Clinical Practice Guidelines
A reference manual of best practice in pedorthic care
Second Edition

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Citation:

About the Pedorthic Association of Canada

The Pedorthic Association of Canada is a national association of pedorthists committed to patient well-being and the advancement of the profession of pedorthics.

Our values

We believe in:

• Integrity
• Leading the field of foot orthoses and footwear
• Being the trusted source for information on foot orthoses and footwear
• High standards of education
• The highest ethical standards
• Innovation
• Excellence in reputation
• Visionary leadership

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Disclaimer

The Clinical Practice Guidelines (“CPGs”) of the Pedorthic Association of Canada ("PAC") are intended to help pedorthists make decisions about assessing and treating patients. The CPGs are not intended to be used directly by patients, nor are they intended to serve as an exclusive source of information for pedorthists. PAC sees the CPGs as a useful input into pedorthic decision-making, but makes no claim that the information contained herein offers the only correct or suitable courses of action. The CPGs are not intended to dictate specific courses of treatment. The advice offered in the CPGs is subject to change.

A Note About The Term “Diagnosis”

As pedorthists, we are not licensed to communicate a diagnosis. It is, however, essential that we have a working knowledge of the diagnoses that are presented to us by physicians and other health professionals who are in fact permitted to diagnose. In this publication we therefore use “diagnosis”, “differential diagnosis”, and other related terms in a general way. The intent is to discuss and respond to diagnoses, not to actually diagnose.

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In Memoriam

This second edition of PAC’s Clinical Practice Guidelines is dedicated to the memory of pedorthist Michael Forgrave.

Mike chaired the Task Force that produced our first edition in 2012. He was passionate, energetic, and cared deeply about the advancement of pedorthic practice in Canada and beyond.

Mike rallied volunteers together in common cause and we are grateful. When he passed away in 2015, we lost a leader and a friend.
The PAC Clinical Practice Guidelines Team

The Pedorthic Association of Canada is grateful for the remarkable volunteer contributions of our writers, reviewers, photographers, and Task Force members. Including our professional team and external suppliers and contractors, more than 100 individuals have participated in this project.

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Preface

As Canadian Certified Pedorthists, we are fortunate to work in a dynamic profession where dialogue about best practice is robust and where we share a common interest in meeting the needs of our patients.

In 2010, the Pedorthic Association of Canada (PAC) first expressed the need for a formal Clinical Practice Guidelines (CPGs) document to create a platform for greater dialogue and to advance the profession in the eyes of the general public, other health care providers, and policymakers. The result was a 2012 manual that has since stimulated discussion, gained acclaim, and has had a positive impact on the practice of pedorthics in Canada.

In 2017, the PAC Board of Directors determined that there had been enough developments in the field that it was appropriate to review our CPGs and produce a new edition for launch in 2018.

We assembled as a volunteer Task Force to oversee the project. This work included:

- distributing a survey to all Canadian Certified Pedorthists to gather their feedback to the first edition and their suggestions for the second;
- based on that feedback and our own assessments, determining together what chapters should be removed, combined, updated, and added;
- marking up individual chapters with recommendations for updates to reflect new research and changes in best practice;
- working with original writers to update their chapters accordingly;
- working with new writers to update chapters from 2012 where the original writers were not available;
- identifying and inviting new writers to produce our seven new chapters;
- identifying and inviting reviewers to participate in a double-blind review process for our new chapters; and
- working alongside our Managing Editor to bring the project to closure.

These efforts led to this new reference manual. We hope that it will make a positive difference for Canadian Certified Pedorthists and their patients.

We thank all of our writers and reviewers for their commitment to this initiative. We are also very grateful for the generosity of those people who shared photos with us for the first and second editions. It has taken literally thousands of volunteer hours to develop and update these guidelines – a remarkable contribution for all involved. It should be a source of pride for our entire profession.

President Peter Morcom, C. Ped (C) and the entire PAC Board of Directors have been enthusiastic supporters and we thank them. We are also very grateful for the efforts of Jonathan Strauss, our Chief Executive Officer, and his team at the PAC office; and for the contribution of our Managing Editor, Stu Slayen. We also acknowledge the generosity of Western University for offering library access to our writers.

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April 2018
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Diabetes Mellitus

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Key Messages

• The number of individuals in Canada affected by diabetes is projected to rise to more than 4.2 million by the year 2020.
• Diabetic foot ulcerations are a frequent complication of diabetes and are the leading cause of hospitalization of people with diabetes mellitus.
• Diabetes and diabetes foot complications place a financial burden on the Canadian health care system.
• Many diabetic foot ulcers and complications are preventable and treatable with patient education, custom-made orthoses, orthopaedic footwear, modified orthopaedic footwear, and custom-made orthopaedic shoes.
• Pedorthists are important members of the diabetic health care team.

Keywords

autonomic, arterial, metabolic, motor neuropathy, neuropathy, numbness, peripheral, sensitivity, sensory, ulceration, vascular

Introduction

Diabetes mellitus is a metabolic disease in which the body does not produce or properly use insulin that it produces. This leads to high levels of glucose in the blood, which can cause damage to organs, blood vessels, and nerves. The number of individuals with diabetes in Canada is projected to rise to 4.2 million by the year 2020 (Canadian Diabetes Association & Diabetes Quebec, 2011).

Foot and ankle complications of diabetes are influenced by many factors including peripheral neuropathy, peripheral vascular disease, infection, and minor trauma (Reid et al., 2006). Peripheral neuropathy and peripheral vascular disease are the two most common underlying complications of diabetes mellitus and the primary causative factors for foot ulcerations and amputations in diabetic patients. Diabetic foot ulcerations are a frequent complication of diabetes and are the leading cause of hospitalization in people with diabetes mellitus. The management of diabetic foot complications and foot ulcers is primarily focused on avoiding lower extremity amputations, prompt and effective treatment of the acutely diseased foot, prevention of further problems, and patient education (Giurini & Lyons, 2005). According to the Canadian Diabetes Association, by the year 2020 it’s estimated that diabetes will cost the Canadian health care system $16.9 billion a year. For people with diabetes neuropathy and/or peripheral vascular disease, minor trauma to the foot can potentially lead to skin ulcerations, infections, and ultimately gangrene, resulting in lower extremity amputations. Diabetic foot complications place financial and economic burdens on the health care system and as well as social and financial burdens on the diabetic patients with foot complications and their families (Giurini & Lyons, 2005). Foot and ankle complications of diabetes are influenced by peripheral neuropathy, peripheral vascular disease, infection, and minor trauma (Reid et al., 2006).

The objectives of pedorthic care for the diabetic foot is to relieve areas of excessive plantar pressures, reduce shock, reduce shear or horizontal movement of the foot within the shoe, to accommodate deformities by stabilizing and supporting both fixed and flexible deformities, limit motion of unstable joints, and educate patients regarding proper shoe selection and fit (Janisse, 1994). Many of the diabetic foot ulcers and complications in diabetic patients are preventable and treatable with patient education, properly designed and fitted custom-made orthoses, orthopaedic footwear, modified orthopaedic footwear, and custom-made orthopaedic shoes. Canadian Certified Pedorthists are important members of the
diabetic health care team, providing custom orthoses, orthopaedic shoes, orthopaedic shoe modifications, and custom-made orthopaedic shoes. Pedorthic treatment modalities can be crucial in preventing both the initial foot ulcerations and foot ulcer recurrence in diabetic patients (Janisse, 1994).

**Peripheral Neuropathy**

Peripheral neuropathy is the gradual loss of nerve function in the feet and legs due to diabetes. Peripheral neuropathy affects the sensory, motor, and autonomic nerves. Nerve damage subsequently affects the patients’ ability to perceive pain, pressure, touch, and temperature (O’Loughlin, McIntosh, Dinneen, & O’Brien, 2010). The most common and significant change is the loss of feeling or sensation of touch. The loss of protective sensation is the most significant predictor of foot ulcerations and complications in people with diabetes. The inability of a diabetic patient to feel pain sensation places this patient at significant risk for foot problems and complications in the future (Giurini & Lyons, 2005).

Peripheral sensory neuropathy is one of the major underlying causes of foot ulcerations in patients with diabetes because the lack of painful feedback allows for unnoticed repetitive trauma to occur to the feet (Giurini & Lyons, 2005).

**Etiology**

Reid *et al.* (2006) found that peripheral neuropathy presented in 50% of patients by 25 years after the diagnosis of diabetes, increases the risk of diabetic foot ulcers sevenfold. Peripheral neuropathy affects more than 50% of people with type 2 diabetes mellitus over 60 years of age (Paton, Bruce, Jones, & Stenhouse, 2009).

Sensory neuropathy is the loss of sensation, leaving the patient incapable of sensing pain and pressure. The patient has no sense of identity with their feet (Elftman, 1992). Sensory nerves send messages from the muscles back to the spinal cord and the brain. Sensory nerve damage often results in tingling, numbness, pain, and extreme sensitivity to touch. Loss of pain and temperature sensation predisposes the area of involvement to repeated injuries such as burns, abrasions, and mechanical stresses (Birke, Novick, Hawkins, & Patout, 1991). Altered proprioception makes the patient unable to determine the position of the foot (O’Loughlin *et al.*, 2010).

Motor neuropathy can produce foot deformities that can place the diabetic foot at a significant higher risk for foot ulcerations. Motor neuropathy is characterized by intrinsic muscle atrophy and results in contracted digits and a displaced fat pad (Botros *et al.*, 2010). Motor neuropathy affects the smaller muscles of the foot and causes weakness, atrophy, and deformity. The deformities include clawing of the lesser toes, prominent metatarsal heads, and limited joint mobility (O’Loughlin *et al.*, 2010). This makes the metatarsal heads more prominent, thus leading to increased plantar pressures and the potential risk for foot ulcerations. The loss innervations of the intrinsic musculature of the foot, resulting in common foot deformities such as hammer toes, claw toes, and plantarflexed metatarsal heads (Giurini & Lyons, 2005). Motor nerves send impulses from the brain and spinal cord to all of the muscles in the body. Motor nerve damage can lead to muscle weakness, difficulty walking or moving, cramps, and spasms. Motor neuropathy produces common abnormal gait characteristics in the neuropathic population (Elftman, 1992).

Autonomic neuropathy is the loss of the autonomic system function, resulting in the absence of sweat and oil production and leaving the skin dry and non-elastic (Elftman, 1992). Autonomic nerves control involuntary or semi–voluntary functions, such as heart rate, blood pressure, digestion, and sweating. Autonomic neuropathy may reduce sweating and increase the temperature of the patient’s foot, predisposing to infection. The reduction in sweating and increased temperature predispose to cracking of the skin and consequently ulceration (O’Loughlin *et al.*, 2010).

**Key messages**

- Loss of protective sensation places the diabetic patient at a higher risk for developing foot ulcers and complications.
- Treatment includes accommodation, reduce plantar pressures and shear forces, and shock absorption.
- Patient education regarding proper footwear selection and fit, custom-made orthoses, footwear modifications, and custom-made orthopaedic shoes.

**Signs and symptoms**

- Numbness or tingling in the toes and feet
- The neuropathic foot is warm to touch and well perfused with palpable pulses
- Burning sensation or freezing pain
- Sharp, jabbing pain
- Extreme sensitivity to touch
- Difficult sleeping because of feet and leg pain
• Loss of balance and coordination
• Muscle weakness
• Difficulty walking
• Symptoms depend on the nerve or nerve group that is damaged

**Differential Diagnosis**

Any condition causing loss of sensation in the lower limbs such as alcoholism, Guillain–Barre syndrome, leprosy, and Charcot–Marie–Tooth disease can mimic the signs and symptoms of peripheral neuropathy (Decker & Albert, 2002).

**Common Testing**

Peripheral sensory neuropathy can be adequately detected by using a 5.07 (10g) Semmes–Weinstein monofilament wire as a screening tool to identify at-risk patients (Schaper, van Netten, Apelqvist, Lipsky, & Bakker, IWGDF, 2017). The 5.07 (10g) Semmes–Weinstein monofilament wire is an effective and inexpensive device to identify diabetic patients at risk for foot ulcerations. Monofilament testing is a single point perception test and requires the examiner to place the monofilament on the skin, press until the monofilament bends, and remove the monofilament from the skin surface. Diabetic patients unable to perceive the monofilament are at a significantly higher risk for the development of foot ulcerations than patients who are able to detect the monofilament (Schaper et al., 2017). The inability to feel the wire implies a lack of adequate protective sensation in the foot. Testing for vibratory sensation can be accomplished by using a biothesiometer. A biothesiometer provides more objective documentation of a patient’s ability to perceive vibratory sensation (Giurini & Lyons, 2005). This instrument is essentially an electric tuning fork that uses repetitive mechanical indentation of skin delivered at a prescribed frequency and amplitude.

**Common Treatment**

• Custom orthoses
• Properly fitted therapeutic or orthopaedic shoes
• Footwear modifications
• Custom-made orthopaedic shoes

The management of peripheral neuropathy requires the need for regular follow-up appointments for the prevention and early detection of foot ulcerations (Dang & Boulton, 2003). Offloading is an important component of the treatment to prevent and heal plantar ulcers in patients with diabetes. Treatment of the diabetic neuropathic foot requires accommodation, relief of pressure or shears forces, and shock absorption with modalities such as custom-made orthoses, therapeutic or orthopaedic shoes, footwear modifications, and possibly custom-made orthopaedic footwear.

**Footwear**

Appropriate footwear is essential for people with diabetes and peripheral neuropathy. The footwear should be of adequate width and depth for the diabetes feet. A research study conducted by (Praet & Louwerens, 2003) found that the most effective way to offload the forefoot of patients with neuropathic feet is through the use of the rocker sole shoe principle. They found that shoes with the rocker bottom principle reduced plantar pressure by 35–65% underneath the heel and the central metatarsal heads (Praet & Louwerens, 2003). Patients with an at-risk diabetic foot should be urged to not walk barefoot but to wear protective footwear both at home and outside at all times (Bus et al., 2008). Diabetic patients have a tendency to wear their shoes too short, especially if they have any degree of neuropathy present. There should be at least one finger to a thumb space from the patient’s longest toe to the end of the shoe and the widest part of the shoe should fit the widest part of the patient’s feet. This is especially important for patients with diabetes and peripheral neuropathy. Patients with an at-risk diabetic foot should be urged
to not walk barefoot but to wear protective footwear at all times, both at home and outside (Bus et al., 2008). Therapeutic or orthopaedic shoes with the rocker sole feature, firm heel counter, good midfoot stability, and only flexes in the forefoot will benefit patients with diabetes and peripheral neuropathy. Therapeutic or orthopaedic shoes designed with extra width and depth, seamless uppers, stable supportive midsoles with rocker soles should be recommended for patients with diabetes and peripheral neuropathy. Patients with diabetes and peripheral neuropathy need to make sure that their shoes are properly fitted by a footwear specialist.

**Custom orthoses**

Custom orthoses are frequently prescribed for individuals with diabetic neuropathy to offload high pressures from the metatarsal heads and other areas, which reduces the risk of plantar foot ulcerations. According to Paton et al. (2011) insoles are effective in reducing ulceration rate and peak pressure in people with diabetes and peripheral neuropathy. Treatment objectives for diabetic accommodative custom orthoses include attenuating ground reaction forces, reducing shear forces, relieving areas of excessive pressure, and accommodating, stabilizing, and supporting foot deformities. Custom orthoses provide the important interface between the foot and the shoe, and together with orthopaedic shoe modifications, offer the most direct approach to the accommodation of any lesions or deformities (Giurini & Lyons, 2005). Custom orthoses are the most effective way to significantly reduce peak plantar pressures and facilitate the healing process.

**Custom-made orthopaedic shoes**

Diabetic patients with peripheral neuropathy are sometimes prescribed custom-made orthopaedic shoes due the foot deformities and inability to fit into regular off the shelf therapeutic or orthopaedic shoes. The goals of custom-made orthopaedic footwear are to redistribute and reduce plantar foot pressures, and to prevent foot ulcer recurrence (Bus, 2008).

**Vascular Impairment**

Peripheral vascular disease (PVD) is a condition of the blood vessels that leads to the narrowing and hardening of the arteries that supply the legs and feet. Peripheral vascular disease occurs as the circulation to the lower extremities is impaired. Peripheral vascular disease is caused by arteriosclerosis or hardening of the arteries. Peripheral vascular disease is commonly associated with diabetes mellitus. Diabetes can contribute to the narrowing of the arteries and decreased circulation in the lower legs and feet. The vessels commonly affected by diabetic vascular disease are the anterior and posterior tibial arteries (Giurini & Lyons, 2005).

**Etiology**

Peripheral arterial disease (PAD) is four times more common in people with diabetes than in those without diabetes (Botros et al., 2010). The prevalence of PAD in people with diabetes has been reported to be as high as 30% and it is known to have a negative impact on health-related quality of life as a result of chronic pain, intermittent claudication, loss of mobility, and function (Burns, Wegenert, Begg, Vicaretti, & Fletcher, 2009). Peripheral arterial disease usually appears in older adults with a 2:1 male predominance (Decker & Albert, 2002). Poor circulation to the toes is common in people with peripheral arterial disease. Peripheral arterial disease is an important risk factor for lower extremity amputations in patients with chronic diabetic foot ulcerations (Dang & Boulton, 2003). Diabetic patients with peripheral arterial disease might not have the classical symptoms (claudication, rest pain) because of the presence of severe peripheral neuropathy (Dang & Boulton, 2003). Diabetic patients present late as the presence of neuropathy may mask the symptoms of intermittent claudication, and hence these patients do worse than their non-diabetic counterparts (Dang & Boulton, 2003). Arterial insufficiency can lead to non-healing of ulcerations once they have developed.
Foot ulcers in patients with diabetes mellitus often have mixed ischemic and neuropathic components. Arterial insufficiency occurs in the diabetic lower limb. Wound healing depends on an adequate blood supply and ischemia impairs healing by reducing the supply of oxygen, nutrients, and soluble mediators that are involved in the repair process (O’Loughlin et al., 2010). Peripheral vascular disease also contributes to tissue breakdown and delayed wound healing, but is rarely the primary or initial cause of foot ulcers in patients with diabetes (Birke, Patout, & Foto, 2000). Peripheral vascular disease is also an important risk factor for diabetic foot ulcers and lower extremity amputations in patients with diabetes.

**Key messages**
- Peripheral vascular disease is an important risk factor for diabetic foot ulcers and amputations.
- Arterial insufficiency can lead to non-healing of ulcers once they have developed.

**Differential Diagnosis**
- The presence of peripheral neuropathy may mask symptoms of intermittent claudication
- Raynaud’s disease/phenomenon
- Pseudoclaudication
- Metatarsalgia

**Subjective findings**
- Colour changes in the skin
- Pain associated with walking a short distance relieved with a period of rest in either the calf or foot
- Pain at rest, especially at night that is relieved with dependency of the legs
- Toe and foot ulcers that do not heal

**Objective findings**
- Absent pedal pulses
- Intermittent claudication
- Purple or reddish purple colour changes in the feet
- Cool skin to touch in specific areas of the feet and legs
- When the leg is lowered from an elevated position there is delayed venous filling
- Decreased hair growth on the legs

**Common Testing**
Examination of the vascular status of the foot includes palpation of pulses, and the measurement of the ankle-brachial pressure index (ABPI) using Doppler ultrasound or toe blood pressure index (TBI) to factor out falsely high ABPI resulting from medial calcinosis (O’Loughlin et al., 2010). Vascular impairment should be suspected when one limb is significantly colder or distal portions of the foot show an extreme drop in temperature (Elftman, 1992). Palpation of the pedal pulses and/or the measurement of the ABPI will assess the lower limb vascular supply. The absence of both pedal pulses and/or an ABPI <0.8 in the affected foot is clinical evidence of ischemia (Dang & Boulton, 2003). A blanching test is performed by pressing a finger on the dorsum of the dependent foot to produce a noticeable lightening of the skin colour. Normally, erythema should return within five second if not there is decreased local perfusion microcirculation time. (Botros et al., 2010). Diabetic patients with intermittent claudication, ischemic ulcers, or gangrene need a referral to a vascular surgeon (Dang & Boulton, 2003).

**Common Treatment**
The management of vascular disease in the patient with diabetes mellitus is an essential and important consideration. Peripheral vascular disease by itself is responsible for only a small percentage of diabetic foot ulcerations. Only 15% of all diabetic foot ulcers are purely ischemic (Giurini & Lyons, 2005).

**Surgery**
All patients with tissue loss and arterial disease should be considered for arterial reconstruction. In patients with extensive tissue loss or gangrene of the foot, restoration of pulsatile blood flow to the foot is required for proper healing (O’Loughlin et al., 2010). The effective revascularization procedures include angioplasty (balloon and laser), stenting, atherectomy, and bypass grafts to foot vessels. If vascular reconstruction surgery is not possible, then gangrenous toes may have to be amputated (O’Loughlin et al., 2010).

**Footwear**
A research study conducted by (Praet & Louwerens, 2003) found that the most effective way to offload the forefoot of patients with neuropathic feet is through the use of the rocker sole shoe principle. They found that shoes with the rocker bottom principle reduced plantar pressure by 35–65% underneath the heel and the central metatarsal heads (Praet & Louwerens,
Custom orthoses

Custom orthoses are frequently prescribed for individuals with diabetic neuropathy and peripheral vascular disease to offload high pressures from the metatarsal heads and other areas, which reduces the risk of plantar foot ulcerations. Custom orthoses provide the important interface between the foot and the shoe, and together with orthopaedic shoe modifications offer the most direct approach to the reduction of potentially damaging tissue stresses on the plantar aspect of the foot (Owings et al., 2008). Custom-made orthoses can effectively help to alleviate excessive plantar pressures by evenly distributing the pressure, reducing shock absorption through the use of shock-absorbing materials, and accommodating foot deformities. Custom-made orthoses should be made of a soft, closed-celled material that will allow for good shock absorption and accommodation of any lesions or deformities (Giurini & Lyons, 2005).

Footwear modifications

Footwear modifications including rocker soles, steel shank rocker soles, medial or lateral buttresses, balloon patches, and lace-to-Velcro conversions are each very effective methods of optimizing orthotic and orthopaedic shoe treatment methods. Modifications to the upper of the shoe are commonly used to accommodate bony prominences and deformities such as bunions, hammer toes, and claw toes. Rocker sole and steel shank rocker sole modifications help to reduce excessive forefoot plantar pressure in patients with diabetes, peripheral neuropathy, and peripheral vascular disease.

Diabetic Foot Ulcers

A diabetic ulcer is a break or hole in the skin which leaves the foot and/or body vulnerable to invasion from harmful bacteria that can cause infection. Ulcers can be either superficial or deep in the skin. Diabetic foot ulceration is a serious and debilitating complication of diabetes mellitus. Approximately 15% of individuals with diabetes will develop foot ulcers in their lifetime (Dang & Boulton, 2003). Fifteen to 20% of these ulcers will result in lower extremity amputations (Decker & Albert, 2002). Diabetic foot ulcers are the most common cause of lower extremity complications in patients with diabetes.

Etiology

The etiology of diabetic foot ulcers is caused by many factors. Peripheral neuropathy and peripheral vascular disease are the two major underlying risk factors for diabetic foot ulcers, subsequently leading to infections and amputations. Foot deformities, limited joint mobility, partial foot amputations, and other structural deformities often predispose diabetic patients with peripheral neuropathy to abnormal weight-bearing, areas of concentrated pressure, and abnormal shear forces that significantly increase their risk of ulceration (Wu, Crews, & Armstrong, 2005). The following foot conditions are associated with an increased risk of amputation: proprioceptive loss; altered biomechanics; evidence of increased pressure (erythema, hemorrhage under a callus); bony deformity; peripheral vascular disease (decreased or absent pedal pulses); a history of ulcers or amputation; and severe nail pathology (O’Loughlin et al., 2010). Many diabetic foot ulcers have footwear-related causes such as improperly fitting shoes or inappropriate shoes (Janisse, 1994). Foot stress results from extrinsic factors such as footwear and intrinsic factors such as deformity and limited joint mobility (Birke et al., 2000). The most common mechanism of injury results from repetitive unperceived pressure on bony prominences and pre-existing deformities (O’Loughlin et al., 2010). Ill-fitting footwear is also a major cause of foot ulcers, foot complications, and amputations in patients with diabetes. Most diabetic ulcerations are either neuropathic or neuroischemic.

There are three main types of ulcers: neuropathic, arterial, and venous.

Neuropathic ulcers in diabetes are due to chronic repetitive trauma and/or friction on weight-bearing areas of the foot. Neuropathic ulcers are generally painless, round, surrounded by callus, and located...
over prominent bony areas of the toes or plantar surface of the foot. The most common sites of ulceration are the first metatarsal head and the plantar aspect of the great toe. The foot is warm, dry, and pink.

Arterial ulcers occur when there is diminished arterial flow to the foot and lower leg resulting in ischemia. Arterial ulcers occur near bony prominences and are due to a lack of blood supply. Arterial ulcers are located on the tips or between toes, heels, metatarsal heads, side or sole of foot, and above the lateral malleoli (Elftman, 1992). Arterial ulcers have a poor prognosis. The misdiagnosis of an arterial ulcer as a venous ulcer can lead to serious complications (Elftman, 1992).

Venous ulcers occur due to the failure of the deep veins of the leg to return blood, resulting in pooling or stasis in the lower leg. Venous ulcers typically occur on the medial ankle and lower leg and proceeded by itching and swelling. The surrounding skin may be a brownish colour, flaky, and edematous with pain.

**Key messages**
- Loss of protective sensation, history of ulceration, and reduced circulatory perfusion are important factors in the development of foot ulcers.
- Ill-fitting footwear is a major cause of foot ulcers and amputations.
- Important risk factors for ulcerations and lower extremity amputations include loss of protective sensation, peripheral vascular disease, previous ulcer, and foot deformity.

**Subjective findings**
- Painless sore on skin of foot that does not heal

**Objective findings on assessment**
- Superficial or deep ulcer, usually plantar to the metatarsal heads and hallux
- Diabetic with loss of protective sensation in feet
- Drainage with or without blood on socks or in shoes
- Foot deformity such as contracted toes and prominent metatarsal heads

**Development of foot ulcers**
- Neuropathy, deformity, callus, and plantar pressures
- Peripheral arterial disease
- Ill-fitting shoes
- Trauma

Diabetic foot ulcers occur as a result of various factors. Diabetic foot ulcers tend to occur in the following areas: areas subjected to weight-bearing, such as the heel, plantar metatarsal heads, the tips of the most prominent toes, and tips of hammer toes. Peripheral neuropathy and peripheral arterial disease are the major contributors to the risk of non-healing diabetic foot ulcers. Important predictive factors for the development of foot ulceration include high plantar pressure, which usually occurs at sites with bony prominences such as the metatarsal heads, and the presence of callus build-up, which acts as a foreign body ultimately causing ulcerations (Dang & Boulton, 2003). Many diabetic foot ulcerations occur at the toes due to poorly fitting footwear. Shoes lacking adequate width and depth in the toe-box area or made of non-stretchable materials pose the greatest danger for the development of foot ulcerations.

**Common Treatment**
The treatment of superficial ulcers entails regular debridement, dressings, and relief of pressure around the ulcer by using a total contact cast (TCC) or walking boot. Patients with neuroischemic foot ulcers need proper vascular assessment, as these patients may require vascular reconstruction to aid the healing of their ulcers, thus reducing the probability of amputations (Dang & Boulton, 2003). A Harris mat is a simple and inexpensive tool that can be helpful in identifying areas of high plantar pressures in patients who present with diabetic ulceration. Photo by Ryan T. Crews.
with diabetes, peripheral neuropathy, and peripheral vascular disease.

**Total contact casting (TCC)**

Total contact casting (TCC) is considered by many to be the “gold standard” in achieving pressure redistribution. (Wu et al., 2005). Total contact casting (TCC) method is a common and successful treatment plan for plantar ulcerations. The technique has become known as TCC because it employs a well-moulded, minimally padded cast that maintains contact with the entire plantar aspect of the foot and lower leg (Wu et al., 2005). Total contact casting redistributes plantar pressures, prevents direct trauma to the wound, reduces edema, and provides immobilization to the joints and soft tissue. TCC is quite effective in treating the majority of non-infected, non-ischemic plantar diabetic foot wounds, with healing rates ranging from 72% to 100% over a course of 5–7 weeks (Wu et al., 2005).

**Surgery**

Repeated ulcerations may warrant consideration of surgical correction of any underlying structural deformities. Metatarsal osteotomies, digital arthroplasties, and metatarsal head resections have all proven useful in the prevention of recurrent foot ulcerations (Giurini & Lyons, 2005).

**Pedorthic management**

- Custom-made foot orthoses to reduce plantar pressure
- Properly fitting therapeutic or orthopaedic footwear
- Shoe modifications such as rocker soles, steel shank rocker soles, flares, and buttresses
- Custom-made orthopaedic footwear

**Footwear**

Inappropriate footwear causing increased mechanical stress at the plantar and dorsal surfaces of the foot has been reported as probably the most common cause of foot ulcerations in patients with diabetes. Therefore, the reduction of pressures through appropriate therapeutic footwear seems to be of paramount importance for preventing diabetic foot ulcerations (Bus, 2008). Therapeutic shoes can be used for preventing plantar ulcerations in the at-risk diabetic foot (Bus et al., 2008). Properly fitting shoes should be at least 1 cm longer than the longest toe; the widest point should correspond with the widest part of the foot, and match the contours of the feet. Patients with an at-risk diabetic foot should be urged to not walk barefoot but to wear protective footwear at all times, both at home and outside (Bus et al., 2008). Patients should be encouraged to wear shoes with laces as opposed to loafers, as a better more supportive fit is provided by a laced-up shoe than a slip-on shoe (Giurini & Lyons, 2005). Footwear with a soft upper and deep toe or rounded toe-box is recommended to accommodate deformities such as bunions or hammer and claw toes.

**Custom orthoses**

Custom-made orthoses are frequently prescribed for patients with diabetes and peripheral neuropathy to offload high pressures from the metatarsal heads and other areas, which reduces the risk of plantar ulcerations (Owings et al., 2008). The treatment objectives for diabetic accommodative orthoses include: attenuating ground forces, reducing shear forces, relieving areas of excessive pressure, and accommodating, stabilizing, and supporting deformities. Proper offloading is vital for those patients with neuropathic foot lesions. The custom orthoses provide the important interface between the foot and the shoe and, together with outsole modifications, offer the most direct approach to the reduction of potentially damaging tissue stresses on the plantar aspect of the foot (Owings et al., 2008). Custom-made orthoses should be made of a soft, closed-celled material that will allow for good shock absorption and accommodation of any lesions or deformities (Giurini & Lyons, 2005). The diabetic tri-density custom orthoses is the gold standard total contact accommodative device designed specifically for the “high-risk” diabetic patient wearing extra-depth footwear. Excavation accommodations are effective for offloading and/or accommodating bony prominences such as Charcot foot deformities or bony prominences. Sweet spots (poron-filled cavity) are an effective way to decrease ground reaction forces and shearing forces on sensitive or high-risk bony prominences in patients with diabetes and peripheral neuropathy, for example the navicular or cuneiform.

**Footwear modifications**

Footwear modifications including rocker soles, steel shank rocker soles, medial or lateral buttresses, balloon patches, and lace-to–Velcro conversions are each very effective methods of optimizing orthotic and orthopaedic shoe treatment. Modifications to the upper of the shoe are commonly used to accommodate bony prominences and deformities such as bunions, hammer toes, and claw toes. In recalcitrant forefoot plantar ulcerations, a steel shank rocker sole modification combined with total contact orthoses is the most effective way to significantly reduce peak
plantar pressures and facilitate the healing process. Rocker sole and steel shank rocker sole modifications help to reduce excessive forefoot plantar pressure in patients with diabetes, peripheral neuropathy, and peripheral vascular disease. The rocker sole modification helps to reduce forefoot planar pressure in patients with diabetes. The size and positioning of the rocker affects where it acts as a fulcrum, and hence the area of the foot that receives pressure relief.

**Charcot Foot**

Charcot foot – or Charcot arthropathy – is a syndrome consisting of fractures and dislocations in the diabetic and non-diabetic neuropathic foot and ankle that could result in deformity, ulceration, and the risk of amputation (Verity, Sochocki, Embil, & Trepman, 2007). It is a progressive condition that is characterized by pathological fractures, joint dislocation, and destruction of the pedal architecture. In 1868, Jean-Martin Charcot identified neuropathic joints with an unusual pattern of bone destruction in patients with tabes dorsalis. The first description of neuroarthropathy occurring in diabetes mellitus was published in 1936. Charcot foot is one of the major complications of diabetes mellitus. The Charcot foot in diabetes patients poses many clinical challenges in its diagnosis and management (Rogers et al., 2011).

**Etiology**

The Charcot foot deformity occurs most often in patients with diabetic neuropathy and patients with other neuropathic predisposing conditions. The Charcot foot deformity has been documented to occur as a consequence of various peripheral neuropathies; however, diabetic neuropathy has become the most common etiology (Rogers et al., 2011). The Charcot foot deformity sometimes occurs for no apparent reason and often appears with redness, swelling, and pain. The interaction of several factors (diabetes, sensory-motor neuropathy, autonomic neuropathy, trauma, and metabolic abnormalities of bone) results in an acute localized inflammatory condition that could lead to varying degrees and patterns of bone destruction, subluxation, dislocation, and deformity (Rogers et al., 2011). Predisposing factors for this condition include peripheral neuropathy, increases in local blood flow, excessive osteoclastic activity, unrecognized injury, and continued repetitive stress. The hallmark deformity associated with the Charcot foot deformity is midfoot collapse, described as a “rocker-bottom” foot. The “rocker-bottom” deformity is prone to increased pressure and ulceration. Two well recognized deformities develop: the “rocker bottom” foot associated with midtarsal bone destruction and subluxation; and a marked, pronated deformity resulting from medial displacement of the talonavicular joint or laterolantar calcaneocuboid dislocation. The tarsometatarsal (Lisfranc) joint is the most common site of arthropathy, with the initial involvement usually occurring on the medial column of the foot (Birke et al., 1991).

Two theories (neurotraumatic and neurovascular) explain the pathogenesis of the Charcot foot deformity. The neurotraumatic theory attributes bony destruction to the loss of pain sensation and proprioception combined with repetitive and mechanical trauma to the foot. The neurovascular theory suggests that joint destruction is secondary to an autonomically stimulated vascular reflex that causes hyperemia and periarticular osteopenia with contributory trauma (Sommer & Lee, 2001). Repeated unrecognized microtrauma or an identifiable injury may be the inciting factors of Charcot foot.

Approximately 50% of patients with Charcot foot will remember a precipitating event such a slip, or a minor traumatic event such as an ankle sprain or previous unrelated foot procedure as an antecedent event (Caput et al., 1998). Charcot foot is frequently misdiagnosed and mistreated, leaving the patient with deformities requiring additional medical intervention or expensive footwear (Elftman, 1992). Inflammation plays a key role in the pathophysiology of the development of the Charcot foot deformity and is the earliest clinical indication (Rogers et al., 2011).

**Key messages**

- Early intervention and treatment
- Offloading is the most important initial treatment recommendation
- Support flexible joints and deformities and accommodate rigid joints and deformities
- Relieving pressure and improving gait mechanics

**Signs and symptoms**

- Feet look swollen, warm and red
- Pain and tenderness are usually absent because of sensory neuropathy
- Rocker bottom midfoot deformity might be present (Hallmark deformity)
- Ulceration in the midfoot might be present
Differential Diagnosis

- The acute Charcot foot can mimic cellulitis or less commonly deep venous thrombosis.
- Laboratory tests, including radiographs, might not show changes in the acute stage to differentiate Charcot foot disease from other diagnoses.
- Osteomyelitis, arthritis, and soft tissue infection are mimicking conditions.

Common Testing

Radiographs are the primary initial imaging method for evaluation of the foot in diabetic patients (Rogers et al., 2011). X-rays should be the initial imaging performed, and one should look for subtle fractures or subluxations if no obvious pathology is visible. Skin temperature assessment, as increased warmth is the first indicator of inflammation in an insensate foot and may be the first sign of an acute Charcot foot. The diagnosis of active Charcot foot is primarily based on history and clinical findings but should be confirmed by imaging (Rogers et al., 2011). Magnetic resonance imaging (MRI) or nuclear imaging can confirm clinical suspicions in the presence of normal-appearing radiographs (Rogers et al., 2011). An MRI allows detection of subtle changes in the early stages of active Charcot foot when X-rays could still be normal (Rogers et al., 2011). An MRI is very useful in making the diagnosis at its earliest onset before such changes become evident on plain films (Rogers et al., 2011).

Stages of Charcot foot

Stage 0 – Prodromal period: includes dermal flush/redness and increased skin temperatures, with or without local edema and bounding pulses. There is evidence of instability of the foot. X-ray evidence might be seen.

Stage 1 – Developmental stage: an acute destructive period that is induced by minor trauma resulting in fragmentation of bone and joint dislocation and subluxation. This is the most important stage for clinicians to recognize and where they can make the greatest difference in prevention.

Stage 2 – Coalescence stage: The patient presents with lessening of edema and healing of fractures.

Stage 3 – Reconstruction stage: Healing of bone and remodelling on X-ray and evidence of deformity (Botros et al., 2010).

Pedorthic management

- Custom-made foot orthoses
- Properly fitting orthopaedic shoes
- Shoe modifications such as rocker soles, medial and/or lateral stabilizers, buttresses
- Custom-made orthopaedic shoes

The management goal of the Charcot foot deformity involves early intervention and immobilization. Offloading the foot and immobilization are the most important treatment recommendations in active Charcot neuropathic osteoarthropathy and can prevent further bone destruction (Rogers et al., 2011). Offloading at the acute active stage of the Charcot foot is the most important management strategy and could arrest the progression to deformity. The key goals of treatment are to prevent foot deformity and subsequent foot ulcerations. The medical treatment of Charcot foot deformity is aimed at offloading the foot, treating bone disease, and preventing further foot fractures (Rogers et al., 2011). The basic rule is to support the flexible joints and deformities and accommodate rigid joints and deformities. Relieving pressure and improving gait are also important in the treatment of the Charcot foot (Janisse, 1994).

Total contact casting (TCC)

Total contact casting is an effective method for the treatment of neuropathic ulcers and is also very useful in the treatment and healing process of Charcot foot deformity (Verity et al., 2007). The treatment plan for acute Charcot foot is total contact casting until the foot has healed properly. The total contact cast immobilizes the affected foot and ankle, reduces plantar foot pressures and swelling, protects from additional trauma, limits shear stresses on the skin, and maintains patient mobility (Verity et al., 2007). The TCC method provides decreased plantar pressures by increasing weight-bearing over the entire lower leg (Elftman, 1992).

Surgery

Surgical treatment of Charcot arthropathy of the foot and ankle is based primarily on expert opinion. Chronic Charcot foot changes might require surgical treatment including bony reduction, fusion, and reconstruction (Botros et al., 2010). Surgery has generally been advised for resecting infected bone (osteomyelitis), removing bony prominences that could not be accommodated with therapeutic or orthopaedic footwear or custom orthoses, or correcting deformities that could not be successfully accommodated with therapeutic or orthopaedic.
footwear and/or custom orthoses (Rogers et al., 2011). Surgery can be helpful in early stages involving acute fractures of the foot or ankle or in later stages when offloading is ineffective (Rogers et al., 2011).

**Custom orthoses**

Custom-made orthoses are frequently prescribed for patients with diabetes and peripheral neuropathy to offload high pressures from the metatarsal heads and other areas, which reduces the risk of plantar ulcerations (Owings et al., 2008). The treatment objectives for diabetic accommodative orthoses include attenuating ground forces, reducing shear forces, relieving areas of excessive pressure, and accommodating, stabilizing, and support deformities. Custom-made orthoses and orthopaedic footwear are needed to accommodate deformities after healing. Proper offloading is vital for those patients with neuropathic foot lesions. The custom orthoses provide the important interface between the foot and the shoe and, together with outsole modifications, offer the most direct approach to the reduction of potentially damaging tissue stresses on the plantar aspect of the foot (Owings et al., 2008). The diabetic tri-density custom-made orthoses is the gold standard total contact accommodative device designed specifically for the “high-risk” diabetic patient wearing extra depth footwear. Custom-made orthoses should be made of a soft, closed-celled material that will allow for good shock absorption and accommodation of any lesions or deformities (Giurini & Lyons, 2005). Excavation accommodations are effective for offloading and/or accommodating bony prominences such as Charcot foot deformities. Sweet spots (poron-filled cavity) are an effective way to decrease ground reaction and shearing forces on sensitive or high risk bony prominences, for example the navicular or cuneiform.

**Footwear modifications**

Footwear modifications including rocker soles, steel shank rocker soles, medial or lateral buttresses, balloon patches, and lace-to-Velcro conversions are each very effective methods of optimizing orthotic and orthopaedic shoe treatment methods. Modifications to the upper of the shoe are commonly used to accommodate bony prominences and deformities such as bunions, hammer toes, and claw toes. Rocker sole and steel shank rocker sole modifications helps to reduce excessive forefoot plantar pressure in patients with Charcot foot deformity, diabetes, and peripheral neuropathy. The rocker sole modification helps to reduce forefoot planar pressure in patients with Charcot foot deformity and diabetes. The size and positioning of the rocker affects where it acts as a fulcrum, and hence the area of the foot that receives pressure relief.

**Custom-made orthopaedic shoes**

Custom-made orthopaedic shoes may be the best alternative for patients with Charcot foot deformity due to the severe foot deformities that are present.

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Rheumatoid Arthritis

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Key Messages

- Early diagnosis and treatment is vital to reducing disabling effects of rheumatoid arthritis.
- Even in low-disease states or remission, foot joint damage still seems to progress.
- Recognition of signs and symptoms of possible rheumatic disease may speed appropriate diagnostic tests in previously undiagnosed individuals.
- There is strong evidence that foot orthoses and specific footwear can reduce foot pain and improve functional ability.
- Orthosis efficacy may be as much a result of changes in muscle activation, proprioception, and motion facilitation as skeletal alignment and kinematic changes.
- Semi-rigid and rigid orthoses in combination with rocker sole footwear were the most effective in relieving forefoot pain where soft orthoses (Plastazote, Poron) were less effective.
- Footwear modifications are an effective treatment option for the rheumatoid foot.

Keywords

articular, autoimmune, chronic, joint inflammation, nodules, pannus, polyarthritis, progressive, synovial joints

Introduction

Rheumatoid arthritis (RA) is a chronic progressive autoimmune disease of unknown cause in which the body's immune system attacks the joints and surrounding tissues, leading to joint inflammation, pain, joint damage, and disability. RA is a systemic disease that targets synovial joints, but can have extra-articular ramifications in other organs. RA affects approximately 1% of Canadians (Health Canada, 2003) and is more than twice as likely to affect women as men. Rheumatoid arthritis can occur at any age, but tends to begin between the ages of 25 and 60. Children can be affected by a distinct but closely related inflammatory arthritic condition called juvenile idiopathic arthritis, or juvenile rheumatoid arthritis. Rheumatoid arthritis affects all ethnic groups and is found worldwide (Thould & Thould, 1983). Rheumatoid arthritis has significant impacts on pain levels, activities of daily living, social interaction, self perception, mobility, overall health (Graham & Williams, 2016).

Symptoms of initial onset of RA most commonly include insidious fatigue, morning stiffness lasting 30–60 minutes or more, and joint pain and swelling involving the small distal joints including the wrist, metacarpophalangeal joints, proximal interphalangeal joints, and metatarsophalangeal joints (Rheumatoid Arthritis, 2011; Tehlirian & Bathon, 2008). Inflammation of the small joints of the hands and feet in a symmetrical pattern is specifically characteristic of RA (Robinson, 2008; Woodburn, Hennessy, McInnes, & Turner, 2010) and occurs early in the onset of the disease (Otter, Lucas, Springett, Morre, Davies, Cheek, & Walker–Bone, 2010). Other joints that may be affected include elbows, shoulders, hips, knees, ankles, jaw, and neck. In addition to articular symptoms, the early symptoms of RA can also include low grade fever, fatigue, loss of appetite, weight loss, swollen glands, weakness, malaise, myalgias, and anemia. Muscle weakness and atrophy is a common complaint and may be a result of pain from articular and/or periarticular inflammation.

RA is a chronic and potentially debilitating disease for the individual sufferer and also represents a significant economic and health burden on society (Conceicao, Neto, Mendes, Sa, & Baptista, 2015). Foot involvement in rheumatoid arthritis is a major contributing factor to disability causing changes to gait mechanics, difficulties walking and restrictions in activities of daily living (Clark, Rome, Plant, [Rheumatoid Arthritis]...
Rheumatoid Arthritis

O’Hare, & Gray, 2006; Williams, Davies, Graham, Dagg, Longrigg, & Lyons, 2011; Woodburn et al., 2010. Over 90% of patients report foot problems within the first 10 years of disease onset (Clark et al., 2006; Goksel Karatepe, Gunaydin, Adibelli, Kaya, & Duruoz, 2010; Otter et al., 2010; Williams et al., 2011) and 57% report foot and ankle pain in the first year (Riskowski, Dufour, & Hannan, 2011). Disease severity is not the only factor that affects disability. Other contributing factors may include socioeconomic factors, depression, and access to appropriate medical care and supportive resources, which all contribute to the quality of patient care and patient compliance with recommended treatments (Jones, Halbert, Crotty, Shanahan, & Batterham, 2003; Wilson, Hewlett, Woodburn, Pollock, & Kirwan, 2017).

The treatment of RA has taken a more aggressive pharmacological approach utilizing disease-modifying antirheumatic drugs, and more recently biologic agents, which can lead to disease remission in some patients (Hennessy, 2012). Evidence shows that early diagnosis and treatment offers the best chance of limiting the disabling effects of RA on joints and damage to organs (Breedveld & Combe, 2011; Rheumatoid Arthritis, 2011). Early detection of disease leads to better treatment options, so foot care professionals need to be aware of possible signs and symptoms of rheumatic disease in previously undiagnosed patients. For patients who are referred for pedorthic treatment complaining of bilateral joint pain in the metatarsophalangeal and/or interphalangeal joints but have not been diagnosed with rheumatoid arthritis, the clinician should look for the cardinal signs of articular inflammation. These signs include warmth, joint line tenderness, pain on motion (particularly at end range), and intra-articular swelling or effusion. The history should also include questions regarding other joints in the body, mode of onset of pain, quality of pain, and general health of the patient. Episodic swelling, stiffness, weakness, and more generalized symptoms are warning signs of a possible rheumatic condition. A thorough history and assessment of the painful joints may elicit responses that indicate that a referral back to the physician is necessary. If the physician suspects RA, an immediate referral to a rheumatologist will expedite appropriate treatment. With respect to non-pharmacological treatment, research suggests that pedorthic treatment of the foot in RA, using foot orthoses and specific shoe types that reduce plantar pressures and redistribute forces, may play a significant role in the treatment of the RA foot (Riskowski et al., 2011; Williams et al., 2011; Woodburn, Barker, & Helliwell, 2002). Other health care professionals are integral to the team treating the RA patient and may include the rheumatologist, physiotherapist, occupational therapist, dietician, alternative therapist, social worker, and other complementary professionals.

Pathology

In rheumatoid arthritis, the immune system attacks in particular the synovium, a thin layer of tissue that lines the joints. People with RA experience “flares,” or intermittent active phases of the disease where joints may become hot, red, inflamed, and painful. The initial trigger for the autoimmune reaction that leads to rheumatoid arthritis is unknown, but viral infection, bacterial infection, stress, hormones, environmental factors, and smoking have all been suggested as possible triggers (Carty, Snowden, & Silman, 2003; Söderl, Bergsten, & Svensson, 2011).

As a result of the immune response attacking the synovium, a host of inflammatory cells infiltrate the joint, flooding it with inflammatory chemicals that damage the cartilage, subchondral bone, articular capsule, and ligaments. Synovial fluid builds up within the joint, leading to swelling and pain in the joint. The synovium thickens and develops into a granular tissue called a pannus, which releases inflammatory mediators, eroding the cartilage in particular, but also the bone underneath. Once a pannus has formed, scar tissue can develop which can eventually ankylose, fusing the bone ends together. Not all cases of RA progress to the ankylosis stage, but joint erosion, subluxation, and subsequent deformity are common in RA (Marieb & Hoehn, 2010). The pattern of joints affected can help in differentiating rheumatoid arthritis from other conditions. Symmetrical involvement of the small joints of the hands and feet is a hallmark of RA. RA can, on occasion, begin as a sudden onset polyarthritis, or a transient episode of single joint or polyarthritis lasting only a few days or weeks, but this is not the standard presentation (Tehlirian & Bathon, 2008). RA tends to affect joints symmetrically in the body, as opposed to other arthritic diseases such as osteoarthritis which may act asymmetrically, targeting one or more individual joints. The progression of RA is highly variable among individuals with the disease. There are certain biological markers and clinical indicators that can identify individuals who are at high risk of rapid disease progression.

The complications of RA are many. In longstanding disease extra-articular symptoms occur in approximately 40% of RA patients and may include
the formation of firm, fibrous rheumatoid nodules on the elbows, Achilles tendon, metatarsal heads and fingers; shortness of breath or chest pain due to pulmonary fibrosis; pleuraleffusion; pain and redness around the eyes due to scleritis; dry eyes and dry mouth; congestive heart failure as a result of pericarditis; bleeding stomach ulcers; and skin ulcers and infections due to rheumatic vasculitis (Tehlirian & Batson, 2008). Aviña–Zubieta, Choi, Sadatsafavi, Etminan, Esdaile, & Lacaille (2008), in a review of cardiovascular mortality in RA patients, reported that most studies found a 30–50% increase in mortality rates due to cardiovascular disease in RA patients as compared to the general population; however, more recent studies indicate improvements to mortality rates from cardiovascular disease for RA patients, and the authors suggest that earlier diagnosis and the use of more aggressive and newer antirheumatic treatment regimens have improved survival rates (Aviña–Zubieta et al., 2008). The key to achieving longterm disease control is to reduce inflammation promptly and effectively; therefore, early therapeutic intervention with appropriate medications reduces longterm disability and joint and organ damage (Breedveld & Combe, 2011; Rheumatoid Arthritis, 2011; Winston, 2011).

Common Testing and Differential Diagnosis

There is no single test for RA; rather it is a diagnosis reached after combining the results of a thorough patient history, physical examination, blood tests, and diagnostic imaging. A careful examination of musculoskeletal complaints by a physician will determine the direction of the clinical exam and the appropriate lab tests and diagnostic imaging that is required. Polyarthritis of the hands and feet is also a common feature of other rheumatic diseases, as are the constitutional symptoms of fever, weight loss, loss of appetite, swollen glands, anemia, and myalgias. Polyarthritis associated with RA needs to be differentiated from similar rheumatic diseases such as systemic lupus erythematosus (SLE), systemic sclerosis (scleroderma), psoriatic arthritis, rheumatic fever, reactive arthritis, infectious arthritis, and Sjogren syndrome. Blood tests are used to determine levels of inflammation and to identify antibodies and other markers that will help determine a diagnosis of RA. Rheumatoid factor (RF) is an antibody that is not normally present in healthy individuals, but 70–80% of individuals with RA have high levels of rheumatoid factor; however, destructive arthritis can still occur in the absence of RF. A positive anticyclic citrullinated peptide (CCP) antibody test, in combination with elevated erythrocyte sedimentation rate (ESR) and C–reactive protein (CRP) rates and a positive RF, supports a diagnosis of RA (Rheumatoid Arthritis, 2011; Tehlirian & Batson, 2008); however, high levels of RF may also be present in other autoimmune diseases including Sjogren’s syndrome, systemic lupus erythematosus, scleroderma, polymyositis, and dermatomyositis. Radiographs for RA diagnosis may reveal joint space narrowing, bone erosions, deformities or periartricular osteoporosis (Tehlirian & Batson, 2008). MRI and ultrasound may be more sensitive tests in early stages of the disease when some blood tests may not yet be positive.

Contraindications

Maintaining a healthy lifestyle can benefit RA patients including cessation of smoking, appropriate exercise, maintaining a normal weight, healthy diet, and utilizing aids for daily living where necessary. Appropriate referrals for these issues are necessary. Some medications used to alter the disease process or treat symptoms may be contraindicated in some patients due to comorbidity, intolerance, and other factors. For patients taking biologic therapy, immediate referral to their rheumatologist or family physician is necessary if they develop foot or leg ulcerations. Do not try pressure–relieving orthosis or footwear as a first–line treatment for ulcers if the patient has not consulted with their doctor or rheumatologist for possible medication–related issues first. In addition to drug therapies, patient tolerance for conservative or alternative therapies may also dictate the nature of the treatment plan for each individual.

The Patient Perspective

Bone and joint erosion caused by RA is particularly evident in the feet. 16–19% of RA patients initially present with foot complaints (Goksel Karatepe et al., 2010) and 20% already have radiological damage at diagnosis (Baan, Drossaers-Balke, Dubbeldam, & van de Laar, 2011). Otter et al. (2010) found that women reported significantly higher scores for foot pain than men. Patients initially may report forehead pain. The metatarsophalangeal joints (MTP) are one of the most common sites for complaint, especially in early RA (Magalhaes, Davitt, Filho, Battistella, & Bertolo, 2006; Otter et al., 2010). Foot joint involvement impacts negatively on quality of life, leading to walking disability in early disease. One study reported a prevalence of disability in walking of 56.7% in a cohort of 848 patients with RA of less than
The development of subcutaneous nodules occurs in approximately 30% of patients and can further complicate already painful joints. The patient may complain of callus formation under high pressure areas, pain from nodules, and lesions on high friction areas from footwear irritation. Inflammation alters pain perception, lowering the threshold for pain (Hodge, Bach, & Carter, 1999), so higher plantar pressure associated with joint changes is not the only factor at play in the painful RA foot. Dorsal subluxation of the lesser MTP joints can occur, exposing the distal end of the metatarsals to increased pressure leading to callosities and possible ulceration of overlying skin under the metatarsal heads as well as over claw- and hammer toes (Magalhaes et al., 2006). Common foot deformities in RA include hallux valgus, hallux rigidus, splaying of the forefoot, metatarsal subluxation, clawing of lesser toes, rearfoot valgus, and damage to the ankle joint (Baan et al., 2011; Goksel Karatepe et al., 2010).

Most patients with involvement of the rearfoot complain of ill-defined ankle pain, lateral ankle pain associated with valgus deformity and lateral impingement (Li, 2007), plantar heel pain, generalized foot pain, difficulty walking on uneven ground, and difficulty finding footwear. Changes to ankle stability, arthritic involvement of the peritalar joints and tendinopathy of the tibialis posterior tendon may result in damage which contributes to foot deformity (Magalhaes et al., 2006) and specifically to acquired pes planovalgus (Woodburn, Hennessy, McInnes, & Turner, 2010). Suspected dysfunction of the tibialis posterior tendon can be confirmed by an inability to perform a single heel raise or by a lack of inversion of the rearfoot during heel raise.

Patients report a decreased tolerance for walking and standing (Cho, Hwang, Chang, Koh, & Park, 2009; van der Leeden et al., 2008). Joint damage and foot deformities cause functional impairment, disrupting normal gait patterns and restricting activities of daily living (Baan et al., 2011; Clark et al., 2006; Woodburn, Helliwell, & Barker, 2003). They may have generalized foot pain on weight-bearing, joint damage that causes pain on weight-bearing, and pressure sensitive areas of the foot which limit standing and walking activities as well as difficulty finding appropriate footwear to accommodate pain and deformity.

In addition to pain, patients report numbness, stiffness, weakness, and decreased range of motion in the joints. Limited mobility has far reaching implications for the lifestyle of rheumatoid arthritis sufferers. Not only do patients suffer from pain, stiffness, and deformity of the feet, these complications have severe consequences for independence, employment and maintaining social integration (Otter et al., 2010, Wilson et al., 2017). In RA, deformity and pain have an impact on functional ability but it also have a detrimental effects on how the body looks, the perceptions of an individual’s body image, moods, and general quality of life (Goksel Karatepe et al., 2010; Williams & Nester, 2006.). Footwear is often cited as a major factor in dissatisfaction with appearance and can lead to limited social engagement as patients feel they cannot meet fashion requirements for specific activities (Graham & Williams, 2016).

**Common Treatment**

Early and aggressive treatment of RA is the overwhelming recommendation of the rheumatology community (Breedveld & Combe, 2011; Lacaille, 2000; Mäkinen, Hannonen, & Sokka, 2006; Woodburn et al., 2010) to prevent joint destruction and limit disease progression (Breedveld & Combe, 2011; Lacaille, 2000; Rheumatoid Arthritis, 2011). Primary treatment of RA includes early intervention with drug therapies, lifestyle changes, and patient education. Overall treatment goals include reducing inflammation, preventing damage to joints and organs, relieving pain, maintaining mobility, and limiting harmful side-effects of medications (Williams et al., 2011). There is no one single medication that will address the complex progression of RA, but standard treatment paradigms include the use of disease-modifying antirheumatic drugs (DMARDs) (Jones et
DMARDs target biologic processes that fuel inflammation in joints and have an effect on the progression of the disease. They are often combined with non-steroidal anti-inflammatory drugs (NSAIDs) and/or steroids which work to control symptoms of the disease. While NSAIDs work quickly to relieve symptoms they do not have an effect on the underlying disease and do not prevent damage to the joints or organs. Steroids work quickly, but often have side effects, so their use is often short term. There are a number of DMARDs available, and depending on patient response, more than one DMARD may be prescribed, or a combination of drugs including DMARDs and NSAIDs or steroids. Patients need to be carefully monitored due to possible toxicity of DMARD therapy.

There has been a major shift in the treatment of RA in the past two decades as newer biologic agents have been developed which have proven to be successful, preventing damage to the joints and improving the quality of life and lifespan of RA patients, even producing remission (Mäkinen et al., 2006; Winston, 2011). “In patients with well-established RA, biologic agents have been shown to effectively improve clinical, functional and radiographic outcomes and to retard radiographic progression” (Breedveld & Combe, 2011). In more advanced disease, orthopaedic surgery may be indicated due to joint or soft tissue destruction or deformity. Shourt, Crowson, Gabriel, & Matteson (2012) showed a marked decline in orthopaedic surgeries for RA in a more recent cohort of patients compared to those followed from 1980 to 1994, suggesting that recent treatment advances may be reducing the rate of disease progression and therefore the need for as many surgeries (Shourt et al., 2012). In a 2015 review of foot orthosis literature for RA, Conceicao et al. note that treatments for RA have decreased disease activity scores, hospitalization, and sick leave. Despite this advance in treatment, those with RA still suffer foot and ankle pain even though clinical remission may be reached (Hennessy, 2012). Treatment options for RA also include conservative treatment including the use of analgesics, physical therapy, occupational therapy, access to social workers, patient education regarding lifestyle changes, splinting, alternative therapies, specialty footwear, and foot orthoses for foot pain relief.

The relatively small amount of research about the foot in RA is disproportionate to the contribution of the foot to pain and disability (Baan et al., 2011; Riskowski et al., 2011). Inconsistencies in methodology, orthoses terminology, orthoses fabrication techniques, trial quality, trial sizes, and outcome measures have made evaluating the available evidence and applying to clinical practice challenging (Clark et al., 2006; Riskowski et al., 2011; Hennessy et al., 2012; Conceicao et al., 2015). In a review of studies of foot orthoses in the RA foot, Clark et al. (2006) state that there is strong evidence that foot orthoses do reduce foot pain and improve functional ability, despite the range of types of orthoses being assessed in the studies examined. Conceicao et al. (2015) also found that orthoses significantly improved pain compared with controls, but their effect on function was more limited. However, a Cochrane review of research on custom foot orthoses for RA in the foot only awards foot orthoses a “silver level” of evidence for their use, finding results of clinical trials not strong enough to develop guidelines (Hawke, Burns, Radford, & du Toit, 2008; Riskowski et al., 2011). Regardless, repeated findings and clinical observations in these studies may be helpful in making treatment decisions regarding foot orthoses and footwear to treat foot pain in RA.

The purpose of providing orthosis is to relieve pain, maintain or improve function and mobility, reduce plantar pressures, prevent or minimize deformity, and reduce ulceration risk (Magalhaes et al., 2006; Williams et al., 2011; Woodburn & Helliwell, 1997, Hennessy, 2012). Several studies found that stabilizing deformities and providing pain relief increased walking tolerance and improved gait measures (Chalmers, Busby, Goyert, Porter, & Schulzer, 2000; Cho et al., 2009; Woodburn et al., 2003); however, the mechanism by which orthoses and footwear achieved these goals is not clear. While it is commonly believed that orthoses have an effect on kinematics and mechanical alignment, these effects may be small, and may not be the primary influence of orthoses. Other research provides alternative or perhaps complementary explanations for the efficacy of foot orthoses and specialized footwear. Orthoses may change muscle activation patterns in the foot and leg (Nurse, Hulliger, Wakeling, Nigg, & Stefanyshyn, 2005; Nurse & Nigg, 2001) as well as gait pattern changes which may influence joint loading, reducing stresses to the joints (Woodburn et al., 2002). This may explain why orthoses of various densities, designs, and materials have shown positive results in a wide range of studies. There may be a variety of influences at play to achieve improved results (Riskowski et al., 2011). In a review, Egan supported the current practice of recommending orthoses recognizing their potential to provide pain relief at a relatively low cost (Egan, 2010).

Both rigid (carbon graphite) and semi-rigid
Foot orthoses have also been shown to reduce pain and improve function in adults with RA (Chalmers et al., 2000; Cho et al., 2009; Woodburn et al., 2002) and in children with JIA (Powell, Seid, & Szer, 2005). In early disease, patients responded well to harder orthoses, showing a reduction in metatarsal and rearfoot pain; however, Woodburn et al. (2002) cautioned that rigid devices should only be used in patients where the tarsal joints are not affected by disease. Soft orthoses (Plastazote and polyurethane foam) did not produce as positive a reduction in forefoot pain as semi–rigid or EVA orthoses with metatarsal pads (Chalmers et al., 2000; Hodge et al., 1999). Magalhaes et al. utilized EVA orthoses and found the orthoses effective in reducing pain, perceived disability and activity limitation. Orthoses with medial arch support and metatarsal arch support were found to decrease pain during walking and at rest in RA patients (Moreira, Jones, Oliveira, Jennings, Fernandes, & Natour, 2016). Foot type may also play a role in the selection of orthotic materials. Lower arched or pronated feet generally respond well to harder materials whereas rigid deformity and pes cavus foot types do better with softer materials (Riskowski et al., 2011). As Chalmers et al. (2000) found, too soft a material does not produce a therapeutic effect.

Wearing time appears to have a dramatic effect on the efficacy of custom foot orthoses with increased wearing time resulting in decreased pain, disability, and deformity (Clark et al., 2006; Moreira et al., 2016). Other authors support the continuous use of custom foot orthoses to reduce pain and improve function (Woodburn et al., 2002; Clark et al., 2006), one suggesting an average wearing time of six hours per day for optimal treatment effect. Williams et al. (2011) found that patients who wore their prescribed orthoses and footwear for longer periods of time had better pain reduction than those who did not wear them as often.

Foot orthoses have also been found to reduce plantar pressures under the feet of RA patients. Li (2007) found that orthoses provided greater pressure and loading stress relief in RA patients than in control subjects with no foot problems. If calluses are removed from the plantar aspect of the feet, then pressure–relieving orthoses should be provided to prevent ulceration in at risk feet (Williams, 2011). Surprisingly, Hodge et al. (1999), using in–shoe pressure measurement, found no relationship between foot pain and plantar peak pressure; however, a relationship was found between average pressure and foot pain. They postulate that pain perception in RA is not well understood, and that while peak pressures may be an important variable in the insensate foot when evaluating risk of ulceration, average pressure appeared better correlated to pain in the RA foot. In–shoe pressure–measuring systems can provide vital information when working with patients at high risk of ulceration, but according to Hodge et al. (1999), those using in–shoe pressure systems as a clinical tool should look to average pressure as the more important variable in the management of foot pain in RA.

Several other authors found the combination of orthoses and specific footwear to be the most successful at alleviating foot pain (Cho et al., 2009; Li, 2007; Woodburn et al., 2002, Cham, Ghasemi, Forogh, Sanjari, Yeganeh, & Eshraghi, 2014). Orthoses and footwear cannot be easily separated in the treatment of the rheumatoid foot. These devices should work together to provide a more appropriate therapeutic environment for the RA foot. Despite the need for orthoses and footwear in the RA population, Otter et al. (2010) found poor adherence with prescribed orthoses and footwear, with footwear having poorer adherence than orthoses (de Boer, Peeters, Ronday, Mertens, Huizinga, & Vliet Vlieland, 2009). Reasons for non–adherence to footwear include poor patient education, poor fit of orthoses in shoes, poor fit of footwear, aesthetics, design, comfort, weight, and poor efficacy (Williams & Nester, 2006; de Boer et al., 2009; Graham & Williams, 2016).

The definitions of orthopaedic and therapeutic footwear in the literature varies but most often include footwear features such as extra depth, increased volume in the toe box, heat mouldable uppers, and rearfoot stability. Foot deformity and pain obviously have a negative impact on finding appropriate footwear to accommodate the structural changes and pain relief demands. Difficulty in finding footwear can lead to prolonged use of inappropriate footwear which can be a major contributing factor to foot impairment (Silvester, Williams, Dalbeth, & Rome, 2010). Patient involvement in the process of selecting and designing orthotic and footwear choices may improve adherence to treatment. Patients who perceive the footwear to “look comfortable” are more likely to wear the footwear chosen (Williams & Nester, 2006). Overall mobility needs to be considered when selecting footwear. Limited hand function may restrict closure choices for footwear, eliminating laces as an option, for example. Ease of donning and doffing footwear is important in those with spinal or hip involvement where impaired flexion at the hip and back makes reaching the feet impossible.
Footwear may need to be modified with Velcro closures, or alternative closures to accommodate the needs of the individual and improve compliance.

In a study comparing running shoes to orthopaedic specialty footwear, using a single blind comparison design, running shoes were the preferred footwear for comfort and weight (Hennessy, Burns, & Penkala, 2007). Another footwear condition that also showed a decrease in forefoot loading was custom orthosis in combination footwear that had a forefoot rocker (Cho et al., 2009). Shoes with a forefoot rocker have been shown to reduce plantar pressures in the forefoot independent of orthoses as well (Brown, Wersch, Harris, Klein, & Janisse, 2004; Cham et al., 2014) but studies repeatedly show better results with a combination of orthoses and footwear with forefoot rockers. One of the biggest barriers to compliance for using rocker soles is cosmetics. Patients do not want to “look different” and would prefer to wear shoes they feel are more fashionable, or more acceptable to them. There needs to be more care taken on the part of shoe manufacturers and those in the orthopaedic appliances industry to better design footwear that meets the needs of rheumatoid arthritis patients, improving the look of footwear that also meets the biomechanical and comfort needs of patients.

There are several footwear modifications that are commonly used to treat painful RA feet. To decrease pressure on the forefoot, rocker soles have proven to be beneficial (Cham et al., 2014), provided balance and gait parameters permit the use of rocker soles. In-shoe accommodations can be used to relieve pressure on specific painful metatarsal heads, by physically removing material from the inside of the footwear directly under the affected area. Modifications to the shape of the shoe may be necessary in order to accommodate foot deformity such as a splayed forefoot with a narrow heel. The sole can be split and widened in the area that impinges on the foot and the shoe can then be re-soled to secure the modification and seal the bottom of the shoe. In some cases of forefoot pain, where extra-depth footwear was not suitable, heat mouldable footwear has been shown to reduce forefoot pain (Riskowski et al., 2011). Due to the extent of deformity of the foot in some cases, custom orthopaedic footwear is the best treatment option.

The provision of specialized footwear and orthoses has larger implications than simply accommodating the feet; it also influences issues such as body image, moods, a sense of well-being, and quality of life (Otter et al., 2010; Williams & Nester, 2006; Wilson, 2016). The Canadian Certified Pedorthist is uniquely positioned to provide orthotic and footwear treatment for the RA foot and participate as part of the multidisciplinary team treating the patient. The ability to design and dispense patient specific devices, and to adjust and modify those devices in a timely manner, is a powerful tool in treating the painful RA foot.

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Osteoarthritis is a chronic disease that affects many people and over 60% of people with osteoarthritis are limited in their activities. Foot and ankle osteoarthritis can affect normal range of motion in the specific joint, which can then lead to mild or severe discomfort of that joint. A physical examination and a roentgenogram of the foot and ankle joint are commonly used to diagnose osteoarthritis. Education is key in osteoarthritic treatment; early education may help to limit or delay the onset of OA while continuing education is paramount for self-management after diagnosis. Treat osteoarthritis patients with conservative and pharmacological management first and only pursue surgical treatments under the guidance of an appropriate healthcare professional, as it may lead to future complications.

Key Messages

- Osteoarthritis is a chronic disease that affects many people and over 60% of people with osteoarthritis are limited in their activities.
- Foot and ankle osteoarthritis can affect normal range of motion in the specific joint, which can then lead to mild or severe discomfort of that joint.
- A physical examination and a roentgenogram of the foot and ankle joint are commonly used to diagnose osteoarthritis.
- Education is key in osteoarthritic treatment; early education may help to limit or delay the onset of OA while continuing education is paramount for self-management after diagnosis.
- Treat osteoarthritis patients with conservative and pharmacological management first and only pursue surgical treatments under the guidance of an appropriate healthcare professional, as it may lead to future complications.

Keywords

ankle, cartilage, compression, crepitus, degenerative, friction, joint tenderness/pain, metatarsophalangeal, midtarsal, osteophytes, subtalar

Introduction

Osteoarthritis, also commonly referred to as degenerative arthritis or degenerative joint disease, is a disease that results from the breakdown and loss of cartilage in joints. Cartilage is an elastic connective tissue which helps provide shock absorption and lubrication, covering the surfaces of articular joints and allowing the ends of bones to slide over one another smoothly, helping to ensure proper joint motion. Biological or mechanical factors can impair proper maintenance of healthy cartilage, leading to arthritis. Over time, cartilage may be lost, causing the bones in the joint to rub together; osteophytes may form in some cases, further increasing friction and causing pain.

Osteoarthritis affects as many people as all of the other types of arthritis combined. Although age is not a cause of osteoarthritis, the prevalence of osteoarthritis increases with age. Osteoarthritis is the most common joint disorder in the US, affecting an estimated 12% of U.S. adults aged 25 to 74 years (Cisternas, Murphy, Sacks, Solomon, Pasta, & Helmick, 2016). It is estimated that worldwide, 9.6% of men and 18.0% of women over the age of 60 suffer with osteoarthritis (OA) (Maiese 2016). The prevalence of symptomatic osteoarthritis in those aged over 60 years is predicted to be 30% by the year 2030 (Cooper, Adachi, Bardin, Berenbaum, Flaminon, Jonsson, Kanis, Pelousse, Lems, Pelletier, Martel-Pelletier, Reiter, Reginster, Rizzoli, & Bruyère, 2013).

In the joints of the foot and ankle, osteoarthritis affects range of motion and can cause mild to severe discomfort. At the ankle joint, it mainly affects the foot’s ability to plantar flex and dorsiflex, and can cause significant discomfort and compensated gait. As the subtalar joint is responsible for pronation and supination, allowing the foot to adapt to uneven ground conditions, osteoarthritis here can cause great instability and pain due to limited inversion and eversion. Because motion in the midtarsal joints is very limited even in normal individuals, osteoarthritis across these joints is less common. In cases where it does occur, there is a possibility of some minor aching, but little or no loss of function. In the metatarsophalangeal (MTP) joints, osteoarthritis usually only involves the first MTP joint. Although
this can be an uncomfortable situation, osteoarthritis at the MTP joint does not affect normal ambulation as much as osteoarthritis at more proximal locations of the foot (Moskowitz, 1984).

**Pathology**

Osteoarthritis can affect one side of the body or both, and can be classed into two categories: primary and secondary. Primary osteoarthritis is a result of hereditary factors or stresses on weight-bearing joints. Secondary osteoarthritis follows joint surgery, trauma, or repeated injury (Meszaros, 2006).

Generally speaking, osteoarthritis is characterized by subchondral bone sclerosis, osteophyte formation, progressive loss of articular cartilage, changes in the synovial membrane, increased volume of synovial fluid with reduced viscosity, and, hence, changed lubrication properties (Mei-Dan, Kish, Shabat, Masarawa, Shteren, Mann, & Nyska, 2010). As the disorder progresses, pain can result from deformation of the bones and fluid accumulation in the joints.

**Ankle**

Osteoarthritis in the ankle is characterized by pain localized in the area of the joint. The progression of pain depends on the level of deterioration as the patient continues about normal day to day activities. As the joint deteriorates, the foot’s ability to plantar flex and dorsiflex is decreased. As dorsiflexion is usually more affected by osteoarthritis, the patient may externally rotate the affected foot as a compensatory relief of the dorsiflexion stress on the ankle joint (Moskowitz et al., 1984).

**Subtalar**

Patients with osteoarthritis in the subtalar joint will usually complain of pain in the hindfoot area, made worse by walking on irregular or uneven surfaces as limited inversion and eversion places additional pressure on the joint. Patients will also often report a loss of agility (Moskowitz et al., 1984).

**Midtarsal**

Because motion within these joints is minimal under normal conditions, osteoarthritis is less commonly found in this area. Patients with midtarsal OA may experience mild aching in the middle area of the foot, with little or no loss in normal function (Moskowitz et al., 1984).

**Metatarsophalangeal**

A patient with MTP joint OA usually reports an aching feeling at the joint, which may accompany synovitis and warmth in the area. The joint gradually enlarges, which can make footwear increasingly uncomfortable. This leads to a decreased range of motion, especially in dorsiflexion; patients often externally rotate the foot to toe-off medially or internally rotate to toe-off laterally. These rotations avoid pain by forward motion not having to directly pass over the affected joint (Moskowitz et al., 1984).

Osteoarthritic pain is often relieved by rest and made worse by movement or weight-bearing. In early osteoarthritis, pain is minor and may take the form of mild stiffness in the morning. Inflammation often develops in the later stages. The patient may experience pain even when the joint is not being used and may suffer permanent loss of normal range of motion in that joint. In addition to the physical symptoms, osteoarthritis also causes stress, depression, anger, feelings of helplessness, and anxiety. Those affected may also make changes in lifestyle due to increased pain, which may adversely affect their ability to work and earn an income as well as decreasing community involvement. The financial burden of health care and days lost from work can seriously impact the financial well-being of persons with arthritis and their families (Sniezek, Brady, & Marks, 2002).

**Differential Diagnosis**

Differential diagnoses may include pseudogout (calcium pyrophosphate deposition) (Walker, 2010). Subtalar Joint OA may be misdiagnosed as plantar fasciitis or tendinitis of any of the surrounding muscle complexes. Talonavicular joint OA may present with heat, swelling or, pain that may be mistaken for posterior tibial tendon rupture as this tendon has attachments to the navicular bone (Moskowitz et al., 1984). First MTP joint arthritis may be mistaken for gout, sesamoiditis, or tendinitis of any surrounding muscle complexes about this joint.

**Common Testing**

A thorough history and physical examination as well as radiologic testing is involved in confirming osteoarthritis. The patient’s main complaint is in the location of the arthritic joint, which is aggravated by activity and usually improves with rest. Commonly, the pain is worse when the person first arises after rest and moves around on the extremity. Pain may recur at the end of the day or when there are increased demands on the affected joints. As the condition progresses, the affected joint may show deformity.
(Moskowitz et al., 1984). Radiographs of the affected joints may show joint space narrowing, subchondral sclerosis, and subchondral cysts (Walker, 2010).

The ankle joint
Physical examination will often show a decrease in ankle dorsiflexion and may cause the affected foot to be more externally rotated. Generalized synovial thickening and increased warmth about the joint may occur. Confirm diagnosis by obtaining a roentgenogram of the ankle joint, or a bone scan if there are no roentgenographic changes but pain persists (Moskowitz et al., 1984).

The subtalar joint
Physical findings will likely show restricted motion in the subtalar joint (pronation, supination), at times associated with crepitus. The motion is usually associated with pain. Occasional spasm of the peroneal tendons can occur. Diagnosis is confirmed by a roentgenogram of the subtalar joint region; if a roentgenogram is negative, a bone scan may be used (Moskowitz et al., 1984).

Talonavicular joint
Physical examination will reveal localized pain, swelling, and possibly increased heat over the area of the talonavicular joint. When patient stands, there may be a sag at the talonavicular joint and occasionally a large medial prominence. Diagnosis is confirmed by roentgenographic examination of the joint (Moskowitz et al., 1984).

Tarsometatarsal joints
Physical examination may demonstrate tenderness about the involved metatarsocuneiform joints. A progressive abduction deformity becomes quite rigid and cannot be passively corrected and the deformity is exaggerated upon standing (Moskowitz et al., 1984).

Metatarsophalangeal joints
Physical examination may demonstrate a general thickening of the MTP joint that is not infrequently associated with dorsal spurring. Occasionally a small ulceration can be seen over the area of the spur owing to tight shoes. There is a reduced range of dorsiflexion when compared to a normal foot. Forcing dorsiflexion past the point that the patient can passively achieve usually causes discomfort, particularly with walking. The roentgenographic findings consist of narrowing of the joint space associated with proliferative new bone and varying degrees of dorsal breaking (Moskowitz et al., 1984).

Contraindications
There are no referenced contraindications; however, contraindications can also be seen as the opposite of treatment. Sometimes a treatment for foot arthritis is a rigid–sole shoe with a forefoot rocker; in this case, a contraindication would be the exact opposite—a soft flexible sole with no rocker. These surfaces will increase instability and movement within the foot, increasing frictional forces between affected joints. A foot with an everted hindfoot position as a result of a traumatic injury, muscle tightness, or severe foot deformity would not do well with a deep rigid heel cup to correct the everted position. A more accommodative design of orthoses would be better suited for this situation. OA in the first metatarsophalangeal joint may result in hallux rigidus or hallux valgus. This may cause pain on ambulation or difficulty in finding appropriate well-fitting footwear (Walker, 2011). This type of patient should not be in a shoe with a narrow and shallow toe box, or one that does not have a forefoot rocker.

The Patient Perspective
Osteoarthritis pain varies for each person. There are several ways in which a patient might describe what he/she is feeling, including achy and/or sharp pain; mild or intense joint pain and stiffness; grinding; very debilitating joint stiffness upon waking up in the morning; or symptoms that are different every day. What a patient describes depends on the extent of the joint damage (Eustice, 2010). Pain associated with OA is typically described as being dull, aching, or throbbing, and localized to a specific region which is generally more severe with use of the joint and toward the end of the day, but better with rest (Walker, 2011).

Common Treatment
The goals of OA management are to reduce pain and stiffness, and to maintain or improve joint mobility where possible (Walker, 2011). Management for OA of the foot and ankle may include conservative and nonpharmacological management as well as invasive and pharmacological management. It is important to consider each OA patient on an individual level, and management strategies should be considered in relation to the context of quality of life, functional limitations, and pain experienced (Walker, 2011). Regular osteoarthritis exercises, weight control, proper nutrition, and a healthy diet are all part of a comprehensive treatment plan for osteoarthritis. Patients should always discuss diet, weight loss, and
osteoarthritis exercises plan with their doctors (Das, 2011).

**Non-pharmacological management**

Patients with osteoarthritis in the ankle, subtalar joint, tarsal joints, and metatarsophalangeal joints will have some overlap when it comes to treatment interventions.

The use of aids such as foot orthoses, insoles, special/appropriate footwear, footwear modifications, AFO, ankle braces, gait plates, and canes can assist OA patients who have biomechanical joint pain or instability (Walker, 2011). If joint pain resulting from poor or inefficient foot biomechanics contributes to OA symptoms, then the proper use of foot orthoses and footwear can cause changes in kinematic and skeletal alignment, and may alter muscle activation and gait patterns to reduce joint loading (Riskowski, Dufour, & Hannan, 2011). The use of a cane reduces the force through the affected joint, improving pain, function, stride length, and cadence (Walker, 2011).

If the patient has rather marked limitation of ankle dorsiflexion, a slight elevation of the heel may be useful (Moskowitz et al., 1984). This can be done either by placing a felt pad within the shoe itself or by adding an external elevation to the heel. The use of a soft sole material in the heel, e.g., a SACH heel, is helpful because it decreases the impact on initial ground contact.

In order to further increase the stability of the ankle joint, a polypropylene ankle-foot orthosis (AFO) can be used to further restrict the ankle joint motion (Moskowitz et al., 1984). When such an orthosis is used, the patient should wear a rocker bottom type shoe in order to enable him to smoothly roll over his foot. The use of extra-depth orthopaedic shoes can also help improve ankle stability (Riskowski et al., 2011).

A rigid ankle brace may be useful in reducing movements at the subtalar joint in subtalar OA patients. They may also benefit from footwear with cushioned soles, firm heel counters, and hindfoot rockers. In cases of midfoot and forefoot OA, footwear with a rockered forefoot and a cushioned sole can aid in reducing stress in the affected joints.

Patients often note that they can walk fairly comfortably on level ground, but any type of uneven surface will cause a significant amount of discomfort (Moskowitz et al., 1984). Therefore, the use of a wide-heeled shoe will increase the stability of the subtalar joint by increasing the surface area making contact with the ground, but a moulded leather ankle brace with medial and lateral stays or a polypropylene AFO with a rocker bottom shoe may eventually be necessary (Moskowitz, 1984).

Midfoot OA, according to Riskowski et al. (2011), may be a result of abnormally high mechanical stress. Noninvasive treatment with a full-length orthosis will decrease pain and increase foot function by reducing both the magnitude and duration of medial midfoot loading. Rockerbottom footwear may also increase the mechanical advantage of the great toe and reduce the stress at the medial midfoot. In a study done by Rao, Baumhauer, tome, & Nacwaczenski (2008), using full-length orthoses over a four-week period had provided symptomatic relief to subjects with midfoot OA.

Footwear with cushioned soles and increased padding of the tongue area may reduce the external pressures on the midfoot in certain instances.

In cases where there is excessive motion in the foot and ankle, a semirigid functional foot orthosis may help with reducing strain in the arthritic joints. A foot with a rigid structure, however, may benefit from a more accommodative orthosis to help provide cushioning and distribute pressure.

The talonavicular joint and calcaneocuboid joint will benefit from the same conservative treatment as that mentioned above for ankle and subtalar joints; however, the use of “a UCBL type of insert, particularly if there is progressive collapse in the medial longitudinal arch” (Moskowitz et al., 1984, p.396) might be necessary.

OA of the tarsometatarsal joint usually occurs in the first metatarsocuneiform joint, and is associated with a progressive deformity of the midfoot region (Moskowitz et al., 1984). Conservative treatment generally involves the use of an arch support and rockerbottom shoes to decrease stress in the affected area (Moskowitz et al., 1984).

According to Moskowitz et al. (1984), OA of the MTP joints usually only involves the first metatarsophalangeal joint. A specific “prescription of a shoe that has adequate toe box to prevent excessive pressure is helpful” (Moskowitz et al., 1982, p.400) in reducing external pressure from footwear and “a shoe with a stiff sole or rocker bottom will help diminish motion at the metatarsophalangeal joint and decrease joint pain” (Moskowitz et al., 1982, p.400). According to Welsh, Redmond, Chockalingam, & Keenan (2010), a commonly used orthoses design can
offer a reduction in mechanically induced pain at the first MTP joint. In cases where dorsiflexion of the first MTP joint needs to be reduced, an orthosis design with either a rigid Morton’s or a gait plate may be used.

Other ways to manage pain from OA include the use of heat, cold, and transcutaneous electrical nerve stimulation (TENS) . Heat application positively affects pain and function (Walker, 2011). Local heat and cold applications are widely used by people with OA as a part of self-management. The use of TENS may also be considered as a valuable option to provide symptomatic relief.

Exercise should be considered a core treatment of OA, as it may reduce pain and improve function as long as the magnitude of weight loss is sufficient (Walker, 2011).

There are three main types of physiotherapy—osteoarthritis exercises:

1. Range of motion: To maintain normal joint movement and relieve stiffness, making the joints more flexible.

2. Strengthening exercises: To increase strength in the muscles that support the joints affected by arthritis.

3. Aerobic or endurance exercises: These improve cardiovascular fitness, control weight, and improve overall body function (Das, 2011).

Pharmacological and invasive management

An oral drug such as paracetamol is the first drug of choice for mild OA (Walker, 2011); however, additional nonsteroidal anti-inflammatory drugs (NSAIDs) may help. Topical NSAIDs can also be used, and are associated with fewer adverse effects than oral NSAIDs.

Glucosamine and chondroitin products, as well as the use of vitamins A, C, or E, ginger, or turmeric, are not recommended as there is insufficient evidence for their efficacy (Walker, 2011).

Corticosteroid injections can be used to provide short-term pain relief to the affected joint (Walker, 2011).

Treatment of hallux limitus can involve conservative measures, and/or surgical interventions (Munteanu, Menz, Zammit, Landorf, Handley, Elzarka, & DeLuca, 2009). Pharmacological treatment is also often undertaken for pain relief; however, since gastrointestinal complications remain as a concern here, an alternative treatment termed “viscosupplementation”—the intraarticular injection of hyaluronan into arthritic joints with the aim of restoring the viscoelasticity of the synovial fluid—has been proposed as a treatment for OA.

Surgical management

If conservative and pharmacological management fails to provide adequate levels of symptom relief, then surgical intervention may be considered.

If OA becomes severe with progressive limitation in activities of daily living, restriction of normal functioning due to pain, or effects on health related to quality of life, then the opinion of an orthopaedic surgeon should be considered (Walker, 2011).

Arthroscopic lavage and debridement are not routinely performed as a treatment for OA unless there is evidence to support their use. Osteotomy may relieve pain and delay the need for joint replacement by reducing intraosseous pressure, which is great for young and active candidates. Arthroplasty effectively relieves pain and improves functional ability for patients with OA.

“The surgical management of osteoarthritis of the ankle consists of performing an arthrodesis of the ankle joint. Following a successful ankle arthrodesis, most patients can walk with a mild gait abnormality with little or no pain” (Moskowitz et al., 1984); however, “most total ankle joint replacements that are currently in use do not provide adequate pain relief, significant improvement in motion, or long-term reliability” (Moskowitz et al., 1984). The other issue with ankle arthrodesis is that it alters the biomechanics of foot joints and may lead to arthritis in neighbouring joints (Rouhani, Crevoisier, Favre, & Aminian, 2011). Performing a total ankle replacement causes less alteration of the foot and ankle’s biomechanics, but involves surgical complexities (Rouhani et al., 2010).

When considering surgical treatment for the subtalar joint, “a subtalar arthrodesis can be performed and will usually result in satisfactory relief of symptoms” (Moskowitz et al., 1984. p.395).

Surgical treatments for the tarsal joints all involve arthrodesis of the involved joint articulation, which would include the talonavicular articulation and, in rare and unusual cases, the calcaneocuboid articulation and midtarsal joint (Moskowitz et al., 1984).

Osteoarthritis of the tarsometatarsal joint may require surgical intervention if deformity progresses.
Surgery consists of stabilization of the involved tarsometatarsal joint, followed by the joints in sequence laterally across the foot (Moskowitz et al., 1984).

If pain persists after conservative management of osteoarthritis in the metatarsophalangeal joint, then surgical procedures may be needed. According to Moskowitz et al. (1984), there are four basic procedures. The first is a cheilectomy, which consists of removing the excess bone from around the metatarsal head. If the cheilectomy fails, a different surgical procedure can be performed. A second procedure is arthrodasis of the first metatarsophalangeal joint, which will ensure a painless joint, but with loss of joint motion. This is often the treatment of choice if the patient is involved in heavy manual labour. The third option is placing a Silastic spacer into the base of the proximal phalanx as well as performing a cheilectomy of the metatarsal head in order to permit adequate dorsiflexion, although there are problems associated with the prosthesis, such as a certain degree of soft tissue reaction to foreign material. A fourth surgery is the Keller procedure or excision of the proximal third of the proximal phalanx. This procedure is for the non-demanding, housebound patient, because it does not relieve the pain associated with the arthritic joint.

Adequate support, exercise information, and appropriate analgesia control are the keys for patients to retain their independence and be able to manage daily activities without repeated consultations with health professionals (Walker, 2011).

Key Considerations

There are many key considerations when working with osteoarthritis patients, and what works for one patient won’t necessarily work for another. According to Walker (2011), psychological status can affect a patient’s experience of pain, and consequently the ability to develop effective coping strategies. Patients with high levels of anxiety are more likely to request higher doses of analgesics and be more focused on their pain. Patients need to learn to pace themselves during activities, and to integrate periods of rest into specific exercises/activities.

Age is a key consideration for patients with osteoarthritis. OA occurs in both men and women and usually develops after age 45 (eHealth MD, 2011a).

Emotional and repeated daily stresses may increase joint discomfort and cause arthritic symptoms to worsen (eHealth MD, 2011b).

Weather and climate can cause temporary effects on symptoms but do not actually affect the disease. This means that climate does not improve or worsen arthritis, although it may alter symptoms (eHealth MD, 2011b).

Weight control is important in successful management. Maintaining an appropriate weight can lessen pain by reducing stress on the weight-bearing joints (hips, knees, back, and feet), increase self-esteem, and avoid the risk of psychological suffering and/or depression that can affect overweight individuals (eHealth MD, 2011b).

Foot and ankle biomechanics must always be considered. The type of orthosis relative to foot structure is important (Riskowski et al., 2011). In general, rigid orthoses are used to control excessive motion, reduce forefoot loading, and decrease forefoot and rearfoot pain. Conversely, soft orthoses are typically used in conjunction with rigid foot ailments, such as pes cavus, and reduce both forefoot and generalized foot pain.

Appropriate referral to other members of the multidisciplinary team should be considered to maximize optimal function (Walker, 2011). Referral to an orthopaedic surgeon can be of great benefit for patients with severe osteoarthritis. Shared decision-making and open communication are important for effective management of osteoarthritis.

References


Osteoarthritis – Knee

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Key Messages

• Osteoarthritis of the knee is characterized by degradation of cartilage.
• Typically worse in one compartment of the knee.
• Treat by unloading the affected compartment.

Keywords
cartilage, compression, crepitus, degenerative, friction, joint tenderness/pain, knee, osteophytes, patellofemoral

Introduction and Pathology

Osteoarthritis (OA) is a degenerative disease that affects the joint cartilage, synovium, joint capsule, and subchondral bone (De Almeida Carvalho, Bittar, de Souza Pinto, Ferreira, & Sitta, 2010). With age, wear, or injury, the cartilage of the affected compartment becomes thinned to the point that it no longer allows for smooth, low-friction movement between joint surfaces. Six percent of adults over the age of 30, and 12% of those who are ages 65 and above, are affected by knee OA. In a group of 4,733 adult Canadians, 10% of males and 17% of females had been diagnosed with knee OA (Plotnikoff, Karunamuni, Lytvyn, Penfold, Schopflocher, Imayama, Johnson, & Raine, 2015). Those who are obese are at higher risk than those who are not (Kulie et al., 2011). With continued degeneration, the cartilage can become pitted, torn, or worn away altogether, resulting in bone–on–bone contact. Osteophytes can form at the joint margins, causing further pain and restricting range of motion (Bijlsma, Berenbaum, & Lafeber, 2011). When either of the weight-bearing compartments of the knee is affected more than the other, a change in the frontal plane alignment of the knee can result. The medial compartment is involved about 10 times more often than the lateral compartment. When the medial compartment is involved, medial compartment cartilage degradation ensues and a varus knee alignment is acquired. This varus alignment causes the ground reaction forces of gait to pass through the knee joint more medially, resulting in abnormal compressive and shear forces to the medial knee structures (Raja & Dewan, 2011). This cyclic turn of events causes the condition to progressively worsen. When the lateral compartment is affected, a resultant valgus knee alignment causes ground reaction forces to pass through the lateral aspect of the knee joint, which excessively loads the area (Raja & Dewan, 2011). These frontal plane misalignments are clinically important because they are the basis for several treatment modalities as well as functional testing and diagnosis.

The patellofemoral compartment can also be affected. This compartment is unlike the medial and lateral compartments because it plays no role in weight-bearing. It is symptomatic in 8% of women and 2% of men over the age of 55 (Vaquero, Calvo, Chana, & Perez–Marianes, 2010). OA in this area develops as a result of friction between the posterior surface of the patella and the anterior surface of the femur. Chronic misalignment of this joint will cause increased wear and accelerate the degeneration that leads to osteoarthritis in the area. Prominent genu valgum or genu varum is a risk factor for patellofemoral OA (Vaquero et al., 2010).

Differential Diagnosis

• Iliotibial band friction syndrome (Wittke, 2005)
• Patellar tendinitis (Wittke, 2005)
• Patellofemoral syndrome (Wittke, 2005)
• Patellar plica syndrome (Wittke, 2005)
• Meniscal tears (Dervin, Stiell, Wells, Rody, & Grabowski, 2001)
• Knee rheumatoid arthritis (Jones, Hopkinson, Pattrick, Berman, & Doherty, 1992)
• Popliteal cyst (Calmbach & Hutchens, 2003)
• Pes anserine bursitis (Calmbach & Hutchens, 2003)

Common Testing

When testing for OA of the knee, a few quick checks should be done first. Look for these symptoms:

- Tenderness over the knee
- Creaking and grating (crepitus)
- Bony swelling
- Excess fluid
- Restricted movement
- Instability of the knee
- Thinning of the muscles that support the knee

functional testing

The squat duck walk test can give indications of the intraarticular status in the tibiofemoral joint (Magee, 1997). Ask the patient to squat with heels down, and then heels up. If they are able to do this then ask them to walk forward in this position. Here you are looking for the ability to weight-bear in knee flexion. You should compare affected to non-affected side. Also, in a standing position, check the varus or valgus angle at the knee joint, and compare both legs. Ask the patient to go to a squatting position while palpating the tibiofemoral joint lines, noting any crepitus, tenderness, and range of motion (Magee, 1997).

Assessment and treatment techniques for the lower extremity

When arthritis in the knee begins to present, the patient will begin to lose range of motion, beginning with flexion and followed by extension at the knee (Walsh & Nolan 2001). Many patients will be unable to perform many functional tests, due to pain at the knee, as well as weight issues. If this is the case, diagnostic testing should be done and is normally used for all patients dealing with OA of the knee.

Diagnostic tests

When using diagnostic tests for OA, the most common and useful is an X-ray. X-rays may show changes such as osteophytes, narrowing of the space between bones and calcium deposits within the joint (Arthritis Research UK, n.d.). X-rays aren’t a good indicator of how much pain or disability a patient is likely to have. People can have a lot of pain from minor joint damage but others have little pain from severe damage. An MRI can also be used to show soft tissue damage and changes in the bone that can’t be seen on a standard X-ray (Arthritis Research UK, n.d.). This is quite rare, though. There is no blood test for osteoarthritis but they can be used to rule out other conditions such as rheumatoid arthritis (University of California, 2010).

Osteoarthritis – Knee
To reduce the risk of applying treatments incorrectly, the degree of degradation present in both weight-bearing compartments should be confirmed by a physician before proceeding. The physician may require imaging or other tests to confirm the state of the two compartments.

These contraindications should also be considered for patients who have knee OA as a secondary complaint. Treating other conditions with treatments that will worsen their OA should be avoided if possible.

The Patient Perspective

The following symptoms are common for patients with OA at the knee, yet not all need to be present (University of California, 2010):

- Joint pain
- Joint stiffness
- Joint tenderness
- Limited range of motion
- Crepitus (crackling, grinding noise with movement)
- Joint effusion (swelling)
- Local inflammation
- Bony enlargements and osteophyte formation
- Not getting relief from over-the-counter medications
- Pain changes with weather

Pain is the primary symptom of osteoarthritis and is linked to functional impairment and disability in osteoarthritis patients. Usually osteoarthritis pain develops gradually. With mild to moderate osteoarthritis, pain typically worsens with use of the joint and improves with rest (Arthritis Research UK, n.d.). As the disease progresses, pain is usually more persistent and may not be relieved by rest or basic treatments. As well as a sign of progressed OA, pain at rest can be a sign of local joint inflammation. Morning stiffness is common with osteoarthritis but does not last as long as it does in people with rheumatoid arthritis (Arthritis Research UK, n.d.). Usually morning stiffness in osteoarthritis patients lasts less than 30 minutes. Osteoarthritis is not a systemic disease like rheumatoid arthritis. Symptoms of osteoarthritis are usually localized to the affected joint. Severe cartilage loss in the joint can result in misshapen joints which can cause joint instability. Unstable joints can cause a joint to lock or buckle, especially the knee. Posture, coordination, and ability to walk can be affected by joint pain and stiffness of hips, knees, and spine (University of California, 2010). Non-steroidal anti-inflammatory drugs, including ASA and ibuprofen, often are most effective in the early stages of arthritis. Their effectiveness varies from person to person and may become less effective for patients with severe arthritis. Some people find that changes in the weather (especially damp weather and low pressure) make their pain and stiffness worse. This may be because nerve fibres in the capsule of their knee are sensitive to changes in atmospheric pressure (University of California, 2010).

Common Treatment

Treatments for OA of the knee fall into three broad categories: pharmacological, surgical, and non-surgical physical therapies. The primary focus here is non-surgical physical therapies, though the other treatment modalities will be discussed so that the clinical practitioner has a thorough understanding of the overall treatment process. Treatment modalities will be discussed separately for disease of the weight-bearing (tibiofemoral) compartments and the patellofemoral compartment.

Above all treatments, patient education is essential to successful treatment. Patients who understand their condition will be more likely to comply with treatments of all kinds (Price, 2008). It is the responsibility of each clinician to educate their patients regarding their condition. Patients can also be referred to community organizations such as the Arthritis Society for education as well as social support and other resources.

Weight-bearing compartments

As OA of the knee alters the Q angle and increases loading of the affected compartment, several treatments aim to reduce the angular deformity and reduce the excessive loading. Patients with medial compartment OA have been shown to exhibit a lateral centre of pressure at the feet compared to a healthy control (Lidtke, Muehleman, Kwasny, & Block, 2010). Lateral wedges with or without foot orthoses make use of this by applying valgus stress to the foot and therefore the knee. This treatment has been shown to be effective for all severities, though more pain relief was noted with less severe cases (Krohn, 2005). Addition of custom arch support to a standard lateral wedge insole has shown increased comfort and function and a decrease in pain with pronated feet (Hunt, Krowchuk, Hatfield, Hinman, & Chang, 2017). The treatment is simple, relatively inexpensive, and well tolerated, and is recommended as a first step in treating knee osteoarthritis. Medial wedges can be
used in the same manner for lateral compartment disease.

Another approach to reduce the Q angle is unloading knee bracing. These braces apply frontal plane stress directly to the knee: valgus for medial compartment disease and varus for lateral disease (Schmalz, Knopf, Drewitz, & Blumentritt, 2010). By applying this frontal plane stress, weight-bearing load is shifted from the affected to the unaffected compartment thus reducing pain (Raja & Dewan, 2011). A wide variety of these braces are available in off-the-shelf as well as custom versions.

Reducing total weight being borne by the affected limb can also reduce pain and disability. Weight and fat loss is a very effective way of achieving this (Toda, Toda, Takemura, Wada, Morimoto, & Ogawa, 1998), as is the use of a contralateral cane (American College of Rheumatology Subcommittee on Osteoarthritis Guidelines, 2000).

There are also many treatment modalities that fall outside of the pedorthic scope. Viscosupplementation using a hyaluronic acid–based supplement has been shown to be safe and effective at reducing pain and increasing function (Migliore, Giovannangeli, Granata, & Laganà, 2010). Surgical debridement of loose or uneven cartilage is a common early surgical treatment; however it has been shown to have limited benefit except for specific cases such as meniscal damage (Howell, 2010). A wedge osteotomy, where the angle of the tibial plateau is surgically altered to achieve unloading of the affected compartment, is an effective, more aggressive surgical treatment (Schallberger, Jacobi, Wahl, Maestretti & Jakob, 2011). The most aggressive surgical treatment is a total knee arthroplasty, in which the proximal tibia and distal femur are removed and replaced by prosthesis (Woo, Chung, & Lee, 2010). A physician may also prescribe analgesic and/or anti-inflammatory drugs, both non-steroidal and corticosteroid. Physiotherapeutic treatment, including strengthening of surrounding musculature and inflammatory control, may also form a part of the treatment plan.

**Patellofemoral compartment**

The biomechanical presentation of patellofemoral OA is very similar to patellofemoral pain syndrome, and its treatment is similar. The primary goal of treating OA in this compartment of the knee is to reduce friction force between the patella and the femur. This can be achieved through various treatments. Under the care of a physiotherapist, strengthening, stretching, and balancing of the quadriceps and internal/external hip rotator muscle groups can reduce pain and increase function. For the pedorthist, controlling abnormal foot biomechanics that affect the rotational position of the tibia can reduce friction in this compartment.

External stabilization of the patella by use of patellar stabilizing braces or taping can also provide some relief. For further discussion of treatment for the patellofemoral compartment, see the clinical practice guidelines document for patellofemoral pain syndrome.

**Key Considerations**

- Full length lateral/medial wedges usually provide the best results and are better tolerated compared to heel wedges.
- Using custom arch support with a lateral wedge in pronated feel increases patient comfort and decreased pain levels.
- Semi–rigid (polypropylene/carbon fibre) or accommodative (XPE/PPT/Plastazote) orthoses can be used at the pedorthist’s discretion to stabilize the hindfoot and midfoot, which can help reduce internal rotation of the tibia. This should be done with caution as increasing load on one compartment is needed to unload the other.
- Well–cushioned shoes are helpful in reducing ground shock which can affect the patients discomfort in the affected knee. A shoe with a wide base, stiff heel counter, and rocker sole are preferred as it provides a stable base during midstance while easing the patient’s gait through toe–off.
- Sandals with similar features (as noted above) should be worn indoors or when not wearing shoes. This consistency of support will help lessen the stress on the knee joint while weight–bearing. Since knee OA is seen mostly in older populations, it is a good practice to wear indoor footwear as a significant portion of their daily activities will likely be spent at home or in an indoor environment.

**References**


conditions/osteoarthritis-of-the-knee.aspx


Osteoarthritis – Hip

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Key Messages

- OA of the hip can present as dull or sharp pain in the buttock, groin, thigh, knee, or hip.
- Persons with structural deformities at the femoral head-neck junction are at greater risk of developing hip OA.
- Loading patterns at the foot can have substantial effects on loading at the knee and hip.

Keywords

cartilage, compression, crepitus, degenerative, friction, hip, joint tenderness/pain, osteophytes

Introduction

Osteoarthritis (OA) is a degenerative chronic condition and is the most common form of chronic arthritis (Marieb & Hoehn, 2007). This joint disease mainly affects the cartilage. In a healthy individual, cartilage allows bones to glide over each other and helps with shock absorption. With the wear and tear that this condition causes, more cartilage gets destroyed than is naturally replaced (Marieb & Hoehn, 2007). As the disease progresses, bone tissue will thicken, creating spurs that will reduce the range of motion in the joints (Marieb & Hoehn, 2007). Bits of bone or cartilage can also break off, which can cause more pain and damage as those pieces float inside the joint spaces. Although more women are affected than men, 85% of Americans will develop this condition (Marieb & Hoehn, 2007). The most commonly affected joints are the knees and hips, along with the fingers and the cervical and lumbar spine (Marieb & Hoehn, 2007). By the age of 85, one in four people will develop symptomatic hip OA (Murphy & Hemlock, 2012). Within the knee, the medial compartment is more frequently involved because during midstance of gait, 60–80% of the load is distributed through the medial compartment (Johnson, Leitl, & Waugh, 1980).

Pathology

OA is not exclusively a disorder of articular cartilage (Burr, 2004). Multiple components of the affected joint are adversely affected by OA, including the peri-articular bone, synovial joint lining, and adjacent supporting connective tissue elements (Burr, 2004). The characteristic structural changes in OA include the progressive loss of articular cartilage, increased subchondral plate thickness, formation of new bone at the joint margins (osteophytes), and the development of subchondral bone cysts (Li & Aspden, 1997). In addition, at the junction of the articular hyaline cartilage and adjacent subchondral bone, there is a remnant of calcified cartilage (Lane, Villacin, & Bullough, 1977). As OA progresses, there is evidence of vascular invasion and advancement of this zone of calcified cartilage into the articular cartilage, which further decreases articular cartilage thickness (Lane et al., 1977). These structural alterations in the articular cartilage and periarticular bone are thought to lead to modification of the contours of the adjacent articulating surface (Bullough, 2004). Further, persons with structural abnormalities at the femoral head-neck junction such as certain tilt deformities, femoral anteversion, and other alterations of the femoral neck are at greater risk of developing hip OA (Beck, Kalhor, Leunig, & Ganz, 2005). Bony abnormalities of the acetabulum such as coxa profunda, protrusion, or retroversion are also at a higher risk (Beck et al., 2005). In summary, multiple factors are known to affect the progression of OA such as the presence of polyarticular disease, increasing age, associated intra-articular crystal deposition, obesity, joint instability and/or malalignment, muscle weakness, and peripheral neuropathy (Goldring & Goldring, 2006).

Signs and symptoms

Patients will usually describe hip OA pain as discomfort and stiffness in the hip, buttock, groin, or thigh region first thing in the morning. Also, this pain often worsens with activity and is relieved with rest. Cold weather seems to affect the severity more...
than in summer months. A limp is often noted as the patient tries to transfer body weight during gait with the least amount of pain.

**Differential Diagnosis**

Bursitis and other periarticular disorders of the affected joint as well as referred pain should be considered when diagnosing OA (Felson & Zhang, 1998). OA of the hip can present as a sharp or dull pain in the buttock, groin, thigh, knee, or hip. Therefore the varied nature and site of pain can make early diagnosis difficult (Felson & Zhang, 1998).

**Common Testing**

The hip can be examined reliably for pain on movement (log roll test, pain on flexion, pain on external rotation), range of motion in different directions (internal rotation, flexion, flexion contracture), muscle strength of different muscle groups (abduction, adduction, flexion, extension), and leg length measurements (Cibere et al., 2006). Examinations such as the assessment of gait (walking) and Trendelenburg test for the assessment of hip muscle abduction strength were found to not be reliable despite standardization of techniques (Cibere et al., 2006). Radiographs can provide objective evidence of the disease. Findings consistent with osteoarthritis include the presence of joint space narrowing, osteophyte formation, pseudocyst in subchondral bone, and increased density of subchondral bone (Kraus, 1997). The absence of radiographic changes, however, does not exclude the diagnosis of osteoarthritis. Many patients with radiographic changes consistent with osteoarthritis are asymptomatic or do not exhibit any disability, suggesting that the presence of radiographic changes in the absence of symptoms should not lead to the diagnosis of osteoarthritis (Kraus, 1997).

**Contraindications**

Any activity that leads to increased pain in the affected joint should be avoided. Shoe lift therapy to balance any diagnosed leg length discrepancies should be approached extremely cautiously as further unknown skeletal changes are likely to be present above and/or below the affected joints (Gofton & Trueman, 1971).

**Treatment**

Osteoarthritis progresses very slowly and is irreversible (Marieb & Hoehn, 2007). There is currently no cure for this condition; therefore, the treatment is focused more on reducing pain and improving function (Felson, Lawrence, Hochberg, McAlindon, Dieppe, Minor, Blair, Berman, Fries, Weinberger, Lorig, Jacobs, & Goldberg, 2000). Knowing what osteoarthritis is, how it affects the body/joints, and ways of treating the condition is very valuable information. Patient education on this condition can really help to understand and treat the pain effectively (Brandt, 1998).

A great way to improve and maintain function is to exercise regularly. Physical activity is a key ingredient for managing osteoarthritis and will help strengthen the lower limbs (Zhang, Moskowitz, Nuki, Abramson, Altman, Arden, Bierma-Zeinstra, Brandt, Croft, Doherty, Dougados, Hochberg, Hunter, Kwoh, Lohmander, & Tugwell, 2008). Exercise is also important in losing weight (Brandt, 1998), which can help reduce the load on the affected joints of the lower limbs. Veitienne & Tamulaitiene (2005) explain in a study that exercise, assistive devices (such as canes and walkers), and heat were more effective in reducing osteoarthritic pain. Less effective treatments included splints, resting, joint protection, and cold therapy; however, Brandt (1998) suggested heat and cold can both help with symptoms.

The entire lower extremity acts as a linked kinetic chain; therefore, changes at any site will significantly affect the loading patterns throughout the extremity (Lidtke, Meuhleman, Kwasny, & Block, 2010). One device that can help change these forces is the unloading knee brace. In fact, a 4–6 degree increase in varus alignment can increase medial compartment loading from 70–90% during single limb weight-bearing (Fitzgerald, 2005). The strength of recommendations scored 76%, according to the Osteoarthritis Research Society International’s new guidelines for managing hip and knee OA, in the ability for an unloader brace to reduce pain and improve stability (Zhang et al., 2008). Also, unloader braces have been shown to delay the requirement for surgery (Pollo, Otis, Backus, Warren, & Wickiewicz, 2002).

The ground reaction force vector must go through the foot on its way through the kinetic chain (Lidtke et al., 2010). The loading patterns at the foot can have substantial effects on loading at the knee (Lidtke et al., 2010), therefore foot orthoses and wedges can significantly help in the treatment of knee and hip OA. Insoles or custom foot orthoses can be designed to improve the skeletal alignment and function up the entire kinetic chain. This can be done by controlling...
certain movements such as overpronation and supination or just by increasing shock absorption with better weight distribution and cushioning. Wedging and insoles have been shown to significantly reduce pain, primarily during stair climbing, especially when used in conjunction with cushioned shoes (Fang et al., 2005). Bracing of an OA hip is a treatment that is recommended for mild to moderate hip OA and as an adjunct treatment to a core treatment plan which is stated previously. The goal of such bracing is to optimize the rotational movement of the femur. The straps are constructed in such a way that an external rotation and an abduction force is placed on the hip which can reduce the load on the femoral head (Nerot & Nicholls, 2016).

Some patients will control their pain level with certain anti-inflammatory and pain medications or supplements. ASA, acetaminophen, and moderate activity can help keep the joints mobile, while glucosamine and chondroitin sulphate appear to reduce pain and inflammation in some people with OA (Marieb & Hoehn, 2007).

**Key Considerations**

Although there is no specific cause of osteoarthritis, it is known that malaligned or overworked joints are likely to develop OA (Marieb & Hoehn, 2007). With that in mind, consideration must be given to different factors that can play a part in the progression of OA including, age, sex, activity level, weight, previous injuries, etc. The number of adults with OA is increased in correlation with increased obesity rates (Heller, Taylor, Perka, & Duda, 2003). The percentage of people suffering from the disorder seems to increase in age (Dillon, Rasch, Gu, & Hirsch, 2006).

**References**


Fibromyalgia

Jacquie Snider-Nasseri, BSc (Kin), C. Ped (C)

Key Messages

• Chronic, widespread pain due to a disturbance in pain processing in the central nervous system (CNS).
• Symptoms include fatigue, lack of restorative sleep, sensitivity to light/touch/sound, irritable bowel, neurological symptoms (numbness, tingling, burning), depression, and social isolation.
• Treatment will vary based on the presentation of the syndrome and should have a multi-faceted approach and not just symptom control.
• Fibromyalgia is no longer diagnosed by exclusion.

Keywords

allodynia (pain experienced from a non–painful stimulation), hyperalgesia (increased sensitivity to pain), sensory amplification, tender joints, widespread pain

Introduction

Fibromyalgia is a common syndrome in which people experience long-term, body-wide pain. In addition to pain, people also commonly report other symptoms such as sleep disturbances (especially a lack of restorative sleep), fatigue, stiffness, sensitivity to touch/light/sound, cognitive disturbances, concentration difficulties, headaches/migraines, irritable bowel or bladder, as well as neurological symptoms including numbness, tingling, burning, and paresthesia. Fibromyalgia is the second most common rheumatologic disorder after osteoarthritis and affects up to 6% of the world population, with a higher prevalence among women (Starkweather & Menzies, 2016). The cause of this syndrome is currently the topic of much research as its high prevalence, number of co-morbidities, degree of disability, and global severity create a significant burden on health care around the world (Imamura, Cassius, & Fregni, 2009). Risk factors associated with fibromyalgia include genetics, environmental triggers, and gender. First-degree relatives of people with fibromyalgia are at an 8.5-fold increased risk of developing fibromyalgia (Arnold, L., Clauw, D., & McCarberg, B., 2011). Finally, women are diagnosed with fibromyalgia seven times more often than men (Arnold et al., 2011).

Pathology

Although the full pathophysiology remains unknown, there is increasing evidence that peripheral and central sensitization at the spinal cord, brain stem, and cortical levels result in an amplification of sensory impulses that may alter pain perception (Imamura, M., Cassius, D., & Fregni, F., 2010).

One study suggested that there is a peripheral neuropathic component of fibromyalgia, augmented ascending pain-facilitating pathways and impaired descending pain inhibitory pathways. Aberrant neurochemical processing of sensory signals in the CNS may lower the threshold of pain, amplify normal sensory signals (allodynia), and alter gene expression, thereby leading to hypersensitivity (hyperalgesia) and central sensitization that result in chronic pain (Arnold et al., 2016). Due to the peripheral and CNS sensitization, continuous pain in individuals with fibromyalgia leads to additional neoplastic changes that can sustain pain, and therefore maintain a continuous cycle that is responsible for the chronic and refractory condition of this syndrome (Imamura et al., 2010).
Changes in the CNS that lead to fibromyalgia also likely contribute to multiple associated symptoms, including sleep disturbance, fatigue, cognitive symptoms, and mood disorders (Starkweather & Menzies, 2016). The most common sleep disturbance that occurs is a lack of restorative sleep, or stage 4 abnormality in which the individual has bursts of awake-like brain activity which decreases the amount of deep sleep (NFA, 2017). The fatigue experienced by individuals with fibromyalgia is an all-encompassing exhaustion, much more than typical tiredness at the end of a busy day (NFA, 2017). Commonly experienced cognitive symptoms include difficulty focusing and poor working memory (NFMCPA, 2017). And finally, mood disorders such as depression are often associated with fibromyalgia and affect the individual’s quality of life. When an individual’s symptoms are discredited, a phenomenon known as invalidation often occurs, where the individual has higher levels of distress, frustration, and dissatisfaction, which further diminishes physical and social functioning (Starkweather & Menzies, 2016).

Further research is ongoing to understand the evolution of abnormal pain processing associated with fibromyalgia as the molecular events leading to these changes have not been identified (Starkweather & Menzies, 2016).

**Differential Diagnosis**

There are many conditions that mimic the symptoms of fibromyalgia or are seen with fibromyalgia including chronic neck and/or back pain, chronic fatigue syndrome, depression, hypothyroidism, Lyme disease, sleep problems, tendinitis, ankylosing spondylitis, lupus erythematosus, dermatomositis and polymositis, rheumatoid arthritis, and osteoarthritis (Imamura et al., 2009).

**Common Testing**

The diagnosis of fibromyalgia has recently undergone drastically changes. The American College of Rheumatology initially required a history of at least three months of widespread pain as well as tenderness in at least 11 of 18 tender-point sites (arms/elbows, buttocks, chest, knees, lower back, neck, rib cage, shoulders, thighs) (Choy et al., 2010). This criteria was replaced in 2010 and then modified in 2011 with a symptom–based assessment using the Widespread Pain Index (WPI) and Symptom Severity (SS) Scale which focuses on pain being widespread and accompanied by other symptoms such as sleep problems, cognitive difficulty, and fatigue (Wolfe et al., 2011). Diagnostic criteria for fibromyalgia are satisfied if the following three conditions are met: (1) WPI score of 7 or higher and a SS score of 5 or higher (Type A), or WPI of 3–6 and SS of 9 or higher (Type B); (2) symptoms have been present for more than three months; and (3) the individual does not have a disorder that would otherwise explain the pain (Wolfe et al., 2011).

The majority of individuals with fibromyalgia are affected by Type A fibromyalgia, which includes individuals with higher WPI scores. Type B fibromyalgia was added to identify individuals who have all the symptoms but do not have as many painful sites and, therefore, have lower WPI scores. Regardless of classification, patients with the highest WPI score have the poorest physical and mental health status (Starkweather & Menzies, 2016). It is important that physicians rule out other causes of the symptoms included in the criteria before making a diagnosis of fibromyalgia as there may be a co-morbid condition that is exacerbating the patient’s polysymptomatology. These new criteria account for a wide spectrum of fibromyalgia symptoms, which has been called “fibromyalgianess”. This term defines the varying symptoms and severity patients may experience across the fibromyalgia diagnosis.

**Contraindications**

Contraindications to treatment of a patient with fibromyalgia would be to ensure that any treatment be done in a progressive and gradual manner. If treated aggressively, there could be treatment-related pain that could exacerbate symptoms of the disease.

**The Patient Perspective**

The primary symptoms of fibromyalgia are pain and fatigue. The pain is widespread throughout the body and can be deep-aching, radiating, gnawing, shooting, or burning, ranging from mild to severe, and will vary from day to day. Joints are not affected, although the pain may feel like it is coming from the joints. People with fibromyalgia tend to wake up with body aches and stiffness. For some patients, pain improves during the day and increases again during the evening, though many patients have day–long, non–stop pain (PubMed Health, 2010). Although pain can be present while resting, it is more noticeable during muscular use. Pain tends to increase with excessive or lack of activity, cold or damp weather, anxiety, and stress (PubMed Health, 2010). Fibromyalgia pain and fatigue cause difficulties in accomplishing activities of daily living.
Although the primary symptoms of fibromyalgia are pain and fatigue, the effects are much more widespread and not just physical. Normal sensory messages from all over the body are being processed by the brain as pain. This is difficult to understand by both the individual suffering with fibromyalgia, as well as their friends, family, and co-workers. People with fibromyalgia often look well despite significant levels of pain, making it even more difficult for others to understand the level of pain and suffering. Individuals might experience social isolation from the lack of understanding surrounding fibromyalgia and report a significantly lower quality of life. The uncertainty associated with a fibromyalgia diagnosis can further diminish physical and social functioning. Loss of career from the inability to perform tasks can lead to decreased self-esteem and self-worth (Guymer & Littlejohn, 2013).

In addition to pain, fatigue, and psychosocial adjustments, people with fibromyalgia may also be suffering from sleep disturbances, irritable bowel syndrome, alternating bouts of diarrhea and constipation, memory difficulties, numbness and tingling of the hands and feet, palpitations, reduced exercise tolerance, sad or depressed mood, tension or migraine headaches, vision problems, restless legs, dizziness, and impaired coordination (PubMed Health, 2010; NFA, 2017). Therefore, individuals with fibromyalgia are not only dealing with the pain and fatigue, but also suffering with numerous other debilitating complications.

Common Treatment

There is no cure for fibromyalgia. Treatment once focused on localized pain symptoms but recent research has shown that this could make symptoms worse because symptoms are not localized or consistent. It is important to understand that symptoms vary and the severity fluctuates between sufferers and day-by-day. Each individual, with the input of their healthcare practitioner, should develop a multi-faceted approach to treatment and management (NFA, 2017). Conventional medical intervention might only be part of a treatment plan that also includes pain management, sleep management, psychological support, and symptom management.

Pain management (NFA, 2017)

- Physical therapy
- Regular exercise and stretching
- Medication – non-narcotic pain relievers or low doses of antidepressants which are serotonin builders and can improve sleep and decrease pain. Lidocaine can also be used for localized pain.

Sleep management

- Develop a healthy sleep routine
- Consistent sleep/wake time
- Environment is conducive to sleep
- No caffeine, sugar, or alcohol before bed
- Relaxation exercises
- Sleep medication

Psychological support

- Support groups and educational programs to provide patients with information about active self-management of pain, the importance of physical activities and relaxation techniques, pain coping skills, and individual strategies for behavioural change (Imamura et al., 2009).
- Counselling can help improve communication and understanding about the condition and build healthier relationships by decreasing social isolation and improving self-esteem (NFA, 2017).
- Cognitive–behavioural therapy with a main objective to modify the negative behaviour into positive attitudes (Imamura et al., 2009).

Symptom management

Although developing a treatment plan that considers all of the above is critical, one of the most important factors in controlling symptoms is understanding the need for lifestyle adaptation. People need to learn their limitations, pace themselves, prioritize, learn to say no, and delegate tasks that are difficult (NFA, 2017).

Pedorthic Treatment

Pedorthic treatment of fibromyalgia is poorly defined in the research literature. From what information is available, a pedorthist should ensure the use of appropriate footwear for exercise and activity and, when necessary, the use of a custom foot orthosis. A few points to consider when designing custom foot orthoses for an individual with fibromyalgia would be to avoid a rigid device, focus on redistributing forces and pressure, treat the person progressively and not aggressively, and provide appropriate follow-up.
References


Complex Regional Pain Syndrome

Shari Schroeder Stubbs, HBA, O.S.T., C. Ped, C. Ped (C)

Key Messages

- CRPS has no definitive cause.
- Pain far exceeds the original injury.
- Abnormalities in tissue colour, inflammation/edema, pain, and movement.
- No literature supporting specific pediatric pedorthic treatment.

Keywords

causalgia, chronic pain, complex regional pain syndrome, CRPS, nerve injury, reflex sympathetic dystrophy, RSD, Sudeck's atrophy, tissue changes

Introduction

Complex regional pain syndrome is a chronic pain condition that arises as an abnormally severe bodily response to a preceding injury or trauma. Complex regional pain syndrome (CRPS), was formerly known as reflex sympathetic dystrophy (RSD), causalgia, and Sudeck’s atrophy. CRPS has no definitive cause for occurring; it can arise spontaneously or from an injury. For pedorthic purposes, only CRPS arising from injury will be detailed. The pain of CRPS far exceeds the pain of the preceding original injury or trauma. CRPS arising from injuries has been categorized into two types. Type I, formerly known as RSD, arises from an injury which did not cause any nerve damage. Type II, formerly known as causalgia or Sudeck’s atrophy, begins after an injury where some type of nerve damage has occurred (Palmer, 2015).

There are consistently four key symptoms to CRPS. Fukushima, Bezerra, Villas Boas, Valle, & Vidal (2014) stated:

Complex regional pain syndrome (CRPS) is characterised by constant regional neuropathic pain that does not follow the usual distribution of a dermatome or nerve territory and is usually associated with abnormal sensory, autonomic, motor, and/or trophic changes.

Therefore, changes in tissue colour, inflammation/edema, pain, and movement abnormalities are all identifying features of CRPS.

Pathology

The pathophysiology of CRPS is heavily debated in the literature. There are multiple reported mechanisms of cause. “Suggestions include inflammation and changes in the brain and sympathetic, peripheral and spinal nervous systems, (and) aggravated immobility” (Palmer, 2015). CRPS is an exaggerated inflammatory response to an injury or trauma. Other theories include “an autoimmune response, increased sympathetic activity, neurogenic inflammation with increased nociceptive stimulation, deep tissue microvascular pathology, small fibre neuropathy, dysfunction in central processing, and genetic predisposition” (Dickson, 2017). Research shows that after the initial tissue injury or nerve damage, the central and peripheral nervous systems undergo changes, causing increased inflammatory responses and a greater responsiveness to pain. These nervous system adaptations prevent additional damage by avoiding the injurious activities (Goh, Chidambaram, & Ma, 2017). In the pediatric population, studies have shown that CRPS Type I is far more common in females than males, and the lower extremity is far more involved than the upper (Abu-Arafeh & Abu-Arafeh, 2016). The psychopathological factor of stress has also played a large role in perpetuating or creating CRPS in children (Weissmann & Uziel, 2016). Others have theorized that an early predictor or “red flag”
for the development of CRPS is the overly intense pain in the first week following an injury (Elsharydah, Loo, Minhajuddin, & Kandil, 2017).

**Differential Diagnosis**

There are no routine diagnostic or laboratory tests for CRPS. The major complaint of the patient is pain, but pain is included in many other possible diagnoses. Included in the differential diagnosis for CRPS may be diabetic neuropathy, entrapment neuropathy, chronic vascular disorders, inflammatory arthritis, deep vein thrombosis, cellulitis, osteomyelitis, or malignant tumors (Söylev & Boya, 2016).

In 2004, the International Association for the Study of Pain (IASP) validated a set of criteria for the classification for the clinical assessment of CRPS. This set of criteria is known as the Budapest Clinical Diagnostic Criteria for CRPS (Harden, Bruehl, Perez, Birklein, Marinus, Maihofner, Lubenow, Buvanendran, Mackey, Graciosa, Mogilevski, Ramsden, Chont, & Vatine, 2013).

**Budapest Clinical Diagnostic Criteria table for CRPS:**

1. Continuing pain, which is disproportionate to any inciting event

2. Must report at least one symptom in **three of the four** following categories:
   - **Sensory:** reports of hyperesthesia and/or allodynia
   - **Vasomotor:** reports of temperature asymmetry and/or skin color changes and/or skin color asymmetry
   - **Sudomotor/edema:** reports of edema and/or sweating changes and/or sweating asymmetry
   - **Motor/trophic:** reports of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

3. Must display at least one sign at time of evaluation in **two or more** of the following categories:
   - **Sensory:** evidence of hyperalgesia (to pinprick) and/or alldynia (to light touch and/or deep somatic pressure and/or joint movement)
   - **Vasomotor:** evidence of temperature asymmetry and/or skin color changes and/or asymmetry
   - **Sudomotor/edema:** evidence of edema and/or sweating changes and/or sweating asymmetry
   - **Motor/trophic:** evidence of decreased range of motion and/or motor dysfunction (weakness, tremor, dystonia) and/or trophic changes (hair, nail, skin)

4. There is no other diagnosis that better explains the signs and symptoms (Harden et al., 2010)

The majority of CRPS cases usually resolve (90% of symptoms) within two years, but for those cases which progress further, joint contractures appear and the affected limb becomes cold (Söylev & Boya, 2016).

**Patient Perspective**

There are four major symptom categories that a CRPS patient will experience. These are pain and sensory deficits, motor disturbances, connective tissue and trophic changes, and autonomic symptoms. The most common complaint is of neuropathic pain. Neuropathic pain can be reported as a burning, numbness, tingling, stabbing, or electric shock type of pain. Nociceptive pain, in contrast, is the type of pain reported as a sharp, aching, or throbbing type of pain. Both types may be felt with CRPS, but neuropathic pain is more indicative of the syndrome (Fukushima et al., 2014). The discomfort is not isolated to the area of the injury or trauma, is far worse than the originating injury, and usually affects the whole limb (Söylev & Boya, 2016). The sensory deficits felt due to oedema/swelling can make the patient perceive the CRPS affected limb as if it is oversized or a foreign-feeling body (Birklein, O’Neill, & Schlereth, 2015).

The affected limb will also experience motor disturbances. Reductions in motor strength and ranges of motion of the affected limb are seen as a result of the oedema, pain, or fibrotic contractures. Tremors, myoclonic twitches, and fixed dystonia-like positions are also representative motor deficits (Birklein et al., 2015).

At a very early stage of the disease, the CRPS patient will have connective tissue and trophic changes. The subcutaneous fat in the limb will decrease, the skin colour will change, hair loss may occur, and tendon sheaths and fascias may thicken causing joint contractures (McBride & Atkins, 2005). The autonomic symptoms experienced in the affected CRPS limb will consist of the oedema that arises from the excessive posttraumatic inflammation and, in the majority of cases, hyperhidrosis. All affected CRPS limbs will experience a change in skin temperature and a corresponding skin colour change (reddish/hot or bluish/cold) (Birklein et al., 2015).

When CRPS is present in a paediatric patient, the effects are not only patient specific, but will cause...
a significant impact on the whole family unit. The affected child will miss school due to pain and loss of limb function, will cease to play sports, and may cancel social interactions due to their symptoms. The family unit is also affected, as the parents will need to take time off from work to care for the child and family holidays and social events may be cancelled, all of which create more parental and familial stress (Abu-Arafeh & Abu-Arafeh, 2016). The CRPS patient will often be unable to tolerate the touch of bedding, clothing, or even water on the affected limb. Loss of sleep is quite common due to the pain, sensitivity and the warm/sweaty or cold/blue trophic changes that the limb feels (Palmer, 2015).

During the assessment of the patient’s history, we as pedorthists may be the initial practitioners to encounter the patient (or the parent) reporting information of non-standard injury pain, excessive pain, or pain in areas not normally associated with the injury. This information might give us cause to suggest further medical investigations take place. Factors the pedorthist should keep in mind during the initial assessment are that the CRPS-affected lower limb is usually only in paediatric cases, and most of those are female patients.

### Common Treatments

There is no specific treatment for CRPS type I or type II. Researchers agree that the earlier the diagnosis and treatments are started, the better the outcome. An early referral to a specialty pain clinic for an assessment and developing strategies for the management of the pain are essential. As CRPS is highly individualized and very little research has been done on the efficacy of the treatments used, the research community cannot be certain of treatment success, or whether remission occurs (Palmer, 2015). Other multidisciplinary treatments that may be used in addition to pain clinics are:

- **Physical therapy and occupational therapy** – desensitising contrast water baths, strengthening exercises, compression garments to control the swelling, and sensory reeducation. The patient needs to exercise and move the limbs to regain normal activity and function.

- **Psychological therapy** – patients will experience anxiety, depression, and kinesophobia (fear of movement). Kinesophobia may develop because movements cause pain. Patients will need to be treated accordingly to decrease these fears and anxieties.

- **Medications** – there is also very little scientific significance for the type of drugs that are effective with CRPS. Was the drug useful, or did the CRPS go into remission spontaneously? NSAIDs and opioids have both been used to help manage the initial pain and oedema.

- **Surgical and invasive treatments** – epidural pain blocks, implantable pain management pumps, and electrical stimulators have all been utilized, but again with very little scientific evidence to support their use (Palmer, 2015).

Pedorthic treatment of CRPS, type I or type II, is very poorly defined in the literature. As the vast majority of adult CRPS cases are of the upper extremities, this is not something that would fall into the pedorthist’s scope of practice. When paediatric patients are affected with CRPS in the lower limbs, there is no documentation, nor are there scientific studies reporting pedorthic treatments being utilized. If pedorthic roles were to be extrapolated with any lower limb CRPS cases, the pedorthic scope of practice would, in theory, involve the use of custom-made foot orthoses, over-the-counter foot orthoses, and/or orthopaedic footwear. The pedorthist’s role would be to provide very comfortable, supportive footwear, and/or custom foot orthoses to cushion, protect, and maintain proper stability of the lower limbs. This would allow the patient to participate in all types of physical and occupational therapies for regaining movement and function. Future research is required to determine the efficacy of pedorthic treatments of complex regional pain syndrome.

### References


Connective Tissue Laxity

Rodney Ashfield, BA, Cert. Rehab., C. Ped (C)

**Key Messages**

- A host of complex disorders that result in systemic physiological changes.
- May present clinically and often require specialized intervention and support.
- Common foot-related disorders include hormone-mediated ligament laxity associated with pregnancy, Marfan, Down, Ehlers-Danlos and osteogenesis imperfecta syndromes.

**Keywords**

collagen, connective tissue, Down syndrome, Ehlers-Danlos, genetic disorder, heritable, hypermobility, ligament laxity, Marfan, osteogenesis imperfecta

**Introduction**

Connective Tissue Laxity (CTL) refers to a broad constellation of disorders of either dysregulation or deficiency that vary widely in their etiology. These disorders typically manifest clinically as increased laxity in specific or more systemic connective tissues, resulting in joint looseness and altered morphology and pathomechanics.

CTL disorders have varying effects (Bolar, Van Laer, & Loeys, 2012) on the feet that pedorthic interventions can treat to improve foot function, mobility and comfort. Down & Marfan syndromes often present with hypermobile feet, resulting in functional pes planus, or flat feet, while people with Loeys-Dietz syndrome may be born with talipes equinovarus, or club feet. All of these are relatively rare conditions. From a clinical perspective, joint laxity or hypermobility of the feet are also often associated with women’s oxytocin hormone levels during and after pregnancy. Osteogenesis imperfecta (OI) is a genetic disorder with many types affecting osteoblast function, with underlying genetic mutations affecting the formation of collagen type 1 genes. All patients with OI have brittle bones, but may also have joint laxity and a host of extraskeletal manifestations (Alharbi, 2016). As bones are a highly specialized type of connective tissue, protection and shock absorption become key features of orthoses designed for people with OI, as compared to simply structural support.

As a whole, connective tissue laxity encompasses a host of complex multi-faceted disorders that result in physiological changes from accepted norms that may present clinically and often require specialized interventions, including orthotic treatments.

**Pathology**

Connective tissue laxity is most often the result of genetic-level dysregulation of growth processes and specific proteins, such as fibrillin-1 in Marfan’s syndrome. CTL can also present from a deficiency of specific chemicals along key metabolic pathways affecting collagen formation, as is the case with Ehlers-Danlos syndrome. Also an increase in certain hormone levels during pregnancy may result in tissue laxity to the extent that persistent foot and leg pain may develop and feet can change shape.

**Differential Diagnosis**

(Colombi, Dordoni, Chiarelli, & Ritelli, 2015):

- rheumatoid arthritis
- systemic lupus erythematosus (lupus)
- myositis
- Sjögren’s syndrome
- scleroderma
- arterial tortuosity syndrome
- lateral meningocele syndrome

**The Patient Perspective**

Maladies and injuries of the foot and lower leg associated with CTL include recurring ankle sprains (especially inversion involving the anterior talofibular...
ligament), patellar dislocations, joint hypermobility, and associated pathomechanics. Traumatic or spontaneous fractures may occur with OI. Functional pes planus with medial tibial–talar and lateral talor–calcaneal deviations, along with foot abduction, are also more commonly seen with CTL disorders.

Less apparent concerns may involve higher metabolic rates of muscle activity to compensate for joint laxity resulting in premature fatigue or sub-optimal function. Proprioceptively, ligament laxity can impair balance and cause increased risk for falls, increasing with age.

On a positive note, “double-jointed” patients report an ability to excel in activities that rely on increased ranges of motion such as gymnastics, ballet, yoga, and magic tricks (escape artistry). CTL patients, however, generally lack the physical coordination for team-oriented sports.

**Common Treatment**

When connective tissue laxity is a defining characteristic of a patient’s foot and lower limb condition, reduced internal stability results. Therefore, increased externally-derived stability is often necessary. From the pedorthic perspective, there are a plethora of orthoses materials, types, and design details possible to best provide this external support. UCBL-type orthotics with deeper profiles at the heel and midfoot can stabilize joints, tissues, and foot function. The more pervasive the laxity, the more support is needed proximally, which can be provided by appropriate AFO (ankle-foot orthotic) devices or hinged ankle braces. Due to their brittle bones, OI patients also require increased cushioning and shock absorbing designs, favouring flexible contoured arch support to reduce the chance of fractures.

Appropriate footwear needs to be considered with strong hindfoot control (higher boot style, extended stiff heel counters, wide heel and midfoot base, and adjustable closures).

Physiotherapy to strengthen muscles and tendons that cross the lax joints may be recommended to help stabilize movement or improve balance.

**References**


Psoriatic Arthritis

Suzanne Webster, BSc HK, Dip SIM, C. Ped Tech (C), C. Ped (C) (2012)
Updated by Jacob Simpson, BA (Kin), RKin, C. Ped (C) (2018)

Key Messages

• Psoriatic arthritis follows a chronic, progressive course in the majority of patients, and joint damage occurs early in the disease course.
• Early and aggressive treatment leads to best outcome.
• Psoriatic arthritis typically affects the large joints, especially those of the lower extremities, distal joints of the fingers and toes, and also can affect the back and sacroiliac joints of the pelvis (Canadian Dermatology Association, 2017).

Keywords
dactylitis, enthesitis, onycholysis, psoriasis, psoriatic plaques

Introduction

Psoriatic arthritis (PsA) is a seronegative inflammatory arthritis associated with psoriasis that predominantly affects musculoskeletal structures as well as the skin, nails and mucosae (Gladman, Rosen, & Chandran, 2014). Published studies suggest that the prevalence of psoriatic arthritis among psoriasis patients ranges from 7% to 26%. Studies have also found that 15.5% of patients with psoriasis have undiagnosed psoriatic arthritis (Villani, Rouzaud, Sevrain, Barnetche, Paul, Richard, Beylot–Barry, Misery, Joly, Le Maitre, Aractingi, Aubin, Cantagrel, Ortonne, & Jullien, 2015). This high prevalence suggests that individuals with psoriasis should be screened for psoriatic arthritis.

While assessing individuals with psoriatic arthritis, it is important to note the five clinical subtypes. These subtypes, developed by Moll & Wright (1973) specifically highlight the heterogeneity of the disease. The oligoarticular subtype affects four or fewer joints and is most commonly seen in an asymmetric distribution. The polyarticular subtype affects five or more joints symmetrically and can resemble rheumatoid arthritis. The distal subtype affects distal interphalangeal joints of the hands, feet, or both. Arthritis mutilans is the destructive subtype that involves marked bone resorption or osteolysis and the axial subtype primarily involves the spine and sacroiliac joints.

Pathology

Psoriasis is a disease in which scaly, red and white patches develop on the skin. These patches develop due to the body’s immune system going into overdrive to attack the skin. Some people with psoriasis can also develop psoriatic arthritis, manifested by painful, stiff, and swollen joints (Canadian Dermatology Association, 2017). Approximately 15% of people develop the arthritis prior to the skin manifestations (Siddle & Helliwell, 2009). The onset of psoriatic arthritis generally occurs in the fourth and fifth decade of life, with males and females equally affected (Decker & Albert, 2002).

Certain clinical features help to differentiate psoriatic arthritis from rheumatoid and other forms of arthritis, including the presence of axial skeleton involvement, distal interphalangeal (DIP) involvement; dactylitis (a “sausage-like” appearance of the fingers and toes due to swelling and redness); and enthesitis (an irritation of the point of attachment of ligaments and tendons to bone) (Siddle & Helliwell, 2009).

Articular involvement can vary from isolated involvement of midfoot or rearfoot joints, to destructive polyarthritis, with extensive bone loss. It has been reported that isolated foot symptoms could be the initial manifestation of the disease (Siddle & Helliwell, 2009). These symptoms include plantar heel pain, metatarsal pain, dactylitis, and involvement of the ankle and midfoot.
Enthesitis can be clinically observed at the Achilles tendon insertion, plantar fascia, attachment of the tibialis posterior to the tuberosity of the navicular, and the attachment of the peroneus brevis to the base of the fifth metatarsal (Siddle & Helliwell, 2009). People presenting with isolated, recurrent, or bilateral Achilles enthesitis would immediately suggest a diagnosis of psoriatic arthritis, ankylosing spondylitis, or reactive arthritis (Siddle & Helliwell, 2009). In the presence of psoriasis, the diagnosis is clear, particularly if a dactylitic toe is present. Dactylitis is seen more commonly in the feet than the hands. In the feet, the fourth toes are the most frequently involved (Siddle & Helliwell, 2009).

Classical psoriatic plaques can also occur on the dorsum of the foot. Also, nail involvement, with onycholysis (loosening of the nail from the nail bed), pitting, and hyperkeratosis are typical. When these occur in the great toe along with arthritis of the interphalangeal joint, it is referred to as Bauer digit (Siddle & Helliwell, 2009).

**Differential Diagnosis**

Psoriatic arthritis presents similarly to rheumatoid arthritis, osteoarthritis, and gout. Rheumatoid arthritis is characterized as proximal, symmetric involvement of the joints of hands and feet. At its onset, psoriatic arthritis also tends to be less symmetric than rheumatoid arthritis. When differentiating osteoarthritis from psoriatic arthritis, on palpation of the affected distal joints, psoriatic arthritis patients present with soft swelling, whereas in osteoarthritis, swelling arises from a bony osteophyte and is solid. Psoriatic arthritis may also be misdiagnosed as gout due to the uric acid levels being elevated (Villani et al., 2015).

**Common Testing**

Current criteria for the diagnosis of psoriatic arthritis, is based on the CASPAR criteria. According to CASPAR criteria, presence of three of the following five features in a patient with inflammatory articular disease (joint, spine, or entheses) are required to make a diagnosis of psoriatic arthritis: (a) current psoriasis, or personal history of psoriasis or a family history of psoriasis; (b) psoriatic nail dystrophy, including onychomycosis, pitting, and hyperkeratosis; (c) a negative test for rheumatoid factor; (d) current dactylitis or a past history; and (e) radiological evidence of juxta articular new bone formation (Sharma & Dogra, 2010).

**Contraindications**

An important contraindication for pedorthic treatment is the use of rigid, controlling materials when manufacturing custom foot orthoses. Accommodative support is preferential to cushion and offload any involved joints or high-pressure sites. Building on the notion of offloading, restrictive or ill-fitting footwear are also contraindicated. Footwear with appropriate depth, width and volume is an important consideration. It may also be beneficial to avoid footwear with stitched seams, as they may create sites for pressure or friction.

**The Patient Perspective**

The diagnosis of psoriatic arthritis is based on the recognition of clinical and imaging features, since there are no specific biomarkers (Anandarajah & Ritchlin, 2009). Patients with psoriasis should be questioned about joint pain, morning stiffness and evidence of dactylitis (Coates, Aslam, Al Balushi, Burden, Burden-Teh, Caperon, Cerio, Chattopadhyay, Chinoy, Goodfield, Kay, Kelly, Kirkham, Lovell, Marzo-Ortega, McHugh, Murphy, Reynolds, Smith, Stewart, Warren, Waxman, Wilson, & Helliwell, 2013). Early recognition is key as bone erosions are observed in 47% of patients within the first two years, despite the use of traditional disease-modifying medications in more than half the patients (Kane, Stafford, Bresnihan, & FitzGerald, 2003).

**Common Treatment**

Treatment of psoriatic arthritis includes pharmacologic therapies, as well as patient education, physical therapy, and occupational therapy. Interdisciplinary cooperation among the dermatologist, rheumatologist, and physical and
Similar to rheumatoid arthritis, it has been shown that early and aggressive control of psoriatic arthritis leads to optimal outcomes in patients with moderate to severe psoriatic arthritis (Sharma & Dogra, 2010).

**Non-pedorthic treatment may include:**

- NSAIDs
- DMARDs (disease modifying anti-rheumatic drugs)
- Painful joints may be injected with steroid medications
- In some cases, patients need surgery to repair or replace damaged joints

Based on clinical experience and evidence–based strategies for managing disease of the foot and ankle in patients with rheumatoid arthritis, it can be concluded that it is important to address both the inflammatory and mechanical factors that affect the feet of patients with psoriatic arthritis (Siddle & Helliwell, 2009). The use of splints can be beneficial for stabilizing and immobilizing the rearfoot and ankle in cases of enthesitis and peripheral arthritis. Functional foot orthoses to correct deformities such as rearfoot valgus are appropriate for protecting joints and relieving mechanical stresses on the soft tissues. Orthoses can be used to offload painful areas and reduce plantar pressures (Siddle & Helliwell, 2009). Materials for custom foot orthoses should be soft, not rigid, and designed to accommodate deformities. There are many materials available to accomplish this goal and can be decided upon based on the clinical experiences of the practicing pedorthist.

The recommendation of footwear with extra depth and width to accommodate deformities such as nail dystrophies, hallux valgus, claw toes, and dactylitis for patients is appropriate (Siddle & Helliwell, 2009). Shoe modifications may also be necessary and may include: in–shoe excavations to relieve pressure, rocker soles to transfer pressure and improve gait (Decker & Albert, 2002). SACH heels to reduce shock at heel strike may be appropriate for those with rigid or limited mobility (Decker & Albert, 2002). When modification of stock footwear no longer meets the needs of the psoriatic arthritic patient, custom footwear should be considered (Siddle & Helliwell, 2009).

**References**


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Osteochondrosis

A. Brian Stoodley, Dip Orth Tech, C. Ped Tech (C), C. Ped (C)

Key Messages

• Osteochondrosis can be divided into three groups: articular, non-articular, and physeal (Decker & Albert, 2002).

• Osteochondrosis is a disorder of the epiphysis noted during the growth period (Gartland, 1986).

• Osteochondrosis involves the pathologic process of avascular necrosis, the cellular death that occurs in tissue after interruption of its blood supply by trauma or disease.

• Radiographs (X-rays) are needed to confirm diagnosis; such patients should be under the care of an orthopaedic surgeon and/or paediatric specialist.

• Although these conditions primarily affect growing children, residual symptoms of pain and deformity are often noted during adulthood.

• A pedorthist’s role will include offloading direct stress to the affected area. This will include reducing biomechanical faults that can result in tractional pull and/or accommodative support designed to reduce pinpoint pressures on affected sites.

Keywords

avascular necrosis, Freiberg’s, Köhler’s, Legg–Calvé–Perthes, Osgood–Schlatter’s, osteochondritis, Sever’s

Introduction

Osteochondrosis is a family of conditions that result in defective bone formation in the epiphysis or growing part of the skeleton. No one knows the exact cause, although it is assumed to be the result of circulation disturbance to that part of the bone due to a number of possible problems, including mechanical, anatomic, and traumatic etiologies.

In particular, the term osteochondrosis refers to a disorder affecting the pressure epiphyses of any bone during the growth period. The pathologic process is an avascular necrosis of the developing epiphysis. The term avascular necrosis is used to describe the cellular death that occurs in tissue after interruption of its blood supply by trauma or disease. A distinction between osteochondrosis and osteochondritis dissecans should be made. Osteochondritis dissecans is an inflammatory condition of bone and underlying articular cartilage that affects the joints of patients with immature and mature skeletons alike. Either condition may present with activity related pain, but osteochondritis dissecans may cause joint catching and locking. Lesions from osteochondritis dissecans may or may not resolve with nonoperative treatment, whereas osteochondrosis eventually resolves when the growth plates have closed and skeletal maturity has been reached. (Alfred, Suken, & O’Brien, 2011).

Whatever the underlying causes, interference with the epiphyseal circulation occurs, resulting in avascular necrosis involving the epiphysis. The bone of an affected epiphysis often softens, dies, and is gradually absorbed. However, the cap of the articular cartilage surrounding the epiphysis is often unaffected since its nutrition is derived from the synovial fluid. Because the bony tissue contained within the cartilage cap is soft and degenerating, the epiphysis collapses and flattens under body weight pressure. The body attempts to repair this damage by growing new bone, a repair process called “creeping substitution”. The affected pressure epiphysis heals spontaneously with some permanent residual flattening or deformity (Gartland, 1987).

There are numerous types of osteochondrosis; this disorder has been described in nearly every pressure epiphysis in the body and the describers’ names have clung as identifying tags. Most commonly seen by pedorthists are Freiberg’s disease/infraction, Köhler’s disease, Sever’s disease/syndrome, Osgood–Schlatter’s disease/syndrome, and Legg–Calvé–Perthes disease. All describe the same underlying
pathological process with the only difference being the functional importance of the involved pressure epiphysis. Unfortunately, osteochondrosis is typically not recognized in children or adolescents until the onset of clinical symptoms, at which point the disease is advanced (McCoy, Toth, Dolvik, Ekman, Ellermann, Olstad, Ytrehus, & Carlson, 2013).

The pedorthist’s role includes offloading, reducing biomechanical faults, and accommodation of the affected painful areas; however, non-pedorthic approaches such as anti-inflammatory medications, behavior modification, physiotherapy, massage therapy, and/or surgical intervention can play an active part in the physician’s treatment plan for the patient.

Freiberg’s Disease/Infarction

Pathology

This form of osteochondrosis affects the lesser metatarsal heads, in particular the second metatarsal head. Freiberg’s disease/infarction is most commonly encountered in adolescents aged 13–15, and females are affected three times more often than males (Valmassy, 1996).

Symptoms include localized pain, primarily to the dorsal aspect, but also the plantar aspect of the MTP joint; clinical observation reveals localized swelling and thickening of affected joint area, osteophyte-like prominences over the head can often be palpated, and joint range of motion is typically limited or guarded with these patients (Alexander, 1997; Valmassy, 1996).

Differential Diagnosis

Common etiologies of pain originating in this area include capsulitis, capsular tear, synovitis, arthritis, flexor tenosynovitis, interdigital neuroma, and metatarsal stress fracture (Alexander, 1997).

Common Testing

Radiographic findings include a flattened irregularly shaped metatarsal head with a trumpet-like appearance. It should also be noted that since this process is gradual, initial X-rays may not reveal these signs (Valmassy, 1996). Diagnosis is made clinically and imaging is used to confirm. Early in the process, radiographs are normal however bone scans and magnetic resonance imaging are used to confirm diagnosis (Talusan, Diaz-Collado, & Reach, 2014).

The Patient Perspective

Patients may complain of increased unilateral pain throughout the forefoot with weight-bearing activities, especially during propulsive stage of gait.

Common Treatment

Pedorthic considerations should include the goal of offloading the affected metatarsal head; this can be accomplished by various means which, depending on the severity, may include any or all of the following:

- Ensuring proper shoe fit, in particular ample forefoot width to prevent side to side compression of the metatarsals and toe box height to avoid dorsal shoe upper pressure on the affected metatarsal head
- Reducing biomechanical foot deformities via over-the-counter or custom foot orthoses. Along with reducing underlying biomechanical compensations, orthoses may include appropriately placed metatarsal pads, excavations under the affected metatarsal head, low durometer extensions and/or soft top covers
- External shoe modifications, including appropriately placed metatarsal pad, excavation under the affected metatarsal head, soft insole material, properly apexed rocker sole (Decker & Albert, 2002)

Köhler’s Disease

Pathology

This is a form of osteochondrosis that affects the navicular bone in the foot. It is generally found in children aged 3–9, and most texts report that it affects young males more than females. The condition has a chronic yet self-limited course and rarely persists longer than two years (Decker & Albert, 2002; Valmassy, 1996).

Symptoms include pain at the apex of the medial longitudinal arch at the level of the navicular, tenderness through the talonavicular joint, and often swelling.

Differential Diagnosis

Common etiologies of pain originating in this area include arthritis, posterior tendon maladies, accessory navicular, exostosis, plantar fibromatosis, various tendon disorders, plantar fasciitis, midfoot sprain/strain, and stress fracture (Pedorthic Footwear Association [PFA], 1998).

Common Testing

Typically, the child reports pain with mechanical characteristics in the medial region of the foot; however, intermittent or continuous limping may be the only clinical manifestation. (Santos, Estanqueiro, Matos, & Salgado, 2015). Comparison radiographic findings demonstrate thinning or narrowing of the
ossification centre of the involved navicular, with occasional fragmentation (Valmassy, 1996).

**The Patient Perspective**

Often the child will walk with a limp and with the affected foot inverted, as weight-bearing and walking increase pain (PFA, 1998).

**Common Treatment**

Abnormal pronation generally exacerbates this situation; thus, pedorthic intervention should be focused on reducing biomechanical faults. This may be accomplished by any or all of the following (Decker & Albert, 2002):

- Ensuring proper shoe fit, in particular making sure that the upper does not put pressure on the navicular
- Reducing biomechanical foot deformities via over-the-counter or custom foot orthoses. The device chosen should accommodate the tenderness of the navicular
- In-shoe modifications may include scaphoid pads, navicular excavation, and soft insoles
- Extrinsic shoe modifications may include medial wedges, Thomas heels, and in severe cases medial flares, buttresses, and sole widening may be beneficial
- Severe cases can also result in the use of below-knee walkers and plaster/fibreglass casting to completely rest the affected area

**Sever’s Disease/Syndrome**

**Pathology**

Also known as calcaneal apophysitis, Sever’s disease is a condition that affects the heel of the foot. It is believed to be caused by the pull of the Achilles tendon on the traction epiphysis of the calcaneus. It is generally found in boys 7–15 years old, and can often be bilateral.

Pain and discomfort may increase with activity, and in progressively worse cases, there may be pain upon weight-bearing (Cailliet, 1983). The problem is often aggravated by tight posterior musculature associated with a congenital gastrocnemius equinus or a contracted posterior muscle group (Valmassy, 1996).

**Differential Diagnosis**

Common etiologies of pain originating in this area include insertional Achilles tendinitis, retrocalcaneal bursitis, pump bump, plantar fasciitis, plantar fibromatosis, heel pain syndrome, calcaneal stress fracture, and subtalar joint arthritis (Alexander, 1997).

**Common Testing**

Weight-bearing activity and shoes, particularly soccer cleats, can aggravate the symptoms. Physical examination often reveals point tenderness at the insertion of the Achilles tendon, tight heel cords, and heel pain with medial-lateral compression (Alfred et al., 2011). Radiographic examination may be interpreted as showing a dense fragmentation or an irregular appearing apophysis, but not too much significance has been ascribed to this interpretation as this is generally considered to be a normal appearance for this structure (Gartland, 1986).

**The Patient Perspective**

Symptoms are pain and tenderness throughout the back of the heel at a point just below the Achilles insertion.

**Common Treatment**

Pedorthic considerations should include the goal to decrease stress on the Achilles insertion and to avoid pressure at its attachment on the calcaneus. This can be accomplished by various means which will include a team approach with other medical professionals and may include any or all of the following (Decker & Albert, 2002; Gartland, 1986):

- A physiotherapy recommendation if tight musculature is involved
- Ensuring proper shoe fit, in particular avoiding rearfoot counters and toplines that put direct pressure on the tender site. Additional attention should be given to outdoor field cleats, as cleats often sink into the soft ground, creating a negative heel affect and adding stress to the site. An internal heel lift of 1/8 inch may be beneficial to offset the difference
- Reducing biomechanical foot deformities via over-the-counter or custom foot orthoses
- Internal shoe modifications including posterior heel excavation and heel lifts
- External shoe modifications including additional heel lifts and SACH heels
- Severe cases can also result in the use of below-knee walkers and plaster/fibreglass casting to completely rest the affected area
Osgood–Schlatter’s Disease/Syndrome

Pathology

This form of osteochondrosis affects the tibial tubercle, mostly found in boys between the ages of 10 and 15. It is believed to be caused by the pull of the patellar tendon on the immature tibial tubercle epiphysis. This condition may occur bilaterally, characterized by a tender and enlarged tibial tubercle (Gartland, 1987).

The condition is self-limited and the ultimate prognosis is excellent. Acute pain will disappear with time; however, the residual enlargement and sensitivity of the tibial tubercle may be permanent.

Differential Diagnosis

Common etiologies of pain originating in this area include chondromalacia patellae, patellofemoral pain syndrome, plica band syndrome, patellar tendinitis, iliotibial band syndrome, and arthritis (Hunter, Dolan, & Davis, 1995).

Common Testing

The diagnosis is often made clinically, although plain radiographs should be obtained to rule out fractures, tumours, or osteomyelitis if the presenting signs and symptoms are atypical. Radiographs of patients with Osgood–Schlatter disease may show anterior soft tissue swelling and fragmentation of the tibial tubercle. Magnetic resonance imaging and ultrasonography are not routinely necessary (Alfred et al., 2011). Radiographic examination may reveal some fragmentation, apparent separation of the tibial tubercle, with irregularity of the epiphyseal line (Magee, 2007).

The Patient Perspective

Symptoms include pain accentuated by local pressure such as kneeling, and by any activity that causes forceful contracture of the patellar tendon on the tubercle. It is usually associated with activities that involve kicking, jumping, and squatting.

Common Treatment

Pedorthic considerations should include the goal to decrease the tractional pull of the patellar tendon on the tibial tubercle. This can be accomplished by various means which will include a team approach with other medical professionals and may include any or all of the following (Gartland, 1987; McRae, 1993):

- Mild pain may respond favourably to simple temporary restrictions of activities
- A physiotherapy recommendation if tight musculature is involved
- Properly fit footwear to encourage normal internal and external rotation of the tibia
- Reducing biomechanical foot deformities via over-the-counter or custom foot orthoses
- Recommendation of knee brace specifically designed for condition
- Severe pain may require the use of a knee immobilizer or cylinder cast
- Surgery may be indicated if patient does not respond to conservative approaches

Legg–Calvé–Perthes Disease

Pathology

The most common of the osteochondroses, Legg–Calvé–Perthes disease, affects the capital femoral epiphysis. It is also known as coxa plana (flat hip) and It is one of the most important causes of painful limp in childhood. Incongruity of the hip joint secondary to the residual flattening of the femoral head caused by this disorder may lead to a painful osteoarthritis of the hip during adult life. It most commonly affects children 4–8 years old and occurs more commonly in boys. It is usually unilateral but can be bilateral (Gartland, 1987).
Differential Diagnosis
Common etiologies of pain originating in this area include dislocation of the hip, slipped capital femoral epiphysis, scoliosis, osteomyelitis, and arthritis. (Valmassy, 1996).

The Patient Perspective
Symptoms usually include vague limp or pain in its early stages. Pain is usually in the groin, medial thigh, or referred nerve pain to the knee. Midstance phase is short, with a lurch to the affected side. Decreased range of motion is found in abduction and internal rotation when compared to the unaffected side. Local tenderness over the hip joint and muscle atrophy are common as the disease progresses (Valmassy, 1996).

Common Testing
The diagnosis may be confirmed by a thorough clinical evaluation, a detailed patient history, and/or a variety of specialized tests, such as magnetic resonance imaging (MRI), arthrography, scintigraphy, and/or sonography (Kim, 2016). Radiographic findings will vary with each stage of involvement. Serial radiographs will disclose the changes of the epiphysis brought out by necrosis and repair, or “creeping substitution.” The reossification stage is characterized by areas of new bone formation, the femoral neck tends to shorten and broaden, the femoral head deformity can range from enlargement of the head to complete collapse with mushrooming of the head about the neck, and the acetabulum tends to alter its shape to fit the contour of the femoral head.

Common Treatment
Pedorthic considerations should include minimizing femoral head deformity and preserving joint motion regardless of the stage of the disease. This can be accomplished by various means which will include a team approach with other medical professionals and may include any or all of the following:

• A physiotherapy recommendation if tight musculature is involved
• Properly fitted footwear to encourage normal internal and external rotation of the femur
• Reducing biomechanical foot deformities via over-the-counter or custom foot orthoses, close attention to structural leg length differences
• Recommendation of a brace that maintains the femoral head in the acetabulum by abduction and internal rotation

• Surgery may be indicated if patient does not respond to conservative approaches

References


Osteochondrosis
Charcot–Marie–Tooth Disease

Jay Paul, C. Ped Tech (C), C. Ped (C) (2012)
Updated Andrea Durham, R.Kin, C. Ped (C) (2018)

Key Messages

- Treatment for CMT primarily includes counselling for improved daily living, as well as clinical management of symptomatic soft tissue changes and musculoskeletal position/alignment through conservative treatment and/or surgical interventions.
- CMT is a hereditary disease characterized by a slowly progressing and length–dependent peripheral nerve dysfunction resulting in weakness and loss of sensation in the limbs.
- Common names for CMT are hereditary motor sensory neuropathy (HMSN), peroneal muscle atrophy, and Dejerine–Sottas disease.
- Progression and phenotype are highly variable even within the same family.
- Pedorthic interventions may vary: professional shoe fitting, accommodative or functional foot orthoses, dorsiflexion assistive devices, shoe modifications, custom–made footwear, and ankle–foot orthosis.

Keywords

axonal degeneration, Charcot–Marie–Tooth disease, demyelination, hereditary motor sensory neuropathy, HMSN, nerve conduction velocities, neuropathy, peripheral nerves

Introduction

Charcot–Marie–Tooth disease (CMT), also known as hereditary motor sensory neuropathy (HMSN) or peroneal muscular atrophy, is a hereditary neurological disease named after three investigators, neurologists Dr. Jean–Martin Charcot, Dr. Pierre Marie, and Dr. Howard Henry Tooth who first described it in 1886 (Timmerman, Strickland, & Zuchner, 2014). The first description of distal muscle weakness and wasting which begins in the legs was published by Dr. Jean–Martin Charcot and Dr. Pierre Marie under the name of peroneal muscular atrophy in 1886 (Bertorini, Narayanaswami, & Rashed, 2004). Dr. Tooth was the first to correctly attribute the symptoms to neuropathy, rather than myelopathy (spinal cord disease) as was previously believed (Crabtree, 1989). CMT is among the most common inherited group of disorders of the nerves within the peripheral nervous system (Pareyson & Marchesi, 2009). It is marked by slowly progressing peripheral nerve dysfunction, resulting in weakness and loss of sensation in the limbs (Nagai, Chan, Guille, Kumar, Scavina, & Mackenzie, 2006). CMT disease prevalence is 1 in 2,500 making it one of the most common inherited diseases in humans. (McCorquodale, Pucillo, & Johnson, 2016). Genetic research since the 1990s has identified 30 or more genes that, when flawed, result in CMT (Inherited Peripheral Neuropathies Mutation Database [IPN MDB], 2011; Pareyson & Marchesi, 2009).

Pathology

Definitions

Pes cavus/cavovarus – foot structure characterized by an excessively high medial longitudinal arch, inverted rearfoot, and ankle equinus (Hewitt & Tagoe, 2011).

Axon – A thin cable leaving the cell body where electrical impulses from the neuron travel away to be received by other neurons (Queensland Brain Institute, n.d.).

Myelin sheath – The myelin sheath is the protective layer of protein and fatty substances that forms around nerves in the body, including the nerves in the peripheral nervous system (PNS) and the oligodendroglial cells in the central nervous system (CNS). The main purpose of this sheath is to expedite the fast transmission of impulses along the nerves (Siegel, Agranoff, Albers, Fisher, & Uhler, 1999).

Wallerian degeneration – The degeneration of axons and myelin retrograde to the first node of Ranvier in
the nerve proximal to a crush or transection injury and throughout the distal nerve stump (Kury, Stoll, & Muller, 2001; Stoll, Jander, & Myers, 2002).

**Demyelinating** – Demyelination is a loss of myelin with relative preservation of axons. This results from diseases that damage myelin sheaths and the cells. This impairs the conduction of signals in the affected nerves, causing impairment in sensation, movement, cognition, or other functions depending on which nerves are involved (Love, 2006).

**Schwann cells** – Myelinating Schwann cells wrap around axons of motor and sensory neurons to form the myelin sheath (Susuki, Raphael, Yasuhiro Ogawa, Stankewich, Peles, Talbot, & Rasband, 2011).

**Phenotype** – The attributes of an individual and their genetic makeup contrary to genotype which is the inherited material transmitted by gametes that make up an individual’s genetics (Orgogozo, Morizot, & Martin, 2015).

**Etiology**

CMT is a heterogeneous group of genetically distinct disorders with a similar phenotype (Saporta, Sottile, Miller, Feely, Siskind, & Shy, 2011).

There is a strong family history with CMT; however, there are rare sporadic cases without a family history which represent spontaneous mutations (Saporta et al., 2011; Yale School of Medicine, 2018). There does not appear to be any racial predominance, although certain types have been linked to consanguinity (Pareyson & Marchesi, 2009; Pleasure, 2003). CMT is caused by duplication and/or mutations in genes that produce proteins involved in the structure and function of either the peripheral nerve axon or the myelin sheath (National Institute of Neurological Disorders and Stroke [NINDS], 2018). Although different proteins are abnormal in various forms of CMT disease, all of the mutations affect the normal function of the peripheral nerves (NINDS, 2018). All CMT diseases appear to have a common final pathway of axonal loss (Nave, Sereda, & Ehrenreich, 2007). CMT affects the peripheral nerves, those groups of nerve cells carrying information to and from the spinal cord. CMT decreases the ability of these nerves to carry motor commands to muscles, especially those furthest from the spinal cord located in the feet and hands (Muscular Dystrophy Association [MDA], 2018). As a result, the muscles connected to these nerves eventually weaken. CMT also affects the sensory axons that carry information from the limbs to the brain (MDA, 2018). Therefore, people with CMT also have sensory loss. This causes symptoms such as decreased sensation to hot or cold, sense of positioning, and difficulties with balance (MDA, 2018). There are two parts of the nerve that can be affected in CMT: the axon or the myelin sheath (NINDS, 2018). A nerve can be likened to an electrical wire, with the wire being the axon of the nerve and the insulation surrounding it is the myelin sheath (MDA, 2018). Myelin is an insulating layer or sheath that forms around nerves, including those in the brain and spinal cord. It is made up of protein and fatty substances. This myelin sheath allows electrical impulses to transmit quickly and efficiently along the nerve cells. If myelin is damaged, these impulses slow down (medlineplus.gov Nov 19/17). CMT is classified based on which part of the nerve is affected. Historically, people who have problems with the myelin have CMT type 1 and people who have abnormalities of the axon have CMT type 2 (Bertorini et al., 2004; Nave et al., 2007).

**Inheritance**

CMT follows the principles of Mendelian inheritance patterns or genetic mutation (Szigeti & Lupski, 2009). The principle defines one of four ways in which genes or traits are passed from parent to child.

**Autosomal dominant**

- Both sexes can have the condition and each child has a 50% chance of being affected (Szigeti & Lupski, 2009).

**Autosomal recessive**

- Both parents carry a faulty gene but the parents themselves do not manifest the symptoms. Each child has a 25% chance of carrying both abnormal genes and therefore being affected. Either sex can have the condition. The children of an affected individual are usually unaffected but cousin marriages between affected or unaffected members of such families greatly increase the risk that they will have affected children (Szigeti & Lupski, 2009).

**X-linked**

- These conditions are determined by genes carried on the chromosomes which determine the sex of a child. The result is that only boys are affected and they inherit the disease from their mothers who are known as carriers. Carriers are capable of passing the condition on to their own sons. Women who carry the gene can be either completely unaffected, mildly affected, or they may have the condition as severely as males. Each son of a carrier has a 50% chance of having CMTX and each daughter has 50%
Genetic mutation/de novo

- This is an unpredictable change in the structure of a gene causing a different characteristic to appear for the first time in a family. Subsequent generations will inherit the condition in an autosomal dominant pattern (NINDS, 2018; Szigeti & Lupski, 2009).

CMT typing and subtyping

CMT Type 1

- A disorder of peripheral myelination resulting from a mutation or overexpression in the peripheral myelin protein (PMP) 22 gene (NINDS, 2018). The result is segmental demyelination, which causes slowing of nerve conduction velocity (NCV) (NINDS, 2018). The slowing of conduction in motor and sensory nerves results in weakness and numbness of distal extremities (NINDS, 2018).

CMT Type 2

- Primarily an axonal disorder. It results in peripheral neuropathy through direct axonal death and Wallerian degeneration (NINDS, 2018).

CMT Type 3

- Also called Dejerine–Sottas disease. CMT type 3 causes severe demyelination. Onset is infantile and manifests with delayed motor skills, and symptoms are much more severe than CMT1 (NINDS, 2018).

CMTX

- Related to the defects in the X chromosome. It typically affects males and usually starts in the first two decades of life running a progressive and severe course. Heterozygous female carriers may be completely asymptomatic, present with mild symptoms, or on rare occasions, suffer severe symptoms of CMT (NINDS, 2018).

Genetic Mutation

Occasionally a spontaneous mutation flaw in an associated gene will result in a new inherited family disorder (NINDS, 2018; Szigeti & Lupski, 2009).

Age at onset of CMT is usually within the first two decades of life (Pareyson & Marchesi, 2009). Causative gene and type of mutation will determine clinical course, progression rate, and overall severity (Pareyson & Marchesi, 2009). Further subdivisions are based mainly on causative genes and their location. At present, mutations in more than 30 genes have been identified that cause these various forms of inherited neuropathies and more than 44 distinct loci have been identified (IPNMDB, 2011; Pareyson & Marchesi, 2009).

Clinical manifestations

CMT is highly variable in severity and symptomatic involvement even within the same type and among same family members (Pareyson & Marchesi, 2009); whether through demyelination or various axonal impairments, the final common pathway of axonal loss results in the longest axons being especially prone to damage (Nave et al., 2007). Typically, this leads to progressive motor and sensory deficits and diffuse peripheral neuropathy. Changes occur distally and progress proximally, affecting the lower extremities to a greater degree than the upper extremities (Pareyson & Marchesi, 2009). Muscle weakness is almost always bilateral and symmetrical (Nave et al., 2007). As muscle weakness becomes more severe, it appears to progress from distal extensor to distal flexor muscles rather than progressing proximally (Benstead & Grant, 2001; MDA, 2018; Nave et al., 2007). While observing normal walking, the ankle plantar flexor muscles generate nearly all of the positive work in late stance; however, in conditions
where the distal leg muscles are weakened it has been suggested that the hip flexors compensate for the reduced contribution from the plantar flexors and take over the role of swing initiation, resulting in an earlier onset of hip flexion at the end of the stance phase. This limitation will contribute to shortening walking time and distance that can be travelled. (Ramdharry, Day, Reilly, & Marsden, 2009)

**Prognosis**

The symptoms of CMT usually progress slowly over many years and do not usually shorten life expectancy (Bertorini et al., 2004; Pareyson & Marchesi, 2009). Most people with CMT are able to lead full and productive lives despite their physical challenges.

**Differential Diagnosis**

A conclusive diagnosis needs to differentiate CMT from other hereditary neuropathies, acquired neuropathies, distal myopathies, motor neuron diseases, hereditary ataxias, mitochondrial disorders, hereditary spastic paraplegia, and leucodystrophies (Pareyson & Marchesi, 2009). Multiple sclerosis, Friedreich ataxia, muscular dystrophy, and Guillain–Barre syndrome may present with similar etiology. (NORD, 2018). Clinical phenotype will be a primary indication of CMT (Saporta et al., 2010). However, a thorough workup including genetic testing, electrophysiological testing, nerve and/or muscle biopsy, and hematological assessment will establish or rule out the diagnosis (Pareyson & Marchesi, 2009).

**Common Testing**

For pedorthists, the diagnosis of CMT will likely have been confirmed by a neurologist prior to our interventions; however, initial diagnosis is made based on clinical presentation, and a pedorthist may be on the front line for screening. The key features may be pes cavus feet, symmetrical and progressive weakness of the distal limbs, clumsiness, diminished deep tendon reflexes, altered sensory perception and proprioception, and a positive family history of CMT (Nagai et al., 2006). If these symptoms, along with consistent physical examination findings, are present, then the patient and family physician should be encouraged to pursue further investigations to confirm a definitive diagnosis. Additional investigations may include neurology.
consult, electromyography (EMG) studies, nerve biopsy, genetic testing, and orthopaedic surgeon consult (Pareyson & Marchesi, 2009). The first study performed is usually electromyography/nerve conduction study (Yale School of Medicine, 2013). The results will depend upon the type of CMT. Although it is not usually necessary, a nerve biopsy may be done to clarify the differential diagnosis (Yale School of Medicine, 2013). Genetic Testing/DNA duplication testing for CMT Type 1 is now readily available (Yale School of Medicine, 2013). This provides a definitive diagnosis. Even in asymptomatic individuals, 50–60% of cases will be positive (Yale School of Medicine, 2013). The other 40–50% of patients with CMT have another genetic type and may require a combination of the above tests to determine a definitive diagnosis (Yale School of Medicine, 2013).

**Physical exam and common clinical presentation**

The classic CMT patient will usually present with complaints of lower extremity weakness, foot drop, and foot deformities such as hammer toes and claw toes. Examination reveals sensory deficits and motor weakness distally with associated muscular atrophy and absent deep tendon reflexes (McCorquodale et al., 2015). As the condition becomes more chronic, deformities can become rigid (Mosca, 2001). Excessive foot inversion, secondary to the peroneus brevis being overpowered by the posterior tibialis muscle, results in high arched feet (Holmes & Hansen, 1993; Strauss & Angell, 2001). A pes cavus foot type is common, characterized by symmetrical high arches (Nagai et al., 2006; Strauss & Angell, 2001). Plantar flexion of the first ray, forefoot valgus, and forefoot equinus are secondary to the contraction/shortening of the peroneus longus, which may or may not also be weak, acting on a weakened anterior tibialis (Holmes & Hansen, 1993; Vinci, Serrao, Pierelli, Sandrini, & Santilli, 2006). With its insertion at the plantar aspect of the first ray, the peroneus longus overpowers its antagonist muscle, bringing the rearfoot into varus (Mosca, 2001). A weakened anterior tibialis allows the gastrocnemius to pull the foot into equinus, resulting in a spastic drop foot (Holmes & Hansen, 1993; Mosca, 2001). The weakening/wasting of the gastrocnemius and soleus muscles results in the atrophy of that muscle group (Bertorini et al., 2004). This hypertrophy of the proximal muscles, and marked peroneal muscle atrophy leads to a tapering of the distal extremities giving the appearance of an inverted champagne bottle otherwise known as stork legs (Bertorini et al., 2004). Plantar forefoot fat pad atrophy, distal fat pad migration, and plantar callusing may develop on weight-bearing areas. In the presence of ligamentous laxity, deformity is exacerbated (Strauss & Angell, 2001). Immobility without adequate joint range of motion results in contracture (Strauss & Angell, 2001). Fixed or flexible ankle varus manifestations may be present, resulting in musculoskeletal mal-alignment (Strauss & Angell, 2001). In later more progressive stages of the disease the patient may have decreased strength in the forearms and hands (Bertorini et al., 2004). Scoliosis may also be present in one third of all patients (Karol et al., 2007). Patients may also present with sensory deficits in the lower and upper extremities (NINDS, 2018; Yale School of Medicine, 2013).

**Pedorthic assessment**

The patient should present with a detailed prescription including diagnosis and requested treatment from a neurologist. Once it is determined that the diagnosis is CMT, the history should include patient demographics, past medical history, previous surgical interventions and other related treatments, family history (particularly as it may relate to previous diagnosis of CMT), occupation, recreational activities, activity level, and chief complaint(s).

On completion of history, the pedorthic examination should continue with observation of the patient’s footwear, looking for signs of abnormal wear and structural deviations. Footwear evaluation provides useful indications to the current symptom manifestations due to abnormal mechanics and pathological gait. Special attention should be paid to visual observation as to whether there are lateral/medial upper deviations, toe dragging, evidence of midfoot or toe strike, reduced heel contact, excessive lateral sole wear, toe-box deformation, excessive internal wear on the insole at the first/fifth metatarsal phalangeal joint, and lateral shearing on the internal counter. Once the footwear is removed, the pedorthist can examine the surface anatomy of the feet for abnormalities. CMT patients may have pes cavus, forefoot adduction, midfoot supination, and pes varus deformity. These findings revealed that the pes cavus deformities of CMT patients tend to be worse with disease severity (Joo, Choi, Kim, Jung, Cho, & Hwang, 2011). A biomechanical evaluation should include evaluating the range of motion of the talocrural, subtalar, midtarsal, metatarsal, and digits. Limitations and pain on range of motion testing, crepitus and soft tissue limitations, contractures, and fixations should all be noted. As CMT is a progressive disease involving sensory and motor deficits, examination should include a Semmes–Weinstein 10-gram monofilament test to determine if there is a
stocking/glove sensory impairment (American College of Physicians [ACP], 2017). Neurological testing with a Semmes–Weinstein 10-gram monofilament is a reliable screening tool for identifying sensory deficits or loss of protective sensation (Feng, Schosser, & Sumpio, 2017). Sensory loss is associated with dry skin and hair loss in the affected area due to ischemia (MDA, 2018). Patients with CMT may not complain of numbness or altered sensation as they may have never known normal sensation. Patients may present with painful symptoms secondary to muscle cramping or neuropathic pain as a result of defective axonal signals (MDA, 2018). The manual muscle strength testing (MMT) scale of 0–5 can also be used to assess muscles weakness progression on follow-up (Weber, 1993). Muscle testing should include the muscles specifically targeted by the disease in CMT patients. This should include examination of the following pathological characteristics:

- Claw/hammer toes (including the hallux) present when there is simultaneous contraction of extensors and flexors with weak or insufficient intrinsic muscles (Wheeless, 2012). Weak lumbricals, plantar interossei, and dorsal interossei fail to adequately aid in flexion the metatarsal phalangeal joints and extension of the proximal interphalangeal joints (Kendall, McCreamy, Provance, Rodgers, & Romani, 2005). Proximal interphalangeal joint hyperflexion is caused by contraction or relative excessive pull of the flexor hallucis longus/flexor digitorum longus (Kendall et al., 2005). There is hyperextension of the metatarsal phalangeal joints with relative excessive pull of the extensor tendons as the extensor digitorum longus and extensor hallucis longus are recruited to aid in ankle dorsiflexion and indirectly plantar flex the metatarsal phalangeal joints (Kendall et al., 2005).

- Test for weak intrinsic muscles (lumbricals, plantar interossei, and dorsal interossei), contracture, or excessive pull/activity of extensor hallucis longus/ extensor digitorum longus and flexor hallucis longus/flexor digitorum longus.

- Pes cavus secondary to weak peroneus brevis and the tibialis posterior, creating an increased arch height.

- Test for weak peroneus brevis (patients with cavus and equinus foot types will have great difficulty demonstrating a heel walking test).

- Forefoot valgus, plantar flexed first ray, and forefoot equinus due to peroneus longus overpowering anterior tibialis.

- Test for weak anterior tibialis and tight peroneus longus.

- Test for drop foot due to weak tibialis anterior, allowing stronger gastrocnemius/soleus complex to plantar flex the foot.

- Test for weak anterior tibialis.

- Increased rearfoot inversion secondary to peroneus longus overpowering the tibialis anterior, resulting in rearfoot varus.

- Test for weak tibialis anterior and perform Coleman block test for rearfoot flexibility.

- Coleman block testing involves having the plantar flexed first metatarsal hang free from the block while the lateral part of the foot stands on the block. A flexible hindfoot corrects to neutral with block under lateral aspect of foot while rigid hindfoot will not correct (Alexander, 1990). This test will provide indications to appropriate treatment strategies as the disease progresses.

Gait

As CMT is a slowly progressive disease, patients usually present with an increasingly pathological gait pattern. At the heel-strike phase of gait, there will be excessive lateral initial contact with reduced heel contact and loading, secondary to the cavus foot type. Progressing to midstance, there is decreased eccentric contraction of the tibialis anterior and increased plantar flexion secondary to the equinus deformities. The result is a midstance flat foot or toe-to-heel gait with excessive foot supination throughout the gait cycle (Newman, Walsh, O’Sullivan, Jenkinson, Bennett, Lynch, & O’Brien, 2007). Genu recurvatum may be present during stance phase of gait, coupled with decreased plantar flexion at toe off due to weak gastrocnemius and soleus muscles. During the swing phase of gait there is reduced dorsiflexion due to weak tibialis anterior muscles, resulting in foot drop and poor ground clearance. Patients may exhibit a wide base of gait to encourage increased balance and stability. There is potential for an ataxic gait secondary to sensory deficit or a high stepage gait secondary to foot drop as the patient tries to clear the ground during swing phase (Nave et al., 2007; Newman et al., 2007).

Contraindications

Individuals with CMT, without equinus, and with weak plantar flexors should not wear footwear with heel elevation, as reduced ankle stability increases
the risk of sprains (Vinci & Pierelli, 2002). Individuals with CMT should be professionally fitted with footwear and should not self-select the treatment, as sensory deficits may influence poor decision criteria. Patients that have developed talocrural arthrodesis(es) or have a rigid ankle-foot orthosis would benefit from footwear with lateral torsional stability and appropriate rocker sole or rocker sole modifications. The patient’s positive perception of self-image encourages and improves acceptance and/or treatment compliance. Due to the cavus foot type of individuals with CMT, any footwear with features that limit dorsal volume should be avoided, such as Balmoral or slip-on style footwear. Pronation-control footwear should also be avoided, as this may encourage lateral instability (Vinci et al., 2006). Prefabricated ankle-foot orthoses must be carefully screened and may be contraindicated for patients with altered sensation, fixed deformities, and/or contractures. As most off-the-shelf ankle-foot orthoses have an ankle alignment of 90 degrees, the presence of equinus, contractures, and/or sensory deficits may result in increased potential for pressure sores. Patients should avoid stresses on their peripheral nervous system, such as poorly managed diabetes, excessive starvation, obesity, or drinking excessive amounts of alcohol (Nave et al., 2007). Pedorthists must not practice beyond their scope or skill level and recognize when it is appropriate to refer their patient to an allied health care provider for further treatment.

**The Patient Perspective**

CMT is characterized by slow progressive weakness beginning in the distal limb muscles, typically in the legs before the arms (Pareyson & Marchesi, 2009). Patients may first complain of lesser toe deformities, feeling clumsy, having difficulty with walking, stubbing their toes, tripping or falling, and/or previous ankle sprains. They may complain of pain under the forefoot or on the lateral border of the foot (Karadsheh, 2018). Patients may complain of muscle cramping (MDA, 2018). Patients may also describe difficulty in fitting shoes due to high insteps. In early stages of CMT, when plantar flexors are still strong, patients may feel more comfortable walking in a shoe with a slight heel elevation that would accommodate early plantar flexion contracture. In later stages of the disease progression, patients may complain of decreased abilities to use zippers or buttons, or turn door knobs (Domino, 2010). Patients may also complain of depression due to decreased ability to manage activities of daily living (Vinci et al., 2006; Vinci & Pierelli, 2002; Yale School of Medicine, 2013)

**Common Treatment**

Currently there is no cure for the underlying disorder of CMT (Nave, 2007); however, there are currently studies and trials ongoing to develop treatments for Charcot-Marie-Tooth disease (Sheffield Institute for Translational Neuroscience, 2018). The disease's course and its severity is highly variable even within the same CMT type and among siblings (Pareyson & Marchesi, 2009). It is a progressive disease and, as such, the management is not definitive. A sequential approach to the evaluation and treatment of the condition is required. Consequently, pedorthic management will be greatly varied. Detailed and sound pedorthic assessment, coupled with reasonable patient goals and expectations, will largely influence the choice of treatment. Management should focus on the prevention and treatment of foot deformities, lateral stability, management of foot drop, and the distribution of plantar pressure.

Early stage treatment with lesser digit deformities, mild foot drop, and peroneal weakness may be adequately managed with functional foot orthoses, high-top shoes, a deep toe box, and a small heel lift of 2cm or less that may reduce the need for additional dorsiflexion assist devices (Vinci & Pierelli, 2002). Foot orthoses with lateral forefoot/rearfoot posting will encourage lateral stability and functionally stretch the muscles, causing inversion (Vinci & Pierelli, 2002). However, triceps surae and quadriceps strength must be carefully evaluated to ensure adequate ankle stability and knee strength to prevent inversion sprains and extensor weakness falls (Vinci & Pierelli, 2002). The progression of CMT often leads to increased lateral instability which is best managed with footwear modifications such as lateral flares or buttresses to extend the base of support and discourage varus deforming forces (Weber, 1993). Increasing muscle imbalances may lead to further deformity, inversion, and contractures of the foot resulting in greater plantar pressures, lateral pressures, lateral instability, and drop foot. Surgical intervention may be a consideration as well, such as tendon transfers and/or tendon releases. Ankle-foot orthosis (AFO) may be indicated as the symptoms progress. If a gross deformity is present, custom footwear may be required to accommodate the AFO management. In the late stages of the disease, where deformities may be fixed, surgical interventions are indicated. However, if the patient is not a surgical candidate then above-ankle custom footwear may be indicated. This footwear management may include accommodative orthotic inserts with internal buttressing and an equinus cradle to create a lateral
The plantar column of support and equal distribution of plantar pressures. High lateral counters (over the ankle joint) will encourage lateral stability. Velcro closures allow for easier donning and doffing with weakened hand and forearm strength. Rocker soles with a mild heel rocker provide decreased shock loading at heel strike, a stable midfoot, and an aggressive forefoot rocker to allow forward progression over fixed/fused deformities and adequate clearance during swing phase. Whenever bracing principles are employed to create an arthrodesis, rocker soles will be required to reduce excess loading and allow for adequate motion around the affected joint(s) (Weber, 1993).

Outside of pedorthic treatment, patients could benefit from:

- Orthopaedic surgical consultation – surgical intervention to improve muscle balance and realign bone structure.
- Physiotherapy and massage – preserve muscle strength, balance, and range of motion, and minimize deformity secondary to contractures.
- Orthotist consultation – upper and lower limb bracing for stability, balance, and muscle control.
- Podiatry care – nail and skin care, wound care, and prevention.
- Occupational therapist consultation – daily living and mobility aids.
- Genetic counselling – to make informed choices regarding potential risk of their children having the disease.
- Psychological counselling – to cope with the decreased abilities to manage activities of daily living in the same manner as they had previously been able to.

All healthcare professionals need to participate in patient education in all aspects of disease management for best patient outcomes.

**Key Considerations**

Key considerations will be specific to assessment findings and disease progression; however, they will revolve around patient symptoms and severity of impairment as they relate to age, activity level, occupation, recreation, social support, expectations, goals, patient preference, and willingness to participate in treatment. There may be more than one option for treatment at any stage and each must be weighed carefully as to its potential benefits and drawbacks.

**References**


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Key Messages

- Post-poliomyelitis syndrome typically presents with delayed onset muscle weakness decades following acute polio.
- New cases of acute poliomyelitis have been eradicated in North America following the introduction of a vaccine in 1956.
- Patients can benefit from a multidisciplinary approach to treatment including controlled strengthening programs and joint-stabilizing orthoses.

Keywords

atrophy, denervation, fatigue, neurological, muscle weakness, nociceptive pain

Introduction

Post-poliomyelitis syndrome (PPS) is a neurological disorder that can affect some individuals who had previously contracted poliomyelitis (polio). Polio is a highly infectious viral disease most commonly spread through contact with nasal/oral secretions or the stool of an infected person (LaRocco, 2011). Until the introduction of the Salk inactivated polio vaccine in 1956, polio was considered the most feared childhood disease in the United States. However, after the introduction of the vaccine, a drastic reduction was seen over the following decade (LaRocco, 2011). More than 50 years later, most post-polio cases are limited to an aging population who were first affected by poliomyelitis as children.

While it is likely that paralytic poliomyelitis has been around for thousands of years, first reports of delayed onset muscle weakness, fatigue, and atrophy were not reported until 1875 in Paris, and only in the past two to three decades has there been an increasing acceptance of a new stage of neurological decline following years of stability (Trojan & Cashman, 2005). The two most common complaints of PPS survivors are muscle fatigue and weakness, while the two most affected activities of daily living are walking and climbing stairs. The frequency of neuromuscular complaints in polio survivors 20 to 40 years after the initial illness has been reported to be as high as 80% (Flansbjer et al., 2000) and these findings have been consistently reported in the research. Studies vary in regard to the prevalence of fatigue, pain, and new muscle weakness reported by PPS patients. Muscle and joint pain in the range of 38–86% and 42–80%, respectively, have also been reported by survivors of poliomyelitis. Werhagen & Borg (2010) found that 60% of patients reported pain in a study that looked at the prevalence of nociceptive and neuropathic pain. New onset muscle weakness is a key complaint in a PPS population. Ankle dorsiflexor strength has been frequently used as a measure of strength gains and losses and can be used in the development of exercise and rehabilitation programs or to simply monitor strength changes over time to aid in other treatment recommendations, such as the need for walking aids and braces (Flansbjer et al., 2011). Potential strength gains of the quadriceps muscle group have also been studied, especially important since adequate quadriceps muscle strength is crucial for an individual to stand and walk. A variety of orthotic devices have been shown to improve gait disturbances secondary to muscle weakness (Agre, Rodriguez, & Franke, 1997). Due to the large population of poliomyelitis survivors, PPS is considered to be the most common motor neuron disease in North America today.

Pathology

The cause of post-poliomyelitis syndrome is not clearly understood, but the most accepted hypothesis maintains that during the initial polio illness there is degeneration of motor neurons of the anterior horn cells of the spinal cord and brainstem, and subsequent denervation of the associated muscle fibres (Laffont et al., 2009). During the recovery period following the acute polio virus, the motor
units can become seven to eight times larger than normal, through terminal axonal sprouting and re-innervation of muscle fibres. Typically there is a stable period, often decades, where these motor units can handle the extra workload. Eventually terminal axonal sprouts degenerate and are no longer able to innervate the muscle fibres. For a time, axons in close proximity can sprout and re-innervate these fibres, but eventually the load on the remaining motor units becomes too great and complete denervation occurs. It is this denervation of muscle fibres that is thought to cause delayed onset muscle weakness associated with post-polio myelitis. New onset muscle weakness typically occurs in muscles that were originally involved in the acute case of poliomyelitis, but it is not uncommon to have muscles that underwent a lower level of motor neuron loss also showing delayed onset of symptoms (Trojan & Cashman, 2005). Following the initial illness of paralytic poliomyelitis, there is a recovery period which could last months or years, followed by a longer period of functional and neurological stability which typically lasts for several decades. On average, there is a lapse of 36 years from initial illness to the onset of new symptoms (Trojan & Cashman, 2005). The term “post-polio syndrome” was first used in 1985 by Halstead to describe a set of symptoms including muscle weakness, muscle fatigue, and muscle and joint pain, commonly described by polio survivors following a stable period of more than 15 years after the initial polio symptoms presented. Table 1 summarizes the criteria for diagnosing PPS. In 1991 Halstead went on to revise the criteria to include the gradual or abrupt onset of neurogenic weakness as a requirement, regardless of co-existing symptoms.

**Table 1: Diagnostic criteria for post-polio syndrome**

<table>
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<tr>
<td>A prior episode of polio as confirmed by history, physical exam, and EMG findings.</td>
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<tr>
<td>A period of neurologic recovery following the acute polio episode.</td>
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<tr>
<td>A period of at least 15 years of neurological and functional stability.</td>
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<tr>
<td>Complaints of two or more of the following symptoms: fatigue, muscle/joint pain, new muscle weakness, new muscle atrophy, cold intolerance, and functional decline.</td>
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<td>Elimination of other possible co-morbidities.</td>
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(Halstead, 1991)

While patients often report symptoms of muscle and joint pain, muscle weakness, and fatigue, these are only the most common complaints. Weight gain in polio survivors tends to be high. Laffont *et al.* (2010) reported a rate of 28.4% of patients in one study having a body mass index (BMI) greater than 25kg/m². Tendon diseases are also more common in the post-polio population although the upper body seems to be more involved in these cases. Arthritis has a high incidence rate and is more likely to affect joints of the lower limb. Arthritis can affect the joints of the midfoot and can make for greater footwear complications and difficulties in ambulation (Laffont *et al.*, 2010). Less frequently reported are symptoms of muscle atrophy, respiratory insufficiency, sleep abnormalities, dysarthria, dysphagia, muscle cramps, intolerance to cold, muscle fasciculations, and joint deformities (Trojan & Cashman, 2005). Individuals with PPS can live highly independent lives as symptoms of post-polio syndrome are slowly progressive and can often take 20–40 years to develop following the initial illness (Gonzalez, Olsson, & Borg, 2010).

Activities of daily living (ADLs) do not seem to be greatly affected, however, stair climbing and walking long distances can be more difficult for PPS patients.

### Differential Diagnosis

There are a number of difficulties with diagnosing PPS. Evidently, confirmation of an initial bout of poliomyelitis is a necessary factor for the development of PPS, however, it is difficult to relate all of the symptoms that might present in a patient with PPS to the initial illness. Pain, for instance, is one of the most common complaints associated with PPS, however pain is a common complaint in many conditions. In a pain study conducted by Werhagen & Borg (2010), 67% of subjects reported pain and the majority of these people reported that the pain was nociceptive in nature. For the small percentage of subjects that reported both nociceptive and neuropathic pain and neuropathic pain only, further clinical and neurological testing revealed that another underlying cause of this pain was present, such as disc herniation with medullar compression, stroke, and meningioma. It is therefore important to conduct a thorough clinical and neurological assessment.
pertaining to the type of pain patients experience.

The normal aging process can evidently lead to many of the same symptoms seen with patients with PPS. Muscle weakness, atrophy of muscles, fatigue, difficulty with ambulation, and increased pain are all more common complaints in the elderly population. While those with PPS tend to develop more rapidly progressive muscle weakness (Flansbjer et al., 2000) it might still make for some difficulty in determining whether weakness is part of the normal aging process or part of this new syndrome.

There are many other diseases that mimic PPS in some way, considering that PPS can have an effect on many muscles and systems of the body. Osteoarthritis, fibromyalgia, and multiple sclerosis are a few other diseases that could confound the diagnosis. Fatigue, which is a common symptom of PPS, is also closely associated with depression and it should be treated appropriately (LaRocco, 2011). Amyotrophic lateral sclerosis (ALS), more commonly known as Lou Gehrig’s disease, can present similarly to PPS. ALS also affects the anterior horn cells and leads to degeneration of the motor nerve cells. Muscle cramps, fasciculations, weakness, and fatigue are common symptoms associated with ALS. Progression of ALS is typically more rapid than with PPS and patients typically die within three years of the onset of symptoms (Decker & Albert, 2002). Guillain–Barre syndrome is a condition that affects the peripheral nerves. It has a rapid onset and can present as muscle weakness and possible paralysis of the legs, arms, and respiratory muscles. Numbness and sensation changes in the feet are not uncommon. A rapid development of symptoms is followed by a stable period of no change and a final recovery period which could last up to six months. A minor pulmonary or gastrointestinal infection could precipitate the development of Guillain–Barre syndrome (Decker & Albert, 2002). It is therefore important that thorough history-taking be conducted to help rule out other conditions that present similarly to post-polio myelitis syndrome.

**Common Testing**

There are currently no tests to accurately diagnose PPS. It is largely a diagnosis of exclusion. There are, however, certain criteria that can help to establish a diagnosis. These are: confirmation of a prior episode of poliomyelitis; a stable period of recovery of at least 15 years from the initial infection; gradual development of new muscle weakness and fatigability for a period of more than one year; and the exclusion of medical conditions that present similarly to PPS. In instances where the history of illness is unsupported or doubt remains, EMG (electromyography) studies are also used to confirm neurological impairment as indicated by motor neuron loss and signs of denervation (Gonzalez et al., 2010; LaRocco, 2011).

It is important to conduct a thorough initial assessment in order to rule out other co-morbidities such as radiculomyelopathy, peripheral nerve compression syndromes, and degenerative joint disease. Other conditions that might cause muscle pain and fatigue, and therefore make diagnoses of post-polio myelitis more difficult, include hypothyroidism, polymyalgia rhematica, and other connective tissue disorders (Bridgens, Sturman, & Davidson, 2010).

**Contraindications**

More than a century after Raymond (1875) first described new muscle weakness thought to be associated with an earlier polio illness, and 30 years after Halstead introduced the term “post-polio syndrome”, one of the most controversial aspects
of treatment of this condition surrounds the use of muscle strengthening as a potential treatment for the delayed onset muscle weakness. Several studies have looked at the use of a muscle strengthening exercise program to determine if strength gains could be achieved, but there are few studies that have also considered whether this exercise had deleterious effects on the integrity of the muscle fibres. Measuring levels of creatine kinase indicates if there has been damage to the muscle (Agre, Rodriguez, & Todd, 1997). A longitudinal study carried out by Agre et al. (1997) looked at the effects of a moderate intensity muscle strengthening program on the quadriceps muscle group. Creatine kinase levels were measured prior to and at the completion of the program. The exercises were preformed four times per week for 12 weeks. They concluded that there were increases in muscle strength, work performance, endurance, and isometric tension time index without any measured increases in serum creatine kinase levels. It appears that the exercise program must be of adequate intensity to positively affect strength, without being too intense as to cause further muscle damage and stress to affected muscles. The difficulty facing many health care providers is not having a lot of long-term data, combined with the inevitable aging process facing nearly all polio survivors in North America.

Focusing again on the negative effects that excessive muscle exertion may cause, it is important to look at other activities that the patient with PPS is involved in. High levels of muscle exertion may not only be present in a controlled muscle strengthening program—the patient’s type of employment might be physically demanding as well. Failing to assess the work being performed on a daily basis could lead to greater muscle stress and damage to already vulnerable muscles. It might even be necessary to change tasks or occupation to eliminate the potential for overload (Trojan & Cashman, 2005).

The Patient Perspective

The most common complaints associated with post-polio syndrome include fatigue, muscle weakness, atrophy, and pain (Agre et al., 1997; Trojan & Cashman, 2005). Sleep disturbances, respiratory issues, dysphagia, and cold intolerance have also been reported (Bridgens et al., 2010). Fatigue can be described as general exhaustion made worse with activity and improved with rest, aching and heaviness in the muscles, and mental fatigue (Bridgens et al., 2010). Muscle weakness has been found to affect muscles that were affected by the initial polio virus but can also affect muscles not involved initially. Patients might report greater difficulty walking as muscle weakness can lead to difficulty with ambulation (Flansbjer et al., 2000) as well as stair climbing (Agre et al., 1997). Muscle atrophy might concern a patient as they find activities that were once easy now more difficult to accomplish. Muscle atrophy can lead to such alignment issues as genu recurvatum as the patient might use this compensation to lock the knee back into hyperextension in order to stabilize the joint in weight-bearing (Laffont et al., 2010).

A study conducted by Werhagen & Borg (2010) reported that pain is the most common complaint associated with PPS. Pain was divided into three groups; nociceptive, neuropathic, and a combination of both. The majority of patients reported nociceptive pain. They looked at the relationship between pain and gender, age at time of initial illness, age at time of examination, and disability. Disability was rated using such tests as the visual analogue scale (VAS) and WISCI (walking index for SCI). It was concluded that 67% of subjects reported pain. Women were nearly twice as likely as men to report pain (88% and 49% respectively). Younger age at the time of the initial polio virus correlated with greater reported pain. Interestingly, patients who were older at the time of examination actually reported pain less often than younger patients.

Common Treatment

There is no specific treatment for PPS. Since the medical problems affecting individuals with PPS can affect varying systems of the body, a multidisciplinary approach to treatment is recommended in the literature. Muscle strengthening and physical activity has been suggested to improve overall functioning (Gonzalez et al., 2010). Agre et al. (2007) reported that a program of muscle strengthening can help to increase overall muscle strength and endurance, without any negative impact on the motor units of the muscle. Farbu et al. (2005) reported positive patient response to warm climate and warm water exercises with respect to reduced pain, health-related problems, and depression. Application of controlled exercise to improve function and minimize muscle damage is put into practice by the American Physical Therapy Association (APTA) section of neurology with exercise guidelines for PPS patients. These guidelines include aquatic exercise, aerobic exercise, and stretching. APTA also warns against exercise when fatigue is significant. Energy conservation and lifestyle changes should be considered a priority.
Flansbjer et al. (2000) studied the reproducibility of ankle dorsiflexor muscle strength measurements since the strength of the ankle dorsiflexors can have a great impact on mobility and the risk of falls. The results were in favour of reproducibility of isometric and isokinetic strength measurements. In a clinical setting this could have future benefits. It might be possible to determine increased weakness in the muscle being tested, and use this to encourage the patient to take greater control of their treatment regime. This might also allow clinicians and researchers to detect changes in strength following a muscle strengthening routine to reinforce the inclusion of strengthening in the patient’s treatment protocol. There has been some concern, however, that too much demand placed on the already overloaded motor units could create more damage and lead to greater muscle weakness and pain. Depending on the method of locomotion, studies have shown that pain is more commonly found in the lower extremities in patients who ambulated and in the upper extremities in patients who used crutches or wheelchairs. It has been suggested that muscle pain might be a sign of overexertion and therefore a fine balance seems to exist between strengthening and inclusion of exercise into a treatment program, and the avoidance of becoming too physically active which could lead to strength losses and increased muscle pain (Trojan & Cashman, 2005). Greater investigation in this area should be conducted to confirm best practices. Obesity management is another key component in the management of PPS (Bridgens et al., 2010), so a balance must be found in order to maximize weight management and maintenance of strength without over-stressing the muscles.

In some cases where there has been greater weakening and atrophy of muscles, the integrity of the joints on the lower extremity can be affected. Bracing and orthoses for the lower limb can be beneficial in providing improved stability to the joints. Kelley & DiBello (2007) developed a classification system to help health care professionals determine the type of orthoses that could provide the greatest advantage to the patient. This report looked at the effect of orthoses with respect to reduction of pain, falls or near falls, and muscle weakness and fatigue. Orthoses can help to stabilize joints and reduce painful motions. Falls can be due to such things as instability of joints causing buckling of the joint, or in swing phase, the inability to dorsiflex the ankle to achieve ground clearance causing tripping. Difficulty with walking can also be due to muscle fatigue or weakness. An appropriate orthoses can help to reduce the muscle expenditure necessary for ambulation. Depending on the severity of symptoms, an appropriate orthoses could be for the foot only, or in more extreme cases of weakness and atrophy, an AFO (ankle–foot orthosis) or KAFO (knee–ankle–foot orthosis) might be indicated. Lightness of the materials used will enhance compliance and minimize increased fatigue in already weakened and fatigues muscles.

Trojan & Cashman (2010) also recommend the use of assistive devices and orthoses as indicated which can improve the patient’s ability to walk, reduce their pain, and accommodate joint deformities.

Weakness of the quadriceps muscles is a common finding in PPS patients. This can lead to difficulties with standing and walking and thus have significant impacts on ADLs. Rahul, Mohanty, & Tharion (2014) looked at the effects of an articulating KAFO to determine the effect of controlling knee hyperextension during gait. Benefits of the articulating option allow for a free swing phase and ease of sitting. This case study evaluated the effects of an articulating knee hyperextension orthosis on energy expenditure during gait. They were able to conclude that while the KAFO did reduce knee hyperextension and reduce walking heart rate, there are limitations to the conclusions drawn from a study with only one subject. The walking speed actually increased from 9.6 m/s to 7.2 m/s. This was most likely due to the brace being new and the subject not having time to adjust to it. Reduced energy expenditure by producing a more normalized gait pattern should be the primary goal of orthosis use.

Footwear and footwear modifications are important considerations in a post-polio population. As muscles become weak and atrophied, footwear can provide added stability and a stable platform for the foot. Footwear that comes above the ankle may be necessary to support the ankle. Modifications to footwear can be beneficial in compensating for muscle weakness. Medial and lateral flares or wedges could be added to footwear to compensate for muscle weakness and will depend on the muscles most affected (Decker & Albert, 2002). There are various other footwear modifications possible and decisions will be dependent on possible deformities and level of weakness present. An individual approach is important and hence a thorough examination is necessary to determine what changes may be required to make walking more comfortable and less fatiguing to the patient.
Key Considerations

In North America, it is an older population that is affected by post-polio myelitis, considering the last cases of polio were seen over 50 years ago, and only 61 cases of polio were reported in the United States in 1965 (LaRocco, 2011). Compliance with treatment protocol could be an issue within this population as activities become more difficult to carry out due to increasing age and potentially the severity of symptoms. Kelley & DiBello (2007), for instance, suggest that some patients who had polio and recovered to a point that they did not require the use of orthoses might feel that they are regressing and take the use of orthoses as a sign of weakness.

Gender differences and age-related changes are two areas of interest for researchers as they both affect patient reports of pain. Werhagen & Borg (2010) concluded that pain was more often reported by female PPS patients compared to males. 88% of women and 49% of men reported pain in this study. These findings however do not prove that women are more likely to suffer from pain, as women in general tend to report pain more often than men (Werhagen & Borg, 2010). This is an important consideration in determining best treatment for an individual, as women may require an intervention that incorporates pain management (LaRocco, 2011). Interestingly, age also appears to affect perceived pain; increasing age correlates with lower perceived pain as found using the VAS (visual analogue scale). Quality of life differences also exist between young and old PPS patients, however these differences diminish over time as results for PPS patients approach those of a normal aging control group (Werhagen & Borg, 2010).

Post-polio myelitis is a condition that impacts everyone who suffers from it differently. One of the most important considerations is in the approach to assessment and treatment. Assessment must be thorough enough to rule out closely mimicking conditions. The involvement of a number of health care professionals representing a range of disciplines will ultimately ensure that the best treatments are undertaken to produce the most favourable results, including maintenance of muscle strength, endurance, and overall quality of life.

References


Post-Poliomyelitis Syndrome
Parkinson’s Disease

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PWR! Moves Certified Parkinson’s Therapist

Key Messages

• Parkinson’s disease is an idiopathic neurodegenerative condition of the midbrain affecting motor control, muscle tone, coordination, and gait patterns.

• This condition is not found exclusively in the elderly; young-onset Parkinson’s disease can occur before the age of 40.

• Symptoms of parkinsonism are associated with gait disorders, commonly characterized by shuffling steps, low walking speed, small stride length, reduced arm swing, rigidity in trunk movements, and freezing gait.

• Persons with a Parkinsonian gait and altered motor control are at high risk of falls and injuries arising from the falls. Prevention of falls is key, and a multidisciplinary team approach is ideal.

• Pedorthic interventions include: Footwear education and assessment for custom orthoses, custom-modified, or custom-made footwear. Exercises specific to the intrinsic muscles of the foot, lower leg, and ankle range of motion (ROM) should also be considered.

Keywords

bradykinesia, dopamine, gait disturbance, hesitation, neurodegenerative, rigidity, Romberg’s test, shuffling, tremors

Introduction

First termed “shaking palsy” in 1817 by British physician James Parkinson, in 2018 the condition is known as Parkinson’s disease, affecting more than 10 million people worldwide, making it the second most common neurologic condition after Alzheimer’s disease (Lewis, 2017). Parkinson’s disease (PD) generally develops in the elderly, with most cases occurring in people over the age of 50, and is also commonly termed idiopathic parkinsonism because it has no known cause; however, research shows that Canadians are being diagnosed at a younger age, with 5–10% showing symptoms of young-onset Parkinson’s before the age of 40, and 20% under the age of 50 (Parkinson Canada, 2017). Through early screening, the Unified Parkinson’s Disease Rating Scale (UPDRS) (Ramaker, Marinus, Stiggelbout, & van Hilten, 2002) is the gold standard assisting clinicians and researchers in detecting young-onset PD and male-to-female ratio statistics. From a male-to-female ratio perspective, males are one and a half times more likely to have PD than females (Parkinson’s Foundation, 2018). Although traditionally considered a non-genetic disorder, recent research suggests that there are several factors contributing to the disease which involve both genetic and environmental variables; around 15% of individuals with PD have a first-degree relative who has the disease (Samii, Nutt, & Ransom, 2004), and at least 5% of individuals with PD are now known to have forms of the disease that occur because of a mutation of one of several specific genes (Lesage & Brice, 2009). Certain environmental factors have also been associated with an increased risk of developing PD, including pesticide exposure, head injuries, living in the country, and farming (Maele-Fabry, Hoet, Vilain, & Lison, 2012).

Parkinson’s is a condition which attacks the nervous system, causing various levels of motor control deficits in individuals (Winogrodzka, Wagenaar, Booij, & Wolters, 2005). The classic picture of parkinsonism includes bradykinesia (slow movements), rigidity, difficulty initiating movements, and delayed postural corrections (Reeves & Swenson, 2004). Most of these Parkinsonian symptoms lead to poor balance, loss of coordination and gait disturbances, causing those with this condition to be at an increased risk for falls. As part of a multidisciplinary team, the pedorthist...
can play a significant role in helping persons with Parkinson’s disease. Assessing the need for custom-made or modified orthoses, and/or custom-made or modified footwear to improve base of support and ease of gait, increase range of motion (ROM), reduce muscle tension, and lessen fatigue, could prove beneficial. Performing any necessary offloading of plantar pressures of the foot, or relieving pressures against the shoe, should also be considered by the pedorthist. However, the role of the pedorthist is not only intervention; pedorthists may be among the first health care providers who can recognize early changes in postural and gait characteristics as “red flags” in those with early onset PD, and those who remain undiagnosed.

**Pathology**  
**Condition, causes, and impact on the patient**

Parkinson’s disease is a severe progressive neurodegenerative disorder (Winogradzka et al., 2005). The condition targets the neurons of the basal ganglia in the substantia nigra (a structure in the midbrain) responsible for producing a chemical neurotransmitter called dopamine. Released from the brain to direct movement patterns, dopamine commands the motor control centre (National Institutes of Health (NIH), 2011). In fact, it is the amount of cell loss in the substantia nigra which dictates the severity and determines the evolving stages of PD. Over the course of this condition, until the time of death, those with PD will suffer a loss of up to 50–70% of the neurons in the substantia nigra, as compared to a person unaffected by the condition (Davie, 2008). With the progression of PD, pathological changes within the brain’s nerve cells (mainly those involved in thinking and moving), are observed to be affected by deposits of proteins called Lewy bodies (Davie, 2008; Nilsson, Hariz, Wictorin, Miller, Foresgren, & Hagell, 2010). Except when Lewy body dementia (LBD) is present, these protein deposits are hallmarks of PD and are not found in any other neurodegenerative disease (Davie, 2008). Because of these abnormal deposits and the death of the brain cells, notable symptoms occur early on in the progression of Parkinson’s, causing those diagnosed with PD to suffer from muscular rigidity, tremors at rest (involuntary, rhythmic movements), and postural instability. Collectively, these characteristic symptoms of PD which disrupt primary motor patterns are termed “parkinsonisms” (Davie, 2008; Winogradzka et al., 2005). More subtle signs can also appear during the early stages of PD, and often go undetected: changes in handwriting, loss of facial expression, unilateral loss of arm swing, postural changes, reduced base of support when walking, and reduced sense of smell are among some of the “red flags” often associated with the early stages of this disease (Davie, 2008).

**Later stages**

In the later stages, those affected with PD may have hypophonia (abnormally soft speech from the weakened muscles surrounding the vocal cords), drooling (due to weakening of the muscles of the throat), and poor postural reflexes (Davie, 2008). Despite treatment with medications that replenish dopamine (Winogradzka et al., 2005), there still exists a steady loss of dopamine neurons; PD patients suffer from an unyielding deterioration in mobility and quality of life, particularly once bradykinesia, hypokinesia, dyskinesia, and dementia begin (Winogradzka et al., 2005). Bradykinesia is one of the most characteristic symptoms of PD; it is defined as reduced speed when initiating and executing a single movement and progressive reduction of amplitude, up to complete cessation, during repetitive simple movements. Bradykinesia responds well to dopaminergic medication and deep-brain stimulation (DBS) surgery (Lin, Dai, Xiong, Xia, & Horng, 2017). Degeneration of the dopaminergic system causes long-term, irreversible neurologic symptoms (Winogradzka et al., 2005). Some of the visible effects of this disruption of the primary motor patterns are notable postural disturbances and significant decline in both motor and cognitive tasks (Holmes, Jenkins, Johnson, Adams, & Spaulding, 2010).

**Non-motor complications**

There are many symptoms which may arise regardless of whether the person has their motor symptoms well under control with medication; these symptoms are not directly related to the dopaminergic pathways, but often related to side effects of PD medication. With the progression of PD, 60–98% of people experience nocturnal sleep disturbances, which are related to disease progression and levodopa medication intake. Because the presence of Lewy body proteins in the brains of those with PD is also consistent with those found in the brains of people with Alzheimer’s and Lewy body dementia, these three conditions and their symptoms are closely linked and crossover may occur, placing those with PD at high risk for developing memory loss and visual hallucinations (Alzheimer Society of Canada, 2011). In fact, psychosis, hallucination, and confusion can occur in up to 30% of those diagnosed with PD, often causing paranoia, agitation, and aggression toward others. Depression occurs in 50% of those with PD, and can develop at any stage of the disease (Davie, 2008).
The Patient Perspective

Gait disturbances

The symptoms of Parkinson’s are associated with gait disorders, characterized by shuffling steps, slow walking speed, small stride length, reduced arm swing, rigidity in trunk movements, and reduction in propulsion and retropulsion (Winogrodzka et al., 2005). During gait, it is believed that bradykinesia might affect the adaptation of a coordination pattern to changing walking speed, while rigidity has been found to affect single upper limb movements (Winogrodzka et al., 2005). Once the basal ganglia in the brain is impaired by the loss of dopaminergic innervation, gait disturbances can occur in two ways: episodic and continuous (Hausdorff, 2009).

- Gait festination (freezing gait) is considered an episodic gait disturbance. The nature of freezing gait is random and unpredictable, and can occur during any moment of the gait cycle (Hausdorff, 2009; Nilsson et al., 2010).

- Continuous gait disturbances are more consistent, such as a decreased stride length and increased gait variability (Hausdorff, 2009).

Freezing gait

The freezing of gait occurs in more advanced stages of PD and is one gait disturbance most highly correlated with falls (Nilsson et al., 2010). The term “freezing gait” is a common description among those with PD and adequately describes the following characteristic symptoms of gait (Tanioka, Kai, Matsuda, Inoue, Sugawara, Takasaka, Tsubahara, Matsushita, Nagamine, Tada, & Hashimoto, 2004):

- Start hesitation: difficulty in taking the first step at the beginning of walking and then stopping after taking only a few small steps (Nilsson et al., 2010; Tanioka et al., 2004)

- Destination hesitation: the state in which the patient’s feet appear to become “fixed” to the floor, not allowing them to move in any direction. This occurs primarily upon turning or when approaching a targeted destination (Nilsson et al., 2010; Tanioka et al., 2004)

- The inability to step forward, and consequently falling, during walking is typically triggered when in confined or crowded spaces and in time-sensitive situations such as crossing the road (Nilsson et al., 2010; Tanioka et al., 2004)

The effects of freezing gait on gait timing are primarily seen during the swing phase of gait. It has been shown that in patients with PD, “heel-to-toe” response time was about 2.4 times longer than in healthy subjects at the beginning of walking and freezing gait could be recorded as the difficulty in lifting the foot by the toes and characterized by a double-peak in gait pressures occurring at a short interval, indicating double landings” (Tanioka et al., 2004). In advanced stages of PD, when dopamine medication proves less effective, motor fluctuations cause more frequent disruptive gait compensation patterns. It has been observed in patients wearing a 24-hour portable gait rhythmogram device that when PD patients experience bradykinesia or instability, they tend to shift to a slower gait cycle, and during a bout of freezing gait, a shift to a faster gait cycle occurs (Mitoma, Yoneyama, & Orimo, 2010). Because of this “shifting” pattern, freezing gait is primarily responsible for most falls in those with PD, regardless of whether the patient is using a gait aid such as a walker equipped with brakes, because in states of panic, persons with PD cannot operate the brakes fast enough and are unable to follow an accelerating walker to regain stability (Tanioka et al., 2004). It is important to note that freezing gait is one of the most distressing symptoms for people with this condition, as it has a substantial negative impact on their overall perceived health (Nilsson et al., 2010).

High prevalence of falls

An 87% prevalence of falls has been reported in a study with persons newly diagnosed with PD who were involved in over a 20-year follow-up study; 35% of the reported falls also led to fractures (Hely, Reid, Adena, Halliday, & Morris, 2008). The possible injuries sustained from a fall can cause secondary immobility, depression, and reduced quality of life. Falls in patients with PD are caused by many factors. Motor dysfunction from PD creates balance and gait disorders, and disruptions in stepping patterns, as observed in freezing gait. Because of these motor deficiencies, patients with PD also need to be exceptionally aware of environmental risk factors such as slippery or uneven floor surfaces, loose rugs or heavy carpeting, poor lighting, and inadequate footwear. Another key factor is cognitive function, as persons with PD who are more severely affected by dementia are also at higher risk of falling due to the lack of dopaminergic innervation to the brain; the caudate nucleus, partially responsible for motor control, is the area most affected (Snijders, Nonnekes, & Bloem, 2010).
Fall prevention

Although balance deficits are resistant to dopaminergic medication, optimal doses of these agents may reduce the frequency of freezing gait episodes (Snijders et al., 2010). Due to the complex nature of fall prevention, a multidisciplinary team approach is preferable in preventing them; the involvement of family doctors and neurologists, increasing PD medication for dopamine-sensitive signs, stopping sedative drugs, tailored physiotherapy, occupational and speech therapy, and pedorthic interventions are among some of the options. Foot orthoses have been shown to increase sensorimotor function of the foot, ankle control, and sensorimotor feedback around the ankle, which can also help with poor balance and decrease the risk of falling (Spink, Menz, & Lord, 2008).

Common Treatment

Physiotherapy and exercise

Increasing overall mobility through regular exercise and/or tailored physiotherapy should be high on the list of priorities for those diagnosed with PD; by increasing muscle strength and flexibility, it has been proven that exercise in persons with PD results in improvement of postural stability and balance task performance (Snijders et al., 2010). In addition to improving gait, leg strength, and overall physical functioning in those with PD, exercise can also improve cognitive function and health-related quality of life (Reuter, Mehnert, Leone, Kaps, Oeschner, & Engelhardt, 2011). Some physiotherapy based strategies consist of cueing techniques and cognitive movement strategies; rhythmic, auditory, or visual cues such as listening to music during walking for exercise or using an electronic metronome to help with pacing, are some of the evidence-based techniques used to improve gait (Snijders et al., 2010). Some studies suggest that attentional strategies and visual cues may facilitate the normal stepping pattern (Morris, Huxham, McGinley, Dodd, & Iansek, 2001; Morris, Iansek, Matyas, & Summers, 1996). Visual cues can include activity stimulation such as stepping over small objects (typically placed in a stride sequence in front of the patient), or kicking a ball that lies in the way (to open up stride length). These simulations attempt to break the episodic gait disturbance, such as destination hesitation and freezing of gait. Examples of some attentional strategies include speech tasks such as numerical recitation or a simple monologue in which individuals with PD have been shown to “prepare themselves” prior to performing cognitive tasks in order to focus. This preparation, in turn, has been shown to stabilize the posture of those with PD gait disturbances beyond normal levels (Holmes et al., 2009). This may be why attentional gait strategies such as Nordic walking (walking with the use of rubber- or spike-tipped poles), has been proven to dramatically improve stride length, gait variability, and maximal walking speed levels in patients who practice the technique often and become proficient at it (Reuter et al., 2011). Nordic walking adds another dimension to gait, by further activating the muscles of the upper body and increasing the length of each step, resulting in an overall faster gait.

Nordic walking and spin cycling

Nordic walking, walking, and participating in a flexibility and relaxation training group are important activities for patients with PD. The overall results showed that all training programs provided benefits, but a remarkable 30% of the PD patients became pain free post Nordic walking (NW) training, and for those who still reported joint and muscle pain after assessment, the intensity of their pain had decreased more than those in the other groups. Furthermore, patients in the NW group showed the most improvement in posture, postural stability, and balance. The poles in NW represent external triggers to cue the patients to increase the involvement of the more affected arm/hand which may have been responsible for an improvement in hand agility in everyday tasks (van Eijkeren, Reijmers, Kleinveld, Minten, Bruggen, & Bloem, 2008). The NW group was also the group with the greatest perceived exertion, which translated to better concentration and memory than the other groups (Reuter et al., 2011). If Nordic walking or walking as exercise is difficult, or the fear of falling is too great, particularly for those with freezing gait, the skill of cycling proves to be exceptionally preserved in those with PD. When cycling, the bicycle’s rotating pedals may act as an external pacing cue which seems to trigger the motor-control mechanisms involved in cycling which seem to be less affected by the disease than the patterns involved in gait (Snijders & Bloem, 2010).

Current evidence-based rehabilitative techniques for persons living with PD

Strength, Power & Agility Training

A study published in 2016 reveals multiple levels of improvement from one repetition at maximum strength focused on peak power output (Signorile & Ni, 2017); the results of this study support the theory that targeted high-speed training intervention (light weight/fast repetitions) can improve strength.
and power in Parkinson’s patients while reducing bradykinesia and providing positive outcomes to activities of daily living, mobility, social support and overall quality of life. In addition to using basic open and closed kinetic chain strength exercises (such as bicep curls, chest press, seated calf raises and leg press), the patients in this study were also involved in agility and balance exercises using cones, agility ladders and ball drills to specifically target challenges associated with bradykinesia. This study demonstrates the benefits of applying strength and agility training to achieve “like” movements - where the goal is to break slow, rigid movements and use light weight to achieve maximal speed while improving strength.

High Intensity Treadmill Exercise
The effects of treadmill exercise four days per week at 80–85% of maximum heart rate have been recently studied along with moderate intensity treadmill exercise (60–65%) at the same frequency for a trial period of 6 months. The results of this study have shown a positive mean change in the gold-standard UPDRS (Unified Parkinson’s Disease Rating Scale – see subsection III) from 0.3 (95% CI, −1.7 to 2.3) in the high intensity group, compared with 3.2 (95% CI, 1.4 to 5.1) in the moderate intensity group (Schenkman, Moore, Kohrt, Hall, Delitto, Comella, Josbeno, Christiansen, Berman, Kluger, Melanson, Jain, Robichaud, Poon, & Corcos, 2017). While several factors including patient compliance for frequency of exercise and non-severe adverse musculoskeletal events were challenging to this research, the outcomes prove that high-intensity treadmill exercise is feasible and produces meaningful clinical benefits when performed safely.

Dance Lessons
Recent research at the Montreal Neurological Institute and Hospital as well as McGill University has found that PD patients who learn to dance the Argentine tango can improve balance and functional mobility, and may have modest benefits upon cognition and fatigue in Parkinson’s disease (Romenets, Anang, Fereshtehnejad, Pelletier, & Postuma, 2015). The study looked at whether a social and physical activity linked to music, such as tango, could have possible therapeutic value for PD patients who suffer from motor and gait dysfunctions, as well as from non-motor symptoms, such as depression, fatigue and cognitive degeneration. Because Tango requires specific steps that involve rhythmically walking forward and backward, this form of therapy can address specific balance-related issues, especially for freezing of gait and prevention of backward falls. Tango requires memory, focus, and multitasking to stay in rhythm with the music and maneuver around others on the dance floor – a fun, unique, and social way of staying active while improving balance and function.

Virtual Reality Training
Perhaps one of the most recent developments in PD rehabilitation techniques is using a type of virtual reality called “augmented reality”, which focuses on both cognitive and motor function training. Integrated augmented reality (IAR) is one type of virtual reality where virtual and real-world (RW) objects are integrated into one environment (Boucher, Roberts, Ayala, Katchabaw, & Jog, 2013). The goal of augmented training research is to take PD patients through activities of daily living (ADLs) tasks in a controlled environment, during which performance of these tasks can be monitored and evaluated. In performing this type of cognitive–motor rehabilitation on a regular basis, researchers are attempting to determine if the positive results they are measuring in the lab can translate to ADLs outside of a laboratory setting. A group of researchers at Western University in London, Ontario, effectively set up augmented reality training once a week for three weeks for a group of PD patients – one task was watering a plant in a living room environment, and the other choosing a series of cereal boxes in the aisle at a grocery store. The goal of this research-based rehabilitation was to prove that in order to best transfer gait and other movement skills acquired in a virtual environment to everyday activities, participants would ideally be able to move freely within the virtual environment (unlike other VR research using treadmills or desktop based systems (Mirelman, Maiden, Herman, Deutsch, Giladi, & Hausdorff, 2011). Because people with PD find it challenging to focus on the most relevant sensory information in a given environment (Cools, Rogers, Barker, & Robbins, 2010), the IAR system may have “overloaded” the sensory information, distracting the patients from the working memory task, resulting in an effect on the ability of the PD group to accurately fill the grocery baskets with even better results than the control group (Boucher et al., 2013). These and other ongoing studies can be followed at the Movement Disorder Centre in London, Ontario, as can Dr. Mandar Jog’s PD research (MDC, n.d.).

Voice Treatment
Recent therapy gaining momentum in North America also includes an intervention researched by speech
therapists; specific voice control exercises and precise over-exaggerated arm and body movements have been shown to reset motor patterns and improve hypophonia in those affected by PD, as well as addressing the needs of similar conditions such as stroke, multiple sclerosis, cerebral palsy, and Down syndrome. A group of North American speech therapists from Lee Silverman Voice Treatment (LSVT) Global Inc., have researched and designed a program to train physical and occupational therapists on a derivative of a speech treatment that is directed at improving the whole-body movements of persons with PD (LSVT Global, 2011). LSVT LOUD is based upon a specified form of speech therapy to re-train lost voice control and amplitude, and has become a standardized, research-based speech treatment protocol with established efficacy for PD (Fox, Ramig, Ciucci, Sapir, McFarland, & Farley, 2006). Recently LSVT Global launched the LSVT Companion, a computer program that allows PD subjects to independently progress through treatment sessions at home as an independent therapy option. When PD subjects do not meet their target goals (vocal loudness, pitch, duration), the LSVT Companion provides feedback to encourage them to appropriately adjust the characteristics of their phonation, e.g., louder, longer, higher, or lower (Tsanas, Little, Fox, & Ramig, 2014). LSVT BIG follows a specific protocol of numerous movement patterns: seated arm thrusts, rotational arm extension, step and reach backwards with a bowing motion forwards, and a standing lunge with arm extension are examples of multidirectional, sustained, and repetitive movement patterns common to the program. Some examples of functional movement patterns include rolling, supine to sitting position, walk and turn, and sit to reach. The focus of these movement patterns is to create as “BIG” a movement as possible with maximum magnitude, speed, and extension to recalibrate and maximize motor output (LSVT Global, 2011; PWR!, 2018).

Deep brain surgery

Deep brain stimulation (DBS) surgery is currently the only acute intervention that results in rapidly progressive reduction in the severity of dyskinesia in PD patients (Delrobaei, Baktash, Gilmore, McIsaac, & Jog, 2017). Implantation of deep brain stimulators is reserved for those whose gait and balance deficiencies are still responsive to levodopa medication pre-operatively; stimulus of the internal globus pallidus shows the most promise for long-term improvements in gait and balance (Snijders et al., 2010); however, the real challenge is within the actual pre-programming of deep brain stimulators, because currently the devices are preprogrammed based on subjective assessments of PD symptoms such as the severity of dyskinesia pre-operatively. At this time, there are several studies using cost-effective wearable technology as a means of a postoperative home monitoring system to effectively gather real-time data on the severity of the patient’s dyskinesia. Using these wearable motion capture systems with the developed software could allow health professionals to view accurate animations developed from the sensor data reviewed, which can be monitored remotely by the treating physician (Delrobaei et al., 2017).

Clinical Testing for Pedorthists

For medical professionals, the diagnosis of PD remains clinical, based upon recognition of the previously described characteristic physical findings. The recognition of early features, signs, and symptoms is essential to the early detection of this disease. While some but not all characteristic features of bradykinesia, resting tremors, rigidity and postural instability may be present, most symptoms of PD are found asymmetrically (Davie, 2008). Although not a completely accurate scientific measure of motor neuron dysfunction, the Babinski sign is commonly used to complete a neurological examination by neurologists and clinicians alike. A positive Babinski test occurs when the great toe dorsiflexes and the lesser toes fan out when the clinician applies a strong upward stroke to the lateral plantar aspect of the foot from heel to metatarsals with a blunt instrument. Another simple test, and possibly a more reliable method of evaluation, is comparing the slowness of foot tapping on each foot (Miller & Johnston, 2005). Pedorthists can use both the Babinski test and foot tapping test to help recognize some early signs of PD; the implementation of objective screening tests such as these places the pedorthist in a position, clinically, to determine if the patient should be referred to see a neurologist for further examination. The Romberg test is used by physicians in neurological examinations, testing the body’s sense of positioning (proprioception), which requires healthy functioning of the dorsal columns of the spinal cord. The exam requires the patient to use at least two of the three senses (proprioception, vestibular function, and vision) to pass the test. A patient who has a problem with proprioception can still maintain balance by using vestibular function and vision. During the testing, the patient stands upright with eyes closed, and a loss of balance is interpreted as a positive Romberg sign (Reeves & Swenson, 2008).
Clinical Gait analysis – gait asymmetries in PD

Although symptoms of PD are extremely varied, gait mechanics are one of the most affected and observable, because patients with PD exhibit gait characteristics that are markedly different from normal heel-to-toe walking. In Parkinsonian gait, to compensate for postural imbalances, patients generally show a trend towards higher relative loads in the forefoot; normal gait mechanics are replaced by a flat foot strike (where the entire foot is placed on the ground at the same time), or in the more advanced stages of the disease, the toe-to-heel walking pattern (where the toes touch the ground before the heel) is common (Kimmeskamp & Hennig, 2001). Whereas most research has been on studying the anterior–posterior movement changes in PD, recent research using multiple plantar foot sensors has been able to track pathological changes in the lateral–medial direction; this might be because the locomotion toward the medial foot can help the PD patients to keep balance (Ren, Karahan, Chen, Luo, Geng, Bayard, Bringas, Yao, Kendrick, & Valdes-Sosa, 2017). During a patient’s gait assessment, Pedorthists can use this information make note of possible PD red flags, particularly in the undiagnosed population, and refer the patient for further testing.

Misdiagnosis

Many of the typical early features of PD overlap with other signs and symptoms of conditions associated with aging such as developing an essential tremor, postural instability, dementia, and Alzheimer’s disease. Very few medical diagnostic testing methods have been found scientifically relevant to the detection of PD; this may be why many of those diagnosed with the condition have later been found to have been misdiagnosed, while others in the community remain undiagnosed (Schrag, Ben-Shlomo, & Quinn, 2002). Unfortunately, without a readily available biological marker for Parkinson’s disease, the only certain means of diagnosis at present is the finding of depletion of brain stem pigmented neurons with Lewy bodies at autopsy.

Although the typically prescribed anti-Parkinsonian drugs have the ability to temporarily alleviate many motor related symptoms, it has also been shown that long–term use of these drugs can produce disabling side effects and can actually induce dyskinesia (Davie, 2008; Meara, Bhowmick, & Hobson, 1999). Although most physicians are cautious by delaying dispensing anti-Parkinson’s medication and are careful about progressing the doses, if misdiagnosed, a patient taking dopaminergic drugs will be at risk for impaired cognitive function and postural hypotension. Such risks underline the importance of accurate early diagnosis for effective long–term treatment and management with the support of a multidisciplinary team (Meara et al., 1999).

Observations

Depending on the stage of PD and variations in treatment protocols (whether or not the patient is receiving medication, exercising, stretching, etc.), the following are some of the observations you may see during an assessment with a person with PD:

- Foot and leg cramping – Muscular rigidity, causing cramping and stiffness in the feet and legs, is quite common.

- Hammer toes and progressive dropping of the transverse arch – As balance decreases and stride length is shortened, there is a tendency to try and grip the ground with the toes for balance and support. With repetitive toe gripping, the extensor digitorum longus muscle loses its ability to maintain a compressive force to straighten the proximal and distal interphalangeal joints, causing the proximal interphalangeal joint to plantar flex (Michaud, 1997). Plantar flexed metatarsophalangeal joint (MTP) heads and toe tips may become painful with increased mechanical pressures; dorsal prominence of the proximal joint may also cause rubbing and pressure points against the shoe. Checking appropriate footwear fit, particularly in the toe box, is highly recommended.

- Peripheral edema, dark and brittle nails – Possibly due to poor circulation and/or lack of mobility and exercise.

- Weak musculature and excessive tone – Excessive muscular tone and rigidity and, for most persons with PD, being forced to adapt to a more sedentary lifestyle, causes a loss in muscular strength. Loss of strength can be seen primarily in a decreased ability to invert the calcaneus upon weight-bearing heel raise test and/or poor dorsiflexion (ROM and/or muscular endurance) due to lack of tibialis anterior strength causing toe drag in gait. Poor hip stability and a possible Trendelenburg gait may also be observed.

- Changes in joint ROM – with increased muscle tone, rigidity and major compensatory motor patterns, joint range of motion, particularly at the hip, ankle mortise, and transverse arch may be observed.
• Unsteady gait – Patient may demonstrate a shuffling gait, shortened stride length, difficulty in lifting the foot in propulsive phase, poor swing phase and recovery, freezing gait, and a fear of falling.

• Flexed at trunk – A forward flexed position causes changes in the patient’s centre of gravity causing decreased stability, as well as a possible increase in tone and spasticity of the hamstring and posterior compartment.

• Possible leg length discrepancy (LLD) – Considering that PD is primarily a unilateral disease, it may not be uncommon to find a notable LLD from compensatory gait mechanics, use of a cane, and possibly from a greater loss of joint rage of motion and flexibility on the affected side.

Custom-made orthoses

For persons with PD, custom-made orthoses should be considered to help with weight redistribution, to increase base of support, and to aid with balance. Other benefits include reduction of muscle fatigue and increase in shock attenuation. Most often, those with PD will benefit from a lightweight/flexible and accommodative EVA orthosis with a deep heel cup and higher sidewalls for added stability upon heel strike, and to increase sensorimotor function of the foot and ankle. The need for forefoot and metatarsal arch support should be addressed particularly in cases where patients are “clawing” the ground for support when feeling unstable, particularly if the overuse of flexor or extensor digitorum brevis, or excessive transverse arch motion, has created a metatarsalgia (Michaud, 1997). Upon further investigation, dorsal or plantar forefoot pain, neuroma, pain with pressure under the metatarsal heads, tendinitis, or capsulitis may often be present. Offloading with a metatarsal pad and/or sulcus crest to support the metatarsals, PIP, or DIP may be necessary, particularly if the toe “clawing” has created a fixed hammer toe deformity; excavation in the forefoot cushion of a custom-made orthosis may also be necessary to decrease pressure and friction on lesser toe deformity toe tips.

Proper footwear

Several studies have assessed the influence of footwear, or lack thereof, on the incidence of falls in the elderly. In general, going barefoot or in stocking feet, wearing shoes with increased heel height, or wearing shoes with a small base of support have been shown to increase the risk of falling, and can be assumed to be equal risk factors for those with PD. Other factors which have been shown to affect balance are heel collar height, fixation, and the slip resistant properties of the sole (Spink et al., 2008).

Although this research has been thoroughly examined in the elderly, the fact that those with parkinsonisms and gait disturbances suffer from muscular rigidity, shuffling steps, shortened stride length, and episodes of freezing gait, the importance of proper shoe fit and features, should be highlighted to a patient with PD by the pedorthist:

• Light weight – Easier to pick feet up off the ground and lessens fatigue.

• Wide base of support – Avoid midfoot cut-outs and heels to ensure the maximal amount of shoe-to-ground interface for increased balance and control.

• Rocker sole and beveled heel – Creates better ease of movement including the following benefits: easier transition from heel-to-toe, tibialis anterior does not have to work as hard upon heel strike, provides better shock attenuation, and the forefoot rocker helps to reduce pressure under the metatarsal arch. Also, it has been shown that on dry household surfaces, a beveled heel may also enhance grip during stepping because it provides a greater coefficient of friction than a shoe with a square-edged heel (Menant, Steele, Menz, Munro, & Lord, 2008).

• Good torsional stability – Board lasted to provide maximal amount of support to the rocker. Also, firmer footwear may also have a positive effect on balance (Menant et al., 2008).

• High heel collar – Higher cut footwear provides more sensorimotor feedback around the ankles because it involves the skin receptors of the ankle and creates a more supportive feel (Menant et al., 2008).

• Slightly raised heel – To reduce tension on tight muscles in the posterior compartment.

• Extra-depth and rounded toe box – PD patients have a tendency is to grip the ground with the lesser toes when feeling unbalanced, therefore a deep, wide, rounded toe box will allow the MTPs to stabilize the foot and also provide a better fit for any existing hammertoes and mechanical pressures.

• Appropriate outsole for walking conditions – Because foot clearance is low and tibialis anterior is typically weak, it is important that the shoe tread is not too heavy for indoor walking, which creates risk of catching the toe on carpeted surfaces.
Velcro closure or elastic laces – If the person is unable to properly secure and adjust the shoe due to muscular rigidity and tremors in the hands.

Modification to footwear

The following footwear modifications should be considered for those with greater needs than what off-the-shelf footwear can meet:

Medial/lateral flare (buttress) – To increase overall ground contact for added stability and support. A lightweight EVA material added to the midsole and the upper on the medial and/or lateral aspect of the midfoot. A shoe with a leather upper is recommended to enhance existing support without bulking up the shoe with extra weight.

External heel lift – If unable to provide an adequate heel lift internally for tight posterior compartment muscles, or if a significant LLD exists. Be careful not to increase heel height more than 4.5cm, so as not to shift the centre of mass too far forward and potentially cause an adverse effect on balance, postural sway, and plantar pressures (Menant et al., 2008).

Shoe stretch or spot stretch – To accommodate lesser toe deformities causing areas of pressure against the shoe’s upper.

Toe slider – Adding a hard polymer modification under the toe to help with any “toe catching” and excessive shoe wear when lack of tibialis anterior strength in swing phase is present. Be cautious not to place slider too proximal, or the slider could create an unsteady surface and be a further risk to falling.

Custom rocker sole – A rocker sole may be added to the existing footwear’s midsole to enhance the rocker effect of the midsole as well as providing additional cushioning for shock attenuation.

Custom-made footwear – In extreme cases where nothing over the counter fits or provides enough support, or perhaps where a foot deformity is present, then custom-made footwear may be a suitable option.

Foot, ankle, and lower leg strength and range of motion

Although generally overlooked, some research shows that exercise can decrease the risk of falling by improving the patient’s balance and strength. This notion could carry over to the PD population because they are found in a higher risk group for fall due to lack of motor control. Research shows that postural imbalances are highly correlated with falls (Snijders et al., 2010). It has been proven that toe grasp training activates the mechanoreceptors of the plantar foot and the muscular reaction of the lower limb, which leads to an improvement in postural adjustment. One study demonstrates that by training toe plantar flexor strength (gathering a towel with a weight on it and passing a bean bag with the toes) 10 minutes a day, three times a week, over a period of eight weeks, there exists an improvement in spontaneous postural sway in the tested elderly population (Kobayashi et al., 1999). Ankle strength and range of motion should also be tested and a regular exercise program considered since reduced ankle flexibility has also been associated with falls (Spink et al., 2008).

Key Considerations

1. Be aware of the red flags in the initial stages of PD; an accurate, early diagnosis is important for effective long-term treatment and management with the support of a multidisciplinary team (Meara et al., 1999). Some of the subtler signs include changes in handwriting, loss of facial expression, unilateral loss of arm swing, postural changes, changes to midfoot-forefoot striking, medial centre of plantar pressures, reduced base of support when walking, and reduced sense of smell (Davie, 2008; Ren et al., 2017).

2. Freezing gait is a common and frustrating occurrence for those with PD and is highly correlated with falls. Tailored physiotherapy programs focused on verbal or visual cueing, recalibrating, and maximizing motor output with programs such as LSVT BIG, or external/attentional strategies such as Nordic walking or treadmill training, are some of the recent evidence-based rehabilitative techniques creating much attention and producing results.

3. Pedorthic goals specific to the needs of most patients with PD include:

- Improving the patient’s base and ease of gait
- Increasing range of motion and reducing muscle tension and fatigue
- Increasing sensorimotor function of the foot and ankle
- Offloading of plantar pressures of the foot or against the shoe
- Referring out to neurologists or other specialists for testing when early onset PD symptoms are observed
• If practising in the U.S. or Canada, you may find some LSVT BIG/PWR! Moved trained therapists as a referral source at http://www.pwr4life.org/professional-directory/

4. The use of lightweight walking shoes with a wide base of support and a deep toe box, combined with lightweight/ flexible accommodative EVA orthoses with a deep heel cup and higher sidewalls is the most commonly recommended pedorthic intervention to improve stability and sensorimotor function, and to lessen muscle fatigue.

References


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Multiple Sclerosis

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Key Messages

- Multiple Sclerosis (MS) is an autoimmune disease effecting the central nervous system (CNS).
- Canada has the highest incidence of MS in the world.
- The cause and effective treatments are largely unknown.
- Each case is unique, affecting individuals differently depending on where CNS lesions are located.
- Treatment must be tailored to the patient’s individual presentation.

Keywords

ataxia, autoimmune disease, balance, contracture, fatigue, myelin lesions

Introduction

MS is an autoimmune disease that attacks the nervous system, specifically the myelin sheath around the axons of neurons, in the CNS. The cause is unknown, but many correlations have been identified. MS is much more common in higher latitudes and amongst people of northern European descent. Canada has the highest incidence of MS in the world with over 100,000 people living with the disease (Multiple Sclerosis Society of Canada [MSSC], 2018). It occurs twice as often in women as it does in men. It is most often diagnosed in individuals between the ages of 25 and 35 years old, but can occur rarely in children and people over the age of 50 (Porter, 2005).

The effects of MS can be unpredictable depending on where the specific lesions are located. It can result in pain, weakness, mobility issues, vision problems, depression, and cognition issues (MSSC, 2018). Pedorthic intervention must be tailored to each patient’s individual presentation, but most commonly is focused on improving balance, proprioception, and avoiding falls.

Pathology

Multiple sclerosis is derived from the term multiple sclerae or scars that are seen on the white CNS tissue in MRI imaging. It is an auto-immune attack on the fatty tissue called the myelin sheath of neurons. These lesions affect the nerve’s ability to conduct electrical signals. It mainly affects white matter nerves, including the optic nerve, brain stem, basal ganglia, and spinal cord (Compston & Coles, 2002).

The disease can manifest in different ways depending on the individual. Most commonly it results in weakness, loss of eyesight or vision effects, loss of bladder and bowel control, difficulty in memory and cognition, difficulty with coordination and balance, ataxia (non-specific gait abnormality due to a lack of voluntary, coordinated movements), spasticity, and depression (Ziemssen, 2011; Khan, & Smith, 2013).

In terms of mobility, one-third of people diagnosed with MS require a walking aid within 10 years of initial diagnosis; this rises to 85% after 30 years. The greatest factors limiting mobility are fatigue, weakness, spasticity, impaired balance, ataxia, and tremor (Kremenchutzky, Rice, Baskerville, Wingerchuk, & Ebers, 2006).

Foot pathology changes are caused by muscle spasticity and contracture, resulting in muscle retraction and a cavus foot structure, coupled with poor pressure distribution. Neuralgia can increase sensitivity causing pain, while neuropathy can place individuals at risk of ulceration (Khan & Smith, 2013; Freeman 2001). Conversely, muscle weakness may result in drop foot, with or without equinus contracture, adult acquired flat foot, and increased risk of trips and falls.

Differential Diagnosis

Diagnosis is reached by evaluating the clinical presentation of initial signs and symptoms, which are then confirmed by MRI. Early diagnosis can be difficult as the early symptoms can be similar to other diseases. Early symptoms (weakness, vision
problems, difficult in memory and cognition, poor coordination, and balance) will be episodic and self-limiting. In fact, most early signs and symptoms will not result in the patient visiting the doctor and it is only in hindsight that it is realized these are early attacks.

The condition is broken down into 4 major subgroups: (Kremenchutzky et al., 2006)

1. Clinically isolated syndrome (CIS) – This is the first attack, and rarely diagnosed before a client is in relapsing–remitting multiple sclerosis (RRMS).

2. Relapsing–remitting multiple sclerosis (RRMS) – This subgroup is composed of unpredictable relapses, followed by years of remission. This accounts for 80% of initially diagnosed cases of MS.

3. Primary–progressive multiple sclerosis (PPMS) – This subgroup is characterized by progressive symptoms of MS after the initial onset, with no periods of remission. This subgroup is fairly rare, and only accounts for approximately 5% of cases.

4. Secondary–progressive multiple sclerosis (SPMS) – This is the progression after RRMS. The periods of remission become shorter and less frequent, until there are no periods of remission of symptoms.

The Patient Perspective

Multiple sclerosis is highly associated with depression. This may be in part due to the emotional distress attached to the diagnosis. MS is often diagnosed at a fairly young age, drastically changing the patient’s outlook for the future. Progression of the disease is unpredictable and often the patient’s expectations are actually worse than the reality. Patients feel a loss of control and connection as the diagnosis can affect their interactions with friends and family members (Freeman, 2001).

Changes in nerve function can cause neuralgia, resulting in increased pain and/or a change in sensory response to mundane stimuli. Vision and balance problems reduce a patient’s ability to perform activities of daily living. It is notable that patients will often complain of numbness on the skin, but are unaware of joint proprioceptors becoming “numb”, affecting their ability to balance and ambulate. For example, a clinical observation of this is a patient who cannot walk well in the dark or stumbles at night, but during the daytime, when they can see, can walk very well (Everydayhealth.com, 2008).

Ataxia, spasticity, and muscle weakness restrict mobility. These will manifest differently in each individual depending on what part of the CNS is affected with lesions. Due to the episodic nature of attacks, patients can develop joint contractures during periods of illness and inactivity. For example, contractures can restrict the ability of patients to straighten their knees beyond 15 degrees of flexion. Consequently, patients are required to stand and ambulate with their knees constantly flexed, resulting in rapid muscle fatigue. In a similar case, hip flexor contracture can force a forward lean, resulting in the inability to get the center of gravity over the ankles. This can get into a vicious cycle, in which early onset fatigue leads to inactivity, inefficient walking, and contracture, which further contributes to the early onset of fatigue (Everydayhealth.com, 2008).

Common Pedorthic Treatment

Treatment for MS is a long-term (rest of life) measure. While their interactions with various medical professionals will be relatively transient, patients and their caregivers must take on self-management. As there is very little evidence-based treatment, patients must make their own treatment decisions. The progressive but unpredictable nature of the disease can result in changes that even the patient will not notice. All treatment measures will require regular re-evaluation.

Common treatments include drug therapy, physiotherapy, and mobility aids. Few drug therapies have been clinically proven effective to aid in mobility, and medications are expensive and can have many side–effects. Physical therapy is a well–accepted and anecdotally effective treatment. Due of a vacuum of reliably safe, effective treatments, many patients seek alternative therapies including acupuncture, reflexology, cannabis, hyperbaric Oxygen, bee venom, and infecting themselves with hookworm (Helmby, 2005).!

One role of a pedorthist in treatment is to improve the safety and comfort of activities of daily living and to ultimately facilitate the patient’s ability to perform other physical therapy. These both can have considerable impact on the mental well–being of the patient. Pedorthists can address symptoms of muscle weakness through the use of bracing (AF0 or foot–drop braces for extensor weakness, ankle bracing for inversion/eversion control) (Bregman, Harlaar, Meskers, & de Groot, 2012), and custom foot orthoses (UCBL for posterior tibialis tendon dysfunction [PTTD], semi–rigid devices for arch...
support) (Ramdharry, Marsden, Day, & Thompson, 2006). Pedorthic use of shoe modification can improve balance through offering a wider base of support or skid plates to reduce toe catching and resultant trips and falls. Pedorthists can also educate the patient on the importance of supportive indoor footwear. A review of the literature indicates that orthoses and footwear can have both positive and negative impacts on balance and comfort (Hijmans, Geertzen, Dijkstra, & Postema, 2007). Soft orthoses will improve comfort while reducing proprioceptive feedback, a key educational point for this client base. Footwear with a firm sole can protect the foot against accidentally stepping on something or stubbing a toe. If the individual presents with contracture in the foot, orthoses may be used to reduce peak pressures and to improve ground contact and proprioceptive feedback (Rome & Brown, 2004). Use of toe separators, cradles, crest pads, and other devices may be helpful in reducing pain and pressure due to toe contractures and resultant pressure points.

There is little research surrounding the role of pedorthic care for people with multiple sclerosis. As each case presents different challenges, pedorthists can use their knowledge of existing pedorthic principles to guide a treatment plan.

References


Duchenne Muscular Dystrophy

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Key Messages

• Treatment of the child with Duchenne muscular dystrophy aims to slow down the progression of muscle wasting, to maintain independent standing and gait for as long as possible, and to manage the progressive loss of physical function and the development of contractures, respiratory insufficiency, and cardiac myopathy.

• Customized orthopaedic footwear and orthoses are used to manage ankle joint instability, to correct the varus/equinus position of the foot and ankle, and to increase the base of support of the foot. This can improve standing and gait capacity [level 4 and 5 evidence].

• Knee–ankle–foot orthoses (KAFOs) are used to prolong assisted walking and standing [level 3b, 4, and 5 evidence], which slows the progression of muscle wasting and delays the development of contractures at the knee and ankle joints [level 5 evidence].

• Ankle–foot orthoses (AFOs) are used at night, in combination with daily passive stretching, to correct/delay the development of tendo-achilles contracture [level 2b, 2c, and 5].

Keywords

balance, dystrophin, muscle degeneration, scoliosis

Introduction

Muscular dystrophy defines a group of disorders that result in the progressive destruction of muscle cells (Roland, 2000; Mercuri & Muntoni, 2013a). Muscular dystrophies vary widely with regard to the severity of symptoms, the age of onset, the pattern of inheritance, predominant muscle groups affected, and extent of involvement of other organ systems (Mercuri & Muntoni, 2013b). Duchenne muscular dystrophy (DMD) is the most common muscular dystrophy of childhood, with an estimated worldwide incidence that ranges from 1.7 to 4.2 per 100,000 individuals, and a specific incidence of 1.7 per 100,000 individuals in Canada (Theadom, Rodrigues, Roxburgh, Balalla, Higgins, Bhattacharjee, Jones, Krishnamurthi, & Feigin, 2014). In males only, the worldwide incidence of DMD is estimated at 1:3,500 live male births (Manzur, Kinali & Mutoni, 2008).

The natural clinical trajectory of DMD is well characterized, being predominantly defined by a relentless wasting of muscle that leads to severe disability and eventual respiratory insufficiency and death (Manzur et al., 2008; Garralda, Muntoni, Cunniff, & Caneja, 2006; Parker, Robb, Chambers, Davidson, Evan, O’Dowd, Williams, & Howard, 2005; McPhee, Lingappa, & Ganong, 2002; Sussman, 2002; Roland, 2000). Improvements in the general care of children and adolescents with DMD, including the use of glucocorticosteroid treatment, non-invasive ventilator support, and the management of cardiomyopathy and scoliosis, have significantly improved clinical outcomes and survival of patients into their second or third decade (Manzur et al., 2008). Although novel therapies are being developed and evaluated (see Nowak & Davies, 2004, and Bogdanovich, Perkins, Krag, & Khurana, 2004), there is currently no cure for DMD.

Pathology

Classically, DMD is defined as a recessively inherited mutation or absence of the dystrophin gene on the X chromosome (Blake, Weir, Newey, & Davies, 2002; Nowak & Davies, 2004). However, the dystrophin gene has a high rate of spontaneous mutation, with approximately two-thirds of DMD cases being attributed to new mutations and with no positive family history at presentation (Roland, 2000; Manzur et al., 2008). These spontaneous mutations can lead
to a failure of early diagnosis of young children with symptomatic DMD (Bushby, Hill, & Steele, 1999).

Box 1. Levels of evidence to evaluate the effectiveness of therapeutic interventions

The Centre for Evidence-based Medicine (2009) defines levels of evidence to evaluate the effectiveness of therapeutic interventions as follows:

- **1a** – systematic review with homogeneity of randomized controlled trials (RCT) included;
- **1b** – individual RCT with a narrow confidence interval of outcomes;
- **1c** – all–or–none study;
- **2a** – systematic review with homogeneity of cohort studies included;
- **2b** – individual cohort study or lower quality RCT;
- **2c** – outcomes research or ecological study;
- **3a** – systematic review with homogeneity of case control studies included;
- **3b** – individual case control study;
- **4** – case series and poor quality cohort and case control studies; and
- **5** – expert opinion without explicit critical appraisal or based on physiology, bench research, or “first principle”.

With regard to function, dystrophin is a large cytoskeletal protein that acts as a molecular bridge between the contractile apparatus inside the muscle fibre (more specifically the actin cytoskeleton) and the extracellular matrix of the muscle cell (Ervasti & Campbell, 1993a, 1993b; Pasternak, Wong, & Elson, 1995; Silverthorn, 2004; Weller, Karpati, & Carpenter, 1990). In addition to being essential for force transduction, this molecular bridge enhances the stiffness of the muscle cell membrane and redistributes the stress that develops across the cell membrane during a muscle contraction over a wider area of the cell (Blake et al., 2002; Pasternak et al., 1995; Bloch & Gonzalez–Serratos, 2003; Ervasti, 2003). In the absence (or with a severe reduction) of dystrophin, the muscle membrane becomes fragile and susceptible to contraction–induced injury, with its regenerative potential progressively depleted under repeated cycles of degeneration and regeneration (Petrof, Shraget, Stedman, Kelly, & Sweeney, 1993; Sussman, 2002; Manzur et al., 2008). The weakened muscle fibres become abnormally permeable, allowing leakage of intracellular contents, including creatine kinase, which triggers a strong inflammatory response that results in severe muscle weakness, fibrosis, and atrophy, and further compromises muscle function (Sussman, 2002). Extracellular calcium, enzymes, and signalling ions also enter the muscle cell, causing intracellular degradation and eventual cell death (Manzur et al., 2008; Moen, Baatsen, & Marechal, 1993; Sussman, 2002; Weller et al., 1990).

Clinical Diagnosis

The diagnosis of DMD is based on a systematic evaluation of the child. It considers the family history, findings of the physical and laboratory examinations, muscle tissue histology, the child’s developmental progression (and regressions) in motor performance and functional ability, and genetic testing as indicated (Scott, Goddard, & Dubowitz, 1982; Sussman, 2002; Bushby et al., 2005).

In the absence of a clear family history, late development of independent ambulation, the emergence of an abnormal gait pattern early in childhood, and physical clumsiness are common concerns that lead parents to seek medical consultation (Read & Galasko, 1986; Sussman, 2002). Signs of proximal muscle weakness are observable as the child tries to run, jump, climb stairs, and rise from the floor (Bushby, Bourke, Bullock, Gibson, & Quinby, 2005).

Clinical course of the physical impairments of DMD

DMD follows a fairly typical clinical trajectory. It is defined by an unremarkable history of early child development with walking first achieved at a median age of 18 months (Parker et al., 2005). As muscle wasting progresses, so does the loss of functional abilities (Roland, 2000; Parker et al., 2005). Moreover, the greater relative weakness of proximal over distal muscles leads to a characteristic evolution of observable clinical signs (Manzur et al., 2008), including frequent falls, progressive gait impairments such as walking on toes and waddling gait, and difficulty running and rising from the floor (Gower’s sign). Typically, patients are confined to wheelchairs by the age of 12 years. The loss of ambulation is a turning point in the clinical course of DMD, with a rapid progression in the development of muscle contractures, fixed skeletal deformities, and scoliosis, as well as the development of secondary respiratory complications (Rodillo, Fernandez–Bermejo,
Evidence-based Recommendations for Intervention

The goals of the inter-professional care of the child with DMD are to enhance the child’s level of participation in activities of daily functioning and physical activity, to slow the development of disease-associated complications, and to limit their consequences on the child’s health, function, and overall quality of life (Bushby, Finkel, Birnkrant, Case, Clemens, Cripe, & Constantine, 2010; Sussman, 2002; Roland, 2000). In the early course of DMD, the role of the health care team, including pedorthists, is to prolong ambulation (Vuillerot, Girardot, Payan, Fermanian, Iwaz, de Lattre, & Berard, 2010).

The use of deflazacort, a glucocorticosteroid, can improve muscle strength and prolong the period of ambulation, although its use is associated with side effects (Aschenbrenner, 2017). The clinical benefits of deflazacort have been reported in level 1b, 2b, and 4 studies, with the evidence summarized in Dubow and Meyers (2016). In combination with the use of deflazacort, orthopaedic management aims to prevent (and to correct as possible) skeletal deformities related to the progressive loss of muscle strength and extensibility, and to enhance functional independence (Bushby et al., 2010; Sussman, 2002). As part of the orthopaedic management, knee–ankle–foot orthoses (KAFOs), ankle–foot orthoses (AFOs), and customized footwear are prescribed with the goal of providing extra stability at the hip, knee, and ankle to prolong standing and walking ability (Garralda et al., 2006).

Use of orthopaedic footwear and orthoses to prolong independent standing and walking

Early in the course of the disease, as impairments in posture control and gait emerge, the use of orthoses (KAFOs and AFOs) is not recommended as they can lead to the development of an equinus deformity of the foot and ankle, hinder gait, and restrict the organization of functional compensations in posture and gait for the progressing weakness (Eagle, 2002; Bushby, Finkel, Birnkrant, Case, Clemens, Cripe, Kaul, Kinnett, McDonald, Pandya, Powsky, Shapiro, Tomeszko, & Constantine, for the DMD Care Considerations Working Group, 2010). During this early phase, customized footwear, including the use of customized orthoses, is useful to manage ankle joint instability and to correct the varus/equinus position of the foot, as well as to improve comfort (Bakker, de Groot, de Jong, van Tol-de-Jager, & Lankhorst, 1997; Schaars & Postema, 1993).

Impairments in lower extremity function, posture, and gait

The impairments in lower extremity function, posture, and gait in DMD are progressive, arising from the effects of muscle weakness, muscle shortening, and joint contractures on the biomechanics of posture control and gait. Early in the disease process, typically around the age of four years, a wide-base gait emerges to increase stability as a compensation strategy for proximal weakness around the pelvis. With progression of proximal muscle weakness, a lateral lurch emerges during ambulation. This lurch consists of a lateral shift of the body’s centre of mass over the stance leg to lower the mechanical demand on the hip abductor muscles. The child also experiences increasing difficulty rising from the floor, running, and stair climbing. A stereotypical pattern for rising from the floor emerges, known as Gower’s sign, in which the child uses his hands/arms to climb up his legs to extend the trunk and hip against gravity.

As lower limb muscle weakness progresses to include the quadriceps, the knee is maintained in full extension throughout stance phase, with loss of the initial knee flexion wave during the weight acceptance phase of gait (Shamaei & Dollar, 2011). The loss of shock absorption during weight acceptance results in postural instability which further increases the demand on proximal muscles. A set of postural adaptations develops to maintain postural control in the face of increasing muscle weakness, including hyperlordosis of the lumbar spine, genu recurvatum, and an equinus position of the foot. These adaptations maintain the ground reaction force vector anterior to the axis of rotation of the knee and posterior to the axis of rotation of the hip, allowing the lower limb to maintain a position of extension in standing and walking despite extensive proximal muscle weakness (Khodadadeh, McClelland, Patrick, Edwards, & Evans, 1986). Therefore, an effective treatment plan to extend walking capacity should focus on optimizing muscle strength, preventing the development of lower extremity muscle and joint contractures, allowing compensatory strategies to emerge, and providing external support (such as orthoses and gait aids) for posture control and balance.
A lightweight, wide-soled shoe with variable upper closures for ankle stability and ankle equinus aid is recommended, with the addition of toe sliders as needed. Customized foot orthoses, including bilateral heel lifts and varus wedging, can provide a base of support to an equinus ankle and may therefore enhance stability. The research on the use of orthopaedic footwear and orthoses is scarce (only level 5 evidence available) with the main study (Bakker et al., 1997) being based on self-report questionnaires of rehabilitation specialists in the Netherlands regarding their prescription pattern for orthoses. Theoretically, as customized shoes and orthoses improve the base of support to the foot, it is reasonable to assume that they would also affect the profile of the ground reaction force and the progression of the centre of pressure during gait. Research to fully investigate the role of customized footwear is warranted.

Although the daytime use of ankle-foot orthoses (AFO) during the early phase of disease progression is usually not recommended, for the reasons previously described, AFOs can provide certain benefits which should be evaluated on a per case basis (Eagle, 2002). AFOs can prevent knee hyperextension (which protects the soft tissues at the posterior aspect of the knee) and also maintain the position of the foot in neutral or slight dorsiflexion, with limited plantarflexion range available. The resultant improvement in lower limb alignment during weight bearing can improve both lower limb kinematics during gait and ankle dorsiflexion moment during the swing phase (de Souza, Figueiredo, de Jesus Alves de Baptista, Aldaves, & Mattiello-Sverzut, 2010). This could delay the development of an equinus foot position during the swing phase of gait which is a characteristic component of the progression of gait impairment in DMD.

As the disease progresses, KAFOs are commonly used as part of the rehabilitation strategy for children with DMD (Garralda et al., 2006). The strength of the evidence informing the use of KAFOs, however, is poor overall (level 3b, 4, and 5). Available evidence indicates that offering KAFOs at the end of independent ambulation can prolong independent walking for 24 months (a range of 19.2 to 32.6 months), assisted walking for 36.2 months (a range of 0 to 90 months), and standing for 50.5 months (a range of 31.5 to 58.6 months) (see Bakker, de Groot, Beckerman, de Jong, & Lankhorst, 2000; Garralda et al., 2006, and Katz, 2006 for a summary). KAFOs provide the extra stability at the hip, knee, and ankle necessary for the child to remain upright and ambulatory. This prevents muscle disuse atrophy and delays the development of knee and ankle joint contractures and scoliosis (Bushby et al., 2005; Do, 2002; Garralda et al., 2006). Again, customized footwear and orthoses are important to improve the positioning of the foot and ankle which improves the function of the KAFOs, as well as overall comfort.

**Use of orthopaedic footwear and orthoses to delay and manage joint and muscle contractures**

Joint and muscle contractures develop in DMD from multiple factors including loss of ability to actively move a joint through its full range of motion, muscle imbalance across a joint, static positioning of the limbs, and fibrotic changes in muscle tissue (Brooke, Fenichel, Griggs, Mendell, Moxley, Florence, & Arfken, 1989; Bushby et al., 2010; Hsu & Furumatu, 1993; Sutherland, Olshen, Cooper, Wyatt, Leach, Mubarak, & Schultz, 1981). The management of ankle plantarflexion deformity, which occurs early in the disease process (Siegel & Bernardoni, 1997), is critical as plantarflexion contracture is the most significant precipitating cause of loss of ambulation in children with DMD when the level of voluntary muscle strength is accounted for (Hyde, Flotrup, Glat, Kroksmark, Salling, Steffensen, & Erlandsen, 2000).

To minimize the progression of equinus contractures of the foot and ankle, it is appropriate to recommend lifelong wearing of AFOs at night in conjunction with daytime stretching, especially during the ambulatory phase of the disease (Bakker et al., 1997; Bushby et al., 2005; Hyde et al., 2000; Sussman, 2002). AFOs should be custom-moulded and fabricated for comfort and optimum foot and ankle alignment (Bushby et al., 2005). The recommendation for nighttime AFOs is supported by level 2c and 5 evidence. Of note, however, is that experts have not consistently supported the benefits of nighttime AFOs.

In their review on the “diagnosis and management of Duchenne muscular dystrophy”, Bushby et al. (2010) recommend that passive stretching be used until the child demonstrates loss of ankle dorsiflexion range. At this time, AFO night bracing should be introduced, typically around the age of 9–13 years. In contrast, other experts recommend that nighttime use of AFOs should be introduced upon diagnosis (typically 5–6 years of age) to delay the loss of ankle dorsiflexion range of motion and to limit the development of foot deformity (Stevens, 2006; Sussman, 2002). Of the 21 studies identified addressing the management of ankle joint function in DMD, four studies further evaluated the effectiveness
of stretching, with or without supplemental night bracing, using a prospective design (Brooke et al., 1989; Hyde et al., 2000; McDonald, 1998). Of these, only the study by Hyde et al. (2000) was randomized and therefore considered to provide evidence of sufficient methodological quality to evaluate the recommendation for early bracing.

Hyde et al. (2000) compared the effects of stretching and night bracing (n=15) to stretching alone (n=12) on the evolution of equinus deformity over a period of 30 months in young children with DMD who were ambulatory at the onset of the study (level 2c evidence). Supplementing passive stretching with night bracing reduced the equinus deformity by 23%. These results agree with the previous recommendations made by Brooke et al. (1989) and Scott, Hyde, Goddard, & Dubowitz (1981), based on longitudinal monitoring of functional outcomes of large groups of children with DMD, that night bracing should be included early in the course of DMD. In their non-randomized pilot study, Main, Mercuri, Haliloglu, Baker, Kinali, & Muntoni (2007), however, reported that stretching was only effective for patients who have moderate contractures of the ankle plantarflexors and with no significant tightness of the iliotibial band (level 2b evidence).

In summary, although nighttime AFOs are generally prescribed, this recommendation is not supported by high-quality research evidence. As well, research is also needed to define the parameters for passive stretching that would achieve clinically meaningful delays in ankle plantarflexion contracture while minimizing the burden of time on children and families. Moreover, some researchers have advocated that stretching of the plantarflexors should be balanced with the functionality of ankle plantarflexor stiffness, with greater stiffness improving knee stability during weight-bearing and increasing the total extension support moment of the lower limb during stance – factors that are essential to prolonging walking capacity (Goudreault, Gravel, Nadeau, & Houde, 2006; Lamontagne, Malouin, & Richards, 2000). However, objective criteria to guide clinicians in maintaining an optimal balance between the length of plantarflexor muscles and their function in posture and gait do not currently exist. Additional research is required (supplementing Bromwich, Emery, Stewart, & James, 2010) to understand the balance between plantarflexor muscles and the role of orthoses approaches in achieving balance.

**Concluding Statement**

Pedorthists have a professional role in the treatment of children with DMD, and more specifically in the early phase of disease progression. The prescription of customized footwear and orthoses provides support to the ankle and foot, which can potentially slow down the progression of equino-varus deformity and provide a base of support needed to assist in prolonging the child’s capacity for independent standing and gait.

**References**


Foot Drop

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Key Messages

• Foot drop can be broken down into three main categories: neurologic, muscular, and anatomic (Mayo Clinic, 2017). Understanding which category of foot drop you are treating, understanding foot drop’s root presentation, and understanding the severity of the presentation are key factors for facilitating best outcomes. Foot drop may originate from a variety of influences, such as trauma (Cush & Irgit, 2011), degenerative neurological conditions, e.g., Charcot-Marie Tooth Disease (Han, Kim, Cho, & Sung, 2015) or poor habits (Stewart, 2008).

• There are many treatments available for foot drop, ranging from common sense to great expense. Being aware of the wide range of treatment options will help match realistic goals for patient success.

Keywords
don foot, gait disorder, knee dislocation, lumbar disc herniation, lumbar stenosis, neuropathies, peroneal nerve palsy, stroke

Introduction

Across the literature there exists a wide variety of specific definitions of foot drop. A direct description of foot drop (or drop foot) is a weakness or inability of the foot adequately dorsiflex (Stewart, 2008). The weakness or inability for the foot to adequately dorsiflex may be a permanent or temporary condition.

Males are more affected than females with foot drop at a ratio of approximately 2.8:1, with both feet equally disposed to the disorder (Gale Encyclopedia of Neurological Disorders, 2005). There exist myriad etiologies that can contribute to the presentation of foot drop, and globally these can influence the body at lower (i.e., peripheral neuropathies), central (i.e., spinal stenosis), or upper (i.e., stroke) regions (Wang & Nataraj, 2014). Foot drop may occur at any time in life, as influenced by rare congenital presentations such as unique anatomy (Hawkes, McNamara, O’Mahony, & Dempsey, 2013), compressions or trauma to the common peroneal nerve as with knee dislocations (Cush & Irgit, 2011), or from degenerative / disease processes (Wang & Nataraj, 2014) such as lumbar spinal diseases affecting the sciatic nerve, to name a few.

As outlined in the journal of Clinical Biomechanics in an article by Błażkiewicz, Wizsomirska, Kaczmarczyk, Brzuszkiewicz-Kuźmicka, & Wit (2017), the gait compensations for an individual with foot drop can be remarkable and cause great disturbances to desired walking patterns.

Foot drop is often visually identified by its unique presentation of a high steppage gait pattern, in which the hip on the affected side is raised to assist swing phase clearance and there is notable increased flexion of the knee. The foot remains in varying degrees of plantar flexion during swing phase as the dorsiflexors of the foot have been compromised. With contact on the ground the desired heel strike (again to varying degrees) is replaced by a forefoot or flat foot strike. This toe-to-heel progression of forces can create unwanted lower limb loading to both the affected and unaffected sides of the body (Błażkiewicz et al., 2017; Whittle, 2006).

Again, in a direct view of understanding foot drop it can be viewed as a “compensation seen as a chain originating from a change of movement within the ankle joint” (Błażkiewicz et al., 2017).

Pathology

The range of pathologies that can contribute to foot drop could fill an entire book. In efforts to provide a framework in which to categorize foot drop, the main influences of foot drop pathologies can be broken down into three main categories: nerve, muscle, and brain/spinal function. These categories can be compromised by trauma, disease, or events such

In terms of nerve damage that can lead to foot drop, from a top-down perspective the sciatic nerve branches to the tibial nerve and the common peroneal nerve with the deep peroneal nerve and the superficial peroneal nerve inferior to these structures. Damage to these nerves that ultimately innervate the dorsiflexors of the foot (tibialis anterior, extensor digitorum longus, extensor hallucis longus, peroneus longus, peroneus brevis, extensor digitorum brevis) can lead to foot drop presentations (Stewart, 2008).

While there exist wide-ranging possibilities for the presentation of foot drop, the most frequent influence of foot drop is an interruption to the function of the common peroneal nerve (Masakado et al., 2008).

Some influences of nerve damage are listed below:

Nerve injuries involving foot drop can be sustained from surgical procedures. Lumbar operations aimed at correcting disc health or spinal stenosis may interact with the L4–L5 nerve root (the deep peroneal nerve), causing foot drop (Westhout et al., 2007; Liu et al., 2013). Hip procedures that may interact with the sciatic nerve (Merchant, Lui, Ismail, Wong, & Sitoh, 2005), and knee procedures that may involve the peroneal or common peroneal nerve (Cush & Irgit., 2011). In efforts to repair or relocate the joint, the nerve may be severed or compressed, contributing to foot drop.

Damage to the lower limb function involving pressure at the superficially exposed common peroneal nerve at the lateral fibular head can also lead to foot drop (Stewart 2008, Merchant et al., 2005). Sitting back on the knees, sitting cross-legged too long, or too-tight casting below the knee may cause an interruption to the common peroneal nerve (Stewart 2008).

Other opportunities for lower limb nerve damage involving foot drop are childbirth presentations, complications from diabetes, and sports/work injuries to name a few (Hawkes et al., 2013; Zernich, 2008).

Following is a list of possible diseases/conditions you may see in your clinic, with brief detail of symptoms associated with foot drop:

Amyotrophic lateral sclerosis (ALS) is a fatal progressive neurological disease, causing death 2–7 years after diagnosis. Over the duration of the disease the muscle presentation will atrophy becoming weaker and smaller. Erosion of nerve cells in the brain and spinal cord that control voluntary muscle movement may lead to foot drop and "slapping gait". The cause is unknown and there is no known cure. One early symptom of ALS is tripping, which can be associated with foot drop. As the disease progresses greater muscle control is lost including the ability to breathe (ALS Society of Canada, n.d.; Armon, 2017).

Muscular dystrophy (MD) is a hereditary disease in which the proteins of the muscle are interfered with, altering muscle function and potentially leading to foot drop. It is most commonly seen in boys in early childhood. In severe cases, patients will be unable to walk, breathe, or swallow. MD can be broken down into two types depending on the presentation: Duchenne and Becker. Subcategories can include myotonic, facioscapulohumeral, congenital or limb-girdle depending on presentation. There is no known cure but therapy but narcotics and therapy can help slow the progression (Muscular Dystrophy Canada, n.d.).

Multiple sclerosis (MS) is an autoimmune disease of the brain. It is most often diagnosed in patients aged 15–40, but also affects younger children and older adults. It is a progressive disease, damaging the sheath of nerve cells in the brain and spinal cord. It affects muscle coordination and muscle fatigue and may lead to foot drop (Multiple Sclerosis Society of Canada, n.d.).

Polio is a contagious viral illness that can lead to muscle paralysis that may lead to FD. Last reported naturally occurring case in North America was in 1979. Prevented through vaccination. Polio is still prevalent in parts of Asia and Africa; as such patients you may see in your clinical setting may be older or may have immigrated. (World Health Organization (WHO), n.d.)

Stroke refers to sudden death of brain cells due to lack of oxygen caused by blood flow rupture or blockage. There are two main presentations of stroke: ischemic stroke involves clots or blockages to the blood vessels; in hemorrhagic stroke, blood vessels break or rupture. Leg weakness and FD may occur from the zones of the brain directing motor function (Heart and Stroke, n.d.).

Cerebral palsy is a non-progressive condition that involves damage to the fetal or infant brain that controls motor function. Loss of certain motor function may lead to FD. The deficits can be caused by infection, genetics, or the mother’s health to list a few. While the muscle ability itself may not be...
compromised (no wastage) typically the muscle tone is flaccid from poor firing innervation. Typically, there is no lifetime degeneration of muscle function (MyChild™, n.d.).

Charcot–Marie–Tooth disease, or peroneal muscular atrophy, is a sensory motor neuropathy that can cause foot drop by damaging the nerve axon and myelin sheath in the periphery. As the disease progresses, there is notable weakness and wastage of the lower extremity muscle groups (Don, Serrao, Vinci, Ranavolo, Cacchio, Ioppolo, Paolini, Procacci, Ioppolo, Frascarilli, Pierelli, Frascarilli, & Santilli, 2007). CMT can be broken down into several subcategories depending on the presentation: CMT1, CMT2, CMT3, CMT4, and CMTX. Incidence is 1 in 2,500 and presents in children but mostly in adolescence or early adulthood (NINDS, 2007). Its progression of dysfunction, pain, and lower limb wastage tends to be gradual and varies dramatically from person to person. A normal life expectancy is typical. As this disorder lies outside the brain and spinal cord, it is considered a peripheral neuropathy (NINDS, 2007).

In efforts to better organize the vast influences of FD it is suggested by Wang et al. (2014), to categorize FD into peripheral, central, or upper etiologies.

Differential diagnoses of foot drop are listed below; while this is not an exhaustive list it does describe common presentations (Ying, 2018; Westhout et al., 2007; Wang & Nataraj, 2014; Aquino, Slow, & Long, 2015):

- Anterior horn cell disease (disorders involving the anterior horn of the spinal cord)
- L5 radiculopathy (degenerative disc disease at L4-5)
- Lumbar plexopathy (injury to the lumbar or sacral plexuses)
- Sciatic nerve injury as influenced by trauma (lumbar whiplash), hip surgery, tumours, interruption via injections
- Common peroneal nerve injury via external compression, tight-fitting casts below the knee, habits (compressed sitting or leg crossing), trauma (impact injury or cuts), knee surgery side effects (knee dislocation, traction complications, injury to the fibular head, nerve impingement, fibular tunnel impairment, anterior compartment pressures)
- Disease processes such as Charcot–Marie–Tooth, diabetes, rheumatoid arthritis, multiple sclerosis, muscular dystrophy, amyotrophic lateral sclerosis
- Brain disorder – neurological disruption from the brain causing FD such as a stroke, multiple sclerosis or cerebral palsy
- Complications from chemotherapy, adverse drug outcomes
- Direct trauma to a dorsiflexor muscle of the foot (i.e., severed tibialis anterior)
- Dystonic pseudo foot drop mimics foot drop presentation, but with no strength deficits, and can be an early indicator of Parkinson’s disease

The Patient Perspective

A primary complaint for people with FD is tripping and difficulty with walking when the foot is unsupported. Patients have a sense of weakness, poor gait symmetry, and awareness of sensory disruption. There may or may not be pain present in the lower
extremity. The symptoms may present on the affected or unaffected side, and may be unilateral or bilateral. Among the words used to describe this dysfunction are clumsy, wooden, and dead. People with unsupported foot drop report difficulty managing curbs, stairs, or small rises, especially when distracted or fatigued. A slapping or high steppage gait is another frequent presentation of foot drop. In efforts to assist gait stability, some patients will purchase high-top shoes or boots, but the relative weight of these shoes may have a negative effect with prolonged use, depending on the severity of the foot drop. Other challenges can be getting up out of chairs, automobile seats and rising from a squatting type position. The ability to rise from a seated position can be influenced by age, upper body strength, injury, or an upper neurological condition (Mendell, Kissel, & Cornblath, 2001).

Common Treatment

The first step in treatment is determining the cause of foot drop in each patient, as it does not always originate with the foot itself (Love, 2014).

Treatment options for patients with foot drop vary from simple to complex, depending on the type presenting. Some questions to ask are: What are the patient’s goals for treatment? Better gait? Total recovery? What is possible, and what does it involve?

It is important to be systematic in your approach to determining the level of dysfunction presenting. In terms of treatment, you may be the first healthcare professional seeing the foot drop, or you may be just one of many.

In examining the literature, it becomes clear that being part of a strong group of allied health professionals providing a wide range of treatments will help ensure best outcomes for your patient. Some of the professionals that may be involved in foot drop treatment include pedorthists, occupational therapists, physiotherapists, registered massage therapists, osteopaths, chiroprists, orthopaedic surgeons, neurosurgeons, physiatrists, chiropractors, and family physicians. Some of the treatments these providers may offer include strength training (involving balance and symmetry), gait training, support devices, walking aids, suitable footwear, muscle trigger point release, stretching, joint manipulation, surgery, medications, and counselling.

As suggested in an article by Love (2014), one goal of treating foot drop is to get patients back to a regular gait cycle. This approach aligns with the scope of practice for Pedorthists as set out by the Canadian College of Pedorthics:

- Alleviating painful or debilitating conditions of the lower limb.
- Accommodation of foot deformities.
- Re-alignment of anatomical structures.
- Redistribution of external and internal forces.
- Improvement of balance.
- Control of biomechanical function.
- Accommodation of circulatory special requirements.
- Enhancement of the actions of limbs compromised as a result of accident, congenital deformity, neural condition, or disease (The College of Pedorthics of Canada, 2017).

There are many options for management of foot drop. While the following list is not all-inclusive, it illustrates some examples of foot drop treatment.

Ankle-foot orthoses (AFOs) are often the most common item prescribed to assist with varying levels of dysfunction associated with foot drop (Stewart, 2008). These offer stability and foot clearance for individuals with damage or deficits to the dorsiflexors of the foot (Ruble, 2016).

Mild-control AFO designs can include products with a simple ankle cuff and a short strap that attaches to the dorsal lace of the shoe. This low-profile device helps support patients with slight foot drop, typically when fatigue sets in after a prolonged period of walking.

Another consideration for AFO in patients with mild symptoms is a leaf-spring design. This is a traditional AFO offering a lightweight frame suitable for mild or recovering foot drop, such as in common peroneal nerve compression from poor habits, for which recovery can take 2–3 months (Stevens, Weerkamp, & Cals, 2015). The leaf-spring AFO offers more corrective dorsiflexion support than the cuff style of brace, but it will not address movement outside the sagittal plane.

More involved AFOs may include rigid polypropylene, which reduces dynamic motion of the ankle and can provide aggressive medial longitudinal arch support along with inversion/eversion stability; articulated bi-channel metal, typically a permanent addition to the shoe, and heavier, but addresses swelling concerns; articulated polypropylene, which allows for greater ankle range of motion; and dynamic response ankle-foot orthoses (DRAFO), which provide more flexible...
heel function, permitting a more dynamic ground interaction than traditional AFOs (Ruble, 2016).

For patients that have difficulties with posterior calf support, anterior AFO designs are an option in some foot drop presentations. If an anterior design is being considered, there are those available that will place the support strut in either a medial or lateral presentation, depending on the patient’s anatomy and support needs.

With minor cases of foot drop, some patients have been proven to benefit from taping techniques to assist in reducing ankle plantar flexion and ankle inversion. Positive outcomes have been achieved using kinesiology taping techniques, taping the ankle into dorsiflexion and eversion. While kinesiology taping has its limitations as far as outcome, for the right patient it can be a great introduction to improved gait mechanics during recovery and physiotherapy-focused interventions (Shin, Kim, & Kim, 2017).

Depending on the root cause of the foot drop, varying levels of strength, elasticity, and symmetry training for both sides will greatly assist patients to create consistent, repeatable gait patterns. As with most interventions, multiple treatment options typically produce more comprehensive results.

For some acute presentations of FD, after investigations of more permanent obstacles have been ruled out, manipulative therapy may be an option for presentations thought to be mechanical in nature. A report by Francio (2014) presents a rather immediate relief outcome from a foot drop presentation resulting from an inversion ankle sprain. A manipulative procedure was performed at the determined tibiofibular joint restriction, freeing the peroneal nerve from its entrapment and consequently improving ankle dorsiflexion with improved strength noted as well.

With more severe foot drop, functional electrical stimulation may be a viable option. As outlined by Prenton, Hollands, & Kenney (2016), patients with foot drop have had positive results from electrical impulses sent into paralized muscles, producing more normative gait phasing for the deficit side. For impulse treatment to be successful, the affected nerve must be healthy enough to still accept the artificial nerve impulse. In cases where the nerve is damaged beyond function, surgical options may provide better outcomes.

For patients with severe FD presentations there are surgical options, such as tendon transfers. There have been positive results with transferring the tibialis posterior tendon to various regions of the foot, where it acts as a new innervated foot dorsiflexor. This may be preferred when nerve repair is not possible.

To support the treatment options presented above, patients will require shoes that are well matched to their presentation.

Orthopaedic shoes can offer many attractive support features to assist in function for patients with foot drop (Pedorthic Association of Canada, n.d.). Some key features for the walking needs of these patients may be:

- Heel rockers to ease strike angles/loading
- Forefoot rockers to ease toe-off and improve swing phase clearance
- Strong heel counters to support potential AFO devices
- Wide base of support to increase stability for challenging gait patterns
- Extra depth to accommodate potential AFO devices or the potential for in-shoe lifts
- Lightweight or thin tread outsoles to reduce fatigue and tripping
- Do the shoe requirements go beyond stock or modified orthopaedic footwear requiring a custom shoe to address the patients’ needs?

In a unilateral foot drop presentation, custom foot orthoses may be recommended on the contralateral side. Treatment considerations for these custom foot orthoses may include:

- Whether the contralateral side is symptomatic or asymptomatic
- Level of control desired
- Density of material used (functional or accommodative)
- Length of design
- Volume of design
- Whether there is a gait imbalance that involves a leg length discrepancy, e.g., whether the lift will fit in the shoe with the foot orthosis or an external modification will be required
- Follow-up and management of the device
Key Considerations

Foot drop can be extremely simple or fantastically complex in its presentation.

Foot drop is seldom seen in isolation and is usually associated with another injury, condition, or disease. Knowing the root cause will help guide the pedorthist in determining the best treatment plan.

Having a large tool box of options to offer patients and being able to help patients communicate with other health professionals will aid patients in obtaining complete treatment and recovery.

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Foot Drop
Cerebral Palsy

Lucie Pelland, PT, PhD and Linda Deschamps, BSc (Kin), C. Ped C

Key Messages

• For ambulatory children with cerebral palsy (Gross Motor Classification I, II, and III), corrective custom foot orthoses, used in combination with ankle-foot orthoses and customized orthopaedic footwear, can improve the functional alignment of the lower limb and motions of the foot, ankle, and lower leg in weight-bearing to improve posture control and the energetic cost of gait [Level 3b, 4 and 5].

• Custom contoured orthoses can be incorporated into ankle foot orthoses to reduce the influences of abnormal muscle tone and spasticity, as well as to control the motion of the midfoot and forefoot to reduce the risk for deformity and improve weight-bearing function [Level 4].

Keywords

custom foot orthoses, customized orthopedic footwear, lower limb alignment, posture and gait

Introduction

Cerebral palsy (CP) is a neurodevelopmental disorder that describes a group of motor impairment syndromes resulting from a permanent but non-progressive injury to the brain, sustained early in development (Rosenbaum, Paneth, Leviton, Goldstein, Bax, Damiano, & Jacobsson, 2007). Although CP results from many factors, clinically, it is characterized by impairments of posture control and movement of varying degree, with abnormal muscle tone, spasticity, muscle weakness, and loss of selective motor control being dominant features (Druzbicki, Rusek, Szczepanik, Dudek, & Snela, 2010; Tilton, 2004).

CP is the most common cause of physical disability in children in Canada, affecting 2 to 2.5/1000 children every year, with a worldwide prevalence ranging between 1.5 to >4/1000 live births (Krägeloh-Mann & Cans, 2009; Quinby & Abraham, 2005). Specifically, among infants born prematurely or with a very low birth weight, the incidence rate of CP increases to 40-100/1000 live births (Krägeloh-Mann & Cans, 2009; Rosenbaum, 2003). Among children with CP, it is estimated that 58% can walk independently and 11% using a hand-held mobility device, with about 30% of children having limited or no walking ability (Christensen, Van Naarden Braun, Doernberg, Maenner, Arneson, Durkin, Benedict, Kirby, Wingate, Fitzgerald, & Yeargen-Allsopp, 2008). Due to

shared developmental pathways between different brain system, the motor disorders of CP are often accompanied by impairments in sensory function, cognition, communication, perception, and behaviour (Bax, Goldstein, Rosenbaum, Leviton, Paneth, Dan, & Damiano, 2005), with an estimated 41% of children with CP having epilepsy and 7% a dual diagnosis of autistic spectrum disorder (Christensen et al., 2008).

While basic science research is advancing knowledge on the emerging role of neuroprotective agents in the primary prevention of CP, there is currently no cure for CP. Therefore, the therapeutic management of the child with CP aims to minimize the negative outcomes of altered sensory–motor control on musculoskeletal development and to facilitate the development of gross motor function, posture control, and gait (Koman, Smith & Shilt, 2004).

The purpose of this structured review is to identify practice trends and recommendations for the use of foot orthoses and customized orthopaedic footwear in the clinical management of the child with CP, as they pertain to the management of abnormal muscle tone, malalignment of the lower limb, and impairments in posture and gait, and to critically appraise these recommendations within the context of current research evidence.
CP results from a static permanent insult to the brain that is sustained early in development, either in utero, perinatally, or within the first 3 years of life (Flett, 2003). Common causes of CP include hypoxic-ischemic stroke, haemorrhagic stroke, mechanical damage to the brain and spinal cord, deep central nervous system hypoxia, and birth defects of the central nervous system (Koman et al., 2004). While the severity of motor impairments depends primarily on the location, magnitude and extent of the injury to the brain, the timing of the injury is also critical, as different parts of the brain are selectively vulnerable at different periods of development (Back, Luo, Borenstein, Levine, Volpe, & Kinney, 2001; Dammann & Leviton, 2000; Koman et al., 2004). In particular, the ascending and descending white matter tracts that course in the internal capsule, located in proximity of the lateral ventricles, are vulnerable to ischemic-hypoxic events during the perinatal period due to the high metabolic demand during myelination of these tracts (Englander, Pizoli, Bachatreanko, Sun, Worley, Mikati, Kurtzberg, & Song, 2013). As the corticospinal tract constitutes a large part of the internal capsule, carrying information from the primary motor cortex to the lower motor neurons in the spinal cord, an ischemic-hypoxic lesion in this area, termed periventricular leukomalacia (PVL), is strongly associated to the motor impairment of CP, with 60-80% of infants with PVL later diagnosed with spastic diplegia (Rosenbaum, 2003).

Premature neonates, born at <32 weeks of gestation, and those requiring mechanical ventilation, which is associated with hypotension, hypoxemia, acidosis, and hypocarbia, are at highest risk for PVL. Other risk factors for PVL include: low birth weight, intrauterine infection, infection around the time of delivery, placental vascular anastomoses, antepartum hemorrhage, chorioamnionitis, funisitis, sepsis, twin gestation, and maternal cocaine use (Koman et al., 2004).

Pathology

Clinical Diagnosis

PVL is usually diagnosed by cranial ultrasound, magnetic resonance and computed tomography imaging. As PVL results from an ischemic-hypoxic event, it can take up to 4-8 weeks to be detectable on imaging. Therefore, neonates with known risk factors for PVL typically undergo examination by medical imaging around 30 days after birth. However, the clinical diagnosis of CP itself is typically made within the first 12 to 18 months of life, as the child fails to attain expected milestones of early development and exhibits qualitative differences in motor development, such as asymmetry in gross motor movement, abnormal tone such as stiffness or floppiness, and impairments in postural control (Rosenbaum, 2003).

The diagnosis of CP is based on a systematic evaluation of the child, and considers the gestational, birth and perinatal history; the early developmental trajectory and attainment of developmental milestones; and performance on standardized physical and functional evaluations. The physical examination is based on a thorough review of the musculoskeletal system, including limb alignment and muscle flexibility and power, and a complete neurological assessment, including muscle tone, sensation, coordination, fine/gross motor movement, posture, and gait as age-appropriate. Medical imaging and other specialized tests are used to assess the location, nature, and extent of central nervous system damage (Koman et al., 2004).

Clinically, CP is described by type, topography, and severity (Rosenbaum et al., 2007; Rosenbaum, 2003). The type describes the dominant motor disorder: spastic, dystonic, dyskinetic, athetoid, or ataxic. The topography describes the distribution of impairments: hemiplegia, diplegia, triplegia, and quadriplegia. The severity describes effects of the impairments on the motor function of the child, which is classified using the Gross Motor Function Classification System, (GMFCS) (Palisano, Rosenbaum, Walter, Russell, Wood & Galuppi, 1997).
Clinical course of the motor impairments of CP

While the injury to the brain in CP is non-progressive in nature, the clinical manifestation of CP is often changing (Rosenbaum et al., 2007). The GMFCS classifies children along a continuum of gross motor function ability, from level I (child walks without restrictions, indoors and outdoors, and is able to climb stairs, run, and jump without limitations, although speed, balance, and co-ordination might be impaired) through level V (severe restrictions in all areas of motor function, with limited anti-gravity control of the head and trunk and no means for independent mobility). A complete description of the GMFCS is available from the CanChild research Centre at McMaster University (https://canchild.ca/).

For children having the capacity for independent standing and ambulation, GMFCS levels I, II and III (Palisano et al., 1997), emerging muscle contractures, torsional deformities of long bones, and joint subluxations cause additional biomechanical constraints which impose further demands on the nervous system to organize effective postural control and movement strategies. These common musculoskeletal problems develop as the mechanical demands on the neuromusculoskeletal system increase with child growth and the demands of weight-bearing (Druzbicki et al., 2010; Quinby & Abraham, 2005). Therefore, the prevention and management of these biomechanical constraints on lower limb alignment and function are key factors to address in the treatment of children with CP.

Evidence-based recommendations for the orthotic management of the child with CP

Custom designed foot orthoses

The use of orthoses is one component of a multidisciplinary approach to the management of lower limb function in children with CP, supporting normal joint alignment, delaying the development of muscle contractures and torsional deformities of the long bones, redistributing mechanical loads on joints and soft tissue, and improving overall lower limb biomechanics for posture control and gait. As the mechanics of the foot are implicitly linked to those of the knee, hip, and pelvis, custom orthoses are usually integrated into University of California at Berkeley Laboratory (UCBL), supramalleolar, and ankle-foot orthoses. Individually-contoured orthoses have also been recommended to inhibit abnormal muscle tone and activity that can disrupt the biomechanics of the foot during weight-bearing (Crenshaw, Herzog, Castagno, Richards, Miller, & Michaloski, 2000; Westberry, Davids, Shaver, Tanner, Blackhurst, & Davis, 2007).

Tone-inhibiting foot orthoses

The principles of tone-inhibiting orthoses are based on the theory and practice of inhibition casting (Duncan & Mott, 1983). Generally, tone-inhibiting orthoses include the following features (Hylton, 1989; Duncan & Mott, 1983; Lohman & Goldstein, 1993): custom heel cup with a recessed area under the calcaneal pad; build-up support for the medial longitudinal arch to distribute weight-bearing pressure toward the lateral column of the foot; peroneal notch to stabilize the rear-foot and subtalar joints; and a build-up under the transverse metatarsal arch which, in combination with recessed areas under the metatarsal heads, improves midline stability of the forefoot and reduces PF and inversion tone. As needed, a mild build-up under the toes reduces tone of the long toe flexor muscles, allowing dorsiflexion across the metatarsal–phalangeal joints for push-off. While the theoretical basis for tone-inhibiting orthoses is grounded in sound principles of biomechanics and neurophysiology, (Morris, Bowers, Ross, Stevens, & Phillips, 2011), the evidence remains principally based on expert and parent opinion (Level V; Näslund, Tamm, Ericsson, & von Wendt, 2003).

Optimizing lower limb alignment for function

The effectiveness of custom designed orthoses could be further improved by adapting the ankle-foot orthosis to optimize the sagittal plane alignment of the lower limb (Butler, Farmer, Stewart, Jones, & Forward, 2007; Morris et al., 2011; Owen, 2002; Stallard & Woollam, 2003). The optimal alignment of the lower limb can be defined by the shank-to-vertical angle (SVA), which defines the inclination of the tibia over the foot in the sagittal plane and, hence, the orientation of the ground reaction force vector at midstance (Jagadamma, Coutts, Mercer, Herman, Yirrel, Forbes, & van der Linden, 2009).

An SVA of 12° of forward tibial inclination in standing, in combination with control over ankle dorsiflexion, is recommended to allow sufficient advancement of the
tibia over the foot during the second rocker of gait to prepare for push-off, while reducing the magnitude of external knee flexion (Butler et al., 2007; Jagadamma et al., 2009; Rogozinski, Davids, Davis, Jameson, & Blackhurst, 2009; van Gestel, Molenaars, Huenarts, Seyler, & Desloovere, 2008). This controlled motion of the ankle and knee reduces the demands on quadriceps activity required to stabilize the knee (Butler et al., 2007).

The optimal SVA, to align the ground reaction force vector with the centre of rotation of the knee in order to minimize the magnitude of abnormal external knee flexion and hyperextension moments (Jagadamma et al., 2009; Owen, 2002; Wesdock & Edge, 2005), can be achieved through an iterative modification of the height, type, and design of the heel of the custom orthotic and shoe, as well as the type of shoe rocker provided at the metatarsal heads (Jagadamma et al., 2009). As an example, a positive inclination of the heel (i.e., wedging to increase heel height) enables full foot contact with the floor in equinus foot posture and reduces the need for excessive hip and knee flexion (i.e., crouch gait posture) by shifting the ground reaction force vector slightly anteriorly (Wesdock & Edge, 2003). These improvements in distal alignment and vertical posture facilitate standing balance, while the buildup of the rocker at the metatarsal heads facilitates advancement of the tibia and the mechanics of roll-off.

Tuning may not be suitable for all children. Small cohort controlled studies (n=5 and n=11) did not demonstrate any appreciable outcomes of tuning on posture alignment (Jagadamma et al., 2009; Wesdock & Edge, 2003). The presence of ataxia and flexion contractures at the hip and knee may limit the ability to achieve an optimal SVA (Butler et al., 2007). Moreover, Butler et al. (2007) reported that children with knee flexion angles greater than 20° in the first third of stance and who are unable to produce sufficient knee extensor moment to reach a minimum of 10° of residual knee flexion in the second third of stance will not respond to tuning.

**Concluding Statement**

Pedorthists have a professional role in the treatment of children with CP, specifically in providing sufficient support to protect the foot, optimizing lower limb alignment and the orientation of the ground reaction force for posture control and gait, and reducing the effects of abnormal tone on lower limb alignment and function.

**References**


Clubfoot (Congenital Talipes Equinovarus)

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Key Messages

- Congenital talipes equinovarus (CTEV) is a congenital condition characterized by deformities in the frontal plane (hindfoot varus), sagittal plane (ankle equinus), and transverse plane (forefoot adduction).
- Exact cause of the condition is unknown, but several genetic and environmental factors are suspected to play a role.
- The Ponseti method is the most widely used conservative treatment for CTEV, utilizing serial casting and gentle progressive manipulation to correct joint position.
- Surgical methods including percutaneous Achilles tenotomy (PAT), anterior tibialis muscle surgery and posteromedial soft tissue release (PMR).
- Other treatments include the French method, the Kite method, & the Copenhagen method.
- Pedorthic interventions typically include custom foot orthoses, footwear recommendations, footwear modifications (lift to compensate for leg length discrepancy if present or lateral buttress, if mechanics permit), and ankle foot orthoses dependant on patient-specific factors.

Keywords

calcaneal varus, clubfoot, congenital deformity, congenital talipes equinovarus (CTEV), genetic, osseous deformity, Ponseti, varus alignment

Introduction and Pathology

Congenital talipes equinovarus (CTEV), also known as clubfoot deformity, is a congenital condition characterized by forefoot and midfoot adductus, hind foot varus and ankle equinus (Bacino & Hecht, 2014). Two main classifications of CTEV have been identified: idiopathic (isolated) and syndromic (those associated with other conditions). In both classifications, the cause is unknown. (Gray, Pacey, Gibbons, Little, & Burns, 2014). Two classification systems exist to evaluate the severity of the deformities. Both classification systems utilize a point score based on gross physical findings that correlates with clubfoot severity (Dobbs & Gurnett, 2009). CTEV is recognizable at birth, and can vary from flexible to extremely rigid. When left untreated, children with CTEV walk on the lateral sides of their feet, resulting in complications such as callusing, skin and bone complications, and limitations in mobility and functional ability (Dobbs & Gurnett 2009).

CTEV is one of the most common orthopaedic disorders, affecting 1-2 in 1000 newborns (Gray et al., 2014). Racial differences occur, with an increased incidence in Maori populations of up to 7 in 1000 (Gibbons & Gray, 2013). While some cases of CTEV may be associated with myelodysplasia, arthrogryposis or multiple congenital abnormalities, most cases of this congenital condition are both idiopathic and
isolated (Dobbs & Gurnett, 2009). CTEV affects males twice as often as females and occurs bilaterally in approximately 50% of cases (Gibbons & Gray, 2013). The etiology of CTEV is largely unknown, although hypotheses include transient gene activity, environmental factors, neuromuscular disease, lack of fetal movement and multifactorial genetic causes (Gray et al., 2014; Gibbons & Gray 2013).

Recent research has identified several genes as possible factors in CTEV, but origins are still considered to be a multifactorial combination of environmental and genetic factors (Bacino & Hecht, 2014).

**Differential Diagnosis**

Prenatal diagnosis of CTEV is becoming increasingly more common, with clinical observations being made at 20 weeks gestation in up to 77% of cases. Clubfoot is more likely to be diagnosed prenatally if the deformity is bilateral, or if other abnormalities are present. The post-natal severity of the deformity cannot be determined from prenatal ultrasound findings (Gibbons & Gray, 2013).

Postnatal diagnoses of CTEV are performed by physical examination, typically at birth. Patients with CTEV present with ankle equinus, hindfoot varus and forefoot adduction. Severity of the deformity is often determined by one of two scored grading systems. The Dimeglio classification system uses a 4-point system to evaluate the severity of equinus, varus, derotation, and forefoot adduction. The Pirani scoring system assesses both the severity of the deformity as well as the progress of treatment by the Ponseti method. This scoring system uses a simple scoring system based on the physical appearance of the foot using 3 clinical signs in the both the hind foot and midfoot (Balasankar, Luximon, & Al-Jumaily, 2016).

Cases of CTEV include deformities in all three planes of motion; hindfoot varus in the frontal plane, ankle equinus in the sagittal plane and forefoot adduction in the transverse plane. CTEV should not be confused in patients with metatarsus adductus, with the absence of any frontal or sagittal plane deformities. Considerations must also be made for co-morbid conditions that affect the foot and lower limb such as cerebral palsy, arthrogryposis, spina bifida and other neuromuscular disorders.

**Common Testing**

Common testing for CTEV is centred around physical presentation, joint positioning, and range of motion testing. Physical examination and assessment of the severity of the deformities will guide treatment. Patients with CTEV may present with varying degrees of hindfoot varus, ankle equinus, and forefoot adduction, depending on the implementation and success of previous treatments. Physical assessment of the foot and ankle will provide information on the severity of joint deformity, flexibility, and available range of motion in the affected joints.

Comprehensive range of motion testing should include passive assessment of all of the affected joints of the foot and ankle. As CTEV primarily affects the hindfoot and midfoot, the pathological changes are seen primarily in these areas. In severe cases, the deformity can extend distally into the metatarsals, resulting in inversion and plantar flexion of the metatarsals, or proximally to the calf, presenting with a shortened Achilles and atrophy of the gastrocnemius.

In addition to standard weightbearing observations of positional deformity and dynamic gait assessment, non-weightbearing assessments of the joints of the hindfoot and midfoot will reveal the severity of the deformity and the available ranges of motion. These areas should be examined during a pedorthic assessment to determine the severity of the deformity and available range of motion.

**Subtalar joint**

Calcaneal inversion is a primary indicator of CTEV. The calcaneus may be congenitally smaller, and the subtalar joint is often restricted in eversion or fixed in a degree of inversion. Passive manipulation of the subtalar joint, moving the calcaneus through its available range of motion will illustrate the severity of the joint restriction and available range of motion.

**Talocrural joint**

Another cardinal sign of CTEV is plantar flexion of the ankle at the talocrural joint. This deformity may be osseous/rigid, or as a result of shortening in posterior lower leg musculature. Active and passive testing of the range of motion of this joint will demonstrate the severity of the deformity.

**Talonavicular & calcaneocuboid joints**

Medial subluxation of these joints results in varying degrees of adduction and inversion of the midfoot in patients with CTEV. Palpation of these joints to determine the presence and severity of the subluxation. Observations of the forefoot positioning relative to any subluxation is also important in identifying any forefoot adductus.
 Coleman block test
When assessing the degree of hindfoot varus, the Coleman block test can be used to determine the presence of fixed forefoot valgus and its effect on the varus position of the hindfoot. If the hindfoot is rigid, the varus deformity will remain present. A flexible hindfoot will correct towards a neutral position.

Imaging
In paediatric cases of CTEV, imaging may be used to observe the progress during treatment. The usefulness of X-rays in the first few months of life is debatable, as the ossification of the bones is not yet complete throughout the foot. Ultrasound may also be used to observe the foot in multiple positions and in cases of unusual evolution during treatment (Bergerault, Fournier, & Bonnard, 2013).

Contraindications
Contraindications will be largely dependent on the limitations and severity of the cavovarus foot deformity present in the patient. With some cases of CTEV, serial casting, or surgical procedures may restore the majority of joint range of motion, thus reducing potential contraindications. With any cavovarus foot type, foot Pedorthic interventions which exacerbate varus of the hindfoot, adduction of the midfoot or plantar flexion of the ankle would be contraindicated.

The Patient Perspective
CTEV is typically diagnosed prenatally, at birth, or when the child begins to ambulate. As such, patients with this pathology will likely have an extensive history of treatment, including surgeries, bracing, serial casting, tendon release/transfer, physiotherapy, and other modalities.

Despite its often severe presentation, CTEV does not always present with pain or functional disability. Half of all cases will present bilaterally, and in the other 50% of cases, the affected foot will be smaller and shorter than the unaffected side. Even with surgical intervention or serial casting, there are often minor functional and structural differences in the affected limb post intervention. In some cases, the affected limb will suffer growth deficiencies and result in a leg length discrepancy.

Adult patients with a history of CTEV may present with a cavovarus foot type with varying degrees of joint restrictions. Patients may have difficulty finding footwear that is comfortable and functional, depending on their individual joint restrictions. Callusing, discomfort, and history of ankle instability may be present. Comfort and stability are two of the primary concerns in patients with a history of CTEV. Appropriate footwear, modifications, and custom foot orthoses can assist in improving comfort and function.

Common Treatment
For more than a decade, conservative management strategies have been the most widely used methods to correct clubfoot deformity (Balasankar et al., 2016). Over the past century, there have been several important developments in treatment protocols that have made it easier to correct this significant condition.

In the beginning, attempts at non-operative strategies often relied on painful and forceful manipulations. In the late 1930s, Kite introduced a method of gentle manipulations and serial casting in an attempt to gradually correct each component separately. Shortly after the Kite method was introduced, Ignacio Ponseti developed and refined his method for clubfoot. The Ponseti method remains the most widely used method for treating CTEV, with other conservative methods also being utilized recently (Dobbs & Gurnett, 2009).

Kite Method
The Kite method was the first conservative method developed for the correction of clubfoot deformity. This method focused on the systematic and sequential correction of the components of the deformity. First, the forefoot adduction was addressed, followed by the hindfoot varus, and finally the ankle equinus (Sanzarello, Nanni, & Faldini, 2017). Patients were cast in a toe–to–groin plaster cast, changed every 7–10 days until full correction, which could take up to two years (Sanghvi 2009, Dobbs, 2009). The Kite method applied corrective manipulations around the calcaneocuboid joint and was followed by full-time splinting to avoid recurrence. (Sanghvi & Mittal, 2009). Kite reported good outcomes initially, but his results were not reproducible. Up to 90% of patients needed additional surgical intervention. These outcomes were attributed largely to the anatomically inaccurate method of manipulation and inadequate casting (Herzenberg, Radler, & Bor, 2002).

Ponseti Method
Similar to the Kite method, the Ponseti method involves weekly gentle manipulations followed by the application of serial long leg casting (Balasankar et al., 2016). This method is typically initiated during the
first weeks of life, but may also be used after walking age (Bergerault et al., 2013). In contrast to the Kite method, the Ponseti method abducts the foot around the head of the talus, avoiding forceful pronation (Sanghvi & Mittal, 2009). This method focuses first on the reduction of the foot adduction and hindfoot varus followed by a correction of the ankle equinus. In severe cases, or when gentle manipulation is not able to fully correct the ankle equinus, a percutaneous Achilles tenotomy is incorporated into this method (Sanghvi & Mittal, 2009; Balasankar et al., 2016). Once the deformity is corrected, abduction braces are to be used 23 hours a day for 12 weeks, after which the braces are used at night until the age of 4 (Balasankar et al., 2016).

The Ponseti method is currently the gold standard for treating CTEV at birth (Sanzarello, Nanni, & Faldini, 2017).

**French Functional Method (also known as physiotherapist method)**

The French functional method states that CTEV occurs due to contractures in the posterior lower leg, weakness of the peroneals, and deviation of the mid–tarsal joints. This method incorporates daily stretching of the medial foot, elastic taping, and splinting. Duration of treatment is 1-3 months of daily treatment and home exercise (Balasankar et al., 2016).

**Copenhagen Method**

Based on the same principles as the aforementioned, the Copenhagen method utilizes daily manipulations, muscle stimulation, and plaster casting to correct the deformity (Balasankar et al., 2016). Following the protocols of serial manipulation and casting, the Copenhagen method uses bandaging rather than bracing until the patient is walking (Utrilla-Rodiguez, Martinez-Cañavete, & Casares, 2012).

**Pedorthic treatment**

As primary treatment for CTEV is performed in early childhood, pedorthic treatment is most often focused on supporting any remaining elements of rearfoot valgus, ankle equinus, or midfoot/forefoot adduction. Adult patients with a history of CTEV may present with a pes cavus foot structure and may benefit from custom foot orthoses. Patient–specific footwear recommendations and/or modifications may also be indicated, depending on the severity of remaining deformity and the associated symptoms.

Custom foot orthoses have been shown to decrease foot pain associated with pes cavus (Hawke, Burns, Radford, & du Toit, 2008). Devices designed to provide support and offloading for the cavus foot structure, accommodate for any remaining ankle equinus, and reduce the patient’s symptoms are indicated.

Footwear recommendations to accommodate a voluminous cavus midfoot, increase shock absorption, and improve gait kinematics are often utilized, as indicated by the patient’s symptoms and presentation of foot deformity. In cases where hindfoot varus or ankle equinus are significant, modifications to footwear such as accommodative heel lifts or lateral buttressing may be indicated. In severe cases of uncorrected CTEV or significant residual deformity, custom footwear may be indicated.

**References**


Hammer Toe, Claw Toe, Mallet Toe

Amy Chapman, C. Ped (C)

Key Messages

• Toe deformities result from the imbalance of toe flexors and extensors.
• Lesser toe deformities have a negative impact on activities of daily living.
• Lesser toe deformities are a risk factor for falls in older people.
• Very common foot deformity in older populations.
• Higher prevalence among women.

Keywords
distal interphalangeal joint (DIP), extrinsic muscle, imbalance, intrinsic muscle, lesser toe deformities, metatarsal callusing, metatarsophalangeal joint (MTP), proximal interphalangeal joint (PIP), toe cramping/bending/buckling

Introduction

Lesser toe deformities (hammer toe, claw toe, and mallet toe) are common deformities of the foot. Also referred to as buckled toe, crooked toe and bent toe, they are amongst the most commonly found foot problems in the elderly population (Mickle, Munro, Lord, Menz, & Steele, 2009; Montgomery & Davies, 2016). These sagittal plane deformities can be rigid or flexible and often result in the development of corns or calluses at protruding joints (Decker & Albert, 2002). Although the terms are often interchanged in literature, each deformity has its own unique features. A hammer toe is defined as a deformity in which the proximal interphalangeal joint (PIP) is in a flexed position. A claw toe deformity involves flexion at the PIP and distal interphalangeal joints (DIP) while the metatarsal phalangeal joint (MTP joint) is in a neutral or extended position. A mallet toe involves only the DIP, which is in a flexed position (Barakat & Gargan, 2006). A curly toe is a deformity of the IP joints in the coronal plane (Montgomery, 2016), often hereditary. Lesser toe deformities are better treated when caught early. If fixed in a flexed position for an extended period of time, osteoarthritic changes can occur forming a rigid deformity which can become extremely difficult to accommodate (Montgomery & Davies, 2016; Lorimer, 1993). Decker & Albert (2002) have stated that one in every 15 individuals between the ages of 31–60 have at least one lesser toe deformity. Once over the age of 60, one in every 10 individuals will acquire a lesser toe deformity, although other studies have found prevalence rates of 60% (Dunn, Link, Felson, Crincoli, Keysor, & McKinlay, 2004; Mickle et al., 2009). There is a much higher prevalence rate among females, ranging from 2.5:1 to 3:1 compared to males (Decker & Albert, 2002).

Pathology

Lesser toe deformities are typically the result of a muscle imbalance or improperly fitted footwear (AAOS, 2001) but are certainly exacerbated by poorly fitting footwear (Montgomery & Davies, 2016). Other conditions that may cause lesser toe deformities include congenital malformations, diabetes, peripheral neuropathy, neuromuscular disorders, inflammatory arthritis, and acute injuries (Alexander, 1997).

Neuromuscular disorders create an imbalance in the muscles of the foot by weakening extensor muscle groups, causing an overpowering flexor pull in the toes. Inflammatory arthritis, including seropositive and seronegative rheumatoid arthritis, can cause changes in the joint capsule which can lead to a disruption in the structure of the joint, hyperextension at the MTP joint, and finally cause the characteristic flexion at the PIP joint of a hammer toe.
Acute trauma to the foot can cause ligament or soft tissue disruption which may also lead to any of the lesser toe deformities (Hurwitz, 1999).

Women are more likely to acquire lesser toe deformities. Hammer, claw, and mallet toes may occur at any age but are more common within the older adult population. Studies have shown toe deformities are very uncommon amongst non–shoe wearing populations (Hurwitz, 1999). When the deformity is an isolated incident, the second and fifth digits are often the most commonly affected. A second toe deformity can be due to a long second phalanx combined with incorrect footwear length or when a hallux valgus forces the second toe dorsally. A fifth–toe deformity can be due to an inadequate toe–box width (Hurwitz, 1999; Lorimer, 1993).

One of the proposed causes of lesser toe deformities is an imbalance between static and dynamic forces acting on the joint. Static forces are determined by the bony and ligamentous architecture of the joint. Dynamic forces are controlled by muscle/tendon units and the movements they generate across the joint (Montgomery & Davies, 2016; Kwon, Tuttle, Johnson, & Mueller, 2009). The intrinsic foot muscles, interossei and lumbricals, influence the extrinsic muscles through their attachment into the extensor hood of the metatarsals. The extensor hood is formed by the extrinsic extensor tendons and the intrinsic muscles contribute slips to this structure which balance tension in the extensor hood. Weakness or dysfunction of the intrinsics can lead to a loss of tension in the extensor hood which allows the extrinsic extensors to extend the MTP joint, but flex the PIP and DIP, which can produce a claw deformity (Montgomery & Davies, 2016). When the intrinsic flexors are weak, the extensor digitorum longus and brevis overpower the flexor muscles and in turn cause hammering at the toes (Kwon et al., 2009). A decrease in toe flexor strength is a contributing factor to the development of lesser toe deformities (Mickle et al., 2009). Mickle, Munro, Lord, Menz, & Steele (2011) showed that individuals with hammer toes demonstrate a greater postural sway during gait which may contribute to increased falls in the elderly. Mickle et al., (2009) showed that the paper–grip test (resisting a piece of paper being pulled out from under the toes) is a good predictor of toe flexor strength which correlates to balance during functional tests in elderly people. Toe flexor strength is needed to maintain good balance and assist with propulsive force (Misu, Doi, Asai, Sawa, Tsutsumimoto, Nakakubo, Yamada, & Ono, 2014). Those with weaker flexor strength are more likely to have lesser toe deformities but they are also at higher risk for falls (Mickle et al., 2009).

Mallet toe deformity is often an isolated incident involving flexion of the DIP only. It can occur gradually due to a contracture of the flexor digitorum longus tendon, or after an acute avulsion of the extensor digitorum longus insertion (Lorimer, 1993).

Short, narrow footwear with a pointed toe box force the lesser toes into a flexed position. When toes are forced into this position for long enough, the structures adapt; muscles cannot operate appropriately and imbalances occur which contribute to the development of toe deformities (Montgomery & Davies, 2016). Women’s high heeled shoes increase stress to the forefoot and force the foot forward into a pointed toe box, increasing the chances of developing lesser toe deformities with repetitive use (Hurwitz, 1999).

Cavus foot types tend to be more prone to lesser toe deformities due to the increased stress on the MTP joints and the soft tissues supporting them (Decker & Albert, 2002). A cavus foot has a large decline angle at the metatarsals which forces the proximal phalanx into dorsiflexion. This position held at the proximal phalanx causes the lumbral muscles and interosseous tendons to migrate superior to the phalanx, which in turn inhibit their abilities as plantar flexors. If superior displacement is mild, the muscles will only create a compressive force at the joint; however, if the superior displacement is severe, the tendons will act as dorsiflexors of the proximal phalanx. This placement can lead to hammering of the lesser toes, especially the fourth and fifth phalanges (Michaud, 1993).
It is always important to evaluate metatarsal head tenderness, especially when a hammer toe deformity is present. It is possible that with increased pressure on the metatarsal heads, MTP joint subluxation or capsulitis may occur. Lesser toe deformities can be fixed or flexible. A flexible deformity usually involves soft tissue imbalance or damage. Pain with passive movements is typically the result of joint motion or tension on tendons or joint capsules. If flexible, a lesser toe deformity may be passively corrected by pushing up just behind the metatarsal heads on the plantar surface of the foot. Fixed lesser toe deformities are typically the result of joint or tendon contracture, dislocation of MTP joint, shortening of plantar capsules or flexor tendons, or wear and tear of the joint surfaces (Hurwitz, 1999).

Differential Diagnosis

Lesser toe deformities may be asymptomatic or present with extreme pain over high pressure areas. Lesser toe deformities may be confused with mimicking conditions which can lead to improper choice of treatment methods and unsuccessful results. Although these deformities are often associated with callusing under the metatarsal head, plantar callusing may be misdiagnosed as plantar wart. Pain associated with lesser toe deformities may be confused with the numbing, burning, and shooting pain commonly found in Morton’s neuroma. This type of pain is not typically present in lesser toe deformities.

Another source of pain at the MTP joint, which may be confused with the MTP joint pain associated with lesser toe deformities, is metatarsal stress fracture. Stress fractures may be ruled out by a bone scan or MRI but typically will not show until two months after the injury occurred (Hurwitz, 1999). Curly toes are a common lesser toe deformity that could possibly be confused with hammer, claw, and mallet toe deformities. Curly toes are usually congenital deformities which run in families and typically involve a torsional medial deviation of the third, fourth, or fifth toes. This deformity may resolve itself with age but typically is a continuing deformity (Barakat & Gargan, 2006).

Common Testing

To first examine the possibility of a lesser toe deformity, a visual inspection should be performed. Observe the sagittal plane position of the MTP, PIP, and DIP. Also check for any callusing or corns on the plantar surface of the MTP, dorsal PIP, and distal ends of the toes. Be sure to examine the toes in both non-weight-bearing and weight-bearing positions, as weight-bearing will typically accentuate the deformity and elicit pain beneath the metatarsals and distal toe (Hurwitz, 1999). The clinician needs to determine if the deformity is fixed or flexible.

In some cases it may be necessary to perform tests to rule out neurological disorders as this may be the cause of muscle imbalances leading to hammer, claw, or mallet toes (AAOS, 2001).

Contraindications

Shoes with shallow, narrow toe-boxes are not recommended for those with lesser toe deformities. Ulceration is a common concern when dealing with poor circulation or neuropathy. In these cases it is important to monitor the use of any treatment method which may cause rubbing or high pressure areas such as sulcus crests (sulcus bar, toe crests) or materials used to deflect pressures that have aggressive edges which may themselves be a cause of excess pressure (Hurwitz, 1999).

The Patient Perspective

In addition to trouble finding well-fitting footwear, patient complaints often come from functional impairments such as stress on affected joints, pressure on the skin, stretching of connective tissue, corns, and callusing (Hurwitz, 1999). Patients with lesser toe deformities may describe bending, cramping, or buckling of the lesser toes (Decker & Albert, 2002), especially in standard footwear. The primary complaint is usually of irritation and callusing on the protruding joints from repetitive friction and pressure points. Hammer toes can develop irritations at the dorsal PIP with possible callus formation at the metatarsal head and tip of toe. Claw toes can present with irritations and calluses can develop on the end of the toe, under the edge of the toenail; the nail can
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become thickened, making trimming difficult, or the nail bed can become damaged due to constant pressure from the end of the toe driving into the ground resulting in toenail deformities or death of the toenails (black nails). Mallet toes will typically present with corns or discomfort at the tip of the toe (Decker & Albert, 2002).

**Common Treatment**

Treatments for hammer, claw, and mallet toes include orthoses with metatarsal pads and accommodations for any painful lesions, footwear modifications, commercial accommodative devices, physical therapy, and surgical intervention. Typically the more conservative treatment options are attempted before surgery becomes an option. It is important to know whether the lesser toe deformity being treated is flexible or fixed as this will change the treatment. A flexible deformity can be passively corrected and therefore treatment should focus on correcting the deformity to avoid pressure points and fixation in the deformed position. If the deformity is flexible, metatarsal pads placed just proximal to the MTP joints can help the resting position of the toe and reduce contact of the toe and the shoe upper, which reduces painful friction. The toe can be splinted to reduce friction on the dorsal aspect of the toe, or toes can be taped together (buddy taping) to try and keep the affected toe straighter. A fixed deformity needs to be accommodated to decrease high pressure areas and avoid further deformation. Footwear choices need to include shoes with deeper toe boxes, uppers made of soft material to accommodate the toe deformities, and no seams over the areas of concern.

**Orthotic intervention**

Accommodative orthoses for hammer, claw, and mallet toes include deflecting, cushioning, or supporting the affected areas.

A metatarsal pad placed proximal to the metatarsal heads will offload weight and support a plantar flexed or irritated metatarsal head (AAOS, 2001). If lesser digits are affected, a metatarsal bar, which extends more laterally, spanning from the second to fifth MTPs, would be appropriate (Deschamps, 2010).

If plantar metatarsal head or DIP irritation is extreme, or the metatarsal is in a fixed plantar flexed position, it may be necessary to excavate under the affected area (Decker & Albert, 2002). Excavations can be done in the orthosis, or in more extreme cases in the inside of the shoe itself.

If fat pad atrophy has occurred underneath the metatarsal head or the distal end of the toe is irritated, cushioning of pressure points can provide relief (Deschamps, 2010).

A toe crest can be used when the deformity is flexible, to limit flexion of the PIP by providing a physical barrier and by unweighting the tips of the toes which are often affected either by pressure lesions of the nails or callusing at the tips of the toes. When a deformity is rigid, a toe crest can also be used to redistribute pressure away from the distal ends of the toes. Be sure not to make the toe crest too high, as it will then put pressure on the superior aspect of the toe and into the upper of the shoe. Toe crests are also helpful to decrease flexion strain on toes by providing a grip (Deschamps, 2010).

**Footwear modifications**

Footwear modification is a very common treatment for lesser toe deformity. It is vital to ensure all footwear is fitted properly, assuring a half inch is left beyond the longest toe. This is especially important with flexible deformities of the lesser toes which are reduced using metatarsal pads or bars. If the toes are flexed at the PIPs but straighten by supporting the metatarsal arch, then the toes will elongate, and may affect shoe fit. Be sure to check that the toe-box material is soft, with extra depth, and avoid stitching that may irritate the toes (AAOS, 2001). In a case where these standards are not met, or are not sufficient to accommodate the deformity, further modifications may be necessary. A vamp stretch, general or localized, is a non-invasive way to provide extra depth to adapt for lesser toe deformities, although a manual stretch may be helpful more structurally invasive methods may be necessary to accommodate some deformities (Decker & Albert, 2002). When proper fit and manual stretching is not enough, other more extreme methods of increasing
space in the toe-box include bubble patches and re-lasting (split sole). If the deformity is extremely severe or other methods have been exhausted, custom footwear can be made from a cast of the foot and designed to fit around the protruding lesser toe deformities (Cailliet, 1983). When using regular off-the-shelf footwear, discomfort accompanying lesser toe flexion deformity can also be moderated using rigid plates to stiffen the midsole of the shoe and minimize extension at the metatarsal heads. Full steel shanks and carbon fibre plates can be used beneath the sock-liner or orthosis to stiffen the midsole (Decker & Albert, 2002). Footwear with a stiffened forefoot and aggressive forefoot rocker have been shown to reduce plantar pressure beneath the forefoot, thereby reducing active lesser toe flexion during late stance and toe-off (Brown, Wertsch, Harris, Klein, & Janisse, 2004).

**Surgical intervention**

When conservative methods have been unsuccessful in relieving symptoms associated with lesser toe deformity, surgical intervention can provide joint fixation or proximal interphalangeal joint resection arthroplasty (Doty & Fogleman, 2018). While surgical interventions are typically reserved for fixed deformities with high pain scores, flexible deformities may also qualify for surgery (Cailliet, 1983). When the flexion deformity is rigid, surgical treatment often results in osseous fixation with the joint in an extended position (Dayton & Smith, 2009). When joint mobility can be preserved, proximal interphalangeal joint resection can be used with varying degrees of success (Doty & Fogleman, 2018). The actual procedure will vary according to the type and severity of the deformity (AAOS, 2001). Surgical fixation, the more frequently used procedure, is typically performed in an outpatient setting, resulting in quick return to weight-bearing activity, up to 90% success rates, and high patient satisfaction scores (AAOS, 2001; Kronkel, Menger, & Retzlaff, 2007; Kronkel, Sover, Menger, & Halfert, 2011; Doty & Fogleman, 2018). Other soft tissue surgeries may include tendon lengthening, capsulotomy, and/or ligament release (Anderson, Hall, & Martin, 2005).

**Alternative treatment methods**

Stretches and strengthening exercises such as manual toe extensions and toe curls can be used to correct or prevent the earlier stages of lesser toe deformities (AAOS, 2001). Passive stretching of the Achilles tendon and plantar fascia are also beneficial in reducing the pull on the tendons associated with lesser toe deformities (Hurwitz, 1999). Strength testing of the lesser toes is recommended to determine if there is a significant imbalance between the flexors and extensors of the toes (Kwon et al., 2009). Alternatively, off-the-shelf accommodative devices such as toe crests, cushioning, and pads can be used to relieve symptoms caused by lesser toe deformities (AAOS, 2001). In many cases, referral to a physiotherapist may complement pedorthic treatment methods.

**Key Considerations**

Although this is a very common condition in older people, the potential consequences should be taken seriously. Lesser toe deformities have been shown to affect balance, gait, and physical performance, and to be an independent risk factor for falling in older people (Menz, Morris, & Lord, 2005; Mickle et al., 2009). Lesser toe deformities can affect footwear selection, activity levels, and can have a negative impact on activities of daily living.

**References**


Hammer Toe, Claw Toe, Mallet Toe
Crossover Toe Deformity

Melissa Bendo, BKin (2012)
Updated by Kaitlyn Witkowski, BA Kin, C. Ped (C) and Ishvarjot K. Sehmbey, BSc (Kin), C. Ped (C) (2018)

Key Messages
• Most commonly the second toe medially drifts towards and eventually crosses over the great toe due to plantar plate insufficiency.
• Progressive deformity involving plantar plate tear or degeneration.
• Often incorrectly included in the umbrella term metatarsalgia.
• Conservative treatment goals are to prevent progression of the deformity and alleviate symptoms.

Keywords
dorsomedial/metatarsal subluxation, fibrocartilaginous thickening, hallux abductovarus, metatarsal joint dislocation, plantar plate, synovitis

Introduction
Crossover toe deformity was first described in the literature by Coughlin (Doty & Coughlin, 2014) in 1987 as it related to the second toe medially drifting towards the great toe, and eventually crossing over to sit on top of the great toe due to a plantar plate tear (subluxation). However, this deviation is not limited to the second metatarsal and may occur in the other lesser toes, although with less frequency (Coughlin 1993, Doty & Coughlin, 2014), and can be a result of combined sagittal and transverse plane deformities or may occur in a single plane with either dorsal or medial/lateral deviation (Coughlin et al., 2012).

The plantar plate is described as a fibrocartilaginous thickening of the metatarsophalangeal (MTP) joint capsule that is firmly attached to the base of the proximal phalanx but only loosely attached to the metatarsal head. The primary function of the plantar plate and the collateral ligaments is to stabilize the metatarsal joint. The plantar plate originates on the metatarsal head via a thin synovial attachment proximal to the articular surface and inserts on the base of the proximal phalanx. It also serves as an attachment for a number of important structures including the distal fibres of the plantar fascia, collateral ligaments, transverse metatarsal ligaments, interosseous tendons, and the fibrous sheath of the flexor tendons (Foot & Ankle Institute, 2018). Through mechanical testing, Bhatia, Myerson, Curtis, Cunningham, & Jinnah (1994) found the plantar plate to be the main stabilizing force of the MTP joint and concluded that the MTP joint collateral ligaments, which have an insertion into the plantar plate, are the second most powerful structures that stabilize the MTP joint. Coughlin, Schutt, Hirose, Kennedy, Grebing, Smith, Cooper, Golano, Viladot, & Alvarez (2012) noted that plantar plate tears of a greater magnitude were associated with greater angular deformities of the digit.

In cases where a subluxation or dislocation of one or more of the lesser MTP joints has occurred, a hammer toe deformity may be found in association with the tearing of the plantar plate, but is usually not a significant component. Also, a crossover toe deformity can be present with a hallux valgus deformity and/or a first ray insufficiency. They may contribute to the problem but not necessarily be precursors or a prerequisite (Coughlin et al., 2012).

Yu, Judge, Hudson, & Seidelmann (2002) state that this progressive subluxation or dislocation is frequently unrecognized or misdiagnosed as metatarsalgia resulting from the idiopathic inflammation of the lesser MTP joints. The resulting crossover toe deformity has been described as a common condition that can occur at any age, although it is most often seen in adults, especially women over the age of 50 (Weber, Aubin, Ledoux, & Sangeorzan, 2012).
Pathology

Researchers have hypothesized that the most common pathological indicator of instability is plantar plate insufficiency (Doty & Coughlin, 2014; Lui, 2016). A plantar plate tear can be caused by an acute trauma to the first MTP joint as a result of forced hyperextension, causing the second toe to cross over. The plantar plate ruptures, and as a result, the second MTP joint drops down and becomes fixed in that position. The patient may have an idiopathic onset secondary to chronic inflammation. Authors have proposed that overloading of the second MTP joint can result from a long second metatarsal, hypermobility of the first ray, pes planus, or genetic predisposition (Doty & Coughlin, 2014). When excessive pressure is placed on the metatarsals, the plantar plate ligament can become overloaded and tear. Consequently, the intrinsic muscles will provide a dorsiﬂexory force at the MTP joint, and in the late stages of the deformity, the lumbral muscle and toe ﬂexors will provide an eccentric pull (Klinge, McClure, Fellars, & DiGiovanni, 2014).

Instability may also be caused by hyperextension forces at the second MTP joint that may lead to attenuation of the plantar plate due to trauma or chronic inﬂammation. When trauma or chronic inﬂammation occurs at the second MTP joint, the plantar plate weakens. The main function of the plantar plate is to resist the force of toe-off and pull the proximal phalanx back into a neutral position at the MTP joint. The end result is a dorsomedial subluxation of the proximal phalanx on the metatarsal head. This whole process can progress very slowly or rapidly, depending on the individual.

The research is unclear as to the effect of foot structure on MTP dislocation (Kokubo, Hashimoto, Suda, Waseda, & Ikezawa, 2017); however, previous literature has indicated that a long second metatarsal is often cited as a common ﬁnding in individuals with instability of the second MTP joint (Weber et al., 2012). An elongated second metatarsal (or a short ﬁrst metatarsal) may alter normal foot loading patterns by transferring load from the ﬁrst to the second metatarsal head, resulting in chronic overloading to the second ray (Klinge et al., 2014). Hallux abductovalgus may also cause abnormal foot loading patterns (Coughlin & Mann, 1999). Lateral deviation of the hallux into the second digit may cause multiplanar deviation of the second toe, which results in retrograde buckling at the second MTP joint level on weight-bearing (Bhatia et al., 1994).

When the foot is placed on the ground, the crossover toe may not touch the ground, and it may shift to one side. When the toe is elevated in this way, increased pressure is placed on the head of the metatarsal from ground forces which may cause a bone bruise, pain, and/or callusing. As the deformity progresses, the cartilage in the joint becomes eroded from the malalignment, which may lead to arthritis in the joint and increased pain. This deformity is often associated with hallux valgus or a severe bunion.

Biomechanical hypermobility may also predispose a patient to altered forefoot loading patterns. Excessive pronation may alter the angle of approach of the peroneus longus tendon, which in turn alters the length of the lever arm to the axis of the ﬁrst ray in such a way that it loses its ability to stabilize the ﬁrst ray during the forefoot-loading phase of gait.

A 2012 study outlined the following risk factors for developing second MTP joint dislocation: a high body mass index (BMI), a large hallux valgus angle as well as large angles associated with the second MTP joint, and second MTP joint declination (Kokubo et al., 2017). The deformity can be found in many different demographics, but primarily affects women over the age of 50 (Weber et al., 2012).

The key developmental factor for crossover toe deformity is progressive inﬂammation in the MTP joint with subsequent attenuation and rupture of the plantar plate and collateral ligaments. Thus, any structural or biomechanical deformity that increases loading forces within the forefoot and results in inﬂammation of the plantar plate can predispose an individual to progressive weakening of the periarticular structures and joint instability (Bhatia et al., 1994; Coughlin, 1989).

Differential Diagnosis

Crossover toe is often misdiagnosed, especially in the early stages when there is pain but the toe has not yet crossed over. The pain experienced in crossover toe mimics symptoms related to Morton’s neuroma and is often included in the umbrella term of metatarsalgia.

Common Testing

Specific and accurate pain localization during a physical examination is paramount for determining the structures affected. On physical examination, excruciating pain on palpation just distal and plantar to the metatarsal head is thought to be a result of bursitis or inflammation of the plantar plate. Occasionally, inflammation of an intermetatarsal
bursa may cause local nerve irritation, resulting in neuritis or neuroma-type symptoms (Thompson & Hamilton, 1987); however, patients presenting with pain caused by inflammation of the plantar plate generally do not have subjective complaints of numbness or shooting pain, or objective sensory deficits in the affected digits on clinical examination. In addition, on physical examination, the focal point of tenderness is clearly and distinctly the MTP joint area, not the adjacent interspace (Root, Weed, & Orien, 1977).

Active flexor and extensor tendon functions of the affected toe are present with minimal or no compromise. Palpation and range of motion of the MTP joint may elicit capsular crepitus. A painful decrease in range of motion of the joint, particularly in plantar flexion, is often noted (Root et al., 1977).

Testing can be performed to confirm instability of the second MTP joint:

- A “drawer test,” similar to testing for instability in the knee. Start by performing this test on the non-affected foot to compare the instability of the affected foot. This test is performed by firmly stabilizing the second metatarsal head between the thumb and forefinger of the left hand. Grasp the base of the proximal phalanx with the thumb and forefinger of the right hand, and perform a retrograde and purely vertical (not dorsiflexion) maneuver to evaluate laxity and the ability of the MTP joint to dislocate. A positive test indicates a toe that is at high risk to dislocate, and will commonly elicit pain when dorsal/plantar stress is applied, or subluxation of the joint may occur (Coughlin et al., 2012). Gripping the second toe with one hand and the second metatarsal head with the other and moving up and down will show a laxity in the ligaments which should then be compared to the asymptomatic second MTP joint.

- For confirmation of a plantar plate tear, several imaging modalities may be required. The most practical diagnostic tool for ruling out this pathology is an X-ray of the affected MTP joint. Weight-bearing lateral X-rays clearly indicate dorsal subluxation of the proximal phalanx, and weight-bearing anteroposterior X-rays will show both dorsal and transverse deviation of the affected toe (Yu et al., 2002).

- Yu et al. (2002) also suggested a more advanced imaging modality that may be warranted when there is no clear clinical or X-ray evidence of instability. An arthrogram with iodinated contrast material injected dorsally into the MTP joint under fluoroscopy may be used to evaluate the integrity of the MTP joint capsule. A rupture of the MTP joint capsule will show leakage of the contrast material outside the joint capsule into the intermetatarsal space, indicating a collateral ligament rupture.

- Another alternative to confirm diagnosis would be the use of magnetic resonance imaging (MRI). A rupture of the plantar plate will demonstrate increased signal intensity within the plate with a loss of continuity. Synovitis of the flexor tendon sheath of the MTP joint capsule are common associated findings in plantar plate rupture and, if present, are readily identified on MRI.

Coughlin et al. (2012) outline the clinical staging system (Table 1) that is used to assess the level of crossover toe deformity:

<table>
<thead>
<tr>
<th>Grade</th>
<th>Alignment</th>
<th>Physical Examination Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No MTP joint malalignment; prodromal phase with pain but no deformity</td>
<td>MTP joint pain, thickening or swelling of the MTP joint, diminished toe purchase, negative drawer test</td>
</tr>
<tr>
<td>1</td>
<td>Mild malalignment of MTP joint; widening of web space, medial deviation</td>
<td>MTP joint pain, swelling of MTP joint, reduced toe purchase, mildly positive drawer test result (&lt;50% subluxated)</td>
</tr>
<tr>
<td>2</td>
<td>Moderate malalignment; medial, lateral, dorsal, or dorsomedial deformity, hyperextension of MTP joint</td>
<td>MTP joint pain, reduced swelling, no toe purchase, moderately positive drawer test (&gt;50% subluxated)</td>
</tr>
<tr>
<td>3</td>
<td>Severe malalignment; dorsal or dorsomedial deformity; second toe can overlap hallux; might have flexible hammer toe</td>
<td>Joint and toe pain, little swelling, no toe purchase, can dislocate MTP joint, flexible hammer toe</td>
</tr>
</tbody>
</table>

Table 1.

**Contraindications**

A cascade of deforming events can be accelerated by cortisone injections into the second MTP joint in attempts to alleviate the acute pain noted in the early and mid-term development of this condition. It has also been found that the use of high-heeled
shoes or heel lifts, which place the MTP joints in an extended position, have also been implicated in the development of crossover toe deformity and should be avoided by the patient (Coughlin, 1993).

The Patient Perspective

Pain and inflammation of the second MTP joint are the first signs of strain on the plantar plate. In a mild case, there may be little to no deformity that can be seen on the second (or affected) toe. A patient may feel as though there is not enough cushion between the head of the second metatarsal and the ground and may comment on the feeling of a “stone in the foot.” Over time, as the tear increases, the toe will begin to shift dorsally and/or medially or laterally.

In general, pain is most noticeable during ambulation and subsides during rest (Coughlin & Mann, 1999). Patients may also complain of swelling about the base of the toe, more plantarly than dorsally. Some patients will note that the toe feels as though it is “trying to go out of position,” and others may have even noted a distinct change in position over the course of several weeks.

Onset of symptoms may follow a recent increase or change in activity level. Yu et al. (2002), indicate that this problem may develop following participation in such activities as jogging, tennis, and basketball. In other cases, patients may recall a minor traumatic event, such as a misstep on a stair, stepping into a hole, or tripping on a protruding object such as a garden hose or an electrical cord prior to the development of symptoms.

In later stages, severe pain may lead to an antalgic gait with the patient compensating by walking on the lateral aspect of the foot. Barefoot weight-bearing often becomes intolerable, especially on hard surfaces. Dorsal callusing at the proximal interphalangeal joint of the affected toe are often evident (Kokubo et al., 2017).

The patient may have sought treatment for the problem in the past, after being treated by a variety of medical and surgical specialists to no avail. Treatments may have been either conservative or surgical. It is not uncommon for the patient to have received a diagnosis of second interspace or adjacent interspace neuromas, in spite of the obvious absence of the subjective complaints classically associated with neuromas.

Common Treatment

The goal of conservative treatment for crossover toe deformity is to prevent progression of the deformity and alleviate symptoms. Previously, nonsteroidal anti-inflammatory drugs and/or corticosteroids have been supported for the management of the pain and inflammation associated with this disorder; however, it should be noted that dislocation of the toe following repeated intra-articular administration of corticosteroids has been documented in the literature (Reis, Karkabi, & Zinman, 1989), along with other negative side effects (Doty & Coughlin, 2014). The physician must realize that such an injection may weaken the already compromised plantar plate, and is seen as a controversial method of treatment (Shane, Reeves, Wobst, & Thurston, 2013).
Other types of treatment consist of offloading the MTP joint and supporting the toe in terms of sagittal plane stability (Doty & Coughlin, 2014). This can be done in the early stages of the deformity through taping the affected digit and its neighbouring digit. It is believed that taping the two toes together stabilizes the digit enough to allow fibrosis of the periarticular structures to occur; however, there are two main drawbacks to taping of the digits: 1) Taping may take several months before stability and pain-free ambulation are achieved; 2) a prolonged course of taping may result in edema and possible ulceration of the digit (Doty & Coughlin, 2014). At later stages of the deformity, in which it is no longer reducible, taping is not a viable treatment method, and a medical–grade silicone toe sleeve may be utilized to reduce friction on the dorsal joint.

The literature indicates that custom foot orthoses with accommodative padding may also be used to alleviate symptoms at the affected MTP joint. The use of a metatarsal pad, possibly with a lesser MTP joint cutout (horseshoe pad) can be used to effectively decrease the weight-bearing stress on the metatarsal head. In addition, the use of a stiff forefoot plate may help to further alleviate stress through the forefoot (Doty & Coughlin, 2014). The combination of a custom foot orthosis with an extra-depth rocker-bottom shoe and wider toe box may help to decrease pain and provide further offloading (DiPreta, 2014). Wearing a shoe with a reduced heel height will also help to decrease the overall level of pressure through the symptomatic forefoot (Doty & Coughlin, 2014).

In chronic cases of severe deformity, patients who have not responded to conservative treatment may be candidates for surgical correction, in which the deformity is reduced and the affected toe is realigned into the joint and stabilized. Surgical treatment is commonly noted in the literature, but in most regions of Canada, where foot surgeries are less common than in the United States, conservative treatments are more commonly employed.

Key Considerations

Crossover toe deformity has been under-recognized as a cause of lesser metatarsalgia. Although the second digit is the most commonly and profoundly affected, the adjacent third and fourth digits can also be affected. Additionally, one or more of the lesser digits can be simultaneously involved.

Coughlin (1993) has identified two distinct populations at risk for developing instability of the second MTP joint. The first group consists mainly of sedentary women aged 50 to 70 years, with an average age of 60 years. The high proportion of elderly women in this group was attributed to the prevalent use of high-heeled shoes in this population. The second group consists predominantly of athletic men aged 25 to 64 years, with an average age of 50 years. A majority of this group were found to have a long second metatarsal. Coughlin hypothesized that, in the second group, repetitive physical activity resulted in inflammation about the second MTP joint, eventually resulting in a weakening of the periarticular structures that stabilize the toe. The author believed that both structural alteration and biomechanical dysfunction of the first ray could contribute to the development of this entity at the second MTP joint. The condition can, however, occur in the absence of either of these predisposing factors. The clinical signs and symptoms present on examination can help to stage and chart the evolution of lesser toe MTP joint subluxation and dislocation. The treatment of crossover toe deformity is dependent on the stage of the deformity.

References


Crossover Toe Deformity
Bunionette

Crystalle Ripak, BA (Kin) (2012)
Updated by David Deir, BA (Kin), C. Ped (C) (2018)

Key Messages

• Characterized by a lateral prominence of the fifth metatarsal head and a medial deviation of the fifth toe.
• Rarely symptomatic.
• Conservative treatment goals are to accommodate and offload.

Keywords
callusing, lateral prominence, medial deviation, tailor’s bunion

Introduction

A bunionette is characterized by a lateral prominence of the fifth metatarsal head and a medial deviation of the fifth toe. It is similar to a bunion but on the lateral side of the foot. It is also known as a tailor’s bunion due to the fact that it was common in tailors, who sat in a cross-legged position for long periods of time, causing them to develop this deformity (Ajis, Koti, & Maffulli, 2005).

Pathology

Bunionettes can be classified into four types (Ajis et al., 2005). Type one is an enlarged lateral surface of the fifth metatarsal head. Type two is a lateral bowing of the distal portion of the fifth metatarsal with no hypertrophy of the surrounding soft tissue. Type three is an increase in metatarsal angle. Finally, type four is a deformity that includes two or more of the previously listed types. Determining the type of bunionette makes it easier to determine the proper and most effective treatment for the patient. The higher the classification of bunionette, the more likely that it will not respond to regular conservative treatment, and surgical referral may be indicated (Ajis et al., 2005).

Bunionettes are common in adolescents and adults, with women reporting them at least twice as often (Thomas, Blitch, Chaney, Dinucci, Eickmeier, Rubin, Stapp, & Vanore, 2009) and up to 10 times as often (Cooper, 2010). In athletes with pes cavus and a forefoot splay it is also common to develop bunionettes (Lorimer, French, O’Donnell, Burrow, & Wall, 2006).

Determining a singular cause of bunionette may not be possible, as most of the literature deems it multifactorial. The various causes can be separated into structural and biomechanical causes. Structural causes include shape and length variations, including lateral bowing of the fifth metatarsal (Thomas et al., 2009), dumbbell shape, and a short fifth metatarsal (Ajis et al., 2005). Other structural causes include uncompensated rearfoot varus (Lorimer et al., 2006), plantar flexed first ray, forefoot valgus (Michaud, 2011), and increased deviation between the fourth and fifth metatarsals (Ajis et al., 2005). Two-thirds of the population with bunionettes are also said to have pes planus (Cooper, 2010).
Biomechanical causes include: abnormal pronation due to hypermobility in the midtarsal and subtalar joints (Ajis et al., 2005); splay forefoot (Thomas et al., 2009); ill-fitting, tight footwear; and footwear that is unyielding (Lorimer et al., 2006). Certain foot positions that are maintained over long periods of time, like sitting tailor fashion, are also considered a cause (Ajis et al., 2005). Biomechanically, activities that increase the pressure and load on the fifth metatarsal are listed as causes of bunionettes. Bunions may also be co-occurring with bunionettes as they can have the same root causes.

**Differential Diagnosis**

Bunionettes may not be the only cause of pain and discomfort in the area of the fifth metatarsal head. If the onset of pain was sudden after a trauma, then referral to a doctor and an X-ray would be best to rule out the possibility of a fracture (Lorimer et al., 2006). If the level of deformity is minimal but there is localized inflammation without callusing, then bursitis or capsulitis could be present (Thomas et al., 2009). Inquire about painful or limited motion from increased pressure to the fifth metatarsal. A thick callus could cause pain and discomfort; referral to a medical professional (family physician, foot care nurse, podiatrist, chiropodist) to debride the callus could increase comfort. Debriding the callus will only be a temporary solution if the root biomechanical cause of the irritation is not addressed, as it will continue to form.

**Common Testing**

Before starting any testing and evaluation, a thorough history should be taken. History of the pathology would include questions regarding the onset, duration, intensity, and location of any pain. Past injury or surgery to the back, lower limb, or foot may have caused a change in gait, thereby affecting the bunionette area. Determine what styles of footwear are worn on a regular basis for specific activities of daily work and leisure. Determine activity level, type, intensity, and frequency for work and leisure. A thorough history will give the clinician insight into the possible mechanisms of injury and inform an appropriate treatment plan.

Evaluate patient footwear for proper fit by reviewing previously worn footwear and those that the patient came in wearing. If a large deformity is present, wear patterns should be evident on/in the footwear. If there is a large exostosis and lateral deviation of the fifth metatarsal head, the upper may look stretched or misshapen and the upper lining may be damaged or worn away. If there is a plantar flexed fifth metatarsal head or callus on the plantar surface, a depression under that area may be on the insole of the shoe.

Look for signs of redness and swelling around the fifth metatarsal head indicating that there is inflammation present. Look for signs of callusing and the location of it. A callus on the plantar surface suggests increased loading and pressure from gait, an enlarged fifth metatarsal head, a plantar flexed fifth metatarsal, or a combination of the three. Callusing on the lateral aspect of the fifth metatarsal head could indicate increased rubbing against the footwear, or that during gait weight is staying on the lateral side of the foot. To have a closer look at the pressure distribution on the plantar surface of the foot a Harris Mat impression could be taken both statically and dynamically (Cooper, 2010).

When evaluating joint range of motion look for hyper- and hypomobility. Rigidity may suggest the presence of arthritis in the joint (Thomas et al., 2009) as increased swelling in the area may impede range of motion. Patients with rheumatoid arthritis commonly have bunionettes (Ajis et al., 2005). Hypermobility in the foot can be an indicator of possible increased pronation and other biomechanical issues.

Perform gait analysis to determine if hypermobility of the subtalar and midtarsal joints are causing increased pronation, one of the causes of bunionettes (Ajis et al., 2005). Also check to determine if there is an uncompensated rearfoot varus (Lorimer et al., 2006), plantar flexed first ray, or forefoot valgus (Michaud, 1997) causing increased loading to the lateral side of the foot.

**Contraindications**

Patients with diabetes should be treated with caution due to the high-risk nature of the disease. Because neuropathy or loss of feeling can develop in diabetics, these patients may have gait alterations, bony deformities, muscle imbalances, or other biomechanical problems. They may also be wearing inappropriately fitting footwear, as feelings of light touch may not be appreciated and they have chosen tight-fitting shoes. Checking for neuropathy with a 10-gram monofilament or referral to their doctor to have their sensation checked is advised.

**The Patient Perspective**

This morphology is rarely symptomatic. Patients will seek treatment when it becomes symptomatic, when
they are unhappy with its visual appearance (Ajis et al., 2005; Cooper, 2010) or when it hampers wearing their desired footwear. Bunionette pain is location specific—pain in and around the affected fifth MTP joint. It is common for the patient to complain of pain when wearing certain types of footwear, with relief upon the removal of said footwear.

**Common Treatment**

While most patients presenting with bunionettes are asymptomatic, patient education is key to maintaining the absence of pain (Thomas et al., 2009). This could include talking about proper selection of footwear, proper fit of footwear, and possible footwear modifications to aid in offloading and pressure relief of the bunionette. When bunionettes are symptomatic or treatment is desired to slow its progression, conservative treatment can be utilized. This can include footwear, footwear modifications, orthoses (custom made and over the counter), activity modifications, NSAIDs, and referral to other practitioners.

Footwear that is ill-fitting and tight should be avoided, as it is one of the contributing factors of developing a bunionette deformity. Width is of particular importance when it comes to evaluating footwear. The bunionette is generally the widest part of the foot, thus it should sit in the widest part of the shoe. The upper material that sits over the bunionette should not be strained or taut as this would indicate that it is exerting increased pressure on the bunionette. With regards to shape, a deep and wide toe box is best (Sferra & Shute, 1999). A deep and wide shoe may allow for enough room to house the bunionette deformity with ease. Shoes with pointed toes should be avoided; look for a rounded toe box. Ideally the upper should be seamless in the forefoot area (Porter & Schon, 2008). Encourage the patient to also feel inside the shoe to ensure that the lining of the upper is smooth and that there are no interior seams or folds that could cause irritation to the bunionette. High heels cause more pressure to be shifted forward onto the forefoot and can cause increased pain in the bunionette. While eliminating wearing high heels may be an unrealistic expectation for some female patients, a decrease in the height of the heel and duration of wear (Ajis et al., 2005) could be a compromise.

Matching footwear to activity is important. Both the style of shoe and the duration of wear should be taken into account. To create more realistic expectations for the patient, the pedorthist should stress that the more one wears improper footwear, the more likely that pain and discomfort will continue.

Footwear modifications aid in the relief of bunionette pain, and may increase the suitability of footwear. Footwear modifications for bunionettes include stretching, padding, and excavating. Stretching is to decrease pressure of the upper on the bunionette (Porter & Schon, 2008). Stretching can be done in two ways: an all over or a point stretch. Stretching the upper width in the forefoot will allow more room for the bunionette. If the upper is tight all over, a general stretch of the width is best. Point stretching will stretch only a small targeted area of the upper, basically creating a pocket for the bunionette to sit in. It is usually done with a bunion stretcher, or a ball and ring stretcher. With point stretching it is best to have the patient don the footwear so you can see the specific location that needs to be stretched. If stretching is not enough, then a balloon patch may be of benefit. Take into consideration the upper material and if there are any seams present. Soft leather may stretch very easily, so care should be taken that it is not overstretched. Materials such as mesh and fabric do not respond to stretching very well and may tear under pressure.

The use of padding is generally said to decrease the likelihood of callus formation (Porter & Schon, 2008). In footwear that has a thin mid- and outsole, the addition of padding under the forefoot can make the shoe more comfortable. Be mindful that adding padding inside the footwear decreases the volume of the forefoot space and my make the shoe feel tighter. If too much padding is added, pain could increase as the bunionette experiences greater pressure exerted from the footwear’s upper. A combination of properly increasing the padding and stretching the footwear to accommodate the bunionette may reduce symptoms. Adding padding along the lateral side of the fifth metatarsal ending just before the metatarsal head may aid in decreasing pressure on the bunionette. This will more evenly distribute the pressure of the upper along the whole length of the fifth metatarsal.

Midsole excavation under the fifth metatarsal head can provide symptom relief. This excavation increases the volume of the toe box slightly by allowing the metatarsal head to sit in a depression in the midsole. Due to the location of the excavation in the shoe, care needs to be taken that the upper material along the lateral edge remains well adhered. The excavation may be left as a small depression inside the shoe or can be filled with a soft material to provide increased padding. In either case, ensure that there are no edges
or areas that are considerably uneven or sharp that could become areas of irritation.

The use of orthoses can be beneficial in providing relief when biomechanics are contributing to the bunionette pain. The use of an over-the-counter (OTC) or custom-made foot orthoses will depend on the patient’s pain and deformity. For patients with uncompensated rearfoot varus, accommodative orthoses are recommended (Lorimer et al., 2006), as the main goal will be to redistribute weight on the plantar surface of the foot; however, most patients would benefit from semirigid orthoses (Ajis et al., 2005), primarily to control any abnormal motion of the foot such as pronation (Michaud, 2011).

Posting is frequently used to aid in the control of abnormal foot motion. In patients with forefoot valgus, it is recommended to add a forefoot valgus post to keep the subtalar joint in a neutral position while maintaining lateral stability (Michaud, 2011). To maintain depth in the toe box of the footwear, use of intrinsic forefoot posting is recommended when possible. If extrinsic posting is needed, extending and tapering it to sulcus length could provide a smoother transition. The extrinsic post could also be modified to include offloading properties such as a minor excavation under the fifth metatarsal head for increased offloading.

To increase the offloading properties of the orthoses, addition of a metatarsal pad or bar to appropriate (Ajis et al., 2005). The metatarsal addition increases contact with the shafts of the metatarsals, thus redistributing the load from the distal heads more uniformly along the entire length. To achieve this, the apex of the metatarsal pad should be placed just proximal to the metatarsal heads (Michaud, 2011). To provide increased offloading with an enlarged or plantar flexed fifth metatarsal head, a 1–4 bar may be used.

Athletic patients that are experiencing pain may benefit from short-term modifications to their training routines; talk to them about substituting some low- or non-impact activities for high-impact ones or reducing workout times (Porter & Schon, 2008). In conjunction with utilizing some of the other conservative treatments, decreasing the load on the bunionette should decrease irritation and provide a better environment for healing. Once the pain has subsided and the patient has adjusted to the changes made in footwear, a slow transition back to the usual training activities is best, and if pain recurs, the activity modification may need to be more permanent.

There are a few circumstances where referral to another practitioner is warranted. If there is suspicion of a fracture, referral to a doctor for X-rays is critical. If callus formation is problematic, an appropriate medical professional should perform callus debridement. A physiotherapist or athletic therapist can create modified or low impact workout routines for the athletic patient.

When conservative treatment fails, referral to an orthopaedic surgeon is warranted. The goal of surgical treatment is to decrease the prominence of the fifth metatarsal head laterally (Thomas et al., 2009). Although surgery is uncommon, there are numerous types of surgery that can be performed, depending on the patient’s presentation. It is of benefit to note that not all patients that undergo surgery find full relief of their symptoms. One study found that 13% of patients still had pain after the surgery, and 28% still needed comfortable footwear and were not able to tolerate fashionable, conventional footwear (London et al., 2003). Thus, once a patient is referred to a surgeon, the role of the pedorthist is not over, as patients may still need ongoing care.

**Key Considerations**

Bunionettes, while rarely symptomatic, can present in a wide number of adult patients, due to a multitude of causes. Patient education is key, with appropriate selection of properly fitting footwear being the consistent message. Conservative treatments revolve around both accommodation and offloading of the bunionette using footwear modifications, alone or in combination with custom or over-the-counter foot orthoses. Most patients respond to conservative treatment, but in some cases, referral to a surgeon may be needed.

**References**


Interdigital (Soft) Corns

Nancy Kelly, BSc (Kin), C. Ped Tech (C), C. Ped (C)

Key Messages

- Goal of all treatments is to reduce interdigital pressures.
- Footwear is a key factor in preventing and treating interdigital corns.
- In some cases, referral to a footcare nurse, podiatrist, and/or surgical consultations by a physician might be necessary.

Keywords
abscess, exostosis, hallux abductovalgus, heloma molle, hyperkeratotic, hypertrophied condyles, interdigital pressure, ulceration

Introduction

An interdigital or soft corn is a variant of corn, also known as a heloma molle (Decker & Albert, 2002). Interdigital corns can occur in any interdigital space, but are most commonly found between the fourth and fifth digits (Dawber, Bristow, & Turner, 2001). Increased friction and pressure in the interdigital space, stemming from a number of intrinsic and extrinsic factors (ePodiatry.com, 2003; Singh, Bentley, & Trevino, 1996), can result in the accumulation of hyperkeratotic tissue or a corn (Day, Reyzelman, & Harkless, 1996). The interdigital corn typically has a macerated (Freeman, 2002) or rubbery appearance, resulting from the trapping of moisture in the interdigital spaces (Dawber et al., 2001).

Although interdigital corns can occur in both sexes, a higher incidence has been found in women (Coughlin, 2006). Interdigital corns have also been found to be more prevalent in middle aged and older populations (Dawber et al., 2001).

Pathology

Corns occur in response to pressure or friction in an area of the foot (ePodiatry.com, 2003; Singh et al., 1996). The corn itself is a thickening or accumulation of spongy, hyperkeratotic tissue (Day et al., 1996) which serves as a protection to the underlying structures (ePodiatry.com, 2003). In the case of interdigital or soft corns, this lesion occurs between adjacent digits in the region around the condyles of the phalanges or interphalangeal joints, where there are hard or bony prominences (Dawber et al., 2001; Singh et al., 1996; Day et al., 1996). Interdigital corns can also occur within the sulcus, which are referred to as webspace corns (Day et al., 1996). The softer or rubbery texture of an interdigital corn occurs as a result of the build-up of moisture between the digits (Dawber et al., 2001). The thickening of the skin resulting from being trapped between opposing hard surfaces can eventually result in pain, and the body will begin to treat this area as a foreign body (ePodiatry.com, 2003). Should this go untreated, ulcerations or abscesses could develop. This can then result in infection, which could be particularly problematic for those with peripheral neuropathy, diabetes, and compromised circulation (ePodiatry.com, 2003).

There are several intrinsic and extrinsic factors that can contribute to the increased friction and pressure within the interdigital spaces, resulting in the formation of an interdigital corn. Structures of the foot that contribute to increased pressure between joint surfaces include “…hypertrophied condyles, exostosis, ankylosis of the fifth interphalangeal joint, unreduced fractures, or dislocated digits” (Day et al., 1996). Adductovarus of the fifth, hallux abductovalgus, an enlarged fifth metatarsal head, and positional deformities of the digits (Day et al., 1996) can also contribute to increased pressure between the joint surfaces in the interdigital spaces. It is thought that length of the metatarsals and phalanges can contribute to the formation of interdigital corns. However, an analysis of patients with webspace corns performed by Zeringue and Harkless “…found no correlation with fourth intermetatarsal angle, relative length of the fourth and fifth metatarsals, ankylosis
of the fifth distal interphalangeal joint, adductovarus rotation of the fifth toe, metatarsus adductus angle, or forefoot width” (Day et al., 1996). Hallux rigidus, positional toe deformities, and altered foot mechanics have also been cited as intrinsic factors, contributing to callosities.

Footwear is the primary extrinsic factor contributing to the formation of interdigital corns. Footwear is felt to be a key factor in various forefoot pathologies including corns (Menz & Morris, 2005). Ill-fitting footwear has been found to be significant in incidence of foot pain and ulcerations (Menz, & Morris, 2005), as this can result in decreased air flow and increased interdigital pressures (Dawber et al., 2001). Fashion footwear, in particular, has been considered a cause of interdigital corns due to the high incidence of corns in women (Coughlin, 2002). Tight socks or stockings can also contribute to closed and high pressure interdigital conditions (Dawber et al., 2001).

Interdigital corns have been found to be more painful than hard corns (Dawber et al., 2001). Lateral compression of the digits will often provoke pain (Dawber et al., 2001). As the corns result from a thickening of skin there is potential for this area to ulcerate (ePodiatry.com, 2003). This is a serious complication, particularly for those with any form of peripheral neuropathy, compromised circulation, and/or diabetes (ePodiatry.com, 2003).

Differential Diagnosis

Corns might be misdiagnosed as plantar warts. Dawber et al. (2001) described the following features of corns, which will assist in a differential diagnosis from plantar warts: corns primarily occur in areas of high pressure; pain is provoked with direct pressure to the affected area; and skin lines will continue over the lesion. The reduction or elimination of the causative pressure is necessary to resolve the interdigital corn. According to Coughlin (2002) and Day et al. (1996), interdigital corns might be misdiagnosed as fungal infections, due to the macerated appearance of the tissue of the interdigital space. For example, if a patient were to complain of itching in the interdigital space in the presence of maceration, they might not have an interdigital corn, but instead have athlete's foot or tinea pedis (Day et al., 1996).

Common Testing

The most common testing for interdigital corns is to palpate the region under the lesion. According to Day et al. (1996), “…if one can palpate a firm bony prominence underneath the macerated lesion and it is painful to palpate, the lesion is a soft corn until proven otherwise.” Day et al. (1996) have also suggested that radiographs can be used in the diagnosis of interdigital corns and that shod radiographs can indicate changes in the alignment of the digits due to external forces.

Contraindications

Infection is a concern with soft corns, as the body might start to treat the accumulation of skin as a foreign body and it might eventually develop into an ulcer (ePodiatry.com, 2003). According to the Podiatry Network (2017), “over-the-counter corn removers should never be used in this area because of the risk of increased damage to the skin resulting in infection.”

The Patient Perspective

Patients with interdigital soft corns might complain of pain or discomfort at the medial or lateral aspect of a digit. However, not all patients will report pain. Pain can be provoked by increased pressure in this region, for example from tight-fitting socks and/or footwear.

Common Treatment

There are a number of treatments for interdigital soft corns ranging from conservative measures to more invasive, surgical procedures.

The goal of many of the conservative measures is to decrease pressure in the interdigital spaces. There are a number of methods that can be implemented to reach this goal.

Toe spacers can be used to decrease the friction and pressure in the interdigital spaces. Toe spacers can be purchased over-the-counter or fashioned from a variety of materials. Mass-produced toe spacers...
are commonly made from silicone. Cotton and foam-based materials can also be used to decrease the interdigital pressure (Podiatry Network, 2017). Mouldable putty can be used to construct a custom toe spacer. Silicone toe sleeves are also used to decrease the pressure on the bony surfaces between the digits.

As footwear can be a contributing factor to the formation of interdigital corns, appropriately fitting footwear is an integral piece of the treatment plan. Footwear should be wide and roomy in the toe-box (Coughlin, 2002; Dawber et al., 2001; ePodiatry.com, 2003), which will serve to decrease compression of the digits and pressure between the digits. Soft upper materials and lower heel height are also desirable features of footwear to treat and avoid interdigital corns (Singh et al., 1996).

Proper hygiene is key when treating interdigital corns, as these soft corns have potential to become infected and develop into ulcers. There are several accepted practices when it comes to footcare and hygiene, some of which may not be appropriate for all populations, for example those with peripheral neuropathy and/or diabetes. Emollients can be helpful in maintaining the condition of the skin (ePodiatry.com, 2003) and anti-bacterial, anti-fungal products used after cleansing can help to reduce the likelihood of infections (Hogan, 2016).

Tight-fitting socks or hosiery have also been identified as contributing factors of soft corns as wearing them can result in increased pressure in the interdigital space (Dawber et al., 2001). They should be avoided by people with interdigital corns. Therefore, changing the type of hosiery or socks being worn has also become an accepted part of the treatment plan for addressing soft corns. Socks made from materials that are breathable and/or have moisture-wicking technology can be effective in reducing the accumulation of moisture in the interdigital spaces which in turn will assist in prevention of maceration of the tissues.

Debridement or trimming down of the affected area by a trained footcare professional (e.g., podiatrist, chiropodist, or footcare nurse) can be an appropriate referral for those with interdigital corns. This form of footcare is particularly recommended for diabetic patients (Podiatry Network, 2017).

In some cases of interdigital corns, more invasive surgical procedures are necessary. The ultimate goal of these surgical treatments is to reduce the physical cause of the high pressure in the interdigital space. There are several options for surgical procedures. Removal of prominent condyles or irregularly shaped bones can reduce high interdigital pressures (Coughlin, 2002). Along with condylectomy, which is more common, syndactylization or fusion of adjacent digits can be performed as a way to address the structural considerations (Coughlin, 2002; Day et al., 1996). Fusions can also be used in the treatment of those soft corns that occur in the webspaces (Coughlin, 2002). In rare cases of interdigital soft corns, amputation of a digit might be needed (Coughlin, 2002).

**Key Considerations**

Footwear is a key consideration in the formation of interdigital soft corns, as this is a primary factor in high interdigital pressures, which can result in soft corns (Menz & Morris, 2005). Dawber (2001) also identified that corns are more common in middle-aged and older populations.

**References**


Intractable Plantar Keratosis (Hard Corn), Callus, Plantar Wart

Jodi Basha, BPE, Dip Ped, C. Ped (C)

Key Messages

- Hard corns are usually 1cm or less in diameter, and are usually cone shaped.
- Calluses can range from small to large, even across the entire plantar forefoot. Their borders are undefined.
- Plantar warts are the result of a viral infection and present with a disruption in the skin lines along with a dark central core (hemorrhage).
- Plantar warts have a rapid onset, whereas corns and calluses develop gradually.

Keywords

bony prominence, bubble/balloon patches, callosities, contagious, corns, epithelial tumor, excavation, hyperkeratosis, planaris, toe crests, viral infection

Introduction

The feet are our primary contact with the ground and as such they are exposed to unique pressures and forces. These frictional and other forces often result in the formation of corns and calluses on the toes and plantar aspect of the foot. Warts are a common foot complaint affecting the bottom of the foot and the toes, though they do not share the same causes as corns and calluses. A callus is a thickened area of skin which usually forms on the sole of the foot (Decker & Albert, 2002) or other areas of high pressure. A hard corn is a dry, thick mass of hyperkeratosis with a hard central core, commonly found on the dorsolateral aspect of the fifth toe, the dorsum of the interphalangeal joints of the lesser toes (Scheinfeld, Hyer, & Loebenburg, 2007; Freeman, 2002), and the plantar forefoot (Kang, Chen, Chen, & Hsi, 2006). These skin pathologies result from areas of high friction and/or pressure often experienced at the forefoot or around the heel due to mechanical foot abnormalities, ill-fitting footwear, or increased activity. The longer the pressure continues, the worse the callosity will become. These conditions generally form on the fatty pads of the bottom of the foot or on the tips and sides of the toes, especially the fifth toe, where continual shearing forces take place.

A plantar wart occurs on the sole of the foot and is caused by direct contact with HPV (human papilloma virus), a viral infection that is transferred between people through skin-to-skin contact. A plantar wart will routinely form on areas of high pressure on the plantar aspect of the foot, mainly the ball or heel of the foot. Repetitive increased pressure can cause the wart to develop a painful callus around the soft centre which may attack blood vessels deep into the skin, becoming extremely painful. A hard corn, or keratosis, has a small defined area of extremely thickened skin, whereas a callus has borders that are much less defined and is generally not as thick. In both conditions, however, the skin lines run through the thickened skin. A plantar wart is noticeably different in that the skin lines surround the viral mass, as opposed to running through or over it. However, in areas of weight-bearing, a plantar wart may be masked by the thickened skin present over it.

Callosities become a problem when the pressure persists and the skin continues to become thicker. According to Scheinfeld et al. (2017), these callosities can limit ambulation and cause changes in gait; if the callosity is present directly beneath a weight-bearing area of the foot, then compensatory changes in gait patterns can occur due to functional limitations. Hard surfaces and footwear can further irritate
these conditions. With a continual shearing force followed by a viral infection, a plantar wart can occur at these high-pressure areas, thus causing the same compensatory gait changes due to pain and/or pressure.

According to Scheinfeld et al. (2017), intractable plantar keratosis is not uncommon; however, Spink, Menz, & Lord (2009) indicated that there is very little information documented in relation to the frequency of corns and calluses. Furthermore, Silverberg (2017) stated that corns are among the most common foot conditions in the United States and that they are usually more common among the elderly population. It was also noted that corns and calluses are common around the world among all individuals who are able to bear weight.

Plantar warts are usually picked up in areas where people are more likely to walk around barefoot (i.e., swimming pools), where the viral infection is usually spread. These warts are typically most common among children and teenagers.

**Pathology**

The skin on our feet needs to withstand frictional and other forces, and the anatomy of the skin reflects this need. Skin is composed of two layers, the dermis and the epidermis. The epidermis is the outermost protective layer; the dermis lies under the epidermis and is a thicker, tougher layer of fibrous connective tissue. The skin on the feet is required to combat frequent friction and does so by accelerating cell production and the formation of keratin, a fibrous protein that helps protect tissues. Persistent friction causes a protective response to occur where the epidermis layer of the skin thickens, causing a callus. Corns and calluses are both a direct result of hyperkeratosis (abnormal thickening of the outer layer of the skin), which is a normal physiological response to chronic excessive pressure or friction on the skin. What may start off as a mild callus can lead to a corn if pressure continues. In both cases, the skin is unable to shed, and the intact skin continues to thicken and can become very painful if left untreated (Decker & Albert, 2002). Once the thickened skin forms, whether due to ill-fitting footwear or mechanical abnormalities, it then causes more pressure in the shoe. A vicious cycle then ensues which can result in tremendous pain as well as an increase in the size of the callosity (Singh et al., 2009).

Calluses form as a means of protection against shearing or compressive forces (Singh, Bentley, & Trevino, 2009). There are numerous causes of callus formation, including mechanical abnormalities, prominent metatarsal heads, ill-fitting footwear, cavus foot type, dropped transverse metatarsal arches, hammer toe deformities, long lesser metatarsals, hypertrophic plantar metatarsal head condyles, malunion of a metatarsal fracture, and accessory sesamoids. Calluses may also form as a result of a hypermobile first ray as well as a hallux with limited range of motion, as pressure shifts laterally, thus increasing the amount of pressure under the lesser metatarsals (Scheinfeld et al., 2017; Decker & Albert, 2002). Another cause is overpronation, which forces too much pressure to be shifted medially during toe-off, causing a callosity to develop on the medial side of the hallux. A callus can become so severe that it cracks, causing pain and even sometimes leading to infection (DeMello, 2009).

Corns are very similar to calluses, except that they have more of a cone-shaped localized hardening and, unlike calluses, they have a tendency to press against the nerves located within the foot, especially in cases of ill-fitting footwear (DeMello, 2009). According to Scheinfeld et al. (2017), intractable plantar keratosis usually approximates 1cm or less in diameter. This discrete hard area on the skin is typically caused by wearing footwear too tight or too loose, footwear with very pronounced seams, malaligned metatarsal
phalangeal or interphalangeal joints; hammer, claw, or mallet toes; or prominent underlying bones (Decker & Albert, 2002). Corns are formed when the skin’s epidermis accumulates to protect the foot from external or underlying forces.

Plantar warts differentiate themselves from calluses and corns. Plantar warts are benign epithelial tumors that occur when the skin encounters a viral infection, commonly occurring when the contagious virus (human papilloma virus) comes in contact with an existing crack or break in the skin. Plantar warts are usually covered by a thick layer of callus, and may appear as a single wart or a group of warts (referred to as a cluster). Typically, there is a clear disruption in the skin lines and often times a dark central core (hemorrhage) is present within a plantar wart (Decker & Albert, 2002).

**Differential Diagnosis**

Corns, calluses, and warts need to be differentiated from other conditions of the skin of the foot. DeHeer, Leshikar, & Patel (2016) emphasize the importance of differentiating biomechanical and dermatological plantar keratosis. Dermatological conditions that present as similar to keratosis include psoriasis, foreign bodies in the skin, porokeratosis, skin cancers, keratoderma plantaris and punctate keratoderma plantaris, and plantar scars. Calluses and warts are commonly confused with each other. Treatment for warts differs from callus treatment, and therefore diagnosing each correctly aids effective treatment.

**Common Testing**

According to Singh et al. (1996), warts have a rapid onset, whereas corns and calluses develop gradually. Warts may or may not be under bony prominences, but corns and calluses are usually directly against a bony prominence. Corns are usually on the interphalangeal joints or on the sides of the toes, while calluses are usually on the plantar aspect of a bony prominence, such as a prominent metatarsal head or an area which experiences greater friction. Pain can be reproduced with side to side squeezing if a wart is present, but direct pressure on a corn or callus will cause pain. The direct location of the pain, along with a thorough biomechanical evaluation, gait analysis, and footwear evaluation can be a good indicator of which condition exits.

**Contraindications**

Avoidance of surgical treatment is strongly recommended when there is infection, vascular insufficiency, painless lesions, or neuropathy, such as that commonly found among diabetics (Scheinfeld et al., 2017). Diabetics have to be especially careful, as even too much filing of a corn or callus can cause an infection which can lead to ulcers and potential amputation. Great care must be taken when treating one of these conditions in persons with diabetes, due to possible neuropathy and poor blood supply which can delay healing and lead to other complications.

According to Scheinfeld et al. (2017), injectable anti-inflammatory steroids to or around an intractable plantar keratosis should not be recommended. This can lead to another painful condition known as fat pad atrophy, which can further irritate and cause pain to the foot.

**Common Treatment**

There are many methods of treatment for corns, calluses, and warts, and so the treatment chosen is often dependent on the severity of the condition.
If left untreated, pain can become so severe that it can affect a person’s gait as well as which types of activities they decide to partake in (Singh et al., 1996). According to Singh et al. (1996), treatment should provide symptomatic relief and alleviate the underlying cause.

The first step in treating the condition involves finding the underlying cause. Footwear should fit properly. If mechanical plantar pressure exists, proper foot orthoses should be fitted to evenly distribute pressure among the foot and re-distribute abnormal mechanical forces to the body (Singh et al., 1996). Metatarsal pads and metatarsal bars positioned properly on the orthoses, to offload pressure on the weight-bearing plantar aspect of the forefoot, can help significantly (Lee, Landorf, Bonanno, & Menz, 2014; Kang et al., 2006; Decker & Albert, 2002). Additional padding may be necessary, depending on the severity of the condition. Plastazote top covers and excavation under the high-pressure area will also help alleviate pain and decrease pressure in addition to wearing rocker soled footwear (Decker & Albert, 2002).

If calluses and corns are present at the forefoot, properly fitting footwear should consist of soft heeled, soft seamless uppers, and an extra-deep, roomy toe-box. Some general modifications to footwear to further address the problem might consist of stretching the footwear to provide adequate room in the toe-box, spot stretching directly above or alongside the area of pressure with a ball-and-ring stretcher, and forefoot rockers to help decrease plantar pressure during propulsion (Freeman, 2002). Further footwear modifications to offload corns consist of bubble/balloon patches, excavations, metatarsal pads, and toe crests (Decker & Albert, 2002). Footwear modifications for treating calluses also consist of tongue pads, lacing techniques to avoid direct pressure, metatarsal pads, excavations to the insole or the orthosis, internal and external wedges to correct foot mechanics, and soft top covers for cushioning (Decker & Albert, 2002).

Corns can be treated through the use of creams containing salicylic acid, found in over-the-counter products or provided by a physician or other medical professional (chiropractor or podiatrist). Podiatrists, however, do not usually recommend using chemicals for removal of corns as they can burn the skin (DeMello, 2009).

Calluses can often be treated or lessened by lubricating the area with lotion containing urea or salicylic acid. This increases the amount of water in the skin and helps to soften the callus. Soaking the feet in lukewarm water or a footbath and then scrubbing the calluses with a pumice stone or emery board can help remove the dead skin. Ped Eggs and callus scrapers are over-the-counter devices which can also be used to aid in the removal of dead skin; however, care must be taken when using sharp objects that could possibly cut the skin. Some podiatrists believe this makes the skin more vulnerable (DeMello, 2009). Regular maintenance to keep the callosity reduced is an important part of managing the condition.

When a plantar wart exists, the patient should seek treatment through their family physician or footcare specialist. Plantar warts are most commonly treated using over-the-counter wart removal products such as wart medication with salicylic acid (over the counter or prescribed by a physician or other health care professional), cryotherapy, acid treatments, laser treatments, and minor surgeries. Proper offloading of the wart by wearing proper fitting extra-depth footwear, and accommodative orthoses with soft top covers, metatarsal pads, and rocker soles can aid in treatment as well as providing relief from a painful plantar wart. The spread of plantar warts can often be prevented by wearing footwear when around areas of high-barefoot traffic (swimming pools, playrooms, gymnasium changing rooms).

For patients suffering from one or more of these conditions, proper supportive footwear worn around the house to offload abnormal pressure is a great asset. In order to pare down the thickened lesion, a footcare nurse, chiropodist, or podiatrist can debride the thickened skin, offering relief of pressure on the affected area. The debrideement often needs to be repeated on a regular basis. This is especially beneficial for people who have limited mobility and are unable to reach the affected area themselves, or if the lesion is too thick to manage at home.

If a keratotic lesion still persists and continues to be painful, then surgery may be required to relieve pressure cause by a bony prominence (Scheinfield, Hyer, Loebenberg, & Stephens, 2017). Most often, hard corns and calluses are treated without having to turn to surgery (Scheinfield et al., 2017). If they do not go away with a more conservative method of treatment, then surgery may be required to treat the condition. Such surgeries may include partial metatarsal excisions, metatarsal osteotomies, or sesamoid surgery (Scheinfield et al., 2017; Scheinfeld et al., 2017).
The Patient Perspective

With intractable plantar keratosis, patients usually experience a lot of pressure under the affected area and they usually describe the area as being very sharp and painful and having the same feeling as “walking on a marble” (Scheinfeld et al., 2017). Patients will also pinpoint localized pain directly above or below the affected area on the foot. They may even be able to describe the pain as occurring during certain movements, both static and dynamic, as well as when wearing certain footwear.

Key Considerations

While calluses and corns are the most common foot problem (Decker & Albert, 2002), according to a study done by Spink et al. (2009), intractable plantar keratosis affects approximately 60% of the elderly population. It is usually a result of abnormal biomechanics, including hallux valgus toe deformities and time spent on feet. Corns and calluses are more common among females. This could be due to the fact that females are more likely to wear ill-fitting footwear such as high heels or pointy-toed shoes. Plantar warts, on the other hand, are most common among children and adolescents (Decker & Albert, 2002). It is easy to pick up this virus in and around swimming pools, gymnasium changing rooms at school, or anywhere else where people are more likely to go barefoot.

References


Hallux Valgus and Bunion

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Key Messages

- Family history is a greater factor (88%) than ill-fitting shoes for the cause of hallux valgus; poor biomechanics of the first MTP joint are the main contributing factor.
- Hallux valgus and bunions may not cause pain unless wearing tight/inappropriate footwear.
- Pedorthic treatments include custom foot orthoses, which control the underlying poor biomechanics of the foot, as well as proper-fitting footwear and footwear modifications, which reduce pressure on the bunions.

Keywords

angulation, bunion, family history, great toe, hallux valgus angle, hypermobile, intermetatarsal angle, lateral shift, metatarsocuneiform joint, metatarsophalangeal joint, osteotomy, plantar flexed, subluxation

Introduction

Hallux valgus (HV) is a lateral deviation of the distal hallux at the first metatarsophalangeal (MTP) joint toward the lateral foot (Alexander, 1997; Ferrari, 2014). Hallux valgus is a very common finding in the general population with 2% of children aged 9 to 10 showing evidence of hallux valgus and half of adults (Ferrari J. 2014). Hallux valgus is also commonly associated with bunion. A bunion is defined as a medial bony prominence of the first metatarsal head (Alexander, 1997). The first MTP joint is composed of the first metatarsal and the proximal phalanx, and the hallux is composed of the distal and proximal phalanx (Northcoast Footcare, 2011).

Hallux valgus and bunion are a common foot deformity seen in the pedorthic clinic. Most patients who present with this deformity are women, and though past literature has blamed poor fitting high heeled shoes, this is often not the case (David-West, 2011). Family history of hallux valgus is a significantly greater predeterminant of hallux valgus than wearing high-heeled shoes (Wu & Louie, 2010). Poor biomechanics of the first MTP joint is the main contributing factor; poorly fitting footwear will only exacerbate the problem. Anything which interferes with the normal function of the first ray, the first MTP joint, and/or the subtalar joint can contribute to the development of HV. In particular, a hypermobile first ray and plantar flexed first ray can be contributing factors to the development of HV. The hypermobile first ray is unable to resist ground reactive forces and allows for increased subtalar pronation. If calcaneal eversion is present, the range of subtalar pronation is increased, which forces the first MT into a dorsiflexed and inverted position (Michaud, 1997). This would result in the progression of HV to a grade 3 due to the subluxation of the proximal phalanx. With a plantar flexed first ray, significant shearing forces contribute to the development of HV and bunion when the first MT head is forced upward during midstance of the gait cycle, which results in irritation to the bursa of the first MTP. Coughlin & Jones (2007) indicated in their demographic, etiological, and radiographical research that genetics, a long first metatarsal, a curved MTP joint articular surface, increased first ray mobility, and gapping of the first metatarsocuneiform joint were contributing factors for development of HV in the 122 feet they studied. Secondary etiological factors can be seen in patients with rheumatoid arthritis or any neuromuscular disease process.

The hallux and first MTP joint are important for the propulsive phase of gait and aid in stabilization of the forefoot. The muscles which act on the hallux and first MTP joint are the peroneus longus, abductor hallucis, adductor hallucis, flexor hallucis longus and brevis, and extensor hallucis longus. The peroneus longus muscle helps to stabilize the first ray through the propulsive phase of gait (Michaud, 1997). The flexor hallucis brevis muscle is a strong plantar flexor of the first MTP joint. The abductor and adductor hallucis muscles work together to stabilize the proximal phalanx of the hallux against the ground (Michaud, 1997). The first MTP joint is stabilized...
by the medial and lateral metatarsophalangeal ligaments, the medial and lateral metatarsosesamoid ligaments, the plantar plate which contains the sesamoid bones, and the deep transverse ligament which connects the plantar plates of the metatarsophalangeal joints (Steinbock, 2003). It has been found that in feet with hallux valgus, there is an imbalance between the adductor and abductor hallucis muscles and the flexor hallucis brevis muscle. Kilmartin & Wallace (1993) indicate that “there is a weak medial flexion force, a strong lateral flexion force, a weak adduction force, and no abduction force operating around the metatarsophalangeal joint.”

The severity of hallux valgus is determined via the hallux valgus angle (HVA) and the intermetatarsal angle (IMA). The HVA is the angle along the axis of the proximal phalanx and the first MT, while the IMA is the angle formed between the first and second metatarsals (Laughlin, 2009). There is a range of values for hallux valgus found within the literature. Most agree that 9–13 degrees is mild, 13–20 is moderate, and greater than 20 is severe (O’Donnell, Hogan, Solan, & Stephens, 2010).

Because the proximal phalanx normally acts as a stable origin for this muscle, if the hallux is unstable due to increased dorsiflexion and inversion of the first MTP joint, then a lateral force occurs (Michaud, 1997). During the second stage, the flexor hallucis longus tendon, extensor hallucis longus tendon, and adductor hallucis muscle shift laterally which abducts the hallux. This results in a medial widening and lateral compression of the first MTP joint. Additional bone is added at the first MT head medially and dorsally (Michaud, 1997). The third stage consists of the formation of primus metatarsus adductus with a cuneiform split. If the hallux continues to bear weight, the hallux will completely dislocate from the metatarsal head in the fourth stage of hallux valgus (Michaud, 1997). A hypermobile first ray can exacerbate the progression of hallux abductovalgus because the first ray is unable to resist ground reactive forces, which allows for increased subtalar pronation. This forces the first MT into a dorsiflexed and inverted position, which allows for the progression to stage three as the propulsive forces sublux the proximal phalanx (Michaud, 1997).

The Patient Perspective
Many patients who present with a bunion and hallux valgus may not experience pain symptoms unless wearing tight and/or inappropriate footwear. These patients may complain more of the look of the deformity than of pain. However, those that do have pain symptoms may describe tenderness and redness on the medial aspect of the bunion and sometimes edema at the bunion, and have a hard time fitting shoes. Patients with pain symptoms may also have limited range of motion of the first MTP joint and be diagnosed with hallux limitus.

As the hallux deviation progresses, symptoms occur along the dorsomedial aspect of the first MTP joint, plantar aspect of the first MT, pain with plantar flexion of the first MTP joint, and tenderness occurring in the first MT interspace and at the second MT head. Severe hallux valgus can contribute to lesser toe deformities.

Common Treatment
Custom foot orthoses which control the underlying poor biomechanics of the foot can reduce the pain symptoms of hallux valgus and help slow down the progression of the deformity in the early stages of hallux valgus. In cases of stage 3 hallux abductovalgus deformity, in which the intermetatarsal angle has progressed beyond 13 degrees, the effectiveness...
of functional foot orthoses decreases (Valmassy, 1995). Orthoses can also be used following surgical intervention to control biomechanical abnormalities. A common foot deformity, pes planovalgus, can be controlled with a medially posted orthosis. A metatarsal pad extrinsically placed under the top cover of the orthosis just proximal to the metatarsal heads can help reduce pressure off the forefoot to aid the propulsive phase of gait and thus reduce the abductory forces at the first MTP joint.

Properly fitting footwear is an important treatment component which cannot be overlooked. Narrow fitting shoes will increase pressure on the medial side of the foot, and therefore a shoe with a wide toe box can help alleviate the redness and edema at the bunion (Menz, Roddy, Marshall, Thomas, Rathod, Peat, & Croft, 2016). Other footwear features that can be beneficial include no seams over the bunions, a rocker sole, and a deep toe box. The material of the shoe over the bony prominence can be stretched by using a ball–and–ring stretcher to allow more room for the bunions. Many footwear companies are also using stretch fabrics in the toe box to help accommodate such deformities as bunions and hallux valgus. Many brands also provide sandals which can accommodate a custom foot orthosis and these sandals may have less material over the bunion area. In severe hallux valgus cases, increasing the forefoot rocker of the shoes while at the same time stiffening the sole can aid propulsion and reduce pressure on the first MTP joint and lesser toes that are also deformed. Removing a portion of the upper over the bunions and adding a balloon patch, preferably with a stretchy material or a soft leather, can also be beneficial.

**Surgical intervention**

Surgical intervention of hallux valgus can be performed, either for aesthetic reasons or due to a high level of pain. Although an in–depth description is beyond the scope of this paper, a brief description follows. There are a few types of interventions including bunionectomy, soft tissue repair, arthroplasty, arthrodesis, silicone elastomer implant, and osteotomy (Kilmartin & Wallace, 1992).

Complications with hallux valgus correction include metatarsalgia, non–union, recurrence of the valgus position of the hallux, and avascular necrosis of the first metatarsal (David–West, 2011). Another common complaint of patients is decreased range of motion of the first MTP joint following corrective surgery (Laughlin, 1995). This is a complication which affects podorthists and our treatment options following bunion correction because it can negatively affect the propulsive phase of gait for these patients. Unfortunately, the literature does not agree on the average expected limitation and changes to the first MTP joint following metatarsal osteotomies (Laughlin, 1995). A high recurrence rate of hallux valgus occurs due to factors such as untreated pes planovalgus, insufficient lateral displacement of the first metatarsal head, fracture of the metatarsal osteotomy, and insufficient soft tissue techniques (David–West, 2011). Another common reason for failure of hallux valgus corrective surgery is the failure of the surgeon to recognize the presence of hallux rigidus or osteoarthritis of the first MTP joint (Alexander, 1997).

**Key Considerations**

One struggle in treating a bunion and hallux valgus from a podorthic standpoint is changing the mindset of the patient to accept a change in footwear. It helps that more footwear brands today offer more stylish options that accommodate a custom foot orthosis; however, realistically, if a patient does not want to wear footwear with a wider fit, along with wearing the orthoses all the time, then the deformity will progress at a faster rate. Another struggle is recognizing that footwear and orthoses can only do so much and the deformity cannot be corrected unless an orthopaedic surgeon feels it necessary. If the patient continues to have concerns and issues about their bunion and hallux valgus, refer them back to their family or sports physician for a referral to an orthopaedic consultation.

**References**


Hallux Limitus/Rigidus

Maclean Graydon, BKin, C. Ped (C)

Key Messages

• Hallux limitus/rigidus is a continuum assessed by first MTP joint range of motion and pain.
• Hallux limitus should not be confused with functional hallux limitus.
• Orthoses are an effective treatment for hallux limitus by promoting first MTP joint dorsiflexion; and for hallux rigidus by restricting this motion with a rigid Morton’s extension.

Keywords

antalgic gait, joint immobility/restriction, osteophytes, overpronation, propulsion pain, rigid hallux extension, synovium, turf toe

Introduction

Hallux limitus/rigidus is a condition of the big toe joint where degeneration of articular cartilage and formation of osteophytes cause the first metatarsophalangeal (MTP) joint to become stiff and painful. This is a progressive disorder that has a gradual onset, even when caused by trauma. The condition exists on a continuum where increasing pain and decreasing range of motion dictate the severity. In spite of the simplicity of measuring hallux dorsiflexion, there is no clear delineation between hallux limitus and hallux rigidus. These are clinical terms that are vaguely differentiated based on the degree of joint immobility and arthritis (Banks, 2001). Several classification systems exist (the most up to date being Yee & Lau, 2008), however they are primarily a tool for orthopaedic surgeons and do not offer range of motion parameters for foot practitioners. Due to the lack of consensus on a true definition of hallux rigidus, clinicians rely mostly on experience rather than formal research (Beeson, 2008).

Hallux rigidus can be debilitating and next to hallux valgus is the second most common reason for a visit to an orthopaedic surgeon for forefoot pain (Beeson, 2008). In its early stages, the discomfort predominates at the dorsal aspect of the joint and becomes more diffuse with progression of the condition (Hunt, 2010). Due to pain and reduced mobility, patients with hallux rigidus often have secondary symptoms as a result of gait alterations, such as ankle and knee pain (DeFrino, 2002).

Pathology

Generally, the process of first MTP joint range of motion reduction begins long before symptoms exist. Joint restriction often begins with functional hallux limitus (Flavin, Halpin, O’Sullivan, FitzPatrick, Ivankovic, & Stephens, 2008). Patients with functional hallux limitus have reduced dorsiflexion of the first MTP joint in the propulsion phase (Aquino, 2001). Inadequate dorsiflexion causes increased pressure beneath the hallux (Zammit, 2008) and an increased rate of arthritis of the inter-phalangeal joint (Shurnas, 2009) as the tension from tight plantar soft tissue compresses the first MTP joint. Mechanical constriction of the first MTP joint from an impaired flexor hallucis longus tendon has been shown to load the joint (Kirane, 2008). Plantar soft tissue tension due to excessive pronation has a similar effect. Overpronation can lead to functional hallux limitus in individuals by over-loading the flexor hallucis longus muscle which increases tension in the tendon and restricts first MTP joint range of motion (Flavin et al., 2008). In a study measuring pronation, subjects with positive hindfoot valgus were 23% more likely to develop first MTP joint osteoarthritis than individuals with normal hindfoot alignment (Mahiquez, 2006). Patients with first MTP joint osteoarthritis have been shown to have higher medial forefoot pressures and more pronated foot postures in comparison to normal participants (Bryant, Tinley, & Singer, 1999). Nawoczenski, Ketz, & Baumhauer (1999) found a moderate relationship (Pearson correlation r = 0.61) between maximum passive first MTP joint dorsiflexion during standing and maximum first MTP joint dorsiflexion during
gait. These biomechanical deficiencies are the most likely causes of hallux rigidus.

Other etiologies have been suggested in the development of hallux rigidus from trauma to structural causes such as a long first metatarsal or metatarsus primus elevatus (Horton, 1999). A long first metatarsal relative to the second metatarsal was more common in a group with hallux limitus when compared to normal feet, and the correlation was greater when the hallux was also long (Munuera, 2007).

Hallux rigidus is associated with hallux valgus, familial history, trauma (often unilateral), and female gender. Metatarsus adductus is more common in patients with hallux rigidus likely due to its correlation with hallux valgus (Coughlin & Shurnas, 2003). These authors, as well as several others, (Flavin et al., 2008; Mann & Clanton, 1988) also suggest that a flat or chevron shaped distal first metatarsal will inhibit hallux glide as it dorsiflexes in the propulsion phase. This creates irregular joint wearing and limits range of motion.

In addition to range of motion limitations, there are also bone adaptations to consider. As hallux limitus progresses to hallux rigidus the joint often becomes visibly enlarged and is painful with dorsiflexion. As joint cartilage erodes the body responds by manufacturing osteophytes on the dorsal and medio-lateral sides of the distal first metatarsal and proximal hallux which further restricts range of motion and can become painful if they irritate the joint capsule and synovium.

The normal sagittal arc of movement of the hallux is 90 degrees of dorsiflexion to 30 degrees of plantarflexion (Shereff, 1990), however clinicians usually measure hallux range of motion in a non-weight-bearing position. This does not always provide a good indication of how the first MTP joint will perform during gait (see Common Testing, below). The average step requires 44 degrees (Nawoczenski, 1999) to 65 degrees (Hopson, 1995) of extension at the first MTP joint depending on factors like terrain and gait speed (this may even be greater if the ground is used as a reference point instead of the first metatarsal shaft). We can use this range as a clinical guideline, therefore, and suggest that anything less than 45 degrees should be considered hallux limitus, and when there is pain and/or visible exostoses of the distal first metatarsal or proximal hallux coupled with decreased range of motion, this should be considered hallux rigidus.

Patients with hallux rigidus have altered gait characteristics due to restricted range of motion and pain avoidance strategies. In normal gait the MTP joint dorsiflexes at heel-off allowing the ankle to roll over the foot in a larger arc, in turn generating greater torque. A functioning MTP joint allows the hallux to contact the floor for a longer period without generating excessively high peak pressures (Brodsky, 2007). With hallux rigidus the lever arm of the foot is effectively lengthened, and patients push off with a longer step length (Defrino, 2002). Without a compensatory decrease in ankle plantarflexion, increase in subtalar inversion, or toe-off angle, a vertical displacement in the body's centre of gravity occurs, manifesting as a lurch with each step taken. The patient’s gait may become increasingly antalgic as the first MTP joint stiffens and progressive metatarsalgia, lesser toe deformities, or malalignment of the foot may become evident (Fortin, 1995). As pain increases in the joint, patients may compensate by attempting to hold the foot in a supinated position or by changing the foot progression angle to avoid dorsiflexing the first MTP joint in the sagittal plane (Hunt, 2009). This manifests as increased wear on the lateral surface of the sole of shoes as well as an oblique crease in the toe-box (Sheriff & Baumhauer, 1998). In advanced hallux rigidus, prolonged activity while barefoot or in shoes with a soft or flexible forefoot is painful and wearing high-heeled shoes is almost impossible.

Differential Diagnosis

Hallux limitus is a precursor to hallux rigidus and should not be confused with functional hallux limitus which is a functional restriction of hallux dorsiflexion. In the latter condition, the first MTP joint is impinged due to plantar soft tissue tension either due to intrinsic tautness, or to overpronation taking up slack in the plantar fascia (Danazanberg,1993). Feet with functional hallux limitus dorsiflex adequately at the first MTP joint in the sagittal plane (Hunt, 2009). Although functional hallux limitus is a contributing factor in hallux rigidus, feet with functional hallux limitus may not necessarily be experiencing any joint erosion or osteophyte formation.

Turf toe is an injury which damages the first MTP joint and surrounding soft tissues. Although the symptoms can be similar to hallux limitus, it is considered an acute injury, not a degenerative condition of the joint.
Common Testing
In a clinical setting the most basic way to test first MTP joint range of motion is to grasp the foot with one hand on the first metatarsal and dorsiflex the hallux with the thumb and index finger of the other hand, being careful to push on the proximal segment of the hallux (so that dorsiflexion is not exaggerated by bending the hallux at the inter-phalangeal joint). The joint angle can be measured with a goniometer by placing one end along the medial side of the first metatarsal shaft and the other along the hallux with the fulcrum at the joint line. This method allows the clinician to assess joint-line pain as well as the presence of osteophytes.

There is some dispute that passive non-weight-bearing joint range of motion is an accurate depiction of range of motion during gait. Nawoczenski (1999) found that there was only a moderate correlation (Pearson correlation r = 0.67) between maximum passive non-weight-bearing range of motion and hallux dorsiflexion during gait; while there was a 0.80 correlation for active weight-bearing motion and 0.87 for a heel–rise test. The heel–rise test (Nawoczenski, 1999) is easy to perform in a clinical setting and more closely resembles the amount of first MTP joint dorsiflexion that would be found using gait analysis equipment in a laboratory setting as it takes into account the soft tissue structures of the plantar foot.

Using a goniometer placed along the side of the forefoot with the fulcrum at the first MTP joint the patient lifts the ipsilateral heel off the ground as high as possible while keeping the hallux flat on the ground. One caveat to using this method is that a foot with functional hallux limitus may appear to have hallux rigidus due to soft tissue restriction of first MTP joint dorsiflexion. The heel–rise test seems to mitigate this confusion, however, as once the heel is off the ground and the windlass mechanism is in effect, the joint should only be blocked by bony formations, not the soft tissue. The patient may need to hold on to something for balance while they perform this test.

Contraindications
Whenever possible it should be the clinician’s goal to improve first MTP joint mobility. The windlass effect is not possible without sufficient hallux dorsiflexion (Dananberg, 1993). Rigid rocker soles and orthoses with rigid hallux extensions are valuable tools for reducing pain in the first MTP joint by impeding dorsiflexion of the hallux, but they also serve to encourage hallux rigidus by reducing synovial fluid production in the joint as it is less mobile and reducing stretching of the plantar soft tissue structures. As the plantar fascia, flexor hallucis longus and brevis become tighter, they place more pressure on the joint which causes further breakdown. Clinicians should be wary of employing joint immobilisation techniques before it is necessary.

The Patient Perspective
Patients rarely notice a reduction in first MTP joint range of motion until the joint is painful or osteophytes begin to form. It is usually through palpation of the joint and moving the big toe up and down that they notice it does not move as far in dorsiflexion as it does in plantarflexion. The patient will usually describe pain during the propulsion phase of gait, especially as the heel comes off the ground and body weight is supported by the metatarsals and toes. The windlass mechanism compresses the joint, and if there is cartilage breakdown or an irregularly shaped distal first metatarsal bone, pain can be elicited as the hallux glides upwards.

Common Treatment
NSAIDs and cortisone injections can offer temporary relief to joints if they are inflamed (Shereff & Baumhauer, 1998; Solan, 2001); however hallux rigidus is a chronic condition and is only reversible with surgery. For long-term pain relief and improved mobility clinicians often choose footwear modification or orthotic inserts (Smith, Katchis, & Ayson, 2004). There is evidence that orthoses can alleviate symptoms (Torkki, Malmivaara, Seitsalo, Hoikka, Laippala, & Paavolainen, 2001; Torkki, Malmivaara, Seitsalo, Hoikka, Laippala, & Paavolainen, 2003), and delay deterioration of the first MTP joint (Munuera, 2006; Sherer, 2006). When reviewing the literature it is important to note the intent of the orthotic intervention (Landorf & Keenan, 2000). Some trials attempt to increase dorsiflexion through the first MTP joint in order to improve long-term joint function in patients with hallux limitus (Thompson, 1992). Other studies attempt to limit first MTP joint range of motion with a rigid hallux extension in order to alleviate symptoms due to joint irritation from contact of the bones and osteophytes (Shereff & Baumhauer, 1998). In the first category, Munera (2006) found that when functional hallux limitus was caused by abnormal subtalar pronation, hallux dorsiflexion was gradually restored by the use of foot orthoses. By correcting subtalar eversion and medial arch depression, the windlass mechanism
can be restored, thereby improving first MTP joint range of motion during push-off (Van Gheluwe & Dananberg, 2004). The research is not conclusive, however, as several other studies have found high variability or nonsignificant results (Halstead, Turner, & Redmond, 2006; Hogan and Kidd, 2001; Nawoczenski & Ludewig, 2004) when attempting to improve joint range of motion with orthoses. Overall, there is a trend towards improving hallux dorsiflexion during push-off with the use of orthoses that correct or limit pronation. Hallux dorsiflexion can be further encouraged by adding a first ray cutout (also known as a reverse Morton’s extension, kinetic wedge, or 2-5 bar) to the distal shell of the orthosis. In contrast, with a rigid joint that would not respond to increased dorsiflexion, or where pain management is the goal, orthoses with a rigid hallux extension can be effective at reducing first MTP joint range of motion and alleviating pain (Thompson, 1992). Whereas the shell of an orthosis usually ends just proximal to the metatarsal heads, with a rigid hallux extension the shell is cut so that it extends under the hallux. This reduces the joint’s ability to dorsiflex in the propulsion phase of gait and therefore there is less compression of osteophytes on the dorsal surfaces of the first metatarsal and hallux.

Footwear modifications such as a rigid rocker sole can also be effective at reducing the symptoms of hallux rigidus. Adding a stiff shank and rockered forefoot to the shoe will decrease joint motion and encourage forward propulsion during gait. It should be noted that the intention of this intervention is to decrease hallux dorsiflexion in the propulsion phase which should only be done once the patient is asymptomatic. Until that point, all efforts should be made to increase first MTP joint range of motion.

Footwear with a deep toe-box and stiff rocker sole reduces bending at the MTP joints, and allows the patient to continue to ambulate without having to make gait alterations to avoid pain (Shereff & Baumhauer, 1998).

Key Considerations
As with most cases of joint breakdown (osteoarthritis), age is a factor in the development of hallux rigidus. With increasing age the first MTP joint is exposed to more wear-and-tear, and there is a greater likelihood of it being subjected to trauma. Although an active lifestyle is important in maintaining healthy joints, overuse due to occupation or sports can accelerate joint breakdown. Occupations that require working on uneven terrain like construction can expose the first MTP joint to forces from various angles, and sports such as soccer and football where the joint may be hyper-extended or compressed may damage cartilage or the joint capsule.

References


Sesamoiditis

Darlene Rempel, BHK, C. Ped Tech (C), C. Ped (C)

Key Messages

- A condition of the first metatarsal area that is often a diagnosis of exclusion because it so closely resembles so many other forefoot pathologies.
- Primarily a condition of repetitive overuse with insidious onset.
- Impact activities (such as running and dancing), plantarflexed first ray, improper footwear and overpronation may predispose sesamoiditis.
- Treatment can be as simple as offloading the affected area.

Keywords

bony feet, distal glide, first metatarsophalangeal/MTP, first ray, hallux, inflammation, ossification, osteoarthritis, plantar point tenderness, sesamoids, tibial (medial)/fibular (lateral), toe-off

Introduction

The foot encounters substantial forces during walking that increase with the pace. Peak forces can reach 120% of body weight during walking and are up to 220% during running. At toe off in the gait cycle, the centre of pressure occurs over the hallux (Hunt, McCormick, & Andersen, 2010). With such great load landing over the first metatarsophalangeal (MTP) joint, the occurrence of injury can be frequent.

The word “sesamoid” comes from the Greek word “sesamoëides,” which means “sesame seed-like.” With two pea-shaped bones or sesamoids imbedded in the tendon under the first MTP, injury to these bones are often labeled as sesamoiditis. Specifically, sesamoiditis is an inflammatory condition of the periosteum or bone lining of the sesamoid bone.

First MTP joint pain can originate from a variety of etiologies such as overuse syndromes, minor traumas, degeneration, and fractures. Therefore sesamoiditis is often a diagnosis of exclusion as many first MTP joint symptoms can originate from other conditions.

Pathology

The first MTP joint is a hinge joint, characterized by a convex cartilage-covered surface of the first metatarsal bone that interacts with the concave surface of the proximal phalanx. The abductor and adductor hallucis muscles, combined with the collateral ligament from the metatarsal heads, provide dynamic support to the medial and lateral joint (Coughlin, 2007). The medial and lateral sesamoids are imbedded in the flexor hallucis brevis and help to dissipate forces at the MTP joint by elevating the metatarsal head (Hunt et al., 2010). Ossification of the sesamoids is seen in females at age eight and in males at age ten, with the lateral forming before the medial (Dharap, Al-Hashimi, Kassab, & Abu-Hijleh, 2007).

The medial (tibial) sesamoid is larger and longer and therefore tends to sit more distally. It is imbedded into the medial head of flexor hallucis brevis and receives attachment from abductor hallucis and from here extends via a thick strong tendinous band to the plantar medial aspect of the base of the proximal phalanx. The medial portion of the plantar aponeurosis also blends into the plantar non-articular surface of the sesamoid (Hunt et al., 2010). The fibular (lateral) sesamoid is imbedded into the lateral head of the flexor hallucis brevis and receives attachment from the lateral head of the flexor hallucis brevis and the adductor hallucis via the intermetatarsal ligament. From here, the band extends to the plantar lateral aspect of the base of the proximal phalanx where it also is blended with the plantar aponeurosis and by the deep portions of the intermetatarsal ligament. (Hunt et al., 2010).

Primarily, the sesamoids of the hallux reduce friction at the first MTP joint and provide a biomechanical advantage to the function of the flexor hallucis brevis in plantarflexion activities using fulcrum mechanics (Richardson, 1999). The movement and tracking
of the sesamoids has been likened to that of the patella. As the first MTP joint flexes, the sesamoids glide distally (Sims & Kurup, 2014). A complex set of tissues maintains their functional and dynamic integrity, including tendons, ligaments, and fascial tissue. Mediolateral stability comes from the collateral ligaments of the metatarsal heads, and is extremely important in cutting activities in sports, such as football and basketball (Hunt et al., 2010). Sesamoid function is especially fundamental in sports or activities involving jumping, sprinting, spring board diving, ballet, and certain martial arts.

During the normal gait cycle, the forefoot experiences most of its stresses during the later gait phase, while the foot is rigid. As gait progresses to heel raise and toe off, the hallux and lesser toes reach their maximum dorsiflexion (Hunt et al., 2010). It is in the later gait phase that the sesamoids are under the most stress, with the medial sesamoid bearing more weight than the lateral (Boike, Schnirring-Judge, & McMillin, 2011).

An injury to the sesamoids is usually an insidious onset of plantar pressures to the first MTP joint. Footwear choices may inflict undue pressures to the forefoot such as high-heeled shoes or cleats. A job that requires frequent squatting or carrying heavy loads can trigger sesamoiditis over time.

Although rare, adding to the challenges of other pathologies masquerading as sesamoiditis are congenital variations. Variations such as an enlarged, bipartite (divided sesamoid bones), or complete absence of sesamoids can occur, causing similar symptoms at the first MTP joint. Hereditary defects such as high arches, or a planterflexed first ray will put excessive pressures at the metatarsal heads which may lead to pain involving the sesamoids. Furthermore, a pronated foot will advance gait through the medial column of the foot producing a medial toe-off in gait. This excessive pressure at the first MTP joint may produce inflammation at the sesamoids.

Osteoarthritis of the sesamoid bones is common in older populations and may involve the formation of bone spurs. These osteophytes can cause inflammation to the surrounding muscles and tendons (Lee, 2008). Osteoporosis is the loss of calcium and weakening of the bones and may result in a stress fracture.

Pain associated with sesamoiditis may be present with tenderness, redness, swelling and point tenderness on the plantar side of the first MTP joint, (particularly at the sesamoids) in the toe off phase of gait. Tenderness of the surrounding tissues may also be present and intensify with pressure. Usually, when pressure is removed, the pain dissipates quickly as well. However, in some chronic situations, the pain may continue even in non-weight-bearing conditions.

**Screening and diagnosis**

Upon assessing a patient, it is important to take a comprehensive medical history which should include the identification of the mechanism of pain, congenital defects, length of symptoms, which activity and stage of gait produces the symptoms, and any other relevant history. Careful examination of the patient’s shoes should be performed. The outsole will indicate high wear patterns under the first MTP joint, or in cases of avoidance gait due to symptoms, a lack of wear is seen. With avoidance gait patterns for first MTP pain, an oblique crease in the shoe’s vamp may be seen (Oh Park, 2001).

Epidermal surfaces of the plantar forefoot may demonstrate hyperkeratosis (callous) at the first MTP joint, or an intractable plantar keratosis (IPK) or corn when there is excessive pressure (Richardson, 1999). Range of motion should be examined where normal range of active extension of the first MTP joint is 80 degrees and another 25 degrees of passive range (Coughlin, 2007).

Etiological factors which may predispose sesamoiditis include: plantarflexed first metatarsal, hypermobile subtalar joint, cartilage abnormalities, enlarged or multiple sesamoids, acute or repetitive trauma, and inappropriate shoes (Michaud, 2011; Sims & Kurup, 2014). Although a sudden impact trauma may cause sesamoiditis, it is more common to have a gradual onset of pain. Insidious onset sets it apart from most other forefoot conditions. A sudden trauma to the first MTP factored with increased edema and redness with acute point tenderness may indicate a fracture. The patient should be advised to check in with the physician who may order an X-ray, or bone scan (Decker & Albert, 2002).

In all stages of pain, point tenderness at the sesamoids with forced dorsiflexion is indicative of sesamoiditis. However, in the early stages, the patient may experience mild pain with walking barefoot or thin-soled shoes. Pain worsens with an increase in activity intensity, but often subsides when activity finishes. Some mild swelling may also be present (Dedmond, Cory, & McBryde, 2006).

In more chronic cases, the pain under the first MTP joint may be constant and intensifies with activity.
Applying pressure with dorsiflexion of the first MTP joint is painful. Swelling may be apparent, along with redness and warmth at this joint and potentially the entire first toe (Cohen, 2009).

**Differential Diagnosis**

The symptoms of sesamoiditis are often misdiagnosed as other pathologies and vice versa. Sesamoiditis is also a very general term meaning the inflammation of the sesamoids which sounds more like a description than a diagnosis.

Acute trauma like hyperextension under force or a stress fracture from repeated trauma over time may also be diagnosed initially as sesamoiditis. However, if symptoms persist over time despite using conservative treatment, further investigation is warranted. It may not present itself in an X-ray and may require a bone scan to determine a stress fracture. Turf toe is another common injury of the first MTP joint which is caused by the hyperextension of the joint by flexible shoes on “sticky” artificial turf (Coughlin, 2007). In this case a possible lesion to the plantar plate may be found causing instability to the joint. 7–33% of the population present with sesamoids which have failed to ossify in adolescence, causing bipartite, tripartite or quadripartite sesamoids. When examined in radiographs, sesamoids may be diagnosed as fractured, only to realize that proper ossification did not occur. If symptoms of lateral plantar pain under the lateral sesamoid are accompanied by partial loss of sensation under the medial border of the hallux, a parasesamoid lesion (of the medial plantar digital nerve) may exist. Further investigation may be warranted (Boike et al., 2011).

Pain in the MTP joint can result from flexor hallucis brevis tendinitis, osteochondritis dissecans, avascular necrosis of sesamoids, inflamed bursae, intractable keratoses, infection, sesamoiditis, gout arthropathy, tumours, and rheumatoid arthritis (Richardson, 1999). This is why taking a detailed history of the patient’s medical history is important.

**Common Testing**

Range of motion testing will be important to determine the extent of the injury at the first MTP joint. 80 degrees of active range of motion with an additional 25 degrees of passive motion is average at the first MTP joint although this number declines with age (Coughlin, 2007). A reduction of range or pain at the joint may indicate sesamoiditis.

Pain on palpation would be present on the plantar side of the first MTP joint with point tenderness at the sesamoids (both or one only). In passive range of motion, the sesamoids migrate distally toward the first phalange. Typically, pain will increase as the joint extends. Furthermore, at toe-off the centre of pressure is located at the hallux and would therefore recreate the typical symptoms of sesamoiditis (Hunt et al., 2010).

**Contraindications**

Applying direct pressure to the first MTP joint, such as firm padding primarily under the joint or walking on hard surfaces would be contraindicated. Also, cortisone injection to the joint could compromise the integrity of the ligaments and soft tissue supporting the joint. (Porter & Schon, 2008).

**The Patient Perspective**

Patients may use general terminology when describing their pain at the first MTP joint. It is important to determine where the pain is precisely, dorsal or plantar. Patients would describe dull, aching pain with walking, particularly at toe-off or stair climbing. Depending on the severity of the problem, there may be constant pain, even when at rest.

**Common Treatment**

When considering treatment, the severity of the problem should be considered. In mild cases footwear changes may produce dramatic results while in severe cases further investigation by a physician will be required.

Rest and ice are recommended for any inflammatory condition. Use of topical analgesics or oral anti-inflammatories may be helpful to dissipate pain. Avoidance of repetitive activities which irritate the first MTP is imperative. Cross training may be required during the “healing” period. Physical therapy is often recommended to help restore mobility through simple range of motion stretches. Also exercises such as toe crunches and heel raises are suggested after pain has subsided.

**Orthoses techniques**

When considering orthotic techniques, over-the-counter orthoses may be helpful in initially offloading the first MTP joint by reducing pronation. An addition of a specifically placed metatarsal dome or bar just proximal to the metatarsal heads will further offload the affected structures (Porter & Schon, 2008).
In cases of moderate to severe pain, a custom-made product will be more likely to address sesamoiditis symptoms. An intrinsic and/or extrinsic varus posting of the rearfoot with medial arch posting will control excessive medial forces from overpronation during the gait cycle (Decker & Albert, 2002; Michaud, 2011). Limiting cast fill and/or an addition of a medial skive will allow the CFO to fit closely to the arch and effectively reduce pressure to the medial metatarsal heads. Higher medial arch posting will transfer forces from the metatarsal head to the proximal shaft of the bone and to the lateral forefoot (Porter & Schon, 2008). Furthermore, adding forefoot padding such as Poron may protect the affected area from hard surfaces.

If extension of the first MTP joint causes the most severe symptoms, consider reducing motion at the joint by designing the CFO with a rigid Morton’s extension (Coughlin, 2007). If plantar pressure reproduces sesamoid symptoms, place a metatarsal bar just proximal to the metatarsal heads to offload forces at the forefoot during toe-off (Boike et al., 2011). A valgus forefoot posting may also create a transfer in pressure to the other metatarsal heads. In more severe cases, metatarsal padding may be fashioned to create an excavation (“J” or “donut” pad) under the affected joint, allowing the first MTP joint a decrease in load (Baumgartener, Moller, & Stinus, 2016).

**Footwear and modifications**

Early, mild cases of sesamoiditis may do very well with a change in footwear. A flat, wide stiff-soled shoe with forefoot rocker will reduce motion and pressure at the joint (Porter & Schon, 2008). A shoe with a good shock absorbing midsole may provide enough cushioning to reduce plantar pressures.

A footwear excavation inside the shoe at the location of the painful area will decrease ground reaction forces to the joint. If motion of the joint is particularly painful, a rigid shank with a rocker sole modification with the apex just proximal to the first MTP joint will prevent extension and decrease plantar pressures (Porter & Schon, 2008).

**Further investigation**

In extreme cases of pain over a long duration of time where conservative treatment has failed, a surgical consult may be warranted.

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**Key Considerations**

- Sesamoid pain may occur in an otherwise normal foot placed under unusual impact stresses
- Radiographic findings and duration of symptoms prior to treatment with orthoses did not correlate with the success of treatment. The success of treatment was not dependent upon the distinction of whether the sesamoid was fractured or was a congenital variation with subsequent inflammation
- Orthoses can provide relief of symptoms in the treatment of patients with pain beneath the first metatarsal head, providing the activity does not involve toe walking
- Athletes treated with orthoses for sesamoid pain were able to continue training or participating with minimal, if any, loss of time. Casting and/or surgery would require 4 or more weeks of time lost from participation

**References**


Morton’s (Foot) Syndrome

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Key Messages

• Morton’s syndrome is a short first metatarsal, not a long second toe.
• The protrusion of the metatarsals is more important than the actual bone length.
• The first metatarsal is measured relative to the second metatarsal.
• A short first metatarsal puts increased pressure on the second metatarsal and can lead to foot pain including metatarsalgia and midfoot arthritis; the second metatarsal head is at greater risk of developing an ulcer in diabetic patients.
• Orthoses with a soft rubber extension under the first metatarsal head could be used to treat Morton’s syndrome by increasing the load the first carries in relation to the lesser metatarsals. A metatarsal mound can also be used to offload the second metatarsal head.

Keywords

arthrosis, chevron, distal protrusion, inter-metatarsal angle, lever arm, metatarsus adductus, windlass mechanism

Introduction

Morton’s syndrome was introduced by Dudley Morton in a paper published in the Journal of Bone and Joint Surgery identifying a foot type which he named metatarsus atavicus (Morton, 1927). This foot type was characterized as having a shortened first metatarsal. Morton asserted that in a normal foot, the first and second metatarsal heads should be positioned evenly in the sagittal plane (neither more distal nor proximal to the other), but that in metatarsus atavicus the first would be positioned noticeably behind the second. He also proposed that due to its distal protrusion, the second metatarsal would bear more weight, causing hypertrophy of the bone and pain or callusing under the head of the second metatarsal. This is now called Morton’s syndrome. Morton’s syndrome has been blamed for predisposing feet to stress fractures of the second metatarsal (Morton, 1927), metatarsalgia (Hurst, 2007), diabetic ulcers of the second metatarsal (Katoulis, Boulton, & Raptis, 1996), hallux valgus (Klaue, Hansen, & Masquelet, 1994), midfoot arthrosis (Davitt, Kadel, Sangeorzan, Hansen, Holt, & Donaldson-Fletcher, 2005), and overpronation (Simons, Travell, & Simons, 1999). Various publications have estimated that Morton’s foot occurs in 4–30% of the population (Grebing & Coughlin, 2004) but there has not been an epidemiological study to confirm this number.

Dudley Morton did not specifically define normal and abnormal protrusion of the metatarsal heads. Moreover, his method of measuring feet, in spite of the accuracy of X-rays, was disputed by his contemporaries (Hardy & Clapham, 1951; Harris & Beath, 1949). The lack of a clear delineation makes it difficult for clinicians to draw a line between a normal foot and a disordered foot, as someone might appear to have a Morton’s foot due to a short first metatarsal, a long second metatarsal, or they might simply have a large inter-metatarsal angle (Harris & Beath, 1949; Coughlin & Jones, 2007; Munuera, Polo, & Rebollo 2008). There is also a problem of definition: are we describing metatarsal length, or metatarsal protrusion? The base of the second is located proximally to the first, so even in a foot with metatarsal heads in perfect alignment in the sagittal plane, the absolute length of the second metatarsal bone will be greater than the first in the majority of feet even if it does not protrude more distally (Dogan, 2007). Scientists are most concerned about a second metatarsal that protrudes beyond the first regardless of actual metatarsal length (Rodgers & Cavanagh, 1989). Dogan (2007) measured the length of metatarsals of 100 subjects and showed that,
on average, the second metatarsal was the longest metatarsal bone, but they did not measure protrusion, only absolute bone length. For clinical purposes this chapter will define Morton’s foot as having a first metatarsal head located even with or behind the third metatarsal head. Using the third metatarsal allows clinicians to use an anatomical reference without encountering the errors that previous researchers have found using the second metatarsal head as a landmark. It also shows that the first is short enough to be clinically significant.

Pathology

The first metatarsal forms the distal segment of the medial arch and acts as a lever during propulsion (Kirby, 1987). When this bone is excessively short, the effect of the lever arm is reduced and less torque is generated at push-off. A significant amount of leverage is provided by the first ray and its musculo-tendinous structures from late-stance through toe-off (Dicharry, 2010). The first metatarsal head, along with the sesamoids, creates a larger lever arm than the lesser metatarsals and the windlass mechanism (Hicks, 1954; Cheng, 2008) is rendered more effective. The thickest part of the plantar fascia attaches into the first metatarsal and hallux to give support to the first ray. In Morton’s foot, as tension is taken up by the plantar fascia during metatarsophalangeal (MTP) joint dorsiflexion, greater tension is put in the weaker area of the fascia where it attaches into the second and third metatarsals (Christensen & Jennings, 2009). If the windlass mechanism is less effective, the foot is allowed to continue to pronate through the late stages of propulsion, and the push-off comes from the medial side of the hallux (Kirby, 1987), rather than the plantar/distal tip. This proximal location of the first MTP joint also means that it dorsiflexes earlier in the gait cycle, and the second MTP and those of the lesser toes dorsiflex later, which causes pronation of the forefoot, proportionally placing more weight on the lesser metatarsals while rolling towards the medial hallux (Weijers, 2004). The effect is similar to a chair with one short leg; the chair will balance on the three longer legs, and tip towards the short one. During propulsion in a Morton’s foot there is a shear force across the metatarsals, as well as a greater medial force to the hallux. This excessive pressure to the medial hallux can contribute to hallux valgus, and the greater work performed by lesser toes as the fascia attaches into them can contribute to hammer and claw toes as they grip.

In Morton’s foot, the first metatarsal head is located even with or behind the third metatarsal head which gives the forefoot the appearance of a chevron. The foot bends and pivots with the second metatarsal as its fulcrum. Morton (1927) described this as the “axis of balance”. As the second metatarsal protrudes furthest distally, it bears a greater proportion of the weight across the forefoot, when standing or ambulating. Bojesen-Moller (1979) proposed a two-gear system where humans use either an in-toeing or out-toeing strategy depending on several gait factors, including speed and power. There would be two axes across the MTP joints: the transverse axis drawn through the first and second MTP joints, and an oblique axis drawn through MTP joints two through five. If Bojesen-Moller’s theory is correct, then with a short first metatarsal the transverse axis would not be perpendicular to the direction of travel, rather it would be angled more posterior through the more proximal first MTP joint causing a second oblique axis. This would give the axes the appearance of an arrowhead with the second MTP joint being the tip. In either of Bojesen-Moller’s scenarios, the second metatarsal is the pivot point, and therefore will bear more pressure and friction when weight is on the forefoot. Rodgers and Cavanagh (1989) concluded that while the second metatarsal head experiences greater pressure than the first in the majority of feet, in Morton’s feet it produces a significantly higher magnitude of peak pressure when compared with normal feet (320 kPa versus 243 kPa) making them more prone to callusing and even stress fractures.

A handful of studies have shown that Morton’s feet are predisposed to more serious wear and tear than simple callus build-up. Weber, Aubin, Ledoux, & Sangeorzan (2012) used cadaver feet to show that an elongated second metatarsal increased pressure under the second metatarsal head, and resulted in a more medially deviated second toe (crossover second toe). In a study by Chuckpaiwong, Cook, Pietrobon, & Nunley (2007), the average length of the first metatarsal compared with the second metatarsal, in patients with proximal stress fractures of the second metatarsal was 80%. In a relative metatarsal length study by Davitt et al. (2005), it was shown that a short first metatarsal changes weight distribution across the forefoot, which alters foot mechanics, causing the second metatarsal to bear a greater proportion of weight. This study shows that there may be a mechanical aetiology for midfoot arthrosis. The authors found that subjects in the study group who had midfoot arthrosis had on average an 18.6% longer second metatarsal when compared to the first metatarsal, while subjects in the control group had only a 4.1% greater second versus first metatarsal length. These studies as well as Rodgers and
Cavanagh (1989) show that where the first metatarsal shaft is significantly short relative to the second there is in fact increased stress on the second metatarsal and therefore on the second tarso-metatarso and metatarsophalangeal joints.

The combination of increased pressure due to the distal position of the first metatarsal, and friction due to forefoot pronation in the propulsion phase make Morton’s feet prone to ulceration in people with diabetes mellitus. Morton’s theories of callus build-up are confirmed by Ledoux (2005) and Bolton & Baine (2012) who showed that foot deformities can make someone with diabetes more prone to developing ulcers on the plantar foot. If a long second toe co-exists with Morton’s syndrome, a patient who has diabetes is also prone to ulceration of the tip at the second toe due to hammer/claw/mallet toe.

**Differential Diagnosis**

There is a common misconception that Morton’s feet can be identified by a long second toe. Using the toes as a reference can be misleading, as we are most concerned with the length of the metatarsals. For instance, a foot might have an abnormally short hallux or long second toe, but metatarsals of equal length, which is not considered a Morton’s foot. For this reason, it is important to take a careful measurement of metatarsal length with specific attention to metatarsal protrusion.

One aspect of Morton’s syndrome is that the second metatarsal will have increased callosity due to greater pressure on the pad. As Harris & Beath (1949) showed, not all feet with a callus on the second metatarsal head should be considered Morton’s feet. However, a fallen second metatarsal head, or collapsed metatarsal arch may place undue stress on the second metatarsal head and cause the skin to thicken here. Second metatarsal head calluses are a symptom of Morton’s syndrome, but not proof that a patient suffers from this condition.

There are other foot abnormalities that may disguise or exaggerate the appearance of Morton’s foot. Metatarsus primus varus and metatarsus adductus make it very difficult to accurately measure metatarsal length, even with the benefit of X-rays. Harris and Beath (1949) claimed that Morton overestimated the rate of patients with a short first metatarsal in cases of metatarsus adductus as the larger inter-metatarsal angle makes the first appear abnormally short. Hardy & Clapham (1951) developed a different measuring technique from that of Morton to reduce false positives when measuring metatarsal length.

Given these examples of common errors, one should be wary of over-diagnosing Morton’s foot, as even with the accuracy of X-rays, there are arguments concerning how many feet should truly be considered Morton’s. However, using the third metatarsal as a marker for comparison rather than the second in a clinical examination reduces this potential error somewhat.

**Common Testing**

In a clinical setting without the benefit of X-ray technology, there are a few simple ways to assess the foot for Morton’s syndrome. With the patient sitting in front of the clinician, the foot is held with one hand, and the toes are plantarflexed to expose the MTP joints. Using an indelible pencil or a ball point pen, the metatarsal is bisected and a mark is placed in the MTP joint space of the first and third. With the patient standing, the clinician examines the dorsum of the foot to determine if the first is located distally or proximally to the third MTP joint. This may be done with eyesight, or by using a ruler or tape-measure to measure from the talo-crural joint to the MTP joint space.

Another simple clinical method is to use a Harris mat, or ink pad. The patient stands on the pad and an image of the foot is produced by the weight-bearing surfaces of the foot causing the ink to transfer onto a piece of paper. Depending on foot architecture, it should be easy to identify the heel, lateral border of the foot (fifth metatarsal), metatarsal heads, and toes.
On the image the clinician bisects the heel and places a mark at the most posterior point of the outline of the foot. The same is repeated with the first, second, and third metatarsal heads at the centre point (this is usually the darkest part of the ink-blot). Three lines are then drawn using a ruler to connect the dots between the heel and the metatarsal heads. The middle line to the second metatarsal head is the reference line. A line is drawn from the reference line to the dots on the metatarsal heads at a right angle to the line. The clinician then determines which dot is most distally located.

A slightly more complicated, but arguably more accurate and reproducible method can be performed using digital photography. This is useful because it can be stored with the patient’s records, either in digital or print form. A 1”x 5” piece of Subortholen bent at 45 degrees with a hole drilled at the bend is placed on the dorsum of the MTP joint while the toe is passively dorsiflexed, and a pen mark is placed on the foot through the hole. This should mark the distal tip of the metatarsal shaft. These marks are placed on the dorsum of the foot at the first and third MTP joint lines as well as on the navicular tubercle which is easy to palpate on most feet.

Using a tripod, a photograph is taken from a height of approximately 40cm pointing straight down at the foot. Using GIMP software (free, open-source software easily downloaded at http://www.gimp.org), a reference line is drawn from the navicular to the first and third MTP joint marks. The software has a tool that can measure the absolute distance from the navicular tubercle to the marked spot on the first and third metatarsals. A ratio can then be calculated showing relative metatarsal protrusion.

A final method is to use software programs that rely on pressure to show where metatarsal heads are located and to describe the stress they bear. These programs have tools which can help with quantifying which metatarsal (first or third) is located most distally on the foot. Pressure mapping is the most accurate way to measure weight distribution across the forefoot (Guldemond, 2006) but simply having increased pressure on the second metatarsal head is not a guarantee that someone has Morton’s syndrome. This software is expensive and not accessible to all clinics.

**Contraindications**

A rigid plastic extension from the medial/distal part of an orthotic shell that protrudes under the hallux is often called a Morton’s extension. Clinicians can be confused by the name. This is designed to reduce dorsiflexion of the first MTP joint when treating symptomatic hallux rigidus, and should not be used with Morton’s syndrome. A Morton’s extension for Morton’s syndrome should be made from a flexible rubber material.

Rigid or semi-rigid metatarsal pads with a peak or summit line are designed to treat Morton’s neuroma. For Morton’s syndrome the metatarsal pad should be constructed from a soft or semi-rigid material and its apex should be under the second metatarsal shaft—6–11mm behind the metatarsal heads—to reduce pressure on the second metatarsal head (Hastings, 2007).

**The Patient Perspective**

There are no direct symptoms in Morton’s feet as this is a congenital condition, so patients usually complain of secondary symptoms. Most patients are not aware that they have Morton’s foot unless they have a noticeably short hallux or long second toe (which does not guarantee a Morton’s syndrome anyways). Most often if they have secondary symptoms, patients
complain about pain or burning of the second metatarsal due to excess pressure or friction. There may be visible callusing of the second metatarsal head and this area may be painful with palpation.

Some patients may describe a history of pain or stress fractures of the second metatarsal, pain in the midtarsal (Lisfranc joint), or hallux valgus. They may also complain of a hammer or claw toe of the second toe as a result of tightening of the flexor digitorum muscles from overuse. An inspection of the structures of the foot, as well as the biomechanics, will determine if Morton’s syndrome exists and if it is contributing to the patient’s symptoms.

## Common Treatment

Clinicians often use a Morton’s extension to treat Morton’s syndrome. This is a flexible pad that extends under the first metatarsal head and sometimes under the hallux to the inter-phalangeal joint. This pad is designed to raise the first metatarsal head approximately 2–6mm. Its purpose is to effectively lengthen the lever arm of the first ray. By raising the first metatarsal up, it contacts the ground earlier and therefore longer into the propulsion phase which gives the medial arch a longer torque curve. This type of Morton’s extension pad may be purchased commercially from the Internet; attached to inserts; or through a custom device in a foot clinic (Decherchi, 2005). It is usually made from a flexible rubber material like EVA and attached to the distal part of the shell of an orthotic device, similar to a forefoot post. This extension braces the forepart of the orthoses in the latter stage of mid-stance and during the early part of propulsion as the forefoot is everting. The extension also serves to change the proportion of weight distribution across the metatarsal heads in standing or in gait. As in Rodgers & Cavanagh (1989) the second metatarsal head bears a greater amount of pressure and a Morton’s extension aims to distribute more of the pressure to the first metatarsal which is a much stronger bone and therefore able to carry a greater load.

When pain or callusing under the second metatarsal head occurs, a metatarsal pad could be placed under the forefoot to relieve pressure. This pad is designed to disperse pressure away from the head of the metatarsal by lifting up under the shaft of the bone 6–11mm behind the metatarsal line. It can support metatarsals two through four, but should be highest under the second (Hastings, 2007). This pad can be used in conjunction with a flexible Morton’s extension or a forefoot varus post attached to an orthosis.

Rocker soles may be used to treat pain and callusing of the second metatarsal head, as well as diabetic ulcers (Bus, van Deursen, Kanade, Wissink, Manning, van Baal, & Harding, 2009). In the treatment of Morton’s syndrome, this rocker should start at approximately 5/8 of the length of the shoe at the first MTP joint line. The start of the rocker should be proximal to a normal rocker sole to compensate for the short first metatarsal. As the foot prepares for propulsion weight starts to shift onto the forefoot.

In severe cases of Morton’s syndrome where symptoms are debilitating, an orthopaedic surgeon may operate to lengthen the first metatarsal (Benson, 2008; Decherchi, 2005; Hurst, 2007). This falls under the category of brachymetatarsal surgeries. In cases where the second metatarsal is symptomatic (recurrent corns and calluses, hammer toes, recalcitrant metatarsalgia, or metatarsal fractures) and the second metatarsal bone is excessively long compared to the first and the other lesser metatarsal bones, surgeons may choose to shorten the second by an osteotomy. Where more correction is required, the distraction lengthening technique is preferred, as it is possible to gain 10mm in length of a short metatarsal bone, while osteotomies are limited to 5mm due to potential complications (Casteel, 2010). The purpose of the surgery is to establish a normal parabola of the metatarsal heads. Since Morton’s syndrome is most often due to a congenitally short first metatarsal bone, distraction lengthening of the first would be the preferred treatment for this condition if conservative methods have failed to treat the symptoms.

## References


Morton’s (Foot) Syndrome


Morton’s Neuroma

Brad J. Gibbs, BSc (Kin), C. Ped (C)

Key Messages

• Morton’s neuroma is a compression neuropathy of the common digital nerve, typically seen in the webspace between the third and fourth metatarsal heads.
• Utilizing the four clinical tests yields a high degree of accurate diagnosis.
• Insight into the differential diagnoses is important.
• Foot pathomechanics, footwear, and lifestyle are important considerations.
• Using diagnostic ultrasound and magnetic resonance imaging and complementary treatment modalities enhances pedorthic treatment outcomes.

Keywords

fibrous nerve tissue, interdigital neuropathy, Mulder’s click, pinching, plantar nerves, squeeze test, thickening, webspace

Introduction

Thomas Morton was an orthopaedic surgeon practising in Philadelphia, Pennsylvania, in the 1870s. He described “a number of cases of a peculiar and a painful affection of the foot, which, so far as I am aware, has not been described” (Morton, 1876). The painful condition related to a lesion to the common plantar digital nerve of the third interspace bearing his name. In reality, he described the pain as being localized to the fourth metatarsophalangeal articulation, with possible neuralgia due to the bruising or pinching of the digital branches of the plantar nerve. In his examination of 15 cases, he described a greater frequency occurring in women. Morton attributed the pain to either injury to the fourth toe, or gradual onset from shoe pressure, or to no recognized cause. He believed that a greater prevalence in females was attributed to both the greater delicacy and pliability of the female foot and to wearing tighter fitting narrow shoes. Dr. Morton treated this painful condition by blood-letting and extended rest until sensitivity had subsided, or by complete excision of the metatarsophalangeal joint, the nerves (likely on either side of that joint), and amputation of the fourth toe (Morton, 1876).

A review of the historical literature demonstrates that Morton was not the first to describe a neuralgia–like condition of the third webspace. In 1835, a paper written by Dr. Civinini entitled “On a ganglionic swelling of the sole of the foot” describes and illustrates the third common digital nerve neuroma as a fusiform swelling that was also associated with a valgus deformity of the big toe (Pisani, 2010). In 1845, Durlacher described a neuralgia involving the plantar nerve in the space between the third and fourth metatarsal joints, and specified the clinical signs known today (Adams, 2010; Pisani, 2010). The first description of the presence of a mass in the third interspace was by Tubby in 1912 (Pisani, 2010). Although originally referred to III–IV interdigital neuropathy, today the term “Morton’s neuroma” is used for all interdigital neuropathy (Oh, 2007). Morton’s intermetatarsal neuroma is a mechanically induced compression neuropathy of the common digital nerve (Owens, Gougoulias, Guthrie, & Sakellariou, 2010), most commonly seen in the third intermetatarsal space. The second most common site for an interdigital neuroma is the second webspace, described by Dr. EDW Hauser in 1950 (Larson, Barrett, Battiston, & Maloney, 2005). A neuroma may occur in more than one intermetatarsal space and may be bilateral. Studies have suggested the occurrence at approximately 30% (Rout, Tedd, Lloyd, Ostlere, Lavis, Cooke, & Sharp, 2009). The occurrence of an interdigital neuroma is rare in the first or fourth intermetatarsal spaces.
Pathology

Anatomically, the tibial nerve enters the foot by passing through the tarsal tunnel, the space between the medial malleolus and the flexor retinaculum. It then bifurcates into the medial and lateral plantar nerves (Netter, 2014). The medial plantar nerve supplies sensory innervation to the skin along the plantar side of the first three toes and the medial half of the fourth toe as well as to the medial anterior two-thirds of the sole. The distal branches of the medial and lateral plantar nerves ultimately form the interdigital nerves.

These pass between the distal heads of the metatarsal bones, cross the plantar surface of the transverse metatarsal ligaments, and supply sensation to the webspace between the toes. The III–IV interdigital nerve is formed by a branch from the medial and lateral plantar nerves.

The term “neuroma” is actually a misnomer for this condition. Neuroma implies that the swelling of the nerve distal to the transverse metatarsal ligament consists of an abnormal overgrowth of nerve tissue. In fact, the symptoms are brought about by degenerative nerve fibres with extensive perineural fibrosis which is the development of fibrous tissue surrounding the nerve (Adams, 2010). The lesion is likely due to repeated trauma or compression of the nerve before it divides to innervate the adjacent toes, typically within the third and fourth intermetatarsal webspace (Adams, 2010; Ahearne & Rosenfeld, 2010; Kincaid & Barrett, 2005; Oh, 2007; Wu, 1996).

The high frequency of occurrence of Morton’s neuroma in the third intermetatarsal space is explained by both anatomical and biomechanical contributing factors (Wu, 1996). As noted above, the third common digital nerve is potentially thicker because of its formation by branches of both the medial and lateral plantar nerves. Although the correlation between a thicker nerve and the development of Morton’s neuroma is not supported by all literature (Frank, Bakkum, & Darry, 1996), this potentially thicker nerve, which is tethered in the third webspace, is more vulnerable to repetitive trauma of activities of daily living.

Secondly, the fourth metatarsal is more mobile than the third, thus subjecting the third common digital nerve to more stretching and shearing forces.

Thirdly, high heeled shoes create a rise in the body’s centre of gravity. This results in a significant reduction of the base of support of the weight-bearing foot and hyperextension of the metatarsophalangeal joints which bring the common digital nerve against the unyielding transverse intermetatarsal ligament. Couple this with compression of all the metatarsal heads from a narrow toe-box and the combined contribute to harmful trauma to the well-being of the third common digital nerve (Wu, 1996; De Maeseneer, 2015).

Pain is typically worse with weight-bearing and ambulation, but it can also occur while at rest (Adams, 2010; Woods, Cervone, & Fernandez, 2004). There may be a sensory deficit in the involved toes (Ata, Onat, & Özçakar, 2016; Thomas, Blitch, Chaney, Dinucci, Eickmeier, Rubin, & Vanore, 2009). The affected digits may be splayed (Thomas et al., 2009).

Differential Diagnoses

The differential diagnosis of Morton’s neuroma includes inflammatory, neurological, and biomechanical causes:

- Metatarsal stress fractures (Thomas et al., 2009; Wu, 1996)
- Neoplasm (e.g., Rheumatoid nodule, soft tissue tumours) (Sharp, Wade, Hennessy, & Saxby, 2003; Thomas et al., 2009; Wu, 1996)
- Bursitis (Adams, 2010; Thomas et al., 2009; Wu, 1996)
- MTP joint pathologies (e.g., Freiberg’s disease) (Sharp et al., 2003; Thomas et al., 2009)
- Infection (Sharp et al., 2003)
- Metabolic neuropathy (Thomas et al., 2009)
- Fibromyalgia and other chronic pain syndromes (Thomas et al., 2009)
- Tarsal tunnel syndrome (Adams, 2010; Wu 1996)
- Lumbar radiculopathy (Adams, 2010; Wu, 1996)
- Peripheral neuropathy (Adams, 2010; Wu, 1996)
- Capsulitis (Adams, 2010)
- Metatarsalgia (Adams, 2010)
- Painful plantar callosity associated with hammer toes and claw toes (Wu, 1996)

Common Testing

i) Webspace tenderness test

Webpace compression will produce severe pain as the affected nerve is squeezed between thumb and finger from a dorsal and plantar position. A palpable mass
may be felt between the fingers when massaging the webspace. Occasionally, this causes paresthesia into the toes (Adams, 2010; Rout et al., 2009; Wu, 1996). Simultaneously squeezing the metatarsal heads together with the other hand or translating the two adjacent metatarsal heads relative to each other may produce a painful and palpable click called a “Mulder’s sign”. This causes a sudden displacement of the neuroma from between the two adjacent metatarsal heads (Mulder, 1951; Thomas et al., 2009; Wu, 1996).

### Webspace tenderness test. Photo by Deborah Wolf.

#### ii) Foot squeeze test

Squeezing the metatarsal heads together (medial to lateral pressure) with one hand (Gauthier’s test) while actively dorsiflexing and plantarflexing the digits for 30 seconds will replicate symptoms as the nerve is pinched between the adjacent metatarsal heads. A positive test results in pain to the patient or a sensory abnormality (DeHeer & Werber, 2006; Thomas et al., 2009).

### Foot squeeze test. Photo by Deborah Wolf.

#### iii) Plantar percussion test

Percussion over the site of the neuroma while passively extending the toes may cause paresthesia (a positive Tinel’s sign) in the associated toes, or pain (Owens et al., 2010; Thomas et al., 2009). A modified version involves passively hyperextending the toes and rolling the thumb of the examiner in the area of symptoms. This maneuver may reveal a tender, thickened, longitudinal mass.

### Plantar percussion test. Photo by Deborah Wolf.

#### iv) Toe tip sensation deficit test

Applying a pin prick or the extended end of a paper clip to the adjacent aspects of the toes or the plantar aspect of the adjacent toes may reveal reduced sensation (numbness, paresthesia) (Adams, 2010; Di Caprio, 2017; Owens et al., 2010).

### Toe tip sensation deficit test. Photo by Deborah Wolf.

#### v) Digital nerve stretch test

The digital nerve stretch test is performed with the patient seated with the feet upon the examiner’s knees. Both ankles are held in full dorsiflexion, whilst the lesser toes on either side of the suspected webspace are passively and fully extended on both feet. The test is positive if the patient complains of discomfort in the webspace of the affected foot (Cloke & Greiss, 2006).
The literature confirms diagnostic ultrasound as a valuable and highly accurate diagnostic tool (Kincaid & Barrett, 2005). The neuroma appears as an ovoid mass parallel to the long axis of the metatarsals (Adams, 2010; Rout et al., 2009). Interpretation of an ultrasound scan is straightforward and uncomplicated (Gomez, Jha, & Jepson, 2005).

Radiographs are useful to rule out metatarsal stress fractures and other metatarsal pathologies but neuromas will not be visible on X-ray (Adams, 2010). MRI may reveal the neuroma as a low density mass (Adams, 2010).

**Contraindications**

Additional forefoot padding or a neuroma pad that is too thick may make the shoe too tight.

**The Patient Perspective**

The literature emphasizes diagnosing Morton’s neuroma based on the patient’s history and physical findings. These details are often enough to establish the correct condition.

The diagnosis might be validated when a patient describes any or all of the following:

- A perceived “lump” or feeling of walking on a rolled-up or wrinkled sock or the seam of the sock
- A feeling of relief walking barefoot or with massage on removing footwear
- Sharp, dull, or throbbing pain
- Radiating, aching, burning pain localized to the plantar aspect of the intermetatarsal space
- Numbness and tingling (paresthesia)
- Symptoms may radiate into the adjacent toes, described as shooting sensation (Ata et al., 2016; Di Caprio, 2017; Gabriel, 2004; Rout et al., 2009; Thomas et al., 2009; Woods et al., 2004; Wu, 1996).

**Common Treatment**

Pedorthic treatment protocol may initially consist of the use of a neuroma (or metatarsal) pad placed in existing footwear that has sufficient toe-box width and depth. This pad is placed proximal to the metatarsal heads (ideally peaked at the third webspace) to provide space between the metatarsals to relieve pressure on the pinched nerve. This can provide a more cost effective method of controlling the symptoms.

If the severity of the pathology warrants further intervention, pedorthic treatment would consist of custom foot orthoses that are designed to control excessive rearfoot motion, typically eversion. The orthoses will address forefoot equinus (if present) and will feature a metatarsal pad or neuroma pad (either elongated or narrow) placed between and just proximal to the affected metatarsal heads (within 10mm). This positioning likely separates the metatarsal heads to decrease pressure and trauma on the involved digital nerve. Appropriate footwear consists of a wide, deep toe-box with lower heels to minimize metatarsal head pressure. The addition of a metatarsal bar or shoes with rockerbottom soles or stiff soles may enhance the orthosis efficacy by reducing MTP extension during the push-off phase of gait (Decker & Albert, 2002; Rout et al., 2009; Valmassy, 1996; Wheeless, 2011; Woods et al., 2004).

If pedorthic treatment fails to improve symptoms, consider referral to an allied health care professional for appropriate modalities like ultrasound or laser treatment.

Three cycles of radiofrequency ablation provide a high rate of patient satisfaction (Brooks, Parr, & Bryceson, 2017).

The literature also recommends invasive treatment of ultrasound guided injection using a combination of steroids and local anaesthetic to relieve symptoms. Other surgical options include neurolysis, percutaneous electrocoagulation, cryogenic denervation of the intermetatarsal neuroma, and surgical decompression (Kasparek & Schneider, 2013; Kincaid & Barrett, 2005; Rout et al., 2009).

**Key Considerations**

Neuromas are more prevalent in adults beginning in the third decade of life, and are more common
in females than males (Owens et al., 2010; Thomas et al., 2009). Typically, neuromas most often afflict fashion-minded women between the ages of 40 to 60 who wear high-heeled narrow-fitting footwear (Wu, 1996). Ultrasound is as accurate as MRI in diagnosing Morton’s neuroma (Bignotti, Signori, Sormani, Molfetta, Martinoli, & Tagliafico, 2015).

References


Metatarsophalangeal (MTP) Joint Synovitis

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Key Messages

- MTP joint synovitis is mainly a second MTP joint issue.
- MTP joint synovitis tends to affect those with rheumatoid arthritis, gout, and osteoarthritis.

Keywords

anterior drawer test, crossover toe, gout, hammer toe, inflammation, medial displacement, Mulder’s click, osteoarthritis, rheumatoid arthritis, second MTP joint

Introduction

Synovitis is the inflammation of the synovial membrane. This membrane lines joints which possess cavities, which are known as synovial joints. The disorder can be (and usually is) quite painful. The joint usually swells due to the influx of synovial fluid.

It may be caused by rheumatic fever, rheumatoid arthritis, tuberculosis, trauma, gout, or other conditions (Dorland’s Medical Dictionary, n.d.).

Usually, but not always, synovitis affects the second MTP joint. Instability and synovitis of the lesser MTP joints is a common cause of forefoot pain. Some studies have shown that close to 30% of patients presenting with metatarsalgia were diagnosed with MTP joint synovitis. Peck, McLeod, & Barrie (2011) found that the condition appears to be common in middle-aged women (82% of patients in this study) with the median age of 56.5 years.

Although this is also a common condition occurring in males and females of all age groups, some feel there is a distinct group of mainly male athletes with a lower average age (Foot and Ankle Hyperbook, 2005).

Pathology

MTP joint synovitis with vertical instability was first characterized by Mann & Mizel (1985), who deemed this instability a result of synovitis, possibly linked with hallux valgus.

Early research concentrated on the idea that synovitis stretched the joint capsule and the collateral ligaments. The collateral ligaments are significant stabilizers of the MTP joint (Deland, Sobel, Arnoczky, & Thompson, 1992). Bhatia, Myerson, Curtis, Cunningham, & Jinnah (1994) found that separation or tear of the plantar plate lessened MTP joint stability by 30% and division of the collateral ligaments by 46%. In the mid-1990s, greater emphasis was placed on the involvement of the plantar plate. At first, the consensus was that the plantar plate detached at its proximal border (Fortin & Myerson, 1995). However, Yao, Do, Cracchiolo, & Farahani (1994) demonstrated that plantar plate tears most often occur nearer to the proximal phalanx insertion point.

Some studies have revealed that plantar tears are not entirely uncommon in feet that are otherwise normal. Evidence also suggests that there is an increase of plantar tear incidence in older population groups. Gregg, Silberstein, Schneider, & Marks (2006) discovered plantar plate tears in 35% of normal feet examined by ultrasound or MRI. Powless & Elze (2001) found plantar plate tears in 41%, collateral tears in 33%, and combined lesions in 26% of the 58 feet tested by plain arthrogram (Foot and Ankle Hyperbook, 2005).

Synovitis is a significant complication in both rheumatoid arthritis and psoriatic arthritis. It may also be associated with trauma, lupus, or gout. In rheumatoid arthritis, the synovial membrane lining the joint becomes inflamed. The cells in this lining then divide and inflammatory cells move into the joint from other areas of the body. The inflammation
stretches the ligaments of the joint, thus reducing its inherent stability. Inflammation is accompanied by an increase in blood flow and this causes the affected joint to feel warm. The joint also has a swollen appearance and feels puffy or spongy to the touch. In the case of rheumatoid arthritis, the cells discharge enzymes into the joint, causing more pain and irritation. This process, if continued over a long period of time, may allow for the enzymes to slowly absorb the cartilage. Synovial fluid is a transparent fluid secreted by the synovial membrane and found in joint cavities, bursae, and tendon sheaths.

Relevant anatomical changes resulting in MTP joint synovitis:

• Failure of the lateral collateral ligament
• This causes medial deviation of the second toe
• The plantar plate and its flexor tendon attachments, displace medially
• Medial displacement of the proximal phalanx relative to the metatarsal
• The medial structures then become contracted or tightened
• The interosseous and lumbral tendons, the medial cruciate ligament (MCL), and the medial capsule contract and become tight, thus producing adductor force
• The plantar plate ultimately fails
• Dorsal instability results from continuous force of hyperextension on the proximal phalanx (Orthobullets, n.d.)

Differential Diagnosis

Articular (joint) misdiagnoses:

• Capsular tear
• Avascular necrosis/Freiberg’s infarction
• Predislocation syndrome
• Plantar plate tear/rupture
• Arthrosis
• Capsulitis
• Pigmented villonodular synovitis

Extra-articular misdiagnoses:

• Flexor tenosynovitis
• Metatarsal stress fracture
• Morton’s neuroma or interdigital neuroma
• Forefoot fat pad atrophy

Common Testing

Observation:

• Toe may be thickened, more curled than the adjacent toes, and may have hammer toe and/or crossover toe deformity
• Red and swollen joints
• Elevated toe in comparison to adjacent toes

Palpation:

• Tenderness to the touch, especially under the plantar plate
• Instability of the joint
• Pain in and around the MTP joint (usually the second), accompanied by palpable fullness (puffiness), warmth, and tenderness

Range of motion:

• Instability, subluxation, or dislocation of the MTP joint in extension.
• In plantar flexion, the range of motion may be limited or decreased.

Special tests:

Anterior drawer test: hold the metatarsal head between the index finger and thumb; grasp the base of the proximal phalanx with the other hand and apply a dorsally directed force. Dorsal displacement and pain indicate MTP joint instability or MTP joint synovitis. Plantar flexion ROM is decreased (Mizel, 1995).

The Thompson draw test evaluates instability with the MTP joint flexed 20 degrees and the proximal phalanx drawn up and down.

• Grade 1 – <50% vertical translocation
• Grade 2 – 50–100% translocation
• Grade 3 – dislocatable (Mizel, 1995)

Mulder’s click or pain is elicited on palpation, most commonly at the third interspace. This is done to differentiate between synovitis and neuroma and confirms the latter.

A joint disorder is indicated when some or all of the following are present:

• Pain is elicited upon palpation of the affected metatarsal head
• Range of motion in plantar flexion is limited
• Plantar flexion results in pain

Metatarsophalangeal (MTP) Joint Synovitis
• The toe associated with the affected joint is superior to (or elevated above) the adjacent lesser toes
• The associated toe is crossed over or is a hammer toe
• Range of motion in dorsiflexion is excessive (Alexander, 1997).

Medical imaging:
• Synovial fluid analysis examines the lubricating fluid secreted by synovial membranes. This test is useful in the diagnosis of some types of arthritis, primarily those caused by infection, gout, or pseudogout (Orthosports, 2009).

Contraindications
• Avoid adding metatarsal pads and padding to shoes that are already too tight, too shallow, or too short.
• Avoid simply fitting your client into shoes with a lower heel, especially with those who have limited sagittal plane range of motion, as this can actually result in greater force being transferred to the metatarsals.
• Avoid inflexible footwear with hard, flat soles. A stiff soled shoe with a firm toe rocker feature is desirable and recommended. The inner sole and underlay should always be soft.
• Be cautious with a sulcus placement: ensure it is not too proximal, which irritates the synovitis area.

The Patient Perspective
The patient might describe:
• Pain at or under the affected joint
• Walking on a “marble” in the ball of their foot
• Tender, warm, swollen sensation
• Hammer toe or crossover toe deformity; redness at PIP joint from rubbing on the shoe (Orthobullets, n.d.)

Common Treatment
Pedorthic
• Calf muscle stretching to reduce forefoot pressures. It is best to demonstrate the proper way to stretch and to have your client try it to be sure they are doing it correctly.
• Icing of the forefoot.
• Taping the affected toe in mild plantar flexion (crossover taping: loop the tape over the dorsal aspect of the relevant proximal phalange and cross over on the plantar side, adhering to foot).
• Cushioned forefoot padding.
• Rocker–soled shoes, or rocker sole modifications to existing shoes. A proper toe rocker shoe will reduce toe dorsiflexion, and thus reduce MTP plantar flexion. The rocker sole will take much of the burden of propulsion off the metatarsal heads.
• Metatarsal dome pads or metatarsal bars, if placed correctly.
• Custom–made orthoses or modified over–the–counter arch supports incorporating the necessary offloading attributes (including but not limited to metatarsal arch, sulcus, or toe crest, and indenting at the site of inflammation).
• Appropriate footwear: proper width across the ball of the foot, proper length, removable and modifiable insole, forefoot rocker sole, soft midsole.

Note: If the synovitis occurs at the second MTP joint, check the following:
• First ray incompetence, weak plantar flexion of the 1st ray – try using a first ray cutout, 2–5 bar, or dancer’s pad.
• Hallux rigidus – try using a Morton’s extension modification to orthoses or insole.
• Prominent second metatarsal head – try using a “U” pad.

Medical
• Initial treatment: NSAIDs and a rheumatologic workup to rule out systemic causes
• Secondary treatment: intra–articular corticosteroid injection
• Surgical treatment: MTP joint exploration via a dorsal approach with debridement, synovectomy, and MTP joint reduction. May require release of the collateral ligaments and the extensor digitorum longus tendon for MTP joint reduction (Mizel, 1995). Usually about 70% of patients can be managed non–surgically; the remainder may require surgery. Common surgical procedures include shortening osteotomies and tendon transfers. Surgery is effective 80–90% of the time (Orthosports, 2009).

Key Considerations
• Often MTP joint synovitis is associated with rheumatoid arthritis conditions; as such, the onset of RA is usually between the ages of 40–50.
• Improper footwear (too small, too stiff) can aggravate this condition.

References


Flexor Tenosynovitis

Ernest Porthouse, Dip AA, C. Ped Tech (C), C. Ped (C)

Key Messages

- FT for this paper refers only to the flexor digitorum longus and flexor hallucis longus tendons.
- FT is common in athletes, especially those with frequent ankle injuries; however, FT is also associated with chronic inflammation resulting from diabetes, arthritis (especially rheumatoid), repetitive stress, gout, thyroid problems, and infection.
- Early intervention is directly related to a favourable outcome; reducing stress to the tendon is paramount; pedorthic treatment would include both rigid rocker bottom footwear and orthoses for maintenance of proper biomechanical alignment (especially when overpronation and ankle equinus are contributing factors).

Keywords

ankle pain/swelling, flexor tendons, synovial sheaths, synovium inflammation, tendon pathway

Introduction

Flexor tenosynovitis (FT) is common and often a source of pain in the soft tissues of the hindfoot and ankle. This condition is generally assumed to involve the flexor digitorum longus and flexor hallucis longus tendons. Early intervention with regard to FT is directly related to a favourable outcome. A complete history is instrumental in making an accurate assessment, as flexor tenosynovitis is common in the athlete and particularly where frequent ankle injury is present.

Flexor tenosynovitis is also associated with (and can be secondary to) chronic inflammation resulting from diabetes, arthritis (especially rheumatoid), repetitive stress, gout, and thyroid problems, and is often the result of infection (Bibbo, Delmi, Hyer, Jacobs, & Roukis, 2009). Steroid use also increases the chance of developing FT.

There are many variations in the terms that are commonly used to describe this condition (tendinitis, tendinosis, paratenonitis, tenovaginitis). This chapter uses “tenosynovitis,” which is defined as “inflammation of the fluid-filled sheath (synovium) that surrounds a tendon and disrupts normal tendon function.” It is estimated that up to 36% of tenosynovitis occurs in the foot and ankle (Di Giovanni & Greisberg, 2007).

Pathology

The flexor tendons are subjected to tensile, compressive, and shearing forces, as well as traumatic damage from proximal bony structures, as they pass around and under the ankle. These all contribute to pathology. Anatomical abnormalities are often contributors as well (Di Giovanni & Greisberg, 2007).

Because the varied causes and contributors of FT are common among all age groups, the age of the patient should in no way prejudice the clinician during assessment. The proximity of these tendons to one another, and to other structures, complicates the process of assessment (Di Giovanni & Greisberg, 2007).

Inflammation of the proximal sheath of the flexor hallucis longus (FHL) is uncommon in the general population; however, it is most often seen in ballet dancers, as the common “en pointe” position is extremely offensive to the FHL (Tudisco & Puddu, 1984). Inflammation of the great toe flexor occurs at the posterior margin of the ankle where it passes in a groove between the medial and lateral tubercles of the posterior process of the talus. In the early stages, tenderness in the deep posterior ankle, aggravated by passive extension of the hallux, as well as resisted plantar flexion of the hallux, is the primary physical sign. In advanced cases, localized tendon swelling proximal to the talar tubercles can result in stenosing tenosynovitis, triggering of the hallux, and functional hallux rigidus. When this occurs, passive extension of
Eroded bone may fray tendons by a process of attrition and periarticular inflammation, thus weakening tendons. Tendon rupture is a frequent and serious consequence of tenosynovitis (Gluck, Heckman, & Parekh, 2011). Systemic inflammatory disease is frequently revealed in the feet and can be a cause of FT. If suspicion arises, then it is paramount that the clinician asks about family history, and symptoms affecting other joints (hips, wrists, knees) and associated tendons. Referral back to the physician is essential if systemic inflammatory disease is suspected (Bibbo et al., 2009).

**Presentation**

Patients may complain of stiffness or pain during or after activity. Rubor or swelling may be present about the synovia. Crepitus is also common in FT when the affected tendon is in motion.

**Anatomy**

FT is extremely difficult to distinguish from many other conditions. For this reason, a detailed understanding of the anatomy of both FHL and flexor digitorum longus (FDL) is necessary. The FHL originates from the distal posterior surface of the fibula with a portion coming off the covering fascia and adjacent fascial septum. Its main action is to flex the great toe at all joints and weakly plantar flex the ankle. It also helps support the medial longitudinal arch. Innervations are the tibial nerve, S2, and S3. The tendon lies in the most posterolateral portion of the flexor retinaculum, cradled between the medial and lateral talar tuberosities on the posterior aspect of the talus. The tendon runs through a fibro-osseous tunnel as it passes inferior to the ankle. Its boundaries are: medially, medial tubercle of the talus; anteriorly, talar body; posteriorly, flexor retinaculum; and laterally, lateral tubercle of the talus. It inserts in the proximal plantar aspect of the distal phalanx.

The FHL has two distinct synovial sheaths. The proximal sheath starts 2cm proximal to the ankle joint, continues through the fibro-osseous tunnel past the master knot of Henry, and terminates at the naviculocuneiform joint. The distal sheath starts at the base of the first metatarsal, continues with the tendon as it passes through the intersesamoid space, and terminates with the insertion of the tendon on the distal phalanx.

The proximal attachment of the FDL is the medial part of the posterior surface of the tibia, inferior to soleal line and by a broad tendon to the fibula. It passes behind the medial malleolus just posterior/inferior to the tendon of the posterior tibialis. It then passes anteriorly and crosses the tendon of the FHL, to which it is attached. From this point it proceeds obliquely and is joined by the quadratus plantae muscle, as it divides into the four lesser digits.

The FDL terminates at the bases of the distal phalanges of the four lateral digits. It has one primary synovial sheath, starting 2cm proximal to the ankle joint, continues through the fibro-osseous tunnel past the master knot of Henry, and terminates at the naviculocuneiform joint.

FDL is a strong lesser toe and foot plantar flexor and midtarsal joint supinator.

**Differential Diagnosis**

Flexor tenosynovitis is difficult to distinguish from plantar capsulitis unless one looks for specific distinguishing historical features and physical findings with regard to tenosynovitis of the most distal tendon sheath. The most important distinguishing diagnostic feature between FHL and plantar capsulitis is tenderness along the course of the tendon proximal and distal to the metatarsal head, which is accentuated by resisted plantarflexion of the involved digit (Alexander, 1997).

**Common Testing**

Early intervention with regard to FT is directly related to a favourable outcome. A complete history is instrumental in making an accurate assessment, as flexor tenosynovitis is common in the athlete and particularly where frequent ankle injury is present (Alexander, 1997).

Tendon rupture is a frequent and serious consequence of tenosynovitis. Accurate assessment is often clouded by confined tendon pathways and highly varied symptoms. Patients regularly report generalized discomfort, and edema of adjacent structures can be confused with otherwise palpable tendons. Pain may be located posteromedially and is frequently described as being within or just behind the ankle (Di Giovanni & Greisberg, 2007).

Imaging would appear to be the most conclusive method for confirming cases of flexor tenosynovitis. Although MRI can be used with some success (Masala, Fiori, Marinetti, Uccioli, Giurato, & Simonetti, 2003),
the gold standard for confirming cases of flexor tenosynovitis would appear to be ultrasonography. If there is clinical suspicion of tendon pathology, the ability to dynamically assess tendons by passive and active movements during imaging allows for a reliable assessment of tendon integrity (Loredo, Rahal, Garcia, & Metter, 2010). In stenosing tenosynovitis, synovial proliferation and fibrosis within the tendon are evident, causing entrapment. An X-ray technician experienced in imaging for tenosynovitis would be invaluable for accurate diagnoses (Brown & Brown, 1999).

**Common Treatment**

Management of flexor tenosynovitis is highly dependent on the severity of the condition, ability of the patient to avoid stressing the tendon, and (in such cases) whether the patient has a systemic inflammatory condition and whether it is under control. Pedorthists should recognize the limits of the profession. With this in mind, it is imperative that the pedorthist provide other health care practitioners with quality information gained from exhaustive and comprehensive assessment. Physiotherapy/athletic therapy (or similar) is often involved in treatment. Surgery is obviously a possible treatment in more advanced cases, especially when anatomical anomalies are detected.

Pedorthic management of flexor tenosynovitis can be an effective tool and can provide patients with excellent care for this condition where surgery is not recommended. Both footwear and foot orthoses should be considered. Rigid rocker bottom shoes, or modifications to existing shoes, can accomplish much in treating FT by reducing flexor stresses. Foot orthoses used to maintain proper biomechanical alignment (particularly in cases of severe overpronation or ankle equinus) can help reduce stress to the flexor tendons along their pathways. Off-weighting of painful inflamed tendons near insertion is suggested. When rigid forefoot rocker footwear is not possible or desired, a rigid carbon plate (or similar) inside the footwear, or a rigid extension on the orthoses, is recommended.

**References**


Plantar Fibromas

Linda Deschamps, BSc (Kin), C. Ped (C)
Julia Miller, PTA, OTA

Key Messages

• A plantar fibroma is a benign proliferative condition of a plantar nodule of the foot.
• The main cause of this condition is uncertain.
• A patient who presents with plantar fibroma should be investigated for Dupuytren’s contracture
• A conservative treatment plan should include properly fitted footwear and corrective foot orthoses to help cushion and offload symptomatic plantar nodules.

Keywords

benign, Dupuytren’s, fibromatosis, Ledderhose’s, Morbus Ledderhose’s, nodule, plantar

Introduction

The plantar fibroma condition was first described in 1897 by the German surgeon Georg Ledderhose. A plantar fibroma is a single lesion of a proliferation of mature fibroblasts associated with mature collagen (ligamentous material) typically present on the plantar fascia. It is a non-malignant (benign) thickening of the foot’s deep connective tissue which grows on the plantar surface, or fascia, of the foot. Nodules can develop in one or both feet and, although generally asymptomatic, can be painful to walk on. They are usually slow growing and typically measure less than an inch in size. These common growths are usually singular, although multiple nodules are possible in the same foot. Plantar fibromas usually appear in people aged 20–60, and men are more likely to be affected. More invasive, rapid-growing, and multi-planar fibromas are considered plantar fibromatosis or Ledderhose’s disease. This more aggressive form of the plantar fibroma condition consists of one or more painful masses on the bottom of the foot, roughly in the middle of the medial longitudinal arch. This mass can cause a soft convexity in the contour of the bottom of the foot that may be painful with pressure or shoe wear. (American Orthopaedic Foot & Ankle Society, n.d.).

Pathology

Etiology of the plantar fibroma condition remains controversial, with a theory of a possible genetic predisposition. Northern Europeans note a higher than normal rate of fibrotic conditions. Although prior trauma to the plantar fascia is considered an unlikely cause, some suspect these tears in the fascia may promote fibroma growth. Excess collagen and fibrous tissue growth may occur with the use of certain high blood pressure medications, anti-seizure medications, vitamin C, and glucosamine. Other predictors of plantar fibroma (coexisting morbidities) include diabetes mellitus, epilepsy, thyroid conditions, keloids, smoking, chronic stress to the feet, and alcoholism with liver disease.

Plantar fibroma in the foot is similar to Dupuytren’s contracture in the hand, with both deformities developing over years. Where Dupuytren’s contracture can result in thickened nodules in the palm of the hand with possible bent “frozen” fingers, plantar fibroma can cause similar results in the foot. For reasons unknown plantar fibroma is less frequent than Dupuytren’s disease but appears often in combination with Dupuytren’s contracture (International Dupuytren Society, 2016a).

Although plantar fibromas can occur in
children, incidence increases with advancing age. In a large study (501 patients) 44% of patients were less than 30 years of age. Men are affected twice as often as females and these nodules or lesions are bilateral in 20–50% of cases. Patients present with one or more subcutaneous nodules, which most frequently arise in the medial aspect of the plantar arch (78%) and can extend to the skin or deep structures of the foot. Nodules are multiple in 33% of cases. Palmar fibroma is also present in 10–65% of patients with plantar fibroma. Most nodules are asymptomatic, only becoming symptomatic when it invades the adjacent structures such as neurovascular bundles, muscles, or tendons (Walker, Petscavage, Brian, Logie, Montini, & Murphy, 2012).

**Different Diagnosis**
The first and most important step is a correct diagnosis. Not every nodule in the arch of the foot is a plantar fibroma, plantar fibromatosis, or Ledderhose’s disease. Plantar “bumps and lumps” of the foot can also be:

- rheumatoid nodules
- fibrosarcoma (among other cancers)
- neurofibroma
- serous, tendinous, or mucoid cyst
- calcinosis
- scar or keloid
- fasciitis
- osteoma or calcaneal spur
- gout
- ganglion
- schwannoma

**Common Testing**
A thorough physical examination of the plantar foot often results in a palpable nodule or cyst on the plantar fascia. Possible imaging tests include MRI scan, X-ray, diagnostic ultrasound, or bone scan. A tissue biopsy may be performed for further investigation.

Many clinical conditions may have similar signs and symptoms. Your healthcare provider may perform additional tests to rule out other clinical conditions to arrive at a definitive diagnosis.

**The Patient Perspective**
In the early stages, a plantar fibroma usually causes little discomfort. It often appears as little more than a small bump along the plantar fascia. Patients may not notice the nodule unless it begins to grow in size. They may begin to experience pain or discomfort if the nodule becomes larger or if external pressure is applied to the affected area. This includes friction from wearing shoes, walking for extended periods, and standing barefoot. Although plantar fibromas usually do not cause discomfort, some patients experience persistent pain with little relief from conservative treatments.

Plantar fibromas are benign; however they generally will not improve (or reduce in size) without treatment.

**Common Treatment**
The treatment of a plantar fibroma is often conservative and consists of footwear modifications and orthoses aimed at reducing pain and discomfort through pressure relief and cushioning. Anecdotal evidence supports these pedorthic modalities and there is also some evidence base. Some of the most common conservative and non-conservative approaches to treating a plantar fibroma include:

**Conservative Treatments:**
- Properly fitted footwear with cushioned soles to help reduce soft tissue stress. As well, it is possible that rocker soles may reduce plantar stress
- Custom foot orthoses to maintain corrected foot structures and offload the nodules. Support to the medial longitudinal and metatarsal arches may help to reduce soft tissue strain of the plantar fascia, along with indentations at the plantar nodules
- Physiotherapy for strength training and stretching exercises for the plantar fascia to reduce its gradual tightening
- Rest/reduction in high impact and weight bearing activities to reduce stress to the plantar fascia

**Non-conservative Treatments:**
- Cortisone injections to manage pain and decrease inflammation
- Collagenase injections
- Radiotherapy to reduce the nodule’s size. The side effects of radiotherapy are minor, and this
treatment has been shown to be approximately 80% effective (Panizzon & Seegenschmiedt, 2014). This type of treatment can stop the disease progression and possibly dissolve the nodule in the initial stages when the nodule is tiny. In progressed stages with large nodules radiotherapy can shrink the nodules in size and relieve pain. Radiation therapy is also applied if surgery cannot be repeated due to excessive scar tissue (International Dupuytren Society, 2016b).

- Surgery to remove the plantar fibroma is a last resort. It may cause the medial longitudinal arch to flatten and fibromas can recur. Surgical resection is reserved for large nodules which cause significant disability and are refractory to non-operative methods of management. Surgical treatment consisting of simple excision resulted in high rates of local recurrence (20–40%), with the majority of lesions recurring within the first post-operative year (Walker et al., 2012).

- Cryosurgery is occasionally used to treat a plantar fibroma. A probe is inserted into the nodule and surrounding tissue. It is deeply frozen, typically with liquid nitrogen, and more recently with thinner probes using Argon or other gases. Cryosurgery “will destroy nerve tissue by causing extensive vascular damage to the endoneural capillaries or blood vessels supplying the nerves” and thus killing the nerve endings in this area. The purpose is to reduce pain and shrink the nodules (International Dupuytren Society, 2017).

References


Plantar Fibromas
Metatarsophaalangeal (MTP) Joint Capsulitis

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Key Messages

• Capsulitis is most commonly found in the plantar forefoot region of the foot, usually under the second metatarsal head.
• Common complaints include a “bruised feeling” or a “lump” under the second MTP joint, pain and swelling around the affected joint, and difficulty wearing closed-toe shoes.
• The condition is commonly associated with a hallux valgus deformity.
• Conservative treatment options are re-aligning the affected joint, preventing or slowing deformity, and cushioning the affected MTP joint.

Keywords

ball of the foot, hallux valgus, hyperextension, hypermobility, joint capsule inflammation, lump, plantar forefoot, plantar plate degeneration, vertical stress test

Introduction

Metatarsophaalangeal (MTP) joint capsulitis is the local inflammation of the plantar joint capsule in the ball of the foot (Valmassy, 1996). While capsulitis is very common and can occur at any joint in the forefoot, it often occurs beneath the second metatarsal head. Capsulitis is the inflammation of the capsule of a joint. The capsule is the fluid-filled outer covering that joins two bones to make a joint.

Pathology

The development of capsulitis is dependent on a number of contributing factors such as abnormal or poor foot mechanics, chronic stress to the joint, overloading the joint from an associated foot deformity, and/or inappropriate footwear (Premier Podiatry, 2005). Certain medical conditions such as rheumatoid arthritis and osteoarthritis can also predispose the joint to inflammation.

MTP joint capsulitis can be caused by the degeneration of the plantar plate complex of the MTP joints. The plantar plate complex is a fibrocartilaginous structure that helps support, stabilize, and reduce compressive loads on the metatarsal heads (Dilnot & Michaud, 2003). If attrition of this sling occurs, the metatarsal heads become unsupported and thus drop plantarly. With this plantar drop of the metatarsal heads, there is an increase in pressure and possible shearing of the metatarsal heads during weight-bearing (Valmassy, 1996).

Capsulitis in the second MTP joint can also be associated with hallux valgus. The hallux valgus deformity causes an increase in stress on the second metatarsal head, because of its varus/medial positioning relative to the second MT head. Another
cause of capsulitis can be longer second metatarsal bone, relative to the first metatarsal. This is called a Morton’s foot or Morton’s toe (Schuler, 2009). The longer the metatarsal bone the greater the tendency for capsulitis to occur. If the metatarsal heads are not loading properly or the metatarsal bones are too long or prominent, then overloading and inflammation of the joint can occur.

Excessive hypermobility of the foot leads to prolonged pronation during the propulsive phase of gait and places considerable stress on the second, third, and fourth metatarsals (Roy & Irvin, 1983). Repetitive hyperextension of the MTP joint – more likely associated with athletes – increases the chance of developing capsulitis.

MTP joint capsulitis is a progressive disorder if left untreated. If the inflamed capsule ruptures, it causes the supportive ligaments surrounding the toe to weaken and eventually dislocation of the toe occurs (Premier Podiatry, 2005). Once the second toe crosses over the top of the big toe, the joint has become dislocated and permanent damage has occurred. Surgery is then needed to reduce the deformity (Foot and Ankle Academy, 2017).

Differential Diagnosis
Capsulitis can be difficult to diagnose because of other related structures in the forefoot that can also become inflamed from biomechanical problems. Metatarsalgia is often diagnosed for general metatarsal pain. However, it is a non-specific diagnosis of pain in and about the metatarsal head or MTP joint and adjacent soft tissue structures. It is a description of a region of pain, not a specific diagnosis.

A nerve symptom such as a neuroma could be diagnosed, as the swollen capsule can put pressure on the adjacent nerves in the forefoot. There are also numerous fluid-filled tissue sacs in the forefoot called bursae. A misdiagnosis of bursitis could occur when one of these sacs becomes inflamed, as well as the surrounding tissues (Meditech Healthcare, 2011). Severe hallux valgus and/or bunions could cause capsulitis to be dismissed. They both result with the first metatarsal being affected and not bearing its share of weight causing uneven pressure across the ball of the foot and deviating medially by crossing under the second toe.

Other differential diagnoses can include a stress fracture, hammer toe deformity, degenerative joint disease, avascular necrosis, and synovitis. MTP joint synovitis is very similar to capsulitis. However, synovitis is usually described by a sharp or aching pain located directly in the ball of the foot due to an increase in the synovial fluid inside the joint (McKean, 2017). It is commonly found in association with a specific disease such as rheumatoid arthritis or gout.

Common Testing
A detailed patient history, with plantar palpation to the affected area, and a positive vertical stress test can be used to confirm diagnosis. The vertical stress test involves stabilizing the proximal metatarsal and elevating the related digit dorsally. Translocation of the digit greater than two millimeters is felt to be suggestive of a rupture (Dilnot & Michaud, 2003). X-rays can be helpful to evaluate the extent of the deformity and the amount of arthritis or damage within the joint. The amount of damage done to the capsule can be seen on the X-ray if the affected joint is injected with dye (Premier Podiatry, 2005). An ultrasound or MRI can also help to diagnose any ligament tears in the plantar plate structure.

Contraindications
It is very common to develop discomfort in the metatarsal region of the foot and to ignore the pain hoping for it to settle while the patient continues to apply a load to the affected area. However, continuous pressure and long-term inflammation causes damage to the joint resulting in weakening of the surrounding ligaments and ultimately a rupture to the capsule (Premier Podiatry, 2005). Following a rupture, the toe becomes de-stabilized, deformed, and prominent. Walking barefoot on hard floors would not be advised. Hard, thin-soled shoes are also not recommended.

The Patient Perspective
Common symptoms reported with capsulitis are pain under the central ball of the foot, swelling and redness around the joint, and the sensation of walking on a stone (Meditech, 2011). This last symptom is particularly noticeable when standing barefoot or in thin-soled shoes on hard surfaces. There could also be the development of a painful callus under the affected MT head that has dropped. With more advanced stages of capsulitis, patients might also notice a gradual hammering of the second toe (Premier Podiatry, 2005). This causes difficulty finding footwear to fit properly.

A patient might find their pain to be intermittent and not consistent, depending on their level of activity and chosen footwear. Capsulitis can definitely cause a
decrease in activity level, as well as changes in gait in order to offload the affected area. The changes in gait could lead to pain in other areas of the foot, ankles, knees, hips, and back (Schuler, 2009).

**Common Treatment**

Conservative treatment is the first approach for treating MTP capsulitis. Initially, rest and ice should be suggested, along with gastrosoleal stretching for patients with tight calf muscles. Avoiding high-heeled or any shoe with flimsy soles will also help. Further treatments could include taping the affected area, increased cushioning under the ball of foot, and custom-made orthoses to offload the affected area and to help re-align the affected joint. A cross-over taping technique is used in order to plantarflex the affected joint and reduce tension on the ruptured plantar plate (Dilnot & Michaud, 2003). A metatarsal pad or bar (alone or incorporated with a full-contact orthosis) to offload the second MT head can be invaluable as a primary choice of treatment. Depending on the positioning and mobility of the first metatarsal bone, a custom-made orthosis incorporated with a first ray pad under the first MT bone will force the first MT bone to meet the ground properly and bear weight normally thus shifting the abnormal weight off the second MT bone (Shuler, 2009). The use of a toe crest can also be incorporated to redistribute pressure if the metatarsal padding is not sufficient, or if a fixed toe deformity co-exists (Valmassy, 1996). Footwear design also plays a very effective role in relieving pressure in the forefoot. Shoes with a relatively thick, stiff rocker sole reduce bending at the MTP joints thereby allowing the patient to ambulate without having to make gait alterations to avoid pain. Footwear modifications such as external metatarsal bars or the addition of a forefoot rocker to the sole of the shoe will help reduce painful pressure under the ball of the foot. Conservatively, a treatment of injectable cortisone at the site of inflammation can often offer immediate and long-standing relief, especially when used in conjunction with an appropriate orthosis and footwear treatment plan. Physiotherapy treatment might also be considered. Once conservative treatments have been exhausted, surgical intervention can be an option. A long metatarsal bone could be shortened (shortened osteotomy) or a prominent metatarsal could be surgically raised (dorsiflexion osteotomy). If the metatarsal is too prominent, then an operation to raise it can be performed (dorsiflexion osteotomy) (Premier Podiatry, 2005). Other surgical approaches include straightening of a hammer toe, flexor-to-extensor tendon transfer, or a simple repair of the plantar plate or collateral ligament tears (Dilnot & Michaud, 2003).

**Key Considerations**

- MTP joint capsulitis most commonly affects the second toe.

- The patient who commonly presents with MTP joint capsulitis has abnormal foot mechanics increasing the stress to the ball of the foot region, hallux limitus, or hallux valgus deformity, a longer second toe (relative to the first), history of chronic use of ill-fitting footwear, and/or tight calf muscles.

- Conservative treatments work well for capsulitis, especially during the early stages, before the second toe starts to drift towards the big toe.

**References**


Stress Fractures

Graham Archer, BSc, C. PedTech (C), C. Ped (C)

Key Messages

- 95% of stress fractures occur in the lower limb, and they are frequently seen in athletes involved in high impact repetitive activities, military personnel, and osteoporotic patients.
- Overpronation, high medial arches, limb length discrepancies, structural forefoot varus, nutritional deficiencies, footwear, and training environments are among the factors that can contribute to stress fractures.
- Stress fractures are not often found on an X-ray, as radiological evidence does not occur until 4–6 weeks after injury when bone remodeling occurs. Most stress fractures are diagnosed via bone scan and MRI.
- Treatment programs must include a multifaceted approach and should include rest, gradual return to non-impact activities before a cautious return to impact activities, and splinting or bracing when necessary for pain-free ambulation and biomechanical correction.
- Custom orthoses have been shown to reduce the risk of stress fractures and to reduce symptoms when a patient is recovering from a stress fracture.

Keywords

heel impact, intense contraction, interarticular, localized pain, overuse, prolonged swelling, repetitive loading, rotational forces

Introduction

Stress fractures are bone injuries caused by repetitive stress, as seen in sports, strenuous exercise, and physical labour (MedicineNet.com, n.d.). They are common in athletes, military personnel (Kazimoglu, Karapinar, Sener, & Bozkurt, 2009) and other individuals that are involved in repetitive impact activities. In fact, it has been shown that 5% of all sport-related injuries are stress fractures (Helstad, Ringstrom, Erdmann, Jacobs, & Julsrud, 1996). Patients with osteoporosis are particularly susceptible to stress fractures in the feet and lower legs (Kazimoglu et al., 2009).

Risk factors for stress fractures can be training related, environmental, or biomechanical. Sudden onset of training or a change in training patterns, such as a change in terrain or intensity, can contribute to stress fractures. Poor footwear and nutritional deficits have been shown to be contributing factors. Biomechanical factors, such as leg-length difference, excessive forefoot (FF) varus, hypermobile flat arches, and high rigid arches will increase the likelihood of injury (Heaslet & Kanda-Mehtani, 2007). As with many injuries, often a combination of factors contribute to stress fractures. A thorough history and examination covering all these areas is important.

95% of all stress fractures occur in the lower extremities, but the distribution of where these occur in the lower extremity varies in the literature. One study showed that tibial stress fractures have the highest rate of occurrence at 35%, while fibular stress fractures follow at 25–30% (Losito, Laird, Alexis, & Mora, 2003). Another study showed that following tibial stress fractures, second metatarsal and third metatarsal were the next most common (Heaslet & Kanda-Mehtani, 2007). It is also shown that more than 18% of all stress fractures in the body occur in the feet (Arnheim & Prentice, 1997).

Tibial stress fracture

Tibial stress fractures are the most common stress fractures seen in the body (Heaslet & Kanda-Mehtani, 2007) and have been shown in at least one study to occur mostly in runners (Heaslet & Kanda-Mehtani, 2007). Most tibial stress fractures are located in the middle third and proximal third of the tibia (Kor, Saltzman, & Wempe, 2003). Stress fractures of the
tibia have been associated with a high arched rigid foot structure (Arnheim & Prentice, 1997; Heaslet & Kanda–Mehtani, 2007).

**Fibular stress fracture**

Fibular stress fractures have been reported as the second most common form of stress fracture, although they are often misdiagnosed. They are often found approximately 8cm proximal to the distal tip of the lateral malleolus (Palamarchuk & Sabo, 1998). A hypermobile overpronated foot structure is the most common mechanical condition seen with fibular stress fractures (Arnheim & Prentice, 1997).

**Navicular stress fracture**

Navicular stress fractures commonly occur in activities with explosive movements. Athletes that participate in activities that involve sprinting or jumping are particularly susceptible. It has also been shown that a short first metatarsal or long second metatarsal can also contribute to navicular stress fractures, possibly due to an increase in impact through the second metatarsal.

Although less common, other bones that can sustain stress fractures are the cuboid, sesamoids and patella.

**Pathology**

While there are a few theories as to why stress fractures occur, it is generally agreed upon that when the occurrence of repetitive stress is greater than the bone’s ability to repair itself, a stress fracture can occur (Heaslet & Kanda–Mehtani, 2007). They can occur as a result of axial compression on the bone or by tension created by the pull of muscles (Arnheim & Prentice, 1997).

Overpronation, high medial arches, limb length discrepancies, structural forefoot varus, short first metatarsal (long second metatarsal), nutritional deficiencies, footwear, and training environments are among the factors that can contribute to stress fractures. If you consider that stress fractures occur as a result of axial compression on a bone, muscle tension at the site of insertion on a bone, or reduced ability for a bone to be able to repair itself, then the mechanics of injury can likely be determined.

Overpronation creates an increase in tension in the muscles of the lower leg and ankle (posterior tibialis, anterior tibialis, and peroneus longus and brevis). This increase in tension will therefore increase the tensile forces at the origin and insertion of these muscles and tendons. The foot’s ability to absorb shock is also limited in a hypermobile overpronated foot. An increase in ground impact may be a contributing factor in metatarsal stress fractures.

Axial compression on a bone will in part be the result of a foot structure that does not effectively absorb shock. An example of a foot type with poor shock absorption is rigid pes cavus. As the foot strikes the ground, the forces from contact will transfer through to the tibia and increase the risk of tibial stress fracture. Another example is a short first metatarsal, which will increase the load on the second metatarsal and increase the compressive forces through the second metatarsal, particularly in the propulsive phase of gait.
Training environments and training surfaces have a great effect on the forces applied to the body while walking, running, or jumping. Harder surfaces will reduce the body's ability to absorb the shock of impact and increase the risk of stress fracture in both the foot and lower leg. Improper footwear, such as shoes that are worn out or that are inappropriate for an individual's foot type and mechanics, will also inhibit the body's ability to absorb shock and/or exacerbate existing mechanical conditions like overpronation.

The risk of stress fracture also increases from a decrease in the body’s ability to repair itself. Individuals with eating disorders, such as bulimia or anorexia nervosa, may not possess the required nutrients for normal bone growth and repair. Bone mineral density and bone composition are factors that contribute to bone strength. The decrease in mineral content in an osteoporotic bone decreases its ability to resist daily load and increases the risk of stress fracture. Remodeling capability in osteoporosis patients is also diminished, again increasing the risk of stress fracture (Kazimoglu et al., 2009). It is theorized that this may partly account for why stress fractures are more commonly seen in women versus men (Heaslet & Kanda-Mehtani, 2007).

Differential Diagnosis
It takes 2–4 weeks for a stress fracture to be seen on an X-ray. As a result, stress fractures are often misdiagnosed. A stress fracture can be misdiagnosed as a soft tissue strain, muscle contusion, or bone contusion. Shin splints, compartment syndrome, and plantar fasciitis are also common misdiagnoses.

Common Testing
Initial diagnosis of a stress fracture can be difficult. Radiographical evidence is delayed in the progression of the condition, and as such, stress fractures can be missed with this testing alone. If a stress fracture is suspected, radiographic examination should be followed up with a bone scan and subsequently an MRI (Helstad et al., 1996; Kor et al., 2003).

Clinically, without the help of a bone scan or MRI, it is difficult to properly screen for a stress fracture. The most common method is to apply percussion or vibration (tuning fork) in the surrounding area. This stimulus irritates the damaged bone and will produce pain at the affected area (Arnheim & Prentice, 1997).

Contraindications
There are no contraindications for stress fractures found in the research that is available.

The Patient Perspective
Individuals with stress fractures rarely report sudden onset of pain, rather a progressive level of discomfort and swelling with a corresponding decrease in level of activity (Heaslet & Kanda-Mehtani, 2007). Many patients will report having tried to exercise or work through the pain and experiencing an increased level of pain as a result (Palamarchuk & Sabo, 1998). Early stages of a stress fracture will produce pain during activity and none at rest. As the injury worsens, pain becomes more constant, even after the activity, and can be worse at night (Arnheim & Prentice, 1997).

Common Treatment
Conservative treatments of stress fractures should start with removing the patient from the activity that contributed to the stress fracture in the first place. After 2–4 weeks, once symptoms have subsided, the patient can resume activity within defined pain limits (Arnheim & Prentice, 1997). Exercise can come from low impact activities such as cycling or swimming (Arnheim & Prentice, 1997). Patients that are having difficulty walking pain free can be placed in a cast with crutches, a walking boot or a brace (Martinez, 2015).

Biomechanical problems in the feet should be corrected (Arnheim & Prentice, 1997). Custom foot orthoses have been shown to reduce the rate of stress fracture injuries (Baxter, Baycroft, & Baxter, 2011; Heaslet & Kanda-Mehtani, 2007) and have been shown to reduce stress fracture symptoms (Heaslet & Kanda-Mehtani, 2007). It has been stated that hypermobile overpronated feet, rigid cavus feet, a long second metatarsal, and structural FF varus can contribute to stress fractures. All these conditions can be treated with custom or over-the-counter foot orthoses. Common orthotic additions to help redistribute forces and provide shock absorption are metatarsal pads or bars, heel wells, cushioned top covers, and cushioned forefoot extensions.

Improper footwear also plays a role in stress fractures (Heaslet & Kanda-Mehtani, 2007). A patient's footwear must be assessed for appropriateness for activity, foot biomechanics, and life span. Shoe modifications can be added to further improve the function of the footwear. Rocker soles, heel and
forefoot wells and metatarsal bars can all redistribute forces, provide shock absorption, and reduce impact of bony structures.

Pedorthic treatment of stress fractures does need to follow the guidelines of best pedorthic practices. Metatarsal pads must be properly located proximal to the metatarsal heads, hindfoot and forefoot posting must compliment the correction needed in a patient’s foot mechanics, and orthotic shell materials must be compatible with a patient’s foot structure. Proper break-in guidelines are to be communicated to the patient, appropriate patient followup is to occur, and orthosis adjustment should performed when necessary.

**Key Considerations**

Age, activity, and medical conditions should be factored in when assessing a patient for lower limb stress fractures. Athletes involved in high impact repetitive activities, military personnel, and osteoporosis patients are the populations most prone to stress fractures (Kazimoglu et al., 2009). Age and diet also affect the bones’ ability to respond to repetitive stress, and will also be factors in stress fracture formation. Training practices and environments, including footwear, also need to be explored and evaluated.

**References**


Fracture: Calcaneus, Fifth Metatarsal Avulsion, and Jones

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Key Messages

• An accurate and well-narrated history is a key factor in determining the best course of treatment for the patient.
• The classification of traumatic fracture versus stress fracture is a critical component in determining the best treatment practice.
• Acute fractures of the calcaneus or fifth metatarsal may initially be masked by secondary symptoms and could be misdiagnosed.
• The primary goal of pedorthic intervention in managing patients who have sustained a calcaneal or fifth metatarsal fracture is to provide functional pain relief.
• The use of custom-made foot orthoses and orthopaedic footwear following a calcaneal or fifth metatarsal fracture has been widely supported in literature.

Keywords
calcaneal fracture, fifth metatarsal avulsion fracture, fracture, Jones fracture, pedorthics

Introduction

The foot serves as the foundational support of the human body. For most, it is the primary means of mobility. When a fracture occurs within the foot it can have a multifaceted effect on a person’s life. A fracture of the foot can significantly limit a person’s ability to ambulate acutely and often chronically (Sanders & Clare, 2010). This can have many implications on their social interactions, employment status, economic state, and general health. Fractures of the foot, particularly of the calcaneus or fifth metatarsal, are notoriously difficult to manage. According to an article by Judd & Kim (2002), foot and ankle fractures are often initially misdiagnosed as sprains or other soft tissue injuries and, therefore, may be mistreated. Furthermore, a person may be unaware of the severity of a traumatic injury and fail to immediately seek the necessary medical attention. Judd & Kim (2002) also report that a delay in appropriate treatment may cause a patient to have a more complex clinical course. While misdiagnoses of foot fractures have been identified in the available literature as one problem area in the management of calcaneal and fifth metatarsal fractures, standards of practice have been a point of contention. There has been much debate over the course of the past few decades in regards to best treatment practices. Many studies have argued that initial surgical management of more complex fractures is critical to a patient’s positive prognosis (Algan, 2011; Sanders & Clare, 2010; Zwitser & Breederveld, 2009). Others suggest that there is minimal difference between the long-term functional outcomes of patients who are initially managed surgically versus conservatively (Khan, Oragui, & Akagha, 2010). With controversy and debate over best treatment practices still existing today, a certified pedorthist’s expertise in providing conservative management options of foot dysfunction can be a particular asset in the overall management of patients who have sustained calcaneal or fifth metatarsal fractures.

Pathology

Fracture is defined as either a complete or incomplete break in the continuity of a bone as a result of the application of excessive force (Canadian Orthopaedic Foundation [COF], 2015a; COF, 2015b). Fractures are clinically classified based on their location, alignment, direction, and articular involvement (Terrell, 2002). They can be further categorized as open or closed; an
open fracture refers to the bone breaking through the skin, creating an open wound, whereas in a closed fracture the bone remains contained within the skin (Scolaro & Mehta, 2010). An avulsion fracture occurs when excessive tension on a tendon or ligament results in a rupture of the tuberosity (Bahel & Yu, 2010).

Calcaneal fractures account for approximately 2% of all fractures and are the most common fracture of the foot (Juliano & Nguyen, 2001; Scolaro & Mehta, 2010; Schepers, Van Lieshout, Van Ginhoven, Heetveld, & Patka, 2008b). Likewise, the fifth metatarsal is the most frequently fractured metatarsal bone (Zwitser & Breederveld, 2010). In their article, Zwitser & Breederveld (2010) state that 45–70% of metatarsal fractures involve the fifth metatarsal.

The epidemiology of calcaneal and fifth metatarsal fractures shows a large bias toward the working-age male demographic. Several studies have shown that men are more likely than women to sustain either a calcaneal or fifth metatarsal fracture (Mitchell, McKinley, & Robinson, 2009; Schepers, Ginai, Van Lieshout, & Patka, 2008a; Smith, Timmons, Lombardi, Mamidi, Matz, Courtney, & Perry, 2006; Zwitser & Breederveld, 2010). The common rationale for this demographical divide has been the primary mechanism of injury. In their study on the incidence of calcaneal fractures, Mitchell et al. (2009) found that males were 2.4 times more likely to sustain a traumatic calcaneal fracture than females, with the primary mechanism of injury being an uncontrolled fall from height. Likewise, Zwitser & Breederveld (2010) noted that young males involved in organized sports were more prone to fractures of the fifth metatarsal secondary to inversion injury. However, it should be noted that the prevalence of both calcaneal and fifth metatarsal fractures in females has been shown to increase with age (Mitchell et al., 2009; Schepers et al., 2008a; Zwitser & Breederveld, 2010). This has primarily been linked to rate of osteoporosis amongst women (Mitchell et al., 2009; Osteoporosis Canada, n.d.).

The etiology of calcaneal and fifth metatarsal fractures can be divided into two categories: traumatic fracture and stress fracture.

Traumatic fractures of the calcaneus:

• Traumatic calcaneal fractures are most often caused by falls from a height in which the subject absorbs the primary force of impact through the heel(s) (Sanders & Clare, 2010). However, motor vehicle accidents and sports injuries account for a smaller portion of reported traumatic calcaneal fractures (Mitchell et al., 2009). These fractures can further be divided into two descriptive classifications; inter-articular and extra-articular fractures (Essex-Lopresti, 1993). Intra-articular fractures are those in which the fracture line extends into the articular surface of the bone, while extra-articular fractures do not extend to the joint surface. The etiological classification of the fracture is generally determined by the magnitude of the force that is applied to the bone (Barei, Bellabarba, Sangeorzan, & Benirschke, 2002). For instance, a high-energy injury is more likely to result in an intra-articular fracture (Sanders & Clare, 2010). The classification of a traumatic calcaneal fracture is a critical component in determining the best acute treatment practice, which may ultimately affect the patient’s long-term prognosis.

Stress fractures of the calcaneus:

• Stress fractures of the calcaneus typically occur secondary to excessive repetitive loading at the heel and are often referred to as overuse injuries (Serrano, Figueiredo & Páscoa Pinheiro, 2016). However, Alexander (1990) notes that regular activities are often enough to cause a stress fracture of the calcaneus in osteoporotic individuals. Stress fractures of the calcaneus can often be misdiagnosed as plantar fasciitis or other forms of soft tissue injury due to their gradual onset and failure to present on plain X-ray prior to an onset of approximately 3–4 weeks (Dec, 2007; Judd & Kim, 2002; Lichniak, 1994).

Fractures of the fifth metatarsal:

• Fractures of the fifth metatarsal primarily occur as a result of excessive inversion of the rearfoot combined with plantarflexion at the ankle (Bahel & Yu, 2010). High-energy rotational forces apply extreme tension to the peroneus brevis tendon, as well as the lateral band of the plantar fascia along its insertion at the proximal fifth metatarsal (Khan et al., 2010). This typically results in one of two types of fracture; Jones or avulsion fracture (Khan et al., 2010). A Jones fracture is a transverse fracture of the fifth metatarsal distal to the tuberosity at the metaphyseal–diaphyseal junction (Algan, 2011; Dec, 2007). This fracture was first documented by Sir Robert Jones in 1902 after personally sustaining a fracture of his fifth metatarsal (Khan et al., 2010). Jones fractures are often susceptible to delayed healing or non-union as the blood supply to the fragmented bone from the proximal nutrient artery can be interrupted by the anatomical location of the fracture (Algan, 2011). Avulsion fractures often
occur at the tuberosity of the fifth metatarsal base (Bahel & Yu, 2010). An avulsion fracture of the fifth metatarsal base occurs secondary to intense contraction of the peroneus brevis and lateral band of the plantar fascia during excessive inversion of the subtalar joint resulting in fragmentation through the tuberosity (Zwitser & Breederveld, 2010).

Failure for fractures of the foot to be apparent on plain X-ray during initial emergency room visits has been a noted problem in the misdiagnosis of both calcaneal and fifth metatarsal fractures (Dec, 2007; Lichniak, 1994). However, improvements in radiographic technology and the use of computed tomography (CT) scanning as well as magnetic resonance imaging (MRI) and nuclear scans have greatly improved accuracy in diagnosing fractures of the foot (Sanders & Clare, 2010). The development and implementation of emergency department clinical assessment guidelines like the Ottawa Ankle Rules (1992) have also improved accurate diagnosis of foot and ankle fractures (Bachmann, Kolb, Koller, Steurer, & ter Riet, 2003; Judd & Kim, 2002). However, despite the best efforts of emergency and/or primary care physicians, acute fractures of the calcaneus or fifth metatarsal continue to present in pedorthic and other foot care centres. In general, the type of acute fracture that is most likely to present in pedorthic clinics is a stress fracture. As previously mentioned, stress fractures of the calcaneus are predominantly linked to excessive repetitive loading (Serrano et al., 2016). These injuries most often present in athletes whose sport involves jumping such as basketball and volleyball, or runners who have increased their intensity in training, like those preparing for a marathon (Dec, 2007). However, patients over the age of 50 are at higher risk of stress fracture due to the increased likelihood of osteoporosis (Alexander, 1990; Osteoporosis Canada, 2011). Likewise, fractures of the fifth metatarsal are often associated with excessive inversion forces (Bahel & Yu, 2010; Zwitser & Breederveld, n.d.). These injuries may not present as the result of one specific incident or onset and therefore, the patient may be directed to a pedorthist for management.

Differential Diagnosis

A differential diagnosis is the process of ruling out alternative possibilities in order to confirm the most likely diagnosis. In the case of an acute or subacute calcaneal stress fracture, the patient may present with symptoms of plantar fasciitis, tarsal tunnel syndrome, Achilles tendinitis, calcaneal neuritis, Sever’s disease, or other forms of soft tissue injury (Dec, 2007). A heel squeeze test in which pain is elicited when the medial and lateral aspects of the calcaneus are compressed between the palms of the practitioner’s hands can differentiate a stress fracture from a soft tissue injury (Alexander, 1990). Suspicion of a calcaneal stress fracture is also suggested when pain is constant regardless of weight-bearing (Alexander, 1990). Fifth metatarsal fractures may initially be overlooked on examination or misdiagnosed as severe ankle sprains (Zwitser & Breederveld, 2010). Marked tenderness to palpation over the base of the fifth metatarsal should raise suspicion of the potential for fracture (Zwitser & Breederveld, 2010). The additional elicitation of pain to axial loading of the fifth metatarsal may differentiate a fracture from peroneal brevis tendinitis (Zwitser & Breederveld, 2010). The Ottawa Ankle Rules were developed in 1992 as a guideline to aid emergency department physicians in determining when an X-ray is clinically indicated (Bachmann et al., 2003). This guideline should also be used as part of the differential diagnosis; however, if practitioners question a previous diagnosis, it is their obligation to refer the patient back to the prescribing physician for review with appropriate clinical recommendations.

Calcaneal fracture, repaired through internal surgical fixation. Photo courtesy Dennis J. Janisse.

Common Testing

As with any clinical evaluation, the pedorthist should begin his/her examination by taking a detailed history. In the case of a previous fracture of either the calcaneus or fifth metatarsal, questions regarding the mechanism of injury, onset, previous treatments, surgical procedures, and present physical limitations should be asked. It is also beneficial to have patients quantify their level of discomfort using the 0–10 measure, as in the Numeric Rating Scale (NRS) (Ware, Epps, Herr, & Packard, 2006). This measurement can serve as a useful basic outcome measure when assessing a patient’s progress on follow-up.
With a detailed history well documented, the clinical evaluation can progress to a physical examination of the patient. Special note should be made of any evidence of swelling (edema), redness (erythema), scarring, bony prominences, or marked tenderness to palpation (Alexander, 1990). When assessing patients with a history of unilateral fracture, it is important to examine both feet and compare the findings to the contralateral side. A thorough assessment of the rearfoot mechanics is especially important in patients who have sustained a calcaneal fracture. Subtalar joint mobility can be greatly reduced by changes in the physiology of the calcaneal and talar articulating surfaces as a result of traumatic intra-articular fracture, post traumatic osteoarthritis, surgical arthrodesis, and internal surgical fixation (Barei et al., 2002; Cailliet, 1983). Secondary soft tissue injury is common with both calcaneal and fifth metatarsal fractures (Dec, 2007; Sanders & Clare, 2010). Therefore, a cursory examination of major tendons and ligaments should also be conducted, including the peroneals, tibialis anterior and posterior, Achilles, deltoid ligament, and the lateral collateral ligaments of the foot/ankle. Notes should be made of any irregularities or elicitation of discomfort with palpation of these regions. Active isometric muscle strengths can also be tested using the Manual Muscle Testing (MMT) scale of 0–5 (Mangusan, 2011; Weber, 1993). This measure, like the NRS, can be used during follow-up to assess a patient’s progress or possible regression.

With the patient in a weight-bearing position, range of motion and muscle strength can once again be affirmed using simple functional tests such as a double-heel raise or subtalar twist test. The subtalar twist test is useful in further evaluating the closed kinetic chain mobility of the subtalar joint. The test is performed by having the patient position their hands over their iliac crests and then rotate their pelvis from side to side, while their feet remain pointed forward. As the pelvis is rotated to the right, the right leg should externally rotate and the right subtalar joint should supinate (Weber, 1993). On the contralateral side, internal rotation of the leg will force pronation (Weber, 1993). The rotation is performed in both the left and right directions to review pronation and supination of each subtalar joint. These movements can be visually assessed and compared for asymmetry. Discomfort while performing this test should also be noted. Functional testing should only be performed as tolerated by the patient.

Observation of a patient’s gait is an important part of the overall examination process. Assessing the many complexities of the gait pattern in a patient who has sustained a fracture of either the calcaneus or the fifth metatarsal can be simplified by breaking the gait cycle down into its basic phases; heel strike, midstance, push-off/propulsion, and swing phase (Weber, 1993). If it is too uncomfortable for a patient who is recovering from a fracture of the calcaneus or fifth metatarsal to walk barefoot, it is beneficial to permit the patient to walk in footwear. This will certainly compromise the pedorthist’s ability to assess the dynamic function of the rearfoot; however, it may provide a truer impression of the patient’s gait pattern.

**Contraindications**

In the management of calcaneal or fifth metatarsal fractures, a pedorthist should not assume a primary care role, especially in the case of acute injury. The pedorthist’s scope of practice is to provide conservative management of the foot and ankle at the direction of physician (College of Pedorthics of Canada, 2011). Pedorthists are not qualified to diagnose; however, it is their responsibility to report clinical findings to referring physicians and advise patients when it may be necessary to follow up with their physicians. More specifically related to the subacute or long-term treatment of these injuries, it would be contraindicated to attempt to functionally correct a joint alignment that has been surgically fused or that is limited by bony deformity. Doing so may cause iatrogenic injury to the patient (Michaud, 1997). Conversely, several studies suggest that early mobilization of joints provides more favourable outcomes (Barei et al., 2002; Schepers et al., 2008a). Therefore, patients should be encouraged to perform both passive and active range of motion exercises in order to maintain joint mobility as applicable and within their own comfort levels. Referring the patient to a physiotherapist or other allied health care provider may be necessary for more specialized treatment that would extend beyond the scope of pedorthic practice. The best approach is always the safe approach.

**The Patient Perspective**

The patient might describe:

- Onset of pain within the past 4–6 weeks
- Localized sharp pain at onset of injury
- Incident of acute injury (e.g., fall from a height, inversion ankle sprain)
- Pain following sudden increase in activity level
of non-union and potential for recurrence (Algan, 2011; Khan et al., 2010). Therefore, these injuries often require long-term management. The use of a custom-made foot orthosis, either rigid or semi-rigid, with a rearfoot to forefoot valgus post is indicated to reduce excessive rearfoot varus/subtalar inversion that permits increased loading at the fifth metatarsal (Algan, 2011). However, adequate eversion of the subtalar joint must be available in order to appropriately transfer lateral forces on the foot. If sufficient subtalar eversion is not permissible then an accommodative approach to custom-made foot orthoses is indicated.

Due to the great variability in the acute management of calcaneal and fifth metatarsal fractures, both surgically and conservatively, and the ongoing controversy about best clinical practice, it is difficult to determine whether a functionally corrective or accommodative pedorthic approach is most effective (Khan et al., 2010). The key determinants to be used in making this decision should be based on the patient’s tolerance to joint manipulation, their activity level, body weight, and footwear selection (Weber, 1993). Material selection for foot orthoses should be left to the discretion of the pedorthist, as personal preference and experience in working with certain materials differs between practitioners. In general, it is widely believed that functional foot orthoses are typically fabricated using rigid materials like polypropylene, Subortholene, copolymer, or composites (Michaud, 1997; Weber, 1993), while accommodative or soft foot orthoses are constructed from Plastazote, ethylene vinyl acetate (EVA), and other types of Thermofoam (Olsen, 1996; Valmassy, 1989). Lastly, well-made custom foot orthoses are only as effective as the footwear in which they are worn (Michaud, 1997). It is important to consider the patient’s footwear as an integral component in the overall pedorthic management. Extra depth and width orthopaedic footwear provide more adequate internal volume to appropriately accommodate custom-made foot orthoses. Orthopaedic footwear can also provide additional stability to the foot and ankle. Footwear modifications such as buttresses/hares, rocker soles, or rigid shanks may also be required in the ongoing management of more complex calcaneal or fifth metatarsal fractures (Michaud, 1997; Zwitser & Breederveld, 2010). A custom rocker–sole modification helps to more evenly transfer plantar pressures over the foot as it progresses through the gait cycle, thereby offloading sensitive areas (Long, Klein, Sirotta, Wertsch, Janisse, & Harris, 2004). The incorporation of a rigid shank within the rocker sole ensures that the shoe does not excessively flex at the fulcrum of the rocker.

Common Treatment

The primary goal of pedorthic intervention, in managing patients who have sustained calcaneal or fifth metatarsal fractures, is to provide pain relief through improving biomechanical alignment, accommodating fixed deformities, improving balance, and optimizing foot function (Colaizzi, 2002; Wu, 1990). When managing patients who have recovered from or are in the process of recovering from calcaneal or fifth metatarsal fractures, it is essential that a thorough and complete biomechanical and gait assessment has been performed to identify any functional limitations or restrictions within the foot. The findings of the clinical assessment will form the basis of the treatment plan that best suits a particular patient’s needs. It is important to outline treatment goals with patients and discuss their expectations prior to initiating treatment. This can help to prevent misunderstanding regarding the purpose and intention of the purposed treatment.

As each patient and injury is different, there is no one method of pedorthic treatment that can be universally employed; however, the use of custom-made foot orthoses and stiff rocker-soled orthopaedic footwear following either a calcaneal or fifth metatarsal fracture has been widely supported in available literature (Algan, 2011; Bahel & Yu, 2010; Barei et al., 2002). When determining the most appropriate course of management, it is important to consider the patient’s subjective symptoms and primary complaint as they relate to the objective clinical findings (Neale & Adams, 1989). For example, a patient presenting with a history of a traumatic calcaneal fracture, a chief complaint of ongoing rearfoot pain, and clinical findings significant for discomfort with subtalar joint inversion/eversion would be best managed by limiting subtalar motion with a functional rigid foot orthosis in an effort to minimize discomfort (Weber, 1993); however, if the patient has undergone an arthrodesis of the subtalar joint, then the rearfoot alignment cannot be functionally manipulated and, therefore, an accommodative approach that focuses on protection of the sensitive calcaneal region and redistribution of plantar pressures is most appropriate (Michaud, 1997).

Fractures of the fifth metatarsal, particularly Jones fractures, have been noted to have a high incidence of non-union and potential for recurrence (Algan, 2011; Khan et al., 2010). Therefore, these injuries often require long-term management. The use of a custom-made foot orthosis, either rigid or semi-rigid, with a rearfoot to forefoot valgus post is indicated to reduce excessive rearfoot varus/subtalar inversion that permits increased loading at the fifth metatarsal (Algan, 2011). However, adequate eversion of the subtalar joint must be available in order to appropriately transfer lateral forces on the foot. If sufficient subtalar eversion is not permissible then an accommodative approach to custom-made foot orthoses is indicated.

Due to the great variability in the acute management of calcaneal and fifth metatarsal fractures, both surgically and conservatively, and the ongoing controversy about best clinical practice, it is difficult to determine whether a functionally corrective or accommodative pedorthic approach is most effective (Khan et al., 2010). The key determinants to be used in making this decision should be based on the patient’s tolerance to joint manipulation, their activity level, body weight, and footwear selection (Weber, 1993). Material selection for foot orthoses should be left to the discretion of the pedorthist, as personal preference and experience in working with certain materials differs between practitioners. In general, it is widely believed that functional foot orthoses are typically fabricated using rigid materials like polypropylene, Subortholene, copolymer, or composites (Michaud, 1997; Weber, 1993), while accommodative or soft foot orthoses are constructed from Plastazote, ethylene vinyl acetate (EVA), and other types of Thermofoam (Olsen, 1996; Valmassy, 1989). Lastly, well-made custom foot orthoses are only as effective as the footwear in which they are worn (Michaud, 1997). It is important to consider the patient’s footwear as an integral component in the overall pedorthic management. Extra depth and width orthopaedic footwear provide more adequate internal volume to appropriately accommodate custom-made foot orthoses. Orthopaedic footwear can also provide additional stability to the foot and ankle. Footwear modifications such as buttresses/hares, rocker soles, or rigid shanks may also be required in the ongoing management of more complex calcaneal or fifth metatarsal fractures (Michaud, 1997; Zwitser & Breederveld, 2010). A custom rocker–sole modification helps to more evenly transfer plantar pressures over the foot as it progresses through the gait cycle, thereby offloading sensitive areas (Long, Klein, Sirotta, Wertsch, Janisse, & Harris, 2004). The incorporation of a rigid shank within the rocker sole ensures that the shoe does not excessively flex at the fulcrum of the rocker.
**Key Considerations**

There are several factors to consider when assessing and treating a patient who presents with a history of a either a calcaneal or fifth metatarsal fracture. Key considerations are:

- **Specific injury and acute management** (i.e., location of fracture, surgical interventions, immobilization, rehabilitative therapies, etc.)
- **The onset of injury** (how long ago did the fracture occur? how well has the fracture healed?)
- **The patient’s age, activity level, occupation, and preferred or required footwear** are factors that will dictate or determine the design of a custom-made foot orthoses and/or orthopaedic footwear selected
- **Normal and abnormal ranges of motion** (how can the foot structure be manipulated to relieve stress and force?)
- **The patient’s preference as to treatment options and expectations of treatment outcomes**

**References**


Lisfranc Injury

Patrick Bergevin, HBSc (Kin), C. Ped Tech (C), C. Ped (C)

Key Messages

- Signs and symptoms include inability to bear weight, redness and edema at the dorsum of the foot, as well as dorsally prominent medial midtarsals.
- Common causes of injury include stepping in a hole, motor vehicle accidents, uneven terrain, or falls from heights with construction workers.

Keywords

Lisfranc fracture dislocations, midfoot fusion, midfoot hyperflexion, tarsometatarsal joint sprains

Introduction

Lisfranc injuries occur along the tarsometatarsal (TMT) joint complex, which indirectly bisects the midfoot in the frontal plane, and can involve one or many of the five tarsometatarsal joints (Trevino, 2009). The mechanism of injury can be direct (direct trauma such as motor vehicle or crush injury), or indirect (axial loading of a plantarflexed ankle and forefoot, with toes extended) (Aronow, 2006). An indirect injury can be summarized as a convex, or dorsal, bending of the midfoot, with the forefoot and rearfoot relatively plantarflexing. Specifically, the metatarsal bases dislocate dorsally or transversely from the midtarsals.

Often referred to as a Lisfranc fracture dislocation, it presents in varying degrees, and not always involving fractures. The amount of axial load applied to the foot can proportionally cause a mild sprain of the soft tissue, or complete disruption of the TMT joint complex, with metatarsal fractures (Burroughs, Reimer, & Fields, 1998; Trevino, 2009). Unlike the plantar surface of the foot which has extensive musculature and fasciae to prevent the concave or plantar collapsing of the foot, there is limited tissue on the dorsal aspect to prevent convex, or dorsal dislocation. Specifically, the number and sizes of the dorsal ligaments versus plantar ligaments are much less (Raikin, Elias, Dheer, Besser, Morrison, & Zoga, 2009). Raikin et al. (2009) also state that the plantar ligaments are the primary stabilizing ligaments of the midfoot. Trevino (2009) says that the Lisfranc ligament is the only structure to offer ligamentous support between the first and second rays at the midfoot level. There are no transverse or interosseous ligaments between the first and second metatarsal bases, and therefore the strength of the second TMT joint (first Lisfranc joint) comes from its keystone type morphology (Raikin et al., 2009).

Aronow (2006) offers a good summary of the support structure of the midfoot: “The TMT joints consist of the five metatarsals and their articulation with the corresponding cuneiforms and cuboid. TMT joint stability comes from a combination of bone morphology, ligaments, and soft tissue support. The second metatarsal base forms a keystone with all three cuneiforms. The metatarsal bases, cuneiforms, and cuboid are wedge-shaped bones that form a stable “Roman arch” configuration. Ligaments cross the TMT, naviculocuneiform, and intercuneiform joints. Ligaments connect the second through fifth metatarsal bases, but not the first and second. The strong interosseous Lisfranc ligament connects the medial cuneiform to the medial second metatarsal base. There is a plantar ligament from the medial cuneiform to the plantar aspect of the second and third metatarsal bases and a weaker dorsal ligament from the medial cuneiform to the medial second metatarsal base. Additional plantar arch support comes from the intrinsic foot muscles; the plantar fascia; and the posterior tibial, anterior tibial, and peroneus longus tendons. The first, second, and third TMT joints undergo slight dorsiflexion, plantarflexion, supination, and pronation motion that helps to absorb shock. They principally function as a rigid lever that connects the hindfoot to the forefoot. The fourth and fifth TMT joints have more motion which accommodates the forefoot on uneven terrain.”

Englanoff et al., as well as Mantas et al. (as cited in Burroughs et al., 1998), report the incidence of
Lisfranc fracture dislocations to be at a rate of one in 55,000 per year.

Richter, Wippermann, Krettek, Schratt, Hufner, & Therman (2001) report Lisfranc fracture dislocations account for 0.2% to 0.8% of all fractures. Although not as common as ankle (talocrural) joint sprains, Lisfranc cases may be seen more often in clinics receiving many referrals from orthopaedic surgeons, motor vehicle accident specialists, athletes, or workers’ compensation.

Pathology

The injury was identified by Jacques Lisfranc de St. Martin (1790–1847), a French army field surgeon and gynecologist. He noted the injury occurred when soldiers fell from their horses and the foot did not release from the stirrup (Burroughs et al., 1998; Cluett, 2005). As the TMT joints are plane joints, they allow only for gliding movement so when excessive gliding (or rotation) is applied to the joint, it can damage the surrounding soft tissue.

Burroughs et al. (1998) state that TMT joints 1 through 5 should be referred to as the Lisfranc joint complex, while the Lisfranc joint refers to the first and second only. Conversely, Raikin et al. (2009) refer to the second TMT joint as the first Lisfranc joint, excluding the first TMT joint. The anatomy of the second tarsometatarsal joint is multi-faceted, and is described as a keystone; it is therefore difficult to truly dislocate the medial midtarsal joints as they are more secure than the lesser joints. Wheeless (2011) reports true severe sprains of the Lisfranc joint to be somewhat uncommon because the base of the second metatarsal is recessed between the medial and lateral cuneiforms; it is therefore difficult to dislocate this joint without fracturing the second metatarsal base. This same author also reports that when true Lisfranc sprains occur (with disruption of the Lisfranc ligament), the injury will most often be due to high-energy trauma (i.e., a motor vehicle accident) rather than from sporting events.

Modern day causes (footEducation, 2011):

- A twisting injury such as in sports (football)
- Jamming the foot on the brakes as in a head-on vehicle collision
- Loading through the heel leading to an increased force through the midfoot (as in stepping in a small hole)

Classifications

Authors report the importance of proper and early diagnosis of the injury, placing great emphasis on the proper identification of the severity of the injury (Aronow, 2006; Raikin et al., 2009). The diagnosis must be made whether the TMT joints are non-displaced, displaced, or Lisfranc fracture dislocations are present. Lisfranc fracture dislocations are treated operatively; the challenge is determining the level of instability within the first two categories. Within these two categories, it is the level of instability that dictates the need for surgical intervention, not simply the level of displacement (Aronow, 2006; Raikin et al., 2009). Some non-displaced injuries can be completely unstable. Raikin et al. (2009) provide a very clear diagram (algorithm) to depict the decision process whether treatment should be done conservatively or operatively.

The mechanism of injury can also be described as being of a direct or indirect cause (Aronow, 2006; Lattermann, Goldstein, Wukich, Lee, Bach, 2007). Lattermann et al. (2007) classifies the causes as low or high energy, associating low-velocity injuries to sports, and high velocity injuries to motor vehicle accidents. They further divide low-velocity injuries into forced hyperplantarflexion of the midfoot in athletes using foot straps (surfers, windsurfers, and equestrians, for example). These authors further refer to Shapiro’s classification who has identified the high frequency of this injury in football players where the forefoot is trapped between the ground and a force applied at the heel, such as a player falling on another player’s heel (especially common in linemen).

Richter et al. (2001) presents much more detailed charting of the pathology using the Quenu and Kuss Lisfranc fracture dislocation chart. This classification method specifically described the pattern of dislocation with three sub-classifications: homo–lateral; isolated; and divergent. Soft tissue and fracture severity classification can further be described using the extensive Tscherne and Oestern chart. This classification system identifies specific qualities of the skin, such as open or closed fracture, extent of the soft tissue damage, fracture severity, and the presence or absence of contamination.

Nunley (as cited in The Sports Physiotherapist, 2011) offers a three-stage classification chart, which is based on the findings of the severity of the ligament damage, the anteroposterior and lateral X-ray findings, as well as bone density results.
Raikin et al. (2009) state: “Occasionally, despite an appropriate mechanism of injury and clinical findings suggestive of local tenderness over the articulation and inability to bear weight on the foot, plain radiographs are not diagnostic. In these cases, manual stress radiographic evaluation with the patient under anesthesia has been shown to be the most accurate determinant of midfoot stability.”

Lattermann et al. (2007) recommend the following for athletes:

- Initial treatment: icing and elevating, no weight-bearing.
- Patient to visit a physician to diagnose severity, and the possibility of compartment syndrome.
- Non-operative treatment (Stage 1 Nunley classification): non-weight-bearing fiberglass cast for six weeks. If there is no tenderness at palpation after removing the cast, custom foot orthoses should be manufactured. If tenderness is present, these authors recommend the use of a custom-made ankle-foot orthosis for an additional four weeks.
- They caution that Nunley Stage 2 and 3 sprains lead to instability if treated conservatively, and an operative treatment is recommended.
- When corrected operatively (midfoot fusions either percutaneously or by open reduction), the authors recommend post-operative treatment using a non-weight-bearing cast or boot for a minimum of eight weeks.

After this, partial weight-bearing is suggested for weeks 8–12. The patient can move to full weight-bearing after three months with the use of custom foot orthoses. Hardware removal is advised 12–16 weeks post-fixation.

Aronow (2006) recommends (non-operative):

- Immobilization in a cast or cam walker may be particularly helpful to promote ligament healing in patients whose injuries are only a few weeks or months old.
• An insole orthosis or University of California Biomechanical Laboratory (UCBL) brace can support the arch, accommodate a fixed deformity, limit subluxation that is due to ligamentous instability, and decrease painful motion of arthritic joints.

• A shoe with a stiff rocker sole may aid ambulation, as would a cane.

Complications

• Burroughs et al. (1998) suggests that a post-traumatic arthrosis is the most common complication of a Lisfranc joint injury. He further reports the complications are directly related to the degree of comminution of the articular surface in the joint.

• Wheeless (2011) reports that possibly up to 50% of cases result in arthritis or pes planovalgus deformities.

• Several authors report compartment syndrome to be a very common sequela, the main cause being disruption of the dorsalis pedis neurovascular complex (Cluett, 2005; Teng, Pinzur, Lomasney, Mahoney, & Havey, 2002). Lattermann et al. (2007) write: “Particularly after high energy trauma the foot can show massive swelling that should alert the treating physician or athletic trainer to a possible compartment syndrome. Diagnosis of a foot compartment syndrome is difficult and should be left to an expert. The usual clinical tell–tale sign of a compartment syndrome: ‘pain with passive stretch’, is not accurate. Fullness of compartments is difficult to assess due to the inaccessibility of the actual muscular compartments. If neurovascular symptoms are described, the compartment syndrome is already established.”

• Teng et al. (2002) say that even in cases where the force plate scan is normal and radiographs look good, poor patient outcomes are quite common.

Differential Diagnosis

Geerling et al. (as cited in Burroughs et al., 1998) say that even today, these injuries are frequently missed because they often occur in patients with multiple traumatic injuries, but low–energy trauma to the foot might also lead to difficulties in diagnosis due to a heterogeneous clinical appearance.

Englanoff et al., as well as Trevino et al. (as cited in Burroughs et al., 1998), report that as many as 20% of Lisfranc joint injuries are missed on initial anteroposterior and oblique radiographs. Richter et al. (2001) states the same percentage, but cites polytrauma as the reason for misdiagnosis.

Lattermann et al. (2007) state that high–velocity injuries (as in motor vehicle accidents) are much easier to diagnose due to the evident physical symptoms such as redness, swelling or ecchymosis, or midfoot instability, where the low–velocity injuries might be subtle and hard to detect due to the lack of external evidence (using the Nunley description of low and high velocity).

Patel, Rao, Nawoczenski, Flemister, DiGiovanni, & Baumhauer (2010) note that post–traumatic midfoot injuries are the leading cause of midfoot arthritis.

Common Testing

• Palpation of dorsal and plantar midtarsal tarso–metatarsal joints.

• Inversion/eversion of forefoot, while securing the rearfoot. The Sport Physiotherapist (2011) recommends: “Passive Accessory Motion: The stability of the “Lisfranc row” i.e., the first to fifth TMT joints can be assessed by passive pronation and supination of the forefoot. Additionally the Lisfranc joint can be stressed via passive dorsiflexion and abduction of the forefoot. Pain with these movements should increase your clinical suspicion”.

• Burroughs et al. (1998) recommend that “palpation of the foot begin distally and continue proximally to each tarsometatarsal articulation. Tenderness along the tarsometatarsal joints supports the diagnosis of a midfoot sprain with the potential for segmental instability. Pain can localize to the medial or lateral aspect of the foot at the tarsometatarsal region on direct palpation, or it can be produced by abduction and pronation of the forefoot while the hindfoot is held fixed. Another diagnostic clue is the patient’s inability to bear weight while standing on tiptoe.”

Contraindications

• Weight–bearing too early

• Barefoot walking in early stages or recovery

• Metatarsal pads in acute cases may not be tolerated

• From a surgeon’s perspective, disrupted skin and excessive swelling are relative contraindications for open reduction internal fixation (ORIF) (Wheeless, 2011)
The Patient Perspective

Acute:
- Pain, redness, and swelling at dorsum
- Ecchymosis (blood pooling) that can present as far back as at the calcaneus or as far forward as the toes
- Cannot tolerate forefoot inversion/eversion against the rearfoot
- Inability to bear weight

Stabilized:
- Inability to bear weight, antalgic gait

Post:
- Permanent dorsal exostosis. Burroughs et al. (1998) describes this as a “step-off”, where a prominence is visible on the dorsal aspect of the foot as the dorsal surface of the proximal second metatarsal is higher than the dorsal surface of the middle cuneiform
- At risk for midfoot collapse, especially when Chopart joint complex is involved (Richter et al., 2001)
- Compartment syndrome

Common Treatment

Most researchers agree that stable injuries (2mm of diastasis or less) do well with non-operative treatment (Burroughs et al., 1998; The Sports Physiotherapist, 2011).

Pedorthic treatment recommendations:

Acute care:
- Non-weight-bearing until condition is diagnosed and classified (if the pedorthist is the first professional the patient sees)
- Non-weight-bearing short leg cast or walking boot for six weeks (Lattermann et al., 2007)
- “The patients are still often symptomatic with pain and swelling 8–12 weeks (or even more) after their injury” (footEducation, 2011)
- Walking boot with off-the-shelf or direct-moulded footbed to support midfoot
- In lieu of or in conjunction with the walking boot, a rigid graphite plate can be added to the patient’s shoe to limit distal metatarsal movement. An aggressive rocker sole modification should be used
- Bullock-Saxton, Janda, & Bullock report (as cited in Wadsworth & Eadie, 2005) that proprioception should be re-trained by using wobble boards, Swiss balls, or balance shoes

Stabilized condition:
- Custom, corrective foot orthoses (intrinsic or extrinsic posting). These devices are advised for both stable or surgically-repaired injuries. Metatarsal pads also help support the distal metatarsals, but may not be tolerated in more acute cases
- Wadsworth & Eadie (2005) specifically used custom-made orthoses with standard 4 degrees of medial posting, 15-degree medial heel skives, as well as metatarsal pads for his study
- Taping of the plantar portion of the foot to support the midfoot and stabilize the forefoot. Wadsworth & Eadie (2005) state that the main goals of taping as being increased plantarfexion of first ray to stabilize forefoot during push-off, to promote first MPJ extension and to provide support and shortening of the longitudinal arch of the foot
- Icing: 10–15 minutes to area, especially at night
- Avoid barefoot walking; moulded cork footbed type sandals should be worn indoors
- If footwear causes dorsal midfoot discomfort, “skip lacing” (not crisscrossing at the dorsal prominence) should be used on the footwear.
- Physiotherapy for gait and proprioceptive retraining (Wadsworth & Eadie, 2005)

Key Considerations

In a study of 93 subjects by Richter et al. (2001), it is reported that men were twice as often affected, there were no significant differences between the number of right and left injuries, and only 8% of the study group presented with bilateral injuries. The mean age at time of injury was 28 years (16–78) and the patients were primarily injured in traffic accidents.

As this injury, in its mild form, can be assumed to be a foot “sprain”, mild cases may present clinically without proper examination from an appropriate medical professional. In these cases, the certified pedorthist must use basic testing to identify the possibility of this injury, and refer them to the appropriate medical professionals for a diagnosis.

Richter et al. (2001) conclude that the treatment of such joint dislocation or fracture dislocations presents problems in trauma care, and influences the functional outcome of the entire foot in the mid- and long-term follow-up. He also reports that in particular, the Chopart–Lisfranc fracture dislocation results in a high degree of residual impairment.
References


Lisfranc Injury
Posterior Tibial Tendon Dysfunction

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Key Messages

- Posterior tibial tendon dysfunction (PTTD) can cause pain in the medial rearfoot to midfoot (distal to the medial ankle) as well as the lateral rearfoot in later stages.
- PTTD is a manageable condition in the early stages. It is imperative that patients complaining of loss of active ankle inversion function are examined appropriately and screened for PTTD.
- A reduced arch height may be a predisposing factor in the later stages of PTTD, but in stage 1 an average arch height may be more expected, therefore early prevention is paramount (Rabbito, Pohl, Humble, & Ferber, 2011).
- An orthosis (AFO or FO) in conjunction with stretching, strengthening or loading exercises have shown to be successful in pain management and to restore function in those with stage I and II PTTD.

Keywords

heel raise test, ligament dysfunction (especially the spring ligament), medial longitudinal arch, midfoot, rearfoot, talus tilt, tendinopathy

Introduction

Posterior Tibial Tendon Dysfunction (PTTD) is a foot condition commonly seen in middle aged adults. The reported prevalence of PTTD in elderly patients is about 10% and is more commonly seen in middle aged women (Edwards, Jack & Singh, 2008). Whereas stage 1 PTTD can be found in younger, active adults, especially runners (Rabbito et al., 2011). The etiology of PTTD is not entirely clear in the research; however, some believe it is caused by both intrinsic and extrinsic factors, and there are 3 theories, including mechanical, vascular, and neural. These can include: tendon load (due to excessive rearfoot eversion), degenerative changes (due to age or mechanical strain), inflammatory response, systemic disease (diabetes, rheumatoid arthritis), and trauma (Beeson & Kumar Varma, 2011). PTTD can be classified into four stages that aid in the early diagnosis and treatment of this condition. Without early diagnosis PTTD can go from an average arch height and weakness of the posterior tibialis muscle, to complete rupture of the tendon, to mid-tarsal osteoarthritis and the need for surgical intervention (Balen & Helms, 2001; Edwards et al., 2008; Kohls-Gatzoulis, Woods, Angel, & Singh, 2009).
load on the tendon, potentially reducing the risk of progression of PTTD when caught and treated early.

The effectiveness of foot orthoses in PDDT is not conclusive due to a limited number of investigations. A study by (Houck et al., 2015) stated that above and below the ankle orthosis prescriptions are the most recommended non-surgical treatment for PTTD. The issues they noted are whether some types of orthoses are more effective than others and whether adding a stretching and/or strengthening routine to the orthosis prescription would be beneficial. Houck et al.’s (2015) study suggests there is weak evidence that augmenting the orthosis with stretching and strengthening was beneficial in its effects on pain and function in the participants.

Pathoanatomy

The tibialis posterior muscle is part of the deep posterior compartment of the lower leg. It is the largest muscle of this group, which also includes flexor digitorum longus (FDL) and flexor hallucis longus (FHL). It originates from the posterior interosseous membrane and the proximal 2/3 of the posterior tibia and fibula. It forms a tendinous structure in the distal third of the calf, passes posterior and inferior to the medial malleolus, and inserts into many locations of the plantar foot: the navicular tuberosity, the plantar surface of the three cuneiforms, and the base of the second to fourth metatarsals (Edwards et al., 2008). Edwards et al. give a possible explanation behind the degeneration of this tissue: “Proximally the tendon is supplied by branches of the posterior tibial artery, which continue distally within the tendon by branches from the dorsalis pedis. Four centimeters from its insertion there is a watershed in the vascular supply. It is thought that the acute change in direction of the tendon as it hugs the medial malleolus causes a zone of relative hypovascularity rendering the tendon susceptible to degenerative change” (Edwards et al., 2008).

The role of the tibialis posterior tendon is to support and stabilize the medial longitudinal arch. It elevates the arch by inverting and plantar flexing the ankle. During gait, its role is to resist ankle eversion by eccentrically contracting, and then during heel lift concentrically contracting to resupinate (invert the ankle) in order to create a rigid lever for strong propulsion. The tibialis posterior is the largest and strongest muscle of the deep posterior compartment and has the largest inversion moment arm. The action of the tibialis posterior is important to the proper function of the foot. Inversion of the rearfoot begins the process of creating a rigid midfoot and rearfoot by locking the mid-tarsal joints, inverting the subtalar joint, and stabilizing the rearfoot. The tibialis posterior muscle is active from mid- to terminal stance (Marks, Long, Ness, Khazzam, & Harris, 2009) and during the propulsion phase of gait the foot acts as a rigid lever, allowing the gastrocnemius/soleus complex to efficiently plantar flex the ankle. Weakness or lengthening of posterior tibialis muscle-tendon unit may compromise the locking of the mid-tarsal joints.

Pathology of the tibialis posterior tendon may be the result of the difference in size and moment arms of the other muscles in this compartment. The FDL and FHL may not be able to compensate for the loss of function of the posterior tibialis muscle/tendon, causing a weakness of the entire compartment and leading to PTTD (Neville & Houck, 2010). Rabbito et al. (2011) found that rearfoot kinematics during gait were a distinguishing factor between the participants with stage 1 PTTD and the control group. They had greater rearfoot eversion and greater peak eversion throughout stance as well as approximately 4 degrees less inversion at heel strike, and reached 92% of their maximal rearfoot eversion range of motion vs. 60% in the control group. Due to these altered mechanics, Ferber et al. speculate that this could not only mean
pathology to the tibialis posterior tendon but also damage to supporting ligamentous structure, leading to the progressive nature of the condition (Rabbito et al., 2011).

Injury to the tibialis posterior tendon is a continuum that starts at paratendinitis and may end at a complete rupture of the tendon (Kong & van der Vliet, 2008). Over time, these forces may cause adult acquired flatfoot deformity (AAFD), as the plantar ligaments of the foot are repeatedly subjected to this increased stress (Balen & Helms, 2001; Edwards et al., 2008; Kohls-Gatzoulis et al., 2009). Williams, Evans, & Platt (2014) have looked at whether ligaments are causative in PTTD and AAFD, and note that failure of the ligaments to support the MLA can occur concurrently, which can continue the progression of this condition. The primary ligaments involved include: spring, superficial deltoid, plantar metatarsal, and naviculocuneiform ligaments. Especially in the later stages of PTTD, the tibialis posterior tendon cannot be at total fault for the resulting deformity without ligament attenuation occurring as well, especially in the spring ligament. Williams, Evans, and Platt (2014) were unable to conclusively say which came first, spring ligament damage or posterior tibial tendon damage; however, they were able to conclude that the spring ligament is an important part to the puzzle of PTTD. Balen & Helms (2001) also concur that “this progression results from failure of the other foot structures that play a role in maintaining the arch. Three of these structures are: the spring ligament, ligaments of the sinus tarsi, and the plantar fascia”. Secondary injuries to PTTD can and will occur due to the increased load on these supporting structures. The above noted structures will be put under undue stress in the absence of a functioning posterior tibial tendon. Time will cause inflammation and degeneration to the spring ligament, sinus tarsi ligaments, and plantar fascia, which allows further progression of PTTD (Kong & van der Vliet, 2008).

There are four classifications of this condition that Johnson and Strom developed, with Myerson later modifying by adding the fourth stage (Edwards et al., 2008; Kohls-Gatzoulis et al., 2009; Kong & van der Vliet, 2008; Kulig, Pomrantz, Burnfield, Reischl, Mais-Requejo, Thordarson, & Smith, 2006; Marks et al., 2009; Neville, Flemister, Tome, & Houck, 2007; Ritchie, 2007):

- **Stage I** – mild weakness of the tibialis posterior tendon; no deformity; medial longitudinal arch is maintained; normal tendon length; inflammation of the tibialis posterior tendon or tendon sheath; able to perform both double and single heel raises but may have some weakness; pain medially along the tendon; negative “too many toes sign”; no valgus deformity or arthritis.

- **Stage II** – tendon is lengthened with degeneration and becoming dysfunctional; varying degrees of flexible rearfoot eversion (or valgus) and forefoot abduction; an acquired flat foot develops; inability to perform a single heel raise test although may be able to perform a double heel raise; pain is medial along the tendon, laterally around the calcaneus (inferior to lateral malleolus) or both; positive “too many toes sign”; no rigid valgus deformity or arthritis.

- **Stage III** – the stage II deformity progresses into a fixed deformity; the rearfoot valgus cannot be passively reduced; marked degeneration and lengthening of the tendon; cannot invert the heel when performing a double heel raise; pain along the tendon track on medial ankle, laterally around the calcaneus (inferior to lateral malleolus), or both; positive “too many toes sign”; no valgus deformity or arthritis.

- **Stage IV** – same as stage III but with arthritic changes in the ankle; additional deltoid ligament incompetence resulting in a valgus tilt of the talus leading to lateral tibiotalar degeneration; arthritis of the ankle is present.

Kulig et al. (2006) describe an addition to the above table with Stage IIa and Stage IIb as follows:
• **Stage IIa** – the foot is flexible, and the tendon is functionally impaired

• **Stage IIb** – the tendon is incompetent or possibly ruptured

### Differential Diagnosis

Misdiagnosis can occur if the patient’s complaints do not immediately match the signs and symptoms of PTTD. Practitioners must be sure to eliminate the following conditions that may cause pain in the medial foot, deformity or loss of function:

- talonavicular, naviculocuneiform, or cuneiform–first metatarsal joint disease
- deltoid ligament rupture
- Charcot arthropathy
- neuromuscular disease
- spring ligament injury (Alvarez, Marini, Schmitt, & Saltzman, 2006)

In addition, Edwards *et al.* state “One must always consider tibialis posterior tendon dysfunction in middle-aged or elderly patients with lateral hindfoot pain and not forget that other foot pathologies may initiate medical attendance, and incorrect initial diagnosis of hallux valgus, hallux rigidus, and metatarsalgia may be the initial diagnosis in primary care.” It is important to gain an understanding of PTTD to prevent misdiagnoses of this condition. While awareness has improved, the diagnosis is often missed, leading to further deterioration of this condition. Edwards *et al.* report, “As prompt diagnosis and early treatment can prevent progression, delay can result in progression of the condition and a poor outcome.” They also state that the patient may not complain of a change in their foot shape, but are more likely to present with pain and swelling around the medial rearfoot (posterior and distal to the medial malleolus), lateral ankle pain, or a decreased ability to walk (Edwards *et al.* , 2008).

### Common Testing

History and examination are the primary means to diagnosing PTTD; however, radiographs, magnetic resonance imaging (MRI), or ultrasound may help the practitioner assess the stage and damage of PTTD (Kohls–Gatzoulis *et al.*, 2008). MRI and radiographs show bony changes, especially arthritis and/or changes of the navicular. An MRI is considered the “gold standard” for assessing PTTD. As Kong & van der Vliet explain, “Bony irregularity and hypertrophic change may be seen at the navicular attachment of the tibialis posterior tendon, suggesting an enthesopathy. A tibial spur seen adjacent to the tibialis posterior tendon in the retromalleolar groove is also a secondary sign of longstanding tibialis posterior tendinopathy.” Ultrasound is another useful method for assessing the tibialis posterior tendon and other relevant soft tissue structures. Early detection and diagnosis may prevent deformity from PTTD and resultant surgery (Kong & van der Vliet, 2008).

Ritchie describes ways to evaluate common features of an adult acquired flatfoot deformity that may help in assessing the level of deformity. The tests are as follows (Ritchie, 2007, p.633–640):

- **Too–many–toes sign** – abduction of the forefoot (more than two lesser digits are visible when looking from behind), which is an indication of transverse plane subluxation of the midtarsal joint, occurring worse on one foot over the other.

- **First metatarsal rise sign** – supination of the foot by means of passive inversion of the heel will cause the first metatarsal to rise off the floor when the posterior tibial tendon and the spring ligament are ruptured, also determines if the deformity can be reduced, and the level of forefoot deformity (varus or supinatus) that accompanies the AAFD.

- **Double/single heel raise** – with a functioning posterior tibial tendon, the rearfoot will raise at least four inches off the floor and the calcaneus inverts. In a stage II deformity, the rearfoot will plantar flex on the forefoot with no aid from the supporting structures; a collapse of the midfoot will be apparent with little to no elevation in the heel off the ground.

- **Hubscher manoeuvre** – passive dorsiflexion of the hallux normally activates the windlass effect and causes an external rotation of the leg (through ligamentous connection and movement transfer). In a stage II PTTD deformity, passive elevation of the hallux is very difficult, if not impossible, due to severe pronation causing maximal tension on the plantar fascia which resists dorsiflexion of the hallux.

- **Modified Romberg test for balance** – the patient stands on one leg with arms crossed over chest and eyes closed; in an adult acquired flat foot, balance is compromised severely (used for evaluation purposes only, not diagnosis of PTTD).

- **Supination lag examination** – with the legs relaxed and bent over a table, the patient actively supinates the foot; in a stage II deformity there will be no
medial movement. Look for asymmetry between the two feet as well as noting the curvature of the affected foot.

- **Test for strength of the tibialis posterior muscle/tendon** (positioning is very important to eliminate the contribution of the tibialis anterior) – place the thumb on the plantar medial half of the first metatarsal phalangeal joint and push the foot into abduction, allowing the patient’s foot to plantar flex, then ask the patient to adduct their foot against your resistance (palpate tibialis anterior tendon to ensure it hasn’t been recruited). Feel for the quality of resistance, especially compared to the contralateral side.

- **Measure medial longitudinal arch height** – by placing a hand under the arch of each foot, the examiner can use their finger creases as a reference, which allows comparison of height between both feet (Edwards et al., 2008).

- **Silfverskiold’s test (ankle dorsiflexion)** – assess ankle dorsiflexion with knee flexed and extended to determine whether gastrocnemius or soleus or both are tight (Edwards et al., 2008).

- **Palpation for pain** – in earlier stages along the tibialis posterior tendon, in later stages moving lateral to the sinus tarsi and distal fibula.

### Contraindications

The main contraindication for PTTD lies within surgical treatment. There do not seem to be contraindications for the more conservative treatments. Treatments can be provided based on the stages of PTTD and patients should be treated conservatively at first, before surgical treatment is an option.

### The Patient Perspective

Patient complaints may include: pain and swelling along the posterior tibial tendon (inside of the foot), decreased walking distance or function, abnormal wear of the medial heel on shoes, lateral rearfoot pain (on the outside of the heel—this is secondary to impingement of the calcaneus on the fibula), and significantly reduced stride length, cadence, and walking speed, which may explain why patients may present with a decreased function rather than a changing foot shape (Edwards et al., 2008). Later changes with a pathologic flatfoot may include sinus tarsi inflammation, lateral rearfoot impingement, and arthrosis (Neville, Flemister, & Houck, 2010; Kohls-Gatzoulis et al., 2008; Kong & van der Vliet, 2008; Marks et al., 2009). Patients may also describe a feeling of “rolling in” on the affected foot, weakness, and instability. As the PTTD progresses they may not feel as though they can reach for things that require them to go up on their toes. Standing ankle plantar flexion may cause feelings of instability, weakness, and possibly pain.

### Common Treatment

The stages of PTTD can be used as a guideline for treatment management and further progression of PTTD. Treatment ranges from conservative to surgical. Most patients can likely start with conservative management before moving to surgical considerations. Conservative management includes (Edwards et al., 2008):

- **Orthoses** – support medial longitudinal arch and correct rearfoot eversion in stages I and II; in the later stages, the role of the orthosis is to accommodate the deformity and prevent progression of the condition

- **AFO** – custom to correct or accommodate deformity

- **Footwear** – lace-up shoes that allow enough width through the midfoot to accommodate any deformity

- **Footwear modifications** may be necessary to accommodate a deformity depending on the stage of PTTD. Possible modifications could include a medial buttress or a midfoot split and widening.

- **Walking cast** – immobilization for 6–8 weeks

- **Non-steroidal anti-inflammatories** – if there is tenosynovitis

- **Physiotherapy** – muscle strengthening and stretching

Edwards et al. describe the above conservative methods with a note that physiotherapy is controversial and that “surgical treatment can produce good to excellent results in more than 80% of patients at up to 5 years” (Edwards et al., 2008). They report many different types of surgeries but note that stage III and IV typically require a triple arthrodesis (subtalar, calcaneocuboid, and talonavicular fusion) (Edwards et al., 2008).

Below is a summary from various authors and research articles in regards to treatment of PTTD, although the reader should note that there is no consensus on best management strategies for PTTD:

- **Orthoses** – Ringleb, Kavros, Kotajarvi, Hansen, Kitaoka, & Kaufman (2007) note that “non-
operative treatment of patients with PTTD should not only consider reducing posterior tibialis activity with arch support, but should also use a similar approach to reduce the activity of the peroneals, tibialis anterior, and the gastrocnemius. When designing an orthosis, the above structures must be addressed, as well as the rearfoot and medial longitudinal arch. Neville (2007) suggests that any treatment that limits abnormal foot pathomechanics that lengthen the posterior tibialis tendon would be a beneficial treatment method (i.e., controlling rearfoot eversion and forefoot abduction) compared to those that act to only support the medial longitudinal arch. Orthoses such as the Arizona brace (AFO) and UCBL may provide support for excessive rearfoot eversion when the rearfoot contacts the ground. This may in turn limit the amount that rearfoot eversion affects the length of the posterior tibialis tendon. Neville et al. (2007) also note that bracing and orthoses have not yet been studied on their ability to control forefoot abduction, especially while the foot is off the ground during later phases of gait. Orthoses supporting the medial longitudinal arch may also be able to protect the supporting structures (spring ligament, sinus tarsi ligaments, and plantar fascia) while the posterior tibialis tendon is failing.

• AFO – Many clinical guidelines recommend the use of orthoses for conservative treatment of PTTD, but choosing the appropriate orthotic device may be difficult. Evidence suggests that the use of a rigid AFO that crosses the ankle joint will be the most successful in treatment of PTTD. Neville & Houck (2010) report that the goal of the orthosis is to unload the tibialis posterior tendon by correction of rearfoot eversion and forefoot abduction. By supporting the medial longitudinal arch as well, the orthosis will unload the spring ligament. They note, “Correction of forefoot abduction is thought to occur by controlling the hindfoot and MLA” (Neville & Houck, 2010, p.2). Although rigid AFOs may be the most appropriate way to unload the posterior tibialis tendon, they also restrict motion at the ankle joint, thereby inducing plantar flexor weakness and increasing patient dependence on the device. Neville et al. (2009) completed a case study on one patient with stage II PTTD. The purpose was to recommend the best of three AFOs for this patient, one being an OTC AFO, one a custom solid AFO, and one a custom articulated AFO. They found the articulated AFO, which allowed ankle movement, to be better at reducing rearfoot eversion as compared to the solid AFO. The custom articulated AFO also achieved reduction of forefoot abduction and support of the medial longitudinal arch. The authors do not have an explanation as to why the articulated orthosis managed the foot mechanics better; however, they hypothesize that the allowance of the muscle dynamic added to the support of the AFO (Neville et al., 2009).

• Physiotherapy – a report by Neville et al. (2007) report suggests that doing strengthening exercises while wearing orthoses increases the contribution of the posterior tibialis muscle. Alvarez et al. (2006) completed a study with stage I and II PTTD patients without a complete rupture of the tendon. The treatment consisted of an AFO or foot orthosis with a strengthening program. The results concluded that most of this group responded well to the treatment “of an orthotic device and a graduated strengthening and neuromuscular facilitation program” (Alvarez et al., 2006). After approximately 4 months on this treatment plan, most patients had no pain or a reduction in pain, could walk on their toes, had no functional limitation of activity, and could perform a single heel raise test (Alvarez et al., 2006). In the 2010 study, Neville & Houck suggest that muscle strengthening alone may only partially correct flat–foot mechanics in patients with stage II PTTD, as there are other factors that contribute to poor foot mechanics during gait. Kulig et al. (2009) studied the effect of eccentric and concentric loading of the tibialis posterior and stretching of the gastrocnemius/soleus complex on pain and function in patients with PTTD. They had every patient also wear custom-made orthoses for 90% of their day. They found great improvement in function and pain with the strengthening, stretching, and orthotic device, but could not determine whether eccentric or concentric loading was more beneficial. The authors did note that the patients were able to perform both loading exercises without pain. Another interesting point was in regards to eccentric loading and the overload principle. “The overload principle speaks to the necessity to stress biologic tissues beyond their current thresholds to increase tolerance to subsequent stresses and avoid future injuries” (Kulig et al., 2009). Houck et al. (2015) looked at augmenting orthosis use with stretching only vs. stretching and strengthening and found that there were minimal improvements with the orthosis, stretching, and strengthening program; however, found limitations to their study, and suggest that a longer term and more intense activity regimen might improve the results. The orthosis used in their study was a DJO ankle brace (Airlift PTTD Brace).
• Surgical – Marks et al. (2009) discussed different surgical methods and concluded that treatment for stage II PTTD requires a combination of soft tissue repair of support structures (spring ligament and talonavicular capsule) and an FDL tendon transfer.

References


Accessory Navicular

Nicola Smith, BSc (Kin), C. Ped (C)

Key Messages

- Most prevalent in adolescent females.
- There is no definite functional test to conclusively diagnose an accessory navicular: it requires medical imaging for confirmation.
- Accessory naviculars are classified into three subtypes: Type I, II, and III; Type II being the most symptomatic.
- Pain presents primarily at the medial midfoot localized to the navicular.
- Conservative treatment can manage symptoms; however, surgical intervention may be required for recalcitrant cases.

Keywords

bony, cornuate, inflammation, ossification, osteochondrosis, sesamoid, synchondrosis, TPT

Introduction

Accessory bones are a common occurrence within regular anatomy and are considered to be developmental anomalies. Accessory bones are most frequently found within the feet and hands. The accessory navicular is the most commonly occurring accessory bone of the foot and the most likely to present symptomatically as pain (chronic or acute) on the medial aspect of the foot. Conservative treatment can manage symptoms; however, surgical intervention may be required if symptom relief is insufficient or short-term.

Pathology

The accessory navicular (AN) was first documented by Bauhn in 1605. Over the years “navicular secundum,” “pre-hallux,” “bifurcate navicular,” “divided navicular,” “os tibiale externum,” “accessory scaphoid,” and “accessory tarsal scaphoid” have all been used to describe the accessory navicular (Evans, Averett, & Saunders, 2002; Fredrick, Beall, Ly, & Fish, 2005). AN is one of 21 potential accessory bones of the foot (Gluck, Heckman, & Purekh, 2010) occurring with a reported incidence of 4–21% (Issever, Minden, Eshed, & Hermann, 2007; Leonard & Fortin, 2010). AN is one of 21 potential accessory bones of the foot (Gluck, Heckman, & Purekh, 2010) occurring with a reported incidence of 4–21% (Issever, Minden, Eshed, & Hermann, 2007; Leonard & Fortin, 2010). Reported incidence varies greatly depending upon the research; however, only 1/1,000 AN becomes symptomatic (Butler, 2003).

The navicular derives its name from its unique boat shape and lies within the medial aspect of the midfoot. Several cartilaginous surfaces allow it to articulate with the talus (proximally), the cuboid (laterally), and the cuneiform bones (distally). The navicular has the following attachments: talonavicular, navicular–cuneiform, and calcaneonavicular ligaments which are situated on the dorsal aspect of the bone, while the spring ligament inserts on the plantar surface. The tibialis posterior tendon inserts onto the plantar and inferior surface of the navicular, providing both plantar flexion and inversion of the foot (Scott-Moncrieff, Forster, Andrews, & Khan, 2007).

AN exists as a cartilaginous bony precursor as early as the second month of fetal development but is not apparent radiographically until 9–12 years of age (Baker, Dupras, & Tocheri, 2005). The navicular is the last of the tarsal bones to ossify; ossification occurs approximately two years earlier in females than males, between 1–3 years of age for girls and 3–5 years of age for boys (Ugolini & Raikin, 2004). AN is a congenital anomaly in which the tuberosity of the navicular develops from a secondary centre of ossification. Incidence of bilateral AN has been reported to vary from 50–89% (Romanowski & Barrington, 1992). Studies by Dobbs and Kiter confirm that AN is an inherited trait, and that a single gene is responsible (Dobbs & Walton, 2004; Kiter, Erduran, & Gunal, 2000).
AN is classified into three types according to size and location (Sella, Lawson, & Ogden, 1986):

**Type I** is a true sesamoid bone located within the tibialis posterior tendon. It is well-defined, round or oval in shape, and completely separate from the navicular. It measures 2 to 6mm in diameter and is situated posteromedially to the medial border of the primary navicular. Type I AN is very rarely symptomatic.

**Type II** are united to the navicular by a fibrocartilaginous structure called a synchondrosis. This synchondrosis is approximately 1–3mm thick. Type II AN ossicles are heart or triangular in shape and approximately 8–12mm in size. Type II has been further subdivided into two subclasses: Type IIa- is connected to the navicular by a less acute angle which results in a greater tension force across it making it more susceptible to avulsion injury. Type IIb- is situated more inferiorly and therefore more prone to injury from shear forces.

**Type III** is united to the navicular by a bony bridge producing a cornuate navicular. This is considered to be an end stage Type II.

Type I makes up 30% of all AN with Type II and III compromising the remaining 70%. Pain from the presence of AN arises almost exclusively from Type II, although very prominent type III may cause overlying soft tissue inflammation of an associated bursa (Romanowski & Barrington, 1992).

Historically, it was reasoned by Kidner (the surgeon who pioneered the initial surgical treatment for AN) that the AN led to a pes planus foot structure. There may be a co-existent flexible flat foot, but there is no conclusive evidence of a cause and effect relationship between the two conditions (Dobbs & Walton, 2004). It is more accurate to infer pes planus and overpronated foot structures possess the pathomechanics and/or required forces and friction to cause an AN to become symptomatic.

The four most common theories as to the etiology of the pain are: tibialis posterior tendinitis (TPT), pressure or inflammation secondary to bony prominence, abnormal biomechanics of the foot, and trauma to the synchondrosis.

**Tibialis posterior tendinitis**

TPT primarily inserts onto the navicular and is the main inverter of the foot. It has been found that when an AN is present, the TPT inserts directly onto the AN without any continuity onto the sole of the foot, and the resultant forces of a TPT contraction are exerted onto AN and the synchondrosis. MRI findings of a symptomatic AN show persistent edema patterns in the AN indicating osteonecrosis, inflammation, and destruction of the cartilage (Lawson et al., 1984). Pain associated with AN may be secondary to direct trauma or irritation and indirectly from tibialis posterior tendinitis or tears (Requejo, Kulig, & Thordarson, 2000).

**Pressure of inflammation secondary to the bony prominence**

Examination of Type III AN shows chronic inflammation surrounding the AN. As with any bony protuberance, pressure or friction (caused most notably by footwear) will create points of pressure, and as a result, create irritation and inflammation. MRI findings of an AN show a persistent edema pattern in the AN indicating osteonecrosis, inflammation, and destruction of the cartilage (Lawson et al., 1984).

**Abnormal biomechanics**

There are three types of biomechanical forces acting simultaneously on the Type II AN synchondrosis when pes planus or excessive pronation is present. The TPT produces tension and shear, while increased pronation of the subtalar joint adds compression. The result of these combined forces is trauma to the synchondrosis, producing a clinically painful state. Once the trauma has begun and bony disruptions are established, the same biomechanical forces prevent it from healing (Sella et al., 1986; Sella & Lawson, 1987).

**Trauma to synchondrosis**

Recent histological analysis of excised ANs suggests an injury to the area of the cartilaginous synchondrosis, and in most cases, the patient did not recall a specific trauma (Requejo et al., 2000).

**Differential Diagnosis**

**Navicular fracture**

Primary differential diagnosis for the symptomatic AN is a fracture of the medial tuberosity of the navicular. The fracture usually results from a fall from a low height with forced eversion (Requejo et al., 2000). In contrast to an AN, tenderness associated with a fracture is more diffuse and there is often ecchymosis (bruising) on the medial side of the foot (Requejo et al., 2000). Type II AN are most commonly mistaken for navicular fractures (Leonard & Fortin, 2010), however upon imaging, AN have smooth rounded borders as opposed to fractures which have sharp ragged edges (Leonard & Fortin, 2010).
Kohler’s disease
Osteochondrosis of the navicular is of unknown etiology, most commonly unilateral affecting children 5–6 years old. It presents as pain at the midfoot and generally resolves spontaneously with time. Radiographs will be able to differentiate between AN and Kohler’s (Sharp, Calder, & Saxby, 2003).

Tibialis posterior dysfunction/rupture/avulsion
This is more difficult to differentiate as many symptomatic AN will have simultaneous TPT inflammation and varying degrees of dysfunction. A study by Evans, Averett, & Saunders (2002) found that the accessory bone is present two to four times more frequently in patients with TPT tears.

Other differential diagnoses include tarsal coalition, arthritic changes, tight heel cords, osteoid osteomas and other tumors, or injury to the deltoid or spring ligament.

Common Testing
Upon examination there will be tenderness over the navicular area and pain will be typically localized rather than diffuse. It may be possible to feel motion between a prominent accessory and primary navicular. Resisted inversion may be painful with or without accompanying tenderness over the tibialis posterior tendon (Herring, 2008). Swelling and erythema can also be present (Leonard & Fortin, 2010).

Many children may have asymptomatic AN that may be noticed incidentally on clinical or radiographic examination. It is important to note that a true navicular extends well medially and toward the plantar surface of the foot, and a prominence in this area may be a prominent navicular tuberosity (Herring, 2008). This is especially true when the patient also presents with a planovalgus foot alignment which in itself causes the navicular to become more prominent. Direct palpation of an AN will generally reproduce pain symptoms; however, pain upon palpation is not clinically conclusive due to the multiple differential diagnoses.

If an AN is suspected, radiographic views should be acquired to confirm its presence (Issever et al., 2007). Plain radiographs are generally the only imaging required to diagnose an AN.

MRI is considered to have highest sensitivity and specificity for diagnosis of a painful AN (Issever et al., 2007). MRI is the imaging of choice for the evaluation of acute and chronic injuries of the midfoot as it offers superior soft tissue resolution (Chiu, Jou, & Lee, 2000).

Ultrasound may demonstrate possible involvement of synchondrosis in symptomatic type II, or help identify soft tissue involvement (Issever et al., 2007).

Contraindications
Conservative treatment contraindications are not well documented; however, logically it follows to avoid direct pressure to the AN and activities which intensify the symptoms. Rigid foot orthoses tend to exacerbate symptoms by increasing pressure on the AN (Hsu, Michael, & Fisk, 2008).

Contraindications to surgery are few but those relative include: tendinosis, fixed hindfoot deformity, tarsal coalition, moderate to severe flexible hindfoot deformity and associated gastrocnemius or Achilles tendon contracture or neuromuscular spastic disorders (Shurnas & Saunders, 2007). These all lead to poor outcomes from surgical intervention.

The Patient Perspective
Although medial foot pain, often localized to the navicular, is common to both groups, presentation in the child and adult may differ considerably (Ugolini & Raikin, 2004). In children, the presenting complaint is often more chronic, described as bruising or aching and related to pressure or rubbing of a medial prominence against the shoe. This generally accompanies a recent increase in activities but is less likely to be related to a traumatic event. A symptomatic adult is more likely to have an acute onset of pain, described as sharp, often after an eversion injury or other foot trauma (Gluck et al., 2010; Ugolini & Raikin, 2004).

The most distinguishing feature in patients with symptomatic AN is a palpable, localized tenderness at the site of the navicular. Specifically, there is inflammation over the medial navicular with swelling, redness, and palpable protuberance (Lawson et al., 1984; Requejo et al., 2000; Ugolini & Raikin, 2004). Pain is often reproducible by direct pressure over the AN and TPT insertion, or by resisted inversion testing (Ugolini & Raikin, 2004).

Pain resulting from an AN is almost exclusively related with a type II AN (Romanowski & Barrington, 1992). AN rarely present as symptomatic prior to adolescence (Sullivan & Miller, 1978). The majority of
patients become symptomatic in the second decade of life, although incidents have been documented in up to the sixth decade of life (Lawson et al., 1984).

**Common Treatment**

Treatment modalities (appropriate footwear, foot orthoses, and walking casts) in combination with analgesics, anti-inflammatory medication, and ice should be expected to provide symptomatic relief. Reported success with conservative treatment intervention ranges from 13–33%. The reported acuity of the symptoms and presentation of the AN can determine the most appropriate order of intervention.

**Footwear**

If the AN presents as a large bony prominence, the majority of the patients can be managed conservatively by changes or adjustment to footwear. When symptoms are caused by pressure over the navicular, a shoe that avoids pressure over the area should be dispensed. Relieving shoe pressure over the prominences (Herring, 2008) with the use of soft pads between the foot and the shoe, usually with a horseshoe accommodation, can negate direct pressure to the AN. Elevated arch pads are not beneficial for these patients because the pads can cause increased pressure or further aggravate the AN. Appropriate fitting footwear can be further modified to accommodate for the AN. Creating a pocket at the AN either through stretching of the upper, excavation into the midsole or balloon patching are all viable footwear modifications. Any of these modifications either independently or in unison can minimize friction and pressure created by the shoe gear (Herring, 2008).

**Change in client routine**

An adjustment of activity levels, intensity, or frequency can manage pain and discomfort (Lawson et al., 1984). Avoidance of activities that aggravate or require footwear that increases symptoms can minimize symptoms.

**Low-dye taping**

When pathomechanics are thought to be part of the etiology of pain symptoms and success from a foot orthosis unclear, low-dye taping can be a temporary quick technique to gauge responsiveness to foot orthosis therapy.

**Orthoses**

Foot orthoses should be utilized in conjunction with therapy and footwear modification. When a planovalgus foot structure is present, a UCBL type orthosis may prove effective in alleviating pressure over the navicular. A UCBL foot orthosis inverts the patient’s heel during gait rather than correcting through pressure or contact through the arch (Herring, 2008). Little research exists on the material usage for treatment of an AN. Common foot orthosis practice is accommodating for the AN with pocketing and soft top covers. Care should be taken not to increase direct pressure to the AN as this may exacerbate symptoms rather than decrease them. Treatment of the individual patient is empirical, and no literature supports the use of one orthosis over another (Hsu et al., 2008).

**Walking cast**

For a client in acute pain, especially during early onset of symptoms, immobilization in a short-leg cast is also recommended. Non-surgical treatment with a short-leg cast may lead to abatement of the symptoms (Fredrick et al., 2005; Canale & Beaty, 2007).

**Injection/medication**

In more recalcitrant cases, an injection to the synchondrosis with steroids and an analgesic agent may be indicated (Herring, 2008). The inflammatory changes in the soft tissues, including tenosynovitis and bursitis, may respond to anti-inflammatory medication.

**Surgery**

There are numerous surgical procedures to address an AN when conservative intervention has failed. Surgeon preference, type and size of the ossicle, age, and activity level of the patient all factor into which procedure is performed. The Kidner procedure involves the removal of the accessory navicular and reattachment of the tibialis posterior tendon back onto the medial plantar aspect of the navicular. A new method of treatment is percutaneous drilling of a symptomatic AN in order to induce or accelerate bony union. This is yielding positive results and is advantageous as it is minimally invasive, easy, and economical. Further studies are needed to validate this method (Leonard & Fortin, 2010). Arthrodesis of the AN to the primary navicular along with advancing the tibialis posterior tendon distally and plantarly is also a more recent procedure. A study by Fredrick et al. (2005) suggests excision of the AN results in best return to function, and is the most popular procedure. It has few associated risks and complications, and it effectively reduces pain.
It is not unusual for symptoms to persist after surgical intervention of the AN. If the primary navicular is prominent medially, pain and tenderness may continue over the area even though the AN has been removed. In some cases, the scar itself and the area beneath the scar remain tender. Causes of this are unclear; however, these symptoms usually diminish with time (Herring, 2008).

**Key Considerations**

When diagnosing medial midfoot pain it is important to consider AN as a possible cause.

Pain etiology will present differently between children and adults. In adolescence, patients’ pain stems from increased activity or shoe pressure, whereas adult symptoms are generally caused by a trauma.

Surgical intervention should be considered a viable option when conservative measures offer transient or insufficient symptom relief.

**References**


Tarsal Coalition

Melissa Rabbito Lujan, MSc, C. Ped (C)

Key Messages

- Tarsal coalitions typically present in patients aged 9–16, with the calcaneonavicular and talocalcaneal joints being affected the most often.
- Not all cases of tarsal coalition are symptomatic, and non-symptomatic cases should not be treated aggressively.
- Diagnosis should be suspected if the patient is in their preteen or teenage years, complaining of insidious onset ankle pain in association with a lack of subtalar joint range of motion.

Keywords

heel impact, intense contraction, interarticular, localized pain, overuse, prolonged swelling, repetitive loading, rotational forces

Introduction

A tarsal coalition is defined as a union between two or more tarsal bones of the foot and can be further classified as osseous (bony), non-osseous (cartilaginous) or fibrous (syndesmosis) (Zaw & Calder, 2010). The condition is congenital via autosomal dominant inheritance (only one parent needs to have the gene to pass it on) (Bohne, 2001). The most common sites of coalition are the calcaneonavicular (CN) joint and the talocalcaneal (TC) joint, and very rarely it may occur in the talonavicular (TN) joint (Cass & Camasta, 2010). Tarsal coalitions typically affect adolescents, with presentation of symptoms occurring between the ages of 9 and 16, appearing to correlate with bone ossification (Bohne, 2001; Vincent, 1998). Patients will typically present with a rigid pes valgoplanus foot type, limited ankle range of motion, and possible peroneal spasm. Although the condition can create symptoms of ankle pain, not all patients are symptomatic. In rare cases, patients with tarsal coalition will not demonstrate a pes valgoplanus foot type; therefore, a definite diagnosis requires diagnostic imaging with oblique X-ray, MRI, or CT scan of the foot.

Pathology

Several theories have been proposed for the etiology of tarsal coalitions (Cass & Camasta, 2010). Tarsal coalition can be described as either congenital or acquired (Zhou, Tang, & Hardy, 2014). Talocalcaneal coalitions can be acquired by degenerative joint disease, inflammatory arthritis, infection, fibular hemimelia, and clubfoot deformities (Zhou et al., 2014).

The most widely accepted theory of congenital coalitions was proposed by Lebouq in 1890 (Cass & Camasta, 2010; Bohne, 2001). Lebouq proposed that tarsal coalitions occurred due to an incomplete segmentation of the embryonic mesenchymal tissue (Bohne, 2001; Cass & Camasta, 2010). As previously mentioned, the majority of coalitions occur in the CN or TC joints, making up 90% of all coalitions (Cass & Camasta, 2010). Not all coalitions are symptomatic or painful, but if pain is present, it is proposed to result from the mechanical stress imposed on the periosteum surrounding the ossifying joint. Typically there is no inflammation associated with the condition (Cass & Camasta, 2010). Histopathological studies have noted the absence of inflammatory cells in many cases of talocalcaneal coalition (Zhou et al., 2014).

Incidence rates range from 0.04–14.54%, according to the literature, and coalitions are found bilaterally in 50–80% of cases (Cass & Camasta, 2010). A commonly quoted statistic is a 1% incidence rate in the general population (Sakellariou & Claridge, 1999; Solomon, Ruhli, Taylor, Ferris, Pope, & Henneburg, 2003; Vincent, 1998; Zhou et al., 2014); however, these authors may have been reporting only symptomatic patients who presented to their physician with pain. Since not all tarsal coalition cases are symptomatic, many may go undiagnosed. To estimate incidence
rates not associated with symptoms, Solomon et al. (2003) examined 100 feet in 55 cadavers and found a rate of 12.72%. The authors did not know the medical history of the cadavers, thus were more likely to include people who had tarsal coalitions but never presented with symptoms. In 2008, Nalaboff & Schweitzer suggested that incidence rates could be as high as 11% based on a retrospective study of ankle MRIs.

Tarsal coalitions can exist with multiple combinations of structure and symptoms. Patients may present with or without pes valgoplanus foot structure, and with or without symptoms. Cases have been noted where asymptomatic coalitions were reported during imaging for unrelated purposes (Cass & Camasta, 2010).

Tarsal coalitions occur in the joints of the rear (or mid) foot and symptoms are typically noted in the midfoot, rearfoot, and/or ankle. The triple joint complex (subtalar, talonavicular, and calcaneocuboid) is responsible for the foot’s ability to adapt to uneven surfaces and motions of pronation and supination (Aston, Deland, Otis, & Kenneally, 1997). Fusion (as in a tarsal coalition) or decreased range of motion in any of these joints will result in excess stress placed on the hindfoot joints. If motion is decreased or absent in the subtalar joint, stress is often placed on the neighboring ankle joint, thus sprains or ankle joint degeneration can occur (Aston et al., 1997; Bohne, 2001). The TN joint is noted as the most significant joint in the triple joint complex, as it has the greatest range of motion. Fusion of the TN joint essentially eliminates range of motion in the remaining two joints of the triple joint complex (Aston et al., 1997), thus coalition of this joint is particularly troublesome to patients. Luckily, TN coalitions are rare, with fewer than 50 cases documented in the literature (Cass & Camasta, 2010).

Symptomatic patients will typically present with “ankle” pain which is aggravated by activity. The onset may be insidious, but is commonly exacerbated by a traumatic incident such as a sprained ankle.

CN coalitions typically present at ages 8–12, while TC coalitions present around ages 12–16 (Cass & Camasta, 2010). Patients who reach their 20s without symptoms are likely to stay asymptomatic throughout the rest of their lifetime (Bohne, 2001). It is believed that asymptomatic patients possess enough compensatory motion in the adjacent joints to prevent symptoms from occurring. In contrast, it is believed pain occurs when not enough compensatory motion is available in adjacent joints (Sakellariou & Claridge, 1999).

Talocalcaneal coalition is the most common cause of spastic flatfoot and peroneal spasm is the most common clinical symptom in patients with talocalcaneal coalition (Zhou et al., 2014). As previously mentioned, tarsal coalitions often restrict motion in the subtalar joint. If internal rotation of the subtalar joint is restricted, the tarsal joints of the foot will compensate by flattening, which in turn will lead to lowering of the medial longitudinal arch and planovalgus appearance. This can lead to adaptive shortening of the peroneal tendons and reactive peroneal spasm or spastic flatfoot (Afolayan et al., 2016).

Patients with peroneal spasm and talocalcaneal coalition will have consistent pain located in the sinus tarsi and over the sustentaculum tali (Zhou et al., 2014).

To date, there is no commonly accepted classification system for tarsal coalitions. They are most often described by etiology, anatomy and/or tissue type (Afolayan et al., 2016).

**Differential Diagnosis**
- Rigid pes planus
- Ankle sprain
- Posterior tibial tendon dysfunction

**Common Testing**

Clinical examination should focus on the motion and function of the subtalar joint. Range of motion testing will reveal limited subtalar joint range of motion, particularly passive ankle inversion (Cass & Camasta, 2010). If the condition is determined to be unilateral by diagnostic imaging, refer to the non-affected limb to establish a normal range of motion for the individual. Palpation may reveal a mass and/or tenderness at the site of the coalition (distal to the medial malleolus for TC coalitions or the posterior portion of the navicular for CN coalitions) (Bohne, 2001). If the patient demonstrates a pes valgoplanus foot type, no arch will be present in the non-weight-bearing scenario. When the patient rises to a standing, weight-bearing position, there will likely be no change in foot structure and the foot will still demonstrate no arch. During static standing, the clinician will often observe a positive “too many toes” sign where the forefoot is in an abducted position relative to the rearfoot.

When in the standing position, there are several tests which can assist in confirming a diagnosis. Patients
with tarsal coalition will typically demonstrate a non-responsive Hubscher manoeuvre and a non-responsive heel tip test.

The Hubscher manoeuvre is completed with the patient in a standing, weight-bearing position. The clinician will try to passively dorsiflex the great toe while the tibia is externally rotated. Typically, due to the windlass mechanism, dorsiflexion of the toe will tighten the plantar fascia and cause a rise in the medial longitudinal arch (MLA). In patients with tarsal coalition, this response will not occur and the MLA will remain flat.

During the heel tip test the patient is standing while the clinician supinates the foot by raising the medial border of the foot. Due to the coupled motions of supination and tibial external rotation, the tibia should externally rotate during the forced supination and is generally visible at the level of the patella. In a patient with tarsal coalition, no external rotation of the tibia or patella will occur during supination (Sakellariou & Claridge, 1999; Afolayan et al., 2016).

A third test to assess subtalar joint function would be the heel raise test. While standing, the patient is asked to rise on to their tiptoes. During this action, the heel should invert in response to the medial line of pull in the Achilles. In a patient with tarsal coalition, the heel will not invert and will stay in a valgus position. This is clearly visible in patients with a unilateral condition, as the normal side will invert normally. This needs to be distinguished from posterior tibial tendon dysfunction, in which patients will not be able to do a single leg heel rise (Afolayan et al., 2016).

**Contraindications**

Off-the-shelf inserts are not recommended for this population, as the prefabricated arch support will likely be too aggressive and thus uncomfortable for the pes valgoplanus foot structure.

**The Patient Perspective**

If patients are symptomatic, they will typically describe a vague heel or ankle pain that is not localized and is aggravated by activity or prolonged standing. Patients may have a history of frequent ankle sprains or feel as if they are unstable on uneven surfaces (Sakellariou & Claridge, 1999). As mentioned, the two main types of coalitions typically coincide with different stages of ossification. Coalitions of the CN joint will typically present at ages 8–12, while TC coalitions will present at ages 12–16. In the rare case of TN coalitions, these patients will present much younger, at ages 3–5 (Cass & Camasta, 2010; Sakellariou & Claridge, 1999).

If the patient is asymptomatic, the complaint may come from the parent who is concerned about the excessively flat appearance of their child’s foot. Cases have been noted where coalitions are present without the associated pes valgoplanus foot structure, thus patients with all other signs and symptoms should still be sent for diagnostic imaging.

**Common Treatment**

Asymptomatic patients should not be treated aggressively. For young children, supportive footwear and regular annual observation are the recommended course of action. For symptomatic patients, all conservative treatment options should be explored prior to surgical consultation. Conservative treatment options include oral anti-inflammatories, activity modification, orthoses, and shoe modifications (Sakellariou & Claridge, 1999, Afolayan et al., 2016). Afolayan et al. (2016) point out that non-operative treatment does not equate to “non-treatment” and that all symptomatic patients should be managed nonoperatively as a first step. This is an important message for patients to understand.

There is a paucity of research regarding conservative treatment for tarsal coalitions; the majority of the treatment research focuses on surgical procedures. Several authors refer to orthosis as a conservative treatment option (Bohne, 2001; Sakellariou & Claridge, 1999; Vincent, 1998; Afolayan et al., 2016); however, no study has reported specific data on orthosis as a treatment option for this condition. In a detailed case report, David, Clark, & Bier (1998) assessed and treated a five-year-old girl with a symptomatic talonavicular coalition. The patient was treated exclusively with a custom orthosis and the authors report that the patient had full resolution of symptoms after three weeks of use. The orthosis used to treat the patient was a University of California Biomechanics Laboratory (UCBL) style orthosis made of polypropylene with the modification of a navicular cut-out to prevent irritation from occurring in the area. The authors also described a dual-density heel post, which was softer on the lateral side to provide shock absorption at heel strike, and firmer on the medial side to provide support as the foot moved into pronation.

In theory, the purpose of an orthosis for this condition would be to limit painful motion. Afolayan
et al. (2016) suggest that accommodative orthosis and ankle braces can alleviate pain by stabilizing the rearfoot and providing proprioceptive feedback. Michaud (1997) recommends casting the foot in the most comfortable position, and building the orthosis to maintain this position during ambulation. The orthosis should be manufactured from a rigid material such as Subortholene or polypropylene with high medial and lateral borders to help control rearfoot motion. If no forefoot compensations or deformations have occurred, a three-quarter-length orthosis would be suitable. Orthoses should be used in combination with professionally fitted, supportive footwear.

In cases where custom-made orthoses are not an option, motion control or modified footwear could be an alternative. As previously mentioned, off-the-shelf inserts are not recommended for this population as the prefabricated arch support will likely be too aggressive. Afolayan et al. (2016) postulate that corrective orthosis with features such as a medial heel wedge, Thomas heel, and/or medial arch support can actually increase discomfort in patients with stiff hindfeet. Pedorthists and other orthosis providers should exercise caution when considering these features in a custom or off-the-shelf device for patients with a tarsal coalition; however, the same authors note that a medial heel wedge, Thomas heel, and/or medial arch support could be useful following surgical correction as they would help maintain correct mechanics of the rearfoot.

Potential footwear modifications may include a medial flare or buttress, or even sole widening for the very difficult-to-fit cases.

In very severe cases, or cases where initial treatment options are not successful, a short-leg walking boot is recommended for 2–4 weeks, followed by the use of a UCBL orthosis (Vincent, 1998). Higher success rates with conservative treatment have been noted in patients with TC coalitions as compared to those with CN or TN coalitions (Bohne, 2001). If one year of conservative treatment is not successful in relieving painful symptoms, surgical consultation would be the next step. There are several surgical procedures available, depending on whether degeneration of the surrounding bones exists or not. Two common surgical procedures are resection of the coalition and arthrodesis (Sakellariou & Claridge, 1999).

Adjunct therapies such as physiotherapy, athletic therapy, or massage should be included in the treatment plan to address spasm or shortness of the peroneal or calf muscles (Afolayan et al., 2016).

**Key Considerations**

Diagnosis should be suspected if the patient is in their preteen or teenage years, complaining of insidious or sudden onset ankle pain in association with a lack of subtalar joint range of motion.

However, since tarsal coalition can occur with any combination of the previously mentioned signs and symptoms, the only way to get a truly accurate diagnosis is through diagnostic imaging such as X-rays or MRI.

**References**


Tarsal Tunnel/Medial Calcaneal Nerve Entrapment

Dana Hall, HBKin, C. Ped (C)

Key Messages

- Commonly misdiagnosed as plantar fasciitis, heel pain syndrome, or heel spurs.
- Involves entrapment (impingement) of nerves, most commonly the medial calcaneal nerve.
- Overpronation, rearfoot valgus or varus, growths or swelling within the tarsal tunnel, diabetes, and arthritic changes can predispose a patient to this condition.
- Often originates with trauma to the foot and ankle, rather than an unknown cause.
- The Tinel tap test, nerve conduction, diagnostic ultrasound, and a thorough patient history can be instrumental in determining the proper diagnosis.
- Symptoms are nerve-like: burning, pain, tingling, and numbness, and can travel along the nerve pathways along the entire foot.
- Orthotic and footwear intervention, along with manual/massage therapy, are key conservative treatment options.

Keywords

burning, heel pain, impingement, numbing, Tinel test, tingling

Introduction

The tarsal tunnel is located on the medial side of the ankle joint and consists of a small space extending from the posteromedial ankle to the plantar aspect of the foot (Delfaut, Demondion, Bieganski, Thiron, Mestdagh, & Cotten, 2003). It is generally located between the medial flexor retinaculum and the calcaneus (Decker & Albert, 2002). More specifically, the tunnel is a fibro-osseous structure with four canals separated by a fibrous septum/neurovascular bundle in the third canal. The tunnel is divided into the tibiotalar (upper) portion and the calcaneal (lower) portion (Delfaut et al., 2003). The border of the upper portion is covered by a deep aponeurosis (laciniate ligament, flexor retinaculum) and has an osseous floor formed by the posterior tibia and talus and contains the tendons of the posterior tibialis, flexor digitorum longus, and flexor hallucis longus muscles, as well as the posterior tibial neurovascular bundle (Delfaut et al., 2003). The lower portion is covered by the fusion of the flexor retinaculum (superficial and deep aponeuroses of the leg) and the superior border of the abductor hallucis muscle and fascia (Delfaut et al., 2003). The osseous floor is the posteromedial talus, inferior navicular, medial sustentaculum tali, and the calcaneus (Delfaut et al., 2003). Tarsal tunnel syndrome or entrapment occurs when the posterior tibial nerve becomes compressed within the space of the tunnel, causing a variety of symptoms in the foot and lower leg.

The medial calcaneal nerve most commonly branches from the posterior tibial nerve, but can also branch from the lateral plantar nerve (Martin-Olivia, Elgueta-Grillo, Veliz-Ayta, Orozco-Villasenor, Elgueta-Grillo, & Viladot-Perice, 2013). The nerve tends to have a single end point, but approximately 21% of people can have multiple end branches to the MCL (Martin-Olivia et al., 2013).

Approximately 10% of the general population will experience heel pain at some point during their lifetime, with tarsal tunnel or medial calcaneal nerve entrapment accounting for a less common but still notable cause of this discomfort (Diers, 2008). It is estimated that 2 million people in the United States will seek treatment for heel pain with 90% responding well to conservative treatment and 10%
requiring further intervention (Mowen, 2007). Tarsal tunnel entrapment is the most common entrapment condition of the foot and lower extremity (Medical Disability Guidelines, 2010).

Pathology
Tarsal tunnel entrapment “…occurs for a variety of reasons...excessive pronation, ligamentous injury to the ankle, ganglion, engorged veins, arthritic problems...trauma and even obesity” (Decker & Albert, 2002). Diabetes and rearfoot valgus or varus deformities can also cause entrapment symptoms (Hudes, 2010). Injury, diabetes, arthritis, and obesity generally result in swelling and decreased space in the tarsal tunnel that can cause or exacerbate symptoms involving the nerve. Abnormal biomechanics (overpronation, rearfoot deformities, etc.) can place undue stress on the tunnel, resulting in swelling and decreased space for the nerve to pass through without impingement.

In the upper portion of the tunnel, it is most common for space-occupying lesions to cause nerve entrapment, while in the lower portion mechanical causes such as overpronation are most likely to cause entrapment (Myerson, 2005).

Differential Diagnosis
Compression of the artery or vein can occur without nerve involvement, in which case the patient presents with sensation symptoms without movement or motor compromise (Hammer, 2007).

Because of the location of symptoms with tarsal tunnel syndrome, plantar fasciitis, heel spurs, and heel pain syndrome are commonly diagnosed if proper diagnostic testing is not conducted (Cluett, 2005).

Sciatica or peripheral neuropathy can also mimic this condition (Decker & Albert, 2002).

Other possible differential diagnoses are supernumerary muscles, tenosynovitis, arterial aneurysm, arteriovenous fistula, lipoma, neurilemmoma, post-traumatic fibrosis, peripheral arterial disease, hematoma, and radiculopathy (McGlamry, 2001).

Common Testing
A thorough clinical exam is one of the most important tools a practitioner can utilize to differentiate between nerve entrapment and other misdiagnoses (Kitaoka, 2002). One valid test to consider during the assessment is the percussion test (also called Tinel’s sign) of the medial calcaneal nerve pathway. This test consists of the practitioner tapping on the nerve as it passes along the medial portion of the ankle. A positive test occurs when the patient reports reproduction of the symptoms (Cluett, 2005). A positive Tinel’s test, or Tinel–type test, is present in approximately 67% of tarsal tunnel cases (Hudes, 2010). Due to the fact that a positive tap test is not present in all cases of nerve entrapment, many times this condition can be misdiagnosed (Hudes, 2010). As well, the nerves passing through the tarsal tunnel are small compared to other nerves, making an accurate diagnosis of which nerve is affected very difficult (Mowen, 2007). Practitioners can also use the Valleix sign, similar to the Tinel test, where tapping on the tarsal tunnel produces symptoms proximal to the area of entrapment (McGlamry, 2002).

Another valid test is the triple compression stress test where the ankle is inverted and plantar flexed, with pressure placed along the posterior tibial nerve. This test can reliably diagnose nerve entrapment in over 85% of cases (Abouelela & Zohiery, 2012). Conversely, the dorsiflexion–eversion test where the patient’s foot is dorsiflexed, everted, and toes extended for 5–10 seconds can reproduce symptoms (Kinoshita, Okuda, Morikawa, Jotoku, & Abe, 2001).

If nerve entrapment is suspected, or the patient is not responding to other typical conservative treatments for heel pain, referral for nerve conduction testing can be valuable in determining the specific diagnosis and achieving relief for the patient’s symptoms (Hudes, 2010). With this injury, conduction tests will often show decreased firing of the intrinsic muscles and inhibition of the medial and lateral plantar nerves (Therimadasamy, Seet, Kagda, & Wilder-Smith, 2011). Diagnostic ultrasound can also determine if there are cystic growths or other interference within the tarsal tunnel, and can show enlargement of the nerve due to trauma or entrapment (Therimadasamy et al., 2011). Diagnostic nerve blocks or cortisone shots to specific nerves can also be useful to determine different levels of entrapment (Myerson, 2005).

Because of symptom location and similarities to plantar fasciitis, it can be very difficult to obtain proper diagnosis without ultrasound and/or nerve conduction testing readily available to the consulting practitioner. Referral for MRI can be useful in determining if there are lesions or space–occupying tissues, and to rule out other pathology (Delfaut et al., 2003). Obtaining a proper medical history can yield
important clues as to symptom origin. For example, plantar fasciitis and heel spurs usually occur as the result of overuse over a longer time period, whereas nerve entrapment is commonly due to an injury, with symptoms developing suddenly (Diers, 2008). As well, symptoms with nerve entrapment will commonly present along the nerve pathway, without plantar fascia involvement, while plantar fasciitis will occur along the fascia bands or at the calcaneal insertion (Diers, 2008). Plantar fasciitis pain will often decrease with weight-bearing as the tissue flexibility increases, where nerve entrapment symptoms will increase with prolonged weight-bearing (Diers, 2008).

Another diagnostic tool that is gaining popularity is the pressure-specified sensory device. Preliminary testing has revealed 100% effectiveness compared with older sensation testing devices (Mowen, 2007). This device measures sensitivity to the area that the nerve innervates and quantifies the sensation (Mowen, 2007).

Electrodiagnosis is not considered reliable, as it is too specific. Usually a positive test will be present only with advanced neurological damage (McGlamry, 2001; Myerson, 2005).

**Contraindications**

Patient history, along with conditions or disorders such as diabetes or arthritis, will guide choices of footwear and orthotic needs. For example, a diabetic foot may require a functional semi-rigid orthosis. However a more cushioned full-contact top cover choice can help alleviate entrapment symptoms while also avoiding other complications associated with the diabetic foot.

Referral for massage therapy by the pedorthist should be avoided or postponed if the patient has current fever, open cuts/wounds, past history of thrombosis or blood clots, varicose veins, infectious skin disease, cancer, or bone fracture (Wieting, 2017).

Invasive treatment protocol involving medication, surgery, or injection should be discussed and ultimately decided upon with the referring physician before recommendation is advised to the patient.

**The Patient Perspective**

The patient will often describe this injury with words such as:

- Painful, burning, numbness, tingling
- Wakes them or keeps them up at night
- Rest does not resolve the symptoms
- Symptoms worsen as they stand, walk, or perform activity
- Ankle/foot injury
- Other treatments have failed to provide long-term relief

A key characteristic of tarsal tunnel conditions is that the discomfort and symptoms felt due to entrapment do not subside when the patient is non-weight-bearing (Decker & Albert, 2002). Nerve-like symptoms are commonly present around the ankle, heel, and/or into the foot: burning, tingling, numbness, and pain (Decker & Albert, 2002). In cases where there may be entrapment of more than just the medial calcaneal nerve, the patient can experience nerve-like symptoms all the way into the toes and/or the entire plantar surface of the foot (Therimadasamy et al., 2011). In chronic cases, atrophy and weakness of the intrinsic musculature of the foot will occur (Hudes, 2010). Night pain, cramping in the foot, and focal or diffuse discomfort can all be described by the patient.

**Common Treatment**

Conservative treatments include orthotic and footwear interventions, while more invasive treatments include possible surgical release of the retinaculum or neurolysis to release pressure on the nerve (Cluett, 2005). If surgical release is required, the medial plantar fascial band located deep to the abductor hallucis at the roof of the porta pedis should also be released to relieve both the lateral and medial plantar nerves (Myerson, 2005). Manual therapy and various types of massage or physiotherapy may also be helpful in releasing the retinaculum and reducing scar tissue buildup (Hudes, 2010). There are some case studies that have demonstrated that orthotic and/or footwear interventions alone were not enough to resolve symptoms in a timely manner. Massage and manual therapy are usually integral parts of the treatment plan and should be referred to as necessary by the initial practitioner (Hudes, 2010). Braces, NSAIDs, ultrasound guided cortisone injections, self-massage, and elevation of the limb are also treatment options to be used alone or in conjunction with the above (Delfaut et al., 2003).

In terms of pedorthic treatment, properly fitted footwear is a very important factor in avoiding further nerve entrapment problems. The footwear must have good fit at the heel counter, notch, and collar that controls the foot mechanics, but also must not further
impinge the nerve (Decker & Albert, 2002). When treating with custom foot orthoses, the practitioner must accurately identify and accommodate/correct forefoot and rearfoot deformities to achieve successful treatment. Shell, cushioning, and cover material choices are also important to enable the orthosis in correcting biomechanical abnormalities resulting in tarsal tunnel entrapment. The custom foot orthoses must address any biomechanical malalignments (especially at the heel and midfoot). They must also offer reduced or even expanded heel cups with a deflection and cushion at the nerve entrapment site.

In a case study by Diers (2008), a female patient presented with chronic heel pain over the course of eight years. This patient had tried orthoses (custom and prefabricated), physiotherapy, massage therapy, ultrasound, shockwave therapy, and cortisone injections with no long-term symptom relief. Each treatment would provide temporary symptom resolution, but the symptoms would always return. Due to the lack of positive treatment results, a clinician performed a modified Tinel tap test and yielded a positive result of reproduction of symptoms. Nerve entrapment was suspected and an injection of lidocaine to reduce sensation in the nerve yielded complete symptom resolution and confirmed the diagnosis of medial calcaneal nerve entrapment. From here, the consulting practitioners referred the patient for specific physiotherapy massage of the tarsal tunnel to release it, and administered another injection of cortisone directly to the nerve. With both of these treatments, the patient had complete resolution of the injury and related symptoms.

A modern approach to a more invasive treatment option is cryosurgical ablation, or removal of the entrapped nerve tissue, which has a 90% success rate when conservative treatment has failed (Mowen, 2007). This procedure is conducted in the office and allows patients to return to daily activity in a short amount of time, when compared to conventional surgical release (Mowen, 2007). If extensive damage to the nerve has occurred, this procedure is less effective, and thus surgical decompression would be the next viable option (Mowen, 2007).

**Key Considerations**

- Recent trauma to foot or lower leg
- No relief with rest
- Nerve–like symptoms in foot or around lower leg
- Overpronation, rearfoot valgus/varus, or other deformity
- Diagnosis of obesity, diabetes, or arthritis
- Positive Tinel’s test
- No long-term relief with other conventional treatments
- Treatment plan includes standard pedorthic treatment, along with manual therapy
- Consider nerve conduction testing and/or diagnostic ultrasound if this condition is suspected
- There is some evidence that this injury is more prevalent in women than men (Hudes, 2010)

**References**


Plantar Fasciitis

Adam Janke, C. Ped (C) (2012)
Updated by Michael Ryan, PhD, C. Ped (C) (2018)

Key Messages

• Plantar fasciitis has been documented in all foot types and may be caused as much by environmental factors (i.e., prolonged standing, footwear) as intrinsic foot posture. Thorough history and examination in the context of custom or over-the-counter (OTC) insoles, rockered footwear, and activity modifications should be considered where appropriate.

• Less than 10 degrees of ankle dorsiflexion has been identified as a contributing factor to plantar fasciitis. Identifying limited ankle dorsiflexion, and treating the underlying cause of the limitation, will improve patient outcome. From a pedorthic perspective, dorsiflexion night splints or socks, at-home stretches, and referral for soft-tissue release where gastrocnemius or soleus contracture is thought to be a component of the issue must also be considered.

• Leg length discrepancy (LLD) has been found to be a potential risk factor for plantar fasciitis. A pedorthist should be assessing for LLD with any patient presenting with plantar fasciitis, in particular those with unilateral symptoms.

• Plantar fasciitis can exist bilaterally, but differential diagnoses should be considered to rule out other conditions in cases with bilateral symptoms.

• If radiographic images are available, care should be taken by the pedorthist to accommodate any subcalcaneal spurs in custom orthoses design, OTC insole modifications, and footwear modifications.

Keywords

Apley’s, bounce home test, bucket handle, compression, fibrocartilage, McMurray’s test, parrot-beak, rotation, shear force

Introduction

Plantar fasciitis (PF) is a painful localized syndrome affecting the plantar fascia or plantar aponeurosis, a thick fibrous band of connective tissue that originates at the medial tubercle of the plantar calcaneus and inserts into the plantar plates of the metatarsophalangeal joints, spanning the majority of the plantar surface of the foot. PF is a degenerative syndrome resulting from repeated microtrauma typically to the origin of the plantar fascia on the inferior calcaneus (Cornwall & McPoil, 1999). The condition is estimated to affect as much as 10% of the population over the course of a lifetime (Crawford, Atkins, & Edwards, 2000) and is reported to be the most common cause of plantar heel pain (Singh, Angel, Bentley, & Trevino, 1997) accounting for 11–15% of professional visits related to foot pain on an annual basis in North America (Pfeffer, Bachetti, Deland, Lewis, Anderson, Davis, & Smith, 1999).

Plantar fasciitis is relatively prevalent in athletically active individuals and military personnel but is also diagnosed in the non-active general population as well (Buchbinder, 2004; Riddle et al., 2004; Singh et al., 1997). Recent research suggests that the term “fasciitis”, which denotes an inflammatory condition, may not be entirely accurate, as many cases present with non-inflammatory, degenerative processes that could be more appropriately named “plantar fasciosis” (Aldridge, 2004; Lemont, Ammirati, & Usen, 2003).

Pathology

The etiology of plantar fasciitis is not clearly understood, but is believed to be associated with a wide variety of factors. Mechanical stress and microtrauma associated with obesity and/or sudden weight-gain, occupation-related activity (prolonged weight-bearing), anatomical factors such as pes
planus and pes cavus, and reduced ankle dorsiflexion related to gastrocnemius and soleus contracture have all been identified as the greatest risk factors associated with plantar fasciitis symptoms (Buchbinder, 2004; Cornwall & McPoil, 1999; Riddle et al., 2003; Tallia & Cardone, 2003; Young, Rutherford & Niedfeldt, 2001). Secondary risk factors that have been identified are leg length discrepancy, excessive lateral tibial torsion, excessive femoral anteversion, and overtraining in athletic individuals (Messier & Pittala, 1988; Warren, 1984; Young et al., 2001). In the elderly, symptoms are often attributed to reduced foot intrinsic muscle strength and acquired pes planus (Young et al., 2001).

For years, calcaneal spurs were thought to be a risk factor for PF, however, many patients diagnosed with plantar fasciitis do not have calcaneal spurs (DeMaio, Paine, Mangine & Drez, 1993) and many others with calcaneal spurs do not have plantar fasciitis (Cornwall & McPoil, 1999). Furthermore, the appearance of heel spurs does not appear to influence treatment success given significant reduction of pain can occur despite no reduction in calcaneal spur size (Ryan, Wong, Gillies, Wong, & Taunton, 2009).

Consistent with the “fasciosis” suffix, structural changes to the plantar fascia are becoming a hallmark of chronic injury. Recent ultrasonographic studies have found positive correlations between medial band fascial thickness, structural changes in collagen and ECM (extracellular matrix) thickening, microtearing, and pain typically associated with PF symptoms. Plantar fasciitis shares many of the clinical, histological and radiological attributes of overuse injuries of tendinous structures (Abdel-Wahab, Fathi, Al-Emadi, & Mahdi, 2008; McMillan, Landorf, Barrett, Menz, & Bird, 2009; Wearing, SMEathers, Sullivan, Yates, Urry, & Dubois, 2007). Indeed, the hyper-sensitivity to pressure in patients with plantar fasciitis, especially those with long-standing symptoms, is suspected to involve both local and central nervous system changes similar to those seen in other chronic soft-tissue injuries (Coombes, Bisset, & Vicenzino, 2012). Achieving pain-free movements at the forefoot and ankle should be the goal of any treatment plan to mitigate further neurosensory sensitization.

**Differential Diagnosis**

The following conditions and symptoms must also be considered when a patient presents with suspected plantar fasciitis:

- Calcaneal stress fracture
- Calcaneal fat pad irritation
- Intrinsic foot muscle strain
- Idiopathic cavus foot pain
- Peripheral nerve entrapment
- Tarsal tunnel syndrome
- Reiter’s syndrome
- Rheumatoid arthritis
- Plantar fibromas
- Infection

**Common Testing**

Plantar fasciitis is characterized by plantar foot pain most often focused at the antero-medial subcalcaneal region corresponding to the area of highest stress concentration to the plantar fascia (Cheng, Lin, Wang, & Chou, 2008). Note that while plantar fasciitis is a diagnosis that can be made clinically, ultrasound or MRI imaging is necessary to confirm actual structural abnormalities and tissue degeneration.

Plantar fasciitis commonly presents with palpable pain at the antero-medial aspect of the calcaneus. 
*Photo by Mary Chau*.
From a pedorthic perspective, a detailed history is necessary to rule out possible misdiagnoses and contributing factors outside of the pedorthic toolbox. Soft tissue imaging reports should be considered, particularly if they are negative for structural abnormalities. Both open and closed kinetic chain biomechanics must be considered.

**The following biomechanical tests and evaluations are appropriate in plantar fasciitis cases:**

- With the patient in non-weight-bearing (NWB) position, palpate around the medial tubercle of the calcaneus and note localized pain.
- In NWB, passively dorsiflex the toes to create tension along the plantar fascia and palpate distally along the medial, central, and lateral bands. Take note of any nodules or localized pain.

*With dorsiflexion of the hallux and lesser digits, the plantar fascia (and long flexor tendons) become visible (arrow). Photo by Mary Chau.*

- Open chain passive ankle dorsiflexion must be evaluated in the subtalar neutral position as described in the literature (Riddle *et al*., 2003) to reduce the chances of a false positive range-of-motion finding. A finding of less than 10 degrees of dorsiflexion is clinically significant and the gastrocnemius and soleus must also be palpated to identify or rule out isolated contractures in these muscles.

- Closed chain biomechanics and gait should be evaluated. Findings of excessive pronation or excessive supination should be noted and addressed to reduce additional mechanical strain on the plantar fascia.

- Asymmetrical gait patterns should also be noted and may be indicative of an underlying leg length discrepancy (LLD), especially if PF symptoms are unilateral.

- If LLD is suspected, further assessment of limb length should be completed.

**Contraindications**

- Activity levels, especially intense high-impact activities should be minimized in symptomatic athletic individuals. If steroid injections have been administered by the presiding MD, activity levels must be severely reduced to reduce the risk of plantar fascia rupture (Crawford *et al*., 2000).

- Prolonged weight-bearing should be minimized in the working patient.

- Prolonged barefoot walking on hard surfaces should be eliminated until symptoms reduce.

- Heel spur accommodations and heel pads should be avoided where loss of sensation – such as in the diabetic population – is a concern.

**The Patient Perspective**

- Dorsiflexion night splint to keep plantar fascia, gastrocnemius, and soleus in an elongated position for a prolonged period of time to improve passive ankle dorsiflexion (Roos, Engström, & Söderberg, 2006).

- Lift(s) if leg length discrepancy is confirmed.

- Footwear incorporating a strong forefoot rocker is recommended, where appropriate, to reduce additional plantar fascia stress during toe-off.

- In–home footwear with either built–in arch support, or the ability to accommodate a foot orthosis, is strongly recommended in cases where the patient spends prolonged weight-bearing indoors on hard surfaces.

- Plantar fascia and foot–specific strengthening exercises (Ryan, Fraser, McDonald, & Taunton, 2009; Ryan, Hartwell, Fraser, Newsham-West, & Taunton, 2014).

- Stretching exercises for the plantar fascia, gastrocnemius, and soleus (DiGiovanni, 2006).
Other Treatments (outside pedorthic scope of practice):

- Icing
- Rest/reduction in activity and/or work-related weight-bearing
- Anti-inflammatory medication
- Heel cups or heel lifts
- Manual therapy from either physiotherapist or chiropractor (Active Release Technique, intramuscular stimulation, Graston technique, deep massage)
- Registered massage
- Cortisone or steroid injections
- Extracorporeal and radial shockwave therapies
- Ultrasound guided dextrose injections (prolotherapy)
- Taping
- Surgical intervention: fascial release, fasciotomy
- Future direction of treatment to watch: platelet-rich plasma and stem cell research

Key Considerations

- Plantar fasciitis is now understood to involve a combination of structural abnormalities to the plantar fascia, as well as increased sensitivity to pressure due to neurosensory adaptations. Goals for treatment should include reducing pain (through footwear and orthotic therapy), as well as encouraging structural rehabilitation through progressive pain-free movements.

- Plantar fasciitis is common among individuals who are required to weight–bear for prolonged periods of time during the work–day. Discussing appropriate footwear options to minimize foot loads is important.

- The principal goal of foot orthosis treatment is to reduce strain on the plantar fascia through stabilization of the medial longitudinal arch.

- Referral for progressive tissue rehabilitation and clinical modalities such as shockwave therapy and manual therapy techniques should be explored in cases of calcaneal pain to foot orthoses.

References


Plantar Fasciitis
**Calcaneal Fat Pad Contusion**

**Mike Daigle**, C. Ped. (C) (2012)

Updated by **Davis DesRochers**, C. Ped. (C) (2018)

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**Key Messages**

- Heel pain is the most common complaint with which pedorthic patients present. The general term heel pain is not always plantar fasciitis, which is usually what the prescription states as the diagnosis.
- There are key differences in the presentation of fat pad syndrome and the other pathologies that affect the heel, but the treatments may be similar.
- Generalized centre heel or subcalcaneal aspect of the foot is the most common location of fat pad syndrome.
- Treatable with rest, taping, supportive footwear, and foot orthoses with a deep heel cup.

**Keywords**

bruise, calcaneodynia, ground reaction forces, heel pain, septa

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**Introduction**

Heel pain, or calcaneodynia, is one of the most frequent complaints and disabling clinical conditions presented to pedorthists. There are several pathologies related to heel pain but only one may be correctly referred to as calcaneal fat pad contusion. Hossain and Makwana (2011) suggest that pain in the plantar heel area be initially named heel pain syndrome until clarification of the symptoms can establish a firm diagnosis. Sometimes pathologies are indistinguishable, and in most medical texts they are used interchangeably. They include plantar fasciitis (which is the most common diagnosis), tarsal tunnel syndrome, subcalcaneal bursitis, and calcaneal fracture. These disorders are classified on the basis of anatomical origin and predominant location of heel (calcaneal) pain; that is, in the subcalcaneal aspect of the foot (Alexander, 1997). Onset may be insidious or acute and is usually unilateral.

Calcaneal fat pad contusion presents primarily as pain in the centre heel, often generalized over the entire heel fat pad. It is sometimes referred to as fat pad syndrome, bruised heel, stone bruise, policeman’s heel, or runner’s heel. It is a frequent ailment of soldiers caused by excessive marching. It refers to damage, disruption, atrophy, or flattening of the calcaneal fat pad. The calcaneus is the largest tarsal bone that bears all the weight during heel strike. It is the only bone to make initial contact with the ground, so it is at risk for injury when a person walks, runs, or lands from a jump (Alexander, 1997).

The skin on the sole of the foot is thicker than anywhere else in the human body, particularly the heel. The heel pad is uniquely designed to absorb shock, protect the underlying structures, and allow pain-free ambulation. The heel pad has a honeycomb structure and consists of fibroelastic septae extending from the calcaneum to the plantar skin. Enclosed between the septae are fat globules. Each fibroelastic chamber is further reinforced by diagonal and transversely running fibres (Hossain and Makwana 2011).

**Pathology**

Symptoms of calcaneal fat pad contusions include:

1. Swollen heel
2. Painful walking, running or jumping
3. Pain while standing
4. Tenderness on the heel when subjected to pressure
5. Redness or bruising on the bottom of the heel

There are two categories of fat pad injury: degenerative and traumatic. Degenerative injuries refer to normal thinning or atrophy of the fat pad which leads to greater impact loads on the heel. Degeneration is more frequent after 40 years because...
of atrophy and thinning of the fat pad with loss of water, collagen, and elastic tissue, reducing shock absorbency and protection of the calcaneus. It is documented that fat pad atrophy is the second most common cause of heel pain following plantar fasciitis (Yi, Lee, Seo, Huh, Yoon, & Kim, 2011). Risk factors are increased age, obesity, biomechanical anomalies, and certain occupation–related or sport specific activities. Yi et al. discussed the clinical diagnosis of plantar heel fat pad atrophy and how to differentiate it from other causes of heel pain, such as plantar fasciitis. They found that people with plantar heel fat pad atrophy presented with bilateral centralized heel pain (as opposed to unilateral medial tubercle pain) that occurred after prolonged standing or walking, and at rest.

Traumatic onset can be a sudden severe impact to the heel, such as a fall from a ladder or stepping barefoot on a stone, or due to rapid increased activity levels with walking or running. It is not specific to athletes or training in general. Many clients can have a change in lifestyle such as retiring and find that their daily activities have changed, which can bring on discomfort. Anecdotally, the pedorthist may discover a recent history of the patient working a double or triple shift on their feet. These “extra” hours on one’s feet are responsible for the added stress on the bottom of the heel, with the ground reaction force serving to flatten the heel pad. Work environment can be a relevant factor as well. Many clients working on hard surfaces such as nurses, care aides, cooks, and grocery clerks can be at a high risk for this type of heel pain.

Calcaneal fat pad contusion pain can limit job function and prevent an active lifestyle. The patient presenting with fat pad contusion will always describe the pain as “like walking on a deep bruise” or “a dull ache.” History taking will often discover a trauma like a blow to the heel, landing hard while barefoot, or jumping in dress shoes that have a hard heel. Pain is aggravated by walking barefoot on a hard surface like tile or hardwood floor, and the gait will be antalgic. Upon examination you will discover the patient is experiencing pain over the entire heel pad. A thumb pressed in the centre will re-create the pain.

Differential Diagnosis

Tarsal tunnel syndrome is an entrapment of the posterior tibial nerve causing pain paraesthesia, mostly inferior to the medial malleolus in the area known as the tarsal tunnel, but it often radiates into the subcalcaneal region. Pain is usually described as burning or numbness. Excessive pronation contributes to this. A positive Tinel’s sign can be elicited by tapping the nerve at the site.

Calcaneal fracture often accompanies a bruised heel. Determination is possible by compressing the centre heel with your thumb or by cupping the whole heel and squeezing. A fracture will always elicit pain. There will be swelling and possible discoloration. A radiograph like magnetic resonance imaging may be the only way to determine a fracture and the severity of the damage.

The plantar fascia is a band of tissue that is attached to the heel bone and connects to each of the toes, and its job is to provide support and stability to the longitudinal arch. Plantar fasciitis occurs when this band of tissue gets strained or irritated. Pain is usually described as burning or numbness. Excessive pronation contributes to this. A positive Tinel’s sign can be elicited by tapping the nerve at the site.

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Common Treatment

Most clinicians would agree that conservative or non-operative modalities are the most effective means of treating plantar heel pain.

Rest is a must when one is treating any type of heel pain. The patient should change or modify their regular routine to accommodate their damaged tissues.

Physiotherapy modalities like transcutaneous electrical nerve stimulation (TENS), ultrasound, cryotherapy (ice), and strapping the heel with low-dye tape all are effective in reducing the pain and promoting healing. Low-dye taping the heel to hold the fat pad underneath the heel and limit the movement of the fat pad can be very effective. If taping reduces or relieves the pain, then one’s impressions of fat pad contusion may be clearer.
An over-the-counter (OTC) gel heel cup is helpful in compressing the fat pad around the calcaneus to offload the impact of walking. It is important to note that fat pad contusion will heal, but reducing the inflammation and pain in the interim is our goal.

Footwear must be examined to determine whether it is a contributing factor. Hard heels are to be avoided. Educating the patients to wear shoes with a strong heel counter and heel cup, as well as a steel shank and rocker forefoot, will contribute to their successful healing. Running shoes have proved useful to wear for casual use, as they are cushiony and supportive. In-house footwear should provide more than a foot cover as well; most slippers do not provide the needed cushioning.

Custom foot orthoses that are designed to attenuate the shock and support the medial longitudinal arch have proved efficacious. The casts should be dressed to round the heels more so that the integrity of the fat pad is increased. This is sometimes lost in the foam casting process because the method of applying downward pressure to push the foot into the resisting foam tends to flatten the fat pad. Customized orthoses provide greater contact area with the sole, allowing plantar forces to be more evenly distributed and resulting in lower peak rearfoot pressure (Chia, Suresh, Kuah, Phua, & Seah, 2009). Also, moulding 6mm microcellular urethane foam heel pads in the centre of the ethylene vinyl acetate (EVA) foam shell will increase the shock absorption capability of the heel. The orthosis should have a deep heel cup and control the rearfoot to reduce long midstance pronation. A neoprene microcellular urethane foam (unless a diabetic), or other 3mm cushion top cover is suggested.

References


First Metatarsal–Medial Cuneiform Dorsal Exostosis

Amy Ellis, BA (Kin), C. Ped (C)

Key Messages

• First metatarsal–medial cuneiform dorsal exostosis may occur without symptoms while extensive degeneration can result in severe pain and loss of function.
• First metatarsal–medial cuneiform dorsal exostosis is associated with neuritis, tendonitis, bursae, swelling, and ganglion cysts.
• First metatarsal–medial cuneiform dorsal exostosis can be detected by palpating the dorsal joint margin. More detailed information can be obtained through radiographic assessment.
• Conservative treatment includes custom foot orthoses and footwear designed to stabilize and offload the midfoot; footwear must also accommodate dorsal prominence.
• First metatarsal–medial cuneiform dorsal exostosis is linked to midfoot instability and is commonly overshadowed by other complaints such as plantar fasciitis or hallux rigidus.

Keywords
arthritis, bony proliferation, bursa, degeneration, first ray, ganglion cyst, medial column, medial column hypermobility, osteophytes, tendinitis

Introduction

First metatarsal–medial cuneiform dorsal exostosis refers to the proliferation of bone at the dorsal joint margins of these joints (Michaud, 1997). As this condition progresses, the degenerative changes encompass progressively more of the articulating surfaces. The degeneration may be secondary to systemic diseases such as inflammatory arthritis or diabetes (and associated Charcot deformity). It may also result from the intermittent compressive forces that occur during stance and gait and is associated with both planus and cavus foot types (Banks, Downey, Martin & Miller, 2001). The first metatarsal–medial cuneiform joint can also develop post-traumatic arthritis.

Pathology

Primary degeneration of the first metatarsal–medial cuneiform joint is most commonly associated with lower arches (Rao, Nawoczenski & Baumhauer, 2008). If the foot remains pronated throughout propulsion rather than functioning as a rigid lever, the first ray goes through excessive ranges of dorsiflexion and inversion (Michaud, 1997). This causes sagging at the plantar aspect of the first metatarsal–medial cuneiform joint and compression of the dorsal surfaces (Rao et al., 2008). The first metatarsal–medial cuneiform, talonavicular, navicular–cuneiform, and first inter–cuneiform joints all contribute to motion of the medial column. Hence, isolated fusion of the first metatarsal–medial cuneiform joint can effectively treat hypermobility of
the medial column but sometimes additional joints must also be fused (Blitz, 2005). It is of interest to note that the first metatarsal-medial cuneiform joint must endure nearly twice as much compressive force as the other tarsal-metatarsal joints (Banks et al., 2001). Pes cavus feet are also prone to first metatarsal-medial cuneiform exostosis. If the forefoot is in a more plantar flexed position relative to the rest of the foot a jamming force may be transmitted through the first metatarsal to the medial cuneiform (Banks et al., 2001).

The symptoms of first metatarsal-medial cuneiform dorsal exostosis range from no discomfort to severe pain and loss of function (Banks et al., 2001). The amount of disability primarily depends upon the extent of degenerative changes within the joint plus the overall stability of the medial column (Rao et al., 2008). First metatarsal-medial cuneiform dorsal exostosis can be classified according to the degree of degeneration within the joint, and according to McGlamry’s Comprehensive Textbook of Foot and Ankle Surgery (2001), there are five types. In Type I there are dorsal osteophytes, but the remaining joint surface is unaffected. In Type II, the dorsal exostosis may extend circumferentially around the joint and there are also degenerative changes within the joint. In Type III, the dorsal exostosis and degenerative changes within the joint occur along with poor alignment (in any direction) of the first metatarsal. In Type IV, there are degenerative changes in multiple tarsal-metatarsal joints. In Type V, also referred to as pseudoexostosis formation, the high-arched foot is afflicted with a bony prominence at the apex of the arch, but without bone spurs. In this case, the high arch makes the dorsum of the foot appear more prominent and there may be signs of superficial irritation from footwear (Banks et al., 2001).

A first metatarsal-medial cuneiform dorsal exostosis can afflict the foot itself and cause a variety of associated conditions. This exostosis can lead to extensor hallucis longus tendonitis from rubbing against the sharp dorsal bone spur. It can result in acute pain superficial to the joint that can be incapacitating when the patient moves their foot (Banks et al., 2001). The medial dorsal cutaneous nerve branch and/or saphenous nerve can become compressed between the exostosis and footwear (Reyzelman, Fiorito, Hoover & Brewer, 2007). The deep peroneal nerve can also become entrapped between the dorsal exostosis and extensor hallucis brevis (Parker, 2005) or extensor digitorum brevis (Reyzelman et al., 2007). Hence, patients afflicted by first metatarsal-medial cuneiform dorsal exostosis may have neuritic symptoms including pain that shoots into the toes (Parker, 2005). In other cases, the first metatarsal-medial cuneiform dorsal exostosis may be accompanied with swelling (Banks et al., 2001), a ganglion cyst, or bursa; however, as first metatarsal-medial cuneiform dorsal exostosis is linked to midfoot instability (Blitz, 2005), it is often overshadowed by other complaints such as plantar fasciitis or hallux rigidus (Banks et al., 2001).

**Differential Diagnosis**

Ganglion cysts and bursae are both fluid-filled masses that are associated with dorsal exostoses of the various tarsal-metatarsal joints but can also occur in the absence of an exostosis. Either a ganglion cyst or bursa may compress the overlying nerves leading to neuritic symptoms (Banks et al., 2001). Tophi can also mimic a first metatarsal-medial cuneiform exostosis (Decker & Albert, 2002). Tophi are deposits of uric acid crystals that form nodules within various tissues of the body including the joints. These masses typically develop in individuals with an extended history of gout.

**Common Testing**

If a first metatarsal-medial cuneiform dorsal exostosis is present, there will be a palpable proliferation of bone along the dorsal joint margins. If the exostosis is associated with neuritic symptoms, pressing on the area may reproduce these (Decker & Albert, 2002). Depending upon the severity, the person may complain of aching deep within the joint (Banks et al., 2001). More detailed information regarding the extent of the degenerative joint disease can be obtained with radiographic assessment (Banks et al., 2001; Patel, Rao, Nawoczenski, Flemister, DiGiovanni, & Baumhauer, 2010).

**Contraindications**

Footwear that compresses or frictions the soft tissue overlaying the exostosis is contraindicated even if the shoe is otherwise suitable (i.e., supportive). Hence, it would be prudent to assess the patient’s current footwear and advise them to opt for shoes with sufficient volume and stability features and to avoid those which rub on the area. Laced footwear is generally more accommodating than slip-ons, particularly with the addition of an orthotic device.

**The Patient Perspective**

Even with a palpable dorsal exostosis, the patient
may be pain free. As the degeneration progresses, the patient may complain of aching deep within the joint. Neuritic symptoms may occur from compression of the overlying nerves; these symptoms may be aggravated by certain types of footwear, such as sandals with straps over the exostosis, or simply by footwear in general (Banks et al., 2001). Advanced degeneration of the first metatarsal–medial cuneiform may be associated with hypermobility and collapse of the medial column (Blitz, 2005). In this case, the patient will have extreme discomfort and disability. This is especially true when performing activities like walking and stair climbing (Patel et al., 2010).

**Common Treatment**

In feet that overpronate, the goal of treatment is to stabilize the midfoot. This requires the use of custom foot orthoses and supportive footwear. Footwear should be stiff–soled. Alternatively, carbon fibre plates can be worn under the orthoses to stiffen the shoes. This has been shown to decrease the average plantar pressure and reduce contact time of the medial midfoot. For more advanced metatarsal–medial cuneiform dorsal exostosis including degeneration within the joint, and possibly involving additional tarsal–metatarsal joint(s), one should consider footwear with rocker bottoms. More advanced cases of midfoot instability involving degeneration of one or more tarsal–metatarsal joints and medial column hypermobility may require ankle foot orthoses (Patel et al., 2010).

In the case of pes cavus feet, custom foot orthoses have shown to decrease plantar pressure in all areas of the foot. (Burns, Crosbie, Ouvrier, & Hunt, 2006). Pes cavus feet are prone to degeneration of the first metatarsal–medial cuneiform joint because the forefoot may be more plantar flexed relative to the hindfoot. The design of the orthosis will depend upon the specific deformity. A stiff plantar flexed ray, for example, should be treated with a first ray cut-out and/or a 2–5 bar post (Michaud, 1997). If a forefoot equinus deformity exists, the footwear prescription should include a raised heel relative to the forefoot (Branthwaite & Chockalingam, 2010).

Patients with tendinitis or neuritis may benefit from oral anti-inflammatories, corticosteroid injections, and physical therapy modalities (Banks et al., 2001). If conservative measures fail, the affected joint(s) may require surgical excision of the dorsal osteophytes (Banks et al., 2001) and fusion to limit/reduce painful motion and address midfoot instability (Patel et al., 2010).

Additional conservative treatments include padding the tongue of the shoe, avoiding sandals with straps that rub on the dorsal bump, and changing the method of shoe lacing (skip lacing) to avoid lace pressure across the tender site. Stretching or modifying the shoe may also be required to accommodate the dorsal prominence (Decker & Albert, 2002).

**Key Considerations**

First metatarsal–medial cuneiform joint exostosis is most commonly associated with excessive pronation and instability of the medial column (Rao et al., 2008). As such, conditions such as a hypermobile first ray or semi–rigid forefoot valgus deformity make a patient more susceptible to degeneration of this joint. Patients with pes cavus feet are also prone to first metatarsal–medial cuneiform joint exostosis; this is especially true if the patient has a plantar flexed first ray (Banks et al., 2001). Degeneration of the first metatarsal–medial cuneiform may eventually require surgical treatment, particularly if it is associated with medial column hypermobility (Banks et al., 2001; Patel et al., 2010). However, conservative treatment using a custom foot orthosis and appropriate modified footwear may suffice.

**References**


Tibialis Anterior/Extensor Tendinopathy/Tenosynovitis

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Key Messages

- Tibialis anterior injury is often a result of overuse. A detailed patient history often reveals a change in training regimen, resulting in increased demands of the muscle.
- Tibialis anterior tendinopathies usually present themselves with compensatory overpronation. Pedorthic treatment is thus geared towards reducing this abnormal midfoot movement.
- Extensor tendinopathy is rare. Minimal research supports prevalence, etiology, and treatment.

Keywords
overuse injury, paratenonitis, tendinitis, tendinopathy, tendinosis, tibialis anterior

Introduction

Overuse tendon injuries, commonly termed tendinopathies (Simpson & Howard, 2009), are common musculoskeletal injuries seen in a pedorthic clinic. The term tendinopathy “describes a spectrum of diagnoses involving injury to the tendon (e.g., tendinitis, peritendinitis, tendinosis)” (Simpson & Howard, 2009). These tendon injuries are often influenced by trauma, or, “exacerbated by abnormal function of the lower extremity” (Valmassy, 1996). Tendinopathic terms are often misused in clinical context, leading to a somewhat confusing terminology. Clarification of these terms follows.

- Tendinitis: The tendon, as primary area of pain, shows inflammatory features. This term is used in a clinical context, referring to the clinical syndrome, rather than the pathological condition.

- Tendinosis: May or may not be symptomatic. Degeneration is evident at the collagen fibre level.

- Paratenonitis (term includes peritendinitis and tenosynovitis): The paratenon suffers from acute edema and hyperemia. Inflammatory cells are present at the site of injury (Maffulli, Khan, & Puddu, 1998).

Differentiation between these terms is formally determined at a biopsy level but recent advances in ultrasound imaging enable clinical distinction (see Differential Diagnosis for additional details). In clinical context, the term “tendinopathy” describes the clinical syndrome, which is more relevant to the pedorthic clinician. A “clinical syndrome characterized by a combination of pain, swelling (diffuse or localized), and impaired performance should be labeled tendinopathy” (Maffulli, Khan & Puddu, 1998). These clinical syndromes commonly present themselves as overuse injuries to the tendon in question.

Extensor tendinopathy is extremely rare and minimally reported in the literature. Apart from a few case studies (Kobayashi et al., 2007; Lee et al., 2009; Ross, 2006), the literature lacks accurate prevalence rates, etiology, and supportive treatment protocols. Consequently, this chapter will focus on tibialis anterior overuse.

Tendinopathic injuries to the tibialis anterior (TA) tendon are most predominant in overweight women, between the ages of 50–70 (Varghese & Bianchi, 2014). While TA overuse has been reported across the literature, TA tendon dysfunction and/or complete rupture is rare. This chapter contains brief anatomical details, typical patient symptomology, and pedorthic treatment for tibialis anterior tendinopathic injuries and overuse clinical syndromes.

Pathology

The tibialis anterior is a superficial muscle of the anterior compartment of the lower leg. The muscle
originates on the tibia’s lateral condyle and the upper two-thirds of the tibial shaft and interosseous membrane (Porter & Schon, 2008). Tibialis anterior runs parallel to the tibia, and inserts into the inferior surface of the medial cuneiform and first metatarsal head making it the prime mover for ankle dorsiflexion. Secondary actions include inverting the subtalar joint, and supporting the medial longitudinal arch (Marieb, Mallatt, & Wilhelm, 2004). 

Locomotion speed (walking vs. running) alters the TA muscular demand and rate of fatigue (Tusji et al., 2015). During walking mechanics, the tibialis anterior muscle is most active immediately following heel strike. This muscle allows for a gradual lowering of the forefoot to the ground surface. During these early stages of the gait cycle, this muscle functions to decelerate ankle plantarflexion and “maintains an inverted position about the longitudinal midtarsal joint axis” (Michaud, 1997). At terminal stance, the tibialis anterior muscle assists in ankle dorsiflexion initiating the early swing phase. During late swing phase, the muscle produces “marked inversion of the forefoot about the longitudinal midtarsal joint axis” (Michaud, 1997) preparing for the following heel strike.

The average muscle activity of the tibialis anterior increases in running compared to walking (Tusji et al., 2015). Consequently, it is not surprising that TA tendinopathy is most common in runners. During running activities, the TA must concentrically dorsiflex the ankle in early swing, and provide sufficient ground clearance prior to the next initial contact (Novacheck, 1998). An increase in mileage or a change in running techniques is often discovered in the patient’s history. Running stairs and running uphill and/or downhill are additional activities resulting in increased demands of the muscle (Porter & Schon, 2008). Injury often results from a dorsiflexion force placed eccentrically on a resisted plantarflexed foot (Simpson & Howard, 2009). Recovery periods are necessary for our lower limb tissues to meet the increased training demands. Breakdown at the tissues cellular level is believed to be the consequence of inadequate rest to these tissues (Simpson & Howard, 2009). Additionally, “a tight gastrocsoleus [complex] also contributes to increased strain in the myotendinous unit” (Porter & Schon, 2008). In such circumstances, the muscular demands of the tibialis anterior greatly increase in dorsiflexing the ankle and controlling the forefoot during the swing phase of gait (Porter & Schon, 2008). Overuse of the tibialis anterior tendon is associated with increased supination in compensation for overpronation (Valmassy, 1996). In the case of highly pronated gait, there is an increased demand on the tibialis anterior muscle resulting in longer muscle contraction “attempting to reduce abnormal subtalar and midtarsal joint pronation” (Valmassy, 1996). In such instances, the tibialis anterior will aid in subtalar supination to avoid painful weight-bearing of the medial aspect of the foot.

Overuse injuries to the tibialis anterior tendon can be associated with the following foot types (Michaud, 1997):

- Partially compensated, or fully compensated forefoot varus: The forefoot is maintained in an inverted position relative to the rearfoot in a forefoot varus deformity. Consequently, the subtalar joint must overpronate through midstance and toe off in order to bring the medial forefoot to ground contact. This overpronation throughout the gait cycle results in excessive demands on the tibialis anterior musculature.

- Rigid forefoot valgus: As means of avoiding a “supinatory rock,” the subtalar joint is forced into rapid supination. These patients will often excessively dorsiflex the ankle at heel strike. This action delays forefoot contact with the ground, thus allowing a longer time period for the subtalar joint to pronate. By increasing natural shock absorption at heel strike, this compensatory action increases the demands of the tibialis anterior muscle. A prolonged heel strike results in a prolonged contraction of the muscle, as it lowers the forefoot to the ground.

- Flexible forefoot valgus: When this deformity is flexible, the forefoot compensates for the valgus position by inverting the foot along its midtarsal joint axis. However, with larger ranges of longitudinal midtarsal joint inversion, the excessive pronation can still be observed through all stages of the gait cycle. This deformity directly affects tibialis anterior in cases where the subtalar joint is not sufficiently resupinating at toe-off, demanding excessive contraction of the tibialis anterior muscle.

- Ankle equinus: The ankle joint must dorsiflex a minimum of 10 degrees to ambulate without a compensated gait pattern. If the gastrocsoleus complex does not allow sufficient dorsiflexion, the subtalar joint compensates with overpronation. This overpronation results in excessive demands of the tibialis anterior muscle.

- Abnormal subtalar joint pronation: Pathological conditions resulting in abnormal motion of the
subtalar joint results in greater demands of the tibialis anterior muscle, as it attempts to resupinate the foot.

**Differential Diagnosis**

Possible misdiagnoses are as follows, but are not limited to: exertional compartment syndrome; tibial stress fractures; and medial tibial stress syndrome (MTSS). If pain is present in multiple joints, a rheumatoid condition is worth considering (Simpson & Howard, 2009). Due to the TA’s superficial morphology, diagnostic ultrasound can screen for tendinopathy, and further confirm the location of tendon hypertervascularization. Positive TA tendinitis/tenosynovitis ultrasound results, typically reveal tendon and synovial sheath thickening, increased fluid uptake within the tendon sheath, and/or peritendinous subcutaneous edema (Varghese & Bianchi, 2014).

**Common Testing**

Non-weight-bearing muscle testing is composed of three different types: active range of motion, passive range of motion, and isometric resistive testing. Active range of motion consists of an active contraction of the muscle, performed by the patient. Patients are asked to actively dorsiflex and plantarflex the ankle. The examiner would ask the patient to “bring their toes towards their knee” and “point their toes away from the knee”. A patient suffering from TA tendinopathy will often experience pain with active dorsiflexion, and may experience pain with plantarflexion. Passive range of motion consists of the examiner moving the joint in the desired motion, rather than the patient. The examiner moves the patient’s ankle in both dorsiflexion and plantarflexion. Patients will often report no pain with passive dorsiflexion, however may experience pain with a passive plantarflexion stretch. Isometric resistive testing consists of the patient contracting the muscle without changing muscle length. When testing resisted dorsiflexion, the examiner would explain the following: “I am going to pull your toes downward, away from the knee—try not to let me do it.” The opposite would occur when testing resisted plantarflexion: “I am going to push your toes towards your knee—try not to let me do it.” With tibialis anterior injury, patients will often experience pain with resisted ankle dorsiflexion. When comparing muscle strength bilaterally, resisted dorsiflexion can further highlight any TA weakness. The leg with muscle weakness will have difficulty resisting the dorsiflexion force against the muscle.

Resistive plantarflexion should not cause any painful symptoms.

A toe-up test can be used to confirm the tibialis anterior muscle does not suffer from rupture/dysfunction. While weight-bearing, patients are asked to raise their forefoot off the ground. Patients with tibialis anterior rupture/dysfunction will be unable to perform this test.

**Contraindications**

Tibialis anterior dysfunction/rupture is extremely rare, however possible. The mechanism of injury can be spontaneous or a result of local trauma. Local trauma is more common within a younger population, whereas spontaneous rupture occurs in middle-aged to older patients. In cases of complete rupture, patients may not experience any pain. Immediate pain is common at the time of trauma, however the patient may only describe slight localized tenderness at the time of their clinical visit. On physical examination of a complete rupture, obvious indentation will be evident at the tendon site. If the tendon is partially torn, patients will report extreme pain upon palpatation of the tibialis anterior insertion. A detailed history on patients with spontaneous rupture often reveals a co-founding factor; “diabetes, inflammatory arthritis, gout, hyperthyroidism, obesity or steroid use” (Porter & Schon, 2008; Varghese & Bianchi, 2014). If a rupture is suspected, treatment should no longer continue. The patient should be advised to return to primary care.

Patients suffering from a stress fracture may or may not report night pain. Furthermore, a patient’s history may be very similar if they are suffering from tendinopathy versus a stress fracture. The red flag to immediately stop treatment is apparent during physical examination. Upon palpation, localized bony tenderness should trigger the suspicion of a stress fracture. It’s important to note that a stress fracture may not affect joint range of motion, and localized swelling may not be present (Porter & Schon, 2008).

If history details alerts a suspicion of rheumatoid arthritis or gout, the patient should be encouraged to return to primary care. Patients suffering from rheumatoid arthritis will often describe flare-ups of pain and swelling, followed by a remission period. Stiffness upon waking is very common. Joint swelling is often symmetric and found in multiple joints of the body (Lorimer, French, O’Donnell, Burrow, & Wall, 2006). Patients suffering from gouty arthritis will typically describe a rapid onset of pain. During a
flare-up period, the affected joints are marked with heat, swelling, and redness. X-ray imagery reveals excessive crystal deposits within the synovial joints (Lorimer et al., 2006).

**The Patient Perspective**

Overuse injury to the tibialis anterior muscle initially presents itself with insidious onset of pain, often during prolonged activity (see contraindications if rupture is suspected). In the early stages of the condition, patients will describe minimal pain during the “warm-up” of their activity, with minimal, if no, pain afterwards. In the later stages of the condition, patients will observe pain during and following activity. Pain relief is only found with prolonged rest. Commonly reported symptomology includes “dull” pain at rest, but “sharp” pain during activity (Simpson & Howard, 2009). Pain “deep in the joint” may also be reported. Anterior ankle pain can present itself during running activity, which may continue for a few hours following activity. On physical examination, pain is often reported with palpation along the tendon. Inflammation and palpable crepitus may be additionally observed (Porter & Schon, 2008). A decreased range of ankle dorsiflexion may be observed on the affected limb (Simpson & Howard, 2009). Pedorthists may additionally notice a flexible plantarflexed first ray in patients with tibialis anterior weakness. A plantarflexed first ray can be caused by weakness in the tibialis anterior muscle, providing a mechanical advantage to the peroneal longus muscle. If weakened, the tibialis anterior cannot dorsiflex the first ray–to–ground contact (Michaud, 1997).

Typical distal tendinopathy symptoms include burning on the medial side of the midfoot, commonly reported in the evening. Injury is often spontaneous in nature and symptoms present themselves unilaterally. Palpation over the TA’s distal insertion will often reproduce pain. Typically, there is no loss in muscle strength, however active ankle dorsiflexion may be limited. (Varghese & Bianchi, 2014).

**Common Treatment**

Pedorthic treatment is targeted towards decreasing the muscular demands of the tibialis anterior muscle when abnormal foot function exacerbates the overuse injury. Custom orthoses for these patients are designed to control the excessive pronation, which has proven effective in minimizing the duration of tibialis anterior activity in single stance (Dedieu et al., 2013). Specific orthotic design features are determined based on clinical findings. If a forefoot deformity is revealed during assessment, treatment is based on the rigidity of the condition. A rigid forefoot varus is treated with an accommodative varus forefoot post. A flexible forefoot varus is treated by supporting the medial longitudinal arch and decreasing midfoot pronation. No forefoot posting is necessary. A valgus forefoot post is used to treat both a flexible and rigid forefoot valgus deformity. If a flexible rearfoot valgus is determined at assessment, a varus rearfoot post is used to control the excessive pronation (Michaud, 1997).

Supplementary to orthoses treatment, stability footwear can further minimize midfoot pronation. As vamp irritation can lead to tendon irritation (Valmassy, 1996), lacing techniques can decrease pressure over tender area. A “skip lacing” technique is a common and effective lacing change to minimize pressure over the dorsal midfoot.

During the acute stage of injury, ice may be placed over the area to decrease inflammation and pain (Porter & Schon, 2008). In more severe cases, additional treatment may be required. Walking casts during weight-bearing (Simpson & Howard, 2009) are effective offloading devices. There are contradictory findings on the efficaciousness of steroid injections decreasing severe pain (Porter & Schon, 2008; Simpson & Howard, 2009). If a rupture is suspected, an orthopaedic consult is suggested.

Overuse injuries can benefit from massage therapy and physiotherapy. Referrals to these professionals are great compliments to pedorthic treatment. An important note: Similar offloading principles can be applied if a pedorthist suspects an overuse injury to extensor musculature.

**Key Considerations**

- Tibialis anterior and extensor tendinopathies are rare, with minimal research supporting best practice.
- Any physical activity that places excess stress on the musculature can increase the likelihood of an overuse clinical syndrome.

**References**

Tibialis Anterior/Extensor Tendinopathy/Tenosynovitis
Cuboid Syndrome
Freeman Churchill, BSc, BPE, C. Ped (C)

Key Messages
• Cuboid syndrome is a painful articular injury located on the lateral midfoot between the calcaneus, base of the fourth and fifth metatarsals, and the third cuneiform bone.
• Occurs frequently after lateral or inversion ankle sprains, and in cavus feet that have a plantar flexed first ray.
• Occurs with significant rearfoot pronation when there is stress on tissues surrounding the calcaneal cuboid midtarsal joint.
• Cuboid syndrome must be correctly identified and not confused with other conditions related to the same area and pain presentation.
• Prognosis for treatment, when identified, is very good.

Keywords
interosseous ligaments, inversion sprain, peroneus longus, point tenderness, pop, subluxation

Introduction
Cuboid syndrome is a very specific articular pathology that exists when the cuboid bone is out of normal or desirable articular congruity or integrity with the calcaneus, navicular, fourth and fifth metatarsal bases, and the third or lateral cuneiform. Left uncorrected, a “subluxed cuboid” may lead to arthritic changes within articulating structures, peroneus longus tendinitis, and compensated gait patterns with potential for further pathologies associated with gait.

Cuboid syndrome is a common condition of the lateral midfoot that is commonly misdiagnosed and mistreated. It is also known as subluxed cuboid, peroneal cuboid syndrome, dropped cuboid, faulty cuboid syndrome, and lateral plantar neuritis.

Cuboid syndrome is very common in people who have suffered an inversion sprain or multiple inversion sprains. Sports that sustain more inversion type injuries include soccer, volleyball, basketball, and jazz and ballet dancing. Severe inversion sprains interrupt and injure lateral foot and ankle ligaments and tendons. Another suggested mechanism is that the inversion stress at the ankle may cause a reflex contraction of the peroneus longus muscle in an attempt to restore balance and medial ground contact. This forceful contracture of the peroneus longus muscle creates a medial rotation of the cuboid. Then the peroneus longus tendon — within the osseous fibrous tunnel on the inferior surface of the cuboid — imparts a dorsal and lateral force on the cuboid, which results in the inferomedial subluxation.

Repeated inversion injuries to the ankle increase the wear and tear on the joint capsule and ligaments around the cuboid, increasing frequency in the number of episodes of cuboid subluxation.

Pathology
Posture and function of the cuboid bone is greatly affected by the peroneus longus, the extrinsic plantar flexor, and the forefoot evertor of the foot. The peroneus longus originates in the lateral proximal fibula, runs down the fibula posterior to the lateral malleolus, and wraps under the cuboid in the peroneal groove of the cuboid. It then runs obliquely across to the base of the first metatarsal and first cuneiform (Blakeslee & Morris, 1987).

It is theorized that there are two main causes for cuboid syndrome, one being the resultant interruption of stability and normalcy following a plantar flexion inversion sprain of the ankle. The second, and less commonly reported, is from overuse, primarily from excessive repetitive subtalar pronation (Patterson, 2006).
As the foot and ankle are placed into plantar flexion and inversion, the peroneus longus tendon places a dorsal and lateral force on the forefoot, creating a close-packed position and forcing the cuboid in an inferomedial direction, tearing the interosseous ligaments (Caselli & Pantelaras, 2004). In this scenario, the actual acute trauma is thought to be responsible for the disruption of the cuboid position (Jennings & Davies, 2005).

The stretch reflex is another theory regarding plantar flexion and inversion ankle sprains (Blakeslee & Morris, 1987; Caselli & Pantelaras, 2004). When the foot and ankle are forced rapidly into plantar flexion and inversion, there is a reflex contraction of the peroneus longus muscle, attempting to get the medial aspect of the foot to the ground (Blakeslee & Morris, 1987; Caselli & Pantelaras, 2004). During the forceful contraction of the peroneus longus, the cuboid is used as a fulcrum to increase the mechanical advantage, causing a medial rotation (Caselli & Pantelaras, 2004). The peroneus longus, once again, exerts a dorsal and lateral force on the cuboid, hypothetically resulting in inferomedial displacement of the cuboid (Caselli & Pantelaras, 2004). More specifically, the pathomechanics of cuboid syndrome may stem from an eversion of the cuboid from an inverted foot position, such as the mechanism of injury for a lateral ankle sprain (Jennings & Davies, 2005; Subotnick, 1989).

Several factors may increase the likelihood of cuboid syndrome, including midtarsal instability, excessive body weight, ill-fitting or poorly constructed orthoses or shoes, exercise (i.e., intensity, duration, and frequency), inadequate exercise recovery, training on uneven surfaces, and sprain of the foot or ankle. Cuboid syndrome may be more prevalent in individuals with pronated feet due to the increased moment arm of the peroneus longus. In one study, 80% of the patients with cuboid syndrome presented with pronated feet, but it can also occur with pes cavus (supinated) feet.

It is theorized that when the subtalar joint is neutral and the forefoot is pronated, the midtarsal joints are most stable. When the subtalar joint is pronated, the forefoot is inverted relative to the rearfoot and the midtarsal joint is unlocked, enabling the foot to adapt to uneven surfaces and act as a shock absorber at ground contact. The supination that occurs at the subtalar joint decreases both supination and pronation at the midtarsal joint, thus creating a more stable midfoot during the propulsive phase of gait (Blakeslee & Morris, 1987). Biomechanically, the line of pull exerted by the intrinsic musculature plays an important role in the dynamic stabilization of the calcaneocuboid or midtarsal joint (Mann & Inman, 1964).

Every degree of subtalar pronation that occurs produces an exponential increase in midtarsal joint instability (Blakeslee & Morris, 1987). If the calcaneocuboid joint is not completely congruous, the excessive ground reaction forces occurring during the propulsive phase of gait overwhelm the soft tissues surrounding the cuboid, predominantly the joint capsule and ligaments that secure the cuboid in the lateral column. Theoretically, this can lead to varying degrees of subluxation or cuboid displacement (Blakeslee & Morris, 1987).

**Differential Diagnosis**

Cuboid syndrome is commonly mistaken for cuboid stress fracture, inversion sprain, tarsal coalition, Jones fracture, fracture of the anterior calcaneal process, peroneal and extensor digitorum brevis tendinitis, subluxing peroneal tendons, sinus tarsi syndrome, lateral planter nerve entrapment, Lisfranc injuries, meniscoid of the ankle, and malalignment of the lateral ankle and subtalar joints (Caselli & Pantelaras, 2004; Dewar & Evans, 1968; Jennings & Davies, 2005; Leerar, 2001; Main & Jowett, 1975; Phillips, 1985).

**Common Testing**

The diagnosis of cuboid syndrome is based primarily on the patient’s history and findings during the physical examination (Blakeslee & Morris, 1987; Jennings & Davies, 2005). X-rays, CT scans, and MRIs are useful in ruling out other injuries that may mimic cuboid syndrome. The degree of subluxation of the cuboid is usually so minute that it is very difficult to use imaging tools for differential diagnosis (Blakeslee & Morris, 1987; Marshall & Hamilton, 1992; Mooney & Maffey-Ward, 1994; Newell & Woodle, 1981).

**Contraindications**

If the patient has a confirmed subluxed cuboid, running, jumping, and dance activities should be stopped until its congruity has been reestablished either through manipulation or physical or manual therapy to establish normal muscle function. Athletes should not try to “run through” this injury for fear of distressing articular structures and possibly injuring other related structures through compensation. Normal flex patterns of the fourth
and fifth metatarsal bones may be reduced such that metatarsal fractures become a possibility.

**The Patient Perspective**

Patients suffering from cuboid syndrome will describe a direct point tenderness or boney ache directly on the cuboid. It may be sore to touch. There may be bruising, redness or swelling around the cuboid. Soft tissues around the cuboid may also be swollen. Pain may radiate distally to the fourth and fifth metatarsal bases and posteriorly to the calcaneal cuboid articulation. It may also be painful on the plantar surface of the foot directly under the cuboid. Patients may describe the cuboid areas as painful to walk, run, or bear weight. Pain may also be present at rest or in bed. Transferring weight from the hindfoot to the forefoot may be particularly painful as the calcaneal-cuboid articulating gliding motion may be restricted, creating an asymmetry in gait. The opposite, unaffected foot has motion during the transfer from midstance to heel-off that the affected foot does not.

**Common Treatment**

Successful treatment for cuboid syndrome is best achieved through a combined approach of pedorthic care, to provide a corrective and support environment for the foot, and manual therapy to address joint incongruity and misalignments. The pedorthic care may include: comfortable stable footwear, corrective functional orthoses, taping, or strapping to support the cuboid and medial long arches. Appropriate manual therapy can be provided by modalities such as chiropractic care, physiotherapy, athletic therapy, and osteopathy to restore desirable joint congruity to the calcaneal-cuboid, cuboid, metatarsal, and cuneiform structures.

To date, there is a lack of research or statistical data to support the claim that foot orthoses will prevent cuboid syndrome; however, there appears to be a clinical consensus that the use of taping, foot orthoses with soft cuboid padding, and proper fitting of therapeutic footwear can be helpful in the treatment of a pre-existing condition. Emphasis points to reducing excessive inversion of the rearfoot while the forefoot everts throughout the midstance and heel off phase of propulsion.

Footwear should be stable and structured in the rearfoot and midfoot to support the foot, yet not so supportive that it overcorrects in combination with an orthosis. It may be necessary to avoid motion-controlling footwear. There should be adequate room and flexibility in the forefoot for normal extension and dorsiflexion of the metatarsal heads.

Orthoses should be designed to address the biomechanical abnormalities of the foot, while helping reduce recurrent ankle sprains by supporting the lateral column of foot. A cuboid pad should be placed directly beneath the cuboid, avoiding being placed under the calcaneus posteriorly, or the fourth and fifth metatarsal base anteriorly. Forefoot balance should be such that it allows normal function of the first ray and prevents abnormal loading of the first ray.

Physical therapy should include restoring desirable or normal musculoskeletal balance of the peroneals, gastrocnemius, and soleus. Simple joint manipulation that does not address the soft tissue component may result in the cuboid quickly sliding back “out” of proper alignment. During the course of treatment it is important that the patient wears orthoses and footwear which help maintain an appropriate articulation with the cuboid. It may help to use a soft pad or support immediately beneath the cuboid to maintain optimal positioning while soft tissue changes occur and strength and flexibility are established.

**Key Considerations**

- Cuboid syndrome is very common in people who have suffered inversion sprains or repeated inversion sprains.
- Interestingly, in ballet dancers, there was a greater incidence with male dancers suffering from cuboid syndrome than female dancers.
- It can occur in virtually all foot types (i.e., feet that either supinate or pronate).

**References**


Peroneal (Fibularis) Tendinopathy

Paul Lucas, C. Ped (C)

Key Messages
• Peroneal tendinopathy usually presents as pain posterior and/or inferior to the lateral malleolus.
• It is often associated with inversion instability and with a cavovarus hindfoot.
• Usually caused by either single traumatic inversion injury, or by overuse (e.g., with runners and ballet dancers).
• Pedorthic treatment is aimed at controlling or reducing inversion with footwear, custom modifications to footwear, and/or orthoses.

Keywords
cavovarus hindfoot, hindfoot pain, inversion instability, lateral malleolus, ligament injury, peroneus brevis insertion, repetitive strain, weak ankles

Introduction
Peroneal tendinopathy presents as lateral foot pain, typically exertion influenced and often without a specific antecedent injury (Alexander, 1990; Roster, Michelier, & Giza, 2015; Taljanovic, Alcala, Gimber, Rieke, Chilvers, & Latt, 2015). It involves pathology of the peroneus longus and/or peroneus brevis tendons. It more often affects one of the peroneal tendons, but can involve both (Alexander, 1990; Taljanovic et al., 2015). It can be chronic, typically with foot types that remain inverted at stance (Brandes & Smith, 2000) or acute, often caused by a specific traumatic inversion incident. In either case it occurs when one or both of these peroneal tendons are overloaded (Molloy & Tisdel, 2003; Selmani, Gjata, & Gjika, 2006). The peroneal muscles stabilize the foot in weight-bearing and prevent it from rolling too far into inversion, particularly on uneven surfaces (Alexander, 1990). Peroneal tendinopathy is a common cause of lateral hindfoot pain and dysfunction (Molloy & Tisdel, 2003; Taljanovic et al., 2015), more specifically with pain posterior or distal to the lateral malleolus along the course of the peroneal tendons (Wilder & Sethi, 2004). It is relatively uncommon in comparison to tendinopathies of the Achilles or posterior tibial tendons (Wilder & Sethi, 2004), and is also less common than the ubiquitous lateral ankle ligament sprain (Fallat, Grumm, & Saracco, 1998; Molloy & Tisdel, 2003). It can be an often-overlooked cause of persistent lateral ankle pain and chronic ankle instability in overuse situations or after trauma (DiGiovanni, Fraga, Cohen, & Shereff, 2000; Karageanes, 2017). Pedorthic management is a key component of the treatment regime for this condition and is primarily aimed at controlling inversion and reducing traction on the affected tendon.

Pathology
Peroneal tendinopathy is a degenerative process involving the peroneus longus and/or brevis tendons (Dirks & Warden, 2011). It more commonly affects one, but can affect both tendons (Alexander, 1990). Notably, the peroneus tertius is absent in roughly 10% of the population (Sarrafian, 1983). Its absence has been found to be clinically insignificant with regard to both risk of ankle injury and to dorsiflexion/eversion strength (Witrouw, Vanden Borre, Willems, Huysmans, & Broos, 2006). Peroneal tenosynovitis may have a similar presentation, and can affect both tendons as they share a common sheath passing behind the lateral malleolus (Ardizzone & Valmassy, 2005; Shewmaker, Guderjahn, & Kummer, 2016). While overall patient management will vary based on the distinction between tendinosis and tenosynovitis, for pedorthic purposes this distinction will not significantly affect our management.

Peroneal tendinopathy is often associated with lateral
ankle instability (DiGiovanni et al., 2000) as the muscles can be overtaxed in attempting to stabilize the joint. Chronic injuries typically result from repetitive strain, as commonly seen with runners and ballet dancers (Heckman, Gluck, & Parekh, 2009; Karageanes, 2017). Acute injuries usually result from an inversion injury, often with involvement of the lateral collateral ankle ligaments (Karageanes, 2017; Selmani et al., 2006; Ziai, Benca, Wenzel, Schuh, Krall, Auffahrt, Hofstetter, Windhager, & Buchhorn, 2016). During an inversion injury, the peroneal muscles can contract forcibly in an attempt to evert the foot and prevent injury. This can in fact result in a small tear to one of the tendons. It is possible that the tendinopathy may not be recognized until some time later after the coinciding ligament injury has been healed and normal activity resumed (Ardizzone & Valmassy, 2005).

There are three common sites of pathology: one on the brevis tendon as it bends around the lateral malleolus; and two on the longus tendon, the first as it winds around the malleolus and the second as it turns around the cuboid bone on its way to the base of the first metatarsal (Peterson, Bobka, Stein, & Tillman, 2000; Shubert, 2013). These sites correspond to areas of poor vascularization of the tendons (Peterson et al., 2000), and as a result may explain why this condition can be slow to heal. There can also be insertional tendinopathy, most commonly at the brevis insertion on the base of the fifth metatarsal (Koepsel, 2002).

Several anatomical variants are associated with an increased incidence of peroneal tendinopathy. A low-lying peroneus brevis muscle belly (Geller, Lin, Cordas, & Viera, 2003) or the presence of an accessory peroneus quartus muscle (present in approximately 7% of the population) (Zammit & Singh, 2003). Both of these enlarged or additional structures further restrict space as the tendons pass under the retinaculum (Baumhauer, Nawoczenski, DiGiovanni & Flemister, 2004) thereby increasing irritation to the tendons. Also, hypertrophy of the peroneal tubercle increases mechanical irritation of the tendons (Hyer, Dawson, Philbin, Berlet, & Lee, 2005; Taljanovic et al., 2015). A flat or convex retromalleolar groove, and an accessory bone on the longus tendon called the os peroneum, have also been associated with higher incidence of peroneal tendinopathy (Taljanovic et al., 2015; Schubert, 2013).

Peroneal tendinopathy is also far more commonly seen in those with a cavovarus hindfoot alignment. One study reported 82% of cases of peroneal tendinopathy patients having significantly cavovarus hindfeet (Brandes & Smith, 2000). Forefoot valgus deformity and a rigid plantar flexed first ray can result in the foot functioning in a more inverted position, hence predisposing to peroneal injury (Ardizzone & Valmassy, 2005). As mentioned, the peroneal tendons can be injured with excessive or prolonged inversion as they are subjected to undue traction and strain. Injuries to the peroneal tendons can also be commonly seen alongside fractures of the calcaneus or lateral malleolus (Heckman et al., 2009).

While one might typically associate peroneal tendinopathy with the cavovarus foot, severe pes planus or feet with a valgus hindfoot can also be prone to injury resulting from impingement of the tendons between the fibula and everted calcaneus as they curve below the lateral malleolus (Karageanes, 2017). Similarly, peroneal tendinopathy may occur following an inversion ankle injury as the subject compensates by contracting the peroneals to guard against further ligament injury (Ardizzone & Valmassy, 2005). Ziai et al. (2016) found a correlation between peroneal tendinosis and ankle sprain trauma.

In cases of unilateral peroneal tendinopathy the clinician should consider the possibility of a leg length discrepancy. The foot of the shorter limb will often function in a more inverted position in an attempt to elevate the talus and equalize the leg lengths (Ardizzone & Valmassy, 2005). In doing so, it may place the shorter limb at risk of injury.

Peroneal tendinopathy typically results in pain posterior or distal to the lateral malleolus as well as instability of the affected ankle. Symptoms can result in reduced function, particularly in sport and dance settings, as well as predispose to injury of the lateral ankle ligaments (DiGiovanni et al., 2000; Molloy & Tisdal 2003).

**Differential Diagnosis**

Peroneal tendinopathy is most commonly confused with lateral ankle ligament injuries, and often can occur in conjunction with injuries to the anterior talofibular and calcaneofibular ligaments (Karageanes, 2017). One must also rule out small fractures to the calcaneus and lateral malleolus, as well as to the base of the fifth metatarsal (Heckman et al., 2009). The os peroneum is an accessory bone located in the peroneus longus tendon in approximately 20–30% of the population (Karageanes, 2017; Schubert, 2013). A fracture of the os perineum can sometimes mimic tendinopathy symptoms (Sobel, Pavlov, Geppert,
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Peroneal (Fibularis) Tendinopathy

Thompson, & DeCarlo, 1994). Other conditions that may appear with similar presentations are a subluxed peroneal tendon and lateral subtalar joint osteoarthritis (Ardizzone & Valmassy, 2005; Karageanes, 2017). Calcaneofibular impingement syndrome may also involve impingement of the peroneal tendons between an extremely everted calcaneus and the fibula, resulting in similar symptoms (Karageanes, 2017), albeit with quite different pathomechanics than what one might typically see in chronic peroneal injury.

Common Testing

Patients will typically present with pain and tenderness along the course of the peroneal tendons, most commonly posterior and inferior to the malleolus or as the longus passes under the cuboid (Peterson et al., 2000). Pain may be elicited with stretching of the peroneals. This is done with passive inversion, adduction, and dorsiflexion of the foot (Alexander, 1990). Pain may also be elicited with active eversion, abduction, and plantar flexion, with the pedorthist resisting motion (one hand stabilizes the leg above the ankle while the other hand applies resistance by holding the lateral forefoot) (Alexander, 1990). Contracting the tendons will also make them taut and easier to palpate for tenderness. The brevis is easily palpable between the tip of the malleolus and the base of the fifth metatarsal and the base of the fifth metatarsal. The longus can be identified with tenderness between the tip of the malleolus and the groove in the cuboid just proximal to the base of the fifth metatarsal, or from the plantar aspect of the cuboid to its insertion at the base of the first metatarsal (this segment can be made taut with plantar flexion of the first ray) (Alexander, 1990). Tenderness on the tendon will support a diagnosis of tendinopathy. In some cases there may be a palpable thickening of the affected tendon. Swelling is not common, but with more proximal tendon involvement, there may be loss of concavity behind the lateral malleolus (Alexander, 1990).

Bilateral strength testing of the peroneals can be performed in the same manner; the clinician provides resistance against eversion/abduction/plantar flexion of the foot. Weakness of these muscles can predispose to injury.

With a cavovarus hindfoot, the pedorthist should also perform a Coleman block test to assess flexibility of the deformity (Coleman & Chestnut, 1977).

With unilateral peroneal tendinopathy the clinician should assess for a leg length difference (Ardizzone & Valmassy, 2005), as the foot of the shorter limb may function in a more inverted position and be prone to injury.

Contraindications

Contraindications will be subject to each patient’s individual alignment and biomechanics. With a typical cavovarus foot type, any pedorthic treatment which results in pushing the foot further into varus, such as medial heel wedges, excessive medial arch support, medially posted orthoses or medially posted footwear, would be contraindicated as it would increase strain on already damaged peroneal tendons. Walking barefoot and wearing flat heels can increase load on the tendons and should be avoided. Training on uneven surfaces, or running on the sides of graded roads can increase strain on the peroneal muscles (of the foot on the lower side of the grade) and should be discouraged, as should sports requiring rapid lateral movement which may demand sudden, strong contraction of the peroneals.

The Patient Perspective

The patient with peroneal tendinopathy will typically describe pain behind and below the lateral malleolus, possibly as far distal as the base of the fifth metatarsal. They will also commonly report a feeling of ankle instability or “weak ankles” (Karageanes, 2017). They may have no definite antecedent traumatic episode (Alexander, 1990), or they may attribute pain to an inversion injury.

Common Treatment

Pedorthic treatment should be aimed at the patient’s individual pathomechanics, but typically treatment will be aimed at reducing traction on the peroneal tendons by controlling inversion or correcting the foot into some degree of eversion. In very mild or recent onset cases this might be achieved with lateral heel wedges in the shoes (Heckman et al., 2009). With increasing severity and persistence, treatment options could include external footwear modifications such as a lateral flare or buttress, or lateral sole wedging. Increased dorsiflexion places strain on the peroneals. While this might lead one to consider heel lifts to help reduce strain, introducing heel lifts will predispose to inversion instability (Bonnel, Toullec, Mabit, & Tourne, 2010) and further tendon injury. Dorsiflexion night–splinting may aid healing as the peroneals are secondary plantar flexors of the ankle. Restricted dorsiflexion is a risk factor with inversion.
 installations (Willems, Witrouw, Delbaere, Mahieu, & DeBourdeaudhuij, 2005).

As mentioned, medially posted footwear should be avoided. Shoes with wide lateral bases and firm heel counters should be favoured to prevent lateral breakdown and to stabilize the hindfoot. Motion control shoes designed to inhibit pronation are contraindicated, and cushioned shoes with lower durometer midsoles should also be avoided as they may compress laterally with a varus foot type.

Orthoses should be designed with the underlying foot type and mechanics in mind, but supporting the lateral column of the foot should be common to any orthosis with this diagnosis (Ardizzone & Valmassy, 2005). One study (Moisan & Cantin, 2016) found that orthosis with a lateral bar resulted in reduced peroneus longus activity during midstance and terminal stance. A cavovarus deformity should be posted laterally as tolerated to limit inversion. A rigid forefoot valgus should be managed with a lateral forefoot post to bring the hindfoot into straighter alignment. A plantar flexed first ray should be accommodated to permit eversion of the hindfoot (Coleman & Chestnut, 1977). A deep heel cup will assist in stabilizing and controlling the hindfoot, and a high lateral flange may assist in preventing the foot from inverting off of the foot bed. In some cases the practitioner may want to cast the foot in a pronated position and then post the orthosis to stabilize the foot in this pronated alignment (Ardizzone & Valmassy, 2005). Care should be exercised here not to overload medial structures (e.g., posterior tibialis tendon, medial ankle ligaments, and medial band of plantar fascia) or to adversely affect structures above the foot such as the knee and patellofemoral joints.

Ankle braces designed to provide inversion stability can be helpful in partially unloading the peroneals (Karlsson & Andreason, 1992). In more severe cases, the foot and ankle can be immobilized with a short walking cast or a CAM walker (Heckman et al., 2009).

Other treatments may include NSAIDs, physiotherapy, and activity restriction. If symptoms persist beyond three to six months, a surgical consult should be considered (Heckman et al., 2009).

**Key Considerations**

Peroneal tendinopathy is most typically seen in overuse situations, particularly after periods of inactivity (Molloy & Tisdel, 2003). This can be seen in sports settings where training is increased or changed, or in return to work settings where undue strain is placed on the tendons due to the nature of the job or the individual’s malalignment.

Cavovarus hindfeet, valgus forefeet and rigid plantar flexed first ray deformities will all tend to result in a more inverted foot and place load on the peroneals (Ardizzone & Valmassy, 2005; Brandes & Smith, 2000). In the absence of these mechanics, the clinician should also consider work or training surfaces that might force the foot into inverted positions, such as uneven terrain, the lower leg on a graded road, sloped roofs for roofing contractors, etc.

Age is also a predisposing factor, as with any tendinopathy (Almekinders & Temple, 1998), as is significant weight gain, with greater load being placed on the lower limb.

One may also consider a leg length discrepancy in unilateral presentations. The foot of the shorter limb will tend to supinate to elevate the talus, and in doing so place strain on the peroneals (Ardizzone & Valmassy, 2005).

If the foot is actively inverted to compensate for a more medial or pronation induced injury such as plantar fasciitis, posterior tibial tendon dysfunction, or sesamoiditis, this may eventually lead to excessive strain on the peroneals as well.

**References**


Retrocalcaneal Bursitis

Jill Rick, HBPE, C. Ped (C) (2012)
Updated by Jennifer Gould Andrew, BSc (Kin), C. Ped Tech (C), C. Ped (C) (2018)

Key Messages

• The retrocalcaneal bursa shares tissues and therefore pathology of the bone (Haglund’s exostosis) and tendon (Achilles tendinopathy) and therefore is part of a triad of conditions.
• Differential diagnosis to identify which or any of the closely related conditions will help in treatment and also in avoiding contraindications.
• Prevention is key, with gradual introduction to possible overuse activities such as running on an incline and avoiding poorly fitted and functioning shoes, especially at the heel counter.
• Retrocalcaneal bursitis is tender on palpation specifically with the two-fingered pinch test, anterior to the Achilles tendon, closely superior to its insertion.
• Treatment is most commonly non-surgical and includes ice, rest, flexibility, strength, orthoses, and discontinuation of irritating factors such as overuse activities and unsuitable footwear.

Keywords

Achilles, bursa, calcaneal tuberosity, exostosis, Haglund’s, inflammation, Kager’s, spongy

Introduction

History

The history of RB needs to be extracted from the literature, which identifies a spectrum of pathologies of the heel and the Achilles tendon. It was first identified by Albert in 1893 as “Achillodynia,” although Rossler later identified the cause more specifically as a bursitis in 1895 (van Dijk, van Sterkenburg, Wegerinck, Karlsson, & Maffulli, 2011). Described in much of the literature as one part of a spectrum disorder, RB has been included in the terms: Achilles tendon disorders, heel pain, bursitis, winter heel, Albert disease, calcaneus altus, pump bump (coined in 1954), cucumber heel, high-prow heel, knobby heel, prow beak deformity, tendo-Achilles bursitis, and hatchet-shaped heel (Aaron, Patel, Kayiaros & Calfee, 2011; Kachlik, Baca, Cepelik, Hajek, Mandys, Musil, & Stingl, 2008; Leitze, Sella, & Aversa, 2003; van Dijk et al., 2011). A universal terminology has not yet been identified regarding the pathologies affecting the area of the Achilles insertion and the posterior portion of the os calcis, or calcaneus, so inconsistencies in uses of the various terms are found throughout the literature (Kachlik et al., 2008; van Dijk et al., 2011).

One term of importance, because of its continued association with RB, is Haglund’s syndrome (also known as Haglund’s disorder, disease, exostosis, or deformity). It was originally defined as Haglund’s disease in 1928 by Patrick Haglund (Leitze et al., 2003) and renamed Haglund’s syndrome in 1982 (van Dijk et al., 2011). The term is used to refer to an exostosis, or bony prominence, at the posterior lateral aspect of the calcaneal tuberosity, and was originally described as an isolated condition caused primarily by friction from a low, rigid heel counter (e.g., a women’s pump) in combination with certain biomechanics of the foot and leg.

Haglund’s syndrome is now more commonly (but not universally) used to refer to a triad of conditions including RB, superficial Achilles bursitis, and/or Achilles insertional tendinopathy due to the close association and commonly co-existing pathologies of these disorders (van Dijk et al., 2011).

For the purpose of this discussion, “Haglund’s exostosis” will refer to the pathological bony protrusion of the supero-posterior lateral aspect of the calcaneal tuberosity, and “Haglund’s syndrome” will refer to the triad of RB, Achilles tendinopathy, and superficial Achilles bursitis.

One of the limitations in the literature is a detailed anatomical study of the retrocalcaneal bursa (RB) specifically. Reference to the RB as a separate
anatomical and pathological consideration with
detailed micro- and macroanatomy has been
significantly limited. Although the end of the
19th century brought studies on the pathology
and treatment of Achilles enthesopathies,
there has since been limited information on the
normal retrocalcaneal bursa, its detailed macro/
microanatomy, and its histology in the normal, non-
pathological state (Kachlik et al., 2008).

Definition

A bursa is a synovial fluid–filled sac that acts as a
cushion between bone and soft tissue. At a joint,
where hard bony tissues move next to softer, more
mobile tissues such as muscle, tendon, and skin, the
bursae help to protect these surfaces from shearing
and irritation (Michaud, 1993; National Institute
of Arthritis and Musculoskeletal and Skin Disease
[NIAMS], 2014). RB is defined as an inflammation
specifically of the retrocalcaneal bursa situated
supero-posterior to the calcaneal tuberosity and
anterior to the Achilles tendon, just superior to its
insertion (Aaron et al., 2011; Kachlik et al., 2008;
Michaud, 1993; Pedowitz, Ökereke, Lotke, Abboud, &
Ende, 2008; van Dijk et al., 2011). Studies have shown
an increase in the pressure of the retrocalcaneal
bursa in patients suffering from chronic RB (Lohrer
& Nauck, 2014). For purposes of this discussion, RB
will be referred to in isolation as regards the spectrum
of retrocalcaneal pathology and conditions closely
associated, as previously mentioned, but namely
Haglund’s syndrome.

The painful condition of RB limits function and,
in more severe cases, the bursa itself can become
hypertrophied, septic, calcified, and adherent to the
Achilles tendon and the bony tissue of the calcaneus
(Aaron et al., 2011; Kachlik et al., 2008; Michaud, 1993;
Pedowitz et al., 2008; van Dijk et al., 2011).

There is an increased prevalence of RB when
Haglund’s exostosis and Achilles tendinopathy
are present, but no causal relationship has been
determined according to some literature (Aaron et al.,
2011), although is listed as causal in others (Leitze
et al., 2003; Pedowitz et al., 2008; van Dijk et al., 2011).
It is thought that the closely associated, merging
tendinous tissues of the posterior aspect of the
retrocalcaneal bursa and the anterior Achilles can lead
to degenerative changes within the Achilles (Pedowitz
et al., 2008). Most of these pathologies are closely
linked in both diagnostics and occurrence given the
close anatomical structure, difficulty of differential
diagnosis, and merging of tissues of the calcaneus
and Achilles tendon attachment/insertion, hence a
“sharing” of inflammatory conditions (Pedowitz et
al., 2008). The prominence of the posterosuperior
lateral portion of the calcaneal tuberosity in Haglund’s
exostosis can act as a mechanical irritant of the
retrocalcaneal bursa by altering the angle of insertion
of the Achilles tendon (Pedowitz et al., 2008).

Functional anatomy

The posterior calcaneal tuberosity acts as the most
proximal attachment site for the Achilles tendon (also
known as the calcanean tendon). The retrocalcaneal
bursa is located in the narrow space between the
upper posterior aspect of the calcaneal tuberosity
and anterior to the distal portion of the Achilles
tendon, and is “wedge-shaped” (Kachlik et al., 2008;
Theobald, Bydder, Dent, Nokes, Pugh, & Benjamin,
2006). Its lateral edges match the lateral edges of
the calcaneus and Achilles tendon (Kachlik et al.,
2008). It sits in the surrounding adipose tissue (called
Kager’s fat pad or the retromalleolar fat pad) (Canoso,
Liu, Traill, & Runge, 1988) of the distal aspect, or
the tip of Kager’s triangle, which is well known and
used by radiologists as a landmark in posterior heel
radiographs.

Kager’s triangle refers to the space between the
anterior surface of the Achilles tendon, the posterior
surface of the flexor hallucis longus (FHL), and a
wedge of adipose tissue adjacent to the calcaneus
(Kachlik et al., 2008; Theobald et al., 2006). This fat
wedge is a mobile portion of Kager’s fat pad and
is a continuation of the FHL portion of Kager’s fat
pad functions. The calcaneal bursal wedge in which
the retrocalcaneal bursa sits is a fusion of the FHL–
associated portion and the Achilles–associated portion
of Kager’s fat pad. This distal wedge of fat has been
found to retract during dorsiflexion and extend into
the retrocalcaneal fat pad during plantar flexion
(Canoso et al., 1988; Theobald et al., 2006), more so
under active loaded conditions due to contraction of
the FHL (Theobald et al., 2006). This movement of
Kager’s fat pad helps to minimize pressure on the
retrocalcaneal bursa during dorsiflexion and plantar
flexion (Theobald et al., 2006).

Inflammation within the fat pad can negatively affect
blood flow to the Achilles tendon, albeit temporarily
(Theobald et al., 2006), which in turn can affect
the retrocalcaneal bursa. This can occur through
inflammation of the FHL, which often occurs during
extended plantar flexion, as occurs in soccer and
classical ballet (Theobald et al., 2006).

The retrocalcaneal bursa helps to maintain a
constant distance between the axis of the ankle joint.
and the Achilles tendon. This allows for constant tension of the calf muscles (gastroc/soleus) during dorsiflexion and plantar flexion (Pedowitz et al., 2008). The movement of the Kager’s fat pad into the retrocalcaneal bursa during plantar flexion also offers a mechanical advantage by maximizing the angle of the Achilles tendon insertion in this position (Canoso et al., 1988; Theobald et al., 2006). When there is inflammation of the retrocalcaneal bursa, the sliding motion of the fat pad is reduced.

The sac containing the synovial fluid, also known as the synovial fold, is 1–2cm in size with a volume of 1–1.5mL. The anterior wall of the retrocalcaneal bursa is a thin layer of cortical bone covered by a layer of fibrous cartilage which attaches to the ventral aspect of the calcaneal tuberosity (Aaron et al., 2011; Canoso et al., 1988; Kachlik et al., 2008; Theobald et al., 2006). The posterior wall is tendinous in nature and merges with the Achilles tendon (Aaron et al., 2011; Canoso et al., 1988; Kachlik et al., 2008; Theobald et al., 2006). Some of the literature differs with respect to the microanatomy and histology of the retrocalcaneal bursa, but the above description concurs with the more detailed microscopic anatomical studies of the retrocalcaneal bursa (Kachlik et al., 2008; Theobald et al., 2006).

The surface of the Achilles tendon, as well as the surface of the cartilaginous portion of the calcaneal tuberosity, covered by the retrocalcaneal bursa, is smooth. The outer surface of the retrocalcaneal synovial membrane, which meets the fibrocartilage of the calcaneus and the tendinous tissue of the Achilles, is also smooth (Kachlik et al., 2008). The thin fibrous cartilage of the calcaneal tuberosity continues posteriorly to cover the entire bottom of the retrocalcaneal bursa, into which the Achilles tendon attaches (Kachlik et al., 2008).

The inner surface of the synovial membrane contains a network of large and small villi, the larger ones vascularized from the extended vascularization of the synovial fold. A very small portion of the ceiling of the inner membrane contains delicate skeletal muscle fibres, contributing to the theory of a functional extension of the shape of the retrocalcaneal bursa during maximal dorsiflexion of the ankle joint (Kachlik et al., 2008).

Pathology

Description of symptoms

The symptoms of RB are described as a dull, aching local pain anterior to the Achilles tendon, just superior to the Achilles attachment, and can exhibit a “spongy” feel. It is more pronounced with passive dorsiflexion (Leitze et al., 2003) due to the compression of the inflamed retrocalcaneal bursa between the calcaneus and the Achilles tendon. It generally appears as start-up pain after sleep and when rising from being seated (Aaron et al., 2011; Pedowitz et al., 2008).

Onset often occurs after an episode of activity on an incline (e.g., a weekend hike or the commencement of a treadmill program) and so can be somewhat sudden; however, the development of RB is usually associated with overuse syndrome and more gradual in nature (Pedowitz et al., 2008).

Etiology

There are several causes of RB, the main one being irritation of the bursa through overuse, and so it is more common in runners and in younger populations in their 30s (Aaron et al., 2011; Pedowitz et al., 2008), although the susceptibility of RB increases with age in the active population, due to a decrease in elasticity of soft tissue (NIAMS, 2014). Ankle dorsiflexion and impact activities increase stress on the retrocalcaneal bursa and this can occur through extended activities such as using a treadmill with an inclined surface. Hiking in mountainous regions, especially without prior habituation (e.g., vacationers, “weekend warriors”) as well as occupational stressors (labour/trades, e.g., using a ladder, roofing, landscaping) can be the cause of its onset. Other biomechanical irritants such as the type of foot function are also a consideration. Compensated rearfoot varus and a rigid plantar flexed first ray (commonly found together in the flexible and rigid pes cavus foot types) are known to be susceptible to RB (Aaron et al., 2011; Leitze et al., 2003), as well as rearfoot equinus, compensated forefoot valgus, apophyseal trauma during growth, and frontal plane hypermobility (Lietze et al., 2003).

Achilles tendinitis is found in 10% of runners (Mazzone & McCue, 2002) and amongst many athletes participating in high-impact sports, which may give an indication of the prevalence of posterior calcaneal disorders.

Impact on the patient

RB hinders normal activity at its height of pain and can also affect choice of footwear and daily activity as well as choice of exercise. At its worst, RB can affect the ability to work certain types of jobs (e.g., retail, trades) so the cost of interrupted employment and lifestyle change can also have an impact. All treatments require proper diagnosis, and
possible action or intervention may include referrals, prescriptions, foot orthoses and footwear purchases (if no current footwear is deemed suitable). These interventions cost patients time as well as money. Non–surgical treatment necessitates necessitates regular time and attention for modalities such as ice, elevation, stretching, and activity/exercise modification, in addition to physiotherapy treatments such as ultrasound and even immobilization with walking casts. Surgical intervention for the non–responsive cases necessitates a more dramatic recovery depending on the procedures involved and the presence of additional associated conditions (Pedowitz et al., 2008).

**Differential Diagnosis**

RB must be distinguished from Achilles paratendinopathy and tendinopathy, arthritis, fracture, infection of bony and/or soft tissue, ligament injury, and nerve pathology (Aaron et al., 2011; van Dijk et al., 2011), although this differentiation can be difficult due to the possible co–existence and causal relationships of some of these conditions (Botchu, Khan, & Bhatt, 2010; Leitze et al., 2003; Schepsis, Jones, & Haas, 2002). One study on Achilles tendinopathies found that 15% of cases exhibited a combination of conditions (Schepsis et al., 2002).

Another closely associated condition is insertional tendinopathy of the Achilles, just inferior to the location of the retrocalcaneal bursa. The portion of the retrocalcaneal bursa closest to the anterior aspect of the Achilles tendon is tendinous in nature (Aaron et al., 2011) and this may be why RB often presents with pre–existing Achilles tendinopathy (Aaron et al., 2011; Pedowitz et al., 2008).

The presence of Achilles tendinopathy and Haglund’s exostosis are also thought to be causal in some of the literature due to the merging tissues of tendon and bone at the respective sites where they meet the retrocalcaneal bursa (Leitze et al., 2003; Pedowitz et al., 2008; Theobald et al., 2006).

One distinction in the literature on Achilles tendinopathy is the non–inflammatory character of Achilles tendinosis. This condition is considered to be degenerative in nature, unlike Achilles tendinitis and RB, which are inflammatory (Leitze et al., 2003; Pedowitz et al., 2008).

Haglund’s syndrome includes inflammation associated with the protrusion, or exostosis, of the posterosuperior aspect of the calcaneal tuberosity and often can have a superficial callus. Haglund’s syndrome is associated mainly with three inflammatory conditions: RB, insertional Achilles tendinitis, and superficial Achilles bursitis (also known as adventitial, retroachilleal, and subcutaneous calcaneal bursitis) (Pedowitz et al., 2008). Each of these can occur independently. Superficial Achilles bursitis is sensitive to direct pressure (as opposed to medial/lateral pressure anterior to the Achilles for RB).

Insertional Achilles tendinosis should be investigated by palpating below the retrocalcaneal bursa, and is sensitive to direct pressure (Pedowitz et al., 2008). Differentiating between insertional Achilles tendinopathy, osseous impingement, and RB can be difficult due to the close association of these conditions and their possible concurrence (Leitze et al., 2003). Non–insertional Achilles tendinopathy is recognized by tenderness and swelling above the retrocalcaneal bursa and along the thinner portion of the Achilles tendon.

RB causes mostly a dull pain, and if onset is sudden or there is an acute pain, then Achilles rupture should be considered, and referral to a physician should follow (Pedowitz et al., 2008).

Possible misdiagnoses and associated conditions:

- **Bony complications:** Calcaneal fracture, periostitis, calcaneal epiphysitis, Haglund’s exostosis, osteomyelitis (Aaron et al., 2011; Michaud, 1993).

- **Nerve pathology:** Tarsal tunnel syndrome, nerve entrapment.

- **Systemic complications:** Inflammatory arthrosis, infection, connective tissue disease, ankylosing spondylitis, Reiter syndrome (Pedowitz et al., 2008). Gout, thyroid disease, and diabetes and vascular insufficiency can also be causal conditions (Aldridge, 2004; NIAMS, 2011). These are more likely considered when there is no obvious cause (Aldridge, 2004).

RB can have both infectious and non–infectious etiologies, and distinguishing between them is both important and challenging (Aaron et al., 2011). Some red flags for the diagnostician are open skin wounds or lesions, redness or warmth, erythema, and apparent cellulitis (Aldridge, 2004).

Bilateral RB can be indicative of inflammatory arthritis which can impact tendinous tissue (Aaron et al., 2011).

**Common Testing**

A thorough history and assessment of the non–
weight–bearing foot to observe passive and active ranges of motion, swelling, tenderness, pain on palpation, bony and soft tissue protrusions and deformities, skin irregularities or skin breaks, ulcers, bruising, pulses, and circulatory irregularities or swelling. Identification of the biomechanical foot types, joint rigidities, or hypermobilities affecting ranges of motion followed by a static weight–bearing and gait analysis can help to identify possible biomechanical irregularities and provide further diagnostic indicators (Aldridge, 2004).

Some of the diagnostic tests include:

• The two–finger pinch test is the test most indicative of RB. Pressure is applied medially and laterally, anterior to the Achilles insertion (Aaron et al., 2011; Pedowitz et al., 2008).

• Active resisted plantar flexion as well as passive dorsiflexion of the ankle joint may also trigger pain (Aaron et al., 2011; Leitze et al., 2003).

• Full weight–bearing radiographs taken from a lateral view will confirm the presence of Haglund’s exostosis, which causes greater posterior calcaneal angles.

• Musculoskeletal ultrasonography (dynamic USG) can be highly successful in identifying pathologies in the soft tissues in the foot, and specifically RB (Botchu et al., 2010; Kachlik et al., 2008; Schepsis et al., 2002; Theobald et al., 2006).

• MRI scans are not necessary for a positive diagnosis of RB; however, fat is easily identified on MRI scans, so the pathology of RB relating to the surrounding fat pads is easily identified. On an MRI taken laterally in a plantar flexed position, the presence of retrocalcaneal radiolucency (known as the “retrocalcaneal recess”) is a reliable indication of a negative RB radiograph. RB is positive with the absence of the lucent retrocalcaneal recess anterior to the Achilles tendon (Canoso et al., 1988).

Contraindications

Although there is still a debate in the literature, conservative practitioners would adhere to the corticosteroid injection as being contraindicated due to the increased incidence of Achilles tendon rupture following the injection (Pekala, Henry, Pekala, Piska, & Tomaszewski, 2017; Aaron et al., 2011; Pedowitz et al., 2008; Schepsis et al., 2002; Simpson & Howard, 2009). A weakening and/or rupture of the Achilles tendon can occur with corticosteroid injection (Leitze et al., 2003; Pedowitz et al., 2008; Schepsis et al., 2002), as well as atrophy of fat tissue in which the retrocalcaneal bursa sits, affecting its efficacy (Theobald et al., 2006). One recommended use of a corticosteroid injection was for pre–surgical refractory cases, and limited to only one injection to be made directly into the bursal fold and not to contact the Achilles tendon, although the issue of shared tissues between the retrocalcaneal bursa and the Achilles tendon, as well as the impact on the surrounding fat tissue, were not addressed (Schepsis et al., 2002).

Prevention and recommendations

• Prepare for increases in activity (especially repetitive dorsiflexion) whether a beginner, vacationer, labourer, or advanced athlete on a progressive training program.

• Avoid the “weekend warrior” syndrome. Keep active between more intense episodes such as a golf tournament, an active vacation, or seasonal events like spring gardening or shoveling snow.

• Protect the area from external friction with a well fitted shoe, an appropriate heel counter, and foot orthoses to address certain associated foot functions.

• Avoid flimsy or flat footwear, which causes increased movement of the heel as well as decreased shock absorption.

• Avoid extended periods of dorsiflexion (such as an inclined treadmill) or plantar flexion (such as classical ballet) without a gradual introduction.

• Be vigilant during a period of increased or changed activity like a new or progressive training program or a change to harder surfaces such as with a new home or new employment.

• Review activity or sport technique, such as a heavy heel strike in running.

• Allow for a proper warm–up of muscles and tissues before more intense activity begins.

• Take breaks and change positions from repetitive motions or tasks (e.g., roofing, gardening).

• Stretch after intense activity to re–establish muscle flexibility and mobility.

• Take immediate action if pain begins, such as rest, ice, elevation, massage, stretching, activity reduction and/or modification; seek early treatment if symptoms are not managed and reduced.
• Be aware of whether associated conditions such as Haglund’s exostosis, superficial Achilles bursitis, or Achilles tendinitis are present, in which case a more proactive approach to the onset of symptoms is advised.

Treatment
Non-surgical intervention
When present due to overuse, the majority of cases of RB respond well to conservative treatment (van Giffen et al., 2009). Modifying activities to reduce those on an incline, rest, ice, padding and NSAIDs (if necessary) for the inflammation (Aaron et al., 2011; Leitze et al., 2003; Pedowitz et al., 2008). Biomechanical relief can be achieved with foot orthoses and appropriate footwear (open-backed shoes) with cessation of friction at the heel counter and improved shock absorption at heel strike, although a general reduction of impact activities is recommended (Leitze et al., 2003). A slight incline of the heel allows for a decrease of tension of the Achilles tendon. Night splints in dorsiflexion can reduce the start-up pain when arising after sleep or long periods of rest. Immobilization may be necessary for refractory cases by use of a walking cast, positioned in slight plantar flexion (Aaron et al., 2011; Pedowitz et al., 2008). Strengthening and stretching of the gastrocnemius and the soleus, as well as avoidance of flat shoes or bare feet can assist in relieving inflammation. Custom foot orthoses are also useful in diminishing poor biomechanics of the foot. The pes cavus foot type, compensated forefoot valgus, compensated rearfoot varus, rearfoot equinus, frontal plane hypermobility, and a rigid plantar flexed first ray can all be functionally improved with orthoses. Custom foot orthoses can also reduce external friction of the shoe as well as stress on related soft tissue such as the Achilles tendon, and improve shock absorption, weight distribution, and gait patterns which all contribute to reduction of stress on the injured tissues. Physiotherapy can also help with therapeutic ultrasound and phonophoresis (Leitze et al., 2003; NIAMS, 2014).

Surgical intervention
Cases that do not respond to non-surgical and pharmacological interventions, approximately 10% (Leitze et al., 2003), can be referred for surgical interventions, although each case needs to be assessed independently due to the spectrum of related posterior calcaneal pathologies, which can occur independently but are often concurrent. The surgical assessment will determine the suitability of the various surgical procedures such as:
• Open surgery with greater complication possibilities and longer recovery time vs. endoscopic approach with less predictable results but reduction in recovery time (Aaron et al., 2011; Leitze et al., 2003).
• Resection of the calcaneal superior tuberosity for treatment of Haglund’s exostosis, generally reported to be a poor-outcome procedure (Theobald et al., 2006) with a greater possibility of complications (Leitze et al., 2003).
• Resection of soft-tissue and tendon-splitting procedures, usually recommended with more extensive symptoms such as calcific insertional Achilles tendinosis.
• Calcaneal wedge osteotomy.
• Dorsal closing wedge osteotomy which rotates the posterior calcaneus to a lesser prominence (Aaron et al., 2011).
• Achilles tendon debridement.
• Endoscopic decompression of the retrocalcaneal space, usually preferred for simpler diagnoses such as RB with or without minimal Achilles insertional tendinosis and/or Haglund’s spur (Lietze et al., 2003).
• Complete excision of the retrocalcaneal bursa (Aaron et al., 2011).

Possible surgical complications
• Surgical alteration of the adipose tissue and resulting scar tissue adhesions can affect the movements of the fat pad which in turn, can reduce the lubricating function of the retrocalcaneal bursa (Theobald et al., 2006).
• Alteration of the movements of Kager’s fat pad as well as risk tearing of the retrocalcaneal bursa due to postoperative exposure to the underlying cancellous (spongy) bone (Theobald et al., 2006).
• Skin/wound breakdown.
• Infection.
• Painful scar tissue and altered sensation.
• Achilles tendon avulsion or rupture.
• Joint stiffness (Pedowitz et al., 2008).

Recovery time varies according to the etiology of the
symptoms, and length of time prior to diagnosis and commencement of treatment. Patient compliance, timely access to professionals, and treatment efficacy are elements that affect recovery time and their absence can exacerbate a refractory case. The progression from the lowest level of non-operative intervention through to full surgical intervention, followed by postoperative recovery time and possible complications can be significant for the worst-case scenario. A significantly longer recovery time was found in cases which had pre-existing posterior heel pain due to calcific Achilles tendinitis, Achilles tendinosis (Aaron et al., 2011), or use of a corticosteroid injection (Leitze et al., 2003).

Non-surgical recovery time can vary, but anywhere from four weeks (without Achilles tendinopathy) to 9 months for relief of symptoms is common (Pedowitz et al., 2008). Surgical recovery time will vary from a simple isolated condition, to treatment of a multi-faceted condition, and management of possible complications, which increase the intervention necessary. This, in turn, extends the recovery time.

**Key Considerations**

- RB is most prevalent in runners and athletes and those inclined to overuse syndrome.
- Repetitive or extended dorsiflexion (inclined surfaces) or plantar flexion (classical ballet) are associated with a higher incidence of retrocalcaneal bursa irritation.
- The presence of Haglund’s exostosis, superficial Achilles bursitis, and/or Achilles insertional tendinosis is often concomitant.
- To be differentiated from related inflammatory conditions such as Achilles insertional tendinopathy and superficial Achilles bursitis, although these conditions often are concomitant.
- Sensitive to two-fingered pinch test anterior to Achilles tendon.
- Rigid pes cavus foot types with rearfoot varus and/or plantarflexed first ray with rigid forefoot valgus types are more susceptible to RB.
- Be aware of infection, tendon rupture, bone disorders, and inflammatory arthroses as possible misdiagnoses of more serious conditions.
- Corticosteroid injection is generally contraindicated.

**References**


Retrocalcaneal Bursitis


Haglund’s Deformity

Janelle Coultes, BSc (Biol), C. Ped (C)

Key Messages

• Haglund’s deformity is a bony prominence with isolated pain and irritation at that site of the exostosis.
• Haglund’s deformity differs from retrocalcaneal bursitis in that there is no swelling around the prominence.
• Patients tend to feel better when wearing sandals or slippers that do not have an aggravating heel counter.

Keywords

Achilles, Bauer bump, bony, bursitis, calcaneus, exostosis, pump bump

Introduction

Haglund’s deformity is characterized by a bony prominence, or exostosis, lateral and proximal to the insertion of the Achilles tendon on the calcaneal tuberosity (Alexander, 1997). It is sometimes also called pump bump or Bauer bump, due to the rigid heel counters of high heels and ice skates causing the deformity. Erythema, irritation, and pain occur only at the exostosis itself. This deformity is named after Patrick Haglund because in 1928, he was the first to stress that this “high pointed calcaneus” was a precursor to the occurrence of a painful heel (Chauveaux, Leit, Le Huec, & Midy, 1991).

Pathology

Haglund’s deformity is usually located just lateral to the Achilles tendon proximal to its insertion. The exostosis is oriented longitudinally, parallel to the tendon (Alexander, 1997). A rigid posterior heel counter on footwear is believed to be an inducing factor (Lohrer, Arentz, Nauck, Dorn-Lange, & Konderding, 1998), and can sometimes arise secondary to malunion of the calcaneus after fracture (Lui, 2008). Haglund’s deformity is believed to be the initial mechanical cause that leads to retrocalcaneal bursitis (Lohrer et al., 1998) and once retrocalcaneal bursitis is present, the deformity itself can also increase the irritation of the bursitis (Heckman, Gluck, & Parekh, 2009).

Haglund’s deformity is a frequent cause of posterior heel pain and it is often important in the development of calcaneal tendinopathies (Chauveaux et al., 1991). Pain in the posterosuperior portion of the calcaneus can be caused by the enlargement of the superior bursal prominence of the calcaneus (Haglund’s deformity) (Jerosch & Nasef, 2003). Insertional Achilles tendinopathy (IAT) is among the most common posterior heel conditions while walking and running, and is located at the insertion of the Achilles tendon onto the calcaneus, involving pain and swelling of the Achilles tendon itself, the formation of bone spurs, and calcifications at the insertion site (van Dijk, Van Sterkenburg, Wiegerinck, Karlsson, & Maffulli, 2011). The study of Sundararajan and Wilde exhibited that Haglund’s deformity was present in 25% of IAT patients (Sundararajan & Wilde, 2014).

Differential Diagnosis

Retrocalcaneal bursitis is a common misdiagnosis, given that both conditions have redness, irritation, and prominences at or near the insertion of the Achilles tendon on the calcaneus. Haglund’s deformity does not have swelling that spreads beyond the exostosis itself, and pain occurs only when external pressure is applied to that area (e.g., in the form of the heel counter of a shoe) (Alexander, 1997).

Haglund’s deformity should not be confused with Haglund’s syndrome or Haglund’s disease. Haglund’s syndrome combines inflamed retrocalcaneal bursa and insertional tendinopathy of the Achilles tendon, but may or may not have the presence of Haglund’s deformity. Haglund’s disease is actually osteochondritis of the accessory navicular (van Dijk et al., 2011).

Common Testing

There are three criteria that are fundamental in determining whether Haglund’s deformity will lead to
calcaneal tendinopathies. They are the severity of the deformity, its location in the sagittal plane, and the position of the calcaneus in relation to the ground. A higher vertical angle of the calcaneus causes the dorsal end of the bone to become prominent, and this is an essential pathogenic factor (Chauveaux et al., 1991).

Simple X-ray images can show shape changes and deformities of the bony structures, such as Haglund’s deformity (Kachlik et al., 2008). An MRI is useful in outlining Haglund’s deformity, especially when it is causing impingement of the Achilles tendon (Sullivan, 2010).

The Patient Perspective

The patient would complain of pain on the deformity with pressure, as when the heel counter of a shoe is causing friction. There would be full relief of pain when barefoot or wearing shoes without a heel counter.

Common Treatments

Not a lot of literature exists that focuses solely on treatment of Haglund’s deformity. This is most likely because the exostosis is a precursor to more painful conditions, and that is when patients would seek treatment. From clinical practice, it is recommended to remove external pressure from the area of the deformity itself, i.e., wear shoes with no heel counters, softer heel counters, or modified heel counters that are stretched, cut out, or cushioned in the area of irritation.

Haglund’s deformity might be difficult to treat effectively by non-operative measures alone, which would include analgesia and modified footwear (Anderson, Suero, O’Loughlin, & Kennedy, 2008). For patients with Haglund’s deformity who do not respond to non-operative therapy, there are many surgical options available, including calcaneal ostectomy, excision of the retrocalcaneal bursa (if present), and calcaneal osteotomy. However, the results of these procedures have not been consistent (Anderson et al., 2008). Surgery is recommended for patients who have not responded to conservative treatment for a minimum of six months, however the removal of Haglund’s deformity is a technique that has a learning curve and can be demanding (Sullivan, 2010). The results of a study by Natarajan and Narayanan suggest that a lateral approach to calcaneal ostectomy can be an effective treatment for patients suffering from Haglund’s deformity who are not responding to conservative treatment (Natarajan & Narayanan, 2015).

Because the deformity is closely associated with things like adventitious Achilles tendon bursitis (Michels, Guillo, King, Jambou, & de Lavigne, 2008), most treatment in the literature focuses on reducing the strain on the Achilles.

Treatment that focuses on the Achilles tendinopathy that may result from Haglund’s deformity includes PRICEMM (Protection, relative Rest, Ice, Compression, Elevation, Medication, and rehabilitative exercise Modalities), gastrocnemius and soleus stretching, and an eccentric strengthening program (Simpson & Howard, 2009). Adding a heel raise of 6mm using a dense material such as EVA can reduce the strain on the Achilles, but this can also change the position of the calcaneus within the shoe, meaning that further modification of the heel counter to accommodate the deformity might be warranted.

Extracorporeal shock wave therapy (ESWT) for IAT has been shown to improve clinical outcomes with or without the presence of Haglund’s deformity. However, it has been shown that the presence of Haglund’s deformity may worsen the therapeutic effect of the ESWT for IAT, and VISA-A scores in patients with the deformity were inferior to those without Haglund’s deformity (Wu, Yao, Chen & Li, 2016).
Key Considerations

When Haglund’s deformity leads to insertional tendinitis of the Achilles, it is most often found to occur in obese patients and older or recreational athletes. In addition, running on hills, doing interval training, and general training errors could exacerbate the tendinitis (Heckman et al., 2009).

References


Achilles Tendinopathy

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Key Messages

• Recommendations for clinical use in a pedorthic setting should include a broad spectrum of treatment points focusing on the findings of the clinical examination in an attempt to address multiple potential etiological factors.
• Current level of activity should be discussed and any training errors should be addressed and potentially modified.
• Given the current research, it is reasonable to incorporate a conservative eccentric loading program whenever possible to a pedorthic treatment plan.
• Regular follow-ups should be encouraged to assess progression of treatment or deterioration of condition and adjust accordingly through a modified treatment plan or referral back to family physician with a detailed account of the current treatment history.

Keywords
Achilles tendon, endotenon, epitenon, gastrocnemius, microtears, paratenon, soleus, tendinopathy, tendinosis, tendon rupture, triceps surae

Introduction

Achilles tendinopathy is a common musculoskeletal disorder among both physically active and sedentary individuals (Rolf & Movin, 1997).

Disorders of the Achilles tendon have been studied extensively over the years to establish specific etiology and pathogenesis in an attempt to provide successful prevention and treatment strategies. Unfortunately to date there remains a lot of uncertainty, confusion and debate regarding classification, causation, treatment and prevention.

In general, activities that involve running and jumping pose a greater risk, with recent estimates of up to 9% of recreational runners affected, and cause up to 5% of professional athletes to end their careers (Li & Hua, 2016). Those aged 35 years and over are at an increased risk for developing disorders of the Achilles tendon (Kvist, 1994).

The posterior compartment of the lower leg consists of muscles that are divisible into superficial and deep groups. The gastrocnemius, which originates from the lateral and medial condyles of the femur and capsule of the knee, is the most superficial muscle and provides the shape of the calf. The soleus, which originates from the head of the fibula and medial border of the tibia is broad, flat, and lies deep to the gastrocnemius. Combined these superficial muscles form the triceps surae and share a common tendon of insertion, the Achilles (calcaneal) tendon which inserts onto the posterior aspect of the calcaneus (Tortora, 1999) via an enthesis which anchors the tendon to the bone (Rees, Wolman, & Wilson, 2009).

The primary function of the gastrocnemius is to plantarflex the foot at the ankle joint as well as flex the leg at the knee joint. The primary function of the soleus is to plantarflex the foot at the ankle joint (Tortora, 1999).

The Achilles tendon is the largest and strongest tendon in the body. It measures approximately 15cm in length, with an average thickness of 6mm (Pierre-Jerome, Moncayo & Terk, 2010).

The lateral and medial bellies of the gastrocnemius combine with the deeper soleus muscle in an aponeurosis approximately 12–15cm from the insertion of the tendon onto the calcaneus (Sartoris, Schepsis, & Harris, as cited in Pierre-Jerome et al., 2010).

As the tendon descends it may spiral laterally up to 90 degrees, so that the medial aspect becomes more posterior at the distal end. The amount of rotation is
highly variable and depends largely on the amount and location of fusion between the gastrocnemius and soleus (Reynolds & Worrell, 1991).

This rotation of the tendon is clinically significant as the convergence of the two tendons is most evident between 2–5cm proximal to the calcaneal insertion and this area of concentrated stress usually corresponds with a region of diminished vascular supply (Curwin & Stanish, as cited in Reynolds & Worrell, 1991).

The entire Achilles tendon is enveloped by a thin membrane called the paratenon. The paratenon acts as a sleeve to allow movement of the tendon within the surrounding tissue. Under the paratenon lies a thin sheath called the epitenon which connects the paratenon to the endotenon and provides the neural, vascular and lymphatic supply to the tendon structure (Paavola et al., 2002).

The vascular supply to the Achilles tendon comes from the posterior tibial and peroneal arteries. There exist multiple areas for vascular supply to the tendon. These consist of the musculotendinous junction, the osteotendinous junction and along the length of the tendon through the paratenon. The paratenon serves as the primary blood supply to the Achilles tendon, where branches from the posterior tibial and peroneal arteries transect the paratenon to form a mesh-work vascular system along the body of the entire Achilles tendon (Smart, Taunton, & Clement, as cited in Reynolds & Worrell, 1991).

Pathology

Definitions and classifications

The various disorders related to the Achilles tendon must be differentiated in order to properly research and understand the mechanisms which cause these conditions allowing for efficient and reliable treatment protocols.

Achilles tendinopathy is a general term used to describe the combination of pain, swelling (diffuse or localized), and a decrease in performance surrounding the Achilles tendon (Maffulli, Khan, & Puddu, 1998).

Mid-portion Achilles tendinopathy denotes the location of the tendinopathy in the region of 2–7cm from the insertion onto the calcaneus. Clinical signs and symptoms should include diffuse or localized swelling accompanied by pain and impaired performance (van Dijk, van Sterkenburg, Wiegerinck, Karlsson, & Maffuli, 2011).

Insertional Achilles tendinopathy is located at the insertion of the Achilles tendon onto the posterior aspect of the calcaneus. Often accompanied by calcification in the tendon at the insertion site. Clinical signs and symptoms include pain and stiffness, notable swelling, tenderness around the calcaneal insertion; a bony spur is often palpable (van Dijk et al., 2011).

Achilles paratendinopathy is an acute inflammation (paratendinitis) or a chronic degeneration of the paratenon (paratendinosis) (Maffuli, Sharma, & Luscombe, 2004) is characterized by edema and hyperaemia of the paratenon. Inflammatory cells are present within the space between the sheath and the tendon causing palpable crepitus upon examination (Maffuli et al., 2004).

With chronic paratendinopathy exercise induced pain is the primary symptom and crepitus and swelling are less evident (van Dijk et al., 2011).

Tendinosis indicates a degenerative non-inflammatory process of a tendon. There are no clinical or histological signs of inflammation (van Dijk et al., 2011).

Tendinosis can be either symptomatic or asymptomatic with a risk of spontaneous rupture (Kannus & Josza, 1991).

Etiology and risk factors

Biomechanics

To understand the biomechanical risk factors associated with Achilles tendinopathy it’s important to first understand the role the Achilles tendon plays during the gait cycle.

The gastrocnemius and soleus achieve peak activation during terminal midstance at which point they act to create heel lift. The soleus slows the forward motion of the proximal tibia which eventually stops ankle dorsiflexion, meanwhile the gastrocnemius, which crosses two joints, flexes the knee and plantar flexes the ankle initiating heel lift. These muscles are also activated during the contact phase to limit the amount of internal rotation of the tibia and femur.

Throughout midstance, the soleus assists in supination of the subtalar joint, external rotation of the tibia and stabilization of the lateral forefoot against the ground. The gastrocnemius also assists with supination of the subtalar joint as well as external rotation of the femur. The ability of the gastrocnemius to produce both plantar flexion of the ankle and flexion at the knee greatly assists with hip
flexion and thus ground clearance during the swing phase of the gait cycle (Michaud, 1997).

During the normal gait cycle the Achilles tendon is subjected to substantial loads and may reach up to 12.5 times body weight during running. The magnitude of these loads and the repetitive stress placed on the tendon during regular locomotion are what makes the Achilles tendon susceptible to overuse injuries and ruptures (Arya & Kulig, 2009).

Since the exact etiology of tendinopathy remains unclear, the following parameters are currently some of the most widely accepted causes for the development of Achilles tendon disorders. Structural abnormalities may alter the way in which the gait cycle is preformed, thereby increasing the mechanical load placed on the musculotendinous unit during locomotion and hence increasing the risk of mechanical injury.

**Forefoot varus deformity** occurs when the plantar forefoot locks in an inverted position relative to the plantar rearfoot. This structural malalignment causes prolonged subtalar joint pronation which in turn produces a whipping action or a bowstring effect on the Achilles tendon as the calcaneus remains in an excessively everted position. This snapping of the tendon may potentiate microtears along the medial aspect of the tendon and due to the significant amount of forces the Achilles tendon is subjected to during the propulsive period; these microtraumas may initiate an acute inflammatory reaction and/or a chronic degenerative process that may ultimately lead to complete rupture. Individuals that possess a forefoot deformity may even be at risk for medial Achilles tendon damage during static stance due to the everted position of the calcaneus placing excessive strain on the medial aspect of the tendon (Michaud, 1997; Clement, Taunton, & Smart, 1984.)

Ryan, Grau, Krauss, Maiwald, Taunton, & Horstmann (2009) found evidence of an increase in eversion displacement of the sub–talar joint in runners with Achilles mid–portion tendinopathy. The significant difference of movement of the sub–talar joint between runners with and without Achilles mid–portion tendinopathy found in this study supports the common clinical concept of the whip–lash effect postulated by Clement et al.(1984).

**Forefoot valgus deformity** exists when the plantar forefoot rests in an everted position against the plantar rearfoot. This is typically seen with a pes cavus structure and/or a rigid plantar flexed first ray with a characteristic high medial longitudinal arch and rearfoot varus presentation. In cases of a rigid forefoot valgus deformity the amount of calcaneal inversion necessary to bring the lateral forefoot to the ground can significantly increase the amount of tensile load placed on the lateral aspect of the Achilles tendon, leaving the tendon more susceptible to lateral Achilles tendinitis/peritendinitis. Over time, this can reduce vascular supply to the tendon and result in an increased risk for degenerative changes to occur (Michaud, 1997).

**Limitations to minimal ranges of motion**
Restrictions in ankle dorsiflexion whether from a structural bony block or a functional tightness and/or shortening of the Achilles tendon, results in a compensatory increase in midstance pronation leading to the same risks associated with a forefoot varus deformity (Michaud, 1997).

In addition, inappropriate footwear has been noted as a potential extrinsic risk factor for the development of Achilles tendinopathy. Footwear that cannot control excessive rearfoot motion may contribute to the development of Achilles tendon disorders (Clement, Taunton, & Smart, 1984; Maffuli & Kader, 2002).

**Training errors**
Changes in activity levels that cause fatigue and weaken the musculotendinous unit, such as a rapid changes in frequency, duration, intensity, or terrain including softer/harder surfaces to a training or work environment or the addition of hills to an exercise program has been suggested as potential extrinsic risk factors for developing Achilles tendinopathy (Maffuli & Kader. 2002; Welsh & Clodman, 1980).

**Pharmacological factors**
Pharmacological risk factors that have been associated with Achilles tendon ruptures include the use of fluoroquinolones (Casparian, Luchi, Moffat, & Hinthorn, 2000) i.e., ciprofloxacin, a class of antibiotics used for the treatment of bone and joint infections, skin infections, urinary tract infections, bronchitis, pneumonia as well as several other infections may induce tendon cell death, oxidative damage and collagenolysis which weakens the tendon structure leaving it vulnerable to ruptures (Fu, Rolf, Cheuk, Lui, & Chan, 2010).

Tsai & Yang (2011) propose that the use of fluoroquinolones, in particular ciprofloxacin increases the risk of Achilles tendon rupture by inhibition of cell proliferation and inhibition of the spread and migration of tenocytes as well as enhancing the enzymatic activity of metalloproteinase–2 with degradation of type I collagen.
**Historical findings**

Other reported intrinsic risk factors include changes in vascularity, age, gender, body weight, dysfunction of the triceps surae, and lateral instability of the ankle (Maffulli & Kader, 2002).

Previous injury is another risk factor for developing further injury, where a poor or non-existent healing response results in further disruption or compensatory disorders including but not limited to Achilles tendinopathy (Maffulli et al., 2004).

Achilles tendinopathy is classically known for its prevalence among active individuals, however; it has been associated with many other disorders including rheumatic diseases such as, rheumatoid arthritis, ankylosing spondylarthropathy, psoriatic arthritis, reactive arthritis and gout (Ames, Longo, Denaro, & Maffulli, 2008). Also, metabolic diseases such as familial hypercholesterolemia, dislipidemia, and diabetes mellitus have been linked to Achilles tendinopathy (Ames et al., 2008).

Achilles tendinitis is listed as one of the musculoskeletal manifestations of inflammatory bowel disease. Musculoskeletal involvement may occur in 30% of patients with IBD. It is thought to be the most common extraintestinal manifestation of IBD. In the majority of cases intestinal symptoms coincide with or precede articular manifestations (Fornaciari et al., 1999).

Gaida, Ashe, Bass, & Cook (2009) conducted a systematic review to determine the relationship between increased adiposity and tendinopathy. Their review highlights that often people with tendinopathy have higher adiposity. They go on to state that the mechanism linking higher adiposity and tendinopathy may be mechanical or systemic. They mechanical hypothesis is that tendons are exposed to higher loads with an increase in adiposity possibly leading to tendinopathy. Whereas the systemic hypothesis suggests that bioactive peptides released by adipose tissue may influence tendon structure through a direct mechanism or that systemic metabolic alterations associated with elevated adiposity may affect tendon structure through an indirect mechanism.

More research is needed to establish a cause and effect relationship and to determine through which biological pathway they exert change.

Gaida et al. (2009), propose that although the evidence linking adiposity and tendinopathy is not conclusive, unlike some of the other risk factors listed above increased levels of adiposity are somewhat preventable and reversible.

**Pathogenesis**

Achilles tendinopathy is a general term that encompasses multiple subclasses of pathology which can affect any part of the tendon. There are several theories of the pathogenesis of tendinopathy. However, to date the exact etiology and pathogenesis remains unclear.

Tendinopathy can be categorized as symptomatic or asymptomatic in a clinical setting. Where symptomatic tendinopathy includes chronic pain and degenerative changes confirmed through imaging and/or histological findings, asymptomatic tendinopathy is only recognized after rupture or partial tear with non-symptomatic degenerative changes (Fu et al., 2010).

Kannus & Josza (1991) reported that an "absolute majority" of patients with Achilles tendon ruptures had pathological changes similar to that of tendinosis, which weakens the tendon structure and leaves it vulnerable to ruptures.

This suggests that the degenerative changes associated with tendinopathic tissue aren’t necessarily associated with increased nociception and the warning signs to protect the compromised tissues (Fu et al., 2010).

Histological examination of clinical samples of tendinopathic tissues show classical signs of tendinosis, including but not limited to changes in the collagenous matrix, hypercellularity, hypervascularity, and most importantly a lack of inflammatory cells (Fu et al., 2010).

The pain associated with chronic Achilles tendinopathy when there is a lack of inflammatory cells is thought to be caused by numerous biological reactions. Alfredson & Lorentzon (2007) show a link between vasculo-neural ingrowth and tendon pain as well as the presence of neurotransmitters such as substance P, calcitonin gene related peptide (CGRP), and glutamate and its receptor N-Methyl d-aspartate within tendinopathic tissue. Such substances are involved in the transmission of pain impulses from the periphery to the central nervous system (Webborn, 2008).

Webborn (2008) proposes that the pain associated with chronic Achilles tendinopathy could be classified as a neuropathy due to the neoneurovascularization present in chronic symptomatic mid-portion Achilles tendinopathy.
Dean, Gettings, Dakin, & Carr (2016) performed a systematic review to determine whether inflammatory cells are increased in painful human tendinopathy as well as to assess the link between the presence of inflammatory cells and symptoms or degree of structural tendon failure. They found evidence to support the hypothesis that increased inflammatory cells are present in pathological tendons, which is consistent with a chronic inflammatory process. Due to the small number of studies reported in this review, much more research is needed to fully understand the role inflammatory cells play in tendinopathy.

The traditional biomechanical theory suggests that excessive and repetitive stress to the tendon results in accumulated microtrauma that is incapable of sufficient repair, leading to pain and pathology (Clement et al., 1984).

The stress shielding theory proposes that there are focal regions of tendon that are subject to abnormal loading. Areas of reduced activation shielded from stress may cause enough tendon atrophy to allow injury to occur more readily (Maganaris, Narici, Almenkinders, & Maffuli, 2004; Wyndow, Cowan, Wrigley, & Crossley, 2010).

The theory of hyperthermic damage suggests that changes in joint angles create internal shear forces that are capable of inducing thermal damage to the tendon (Maganaris et al., 2004; Wyndow et al., 2010).

Among all these theories are some basic commonalities that may provide a foundation for a valid and reliable explanation of the pathogenesis of Achilles tendinopathy, leading to more successful and reliable treatment protocols.

Fu et al. (2010) proposed a comprehensive model for the pathogenesis of tendinopathy. The basis of this model focuses on the interaction between tendon injuries and unfavourable mechanical environments which sets in motion a cascade of events leading to tendinopathy.

They suggest that tendinopathies are part of an active continuum of “adaptive healing responses to tendon injuries.” This includes vascular, neural, and peritendinous reactions during various stages of healing (Fu et al., 2010).

These healing responses may be diverted through abnormal pathways due to unfavourable mechanical environments, disruption of inflammatory responses, oxidative stress and or pharmacological influences, leading to both an inadequate and incorrect healing response (Fu et al., 2010).

Degenerative changes as well as active healing can be observed in pathological tissues (Fu et al., 2010).

Finally, pathological tissue may include an infiltration of nociceptive substances which can be resistant to some conventional treatments, leading to chronic pain, or may be devoid of such substances, which leaves the tendon more susceptible to partial tears and/or rupture (Fu et al., 2010).

The failed healing theory consists of three stages: initial injury, failed healing and clinical presentation (Fu et al., 2010).

**Stage I - injury**

Tendinopathy begins with the development of collagenolytic injuries (Fu et al., 2010).

There are multiple triggers for these collagenolytic injuries which may include, repetitive mechanical overload, that may release pro-inflammatory mediators such as PGE (2) resulting in degenerative changes to the tendon (Zhang & Wang, 2010).

Chemicals such as fluoroquinolones have been associated with weakening Achilles tendon structure (Casparian et al., 2000).

Previous traumatic injuries that have not been healed completely are also susceptible to further failed healing responses (Taunton et al., 2002).

During this stage tendon pain and weakness is not significant and a healthy tendon repair process is possible (Fu et al., 2010).

Cook & Purdam (2008) refer to this stage within their continuum of pathology as reactive tendinopathy and propose a noninflammatory proliferative response in the cell and matrix due to acute tensile or compressive overload.

**Stage II - failed healing**

During this stage, the natural healing responses are activated but cannot completely repair the collagenolytic injuries within the tendons (Fu et al., 2010).

The causes related to this failed healing response remain unclear and further research and investigation is required to fully understand the series of pathways involved in this failed response to injury. Current research suggests that unfavourable mechanical
environment, genetic predisposition, hormonal background, and pharmacological intervention may play a role in the healing process (Fu et al., 2010).

Since the repair response of tendons includes many sequential processes including inflammation, neovascularization, nerve ingrowth, sensory mediators that regulate regeneration (Ackermann, Salo, & Hart, 2009), matrix synthesis, and remodelling, diversion from the normal pathways or failure to respond at various stages of the healing response may result in different manifestations of tendinopathy (Fu et al., 2010).

Stage III - clinical presentation

In this final stage, symptomatic tendinopathy can be diagnosed as chronic activity-related pain, while with asymptomatic cases, mechanical weakness accumulates undetected until the tendon threshold is met, resulting in spontaneous rupture (Fu et al., 2010).

In a clinical setting, pain is the primary symptom of Achilles tendinopathy and can be used as a determinant of the level or severity of the disorder. In the early stages, pain is present at the beginning and after periods of activity, with relief during physical activity (Rompe, Furia, & Maffuli, 2008).

In the early stages of Achilles tendinopathy, there is diffuse thickening of the tendon. The tendon body is tender upon palpation, usually greatest approximately 2–6cm from the tendon insertion onto the calcaneus (Maffuli, 2004).

Crepitus may be present, especially in cases of paratendinopathy (Maffuli, 2004).

The area of diffuse thickening and tenderness does not move during ankle joint dorsiflexion (Maffuli et al., 2004; Paavola et al., 2002).

As the degeneration progresses, pain becomes more consistent throughout most activity and may even limit ability to perform activities of daily living (Rompe et al., 2008).

The presence of diffuse thickening and crepitus is diminished and the appearance of a focal nodule becomes more prevalent and the tendon becomes thicker and firm (Rompe et al., 2008).

In cases of tendinosis the tender nodules may move with dorsiflexion and plantar flexion of the ankle joint (Paavola et al., 2002).

Clinical examination

A detailed history should be taken in order to gather pertinent information regarding the background surrounding the injury as well as the mechanical environment that may be associated with the presenting signs and symptoms.

Clinical examination should include physical tests to rule out any other condition and to establish a correct differential diagnosis.

Familiarity with underlying anatomy and detailed palpation of the soleus–Achilles muscle tendon junction (MTJ), Achilles mid-substance and insertion, are important in an accurate diagnosis. Most patients will present with palpable soreness along the tendon proper in mid-substance injury or directly on posterior calcaneal insertion with insertional injury. Soreness at the soleus MTJ is indicative of myofascial irritation and not injury to the tendon proper.

The Thompson squeeze test is an important test for ruling out acute tendon ruptures. With the patient lying in the prone position, and feet hanging over the edge of the table, squeezing the calf should produce a forcible plantar flexion of the foot. Absence of this motion is consistent with a complete rupture of the Achilles tendon. With partial tears of the tendon, these results are inconsistent (Alexander, 1997).

A thorough biomechanical examination and gait analysis should be performed by the practitioner to determine whether there is a structural or mechanical component involved with the onset of the presenting signs and symptoms.

Further examination should be focused on reproducing tendon pain in an attempt to pinpoint location and identify the exact structures involved. Single heel raises may be sufficient to reproduce pain; however, more aggressive loading tests may be required to provoke pain symptoms such as single-leg hopping or jumping forward (Rompe et al., 2008).

Mimicking injuries and or conditions that may produce pain at or around the Achilles tendon include: dislocation of the peroneal tendon, tendinopathy of the plantar flexors, tenosynovitis, irritation or neuroma of the sural nerve, and systemic inflammatory disease.

A key distinguishing feature is that true Achilles tendon pain is almost always confined to the tendon itself (Alfredson & Cook, 2007).
To date, the most common form of imaging used to confirm the diagnosis of Achilles tendinopathy is diagnostic ultrasound. Ultrasound imaging can detect focal tendon thickening, disrupted collagen organization, tears, and changes in vascularity. A recent study by Wang, Luh, Chen, & Li (2011) shows promising results with photoacoustic micro-imaging that may detect, with more sensitivity, morphological as well as microvascular changes within the tendon structure.

**Common Treatment**

Given the limited data and scientific evidence surrounding Achilles tendinopathy there is no universal, multidisciplinary set of treatment protocols. More research and collaboration aimed at determining the exact etiology is required in order to provide effective, reproducible, and reliable treatment results.

Many authors agree that early detection and therapeutic action is paramount in controlling the acute phases of Achilles tendinopathy (Alfredson, Pietila, Jonsson, & Lorentzon, 1998).

There are a number of conservative treatment options available and new concepts and modalities appear frequently in the literature; however, evidence based support for these methods, although widely researched, is scarcely proven.

The initial treatment should be focused on the presumed etiological factors (Alfredson & Lorentzon, 2000).

The most agreed upon first course of conservative action is rest or modified activity, ice (acute phase), and assessment and recognition of potential biomechanical impairments (i.e., forefoot varus deformity, prolonged pronation), training errors such as a rapid increase in intensity, frequency, and duration, and inappropriate footwear or old footwear that has lost the ability to stabilize and cushion the body (Alfredson & Lorentzon, 2000; Clement et al., 1984).

Initial conservative treatment should involve a multidisciplinary approach that incorporates a combination of treatments to ensure the best possible outcome (Alfredson & Lorentzon, 2000).

**Custom foot orthoses**

The use of custom foot orthoses is frequently used for the treatment of Achilles tendinopathy when there is a structural or mechanical deficit detected during the clinical examination despite the fact that the mechanism by which they produce their therapeutic effect is relatively unknown.

Clement et al. (1984) studied 109 runners with overuse injury to the Achilles tendon. They determined that the most prevalent etiological factors included overtraining, excessive pronation, and triceps surae insufficiency. They hypothesized that runners are susceptible to Achilles tendinitis with peritendinitis due to microtrauma caused by these structural and biomechanical abnormalities and that treatment should be focused on addressing these issues. Their conservative treatment strategies included rehabilitation of the gastrocnemius/soleus musculotendinous unit, control of inflammation, and biomechanical control with custom foot orthoses. They reported excellent results in 73 of the 109 with a mean recovery period of five weeks.

Gross, Davlin, & Evanski (1991), in their study of long distance runners with lower extremity complaints, found that the use of custom foot orthoses were effective in over 75% of participants across all injuries and effective in just over 73% of participants with Achilles tendinitis. They consider the ability of the orthoses to address biomechanical abnormalities such as excessive pronation as the therapeutic effect.

Mayer, Hirschmuller, Muller, Schuberth, & Baur (2007) conducted a small study and found significant improvement in pain scores with the use of custom foot orthoses in patients with Achilles tendinopathy.

Ryan et al. (2009) found evidence that devices used to control subtalar eversion may be a reasonable treatment option for patients with Achilles mid-portion tendinopathy who present with overpronation during the midstance phase of running gait.
Munteanu et al. (2009) designed a parallel group, participant blinded, randomized controlled trial with a 12 month follow-up study to determine the efficacy of customized foot orthoses in the treatment of Achilles tendinopathy. This study evaluated the effectiveness of custom foot orthoses in conjunction with an eccentric training program thus increasing the clinical relevance by combining orthosis prescription with a widely accepted treatment program for Achilles tendinopathy. Results of this study were published in 2014 and they found that there was no statistically significant difference between the customized foot orthoses and sham orthoses groups when coupled with an eccentric exercise program. Munteanu et al. (2014) state that limitations to the study include but are not limited to: first, that there are no empirically supported guidelines for the prescription of customized foot orthoses, so the observed lack of effect vs. sham may not be applicable to alternative orthosis prescription protocols; second, that there were no biomechanical assessments such as kinematic analyses preformed on the participants.

Although the use of custom foot orthoses has been frequently used in the treatment of Achilles tendinopathies, more research is required to determine what elicits the therapeutic benefit that has been documented clinically for so many years.

There are numerous theories surrounding the correlation between custom foot orthoses and improved function. Do they improve skeletal alignment? Do they alter magnitude or rate of loading of external forces? Do they decrease muscle activity and support a preferred movement path? Do they reduce joint loading? Do they improve sensory feedback and proprioception? Answers to these questions need to continue to be studied in depth to validate the efficacy and reliability of custom foot orthoses in the treatment of Achilles tendinopathy.

Given the longstanding positive clinical outcomes regarding the use of custom foot orthoses in the treatment of Achilles tendinopathy, it would be reasonable to continue to incorporate these devices as part of a conservative treatment plan, given their ability to address biomechanical and structural abnormalities.

**Footwear**

Footwear prescribed by foot type has long been used as an adjunct to many forms of treatment for various musculoskeletal disorders of the lower limbs, including Achilles tendinopathy. Current research shows that the combination of a posted orthotic device placed within a motion control shoe may be blocking natural rearfoot motion and thus increasing the risk of injury. Neutral running shoes may complement a posted orthotic device with greater efficiency of movement by guiding the foot through a healthy movement pattern rather than restricting the normal mechanics of the foot (Baker, 2005).

Another study by Ryan, Valiant, McDonald, & Taunton (2011) suggests that the prescription of footwear based on foot type may be ineffective and overly simplistic. They found that the use of motion control shoes increased pain and number of training days missed across all foot types, including the group with a highly pronated foot structure.

Given the direction that the research is heading, further investigation is required to understand the role of footwear and its relationship to individual foot type and biomechanical parameters. Furthermore, additional research should address the use of stability footwear in conjunction with custom foot orthoses in order to determined how to prescribe the combination, i.e., pairing level of shoe stability with level of orthotic support or when to prescribe one versus the other.

**Eccentric exercises**

Currently, one of the most commonly used forms of conservative treatment is an eccentric exercise program. First established by Curwin & Stanish in 1984 and then modified by Alfredson et al. (1998) and Alfredson & Lorentzon (2000) and implemented into a treatment algorithm for Achilles tendinopathy by Alfredson & Cook in 2007.

Alfredson et al. (1998) suggest that a heavy load eccentric calf muscle training program be used as an initial treatment regimen for mid-portion Achilles tendinopathy. This model involves no concentric loading and is completed regardless of tendon pain. The exercises are performed twice daily 7 days per week for a total of 12 weeks. If the exercises are completed without pain, then the load is increased.

Alfredson et al. (1998) found that after 12 weeks of a heavy load eccentric training regimen that all 15 patients were back to pre-injury levels of activity with no pain versus the control group that was treated without success.

The exercises were performed by having the patient position themselves on a platform with the affected limb placed with the ankle joint in a plantar flexed position by the uninjured limb. The calf muscle was
then eccentrically loaded by lowering the heel beneath the platform lever. The training program involved 3 sets of 15 with the knee extended followed by three sets of 15 with the knee flexed.

A follow-up study by Ohberg, Lorentzon, & Alfredson (2004) focused on individuals suffering from chronic mid-portion Achilles tendinopathy who had been treated with an eccentric training program showed a significant decrease in tendon thickness, structural abnormalities as well as pain.

Langberg, Ellingsgaard, & Madsen, et al. (2007) demonstrated with a small study that Achilles tendons suffering from degenerative changes responded to 12 weeks of an eccentric training program by increasing collagen synthesis rate. Collagen metabolism in the healthy control tendons seemed unaffected by the eccentric loading.

Cook & Purdam (2008) hypothesize that athletes in an early stage of tendinopathy who are already significantly loading the tendon may not respond to an additional eccentric exercise program. They explain that exercise is a potent stimulus to the already upregulated tendon cells and that this may over stimulate a fundamentally intact, but reactive matrix.

The mechanism by which eccentric loading has shown to be effective in the management of Achilles tendinopathy remains unclear. Allison & Purdam (2009) propose that the rehabilitative function is in relation to the extreme range of motion that the tendon is subjected to and the benefits may be derived from the stretching stimulus of the eccentric loading program. They suggest that improved homogeneity of the passive structures within the musculotendinous unit, modulation of the neurological stretch responses and increased shear forces between the tendon and paratenon are possible mechanisms of action for the therapeutic response to eccentric loading programs.

Furthermore, they propose that this may be why insertional tendinopathies respond poorly to eccentric loading and cite the 2003 study by Fahlstrom, Jonsson, Lorentzon, & Alfredson that shows very poor results for insertional Achilles tendinopathy treated with eccentric training. However; a modified version by Jonsson, Alfredson, Sunding, Fahlstrom, & Cook (2008) where patients with insertional Achilles tendinopathy preformed the eccentric loading without going into ankle dorsiflexion showed promising results.

Rees, Lichtwark, Wolman, & Wilson (2008) hypothesized that eccentric loading results in different physiological changes than that of concentric loading and found that the eccentric movement pattern possessed a sinusoidal wave pattern. They suggest that these fluctuations in force may provide the stimulus for the remodelling of the tendon and explain the therapeutic benefits of an eccentric training program.

Eccentric training programs have shown significant results in the treatment of Achilles tendinopathy; however; further research is needed to determine the mechanisms that provide the therapeutic benefits as well as optimal exercise, parameters such as number of repetitions, speed of movement, load progression, frequency and duration of treatment.

Injection-based treatments

Sonographically guided dextrose injections have shown positive results in severe cases of Achilles tendinosis where other forms of conservative treatments have failed. Dextrose injections are believed to induce a cascade of reactions to initiate a healing response. A recent study showed significant long-term effects with dextrose injections improving pain scores and sonographic appearance in both mid-portion and insertional Achilles tendinosis (Ryan, Wong, & Taunton, 2010).

There are many more conservative forms of treatment for Achilles tendinopathy that have shown positive effects in a clinical setting but lack conclusive scientific evidence to reliably and consistently recommend them within a pedorthic setting.

These include but are not limited to the following:

- Corticosteroid injections remain somewhat controversial. Gill, Gelbk, Mattson, Anderson, & Hurwitz (2004) conducted a safety and efficacy profile and found that fluoroscopically guided low volume injections into the peritendinous space were deemed to be safe however the efficacy of the injections were low.

- MMP-inhibitor aprotinin injections. Orchard, Massey, Brown, Cardon–Dunbar, & Hofmann (2008) found improvement in 84% of patients with mid-portion Achilles tendonopathy and 69% in insertional Achilles tendonopathy; however, this study lacked a control group and listed several undesirable side effects. Further research is needed.

- NSAIDs are used in the acute phase to control inflammation but have shown little effect in the chronic degenerative stages of Achilles tendonopathy. Astrom & Westlin (1992) found
success rates that matched those of the placebo group when compared to those in the NSAID group.

- Sclerosing injections of small vessels to treat chronic painful tendons have shown good short and mid-term clinical results. Further research on the mechanism of action is required (Alfredson & Lorentzon, 2007).

- Platelet rich plasma injections have been frequently used; however, Sterkenburg & van Dijk (2011) suggest further investigation into methods and results.

Other forms of treatment with limited research or inconclusive results include but are not limited to the following:

- Heel lifts
- Athletic taping
- Kinesio taping
- Compression garments
- Deep friction massage
- Augmented soft tissue mobilization
- Low-level laser therapy
- Extracorporeal shockwave therapy

Surgical intervention

After all forms of conservative treatment have failed, surgical intervention may be required. Approximately 24–45% of patients with Achilles tendinopathy are unresponsive to conservative treatment (Maffuli et al., 2004).

The objective of surgical procedures for chronic Achilles tendinopathies is to excise fibrotic and degenerative tissue and make longitudinal incisions in an attempt to restore normal vascularity, providing improved nutrition and a favourable environment to initiate a healthy healing response (Maffuli et al., 2004).

Results of surgical intervention tend to favour those without a focal area of tendinopathy. Post-surgical rehabilitation is extensive and long-term results may be only as good as the strength and functional capacity that is regained (Alfredson & Cook, 2007).

References


Achilles Tendinopathy


Peroneal Tendon Subluxation

Lise C. Tremblay, C. Ped Tech (C), C. Ped (C)

Key Messages

• Starting by asking the patient’s chief complaints: the main presenting symptom and how long they have been experiencing the pain or discomfort. Finding out what type of activity they were doing when the injury occurred can also help determine if there’s a possible lateral column strain.

• If the activity or movement that created the injury entailed an abrupt inversion movement, an ankle sprain can be determined as a definite possibility for a primary diagnosis.

• It’s important to address any biomechanical foot structure that could be a key factor resulting in a lateral ankle sprain and the possibility for an acute or chronic subluxation. For example, a high-arched foot structure that remains in the rearfoot varus position (inverted) under full weight bearing can be at risk for a peroneal tendon subluxation, especially if they participate in sports with a lot of lateral movements.

Keywords

ankle sprain, eversion, fibular groove, inversion, lateral compartment, peroneal retinaculum, popping, supination

Introduction

Peroneal tendon subluxation was first described by Monteggia in 1803 in a ballet dancer and is relatively uncommon (Oliva, Del Frate, Ferran, & Maffulli, 2009).

Peroneal tendon subluxation occurs during the gait cycle while the peroneal longus and peroneal brevis tendons are contracted and the tendons jump or dislocate out from the peroneal groove.

Most sports have elements of running and lateral movement. Sports such as soccer, basketball, and football can be highly demanding on the lower extremity. The role of the peroneus muscles is to evert the ankle and stabilize its subtalar motion. In balancing the foot, they play off the posterior tibialis muscle on the opposite side of the tibia. Maximal exertion occurs with side-to-side movement and jumping (Sports Medicine, 2013).

Peroneal tendon subluxation most commonly occurs in individuals who participate in sports that involve repetitive ankle motion. In addition, people with higher arches are at risk for developing peroneal tendon injuries. Basic types of peroneal tendon injuries are tendonitis, tears, and subluxation (American College of Foot and Ankle Surgeons, n.d.).

Pathology

The lateral compartment muscles comprise the peroneal longus muscle and peroneal brevis muscle. The tendons are connective soft tissues that attach muscle to bone. The peroneal muscles are the two muscles located in the lateral compartment of the lower leg which are the primary muscles that support the ankle and help resist inversion of the foot.

The peroneus longus tendon runs along the lateral aspect of the fibula, down into the cuboid groove joint and along the plantar aspect of the foot, and attaches to the plantar surface of the base of the first metatarsal and the lateral face of the medial cuneiform.

The peroneal groove (also called retromalleolar) is located behind the fibula on the outside lower aspect
of the lower leg at the lateral malleolus (the outside ankle bone) and the peroneal tendons relocate by snapping back into the groove.

The peroneus brevis tendon is much shorter and runs in front of the peroneus longus and inserts into the base of the fifth metatarsal head on the lateral side of the foot (Wheeless Online, n.d.). The two tendons meet at the lateral aspect of the ankle and enter in a sheath that forms a tunnel around both tendons. The retinaculum holds the two tendons in the groove. Its normal range is 5–10mm wide and up to 3mm deep.

The superior peroneal retinaculum is the primary constraint to peroneal tendon subluxation and originates from the distal lateral surface of the fibula. It varies considerably in thickness and normally has 2 bands: the superior band inserts on the Achilles tendon and the inferior band inserts on the peroneal tubercle on the lateral surface of the calcaneus.

The lateral compartment muscles lie in the outer part of the lower leg and are stimulated by the superficial peroneal nerve. Contracting the lateral compartment muscles makes the tendons glide in the retromalleolar groove like a pulley. The peroneal tendons evert the foot and plantar flex the ankle. In addition to these functions, they also help to stabilize the metatarsophalangeal joint, aid in the resupination of the foot, and help stabilize the lateral column during the gait cycle.

Acute dislocation of the peroneal tendons occurs by a sudden forced dorsiflexion and simultaneously with an unusual contraction of the peroneal muscles. The dislocation of the peroneal tendon can occur from a combination of dorsiflexion either in the eversion of the ankle joint (like skiing or skating) or in the dorsiflexion and inversion of the ankle joint. Dorsiflexion and the eversion position slacken the peroneal tendon, allowing it to pop out of its groove. However, the injury can also occur from dorsiflexion and inversion of the ankle whereas the tendon is exaggeratedly stretched and pops out of the retromalleolar groove. In retrospect, this injury is often seen with ankle instability during certain sports allowing the ankle to dorsiflex and overly invert or evert.

Peroneal tendon subluxation happens when there is damage to the peroneal retinaculum. The peroneal retinaculum is often injured in a lateral ankle sprain, foot inversion with a sudden contraction of the peroneal muscles, or a direct hit to the posterior lateral malleolus. Lateral ankle pain, tenderness, bruising, and swelling may occur. Often, a snapping or popping sound is heard when the ankle is in motion (Advanced Dynamic Therapies, n.d.).

Chronic peroneal tendon dislocation is often associated with recurrent ankle sprains, which lead to incompetency and laxity of the peroneal retinaculum, subsequently causing peroneal tendon subluxation (Oliva et al., 2009).

Recurrence of subluxation presents with instability during the gait cycle and an inversion of the ankle joint. Chronic injuries of the retinaculum cause instability, pain, swelling, bruising, and a snapping sound across the ankle, which are classic indications of the peroneal subluxation condition.

The rearfoot varus is a structural deformity of the heel positions in a supinated and inverted alignment with the medial border of the foot off the ground, in which excessive shoe wear occurs on the outer aspect of the sole and heel (Wu, 1990). It is an often overlooked contributing factor in recurrent lateral ankle sprains.

Uncompensated rearfoot varus is an issue that is often corrected in conjunction with the treatment of the peroneal tendon subluxation.

The rearfoot varus produces dysfunction of the subtalar joint primarily during the contact period, as this joint often returns to a stable position by mid-propulsion. The contact period associated with the rearfoot varus deformity may produce injury partly because the subtalar joint moves through this exaggerated range in less than 0.15 seconds (Michaud, 1997).

The Patient Perspective

Patients may report a feeling of popping or snapping at the time of the acute event or while walking after the injury (Oster, 2011). There may be swelling and pain which increase with activity, especially to the rear of the fibula. Weakness and instability of the foot and ankle may also be a common complaint. The tendon may even be seen to split out of place along the lower tip of the fibula. It is common to feel pain and tenderness along the tendons. There may also be swelling just behind the bottom edge of the fibula.

If the pain is concentrated along the lateral aspect of the ankle joint and around the lateral malleolus, it would be the first indicator of possible lateral ankle sprain.
Common Testing

Peroneal subluxation falls in one of two categories—acute and chronic. Upon examination, there will be tenderness posterior to the lateral malleolus.

Palpating and following the tendon along the lateral side of the ankle joint, down the lateral longitudinal arch on the plantar aspect of the cuboid, and continuing across the foot up to the base of the first metatarsal, may produce pain which would indicate a positive sign of a strained peroneus longus tendon.

With acute injury, pain and swelling are inferior and posterior to lateral malleolus. The patient may have had pain before the injury, but now the pain is debilitating and strength is decreased.

Subluxation of the peroneal tendons may be provoked by having the patient dorsiflex and evert the foot from a position of plantar flexion and inversion (Michaud, 1997). Look for a prominence of the tendon with dorsiflexion and internal rotation. With chronic peroneal tendon subluxation, there will often be signs of ankle instability.

Pain is noted at the retromalleolar region with dorsiflexion and eversion against resistance. Movement of the tendon on the dorsal/lateral aspect of the malleolus can often be seen. A snapping sound upon moving the ankle is the classic sign of the peroneal tendon subluxation.

Chronic injury results in the subtle, insidious onset of pain posterior to lateral malleolus that progressively worsens in terms of both function and the level of pain (Neale & Adams, 1989).

Tenograms and CT scans may be used to help diagnose the condition but MRI scans are of the greatest value to evaluate the soft tissue (Oliva et al., 2009).

Diagnostic imaging is used to help identify the peroneal subluxation. However, it can give false results if taken without tension on the tendon because the subluxation occurs when the muscle is being contracted.

Dynamic high resolution ultrasound can also be used to diagnose the injury and is effective in showing subluxation and associated tendon splits (Oster, 2011).

These are common methods used by most physicians especially for the patients who are considering undergoing surgical treatment.

Examination of the shoes

Since the shoes enclose and protect the feet during our daily activities, deformities or dysfunctions of the foot often produce predictable distortions or abnormal wear patterns on various parts of the shoe (Wu, 1990).

To be able to address the injury is of the utmost importance to avoid the need for any surgical treatment for repair, but to be able to treat the cause would considerably decrease or possibly even avoid recurrences.

Differential Diagnosis

Peroneal subluxation is often misdiagnosed as an ankle sprain, which is its main cause. An ankle sprain where there are tendon ruptures can be treated both with surgical techniques and more conservative methods like ankle strapping, ankle bracing, exercise, and custom-made orthoses, along with supportive footwear for related biomechanical issues.

If the examiner pulls or pushes on the ankle joint in certain movements, gross instability occurs. Symptoms and signs may include: total rupture of a tendon and the retinaculum, gross instability of the joint, severe pain initially, severe swelling, and usually extensive bruising.

Common Treatment

Non-athletic individuals may have greater tendencies to more conservative care, whereas highly athletic individuals may prefer surgical resolution for better and quicker outcomes.

Ankle instability consistently causes lack of postural control (Werd & Knight, 2010). Studies of foot orthoses have shown positive effects in improving postural control in both injured and non-injured subjects (Werd & Knight, 2010). Methods by which foot orthoses can improve postural control including optimizing the position of the foot, reducing strain and load on supportive soft tissue structure, and improving the receptor sensory field on the plantar surface of the foot (Werd & Knight, 2010).

When addressing these biomechanical issues, a corrective custom-moulded foot orthosis may be prescribed along with an extrinsic lateral wedge or posting to position the foot in a more neutral alignment. The thickness of the wedge would vary with the degree of supination.
Treatments may be divided into conservative (or non-surgical) and surgical, depending on the severity and acuteness of the injury.

Conservatively, treatment of peroneal tendon subluxation is usually similar to the treatment of an acute ankle sprain with ice, anti-inflammatory medications, and compression.

When the rearfoot varus is present, or a tibia varum, or possibly even both, it creates strain along the lateral column. The peroneus longus and the peroneus brevis tendons can snap out of the groove along the lateral aspect of the ankle joint. Therefore, there are a few different ways to accommodate the rearfoot varus in order to minimize the inversion.

If the pain is subtle and there is no obvious need for orthotic therapy, or simply to create a temporary lateral support until the custom-moulded foot orthosis is fabricated, the use of felt with an adhesive backing is very quick and effective. Considering an ankle brace along with the felt might be prudent because the latter isn’t a very sturdy material.

Lateral wedges fabricated with a stronger material (i.e., 45–65 Durometer Proform Evalite) can also be inserted in a shoe to create a lateral support.

A custom-made foot orthotic device constructed with strong materials (i.e., 65 Durometer Proform EVA or Thermoplastics) that is prepared by a qualified and certified professional may be prescribed for those individuals with misalignment or instability in order to correct the alignment on the lateral aspect. If there is enough range of motion in the subtalar joint to allow a correction, increasing eversion of the rearfoot varus, the strain of the peroneal tendons can be relieved.

Feet identified in a rearfoot varus are more susceptible to lateral ankle sprains. Because of the severe supination during the gait cycle, there is a constant strain on the peroneal muscles, which can cause peroneal subluxation and recurrence if the varus alignment is not supported properly.

Therefore, for those who display an uncompensated rearfoot varus foot structure, a custom orthosis can be prescribed to help correct the biomechanics of the ankle joint and increase the stability of the rearfoot. The lateral support decreases the risk for lateral ankle sprains and strain on the lateral compartment muscles.

Along with the custom orthotic device, proper footwear selection should also be taken into account in order to accommodate the foot structure. The combination helps to provide a greater support benefit and therefore improved outcomes.

The rigid high-arch foot type is an underpronator and considered to be a poor shock absorber. It is susceptible to the overuse injuries that are associated with running. This foot type does well with neutral and stable running shoes.

These styles of running shoes allow motion to occur, therefore decreasing the stress placed on the lower extremities (Werd & Knight, 2010).

A neutral posting shoe where the upper is positioned at a 90-degree angle with the outer sole and is constructed with a forefoot rocker sole along with the lateral supportive orthosis will work best to keep the rearfoot in a neutral position during stance. This allows the ankle joint to remain stabilized which decreases the chance of subluxation and lateral ankle sprains during the gait cycle.

**Other non-operative treatment**

- A walking cast with a hard shell can be used to stabilize the foot; however, a lateral wedge should be inserted into the boot so that the foot sits in a neutral position, allowing the peroneal tendon to relax.

- Ankle strapping may also be used as a preventative measure for chronic peroneal subluxation sufferers.

To prevent further injury, consider strapping the lower leg using a Tensor bandage or sport tape as a temporary measure until the patient is seen by either a physiotherapist or a sports medicine doctor. Strapping the ankle joint will encourage the tendon to stay in the peroneal groove located behind the fibula.

1. Anchor the band on the medial aspect of the midfoot, clockwise for the right and counter-clockwise for the left. Wrap the midfoot, overlapping the tape at least two times before going up the ankle joint. Starting at the lowest extremity and going up the ankle allows for blood flow and decreases swelling.

2. Ensure that the tendons are reduced before immobilization by placing the foot in plantar flexion and in a slightly inverted position continuing to wrap the lower leg.

3. Continue to use the band for the next six weeks to allow the injured retinaculum to heal and to prevent chronic subluxation.
Therefore, explaining to the patient how to secure the tendon correctly is very important for proper healing.

If using athletic tape, applying a skin prep product will protect the skin from adhesive trauma. Stabilizing the ankle joint in a plantar and inverted position will ensure that the tendon is located in the peroneal groove.

• Using 1.5-inch tape, wrap anchor strips, one around the leg just above the ankle (layered twice) and another two double-layer strips around the arch and the instep of the foot. On the lateral aspect of the leg, apply a band, creating a stirrup that starts at the top anchor strip and travels down under the foot just in front of the heel, coming up on the medial side. Anchor the stirrup with a layer from the lateral malleolus around the back of the ankle joint up to the medial malleolus. Repeat this horseshoe-like method three times to ensure that the joint is stabilized and secure. Continue wrapping the foot and the ankle starting from the midfoot, and then upward past the ankle joint.

• Physical therapy with a certified therapist may be prescribed to help regain normal ankle function, because it is important to improve the strength and the coordination in the ankle joint (H.E.M Ankle Rehab System, n.d.).

• Ice and ibuprofen or another anti-inflammatory medication may be prescribed to ease the swelling and discomfort more quickly.

• Even with conservative treatment, ankle strapping or ankle bracing done for acute injuries in active young athletes peroneal subluxation continues to have a relatively high recurrence rate of 50% (Wheeless Online, n.d.).

**Muscle-strengthening therapy**

After the injury, a physiotherapist may use massage and other hands-on treatments to ease muscle spasm and pain, and ankle rotation to help improve ankle range of motion without putting too much strain on the area.

After about six weeks, more active exercise can be started. Exercises are slowly added to the therapy to improve strength in the peroneal muscles and to help regain position sense in the ankle joint, improving its overall stability. The physical therapist’s goal is to help keep the pain under control, to improve range of motion in the ankle joint, and to maximize strength and control in the ankle by building the lateral column muscles.

This will typically progress from sitting, to standing on stable ground, to standing on a rocker board, then to agility drills. Only when one achieves full range of movement, good strength, and ability to complete sports-specific agility drills can one be allowed to return to sports.

**Seated heel raises**

Seated heel raises strengthen the soleus or the lower calf muscle. These are performed by comfortably sitting in a straight back chair with knees bent and pointing feet straight in front on the floor while placing them shoulder width apart, then slowly elevating the heels as far as can be tolerated, holding for about three seconds at a full range of motion and gradually bringing the heels down until they are just shy of hitting the floor. Complete as many repetitions as proper form will allow. This exercise can be progressed by wrapping weight cuffs around each ankle.

![Strapping of the lower leg with a tensor bandage as a temporary measure. Photo by Lise C. Tremblay.](image)

**Exercise band dorsiflexion**

Ankle dorsiflexion increases mobility in the lower limbs. While sitting on a long table with the affected leg straight and off the end of the table, secure the exercise band around the forefoot. Hold the ends of the band with both hands and plantar flex the foot through a pain-free range of motion. The band provides light resistance. Dorsiflex the foot back to a 90 degree angle.

Make sure the thickness of the band doesn’t compromise the ankle motion.

**Exercise band abduction**

Secure the exercise band along the lateral aspect of the midfoot. Hold both ends of the band with the opposing hand. Abduct the foot against light lateral
resistance and allow the band to provide resistance back into adduction.

**Standing heel raises**

Standing heel raises engage the gastrocnemius muscle. These are performed by standing with back and legs straight, placing hands flat on a stable wall with feet shoulder width apart, lifting both heels deliberately as much as possible, and lowering heels down after holding for about three seconds. Repeat once heels are about to glance on the floor. Do not lean forward while raising heels. Increase performance by doing heel rises on a step.

**Wobble board stance**

A wobble board is a balance tool that enhances lower leg stability by standing with feet flat on top of the flat device and straightening legs and back while looking forward.

Tilt the wobble board side to side by shifting the weight of the body. The idea is to attempt the side to side motion without holding on to anything. Complete four to five reps of 30 seconds side to side balancing motions.

**One-foot stance**

Standing on one foot engages lower leg the posterior and the lateral compartment muscles and increases stability.

Stand flat with one leg raised with back and the other leg straight, placing both hands flat on a stable wall. Lift the heel deliberately as much as possible and lower it down after holding for about three seconds. Repeat once the heel is about to touch the floor. Do not lean forward while raising the heel. Increase performance by doing the heel raise with added ankle weight.

**Surgical treatment**

Surgical treatment may be considered for chronic peroneal subluxation sufferers and for serious athletes, due to the likelihood of a speedy recovery time with superior results. Postoperatively, patients may be kept in a boot cast for six weeks with partial weight-bearing and physical therapy with supervised ankle joint mobility. They will then require the use of a walking cast for the following 3–6 months.

When treating post-surgery peroneal tendon subluxation, it’s important to remember to accommodate any biomechanical factors that may contribute to the lateral instability and ankle sprains.

It is important to recognize that the biomechanical factors may have been initial causative or contributing factors to the peroneal tendon subluxation. If the ankle instability is not addressed, the surgery to correct the peroneal tendon subluxation is likely to fail (Wheeless Online, n.d.).

**Contraindications**

The peroneus longus and brevis muscles are overworked and become fatigued in side–to–side sports, classically tennis, but also aerobic dance with frequent hopping from one foot to the other (Neale & Adams, 1989).

Those who display lateral ankle instability should avoid activity that involves a lot of lateral side–to–side movement and/or effectively use ankle bracing or taping to reduce recurrence.

Avoid any footwear with a cushioned sole or paws at the heel. These shoes contain soft materials that compress laterally, which reduces control during heel strike and increases the risk for an ankle inversion.

If a custom orthotic device is constructed in such materials, allowing the foot to invert, it will increase the potential for subluxation. The lateral compartment should be supported to avoid allowing the peroneal tendon to slip along the lateral malleolus.

Another concern is that if the shoe is not properly fitted, if it is medially posted causing the foot to invert, or if the heel height is over 1–1½ inches, allowing the foot to plantar flex and invert, stress on the lateral column may cause a negative effect on pain management and creating the potential for lateral ankle sprains and recurrence of subluxation.

**Key Considerations**

When examining a patient suffering from possible peroneal tendon subluxation, the first step should be taking a careful detailed history, paying particular attention to their activity level and the type of sports they participate in. Given that peroneal tendon dislocation is caused by the rupture of the peroneal retinaculum ligament, which acts to hold the peroneal tendons behind the ankle bone, one would be wise to encourage the use of preventative ankle bracing or taping at the initial visit.

Surgery generally should be seen as an avenue for those suffering from acute symptoms and for seriously athletic individuals. Postoperatively, the patient should have physical therapy and be
reassessed for any biomechanical factors that may contribute to the recurrence of the injury.

Walking boots have been shown to be as effective as casts in reducing soleus and peroneal muscle activity during the stance phase of gait, while actually significantly reducing gastrocnemius activity compared to casting (Werd & Knight, 2010).

The avid athlete may also consider stabilizing the ankle joint with taping while exercising to decrease the possibility of future tearing.

Long-term therapy for most patients, with or without surgical repair of the soft tissue, would likely include a customized lateral motion-control orthosis that decreases supination and allows the foot to be in a more neutral position during stance, along with properly fitted and slightly cushioned supportive footwear build with a forefoot rocker sole, allowing the propelling motion of the foot during toe-off phase to relieve strain on the ankle joint. Patients should also incorporate muscle-strengthening exercises into their daily routine, along with other conservative methods like ankle bracing during sport activities.

References


Chronic Ankle Instability

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Key Messages
- Commonly described as feeling of giving way.
- Typically affects physically active individuals.
- A result of mechanical and/or functional deficit.
- Majority of instability affects lateral ankle.
- One may reduce the incidence of ankle sprains by way of physiotherapy exercises, bracing, taping, footwear, and/or custom orthosis use.

Keywords
ATFL, eversion, inversion, lateral/medial/syndesmotic ligaments, laxity, ligament instability, proprioception, sprain

Introduction

Chronic Ankle Instability (CAI) is described as a feeling of giving way in the ankle either laterally or medially and may result in persistent pain, swelling, limited and/or increased range of motion after having experienced a previous ankle sprain (Guillodo, Varache, & Saraux, 2010). It is a result of mechanical instability, functional instability, or a combination of both. Ankle sprains account for nearly 75% of all ankle injuries (Hintermann, Boss, & Schafer, 2002; Garrick, 1977). This includes eversion sprains (mainly affecting the deltoid ligaments), high ankle sprains, (injuring the syndesmotic ligaments) and most commonly inversion sprains (Hupperets, Verhagen, Heymans, Bosmans, van Tulder, & van Mechelen, 2010; Konradsen, 2002; Sprained ankle, 2011). Inversion sprains are the most prevalent ankle injury, making up 85% of ankle sprains (Hintermann et al., 2002) with reocurrence rates estimated to be as high as 70% (Cordova, Sefton & Hubbard, 2010).

Those affected by CAI are typically physically active individuals and participate in activities that require jumping, landing, and/or cutting movements as well as running on uneven surfaces (Sprained ankle, 2011); however, this is not exclusive. CAI also affects those in work related injuries, injuries in the home, and motor vehicle accidents (Hintermann et al., 2002).

Pathology

The stability of the ankle is a product of mechanical and neuromuscular factors. From this, research has described the mechanism for repeated instability as mechanical (ligament laxity) and functional or neurological (proprioception). Foot type may also be considered a risk factor, as those with a cavus foot are considered more prone to lateral ankle instability. First, it is important to recognise the mechanical stability of the ankle.

Lateral ligaments

The mechanical stability of the ankle is based on joint congruency and supporting ligaments. The weakest and most commonly injured of these ligaments is the anterior talofibular ligament (ATFL), often as a result of an inversion sprain (Hockenbury, O’Connor, Sallis, Wilder, & St. Pierre, 2005). Inversion sprains occur when the foot is inverted and plantar flexed to create inversion rotation, thereby causing the talotibial joint to be in an unstable position (Konradsen, 2002). “Most ankle sprains occur when a supination moment force is applied to the foot while an external rotation is applied to the leg” (i.e., landing from fall or from a jump in an unbalanced position, stepping down on uneven terrain or into a hole) (Hockenbury et al., 2005; Santilli, Frascharelli, M., Paoloni, Frascharelli, F., Camerota, De Natale, & De Santis, 2005). The calcaneofibular ligament (CFL) is the next weakest ligament of the lateral ankle and an injury of it is often preceded by a tear of the ATFL.
It is rare for the CFL to have an isolated injury. The posterior talofibular ligament (PTFL) is the third weakest ligament, respectively, of the lateral ankle (Hockenbury et al., 2005).

The degree to which the lateral ankle is injured is affected by the relative position of the foot as it inverts. For example, if the sagittal plane (plantar and dorsiflexion) of the ankle is in a plantar flexed position, the ATFL is affected. In a more neutral position, the CFL may be more likely to tear, and in a dorsiflexed position, the PTFL is placed in a more vulnerable position.

**Medial ligaments**

The ankle joint is also stabilized by the deltoid ligaments at the medial aspect. This group of ligaments is the least commonly injured. It is comprised of the (superficial) tibiocalcaneal ligament, tibionavicular ligament, and superficial tibiotalar ligament as well as the (deep) posterior tibiotalar ligament and spring ligament, also known as the plantar calcaneonavicular ligament. It is also rare to have an isolated tear to this ligamentous group; however, mechanism of injury is a result of eversion of the ankle joint or external rotation of the foot, such as running down stairs or dancing while the body rotates in the opposite direction (Hintermann, 2003; Hockenbury et al., 2005). Injury is suspected by a feeling of “giving-way,” especially medially when walking on even ground, downhill, or down stairs. Due to the force required to injure the deltoid ligaments, an eversion sprain may be accompanied by a distal fibular fracture or syndesmotic ligament tear (DeGnore & Almekinders, 1996).

** Syndesmotic ligaments**

Syndesmotic ankle sprains, also known as high ankle sprains, are also less common relative to inversion sprain injuries. These ligaments are made up of the anterior tibiofibular ligament, posterior tibiofibular ligament, interosseous ligament, and interosseous membrane. Mechanism of injury occurs when there is forced external rotation of the foot or internal rotation of the tibia on a fixed planted foot (Hockenbury et al., 2005).

**Mechanical deficits**

Mechanical instability describes the ligamentous disruption and laxity of the ligamentous structure. The mechanical damage of chronic ankle instability varies on the degree of injury intensity, including factors such as weight, foot position and momentum (DeGnore & Almekinders, 1996). Hertel, Denegar, Buckley, Sharkey, & Stokes (2001) note that a “loss of structural integrity of the ankle ligaments after injury [predisposes] individuals to further injuries because the ankle is allowed to go through a greater range of motion.” As noted earlier, the ATFL is the most common site of damage (Guillodo et al., 2010) and the CFL is second (Cordova, Sefton, & Hubbard, 2010). Ligament laxity is not the only cause thought to attribute to mechanical instability. Hertel (2002) and other researchers have recognized synovial hypertrophy and impingement, osteophytic spurs, and soft tissue impingement as a source for instability (Richie, 2007).

As a clinician it may be difficult to differentiate the cause of instability; however, the integrity and laxity of this talocrural joint can be tested using the clinical exam tests, as described below. After the assessment, a referral back to the physician may be required along with the recommendation for stress radiographs. In the event that the ligaments are too lax to support the ankle, reconstructive surgery may be required (Hockenbury et al., 2005).

**Functional/neurological deficits**

Neurological deficits affect joint proprioception, reflexes, balance, muscular strength, and muscular reaction time (Hertel et al., 2001; Konradsen, 2002; Mattacola & Dwyer, 2002; Richie, 2007; Santilli et al., 2005; Feger, Donovan, Hart, & Hertel, 2015). Decreased impulses from a muscle, fascia, tendon, or articular receptor can result in sensory deficit and produce greater postural sway or reduced postural control. Postural control is defined as “the ability of an individual to maintain his or her center of mass over a single supporting foot” (Richie, 2006, p.48). This is achieved by performing a modified single leg stance test, called a Romberg test (described below). Postural control is considered the best estimate of loss or restoration of functional ankle control (Garn & Newton, 1988; Richie, 2006; Tropp & Odenrick, 1988) and predicts risk for future ankle injury (Richie, 2006). Much of the research shows that postural control can be improved using measures such as bracing, taping and orthoses.

The cavovarus foot is regarded as a risk factor in lateral ankle instability. This foot type is the result of a range of etiologies and pathologies, however, common features include a varus hindfoot, plantar flexed first metatarsal, and high medial longitudinal arch (Burns, Crosbie, Ouvrier, & Hunt, 2006). The varus hindfoot causes overload of the lateral border of the foot, placing the lateral collateral ligaments under continuous stress by the medially displaced moment.

**Chronic Ankle Instability**
arm of the Achilles tendon (Younger & Hansen, 2005). To determine whether a hindfoot varus is flexible or fixed, a Coleman block test can be performed, thus affecting treatment. Nevertheless, there is some controversy to the claim that the cavus foot is more prone to lateral ankle instability. Richie (2006) states that the pes planus foot type is just as susceptible to inversion sprains and based on research reviews has concluded the medical literature unable to verify one foot type more vulnerable to lateral ankle sprains than another.

Individuals affected by CAI report symptoms of pain either chronically or with acute episodes. Common causes for chronic ankle pain are “occult fractures, tendon tears, or ankle soft tissue impingement” (Hockenbury et al., 2005). Typically the location of pain is at the lateral ankle anterior and posterior to the fibula. In the case of lateral ankle sprains, scarring of the torn ATFL and joint capsule can lead to inflamed tissue pinched between the talus, tibia, and fibula, and result in anterolateral impingement (Wolin et al., 1950, as cited in Hockenbury et al., 2005). Chronic pain may also result from an accessory peroneal tendon. This would result in posterolateral ankle pain (Trono, Tuche, Quintart, Libotte, & Baillon, 1999). In cases of medial instability, pain may be felt at the anteromedial aspect of the ankle, along the tibialis posterior tendon and in some cases at the lateral ankle during dorsiflexion of the foot. Individuals with CAI also express feelings of insecurity in their ankle support, repeated turning over on the ankle, swelling, and/or sense of locking, sticking or grinding (Baravarian, 2008; Hintermann et al., 2002; Hintermann, 2003; Chronic Ankle Instability, 2009).

Not only does CAI produce these symptoms, it can have a devastating effect on the joint over time. Chronic lateral instability is an etiological factor in ankle arthrosis, osteochondral lesions of the talar dome, fissures of the fibular tendons, or, as mentioned above, anterior or posterior impingement. Incidence of degenerative arthritis is reported between 13–78%; however, the degree of lateral ligament injury does not correlate with the degree of cartilage damage, and it is unclear as to why (Hintermann et al., 2002). In a study conducted by Hintermann et al., 148 patients with symptomatic CAI that lasted 6 months or more were examined arthroscopically to record and compare structural changes. It was found that 86% of ankles had ruptured or stretched the ATFL, 64% the CFL, and 40% the deltoid ligaments. Of those with lateral ligament injuries, 66% had cartilage damage, and those with deltoid ligament injuries had 98% cartilage damage. In general, medial instability is underestimated. This research shows there is a high correlation between deltoid ligament injuries and cartilage damage of the medial talus (Hintermann et al., 2002).

**Testing**

During the clinical exam, a comparative between right and left ankles should be noted. Standard range of motion (ROM) testing should be performed with attention to articular motion during inversion and eversion, dorsiflexion and plantarflexion of the ankle. Note limitations, rearfoot mobility, and dislocation of the peroneal tendons (Tourne, Besse, Mabit, & Sofcot, 2010). Individuals may express tenderness or pain on palpation around the anterior talofibular ligament (lateral gutter), on the anterior border of the medial malleolus (medial gutter), along the posterior tibial tendon, and/or along the peroneal tendon. Laxity of the joints can be tested as well as postural sway.

**ATFL Instability**

The anterior drawer test allows the examiner to test the integrity of the anterior talofibular ligament. To administer the test, the examiner stabilizes the lower leg of the patient by grasping the anterior tibia just above the ankle with one hand while the other hand cups the heel. The leg being tested should be relaxed and the knee slightly bent. With the ankle in slight plantar flexion, an anterior force is applied to the heel while attempting to move the talus anteriorly in the ankle mortise. Normal anterior talar translation is less 5mm. This test is administered bilaterally and the results are compared (Hockenbury et al., 2005; Sailer, 2007).
An inversion stress test can be used clinically to determine the integrity of the lateral ankle ligaments. With the client seated on the table with their feet hanging, grasp the heel with one hand and the tibia with the other hand. Invert the heel, comparing the results with the contralateral side. In healthy conditions, relative comparisons of two-thirds inversion are to be observed. If greater inversion movement on the affected side is noted, this indicates greater lateral ligament laxity.

**Deltoid ligament instability**

An eversion stress test can be used clinically to determine the integrity of the deltoid ligaments. The client is seated on the table with their feet hanging (Hintermann, 2003, as cited in Hintermann et al., 2002). Grasp the heel with one hand and the tibia with the other hand. Evert the heel, comparing the results with the contra-lateral side. Relative comparisons of one-third eversion are to be observed. Greater eversion movement on the affected side indicates greater deltoid ligament laxity.

Asymmetrical rearfoot valgus and pronation is symptomatic of medial ankle instability. To test, simply observe the rearfoot in weightbearing, looking for asymmetrical planus position of the affected foot. Testing of the tibialis posterior tendon should be performed to exclude its dysfunction.

**Posterior tibial tendon function**

Due to the proximity of the deltoid ligaments and potential involvement in an eversion sprain, its strength may be tested. To do so, palpate the tendon while testing inversion strength by grasping the leg above the ankle and resist the inversion movement of the foot with other hand. Compare both sides, noting pain and weaker strength on affected side.

The strength to the posterior tibialis can also be assessed by performing a double or single heel raise test. Simply view the client from behind and have them either rolling up onto one foot or both, observing the rearfoot motion. Inversion of the rearfoot should be noted. Compare the unaffected side with the affected side. A positive test demonstrates reduced rearfoot inversion and impaired heel rise on the affected side.

**Syndesmotic ligament instability**

The squeeze test is used to test the integrity of these ligaments although it is more commonly demonstrated for an acute injury. The test is performed by placing the fingers over the proximal half of the fibula and the thumb around the tibia, squeezing the bones together. The compression causes the distal tibia and fibula to separate. Pain provides a positive test result. If a positive test result is found, a referral back to the physician is recommended as further diagnostic tests, such as a stress radiograph, may be required.
Postural sway

The Romberg test is used to test the amount of postural sway. This is accomplished by having the individual standing on one foot with their arms crossed over the chest and their eyes closed. A healthy person should be able to hold this position for at least 10 seconds (Richie, 2006, p.48). The ability to maintain their centre of mass over a single support leg shows postural control.

Coleman block test

A Coleman block test is performed to determine if the rearfoot varus position is fixed or flexible. View the client from behind and place a block under the foot but not under the first ray. If the hindfoot varus corrects on the block, it is a flexible varus and considered a forefoot driven varus (Sangeorzan, 2010). Failure to correct indicates a fixed hindfoot varus. Custom orthoses may be used to provide even pressure distribution under the foot and accommodate for the plantar flexed first ray.

Other Diagnostic Tests

Ultrasound, MRI, and stress radiographs are tools often used to determine damage to ligaments and tendon structures (Brotzman, 2011; Tourne et al., 2010). To assess cartilage damage, computed arthrotomography (CTA) has been found to provide a better assessment than that of an MRI (Guillodo et al., 2010).

Treatments

Foot orthoses

Foot orthoses (FO) have been shown to address both mechanisms of CAI by reducing postural sway and improving alignment (Guskiewicz & Perrin, 1996; Orteza, Vogelbach, & Denegar, 1992; Ochsendorf, Mattacola, & Arnold, 2000); however, the literature is limited in addressing the long-term effects of orthosis use and the efficacy of FO in tasks that require cutting movements as it focuses on closed kinetic chain actions.

Guskiewicz and Perrin (1996) reported reduced postural sway in subjects with acute ankle sprains while wearing semi-rigid orthoses (materials not disclosed) derived from 3-dimensional foam block casting in subtalar joint neutral, and Orteza et al. (1992) noted improved balance and reduced pain with jogging when his subjects wore custom direct moulded 1/8-inch Aquaplast orthoses. It is suggested that due to the fact that custom-moulded orthoses add structural support to the sides of the foot and improve alignment, they enhance mechanical stability at the ankle mortise and influence joint mechanoreceptor function and neural feedback (Guskiewicz & Perrin, 1996; Ochsendorf, Mattacola, & Arnold, 2000). It is suggested that “[t]he cradling effect of the orthosis improves alignment of the talocrural joint and places the ankle mortise in a more neutral position. Neutral positioning places the muscle spindles in a position of decreased stretch and activity... application of the orthosis results in increased tactile pressure to the bottom of the
foot. The increased area of stimulation or increase in the receptive field might theoretically result in increased activation of nearby parent afferent fibers” (Ochsendorf et al., 2000). This increase in proprioception may help to reduce reliance on supporting musculature.

FO attribute to improved evertor muscle function as well, since weakness of these muscles is thought to contribute to CAI. “The peroneus longus is normally activated during the middle and terminal stance phases of the stride, providing lateral support during the single-limb stance, when ankle sprains mostly occur... the ankle is protected from lateral sprains when the talocrural joint is in inversion through a dynamic defence mechanism that activates a peroneal eversion response induced by the proprioceptive input of the inversion moment... an inversion sprain may occur while the ankle is in inversion during the weightbearing phase of gait cycle and the evertor muscles are unable to contrast the inversion moment” (Santilli et al., 2005). In newer research, Feger et al. demonstrated that individuals with CAI had sooner muscle activation of the peroneal longus muscle during the entire stride cycle of walking gait than non-CAI individuals. The authors conclude that preactivation of the peroneal longus at heel strike may increase fatigue of that muscle and decrease any available motor-neuron units that could protect against an ankle inversion moment. It is, then, reasonable to think that the neutral positioning of the orthoses may improve the firing of peroneus longus, especially at heel strike.

The same philosophy applies to invertor muscles. There is disparity within the research, as findings show that weakness of ankle invertor muscles contribute to CAI, especially in those with pronated feet (Richie, 2006; Wilkerson, Robert, & Caturano, 1997). Orthoses achieve more neutral positioning and control the rate of pronation. The idea is that pronated feet use up all of their available ROM in the subtalar joint (STJ) because the STJ is functioning at its end ROM. Functioning at an extreme ROM will reduce proprioceptive input and increase lateral sway. Lateral sway over a fixed single leg stance that produces closed kinetic chain rearfoot pronation activates the medial leg muscles that fire and pull the tibia medially over the fixed foot. If these muscles (tibialis posterior, flexor digitorum longus, and flexor hallucis longus) are unable to perform effectively due to weakness, the lateral sway would force the foot and ankle into sudden inversion. This reduces the ability of the invertor muscles to function (Richie, 2006).

Overall, the foot orthoses should enhance sensory input from the plantar surface of the foot, reduce strain or load on the soft tissue supportive structures around the ankle, reduce compensation, improve range of motion, and provide a stable base of support to resist body sway (Richie Jr., 2013).

**Type of FO**

When it comes to the research, no one style of orthoses has been shown more effective than another. Further research is required to determine this; however, in studies focused on orthosis use, custom-moulded seem to improve balance more than OTC (Richie, 2006). Orteza et al. (1992) used neutral moulded (1/8-inch Aquaplast, custom) and unmoulded (flat inserts) orthoses. Comparatively, it was reported that the moulded orthoses significantly reduced pain during jogging in injured people and showed improvement in balance (Orteza et al., 1992). Gukiewicz and Perrin (1996) used custom-made semi-rigid polypropylene orthoses and showed improved balance and postural control, although Hamlyn et al. (2012) also showed improved postural stability when subjects wore full length semi-rigid neutral shell, deep heel cup prefabricated orthoses made from a urethane base and ethylene vinyl acetate top cover. It has been reported that conditions or inventions that decrease pressure under the foot may compromise the somatosensory signals from the plantar foot surface, thereby affecting balance and postural control (Richie, 2007). Researchers also note that although a soft foam surface reduces plantar pressure, it may also cause a decrease in muscular response in correcting body alignment (Wu & Chiang, 1997). This would suggest that the type of material used to fabricate the orthoses should be more firm. For example, Subortholen is a rigid material that is easily heat mouldable and easy to grind. It can be used to fabricate a half-length rigid orthosis or be used as the main stabilizer when fabricating a composite device. High-density ethyl vinyl acetate (EVA) can also be used to fabricate a firmer orthosis while providing some impact absorption. A firmer orthosis may provide better durability, less compression with wear and greater control of abnormal foot mechanics. Based on the literature, Richie (2006) recommends using custom semi-rigid or rigid devices closely moulded to the foot in a subtalar joint neutral position, as well as ensuring the positive cast is intrinsically balanced to perpendicular with no posting. If a post were to be used, a mild medial post is suggested, especially for pronation control. Richie Jr. (2013) recommends using a medial arch flange for most cases of CAI. He states that “a medial flange will act as a block to foot inversion and increase the total contact surface area of the
orthosis,” where a lateral flange will cause the foot to “resist abduction and may force the foot to invert.”

**Modifications to FO**

Research provides anecdotal suggestions to posting, such as adding a lateral rearfoot post (Clanton, 1989). This is especially recommended for those with a flexible rearfoot and lateral heel strike. Contrary to these findings, Hertel et al. (2001) found no decrease in postural sway using rearfoot orthoses, regardless of medial posting (7 degrees), lateral posting (4 degrees), and neutral position (no posting). The authors used posted prefabricated Superfeet Synergizer footbeds and a custom mould orthosis using Aquaplast. The authors suggested that rearfoot posted orthoses are unlikely to improve function related to improved postural control in injured individuals. In a followup study done by Hertel et al. (2001), the same shoe inserts and degrees of posting were re-examined using non-injured subjects. This time the findings demonstrated a significant decrease in frontal sway with the medially posted inserts. The findings showed the greatest frontal plane sway, amplitude and velocity when using prefabricated sprayed ankle orthotic shoe inserts with laterally posted heel wedges. The study also revealed no change in sagittal plane sway for any condition. In comparing the two studies, the authors suggest that the ability of an OTC or non-posted insert may not be as effective to control sway in injured patients and that by reducing rearfoot pronation by way of medial posting, it positively affects balance and postural control compared with laterally posted inserts with poor medial arch contouring (Hertel et al., 2001). Despite these findings, further research is warranted as there is limited information on how well lateral posting affects CAI. However, we may deduce the following: Those with medial ankle instability and/or those with CAI and overpronation may benefit from medially posted FO. Likewise, those that present with lateral ankle instability may benefit from a laterally posted orthosis that fully contacts the arch.

In cases of a cavus foot presenting with a varus hindfoot and plantar flexed first metatarsal, a first metatarsal head cutout is recommended (Younger & Hansen, 2005). Much like the Coleman block test, this allows the first ray to plantar flex thus permitting the rearfoot to shift to a near neutral position. The custom contouring of the FO also provides pressure redistribution by unloading the metatarsal heads and heel and increasing pressure into the midfoot (Burns et al., 2006).

The top cover has also been shown to influence function. Based the findings of Okubo, Watanabe, & Baron (1980), the clinician should consider a more textured top cover. These researchers found improved posture control when their subjects stood on platforms covered with shotgun pellets. It is then presumed that a similar texture enhances pressure sensory input.

**Contraindications to FO**

Based on the above evidence for custom foot orthoses in the treatment of chronic ankle instability, one may deduce that it would be ineffective to provide very soft shells with minimal pronation control or medial arch contact, especially for those individuals that over pronate. However, hard orthoses are often poorly tolerated by the cavus foot due to its naturally limited shock absorption. Researchers also warn against a very high-arched orthosis that may actually increase ankle instability by way of over correction (Burns et al., 2006; Younger & Hansen, 2005). A clinician may be able to determine if over correction is occurring by observing excessive supination of the STJ in static weight bearing and during gait while wearing the orthosis in the shoe. Over time, if left uncorrected, the footwear may show signs of lateral midsole compression.

Despite the lack of evidence, it can be theorized that adding a medial post on orthoses used for lateral cutting movements would negatively impact the foot. It would theoretically place the foot into a more inverted position, thereby encouraging inversion. Similarly, by medially posting a varus rearfoot it would encourage inversion of the foot and promote lateral instability.

**Footwear**

The features of footwear recommended for CAI include a firm heel counter, stable midfoot, and neutral support system. In a study conducted by Robbins & Waked (1998), the researchers found that a firm midsole enhanced tactile sensitivity and proprioception, thereby increasing the awareness of foot position and reducing the time to initiate intrinsic foot muscles (as cited in Hockenbury et al., 2005). High-top shoes have been shown to significantly reduce the amount and rate of ankle inversion. In a comparative study between the Reebok Turf Rat Hi and Reebok Turf Rat Lo football shoes, Ricard, Schulthies, & Saret (2000) were able to show a decrease of inversion by 4.5 degrees and an average reduced rate of inversion by 73 degrees per second. The authors believe that these reductions may allow time for the body’s protective mechanism to respond, thereby reducing potential ankle sprains.
When fitting for the high-arched foot, footwear should include lace up closures as well as extra-depth and removable insoles to accommodate a semi-rigid orthosis (Younger & Hansen, 2005). If off-the-shelf footwear is insufficient in providing adequate lateral stability, Janisse & Janisse (2008) recommend modifying the lateral aspect to increase stability. A lateral flare or buttress can be applied to an everyday shoe for those with extreme lateral instability.

**Contraindications to footwear**

It is contraindicated that individuals be fitted with a pronation control shoe if the foot is in rearfoot varus (Janisse & Janisse, 2008). Anecdotally, this would result in overcorrection of the foot and force an increased varus or lateral heel strike thereby promoting lateral ankle instability. Similarly, those that hypersupinate during midstance are to avoid pronation control shoes. Even when treating overpronation or medial instability, neutral footwear combined with foot orthosis is suggested. When considering the highly active individual, a wide base of support has the potential to increase ankle inversion. If they were to land on the outer corner of the footwear it could increase the moment arm and potentially increase inversion velocity.

**Physiotherapy**

The goal of rehabilitation is to develop neuromuscular control and strength, thereby allowing the ankle and foot to be better controlled and protected during stance and impact. Physiotherapy exercises are performed to improve ROM & strength, neuromuscular control, proprioceptive and balance training, and activity specific training. (Mattacola & Dwyer, 2002; Webster, Pietrosimone, & Gribble, 2016). Based on the works of Feger et al. (2016) and Webster et al. (2016), the physiotherapist should not only focus on the ankle’s local structures, but also account for all altered lower extremity muscle activity, such as with the anterior tibialis, lateral gastrocnemius, rectus femoris, biceps femoris, gluteus medius, and gluteus maximus. Implementing alternative therapies, such as trigger point dry needling, has shown to improve pain and functional outcomes. Salom–Moreno, Ayuso–Casado, Tamaral–Costa, Sanchez–Mila, Fernandez–de–las–Penas, & Albuquerque–Sendin (2015) proved that one month after completing proprioceptive and strengthening exercise therapy in combination with trigger point dry needling into the lateral peroneus muscle, individuals had greater improvements in function and decrease in pain intensity than those doing only the exercise rehabilitation.

**Taping**

Ankle taping, especially as used by athletes, has its advantages and disadvantages. The pros include greater perceptions of stability and confidence during dynamic activity (Halim–Kertanegara, Raymond, Hiller, Kilbreath, & Refshauge, 2017), controlled joint mobility, and less bulk around the ankle. The disadvantage to tape is that it can weaken throughout the activity and continued use may cause a skin allergy (Dizon & Reyes, 2010). In a study conducted by Ricard, Sherwood, Schulties, & Knight (2000) there was no difference between the effectiveness of taping on the skin versus prewrap. Both versions reduced the amount and rate of dynamic ankle inversion. Tape conditions allowed greater time to maximum inversion than the non–taped conditions and decreased ankle inversion velocity by 38–40%. It is suggested then that this decrease in inversion velocity and increase in time to maximum inversion may allow more time for the body’s natural reflex mechanism to activate and possibly prevent or reduce the severity of an inversion injury (Ricard et al., 2000).

**Bracing and ankle–foot orthoses**

Bracing and ankle–foot orthoses (AFO) may be used to improve ankle stability. A hinged AFO, like a Richie brace, will provide medial–lateral stability while allowing for ankle dorsiflexion and plantarflexion (Janisse & Janisse, 2008; Mattacola & Dwyer, 2002). If the motion required to control is more than frontal plane, then a solid AFO using moulded thermoplastic is recommended, such as the Arizona brace (Janisse & Janisse, 2008).

In a research review of ankle injury prevention strategies, the authors concluded that ankle bracing constructed with semi–rigid or rigid stabilizers, lace-up braces, and stirrups were effective in resisting ankle inversion and provided a certain degree of prevention for ankle sprain recurrence (Dizon & Reyes, 2010). Bracing can also improve proprioception and sensory feedback (Mattacola & Dwyer, 2002). The benefit to bracing is that, once fitted, the brace can be reapplied by the individual themselves. It is adjustable, provides stable restriction, and can be used for a longer period of time. The potential downside to ankle bracing is the risk to injury above the ankle, such as the knee; however, this requires further investigation (Dizon & Reyes, 2010).

**Surgeries**

With chronic ankle instability, surgery may be required, especially if nonsurgical management fails to alleviate symptoms. The goals are to achieve
tightened ligaments or capsular tissue. Tightening can be achieved by way of reconstruction and tendon grafts, typically using the peroneus brevis (Tourne et al., 2010).

Chronic ankle instability is the most common ankle injury resulting in repeated injury with potential to lead to osteoarthritis, persistent pain and reduced postural control. Several treatment options are available and are shown to reduce the reoccurrence of ankle injury; however, it would be beneficial for future studies to examine the long-term effects of orthosis use on CAI, the efficacy of lateral posting on FO, and the effectiveness of FO on cutting and jumping movements.

References


Ankle Ligament Sprains

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Key Messages

- Ankle sprain is a mechanism of injury rather than a diagnosis (Rolf, 2007). Sprains most commonly occur with the foot in inversion and plantarflexion (Matharu, Najran, & Porter, 2010; Tenforde, Yin, & Hunt, 2016). This position pulls the ligaments tight and the talus is wedged anteriorly thereby positioning it also into a less stable position (Gray, 1918).
- Lateral ligament structures are seen to be damaged in as high as 75% of ankle sprain injuries (Matharu et al., 2010).
- Ankle injuries may have permanent effects on athletic performance (Tenforde et al., 2016).
- In sports involving running and jumping activities, 25% of the injuries are at the ankle (Tenforde et al., 2016).
- In sports like basketball and soccer, 30–45% of the injuries occur at the ankle (Tenforde et al., 2016; Nery, Raduan, & Baumfield, 2016; Simons & Kennedy, 2004).
- 95% of injuries causing foot, heel and ankle pain will heal themselves over time (Scribd, n.d.; Thompson, n.d.).
- Healing may take 6–12 months for recovery. It is possible for a complete recovery as fibers remodel and strengthen.

Keywords

anterior drawer test, ATFL, eversion, external rotation, inversion, PTFL, squeeze test, talar tilt, talocrural joint

Introduction

The ankle joint is a hinge joint comprised of the distal tibia and distal fibula bones of the leg and the talus which fits in between them (Gray, 1918; Bull p.349). Together these bones comprise the talocrural joint (Bull p. 349, Gray 1918). Helping to stabilize these bones are connective tissues that play a role in the ankle’s function. The ligaments of the ankle play an important function of holding the bones and joint in a stable position. The talus is held directly into place through ligamentous connections to both the tibia and fibula. Ligaments supporting the ankle include the following:

- Anterior talofibular ligament (ATFL) is the most common site of injury when the ankle is “sprained” It is on the lateral side of the ankle joint and this is the only ligamentous structure at the front of the ankle (Taylor, Ford, Nguyen, Terry, & Hegedus, 2015; Simons & Kennedy, 2004)
- Calcaneofibular ligament (CFL) (Taylor et al., 2016). This is the second most commonly injured ligament (Taylor et al., 2016)
- Anterior and posterior talofibular ligament (Gray, 1918; Simons & Kennedy, 2004). The posterior talofibular ligament is the third most commonly injured
- Anterior inferior tibial fibular ligament (Gray, 1918; Simons & Kennedy, 2004).
- Deltoid ligament – this is the ligament on the medial side. It is relatively resistant to sprains (Gray, 1918; Simons & Kennedy, 2004).
- Talocalcaneal ligament – this runs from the posterior aspect of the talus to the calcaneus. (Gray, 1918; Simons & Kennedy, 2004).
- Talocalcaneal interosseous ligament – this is a strong ligament deep within the subtalar joint (Gray, 1918; Simons & Kennedy, 2004).
Ligaments can be injured when they are forced to move further than their normal range of motion will allow for. When a stretching, tearing or rupture of a ligament occurs it is referred to as a sprain (Scribd, 2003; Stone Clinic, n.d.).

**Grade 1** is the mildest (Scribd, 2003; Stone Clinic, n.d.; Simons & Kennedy, 2004) and it roughly corresponds to a stretch of the ligament.

**Grade 2** is a moderate injury (Vancouver Sport Physio, n.d.) and it roughly corresponds to a partial tear of the ligament.

**Grade 3** is a severe injury (Scribd, 2003; Simons & Kennedy, 2004) and it roughly corresponds to a complete rupture of the ligament. It may be accompanied with fractures or a dislocation in some cases.

The Interosseous membrane (Gray, 1918; Simons & Kennedy, 2004), which joins the tibia and fibula, is also a stabilizer of the ankle joint. It is the fourth most commonly injured region of the ankle.

When Grade 1 sprains occur the ligament is stretched but not torn. There is minimal swelling and some pain. It is possible to gradually return to activities within 2 weeks (Scribd, n.d.; Simons & Kennedy, 2004).

Grade 2 sprains cause the ligament to be partially torn. Some bleeding from the ligament may occur. Bruising may appear in a few days (Dubin, Comeau, McClelland, Dubin, & Ferrel, 2011; Scribd, n.d.; Simons & Kennedy, 2004; Bouchard, 2004). Due to gravity, the bleeding may not be near the site of injury but distal (Scribd, n.d.). A return to activities with proper rehabilitation can often be possible within 2–6 weeks.

When Grade 3 sprains occur the ligament is completely torn or ruptured. There is severe pain initially. Pain may subside more quickly than a grade 2 injury because the ligament is not under stretch but in fact ruptured. Surgery may be required and the return to activity may exceed six weeks. There may be no palpable end point see with ligament testing (Scribd, n.d., Simons & Kennedy, 2004). It may not be possible to weight bear on an unstable ankle (Bouchard, 2004).

While less common, eversion ankle sprains comprise 5–10% of all ankle sprains but they may take longer to heal than inversion sprains (Scribd, n.d.). Often if this kind of injury is seen, it represents a major injury (Mendel et al., 2010).

**Pathology**

Due to the anatomical configuration of the talocrural joint, it is vulnerable to a variety of injuries. Forces that the foot encounters can result in traumatic and overuse injuries. Examples of injuries include landing on another person’s foot, stepping on uneven ground (Mendel et al., 2010), sudden changes of direction, and stepping off the side of a curb. This puts excessive force on the ligaments and it takes ligaments and tendons beyond their anatomical limits. This results in sprains and tears.

Predisposition to ankle sprains and an increased incidence is seen in individuals with varus alignment of the lower limbs, calf tightness, and a history of previously injured ankles that have not healed. Sports that involve quick changes in direction may also contribute to the injury.

**Differential Diagnosis**

Due to the complex nature of the surrounding anatomical structures it is possible to see some potential for misdiagnosis with injuries in the ankle. Avulsion fractures, where a small component of bone is pulled off at the ligament or tendon attachment is one such type injury. A fibular avulsion fracture can be diagnosed for some sprains where the pain is severe accompanied with a hematoma in the skin. The symptoms of a fracture are similar, but more extreme, than a sprain of the ankle.

Another bone susceptible to avulsion fracture through similar mechanism as an inversion sprain is the fifth metatarsal. Fracture here may occur as the peroneus brevis muscle resists ankle inversion. Its insertion is into the base of the fifth metatarsal. A hematoma is often found over the fifth metatarsal and palpation pain can be over the cuboid and fifth metatarsal.

Calcaneal fracture may also be misdiagnosed where heel contusion exists (Scribd, n.d.). Two other misdiagnoses to consider are osteochondral lesions and cuboid syndrome/subluxation.

**Common Testing**

Identification of the joint position at the time of injury can help the examiner to determine possible ligamentous damage. Prior injuries are a major predictor of problems (Simons & Kennedy, 2004) Passive range of motion tests such as ankle inversion from a neutral and plantar flexed position can help isolate specific ligaments. Palpation at the exact location of pain is also helpful indicators of ligament...
damage. Methods that test the integrity of the ankle ligaments include the anterior drawer test for ATFL sprains. It is done by holding the lower leg stable and cupping the calcaneus with the other hand while applying a posterior to anterior force to test the ankle. The anterior drawer test is specific for the ATFL and can be done with minimal pain or guarding (Simons & Kennedy, 2004, Ankle and Foot Injuries, n.d.)

The talar tilt test is more specific for calcaneofibular ligament sprains. The examiner holds the lower leg and cups the calcaneus while inverting and everting the foot.

Two provocative tests for syndesmotic ligament injury are the squeeze test and the external rotation stress test (Ankle and Foot Injuries, n.d.).

Direct palpation along the plantar aspect of the foot can help identify bones that may be fractured such as the base of the fifth metatarsal.

Ottawa ankle rules are a good method to rule out fractures (Matharu et al., 2010) and to determine the need to refer to others if needed. In short, the Ottawa rules suggest that if there is pain near the malleoli (Briggs & Combs, 2009; Mai & Cooper, 2009), bony tenderness at the point of the malleoli or in the distal 6 inches of either malleolus (Briggs & Combs, 2009; Matharu et al., 2010), and the inability to bear weight for 4 steps after injury (Briggs & Combs, 2009; Mai, 2009), further investigation with X-rays may be required. These rules are regarded by these sources as being accurate for older children, but one study has expressed doubt if they are as accurate for young children (Runyon, 2009). In subsequent follow-up, Ottawa Ankle Rules are the most valid predictor (Jonckheer, Willems, De Ridder, Paulus, Holdt Henningsen, San Miguel, De Sutter, & Roosen, 2016)

Other simple tests that can be incorporated into the physical exam are the Thompson squeeze test which is used to rule out complete rupture of the Achilles tendon. Checking sensation in the foot can be used to rule out possible nerve damage. Calcaneal contusion may also be suspected if there is pain in the heel without the ability to bear weight (Scribd, n.d.). Lastly, if immediate swelling interferes with physical testing, rechecking the ankle in 5 days may be required to accurately assess the ankle (Matharu et al., 2010). If further testing is needed, a Telos stress X-ray may be taken. The Telos is a device that stresses the ligament on X-ray.

**Contraindications**

Ultrasound is often suggested to reduce the swelling on sprained ankles. The use of ultrasound is contraindicated on children under 18 years of age.

Taking of non-steroidal anti-inflammatory drugs (NSAIDs) within the first 48 hours after injury (Bupa, n.d.) is contraindicated. They decrease the inflammation that is an important part of the healing process.

Wearing shoes that are excessively worn on one side of the heel is also contraindicated in the healing process as they may inadvertently destabilize the ankle (Bupa, n.d.).

Exercising when you are tired is also not recommended because the ankle muscles offer decreased support when fatigued (Bupa, n.d.).

The mnemonic HARM outlines other contraindications in the first 72 hours following a suspected ankle sprain (Bupa, n.d.):

- **H is for Heat.** Heat encourages blood flow in the area and increases swelling
- **A is for Alcohol.** Drinking alcohol increases bleeding into the tissues
- **R is for Running.** Running can increase the damage level if it is pursued in the first 72 hours
- **M is for Massage.** Massaging a sprain can increase the level of bleeding and swelling

Casting a sprained ankle is contraindicated. Ankle injuries casted for six weeks or more can cause the repairing collagen fibres to grow in a disorganized fashion. This leads to scar formation and weakness (Stone Clinic, n.d.). Casting has also been shown to affect proprioception negatively. Proprioception is required to avoid repeat injuries that can lead to chronic instability (Holmes, 2009; Zhao & Wang, 2015).

**The Patient Perspective**


**Instability** - The feeling of the ankle giving way—especially in the early phase of recovery (Bupa, n.d.; Taylor et al, 2015). The instability is more commonly described after multiple injuries (Zhao & Wang, 2015). If the instability is chronic, you will want to refer the patient to a doctor. (Zhao & Wang, 2015).
Stiffness – Usually described after resting and gets better temporarily after exercise (Bupa, n.d.).

Numbness – Any sensation of “pins and needles” indicates the nerve is involved.

Popping and Cracking – Normal ankle popping has been reported where it takes place without pain and instability. Sensations like these at the time of injury usually indicate the tearing of tissues and/or fractures. Abnormal popping is where the tendons jump out of position and is accompanied by pain. Such cases need to be referred to the doctor. (Simons & Kennedy, 2004).

Causes the patient may describe:

- Walking on uneven surfaces.
- Foot slipping on a curb.
- Slip while going down the stairs.
- Losing balance on high heels (Bupa, n.d.).
- “Turned ankle over” while running or during a sudden direction change in sports.

**Common Treatment**

Each ankle sprain injury is unique and its treatment is not always the same. Some self-help suggestions can be detrimental if not introduced at the appropriate point in the stages of healing. A referral to a qualified rehabilitation provider (i.e., physiotherapist, athletic therapist) is highly recommended since the injured person may require specific guidance. Refer to a physician if the patient can’t walk 4 steps (Bupa, n.d.), or if it remains significantly sore for more than 4 days after injury or if the ankle is numb (Richards, Marks, Sekyi-Out, & Yee, 2004; Bouchard, 2004; Bupa, n.d.).

When mild sprains such as a grade 1 occur, protection, rest, ice, compression, and elevation (commonly known as PRICE) for the first 48 hours are recommended as standard early treatment (Bupa, n.d.; Dubin et al., 2011; Stone Clinic, n.d.). It has been shown that this methodology gives results not significantly different than using intensive recovery physiotherapy early in the recovery (Hing, Lopes, Hume & Reid, 2011). Once 48 hours post injury has occurred, a gradual reintroduction to movement can commence (Bupa, n.d.; Dubin et al., 2011; Stone Clinic, n.d.). Footwear during this time should accommodate swelling initially and assist in supporting the ankle by having a wide stable base (Bupa, n.d.). The outsole of footwear should be inspected and should not be accessibly worn down at the heel (Bupa, n.d.). Properly fitted shoes decrease the chance of injury and increase the stability of the ankle (Thompson, Vancouver Sports Physio, n.d.).

Protection to the ankle joint can be accomplished through use of various types of ankle braces (Bupa, n.d.; Dizon & Reyes, 2010; Gross & Liu, 2003; Taylor.; Knapik et al., 2010; Rolf, 2007) (7) and for a short-term ankle taping can be utilized (Bupa, n.d.; Dizon & Reyes, 2010; Ewalt, 2010; Stone Clinic, n.d.). In cases of a severe sprain, a walking boot or short-leg cast is sometimes utilized. Early protection that allows for some dorsiflexion and plantar flexion of the ankle is superior to rigid cast immobilization in terms of patient satisfaction, strengthening, and return to activities (Richards et al., 2004; Bouchard, 2004).

Ice packs can be put on the ankle, wrapped in a wet towel, for 15–20 minutes every 2 hours for the first 48–72 hours. When icing, elevation of the affected ankle will also help to reduce excessive swelling. Utilizing an ice pack on your ankle while sleeping is contraindicated (Bupa, n.d.).

Compression is given through compressive wrap (commonly known as a Tensor bandage) (Dubin et al., 2011).

Once the initial injury is over, home exercises prescribed by a qualified professional such as a physiotherapist can be done (Bupa, n.d.). Heating the area before exercise and icing afterwards is recommended during the rehabilitative process (Stone Clinic, n.d.).

Early mobilizations for the ankle are listed below:

- Move foot up and down like on a gas pedal
- Move the ankle in clockwise and counterclockwise directions
- Stretch the Achilles tendon with a belt under the foot and pull it towards your body. Do this while you are non-weight-bearing
- Stand up and shift your weight forward and back, left and right

More advanced exercises can be done with Thera-Bands and balance spheres. Gradual strength training in the lower leg muscles is important for return to running and jumping activities (Rolf, 2007).

Proprioceptive and functional balance training is a key way to prevent injuries (Bouchard, 2004, Hrysomallis, 2007; Rolf, 2007; Valovich McLeod,
There are a number of different methods to do this. A wobble board is one way. It is a flat board with a non-slip surface on which a person stands. There is a half sphere of various radii under the board. The person stands on it and attempts to remain standing erect. In order to do that, the person needs to coordinate the flexion of muscles to maintain balance. Proprioception is required to achieve balance (Hübscher, Zech, Pfeifer, Hänsel, Vogt, & Banzer, 2010; Valovich McLeod, 2008). A different way to do this is to put the sphere on sandals. The subject is to remain as motionless as possible. It has been shown that training with these sandals can decrease the incidence of ankle sprains. It also increases the strength and activity of the intrinsic foot flexors (Michell, Ross, Blackburn, Hirth, & Guskiewicz, 2006).

Walking, running, and jumping on multiple surfaces makes for gradual progression to normal activities (Thomas, n.d.). In sports, it may be wise to wear a brace for 6 months to prevent further injury (Simons & Kennedy, 2004).

Footwear must be appropriate for the person (Michel, Kälin, Metzger, Westphal, Schweizer, Campe, & Segesser, 2009; Scribd, n.d.):

- Rigid shoe for pronators.
- Flexible cushioned shoe for supinators.
- Heel counter design is important to control heel motion.
- The last type is an important consideration. A slip-lasted shoe is more flexible, a board-lasted shoe is less flexible, and a combination-lasted shoe is between the two (Scribd, n.d.).
- The midsole needs to be designed to control the medial aspect of foot motion (Scribd, n.d.).
- The last shape is also an important consideration and the shoe needs to match the shape of the foot. If a person who needs a curved-lasted shoe wears a straight-lasted shoe, it will cause increased wear on the lateral side and increased risk of ankle sprains (Pattison, 2005).
- High top shoes for basketball with or without taping or bracing can decrease injuries, but they do impair athletic performance. It may be wise to tighten the brace/shoes in midgame (Richards et al., 2004).

Training for sport-specific stressors of the ankle is a key factor in the rehabilitation and further prevention of further ankle sprains (Hübscher et al., 2010; Hrysomallis, 2007; McHugh, 2010; Rolf, 2007; Vancouver Sports Physio, n.d.). An adequate warm-up will decrease the chance of injury. The best preventative factor is the skill of the person. Safe controlled movements and looking at the environment that the exercise is performed in decrease the chance of injury. Doing strength and endurance exercises before the season begins if the patient performs seasonal sports (Bupa, n.d.). Muscle endurance can be gained by bicycling with the pedal under forefoot, and ankle raises with knees bent and straight (Rolf, 2007).

Biomechanical and structural deficiencies need to be dealt with through the use of orthoses (Scribd, n.d.).

**Key Considerations**

- Training.
- Warm-up.
- Proprioceptive exercise.

Sprains are most commonly seen in the “weekend warrior” and those who don’t do adequate training and warm-ups!

**References**


Ankle Impingement Syndromes

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Key Messages

- Impingement syndromes in the ankle can occur at various anatomical regions of the joint.
- Region developments can often be traced to the nature of a single or multiple ankle joint injuries.
- Identifying other soft tissue limitations to joint range of motion can improve outcomes.
- A commonly occurring ankle inversion sprain injury may lead to impingement syndrome.
- Imaging results to discover the reason for the impingement help dictate the preferred treatment.
- Pedorthic intervention to reduce joint impingement can aid in pain reduction and in reducing joint irritation.

Keywords

antero-lateral/medial complex, compression, fibrosis, meniscoid lesion, microtrauma, os trigonum, osteophytes, spurs, Stieda process, synovitis, tibiotalar joint complex

Introduction

Ankle impingement syndromes are classified by the injury site as anatomically defined by the relation to the tibiotalar joint complex. It is important to note that these injury sites may have similar causes but they do present with different signs and symptoms and radiological imagery findings. These conditions are due to post traumatic events that cause chronic ankle pain and instability that lead to biomechanical range of motion (ROM) restrictions affecting the tibiotalar joint. Persistent symptoms can be caused by soft tissue or osseous abnormalities. The ankle impingement can result from tendon injuries, synovial proliferation, bone spur (osteophyte) formation, mechanical instability, ligamentous scarring, and hypertrophy (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006).

Soft tissue and osseous diagnosis can be aided by various imaging tools such as the use of normal radiology (X-rays) to detect osseous proliferation, MRI (magnetic resonance imaging) and CT scan (computed tomography) for soft tissue abnormalities, and MRA or CTA (magnetic resonance arthrography or computed tomography arthrography) for intra-articular abnormalities (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson, 2007; Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006).

Management and treatment of the ankle impingement syndrome are mostly conservative and non–invasive, but the type of injury may lead to arthroscopic surgery and debridement of the chronic, unresolved, diagnosed ankle impingement (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006).

Pathology

Anterior ankle impingement is a common cause of occult fractures, osteochondral talar lesions, and intra-articular foreign bodies (Donovan & Rosenberg, 2010; Robinson, 2007; Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006). Clinical diagnosis can be accomplished for some of the ankle impingements but there are those that are more difficult to detect and determine. This is because differential diagnosis and physical findings can question the exact cause due to mimicking or co-existing with a variety of other conditions such as chronic ankle dysfunction, cartilage damage,
ankle pain in athletes such as soccer players, runners, volleyball players, and ballet dancers (Robinson, 2007; Tol & van Dijk, 2006; van Dijk, 2006). The injury is due to direct microtrauma or repeated stress of the ankle in supination or dorsiflexion (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006). It is characterized by stiffness with limited ankle dorsiflexion ROM and instability. Soccer players develop the condition due to the biomechanics of the striking action and the force of the ball impact over the anterior tibiotalar joint area. Ballet dancers develop the condition due to the forces of the foot ROM during routines.

Anterolateral ankle impingement is a less common occurrence causing chronic ankle pain. Primarily, this painful condition results from hypertrophied soft tissue impingement rather than ankle instability or tendon subluxation (Robinson & White, 2002).

Anteromedial ankle impingement is an uncommon injury for chronic ankle pain and instability. It is a rare occurrence but when diagnosed it is found to be associated with other underlying causative factors (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson & White, 2002; Umans, 2002).

Posteromedial ankle impingement is the least recognized type of ankle trauma. It has been speculated that the injury may be a series of events due to a severe inversion mechanism (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Umans, 2002; van Dijk, 2006).

Posterior ankle impingements are synonymous with names such as os trigonum syndrome, talar compression syndrome, posterior tibiotalar compression syndrome, and posterior block. Symptoms are caused by compression of soft tissue and bony abnormalities between the posterior tibia and calcaneal tuberosity (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson, 2007; Robinson & White, 2002; Umans, 2002; van Dijk, 2006).

Extra-articular ankle impingement, via severe pes planus deformity, may present with lateral ankle pain in the region bounded by the anterior fibula and the sinus tarsi (Donovan & Rosenberg, 2010). Lateral ankle pain can be attributed to extra-articular hindfoot impingement including talocalcaneal and subfibular impingements (Donovan & Rosenberg, 2010).

Each ankle impingement syndrome, as similar as it may be, has subtle differences and causative factors.

Impingement of the anterior ankle causes tibiotalar spurs to form at the margin of the tibial plafond and at the opposed margin of the talus within the anterior ankle joint capsule (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson, 2007; Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006). The spur locations contribute to the painful limitation of the ankle ROM (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson, 2007; Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006).

Repetitive direct microtrauma such as kicking a soccer ball in plantar flexion can induce bone spurs at the cartilaginous rim of the articular margins between the anterior tibia and talus of the ankle joint without concomitant degenerative changes (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson & White, 2002; Umans, 2001). Osteophytes form within the anterior joint capsule, believed to be caused from the repetitive supination impaction injury that repairs itself with scar tissue, proliferative fibrosis, and subsequent ossification (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson & White, 2002; Umans, 2001).

The anterolateral ankle complex is a triangular shaped recess of the ankle that is made up posteriorly by the anterolateral tibia and talus and anteromedial fibula, and anteriorly bounded and reinforced by the joint capsule which is comprised of the anterior inferior tibiofibular, anterior talofibular, and the calcaneofibular ligaments (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson, 2007; Robinson & White, 2002; Umans, 2001).

The injury is common in young athletes and results from a typical ankle inversion sprain when the ankle is forced into plantar flexion and supination. This mechanism of injury sprains the lateral collateral ligaments of the ankle, namely the anterior talofibular ligament, and can lead to disruption of the syndesmotic fibrous tissue (Donovan & Rosenberg, 2010; Robinson & White, 2002; Umans, 2001). In most cases the initial sprain is minor and may not be recalled, but repetitive microtrauma and subclinical microinstability may cause a cycle of scarring and inflammation leading to soft tissue abnormality (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson, 2007; Robinson & White, 2002; Umans, 2001). These chronic soft tissue changes create hypertrophied synovial tissue that forms a fibrous mass within the joint capsule that causes pain and a mechanical impingement, but not instability (Donovan & Rosenberg, 2010; Robinson,
Ankle Impingement Syndromes

2007; Robinson & White, 2002; Umans, 2001). During eversion or dorsiflexion of the ankle, compression of the affected anterolateral soft tissue causes the symptoms (Hopper & Robinson, 2008).

Other described causes of the impingement include a hyalinized connective tissue mass called a meniscoid lesion, an accessory fascicle of the anterior inferior tibiofibular ligament, contact between the talus and anteroinferior tibiofibular ligament surfaces if laxity about the anterolateral ankle, and a chondral injury of the talus due to abrasion (Hopper & Robinson, 2008).

Anteromedial ankle impingement is due to injury of the five superficial and deep deltoid ligament structures of the ankle which originate from the tip of the medial malleolus. The impingement arises from lesions related to the injury of the anterior tibiotalar ligament or of the overlying anteromedial joint capsule (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006). The common mechanism of injury is inversion with a possible rotational component about the tibiotalar joint and capsule (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson & White, 2002; Umans, 2001).

Inversion ankle injury to the anterior taloﬁbular ligament creates compression of the joint structures causing hypertrophic changes and ﬁbrosis of the posteromedial tibiotalar capsule and posterior deltoid ﬁbres, speciﬁcally the posterior tibiotalar ligament and to a lesser extent the ﬂexor tendons that course between the talus and medial malleolus (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008).

The posterior ankle impingement syndrome is related to osseous and soft tissue pathology. Several possibilities exist that can impinge the ankle and its ROM, such as a large “os trigonum” or Stieda process, an enlarged lateral talar tubercle, a prominent calcaneal tuberosity, and a fracture or bony fragment (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Umans, 2002; van Dijk, 2006). Soft tissue pathology found in conjunction with the osseous types are the posterior capsule, posterior ligaments of taloﬁbular and tibioﬁbular and the intermalleolar that become compressed (Hopper & Robinson, 2008). Soft tissue changes include posterior ankle and subtalar synovitis, FHL tenosynovitis, and localized posterolateral ankle sinusitis.

Impingement of the ﬂexor hallucis longus (FHL) tendon occurs as it anatomically runs in its groove between the smaller medial and larger lateral talar tubercles. The “os trigonum” or accessory ossicle forms and articulates with the talus by a cartilaginous synchondrosis. As the name suggests, the “os trigonum” is triangular in shape and has three articulating surfaces. These consist of the anterior surface with the talus, the posterior facet attaching the posterior taloﬁbular and talocalcaneal ligaments, and the inferior articulation with the superior surface of the calcaneus (Hopper & Robinson, 2008). A retinaculum joins the space between the bony prominence the adjacent lateral talar tubercle. Pain is caused by the disruption of the synchondrosis due to repeated trauma and chronic inﬂammation (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Umans, 2002; van Dijk, 2006). This injury is noticed in ballet dancers.

The condition arises from forced, repetitive plantar ﬂexion of the foot and ankle, commonly seen in ballet dancers, which compresses the soft tissue between the posterior process of the calcaneus and the posterior tibia needed en pointe. This entraps and inﬂames the intervening soft tissue. Soccer players and runners subacutely experience trauma due to compression of the posterior ankle involving the posterior taloﬁbular and calcaneofibular ligaments (Hopper & Robinson, 2008; van Dijk, 2006).

Extra-articular ankle impingement, otherwise known as lateral hindfoot impingement, is believed to be a result of a lateral shift of weight-bearing forces from the talar dome to the lateral talus and ﬁbula, and possibly to talocalcaneal joint subluxation (Donovan & Rosenberg, 2010). Causes of this impairment include PTTD, healed intra-articular calcaneal fractures, neuropathic arthropathy, and inﬂammatory arthritis (Donovan & Rosenberg, 2010).

Common Testing

Diagnosis of anterior ankle impingement is mainly clinical. Anterior ankle and midfoot pain radiates to the lateral malleolus and pain is elicited by pressure on the anterior ridge of the tibia while going through forced ROM of dorsi/plantar flexion of the foot (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006). Symptoms are progressive and are caused by soft tissue swelling of the associated anterior synovial thickening and scarring (hypertrophy), decreased ankle dorsiflexion ROM, and a possible palpable bone spur over the anterior ankle (Donovan & Rosenberg,
Anterolateral ankle impingement through clinical assessment diagnosis in most cases is sufficient. It is characterized by anterolateral ankle tenderness, swelling, and pain. The symptoms are exacerbated with ankle eversion or dorsiflexion and single-leg squatting with no associated ligamentous ankle instability (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006). Patients present with anteromedial pain over the anterior tibiotalar ligament that is exacerbated by dorsiflexion and inversion as the inflamed joint compartment is compressed (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002). Symptoms worsen with weight-bearing activities and joint ROM is limited.

The usual lateral ankle symptoms of posteromedial ankle pain are noted early but mild discomfort related with activity continues 4 to 6 weeks post-injury. There is pain and tenderness upon posteromedial palpation during inversion and plantar flexion (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008). Clinically, the main differential diagnosis is posterior tibialis tendon dysfunction (PTTD). Patients with PTTD present with a pes planus foot type and lack heel inversion when standing on tiptoes.

Patients with posterior ankle impingement present with gradual onset of posterior ankle pain that can radiate to the posterolateral ankle, swelling, and tenderness on palpation between the Achilles and peroneal tendons. Pain can be elicited by passive plantar flexion of the foot and by plantar flexion or dorsiflexion of the great toe and limited ROM of these great toe actions (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Umans, 2002; van Dijk, 2006). This is associated with the FHL tendon as it courses within its retinacular groove behind the talus (Hopper & Robinson, 2008).

Extra-articular ankle impingement depends on the flatfoot and rearfoot valgus deformity. Rigid pes planus foot deformities have decreased ROM about the midfoot, hindfoot, and ankle dorsiflexion. Patients with PTTD in the early stages present with medial ankle pain, while the chronic patients present with lateral ankle pain and lateral impingement (Donovan & Rosenberg, 2010). Pain contribution is due to osteoarthritic changes of the subtalar, talonavicular, and calcaneocuboid articulations.

**Imaging**

Primarily, diagnosis for ankle impingement syndrome is attempted clinically; however, imaging and ultrasound can aid in the differential diagnosis of other causes. The conventional weight-bearing ankle radiograph is useful for assessing the medial longitudinal arch, rearfoot valgus, and the lateral hindfoot bony impingement. Also, evaluation of the impingement spurs including appearance, size, and positioning can be done through X-rays of the tibiotalar joint space. Other internal derangement is better viewed by CT, MRI or MRA.

The MRI or MRA seems the more useful tool to detect soft tissue abnormalities such as hypertrophy, evaluation of the anterolateral recess, and detection of pathological soft tissue or capsular irregularities such as osteophytes such as bony prominences or spurs, and chondral damage (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Umans, 2002) such as fractures and fragments, and the os trigonum, and scarring and synovitis within the joint capsule, cartilaginous synchondrosis, the FHL integrity, and pseudoarthritis (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006).

**Common Treatment**

Conservative therapy and management practices are most successful for ankle impingement syndromes. The conservative measures include rest and limiting the stress–causing activity, physiotherapy, non-steroidal anti-inflammatory drugs (NSAIDs), and ankle bracing, custom foot orthoses, and functional footwear. The treatment plan duration may be 4 to 6 months to return to activity (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007);
Physiotherapy will include stretching and strengthening exercises that aid the ankle healing, foot and ankle proprioception and ROM such as Thera-Band, wobble board, calf stretches, lunges, and dorsi-/plantar flexion exercises.

Ankle bracing will aid the chronic foot and ankle instability by providing additional medial and lateral support. The bracing options could be custom ankle-foot orthosis (AFO) such as the Richie brace and other custom plastic AFO device styles, depending on the prescribed condition and clinical assessment that elicits the ankle pain. There are a variety of off-the-shelf options that could be attempted, for example, Active Ankle brace, DonJoy, and Aircast products.

Pedorthics provides custom foot orthoses that support the foot and ankle from deviating in a more varus or valgus rearfoot position that can lead to the pain and discomfort of an impingement. The foot orthosis prescription may include: deep heel cups; first met cut outs; forefoot varus posting for a significant rigid pes planus foot type or forefoot valgus posting, or lateral valgus wedge for an inverted pes cavovarus foot type; bilateral heel lifts in order to bring the ground up to meet the feet and to decrease the Achilles stretch on the rearfoot or help with the compensated rigid flat foot type. Footwear could be modified with use of bilateral balanced lifts, rocker soles and perhaps ankle-high uppers for additional foot and ankle support.

Based on literature review and further investigation, scientific evidence-based results are difficult to find as to what works as a best practice outcome. It might be suggested that pedorthists may need to “trial and error” with additional forefoot posting attempts in order to alleviate the pain or discomfort of the impingement as the individual patient may respond differently to specific adjustments. Foot orthoses therefore can be used to help accommodate the deformity. As an example, the pedorthist may find that a 3-degree external forefoot varus post to sulcus for a pes planus foot type alleviates the discomfort better than for the patient who seeks to be alleviated by an internal post or external tip post along the metatarsals.

If the conservative treatment fails, then surgical intervention is necessary (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Umans, 2002). Excision of the impingement bone spurs, os trigonum, or the non-union fractures or bone fragments is performed and soft tissue abnormalities either arthroscopically or open surgery is usually effective and outcomes are good. Soft tissue entrapment of the FHL tendon can be relieved by surgical release (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Umans, 2002).

Outcomes are dependent on the degree of degenerative changes within the tibiotalar joint capsule (Donovan & Rosenberg, 2010; Hopper & Robinson, 2008; Robinson (2007); Robinson & White, 2002; Tol & van Dijk, 2006; Umans, 2002; van Dijk, 2006).

Progressive deformity and disability in those patients with significant PTTD require surgical correction consisting of a calcaneal osteotomy (Donovan & Rosenberg, 2010). Fixed osseous deformities or deformities associated with arthritis are best managed with an arthrodesis of the talonavicular joint, Chopart joint complex, or subtal joint, or a triple arthrodesis (Donovan & Rosenberg, 2010)

Key Considerations

Ankle impingement syndromes, as anatomically defined about the tibiotalar joint, are very complex and difficult to clinically diagnose and treat. Radiography images can help detect the impingement site and determine the osseous or soft tissue injury that will need prescribed non-invasive or invasive management and treatment measures.

References


Ankle/Subtalar Arthrodesis

Corrie Weames, MSc, C. Ped (C)

Key Messages

- Ankle and subtalar fusion is very successful in treating the severe pain of arthritis in the ankle and subtalar joint.
- Development of arthritis at joints surrounding fused joint is common in the long term, but may not be a result of arthrodesis.
- Shoes with rocker soles, solid ankle cushioned heel (SACH), and heel lifts when necessary are very effective pedorthic treatment modalities.
- Multidensity orthoses may provide additional stability to the foot, provide additional cushioning and shock absorption, and protect against further development of arthritis in the foot.

Keywords
dorsi-/plantar flexion, equinus, fixed ankle, subtalar fusion

Introduction

Arthrodesis is the surgical fixation of a joint to promote bone fusion. It can also be called artificial ankylosis or syndesis. The fusion of a joint is a procedure commonly used to treat severely painful joints that do not respond to conservative treatments. Fixation of the ankle and subtalar joint has been around for more than a hundred years. The goals of arthrodesis in treating painful ankle and subtalar joint arthritis include correction of severe deformity, alleviation of pain, improvement in kinematic and kinetic gait parameters, and maintenance of stability (Donatto, 1998; Mangone, Fleming, Fleming, Hedrick, Siler, & Bailey, 1997; Bhatia, 2014; Brodsky, Kane, Coleman, Bariteau, & Tenenbaum, 2016; Chopra, Rouhani, Assal, Aminian, & Crevoisier, 2013, Stegeman, Louwerens, van der Woude, Jacobs, & van Ginneken, 2015; Strasser & Turner, 2012; Tenenbaum, Coleman, & Brodsky, 2014).

Arthritis of the ankle joint can be severe and debilitating, but unlike other joints of the body, primary osteoarthritis of the ankle is rare (Cheng, Huang, Hung, Chen, & Lin, 2000; Katcherian, 1998; Saltzman, Salamon, Blanchard, Huff, Hayes, Buckwalter, & Amendola, 2005; Grunfeld, Aydogan, Juliano, & Bustillo, 2013). Ankle arthritis most frequently results from fractures of the joint (post traumatic) and less commonly is the result of degenerative changes (osteoarthritis) or inflammation (rheumatoid arthritis) (Boobbyer, 1981; Katcherian, 1998; Saltzman et al., 2005).

As with the ankle joint, subtalar joint arthrodesis is commonly performed as a treatment for severe pain resulting from degenerative bone disease, arthritis or fracture. Post traumatic arthritis resulting from calcaneal fractures is the most common reason for subtalar arthrodesis surgery. Subtalar arthrodesis is also used to treat conditions such as subtalar arthritis resulting from talipes equinovarus, posterior tibial tendon dysfunction, Charcot joint, tarsal coalitions, osteoarthritis, rheumatoid arthritis, and symptomatic severe pes planovalgus (Dahm & Kitaoka, 1998; Dogan, Zorer, Mumcuoglu & Akman, 2009; Donatto, 1998; Mann & Baumgarten, 1988; Mangone et al., 1997; Sammarco & Tablante, 1998).

Pathology

The ankle joint or talocrural joint is a hinge type joint located between the inferior ends of the tibia and fibula and the superior part of the talus. This joint mainly produces movement of dorsiflexion and plantar flexion (Moore, 1985). Surgical fixation of the talocrural joint has been shown to have great success at relieving severe pain as a result of arthritis at this joint (Coester, Saltzman, Leupold, & Pontarelli, 2001; Fuchs, Sandmann, Skwara, & Chylarecki, 2003; Lynch, Bourne, & Rorabeck, 1988). It is widely accepted as the treatment of choice for severe ankle
Arthritis that does not respond to more conservative treatment modalities (Easley, Trnka, Schon, & Myerson, 2000; Donatto, 1998; Fuchs et al., 2003; Katcherian, 1998; Martin, Stewart, & Conti, 2007; Mazur, Schwartz, & Simon, 1979; Thomas, Daniels, & Parker, 2003; Thomas, Daniels, & Parker, 2006). Ankle arthrodesis tends to have three main goals when performed: correction of deformity, alleviation of pain, and maintenance of stability (Martin et al., 2007; Mazur et al., 1979; Thomas et al., 2006). Ankle arthrodesis aims to remove the degenerated surface tissue and fixates the talocrural joint to prevent ankle dorsi- and plantar flexion from occurring.

With ankle arthrodesis, the joint is ideally fixated with the hindfoot in five to ten degrees of valgus and external rotation of five to fifteen degrees or equal to the contralateral limb. In the sagittal plane, a neutral to a slight equinus position is optimal (Bayaert, Sirveaux, Paysant, Mole, & Andre, 2004; Dahm & Kitaoka, 1998; Hefti, Baumann, & Morscher, 1980; Katcherian, 1998; Thomas, Daniels, & Parker, 2003; Wheelees' Textbook of Orthopaedics, n.d.; Grunfeld, 2013; Hendrickx, Stufkens, de Bruijn, Sierevelt, van Dijk, & Kerkhoffs, 2011; Jastifer, Gustafson, & Gorman, 2013). This alignment for ankle arthrodesis is recommended as the best way to use midfoot motion to simulate the normal ankle movement thereby creating gait patterns that are not substantially altered from the unaffected side (Mazur et al., 1979; Thomas et al., 2003).

If the ankle has been fixed in varus, the transverse tarsal joint is locked and the foot becomes rigid, resulting in a vaulting type gait pattern. This position is poorly tolerated and places increased stress on the subtal joint and may cause pain under the cuboid and fifth metatarsal. Excessive valgus positioning of the tibiotalar joint contributes to medial knee pain (Boobbyer, 1981; Buck, Morrey & Chao, 1987; Katcherian, 1998). The valgus position of five to ten degrees has been demonstrated by Jastifer et al. (2013) to maximize extrinsic muscle strength.

In the past, ankles have sometimes been fixed in a more exaggerated equinus position, particularly for female patients (Hefti et al., 1980). It was believed that this would be most beneficial to accommodate the shoe styles of women, but with the ankle fixed in equinus, the midtarsal joints cannot compensate since they do not normally dorsiflex past neutral (Mazur et al., 1979; Thomas et al., 2003). With an equinus position the patient may hyperextend the knee to achieve a plantigrade foot or walk on the toes (Buck et al., 1987; Mazur et al., 1979). Equinus also increases pressure on the metatarsophalangeal joints potentially leading to forefoot pain (Katcherian, 1998). Equinus position leads to premature heel-off and a vaulting gait (Hefti et al., 1980). A dorsiflexed position has been shown to be associated with heel pad pain and stress associated with excessive dorsiflexion (Katcherian, 1998).

The long-term complication of ankle arthrodesis is believed to be an acceleration of arthritic changes in the ipsilateral foot (Buck et al., 1987; Coester et al., 2001; Mazur et al., 1979; Thomas et al., 2003, Aarts et al., 2016, Ebalard et al., 2014, Fuentes-Sanz et al., 2012). Studies have shown that most individuals with ankle arthrodesis show signs of arthritis in the subtalar and midtarsal joints in follow up studies (Martin et al., 2007; Mazur et al., 1979; Lynch et al., 1988). The functional severity of the arthritis and if it was present prior to arthrodesis is variably demonstrated in the literature (Sheridan, Robinson, Hubble & Winson, 2006, Hendrickx et al., 2011, Houdek et al., 2014). Researchers have shown that degenerative changes often exist prior to arthrodesis and show only mild progression and minimal symptomology in follow up studies years later (Aarts, Heesterbeek, Jaspers, Stegeman, & Louwerens, 2016; Ling, Smyth, Fraser, Hogan, Seaworth, Ross, & Kennedy, 2015; Yasui, Hannon, Seow, & Kennedy, 2016).

Even with shoe modifications such as a heel lift or
rocker sole, fast walking is usually the highest level of activity that can be achieved (Martin, Stewart, & Conti, 2007). Given these long-term complications, it is important to note that many researchers have found that patients who underwent ankle arthrodesis surgery and went on to develop distal joint arthritis still were happy with the decision to have the surgery and would do it again given the same symptoms they were having prior to the initial surgery (Coester et al., 2001; Fuchs et al., 2003; Katcherian, 1998; Mazur et al., 1979).

Research by Bayaert et al. (2004) showed that, following arthrodesis of the ankle, certain gait parameters are changed when walking barefoot. Fixation of the ankle joint eliminates the normal plantar flexion and dorsiflexion usually required during stance phase of gait. With the ankle joint fixed in neutral, the forward progression of the tibia at mid–stance happens through subtalar movement in the sagittal plane and dorsiflexion of the mid–tarsal joints (Beyaert et al., 2004; Cheng et al., 2000; Kitaoka et al., 1999; Mazur et al., 1979). Heel–off occurs significantly earlier compared to the unaffected side, although knee flexion is unchanged. Walking speed is reduced in people with an ankle arthrodesis due to a significant reduction in stride length and opposite step length. There is decreased knee flexion before heel strike, less time in single leg stance and decreased ground reaction force (Buck et al., 1987; Hefti et al., 1980). The ground reaction force application at heel–off is significantly different between ankle arthrodesis groups and controls. In the arthrodesis group, the distance from the ankle to the application of the GRF is lower. In controls, the GRF is applied near to the metatarsal heads, but for a fused ankle, the GRF is applied more posterior to this point. At early heel–off then, there is greater dorsiflexion stress applied to the joints between the ankle and the metatarsal heads (Beyaert et al., 2004; Wu, Rosenbaum & Su, 2004).

On the other hand, walking in shoes compared to barefoot walking found significant increases in walking speed, stride length, and opposite step length. Gait parameters are more “normal” in comparison to the contralateral limb, yet differences remain compared to controls (Beyaert et al., 2004).

Distal to the ankle joint lies the subtalar joint. The subtalar joint or talocalcaneal joint is found where the talus articulates with the calcaneus (Moore, 1985). This joint produces movements of pronation (eversion, dorsiflexion, and abduction) and supination (inversion, plantar flexion, and adduction). With subtalar arthrodesis, the talus and calcaneus are surgically fused together.

The subtalar joint is ideally fused in a slight valgus position. By placing the calcaneus in 5 degrees of valgus, locking of the transverse tarsal joint is avoided and forefoot mobility remains (Donatto, 1998; Mann & Baumgarten, 1988; Sammarco & Tablante, 1998; Wheeless’ Textbook of Orthopaedics, n.d.).

A fixed and excessive valgus position (more than 10 degrees) creates pronation in the forefoot and may lead to lateral ankle impingement pain and pain the medial longitudinal arch (Wheeless’ Textbook of Orthopaedics, n.d.; Easley, Trnka, Schon, & Myerson, 2000; Mann & Baumgarten, 1988). The development of hallux valgus, medial ankle or knee pain, and arch pain is also possible with excessive valgus positioning.

With the calcaneus positioned in varus, the transverse tarsal joint is locked and the forefoot becomes rigid, leading to significant lateral foot pain and callusing under the fifth MT head (Mann & Baumgarten, 1988). Easley et al. (2000) reported that with the calcaneus fixed in a varus position, the lateral forefoot is overloaded and may remain symptomatic despite orthotic treatment and shoe modifications.

Additional complications with subtalar fusion may occur if the screws used are too close to the anterior margin of the tibia and therefore block ankle dorsiflexion. Sural nerve neuralgia was also observed as a complication in the Easley et al. (2000) study.

Long-term complications of subtalar fusions alone have been believed to include degenerative changes at either the ankle or transverse tarsal joints (Mann & Baumgarten, 1988; Ross & Lyne, 1980). As a result of this, triple arthrodesis used to be commonplace, but it has been shown that these changes tend to be mild, and often the degenerative changes were often present prior to arthrodesis (de Heus, Marti, Besselaar, & Albers, 1997; Joveniaux, Harisbourne, Ohl, & Deboux, 2010).

Finally, non–union as a common complication (for both ankle and subtalar arthrodeses) reportedly occurs on average in 20% of cases and as high as 30% of cases (Fink, Niggemeyer, Schneider, Strauss, & Ruther, 1996; Scheppers & Patka, 2008; Paley, Lamm, Katsenis, Bhave, & Herzenberg, 2006). Non–union is associated with pain, limb length discrepancy, and deformity (Paley et al., 2006). Significantly more non–unions are reported in smokers than non–smokers (Easley et al., 2000). Non–unions require additional
Subtalar motion is important in the gait cycle to absorb shock and to allow the foot to adapt to uneven terrain. Its motion is closely linked to the transverse tarsal joint and a change in one of the joints affects the other joints. Although the subtalar joint is an important component of the foot’s adaptation to the ground, it only partially influences motion at the remaining hindfoot joints (Donatto, 1998). Following subtalar arthrodesis, talonavicular, and calcaneocuboid motion still occurs and typically makes up for the lost motion of the subtalar joint quite effectively (Aston, Delan, Otis, & Kenneally, 1997; Joveniaux et al., 2010). Dubois, Revuelta, Blatt, Maynou, Migaud, & Thevenon (2001) found no significant differences in a wide range of kinematic and kinetic variables when comparing the arthrodesis side to the contralateral side. They did find significant decreases for peak plantar flexion moment and peak power generated on the arthrodesis side.

Fusion of the subtalar joint has been shown to reduce midtarsal joint motion. Wheeless’ Textbook of Orthopaedics (n.d.) reports that subtalar joint fusion reduces talonavicular motion to about 26% of normal and limits calcaneocuboid motion to about 56%. Mann & Baumgarten (1988) showed that isolated subtalar fusion resulted in an absence of inversion and eversion of the subtalar joints, decreased ankle dorsiflexion/plantar flexion, and approximately 50% reduction in forefoot abduction/adduction (compared to the contralateral limb).

**Differential Diagnosis**

Symptoms following arthrodesis of either the ankle or subtalar joint are usually greatly reduced compared to prior to fixation. Typically, additional symptoms may surface many years following surgery. These symptoms may include medial knee pain, forefoot pain (metatarsalgia), lateral ankle impingement, and arthritis symptoms at surrounding joints. Many of these symptoms will be related to the fixation position of the earlier arthrodesis (as described above in Pathology).

**Common Testing**

It is important to determine the exact level of arthrodesis if the patient is unclear on this point. Communication with the referring physician should provide this information.

Detailed examination of the foot will provide evidence of the level of arthrodesis. Absence of ankle dorsiflexion/plantar flexion coincides with ankle arthrodesis. The clinician must be careful, in assessing ankle motion, to not include motion from the midtarsal joints. This would give a false impression of ankle motion. Although the talocrural joint should have no motion, there is typically significant dorsiflexion and plantar flexion possible through the midtarsal joints to allow near-normal walking style.

The subtalar joint is most clearly assessed through examining the rearfoot range of motion into inversion and eversion. Subtalar fusion will eliminate the inversion and eversion movement of the hindfoot, but appropriate positioning in fusion allows the midtarsal joints to continue to function well. Care must be taken to isolate the movement of the rearfoot in inversion and eversion as the way to assess subtalar joint motion. Movement of the heel fat pad or of the midtarsal joints can give the illusion of subtalar motion if not careful.

**Contraindications**

Leg length discrepancy is possible in a fused ankle or subtalar joint but is not always present. An equinus foot is also a common result of old-style arthrodeses. It is important to evaluate whether a true LLD is present prior to adding a lift. Overly rigid orthoses are generally not recommended. It is important to balance the creation of motion (often with rocker soles or SACH heels) with control of motion (to reduce stress to joints adjacent to the arthrodesis).

**The Patient Perspective**

The presence of an ankle or subtalar arthrodesis is best confirmed through detailed history and assessment of the patient. The patient will usually provide information that the ankle or foot had been fused following an accident or after an injury that caused recurrent severe pain that was unmanageable through other means. If the arthrodesis is relatively new, the patient may report that their pain is greatly reduced compared to their pain prior to fusion. If the arthrodesis is older, the patient may report symptoms of arthritis at other joints of the foot surrounding the site of the fusion.

Most patients with arthrodesis of the ankle will have no pain with walking initially (after completely healed from surgery). Most will find that they prefer walking with shoes rather than barefoot. They will typically naturally avoid walking at high speed and will generally have a slower natural walking speed compared to controls (Beyaert et al., 2004). Patients
who have had an ankle arthrodesis often walk with only a slight limp even without modified footwear (Lynch et al., 1988).

Common Treatment

Most patients with ankle or subtalar arthrodesis will have no pain with walking in the short-term following recovery from surgery and most will prefer to walk with shoes rather than go barefoot (Fuentes-Sanz, Moya-Angeler, López-Oliva, & Forriol, 2012). For the long-term, additional arthritis symptoms may present themselves at the ankle, subtalar, or midtarsal joints in particular (Coester et al., 2001; Thomas et al., 2003). There is limited evidence to support or refute the use of custom-made orthoses for the treatment of patients following ankle or subtalar arthrodesis. Most of the evidence available does focus on the use of footwear following arthrodesis. Several studies have examined the effects of rocker soles, solid ankle cushioned heel (SACH heel), and heel lifts in relation to gait kinematics and kinetics (Long, Klein, Sirota, Wertsch, Janisse, & Harris, 2007; Myers, Long, Klein, Wertsch, & Harris, 2006; Sirveaux, Beyaert, Paysant, Mole, & Andre, 2006; Wu et al., 2004, Arazpour, Hutchins, Ghomshe, Shakly, Karami, & Aksenov, 2013). Some of these studies relate primarily to pressure relief in patients with diabetes. Baker, in 1970, reported that a SACH heel improves gait following ankle fusion but only reported anecdotal evidence. Beyaert et al. (2004) reported that wearing of shoes delayed heel–off in the arthrodesis group. Beyaert et al. (2004) also showed that the GRF progression was improved by wearing shoes and was comparable to controls. However, the GRF was still positioned posterior to the metatarsal heads in the arthrodesis group, compared to controls with shoes. This is important as Beyaert et al. (2004) suggests that the location of GRF may contribute to increased stress on the joints between the arthrodesis and the MT heads.

Four common footwear modifications are recommended in the literature for treatment of ankle or subtalar arthrodesis. The most common modification following ankle fusion is a rocker sole. Specific design elements such as the location of the fulcrum of the forefoot rocker have not been well examined in the literature. There is some evidence to suggest that a rocker sole placed incorrectly (distal to the MTP joints of the foot) increases the bending stress to the midtarsal joint and accelerates deterioration of these joints (Beyaert et al., 2004). A double rocker (heel rocker and forefoot) has been shown to reduce ankle plantar flexion moment during walking and decreases midfoot loading (Long et al., 2007, Arazpour et al., 2013). Wu et al. (2004) postulates that moving the rocker fulcrum more proximal to the metatarsal heads may be necessary when ankle movement is lost but evidence is not provided. Runners alone have been shown to be beneficial by Arno & Roman (2015) and Talaty, Patel, & Esquenazi (2016). Finally, Jones, Moed, & Karges (2016) found that, in comparing barefoot walking and walking in rocker bottom shoes, the rocker bottom shoe conditions allowed for a more physiologically normal gait in patients with ankle arthrodesis.

A second common footwear modification for a fused ankle is a SACH heel (Baker, 1970; Wu et al., 2004). This modification helps to simulate plantar flexion at heel contact, cushions heel impact and reduces the flexion moment generated at the knee at heel contact.

Thirdly, if there is a leg length difference following ankle or subtalar arthrodesis, a heel and sole lift to the affected side on the shoe should be added. Although in the past it was more common to fix the ankle in equinus, making the addition of a heel lift reasonable, it is currently less common to see this position. Now it is recommended that the foot be fixed in a neutral sagittal plane position. Given current information, it is important to assess the fixed position of the ankle, whether it is planter flexed or neutral, to determine if a heel lift would be beneficial or not. Hefti et al. (1980) suggested that with a neutral fusion position, a shoe with a heel height of 1.5–2.0cm was still optimal. Beyaert (2004) has also suggested that increasing heel height may be beneficial in reducing stress to the midtarsal joints but cautions that the limitation of foot plantar flexion would have to be taken into account. If there is no LLD, then care should be taken to modify the rocker of the shoes without adding any lift in the process.

Finally, a fourth common modification for fused ankles is outflaring of the sole of the shoe to provide medial or lateral stability as needed. If residual varus alignment is present, then a lateral outflare can minimize problems related to this positioning (Baker, 1970; Long et al., 2007; Marzano, 2002; Mazur et al., 1979; Myers et al., 2006; Sirveaux et al., 2006; Wu et al., 2004).

Custom orthoses for a patient with ankle or subtalar arthrodesis should be aimed towards protecting the surrounding joints from deterioration. If the shoe can be made to accommodate the loss of ankle dorsiflexion motion, then the orthosis can be focused on the control of midtarsal joint motion. Generally a softer, multildensity foot orthosis...
is recommended for several reasons. The softer materials complement the lost shock absorption properties of the ankle and subtalar joints. The materials can also be used to accommodate small loss of height (leg length discrepancy) on the affected side. The orthosis should also be geared toward correction and control of any forefoot-to-hindfoot alignment concerns to reduce stress to the midtarsal joints (Marzano, 2002).

References


Talar Dome Lesion

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Key Messages
- A talar dome lesion is an injury to the cartilage and underlying bone of the talus within the ankle joint.
- It is also called an osteochondral defect (OCD) or osteochondral lesion of the talus (OLT). “Osteo” means bone and “chondral” refers to cartilage.
- Talar dome lesions are usually caused by an injury, such as an ankle sprain.
- If the cartilage doesn’t heal properly following the injury, it softens and begins to break off.
- Sometimes a broken piece of the damaged cartilage and bone will “float” in the ankle (Konig, 1887).

Keywords
antalgic gait, anterolateral, clicking, instability, locking, osteochondral, posteromedial

Introduction and Pathology

The talus is the second largest of the tarsal bones that connects the leg to the foot. The portion of the talus that articulates with the tibia is known as the trochlea or talar dome. Because of the shape of the articular surface of the trochlea, the ankle joint is more stable and has more articular surface contact while in dorsiflexion. For the purpose of osteochondral lesions, it is the talar dome and the talo-crural joint or ankle joint that are important to review.

The ankle is a joint which is formed by the tibia and fibula (bones above the ankle in the foreleg) and the talus (below the ankle joint). The ankle joint allows for the upward (dorsiflexion) and downward (plantarflexion) motion. The end of the shin bone (tibia) forms the inner bony prominence of the ankle called the medial malleolus. The outer bony prominence is called the lateral malleolus and is formed by the small outer bone in the foreleg called the fibula (Chew, Tay, & Won, 2008).

In gait, the ankle joint functions primarily as a hinge. As we travel from heel strike to toe-off, the tibia articulates with the dome of the talus.

Osteochondral fractures of the talus have been classified into four stages by Berndt and Harty (1959):

**Stage I:** a small area of subchondral compression

**Stage II:** a partially detached fragment

**Stage III:** a completely detached fragment remaining in the crater

**Stage IV:** a fragment that is detached and also loose in the joint

Below are images of these classifications:

Osteochondral fractures of the ankle are seen with eversion or inversion injuries. There is a high association with ligamentous injury and there might also be a bone contusion or a true linear fracture. The osteochondral fragment might remain stable or be slightly displaced. If the fragment remains attached to the underlying bone, revascularization and healing occur. If completely detached, complete healing does not occur. The intra-articular loose body can increase in size with nourishment from the synovial fluid within the ankle joint. In some cases, the fragment might attach to the synovial lining at a distant site and become absorbed. Treatment of this lesion depends on the status of the fragment.
Talar dome lesions commonly occur in two areas of the talus:

1. the medial talar dome
2. the anterolateral talar dome

The average age of occurrence of talar osteochondral lesions usually lies between 20 and 30 years of age. They are bilateral in 10% of cases, with a slight preponderance in males. These lesions are seen in 6.5% of ankle sprains. However, this incidence could be underestimated as many of these lesions may be subclinical, masked by other more obvious associated injuries of the foot and ankle, or missed because of limitations in conventional radiological investigations. Osteochondral lesions of the talus can present as a late complication of ankle injuries. As the talus is largely covered by articular cartilage, it has a limited ability for repair. Early and accurate diagnosis is important as talar integrity is required for optimal function of the ankle. The common presentation is chronic ankle pain with a history of ankle trauma. Conservative treatment involving a period of casting and non-weight-bearing is recommended for acute, non-displaced osteochondral lesions. Surgical management is recommended for unstable lesions or failed conservative management (Barnes & Ferkel, 2003).

Flick and Gould (1985) reviewed the literature in reports of more than 500 patients with these lesions and found that 98% of lateral dome lesions and 70% of medial dome lesions were associated with a history of trauma. This could either be a single injury or repeated microtraumas.

Differential Diagnosis

• Ankle syndesmosis injury
• Compartment syndrome
• Deltoid ligament injury
• Dislocation of the talus
• Second- or third-degree ankle sprains
• Tarsal bone fracture

Common Testing

Examination

A typical presentation is of chronic ankle pain persisting after a prior history of an inversion injury of the ankle. Pain is usually experienced at the specific locations of the lesions. Recurrent swelling, weakness, stiffness, and catching of the ankle joint are often described. Patients with a history of recurrent ankle sprains often complain of ankle instability. Point tenderness can usually be elicited and should be sought over the common sites of the lesions.

Anterolateral lesions can be palpated at the anterolateral talar dome with the ankle in plantarflexion. Posteromedial lesions can often be palpated behind the medial malleolus with the ankle in dorsiflexion. Tests for instability should be performed, including the anterior drawer test as well as the inversion and eversion stress tests. Range of motion of the ankle should be documented and compared with the contralateral side. After the initial pain and discomfort of a strain or sprain subsides, individuals usually resume or even increase their activity level. If an osteochondral lesion has occurred, however, everyday activities that put pressure on the joint, may lead to pain and swelling, although the joint usually is fine when at rest. A patient with an osteochondral lesion will often feel a dull ache in the joint and might also experience a mild locking or clicking of their ankle joint. The affected joint could also seem to be loose.

It can be challenging to diagnose an osteochondral lesion at the time of injury. Many scans miss the damage caused by the lesion, which is also masked by the sprain or trauma that caused the injury.

A physician will examine the joint for instability and range of motion. An X-ray might be ordered, but a cartilage tear is difficult to see on an X-ray, so an MRI or CT scan might be required. Check other joints and limb alignment. Look at the gait—most patients have an antalgic gait. Examine the skin and soft tissues carefully for the effects of previous surgery, injury, or infection. Check the alignment of the tibia to the hindfoot, the midfoot, and the forefoot. What is the overall foot shape? If there is malalignment, is it correctable or fixed? It might be correctable at some levels but not at others.

Palpate for tender areas, synovitis, effusion, and loose bodies. If there is anterior tenderness, dorsiflex the ankle while pressing on the tender area (the Molloy impingement test), looking for an impingement lesion. Assess the range of ankle motion; check that any dorsiflexion/plantarflexion is in the ankle rather than the midtarsal joint.

Perform the anterior draw and talar tilt tests for instability.

Check what movement remains in the subtalar and midtarsal joints.

Assess circulation, sensation, and power.
The Patient Perspective

Your patients might complain of symptoms such as:

- Chronic pain deep in the ankle typically worse when bearing weight on the foot (especially during sports) and less when resting
- An occasional “clicking” or “catching” feeling in the ankle when walking
- A sensation of the ankle “locking” or “giving out”
- Episodes of swelling of the ankle—occurring when bearing weight and subsiding with rest

Common Treatment

In the acute phase, the standard of care would be immobilization for 4–6 weeks. This could be done using a walking cast or a removable immobilization brace. Where a cartilage defect is not detectable on X-ray, an MRI might be ordered by a physician if the symptoms persist. When the acute phase is over and the patient is ready for mobilization further examination needs to be done.

Physiotherapy should be ordered after immobilization to regain range of motion and strength in the ankle joint and surrounding structures. The gait pattern of a patient with a talar lesion will be altered due to the lack of dorsiflexion at the ankle. To help with toe-off, a patient might walk with their foot externally rotated in an attempt to avoid dorsiflexion of the ankle. This would produce an antalgic gait. If the patient doesn’t externally rotate the foot then this may cause a hyperextension in the knee and an early toe-off due to the lack of dorsiflexion. Gait retraining might be required by the physiotherapist.

A pedorthist should also be aware of any pronation that the foot might undergo. The talus sits at an angle of approximately 21 degrees internally rotated when in subtalar neutral (Root, Orien, & Weed, 1977). When the foot pronates the talus will internally rotate causing a shear force to be placed on the talar dome. If the talar dome is compromised with a talar lesion or osteochondral fracture then this will create an unhealthy situation on the talar dome as the talus rotates under the tibia.

A foot orthosis with a heel raise will allow the foot to be more plantar grade where it lacks end-stage dorsiflexion in gait. Controlling pronation using a UCBL-style foot orthosis would limit internal talar rotation and provide maximum control of the foot during the stance phase of gait. A rocker sole shoe modification would limit dorsiflexion at the ankle and yet allow for a fluid transfer of body weight throughout toe-off. A Richie brace with a limited range of motion hinge would provide full control medially and laterally. The hinge can be locked in a position of comfort or within a prescribed range of motion.

A subtalar control ankle–foot orthosis (SCAFO) is a leather gauntlet style of ankle–foot orthosis that provides minimal motion in all planes through the ankle and subtalar joints. This would give the patient very limited motion but would maximize the potential for pain relief.

Contraindications

With all devices at our disposal for treatment, we must provide the patient with the ability to get through a range of motion with as little pain and discomfort as possible. This might take a lot of trial and error—even more than other conditions—but it is our job as lower limb professionals to go through this trial and error with the patient’s well-being in mind.

A foot orthosis must provide the proper motion control, or shock attenuation to help the patient. For example, positioning a patient’s rearfoot in varus, thereby adding pressure to the talar lesion, would obviously be detrimental to treatment.

Key Considerations

Talar dome lesions are injuries one should not miss. Clients that are suspected of having this injury and have not yet seen their physician for diagnosis should be referred to them. Ankle sprains that seem to give ongoing pain in atypical patterns, pain with certain ankle joint positions, and reports of catching with sharp pain should not be passed off as conditions that simply need more time to heal.

References


Shin Splints

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Key Messages

• Shin splints refers to exercise-induced pain in the lower leg.
• It is usually an overuse injury caused by abnormal biomechanics.
• Commonly found in running and jumping sports.
• Custom orthoses are an effective treatment of shin splints.
• The term shin splints is commonly used to describe activity-induced pain in the lower leg. For the purpose of this document, shin splints will be broken down into three parts: (1) medial tibial stress syndrome; (2) compartment syndrome; and (3) peroneal muscle strain.

Keywords

Achilles, bursa, calcaneal tuberosity, exostosis, Haglund’s, inflammation, Kager’s, spongy

Medial Tibial Stress Syndrome (MTSS)

Introduction

Medial tibial stress syndrome (MTSS) is one of the most common sport-related injuries of the lower leg. Clement, Taunton, Smart, & McNicol (1981) reported that MTSS accounted for 13.2% of lower extremity injuries in male and female high school runners, making it the second most common injury in this study. In 1913, Hutchins (as cited in Thacker, Gilchrist, Stroup, & Kimsey, 2002) described shin pain as “spike soreness” in runners. There is no official definition of MTSS; however it is commonly referred to as pain along the distal two-thirds of the posterior medial border of the tibia that occurs during exercise (Kortebein, Kaufman, Basford, & Stuart, 2000).

Pathology

The exact pathogenesis of MTSS is still unclear (Tweed, Avil, Campbell, & Barnes, 2008). Until recently, inflammation of the periosteum (periostitis) was considered the most common cause of MTSS; however, more recent studies point to a stress reaction of bone that becomes painful (Tweed et al., 2008). Even though there is not a definitive pathophysiologic cause of MTSS, it is clear that MTSS is an overuse injury with excessive strain on the posterior medial border of the tibia.

Factors that contribute to MTSS can be broken down into two groups, extrinsic and intrinsic.

Extrinsic factors are environmental or outside the body. Running on hard or uneven surfaces and running up- or downhill can cause MTSS. Improper running and training techniques can also cause pain. These include increasing duration and intensity of activity too quickly, too much hill training, and not enough rest between training sessions (Thacker et al., 2002). The use of worn or inadequate shoes can contribute to MTSS (Thacker et al., 2002). If the shoe is too hard or too old, there will not be adequate shock absorption causing increased strain on the lower leg. Footwear that has insufficient stability or support is also a factor contributing to MTSS.

Intrinsic factors are within the person and include abnormal biomechanics (such as subtalar joint overpronation), decreased muscle strength, decreased muscle flexibility (especially in the gastrocnemius and soleus), and low bone mineral density. Other factors include increased BMI, female gender, navicular drop, ankle plantar flexion, hip external rotation ROM, and previous running injury (Winkelman, Anderson, Games, & Eberman, 2016; Reinking, Austin, Richter, & Krieger, 2017).

Common Testing

It is important to get a complete history from the patient so there is a better understanding of the problem. Questions include where the pain is, what type of pain it is, when it occurs, how long it lasts, and what type of footwear is being used. Patients will usually run their finger along the medial aspect of the tibia when asked where the pain is. In the early stages
of the injury, the patient will usually describe the pain as an ache during the beginning of the activity, slowly diffusing as they continue in the activity. If the condition is left untreated, the pain can be present for the duration of the activity and also be present after the activity. It is important for the patient not to continue the activity if the pain is significant. If the pain is mild, then it may be possible to continue with the activity at a reduced intensity level and duration, as long as the symptoms do not get worse.

The best way to test for MTSS is to palpate the posterior medial aspect of the tibia. The pain is usually a 4–6cm area along the middle to distal third of the tibia (Rompe, Cacchio, Furia, & Maffulli, 2010). Other tests include passive stretching of the soleus, single stance heel raises, and hopping up and down on one foot (Rompe et al., 2010). These tests can reproduce symptoms of MTSS. A possible misdiagnosis is medial tibial stress fracture; however, a stress fracture is more localized as compared to MTSS, and the pain is usually described as very sharp and continuous throughout the activity.

**Common Treatment**

Orthoses are one treatment option for MTSS. One of the factors that causes MTSS is subtalar joint overpronation. Studies have found greater than 10mm of navicular drop may double the chance of developing MTSS (Winkelman et al., 2016). Overpronation causes strain on the tibialis posterior muscle and other muscles of the lower leg, which can lead to excessive loading and strain of the medial posterior border of the tibia. The orthoses can reduce the amount of over-pronation thus reducing the strain on the soft tissue of the lower leg. These will need to be functional orthoses made from a three-dimensional mould of the foot. The materials that are used can vary based on the specifics of the patient and the experience of the practitioner. Regardless of the type, the orthoses should be made from raw materials to the specifications of the practitioner. The orthoses should have full medial longitudinal arch contact and can be posted medially in the hindfoot and/or forefoot. If there is a rigid forefoot varus that is contributing to the overpronation, then adding a sulcus-length medially posted forefoot extension may be required. Another option, aside from custom orthosis, is an off-the-shelf orthosis. Deciding on which one to use will depend on the severity of the overpronation and the patient’s ability to acquire custom orthosis.

Footwear will also be a very important component in the treatment of MTSS. It is important to evaluate the footwear in use during the time of pain. Is it the appropriate shoe for the activity? Is it worn out and in need of replacing? A common mistake that runners make, especially new runners, is that they do not run in the appropriate shoe. Runners need to be in an appropriate running shoe; basketball players should wear a basketball shoe. Regardless of the activity, the shoe needs to fit properly. Make sure that there is a thumb width between the longest toe and the end of the shoe. The width also should be fitted properly, making sure the toe-box of the shoe is not narrower than the foot. The shoe also needs to provide enough cushioning for the specific person based on their weight, type of activity, and frequency and duration of the activity. The rule of thumb for runners is to replace shoes after 500 km of running. Another aspect of the shoe that is important is the stability or torsional stability of the shoe. Because overpronation is one of the causes for MTSS, the shoe needs to have enough support to help reduce pronation forces. This can be tested by twisting the shoe in the middle (it should be difficult to twist). For severe overpronators, a motion control shoe may be necessary.

Another treatment that can be helpful is icing the affected area. Icing two to three times a day for approximately 10 minutes is recommended. With MTSS, there is inflammation of the soft tissue and icing can help with reducing this inflammation.
Stretching is also important, as tight muscles, especially the gastrocnemius and soleus, are factors in causing MTSS. Stretch the gastroc/soleus, holding for 30 seconds, two to three times per leg. Repeat this stretch two to three times per day.

Other health care professionals can also play an important role in treating MTSS. Physiotherapists can assess for muscle weakness, imbalance, and tightness, and provide the appropriate treatment plan. Massage therapy is another option to decrease muscle tightness and break down adhesions. If the pain persists, referring back to the physician for X-rays or bone scans may be necessary to rule out tibial stress fractures.

Compartment Syndrome

Introduction

Compartment syndrome occurs “when the pressure within a fixed fascial compartment is raised sufficiently to result in tissue ischemia leading to neuromuscular compromise” (Power & Greengross, 1991, p.218). Compartment syndrome is broken down into acute and chronic. Acute compartment syndrome occurs with trauma to the lower leg. Chronic compartment syndrome presents as pain during activity which is relieved with rest. Of the four compartments of the lower leg, the anterior compartment is the most likely to be affected (McLaughlin, Heard, & Kelham, 2014).

Pathology

Acute compartment syndrome is most often caused by a severe trauma to the lower leg, such as tibial fracture or severe muscle contusion (Power & Greengross, 1991). In chronic compartment syndrome, exercise or even repetitive muscle contraction causes the tissue pressure within a compartment to increase to an abnormally high level, but because the fascia can’t stretch, the tissues in that compartment aren’t able to expand sufficiently under the increased pressure. Poor biomechanics, such as overpronation, enlarged muscle, a thicker than usual fascia, and high venous pressure can cause chronic compartment syndrome. If left untreated, the pain can prevent the patient from participating in their activity.

Clinical Observations

It is important to get complete history from the patient. Patients will complain of an aching or burning pain during activity. They may also describe numbness or tingling and muscle weakness. The symptoms usually start soon after beginning activity, worsen as the patient continues with activity, and subside 15–30 minutes after ceasing the activity. Patients may have edema and neurological dysfunction when examined directly after activity (Flick & Flick, 2015). If there is numbness and tingling that does not subside, prompt medical attention is recommended. Another thing to look at is the skin, as it can be shiny, swollen, and red. It is important to cease participation in the activity if these symptoms persist, as continuing could cause permanent damage to the muscle.

Common Treatment

As poor biomechanics can cause chronic compartment syndrome, orthosis can be a very effective treatment. Overpronation can be reduced with an effective functional orthosis. The orthosis should be fabricated from a three-dimensional mould of the foot and be constructed from raw materials. The type of materials used will depend on the specifics of the patient and the experience of the practitioner. The orthosis should have full medial longitudinal arch contact and can be posted medially in the hindfoot and/or forefoot. If there is a rigid forefoot varus that is contributing to the overpronation, then adding a sulcus-length medially posted forefoot extension may be required. Another option in lieu of a custom orthosis is an off-the-shelf orthosis. Deciding which to use will depend on the severity of the overpronation and the patient’s ability to acquire a custom orthosis.

Footwear will also play an important role. The patient needs to wear specific activity related footwear like running shoes for runners and basketball shoes for basketball players. The shoe also needs to provide enough cushioning for the specific person based on their weight, type of activity, and frequency and duration of the activity. Another aspect of a shoe that is important is the stability or torsional stability of the shoe. Because overpronation is one of the causes of chronic compartment syndrome, the shoe needs to have enough support to help reduce pronation forces. This can be tested by twisting the shoe in the middle. It should be difficult to twist it. For severe overpronators, a motion control shoe may be necessary.

Other health care professionals that may be helpful include physiotherapists.

If these conservative treatment options do not work, then surgery may be needed. If pain persists for six months, intracompartmental pressure should be measured by a physician. If the anterior compartment pressure is greater than 50mmHg, patients usually

Shin Splints
respond well to a subcutaneous fasciotomy (Allen & Barnes, 1986).

**Peroneal Muscle Strain**

**Introduction**

Peroneal muscle strain causes muscle pain in the lateral aspect of the lower leg. It is usually found in people who participate in running and jumping sports. Hindfoot inversion and supination can cause excessive strain on the peroneal muscles, as can running on uneven terrain. Shoes that are worn out, especially on the lateral side, can also cause peroneal strain. The peroneal muscles are evertors of the foot so excessive supination will stretch the peroneal tendons and fatigue the muscles because they are trying to stabilize the foot during gait.

**Clinical Observations**

Patients will complain of pain along the lateral aspect of the leg. They will describe it as tightness or aching. The pain usually occurs during the duration of the activity and can still persist after the activity. It is important to assess the footwear, taking notice of the lateral aspect of the shoe.

**Common Treatment**

Treatment includes orthosis, proper footwear, icing, stretching, and rest. It is important to be careful with stretching, as too much too early can reaggravate the injury. Custom orthoses should be made from a three-dimensional mould of the foot and fabricated from raw materials. Supination is one of the causes of peroneal strain and people who supinate usually have a high arch and a rigid foot. Another common characteristic of a cavus foot structure is a plantar flexed first ray and a valgus forefoot. The orthosis is usually made from softer materials to accommodate for the lack of shock absorption. If there is a plantar flexed first ray or forefoot valgus, a 2–5 sulcus extension may be needed. The orthosis can also be laterally posted. The lateral post will be more effective if it is extended through the entire lateral aspect of the orthosis.

Footwear is also important in treating peroneal strain. Because a supinated pes cavus foot structure is more common, a neutral cushioned shoe is usually required. It is still important for it to be torsionally stable and not too soft laterally. Regardless of the activity, the shoe needs to fit properly. Make sure that there is a thumb width between the longest toe and the end of the shoe. The width of the shoe also has to fit properly, making sure the toe-box of the shoe is not narrower than the foot. It is also important to make sure that the shoe is not worn out. The rule of thumb for runners is to replace shoes after 500 km of running.

Icing can be helpful in reducing pain. It is best if the patient can ice two to three times a day for 10 minutes each time. It is also important to stretch the peroneal muscles as they become tight from the excessive strain. A good way to stretch the peroneal muscles is to stand beside something against which you can brace yourself and invert the foot so you are standing on the lateral side of the foot. It is important to brace oneself so as not to roll too far laterally. This stretch can also be easier if wearing shoes.

Other health care professionals can also play an important role in treating peroneal muscle strain. Physiotherapists can assess for muscle weakness, imbalance, and tightness and provide the appropriate treatment plan. Massage therapy is another option to decrease muscle tightness and break down adhesions.

**References**


Patellofemoral Pain Syndrome

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Key Messages

- Patellofemoral pain syndrome is a malalignment of the femur and the patella.
- Knee pain in children may be referred from the hip or other sources.
- Internal tibial rotation contributes to patella malalignment.
- Orthoses controlling pronation have been found to be helpful.
- Multi-disciplinary treatment yields best results.

Keywords

anterior knee, chondromalacia, crepitus, femoral anteversion, genu recurvatum, Q angle, subluxation, synovial plicae

Introduction

Patellofemoral pain syndrome (PFPS) is defined as retropatellar or peripatellar pain in the absence of other pathologies, including patellar tendinopathy, ligament insufficiency, and internal derangement (Barton, 2011; Lee, Ren, Wilson, Kang & Zhang, 2011). Pain is most commonly produced by activities that increase patellofemoral joint (PFJ) loading, such as running, squatting, and ascending stairs (Lee et al., 2011; Powers, 2003; Servi, 2009), thus most prevalent in active populations particularly runners (Potter, 2009; Powers, 2003; Vega, 2009). PFPS is highly significant to sports medicine as it is reported to represent around 14–25% of all knee injuries and 5–17% of all injuries seen in sports medicine clinics (Barton, 2011; Lee et al., 2011; Sheehan, 2010; Vega, 2009). An almost 50% greater incidence in active females has been reported and is generally most prevalent under the age of 34 in all groups (Patel, 2011; Vega, 2009). The significance of this condition and the need to develop effective treatment protocols is emphasized by research indicating that 71–91% of individuals with PFPS continue to report recurring pain up to 20 years after initial diagnosis (Barton, 2011; Lee et al., 2011).

PFPS is distinct from the term chondromalacia patella (CMP), the abnormal softening of the patellar fibrocartilage that was first described in 1928 by Aleman. PFPS is also accepted as a precursor to, although on a poorly defined continuum with, CMP and patellofemoral arthritis, the osseous wear and tear of the patella or femoral condyles (Lee et al., 2011; Patel, 2018).

In 1968, Hughston focused on extensor pathomechanics as a cause of anterior knee pain. The renaissance of patellofemoral mechanics and structure as the primary cause of anterior knee pain was promoted by the work of Merchant et al. and Ficat and Hungerford (Patel, 2018).

Advanced radiographic techniques permitted analysis of patellofemoral pathomechanics, which, in turn, led to the understanding that “chondromalacia was generally the result of advanced patellar pathomechanics and not a primary disease process of the patella.” In fact, abnormal softening of the cartilage does not correlate with pain (Chester Knee Clinic & Cartilage Repair Center, 2013; Patel, 2018).

PFPS is not associated with specific races, and while there are no statistics on mortality and morbidity rates for this condition, it is suggested they would be the same for any group that is unable or unwilling to exercise (Potter, 2009). Obesity may be a factor as it is a risk factor for osteoarthritis, which is often a progression from PFPS (Chester Knee Clinic & Cartilage Repair Center, 2013; Patel, 2018).

The overwhelming consensus of past literature agrees that PFPS is a structural/mechanical condition which is based on kinematics (Sheehan, 2010). More recently, however the role that kinetic forces play in the knee, hip, and foot are opening up new understandings of this complex condition. Although there is some conjecture about the role and function of the specific mechanics, how they interrelate and how to measure them, the condition is recognized as
multi-factorial in origin (Barton, 2011; McClay, 2000; Potter, 2017; Sheehan, 2010).

Pathology
The patellofemoral joint is the articulation of the patella with the femoral condyles which form a “groove” known as the femoral trochlea (Servi, 2015). The patella is the largest sesamoid bone in the body, embedded in the quadriceps and patella tendons. It has a configuration of a triangle with its apex directed inferiorly. Superiorly, it articulates with the trochlea (Servi, 2015). A subcutaneous bursa separates the patella from the overlying skin. The articular surface of the patella consists of four facets: inferior, middle, superior, and medial vertical (Chester Knee Clinic & Cartilage Repair Center, 2013; Manske, 2008).

Functional anatomy
The patella acts as a fulcrum for extension of the quadriceps. The patella travels in a J-shaped pattern, moving laterally with knee extension (Manske, 2008; Patel, 2018). The lateral aspect of the trochlea, the vastus medialis, and the medial patellofemoral ligament (retinaculum) prevent excessive lateral translation. Lateral tracking is guided by both the vastus lateralis and the iliotibial band. Patellar motion is further constrained by the patellofemoral ligament, the patellotibial ligament, and the retinaculum.

While anatomy of the distal femur, the vastus lateralis and lateral patellofemoral ligaments (retinaculum) provides restraint against medial subluxation (Patel, 2018; Servi, 2015) largely thought to be controlled by the vastus medialis oblique (VMO) muscle (Chester Knee Clinic & Cartilage Repair Center, 2013; McClay, 2000; Powers, 2003; Servi, 2015). More recent analysis shows that this is a much smaller effect than previously understood. In fact it has been clearly shown that the femur internally rotates under the patella and the patella itself has a very small actual lateral excursion during extension (Williams, McClay Davis, & Baitch, 2003). The patella itself is engaged with the trochlea at 20°–30° of knee flexion while at 90 degrees the patella contacts the lateral and medial femoral facets within the condylar fossa. At 130°–135 degrees of knee flexion, the medial facets of the patella contact the articulating surface of the femoral condyles. In knee extension, the patella abuts the suprapatellar fat pad (Manske, 2008; Servi, 2015).

Patellar loading/contact pressure studies showed excessive loading occurred at greater than 50 degrees knee flexion, while lower contact pressures on a loaded knee that are less harmful to the PFJ occurred between 90–50 degrees and 20–0 degrees (Manske, 2008, Potter, 2009; Servi, 2015).

According to a study by Lin, Wilson, Mohsen, Press, Koh, Nuber, & Li-Qun Zhang, (2010), as knee flexion angles increase during squatting, the patella shows gradual medial shift in healthy subjects and continuous lateral shift in subjects with patellofemoral pain. Concomitantly, subjects with patellofemoral pain demonstrated greater lateral excursion during deep squatting than those without. These results suggest that the patella is not adequately stabilized during active knee flexion/extension in patients with PFPS (Lee et al., 2011). Previous literature focused on weakness of the VMO muscle as the cause of a lateral shift and patellar rotation during knee flexion. It has also been suggested that an altered VMO muscle reflex response time is a factor (Vega, 2009).

Since the patella lies within the quadriceps tendon it has the function of increasing the mechanical advantage of the quadriceps extensor mechanism. Not only does the patella increase the force of knee extension by 50%, by increasing the lever arm of the quadriceps muscles, it also provides stability to the patellar tendon, acting as a guide for the quadriceps tendon and spreading out the compressive forces placed on the femoral condyles (Patel, 2018; Servi, 2015).

Pathomechanics
The pain experienced with PFPS is generally believed to originate from two sources. The first source includes injury to the various soft tissues associated with the patellofemoral (PF) joint, whereas the second arises from compressive forces between the articulation of patellar facets and the femoral condyles, which vary between knee positions (Patel, 2018; Sheehan, 2010). Abnormal patellar tracking, particularly lateral tracking, can increase patellofemoral contact pressure and subsequently trigger pain by activating nociceptive (pain) fibres in the subchondral bone located underneath the patellofemoral articular surface (Lee et al., 2011; Patel, 2018; Sheehan, 2010). Articular cartilage lesions themselves are not symptomatic, although the alteration of the normal protective load-bearing characteristics of thinned and damaged articular surfaces remains a potential source of pain over time (Chester Knee Clinic & Cartilage Repair Center, 2013; Patel, 2018). Any biomechanical factors or strength imbalances that increase the lateral compressive forces or soft tissue structures on the PF joint are capable of producing symptoms.
Until approximately 12 years ago PFPS had been studied primarily using 2-D information that described joint kinematics, i.e. descriptions of motion and position. These include dynamic internal tibial rotations via coupling with foot overpronation and hip internal femoral rotation as well as dynamic increased Q angles (Barton, 2011; Lee et al., 2011; Sheehan, 2010).

The Q angle is perhaps the most well-known single factor. The Q angle is a line created from the anterior superior iliac spine (ASIS) to the mid-patella, which intersects with a line from the mid-patella to the tibial tubercle when the knee is in full extension. An average Q angle for a male is 14 degrees, whereas that for a female is 17 degrees. Q angles larger than average can indicate abnormal patellar tracking. Excessive femoral anteversion, genu valgum, external tibial torsion, and hyperpronation of the foot can effectively increase the dynamic Q angle component (Chester Knee Clinic & Cartilage Repair Center, 2013; Lee et al., 2011; Manske, 2008; Scherer, 2009; Thabit & Micheli, 1992; Williams et al., 2003).

Other primary structural and positional (kinematic) abnormalities thought to promote PFPS include any one, or a combination of the following:

- Genu valgum – a primary source of increased Q angle
- Femoral anteversion
- Internal tibial torsion
- External tibial torsion
- Hyperpronation of the foot
- Patella position (patella alta and baja [infera] or subluxation)
- General ligamentous laxity
- Patellar laxity (excessive side-to-side patellar glide measured in quadrants)
- Genu recurvatum
- Quadriceps atrophy

There has been strong evidence to indicate that femoral anteversion and/or internal tibial rotation caused by overpronation of the foot may be at fault in creating or exacerbating PFPS (Barton, 2011; Manske, 2008; Powers, 2003; Scherer, 2009; Servi, 2015). The amount of tibial internal rotation and the resulting transverse plane moment at the knee joint is determined by pronation of the subtalar joint and consequential collapse of the midtarsal joint. It has been further suggested that the amount and velocity of excessive foot pronation increases the amount and force of tibial internal rotation. This process increases the dislocation moment at the knee in the transverse plane (Scherer, 2009).

Pes equinus, produced by an osseous restriction in the ankle or a functional one via a shortened gastrocnemius or Achilles/soleus unit restriction of dorsiflexion in the sagittal plane indirectly increases internal tibial rotation as it is often compensated by subtalar overpronation.

To offset what were felt to be abnormal motions within these kinematic variables, traditional treatment focused on modifying them. For example using foot orthoses to reduce internal tibial rotations and perhaps the best known, vastus medialis obliques (VMO) strengthening to reduce the lateral vector forces on the patella. Inflexibility of the quadriceps, hamstring, and iliotibial band has also been implicated (Powers, 2003). Clearly there are myriad factors thought to contribute to PFPS.

**Differential Diagnosis**

Several patellofemoral conditions have been identified that have distinguishing symptoms and etiologies including instability (patellar subluxation/dislocation, giving out, pseudolocking) and patellar arthritis although the symptoms of PFPS may be concomitant with them. Similarly, patients with PFPS typically have more than one structural similarity with conditions associated with instability. For example, patellar tilt, patellar laxity, genu recurvatum, patella alta, patella baja (infera), and increased Q angle.

It is important to differentiate conditions that affect the knee. Within PF conditions, PFPS is not a diagnosis that includes features of instability (subluxation/dislocation/ “giving out”/ pseudolocking). Other non–PFPS pathologies include retropatellar crepitus, infrapatellar fat pad syndrome, and osteoarthritis (patella, femoral condyles). External to the PFJ are tendinopathies (iliotibial band, suprapatellar, infrapatellar), bursitis (pre–patellar, suprapatellar, pes anserine), systemic arthritis (lupus erythematous, rheumatoid arthritis, psoriatic arthritis, septic arthritis including Lyme disease) or internal derangements (ACL, PCL, MCL, LCL, meniscal, osteochondritis dissecans), Osgood–Schlatter’s, apophysitis, or synovial plica syndrome (Rush, 2017; Bigelow, 2017; Potter, 2017; Servi, 2015; Stefanyshyn, Stergiou, Lun, Meeuwisse, & Worobets, 2006; Williams et al., 2003)

Of the aforementioned conditions, synovial plica is a condition that mimics PFPS closely in several aspects.
Synovial plicae are normal structures found in many knees. Under normal circumstances, they are not associated with any painful conditions. However, with the right combination of events they can become quite problematic (Bigelow, 2017). Plica syndrome of the knee is a constellation of signs and symptoms that occur secondary to injury or overuse. An otherwise normal structure, a plica can be a significant source of anterior knee pain and is commonly interpreted as PFPS. Once an inflammatory process is established, the normal plical tissue may become hypertrophied into a truly pathologic structure. Often, symptoms resemble or overlap with those of other pathology. Reported symptoms include anterior or anteromedial knee pain; intermittent or episodic pain, clicking, snapping, occasional giving way, pseudolocking, and catching (Bigelow, 2017). Catching and true locking are common meniscal tear signs. Factors that aggravate synovial plicae may overlap with PFPS; symptoms include activity, stair climbing, squatting, or sitting (Bigelow, 2017). The most commonly affected site is the anteromedial aspect, which is anatomically in line with the medial retinaculum and anterior horn of the medial meniscus. Synovial plicae are typically diagnosed at the sports medicine or orthopaedic specialist level. There is no negative or positive link between synovial plicae syndrome and lower body mechanics to this date, although the abnormal biomechanics that have been implicated clearly affect all other PFJ tissues.

**Common Testing**

Although most tests for PFPS should be done by a sports medicine specialist, there are a couple of tests that a certified pedorthist can do safely during exam.

Patellar tilt test: With the patient in a supine position, the knee is extended and fully relaxed. The medial and lateral patellar borders are examined to determine a difference in height. The examiner places the thumb and index finger on the medial and lateral border of the patella. Both digits should be at the same height. If the medial digit is more anterior then the patella is laterally tilted; if the lateral digit is anterior to the medial digit then the patella is medially tilted (Grelsamer & McConnell, 1998).

Patellar compression test: With the patient supine and the knees extended, the patella is moved superiorly and inferiorly with pressure placed on it, causing a compression of the patella onto the femoral condyles. A positive result would be pain. There is some question in the medical community as to the validity of this test, as people presenting with pain in one knee may have a positive result in both knees (Hand & Spalding, 2004).

**Contraindications**

Contraindications include squats, squatting, and being seated for an extended period of time. The patient should avoid jumping sports such as basketball and volleyball.

**The Patient Perspective**

Patellar instability is associated with intermittent sharp pain at the patella, but can also occur with PFPS at a lower level of intensity. A feeling of “giving way” can be related to muscle weakness or to instability. Recurrent patellar subluxation or dislocation may cause an acute osteochondral fracture and subsequent chronic cartilage damage as a result of repeated microtrauma (Patel, 2018). Distinguishing PFPS from patellar arthritis requires an X-ray diagnosis. Retropatellar crepitus is meaningless without radiograph confirmation of osseous wear and tear. Patellar arthritis requires complete erosion of the patellar cartilage of at least one facet, in order for the patella itself to be eroded. Typically it is the femoral condyle that has eroded, but both are eventually involved.

Assessing a child’s pain and/or limp has a myriad possibilities, and a thorough medical investigation is often required (Rush, 2017). For example, hip pathology often presents with referred thigh or knee pain.

**Common Treatments**

The multifactorial nature of PFPS responds when treated in kind (Barton, 2011; Potter, 2017; Scherer, 2009). Primary treatments have traditionally included immediate pain management (physiotherapy, medications e.g. NSAIDs, ice, rest), attempting to normalize abnormal biomechanics (strengthening, foot orthotics, specific footwear recommendations, surgery), techniques to reduce the influence of tissues that pull the patella laterally, patellar taping and bracing, strengthening and activity modification (type, duration, intensity, frequency), avoidance (minimizing kneeling and squatting, learning landing/jumping techniques) (Barton, 2011; Lee et al., 2011; McClay, 2000; Patel, 2018; Powers, 2003; Scherer, 2009; Servi, 2015; Sheehan, 2010).

While the mechanics causing PFPS are still not fully understood, and the literature lacks consensus, there
are treatments that have been considered effective for many years. Researchers have more recently revealed greater understanding of the kinetics affecting the PFJ via improved methods of measuring. The general consensus is that there are even more complex variables at play than originally thought. Perhaps this explains why treatment results, effective or ineffective, are not reliably predictable. Since there are no absolute indicators of success, it is generally agreed that PFPS is best understood, assessed, and treated on a case-by-case basis. In other words, a treatment approach may work for one patient, but not the other. A repertoire of tools is essential.

Kinetic based force analyses suggests that treatment should focus heavily on strength training. This may in turn affect the timing and degree of movements acting on the knee. This has also been described as providing more controlled motion (Powers, 2003; Williams et al., 2003). These include coupled joint interactions between the foot, knee, and hip joint musculature with particular emphasis on the hip external rotators, gluteus medius and tensor fascia latae, and gluteus maximus which exhibit significant control of the knee in the transverse plane (Barton, 2011; McClay, 2000; Powers, 2003).

While not all knee pain should be attributed to abnormal mechanics, there is strong long-term evidence that foot orthoses can reduce or eliminate PFPS symptoms and even prevent injury by modifying frontal plane alignment and controlling overpronation, which in turn affects tibial rotation, dynamic genu valgum, and dynamic Q angle (Barton, 2011; Potter, 2017; Scherer, 2009).

Foot orthoses designed to control excessive pronation of the subtalar joint, with subsequent collapse of the midtarsal joint, are thought to prevent the coupling sequence described above. Ortotic control limits both the amount and velocity of the internal rotation (Barton, 2011; Scherer, 2009). More recent studies have questioned how orthotics function (McClay, 2000). Rather than orthotics simply blocking or reducing abnormal motion, they may be affecting the timing or “control” of subtalar joint overpronation by modifying the joint angle moments at the knee, hip, and foot (McClay, 2000).

A common approach in many areas of medical care involves short-term trials which can be good indicators of what treatment to pursue. This is a common technique within pedorthics. Evaluating to guide decisions related to foot orthoses prescription is not new. Vicenzino proposed the use of the treatment direction test (TDT) to identify individuals most likely to benefit from foot orthoses prescription (Barton, 2011). The basis of the TDT is to use an external control (e.g., adhesive strapping tape or temporary orthoses) to facilitate the desired motion control (e.g., reduce foot pronation in individuals with PFPS). Pain and quality of movement are assessed with and without this external control. If the trial produces a significant improvement, both patient and clinician have increased confidence that prescribing foot orthoses is likely to be effective (Barton, 2011; Scherer, 2009).

Barton (2011) studied dynamic foot function and the effects of foot orthoses on PFPS over a period of 12 weeks which demonstrated that “greater dynamic peak rearfoot eversion” was a reliable predictor of marked improvement following prefabricated foot orthoses prescription and that this type of measurement was more appropriate than static clinical measures of foot type. At this time there are no in-office validated methods of evaluating dynamic foot function so visual observation remains the norm. Barton (2011) also developed a set of criteria that predicted a significant improvement in PFPS symptoms from 25–78% if three of the following four criteria were met: immediate reduction of pain using an orthosis during a single leg squat, subjects used unsupportive footwear, their baseline pain was lower (less than 22/100), and reduced weight-bearing ankle dorsiflexion (less than 41 degrees) was present (pes equinus). Of these four predictors, immediate reduction of symptoms during a one-legged squat using an orthosis was the most predictive, meaning symptoms were reduced from 25% to 45% if orthoses were used over a 12-week period (Barton, 2011).

Although actual orthotic devices used vary greatly with materials, construction, casting, and flexibility, all are designed to limit foot motion between heel contact and heel lift, if not specifically to limit pronation of the subtalar joint (Scherer, 2009).

The recent predictors developed by Barton and TDT techniques that have been used in the past are useful tools that may quantify and therefore legitimate custom orthoses construction. The degree of subtalar eversion as a predictor of orthoses success (Barton, 2011) for example, may suggest that practitioners with the highest levels of competence in orthoses construction combined with competence in foot and lower limb mechanics, may be the best suited to creating positive outcomes. It may also seem logical that if non-custom moulded (pre-fabricated, generic, OTC) devices are beneficial, then custom moulded foot orthoses (CFOs) should provide a superior
benefit. However, the infinite levels and methods of correction in custom devices made today will ensure that studying the effectiveness of orthoses in PFPS, or any condition, will remain daunting. Theoretically at least, CFOs should be superior as the degree of subtalar and midtarsal control can be manipulated for maximum effect as needed whereas non-CFOs are typically very low control standardized devices.

Foot orthoses are often prescribed in an effort to augment other treatments or they may be a frontline treatment. Certainly the use of orthoses as an important element in the overall treatment approach is validated (Barton, 2011; Scherer, 2009; Sheehan, 2010; Vega, 2009). The direction of research suggests we will eventually be more successful in predicting their effectiveness and therefore avoid the manufacture of unwarranted orthotic devices than we are capable of today. One approach pedorthists may benefit from today relies on a thorough understanding of PF mechanics. The greater the number of abnormal mechanical factors at the level of the knee or hip that contribute to PFPS, the more patients can be educated as to what CFOs may or may not do. For example, CFOs in the presence of significant genu recurvatum, patellar laxity, Q angle, etc. could be assigned only a partial expectation of success. Orthotics are best considered an adjunct treatment (Barton, 2011) which is in line with the multi factorial nature of the condition, as are all treatments.

Footwear and footwear education plays as important role in managing most foot related issues including PFPS. Classic elements of good shoe construction are second nature to pedorthists. These include reliable heel counter reinforcements in a torsionally stable laced shoe, typical of larger sports and walking shoes. The only footwear that provides choice in the level of subtalar and midtarsal support and/or attempt to modify the timing of overpronation are running shoes. In patients presenting with overpronation and extremely poor footwear, simply prescribing a highly supportive shoe will have better results than in those presenting with excellent footwear. upgrades will often result in a significant reduction of symptoms. There is no literature that asks whether a minimal approach, i.e., a footwear-only prescription, is in the best long-term interests of the patient. Knowing that individuals with PFPS are often symptomatic 20 years after onset (Barton, 2011; Servi, 2015) suggests that, when foot mechanics are demonstrated to ameliorate symptoms, they need to be controlled long term as accurately and consistently as possible within the practical limits of each individual.

The control level of recommended footwear depends on hip, knee, and foot biomechanics. If orthoses are introduced, these recommendations typically change to avoid overcorrection issues (assuming the orthosis is custom moulded with a significant commitment to subtalar, midtarsal, or forefoot control). Therefore it is generally accepted that CFOs should be paired with neutral running and walking shoes in the majority of cases. Non-custom foot orthoses may be best paired with stability/motion control shoes if subtalar control is desired. Experience is often the only teacher when it comes to knowing when to break these guidelines. A clear example could include a PTTD acquired flatfoot where even aggressive orthotic devices need additional control from the footwear. In this example, a normal or retroverted PFJ could theoretically be overcorrected and produce PFPS symptoms or medial compartment compression, both undesirable consequences.

In recent years, shoe manufacturers have installed very soft durometer “crash pads” at the posterolateral heel of the midsole, extending more laterally than in the past. During heel strike, the crash pad compresses and delays the rapid STJ overpronation that often follows heel strike. As a result, midfoot overpronation is delayed and then often met by a medial midfoot stability device, e.g., a higher durometer midsole material. These changes reduce the speed and timing of STJ overpronation and are likely to be helpful in the subgroup experiencing excessive patellar anteverision caused by STJ overpronation. Pedorthists need to be aware of the potential for overcorrection in these cases.

**Physiotherapy/athletic therapy (PT/AT)**

A prospective randomized double-blind study of non-operative treatment in younger patients (aged 20–55) with patellofemoral pain (non-arthritic) revealed that symptoms resolved in 67% of patients within six months of initiating physical therapy, and 80% graded their knee function as excellent after seven years. However, clinical findings in these patients worsened with time. Significantly more patients had positive apprehension and compression test results and had crepitus at seven-year follow-up, and 5% of patients developed arthritis (Servi, 2015).

Physiotherapy has also been documented as being successful in the treatment of PFPS between 80–90% in other short-term studies (Servi, 2015). However, it is more difficult to know the long-term results. Between 71% and 90% continue to be symptomatic 20 years from diagnosis (Barton, 2011). Do some individuals become less symptomatic as the result of
dropping out of aggravating yet potentially healthful activity? Claiming success in the treatment of PFPS over the long term is difficult as many factors are yet to be determined, the greatest perhaps being whether the patients are able to maintain the levels of treatment required to keep them symptom free. Patients that carry on effective exercise prescriptions following initial therapy and exercise below the threshold of activity that develops symptoms will generally have the best outcomes (Servi, 2015; Vega, 2009). Although conservative management may help in resolving symptoms, no evidence indicates that it prevents the progression of arthritic changes in the joint (Patel, 2018).

PT/AT utilizes modalities: (ice, rest, interferential and TENS currents, ultrasound, laser etc.) to reduce inflammation, particularly the component of pain which promotes disuse atrophy and inhibits strengthening exercises. Disuse atrophy occurs rapidly after any knee injury. Strengthening exercises have traditionally focused on the VMO. Weak quadriceps strength has traditionally been considered a fundamental precursor to PFPS symptoms, and in some cases is the only cause of symptoms. However, where quadriceps is above average it is important to look elsewhere. As previously discussed, the shift to address kinetics influencing PFPS, for example attempting to counter knee and hip adduction moments with abductor and external hip rotator strengthening, has provided additional answers. It is perhaps important to maintain perspective in these discussions as no amount of strengthening can overcome all kinematic abnormalities.

Patellofemoral taping and bracing: Patellar taping to realign and stabilize the patella are among the first-line conservative treatments to reduce pain among patients with PFPS (Lee et al., 2011; Sheehan, 2010). Both are designed to stabilize and minimize lateral tracking of the patella. Taping can therefore reduce pain and allow strengthening exercises to begin earlier, and may function as a good indicator for patellofemoral bracing at a later date. Taping is believed to reduce pain through two mechanisms, similar to those thought to reduce pain in bracing: In the short term, the force of the tape medializes the patella within the femoral groove, temporarily unloading the inflamed peripatellar tissues, which leads to a reduction in pain. This short-term pain reduction enables the patient to more actively participate in rehabilitation (Sheehan, 2010; Vega, 2009). Pain is reduced or eliminated via lasting changes in muscle control, which in turn improves the knee’s dynamic stability to a point where taping is no longer needed (Lee et al., 2011; Servi, 2009; Sheehan, 2010). The second mechanism is suggested as a shifting of the location of the highest contact pressures to a different region of the patella or decreasing the pressure on the patella (Lee et al., 2011).

One widely used method is McConnell taping. This involves pushing the patella medially, then securing it in this position with tape on the skin (Potter, 2017; Sheehan, 2010).

Several types of braces used at this time include a patellar stabilization sleeve with a J-shaped buttress pad and medial external stabilization strap, a patellar stabilization sleeve with a C-shaped buttress pad and a lateral to medial external stabilization strap, and a wrap-style patellar stabilization brace consisting of a bifurcated strap with a C-shaped buttress pad positioned lateral to the patella (Lee et al., 2011; Potter, 2017; Sheehan, 2010).

Key Considerations

PFPS describes pain in and around the patella in the absence of other anterior knee pathologies, tendinopathies, or internal derangements of the knee. Medical diagnosis is required to rule out non-PFPS pathologies prior to pedorthic treatment. This allows treatment to move forward with confidence.

PFPS is not the correct descriptor if symptoms of instability are present regardless of post-instability episodes (e.g., patellar subluxation) reproducing many of the same symptoms. PFPS is very common particularly in runners (14–25% of all sports medicine injuries seen).

PFPS relates to the long-term progression of symptoms including patellar arthritis, suggesting it has a significant impact on overall health. It affects active adolescent females more than males but has no specific barriers to race or age. Activity levels and type are heavily implicated, as well as primarily affecting individuals under the age of 34.

PFPS is occasionally mimicked by other pathologies and the pedorthist should routinely refer patients back to the referring source if pedorthic treatment is not effective. An appropriate interval is two months when treatment is stalled.

Knee pain in children may be referred from the hip and other sources so a thorough medical examination is warranted. Lack of a quick response to pedorthic
treatment should definitely be questioned and referred on.

Patients describe PFPS in many ways but almost always as in and around the anterior knee. The vagueness of anterior knee ache, discomfort, stiffness, or pain is often clarifying. However it may be specific as “under the knee cap,” specifically slightly medial or lateral to the patella or both. An ache, stiffness, or sharp pains, are always worse after activity, but much less during. A classic sign is an annoying, persistent ache when sitting with the knee flexed at 90 degrees, often referred to as a positive moviegoer’s sign.

Pain associated with PFPS is multi-factorial in origin and primarily of a structural/mechanical origin being less affected by age and weight.

The pain associated with PFPS is thought to originate from two sources; compression of nociceptors of the patella underlying the articular cartilage, and soft tissues stresses created by kinematic and kinetic factors associated with the PFJ’s osseous, ligamentous, and musculotendinous components.

PFPS is most often a condition of management not cure. Multi-disciplinary treatment yields best results; pedorthists will be most effective working in cooperation with physicians and other allied health care professionals.

The mainstays of treatment include physio/athletic therapy, independent strengthening programs, activity modification, taping, bracing, pharmacotherapy, foot orthotics, and footwear prescription.

Management needs to focus on both kinematic and kinetic factors affecting the structure and function of the PFJ including the coupling of knee, foot, and hip control and motion.

Recognizing abnormal patellar alignment caused or exaggerated by abnormal foot mechanics, is sufficient evidence at this time to suggest foot orthotic therapy (at minimum trial using non-custom devices). New research suggests that a correctly interpreted biomechanical examination can be predictive of success with foot orthoses.

Encouraging patients to develop life-long habits including specific strengthening routines, consistent use of foot orthotics where applicable, avoidance of kneeling and activities below symptom provoking levels will assist in the long-term management of PFPS and likely minimize the development of patellofemoral osteoarthritis.

References


Patellofemoral Pain Syndrome
Iliotibial Band Syndrome

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Key Messages

- Iliotibial band syndrome is inflammation and pain above the lateral knee joint.
- It is a condition caused by overuse and biomechanical factors such as running, cycling, overpronation, leg length discrepancy, and genu varum.
- ITBS is slightly more common in women than men.
- Orthoses, proper footwear, rest, ice, and a change in activity can help decrease symptoms.

Keywords

femoral/femur condyle, hip, lateral knee, leg length discrepancy, overuse

Introduction

The iliotibial band (ITB) is a reinforced tract of fascia connecting proximally to the tensor fascia lata and gluteus maximus muscle, and distally to the lateral condyle of the tibia (aka Gerdy’s tubercle). Iliotibial band syndrome is inflammation and pain where the iliotibial band crosses the lateral femoral condyle; movement of the knee joint then becomes painful (Rattray & Ludwig, 2000).

Iliotibial band syndrome (ITBS) affects 12% of runners and is twice as likely to occur in female runners. (Jackson, 2017). Studies have indicated a 4.3–7.5% occurrence rate for ITBS in long-distance runners. ITBS is less common in shorter-distance or sprint-distance runners. The higher rate in long-distance runners is primarily because of the increased stance phase during longer-distance running; this has increased over the past 30 years for reasons that are unknown (Jackson, 2017). ITBS also has been reported in military recruits, cyclists, tennis players, Nordic skiers, and soccer players. The frequency of ITBS is also increased in adolescents undergoing the rapid growth phase (Martinez, 2009). It is also reported to be the leading cause of knee pain and the primary cause of lateral knee pain. Overall rates of ITBS vary from 2–25% in physically active individuals and have not been reported in individuals who are not active. It is said to be the second most common cause of knee pain due to overuse after patellofemoral pain syndrome (Jackson, 2017).

Pathology

The ITB supports the knee laterally, especially when the knee is extended; it also assists in flexion of the knee joint. ITBS is inflammation and pain where the ITB crosses the lateral femoral condyle; movement of the knee joint then becomes painful. Usually the pain is worse with continued movement, and resolves with rest. Some causes of ITBS are prolonged, repetitive activities where the knee and hip are flexed, such as running or cycling (Rattray & Ludwig, 2000), or a combination of overuse and biomechanical factors (Khaund & Flynn, 2005). Symptoms include pain on the lateral aspect of the knee with possible swelling at the site. There may also be a snapping or popping sensation as the knee is bent. Symptoms will often present after a certain mileage for long distance runners, cyclists, and in other athletes who squat repeatedly, such those who row and lift weights (Wedro, 2017).

Although the onset of this syndrome has been linked to problems with training, such as an increase in mileage on a pitched surface, research has indicated that poor mechanics and anatomical factors such as knee, forefoot and rearfoot misalignment, increased Q angle, IT band tightness, and size of the lateral femoral condyle all play a role in the syndrome. (Wedro, 2017). There can be a predisposition to developing IT band syndrome. Anatomy issues may include leg length discrepancy, an abnormal tilt to the pelvis, or bowed legs (genu varum). These situations can cause the IT band to become excessively tight, leading to increased friction and irritation as the band crosses over the femoral condyle with movement.
Training errors may cause runners to develop ITBS; on roads that are canted, when the runner runs the same way every time, the slope mimics a leg length difference. Running too many hills, especially downhill, can also cause runners to develop ITBS. Many patients will show a change in lower extremity kinematics, for instance, great peak hip abduction and greater peak knee internal rotation and femoral external rotation, when compared to control groups. Poor muscle performance, such as hip abduction, can further exacerbate the faulty biomechanics (Wedro, 2017). Cyclists may experience ITBS due to improper positioning on their bike, “toe-in” when they pedal, or if the foot placement is too narrow. If the toe clips are not aligned properly, the foot will be forced to internally rotate, and this can cause the same effect as bowed legs in that it increases the angle of the IT band as it crosses the knee, which in turn increases the risk of inflammation. (Wedro, 2017). If the problem is in internal rotation, externally rotating the cleat will help, or if clipless pedals are being used, switching to a floating pedal. Widening the cyclist’s bike stance and improving both hip and foot alignment will also help. This can be accomplished by placing spacers between the pedal and the crank arm. Saddle or seat position can also play a role and should be considered. Ideal position is when the knee has 30–35 degrees of flexion at the bottom of the pedal stroke (Stirling, 2015).

Long-distance runners have a higher incidence of ITBS than do short-distance runners and sprinters. This higher incidence may be due to the change in the biomechanics of running versus sprinting. Long-distance runners tend to have a more prominent and extended heel strike and stance phase in comparison with sprinters. The ITB is under its greatest tension during the first third of the stance phase.

Weakness of muscle groups in the kinetic chain may also result in the development of ITBS. Weakness in the hip abductor muscles, such as the gluteus medius, may result in higher forces on the ITB and the tensor fascia lata (Martinez, 2009). Studies have shown that long distance runners with ITBS have weak hip abductors on the affected side (Fredericson, Cookingham, Chaudhari, Dowdell, Oestreicher, & Sahrmann, 2000).

Differential Diagnosis

IT band syndrome can sometimes cause referred hip pain when the inflammation is at its proximal origin (Khaund & Flynn, 2005); however, ITBS will cause pain at the level of the knee and not the hip. Even though tightness of the band can have an influence on hip bursitis, it should not be confused with it (Joint Pain Solutions, 2011). In addition, symptoms that radiate down the lateral leg may not always involve the ITB. There are nerves that originate from the low back and superficial nerves that pass just below the knee joint that may create symptoms similar to ITBS. If the pain is non-specific, or if it spreads, it could be coming from these nerves (Joint Pain Solutions, 2011). ITBS can also be confused with popliteus tendinitis (Rattray & Ludwig, 2000). Other possible considerations could be biceps femoris tendinopathy, degenerative joint disease, lateral collateral ligament sprain, popliteal tendinopathy, or superior tibiofibular joint sprain (Saikia & Rodger, 2013).

Common Testing

ITBS is diagnosed on the basis of history and presentation complemented by the clinical findings. In most studies, Noble’s compression test is used to confirm the diagnosis of IT band syndrome. With the patient supine (lying on their back), flex the knee to 90 degrees and flex the hip. Pressure is applied 1–2cm proximal to the lateral femoral epicondyle. While the pressure is maintained, passively extend the knee. The patient will report severe pain at approximately 30 degrees of flexion (van der Worp, van der Horst, de Wijer, Backx, & Nijhuis–van der Sanden, 2012). Supplementary tests include Ober’s test and Renne’s test. Ober’s test is done with the patient lying with the affected side down and the unaffected hip and knee at 90 degrees. The examiner stabilizes the pelvis, then abducts and extends the affected leg until it is aligned with the rest of the patient’s body. The affected leg is lowered into adduction. If the leg remains in abduction and they have lateral knee pain, the test is positive for IT band syndrome. (Saikia & Rodger, 2013). Renne’s test is one that evokes the pain experienced while running. Have the patient stand on the affected leg, flex the knee to 30–40 degrees, and hold. If pain is experienced, the test is positive (van der Worp et al., 2012). A positive Trendelenburg’s sign suggests weakness of the gluteus medius, and therefore compensation will further alter lower extremity kinematics and aggravation of the IT band.

Contraindications

Few contraindications have been noted; however, patients should ensure that their footwear is appropriate and not worn out. Shoes that are too soft and unstable, or that are worn out (especially in the lateral aspect) can cause the foot to oversupinate; this forces the knee out laterally. Alternatively, if the
foot is allowed to go into excessive pronation, this will cause an increase in tibial internal rotation and, therefore, stress on the IT band. Obvious infection or transient pain near the lateral knee joint should not be treated as ITBS.

The Patient Perspective

Patients may report pain with activity, localized over the lateral femoral epicondyle, while climbing stairs or running downhill, they may also report pain at rest (Stirling, 2015). Patients may tell you that it is worse with time and continued activity. The initial lateral tenderness can progress into a more painful, sharp, and localized discomfort over the lateral femoral epicondyle and/or the lateral tibial tubercle. Typically, the pain begins after completion of a run or several minutes into a run; however, as the ITB becomes increasingly irritated, the symptoms typically begin earlier in an exercise session and can even occur when the person is at rest. If left untreated, the pain can radiate to the distal tibia and calf, and up to the lateral thigh. Patients often note that the pain is aggravated while running down hills, lengthening their stride, or sitting for long periods of time with the knee in a flexed position (Khaund & Flynn, 2005). There may also be a snapping or popping sensation as the knee is bent. Symptoms will often present after a certain mileage for runners and cyclists (Wedro, 2017).

Common Treatment

Custom-made foot orthoses can be used to help control excessive pronation and supination of the foot. Orthoses control the magnitude and timing of excessive pronation and reduce the internal rotation of the tibia, decreasing the pull of the IT band, and therefore the rubbing of the band across the femoral epicondyle. Orthoses that provide shock absorption and reduce varus alignment will help the supinated foot to take strain off the ITB. D’Ambrosia (2005) reported that of 200 individuals who used orthoses to manage a variety of running injuries, almost all had severe pronation with forefoot varus. Of the individuals with ITBS, 66% reported improvement with orthoses (Lusardi, Jorge, & Nielsen, 2013).

Proper footwear selection is also an important treatment for ITBS. Footwear selection is based on mechanics and also used to complement orthoses. Footwear with stability features such as a firm heel counter, medial lateral torsion, and medial support are an ideal choice for the overpronated foot. For a foot that supinates, it is best to choose a neutral, shock-absorbing shoe.

Other treatments could include: physiotherapy, anti-inflammatory medicines, corticosteroid injections, a change or decrease in activity, and (in rare cases) surgery. Physiotherapy treatments include modalities to decrease inflammation, such as ultrasonography, phonophoresis, iontophoresis, and icing. After the acute inflammation has subsided, one can begin stretches for the hamstring, glutes, and hip adductors (Stirling, 2015). Physical therapists may evaluate underlying causes and evaluate muscle strength and balance and/or flexibility and gait. A physiotherapist may also recommend orthoses if they find a gait problem, pelvic tilt, or leg length difference (Wedro, 2017). Stretching and active release therapy can also be of benefit, as well as strength training, especially for the hip abductors (Fredericson et al., 2000). Rest and ice are also helpful in treating ITBS.

Surgical intervention is only done in very rare cases, in which prolonged conservative treatment has failed to resolve the symptoms. The procedures include removing a portion of or Z-lengthening the IT band where it comes into contact with the lateral femoral epicondyle (Stirling, 2015).

Key Considerations

Iliotibial band syndrome usually is seen in individuals aged 15–50 years, an age range that generally includes active athletes (Martinez, 2009). ITBS has been reported in men and women equally; however, women may be more susceptible to developing the syndrome because of anatomical differences of the thigh and knee, such as genu varum and increased internal tibial rotation. These anatomical differences result in weakness of the quadriceps muscle and an increased varus angle of the femur in relation to the knee.

References


Chondromalacia Patella

Scott J. Harvie, BSc (HK), C. Ped (C)

Key Messages

• Chondromalacia patella is characterized by the degeneration of the patellar articular cartilage; by comparison, patellofemoral pain syndrome (PFPS) is defined as retropatellar pain resulting from physical or biochemical changes in the patellofemoral joint.
• One-quarter of all knee injuries are diagnosed as PFPS, with prevalence skewed toward the young, active female.
• Management of chondromalacia patella is directed by a conservative, multi-disciplinary treatment approach, addressing musculoskeletal and biomechanical factors.

Keywords

articular cartilage, cartilage, chondrocytes, compression, crepitus, edema, femoral anteversion, PFPS, Q angle, subluxation, tibial torsion

Introduction

First used by Aleman in 1917, chondromalacia patella was the name given to a degeneration of the patellar articular cartilage (Kipnis & Scuderi, 1995). Distinguished from patellofemoral pain syndrome (PFPS), true chondromalacia patella involves the degeneration of the articular facets of the patella, with resulting ragged fronded edges (Welsh, Hutton, Torg, Welsh, & Shephard, 1989) and is a very common injury amongst both male and female fitness enthusiasts (Williams, 1989).

By comparison, patellofemoral pain syndrome can be defined as retropatellar or peripatellar pain resulting from physical or biochemical changes in the patellofemoral joint. Patients with patellofemoral pain syndrome have anterior knee pain that typically occurs with activity and often worsens when descending steps or hills. It can also be triggered by prolonged sitting, referred to as “movie-goer’s knee.” One or both knees can be affected. Consensus is lacking regarding the cause and treatment of the syndrome (Cutbill, Lady, Bray, Thorne, & Verhoef, 1997). Patients presenting to a pedorthic clinic with knee pain will commonly be diagnosed with patellofemoral pain syndrome, as nearly 10% of all visits to an orthopaedic or musculoskeletal clinic will be for anterior knee pain; of those, 25% will be diagnosed as PFPS. The incidence of PFPS has been reported to be 2.2 times greater for females, and is more common in adolescence and early adulthood.

Pathology

Pathophysiology and etiology

The patella articulates with the trochlear notch of the distal femur. As the patella moves over the femur during flexion and extension, different parts of the femur are exposed to the patella, and in full flexion the patella and femur are most stable. This pattern of the patella moving over the femur is referred to as patellar tracking. Two important components of the patella’s movements over the femur are quadriceps force on the patella and the direction of the patella tendon. The patellar tendon (patellar ligament) inserts into the tibial tubercle, which, in the last 30 degrees of extension, rotates externally with the tibia. This creates what is known as the Q angle, “the angle between the line of application of the quadriceps force and the direction of the patella tendon” (Tria, Palumbo & Alicea, 1992). Regarded by many as the single most important factor contributing to patellofemoral knee pain, the outer limit for normal Q angle is 13–15 degrees (McConnell, 1986). An increase in Q angle, which may be associated with increased femoral anteversion, external tibial torsion, and lateral displacement of the tibial tubercle, increases the lateral pull of the patella (Ficat & Hungerford, 1977; Gruber, 1979; Insall, 1979; Malek & Mangine, 1981). Once all the alignment variables are accounted for, greater tibiofemoral angle and femoral anteversion are significant predictors of greater Q angle, in both males and females. Pelvic angle, genu
recurvatum, tibial torsion, navicular drop, and femur-to-tibia length ratio are not significant independent predictors of Q angle in males or females (Nguyen, Boling, Levine, & Schultz, 2009).

Many theories have been proposed to explain the etiology of patellofemoral pain, including biomechanical, muscular, and overuse theories. Trauma is also a cause of patellofemoral pain. Acute injuries normally occur when the front of the kneecap suffers an impact, such as falling directly onto it, or being hit from the front. This can result in small tears or roughening of the cartilage. The literature suggests that the etiology of PFPS is multifactorial.

**Cartilage changes in chondromalacia patella**

Normal cartilage is characterized by tangentially oriented collagen bundles at the articular surface and more deeply oriented fibres running down to subchondral bone (Reid, 1992). Cartilage changes occur due to excess compression which disrupts the intermediate and deep layers of cartilage of the patella’s articular surface (Reid, 1992). Changes occur to the subchondral bone—microfractures and sclerosis—both of which make the bone less resilient and lead to greater shock to the cartilage. Most chondromalacic changes are seen along the median ridge of the patella, and here cartilage is thickest (Kulund, 1988).

The severity of articular cartilage softening, or chondromalacia patella, can be classified using Outerbridge’s grading system:

- **Grade 1** – softening and swelling
- **Grade 2** – fissuring
- **Grade 3** – fibrillation (“crab meat” appearance)
- **Grade 4** – erosive changes and exposure of the subchondral bone

**Grade 1**

Chondromalacia patella begins with edema of the cartilage, which is easily damaged. Microscopically, there are small fissures in the cartilage and the chondrocytes appear normal (Vigorita, Morgan, & Scuderi, 1995). When probed, the cartilage will feel spongy (Reid, 1992).

**Grade 2**

Fissuring of the cartilage is present, but does not extend into the subchondral bone.

**Grade 3**

Fissuring, fragmentation and fibrillation, extending to the subchondral bone, can now be seen, but this degeneration covers less than 50% of the patella (Reid, 1992). Chondrocytes become hyperactive and begin to degenerate, seen as necrosis of the cartilage and some chondrocytes may become fibrous (Vigorita et al., 1995).

**Grade 4**

Chondromalacic changes extend into bone and involve more than half of the patella surface. Osteoarthritic-sclerotic changes take place and osteophytes are formed (Vigorita et al., 1995).

**Factors affecting patella alignment**

**Q angle**

Although some investigators believe that a large Q angle is a predisposing factor for patellofemoral pain, others question this claim. One study by Caylor, Fites & Worrell (1993) found similar Q angles in both symptomatic and asymptomatic patients. Another study (Thomeé, Renström, Karlsson, & Grimby, 1995) compared symptomatic and asymptomatic legs in 40 patients with unilateral symptoms and found similar Q angles in each leg. Furthermore, normal Q angles vary from 10 to 22 degrees (Reid, 1992), depending on the study, and measurements of the Q angle in the same patient can vary between practitioners (Tomsich, Nitz, Threlkeld, & Shapiro, 1996). Therefore, practitioners should be wary of placing too much emphasis on such biomechanical variants, as this can lead patients to believe that nothing can be done about their pain.

**Muscle tightness**

There are structures which, when tight, are reported to have an effect on patellar alignment. These are:

- The rectus femoris, which, if tight, affects patellar movement during knee flexion.
- The iliobibial band, which, if tight, will pull the patella laterally during knee flexion (McNicol, Taunton, & Clement, 1981; Noble, 1980).
- The hamstrings, which, if tight, will during running cause increased flexion of the knee, thus increasing compressive forces in stance (Winter, 1983). It has been suggested that the increased knee flexion will cause an increase in ankle dorsiflexion which cannot be adequately fulfilled by the talocrural joint, so that the subtalar joint assists, resulting in compensatory pronation (Root, Orien, & Weed, 1977).
The gastrocnemius which, if tight, will also result in compensatory pronation because dorsiflexion of the talocrural joint cannot occur and the movement is translated to the subtalar joint (Root et al., 1977).

**Hip joint kinematics**

Recent research has demonstrated a link between abnormal hip motion and strength and PFPS. Using dynamic magnetic resonance imaging techniques, researchers found that the primary contributor to lateral patellar tilt and displacement during weight-bearing was internal rotation of the femur underneath a stable patella (Powers, Ward, Fredericson, Guillet, & Shellock, 2003), suggesting that control of femur rotation may be important in restoring normal patellofemoral joint kinematics. As well, a systematic review of six studies that compared hip muscle strength between females with PFPS and control subjects concluded that there is strong evidence that females with PFPS exhibit impaired strength of the hip extensors, abductors, and external rotators (Prins & van der Wurff, 2009). Findings from these studies are consistent with investigations that have reported successful clinical outcomes in patients who have undergone hip focused training (Boling, Bolgla, Mattacola, Uhl, & Hosey, 2006; Mascal, Landel & Powers, 2003; Tyler, Nicholas, Mullaney, & McHugh, 2006).

**Excessive pronation**

Prolonged pronation of the subtalar joint is accompanied by a prolonged internal rotation of the leg resulting in malalignment of the patella and internal rotation of the femur. The quadriceps, therefore, will pull the patella laterally (Buchbinder, Napora, & Biggs, 1979; Subotnick, 1980). In adolescent boys, subtalar pronation, not Q angle, was found to be the single most significant predictor of patellofemoral pain (McConnell, 1984).

**Patella alta**

This is measured by means of a lateral X-ray where the height of the patella and the distance from the inferior pole of the patella to the tibial tubercle (i.e., patellar tendon length) are determined. Patella alta is present when the length of the patellar tendon is 20% greater than the height of the patella. The consequent high position of the patella predisposes the individual to patellar subluxation (Insall, 1979).

**Vastus medialis obliquus (VMO) insufficiency**

The function of vastus medialis obliquus is to realign the patella during extension of the knee (Basmajian, 1970; Lieb & Perry, 1968). It is the only dynamic medial stabilizer; any insufficiency of this muscle will increase the lateral drift of the patella (Gruber, 1979; LeVeau & Rogers, 1980).

**Differential Diagnosis**

There are numerous other causes of anterior knee pain (Reid, 1992), including:

- Osteoarthritis
- Peripatellar tendinitis and bursitis
- Fat pad syndrome
- Sympathetic dystrophy
- Vascular related syndromes
- Fractures
- Dislocation and subluxation
- Referred pain from hip or low back pathology

**Clinical features on examination**

It is important to remember that knee pain does not always accompany chondromalacia patella, and often the degree of chondromalacia patella does not correlate with the symptoms (Kipnis & Scuderi, 1995).

**The Patient Perspective**

Symptoms are experienced by the patient due to changes in the articular surfaces of the patella and/or the femoral groove (Appenzeller, 1988):

1. pain at the anterior aspect of the knee, during walking and running
2. anterior knee pain with squatting and walking up or down stairs or hills
3. recurrent effusion
4. crepitus during flexion and extension
5. rarely do patients experience symptoms at rest

On examination, the clinician may find (Appenzeller, 1988):

1. crepitus as the patella moves in the femoral groove
2. easy lateral subluxation of the patella
3. pain and recurrence of symptoms with passive movement of and simultaneous pressure to the patella within the femoral groove
4. pain with contraction of the quadriceps as the patella is held in the femoral groove and the knee is fully extended (Clarke’s sign)
5. Q angle greater than 15–20 degrees
6. genu valgum
7. femoral anteversion, external tibial torsion, and external rotation of the tibial tuberosity

When examining the patient for chondromalacia patella, the entire knee should be assessed in its position and movements, both passive and dynamic (Reid, 1992). The entire lower limb should be assessed, including muscle testing and range of motion at the hip, knee, and foot, as abnormalities in these areas may be reflected in the knee biomechanics. Patients should be assessed while walking and squatting, as pain is generally only present on movement.

**Common Testing**

**Patellofemoral compression test** – To perform the patellofemoral compression test, the patient should remain seated on a plinth, with legs hanging off the edge. As the patient’s knee moves through active and/or passive flexion and extension (within a range of 35 degrees), the pedorthist puts pressure directly on the patella. The test is positive for PFPS if the patient reports pain or discomfort while performing this test.

**Clarke’s sign** – Clarke’s test is performed to confirm a positive chondromalacia patella diagnosis. The patient should lie prone on the plinth while the pedorthist applies inferior compression (towards the toes) directly on the patella. While maintaining this level of compression, the patient should attempt an isometric quadriceps contraction. As contraction continues, the pedorthist should slowly increase the pressure applied to the patella. The test is considered positive if the patient exhibits hesitation to complete the contraction, or reports pain or discomfort while maintaining the contraction.

**Medial glide test** – This test is performed with the patient seated and involves isometric quadriceps contractions at five different flexion angles: 120, 90, 60, 30, and 0 degrees, while the femur is externally rotated. The contraction is sustained for at least 10 seconds as pain is often delayed in onset. It has been found in preliminary investigations that the VMO has phasic rather than tonic activity in patellofemoral pain sufferers, whereas VMO activity is tonic in subjects with no patellofemoral pain (Richardson, 1985).

While completing the medial glide test, the patient’s quadriceps musculature must remain relaxed during patellar manipulation. This test is best performed while standing on the lateral side of the effected leg, while the patient lies supine on the plinth. One hand is used as patella stabilization, placed on the medial surface of the femur. The opposite hand lies inferior to the patella, with both thumbs serving to mobilize the articulation. Without adding compression to the knee joint, the pedorthist can use both thumbs to glide the patella medially. While maintaining this medial position of the patella, the patient can repeat the painful isometric contraction of the quadriceps. If the patient reports a reduction and/or absence of pain, the medial glide test is positive for PFPS.

**Common Treatment**

There remains a strong emphasis on clinical experience to guide the testing and appropriate treatment for chondromalacia patella. It is strongly encouraged to consult recent publications, including several meta-analyses, supporting various treatment options for this condition.

**Relative rest**

Initially, knee activity should be reduced, at least relatively, because the theory that patellofemoral pain is an overuse/overload syndrome has merit (Buchbinder et al., 1979; Bockrath, Wooden, Worrell, Ingersoll, & Farr, 1993; Caylor et al., 1993; Cutbill et al., 1997). If patient’s experience pain with prolonged sitting, i.e., a positive “movie-goer’s” sign, periodic movement is commonly reported to alleviate knee discomfort. If the patient is a runner or engages in impact activity and insists on continuing some rigorous activity, swimming, cycling, or other non-impact aerobic activities are a reasonable recommendation.

**Ice and NSAIDs**

Icing the knee is the safest anti-inflammatory agent, but its successful use requires discipline. Applying ice for 10–20 minutes after activity is reasonable.

A frozen gel pack, crushed ice in a wet towel or plastic bag, or a bag of frozen peas works well. For pain, non-steroidal anti-inflammatory medication (NSAIDs), as directed by a physician, can help decrease discomfort.

**Exercises and physical therapy**

Quadriceps strengthening is most commonly recommended because the quadriceps muscles play a significant role in patellar movement; strengthening of the hip abductors, adductors, and flexors should
also be considered. Hip, hamstring, calf, and iliotibial band stretching may also be important, and referral to a physical therapist for an accurate physical examination and exercise prescription is highly recommended.

**Knee sleeves and braces, and taping the knee**

The use of knee sleeves and braces in patients with patellofemoral pain is controversial (Appenzeller, 1988; Buchbinder et al., 1979; Milgrom, Finestone, Shlamkovitch, Giladi, & Radin, 1996). Typically, knee braces have a C- shaped lateral buttress that keeps the patella from deviating too far laterally. However, the patellofemoral mechanism is not that simple, as the patella moves in several planes (Arroll et al., 1997; Basmajian, 1970).

Taping the patella into a certain position to reduce friction may be helpful, although results of studies have varied (Greenwald, Bagley, France, Paulos & Greenwald, 1996; Nobel, 1980; Powers et al., 2003; Prins & van der Wurff, 2009; Reid, 1992). A technique embraced by some physical therapists is known as “McConnell taping” (Reid, 1992). When performed correctly, taping may offer short-term pain relief; most physical therapists are trained in taping and can teach patients to tape themselves.

**Orthoses and footwear**

Arch supports or custom orthoses can be helpful in patients with a wide variety of lower extremity complaints, including patellofemoral pain (Root et al., 1977; Subotnick, 1980). Over-the-counter and/or custom foot orthoses may prove beneficial to patients with either a pes planus or pes cavus foot type. In pes planus individuals, the orthoses serve to minimize overpronation, consequently improving lower limb biomechanics, and improving patellar alignment. In pes cavus feet, increasing the plantar foot contact to the ground surface, widens the base of support, and consequently improves shock absorption to the lower leg. Orthoses to control motion in the planus foot will generally be posted medially to limit overpronation and the resulting internal rotation of the tibia. For the normal or cavus foot, total contact orthoses balanced to neutral will improve base of support and also provide an extra degree of cushioning.

Although correction of abnormal lower limb internal rotation in the presence of excessive pronation is the long-standing hypothesis for the mechanism of foot orthoses effectiveness (Caylor et al., 1993; Powers et al., 2003), the validity of this hypothesis remains unclear (Prins & van der Wurff, 2009; Reid, 1992). More recently, alternative hypotheses for the efficacy of foot orthoses in PFPS treatment have been proposed, including enhanced activation of the vasti and gluteal musculature as a result of improved plantar cutaneous afferent feedback (Richardson, 1985), and reduced lower limb muscle activity and joint moments through to enhanced footwear comfort and facilitation of preferred movement pathways (Root et al., 1977).

Footwear considerations include stability, or motion control characteristics for the planus, overpronated foot. These may include a stiff long medial heel counter, multi-density midsole, or anti-pronation bridging. Careful attention to fit is encouraged, as ill-fitting footwear may induce a biomechanical fault. Running on banked, uneven, rough surfaces can accentuate pronation of the foot. As well, running up or down hills will add to the stress on the patellofemoral joint, through constant flexion on the sloped terrain.

Even though the etiology and treatment of chondromalacia patella remain uncertain, most patients do well with a conservative treatment approach, including physical therapy to address muscle imbalances and pedorthic management through the use of appropriate orthoses and footwear.

**References**


Chondromalacia Patella


Chondromalacia Patella
Patellar Tendinopathy

Melissa Bendo, BKin (2012)
Updated by Winnie Law, B.Sc (BP), C. Ped (C) (2018)

Key Messages

- Patellar tendinopathy is localized to distal position of patellar tendon and its insertion to the inferior pole of the patella.
- It usually affects athletes whose sport involves repetitive explosive extension or eccentric flexion of the knee.

Keywords

anterior patella, eccentric contraction, high impact, jumper’s knee, knee flexion, non-inflammatory condition, Osgood–Schlatter’s disease, tendon overload, tendon regeneration

Introduction

Patellar tendinopathy is a degenerative change in the patellar tendon which originates on the distal end of the patella and inserts into the tibial tuberosity. It is also referred to as jumper’s knee and is typically associated with repetitive, forceful eccentric muscle contractions of the quadriceps during activity. It is characterized by local tenderness at its origin which is located on the inferior pole to the patella. Though the cause of the condition has not been clearly defined, tendon overloading during jump takeoff and landing is suspected to result in microtears within the tendon (Lian, Engebretsen, & Bahr, 2005). This has been suggested to be the cause of patellar tendinopathy in its chronic stages. Clinical studies have indicated that athletes who subject their patellar tendons to higher loads of increased knee flexion are at a higher risk of tendinopathy; however, the link between the mechanical loading conditions and the pathologic response remains unclear. This is often seen in volleyball and basketball where the players, typically at the elite levels, intensively train to develop high vertical jumps (Peers & Lysens, 2005). With these types of sports, focus is placed on both the concentric muscular action (with increased knee flexion) as well as the eccentric muscular contraction (during the landing phase of each jump). A study completed by Young & Ranson (2011), stated that it is the eccentric loading of the muscle groups that needs to be managed to improve the function of the patellar tendon which will then lead to reduction of patellar tendon pain.

The prevalence of jumper’s knee across different sports is mostly unknown. However, early studies from volleyball have shown that among male players at the elite level had 40–50% patellar tendinopathy (Ferretti, Ippolito, Mariani, & Puddu, 1983; Ferretti, Papandrea, & Conteduca, 1990). A study completed in Finland described 182 patients undergoing surgery for jumper’s knee and found that 46% were from athletics (track and field), 37% from volleyball, 5% from soccer, and the rest from other sports (Raatikainen, Kärpäkka, Puranen, & Orava, 1994). Another study completed in Belgium found that only 8% of the 90 surgically treated patients were from athletics, while 34% were volleyball players and 32% soccer players (Martens, Wouters, Burssens, & Mulier, 1982). The differences observed in the proportion of patients from different sports may simply reflect how popular these sports are in the different countries.

Pathology

The diagnosis of jumper’s knee is based on a history of pain localized to the lower patellar insertion of the quadriceps tendon in connection with athletic activity and distinct palpation tenderness corresponding to the painful area (Blazina, Kerlan, Jobe, Carter, & Carlson, 1973). The diagnosis of patellar tendinopathy requires a typical history taking with clinical signs.
and structural changes in the tendon that can be confirmed by an MRI or ultrasound. A diagnosis, however, is often based on history and clinical findings rather than ultrasound imaging, as structural abnormalities can be present even in asymptomatic individuals (Malliaras et al., 2015).

The severity of patellar tendinopathy can be assessed using the Victorian Institute of Sport Australia (VISA) Score or Roels’ clinical grading system (Roels et al., 1978). Roels’s grading system reflects the clinical seriousness of the disease, but is based primarily on clinical experience rather than research. In describing patients with jumper’s knee, Lian (2007) proposed a modification to the clinical grading system. There are several patients who are able to play matches and practice despite having pain throughout the activity, but there was no available classification category. The content in Table 1 was designed originally in 1978 and then later modified, splitting grade III into two subgrades (Lian, 2007). Grade IIIa was added for patients with pain during activity, but who are still able to train and play matches. Grade IIIb was added for those with disabling pain. This modification was added to categorize more precise patient classification in future clinical studies. The VISA score has been proven to be a reliable tool when assessing severity by measuring individuals’ (i) symptoms, (ii) performance in a simple test of function, and (iii) ability to undertake physical activity (Visentini et al., 1998). It uses a questionnaire and scale from 0 to 10 to measure the response of the individual. This provides a value that corresponds to the individuals’ health and it can range from 0 to 100 to represent health. This allows the VISA score to be highly reproducible and useful for determining the severity and measuring the outcome of therapy (Malliaras et al., 2015).

Extrinsic predisposing factors act externally on the human body (Nigg, 1988). The most common extrinsic factors are thought to be training errors, excessive loads on the body, poor environmental conditions, and poor equipment. Training errors are assumed to contribute 60–80% of tendon and other overuse injuries, and the main problems are thought to be too-high intensity and too-fast progression.

The overall current incidence of jumper’s knee was 23% (74 men and 19 women) in a study of 407 volleyball players from different playing levels. In athletes playing five times a week or more the incidence of jumper’s knee was 41% and an association was seen between the development of jumper’s knee and the training volume. However, the study did not find a correlation between career duration and the development of jumper’s knee (Ferretti et al., 1983).

The ground reaction forces during landing and take-off are different on different surfaces. The impact force, or the force at first contact, is much higher for running on asphalt compared with running on grass or sand (Nigg, 1988). The ground reaction force is at the same magnitude for those different surfaces, therefore it is speculated that these high-impact forces on harder surfaces could be a cause of overuse injury. It was found that 60.7% of the players with jumper’s knee played on a cement or linoleum floor, while only 4.7% of those diagnosed with jumper’s knee played on a wooden floor, suggesting a positive correlation between the hardness of the floor and the prevalence of jumper’s knee among volleyball players.

### Table 1. Classification of jumper’s knee according to symptoms as outlined by Roels et al. (1978) as modified by Lian (2007).

<table>
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<tr>
<td><strong>Grade I:</strong> Pain at the infrapatellar or suprapatellar region after practice or after an event</td>
<td>Same</td>
</tr>
<tr>
<td><strong>Grade II:</strong> Pain at the beginning of the activity, disappearing after warm-up and reappearing after completion of activity</td>
<td>Same</td>
</tr>
<tr>
<td><strong>Grade III:</strong> Pain remains during and after activity and the patient is unable to participate in sports</td>
<td>IIIa: Pain during and after activity, but the patient is able to participate in sports at the same level</td>
</tr>
<tr>
<td></td>
<td>IIIb: Pain during and after activity and the patient is unable to participate in sports at the same level</td>
</tr>
<tr>
<td><strong>Grade IV:</strong> Complete rupture of the tendon</td>
<td>Same</td>
</tr>
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Patellar Tendinopathy
(Ferretti et al., 1983), while it has recently been shown that jumper’s knee among elite beach volleyball players playing on sand is only 9%, considerably lower than for indoor volleyball players (Bahr & Reeser, 2003).

Young & Ranson’s (2011) study considered that the mechanism in patellar tendinopathy had more to do with the undeveloped loading of the quadriceps, hamstrings, and calf muscles upon the eccentric contraction phase when landing from a jump. It was stated that heavy loading of eccentric quadriceps muscle training for the treatment of chronic patellar tendinopathy was a popular type of exercise management for tendinopathy. However, few training programs have involved purely eccentric muscle actions that utilize loads anywhere near the eccentric capacity of the targeted muscle-tendon complex, or worked the associated muscle group in the most appropriate eccentric length-tension range of motion to promote strength improvements. Therefore, this type of approach could equally, and perhaps more appropriately, be termed high-volume, large-amplitude (range of motion), heavy-load, painful training for tendinopathy. A willingness to not only allow but encourage exercise into tendon pain followed the emergence of findings that most cases of tendinopathy probably were not due to a primary inflammatory process, and that pathological changes found on imaging were poorly correlated with pain.

**Differential Diagnosis**

Athletes with patellar tendinopathy usually present with localized knee pain, most frequently located at the proximal insertion of the patellar tendon to the patella, which is exacerbated by activity or sometimes by prolonged knee flexion. The onset of pain is mostly subtle, but often patients can relate it to a period of increased sport activity. In mild cases, pain will be present only after sport activities. When the tendinopathy progresses, patients also experience pain at the beginning of specific activities or even throughout their sport activity. At that time, patellar tendinopathy can hinder their performance. In severe cases, pain is present during daily activities and at rest (Peers & Lysens, 2005).

Anterior knee pain can be present in individuals of different ages and types of activities, and may involve varying anatomic structures of the knee (Malliaras et al., 2015). Anterior pain localized away from the inferior pole may indicate quadriceps tendinopathy or distal patella tendinopathy, and can be differentiated by the type of aggravating activity.

In contrast to the load-dependent movements that affect patellar tendinopathy, deep knee flexion and endurance activities such as running are the typical causes of quadriceps tendinopathy and distal patellar tendinopathy respectively. The infrapatellar fat pad can also be a cause for anterior knee pain, but is diffusely felt in the area of the patella and increases at the end of extension or with direct pressure. The most common differential diagnosis is perhaps patellofemoral joint pain as there are no specific clinical tests to diagnosis the condition and the general description of pain is very similar to that of patellar tendinopathy. The age of the individual should also be considered when diagnosing anterior knee pain; osteochondral lesions may be indicative for an older patient, while Osgood–Schlatter syndrome or Sinding–Larsen–Johannson syndrome are common in youth (Malliaras et al., 2015).

**Common Testing**

Common testing for patellar tendinopathy to confirm diagnosis consists of four tests:

1. Functional testing of the patellar tendon can be performed by decline squatting. Typically patients can only perform a limited number of pain-free decline squats (Cluett, 2006). Assessment of the supporting musculature such as the gluteus maximus, quadriceps, and calf muscles by repeated bridging, single leg squat, or repeated calf raises helps to identify relevant deficits. Foot posture, flexibility of the quadriceps and hamstrings, and ankle range of motion are also associated with patellar tendinopathy and should be assessed (Malliaras et al., 2015).

2. X-rays can be useful to identify associated bony abnormalities, such as in severe cases of Osgood–Schlatter disease or to detect the occasional intratendinous calcification.

3. Ultrasound can also be used as a tool for evaluation of the patellar tendon due to the tendon lying superficially and in parallel with the skin surface. The increased ground substance with degeneration of collagen fibers is often associated with tendon thickening (Campbell & Grainger, 2001).

4. MRI also documents patellar tendinopathy as a thickened tendon with areas of increased signal intensity. MRIs are not sensitive enough to detect small calcifications if present within in the tendon (Khan, Bonar, Desmond, Cook, Young, Visentini, & Wark, 1996).
Contraindications

The use of anti-inflammatory medication for a degenerative, non-inflammatory condition seems paradoxical and must be questioned. No conclusive evidence is provided in the literature to support any effect of NSAIDs in the treatment of chronic patellar tendinopathy (Weiler, 1992). NSAIDs might also have other effects than simply anti-inflammatory or analgesic effects. In the clinical management of chronic tendinopathy, the use of an NSAID might mask symptoms through analgesia and consequently hamper necessary therapeutic management. Therefore, until further evidence of true effects of NSAIDs in tendinopathy is provided, their widespread use in patellar tendinopathy is not evidence based. The use of corticosteroids is probably the most debated issue in the treatment of tendinopathy (Almekinders, 1990). Long-term effects of corticosteroids seem less favourable. Corticosteroid injections were compared with local physiotherapy and a wait-and-see policy (Smidt, van der Windt, Assendelft, Deville, Korthalk-de Bos, & Bouter, 2002). As a caution, injections cause increased risk for rupture until the tendon is reconditioned through strengthening. Patellar tendinopathy is a non-inflammatory property and corticosteroids have negative effects on collagen synthesis and tendon strength (Jozsa & Kannus, 1997).

The Patient Perspective

The previous lack of basic knowledge of the pathophysiology and pain mechanisms associated with patellar tendinopathy is reflected in the existence of many different treatment protocols. For less than 25 years, athletes with patellar tendinopathy have required exhaustive conservative treatment before proceeding to surgery (Ferretti et al., 1983). The current trend in the therapeutic approach for patellar tendinopathy suggests a more comprehensive approach with emphasis on strengthening exercises. As tendinopathy results in a failed healing response of overloaded tendons, it seems only logical that strengthening should be the key element in management of patellar tendinopathy.

Common Treatment

The most important first step in treatment is to avoid activities that aggravate the problem. The body is the best guide to know how much to rest the injured knee: if an activity hurts in the area of the injured patellar tendon, then you should rest from that activity.

Stretching the quadriceps, hamstring, and calf muscles is suggested in any stage of patellar tendinopathy (Cluett, 2006).

Ice is also recommended to control the inflammation and decrease swelling. By minimizing inflammation and swelling, the tendon can return to its usual state and perform its usual function.

Strength training focusing on eccentric loading of the knee (e.g., squats, lunges, knee extensions, leg presses) has been proven for rehabilitation of patellar tendinopathy, specifically the single-leg decline squat. A comparison of heavy slow resistance (HSR) training to cortisone injections in a study of 52 individuals demonstrated the long-term benefits of HSR. Although the short-time effects were similar in a 12-week period, the improvements observed from HSR remained at six-month follow-up (Kongsgaard et al., 2009). The physiological, neural, and mechanical characteristics of eccentric muscle actions have a positive effect on the muscle-tendon complex. There is a high specificity of strength gains with eccentric training, which may be due to the specialized neural pattern of eccentric actions. This specificity may be a reason to prescribe predominantly eccentric muscle actions in patellar tendon rehabilitation (Young & Ranson, 2011).

Depending on the severity of the tendinopathy, a support strap (called an infrapatellar strap or a Cho-Pat strap) or a knee brace will be suggested to wear while performing sport or activity.

A pair of custom-made foot orthoses can be prescribed to control the biomechanics of the foot and lower limb keeping the foot stable and allowing for any exaggerated movement at the knee to be limited.

Surgery is exceedingly rare for patients with patellar tendinopathy but regenerative injections have become increasingly common among athletes. Platelet-rich plasma injections have been shown to enhance the rate of recovery when injected at the tendon. It is believed that the ability of platelets to release growth factor increases the initial healing process but dissipates over time (Dragoo et al., 2014). Prolotherapy using dextrose has also been shown to successfully reduce pain and initiate tendon repair. Improvements in pain scores and ultrasound imaging demonstrate tissue healing specifically remodeling but require further histological analysis (Ryan et al., 2011). In other instances, direct injection of stem cells can also be used to improve pain and tendon repair. Direct placement of fibroblasts at the site...
of tendinopathy allows new collagen I and III to be added and appears to enhance the response to treatment (Clarke et al., 2010). On some occasions, patients have persistent patellar tendinopathy symptoms despite thorough conservative treatment. In these chronic cases, where the above treatments have been tried for at least one year, surgery can be considered. Surgery is usually considered if a portion of the tendon can be seen as degenerative on the MRI.

**Key Considerations**

Patellar tendinopathy is a threat to many athletes. Its diagnosis is basically clinical; however, technological advancement of imaging techniques has provided more diagnostic options. Improved knowledge of the non–inflammatory, degenerative pathology of chronic tendinopathy must provoke clinicians to shift their therapeutic accents from anti–inflammatory approaches towards a more complete rehabilitation based on eccentric exercises. Pain in tendinopathy patients remains largely unexplained and further explanation is needed. New emerging techniques can lead to improved curative and preventive measures and can decrease patellar tendinopathy in the future of sports and athletes.

**References**


Infrapatellar Bursitis

Nancy Kelly, BSc (Kin), C. PedTech (C), C. Ped (C)

Key Messages

• Goals of treatment should be to reduce stress and pressure on the infrapatellar bursa.
• Footwear and orthoses may be beneficial in addressing abnormal biomechanics which may increase stress on the bursa.
• Infrapatellar bursitis may occur concurrently and secondarily to other knee conditions, such as Osgood-Schlatter disease.
• Medical interventions such as injections, aspirations, and possible surgery may be necessary in instances where conservative and nonsurgical interventions are unsuccessful or infection is suspected.

Keywords
clergyman’s knee, crepitus, kneeling, OSD, osteophyte

Introduction

The infrapatellar bursa is comprised of two portions: a deep or posterior bursa, and a superficial or anterior portion (LaPrade, 1998). The deep portion lies between the patellar ligament and the proximal tibia (LaPrade, 1998), while the superficial portion lies between the patellar ligament and the subcutaneous tissues (Waldman & Campbell, 2011).

Infrapatellar bursitis, also known as “clergyman’s knee,” is a condition where one or both portions of the infrapatellar bursa are inflamed. Derscheid & Malone (1980) described bursitis in general as a traumatic condition as the inflammation results from stress to the bursa. Arnold (2006) described bursitis as a common and often overlooked or misdiagnosed cause for pain surrounding the joints of the lower extremity. This condition has also been described as one of many different causes of anterior knee pain (LaPrade, 1998; Wai, 2004).

Infrapatellar bursitis has been described more frequently in individuals who have repetitive strain/trauma to the anterior and inferior aspect of the knee, such as those whose occupation requires regular kneeling (Waldman & Campbell, 2011). This is likely where the term “clergyman’s knee” evolved.

It is important to note that in the absence of inflammation or disease, the bursa contains little fluid (McCarthy, Murphy, Doran, & Cunnane, 2011).

Pathology

There are a number of factors that can result in the bursa becoming inflamed. Trauma to the bursa, overuse injuries, infection, and inflammatory disorders (e.g., arthritis) can result in infrapatellar bursitis (Arnold, 2006; McCarthy et al., 2011; Patel & Holman, 2008).

Structural changes and/or altered biomechanics have also been associated with it (Arnold, 2006; McCarthy et al., 2011). The mechanics of recreational and occupational activities can be a factor in the development of this condition. For example, individuals who have occupations that involve a great deal of dropping onto the inferior aspect of the knee, kneeling, and/or crawling (such as floor layers/installers) have a higher incidence (Arnold, 2006; Berg, 2004; Nordin, Andersson, & Pope, 2007; Waldman, 2008). Recreational endeavours that involve repetitive jumping and running on uneven surfaces have also been linked to infrapatellar bursitis (McCarthy et al., 2011; Waldman & Campbell, 2011). Anatomical malalignment, such as tibia varum, pes cavus, and leg length differences that contribute to varus strain at the knee have been found to contribute to this injury (Arnold, 2006; Weiss, Weiss, & Pobre, 2010). Muscular weakness, strength imbalances, and decreased flexibility in the musculature surrounding the knee may also influence the biomechanics and structural alignment of the knee (Arnold, 2006; Weiss et al., 2010).
The literature reveals that infrapatellar bursitis can occur in conjunction with other knee conditions. Lagier & Albert (1985) described bursitis of the deep infrapatellar bursa accompanying enthesopathy of the tibial tuberosity in some patients with juvenile ankylosing spondylitis. Infrapatellar bursitis has also been described to occur secondary to Osgood–Schlatter disease (Kujala, Kvist, & Heinonen, 1985).

Arnold (2006) has described several other physical conditions as contributing factors. Degenerative joint disease and osteophytes at the knee can result in increased pressure in the region of the infrapatellar bursa and the exacerbation of symptoms. Obesity may also increase pressure at the anterior knee, resulting in exacerbation of symptoms.

Common symptoms of infrapatellar bursitis include localized pain with tenderness on palpation of the inferoanterior knee (Arnold, 2006; Waldman & Campbell, 2011). Palpation of the region of the bursa provokes tenderness (Frontera & Silver, 2002) and may be exacerbated with movement of the knee joint. Specifically, full–range passive flexion, full–range active extension, and resisted knee extension may elicit a painful response (Derscheid & Malone, 1980; Waldman & Campbell, 2011). Pain complaints are often worse at night (Waldman & Campbell, 2011). Range of motion may be decreased with this condition but full range of motion does not rule this out as a diagnosis (Patel & Holman, 2008). An intermittent decrease in active range of motion of the knee, with full passive range, can be characteristic of infrapatellar bursitis (Patel & Holman, 2008). Warmth, swelling and edema may be present; however, swelling is more easily identified in the superficial bursa (Patel & Holman, 2008; Waldman & Campbell, 2011). Patients with this condition will commonly have difficulty or inability to climb stairs and/or kneel (Waldman & Campbell, 2011). Some patients also report crepitus or catching of the knee (Waldman & Campbell, 2011).

**Differential Diagnosis**

As the symptoms related to infrapatellar bursitis are also symptoms associated with a number of other knee complaints, it is often known to be misdiagnosed (Arnold, 2006). The inflammation and pain complaints that have been associated with this condition are also identified in those patients with Osgood–Schlatter disease (LaPrade, 1998).

Anterior knee pain is another common complaint associated with a diagnosis of this condition; however, there are many other conditions that can result in an anterior knee pain complaint, including patellofemoral pain syndrome, patellar tendinopathy, Osgood–Schlatter disease, and Perthes disease (Magee, 2002; Wai, 2004). Tendinopathy is another possible misdiagnosis as the sporadic loss of active range of motion with maintained passive range of motion is characteristic of both conditions (Patel & Holman, 2008).

Sanchis-Alfonso (2010) also noted the less common possibility of musculoskeletal tumors. Clinically, tumors may have a presentation mimicking anterior knee pain syndromes.

**Common Testing**

Common physical examination testing for infrapatellar bursitis includes palpation of the deep portion of the bursa, as this region will be tender on palpation (LaPrade, 1998; Waldman & Campbell, 2011). This is pertinent to diagnosis and proper treatment (LaPrade, 1998). Range of motion testing is often included. Movements that stretch the muscles overlying and/or compressing the bursa should be included, as these can provoke pain in the affected bursa (Arnold, 2006).

Other recommended testing involves occasional imaging of the knee joint using ultrasound or MRI (Berg, 2004; Waldman & Campbell, 2011). Although not routine, imaging can be important as bursitis may be clinically difficult to differentiate from tendinopathies and ligamentous injuries (Arnold, 2006).

A tap or aspiration with analysis of the bursal fluid may be recommended, particularly if the symptoms are unilateral and infection is suspected (Arnold, 2006; Berg, 2004; Waldman & Campbell, 2011). If inflammatory arthritis is suspected, further lab tests may be necessary (Waldman & Campbell, 2011).

**Contraindications**

Although compression of the knee joint has been suggested as part of the treatment plan for acute cases of infrapatellar bursitis, it is important that this be done in such a way as to not increase pressure directly on the region of the bursa (Patel & Holman, 2008; Weiss et al., 2010).

**The Patient Perspective**

Patients with infrapatellar bursitis generally present with pain at the anterior aspect of the knee. Pain is

Infrapatellar Bursitis
often reported to be constant but may be intermittent (Patel & Holman, 2008). A patient might report sharp pain with activity or movements that increase pressure/compression of the bursa; however, in some cases all movements of the knee may provoke pain (Waldman & Campbell, 2011). Decreased tolerance or an inability to kneel or perform other activities that increase pressure at the anterior–inferior aspect of the knee are common in those with this condition. Stiffness and/or aching pain is often reported following activity and at the end of the day. As well, some patients will report decreased range of motion and decreased tolerance for stairs. Patients may also note swelling at the anterior aspect of the knee and occasionally at the medial aspect of the knee.

**Common Treatment**

Initial treatment for acute infrapatellar bursitis includes local anti-inflammatories (heat or ice) and non-steroidal anti-inflammatory medications for reduction of inflammation (Patel & Holman, 2008; Waldman & Campbell, 2011). Rest and avoidance of those activities that provoke pain and symptoms, such as sporting activities and kneeling, are encouraged (Berg, 2004). Protection of the overlying skin and elevation of the limb can also be beneficial in treating the acute symptoms (Weiss et al., 2010). Protective equipment, such as knee or kneeling pads, should be used to reduce further injury to the infrapatellar bursa (Berg, 2004).

Physiotherapy to decrease inflammation and an exercise program to increase range of motion and muscular flexibility can decrease stress on the bursa and reduce pain (Waldman & Campbell, 2011; Weiss et al., 2010).

As altered biomechanics have been found to contribute to the development and exacerbation of symptoms of this condition, footwear and/or orthoses may be appropriate, although there is little discussion of this in the literature. In those who have a cavus foot type and decreased pronation of the foot, increased varus strain can result at the knee joint. Footwear that has a strong lateral column and custom orthoses to decrease the supinated position of the foot can be beneficial in treating infrapatellar bursitis. For those individuals who have increased pronation of the foot, this may result in increased internal rotation of the tibia and femur, which may increase pressure on both the deep and superficial infrapatellar bursae. Increased pressure can result in increased inflammation. In this case, orthoses and/or footwear that provide increased support to the medial longitudinal arch, and assist in limiting pronation and increased internal rotation of the tibia and femur, can be effective in reducing inflammation of the infrapatellar bursae and accompanying symptoms.

In more chronic cases where conservative treatments have not been successful, injections of local anesthetics and steroid medications may be necessary (Waldman & Campbell, 2011; Weiss et al., 2010; Wheeless, 2013). For those cases where there is persistent pain and/or progressive reduction in function, surgery to perform a partial or full excision of the bursa may be necessary (Waldman & Campbell, 2011; Wheeless, 2013).

**Key Considerations**

Both deep and superficial infrapatellar bursitis can occur through all age groups and it has been reported to occur more in men than women (Waldman & Campbell, 2011). Younger athletes and those whose occupational demands require kneeling and/or crawling have also been found to have a higher incidence (Waldman & Campbell, 2011). Obesity has also been identified as a contributing factor (Arnold, 2006). As the structure and mechanics of the foot can be influential on the biomechanics of the knee joint, it is important to assess the entire lower extremity.

Other conditions that can increase pressure or mechanical stress on the infrapatellar bursae are osteophytes or degenerative joint diseases (Arnold, 2006). This should also be considered for those presenting with anterior knee pain in the region of the bursa.

**References**


Infrapatellar Bursitis
Patellar Instability

Stan Sklepowicz, HBSc (Kin), BSc (PT), PT (reg), C. Ped (C)

Key Messages

- Most often seen in adolescence.
- Recurrent subluxations have anatomical variations (patella alta).
- Reducing lower extremity internal rotation can reduce lateral patellar tracking.
- Multiple treatment approach is most effective (foot orthoses, quadriceps strengthening, bracing and activity modification).
- Surgical intervention may be required.

Keywords

dislocation, femoral notch, IT band, medial patellofemoral ligament, Q angle, squinting patella, subluxation, vastus medialis

Introduction

Patellar instability is a condition that allows the kneecap (patella) to move into excessive ranges due to a decrease in patellar control. This can result in patellar subluxation. Patellar subluxation is a partial dislocation, usually lateral, of the kneecap. The subluxation is as a result of a complex interaction of the muscles and ligaments, the bony fit of the patella into the femur, and the lower leg alignment (Wunschel, Leichtle, Obloh, Wulkern, & Muller, 2011). This combination of factors causes the line of pull on the patella to move in a more lateral direction. Patellar subluxation is part of a spectrum that includes patellar instability at one end and patellar dislocation at the other end.

Generally this condition is seen in adolescents, with an incidence rate of 31 per 100,000 in the 10–19 age group, with an equal distribution among the sexes (Fithian, Paxton, & Cohen, 2004). Patellar subluxation and dislocation were also identified in a population that is active in sports, having a family history of patellar dislocation (9%), lower extremity problems as an infant (3%), and a history of birth complications (4%). Over 50% of this population also had radiographic evidence of patella alta (Atkin, Fithian, Marangi, Stone, Dobson, & Mendelsohn, 2000). Patellofemoral subluxation is probably the most common knee problem seen today (Henry, 1989; Werner, 2014; Antinolfi, Bartoli, Placella, Speziali, Pace, Delcogliano, & Mazzola, 2016).

Pathology

Kneecap stability is due to joint geometry, connective tissue support, and muscular control. The best fit between the kneecap and the groove in the femur occurs at approximately 20 degrees of flexion where the patella enters the femoral notch. When the knee is locked out, the kneecap sits above this notch and is susceptible to subluxation. Whether the patella will sublux is determined by the height and slope of the notch in the femur. The soft tissue support consists of the retinaculum, the medial and lateral patellofemoral ligaments, the iliotibial band, and the quadriceps tendon through the patellar tendon. The muscles that control the kneecap are the quadriceps muscles, especially the VMO muscle (vastus medialis obliquus) (Canadian Physiotherapy Association, n.d.). Any abnormality of any of these structures could result in patellar instability and subluxation.

Usually, there are multiple causes for patellofemoral instability. There is a complex interaction of the active, passive, and static structures. This interaction can be disturbed by anatomical variation or trauma (Schoettle & Weiler, 2007). The main predisposing factor is insufficient trochlear geometry, i.e., trochlear dysplasia (Schoettle & Weiler, 2007). Also, it may involve patella alta, lateral femoral condyle dysplasia, vastus medialis insufficiency, general joint laxity, tight lateral structures like the iliotibial band, and/or trauma. (Davida & Nathwani, 2012) The medial patellofemoral ligament is the primary soft tissue control of the kneecap; it provides 50–60% of
the passive medial restraint (Buckens & Saris, 2010). The ligament guides the patella into the groove as the knee bends. Once the patella is engaged in the groove there is increased pressure on it as a result of the combined forces of the quadriceps and the patellar and femoral tendons, and increasing bony contact. Thus, the patellar stability increases as the knee bends. With further knee bend, the lateral wall of the femoral notch provides the main resistance to lateral movement of the kneecap (Andrish, 2008).

The VMO has been identified as a contributing factor in the stability and positioning of the patella. As it contracts, it pulls the kneecap medially and prevents the patella from tilting upwards. This helps maintain the fit between the lateral side of the patella and the femoral notch (Pal, Draper, Fredricson, Guillett, & Shellock, 2003).

Obviously, any abnormalities in the supporting structures or joint fit will result in patellofemoral instability and could lead to joint subluxation and dislocation. Medial patellofemoral ligament injury has been identified as a primary cause of patellar subluxation (Garth, Connor, Futch, & Belarmino, 2011). The congenital under-development of the vastus medialis muscle has also been identified as a contributor to an abnormally shaped patella and femur. This results in the increased pull of the vastus lateralis muscle, causing abnormal growth and development of the lateral femoral condyle and patella, causing lateral patellar migration (Fox, 1975).

Lower extremity alignment has an effect on patellofemoral instability. External tibial torsion has been recognized as a cause of recurrent patellar dislocation (Canadian Physiotherapy Association, n.d.). Surprisingly, an increased Q angle has been shown not to have an effect on lateral patellar movement (Pal et al., 2003). In fact, the Q angle was decreased in knees that had suffered dislocations, whether traumatic or habitual. The non–traumatic group also showed greater rotation between the tibia and femur and increased lateral patellar movement (Sanfridsson, Arnbjornsson, Friden, Ryd, Svahn, & Jonsson, 2001). Repeated patellar subluxation and instability is directly related to an elevated kneecap and a shallow notch of the femur (Fox, 1975). The pain associated with patellar instability is caused by elevated fluid pressure and stress on the joint (Farrokhi, Keyak, & Powers, 2011). With increased patellar pain and joint pressures, there is impairment of the VMO, causing weakness, poor kneecap tracking, and increased internal tibial rotation.

Although many lower extremity misalignments have been associated with patellar instability, further complications are required to cause subluxation and dislocation. There must be a weakness in the soft tissues surrounding the patella, due to either a traumatic event or an underlying hypermobility (Fithian, Nomura, & Arendt, 2001). With a traumatic event, at least one of the medial restraining structures must be injured, usually the medial patellofemoral ligament (Wheeless, 2011). If the initial injury does not completely heal or if the characteristics of the joint allow for it, the patellofemoral joint becomes unstable and begins to subluxate. This can become a chronically recurring condition such as chondromalacia patella, which is characterized by anterior knee pain and swelling, particularly with prolonged sitting or stair climbing (Wheeless, 2011).

It is therefore important to identify an unstable patella to prevent the further complications that we have listed, if possible.

**Assessment**

Patellar instability is a clinical diagnosis that is based on an accurate history and supported by some examination findings. The single most important subjective report in patellar subluxation is the sudden unexpected giving out of the knee during daily activities, with or without minimal trauma. Statements like: “I was just walking down the street and my knee gave out for no apparent reason!” are common with patellar instability and subluxation. This is different from ligamentous tears, which result in knee instability occurring with aggressive knee movements such as in sports activities (“I was pivoting during my basketball game and I felt a pop and my knee gave out”). This sudden kneecap giving-way does cause a great deal of apprehension (Andrish, 2008). The patient’s report of pain is usually secondary to sudden instability that occurs with innocuous activities. Typically, there is some anterior knee pain and swelling around the kneecap. There can be acute severe pain associated with each episode that subsides fairly quickly.

The physical examination of a patient with a subluxing patella has some notable features. The patient may appear apprehensive, since they may have had a previous experience of the patella suddenly subluxing and the knee giving out. The gait pattern may be abnormal with unilateral differences being seen on the affected side. The client may use an unusual pattern that they feel will help to avoid further episodes (Andrish, 2008). Often the lower limb
Patellar Instability

is held in a completely internally rotated position, which allows the client to maintain the kneecap in a stable position against the femur. If the client is slightly uncomfortable, the gait pattern most often seen is a kneeling-in gait (Andrish, 2008). Typically, at midstance, there is significant pronation of the foot, with internal tibial rotation and a valgus knee. This is frequently described as being secondary to femoral anteversion as well as internal tibial torsion and the numerous foot pathologies that may result in a prolonged and exaggerated pronation (James, 1995). This gait pattern with the internal rotation and valgus thrust generates an external rotatory force about the knee and a lateral force against the kneecap (James, 1995). Although this pattern is attributed to structural abnormalities, it may be a result of neuromuscular impairment (Zeller, McCrory, Kibler, & Uhl, 2003). Timing differences were seen with people who suffered from patellofemoral pain. These people spent a significantly longer time in stance phase while walking as compared to a healthy population. It was felt that this was due to a generalized quadriceps weakness rather than a quadriceps muscle imbalance (Mohr, Kvitne, Pink, Fideler, & Perry, 2003). Weakness of the core musculature could also directly impact this gait pattern (Steinkamp, Dillingham, Markel, Hill, & Kaufman, 1993).

In standing, the posture demonstrates the “squinting patella” sign and may also include a genu valgum, tibial varum, femoral anteversion, excessive rearfoot eversion, a lowered longitudinal arch, and internal rotation of the tibia. Although these characteristics are present with clients with anterior knee pain, they are not necessarily the cause of the subluxation (Sheehan, Derasari, Fine, Brindle, & Alter, 2007).

With the client lying supine the natural position of the patella can be observed, and also if atrophy of the quadriceps muscles is present. Any joint effusion can also be observed at this time.

On palpation the patient demonstrates a high level of apprehension when the area around the patella is palpated. If the patella is moved laterally there will be a sudden protective contraction of the quads and the client will have an apprehensive look (a positive Fairbanks apprehension test) (Dutton, 2008). When the patella is moved outwards there should be a firm feel of resistance and the amount of lateral movement should be 25-50% of the width of the patella (Wheeless, 2011). The outward movement should not only be checked when the knee is fully straight but also with the knee is bent to 30 degrees, where the patella should be fully stable. A more sensitive test for patellar instability is to move the kneecap down and outwards (Tanner, Garth, Soileau, & Lemons, 2003).

When the patella is palpated, it can be determined if it is in a patella alta position. Feeling the bottom tip of the patella and comparing it to the joint line of the knee can determine patella alta. The tip should rest below the level of the knee joint; if the tip is at or above the joint line, there is patella alta. Having the client bend the knee while the patella is being palpated will allow the examiner a close look at the amount of lateral movement that occurs during movement and the final outward position of the patella.

Having the client contract the quadriceps may elicit patellofemoral joint pain or a grating feeling under the kneecap, which would indicate damage to its undersurface. It also allows evaluation of how effective the quadriceps contraction is. A quad lag may be present, which indicates a significant weakness of the quadriceps mechanism (Andrish, 2008). At this point the J sign may become evident. This is a sudden outward jerk of the kneecap at the end of the knee locking out (Sheehan et al., 2007), and a reverse J sign can be seen with knee bending and a sudden inward movement of the kneecap is seen (Wheeless, 2011). The excessive outward movement is seen more easily when there is no weight on the leg and the knee is straightened, as opposed to straightening the knee in standing. This is due to the patella rotating on the femur in sitting and the femur rotating underneath the patella in standing (Powers, Ward, Fredricson, Guillet, & Shellock, 2003).

Traditionally, a Q angle measurement was also taken but recent research has invalidated this measure as an identifier of patellar instability (Powers et al., 2003; Cooney, Kazi, Caplan, Newby, St. Clair Gibson, & Kader, 2012). When motion analysis was done to the kneecap during regular movement of the knee, the data indicated that Q angle did not relate to lateral kneecap instability (Sheehan et al., 2007; Freedman, Brindle, & Sheehan, 2014). In fact, outward movement of the patella was opposite to a measure of a large Q angle (Eng & Pierrynowski, 1993). Dynamic measures of knee valgus during stair ascent and squats seem to provide a more accurate picture of patellar lateralization. (de Oliveira Silva, Briani, Pazzinatto, De Azevedo, Ferrari, & Aragão, F., 2015). Radiographic measures of tibial tubercle/trochlear groove distance of more than 20mm and ratios involving patellar width or trochlear width greater than 0.5 provide a better predictor for patellar instability. (Camp, Heidenreich, Dahm, Stuart, Levy, & Krych, 2016). Although any one of the findings could be present...
with an unstable kneecap, none can be said to be universally present with all patellar subluxations. To arrive at a diagnosis of kneecap instability, an overall picture must be developed between the history, subjective, and objective findings.

X-ray reports are also important as they may identify abnormal bony features that are associated with patellar subluxation. These include a high-flying kneecap (patella alta) or an abnormal flattening of the notch that kneecap sits in. Kneecap positioning and outer placement of the kneecap attachment have also been identified as causative factors (Nove-Josserand & Dejour, 1995). These characteristics are relevant to this condition (Dejour, Walch, Nove-Josserand, & Guier, 1994) as they will cause instability but will not be altered by pedorthic intervention.

**Differential Diagnosis**

Subluxation of the patella needs to be distinguished from:

- Medial synovial plica
- Multipartite patella
- Chondromalacia
- Jumper’s knee
- Patellar ligament rupture
- Sindig-Larsen-Johanssen disease (apophysitis of the growth plate at the inferior pole of the patella)
- Quadriceps contracture (patient shows habitual patellar dislocation in flexion)
- Medial collateral ligament tears
- Medial or lateral meniscus tears
- Anterior/posterior cruciate ligament tears

(Wheless, 2011)

**Precautions**

Care must be taken in the care of individuals with patellar instability. An individual who demonstrates a general joint laxity could easily dislocate if the patella is aggressively moved (Schoettle, Werner, & Romero, 2005). Strong palpation could flare the pain and inflammation associated with this condition. Aggressive over-correction of foot pronation and tibial internal rotation in a client who has patellar hypermobility could cause an opposite inward (medial) subluxation.

**Common Treatment**

With patellar subluxation and dislocation, whether this is a first episode or a recurrent injury will determine what type of treatment is necessary. A traumatic versus idiopathic history also plays a factor in the selection of treatment. Typically, first–time injuries are immobilized to allow the medial restraints to heal, and early mobilization is contraindicated (Maenpaa & Lehto, 1997). Control of immediate symptoms such as pain and swelling are addressed with the typical anti-inflammatory treatments of rest, ice, compression and elevation as well as anti-inflammatory medication. A history of a dislocation results in patellar instability, anterior knee pain, and daily activity limitations in 50% of the patients and a re-dislocation rate of between 15% and 40% (Hawkins, Bell & Anisette, 1986).
A non-surgical approach may be successful with patients who have an isolated series of subluxations that have no significant mechanical or joint abnormalities that would automatically make them surgical candidates.

In the presence of biomechanical dysfunction of the lower leg, proper footwear needs to be addressed, and if there is significant biomechanical derangement to the foot, an orthosis is required (Dutton, 2008). Stability in running shoes has been shown to decrease the peak knee internal rotation throughout stance phase. (Hutchison, Scharfbillig, Uden, & Bishop, 2015). With increased subtalar joint pronation, there is a compensatory femoral internal rotation that results in a change in patellar alignment, an increase in the lateral tracking of the patella, and an abnormal pull of the quadriceps (Tiberio, 1987). These combined factors together with bony differences in the kneecap can result in patellar subluxation. It can be surmised that a reduction in foot pronation may influence patellofemoral tracking by decreasing the rotation in the lower leg segment (Buchbinder, Napora, & Biggs, 1979). A foot orthosis has been shown to affect the knee adduction moment and move the line of the ground reaction force laterally. (Roodsari, Esteki, Arminian, Ebrahim, Mousavi, Najdoleslam, & Bahramian, 2017). A foot orthosis can reduce foot and ankle pronation and move the center of pressure laterally (Delacroix, Lavigne, Nuytens, & Cheze, 2014). Controlling foot pronation will also limit hip adduction and internal rotation of the knee (Lack, Barton, Woledge, Laupheimer, & Morrissey, 2014), and thus lateral kneecap tracking (Eng & Pierrynowski, 1993). However, although foot orthosis has been shown to have a major effect on the foot kinematics, the effects on hip, knee, and pelvic kinematics are generally minimal. Changes seen in the leg are presumed to be as a result of changes in the proprioceptive mechanism affecting the active and passive soft tissues of the lower limb. (Nester, van der Linden et al., 2014). Muscular activity has been shown to have an altered temporal pattern of a shorter activation time and a delayed start time when an orthosis is used in the leg and hip (Dedieu, Drigeard, Gjini, Dal Maso, & Zanone, 2013; Lack et al., 2014). This distal to proximal action of foot orthosis allows for a reorientation of the bony segments and muscular rehabilitation (Delacroix, Lavigne, Hasdenteufela, Legranda, & Nuytens, 2011). With a regular population, a medial wedge under the bare foot resulted in a significant internal movement of the kneecap (Klingman, Liao, & Hardin, 1997). In a running population, semi-rigid custom orthoses decreased rearfoot movement and impact measures as well as maintaining knee position (MacLean, Davis, & Hamill, 2008). This not only decreased knee pain but also likely reduced excessive kneecap outward movement. A foot orthoses can change anterior knee pain originating from the kneecap (Collins, Crossley, Beller, Darnell, McPoiil, & Vincenzino, 2009; McPoli, Vincenzino, & Cornwall, 2011; Saxena & Haddad, 2003). This is most likely due to decreasing the rotation of the lower leg, reducing kneecap pressure and lateral tissue stress (Gross & Foxworth, 2003).

Kneecap pain responded best to an off-the-shelf orthosis that allowed the foot a forefoot valgus of at least 2 degrees, passive great toe extension of 78 degrees, and a navicular drop of 3mm (Sutlive, Mitchell, Maxfield, McLean, Neumann, Swiecki, Hall, Bare, & Flynn, 2004). Also, decreased ankle flexion and decreased pain using a foot orthosis while performing a single leg squat can predict success using an orthosis with patellofemoral pain syndrome (Barton, Menz, & Crossley, 2011) and indirectly with patellar subluxation.

We can infer that some of the kneecap pain is related to patellar instability but the studies on orthoses and patellofemoral pain indicate there is little direct evidence that custom foot orthoses will affect patellar subluxation. In fact, due to the limited number of studies with controlled random trials at present, the evidence for usefulness of a variety of foot orthoses in the treatment and prevention of various lower limb injuries is still not very strong (Hume, Hopkins, Rome, Maulder, Coyle, & Nigg, 2008); however, biomechanically speaking, there seems to be some intuitive justification in using a shoe insert that reduces foot pronation, and thus lower leg internal rotation and lateral patellar movement.

**Positive lateral patellar instability. Photo by Stan Sklepowicz.**
Non-surgical treatment for patellar instability also requires a rehabilitation program. Generally a physiotherapy program would consist of quadriceps sets, straight leg raises, hip strengthening exercises, and flexibility work of the hip flexors and hamstrings (Henry, 1989). Vastus medialis strengthening has been identified as a key to decreasing poor kneecap movement when there is evidence of abnormal patellar positioning (Sutlive et al., 2004). Strengthening the vastus medialis leads to control of tibial rotation and thus kneecap movement (Eng & Pierrynowski, 1993; Wunschel et al., 2011). The VMO is an important stabilizer of the patella but is no longer identified as the key to stabilizing the kneecap (Sakai, Luo, Rand, & An, 2000). Improving core stability should also be included as this improves control of hip rotation and the amount of movement in the knee. Together, these should improve control over patellar mobility (Dutton, 2008). Activities such as jumping, repeated stair climbing, kneeling, and prolonged sitting with excessive knee bend should be avoided after a recent episode of patellar subluxation.

Footwear recommendations for patellar subluxations would be in line with trying to assist in the correction of the biomechanical abnormalities previously discussed. Wearing shoes that assist in controlling abnormal foot movement on a regular basis may assist in reducing the incidence of patellar instability and subluxation. Less supportive footwear has been related to patellofemoral pain (Barton et al., 2011). Shoes that have a strong heel counter and shank would reduce the amount of rearfoot eversion and foot pronation that results in internal leg rotation and lateral patellar displacement. A technical athletic shoe that has motion control features like a dual density midsole, medial posting, reinforcement of the medial longitudinal arch, a medially reinforced rigid heel counter, a closure system that fits the shoe snugly to the foot, and a flared outsole would assist in the reduction of patellar instability. Improving the existing footwear by adding an internal medial heel wedge and an arch cookie should assist in maintaining kneecap positioning during gait. Adding medial posting, a medial flair or even medial buttressing of the shoe if the foot pronation is extreme can modify midsoles of existing shoes (Decker & Albert, 2002).

Patellofemoral bracing using some type of external patellar support is often recommended as an adjunct to the rehabilitation program. A brace offers a restraining force against lateral displacement with varying degrees of success. Patella bracing usually prevents the kneecap from moving excessively and/or corrects the positioning of the kneecap. Orthopaedic surgeons commonly prescribe bracing for acute kneecap subluxation, and patellar supports are one of the more common treatments for patellar instability. At present there is minimal evidence-based support for bracing in the literature (Shellock, 2000).

**Referrals**

Referral to the treating physician can occur for X-rays, which will help to identify the extent of the bone and joint alterations, i.e., patella alta, trochlear dysplasia, or torsional deformities of theibia or femur (Dejour et al., 1994; Malghem & Maldague, 1989). This could affect the outcome of a pedorthic treatment. Further referral to an orthopaedic surgeon could also occur if there is suspicion of medial patellofemoral ligament laxity or injury. A referral should also occur if there is reported or evident of major instability of the patellofemoral joint (Andrish, 2008). The most important referral would be for physiotherapy as the improvement in patellofemoral pain and subluxation is significant when there is a combination of vastus medialis strengthening with the introduction of a foot orthosis (Eng & Pierrynowski, 1993; Henry, 1989; Neptune, van der Linden, & Bowker, 2000; Pal et al., 2011; Sutlive et al., 2004).

**Recommendations**

Further research is required with random control trials to determine the effectiveness and corrective mechanism of foot orthosis on patellar subluxation and maltracking. Conservative treatment of patients with isolated subluxations with minimal mechanical or joint abnormalities can be effective.
Abnormal pronation resulting in internal rotation of the lower extremity and lateral patellar displacement can be helped with foot orthoses and quadriceps strengthening.

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Meniscal Injuries

Amy Chapman, C. Ped (C) (2012)
Updated by Sonia Page, BSc. Kin., C. Ped Tech (C), C. Ped (C) (2018)

Key Messages

• The menisci are discs of fibrocartilage located within the knee to provide cushion between the tibia and femur.

• Meniscal injuries are categorized into four different types of tears according to age, location, or orientation: longitudinal, bucket-handle, horizontal, and parrot beak.

• Excessive compression and shear forces combined with rotation of the knee are often the most common mechanism of meniscal knee injuries.

• Although acute injuries may be able to heal on their own without intervention, surgery, orthoses, or physical therapy may aid in a full recovery or relief of symptoms.

Keywords

Apley’s, bounce home test, bucket handle, compression, fibrocartilage, McMurray’s test, parrot-beak, rotation, shear force

Introduction

The menisci, also known as semi–lunar cartilage, are discs of fibrocartilage located within the knee to provide cushion between the tibia and femur. The menisci are attached to the tibial plateaus by various ligaments including the coronary ligaments and the joint capsule (Anderson, Hall, & Martin, 2005).

Table 1. Menisci shape, attachments, and pathway.

<table>
<thead>
<tr>
<th>Shape</th>
<th>Attachments</th>
<th>Pathway</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Medial Meniscus</strong></td>
<td>Semi–circular and narrows anteriorly, Posterior horn is larger than the anterior horn.</td>
<td>Intercondylar area just in front of the anterior cruciate ligament (ACL), and stabilized medially by its attachment to the medial collateral ligament (MCL).</td>
</tr>
<tr>
<td><strong>Lateral Meniscus</strong></td>
<td>Circular in shape and covers a larger portion of the tibial plateau than the medial meniscus.</td>
<td>Joint capsule, intercondylar tubercles and transverse ligament.</td>
</tr>
</tbody>
</table>
The menisci are attached to each other anteriorly via the transverse ligament and to the infrapatellar tendon via the patellomeniscal ligament (Anderson et al., 2005). The lateral meniscus’s attachment to a lax capsule and lack of attachment to the lateral collateral ligament (LCL) increase its mobility, therefore decreasing the chance of lateral tears (Hall & Brody, 2005). The medial meniscus is securely attached to the tibia, making it less mobile and more prone to injury (Anderson et al., 2005).

Table 2. Types of meniscal injuries.

<table>
<thead>
<tr>
<th>Description</th>
<th>Source</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Longitudinal</strong></td>
<td>Extend parallel to the C-shaped circumference.</td>
<td>Acute Can be partial or incomplete tears.</td>
</tr>
<tr>
<td><strong>Bucket-Handle</strong></td>
<td>Complete tear of the meniscus support / ligament resulting in the meniscus flipping over and being stuck in the middle of the knee.</td>
<td>Typically traumatic Largest meniscal tear. Occur in approximately 40% of meniscal injuries.</td>
</tr>
<tr>
<td><strong>Horizontal</strong></td>
<td>Occur parallel to the tibial plateau, separating the upper and lower parts of the meniscus.</td>
<td>Degenerative Most common meniscal tear. Most often affects the posterior medial portion.</td>
</tr>
<tr>
<td><strong>Parrot Beak (Oblique)</strong></td>
<td>Lie perpendicular to the free edge of the meniscus but then curve around to run parallel to the C-shaped fibers of the meniscus.</td>
<td>Acute Often found in adolescents with a history of meniscal trauma.</td>
</tr>
</tbody>
</table>


Excessive compression and shear forces combined with rotation of the knee are often the most common mechanism of meniscal knee injuries (Thompson & Floyd, 2004). Meniscal injuries are categorized into four different types of tears according to age, location or orientation: longitudinal, bucket-handle, horizontal, and parrot beak (Anderson et al., 2005).

Knee injuries account for almost 5% of all acute injuries treated in physicians’ offices, emergency rooms, and out-patient clinics. Ten percent of these injuries are severe soft-tissue injuries including meniscal tears and ACL injuries, two of the most common soft-tissue injuries of the knee. Half of all meniscal and ligamentous injuries to the knee are a result of sport injuries. The annual incidence for meniscal injuries is 1/1,000. Seventy percent of all ACL injuries are coupled with meniscal trauma due to their articulations. MCL tears are also coupled with meniscal tears when a valgus external rotation force is applied to the knee, such as in a ski accident where the ski gets lodged in the snow and creates a twisting motion in the leg (Bahr & Maehlum, 2004).

Chronic, degenerative tears typically do not elicit pain or dysfunction as they result from multiple episodes of minimal trauma. Constant shearing forces tear away at the inner meniscus which may lead to detachment and momentary locking, pain, and instability. A horizontal cleavage tear is a result of degeneration which often affects the posterior medial portion of the meniscus (Anderson et al., 2005). Acute tears are classified based on the type of tear present. Longitudinal tears happen when a twisting motion occurs with the knee flexed and the foot in a fixed position. These tears can be partial or complete, and can affect the peripheral or inner areas of the meniscus. Bucket-handle tears occur in only 40% of meniscal injuries. The entire longitudinal segment of the meniscus is displaced medially toward the centre of the tibia which may cause the knee to lock at 10 degrees flexion. Parrot-beak tears are another form of an acute meniscal tear but often found in adolescents with a history of meniscal trauma, or whose menisci may be less mobile at the periphery. In this form of meniscal tear, two tears often occur in the middle of the lateral meniscus forming the parrot-beak shape (Anderson et al., 2005) (See table 2).
Pathology

As in the jaw and sternoclavicular joints, the meniscus is a form of knee joint cartilage which grows inward from the joint capsule and forms a pad (Saladin, 2004). This “pad” deepens the surface of the tibia and provides a greater, more stable, surface area for femoral articulation. The meniscus distributes forces evenly across the entire knee joint aiding in shock absorption and reducing friction (Anderson et al., 2005). The meniscus transmits 40–50% of a compressive load across the knee when in full extension and 85% when 90 degrees flexed (Hall & Brody, 2005). Other functional advantages of the menisci are prevention of hyperextension, resistance of anterior tibial displacement, and prevention of joint capsule intrusion into the joint during the locking mechanism (Anderson et al., 2005). The locking mechanism of the knee joint refers to the femur rotating slightly medially on the tibia to lock the knee joint in place during full extension (0 degrees) and 20 degrees of knee flexion.

Seventy-four percent of the total weight of the meniscus is made up of water which is squeezed into the joint space when the knee is compressed. This aids in lubrication while providing a smooth surface for the bones to glide over (Anderson et al., 2005). The meniscus is primarily composed of type 1 collagen and is more fibrous than articular cartilage. The meniscus obtains most of its nutrition by diffusion and has a low metabolic and repair rate. It makes up for its low repair rate by using its blood supply to assist in the healing of tears. The meniscus receives its blood supply from the medial and lateral superior and inferior geniculate arteries. The outer one-third of the meniscus is also referred to as the red zone as the meniscus’s vascular supply only penetrates 10–30% of the width of the medial meniscus and 10–25% of the lateral meniscus. The middle area is called the red-white zone while the centre is the white, or avascular zone (Hall & Brody, 2005). The outer one-third of the meniscus is also the only area to contain nociceptors which innervate this area and send signals of pain to the brain upon injury (Anderson et al., 2005).

Differential Diagnosis

Patellar injuries may be misdiagnosed as a meniscal tear as both injuries present with acute, sometimes sharp pain in the knee. When a patient has a patellar injury, the onset of pain is typically a direct blow, overuse, or training errors versus a twisting pain. Pain is commonly located in the anterior knee or spread out throughout the joint. Patellar pain will not elicit a true locking of the knee as in a meniscal injury, just catching and stiffness after immobility. Meniscal pain is stimulated with weight-bearing, where patellar pain will also extend into the evening with rest. Pain with activity is not directly related to cutting movements but is intensified with use of extensors when squatting, jumping, and climbing stairs, meniscal injuries are typically not irritated by these movements. Pain is also present with patella injuries when the patella is compressed such as with kneeling or sitting. These movements do not irritate a meniscal tear as they do not load or stress the meniscus (Starkley & Ryan, 2003).

Ligamentous injuries in the knee may cause knee pain which may be confused with meniscal pain. However, general clinical tests such as anterior and posterior drawer test, and valgus or varus stress tests can confirm a ligamentous injury (Starkley & Ryan, 2003).

Common Testing

There are three common clinical tests used to confirm meniscal lesions. McMurray’s test places the patient supine while the practitioner moves the leg through various ranges of motion attempting to trap the meniscus. The knee is flexed and extended while internally rotated, the same range of motion is performed while the knee is externally rotated. If a meniscal tear is confirmed the test will produce a clicking, popping, or locking in the knee or a sensation of pain (Anderson et al., 2005; Starkley & Ryan, 2003). A false positive result may produce an audible clicking sound if the patella is tracking improperly or if the client has chondromalacia. Acute swelling may not allow the required range of motion needed to complete this test, as full flexion is needed to isolate the posterior horn (Anderson et al., 2005).

Apley’s compression test is performed with the patient lying prone with their knee flexed at 90 degrees. The practitioner stabilizes the thigh, and rotates the lower leg internally and externally, all while applying downward pressure to compress the meniscus between the tibia and femur. Pain with compression and rotation that is relieved when the lower leg is distracted is a positive result for a meniscal tear. If the pain occurs when the leg is distracted, this is a positive sign for ligamentous or joint capsule damage (Anderson et al., 2005; Starkley & Ryan, 2003).

The bounce home test has the patient lying supine with their heel resting in the practitioner’s hand. Meniscal Injuries
The knee is flexed through its range of motion then passively allowed to extend. If complete extension is not achieved with a spring-like end feel, the test is positive for a possible meniscal tear (Anderson et al., 2005).

**Contraindications**

Deep squats are discouraged when a meniscal injury is present. The knee flexion increases sheer forces, which promote anterior displacement of the femur, and stresses the ligaments and menisci (Anderson et al., 2005).

**The Patient Perspective**

Meniscal injuries are most commonly paired with pain and joint line tenderness along the medial or lateral joint line near the collateral ligaments. Anterior joint line pain is rare, unless a bucket-handle or ruptured bucket-handle tear has occurred (Anderson et al., 2005). If acute, the patient will recall a twisting injury to the knee causing a sharp pain with loaded weight-bearing or twisting (Starkley & Ryan, 2003). The patient may experience a popping, grinding, clicking, or locking of the knee, while they also may be unable to fully extend the knee. Duck walk (out-toeing) and deep squats will elicit pain as they involve rotation and flexion, two movements known to irritate a meniscal tear. Swelling may be present more than 12 hours after the injury, as the tensile forces associated with a meniscal injury can irritate the synovial lining of the knee joint (Anderson et al., 2005).

**Common Treatments**

When deciding on the treatment method for a meniscal tear it is important to know the type of tear and extent of damage as treatment will vary from one situation to the next (Hall & Brody, 2005). Acute cases are managed like many acute soft tissue injuries using RICE (acronym for Rest, Ice, Compression, and Elevation), non-steroidal anti-inflammatories (NSAIDs), and offloading with crutches as needed. An immediate referral may be necessary if the injured knee is locked and unable to be spontaneously mobilized, or if joint effusion is excessive (Anderson et al., 2005). Acute trauma to the meniscus may heal without repair if the tear is longitudinal or peripheral (Hall & Brody, 2005). Surgical

Degenerative (chronic) tears often require surgery to remove loose fragments and stimulate the healing of articular cartilage which may have also sustained lesions over time (Hall & Brody, 2005). Bucket–handle tears can be fixed surgically without removing the meniscus if found within 1–2 weeks, although the internal (“white zone”) portion of the tear will not regenerate (Anderson et al., 2005; Bahr & Maehlum, 2004). Full and partial meniscectomy are surgical treatment options which may be appropriate in some situations (Hall & Brody, 2005). Arthroscopic meniscectomies are done as an outpatient procedure and use local anaesthesia, with return to function after 2–6 weeks (Anderson et al., 2005). When a partial tear creates mechanical inabilities, meniscal repair or a partial meniscectomy may be performed. Although the goal of a partial meniscectomy is to treat the tear along with its symptoms, removing a portion of the meniscus will change the load distribution at the knee joint, and increase peak stress in certain areas. If necessary, a full meniscectomy may be the treatment of choice. When 10% of the surface area of a meniscus is removed by a partial meniscectomy, the peak local stress is increased by 65%. If a total meniscectomy is performed, this decreases the contact area by 75%, and increases local peak stress by 235%. With a full meniscectomy, changes to the femur include osteophyte ridging, flattening of medial condyles, and narrowing of joint space. These changes are known as the Fairbank changes (Hall & Brody, 2005).

Adaptation to meniscal surgery depends on a number of factors. Recovery time is important, if overused, the joint may become irritated and inflamed, causing pain and a possible overload leading to a rupture at the original trauma site. Those with malalignment, degenerative joint disease, limited range of motion, and poor quadriceps or neurological function, will have a difficult time adapting to the changes after surgery. Injuries to the “white zone” and complex radial tears should be allowed longer recovery time from surgery (Hall & Brody, 2005).

**Orthoses**

Many studies have focused on the effects that orthoses have on preventing knee injuries. There have been mixed results although the different parameters of each study must be kept in perspective. Devices and methods in each study varied greatly regarding materials, construction, casting, and flexibility, however all were made to limit foot motion between heel contact and heel lift (Scherer, 2011).
Data collection also varies amongst every study, some studies focus on three dimensional ankle and knee kinematics (MacLean, Davis, & Hamill, 2008; MacLean, van Emmerik, & Hamill, 2010; Williams, McClay Davis, & Baitch, 2003), others measuring ankle and knee joint angles and moments (MacLean, Davis & Hamill, 2006), while some use injury as a measurement of effectiveness (Jenkins, Raedeke, & Williams, 2008). Most studies mentioned above used an inverted rearfoot angle and saw results in decreased rearfoot maximum eversion angle and velocity. The rearfoot inversion angle used in each study seemed to be related to the decrease in knee kinematics noted. Williams, McClay Davis, & Baitch (2003) believe that there is a coupling between the rearfoot and knee motions. As the foot pronates, the talus rotates internally and in turn, due to its tight articulation with the tibia causes an internal rotation to occur. These motions may lead to abnormal alignments at the knee joint, promoting shear forces at the knee and increasing risk for common knee injuries including meniscal tears.

It has been found that pronation of the subtalar joint, combined with midtarsal joint collapse, produces excessive internal rotation of the tibia causing the knee to move into a greater valgus position. It has also been suggested in previous studies that the amount and velocity of excessive pronation increase the force of internal tibial rotation, producing a greater mechanical moment in the transverse and frontal plane (Scherer, 2011).

Although most studies explore the relationship between custom foot orthoses and ligament injuries or patellafemoral pain syndrome, most seem to conclude that the use of custom foot orthoses decrease torsion and sheer forces at the knee joint which in turn decreases the chance for meniscal injury. More specific research is needed to determine the exact link between abnormal foot mechanics and different knee injuries, including meniscal tears (Scherer, 2011).

Knee orthoses is another consideration for the treatment of meniscus injuries. The American Journal of Sports Medicine published a systematic review on the treatment of surgical intervention compared to non-operative management as the first line of treatment for patients with knee pain and degenerative meniscal tears (Monk, Garfjeld Roberts, Palmer, Bayliss, Mafi, Beard, Hopewell, & Price, 2017). The results found there was no difference between arthroscopic meniscal debridement compared with non-operative management as a first strategy for patients with knee pain and degenerative meniscal tears (Monk et al., 2017). Evidence was found to indicate that patients with resistant mechanical symptoms who initially fail non-operative management may benefit from meniscal debridement (Monk et al., 2017). Research is fairly limited in relation to the effect of knee orthoses on meniscal injuries but there are more studies being initiated in this area.

There is a significant amount of literature that supports the impact of knee orthoses on the symptoms of osteoarthritis and one could stretch that as the mechanics of the knee joint and orthoses interact the same, that the treatment could produce similar results. The mechanisms by which valgus bracing alter the knee joint biomechanics are listed as improvements in malalignment, reduction in knee adduction moment, increase joint stability, decrease muscle co-contraction and improve proprioception (Moyer, Birmingham, Bryant, Giffin, Marriot, & Leitch, 2015). Canadian Certified Pedorthists are encouraged to operate in their scope of practice. If training and experience is not inclusive of knee orthoses, a referral to a qualified facility can provide the patient with further investigation to knee orthoses.

**Therapy**

Physical therapy management after a partial meniscectomy can be broken down into four phases. The first phase is acute injury management with pain free basic range of motion exercises. The second phase moves on to continued range of motion as tolerated, balance activities, strength training exercises, and upper body or low-impact cardiovascular exercise avoiding full knee extension. Phase three is the same as the second, however range of motion, strength training and cardiovascular fitness are more advanced and challenging. The fourth phase adds functional activities to those listed in phase three (Anderson et al., 2005).

**Key Considerations**

Meniscal injuries occur most often in men between the ages of 21 and 40. Peak incidences for females border on either side of the male statistics at 11–20 years of age and again between the ages of 61 and 70. Degeneration of the meniscus occurs from constant wear and tear grinding which comes with age and can be accentuated by activities requiring large weight-bearing forces (Anderson et al., 2005). These chronic injuries are often complex tears which can be aggravated by very minor stresses to the knee,
something as simple as changing directions can elicit pain (Hall & Brody, 2005). Acute traumatic tears, the more common method of injury, typically occur between the ages of 13 and 40 in people who are physically active or involved in accidental incidences such as a fall or motor vehicle accident (Hall & Brody, 2005).

References


Gluteal Tendinopathy

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Key Messages

• Corrective moulded foot orthoses are recommended for management of gluteal tendinopathy to reduce hip abduction and internal rotation that often place increased strain on gluteus medius/minimus tendons.

• Clinicians should be aware of other conditions that can manifest as lateral hip pain, including greater trochanteric bursitis and hip osteoarthritis, as these have similar presentation but different treatment strategy.

• Functional testing that stresses hip abductor muscle groups is recommended to improve diagnostic accuracy of gluteal tendinopathy.

Keywords

gluteus medius, gluteus minimus, hip abduction, hip pain, tendinopathy

Introduction

Gluteal tendinopathy is a lower limb tendinopathy that predominantly affects women aged 40–60. It is characterized by pain laterally around the greater trochanter with localized discomfort upon palpation (Mellar, Grimaldi, Wajswelner, Hodges, Abbott, Bennell, & Vincenzino, 2016). People with gluteal tendinopathy typically complain of lateral hip pain with walking or ascending stairs, and extreme discomfort with side sleeping. This can not only affect physical activity, but also activities of daily living. With gluteal tendinopathy structural abnormality in the tendon may be seen via imaging (Lequesne, Mathieu, Vuillemin-Bodaghi, Bard, & Dijan, 2008).

Gluteal tendinopathy can affect both the active and sedentary population. However, it is postulated that with increased female participation in long distance running and triathlons, the prevalence of gluteal tendinopathy will also increase within these populations (Mellar et al., 2016).

Pathomechanics

Gluteal tendinopathy involves tendinopathic change of the gluteus medius and/or gluteus minimus tendons (Allison, Vincenzino, Wrigley, Grimaldi, Hodges, & Bennell, 2016), two of the main hip abductors. The hip abductors are necessary to stabilize the pelvis during gait. The gluteus medius originates on the ilium inferior to the iliac crest, and inserts on the lateral aspect of greater trochanter of the femur. The gluteus minimus originates on the ilium between the anterior and inferior gluteal lines, and inserts on the anterior surface of the greater trochanter of the femur. The gluteus medius lies deep to the gluteus maximus, while the gluteus minimus lies deep to the gluteus medius. Together, the gluteus medius and gluteus minimus muscles provide an estimated 70% of the hip abductor force needed to preserve pelvic alignment during single-leg stance (Allison et al., 2016).

Excessive hip adduction is a key factor in the development of gluteal tendinopathy. Weak hip abductor muscles have been noted on both symptomatic and asymptomatic sides (Allison et al., 2016). There is greater contralateral trunk lean and pelvic drop during first and second (respectively) peak hip adduction moments of gait. Individuals with gluteal tendinopathy demonstrate a greater hip adduction moment throughout the stance phase of walking than healthy controls (Allison et al., 2016). Greater contralateral pelvic drop may increase gluteal tendon tension load as the muscle–tensile units lengthen to the point where their length–tension relationship relies more on passive tensile loading than active cross–bridge overlap; and/or result in compression of the gluteal tendons against the greater trochanter. Tendon loads can be particularly elevated when the muscle is not only activated, but the tendon is also concurrently lengthening. Therefore, it is understood that poor gait patterns of pelvic drop and hip adduction increase the tension load on the gluteal tendons of the weight-bearing
limb which is the likely culprit of the development or persistence of gluteal tendinopathy.

Another contributing factor is bone morphology. Studies by Birnbaum et al. found that a lower femoral neck shaft angle is present in patients with gluteal tendinopathy compared to patients with hip osteoarthritis, or pain free controls. A typical femoral neck angle is 128° with studies showing increased compressive force noted at 115°. Viradia et al. further demonstrated that the offset between the greater trochanters and the width of the iliac wings is greater in individuals with lateral hip pain (mean 28mm difference) compared to a pain-free control population (mean 17mm difference). These morphological considerations are important when noting that gluteal tendinopathy is more prevalent in women (23.5%) than in men (8.5%), as women are shown to have a greater Q angle at the hip.

**Differential Diagnoses**

**Hip Osteoarthritis** pain often characterized by insidious onset of groin or buttock pain. Pain is worsened by activity and can sometimes come in conjunction with decreased hip range of motion (Murphy, Eyles, & Hunter, 2016). Interestingly, patients with gluteal tendinopathy do not have trouble putting on shoes and socks, where patients with hip osteoarthritis will (Mellor et al., 2016).

**Trochanteric Bursitis** characterized by chronic lateral hip pain worsened by active abduction, passive adduction and direct palpation (Lustenberger, Ng, Best, & Ellis, 2011). Noted inflammation/collection of fluid of the trochanteric bursa; located beneath the gluteus maximus muscle and the iliotibial tract; covering the posterior and lateral facets of the greater trochanter as well as the gluteus medius tendon (Lequesne et al., 2008).

**Femoral stress fracture** pain in the groin/anterior hip. Active & passive range of motion may be painful and limited, with weakness and pain in the hip musculature (Konetsky & Miller, 2013).

**Testing**

**30-second single-leg stance** Request your patient stand on the affected leg for 30 seconds. Any ipsilateral trunk deviation must be prevented as this can delay or prevent onset of pain. As the examiner, you may gently hold your patient’s hands for balance. The gluteus medius is the primary pelvic stabilizer during single-leg stance and prevents downward tilt of the contralateral pelvis. During gait, it contracts with every step. Pain occurring with steady single-leg stance is indicative of gluteal tendinopathy.

**Resisted external derotation** Request your patient lie supine on the plinth with the affected hip and knee flexed at 90°. Additionally, the hip should be held in external rotation. This external rotation is typically painful for patients with trochanteric tendinobursitis. Slowly reduce the amount of external rotation until you are just able to relieve the pain (if at all). Ask your patient to then actively return the leg to neutral along the axis of the plinth. If this action replicates spontaneous pain, the test is considered positive. If a negative result is achieved, the test should be repeated with the patient lying prone with the hip extended and the knee flexed at 90° (Lequesne et al., 2008).

**Resisted Abduction** Request your patient lie on their unaffected side. With the affected leg, ask them to perform active abduction up to 25°, with the thigh extended. Pain is then assessed during resisted abduction with the thigh successively extended, then flexed at 45° and then flexed at 90°.

**Trendelenburg** ask your patient to stand on one leg. If the pelvis drops on the non-weightbearing leg, the Trendelenburg sign is positive. The pelvic drop indicates weakness in the hip abductors of the standing leg.

**Patient complaints**

Lateral hip pain at greater trochanter when walking, ascending stairs, or other activities that require single-leg loading. Pain upon palpation primarily at the greater trochanter. Pain may also radiate down the lateral thigh or around the greater trochanter itself. Patients may also complain of notable discomfort when lying on the side, as when sleeping. Onset of pain is gradual, with worsening occurring over time, and in active populations, sometimes with increased training load.

**Treatment**

As one of the primary mechanisms of injury is tension loads on the tendons, secondary to adduction of the hip, it is imperative to reduce hip adduction moment during gait, as well as reduce tension in the tendon sheath.

- Custom orthoses can assist in reducing medial ankle shift and subsequent internal leg rotation during stance and gait. This reduced adduction of the leg can decrease tension on the gluteal tendons. Custom
orthoses may also help attenuate shock more appropriately at foot level, therefore reducing shock translation up the kinetic chain. This may decrease secondary discomfort from ground reaction forces.

- Physiotherapy should be recommended to strengthen weak hip abductor muscles; primarily the gluteus medius and gluteus minimus. Furthermore, Murphy et al. (2016) note that while there is a lack of research in the area, the deep stabilizing muscles of the hip likely contribute to shock absorption and joint protection. Exercise and load modification is noted by Allison et al. (2016).

**Considerations**

- Sensitivity over the lateral trochanter is most often present but is not specific. It can be noted in patients with gluteal tendinopathy, hip osteoarthritis, trochanteric bursitis or a femoral neck stress fracture. It can therefore make it challenging to use as a diagnostic tool.

- Recent research by Pohl, Kendall, Patel, Preston Wiley, Emery, & Ferber (2015) at the University of Calgary has shown preliminary evidence that reduction in hip abductor strength without a concurrent pain response, may not result in a compensatory trunk lean. In fact, their results showed that maximal isometric hip–abductor strength was not related to external hip adduction moment during gait. Further study is needed to validate the reliability of a Trendelenburg test.

- Dynamic activities that cause increased unilateral hip adduction, such as running around a track in the same direction, or running on a cambered surface in the same direction, or with a narrow midline strike pattern should be considered as this increases the risk for development of lateral hip pain. (Mellor et al., 2016)

**References**


Piriformis Syndrome

Elisa Harrison, HBSc (Kin), PCP, C. Ped (C)

Key Messages

• Hip pain can be related to a number of pathologies including arthritis, avascular necrosis of the femoral head, stress or occult fracture, vascular insufficiencies, metastasis, various soft tissue diseases, and repetitive strains and trauma that affect nerve, muscle, bursa, and connective tissue. In some cases, hip pain is referred pain from adjacent structures such as the spine, as the hips act as the bridge and conduit between the trunk and lower extremities.

• Piriformis syndrome may mimic or be comorbid with other disorders and or conditions such as intervertebral disc disease or herniation, interspinal nerve impingement, lumbosacral radiculopathies, trochanteric bursitis, myofascial pain, compression fracture, and spinal stenosis.

• A full history and physical assessment, electromyography (EMG), magnetic resonance imaging (MRI), and computed tomography (CT) may be beneficial in differentiating piriformis syndrome.

Keywords

Beatty test, deep gluteal syndrome, Freiberg’s sign, ischemia, peripheral neuritis, Pace’s sign, Q angle, retrotrochanteric pain, sacroiliac joint dysfunction, sciatic nerve pain, wallet neuritis

Introduction

Piriformis syndrome is a peripheral neuritis (entrapment neuropathy) of the sciatic nerve caused by the adjacent piriformis muscle compressing or irritating the sciatic nerve. It is characterized by posterior hip and buttock pain similar to that of sciatica but not originating from spinal roots. Although epidemiological figures of the prevalence are unknown, they are estimated to be about 12.2% to 27% (Knudsen, Mei-Dan, Brick, & Matthew, 2016). Piriformis syndrome occurs most frequently during the fourth and fifth decades of life and affects individuals of all occupations and activity levels (Boyajian-O’Neill, McClain, Coleman, & Thomas, 2008). Piriformis syndrome is more common in women than in men possibly because of the biomechanics related to the quadriceps femoris muscle angle or Q angle (Dey, Das, & Bhattacharyya, 2013).

Pathology

The piriformis muscle acts as an external rotator of the femur when the hip is in extension, and as an abductor of the femur when the hip is flexed. Its main function is to provide postural stability (stabilization of the hip) during ambulation and static stance. Piriformis syndrome occurs as a direct relationship between the sciatic nerve and the piriformis muscle. In 96% of the population, the sciatic nerve exits the greater sciatic foramen deep along the inferior surface of the piriformis muscle. In 22% of the population, the sciatic nerve pierces or bisects the piriformis muscle, or the sciatic nerve might split into two branches or both. It is this population that is most predisposed to piriformis syndrome (Boyajian-O’Neill et al., 2008).

There are two types of piriformis syndrome:

1. Primary piriformis syndrome occurs due to anatomical problems such as a split in the piriformis muscle, a split in the sciatic nerve, or anomalies in the sciatic nerve path (as described above). These abnormalities cause the piriformis muscle to irritate or entrap the sciatic nerve such that with increased tension in the piriformis muscle, pain is exacerbated. This can be accentuated with overuse. Other contributing factors could include cerebral palsy, myosotis ossification, and pseudoaneurysms of the inferior gluteal artery adjacent to the piriformis muscle.

2. Secondary piriformis syndrome is a result of any precipitating cause, including trauma, repetitive use causing hypertrophy of the piriformis muscle, and/ or ischemia. In many cases, secondary
Piriformis Syndrome

Piriformis syndrome is the result of blunt trauma to the buttock (or heavy fall) causing inflammation of soft tissue, muscle spasm, or both leading to compression and irritation of the sciatic nerve. Trauma caused by repetitive activities such as competitive cycling, prolonged sitting, running, and/or walking could result in an overuse injury of the piriformis muscle leading to compression to the sciatic nerve. Compression of the nerve may also be a direct result of sitting for long periods of time on a hard surface. The latter is an example of what is referred to as “wallet neuritis.”

Symptoms
- Pain with prolonged sitting or standing
- Pain and or paresthesia radiating from sacrum through gluteal area down posterior thigh to knee
- Pain may improve with movement and worsen with rest
- Increased pain with rising from seated position or with deep squatting
- Increased pain with sitting on a wallet (“wallet neuritis”)
- Change in position does not relieve pain completely
- Difficulty walking, antalgic gait, or foot drop
- Contralateral sacroiliac pain
- Numbness in lower extremity
- Weakness in ipsilateral lower extremity
- Pain with bowel movements
- Retrotrochanteric tenderness

Differential Diagnosis
The following are other conditions/issues that may present similar clinical features to piriformis syndrome. The most common is sciatica.

- Sciatica: Symptoms of sciatica can be very similar to that of piriformis syndrome since they are both causing compression of the sciatic nerve. However, the origin of this pain is different. Sciatica originates from compression or irritation of the sciatic nerve in the lumbar spine from conditions such as disc herniation or spinal degeneration, whereas piriformis syndrome originates in the buttock area where the piriformis muscle is located.
- Vertebral disc disease or herniation
- Inter spinal nerve impingement
- Lumbosacral radiculopathies
- Trochanteric bursitis
- Myofascial pain
- Compression fracture and spinal stenosis

Common Testing

1. **Pace’s sign**: is determined with the FAIR test of flexion, adduction, and internal rotation of the hip (also known as the piriformis test). The patient is side lying, affected side on top, with hip and knee flexed at 60 degrees. The examiner, while stabilizing the hip, internally rotates and adducts the hip by applying downward pressure to the knee. A positive test is indicated by the presence of pain. Another version of this test can be performed in sitting or supine position. With the knee and hip flexed and the hip medially rotated, the patient resists the examiner’s pressure to the knee and attempts to externally rotate and abduct the hip. This test is positive when sciatic nerve pain is reproduced and weakness is displayed.

2. **Freiberg’s sign**: while the patient is supine the examiner will passively internally rotate and extend the hip. Pain is reported in the lower back and or buttock as the piriformis stretches. Pain might be accentuated by the examiner continuing internal rotation while increasing flexion of the hip. This is a positive sign for compression of the sciatic nerve often associated with piriformis syndrome.

3. **Beatty test**: the patient lies with the painful side up, the bottom leg straight and the painful leg flexed at the hip and knee, with the knee resting on the table. Buttock pain is produced when the patient lifts and holds the knee several inches off the table. This test relies on contraction of the muscle, rather than stretching which reproduces one of the proposed mechanisms for piriformis syndrome.

4. **Functional vs. structural leg length discrepancy (LLD)**: It is important when assessing piriformis syndrome to identify any structural and functional leg length differences. During gait analysis and static stance, one might observe a shortening of the involved lower extremity. In this case, it is important to do the Galeazzi test (also known as Allis sign or the skyline test) to determine if the LLD is indeed structural or if it is functional due to differing amounts of pronation or supination in the foot with weight-bearing.

4. **Palpable point tenderness**: Tenderness in area of sacroiliac joint, greater sciatic notch, and piriformis muscle.
5. **Gait**: Piriformis syndrome may be exacerbated by simple movement including walking and or stooping. Patient may show signs of an antalgic gait as they attempt to reduce symptoms and shorten midstance on the affected side. They might show signs of neuromuscular weakness including a high steppage gait or, in severe cases, drop foot or slap on the affected side. LLD might also be observed as the patient falls to the shorter limb in strike phase or circumducts the longer limb in swing phase.

**Signs**
- Asymmetrical weakness in affected limb
- Positive Pace’s sign
- Positive Freiberg’s sign
- Positive Beatty test
- Limited medial rotation of the ipsilateral lower extremity
- Ipsilateral short leg
- Persistent sacral rotation toward the contralateral side with compensatory lumbar rotation
- High steppage gait, inability to stand on heels or toes
- Sensory alteration in entire foot except medial longitudinal arch, and medial malleoli

**Contraindications**
Michaud (1993) suggests “when a structural LLD is present, it is recommended to treat with a balance full shoe lift vs. a partial heel lift to prevent contracture of the calf muscles and prevent contralateral risk of injury to hip flexors and adductors” (Michaud, 1993).

**The Patient Perspective**
Patients report pain in the sacroiliac joint, greater sciatic notch and the piriformis muscle, extending down the leg and causing difficulty walking. Patients might report acerbating pain and symptoms with stooping or lifting.

**Common Treatment**
Acute treatment could include the prescription of anti-inflammatory medication, analgesics, and muscle relaxants by a qualified physician to reduce local inflammation, pain, and spasm. Piriformis stretching exercises with heat therapy might be sufficient to relieve the pain. In addition, strengthening of the gluteus maximus and gluteus medius to decrease excessive adduction and internal rotation of the hip can help to decrease the high eccentric loads being transferred to the piriformis muscle during gait (Michaud, 2011). Other relatively conservative treatments include therapeutic ultrasound, massage, foam roller, core strengthening, hip mobilization, lumbar and sacroiliac manipulation, rectal diathermy, and transcutaneous electrical stimulation. Piriformis syndrome is often successfully treated by physical therapists, massage therapists, chiropractors, and osteopathic manual practitioners. Studies show that 79% of physiotherapy patients have a 50% improvement in their symptoms (Knudsen et al., 2016).

Pedorthic treatment includes correction of any abnormal biomechanics caused by posture, pelvic obliquity, LLD, or ankle/foot pathomechanics and anomalies including internal and external modification to footwear such as lifts and custom foot orthoses.

If a structural LLD is present by itself, treatment consists of a balance lift under the short limb. The ideal lift will level the iliac crest and bring the lumbar spine to vertical. Structural LLD less than 1 cm does not require treatment unless the LLD is contributing to the pathology.

In a report by Shah (2017), the presence of a Morton’s foot was suggested as a contributing factor. The long second metatarsal bone is thought to cause instability during terminal stance and push-off, resulting in a reactive contraction of the external hip rotators, namely the piriformis muscle, in order to stabilize. In this case, a full-contact foot orthosis can help to stabilize the metatarsals, decrease the excessive pronation and internal rotation of the lower limb, thereby relieving the piriformis syndrome.

In a study by Klein (2001) the following was found: “a Morton’s foot may predispose the patient to developing piriformis syndrome. The prominent second metatarsal head destabilizes the foot during the push–off phase of the gait cycle, causing excessive foot pronation and internal rotation of the lower limb. The piriformis muscle (external hip rotator) reactive contracts repetitively during each push–off phase of the gait cycle as a compensatory mechanism, leading to piriformis syndrome.” In this case, a full-contact foot orthosis can help to stabilize the metatarsals, decrease the excessive pronation and internal rotation of the lower limb, thereby relieving the piriformis syndrome.
Foot types that are hypermobile or excessively pronate require functional control with foot orthoses. Michaud (1993) summarized that “asymmetrical pronation of the foot forces the entire lower extremity to excessively internally rotate and drop inferiorly, the posterior superior iliac spine moves anterior–superior. This pathomechanic eventually produces ligamentous instability and chronic sacroiliac joint dysfunction and may entrap the sciatic nerve between the piriformis muscle leading to piriformis syndrome.”

When hip pain is refractory, consider referral to specialized medical professionals such as a physiatrist, rheumatologist, physical therapist, massage therapist, chiropractor, osteopathic manual practitioner, and/or orthopaedic specialist.

In some cases, surgical interventions such as a release of the piriformis muscle with exploration of the sciatic nerve is required when conservative treatments have failed.

Key Considerations

- Piriformis syndrome is more common in women than in men.

- Piriformis syndrome might mimic or be comorbid with other disorders and/or conditions such as sciatica, intervertebral disc disease or herniation, interspinal nerve impingement, lumbosacral radiculopathies, trochanteric bursitis, myofascial pain, compression fracture, and spinal stenosis.

- When a structural LLD is present, it is recommended to treat with a balanced full shoe lift vs. a partial heel lift.

References


Key Messages

- Partial foot amputation is the most common form of amputation and is usually the result of diabetic neuropathy.
- Amputation of lesser toes has little impact on weight-bearing or gait, and can be treated with custom orthoses, toe fillers, and orthopaedic shoes.
- Hallux amputation impairs balance and limits push-off.
- Once the metatarsal heads are removed, gait is significantly impacted and referral to an orthotist/prosthetist may be necessary to produce an above ankle offloading device that is capable of restoring effective foot length.

Introduction

The goal of amputation is to remove non-viable tissue and maintain as much function as possible (Funk & Young, 2001). Trauma, infection, tumours, and peripheral vascular disease are common causes of limb amputations (Yusof, Sulaiman, & Muslim, 2007). There are 110,000 lower limb amputations performed in the United States every year (Karam, Wiley, & Shurr, 2010). Most lower extremity amputations are due to peripheral arterial occlusive disease, soft tissue sepsis, and neuropathy (Moxey et al., 2011). The incidence of lower limb amputations due to peripheral vascular disease ranges from 20 to 35 per 100,000 people (Johannesson et al., 2009).

Lower extremity amputations can take place at many levels, including: transfemoral (above knee), through knee, transtibial (below knee), Syme (ankle), midtarsal (Chopart), tarsometatarsal (Lisfranc), transmetatarsal, and toe/ray (Bowker, 2008). Amputations due to foot injury or infection used to be done at the below knee level because of complications following partial foot amputations (Sobel, Japour, Giorgini, Levitz, & Richardson, 2001). Prior to the 1950s, partial foot amputations were generally only done as a result of traumatic injuries (Bowker, 2007). In the United States today, there are about twice as many partial foot amputations as transfemoral or transtibial (Janisse & Janisse, 2010). Partial foot amputations are a viable alternative now, thanks to improved surgery and prosthetics (Sobel et al., 2001). Partial foot amputations offer the benefits of a weight-bearing residuum (Mueller & Straube, 1997), maintaining proprioception, and the least change in body image (Bowker, 2008).

Diabetes is the primary cause of non-traumatic lower limb amputations in developed countries (Meltzer et al., 2002). The prevalence of diabetes in the United States is approximately 7.3% of adults (Imam, Elsawy, & Balb, 2007). In 2005 there were 1.8 million Canadian adults with diagnosed diabetes (Canadian Diabetes Association, 2008). People with diabetes are 15 times more likely to have a lower extremity amputation (Kanade, Van Deursen, Price, & Harding, 2006). In Canada in 2006, over 4,000 people with diabetes had an amputation (Canadian Association of Wound Care, 2011).
Pathology

Diabetes can lead to neuropathy, which is an impairment of the peripheral nerves (Tanenberg & Donofrio, 2008). Over time, ulcers can develop on the plantar surface of the foot at high pressure areas (Armstrong et al., 1999). The ulcer provides an entry for bacteria, which can lead to infection and amputation (Armstrong, Hadi, Nguyen, & Harkless, 1999). Approximately 85% of patients have an ulcer prior to limb amputation (Lazaro-Martinez et al., 2011).

Amputations due to trauma mainly affect young males (Hebert & Ashworth, 2006). Trauma causes 16% of amputations, but because it affects a relatively young population, 45% of people living with amputations suffered traumatic injury (Ziegler-Graham, MacKenzie, Ephraim, Travison, & Brookmeyer, 2008). Foot amputation due to trauma is more common during a war, thanks to landmines, bullets, and frostbite (Soderberg, Wykman, Schaarschuch, & Persson, 2001).

Partial foot amputation (PFA) refers to the removal of part of the rearfoot or forefoot (Dillon & Barker, 2008). PFAs are usually the result of infection and tissue necrosis in diabetic patients (Bowker, 2007). PFA is the most common form of amputation surgery, affecting two in 1000 people (Dillon, 2010). It is believed that PFA leads to increased plantar pressure because of the decrease in surface area (Imam et al., 2007). PFA has been found to increase midfoot pressure of the affected foot and heel pressure of the opposite foot (Kanade, Van Deursen, Price, & Harding, 2008). Generally, the level of amputation is selected based on the most distal area of salvageable tissue (Baima, Trovato, Hopkins, & delLateur, 2008). Several levels of amputation are discussed below.

Transfemoral

Ambulation following transfemoral amputation requires 65% more energy (Bowker, 2008). There is a short residual limb and therefore a heavier prosthesis is required than with more distal amputations (Smith, 2006). Because of the extreme energy cost, only 25% of transfemoral amputees become functionally mobile with their prosthesis (Bowker, 2008).

Transtibial

If the foot is not salvageable, then amputation may be performed at the tibia (Bowker, 2008). Following transtibial amputation, the patient must learn to walk without plantarflexors, which usually generate 80% of the power required for ambulation (Soares, Yamaguti, Mochizuki, Amadio, & Serrao, 2009). The lack of plantarflexors is compensated for with increased hip extensor moments (Mueller, Salsich, & Bastian, 1998). At this level of amputation, there is a two–year mortality rate of 36% (Pinzur, Stuck, Sage, Hunt, & Rabinovich, 2003). Early prosthetic fitting and maintaining as much tibial length as possible lead to more positive outcomes (Bowker, 2008). Half of below knee amputees will develop a wound in the contralateral limb within two years (Sobel et al., 2001). There is a greater risk of developing osteoarthritis in the contralateral lower limb joints (Karam et al., 2010). Kanade et al. (2006) stress the importance of protecting the remaining leg. Pedorthic treatment could include shoe fitting and a foot orthoses for the remaining foot. Care must be taken to avoid creating a limb length discrepancy when dispensing a unilateral foot orthosis.

Syme

Syme refers to an amputation at the ankle joint with the medial and lateral malleoli removed (Pinzur et al., 2003). The heel pad is used on the stump to facilitate weight–bearing (Bowker, 2008). Compared with a more proximal amputation, there is a simpler rehabilitation and patients have greater ambulatory independence (Pinzur et al., 2003). The Syme amputation requires a firmly fitted prosthesis to maintain the heel pad beneath the tibia (Bowker, 2008).
Chopart

Chopart refers to an amputation at the calcaneocuboid and talonavicular joints (Bowker, 2008). With only the talus and calcaneus remaining, patients are able to walk short distances barefoot (Bowker, 2008). Chopart amputation can be selected for children, because of the risk of metatarsals growing and requiring further surgeries (Baima et al., 2008). This amputation is rarely used for diabetic patients (Bowker, 2008); it is usually performed due to injury or tumors (Burger et al., 2009). There is difficulty fitting a prosthesis for a Chopart amputation. The limb must be stabilized in the prosthesis, otherwise it will piston up and down and risk ulceration (Sobel et al., 2001).

Transmetatarsal

Transmetatarsal refers to an amputation through the shafts of metatarsal bones (Dillon, 2010). Problems with this surgery include equinus contractures and callosities at the distal end of the remaining foot (Sobel et al., 2001). Equinus contractures can develop due to the muscle imbalance between intact gastrocnemius/soleus and severed dorsiflexors (Tang et al., 2004). Patients are unable to progress their centre of pressure beyond the distal end of the stump, until the contralateral heel touches the ground (Dillon & Barker, 2008). Active push-off is not possible when the metatarsal heads have been removed (Michael, 2003). The lack of toe-off is compensated by pulling the leg forward with the hip flexors (Mueller et al., 1998). There is high pressure at the distal stump at the end of the stance phase of the gait cycle (Sobel et al., 2001). Peak forefoot pressures are 30% higher following partial foot amputation (Armstrong et al., 1999). Using an AFO can spread the pressure over a larger surface area (Sobel et al., 2001). To restore effective foot length, an above ankle device is required that includes: an anterior shell, stiff forefoot to accept body weight, and a locked ankle or dorsiflexion stop (Dillon & Barker, 2008).

Longitudinal

Longitudinal refers to an amputation along the length of the foot, such as ray resections (Dillon, 2010). This results in a narrow foot, placing excess pressure on the remaining metatarsals and leading to forefoot calluses and ulcers (Sobel et al., 2001). A narrow foot also makes shoe fitting a challenge. Longitudinal amputations allow the retention of the lever arm, enabling normal propulsion and gait (Sobel et al., 2001). Amputation of the first ray can result in prominent metatarsal heads and clawing of the remaining toes (Paola et al., 2003).

Phalangeal

Amputation of the hallux causes much greater impairment than removal of the lesser digits (Sobel et al., 2001). The first toe is important for balance and push off during gait (Atnip, 2005). Amputation of the great toe leads to increased peak pressures at the metatarsal heads and lesser toes (Funk & Young, 2001). Ideally, surgeons remove the hallux at the base of the proximal metatarsal, if possible (Atnip, 2005). If an amputation is required at the first metatarsophalangeal joint, then the windlass mechanism is lost (Bowker, 2008). Biomechanical changes can result in increased pressure and risk of ulcer formation on the opposite foot (Paola et al., 2003).

Amputation of the lesser toes does not greatly alter gait (Funk & Young, 2001). However, pressure imbalances are created that can lead to callosities at the metatarsal heads and risk of ulceration (Sobel et al., 2001).

Common Treatment

Shoe fitting is an important pedorthic consideration for all levels of amputation. Given the fact that lower limb amputation is associated with diabetic neuropathy and the risk posed to the contralateral limb, extra deep and wide orthopaedic footwear can help to protect the sound foot and/or residuum. The average time following a partial foot amputation for a second amputation is two years (Meier & Kennedy, 2009). Janisse & Janisse (2010) indicate that the main treatment goals for partial foot amputees are to “restore stability and function lost due to an amputation, facilitate energy-efficient gait, maintain support, and prevent any further complications.”

Treatment for partial foot amputation may include: immobilizing devices such as AFOs, sockets with a filler prostheses, carbon fiber footplates incorporated in an orthosis or in the shoe (Meier & Kennedy, 2009). For toe amputations, a custom foot orthosis with toe filler can be used. The filler prevents the forefoot of the shoe from collapsing. When placing the forefoot filler on the orthosis, extra room must be given to allow for the residual foot sliding forward and socks bunching up. The plantar surface of the orthosis may be stiffened to prevent breakdown of the shoe at the flex point, though this will not serve to restore effective foot length. Patients with midfoot amputations are candidates for an AFO with toe filler, because the distal stump is susceptible to breakdown and an above ankle device is conducive to transferring forces to a larger surface area. Patients
with more proximal amputations tend to prefer high top footwear, as it secures the foot in the shoe (Sobel et al., 2001).

Shoe modifications can improve comfort and gait. For the partial foot amputee, a rocker sole with the apex proximal to the distal residuum and sole stiffening are beneficial (Janisse & Janisse, 2010). If the orthotic/prosthetic device does not adequately control medial/lateral movement, then a sole flare may be added to the shoe (Soderberg, 2001).

**Prevention**

Patients with diabetic neuropathy need the services of a multidisciplinary health care team to minimize complications (Dupre, Deschamps, Pillu, & Despeyroux, 2003). The services of a wound care team have been found to lower amputation rates among high risk individuals (Meltzer et al., 2002). Prevention and treatment of ulcers is the approach used by programs aiming to prevent amputations (Armstrong et al., 1999). A neuropathic foot must be protected, and the correct shoes play an important role in this goal (Dahmen & Haspels, 2001). Orthopaedic shoes can protect the diabetic foot from injury by reducing plantar pressures during ambulation (Mueller, 1997). Paola et al. (2003) found lower than expected re-ulceration rates in patients who had a first ray amputation. The patients in their study had extensive follow-up appointments and were fitted with accommodative foot orthoses and rocker sole orthopaedic shoes.

**Key Considerations**

Factors such as a patient’s age, health, activity level, and financial means must be taken into account when deciding upon the best treatment approach. Although an AFO might offer the greatest offloading for a midfoot amputee, the patient may prefer a foot orthoses for cosmetic or financial reasons. Generally, the more active the individual is, the more “biomechanically sophisticated” the device must be (Michael, 2003). The rate of PFA increases significantly after age 40 and peaks at the 75 to 79 age range (Dillon, Fatone, & Hodge, 2007).

**References**


# Leg Length Discrepancy

**Colin Dombroski, PhD, C. Ped (C)**

## Key Messages
- Identify the discrepancy (Allis test, gait abnormalities, asymmetry in structural findings, i.e., significant differences in pronation).
- Classify the discrepancy (structural/functional, scoliosis, congenital/acquired, and when).
- Measure (scanogram or the TMM).
- Decide to lift and the total amount (congenital = 60% to start, recently acquired = 80% with caution that things may change).
- Decide how to lift (internal or external to shoe).
- Follow up on lift for progress and further lift based on tolerance and improvement or change in measurement due to arthroplasty.

## Keywords
acquired, Allis test, anisomelia, asymmetry, compensation, congenital, functional LLD, osseous malformation, structural LLD

## Introduction and Pathology

Limb length discrepancy, or anisomelia, is a condition defined as a paired set of limbs that are unequal (Gurney, 2002). When this unequal pairing of limbs occurs in the lower extremity, anisomelia is known clinically as a leg length discrepancy. Leg length discrepancy can be further subdivided into two etiological categories: functional leg length discrepancy, and structural leg length discrepancy (Gurney, 2002).

Functional leg length discrepancy is described as a discrepancy caused by an asymmetry in soft tissue, giving the appearance of a discrepancy (Gurney, 2002). This asymmetry may be caused by muscle or joint tightness or muscle weaknesses that may affect joints in the lower kinetic chain (Hanada, Kirby, Mitchell, & Swuste, 2001). More common causes of functional leg length discrepancy are asymmetries in strength, flexibility, and asymmetrical subtalar joint pronation/supination that may cause an increase or decrease (respectively), in rotational torques in the affected limb (Gurney, 2002).

Structural leg length discrepancy is defined as a discrepancy due to an osseous malformation of the load bearing bones of the lower extremity, the femur and/ or the tibia (Gurney, 2002). Etiology of structural leg length discrepancy is attributed to, but not limited to congenital dislocation of the hip, fractures, avascular necrosis of the femoral head, infections, tumors, and surgical procedures such as a total hip arthroplasty.

These categories of leg length discrepancy can be further divided into “congenital” and “acquired” groups. It should be noted that acquired leg length discrepancy that develops later in an individual’s life (due to trauma or surgery) is thought to be the more debilitating of the two (Gurney, 2002).

Scoliosis based leg length discrepancy can in fact, cause a functional discrepancy wherein the spine is curved and fixed, causing a malalignment in the hips. Lifting for scoliosis is based on many factors and may include length of time one has had the scoliosis, severity of the deformity, function and pain, and the nature of the curve. See the chapter on scoliosis for descriptions of how to deal with this deformity, but understand that lifting therapy, if done incorrectly, may worsen the apparent leg length discrepancy, so caution is to be taken.

Leg length discrepancy is a condition shown to affect 25–70% of the general population (Gurney, 2002). Due to the prevalence of leg length discrepancy, clinicians commonly test for leg length discrepancy during standard musculoskeletal assessments. The
ubiquitous nature of leg length discrepancy can prove frustrating to many clinicians, particularly due to lack of consensus surrounding the amount of discrepancy that necessitates treatment.

Structural leg length discrepancy, and the alterations in biomechanical function associated with it, is thought to be a contributing factor to many clinical pathologies (Etnier & Landers, 1998; Friberg, 1983; Giles & Taylor, 1981; Gurney, 2002; Hanada et al., 2001; Kakushima, Miyamoto, & Shimizu, 2003; Walsh, Connolly, Jenkinson, & O’Brien, 2000). These pathologies include lower back pain, osteoarthritis of the hip, aseptic loosening of hip prostheses, lower limb stress fractures, knee pain, and poor running economy. Many authors have linked leg length discrepancies to these pathologies by way of the compensatory mechanisms developed by the patient (Friberg, 1983; Giles & Taylor, 1981; Kakushima et al., 2003; Kaufman, Miller, & Sutherland, 1996; Papaioannou, Stokes, & Kenwright, 1982).

The leg length discrepancy literature is typified by a general lack of agreement as to the point at which treatment is warranted. Blake & Ferguson (1992) reported that a leg length discrepancy of only 3mm can be clinically relevant to runners due to the increase in ground reactive forces upon heel strike, and Friberg (1983) reported that a 5mm leg length discrepancy is enough to be a contributing factor in the development of low back pain. White & Dougall (2002) reported, however, that a leg length discrepancy of up to 19mm was acceptable in patients who have recently undergone a THA.

LLD post joint replacement is a clinical reality, and one that is attracting greater attention in the literature. Post THA, the mean leg length discrepancy for hip arthroplasty varies in the literature from 1mm to 15.4mm. The mode leg length discrepancy post hip arthroplasty has been reported at 9.7mm (Clark, Huddleston et al. 2006)

In a 2017 study titled “Is a long leg a risk for hip or knee osteoarthritis,” researchers found that replacements surgeries due to osteoarthritis was performed three times more often on a longer, rather than a shorter, leg. In the aforementioned study the LLD breakdown was as follows: 40% 5-8mm, 15% greater than 10mm, and one participant with an LLD of 26mm. The researchers posited that increased focal loading of articular surface area in the hip may be associated with early osteoarthritic changes (Tallroth et al., 2017). Previous research has concluded that the prevalence and severity of osteoarthritis with leg length discrepancy is greater than those with equal length limbs (Murry et al., 2015).

The current cutoff for clinical significance (i.e., the point at which treatment is warranted) is a heated topic. Even though one may “reasonably assume neuromuscular control and foot loading patterns can be greatly affected by leg length discrepancy” (Perttunen, Anttila, Sodergard, Merikanto, & Komi, 2004), some authors have been bold enough to suggest that leg length discrepancy does not matter (White & Dougall, 2002), while others have concluded smaller leg length discrepancy (3mm), combined with the compounded ground reaction forces associated with running, may require treatment (Blake & Ferguson, 1992). At the end of the day, pain, function and disease progression in patients with
osteoarthritis are the clinical considerations when deciding when and how to lift.

**Clinical Presentation – Static Weight-bearing:**
- Windswept feet (longer leg pronated, shorter leg supinated)
- Unilateral knee flexion (longer leg)
- Unilateral knee hyperextension (short leg)
- Hip drop to the short side
- Shoulder drop to the short side
- Possible head tilt
- Asymmetrical foot pronation

**Clinical Presentation – Gait Analysis:**
- Decreased stance phase on shorter leg
- Increased stance phase on longer leg
- Possible Trendelenburg sign (hip drop) if hip musculature is weak
- Inability to fully extend long leg knee
- Pelvis drop the shorter side
- Possible circumduction of longer leg
- Greater out-toeing of longer leg
- Increase of stance width
- Asymmetrical foot pronation

**Differential Diagnosis**
The obvious causes of misdiagnoses of leg length discrepancy are improper classification of a discrepancy and either over- or underestimation of the magnitude. If a clinician is unfamiliar with physical measurements, imaging studies will prove more reliable.

It is possible for leg length discrepancy to be comorbid with many other clinical pathologies. A patient’s leg length may be one contributing factor to a larger, multifactorial puzzle.

**Common Testing**

**Measurement of Leg Length Discrepancy**

**Allis test**
The most commonly used leg length discrepancy test is the Allis test. The Allis test is a method in which the patient is positioned supine on a plinth with both knees flexed to 110 degrees and with both medial malleoli placed together. The heights of the tibial tubercles are used to compare the lengths of the tibiae, while the placement of the knees (in front or behind) is used to gauge femoral discrepancy. From this position, the patient is asked to raise the pelvis up off the plinth, and then to firmly place the pelvis down. The patient’s legs are then fully extended by the examiner. Visual landarking of the most inferior part of the medial malleoli is taken. The patient is then instructed to sit upright. The medial malleoli in a patient with a FLLD may equal out, whereas a patient with a SLLD will not. It is important that clinicians test for the factors that may influence functional discrepancy.

The Allis test, however, is only a qualitative measurement, and can therefore only alert the clinician to a potential difference in leg length. Quantitative measuring is described below.

**Medical imaging**

Scanogram is a common method utilized by clinicians (Beattie, 1990). A scanogram is an X-ray that captures the hip, knee, and ankle, non-weight-bearing, in three separate exposures. A radiographic ruler is placed in the midline of the patient’s body so that measurements may be taken right off the X-ray. The scanogram lessens the chance of magnification error, but does increase the cost of the procedure, as well as the patient’s exposure to radiation (Beattie, 1990). Also, determinations of leg length from a scanogram only account for the overall length of the femur and tibia, and may not take into account the differences in joint space that may be present.

Computerized tomography (CT) has also been utilized in the detection of leg length discrepancy (Tokarowski, 1995). CT has been shown to have a precision of less than 1mm with 66% less radiation.
exposure to the patient when compared to radiograph (Porat, 1989). Although CT is more reliable (and arguably safer) than X-ray, it is used less often due to the cost of the procedure, and the longer wait times that are typically seen for CT (Tokarowski, 1995).

Physical measurements

Some researchers argue that imaging techniques are costly, time consuming, and may expose the patient to unneeded radiation. Due to the aforementioned factors, different measurement protocols have been developed to measure leg length discrepancy.

The tape measure method is a method described often in the literature as an alternative way to measure structural leg length discrepancy (Beattie, 1990). This method involves taking a measurement from the individual’s anterior superior iliac spine to the ipsilateral medial malleolus, while the individual is supine on a plinth. The tape measure method is subject to errors due to differences in circumference between the lower extremities, and unilateral deviations along the long axis of the leg, such as genu valgum or varum. Furthermore, pelvic differences and difficulty land-marking bony prominences (such as the anterior superior iliac spine), especially in overweight individuals, might prove to be difficult, and contribute to error (Eichler, 1972).

To investigate the real impact of this error variation, Beattie et al. (1990) measured nineteen individuals (ten individuals with a leg length discrepancy and nine controls) from the anterior superior iliac spine to the medial malleoli, using the tape measure method, and then compared this measurement to mini-scanogram (non-weight-bearing radiograph). Beattie et al. (1990) reported intraclass correlation coefficients of 0.68 for both groups when only one measurement was taken. When the means of two measurements were compared however, this association increased to 0.79, suggesting that the tape measure method demonstrates acceptable concurrent validity. It is important to note, however, that 0.79 is not the gold standard, and if your patient has a long complicated history and you wish to undertake the best due diligence, scanogram is your best bet.

Contraindications

Lifting a patient with a functional leg length discrepancy may cause iatrogenic effects. Care should be taken when classifying discrepancies to properly identify the appropriate category.

Lifting a patient too soon after joint arthroplasty may lead to pain, if the lift is too high. Post THA, LLD followups should be conducted at 4-, 8-, and 12-month intervals to investigate changes in discrepancy. Care should also be taken not to lift too much, too soon after surgery.

Lifting a patient who has a fixed scoliosis may increase lower back pain. Please refer to the section on scoliosis for more information.

The Patient Perspective

Oftentimes, patients with a small congenitally acquired discrepancy are not aware that they have one. When a patient is aware, they might use descriptors such as:

• I need to have clothes tailored differently
• I feel my hip drop to one side
• I feel unbalanced when I walk
• Lower back pain
• My shoes are worn out unevenly
• I need to set up my bike differently to avoid pain (cyclist)
Common Treatment
Common treatments for functional leg length discrepancy are:

• Physiotherapy (to work out imbalances in strength and flexibility)
• Massage therapy (deep tissue release)
• Off-the-shelf or custom foot orthoses (to correct large asymmetrical foot pronation that may lead to functional discrepancy)

Common treatments for structural leg length discrepancy (including the above listed) are:

• Footwear lifts (internal and external)

Key Considerations
Amount of discrepancy to lift, use the following guidelines:

• Congenital – Begin with 60% of the total amount and increase based on objective outcomes (decrease in pain, increase in function, etc.).
• Acquired – Begin with 80% of the total and increase based on objective outcomes and follow up post surgery to assess change.

Age:

• Younger patients – Use caution in lifting as growth may alter the discrepancy. Close dialogue with the patient’s physician is encouraged.
• Older patients – Use caution and do not lift too much too soon as this may alter postural stability and increase muscle activity.

Type of lift: Internal

• Depending on the shoe, 10mm is typically the cut-off for internal heel lifting. If the shoe is extra deep, more may be added.
• Heel-only lifting may put the patient at risk for forefoot symptoms over time.

References


Sciatica

Kelly Robb, MSc, C. Ped (C)

Key Messages

- True clinical sciatica involves disc herniation and nerve-root compression. This condition is often confused with low back injuries causing referred peripheral neuropathy.
- The term “sciatica” describes symptomology, rather than a specific diagnosis.
- Pedorthic treatment can vary substantially from patient to patient. Treatment is dependent on the location of spinal impingement and the patient’s location of referred discomfort.

Keywords
disc herniation, lumbar radiculopathy, pain, piriformis syndrome, sciatic nerve injury, sciatica

Introduction

The term “sciatica” literally means “of the hip” (Marieb, Mallatt, & Wilhelm, 2004). The simplicity of this term is misleading, as there seems to be confusion around its definition between researchers, clinicians, and patients. Although approximately 70% of the adult population experiences low back pain once or more during their life, no specific pathology is identified in up to 85% of the patients (Devillé, van der Windt, Dzaferagi, Bezemer, & Bouter, 2000). The clinical definition of sciatica includes disc herniation and referred lower limb pain resulting from nerve-root compression. Therefore, inflammation and compression of the nerve root is clinically important in order to differentiate these terms. However, confusion occurs when patients and clinicians describe sciatica as “referred pain from the lower back and is neither related to disc herniation nor does it result from nerve-root compression” (Valat, Geneway, Marty, Rozenberg, & Koes, 2010).

Consequently, there have been numerous generalizations of findings across studies and conflicting prevalence estimates (values ranging from 1.6% to 43%) of patients suffering from this condition (Konstantinou & Dunn, 2008). Furthermore, “piriformis syndrome” is regularly under-diagnosed, and unclear as to its “distinct clinical entity [of] neuropathic pain from compression of the sciatic nerve as it courses through the piriformis muscle” (Kirschner, Foye, & Cole, 2009). Researchers and clinicians agree that the term sciatica describes symptomology, rather than a specific diagnosis. This statement is important to keep in mind during pedorthic clinical treatment.

Pathology

The sciatic nerve is the largest branch of the spine emerging from the sacral plexus, spinal nerves L4–S3. The sciatic nerve, composed of the tibial and common fibular nerves, is the thickest and longest nerve in the body. Innervation is supplied to the majority of the lower extremity, excluding the anterior and medial regions of the thigh. Leaving the pelvis region, this nerve passes through the greater sciatic notch, coursing deep to the gluteal maximus muscle as it enters the thigh. The sciatic nerve innervates the hamstrings as it passes deep to the musculature. The tibial and common fibular nerves diverge from the sciatic sheath just proximal to the knee.

The tibial nerve passes through the popliteal fossa, descends deep to the soleus muscle, and continues to descend posterior to the medial malleolus of the ankle. The tibial nerve branches into the medial and lateral plantar nerves. This nerve innervates almost all posterior lower limb musculature.
The common fibular nerve (also called the common peroneal nerve) descends the leg lateral to the popliteal fossa, and wraps itself around the head of the fibula. The nerve branches into superficial and deep branches, innervating the lateral and anterior compartments of the lower leg (Marieb et al., 2004).a

Table 1. Branches of the sacral plexus.

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<tr>
<th>Nerves</th>
<th>Ventral Rami</th>
<th>Structures Served</th>
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</thead>
<tbody>
<tr>
<td>Sciatic</td>
<td>L4, L5, S1–S3</td>
<td>Composed of two nerves (tibial and common fibular) in a common sheath that usually diverge just proximal to the knee.</td>
</tr>
<tr>
<td>Tibial (including medial and lateral plantar branches)</td>
<td>L4–S3</td>
<td>Motor branches: to muscles of back of thigh, leg, and foot (to hamstrings except short head of biceps femoris, to posterior part of adductor magnus, to triceps surae, fibialis posterior, popliteus, flexor digitorum longus, flexor hallucis longus, and intrinsic muscles of the foot). Cutaneous branches: to skin posterior surface of leg and sole of the foot.</td>
</tr>
<tr>
<td>Common fibular (superficial and deep branches)</td>
<td>L4–S2</td>
<td>Motor branches: to short head of biceps femoris of thigh, fibular muscles of lateral compartment of leg, fibialis anterior, and extensor muscles of toes (extensor hallucis longus, extensors digitorum longus and brevis). Cutaneous branches: to skin of anterior and lateral surface of leg and dorsum of foot.</td>
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Table 2. Subjective and objective findings based on location of spinal impingement

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<th>Nerve Impinged</th>
<th>Subjective Findings</th>
<th>Objective Findings</th>
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<tr>
<td>L4</td>
<td>Back pain down to knee and to medial ankle, with or without numbness or tingling, and difficulty with subtalar joint inversion.</td>
<td>Weakness in inversion of the foot, and diminished or absent deep tendon reflex at the knee.</td>
</tr>
<tr>
<td>L5</td>
<td>Sharp shooting pain with or without numbness or tingling in the lateral leg to dorsum of foot and hallux, and weakness/difficulty raising foot and toes.</td>
<td>Weakness in dorsiflexion of foot and toes (tibialis anterior and extensor hallucis longus) positive straight leg test.</td>
</tr>
<tr>
<td>S1</td>
<td>Sharp shooting pain with or without numbness or tingling in posterior compartment to lateral foot and fifth digit, and weakness/difficulty in plantarflexion and/or eversion.</td>
<td>Weakness in plantarflexion and eversion of foot and diminished or absent deep tendon reflex at the ankle.</td>
</tr>
</tbody>
</table>


The common fibular nerve (also called the common peroneal nerve) descends the leg lateral to the popliteal fossa, and wraps itself around the head of the fibula. The nerve branches into superficial and deep branches, innervating the lateral and anterior compartments of the lower leg (Marieb et al., 2004).a

The sciatic nerve can be implicated through a number of mechanisms but injury most commonly results from nerve-root compression. Structural irregularities within the intervertebral foramina may also result in sciatica (Prentice, 2006). Additional causes include “metabolic, toxic, or infectious disorders” (Decker & Albert, 2002) and “lumbar canal, foraminal stenosis and (less often) tumours or cysts” (Valat et al., 2010). A blunt force to the gluteal region or posterior thigh can sever the nerve. In such cases, due to the fibular nerve’s superficial location in the lower leg, this nerve may be susceptible to compression against the fibular neck.

True sciatica occurs when a herniated lumbar disc causes pressure on the “sacral dorsal roots within the vertebral canal” (Marieb et al., 2004). Consensus
among researchers acknowledges that the term “sciatica” describes symptomology, rather than a specific diagnosis. Patients will often describe a sharp, stabbing pain along the course of the nerve pathway. The condition may begin with or without definitive mechanism of injury, beginning abruptly or gradually over time. Small “movements of the limb, coughing, sneezing, and sitting generally intensified the suffering” (Decker & Albert, 2002). The term “piriformis syndrome” (PS) is regularly underdiagnosed, and there is “disagreement as to whether PS is a distinct clinical entity and whether it involves neuropathic pain from compression of the sciatic nerve as it courses through the piriformis muscle, or myofascial pain from a tight, hypertrophic, and tender piriformis without nerve entrapment” (Kirschner, Foye, & Cole, 2009). Patient symptomology in each diagnosis is extremely similar, making it difficult to differentiate one from another. Patients may be unable to flex the knee, due to the lack of hamstring innervation, and lose the ability to move the ankle about the talo-crural joint. The condition is usually unilateral. Additional symptoms include weakness, numbness and tingling in the affected leg (Decker & Albert, 2002). Pain with palpation along the course of the nerve might additionally be noted. A straight leg test (Lasègue’s sign) usually intensifies the pain (Prentice, 2006). Risk factors include smoking, obesity, and work-related activities (Cook et al., 2014).

In cases where the tibial nerve is solely injured, the “paralyzed muscles in the calf cannot plantarflex the foot, and a shuffling gait results” (Marieb et al., 2004). In comparison, when the common fibular nerve is injured, “dorsiflexion is lost and the foot drops into plantarflexion” (Marieb et al., 2004), known to pedorthists as “foot drop.” With prolonged injury to the sciatic nerve, muscle atrophy and joint rigidity could become apparent in the affected leg (Decker & Albert, 2002).

The location and description of the patient’s pain varies greatly depending on the location of nerve impingement (see Table 2). The source of the pain is the lower back, however, patients might present with lower limb pain, weakness, numbness, or paresthesia. The term “lumbar radiculopathy” describes injury to any lower back nerve, either by disc herniation or a narrowed spinal canal in the lumbar spinal cord. Ninety percent of impingements involve the sciatic nerve, specifically, L4–L5 and L5–S1 (Decker & Albert, 2002).

### Differential Diagnosis

Possible differential diagnoses include, but are not limited to:

1. **Spinal root compression of the lumbar plexus** – Major disturbances in gait are often noted with herniated discs in the first four lumbar spinal nerves (L1–L4). As the femoral nerve is impaired, muscles flexing the thigh at the hip (rectus femoris, iliacus) and extending the leg at the knee (quadriceps) are not adequately innervated. Obturator nerve impairment can additionally cause symptoms of pain and anesthesia in the anterior or medial thigh (Marieb et al., 2004).

2. **Diabetic peripheral neuropathy** – Patients suffering from diabetes can experience circulatory changes in their lower limbs. When neuropathy is present, the patient’s feet will be “warm, numb, dry, and usually painless” (Lorimer, French, O’Donnell, Burrow, & Wall, 2006).

3. **Lumbar muscular strain/low back pain** – Musculoskeletal injuries in the lumbar region can create similar symptomology as piriformis syndrome. Testing and history-taking are important in differentiating between these two conditions.

4. **Cauda equina syndrome (CES)** – This condition can often get confused in the early diagnosing of sciatica. Patients can experience a wide spectrum of non-specific signs and symptoms. Patients might experience any “combination of motor weakness and sensory disturbance in the lower extremities, back pain, unilateral or bilateral radicular pain and sphincter disturbance” (Balasubramanian, Kalsi, Greenough, & Seetharam, 2010). An important note: true CES is extremely rare, and has a higher prevalence in adults compared to children. This condition is outside the scope of pedorthic treatment, and when suspected, patients are recommended to seek urgent medical care.

5. **Syringomyelia** – Syringomyelia is a rare, but chronic condition where cyst formation within the spinal cord intrudes on the natural flow of cerebral–spinal fluid around the spine. As the condition progresses, the nerve fibers within the spine are slowly damaged. This neurological condition remains without cure.
Common Testing

History-taking, imaging, and physical examination are the three means of diagnosing sciatica, however “no history items or physical examination tests have both high sensitivity and high specificity” (Valat et al., 2010). The straight leg raise test (Lasègue’s sign) remains controversial, yet widely used in diagnosing low back pain and/or herniated discs. This test is commonly used in primary and secondary care, aiding in decision-making between diagnostic imaging and/or hospital referrals (Devillé et al., 2000). When completing the straight leg test, patients are asked to lie in a supine position. The patient’s leg should remain extended, and the pedorthist passively lifts this extended leg from the treatment table. The test is considered positive if pain is reproduced along the lumbar region. The test is complete at the point of maximal hip flexion, and/or pain is reproduced (Kamath & Kamath, 2017). This test must be performed with caution and control and should only be used if within the comfort zone of the attending pedorthist.

As Valat et al. explain, “signs and symptoms that help to distinguish between sciatica and non-specific low back pain are unilateral leg pain greater than low back pain, pain radiating to foot or toes, numbness and paraesthesia in the same distribution, straight leg raising test induces more leg pain and localized neurology, which is limited to one nerve root” (Valat et al., 2010).

Contraindications

Patients should have medical clearance and a direct referral prior to visiting the pedorthist. In cases where you suspect additional medical problems – for example, cauda equina syndrome – further testing and examinations should be considered.

The Patient Perspective

Patients will often enter the clinic initially describing low back pain, with or without a reported mechanism of injury. As pain often radiates down a unilateral leg, patients will often rub their hand down the gluteal and hamstring region of the affected limb. Along with these actions, patients will describe that the pain, numbness, or tingling seems to “continue” or “shoot” down the affected leg. A weakness in the leg may be evident to the patient. In addition to the patient physically moving their hand along the course of the nerve, they may describe an increase in pain with prolonged sitting and movement of the affected limb.

Common Treatment

Pedorthic treatment

It is of primary importance to differentiate a patient’s radicular pain from their referred pain, as “the epidemiology, clinical course and, most importantly, therapeutic interventions are different for these two conditions” (Valat et al., 2010). This can create problem areas for some clinicians, as true sciatica is mainly diagnosed by history taking and physical examination. Physicians and allied health professionals agree that treatment should remain conservative in the first six to eight weeks of acute sciatica (Valat et al., 2010). It is important to remember the nature of pedorthic treatment. As pedorthic treatment is conservative, we are not directly addressing the cause of patient’s neurological symptoms. Treatment is thus targeted towards addressing these locations of referred pain, as means of decreasing patient discomfort, and promoting a more efficient ambulation. In some patient cases, it might be difficult to differentiate between true clinical sciatica and lumbar radiculopathy.

Consequently, pedorthic treatment is dependent on the location of spinal impingement (see Table 2), and the patient’s location of referred discomfort. For example, if the spinal impingement occurs at L5, patients will have weakened neuromuscular control of the tibialis anterior and extensor hallucis longus. Abnormal amounts of pronation may be secondary to these weaknesses, as the musculature cannot minimize the excessive midfoot movement. In this example, custom orthoses and stability footwear may help to control the excessive midfoot movement during gait, consequently decreasing muscular demands. This treatment may also apply in patients with secondary foot drop, a result of neuromuscular weakness. Patients suffering from piriformis syndrome may demonstrate muscular tightness resulting from piriformis overuse. Decreasing the biomechanical demands of the muscle, allowing it to relax, can consequently decrease the pressure on the sciatic nerve.

If the patient has neuropathy on the plantar aspect of the foot, extra care is recommended with material choices for custom orthoses. A moisture wicking, open celled material is highly encouraged, as it increases airflow between the plantar aspect of the foot, and the surface of the orthosis. Fungal resistant materials are also available. Furthermore, pedorthists can educate their patients in the importance of foot hygiene and regular observations on the plantar surface of both feet.
**Additional considerations**

The sequence of muscle actions defining the “ankle strategy” is altered in patients with a history of sciatica (Huber et al., 2016). Consequently, balance and proprioceptive training may provide additional benefits to pedorthic treatment. If work-related activities are associated with the diagnoses, patients may benefit from occupational therapy to address the physical exposures at the site of employment.

As sciatica often involves inflammation around the affected area of the spine, rest is crucial in the acute stage of injury. Lumbar traction is effective at decreasing disc protrusion (Prentice, 2006). If nerve entrapment is a result of a tight piriformis muscle, the first course of treatment is to address the underlying cause, which could include stretching the musculature. The goal of stretching is to correct the underlying pathology, as the muscle tightness causes mechanical compression on the sciatic nerve. Following stretching, a physiotherapist will use a combination of “lumbosacral stabilization, hip strengthening exercises, and myofascial release" techniques (Kirschner, Foye, & Cole, 2009).

**Key Considerations**

As clinicians, it is important to keep in mind that the term “sciatica” describes symptomology, as opposed to a specific diagnosis. A new patient in your clinic might be suffering from “piriformis syndrome” or “lumbar neuropathy” yet describe all sciatica-related symptoms. Regardless of medical diagnosis, the patient’s biomechanics might be an exacerbating factor, adding to discomfort or pain. In other words, as pedorthists, we might not have the ability to remove the cause of our patient’s pain, however, we can use our knowledge towards enhancing ambulation, decreasing muscular demands, and improving quality of life.

**References**


Sciatica
Spinal Stenosis
Heather Orosz, BSc, C. Ped (C)

Key Messages
• Spinal stenosis is a narrowing of the spinal column which can impinge nerve roots or the spinal cord and may cause sensory and motor impairments.
• The severity of the stenosis does not correlate to the intensity of symptoms.
• The most common region for stenosis is in the lumbar spine, most often producing symptoms of neurogenic claudication.
• Neurogenic claudication symptoms are worsened with spinal extension and improved with spinal flexion.
• Conservative treatment of spinal stenosis can include custom foot orthoses and stable footwear to maintain lower limb alignment.

Keywords
cervical, claudication, degenerative disc, foramen, kyphosis, lordosis, lumbar, myelopathy, nerve root, neurogenic, radicular pain, spinal column, spinal cord, spondylosis, thoracic, vertebrae

Introduction
The vertebrae that compose the spine contain a channel, called the vertebral foramen, through which the spinal cord passes. Between each vertebra are two lateral foramina which allow space for the nerve roots to exit the spinal canal. Spinal stenosis is a narrowing (“stenosis”) of these channels, which on its own does not cause pain; however, if this narrowing progresses, it can impinge and compress nerve roots or the spinal cord, and can lead to pain or dysfunction in the areas of the lower limb innervated by these nerves. The most common symptoms are neurogenic intermittent claudication (or pseudoclaudication), radicular leg pain, and disruption of motor and sensory processes. Pain in the buttocks or legs is typical with numbness, tingling, or weakness in one or both legs, which is worsened during spinal extension and improved with flexion. Spinal stenosis can be congenital or acquired, but is most commonly the result of disc degeneration and osteoarthritis. As such, it is more prevalent in older populations and is becoming increasingly common as the population ages (Cadogan, 2011). In a study by Sasaki (1995), magnetic resonance imaging (MRI) showed spinal stenosis in 80% of patients over 70 years old. Lumbar spinal stenosis is uncommon before age 50 (Melancia et al., 2014), but cervical spinal stenosis can start showing symptoms during the third decade (Melancia et al., 2014). Congenital spinal stenosis is generally diagnosed earlier than degenerative spinal stenosis, but is still rarely apparent in childhood (Melancia et al., 2014). Not all patients with spinal stenosis are symptomatic. In fact, the degree of spinal stenosis does not correlate with the severity of symptoms, so a patient whose MRI shows mild stenosis might present with more severe symptoms than a patient with a greater degree of stenosis (Cuccurullo, 2014). At its mildest, spinal stenosis can be asymptomatic at its most severe, it can cause debilitating pain or numbness in the back and legs, impaired ambulation, incontinence, loss of bowel control, and reduced quality of life.

Pathology
Stenosis can occur at any level of the spine, but is found in the lumbar spine in about 75% of cases (Lohr, 2015) and at L4/5 in 91% of cases (Weinstein, 2008). It can also occur in the cervical spine, but it is rarely found in the thoracic spine. The lumbar and cervical spines are more mobile than the thoracic spine and consequently are more susceptible to wear and degeneration. Spinal stenosis can occur in only one vertebra or it can be multi-segmental. It can be unilateral or bilateral. A stenosis can form in the central canal of the vertebrae, the lateral recess, or foramina (Melancia et al., 2014).

Degeneration due to osteoarthritis and the aging
process is the most common cause of spinal stenosis (Lee, Kim, Oh, Lee, & Park, 2015). As we age, the discs between each vertebra become depleted of water and nutrients, causing them to thin and become stiff (Buckwalter, 1995). Once thinned they lose their ability to absorb shock and become unstable, leading to increased stress, hypertrophy of the facet joints, and narrowing of the spinal canal. This loss of disc height can also cause the ligamentum flavum to buckle and encroach on the space within the spinal canal, limiting the space for nerve roots (Buckwalter, 1995). The spinal canal can be further narrowed by osteophytes, resulting from osteoarthritis, entering the spinal column. The ossification of spinal ligaments and deterioration of facet joints will also contribute to spinal stenosis (Conrad, Shokat, Abbasi, Vincent, Seay, & Kennedy, 2013).

Other common causes of acquired spinal stenosis include herniated discs, bulging discs, scoliosis, spondylolisthesis (when one vertebra slips forward on another), tumors, and trauma or injuries such as vertebral fracture or dislocation. The processes of diseases such as rheumatoid arthritis or Paget's disease can also lead to spinal stenosis (Siebert, Prüss, Klingebiel, Failli, Einhäupl, & Schwab, 2009).

Less commonly, spinal stenosis is congenital. Individuals can be born with abnormally short and thick vertebral pedicles, reducing the space within the spinal column and intervertebral foramina, predisposing them to spinal stenotic symptoms (Melancia et al., 2014). Although spinal stenosis may be present at birth, it usually does not produce symptoms until patients are in their 30s or 40s (Melancia et al., 2014).

The compromised nerve roots at the site of the stenosis have reduced ability to send sensory and motor neural signals to the lower body, and can impair the function of the muscle groups innervated by the affected nerves. Symptoms of spinal stenosis will vary depending on the location of the stenosis and which nerve roots are involved, and can affect any part of the body innervated by the affected nerves. Nerve root and/or spinal cord compression can occur with cervical and thoracic stenosis, producing symptoms of pain, radiculopathy, myelopathy, or myeloradiculopathy (Melancia et al., 2014). Patients with lumbar spinal stenosis can present with progressive back pain, particularly when stenosis is a result of spondylolisthesis (Melancia et al., 2014), but more often they experience symptoms of neurogenic claudication. Patients will typically experience numbness, weakness, and radicular pain or sciatica from the spine to the buttocks in one or both legs while standing or walking. This can result in poor balance and gait abnormalities such as foot drop, reduced coordination, and frequent falls (Kim, Chun, Han, Moon, Kang, Kim, Park, Moon, Kim, Sohn, Shin, Jang, Lee, & Lee, 2011). These symptoms are worsened during spinal extension as this posture further reduces the space within the spinal canal. Symptoms are relieved during spinal flexion, or simply bending forward, which reduces lumbar lordosis and thoracic kyphosis, alleviating compression of the nerve roots or spinal cord.

Although very rare, severe cases of central lumbar spinal stenosis can compress the nerve bundle below the spinal cord and can cause cauda equina syndrome (Siebert et al., 2009). This results in sensory and motor deficits in the lower body, which can include incontinence and loss of bowel control.

Cervical stenosis is not as common as lumbar stenosis, but it can be more serious as it can result in not only nerve root compression, but also compression of the spinal cord itself, or cervical spondylotic myelopathy. Myelopathy can produce symptoms of radiculopathy, with shooting pains or paralysis in the extremities, reduced fine motor skills, uncoordinated gait, and balance problems (Melancia et al., 2014). If it progresses, later stages of cervical stenosis with myelopathy can result in sphincter deficits and paralysis of one or more limbs.

Differential Diagnosis

In cases of claudication, it is important to determine whether it is neurogenic and caused by spinal stenosis or vascular and caused by atherosclerosis and impaired blood circulation. If the claudication is neurogenic, the patient should feel symptom relief during sitting or leaning forward, whereas vascular claudication is unaffected by spinal flexion or extension. On the other hand, vascular claudication symptoms will worsen while walking uphill or during increased physical exertion due to the increased vascular demands, while neurogenic claudication improves while walking uphill due to increased spinal flexion and is unaffected by exertion (Melancia et al., 2014). Vascular claudication symptoms stop when activity is ceased, even while standing, whereas neurogenic claudication symptoms will continue while standing, and will only subside when the patient sits down or leans forward.

In addition to vascular claudication, other conditions that can mimic symptoms of lumbar spinal stenosis include (Ammendolia, 2014; Melancia et al., 2014):
Peripheral neuropathy – can produce similar neurological symptoms to spinal stenosis, but is unaffected by spinal posture

Hip osteoarthritis – buttock, groin, hip, and leg pain is increased during hip flexion and internal rotation, unlike spinal stenosis pain which is unchanged with hip rotation

Trochanteric bursitis (or greater trochanteric pain syndrome) – produces lateral hip pain that can radiate down to the knee and occasionally below the knee; patients will experience pain at night and while lying on the affected side, whereas patients with neurogenic claudication are usually pain-free when lying down

Learning which postures worsen symptoms will help to differentiate spinal stenosis patients from patients with non-specific low back pain, who typically experience more pain with prolonged sitting (Genevay & Atlas, 2010).

Differential diagnoses for cervical spondylotic myelopathy include amyotrophic lateral sclerosis (ALS) multiple sclerosis, herniated cervical disc, strokes and brain lesions (Toledano & Bartleson, 2013; Melancia et al., 2014).

Common Testing

Although diagnostic imaging such as MRI is a method of ascertaining the presence of spinal stenosis, the degree of stenosis within the spine is not reflective of the severity of symptoms (Genevay & Atlas, 2010). Some patients are unaware that they have spinal stenosis until they have an MRI or other imaging for an unrelated condition which reveals the narrowed spinal canal. Studies have found spinal stenosis in imaging findings in up to 20% of asymptomatic patients (Genevay & Atlas, 2010). Because of this discrepancy between the intensity of symptoms and the severity of the anatomical abnormality seen on imaging, there is risk of spinal stenosis being misdiagnosed as the cause of the patient’s symptoms, when in actuality, the spinal stenosis may be asymptomatic and the symptoms have an altogether different cause (Ammendolia, 2014). Therefore, diagnostic imaging is helpful to confirm a diagnosis but it is not sufficient to diagnose spinal stenosis as the cause of a patient’s symptoms. It is critical to perform a thorough medical history and physical exam, rather than relying solely on MRI findings for diagnosis.

The most common symptom of lumbar spinal stenosis is neurogenic claudication which is present during standing and walking upright and absent while leaning forward or sitting due to the delordosis that occurs in these positions, which opens the vertebral foramen. Patients with lumbar spinal stenosis may display the “shopping cart sign” (Ammendolia, 2014), and walk with a forward lean as though pushing a shopping cart, which increases the space of the spinal canal and reduces symptoms. Other gait abnormalities can vary depending on the area of spinal compression, but common gait compensations include decreased stride length and decreased gait velocity as functional limitations increase (Conrad et al., 2013). Foot drop can be displayed by patients with stenosis at the level of L4/5, as the dorsiflexing function of tibialis anterior and extensor hallucis longus are impaired (Wang & Nataraj, 2014).

Other physical characteristics that can be identified during a physical exam include limited range of motion at the lumbar spine during extension, limited sensation in the lower limb, and muscle weakness (Melancia et al., 2014). Ankle reflexes may be weak or absent; however, this can also be the case in older asymptomatic patients (Genevay & Atlas, 2010). Cervical stenosis can also result in upper extremity weakness or numbness.

Contraindications

Patients with spinal stenosis should avoid any activity that exacerbates symptoms. Contact sports and high impact activities should be avoided, as well as any activity that requires extension of the spine. Prolonged walking and standing tend to increase symptoms, so patients should limit themselves to short walks and take frequent breaks to sit down and alleviate compression of the nerve roots.

The Patient Perspective

Patients with spinal stenosis will typically complain of leg pain, numbness, weakness, or other neurogenic symptoms related to the location of the stenosis within the spine. Some patients will describe difficulty walking, a feeling of heaviness in the legs, or feeling clumsy due to the reduced sensory feedback caused by the stenosis. They may describe increased pain while walking and improvement upon sitting. They might describe increased pain while walking downhill and less pain walking uphill, as the spine will have greater forward flexion during uphill walking, increasing the foraminal space and reducing symptoms. This forward-leaning posture can also allow a patient who is unable to walk even a short
distance without symptoms to ride a bicycle for long distances without pain.

**Treatment**

Unless symptoms are severe, conservative treatment is effective as a first approach to managing spinal stenosis (Backstrom, Whitman, & Flynn, 2011). Zaina, Tomkins-Lane, Carragee, & Negrini (2016) note that the difference in outcomes between surgical and conservative treatment for lumbar spinal stenosis is insignificant; however, with surgical intervention, 10–24% of patients experience side effects, compared to no side effects with conservative treatment.

Conservative treatment can include pain medication and non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroid injections to reduce inflammation, spinal bracing, and physical therapy (Melancia et al., 2014). Physical therapy can be beneficial in improving flexibility and strengthening spinal musculature to improve stability of the spine as well as strengthening the abdominals to compensate for excessive lumbar extension (Melancia et al., 2014). Lumbar hyperextension and spinal stenosis symptoms can also be exacerbated when there is a limitation in hip extension, which causes compensations to the lumbar spine during gait (Michaud, 1997). By improving hip range of motion, these symptoms may be reduced.

As good posture and optimal spinal alignment are beneficial to limit the symptoms of spinal stenosis, custom foot orthoses can be part of the treatment plan to improve the body’s posture by way of the kinetic chain. Overpronation of the subtalar joint and excessive eversion of the calcaneus causes internal rotation of the tibia and lower limb, increasing pelvic anteversion, and leading to an increase in lumbar lordosis and thoracic kyphosis (Pinto, Souza, Trede, Kirkwood, Figueiredo, & Foseca, 2008; Farokhmanesh, Shirzadian, Mahboubi, & Shahri, 2014; Khamis & Yizhar, 2007), which can increase the compression of spinal nerve roots. Custom orthoses fabricated with a varus rearfoot post will reduce overpronation and eversion of the calcaneus (Michaud, 1997), and may lead to delordosis, lessening the compression of spinal nerve roots. Custom orthoses can also be beneficial for patients with neurogenic claudication by improving balance and reducing muscle fatigue. Orthosis design for patients with spinal stenosis will vary depending on their biomechanics and the symptoms they are experiencing. Those with neuropathy affecting the foot will require softer orthoses to reduce pressure points and shearing forces, and to improve shock absorption.

Other pedorthic treatment can involve providing the patient with stable footwear with a wide base of support, particularly when balance or proprioception has been impaired. Footwear with rocker soles may improve ambulation. Shoe fitters and pedorthists must be extra cautious with patients who have sensory deficiencies in the feet, as the lack of sensory feedback could lead to irritation or sores in an improperly fitted shoe.

If conservative treatment fails to improve symptoms to an acceptable level, surgery is commonly performed. Depending on the anatomy and location of the stenosis, there are various surgical procedures available. Spinal decompression surgery (such as laminectomy, discectomy, or facetectomy) involves removing tissue from the posterior spine to open the foramen. In some cases, a spinal fusion is also required to reduce instability in the affected area (Zaina et al., 2016).

**Key Considerations**

Spinal stenosis is, in many cases, a result of the degeneration that occurs as part of the aging process and cannot be prevented. It can affect any region of the spine and can produce symptoms throughout the body, but most commonly neurogenic claudication. Radiographic imaging can detect the presence of a stenosis; however, it is not necessarily symptomatic. Conservative treatment is best as a first approach, including physical therapy, posture and activity modification, custom orthoses, and stable footwear. However, surgical intervention may be necessary in many cases to decompress the spine and provide more space for nerve roots and the spinal cord.

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Clinical Practice Guidelines
A reference manual of best practice in pedorthic care
Second Edition

The Pedorthic Association of Canada (PAC) first published Clinical Practice Guidelines in 2012, under the leadership of a volunteer Task Force.

This second edition includes updates to original chapters as well as several new chapters. As in 2012, this has been a volunteer-driven exercise with the participation of Canadian Certified Pedorthists from coast to coast along with other health care professionals.

PAC’s Clinical Practice Guidelines aim to:

• improve and standardize pedorthic practice across Canada;

• identify acceptable standards of care on a condition-by-condition basis;

• advance the profession in the eyes of the general public, other health care providers, and policymakers;

• help patients and the public better understand and appreciate the practice of pedorthics;

• strengthen the community of practising pedorthists; and

• serve as a focus of ongoing discussion about best practice, new ideas, and improving patient care.

Ultimately, PAC’s leadership believes that these Clinical Practice Guidelines will help pedorthists serve their patients with increasing skill and with even a sharper focus on best practice. This publication is seen as a focal point for dialogue; a foundation for new research and study; and as an effective, user-friendly, patient-centred resource.