Veterans Review and Appeal Board

"Your right to be heard"

ORTHOPAEDIC HANDBOOK

Revised May 2019

Prepared by: Stanish Orthopaedic Inc.

Dr. William Stanish

Professor of Orthopaedic Surgery, Dalhousie University

Director, Orthopaedic and Sport Medicine Clinic of Nova Scotia

Halifax, Nova Scotia, Canada

Veterans Review and Appeal Board Canada

Tribunal des anciens combattants (révision et appel) Canada

Canada

The objective of Discussion Papers is to provide general information on medical issues. Their aim is to present a balanced view of the current medical knowledge on a particular topic. They are written to be understood by lay individuals. They have been prepared by experts selected by the Veterans Review and Appeal Board. They are not peer-reviewed. A Panel of the Board may consider and rely on the medical information provided in a discussion paper, but the Board is not bound by an opinion expressed in a discussion paper in any particular claim. Every decision of the Board must be based on the facts of the particular case.

Table of Contents

OSTEOARTHRITIS

Introduction

Osteoarthritis is a chronic and progressive degenerative process that afflicts more than 4 million Canadians.[1]

These numbers are expected to double in the next thirty years.[1]

There are two main types of arthritis which are as follows:[2]

a) **Osteoarthritis** - This is a degenerative type of arthritis that develops over time with progressive physical damage to the joint cartilage. Osteoarthritis has traditionally been thought to be to be a progressive condition due to "gradual wear and tear on the joints" over the years. It is now recognized that other factors also may be involved, such as progressive damage to the underlying bone.

b) **Inflammatory Arthritis** - This type of arthritis is initially inflammatory in nature, which then leads to progressive erosion of the joint cartilage. Such entities as rheumatoid arthritis, gout and psoriasis are considered to produce the inflammatory type of arthritis.[2]

Osteoarthritis has a significant impact on day-to-day functioning and has no known cure.[3]

Anatomy and Pathoanatomy

Joints are the connections between any two bones in the body.

The ends of the bones are covered with articular cartilage, which provides a smooth movement that is friction-free.

The first change that occurs in osteoarthritis is damage to the smooth articular cartilage.

Once the cartilage is damaged, it is no longer as effective at taking loads. This leads to further deterioration over time, hence the name degenerative osteoarthritis.

Osteoarthritis was once thought to be caused by wear and tear alone - considered to be a normal part of aging.[3]

We now appreciate that osteoarthritis is due to abnormal joint loading which occurs after joint injury or with obesity.[4]

Systemic factors such as inflammation, early aging and sex also enter the picture in terms of contributing factors to the development of osteoarthritis.[4]

Figure 1 Abnormal joint loading and other factors lead to a gradual breakdown of the smooth articular cartilage at the end of the bones making up the joint.

Key Points

- Normal joints are very effective at distributing loads and providing friction- free movement.
- Once the joint is damaged a degenerative process begins that gradually progresses over time.
- Osteoarthritis most commonly affects weight-bearing joints such as hips and knees.

Incidence and Prevalence

Joints most commonly affected by osteoarthritis arc the weight-bearing joints.

Nearly 1 in 100 Canadian adults (over the age of 20 years) have experienced at least moderate to severe pain, limiting their activities due to osteoarthritis.[4]

Osteoarthritis is a degenerative process that progresses over time. By age 65 years approximately 32% of women and 22% of men in Canada will have been diagnosed with osteoarthritis. By age 70 years, over half of people are affected.[1,5,6]

Key Points

- More than 1 in 10 Canadians have osteoarthritis after age 20.
- Symptoms can occur at any time, but usually appear after the age of 40 years. Hip and knee are most commonly affected.

Risk Factors

Osteoarthritis has a multi-factorial etiology with different sets of factors associated with its incidence. These factors include age, sex, obesity and genetics.[4]

Some risk factors arc modifiable, such as a decrease in body weight.

Occupational factors may also play a role, as osteoarthritis seems to be more common in people whose job involves heavy lifting or increased joint stresses.[7]

However, controversy exists as to whether specific occupations do indeed render the knee joint (cartilage) more prone to arthritis than others.[4,7]

It has been argued that the cumulative load of standing all day and every day is a causative factor in the development of arthritis. No such correlation has ever been proven scientifically.[7]

Crouching and kneeling have always been incriminated in being responsible for provoking premature cartilage wear. Although appealing in theory, there are no studies that have provided convincing evidence that the everyday knee stressors experienced by plumbers or electricians cause early knee arthritis.

However, if a worker undergoes knee surgery that removes or debrides cartilage/meniscus, it is likely that the individual will experience progressive knee arthritis.

Key Points

- Obesity increases the risk of osteoarthritis in the knee.
- Obesity worsens the symptoms of osteoarthritis in the hip and back.
- Previous joint injuries can lead to osteoarthritis.

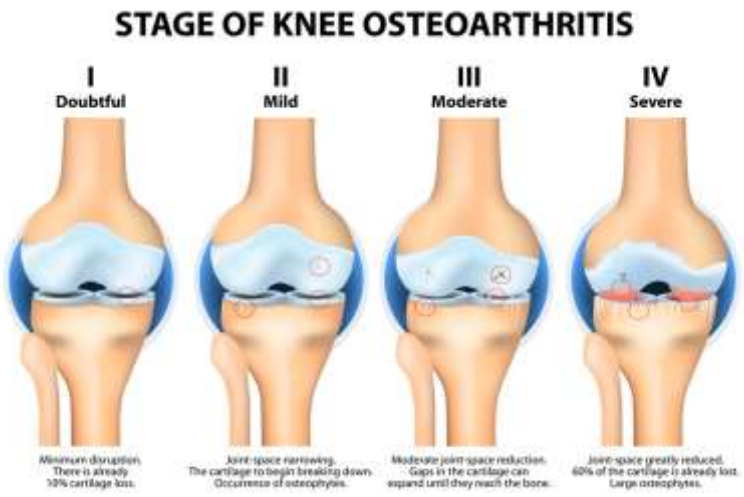


Figure 2 Osteoarthritis progresses through stages, which may take many years to develop. Symptoms are rarely present until the moderate or severe stages.

Natural History

The natural history of osteoarthritis is not well documented nor easy to generalize. This is due to the fact that the development of osteoarthritis is gradual, taking years to evolve, and the progression differs at varying joint sites.[2,4,8]

Significant cartilage damage may have occurred before relevant signs and symptoms appear.

There are known inconsistencies between findings on x-rays and clinical symptoms, with only 50% of subjects with radiographic osteoarthritis being clinically symptomatic.[5] Clinical symptoms, which may be recurrent or continuous, may precede x-ray findings by up to 10 years.[9]

Figure 3
X-ray changes can take many years to develop, and many people will have changes on x-ray, yet remain asymptomatic.

Of the 4 ½ million Canadians with osteoarthritis, approximately 600,000 will have severe enough pain such that it significantly limits their activities.[1]

The frequency and progression of osteoarthritis is unpredictable, though joint changes continue to progress over time. In a 15-year follow-up study, about 50% of patients with knee osteoarthritis experienced joint deterioration while the other 50% showed no change.[5]

Key Points

- The amount and rate of osteoarthritis progression is unpredictable.
- Osteoarthritis begins clinically with joint pain and swelling after vigorous activity.
- Later stages of osteoarthritis involve loss of joint motion, with episodes of more severe pain and swelling, usually after more intense activities.

Treatment

Recommendations for treatment for osteoarthritis have been published by many groups and are consistent.[10-15] Figure 4 illustrates the hierarchy of the treatment approach. Patient education programs related to exercise, healthy diets and strategies to avoid joint stresses have been shown to be effective for managing symptoms and improving function.[16]

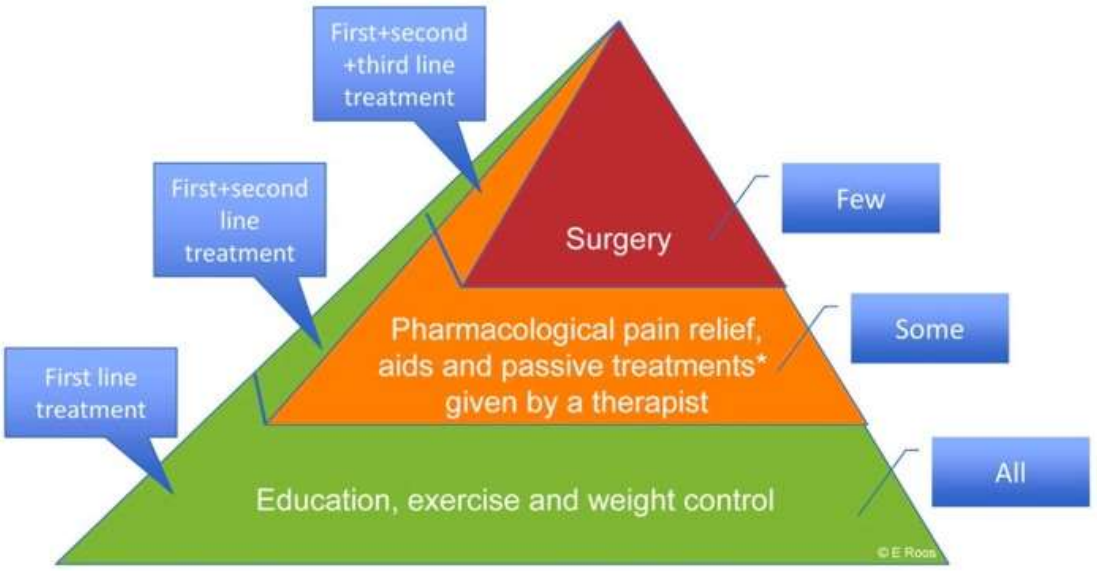


Figure 4 This figure illustrates the treatment approach for osteoarthritis. Items at the bottom of the pyramid are recommended for everyone with OA, while only a few patients will require surgery. (GLA:D Canada. https://gladcanada.ca)

For advanced joint degeneration, surgery such as total joint replacement, may be recommended. Arthroscopic procedures for osteoarthritis of the knee are no longer routinely recommended, though they may be needed for some patients.[18]

Summary

- Osteoarthritis is the most common joint disorder in adults and may progress with age.
- This very common disorder afflicts the joint cartilage, making it prone to uneven wear.
- Symptoms are most bothersome in the hips and knees.
- Symptoms tend to progress as the osteoarthritis becomes more advanced.
- However, there are some cases with severe osteoarthritis but the individual has few symptoms.
- Osteoarthritis can be treated very successfully in many cases with exercise,

weight loss and walking aids.[19]

- Surgery may sometimes be necessary.

Patient Profile #1

A 49-year-old patient presents with pain about the medial side of the knee.

The patient had surgery at the age of eighteen, where his medial meniscus was removed. Clinical examination and x-ray revealed degenerative arthritis on the medial side of the joint with a varus deformity. Knee bracing is recommended to redistribute forces from the medial to the lateral side of the knee.

This is a story of a person who suffers from osteoarthritis due to increased loading on the medial side of the knee after removal of the medial meniscus, which is a common after-effect of that type of surgical procedure.

Patient Profile #2

This gentleman is a retired 66-year-old former floor installer. He has been experiencing progressive pain and swelling in both knees. His pain is increased with squatting, kneeling and going down stairs.

He is 5'9" and 225 pounds with a body mass index of 33.2.

He is unable to fully straighten his right knee and has progressive genu varum (bow legs). An x-ray shows joint space narrowing and early osteophyte formation (beaking at edge of joint). He is referred to a dietitian for weight loss and to a physiotherapist for education and a personalized exercise program. He is advised to use acetaminophen to reduce his pain.

References

1. Bombardier C, Hawker G, Mosher D. *The Impact of Arthritis in Canada: Today and Over 30 Years*. Arthritis Alliance of Canada, 2011. Available at: http://www.arthritisalliance.ca/images/PDF/eng/Initiatives/20111022_2200_impact_of_arthritis.pdf .
2. Felson D. Epidemiology of Rheumatic Diseases. In D. McCarty, W Koopman (Eds.) Arthritis and Allied Conditions; 12th edition. London: Lea & Febiger; 1993, pp. 37-39.
3. Gignac M, Davis AM, Hawker G, et al. "What do you expect? You're just getting older": A comparison of perceived osteoarthritis-related and aging-related health experiences in middle- and older-age adults. *Arthritis Care and Research.* 2006; 55(6):905-12.
4. Man GS and Mologhianu G. Osteoarthritis pathogenesis – a complex process that involves the whole joint. J Med Life. 2014; 7(1):37–41.
5. Public Health Agency of Canada. *Life with Arthritis in Canada: A Personal and Public Health Challenge.* 2011. Available at: http://www.phac-aspc.gc.ca/cd-mc/arthritis-arthrite/lwaic-vaaac-10/index-eng.php.
6. Bellamy N, Wilson C, Hendrikz J. Population-Based Normative Values for the Australian/Canadian (AUSCAN) Hand Osteoarthritis Index: Part 2. *Seminars in Arthritis and Rheumatism*, 2011; 41(2):139-148.
7. Maetzel A, Makela M, Hawker G, Bombardier C. Osteoarthritis of the hip and knee and mechanical occupational exposure – a systematic review of the evidence. *Journal of Rheumatology*. 1997; 24:1599-1607.
8. MacDonald KV, Sanmartin C, Langlois K, Marshall DA. Symptom onset, diagnosis and management of osteoarthritis. Health Reports, 2014; 25(9): 10-17, Statistics Canada, Catalogue no. 82-003-X.
9. Hannan MT, Felson DT, Pincus T. Analysis of the discordance between radiographic changes and knee pain in osteoarthritis of the knee. *Journal of Rheumatology*. 2000; 27(6): 1513-1517.
10. Zhang W, Moskowitz R, Nuki G, et al. OARSI recommendations for the management of hip and knee osteoarthritis, Part I: Critical appraisal of existing treatment guidelines and systematic review of current research evidence. *Osteoarthritis and Cartilage*. 2007; 15(9):981-1000.
11. Zhang W, Moskowitz R, Nuki G, et al. OARSI recommendations for the management of hip and knee osteoarthritis, Part II: OARSI evidence-based, expert consensus guidelines. *Osteoarthritis and Cartilage* 2008; 16(2):137-62.
12. Zhang W, Nuki G, Moskowitz R, et al. OARSI recommendations for the management of hip and knee osteoarthritis, Part III: changes in evidence following systematic cumulative update of research published through January 2009. *Osteoarthritis and Cartilage* 2010;18(4): 476-99.
13. Hochberg MC, Altman RD, April KT, Benkhalti M, Guyatt G, McGowan J, et al. American College of Rheumatology 2012 recommendations for the use of nonpharmacologic and pharmacologic therapies in osteoarthritis of the hand, hip, and knee. *Arthritis Care Res*. 2012; 64(4):465-74.

14. Nelson AE, Allen KD, Golightly YM, Goode AP, Jordan JM. A systematic review of recommendations and guidelines for the management of osteoarthritis: The chronic osteoarthritis management initiative of the U.S. bone and joint initiative. *Semin Arthritis Rheum*. 2014; 43(6):701-12.

15. Kaulback K, Jones, S, Wells C, Felipe E. Viscosupplementation for knee osteoarthritis: a review of clinical and cost-effectiveness and guidelines. Ottawa. CADTH; 2017 (CADTH rapid response report: a summary with critical appraisal). Accessed March 22, 2019: available at: https://www.cadth.ca/sites/default/files/pdf/htis/2017/RC0895%20Viscosupplementation%20for%20Knee%20Osteoarthritis%20Final.pdf

16. Public Health Agency of Canada. *What is the Impact of Arthritis and What are Canadians Doing to Manage Their Condition?* 2010. available at: http://www.phac-aspc.gc.ca/cd-mc/slcdcfs-epamccfi/pdf/SLCDCFactSheet2009-Arthritis-eng.pdf.

17. Felsen DT, Zhang Y, Hannan MT et al. The incidence and natural history of knee osteoarthritis in the elderly. The Framingham Osteoarthritis Study. *Arthritis and Rheumatism*. 1995; 38:1500-1505.

18. Wong I et al. Position Statement : Arthroscopy of the Knee Joint. Developed by the Arthroscopy Association of Canada (AAC) September 2017. Approved and Endorsed by the Canadian Orthopaedic Association (COA) Board of Directors January 2018. Accessed March 22, 2019. available at: https://coa-aco.org/wp-content/uploads/2017/05/Position-Statement-on-Arthroscopy-of-the-Knee-Joint-–-AAC-COA-march2018.pdf

19. GLA:D Canada. https://gladcanada.ca

INJURIES TO THE MENISCUS OF THE KNEE

Introduction

Injuries to the menisci of the knee are common.

Usually a torn meniscus does not require surgical treatment.[1]

In fact, history has suggested that the surgical removal of the knee meniscus can result in the development of arthritis.[2]

This chapter will explain the contemporary understanding and management of disorders of the knee meniscus.

Anatomy and Function of the Meniscus of the Knee

The knee joint has two menisci - one on the inside (medial) and another on the outside (lateral).[3]

These discs of fibrocartilage act as shock absorbers. They also function as stabilizers of the knee.

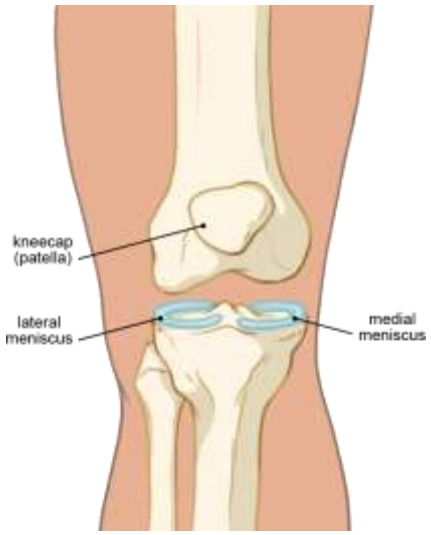


Figure 1 There are two menisci – a medial meniscus on the inner side of the knee and a lateral meniscus on the outer side. They both sit inside the knee joint between the femur and tibia and ensure the two bones match well.

The menisci cushion the loads between the femur (thigh) and the tibia (shin).[3] Thus they must absorb more load with running and jumping compared to walking. As stabilizers, the knee menisci come into play with rotation and pivoting (squatting as well).

It is important to understand that the knee meniscus has a poor blood supply (circulation).[4] As such, its ability to heal (once injured) is impaired.

The outer rim of the meniscus has a much richer circulation, thus a better chance for repair (versus the inner part which has no blood supply).[4]

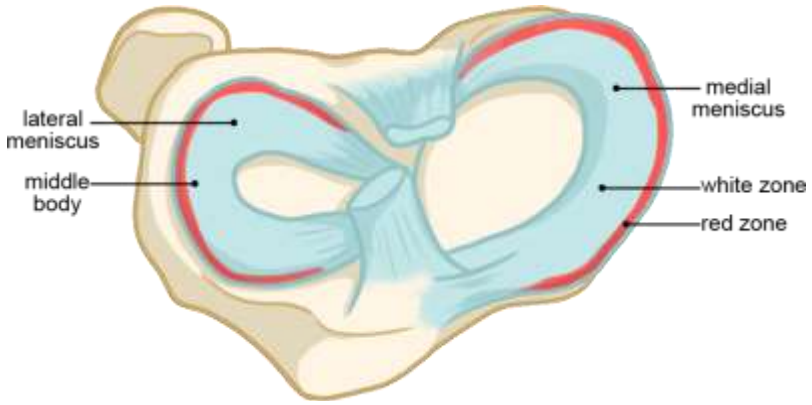


Figure 2 The outer rim of the meniscus is more likely to heal if injured because it has a blood supply. The inner parts of the meniscus are unlikely to heal once damaged because they have no circulation.

Incidence and Prevalence of Meniscal Injuries

Tear of the meniscus is more common in males than in females, with the ratio ranging from 2.5:1 versus 4:1.[1]

The incidence of traumatic tears of the meniscus is approximately 60 cases per 100,00 people, peaking in men between 20-30 years of age. For both sexes, degenerative tears become more common after the age of 35.[1]

Degenerative tears are most prevalent amongst the elderly - they are usually associated with osteoarthritis.[2]

One study found that 76% of people with an average age of 65 years, who were asymptomatic, had x-ray evidence of a tear of the meniscus.[5]

Key Points

- Tears of the meniscus are most common in males
- 75% of persons over 65 years old have tears of the menisci
- Most tears are degenerative in type

Mechanism of Injury

Generally there are two types of disorders (tears) of the meniscus.[5]

The first type occurs in the younger population; the second type is more frequent in the aging individual.

Type #1 ~ Acute Injury

The clinical characteristics of this type of meniscal disorder are as follows:

- Younger population, less than 35 years old.
- Follows an acute injury.
- The mechanism is usually twisting in nature and is of relatively low energy (unless associated with a ligament tear).
- The pain is not severe and the joint has mild swelling.
- Locking may occur.

Type #2 ~ Degenerative Tears

The clinical characteristics of this type of tear include:

- Older population, greater than 35 years of age.
- Rarely has a history of acute trauma.
- Associated with mild discomfort, which is intermittent (sometimes with swelling).
- Usually associated with an element of arthritis.

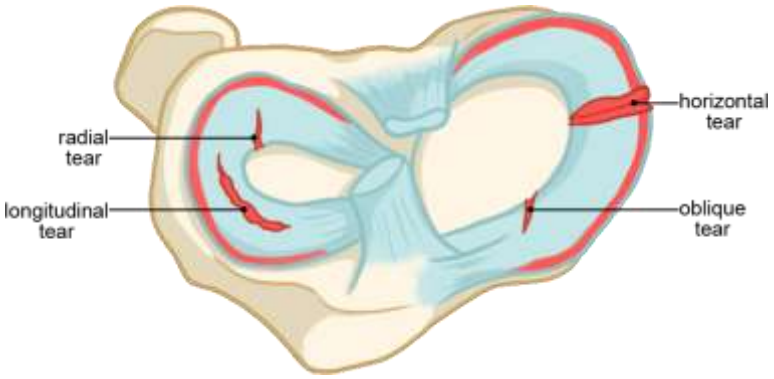


Figure 3 Various types of tears in the meniscus can occur gradually over time.

Natural History

The natural history of meniscal tears is variable, depending on the location, size and type of tear.[6,7,8]

As mentioned previously, injuries of the meniscus can result from either a single traumatic event, which most commonly occurs in the younger population, or degeneration within the meniscus (older population).
Tears of the meniscus can be asymptomatic or they can be accompanied by varying degrees of pain, swelling and catching or locking within the joint.[5,7]

Degenerative tears occur in the meniscus as they lose their elasticity with age and are often accompanied by knee osteoarthritis. These degenerative tears are common and can occur from routine activities. They may very well remain asymptomatic throughout life.[2]

Treatments

Medical and/or surgical management for disorders of the meniscus has changed considerably over the past two decades.[8-18]

Many guiding principles have been established.

Principle #1

The meniscus should be preserved, if at all possible.[7,8]

Principle #2

The meniscus, when injured, should be salvaged and repaired. (In the younger person.)[10,12,17]

Principle #3

If necessary, minimal surgical excision is fundamental.[18]

Principle #4

Replacement (transplantation) of the meniscus would be a consideration in unique cases.[9]

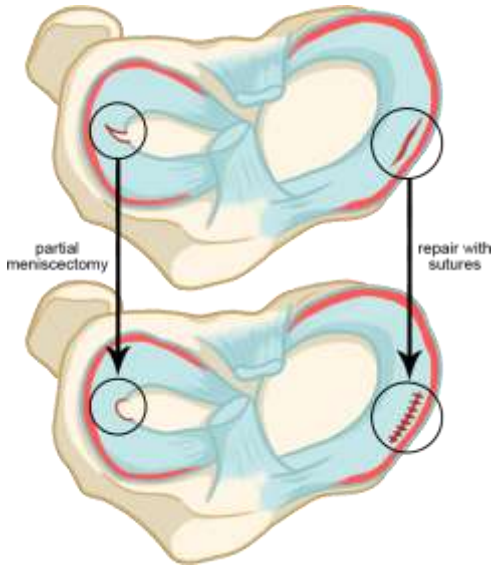


Figure 4 Damage to the meniscus should be repaired whenever possible. Tears in the inner portion of the meniscus may need partial removal, but the majority of the meniscus should be preserved to prevent later osteoarthritis in the knee.

Outcomes

The knee meniscus is a vital structure for proper knee mechanics. It is critical to understand the importance of the knee meniscus in providing the knee joint with stability and the ability to absorb stress (load).

If injured, the meniscus should be preserved whenever possible.

Meniscal repair, reconstruction and/or replacement should always be considered.

If the meniscus is removed surgically, a favorable functional outcome could be compromised.[2,7] Arthritis is likely to develop in that compartment of the knee joint (that has undergone meniscectomy).

Summary

- Tears of the meniscus are common.
- Meniscus tears are often asymptomatic and do not require treatment.
- Minor tears of the meniscus are stable and can be left untreated.
- Treatment depends on the type of tear and the symptoms of the individual.
- The meniscus should be preserved whenever possible

Patient Profile #1

A 50-year-old engineer is working in an awkward position (squatting) and experiences pain about the medial side of his knee (inside).

Initial treatment is ice, physiotherapy and a mild anti-inflammatory.

An MRI reveals a degenerative tear of the medial meniscus with early osteoarthritis.

He was treated without surgery, using a small knee brace.

He is essentially pain-free except for discomfort on deep squatting.

Patient Profile #2

A 20-year-old military recruit is playing flag football. He incurs a high energy to his knee. There is an immediate swelling within the joint and he is unable to gain full extension.

The x-ray/MRI reveal a peripheral detachment of the meniscus with an associated tear of the anterior cruciate ligament.

At surgery, the meniscus is repaired and sutured in place; at the same time his anterior cruciate ligament is reconstructed.

Two years later he has a full range of motion and essentially normal function of his knee for all activities of daily living. There is no evidence of osteoarthritis.

References

1. Brockmeier S, Rodeo S. Meniscal Injuries. In: DeLee J, Drez D, Miller M, editors. *DeLee and Drez's Orthopaedic Sports Medicine*; 3rd ed. Electronic: Saunders, 2009

2. Englund M, Guermazi A, Lohmander LS. The Meniscus in Knee Arthritis. *Rheumatic Diseases Clinics of North America,* 2009; 35(3):579-590.

3. Wojtys EM, Chan DB. 2005. Meniscus Structure and Function. *Instructional course lectures,* 2005; 54:323-330.

4. Arnoczky SP, Warren RF. Microvasculature of the Human Meniscus. *American Journal of Sports Medicine*, 1982; 10(2):90-95.

5. Howell R, Kumar NS, Patel N, Tom J. Degenerative meniscus: Pathogenesis, diagnosis, and treatment options. *World J Orthop*, 2014; 5(5):597-602

6. Salata MJ, Gibbs AE, Sekiya JK. A systematic review of clinical outcomes in patients undergoing meniscectomy. *American Journal of Sports Medicine*, 2010; 38(9):1907-1916.

7. Stanish WD, Vincent NC. Is the Meniscus Worth Saving? *The Nova Scotia Medical Bulletin*. 1984; 63(5):139-142.
8. Seil R, Becker R. Time for a paradigm change in meniscal repair: save the meniscus! *Knee Surg Sports Traumatol Arthrosc.* 2016;24(5): 1421-1423.

9. Beaufils P, Becker R, Kopf S, et al. Surgical management of degenerative meniscus lesions: the 2016 ESSKA meniscus consensus *Knee Surg Sports Traumatol Arthrosc*, 2017; 25(2):335-346.

10. Hulet C, Menetrey J, Beaufils P, et al.; French Arthroscopic Society (SFA). Clinical and radiographic results of arthroscopic partial lateral meniscectomies in stable knees with a minimum follow up of 20 years. *Knee Surg Sports Traumatol Arthrosc.* 2015; 23(1):225-231.

11. Pujol N, Lorbach O. Meniscal repair: Results. In: Hulet C, Pereira H, Peretti G, Denti M, editors., eds. *Surgery of the meniscus*. Berlin, Heidelberg: Springer Verlag, 2016:343-355.

12. Westermann RW, Wright RW, Spindler KP, Huston LJ, Wolf BR; MOON Knee Group. Meniscal repair with concurrent anterior cruciate ligament reconstruction: operative success and patient outcomes at 6-year follow-up. *Am J Sports Med* 2014; 42(9):2184-2192.

13. Khan M, Evaniew N, Bedi A, Ayeni OR, Bhandari M. Arthroscopic surgery for degenerative tears of the meniscus: a systematic review and meta-analysis. *CMAJ.* 2014; 186(14):1057-1064.

17. Nepple JJ, Dunn WR, Wright RW. Meniscal repair outcomes at greater than five years: a systematic literature review and meta-analysis. *J Bone Joint Surg Am*. 2012; 94(24):2222-2227.

18. Paxton ES, Stock MV, Brophy RH. Meniscal repair versus partial meniscectomy: a systematic review comparing reoperation rates and clinical outcomes. *Arthroscopy* 2011; 27(9):1275-1288.

LEG LENGTH DISCREPANCIES

Introduction

Leg length inequality is a difference between the lengths of the right and left legs.

Almost all individuals have a slight difference in their leg lengths, usually less than 1 cm.[1]

The degree of leg length difference may contribute to musculoskeletal problems such as low back pain. This remains controversial. Some authors suggest that the leg length discrepancy must be 2 to 3 cm. to be of clinical significance.[1,2]

Incidence and Prevalence

The incidence of leg length inequality in the normal population is estimated to be between 60 and 90 percent.[1]

There are two types of leg length inequality.[2]

Type #1 ~ Structural

The structural type is due to actual difference in the length of the bones of the leg. This can be a consequence of injury, growth disturbance or surgery such as hip/knee replacement.

Structural leg length discrepancy affects up to 90% of the general population with a mean discrepancy of 5.2 mm.[1]

Thirty-two percent of military recruits in one study had a .5 - 1.5 cm. difference between the lengths of their legs.[3] This is a normal variation.

It has been suggested that approximately 30% to 50% of patients may experience a leg length difference after total hip replacement.[4] The operated limb is usually lengthened after surgery.

Type #2 ~ Functional

Apparent leg length inequality (functional) is a result of muscle tightness or weakness. There is no difference in actual bony length, but soft tissue changes create the equivalent of a leg length difference.

Key Points

- Leg length inequality is common; most people have one leg 5 to 10 mm. difference
- Most leg length discrepancies are 2 cm. or less and are not considered clinically significant in an adult.
- Only 1 out of 1000 people has a leg length inequality over 2 cm.

Measuring Leg Length Inequality

The most common method for measuring leg length is by using a tape measure (see right).[6] The distance between the front hip bone (anterior superior iliac spine (ASIS)) and inside ankle bone (medial malleolus) is compared on the left and right.

The most accurate way to measure leg length is via a standing x-ray.[6] This is seldom required.

A difference in leg length usually results during childhood growth. Trauma (such as a fracture) or growth plate abnormalities may result in unequal leg lengths.[1]

There is a disagreement in the literature as to what amount of leg length inequality is of significance.

The longer leg is subject to larger forces during walking, which may result in problems in the hip, knee, foot or lower back.[7-12]

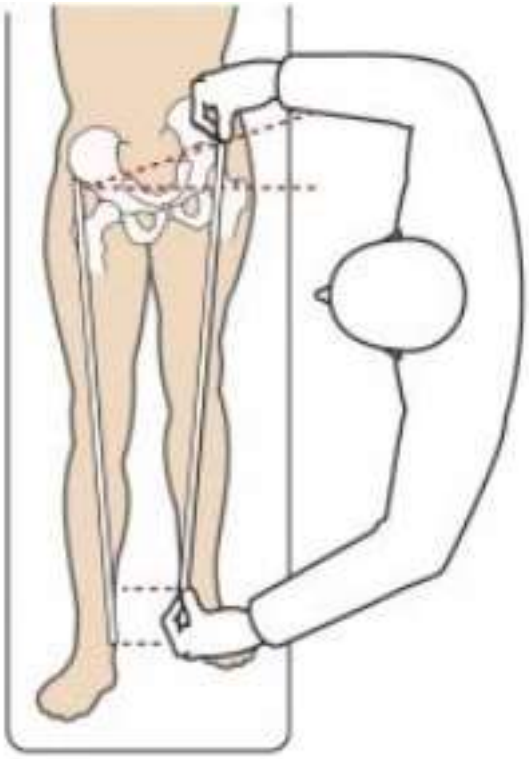


Figure 1
A tape measure is used between bony points on the left and right legs to measure leg length.

A study of military recruits revealed the occurrence of stress fractures 73% in the longer limb, 16% in the shorter limb and 11% in limbs of equal length.[10] Knee problems may be more common in the shorter leg.[12,13]

Leg length discrepancies are fairly common after total hip replacement.[14,15]

Leg length discrepancies under 2 cm. are believed to have limited significance, as individuals tend to compensate with other joints and muscles. Walking patterns are essentially unchanged.[16]

Key Points

- Leg length discrepancies typically develop during childhood.
- Leg length discrepancies less than 2 cm. are well tolerated.
- Leg length inequalities have been implicated in a variety of disorders including low back pain.
- There is little research to predict how much of a leg length inequality will cause a long-term problem.

Treatments and Outcomes

There is disagreement regarding the amount of leg length inequality adults can tolerate without treatment. Finding a difference in leg length in someone who is on their feet during the workday should probably be corrected. This can be done by using a foot orthotic that can be inserted inside a shoe; larger leg length differences may require a shoe's sole to be built up.[17,18]

Figure 2
Internal heel lifts or orthotics can be placed inside shoes (left), while larger differences may require the sole of the shoe to be thickened. A combination of the two approaches also may be used.

Reid and Smith[18] suggested dividing leg length discrepancy into three categories.

a) Mild, less than 3 cm. - These cases should go untreated or treated non-surgically.

b) Moderate, 3 to 6 cm. - These cases should be treated on a case by case basis with some requiring surgery.

c) Severe, greater than 6 cm. - These cases will likely need surgical correction.

Key Points

- Leg length discrepancies under 2 cm. are believed to be of limited significance.
- Foot lifts (see right) or orthotics are a simple and effective treatment for most leg length difficulties.
- More than a 5 - 6 cm. difference may require surgery.

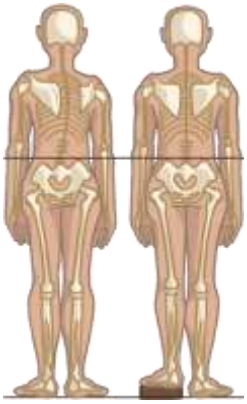


Figure 3
Leg length differences can be compensated by lifts or orthotics once the desired thickness is established.

Summary

- Leg length discrepancies are common and do not cause problems long-term in most people.
- In the adult population, up to 90% of people have a leg length inequality of at least .5 cm., while up to 70% have a leg length difference of .5 to 1 cm.
- Most people can tolerate up to 2 cm. of difference and usually do not require treatment.
- If treatment is necessary, shoe lifts are a simple, inexpensive and effective means for improving function.

Profile #1

A 26-year-old female recruit had a one-year history of right knee pain.

Thorough medical history revealed that she had had an orthopedic knee intervention as a child.

A plain film x-ray confirmed the presence of a leg length discrepancy which was 3 cm. The leg length discrepancy was likely secondary to a growth arrest after her childhood surgery.

The patient was managed with a heel left and physiotherapy.

Profile #2

A 58-year-old semi-retired male had previous surgery twenty years ago to his right knee - removal of the medial meniscus.

This resulted in considerable osteoarthritis and leg deformity. His right knee was 3.6 cm. shorter than the left.

Following total knee replacement, the patient's leg length discrepancy was corrected, bringing the leg lengths back to within .5 cm. of each other.

References

1. Blake RL, Ferguson H. Limb length discrepancies. *J Am Podiatr Med Assoc*. 1992; 82(1):33-38.

2. Schuster RO. Legs, the long and the short of it. *The Runner*. 1980; 2:70.

3. Helsing AL. Leg length inequality: a prospective study of young men during their military service. *Ups J Med Sci*. 1988; 93(3):245-253.

4. Desai AS, Dramis A, Board TN. Leg length discrepancy after total hip arthroplasty: a review of the literature. *Curr Rev Musculoskelet Med* 2013;6(4):336-341.

5. Rothbart B, Estabrook L. Excessive pronation: a major biomechanical determinant in the development of chondromalacia patella and pelvic lists. *J Manip Phys Ther*. 1988; 11(5):373-379.

6. Woerman AI, Binder-MacLeod SA. Leg length discrepancy assessment: accuracy and precision in five clinical methods of evaluation. *J Orthop Sports Phys Ther*. 1984; 5(5):230-239.

7. Mahmood S, Huffman LK, Harris JG. Limb-length discrepancy as a cause of plantar fasciitis. *J Am Podiatr Med Assoc* 2010; 100(6):452-455.

8. Korpelainen R, Orava S, Karpakka J, et al. Risk factors for recurrent stress fractures in athletes. *Am J Sports Med*. 2001; 29(3):304-310.

9. Minkowsky I, Minkowsky R. The spine: an integral part of the lower extremity. In: Valmassy RL (ed.) *Clinical Biomechanics of the Lower Extremity*. Mosby, Philadelphia, 1965, pp. 101-107.

10. Friberg O. Leg asymmetry in stress fractures: a clinical and radiographic study. *J Sports Med Phys Fitness*. 1982; 22(4):485-488.

11. Tallroth K, Ylikoski M, Lamminen H, Ruohonen K. Pre-operative leg length inequality and hip osteoarthritis: a radiographic study of 100 consecutive arthroplasty patients. *Skeletal Radiol*. 2005; 34(3):136-139.

12. Murray KJ, Azari MF. Leg length discrepancy and osteoarthritis in the knee, hip and lumbar spine. *J Can Chiropr Assoc*. 2015; 59(3):226-237.

13. Subotnick SI. Limb length discrepancies of the lower extremity (the short leg syndrome). *J Orthop Sports Phys Ther*. 1981; 3(1):11-16.

14. Maloney WJ, Keeney JA. Leg length discrepancy after total hip arthroplasty. *J Arthroplasty*. 2004;19(4 Suppl 1):108-110.

15. Roder C, Vogel R, Burri L, Dietrich D, Staub LP. Total hip arthroplasty: leg length inequality impairs functional outcomes and patient satisfaction. *BMC Musculoskelet Disord*. 2012; 13: 95.

16. Gurney B, Mermier C, Robergs R, et al. Effects of leg length discrepancy on gait economy and lower-extremity muscle activity in older adults. *J Bone Joint Surg Am* 2001; 83(6):907-915.

17. Akinola B, Jones HW, Harrison T, Tucker K. Shoe raises for symptomatic leg length discrepancy after total hip replacement: do patients find them useful? *Internet J Orthop Surg* 2104; 22(1).

18. Reid DC and Smith B. Leg length inequality: a review of etiology and management. *Physiotherapy Canada*. 1984; 36(4):177-182.

DISORDERS OF THE ROTATOR CUFF

Introduction

The shoulder is the most mobile joint in the body.[1]

Due to the anatomical structure of the shoulder, it is able to move in many different directions.

The shoulder is held in place by soft tissues which include tendons, ligaments and cartilage.

These shoulder tissues can be injured in a variety of ways - for instance, by an acute sudden insult or by aging alone (degeneration).

Anatomy of the Rotator Cuff and Shoulder

Important Features:

The shoulder joint has a very shallow cup (glenoid) which is deepened by a cartilage rim (labrum). The ball of the shoulder (humerus) sits in the glenoid and is stabilized by four tendons which constitute the rotator cuff.[1]

These tendons are in action with every movement of the shoulder. They work in harmony as the shoulder joint moves in all directions.

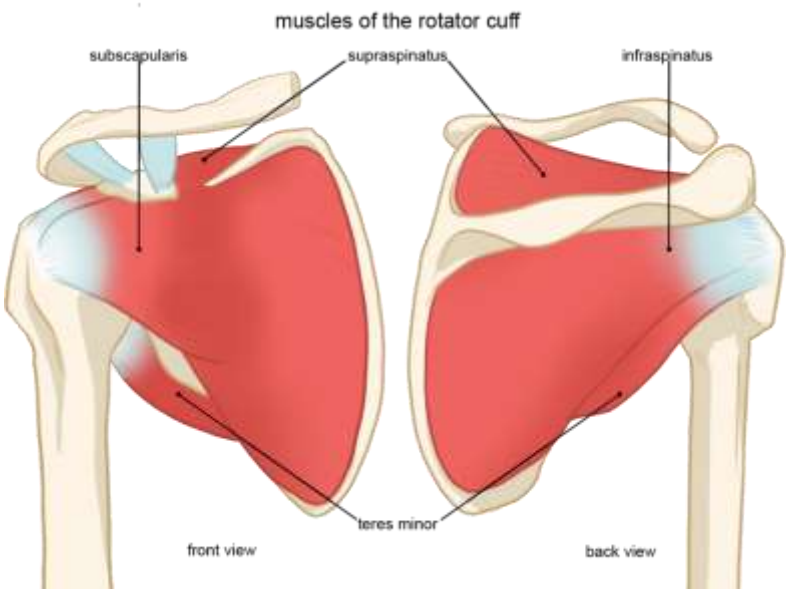


Figure 1 The humerus (arm) contacts the glenoid of the scapula and is held in place and controlled by the four rotator cuff muscles: supraspinatus, infraspinatus, teres minor and subscapularis.

The prime mover of the shoulder is that component of the rotator cuff that pulls the arm away from the body and into the overhead position - the supraspinatus tendon.

The supraspinatus tendon works the hardest in shoulder movements and thus is the most frequently injured.[2]

Another important feature of the anatomy of the supraspinatus tendon is the fact that it has a very poor blood supply. This reality predisposes the tendon to early wear (degeneration) and prolongs recovery after injury.[3]

To complete our understanding of shoulder anatomy, it is important to note the role of the labrum.[1]

The labrum provides a rim of cartilage that functions to stabilize the shoulder.

The labrum can be injured when the shoulder is dislocated (comes out of the joint).

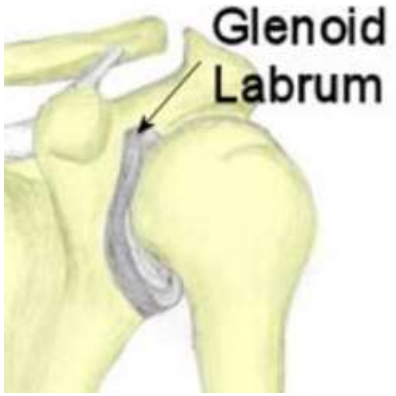


Figure 2
The glenoid labrum makes the shoulder more congruent and stable.

Further, repetitive forces (such as in a laboring job) can also injure the labrum, as well as the rotator cuff.

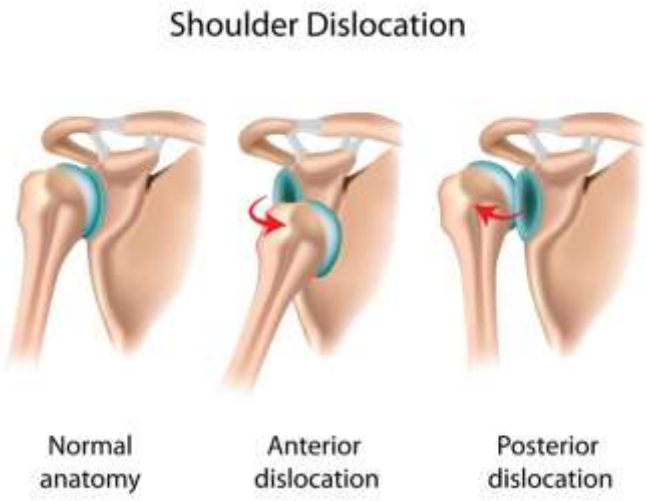


Figure 3 The labrum is a ring of cartilage that supports the shoulder. It can be damaged with dislocation or through repetitive use of the shoulder.

The biceps tendon is attached to the upper part of the shoulder and can be injured in isolation or in conjunction with the labrum.[4]

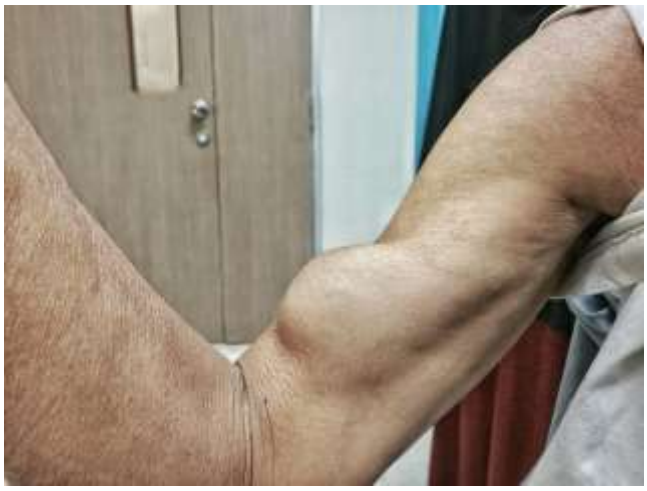


Figure 4 The biceps tendon, which is attached at the front of the shoulder, can rupture. This lets the muscle slide down the arm creating a 'popeye' appearance.

Incidence and Prevalence of Rotator Cuff Disorders

In a review of the general population, degeneration (natural aging) of the rotator cuff is common. Over 60% of individuals beyond the age of 60 years will have radiological evidence of tears of the rotator cuff.[5,6,7]

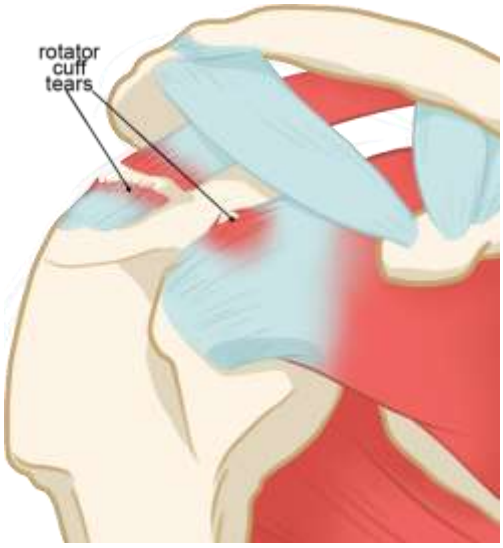


Figure 5 The rotator cuff (usually the supraspinatus portion) can develop tears through wear over time, or through use of the shoulder in overhead positions, especially if lifting repetitively.

Typically rotator cuff tears develop from natural wear and tear (degeneration) of tendons. As a result, prevalence of cuff disorders does increase with age.[8]

That said, there are some occupations that tax the shoulder excessively and can be responsible for premature deterioration of the tendons. Athletes and laborers, especially those who use overhead motion, have a higher incidence of rotator cuff disruptions.[9] Fishermen, for example, are known to have a very high incidence and prevalence of rotator cuff difficulties.[10]

Rotator cuff injuries in the younger population are exceedingly rare and generally result from traumatic shoulder injuries, but are quite uncommon.[11]

Sher et al[12] reported only 4% of individuals, between the ages of 19 and 39, had a tear of the rotator cuff.

Disease progression has not been well studied.

Many individuals with full thickness tears of the rotator cuff can be functioning normally with no pain.[13]

Another important fact is that few, if any, rotator cuff tears actually heal or decrease in size over time.[13]

Larger rotator cuff tears can be associated with decreased strength and increased pain.[14]

It is important to note that upwards of 75% of rotator cuff tears are asymptomatic and the presence of a tear (radiographically) should not be the sole guide to treatment.[15]

Many individuals with a full thickness tear can have normal functioning with no pain.[13,15]

Key Points

- More than 60% of persons over 60 years old will have rotator cuff tears
- Not all individuals with rotator cuff tears experience pain
- Persons with rotator cuff tears may function normally

Mechanism of Injury

Most disorders of the rotator cuff are degenerative in nature (normal aging).[16,17] Co-morbidities that may accelerate the aging process include smoking, obesity, diabetes and some medications.[18,19]

Repetitive occupational challenges that extend over many years and are arduous in nature (heavy lifting, overhead or pulling) have been incriminated in premature tendon degeneration.[9,10,20]

Acute tears of the rotator cuff are less common but do occur. They usually occur as a consequence of a fall on an outstretched arm or an abrupt traction injury to the shoulder; i.e., grabbing a handrail.[21]

That patient is usually younger and has immediate pain. This is in contrast to the degenerative tear that triggers milder pain.

Acute ruptures of the long head of the biceps usually occur with a similar mechanism of injury; i.e., acute traction/pulling.[4] (See Figure 4)

Natural History of Injuries

Most degenerative tears of the rotator cuff do not require surgical repair.

Recent data suggests over 90% of individuals over the age of 60 years, with a full thickness rotator cuff tear, have a favorable outcome with a non-surgical program. This non-surgical program basically emphasizes exercise.[8,13,21]

These individuals are not limited or restricted in all activities of daily living, whether occupational, recreational or domestic.

The young patient with a symptomatic rotator cuff tear, whether complete or partial, may require surgical intervention.[23] That decision assumes that the younger patient has failed a non-surgical rehabilitation program and is well motivated. It is important to note that most individuals that have a partial or full thickness disruption of the rotator cuff can be completely asymptomatic (such as pain) thus they continue to function normally in all activities of daily living.[24,25]

When conservative treatment is ineffective, surgical options can be considered. Surgical decisions are based on symptoms, level of physical activity, size of the tear, quality of tissue and response to conservative treatments.[26] Surgery is generally recommended for younger individuals with a traumatic tear because they have quite a good chance of healing and usually demand higher shoulder function than the older patient.[23,27]

Summary

- Degenerative rotator cuff tears are very common.
- More than 50% of people over the age 60 years have some wear of the rotator cuff.
- Full thickness tears may be asymptomatic.
- Individuals with full thickness tears can still have full function of the shoulder.
- Conservative treatment is highly effective.

- Surgical options exist when conservative treatment is ineffective.

Patient Profile #1

A 38-year-old military truck driver slips on the threshold of his truck, grabs on to the rearview mirror and has severe pain in his shoulder.

Upon presentation, he is unable to move the shoulder away from his side beyond 25 degrees. The MRI of the shoulder reveals a complete tear of the rotator cuff. Within three weeks of injury, the patient undergoes surgery to repair the rotator cuff disruption.

Six months after the repair, he is back to full and unrestricted duties.

This is a typical story of an acute rotator cuff tear in a relatively young person, that responds best to a surgical program.

Patient Profile #2

A 63-year-old retired military veteran suffers with pain about his right shoulder, after he fell in his garden on his outstretched arm.

He had a history of intermittent shoulder pain, dating back for fifteen years.

On examination, his range of motion is 90 degrees of forward elevation.

MRI of the shoulder reveals a tear of the rotator cuff, with retraction and evidence of chronic impingement.

This patient responded well to a physiotherapy and exercise program, designed to strengthen his surrounding muscles.

Five months after the injury the patient is functioning well, with a range of motion of 140 degrees and he has returned to all his daily activities, including gardening and golf.

This is a typical story of a chronic rotator cuff problem, with an acute injury in an older patient. Most patients in this age group respond best to a conservative, non-surgical program.

References

1. Terry GC, Chopp TM. Functional anatomy of the shoulder. *J Athl Train*, 2000; 35(3): 248-255.

2. Lin KC, Krishnan SG, Burkhead WZ. Impingement Lesions in Adult and Adolescent Athletes. In: DeLee J, Drez D, Miller M, editors. *DeLee and Drez's Orthopaedic Sports Medicine*; 3rd Ed., Saunders. 2009

3. Lohr JF, Uhthoff HK. The microvascular pattern of the supraspinatus tendon. *Clin Orthop Relat Res.* 1990; 254:35-38

4. Krupp RJ, Kevern MA, Gaines MD, Kofara S, Singletin SB. Long Head of the Biceps Tendon Pain: Differential Diagnosis and Treatment. *Journal of Orthopaedic & Sports Physical Therapy*, 2009; 39(2):55–70

5. Moosmayer S, Smit HJ, Tariq R, Larmo A. Prevalence and characteristics of asymptomatic tears of the rotator cuff: An ultrasonographic and clinical study. *The Journal of Bone and Joint Surgery Br.* 2009; 91(2): 196-200.

6. Yamaguchi K, Ditsios K, Middleton WD, et al. The demographic and morphological features of rotator cuff disease. A comparison of asymptomatic and symptomatic shoulders. *The Journal of Bone and Joint Surgery Am.* 2006; 88(8): 1699-1704.

7. Reilly P, MacLeod I, McFarland R, Windley J, Energy RJ. Dead Men and Radiologists Don't Lie: A review of cadaveric and radiological studies of rotator cuff tear prevalence. *Annals of the Royal College of Surgeons of England,* 2006*;* 88(2): 116-121.

8. Teunis T, Lubberts B, Reilly BT, Ring D. A systematic review and pooled analysis of the prevalence of rotator cuff disease with increasing age. *Journal of Shoulder and Elbow Surgery.* 23(12):1913-1921.

9. Linaker CH, Walker-Bone K. Shoulder disorders and occupation. *Best Pract Res Clin Rheumatol.* 2015; 29(3):405-423.

10. Eckert C, Baker T, Cherry D (2018) Chronic Health Risks in Commercial Fishermen: A Cross-Sectional Analysis from a Small Rural Fishing Village in Alaska, *Journal of Agromedicine*, 2018; 23(2):176-185

11. Yamamoto A, Takagishi K, Osawa T, Yanagawa T, Nakajima D, Shitara H, Kobayashi T. Prevalence and risk factors of a rotator cuff tear in the general population. *J Shoulder Elbow Surg.* 2010; 19(1):116-120.

12. Sher JS, Uribe JW, Posada A, Murphy BJ, Zlatkin MB. Abnormal findings on magnetic resonance images of asymptomatic shoulder. *Journal of Bone and Joint Surgery Am.* 1993; 77(1): 10-15.

13. Yamaguchi K, Tetro AM, Blam O, et al. Natural history of asymptomatic rotator cuff tears: A longitudinal analysis of asymptomatic tears detected sonographically. *Journal of Shoulder and Elbow Surgery*, 2001; 10(3):199-203.

14. Favard J, Berhouet M, Colmari E, et al. Massive rotator cuff tears in patients younger than 65 years. What treatment options are available? *Orthopaedics & Traumatology: Surgery & Research*, 2009; 95(4):19-26

15. Tashjian RZ Epidemiology, natural history, and indications for treatment of rotator cuff tears. *Clin Sports Med*, 2012; 31(4):589-604.

16. Teunis T, Lubberts B, Reilly BY, Ring D. A systematic review and pooled analysis of the prevalence of rotator cuff disease with increasing age. *J Shoulder Elbow Surg*. 2014; 23(12):1913-1921.

17. Fehringer EV, Sun J, VanOeveren LS, Keller BK, Matsen FA. Full-thickness rotator cuff tear prevalence and correlation with function and co-morbidities in patients sixty-five years and older. *J Shoulder Elbow Surg*. 2008; 17(6):881-885.

18. Bishop JR, Santiago-Torres JE, Rimmke N, Flanigan DC. Smoking Predisposes to Rotator Cuff Pathology and Shoulder Dysfunction: A Systematic Review. *Arthroscopy*. 2015; 31(8):1598-1605.

19. Sayampanathan AA, Andrew THC. Systematic review on risk factors of rotator cuff tears. *Journal of Orthopaedic Surgery*, 2017; 25(1):1–9.

20. Craik JD, Mallina R, Ramasamy V, Little NJ. Human evolution and tears of the rotator cuff. *Int Orthop*. 2014; 38(3):547-552.

21. Aagard KE, Abu-Zidan F, Lunsjo K. High incidence of acute full-thickness rotator cuff tears. *Acta Orthop*, 2015; 86(5): 558-562.

22. Clement ND, Nie YX, McBirnie JM. Management of degenerative rotator cuff tears: a review and treatment strategy. *Sports Med Arthrosc Rehabil Ther Technol.* 2012; 4(1):48.

23. Murray J, Gross L. Optimizing the management of full-thickness rotator cuff tears. *J Am Acad Orthop Surg.* 2013; 21(12):767-771.

24. Safran O, Schroeder J, Bloom R, Weil Y, Milgrom C. Natural history of nonoperatively treated symptomatic rotator cuff tears in patients 60 years old or younger. *Am J Sports Med.* 2011; 39(4):710-714.

25. Kuhn JE, Dunn WR, Saunders R, AnQ, et al. Effectiveness of physical therapy in treating atraumatic full-thickness rotator cuff tears: a multicenter prospective cohort study. *J Shoulder Elbow Surg*. 2013; 22(10):1371-1379.

26. Lambers Heerspink FO, Dorrestijn O, van Raay JJ, Diercks RL. Specific patient-related prognostic factors for rotator cuff repair: a systematic review. *J Shoulder Elbow Surg.* 2014; 23(7):1073-1080.

27. Lee TQ. Current biomechanical concepts for rotator cuff repair. *Clin Orthop Surg*. 2013; 5(2):89-97.

PLANTAR FASCIITIS

Introduction

Pain about the heel is not unusual in the general population.

Plantar fasciitis is arguably the most common cause of pain about the heel in adults.[1,2]

It may be caused by a variety of triggers including a sudden increase in activity, a change in footwear or an abrupt increase in body weight.

Inflammation of the plantar fascia - is said to afflict approximately 10% of the population at some point in their adult life.[1]

This disorder - plantar fasciitis - can be successfully treated with simple remedies such as footwear modification, normalization of body weight and sometimes physiotherapy.[3-6]

Due to the fact that in 90% of cases the condition is self-limiting, surgery is rarely necessary.

Anatomy of Plantar Fascia

The plantar fascia is a fibrous band that originates on the front part of the heel (calcaneus) and moves forward to the front part of the foot in the toe region.

This fascia, by nature, is tight and assists in the support of the longitudinal arch of the foot.

Anatomically it has a poor blood supply, is rigid in nature and thus when the fascia becomes stretched, the insult normally occurs as the fascia comes off the heel.

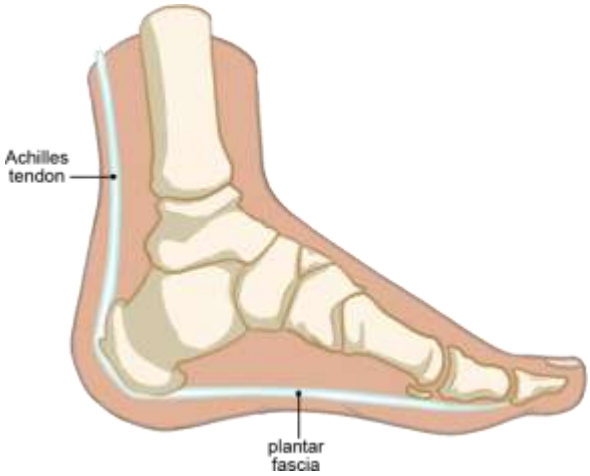


Figure 1
The plantar fascia is a tough band of tissue that supports the arch of the foot

If the inflammation is chronic and prolonged, a small spur can develop at the origin of the plantar fascia - referred to as a calcaneal spur.

Function

As one walks or runs, the plantar fascia springs into action.[7]

Due to the fact that it is quite rigid, it tends to focus the stress/strain at its origin which is off the heel bone (calcaneus).

If the plantar fascia is overly tight, as with a high-arched foot, it seems to be more prone to injury.

Likewise, a person with a flat foot can experience excessive strain on the arch resulting in plantar fasciitis.

Mechanism of Injury Producing Plantar Fasciitis

When the plantar fascia is stretched beyond its intrinsic strength, injury can occur.

Such an insult can occur with such simple triggers as adopting new footwear, walking on different surfaces or carrying excessive body weight.[8]

These injuries are usually micro tears in the plantar fascia and not complete full disruptions or tears. A complete tear (severing) of the plantar fascia is exceedingly rare.

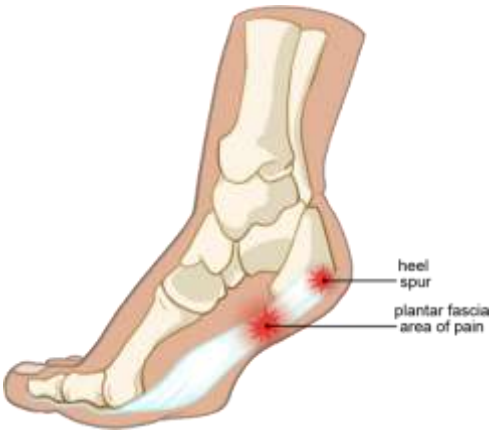


Figure 2
Injury to the fascia usually occurs near the attachment to the calcaneus (heel bone)

As mentioned, the micro tears are usually at the origin of the fascia, as it arises from the heel.

This can result in inflammation and, as mentioned, a calcaneal spur. (see Figure 3)

Natural History of Plantar Fasciitis

In most cases plantar fasciitis will settle with time alone; i.e., self-limiting.[9,10]

Simple treatments such as footwear modification, soft orthotics and reduction in body weight will ordinarily settle the fasciitis.

In the short term, the patient may find it necessary to alter their activities; i.e., bicycling rather than running. However, in the long term, modification of activity is not necessary.

Rarely, mild medications may be suggested but only in acute/subacute cases. Physiotherapy modalities such as stretching, can likewise prove beneficial.

Cortisone injections or surgery should be used sparingly and only in unique cases.[3]

Outcomes, as suggested in most cases of plantar fasciitis, will be self-limiting. Modification or limitations of activities are rarely necessary in the long term.

Summary

- Plantar Fasciitis is a common condition of the foot.
- It is readily treated with non-surgical techniques.
- The high-arched foot (pes cavus) and excessive weight gain can be precipitating causes.
- The vast majority of cases are self-limiting and do not require aggressive treatment.

Profile Patient #1

A twenty-two year old male military recruit presented with heel pain after a 5 mile run.

This is double the distance he had ever run before.

On examination he had a high-arched foot and was 20 pounds overweight.

X-rays showed that he had a calcaneal spur.

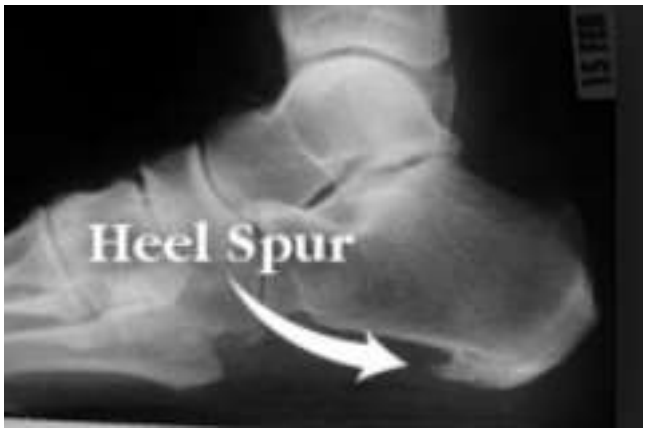


Figure 3
The plantar fascia pulls on the calcaneus (heel bone) and creates a small spur of bone – a heel (calcaneal) spur

His program of treatment included slight adjustment in his running program, a soft heel sponge (see Figure 4) and a stretching program designed by his physiotherapist.

Over four weeks the foot pain had settled entirely and he has since ramped up his program of running.

This is a typical story of a person who develops acute plantar fasciitis when an exercise program was ramped up too quickly and prompted micro-tearing of the plantar fascia.

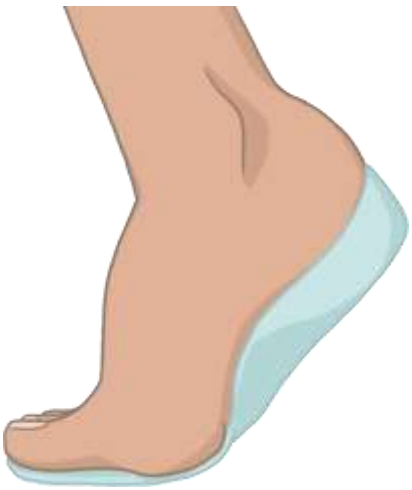


Figure 4
A soft insert is used to cushion the plantar fascia and to help support the arch of the foot

Profile Patient #2

A 43-year-old nurse in the military adopted new footwear that had a heel lower than what she was accustomed. After a full week she commenced having pain about her plantar fascia, as well as the Achilles tendon.

Cortisone injections to the fascia gave her but short-term relief. She was eventually placed on a program of strengthening and stretching her plantar fascia, as well as her Achilles tendon.

After six weeks she was entirely pain-free. She resumed her program of exercise and progressive weight reduction.

This is the story of an individual with a change in footwear that placed excessive strain on the soft tissues about the heel. The successful program of treatment was strengthening those tissues rather than prolonged rest/immobilization.

References

1. Riddle DL, Schappert SM. Volume of ambulatory care visits and patterns of care for patients diagnosed with plantar fasciitis: A national study of medical doctors. *Foot and Ankle Int.* 2004; 25(5):303-310

2. Buchbinder R. Clinical Practice: Plantar Fasciitis. *N Engl J. Med* 2004; 350(21):2159-2166

3. David JA, Sankarapandian V, Christopher PR, Catterjee A, Macaden AS. Injected corticosteroids for treating plantar heel pain in adults. *The Cochrane Database of Systematic Reviews.* 2017; 6: CD009348**.**

4. Anderson J, Stanek J. Effect of foot orthoses as treatment for plantar fasciitis or heel pain". *Journal of Sport Rehabilitation*. 2013; 22(2):130-136.

5. Thomas JL, Christensen JC, Kravitz SR, Mendicino RW, Schuberth JM, Vanore JV, Weil LS, Zlotoff HJ, Bouche R, Baker J. The diagnosis and treatment of heel pain: a clinical practice guideline-revision 2010. *The Journal of Foot and Ankle Surgery.* 2010; 49 (3 Suppl):S1-19.

6. Lim at, How CH, Tan B. Management of plantar fasciitis in the outpatient setting. *Singapore Medical Journal.* 2016; 57(4):168-170.

7. Aquino A and Payne C. Function of the plantar fascia. *The Foot,* 1999; 9(2):73-78

8. Beeson P. Plantar fasciopathy: revisiting the risk factors. *Foot and Ankle Surgery.* 2014; 20 (3):160-165.

9. Rosenbaum AJ, DiPreta JA, Misener D. Plantar heel pain. *The Medical Clinics of North America.* 2014; 98 (2):339-52.

10. Lareau CR, Sawyer GA, Wang JH, DiGiovanni CW (June 2014). Plantar and medial heel pain: diagnosis and management. *The Journal of the American Academy of Orthopaedic Surgeons.* 2014; 22(6):372-80.

HALLUX RIGIDUS (Limitus)

Introduction

Hallux Rigidus (Limitus) is not a rare condition.

By definition, it is an arthritic condition that affects the first joint of the big toe. As the wear of the joint cartilage progresses, the movement of the joint decreases.[1]

The terminology 'Hallux Rigidus' essentially means 'stiff big toe'.

It can be an incidental finding on routine x-rays of the foot.

Symptoms from this condition vary widely. The extent of the degeneration (arthritis) does not always parallel the patient's symptoms.

Hallux rigidus is seen in two distinct populations. When seen in adolescence, there is invariably a family history of this condition.[2]

Hallux rigidus might also present in adulthood.

Anatomy & Function

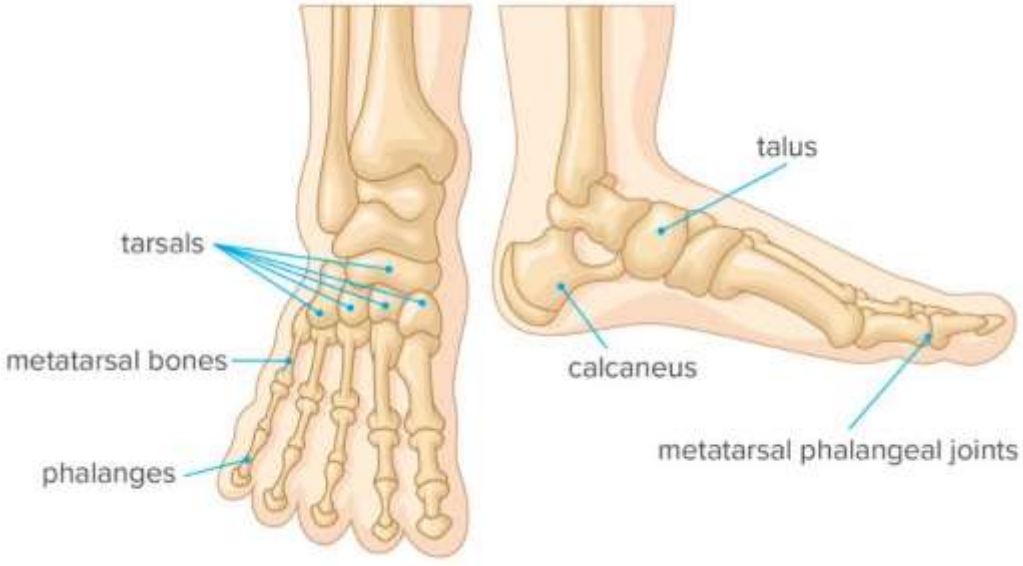


Figure 1 The bones of the foot are divided into the tarsal bones (near the ankle), the metatarsals (flat part of the foot) and the phalanges (toes). The first metatarsophalangeal joint attaches the big toe to the flat part of the foot.

The joint of the first toe (metatarsophalangeal joint) is lined by cartilage which is smooth. The hyaline cartilage is without blood supply and resembles gristle in consistency. It is nourished by the fluid within the joint and by movement.

Unfortunately, once degradation of the cartilage begins, the process continues as cartilage has no ability to heal.

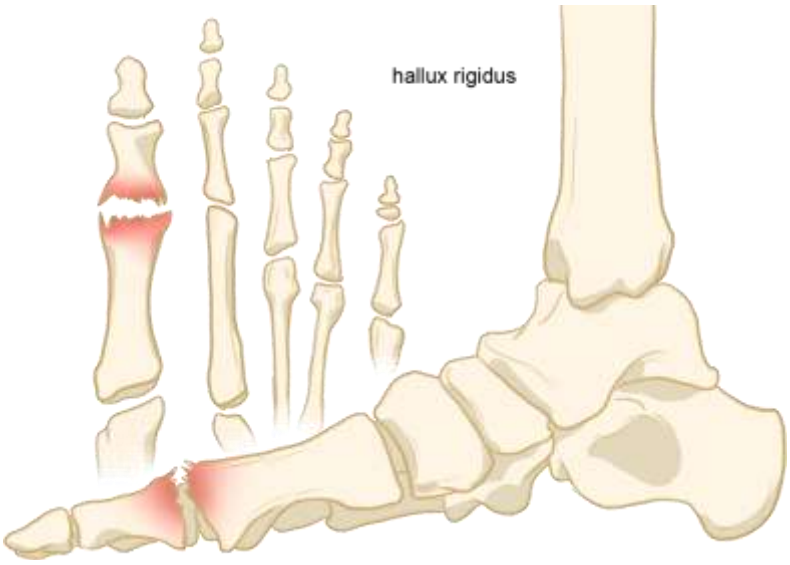


Figure 2 The cartilage surfaces of the metatarsal or phalanx can break down over time, leading to arthritis and limiting the movement.

The metatarsophalangeal joint moves upward and downward in gait. Quite naturally it experiences greater loads with running, jumping and climbing hills.[3]

Mechanism of Injury

Repetitive kicking (direct impact on the toe joint) or perpetual squatting challenges the joint excessively and can be incriminated in the development of early arthritis.[4]

It should be stated again the degree of wear of the joint (degeneration/arthritis) does not always coincide with the symptoms of the patient.[5,6,7]

Incidence and Prevalence

Hallux rigidus is the most common arthritic condition affecting the feet and the second most common condition of the big toe, second only to hallux valgus (bunions).[2]

There is contradicting information regarding the gender distribution of hallux rigidus, with multiple studies suggesting an increased prevalence existing with females, while others cite a greater prevalence amongst males.

As with most arthritis conditions, the prevalence of hallux rigidus appears to increase with age.

It has been estimated that 10% of people aged 20-34 have evidence on x-ray of the condition.

Upwards of 44% of people over 80 years of age have radiographic features of hallux rigidus.[2,8]

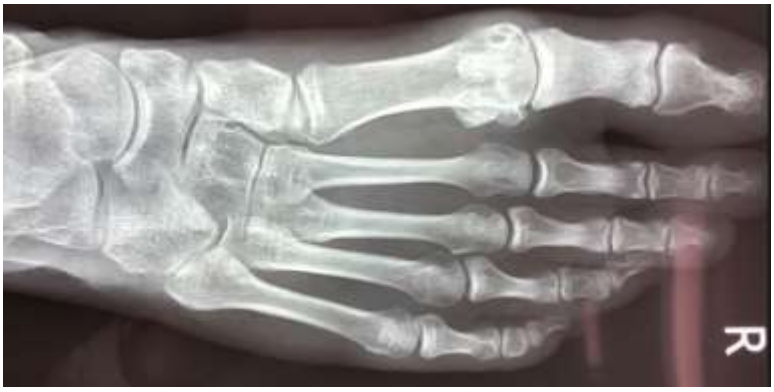


Figure 3 This x-ray of the right foot, taken from above, shows signs of arthritis in the joint between the big toe and the foot (first metatarsophalangeal joint). The narrow (black) space between the bones, compared to the other toes, indicates the cartilage is worn down. These x-ray changes are quite common.

It has been reported that for younger people the incidence of hallux rigidus increases with a history of hyperextension injuries (bending the toe upward too far) or by repeated stubbing of the big toe. This is controversial.

It was estimated that upwards of 39% of the population have hallux rigidus to some degree; the majority of the studies were making the diagnosis by x-ray alone. In upwards of 80% of cases of hallux rigidus, the problem is in both feet.

Key Points

- Arthritis of the big toe (1st MTP) is common
- It is not always painful
- Hallux Rigidus can run in families

Natural History of Hallux Rigidus and the Treatments

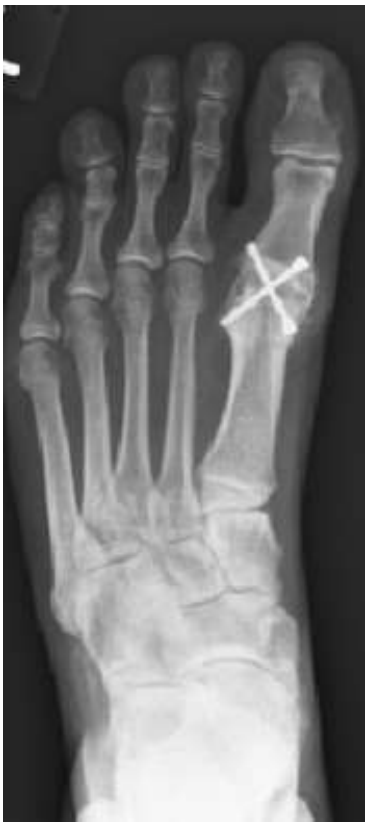
The natural course of this disorder - hallux rigidus - is loss of the articular cartilage of the joint, joint space narrowing and bone spur formation on the first metatarsophalangeal joint.[1] This may cause pain with activities sufficient to impair some activities of daily living. The symptoms can be adequately controlled by modification of footwear.[5,6]

Hallux rigidus has been shown to be manageable through non-surgical procedures. A study by Smith et al[6] found that although there was radiographic evidence of further degeneration of joint space, 90 percent of the patients surveyed stated that their pain had not changed 15 years after their diagnosis. The indication for surgical intervention with hallux rigidus is principally a marked disturbance of daily activities.

Hallux rigidus has been shown, as mentioned, to be manageable through non-surgical (conservative) procedures.
If conservative treatment measures do fail, then surgical interventions, such as fusion of the joint, are highly successful and are tailored for the individual patient.[5]

Figure 4

The x-ray at right shows surgery for painful hallux rigidus. Small screws are placed across the joint, creating a joint fusion. Movement is prevented, but pain is eliminated.



Summary

- Hallux rigidus is a chronic degenerative disorder of the big toe.
- 44% of patients over the age of 80 years have x-ray evidence of hallux rigidus.
- The degree of pain does not always coincide with the degree of degeneration within the joint.
- It remains unclear as to whether trauma or injury caused hallux rigidus.
- Hallux rigidus is effectively managed through conservative measures; in severe cases surgical interventions have been shown to improve symptoms and restore motion

Patient Profile #1

A 20-year-old male presents with pain and limitation of motion of the great toe. Physical examination reveals enlargement of the toe and reduced range of motion. The patient claims discomfort when the great toe is forcibly moved. X-rays reveal degeneration of the first metatarsophalangeal joint, compatible with osteoarthritis; i.e., hallux rigidus. The patient is markedly improved with the use of a rigid walking shoe.

Patient Profile #2

A 50-year-old male presents with pain and limitation of motion of the first metatarsophalangeal joint. X-rays reveal evidence of early osteoarthritis of the first metatarsophalangeal joint. Attempts at modification of his footwear does not improve his circumstance. He undergoes a surgery to fuse the joint (See Figure 4). This allows him to return to all activities without limitation.

References

1. Triveldi B, Marshall M, Belcher J, Roddy E. A systematic review of radiographic definitions of foot osteoarthritis in population-based studies. *Osteoarthritis & Cartilage*, 2010; 18:1027-1035.

2. Coughlin MJ, Shurnas PS. Hallux rigidus: demographics, etiology, and radiographic assessment. *Foot and Ankle Int*, 2003; 24(10):731-743.

3. Towers JD, Deible CT, Golla ST. Foot and ankle biomechanics. *Seminars in Musculoskeletal Radiology*. 2003; 7(1):67-74.

4. Riskowski J, Dufour AB, Hannan MT. Current musculoskeletal research on feet. *Curr Opin Rheumatol*. 2011; 23(2):148-155.

5. Zamet GV, Menz HB, Munteanu SE, Landorf KB, Gilheany MF. Interventions for treating osteoarthritis of the big toe joint. *Cochrane Database Syst Rev*. 2010; Sep 8;(9):CD007809

6. Smith RW, Katchis SD, Ayson LC. Outcomes in hallux rigidus patients treated nonoperatively: A long-term follow-up study. *Foot and Ankle Int*, 2000; 21(11):906-913.

7. Gilheany MF, Landorf KB, Robinson P. Hallux valgus and hallux rigidus: a comparison of impact on health-related quality of life in patients presenting to foot surgeons in Australia. *Journal of Foot and Ankle*, 2008; 1(1):14-19.

8. Gould N, Schneider W, Ashikaga T. Epidemiological survey of foot problems in the continental United States, 1978-1979. *Foot and Ankle*, 1980; 1(1):9-10.

DEGENERATIVE DISC DISEASE

Introduction

Degenerative disc disease is one of the most common causes of low back pain and neck pain.[1]

More than 80% of people will experience an episode of back pain at some point in their lifetime that interferes with work and other activities.[2]

Degenerative disc disease refers to symptoms of back or neck pain caused by wear and tear of the intervertebral disc.

Most commonly degenerative disc disease consists of low-level pain with occasional episodes of more severe pain.

Despite what the name suggests, degenerative disc disease is not a disease but a condition in which the natural, age-related wear on a disc causes discomfort and other symptoms.[3]

Approximately 30% of adults will show some evidence of disc degeneration by the time they are 35 years of age, and almost all people will show disc degeneration by the time they are 65 years old.[2]

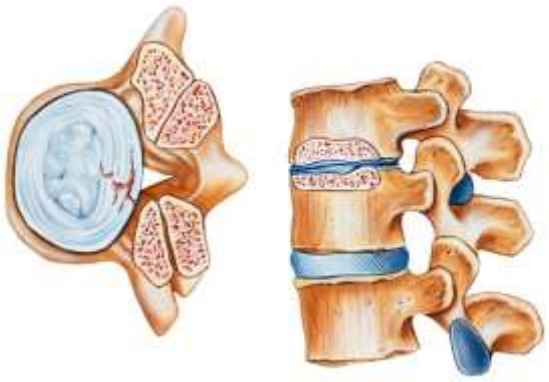


Figure 1 The discs of the spine begin to degenerate early in life, with small tears in the outer part of the disc.

This condition usually does not result in long-term disability, and most causes can be managed using non-surgical treatment methods. While it is true that disc degeneration is likely to progress over time, the pain from degenerative disc disease usually does not get worse.[4]

Key Points

- Disc degeneration is a slow process that develops over many years.
- Almost all people have some amount of disc degeneration after age 35.
- Disc degeneration does not mean progressive deterioration in function.

Anatomy

The spine consists of bones called vertebrae which are connected by ligaments and discs. Soft tissues about the spine - including muscles, tendons and ligaments control movement of the spine. A pair of vertebrae and the disc between them is referred to as a motion segment.[5]

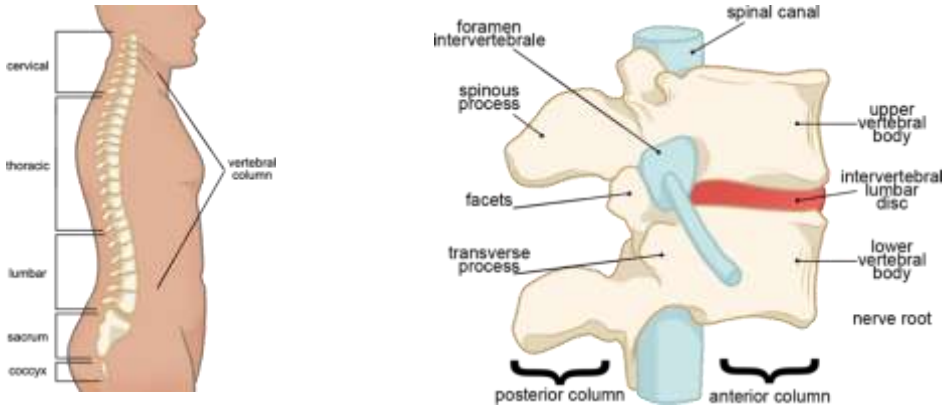


Figure 2 The spine is divided into different regions; the cervical (neck), thoracic (chest/back) and lumbar (low back) regions are the mobile areas of the spine. Pairs of vertebrae are firmly attached together by the intervertebral disc, plus ligaments, tendons and muscles. A pair of vertebrae with their disc between is called a motion segment.

The discs, because they are between two vertebrae, are called intervertebral discs. Discs act like spacers and shock absorbers, and are flexible enough to allow movement. The intervertebral disc is made up of three portions:

1. A tough outer ring called the annulus fibrosis;
2. An inner, gel-like portion called the nucleus pulposus;
3. An end plate that attaches to the bones above and below the disc.

Adjacent to the discs are two joints called the facets. These joints dictate the direction and the amount of movement possible between the two vertebrae.[5]

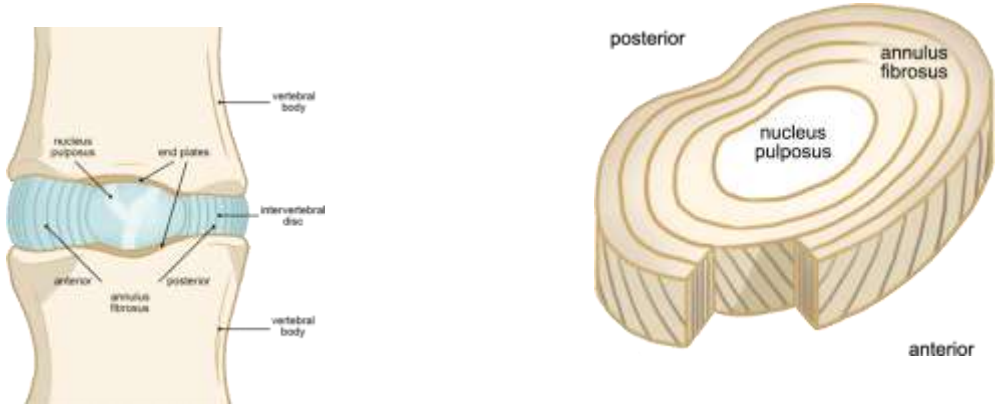


Figure 3 The disc has a tough outer ring, made of layers, called the annulus fibrosis. This ring surrounds and contains the nucleus pulposus.

Key Points

- Discs allow spinal motion and provide stability.
- Discs attach to adjacent vertebrae.
- Discs are responsible for 25% of the height of the spinal column.
- Discs have three parts, annulus fibrosis (on the outside); the nucleus pulposus (in the centre); and the end plate which attaches to the vertebrae.

Function

The intervertebral discs are fibrocartilaginous cushions which function as shock absorbers and stabilizers.[5,6]

The disc absorbs the forces that compress vertebrae together.

Individual discs have very limited movement, which is governed by the supporting muscles, tendons and ligaments. As the discs are compressed, the nucleus pulposus pushes outwards and upwards. The movement of the nucleus pulposus will be dictated by whether the spine is bent forward, backwards or to the side.

The nucleus pulposus and the surrounding annulus fibrosis are very different anatomically but they have an efficient functional relationship. Over time and with daily challenges, the intervertebral disc loses some of its water and thus becomes less able to take load.[7]

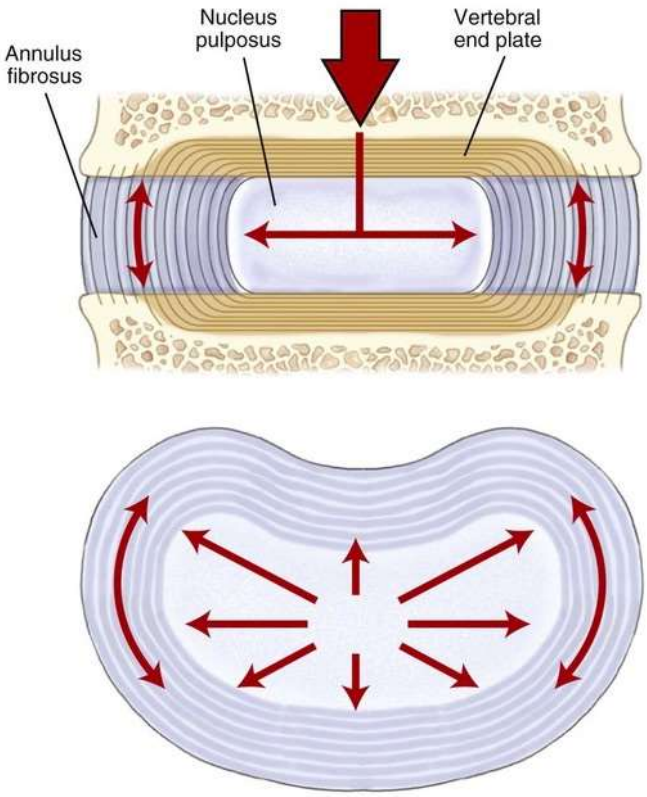


Figure 4 The two parts of the disc work together to resist compressive forces on the spine

Key Points

- Discs resist compression with loading.
- The annulus fibrosis part of the disc is more likely to be injured with twisting and bending.
- If the annulus fibrosis is damaged, the nucleus pulposus can press outward, resulting in a disc herniation.

Mechanism of Injury

Discs are generally injured during a combination of twisting and bending. Repeated twisting and bending can wear out the casing; i.e., annulus fibrosis.[8]

A combination of disc aging and repeated loading can lead to structural changes in the disc and eventual further degeneration of the disc structure. When there are fissures (splits) in the annulus fibrosis, a herniation of the nucleus pulposus can occur.[9,10]

Such a disc herniation may be fairly small and thus the nucleus pulposus remains contained.

If the split is larger, then the nucleus pulposus can escape, putting pressure on the adjacent nerve root.

The nerve root is called the sciatic nerve and thus the clinical picture is said to be sciatica.[11]

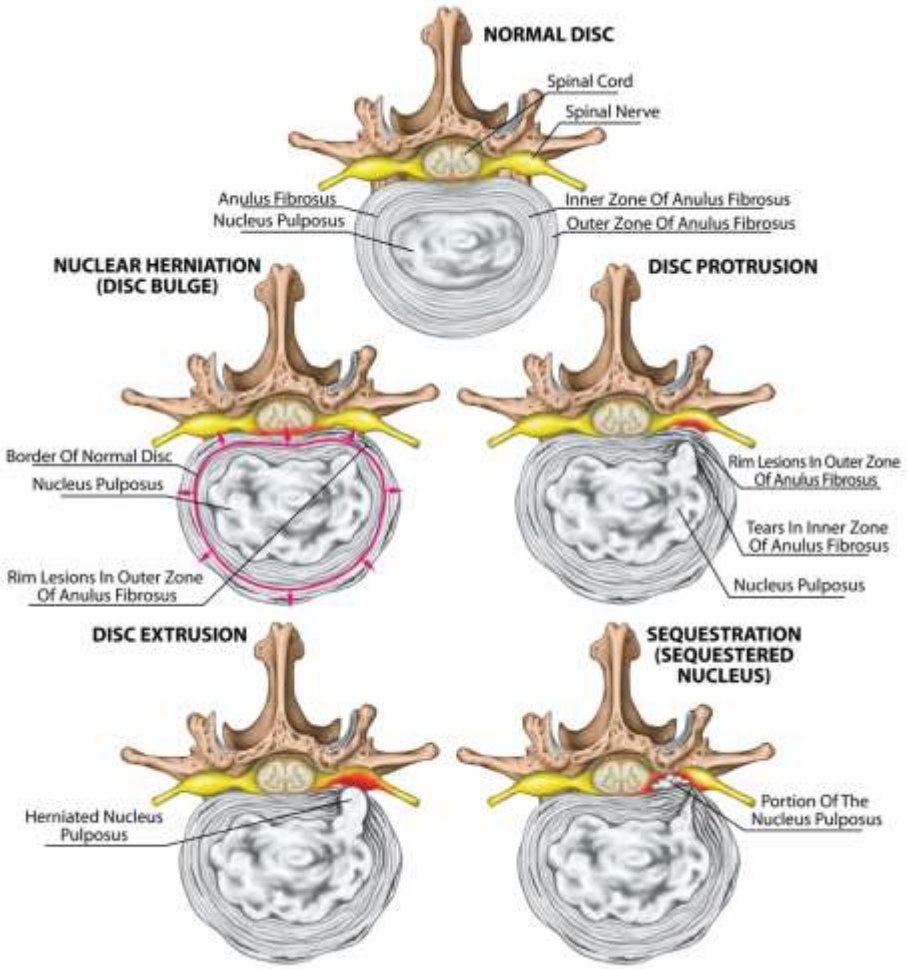


Figure 5 The outer rings of the disc can weaken and 'sprain', allowing the inner nucleus pulposus to stretch the annulus fibrosis (disc bulge, disc protrusion). This can progress to disc extrusion if the nucleus pulposus migrates through the injured outer part of the disc.

Key Points

- Discs can be injured with bending and twisting. If a frank disc herniation or rupture occurs, it can put pressure on the adjacent sciatic nerve.

Risk Factors for Low Back Pain

The following factors may play a role[12]:

1. Excess body weight.
2. Occupations with prolonged sitting, coupled with vibration - truck drivers.
3. Family history/genetics.

Types of Disc Injuries

1. Acute disc injuries (less than one month).

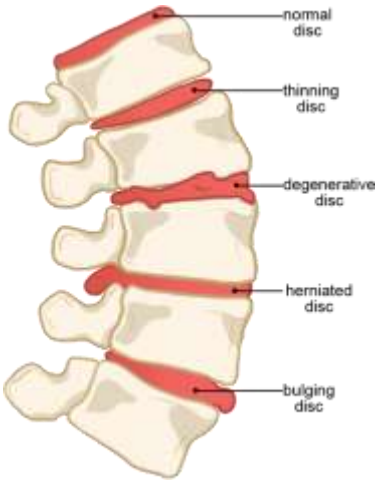
Sprains of the lower back are very common and simple injuries.[13]

This usually causes discomfort in the lower back and resolves after a few days or a week or two. Any activity that increases pressure on the intervertebral disc can cause low back pain; this includes coughing, sneezing, lifting heavy objects, bending and twisting.[14,15] These disc injuries can occur in younger people, as well as those who are middle-aged.

They are normally self-limiting and resolve without long-term implications.

Figure 6

Different stages of disc degeneration and damage can occur at different levels of the spine.



2. Subacute Disc Injuries (2-3 months)

In cases of early damage to the disc, small tears can occur in the annulus fibrosis. The recovery from this type of disc injury is somewhat longer (2-3 months). The individual presents with spasm of the back muscles, limitation of motion, but usually does not have sciatica (lack of nerve pressure). This type of injury is self-limiting and thus does not require extensive physiotherapy or more aggressive treatments such as nerve blocks.[16]

Key Points

- The outer layers of the disc may tear to varying degrees, like a sprained ligament.
- The more stretch on the annulus fibrosis, the more pain.
- Larger tears of the disc may allow the inner nucleus pulposus to migrate, causing nerve root pressure.

3. Cumulative Trauma - The Degenerative Process

Repeated wear and tear to the annulus fibrosis can lead to a change in the function of the intervertebral disc.[10]

The intervertebral disc likewise can change with age, such that the inner nucleus pulposus dries out.[1]

This makes the disc a less effective spacer and allows more movement between two vertebrae.

Both of these situations can be a source of back pain.

As we age, the spine stiffens and thus movement between vertebral segments is lessened in this process.[3]

As the intervertebral disc degenerates, the facet joints at that level of the spine accept increased load.

The facet joints, which are lined with cartilage, become more compressed. This increased stress (load) leads to osteoarthritis of that particular facet joint. These changes can be viewed on x-ray or MRI (see right) but are not necessarily a source of low back pain.[17]

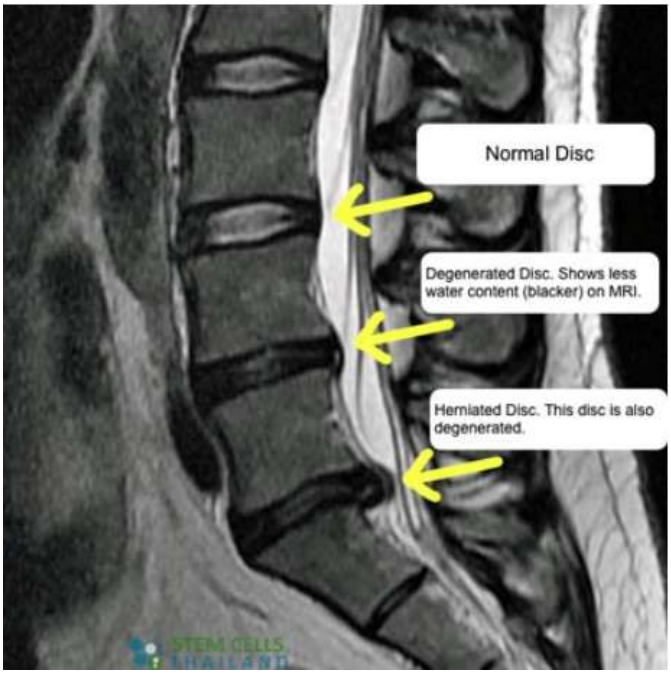


Figure 7
Magnetic Resonance Image of the lumbar spine showing disc degeneration

Key Points

- There is no standard definition of degenerative disc disease, rather, it denotes a process.
- The intervertebral discs change with age.
- In the process of degenerative disc disease, the intervertebral disc becomes thinner and the facet joints can develop secondary arthritis.

Natural History

The natural history of lumbar degenerative disc disease is relatively benign.[18]

While the disc degeneration may progress, symptoms such as low back pain do not necessarily get worse. If individuals are able to manage their back pain, whilst maintaining their function, the natural history is really quite favorable.

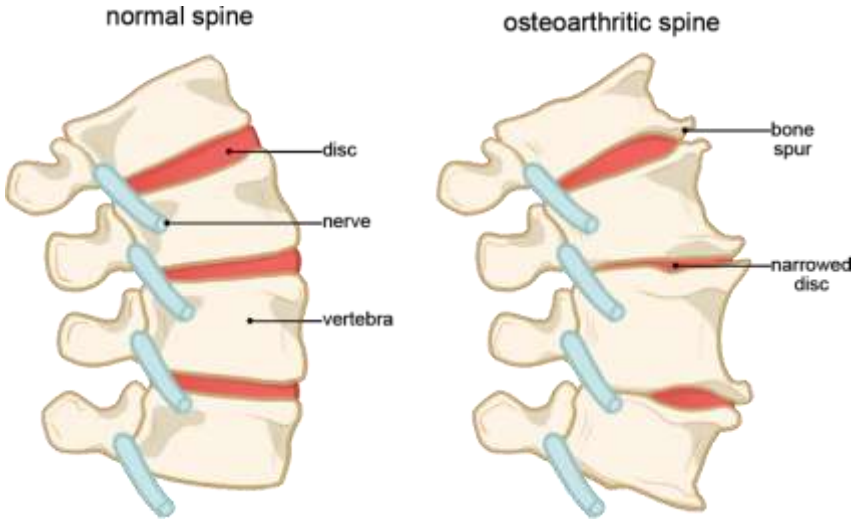


Figure 8
As discs degenerate over time, the space between vertebrae narrows, and osteoarthritis gradually develops through increased pressure on the bones.

Degenerative intervertebral disc disease usually goes hand in hand with facet joint osteoarthritis.

Certainly, as the individual moves beyond 40 years of age, the intervertebral disc thins and the load on the facet joint increases.

By definition, the facet joint develops osteoarthritis.

The degree of osteoarthritis and the individual's symptoms can vary widely.

In fact, someone who is 65 years of age is actually less likely to have low back pain as a consequence of degenerative disc disease - than someone who is 35 years of age.[10]

Most individuals with acute episodes of low back pain do not seek medical care because symptoms are often brief and self-limiting.

Within the first month, 82% of those off work return to gainful employment.[19]

It is noted that up to 30% of patients can report persistent back pain of a mild to moderate intensity for one year after an acute episode.[20]

Recurrences of pain are also common with upwards of 60% of patients experiencing at least one relapse within 12 months.[19,20]

Factors associated with the development of chronic disability due to low back pain include pre-existing psychological conditions, job dissatisfaction and disputes over compensation issues.[21]

Key Points

- Back pain usually resolves within 2-4 weeks.
- Most people have a subsequent episode of back pain.
- A small proportion, less than 7%, will develop chronic low back pain.

Treatments

Most cases of low back pain, secondary to degenerative disc disease, will recur in the future due to progressive changes in the disc.[3,4,20]

Emphasis should be placed on treatments that reduce the incidence or severity of future episodes.[22]

Tips for treatment to reduce future low back pain.

- Reassure the patient that the problem is not dangerous and the back is a strong structure.
- Explain degenerative disc disease (patient education is very important) is not really a disease.
- Explain that most people can prevent low back pain or minimize its symptoms.
- Emphasize an active approach involving exercise.

Conservative (Non-surgical) Treatment

At all stages, pain and muscle spasm can be managed symptomatically with physiotherapy modalities, mild medications and progressive activity.[23,24]

Surgical Treatment

Very few people with degenerative disc disease will require surgery. If there is evidence of progressive nerve deficit, then lumbar spine surgery would be a consideration.[25]

Key Points

- Treatment should always include exercise and activity.
- Treatment almost always includes patient education and an exercise program that is individualized to the patient.
- Traditional treatments, such as modalities and/or medications may help the patient reduce pain and muscle spasm. These treatments are often helpful, but not essential so may be considered optional.
- If surgery is required, it would be in very specific circumstances.

Summary

- Degenerative disc disease is a typical part of the aging process for most people.

- Symptoms and physical findings of degenerative disc disease vary with the age of the patient.

- Treatment of low back pain, without surgery, is commonly successful.

Patient Profile #1

A 26-year-old man complains of acute low back pain of two days following a week of bending and twisting on his job. He presents with spasm in his low back muscles, and difficulty bending forward. He feels better while he is walking. There is no evidence of any nerve root irritation.

This gentleman presents with a classic story of a small tear of the annulus fibrosis, allowing the central part of the disc to protrude backward, stretching the disc and causing back pain. There is a mild inflammatory response as part of the healing process that is causing the muscle spasm.

This patient is treated with anti-inflammatory medication, exercises aimed at reducing the stretch on the disc, and a temporary avoidance of activities that increase disc pressure. Once symptoms resolve, regular activities are re-introduced. He is back to work in three weeks.

Patient Profile #2

A 62- year-old female presents with recurrent episodes of low back pain over the past 20-30 years. The patient is overweight and there is x-ray evidence of degenerative disc disease at L4-L5 (see Figure 9 at right). There is no evidence of any neurological deficit. Her back pain is worse after standing and walking.

This patient suffers with mechanical low back pain, as a consequence of degenerative disc disease and facet joint osteoarthritis.

This situation is treated with education about degenerative disc disease, weight reduction, enhanced fitness and muscle strengthening and advice about postures to reduce pressure on the joints.

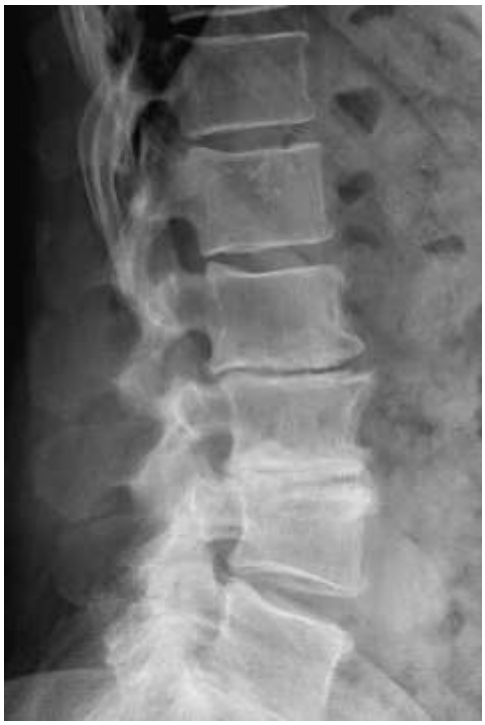


Figure 9
Degenerative changes between the 4th and 5th lumbar vertebrae are evident through the narrowing of the space between the vertebrae.

References

1. Adams MA, Roughley PJ. What is intervertebral disc degeneration, and what causes it? *Spine*, 2006; 31(18):2151-2161

2. Battie MC, Videman T, and Parent E. Lumbar disc degeneration. Epidemiology and genetic influences. Spine, 2004; 29(23):2679-2690

3. Prescher A. Anatomy and pathology of the aging spine. *European Journal of Radiology*. 1998;27(3):181–195.

4. Taher F, Essig D, Lebi DR, et al. Lumbar degenerative disc disease: current and future concepts of diagnosis and management. *Adv Orthop*, 2012; 2012: Article ID 970752, 7 pages doi:10.1155/2012/970752.

5. Bogduk N. *Clinical anatomy of the lumbar spine and sacrum*. Third Edition. Churchill Livingstone, 1997.

6. Waxenbaum JA, Futterman B. Anatomy, Back, Intervertebral Discs. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jan, 2019.

7. Urban J and Roberts S. Degeneration of the intervertebral disc. *Arthritis Res Ther*2003; 5:120

8. Ito K and Creemers L. Mechanisms of intervertebral disc degeneration/injury and pain: a review. *Global Spine*, 2013; 3(3):145-152

9. Boos N, Weissbach S, Rohrbach H, Weiler C, Spratt KF, Nerlich AG. Classification of age-related changes in lumbar intervertebral discs: 2002 Volvo Award in basic science. *Spine*, 2002;27(23):2631–2644.

10. Martin MD, Boxell CM, Malone DG. Pathophysiology of lumbar disc degeneration: a review of the literature. *Neurosurg Focus*, 2002; 13(2):1-6

11. Stafford MA, Peng P, Hill DA. Sciatica: a review of history, epidemiology, pathogenesis, and the role of epidural steroid injection in management. *BJA: British Journal of Anaesthesia*, 2007; 99(4):461–473

12. Lionel KA. Risk factors for chronic low back pain. *Journal of Community Medicine & Health Education*, 2014; 4(2):271

13. Bogduk N: The anatomy of the lumbar intervertebral disc syndrome. *Med J Aust.* 1976;1(23):878–81

14. Marshall LW, McGill SM. The role of axial torque in disc herniation. *Clin Biomech*, 2010; 25(1):6–9

15. Adams MA, Hutton WC. Prolapsed intervertebral disc. A hyperflexion injury 1981 volvo award in basic science. *Spine*, 1982;7(3):184–191

16. Karppinen J., Shen F.H., et al. Management of degenerative disk disease and chronic low back pain. *Orthop Clin North Am*, 2011; 42(4):513-28

17. Modic M et al, Acute low back pain and radiculopathy: MR imaging findings and their prognostic role and effect on outcome, *Radiology*, 2005; 237:597-604

18. Saal JA. Natural history and nonoperative treatment of lumbar disc herniation. *Spine*, 1996; 21(24S):2s-9s

19. Maetzel A, Li L. The economic burden of low back pain: a review of studies published between 1996 and 2001. *Best Practice & Research Clinical Rheumatology*, 2002; 16(1):23-30

20. Wasiak R, Pransky GS, Webster BS. Methodological challenges in studying recurrence of low back pain. *Journal of Occupational Rehabilitation*, 2003; 13(1):21-31

21. Wong AYL, Karpinnen J, Samartzis D. Low back pain in older adults: risk factors, management options and future directions. *Scoliosis and Spinal Disorders*, 2017; 12:14. doi: 10.1186/s13013-017-0121-3. eCollection 2017.

22. McGill S. *Low back disorders: evidence-based prevention and rehabilitation*. 2nd edition. Human Kinetics, 2007.

23. Delitto et al. Low Back Pain. Clinical Practice Guidelines Linked to the International Classification of Functioning, Disability, and Health from the Orthopaedic Section of the American Physical Therapy Association. *J Orthop Sports Phys Ther*. 2012; 42(4):A1-A57

24. Guideline for the evidence-informed primary care management of low back pain. 3rd edition. http://www.topalbertadoctors.org/download/1885/LBPguideline.pdf?_20180625085852

25. Sabnis AB, Diwan AD. The timing of surgery in lumbar disc prolapse: A systematic review. *Indian J Orthop.* 2014; 48(2):127–135

DEGENERATIVE DISC DISEASE OF THE CERVICAL SPINE

Introduction

Degenerative disc disease of the cervical spine is common, affecting virtually every individual over the age of fifty years to some degree.[1]

This condition is usually asymptomatic, with most individuals complaining of some stiffness with their neck movement, combined with crepitus (crackling).

Rarely, the degenerative disc disease of the cervical spine may become more severe which can lead to increased pain, limitation of movement and, infrequently, arm numbness.[2]

Anatomy and Pathoanatomy

As discussed within the earlier section on Degenerative Disc Disease of the Lumbar Spine, the anatomy of the intervertebral disc in the neck is similar and thus degeneration occurs through a similar process.

This includes thinning of the disc, which robs it of its ability to accept load.

As with the lumbar spine, the adjacent facet joints face increasing stress and start to develop osteoarthritis.

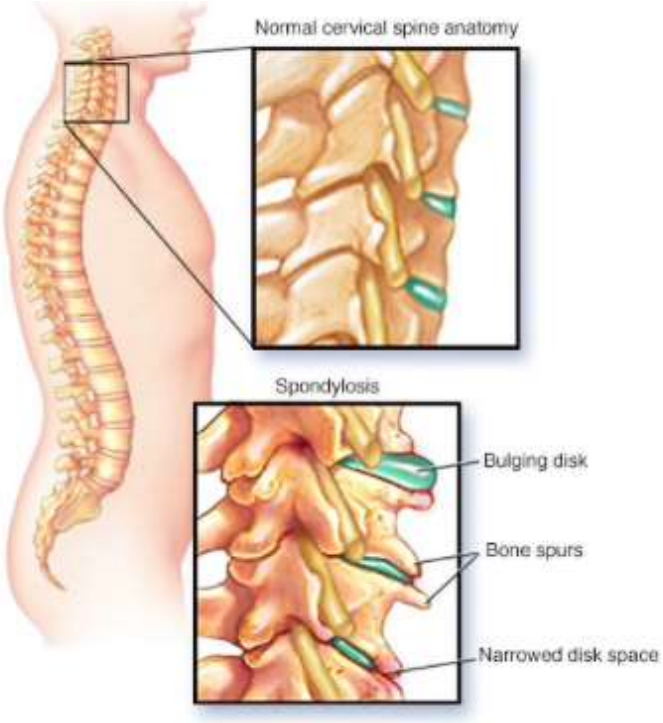


Figure 10 Degenerative changes in the cervical spine (spondylosis) involves narrowing of the disc space, leading to increased stress on the facet joint, creating osteoarthritis.

Incidence and Prevalence

Cervical degenerative osteoarthritis incidence varies with age. Population-based studies using magnetic resonance imaging (MRI) show nearly 100% of adults aged >40 years have moderate to severe degeneration of at least 1 cervical level (commonly C5/6).[3]

However, patients are usually asymptomatic even though cervical radiographs and MRI may show advanced, spontaneous degenerative disease.[3]

Neck pain is common in the general population. The lifetime prevalence for adults has been reported to range from 26% to 71%, the 1-year prevalence from 12% to 34% (4—8), and the daily prevalence from 10% to 22%[4,5]

In a systematic review by Haldeman et al[6], prevalence depended on the definitions used; for neck pain, the 1-year prevalence ranged from 30% to 50% in the general population. For neck pain with associated disability, the 1-year prevalence ranged from 2% to 11% in the general population, and from 11% to 14% in workers who reported being limited in their activities because of neck pain.

Risk Factors

Age is the most common risk factor for cervical degenerative disc disease. The condition is extremely common in patients who are middle-aged and older.

Other factors that may increase the risk for developing neck pain include:

- Genetics—a family history of neck pain
- Smoking—clearly linked to increased neck pain
- Depression or anxiety
- Previous injury or trauma to the neck or back

While these factors may increase the risk of neck pain, there is no scientific evidence that they cause degenerative changes in the cervical spine.[7,8]

Affect of Cumulative Load

Due to the fact that degenerative disc disease of the cervical spine is so prevalent, it is almost impossible for scientists to substantiate that certain occupations cause accelerated degeneration of the cervical spine.

Occupational physical activities have not been shown to be associated with increased cervical degenerative changes.[9,10]

Key Points

• Neck pain is common, with 30-50% of people experiencing some pain each year
• Cervical degenerative arthritis is present in most people after age 40 years old, and virtually everyone by 70 years old
• Occupational factors have not been linked to neck pain or cervical osteoarthritis

Symptoms

For most people, cervical spondylosis causes symptoms only occasionally, and these are self-limiting.

When symptoms do occur, they typically include pain and stiffness in the neck. This pain can range from mild to severe. It is sometimes worsened by looking up or looking down for a long time, or by activities in which the neck is held in the same position for a prolonged period of time—such as driving or reading a book. The pain usually improves with rest or lying down.

Other symptoms may include:

- Headaches
- Grinding or popping noise or sensation when you turn your neck (crepitus)
- Numbness/tingling and weakness in the arms, hands, and fingers
- Trouble walking, loss of balance, or weakness in the hands or legs
- Muscle spasms in the neck and shoulders

Management and Treatment

Most individuals afflicted with degenerative disc disease of the cervical spine and osteoarthritis respond favourably to a program of joint mobilization (manual therapy), strengthening exercise and mild medication.[11,12,13]

Rarely, surgery may be necessary for progressive neurological deficit.[14]

Summary

• Cervical degenerative disc disease/osteoarthritis is found in nearly all people

• Most people will occasionally have neck pain, but this is usually short-lived

• Education, joint mobilization and exercise will successfully treat most cases

• Occupational physical factors have not been linked to cervical osteoarthritis

Patient Profile

A 50-year-old woman presented with a three-month history of neck and right shoulder pain. She reported no pins and needles or weakness in her arm. She described that symptoms are worse when she first wakes up and at the end the day after knitting or reading.

She also reported that looking over her shoulder when reversing her car had become particularly difficult, as was using her computer at work.

She had visited her family doctor who advised non-steroidal anti-inflammatory medication as needed and he had also sent her for an X-ray. The X-ray confirmed cervical degeneration of the discs C3-C6 with mild osteophyte formation.

She was advised that physiotherapy was the best management at this stage and her symptoms were successfully resolved with education, manual therapy and exercise.

References

1. Michael J. Lee, K.Daniel Riew. The prevalence cervical facet arthrosis: an osseous study in cadaveric population. *The Spine Journal*. 2009; 9(9):711-714

2. Binder AI. Cervical spondylosis and neck pain. *BMJ*. 2007; 334(7592):527-531.

3. Matsumoto M, Fujimura Y, Suzuki N, et al. MRI of cervical intervertebral discs in asymptomatic subjects. *J Bone Joint Surg Br*. 1998; 80(1):19-24.

4. Bovim G, Schrader H, Sand T. Neck pain in the general population. *Spine* 1994; 19(12):1307-1309.

5. Côté P, Cassidy JD, Carroll L. The Saskatchewan health and back pain survey. *Spine* 1997; 23(15):1689-1698.

6. Haldeman S, Carroll L, Cassidey JD. Findings from the Bone and Joint Decade 2000 to 2010 Task Force on Neck Pain and Its Associated Disorders. *J Occup Environ Med*. 2010; 52(4): 424-427.

7. McLean SM, May S, Klaber-Moffett J, Sharp DM, Gardiner E. Risk factors for the onset on non-specific neck pain: a systematic review. *J Epidemiol Community Health*. 2010; 64(7): 565-572.

8. Singh S, Kumar D, Kumar S. Risk factors in cervical spondylosis. J Clin Orthop Trauma. 2014; 5(4):221-226.

9. Williams FM, Sambrook ON. Neck and back pain and intervertebral disc degeneration: role of occupational factors. *Best Pract Res Clin Rheumatol*. 2011; 25(1):69-79.

10. Palmer KT, Walker-Bone K, Griffin MJ, et al. Prevalence and occupational associations of neck pain in the British Population. *Scand J Work Environ Health* 2001;27(1):49-56.

11. Guzman J, Haldeman S, Carroll LJ, et al. Clinical practice implications of the Bone and Joint Decade 2000-2010 Task Force on Neck Pain and Its Associated Disorders: from concepts and findings to recommendations. *J Manipulative Physiol Ther*. 2009; 32(2 Suppl):S227-S243.

12. Blanpied PR, Gross AR, Elliott JM, et al. Neck Pain: Revision 2017. Clinical Practice Guidelines. *Journal of Orthopaedic & Sports Physical Therapy*. 2017; 47(7):A1–A83.

13. Gross A, Kay TM, Paquin JP, et al. Exercises for mechanical neck disorders. *Cochrane Database Syst Rev*. 2015;(1):CD004250.

14. Nikolaidis I, Fouyas IP, Sandercock PA, et al. Surgery for cervical radiculopathy or myelopathy. *Cochrane Database Syst Rev*. 2010;(1):CD001466.

PAIN

Introduction

Pain is a subjective response to a physical insult/injury.

It is normal and varies according to the degree of injury.

However, reported pain can vary widely from individual to individual.

Figure 1
The low back is a common site of pain

It has been recognized that psychological factors, amongst others, are intrinsically involved in the impact that pain has on an injured person.

The transition from acute pain to a chronic state is very concerning.

It is suggested that over 7 million Canadians suffer with Chronic Pain Syndrome.[1] This number is likely to increase in the future.

The introduction of opioid therapies to control chronic pain sufferers has done little to quell this explosion. In fact, the indiscriminate use of opioids may have potentiated the issue of chronic pain.[2]

The official definition of pain, as described by the International Association for the Study of Pain is an "unpleasant sensory and emotional experience associated with actual or potential tissue damage".[3]

As mentioned, interpretation of pain depends on the person's subjective experience. There are no objective measures of pain.

Anatomy and Physiology

Upon injury the painful stimulus acts on unique pain receptors called nociceptors.[4]

These receptors then transmit a signal to the central nervous system.

The central nervous system, upon alert, then transmits the alert to the brain.
As depicted in Figure 2, in the acute circumstance when a painful stimulus is experienced, there is a reflex withdrawal away from the painful stimulus. Depending on the degree of insult (whether mechanical, thermal, or chemical) the pain gradually subsides.

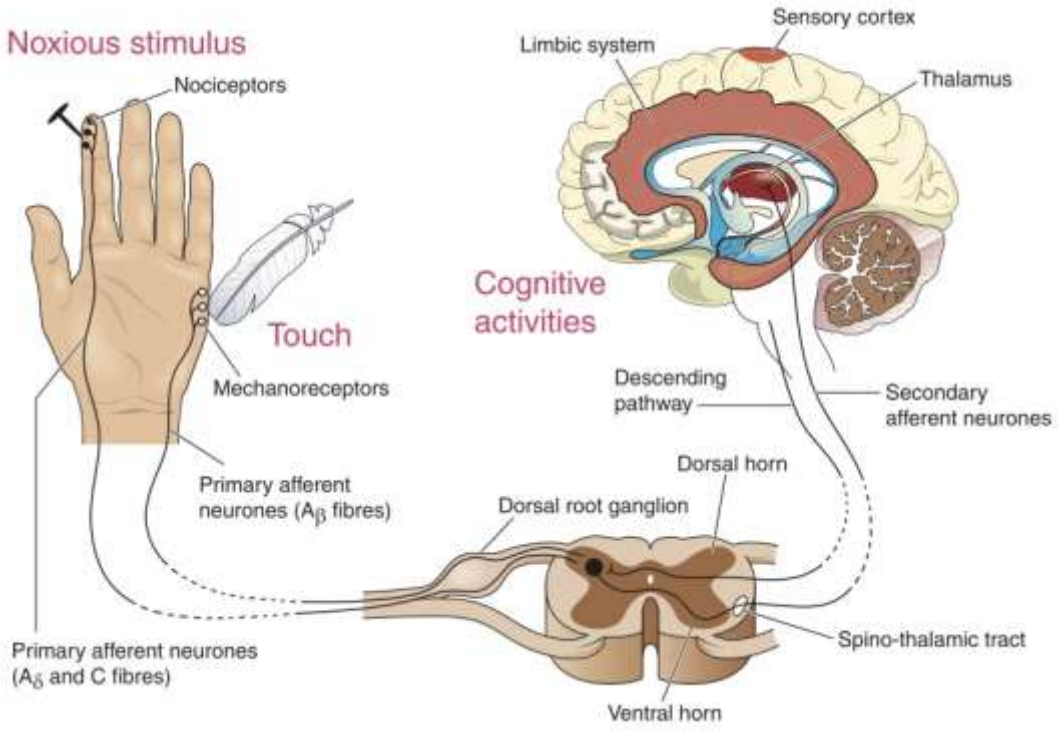


Figure 2 Different types of sensation are transmitted to the brain via the spinal cord. This includes noxious stimuli that activate nociceptors. This input is usually interpreted as pain by the brain.

Key Points

- Pain is a subjective experience.
- Acute pain is usually in response to an injury/insult.
- Acute pain is usually self-limiting.
- There are no objective measures of pain.

Chronic Pain Syndrome ~ Mechanism, Incidents and Prevalence

Chronic pain is defined as subjective pain lasting longer than 3 months.[3]

As mentioned, it is suggested that currently 7 million Canadians suffer with Chronic Pain Syndrome.

The development of chronic pain after an acute injury is poorly understood.

Frequently, the degree of objective evidence of tissue damage does not parallel the individual's complaints of chronic pain.

After an acute injury, it is unknown why some individuals develop chronic pain while others do not.[5]

Several studies have acknowledged co-morbidities that increase the likelihood of developing Chronic Pain Syndrome.[6-9]

These include a past history of depression, anxiety and poor social support. Added to that list have been chronic stress, obesity, smoking and catastrophic thinking.[6-9]

There is some suggestion that after an acute injury the pain receptors become more sensitive. Such studies have yet to be confirmed.

The Importance of Psychological Factors

The literature suggests that individuals that attend pain clinics, approximately 50% have psychological distress such as depression and/or anxiety.[10] Thus, treatment for psychological factors should be addressed when managing persistent pain.[11]

Figure 3 Chronic pain is intertwined with psychological factors, with each amplifying the effects of the other.

As noted in Figure 3, psychological distress increases the likelihood of developing Chronic Pain Syndrome and thus can fuel persistence of symptoms.

Key Points

- Pain lasting for longer than 3 months is classified as chronic pain.
- 50% of individuals seeking chronic pain management have psychological disorders.
- Treatment of psychological distress in patients suffering with chronic pain is vital.
- There are no objective measures of pain.

Natural History of Injury and Treatment

1) Low Back Pain

Low back pain is a very common medical condition.[12]

Approximately 80% of the Canadian population will develop low back pain at some point in their adult life.[9]

Most low back pain is mechanical, as the lower back has many pain sensitive areas.

These include the joints (facets), the supporting soft tissues and the intervertebral disc.

The lumbar spine is prone to develop pain after a relatively trivial insult.[13]

The majority of acute low back injuries get better within a few weeks.[14]

It is suggested in the literature that 20% develop persistent low back difficulties; i.e., pain.[15] This group is responsible for over 80% of costs related to low back pain.[15,16]

The group that develops acute low back pain that progresses to chronic discomfort are noted to have several pre-existing co-morbidities.[16]

These include: psychological conditions, poor general health - including obesity, and proclivity to catastrophize or exaggerate pain.[16,17]

Chronic low back pain can be effectively treated with exercise, psychological support and social strategies.[11]

Key Points

- Low back pain is common in the general population (80%).
- Acute low back sprains/strains resolve within a few weeks.
- Chronic low back pain is usually associated with several co-morbidities such as psychological issues, poor coping strategies and catastrophizing.

2) Carpal Tunnel Syndrome

Carpal Tunnel Syndrome, which is entrapment of the median nerve, is common.

The prevalence of Carpal Tunnel Syndrome in the general population is approximately 6% in women and 1% in men.[18]

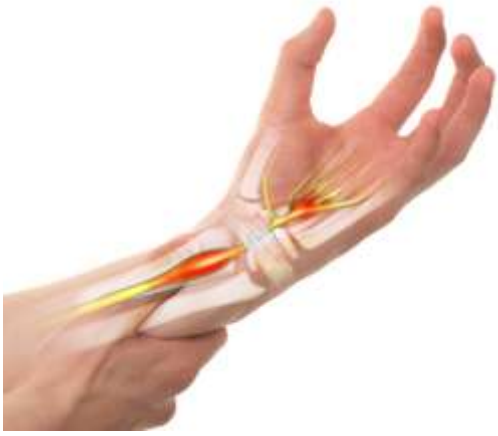


Figure 4
Pressure or tension on the front of the wrist can irritate the median nerve and cause pain in the wrist and hand

Carpal tunnel syndrome is usually triggered by repetitive activities of the hand and wrist.

Hypertrophy and swelling of the tissues about the wrist can compress the medial nerve. This causes burning, tingling, numbness and sometimes muscle weakness in the hand.

Conservative management includes splinting, physio (to control the swelling) and when ineffective, surgical options are available.

3) Neuropathies – Peripheral and Central

Neuropathies (of the peripheral nerves) are not common. They can cause pain, weakness and unusual sensations in the hands and feet.[19]

They ordinarily are a consequence of either diabetes[20,21] or alcohol consumption.[22] Rarely they can be drug induced.

Neurological conditions such as multiple sclerosis, stroke and spinal cord injury can also result in peripheral and central neuropathies.

Common symptoms include numbness, burning and/or loss of motor power.

With alcohol-induced neuropathy, the treatment of choice is discontinuing alcohol consumption.[22]

For diabetic neuropathy the treatment of choice is improved physical activity, ceasing smoking and paying very close attention to control of blood sugar levels.[23]

Summary

- Chronic low back pain is a common type of pain syndrome.
- Individuals suffering with chronic pain very commonly have associated co-morbidities.
- Psychological factors are common and must be carefully managed.
- The key to controlling and thwarting the development of Chronic Pain Syndrome is early intervention.

Patient Profile #1

A 38-year-old female office worker has a minor injury to her left elbow when she bumps her arm against a filing cabinet.

She states she had severe pain in her arm and hand. She reports the injury to her employer with whom she has a very poor rapport.

Initial x-rays and physical examination are normal.

She is started on a program of physiotherapy, which is basically passive modalities without emphasizing mobilization.

After two years, she continues to complain bitterly of pain about her left elbow and arm. She has been unable to return to the workplace.

On physical examination the arm is held in a protected position. The skin is glossy. The range of motion of the finger joints is reduced and the hair, as well as her fingernails have overgrown.

This is a classic story of an individual suffering with a chronic pain syndrome, secondary to a regional dystrophy following an episode of minor trauma.[25]

Patient Profile #2

A 48-year-old mechanic injures his neck, upper back and lumbar spine in a low energy motor vehicle collision. He initially complained of pain in his neck, shoulders and lower back, which failed to respond to physiotherapy, medications and absence from the workplace.

He is started on a program of opioids and encouraged to stay away from exercises. 18 months later, he remains unemployed and is now treated for depression. All investigations are normal.

This is a classic story of a person with chronic low back pain. There is no objective evidence of disease.[26]

References

1. Schopflocher D, Taenzer P, and Jovey R. The prevalence of chronic pain in Canada. *Pain Res Manag*, 2011; 16(6): 445-450.

2. Canadian Guideline for Safe and Effective Use of Opioids for Chronic Non-Cancer Pain. Canada: National Opioid Use Guideline Group (NOUGG); 2010 [cited April 20, 2019]. Available from: http://nationalpaincentre.mcmaster.ca/opioid/

3. International Association for the Study of Pain. 2011. Available at: https://www.iasp-pain.org/Education/Content.aspx?ItemNumber=1698; accessed April 20, 2019.

4. Yam MF, Loh YC, Tan CS, et al. General pathways of pain sensation and the major neurotransmitters involved in pain regulation. *Int J Mol Sci*, 2018; 19(8):2164.

5. Marcus DA. *Chronic Pain: A primary care guide to practical management.* 2nd Ed. New York: Humana Press; 2009.

6. Gureje O, VonKorff M, Simon GE, Gater R. Persistent Pain and Well-Being: A World Health Organization study in primary Care. *JAMA: The Journal of the American Medical Association*, 1998; 280(2):147-151.

7. Voscopoulos C, Lema M. When does acute pain become chronic? *British Journal of Anaesthesia*, 2010; 105(Suppl 1):i69-85.

8. Torrance N, Smith BH, Bennett MI, Lee AJ. 2006. The Epidemiology of Chronic Pain of Predominantly Neuropathic Origin. Results from a General Population Survey. *The Journal of Pain: Official journal of the American Pain Society*, 2006; 7(4):281-289.

9. Elliott AM, Smith BH, Penny KI, Smith WC, Chambers WA. The Epidemiology of Chronic Pain in the Community. *Lancet*, 1999; 354(9186):1248-1252.

10. Tunks ER, Crook J, Weir R. Epidemiology of chronic pain with psychological comorbidity: prevalence, risk, course, and prognosis. *Can J Psychiatry*, 2008; 53(4):224-234.

11. Linton SJ and Shaw WS. Impact of psychological factors in the experience of pain. *Phys Ther*, 2011; 91(5):700-711.

12. Manchikanti L. Epidemiology of low back pain. *Pain Physician*. 2000; 3(2):167-192.

13. Waddell G. *The Back Pain Revolution*, 2nd ed. Edinburgh: Churchill Livingston, 2004.

14. Pengel LH, Herbert RD, Maher CG, Refshauge KM. Acute Low Back Pain: Systematic review of its prognosis. *BMJ*. 2003; 327(7410):323.

15. Chou R, Shekelle P. Will this patient develop persistent disabling low back pain? *JAMA: the Journal of the American Medical Association*, 2010; 303(13):1295-1302.

16. Heitz CA, Hilfiker R, Bachmann LM, et al. 2009. Comparison of risk factors predicting return to work between patients with subacute and chronic non-specific low back pain: Systematic Review. *European Spine Journal*, 2009; 18(12):1829-1835.

17. Diamond S, Borenstein D. Chronic Low Back Pain in a Working-Age Adult. Best Practice & Research. *Clinical Rheumatology*, 2006; 20(4):707-720.

18. Bickel KD. Carpal tunnel syndrome. *J Hand Surg*, 2010; 35(1):147-152.

19. Cooper G, Eichhorn G, Rodnitsky RL. Peripheral Neuropathy. In: Conn PBM, editor. *Neuroscience in Medicine*, 3rd ed. New Jersey: Humana Press, 2008.

20. Dyck PJ, Kratz KM, Karnes JL, et al. The prevalence of staged severity of various types of diabetic neuropathy, retinopathy, and nephropathy in a population-based cohort: The Rochester Diabetic Neuropathy study. *Neurology*, 1993; 43(4):817-824.

21. Veves A, Backonja M, Malik RA. Painful Diabetic Neuropathy: Epidemiology, natural history, early diagnosis and treatment options. *Pain Medicine*, 2008; 9(6):660-674.

22. Chopra K and Tiwari V. Alcoholic neuropathy: possible mechanisms and future treatment possibilities. *Br J Clin Pharmacol*, 2012; 73(3):348-362.

23. Fields H, Baron R, Rowbotham M. Peripheral Neuropathic Pain: An approach to management. In: Wall P, Melzack R, editors. *Textbook of Pain*, 4th ed. Toronto, Ontario, Canada: Churchill Livingston; pp 1523-1533.

24. Watson JC and Sandroni P. Central neuropathic pain syndromes. *Mayo Clin Proc*, 2016; 91(3): 372-385.

25. Bruehl S. Complex regional pain syndrome. *BMJ*, 2015;351:h2730.

26. O'Sullivan P. It's time for a change with the management of chronic non-specific low back pain. Editorial in *Brit J Sports Med*, 2010; 46(4):224-227.

CHONDROMALACIA PATELLA AND PATELLOFEMORAL OSTEOARTHRITIS

Introduction

Patellofemoral osteoarthritis is a common cause of anterior knee pain, and is similar to other forms of osteoarthritis that develop gradually over time.[1]

Patellofemoral osteoarthritis can accompany tibiofemoral (knee) arthritis, or may exist as an isolated entity.[2]

Arthritis affects the patellofemoral joint less frequently than other parts of the knee. The characteristics of this isolated arthritis remain poorly understood, with few references in the literature.[3]

Patellofemoral pain (PFP) typically presents as diffuse anterior knee pain, usually with activities such as squatting, running, stair ascent and descent. It is common in active individuals across the lifespan. It is not known whether young people with patellofemoral pain will develop arthritis.[1]

Patellofemoral pain and patellofemoral arthritis are not synonymous; in other words, one can have pain without arthritis and vice versa.

Chondromalacia patella refers to any disorder of the cartilage covering the patella, and may eventually lead to osteoarthritis.[2]

Anatomy and Pathoanatomy

The patellofemoral joint is the joint between the patella (kneecap) and the femur (thighbone). It depends for stability partially on ligaments and other connective tissues, and on the quadriceps muscle at the front of the thigh. The stability of the joint is also influenced by lower limb alignment including varus/valgus knee alignment and rotation of the hip and foot.[1]

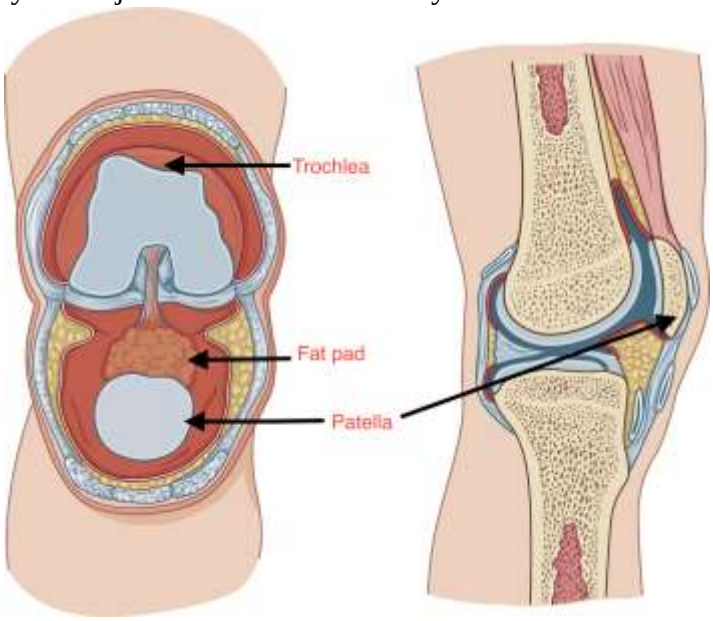


Figure 1
Anatomy of the patellofemoral joint. The left image shows a front view of the knee, with the patella folded down to show the groove on the femur (trochlea) that forms one half of the joint. The image at right shows a side view of the joint.

In addition, the relation of the knee to the position of the pelvis deserves consideration. Strength of hip and thigh muscles is another important stabilizer for the knee. Therefore, patellofemoral joint abnormality can be associated with one or a combination of these factors.[4]

The patella sits in a shallow groove on the femur (trochlea, see Figure 1), and its position is easily influenced by the factors noted above. This can lead to excessive pressure being placed on one part of the patella, rather than being evenly distributed across the entire patellofemoral joint. This excessive pressure leads to breakdown in the articular cartilage of the joint – osteoarthritis.

The relationship between abnormal joint structure and pain is imprecise, and thus highly individual.[5]

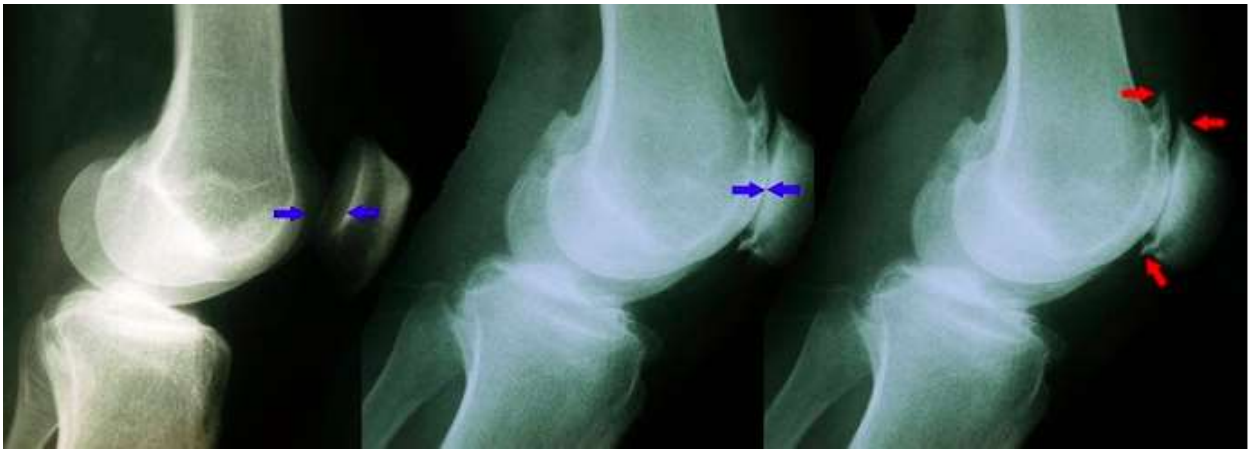


Figure 2
An X-ray of the knee from the side showing the patellofemoral joint. In a healthy joint (left image), there is a space between the patella and femur – this space represents the articular cartilage of the joint. In the middle image, the cartilage has deteriorated and the bones contact each other and are much closer together. Bony changes such as bone spurs (right image) also can form.

Key Points

- The back of the patella is covered with articular cartilage that matches the cartilage of the groove on the femur (trochlea)
- Anything that moves the patella from its balanced position in the trochlea can lead to abnormal pressure on cartilage and eventually cause arthritis
- A variety of factors can lead to malpositioning of the patella
- Changes in pressure distribution at the joint may result in cartilage deterioration

Clinical Manifestation

Anterior (front) knee pain is a common complaint among patients with patellofemoral problems. The pain is aggravated by anything that puts increased pressure on the patellofemoral joint. This includes stair ascending and descending, hill climbing, standing from a seated position, and kneeling or squatting. Often, patients will have little or no difficulty walking on level surfaces.[1-4]

As the cartilage deteriorates, there is increased friction in the joint, which may lead to a feeling of 'roughness' during knee movements, this is called *crepitus*. Some patients complain of stiffness of the knee, especially first thing in the morning or after sitting for long periods.[5]

Patients will typically have difficulty sitting for long periods with the knees bent.
Patients may have some mild swelling in the knee.

Many people have patellofemoral osteoarthritis without symptoms.[6]

Incidence and Prevalence

Patellofemoral pain (PFP) is common in young adolescents, with a prevalence of 7–28%, and incidence of 9.2%.[7]

Few studies have evaluated prevalence or incidence of patellofemoral pain in adult populations, except in the military, where the annual incidence in men is 3.8% and in women is 6.5%, with a prevalence of 12% in men and 15% in women.[7]

It appears that adolescents who experience patellofemoral pain may be more likely to develop arthritis in the joint in later life. It has been suggested that an individual may be 7.5 times more likely to develop patellofemoral joint osteoarthritis if they have suffered from adolescent anterior knee pain, though this remains to be clearly established.[8]

Previous trauma to the knee can lead to patellofemoral osteoarthritis. Post-traumatic etiology (including fractures, excluding patellar dislocations) accounted for 9 % of a large cohort of patients with isolated PF osteoarthritis, and is quite common after ligament surgery.[7,9]

Injuries can include:

- direct blows, such as falling on the knee
- patellar dislocation
- patellar fracture
- anterior cruciate ligament injuries

Experiencing a patellar dislocation increases the likelihood of development of patellofemoral osteoarthritis up to three-fold.[10]

Isolated patellofemoral arthritis is not rare, though less common than tibiofemoral (knee) osteoarthritis.[2]

Patellofemoral osteoarthritis is generally asymptomatic.[4]

Radiographic (x-ray) evidence of patellofemoral osteoarthritis can be observed in 17.1-34% of female patients and 18.5-19% of male patients in the age of ≥55 or ≥60 years old according to some studies.[2]

Noble and Hamblen[11] reported patellofemoral osteoarthritis in 79% of 100 cadavers aged ≥65 years.

Evidence from a study including participants aged ≥50 years with knee complaints suggests that osteoarthritis in the knee starts in the patellofemoral joint and subsequently progresses to the tibiofemoral (knee) joint[12]. Lankhorst et al[13] followed 706 participants over time, 116 (16.4%) had isolated patellofemoral osteoarthritis.

Key Points

- Osteoarthritis in the knee frequently includes the patellofemoral joint
- Patellofemoral pain early in life may increase the likelihood of developing patellofemoral osteoarthritis
- Patellofemoral osteoarthritis is frequently asymptomatic

Risk Factors

Like other forms of osteoarthritis, genetics, age, gender and weight appear to play a role in the development of patellofemoral arthritis.[14]

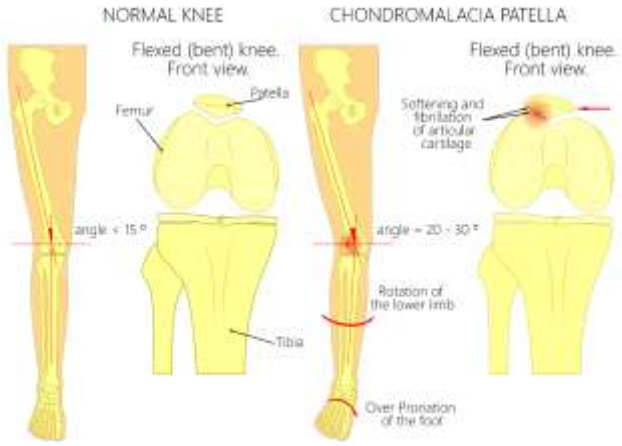
Patellofemoral osteoarthritis is more common in females.[1,14]

Increased body weight increases the pressure on the patellofemoral joint and can lead to increased cartilage wear.[15]

Alignment issues in the pelvis, hips, legs and feet can lead to increased pressure on parts of the patellofemoral joint, starting the degenerative process.[16,17]

As noted above, previous knee injury may increase the risk of patellofemoral osteoarthritis.[11,14]

Figure 3
Alignment issues of the legs can cause abnormal pressure distribution at the patellofemoral joint, leading to chondromalacia ('sick cartilage') on the back of the patella.



Natural History

It has been proposed that patellofemoral pain at younger ages may be a precursor to later patellofemoral osteoarthritis; however, this has not yet been established clearly due to a lack of long-term longitudinal studies.[1,18]

Patellofemoral pain in young people has long been regarded as a benign and self-limiting condition. However, this may not be accurate, as the proportion of those reporting later chronic knee problems varies widely, from 20% after one-year follow-up to 91% after 18 years.[8,10,13]

While no current studies have prospectively studied people with PFP through to the development of patellofemoral osteoarthritis (and thus verified this relationship), individuals undergoing arthroplasty (surgery) for PFOA were more than twice as likely to report having had patellofemoral pain as an adolescent than patients undergoing an arthroplasty for isolated tibiofemoral (knee) osteoarthritis.[18,19]

Radiographic and MRI patellofemoral osteoarthritis features were evident in 20-30% of adults aged 26-50 years old with persistent patellofemoral pain (chondromalacia patella), with greater prevalence observed in those who were older, female, or with higher BMI.[21]

More than half of participants with patellofemoral pain report an unfavourable recovery 5–8 years after onset, but do not have radiographic knee OA. Longer pain duration and worse pain scores predict a worse outcome over time.[13]

Nimon et al[21] followed a series of adolescent girls with anterior knee pain, known as chondromalacia patella, and found that about one in four continued to have significant symptoms at 16-20 years after onset, based on clinical findings.

In patients who do develop patellofemoral osteoarthritis, the progression of joint changes and clinical symptoms over time is not clear. Patellofemoral osteoarthritis progression over 3 years, defined by magnetic resonance imaging (MRI), occurred in 15.5% of subjects.[22]

Key Points

- It remains unclear how many patients with anterior (patellofemoral) knee pain – so-called chondromalacia patella - will develop osteoarthritis.
- It is emerging that there are different causes for patellofemoral pain, which may affect progression to osteoarthritis.
- The physical demands placed on the patellofemoral joint are the most likely reason for patients continuing to experience pain.

Treatment

Treatment for patellofemoral osteoarthritis is similar to that for other forms of osteoarthritis. Patients are advised to lose weight (if necessary) and to use medications such as acetaminophen and non-steroidal anti-inflammatories (NSAIDs) to control pain.[1,2]

A conservative treatment program for patellofemoral pain and/or patellofemoral osteoarthritis will typically involve a combination of:

- strengthening exercises for the hip, knee and abdominal muscles
- balance exercises
- aerobic exercise such as walking, swimming or skipping rope
- mobility (manual) therapy of the patellofemoral and knee joints

Exercise is important for patients with patellofemoral arthritis, with strengthening of the outside hip muscles (abductors) and front thigh muscles (quadriceps) being especially important.

Alignment of the knee joint also is important. It appears that even a small shift in the position of the patella can relieve pain. Changes in alignment can involve the use of foot orthotics in those with flat feet, and, occasionally, braces that keep the patella in position. Taping of the patella is frequently used to enable patients to perform exercises without pain.[23]

Patients also can decrease the pressure on the patellofemoral joint by using higher seats to avoid pressure on the joint when standing from sitting.[1]

Younger patients with patellofemoral pain (chondromalacia patella) often have ongoing or recurring symptoms, which suggests that treatment may need to be ongoing or delivered at regular intervals.

Surgery is seldom indicated for early patellofemoral pain or osteoarthritis, severe pain and degenerative changes in older individuals may be treated with partial or complete knee arthroplasty (replacement).[2]

Summary

- Patellofemoral pain is a fairly common knee problems that may progress to patellofemoral osteoarthritis in about 15-25% of patients

- Symptoms are dependent on the physical demands placed on the joint

- Many patients are asymptomatic with appropriate exercise and activity modification

- Patellofemoral pain is an ongoing problem that may require regular treatment

- Similar to other forms of osteoarthritis, surgery is required for only a small percentage of patients

Patient Profile

Mrs. Jones, 55-years-old, has a 3-year history of progressively worsening pain in both knees. Her knees are stiff for about 20 minutes when she first arises in the morning and for a few minutes after getting up from a chair during the day. She has difficulty walking > 30 minutes because of pain, and her symptoms are exacerbated by kneeling, squatting, or descending stairs.

Mrs. Jones was slightly obese, and physical examination of the lower extremities revealed mild genu varum. Patellar facet tenderness was determined by palpation.

Mrs. Jones had moderate bilateral knee osteoarthritis, including the patellofemoral joint. She was educated about various pharmacologic and lifestyle modifications that may result in better control of her osteoarthritis pain.

The importance of maintaining a regular exercise program to maximize aerobic conditioning, strengthen her muscles and increase caloric expenditure was stressed.

Mrs. Jones was referred to a physiotherapist for assistance with developing an appropriate exercise program.

Mrs. Jones is still receiving conservative care and is doing well.

References

1. Crossley KM, Callaghan MJ, van Linschoten R. Patellofemoral pain. *Br J Sports Med*. 2016; 50(4):247-250.

2. Kim Y-M, Joo Y-B. Patel. Patellofemoral osteoarthritis. *Knee Surg Relat Res*. 2012; 24(4):193-200.

3. Crossley KM, Hinman RS. The patellofemoral joint: the forgotten joint in knee osteoarthritis. *Osteoarthritis Cartilage*. 2011; 19(7):765-767.

4. Crossley KM, Stefanik JJ, Slefe J et al. 2016 Patellofemoral pain consensus statement from the 4th International Patellofemoral Pain Research Retreat, Manchester, Part 1: Terminology, definitions, clinical examinations, natural history, patellofemoral osteoarthritis and patient-reported outcome measures. *Br J Sports Med*. 2016; 50(14):839-843.

5. Schiphof D, van Middlekoop M, de Klerk BM et al. Crepitus is a first indication of patellofemoral osteoarthritis (and not of tibiofemoral arthritis). *Osteoarthritis Cartilage*. 2014; 22(5):631-638.

6. Stefanik JJ, Gross KD, Guermazi A et al. The relation of MRI-detected structural damage in the medial and lateral patellofemoral joint to knee pain: the Multicenter and Framingham Osteoarthritis Studies. *Osteoarthritis Cartilage*. 2015; 23(4):565-570.

7. Smith BE, Selfe J, Thacker D et al. Incidence and prevalence of patellofemoral pain: A systematic review and meta-analysis. *PLoS One*. 2018; 13(1): e0190892. https://doi.org/10.1371/journal. pone.0190892.

8. Eijkenboom JFA, Waarsing JH, Oei EHG, Bierma-Zeinstra SMA, van Middelkoop M. Is patellofemoral pain a precursor to osteoarthritis? Patellofemoral osteoarthritis and patellofemoral pain patients share aberrant patellar shape compared with healthy controls. *Bone Joint Res.* 2018;7(9):541–547.

9. Olestad BE, Holm I, Engebretsen L et al. The prevalence of patellofemoral osteoarthritis 12 years after anterior ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc.* 2013; 21: 942-949.

10. Conchie H, Clark D, Metcalfe A, Eldridge J, Whitehouse M. Adolescent knee pain and patellar dislocations are associated with patellofemoral osteoarthritis in adulthood: A case control study. *Knee.* 2016; 23(4):708-11

11. Noble J, Hamblen DL. The pathology of the degenerative meniscus. *J Bone Joint Surg Br*. 1975; 57(2):180-186.

12. Stefanik JJ, Guermazi A, Roemer FW et al. Changes in patellofemoral and tibiofemoral joint cartilage damage and bone marrow lesions over 7 years: the Multicenter Osteoarthritis Study. *Osteoarthritis Cartilage*. 2016; 24(7):1160-1166.

13. Langhorst NE, Damen J, Oei EH et al. Incidence, prevalence, natural course and prognosis of patellofemoral osteoarthritis: the Cohort Hip and Cohort Knee study. *Osteoarthritis Cartilage*. 2017; 25(5):647-653.

14. van Middlekoop M, Bennell KL, Callaghan MJ et al. International patellofemoral osteoarthritis consortium: Consensus statement on the diagnosis, burden, outcome measures, prognosis, risk factors and treatment. *Seminars Arthritis Rheum*. 2018; 47(5):666-675.

15. Teichtahl AJ, Wluka AE, Wang Y, et al. Obesity and adiposity are associated with the rate of patella cartilage volume loss over 2 years in adults without knee osteoarthritis. *Ann Rheum Dis*. 2009; 68:909–13.

16. Elahi S, Cahue S, Fleso DT, Engelman L, Sharma L. The association between varus-valgus alignment and patellofemoral osteoarthritis. *Arthritis Rheum*. 2000; 43(8):1874-1880.

17. Hinman RS, Crossley KM. Patellofemoral joint osteoarthritis: an important subgroup of knee osteoarthritis. *Rheumatology*. 2007; 46(7):1057-1062.

18. Thomas MJ, Wood L, Selfe J et al. Anterior knee pain in younger adults as a precursor to subsequent patellofemoral osteoarthritis: a systematic review. *BMC Musculoskelet Disorders.* 2010;11:201

19. Crossley KM. Is patellofemoral osteoarthritis a common sequela of patellofemoral pain? *Br J Sports Med*. 2014; 48(6):409-410.

20. Collins NJ, Oei EHG, de Kanter JL, Vicenzino B, Crossley KM. Prevalence of radiographic and MRI features of patellofemoral osteoarthritis in young and middle-aged adults with persistent patellofemoral pain. *Arthritis Care Res*. 2018; Aug 21. doi: 10.1002/acr.23726. [Epub ahead of print]

21. Nimon G, Murray D, Sandow M, et al. Natural history of anterior knee pain: a 14- to 20-year follow-up of nonoperative management. *J Pediatr Orthop* 1998;18(1):118–22.

22. Cibere J, Sayre EC, Guermazi A, et al. Natural history of cartilage damage and osteoarthritis progression on magnetic resonance imaging in a population-based cohort with knee pain. *Osteoarthritis Cartilage*. 2011; 19(6):683-688.

23. Collins NJ, Barton CJ, van Middlekoop m e ta. 2018 Consensus statement on exercise therapy and physical interventions (orthoses, taping and manual therapy) to treat patellofemoral pain: recommendations from the 5th International Patellofemoral Pain Research Retreat, Gold Coast, Australia, 2017. *Br J Sports Med*. 2018; 52(18):1170-1178.