

Knee

Musculoskeletal pain syndromes are seldom caused by isolated precipitating events, but are the consequences of habitual imbalances in the movement system. The one off injury such as the torn anterior cruciate ligament can and does occur, but more often than not, physiotherapists are dealing with more complex pain syndromes such as patellofemoral pain, shin splints, and back pain, so the problem is often multifactorial and the cause of the pain may be remote from the site of the symptoms. Identifying why a particular patient's knee problem is more difficult to treat is a challenge facing all clinicians. A thorough assessment is the key to unravelling the issues, remembering too that pain (and its chronicity) affects individuals quite differently.

Why are some individuals more susceptible to pain?

- An individual's mechanics has a marked effect on his/her inherent stability and passive control and hence propensity for experiencing pain. A joint can be passively unstable, but dynamically stable, as muscles via the neural system can compensate for the lack of stability in the passive structures (Panjabi, 1992). Equally, a passively stable joint can be dynamically unstable when muscle control is poor.
- The amount of load through the soft tissues or the frequency of the loading will also affect joint structures and may result in tissue failure, as an individual may breach his/her threshold and stray out of the zone of homeostasis (Dye, 1996). Dye contends that symptoms will not be present if an individual is operating inside his/her envelope of function, but as soon as a threshold is reached a complex biological cascade of trauma and repair will occur, which will be manifested clinically by pain and swelling.

What factors can contribute to patellofemoral pain?

- Proximally, femoral anteversion or internal femoral rotation affects the position of the trochlea relative to the patella. Internal femoral rotation is associated with poor posterior gluteus medius activation, affecting the stability of the pelvis. Improving hip extension and external rotation mobility as well as gluteal control in weight bearing by simulating the stance phase of gait, can significantly improve the symptoms of many "difficult" knees.

- Distally, pronation problems of the foot – too much, too long or too late can cause an increase in the dynamic valgus vector force on the patellofemoral joint. Orthotics may be used to help foot control. Clinically, comfort is the most important and relevant feature when prescribing foot orthoses (Mündermann et al 2003).
- Locally, tightness of the lateral retinacular structures and the iliotibial band as well as delayed onset of the VMO relative to the VL can cause pressure distribution variations of the patella on the femur. Changing the activity of the VMO relative to the VL may not only require up-training of the VMO but may require down-training the VL. Some “difficult” knees may require inhibitory tape on the VL to improve the coordination of the quadriceps activation.

Where is the pain coming from?

Multiple local anatomical structures are capable of producing anterior knee pain. Those implicated include the medial and lateral retinaculum, the patellar subchondral bone, the anterior synovium, the joint capsule, the patellar tendon, the infrapatellar fat pad and scarring of the infrapatellar branch of the saphenous nerve following

arthroscopy. The fat pad is highly nociceptive and vascular. A recent study showed that injecting 0.25 ml hypotonic saline into the fat pad of asymptomatic individuals caused moderate to severe anterior knee pain (Bennell et al 2004). Additionally, one cannot forget in some “difficult” knees, the possibility of referred pain from the lumbar spine or in prepubescent sufferers the possibility of a slipped femoral epiphysis.

Fat pad irritation

- The fat pad is an under-diagnosed, poorly recognised source of patellofemoral pain, so often falls into the category of the “difficult” knee. Fat pad problems are frequently confused with patellar tendinosis.
- The fat pad stabilises the patella at the extremes of knee motion and alters knee biomechanics, improving tibial external rotation relative to the femur (Bohnsack et al 2004). Imaging abnormalities seen are usually the consequence of trauma and degeneration. The commonest traumatic lesions follow arthroscopy. Fat pad impingement can also occur after patellar dislocation (Saddik et al 2004).
- If the patient describes an acute onset of pain in the history, this is associated with rapid extension of knee such as a tumble turn in swimming or landing on a straight knee during a running or jumping action. On examination, the knees are hyper-extended or “locked back” in standing, the inferior pole of patella is embedded in underlying tissues, the fat pad has a puffy appearance and there is often focal tenderness of inferior pole of the patella. Quadriceps setting and extension overpressure manoeuvres can exacerbate the symptoms. Individuals with fat pad symptoms have very poor inner range quadriceps control and often walk with a flexed knee gait to avoid irritating the fat pad.
- Unless the fat pad is unloaded it is difficult for patients to progress as the fat pad is easily irritated during daily activities and rehabilitation.

Other factors compounding treatment of the “difficult” knee

- It has long been established that pain has an inhibitory effect on muscle activity. Activity of the VMO and VL is decreased when pain is induced in asymptomatic individuals by injecting hypotonic saline in the fat pad (Hodges et al 2009).
- Additionally fear of pain changes muscle activation patterns. Randomly applying an electric shock across the knee during stair ascent and descent in subjects with no history of knee pain causes a significant reduction in VMO EMG activity but not VL activity (Hodges et al 2009). Thus, not only will pain decrease quads activation but fear of pain will increase the imbalance between the VMO and VL making rehabilitation difficult. The clinician has to recognize that fear of pain can be retarding the patient’s recovery, resulting in the label of the “difficult” knee. Minimising the patient’s pain should help resolve the fear of pain and decrease the imbalance between the VMO and VL, accelerating the patient’s rehabilitation.

- Fear-avoidance has long been recognized as an important factor in the development of pain-related disability (Boersma et al 2005). Exposure to stress initiates the secretion of several hormones, including corticosterone/cortisol, catecholamines, prolactin, oxytocin, and renin, as part of the survival mechanism. (Van de Kar et al 1999). Release of cortisol can be detrimental to a patient's recovery. In fact it has been found that stress-related hormones can alter inner ear fluid homeostasis and auditory function. (Juhn et al 1999). This could have implications for the balance of individuals exhibiting fear/avoidance behaviour and may additionally add to the "difficult" knee problem.

- Negative self talk affects a patient's belief system can affect outcome of treatment. Positive affirmations about the knee and desensitising techniques can be useful in shifting the focus for the "difficult" knee patient.
- Centrally maintained pain can significantly affect the progress of a "difficult" knee. Clinicians need to be aware of the treatment options available for this group of patients (Butler & Moseley, 2003).

Some long term strategies for the "difficult knee"

- Show patients how to get out of end of range positions to minimise the stress on soft tissue structures, by giving them simple strategies such as how to stand, get out of a chair and negotiate stairs.
- Give specific exercises which fit into daily life and can be performed frequently, in small quantities, building for endurance, to change the behaviour pattern (the synergistic recruitment pattern) of the muscles.
- Ensure the patient is aware the condition is managed not cured – so an on going maintenance is required much like cleaning one's teeth. The program must be simple, requiring very little equipment so it is transportable and takes no more than 5 minutes per day.
- Once the "difficult" knee problem has been resolved the clinician needs to review the patient every 6 months to ensure the patient is still doing the maintenance exercises, to keep him/her symptom free.

References

- Bennell K, Hodges P, Mellor R, Bexander C, Souvlis T. 2004 The nature of anterior knee pain following injection of hypertonic saline into the infrapatellar fat pad. *J Orthop Res* 22(1):116-21.
- Boersma K, Linton SJ. 2005 Screening to identify patients at risk: profiles of psychological risk factors for early intervention.: *Clin J Pain* 21(1):38-43; discussion 69-72.
- Bohnsack M, Wilharm A, Hurschler C, Rühmann O, Stukenborg-Colsman C, Wirth CJ. 2004 Biomechanical and kinematic influences of a total infrapatellar fat pad resection on the knee. *Am J Sports Med.* 32(8):1873-80.
- Butler D, Moseley L, 2003 Explain Pain. NOI Group Publications
- Dye S. (1996) The knee as a biologic transmission with an envelope of function: a theory. *Clinical Orthopaedics*(325):10-18.
- Hodges PW, Mellor R, Crossley K, Bennell K. 2009 Pain induced by injection of hypertonic saline into the infrapatellar fat pad and effect on coordination of the quadriceps muscles. *Arthritis Rheum.* 15;61(1):70-7.
- Juhn SK, Li W, Kim JY, Javel E, Levine S, Odland RM. 1999 Effect of stress-related hormones on inner ear fluid homeostasis and function. *Am J Otol* 20;6:800-6.

Kopec JA, Sayre EC. 2004 Work-related psychosocial factors and chronic pain: a prospective cohort study in Canadian workers. : J Occup Environ Med. 46(12):1263-71.

McConnell J 2002 The physical therapist's approach to patellofemoral disorders. Clin Sports Med 21(3):363-87

Mündermann A, Nigg BM, Humble RN, Stefanyshyn DJ. 2003 Orthotic comfort is related to kinematics, kinetics, and EMG in recreational runners. Med Sci Sports Exerc. 35(10):1710-9.

Panjabi M (1992a) The stabilising system of the spine . Part I, Journal of Spinal Disorders 5(4) 383-389

Saddik D, McNally EG, Richardson M. 2004 MRI of Hoffa's fat pad. Skeletal Radiol. Aug;33(8):433-44.

Van de Kar LD, Blair ML. 1999 Forebrain pathways mediating stress-induced hormone secretion. Front Neuroendocrinol, 20(1):1-48.



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