The Independent Medical Expert Group (IMEG) 5th Report

Report and recommendations on medical and scientific aspects of the Armed Forces Compensation Scheme

February 2020

Topic 4 - Musculoskeletal (MSK) Disorders Part 2

Key Points

1. Musculoskeletal (MSK) Disorders Part 2 has two sections, A and B. Section A covers general aspects of MSK disorders in the UK military at this date; the challenges of recruitment and retention, gender issues, medical downgrading and discharges, AFCS compensation awards and occupational aspects of MSK disorders.

2. Section B comprises summaries of disorders seen in military practice, with definition, a brief description of clinical features, aetiology, prevention, best practice clinical management and prognosis.

3. MSK disorders are the most common category in military populations often leading to medical downgrading and discharge. Between 1 April 2018 and 31 March 2019 there were 1,869 medical discharges from the UK armed forces made up of 391 from the naval service (RN and RM), 1,316 from the army and 162 from the RAF. This equates to rates of 12 per 1,000 for the naval service, 17 per 1,000 for army and 5 per 1,000 for RAF (1).

4. "At risk" age groups are for the naval service, 25-34 years, for the army, 20-24 years and for the RAF, 45-49 years. In line with this age distribution, discharge from the army is high in untrained personnel with short service. Since AFCS began in April 2005, awards for MSK diagnoses account for 45% of total awards made, and 51% of awards made in 2018/19.

5. To understand the generation of pain from affected musculoskeletal structures and why pain in these disorders so frequently becomes chronic, even when objective signs and radiological findings are minimal or absent, a brief overview of pathophysiological evidence is included. This considers local abnormalities in affected structures, the role of the peripheral nervous system in signalling pain, and the increasing evidence on the part which may be played by consequent changes in the central nervous system. Wider aspects of chronic pain and its co-morbidities and its diagnosis, treatment and rehabilitation are also considered.

6. Pain is the leading symptom and cause of disabling effects in MSK disorders. A key aim of treatment is that the patient should be able to accept and manage his pain. Multidisciplinary approaches including psychological therapy are key, and clinical management to reduce pain and/or improve function, education to inform about the disorder and emphasise the positive effects of maintained activity, especially work activity, are the norm.

Introduction

1. This report follows from Musculoskeletal (MSK) Disorders Part 1 published as part of the Fourth IMEG report, December 2017. In AFCS compensation many MSK disorders (Tariff Table 9) arise over time from overuse or repetitive attrition damage rather than from a discrete physical traumatic injury listed in Tariff Table 2, Injuries, wounds and scarring. Musculoskeletal (MSK) Disorders Part 2 has two sections, A and B. Section A covers general aspects of MSK disorders in the UK military at this date; the challenges of recruitment and retention, gender issues, medical downgrading and discharges, AFCS compensation awards and occupational aspects of MSK disorders. Because pain is the leading

symptom and cause of disabling effects in MSK disorders, an overview is also included. Section B comprises summaries, with definition, a brief description of clinical features, aetiology, prevention, best practice clinical management and prognosis, on a further list of specific injuries seen in the military context. As previously, the report is informed by literature scrutiny and discussion within the group and with military and civilian clinical experts on the various topics.

Background

2. MSK disorders are common across the age groups in Western societies but are a particular issue amongst working age adults and a major cause of sickness absence and ill-health retirement in civilian society despite the reduction in physical workloads. Similarly, MSK disorders continue to be the most common category of injuries in military populations often leading to medical downgrading and discharge. Between 1 April 2018 and 31 March 2019 there were 1,869 medical discharges from the UK armed forces made up of 391 from the naval service (RN and RM), 1,316 from the army and 162 from the RAF. This equates to rates of 12 per, for the naval service, 17 per 1l000 for army and 5 per 1,000 for RAF (1). For all three services, groups at significantly higher risk of medical discharge include women and other ranks. "At risk" age groups are for the naval service, 25-34 years, for the army, 20-24 years and for the RAF, 45-49 years. In line with this age distribution, discharge from the army is high in untrained personnel with short service. Given the demanding physical requirements of army and RM training these facts are not unexpected, especially against a background of societal change with decline in school team games and less physically active adolescent recreational pursuits. In all three services the main cause of medical discharges remains MSK disorders. In some cases, MSK disorders are accompanied by a mental health disorder, with both disorders contributing to the need for medical discharge. In 2018/19 MSK disorders made up 56% of naval service and army discharges and 49% RAF discharges. Back and knee disorders are the most common diagnoses.

3. MSK awards continue to be the most common category of injuries awarded under the AFCS, particularly in the army (2). Since the scheme began in April 2005, they account for 45% of total awards made, and 51% of awards made in 2018/19. Although the most commonly awarded disorders in terms of lump sums, MSK descriptors accounted for only 14% of Guaranteed Income Payments (GIP). This reflects the high physical fitness requirements of service compared with civilian work and the fact that Defence and single service policy is to accommodate personnel with disability or chronic illness by fully exploring possible alternative roles ahead of service termination. In some services, e.g. Royal Marines (RM) and parachute regiments, military options are limited, although the person may be functionally capable of a range of suitable full-time civilian work, where the Equality Act and the employer requirement to make reasonable adjustments to support his or her being in work, may apply.

4. While some MSK disorders which occur in the military are well defined with known clinical course, accepted best practice management and swift return to training or duty, many of the most common problems, typically affecting knees are much less matters of consensus in terms of most appropriate investigation, definitive diagnosis, recognised effective treatment and successful outcome. Terms still common in military practice but less used in civilian orthopaedic and sports injuries include anterior knee pain and chondromalacia patellae. In recent years, as in civilian practice, the use of MRI has become almost universal although many pathologies reported on scans are not confirmed on direct arthroscopic inspection. Over-reliance on MRI scans to inform clinical management may impact recovery, which may be incomplete and prolonged, with medical downgrading and ultimate discharge. This may result in long term compromise and cost to military operational capability, disruption and detriment to the person and his family, and post service civilian employability.

5. In terms of treatment, surgery has a limited role in overuse injuries. Physical therapies are first choice based on exercise, massage and medication, with surgery usually only considered when disorders remain resistant to conservative treatment. There is a lack of treatment evaluation studies and for some common injuries and disorders individual specialists use different diagnostic terminology and vary in their recommendations for the range of possible treatments and timelines for more specialist referral. To address some of these issues and better standardise Defence care, Defence Primary Health Care (DPHC) has developed best practice clinical guidelines and quality standards on a range of topics, e.g. exercise induced leg pain. Regardless of specific diagnosis, common principles apply with management first in primary care including at Primary Care Rehabilitation Facility (PCRF). If at a certain interval, (e.g. six or twelve weeks), progress is limited, then the guidance will suggest onward referral to the Regional Rehabilitation Unit (RRU) and, dependent on further progress, patients may be seen at the Defence Medical Rehabilitation Centre, (DMRC) Stanford Hill.

6. Where progress and recovery from MSK disorders are slow there may be a need to confirm the diagnosis, with exclusion of other pathologies and systemic disorders. Evidenced best practice guidelines stress the need to be alert to symptoms or physical signs which suggest serious underlying pathology. For MSK disorders these may include fever, skin erythema, history of weight loss, a mass or swelling over soft tissue or bone, intense bone pain, and particularly in relation to low back pain, symptoms of bladder or bowel disturbance and the presence of focal neurological signs. Important disorders to exclude include deep vein thrombosis, acute compartment syndrome, osteomyelitis, bone tumours and compression of spinal nerve roots (3).

7. Physical disorders or injury often have emotional sequelae and patients may be anxious or low in mood. Relevant psychosocial factors may include attitudes and beliefs, comorbid discrete psychiatric disorders, unresolved financial compensation, work issues (including poor job satisfaction, alleged lack of peer or management support, complaints of bullying or harassment) and family attitudes, which may be critical or overprotective. Clues that psychosocial factors may be hindering progress include complaints of prolonged pain, sometimes associated with other sensory or motor symptoms, but without consistent objective neurological signs being found at different examinations. A tendency to catastrophize may induce a fear that movement or activity will cause worsening, with over-reliance on aids and appliances, adoption of a disabled lifestyle of prolonged incapacity, overall decline in fitness and failure to return to work either in the military or the civilian community. Pain cannot always be effectively relieved, and a key aim of treatment is that the patient should be able to accept and manage his pain. Multidisciplinary approaches including psychological therapy are key, and clinical management to reduce pain and/or improve function, education to inform about the disorder and emphasise the positive effects of maintained activity, especially work activity, are the norm.

8. As well as discrete diagnosed disorders, MSK aches and pains, often recurrent and without objective signs or imaging abnormalities are a particular issue amongst the working age population and, both in civilian and military populations, because they may impact working capacity at least temporarily, and it is commonly believed that these are traumatic physical injuries caused by work. Focus on prevention and clinical management has failed to substantially reduce incidence and prevalence of disabling MSK symptoms, and it is increasingly clear that psychosocial factors are relevant with the best practice approach, collaboration between patient, clinicians, and the management or chain of command. Wherever possible, workplace or task modification should support maintaining the person in work or expediting their early return.

9. Many aspects of UK military MSK problems, downgrading and discharge etc are common globally. At a time when young people's choice of career routinely takes into account lifestyle, family and work life balance, recruitment and retention in the military can be challenging. Other factors include the changing shape of war, with more reliance on technical expertise as opposed to physical prowess and the need to reflect equality and diversity principles. There remains much debate about required mental and physical fitness standards, including whether these should be generic, regardless of

gender and related to combat roles or otherwise occupation specific. Training programmes and how fitness might most effectively and efficiently be achieved and maintained over time are also subjects of discussion. In terms of physical requirements, the physiology, muscle composition and bone density of the genders differ, and UK data confirms higher rates of MSK overuse injury in women, at a time when most armed forces aspire to an increasing proportion of women, with the option of traditionally male roles e.g. in front line combat. In training, "gender free" policies have been trialled where similar fitness levels are required of both men and women. As expected, this leads to higher female discharges for MSK overuse injuries. The alternative "gender fair" policies adopt lower entry and retention standards for females compared with males and while discharges for overuse injuries amongst women compared with men may be lower than with "gender free", the experience is that discharge rates are still higher than for men (4). These approaches illustrate the conflict between health and safety and equality legislation and make the point that selection tests and standards should take into account gender difference.

10. Beyond gender there are more general issues about MSK injury in initial military recruit training across the world, with some studies recording 60-70% of trainees being injured in the initial eight-week period (5), (6). Not only do we need consistent best practice management of injury and disorder, but prevention measures, including well designed training programmes are key. Programmes should reflect the overall reduced levels of fitness of young people today, changing gender balance amongst entrants and service occupations. Both best practice clinical management and injury prevention are informed by data on specific injury type and incidence and rehabilitation times.

11. Such studies were recently carried out in British army recruits at Catterick Infantry Training Centre. The first was a prospective study of diagnoses, giving specific incidence and rehabilitation times (7). Over a two-year period in 2006-2008, 6,608 military recruits were followed up. The overall incidence of MSK injuries in the 26-week initial training period was 49%. The most common diagnosis was iliotibial band syndrome (6%) and a significant proportion of injuries occurred in the first week of training. The longest rehabilitation times were for stress fractures of the femur (mean 116 days with standard deviation (SD) 17 days), calcaneus (mean 92 days with SD 12 days and tibia (mean 85 days with SD 11 days). Medial tibial stress syndrome accounted for almost 20% of days spent in rehabilitation. The second study was retrospective and considered the incidence of MSK injuries and training outcome in different infantry regiments over five consecutive training years (2013-2017) (8). From a total of 12,501 recruits there were 4,777 injuries. Overuse injuries were the most common, mainly affecting the lower limb and especially the knee. Almost half the total injuries occurred during the first eight weeks of training. Successful training outcome varied widely across the different regiments e.g. Parachute regiment 37%; Guards 53%; Line regiments 65%; and Ghurkha's 98%.

12. Prevention measures for training injuries divide into extrinsic and intrinsic. Extrinsic factors relate to the training programme itself and include footwear, hard surfaces, failure to warm up, cold weather training, too rapid increase in training intensity and its maintenance at a high level especially where there is no prior experience of such training. Intrinsic factors are about the individual e.g. BMI (body mass index), pre-injury level of fitness, pre-service injury or congenital biomechanical abnormality. MSK disorders also occur in all other groups of service personnel and some services, corps and regiments are innately more physically demanding, such as RM, commando and parachute regiments. Personality and behavioural issues are also important, with desire for career progression or to deploy, often leading to delay or failure to present MSK symptoms early or continuing at a high level of physical training when rest and activity reduction have been advised.

13. It is sometimes assumed that armed forces' service in any capacity is innately very physically demanding and, even without significant discrete traumatic physical injury, likely to lead to overuse attrition-related damage and osteoarthritic change in joints. As always there are limitations due to study design, size, selection bias etc, but overall, the extensive evidence from international civilian occupational studies in heavy industry, e.g. construction and mining, does not confirm inevitable

disabling degenerative change over time (9). Civilian study subjects' work pattern centres on the same tasks and activity for around 40 hours a week, 45 weeks a year for many years, a rather different pattern from military service. Similarly, sporting studies up to elite level do not consistently support degenerative change in the absence of documented traumatic physical injury (10). In terms of compensation under AFCS, all cases are considered on their individual specific facts including type, duration and activities of service, whether there are documented injuries, the applicable legislation and contemporary accepted medical understanding of causation progress and likely outcome, including best practice treatment. Where, on balance of probabilities, AFCS service is the predominant cause of the claimed disorder, an award will be made and with military and civilian clinical experts on the various topics.

References:

1. MOD Defence Statistics. Annual medical discharges in the UK regular armed forces. 1 April 2014 - 31 March 2019. Available from: https://www.gov.uk/government/publications /annual medical discharges in the UK regular armed forces. 1 April 2014-31 March 2019. (accessed 5 July 2019).

2. MOD Defence Statistics. AFCS compensation awards. Available from: https://www.gov.uk/ government/publications/ AFCS compensation awards. (accessed 5 July 2019).

3. Waddell, G. The back-pain revolution. Edinburgh Churchill Livingstone 1998

4. Gemmell, I. M. M. Injuries among female army recruits: a conflict of legislation. J. R. Soc. Med. 2002 Jan; 95(1):23-27.

5. Knapik, J. J. et al. Association between ambulatory physical activity and injuries in army basic training. Am. J. Prev. Med .2011;18:147-55.

6. Taanila, H. et al. Musculoskeletal disorders in physically active recruits: a one -year follow-up study in the Finnish Defence Forces. BMC Musculoskeletal Disorders. 2009; 10:89.

7. Sharma, J. et al. MSK injuries in British army recruits: a prospective study of diagnosis, specific incidence and rehabilitation times. BMC Musculoskeletal Disorders. 2015; 16:106.

8. Heagarty, R.D.H. et al. MSK injuries in British army recruits: a retrospective study of incidence and training outcome in different regiments over five consecutive training years. Int. J. Phys. Med. Rehabil. 2017; vol5(6):440.

9. Battie, M. L. et al. Lumbar disc degeneration: Epidemiology and genetic influences. Spine 2004; 29 (23) :279-290.

10. Roos, H. Are there long-term effects from soccer? Clin. Sports. Med. 1998; Oct 17(4); 819-31.

Pain in Musculoskeletal Disorders

14. To understand the generation of pain from affected musculoskeletal structures and why pain in these disorders so frequently becomes chronic, even when objective signs and radiological findings are minimal or absent, a brief overview of pathophysiological evidence follows. This is a technical and complex topic, and a more detailed discussion is given in Wall and Melzack's Textbook of Pain (2013) 6th edition (1). This overview considers local abnormalities in affected structures, the role of the peripheral nervous system in signalling pain, and the increasing evidence on the part which may

be played by consequent changes in the central nervous system, in particular, central sensitization. Wider aspects of chronic pain and its co-morbidities and the benefits of a biopsychosocial approach to diagnosis, treatment and rehabilitation are also considered.

15. Pain has been defined as "an unpleasant sensory and emotional experience associated with acute or potential tissue damage or described in terms of such damage" (2). Acute pain has a key bioprotective function, alerting an individual to the presence of injury, and prompting appropriate action. Following an injury, in the short term, continuing pain may protect the injured body part by causing immobilization of the affected part, with avoidance of further injury and promotion of healing. Peripheral sensory nerves signal acute pain. With skin injury, there is usually precise localisation of the injury, but deep injury to muscle, joints, fascia and bone is often less well localised.

Nociceptive and Inflammatory Pain

16. Nociceptive pain is pain that is detected by nociceptors, which are small diameter myelinated nerve fibres and unmyelinated nerve fibres and their terminals in peripheral tissues. These fibres respond specifically to high threshold, potentially tissue-damaging stimuli. Following injury in which there is tissue damage, an inflammatory response develops. In the skin, this is obvious, as with an insect bite or sunburn. In deep tissues there is often no obvious sign other than limited mobility and pain provoked by use of the affected part. An inflammatory response to injury is normal and potentially completely reversible with healing of the injury. This inflammatory response leads to "primary hyperalgesia" in which the threshold of nerve fibres to stimulation is decreased, so that normally non- painful stimulation is perceived as being painful (allodynia), and ongoing pain at rest may develop. The cascade of injury-related chemical changes in acute inflammation is complex with, within a few minutes of acute injury, release of inflammatory substances including bradykinin, prostaglandins, and histamine and serotonin from circulating mast cells and platelets (3). In primary hyperalgesia the heightened pain and tenderness is fairly well-localised to the injured tissue. With a persistent intensely painful stimulus, over minutes and hours secondary hyperalgesia develops, in which pain, tenderness and allodynia spreads well beyond the region of the original injury (4). This involves spinal cord mechanisms, notably central sensitisation, described below.

Neuropathic Pain

17. Neuropathic Pain is pain is caused by trauma or disease-related damage to the somatosensory system, comprising peripheral sensory nerves, and sensory pathways in the spinal cord and brain (5)(6)(7). The practical consequence of this definition is that in order to label pain as being neuropathic, there must be clinical evidence of a lesion in some part of this system. In other words, a neurological diagnosis is required. This can be challenging clinically, because sensory deficits and associated neuropathic signs may be subtle and difficult to detect. Diagnosis may be improved by specialist investigation such as quantitative sensory testing, histological examination of biopsies for nerve fibre density, and identification of abnormal ion channels as in some small fibre neuropathies (8). Persistent pains of obscure origin are sometimes labelled as being neuropathic in the absence of a rigorous clinical approach to diagnosis. This is unhelpful, particularly for the individual mis-diagnosed with such pain, because subsequent treatment may be inappropriate and less successful.

18. Chronic nociceptive pain is potentially completely recoverable, while the same may not be true for many patients with neuropathic pain. It has long been recognised that neuropathic pain is associated with a poorer prognosis, and the reasons for this are now becoming clearer, largely through experimental investigations (9). A range of abnormal properties may develop following primary damage to peripheral and central nervous system sensory pathways involved in pain transmission. These include ectopic impulse generation (spontaneous activity unprovoked by peripheral

stimulation) in damaged peripheral nerve fibres; abnormal chemical sensitivities of damaged fibres; electrical cross-talk between damaged nerve fibres; hyperexcitability of second order receiving neurons situated in the dorsal horn of the spinal cord; and regenerative nerve fibre sprouting. This may lead to aberrant and potentially irreversible reconnection, causing peripheral nerve fibres normally signalling information provoked by non-painful stimuli to lead to painful sensations. In addition, the normal descending inhibitory pathways for pain, originating in the brain stem, that are activated by incoming painful stimuli, become less effective, increasing the perception of pain. These peripheral nervous system and spinal cord abnormalities may further induce abnormalities at higher levels in the central nervous system including the thalamus and cortical areas involved in pain perception (10),(5).

19. This brief account of the neurophysiology of neuropathic pain provides the basis of an explanation for its poor prognosis. If irreversible changes in neural connectivity have become established, it is not surprising that standard pain treatments may have limited efficacy. In the case of nociceptive pain, hyperalgesia, allodynia and central sensitization (described in paragraph 20) are essential features, in which the nervous system responds in a normal bio-protective fashion. If the peripheral cause of the pain can be treated or recovers spontaneously, the nervous system reverts to a normal state. If this were not the case, each new injury or other cause of pain experienced would result in a gradually increasing pain state during life, which is clearly not the case. It is emphasised that all the MSK conditions discussed in Section B of this paper give rise to nociceptive pain, except where specifically noted.

Central Sensitization

20. In musculoskeletal pain, and more generally in patients with chronic pain of benign origin, there is often a disparity between the intensity of the reported pain and the demonstrable severity of tissue damage. One reason for this mis-match is central sensitizationthat is, the amplification of central nervous system sensory signalling, an “increased responsiveness of nociceptive neurons to their normal or subthreshold input” (11). Although there is extensive experimental evidence indicating likely mechanisms of Central Sensitization (CS), clinical evidence is less complete and as yet, there is no applicable agreed method or guideline for its definitive diagnosis (12). The term was first used to describe a single specific spinal cord mechanism, for clarity, referred to here as spinal CS (10).

21. Under experimental conditions, activation of sensory unmyelinated C-fibres is needed to induce CS; the noxious stimulus has to be intense, repeated and sustained over at least tens of seconds. Peripheral tissue injury is not necessary, although noxious stimuli that produce tissue injury almost invariably produce CS. The neuropharmacology of synaptic transmission of primary afferent nociceptive nerve fibres is highly complex. However, of particular importance in the induction of CS, glutamate, the fast transmitter of primary afferent neuron terminals in the spinal cord, binds to N-Methyl-D-Aspartate (NMDA) receptors (13). The therapeutic significance of this is that drugs that antagonise this mechanism may produce analgesia, by reducing CS. Such drugs include ketamine and the gabapentinoids (gabapentin and pregabalin). A partial, though variable analgesic effect of these drugs has been reported in nociceptive as well as neuropathic pain, notably in post-operative pain, though current clinical evidence is weak (14)(15). At present, the recommended, licenced use of gabapentinoids is for neuropathic pain, in particular for patients with post-herpetic neuralgia and painful diabetic neuropathy. Whether or not neuropathic pain in other small fibre neuropathies, including non-freezing cold injury (NFCI), responds to these drugs remains to be investigated in controlled clinical trials.

22. Numerous other neurotransmitters are now known to be involved in the mechanism of CS. It is recognised that there is no single neurotransmitter involved in producing CS in the dorsal horn of the spinal cord. The recognition of many other central nervous system knock-on effects of a persistent painful input from peripheral tissues has rather confusingly led to a wider common usage of the term

CS, which we may refer to here as supra-spinal CS. By a variety of molecular mechanisms, the core features of CS can be produced: in response to a prolonged, sustained peripheral nociceptor input there is an increase in membrane excitability, synaptic transmission can be facilitated, and inhibitory influences on dorsal horn neurons can be decreased (13). Similar mechanisms in the brain may underpin changes in neuronal responses to prolonged nociceptive inputs, producing supraspinal CS. 23. Of particular relevance to MSK disorders, there is evidence that deep somatic pathological conditions affecting joints and muscles are particularly likely to induce CS, particularly low back pain (16), (12). CS associated with pain of musculoskeletal origin is potentially completely reversible, while by contrast, there is evidence that ongoing CS can become a persistent feature of neuropathic pain (17).

Damaged Musculoskeletal Structures that Generate Pain

Joints

24. Normal joints do not give rise to conscious sensation; joint position sense is essential for postural control but is not consciously perceived. Dull aching and poorly localised pain is the major sensation from damaged and diseased joints (4). Cartilage, which is aneural, does not cause pain in health or disease (18). Joint capsules, synovium and ligaments have nociceptors, and pain can sometimes be referred from adjacent joints, muscle, ligaments and fascia (19). In osteoarthritis (OA), cartilage is initially affected, and then multiple structures in the joint are involved, inflammation and cytokine production leading to pain (20), (21), (22). In joint disease, normal afferent nerve fibres become sensitised by bradykinin, prostaglandins and other inflammatory substances, so that normally 'silent' nociceptors become sensitised to movement, causing pain. These changes powerfully induce spinal CS (23). Subchondral bone pathology causes pressure changes and microfractures, osteophytes may stretch the periosteum, and the joint capsule may undergo distension and inflammation. Inflammatory change in synovium may also contribute to pain.

Muscles

25. Muscle pain (myalgia) is signalled via small diameter myelinated and unmyelinated nerve fibres. These are normally silent but are activated and sensitised by inflammatory substances and accumulation of lactic acid, becoming active at rest. The sensory input from damaged muscle induces spinal CS, together with the unmasking of normally ineffective synapses in the dorsal horn of the spinal cord (24). This is the basis of referred pain to sites remote from the damaged muscle (25). Persistent muscle pain has been shown experimentally to lead to alterations in activation in the cerebral cortex, notably the cingulate cortex. Chronic work-related myalgia (occupational muscle pain) may occur on a background of monotonous repetitive work at a low level of force (26) together with the additional factor of psychological stress (27).

Tendons

26. The tensile strength of tendons is related to thickness and collagen content; a tendon with a cross-sectional area of 1cm2 is capable of supporting 500-1000kg. Loading of the Achilles tendon during running may reach 9kN, equivalent to 12.5 times the body weight (28). Thus, tendons are extraordinarily resilient structures. Pathological mechanisms other than inflammatory change are important in acute and chronic tendon injury, so the term 'tendinopathy' is generally now preferred to tendinitis or tendonitis (29). Trauma due to overload and overuse is the leading cause of tendinopathy.

In a military population, other recognised causes are rarely seen; these include gout, thyroid disease, diabetes and treatment with quinolone antibiotics. Once damaged, tendons heal slowly and are prone to re-injury. With overuse injury, the microscopic changes in tendinopathy are an absence of inflammatory cells and a poor healing response, with non-inflammatory collagen fibre degenerationand thinning, hypercellularity, neurovascular in-growth and increased intrafibrillar glycosaminoglycans or mucopolysaccharides (30),(31). An obvious inflammatory response is usually seen only in complete tendon rupture.

Co-Morbidities of Chronic Pain

27. The co-morbidities of chronic pain may contribute substantially to loss of function and impaired quality of life. A summary of 17 reviews found that factors consistently associated with poor outcome after Low Back Pain (LBP) included poor general health, psychological distress, poor relations with work colleagues, physically heavy work, and worse baseline functional disability (32). Anxiety and depression are common in patients with chronic pain and have been found to predict both pain severity and disability (33). In a study of US veterans with chronic MSK pain disorders, both post-traumatic stress disorder (PTSD) and depressive illness were independently associated with worse pain, poorer quality of life, and disability (34). Depression and anxiety may also increase the prevalence of opioid misuse in patients with chronic pain (35). A systematic review of 38 studies found the prevalence of opioid misuse in patients with chronic pain was between 21% and 29% (95% confidence interval 13%-38%) (36). There is a two-way association between chronic pain and insomnia, thought to be partially mediated by not only pain intensity, but also anxiety, depression and health anxiety (37), (38).

28. Personality may play a role in maintaining chronic pain. A systematic review of 15 studies found that maladaptive emotional regulation (e.g. catastrophizing and negative mood) was associated with perceived pain severity (39). A meta-analysis showed significant associations between self-efficacy and disability, pain severity, and affective disturbance in 15,616 chronic pain sufferers (40). In one study of over 7,000 people with low back pain in Norway, followed up for 11 years, depression, anxiety and number of sites of pain were associated with less likelihood of recovery (41). Effective psychotherapies useful for chronic pain patients may include cognitive behavioural, meditation and acceptance and commitment therapies. Successful treatment depends on a multidisciplinary approach, often involving a variety of health professionals (42).

References:

1. McMahon, M.B. et. al. (eds). Wall and Melzack's Textbook of Pain. 6th edition. Elsevier Saunders, 2013.

2. International Association for the Study of Pain (IASP) Classification of chronic pain. 2nd edition (revised) Wolters Klower,1994.

3. Fields, H. L. Pain. McGraw-Hill,1987.

4. Lewis, T. Pain. Macmillan, 1942. p192.

5. Scadding, J.W. Neuropathic pain. In Brain's diseases of the nervous system. 12th edition, ed Donaghy, M. Chapter 17, pp 453-476. Oxford, 2009.

6. Jensen,T.S. et al. A new definition of neuropathic pain. Pain. 2011; v.152 issue 10:2204-5.

7. Koltzenburg M., Scadding J. W. Neuropathic pain. Current opinion in Neurology. 2013; 14:691-7.

8. Vale, T. et al. Chronic non-freezing cold injury results in neuropathic pain due to a sensory neuropathy. Brain. 2017; 140:2557-2569

9. Hökfelt T. et al. Central consequences of peripheral nerve damage. Chapter 63, pp 902-904. Wall and Melzack's Textbook of Pain. 6th edition. Eds McMahon S.B et al. Elsevier Saunders, 2013.

10. Doubell,T.P. et al. The dorsal horn: state dependent sensory processing, plasticity and the generation of pain. In Wall, P.D. et al. (eds.) Textbook of Pain 4th ed Churchill-Livingstone, 1999.

11. Sandkühler J. Spinal cord plasticity and pain. Chapter 6, pp 94-110. Wall and Melzack's Textbook of Pain 6th edition. Eds McMahon S.B et al. Elsevier Saunders, 2013.

12. Arendt-Nielsen, L. et al. Assessment and manifestation of central sensitisation across different chronic pain conditions. Eur. J. Pain (2018); Feb, 22(2):2016-241.

13. Latromoliere, A. et al. Central sensitisation: a generator of pain hypersensitivity by central neural plasticity. J. Pain. (2009); Sep,10(9):895-926

14. Chang, C.Y. et al. Gabapentin in acute post-operative pain management. 2014. Biomed. Research International Dx.doi.org/10.1155/2014/63w1756

15. Liu,B. et al. A meta-analysis of the pre-operative use of gabapentinoids for the treatment of acute post-operative pain following spinal surgery. Medicine 2017; 96:1-11.

16. Strong, J. et al. Pre-clinical studies of low back pain. Molecular Pain. 2013; 9:17. doi:10.1186/1744-8069-9-17.

17. Woolf,C. Central sensitisation: implications for the diagnosis and treatment of pain. Pain. 2011 Mar;152 (3 supp):s2-15.

18. Kellgren, H. et al. The sensitivity and innervation of the articular capsule. J. Bone and Joint Surgery. 1950; 4:193-205.

19. Kellgren, H. Some painful joint conditions and their relation to osteoarthritis. Clinical Science.1939; 4:35-46.

20. Attur, M.G. et al. Osteoarthritis or osteoarthrosis: the definition of inflammation becomes a semantic issue in the era of genomic medicine. Osteoarthritis Cartilage. 2002; 210:1-4.

21. Bondeson, J. et al. The role of synovial macrophages and macrophage produced mediators in driving inflammatory and destructive responses in osteoarthritis. Arthritis and Rheumatism 2010; 62:647-657.

22. Felson, D. The sources of pain in knee osteoarthritis. Curr. Opin. Rheumatol. 2005 Sep;17(5):624-8.

23. Schaible, H.G. Joint pain. Basic mechanisms. Chapter 44 pp 609-619. In McMahon, S.B. et al. (eds). Wall and Melzack's Textbook of Pain. 6th ed. Elsevier Saunders, 2013.

24. Wall, P.D. The presence of ineffective synapses and the circumstances which unmask them. Phil. Trans. Roy. Soc. Series B. Biological Sciences. 1977; 278:361-372.

25. Mense, S. Basic mechanisms of muscle pain. Chapter 45, pp 620-628. In McMahon, S.B.et al (eds.). Wall and Melzack's Textbook of Pain. 6th edition Elsevier Saunders, 2013.

26. Blair, S. et al. Neuromuscular mechanisms behind chronic work-related myalgias: an overview. Pp 5-46. In Chronic work-related myalgia. Eds Johansson, H. et al. Gavle University Press, 2003.

27. Hagg,G.H. The Cinderella Hypothesis. Pp 127-132. In Chronic work-related myalgia. (eds.) Johansson, H. et al. Gavle University Press,2003.

28. Komi, J. et al. Biomechanical loading of Achilles tendon during normal locomotion. Clin. Sports Med. 1992 Jul;11(3):521-31.

29. Maffulli, N. et al. Achilles tendinopathy: aetiology and management. J. R. Soc. Med. 2004 Oct; 7(10):472-476.

30. Leadbetter, W.B. Cell -matrix response in tendon injury. Clin. Sports Med. 1992 Jul; 11(3):533-78.

31. Maffulli, N. et al. Overuse tendon conditions: time to change a confusing terminology. Arthroscopy 1998 Nov-Dec; 14(89):840-3.

32. Hayden, J.A. Systematic reviews of low back pain prognosis had variable methods and results- guidance for future prognosis reviews. Journal of Clin. Epidemiol. 2009 Aug1; 62(8):781-96.

33. Lerman, S.F.et al. Longitudinal association between depression, anxiety, pain and pain related disability in chronic pain patients. Psychosomatic Med. 2015 April 1; 77(3):333-41.

34. Outcault, S.D. et al. Chronic pain and co-morbid mental health conditions: independent association of PTSD and depression with pain, disability and quality of life. J. of Behavioural Med. 2015 Jun 1; 38(3):535-543.

35. van Rijswijk, S.M. et al. Iatrogenic opioid use disorder, chronic pain and psychiatric co-morbidity. A systematic review. Gen. Hosp. Psychiatry 2019 Jul-Aug; 59:37-50.

36. Vowles, K.E. et al. Rates of opioid misuse, abuse and addiction in chronic pain and psychiatric co- morbidity: a systematic review. Pain 2015 April 1; 156(4):569-76.

37. Taylor D.J. et al. Comorbidity of chronic insomnia with medical problems. Sleep. 2007 Feb 1; 30(2):213-8.

38. Tang, N.K. Prevalence and correlates of clinical insomnia co-occurring with chronic back pain. Journal of Sleep Research. 2007 Mar;16(1):85-95.

39. Koechlin, H. et al. The role of emotion regulation in chronic pain: a systematic literature review. Journal of Psychosomatic Research. 2018 Apr 1; 107:38-45.

40. Jackson, T. et al. Self-efficacy and chronic pain outcomes: a meta-analytic review. The Journal of Pain. 2014 Aug 1; 15(8):800-14.

41. Norstodga, A.L. et al. The influence of multisite pain and psychological co-morbidity on prognosis of chronic low back pain: longitudinal data from the Norwegian HUNT study. BMJ Open 2017; 7(5) eo,5312. Published on line 2017 Jun 6;7(5): e015312.doi:10.1136/bmjopen-2016-015312.

42. Lemmens, L.C. et al. Integrated care programmes for patients with psychological co-morbidity: a systematic review and meta-analysis. J of Psychosomatic Research 2015; Dec1; 79(6): 580-94.

Occupational Aspects of MSK Disorders

29. AFCS makes full and final awards for any disorder or injury predominantly caused by military service on or after 6 April 2005. To support consistency and equity, awards reflect not specific diagnoses but rather the disabling functional restriction or limitation due to the accepted disorder and its duration, particularly in the context of suitable civilian occupation. This is assessed following an adequate course of best practice clinical treatment and rehabilitation and so when the person is in an optimal medical and functional state. Awards also take account of mental health symptoms, short of a discrete disorder. The military goal is to maximise and maintain operational capability, and so from the outset, the management of MSK disorders in the armed forces is work-orientated, with clinical management addressing symptoms, supporting improvement and natural healing, while patients are reassured about the benefits of early active participation, expectation of recovery and return to work (1).

30. Physical disorders including MSK conditions or injuries are almost always accompanied by a degree of anxiety or low mood, and this may be influenced by factors such as how the injury occurred, how it was initially treated and how serious or disabling the injured person perceives it might turn out to be. Psycho-social factors are important in determining prognosis. These include the attitudes and beliefs of the injured person and of his or her family and colleagues. There may be issues of financial compensation involved or legal complications, and/or work issues such as poor job satisfaction, perceived lack of support at work, or even the possibility of bullying or harassment in the background. There may of course be co-morbid discrete psychiatric disorders such as depression. Some individuals may have a tendency to catastrophize, and that can induce a fear that movement or activity (because it is painful) will cause further damage or worsening of the pain or disability. This can lead to reduced physical activity, over-reliance on aids and appliances, and eventually in some cases to a disabled lifestyle of prolonged incapacity, decline in overall fitness and failure to return to work either in the military or civilian community.

31. In the past, treatment was based on the biomedical model, with doctors thoroughly investigating MSK problems and explaining to the patient how their pain was linked to physical damage, ideally “visible” as abnormalities on imaging. Reflecting this, MSK disorders are still commonly referred to as “injuries” although the great majority have insidious onset with no identified initiating event. Even where a sprain or strain has occurred, that often serves to draw attention to symptoms arising from an underlying pre-existing asymptomatic pathology. Correlation between MRI scan appearances of MSK disorders and lesions, let alone symptoms, is poor.

32. Orthopaedic, rheumatological and pain services in the NHS and military increasingly adopt a multidisciplinary biopsychosocial approach. Over-medicalisation is avoided, and key messages include the importance of self-management and active rehabilitation, maintained activity and retention in, or early return to, work. Because of the youth and general high level of physical fitness of military personnel, a stepped care approach is adopted from the outset, beginning with simple interventions supporting natural healing and importantly, providing information concerning the likely course. This will progress to more intensive clinical treatment should there be failure to respond. Modalities of treatment include manual therapy (physiotherapy and related disciplines), exercise therapy (with emphasis on rehabilitation and restoration of function) and, sometimes, other measures such as transcutaneous electrical nerve stimulation (TENS), acupuncture, ultrasound, and laser therapy, although the evidence for these is weak. Drug treatment will include simple analgesia and sometimes non-steroidal anti-inflammatory drugs (NSAIDs), but stronger analgesics should be avoided, as adverse effects frequently outweigh any benefits. Antidepressant drug treatment of associated depression may be appropriate, but again there is a risk of adverse effects. Adjunctive drug treatments such as

gabapentinoids (gabapentin and pregabalin) are increasingly used for MSK pain, but in the absence of a secure evidence base of their efficacy. Adverse effects are frequent and may negate any therapeutic benefits. For military personnel, temporary restrictions in employability grading and graduated return to work programmes as part of a "stepped approach" to rehabilitation may be appropriate. In some cases, return to the principal service occupation may not be possible and there will first be an effort to redeploy to a more suitable role within the military before, as a last resort, medical discharge back into civilian life.

33. Reintegration into civilian life for individuals with disabling musculoskeletal conditions can be challenging and not only for reasons directly associated with physical incapacity. All those who are medically discharged, regardless of length of service, are entitled to the full package of resettlement services, including training for suitable civilian employment, CV writing etc and transition support. Help is also available on housing, health and well-being, and financial advice. Currently these are not co-ordinated in a single policy. As part of the Veterans Strategy, a new unified Defence Transition Service delivered by Veterans UK is being rolled out. This is aimed at the most vulnerable service leavers and will provide co-ordinated, tailored one-to-one support as required, again on employment, health and well-being, housing and financial advice.

34. The underlying aim of the Defence Transition Policy is to enhance personal resilience and independence. Successful transition, long term clinical prognosis and securing and sustaining employment may be influenced by societal and psychosocial factors. Having experienced the "family" aspects of military life and perhaps expectations of a longer military career, a move back into civilian job-seeking can be daunting to some. Added to this are the vagaries of the labour market in which, despite high rates of general employment, the nature of jobs is changing fast and may not on the whole be to the advantage of young men and women exiting the military, particularly with short service and having failed to complete initial or trade training. Increasingly, repetitive and/or physical civilian work is being replaced by robotics or giving way to more jobs in the knowledge economy. Local unemployment rates in the areas of the country from where military personnel originate or settle may be higher than the average. Willingness, however, to accept physically disabled people into the workforce is improving and these efforts have had government support and legislative enforcement through the Equality Act (2010) and vocational rehabilitation schemes, such as Access to Work (2).

35. Musculoskeletal pain affects up to 50% of the civilian population at any one time and work, both physical and mental, sometimes modified, can be undertaken by most of the potential workforce. Recurrent back and neck pain present the biggest challenge to employers. Ideally a functional assessment of the individual plus a consideration of exactly what the work entails will enable a good person/job fit. Not only can the work be modified: many adaptations to allow access to the work place and workstations, and flexible working arrangements, as well as aids and appliances, are now available to physically impaired workers.

36. Currently, evidence on the effectiveness, particularly long-term of UK military medical discharge policies is sparse. A recent paper on veterans and benefits may provide some insights (3). The report linked records from the King's Centre for Military Health Research (KCMHR) cohort study, which includes about 11,000 personnel entering service between 2003 and 2016, with DWP data on uptake of income replacement, unemployment and disability benefits. These service leavers were not differentiated in terms of type of discharge or in the case of those medically discharged, disorders leading to medical discharge. The maximum follow-up time since leaving service for study subjects was 12 years.

37. Information on about 8,000 veterans was studied. About 23% of them had received unemployment benefits at some point in the post service period. Most unemployment benefit (7%) was paid soon after service release while at two years post-service, only 1.5% were in receipt. On the other hand, rates of disability benefit uptake were slightly less frequent (5%) but tended to persist. The study identified

factors associated with receipt of unemployment and disability benefit. For both benefit types, being male, of lower rank, shorter service, less educated, as well as having claimed benefits before service, were relevant. The study also found that veterans with mental health disorders had a higher likelihood of claiming benefits. The study did not consider separately MSK problems or any other category of injury or disorder. Comparison with civilian benefit uptake is difficult owing to the limited comparison official statistical data and different demographics of the two groups. The researchers concluded that "…. veterans are, at the very least, not at higher risk of benefit claims than the general population".

References:

1. Burton, K et al. Musculoskeletal Disorders chap.8 in Snashall, D. et al.(eds.) ABC of Occupational. and Environmental medicine. 3rd.edition. John Wiley and Sons, 2012. pp 45-50.

2. Get help from Access to Work 2019 available from www.gov.uk/access to work.uk.

3. Burdett, H. et al. (2018). Veterans and Benefits a report by Kings Centre for Military Mental Health Research ((KCMHR) for the Forces in Mind Trust.

MSK Disorders - Part 2 Section B - Further Clinical Summaries

Introduction

This section of the paper follows from MSK Part 1 (2017), considering additional MSK diagnoses seen in a serving military population. Topics covered include lower limb exertional tendinopathy; iliotibial band syndrome; shin splints (or medial tibial stress syndrome); stress fracture; compartment syndrome; plantar fasciitis; Lis Franc injury; hypermobility and Ehlers-Danlos syndrome. Complex regional pain syndrome (CRPS) is also included here.

Topic 1 - Lower Limb Tendinopathy including Damage to Achilles, Patellar and Hamstring Tendons, and Lateral Trochanteric Pain

a) Achilles Tendinopathy

1. DEFINITION: The Achilles tendon is formed from the tendons of the gastrocnemius and soleus muscles and inserted into the calcaneal tuberosity. Achilles tendinopathy covers the range of overuse injuries which may affect the tendon ranging from mild tendon irritation to complete rupture. The disorder is common as a sporting injury, particularly in middle-aged recreational distance runners or those who play tennis, squash or soccer, especially on an occasional basis (1).

2. CLINICAL: The tendon is a relatively avascular structure throughout its length (2). This makes it liable to be damaged by repetitive trauma over time, leading to thinning, degeneration and ultimately, possible rupture. Tendinopathy develops insidiously. Various stages in development are described, affecting different parts of the tendon and leading to pain, fullness or rupture. Pain is usually mid-structure or at the insertion. Partial Achilles tears are very rare and, counter-intuitively, although tears only occur in

damaged tendons, few tears occur in patients with a history of tendinopathy symptoms (3). Pain tends to be present on waking or after rest or there is pain on activity which settles over time.

3. AETIOLOGY: The precise cause of Achilles tendinopathy remains unknown. Although well studied in the sports context where it is often associated with squash, badminton or running, there are few occupational studies. Proposed risk factors include age, and a number of congenital anomalies of foot and lower limb are thought to occur more frequently with Achilles tendinopathy, e.g. Varus forefoot and foot hyper-pronation. Overuse or poor technique are possible sports-related risk factors e.g. poor conditioning, training errors, failure to warm up, over-training and training in adverse weather conditions (4). Achilles tendon fibres may be damaged by diabetes and the medical use of corticosteroids eg for asthma or auto-immune disorders.

4. TREATMENT AND PROGNOSIS: Best practice treatment for Achilles tendinopathy remains to be fully determined but it is known that early treatment, of whatever nature, leads to a more favourable outcome. Tendinopathy without rupture is treated with avoidance of the provoking cause and further overuse. However, complete rest is not advised, but rather active physiotherapy with massage, ultrasound, eccentric lengthening of the Achilles tendon with load-bearing, and orthoses to correct abnormal foot alignment.

5. Usually the approach is initial rest, management of tendon loading, followed by physiotherapy, even for rupture. If conservative management fails, surgical treatment may be appropriate. Both conservative and operative treatments produce reasonably successful results with, in most cases, patients being able to return to previous levels of activity (6).

References:

1. Paavola, M. et al. Achilles tendinopathy. J. Bone and Joint Surgery. 2002; 84-A (1):2062-76.

2. Ahmed, I.M. et al. Blood supply of the Achilles tendon. J. Orthop. Res. 1998; 16(5):591-6.

3. Maffulli, N. et al. Overuse tendon conditions: time to change a confusing terminology. Arthroscopy. 1998; 14:840-3.

4. McCrory, J.T. et al. Aetiologic factors associated with Achilles tendonitis in runners. Med. Sci. Sports. Exer. 1999; 31:1374-81.

5. Maffulli, N. et al. Achilles tendinopathy: aetiology and management. J. R. Soc. Med. 2004 Oct; 97(10):472-476.

6. McLauchlan, G. J. et al. Interventions for treating acute and chronic Achilles tendinitis. (Cochrane Review). From the Cochrane Library. Issue 4. 2003. Chichester UK. John Wiley and Sons Ltd.

b) Other Less Common Tendinopathy Variants

6. These include patellar tendinopathy where pain is at the inferior pole of the patella and similar in pattern to Achilles tendinopathy ie present on waking, or activity, which settles with time. It is common in running, jumping and tabbing and may be triggered by a period of intense activity. Pain from hamstring tendinopathy occurs after a period of intense loading or activity with pain radiating from lower buttock into the back of the thigh. There may be localised tenderness at the ischial tuberosity. Lateral trochanteric pain is more common in women. This tends to have insidious onset, is localised around the greater trochanter radiating down the lateral thigh. Physiotherapy is key (1).

Reference:

(1) Nicol, A. et al. DDR Best practice guidelines - management of lower limb tendinopathy. MOD. London. 2018

Topic 3. Exertional Lower Limb Pain e.g. Ankle Sprain - Medial Tibial Stress Syndrome - Compartment Syndrome - Stress Facture

1. Exertional or exercise induced lower limb pain is the subject of a Defence Primary Quality Standard Indicator and Best Practice Guideline whose aim is to enhance accurate diagnosis and consistent quality management with early return to duty. The guideline uses anatomical boundaries to improve injury categorization. Ankle sprain is common in UK recruits and of note because such injuries may require significant rehabilitation times.

a) Shin Splints or Medial Tibial Stress Syndrome

1. Shin splints or medial tibial stress syndrome is a relatively common overuse injury resulting in lower limb pain on exercise, usually running or marching. It is associated with an increase in intensity or duration of strenuous exercise and affects ballet dancers, athletes and military recruits. In US and UK basic military training about 5% of recruits may be affected and shin splints account for about 10% of injuries in runners. Rates are lower where there has been pre-enlistment physical training (1). No universally agreed definition has been proposed.

2. CLINICAL: Symptoms are mainly musculotendinous pain affecting the postero-medial border of the tibia mid-leg. Pain is brought on by exertion and thought to be due to stress of the bone or attached muscle. The differential diagnosis of shin splints includes stress fracture and compartment syndrome both of which must be excluded, as well as lumbar spine disorder.

3. AETIOLOGY: There remains controversy as to the underlying pathology eg ischaemia or microfracture or soft tissue injury, but most clinicians agree that shin splints are a periostitis along the tibia caused by musculotendinous strain and inflammation. The site of tenderness to palpation is the medial origin of the soleus muscle and the pain may stretch over much of the tibia. This is in contrast to a stress fracture characterised by often exquisite but much more localised pain. MRI scans are the preferred method of evaluating and distinguishing these injuries. Shin splints occur when there is a change in a person's level of activity. Unconditioned people, or those with a previous injury are most at risk, with the most common contexts, running and military recruit training. Maximal pronation velocity and pronation, both of which result in increased stress on the lower limb musculature, correlate with shin splints. Other factors include excessive running distances especially on hard surfaces. Body build and anatomical variation such as genu valgum or pes cavus may also increase risk. There is no recognised gender difference in incidence or prevalence (2).

4. PREVENTION: A number of prevention measure have been advocated e.g. warming up, well-fitting footwear, avoidance of training in poor, especially cold and wet, conditions. A few preventative measures have been trialled in military establishments, but results have been disappointing and there have been study limitations (3). There is some evidence that shoe orthoses may prevent the development of shin splints, but any benefit needs to be weighed against cost (4).

5. TREATMENT AND PROGNOSIS: Treatment is usually conservative, focusing on relative rest and pain free activity only, for about 10 days, and then a gradual return to full activity over 6-8 weeks. Where the problem is resistant or recurrent, despite adequate management, surgical release of the fascia surrounding the soleus insertion is the intervention of choice usually achieving high levels of pain reduction and patient satisfaction (5).

References:

1. Andrish, J.T. et al. A prospective study on the management of shin splints. J. Bone Joint Surg. Am. 1974; 56:1697-1700.

2. Hester, J.T. Diagnostic approach to chronic exercise -induced leg pain. A review. Clin. Podiatr. Med. Surg. 2001; 18:285-306.

3. Thacker, S.B. et al. The prevention of shin splints in sport: a systematic review of literature. Med. Sci. Sports Exerc. 2002; 34:32-40.

4. Larsen, K. et al. Can custom made biomechanical shoe orthoses prevent problems in the back and lower extremities? A randomized controlled intervention trial on 146 military conscripts. J. Manipulative Physiol. Ther.2002; 25(5):326-31.

5. Yates,B. et al. Outcome of surgical treatment of medial tibial stress syndrome. J. Bone Joint Surg. Am. 2003; 85-A (10):1974-80

b) Other Less Common Tendinopathy Variants

1. Compartment Syndrome has two forms, Acute Compartment Syndrome (ACS), which can be limb and life threatening and a medical emergency and the more benign, Exertional form (ECS) (1).

2. DEFINITION: ACS occurs when there is increased tissue pressure within a bounded myofascial compartment. This compromises the vascular supply and so the function of the contained structures, muscles, nerves and blood vessels. Normal lower limb compartment pressure is 10-15 mmHg. Serial monitoring is the usual approach for diagnosis. Pressure above 40 mmHg diagnoses ACS while pressures in the 20-30 mm range are indicative.

3. In ECS there is exertional limb pain during and after exercise. This may be accompanied by paraesthesiae and swelling. The underlying mechanism is thought to be ischaemia but remains to be established.

4. AETIOLOGY: ACS is associated with increased volume within the limb compartment e.g. due to haemorrhage, haematoma or oedema and is seen most commonly in relation to high energy or penetrating trauma, especially comminuted fractures of the tibia. Other possible causes are crush injury, burns, intra-arterial injection and venous obstruction. The compartment may be reduced in size by lying on a limb or tight dressings or casts (2).

5. The pathology of ECS is unknown but again structures in a closed myofascial compartment are compressed. Exercise leads to increased muscle bulk and the associated muscle contraction may raise pressure leading to a degree of ischaemia and reduced oxygen supply. Other experts link ECS to oedema where there is sustained high intensity training e.g. running on hard surfaces causing increased osmosis and oedema (3).

6. CLINICAL: (1) ACS. Limb compartments are most at risk, but any muscle mass confined by fascia can be affected. The key features are pain, apparently disproportionate to any trauma and aggravated by passive muscle stretching. This produces tightness and tenderness. Later there may be hyperaesthesia and muscle weakness with, very late on, tissue necrosis, irreversible muscle injury and contractures. ACS is a medical emergency best treated by fasciotomy within 6-12 hours of symptom onset sometimes with interval skin graft to close the incisions.

(2) ECS most frequently involves the lower limb and often occurs in high intensity athletes, notably long-distance runners. The clinical features of pain, cramps and paraesthesia usually occur after a certain time or distance run. Symptoms are usually bilateral and remit within about an hour of stopping the activity but recur when exercise is resumed. Pain is also increased by both passive and active movement. It may be useful diagnostically to measure compartment pressure at rest and on exercise. Typical pre-exercise pressures are more than 14-15 mmHg: 1-minute post-exercise we would expect a pressure of more than 30 mmHg and at 5 minutes post-exercise, pressure should have decreased to no more than 19-20 mmHg.

7. TREATMENT AND PROGNOSIS: (1) ACS. The success of treatment depends on early diagnosis and surgery and the presence of complications, which are more common where there is treatment delay. Infection can be a problem, its presence increasing the likelihood of amputation. Rare problems include renal failure, disseminated intravascular coagulation and respiratory distress syndrome (4).

(2) ECS. Surgical fasciotomy is the treatment of choice with usually high patient satisfaction and up to 85% patients able to resume pre-treatment levels of activity, although owing to fascia removal, there may be some muscle weakness. The response to surgery where the posterior compartment is involved is less good than for anterior or lateral compartments (5).

References:

1. Velmahos, G.C. et al. Vascular trauma and compartment syndromes. Surg. Clin. of North Am. 2002; 82(1):125-41.

2. Pearse, M. F. et al. Acute compartment syndrome of the leg. Br. Med. J. 2002; 89:397-412.

3. Schissel, D.J. et al. Effort related chronic compartment syndrome of the lower extremity. Mil. Med.1999; 164(11):830-2.

4. Tiwari, A. et al. Acute compartment syndromes. Br. J. Surg. 2002; 89:397-412.

5. Turnipseed, W.D. et al. Diagnosis and management of chronic compartment syndrome. Surgery 2002; 132(4): 613-7.

c) Stress Fracture

1. Stress Fractures, also called atraumatic/incomplete fractures are associated with bone fatigue, due in a military context to high exercise stress or bone insufficiency such that normal stress can cause damage. Stress fractures comprise 10% of all sports injuries and are often associated with running. They are seen particularly in recruits as opposed to trained strength military personnel, are more common in women and typically affect the lower limb. The mechanism is thought to be that the bone responds to muscle pull and becomes deformed. If the force exceeds the bone elasticity, microfracture occurs. Suspected stress fractures should have MRI which will show osteitis of bone. X-ray changes typically take some weeks to become visible.

2. Extrinsic factors increasing risk of stress fractures include marked change in level and intensity of training, sustained intensive training, worn or unsuitable, ill-fitting footwear, hard training surface, low dietary vitamin D/calcium and muscle fatigue while intrinsic factors include low bone density, low Basal Metabolic Index, anorexia and cigarette smoking.

3. An important aspect of management is prevention of further fracture. This is achieved by prescribing rest until symptoms subside then gradually building up to the previous level of activity. This usually takes three to twelve weeks. In some cases, surgical correction may be required. High risk sites for stress fracture include the femoral neck (supero-lateral), anterior tibia, medial malleolus, talus, proximal 5th metatarsal, navicular bone, and sesamoids. The femoral neck factures may require surgery and have a long rehabilitation period (in excess of 100 days). This makes them a very significant injury particularly in recruits. They often lead to back-coursing and ultimate medical discharge. Low risk sites include the fibula where management is conservative.

Reference:

1. Asturt, D.A. et al. Stress fractures; definition, diagnosis and treatment. Rev. Bras. Ortop. 2016; 51(1):3- 10.

Miscellaneous Diagnoses

a) Plantar Fasciopathy

1. Plantar fasciopathy is the commonest cause of inferior heel pain. Again, it is now known it is not an inflammatory disorder, so the term fasciopathy is preferred to fasciitis. There is high risk in middle aged women, especially if they stand at work e.g. cooks and chefs and if they have flat feet, have gained weight or have a foot biomechanical abnormality. It is also common in the recruit population e.g. young, predominantly male, runners. While usually self-limiting by 6 weeks, 10% go on to develop chronic pain. In terms of interventions the disorder may respond to plantar fascia stretching, over-the-counter orthotics or extracorporeal short-wave therapy. There is no published randomised controlled trial, but patients report satisfactory outcomes from short wave therapy (1), (2).

References:

1. Martinelli, N. et al. Current therapeutic approaches for plantar fasciitis. RR. 2014; 6:33-40.

2. Singh, D. et.al. Plantar fasciitis. Fortnightly review Brit. Med. J. 1997; vol 315:172-175.

b) Iliotibial Band Syndrome (ITBS)

1. ITBS is considered one of the most common overuse injuries in the lower limb. It is seen in runners but also in cyclists, soccer and hockey players. It causes hip, lateral thigh and knee pain and leads to inability to participate in sport. In runners, lateral knee pain may be reported with onset around a consistent mileage which requires them to stop. The aetiology remains poorly understood with the theoretical model of friction between the iliotibial band and lateral femoral condyle when the knee is partially flexed, not borne out by cadaver studies (1), (2). Abnormal lower limb biomechanics have also been implicated but results have been conflicting with diametrically opposite results on hip internal rotation and adduction and hip abduction muscle strength in runners with a history of ITBS (3), (4).

2. Because of failure to identify the underlying mechanisms, there is at present no best practice treatment. Patients often report pain and limitation at adjoining areas e.g. low back and hip or abdomen. On examination there may be reduced strength in hip flexors, external rotators and abductors as well as limited low back active range of motion. These observations have led to multifaceted treatment protocols involving the lumbar spine and lower limb regional treatment as well as information and education about shoes, training surfaces, warming up and training schedules. Using these techniques there is good evidence of early response e.g. in one series response within at most eight weeks following a programme of hip abductor and external rotator strengthening with return to normal activity. This was maintained at one year (5).

References:

1. Strauss, E. J. et al. Iliotibial band syndrome: evaluation and management. J of the Am. Ac. of Ortho. Surgeons 2011; 19,12:728-36.

2. Lavine, R. Iliotibial band friction syndrome. Curr. Rev. Musculoskeletal Med. 2010; 3(1-4):18-22.

3. Grau, S. et al. Hip abductor weakness is not the cause for iliotibial band syndrome. Int. J. Sports Med. 2008; 29(7):579-583.

4. Noehren, B. et al. Prospective study of biomechanical factors associated with iliotibial band syndrome. Clin. Biomech. 2007; 22:951-956.

5. Khayambashi, K. et al. The effects of isolated hip abductor and external muscle strengthening on pain, health status, and hip strength in females with patellofemoral pain; a randomised controlled trial.
J. Orthop. Sports. Phys. Ther. 2012; 42(1):22-29.

c) Piriformis Syndrome

1. Piriformis Syndrome remains a controversial diagnosis and there is variability in the use of the term. Some reserve the diagnosis for pain associated with neurological signs indicating sciatic nerve compression by the piriformis muscle, but this is a very rare presentation. Others use the term to refer to pain in the buttock, presumed to originate in the piriformis muscle and other muscles acting as external rotators at the hip joint. Buttock pain is usually more prominent than low back pain. Characteristic features include i) tenderness in the region of the greater sciatic notch; ii) pain which is aggravated by sitting; and iii) pain increased by external rotation at the hip joint. There is no proven effective treatment (1), (2).

References:

1. Hopayian, K. et al. The clinical features of the piriformis syndrome: a systematic review. Eur.Spine J. 2010; 19:2095-2109.

2. Cass, S. Piriformis syndrome: a cause of non-discogenic sciatica. Curr. Sports Med. reports. 2015; 14 issue 1:41-44.

d) Groin Pain Syndrome

1. Although the term, Groin Pain Syndrome is frequently used it is not a single discrete diagnosis with defined criteria but simply refers to pain in the groin. It is a common sporting complaint, particularly in soccer. There are multiple causes eg hip pathology (particularly osteoarthritis), femoro-acetabular impingement, labral tear and reduced hip muscle strength or loss of range of movement e.g. due to osteitis pubis. In any one patient multiple causes may co-exist and contribute to the symptoms. Differential diagnoses include iliotibial band syndrome (ITBS), rectus adductor tendinopathy, i.e. stress or imbalance between adductor and abdominal muscles, groin injury e.g. stress fracture, tendon avulsion, nerve entrapment syndrome affecting the ilio-inguinal nerve, intra-abdominal pathology, and referred pain e.g. from lumbar spine, sacroiliac joint or pubic bone.

2. The disorder should be investigated by imaging and clinical evaluation. There may be difficulty in precise diagnosis and identification of aetiological factors in any one case. Multiple approaches to treatment, both physiotherapy and surgical may be required and even then, the disorder may not fully remit (1).

Reference:

1. Bisciotti, G. et al. Groin pain syndrome: an association of different pathologies and a case presentation. Muscle Ligt. and Tendons Journal. 2015; 5(3): e14-22.

e) Lisfranc Injuries

1. Lisfranc injuries form a spectrum of acute, usually but not always, high energy traumatic injuries involving bony or ligamentous compromise of the tarsal, metatarsal and inter-cuneiform foot joint complex. If diagnosed early and treated appropriately they usually have a good outcome. They demonstrate a wide range of severity. They may be stable, or a partial sprain, or a grossly displaced and unstable fracture-dislocation of the midfoot.

2. Investigation is important in order to establish the diagnosis, and fractures may be missed on plain X-ray unless several views are obtained. MRI is the investigation of choice. Typical mechanisms are falls from a height or road traffic accident (RTA) but Lisfranc injuries may also be seen following relatively low energy sports-related sprains and strains in baseball, wind and kite surfing. The injury may involve ligaments only, bone fracture only, or a combination. It may be partial, and essentially a sprain, i.e. a stable injury seen where the mechanism of injury is via an axial load and plantar flexion. The plantar tarso-metatarsal ligaments remain intact. Clinically there may be pain and diffuse swelling in the midfoot and inability to weight-bear. Plantar ecchymosis at midfoot is highly suggestive of Lisfranc injury.

3. Stable variants are treated non-operatively with boot immobilization followed by weight bearing as pain permits. Often there is prolonged recovery time but generally recovery is complete with return to pre-injury activity. Unstable or displaced injuries or misdiagnosed injuries with delayed or inadequate treatment often develop persistent pain and post traumatic arthritis requiring arthrodesis. For unstable injury the treatment of choice is Open Reduction with Internal Fixation (ORIF) with trans-articular screw fixation. More recently ORIF and primary arthrodesis has become increasingly popular and effective. Where ligaments are involved this procedure has lower subsequent rates of surgery for removal of

the screws etc. Most patients return to previous levels of athleticism and sport with typical timeline of about a year. There are high rates of radiographic post traumatic arthritis and symptoms (1).

Reference:

1. Clare. M. P. Lisfranc Injuries. Curr. Rev. Musculoskeletal Med. 2017; 10:81-85.

f) Hypermobility and Ehlers-Danlos Syndromes

1. Joint hypermobility simply means that joints have an unusual range of mobility. Most people with this condition have no problems and may benefit from the flexibility e.g. ballet dancers, gymnasts and musicians. They may also experience pain and stiffness in joints and muscles, clicking joints, joints that dislocate or fatigue. Symptoms may be recurrent and similarly joints may be prone to sprains. The related Ehlers-Danlos syndrome affects 1 in 5000 people globally. This is not a single diagnosis but group of genetically determined connective tissue disorders. Features include joint pain, stretchy abnormal skin and scar formation, usually diagnosed at birth or in early childhood (1).

2. Patients with Ehlers-Danlos Syndrome may also have autonomic dysfunction, aortic dissection, lens dislocation, recurrent joint dislocation, scoliosis, chronic pain and early osteoarthritis. There is no cure (2), (3). In terms of MSK problems treatment is supportive. If dislocation occurs, such as patella or elbow, and there is structural damage the treatment of choice is surgery. If there is no structural damage surgery is not indicated (3).

References:

1. Byers, P. H. et al. Heritable collagen disorders: the paradigm of Ehlers-Danlos syndrome. J. of Investigative Derm. 2012; 132(E1): E6-11.

2. Camerato, F. et al. Heart rate conduction and ultrasound abnormalities in adults with joint hypermobility syndrome or Ehlers-Danlos syndromes. Clin. Rheumatol. 2014; 33(7):981-7.

3. Gedalia, A. et al. Joint hypermobility and fibromyalgia in school children. Ann. of the Rheum. Diseases. 1993;52(7):494-6.

g) Fibromyalgia and Myofascial Pain

1. Fibromyalgia Syndrome (FMS) and Myofascial Pain Syndrome (MPS) are enigmatic pain states, essentially diagnoses of exclusion that continue to defy clear definition (1). For research in these conditions, based on a consensus research study, the American College of Rheumatology (1990) established two essential criteria for FMS: a history of widespread pain of at least three months' duration and painful sensitivity to 4kg of digital pressure at 11 or more of 18 anatomically defined tender points (2). While these criteria meant that comparable patient groups were being studied in research on FMS, there was criticism that some other common associated features were not represented, including dysfunctional sleep, fatigue and cognitive dysfunction (3). A second criticism was that the allegedly diagnostic tender points were hard to standardise, and more related to distress than the illness itself. There have been several revisions of the diagnostic criteria (4), which have now discarded tender points and moved towards recognising chronic widespread pain (5).

2. Myofascial Pain Syndrome (MPS) presents similar diagnostic difficulties and is rarely diagnosed in UK military practice. In a large survey of members of the American Pain Society, 88% of responders considered MPS to be a valid clinical disorder, and 81% that it was different from FMS. Given the uncertainties concerning diagnosis of FMS and MPS, it is not surprising that there is little consensus on pathogenesis (6) or causation. Central sensitization (see paragraphs 20-23 above) seems very likely to play a part (7), but the factors leading to the development, severity and chronicity of these occasionally extreme states of pain and disability remain elusive. Evidence for effective management is accumulating (8), particularly for non-pharmacological approaches, such as exercise therapy and cognitive behaviour therapy (9).

References:

1. Russell, I. J. Chapter 48, pp 658-682. In Wall and Melzack's Textbook of Pain, 6th ed. Eds McMahon S.B. et al. Elsevier Saunders, 2013.

2. Wolfe, F. et al. The American College of Rheumatology 1990 Criteria for the classification of fibromyalgia. Report of the Multicentre Criteria Committee Arthritis and Rheumatism. 1990; 33:160-172.

3. Wolfe, F. et al. The relation between tender points and fibromyalgia symptom variables: evidence that fibromyalgia is not a discrete disorder in the clinic. Annals of the Rheumatic Diseases. 1997 April1; 56(4):268-71.

4. Mease, P. et al. Development of the fibromyalgia survey diagnostic criteria, a modification of the ARC (2010) preliminary diagnostic criteria for fibromyalgia. Annals of the Rheumatic Diseases 2010; 69:144.

5. Wolfe, F. et al. Revisions to the 210/11 fibromyalgia diagnostic criteria in Seminars in arthritis and rheumatism 2016 Dec1; vol 46, no3:319-329.

6. Grayston, R. et al. A systematic review and meta-analysis of the prevalence of small fibre pathology in fibromyalgia: implications for a new paradigm in fibromyalgia etiopathogenesis. In Seminars in arthritis and rheumatism. 2019 April 1; vol 48, no5: 933-940.

7. Bourke, J.H. et al. The common link between functional somatic syndromes may be central sensitisation. J. Psychosom. Res. 2015 Mar; 78(3):228-236.

8. Macfarlane, G. J. et al. EULAR revised recommendations for the management of fibromyalgia. Ann. of the Rheum. Diseases 2017 Feb 1; 76(2):318-328.

9. Thieme, K. et al. Evidence based guidelines on the treatment of fibromyalgia patients: are they consistent and if not, why not? Have effective psychological treatments been overlooked? The Journal of Pain. 2017 Jul1; 18(7):747-756.

h) Complex Regional Pain Syndrome (CRPS)

1. Complex Regional Pain Syndrome (CRPS) is the term now generally adopted to include a number of conditions previously known as reflex sympathetic dystrophy, causalgia, algodystrophy and Sudeck's atrophy. The clinical features include severe chronic pain associated with painful sensitivity of skin and deep tissues (hyperalgesia and allodynia), local disturbances of autonomic function (blood flow, temperature and sweating), swelling of the affected part (oedema) and severe loss of function, usually affecting the whole limb (1).

2. Exclusive definition of CRPS is difficult. CRPS describes a variety of painful conditions that usually follow injury, occur regionally, have a distal predominance of abnormal findings, exceed both in magnitude and duration the expected clinical course of the inciting event, often result in significant impairment of motor function, and show variable progression over time.

3. In CRPS type 1, there is an initiating noxious event. Ongoing pain and hyperalgesia occur beyond the territory of a single peripheral nerve; there is or has been clinical evidence of abnormal blood flow, temperature and sweating; and the diagnosis is excluded if there is an alternative condition that would explain the symptoms and signs. CRPS type 2 follows a clearly identifiable nerve injury, usually of a major limb nerve, and is synonymous with causalgia. It is more regionally confined than CRPS type 1, but in other respects is similar to CRPS type 1. There is a neurological deficit appropriate to the affected nerve.

4. Being descriptive of symptoms and signs, these definitions suffer from lack of clarity concerning the definable limits of the conditions, particularly CRPS type 1, but they do have the advantage of avoiding unjustified assumptions about pathophysiology. Confusion also results from making CRPS type 2 and causalgia synonymous, emphasizing the difficulty arising from the use of a word originally intended to refer to a single symptom (causalgia means 'burning pain'), to a clinical syndrome. However, this is the widely accepted current terminology. There are now generally accepted diagnostic criteria for CRPS (2)(3)(4).

5. CRPS type 2 is very rarely seen in service. CRPS type 1 is uncommon but may occur following injury including fractures, tendon and ligament injury, fasciopathy, arthritis, deep vein thrombosis and prolonged immobilization. However, it is a rare consequence of all these conditions, and the factors leading to its development are unclear.

6. The pathogenesis and pathophysiology of CRPS remain uncertain. Initial and possibly persistent inflammation, immune mediated, is now generally thought to be an important factor. Central sensitization undoubtedly occurs, and there is some evidence supportive of a reorganized state at several levels within the central nervous system that could potentially become irreversible (5). Treatment is always difficult and demands a coherent multidisciplinary approach, including systemic drug treatment, local physical measures, psychological management, usually cognitive behaviour therapy, and family involvement. Some evidence suggests that early intervention yields a better prognosis, but for some, recovery is limited (5)(4).

References:

1. Veldman, P.H. et al. Signs and symptoms of reflex sympathetic dystrophy: prospective study of 829 patients. Lancet .1993 Oct 23; 342(8878):1012-6.

2. Pergolizzi, J. et al. The Budapest criteria for complex regional pain syndrome: the diagnostic challenge. Anaesthesiol. Clin.Sci.Res. 2018; 2(1):1-10 rES

3. Harden, R.N. et al. Validation of proposed diagnostic criteria (the Budapest criteria) for Complex Regional Pain Syndrome. Pain. 2010 Aug; 150(2): 268-274.

4. Birklein, F. et al. Complex regional pain syndromes up-to-date. Pain Rep. 2017 Oct.5; 2(6): e624. doi:10.1097/PR9.000000000624.

5. Giermuhlen, J. et al. Mechanism - based treatment in complex regional pain syndromes. Nat. Rev. Neurol. 2014 Sep; 10(9):518-28.