Acute musculoskeletal pain and acute spinal pain

Musculoskeletal injury may be present in any patient who has sustained other trauma, and it is managed according to the extent of injury and other injuries. In this section, focus will be on the musculoskeletal aspect alone.

Patients presenting with *spinal pain* (neck, thoracic, lumbar or sacral) must be evaluated by taking a thorough systemic history followed by a clinical examination. Take note of the following: the type of pain (duration, location, constant, intermittent, referred, spreading), chills and fever, local tenderness, physical activity, overuse injury, other skeletal pains, age (aortic aneurism), neurological complaints (spreading or referred pain, numbness, spasticity, weakness), GI complaints (peptic ulcer, jaundice, weight loss, faecal incontinence), genitourinary system (haematuria, incontinence, difficulty to pass urine) and haematological problems (anaemia, bruising). Also examine the breasts and thyroid.

Acute musculoskeletal pain usually follows injury (muscle injury, ligament injury, fracture, haemarthrosis or septic arthritis), overuse, gout and exacerbations of inflammatory arthropathies, such as rheumatoid arthritis and osteoarthritis. However, less common but very important causes must be kept in mind, including malignancy and haematological disorders.

The same approach is followed as with spinal pain. Usually, the diagnosis is obvious from the age of the patient and their history. Important information suggested in the history should not be overlooked. For example, an elderly person presenting with acute hip pain after a fall may have a fractured hip, but why did they fall? *Dizziness and falls* could be caused by, among others, autonomic neuropathy, diabetes mellitus, cardiac disease, carotid disease, epilepsy and drugs such as antihypertensive agents and analgesics (tramadol and meprobamate-containing preparations). Remember, shoulder pain may be referred cardiac pain.

Sports injuries

These are usually associated with musculoskeletal injury and pain and include skeletal injury (stress fractures, fractures, sprains, strains, joint dislocations, muscle haematomas, muscle rupture, etc.). These injuries may heal completely leaving no pain; others may be complicated by long-term morbidity, including dysfunction and chronic pain. Yet, the pain associated with sports injury is not always of a musculoskeletal nature. The *apparent good health* of persons presenting with sports-related pain may be misleading. Moreover, sports-related pain may not be of (the expected) musculoskeletal origin or even sports-related. Pain may also be out of proportion to the injury.

Patients may present with *headache*. Primary headaches (migraine, tension headache and cluster headache [CH]) occur commonly, but secondary headaches should always be suspected in a patient partaking in contact sports. These are trauma-related, vascular or nonvascular

types, or infection and substance abuse. Therefore, persons presenting with sports-related pain should be evaluated by taking a history, a clinical examination, and appropriate special investigations regarding the following:

- *Anatomical*: What sports were played (e.g., contact and collision sports, athletics, running, boxing, wrestling, rock climbing, surfing)? Look for associated injury, such as nerve injury, spinal injury, abdominal injury, thoracic injury, among other. Pain is also not limited to musculoskeletal injury (e.g. surfing complicated by sunburn, bites and stings, ocular trauma, and (important, but rarely) paraplegia due to vascular-related myelopathy).
- *Physiological*: Apart from the obvious or expected musculoskeletal pain, look for other functional disturbances, such as hyper- or hypothermia, neurological (change in consciousness, headache, weakness, paralysis, loss of sensation, pain out of proportion to the injury, radiating or referred pain), CV (dehydration, hypertension, dyspnoea, dysrhythmias, chest pain, blood pressure, pulses, compartment syndrome), respiratory (dyspnoea, chest pain), abdominal (abdominal pain, tenderness, groin pain) and genito-urological (flank pain, haematuria, testicular torsion).
- *Pharmacological*: What has the patient taken for pain? Has the patient taken any analgesics (opioids, NSAIDs, glucocorticoids)? Is there an indication of drug abuse (analgesics (opioids), sedatives (alcohol, benzodiazepines), hormones (anabolic steroids, thyroxine), psychostimulants and diuretics)? Does the patient display any *drug-seeking behaviour*? Athletes usually believe that illicit drug use has pleasant short-term consequences, such as increased physical and mental ability, but usually deny long-term detrimental health consequences. *Alcohol* is often abused and affects several neurological functions, which affects performance, memory, vigilance, reaction time and dexterity. They may also present with sports-related pain due to loss of muscle strength, and incomplete and delayed recovery. Drug abuse has also been implicated in serious adverse effects, including sudden death during competition. Persons taking anabolic steroids usually also use several other substances. They have often been diagnosed with a substance dependence disorder, and often suffer from psychiatric illness. These factors will influence pain management. Therefore, the practitioner who treats sports-related pain must have a sound knowledge of all the drugs used and abused by these patients; including the indications, adverse effects and abuses.
- *Special investigations:* The history and clinical examination will usually be straightforward or may need further investigation to dictate further management.

Professional or serious athletes may be demanding patients. Keep notes of the history, clinical findings, and special investigations, and refer the patient for further investigation or management. Fortunately, the majority of patients seeking help for sports-related pain are uncomplicated, undemanding, 'normal' patients. They can be treated with the usual analgesics.

Pharmacotherapy of sports injury

Paracetamol

Paracetamol is always prescribed. It should be taken around the clock at a dose of 15 mg/kg p.o. 6-hourly.

NSAIDs

Tissue injuries (including fractures) triggers the *inflammatory cascade* starting with phospholipase A2 (PLA2) that starts the formation of prostanoids. Apart from mediating nociception, these mediators of the inflammatory process initiate the repair process by hyperaemia, and recruitment of inflammatory cells that secrete mediators that promote the formation of granulation tissue, collagen formation by fibroblasts, and vascularisation. This early surge in the inflammatory response lays the foundations for eventual healing and return of function. Therefore, early NSAIDs inhibit the early inflammatory response and can impair healing. The clinical impact of this theoretical negative impact of NSAIDs on fracture healing is not clear.

Regarding fractures, retrospective studies have implicated the use of NSAIDs in delayed healing, delayed union, and nonunion of long bone fractures. It has been advised that NSAIDs should be avoided in complete fractures and stress fractures. If an NSAID is prescribed, the dose and duration should be limited as far as possible.

Healing of ligaments takes place in three phases, namely an early inflammatory response, the proliferation phase which is responsible for collagen formation, and finally remodelling. This phase may take up to a year. In one study, patients who received an NSAID for an ankle sprain, had less pain, decreased swelling and earlier return to activity. However, these ankles demonstrated less stability and range of movement. The faster return to activity may be due to analgesia, while joint instability and decreased range of movement points can be ascribed to blunting of the early healing process.

Findings of studies in animals regarding the effect of NSAIDs on ligament injury are conflicting. In humans, the effect on joint stability and range of movement is uncertain. Therefore, as is the case with fractures, an NSAID may be prescribed at the lowest effective dose for approximately 3–7 days. The emphasis should be on early immobilisation of the joint.

Healing of muscle injury also consists of three phases, namely inflammatory response, repair and remodelling. However, the inflammatory phase is accompanied by muscle destruction due to the proteolytic effect of inflammatory cells. Theoretically, by limiting proteolysis, NSAIDs may attenuate local tissue damage, limit pain, and promote healing and recovery.

NSAIDs are useful in muscle injury as these are not only analgesic, but also promote recovery, and earlier return to activity after muscle strain and eccentric injury (lengthening of the muscle during contraction). However, long-term use may be detrimental to repair and regeneration.

NSAIDs are useful in entrapment- or swelling-related disorders (e.g. carpal tunnel syndrome, interdigital neuroma, intervertebral disc prolapses, thoracic outlet syndrome, rotator cuff bursitis, trochanteric bursitis, and iliotibial band syndrome).

The adverse effects of NSAIDs should be discussed with the athlete, namely early advantages, risks and long-term outcomes.

NSAIDs are usually prescribed, taking into account its adverse effects and contraindications, including dehydration, kidney disease, ischaemic heart disease and peptic ulceration. The tNSAIDs are not necessarily safer than the coxibs. These drugs should not be prescribed to contact athletes on the day of competition. In the scenario of musculoskeletal injury, NSAIDs may be prescribed at the lowest effective dose for approximately 3–7 days.

Topical NSAIDs penetrate into soft tissues while systemic drug levels remain low. These are effective in relieving the pain associated with soft tissue injuries, without causing serious systemic or local adverse effects.

Corticosteroids

These agents are potent inhibitors of PLA2, which initiates the production of prostanoids. Corticosteroids have potent anti-inflammatory properties and are prescribed to avoid the well-known adverse effects of NSAIDs. For tissue repair (including scaring) to take place, it needs inflammation to bring along several mediators for granulation tissue and collagen formation, fibroblasts and vascularisation. Glucocorticoids inhibit these processes. Where inflammation is the primary process (not preceded by trauma, e.g., rheumatic and connective tissue disorders), corticosteroids are useful and are often prescribed for long-term use. However, secondary (injury-related) inflammation forms part of the healing process and corticosteroids may be prescribed for a short period of time. A short course (< 7 days) has not been shown to affect ultimate healing. Therefore, athletes should not receive these drugs during the competition season, where repeated injury is unavoidable and short-term repair is important.

Oral glucocorticoids are useful for acute radiculopathy and osteitis pubis, but should not be prescribed for noninflammatory degenerative conditions such as plantar fasciitis and Achilles tendinitis. Chronic back pain caused by disc herniation or radiculopathy can be treated with epidural corticosteroids.

Opioids

Besides the type of injury occurring in particular sports, sports injury is characterised by *repeated injury and pain*. The prescription of opioids every time an athlete seeks help, should therefore be done with circumspection to prevent abuse of opioids. All opioids are habit-forming and related to the number (milligrams) of MMEs – not the type of opioid (so-called weak and potent opioids). When prescribing opioids, the MME for the different opioids should be kept in mind to prevent inadvertent overdosing when switching between opioids.

The use of NSAIDs for sports injuries is controversial. Taking into account the (probable) effects of NSAIDs on healing, paracetamol and opioids are the only analgesics useful for the management of acute sports-related pain. However, the adverse effects of NSAIDs have not been demonstrated to impact significantly on the outcome, and are often prescribed. Paracetamol and the NSAID must be taken around the clock, while the opioid must be reserved for breakthrough pain. Physical measures are opioid-sparing (e.g. limb elevation and cold therapy to decrease swelling). A prescription for more than five days is usually unnecessary and it should never be prescribed for longer than 10 days without a review.

13.3.4 The discharge prescription from the emergency department

A patient should not receive a prescription for analgesics without a diagnosis. If a diagnosis is not clear, the patient must be referred for further evaluation as either an in- or outpatient. If the doctor is of the opinion that it is safe to discharge the patient, they must be advised on the symptoms to look for and instructed to return if necessary.

A prescription consisting of paracetamol and an NSAID is usually safe. The drugs should be taken around the clock for approximately 24 hours and longer if necessary. An opioid may be added for breakthrough pain or when NSAIDs are contraindicated. The number of opioid tablets/capsules should not exceed 10 units containing approximately 5 mg MMEs. This practice may decrease the availability of unused opioids that can land up in the hands of people who misuse opioids.

13.4 The intensive care unit

The duration of sedation and analgesia in the intensive care unit (ICU) should be limited to decrease the duration of ventilatory support and limit the complications thereof. To this end, the availability of well-trained staff, good monitoring and resuscitation equipment allow for more potent agents to be used more safely.

- *Multiorgan physiological derangements* may develop or exist in most patients. These abnormalities affect pharmacokinetics to some extent or may be aggravated by analgesics. Owing to large organ reserves, biochemical indicators may be within normal limits, but show a downward trend or deteriorate rapidly once the reserve has been exhausted. For example,