FIFTH EDITION

SPORTS AND SOFT TISSUE INJURIES

A GUIDE FOR STUDENTS AND THERAPISTS

Christopher M. Norris

ROUTLEDGE

Sports and Soft Tissue Injuries

The fifth edition of the retitled *Sports and Soft Tissue Injuries* sharpens its focus on the treatment of sports injuries, providing the most complete evidence-based guide for physiotherapists, sports therapists and medical practitioners working with athletes.

Opening with chapters that examine the underlying science of tissue healing and principles of rehabilitation, the book employs a systematic approach, with chapters covering each area of the body, from facial through to ankle and foot injuries. Every chapter includes in-depth discussion and guidance on the treatment of common sports injuries through physiotherapeutic modalities, drawing on the author's wealth of personal experience and the latest peer-reviewed research.

A complete pedagogical resource, *Sports and Soft Tissue Injuries* is highly illustrated in full colour, and features a companion website with video examples of therapeutic techniques and a frequently updated blog on current issues in sports injury treatment. It is an important text for students of sports therapy, physiotherapy, sport medicine and athletic training, interesting further reading for sport and exercise science or kinesiology students with an interest in sports injury, and a crucial reference for practising physiotherapists and athletic trainers and the related disciplines.

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A Guide for Students and Therapists

Fifth Edition

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CHAPTER 1

Healing

Following injury, tissue which has been damaged must be replaced by living material. Two processes are possible, regeneration and repair. With *regeneration,* tissue is replaced by the proliferation of surrounding undamaged tissue. Therapy to produce this effect is currently in its infancy with stem cell therapy. With repair, however, lost material is replaced by granulation tissue which matures into a scar (Watson 2016), a process which most commonly reflects healing seen within the field of sports and soft tissue injury.

Therapists need to have knowledge of the processes which occur at each successive stage of healing to be able to select the treatment technique which is most appropriate for the stage the subject is presenting. A technique aimed at reducing the formation of swelling, for example, would be inappropriate when swelling had stopped forming and adhesions were the problem. Similarly, a manual treatment designed to mobilize soft tissue may not be helpful when inflammation is still forming and the tissues are highly irritable.

The stages of healing are, to a large extent, purely a convenience of description, since each stage runs into another in a continuum, the previous stage acting to initiate the next. The term *phasing* rather than separate stages may be more suitable. Traditionally, the initial tissue response has been described as *inflammation*, but some authors see inflammation as a response separate to the processes occurring at the time of injury. Both injury and inflammation may be viewed as a reactive phase of injury, with the classical inflammatory period preceded by a short (ten-minute) period before the inflammatory mechanism is activated. The reactive phase may also be viewed as a lag phase (Hunter 1998), before the strength of the healing tissues begins to change. In any traumatic injury the initial stage is *bleeding*, which is the precursor for the inflammatory cascade seen as both a vascular and cellular response.

The second stage of healing has been variously called repair, proliferation and regeneration. The tertiary stage is normally termed remodelling. The terms injury, inflammation, repair and remodelling will be used in this text

When describing the stages of healing, the terms acute, subacute and chronic are helpful. The acute stage (up to 48 hours following injury) is generally the stage of inflammation. The subacute stage, occurring between 14 and 21 days after injury, is the stage of repair. The chronic stage (after 21 days) may be viewed as the stage of remodelling. The term chronic is also sometimes used to describe self-perpetuating inflammation, where

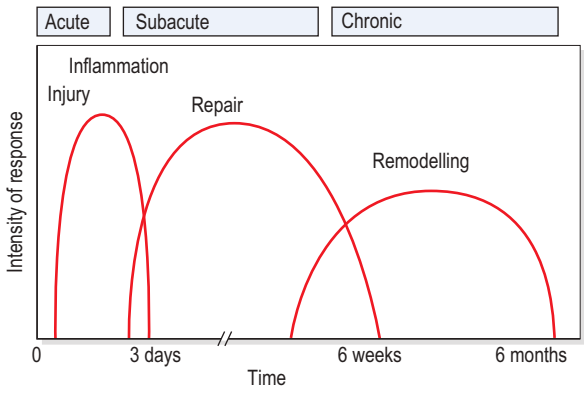

Figure 1.1 Timescale for healing. From Oakes, B.W. (1992) The classification of injuries and mechanisms of injury, repair and healing. In *Textbook of Science and Medicine in Sport* (eds J. Bloomfield, P.A. Fricker and K.D. Fitch). Blackwell Scientific Publications, Melbourne. With permission.

the inflammatory process has restarted due to disruption or persistent irritation of the healing tissues. The total healing process occurs over a continuum, shown in Fig. 1.1.

Keypoint

Treatment must be adapted to the stages of healing, which are injury, inflammation, repair and remodelling.

Injury

This stage represents the tissue effects at the time of injury, before the inflammatory process is activated. With tissue damage, chemical and mechanical changes are seen. Local blood vessels are disrupted causing a cessation in oxygen to the cells they perfused. These cells die and their lysosome membranes disintegrate, releasing the hydrolysing enzymes the lysosomes contained. The release of these enzymes has a twofold effect. First they begin to break down the dead cells themselves, and second, they release histamines and kinins which have an effect on both the live cells nearby and the local blood capillary network.

The disruption of the blood vessels which caused cell death also causes local bleeding (extravasated blood). More vascular tissue such as muscle will bleed more than less vascular tissue such as ligament. On average, bleeding following soft tissue injury stops within four to six hours (Watson 2016). The red blood cells break down, leaving cellular debris and free haemoglobin. The blood platelets release the enzyme thrombin, which changes fibrinogen into fibrin. The fibrin in turn is deposited as a meshwork around the area (a process known as walling off). The dead cells intertwine in the meshwork, forming a blood clot. This network contains the damaged area.

The changes occurring at injury are affected by age. Intramuscular bleeding, and therefore haemorrhage formation, is more profuse in individuals over 30 years of age. The amount of bleeding which occurs will be partially dependent on the vascularity of the injured tissues. A fitter individual is likely to have muscle tissue which is more highly vascularized, and therefore greater bleeding will occur with muscle injury. In addition, exercise itself will affect gross tissue responses. Muscle blood flow is greatly increased through dilatation of the capillary bed, and again bleeding subsequent to injury will be greater.

Keypoint

The tissues of an active individual are more highly vascularized than those of an inactive subject. The subject's tissues will therefore bleed more during injury, and bruising will be more noticeable.

Inflammation

The next phase in the healing sequence is that of inflammation, summarized in Fig. 1.2. This may last from ten minutes to several days, depending on the amount of tissue damage which has occurred, but generally reaches its peak by one to three days.

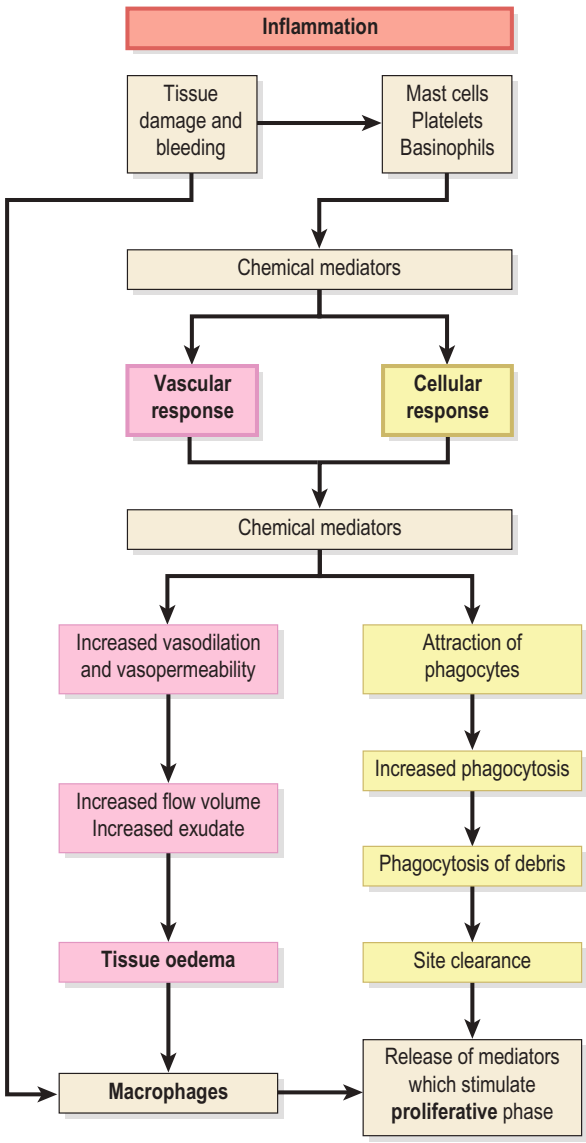

Figure 1.2 Inflammatory elements.

Vascular and chemical cascades occur in parallel to drive the inflammatory process.

Definition:

A *chemical cascade* (signalling cascade) is a series of chemical reactions. As one reaches its completion, it triggers the next in a type of 'chain reaction'.

The inflammatory response to soft tissue injury is much the same regardless of the nature of the injuring agent or the location of the injury itself. Inflammation is not simply a feature of soft tissue injuries, but also occurs when the body is infected, in immune reactions and with infarction. Some of the characteristics of the inflammatory response seen with soft tissue injury may be viewed as excessive and better suited to dealing with infection than healing injury.

The cardinal signs of inflammation are heat (*calor*), redness (*rubor*), swelling (*tumor*) and pain (*dolor*). These in turn give rise to the so-called fifth sign of inflammation: disturbance of function of the affected tissues (*functio laesa*).

Keypoint

Inflammation is often seen as undesirable. However, inflammation is the first stage of healing and so is a vital step on the road to recovery. The aim should be to prevent excessive inflammation and move the subject on through the phases of healing towards eventual full function.

Heat and redness

Heat and redness take a number of hours to develop, and are due to the opening of local blood capillaries and the resultant increased blood flow. Chemical and mechanical changes, initiated by injury, are responsible for the changes in blood flow.

Chemically, a number of substances act as mediators in the inflammatory process. The amines, including histamine and 5-hydroxytryptamine (5-HT or serotonin) are released from mast cells, red blood cells and platelets in the damaged capillaries and cause vessel dilatation and increased permeability. Kinins (physiologically active polypeptides) cause an increase in vascular permeability and stimulate the contraction of smooth muscle. They are found normally in an inactive state as kininogens. These

in turn are activated by the enzyme plasmin, and degraded by kininases.

The initial vasodilatation is maintained by prostaglandins. These are one of the arachidonic acid derivatives, formed from cell membrane phospholipids when cell damage occurs, and released when the kinin system is activated. The drugs aspirin and indometacin act to inhibit this change – hence their use as anti-inflammatory agents in sports and soft tissue injury treatment (see Treatment Note 1.1). The prostaglandins E1 and E2 will stimulate nociceptors and also promote vasodilatation, blood-vessel permeability and lymph flow.

The complement system, consisting of a number of serum proteins circulating in an inactive form, is activated and has a direct effect on the cell membrane as well as helping to maintain vasodilatation. Various complement products are involved, and these are activated in sequence. Finally, polymorphs produce leukotrienes, which are themselves derived from arachidonic acid, help the kinins maintain vessel permeability.

Treatment note 1.1 Medication used in soft tissue injury

Although inflammation is an essential part of the healing process, sometimes it can be excessive. Anti-inflammatory treatments are designed to limit inflammation and interfere with the chemical processes described above. Two groups of drugs are generally used in the treatment of soft tissue injuries in this respect: corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDS). Analgesics are used to limit pain, and may be used in isolation or together with anti-inflammatories.

Non-steroidal anti-inflammatory drugs

NSAIDS have both anti-inflammatory and pain relieving (analgesic) properties, causing both local (peripheral) and mild central effects. They inhibit the cyclo-oxygenase (COX) system, which has an important function in the cascade of chemicals driving the inflammatory process (see above) and works to block the production of prostaglandin. Two types are generally used, COX-1 and COX-2. As COX-1 also has an important function on the gastric mucosa, COX-1 inhibitors can lead to gastritis, and with long-term usage ulceration. COX-2 inhibitors have fewer effects on the gastric mucosa and so are better tolerated, but can increase the risk of thrombosis. NSAIDS are also available as creams and patches which can be used for superficial injury such as muscle injuries, contusions, and knee arthritis. Drugs such as *aspirin*, *Volterol*, *Brufen* and *Naprosyn* are common oral NSAIDS.

NSAIDS can inhibit protein synthesis and affect satellite cell activity, detrimentally changing muscle repair (see Chapter 2). They may alter collagen formation and fibroblast proliferation, so long-term usage should generally be avoided. In addition, tenocyte action during tendon repair may be negatively affected, but pain reduced (Pollock 2017). The role of prostaglandins in bone repair is also a potential concern with NSAID usage, as osteoblast activity may be impaired, delaying callus maturation in bone (Wheeler and Batt 2005).

Targeting pain

Painkillers (analgesics) work on the peripheral or central nervous systems. Drugs such as *paracetamol* have painkilling (analgesic) and fever-reducing (antipyretic) effects, but do not generally reduce inflammation. This type of drug works by blocking a type of cell membrane receptor called a cannabinoid receptor, which drugs such as cannabis work on. *Codeine*, *morphine* and *ketamine* are more powerful painkillers and are opiates. They may be taken alone or combined with paracetamol. Opiate drugs are generally derived from the opium poppy or its synthetic equivalent (one of which is heroin) and are

Treatment note 1.1 *continued*

psychoactive compounds – ones which alter mood or consciousness. As such, one of their side effects is nausea and dizziness. Where pain is from a peripheral nociceptive stimulus, NSAIDS may be effective at targeting pain indirectly by reducing the inflammatory chemicals driving nociception. Their painkilling effect is generally non-addictive, unlike the narcotic group of painkilling drugs such as *morphine*, above.

Where neuropathic pain and central sensitization occurs, medications such as *Gabapentin* (an anti-epileptic) and *Pregabalin* may be chosen as these block the nociceptive signal by binding to the calcium channels on the nociceptor and reducing neurotransmitter release. These drugs can induce fatigue and have a sedative effect so subjects should be aware of this. Locally, counterirritant effects may be provided by massage or self-applied rubifacient rubs. These may reduce nociceptor transmission by depleting neurotransmitter activity.

Corticosteroids

Corticosteroids (such as *triamcinolone* and *hydrocortisone*) also reduce inflammation, but rather than targeting prostaglandin, they reduce activation of leucocytes and alter vascular permeability. These drugs tend to be injected to the site of a pathology and, although generally effective at reducing both pain and inflammation in the short term, can have a number of negative effects. Inhibition of collagen synthesis may occur, impacting on tendon healing, an effect most studied in the case of tendinopathy presenting as tennis elbow (Coombes et al. 2010). Injecting into a contained region such as a joint can reduce synovial inflammation. However, cartilage matrix degradation may occur with prolonged usage in weight-bearing joints. Combining a corticosteroid with a local anaesthetic followed by a quick return to running is said to be detrimental to articular cartilage (Pollock 2017). Fat atrophy and alteration of skin pigmentation can also occur as a result of corticosteroid injection.

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Pollock, N. (2017) 'Therapeutic medication in musculoskeletal injury'. In: Brukner, P., Clarsen, B., Cooks, J. et al. (eds) *Clinical Sports Medicine.*

Wheeler P., Batt, M.E. (2005) 'Do non-steroidal anti-inflammatory drugs adversely affect stress fracture healing? A short review'. *British Journal of Sports Medicine* 39(2):65–69.

Blood-flow changes also occur through mechanical alterations initiated by injury. Normally, the blood flow in the venules, in particular, is axial. The large blood proteins stay in the centre of the vessel, and the plasmatic stream, which has a lower viscosity, is on the outside in contact with the vessel walls. This configuration reduces peripheral resistance and aids blood flow.

In a damaged capillary, however, fluid is lost and so the axial flow slows. Marginalization occurs as the slower flow rate allows white blood cells to move into the plasmatic zone and adhere to the vessel walls. This, in turn, reduces the lubricating effect of this layer and slows blood flow. The walls themselves become covered with a gelatinous layer, as endothelium changes occur.

Definition

Marginalization **is the build-up of white blood cells (leukocytes) on blood-vessel walls at the site of an injury.**

Some four hours after injury, diapedesis occurs as the white cells pass through the vessel walls into

the damaged tissue. The endothelial cells of the vessel contract, pulling away from each other and leaving gaps through which fluids and blood cells can escape (Fig. 1.3). Various substances, including histamine, kinins and complement factors, have been shown to produce this effect (Walter and Israel 1987).

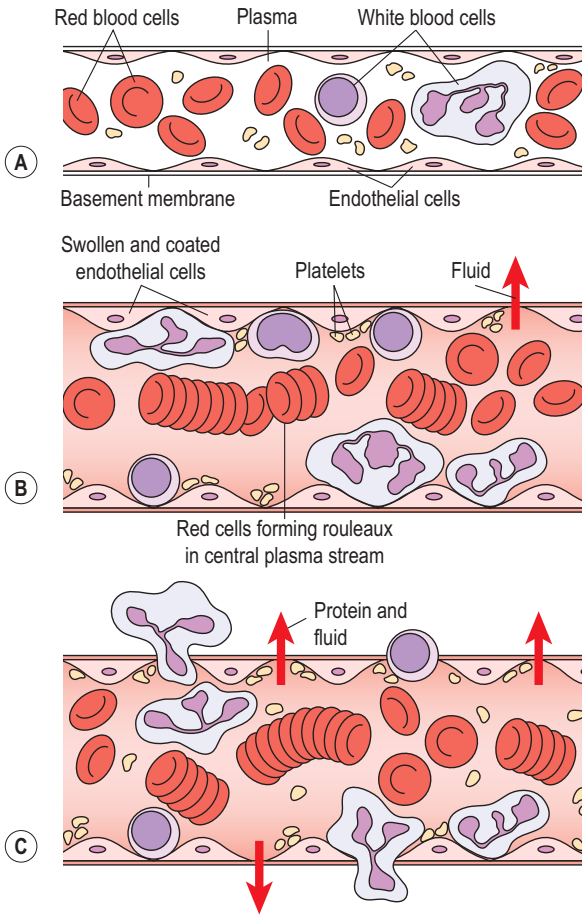

Figure 1.3 Vascular changes which occur in inflammation. (A) Blood vessel starts to dilate. (B) Dilated vessel showing marginalization. (C) White blood cells and fluid pass into tissue. From Evans, D.M.D. (1990a) Inflammation and healing. In *Cash's Textbook of General Medical and Surgical Conditions for Physiotherapists* (ed. P.A. Downie), 2nd edn. Faber and Faber, London. With permission.

Swelling

The normal pressure gradients inside and outside the capillary balance the flow of fluid leaving and entering the vessel (Fig. 1.4). The capillary membrane is permeable to water, and so water will be driven out into the interstitial fluid. However, because the tissue fluids usually contain a small amount of protein, and the blood contains a large amount, an osmotic pressure is created, which tends to suck water back from the tissue fluid and into the capillary once more. The magnitude of this osmotic pressure is roughly 25 mmHg. At the arteriole end of the capillary, the blood pressure (32 mmHg) exceeds the osmotic pressure and so tissue fluid is formed. At the venous end of the capillary, the blood pressure has reduced (12 mmHg) and so, because the osmotic pressure now exceeds this value, tissue fluid is reabsorbed back into the capillary.

During inflammation, the capillary bed opens and blood flow increases (heat and redness). The larger blood volume causes a parallel increase in blood pressure. Coupled with this, the tissue fluid now contains a large amount of protein, which has poured out from the more permeable blood vessels. This increased protein concentration causes a substantial rise in osmotic pressure, and this, together with the larger blood pressure in the capillary, forces fluid out into the interstitium, causing swelling.

Protein exudation in mild inflammation occurs from the venules only and is probably mediated by histamine. More severe inflammation, as a result of trauma, results in protein exudation from damaged capillaries as well.

During inflammation, lymphatic vessels open up and assist in the removal of excess fluid and protein. The lymph vessels are blind-ending capillaries which have gaps in their endothelial walls enabling protein molecules to move through easily. The lymph vessels lie within the tissue spaces, and have valves preventing the backward movement of fluid. Muscular contraction causes a pumping action on the lymph vessels and the

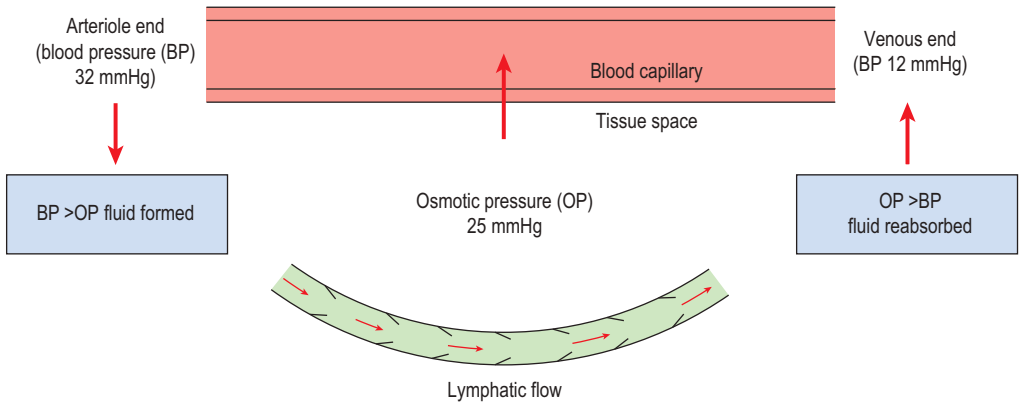

Figure 1.4 Formation and reabsorption of tissue fluid.

excess tissue fluid is removed to the subclavian veins in the neck.

Pain

Pain is the result of both sensory and emotional experiences, and is associated with tissue damage or the probability that damage will occur. It serves as a warning which may cause us to withdraw from a stimulus and so protect an injured body part. Unfortunately, pain often continues long after it has ceased to be a useful form of protection. Associated muscle spasm, atrophy, habitual postures, guarding and psychological factors all combine to make chronic pain a clinical state in itself.

Types of pain

Pain may be classified as somatogenic (acute or chronic), neurogenic or psychogenic. Chronic pain is traditionally said to last for more than six weeks, while acute pain is pain of sudden onset and lasts for less than six weeks. However, rather than distinct timescales, pain behavior is more appropriate as a classification.

Definition

Acute pain is traditionally said to have a sudden onset and lasts for less than six weeks, while chronic pain lasts for more than six weeks.

Musculoskeletal pain is not usually well localized – the surface site where the pain is felt rarely correlates directly to injured subcutaneous tissue. Generally, the closer an injured tissue is to the skin surface, the more accurate the patient can be at localizing it.

Deep pain is normally an aching, ill-defined sensation. It can radiate in a characteristic fashion, and may be associated with autonomic responses such as sweating, nausea, pallor and lowered blood pressure. Pain referral usually corresponds to segmental pathways, most often dermatomes. The extent of radiation largely depends on the intensity of the stimulus, with pain traditionally said to radiate distally, and rarely to cross the mid-line of the body (Cyriax 1982). In the clinic, however, these rules, while a useful guide, are often not adhered to rigidly.

Neurogenic pain is different again. Compression of a nerve root gives rise to ill-defined tingling, especially in the distal part of the dermatome supplied by the nerve. This is a pressure reaction, which quickly disappears when the nerve root is released. Greater pressure often causes the tingling to give way to numbness. Compression or tension to the dural sleeve covering the nerve root gives severe pain, generally over the whole dermatome. In contrast, pressure on a nerve trunk is conventionally said to cause little or no pain, but results in a shower of 'pins and needles' as the nerve compression is released. Pressure applied

to a superficial nerve distally gives numbness and some tingling, with the edge of the affected region being well defined.

Irritability

Irritability may be defined as 'the vigour of activity which causes pain' (Maitland 1991). It is determined by the degree of pain which the patient experiences, and the time this takes to subside, in relation to the intensity of activity or mechanical stimulation. The purpose of assessing irritability is to determine how much activity (joint mobilization, exercise, and so on) may be prescribed without exacerbating the patient's symptoms.

An assessment of irritability may be made at the second treatment session. The amount of movement which the patient was subjected to in the previous session is known, as is the discomfort that he or she feels now. These subjective feelings are then used to determine the intensity of the second treatment session. Similarly, at the beginning of each subsequent treatment session the irritability is again assessed.

Keypoint

Irritability is a measure of the amount of pain a patient experiences as a result of movement (including that of treatment). Irritability can be used to guide the type and intensity of treatment to avoid excessive post-treatment soreness.

Treatment note 1.2 Pain description in examination

During both the subjective examination and the objective examination (see Treatment Note 1.8, p. 53) the patient will usually describe pain as part of their experience. In addition to psychosocial factors (see below), both the type (nature) of pain and its behaviour are important factors in making an accurate clinical diagnosis, and a number of factors should be considered:

- When pain is decreasing, the condition is generally resolving; increasing pain suggests a worsening condition.
- Constant pain which does not change with time, alteration of static posture or activities may suggest a non-mechanical condition such as chemical irritation, tumours or visceral lesions.
- Where pain changes (episodic pain), the therapist should try to determine what activities make the pain worse (exacerbation) and what make it better (remission).
- The therapist should try to determine if the pain is associated with particular events (e.g. movements, visceral function), or time of day.
- Pain with activity which reduces with rest in general suggests a mechanical problem, irritating pain-sensitive structures.
- Morning pain which eases with movement may indicate chronic inflammation which takes time to build up and reduces with movement.

The description of pain itself may indicate the structure causing it (see Table 1.1) and the

Table 1.1 Pain descriptions and related structures

Type of pain	
Cramping, dull, aching, worse with resisted movement	Muscle
Dull, aching, worse with passive movement	Ligament, joint capsule
Sharp, shooting	Nerve root
Sharp, lightning-like, travelling	Nerve
Burning, pressure-like, stinging, with skin changes	Sympathetic nerve
Deep, nagging, poorly localized	Bone
Sharp, severe, unable to take weight	Fracture
Throbbing, diffuse	Vasculature

Source: Magee (2002) and Petty and Moore (2001) with permission.

Treatment note 1.2 *continued*

behaviour of the pain on physical examination clarifies the picture.

Recording pain

The intensity of pain may be recorded on a visual analogue scale (VAS). The patient is asked to indicate the pain description or number which best represents their pain. Where a 10 cm line is used the distance from the left of the scale to the point marked by the patient may be measured in millimetres and used as a numerical value (Fig. 1.5).

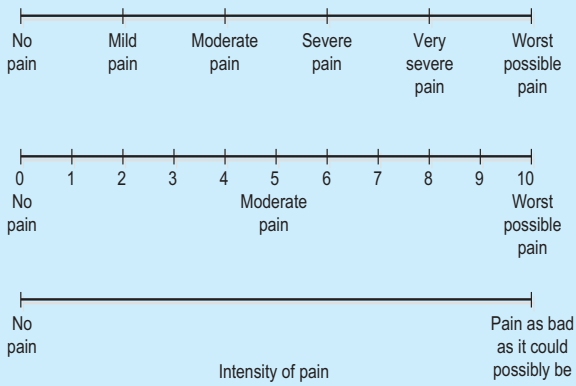

Figure 1.5 Visual analogue scales (VAS) used in pain description. From Petty and Moore (2001) with permission.

Red flags

It is important for the therapist to appreciate when pain and other symptoms may suggest serious pathology which requires medical investigation – so-called 'red flags' (Table 1.2). Where the patient has persistent pain and is generally unwell, the indication is that a pathology other than a musculoskeletal condition may exist. In addition, changes in bladder and bowel habits, alteration in vision or gross changes in gait all require further investigation.

Table 1.2 Red flags in sport examination indicating medical investigation

System/ possible pathology	Pain behaviour
Cancer	Persistent night pain
	Constant (24 hour) pain
	Unexplained weight loss (e.g. 4–6 kg in 10 days)
	Loss of appetite
	Unusual lumps or growths
	Sudden persistent fatigue
	Past history of carcinoma
Cardiovascular	Shortness of breath
	Dizziness
	Pain or feeling of heaviness in the chest
	Pulsating sensations in the body
	Discoloration in the feet
	Persistent swelling with no history of injury
Gastrointestinal/ genitourinary	Frequent or severe abdominal pain
	Frequent heartburn or indigestion
	Frequent nausea or vomiting
	Change in bladder or bowel habits
	Unusual menstruation
Neurological	Changes in hearing
	Frequent or severe headache
	Problems in swallowing or changes in speech
	Gait disturbance, or problems with balance/coordination
	Drop attacks (fainting)
	Sudden weakness

Source: Magee et al. (2002) and Waddell, G., Feder, G. and Lewis, M. (1997) Systematic reviews of bed rest and advice to stay active for acute low back pain. *British Journal of General Practice*, **47**, 647–652. With permission.