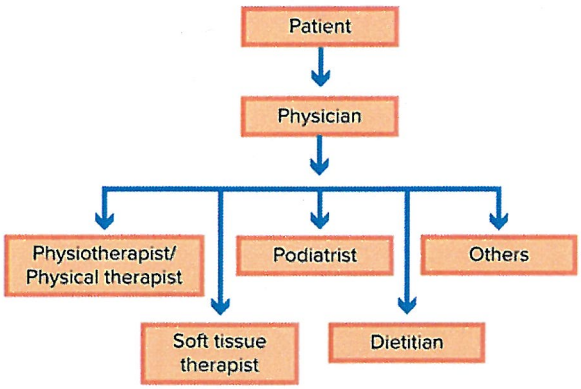


Figure 1.1 The traditional medical model

in its own specialist silo with little focus on holistic athlete health management, effective communication integration and understanding to facilitate decision making. The athlete and coach are often ill equipped to integrate the different contributions in these settings of increasing complexity and multispecialists. They often rely on expert case managers with a very good sport-specific understanding (in big teams the specialist SEM physician) to provide 'health leadership', effectively and efficiently communicating and integrating the contributions of all the key role players in the so-called *integrated performance health management and coaching model*.[9] This new model emphasises the importance of an integrated approach in communication and management to athlete health problems. The focus is on operational integration of the health management and coaching to improve performance with one accountable 'case manager'.

THE CHALLENGES OF MANAGEMENT

The secret of success in SEM is to take a broad view of the patient and his or her problem. The narrow view may provide short-term amelioration of symptoms but will ultimately lead to failure. Examples of a narrow view may include a runner who presents with shin pain, is diagnosed as having a stress fracture of the tibia and is treated with rest until pain-free.

Although it is likely that in the short term the athlete will improve and return to activity there remains a high likelihood of recurrence of the problem on resumption of activity. The clinician must always ask 'Why has this injury/illness occurred?' The cause may be obvious, for example, a recent sudden doubling of training load, or it may be subtle and, in many cases, multifactorial.

The greatest challenge of SEM is to identify and correct the cause of the injury/illness. The runner with shin pain arising from a stress fracture may have abnormal biomechanics, inappropriate footwear, had a change in the training surface, or a change in the quantity or quality of training. In medicine, there are two main challenges: diagnosis and treatment. In SEM it is necessary to diagnose both the problem and the cause. Treatment then needs to be focused on both these areas.

Diagnosis

Every attempt should be made to diagnose the precise anatomical, pathological and functional cause of the presenting problem. Knowledge of anatomy (especially surface anatomy) and an understanding of the pathological and functional processes likely to occur in athletes often permits a precise diagnosis. Thus, instead of using a purely descriptive term such as 'shin splints', the practitioner

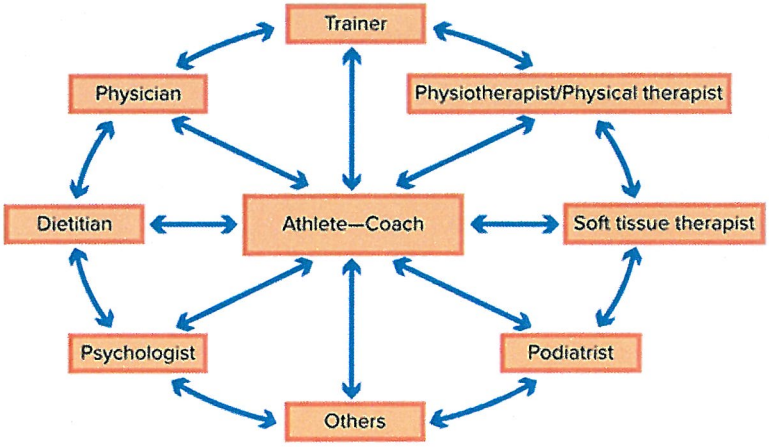


Figure 1.2 The SEM model. In professional sport the player's agent also features prominently in athlete–coach interaction

should attempt to diagnose which of the three underlying causes it could be–stress fracture, chronic compartment syndrome or periostitis–and use the specific term. Accurate diagnosis guides precise treatment.

Some clinical situations do not allow a precise anatomical and pathological diagnosis. For example, in many cases of low back pain, it is clinically impossible to differentiate between potential sites of pathology. In situations such as these it is necessary to establish a functional diagnosis, monitor symptoms and signs through careful clinical assessment and correct any abnormalities present (e.g. hypomobility) using appropriate treatment techniques.

As mentioned, diagnosis of the presenting problem should be followed by diagnosis of the cause of the problem. American orthopaedic surgeon Ben Kibler coined the term 'victim' for the presenting problem and 'culprit' for the cause.[10] Diagnosis of the cause often requires a good understanding of biomechanics, technique, training, nutrition and psychology. Just as there may be more than one pathological process contributing to the patient's symptoms, a combination of factors may cause the problem.

As with any branch of medicine, diagnosis depends on careful clinical assessment, which consists of obtaining a history, physical examination and investigations. The most important of these is undoubtedly the history but, unfortunately, this is often neglected. It is essential that the sports clinician be a good listener and develop skills that enable him or her to elicit the appropriate information from the athlete. Once the history has been taken, an examination can be performed. It is essential to develop examination routines for each joint or region and to include in the examination an assessment of any potential causes.

Investigations should be regarded as an adjunct to, rather than a substitute for, adequate history and examination.[11] The investigation must be appropriate to the athlete's problem, provide additional information and should only be performed if it will affect the diagnosis and/or treatment.

Treatment

Ideally, treatment has at least three components: discussing the planned treatment with the athlete, coach and key role players (also in the context of the immediate and future performance goals); treatment of the presenting injury/illness; and treatment to correct the cause. Generally, no single form of treatment will correct the majority of SEM problems. A combination of different forms of treatment will usually give the best results.

Therefore, it is important for the clinician to be aware of the variety of treatments and to appreciate when their use may be appropriate. It is also important to develop as many treatment skills as possible or, alternatively, ensure access to others with particular skills. It is essential to evaluate the effectiveness of treatment constantly. If a particular treatment is not proving to be effective, it is important firstly to reconsider the diagnosis. If the diagnosis appears to be correct, other treatments should be considered.

Meeting individual needs

Every patient is a unique individual with specific needs. Without an understanding of this, it is not possible to manage the athlete appropriately. The patient may be an Olympic athlete whose selection depends on a peak performance at forthcoming trials. The patient may be a non-competitive business executive whose jogging is an important means of coping with the stress of everyday life. The patient may be a club tennis player whose weekly competitive game is as important as a Wimbledon final is to a professional. Alternatively, the patient may be someone to whom sport is not at all important but whose low back pain causes discomfort at work.

The cost of treatment should also be considered. Does the athlete merely require a diagnosis and reassurance that he or she has no major injury? Or does the athlete want twice-daily treatment in order to be able to play in an important game? Treatment depends on the patient's situation, not purely on the diagnosis.

THE COACH, THE ATHLETE AND THE CLINICIAN

The relationship between the coach, the athlete and the clinician is shown in Figure 1.3. The clinician obviously needs to develop a good relationship with the athlete. A feeling of mutual trust and confidence would lead to the athlete feeling that he or she can confide in the clinician and the clinician feeling that the athlete will comply with advice.

As the coach is directly responsible for the athlete's training and performance, it is essential to involve the

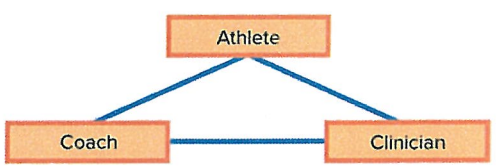


Figure 1.3 The relationship between the coach, the athlete and the clinician

A

Tendinosis: what is it?

This box illustrates the pathology found at end-stage tendinopathy—when symptoms have been present for at least 3 months. The illustration (Fig. 4.10) is based on pathological specimens (Figs 4.11 and 4.12) obtained at surgery for chronic sports-related tendon pain.

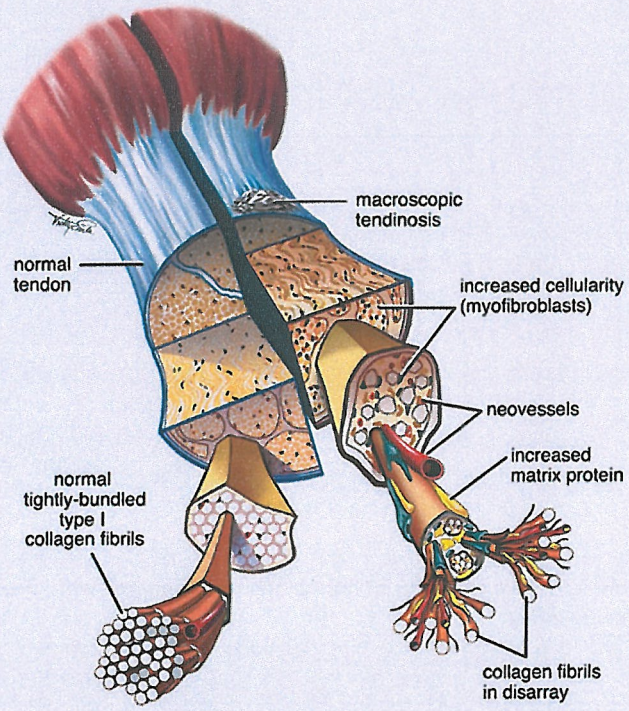
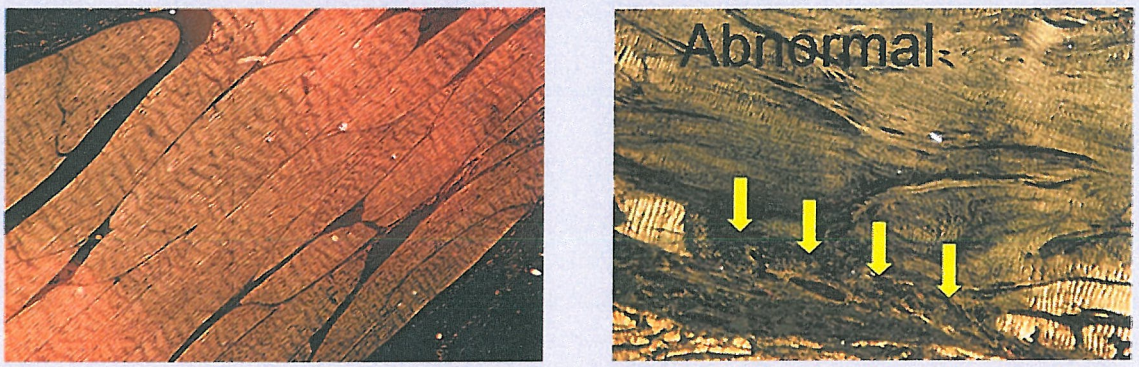


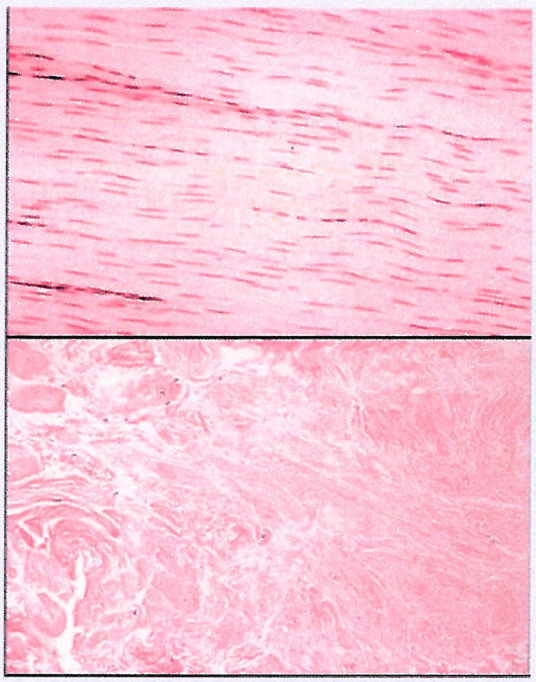
Figure 4.10 The contrasting features of normal tendon (left side) and tendinosis (right side). Characteristic features at this macroscopic level are the collagen fibres of different sizes in disarray, abnormal cell numbers (decreased and increased), abnormally prominent blood vessels and an increase in matrix proteins

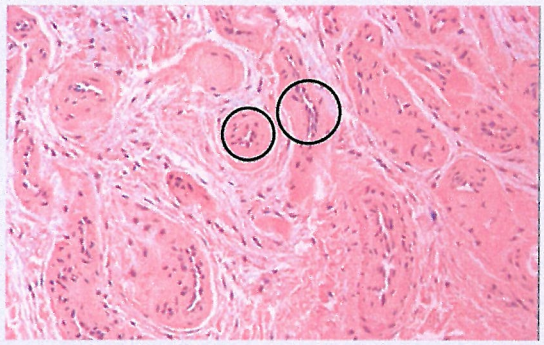


(a) (b)

Figure 4.11 Under polarised light microscopy: (a) normal tendon has tightly bundled parallel collagen fibrils with a characteristic golden reflectivity; (b) a specimen from a patient with chronic patellar tendinopathy showing collagen fibril separation and frank discontinuity within some fibrils

continued





(c)

Figure 4.12 Under light microscopy (haematoxylin and eosin [H&E] stain, hence pink)
(a) normal tendon
(b) collagen fibre disarray comparable with Figure 4.11 (b) but under greater magnification and with this different colour stain; note the loss of parallel bundles of collagen and absent cell nuclei
(c) other areas have increased prominence of cell nuclei and excessive abnormal vascularity

Table 4.9 Five elements of normal tendon compared with the characteristic elements of end-stage tendon overuse injury

Tendon element	Normal tendon	Changes that occur in response to excessive tendon loading
Cells—tenocytes	Tendon cells are spindle-shaped and nuclei cluster in longitudinal chains on microscopy	Tissue has proliferation of cells with abnormally rounded nuclei (Fig. 4.12c) and areas with fewer than normal cell numbers (Fig. 4.12b)
Ground substance or 'matrix' proteins	The ground substance in the matrix is minimal and is not visible when stained for light microscopic viewing	Increased amount of ground substance/matrix proteins which stain and are visible under light microscopy
Collagen	Linear and tightly bundled and has a characteristic crimp under polarised light	Disrupted—both longitudinally and in its bundles (Fig. 4.12b)
Nerves	Minimal intratendinous nerves, some innervation of connective tissue in and around the tendon	Abnormal ingrowth of nerves (mostly sympathetic) and a preponderance of neuropeptides
Vessels	Minimal vascularity when examined histologically or by using ultrasound	Prominent vessels histologically or using ultrasound (Fig. 4.12c)

are producing repair proteins, especially proteoglycans. This results in a short-term thickening of a portion of the tendon that reduces stress. This differs from the normal tendon adaptation to tensile load that generally occurs through tendon stiffening with little change in thickness. This is seen clinically in the acutely overloaded tendon and is more common in the younger person. It also arises when there is trauma to a tendon, such as a direct blow.

At this stage, both ultrasound and MRI show mild fusiform swelling–greater tendon diameter with little disruption of the collagen fascicles. The change in imaging appearance is mainly derived from the increase in bound water within the matrix proteins (proteoglycans).

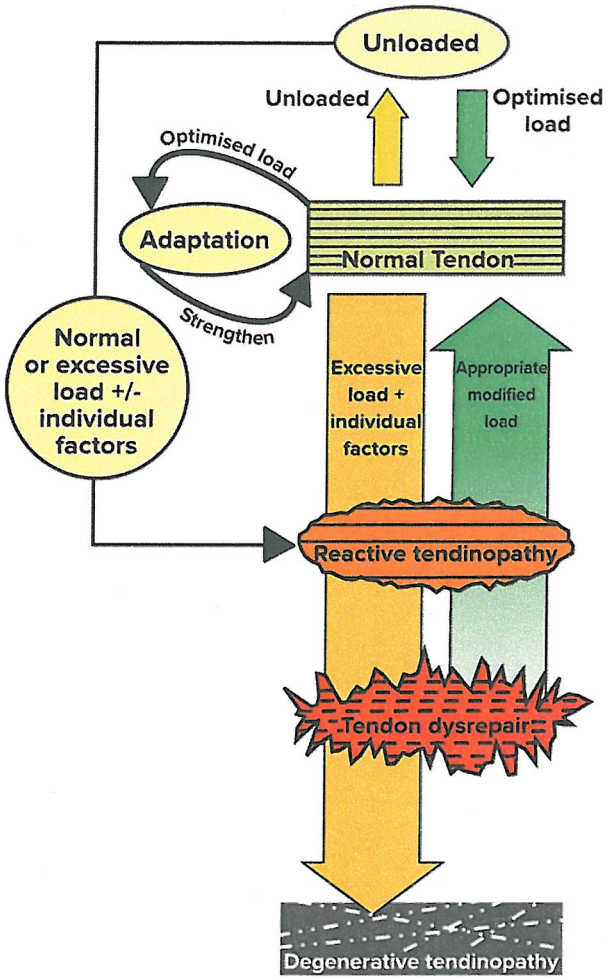


Figure 4.13 The Cook–Purdam model to help clinicians understand the relationship between loading/unloading and the several stages of tendon pathology
REDRAWN FOR *CLINICAL SPORTS MEDICINE*. REPRODUCED WITH PERMISSION FROM *BRITISH JOURNAL OF SPORTS MEDICINE*[127]

Stage 2. Tendon dysrepair

This describes a worsening tendon pathology with greater matrix breakdown. Tendon cells are more prominent and take on a rounded appearance (chondrocytic). Protein production increases–both matrix proteoglycans and collagen. As a result of these changes, collagen separates and the matrix becomes somewhat disorganised. The disruption of the matrix may allow for some ingrowth of vessels and nerves.

This is seen clinically in overloaded tendons in the young, but it may appear across a spectrum of ages and loading environments. The transition from the previous stage may be difficult to detect clinically, but imaging will reveal more focal changes of hypoechogenicity on ultrasound. There may be a mild increase in vascularity on colour or Doppler ultrasound. MRI reveals a swollen tendon with increased signal.

Stage 3. Degenerative tendinopathy

This is the stage that is present in patients who undergo surgery for chronic tendon pain–it is the 'end stage' of tendon overuse injury. The matrix and cell changes described in stages 1 and 2 may progress so that areas of apoptosis (absent cell nuclei due to cell death) are evident. Large areas of matrix are disordered and filled with cells, vessels, matrix breakdown products and disordered type I, II and III collagen. The structure of the tendon is heterogeneous–degenerative pathology is interspersed between other stages of pathology and normal tendon.

This pathology exists in the older person, or the younger person or elite athlete with long-term tendon overload. Typically, it is seen in a middle-aged, recreational athlete with focal tendon swelling and pain (e.g. mid-Achilles region). The tendon may have focal nodular areas with or without general thickening. This is the type of tendon that may rupture if the degenerative pathology is extensive.

In this stage, compromised matrix and vascular changes are obvious on ultrasound scans as hypoechogenic regions with few reflections from collagen fascicles. Larger vessels are usually prominent on Doppler ultrasound. MRI shows increased tendon size and intratendinous signal. The changes are more focal than spread throughout the tendon.

Reactive on degenerative tendon

Degenerative tendinopathy exists in a tendon that has normal tissue. When these tendons are overloaded, the degenerative part of the tendon is unlikely to take load because of a lack of matrix structure, so the load is borne by the normal part of the tendon. Overload can induce a reactive change in this normal tendon, resulting in a reactive on degenerative tendon.

Other terms associated with overuse tendon injuries

Although the most-used clinical label for tendon overuse injuries is 'tendinopathy' as above and is used in specific chapters of this book (i.e. Achilles tendinopathy), the terms 'paratenonitis', 'partial tear' and 'tendinitis' need definition.

Paratenonitis

This term includes peritendinitis, tenosynovitis (single layer of areolar tissue covering the tendon) and tenovaginitis (double-layered tendon sheath). This

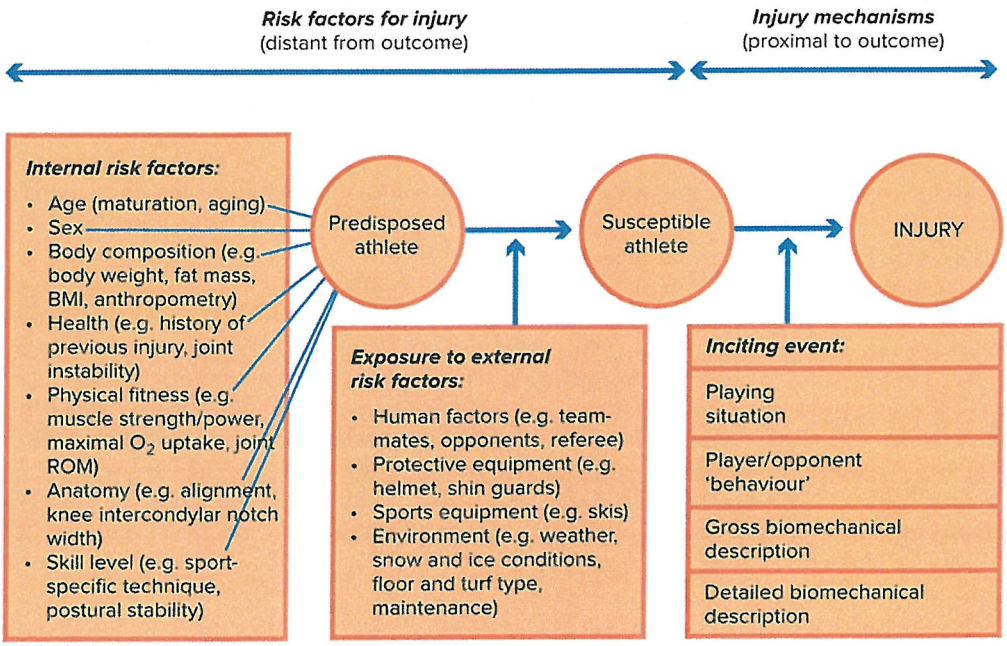


Figure 12.2 A comprehensive injury causation model based on the epidemiological model of Meeuwisse et al.[4] and modified by Bahr & Krosshaug.[7] BMI = body mass index; ROM = range of motion

- developing an injury surveillance program within the team–recording injury and participation data
- season analysis–risk profiling the training and competition program
- performing a periodic medical assessment–mapping current problems and intrinsic risk factors
- developing and initiating a targeted prevention program.

Reviewing the literature—risk identification and assessment

Each sport has its typical injury pattern. Just think of the names of some sports injuries: tennis elbow, runner's knee, jumper's knee. For most sports, there is ample data in the literature to identify and assess the risks. Note that injury risk is not just a question of injury frequency–the severity of injury must also be taken into account.

Injury data can be illustrated by a risk matrix that highlights risks in terms of likelihood and consequences. A risk matrix is a powerful tool for risk assessment. We derive the example shown in Figure 12.3 from soccer. It suggests that injury reductions in the areas of ACL, hamstring and ankle injuries are priorities. By further examining which factors contribute to the causation of these, we can formulate strategies to reduce injury rates. Therefore, in soccer it makes sense to target training programs to prevent ACL tears towards female athletes,

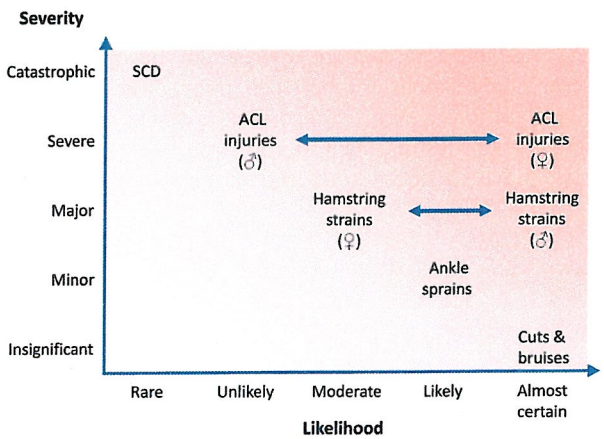


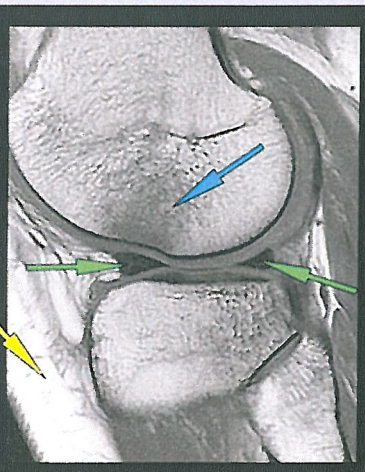
Figure 12.3 Qualitative risk matrix in elite soccer, illustrating the relationship between injury severity (consequence) and injury incidence (likelihood). The darker the colour, the greater the cross-product of severity and incidence, and the greater the priority should be given to prevention. The matrix also illustrates that risk differs between males and females. (SCD = sudden cardiac death)

while the lower risk among males may not justify such interventions. In contrast, a prevention program for male players should definitely focus on preventing hamstring strains.

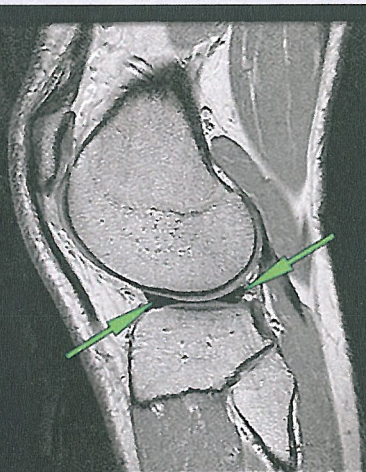
A

What does this MRI tell me? Tips for clinicians

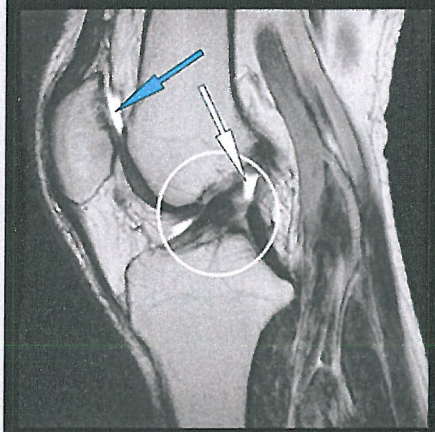
The four most common sequences a clinician will see are outlined below and shown in Figure 15.10.



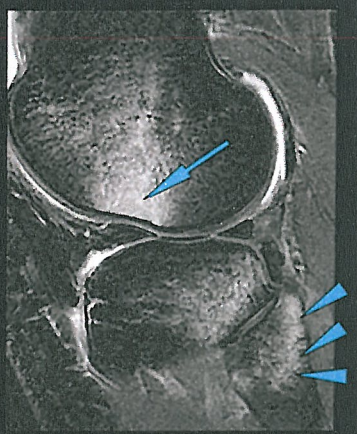
15.10a T1-weighted image showing bone marrow oedema (also called bone marrow lesion (BML) (Chapter 35) after full-thickness ACL tear. Arrows to high signal fat, (yellow arrow), very low signal normal lateral menisci (green arrows), low signal oedema (blue arrow).



15.10b Proton density-weighted image showing normal anterior and posterior horns of the lateral menisci (green arrows).



15.10c T2-weighted image showing full thickness ACL tear (white arrow). Note bright joint fluid (blue arrow).



15.10d STIR image (fat suppressed) showing the same patient as in part (a). Bone marrow oedema (high signal, blue arrow) as a result of the acute knee giving way episode and ACL rupture. Note additional bone marrow oedema (high signal, blue arrowheads) in the fibular styloid process as a result of a concommitant torn arcuate ligament.

Figure 15.10 The legend for the arrows/arrowheads to structures in the four MRI sequences is as follows: fat = yellow; tendon and ligament = off-white; water = blue; menisci = green

1. **T1-weighted** provides sharp anatomical detail, shows bone marrow and is good for meniscal pathology. It lacks the sensitivity to detect soft tissue injury (Fig. 15.10a). MRI signal key: fat = bright; muscle = intermediate; water, tendons and fibrocartilage all dark.
2. **Proton density-weighted** is good for imaging menisci and ligaments (Fig. 15.10b). MRI signal key: fat = bright or intermediate signal, calcium, tendons and fibrocartilage, all dark; water = intermediate.
3. **T2-weighted** is highly sensitive for most soft tissue injury, especially tendons. Abnormal tendons have high signal intensity (bright) which contrasts with normal tendons black (arrowhead) (Fig. 15.10c). MRI signal key: water = bright; fat = intermediate muscle, hyaline and fibrocartilage all dark.
4. **STIR** highlights excess water (blue arrow and blue arrowheads) which can occur due to bone stress and bone marrow oedema (shown), joint fluid and soft tissue pathology. This is the sequence of choice for bone stress injuries or subtle, radiographically occult fractures (Fig. 15.10d). MRI signal key: water = very bright; fat, muscle, menisci all dark.

Figure 21.2 Factors to consider in a headache evaluation

to either underestimate or overestimate their symptoms depending on how they feel at the time they are asked.[17] Hence evaluating a range of headache parameters is likely to give a clearer, bias-free estimate of change. Figure 21.2 illustrates typical measurements taken during evaluation.

Examination

In all patients presenting with headache, a full neurological examination is required, and the skull, cervical spine and orofacial region must always be carefully examined. The examination should consist of some or all of the following components depending on the presence or absence of specific symptoms in the history:

- general appearance
- mental state
- speech
- skull examination
- cervical spine examination
- temporomandibular joint
- gait and stance
- pupils and fundi
- special senses (e.g. smell, vision, hearing)
- other cranial nerves
- motor system
- sensory system
- general examination.

PRIMARY HEADACHE

Primary headache includes migraine, tension-type headache, medication overuse headache, exercise-related headache and some types of post-traumatic headache. In the general population, the 1-year prevalence rate for primary headache is 62%.[20] By survey, approximately 40% of migraine sufferers experience migraine precipitated by exercise, and 34% of Australian rules football players suffered migraine according to IHS criteria, a rate higher than population controls.[7] In that study, almost 60% of subjects experienced regular headaches related to sport.[4]

Migraine

Previously primary headache, in particular migraine, was thought to be a vascular disorder, or due to cortical spreading depression. More recent evidence points to abnormality of brain function leading to a chain of events in the periphery.[21-23] In simple terms, migraine pain can be thought of as an altered perception of normality, such that normal sensory input is misinterpreted as pain.[24] This concept of the variable modulation of sensory input is explained in Lorimer Moseley's YouTube video 'Why things hurt' (http://ow.ly/S9n0h). (See also Chapters 5 and 6.)

The neuroanatomical basis for migraine is the trigeminocervical nucleus. The migraine pain process is likely to be a combination of direct factors, that is, activation of the trigeminal nociceptors, in concert with a reduction in the normal functioning of the centrally mediated endogenous pain control pathways that normally gate that pain.[25] Thus there are both central and peripheral mechanisms involved.

Migraine is broadly categorised into migraine with and without aura. An aura is a specific set of neurological symptoms that typically precede the headache which include visual disturbances (e.g. scotomas), paraesthesia, vertigo, hemiplegia and ophthalmoplegia. Although most people think of migraine as headache alone, the true migraine sufferer usually notices a spectrum of symptoms, including nausea, vomiting, diarrhoea and weight gain. The important point for sports clinicians is that there does not have to be an aura. The IHS criteria for the diagnosis of migraine without aura are shown in Table 21.2.[3]

Clinical features

The typical features of migraine are precipitation by a change in homeostasis such as tiredness, temperature, altitude, thirst, hunger or stress. Ensuing headache pain is described as sharp and intense, throbbing or beating in time with their pulse. Commonly, it begins in the temple or forehead on both sides. When it starts on one side, it may spread to the other side. Occasionally, the headache begins at the back of the head and moves forwards.

The common neurological accompaniments to migraine with aura are visual. Patients speak of bright or dark objects often to one side of the visual field. These objects may shine or flicker and typically move across the visual field. The visual symptoms usually last about 20 minutes

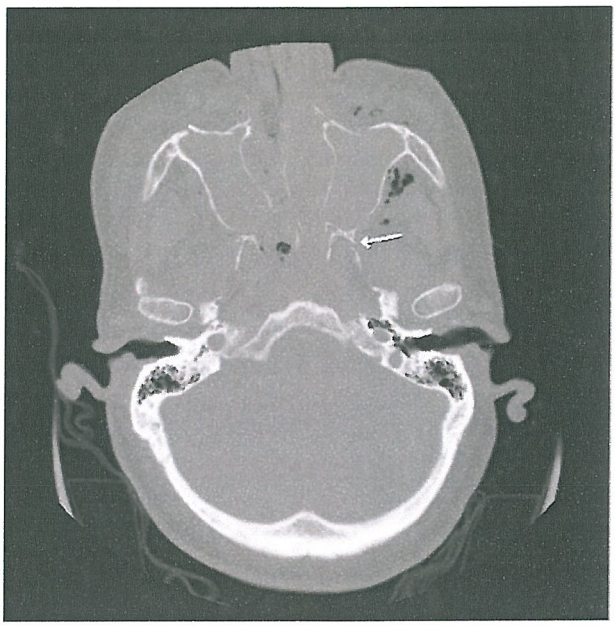


Figure 22.16 CT scan of Le Fort III fracture (arrow)

fractures usually occur on opposite sides of the midline. Fractures may be undisplaced or displaced.

Undisplaced fractures

Minor mandibular fractures are painful, tender and swollen. These are managed conservatively with analgesia and rest. The patient should eat soft food only for up to 4 weeks as symptoms resolve.

Displaced mandibular fractures

Displaced mandibular fractures are severe injuries that result from considerable force. Alveolar (tooth-bearing) fractures are the most common type. These fractures range from single tooth fractures or avulsions to complete segment mobility. The clinical diagnosis is obvious when two or more teeth move as a unit.

Inspection may reveal malalignment of teeth and bruising to the floor of the mouth. Palpation reveals malocclusion, tenderness and defects along the lower border of the mandible. Paraesthesia or anaesthesia of the lower lip and chin suggest damage to the inferior alveolar nerve.

Initial treatment includes maintenance of the airway in a forward sitting position with the patient's hands supporting the lower jaw. A jaw bandage can be used in comminuted or badly displaced fractures, although this is generally not used due to the increased associated risk of compromising the airway by causing backward displacement of the mandible. A cervical collar can be used as an alternative. A concussed or unconscious patient should be placed in a lateral position with head tilt and jaw support after the mouth has been cleared of any dislodged teeth or tooth fragments. Occasionally, the tongue may need to be held forward to maintain an open airway.

Most displaced mandibular fractures require closed reduction and intermaxillary fixation for 4-6 weeks. If adequate closed reduction cannot be achieved, then open reduction and internal fixation is required. A fracture of one condyle usually does not require immobilisation except to control pain. Active jaw exercises should be commenced as soon as pain permits.

During the period of intermaxillary fixation, the athlete may perform mild exercises such as stationary bike riding and light weightlifting. Resumption of contact sport should be delayed until at least 1-2 months after the jaws are unwired. Earlier resumption is possible when internal fixation has been used. The use of a protective polycarbonate facial shield may offer some protection if early return to play is contemplated.

Patients with mandibular fractures who are eating soft food or have their jaws wired can be referred to a dietitian for advice on suitable liquid meals and foods suitable for vitamising.

Temporomandibular injuries

Blows to the mandible can produce a variety of temporomandibular joint (TMJ) injuries. Trauma to the jaw while the mouth is open occasionally produces TMJ dislocation. Other injuries include haemarthrosis, meniscal displacement and intracapsular fracture of the head of the condyle.

Examination of the injured TMJ may reveal limitation of opening, pain and malocclusion. Dislocation of the TMJ causes inability to close the mouth. A dislocated TMJ may be reduced by placing both thumbs along the line of the lower teeth as far posteriorly as possible and applying downward and backward pressure. Longstanding dislocations may require general anaesthesia for reduction. Management of TMJ dislocation includes rest with limitation of mouth opening for up to 7-10 days, a soft diet and analgesics such as aspirin. Contact sport should be avoided for up to 2 weeks depending on the symptoms. Boxers should not attempt sparring for at least 6 weeks.

Chronic temporomandibular pain

Chronic TMJ problems are sometimes referred to as 'temporomandibular joint dysfunction' or 'myofascial pain dysfunction syndrome'. This syndrome appears to affect males more than females with a peak incidence in the early twenties. Patients complain of pain, limitation of movement, clicking and locking of the TMJ. Treatment should include assessment by a dentist to exclude any

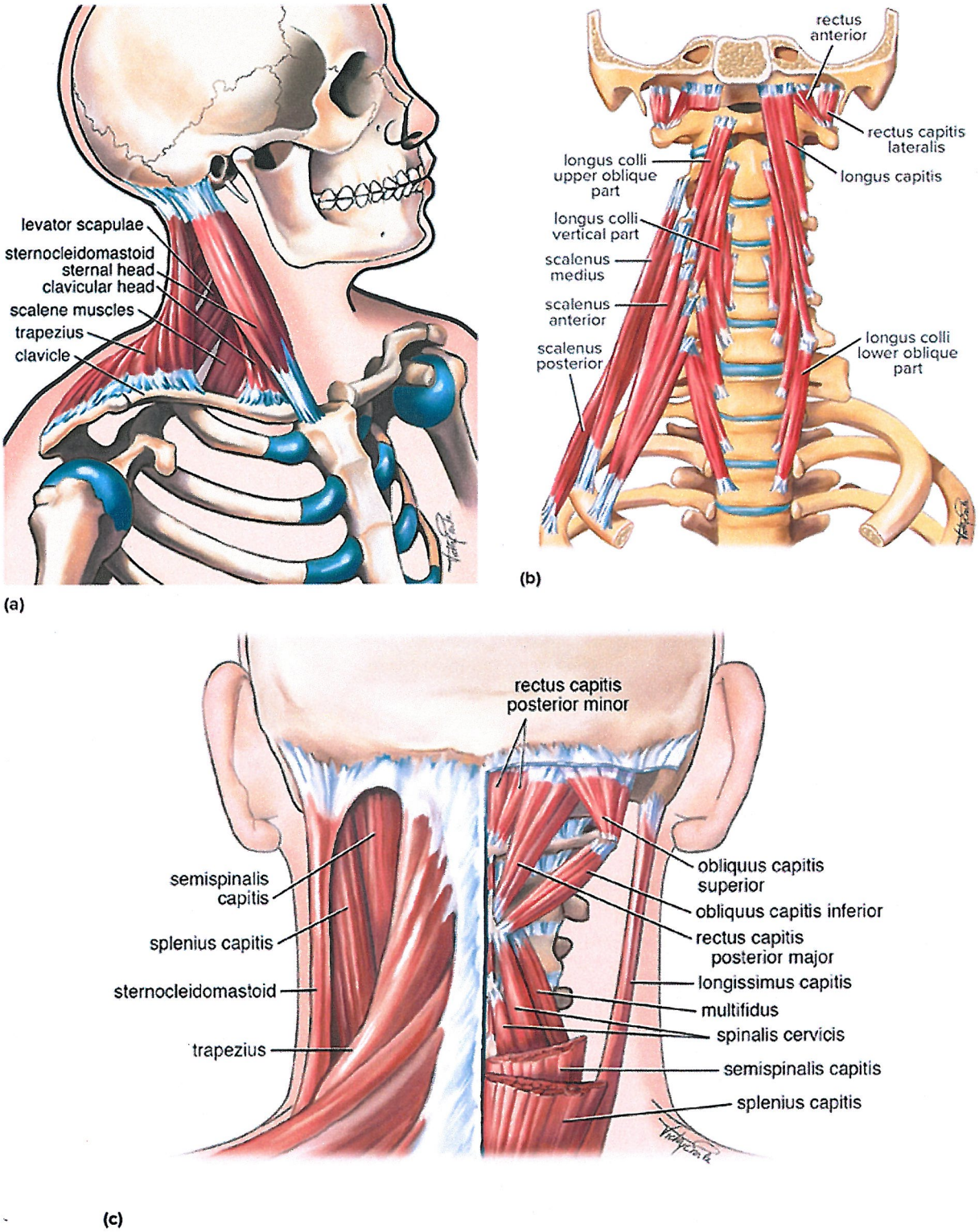


Figure 23.1 Anatomy of the neck (a) Anatomy of the anterior neck—superficial musculature (b) Anatomy of the anterior neck—deep musculature (c) Anatomy of the posterior neck—deep and superficial musculature