ROUTLEDGE INTERNATIONAL HANDBOOKS

Routledge Handbook of Sports Therapy, Injury Assessment and Rehabilitation

Edited by Keith Ward

ROUTLEDGE HANDBOOK OF SPORTS THERAPY, INJURY ASSESSMENT AND REHABILITATION

The work of a sports therapist is highly technical and requires a confident, responsible and professional approach. The *Routledge Handbook of Sports Therapy, Injury Assessment and Rehabilitation* is a comprehensive and authoritative reference for those studying or working in this field and is the first book to comprehensively cover all of the following areas:

- Sports injury aetiology
- Soft tissue injury healing
- Clinical assessment in sports therapy
- Clinical interventions in sports therapy
- Spinal and peripheral anatomy, injury assessment and management
- Pitch-side trauma care
- Professionalism and ethics in sports therapy.

The Handbook presents principles which form the foundation of the profession and incorporates a set of spinal and peripheral regional chapters which detail functional anatomy, the injuries common to those regions, and evidence-based assessment and management approaches. Its design incorporates numerous photographs, figures, tables, practitioner tips and detailed sample Patient Record Forms. This book is comprehensively referenced and multi-authored, and is essential to anyone involved in sports therapy, from their first year as an undergraduate, to those currently in professional practice.

Keith Ward has practised as a Sports Therapist since 1995. He has taught at a number of colleges and universities, and since 2009 has been a full-time lecturer on the BSc Sports Therapy programme at University College Birmingham (UCB), UK. He runs a small private practice in Cannock, Staffordshire. His first book, *Hands-On Sports Therapy*, proved popular with students of sports therapy and became a recommended text for many courses. Since 2012, he has been Managing Editor of the *Journal of Sports Therapy* (the 'JST'). He is a member of the Society of Sports Therapists (SST), a member of the Register of Exercise Professionals (REPS), a Fellow of the Royal Society of Public Health (RSPH), and a Fellow of the Higher Education Academy (HEA). Keith holds a biomedical BSc Rehabilitation Studies degree and Diplomas in Sports Therapy, Sports Massage, Exercise Referral, Naturopathy, and Acupuncture. He is currently completing an MA in Learning and Teaching.

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I dedicate this book with love to my wife Angela, our children Dennie and Arielle, and of course to my mom and dad (Peter and Nina). You have all given me so much love and support.

KW

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CONTRIBUTORS

Jo Baker BSc PG Cert MSc MSST, Lecturer in Sports Therapy, London Metropolitan University, UK.

Amy Bell BSc MSc MCSP BASRAT, Physiotherapist and Graduate Sports Rehabilitator, UK.

Hannah Boardman BSc PG Cert MSc MSST, Lecturer in Sports Therapy, University College Birmingham, UK.

Anthony Bosson BSc PG Cert MSST, Lecturer in Sports Therapy, Technological and Higher Education Institute of Hong Kong.

Aaron J. Caseley BOst MSc BOsC, Senior Lecturer in Sports Therapy, Faculty of Health and Social Sciences, Leeds Metropolitan University, UK.

Rob Di Leva BSc MSc, Sports Therapist, Australia.

Troy Douglin BSc PG Cert MSc MCSP, Lecturer in Sports Therapy, University College Birmingham, UK.

Kate Evans BSc PG Cert MSc MSST, Programme Director and Senior Lecturer in Sports Therapy, University of Wales Trinity David, UK.

Andrew Frampton BSc PG Cert MSST, Lecturer in Sports Therapy, University College Birmingham, UK

Nick Gardiner BSc PG Cert MSST, Lecturer in Sports Therapy, London Metropolitan University, UK.

Mike Grice BA BOst PG Cert BOsC, Lecturer in Sports Therapy, University College Birmingham, UK.

Adam Hawkey BSc MSc FBIS BASES REPS, Lecturer in Biomechanics, Abertay University (Dundee), UK.

Ian Horsley BSc MSc Phd MCSP MMACP MACPSM, English Institute of Sport (Regional Lead Physiotherapist, NW), UK.

Mark I. Johnson, Professor of Pain and Analgesia, Leeds Metropolitan University, Leeds Pallium Research Group, UK.

Jeanette Lewis BSc PG Cert MSST, Lecturer in Sports Therapy, University College Birmingham, UK.

Greg Littler BSc PG Cert MSc MCSP MSST, Senior Lecturer in Physiotherapy, University of Central Lancashire, UK.

Andrew Mitchell BSc PG Cert MSc Phd NSCA, Senior Lecturer, Sport and Exercise Science and Sports Therapy, University of Hertfordshire, UK.

Paul Robertson BSc PG Cert MSc BASES UKSCA, Senior Lecturer in Sports Therapy, University College Birmingham, UK.

Graham N. Smith, Chartered and HCPC Physiotherapist, Rehabilitation and Sports Injury Consultant, Glasgow, UK.

Philip Smith Grad Dip MSc MCSP, Senior Lecturer in Sports Therapy, Coventry University, UK.

Peter K. Thain BSc PhD MSST, Senior Lecturer in Sports Therapy, University of Bedfordshire, UK.

Tim Trevail BSc PG Dip SMA IRMA, Head of Academic Studies (Physical Health), Think Education Group, Australia.

Lee Young BSc PG Cert BASRAT, Lecturer in Sports Therapy, University College Birmingham, UK.

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FOREWORD

Prior to 1990 there was no such thing as a sports therapist. This does not mean that anyone who participated in sport did not sustain injuries or receive primarily basic treatment from trainers, first aiders, physios and doctors. It does mean that, until that point, there was not a specific profession in the UK with its own clearly defined professional title, training, education and scope of practice, dedicated to sports and exercise therapy. However, a well-established and similar model did exist in the United States, which had been in existence since 1950; the model was that of the athletic trainer. This was also a practitioner who was playing an extremely vital role in sports medicine teams at all levels of the American sporting spectrum. Consequently, there was an urgent need for something comparable in the UK. Hence the birth of sports therapy and sports therapists.

Since 1990, sports therapist has become a firmly established occupational title. Similarly, sports therapy is now a respected profession that is firmly established within the sport and exercise medicine family in the UK. A significant reason for this was the development of graduate programmes which, through their validation and accreditation processes, set educational and professional benchmarks that are identifiable and applicable. Significantly, it is this graduate benchmark that is becoming increasingly used by governing bodies in sport and regulatory organizations as the minimum standard required for recognition and professional approval.

It is now acknowledged that sports therapists working in a multidisciplinary sport and exercise medicine team complement the other professionals and practitioners involved, which can only benefit the sports participant and potential patient/casualty. Sports therapists also have the ability to work as autonomous practitioners in an environment and industry that is extremely demanding and frequently unforgiving; hence the need for sports therapists who are appropriately educated and prepared.

A sports therapist requires knowledge, skills, expertise and competencies in five key areas. These are the prevention of injury; the recognition and evaluation of injury; management, treatment and, if appropriate, referral; rehabilitation; and education and professional practice issues. They are 'pillars of knowledge' that need to be built upon strong academic foundations.

In my opinion this *Handbook of Sports Therapy* has managed to establish these foundations. In doing so the editor and contributors have produced a publication that, I believe, will become a valuable asset to anyone studying sports therapy. The content has been carefully compiled to cover the concepts required in each of the five key competency areas by a cadre of contributors

who, predominantly, work in higher education and within the subject area. Consequently, they understand the needs of the students they are responsible for educating and training. More importantly, sport and exercise therapy are linked between each contributor, which can only enhance the credibility of the material produced.

Another significant feature is that the editor has ensured that there is a consistency between each chapter, in both format and referencing, as well in the style of writing. This is extremely difficult to do but so important, especially when there are as many individuals contributing not only to the publication in its entirety, but to each specific chapter. For that achievement alone, the editor must be applauded and credited. I know that students of sports therapy will find this an extremely valuable handbook and one that they will refer to many times during their training and when they qualify. It is also a book that sets an academic benchmark for other specific sports therapy publications to follow. Therefore, I welcome this *Routledge Handbook of Sports Therapy, Injury Assessment and Rehabilitation*, and I will watch, with interest, to see how it evolves in the future alongside the exciting profession that it supports.

Professor Graham N. Smith
Chartered and HCPC Physiotherapist
Rehabilitation and Sports Injury Consultant
Glasgow (UK)

PREFACE

Even in a new age of information and technology, academic textbooks sit proudly and essentially as part of the base of the traditional hierarchical pyramid of research and information sources. Reputable academic textbooks (and handbooks) take a significant amount of time to develop and produce, and aim to provide evidence-based presentations of theoretical concepts and principles on often broad topic sets. I hope that the information in this book, which has been carefully gathered, appraised and presented, may almost inadvertently demonstrate a subtle tribute to all the therapists and scientists, academics and medics, researchers and writers, bloggers and tweeters, athletes and aspirant students who each effectively combine and contribute to an ever-evolving knowledge base.

This book is primarily aimed at *students* of sports therapy. While we have attempted to pitch some advanced concepts and develop a critically evaluative awareness, the main emphasis is on providing foundation and developmental information. The year 2015 is a fantastic time for sports therapists, given the growth and interest in all forms of sport and the clearly established public health need for widespread exercise participation. Not surprisingly, there has been a steady increase in the number of people choosing to study and practise in this exciting area. Since back in the day, athletes have been getting fitter and performing better because of the support they get from their team. The team today (at the higher levels) will be multidisciplinary, professionally organized, evidence-informed, progressive and technically advanced. Even in private practice, sports therapists are steadily gaining the increase in recognition that they deserve for helping their athletes and patients prepare for, and recover from, their chosen sports and physical activities. With increasing recognition and acceptance comes increasing demand and expectation.

The educational demands of graduate programmes in sports therapy obviously require appropriate, reliable and accessible reference resources; moreover, the standards of practice (the so-called 'competencies' of a sports therapist), which continuously undergo re-evaluation, must be achieved and evidenced by all who wish to achieve qualification. To demonstrate true competency in sports therapy is to be able to safely, effectively and autonomously demonstrate the ability to assess, manage and advise patients and athletes; this includes their fitness, their injuries, their needs and goals. Obviously, from the outset this is something much easier said than done, and clearly it takes time, effort and dedication to achieve such competency in such a specific area of health care.

This handbook is in no way any substitute for undertaking a comprehensive and recognized qualification, but along the way we hope that readers can better appreciate the remit of a sports therapist in any given situation, what constitutes our scope of practice, and importantly understand where sports therapists fit in alongside other health professionals.

To maintain focus, we have not attempted to discuss many of the more distinct specialities, methodologies, techniques and applications which are clearly essential component aspects in the wider delivery of sports therapy. Hence there is little mention, for example, of sports nutrition, sports psychology, advanced biomechanics, motor control or fitness assessment; nor are there expansive presentations of rehabilitation applications (whether regional, global or sports-specific) – other resources will provide all of this far more effectively. Simply, we have presented principles, overviews and discussions.

Most of us will recognize that the available 'evidence' (to support or refute anything) does not always equate to acceptable or transferrable validity or reliability – and this boils down to the very quality and type of research that is available, as well as how well it is accessed, translated and integrated. There will always be more questions, more proposals, hypotheses and anecdotes, and more debate. Sports therapists in this era are trained and expected to be relatively autonomous, and to be evidence-based. The concept of 'evidence-informed practice' is trichotomous and complementary and welcomes to the decision table, alongside the evidence-based and expert consensus guidelines, the contribution of individual practitioners' experience, expertise and clinical reasoning, as well as the utility of the 'patient culture' (i.e. the individual patient's or athlete's beliefs, values, situation and goals) which present in every single case.

This handbook has been an honest effort. We make no claim to perfection; and we accept in a critical world that almost any content can be legitimately challenged. We respect the progressive nature of contemporaneous information and enquiry. Whether student, experienced practitioner or lecturer, I think most of us should be able to appreciate that *the more we learn – the more we learn there is to learn*. I've certainly learnt a lot working on this handbook and I hope that you too can learn a little more, wherever you are on your own sports therapy journey.

Best regards to all.

Keith Ward
Cannock, Staffordshire, 2015

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SPORTS INJURIES
Basic classifications, aetiology and pathophysiology

Keith Ward and Andrew Mitchell

This chapter aims to support a foundation understanding of sports-related injuries. It is expected that all professionals involved in the care of active exercising populations will view the topics of injury classification, injury aetiology and injury pathophysiology as fundamental to their knowledge base, and it is hoped that this chapter's overview can clarify some of the essential components of these core topics and explain the mechanisms occurring as the body attempts to repair tissue following injury.

Classifications of sports injuries

Sports injuries occur for innumerable reasons. They can be due to poor preparation for activities undertaken, to over-enthusiastic training, to inherent biomechanical problems or simply due to pure accident, such as a fall from a bike or horse, an unavoidable collision, slip or mistimed tackle. Table 1.1 provides some consideration for the various ways in which sports-related injuries may be presented or categorized, with important terms and concepts identified for the sports therapist to gain familiarity with. Any injury must go through a period of healing, and the resulting outcome will depend on a number of factors, such as: the type and severity of injury; the particular tissues involved in the injury and their capacity for resolution (repair or replacement); the early management provided; the age and general health of the individual; factors impeding the healing and rehabilitation process; and, crucially, how the injury is managed during the days, weeks and months that follow. One important aspect of broader injury assessment and management, especially in professional and team settings, is that of injury surveillance. The 'Orchard Sports Injury Classification System' (OSICS) is one established tool for providing audits and coding of injury diagnosis in sports injury surveillance systems. OSICS incorporates detailed lists of regional injuries and provides coding specific to sports medicine (Orchard, 2010; Rae and Orchard, 2007). The authors suggest that with efficient injury surveillance and documented identification of prevalence and incidence of specific injuries in specific sports, injury prevention strategies can be improved.

All sports have a collection of common injuries associated with them – whether the sport is recreational or professional, individual or team, contact or non-contact, agility, endurance or power-based. Injuries may also be regionally categorized; and by being knowledgeable of the causes and presentations of common injuries associated with each body region, the sports

Table 1.1 Classifying sports injuries

Stage of healing	Immediate / acute / reactive / sub-acute / post-acute / proliferative (repair) / dysrepair / remodelling (chronic) / degenerative / pathological
Severity	Minor, moderate and severe / I–III degree / type 1–4 (muscle disorders and injuries) / insignificant / ongoing / catastrophic / complicated
Tissue type	Skin / fascia / muscle / tendon / bursa / capsule / ligament / fibro-cartilage / hyaline cartilage / bone / blood vessel / nerve / viscera
Aetiology	Mechanism / primary / secondary / macrotrauma / repetitive microtrauma (overuse) / stress (tensile; compression; torsion; bending; shear) / intrinsic issue / extrinsic issue / direct / indirect / functional disorder / structural injury / instability (passive / dynamic) degenerative / insidious / sequelae / complications / predisposition / re-injury / preventable / accidental / compensatory / physiological / psychological
Age, gender, body type and performance level	Male / female / child / adolescent / adult / middle-aged / elderly / disabled / ectomorph / mesomorph / endomorph / novice / veteran / recreational / amateur / elite / professional
Body region	Head / facial / spinal / cervical / thoracic / lumbar / sacral / coccygeal / abdominal / pelvic / upper extremity / shoulder / elbow / wrist / hand / fingers / lower extremity / pelvic / hip / knee / ankle / foot / toes
Sports-related	Individual / team / contact / non-contact / high-risk / endurance-based / power-based / agility-based / linear / multidirectional / environment / terrain / level / position
Training, preparation and competition	Fitness: under-conditioned / over-trained / inadequate recovery, fatigue and physiological change (underperformance / overtraining syndrome) / sports-specific / nutritional deficiencies / dehydration / psychological factors / equipment factors / inadequate warm-up / cool-down / lifestyle factors / inadequate risk assessment or needs analyses / inadequate prehabilitation / access to medical and therapeutic support
Pain and symptoms	Types of pain (e.g. sharp; shooting; deep; throbbing; aching; stinging) / pain scales (e.g. VAS/NRS 0-10) / local pain / referred pain / radicular pain / somatic pain / peripheral neuropathic pain / myofascial pain / trigger point pain / DOMS / phantom pain / pain threshold / pain tolerance / pain syndromes (complex regional pain syndrome; fibromyalgia) / central sensitization / psychogenic pain / allodynia / hyperalgesia / hypoalgesia / causalgia / analgesia / nociception / noxious stimuli / nociceptive pain / dysaesthesia / fatigue / inhibition
Audit	Injury surveillance / statistical analyses – prospective / retrospective (i.e. numbers; percentages); populations (e.g. general; age; gender; sport; level; injury); prevalence (i.e. how common) / incidence (i.e. how often in a given timeframe); time out of action / games missed

therapist is able to clinically reason the differential diagnoses when undertaking assessment, as well as consider optimal prevention and rehabilitation strategies.

Alongside the most well-recognized sports-related injuries, the sports therapist must also be prepared and able to respond to the multitude of other medical conditions and concerns that may present in their work. These can include everything from known pre-existing conditions to acute infections, allergic reactions and the potentially more subtle psychosocial issues. With such a broad spectrum of conditions and the obvious potential for mismanagement and development of complications, the sports therapist must be efficient in screening and assessing

their patients/athletes so as to be sure that individuals are managed as safely and effectively as possible and that medical advice is always sought whenever there is a lack of certainty or any doubt regarding the seriousness of the presenting condition. Conditions where the sports therapist must demonstrate cautiousness in their practice are extremely wide-ranging and not always obvious; they can range from contagious skin conditions (fungal; bacterial; viral) to complex injury presentations or suspicious clinical 'red flag' signs. If the practitioner has any doubt as to the suitability of the patient to receive sports therapy, then it is essential that medical advice is sought.

Functional regional anatomy and pathology are detailed elsewhere in this text, alongside the process and methods for efficient practical clinical assessment and strategies for management. Sports therapists will recognize that confident clinical reasoning strategies are central to being able to autonomously perform competent professional and ethical decision-making during both the assessment and the management of their patients/athletes.

Acute injuries and conditions

One single significantly traumatic episode can result in an acute injury, or aggravation of an existing injury, and the mechanism of that injury may be obvious. The classifications of contact injuries differ and are dependent on the type of tissue that suffers the damage. Acute bone injuries include fractures and periosteal contusions (bone bruising), while articular cartilage can experience osteochondral and chondral damage. Ligament injuries include sprains (graded I–III degree); joints can also suffer capsular irritations, dislocations, labral or meniscal damage. Acute *functional* muscular disorders include fatigue-related conditions, exercise-induced muscle disorders (EIMD) and exercise-associated muscle cramps (EAMC); and *structural* muscular problems include graded (mild, moderate or total) tears and contusions (Mueller-Wohlfahrt *et al.*, 2013). Cook and Purdam (2009) have documented the condition of an acute reactive tendinopathy. Acute skin injuries obviously include cuts, abrasions, lacerations and puncture wounds. Bursae can be subject to traumatic bursitis, which is most common at the hip (i.e. trochanteric), knee (pre-patellar and infra-patellar) and elbow (olecranon). Acute injuries to peripheral nerves can include transient neuropraxia (temporary paralysis ['conduction block'] resulting from direct impact or over-tensioning), axonotmesis (traumatic damage to the axon of the neuron) or neurotmesis (more severe neuronal and sheath damage); these have been traditionally classified by both Seddon (in 1943) and Sunderland (in 1951) (Faubel, 2010).

Traumatic head injuries are extremely common in sports. High-risk sports include combat sports, where the head is a legitimate target (boxing; martial arts); contact sports such as rugby and football; non-contact sports such as motor racing, equestrian, gymnastics and trampolining; and sports where head injury is rare but potentially severe (golf; cricket). Head and facial injuries include: skin wounds; contusions; eye gouging; perichondral haematoma (a blow to the ear causing subchondral haemorrhage [bleeding under the cartilage of the ear] which can separate tissues and restrict nutrient supply to the cartilage, leading to fibrosis and thickening – commonly known as 'cauliflower ear'); epistaxis (nose bleed); cranial or facial bone fractures; dental injury (dislodged teeth); temporomandibular joint (TMJ) dislocation; concussion and intracranial haemorrhage. Concussion is a common presentation and always a serious concern – its assessment and management are discussed in Chapter 4.

Contact injuries

Contact injuries occur as a result of an extrinsic force such as a collision with another player, or being struck by an object such as a hockey stick. Sporting examples of contact injuries include:

- Rugby: where the tackler hits the ball carrier on the anterior aspect of the knee causing a forced hyperextension, and sustains a posterior cruciate ligament sprain.
- Football: where two players go for a tackle and make shin-to-shin contact resulting in one player sustaining a fractured tibia and fibula.
- Basketball: where the player receiving a pass has a finger hyperextended by the ball, resulting in ligamentous sprain.

Non-contact injuries

Non-contact injuries occur as the body interacts with its environment. These may relate to individual predisposition (such as regional static and/or dynamic instability or previous injury), or simply to the combinations of forces and body positioning that occur during sport. Examples of non-contact injuries include:

- Rugby: where the player accelerates rapidly in order to chase or evade an opponent and sustains a hamstring tear.
- Football: where the player performs a change of direction or cutting manoeuvre resulting in forced inversion, and sustains a lateral ankle sprain.
- Basketball: where the player lands in full knee extension, or pivots on a flexed knee with the foot planted, and sustains an anterior cruciate ligament sprain.

Overuse injuries and chronic conditions

Whiting and Zernicke (1998) define use as 'normal functional loading' and overuse as 'repeated overload or force application'. Overuse injuries occur as a result of a repetitive overload or micro-trauma and therefore the mechanism of injury may be less obvious. Classification of overuse injury differs depending on the type of tissue that suffers the damage. Overuse bone injuries include stress fractures, apophysitis, osteitis, periostitis and bone strain. At synovial joints, capsulitis or synovitis can occur, articular cartilage can suffer from chondropathy and fragmentation; osteoarthritis is the most common degenerative musculoskeletal condition. The fibro-cartilaginous intervertebral discs and the meniscal cartilages of the knee are vulnerable to degenerative changes. While delayed onset muscle soreness (DOMS) may not be specifically categorized as an overuse muscle condition (as it can occur due to one single intense bout of unaccustomed exercise), it can present as a recurring problem; chronic exertional compartment syndrome (CECS) is recognized as an overuse condition. Tendinopathy, and in particular degenerative tendinopathy (tendinosis), is a highly prevalent overuse condition. Blisters, calluses and corns are examples of overuse conditions affecting the skin. Bursae are vulnerable to trauma from repeated impact or repetitive friction from tendon movement and can develop bursitis, which typically presents as visibly localized, extra-articular swelling and redness, and associated pain, tenderness and impaired function. Overuse nerve injuries may present as nerve irritations at their mechanical interfaces, which can be associated with adverse neural tension. A range of neural symptoms, such as distal paraesthesia, weakness in motor distribution and radicular pain can occur. Beyond this, peripheral neuropathy is the developmental dysfunction (abnormal firing and pain generation) from peripheral neural tissue (resulting from such situations as prolonged compression, local ischaemia and hypoxia, local neuroma or systemic demyelinating disease) (Butler and Tomberlin, 2007).

Sporting examples of overuse injuries include:

- Tennis: where the adolescent tennis player increases intensity of tennis play using the semi-Western or Western grip and sustains metacarpal stress fracture (Tagliafico *et al.*, 2009).
- Runners and cyclists: where the constant and repetitive flexion and extension of the knee causes the iliotibial band to rub against the lateral femoral epicondyle and the athlete suffers iliotibial band syndrome (ITBS).
- Swimming: where technical faults in conjunction with structural and functional imbalances can contribute to rotator cuff (sub-acromial) impingement.

Children and adolescents

There are a host of injuries and problems associated with younger age groups, for example the common adolescent growth spurt conditions, such as Osgood–Schlatter's lesion – a traction apophysitis, or micro-avulsion, of the patella tendon at the tibial tuberosity; Sinding–Larsen–Johansson lesion – a similar pathology which affects the attachment of the patella tendon at the inferior pole of the patella; and Sever's lesion – affecting the Achilles tendon insertion at the calcaneus. The majority of these conditions tend to settle within months and are generally recognized as being 'self-limiting' – that is they usually resolve within a predictable timeframe (months), and where intervention focuses upon symptom management (i.e. cryotherapy; taping), activity modification and progressive soft tissue and manual therapy. Occasionally, complications such as continued pain or fragmentation can occur (Gholve *et al.*, 2007).

There are several other common children's conditions with characteristic presentations simply attributed to musculoskeletal immaturity. Legg–Calve–Perthes disease is an idiopathic osteochondropathy of the hip, with characteristic flattening of the femoral head. It affects males more than females and is most commonly unilateral. The condition, which is associated with delayed skeletal maturity, typically begins with synovitis of the joint capsule, followed by an avascular (and necrotic) stage, which often leads to fragmentation and malformation. During this process, the child is likely to present with local aching and a painful limp. Once identified, ossification restarts and continues as activities are modified and the condition settles down, typically over a two-year period. Prognosis is generally good, but early onset osteoarthritis can be a concern (Placzek and Boyce, 2006).

Epiphyseal growth plate fractures are not uncommon. The growth plate is a cartilaginous centre of ossification at the metaphysis (between the diaphysis and epiphysis) in the long bones of adolescents. The growth plate can be considered as a vulnerable link – tendons and ligaments are relatively stronger than the growth plate and are also considerably more elastic, and as such the plate, being weaker, can give way when exposed to excessive stress. In adolescents, growth plate disruption is actually more common than ligamentous injury (Shanmugam and Maffulli, 2008). Once full skeletal maturity has occurred, the plate becomes a structurally stronger epiphyseal line (of mature bone) and is no longer active as an ossification centre. The condition can occur following a single trauma or from chronic stress. Certain individuals may have additional predisposing factors for epiphyseal growth plate fractures, such as being overweight, or underweight and tall, or simply sexually immature. Common sites for disruption are the: phalanges of the hand; distal radius; proximal and distal humerus; proximal and distal fibula; proximal and distal tibia; proximal and distal femur. Like many other types of fracture, there are established classification systems, the most recognized being the 'Salter–Harris' system (I–V), which categorizes different fractures according the degree of damage to the growth plate and the line of fracture or fragmentation (Peterson, 1994). A slipped capital femoral epiphysis (SCFE) occurs as the femoral shaft shifts proximally on the epiphysis. It occurs more in males than females and is frequently bilateral (McRae, 2006). The main complication associated with

growth plate fractures is disruption to the normal process of bone lengthening and development and the possibility of deformity (Caine *et al.*, 2006). Growth plate fractures usually require cast immobilization and possibly operative fixation. Osteochondritis dissecans (OCD) is an adolescent idiopathic condition affecting subchondral bone and its adjacent articular cartilage, resulting in fragmentation of articular tissue. The intercondylar region of the femur at the knee, the talus at the talocrural joint and the capitulum at the elbow are the most commonly affected areas (Shanmugam and Maffulli, 2008). It is a condition that occurs more commonly in sporting males. Insidious onset of pain, swelling and restricted joint movements are characteristic symptoms, usually confirmed via plain radiographs or MRI (magnetic resonance imaging). Scheuermann's disease is a physeal osteochondropathy affecting the thoracic vertebral end growth plates. The condition, which is more common in active adolescents and young adults, typically leads to a 'wedging', usually of several adjacent vertebral bodies, and characteristically results in a kyphotic posture (MacAuley, 2007). The so called 'greenstick' fractures of long bones only occur in children and adolescents. These present as incomplete breaks on the convex side of the immature bone.

The sports therapist must also be vigilant to being able to recognize the possibility of symptoms that could be associated with underlying structural or developmental abnormality. Such conditions, although rare, include: lumbarization (abnormal separation and mobilization of S1 from S2); sacralization (abnormal fusion between L5 and S1); cervical rib (abnormal additional costal bone); pars defect (an abnormal neural arch, most commonly at L5); hooked acromion (abnormal inferior angulation of the acromion process – reducing the subacromial space); bipartite or multipartite patella (abnormal segmentation of the patella – usually unilateral and more common in males); discoid meniscus (abnormal discoid configuration of the meniscus); tarsal coalition (abnormal fibrous fusion of adjacent tarsal bones). Such presentations serve to remind us that all individuals are unique and every single patient's anatomy is different and has the potential to be the primary cause of their symptoms.

In addition to their developmental predisposition to particular kinds of injury, children and adolescents have some degree of increased vulnerability to injury simply because of limitations in their experience of situations and their awareness of their body and space (spatial awareness). Furthermore, younger athletes are sometimes 'pushed' inappropriately by parents or coaches. Brukner and Khan (2009) have presented the 'ugly parent syndrome', in which symptoms ranging from simple head, stomach and muscle aches to stress-related sleep and emotional disturbances may occur simply due to the excessive parental pressure to compete. Children should be encouraged but not forced and any participation in sport should be for the child's enjoyment (not the parent's) and healthy development at this stage of life. Additionally, in supervised sport, children should be matched appropriately, be provided with correct equipment and made aware of the rules and the reasoning behind them.

Males and females

MacAuley (2007) states that females may be more prone to sports injuries for anatomical reasons, and it is known that females are more vulnerable to specific types of sports injury when compared to males (for example, ACL injuries, patella dislocation and patellofemoral pain syndrome). The prevalence of males experiencing adolescent traction apophysitis or osteochondritis is significantly higher. It has also been said that males are more prone to injury, experience more injuries and die more frequently from injuries simply because they are more likely to engage in risk-laden behaviour (Udry, 1998). Perhaps the issue of gender predisposition to injury becomes more apparent with inexperienced or recreational athletes, who are less likely

to have access to the training methods and medical support that is normally available to elite athletes.

Males and females of the human species have many anatomical and physiological differences, but, as highlighted by Bennell and Alleyne (2009), they have far more similarities than males and females of many other species. A number of average and very generalized differences are recognized (Table 1.2), many of which are contentious for a number of reasons, not least the uniqueness of every individual and the potential for both anatomical and physiological development via training and nutrition. Hence such generalizations must be recognized as such;

Table 1.2 Average anatomical and physiological differences between males and females

Males	*Females*
Commence adolescent growth spurt around 13 years of age.	Commence adolescent growth spurt around 11 years of age.
No menarche, menstrual cycle, pregnancy or menopause.	Commence menarche between 12 and 14 years of age. Experience regular monthly menstrual cycles. May experience one or more pregnancies (period of 37–42 weeks, culminating in childbirth). Menopause (cessation of normal menstrual cycle) in middle age.
Reach maximal height around 20 years of age.	Reach maximal height around 16 or 17 years of age.
Generally are taller and heavier.	Generally are shorter and lighter.
Higher centre of gravity.	Lower centre of gravity.
Heavier and thicker bones.	Lighter and thinner bones.
Larger articulatory surfaces.	Smaller articulatory surfaces.
More distinct muscle attachment sites.	Less distinct muscle attachment sites.
Proportionally longer limbs.	Proportionally shorter limbs.
Proportionally wider biacromial width.	Proportionally narrower biacromial width.
Lesser elbow carrying angle.	Greater elbow carrying angle.
Proportionally larger thorax.	Proportionally smaller thorax.
Penis and testicles, small amount of breast tissue.	Vagina, ovaries and breasts.
Proportionally thicker waist.	Proportionally narrower waist.
Proportionally deeper pelvis and narrower hips.	Proportionally shallower pelvis and wider hips.
Lesser femoral angle of inclination.	Greater femoral angle of inclination.
Lesser genu valgus and Q angle.	Greater genu valgus and Q angle.
Lower average body fat composition (14%).	Higher average body fat composition (26%).
Greater subcutaneous body fat around abdomen and upper body.	Greater subcutaneous body fat around hips and thighs.
Higher lean body mass.	Lower lean body mass.
Greater muscle mass.	Lesser muscle mass.
Greater percentage of blood in body fluid.	Lesser percentage of blood in body fluid.
Larger heart and lungs.	Smaller heart and lungs.
Greater stroke volume and cardiac output.	Lesser stroke volume and cardiac output.
Lower resting heart rate.	Higher resting heart rate.
Lower blood pressure.	Higher blood pressure.
Greater respiratory rate and vital capacity.	Lesser respiratory rate and vital capacity.
Greater aerobic capacity and VO2 max.	Lesser aerobic capacity and VO2 max.

and clearly stereotypical classifications have their limitations. However, it is important for the sports therapist to recognize the definitive, apparent or potential characteristics of males and females; and in so doing, the aetiology of injuries may be more readily ascertained and the prevention and management of injuries improved.

Within sports medicine, females are frequently discussed as a special population, just as are children, older athletes and the disabled. The average or generalized differences highlighted in Table 1.2 will help the sports therapist appreciate some of the gender-unique issues that females may have to contend with. Clearly, any individual having less than ideal biomechanics for particular sports is at a disadvantage and offers an obvious increased vulnerability to injury. However, it must be recognized that many individuals do not conform to such generalizations; and also the majority of active individuals will choose to undertake sports and training to which they are best suited.

The hormonally regulated menstrual cycle, or monthly period, is a normal part of the female reproductive cycle. During the cycle, a woman bleeds from her uterus via the vagina for a period of three to seven days; this is known as menstruation – a process repeated every month until menopause – unless fertilization of her ovum occurs and she becomes pregnant. There are other normal reasons for absent periods (amenorrhea), such as taking the contraceptive pill. Some females experience occasional pre-menstrual syndrome, which has been defined as a collection of emotional and physical symptoms (for example: anxiety; depression; mood swings; headaches; fluid retention; abdominal and back pain; breast soreness) and which commonly occur prior to the monthly cycle. Interestingly, it has been identified that regular exercise may help to reduce such symptoms. Dysmenorrhea (painful menstrual cramps) can also occur early in the cycle due to transient ischaemia in the smooth muscular wall of the uterus. There are a number of menstrual irregularities which are associated with frequent and intense exercise, as explained by Bennell and Alleyne (2009). These include: delayed menarche (delayed commencement of menstrual bleeding); oligomenorrhea (irregular menstruation); amenorrhea (absent menstruation). Sports therapists, although not expected to be expert in such matters, should recognize, or at least aim to identify, when menstrual issues may be present.

Two main complications of menstrual cycle irregularities include reduced fertility and reduced bone mass. Particularly, amenorrheic female athletes have increased incidence of stress fractures; and osteoporosis is a concern for post-menopausal women with reduced bone mass, and therefore predisposition to pathological fractures (Sherry and Wilson, 1998).

The 'Female Athletic Triad' was first presented in 1992 by the ACSM (American College of Sports Medicine) as a term to describe a condition characterized by a combination of: (1) disordered eating (most commonly anorexia nervosa and bulimia nervosa); (2) delayed menarche or amenorrhoea (intense exercise related); and (3) (amenorrhoeic) osteoporosis or osteopenia.

However, a recent IOC consensus statement regarding 'Relative Energy Deficiency in Sport' and the acronym term 'RED-S' have been presented. RED-S has been defined and designed in an effort to supersede the term Female Athletic Triad (Mountjoy *et al.*, 2014). In presenting the RED-S condition, the aim was to emphasize the pathophysiological complexity and multisystem involvement, and the fact that, while aetiology can vary, the condition may affect both men and women. In RED-S the imbalance in energy availability and energy expenditure is evident; and as Swe Win and Thing (2014) explain, there also occurs a misbalance between training load and training recovery. However, it is important to appreciate the critical response to the IOC statement by De Souza *et al.* (2014a), who ascertained that it was *'ill-conceived and poorly defended'*. They argue that the progressing research platform and established practical management strategies for affected female athletes must continue, and that the 'Female Athletic Triad' concept and definition should not be subsumed under the umbrella of RED-S.

The signs and symptoms of this condition are not always easy to recognize, and indeed individuals can appear 'normal'. Pantano (2009) stated that *'physical therapists must be responsible for recognizing, treating and preventing the Female Athlete Triad'*. Disordered eating, or 'eating distress' may be due to psychological, biological or sociological factors (MacAuley, 2007) and can lead to a whole host of health problems, not least low energy availability. It is, however, more prevalent in sports where athletes are required to maintain a specific body weight and composition, such as gymnastics, synchronized swimming, ballet or distance running (Back and Smethurst, 2004). It is certain that affected individuals are likely to be discreet in their behaviour. Disordered eating results in calorific and nutritional deficiency and signs of such can include loss of muscle tone, skin problems, cold extremities, swollen face and ankles. Where there is imbalance in energy availability and energy expenditure, both short-term (performance deficits; stress fractures) and long-term (osteoporotic fractures; infertility) health consequences can manifest. Other consequences of insufficient energy availability include low immunity and risk of infection, and chronic fatigue. A recent Female Athletic Triad expert consensus statement (De Souza *et al.*, 2014b) recommended a set of pre-participation screening questions for vulnerable female athletes:

- Have you ever had a menstrual period?
- How old were you when you had your first menstrual period?
- When was your most recent menstrual period?
- How many periods have you had in the past 12 months?
- Are you presently taking any female hormones (oestrogen, progesterone, birth control pills)?
- Do you worry about your weight?
- Are you trying to or has anyone recommended that you gain or lose weight?
- Are you on a special diet or do you avoid certain types of foods or food groups?
- Have you ever had an eating disorder?
- Have you ever had a stress fracture?
- Have you ever been told you have low bone density (osteopenia or osteoporosis)?

Taking place over 37 to 42 weeks and involving three trimesters, pregnancy brings with it normal physical and physiological changes, a certain vulnerability to both mother and foetus and a collection of potential complications. Some of the more obvious changes occurring during pregnancy include: weight gain; breast enlargement; lung compression and diaphragm displacement; shift in centre of gravity with progressive lumbar lordosis; fluid retention; ligamentous laxity; increasing joint stress; and emotional changes. It is usual for active women to continue to exercise during pregnancy. Reasons not to exercise include: infection; fetal distress; placenta praevia; pregnancy-induced hypertension; pre-term rupture of membranes; persistent second or third trimester bleeding (Cowey, 2006; MacAuley, 2007). Particular signs of exercise being inappropriate include: dyspnoea; dizziness; chest pain; persistent headache; radicular pain; carpal tunnel syndrome; leakage of amniotic fluid; decreased fetal movement; calf pain or swelling (indicating the possibility of deep vein thrombosis [DVT]); and any other individual concerns. Furthermore, exercise is contraindicated in the first trimester where there is history of spontaneous abortion. Any concerns should be discussed with or referred to the individual's GP, midwife or gynecologist.

The female's breast is an apocrine mammary gland. Its primary function is to produce breast milk (lactation) for breastfeeding. Breasts overlie the pectoral region and are predominantly composed of connective fatty tissue (collagen, elastin and adipose tissue). The superficial

Cooper's ligaments suspend the breast's fascia to the overlying skin superiorly from the clavicular region and each breast contains a nipple, areolar, lactiferous (milk) lobes and ducts, blood vessels, nerve fibres and lymphatic channels. The majority of lymph from the breast region channels to the axillary lymph nodes. Females have to contend with the potential for direct traumatic injuries involving their breasts, which can lead to haematomas. Although males can suffer nipple problems, females have increased predisposition, particularly those undertaking distance running. 'Jogger's nipple' is where nipples are constantly irritated by the friction of clothing. Bleeding is common, and prevention is key. Excessive up and downward movement of breasts during exercise is also an issue and can easily lead to pain and reduced performance; correctly fitting sports bras should be recommended (Bennell and Alleyne, 2009). Finally, breast cancer, although far from being unique to females, is one of the most common cancers; regular self-examination for unusual lumps, skin dimpling or discolouration, nipple changes or bloody discharge must be encouraged.

Complications of injury

Not all sports or exercise-related injuries and problems follow a straightforward course of recovery. Injuries can become chronic, affecting ability to train or compete; secondary, or compensatory, injuries can develop; health problems can worsen; tissues can degenerate or fail to heal effectively; injuries can be recurrent; infection can occur (or reoccur). Recovery can be slow and problematic; setbacks are not uncommon. On return to full sporting action, if underlying aetiologies have not been attended to, or appropriate progressive and final stage rehabilitation undertaken, then re-injury or new injury can result. Comorbidities commonly exist which compound the injury's recovery process. Patients may not have been provided with a confirmative medical diagnosis, meaning increased potential for inappropriate management. Of course, patients may not even receive appropriate intervention or advice in the first instance. Furthermore, patients/athletes may not follow recommendations (for various reasons – intrinsic and extrinsic) which can have significant negative impacts on eventual outcomes. Non-adherence (more historically referred to as 'non-compliance') is a complex topic and involves a host of psychosocial factors (Magee *et al.*, 2008). Greenhalgh and Selfe (2006) present a summary of the psychosocial 'flag system': 'yellow flags' – emotional and behavioural factors; 'blue flags' – social and economic factors; 'black flags' – occupational factors; and 'orange flags' – psychiatric factors. Iatrogenic factors can also occasionally contribute to long-term problems; these relate to adverse effects of therapeutic or medical intervention. Therapists should aim to appreciate how long-term injuries can impact on the individual. Loss of self-esteem, financial implications and developing frustrations, fears and depression are all real concerns, particularly in professional sport. In an ideal setting these will be considered and managed carefully and responsibly.

Even when full and appropriate rehabilitation has taken place, the athlete could still be faced with a range of post-injury detriments, for example: reduced tissue strength; joint instability; excessive repair (scar) tissue; adverse stiffness; muscular weakness; motor control issues; persistant pain; restricted joint mobility; adverse neural tension; soft tissue calcification; early onset osteoarthritis; deformity; fear of use or apprehension. The sports therapist must aim to recognize when injuries are not responding as should be expected and they should be able to make autonomous decisions on appropriate recommendations, such as referral for second opinion, or for imaging or other investigation.

Injury aetiology

With Dr Andrew Mitchell

Forces acting upon the human body

The human body is remarkably resilient to the stresses and strains exerted upon it by not only activities of daily life, but also sports and exercise. In daily life, humans exert force on the ground and stairs while walking, in opening and closing doors and by lifting or carrying objects. During sports and exercise we exert often greater force on the ground, and more often while running than walking, and we also apply force against balls, clubs, racquets, water and wind. In contact sports such as rugby, force is applied against other human beings. On a daily basis the tissues of the body experience innumerable loads of varying intensities and suffer no ill effects. Injury only occurs when a force overloads the tissues' ability to withstand it, resulting in tissue failure and damage. Force may be defined as *'the mechanical action or effect applied to a body that tends to produce acceleration'* (Whiting and Zernicke, 1998).

There are a number of forces that athletes are subjected to that may cause injury. Gravity is the force that has the greatest influence on human movement (Mester and Macintosh, 2000) and is a downward force tending to accelerate objects at 9.8 m s^{-2} (Whiting and Zernicke, 1998). A ground reaction force (GRF) is a force that acts from the ground on any structure that is in contact with it (for example, the hands in a cartwheel, or the feet during gait). If any part of an athlete's body is in contact with the ground it will exert a force upon it, the ground in turn will then exert a force that is equal and opposite.

The impact force from an external source making contact with the body can cause injury (for example, being tackled in rugby or hyperextending a finger while catching a basketball). Compressive forces from structures within the body acting upon each other can cause injuries, for example, landing on one's feet from a high fall could result in compression of the femur, tibia, fibula, tarsals or any other weight-bearing bones, causing fracture. Intrinsic tensile forces, such as those at the musculotendinous junction or across a ligament acting over a joint can result in muscle tears, sprains or avulsion fractures.

Uniaxial forces

An axial force is a force that acts along the longitudinal axis of a structure. There are three forms of uniaxial loading: compression, tension and shearing (Figure 1.1).

Compressive forces cause a crushing or squeezing of the tissues. Compression of a high load can cause a single traumatic injury, for example, being struck on the forearm with a hockey stick, which may result in bruising of the forearm musculature or, more significantly, a fracture of the radius or ulna. Typical compression injuries include fractures, stress fractures, haematoma,

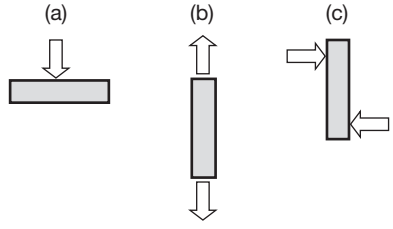

Figure 1.1 Uniaxial force of injury: (A) compression; (B) tension (C) shearing.

puncture wounds and, as a complication, myositis ossificans. Compressive forces can also be more subtle. Abnormal bony projections, for example, can cause repetitive compressive stress to local soft tissues (most commonly tendons) during joint movement. Also, where peripheral nerves pass through narrow anatomical tunnels (mechanical interfaces), compression can cause restriction to normal movement (adverse neural tension) and also restriction of normal blood flow to the nerve.

Tensile forces cause a pulling, longitudinal stretching of tissues. Tension of a high load can cause soft tissue tears and ruptures, whereas intermittent tension can strengthen muscle and tendon. Tensile injuries include ligamentous sprains, musculotendinous tears and avulsion fractures.

Shear forces cause a horizontal or oblique stress movement of one tissue over another, for example an excessive anterior tibial shear force acting on the anterior cruciate ligament can cause sprain or rupture. Shearing injuries include: sprains; meniscal and labral tears; long bone spiral fractures; isthmic spondylolistheses; and certain tendinopathies. Shear forces have been shown to increase within the knee joint if the supporting musculature is fatigued, which can put the knee at a greater risk of non-contact ACL injury (Chappell *et al.*, 2005). Equally, shear forces of a low load, with repetition, can lead to abnormal stress to tissues, such as with frictional foot blisters (Yavuz and Davis, 2010). Tendon and fascial tissues are particularly vulnerable to shear stress, for example the 'whipping' shear stress exposed to the Achilles tendon or the plantar fascia easily created in the presence of excessive foot pronation during running gait. Tendons are also vulnerable to the abusive overloading that can occur during episodes of rapidly repeated activity, especially where less than ideal biomechanics present (Sharma and Maffulli, 2006) – a common cause of acute reactive tendinopathy (Cook and Purdam, 2009).

Compression, tension and shearing stresses often combine and act on the same joint during sporting movements. For example, in the throwing athlete's elbow, tension is applied to the medial stabilizing structures, compression is applied to the lateral structures and a shearing force is exerted to the posterior structures (Cain *et al.*, 2003).

Multiaxial forces

Torque can be thought of as a rotational force and is the product of the magnitude of the force applied and length of the moment arm. When a muscle contracts it produces torque over the joint it crosses and this forms the basis for human movement. Torque caused by muscles rarely causes tissue damage; instead it is usually torque caused by an external source that is responsible for injury. There are two forms of multiaxial loading: bending and torsion (Figure 1.2).

A structure such as a long bone will bend if a force is applied to it, perpendicular to its longitudinal axis. During the bending situation, compression is applied to the concave surface and tension occurs at the convex surface. Bone is better at resisting compression rather than tension, and if the magnitude of force is great enough, fracture will occur on the side of the bone under tension.

A structure will experience torsion if a twisting force is applied to it along its longitudinal axis. A non-anatomical example of this is removing the cap from a bottle of water. A shear stress is created along the bone during a torsion injury. A typical torsional injury is a spiral fracture which is commonly seen in sports such as skiing and snowboarding, and as momentum is occurring and with the leg firmly fixed in the snow, the individual rotates around it during the fall, potentially resulting in excessive force to the lower limb bones. Obviously, such forces can lead to injury of any of the involved tissues (bone; muscle; tendon; ligament; cartilage; skin; nerve; blood vessel; viscera). Different tissues have differing properties and capacity to withstand different forces, and injury is usually sustained to the weakest tissues (or links) in the kinetic chain as these are the tissues that will fail first when put under stress and strain.

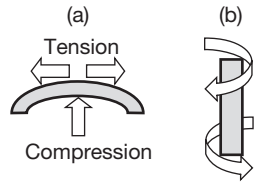

Figure 1.2 Multiaxial force of injury: (A) bending; (B) torsion.

There are a number of factors that determine the nature and severity of injury when force is applied to human tissues:

- **Area**: the risk of injury increases when force is applied to smaller areas.
- **Direction**: some tissues are more efficient at dissipating force in certain directions.
- **Duration**: acute injuries usually occur very quickly (30–50 ms), while overuse injuries occur over a much longer period of time.
- **Frequency**: acute injuries usually occur as a result of a single traumatic overload, while overuse injuries occur as a result of chronic repetitive loading.
- **Location**: where the force is applied to the structure will determine the tissues' ability to withstand it.
- **Magnitude**: injury occurs if the amount of force applied exceeds the tissues' ability to withstand it.
- **Rate**: if a force is applied quickly it may cause injury, but if that same force is applied gradually the tissues may well be able to withstand it.
- **Tissue type**: the large mass of the quadriceps muscles are less likely to suffer injury after a blow compared to the bony prominence of the olecranon process.
- **Variability**: whether the magnitude of the applied force is variable or constant will determine the extent of injury sustained.

Primary and secondary injury

A primary injury is one that occurs as an immediate and direct result of a trauma – for example, sustaining a haematoma on the thigh after being struck by a hockey ball. A primary injury is an acute catastrophic failure of the tissues. A secondary injury has a gradual onset and may appear some time after the initial trauma due to a disruption in blood flow, nerve supply or structural vulnerability. For example, degenerative change in the Achilles tendon (tendinosis) is associated with increased risk of spontaneous rupture (Kannus and Józsa, 1991). Another form of secondary injury is known as a compensatory injury, and can occur when an athlete consciously or unconsciously adjusts their biomechanics in order to accommodate for a primary injury. For example, a long-distance runner recovering from a lateral ankle sprain may subconsciously modify their gait, causing increased risk of stress fractures of the foot, or other problems through the kinetic chain.

Macrotrauma and microtrauma

Macrotrauma is an injury from a single acute force that causes immediate tissue damage such as a fracture, contusion, muscle tear, ligamentous sprain or neurotmesis. While microtrauma following appropriate loading is central to the process for progressive increases in strength and

resilience in tissues (during either rehabilitation or fitness training), inappropriate microtrauma when repetitively or chronically overloading tissues at a microscopic level can lead to a breakdown in tissue structure. The forces involved in a single microtrauma will not cause injury; but it is the repeated inappropriate loading and resulting weakening of the tissue, due to insufficient recovery, that leads to degenerative conditions and in some cases subsequent macrotrauma. Taljanovic *et al.* (2011) present the example of repetitive microtrauma occurring from overhead hitting in female volleyball players, initially causing laxity of the inferior capsule which can lead to subsequent avulsion of the humeral attachment of the inferior glenohumeral ligament.

Stress, strain and deformation

The response of human tissue to progressive force application can be elastic or plastic in nature. Figure 1.3 shows how excessive force application can result in tissue deformation. If the load is applied at point A and released at point X, before reaching the elastic limit or yield point, the tissue will not suffer any long-term deformation and will return to its original shape and size. The response of the tissue to the stress is elastic. If, however, the load is applied at point A, but not released until point Y, which is beyond the elastic limit, the tissue will suffer long term deformation and will not return to its original shape and size. In such a case, the response of the tissue is plastic.

If the applied load exceeds the tissues' ultimate failure point then mechanical failure occurs. Figure 1.4 demonstrates how excessive force application can result in tissue failure. The tendon will cope with a high load applied at point A, before failure at point T. Muscle tissue will deform under low load, then respond stiffly before failure at point M. Bone responds stiffly to the load applied at point A, and undergoes minimal deformation prior to failure at point B (Whiting and Zernicke, 1998).

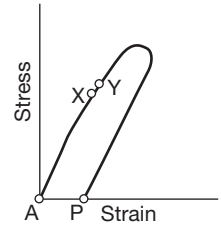

Figure 1.3 Stress–strain curve showing tissue deformation.

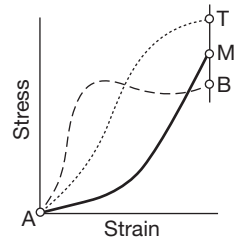

Figure 1.4 Stress–strain curve showing tissue failure (T = tendon; M = muscle; B = bone).

RISK FACTORS FOR INJURY

With Dr Andrew Mitchell

The sports therapist will appreciate that a major component of injury prevention is understanding and assessing the intrinsic and extrinsic risk factors. Leadbetter (1994), cited by Whiting and Zernicke (1998), identified a number of factors that can contribute to injury:

- contact or impact
- dynamic overload
- inflexibility
- muscle imbalance
- overuse
- structural vulnerability
- rapid growth.

Such factors are fundamentally interlinked and may act in unison to cause injury. For example, if an athlete has tightness in a hamstring muscle as a result of a history of previous strains, then inflexibility, muscle imbalance and subsequently dynamic overload may contribute to a new acute hamstring strain. In many cases, these factors may be identified by pre-participation screening, monitoring and evaluation of athletes. If a potential risk factor is observed then the sports therapist can implement prehabilitation techniques in order to reduce the chance of injury occurring. Meeuwisse (1994) presented an original multifactorial model of athletic injury aetiology, which aimed to demonstrate: the intrinsic risk factors predisposing the athlete to injury; the exposure to extrinsic risk factors during training or competition; the susceptible athlete; the inciting event; and the resultant injury. This model has been slightly modified in recent presentations, and remains relevant.

While there are numerous factors that contribute to an athlete sustaining an injury, these may be divided into two basic groups: intrinsic risk factors, which are individual, functional, anatomical, physiological, psychological and pathological and from within the body; and extrinsic risk factors, which are external, resulting from training and preparation errors, inappropriate equipment, environmental conditions and the very nature of competition.

Table 1.3 Intrinsic and extrinsic risk factors predisposing to injury

Intrinsic factors	*Extrinsic factors*
Structural and biomechanical abnormalities (e.g. excessive Q-angle; pes planus; leg length discrepancy)	Equipment
	Footwear
	Surface
Gait abnormality	Terrain
Muscle imbalance	Gradient
Age	Training errors
Excessive or limited flexibility	Coaching errors
Sex, size and body composition	Environmental conditions
Physiological issues	Nutritional issues
Previous injury	Sports officiating errors
Impaired proprioception and joint positional sense	Opposition players
	Inciting event
Sports-specific technique errors	
Psychological issues	

A thorough pre-participation/season screen can identify many intrinsic risk factors for an athlete and in some cases enable the sports therapist to intervene in order to mobilize, strengthen or enhance neuromuscular control where indicated. Formal profiling, particularly in professional settings, is utilized to establish a background picture of the individual athlete's health and injury status and functional ability performance. Profiling can include the individual's: previous medical history; injury history; body composition; blood pressure; blood constituents; nutritional status; lung function; heart function; basic cognitive functioning; balance and coordination; flexibility testing; functional movement screening (FMS); muscular strength and endurance assessment; VO2 max testing; agility testing; and sports-specific fitness assessment.

Structural and biomechanical abnormalities

The following common lower limb abnormalities are generally considered injury risk factors until proved otherwise because they alter biomechanics and may place excessive strain on involved structures during repetitive or excessive loading in sports or exercise. These may be unilateral or bilateral:

- excessive lateral, anterior or posterior pelvic tilt
- coxa valga and vara
- femoral anteversion and retroversion
- genu valgus and varus
- excessive Q-angle
- patella alta and baja
- 'frog-eyed' and 'squinting' patella
- genu recurvatus
- tibia varus and increased tibial external torsion
- ankle equinus
- valgus and varus heels
- rearfoot varus and rearfoot valgus
- plantarflexed first ray
- forefoot varus
- hallux valgus
- hallux rigidus
- pes cavus and planus
- excessive pronation or supination
- leg length discrepancy (true or apparent).

Average Q-angles are in the region of 10° for males and 15° for females; 20° is considered excessive (see Chapter 10 for Q-angle measurement technique). An excessive Q-angle has been shown to be a risk factor for patellofemoral pain syndrome (Haim *et al.*, 2006; Waryasz and McDermott, 2008) and lower limb stress fractures (Cowan *et al.*, 1996). Leg length inequality has been shown to be linked with lower extremity injury in runners (Fields *et al.*, 2010; Wen *et al.*, 1998) and specifically stress fractures (Korpelainen *et al.*, 2001) (see Chapter 9 for true and apparent leg length measurement techniques). Gait abnormalities are common and easily lead to injury problems. Runners with less pronation at heel or mid-foot strike, and those with laterally directed roll-off, may be at a greater risk of sustaining lower limb overuse injuries (Ghani Zadeh Hesar *et al.*, 2009). Equally, runners with excessive pronation and those exhibiting

greater medial pressures are at risk of exercise-related lower leg pain (Willems *et al.*, 2006). Chapter 2 provides a detailed discussion of gait assessment.

Many upper limb injuries are caused by muscular imbalance caused by tightness or weakness, but a number of biomechanical factors may contribute to upper limb injury, including:

- abnormal shape and angulation of the acromion
- reduced subacromial space
- abnormal scapula shape and malpositioning
- excessive elbow carrying angle (>15°)
- cubitus recurvatus and other hypermobility.

Muscle imbalance

Clark (2001) suggested that muscle imbalance is caused by a number of factors such as overtraining, postural stress, poor neuromuscular or technical efficiency and pattern overload. In turn, predictable patterns of kinetic dysfunctions occur, such as those identified by Janda (1983).

Muscle tightness or weakness of one muscle (agonist) can cause inhibition in the antagonist, which in turn causes dysfunction in the kinetic chain. Therefore the tight or hyperactive muscle dominates against the weak or inhibited muscle and disrupts normal function over the joint. The athlete compensates for this dysfunction by making postural modifications and by placing greater emphasis on the synergistic muscles to those that are weak. Clark (2001) referred to this as synergistic dominance and suggested that these muscles are now required to do more work in contributing to acceleration and deceleration and as a result may be more at risk of injury. Janda (1983) divided the muscular system into two: those muscles prone to tightness (movement group) and those prone to weakness (stabilization group).

Movement group characteristics

- Prone to developing tightness or hyperactivity.
- More active during functional movement patterns.
- Dominate during fatigue situations.
- Dominate when performing new movement patterns.
- Usually cross two joints.

Stabilization group characteristics

- Prone to developing weakness or inhibition.
- Less active during functional movement patterns.
- Prone to myokinematic and arthrokinematic inhibition.
- Easily fatigued.
- Usually cross one joint.
- Janda (1983) identified the muscles that are commonly found in each group.

Movement group (prone to tightness)

- Gastrocnemius
- soleus
- hip adductors
- hamstrings
- rectus femoris

- iliopsoas
- tensor fascia lata
- piriformis
- erector spinae
- quadratus lumborum
- pectoralis major
- upper trapezius
- levator scapulae
- sternocleidomastoid
- scalenes
- flexors of the upper limb.

Stabilization group (prone to weakness)

- Peroneals
- anterior tibialis
- posterior tibialis
- gluteus maximus
- gluteus medius
- abdominals
- serratus anterior
- rhomboids
- lower trapezius
- deep cervical flexors
- extensors of the upper limb.

Athletes with a muscle strength imbalance between the ankle inverters and everters have been shown to have an increased risk of lateral ankle sprain, as did athletes with a low dorsiflexion-to-plantarflexion strength ratio (Baumhauer *et al.*, 1995). Weak dorsiflexors and delayed peroneal reaction time contribute to the risk of sustaining an ankle sprain (Willems *et al.*, 2006). Weak adductor muscles are a risk factor for sustaining new groin injury (Engebretsen *et al.*, 2010), while hip extensor muscle imbalance has been shown to be associated with the incidence of low back pain in female athletes (Nadler *et al.*, 2001). Similarly, athletes who sustain knee injury have been shown to have significantly weaker hip abductors and external rotators (Leetun *et al.*, 2004). Furthermore, a hamstring-to-quadriceps isokinetically assessed strength ratio (HQR) below the normal average (where hamstring strength is less than 50 per cent of quadriceps) has been shown to increase the risk of non-contact knee overuse injuries in female athletes (Devan *et al.*, 2004).

Motor control issues

Reduced ability of the athlete to optimally recruit musculature during physical activity, particularly in multidirectional sport, can predispose to injury. In the dynamic and rapidly changing sporting environment, efficient motor control requires at least multi-faceted instant responsiveness. This process is partly informed by trained and ingrained feedback and feed-forward neuromuscular proprioceptive signalling, in addition to all the other multisensory inputs from each concurrent situation (environmental inputs). The neuromuscular system is also further challenged to respond to the varying duration, repetition, intensity and adaptation associated with any movement. Pain, restriction, weakness, neural or circulatory deficits compound the process.

Excessive or limited flexibility

The effects of a limited range of motion at a joint are diverse and may contribute to acute or overuse injury. For example, limited dorsiflexion range of motion is a risk factor for ankle sprains (Willems *et al.*, 2006), while it is also a risk factor for Achilles tendinopathy in volleyball players (Malliaras *et al.*, 2006). Conversely, genu recurvatus (hyperextending knee) has been shown to increase the risk of overuse injuries in the knee in female athletes (Devan *et al.*, 2004).

Joint hypermobility syndrome

Joint hypermobility, when associated with symptoms, is termed joint hypermobility syndrome (JHS). JHS can be due to hereditary, congenital, developmental or acquired factors. Obviously, there is increased potential for decreased static stability following significant damage to joint stabilizing structures. A key component to the development of this condition is where poor recruitment of dynamic stabilizers is evident. JHS, when in its most severe form, presents as a multi-systemic, hereditary connective tissue disorder, which typically results in chronic disability. Recognized genetic connective tissue disorders showing such joint instability include Marfan's Syndrome and Ehler's Danlos Syndrome – these conditions, unfortunately, are likely to present with a host of other complications (including vascular problems, paraesthesias, chronic fatigue, faintness, malaise and even flu-like symptoms) (Simmonds and Keer, 2007).

The more general form of the condition is most prevalent in females. It presents with laxity and increased fragility of connective tissue; patients are also likely to demonstrate reduced proprioceptive functioning and altered (delayed) neuromuscular reflexes. JHS predisposes individuals to damage and injury because of the instability, altered biomechanics and proprioceptive deficits. Characteristic musculoskeletal features also include clicking and clunking, subluxations and dislocations. Such characteristics are normally observed during early development (Simmonds and Keer, 2007). For assessment, the original nine-point 'Beighton Score' (Beighton *et al.*, 1973) has more recently been incorporated into a more comprehensive and validated set of criteria termed the 'Brighton Criteria' (Grahame, 2000; Hakim and Grahame, 2003). The Beighton Score is a simple nine-point screening protocol. It incorporates assessing the patient's ability to: place their hands flat on the floor from standing without bending their knees; extend their elbows (left and right) beyond 10° hyperextension; extend their knees (left and right) beyond 10° of hyperextension; passively approximate their thumbs (left and right) to their forearm; and passively extend their fifth fingers (left and right) to 90°. The 'Brighton Criteria' (Grahame, 2000), categorizes patients with JHS when they are symptomatic and when they present with 'major criteria' (i.e. 4/9 or greater on the Beighton Score; and/or arthralgia for longer than three months in four or more joints); or where in addition to one of the 'major criteria' they also present with two or more 'minor criteria' (including: dislocation or subluxation in more than one joint, or in one joint on more than one occasion; soft tissue rheumatism with three or more lesions (such as epicondylalgia, tenosynovitis or bursitis); or an abnormal skin presentation (such as striae, hypermobility, thin skin or scarring).

JHS is a condition that may be under-recognized and poorly managed. Sports therapists should take care during any objective assessment, manual therapy, soft tissue therapy or exercise prescription so as not to exacerbate the condition or increase instability. While athletes during routine screening sessions should be easily identified with this condition, many patients attending for clinical assessment may be more likely to present with JHS simply as a comorbidity (i.e. as a background to their main complaint) rather than as the primary reason for attendance. Hence, the sports therapist should utilize strategies to improve stability as well as focus on

supporting the management of the primary complaint. General recommendations include improving dynamic stability, proprioception, joint positional sense and muscular endurance (particularly in the core or spinal region and around all vulnerable joints).

Age

Greater age was identified as a risk factor that may contribute to running injuries in a systematic review by van Gent *et al.* (2007), but in contrast they also identified studies that suggest increasing age is in fact a protective factor. Runners with more than ten years of running experience have been shown to have an increased risk of Achilles tendinopathy (Knobloch *et al.*, 2008).

Body size and composition

Body mass index (BMI) is a simple (but limited) measure for identifying where individuals may be overweight – the individual's weight in kilograms is divided by their height in metres squared. Because factors such as individual fitness, muscle mass and ethnic origin are not taken into account with BMI, it is only a proxy indicator of obesity (NOO, 2009). However, a higher BMI has been shown to increase the risk of ankle sprains in high-school athletes (McHugh *et al.*, 2006) and elite athletes (Pefanis *et al.*, 2009). A higher BMI has also been shown to increase the risk of lower extremity injury in male recreational runners (Buist *et al.*, 2010). Heavier and shorter football players are also at a greater risk of thigh muscle injuries (Fousekis *et al.*, 2010); while taller and heavier military recruits, and those with a higher BMI, are at greater risk of ankle sprain (Waterman *et al.*, 2010).

Physiological issues

Cardiorespiratory endurance has been identified as a risk factor for ankle sprains in males (Willems *et al.*, 2006) and male and female military recruits (Waterman *et al.*, 2010). Genetic risk factors for musculoskeletal injuries have been found for the Achilles tendon, the tendons of the rotator cuff muscles and the anterior and posterior cruciate ligaments (Collins and Raleigh, 2009). Reduced dynamic knee stability caused by metabolic fatigue may increase the risk of knee injuries in women by reducing knee flexion angles in single-leg jump landings (Ortiz *et al.*, 2010), although similar results have been shown in both males and females (Brazen *et al.*, 2010).

An increase in non-contact ACL injuries during the ovulatory and pre-ovulatory phases of the menstrual cycle in female athletes has been shown (Adachi *et al.*, 2008; Hewett *et al.*, 2007). Oligomenorrhea, amenorrhea and low bone mineral density have been identified as injury risk factors in female high-school athletes (Rauh *et al.*, 2010).

Neuromuscular fatigue has been identified as a significant factor in functional muscle disorders such as EAMC. Fatigue also contributes to impaired performance and therefore predisposes to all manner of traumatic injuries.

Previous injury

Previous injury is widely identified as a significant risk factor for sustaining a subsequent injury. For example, a previous history of ankle sprain is a significant risk factor for re-injury (McHugh *et al.*, 2006); and previous history of musculotendinous groin injury is a risk factor for new groin

injuries (Engebretsen *et al.*, 2010). A previous injury to the back has also been shown to be a risk factor for back pain in tennis players (Hjelm *et al.*, 2010). It is important to recognize that all significant musculoskeletal injuries undergo a repair process which involves replacement of original tissue with scar tissue (predominantly type 1 collagen), which does not have the same functional properties.

Impaired joint position sense

Athletes with functional ankle instability have been shown to have poor ankle joint positional sense and are more likely to exhibit a more inverted ankle joint position before, at and after heel strike (Delahunt *et al.*, 2006).

Sports-specific technique errors

Brukner and Khan (2009) identify a number of technique faults which may increase the athlete's risk of injury, such as poor lateral pelvic control contributing to iliotibial band syndrome, or too wide a grip in the bench press, leading to pectoralis major tendinopathy.

Psychological issues

A number of psychological factors may contribute to the incidence of injury in sport; these can include anxiety, fatigue, depression, excitation and fear, and can result in abnormal behaviours and impaired performance. It is important to evaluate the risk or threat, personality type and which coping strategies may be applicable (Whiting and Zernicke, 1998).

Nutritional issues

Nutritional status has a significant influence on the risk of injury while training and competing. A lack of protein will delay recovery and rehabilitation (Holmes, 2003), while nutritional deficiencies such as a lack of carbohydrates can cause fatigue. Disordered eating has been shown to be associated with increased risk of musculoskeletal injury in female athletes (Rauh *et al.*, 2010) and female athletes with disordered eating are twice as likely to sustain an injury (Thein-Nissenbaum *et al.*, 2011). Poor diet and hydration are obvious factors which can contribute to short-term poor performance. Persistently disordered eating (such as with anorexia nervosa or bulimia nervosa) and the associated malnutrition can lead to a range of systemic issues including osteoporosis, anaemia, hypotension, irregular cardiac rhythm, renal disease and neural and mental disorders.

Extrinsic risk factors

Extrinsic risk factors are external influences which may contribute to injury. These may be the athletes' immediate environment, such as the surface or terrain, the choice of equipment used or training errors. A simple risk assessment, regular checks of equipment and frequent monitoring and evaluation of the athlete's training programme can all help to reduce the risk of injury as a result of an extrinsic risk factor.

Equipment

Equipment can aid in the prevention of injuries but may also contribute to an athlete sustaining an injury. In the majority of cases protective equipment serves its purpose and protects the athlete from injury. It has been suggested that in a small minority of cases protective equipment such as the helmet and shoulder pads may, in fact, contribute to injury such as heat stress (Whiting and Zernicke, 1998). Sporting equipment has been implicated as an injury risk factor in a number of sports. A common issue is that, although it may be safe and functional, the equipment is often inappropriate for the athlete using it. For example, a tennis player using a tennis racket with too large or too small a grip will be at an increased risk of wrist and elbow injury. Equally, the string tension may be inappropriate or the racquet may be too stiff (Hennig, 2007). In a sport such as cycling there is a higher risk of injury if the bike is not correctly set up for the cyclist. Incorrect bike set-up has been shown to increase the risk of injury in triathletes and multisport athletes (Cipriani *et al.*, 1998). The factors that need to be considered are seat height, seat position, reach and cleat position (Brukner and Khan, 2009). Athletes should be discouraged from borrowing equipment which may be ill-fitting and therefore unsuitable.

Footwear

Although footwear could be classed as part of the athletes' equipment, it is presented separately here because of the diversity of the types of footwear worn by athletes and because footwear choice during the athletes' activities of daily living are equally important to consider. Running shoes are not only worn by runners and triathletes, but by any athlete using running as part of their training programme. More experienced runners are less prone to injury (Fredericson and Wolf, 2005) and it is the less experienced runners who are more inclined to wear ill-fitting or worn-out athletic running shoes. Equally, wearing shoes that are not tied up reduces the support offered by the shoe and increases the loading on the Achilles tendon (Rowson *et al.*, 2010). There is no one shoe that is the perfect running shoe for all athletes due to the diverse array of foot structures and lower limb biomechanics of the running population. Instead, the athlete should look to purchase the most appropriate shoe for their purpose and look to have a simple observational gait analysis and running shoe prescription, which are widely available at specialized running shops. In this way they purchase the most appropriate footwear for their foot structure and gait pattern. In the event that foot structure may be contributing to injury, orthotics or insoles may be required. If so, it is important to seek advice from a specialist sports podiatrist, who can carry out a lower limb biomechanical assessment and prescribe the athlete custom-built orthoses. Off-the-shelf orthotics are not designed individually for the athlete, may not fulfil their required purpose and can contribute to injury by inappropriately modifying the athlete's biomechanics. Athletes with prescribed orthotics may be required to wear them in their general everyday footwear as well as their athletic footwear.

Athletes wearing inappropriate footwear are a common sight: for example, a football player going out for a run while wearing artificial-turf football trainers or a netball player playing netball wearing running shoes. In these cases, they are putting themselves at an increased risk of injury because they are using a shoe for a purpose it was not designed for. Athletes may also wear inappropriate footwear for the type of training they are doing or for the surface they are training on. There is little in the way of evidence-based literature examining the effects of running spikes on the lower limb biomechanics of an athlete. These shoes have spikes over the forefoot and midfoot which strike the ground as the heel normally makes no contact with the ground while running at speed. The 'negative heel' is common in running spikes and observed

when the shoe is placed on a flat surface – the heel is seen to be lower than the forefoot. This may increase eccentric load at the heel and predispose to Achilles tendinopathy and shin pain. Increasing heel lift in running spikes may reduce the risk of sustaining these injuries. Running flats are running shoes worn by athletes in races and at times in training. The use of these types of shoe have been shown to reduce the contact area between the shoe and the ground, increasing the maximum forces experienced by the athlete beneath the entire foot and specifically the lateral forefoot, which is an area of increased stress fracture risk in men (Queen *et al.*, 2010).

Wearing footwear that is too small for the athlete's foot size has been shown to cause injury (Morrison and Schöffl, 2007), while wearing footwear that is too large can cause imbalance, increase the risk of lower extremity injury and potentially induce falls (Paiva de Castro *et al.*, 2010). A study by Teyhen *et al.* (2010) examined the running shoes of 524 US soldiers and showed that poor shoe choices are very common. They showed that 35 per cent of individuals wore incorrectly sized shoes, 57 per cent wore running shoes that were inappropriate for their foot type, 35 per cent had excessively worn shoes and 63 per cent had no idea on the guidelines for running shoe replacement. Excessive shoe wear has also been implicated as an injury risk factor in triathletes (Cipriani *et al.*, 1998) and runners (Taunton *et al.*, 2003).

The type of ski or snowboard boot worn has a significant impact on the risk of injury the individual will be exposed to. Wearing soft boots in either sport puts the individual at a greater risk of injury compared to wearing rigid boots (Pino and Colville, 1989; Sacco *et al.*, 1998). The wearing of rigid boots has reduced the incidence of ankle injuries in skiing; however, in turn, the risk of knee injury has increased dramatically (Pujol *et al.*, 2007).

Surface

Training or competing on surfaces that are too hard or too soft may contribute to an overuse injury, while surfaces that are slippery, sticky, wet or uneven can contribute to acute injury. A number of studies show higher rates of injury sustained on first- and second-generation artificial turf compared to natural grass, while the current data suggest that the injury rate is similar on third-generation and natural grass surfaces. It has also been shown that playing tennis on clay is safer than playing on hard courts or grass (Dragoo and Braun, 2010). It has been suggested that clay causes fewer injuries because it has low frictional resistance and that surfaces with high frictional resistance are a significant risk factor (Nigg and Segesser, 1988).

Terrain

Athletes such as orienteers and hill runners, who predominantly run off-road on rough surfaces, are at an increased risk of injuries such as ankle sprain (Creagh and Reilly, 1998; Fordham *et al.*, 2004). Running on sand has been shown to increase the relative risk of sustaining mid-portion Achilles tendinopathy (Knobloch *et al.*, 2008). Running on a cambered surface has been shown to significantly alter the kinematics of the knee (Gehlsen *et al.*, 1989). This may contribute to lower extremity overuse injury in a similar way as a leg length discrepancy would. Equally, there may be an increased risk of a lateral ankle sprain on the outside (lower) foot as the ankle may be more inclined to invert on heel strike.

Gradient

Excessive uphill or downhill running have been shown to contribute to the risk of injury as the change in gradient significantly alters the biomechanics of the gait cycle. Uphill running has

been shown to significantly increase hip flexion and knee extension torques in the rectus femoris compared to level running (Yokozawa *et al.*, 2007). This is due to the fact that there is greater forward lean of the trunk and less knee extension torque during uphill running. Excessive downhill running has been implicated as a risk factor for sustaining overuse injury (Fredericson and Wolf, 2005), with a –9° gradient causing impact force peaks to increase by 54 per cent and parallel braking force peaks to increase by 73 per cent (Gottschall and Kram, 2005).

Training errors

It has been shown that a significant correlation exists between number of training hours and incidence of injury (Almeida *et al.*, 1999; Fordham *et al.*, 2004; Wolf *et al.*, 2009). Rapid increases in training volume increases the risk of injury (Almeida *et al.*, 1999; Fredericson and Wolf, 2005), while lack of adequate rest has been shown to increase the incidence of overuse injuries. Running excessive distances in the same direction around a running track has been associated with the development of iliotibial band friction syndrome (Fredericson and Wolf, 2005). Once the athlete is running more than 40 miles per week there is a significant increase in the risk of lower extremity overuse injury (Fredericson and Misra, 2007) and runners with high weekly mileage are at risk of recurrent lower extremity stress fracture (Korpelainen *et al.*, 2001). The higher training loads and excessive intensity observed at the start of pre-season training corresponds to higher injury rates (Killen *et al.*, 2010). Running speed is a risk factor for acute injuries such as ankle sprains (Willems *et al.*, 2006). Poor technique while weight training has been shown to increase injury risk in the shoulder (Kolber *et al.*, 2010) and trunk (Quatman *et al.*, 2009).

Inappropriate cross-training can bring about any of the training errors listed above, but also expose the athlete to risk factors such as inappropriate footwear, equipment, surface, terrain or gradient. Cross-training is also a risk where athletes are exhibiting poor technique and are training unsupervised by qualified coaches. A high incidence of injury has been observed as a result of cross-training, with more than one-third of swimming injuries occurring out of the pool during activities such as strength training (Wolf *et al.*, 2009). Poor technique is a risk factor for many sports-specific overuse injuries such as medial epicondylalgia ('golfers elbow') (Bayes and Wadsworth, 2009) and lateral epicondylalgia ('tennis elbow') (Kelley *et al.*, 1994). Faults in sports-specific techniques which are not corrected may contribute to overuse injury, especially when the athlete trains for long periods of time.

Environmental conditions

Environmental conditions have a significant impact on athletes training and competing outdoors as they experience extremes of heat, cold and humidity, all of which may cause injury or illness. Adequate warm-up, cool-down, rest periods, nutrition and the replacement of fluid and salt are widely recommended for athletes training or competing under stressful environmental conditions. Guidelines on the prevention of environmental illness are published by a number of organizations such as the ACSM, the NSCA (National Strength and Conditioning Association) and NATA (National Athletic Training Association). Athletes exposed to cold, wet or windy conditions are at risk of cold injuries such as hypothermia, frostbite, chilblains and immersion ('trench') foot (Cappaert *et al.*, 2008). There are a number of events where athletes are at risk of cold injuries: those taking part in water, such as swimming and sailing; alpine sports; endurance events such as marathons and triathlons and basic military training. Hypothermia has been seen in open water swimming events (Castro *et al.*, 2009) and a higher BMI is associated

with a decreased risk of hypothermia (Brannigan *et al.*, 2009). Extreme cold may cause hypothermia during endurance events and significantly more drop-outs (Agar *et al.*, 2009). It has also been shown that training seasons have an effect on the incidence of Achilles paratendinitis, with significantly more recruits sustaining the condition in winter months (Milgrom *et al.*, 2003). It is thought to be caused by the decreased temperature increasing the viscosity of the lubricant in the paratenon, which increases friction.

In endurance events such as marathon running, 4–15 °C is the most advantageous temperature range, while the risk of drop-outs and the numbers requiring medical attention increase dramatically as the temperature rises above 15.5 °C (Roberts, 2007). Heat stress increases the numbers of drop-outs due to heat stroke and hyponatraemia, while cold stress increases the numbers of drop-outs and when combined with rain increases the numbers of runners seeking medical attention (Roberts, 2007). Marathon performance slows as the temperature rises from 5 to 25 °C in both male and female runners and this affects the slower runners taking part (Ely *et al.*, 2007). As the temperature during the event rises, so does the risk of heat exhaustion and heatstroke in athletes. An athlete is suffering from heatstroke if their core (rectal) temperature is greater than 38 °C and they require immediate cooling until the rectal temperature drops below 38 °C (Armstrong *et al.*, 2007). A number of marathons with starting temperatures of >21 °C have ended with cancellation and mass casualty incident (Roberts, 2010).

Soft tissue repair process

The following section presents an overview of the soft tissue healing process. Generically, Watson (2012) has described four overlapping phases: bleeding, inflammation, proliferation and remodelling. Numerous authors have written extensively on this topic, including good early review articles by Barlow and Willoughby (1992) and Hardy (1989). Anderson *et al.* (2009), Houglum (2010), Knight and Draper (2008), Lieber (2002), Norris (2004) and Prentice (2011) each provide informative book chapters. In order to successfully assess and manage patients with soft tissue injury, sports therapists must understand basic pathophysiological theory, be able to recognize the signs and symptoms in their injured patients/athletes and appreciate the full sequence of events which typically occur during each phase. It is essential to appreciate that the following section describes a generic cascade of events which may typically occur in an average injury involving, for example, an injured ligament or area of skin; it is beyond the scope of this text to explore in detail the subtleties of healing processes involving specific injuries to specific tissues. There are numerous additional healing processes to understand, particularly when presented with injuries involving muscle, tendon, skin, bone, nerve or visceral damage. Readers are therefore directed towards key recent articles for detailed overviews of specific tissue pathophysiology and classification, such as those provided by: Cook and Purdam (2009), Kaeding and Best (2009) and Rees *et al.* (2013) (tendinopathy); Boage *et al.* (2012), Giamberardino *et al.* (2011), Mueller-Wolfahrt *et al.* (2013) and Valle *et al.* (2015) (functional and structural muscle conditions); Enoch and Leaper (2005) and Li *et al.* (2007) (skin healing); Bennell and Kannus (2003) (bone healing); and Butler and Tomberlin (2007) (nerve injuries).

The immediate (bleeding and haemostasis) phase

The immediate phase of a typical traumatic soft tissue injury is characterized first by bleeding and second by haemostasis. At the time of injury, bleeding from ruptured blood vessels in the affected tissues occurs to varying degrees. The amount of bleeding depends primarily on the severity and extent of the injury as well as the inherent vascularity of the involved tissue. The

bleeding phase is normally short-lived (minutes) but may last for a few hours. As haemostasis (the inherent mechanisms involved in the cessation of bleeding) begins, the body is concomitantly initiating an inflammatory response. One of the body's first responses to bleeding is vasoconstriction, which is triggered initially by the release of noradrenaline from mast cells in the connective tissues local to the injury site. Further chemical stimulation for vasoconstriction is provided by serotonin, which is released by platelets. Vasoconstriction (vascular spasm) is the body's attempt to minimize blood loss, but will only last for a few minutes at most. Vasoconstriction also contributes to an additional aspect of the initial response to injury; a process known as secondary ischaemic (hypoxic) death, where local tissue cells on the periphery of the injury site – initially unaffected by the inciting event – begin to die due to an insufficient blood supply. The resulting cell necrosis is caused by a lack of oxygen, and hence hypoxia occurs (Knight, 1995). Digestive enzymes from the gathering phagocytic leucocytes can also spill over from the injury site and contribute further to the death of surrounding cells. Mast cells affected by secondary death release a number of chemical mediators, including histamine, which are essential to the ensuing inflammatory response. At the injury site, further mechanisms contribute to the coagulation cascade and resulting 'platelet plug'. Mediators such as the glycoprotein 'von Willebrand factor' (vWF) are released from the endothelial walls of affected blood vessels and contribute to the activation of circulating platelets (thrombocytes). The platelets proliferate, become sticky, and begin to adhere to the blood vessel walls and to each other (platelet aggregation). This process is further facilitated by the release of a prothrombotic lipid – thromboxane – from the platelets themselves. The process of platelet adherence is termed 'pavementing'. At the same time as platelet plug formation, the process of fibrinolysis occurs. There follows an ongoing series of chemical and enzymatic responses, particularly those involving plasma protein-based clotting factors. Platelet activation also stimulates the Hageman factor XII plasma enzyme to contribute to the coagulation cascade. Prothrombin is then converted to thrombin. Thrombin converts the soluble plasma protein fibrinogen into a more insoluble material called fibrin, which form into strands (Li *et al.*, 2007). The coagulation process results in the formation of a lattice-patterned fibrin mesh, which acts somewhat like a cement and binds together with the platelet plug and all other debris at the site (dead cells and metabolites). The plug and fibrin mesh form a temporary patch, and if bleeding is successfully stemmed, haemostasis is complete. During haemostasis, a careful equilibrium between coagulation and anti-coagulation (blood fluidity – mediated by such chemicals as heparin [also released by mast cells]) needs to be reached so as to achieve the desired state of haemostasis, while also providing necessary blood supply.

The acute inflammation phase

Acute inflammation is the vascularized tissues' initial reaction to any irritant or pathogenic agent or traumatic injury. Inflammation will result from any sufficiently damaging burn or chemical injury, infection, autoimmune disorder or mechanical trauma. In terms of tissue healing, this phase is sometimes referred to as the 'lag phase' because of the inherent weakness and insufficiency of the injury site. Watson (2012) presents a simplified overview of a recognized generic sequence of chemically mediated cellular and vascular responses to soft tissue damage which forms the normal, initial and essential inflammatory phase of healing. Vascular events essentially aim to provide a supply of specialized cellular effectors to the injury site, and the cellular events attempt to contend with pathogenic agents and clear (debride) the affected site of debris. Typically beginning within an hour of injury, peaking in magnitude of activity for two or three days, before gradually fading over two or three weeks, the acute inflammatory

Table 1.4 Five generic and cardinal signs of inflammation

Swelling (tumor)	*Redness (rubor)*	*Heat (calor)*	*Pain (dolor)*	*Loss of function (functio laesa)*
Due to damaged local vessels and the ensuing haemodynamic response (vasodilation, vasopermeability) Leaked plasma proteins increase extracellular osmotic pressure and draw more fluid from local capillaries Extravasated fluids and cells accumulate in the interstitial spaces. Lymphatic obstruction is a contributing factor Swelling is also known as oedema	Due to the haemodynamic response (increased blood flow) at the affected site, near to the skin's surface Reddening of skin is known as erythema Increased blood flow through a region is termed hyperaemia	Due to the haemodynamic response (increased blood flow) at the affected site, near to the skin's surface	Due to mechanical, thermal and chemical stimulation of nociceptors. With any pain experience, there is also a central (nervous system) component	Due to damaged structures, loss of tissue integrity (and reduced tensile strength), pain, swelling and/or muscular spasm

phase offers a defence against foreign substances, a disposal system for dead and dying (necrotic) tissue cells, a warning sign of damage and injury (pain), and attempts to immobilize and compartmentalize the affected area and set up a chain of events for the proliferative repair phase. The five classic, or cardinal, signs of inflammation, which are non-specific to the cause of damage, are: swelling (*tumor*); redness (*rubor*); heat (*calor*); pain (*dolor*); and loss of function (*functio laesa*) (Punchard *et al.*, 2004). The two essential chemically mediated haemodynamic events occurring as the inflammatory response are vasodilation and vasopermeability (Houglum, 2010). The process begins soon after the body has begun contending with the initial trauma and instigating the process of haemostasis. Vasodilation, which is autonomic relaxation and widening of blood vessel walls (arterioles and previously dormant capillaries) – leads to a decrease in vascular resistance and hence an increase in local blood flow (hyperaemia). Fluid leakage from the affected vessels into the interstitial spaces occurs as they become more permeable. Diapedesis is the term to describe the outward passage of blood cells (principally leucocytes), along with plasma proteins and other chemicals, through the vessel walls. The extravasated fluid is referred to as exudate. The increasing vascular hydrostatic pressure forces the fluid and particulate contents from the capillary outwards into the tissues. Gradually, local blood flow slows and a state of haemoconcentration occurs. Importantly, the exudate becomes more gel-like as it binds with hyaluronic acid (released from mast cells), and gradually, due to reducing stimuli, the haemodynamic response fades. The inflammatory exudate's gel-like consistency (sometimes described as the 'inflammatory soup') helps to contain the injury site and reduces the potential for inflammatory reactions occurring in surrounding tissues (Watson, 2012).

The hallmark characteristics of inflammation are the typically visibly obvious swelling (oedema), redness (erythema) and surface heat. Pain may occur due to stimulation of polymodal

nociceptors (mainly involving the fast-conducting A-delta nerve fibres) during the initial trauma, and the resulting acute inflammatory response (with continuing stimulation of sensory nerves – involving slow-conducting unmyelinated C fibres – from the developing oedema and possible presence of muscular spasm); in addition there is chemical stimulation of nociceptors from locally released substances including prostaglandins, histamine and bradykinin. Nociceptors themselves also release the chemical neuropeptide 'substance P', which as well as stimulating further histamine release from local mast cells (to maintain the haemodynamic response), also contributes to the ongoing perpetuation of nociceptive messaging which is transmitted along afferent pathways towards the dorsal horn of the spinal cord; from here pain is modulated in numerous ways (via ascending and descending messaging in the central nervous system).

Substances such as histamine, leucotaxin, bradykinin, prostaglandins and the 'complement cascade' (numerous plasma-derived proteins) are among the main chemical mediators for the haemodynamic response. Leucocytes become chemically attracted to the site, and begin to marginate along blood vessel walls. They are able to pass through the permeable walls of the blood vessels (a process known as diapedesis) and via chemotaxis (chemical attraction) are drawn to the site of damage. The first leucocytes to gather in numbers are neutrophils (also known as polymorphonuclear leucocytes – PMNs). Neutrophils are chiefly responsible for early and aggressive phagocytosis, but may only provide around 7–10 hours of activity, and do not reproduce in the process. Their main phagocytic response is geared towards pathogenic agents, rather than larger cellular debris. As they die, neutrophils release chemical mediators which contribute to the chemotaxic attraction of larger, longer-lasting phagocytic leucocytes (monocytes) to the site (Knight and Draper, 2008). Other chemical mediators of phagocytosis include the complement cascade and a number of growth factors, which are the key substances able to chemically stimulate cell proliferation, differentiation and growth. The monocytes quickly differentiate into macrophages at the site, and continue to combat any ongoing infection and perform the main 'clean up' and debridement of the affected site. The basic process of phagocytosis is to engulf, digest and remove pathogens and tissue debris.

Many of the events occurring within the acute inflammatory phase occur simultaneously and will continuously interact. The acute stage response gradually fades and will normally last until damaged tissue has been contained, tissue debris removed, and new capillary networks have begun to be laid down. Towards the end of this phase, as the initial stimulations subside (chemical and mechanical), the hallmark haemodynamic and cellular responses also reduce.

Practitioner Tip Box 1.1

Chemical mediators are the plasma-derived or cell-derived chemical messengers that act directly on blood vessels, inflammatory cells or other cells. They are involved in propagating the complex cascade of events during all phases of tissue healing. During the bleeding and inflammatory phases they are variously released, mainly from the following:

- Blood plasma
- Endothelial cells (in blood vessel walls)
- Platelets (thrombocytes)
- White blood cells (WBC or leucocytes) including basophils, neutrophils, monocytes and
- macrophages
- Mast cells (in connective tissue)
- Damaged tissue cells

Practitioner Tip Box 1.2

Chemical mediators, which are involved in the complex cascade of events during the bleeding and inflammatory phases of tissue healing, include the following:

- Noradrenaline
- Histamine
- von Willebrand factor
- Thromboxane
- Prothrombin
- Thrombin
- Hageman factor VII
- Serotonin
- Prostaglandins
- Substance P
- Heparin
- Bradykinin
- Leucotaxin
- Nitric oxide
- Complement cascade
- Hyaluronic acid
- Growth factors
- Lactic acid

Table 1.5 The acute inflammatory phase

Duration	Within 1 hour to 2–3 weeks (gradual fade)
Signs and symptoms	Swelling, redness, heat, pain and impaired function
Key features	Chemically mediated vascular and cellular response
	Haemodynamic response:
	Vasodilation
	Vasopermeability
	Hyperaemia
	Haemoconcentration
	Increased hydrostatic pressure
	Production of exudate
	Cellular response:
	Chemotaxis
	Leucocyte margination and diapedesis
	Phagocytosis
	Chemical and mechanical/thermal irritation of nociceptors

Macrophages, which are activated later in the inflammatory phase than other phagocytes, work anaerobically, and as such release lactic acid as a metabolic by-product, as well as vascular endothelial growth factors (VEGF); these act as key stimulants for the next stage of healing – proliferation.

The well-documented 'pain–spasm–pain cycle' is a common characteristic feature of the sub-acute phase, where chemical stimulation of the free nerve endings contributes to the local pain experience, which then triggers a protective mechanism causing involuntary muscle spasm during static postures, leading to further pain. Concurrently, there may also be a reduction of muscle activity during movement (Roland, 1986). The fundamental combination of tissue structural damage, loss of tensile strength, localized swelling, protective muscular spasm and resulting mechanical and chemical perpetuation of pain leads to impaired functioning of the affected tissues in the injured individual. The severity and degree of impairment depends upon numerous factors.

The proliferative repair phase

As the inflammatory phase fades, so begins the proliferation, or early repair, phase. Typically lasting for approximately 3–4 weeks, it is characterized by the establishment of a new vascular network (neovascularization) in order to facilitate the process of delivering and supporting the cells (fibroblasts) involved in the repair of damaged tissue. The actual type of repair depends upon the type of tissue which is predominantly damaged. Knight and Draper (2008) explain that there are two basic types of initial repair process: reconstitution and replacement. Reconstitution may be 'perfect' or 'imperfect'. Perfect reconstitution is where the injured cells are replaced by identical cells – but this can only occur in those tissues which have a normally high rate of turnover and replacement – i.e. tissue cells which have a high regeneration capacity. Such cells are termed 'labile' and are found in such tissues as those forming the epidermis of the skin and in the linings of the respiratory, gastrointestinal and genitourinary tracts. Although dependent on severity of damage and individual capacity to heal, the resultant repair from perfect reconstitution is likely to be perfect! Imperfect reconstitution occurs with tissues formed from 'stabile' cells – such as those forming the liver, pancreas and kidneys. Imperfect reconstitution is where damaged cells are replaced by a combination of identical cells and connective tissue, which will be predominantly type-III collagen at this stage. Imperfect reconstitution can occur in response to skeletal muscle fibre damage, due to the presence of satellite cells, which contribute to a micro-regeneration of tissue, but regeneration capacity is still limited (Barlow and Willoughby, 1992). Replacement is the repair process for 'permanent' cells – cells which have limited or no regenerative capacity, such as those forming nerve tissue, cardiac muscle and ligamentous tissue. This type of repair replaces original cells with 'simpler' connective tissue cells. The replacement repair process also occurs whenever the soft tissue damage is sufficiently extensive to disrupt the basic cellular framework. Scar tissue (collagen) is laid down (synthesized) to replace the original tissue – a process referred to as 'collagenization'. The majority of soft tissue injury sites heal by imperfect reconstitution, with a repair (replacement) process rather than a complete regeneration (reconstitution). Garrett (1990) identified that repair with new collagen does not begin until around five days following injury; Norris (2004) also describes the 'lag phase' as the period of time from immediate injury to the beginning of collagen synthesis (i.e. the bleeding and inflammatory phases). During this period, pain and oedema are present and tensile strength is reduced.

Angiogenesis – the formation of a new vascular network – is an essential factor during this phase of healing. Stimulated by activating growth factors (such as VEGF, angiogenesis factor

[AF] and platelet-derived growth factor [PDGF]), fresh capillary buds (branches) begin to appear and are formed from the dividing endothelial cells from existing vessels in the injured area (Li *et al.*, 2007). As they develop, the new capillary buds migrate towards one another, eventually forming a series of capillary arches. Once fully formed, the capillary arches provide a new and effective and localized vascular supply throughout the damaged tissue. The network of capillary arches is referred to as the capillary arcade (Knight and Draper, 2008), and the neovascularization facilitates delivery of fibroblasts, myofibroblasts, oxygen and nutrients and also provides a drainage system for removal of metabolites and other tissue debris, all of which are essential for effective repair.

Table 1.6 The proliferative repair phase

Duration	3–4 days to 3–4 weeks
Signs and symptoms	Pain and impaired function (dependent on severity); localized swelling and reddening with superficial injury
Key features	Chemical mediation and chemotaxis via cytokines and growth factors Angiogenesis: Endothelial proliferation Neovascularization Capillary budding, arching and arcade formation Repair via reconstitution (regeneration) or replacement (scar tissue) Fibroplasia: Fibroblast and myofibroblast proliferation Granulation tissue Collagenization – type III collagen Degranulation Neoinnervation Devascularization Reduced tensile strength in affected tissue

Table 1.7 Main growth factors released in response to injury

Growth factor	*Abbreviation*	*Function*
Platelet-derived growth factor	PDGF	Early growth factor in healing; stimulates fibroblasts and angiogenesis; promotes remodelling; stimulates production of other growth factors
Transforming growth factor	TGF	Promotes ECM and cell replication
Vascular endothelial growth factor	VEGF	Stimulates angiogenesis
Insulin-like growth factor 1 (also known as mechano-growth factor)	IGF1	Promotes neurogenesis; stimulates myoblast proliferation
Fibroblast growth factor 2	FGF2	Promotes ECM; stimulates fibroblasts and endothelial cells; enhances number and diameter of regenerating muscle fibres
Tumor necrosis factor	TNF	Stimulates inflammatory and immune responses
Nerve growth factor	NGF	Stimulates neurogenesis

Source: adapted from Mangine *et al.*, 2012.

The stimulation for increased fibroblastic activity (fibroplasia) is also provided predominantly by cytokines and growth factors (such as Transforming Growth Factor [TGF], Tumor Necrosis Factor [TNF], Insulin-like Growth Factor [IGF] (also known as Mechanogrowth Factor [MGF]), Nerve Growth Factor [NGF] and basic Fibroblast Growth Factor [bFGF]). Growth factors are released from a range of cells, including platelets, mast cells and macrophages in the final stages of inflammation. Further release of growth factors occurs during the early proliferative phase from the extracellular matrix (ECM) of the injury site and also from the gathering fibroblasts themselves (Boage *et al.*, 2012). As fibroblasts proliferate, granulation tissue begins to form within the existing ECM and platelet plug/fibrin mesh. The tissue being produced is partly fibrous (i.e. collagen, elastin and reticulin) and partly non-fibrous (i.e. fluid and protein – water, mineral salts and glycosaminoglycans or 'GAGs'). The developing structure of the matrix strengthens and supports the tissues and provides a means for diffusion of tissue fluid and nutrients between capillaries and cells (Placzek and Boyce, 2006). Fibroblasts themselves produce collagen, proteoglycans and elastin, which are all essential components of scar tissue formation and maturation (Houglum, 2010). Fibroblasts lay down collagen (principally type III at this stage), which forms a far more significant seal over the injury site than that which was provided by the early fibrin patch and exudate gel during the initial stages of injury. Fibronectin, an adhesive glycoprotein, is plentiful at the site (within the gel) and improves the adhesion of fibroblasts to the fibrin patch. Hyaluronic acid (released from mast cells during inflammation) also assists in facilitating fibroblast and myofibroblast migration to the site due to its fluid-drawing properties. The collagen is laid down in a haphazard manner, essentially creating a quite random matrix. Myofibroblasts also begin to proliferate at the margins of the injury. These are then oriented towards the centre of the damaged tissue and have an actin filament content and resultant contractile properties (similar to that of smooth muscle cells), which means they are able to pull wound edges together. Type-III collagen is fundamentally thin, weak, soluble and lacks robust cross-linkages. It is vulnerable to re-injury, but tensile strength begins to increase after the first week. As the proliferation phase continues, the original fibrin patch is reabsorbed (in a process known as degranulation). In addition to its neovascularization, the replacement tissue also begins to develop a network of pressure- and tension-sensitive nerve cells (a process of localized neurogenesis or neo-innervation), which can lead to additional noxious stimulation when the area is externally compressed or stretched (Houglum, 2010). The area is also likely to remain visibly swollen and reddened, particularly if it is superficial, due to the increased water content, vascularity and significant amount of immature collagen at the site. As the repair tissue matures, typically by day 12, type-III collagen is gradually beginning to be replaced by type-I. A process of devascularization also begins, which involves obliteration of the newly formed capillary network (Watson, 2012).

The remodelling and maturation phase

The remodelling phase begins as the proliferation phase is fading and can last for a year or more. The number of capillaries, fibroblasts, myofibroblasts, macrophages and more all gradually diminish and the activity in the region returns to a near pre-injury state. During remodelling, the gradual organization of collagen takes place – this is termed 'collagen transition'. Type-III collagen is replaced by the stronger, more insoluble, more permanent, type-I collagen – with different tissues (muscle; tendon; ligament; fascia; skin) having different functional and structural requirements. Cross-linkages throughout the fibres occur more readily as fluid content in the area is reduced. Wound contraction via myofibroblast activity continues (Betz *et al.*, 1992) and is generally beneficial to the resulting quality of tissue, unless joint or tissue mobility is

Table 1.8 The remodelling and maturation phase

Duration	2–3 weeks to 1 year or more
Signs and symptoms	Improving function (dependent on severity and effectiveness of healing process)
Key features	Collagen transition: Type-III collagen gradually reinforced and replaced by type-I collagen Reduction in water content and numbers of capillaries, fibroblasts, myofibroblasts and macrophages Increasing scar tissue density, collagen cross-linkages and tensile stress

compromised. As maturation takes place, the collagen fibres model in response to the lines of mechanical stress applied to them – as in the concept of Davis' law of soft tissue and the long-established principle of 'specific adaptation to imposed demands' (SAID) (Stearns, 1940). Optimal scar tissue formation occurs when a physiological balance is maintained between its ongoing synthesis (production) and lysis (breakdown and reabsorption) during the remodelling phase (Hardy, 1989).

Chronic inflammation

In certain cases, the normal process of soft tissue healing does not take place. Chronic inflammation occurs when the healing does not progress successfully into the proliferation stage. Essentially a breakdown in the normal process of acute inflammation, chronic inflammation may persist for months or more and can result in an excess of scar tissue formation and associated functional deficits (Holey and Cook, 2011). Various reasons exist for this occurrence, including: presence of foreign bodies at the injury site; bacterial infection; invasion of microorganisms which are able to survive in large phagocytes; antigen–antibody adverse immune reactions; and perhaps most commonly, ongoing mechanical irritation. Whereas acute inflammation has a short onset and a relatively short duration, by definition, chronic inflammation typically has a long onset and a long duration. However, Watson (2008) explains that there are two forms of chronic inflammation: chronic supervention on an original acute reaction; or chronic inflammation in the absence of acute reaction ('ab initio').

Although the characteristic features of chronic inflammation differ according to type, there are likely to be low concentrations of normal chemical mediators, and neutrophils are replaced with prolonged presence and activity of larger phagocytes at the injury site, which produce harmful cytotoxins. There is continued production of exudate, but this does gradually reduce, albeit later than normal. Concentrations of essential growth factors at the site are reduced. Debridement (phagocytosis) is likely to be incomplete and a low-grade inflammatory response persists. Gathering fibroblasts produce a 'granuloma', or mass of weak type-III collagen, which begins to surround the injury site – this is the start of 'fibrosis' rather than the normal process of fibroplasia. There will be simultaneous tissue destruction, inflammation and attempted healing, and different tissues will respond differently to the process. Prentice (2011) states that there is no recognized timeframe in which acute inflammation transitions to chronic inflammation.

Factors affecting healing

A large number of factors can affect the overall quality of healing, both positively and negatively; these may be local or systemic. During the early phase, the quality of healing response will be

directly influenced by the very nature (tissue involvement) and severity of injury, its initial management and the degree of post-injury mechanical aggravation (as opposed to appropriately graded therapeutic mechanical stress). Early presentations such as excessive oedema or bleeding, haematoma formation, or a large degree of tissue separation or muscle spasm can all impede the normal process. Enoch and Leaper (2005) explain how, in particular, growth factors and cytokines play an essential role in healing processes across each of the phases. Alterations in any of these components or disruption in their functional role can impair the healing response. Additional important factors to consider which have influence on healing (positively or negatively) include: the age of the individual; general health and presence of comorbidity; whether operative intervention was undertaken (repair or reconstruction); infection; immunosuppression; systemic vascular disease; obesity; diabetes; nutritional deficiency (malnutrition) (especially with regard to vitamin A and C, minerals zinc and copper and protein); use of supplementation (there is gathering evidence to warrant consideration of supplements such as arginine, leucine, HMB [hydoxy-beta methylbutyrate] and glutamine) (Williams *et al.*, 2002); and use of medications (especially regarding non-steroidal anti-inflammatories [NSAIDs], corticosteroids and anti-coagulants). Hypertrophic (excessive collagen deposited at the injury site) or keloid (excessive collagen deposited in the surrounding tissue) scarring can also lead to a less desirable local outcome. Placzek and Boyce (2006) explain that glucosamine, found within type-II collagen, has importance in the process of soft tissue repair, being a precursor for such compounds as chondroitin sulphate and hyaluronic acid. Glucosamine also increases proteoglycan production. However, evidence is limited with regard to its effects when taken as a dietary supplement. Bloch (2013) advocates supplemental vitamin C and E as antioxidants to combat the damaging free radicals produced by leukocytes during the early inflammatory phase.

Obviously, the sports therapist has a wide range of specialist interventions available to support the injured athlete back towards optimal function, and any management provided should be based on appropriate assessment and will be carefully and individually tailored to suit each particular phase of healing. Beyond this, an important issue surrounding therapeutic care is that of patient adherence, or non-adherence, to the therapeutic intervention and advice (Magee *et al.*, 2008). Even if the patient or athlete is adhering to the recommended advice, there is still potential for re-injury. The resulting tensile strength of tissue following minor injury is an indicator of outcome. When overviewing average healing timeframes and outcomes in different tissues, muscle tissue may have near normal tensile strength 7–11 days following minor damage, with a 90 per cent contraction ability following the remodelling phase (Houglum, 2010). Tendon strength is likely to be 85–95 per cent normal, and ligaments likely to be near to normal within a year. By comparison, articular cartilage is likely to be near normal within six months, and bone within 8–12 weeks following fracture (depending on type). It is important to recognize that although a functionally successful outcome is highly likely for an uncomplicated soft tissue injury following sports therapy intervention, it cannot be guaranteed!

This chapter has presented an introductory overview of topics fundamental to the understanding of sports injuries: classifications, aetiology and repair processes. It has been beyond the scope of this text to explore in greater detail specific tissue pathophysiology (i.e. the processes involved in the healing and repair of muscle, tendon, ligament, bone, skin, nerve and viscera) and also the relatively complex topic of pain. Obviously, it is an essential requirement that all practitioners involved in the prevention, assessment and management of sports injuries undertake appropriate, evidence-informed and continued professional development. The field of sports and exercise medicine is overtly and continuously re-evaluating its evidence-base, and cutting-edge research

enables new light to be shone on all aspects of this specialized area, hence all aspiring sports therapists must be recommended to consolidate their foundational knowledge in each of the areas discussed, and to keep abreast of the latest and most current information.

References

Adachi, N., Nawata, K., Maeta, M. and Kurozawa, Y. (2008) Relationship of the menstrual cycle phase to anterior cruciate ligament injuries in teenaged female athletes. *Archives of Orthopaedic and Trauma Surgery*. 128 (5): 473–478

Agar, C., Pickard, L. and Bhangu, A. (2009) The tough guy pre-hospital experience: Patterns of injury at a major UK endurance event. *Emergency Medicine Journal*. 26 (11): 826–830

Almeida, S.A., Williams, K.M., Shaffer, R.A. and Brodine, S.K. (1999) Epidemiological patterns of musculoskeletal injuries and physical training. *Medicine and Science in Sports and Exercise*. 31 (8): 1176–1182

Anderson, M., Parr, G. and Hall, S (2009) *Foundations of athletic training: Prevention, assessment and management*, 4th edition. Lippincott, Williams and Wilkins. Philadelphia, PA

Armstrong, L.E., Casa, D.J., Millard-Stafford, M., Moran, D.S., Pyne, S.W., Roberts, W.O. and American College of Sports Medicine (2007) Exertional heat illness during training and competition. *Medicine and Science in Sports and Exercise*. 39 (3): 556–572

Back, D. and Smethurst, M. (2004) The female triad. *Sports Injury Bulletin*. 43: 9–10

Barlow, Y. and Willoughby, J. (1992) Pathophysiology of soft tissue repair. *British Medical Bulletin*. 48 (3): 698–711

Baumhauer, J.F., Alosa, D.M., Renström, P., Trevino, S. and Beynnon, B. (1995) A prospective-study of ankle injury risk-factors. *American Journal of Sports Medicine*. 23 (5): 564–570

Bayes, M.C. and Wadsworth, L.T. (2009) Upper extremity injuries in golf. *Physician and Sports Medicine*. 37 (1): 92–96

Beighton, P.H., Solomon, L. and Soskolne, C.L. (1973) Articular mobility in an African population. *Annals of the Rheumatic Diseases*. 32: 413–417

Bennell, K. and Alleyne, J. (2009) Women and activity-related issues across the lifespan. In Brukner, P. and Khan, K. *Clinical sports medicine*, revised 3rd edition. McGraw-Hill. NSW, Australia

Bennell, K. and Kannus, P. (2003) Bone. In Kolt, G. and Snyder-Mackler, L. (eds) *Physical therapies in sport and exercise*. Churchill Livingstone. Philadelphia, PA

Betz, P., Norlich, A., Wilske, J., Tubel, J., Penning, R. and Eisenmenger, W. (1992) Time-dependent appearance of myofibroblasts in granulation tissue of human skin wounds. *International Journal of Legal Medicine*. 150: 99–103

Bloch, W. (2013) Muscle healing: Physiology and adverse factors. In Mueller-Wohlfahrt, H.W., Ueblacker, P., Haensel, L. and Garrett, W.E. (eds) *Muscle injuries in sports*. Thieme. Stuttgart, Germany

Boage, L., Van Den Steen, E., Rimbaut, S., Phillips, N., Witvrouw, E., Almqvist, K.F., Vanderstraeten, G. and Vanden Bossche, L.C. (2012) Treatment of skeletal muscle injury: A review. *International Scholarly Research Network (ISRN) Orthopedics*. 2012: n.p.

Brannigan, D., Rogers, I.R., Jacobs, I., Montgomery, A., Williams, A. and Khangure, N. (2009) Hypothermia is a significant medical risk of mass participation long-distance open water swimming. *Wilderness and Environmental Medicine*. 20 (1): 14–18

Brazen, D.M., Todd, M.K., Ambegaonkar, J.P., Wunderlich, R. and Peterson, C. (2010) The effect of fatigue on landing biomechanics in single-leg drop landings. *Clinical Journal of Sports Medicine* 20 (4): 286–292

Brukner, P. and Khan, K. (2009) *Clinical sports medicine*, revised 3rd edition. McGraw-Hill. NSW, Australia

Buist, I., Bredeweg, S.W., Lemmink, K.A.P.M., van Mechelen, W. and Diercks, R.L. (2010) Predictors of running-related injuries in novice runners enrolled in a systematic training program: A prospective cohort study. *American Journal of Sports Medicine*. 38 (2): 273–280

Butler, D.S. and Tomberlin, J.P. (2007) Peripheral nerves: Structure, function and physiology. In Magee, D.J., Zachazewski, W.S. and Quillen, W.S. (eds) *Scientific foundations and principles of practice in musculoskeletal rehabilitation*. Saunders, Elsevier. St Louis, MO

Cain, E.L., Dugas, J.R., Wolf, R.S. and Andrews, J.R. (2003) Elbow injuries in throwing athletes: A current concepts review. *American Journal of Sports Medicine*. 31 (4): 621–635

Caine, E.L., DiFiori, J. and Maffulli, N. (2006) Physeal injuries in children's and youth sports: Reasons for concern? *British Journal of Sports Medicine*. 40: 749–760

Cappaert, T.A., Stone, J.A., Castellani, J.W., Krause, B.A., Smith, D. and Stephens, B.A. (2008) National athletic trainers' association position statement: Environmental cold injuries. *Journal of Athletic Training*. 43 (6): 640–658

Castro, R.R.T., Mendes, F. and Nobrega, A.C.L. (2009) Risk of hypothermia in a new Olympic event: the 10-km marathon swim. *Clinics*. 64 (4): 351–356

Chappell, J.D., Herman, D.C., Knight, B.S., Kirkendall, D.T., Garrett, W.E. and Yu, B. (2005) Effect of fatigue on knee kinetics and kinematics in stop-jump tasks. *American Journal of Sports Medicine*. 33 (7): 1022–1029

Cipriani, D.J., Swartz, J.D. and Hodgson, C.M. (1998) Triathlon and the multisport athlete. *Journal of Orthopaedic and Sports Physical Therapy*. 27 (1): 42–50

Clark, M. (2001) Muscle energy techniques in rehabilitation. In Prentice, W. and Voight, M. (eds) *Techniques in musculoskeletal rehabilitation*. McGraw-Hill. New York

Collins, M. and Raleigh, S.M. (2009) Genetic risk factors for musculoskeletal soft tissue injuries. *Genetics and Sports*. 54: 136–149

Cook, J.L. and Purdam, C.R. (2009) Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. *British Journal of Sports Medicine*. 43: 409–416

Cowan, D.N., Jones, B.H., Frykman, P.N., Polly, D.W., Harman, E.A., Rosenstein, R.M. and Rosenstein, M.T. (1996) Lower limb morphology and risk of overuse injury among male infantry trainees. *Medicine and Science in Sports and Exercise*. 28 (8): 945–952

Cowey, C. (2006) Women in sport. In Higgins, R., Brukner, P. and English, B. (eds) *Essential sports medicine*. Blackwell Publishing. Oxford, UK

Creagh, U. and Reilly, T. (1998) Training and injuries amongst elite female orienteers. *Journal of Sports Medicine and Physical Fitness*. 38 (1): 75–79

Delahunt, E., Monaghan, K. and Caulfield, B. (2006) Altered neuromuscular control and ankle joint kinematics during walking in subjects with functional instability of the ankle joint. *American Journal of Sports Medicine*. 34 (12): 1970–1976

De Souza, M.J., Williams, N.I., Mallinson, R.J., Koehler, K., *et al*. (2014a) Misunderstanding the female athlete triad: Refuting the IOC consensus statement on relative energy deficiency in sport (RED-S). *British Journal of Sports Medicine*. 48 (20): 1461–1465

De Souza, M.J., Nattiv, A., Joy, E., Misra, M., Williams, N.I., Mallinson, R.J., Gibbs, J.C., Olmsted, M., Goolsby, M. and Matheson, G. (2014b) 2014 Female athlete triad coalition consensus statement on treatment and return to play of the female athlete triad: 1st International Conference held in San Francisco, California, May 2012 and 2nd International Conference held in Indianapolis, Indiana, May 2013. *British Journal of Sports Medicine*. 48: 289

Devan, M.R., Pescatello, L.S., Faghri, P. and Anderson, J. (2004) A prospective study of overuse knee injuries among female athletes with muscle imbalances and structural abnormalities. *Journal of Athletic Training*. 39 (3): 263–267

Dragoo, J.L. and Braun, H.J. (2010) The effect of playing surface on injury rate: A review of the current literature. *Sports Medicine*. 40 (11): 981–990

Ely, M.R., Cheuvront, S.N., Roberts, W.O. and Montain, S.J. (2007) Impact of weather on marathon-running performance. *Medicine and Science in Sports and Exercise*. 39 (3): 487–493

Engebretsen, A.H., Myklebust, G., Holme, I., Engebretsen, L. and Bahr, R. (2010) Intrinsic risk factors for groin injuries among male soccer players: A prospective cohort study. *American Journal of Sports Medicine*. 38 (10): 2051–2057

Enoch, S. and Leaper, D.J. (2005) Basic science of wound healing. *Surgery*. 23 (2): 37–42

Faubel, C. (2010) Nerve injury classifications – Seddon's and Sunderland's. *The Pain Source* http://thepainsource.com/2010/07/nerve-injury-classifications-seddons-and-sunderlands, accessed July 2011

Fields, K.B., Sykes, J.C., Walker, K.M. and Jackson, J.C. (2010) Prevention of running injuries. *Current Sports Medicine Reports*. 9 (3): 176–182

Fordham, S., Garbutt, G. and Lopes, P. (2004) Epidemiology of injuries in adventure racing athletes. *British Journal of Sports Medicine*. 38 (3): 300–303

Fousekis, K., Tsepis, E., Poulmedis, P., Athanasopolous, S. and Vagenas, G. (2010) Intrinsic risk factors of non-contact quadriceps and hamstring strains in soccer: A prospective study of 100 professional players. *British Journal of Sports Medicine*. 50 (4): 465–474

Fredericson, M. and Misra, A.K. (2007) Epidemiology and aetiology of marathon running injuries. *Sports Medicine*. 37 (4–5): 437–439

Fredericson, M. and Wolf, C. (2005) Iliotibial band syndrome in runners: Innovations in treatment. *Sports Medicine*. 35 (5): 451–459

Garrett, W.E. (1990) Muscle strain injuries: Clinical and basic aspects. *Medicine and Science in Sports and Exercise*. 22: 436–443

Gehlsen, G.M., Stewart, L.B., Vannelson, C. and Bratz, J.S. (1989) Knee kinematics: The effects of running on cambers. *Medicine and Science in Sports and Exercise*. 21 (4): 463–466

Ghani Zadeh Hesar, N., Van Ginckel, A., Cools, A., Peersman, W., Roosen, P., De Clercq, D. and Witvrouw, E. (2009) A prospective study on gait-related intrinsic risk factors for lower leg overuse injuries. *British Journal of Sports Medicine*. 43 (13): 1057–1061

Gholve, P.A., Scher, D.M., Khakharia, S. (2007) Osgood–Schlatter syndrome. *Current Opinion in Pediatrics*. 19 (1): 44–50

Giamberardino, M.A., Affaitati, G., Fabrizio, A. and Costantini, R. (2011) Myofascial pain syndromes and their evaluation. *Best Practice and Research Clinical Rheumatology*. 25: 185–198

Gottschall, J.S. and Kram, R. (2005) Ground reaction forces during downhill and uphill running. *Journal of Biomechanics*. 38 (3): 445–452

Grahame, R. (2000) Pain distress and joint hyperlaxity. *Joint Bone Spine*. 67: 157–163

Greenhalgh, S. and Selfe, J. (2006) *Red flags: A guide to serious pathology of the spine*. Churchill Livingstone, Elsevier. London, UK

Haim, A., Yaniv, M., Dekel, S. and Amir, H. (2006) Patellofemoral pain syndrome: Validity of clinical and radiological features: *Clinical Orthopaedics and Related Research*. 451: 223–228

Hakim, A. and Grahame, R. (2003) A simple questionnaire to detect hypermobility: An adjunct to the assessment of patients with diffuse musculoskeletal pain. *International Journal of Clinical Practice*. 57 (3):163–166

Hardy, M.A. (1989) The biology of scar formation. *Physical Therapy*. 69 (12): 1014–1024

Hennig, E.M. (2007) Influence of racket properties on injuries and performance in tennis. *Exercise and Sport Sciences Reviews*. 35 (2): 62–66

Hewett, T.E., Zazulak, B.T. and Myer, G.D. (2007) Effects of the menstrual cycle on anterior cruciate ligament injury risk: A systematic review. *American Journal of Sports Medicine*. 35 (4): 659–668

Hjelm, N., Werner, S. and Renström, P. (2010) Injury profile in junior tennis players: A prospective 2-year study. *Knee Surgery, Sports Traumatology, Arthroscopy*. 18 (6): 845–850

Holey, E. and Cook, E. (2011) *Evidence-based therapeutic massage: A practical guide for therapists*, 3rd edition. Churchill Livingstone, Elsevier. London, UK

Holmes, S. (2003) Undernutrition in hospital patients. *Nursing Standard*. 17 (19): 45–52

Houglum, P. (2010) *Therapeutic exercise for musculoskeletal injuries*, 3rd edition. Human Kinetics. Champaign, IL

Janda, V. (1983) *Muscle function testing*. Butterworth. London, UK

Kaeding, C. and Best, T.M. (2009) Tendinosis: Pathophysiology and non-operative treatment. *Sports Health: A Multidisciplinary Approach*. 1: 284

Kannus, P. and Józsa, L. (1991) Histopathological changes preceding spontaneous rupture of a tendon: A controlled study of 891 patients. *Journal of Bone and Joint Surgery (American Volume)*. 73A (10): 1507–1525

Kelley, J.D., Lombardo, S.J., Pink, M., Perry, J. and Giangarra, C.E. (1994) Electromyographic and cinematographic analysis of elbow function in tennis players with lateral epicondylitis. *American Journal of Sports Medicine*. 22 (3): 359–363

Killen, N.M., Gabbett, T.J. and Jenkins, D.G. (2010) Training loads and incidence of injury during the preseason in professional rugby league players. *Journal of Strength and Conditioning Research*. 24 (8): 2079–2084

Knight, K.L. (1995) *Cryotherapy in sports injury management*. Human Kinetics. Champaign, IL

Knight, K.L. and Draper, D.O. (2008) *Therapeutic modalities: The art and science*. Lippincott, Williams and Wilkins. Baltimore, MD

Knobloch, K., Yoon, U. and Vogt, P.M. (2008) Acute and overuse injuries correlated to hours of training in master running athletes. *Foot and Ankle International*. 29 (7): 671–676

Kolber, M.J., Beekhuizen, K.S., Cheng, M.S.S. and Hellman, M.A. (2010) Shoulder injuries attributed to resistance training: A brief review. *Journal of Strength and Conditioning Research*. 24 (6): 1696–1704

Korpelainen, R., Orava, S., Karpakka, J., Siira, P. and Hulkko, A. (2001) Risk factors for recurrent stress fractures in athletes. *American Journal of Sports Medicine*. 29 (3): 304–310

Leetun, D.T., Ireland, M.L., Willson, J.D., Ballantyne, B.T. and Davis, I.M. (2004) Core stability measures as risk factors for lower extremity injury in athletes. *Medicine and Science in Sports and Exercise*. 36 (6): 926–934

Li, J., Chen, J. and Kirsner, R. (2007) Pathophysiology of acute wound healing. *Clinics in Dermatology*. 25: 9–18

Lieber, R.L. (2002) *Skeletal muscle structure, function and plasticity: The physiological basis of rehabilitation*, 2nd edition. Lippincott, Williams and Wilkins. Philadelphia, PA

MacAuley, D. (2007) *Oxford handbook of sport and exercise medicine*. Oxford University Press. Oxford, UK.

Magee, D.J., Zachazewski, J.E. and Quillen, W.S. (2008) *Pathology and intervention in musculoskeletal rehabilitation*. Saunders, Elsevier. St. Louis, MO

Malliaras, P., Cook, J.L. and Kent, P. (2006) Reduced ankle dorsiflexion range may increase the risk of patellar tendon injury among volleyball players. *Journal of Science and Medicine in Sport*. 9 (4): 304–309

McHugh, M.P., Tyler, T.F., Tetro, D.T., Mullaney, M.J. and Nicholas, S.J. (2006) Risk factors for noncontact ankle sprains in high school athletes: The role of hip strength and balance ability. *American Journal of Sports Medicine*. 34 (3): 464–470

McRae, R. (2006) *Pocketbook of orthopaedics and fractures*, 2nd edition. Churchill Livingstone, Elsevier. London, UK

Meeuwisse, W.H. (1994) Assessing causation in sport injury: A multifactorial model. *Clinical Journal of Sports Medicine*. 4 (3): 166–170

Mester, J. and MacIntosh, B.R. (2000) Balance and control of movement summary. In Nigg, B.M., MacIntosh, B.R. and Mester, J. (eds) *Biomechanics and biology of movement*. Human Kinetics. Champaign, IL

Milgrom, C., Finestone, A., Zin, D., Mandel, D. and Novack, V. (2003) Cold weather training: A risk factor for Achilles paratendinitis among recruits. *Foot and Ankle International*. 24 (5): 398–401

Morrison, A.B. and Schöffl, V.R. (2007) Review of the physiological responses to rock climbing in young climbers. *British Journal of Sports Medicine*. 41: 852–861

Mountjoy, M., Sundgot-Borgen, J., Burke, L., Carter, S., Constantini, N., Lebrun, C., Meyer, N., Sherman, R., Steffen, K., Budgett, R. and Ljungqvist, R. (2014) The IOC consensus statement: Beyond the Female Athlete Triad – Relative Energy Deficiency in Sport (RED-S). *British Journal of Sports Medicine*. 48: 491–497

Mueller-Wohlfahrt, H.W., Haensel, L., Mithoefer, K., Ekstrand, J., English, B., McNally, S., Orchard, J., van Dijk, C.N., Kerkhoffs, G.M., Schamasch, P., Blottner, D., Swaerd, L., Goedhart, E. and Ueblacker, P. (2013) Terminology and classification of muscle injuries in sport: The Munich consensus statement. *British Journal of Sports Medicine*. 47: 342–350

Nadler, S.F., Malanga, G.A., Feinberg, J.H., Prybicien, M., Stitik, T.P. and DePrince, M. (2001) Relationship between hip muscle imbalance and occurrence of low back pain in collegiate athletes: A prospective study. *American Journal of Physical Medicine and Rehabilitation*. 80 (8): 572–577

Nigg, B.M. and Segesser, B. (1988) The influence of playing surfaces on the load on the locomotor system and on football and tennis injuries. *Sports Medicine*. 5 (6): 375–385

NOO (National Obesity Observatory) (2009) *Body Mass Index as a measure of obesity*. www.noo.org.uk/uploads/doc789_40_noo_BMI.pdf, accessed February 2014

Norris, C.M. (2004) *Sports injuries: Diagnosis and management*, 3rd edition. Butterworth Heinemann. London, UK

Orchard, J. (2010) *The Orchard Sports Injury Classification System (OSICS-10.1)* www.johnorchard.com/about-osics.html, accessed March 2014

Ortiz, A., Olson, S.L., Etnyre, B., Trudelle-Jackson, E.E., Bartlett, W. and Venegas-Rios, H.L. (2010) Fatigue effects on knee joint stability during two jumping tasks in women. *Journal of Strength and Conditioning Research*. 24 (4): 1019–1027

Paiva de Castro, A., Rebelatto, J.R. and Aurichio, T.R. (2010) The relationship between wearing incorrectly sized shoes and foot dimensions, foot pain, and diabetes. *Journal of Sports Rehabilitation*. 19 (2): 214–225

Pantano, K.J. (2009) Strategies used by physical therapists in the U.S. for treatment and prevention of the female athlete triad. *Physical Therapy in Sport*. 10: 3–11

Pefanis, N., Papaharalampous, X., Tsiganos, G., Papadakou, E. and Baltopoulos, P. (2009) The effect of Q angle on ankle sprain occurrence. *Foot Ankle Specialist*. 2 (1): 22–26

Peterson, H.A. (1994) Physeal fractures. Part 3: Classification. *Journal of Pediatric Orthopedics*. 14 (4): 439–448

Pino, E.C. and Colville, M.R. (1989) Snowboard injuries. *American Journal of Sports Medicine*. 17 (6): 778–781

Placzek, J.D. and Boyce, D.A. (2006) *Orthopaedic secrets*, 2nd edition. Mosby, Elsevier. Philadelphia, PA

Prentice, W.E. (2011) *Principles of athletic training: A competency-based approach*, 14th edition. McGraw-Hill. New York

Pujol, N., Blanchi, M.P.R. and Charnbat, P. (2007) The incidence of anterior cruciate ligament injuries among competitive alpine skiers: A 25-year investigation. *American Journal of Sports Medicine*. 35 (7): 1070–1074

Punchard, N.A., Whelan, C.J. and Adcock, I. (2004) Editorial: The Journal of Inflammation. *Journal of Inflammation*. 1: 1–4

Quatman, C.E., Myer, G.D., Khoury, J., Wall, E.J. and Hewett, T.E. (2009) Sex differences in weightlifting injuries presenting to United States' emergency rooms. *Journal of Strength and Conditioning Research*. 23 (7): 2061–2067

Queen, R.M., Abbey, A.N., Wiegerinck, J.I., Yoder, J.C. and Nunley, J.A. (2010) Effect of shoe type on plantar pressure: A gender comparison. *Gait and Posture*. 31 (1): 18–22

Rae, K. and Orchard, J. (2007) The Orchard Sports Injury Classification System (OSICS-10). *Clinical Journal of Sports Medicine*. 17 (3): 201–204

Rauh, M.J., Nichols, J.F. and Barrack, M.T. (2010) Relationships among injury and disordered eating, menstrual dysfunction, and low bone mineral density in high school athletes: A prospective study. *Journal of Athletic Training*. 45 (3): 243–252

Rees, J.D., Stride, M. and Scott, A. (2013) Tendons: Time to revisit inflammation. *British Journal of Sports Medicine*. http://dx.doi.org/10.1136/bjsports-2012-091957, accessed May 2013

Roberts, W.O. (2007) Heat and cold: What does the environment do to marathon injury? *Sports Medicine*. 37 (4–5): 400–403

Roberts, W.O. (2010) Determining a 'do not start' temperature for a marathon on the basis of adverse outcomes. *Medicine and Science in Sports and Exercise*. 42 (2): 226–232

Roland, M.O. (1986) A critical review of the evidence for a pain–spasm–pain cycle in spinal disorders. *Clinical Biomechanics*. 1 (2): 102–109

Rowson, S., McNally, C. and Duma, S.M. (2010) Can footwear affect Achilles tendon loading? *Clinical Journal of Sports Medicine*. 20 (5): 344–349

Sacco, D.E., Sartorelli, D.H. and Vane, D.W. (1998) Evaluation of Alpine skiing and snowboarding injury in a northeastern state. *Journal of Trauma-Injury Infection and Critical Care*. 44 (4): 654–659

Shanmugam, C. and Maffulli, N. (2008) Sports injuries in children. *British Medical Bulletin*. 86: 33–57

Sharma, P. and Maffulli, N. (2005) Current concepts review: Tendon injury and tendinopathy, healing and repair. *Journal of Bone and Joint Surgery*. 87-A (1): 187–196

Sharma, P. and Maffulli, N. (2006) Biology of tendon injury: Healing, modelling and remodelling. *Journal of Musculoskeletal Neuronal Interactions*. 6 (2): 181–190

Sherry, E. and Wilson, S.F. (1998) *Oxford handbook of sports medicine*. Oxford University Press. Oxford, UK

Simmonds, J.V. and Keer, R.J. (2007) Hypermobility and the hypermobility syndrome. *Manual Therapy*. 12: 298–309

Stearns, M.L. (1940) Studies on the development of connective tissue in transparent chambers in the rabbit's ear, part 2. *American Journal of Anatomy*. 67: 55–97

Swe Win, K. and Thing, J. (2014) 'The fatigued athlete' and RED-S: Lessons from the field and the BASEM spring conference. *British Journal of Sports Medicine Blog*. http://blogs.bmj.com/bjsm/2014/04/16/the-fatigued-athlete-and-red-s-lessons-from-the-field-and-the-basem-spring-conference-2, accessed April 2014

Tagliafico, A.S., Ameri, P., Michaud, J., Derchi, L.E., Sormani, M.P. and Martinoli, C. (2009) Wrist injuries in non-professional tennis players: Relationships with different grips. *American Journal of Sports Medicine*. 37 (4): 760–767

Taljanovic, M.S., Nisbet, J.K., Hunter, T.B., Cohen, R.P. and Rogers, L.F. (2011) Humeral avulsion of the inferior glenohumeral ligament in college female volleyball players caused by repetitive microtrauma. *American Journal of Sports Medicine*. 39 (5): 1067–1076

Taunton, J.E., Ryan, M.B., Clement, D.B., McKenzie, D.C., Lloyd-Smith, D.R. and Zumbo, B.D. (2003) A prospective study of running injuries: The Vancouver Sun Run 'In Training' clinics. *British Journal of Sports Medicine*. 37 (3): 239–244

Teyhen, D.S., Thomas, R.M., Roberts, C.C., Gray, B.E., Robbins, T., McPoil, T., Childs, J.D. and Molloy, J.M. (2010) Awareness and compliance with recommended running shoe guidelines among US army soldiers. *Military Medicine*. 175 (11): 847–854

Thein-Nissenbaum, J.M., Rauh, M.J., Carr, K.E., Loud, K.J. and McGuine, T.A. (2011) Associations between disordered eating, menstrual dysfunction, and musculoskeletal injury among high school athletes. *Journal of Orthopaedic and Sports Physical Therapy*. 41 (2): 60–69

Udry, J.R. (1998) Why are males injured more than females? *Injury Prevention*. 4: 94–95

Valle, X., Tol, H. and Hamilton, B. (2015) Muscle injury classification. In FC Barcelona, Aspetar and FIFA Medical Centres of Excellence. *Muscle Injuries Clinical Guide 3.0*. http://media3.fcbarcelona.com/media/asset_publics/resources/000/154/690/original/MUSCLE_INJURIES_CLINICAL_GUIDE_3.0_LAST_VERSION_pdf.v1428569103.pdf, accessed June 2015

van Gent, R.N., Siem, D., van Middelkoop, M., van Os, A.G., Bierma-Zeinstra, S.M.A., Koes, B.W. and Taunton, J.E. (2007) Incidence and determinants of lower extremity running injuries in long distance runners: A systematic review. *British Journal of Sports Medicine*. 41 (8): 469–480

Waryasz, G.R. and McDermott, A.Y. (2008) Patellofemoral pain syndrome (PFPS): A systematic review of anatomy and potential risk factors. *Dynamic Medicine*. 7: 9

Waterman, B.R., Belmont, P.J., Cameron, K.L., DeBerardino, T.M. and Owens, B.D. (2010) Epidemiology of ankle sprain at the US military academy. *American Journal of Sports Medicine*. 38 (4): 979–803

Watson, T. (2008) *Electrotherapy: Evidence-based practice*, 12th edition. Churchill Livingstone, Elsevier. London, UK

Watson T. (2012) Soft tissue repair and healing review. www.electrotherapy.org/assets/Downloads/tissue%20repair%202012.pdf, accessed June 2013

Wen, D.Y., Puffer, J.C. and Schmalzried, T.P. (1998) Injuries in runners: A prospective study of alignment. *Clinical Journal of Sports Medicine*. 8 (3): 187–194

Whiting, W.C. and Zernicke, R.F. (1998) *Biomechanics of musculoskeletal injury*. Human Kinetics. Champaign, IL

Willems, T.M., De Clercq, D., Delbaere, K., Vanderstraeten, G., De Cock, A. and Witvrouw, E. (2006) A prospective study of gait related risk factors for exercise-related lower leg pain. *Gait and Posture*. 23 (1): 91–98

Williams, J.Z., Abumrad, N. and Barbul, A. (2002) Effect of a specialized amino acid mixture on human collagen deposition. *Annals of Surgery*. 236 (3): 369–375

Wolf, B.R., Ebinger, A.E., Lawler, M.P. and Britton, C.L. (2009) Injury patterns in Division 1 collegiate swimming. *American Journal of Sports Medicine*. 37 (10): 2037–2042

Yavuz, M. and Davis, B.L. (2010) Plantar shear stress distribution in athletic individuals with frictional foot blisters. *Journal of the American Podiatric Medical Association*. 100 (2): 116–120

Yokozawa, T., Fujii, N. and Ae, M. (2007) Muscle activities of the lower limb during level and uphill running. *Journal of Biomechanics*. 40 (15): 3467–3475

2

CLINICAL ASSESSMENT IN SPORTS THERAPY

Keith Ward, Mike Grice, Rob Di Leva, Kate Evans, Jo Baker and Adam Hawkey

This chapter will discuss methodology in clinical assessment for sports therapy. A number of strategies, models and approaches – drawn from the wider fields of clinical sports and exercise medicine, orthopaedics, physiotherapy, osteopathy, athletic training and sports science – are presented in context with a consideration for how these may be incorporated into sports therapy practice. The chapter will also present implications of using clinical reasoning to reach working hypotheses and differential diagnoses, alongside an appreciation of factors that can influence the selection of interventions, and how these may affect the overall patient/athlete management plan. The consultation process will be explained, including an exploration of theories of best practice and the subtleties of undertaking a reliable and efficient patient history, including the identification of important nuances which underpin the therapeutic relationship. Following this, the principles and components of objective physical assessment will be presented, including structuring the process of examination and appreciating reliability and validity. Regional assessment approaches and specific techniques are discussed in the regional chapters of this book.

Introduction to clinical assessment

When a patient presents with an injury or complaint, the sports therapist is responsible for carrying out a systematic assessment to identify the reason for the pain or dysfunction and to determine the best possible interventions to assist them back to full function. Magee (2008) identifies the importance of establishing a 'sequential method' so as to ensure that nothing is overlooked. The main components of the consultation process can be separated into four main areas, routinely presented in terms of the acronym 'SOAP' (Quinn and Gordon, 2003):

- **S**ubjective assessment (appropriate personal, lifestyle, medical and injury information provided by the patient).
- **O**bjective assessment (technical, measurable physical examination performed by the sports therapist).
- **A**ssessment (analysis of assessment findings and formation of a 'differential diagnosis').
- **P**lan (formation of a 'problem list' and set of short- and/or longer-term goals).

The information gained from assessment is instrumental in forming a differential diagnosis which, for efficacy, must employ a certain level of clinical reasoning, which in simple terms is the decision-making process based on professional judgement and interpretation of the presenting information. The subjective assessment is an investigatory interview between the therapist and patient/athlete. The therapist should work to find clues during this assessment, which lead to the determining cause of the presenting condition. The main method of data collection in the subjective assessment is through specific and targeted communication between the therapist and patient. The information gathered is mainly qualitative data, and the quality of the information is therefore dependent on such factors as the questions asked, how the patient/athlete recounts their story, and how the therapist interprets their responses.

Following on from the subjective assessment is the objective assessment; and this is focused on gathering measurable clinical data. Objective assessment aims to produce mainly quantitative data with numerical or other graded values (i.e. degrees of joint range of movement; leg length measurements; strength ratings; positive or negative special test results). On completion of the assessment, the sports therapist will evaluate documented data to develop a rationale for the working hypothesis, differential diagnosis and prognosis of the presenting complaint, and determine a plan for the treatment modalities that can be adopted. The prognosis is the predicted (considered and estimated) level of function that the patient may attain within a certain time frame; and this prediction of recovery will help to guide the intensity, duration and frequency of the selected intervention(s), and will also aid in their justification (Dutton, 2008). To enable the sports therapist to obtain the best possible information from both the subjective and objective assessment, it is important to recognize the importance of the developing therapist–patient relationship, and how effective methods of communication influence the whole process.

Communication skills

The consultation process is a major medium of communication between the sports therapist and patient, and it is essential to invest time to perfect the skills which underpin this crucial aspect of practice. The information extracted from the patient in the initial consultation provides the basis for a successful assessment and eventual outcome. Lipkin (1997) identifies three functions of the consultation:

1. information gathering;
2. development and maintenance of the therapeutic relationship;
3. communication of information.

These three functions are intrinsically linked. For instance, a patient who does not particularly trust (or like) a sports therapist is less likely to divulge all necessary information; similarly, a patient who is anxious may not comprehend information clearly. The consultation process can quite frequently present challenges to the novice therapist who must aim to be friendly and flexible but also professional, confident and authoritative. The sports therapist–patient relationship directly determines the quality and completeness of information elicited and understood (Lipkin, 1997).

The communication between the sports therapist and patient is the integral part of the consultation process (Travaline *et al.*, 2005). The role of good communication skills in medical and allied health professions is well documented (Ferrari, 2006). Patients who understand their therapist are far more likely to acknowledge health problems, understand their treatment options and positively modify their behaviours (Bogardus *et al.*, 1999). An effective sports

Table 2.1 Functions of the consultation process

• To determine and monitor the nature of the problem
• To develop, maintain and conclude the therapeutic relationship
• To carry out patient/athlete education and implementation of treatment plans

therapist–patient relationship should aim to give the patient a sense that they have been heard and feel allowed to express their concerns in a respectful, caring and empathetic environment. This should also allow the patient to reflect their feelings and convey their story in their own words.

The Good Back Consultation

The 'Good Back Consultation' was a product of a research study carried out to determine what patients with chronic low back pain perceived as good clinical communication (Laerum, 2006). The guidelines from this research can be applied to other clinical settings and patient presentations. The key findings were:

- Patients wanted to be taken seriously (i.e. be seen, heard and believed).
- Patients wanted to be given an understandable explanation of what is causing their problem (i.e. use of diagrams, models and analogies).
- Patients wanted to have patient-centred communication (i.e. where patient perspectives and preferences are sought).
- Patients wanted to be told what can be done to improve their condition (i.e. by the patient and by the therapist).

Verbal communication

Verbal communication in the context of sports therapy is not confined to face-to-face meetings. Involvement with sports teams can mean that telephone conversations are needed, and it is important that the therapist can articulate themselves in such a way that they can be understood without the need for face-to-face contact. It is important to understand that different patients/athletes will prefer to receive information in different formats; as a therapist it is important to make sure the most effective communication methods are utilized (Laerum, 2006).

It is common in sporting environments to discuss the athlete's status with the medical team on a regular basis. While there are certain issues of informed consent with regard to the sharing of personal information, when appropriate the sports therapist must be able to present information about athletes in a format and language that is understandable to the people in attendance. The language used for the media or management will be different from the language used with the medical team, and may be different again when talking with the athlete. It is also important to consider appropriate use of language when dealing directly with the athlete, particularly when summarizing consultation findings. Adaptive skills are important for effective communication (Travaline *et al.*, 2005).

Developing a conducive and therefore effective patient–therapist relationship allows for a more accurate exchange of information. This dictates how information is delivered, and depending on how the patient/athlete interprets information, the sports therapist must be able to adapt their language terminology and use of jargon for different patients – the patient must at all times be able to understand what is being conveyed. Understanding the patient's belief systems and their interpretation of their problem, their expectations of how long recovery will take and the type of treatment they will receive, allows the therapist to map out a process of

Table 2.2 Adaptive communication skills

- Assess what the patient/athlete already knows
- Assess what the patient/athlete wants to know
- Be empathic
- Speak clearly
- Keep information simple
- Tell the truth
- Be hopeful
- Watch the patient's/athlete's body and face for non-verbal communication
- Be prepared for reactions

Source: adapted from Travaline *et al.*, 2005.

care. Such a process allows goals to be created to determine whether the initial hypothesis is materializing, or whether some confounding variables have materialized which might alter the prognosis (Matthews *et al.*, 1993). The sports therapist can be likened somewhat to a detective, seeking clues in the subjective and objective assessments so as to determine the likely pathology or dysfunction, as well as the aetiology. This is an essential component of clinical practice.

Personal contact

Palpation is a powerful non-verbal stimulus and communication medium (Willison and Masson, 1986). Sports therapists use palpation to ascertain anatomical and symptomatic information and to help provide a therapeutic intervention (Roger *et al.*, 2002). Contact is made with patients throughout the consultation process when examining and during treatment, whether instructing and positioning, facilitating movement or providing soft tissue therapy, mobilization techniques or other interventions. Contact and handling should be carefully explained to ensure that the patient is prepared and informed as to why it is necessary.

The sports therapist may sometimes work in unique settings; this can require that a working space needs to be created in a less than conventional setting. Such settings, where the boundaries between professional palpation and personal/intimate touch could become more easily blurred or obscured, must still be managed professionally. Sports therapists, as do all other health care professionals, require clear definitions and firm boundaries in order to maintain therapeutic efficacy and commitment (Schiff *et al.*, 2010). In best practice, the therapist makes an effort to explain what is likely to happen during the examination (CSP, 2012; SST, 2012). The patient will make assumptions of the therapist's clinical competency based on their handling skills – too vigorous (rough) handling will indicate insensitivity or a lack of empathy; too soft a handling technique may indicate a lack of confidence or inappropriate touch. Whether the handling is too vigorous or too soft, it will at the very least be ineffective.

Non-verbal communication

Alongside face-to-face and telephone communication, the sports therapist will also need to be able to deliver effective email and text communications. As always, the recommendation here must simply be to aim to be clear and professional. There is great potential to utilize electronic communication for confirming appointments and updates, but also for optimizing patient/athlete adherence to therapeutic programmes.

It is estimated that 60–65 per cent of interpersonal communication is conveyed via non-verbal behaviours (Burgoon *et al.*, 2009). Sports therapists should be aware of both their own and their patient's non-verbal behaviours and communication. Non-verbal behaviour ('body

language') includes touch, eye contact, facial expressions, gestures, body positioning and movement, listening, observing and using silence (Duncan, 1969; Exline, 1971). It is important to appreciate how non-verbal behaviour can contribute to the patient–therapist relationship.

Facial expression is one of the more straightforward non-verbal behaviours to identify and interpret. Ekman and Friesen (1971) identified several facial expressions of emotion that are identifiable across cultures, and there are obvious benefits of smiling and presenting pleasant facial expressions during the consultation process. Quite simply, a smile can convey feelings of contentment, happiness and pleasure, and can be very welcoming. Other facial expressions to consider when undertaking a consultation are those that convey genuine interest and concern at the patient's injury or problem. Patients will interpret appropriate facial expressions as indicators of empathy and interest, and this will support the development of rapport and trust. Eye contact can also influence the rapport between the therapist and patient. Effective use of eye contact serves to regulate interaction between the therapist and patient as well as to exercise subliminal social control, and help to facilitate service and task goals (Kleinke, 1986). A therapist may communicate their respectful interest in the patient's story through eye contact and show that there is engagement and an understanding from what is being said. Gestures and postures are used naturally and also deliberately as adjuncts to verbal communication to amplify verbal cues. During the consultation process, the patient may exhibit gestures or postures that indicate pain or apprehension, which may not be conveyed so obviously during verbal or facial expressions. The patient's posture may also demonstrate their level of tension or relaxation. For example, an adopted position of shoulder elevation can be possibly indicative of guarding, or a form of pain apprehension and tension; a slumped shoulder position may indicate a low mood state. Frequency of postural changes may also indicate the patient's level of agitation or anxiety.

Active listening is an important tool in the consultation process. It implies not only listening to the content of what is being said, but also interpreting and understanding feelings from how things are said (Banville, 1978). Positive gestures should be used to show the patient that they are being heard. Nodding in agreement, making detailed notes about what they are saying and then being able to repeat back what they have said are key strategies to demonstrate interest and responsiveness. Inevitably during the consultation process, periods of silence can occur. Periods of silent reflection need to be protected, and the sports therapist should aim to (briefly) embrace such an atmosphere.

Picking up cues in the subjective assessment is a key element to determine the way the therapist reacts and uses the information immediately, or so as to develop the bigger picture and use the information constructively later on. Listening for cues can prompt the therapist to implement individualized processes to engage with the patient. It is usually a collaborative approach that will be favourable to develop effective rapport. Following the initial consultation, the development and fostering of an effective patient–therapist relationship is built on the ongoing communication systems (Travaline *et al.*, 2005); and both verbal and non-verbal (face-to-face and electronic) forms of communication (which must be two-way) constitute this essential feature of practice. All key communicated information and advice regarding patients must be documented on their records; this includes information delivered via telephone, email or text.

Subjective assessment

The first stage of any consultation process is the subjective assessment; this is the patient's opportunity to tell their story. The subjective assessment must aim to provide the sports therapist with all necessary personal details, including medical history, as well as a clear initial understanding of the patient's aetiology and source of symptoms, and their perspective on their condition. By

the end of the subjective assessment the sports therapist should know about the patient's functional abilities and restrictions. This includes what they are able to do in terms of walking, lifting, sitting, training and specific sport activities, as well as work and home activities of daily living (ADL). The subjective assessment should also identify possible barriers, precautions or contraindications to further assessment, treatment or management. An efficient subjective assessment is fundamental to effective injury management (Refshauge and Gass, 2004). It is estimated that 80 per cent of the necessary information to explain the presenting problem can be provided by a thorough subjective assessment (Dutton, 2008). The subjective assessment lays the foundation for effective patient/athlete care. It is also important to note that by the end of the subjective assessment, the patient's interpretation of the severity, irritability and nature (SIN) of their symptoms should have been established. The SIN of symptoms influences which objective tests are to be selected, and also which treatment methods may be applicable. During

Photo 2.1 Undertaking subjective assessment.

Table 2.3 Key factors for effective subjective assessment

- Demonstrate professional appearance and behaviour at all times (overfamiliar, unprofessional behaviour does not inspire confidence).
- Wherever possible, provide a private, quiet and non-distracting environment (subjective assessments can contain highly confidential information and should be conducted in settings that maximize the patient/athlete's privacy).
- Always introduce yourself to new patients/athletes, and identify your role.
- Gain informed consent.
- Position yourself at the same level as the patient/athlete to avoid establishing dominance in the relationship.
- Know and respect the cultural norms and values of individual patients/athletes, and adjust interviewing techniques accordingly.

Table 2.4 Components of subjective assessment

Subjective assessment component	*Reason for information*
Presenting complaint	To determine the main reason for the visit. At this point, it is the patient's perception of their complaint.
Personal details (contact details; GP details; family history; occupation; social history)	To gather essential information, including age, gender, home and work situation, dependents and possible hereditary pathology.
Exercise and sports	To understand the patient's exercise and training routine (types; frequency; intensity) and sporting activity (types; level; position).
Past medical history (PMH)	To ascertain relevant medical history (investigations; interventions; trauma, illnesses), to review general health of body systems (e.g. digestive, respiratory systems) and identify any recent weight loss, dizziness or general malaise.
History of present complaint (mechanism of injury; onset; initial management)	It is important to record the circumstances of injury – both for clarification and for clues as to probable tissue involvement, and for medico-legal purposes.
Body chart (location of symptoms; genesis and any referral of pain)	Type and area of current symptoms, including pain and any paraesthesia (abnormal sensations).
Pain	Many cases of pain will be spontaneous in onset, or be related to perceived injury. The history of chronic pain must be fully explored. Pain of an insidious nature requires further investigation. Severity, irritability and nature (SIN) must be documented. Pain severity is recorded via visual analogue (or numerical rating) scale (VAS/NRS).
Frequency of symptoms	The subjective assessment should show how often the patient suffers pain. Frequency can help to identify the irritability of symptoms and highlight possible aggravating factors.
Duration	The duration of pain carries minimal diagnostic value. However, episodes of limited duration of pain suggest a lesser problem in terms of disability, but not necessarily lesser pathology.
Progression	If the symptoms are worsening, this may indicate an underlying pathological condition.
Precipitating factors	A patient who has pain-free intervals might identify activities that can bring on their pain. A record of precipitating factors provides a description of the patient and their problem.
Aggravating factors (AF)	Particular movements or activities will commonly aggravate a patient's pain. Listing aggravating factors provides a description of the patient and their problem, and foreshadows the assessment of disability. It provides some guidance as to which movements to undertake with care, or to possibly avoid, in objective assessment.
Relieving factors (RF)	Patients might identify factors that relieve their pain. These could include medications and interventions such as ice packs or hot baths. They may also include certain postures or activities. It is expectable that patients with a painful joint will feel better in postures that do not load that joint (e.g. lying down).
Associated features	Associated features suggestive of a more serious disorder may be more reliable than persistence of pain. Associated features can be explored during a systems review and during the general medical history.

the subjective assessment, the sports therapist should try not to ask questions or gather history that solely attempts to prove their hypothesis; it is important to keep an open mind as to what the reasons for the symptoms might be, and from this broad subjective assessment approach they can then begin to plan the objective assessment and start to rule out possible hypotheses. If the therapist starts from a narrow assessment base then the findings can also be narrow and the therapist may miss some vital clues as to the reason for the symptoms. It is important that the therapist develops a routine for the subjective assessment process but not be bound by that routine when they achieve additional clinical findings. The assessment order presented in this text is by no means definitive; however, it may be useful to break down the subjective assessment into a set of identifiable sections. It is worth noting that some of these categories will not be particularly relevant, nor will each question need to be asked for each patient. The subjective assessment must guide the objective assessment; but the temptation can sometimes be to put too much emphasis on the objective assessment and abbreviate the subjective assessment, whereas in reality, quite the opposite should be true.

Record keeping

During the consultation process, sports therapists must complete consultation and assessment forms for each patient. For the newly qualified therapist, the familiar structure and layout of the patient record form may serve as a memory prompt to ensure that all required information is obtained from the patient. It is essential from a legal and professional perspective that an accurate record of the consultation procedure and outcome is kept; importantly, it may be used to support the practitioner's actions should they be required to defend themselves against any claims of negligence or malpractice. It is also essential so that the therapist can revisit notes to compare clinical presentations and maintain clinical vigilance from one assessment to the next. (A sample 'Confidential Patient Record Form' is provided in the Appendix.)

Notation and legal requirements

Any assessment notes should be thorough but concise. It is expected that medical records are full, contemporaneous (up to date), legible and stored securely. All attendances and entries must be dated and initialled; this includes any advice given to the patient (even if provided over the telephone or via email or text). It is the duty of the sports therapist, as part of their professional standards of conduct, performance and ethics (SST, 2012), to ensure that full records are maintained. The use of standardized abbreviations assist note taking to reduce the amount of time required to produce and maintain athlete medical records. It is important that the sports therapist uses standardized abbreviations that facilitate information recording quickly and efficiently, and also so that patient data can be readily disseminated to colleagues and other medical professionals where appropriate. It is equally important that agreed standardized lists of abbreviations are used where a multidisciplinary team is responsible for the care of patients/athletes. The patient record form must be considered as a working legal document. Well-presented patient records safeguard both the practitioner and the patient, and provide a reference to previous treatments and advice. It is recommended that patient records are fully updated within 24 hours of seeing the patient. If in hard copy, all entries should be written in black ink, with any errors in the notes crossed through and initialled by the therapist who is making the correction (correction fluid should not be used). If electronic records are being held, these must be stored in a secure, password-protected system, and each update to the records should be saved as a separate file; robust back-up storage is also to be recommended. Patient records are confidential documents and must be stored in keeping with the Data Protection Act (1998) (ICO, 2013).

The record form must contain a section regarding 'informed consent'. There are two basic components to this; first, there is an information exchange – meaning that information from both sides is clearly and truthfully disclosed and understood. For this, the patient must understand that they need to provide all the necessary personal and medical information; and the therapist must provide the patient with a clear explanation of what is proposed during the assessment and treatment session. Second, consent signifies voluntariness on the part of the patient, i.e. they agree to what has been proposed (Sim, 1996). It is essential that this aspect of the process is effectively undertaken for a number of reasons, not least to offset the potential for accusations of malpractice or negligence from patients who may claim that they were not expecting to be physically examined, for example. There may be situations where informed consent is not so easily achieved, such as when there are language barriers or mental health issues; the sports therapist must in such instances still take steps to achieve informed consent – in the case of mental health issues, perhaps a partner, parent, carer or other chaperone can provide this. In the case of children, the parent or guardian must provide it. Beyond this, it is only for the optimal outcomes that informed consent to assessment and treatment is achieved; and so that the sports therapist can proceed safely. Informed consent must also be achieved prior to the sports therapist sharing any patient records with third parties (such as another health care professional, sports team or insurance company). The sports therapist must also recognize that the patient is entitled to access their records at any time.

It is recommended that if any assessment of 'intimate areas' is required, then this should be clearly noted on the record form and initialled by the patient after an explanation of the procedure. It is advisable that adults' consultation records be kept for eight years; in the case of children (under 18 in Wales and England, under 16 in Scotland) it is considered best practice for all records to be kept until the athlete is 21 years old, and a further eight years after that. After this time, all records should be destroyed as confidential waste.

The patient's personal details

The consultation process involves the initial stage of collecting administrative information. Essential details to be ascertained include: the patient's full name; date of birth; address; telephone number; email address; occupation(s); GP name; GP practice address and contact telephone number. Clarifying the patient's marital status and whether they have any dependents can be important features for clinical reasoning; such information may indicate the various external pressures the patient faces. It is efficient to consider a number of additional aspects of the patient's lifestyle and situation as they may have an effect on their ability to adhere to future advice and rehabilitation; for example: Have they recently moved house? Do they have new-born children? Do they care for elderly relatives? Do they have a long commute? Are they self-employed? It is essential that the physical components of the patient's lifestyle are effectively explored, especially with regard to their exercise and sporting regimes. The sports therapist must understand what exercise, training and recovery the patient/athlete incorporates into their normal routine. Their training history, current programme, fitness level and goals should be discussed, as should their sport(s) activities. If the patient is a competing athlete or belongs to a team, the sports therapist should ascertain to what level they compete, what their particular sport/position entails, whether they are under the care of a medical doctor or sports practitioner as part of the team environment, and, if they have a coach or manager, are they aware of their visit?

Practitioner Tip Box 2.1

Questions for the patient during subjective assessment

- Appropriate questions (e.g. What activities seem to aggravate your condition?)
- Short and clear questions (e.g. On a scale of 0–10, with 10 being the worst pain imaginable, how much pain are you in at this moment?)
- Closed questions (e.g. What sports do you do?)
- Open questions (e.g. Can you explain what happened?)
- Responsive questions (i.e. questions which are presented in response to the previous information provided by the patient)
- Special questions (*'to determine the nature of the patient's condition, differentiating between benign neuromusculoskeletal conditions which are suitable for manual therapy and systemic, neoplastic or other non-neuromusculoskeletal conditions which are not suitable for treatment'*) (Petty, 2011)

Previous medical history and general health

A general medical history needs to be obtained from the patient so that their health condition is understood, and to identify whether they have any congenital, hereditary, developmental or acquired comorbidities of significance. Further to this, are there any familial health issues of relevance (for example, history of cancer or heart disease)? It is important to clarify whether the patient is under the care of another health professional currently, such as their GP – if so, the reason for this must be discussed (and documented). It is essential to record what medications the patient is taking, ideally this will include either the brand (proprietary) name (the name given by the pharmaceutical company that produces it) or the generic (scientific) name (the name of the active ingredient in the medicine that is decided by an expert committee and is understood internationally) (NHS, 2012). The sports therapist needs to identify whether the medication is on prescription or non-prescription ('over the counter' – OTC), and should appreciate why the patient is taking the medication, what the dosage and frequency is, whether there are any adverse or side-effects and how long the patient has been taking them. A brief 'systems review' should also be undertaken. This means that the sports therapist should ascertain the patient's general health regarding their musculoskeletal, neurological, gastrointestinal, endocrine, cardiovascular, respiratory and renal systems. Furthermore, any medical investigations or interventions of significance (recent or major) must be documented; these can include blood tests, imaging, injection therapy or operative interventions. Other information that is important to ascertain can include confirming any history of allergic reactions, recent weight loss or gain, dizziness, malaise, nausea, depression or anxiety and, in females, menstrual problems or pregnancy (Petty, 2011). The sports therapist must be particularly vigilant to the details of any medical condition which may be relevant to the patient's main complaint and how it may be objectively assessed and managed. At this point in the subjective assessment, the therapist may have already gathered enough information to have identified precautions or contraindications to further assessment or treatment; clues to the possibility of serious underlying pathology ('red flag' signs and symptoms) may also be presented during subjective assessment. Sports therapists must be responsible for identifying and recommending or requesting medical assessment when signs, symptoms and history suggest the possibility of any serious pathology, and indeed whenever clarification of a medical condition or approval for sports therapy intervention is required.

Practitioner Tip Box 2.2

Example 'red flag' signs and symptoms

- History of malignancy
- Severe, unrelenting or worsening pain
- Severe pain with no apparent history of injury
- Night pain
- Pain unrelated to movement
- Severe spasm
- Loss of appetite
- Recent unexplained weight loss
- Significant neurological symptoms (e.g. cauda equine syndrome; drop attacks)
- Significant cardiorespiratory symptoms (e.g. shortness of breath; warm, discoloured, swollen limbs)
- Feeling generally unwell (malaise) (e.g. unexplainable fever or fatigue)
- Lesions which have failed to heal
- Unusual lumps or growths

* This list is not exhaustive. Any suspicion of serious underlying pathology requires urgent medical referral.

Questioning may also extend to any problems involving the rest of the kinetic chain; for example, has a patient with a current back problem sought treatment previously for any hip, knee, ankle or foot injuries, even if they are on the contralateral side of the body? Previous injuries may seem insignificant to the patient but they can be very important to the sports therapist who is attempting to put together a picture of the patient's intrinsic biomechanical history. It is also important to ascertain all other current or previous injuries or problems to ensure the therapist does not aggravate these while assessing and treating the main complaint.

Social history and family history

Information about the patient's living circumstances, marital status and details of lifestyle and leisure activities offers information about their social and family history. Certain psychosocial factors may be identified and recognized by the therapist as being 'yellow flag' signs. Throughout the assessment, the therapist should aim to identify predictors of chronicity, elements of risk behaviour, fears and fear avoidance, distorted belief patterns, low mood and possible external stressors, all of which can influence choice of treatment interventions, and can greatly influence the patient's prognosis (George *et al.*, 2008). Patients with underlying psychosocial issues may sometimes elect to withhold certain personal or medical information; such situations can also affect how pain and symptoms are reported (especially with persistant pain); patients may also be less likely to adhere to advice, even if this is unintentional. Certain patients can sometimes be involved in compensation claims with insurance companies following accidents and incidents, and therapists should recognize that this can be an influence in how they respond to assessment (Ferguson, 2009). Therefore there is an onus on detecting such underlining mechanisms. Exploring psychosocial factors forms the cornerstone for a strong patient–therapist relationship; it does, however, require an extremely sensitive, mature and confident approach, but it can be

conducive to the delivery of effective care and optimal outcome, which may not have been otherwise assumed. The 'family history' considers the risk factors and potential for conditions which may have a genetic predisposition (such as ankylosing spondylitis; cancer; heart disease; hypertension; psoriasis; or rheumatoid arthritis); and hence the sports therapist will have increased awareness for suspicion of such conditions in their differential diagnosis. A careful consideration of the patient/athlete's situation and family history may also highlight such background issues as alcoholism, depression or drug dependency. Considering the family history of the patient can help to outline their relationships with others and their domestic situation. Awareness of such factors can help to influence management decisions and home care advice.

The history of the main complaint

In situations where the current injury appears to be a reoccurrence, previous assessment findings, treatment methods, functional outcomes and time frames should be established (this may include orthopaedic investigations and interventions, physiotherapy or even previous sports therapy). Whether the injury or condition is new or reoccurring, an understanding of how it first occurred is essential. The 'mechanism of injury' (MoI) may be simply viewed as a single traumatic event or as a result of repetitive microtrauma. Either way, the onset and history of the presenting complaint must be fully explored with careful questioning; and the therapist must provide full opportunity for the patient to recall and explain their experience. The sports therapist should use open-ended questions wherever possible to elicit information during subjective assessment. It is important that the patient has the opportunity to recount their story in their own words and understanding, particularly in explaining the history of their presenting condition. How and what the patient focuses on provides valuable insight into their perception of the relative importance of the symptoms being experienced.

Symptom assessment

Once the mechanism of the injury is established, the therapist should discuss the symptoms that the patient has experienced. It will be helpful to identify: what symptoms were experienced immediately at the time of injury; how severe they were; where these symptoms were felt; whether there were any abnormal sounds or sensations at the time of injury; how the area looked immediately after the injury; whether the symptoms altered from the time of the injury to present, and if so, how; whether the injury occurred during sport or exercise, and if so, whether they were able to play on.

It is important to establish if the athlete has acute, subacute or chronic symptoms, as the management options for each can be quite different. In the acute injury presentation, where the mechanism of injury is known, the potentially injured structures should be identified. Once the therapist has established the pain type, location, severity and number of pain problems (i.e. P1; P2), they may wish to question the patient on the behaviour (or pattern) of symptoms. The goal here is to establish whether pain is constant or intermittent in nature. True constant pain is unremitting, with no relief. It can be useful to ask someone who says they are in constant pain if it is hurting currently. Many patients will describe pain as constant, but it may not be painful when the question is asked during the subjective history. Constant pain that does not vary in intensity, or where no position of ease can be found, must be viewed as a red flag sign and referral considered. Constant pain that varies in intensity can be indicative of inflammatory or infective processes. Intermittent pain that comes and goes is often mechanically stimulated and is typical of many sports injury presentations. The therapist may also question the patient regarding their 24-hour behaviour of symptoms. This can help to establish how symptoms alter

from morning to evening and night, as well as following activity or rest. In the morning, the therapist should establish the patient's symptoms on waking and on rising (i.e. getting out of bed). Prolonged morning pain and stiffness also suggests inflammation. Conversely, minimal or absent pain in the morning is associated with degenerative conditions such as osteoarthritis (Petty, 2011). Evening symptoms are often dictated by the patient's daytime activity levels and perhaps their occupation. Pain that is aggravated by movement is often worse at the end of the day. Night symptoms can indicate more serious pathology and must be explored fully. The sports therapist should establish if the patient has any difficulty sleeping due to their symptoms. If symptoms are relieved by lying down, this may represent the unloading of load-bearing symptom-producing structures. If the patient can only find comfort and relief and therefore sleep in one position, this can indicate the locality of a structural issue. It is important to note any positions of relief. If the patient is woken by symptoms, these should also be noted. It needs to be established if waking from sleep is associated with movement; for example, it is not uncommon for an injured medial collateral ligament of the knee to be aggravated by turning over onto the unaffected side while sleeping in bed, provoking enough pain to wake the patient. It is important to note the frequency of such painful events; how often per night, per week, and how long it takes for the pain to subside and for the patient to return to sleep. The therapist should also aim to identify any changes to the sleeping environment – this can include the type and number of pillows and the type of mattress, and how recently these may have been changed. These are particularly important with regard to spinal or back pain. Pain that prevents the patient from getting to sleep or, once woken, cannot be alleviated with a change of position or medication and prevents them from returning to sleep should be taken seriously, with referral to their GP. Information regarding the patient's symptoms must be compared on subsequent visits so that an objective view of their progress can take place.

Maitland *et al.* (2005) consider the patient's pain severity and irritability, as well as the nature of the disorder (SIN). These characteristics are important to guide the therapist's clinical impression of the problem. The severity of the injury is determined by the extent of injury, the type of pain and the intensity of pain. The 'visual (or verbal) analogue scale' (VAS) (or numerical rating scale – NRS) is helpful when determining both severity and irritability. The VAS is a numerical pain intensity scale of 0–10, where 0 indicates no pain at all and 10 represents the worst pain imaginable. The VAS is a subjective measurement instrument that is designed to measure the pain characteristic that is believed to range across a continuum of values. The patient identifies on the line the point that they feel represents their perception of their current state. The VAS score is determined by measuring (on a scale of 0–10 cm) from the left-hand end of the line to the point that the patient marks (Wewers and Lowe, 1990). The far left end indicates 'no pain' and the far right end indicates 'worst pain ever'. The score is out of 10 and may be recorded as 2/10 (little pain) or 7/10 (moderate pain) and so on. The VAS has most value when looking at change within individuals, and less value for comparing across a group of individuals at one time point.

A patient complaining of sharp, stabbing pain rated as 8/10 on a VAS can be categorized as having a high severity. A patient complaining of a dull ache which is no more than 3/10 may be deemed low to moderate severity. As a standalone characteristic, the severity of pain carries modest diagnostic weight. Patients may describe their pain as severe, but this does not necessarily suggest a serious or threatening condition. The severity and VAS score must be considered as part of all other clinical findings and in the context of other features. It is, however, helpful to record the severity of pain at baseline and on subsequent visits, using the quantitative measure of the VAS (Carlsson, 1983; Chapman *et al.*, 1985; Strong *et al.*, 1991), as this provides a measure of how the pain is changing. This should be more reliable than the patient's or

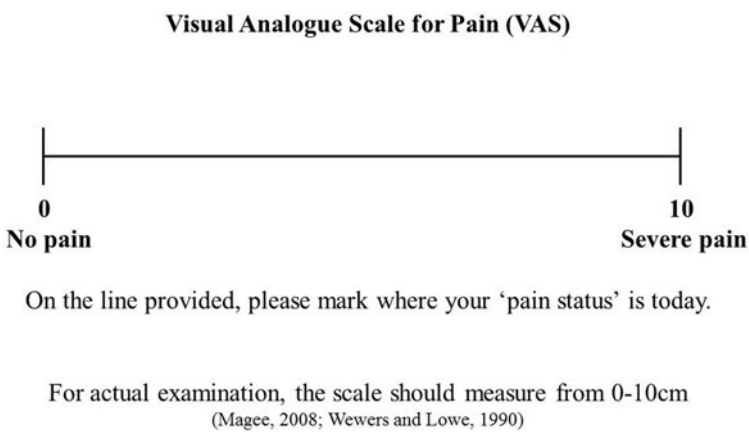

Photo 2.2 The visual analogue scale.

therapist's memory of severity over time. Alongside the severity of pain, it is important to establish the irritability of pain symptoms. Pain irritability is assessed by how vigorous an activity is before symptoms appear, the severity of those symptoms and the time it takes for those symptoms to subside once aggravated (Koury and Scarpelli, 1994; Maitland *et al.*, 2005; Smart and Doody, 2007). Appreciating irritability helps the therapist determine how physically vigorous they can be throughout the objective assessment which follows, as well as the dosage of intervention. There is value in regulating how rigorous the assessment will be; this is chiefly to avoid exacerbating symptoms while still maximizing outcomes (Barakatt *et al.*, 2009).

As part of a full subjective review of the patient's complaint, the sports therapist must explore which factors appear to aggravate or ease the patient's condition, and also what they have done to help manage the condition. How was the injury initially managed? Did any assessment take place? Did the patient/athlete apply a PRICE (protection, rest, ice, compression and elevation) or other regime? Were there any referrals for further assessment such as an orthopaedic consultation or imaging? Were medications prescribed or taken?

Aggravating factors (AF) are particular movements, positions, activities or other things (such as heat applications) that might be responsible for reproducing or increasing the patient's symptoms. The exact movement or position, the delay in the onset of symptoms, intensity of symptoms and the duration of the increase in symptoms should be explored. Where more than one pain problem is established, aggravating factors can help establish the relationship between problems. If all symptoms are aggravated by the same factors it can be that the symptoms are related and are provoking the same structural dysfunction. Aggravating factors may be more functional in nature, so it may be necessary to consider questioning the patient on their ability to perform ADLs or sports-related movements, as well as establishing whether they are able to maintain a normal conditioning programme. Such restrictions can become objective markers of the efficacy of the rehabilitation programme.

Relieving factors (RF) are movements, positions or perhaps certain self-administered interventions (such as ice, heat, massage, strapping or OTC medications) that may help to alleviate symptoms. The sports therapist should establish exact movements or positions which appear to decrease the intensity or delay the onset of symptoms. This information can provide the therapist with insight as to how easily symptoms may be attenuated during treatment. It may also provide an insight into the structures that are implicated. As with aggravating factors,

multiple pain problems eased by the same factors can indicate a relationship and a commonality of dysfunctional tissue.

Pain itself is a complex topic; it can be broadly categorized as being nociceptive, peripheral neuropathic and/or centrally sensitized. Symptoms that are brought on by physical movement and biomechanical loading, but resolve soon after the movement and loading has ceased, can usually be deemed mechanical in nature. Prolonged inflammatory injury pathologies such as bursitis, capsulitis and some tendinopathies can become chronic in nature and may also reflect an acute-on-chronic episode. The therapist can ask the patient to describe the type of pain they are experiencing and try to elicit the qualities this pain has. Newham and Mills (1999) identified general types of pain often associated with particular tissues. For instance, conditions involving bone pain may be associated with reports of deep, boring, nagging or dull pain. Post-acute muscle pain is associated with dull aching. Peripheral nerve root pain is associated with sharp, shooting, lightning-like pain. Sympathetic nerve pain is often reported as being a burning, stinging, pressure-like pain; damaged highly vascular structures can manifest pain as a throbbing and diffuse sensation. It is also interesting to note that Mense (1993) associated deep pain with muscles and superficial pain with joint injury. It is therefore beneficial to question the depth and characteristic of the patient's pain. Obviously, pain is often the primary complaint and reason for the initial sports therapy consultation. Understanding pain requires the sports therapist to appreciate the complexities that are involved in a patient's or athlete's pain. Pain is subjective and different for each individual. There are a host of factors that determine pain. It is particularly helpful for the therapist to aim to gather specific information so as to be able to understand the genesis of the patient's pain (the 'genics'). The general sources of pain, therefore, may be simply categorized as myogenic, arthrogenic, neurogenic, discogenic, viscerogenic or psychogenic, for example. Doubell *et al.* (2002) identified common characteristics of pain mechanisms. Nociceptive pain is generated due to noxious mechanical, thermal and/or chemical stimulation of peripheral receptors and is modulated in the brain. It is typically localized and predictable in its behaviour. Examples of nociceptive pain include that resulting from ligamentous sprains, fractures, impact trauma, inflammation and myofascial pain. Non-systemic peripheral neurogenic pain often follows neural distribution patterns, and can be characterized by burning, sharp or shooting type pain, and may be provoked by movement and positioning (i.e. nerve tensioning and/or compression). Persistent pain that stems from central sensitization can be widespread, more diffuse and likely to be challenging for the patient to explain or understand. Sports therapists will appreciate that, while the central sensitization of pain is very much associated with chronic pain situations, there is a central component (modulation) to all pain presentations. Centrally sensitized persisting pain is complex and multifaceted, but can involve dysfunctional neural processing associated with psychological components which can manifest in hyperalgesia (a heightened sense of pain in the periphery), allodynia (an abnormally painful response to innocuous stimuli) and altered behaviours (such as fear avoidance), which can contribute to the situation.

Somatic referred pain is perceived in a region innervated by nerves or branches of nerves other than those which innervate the primary source of pain, where that source lies in one of the tissues or structures of the body wall (soma) or limbs (Merskey and Bogduk, 1994). A similar definition applies to referred viscerogenic pain, save that the primary source lies in one of the organs of the body. An example of this is the left arm and jaw pain that can occur during myocardial infarction. In both somatic and visceral pain the primary pain is produced by the stimulation of the peripheral endings of nociceptive afferent fibres. In contrast, neurogenic pain is pain produced by the stimulation of peripheral axons themselves, or their cell bodies

(rather than their peripheral endings). Radicular pain is a subset of neurogenic pain, in which pain is evoked by stimulation of the nerve roots or dorsal root ganglion of a spinal nerve. In neurogenic pain, the pain is perceived in the region of the affected nerve. Neurogenic pain is a form of referred pain. It differs, however, from somatic and visceral referred pain in that it does not involve the stimulation of nerve endings, and does not involve convergence. Rather, it is perceived as arising from the periphery because the nerves from that region are artificially stimulated proximal to their peripheral distribution (Merskey and Bogduk, 1994). Centrally sensitized pain tends not to follow a pattern and is less predictable. The patient may also use emotional descriptions to describe their pain, such as it being unbearable or insufferable. This may indicate a habitual, psychosocial or behavioural element to their symptoms. A sclerotome is a region of bone that is innervated by a particular spinal segment (Grieve, 1988). Sclerotomal referred pain extends beyond its locality and is often overlooked for others of a more muscular origin.

When the patient presents with pain, one of the main objectives is to attempt to establish where the pain is coming from as accurately as possible. However, in some instances the patient may have pain arising from a region which may be greater than that initially indicated, such as with referred pain, or pain which only manifests when tissues are aggravated; or they may have more than one pain problem. If the patient has more than one pain, which are presumably separate in origin, a separate history should be taken for each. Each symptom may have a different cause and mechanism requiring different clinical investigation and management, or they may be linked due to the compensational biomechanical changes from the first injury. If the patient has multiple complaints related to one source, the separate histories should be combined.

Location of symptoms

Any relevant signs or symptoms should be recorded on the body charts on the patient record form. These show anterior, posterior and lateral views of the body, and it can be useful to ask the patient to complete this as they can more accurately locate the site of their symptoms. Obviously, the areas identified as painful or otherwise symptomatic by the patient are recorded. It is recommended that therapists label the identified areas of symptoms – making sure that symptoms have been clearly marked, and on the correct side of the body! Areas of scarring (such as surgical, atrophic, hypertrophic or keloid), bruising (contusion) and abnormal and unpleasant sensation (such as paraesthesia or dysaesthesia) should also be recorded. Simple abbreviations may be used to identify symptoms which have been labelled on the charts (i.e. P1 = pain 1; P2 = pain 2; C = constant; I/M = intermittent; P+Ns = pins and needles) (Petty, 2011). As a matter of clinical diligence, the therapist should check all other relevant anatomical areas for related symptoms such as stiffness or discomfort. Once these areas have been cleared a tick (✓) is placed on that area on the body charts. The information documented may not identify exact structures at fault, but the therapist can justify any relationships between symptoms, or decide whether each symptom is individual. Referred pain is the term given when the area of symptoms does not align directly with the structures at fault. Referred pain by definition is perceived in a region remote from the actual source of pain.

Patients will also present when they are not necessarily in pain or otherwise symptomatic. This can be challenging as it may be initially perceived that there is nothing specifically to assess or treat. However, sports therapists may advise completion of a functional movement or intrinsic biomechanical screen to assess the range and quality of movements at key areas, particularly those that are implicated in the patient/athlete's sport, and which may then be able to highlight possible dysfunctions at risk of becoming problematic.

The clinical impression and differential diagnosis

By the end of the subjective assessment the sports therapist should be aiming to have a differential diagnosis and justifiable clinical impression (working hypothesis) of the patient. Good verbal and non-verbal communication skills, along with appropriately selected questions, should enable the therapist to generate hypotheses based upon the MoI, the onset, the signs and symptoms and SIN, the VAS, the AF and RF and the body charts. The therapist should be aiming to link all key patient information so as to contextualize which structures and tissues may be injured and also what the root aetiology may be. The differential diagnosis is a set of named conditions that the sports therapist has clinically reasoned, based on the patient's history. The clinical impression incorporates a full and individualized appreciation of the underlying causes, including the patient's gender, age, general health, fitness, wellness and medical history, previous injuries, biomechanics, lifestyle factors (such as ergonomics, smoking, alcohol and diet), as well as psychosocial and environmental factors. It has been recommended that clinicians aim to generate up to five differential diagnoses (Elstein *et al.*, 1978). The considered differential diagnosis that the therapist establishes should then be prioritized, with the most serious condition being tested first in the objective assessment. It can be useful to categorize the differential diagnoses into the order 'life threatening', 'life changing' or 'treatable'. It may simply be that certain precautions or contraindications to objective assessment or treatment interventions have been identified by this point (Petty, 2011).

The objective assessment is then used to prove or disprove the hypothesis. The objective assessment should begin by attempting to identify any serious condition, and if positive, an immediate referral may be required. Once the therapist is convinced that there is no underlying serious pathology or referral concern, they can continue the objective testing to ascertain if the presenting condition is treatable. Objective testing and prioritizing can be a slow process when the therapist is training or newly qualified, but as more experience is gained these tests can be conducted more efficiently with a higher degree of confidence and competency.

Practitioner Tip Box 2.3

Optimizing the assessment process

- Demonstrate professional appearance, attitude and behaviour at all times.
- Inform and explain to the patient the process of assessment.
- Overfamiliar, unprofessional behaviour does not inspire confidence.
- Wherever possible, provide a quiet, non-distracting private environment. The patient history contains highly confidential information and should be undertaken in settings that show respect for this.
- Understand and respect cultural norms and values of individual patients and adjust consultation techniques accordingly.
- Inform the patient of the process of assessment and treatment and clarify that it is their responsibility to provide all required health information.
- Do not become overly fixated with taking notes at the expense of ignoring the patient.
- Give explanations in a language that the patient can comprehend.
- Be an attentive, non-judgmental, active listener, and an alert observer throughout the consultation process.

- Be mindful not to interrupt prematurely, and control any urges to fill every pause or silence with another question.
- Avoid the use of overly complex medical jargon.
- Give the patient enough time to reflect on their answers to questions.
- Engage patients to become partners in the rehabilitation process.
- Determine their health care goals and expectations about their care.
- Observe non-verbal behaviours throughout the consultation.
- Acknowledge the value of the patient's information through the use of supportive statements during and at the end of the consultation.
- Once all main personal details and general health questions have been completed, the sports therapist may then ask open-ended questions such as 'How can I help you today?' or 'What seems to be the problem?' to explore the history and background of the main complaint.
- The sports therapist should verbally summarize their understanding of the data and ask the patient if it is an accurate portrayal of the information that has been provided.
- If the consultation yields contradictory information, the therapist should revisit earlier areas of inquiry to check for consistency of response and/or ask the patient for clarification.
- Beware of prematurely cutting off a line of diagnostic inquiry.
- Although the patient's presenting symptoms may strongly suggest a particular diagnosis, failure to adequately explore alternative explanations may cause the therapist to falsely reject an important differential diagnosis.
- Provided the correct safeguards are in place, the therapist should ask the patient to email or telephone any additional information pertinent to their care that they may have forgotten to mention during the consultation.

Source: adapted from Lipkin, 1997.

Objective assessment

The adjective 'objective' is defined in the Longman Dictionary (1995) as *'existing independently of the mind'*. Objective assessment is based upon observable phenomena. In order to be objective in assessment, one must observe and be impartial to what is seen. The objective assessment aims to ensure that the therapist has assessed all probable causes of the patient's symptoms in a way that prioritizes risk. Sports therapists should aim to be able to associate the complaint with a specific region and, if appropriate, specific anatomical structures. The physical assessment process is the inspection, palpation and measurement of the body and its parts (Gross *et al.*, 2009). It is the step in the process that follows the subjective assessment, and it precedes the reaching of a clinical diagnosis. The purpose of the objective assessment is to establish the most likely (working) diagnosis using information generated from the clinical impression (hypothesis) formed in the subjective assessment. The process involves the gathering of evidence to support or disprove the differential diagnosis. With any applied test procedure, the therapist is seeking to reproduce symptoms of the primary complaint (these are known as comparable signs) and to compare the injured or problematic to the unaffected side (bilateral comparison). According to Maitland *et al.* (2005), two assumptions can be made when performing physical assessment; the first assumption is that if symptoms can be reproduced or eased, then the test has somehow affected the problematic structures. The assumption here is that all tests have the ability to

isolate structures, be they anatomical or physiological. Any test conducted as part of an objective assessment is likely to stress a number of structures and can therefore implicate more than one locally or remotely (proximal or distal). The second assumption is that any abnormal response detected in a structure which could theoretically be responsible for the symptoms experienced is therefore suspected to be the source of the symptoms. The sports therapist needs to ensure that they have an appreciation of the wider pathological and biomechanical issues that can cause a structure to break down, not just identify which tissue has been damaged.

Table 2.5 Components of objective assessment

Objective assessment component	*Principles*
Observations	General observation of the patient in standing, seated or lying. Observing for skin presentations, muscle contours, asymmetries and other irregularities.
Static postural assessment	General impressions of the patient's musculoskeletal structure, spinal curves, shoulder and pelvic positioning and alignment of joints.
Active, passive and resisted physiological movements	Assessment of the patient's range of movement that includes active and passive ranges as well as active resisted strength, this may also include an analysis of the active and passive range of motion for the joints above and below the injured area (as in 'clearing').
Accessory movements	Movements that cannot be performed by the patient themselves but can be performed and assessed passively by the therapist.
Intrinsic biomechanical screen	A screening tool that takes a joint-by-joint approach to analyse movement dysfunctions.
Meaningful task analysis	The task that the patient functionally needs to be able to perform, but is unable due to symptoms.
Functional task analysis	Usually used as a screening tool to identify the patient's functional ability.
Muscle tests	The response and strength of muscles of the injured area using manual muscle testing. Muscle length tests aim to preferentially isolate and assess specific muscle flexibility.
Neurological tests	These include dermatomal, myotomal, reflex and neural tension testing. Cranial nerve testing may also be employed where applicable.
Palpation	A detailed assessment via touch and feel of the injured area. Involves palpation of the skin and subcutaneous tissues, anatomical landmarks, associated joint lines, ligaments, tendons and muscles.
Special tests	Used to try to confirm diagnosis of structures or the underlying condition that is causing symptoms.

While the sports therapist is guided to follow a reliable and sequential process of assessment, there is a certain clinical autonomy to the process. The patient whose subjective assessment leads the sports therapist to suspect (hypothesize) serious injury (such as a significant joint instability or stress fracture) may wish to prioritize certain stages of this objective assessment process. The prioritizing of stages during the objective assessment should help to confirm the hypothesis without unnecessarily aggravating the patient's symptoms.

The skilled therapist who has generated a comprehensive clinical impression from a detailed subjective assessment may also choose to prioritize the objective assessment in less severe injury presentations. This is a particularly useful skill in a team environment where the opportunity for a comprehensive initial consultation may not be available. The ability to quickly confirm the diagnosis hypothesis without discounting any serious injury or pathology allows the therapist to spend more time treating the patient and addressing the underlying causes.

Petty (2011) explains that prior to objective assessment, the therapist should have a firm plan for which tests to include, and also how they should be carried out. While many assessments will require the therapist to attempt to carefully reproduce patients' symptoms, an awareness of how severe and irritable symptoms are is crucial – in such cases it is important to not cause provocation. The sports therapist should also be mindful to perform fewer tests and to allow for rest periods between tests. Conversely, where symptoms are not so obviously severe or easily irritated, the therapist can consider utilizing combination movements, repetitive movements, and more functional or loaded movements to reproduce symptoms.

Observations

The first part of the objective assessment is the visual inspection; it is essential to conduct an efficient whole-body observational assessment, which then forms the basis of the overall evaluation of the patient. In the first instance, the body should be viewed as one entity as dysfunction at one segment can affect other parts through the biomechanical/kinetic chain. It is important that the sports therapist, possibly knowing prior to the visual inspection which body part is injured, does not isolate their inspection to that segment. The therapist may have formulated initial differential diagnoses from the subjective assessment, and the objective assessment will narrow these down, confirming or denying initial thoughts (Jones and Rivett, 2004). It is essential that the sports therapist remains open-minded at this stage. Observation can inform the therapist about certain specific factors which can then be further investigated throughout the physical assessment, including:

- information about the pathology itself;
- possible causes of the problem and therefore management options to correct it;
- information regarding symptom behaviour;
- information for selecting or requesting specific assessments/tests.

Following a whole-body (general) observational assessment (which should not normally take any longer than a minute or two), the injured area should be subjected to a more specific visual inspection, providing the therapist with a good initial insight into the problem in question. This is an inspection of the surface of the affected area observing for colour, contour and surface markings. It should be noted that aspects of this observation can be inadvertently or mistakenly influenced by the preceding history, leading to a less objective assessment than would be desired.

Observations begin from the moment the patient enters the clinical environment, and clues to problems can present, for example avoidance of certain movements when they are sitting in