Chapter 1

The responsibilities of being a physiotherapist

Ceri Sedgley

INTRODUCTION

This chapter provides an insight into what it means to be a physiotherapist and a member of the physiotherapy profession in the UK.

The chapter explores the development of the profession and how physiotherapy acquired the privileges and responsibilities of autonomous practice, and explores the consequences of that for contemporary professional practice.

Finally, the chapter considers how the changing shape of health services in the UK and society's increasing expectations of health professionals to deliver safe, high-quality health services within finite resources and which patients can trust are shaping physiotherapy practice. The ways in which physiotherapists can demonstrate the quality of both, practice and service delivery, through clinical governance, play vital role and this is also discussed.

The term patient has been used throughout this chapter to describe the individual to whom physiotherapy is being delivered. It is recognised that at times the term service user is more acceptable for some groups to whom physiotherapists provide intervention, e.g. in illness prevention. The term physiotherapist has been used throughout the chapter, but it is recognised that the chapter will also be of relevance to students and support workers, and others involved in delivering physiotherapy services.

BACKGROUND TO THE CHAPTER

Tidy's Physiotherapy has been a key text for physiotherapists over the years. Earlier editions have provided prescriptive descriptions of what physiotherapists should do in particular situations or for specific conditions. This prescriptive approach has become less relevant to the delivery of contemporary healthcare and recent editions of this chapter have demanded critical thinking from the reader. This reflects the development of the profession and the diversity of roles and settings physiotherapists deliver services within, both alone and within teams, providing healthcare for a diverse range of conditions. This change has been reflected in the education of physiotherapists focussing on developing analytical and clinical reasoning skills individualised to the patient. Recent editions of this chapter have, therefore, demanded critical thinking from the reader.

No two patients, clinical situations or professional roles are the same; each requires the physiotherapist to use their skills and knowledge to determine the most appropriate action. In a clinical situation, physiotherapists must use their skills and knowledge to carry out a full and accurate assessment and, using clinical reasoning skills and considering the individual patient, offer appropriate options for management. Throughout the decision-making process the patient should be educated and informed of the options available, and be given the opportunity to participate fully in their management. This includes consideration of the indication for managing the patient in physiotherapy, discharging them or referring them on. The responsibility for this decision-making process lies with the physiotherapist and the physiotherapist is accountable for this decision, hence the dichotomy of autonomy as both a privilege, i.e. the ability to act independently, and a responsibility, i.e. having accountability for the decisions made.

Accepting the responsibility requires maturity and an understanding of the implications of this responsibility. The individual physiotherapist must also understand the concept of scope of practice, competence, and the individual nature of scope (CSP 2008). An individual's scope will change throughout their career and competence

must be maintained through career-long learning, through self-evaluation of both the physiotherapist's learning needs and the service required, for example, maintaining currency with the most effective interventions. This commitment will maintain the trust of the patient and the public in both the individual and the profession.

HISTORY OF THE PHYSIOTHERAPY PROFESSION

This section provides an overview of the development of the physiotherapy profession with a particular focus on the development of autonomy and regulation of physiotherapy. An overview of the early days of the profession can be found in the book *In Good Hands* (Barclay 1994). Further references may be found in *The History of the Physiotherapy Profession* (CSP 2010), which provides an insight into the development of autonomy and, subsequently, scope of practice.

The Chartered Society of Physiotherapy (CSP) was founded in the UK in 1894, under the name of the Society of Trained Masseuses. It was established as a means of regulating the practice of 'medical rubbers'. For many years, doctors governed the profession and one of the first rules of professional conduct stated 'no massage to be undertaken except under medical direction' (Barclay 1994). The Society used the opportunities created by developments in medicine and technology, and the demands of war to extend its manual therapy skills, and to add exercise and movement, electrophysical modalities and other physical approaches to its repertoire during the early years of the twentieth century (Barclay 1994). This scope of practice, which was legitimised by a Royal Charter in 1920, remains the hallmark of contemporary physiotherapy practice (CSP 2008).

Physiotherapy continued to evolve and consolidate its position during the 1930s and 1940s. This was achieved through ongoing patronage of the medical profession and recognition of physiotherapy's contribution to society's health and well-being. The development of the Welfare State during the 1940s created opportunities for physiotherapy to apply and develop its practice across a growing range of medical specialisms (Barclay 1994). Physiotherapy training moved into hospital-based schools during 1948, which effectively meant that newly qualified physiotherapists were prepared for practice in National Health Service (NHS) hospitals. Over time, the NHS became the primary employer of physiotherapists.

Physiotherapy's quest for self-regulation during the 1950s was quashed by the medics who had effectively established control of its practice through sustained involvement in the CSP's governance structures and ongoing patronage. Following intense lobbying by physiotherapy and other healthcare professions, the Council of Professions Supplementary to Medicine (CPSM) opened a physiotherapy register in 1962 which represented a shift in the power of medicine over physiotherapy. Despite the introduction of state regulation, doctors continued to assert full responsibility for patients in their charge, arguing that 'professional and technical staff have no right to challenge his views; only he is equipped to decide how best to get the patients fit again' (Barclay 1994).

It took more than 80 years for the physiotherapy profession to progress from the paternalism of doctors, on whom physiotherapists were dependent for referrals. The first breakthrough came in the early 1970s, when a report by the Remedial Professions Committee, chaired by Professor Sir Ronald Tunbridge, included a statement that, while the doctor should retain responsibility for prescribing treatment, more scope in application and duration should be given to therapists. The McMillan report (DHSS 1973) went further, by recommending that therapists should be allowed to decide the nature and duration of treatment, although doctors would remain responsible for the patient's welfare. This recognised that doctors who referred patients would not be skilled in the detailed application of particular techniques, and that the therapist would therefore be able to operate more effectively if given greater responsibility and freedom.

Eventually, in the 1970s, a 'Health Circular, Relationship between the Medical and Remedial Professions' was issued (DHSS 1977). This acknowledged the therapist's competence and responsibility for deciding the nature of the treatment to be given. It recognised the ability of the physiotherapist to determine the most appropriate intervention for a patient, based on knowledge over and above that which it would be reasonable to expect a doctor to possess. It also recognised the close relationship between therapist and patient, and the importance of the therapist interpreting and adjusting treatment according to immediate patient responses, thus securing professional autonomy. This autonomy brought responsibilities and the ongoing need for physiotherapists to demonstrate competence in decision-making, building up the trust of doctors and those paying for physiotherapy services. This was reflected in the inclusion of skills of assessment and analysis as a key component of the qualifying curriculum introduced in 1974.

Two years after gaining professional autonomy in 1977, and supported by the shifts in physiotherapy education towards polytechnics, the CSP opened the debate on all-graduate entry – an identity traditionally associated with professions (Tidswell 1991). All-graduate entry was finally achieved in 1994 following considerable debate about how degree status would benefit patients and ensure the ongoing development of physiotherapy practice (Tidswell 2009).

In 1996 delegation of activities to healthcare practitioners, including some medical tasks, was facilitated by the document 'Central Consultants and Specialists

Committee: Towards tomorrow – The future role of the consultant' (Marriott 1996). The content of this report, together with the political drivers to contain healthcare service costs and maximise productivity, created new opportunities for physiotherapists to develop new skill-sets to undertake tasks that were previously the domain of medicine. These 'extended' roles were typically found in musculoskeletal medicine: physiotherapists working alongside doctors triaging patients on the waiting list or providing ongoing medical management of people with long-term conditions. Over time, these roles shifted into other medical specialisms, such as neurology, respiratory care and women's health – evidence of the clinical- and cost-effectiveness of this model of practice.

Towards the end of the 1990s, concerns about the quality of patient care, professional power and the need to contain the spiralling medico-legal costs, led to an overhaul of the regulatory frameworks in healthcare. Clinical governance was introduced as a system of quality control in 1997. Discussions about the need to review the regulation of professional groups like physiotherapy who worked alongside medicine, led to a change in terminology in 1999, from 'professions supplementary to medicine' to 'health professions'. Legal protection of the title 'physiotherapy' and 'physical therapist' followed under the Health Professions Order (DH 2002, HCPC 2001) – an outcome that the Chartered Society had been seeking for over 30 years. Alongside protection of title came a whole raft of changes designed to strengthen and modernise the regulation of healthcare professions, including physiotherapy. The CPSM was replaced by the Health Professions Council (HPC) in 2002 and subsequently renamed the Health and Care Professions Council (HCPC) in 2012. One of the most significant changes for registrants was the introduction of a process to audit their ongoing competence to practise and requiring engagement with continuing professional development (CPD) (HCPC 2011).

Once the physiotherapy profession had acquired all-graduate entry, physiotherapy continued its pursuit of professional traits by shifting the debate from examination of skills and techniques to attempting to identify the underpinning knowledge that makes it unique (Roberts 2001). This change is reflected in both the Physiotherapy Framework (CSP 2011c) and the Learning and Development Principles (CSP 2011b).

In 2007 the CSP Council agreed a fresh interpretation of the Royal Charter:

> *… the scope of practice is defined as any activity undertaken by an individual physiotherapist that may be situated within the four pillars of physiotherapy practice where the individual is educated, trained and competent to perform that activity. Such activities should be linked to existing or emerging occupational and/or practice frameworks acknowledged by the profession, and be supported by a body of evidence…*
>
> (CSP 2008)

Most recently, the CSP Council agreed to a new Code of Professional Values and Behaviour (CSP 2011a) that brings to the fore CSP member responsibilities relating to scope of practice, including the responsibility to consult with the CSP if a member is aware that a new area of practice challenges the boundaries of recognised scope of practice.

Physiotherapy has used the opportunities created by changes in society, developments in science and technology, and transformations in the design and delivery of education and healthcare, to evolve into what the profession is today.

RESPONSIBILITIES OF BEING A PROFESSIONAL

Since its inception in 1894, physiotherapy practice has been governed by a set of legal, regulatory and ethical frameworks and these are explored here. As described earlier, physiotherapists, as part of a profession, have certain rights or privileges together with a responsibility to themselves, the patient, the profession and the organisation within which they undertake their professional role. These responsibilities sit within legal, organisational and regulatory frameworks.

Characteristics of a profession

There are various theories on how to describe a profession in the literature. One way reflects work undertaken during the 1950s and 1960s which explored professions by identifying common traits and considering the qualities that distinguished a profession from an occupational group (Koehn 1994; Richardson 1999). A profession is described as:

- licensed by the state;
- a professional organisation which has developed and maintains a code of conduct or standards of practice based on acknowledged ethical principles;
- able to discipline members who contravene the code/standards;
- having exclusive knowledge and a technical base which is protected by the law;
- autonomous in its members' work;
- having members undertaking professional activity which requires them to have responsibilities or duties to those who need assistance;
- having responsibilities which are not incumbent on others.

By creating evidence of these traits, professions have been able to justify their ability to exercise power within society. As illustrated above, physiotherapy has sought to acquire the traits associated with a profession over time. From its inception in 1894 as an occupational group trained and examined in medical massage, physiotherapy has established a distinctive knowledge and skill-base that was first recognised by a charter in 1920, and more recently by achieving all-graduate entry – which also serves to ensure the maintenance and development of its unique knowledge and skills-base. The responsibilities of professional practice are expressed and regulated through standards which are regulated by the state.

Professionalism defines what is expected of a professional. Becoming an autonomous professional requires an acceptance, often implied, of certain responsibilities, in return for certain privileges. These responsibilities require behaviours and attitudes of individuals in whom professional trust is placed. Professionalism is widely understood to require these attributes (Medical Professionalism Project 2005 (cited in CSP 2005b); CSP 2011a):

- a motivation to deliver a service to others;
- adherence to a moral and ethical code of practice;
- striving for excellence;
- maintaining an awareness of limitations and scope of practice, and a commitment to empowering others (rather than seeking to protect professional knowledge and skills).

However, defining and providing evidence of professionalism is often more complex. A recent research report by the HCPC (2011) considered the concept of professionalism as many fitness to practise cases referred to regulators include professionalism. The report summarised that:

> *… professionalism has a basis in individual characteristics and values, but is also largely defined by context. Its definition varies with a number of factors, including organisational support, the workplace, the expectations of others, and the specifics of each service user/patient encounter. Regulations provide basic guidance and signposting on what is appropriate and what is unacceptable, but act as a baseline for behaviour, more than a specification…*

A profession that fulfils these expectations establishes and maintains credibility with the public and demonstrates its capacity to carry the privileges of professional practice – autonomy and self-regulation. In turn, fulfilment of these expectations demonstrates a profession's ability to fulfil the parallel responsibilities of professional practice – accountability, transparency and openness.

A key element of physiotherapy students' preparation for practice on qualification is their being supported in developing their understanding of, and engagement with, the responsibilities and privileges that professionalism encapsulates. The concept of professionalism also relates strongly to the role of physiotherapy support workers.

Possessing knowledge and skills not shared by others

Any profession possesses a range of specific knowledge and skills that are either unique or more significantly developed than in other professions. For physiotherapy, the roots of the profession can be found in massage. Physiotherapists continue to use massage therapeutically, as well as employing a wide range of other manual techniques, such as manipulation and reflex therapy. Therapeutic handling underpins many aspects of rehabilitation, requiring the touching of patients to facilitate movement, and the significance of therapeutic touching of patients still sets physiotherapy aside from other professions.

The World Congress for Physical Therapy (WCPT) states that:

> *Physical therapy provides services to individuals and populations to develop, maintain and restore maximum movement and functional ability throughout the lifespan. This includes providing services in circumstances where movement and function are threatened by ageing, injury, diseases, disorders, conditions or environmental factors. Functional movement is central to what it means to be healthy…*
>
> (WCPT 2011)

Cott et al. (1995) proposed an overarching framework for the profession: the movement continuum theory of physical therapy, arguing that the way in which physiotherapists conceptualise movement is what differentiates the profession from others. They suggest that physiotherapists conceive movement on a continuum from a micro- (molecular, cellular) to a macro- (the person in their environment or in society) level. The authors argue that the theory is a unique approach to movement rehabilitation because it incorporates knowledge of pathology with a holistic view of movement, which includes the influence of physical, social and psychological factors into an assessment of a person's maximum achievable movement potential. They argue that the role of physiotherapy is to minimise the difference between a person's current movement capability and his/her preferred movement capability.

In the UK, one approach to conceptualising physiotherapy is to consider physiotherapy, as defined by the Royal Charter, as the four pillars of practice of:

- massage;
- exercise and movement;
- electrotherapy;
- kindred methods of treatment (CSP 2008).

The acquisition of these knowledge, skills and attributes from qualifying programmes, and subsequently on qualification through a range of learning activities, may be used by physiotherapists to benefit people in a range of specialties or patient groups, for example elite athletes, older people, people with developmental or acquired conditions, or people with mental health problems. A recent definition of the Physiotherapy Framework states that:

> *Physiotherapy is a healthcare profession that works with people to identify and maximise their ability to move and function. Functional movement is a key part of what it means to be healthy. This means that physiotherapy plays a key role in enabling people to improve their health, wellbeing and quality of life.*
>
> (CSP 2011c)

Autonomy

Autonomy, or 'personal freedom', is a key characteristic of being a professional. Professional autonomy is the application of the principle of autonomy whereby a professional makes decisions and acts independently within a professional context and is responsible and accountable for these decisions and actions. Thus, it is both a privilege and a responsibility allowing independence whilst mirrored by responsibility and accountability for action.

Central to the practice of professional autonomy is clinical reasoning, described as the 'thinking and decision-making processes associated with clinical practice' (Higgs and Jones 1995).

Clinical reasoning requires the ability to think critically about practice, to learn from experience and apply that learning to future situations. It is the relationship between the physiotherapist's knowledge, his or her ability to collect, analyse and synthesise relevant information (cognition), and personal awareness, self-monitoring and reflective processes, or metacognition (Jones et al. 2000). A key element of professional autonomy is for a physiotherapist to understand and work within the limits of their personal competence and scope of practice. Physiotherapists are responsible for seeking advice and guidance to inform decision-making and action from others through appropriate forms of professional supervision and mentorship.

Professional autonomy has to be balanced with the autonomy patients have to make their own decisions, that is, patient autonomy. It is the responsibility of a professional to understand and facilitate this. Patient-centred decisions require a partnership between patient and professional, sharing information, with the treatment of patients' values and experience as equally important as clinical knowledge and scientific facts (Ersser and Atkins 2000).

Neither physiotherapy students nor support workers hold professional autonomy. Both groups undertake physiotherapy-related activity with appropriate forms of supervision. The qualifying programme that physiotherapy students undertake prepares them for the responsibilities of professional autonomy on qualification. This preparation includes developing the knowledge, skills, understanding and attributes necessary to accept this responsibility. Although not autonomous practitioners, physiotherapy support workers assume responsibility for undertaking the tasks delegated to them in delivering a physiotherapy service.

Person-centred practice

The professional is characterised as a person with specialised knowledge that can be shared with the patient in a reciprocal 'working with' rather than 'doing to' relationship, and as someone who 'accompanies the patient on their journey towards health, adjustment, coping or death'. Higgs and Titchen (2001) describe the notion of the professional's role as a 'skilled companion'. This patient-centred model facilitates the sharing of power and responsibility between both professional and patient.

Person-centred practice is an approach to healthcare within which the goals, expectations, preferences, capacity and needs of individuals (patients, clients, service users) and their carers are central to all decision making and activity. There needs to be an open partnership between the physiotherapist and the patient, and an acceptance and understanding that, at times, the view of an individual will conflict with the view of the physiotherapist, the profession or the organisation within which a service is being delivered. Furthermore, individual patients will vary as to the degree to which they intend to exercise their autonomy and the physiotherapist may be required to advocate for them on their behalf.

Examples of person-centred practice include ensuring that an individual's perspective is listened to and reflected at all points of intervention and service delivery; ensuring an individual is fully involved in planning, engaging and evaluating their experience and the outcomes of physiotherapy; and actively seeking user involvement to inform how a service is developed and delivered to maximise its effectiveness.

Making a commitment to assist those in need

As stated earlier, one of the characteristics of a professional is to want to 'do good'. This is reflected in the ethical principles of the physiotherapy profession, where there is a 'duty of care' incumbent on the physiotherapist towards the patient, to ensure that the therapeutic intervention is intended to be of benefit. This is a common-law duty, a

breach of which (negligence) could lead to a civil claim for damages.

More generally, Koehn (1994) suggests professionals are perceived to have moral authority, or trustworthiness if they:

- use their skills in the context of the client's best interests and 'doing good';
- are willing to act for as long as it takes to achieve what was set out to be achieved or for a decision to be made that nothing more can be done to help the client;
- have a highly developed internalised sense of responsibility to monitor personal behaviour, for example by not taking advantage of vulnerable patients;
- demand from the client the responsibility to provide, for example, sufficient information to allow decisions to be made (compliance);
- are allowed to exercise discretion (judgement) to do the best for the client, within limits.

Principle 1 of the Code (CSP 2011a) requires that members demonstrate appropriate professional autonomy and accountability. In doing so members are expected to:

1.1.1. Use their professional autonomy to benefit others;
1.1.2. Understand and accept the significant responsibility that professional autonomy brings;
1.1.3. Accept and uphold their duty of care to individuals;
1.1.4. Are responsible and accountable for their decisions and actions, including the delegation of activity to others;
1.1.5. Justify and account for their decisions and actions;
1.1.6. Ensure that their activity is covered by appropriate insurance.

Scope of practice

As a professional body, the CSP defines the scope of practice for physiotherapy in the UK. In doing so it recognises that UK physiotherapy is diverse, and 'requires a dynamic, evolving approach to scope to ensure the profession is responsive to changing patient and population needs and that its practice is shaped by developments in the evidence base'. In taking this approach it 'enables the profession to initiate, lead and respond to changes in service design and delivery, and to optimise opportunities for professional and career development, while being sensitive to the roles and activities of other professions and occupational groups' (CSP 2011c). Scope of practice relates strongly to competence and professionalism.

This concept of scope recognises that:

- the profession's scope of practice is evolving, and needs to evolve, in line with changing patient and population needs, developments in the evidence base, changes in service design and delivery, and changing opportunities for professional and career development;
- practice includes a diversity of activity that is shaped by the collective, shared principles and thinking of the profession;
- individuals have a responsibility to limit their activity to those areas in which they have established and maintained their competence;
- individuals need to evaluate and reflect on their personal activity, taking account of the profession's evolving evidence base, and respond appropriately to their learning and development needs;
- individual competence changes and shifts as they progress through their physiotherapy career;
- individuals have a responsibility to be aware of how their practice may challenge the boundaries of the scope of practice of UK physiotherapy and to take appropriate action (CSP 2011d).

Every physiotherapist has her or his own personal 'scope of practice' (CSP 2011c) – that is, a range (or scope) of professional knowledge and skills that can be applied competently within specific practice settings or populations. When a person is newly qualified, this scope will be based on the content of the pre-qualifying course, but will also be informed by the individual's experience in clinical placements and the amount of teaching and reflective learning that has been possible as part of those placements. As a career progresses, and as a result of CPD and personal interest, these skills and knowledge evolve with a physiotherapist developing some skills, adding new skills and possibly losing competence in some areas. It is the responsibility of the professional to understand his or her personal scope of practice as it changes and evolves throughout their career. To practise in areas in which a physiotherapist is not competent puts patients at risk and is a breach of the HCPC standards (HCPC 2007, HCPC 2008).

For example, some physiotherapists will become competent in highly skilled areas such as intensive care procedures or splinting for children with cerebral palsy, which are unlikely to have been taught prior to qualification. Others will extend their skills in areas in which they already had some experience, for example in the management of neurological conditions. Others will extend their scope to become experts in a specific clinical area and advance their skills of clinical reasoning by participating in research, teaching or management of complex conditions, or undertaking clinical specialist, advanced practice or consultant roles (CSP 2002d).

Competence

Competence is the synthesis of knowledge, skills, values, behaviours and attributes that enable physiotherapists to work safely, effectively and legally within their particular scope of practice at any point in time (CSP 2011a).

Competence changes as a physiotherapist progresses through their career and relates to an individual's professional and life experiences, learning from reading, from evaluating practice and from reflecting on practice, or through more formal ways of learning. Competence in some areas will increase while competence in others will decrease or be lost. To maintain competence a physiotherapist must engage in structured, career-long learning and development to meet their identified learning needs.

Physiotherapists have a duty to keep up to date with new information generated by research, with what their peers are thinking and doing, and by formally evaluating the outcome of their practice. The responsibility for this is dictated by the HCPC (2008) and reflected in the Quality Assurance Standards for Physiotherapy Service Delivery (CSP 2012). For example, Section 3 Learning and Development includes a number of Standards including Standard 3.1 Members actively engage with and reflect on the continuing professional development (CPD) process to maintain and develop their competence to practise.

Responsibility to patients

This chapter has already discussed the importance of the individual physiotherapist, as well as the profession as a whole in maintaining the attributes of professionals. Koehn (1994) argues that trustworthiness is what stands out as a particularly unique characteristic of being a professional – to do good, to have the patient's best interests at heart and to have high ethical standards.

Trust is, perhaps, the most essential characteristic with which to develop a sense of partnership with patients that, in turn, will optimise the benefits of intervention. For physiotherapy, many of the other hallmarks for building and securing trust are set out in the QA standards (CSP 2012).

Responsibility to those who pay for services

Physiotherapists have an ethical responsibility to payers of services, whether these are commissioners of healthcare, purchasers of services, taxpayers or individual patients, to provide efficiently delivered, clinically- and cost-effective interventions and services in order to provide value in an era when resources for healthcare are limited.

Responsibility to colleagues and the profession

A profession has legitimate expectations of its members to conduct themselves in a way that does not bring the profession into disrepute, but rather enhances public perception of it. Physiotherapists have a duty to inform themselves of what is expected of them. Indeed, the expectation of the CSP is that members adhere to the Code (CSP 2011a), and this commitment forms part of the contract of membership of the CSP. Similarly, the CSP expects that all members should meet the Quality Assurance Standards for Physiotherapy Service Delivery (CSP 2012). Where they do not, programmes of professional development should be put in place to facilitate full compliance, as part of the individual's professional responsibility.

Physiotherapists are encouraged to be proactive in supporting each other's professional development and in promoting the value of the profession in local workplace settings, in policy-making forums and in the media. Physiotherapists should not be critical of each other except in extreme circumstances. However, they do have a duty to report circumstances that could put patients at risk. In the NHS there are procedures and a nominated officer within each trust from whom advice can be sought. Outside the NHS, advice can be sought from the CSP.

BELONGING TO A PROFESSION

Regulation: The Health and Care Professions Council (HCPC)

The Health Professions Council (HPC) was created by the Health Professions Order 2001 (HCPC 2001) as the statutory regulator of 13 professions, including physiotherapy. The regulatory process is a government measure to protect patients and the public from unqualified or inadequately skilled healthcare providers. As the number of professions increased to 17, it was renamed the Health and Care Professions Council (HCPC) in 2012 to reflect the diversity of the professions regulated.

In the UK, the titles 'physiotherapist' and 'physical therapist' are protected and only physiotherapists registered (registrants) with the HCPC may call themselves a physiotherapist or physical therapist. As the title is protected, a physiotherapist listed does not, therefore, need to place HCPC after their name as this is implicit within the title.

The HCPC sets standards of professional training, performance and conduct for the 17 regulated professions and maintains a public register of health professionals that meet its standards. The HCPC publishes generic standards for HCPC registrants, standards of conduct, performance and ethics (HCPC 2008) and profession-specific standards (Standards of Proficiency Physiotherapists, HCPC 2007), which members are required to and agree to meet. Registrants are required to keep up to date with the processes and requirements decreed by the HCPC. The HCPC only regulates the practice relating to humans and does not include regulation of physiotherapists practising on animals.

In 2006, the HCPC put in place a system requiring re-registration at intervals of two years (HCPC 2011). Re-registration was introduced partly in response to a lessening of public confidence in the NHS following, for example, the report into children's heart surgery in Bristol (Bristol Royal Infirmary Inquiry 2001). Equally disturbing were the revelations about the murders of so many patients by Harold Shipman, a man who had previously been a trusted general practitioner (GP), where health systems failed to detect an unusually high number of deaths (DH 2004).

These measures demonstrate a commitment to protecting the public through more explicit and independent processes (DH 2002). The re-registration process is linked to an individual's commitment to continuous professional development (CPD), whereby individuals must undertake and maintain a record of their CPD activities and, if required, submit evidence of this and its outcomes to their practice, service users and service. The process of re-registration aims to identify poor performers who may be putting the public at risk, as well as providing an incentive for professionals to keep up to date, maintaining and further developing their scope of practise and competence to practise.

The HCPC takes action when complaints are received and if registered health professionals, including physiotherapists, do not meet these standards (HCPC 2005). The process of registration and the accessible public register provide assurance to the public that a physiotherapist is legally allowed to practise. Disciplinary processes are in place to ultimately remove an individual from the register (HCPC 2005) where necessary.

While the principles of professionalism should be aspired to by physiotherapists anywhere in the world, the existence and/or role of regulators and professional bodies in the locations of practice when outside the UK may vary depending on political, social and financial factors.

Professional membership: the Chartered Society of Physiotherapy (CSP)

The CSP is the professional body and therefore the primary holder and shaper of physiotherapy practice. As such, it is the guardian of the profession's body of knowledge and skills and a number of activities emanate from this. The CSP works on behalf of the profession to protect the chartered status of physiotherapists' standing, which is one denoting excellence. The CSP provides a breadth of support and resources to support members in their working lives whereby its education and professional activity is centred on leading and supporting members' delivery of high-quality, evidence-based patient care and establishing a level of excellence for the profession.

As the guardian of the profession's body of knowledge and skills in the UK, the CSP aims to:

- uphold the credibility, values and high standards of the UK physiotherapy profession;
- ensure new areas of physiotherapy practice draw on the profession's distinctive body of knowledge and skills, and uphold a physiotherapist's accountability for their decision-making and actions;
- enhance the profession's contribution across the UK health and well-being economy;
- optimise the profession's ongoing development;
- ensure the profession's movement into a new area of practice is in the interests of the population and patient groups that it serves (or can potentially serve), while being sensitive to the roles and activities of other professions and occupational groups;
- ensure that the profession's decision to recognise a particular area of practice can be explained and justified in terms of that area's safety, effectiveness and efficacy;
- maintain a record of how the UK physiotherapy profession practice has evolved (CSP 2008).

The relationship between the HCPC and the CSP is essential and, although registration with the HCPC enables a registrant to call themselves a physiotherapist, it is only those physiotherapists who are members of the CSP who may call themselves a chartered physiotherapist and use the letters MCSP.

The CSP continues to handle complaints or consider matters of fitness to practise concerning members of the Society who are not regulated by the HCPC, including physiotherapists treating animals, students and the CSP's associate members. The CSP also holds a disciplinary function to those members who are registrants of the HCPC.

Code of professional values and behaviour

In 2011, the CSP's Council approved the new CSP Code of Professional Values and Behaviour (CSP 2011a). A condition of membership is that all members – qualified members, students and associate members – must agree to meet the Code. The Code defines the values and behaviour that the CSP expects of its members and that underpin their physiotherapy roles and activity. It has been developed to support members in taking responsibility for their actions and to promote their professionalism. Throughout, it reinforces the need for members to meet the requirements of regulation, the law, and their employing organisations and education institutions.

The principles are that:

1. members take responsibility for their actions;
2. members behave ethically;
3. members deliver an effective service;
4. members strive to achieve excellence (CSP 2011a).

Each of these principles is expanded on in sets of layered statements and underpinned by healthcare ethics, values and professional concepts. The Rules of Professional Conduct (the Rules) (CSP 2002a) were endorsed at the very first council meeting of the CSP in 1895 (Barclay 1994). They have been revised and updated at intervals since and have now been superseded by the Code in setting out the expectations of the CSP. The Rules defined the professional behaviour expected of chartered physiotherapists and were founded to safeguard patients. However, they remain for the purpose of supporting the byelaws and taking action against those members who are not regulated by the HPC; that is, students, assistants (associate members) and qualified members practising on animals.

Quality Assurance Standards for Physiotherapy Service Delivery

The CSP initially agreed and published national standards in 1990 which were subsequently revised in 2000 and 2005 (CSP 2005a). The standards underwent a significant revision in 2012 (CSP 2012) to reflect contemporary healthcare and the increasing expectations of the public to be active partners in their healthcare. The standards place greater emphasis on the role of the standards in quality assurance, the integration of members' roles in clinical practice and service delivery and their application to all members of the CSP: physiotherapists, students and associate members.

The CSP QA standards include two resources: the Quality Assurance Standards for Physiotherapy Service Delivery and the Quality Assurance Audit tool (CSP 2012). They are grouped into 10 sections with each section including a number of standards. Each standard then includes a number of measurable criteria which identify how the standards may be met. These measurable criteria enable the comparison of actual performance with the standard through clinical audit.

The standards are organised into 10 sections and provide detailed statements which support members in meeting the expectations of the CSP articulated in the Code and, for physiotherapists, the standards of the HCPC. Patients and service users accessing these have detailed information on the standards of physiotherapy service delivery they can expect.

Physiotherapy Framework

The Physiotherapy Framework (CSP 2011c) has been designed to promote and develop physiotherapy practice and complements the Code, CSP standards and HCPC standards.

The framework defines and illustrates the knowledge, skills, behaviour and values required for contemporary physiotherapy practice: describing physiotherapy practice:

- at all levels – from a new support worker through to a senior level registered physiotherapist;
- across a variety of occupational roles – clinical, educational, leadership, managerial, research and support;
- in a variety of settings – in health and social care, in industry and workplaces, in education and development, and in research environments;
- across all four nations of the UK.

The framework supports CSP members' professional practice in a number of different ways and demonstrates how physiotherapy works to maximise individuals' potential – through its clinical, educational, leadership and research practice.

Physiotherapy education programmes

The CSP provides a set of principles on which physiotherapy qualifying programmes should be based in order to obtain CSP accreditation. These principles are intended to help course providers develop their programmes to prepare their learners for current and emerging physiotherapy roles that meet changing healthcare needs and for practice within an evolving context. For example, Principle 1, Programme Outcomes Qualifying, identifies that 'programmes should aim to develop the knowledge, skills, behaviour and values (KSBV) required to practise physiotherapy at newly qualified level (NHS Band 5 or equivalent), while nurturing the skills, behaviour and values that will enhance career-long development and practice' (CSP 2011c).

Physiotherapy programmes must meet these requirements to be approved by the CSP on behalf of the profession. Physiotherapists completing an approved programme are eligible for membership of the CSP as chartered physiotherapists and, as members, may use the letters MCSP. The Learning and Development Principles for CSP Accreditation of Qualifying Programmes in Physiotherapy 2011 (CSP 2011b) replace the Curriculum Framework for Qualifying Programmes in Physiotherapy published in 2002 (CSP 2002b).

DELIVERING SAFE AND EFFECTIVE PHYSIOTHERAPY SERVICES: CLINICAL GOVERNANCE

So far, this chapter has explored the responsibilities of being a physiotherapist from a professional perspective with the focus on the individual's personal responsibility

ABC Definition

Clinical governance is a framework through which NHS organisations are accountable for continuously improving the quality of their services and safeguarding high standards of care by creating an environment in which excellence in clinical care will flourish (Secretary of State for Health 1998).

[While this definition has been used in England, similar interpretations of the term have been made in Scotland, Wales and Northern Ireland.]

as a professional. This section describes the context of a professional's responsibilities to the organisation within which they undertake their professional role, whether in the public or independent sector.

Clinical governance provides a framework for ensuring the safety, quality and effectiveness of services. Although in the NHS, for example, clinical governance is the responsibility of NHS trusts, its foundation, based on 'the principle that health professionals must be responsible and accountable for their own practice' (Secretary of State for Health 1998) is relevant regardless of the sector where services are delivered and clinical governance processes are in place throughout health and social care.

It can be argued that clinical governance is, at least in part, a response to the loss of public confidence in the NHS, as discussed earlier, which has undermined public perceptions of the NHS as an organisation they can rely on to 'do good' and of the government to protect the public. Clinical governance has a key role in assuring and re-building the public's confidence in health services, providing high-quality and effective care and, above all, reducing the risk of harm through negligence, poor performance or system failures.

A number of key themes were introduced as part of clinical governance:

- the accountability and statutory responsibility of chief executives for quality;
- the introduction of a philosophy of continuous improvement;
- emphasis on an integrated approach to healthcare whereby the whole system of care is examined crossing professions, departments, organisations and sectors to ensure the whole process meets the needs of patients;
- an aspiration to achieve consistency of services across the NHS;
- an emphasis on CPD and life-long learning (LLL) for all healthcare workers in order to keep up to date and deliver high-quality services.

EG Examples of activities within a clinical governance programme

- Ensure physiotherapists are on the HCPC register.
- Consider and learn from patient feedback and complaints.
- Carry out programmes for quality improvement, including clinical audit and evaluation, and report how these have led to improvements for patients.
- Ensure that nationally-produced, high-quality standards and clinical guidelines are implemented locally.
- Have an appropriate skill mix and staffing level to ensure the safety of patients, making appropriate use of human and financial resources in order to provide effective care.
- Have a process for identifying and supporting staff members whose competence is in question.
- Provide an in-service training programme and time for individual CPD activities.
- Ensure appropriate participation in multi-professional clinical audit and quality improvement activities.

Clinical governance should be considered as a package of measures which include:

- openness;
- risk management;
- clinical effectiveness including evidence-based practice, standards and guidelines;
- research and development;
- clinical audit and evaluating the effectiveness and quality of services;
- information management;
- education and training including continuing professional development/life-long learning to ensure the skill mix is appropriate.

Those responsible for physiotherapy service delivery, for example, physiotherapy managers, are responsible for devising, implementing and reporting a departmental clinical governance programme, which should reflect all the aspects of clinical governance. Physiotherapists should play an active part in contributing to physiotherapy clinical governance programmes and also participate in relevant multi-professional clinical governance activities such as clinical audit or local protocol/clinical pathway design.

Evidence-based practice

There is information to show that evidence-based medicine was in place as long ago as the 1940s, and it was Professor Archie Cochrane in 1972 who first introduced

the concept of evidence-based medicine (Cochrane 1972). However, until the early 1990s 'evidence' was largely based on personal experience and opinions derived from that experience, together with the experience of colleagues or those perceived to be experts and opinion leaders.

In 1991, Sir Michael Peckham, then Director of Research and Development for the Department of Health (DoH at the time; changed to DH in 2004), noted that 'strongly held views based on belief rather than sound information still exert too much influence in healthcare. In some instances the relevant knowledge is available but is not being used; in other situations additional knowledge needs to be generated from reliable sources' (DH 1991). At about the same time, a relatively small group of doctors began to write about evidence-based medicine.

ABC Definition

An early definition of evidence-based medicine stated that it is the 'conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients' (Sackett et al. 1996).

A more recent definition of evidence-based practice is 'decisions about healthcare are based on the best available, current, valid and relevant evidence. These decisions should be made by those receiving care, informed by the tacit and explicit knowledge of those providing care, within the context of available resources' (Dawes et al. 2005).

The CSP QA standards state the following within Section 8 Physiotherapy management and treatment:

- **8.2.** There is a system to ensure that physiotherapy care is based on the best available evidence of effectiveness.
- **8.5.** Appropriate treatment options are identified based on the best available evidence, in order to deliver effective care (CSP 2012).

A range of evidence is available to inform practice and a hierarchy of evidence is often described or used in the literature. This ranges from (Ia) systematic reviews, in which evidence on a topic has been systematically identified, appraised and summarised according to predetermined criteria (usually limited to randomised controlled trials (RCTs)) – said to be the strongest evidence (the most reliable estimate of effectiveness) – to (IV) expert opinion, perceived as the least reliable. An example is shown in Table 1.1.

Research takes many forms and is not the only form of evidence. For some questions, such as the efficacy of particular drugs, or a particular modality, such as exercise programmes for the management of back pain, research studies that compare one intervention with another or a placebo (RCTs) can provide reliable information about the degree to which an intervention is effective. But other forms of evidence are also important (Figure 1.1). What patients tell us about their condition, which treatments they find effective and the degree to which interventions improve their ability to get on with their lives also provide important evidence. The physiotherapist also contributes to evidence in the form of clinical expertise, derived from clinical reasoning experience. Thinking and reflecting during or after a clinical encounter will develop such expertise (Jones et al. 2000). Knowledge which arises from and within practice (practice-based and practice-generated knowledge) will become part, along with research evidence, of your rationale for practice (Higgs and Titchen 2001). Sackett and colleagues reflected this in concluding their definition that evidence-based practice requires integration of 'clinical expertise with best available external clinical evidence from systematic research' (Sackett et al. 1996).

The hierarchy does not, therefore, recognise that different research methods are needed to answer different types of questions and that, while a qualitative study may be the best research method for a particular question, it still receives a low rating. It also fails to recognise the importance of expertise derived from clinical reasoning experience, discussed above. Physiotherapists should consider contributing to an ongoing debate to develop a hierarchy that reflects more appropriately a patient-centred approach to identifying the relevant evidence.

Table 1.1 A hierarchy of evidence

Level	Type of evidence
Ia	Evidence obtained from a systematic review or meta-analysis of RCTs
Ib	Evidence obtained from at least one RCT
IIa	Evidence obtained from at least one well-designed controlled study without randomisation
IIb	Evidence obtained from at least one other type of well-designed, quasi-experimental study
III	Evidence obtained from well-designed, non-experimental descriptive studies, such as comparative studies, correlation studies and case studies
IV	Evidence obtained from expert committee reports or opinions and/or clinical experience of respected authorities

Adapted from NICE (2001).

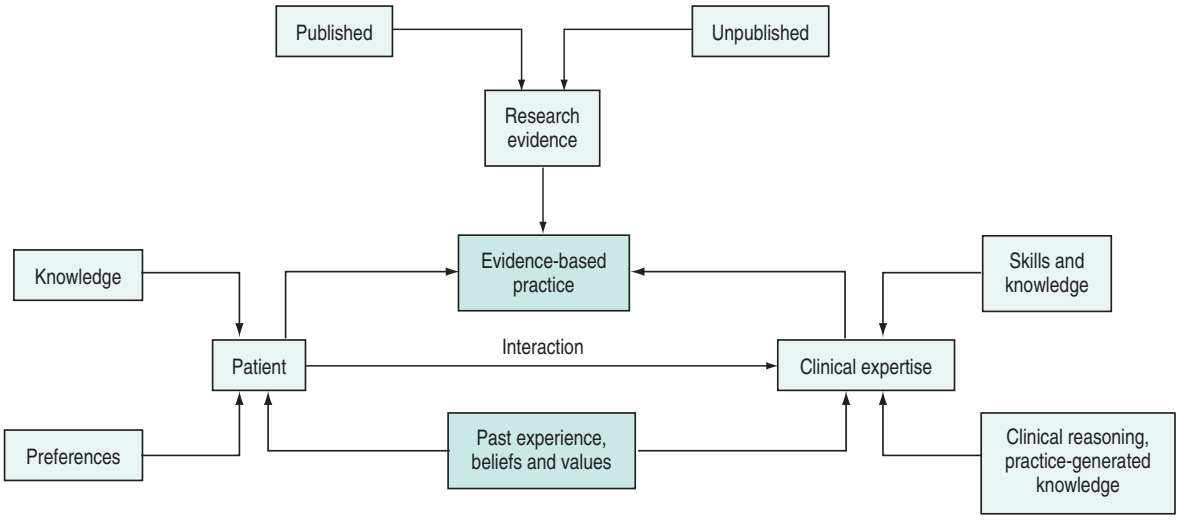


Figure 1.1 What do we mean by 'evidence'? (*Adapted from Bury (1998), with permission.)*

The following steps will be useful in identifying and using research evidence:

- Consider the clinical question for which an answer is sought in the information search. Identify the population (e.g. people with multiple sclerosis with symptoms of urinary incontinence), the intervention (e.g. neuromuscular electrical stimulation) and the outcome sought (e.g. a reduction in symptoms), and use this information to formulate a search strategy.
- Work in partnership with an information scientist to get the best results from a literature search (their information skills and knowledge combined with your clinical skills and knowledge).
- Look first for evidence that has already been synthesised – systematic reviews, nationally-developed clinical guidelines or standards. This will save time looking for individual studies; if it is a high-quality synthesis, it will also provide a more reliable estimate of effectiveness.
- Know the databases well enough to know which will have the most relevant information for any particular topic.
- Check the titles and abstracts for relevance.
- Critically appraise any relevant papers found to assure their quality and the reliability of their conclusions (a list of appraisal instruments can be found at the end of this chapter).
- When the 'best available evidence' has been found, consider it in relation to the patient and past experience. Is it appropriate for that patient? Can the degree of likely benefits and harms (if any) be quantified?
- Discuss the evidence with the patient and agree together the preferred intervention(s).
- Implement the preferred intervention(s).
- Evaluate the effect of the intervention(s) and act accordingly.

More information about evidence-based practice can be found in Herbert et al. (2005) or at www.nettingtheevidence.org.uk, a catalogue of useful electronic learning resources and links to organisations that facilitates evidence-based healthcare. See also 'Sources of Critical Appraisal Tools' towards the end of this chapter.

Clinical effectiveness

Clinical effectiveness, as defined by the DH, sounds very much like evidence-based practice – doing things known to be effective for a particular patient or group of patients. But the fact that an intervention has been shown to work in research studies, in a relatively controlled environment, does not necessarily mean that it will work for a particular patient. Both patients and practitioners are unique beings and there are many additional factors, practical and behavioural, that need to be considered to ensure the patient gets the maximum benefit from an intervention.

While evidence-based practice is a key component of clinical effectiveness, clinical effectiveness also takes account of a range of other influences that could affect the patient's ability to benefit from an intervention based on high-quality research evidence.

ABC Definition

Clinical effectiveness was defined by the DH in 1996 as 'the extent to which specific clinical interventions, when deployed in the field for a particular patient or population, do what they are intended to do – that is, maintain and improve health and secure the greatest possible health gain from the available resources' (NHS Executive 1996).

The following questions will give an insight into the patient's ability to benefit from an intervention. The questions serve to illustrate the complexity of the clinical reasoning process, where highly skilled judgements have to be made based on consideration of the whole person, physically, emotionally and within society, as well as the environment, practitioner skills and resources available in order to provide truly effective physiotherapy management and treatment.

- Who should manage the patient? Is physiotherapy the most relevant approach and, if so, who should treat the patient? Does the individual physiotherapist have the skills and knowledge?
- Why is physiotherapy or a specific intervention indicated or identified?
- What is the most appropriate intervention?
- Which approach is the most appropriate? Does this consider the patient as an individual in terms of lifestyle? Does patient have an opportunity to fully describe the symptoms and the impact of the problem on the person's life, and to ask questions? Does the patient have enough information to be able to give consent? Are other options discussed, that may be more acceptable to the patient, even if less effective?
- Where should the patient be managed? Is the setting for treatment most appropriate, e.g. treatment in a hospital setting may mean a long, exhausting and expensive journey for the patient?
- When should intervention begin? Does the patient have adequate privacy, warmth and comfort during treatment? How long does the patient have to wait for treatment – will any delay affect the effectiveness of the interventions?

Standards

One of the tenets of clinical governance is consistency – for the public, being confident that they will experience the same quality of care regardless of where it is delivered. The availability of high-quality national standards, the use of these locally and the evaluation of their implementation, should ensure an improvement in the standards of service delivery, by identifying where actual services are falling short of the standards and action needs to be taken. Physiotherapists have a key role in implementing standards in the services they provide to the relevant patient population. Implementation will also provide opportunities to promote the value of physiotherapy to this patient population and highlight the contribution physiotherapists can make to a trust's compliance with this particular standard. Two examples of these are set out below.

Nationally-developed standards

The CSP's Quality Assurance Standards for Physiotherapy Service Delivery (CSP 2012) are one example of nationally-developed standards. Others are derived from nationally-agreed guidelines or guidance documents, for example those produced by CSP Professional Networks. There is some evidence to suggest that nationally-developed standards or clinical guidelines are likely to be more robustly developed (Sudlow and Thomson 1997) than those developed locally, and that their universal implementation locally will ensure consistency and effectiveness.

National Service Frameworks

National Service Frameworks (NSFs) and strategies are a government initiative which are based on the best available evidence of what treatments and services work most effectively for patients.

The NSFs aim to address the 'whole system of care' and each sets out where care is best provided and the standard of care that patients should be offered in each setting. They provide 'a clear set of priorities against which local action can be framed' and seek to ensure that patients will get greater consistency in the availability and quality of services, right across the NHS (Secretary of State for Health 1998).

Each strategy is developed in partnership with health professionals, patients, carers, health service mangers, voluntary agencies and other experts. They set clear quality requirements for care including explicit standards and principles for the pattern and level of services required for a specific service or care group. A range of NSFs has been developed, for example Cancer Strategy, Framework for Coronary Heart Disease, National Strategy for COPD (chronic obstructive pulmonary disease), Diabetes Quality Standards, Setting Standards for Kidney Care, Framework for Long-term Conditions, Mental Health Strategy, Framework for Older People and Standards for Stroke Care.

Clinical guidelines

ABC Definition

Clinical guidelines are 'systematically developed statements to assist practitioner and patient decisions about appropriate healthcare for specific circumstances' (Field and Lohr 1992).

The key factors in the development of clinical guidelines are the systematic process for identifying and quality-assessing research evidence, and the systematic and transparent process used for the interpretation of the evidence in the context of clinical practice, in order to formulate reliable recommendations for practice. A number of examples are provided below.

CSP endorsed guidelines

The physiotherapy profession has developed a number of national, physiotherapy-specific clinical guidelines. For users of clinical guidelines, CSP-endorsed clinical guidelines can be considered of high quality and should be implemented locally. To ensure quality and provide confidence for users and in response to guidelines submitted to the CSP, the CSP has established a process for the endorsement of these clinical guidelines which is part of the programme Supporting Knowledge in Physiotherapy Practice (SKIPP). The criteria for assessing whether the quality of a guideline warrants CSP endorsement can be found in an appraisal questionnaire developed by an European consortium known as the AGREE instrument (AGREE: www.agreetrust.org). Further information about the process for the development of clinical guidelines in physiotherapy is available from the CSP website (www.csp.org.uk).

National Institute for Health and Clinical Excellence (NICE)

NICE is an independent organisation established by the government in 1999 to provide health and social care professionals and the public in England and Wales with information to promote good health and prevent ill-health, together with information about the clinical- and cost-effectiveness of healthcare.

NICE provides evidence-based national guidance for the NHS, local authorities, charities and those with a responsibility for commissioning or providing healthcare, public health or social services with the aim of promoting quality of care. NICE also provides support and a range of resources for putting this into practice.

The DH and the Assembly for Wales have given NICE the remit for developing 'robust and authoritative' clinical guidelines, taking into account clinical and cost-effectiveness (NICE 2001). More information about the key principles that underpin the way NICE approaches clinical guideline development can be found on its website (www.nice.org.uk). One of its work programmes is to develop clinical guidelines, which is carried out by a series of collaborating centres.

Scottish Intercollegiate Guidelines Network

The Scottish Intercollegiate Guidelines Network (SIGN) was formed in 1993. Its objective is to improve the quality of healthcare for patients in Scotland by reducing variation in practice and outcome through the development and dissemination of national clinical guidelines. These guidelines contain recommendations for effective practice based on current evidence. Further information can be found on its website (www.sign.ac.uk).

Care Quality Commission (CQC)

The Care Quality Commission (CQC) is the independent regulator of health and social care in the UK. It replaced the Healthcare Commission in 2009. This statutory body was established to raise standards of care provided by the NHS, local authorities, private companies and voluntary organisations throughout England and Wales, and defines its role as 'Checking that hospitals, care homes and care services are meeting our standards'.

The CQC has the role of assessing the implementation of clinical governance in every NHS trust and making its findings public. Teams of trained reviewers visit trusts (and can be called in at any time should concerns be raised) to review trust information and data, talk to staff and patients, and consider the trust's performance in specified categories. In Scotland a similar function is provided by NHS Quality Improvement Scotland and in Northern Ireland the Health and Personal Social Services Regulation and Improvement Authority (HPSSRIA) undertakes regular reviews of the quality of services.

EVALUATING SERVICES

The professional responsibility of a physiotherapist includes knowing whether or not a service or an intervention is effective. This is reflected in both the Code and CSP QA standards. Several of the CSP's QA standards (CSP 2012) of physiotherapy practice include criteria that relate to evaluation, including:

- **Standard 3.1.** Members actively engage with and reflect on the continuing professional development (CPD) process to maintain and develop their competence to practise;
- **Standard 8.4.** Analysis is undertaken following information gathering and assessment in order to formulate a treatment plan, based on the best available evidence;
- **Standard 8.6.** The plan for intervention is constantly evaluated to ensure that it is effective and relevant to the service user's changing circumstances and health status;
- **Standard 9.1.** Effective quality improvement processes are in place, which are integrated into existing organisation-wide quality programmes;
- **Standard 9.2.** There is a clinical audit programme to ensure continuous improvement of clinical quality with clear arrangements for ensuring that clinical audit monitors the implementation of clinical effectiveness;
- **Standard 9.4.** The effect of the physiotherapeutic intervention and the treatment plan is evaluated to ensure that it is effective and relevant to the goals.

The central tenet of evaluation is learning from the process and outcome which leads to improvements in the quality and effectiveness of practice. Evaluation of services and practice should be carried out and the results used in the context of CPD and reflective practice, to improve an individual practitioner's personal practice and/or the delivery of a whole service. Set out below are a number of means (not mutually exclusive) by which physiotherapists can evaluate their practice.

Clinical audit

Clinical audit is a cyclical process which enables the comparison of actual practice or service delivery against standards or criteria based on the 'best available evidence' of effectiveness. In undertaking audit, a reliable benchmark with which to compare practice is required. Earlier, the importance of the local implementation of nationally-developed standards and evidence-based clinical guidelines was discussed. These provide such a reliable benchmark.

ABC Definition

Clinical audit is a cyclical process involving the identification of a topic, setting standards, comparing practice with the standards, implementing changes and monitoring the effect of those changes (CSP 2005a). Further information about clinical audit can be found in *New Principles of Best Practice in Clinical Audit* (Burgess 2011).

Audit has a key role in the continuous cycle of quality improvement when the audit cycle is complete. Firstly, through analysis of the results to identify the extent to which those standards or criteria have been met and the appropriateness of the chosen standards. Secondly, by utilising this analysis to make recommendations to change service delivery. And, finally, to ensure that action is taken on these recommendations.

Patient feedback

Patient feedback is a valuable mechanism for evaluating practice. One method is the use of a validated patient-assessed outcome measure to provide information about the patient's perception of health gain, or the use of a structured questionnaire, based on standards, to determine the patient's perception of the quality of the treatment. Responses from feedback questionnaires can be used by individuals or services to reflect on the extent to which the standards have being met, and to introduce new processes or development opportunities to secure greater conformance, if necessary.

Another valuable source of patient feedback is patients' complaints. These provide opportunities to address the issues contained within them in order to introduce a service improvement. Any issue that becomes a problem for a patient is a problem for the service and should be analysed. The involvement of the patient making the complaint in this process, if willing, will facilitate the finding of a solution that can then be embedded into systems and processes.

Health outcomes

Health outcomes measure a change in the health status of an individual or a group which can be attributed to intervention. Utilising health outcomes of care will determine the impact of the process of care or intervention on the patient's life by using specific measures before and after treatment. Measures must be chosen carefully to ensure the test, scale or questionnaire records what it aims to record (is valid and responsive) and is sufficiently well described to ensure that everyone who uses it does so in the same way (is reliable). Consideration of these factors enables the most appropriate measures for a specific patient or patient group to be identified. Information gathered will provide details on whether the intervention has had the impact intended. More information on using measures can be found on the CSP website (www.csp.org.uk).

The information will be of interest to patients themselves as an objective assessment of their improvement and is increasingly important to demonstrate the benefits and the clinical- and cost-effectiveness of physiotherapy services.

Patient-reported outcome measures (PROMs)

PROMs are measures of a patient's health status or health-related quality of life which are usually short questionnaires completed by the patient from the patient's perspective. They measure health gain and provide information on the patient's health and quality of life. The information contributes to the measurement and improvement of the quality of health services. These were initially introduced for four procedures: hip replacements, knee replacements, hernia and varicose veins. PREMS are patient-reported experience measures. Use of these is included in the CSP QA standards (Standard 8.3. Appropriate information relating to the service user and the presenting problem is collected) (CSP 2012).

NHS outcomes framework

The first NHS outcomes framework sets out the outcomes and corresponding indicators that will be used to hold the

NHS commissioning board to account for the outcomes it delivers through commissioning health services from 2012/13. This framework helps understand what it means for an NHS focussed on outcomes, and its implication for individuals, organisations and health economies (DH 2010b).

Peer review

Peer review provides an opportunity for a physiotherapist to evaluate the clinical reasoning behind their decision-making with a trusted peer. It can be applied most effectively to the assessment, treatment planning and evaluative components of physiotherapy practice where the reasoning behind the information recorded in the physiotherapy record can be explored.

CONTINUING PROFESSIONAL DEVELOPMENT (CPD)

ABC Definition

CPD is the work-oriented aspect of life-long learning (LLL) and should be seen as a systematic, ongoing structured process of maintaining, developing and enhancing skills, knowledge and competence, both professionally and personally, in order to improve performance at work (CSP 2003).

Life-long learning (LLL) is a theme promoted by the government across all sectors of the population, in order to ensure the workforce is equipped to do the jobs that will contribute to high-quality public services and promote prosperity in the UK.

In healthcare, the connection between CPD/LLL and the quality of services is at the centre of the government's view of a new, modernised NHS (CSP 2001).The requirement for re-registration of physiotherapists and other healthcare professional discussed earlier makes CPD an essential component of professional life (CSP 2005b). The introduction of a philosophy of LLL and individual responsibility for it is introduced in qualifying programmes, equipping students for a lifetime of learning in order to maintain and continually improve their competence to practise. Written evidence of learning and development, and its impact on improving practice, is now an essential requirement. Every physiotherapist must establish a portfolio containing such evidence that will need to be maintained throughout a career and is a requirement of the HCPC.

The emphasis on the importance of CPD/LLL within clinical governance is a welcome recognition. The challenge for physiotherapists to keep up to date with the fast pace of change in healthcare is significant – in particular the rapid increase in the volume of information that has to be evaluated and incorporated into practice. Protected time for CPD was recommended by the Kennedy Report (Bristol Royal Infirmary Inquiry 2001) and has been emphasised in the CSP QA standards (CSP 2012).

Another form of professional development is reflective practice, a process in which practitioners think critically about their practice and, as a result, may modify their action or behaviour. Reflective practice is the process of reviewing an episode of practice to describe, analyse, evaluate and inform professional learning. In such a way, new learning modifies previous perceptions, assumptions and understanding, and the application of this learning to practice influences treatment approaches and outcomes (CSP 2002b). 'Reflection enables learning at a subconscious level to be brought to a level where it is articulated and shared with others' (CSP 2001). Learning from experience requires the development of skills such as self-awareness, open-mindedness and critical analysis.

Some key characteristics of CPD (CSP 2003)

- It should comprise a broad range of learning activities (courses, in-service education, reading, supervision, research, audit, reflections on experience, peer review – this is not an exhaustive list).
- It is based on individual responsibility, trust and self-evaluation.
- It links learning with enhancement of quality of patient care and professional excellence while ensuring public safety.
- It should recognise the outcomes of CPD with a focus on achievement.

SKILL MIX

As discussed earlier in the chapter, physiotherapists have a professional responsibility to use their skills appropriately and should only practise to the extent that they have established, maintained and developed their ability to work safely and competently. Additionally there is a professional responsibility to use resources (human, as well as financial) appropriately in delivering healthcare. This means giving consideration to whether a patient should be referred on to another professional with different or higher level skills or to a specialist in a different clinical area. Equally, consideration should be given to whether

there are elements of the treatment programme that can be delegated to a physiotherapy assistant or other support worker.

The decision about whether to delegate, and which tasks or activities to delegate, is entirely the responsibility of the physiotherapist making that decision. The physiotherapist also takes full responsibility for the application of the tasks or activities carried out by the person who has been delegated to. Choosing tasks to be undertaken by a support worker is a complex element of professional activity that depends on an informed professional opinion.

Physiotherapists need to use their own skills and knowledge to carry out an assessment of a patient in order to formulate a clinical diagnosis and a programme of treatment derived from those findings. This process requires skills of analysis and clinical reasoning – key professional attributes. However, an appropriately trained support worker may well have the attributes required to be able to carry out some, or all, elements of the treatment programme based on existing knowledge and skills. This would include the monitoring of the patient's condition and progress with the plan, and advising the physiotherapist of any variations in either of these. As there are no guidelines about what to delegate, the physiotherapist should consider carefully the scope and nature of the task and ensure this is clearly defined and communicated to the assistant. Many organisations have specific policies and procedures for components of physiotherapy management undertaken by support workers and these should be considered.

In deciding who to delegate to, factors to be considered are the competence of the support worker and the nature of the task. The competence of the support worker will be affected by the individual's knowledge, skills informed by experience and training received. The physiotherapist should also consider the individual situation, in terms of the patient and specific circumstances. The support worker, therefore, must be allowed to make an assessment of his or her own competence in relation to the particular task.

The decision about what to delegate and who to delegate to is one that, while ultimately the responsibility of the physiotherapist, also requires the active involvement of the person to whom the task is being delegated. The task should not be delegated if either the physiotherapist or support worker is concerned about the support worker's competence. The physiotherapist will then need to decide whether training is required. Users of physiotherapy services have a right to expect those who deliver them to be competent to do so. The physiotherapist has the ultimate responsibility to the patient for ensuring this is the case, but also needs to consider competence in the context of effective resource use, in terms of both finance and skills.

Newly qualified physiotherapists should recognise and value the skills and knowledge that many support workers possess, particularly those who have extensive experience within the profession, so that effective partnerships between physiotherapists and support workers can contribute to the efficient and effective delivery of physiotherapy services. Support workers, as associate members of the CSP, are required to meet the expectations of the Code in the same way physiotherapists are.

THE FUTURE

Ensuring safe and effective healthcare within the health service continues to be a high priority for the government. Change is constant and a key challenge for physiotherapists is to respond to the opportunities and risks presented to ensure that high-quality services are delivered to patients. The NHS White Paper (DH 2010a) sets out the government's long-term vision for the NHS. This builds on the core values and principles of the NHS; that is, a comprehensive service, available to all, free at the point of use and based on need, not the ability to pay. The paper sets out how the NHS will put patients at the heart of everything the NHS does, be focussed on continuously improving those things that really matter to patients (the outcome of their healthcare) and empower and liberate clinicians to innovate, with the freedom to focus on improving healthcare services.

Many of the government's priority health programmes will be dependent for their success on the provision of effective rehabilitation in order to ensure people can continue to lead independent lives, including services for older people, children and people with long-term conditions. Physiotherapists also have a key contribution to make in keeping people fit for work through, for example, the effective management of musculoskeletal problems or the delivery of cardiac rehabilitation programmes. Ensuring ergonomically safe environments in the workplace and offering a rapid, work-based response when treatment is needed provides another example of the value of the profession.

Structural changes

Continued investment in healthcare will bring with it an increase in the expectations of the public – whose money is being used – and challenges from the government and the public about the need to change and modernise the way in which healthcare is delivered. One initiative is Any Qualified Provider (The NHS Confederation 2011) which aims to drive up quality, empower patients and enable innovation. Management of musculoskeletal back pain has been an area identified for patient choice in this way and one which physiotherapists have a key role in delivering. While this may provide some opportunities for physiotherapists, it may present a challenge to existing services.

It requires that services demonstrate their effectiveness, are responsive to patients' needs, are provided in settings closer to patients' own environments, are delivered more speedily to maximise health benefits, and utilise available resources more effectively.

More services delivered in primary care and community settings

Physiotherapy already has a track record of delivering responsive and effective services in primary care and community settings. The success of domiciliary and community-based physiotherapy services in avoiding hospital admissions and allowing speedier discharges will be further reinforced through intermediate care. Musculoskeletal physiotherapy services delivered in GP practices and health centres, where trust is already established between GPs and physiotherapists, has facilitated more direct access to patients and more appropriate referrals, either from GPs or through patients self-referring, making services more efficient and effective.

The challenges for the future, however, will lie with greater team working and delegation of tasks, with physiotherapists being increasingly flexible, taking on teaching or extended roles in order to allow other staff, such as support workers, to deliver components of physiotherapy services. There will be a need to take on some non-physiotherapeutic roles, for example as a key worker or case manager, in order to deliver a more consistent approach to care for vulnerable people living in the community.

Another challenge will be the experience of physiotherapists working in isolation from their peers and, with this, a greater emphasis needed to identify clear access to peer support, supervision or shared CPD with colleagues. Networking with colleagues with similar interests and case mix at a local and national level will become more important. Where face-to-face contact is not possible, the use of electronic networks for communication and accessing learning resources will need to be embraced. At a time when clinical governance, the requirement for re-registration and the need for systems to assure patients about practitioners' competence and safety are to the fore, physiotherapists will need to work hard to create systems to support their ongoing learning, while also ensuring their managers also accept their responsibilities.

Delivering clinical and cost-effective services

The profession can thrive only if it can clearly demonstrate the 'added value' it offers to patients through increasing their independence, shorter hospital stays, fewer work days lost and so on. In order to achieve this, the profession needs a two-pronged approach. Firstly, it needs to increase its knowledge base about the effectiveness of specific interventions through research. Secondly, it needs to use information from the evaluation of practice to demonstrate the benefit to patients of those interventions. The profession requires high-quality researchers who can access funding in order to increase the knowledge base of the profession. Challenges from commissioners of services to provide evidence for the effectiveness of physiotherapy for particular patient or diagnostic groups remain, and physiotherapy services are in increasing jeopardy without it.

The profession must look critically at the outcomes of interventions. Where research evidence shows that particular interventions are ineffective, these should stop being provided. Where patient outcomes are used as a determinant and demonstrate little or no effect, consideration should be given to possible alternative strategies for securing benefit to those patients, which may lie outside physiotherapy. For physiotherapists to continue to provide services in areas where there is little benefit weakens the image of the profession to the public and to colleagues from other professions. Where there is clear evidence for a service or intervention, these should be explored and, where possible, introduced.

There is a growing emphasis within the NHS and independent providers on working smarter, looking at systems of care from a patient's perspective, breaking down what are perceived as tribal boundaries between professions and redesigning patient-centred delivery systems rather than 'doing things that way because we always have'. To achieve this, physiotherapists have embraced new ways of working, and this needs to continue. Opportunities will emerge from redesign for physiotherapists to adopt new and highly skilled roles in just the same way as the successful creation of extended-scope practitioner and physiotherapy consultant roles.

Influencing the agenda

To influence the healthcare agenda in the future, physiotherapists need to be confident about their roles and be able to articulate to others the value of physiotherapeutic management and interventions or approaches from a science-based, as well as a holistic point of view. Physiotherapists must adopt a political astuteness that makes them aware of the wider national and local drivers for change in order that opportunities for the profession and for services can be identified and seized positively. Physiotherapists need to engage with, and be responsive to, current agendas through contacts with patient and public representatives, as well as senior managers and local politicians.

The delivery of healthcare within organisations, whether state- or independently-funded, will continue to be highly complex, ever-changing and resource-challenged. Qualifying programmes are tasked with equipping

physiotherapy students to deliver these services. Public and private healthcare providers are looking for leaders who are innovative, clear, lateral-thinkers and problem-solvers. Physiotherapists are well placed to adopt such roles and should be proactive in looking for opportunities to do so. The skill is to turn challenges and pressures into opportunities to demonstrate the 'added value' of physiotherapy which, in turn, will provide job satisfaction, and recognition and benefit for patients and the profession.

SOURCES OF CRITICAL APPRAISAL TOOLS

Books and articles

Aveyard, H., Sharp, P., 2009. A Beginner's Guide to Evidence Based Practice in Health and Social Care Professions. Open University Press, Maidenhead.

Burls, A., 2003. What is critical appraisal? Haymarket Group Ltd, http://www.whatisseries.co.uk/whatis/pdfs/What_is_crit_appr.pdf, accessed October 2012.

Crombie, I.K., 1996. The Pocket Guide to Critical Appraisal. BMJ Publishing, London.

DeBrun, C., Pearce-Smith, N., 2009. Searching Skills Toolkit: Finding the Evidence. Wiley-Blackwell, Oxford.

Greenhalgh, T., 2006. How to Read a Paper: The Basis of Evidence-Based Medicine, third ed. BMJ Publishing, London.

Guyatt, G.H., Meade, M.O., Rennie, D., Cook, D.J., 2008. Users' Guides to the Medical Literature: Essentials of Evidence Based Clinical Practice, second ed. McGraw Hill, American Medical Association, New York.

Guyatt, G.H., Sackett, D.L., Cook, D.J., et al., 1993. Users' guides to the medical literature: II. How to use an article about therapy or prevention A. Are the results of the study valid? JAMA 270 (21), 2598–2601.

Guyatt, G.H., Sackett, D.L., Cook, D.J., et al., 1994. Users' guides to the medical literature: II. How to use an article about therapy or prevention B. What were the results and will they help me in caring for my patients? JAMA 271 (1), 59–63.

Herbert, R., Jamtvedt, G., Hagen, K.B., Mead, J., 2011. Practical Evidence Based Physiotherapy, second ed. Churchill Livingstone, Edinburgh.

Jewell, D., 2009. Guide to Evidence-Based Physical Therapist Practice, second ed. Jones and Bartlett Publishers, Sudbury, MA.

Oxman, A.D., Cook, D.J., Guyatt, G.H., 1994. Users' guides to the medical literature: VI. How to use an overview. JAMA 272 (17), 1367–1371.

Oxman, A.D., Sackett, D.L., Guyatt, G.H., 1993. Users' guides to the medical literature: I. How to get started. JAMA 270 (17), 2093–2095.

Weblinks

AGREE (Appraisal of Guidelines for Research and Evaluation): http://www.agreetrust.org/

CSP (Chartered Society of Physiotherapy). CSP Critical Appraisal Skills webpage. CSP webpage explaining what critical appraisal is and why you should do it, and gives links to additional sites: http://www.csp.org.uk/professional-union/library/bibliographic-databases/critical-appraisal

SIGN 50 (Scottish Intercollegiate Guidelines Network), 2008–2011. A Guideline Developer's Handbook. A detailed description of SIGN's methodology, together with examples of checklists, evidence tables and considered judgement forms, is given in the 50th guideline, generally referred to as SIGN 50: http://www.sign.ac.uk/guidelines/fulltext/50/index.html

SPH (Solutions for Public Health). Critical Appraisal Skills Programme: http://www.sph.nhs.uk/what-we-do/public-health-workforce/resources/critical-appraisals-skills-programme

ACKNOWLEDGEMENTS

With thanks to Judy Mead who created the original chapter in the 13th edition; to Ralph Hammond and Gwyn Owen for comments on drafts of this chapter; and to Claire Cox for comments on the final draft of this chapter.

REFERENCES

Barclay, J., 1994. In Good Hands. Butterworth-Heinemann, Oxford.

Bristol Royal Infirmary Inquiry, 2001. The Report of the Public Inquiry into Children's Heart Surgery at the Bristol Royal Infirmary 1984–1995. Stationery Office, London.

Burgess, R., 2011. New Principles of Best Practice in Clinical Audit. Radcliffe Publishing, Abingdon.

Bury, T., 1998. Evidence-based healthcare explained. In: Bury, T., Mead, J. (Eds.), Evidence-Based Healthcare: A Practical Guide for Therapists. Butterworth-Heinemann, Oxford.

Cochrane, A., 1972. Effectiveness and Efficiency: Random Reflections on Health Services. Nuffield Provincial Hospitals Trust, London.

Cott, C.A., Finch, E., Gasner, D., et al., 1995. The movement continuum theory of physical therapy. Physiother Can 47 (2), 87–95.

CSP (Chartered Society of Physiotherapy), 2000. Clinical Audit Tools. CSP, London.

CSP (Chartered Society of Physiotherapy), 2001. Continuing Professional Development and Life Long Learning. CSP, London.

CSP (Chartered Society of Physiotherapy), 2002a. Rules of Professional Conduct. CSP, London.

CSP (Chartered Society of Physiotherapy), 2002b. Curriculum Framework for Qualifying Programmes in Physiotherapy. CSP, London.

CSP (Chartered Society of Physiotherapy), 2002d. Physiotherapy Consultant (NHS): Role, Attributes and Guidance for Establishing Posts. CSP, London.

CSP (Chartered Society of Physiotherapy), 2003. Policy Statement on Continuing Professional Development (CPD). CSP, London.

CSP (Chartered Society of Physiotherapy), 2005a. Standards of Physiotherapy Practice. CSP, London.

CSP (Chartered Society of Physiotherapy), 2005b. Demonstrating Professionalism Through CPD. CSP, London.

CSP (Chartered Society of Physiotherapy), 2008. Scope of Physiotherapy Practice. CSP, London.

CSP (Chartered Society of Physiotherapy), 2010. The History of the Physiotherapy Profession and the Chartered Society of Physiotherapy. CSP, London.

CSP (Chartered Society of Physiotherapy), 2011a. CSP Code of Professional Values and Behaviours. CSP, London.

CSP (Chartered Society of Physiotherapy), 2011b. CSP Vision for the Future of UK Physiotherapy. CSP, London.

CSP (Chartered Society of Physiotherapy), 2011c. Learning and Development Principles for CSP Accreditation of Qualifying Programmes in Physiotherapy. CSP, London.

CSP (Chartered Society of Physiotherapy), 2011d. Scope of Physiotherapy Practice in the UK – CSP Guidance for Members and Governance Arrangements. CSP, London.

CSP (Chartered Society of Physiotherapy), 2012. CSP Quality Assurance Standards for Physiotherapy Service Delivery. CSP, London.

Dawes, M., Summerskill, W., Glasziou, P., et al., 2005. Sicily statement on evidence-based practice. BMC Medical Education 5, 1.

DH (Department of Health), 1991. Research for Health: A R&D Strategy for the NHS. DH, London.

DH (Department of Health), 2002. Health Professions Order Statutory Instrument 2002 No. 254. Her Majesty's Stationery Office, London.

DH (Department of Health), 2004. The Shipman Inquiry: Fifth Report – Safeguarding Patients: Lessons from the Past – Proposals for the Future. Her Majesty's Stationery Office, London. http://www.shipman-inquiry.org.uk/5r_page.asp; accessed October 2012.

DH (Department of Health), 2010a. Equity and Excellence: Liberating the NHS. DH, London.

DH (Department of Health), 2010b. NHS Outcomes Framework. DH, London.

DHSS (Department of Health and Social Security), 1973. McMillan Report: The Remedial Professions (report by a working party set up in March 1973 by the Secretary of State for Social Services). Her Majesty's Stationery Office, London.

DHSS (Department of Health and Social Security), 1977. Health Services Development: Relationship between the Medical and Remedial Professions [HC(77)33]. DHSS, London.

Ersser, S.J., Atkins, S., 2000. Clinical reasoning and patient-centred care. In: Higgs, J., Jones, M. (Eds.), 2000. Clinical Reasoning in the Health Professions, second ed. Butterworth-Heinemann, Oxford.

Field, M.J., Lohr, K.N. (Eds.), 1992. Guidelines for Clinical Practice: From Development to Use. National Academy Press, Washington, DC.

HCPC (Health and Care Professions Council), 2001. Health Professions Order 2001. HCPC, London.

HCPC (Health and Care Professions Council), 2005. What Happens if a Complaint is Made About Me? HCPC, London.

HCPC (Health and Care Professions Council), 2007. Standards of Proficiency Physiotherapists. HCPC, London.

HCPC (Health and Care Professions Council), 2008. Standards of Conduct, Performance and Ethics. HCPC, London.

HCPC (Health and Care Professions Council), 2011. Continuing Professional Development and Your Registration. HCPC, London.

Herbert, R., Mead, J., Jamtvedt, G., Birger Hagen, K., 2005. Practical Evidence-based Physiotherapy. Butterworth-Heinemann, Oxford.

Higgs, J., Jones, M. (Eds.), 1995. Clinical Reasoning in the Health Professions. Butterworth-Heinemann, Oxford.

Higgs, J., Titchen, A., 2001. Rethinking the practice–knowledge interface in an uncertain world: a model for

practice development. Br J Occup Ther 64 (11), 526–533.

Jones, M., Jensen, G., Edwards, I., 2000. Clinical reasoning in physiotherapy. In: Higgs, J., Jones, M. (Eds.), 2000. Clinical Reasoning in the Health Professions, second ed. Butterworth-Heinemann, Oxford.

Koehn, D., 1994. The Ground of Professional Ethics. Routledge, London.

Marriott, C., 1996. Towards Tomorrow: The Future Role of the Consultant. Central Consultants and Specialists Committee. BMA, London.

NHS Executive, 1996. Promoting Clinical Effectiveness. Department of Health, London.

NICE (National Institute for Health and Clinical Excellence), 2001. The Guideline Development Process – Information for National Collaborating Centres and Guideline Development Groups. NICE, London.

Richardson, B., 1999. Professional development: 1 Professional socialisation and professionalisation. Physiotherapy 85 (9), 461–466.

Roberts, P.A., 2001. The practice of physiotherapy. PhD thesis, Sheffield Hallam University, Sheffield, UK.

Sackett, D.L., Rosenberg, W.M.C., Gray, J.A.M., et al., 1996. Evidence-based medicine: what it is and what it isn't. BMJ 312, 71–72.

Secretary of State for Health, 1998. A First Class Service: Quality in the New NHS. Department of Health, London.

Sudlow, M., Thomson, R., 1997. Clinical guidelines: Quantity without quality. Qual Health Care 6 (2), 60–61.

The NHS Confederation, 2011. July, Issue 10 Any Qualified Provider – Discussion Paper. The NHS Confederation, London.

Tidswell, M., 1991. Physiotherapy: A true profession? MA dissertation, University of Keele, Keele.

Tidswell, M., 2009. Adversity the Spur: The History of Physiotherapy Education at Oswestry. Athena Press, London.

WCPT (World Congress for Physical Therapy), 2011. Policy Statement: Description of Physical Therapy. WCPT, London.

Chapter 2

Collaborative health and social care, and the role of interprofessional education

Alison Chambers, Lynn Clouder, Mandy Jones and Jill Wickham

INTRODUCTION

To new physiotherapy students it may seem strange to give prominence to a chapter on interprofessional education (IPE) and team working when they are already convinced they want to be physiotherapists; why would they need to know about other professions? It is well known that students start their professional training with deeply ingrained and historically informed views about what it means to be a particular health and social care practitioner (Hean et al. 2006). However, the inclusion of this chapter, which is new to this edition, signals a change in both professional education and in practice that acknowledges that no one profession can practise effectively in isolation and that a change to a more collaborative way of working is necessary to deliver the best outcomes for clients and service users. In fact, we argue that all practice is collaborative.

Of course, students will find that IPE practices differ with context and it is one of the aims of this chapter to discuss the varied dimensions of IPE so that students might develop critical awareness of their own programme of study and the practice to which they are exposed during clinical placement experience. We begin by clarifying what we mean by IPE. One of the greatest challenges is the conceptual ambiguity surrounding IPE and several related terms that are used interchangeably and without discretion. We will also explore the drivers and challenges that will have influenced the IPE initiatives in which students might find themselves involved.

It is important that students are aware from the outset that IPE is not a taken for granted concept, accepted unconditionally and embraced by all health and social care professions, or even individuals with equal enthusiasm. There has been a long history of concern over poor communication and professional rivalry between disciplines and competing professionals (Cleake and Williamson 2007). Inevitably, this has influenced how health professional education has been delivered and, as a consequence, it has been criticised for being 'designed and delivered within silos' (Ramsammy 2010: 134). Research confirms that silo models remain deeply entrenched in places (Barker et al. 2005). We aim to introduce students to the political, social and economic complexities of IPE that have resulted in its patchy implementation. However, we will also explore principles of best practice, different models, the extent of its global spread and point to evidence of impact where it exists.

Moving full circle in justifying the focus of the chapter, we conclude by discussing the interface between professional and interprofessional identities. Mhaolrunaigh (2001) suggests that embracing the notion of working collaboratively requires students to reflect on their own profession and professional background, and to then see their own profession in relation to 'new others'. These are fundamental issues which might seem of a lower priority than the more immediate concerns of proving competence and accumulating the vast knowledge base necessary for contemporary practice. However, rather than compromising uni-professional knowledge, IPE provides the means of adding to a different type of knowledge and confers a different type of capability. Collaborative learning 'allows students to become members of knowledge communities whose common property is different from the common property of the knowledge communities they already belong to' (Bruffee 1999: 3). IPE does not detract from the status of the professional; it makes for a flexible and open-minded practitioner capable of working

across boundaries and providing collaborative holistic client care.

History of interprofessional education (IPE)

IPE is simply defined as 'Occasions when two or more professionals learn with, from and about each other to improve collaboration and the quality of care' (CAIPE 1997). Although professionals may have shared their learning experiences and expertise since formal healthcare was conceptualised, specifically planned and structured opportunities for IPE were not established in the UK until the 1960s (Barr 2007), when the first interprofessional symposium exploring 'Family Health Care: The Team' was held in London (Kuenssberg 1967). In the UK, the evolution of IPE has been integrally linked with political change and social growth.

Towards the end of the last century it became apparent that many hospital- and community-based services were essentially working in isolation, each with little knowledge of the other's role, expertise or skills. This absence of communication and liaison between healthcare contributors led to fragmented service delivery and substandard patient care (Barr 2007). These collaborative failures sometimes resulted in tragedy, particularly in child protection (Colwell Report 1974; Laming Report, 2003) and psychiatric aftercare (DH 1994). The factors contributing to poor working relationships between health and social care professionals are extremely complex; many professions had their roots entwined with status, class and gender (Barr 2007), or provide identity to their members, promoting prejudice or professional mistrust (Carpenter 1995). Professional isolation was perpetuated through the use of specialist language or jargon (Pietroni 1992), or keeping individual patient records. Indeed, health and social care students were not only entering their professional training with established prejudice regarding other professions, but qualifying and leaving with their prejudices reinforced (McMichael and Gilloran 1984). This notion raised the possibility of using education to improve interprofessional understanding and successful collaborative working.

In 1987, the Centre for the Advancement of Interprofessional Education (CAIPE) was established in the UK. Initially, this charity organisation directed its attention to issues within primary care, but subsequently expanded with interests in local government, higher education and professional associations. The following year, the World Health Organization (WHO) issued a statement which highlighted the theory that if healthcare professionals learned to collaborate as students, these skills would translate to the workplace, facilitating effective clinical or professional team working (WHO 1988). Indeed, throughout the 1980s the Health Education Authority (HEA) launched a series of shared learning workshops throughout England and Wales, focussing on specific patient groups and their problems (Spratley 1990). The concept of collaborative learning was supported by the government white paper 'Community Care in the next Decade and Beyond' (DH 1989), which emphasised the importance of 'multiprofessional training' and was used in the foundations of the 1990 NHS and Community Care Act (DH 1991). These guidelines stated that 'joint training' was an expectation, which must be included in community care plans and training strategies (DH 1991; Barr 2007). The 2001 government white paper 'Working Together, Learning Together: A Framework for Lifelong Learning for the NHS' (DH 2001) provided a strategic framework and co-ordinated approach to continued professional development. It stated that 'core skills, undertaken on a shared basis with other professions, should be included from the earliest stages in professional preparation in both theory and practice settings'.

Most early IPE events were small-scale workshops or short courses focussing on aspects of primary or community care (Barr 2007). However, as many royal colleges, professional and regulating bodies pledged support and contributed to IPE evolution, educational initiatives grew in size and commanded increasing interest. Over the last 30 years, IPE has become an established movement. Shared learning between health and social care professionals is now embedded in most undergraduate curricula and continually extends through professional development. Governments continue to issue clear policy to encourage collaborative practice and partnership working, to develop students able to undertake, and contribute to, effective NHS interprofessional working (Finch 2000).

Key points

IPE is defined as 'Occasions when two or more professionals learn with, from and about each other to improve collaboration and the quality of care'.

The concept of IPE was born out of using education to improve interprofessional understanding and successful collaborative working to improve patient care.

The first specifically planned and structured opportunities for IPE were held in the UK in 1966, when the first interprofessional symposium exploring the 'Family Health Care: The Team' was held in London.

In the UK, the evolution of IPE has been integrally linked with political change and social growth.

Currently, IPE for both undergraduate and postgraduate healthcare professionals is endorsed by the WHO, many Royal Colleges and professional regulatory bodies.

Governments continue to issue clear policy to encourage collaborative practice and partnership working, to develop students able to undertake and contribute to effective interprofessional working

Is IPE an international phenomenon?

The WHO identified IPE as a priority activity in 1984. In 1988, it published a report which drew on examples from developed and developing countries to inform a unifying definition and rationale for IPE (Barr 2009). The report suggested that 'students should learn together during certain periods of their education to acquire the skills necessary for solving the priority health problems of individuals and communities known to be particularly amendable to teamwork' (Barr 2009: 17).

Since the late 1980s the call for a shift towards collaborative interprofessional practices has crossed international boundaries, inspired by a common goal of reducing health inequalities and improving the health of populations. These ideals were reiterated in the recent WHO Framework for Interprofessional Education and Collaborative Care (WHO 2010). WHO member states, eager to achieve equitable, fair, affordable and efficient care, are increasingly focussing on primary care as providing the most suitable means (Gilbert 2010). This focus is evident in the increase in interest in interprofessional collaboration and in policy commitments around the world, for example the Canadian government's call for a move towards patient-centred interprofessional primary care (Health Canada 2003). In Japan, where longevity rates are among the highest in the world, IPE as a means of promoting collaborative working is considered essential to promoting quality of life (Takahashi and Sato 2009). Concerns about quality and safety issues in the USA healthcare system has resulted in resurgence in interest in IPE as a means of promoting interprofessional collaboration and reducing patient errors (Ragucci et al. 2009), while IPE in the UK has emerged through a series of government policies aimed at modernising health and social care.

A recent edition of the *Journal of Interprofessional Care* (March 2010) drew attention to the development of IPE internationally, highlighting that notwithstanding additional challenges and resource constraints in developing countries, recognition of the benefits of a collaborative workforce is not confined to Western countries. In 2009 the WHO established the Health Professionals Global Network with the aim of maximising the potential of all health professionals through a virtual network, fostering interprofessional collaboration and contributing to the global health agenda (see http://hpgn.org/). In 2010, the network held a global consultation on IPE and collaboration, and more than 1000 delegates from 97 countries participated (Gilbert 2010). The recent Framework for Action on Interprofessional Education and Collaborative Practice (WHO 2010) captures a sense of IPE practices at a global level. Representatives from 42 counties provided insight into practices from practice, administration, education and research perspectives.

The 42 countries contributing to the international scan of IPE practices were: Armenia, Australia, Bahamas, Belgium, Canada, Cape Verde, Central African Republic, China, Croatia, Denmark, Djibouti, Egypt, Germany, Ghana, Greece, Guinea, India, Islamic Republic of Iran, Iraq, Ireland, Japan, Jordan, Malaysia, Malta, Mexico, Nepal, New Zealand, Norway, Pakistan, Papua New Guinea, Poland, Portugal, Republic of Moldova, Saudi Arabia, Singapore, South Africa, Sweden, Thailand, United Arab Emirates, UK, USA and Uruguay.

The findings of the scan show that IPE occurs in 'many different countries and health care settings across a range of income categories' (according to the World Bank's Income Classification Scheme) and 'involves students from a broad range of disciplines including allied health, medicine, midwifery, nursing and social work' (WHO 2010: 16). Physiotherapy students in a wide range of countries can expect to find that IPE is a compulsory aspect of their curriculum at undergraduate level that is mostly delivered using a face-to-face approach, although the use of technology to foster IPE is growing, especially where large numbers of students from several professions are involved.

The aim of this section is to encourage you to examine the concept of yourself as a practitioner as an integral member of an interprofessional team by:

- considering the concept of IPE as integral to your own learning;
- exploring how your own practice relates to the concept of interprofessional working;
- reviewing how to anticipate and improve interventions carried out by yourself and others;
- understanding the importance of current evidence and policy, and how to take forward the learning from a simulated environment into the workplace;
- developing the ability to interpret information by the use of student-led interprofessional learning.

Having engaged with the learning you should have developed the ability to explore current practice utilising your reflective skills, knowledge and understanding to improve your personal and professional development, and thus enhance delivery of care to the service user in an interprofessional working environment. A good practitioner is, arguably, someone who is always punctual, who is respectful to clients and to other team members, and is considered to be reliable. What is the difference between a good and an excellent practitioner? An excellent practitioner is one who achieves best possible practice. While working through this section you should be considering what makes an excellent practitioner and at the same time reflecting upon your own practice to identify ways to always strive for excellence. There are certain factors that make being a practitioner easier. Physiotherapy students need to develop skills to enable them to work in an interprofessional environment. As well as the interpersonal skills required to work effectively in a range of environments with a range of personnel (at all levels), there is a need to develop confidence, clinical reasoning, evaluation

and reflective skills (Smith and Green 2003). In fulfilling the ability to self reflect it is imperative to understand therapeutic use of self, self-awareness and constructive use of feedback. Exploration of these skills will make the application of interprofessional working more feasible.

THE IPE CONTEXT WITHIN THE UK

When exploring the literature concerned with IPE, it is evident that there is some confusion over descriptions and definitions. The nomenclature can be confusing, as Barr (2005: xvii) states: 'interprofessional education is bedevilled by terminological inexactitude'. For the purposes of this chapter, the authors have used the CAIPE (1997) definition of IPE, emphasising *learning from, with and about each other*.

Table 2.1 describes the nomenclature you may find in the literature and/or experience as part of your learning experiences.

As described in the previous sections, there is no definitive model of IPE; thus, organisations have developed their own models which best suit their organisational contexts and student mix. The Leicester Model of IPE is one example (Lennox and Anderson 2007); for further examples you are directed to the higher Education Academy occasional paper *Piloting Interprofessional Education* (Barr 2007).

As physiotherapy students it can sometimes be difficult to understand the reasoning behind the inclusion of IPE in your uni-professional programme. The previous sections have provided you with an overview of the history behind the development of IPE, as well as some examples of how you may experience IPE during your studies. This next section will describe the context in which IPE exists and thus enable you to understand its importance to you as an individual, and to the wider health and social care professional community.

Irrespective of how different organisations have implemented IPE they share a common context in which they operate; this section will outline the political, social, economic and technical drivers that underpin the requirement for IPE (Table 2.2).

The imperative to expose pre-registration health and social care students to IPE has, for some time, been directed by a number of policy directives and is not new (DH 2000a, 2000b, 2001, 2009, 2010a, 2010b; Hammick et al. 2002). Since the NHS plan was published in 2000, there has been an increased emphasis on a patient-led NHS and, alongside that, the requirement for IPE to be an integral part of education and training programmes. UK health professional regulatory bodies also demand the inclusion of IPE in all professional curricula (NMC 2004; HCPC 2004). Within higher education, the quality assurance of health and social care education has as a central tenet the requirement to demonstrate where and how IPE is being taught within pre-registration curricula (QAA 2006).

Recent policy documents (DH 2000a, 2000b, 2001, 2006, 2009, 2010a, 2010b) re-emphasise the continuing drive towards an agenda of modernisation and

Table 2.1 IPE common terms

Term	Learning
Multi-professional	Learn side-by-side
Interprofessional	Learn with, from and about each other
Shared learning	Learn alongside each other with NO interaction
Common learning	Learning a common curriculum

Source: Barr (2005).

Table 2.2 IPE context analysis

Political	Economic
Government policy – NHS plan, quality, choice, right care, right place, right professional, equity and excellence liberating the NHS, modernisation and improvement Professional bodies – curriculum frameworks, codes of conduct, scope of practice, continuing professional development requirements Professional regulation – standards and proficiencies, accountability Healthcare organisations – ways of working, service design, workforce design	**Cost improvement** – efficient use of limited resources **Value for money** – best care, best time, best place Increased cost of healthcare – change in demographics
Social	**Technological**
Public and patient empowerment – patient-centred health and social care Expert patient Change in demographics – ageing population, long-term conditions Increased professional accountability – in response to failures in care delivery	Treatment advances – living longer **Improved diagnostics** – earlier diagnosis New service delivery models, e.g. telemedicine Enhanced information technology

improvement in health and social care services, predicated upon the view that interprofessional working is key to its success. Also, the political imperative to ensure continual service improvement cannot be achieved without professionals working together: 'if we are to achieve the aim of health care teams continuously improving the quality of care they provide to their patients, we need to deepen our understanding of what is needed to enable them to work and learn together' (Wilcock et al. 2009: 88). As Davidoff and Batalden (2005: 80) suggest, 'the ideal conditions for achieving this are when everyone working in health care recognises that they have two jobs when they come to work every day, to do their work and improve it'. In addition, the National Institute for Health and Clinical Excellence (NICE) supports the notion of creating a more flexible workforce that can operate across boundaries and promote partnerships (www.nice.org.uk).

Failures in traditional care delivery have contributed to the call for greater collaboration of healthcare professionals. Examples such as the Bristol Royal Infirmary Inquiry (DH 2001), the Victoria Climbie Inquiry (Laming Report 2003) and, more recently, Baby P (Laming 2008) highlight the fact that uni-professional working may hinder optimum care while interprofessional working may help to avoid these failures. These very public failures have led to an increased scrutiny of health and social care professionals, with increased accountability.

The demographic changes in the UK population – an increasingly ageing population and people living with long-term conditions as a result of improvements in diagnosis and treatment advances – has led for a call for healthcare professionals to work across traditional boundaries and create inter-agency partnerships, all requiring improved collaboration between professional groups (Wanless 2004).

The importance of IPE had sufficient prominence to lead, in 2001, to the Department of Health (DH) commissioning four, leading-edge IPE pilot programmes. These were collaborations between universities and the then Workforce Development Confederations (a body responsible for the commissioning of health and social care education on behalf of the DH via Strategic Health Authorities on a regional basis).

Within the Allied Health Professions (AHP) the publication in 2000 of the 'Meeting the Challenge: A Strategy for Allied Health Professions' document further emphasised the importance of collaboration and cross-boundary working outside of traditional uni-professional silos. Although primarily concerned with describing the changes in the roles of AHP within contemporary healthcare, one of the strategy's main tenets called for undergraduate education programmes to be more flexible and more patient- and practice-centred via the inclusion of enhanced opportunities for IPE (DH 2000a, 2000b).

In 2005, the Health and Care Professions Council (HCPC 2012) standards of proficiency under the section relating to professional relationships states that:

> *Registrants need to understand the need to build and sustain professional relationships*
> *Are able to contribute effectively as a member of a multidisciplinary team*
> *Are able to sustain professional relationships as an independent practitioner*

In 2006 the Quality Assurance Agency (QAA) produced generic benchmarking across all health and social care programmes by including common statements while at the same time safeguarding the distinctive learning needs of each profession. These benchmarks include reference to integrated service delivery.

Despite all these initiatives around increasing the opportunity for IPE for health and social care students in practice, there is still a lack of substantive evidence that engaging in IPE impacts positively on interprofessional working in practice. In response to this, the Creating an Interprofessional Workforce (CIPW) project was initiated and findings were published in 2007. CIPW provides a strategic framework for IPE and training to underpin collaborative working practices and partnerships (DH 2006).

In 2008, the NHS next stage review led by Lord Darzi further emphasised an NHS founded upon providing high quality care for all through a patient-led NHS.

Most recently, the new government published its NHS white paper 'Equity and Excellence: Liberating the NHS' (DH 2010a). Although in many ways this strategy moves away from the previous government's strategy for the NHS, the imperative for a patient-centred NHS remains a key priority. The main tenets of this white paper are:

- patients and public first;
- services designed around patient's individual needs, lifestyles and aspirations;
- giving citizens a greater say in how the NHS is run;
- a truly patient-led NHS;
- strengthening the collective voice of patients by creating Health Watch England;
- shared decision-making – '*no decision about me without me*';
- focussed upon outcomes and quality standards.

In 2011, contemporary health and social care is increasingly complex, requiring integrated organisations which require a work force that is adaptable, creative and collaborative. IPE provides the foundations on which to develop graduates who are capable of building strong interprofessional relationships in practice in order to engage in collaborative working.

Over the past 10 years, the modernisation of the NHS has seen a significant cultural shift in the way that healthcare is delivered, requiring healthcare professionals to work in very different ways. Prior to the modernisation movement, healthcare was essentially profession-led, where professionals made decisions about patients for patients. The shift away from profession-focussed care to

one where patients and the public are empowered to make decisions and choices in relation to their healthcare has required professionals to work in different ways. No longer is it acceptable for professionals to exist in professional silos. Service modernisation has resulted in service delivery models that are predicated upon care pathways and not profession focussed pathways. Practitioners increasingly work in multi-professional teams associated with a specific care pathway (e.g. long-term conditions, stroke, cancer, end of life, musculoskeletal) as described in the National Service Frameworks (DH 2006).

The list below provides a checklist of the publications that outline the major drivers:

World Health Organization (WHO)
1988 Learning Together to Work Together for Health
2010 WHO Framework for Action on Interprofessional Education and Collaborative Practice

Department of Health (DH)
2000 A Health Service of All the Talents: Developing the NHS workforce
2000 The NHS Plan: A plan for investment, A plan for reform
2000 Meeting the Challenge: A strategy for the Allied Health Professions
2001 Working together, learning together
2003 The Victoria Climbie Inquiry: Report of an inquiry by Lord Laming
2004 The NHS Knowledge and Skills Framework and the Development Review Process
2006 Our Health, Our Care, Our Say
2007 Creating an Interprofessional Workforce
2008 Allied Health Professions Competence Framework
2008 High Quality Care for All: NHS Next Stage Review
2010 Equity and Excellence: Liberating the NHS
2010 Equity and Excellence: Developing the NHS workforce
2010 Equity and Excellence: Public Health Paper

Professional, Regulatory and Statutory Bodies
2006 QAA benchmark statements
2006 HCPC standards of proficiency
2010 CSP curriculum framework

It is these documents that provide the policy context and major driving force behind the impetus for the inclusion of IPE in the education and training of all health and social care professionals. Contemporary health and social care service delivery models require a workforce capable of working together in the interests of improving healthcare outcomes focussed upon the patient's needs and not the profession's.

All health and social care professionals are required to work in interprofessional teams within integrated service delivery models across and around professional boundaries. All health and social care students are required to be interprofessionally competent (at the very least capable of working collaboratively) – actively engaging in IPE as an integral part of their professional programmes supports the development of such competence.

IPE provides the vehicle for students to:

- gain an understanding of the complexity of interprofessional health and social care delivery;
- develop and practise team-working skills (communication, negotiation, value, respect, collective decision-making, joint goal-setting and outcome measurement);
- be prepared for working across traditional boundaries;
- practise their own professional role within the context of the wider healthcare team in a secure learning context where mistakes will not have real life consequences;
- test out and reflect upon their own professional knowledge, skills and values against others;
- test out negotiating skills around professional boundaries and learn about/from the difficulties inherent in the process.

It can be argued that one of the most important lessons for you as undergraduates to take away from your experiences of IPE is the recognition that no one individual professional or distinct professional group can meet all the health and social needs of individuals requiring health and social care intervention. In order to contribute to the improvement of health outcomes for their patients, today's practitioners must be able and willing to work collaboratively, knowing when they have a contribution to make and, importantly, when they do not and therefore seek help from colleagues. IPE provides real opportunities for undergraduates and postgraduates to learn with and from each other in ways that uni-professional learning will never be able to do (Bokhour 2006; Lennox and Anderson 2007; Barr 2007).

Learning together is never easy; students studying specific professional programmes commence those programmes with well-formed constructions of the professional they are learning to become. This can make learning together a difficult process. It is never easy when cultures collide (Jenkins 2004). IPE can provide an opportunity to improve understanding of self; Chambers (2009) suggests that during interprofessional exchanges students test out their emerging professional identity as a way of working out what they are not in order to work out what they are. She suggests that IPE is pivotal in the process of constructing a particular professional identity.

IPE and collaborative working

A number of significant political and societal and professional changes have taken place over the past ten years and continue to do so. These influence how health and social students are prepared for a career likely to span 40 years. The changes as described above have had a major influence on the education and training of healthcare

professionals as educators are charged with ensuring health and social care graduates are equipped to be effective practitioners in a complex, ever-evolving health and social care system. Educators are required to prepare graduates who are fit for practice and fit for award. The patient and public empowerment agenda continues to be a priority. In order to attend to this, healthcare professionals will need to practise in ways that empower patients to be partners in their own care (DH 2009).

For this to be successful, professionals will need to practise collaboratively, work in partnership with other members of the health and social care team, engage in joint decision-making and goal-setting, and ultimately work towards the same end. Interprofessional learning can support the development of skills enabling graduates to be fit for purpose.

Collaborative working requires individuals to be good at team-working, be aware of their own contribution and the contribution of others, be willing to take a back seat as and when this is in the best interest of the patient, and work towards agreed outcomes and goals with the patient at the centre of any decision-making.

Remember … … 'No decision about me without me' (DH 2009).

Key points for successful collaborative working

• Successful team-working	• Individuals' requirements
• Openness	• Being secure within own professional role but not defensive • A willingness to see things from all sides
• Value and respect different perspectives	• Understanding of other professionals' expertise and contribution • Appreciation of different perspectives • Being able to see things from a different perspective
• Shared outcomes, improved health outcomes for patients	• Negotiated outcomes that matter to the patient not individual professionals • Patients as partners • Shared intelligence
• Agreed roles and responsibilities	• Agreeing professional boundaries, the most appropriate professional providing the right intervention
• Active engagement	• Recognising own professional contributions and limitations
• Flexible	• Willingness to revise own role and priorities for improved patient outcome when required • May involve picking up actions/tasks and giving tasks away to other team members

INTERPROFESSIONAL EDUCATION (IPE) IN PRACTICE

Although the primary motivation behind the evolution and instigation of IPE was to improve health and social care service delivery, the rationale for IPE has firm roots in adult educational theory (www.faculty.londondeanery.ac.uk). In particular, early work by Allport (1954) generated the term 'contact hypothesis'. This theory is often applied to IPE (Street et al. 2007) and 'suggests that attitudes towards diverse groups will improve with contact with that group', particularly where 'there is equal status, focus on differences as well as similarities, the perception that members are "typical" of their professional group, and opportunity to experience successful working together'. Freeth (in Hammick et al. 2007) also believes that positive interprofessional contact may change attitudes and that well-facilitated IPE can channel conflict and stimulate critical debate which promotes professional and personal development. Jarvis (1983) proposed that adults learn effectively when faced with a discrepancy between what they believe they know and what they actually need to know. This draws an interesting parallel with attitudes of established professional stereotyping and the need for collaborative working. Coulshed (1993) stressed the importance of ownership of learning, highlighting that effective adult learning takes place when students can work collaboratively in groups, drawing and reflecting on previous experiences to solve specific problems, thus controlling their work. Indeed, it is suggested that an understanding of adult learning concepts is key for positively received IPE, to ensure learning needs are met through well-structured curriculum development (Hammick et al. 2007). Miler et al. (1999) advocate the use of an interactive approach, such as 'user-centred' scenarios to IPE, to enable students to effectively transfer their collaborative knowledge and skills directly to the healthcare environment.

Over the last 10 years many IPE initiatives have been launched over a wide variety of healthcare settings (Carpenter and Hewstone 1996; Barnes et al. 2000; Gilbert et al. 2000; McNair et al. 2001; Way et al. 2002; Barrett et al. 2003; Kilminster et al. 2004; Street et al. 2007),

providing a framework for successful implementation. The optimal time to commence IPE has induced much debate, but as negative professional stereotyping may evolve early in training, Vanclay (1997) advocates that IPE should be implemented from the beginning of professional education – a view endorsed by the WHO (1988). Carpenter and Hewstone (1996) evaluated a compulsory week-long, shared learning programme for doctors and social workers, where students were given opportunities to 'undertake successful joint work in a co-operative atmosphere'. Working in pairs, they planned the management of a clinical case from their own professional standpoint, co-facilitated by both a doctor and social worker, who focussed their attention and positive feedback on differences and any similarities between their approaches. Prior to starting the programme, the Professor of Mental Health demonstrated institutional support to the medical students and each group was given an overview of the educational background of the other. Although not without difficulty, upon completion both sets of students positively evaluated the programme, gaining an improved attitude toward the other profession. Each rated the other as professionally competent and reported increased knowledge of their skills, role and duties, together with a heightened awareness of what is required for effective collaborative working.

The role of the educator has been highlighted as key to the success of IPE initiatives (Ponzer et al. 2004; Freeth et al. 2005; Buring et al. 2009). Uniting educators from differing backgrounds to teach together is not sufficient to provide a beneficial IPE experience (Buring et al. 2009). Buring et al. believe that IPE educators need to develop and nurture their approach through their own learning in order to educate reflective practitioners who can work collaboratively within the interprofessional healthcare team. Shared knowledge, skills and values are imperative for educators within the collaborative setting (Buring et al. 2009). In addition, through the process of self-development, as IPE educators evolve, implement and teach together, not only do they gain each other's professional trust and understanding, but they also provide a positive role model for the students they educate (Freeth et al. 2005). The importance of staff development and its impact on effective IPE was highlighted in a recent systematic review of interprofessional education (Hammick et al. 2007). However, for this process to work, it requires institutional support which challenges the historical hierarchy among healthcare professionals (Buring et al. 2009). Indeed, management support and other resources, such as time, appear to be of paramount importance in founding and maintaining IPE initiatives (Hammick et al. 2007). Evaluation of a two-day interprofessional team-building workshop attended by 21 students from 13 undergraduate health and human service programmes in Canada concluded that to successfully implement an IPE programme, a university structure which actively fosters collaboration across its own departments is essential (Gilbert et al. 2000).

Interprofessional learning is centred upon an exchange of professional knowledge, understanding, attitudes or skills to improve collaboration and, ultimately, healthcare outcomes (www.faculty.londondeanery.ac.uk). Therefore, delivery of each IPE initiative must be customised and tailored to reflect particular service delivery settings, to provide a positive experience for its targeted practitioners (Kilminster et al. 2004; Hammick et al. 2007). It is much more meaningful to learn interprofessional behaviour in the context of current or future practice, than in an artificial simulated situation (Kilminster et al. 2004; Hammick et al. 2007). Furthermore, the design of the IPE initiative and the timing of its delivery must be appropriate to match the stage of professional development of its participants (Kilminster et al. 2004).

Co-operation and collaboration have been identified as the key features of IPE (Walker et al. 1998; Way et al. 2000). Way et al. believe collaboration is a process for interprofessional communication and decision-making, allowing the knowledge, skills and views of different healthcare professionals to unite and provide holistic patient care. Following the development and evaluation of a series of case studies aimed at collaboration between primary care partners, this group identified seven elements which they deem to be essential for successful collaborative practice: responsibility and accountability, co-ordination, communication, co-operation, assertiveness, autonomy, and mutual trust and respect (Way et al. 2002). This view was supported by Kilminster et al. (2004) who implemented patient-focussed workshops involving pre-registration house officers, student nurses and pre-registration pharmacists. Evaluation of this initiative included the curriculum development, delivery of the workshops and how each influenced the participants. They concluded that the workshops were successful, with participants gaining increased respect for each other's profession. This mutual respect enabled each professional group to feel comfortable with the other to offer and ask for advice. In addition, all professional groups reported enhanced communications skills, both between different team members and with the patient. Similar findings were described by Street et al. (2007), who evaluated a paediatric community practice-based IPE initiative involving medical and paediatric nursing students. These workers highlighted the enhanced learning opportunities generated through interprofessional collaboration, together with a change in attitude between the professions; professional stereotyping was reduced, while understanding of specific roles, knowledge and skills increased.

Healthcare professionals do not work in isolation, but as a contributor to the multi-professional team. The diverse and complex nature of the clinical environment demands effective collaborative working. As such, IPE provides an opportunity for healthcare professionals to

develop the essential skills they require to improve patient outcomes, enhance service delivery and become reflective, collaborative practitioners. However, despite a growing evidence base, the implementation of IPE initiatives remains a challenge. Further robust evaluation of IPE initiatives is required to measure its effect on interprofessional collaboration.

> *Without trust and respect, co-operation cannot exist. Assertiveness becomes threatening, responsibility is avoided, communication is hampered, autonomy is suppressed and co-operation is haphazard…*
>
> (Norsen et al. 1995)

Key points

- The rationale for IPE has firm roots in adult educational theory.
- An understanding of adult learning concepts is key for positively received IPE, to ensure learning needs are met through well-structured curriculum development.
- Positive interprofessional contact may change attitudes, may channel conflict and stimulate critical debate, which promotes professional and personal development.
- The optimal time to start IPE continues to induce much debate, but as negative professional stereotyping may evolve early in training IPE should ideally be implemented from the beginning of professional education.
- The role of the educator has been highlighted as key to the success of IPE initiatives.
- IPE educators need to develop and nurture their approach through their own learning in order to educate reflective practitioners who can work collaboratively within the interprofessional healthcare team.
- Institutional and management support, and other resources, such as time, appear to be of paramount importance in founding and maintaining IPE initiatives.
- Delivery of any IPE initiative must be customised and tailored to reflect particular service delivery settings, to provide a positive experience for its targeted practitioners.
- Furthermore, the design of the IPE initiative and the timing of its delivery must be appropriate to match the stage of professional development of its participants.
- Key elements deemed essential for successful collaborative practice are: responsibility and accountability; co-ordination; communication; co-operation; assertiveness; autonomy; and mutual trust and respect.

IPE and technology

Although sceptics question whether it is feasible to learn with, from and about each other through the use of technology, it has become an important means of interaction and communication in the fast-paced world of health and social care practice. For this reason technology-enhanced IPE is highly relevant and its use is increasing. The potential for e-learning IPE approaches to effectively prepare students for collaborative practice is being endorsed (Hughes et al. 2004; Posey and Pintz 2006), although its use is still being developed, refined and tested (Barr 2010). In some instances, where staff are less confident in the use of technology or unconvinced that it will be effective, technology-enhanced IPE is being developed alongside conventional methods of teaching (Williams and Lakhani 2010).

Initiatives utilising technology have generally done so to overcome the challenge of bringing together large groups of students from geographically dispersed locations. These initiatives often involve a blended learning approach where face-to-face interaction is combined with online activities (see, for instance, Atack et al. 2009). Other IPE programmes, such as the joint Coventry University and Warwick Medical School Interprofessional Learning Pathway (IPLP), occur exclusively online (Bluteau and Jackson 2009) accommodating over 1200 students simultaneously. The IPLP is built around learning resources or learning objects, one of which is known as 'the street', which contains a number of families that provide the focus for online interprofessional collaboration. The Wessex Bay web-based simulated community operates in much the same way in promoting active student engagement in case analysis, discussion, debate, collaborative problem-solving and decision-making (Hutchings et al. 2010). Such resources generally act as triggers for synchronous or asynchronous discussion using discussion forums, boards or chat rooms, or Web 2.0 applications, such as blogs and wikis. These tools allow interaction, including varying degrees of social exchange, as well as the testing and exchange of ideas, probing of assumptions, position taking, the expression of different perspectives, informal argumentation and collaborative sense-making – all of which contribute towards critical discourse. One of the advantages of the online approach to IPE is that it provides a safe space in which to test out ideas. While students can be apprehensive about online learning and doubt their information technology skills (Miers et al. 2007), it provides 'anywhere anytime learning' that is attractive to busy students (MacNeill et al. 2010).

The section will help you to work out strategies to start and enhance your skills as a practitioner integral to the team situation. It will give you some practical guidance; it will not make being a practitioner any easier (the more we learn the more we worry we are inadequate), but it might help!

Practice-based learning

Collaboration in the workplace and in the higher education system is not a choice but a directive. In 2000 the DH (DH 2000a) published a review of workforce planning stating that an unhelpful lack of flexibility in the then current ways of working was not helped by working and educational systems that did not encourage multidisciplinary practices. A following document, 'The NHS Plan' (DH 2000b), offered a commitment to interprofessional working. In reviewing its workforce planning the DH produced 'The Knowledge and Skills Framework' (DH 2004), which reviewed workforce planning and outlined the qualities of all health professionals working in the NHS to incorporate a requirement for interprofessional working. The key areas set out in these documents remain current and underpin educational philosophy associated with interdisciplinary working. A summary of these can be seen in Table 2.3.

Simulated learning

Simulated learning opportunities are becoming increasingly utilised in both undergraduate and postgraduate teaching environments. Simulated learning may be based in a skills laboratory with equipment and dummy models, or may be web-based. Learning within simulated practice environments facilitates opportunities to enhance professional practice and to increase students' confidence when in the real clinical setting. In a study based on a simulated ward setting Ker et al. (2003) reported that students gained valuable experience in collaborative team-working, effective leadership, the ability to prioritise workloads and competence in clinical performance. Simulated learning usually involves students working with a facilitator who will be able to guide the student to utilise the learning in a real practice. Students need to be committed to the experience and provide evidence to support the learning achieved. This evidence might be literature related to the underpinning evidence base or evidence to show competencies have been gained in clinical skills. When working with students from other professions the group should be jointly responsible for providing documentation to summarise the learning on the simulated practice. While on clinical practice, students can share learning from simulated practice with their clinical educator, encouraging the link between theory and practice.

In resource-heavy times where there may be difficulties in accessing clinical hours, simulated learning may seem like a panacea and while the pedagogy has undoubtedly much to recommend, caution must also be exerted. Kneebone (2005) emphasises that simulations should not be accepted without critique and that caution should be taken to ensure that emphasis does not focus on the technology alone without thorough regard for the underpinning evidence. He identifies four key areas for simulated learning: gaining technical proficiency; the place of expert assistance; learning within a professional context; and the affective component of learning that is the effect of an emotive component. He proposes that simulations present opportunity to practise within a safe environment; facilitating consolidation of skills, they should be supported by a tutor with tutor support being reduced as the student becomes more adept. If these premises are followed then simulated learning can offer a link to real-life clinical experience, providing a supportive, motivational and learner-centred environment that will ultimately benefit the service user.

Table 2.3 Key policy drivers for interprofessional working [adapted from NHS plan (DH 2000b)]

All NHS staff must have a curriculum (undergraduate and postgraduate) to enable them to have skills and knowledge to respond effectively to the individual needs of patients
Joint training should occur across professions in communication skills and in NHS principles and organisation
All health professionals should expect their education and training to include common learning with other professions
Common learning should take place in practice placements, as well as in the classroom
Common learning should centre on the needs of patients Multi-skilled teams and networks will be based primarily in communities but supported by, and include, people working in hospitals
Leadership and in working in multi-disciplinary settings a focus of education and training programmes

Problem-based learning

Higher education institutes in the UK utilise a range of different pedagogical methods. Problem-based learning (PBL) is a style used in whole, or in part, on many physiotherapy programmes, regardless of whether or not this is the type of learning experience practised by your educators. A consideration of the approach can be helpful for you as a student as the concept transfers readily into the clinical practice setting. PBL was first developed three decades ago by Barrows and Tamblyn at McMaster University during the 1980s (Major and Palmer 2001: 2). The purpose of this type of learning was to encourage students to be able to respond to real-life clinical situations by drawing upon transferrable knowledge and skills, thus maximising the ability to respond appropriately to the specific needs of a particular individual while responding to the needs of the current context, such as resource constraints or other professionals' skills mix.

Savin-Baden (2000: 5–6) states that PBL depends on students acknowledging responsibility for learning – drawing from experience while intertwining theory and practice. The focus is on processes rather than knowledge acquisition gained through communication and interpersonal skills.

What does PBL mean to the learner? It offers you, the student, the opportunity to critically analyse a situation that might be based on a case study or a scenario, and, in particular, to engage in this learning with others from different professions. Thus, as well as coming to a conclusion on what you might do, you will also be introduced to the multi-dimensional aspect of what others might do; together you will devise a much more informed approach. According to Thompson (2010), there is evidence to suggest that PBL in an interprofessional setting encourages improved attitudes towards other professionals, which she suggests will lead to improved care of the service user as central to the practice of all health professionals.

Student-led interprofessional learning

There are many ways in which students can become proactive interprofessional learners, be it in the clinical or in the university setting. Contemporary methods of communication mean that it has never been easier to form a network of fellow learners. Online networks can offer a stimulating and safe environment for the development of a range of professional skills. In order for students to lead in a learning context it is important to understand that:

- there is always more than one way to deal with a situation;
- no two people will react in exactly the same way to a given situation;
- the way that you react to an incident really matters;
- by reflecting on interprofessional learning incidents you will be better able to cope with similar episodes in the future;
- a positive attitude and self-esteem will enable you to strive for excellence;
- whatever other factors are present, the service user must always be the most important entity.

In student-led interprofessional learning the mentor does not need to be a tutor or health professional – another student may take on this role. A study by Clouder et al. (2010) suggested that student learning is optimised by peer mentoring. They found that where students across a range of disciplines engaged in web-based interprofessional online discussion forums the role of mentor was valued by other students who, at times, found the student added value compared with a professional mentor. A student mentor can offer empathy and awareness of contemporary issues affecting other students, for example, of which a tutor may not have the same insight.

It is important for interprofessional learning to be maximised so that students engage; sometimes there is a reluctance to join in this type of forum. To see the value of this experience join an existing scheme or start your own. Reflect on the benefits that you gain from the experience and feed these back to others. People influence one another – the way that we act as an individual may be very different to the ways in which we act when part of a group. In order to strive to be able to perform in a way that fits both our own values and those of the profession it is useful to gain understanding in the ways in which we and others might be influenced in the group setting of an interprofessional team – the more who engage the better the experience will be!

PROFESSIONAL IDENTITY AND INTERPROFESSIONALISM

Wackerhausen (2009) considers professional identity at macro- and micro-levels. The macro-level is the public face of the profession formed through the profession's official recognition; in other words, its regulations, privileges and duties, public perceptions, related professions' views, and the self image promoted by the profession's leaders and ideologists. Macro professional identity is continually changing as identity is negotiated. Professions collaborate out of necessity, but also compete with one another and have goals that Wackerhausen argues do not promote genuine collaboration.

Micro-level professional identity is dependent on exhibiting the qualities that a person needs to exhibit to be a fully acknowledged member of the profession, as well as the behaviour consistent with the profession's 'cultural dimensions'. He refers to 'a way of speaking, a way of questioning, a way of understanding and explaining, a way of seeing and valuing, a way of telling narratives' and the obligation of the student to become 'one of our kind' (Wackerhausen 2009: 461). Wackerhausen argues that professional identity hampers interprofessional collaboration at both levels. At the micro-level habitual ways of talking, perceiving, valuing, doing and assuming conflict with other professions while, at the macro-level, boundaries and barriers are reinforced by negative narratives and competition. This rather depressing outlook is that 'self complacent, know-all practitioners and professionals see interprofessional collaboration more as an (un)avoidable evil than a desirable necessity' (Wackerhausen 2009).

Is it inevitable that professional identity impacts negatively on IPE and interprofessional working? We believe not. As students are socialised into a profession they are gradually shaped to take on a professional identity

associated with that profession, becoming a nurse, a medic or a physiotherapist. Socialisation is 'the process by which people selectively acquire the values and attitudes, the interests, skills and knowledge – in short – the culture current in groups in which they are, or seek to become, a member' (Merton et al. 1957: 278). The process has been described as 'indoctrination' (Sparkes, 2002) and certainly involves elements of 'internalization' (Hayden 1995) of professional norms.

Inevitably, different training and philosophical approaches have resulted in the professions evolving separately (Fitzsimmons and White 1997) and in the professions being described as 'warring tribes' (Becher and Trowler 2001). The sense of shared identity that develops in groups promotes what William Sumner identified in 1906 as 'in-group' and 'out-group' dynamics (Brewer 1979). Social identity theory explains how such dynamics effect intergroup relations; people seek for their in-group a positive distinctiveness from out-groups; in other words, they distinguish between 'us' and 'them' (Tajfel et al. 1971). However, research suggests that rather than individuals who identify strongly with their in-group – ranking out-groups more negatively, as might be expected – those who are positive about their in-group are also positive about other groups (Hind et al. 2003). This finding is attributed to wider contextual factors, such as membership of diverse student groups of healthcare professionals. These students were also more positive about IPE, suggesting that a strong professional identity does not mitigate against students' willingness to engage positively in it. This finding supports Clouder's (2003) conception of 'socialisation as interaction' based on social constructionist theories that suggest that society creates individuals, who, in turn, create society (Berger and Luckman 1966). Socialisation as interaction allows socialisation to be seen in a less deterministic way, especially if, as is advocated by the IPE movement, students are exposed to one another in a way that allows them to develop awareness of their profession in the context of others.

Clouder's research findings suggest that repressive and deterministic socialisation processes are mitigated by the potential for individual agency. Rather than being enveloped by the influence of professional discourses, 'scarcely aware of changes to their own identity' (Clouder 2003: 220), she has suggested that they are capable of individual agency able to make choices; options include conformity and deviance, commitment or non-commitment, attraction or aversion (Vanderstraeten 2000). Clouder suggests that such choices result in individuals' differential positioning within their professional culture, either identifying wholly with it or sitting on its fringes. Fringe positions may be conducive to making connections with others in different professions as they are inhabited by individuals who have decided where they sit within their profession, who then have the confidence to explore wider interprofessional practice.

Calls to discuss the need for 're-professionalisation' are based on the recognised need for individuals to shift their identity and commitments from being profession-focussed to the organisation in which they work (Hafferty and Light 1995). The previous discussion suggests that professional identity might be the foundation on which individuals can go forward to optimise teamwork, although research by Baxter and Brumfitt (2008) suggests that, again, contextual factors, such as team size and regular contact, were important in developing team identity, rather than professional identity. Professional identity and its interaction with interprofessional relations and interprofessional learning is clearly complex. In agreement with Kreber (2010), we suggest that it might be helpful to think of (professional) identity formation as essentially intersubjective, dialogical and relational in nature. Teamwork is an important element, but, perhaps more importantly, Wackerhausen (2009: 471) reminds us that 'our professional means come and go but our professional *raison d'etre* stays. That is, relentless searching for and using the most excellent means available to do the best we possibly can for the patient'. Accepting this *raison d'etre* is part of our professional identity that takes us closer to effective interprofessional practice.

CONCLUSION

The importance of IPE in preparing undergraduates for a world of work where co-ordinated collaborative practice is the norm should not be underestimated. By engaging in IPE students are able to experience, in a safe environment, the complexity of joint working and the ways in which individual professional sensitivities can, and do, get in the way of decision-making, as well as culminating in a less linked and more fragmented experience for patients. IPE is a useful precursor to improved collaborative working; today's practitioners need to be capable of working across boundaries and providing seamless holistic patient care with colleagues. In 1988, the WHO called for IPE on the basis that learning together translates into improved working together (WHO 1998).

This chapter has sought to provide the reader with an overview of the current thinking around IPE within the context of contemporary health and social care practice, as well as instil in the reader a sense of its importance in the preparation of graduate health and social care practitioners.

As the historical perspective shows, IPE is not a new concept and yet there is still much work to be done on securing a robust evidence base to support the investment both educators and practitioners put into its ongoing inclusion in education programmes, which both prepares new graduates and supports the ongoing professional development of the health and social care workforce.

The authors of this chapter have highlighted the importance of IPE in the undergraduate curriculum to help prepare graduates for a world of work where collaborative practice is becoming increasingly the norm. The publication of the most recent NHS white paper: 'Liberating the NHS' (DH 2010a) reinforces the requirement for collaborative practice in health and social care. For all practitioners this means that every opportunity to develop those skills required for effective collaborative practice should be seized.

For undergraduates concerned with learning to be a particular type of professional with all the inherent uni-professional requirements this demands, the inclusion of IPE can sometimes be viewed as less valuable than those uni-professional aspects. The authors of this chapter hope to convince readers that IPE is an important aspect of their undergraduate education. It is possible to learn to be a particular type of professional within an interprofessional learning context.

Learning to be an effective physiotherapist in the twenty-first century necessitates active engagement in IPE. IPE provides opportunities to practise and experience collaborative working in a safe environment. Learning is a process not performed in isolation from others. As Wenger (1998) asserts, learning involves collaboration and engagement between learners, each influencing and, in turn, being influenced by each other; learning outcomes become shared by communities of practice. IPE is a natural place for this to occur.

Social learning theories (Wenger 1998) suggest that learning with, and through, interaction with others builds communities of practice. This chapter has tried to highlight the importance of interprofessional education as a way of developing the knowledge skills required for collaborative working. It can be argued that building communities of learning through engagement in IPE is the best way to develop such knowledge and skills. IPE communities of learning provide a safe environment where undergraduates can test out their own professional role and identity alongside peers from other professional groups doing the same. Collaborative learning, practice and work are never easy, and are difficult to negotiate on a daily basis. However, it is imperative that today's practitioners are able to practise such professional negotiation on a daily basis if they are to practise person-centred health and social care. IPE offers a vehicle to support this and through collaboration between professionals lead to:

- improved services;
- improved health outcomes;
- effecting change.

The importance of working closely with other professionals to provide care that is truly focussed upon people and their families cannot, and should not, be underestimated (DH 2000b; Wilcock et al. 2009). Being an effective physiotherapist means being equipped with the knowledge, skills and values distinctive to physiotherapy, and recognising, understanding and valuing the knowledge skills and values distinctive to other professional groups. Physiotherapists who are confident in their own professional knowledge, skills and values will be able to recognise when they are the best professional for the job and when it is more appropriate for another professional to do the job. IPE as a mechanism for promoting more effective collaboration and team-working can help to prepare graduates for the daily negotiation of professional boundaries as an integral part of work in order to provide patient-centred care.

The benefits of IPE are well documented and this chapter has introduced readers to the history, drivers, key concepts and practices of IPE alongside an exploration of some of the complexities inherent in any collaborative endeavour (including IPE). The authors acknowledge the need for more evidence to substantiate the benefits of IPE as a precursor to effective collaborative work practices; however, the growing evidential base (Horder 2004; Bokhour 2006; Stone 2006; Lennox and Anderson 2007;

Weblinks

Australasian Interprofessional Practice and Education Network (AIPPEN): http://aippen.net/index.html
Canadian Interprofessional Health Collaborative (CIHC): www.cihc.ca
Centre for the Advancement of Interprofessional Education (CAIPE): www.caipe.org.uk
European Interprofessional Education Network (EIPEN): www.eipen.eu
Global Health Workforce Alliance: www.who.int/workforcealliance/en
Health Professions Global Network: http://hpgn.org/
International Association for Interprofessional Education and Collaborative Practice (InterEd): www.interedhealth.org
Japan Interprofessional Working and Education Network (JIPWEN): http://jipwen.dept.showa.gunma-u.ac.jp/
Journal of Interprofessional Care: http://informahealthcare.com/jic
National Health Sciences Students' Association (NaHSSA): http://nahssa.ca/en/gateway
Nordic Interprofessional Network (NIPNet): http://nipnet.org/
The Network: Towards Unity for Health: www.the-networktufh.org
World Health Organization (WHO): www.who.int/en
London Deanery: Interprofessional Education http://www.faculty.londondeanery.ac.uk/e-learning/interprofessional-education/Interprofessional%20education.pdf

Clarke 2006; Cooper and Spencer-Dawe 2006) is testament to the enduring interest in IPE. Current political and societal drivers (DH 2010a, 2010b) continue to ensure that IPE and interprofessional working remain a high priority in health and social care. As West et al. (2006) assert, more positive patient outcomes are realised through greater and more effective collaboration between professionals. IPE that puts patients at the centre promotes collaboration between professionals, reinforces professionals' collaborative competence and relates collaborative learning to collaborative practice (Horder 2004; Hendrick and Khaleel 2008; Wilcock, 2009).

REFERENCES

Allport, G.W., 1954. The Nature of Prejudice. Addison-Wesley, Cambridge, MA.

Atack, L., Parker, K., Rocchi, M., et al., 2009. The impact of an online interprofessional course in disaster management competency and attitude towards interprofessional learning. J Interprof Care 23 (6), 586–598.

Barker, K.K., Bosco, K., Oandasan, I.F., 2005. Factors in implementing interprofessional education and collaborative practice initiatives: findings from key informant interviews. J Interprof Care 19 (Suppl. 1), 166–176.

Barnes, D., Carpenter, J., Dickinson, C., 2000. Interprofessional education for community mental health: attitudes to community care and professional stereotypes. Social Work Educ 19, 565–583.

Barr, H., 2005. Evaluation, Evidence and Effectiveness. J Interprof Care 19 (6), 535–536.

Barr, H., 2007. OCC 8: Piloting Interprofessional Education-Four English Case Studies. The Higher Education Academy, London.

Barr, H., 2007. OCC 9: Interprofessional Education in the United Kingdom 1966 to 1997. The Higher Education Academy, London.

Barr, H., 2009. Interprofessional education as an emerging concept. In: Bluteau, P., Jackson, A. (Eds.), Interprofessional Education: Making It Happen. Palgrave Macmillan, Basingstoke, pp. 3–23.

Barr, H., 2010. Foreword. In: Bromage, A., Clouder, D.L., Thistlethwaite, J., et al., (Eds.), Interprofessional e-Learning and Collaborative Work: Practices and Technologies. IGI Global, Hershey, PA, pp. xxi–xxii.

Barrett, G., Greenwood, R., Ross, K., 2003. Integrating interprofessional education into 10 health and social care programmes. J Interprof Care 17 (t), 293–301.

Baxter, S.K., Brumfitt, S.M., 2008. Professional difference in interprofessional working. J Interprof Care 22 (3), 239–251.

Becher, T., Trowler, P., 2001. Academic Tribes and Territories, second ed. SRHE and Open University, Milton Keynes.

Berger, P., Luckman, T., 1966. The Social Construction of Reality: A Treatise in the Sociology of Knowledge. Penguin, Harmondsworth.

Bjorke, G., Haavie, N.E., 2006. Crossing boundaries: Implementing an interprofessional module into a uniprofessional bachelor programme. J Interprof Care 20 (6), 641–653.

Bluteau, P., Jackson, A., 2009. An eLearning model of interprofessional education. In: Bluteau, P., Jackson, A. (Eds.), Interprofessional Education: Making It Happen. Palgrave Macmillan, Basingstoke, pp. 107–121.

Bokhour, B.G., 2006. Communications in interdisciplinary team meetings: what are we talking about? J Interprof Care 20 (3), 260–275.

Brewer, M.B., 1979. In-group bias in the minimal intergroup situation: A cognitive-motivational analysis. Psychol Bull 86 (2), 307–324.

Bruffee, K.A., 1999. Collaborative Learning: Higher Education, Interdependence, and the Authority of Knowledge, second ed. John Hopkins University Press, London, Baltimore, MD.

Buring, S., Bhushan, A., Brazeau, G., et al., 2009. Keys to successful implementation of interprofessional education: Learning, location, faculty development, and curricular themes. Am J Pharm Educ 73 (4), 60.

CAIPE (Centre for the Advancement of Interprofessional Education), 1997. Interprofessional Education. A Definition. Bulletin no. 13. UK CAIPE, London.

Carpenter, J., 1995. Interprofessional education for nursing and medical students: Evaluation of a programme. Med Educ 29, 265–273.

Carpenter, J., Hewstone, M., 1996. Shared learning for doctors and social workers: evaluation of a programme. Br J Soc Work 26, 239–257.

Chambers, A., 2009. Becoming a Physiotherapist: Andrew's Story. The Narrative Practitioner Conference, Keele.

Chambers, A., Grey, J., McGlen, I., 2008. Using Service Improvement Learning as a Catalyst for Meaningful Interprofessional Learning. Chartered Society of Physiotherapy Annual Congress Poster Presentation, Manchester.

Clarke, P.G., 2006. What would a theory of interprofessional education look like? Some suggestions for developing a theoretical framework for team training. J Interprof Care 20 (6), 577–590.

Cleake, H., Williamson, D., 2007. Preparing health science students for interdisciplinary professional practice. J Allied Health 36 (3), 141–149.

Clouder, D.L., 2003. Becoming professional: Exploring the complexities of professional socialization in health and social care. Learning in Health and Social Care 2 (4), 213–222.

Clouder, L., Krumins, M., Davies, B., 2010. Interprofessional learning: Exploring the benefits of engaging students in online peer mentoring; http://www.health.heacademy.ac.uk/rp/publications/projectreports/summaries/lclouderexecsum.html, accessed October 2012.

Colwell Report, 1974. Report of the Committee of Enquiry into the Care and Supervision Provided in Relation to Maria Colwell. Her Majesty's Stationery Office, London.

Cooper, H., Spencer-Dawe, E., 2006. Involving service users in interprofessional education narrowing the gap between theory and practice. J Interprof Care 20 (6), 603–618.

Coulshed, V., 1993. Adult Learning: Implications for Teaching in Social Work Education. Br J Soc Work 23 (1), 1–13.

Davidoff, F., Batalden, P., 2005. Toward stronger evidence on quality improvement. Draft publication guidelines: the beginning of a consensus project. Qual Saf Health Care 14 (5), 319–325.

DH (Department of Health), 1989. Community Care in the Next Decade and Beyond. Her Majesty's Stationery Office, London.

DH (Department of Health), 1991. Working Together: A Guide to Arrangements for Inter-Agency Co-operation for the Protection of Children from Abuse. DH, London.

DH (Department of Health), 1994. The Report of Enquiry into the Care and Treatment of Christopher Clunis. Her Majesty's Stationery Office, London.

DH (Department of Health), 2000a. A Health Service for all Talents; Developing the NHS Workforce. Her Majesty's Stationery Office, London.

DH (Department of Health), 2000b. The NHS Plan: A Plan for Investment, A Plan for Reform. Her Majesty's Stationery Office, London.

DH (Department of Health), 2001. Working Together, Learning Together: A Framework for Lifelong Learning for the NHS. Her Majesty's Stationery Office, London.

DH (Department of Health), 2004. The NHS Knowledge and Skills Framework. Her Majesty's Stationery Office, London.

DH (Department of Health), 2006. Our Health, Our Care, Our Say. Her Majesty's Stationery Office, London.

DH (Department of Health), 2007. Creating an interprofessional workforce. An education and training framework for health and social care in England. Her Majesty's Stationery Office, London.

DH (Department of Health), 2009. High Quality Care for All: Our Journey So Far. Her Majesty's Stationery Office, London.

DH (Department of Health), 2010a. Equity and Excellence: Liberating the NHS. Her Majesty's Stationery Office, London.

DH (Department of Health), 2010b. Equity and Excellence: Developing the NHS Workforce. Her Majesty's Stationery Office, London.

Finch, J., 2000. Interprofessional education and team working: a view from the education providers. BMJ 321, 1138–1140.

Fitzsimmons, P., White, T., 1997. Crossing boundaries: Communication between professional groups. J Manag Med 11 (2), 96–101.

Freeth, D., Hammick, M., Reeves, S., et al., 2005. Effective Interprofessional Education: Development, Delivery and Evaluation. Blackwell, Oxford.

Gilbert, J.H.V., 2010. The global emergence of IPE and collaborative care. J Interprof Care, 24 (5), 473–474.

Gilbert, J.H.V., Camp, R.D., Cole, C.D., et al, 2000. Preparing students for interprofessional teamwork in health care. J Interprof Care 14 (3), 223–235.

Hafferty, F., Light, D., 1995. Professional dynamics and the changing nature of medical work. J Health Soc Behav 35, 132–153.

Hammick, M., Barr, H., Freeth, et al., 2002. Systematic reviews of evaluations of interprofessional education: Results and work in progress. J Interprof Care 16 (1), 80–84.

Hammick, M., Freeth, D., Koppel, I., et al., 2007. A best evidence systematic review of interprofessional education: BEME Guide no 9. Med Teach 29, 735–751.

Hayden, J., 1995. Professional socialization and health education preparation. J Health Educ 26, 271–278.

HCPC (Health and Care Professions Council), 2004. Key Decisions from our Consultation on Standards of Education and Training and the Approvals Process. http://www.hpc-uk.org/assets/documents/1000057Fkey_decisions.pdf; accessed October 2012.

HCPC (Health and Care Professions Council), 2012. Standards of proficiency - Physiotherapists. http://www.hpc-uk.org/assets/documents/10000DBCStandards_of_Proficiency_Physiotherapists.pdf; accessed October 2012.

Health Canada, 2003. First Ministers' Accord on Health Care Renewal; http://www.hc-sc.gc.ca/hcs-sss/delivery-prestation/fptcollab/2003accord/index-eng.php; accessed October 2012.

Hean, S.J., Macleod, J., Adams, K., et al, 2006. Will opposites attract? Similarities and differences in students' perceptions of the stereotype profiles of other health and social care professional groups. J Interprof Care 20 (2), 162–181.

Hendrick, L.A., Khaleel, N.I., 2008. Getting it right, educating professionals to work together in improving health and social care. J Interprof Care 22 (4), 364–374.

Hind, M., Norman, I., Cooper, S., et al., 2003. Interprofessional perceptions of health care students. J Interprof Care 17 (1), 21–34.

Horder, J., 2004. Interprofessional collaboration and interprofessional education. BMJ 54, 243–245.

Hughes, M., Ventura, S., Dando, M., 2004. Online interprofessional learning: Introducing constructivism through enquiry based learning and peer review. J Interprof Care 18 (3), 263–268.

Hutchings, M., Quinney, A., Scammell, J., 2010. The utility of disruptive technologies in interprofessional education: Negotiating the substance and spaces of blended learning. In: Bromage, A., Clouder, D.L., Thistlethwaite, J., et al., (Eds.), Interprofessional e-Learning and Collaborative Work: Practices and

Technologies. IGI Global, Hershey, PA, pp. 190–203.

Jarvis, P., 1983. Adult Learning in the Social Context. Croom Helm, Beckenham.

Jenkins, R., 2004. Social Identity, second ed. Routledge, London.

Ker, J., Mole, L., Bradley, P., 2003. Early introduction to interprofessional learning: a simulated ward environment. Med Educ 37 (3), 248–255.

Kilminster, S., Hale, C., Lascelles, M., et al., 2004. Learning for real life: patient focused interprofessional workshops offer added value. Med Educ 38, 717–726.

Kneebone, R., 2005. Evaluating clinical simulations for learning procedural skills: A theory-based approach. Acad Med 80 (6), 549–553.

Kreber, C., 2010. Academics' teacher identities, authenticity and pedagogy. Stud High Educ 35 (2), 171–194.

Kuenssberg, E., 1967. Conference Report. Family Health Care: The Team. Royal College of General Practitioners, London.

Laming, 2003. The Victoria Climbié Inquiry. Report of an Inquiry by Lord Laming. Her Majesty's Stationery Office, London.

Laming, 2008. The Protection of Children in England: A Progress Report. Her Majesty's Stationery Office, London.

Lennox, A., Anderson, E., 2007. Special Report 9: The Leicester Model of Interprofessional Education. The Higher Education Academy, London.

McMichael, P., Gilloran, A., 1984. Exchanging Views: Courses in Collaboration. Moray House College of Education, Edinburgh.

McNair, R., Brown, R., Stone, N., et al., 2001. Rural interprofessional education: promoting teamwork in primary health care education and practice. Aust J Rural Health 9 (Suppl.), 19–26.

MacNeill, H., Reeves, S., Hanna, E., et al., 2010. The Community of Inquiry Framework: A pertinent theory of online interprofessional education? In: Bromage, A., Clouder, D.L., Thistlethwaite, J., et al. (Eds.), Interprofessional e-Learning and Collaborative Work: Practices and Technologies. IGI Global, Hershey, PA, pp. 75–89.

Major, C., Palmer, B., 2001. Assessing the effectiveness of problem based learning in higher education: Lessons from the literature. Academic Exchange Quarterly 5 (4), 4–11.

Merton, R.K., Reader, G.G., Kendall, P.L., 1957. The Student Physician. Harvard University, Cambridge, MA.

Mhaolrunaigh, S.N., 2001. An evaluation of interprofessional education for health and social care professionals: The teachers' views. PhD Thesis, University of Warwick.

Miers, M.E., Clarke, B.A., Pollard, K.C., et al., 2007. Online Interprofessional Learning: The Student Experience. J Interprof Care 21 (5), 529–542.

Miller, C., Freeman, M., Ross, N., 1999. Interprofessional Practice in Health and Social Care: Challenging the Shared Learning Agenda. Arnold, London.

NMC (Nursing and Midwifery Council), 2004. A standard to support learning and assessment in practice. NMC, London; http://www.nmc-uk.org/Documents/Standards/nmcStandardstoSupportLearning%20AndAssessmentInPractice2008.pdf; accessed October 2012.

Norsen, L., Opladen, J., Quinn, J., 1995. Practice model: Collaborative practice. Crit Care Nurs Clinics North Am 7, 43–52.

Pietroni, P., 1992. Towards reflective practice; the languages of health and social care. J Interprof Care 6 (1), 7–16.

Ponzer, S., Hylin, U., Kusoffsky, A., et al., 2004. Interprofessional training in the context of clinical practice: goals and student's perceptions on clinical education wards. Med Educ 38, 727–736.

Posey, L., Pintz, C., 2006. Online teaching strategies to improve collaboration among nursing students. Nurs Educ Prac 6, 372–379.

QAA (Quality Assurance Agency), 2006. Statement of common purpose for subject benchmark statements for the health and social care professions; http://www.qaa.ac.uk/Publications/InformationAndGuidance/Documents/StatementofCommonPurpose06.pdf; accessed October 2012.

Ragucci, K.R., Steyer, T., Wager, K.A., et al., 2009. The Presidential Scholars Program at the Medical University of South Carolina: An extracurricular approach to interprofessional education. J Interprof Care 23 (2), 134–147.

Ramsammy, L., 2010. Interprofessional Education and collaborative practice. J Interprof Care 24 (2), 131–138.

Savin-Baden, M., 2000. Problem-based Learning in Higher Education: Untold Stories. Society for Research into Higher Education and Open University Press, Buckingham.

Smith, S., Green, M., 2003. A Qualitative Study Investigating the Development of Team Working Skills in First Year Physiotherapy Students; http://repos.hsap.kcl.ac.uk/content/m10210/latest/02-08_susansmith.pdf, accessed October 2012.

Sparkes, V.J., 2002. Profession and professionalisation. Physiotherapy 88 (8), 481–492.

Spratley, J., 1990. Disease Prevention and Health Promotion in Primary Care. Health Education Council, London.

Stone, N., 2006. Evaluating Interprofessional Education; the tautological need for interdisciplinary approaches. J Interprof Care 20 (3), 235–245.

Street, K.N., Eaton, N., Clarke, B., et al., 2007. Child disability case studies: an interprofessional learning opportunity for medical students and paediatric nursing students. Med Educ 41, 771–780.

Tajfel, H., Billig, M.G., Bundy, R.P., et al., 1971. Social categorization and intergroup behaviour. Eur J Soc Psychol 1, 149–178.

Takahashi, H.E., Sato, S., 2009. Establishment of the Japan Association for Interprofessional Education and Perspectives. J Interprof Care 23 (6), 554–555.

Thompson, C., 2010. Curriculum: Do interprofessional education and problem-based learning work together? Clin Teach 7, 197–201.

Vanclay, L., 1997. Exploring Interprofessional Education: The Advantages and Barriers. A discussion paper for the UKCC Multi-professional Working

Group of the Joint Education Committee.

Vanderstraeten, R., 2000. Luhmann on Socialization and Education. Educational theory, 50 (1), 1–23.

Wakerhausen, S., 2009. Collaboration, professional identity and reflection across boundaries. Journal of Interprofessional Care 23 (5), 455–473.

Walker, P., Baldwin, D., Fitzpatrick, J., et al., 1998. Building community: Developing skills for interprofessional education and relationship-centered care. J Gerontol Nurs March, 45–49.

Wanless, D., 2004. Securing good health for the whole population: Final report. Her Majesty's Stationery Office, London.

Way, D.O., Busing, N., Jones, L., 2002. Implementing Strategies: collaboration in primary care – family doctors and nurse practitioners delivering shared care. Ontario College of Family Physicians, Toronto.

Wenger, E., 1998. Communities of Practice, Learning, Meaning and Identity. Cambridge University Press, Cambridge.

West, M., 2006. Reflection in action: Developing reflective practice in health and social services. J Interprof Care 20 (2), 213–214.

WHO (World Health Organization), 1988. Learning Together to Work Together for Health. Technical report series 769. WHO, Geneva.

WHO (World Health Organization), 1998. Life in the 21st century: A vision for all. WHO, Geneva.

WHO (World Health Organization), 2010. Framework for Action on Interprofessional Education and Collaborative Practice. WHO, Geneva.

Wilcock, P.M., Janes, G., Chambers, A., 2009. Health care improvement and continuing interprofessional education: Continuing interprofessional development to improve patient outcomes. J Contin Educ Health Prof 29 (2), 84–90.

Williams, J., Lakhani, N., 2010. E-learning for interprofessional education: A challenging option. J Interprof Care 24 (2), 201–203.

Chapter 3

Clinical leadership

Alison Chambers

INTRODUCTION

> *The NHS, like all health care systems, is the sum total of the people who work in it and the day-to-day interactions they have with patients and colleagues. A health care system is a powerful coalition of organisations and professions, not just a set of individual organisations operating in a market. The way to achieve transformation is through the mobilisation of our staff to drive change. For this to happen, our clinical and managerial leaders need to understand what it is that we are trying to do, and how it connects with our wider values.*
>
> (Sir David Nicholson, NHS Chief Executives Annual Report, 2007)

The purpose of this chapter is to introduce readers to the concept of clinical leadership within the context of contemporary healthcare and suggest ways in which clinical leadership can help to support and promote improved health outcomes for patients. It is essentially an introduction to the idea that clinical leadership is an integral aspect of every health professional's professional day-to-day practice.

While the need for strong and effective leadership in healthcare is well recognised there is still the need for ongoing exploration of how leadership in healthcare contributes to improving health outcomes (Hardacre et al. 2010). Work by Hardacre et al. suggests that leadership for improvement is culturally sensitive, inclusive, team-based, personal and collective (p. 27). There is, however, a growing body of evidence that links leadership, culture in organisations and improved outcomes and quality (Corrigan 2000). Research into nursing leadership (Edmonstone 2011) suggests a direct link between effective nurse team leadership and the quality of team work. In essence, effective leadership is seen as essential to improving health outcomes. National Health Service (NHS) organisations have and continue to invest significant amounts of resources to developing existing and aspiring leaders, and leadership competence in healthcare. Work by Hardacre et al. (2010) suggests that some leadership behaviours appear to be positively associated with service improvement work and that service improvement work is beginning to show links to improved health outcomes.

The chapter includes some real life leadership stories that help to bring to life the day-to-day experiences of clinical leaders. These stories are powerful in that they enable real life experiences to be shared to show how different approaches to leadership are enacted in practice. What these stories also do is demonstrate how leaders cannot achieve anything alone, that leaders need teams in order to help them achieve their goals. In the words of Mike Farrar (Chief Executive, NHS North West):

> *Today's NHS leaders don't face their multiple challenges alone: our leaders have teams, directorates or organisations to meet the challenges and achieve goals. Our job as leaders is not to come up with solutions alone, but to inspire people we lead to place ladders together, against appropriate walls…*
>
> (Hartley and Bell 2009)

Narrative research utilises stories as a way of exploring and, therefore, understanding more fully the lived

experiences of human beings (Mishler 1990, 1999; Reissman 1997). Leadership narratives are very powerful as a way of understanding human action and interaction. The stories of real leaders doing the very real job of leading NHS organisations have influenced the writing of this chapter.

What this chapter does not do is provide the reader with leadership theory. Readers are able to access leadership theory through a wide range of leadership and management textbooks, and readers are directed to Jumaa and Jasper (2005) and Jarvis et al. (2003) as a starting point. Constraints of space and constraints of purpose have allowed the author of this chapter to take a practical approach to the subject, drawing upon published literature but, more importantly, drawing upon real examples of clinical leadership. In this way, the chapter attempts to bring clinical leadership to life and to drive home its importance to readers.

By using the NHS leadership qualities framework (LQF) (NHSi 2009) alongside broader leadership concepts, the chapter includes descriptions and illustrations of clinical leadership in contemporary healthcare. It will provide a brief overview of the healthcare reform context (readers are directed to other chapters in this book for more detail), describe, in some detail, the elements of the LQF and, through this, explore some of the implications for today's physiotherapists. The chapter concludes with some practical advice on how physiotherapists can take personal responsibility for developing their leadership credentials.

When reading the leadership literature it is apparent that the concept of leadership is used in a wide variety of ways and tends to be described in behavioural terms (Jumaa and Jasper 2005; McDonald et al 2009; Hardacre 2010; King 2010; Edmonstone 2011). This varied use of nomenclature and definition can be confusing for those new to leadership writings. Put simply, leadership is concerned with individuals and teams achieving what it is they set out to achieve. The leader is someone who provides the focus and sets the direction of travel but achieves an outcome through the collective actions of the whole team.

> *The very essence of leadership is its purpose. And the purpose of leadership is to accomplish a task. That is what leadership does – and what it does is more important than what it is and how it works…*
>
> (Col. Dandridge Malone)

CONTEXT

Contemporary healthcare is complex and undergoing the biggest change since its inception in 1948 (DH 2000, 2001, 2008, 2010). Although we all think of the NHS as one big organisation, in reality it is made up of many different organisations, each with its own culture and ways of working. These NHS organisations have been undergoing a radical change programme for over ten years and are currently in the middle of another reorganisation, probably its most radical yet. As the government's white paper (2010) states, at its best the NHS provides world class care; however, there is still some way to go before world class care is experienced throughout the vast organisation(s) that we call the NHS.

Just like any other large organisation in today's climate of austerity and economic challenge, the NHS is charged with ensuring efficient and effective working, raising standards and redesigning services to improve the quality of the patient experience (DH 2008, 2010). This is a particularly challenging time for the NHS: redesigning services and the workforce that delivers the service, raising standards, and improving efficiencies and real-term savings requires strong leadership. Leadership is not just the responsibility of the most senior managers but the responsibility of all staff, of whatever level, who work within the NHS and who are charged with improving healthcare outcomes (NHS North West Leadership Academy 2008; DH 2008). Also, the creation of a new Public Health Service located within local government rather than as an integral part of the NHS will require professionals to work much more collaboratively, with a greater impetus toward ensuring that multi-service, interprofessional, inter-agency working and collaboration are the norm (DH 2010). Readers are directed to Chapter 2 for more details and references on collaborative working.

All organisations need financial investment, technical knowledge, access to a market, political impetus, professional expertise and quality equipment, etc. (NHSi 2009) and the NHS is no different. All organisations need leaders who can utilise resources effectively and efficiently in order to create success (NHS Alliance 2007). Strong leadership creates an environment where success is more likely to happen. The size and scale of NHS reorganisation and proposed health reform creates a highly challenging and complex context. This context creates an environment where many organisations are focussing on identifying and developing existing and potential leaders as a way of effectively leading and managing the reform agenda. Arguably, organisations that survive are those that have a strong focus on leadership development practices and have a shared understanding of what good leadership means (NHS Alliance 2007; McDonald et al. 2009; Towill 2009). A lot of attention has been focussed upon developing individual leaders; Edmonstone (2011) calls for a rebalancing to refocus attention on context and relationships rather than individual leaders, thus emphasising the importance of a collaborative approach to improving services through a distributed leadership model (NHS North West Leadership Academy 2008; NHSi 2009).

The Department of Health's (DH) 'High Quality Care for All: Next Stage Review Final Report' and the 'High

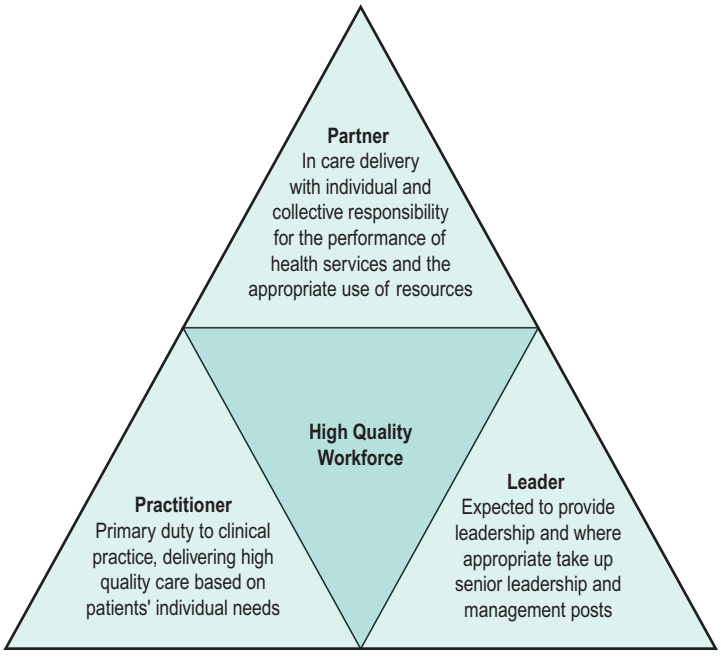


Figure 3.1 Contributory factors to a high quality workforce.

Quality Workforce: Next Stage Review Report' (2008) were two important documents that provided the impetus for leadership development throughout the NHS. These documents put the spotlight on the *quality* of care provided, as well as the concept of *care for all*, as a means of ridding the NHS of inequity and inequality of provision inherent in some parts of the system (DH 2008). The most recent white paper continues to emphasise the centrality of the quality agenda in the provision of patient-centred care. The shift to a truly patient-centred health service – 'no decision about me without me' (DH 2010: 9) – requires clinicians to rise to the leadership challenge.

A 'High Quality Workforce: Next Stage Review' (DH 2008) highlighted three aspects of every clinician's role, now and in the future. These aspects remain relevant today and are likely to remain so for a long time. The three aspects are described as: Practitioner, Partner and Leader. Figure 3.1 describes how each different aspect can contribute to the high quality workforce required to deliver high quality, improved healthcare.

THE ALLIED HEALTH PROFESSIONS AND PHYSIOTHERAPY

The planned NHS reconfigurations as outlined in 'Equity and Excellence: Liberating the NHS' (DH 2010) along with existing NHS service reconfigurations, for example transforming community services and the quality, improvement, prevention and productivity (QUIPP) agenda (DH 2009), provide a context that demands effective leadership.

From an Allied Health Professions (AHP) perspective, leadership development is a high priority. The Department of Health Leadership Challenge events (2009, 2010) are testament to this and readers are directed to the DH website for further details (www.dh.uk). Across England there are regional AHP networks all engaged in regional leadership development as a way of framing the contribution of AHPs in delivering a world-class patient-centred service (e.g. www.nhsnw.uk/ahpnetwork). Also, the AHP career framework (Skills for Health 2007), which was created in response to the 'Modernising Allied Health Professions Careers' project (DH 2002), emphasises the importance of leadership.

The Chartered Society of Physiotherapy's newly published physiotherapy framework (CSP 2010) includes the knowledge, skills and values required of effective clinical leaders. This framework was the product of a lengthy project called 'Charting the Future', and defines and illustrates the knowledge, skills, behaviours and values required for contemporary physiotherapy practice (CSP 2010). This framework not only underpins contemporary practice, it is also the basis from which undergraduate and postgraduate physiotherapy education is developed. It describes physiotherapy practice at all levels, across different professional roles, across a variety of settings and across all four nations of the UK. Readers are directed to www.csp.org.uk for further details.

Key points

- Leadership is seen as being key to the implementation of health reform in the NHS (Firth-Cozens and Mowbray 2001; Gobillot 2007; Hartley and Bell 2009).
- Government health policy reform calls for increased clinical leadership to deliver a world class NHS service (DH 2000, 2002, 2008, 2010; NHS Alliance 2007).
- Regulatory and professional body frameworks include leadership competencies as an integral part of health professionals' initial and continuing professional development and education across a career trajectory (Skills for Health 2007; HCPC 2007; CSP 2010).
- Commissioning high quality healthcare requires frontline clinician engagement (Corrigan 2000; King 2010).
- The NHS requires clinical leaders who infiltrate all areas of NHS organisations (McDonald et al. 2009; Towill 2009; Edmonstone 2011).
- Leadership is every clinician's business (Janes and Mullan 2007; NHSi 2004).
- There are well recognised links between leadership capability and sustained high performance (Fillingham 2007; Gobillot 2007; Hardacre et al. 2010).
- Management and leadership talent are the most important features of organisations (DH 2007; Fillingham 2008)
- The National Clinical Leadership Council supports clinical leadership development.

(N.B. the nomenclature 'clinician' is used throughout this chapter to denote the full range of all healthcare professionals engaged in direct clinical care, irrespective of their professional background. It is intended to emphasise the point that the whole healthcare team shares equal responsibility for improving the health outcomes of patients.)

Just as there is a lack of commonality in the use of the concept 'leadership', the term clinical leadership is still being defined and refined (Cook 2001; Stanley 2006).As this chapter is primarily about leadership in the NHS, it is useful to provide a definition of clinical leadership. The definition below is useful in that it emphasises the importance of leadership as action rather than as leadership relating to position:

> *Someone who whether in a formal or an informal position in an organisation who demonstrates particular skills and behaviours that accord with both professional and organisational values and that result in improvement in service delivery, safety and quality whilst at the same time engendering the active support of colleagues and peers…*
>
> (NHS North West Leadership Academy 2008)

NHS leadership qualities framework (LQF)

The LQF was created specifically to provide a common language and approach for leadership in the NHS (NHSi 2009). It was designed over a two-year period by the Hay Group and launched in 2002 by the then NHS Chief Executive and Permanent Secretary to the Department of Health Sir Nigel Crisp. It was reviewed most recently by the NHS Institute for Innovation and Improvement (NHSi) in 2006.

It is said to reflect the culture of the NHS and is designed to be flexible and intuitive, and allows for individual self-reflection (see Chapter 5 for further details). A widely-available framework, the LQF allows individual clinicians to analyse their own leadership roles and therefore can be a useful self-development tool. The LQF sets the standards for outstanding leadership and describes the qualities expected of existing and aspiring leaders. Within the LQF there are 15 leadership qualities clustered into three groups:

1. Personal Qualities
2. Setting Direction
3. Delivering Service.

Each cluster describes the key characteristics, attitudes and behaviours required for effective leaders at all levels (NHSi 2009). Full details of the LQF can be found at www.nhsi.nhs.uk.

This next section will describe each cluster of leadership qualities and relate them to how you may begin to reflect upon your own abilities and strengths, as well as begin to identify your development needs. The descriptions below are adapted from the LQF. A complete version of this can be found at www.nhsi.uk. Readers are also directed to Griffin (2009) for examples of use in practice.

Personal qualities

The personal qualities required of successful leaders are: self-belief, self-awareness, self-management, a genuine drive for improvement and a high level of personal integrity. Demonstrating these qualities through everyday practice is important. Self-belief means standing up for what you believe in and not being afraid of doing so, even when others seem to hold an alternative view. Being aware of how your behaviour impacts on others is key to effective leadership – your own actions and emotions will have an impact on those you work with; this impact can be positive or negative. Reflection can help you to explore these qualities in yourself by thinking about, and critically evaluating, your own experiences. Being able to manage yourself is vital if you are going to be able to work within, and cope with, increasingly complex environments. Healthcare was never straight forward and today's practice is certainly not. It is forever changing, diverse and increasingly complex.

Task

Think about some of the successful leaders you have come across either in work or in other aspects of your life, for example sport and school, and try to remember how they demonstrated the personal qualities listed above.

Think about how you demonstrate these personal qualities. Reflect upon your own personal qualities and begin to think about which aspects you may need to develop.

Setting direction

Setting direction means being able to articulate a vision and a direction of travel which others can understand, see and feel able to achieve. In order for leaders to be able to do this they need to be able to exploit all opportunities to bring about improvements by understanding and interpreting the drivers for change, such as those described in earlier sections of this chapter. Intellectual flexibility means being receptive to fresh insights – being willing to see things from a different perspective other than your own. It is about being open to professional conversations that challenge our way of seeing the world. This can help us to support and encourage our own creativity, as well as that of our colleagues. It can be extremely liberating to discuss and suggest new insights and solutions to old problems.

In order to set direction appropriately leaders need to keep themselves informed about key developments; they usually have a range of networks that enable them to benchmark themselves and their teams against peers in a systematic way. Being receptive to standing in patient and service user's shoes and seeing things from the point of view of a user of the service they are providing helps to provide a unique insight impossible to get in any other way.

Task

Think about successful teams you have worked with and reflect upon what made the teams successful.

Reflect upon a time when you have taken a lead role in a team. How did you set direction for yourself and the rest of the team?

Delivering service

In order to deliver world class services, leaders need to work through others. Individual's spheres of influence tend to be restricted to a small number of individuals. This means that leaders have to rely upon others to effect a change. Spheres of influence show us that leaders rely upon those they influence to then influence others, and so on – a little like the ripples in a pond when a stone is thrown (NHS Alliance 2007; Hartley and Bell 2009; Hardacre et al. 2010).

Delivery of services relies upon leaders being able to lead change through others, hold people to account for the work they do, empower others to take the lead, and influence through effective partnerships and collaborative working (see Chapter 2). In order to effectively lead, individuals need to be able to gain the support of others, be prepared to share leadership through empowering and enabling others, and be able to hold people to account.

Task

Reflect upon your own team-working successes. How did you go about building relationships and partnerships? What strategies did you use?

Think about a time when you have taken the lead. How did you empower others? How did you measure your success in a leadership role? What, if anything, did you learn from the experience and how do you intend to develop your abilities?

As other chapters in this textbook have highlighted, no one healthcare professional can be a universal panacea for the healthcare needs of patients. Collaborative working is essential if we are to deliver on the promise of world class healthcare. Leaders therefore need to be capable of effective and strategic influencing, able to work in partnership, cope with ambiguity, understand diverse view points and strive to create an environment where successful partnership working can thrive.

The impetus for collaborative partnership working is reinforced by the National Clinical Leaders Network (2009) whose principles reinforce the need for collaboration and describe them as improving clinical leadership and engagement, improving quality, and influencing policy and service reform across professions, specialty, service and organisations (Farrar 2009).

COMMISSIONING WORLD CLASS HEALTHCARE SERVICES: THE ROLE OF CLINICAL LEADERSHIP AND SERVICE IMPROVEMENT

In 2007 the NHS Alliance (see www.nhsalliance.org) published a document that describes and showcases examples of Allied Health Professions (AHPs) leading

change in the NHS. This document describes the relationship between clinical leadership and world class commissioning. Clinical leadership at all levels is an essential component for change leading to improved services and health outcomes. Clinical engagement is required for commissioning and it can only happen if clinical leadership infiltrates the entire workforce (NHS Alliance 2007). Commissioning the right health services for the provision of high quality health services reduces health inequalities and improves the health of local populations. In simple terms commissioning involves:

- identification of need;
- securing services to meet the need;
- performance-management of the service;
- evaluation of the delivery of services.

It requires:

- ***knowledge;***
- ***resources;***
- ***action;***
- ***time-management of change.***

(NHS Alliance 2007)

The proposed health reforms put GPs in the driving seat of commissioning healthcare across the English health system (readers are directed to the Scottish, Welsh and Northern Irish governments for details of these country's health service systems); in other words, putting clinicians in the frontline. This requires clinicians to be engaged in the commissioning agenda. What commissioning involves and requires is what frontline clinicians can provide and every clinician has a responsibility to engage with it. Frontline clinicians have knowledge of patient needs and the services required to meet those needs. Clinicians deliver the service and therefore are well placed to lead service and workforce redesign initiatives. Through research, clinical audit and other outcome-measurement metrics clinicians are also capable of evaluating the service they provide and measuring patient outcomes qualitatively and quantitatively. Thus, clinical engagement is of central importance and can only be achieved if clinical leadership is distributed throughout the workforce.

Clinicians at all levels require enabling and empowering to develop their own leadership competence to be effective.

Leadership is action not position…

Donald H. McGannon

So, as commissioning is the driving force behind the provision of high quality health services there needs to be a high level of clinical engagement in commissioning in order to ensure that the best services are commissioned. Clinical engagement is essential for world class commissioning. Clinical engagement is only possible where clinical leadership is distributed throughout organisations and professional groups.

Service improvement

Clinical leadership is an important requirement to achieving the ambitious healthcare reforms described in the white paper. Another essential aspect of health reform is the modernisation of services through service redesign/reconfiguration, and service improvement and workforce redesign (Batalden and Davidoff 2007; NHSi 2009; Wilcock et al. 2009; DH 2010). The Institute of Healthcare Improvement in Boston pioneered the application of quality improvement in healthcare. From 2001 to 2004 the Modernisation Agency (MA) in the UK promoted the same (NHS MA 2004). Subsequently, the MA's successor, the NHS Institute for Innovation and Improvement, continues to promote quality improvement in healthcare (Batalden et al. 2002; Fillingham 2007). Similar approaches are taken in a number of other regions, including Scandanavia, Australia and the Netherlands.

It is well recognised that service improvement is the responsibility of all healthcare professionals. It transcends professional boundaries, puts patients at the centre of healthcare decision-making, and requires co-operation and collaboration across agencies, organisations, services and professional groups. As Batalden and Davidoff (2007: 80) assert, achieving service improvement requires the commitment of all professionals on a daily basis as part of everyday professional practice: 'the ideal conditions for this are when everyone working in health care recognises that they have two jobs when they come to work every day to do their job and improve it'. Leadership underpins collective action, providing truly patient-centred healthcare requires individuals and groups of professionals to take collective action.

In order to make service improvement an integral part of everyday life of clinicians, the NHS will need strong and effective leadership that transcends professional and organisational boundaries and which pervades every corner of organisations (Batalden et al. 2002; Wilcock et al. 2009). In order to meet the demand for healthcare now and in the future the NHS will need to implement the health reform agenda through staff who are empowered to lead service improvements designed to improve health outcomes in a patient-led NHS (DH 2010). This will require clinical leaders who are able to work collaboratively, across traditional boundaries and, importantly, learn to see the service they provide through the eyes of their patients.

Clinicians at all levels need to learn to see beyond what they normally see. As David Fillingham says when introducing lean healthcare, in his book about his personal leadership journey as the Chief Executive of an NHS trust:

… it is amazing how blind we can become in our day to day work to the errors and problems that exist all around us … we are often so preoccupied with the demands of our job that

> *we stop seeing the service through the eyes of the customer … learning to see the service as they see it is critical to identifying the opportunities for improvement…*
>
> David Fillingham (2008: 55)

Thus, clinical leadership and service improvement go together. As earlier parts of this chapter have outlined, service improvement will only be successfully sustained if clinical engagement is achieved. Clinical engagement is promoted through clinical leadership. Therefore, the development of leadership qualities is fundamental in providing world class healthcare.

High quality healthcare has been described as clinically effective, personal and safe (DH 2008). Despite best efforts, there are still some areas of the NHS that do not achieve this (DH 2010). At its best, the NHS provides a world class service. Ongoing and newly proposed health reforms are the way in which a world class NHS for all is thought to be achievable. Improving care at the frontline requires continuous quality improvement (DH 2000; Janes and Mullan 2007). Continuing quality improvement requires professionals to work together. No one profession has all the answers; professionals have to share knowledge and skills with each other to achieve the best health outcomes for patients.

Since the inception of the MA over ten years ago, there has been an extensive NHS-wide change programme. This modernisation programme has led to a radical overhaul of NHS services and the NHS workforce. NHS organisations have undertaken a radical service improvement programme which has led to redesigned services, the development of clinical pathways of care, and a redesigned NHS workforce to meet the changing and increasing health demands of the public. Professional development of existing NHS staff has included service improvement methodology and, increasingly, service improvement learning is included in the undergraduate programmes which prepare a wide range of professionals for practice (Janes and Wilford 2007; NHSi 2008). As already described, service improvement involves clinical engagement and clinical engagement requires clinical leadership, hence its importance and inclusion in this textbook. Fusing learning about improvement with doing real improvement work has become a key driver (Aron and Headrick 2002).

The case for clinical leadership

The DH Operating Framework 2007 confirmed the importance of clinical leadership in delivering improved services for patients.

> *… it is the very nature of the reforms that improvements must be owned by clinicians, managers and other frontline staff on the ground … it is the responsibility of the NHS community leadership … to engage fully with clinicians. Staff, patients and the wider public to communicate and explain the need for change and the potential of reforms locally to improve services and people's lives…*
>
> (DH Operating Framework 2007/8: 36)

The NHS Next Stage Review Interim Report 2008, led by Lord Darzi, further emphasised the responsibility of clinicians to lead change in the NHS in response to the public's need: 'the essence of clinical leadership is to motivate, to inspire, to promote the values of the NHS and create a consistent focus on the needs of patients' (DH 2007: 37).

SOCIAL INTERACTION, SPHERES OF INFLUENCE AND PROFESSIONAL PRACTICE EXAMPLES

Within everyday life we are continually interacting with people around us. This interaction creates an environment where we both influence and, in turn, are influenced by others (Goffman 1959; Jenkins 2004). This interaction impacts how we behave and what we do. The same can be said of everyday professional practice. Everyday professional practice involves us in interaction. In other words, what we do and how we do it is affected by those around us. This interaction creates what is called a *sphere of influence* for each and every one of us. This sphere of influence is associated with our position in organisations and we each have a limited sphere of influence dictated by where we sit in organisations and with whom we interact. These spheres of influence occur through social interaction.

We all have spheres of influence and it is through these spheres of influence that we can affect those around us. Exercising clinical leadership can help us to use our spheres of influence to effect positive change wherever we find ourselves and whatever our position in organisations. Effective influencing is key to effective leadership (see NHSi 2009).

The new CSP physiotherapy framework (2010) describes clinical leadership as an integral aspect of a definition of physiotherapy. Physiotherapy is defined as 'a health profession that works with people to identify and maximise their ability to move and function' (CSP 2010). It states that physiotherapy has strong clinical leadership and an adaptable workforce able to deliver high quality links between education and research. It describes the four elements of physiotherapy practice, physiotherapy knowledge, skills, values and generic behaviours – knowledge and skills shared by all clinicians working in healthcare. The CSP framework divides these generic elements into two sections relating to interaction, and problem-solving

and decision-making. For more details, readers are directed to Chapter 1.

All the elements described in the physiotherapy framework share aspects that are also integral to clinical engagement and leadership, collaborative working and accountability. The framework reflects the context of contemporary healthcare, including the reform agenda associated with continual service improvement.

The diagrams below provide examples of spheres of influence at different career levels, advanced support worker, advanced practitioner and expert practitioner, thus demonstrating that we can both influence and be influenced in order to effect service improvement (Figure 3.2). Note that these are suggestions of what spheres of influence may look like and are not intended to be exclusive.

Task

Draw on your own sphere of influence and write down ways in which you exercise your influence within the sphere. Reflect upon how you think others influence you in return. Describe the strategies you use to influence effectively. Think of ways of expanding/changing your sphere of influence. What strategies can you develop to improve your influencing?

DEVELOPING YOUR LEADERSHIP COMPETENCIES

The previous sections have outlined some of the reasons and the professional drivers of why we should all be concerned with developing ourselves as leaders. Within our own spheres of influence we can begin to influence those around us by having the courage to share our knowledge and skills and work in a collaborative way across professional boundaries. We can begin to contribute to improving the quality of healthcare and contribute positively to health reforms which are focussed on improving health outcomes through our clinical leadership. This next section will provide you with some practical ideas for you to consider as a way of developing your own leadership competencies.

Organisational requirements

In order for leaders to develop in their organisations a number of factors are required. Not least there needs to be a shared understanding of what leadership looks like. Remember, the NHS is made up of a number of distinct

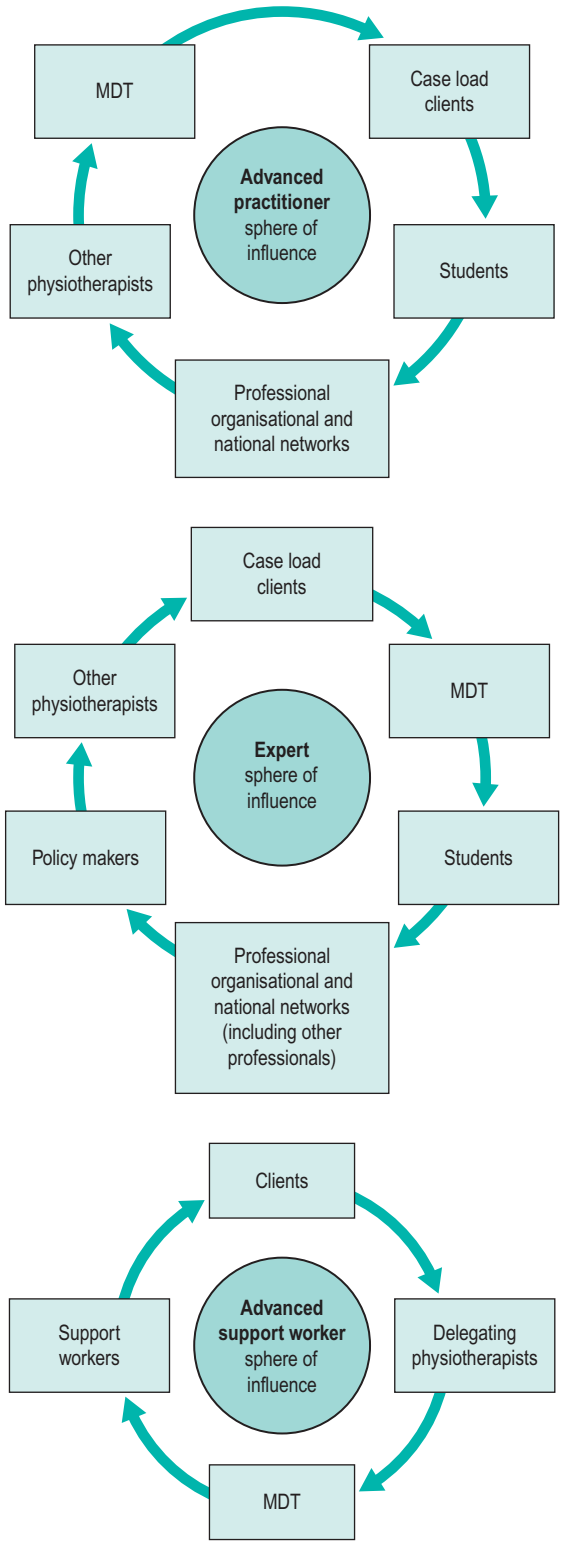


Figure 3.2 Spheres of influence. *Adapted from CSP Framework 2010 (CSP 2010).*

organisations, each with their own culture and ways of doing things and so it cannot be guaranteed that each NHS organisation behaves in the same way as the next. The LQF provides a framework for leadership; it cannot, however, control how it will be interpreted and operationalised in each and every organisation.

Organisations need to value the role of leadership, reward it and be open and willing to share leadership experiences. For example, chief executives of NHS organisations in the north-west of England have all contributed to a book called *Placing Ladders; Harnessing our Leadership Potential* (Hartley and Bell 2009), which shares their leadership stories. This is a very powerful book in that it describes the first-hand experience of very senior leaders, many of whom are humbled by their experiences of listening to and learning from patients about the care they received in their hospitals. These very senior leaders recognise and value the contribution of all their staff and celebrate success collectively. Some share some painful stories as a way of writing about their leadership approach (see Chapter 3 'Making Spaces' in Morley 2009). The power of stories as a legitimate form of understanding and making sense of lived experiences cannot be underestimated (Reissman 2008). These stories provide a very real insight into what some of today's NHS leaders do in the process of leading their organisations. Hardacre et al. (2010) propose ways in which leadership can influence improvements in healthcare outcomes as a result of their research into the links between NHS leadership and improvement.

Figure 3.3 describes the components required for leadership development within organisations.

Individual requirements

There are a number of ways in which we can begin to systematically think about ourselves in a leadership role. Many of the skills we use to support the development of our professional knowledge and skills can also be used to support our leadership competencies and are described in more detail in other chapters of this textbook. This final section suggests ways of supporting your personal learning. This list is not exhaustive but does include some of the more commonly used approaches that professionals use to support their professional development. Some of those listed involve social learning approaches. Social learning theory suggests that learning is a collective endeavour, achieved through, and by, interaction with others via communities of learning. Readers are directed to Wenger (1998) for more details. Other approaches described involve a more individualised approach to learning. A common theme throughout is experiential learning (Boud et al. 1985; Jarvis et al. 2003). Experiential learning is most often associated with adult learning and professional learning in practice (Eraut and Hirsh 2007). The author does not propose that any particular approach is better than another. You will find some of the approaches listed below more helpful than others; this will reflect your own particular learning style and particular organisational context. The important thing to remember is to explore a number of learning approaches to find the one that suits your way of learning and provides you with the support you find most helpful.

- **Action learning sets.** Small groups of clinicians (often from different professions) coming together

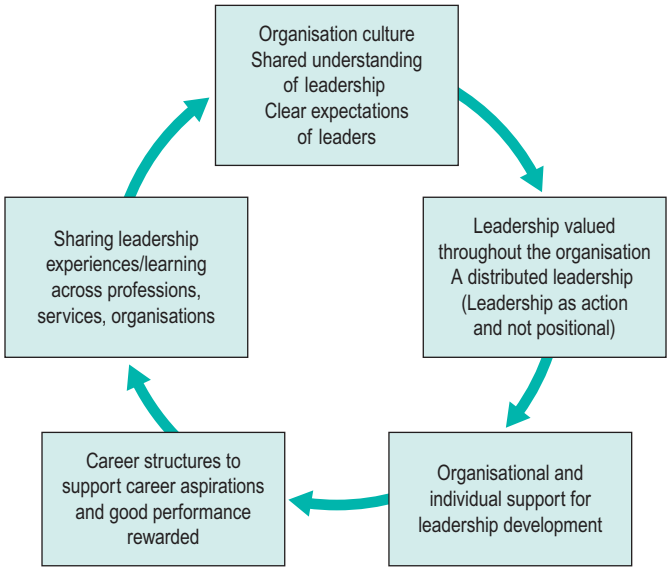


Figure 3.3 The components required for leadership development within organisations. Individual requirements.

on a regular basis to share and learn from the experiences of group members. Often involves a case study type of approach where one member will describe an experience in order to stimulate discussion in the group around possible solutions/future actions.

- **Mentoring.** A supportive one-to-one relationship where either a more senior professional mentors a more junior colleague or peers mentor each other. Can be uni- or interprofessional. Mentor and mentee meet on a formal/informal basis. Both mentor and mentee agree the parameters of the one-to-one; focus is on the needs of the mentee.
- **Coaching.** Similar to mentoring but usually much more structured to provide stretch and challenge to support career development around competency development at a particular stage in a career, for example when seeking promotion or when newly promoted and development needs have been agreed. Often used to support executives and senior staff in organisations.
- **Reflection.** Usually involves individuals describing day-to-day experiences in order to learn from successful and not-so-successful experiences in order to improve their own professional practice. These reflections can often provide the experience that individuals bring to a collective/social learning event (see Chapter 5 for more details).
- **360 degree feedback.** Usually undertaken as part of leadership development programmes and involves self-assessment and colleagues' assessment of skills and abilities to provide a comprehensive assessment of ability that can be used to identify strengths and learning needs. Individuals ask a number of colleagues to assess them, including the line manager, peers and subordinates in order to provide a full, 360 degree perspective. Can be a very powerful tool for individuals to assess self-perception against how others perceive them.
- **Professional networks.** Both uni- and interprofessional in nature. Virtual and physical bringing together of clinicians to share knowledge. Often used as a forum to discuss topical issues, for example health reforms and policy issues. Can be used to provide a collective response to consultations. Examples include regional physiotherapy boards, AHP networks, clinical manager groups, National Clinical Leaders Network and the AHP Confederation. Building and maintaining professional networks are key to benchmarking yourself against peers and can be a very powerful way of triggering reflection.

In practical terms, none of the above are mutually exclusive. For example, while coaching is quite often undertaken on a one-to-one basis, often action learning sets involve individual members coaching each other within the action learning episode. Professional networks usually support a large number of professionals in broadly generic ways through virtual and physical network communities; they also incorporate specific programmes to support members. For example, the NHS North West AHP network has developed a comprehensive coaching network for members to access as part of their specific developmental needs (www.nhsnw/ahpnetwork.uk). Leadership development is important to enhance the quality of leadership in organisations and there are many formal learning opportunities available for clinicians to access. And, of course, there are many formal award-bearing learning programmes available, such as Masters level programmes specifically designed to meet the needs of health service managers.

It is assumed that readers of this text are familiar with the process and practice of reflection as an integral aspect of being a physiotherapist and are directed specifically to Chapter 5 for revision. Clinical leadership is about taking individual and collective responsibility for your day-to-day practice and striving for continual service improvement and improved patient experience.

The development of clinical leadership should begin as an undergraduate and continue throughout your career. Remember, clinical leadership is not about the position you hold in an organisation but rather about a way of behaving and acting. It is acknowledged that often the position a person holds in an organisation does put them in a powerful position from where they can assert their leadership. However, if graduates are to be ready to take on the challenge associated with clinical leadership then it follows that their leadership development must begin as an undergraduate. This can be daunting for undergraduates; however, there are a number of ways in which undergraduates can begin to test out their leadership skills, such as putting yourself forward to be a student representative or taking part in service improvement initiatives as part of your undergraduate programme where opportunities exist (NHSi 2008; Janes and Wilford 2007), seeking opportunities during your clinical placements (on top of, and in addition to, developing your profession-specific knowledge and skills) to observe or get involved in service redesign programmes, actively participate in multidisciplinary team meetings and take the initiative to discuss service improvements with your clinical educator and other clinicians through professional conversations.

Developing leadership qualities in clinicians helps to:

- *engage them in service reform;*
- *address service delivery frustrations;*
- *develop evidence-based effective practice;*
- *reduce service and clinical variations;*
- *increase efficiencies;*
- *promote reflective practice;*
- *increase capacity and capability.*

(NHS Alliance 2007)

CONCLUSION

The focus on developing clinical leaders at all levels of NHS organisations remains a key priority (DH 2010). This chapter has provided the reader with an overview of clinical leadership and the context in which it is developing and evolving. Political, professional and organisational imperatives demand that every clinician, irrespective of professional allegiances, rises to the leadership challenges ahead to improve the quality of healthcare to improve health outcomes. While it is acknowledged that the evidence of a causal link between NHS leadership and improved services remains scant, there is a wealth of evidence through personal leadership stories of NHS leaders to support the ongoing investment in leadership development. Challenges for clinical leaders are vast. The NHS is a complex organisation made up of many distinct, yet connected, organisations charged with unprecedented integration and reform. The shift towards healthcare commissioning through general practitioner consortia puts clinicians in the driving seat. The complexity of today's healthcare requires an interprofessional approach to service development, delivery and improvement. The interdisciplinary nature of healthcare demands wide spread clinical engagement in commissioning; this can be, and is being, achieved through clinical leadership. The clinical leaders of tomorrow need to recognise the importance of leadership as an integral aspect of their professional life and practice.

> *Leadership is about individuals and multilevel leadership … about translating concepts and ideas so that they have meaning for colleagues working with patients, designing services or considering patient needs. It is about building commitments and agreement to go forward addressing population and service needs, changing services, delivering services…*
>
> (NHS Alliance 2007: 13)

REFERENCES

Allan, H.T., Smith, P.A., Lorentzon, M., 2008. Leadership for learning: a literature study of leadership for learning in clinical practice. J Nurs Manage 16, 545–555.

Aron, D.C., Headrick, L.A., 2002. Educating physicians prepared to improve care and safety is no accident: it requires a systematic approach. Qual Saf Healthcare 11, 168–173.

Batalden, P.B., Stevens, D.P., Kizer, K.W., 2002. Knowledge of improvement: who will lead the learning. Qual Manage Healthcare 10 (3), 3–9.

Batalden, P.B., Davidoff, F., 2007. What is quality improvement and how can it transform health care? Quality Saf HealthCare 16, 2–3.

Boud, D., Keogh, R., Walker, D., 1985. Reflection: Turning Experience into Learning. Kogan Page, London.

Cook, M., 2001. The renaissance of clinical leadership. Int Nurs Rev 48, 38–46.

Corrigan, P.W., 2000. Mental Health team leadership and consumer satisfaction and quality of life. Psychiatr Serv 51 (6), 781–785.

CSP (Chartered Society of Physiotherapy), 2010. Physiotherapy Framework; http://csp.org.uk, accessed November 2010.

DH (Department of Health), 1994. The Report of Enquiry into the Care and Treatment of Christopher Clunis. Her Majesty's Stationery Office, London.

DH (Department of Health), 2000. The NHS Plan: A Plan for Investment, A Plan for Reform. Her Majesty's Stationery Office, London.

DH (Department of Health), 2001. Working Together, Learning Together: A Framework for Lifelong Learning for the NHS. Her Majesty's Stationery Office, London.

DH (Department of Health), 2002. Making a Difference. Her Majesty's Stationery Office, London.

DH (Department of Health), 2007. NHS Chief Executive's Annual Report. Her Majesty's Stationery Office, London.

DH (Department of Health) Operating Framework, 2007. Operating Statement 2007/8, Department of Health. Her Majesty's Stationery Office, London.

DH (Department of Health), 2008. Framing the Contribution of Allied Health Professions; Delivering High Quality Health Care Report. Her Majesty's Stationery Office, London.

DH (Department of Health), 2009. High Quality Care for All; Our Journey So Far. Her Majesty's Stationery Office, London.

DH (Department of Health), 2010. Equity and Excellence: Liberating the NHS. Her Majesty's Stationery Office, London.

Edmonstone, J., 2011. Developing leaders and leadership in health care: A case for rebalancing? J Leadership Health Services 24 (1), 8–18.

Eraut, M., Hirsch, W., 2007. The Significance of Workplace Learning for Individuals, Groups and Organisations. ESRC, Oxford.

Farrar, M., 2009. In: Hartley, J., Bell, A. (Eds.), Placing Ladders: Harnessing our Leadership Potential, pp. xi–xii. Kingsham Press, Chichester.

Fillingham, D., 2007. Can lean save lives? Leadership Health Serv 20 (4), 231–241.

Fillingham, D., 2008. Lean Health Care: Improving the Patient's Experience. Kingsham Press, Chichester.

Firth-Cozens, J., Mowbray, D., 2001. Leadership and quality of care. Qual Health Care 10, ii3–ii7.

Griffin, J., 2009. Journeying through Leadership. In: Hartley, J., Bell, A. (Eds.), Placing Ladders: Harnessing our Leadership Potential. Kingsham Press, Chichester.

Gobillot, E., 2007. The Connected Leader: Creating Agile Organisations for People Performance and Profit. Kogan Page, London.

Goffman, E., 1959. Presentation of Self in Everyday Life. Penguin Books, Harmondsworth.

Hardacre, J., Cragg, R., Flannagan, H., et al., 2010. Exploring links between NHS leadership and improvement. Int J Leadership Public Serv 6 (3), 26–38.

Hartley, J., Bell, A. (Eds.), 2009, Placing Ladders: Harnessing our Leadership Potential. Kingsham Press, Chichester.

HCPC (Health and Care Professions Council), 2007. Standards of Proficiency Physiotherapists. HCPC, London.

Janes, G., Mullan, A., 2007. Service improvement is everybody's business. Nurs Manag 14 (6), 22–25.

Janes, G., Wilford, B., 2007. Service improvement education – when should it begin? Synergy March, 28–29.

Jarvis, P., Holford, J., Griffin, G., 2003. The Theory and Practice of Learning, second ed. Kogan Page, London.

Jenkins, R., 2004. Social Identity, second ed. Routledge, London.

Jumaa, M., Jasper, M., 2005. Effective Healthcare Leadership. Blackwell Scientific, London.

King, V.G., 2010. Clinical leadership project. J Nurs Educ 49 (11), 640–643.

McDonald, R., Price, I., Askham, P., 2009. Leadership conversations: the impact on patient environments. Leadership Health Serv 22 (2), 140–160.

Mishler, E.G., 1990. Validation in inquiry-guided research: the role of exemplars in narrative studies. Harvard Educ Rev 60 (4), 415–442.

Mishler, E.G., 1999. Storylines Craft Artists' Narratives of Identity. Harvard University Press, USA.

Morley, P., 2009. Making spaces. In: Hartley, J., Bell, A. (Eds.), Placing Ladders: Harnessing our Leadership Potential. Kingsham Press, Chichester, pp. 21–34.

National Audit Office, 2005. A Safer Place for Patients: Learning to Improve Patient Safety. Her Majesty's Stationery Office, London.

NHS Alliance, 2007. Clinical Leadership for NHS Commissioning; Exploring how Allied Health and other Health Professionals Lead Change Through and Beyond Commissioning for a Patient Led NHS. Her Majesty's Stationery Office, London.

NHS, MA. (Modernisation Agency), 2004. Ten High Impact Changes for Service Improvement and Delivery: A guide for NHS leaders. Her Majesty's Stationery Office, London.

NHS, NW (North West Leadership Academy), 2008. Towards Developing a Senior Clinical Leadership Development Strategy: A Review; http://www.northwest.nhs.uk, accessed December 2010.

NHSi (NHS Institute for Innovation and Improvement), 2008. Evaluation of the Improvement in Pre-registration Education Programme: Final Report. NHSi, Coventry.

NHSi (NHS Institute for Innovation and Improvement), 2009. The NHS Leadership Qualities Framework; http://www.nhsii.nhs.uk, accessed November 2010.

NHSi (NHS Institute for Innovation and Improvement), 2010. The Improvement Leaders Guides; http://www.institute.nhs.uk, accessed December 2010.

Reissman, C.K., 1997. A short story about long stories. J Narrative Life History 71 (1-4), 155–158.

Reissman, C.K., 2008. Narrative Methods for the Human Sciences. Sage, California USA.

Roger, G., 2006. Theory and Practice of Leadership. Sage Publications, London.

Skills for Health, 2007. A Competence Based Career Framework for Allied Health Professions (AHP). Project Information Bulletin 3, London.

Stanley, D., 2006. Recognising and defining clinical nurse leaders. Br J Nurs 15, 108–111.

Towill, D.R., 2009. Enabling effective change in health care delivery systems: Did Gerry Robinson teach us anything new? Leadership Health Serv 22 (2), 176–188.

Wenger, E., 1998. Communities of Practice: Learning, Meaning and Identity. Cambridge University Press, Cambridge.

Wilcock, P.M., Janes, G., Chambers, A., 2009. Health care improvement and continuing interprofessional education: Continuing interprofessional development to improve patient outcomes. J Cont Educ Health Prof 29 (2), 84–90.

Chapter 4

Pharmacology

Nicholas T.L. Southorn

INTRODUCTION

Pharmacology?

This chapter is a new addition to Tidy's which represents the innovative and forward motion of the profession of physiotherapy. Greater independence and autonomy as primary care practitioners with direct access means that physiotherapists have to demonstrate an understanding of medical conditions and treatments, including medicines. The clinical picture painted from the subjective assessment involves drug history; therefore, the need to understand how to interpret this information is essential lest the picture be smudged and the patient's care compromised.

Clinical pharmacology is an entire science; this is a chapter within a book. In no way will it cover the entire breadth and scope of the topic, but rather provide a brief overview of the application of basic scientific knowledge regarding common medicines encountered by the physiotherapist. This information should be adequate for your studies, but I hope to light the touch paper of an explosive interest in pharmacology which you may fuel further by reading the recommended texts at the end of this chapter. A basic physiological knowledge is expected to be enough to understand pharmacology; however, where possible, the relative physiology will be explained. Please note that Chapters 12 and 17 may be a useful starting point.

Please see the end of this chapter for a reference glossary of pharmacology terms and some common drugs.

What is pharmacology?

Simply, it is the study of medicines and their interaction with the body. Information is needed from the moment the drug enters the body and, indeed, how it got there, how it travels to the site of action, the physiological actions and their consequences, and how it is metabolised and excreted.

When talking about a drug, the typically accepted method is as outlined in Table 4.1.

EG Example

Ibuprofen may be taken by adults orally in tablet form at 400 mg three times per day off prescription (four times per day on prescription) or applied to the skin as a cream. Its anti-inflammatory action is that of non-specific inhibition of cyclooxygenase (COX)-1 and COX-2, thus inhibiting prostaglandin production. It is a weak acid and is absorbed largely in the stomach with a peak plasma concentration of one hour. It is metabolised by the liver and excreted in the urine. Owing to the non-specific nature of the COX inhibition, its side effects include alteration of the mucosal lining of the stomach (reduced bicarbonate excretion = reduced lining defence against hostile acidic environment so increased H^+ secretion exacerbates the acidity of the stomach) and renal vasoconstriction. Therefore, it is contraindicated in those with compromised kidney function and prone to gastric ulcers.

This is all you need to know about ibuprofen for the adult in one paragraph, simple!

However, this chapter herein avoids using this method for two reasons:

- you should be making notes that require you to gather information from different sources, which will help you learn in more depth;
- it is rather repetitive for me to write and you to read!

Why would I, a physiotherapist or a student, want to know about this?

If you have a detailed understanding of the action of the drugs in question you may understand how long it takes to act, how some drugs may have multiple uses, why doses of drugs may differ significantly when taken via different routes and how the drugs may be altering the patient's perception of pain. You will also appreciate how your practice impacts upon the uptake of drugs, for example the application of heat near a transdermal patch or the use of relaxation.

The clinical picture, as previously mentioned, needs to be complete; poor medical knowledge may lead the clinician to jump to erroneous conclusions.

EG Examples

DRUG	= THE PATIENT MUST HAVE…
Metformin	= diabetes
Amitriptyline	= depression
Gabapentin	= epilepsy / seizures

These are all quite natural assumptions – ones that would be enforced by looking at the *British National Formulary* (BNF); however, metformin may be used for polycystic ovary syndrome, amitriptyline for sleeping problems and neuropathic pain, and gabapentin for neuropathic pain. So, when you read the past medical history: don't be sold a kipper – understand the possibilities.

On the ward, you must know your patient's past medical history; for example morphine may influence certain respiratory techniques; spinal anaesthetics may make one think twice about mobility assessments; a patient's dizziness may be caused by certain antibiotics, for example streptomycin, etc.

It is common for people to go to their local shop and buy some over the counter (OTC) medication. However, they don't always take these appropriately – you need to know if they are over- or under-dosing and advise appropriately. Also, are they contraindicated by any of the prescribed medication? Does the medical prescriber know what the patient is taking? This point is particularly pertinent when the patient is taking herbal medications – asking them to chat to their family doctor regarding this is *always* recommended.

Picking up on these pharmacological red flags is as important as picking up any other type of red flag. So, the next time a patient tells you that they are taking three 500 mg of paracetamol six times per day or St John's wort to help with their depression, you might decide to advise the patient to discuss these issues with their general practitioner.

So, as a physiotherapist, you should care and the profession as a whole should embrace pharmacology for the sake of patient safety.

Table 4.1 What you should know

Drug	The name of the drug
Route and dosage	Orally, injected, rectally, transdermal, etc.
Pharmacodynamics	What the drug does to the body
Pharmacokinetics	What the body does with the drug
Metabolism/excretion	How is the drug eliminated
Side effects	Including contraindications/ precautions, etc.

BASIC SCIENCE

Where do drugs act (Table 4.2)?

Their action will ultimately have an impact on the cell to which the surface protein is attached, and therefore the tissue, organ and system in which the cell resides. A cell has three main options during normal function: excitation – such as that of a depolarised neurone – contraction or secretion. The drugs will affect the cells' ability to express themselves either making them more active, less active or maintaining normal function by blocking other substances from affecting them (Figure 4.1).

How may drugs get to their destination?

Drugs must be absorbed, i.e. they have to get from the outside in. The main routes of administration are:

- oral;
- sublingual;

Table 4.2 Protein/chemical interactions

Drugs basically target specific proteins, thus:	
Receptors	e.g. morphine
Enzymes	e.g. angiotensin-converting enzyme inhibitors
Ion channels	e.g. local anaesthetics
Transporters (carrier proteins)	e.g. proton pump inhibitors

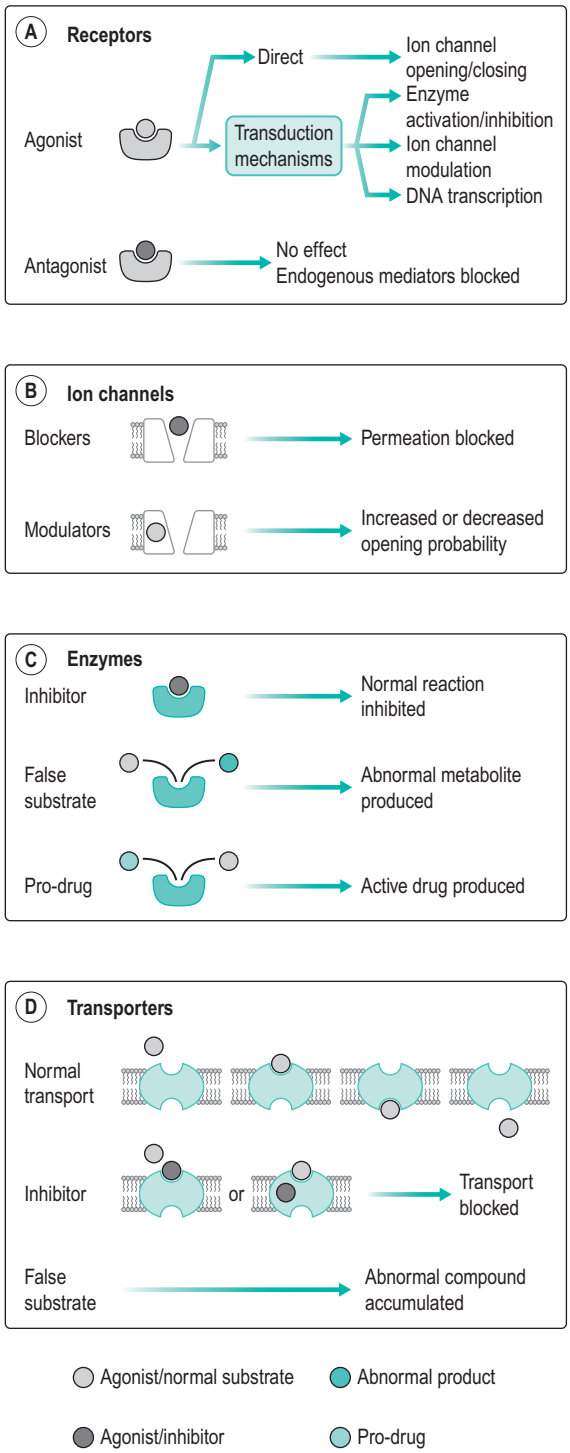


Figure 4.1 Drugs act on many parts of the body including (A) receptors (B) ion channels (C) enzyme and (D) transporters. *Reproduced with kind permission from Rang and Dale's Pharmacology (Figure 3.1: p.25). Copyright Elsevier 2008.*

- rectal;
- epithelial surface application (skin, nasally, corneal, etc.);
- inhalation;
- injection (subcutaneous, intramuscular (i.m.), intravenous (i.v.), etc.).

These routes each have additional factors which will contribute to how well the drug is absorbed, for example:

- blood flow;
- gastrointestinal motility;
- size of drug particle;
- dissociation constant (kP_a/physiochemical factors of the drug; see the Glossary term 'pH and ionisation' for details).

Wherever they are heading, they will need to cross a membrane along their way. They will do this in one of the following ways:

- diffusion through the lipid bilayer;
- transmembrane carrier protein;
- pinocytosis (invagination of the cell membrane creating an intracellular vesicle);
- diffusing through a special aqueous channel.

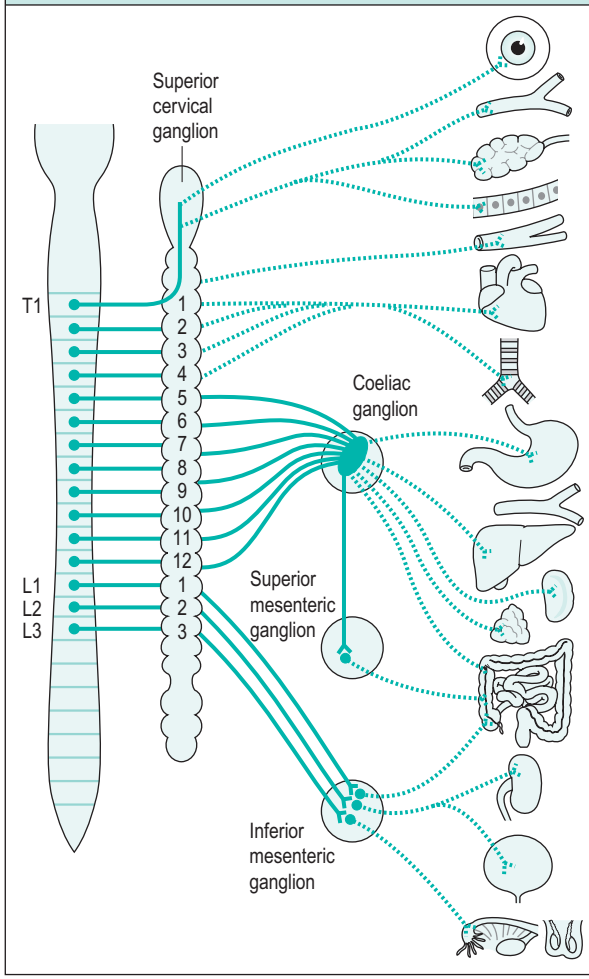
The **ionic status** of a drug molecule is significant when considering membrane transit. To traverse the lipid bilayer, a molecule should be non-polar and unionised (without charge). As many drugs are weak acids or bases, this presents a complication. The pH of the environment will alter the ratio of ionised to unionised molecules, thus influencing the diffusion of the drug across the membrane. Simply, an environment (pH = acidic or basic) that pushes up the relative number of unionised species will increase its ability to permeate the membrane, thus more drug will be absorbed. If the drug is an acid, A^-, (e.g. aspirin) in an environment with a low pH (e.g. the acidic gastric juices of the stomach, pH = 3) it will gain a hydrogen ion, H^+, and thus become unionised, AH, and cross the membrane. To clarify, an acidic drug is better absorbed in an acidic environment and a basic drug is better absorbed in an alkaline environment. It begins to stretch the scope of this chapter to delve any deeper, but for a better understanding see the Henderson-Hasselbalch equation and dissociation constant (kP_a) in any good pharmacology/clinical physiology book.

Blood flow may be influenced in your practice as a physiotherapist via heat, relaxation, massage, chest percussion, acupuncture, electrotherapy, etc. If the area to which the drug has been delivered has a good blood supply, for example gut, skin, mucosa, it will be absorbed more rapidly. Also, a drug that enters the systemic circulation but acts locally, for example ibuprofen, may have a higher concentration in the injured area if the blood flow is enhanced. It is always worth considering if you wish to influence this mechanism of drug absorption. An often-quoted scenario is the patient who has a

transdermal morphine patch and is advised to apply heat to their shoulder. In doing so, the patient's uptake of the drug was enhanced and experienced associated side effects.

The **sympathetic response** is part of the 'fight or flight' reaction. Part of the sympathetic effect is to decrease the gastric blood flow in favour of skeletal muscle and reduce peristalsis (this is the fight or flight response which is explained in exquisite clarity in *Payne's Handbook of Relaxation Techniques: A Practical Guide for the Health Care Professional*). By encouraging relaxation you will allow the parasympathetic nervous system to resume control and allow normal absorption of drugs and food from the gut (Figure 4.2).

Inhaled drugs (including oxygen), bronchodilators and cystic fibrosis transmembrane regulator therapy require the lungs to be operational enough to utilise the vast surface area and be clear enough so that excessive secretions are not blocking the epithelial transport of the drugs. The surface area and the capillary network allow rapid uptake of the drug, which makes inhalational therapy very useful indeed. Localised drug actions, such as dilating bronchioles, are useful as the lungs also expel the drug rather effectively, thus minimising the systemic effects. This is arguably the biggest influence that a physiotherapist can have on drug delivery, as not only can you mobilise secretions, you can facilitate the musculature to enable deeper breaths and expand more lung.



Structures	Effects of stimulation
Iris muscle	Pupil dilated Slightly relaxed
Blood vessels in head	Vasoconstriction
Salivary glands	Secretion inhibited
Oral and nasal mucosa	Mucus secretion inhibited
Skeletal blood vessels	Vasodilatation
Heart	Rate and force of contraction increased
Coronary arteries	Vasodilatation
Trachea and bronchi	Bronchodilation
Stomach	Peristalsis reduced Sphincters closed
Intestines	Peristalsis and tone decreased Vasoconstriction
Liver	Glycogen – glucose conversion increased
Spleen	Contracted
Adrenal medulla	Adrenaline and noradrenaline secreted into blood
Large and small intestine	Peristalsis reduced Sphincters closed
Kidney	Urine secretion decreased
Bladder	Smooth muscle wall relaxed Sphincter closed
Sex organs and genitalia	Generally vasoconstriction

Figure 4.2 Sympathetic outflow; the observable signs of sympathetic stimulation may be useful in assessing the non-communicating patient. *Reproduced with kind permission from Payne's Handbook of Relaxation Techniques (Figure 1.4: p.7). Copyright Elsevier 2010.*

Metabolism and excretion – the body gets hostile

Drug elimination is achieved by either metabolism (change of the molecular shape and function) or excretion (through urine, exhalation, faeces, etc.).

Once a drug has made its perilous journey from outside of the body to within, the body sets about changing it: the liver is the main site of metabolism. The main process of metabolism is mediated by cytochrome P450 – a large group of enzymes that alter the molecule of the drug and the product (metabolite) may be totally inactive, active in a completely different way or even more potent. Drugs that are given for the purpose of being metabolised into an active drug are called 'prodrugs'.

A drug taken orally must pass into the portal circulatory system from the intestines. This is a direct line to the liver and therefore it undergoes pre-systemic metabolism (also known as first-pass metabolism). It is essential to understand that this happens as the amount of available drug in the plasma is a fraction of the amount given orally (the actual fraction is called 'bioavailability'). The result is that the drug which undergoes extensive first-pass metabolism must be given in a higher dose orally than another route.

Other ways that the drug is eliminated is through excretion via urine. The clearance of a drug depends on many factors, including plasma binding, glomerular filtration, lipid solubility and pH, etc.

A note on dosing

All of the above needs to be calculated for efficacious delivery of the drug. The dose needs to be enough to 'work' in the therapeutic window but not too much to create side effects.

Figure 4.3 is a basic representation of delivery of i.v. morphine. An initial bolus dose is given until analgesia is achieved and then a top-up is given, perhaps by a patient-controlled analgesia device. This ensures that the plasma concentration is always in the therapeutic window.

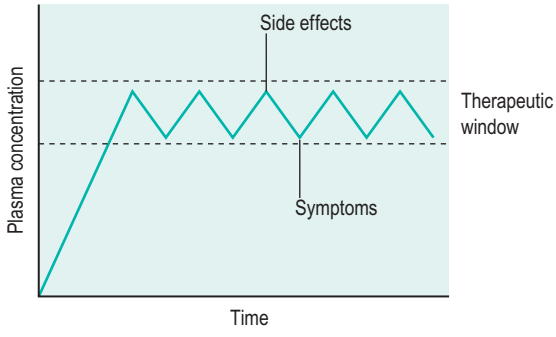


Figure 4.3 The therapeutic corridor must be aimed for and maintained, otherwise the side effects are too great or the symptoms are not controlled.

It is important to convey the information to the patient in an accessible way because they may not understand why they need to take some medications regularly when they only occasionally get symptoms, and some medications as and when required.

MEDICINES YOU WILL (PROBABLY) ENCOUNTER

All medications taken by the patient should be known to all involved in their care.

The heart and vascular systems

You should be aware of cardiac physiology as part of your course. Normal function of the heart depends on the rate, rhythm, contractility, blood supply and autonomic control. Drugs acting on the heart fall into three main groups:

- direct action on myocardial cells (anti-dysrhythmic drugs, inotropes, etc.);
- indirect cardiac function (diuretics, angiotensin converting enzyme (ACE) inhibitors, etc.);
- calcium antagonists.

Anti-dysrhythmic drugs are typically applied during emergency cardiac events in adjunct to, or in place of, physical treatments, such as electrical cardioversion (e.g. defibrillator). They include drugs that block voltage-sensitive sodium channels thus inhibiting the action potential, for example lidocaine (a local anaesthetic) will associate and dissociate within the time frame of a normal heartbeat, thus normalising the rate. It essentially pauses all electrical activity in order to allow normality to resume. It is delivered i.v. as it has a very low bioavailability (see above). It is metabolised rapidly, giving a short half-life, thus allowing rapid alteration of plasma concentration to avoid the central side effects of drowsiness and convulsions. Another action is that of β-adrenoceptor antagonism which effectively reduces sympathetic expression on the heart by increasing the refractory period of the atrioventricular (AV) node (i.e. slowing the heart's pacemaker). These drugs, such as propanolol and atenolol, have the unfortunate side effect of bronchospasm, fatigue and bradycardia, but in the absence of lung disease the risk is relatively low. Another class of anti-dysrhythmic drugs are calcium antagonists and are discussed below in more detail.

Increasing cardiac contraction may require poison. The extract of the foxglove (genus *Digitalis*) is used as a cardiac glycoside (e.g. digoxin). The effects are reduction of conduction, increased force of contraction and disturbance of rhythm. They enhance vagal activity and inhibit the

Na^+/K^+ pump, thus increasing the twitch tension in cardiac muscle. It is given orally (i.v. in emergencies) but unfortunately the clinical risk of such drugs is the fine line between effectiveness and toxicity. It is excreted via the kidneys which poses a problem in patients with less effective renal function in terms of maintaining plasma concentrations at a safe level.

Angina is an important symptom which indicates insufficient oxygen supply for cardiac activity. It is controlled by either increasing perfusion or reducing demand (or both). Two important drugs that you may encounter are glyceryl trinitrate (GTN) and isosorbide mononitrate. These organic nitrates are metabolised into nitric oxide which initiates a cascade of effects resulting in relaxation of vascular muscles and therefore reduced central venous pressure (and thus preload and afterload of the heart), reducing stroke volume and metabolic demand. The relaxation of coronary arteries will enhance the oxygen delivery to the myocardium. GTN is given orally by spray or sublingual tablet (owing to extensive first-pass metabolism it is ineffective if swallowed). It acts within minutes and is rapidly metabolised by the liver and effectively eliminated in half an hour, hence its usefulness in acute situations. Isosorbide is similar, but is taken as a tablet and lasts longer in the system which makes it a useful prophylaxis for angina.

Calcium antagonists block entry of Ca^{2+} to the cell by acting on voltage gated channels. Drugs such as verapamil and diltiazem are used occasionally for their antidysrhythmia action, but this group of drugs is more commonly used for hypertension (e.g. amlodipine and nifedipine). The action on the heart is generally a complicated balancing act but the effects of the arterial and arteriolar smooth muscle is relaxation, thus reducing peripheral resistance (and hence blood pressure). They are well-absorbed and thus taken orally, for example amlodipine is taken once daily because of its long half-life.

The renin-angiotensin system is of significant importance in terms of fluid volume and vascular tone. Renin is released by the kidneys and converts angiotensinogen into angiotensin I (AT_1) which is converted by ACE into the potent vasoconstrictor angiotensin II (AT_2). The ACE is a epithelial membrane-bound enzyme particularly abundant in the lungs and is the site of action for the antihypertensive ACE inhibitors (e.g. ramipril and captopril) whose action is simply to block the active site of the enzyme, thus preventing the formation of AT_2.

Statins help reduce the levels of low-density lipoprotein cholesterol (LDL-C) which adhere to damaged blood vessel walls as part of the athrogenesis chain reaction which ultimately results in significant vessel damage. The action is rather complex and involves an understanding of the mechanism of cholesterol transport and lipoprotein metabolism that exceeds the scope of this chapter. Suffice to say, they reduce circulatory HDL levels (see Chapter 20 of *Rang and Dale's Pharmacology* for more detail). Examples of common statins are simvastatin, atorvastatin and pravastatin. As a physiotherapist, it is worth remembering that statins commonly cause muscle pain, so additional medical history may be required relating to how long they have been taking statins: does the pain correspond? Usually, by alerting the prescriber, the problem may be resolved by reducing the dose or changing the drug.

Diuretics increase NaCl excretion and thus water excretion. They are used to reduce oedema and decrease the load of the circulatory system on the heart. The most potent of these drugs are *loop diuretics* (e.g. furosemide), which inhibit transit of Na^+, K^+ and $2Cl^-$ ions in the loop of Henle. They are typically given orally or i.v. in acute pulmonary oedema. Another example is the *thiazides* (e.g. bendroflumethiazide), which are more often used to treat simple hypertension. They act on the distal tubule binding to, and inhibiting, the Na^+/Cl^- transport system which increases the loss of NaCl. A caution with these diuretics is the loss of K^+ ions which is important to monitor in patients who also have cardiac conditions requiring digoxin. An unwanted side effect of thiazides is erectile dysfunction. Potassium-sparing diuretics, such as spironolactone, are weak diuretics but they inhibit the secretion of K^+. These are particularly important to monitor as hyperkalaemia (too much potassium) can quickly become fatal.

The lungs

Bronchodilators that you will mostly come across are known as β_2-adrenoceptor agonists. They act on the β_2-adrenoceptors and therefore relax the smooth muscle, enhance the ciliary clearance of mucus and reduce the pro-inflammatory action of mast cells. They are usually given by inhalation but oral tablets and injection preparations are also available. For acute episodes, salbutamol and terbutaline are used because of their fast-acting nature and reasonable duration of approximately five hours. Salmeterol and formoterol are used as a dose rather than in acute situations as they take a little longer to act, but typically last much longer – approximately 8–12 hours. Other bronchodilators include xanthenes, such as theophylline or aminophylline (relaxation of smooth muscle but action is unclear), cysteinyl leukotrine receptor antagonists, such as montrelukast, and muscarinic receptor antagonists, such as tiotropium or ipratropium (increases acetylcholine expression and reduces mucus secretion).

Anti-inflammatory drugs, such as glucocorticoids, reduce the formation of pro-inflammatory cytokines and COX-2. Steroids also reduce the allergic response in asthmatics by inhibiting the specific cytokine that is responsible for regulating mast cells – the eventual result is fewer mast cells in the respiratory mucosa. They are given as a daily dose, commonly by inhalation – known as a 'preventer' (beclometasone, fluticasone, etc.). The full required response is only achieved after weeks of continuous use, hence the

essential role of respiratory practitioners to ensure that the patient understands how their drugs are to be taken. In advanced cases, a tablet form is used (e.g. prednisolone) to help bring the symptoms under control. In acute exacerbations i.v. steroids are used for immediate anti-inflammatory action to help maintain a patent airway. It is important to note that side effects are rare but the patient should only ever take steroids as directed by their prescriber. Oral thrush may occur. All steroids have a detrimental effect on the immune system and therefore opportunistic viruses may take advantage.

Neurological medicine

Parkinson's disease is associated with decreased dopamine in the substantia nigra and corpus striatum, and effects are typically tremor, muscle rigidity and hypokinesis (reduced voluntary movement). Levodopa is considered first-line treatment but is, unfortunately, extensively metabolised into dopamine peripherally preventing it passing through the blood-brain barrier and thus (i) requires a very high dose to spare enough levodopa to become centrally active and (ii) results in peripheral side effects. These problems are overcome by combining levodopa with an agent called carbidopa or benserazide that helps prevent its peripheral conversion which reduces the equipotent dose of levodopa to be reduced tenfold. Levodopa is short-acting and so side effects revolve around rapid alteration of symptoms ranging from dyskinesis and hypokinesis to rigidity fairly rapidly. These effects usually progress over time but more instant side effects include nausea and vomiting, and postural hypotension. Other treatment options increasing in popularity include dopamine receptor agonists, such as pergolide, ropinerole and pramipexole.

Anxiolytic and hypnotic drugs are widely used to reduce anxiety, muscle tone, aggression, convulsions and to help sedate patients. *Benzodiazepines* such as diazepam, temazepam, nitrazepam, etc. act on γ-aminobutyric acid ($GABA_A$) receptors enhancing the effect of this inhibitory amino acid in the central nervous system. This relaxant effect helps to break the anxiety (for example dental procedures, flying, phobias, etc.) and sustained muscle contraction/increased tone (e.g. cerebral palsy, acute back pain, etc.). They are typically well absorbed orally and peak plasma concentration occurs within the hour. Side effects include drowsiness and poor coordination, so the patient is advised not to drive.

Antibiotics

The side effects of most antibiotics are nausea and vomiting, gastrointestinal upset and skin irritation. However, if you notice that a patient is taking an aminoglycoside they may require a closer eye than normal. As with any antibiotic, you will want to know exactly why they are having them from an infection-control perspective.

Aminoglycosides are a particular type of antibiotic, the most commonly seen are gentamicin, streptomycin and neomycin. Usually given to treat Gram-positive cocci in aerobic conditions, they inhibit the process of protein synthesis from within the bacterium. Unfortunately, the aminoglycosides are ototoxic, i.e. they damage the sensory cells of the cochlea and vestibular systems which is irreversible and will result in vertigo, ataxia and dizziness, etc.

Analgesia/anaesthesia

Possibly the most common type of medication you will come across in your medical career. Pain medication may range from simple analgesia, such as paracetamol (acetaminophen) and ibuprofen to the extreme opioids and local anaesthetics. You may also encounter adjunct analgesia, such as amitriptyline and gabapentin.

Inhaled anaesthetics, such as nitrous oxide, halothane, isoflurane, etc., work by inhibiting synaptic transmission which peripherally block pain signals and centrally depress areas of the midbrain associated with consciousness and the thalamus associated with analgesia. As a physiotherapist you will probably only encounter nitrous oxide, N_2O, (when combined with oxygen at 1 : 1 ratio it is known as Entonox) as it is used extensively in acutely painful situations, such as childbirth or sporting injuries. The effects are extremely rapid and short-lived and the adverse effects are few: patients with pernicious anaemia (deficiency of vitamin B_{12}) should avoid long-term use (longer than six hours) as bone marrow depression may occur. Repeated and prolonged usage may produce amnesia.

Opioids are any substances that act in a similar way to morphine, while the term opiate is restricted to synthetic, morphine-like drugs. Opium has been used for thousands of years as a pain killer (and a recreational drug). The morphine-like drugs are known as agonists (morphine, diamorphine (heroin) and codeine), partial agonists (buprenorphine) and antagonists (naloxone) depending upon their expression on the cell (see the Glossary at the end of this chapter for details). Opioid receptors have three subtypes: μ, δ, and κ (mu, delta and kappa) which contribute to the analgesia at different levels. The opioid receptors are G-coupled receptors and the net effect is that of inhibition of pre-synaptic release of neurotransmitter, thus preventing nociception at the spinal level. While achieving analgesia, strong opioids also produce euphoria, especially if administered i.v. It is essential for all concerned to understand the importance of the side effects of morphine related drugs:

- sedation;
- respiratory depression;
- nausea and vomiting;
- peristalsis depression (resulting in constipation);
- histamine release (resulting in bronchoconstriction, hypotension and itching).

In the event of an overdose, naloxone, an opioid antagonist, will block the receptors and rapidly reduce the effects.

Unfortunate consequences of prolonged use include tolerance, whereby a higher dose is indicated for an equi-analgesic effect, and dependence, whereby withdrawal produces significant adverse physiological effects, such as irritability, weight loss, shaking, etc. In essence, morphine-like drugs are of vast importance but also a considerable danger to the patient – care must always be exercised when using them. Common opioids are listed below.

Morphine is reasonably fast-acting, is metabolised into a more potent form and has a half-life of approximately four hours. Usually given i.v., orally as a tablet or syrup, or transdermal patch. Commonly used in the community and in acute settings.

Diamorphine (heroin) is used clinically, especially intranasally for children in significant pain. Much faster-acting than morphine and is metabolised into morphine. Typically only used within hospitals. It has a high lipid solubility which allows a much faster 'rush' compared with morphine, but when taken orally its effects are largely similar.

Methadone is commonly used for heroin addicts as it can be much safer and has a much longer half-life (greater than 24 hours). The side effect profile is as above but with less euphoria.

Pethidine (aka meperidine in the USA) is given orally or by i.m. injection, usually by midwives during childbirth as, in contrast to morphine, it causes restlessness rather than sedation and therefore dose not reduce the uterine contraction force. With a shorter acting half-life (2–4 hours), pethidine is better controlled with little chance of accumulation.

Buprenorphine is a partial agonist and is given as a sublingual tablet or as an injection as the first-pass metabolism is so extensive it is rendered inactive orally. It is slow-acting with a predictable 12-hour half-life and less respiratory depression than morphine, hence its common usage in the community.

Fentanyl is delivered by epidural and transdermal patch. It has a short-acting half-life (2 hours) and is highly potent. *Remifentanil* (a variant form) is becoming increasingly popular as it is faster-acting and is associated with more rapid recovery.

Codeine/dihydrocodeine is used for mild pain and is available to buy from pharmacies. It is a prodrug (it is metabolised into morphine) and taken as a syrup or tablets commonly combined with paracetamol. It is approximately a fifth as potent as morphine but more readily taken up when taken orally. Codeine is also used as an antitussive (suppresses coughs) in children.

Tramadol is taken orally or i.v. It is generally well absorbed and has a predictable half-life of 4–6 hours. Although it has a weak opioid action, it also has a centrally-acting action which inhibits the uptake of noradrenaline. Tramadol is extensively used in the community for many musculoskeletal conditions.

Non-steroidal anti-inflammatory drugs (NSAIDs) provide symptomatic relief from mild-to-moderate pain associated with inflammation. The oldest form is aspirin, which has been in use since the late nineteenth century, and the most common is ibuprofen. They typically act by the inhibition of the COX enzyme and thus the production of prostaglandin and thromboxanes, which are pro-inflammatory. There are three identified isoforms of COX (1, 2 and, more recently, 3) although the third (also known as brain COX) may well not actually functionally exist in humans. COX-1 is primarily involved, although not exclusively, in the normal functions of the human body: homeostasis, gastric protection, regulation of renal blood flow and initiation of childbirth. COX-2, among other things, is responsible for producing pro-inflammatory mediators. Research in recent years has shown that a COX-2-selective NSAID gives rise to thrombotic events in those at risk and so most NSAIDs available have little or no selectivity between the two COX isoforms. Therefore, the side effects are considerable and the patient's past-medical history needs to be accounted for if they are taking NSAIDs. The inhibition of prostaglandins at the hypothalamus 'resets' the normal body temperature which will have been affected by a bacterial infection, thus producing an antipyretic effect.

Gastrointestinal disturbances are the most often cited side effect of the NSAIDs, which are as a result of COX-1 inhibition. COX-1 products are responsible for the inhibition of acid into the stomach and maintenance of the cytoprotective layer which protects the stomach lining from the acidic gastric juices. Inhibition of COX-1 is detrimental to these essential components and, as such, the stomach lining is under threat. The prostanoids responsible for renal blood flow are also a product of COX-1, specifically PGE_2 which dilates the vessels. Healthy individuals are at little risk but older patients, infants and those with existing renal insufficiency are at particular risk; therefore, NSAIDs with COX-1 inhibition (such as ibuprofen) are contra-indicated. Approximately 5–10% of patients with asthma will have a reaction to NSAIDs.

While aspirin and ibuprofen are the most common, readily-available NSAIDs from pharmacies, there is a whole range of NSAIDs, each with varying degrees of COX-1 : 2 ratio and potency, etc. As a general rule, those ending in '-fen' are more weakly COX-1 selective and those ending in '-coxib' are COX-2 selective.

Paracetamol (aka acetaminophen in the USA) is one of the most common analgesic drugs in the world. It can be taken for mild-to-moderate pain and fever, and, although classed as a NSAID, it lacks anti-inflammatory action and associated gastric irritation. The action is largely unknown as its action on COX-1 and 2 is limited: taken orally, it is well-absorbed with a half-life of approximately three hours. Between 5% and 15% of paracetamol

is metabolised into n-acetyl-p-benzoquinone imine (NAPQI) which binds to hepatic and renal tubule cell proteins, resulting in necrosis. However, in therapeutic doses the body's glutathione binds to NAPQI, allowing it to pass harmlessly in the urine. In high doses, the concentration of NAPQI exceeds safe levels, resulting in the slow process of multiple organ failure. Paracetamol is commonly seen as a reasonable option for suicide but the reality is rather unpleasant in all respects.

Tricyclic antidepressants (TCAs) (such as amitriptyline) are very useful in the management of neuropathic pain. They are considered to be an adjunctive analgesic as their primary indication is not that of pain. They act centrally by inhibiting the uptake of noradrenaline, which is independent of their antidepressant action. It is rather important to stress this to the patient, as they will automatically assume that the doctor has declared them to be depressed or as having a mental illness. This is entirely wrong and at every opportunity they should be re-assured that this is not the case. Side effects are generally sedation, so this drug is normally taken at night.

Anti-epileptic drugs are also an adjunct used for neuropathic pain. The Na^+ channel blockers (phenytoin and carbamazepine) target excessively depolarising cells – the more frequently they fire, the larger the blockade. Clearly, by blocking the sodium channels, they will arrest the action potential. Calcium channels may also be targeted by drugs, such as gabapentin and pregabalin. They target T-type calcium channels, specifically the $\alpha_2\delta$ subunit which helps to regulate the output of the neurones. The side effects are ataxia and sedation, but the drugs are usually well tolerated. As for the amitriptyline above, these drugs are adjuncts to conventional analgesia and therefore you must reassure the patient that these are also used for pain.

Local anaesthetics such as lidocaine (lignocaine) and bupivacaine deserve a special mention as many orthopaedic medicine physiotherapists are now injecting anaesthetics for a variety of painful conditions. Local anaesthetics block the Na^+ transport channels preventing action potentials close to the site at which they are introduced. They have a higher affinity to small-diameter neurones than to larger fibres and therefore the nociceptive Aδ and C fibres are targeted first. Motor neurones are generally rather resistant. Although they are typically safe, if they do enter the systemic circulation they may present adverse effects, usually to the central nervous and cardiovascular systems. Their effect can be that of depression or stimulation of the central nervous system, which may result in convulsions, agitation and respiratory depression. Cardiovascular effects are vasodilatation and depressed myocardium, and therefore decreased blood pressure. For the purpose of local nerve blocks, etc. an anaesthetist may also mix in adrenaline to create vasoconstriction, thus reducing the systemic distribution and prolonging the effect of the local anaesthetic.

FINAL THOUGHTS

So, there you have it! Pharmacology is both relevant and fascinating to learn about. You are not expected to understand the minutiae of biochemical interactions (that is what clinical pharmacologists/physiologists are for) but please understand that your patients will have these drugs acting within them as you treat: consider why, and how, it will impact your clinical reasoning. Hopefully, you now consider the prescribing route as a legitimate option as a physiotherapist and would like to further enhance your pharmacological knowledge. Above all, if you are unsure about *anything*, make sure you find the answer *before* you act. As a clinician, your career and the patient safety hangs on your every action.

Pharmacology glossary

This glossary is intended to help break down some of the barriers which may be presented by the sometimes inaccessible language of pharmacology.

5-HT (5-hydroxytriptamine) Serotonin – a neurotransmitter

Action potential (AP) The transmission of a signal through the length of an axon. This is an all or nothing affair, i.e. to stimulate an AP the depolarisation must reach a threshold value after which the AP is propagated, regardless of the strength of the initial stimulus. The resting potential of the cell is −70 mV. This is maintained by the active transport of Na^+ out and K^+ in at a ratio of 3 : 2. Stimulus will result in Na^+ channels opening allowing sodium ions to rush in and the membrane will begin to depolarise. If this depolarisation reaches approximately −55 mV – the threshold value – it is a successful stimulus and more Na^+ flood in. At approximately +30 mV potassium channels open and K^+ begins to leave the cell, thus slowing the change of potential across the membrane. Once +40 mV is reached, sodium channels close and repolarisation begins as K^+ is still leaving the cell. At about −55 mV the potassium channels close and normality begins to return following a brief hyperpolarisation. All of this triggers off the same chain of events in adjacent voltage gated channels therefore sending the AP down the axon

Adjunct analgesia A medicine whose indication is something other than pain, but whose use provides analgesia (e.g. amitriptyline)

Agonist, antagonist, partial agonist, inverse agonist An agonist will bind to a receptor and produce an intracellular reaction, whereas an antagonist will bind to a receptor and have no action at all (except for the physical aspect of blocking a receptor site). A partial agonist will exert a submaximal reaction, but a positive one nonetheless, and an inverse agonist will inhibit normal functioning of the cell, i.e. reduce expression. An antagonist will reduce the action of an agonist, partial agonist and an inverse agonist by competitive inhibition

Bioavailability The amount of available drug in the blood expressed as a percentage of the oral amount given. Absorption from the gut and first-pass metabolism may reduce the quantity of the drug reaching the blood

Cyclooxygenase (COX) COX binds to arachidonic acid on the inner surface of the cell membrane acting as a vital step in the production of prostaglandins. COX-1 is the 'good guy', responsible for vascular homeostasis, gastric protection, renal function and platelet function. COX-2 is mostly the opposite, enhancing gastric acid and inflammatory mediators and reducing gastric bicarbonate secretion, etc. There is talk of a 'COX-3' which so far has only been found in the brains of dogs. This third isomer has sparked debate regarding its contribution to persistent pain

Elimination constant (K_{el}) The rate at which a drug is eliminated from the body

First-pass metabolism This is the process that drugs entering the system undergo when taken orally. Once absorbed from the gut, the drug is then transported via the hepatic portal system to the liver where it may undergo extensive metabolism. This pre-systemic assault course reduces the amount of drug that can act on the body and thus gives rise to the concept of bioavailability. This system may be manipulated, however, as inactive compounds may be given which are then metabolised by the liver into an active drug – these are called 'prodrugs'

G-coupled protein receptor (GCPR) The GCPRs are very abundant and highly important in drug actions – they are a receptor positioned on a cell membrane with associated proteins within the cell whose job it is to ignite a cascade which completes whatever task that particular cell does once activated. A particularly important GCPR is the opiate receptor whose ligand may be endogenous (home grown) or exogenous (e.g. a codeine tablet). The resultant action is inhibition of Ca^{2+} influx at the neurone terminus preventing release of neurotransmitters into the synapse

Half-life ($T_{1/2}$) The period of time required for the plasma concentration of a drug to have. This is important for prescribers to understand as it will influence dosing

Metabolism Metabolism is the enzymic conversion of one chemical to another, predominately by the liver. It is part of elimination (the other being excretion) which involves removing the drug from the body, but it may also be responsible for producing active drugs, for example morphine is metabolised into the more potent morphine-6-glucuronide

Neurotransmitters Chemicals that are released across the synapse after an action potential which will excite the adjoining cell. Examples include:

- acetylcholine (ACh);
- noradrenaline (NA);
- dopamine;
- opioids;
- histamine;
- serotonin (5-HT);
- adenosine.

Opiate receptor These are GCPR receptors and found everywhere. There are three main subunits to think of: μ (mu), δ (delta), and κ (kappa). A recent discovery is called ORL-1. Their existence is not to await the time of your life when you rely on codeine or morphine, etc. but to allow cell activation from one of the body's own home grown (endogenous) opioid peptides, such as endorphins, enkephalins and dynorphins. Each subunit has its own particular outcome when stimulated but all are involved in pain analgesia. The overall effect is to prevent the exocytosis at the terminus of the neurone and thus arrest the transmission of nociception

pH and ionisation When considering the transit of drugs from outside the body through wherever it is they are acting to eventually being on the outside again; it is important to mull over the ionisation of the drugs in relation to membrane permeation. As drugs are either weak acids or weak bases they exist in a ratio of ionised and unionised states which varies with the environmental pH. The dissociation constant, kP_a, is the pH at which the dissociated and undissociated are equal and is essential for predicting how a drug will act in the body.

The ionised species ($BaseH^+$ or $Acid^-$) have low lipid solubility and therefore may not traverse the phospholipid bilayer (except when specific ion transporters exist). The lipid solubility of the unionised species depends upon it own chemistry, but most are sufficiently lipid soluble to permit membrane permeation

Pharmacodynamics What the drug does to the body! The physiological action of the drug and its manifestation in terms of cell expression, symptom alteration and pathology, etc. Drugs work by:

- receptor mediation (opioids);
- enzyme inhibition (NSAIDs);
- ion channel modulation/ inhibition (local anaesthetics);

- carrier protein modulation/ inhibition (PPIs);
- physiochemical (antacids).

Pharmacokinetics What the body does with the drug, i.e. how is it absorbed/distributed/ metabolised/excreted, etc. The route of administration plays a huge part in kinetics, for example oral medications undergo first-pass metabolism where they are taken through the portal system and into the liver. They also depend on a number of factors, including pH, gut motility, epithelial conditions etc. Intravenous drugs have 100% bioavailability and are rapid acting. Transdermal drugs have to be lipid-soluble to traverse the subcutaneous tissue

Prodrug A drug that becomes activated once metabolised by the human body

Saturation kinetics The disappearance of a drug from the plasma may not follow the exponential or bi-exponential but rather a linear (removed at a constant rate independent of plasma concentration). This is known as *zero-order kinetics* and the former is known as *first-order kinetics*. However, saturation kinetics is a more user-friendly, and functional, term. Essentially, the plasma C is not relevant when considering the rate of elimination as in exponential first-order kinetics owing to the saturation of the elimination mechanism. This is important as these drugs (and ethanol is a very common example), in high doses, take a very long time to be removed from the system. The time taken for alcohol to leave your system is therefore proportional to the amount you drink (assuming you drink so much that you saturate the elimination mechanism) whereas a drug such as heroin will initially clear much faster from the system if more is taken, meaning that taking twice the amount does not take twice as long to clear

Single compartment model This is a hypothetical idea aimed at simplifying distribution, apparent volume of distribution, V_d, and drug administration. For example, there is a body consisting of a single, well-stirred compartment into which a quantity of drug (Q) is added rapidly by intravenous infusion (IVI) and from which it may escape by either metabolism or excretion. The apparent volume links the total amount of drug in the body to its concentration in the plasma. The quantity of a drug in the body when administered in a single bolus is equal to the administered dose, Q. Therefore, the concentration at time 0 (C_0) is given, thus: $C_0 = Q \div V_d$. In practice, C_0 is calculated by extrapolation of the linear portion of the semi-logarithmic plot of concentration against time, back to the intercept at $t = 0$. Plotting C_t on a log against linear t yields a straight line – the inverse of this rate is the elimination rate constant, k_{el}. While this is important to understand elimination and distribution of a drug, it is very much worth reading it from a dedicated pharmacology book!

Two compartment model This is an approximation of distribution, rather than the hypothetical single compartment model. The compartments are the peripheral (all tissues) and central (plasma). The effect of adding an additional compartment develops a bi-exponential curve, α, where the drug leaves the first central compartment and enters the peripheral (no elimination occurring yet), and, β, elimination from tissues. This provides an estimate of k_{el}. Of course, this is a very simple concept and lumping all tissues into one 'compartment' is bad science

Volume of distribution, V_d Distribution of drugs is to plasma, extracellular fluid and total body water. The volume of distribution depends upon the molecular weight, lipid solubility, ionisation and protein binding. A drug must be lipid-soluble to pass into cells and, in most cases, the blood-brain barrier. Large molecules and protein-bound drugs will not leave the circulation and therefore have a low V_d. The distribution is an exponential curve with an α-phase denoted by the half life ($T_{1/2}$) whereby 50% of the drug is removed from the plasma (by distribution/metabolism). Some drugs show zero order kinetics whereby the elimination from the plasma is constant – ethanol and phenytoin are examples.

Drugs

Some basic information relating to relatively important drugs which you may come across in your practice.

Aciclovir (antiviral) Used to treat infection by herpes virus

Adenosine (anti-arrhythmic) Re-establishes sinus rhythm following supraventricular tachycardias (SVTs)

Adrenaline (epinephrine) Stimulates heart activity during arrest and increases blood pressure by vasoconstriction. Used for anaphylaxis caused by bronchodilation and blood pressure stabilisation. Also used in conjunction with local anaesthetics to prolong the effects and reduce the risk of systemic effects of the anaesthetic

Alendronate bisphosphonate Reduces the rate of bone absorption by inhibiting osteoclast activity. Used primarily for postmenopausal and steroid associated osteoporosis

Alfentanil (opioid analgesic) Rapid onset opioid chiefly used for its respiratory depressive qualities in ventilated patients. Also used peri-operatively to enhance analgesia

Aminophylline Bronchodilator typically used for severe acute asthmatic episodes – given i.v.

Amiodarone An anti-arrhythmic used to slow conduction of the

nerve impulses and treat tachycardia

Amitriptyline A tricyclic antidepressant (TCA) which is used more for neuropathic pain than depression. Amitriptyline competitively blocks binding sites of amine transporters reducing the uptake of 5-HT and noradrenaline, thus increasing the synaptic expression at the synapse. This is cited as being a mechanism of reducing depressive illness by enhancing neurogenesis and reducing apoptosis via gene transcription. Amine re-uptake at the bulbospinal pathway is a reasonable explanation for the downward inhibition of pain

Amlodipine (calcium channel blocker) An anti-hypertensive

Aspirin An anti-inflammatory drug used as an analgesic and antipyretic in higher doses (300–900 mg) and as an anti-platelet in lower daily doses (75 mg)

Atenolol (beta-blocker) Used to treat hypertension

Atorovastatin (statin) Reduces the plasma HDL cholesterol levels protecting against atherosclerosis of the blood vessels

Atropine Blocks the action of acetylcholine and thus relaxes smooth muscle. Typical usages include pupil dilation and irritable bowel syndrome

Beclomethasone A corticosteroid used as an inhaler for asthma. This is used as a dosed drug rather than as required because of the accumulative effects

Bendroflumethiazide (thiazide diuretic) A diuretic that may be used for hypertension and oedema

Captopril (ACE inhibitor) Prevention of the formation of the powerful vasoconstrictor angiotensin II and therefore reduces blood pressure

Carbamazepine An anticonvulsant used for treatment of seizures. Also used for the pain associated with trigeminal neuralgia

Chlorpromazine An antipsychotic that sedates patients offering relief of schizophrenia and severe agitation

Cimetidine (H_2-receptor antagonist) Decreases the secretion of acid into the stomach relieving the symptoms of gastric reflux/peptic ulcers, etc.

Codeine (opioid analgesic) Relatively common opioid which is approximately 20% the potency of morphine. Usually sold in a combined preparation with paracetamol. Used to treat mild-to-moderate pain

Diazepam (benzodiazepine) Used to relax muscles and reduce anxiety. Usually given to patients in the community with acute back pain and muscle spasms

Diclofenac Non-steroidal anti-inflammatory (NSAID) for mild-to-moderate pain

Dihydrocodeine AKA DF118 (opioid analgesic) An opioid that is slightly more potent than codeine but less potent than morphine. Used for moderate pain and often combined with paracetamol in an over-the-counter preparation from pharmacies

Erythromycin (antibiotic) A bacteriostatic antibiotic with similar spectrum effectiveness to penicillin, typically very useful for those with an allergy to penicillin. Very common side effects include diarrhoea, vomiting, abdominal pain and nausea as it is a motilin agonist.

Fentanyl (opioid analgesic) Fast-acting with a short half-life analgesic, typically used in acutely painful situations

Ferrous sulphate This iron salt tablet is prescribed to treat anaemia

Fluoxetine (selective serotonin reuptake inhibitor) An anti-depressant that inhibits the re-uptake of serotonin within the synapse enhancing its effect on the post-synaptic membrane. Commonly known as Prozac in the USA

Furosemide A very fast-acting diuretic that can reduce pulmonary oedema in emergencies

Gabapentin An anticonvulsant used to down-regulate hyperactive nerves that cause seizures and neuropathic pain

Gliclazide An anti-diabetic drug that lowers blood sugar levels. Used in type II non-insulin-dependent diabetes mellitus (NIDDM)

Glyceryl trinitrate (GTN) Used in the treatment of angina by dilating the coronary vessels. Delivered by a spray under the tongue during acute episodes

Heparin (anticoagulant) Used as prophylaxis against clotting (either in deep vein thrombosis (DVT) or pulmonary embolus (PE))

Hydrocortisone A steroid used to treat inflammatory disorders (such as rheumatic conditions, psoriasis, etc.) and immunosuppressant (for example to prevent organ rejection / reaction following transplant)

Ibuprofen (NSAID) A very common, readily available anti-inflammatory drug used for mild-to-moderate pain as a result of inflammation

Insulin (anti-diabetic hormone) Given as an injection to lower blood sugar in type I insulin-dependent diabetes

Lansoprazole (proton pump inhibitor) Used to reduce the amount of acid secreted into the stomach for those with gastric reflux/ulcers, etc.

Metformin (biguanide) Decreases glucose production as a treatment for type II diabetes. This tablet is normally given in conjunction with gliclazide

Methotrexate (cytotoxic and immunosuppressive) This important drug inhibits normal protein synthesis therefore leading to cell death. It can be used in inflammatory rheumatic disorders and tumours, as well as leukaemia

Metoclopromide (dopamine antagonist – anti-emetic) A common anti-emetic used to reduce the symptoms of nausea and vomiting

Morphine (opioid analgesic) A controlled drug used for severe pain. Respiratory depression, nausea and vomiting are the most prominent side effects of note

Naloxone (opioid receptor antagonist) An opioid antagonist that can reverse the effects of an opioid agonist such as morphine. Often used in emergency situations in cases of opiate overdose. More recently, it can be found in oral preparations in conjunction with opioids such as oxycodone to prevent the constipation side effects

Naproxen (NSAID) A prescription-only NSAID used for chronic and generally more severely painful inflammatory conditions

Odansetron A very effective serotonin agonist used to treat nausea and vomiting in postoperative patients

Omeprazole (proton pump inhibitor) Reduces the amount of acid released into the stomach

Paracetamol (acetaminophen) Classed as a NSAID despite its limited anti-inflammatory properties. Used commonly for mild-to-moderate pain and to help reduce temperature (antipyretic)

Pethidine (opioid analgesic) Rapid acting analgesic used for moderate-to-severe pain, especially during childbirth as it lacks the sedative effect seen in other opioids and therefore does not inhibit uterine contractions

Prednisolone (steroid) Suppression of inflammatory, immune and allergic disorders. Also used in organ transplant to help prevent rejection

Pregabalin (anticonvulsant) Used to help prevent seizures in epilepsy and control neuropathic pain

Propofol An anaesthetic induction agent and highly potent sedative

Ramipril (ACE inhibitor) An anti-hypertensive that produces peripheral vasodilatation

Salbutamol (β_2 agonist for acute asthma) Used in metered doses through an inhaler for acute asthmatic episodes. It is fast-acting with a short half-life hence why it is used as required

Salmeterol (β_2 agonist as a dose) Used in metered doses through an inhaler but as a dose to help prevent acute episodes of bronchoconstriction. Compared with salbutamol, it is slower-acting but longer-lasting

Senna Used for constipation, it acts as a stimulant for the colon which will help to initiate bowel movements

Simvastatin Reduces HDL cholesterol levels in the blood as a prevention of atherosclerosis

Streptokinase (thrombolytic) Used in acute myocardial infarction as it breaks down the fibrin within the clot very quickly

Tamoxifen (anti-oestrogen) Used for those with oestrogen-receptive breast cancer to help slow the growth or to prevent recurrence post-removal

Terbutaline (β_2 agonist) A bronchodilator used as a dry powder in a metered dose inhaler to help prevent asthmatic exacerbations

Tramadol (analgesic) Used to treat moderate-to-severe pain. Traditionally classed as an opioid but it also has a secondary serotinergic action which enhances the analgesic properties

Warfarin (anticoagulant) Used to help prevent deep vein thrombosis and pulmonary embolism, stroke, etc. by reducing the clotting risk. Patients taking warfarin are expected to have their clotting rate monitored regularly

ACKNOWLEDGEMENT

The author would like to acknowledge the help of Dr Douglas McBean PhD, Senior Lecturer in Physiology and Neuroscience at Queen Mary's University.

FURTHER READING

Rang, H.P., Dale, M.M., Ritter, J.M., Flower, R.J., 2012. Rang and Dale's Pharmacology. Churchill Livingstone, Edinburgh.

Firstly, this is the book to have in your bag if you are even slightly interested in pharmacology. Reading this book is a pleasure; the relaxed rhetoric, elegant writing and subtle wit is evident throughout the whole book with the added benefit of actually learning something (rather like Tidy's Physiotherapy*). While it is a useful reference text, it is possible to 'read' the book, as it flows effortlessly from the history of pharmacology, through the basics of pharmacological science to the minute detail of the drug types.*

Neal, M.J., 2012. Medical Pharmacology: At a Glance. Wiley-Blackwell, Oxford.

Secondly, those who like concise details with no frills will love this book. The simple diagrams and explanations leave you in no doubt that you are reading a pharmacology book, this is a particularly popular book because of this. This book does rather jump in at the deep end, which may mean you require a physiology book if your knowledge in this area is lacking.

Payne, R.A., Donaghy, M., 2010. Payne's Handbook of Relaxation Techniques: A Practical Guide for Healthcare Professionals. Churchill Livingstone, Edinburgh.

While not explicitly related to pharmacology, this is a great book for understanding the effects of relaxation/stress on the body. A great synopsis of the sympathetic outflow and the clinical implications help the reader to understand the relationship between the patient and their symptoms.

Other books are, of course, available and pre-purchase reading/library usage is recommended.

Chapter 5

Reflection

Sarah Prenton, Lindsey Dugdill and Linda Hollingworth

INTRODUCTION

Reflection is a process that is acknowledged as being fundamental to skilled clinical practice for healthcare professionals. However, the value and application of reflection is widely debated. In some instances, reflection is just seen as the completion of a portfolio submitted at the end of a period of learning; however, it is argued here that reflection should be much more – an attitude or approach that enhances professional development and practice. This chapter aims to introduce physiotherapy students and physiotherapists to the concept of reflective practice, including definitions of reflection and the rationale for reflection in practice. It then highlights the key processes and important principles involved in reflection. It concludes by discussing ways to reflect, including tools and models that can be used to explain and encourage reflection.

This chapter is built around Salford physiotherapy graduates' views on reflection and becoming reflective practitioners in the real world. Consequently, the emphasis of this chapter is on how to successfully start to analyse practice. Ideally reflective practice should involve an emotive, as well as technical element; however, the consideration of 'feelings and emotions' is also frequently cited by physiotherapy graduates as a barrier to initial engagement.

> *… we're 'sciencey' people, we took a 'sciencey' course and so to sit down and write on a piece of paper about how something made you feel doesn't make sense…*
>
> Graduate Reflective Group 2010 (Student 5)

> *… to put an emotional aspect about what I'm doing, what I'm feeling is not in my makeup…*
>
> Graduate Reflective Group 2010 (Student 3)

This chapter aims to engage physiotherapists who are new to these concepts by giving them a toolkit to begin, and develop, as reflective practitioners. Becoming a reflective practitioner is an essential life skill for all physiotherapists. Skills of reflexivity – being able to think about and critically analyse experiences and understand their meaning – is important in becoming more proficient in practice. Reflecting on day-to-day experiences helps professionals to 'think through' decisions that have been made with respect to say a particular patient case or work scenario. This is essential in new or novel situations – which for the student practitioner will be most of day-to-day practice to begin with.

DEFINING REFLECTION

There are numerous definitions of reflection, a selection are provided below.

ABC Definition

Reflection is looking at whole scenarios from as many angles as possible: people, relationships, situation, place, timing, chronology, causality, connections and so on, to make situations and people more comprehensible

(Bolton 2005: 9)

Reflection in the context of learning is a generic term for those intellectual and affective activities in which individuals engage to explore their experiences in order to lead to new understandings and appreciation.

(Boud et al. 1985: 19)

Reflection is a form of mental processing – like a form of thinking – that we use to fulfil a purpose or to achieve some anticipated outcome. It is applied to relatively complicated or unstructured ideas for which there is not an obvious solution and is largely based on the further processing of knowledge, understanding and possibly emotions that we already possess (Moon 1999).

It is clear from the numerous definitions of reflection that people develop personal and individual views of what reflection is; this is influenced by factors such as disciplinary background. The differences in terms used, interpretation and perspective can result in confusion; however, there are clear commonalities and overlap within the work of different authors on the subject. In its simplest form, reflective practice is a process of thinking about and analysing an experience or set of actions. In this case, it would relate to a work scenario or situation. There are many tools that can help processes of reflection, such as keeping reflective logs or diaries, which will be discussed later in the chapter; this is common practice with many other disciplines, such as nursing and sports coaching (Knowles and Telfer 2009).

Knowles and Telfer (2009: 24) make it clear that reflective practice is not just a process of thinking where you literally 'go round in circles', but a cognitive process which encompasses 'deliberate exploration of thoughts, feelings and evaluations focused on practitioner skills and outcomes. The outcome of reflection is not always preparation for change, or action based, but perhaps confirmation/rejection of a theory or practice skill option'. This description gives the process of reflection a feeling of trajectory where the individual undertaking the reflection ends up in a different place from where they started, whether through new understanding of practice, new knowledge or both. This process of moving forward is crucial in gaining a better understanding of effective practice. Understanding what is *not* working well in a service, process or any practice situation is just as important as identifying positive practice – it highlights what needs to change.

For further reading on the topic of reflection and reflective practice see Ghaye (2005), Moon (1999) and Schon (1983).

Rationale for reflection in practice

Effective reflection should improve understanding of the positive and negative aspects of practice, and, subsequently, should enable more informed decisions to be made in the future, leading to better quality of life and health outcomes for the patient. Over time, this enables practitioners to become expert at what they do. Developing excellent clinical reasoning (discussed further in the following section) is one of the fundamental expectations for health professionals and reflecting on practice can help to improve this process. Reflective practice is a lifelong skill, as the need to continually update practice is something all professionals face in their career. Therefore, becoming a skilled reflective learner early in an individual's professional career is essential.

As well as developing individual skills and expertise, it is vital that physiotherapists understand how best to work together in interprofessional teams (Zwarenstein and Reeves 2006). Therefore, reflection should not be just about self and the work context but the wider scope of team activities. Interprofessional working requires an understanding of the boundaries or limits within which any professional both *could* and *should* operate (Dugdill et al. 2009). Reflective practice plays a vital part in enabling professionals to learn and understand the impact of their actions. External factors, such as workplace policy and professional body requirements, as well as internal factors, such as attitudes, skills, experience and team dynamics, can all be issues that are useful to report on.

In time, as the skills of reflective practice develop in an individual, the value of continuing to utilise reflective practice skills becomes obvious as they usually lead to improved clinical work performance and, ultimately, to better patient outcomes. It is much better if the inherent value of reflective practice becomes an accepted way of working for a professional from the early days as a student and it should be continued as a life-skill forever. If reflective practice is just seen as a task to be completed for an assessed piece of work it is unlikely to become a habitual and valued process that becomes embedded in practice. One of the biggest challenges faced by tutors of reflective practice is convincing students of its value in the longer term. Many physiotherapy graduates reported that initially they did not see the value in reflective practice:

> *… you just don't realise how important it is…*
>
> Salford Graduate Reflection Group 2010 (Student 1)

> *… you are there [University] for physio, so anything non-clinical, non-anatomy, non-looking at the hip! – well I can justify why I'm doing this, doing that, but reflection to me really was a waste of time…*
>
> Salford Graduate Reflection Group 2010 (Student 3)

> *Until it was an area that required a formal assessment I considered it to be of less worth than the other aspects of my course.*
>
> Final year reflective assignment 2010 (Salford student)

> *Looking back I believe I underestimated the importance of the reflective process and how it could contribute to my development.*
>
> Final year reflective assignment 2010 (Salford student)

The conclusion from these statements is that reflective practice has to be undertaken to be understood and valued.

CLINICAL REASONING AND EVIDENCE-BASED PRACTICE

A common characteristic of students, and even new graduates, is that they may display knowledge but cannot always apply it in practice. In other words, they struggle with what is known as clinical reasoning.

ABC Definition

Clinical reasoning (or practice decision-making) is a context-dependent way of thinking and decision-making in professional practice to guide practice actions. It involves the construction of narratives to make sense of the multiple factors and interests pertaining to the current reasoning task. It occurs within a set of problem spaces informed by the practitioner's unique frames of reference, workplace context and practice models, as well as by the patient's or client's contexts.

It utilises core dimensions of practice knowledge, reasoning and metacognition, and draws on these capacities in others. Decision-making within clinical reasoning occurs at micro-, macro- and meta-levels and may be individually or collaboratively conducted. It involves metaskills of critical conversations, knowledge generation, practice model authenticity and reflexivity (Higgs and Jones 2008: 4).

As it can be seen it is therefore obviously not something that can come from absorbing.

In order to develop clinical reasoning, novice practitioners first need to acquire a large amount of biomedical knowledge. It is then often assumed that once acquired, and simply with repeated practice, they will then be able to make appropriate links between different aspects of that knowledge. This is usually not the case; hence the difficulties seen in application.

As Higgs and Jones (2008) state, clinical reasoning relies on the integration of three key processes in order to be successful (Figure 5.1).

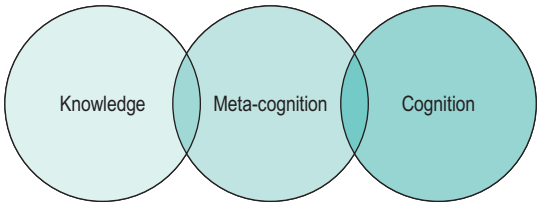


Figure 5.1 Integration of three key processes.

Knowledge: this relates to an individual's personal knowledge of anatomy and physiology, and knowledge of pathological and inflammatory processes. It also includes knowledge of evidence and how to evaluate it appropriately. It should also encompass valuing and considering the patient's knowledge of their health.

Cognition: this relates to an individual practitioner's ability to analyse data, synthesise information and develop strategies of inquiry. In other words, their ability to think through the information you collect and problem solve.

Meta-cognition: this relates to an individual practitioner's ability to review the strategies they are using, consider what might have influenced/biased/limited them, compare this experience against previous ones and generally be critically analytical of their practice.

This last component, meta-cognition, is considered the integrative link between the other two areas. Reflecting on practice takes place within the meta-cognitive domain.

The necessity of this integration is because of the fact that no situation will be like another: each clinical encounter will be unique. It is therefore important to consider all aspects of the experience in order to develop skills (cognitive and knowledge) for the next time. Failure to follow through these lines of inquiry will result in potential errors in the conclusions and plans drawn.

Applying the above concepts to a real world physiotherapy context, for example the painful shoulder

A client's problem could be physical, psychosocial or a combination of these; it could be derived from a number of different origins and could be acute, chronic or an acute flare up of a chronic problem. The first time you are faced with this diagnosis the conclusions you draw might be ineffective or incorrect because of either a limitation in your knowledge base, problem-solving skills or limitations in your interaction with the patient for a variety of reasons. If you fail to reflect on the conclusions regarding a diagnosis and/or treatment plan then you are destined to make them again, even if they are not effectual. Likewise, if the conclusions you draw are correct you need to analyse how you came to them in order to perpetuate this with future patients. This is about understanding *'what works well and why'*.

It might, therefore, be assumed that the very first time you treat a condition your reasoning will be purely knowledge based. However, this is not the case as certain people

will have previous experiences, have read different sources of material, and observed other clinicians. It is this individuality that also requires analysis and reflection after the experience so that it can be utilised and developed for the future.

Novice practitioners tend to use what is often known as hypothetico-deductive clinical reasoning (Jones et al. 2008). This is where hypotheses (initial impressions) are systematically tested out in order to reach a logical, diagnostic conclusion. This requires a significant cognitive demand, tends to focus primarily on physical impairments and may still result in the actual reason for presentation being elusive. By contrast, an expert is said to be someone who is perceived to be *'capable of doing the right thing at the right time'* (Jensen et al. 2008: 123) – their method of clinical reasoning will be different in that it is based on pattern recognition (colloquially termed 'patient mileage') with not only large numbers of possible hypotheses considered but various other components of the individual patient and context taken into account. These include the patient motivations, reasoning based on the rapport and collaboration with the patient, envisioning the future and considering ethical implications (Jones et al. 2008). While seemingly more complex, this process actually requires less cognitive demand at the time of assessment.

The difference between the novice and the expert's clinical reasoning skills is, in part, because of an increased depth of knowledge but also how that knowledge is manipulated. This cannot be achieved through repetitive practice, but rather practice-analysis (reflection) and the re-practice (Boshuizen and Schmidt 2008). In other words, if you do not learn from your experiences you will never develop expertise. Non-reflective clinicians will have a tendency to only look for, and over-emphasise, certain signs and symptoms, disregard others which do not concur with their preconceived ideas and form conclusions which do not consider the individual; in other words, they are not patient-centred. This can result in substandard care for that individual. This occurs most commonly when less experienced practitioners try and 'mimic' expert clinical reasoning without fully understanding the processes necessary to truly achieve expertise.

It is interesting to note, however, that to an expert, who has been reflective, routine clinical practice will actually be relatively non-reflective unless the situation is novel to them. In this instance, however, it should be remembered that their role and responsibilities will be such that they will have many other activities that they will need to reflect upon, such as leadership/team-management, service development and research, which may be deemed as higher practices.

There can be a perceived tension between the drive toward developing the scientifically researched evidence base for physiotherapy practice and the value of professional knowledge developed through experience. Much has been written previously on the processes of reflective practice as being part of a continual learning cycle (Ghaye 2005). Students are taught from an evidence base of 'what works', but the literature is often not complete or not relevant for the working context, so experiential learning 'from practice' and evaluation of that practice is required in order to understand what processes and interventions work best in order to develop clinical reasoning skills.

National and local guidelines/protocols that are based on the best available evidence at any time are designed to ensure consistent high quality care of patients. However, there can be a tendency, as a clinician, to simply follow that guidance without questioning. Firstly, in reality clinicians need to be critical of the evidence itself as it is of varying quality. This is why undergraduates are shown how to evaluate the quality of evidence and this skill requires continued practice once qualified to ensure the application of the most appropriate theory to practice. Secondly, consideration of the evidence/protocol/guideline needs to be taken in the context of individual practice (see below).

EG Example

Mental practice of upper limb movement following a stroke

In the Royal College of Physician's national clinical guidelines for stroke (Intercollegiate Stroke Working Party 2008:76) 'Patients should be taught and encouraged to use mental practice of an activity as an adjunct to conventional therapy, to improve arm function'.

As a clinician should you therefore assume that you should be using this with every patient you encounter who has had a stroke? This recommendation was based on a single paper and therefore this recommendation needs to be considered in the context of the quality/quantity and transferability of the evidence it was based on, for example sample size, the part of the population it was conducted on (generalisability), etc. and also your own knowledge, experience and skills, and, most importantly, your patient's needs, goals and biopsychosocial abilities.

Additionally, as a clinician you need to be critical of current practice and may be working in a brand new or extended role (Figure 5.2). In this case, gaps in the literature base and/or your own experiences of implementing current practice may generate further questions for ongoing research. As Dugdill (2009:49) previously stated:

> *The advancement of a professional role requires that profession to be committed to the continual 'development of thinking' – both generating and testing new knowledge – in order to improve the*

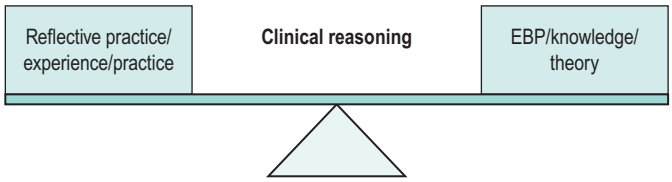


Figure 5.2 The interface between evidence-based practice (EBP) and reflective practice.

delivery, and hence impact, of the role. Sometimes professional practice may involve trying new things, being innovative and challenging the boundaries of existing practice. This may be especially relevant when a client fails to respond to a tried-and-tested approach and the practitioner feels the need to try something different.

Therefore, reflective practice should be seen as a central process for both continuing professional development (CPD) and evidence-based approaches, both of which are fundamental to high quality professional practice. Without all these described processes being in play you will not be able to achieve your full potential as a physiotherapist remembering that *'clinical reasoning and clinical practice expertise is a journey, an aspiration and a commitment to achieving the best practice that one can provide'* (Higgs and Jones 2008: 9).

For further reading on these topics please refer to Higgs et al. (2008).

REQUIREMENTS FOR REFLECTIVE PRACTICE

Becoming an effective reflective practitioner is now an expected requirement of all health professionals in order to drive continued improvement in practice and allow the interface between theory and practice to be constantly analysed by the professional.

In order to practice as a physiotherapist within the UK, practitioners must hold registration with the Health and Care Professions Council (HCPC). HCPC standards ensure that all health professionals must continue to develop their knowledge and skills in order to maintain registration. These standards (HCPC 2006) state that registrants must:

1. maintain a continuous, up-to-date and accurate record of their CPD activities;
2. demonstrate that their CPD activities are a mixture of learning activities relevant to current or future practice;
3. seek to ensure that their CPD has contributed to the quality of their practice and service delivery;
4. seek to ensure that their CPD benefits the service user;
5. present a written profile containing evidence of their CPD upon request.

The HCPC states that physiotherapists must participate in 'a wide range of learning activities through which health professionals maintain and develop throughout their career to ensure that they retain their capacity to practise safely, effectively and legally within their evolving scope of practice' (AHP Project 2003: 9). The use of reflection is identified as one of the key activities, especially in learning from practice in the workplace. The recognition of learning requires a process of reflection and evaluation and it is this process that can prove difficult. Yet, reflecting on workplace practice after it has occurred is integral to workplace learning. It enables subconscious thought to be critically examined, evaluated and articulated. It also allows theories from formal programmes of study to be connected with and integrated into practice.

The physiotherapy graduates discussed how, as well as reflective practice being a mechanism to consider practice, collecting evidence of the process was often the only evidence of learning and achievements. It's a way of 'monitoring you along a timeline' (Student 3) and 'a way of fulfilling your portfolio requirements' (Student 3).

USE OF REFLECTION AS A FORM OF ASSESSMENT

Undergraduate and postgraduate physiotherapy programmes commonly use reflection as a component of assessment; this can be formal, summative assessment or more informal, formative assessment. Because the work is being produced for an external reader who will make a judgement about the 'quality' of that reflection and often give it a mark, the nature and content of the reflection will, inevitably, be altered. There is much debate within the literature regarding the effects of assessment on the development of honest and truthful reflection. A student, knowing the reflective essay or portfolio is to be assessed, will frame the written response to make them (and their practice/expertise) appear in a positive light; hence, the 'messy reality' and mistakes of practice may be lost from the reflective account. Some argue that reflection and assessment are incompatible (Hargreaves 2004); however, many practitioners will admit that without the external motivation that assessment provides, combined with the formal training and feedback received through the process,

they would not have devoted the time and energy to developing the skill. Even the use of 'I' within academic writing can be seen as controversial in academic settings; traditional academic assignments require writing in the third person and discourage the expression of personal or unsubstantiated comment (Lindsay et al. 2010). The following quotes from physiotherapy students and graduates highlight the debate and motivations for undertaking assessed reflection:

> *… when being formally assessed I spent more time asking myself questions of my reflective practice and reflective writing style. I have also discovered that for me to be motivated to do something I need to have some form of praise to encourage me to do it…*
>
> Final year reflective assignment (Salford student 2010)

> *I may have adapted my writing because I knew that someone would read it and I was uncomfortable with talking about my feelings and this would have impacted on the depth of my analysis, this is supported within the literature…*
>
> Final year reflective assignment (Salford student 2010)

> *Some literature suggests that the descriptive nature of a reflection can be the result of its involvement in an assessment process because the reflector holds back on emotions, opinions and is less open and honest as someone will be reading it (Hargreaves 2004). Personally I do not believe this applies to me…*
>
> Final year reflective assignment (Salford student 2010)

> *Perhaps the answer is to almost 'force' me to use written reflection by making the amount completed potentially detrimental to attaining marks. I am then more accountable.*
>
> Final year reflective assignment (Salford student 2010)

> *… if I'm just going to write it for me to stick in my file it's just going to be a box ticking exercise…*
>
> Salford Graduate Reflective Group 2010 (Student 5)

> *… it [assessment] can change what you write…*
>
> Salford Graduate Reflective Group 2010 (Student 1)

Reflection used as assessment may bring additional requirements, such as links to portfolio evidence or to the research literature within a particular area. As a student, you may be asked to consider your reflection on practice within a theoretical context, for example: 'In your management of the patient reflect on the intervention techniques you chose to use and why? Please refer to evidence from the literature to justify your choice of treatment or intervention'.

It is important to remember that the sharing of reflection is within the practitioner's control. Ultimately, it is the individual's decision which aspects of their reflection are private and which are appropriate for sharing with another person, whether that is a mentor, manager or academic tutor. For instance, the reflective diary of a practitioner is normally considered private. The student can then use it as the 'aide memoire' to help them think back over a case or work scenario that they are analysing for their coursework.

> *I would keep a diary every day on placement … I would write up a full reflective piece for my visiting tutor and the others about 18 months later based on my diary…*
>
> Salford Graduate Reflective Group 2010 (Student 2)

PROCESSES OF REFLECTION: USING REFLECTION IN PRACTICE

ABC Definition

Reflection on/following action

Taking a step back from an experience or activity in time and space, using questioning to develop a better understanding of a situation or area of practice (CSP 2005).

Reflection following action/ reflection on action (Schon 1983)

This is the reviewing of an experience or event at some point after it has occurred in order to evaluate and understand it to a greater degree. It allows physiotherapists to explore and analyse their actions in order to investigate and articulate how they completed the task and why they did it in the way they did. There is an aim to make the tacit professional knowledge more open and explicit so that practice can be challenged and performance improved.

Various models have been developed to assist practitioners in developing reflection on action. These generally provide encouragement to follow a cycle of reflection and provide prompt questions to encourage deeper exploration; these are introduced later in the chapter.

EG Example

A standardised physiotherapy treatment protocol is being used for patients following an orthopaedic procedure as a result of the failure to progress for a number of patients. Reflection may be used to consider the following questions:

- What happened with the cases concerned?
- Was the protocol implemented as planned? Why/why not?
- What other factors (internal or external) may have influenced the outcomes?
- Can common features or patterns be identified?
- Can reviewing the literature in the area assist?
- Is the protocol up to date?
- Is the protocol still considered best practice?
- How does our protocol/outcomes compare with other similar departments?
- How does the implementation of the treatment compare to the research/other similar departments
- What other considerations/constraints need to be considered before suggesting/implementing change.

This exploration, which would ideally be done as a team, would then be used as a basis for planning.

Reflection following/reflection on action is useful for developing skills where incremental improvement is possible; however, it is also necessary to reflect during activity and before it takes place.

ABC Definition

Reflection in action

Professionals are able to think what they are doing while they are doing it.

(CSP 2005)

EG Example

A physiotherapist treating a patient who reacts in an unexpected way to an intervention is able to consider the factors influencing the patient's reaction, explore a range of alternative approaches, and implement and evaluate them without interrupting the flow of treatment.

Or

A physiotherapist who receives particular pieces of information during a subjective history may adapt the components of the objective examination or change the order of their assessment.

Reflection during action/reflection in action

Experienced physiotherapists (skilled or expert practitioners) have the ability to 'read' experiences and adapt their intervention or behaviour during that intervention as opposed to needing to evaluate the experience after it occurs. This is sometimes known as reflection in action (Schon 1983). This can be regarded as more than responding intuitively, as it should involve some conscious consideration of what is happening, how effective the behaviour is and whether there might be more appropriate alternatives (Wilson 2008). This skill of 'thinking on your feet' can be taken for granted by experienced physiotherapists but can represent a challenge for the novice practitioner.

Reflection before action/reflection on the future

ABC Definition

Reflecting on the future

The act or process of reflecting on desirable and possible futures with the purpose of evaluating them as well as considering strategies intended to achieve the objective(s).

(Wilson 2008: 180)

Wilson (2008) discusses the importance of considering reflection *before* action and proposes that this natural part of human thought processes has been neglected in the literature on reflective practice. He argues that professionals reflect after and during action but *'should systematically reflect on the future'* (p. 178) (Figure 5.3).

> *Reflecting on what might be is one of the most powerful mental tools we have at our disposal. Without this ability to speculate about what might be, we would be constrained within the present or the past. If we lack the ability to reflect on the future there could be no plans, no hopes, no aspirations, no wants, no dreams and no desires.*
>
> (Wilson 2008: 180)

Reflecting with a supervisor

Participation in clinical supervision is regarded as a vital aspect of professional development.

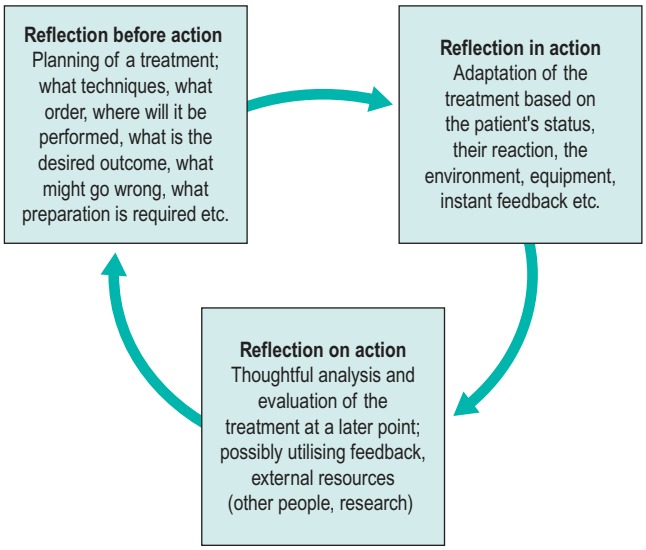


Figure 5.3 The process of reflection in treatment of Description a patient.

Supervisors, therefore, have a huge impact on how novice practitioners view reflective practice.

> *It very much depends on the whole attitude of educators…*
>
> Salford Graduate Reflective Group 2010 (Student 2)

> *… if they (supervisors) can't be bothered, I can't be bothered…*
>
> Salford Graduate Reflective Group 2010 (Student 3)

Supervision is a mechanism that should allow the learner time with a colleague (usually more experienced) to assist in the development of professional skills. The process can be seen as similar in nature to mentorship (Fowler and Chevannes, 1998) with the ideal characteristics of the mentor including feedback-giver, eye-opener, challenger and idea bouncer (Darling 1984). Fowler and Chevannes (1998) suggest that while there is a high degree of compatibility between clinical supervision and reflective practice, there may be some practitioners, especially those at a novice level, for whom the reflective element of the process may be less useful and even detrimental; their recommendation being that the supervision process needs to be tailored to the needs of the individual. Findings from research into clinical supervision within the physiotherapy profession 'highlighted the value that Physiotherapists placed on having formal 'time out' to reflect on their practice. However, clinical supervision was found to fulfil a variety of functions at different times, being tailored to meet individual needs.' (Clouder and Sellars 2004: 263).

Ideally, a clinical mentor should be someone with expertise of their own to bring to the discussion (e.g. senior professional colleague). However, in addition, it is often useful to have a neutral colleague/friend who can act as a confidential 'sounding board', especially for issues which would not be mentioned in front of a senior colleague (see issue of disclosure later).

EG Example

Scenario: Consider a patient you have recently treated for a musculoskeletal problem.

1. Briefly describe the problem and reflect on the process of diagnosis and treatment.
   A. What did you say and why?
   B. What skills did you employ and why?
   C. What treatment or intervention did you prescribe (if any) and why?
2. How did your handling of the patient impact health outcomes?
3. Were you happy with your handling of this case?
4. What would you do differently in future?

Reflection requires honesty from the reflector and it could be argued that it should be built on solid evidence of what has actually happened. However, this can cause dilemmas for the reflector, for example being able to speak about mistakes or processes that have not gone well. There may be circumstances in which it would be very risky to do this, for example disclosing a serious professional error in front of a senior colleague or educator (or during a piece of assessed coursework). You may not want to disclose some issues during a team de-brief session, for instance, if it might effect a future promotion opportunity. The notion of a critical friend as mentor should sit outside the judgemental/assessment process. The mentor has become recognised as being very important in the reflective

practice process. A critical friend is a trusted person who asks provocative questions and can:

> *observe, listen and learn;*
> *demonstrate positive regard for the learner;*
> *help identify issues and explore alternatives;*
> *offer sources of evidence/expertise;*
> *work collaboratively;*
> *encourage the learner to explore possibilities;*
> *offer a thoughtful critical perspective.*
>
> (Stroobants 2004)

In order to be able to disclose the 'truth' during reflection a safe environment needs to be provided in which that reflection can take place, i.e. there needs to be an established mentor-reflector relationship within which the reflector can trust the mentor that absolute confidentiality will be assured, whatever is disclosed. Trust in this context is all about having a mutual exchange (reciprocity) and developing a relationship where the trust increases incrementally over time: different types of trust relationships have previously been identified as being important in reflective practice.

> *Contractual Trust (trust of character), keeping agreements, honouring intentions;*
> *Communication Trust (trust of disclosure) telling the truth, admitting mistakes, give and receive constructive feedback;*
> *Competence Trust (trust of capability) how capable are participants of trust.*
>
> (Reina and Reina 2006)

It should be made clear from the start what the boundaries of the mentor relationship are; there may be issues of disclosure that would require disclosure whatever the relationship, for example criminal acts or patient cruelty/abuse. In order to become a skilled practitioner, the safe environment in which reflective practice takes place should be non-judgemental in order to allow/promote the disclosure of all issues in order for the reflector to be able to move forward and learn from practice, however novice they are.

This safe environment needs to be negotiated at the beginning of your reflective practice journey and finding a skilled mentor who has the skills to help you to reflect well will be crucial to success. An example of a 'confessional' recounted as part of an evaluation study of Health Trainers in Salford (Dugdill et al. 2009) serves to illustrate the importance of the 'safe' environment for de-briefing purposes, especially for novice professionals/practitioners. The following quote was taken from a reflective interview between a health trainer and the evaluator, and highlights how professionals at the chalk face may have to deal with some very difficult issues when dealing with the public.

> *It was scary. I had two referrals from people who said they wanted to kill themselves … I was quite emotional about it. There was a time when I was thinking this isn't the right job for me … I might do more harm than good.*
>
> (Dugdill et al. 2009: 125)

The health trainer went on to say that they had been uncertain what to do and how to de-brief following this incident. Ideally, reflection and de-briefing can become integral processes for professionals and are essential in situations, such as the above scenario, where a novice professional may need guidance and support immediately.

There are a range of different types of mentorship/supervision that can be useful to a reflector. One or more of these relationships can be established to support the reflector at any one time (Richmond 2009):

- line management – performance, competence, outcome driven;
- group supervision – interprofessional team-working situation;
- peer support – interprofessional team-working situation;
- individual supervision – supervisor leads;
- individual mentoring.

It is important to understand the power differentials that may exist between the reflector and each of these different types of supervision situation or mentorship relationship, as this will effect quality of reflective practice.

IMPORTANT PRINCIPLES OF REFLECTIVE PRACTICE

There are certain features to reflective practice that are integral to its success.

Closing the loop

This is about seeing reflection as a process not as a product. Reflective practice is about analysing your practice and also ensuring that you draw conclusions as to what you have learnt about a specific treatment, assessment tool and experience, but also about yourself as a physiotherapy practitioner.

Planning

Whatever the experience you have been through, be it a single assessment or a series of treatments with a patient, you are never going to have exactly the same experience again. As such, the reflection you complete should

Tip

Sometimes identifying an aspect of your practice that you would like to improve and using this as a focus for your reflection can help you be more specific when identifying outcomes. For example, if you feel your clinical reasoning during patient assessment is an area you would like to improve you can focus your thoughts towards this aspect rather than considering other aspects, such as communication.

Tip

I think I assumed that the content of my reflections would inherently bring about a change in my behaviour and facilitate my development as a [reflective practitioner]…

Final year reflective assignment (Salford student 2010)

If you are starting in a new clinical area review older, written reflections to remind yourself what you planned to do and incorporate it into your learning agreement/ contract/development plan or personal development plan.

result in a number of plans for how you will apply what you have learnt about yourself and physiotherapy in other situations. We have all been in situations where we have left a situation and thought 'I said I wasn't going to do that again!' or 'I must remember to do that in that way again'. In other words, we have made the same mistake/ delivered a less than ideal treatment twice (or more!) or done something that worked well (by accident!). The idea is that if you make plans you follow them in order to be mindful and continually developing practitioners. You cannot always ensure you can implement this because sometimes there may be insufficient time between experiences or an experience could be completely unexpected. In addition, the plans you make as a result of reflection might not be 'right'. After all, you can only base future plans on the current experiences so the conclusions you draw might not work as you expect. However, this reiterates the purpose of reflection, which is not about getting it 'right' every time but about developing new skills, attitudes, approaches and techniques honed by each experience. This is a process that will continue throughout your careers.

> *… you will always be a work in progress, you will never be the finished article…*
>
> Salford Graduate Reflective Group 2010 (Student 5)

> *… qualifying is like learning to drive, passing your test is only the beginning…*
>
> Salford Graduate Reflective Group 2010 (Student 5)

Do not worry if you fail to make meaningful plans but when you repeat either an unsuccessful practice or fail to repeat a successful approach revisit previous reflections and use them to help inform your ongoing plans.

Appropriate timing

In terms of reflecting on action (Schon 1983) there is not a specific timescale; however, it should be done ideally within a timeframe where you can remember the details of what happened and potentially how you felt, as this then allows you to draw more from it. That being said, benefit can also be seen from reflecting immediately after an event and then reconsidering it again a short time later with the added perspective of time.

EG Example

A patient falls while you are with them; this has never happened to you before. Your initial reflection will probably be very focussed on your emotions and on details you would want to change. Therefore, the likelihood is it will be negative. A few days later the reflection might be more balanced and you are able to see more of the positives/be more objective and therefore produce more realistic planning strategies.

Certain experiences will tend to make you reflect more than others and over longer periods of time. However, choosing to reflect on an experience weeks or even months after it has occurred dilutes its efficacy. It is probable you have already had further experiences that have had an influence and will feel very differently about it. You may have also perpetuated errors you made initially. As a result, reflections approached in this way tend to be 'sanitised' versions, lacking depth and relevance to the individual.

Others' perspectives

Certainly, some of the models of reflection (see later in chapter) ask you to consider the experiences from the perspectives of other people. If you are using a different

method (see later in chapter) you may still want to consider these individuals. In addition, you might want to consider your decisions/choices in the context of literature/protocols as another perspective.

Knowing yourself

In order to challenge yourself as you progress in practice you need to evaluate yourself now. This may involve using documentation such as SWOT (strengths–weaknesses–opportunities–threats) analyses, learning styles questionnaires, team role questionnaires or personality tests. It might also incorporate evaluation of your previous experience and background. Reviewing these types of aspects on a regular basis will enhance the depth of your reflective practice.

What to reflect on

Students or those new to utilising reflection as a developmental tool sometimes complain that 'nothing significant happened'. In reality, this often translates into 'nothing bad happened' or 'nothing different happened'. This may arise from the term 'Critical Incident Reports', which can be incorrectly interpreted as meaning a dramatic, unusual, significant and usually negative experience. While these experiences do happen and can be usefully harnessed, it is at least as useful to examine the more routine and mundane experiences that are more frequently encountered (Table 5.1). Close scrutiny of those activities which are taken for granted can lead to more meaningful and applicable insights into everyday practice (Donaghy and Morrs 2007).

Table 5.1 Ideas for reflective activity

Experience	Aspect	Evaluations of...
Interaction with a patient	Communication skills	• Interview technique • Motivation skills • Use of counselling techniques • Giving instruction • Reading the patient's non-verbal communication (NVC)
	Assessment skills	• Ability to plan subjective/objective • Clinical reasoning/diagnostic skill • Manual technique/skill development • Accuracy of application • Knowledge of assessment techniques • Time management
	Treatment skills	• Manual technique/skill development • Accuracy of application • Knowledge of treatment approaches
Interaction with the team	Clinical Educator	• Negotiating learning activity • Developing a learning relationship • Understanding expectations • Managing feedback on performance • Reviewing documentation
	Other students	• Managing disagreement or conflict • Negotiating roles/workload • Team working/leadership skills
	Liaison with other professionals	• Receiving information • Providing information • Differences in approach/role • Interaction in team meetings
Attending formal training	Delivery of content	• Self-assessment of understanding/level of learning • Application to own practice • Relevance/quality of the training • Challenge and questioning of 'received wisdom'
	Assessment	• Preparation for assessment tasks • Performance in assessment

As well as reflecting on different issues during practice, professionals may also reflect in different ways depending on the purpose of that reflective process, for example a reflective record might be different if it is for HCPC re-registration as opposed to learning about personal development following an incident with a patient. Choosing the best approach can include consideration of issues such as individual learning preferences, the clinical area or the nature of the experience; however, the purpose of the output is possibly the most significant factor: Is the reflection to demonstrate academic ability? Is it for personal catharsis? Is it for advancing understanding or developing reasoning, or to demonstrate achievement for promotion? Experimenting with different formats and models of reflection allows the practitioner to develop a 'tool box' from which they can select the most effective method for any given set of circumstances.

WAYS TO REFLECT

Verbal reflection

The power and potential of dialogical reflection to develop practice has been recognised (Clouder 2000). Verbal reflection, sometimes also referred to as dialogical reflection, is normally where two or more people engage in verbal discussion about a single experience, a patient, a pathology or a technique/approach. Commonly, this occurs as a 1:1 discussion between more and less experienced clinicians. For example, an educator and their student, tutor and student, or a band 7 and band 5 (see 'Reflecting with a supervisor' in the previous section). The key to its success is having the other person question you and facilitate you in drawing conclusions and making future plans. At a novice level this may involve question and answer sessions, feedback on techniques and more of an informal 'chat'. It may also incorporate the more experienced clinician sharing their expertise with you and even include practising techniques or role-play. As you progress as a clinician, these interactions may be more focussed on challenging your assumptions, examining the theory, considering alternative approaches, or comparing approaches and the reasons for them. The facilitator might find using models of reflection, sets of notes or lists of questions useful when they first start with this process to ensure a reflective cycle is followed.

In terms of producing evidence of dialogical reflection, any written format can be used (discussed later). It may be that the reflector makes notes during the discussion, but, likewise, it could be the facilitator. An alternative to a written record of dialogical reflection is to use an audio device such as a Dictaphone or electronic voice recorder when the discussion is going on. In a study to evaluate the use of a framework to facilitate reflection in physiotherapy students 'the majority of students felt that the structured taped interview was the most effective stage of the framework in stimulating reflective thought…several said that the real value of this step was the opportunity to play and replay the tape, listening and thinking about their own responses' (Donaghy and Morrs 2007: 90). Another option might be to videotape an actual intervention and use this as the basis for the verbal reflection. Again, this provides the opportunity to revisit the experience and analyse it further.

Graphical (mindmaps, spider diagrams, concept maps)

Many people have a preference for learning from visual stimuli; using a more diagrammatic method of documenting thought processes can be a useful activity. This less formal document can be particularly useful for complicated experiences where there is a need to consider multiple possibilities or for situations where it is difficult to identify where something went right or wrong.

Using a diagram can be a quick way of getting ideas down on paper and, as such, is well suited as a starting point for shared (verbal) reflection. For students or physiotherapists who find logic, sequencing and short-term memory challenging, these can be useful tools, allowing all ideas and possibilities to be scribbled down and then significant features, links and patterns to be identified more thoughtfully. Explaining your diagram to another person can also be a helpful starting point for a reflective discussion with a mentor.

Reflective diary

As mentioned earlier on in the chapter, reflective diaries can be used. A reflective log can be for self-analysis only. Sometimes keeping an audio diary (speaking memo notes into a digital voice recorder, for example) can be a quick and useful tool for self-reflection. This audio diary can be played back in private. It is good practice to ensure you debrief with a mentor, however, so do not rely on these self-reflective processes, as one of the crucial elements of reflection is challenging current ideology and practice. Therefore you need to expose your ideas to external questioning either in a formal (senior colleague) or informal (mentor) setting.

Prose/free writing

This builds on the idea of a diary but tends to be based on a single incident where the individual wants to explore it in a greater depth. It simply involves writing down your 'internal dialogue' (what you are thinking). There is a risk of this evolving into a story rather than being reflective in

nature; however, if you are clear about what you want to gain from the process it can be very cathartic, particularly if you have encountered a catastrophic event, and can help you to think more objectively about the details of what occurred.

Example

A patient who was wrongly diagnosed as not having a hip fracture that has subsequently been mobilised over a weekend and you review on the Monday. The reflection might want to consider the other people involved perspectives and your role within the multi-disciplinary team.

Using models of reflection

I think about reflective practice in the same way that I think about swimming; I feel that if my technique were better, if I could manage to get my breathing right and my leg moving the right direction, I would find it easier, put less effort in and would enjoy being able to move through the water sleekly and efficiently. In reality I have realised the only way to achieve this is with lots of practice, a good coach and a great deal of splashing…

Extract from a student diary

Many models and frameworks for reflective practice have been developed to assist practitioners (especially novice ones) in understanding the concepts and processes involved. It can be argued that the slavish use of models is stifling and results in limiting the scope of free and inventive thinking (Johns 2005), which is why this element of the chapter has been left to the end. Many practitioners do find these models, and the prompts to thinking that they provide, helpful in developing a reflective thought process. Ideally, experimentation and practice will lead to an effective individual approach to reflective practice.

Kolb

One of the most often cited models is that of Kolb's learning cycle (Kolb 1984). He argues for a relationship between thinking and experience, and views learning from experience as a cycle involving action and reflection, theory and practice. As a practitioner you can use this cycle to:

- reflect on an experience from practice, drawing on previous, linked experiences;
- consider how the experience is informed and supported by earlier and current practice theories and evidence-based practice from the literature (gained at university, at study days, from reading research);
- consider what conclusions you draw from your reflections and how they link with these other theories;
- consider how the conclusions you have drawn should be used to affect future practice and plan how you will introduce these into practice;
- start the cycle again by undertaking a further review of similar experiences incorporating the changes at a later stage.

The learning process is continuous in a systematic cycle of action, analysis and review (Figure 5.4).

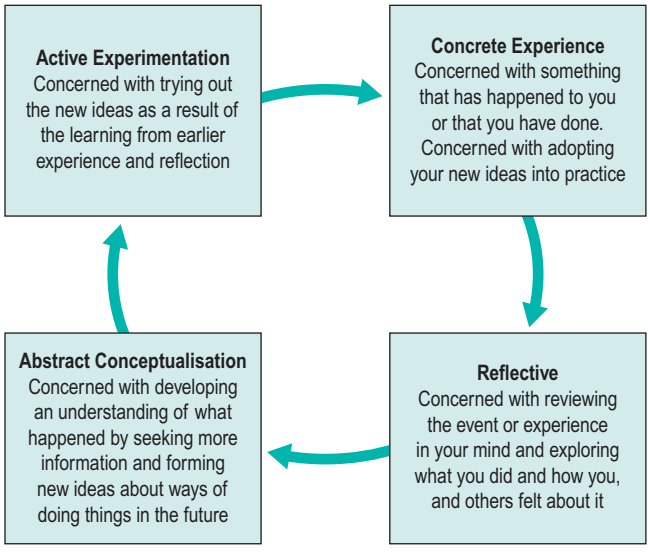


Figure 5.4 Kolb's learning cycle (Kolb 1984).

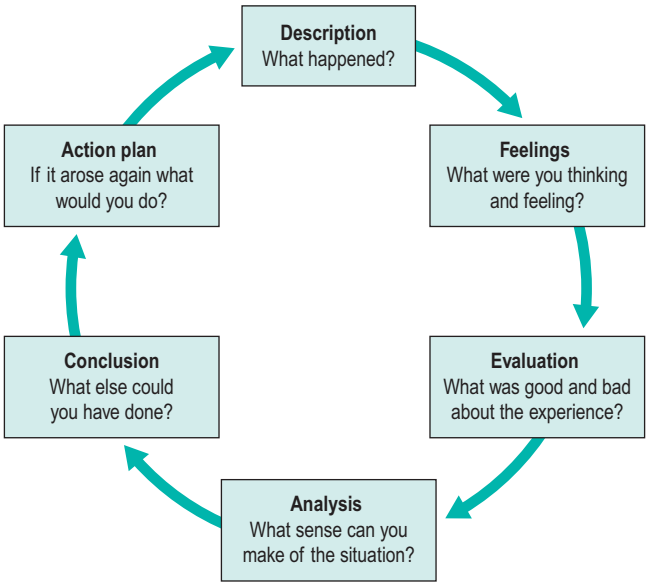


Figure 5.5 Gibb's model of reflection (Gibbs 1988).

Gibbs

Gibbs' model (Figure 5.5) provides a fairly simple cycle, starting with a description of an event or experience, working through stages of evaluation and analysis, and concluding with planning for future action.

Johns

The model of reflection developed by Christopher Johns (1994; Box 5.1) provides a set of questions or cues which help the practitioner work through a reflective process to consider the issues from a variety of perspectives, and to try to understand and learn from the experience.

Rolfe

Rolfe et al. (2001) propose a framework that uses Borton's developmental model. The questions 'What?', 'So what?' and 'Now what?' can stimulate reflection from novice to advanced levels (Figure 5.6; Box 5.2).

Your choice of reflective method(s) depends entirely on you as an individual and the experience(s) you want to reflect on. In the Salford Graduate Reflective Group, student 5 said 'I would prefer just a prosey style', student 2 wanted a few prompts, whereas student 1 felt that 'more questions to help me'. What is important is that you explore different methods in order to determine which you find the most useful.

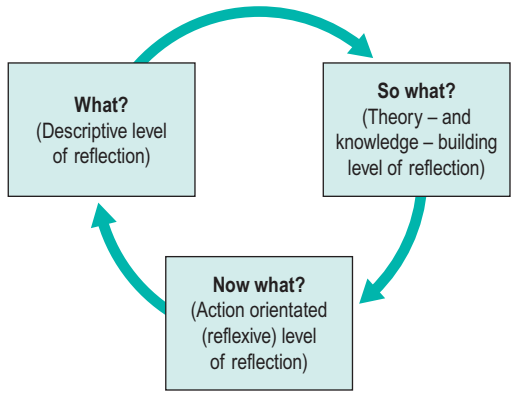


Figure 5.6 Rolfe's model.

CONCLUSION

In summary, this chapter has outlined the rationale and important principles of reflective practice and has given ideas as to how to engage with reflective practice successfully. It is an essential professional skill for all physiotherapists.

Box 5.1 **Johns' (1994) Model of Reflection**

Description

Write a description of the experience

What are the key issues within this description that I need to pay attention to?

Reflection

What was I trying to achieve?

Why did I act as I did?

What are the consequences of my actions

- for the patient and family?
- for myself?
- for people I work with?

How did I feel about this experience when it was happening?

How did the patient feel about it?

How do I know how the patient felt about it?

Influencing factors

What internal factors influenced my decision-making and actions?

What external factors influenced my decision-making and actions?

What sources of knowledge did or should have influenced my decision-making and actions?

Alternative strategies

Could I have dealt with the situation better?

What other choices did I have?

What would be the consequences of these other choices?

Learning

How can I make sense of this experience in light of past experience and future practice?

How do I **now** feel about this experience?

Have I taken effective action to support myself and others as a result of this experience?

How has this experience changed my way of knowing in practice?

(Johns 1994)

Box 5.2 **Rolfe et al. (2001) Model of Reflection**

What …

… is the problem/difficulty/ reason for being stuck/reason for feeling bad/reason we don't get on/etc., etc.?

… was my role in the situation?

… was I trying to achieve?

… actions did I take?

… was the response of others?

… were the consequences

- for the patient?
- for myself?
- for others?

… feelings did it evoke

- in the patient?
- in myself?
- in others?

… was good/bad about the experience?

So what …

…does this tell me/teach me/imply/mean about me/my patient/others/our relationship/my patient's care/the model of care I am using/my attitudes/my patient's attitudes/etc., etc.?

… was going through my mind as I acted?

… did I base my actions on?

… other knowledge can I bring to the situation?

- experiential
- personal
- scientific

… could/should I have done to make it better?

… is my new understanding of the situation?

… broader issues arise from the situation?

Now what …

… do I need to do in order to make things better/stop being stuck/improve my patient's care/resolve the situation/feel better/get on better/etc., etc.?

… broader issues need to be considered if this action is to be successful?

… might be the consequences of this action?

ACKNOWLEDGEMENTS

The editor would like to thank University of Salford graduates and students for their invaluable reflections. Without these we would cease to evolve as a profession.

REFERENCES

AHP (Allied Health Professions) Projec, 2003. Demonstrating Competence Through continuing professional development [CPD]: Final report; http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/dh_4071462.pdf, accessed October 2012.

Bolton, G., 2005. Reflective practice: Writing and professional development, second ed. Sage Publications, London.

Boshuizen, H.P.A., Schmidt, H.G., 2008. The development of clinical reasoning expertise. In: Higgs, J., Jones, M.A., Loftus, S., et al. (Eds.), Clinical reasoning in the health professions, third ed. Butterworth Heinemann, Amsterdam, pp. 113–121.

Boud, D., Keogh, R., Walker, D., 1985. Reflection: Turning experience into learning. Kogan Page, London.

Clouder, L., 2000. Reflective practice: Realising its potential. J Physiotherapy, 86 (10), 517–522.

Clouder, L., Sellars, J., 2004. Reflective practice and clinical supervision: an interprofessional perspective. J Adv Nurs 46 (3), 262–269.

CSP (Chartered Society of Physiotherapy), 2005. Workplace learning; Evidencing through reflection and evaluation. Information paper CPD31. P6 CSP, London.

Darling, L., 1984. What do nurses want in a mentor? J Nurs Admin 14 (10), 42–44.

Donaghy, M., Morrs, K., 2007. An evaluation of a framework for facilitating and assessing physiotherapy students' reflection on practice. Physiother Theor Pract 23 (2), 83–94.

Dugdill, L., 2009. Evaluating professional practice through reflection: Professionalism in the workplace. In: Heaney, C., Oakley, B., Rea, S. (Eds.), Exploring sport and fitness: Work-based practice. Routledge; Open University, Abingdon, Oxon, pp. 48–56.

Dugdill, L., Coffey, M., Coufopoulos, A., et al., 2009. Developing new community health roles: Can reflective learning drive professional practice? Int J Reflect Pract 10 (1), 125–134.

Fowler, J., Chevannes, M., 1998. Evaluating the efficacy of reflective practice within the context of clinical supervision. J Adv Nurs 27, 379–382.

Ghaye, T., 2005. Developing the reflective healthcare team. Blackwell Publishing Ltd, Oxford.

Gibbs, G., 1988. Learning by doing: A guide to teaching and learning methods. Oxford Polytechnic, Oxford.

Hargreaves, J., 2004. So how do you feel about that? Assessing reflective practice. Nurs Educ Today 24 (3), 196–201.

HCPC, 2006. Standards of Continuous Professional Development; http://www.hpc-uk.org/registrants/cpd/, accessed October 2012.

Higgs, J., Jones, M.A., 2008. Clinical decision making and multiple problem spaces. In: Higgs, J., Jones, M.A., Loftus, S., et al. (Eds.), Clinical reasoning in the health professions, third ed. Butterworth Heinemann, Amsterdam, pp. 3–17.

Higgs, J., Jones, M.A., Loftus, S., et al. (Eds.), 2008. Clinical reasoning in the health professions, third ed. Butterworth Heinemann, Amsterdam.

Intercollegiate Stroke Working Party, 2008. National clinical guideline for stroke, third ed. Royal College of Physicians, London.

Jensen, G., Resnik, L., Haddad, A., 2008. Expertise and clinical reasoning. In: Higgs, J., Jones, M.A., Loftus, S., et al. (Eds.), Clinical reasoning in the health professions, third ed. Butterworth Heinemann, Amsterdam, pp. 123–135.

Johns, C., 1994. Becoming a reflective practitioner, second ed. Blackwell, Oxford.

Johns, C., 2005. Balancing the winds. Reflect Pract 6 (1), 67–84.

Jones, M.A., Jensen, G., Edwards, I., 2008. Clinical reasoning in physiotherapy. In: Higgs, J., Jones, M.A., Loftus, S., et al. (Eds.), Clinical reasoning in the health professions, third ed. Butterworth Heinemann, Amsterdam, pp. 245–256.

Knowles, Z., Telfer, H., 2009. The where, what and why of reflective practice. In: Heaney, C., Oakley, B., Rea, S. (Eds.), Exploring sport and fitness: work-based practice. Routledge; Open University, Abingdon, Oxon, pp. 22–35.

Kolb, D.A., 1984. Experiential learning: Experience as the source of learning and development. Prentice Hall, NJ.

Lindsay, G., Kell, L., Ouellette, J., et al., 2010. Using 'I' in scholarly writing: how does reflecting on experience matter? Reflect Pract 11 (3), 271–283.

Moon, J.A., 1999. Reflection in learning and professional development: Theory and practice. Kogan Page Limited, London.

Reina, D., Reina, M., 2006. Trust and betrayal in the workplace: Building effective relationships in your organisation. Berrett-Koehler, San Francisco.

Richmond, D., 2009. Using multi-layered supervision methods to develop creative practice. Reflect Pract 10 (4), 543–557.

Rolfe, G., Freshwater, D., Jasper, M., 2001. Critical reflection for nursing and the helping professions: A user's guide. Palgrave Macmillan, Basingstoke, Hampshire.

Schon, D.A., 1983. The reflective practitioner: How professionals think in action. Basic Books Inc, USA.

Stroobants, H., 2004. Critical friends for comfort and growth. Paper presented at the European Teacher Education Network Conference, February 2004, Viana do Castelo.

Wilson, J.P., 2008. Reflecting-on-the-future: A chronological consideration of reflective practice. Reflec Pract 9 (2), 177–184.

Zwarenstein, M., Reeves, S., 2006. Knowledge translation and interprofessional collaboration: Where the rubber of evidence-based care hits the road of teamwork. J Cont Educ Health Prof 26, 46–54.

Chapter 6

Management of respiratory diseases

Stephanie Enright and Fiona M. Schreuder

Note

Other relevant information such as the active cycle of breathing technique (ACBT) and basic respiratory anatomy and physiology of the lungs can be found in Chapter 9.

INTRODUCTION

Diseases of the respiratory system are a major cause of illness worldwide and are increasingly important as a cause of mortality and morbidity (WHO 2004). In the UK they are the most common reason for consulting a general practitioner (GP) (Yelin et al. 2006) and a recent report on the 'Burden of Lung Disease' suggests that respiratory disease is second only to cardiovascular disease as a major cause of morbidity and mortality in the UK (BTS 2006a).

Respiratory diseases encompass a range of diseases which affect the airways and other structures of the lungs, and although they are characterised by respiratory symptoms, it has become increasingly evident that chronic pulmonary diseases are associated with a range of non-pulmonary effects (Sinden and Stockley 2010; Decramer et al. 2012). In terms of the respiratory manifestations of respiratory disease, however, they can be divided broadly into obstructive and restrictive types – although most patients have elements of both.

1. *Obstructive diseases* include conditions in which there is a resistance to airflow, either through reversible factors such as bronchospasm or inflammation, or through irreversible factors such as airway fibrosis or loss of elastic recoil owing to damage to the airways and the alveoli.
2. *Restrictive disorders* are characterised by reduced lung compliance leading to the loss of lung volume, which may be caused by disease affecting the lungs, pleura, chest wall or neuromuscular mechanisms. These diseases are therefore different from the obstructive diseases in their pure form, although mixed restrictive and obstructive conditions can occur.

Obstructive diseases are by far the most common and have major implications for patients and the economic burden on healthcare, as they require many years of medical intervention (Johnson et al. 2007). Therefore, their pathophysiology and treatment will be discussed initially in some detail. They are:

- chronic obstructive pulmonary disease (COPD);
- chronic bronchitis;
- emphysema;
- asthma;
- bronchiectasis;
- cystic fibrosis.

As the changing pattern of respiratory disease has resulted in opportunistic pneumonias, which are a common presentation in patients with AIDS, the restrictive disorders and their management will then be considered. They are:

- pneumonia;
- pleurisy;
- pleural effusion;
- pneumothorax;
- acute respiratory distress syndrome (ARDS);
- fibrosing alveolitis.

Finally, there are other lung disorders that fit into neither of the first two categories but need to be included because of their prevalence within the community or hospital environment. They are:

- lung abscess;
- pulmonary tuberculosis;
- bronchial and lung tumours;
- respiratory failure (including respiratory failure secondary to neuromuscular disease).

CHRONIC OBSTRUCTIVE PULMONARY DISEASE: BASIC ISSUES

Chronic obstructive pulmonary disease (COPD) is an ill-defined term that is often applied to patients who have a combination of chronic bronchitis and emphysema, which frequently occur together (and may also include asthma). In the majority of cases, chronic bronchitis is the major cause of obstruction, but in some cases emphysema is predominant. There are many patients who report shortness of breath, increasing in severity over several years and, on examination, are found to have a chronic cough, an overinflated chest and poor exercise tolerance. It is often difficult to assess clinically to what extent these patients have chronic bronchitis or emphysema, or a mixture of these. Patients may also present with a more episodic form of disease, which is a characteristic of an asthmatic component. Therefore, 'chronic obstructive pulmonary disease' is a convenient term, which encompasses one or all of these components. Although COPD is an umbrella term for a combination of these clinical entities, both the aetiology and pathological features of chronic bronchitis and emphysema will be considered initially before a discussion of the clinical features and management of COPD is discussed in more detail.

Key point

In patients with COPD, chronic bronchitis, emphysema and asthma often coexist; the disease is progressive and characterised by acute exacerbations. It is not usually diagnosed until irreversible damage has occurred. As well as the respiratory component of COPD, it is also associated with a range of coexisting extrapulmonary effects.

Although historically COPD was considered as a disease of the lungs characterised by irreversible airflow obstruction, there is now increasing evidence that it presents with other associated comorbidities such as skeletal muscle dysfunction, cardiovascular disease, osteoporosis and diabetes (Sinden and Stockley 2010; Decramer et al. 2012). Therefore, because there is increasing recognition of the presence and impact of non-pulmonary manifestations, this has led to the now established international definition from the Global Initiative for Obstructive Lung Disease (GOLD).

Key point

Chronic obstructive pulmonary disease (COPD) is a preventable and treatable disease with some significant extrapulmonary effects that may contribute to the severity in individual patients. Its pulmonary component is characterised by airflow limitation that is not fully reversible. The airflow limitation is usually progressive and associated with an abnormal inflammatory response of the lung to noxious particles or gases. As a consequence of airflow obstruction, dyspnoea and other co-morbidities, patients with COPD have impaired exercise tolerance and reduced activity, which contribute to a reduced quality of life.

(GOLD 2011)

It is difficult to determine the exact prevalence of COPD in the community as the diagnosis of COPD is often only made following a first admission to hospital with an acute exacerbation of respiratory symptoms (Bastin et al. 2010). However, figures from general practice suggest that 5% of men and 2% of women will be diagnosed as suffering from COPD, although in the population as a whole it is estimated that 11% of men and 8% of women have evidence of obstructed airways when specifically tested by spirometry (BTS 2006a).

Cigarette smoke is the main risk factor for COPD and is associated with 80–90% of cases (Ryter et al. 2010). However, not all smokers develop COPD and it is estimated that 15–20% of smokers will develop the pathology (Soares et al. 2010) with some evidence of women being more susceptible to the effects of cigarette smoke than men (Sørheim et al. 2010). However, it is also suggested that 10–20% of COPD cases are a result of occupational exposure to noxious particles, such as the combustion of solid fuels (Kurmi et al. 2010), and long-term exposure to traffic-related air pollution has been implicated in the development of COPD (Andersen et al. 2010).

The survival rate for COPD varies between 5 and 30 years, but eventually cardiac and ventilatory failure will occur. Avoidance of the precipitating factors listed below and early detection of the disease will tend to improve the prognosis:

- stopping smoking and reducing exposure to atmospheric pollution;
- maintaining physical fitness through participation in regular exercise;

- maintenance of good general health, including good nutrition;
- prompt treatment of all acute infections.

As COPD is characterised by airways obstruction, the greater the obstruction, the lower the chance of survival (Pearson and Calverley 1995). Obstruction to flow is measured by *forced expiratory volume in one second* (FEV_1) and together with age, FEV_1 is the most important determinant of survival. Therefore, COPD can be classified according to the GOLD (2011) definition (Table 6.1).

Patients with COPD may also be classified according to the frequency of chest exacerbations each year (NICE 2010) or their level of breathlessness, as assessed by the Medical Research Council (MRC) dyspnoea scale (Table 6.2).

Table 6.1 Spirometric classification of patients with COPD according to GOLD (2011)

Stage	Severity	FEV_1:FVC	FEV_1 % predicted
I	Mild COPD	<0.7	<80
II	Moderate COPD	<0.7	50–80
III	Severe COPD	<0.7	30–50
IV	Very severe COPD	<0.7	<30

FVC = forced vital capacity; FEV_1 = forced expired volume in one second.

Table 6.2 The Medical Research Council Dyspnoea Scale

Grade	Degree of breathlessness related to activities
1	Not troubled by breathlessness except on strenuous exercise
2	Short of breath when hurrying on walking up a slight hill
3	Walks slower than contemporaries on the level because of breathlessness, or has to stop for breath when walking at own pace
4	Stops for breath after walking about 100 m or after a few minutes on the level
5	Too breathless to leave the house or breathless when dressing or undressing

Although chronic bronchitis and emphysema coexist in patients with COPD they can occasionally appear in isolation. Therefore, a brief description of these pathologies will now be presented.

CHRONIC BRONCHITIS

ABC Definition

Chronic bronchitis is a chronic or recurrent increase in the volume of mucus secretion sufficient to cause expectoration when this is not caused by localised bronchopulmonary disease. In the definition of this disease, chronic/recurrent is further defined as a daily cough with sputum for at least three months of the year for at least two consecutive years and airways obstruction that does not change markedly over periods of several months (West 2008). Chronic bronchitis is a clinical diagnosis (unlike the definition of emphysema).

Aetiology of chronic bronchitis

This is more common in middle-to-late adult life and in men more than women (Clarke 1991) although recent data suggest that mortality is now rising faster in women (Dransfield et al. 2006).

Cigarette smoking is the chief culprit and, although in the UK over 20% of the adult population continue to smoke (DH 1997), only 15–20% of smokers may develop chronic bronchitis. The reason for this is probably genetic (Silverman et al. 2000) although the number of cigarettes smoked does have an effect on the progression of the disease.

In the larger, more proximal airways, exposure to noxious particles stimulates mucus hypersecretion, with an increase in the size and number of goblet cells and submucosal glands. This results in a chronic cough with sputum production and is often termed chronic bronchitis (Jeffery 2000). There is also a decrease in the number and length of the cilia, all of which lead to retention of mucus, repeated infections, obstruction of airways and inflammation. In turn, this causes a release of toxins that further damage airway structure and function (Braman 2006).

Atmospheric pollution (e.g. industrial smoke, smog and coal dust) will also predispose to the development of the disease, which is therefore more common in urban than in rural areas. It is more prevalent in socioeconomic groups 4 and 5 (Yelin et al. 2006) and is costly in terms of working days lost annually in the UK.

Exposure to risk: Pack-years

Rather than simply recording a patient's current smoking habits, a much better indicator of any potential deterioration in lung function is an assessment of pack-years, which is the number of packs (20 per pack) smoked daily multiplied by the number of years of smoking. For example, someone aged 60 years who has smoked five cigarettes per day (0.25 of a pack) since the age of 15 years has a lifetime exposure equal to 0.25 × 45 = 11 pack-years. Another person of the same age who smoked 30 cigarettes per day (1.5 packs) between the ages of 15 and 25 years (gave up until aged 40 years, since then has smoked one pack per day) has a lifetime exposure of (1.5 × 10) + (1 × 20) = 35 pack-years.

EMPHYSEMA

The prevalence of the condition is probably highest in England when compared with the rest of Europe, especially in the major centres of industry – although there is often a family history of the disease. It is more common in males (Corda et al. 2006).

ABC Definition

Emphysema is a condition of the lung characterised by permanent dilatation of the air spaces distal to the terminal bronchioles with destruction of the walls of these airways. It is nearly always associated with chronic bronchitis from which it is difficult to distinguish during life (West 2008).

Pathology of chronic bronchitis

The hallmark is hypertrophy, and an increase in number of mucous glands in the large bronchi and evidence of inflammatory changes in the small airways (Thurlbeck 1976). Some irritative substance stimulates over-activity of the mucus-secreting glands and the goblet cells in the bronchi and in the bronchioles, which causes secretion of excess mucus. This mucus coats the walls of the airways and tends to clog the bronchioles, which is functionally more important (West 2008). The cells increase in size and their ducts become dilated and may occupy as much as two-thirds of the wall thickness (West 2008). The airways become narrowed and show inflammatory changes, which results in mucosal oedema, thus further decreasing the diameter of the airways. The ciliary action is also inhibited.

Airflow limitation in chronic bronchitis is more closely related to the dimensions of the distal (small) airways than proximal (large) airways (Hasegawa et al. 2006) and this narrowing of the lumen of the airways is further emphasised during expiration by the normal shortening and narrowing of the airways. Consequently, the airways obstruction is enhanced during expiration, with resulting trapping of air in the alveoli. The lungs gradually lose their elasticity as the disease progresses. They will gradually become distended permanently, which may eventually cause extensive rupture of the alveolar walls. After repeated exacerbations caused by infection there is widespread damage to the bronchioles and the alveoli with fibrosis and kinking occurring, as well as compensatory over-distension of the surviving alveoli. This is closely linked to and contributory to the development of emphysema (Hogg 2004).

Causes and types of emphysema

Causes and predisposing factors

Congenital or primary emphysema may be caused by alpha$_1$-antitrypsin deficiency. This is a rare inherited condition that affects 1 person in 4000 and results in the complete absence of one of the key anti-protease systems in the lung (Corda et al. 2006). The consequence is the early development of COPD, especially if the patient is already a smoker (Senn et al. 2005). Although alpha$_1$-antitrypsin deficiency is responsible for less than 1% of cases of emphysema, its hereditary nature means that it is worth diagnosing. It should, therefore, be considered in any young COPD patient.

Emphysema may be secondary to other factors, such as:

- obstructive airways disease, e.g. asthma, cystic fibrosis, chronic bronchitis;
- occupational lung diseases, e.g. pneumoconiosis;
- compensatory to contraction of one section of the lung, e.g. fibrous collapse or removal when the remaining lung expands to fill the space.

Types of emphysema

ABC Definition

Centrilobular (centri-acinar) emphysema tends to affect the respiratory bronchioles with most of the alveoli remaining normal. *Panlobular* (panacinar) emphysema results in widespread destruction of most alveoli, as well as respiratory bronchioles.

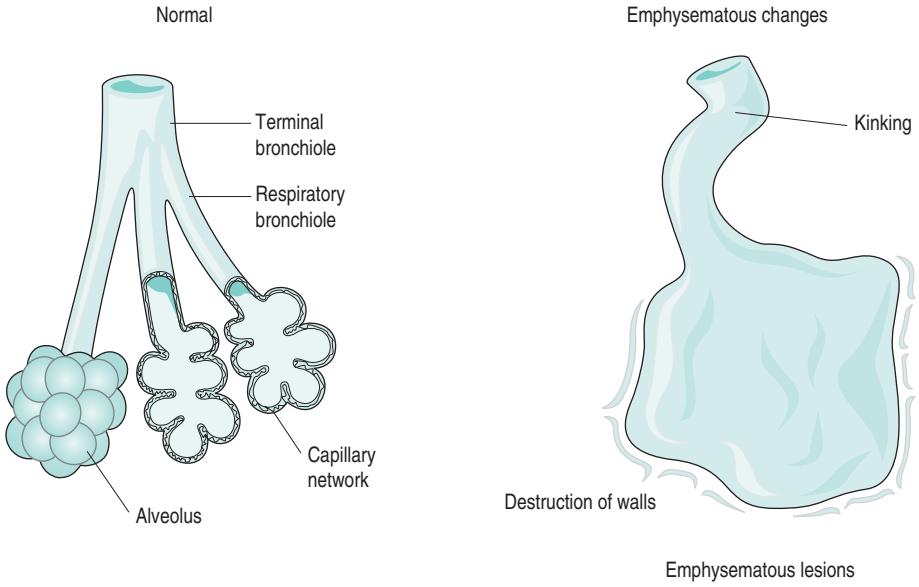


Figure 6.1 Emphysematous changes in the lung.

Emphysema is usually of the panacinar (panlobular) type. In centrilobular emphysema the upper zones of the lung are usually affected. This causes gross disturbance of the ventilation/perfusion relationship as there is a relatively well preserved blood supply to the alveoli but the amount of oxygen reaching the capillary is decreased owing to the damage to airways proximal to the alveoli. Panacinar emphysema predominantly affects the lower lobes; lower lobe involvement is more common in individuals with alpha$_1$-antitrypsin deficiency (Stavngaard et al. 2006). This has a less drastic effect on the ventilation/perfusion relationship as the blood supply in the damaged areas is decreased in proportion to the decreased ventilation in those areas.

Pathology of emphysema

Smoking causes the clustering of pulmonary alveolar macrophages (which are the major defence cells of the respiratory tract) around the terminal bronchioles. These macrophages are abnormal in smokers and release proteolytic enzymes that destroy lung tissue locally. Polymorphonuclear leucocytes, necessary to combat infection in the lung, release an enzyme that also destroys lung tissue. The defence mechanism against the unwanted action of these enzymes lies in the serum alpha$_1$-antitrypsin, which is normally present in the airway lining fluids. Oxidants released by both cigarette smoke and the leucocytes tend to inactivate the anti-proteolytic action of the alpha$_1$-antitrypsin, thus causing destruction of lung tissue as seen in centrilobular emphysema (Stavngaard et al. 2006).

Subsequently, the walls of the airways become weak and inelastic owing to the damage caused by repeated infections. They tend to act as a one-way valve as the walls collapse on expiration. This causes air trapping and consequent increase in the intra-alveolar pressure during expiration. The alveolar septa break down and form bullae (Figure 6.1).

During expiration the pressure from the trapped air in the bullae may compress adjacent healthy tissue, thus causing occlusion and trapping of air in that tissue. The capillaries around the alveolar walls become stretched, causing the lumen to decrease and atrophy to occur. This causes an alteration in the ventilation/perfusion relationship owing to the loss of surface area for gaseous exchange and the decrease in blood supply resulting from damage to the pulmonary capillary network.

CLINICAL FEATURES OF COPD

The most important clinical features arising in the respiratory system are cough, sputum, wheeze and dyspnoea. The respiratory features will be considered initially, prior to a brief description of the extrapulmonary manifestations associated with COPD. Although the clinical features arising in the respiratory system may alter depending on the extent to which a patient has predominantly chronic bronchitis or emphysema, the clinical features of COPD will be broadly discussed. A comparison of the clinical features of chronic bronchitis and emphysema will also be

discussed should one of these clinical entities predominate in the COPD patient.

Dyspnoea or shortness of breath

This occurs in patients with COPD and, together with the energy-requiring consequences of chronic infection and inflammation, leads to increased work of breathing (Donahoe et al. 1989). The patient becomes progressively more short of breath as the disease progresses. Shortness of breath occurs initially on exertion, but as the disease progresses it will gradually occur after less and less activity and finally at rest. This disabling breathlessness is what prevents the patient from working and gradually transforms the patient's state into one of severe exercise intolerance and disability (Folgering and von Herwaarden 1994). In a patient where an emphysematous component predominates, shortness of breath may progress more rapidly during the progression of the disease.

In the patient where emphysema predominates they may present with a 'fish-like' inspiratory gasp, which is followed by prolonged, forced expiration usually against 'pursed lips'. The latter creates back-pressure which tends to prevent airways shutdown during expiration. Owing to increased intrathoracic pressure the jugular veins fill on expiration. A 'flick' or 'bounce' of the abdominal muscles may be seen on expiration as the outward flow of air is suddenly checked by the obstruction of the airways (Figure 6.2).

Cough

The patient will complain of a cough for many years, initially intermittent and gradually becoming continuous. Fog, damp or infection increases it. The patient may also complain of bouts of coughing occasionally on lying down or in the morning. The cough and sputum production are not associated with either mortality or disability, and are reversible in most smokers once they stop smoking. The cough is caused by either irritation of airway nerve receptors because of the release of compounds from inflammatory cells or from the presence of increased mucus production.

Sputum

Some patients with COPD may be unproductive of sputum. However, sputum production may be a common feature (where chronic bronchitis predominates). This is mucoid and tenacious, usually becoming mucopurulent during an infective exacerbation.

Wheeze

Wheezing is a symptom described by as many as 80% of patients with COPD. Wheezing is a characteristic feature of obstructive lung disease, although it is also reported in many other acute and chronic respiratory diseases. Wheezing is caused by the sound generated by turbulent airflow through the narrowed conducting airways and may be worse in the mornings or may be related to weather changes. If the wheeze is reversible following inhaled medication this may be indicative of an asthmatic component in the COPD patient.

Deformity

These patients often develop a barrel chest caused by hyperinflation and use of accessory muscles of respiration. The thoracic movements are gradually diminished and paradoxical in-drawing of the lower intercostal spaces and supraclavicular fossa may occur on inspiration. This is associated with the difficulty of ventilating stiff lungs through narrowed airways. The ribs become elevated by the use of the accessory muscles of respiration, which may become hypertrophied as a result and there is loss of thoracic mobility. There may be a thoracic kyphosis plus elevated and protracted shoulder girdles.

Cyanosis

This is a blue colouration of the skin caused by the presence of desaturated haemoglobin as a result of reduced gaseous exchange. Cyanosis is also related to the development of complications, such as poor cardiac output caused by ventricular failure leading to increased peripheral oxygen extraction. Cyanosis may also be caused by an increase in red blood cells (polycythaemia) in response to chronic hypoxaemia.

Cor pulmonale

This may occur in the later stages of COPD. The impaired gas exchange in COPD caused by the disruption of ventilation and perfusion and the resulting hypoxia leads to widespread hypoxic pulmonary vasoconstriction. This leads to an increase in pulmonary vascular resistance resulting in pulmonary hypertension (Vender 1994). The increase in the pressure within the pulmonary artery will create a resistance, which the right ventricle must overcome. This eventually leads to hypertrophy and dilatation, a condition known as 'cor pulmonale'.

Right heart failure leads to an increased pressure in the peripheral tissues resulting in the development of peripheral oedema. The combination of renal hypoxia and the increase in blood viscosity from polycythaemia increases the systemic blood pressure and eventually leads to left heart failure. The development of pulmonary oedema, which exacerbates the hypoxia and low cardiac output in patients with COPD, leads to a terminal stage of the disease. The mechanism of this cycle is illustrated in Figure 6.3.

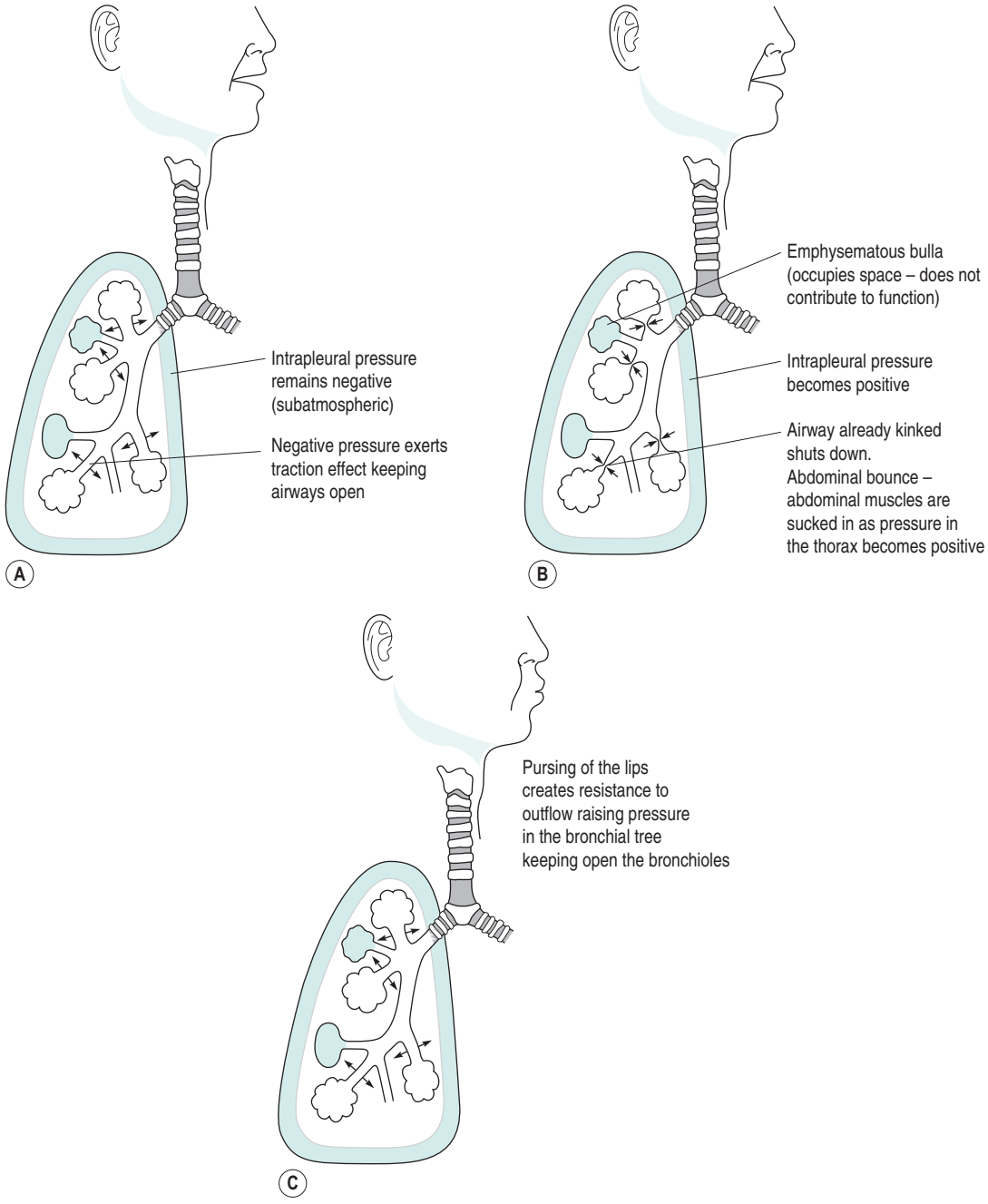


Figure 6.2 End of expiration: (a) normal; (b) in emphysema; (c) pursed-lip breathing.

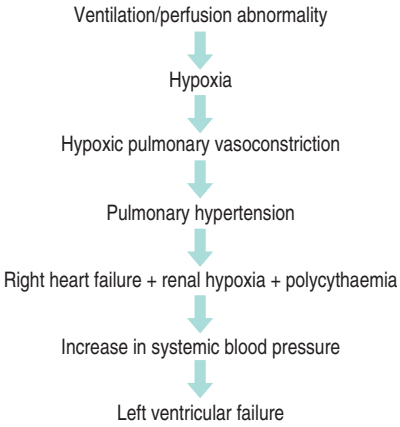


Figure 6.3 Mechanism of development of cor pulmonale and congestive heart failure in COPD.

Lung function

There is reduction of FEV_1 and the forced vital capacity (FVC) is grossly reduced. The residual volume (RV) will be increased at the expense of the vital capacity (VC) because of air trapping and the inability of the expiratory muscles to decrease the volume of the thoracic cavity. The expiratory flow–volume curve is grossly abnormal in severe disease; after a brief interval of moderately high flow, flow is strikingly reduced as the airways collapse and flow limitation by dynamic compression occurs. A scooped-out appearance is often seen (Decramer 1989).

Blood gases

Ventilation/perfusion mismatch is inevitable in COPD and leads to a low arterial oxygen pressure (PaO_2) with or without retention of carbon dioxide (CO_2). As the disease becomes severe, the arterial carbon dioxide pressure ($PaCO_2$) may rise, and there is some evidence that the sensitivity of the respiratory centre to CO_2 is reduced (Fleetham et al. 1980), which may leave the respiratory stimulus dependent upon the hypoxic drive. However, more recent evidence suggests that the administration of high levels of oxygen (>70%) in patients with COPD may increase hypercapnia owing to the reversal of pre-existing regional pulmonary hypoxic vasoconstriction, resulting in greater dead space (Crossley et al. 1997).

Auscultation signs

There will be inspiratory and expiratory wheeze possibly with added coarse crepitations if sputum retention is present. The breath sounds are vesicular with prolonged expiration.

Examination

The percussion note will be normal or hyper-resonant because of air trapping.

X-ray signs

No characteristic abnormality is seen in the early stages of the disease. If there is significant airways obstruction there may be signs of chest over-expansion (flattening of the diaphragm) and an enlarged retrosternal airspace.

Other non-respiratory manifestations of COPD

Loss of skeletal muscle mass

The cause of loss of skeletal muscle mass is not fully understood, but may be caused by a number of factors, including energy imbalance, systemic inflammation, disuse atrophy and hormonal changes (Loring et al. 2009). As a result of pulmonary impairments, patients with COPD often present with dyspnoea and hyperinflation, which results in increased work of breathing (Loring et al. 2009).

Reduced exercise tolerance

As well as loss of muscle mass, skeletal muscle abnormalities have been found in COPD. These include morphological and metabolic abnormalities (Seymour et al. 2009). Bernard et al. (1998) found a 30% decrease in thigh cross-sectional area in patients with moderate-to-severe COPD compared with control subjects. There is also evidence of change in muscle fibre type from fatigue resistant fibres to those which have less oxidative enzyme activity and altered mitochondrial function. These are likely to contribute to an inefficient muscle metabolism and loss of muscle strength and endurance (Maltais et al. 1997).

Increased cardiovascular risk

Cardiovascular disease is a common cause of mortality in patients with COPD, which accounts for approximately 30% of deaths in patients with COPD (McGarvey et al. 2007). This may be attributed partly to common risk factors, such as exposure to cigarette smoke and reduced physical activity. Nevertheless, studies have shown that after controlling for these factors, patients with COPD still have an elevated risk of cardiovascular disease (McGarvey et al. 2007).

Osteoporosis

Loss of bone mineral density and osteoporosis is a significant problem in patients with COPD, affecting between 32 and 60% of patients (Bolton et al. 2004). Bone mineral

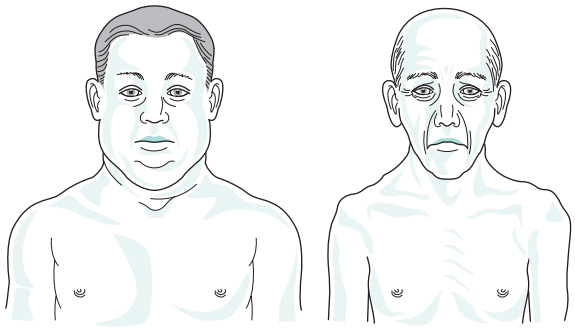


Figure 6.4 Blue bloater and pink puffer.

density has been related to severity of airways obstruction in COPD (Bolton et al. 2004) and to exercise capacity in men with COPD.

Varying clinical presentation in COPD

Within the spectrum of COPD, two extremes of clinical presentation are recognised: type A and type B. At one time these were classified either as 'pink puffers' (type A) or 'blue bloaters' (type B) to correlate with the relative amounts of emphysema and chronic bronchitis respectively (Figure 6.4). While these definitions are over-simplistic, it is worth remembering that patients can present in dramatically different ways (Kesten and Chapman 1993).

Blue bloaters

Patients with this syndrome often show the following symptoms:

- obesity;
- comparatively mild dyspnoea;
- copious sputum which may become infected;
- low PaO_2 and high $PaCO_2$ (PaO_2 > 8kPa; $PaCO_2$ > 6.5kPa) because they tend to hypoventilate;
- central cyanosis with cor pulmonale;
- peripheral oedema;
- an increased residual volume but normal total lung capacity, hence tidal volume is decreased.

Pink puffers

Patients with this syndrome often show the following symptoms:

- an anxious expression;
- general thinness;
- severe breathlessness;
- little or no sputum production;
- relatively normal PaO_2 and $PaCO_2$ (PaO_2 < 8 kPa; $PaCO_2$ normal/low) because of hyperventilation early on in the disease;
- central cyanosis and the development of cor pulmonale in the later stages of the disease;
- generally no peripheral oedema until the late stages of the disease;
- an increased total lung capacity because of hyperventilation.

MEDICAL TREATMENT OF COPD

1. *Decrease the bronchial irritation to a minimum*. The patient should be advised to stop smoking and avoid dusty, smoky, damp or foggy atmospheres. Occupation or housing conditions may need to be changed.
2. *Control infections*. All infections should be treated promptly as each exacerbation will cause further damage to the airways. The patient should have a supply of antibiotics at home and receive a vaccination against influenza each winter. The main organisms of concern are *Streptococcus pneumoniae* and *Haemophilus influenzae,* which are usually sensitive to amoxycillin or trimethoprim.
3. *Control bronchospasm*. Although bronchospasm is not a prominent feature of this disease, drugs (e.g. salbutamol) may be given to relieve the airways obstruction as much as is possible.
4. *Control/decrease the amount of sputum*. Patients with chronic bronchitis may present with excessive bronchial secretions and are usually able to eliminate this by themselves. However, during an episode when secretions may become difficult to eliminate, physiotherapy techniques, including postural drainage; active cycle of breathing technique (ACBT), including Forced Expiratory Technique (FET), Positive Expiratory Pressure (PEP) and oscillating PEP; may aid expectoration (Bott et al. 2009).
5. *Oxygen therapy.* Oxygen must be prescribed and should be given with great care, especially if a normal pH indicates a chronic compensated respiratory acidosis (renal conservation of bicarbonate ions (HCO_3) to maintain pH within 7.35 to 7.45). In this instance HCO_3 is raised above 24 mmol/L whilst PaO_2 is low and the $PaCO_2$ is raised. Controlled oxygen may be given via a Ventimask (or equivalent) with careful monitoring of blood gas levels.
6. *Long-term oxygen therapy* (LTOT). As respiratory function deteriorates, the level of oxygen in the blood falls leading to an increase in pulmonary hypoxic vasoconstriction and deterioration in cardiac function. In 1981, the Medical Research Working Party examined the effects of supplementary low concentrations of oxygen (24%) for 15 hours a day in COPD and found that it reduced three-year

mortality from 66% to 45%. The British Thoracic Society Guidelines (2006b) suggest that patients who have a PaO_2 of less than 7.3kPa, with or without hypercapnia, and a FEV_1 of less than 1.5 litres, should receive LTOT. This therapy should be considered also for patients with a PaO_2 between 7.3 and 8.0kPa and evidence of pulmonary hypertension, peripheral oedema or nocturnal hypoxia.

7. *Noninvasive ventilation (NIV)*. Indicated during an acute exacerbation with type II respiratory failure (see definition in section on Respiratory Failure) (Lightowler et al. 2003; Nava and Hill 2009). NIV offloads the diaphragm enabling it to rest whilst the exacerbation resolves (Fauroux et al. 2003).

Medications

Drugs used in the treatment of respiratory disease fall broadly into two categories: relievers and preventers.

- The *relievers* are used to reduce bronchospasm and include the beta$_2$ (β_2) agonists (which may be short- or long-acting), the anticholinergics and the xanthene derivatives.
- The *preventers* may be used to prevent bronchial hyper-reactivity and reduce bronchial mucosal inflammatory reactions – they include the corticosteroids.

β_2 agonists

β_2-agonists such as salbutamol (Ventolin) and terabutaline (Bricanyl) work by stimulating β_2-receptors, which are widespread throughout the respiratory system. These stimulate adenylate cyclase, which leads to bronchodilation. β_2-receptors are also found in other tissues, including the heart, although these are of the β_1 subtype.

Even though modern bronchodilators are designed to be β_2-selective, they may still cause an increase in heart rate and other side effects, including fine tremor, tachycardia and hypokalaemia (low potassium) after high doses. Inhaled therapy is therefore preferred to oral, as the former limits the amount of drug that finds its way into the general circulation. The long-acting β-agonist agents salmeterol and eformoterol offer a more favourable dose regimen, and respiratory physicians are adding a long-acting β-agonist for patients who have not responded fully to short-acting β-agonists and an anticholinergic used together.

Anticholinergics

Anticholinergic bronchodilators work by preventing bronchoconstriction, mediated by the parasympathetic nervous system. Two agents are currently available: ipratropium bromide and oxitropium bromide. Most studies suggest that these agents are at least as potent as β-agonists when used alone in COPD (Tashkin et al. 1986). A short-acting bronchodilator (β_2-agonist or anticholinergic) used 'as required' is recommended as initial therapy in the British Thoracic Society guidelines (BTS 2001).

Xanthene derivatives

The precise mode of action of the xanthene derivatives such as theophylline and aminophylline remains somewhat uncertain, although they are moderately powerful bronchodilators. They have, however, been shown to improve symptoms in COPD by increasing the contractual ability of the diaphragm (Murciano et al. 1989).

Corticosteroids

The role of inhaled steroids (beclomethasone, budesonide) in COPD will vary from patient to patient. Steroids work by reducing inflammation and reducing bronchial hyperactivity. Trials have shown that about 10–20% of COPD patients will improve significantly following a short course of high-dose oral steroids (Gross 1995).

The most serious limitation to oral steroid therapy is the risk of long-term side effects, which include osteoporosis, adrenal suppression, muscle wasting, poor immune response and impaired healing. However, a positive response to corticosteroids justifies the administration of regular inhaled steroids.

Mucolytic agents

Mucolytic drugs are used to reduce sputum viscosity and improve clearance of secretions. A recent review reported a small reduction in acute exacerbations and a reduction in total number of days of disability (Poole and Black 2010).

Drug delivery systems

The objective of inhaled therapy in COPD is to maximise the quantity of drug that reaches its site of action while minimising side effects from unintended systemic absorption. Most metered-dose inhalers (which are later described in detail for asthmatic patients) are designed to deliver particles of between 0.5 and 10 microns (micrometres). Unfortunately, poor inhaler technique tends to mean that only a relatively small proportion of the drug actually reaches its site of action. It is therefore imperative that a good inhaler technique be adopted (as described for patients with asthma).

In acute exacerbations, when conventional inhalers have proved inadequate, nebulisers may be used to deliver a therapeutic dose of a drug as an aerosol within a fairly short period of time, usually 5–10 minutes (BTS 2006b).

The type of nebuliser for home use consists of a compressor or pump, a chamber and a mask or mouthpiece. The compressor blows air into the chamber, where it is forced through a drug solution and past a series of baffles. The solution is converted into a fine mist, which is then inhaled by the patient through the mask or the mouthpiece.

PHYSIOTHERAPY TECHNIQUES IN COPD

The physiotherapy management for all diseases should be aimed at symptom management once symptoms have been identified during physiotherapy assessment. However, the following general aims of treatment will be described. These aims therefore outline the physiotherapeutic approaches to treatment of the patient with COPD.

General aims of treatment

The general aims are:

- to improve exercise tolerance and ensure a long-term commitment to exercise;
- to give advice about self-management in activities of daily living;
- to increase knowledge of the patient's lung condition and control of the symptoms;
- to relieve any bronchospasm, facilitate the removal of secretions and optimise gaseous exchange;
- to improve the pattern of breathing, breathing control and the control of dyspnoea;
- to teach local relaxation, improve posture and help allay fear and anxiety.

Treatment in the early stages

Key point

The most important themes are maintaining exercise capacity and patient education into self-management. In addition, guidance should be provided with regard to clearing the airways of secretions and establishing a correct breathing pattern.

Increasing/maintaining exercise tolerance

The patient may be treated as an inpatient or as an outpatient, in a health centre or at home by a community physiotherapist. It is important to see the patient regularly. Advice should be given on taking regular exercises, for example a short walk every day. If possible, the patient should be offered participation in a multi-disciplinary comprehensive programme of pulmonary rehabilitation.

ABC Definition

Pulmonary rehabilitation is an evidence-based, multidisciplinary, and comprehensive intervention for patients with chronic respiratory diseases who are symptomatic and often have decreased daily life activities. Integrated into the individualized treatment of the patient, pulmonary rehabilitation is designed to reduce symptoms, optimize functional status, increase participation, and reduce health care costs through stabilizing or reversing systemic manifestations of the disease.

(BTS 2001)

This definition focusses on three important features of successful rehabilitation.

1. A multi-disciplinary approach, which may include respiratory physicians, physiotherapists, occupational therapists, dieticians, nurses, psychologists and therapy assistants.
2. Attention to physical and social function through exercise training, education, nutritional, psychological, social and behavioral interventions.
3. Individualised to meet each patient's needs (BTS 2001).

There is now unequivocal evidence to suggest that pulmonary rehabilitation improves both exercise capacity and health-related quality of life (Lacasse et al. 1996; Guell et al. 2006). In essence, the components of a pulmonary rehabilitation programme include aerobic exercise training, education about the background of the disease, smoking cessation, compliance with medication, nutritional support and energy-conserving strategies for activities of daily living (ADLs). Pulmonary rehabilitation programmes may also include psychosocial support with regard to advice on benefits, sexual function and anxiety management. National Institute for Clinical Excellence (NICE) Guidelines (2010) recommend that all suitable patients with COPD should be offered pulmonary rehabilitation, even after an acute exacerbation.

Inspiratory muscle training

The potential for fatigue of the ventilatory muscles is now recognised as an important component of ventilatory limitation in patients with COPD (Moxham 1990; Green and Moxham 1993). Fatigue may be caused by a combination of:

- increased mechanical load on the respiratory muscles;

- reduced muscle strength;
- reduced energy supply to the respiratory muscles (Roussos and Zakynthinos 1996).

It has also been established that respiratory muscle weakness, which may be a predisposition to muscle fatigue, is present in patients with COPD (Clanton and Diaz 1995; Polkey et al. 1995). It therefore follows that training techniques, which might specifically target the respiratory muscles, may prove beneficial in improving exercise tolerance in patients with COPD who may develop respiratory muscle weakness because of a loss of muscle mass (Geddes et al. 2004).

Removal of secretions

The active cycle of breathing technique (ACBT) (illustrated on Figure 9.9, p.178)

This is a cycle of breathing control, thoracic expansion exercises and the forced expiratory technique (FET), and has been shown to be effective in the clearance of bronchial secretions (Prior and Webber 1979; Wilson et al. 1995) and to improve lung function (Webber et al. 1986).

Thoracic expansion exercises are deep breathing exercises (three or four) which may be combined with a three-second hold on inspiration (unless the patient is very breathless when this may not be tolerated). This increase in lung volume allows air to flow via collateral channels (e.g. the pores of Kohn) and may assist in mobilising the secretions as air is able to get behind the secretions. The increase in lung volume during the inspiratory phase of the cycle may also be achieved by the patient performing a 'sniff' manoeuvre at the end of a deep inspiration. Manual techniques, for example shaking, vibrations or chest clapping, may further aid in removal of secretions.

The FET manoeuvre is a combination of one or two forced expirations (huffs) against an open glottis (as opposed to a cough, which is a forced expiration against a closed glottis). An essential part of the FET manoeuvre is a pause for some breathing control, which prevents an increase in airflow obstruction.

Postural drainage/positioning

This may also aid sputum removal and may be combined with the ACBT technique. The optimum position for effectiveness must be established with each individual, although postural drainage for the lower lobe segments may be difficult as some patients may not tolerate the head-down position or even lying flat. In many patients the ACBT alone may be effective for many in the seated position (Cecins et al. 1999) and changes in position should be used to optimise gaseous exchange. In the lateral position, the lower lung is always better ventilated, regardless of the side on which the subject is lying, although there still remains a bias in favour of the right side because of its larger size when compared with the left lung (Svanberg 1957). Perfusion is also preferential to the lower lung in the lateral position in the spontaneously breathing person (West 2008), although if pathology exists within the lowermost lung gaseous exchange may be compromised because of the presence of pulmonary hypoxic vasoconstriction, which cannot be overcome by gravity (Chang et al. 1993).

Humidification

If the secretions are very thick and tenacious the patient may be given humidification via a nebuliser, usually nebulised saline.

Improving the breathing pattern

The patient is taught how to relax the shoulder girdle in a supported posturally correct position, such as crook half-lying. Breathing control is taught following clearance of secretions. If the patient is breathless, respiratory control is regained starting with short respiratory phases and allowing the rate to slow as the patient's breathing pattern improves.

Treatment in the later stages

It is imperative that patients with COPD are able to maintain as much independence and maximum function as is possible through the support from the hospital or community healthcare team. During acute exacerbations, the ACBT may be continued to assist clearance of secretions. Breathing control should be emphasised so that the patient can walk or climb stairs with confidence. Relaxation positions should be taught for regaining breathing control after activity has made the patient breathless. If the patient becomes very disabled, a walking frame may help to retain some degree of independence as the arms are fixed and accessory muscles of inspiration may be used.

Non-invasive positive-pressure ventilation

Tracheal intubation and mechanical ventilation providing intermittent positive-pressure ventilation (IPPV) is used in intensive care units or high-dependency units to manage patients with deteriorating respiratory failure. However, tracheal intubation may result in complications, including tracheal injury and infection. Furthermore, it may be difficult to wean these patients off IPPV, resulting in a prolonged stay in intensive care.

Non-invasive positive-pressure ventilation (NIPPV) is therefore indicated for the delivery of intermittent positive pressure and may be applied via the nose or mouth using a silicone mask attached to a bedside ventilator. Unlike IPPV, NIPPV can be administered on a general ward for patients in respiratory failure (Sinuff et al. 2000). The ventilator is programmed to supplement the patient's own respiratory effort and, if required, oxygen therapy may be

given in conjunction with NIPPV. NIPPV can be used during an acute exacerbation and has been shown to improve quality of life and arterial blood gas pressures (Meecham-Jones et al. 1995) and to reduce mortality in patients with COPD (Brochard et al. 1995).

Physiotherapy will be required for short spells but frequently throughout the day and sometimes at night. NIV may be used to assist sputum clearance but, if unavailable, intermittent positive-pressure breathing (IPPB) may also be given to assist sputum mobilisation using a mask if the patient is too drowsy to use a mouthpiece. Postural drainage may be necessary, if tolerated, together with rigorous shaking applied during the expiratory phase of the ventilator. Patients should be positioned appropriately in order to facilitate gaseous exchange.

Suction via an airway or nasal suction may have to be used as a last resort to remove secretions if the patient is unable to cough spontaneously or effectively. If $PaCO_2$ is high and PaO_2 is low the patient should not be given a high concentration of oxygen. Drugs such as mucolytic agents or bronchodilators may be provided through the nebuliser attached to the ventilator. The patient should be encouraged to sip drinks because dehydration makes the secretions viscid.

As the patient recovers, treatment should be directed towards that given in the 'early' stages, with special emphasis on a daily maintenance programme of regular exercise, sputum clearance and breathing exercises.

Terminal care

The main theme is to keep the patient as comfortable as possible. Treatment needs to be short and frequent. Non-invasive nasal ventilation may be provided for home use. Inhalations may be used to loosen and liquefy secretions. Suction may be necessary and the GP may provide medication for the patient if the person is being managed at home.

ASTHMA

ABC Definition

Asthma is a clinical syndrome present in all age groups but often starts in childhood and is a disease characterised by recurrent attacks of wheezing and breathlessness. The condition is caused by inflammation and increased sensitivity of the airways. In an attack, the intrapulmonary airways become swollen resulting in reduced air flow during inspiration and expiration. The severity of the narrowing of the airways varies over short periods and is reversible either spontaneously or as a result of treatment (WHO 2004).

Types of asthma

It has been common practice to divide asthma into *extrinsic* and *intrinsic* forms. There is a degree of overlap and many asthmatics, particularly adults, do not fall clearly into either group.

Extrinsic asthma

Extrinsic (atopic) asthma occurs in younger age groups and is caused by identifiable trigger factors, such as specific allergens. Patients are usually sensitive to different factors (e.g. pollen, house dust mites, feathers, fur, dust, pollution and, occasionally, food, drugs and exercise) and have a family history of similar sensitivities. Atopic subjects show an immediate skin reaction, elicited by pricking the skin through a drop of antigenic extract. Exposure to the precipitating factor causes a mucosal inflammatory allergic reaction. This type of asthma tends to be episodic. House dust mites provide the most common positive skin test in Britain, being positive in 80% of children with severe asthma. Extrinsic asthma is common in young people and is associated with a family history of asthma, hay fever and eczema, although new evidence suggests that the presence of more than one of these factors is also associated with the development of asthma in later life (Porsbjerg et al. 2006).

Intrinsic asthma

Intrinsic (non-atopic) asthma tends to occur in the older patient as a chronic condition. This type of asthma is precipitated by, or associated with, chronic bronchitis, strenuous exercise, stress or anxiety. Respiratory infections are also a common factor in precipitating acute attacks, although the majority of these are viral in origin and therefore may represent inappropriate use of antibiotics (Shiley et al. 2010).

Aetiology and prevalence of asthma

The condition can occur at any age but is most common in children, especially boys (ratio of about 3:2). Approximately 10% of children under 10 years of age in the UK have bouts of coughing and wheezing related to narrowing of the airways. Asthma accounts for more absences from school than any other chronic disease, although days lost from school may be under-estimated owing to the under-diagnosis and under-treatment of childhood asthma (Baena-Cagnani and Badelliono 2010).

Childhood asthma generally remits after puberty but it may return in later life. Asthma that starts in middle age is more common in women than men and remission in this age group is rare. The majority of cases of asthma are mild, although the course of the disease is unpredictable. The mortality rate is unacceptably high and has shown a slow rise since the 1960s; in 2008 in England and

Wales there were 1204 reported deaths as a result of asthma (BTS 2008).

The rise in the prevalence of asthma has continued since 1988 but had been shown to decline by 2003 (Burr et al. 2006). However this decline in asthma prevalence is thought to be a result of better disease management, as more children are now using inhaled corticosteroids as a preventive treatment (Burr et al. 2006). However, despite the rise in the use of corticosteroids under-treatment and inadequate appreciation of the severity of asthma by patients and doctors are important factors in determining mortality, with up to 86% of asthma deaths being preventable. Those most at risk are the patients who underestimate their symptoms. About 15–20% of asthmatics do not notice moderate changes in their airflow obstruction (National Asthma Campaign 2002) and may quickly deteriorate until they suddenly present with severe asthma. Patients with inadequately controlled, severe persistent asthma are at a particularly high risk of exacerbations, hospitalisation and death, and often have severely impaired quality of life (Peters et al. 2006).

Pathology of asthma

In all types of asthma an underlying problem seems to lie in abnormal reactivity of the airways; that is, they narrow excessively in response to stimuli which would not affect normal subjects (Bone 1996). The main pathological changes occurring during an asthmatic attack are:

- spasm of the smooth muscle in the walls of the bronchi and bronchioles (bronchoconstriction);
- oedema of the mucous membrane of the bronchi and bronchioles;
- excessive mucus production and mucus plugging.

These changes result in airways obstruction. The bronchial walls become infiltrated with eosinophils and there is thickening of the epithelial basement membrane.

At the end of an attack these changes are almost totally reversible, but if attacks occur frequently then long-standing changes will occur. Such changes are hypertrophy of the smooth bronchial muscle, which increases the effect of bronchial spasm during an attack; permanent thickening of the mucous membrane with an increase in the number of goblet cells and mucous glands; over-distension of the alveoli as a result of the trapping of air; and atelectasis of alveoli when a bronchiole, already narrowed, becomes blocked by mucus plugs.

Where the predominant factor precipitating asthma is an allergic reaction there is antigen-mediated bronchoconstriction. This means that the antigen (allergen or precipitating factor) binds to two IgE molecules (immunoglobulin antibodies) on the membranes of mast cells present in the bronchial lining. This binding releases mediators that act on receptor sites on smooth muscle cells, causing changes in intracellular cyclic adenosine monophosphate (AMP) levels which result in muscular contraction. The mediators histamine, neutrophil chemotactic factor (NCF-A), platelet activating factor (PAF) and eosinophil chemotactic factor (ECF-A) are stored in granules within the mast cells as pre-formed mediators. This antigen-antibody reaction is part of the body's immune response and previous exposure to the antigen results in greater bronchoconstriction.

Clinical features of asthma

Extrinsic asthma

In extrinsic asthma the onset is often sudden and paroxysmal, often at night. An attack starts with chest tightness, dryness or irritation in the upper respiratory tract. Attacks tend to be episodic, often occurring several times a year. Their duration varies from a few seconds to many months and the severity may be anything from mild wheezing to great distress. The most predominant features are summarised below.

Wheeze and dyspnoea

Dyspnoea may be intense and chiefly occurs on expiration, which becomes a conscious exhausting effort with a short gasping inspiration. Wheezing is always present on expiration but may also occur on inspiration in severe asthma.

Cough

At the initial stage of an attack the cough may be unproductive and 'barking' in nature. It causes an increase in bronchospasm and dyspnoea. As the attack subsides, the cough becomes productive of casts or plugs of sputum. Such plugs – made up of yellow viscid mucus and desquamated epithelial cells and eosinophils – are often very occasionally coughed up during acute attacks, which may produce a marked relief of symptoms. A cough may be the only presenting symptom of asthma, particularly in children (Corraco et al. 1979).

Posture

The patient will prefer to sit upright with the shoulder girdle fixed (by grasping a table or bed) to assist the accessory muscles of respiration. The chest is hyperinflated.

Pulse

This is rapid and there may be an increased drop in blood pressure during inspiration (>10 mmHg) owing to an exaggeration in intrathoracic pressure swings caused by severe airways obstruction (pulsus paradoxus). However, pulsus paradoxus may be absent even in very severe attacks of asthma. When it is present, the measurement is easily performed with a sphygmomanometer and provides a guide to progress and response to treatment (Pearson et al. 1993).

Electrocardiogram

This will show a tachycardia and may show signs of right ventricular strain or the development of a large P wave (P pulmonale). These abnormalities will return to normal as the attack subsides.

Cyanosis

This may occur at a very late stage in the progression of the disease because of worsening hypoxaemia (low PaO_2) if this is not corrected with adequate oxygen therapy.

Blood gases

Analysis of blood gases provides important information to help the management of severe asthma. The usual finding is of a low arterial PaO_2 (hypoxaemia) caused by ventilation/perfusion mismatch and a low $PaCO_2$ (hypocapnia) because of the effects of hyperventilation. Later in the disease process, the $PaCO_2$ may be found to be high because the hyperventilation fails to compensate for the fact that there are many under-ventilated alveoli which are distal to the blocked bronchioles. When the $PaCO_2$ is found to be increasing and the pH is low this should be a danger sign that the patient may be becoming tired and be likely to need assisted ventilation if immediate improvement cannot be achieved (BTS 2008).

Breath sounds

These are vesicular with evidence of a prolonged expiration and high-pitched wheeze. Crackles may also be heard if sputum is present. During severe attacks with worsening obstruction, the breath sounds may be diminished and occasionally become inaudible (silent chest) because of diminished airflow.

Percussion note

The note may be hyper-resonant if the patient is hyperinflated.

Chest X-ray

Radiography is not usually helpful in the management of asthma. It usually shows only over-inflation, although may also show a pneumothorax if this is suspected.

Lung function

FEV_1 and FVC drop during a severe attack with little sign of reversibility (Figure 6.5). However, if FEV_1 is measured before and after giving bronchodilators, and there is a 15% increase in FEV_1, this amounts to significant reversibility. The FEV_1 may be less than 30% of FVC. Total lung capacity, FRC and RV may be increased because of over-inflation of the lungs. Recovery is associated with a reduction in these lung volumes. Recordings of the peak expiratory flow rate (PEFR) for a week at home will often make the diagnosis of asthma obvious (Prior and Cochrane 1980). PEFR dips in the morning, particularly during the recovery phase (Figure 6.6). If the dip is severe (less than 33% of predicted) then respiratory arrest may occur (BTS 2008). In a severe attack, the PEFR may drop below 100 L/minute. However, it should be noted that a normal PEFR when a patient is asymptomatic does not exclude a diagnosis of asthma (BTS 2008).

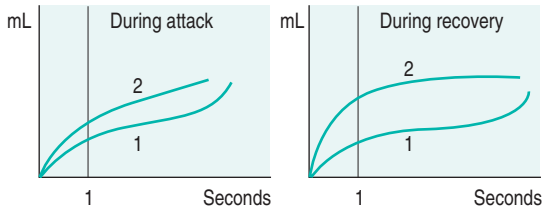


Figure 6.5 Asthma: FEV_1 and FVC reversibility during an attack and recovery. 1 = before bronchodilator; 2 = after bronchodilator.

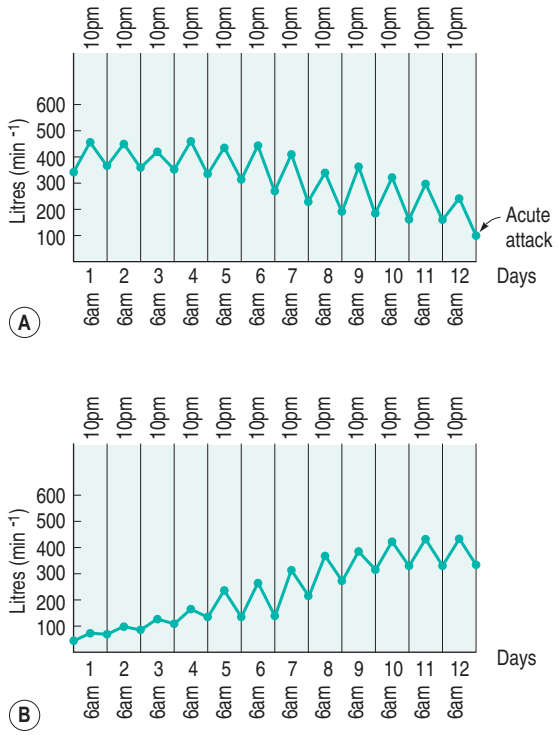


Figure 6.6 Dipping charts in asthma. (a) Recording of peak flow at home showing deterioration before onset of acute attack. Intervention when there is a chart like this can prevent the attack. (b) During recovery there is still diurnal variation. These patients are at risk and should have long-term monitoring.

Table 6.3 Warning signs of an attack of acute severe asthma

Early warning signs	Signs of increasing severity
Increase in symptoms	Dyspnoea at rest
Sleep disturbance	Peak flow below 100 L/min
Increase in bronchodilator use	Deteriorating blood gases
Fall in peak flow	Pulsus paradoxus
Decrease in exercise tolerance	Tachycardia

Between attacks

No abnormality should be detectable between attacks, although children with severe asthma may develop a pigeon chest or have a persistent, low-pitched wheeze with a productive cough.

Intrinsic (chronic) asthma

This is less paroxysmal in character than extrinsic asthma and is often associated with chronic bronchitis.

Clinical features are similar to those described above for extrinsic asthma, but wheeze and dyspnoea tend to be continuous and worse in the morning. Cough produces mucoid sputum, respiratory infections occur with increasing frequency and radiographs may show emphysematous changes.

Acute severe asthma

As asthma is, by nature, a paroxysmal condition, acute attacks that are resistant to bronchodilators may occur. Such attacks are potentially life-threatening, so prompt and effective treatment is imperative. In general, clinical criteria are most helpful and the recognition by the person that his or her asthma symptoms are worsening. Table 6.3 lists warning signs of an acute severe attack.

MEDICAL TREATMENT OF ASTHMA

Oxygen therapy

Unlike patients with long-standing COPD, patients with asthma may tolerate higher levels of oxygen to correct hypoxaemia. During an acute episode it is essential that oxygen therapy be titrated according to the level of PaO_2. It may be evident on clinical examination, however, that the asthmatic patient also has evidence of COPD. In this situation examination of the blood gases will reveal whether the patient has had raised $PaCO_2$ levels for some time (a chronic compensated respiratory acidosis will be evident on an arterial blood gas analysis) where the use of controlled oxygen will be required. If a patient is reaching a stage where the respiratory muscles are starting to fatigue the $PaCO_2$ may rise. In this situation immediate medical attention is required as the asthma is deemed life-threatening and the patient is likely to require invasive mechanical ventilation (Richards et al. 1993).

Medications

Suitable drugs are discussed in the earlier section on the management of patients with COPD. Their use is discussed here particularly with regard to the treatment of asthma.

β_2 agonists

The side effects of salbutamol (Ventolin) and terabutaline (Bricanyl) include fine tremor, tachycardia and hypokalaemia (low potassium) after high doses.

Corticosteroids

In a small proportion of asthmatic people, long-term oral corticosteriods (e.g. beclomethasone and budesonide) will be necessary. In circumstances when an attack supervenes very rapidly, a short course of oral steroids (prednisolone) is required. It cannot be emphasised enough that this approach is safe and certainly much safer than a poorly-controlled attack of asthma. There have been suggestions that the adverse effects associated with long-term steroids, such as osteoporosis, might be less common in asthma but this has been shown to be untrue (Adinoff and Hollester 1983). The introduction of inhaled corticosteroids in 1972 radically changed the management of asthma as side effects from oral steroids were prevented.

Leukotriene antagonists

Oral inhibitors of leukcotriene action may help to reduce the inflammatory component of asthma (Israel et al. 1993).

Mucolytic agents and asthma

Mucolytic drugs are used to reduce sputum viscosity and improve clearance of secretions. A recent review reported a small reduction in acute exacerbations and a reduction in total number of days of disability (Poole and Black 2010).

Other agents

Other types of medication that may be useful include:

- anticholinergic agents;
- long-acting β_2 agonists;
- theophylline;
- salmeterol;
- chromones (sodium chromoglicate);
- antihistamines and ketotifen.

Delivery of medication

Metered-dose inhalers

It is good practice to use inhaled therapy for asthma. This keeps the dose down and reduces side effects.

β_2 agonists, anticholinergic agents and corticosteroids are frequently prescribed in metered-dose inhalers (MDIs). The particles leaving an MDI do so with considerable velocity and even with a perfect technique of inhalation, only about 10% of the dose reaches the respiratory tract – the remainder is deposited in the mouth or swallowed (Davies 1973). The MDI does have the advantage of being small and portable and familiar to many asthmatics. The MDI can also be used with a spacer, which virtually removes oropharangeal deposition, thereby increasing lung deposition to 20–30% (O'Callahan and Barry 1997). Inhaled steroids can cause hoarseness and oral thrush so a spacer device may be prescribed to minimise deposition of large particles of the medication around the mouth and throat.

Breath-activated devices (Figure 6.7) are primed before actuation and the MDI is triggered by inspiratory airflow. The airflow required is low and the triggering of the device quiet enough not to disturb the inspiration. β_2 agonists, anticholinergic agents and corticosteroids can be prescribed in this form of inhaler.

There are various *dry powder systems* (Figure 6.8):

- the Turbohaler is a multidose dry powder system which requires an inspiratory airflow of only 60 L/minute. Patients tend to find this system easy to use;
- the Disk/Accuhaler is a dry powder multidose system used for the delivery of salmeterol and fluticasone propionate;
- with Rotocaps each dose of medication is loaded into the inhaler prior to use and so the inhaler needs to be stored in a dry place. Rotocaps absorb moisture and the particles can become too large to inhale.

Nebulisers

If asthma symptoms become severe, inspiratory airflow may become limited to such an extent that the contents of a MDI cannot be inhaled adequately. With a nebuliser, a high-velocity jet of air or oxygen sucks liquid up a tube and the liquid is broken into tiny particles which are inhaled and deposited in the lungs (Rees et al. 1982) (Figure 6.9). Ultrasonic nebulisers may also be used to deliver medication, although there are currently insufficient data to verify their advantage over other nebulisers in the management of patients with asthma and COPD (Brocklebank et al. 2001).

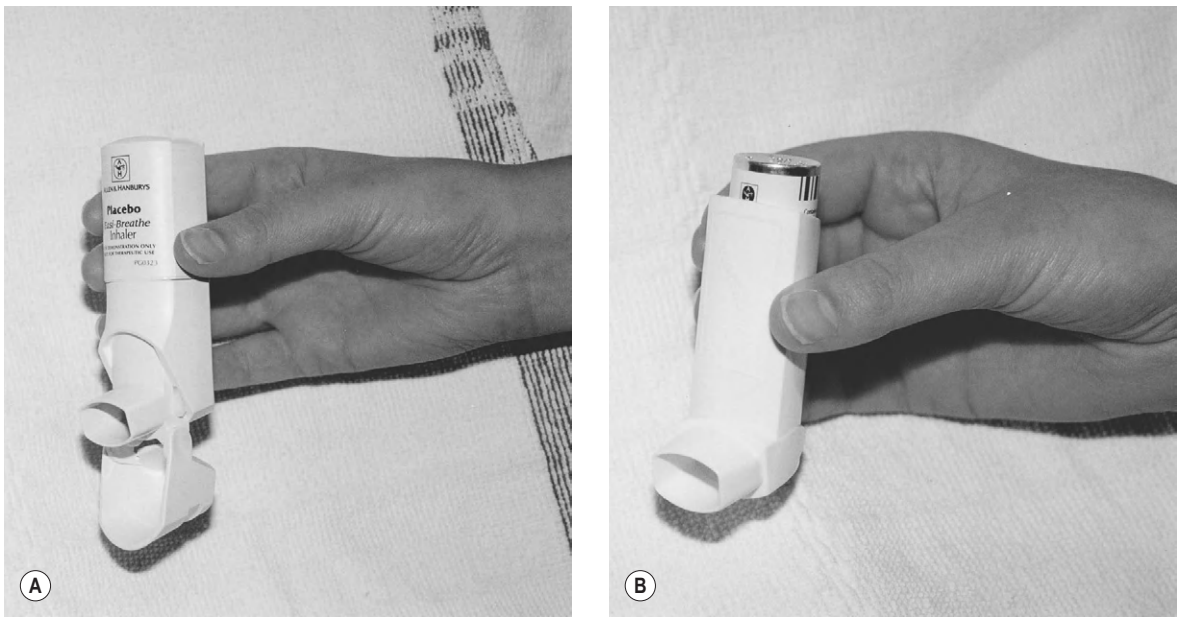


Figure 6.7 Breath-activated metered-dose inhalers. *Photos courtesy of Melanie Reardon and Joanne Kenyon.*

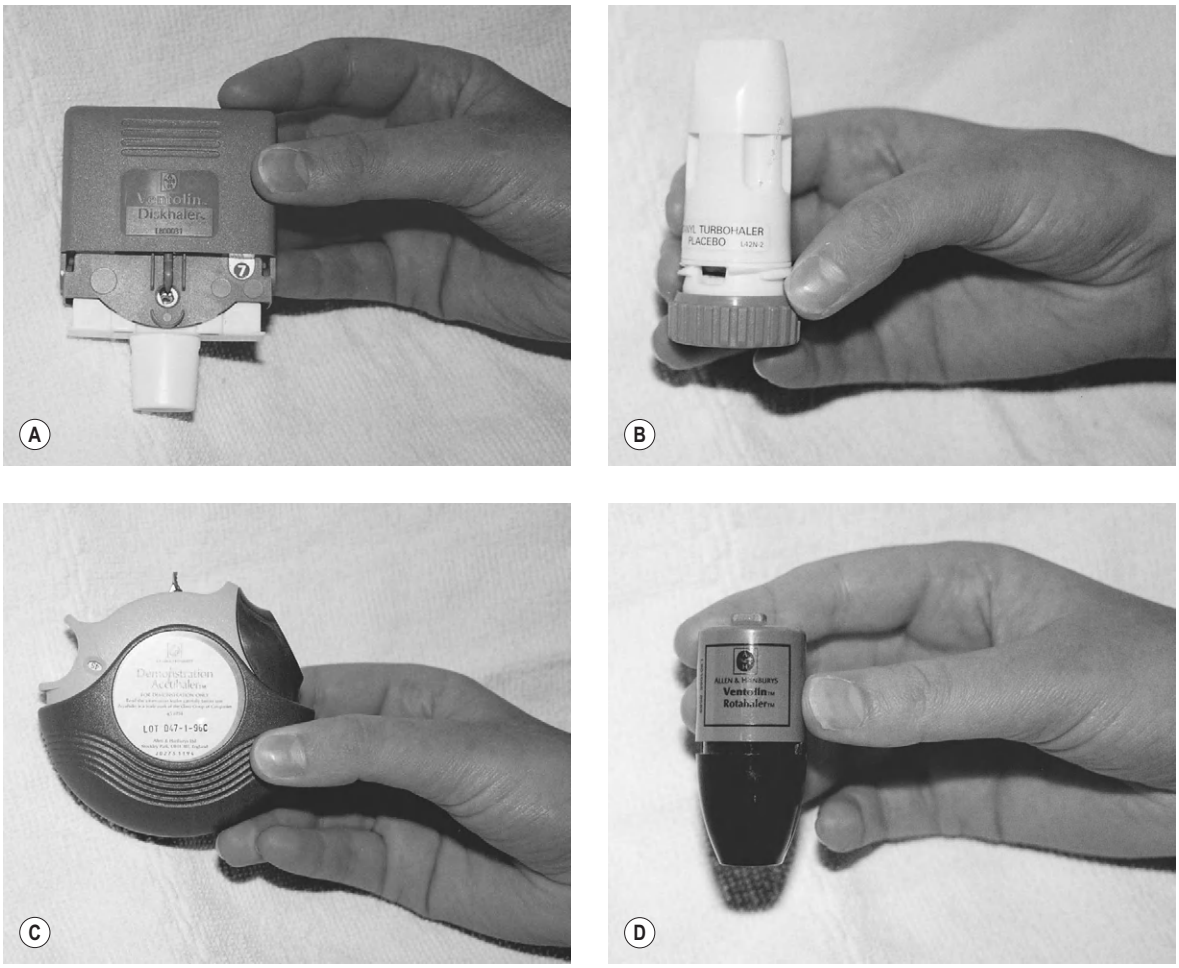


Figure 6.8 Dry powder systems: (a) Diskhaler; (b) Turbohaler; (c) Disk/Accuhaler; (d) Rotocaps. *Photos courtesy of Melanie Reardon and Joanne Kenyon.*

Guidelines for drug therapy

The British Thoracic Society (2001) introduced guidelines on the drug management of asthma. Steps 1–3 below apply to the treatment of less severe asthma, in an attempt to control symptoms. Steps 4 and 5 apply to the treatment of more severe asthma, when it may not be possible to abolish symptoms. Stepping down and up this treatment ladder is recommended to match therapy to the person's current need.

- *Step 1*: Inhaled short-acting β_2 agonists p.r.n. (when required), but not more than once daily. If needed more than once daily, move on to step 2. No prophylaxis (preventers such as inhaled corticosteroids).
- *Step 2*: Inhaled short-acting β_2 agonists p.r.n. Regular low-dose inhaled steroids, such as 100–400 μg beclomethasone twice daily or budesonide daily via a large volume spacer.
- *Step 3*: Inhaled short-acting β_2 agonists p.r.n. Regular high-dose inhaled steroids, such as 800–2000 μg beclomethasone or budesonide daily via a large volume spacer. In a very small number of patients who experience side effects with high-dose inhaled steroids, either the long-acting inhaled β_2 agonists option is used *or* a sustained-release theophylline may be added to step 2 medication.
- *Step 4*: Inhaled short-acting β_2 agonists p.r.n. Regular high-dose inhaled steroids, such as 800–2000 μg of beclomethasone, or budesonide daily via a large volume spacer. Add in long-acting inhaled β_2 agonists *or* sustained-release theophylline or high-dose inhaled bronchodilators.

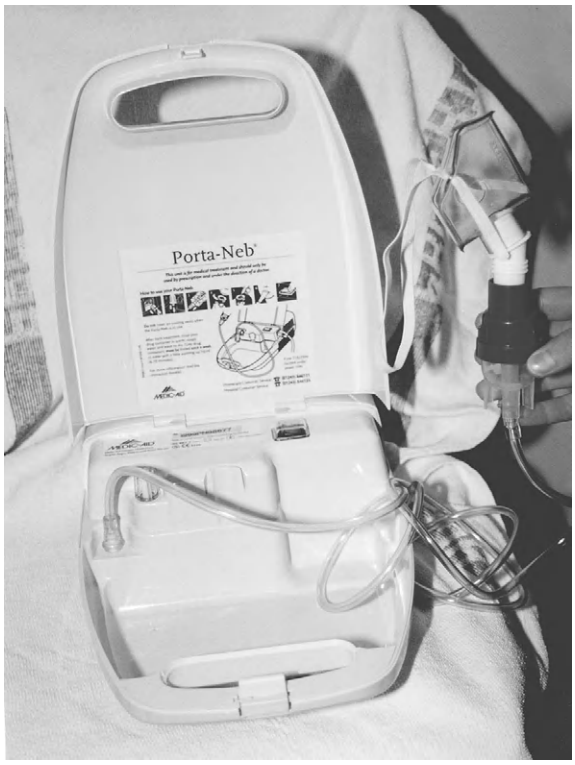


Figure 6.9 Portable nebuliser.

Table 6.4 Peak flow action plan

Daily medication	Seretide (purple inhaler) **One suck every morning and evening**
As-needed medication	Salbutamol (blue inhaler) **Two puffs as needed**
Best peak flow reading when well	390 L/min
If unwell with cough, wheeze, breathlessness or tight chest or peak flow 310 L/min or below	Continue daily medication(s) as above Commence salbutamol (blue) **Four puffs every four hours** Record peak flow readings every four hours before taking blue inhaler
If peak flow falls to 230 L/min	Commence oral steroids **eight tablets once a day** for three days See your doctor within 24 hours of commencing oral steroids
If the action plan does not help relieve your child's symptoms for four hours or peak flow reading falls to 150 L/min, then you must seek urgent medical attention.	

- *Step 5*: Inhaled short-acting β_2 agonists p.r.n. Regular high-dose inhaled steroids, such as 800–2000 μg of beclomethasone, or budesonide daily via a large volume spacer. Add in one or more long-acting bronchodilators, as in step 4, *plus* regular oral prednisolone as a single daily dose.

Management between attacks

The environment

This comprises identifying and removing the cause or trigger, if known. For example, a patient may have to avoid certain foods and damp. The home environment should be cleaned regularly, bedding vacuumed frequently, and synthetic fibres used in place of feathers for pillows and quilts. Gor-tex mattress covers are not inexpensive but are impervious to mites and their faecal particles. It may also be of benefit to avoid certain domestic animals, although getting rid of a loved family pet may provoke emotional problems in children, which can make their asthma worse. Desensitisation may be possible by injection of mild doses of the allergen, which will have been identified by a skin test, although avoidance of these allergens altogether is advised.

Measurement of peak flow

Measurement of peak flow can be useful so that variability of lung function is clearly seen and treatment is titrated accordingly. The patient may then alter medication in order to control his/her symptoms. This is based upon the extent to which the peak flow deteriorates (60%, 40% or 30% of the patient's expected value). An example of a peak flow action plan, which will be kept by the patient for reference and which is based on the British Thoracic Society guidelines (2008), is shown in Table 6.4.

Inhaler technique

When drugs are administered by aerosol/inhaler it is important that the patient be taught how to use the device correctly. The use of various inhaler devices requires some skill on the part of the patient and teaching the correct method of use is an essential part of the prescription of such treatment. Failure to master a metered-dose inhaler occurs in many patients of all ages. The technique for a metered-dose inhaler in which the drug is suspended in a propellant is as follows.

1. *Shake the inhaler.* This disperses the drug uniformly throughout the propellant.

2. *Hold the inhaler upright and direct into the mouth.* If it is not held upright the metering chamber will not fill correctly.
3. *Start inspiration and press the activating mechanism.* The drug will be effective only if it is breathed in during inspiration. Although there is some controversy regarding the lung volume at which the actuation should occur (Newman et al. 1981), it is simpler to teach the person to discharge the inhaler at the beginning of inspiration.
4. *Breathe in slowly through the mouth.* The flow rate of inspiration should be slow (Newman et al. 1982). This helps to reduce impaction on the pharynx and allow for further penetration of the drug into the bronchial tree, as flow is laminar rather than turbulent.
5. *Hold the breath at maximum inspiration for 5–10 seconds.* This allows particles of the drug to settle on the airway walls. Ideally, the breath should be held for 10 seconds.
6. *Relax and allow easy expiration.* Patients need to be aware of when their inhaler is getting close to empty and patients should be instructed to shake their inhaler in order to gauge this. Another method is to place the inhaler in water. If it floats symmetrically upright then it is close to empty. It is always good advice to instruct the patient to have two inhalers at home, to avoid being caught out in an exacerbation without adequate relief.

PHYSIOTHERAPY TECHNIQUES IN ASTHMA

As in patients with COPD, physiotherapy management for asthma should be aimed at symptom-management once symptoms have been identified during physiotherapy assessment.

General aims of treatment

The principal aims are:

- to relieve any bronchospasm and to facilitate the removal of secretions;
- to improve breathing control and the control of dyspnoea during attacks;
- to teach local relaxation, improve posture and help allay fear and anxiety;
- to increase knowledge of the lung condition and control of symptoms;
- to improve exercise tolerance and ensure a long-term commitment to exercise;
- to give advice about self-management.

The management of asthmatic patients should include maintenance of a good general fitness and a vital part of asthma management is to educate the patient.

Patient education

All asthmatic patients and their close relatives should be aware of how to manage their asthma, and the physiotherapist is integral in the education process. Prevention of infection is important. The patient should have plenty of fresh air, avoid smoky atmospheres and keep away from people with infections such as bronchitis and influenza. Stress or anxiety must be minimised as these can precipitate an attack.

Patients must know what therapy to take and how to take it, and where they should seek further help. All this should be carefully planned beforehand and incorporated into a written action plan and self-management strategy (Amado and Portnoy 2006) which guides the patient to increase treatment when their asthma becomes more severe and to reduce treatment when it gets better (refer to Table 6.4).

Acute attacks

Treatment during acute exacerbations will involve the physiotherapist in aiding removal of excessive bronchial secretions using the ACBT technique (see above), with the addition of postural drainage, if tolerated. Percussion and shakings should be applied sensitively as they may increase bronchospasm. Breathing control and the adoption of relaxed positions may be necessary.

Pulmonary rehabilitation

Pulmonary rehabilitation has largely been confined to patients with COPD, but there is now recognition that other patient groups may benefit. The principles are the same as those previously described for patients with COPD, but with some additional considerations.

Patients with asthma are younger but very commonly have a fear and inhibition of exercise (Cochrane et al. 1990) and therefore can benefit from improved cardiorespiratory fitness (Patessio et al. 1993). Also, unlike patients with COPD, individuals with asthma usually show a greater variability in airflow obstruction and are more susceptible to exercise-induced exacerbations (EIEs). Consideration needs to be given to the prevention of exacerbations (such as by the self-administration of β_2 agonists prior to exercise), although certain exercises – for example swimming – are the least likely to cause an EIE. In addition to whole-body programmes, inspiratory muscle training may also be incorporated into the exercise programme (as described previously for COPD patients).

Removal of secretions

Some patients, especially children, have constant excessive secretions and may require sputum clearance techniques, such as the ACBT, on a daily basis. It may be essential to

teach FET for clearing secretions without increasing bronchospasm.

Relaxation

If the patient is able to practise relaxation it may be possible to ward off an attack when there has been exposure to an allergen. The onset of an attack is often preceded by a 'tickle' in the throat or a sensation of tightness in the chest. Relaxation and breathing control in an appropriate position may prevent an attack developing. 'Appropriate position' depends on where the patient is and may have to be against a wall or the back of a chair.

Breathing control

Breathing exercises may be taught with the aim of reducing respiratory rate and/or tidal volume. Encouraging a longer expiratory phase is helpful, but neither inspiration nor expiration should be forced. This may be helped by counting (e.g. 'in 1-2, out 1-2-3') and by manual pressure just under the xiphisternum to encourage diaphragmatic excretion. The patient must breathe at a rate and rhythm that suits him/her. Children may be taught to breathe to a nursery rhyme.

There is evidence that the Buteyko breathing technique may help control symptoms of asthma; however, cost implications of training staff in this technique and time involved should be taken into consideration (Bott et al. 2009). Breathing exercises should be seen as an adjunct to conventional asthma management and not as a replacement for medication (Bott et al. 2009).

BRONCHIECTASIS

ABC Definition

Bronchiectasis is a chronic inflammatory disease of the lungs, defined as dilatation and destruction of bronchi and bronchioles. It results from impaired lung defence which results in repeated infection, inflammation and destruction of the airways.

Bronchiectasis is identified by anatomical and morphological changes, which can be identified by high-resolution computed tomography (HRCT). Although bronchiectasis is distinct from COPD, it shares common symptoms of impaired mucus clearance, chronic cough, recurrent infection, wheeze, dyspnoea, airflow limitation and decreased exercise tolerance. Indeed, up to 50% of patients with COPD have been found to have evidence of bronchiectatic changes on HRCT scan (Patel et al. 2004).

Types and prevalence of bronchiectasis

The condition most commonly affects the lower lobes, the lingula and then the middle lobe. It tends to affect the left lung more than the right, although 50% of cases are bilateral. The upper lobes are least affected as they drain most efficiently with the assistance of gravity. Broadly, there are two types of disease.

Congenital bronchiectasis

This is very rare and occurs in Kartagener's syndrome ('immotile cilia' syndrome) where there is a congenital microtubular abnormality of the cilia that prevents normal cilial beating. It is characterised by bronchiectasis, sinusitis, dextrocardia and complete visceral transposition. There may also be associated male infertility.

Acquired bronchiectasis

Bronchial obstruction and bacterial infection are the principal factors responsible for this disease. Obstruction of a bronchus, which may be caused by a tumour or foreign body, will cause collapse of the lung tissue supplied by that bronchus. Bronchiectasis may also occur following an infection, which causes the production of sticky sputum leading to obstruction of multiple small bronchi. Classically, this is associated with whooping cough, tuberculosis, measles and pneumonia in childhood, when the airways are smaller and therefore more easily 'plug' with sputum (Shoemark et al. 2007). Very occasionally, bronchiectasis may occur as a late complication of tuberculosis, which has affected the right middle lobe causing that segment to collapse. It may also occur following lung abscess and pneumonia, and be associated with immune defects in patients with hypogammaglobulinaemia. Allergic bronchopulmonary aspergillosis, which is associated with an autoimmune response, can cause formation of mucus plugs resulting in bronchiectasis of the medium-sized bronchi.

Prevalence

The prevalence of bronchiectasis following a childhood infection is decreasing as these infections are now treated with antibiotics, but may occur in individuals with an underlying disorder that predisposes them to chronic or recurrent infection. This includes cystic fibrosis, immunodeficiency, HIV infection and primary ciliary dyskinesia (Rosen 2006).

Pathology of Bronchiectasis

Bronchial obstruction may be localised (perhaps because of an inhaled foreign body such as a peanut or broken

tooth, or obstruction caused by a tumour or enlarged gland) or generalised (e.g. pneumonia that is slow to resolve owing to whooping cough or measles).

The bronchial obstruction will cause absorption of the air from the lung tissue distal to the obstruction and this area will therefore shrink and collapse. This causes a traction force to be exerted upon the more proximal airways, which will distort and dilate them. If the obstruction can be cleared and the lung re-expanded quickly then the dilatation is reversible. Secretions may collect distal to the obstruction if it is not relieved quickly and these become easily infected. This causes inflammation of the bronchial wall with destruction of the elastic and muscular tissue. These infections occur repeatedly with the walls becoming weaker and weaker. They will eventually dilate because of the negative intrapleural pressure. As the disease advances, the bronchi become grossly dilated and pockets containing pus are formed. The elastic and muscle tissue is destroyed and the mucous lining is replaced by granulation tissue with loss of cilia. Therefore, the mucociliary transport mechanism is disrupted and passage of mucus out of the lungs is therefore hindered.

Several types are recognised pathologically: tubular, fusiform or sacular. The arterial vessels within the bronchial walls anastomose with the pulmonary capillaries and this results in the common feature of haemoptysis.

Clinical features of bronchiectasis

Key point

Although symptoms often begin in childhood, diagnosis is not usually made until adult life.

Cough and sputum

Patients complain of persistent cough with purulent sputum from childhood. Initially, it would be present only following colds or influenza, but if the disease is allowed to progress in its severity the affected segments continually accumulate purulent secretions, resulting in cough and sputum production. The sputum is usually green, often foul smelling and present in fairly large volume. The breath is fetid. The cough is particularly troublesome on a change of position and on rising first thing in the morning.

Initially, the sputum culture will isolate *Haemophilus influenzae* and/or *Staphylococcus*. In the later stages of the disease *Pseudomonas aeruginosa* and *Klebsiella* spp. may be isolated.

Dyspnoea

Shortness of breath is noticeable only if the disease is particularly severe and widespread. If the bronchiectasis is localised, other well-ventilated and perfused alveoli should maintain blood gases at a reasonable level, although bronchospasm may be a feature particularly during an exacerbation.

Haemoptysis

This occurs quite commonly, usually associated with an acute infection. It can be life-threatening if severe and may require surgical resection of the affected lung tissue.

Recurrent pneumonia

Characteristically this will affect the same sites and is a common feature.

Chronic sinusitis

This occurs in approximately 70% of the patients.

General ill-health

Patients may suffer pyrexia, night sweats, anorexia, malaise, weight loss, lassitude and joint pains.

Clubbing

In about 50% of patients fingers and toes become clubbed. The first sign of clubbing is loss of the angle between the nail and the nail bed. This is followed by curvature of the nail and an increase in the soft tissue of the ends of the fingers to form so-called 'drumstick' fingers.

Thoracic mobility

This gradually decreases, as do shoulder girdle movements.

Radiography

Initially, the X-ray will be normal but the patient gradually develops an increase in the bronchovascular markings and sometimes shows multiple cysts with fluid levels (Armstrong et al. 1995). Bronchography is used for accurate localisation of the area affected and will reveal dilated bronchi. A CT scan will show bronchial wall thickening and dilation of the bronchi and cysts.

Prognosis of bronchiectasis

The vast majority of these patients can lead normal lives with a nearly normal life expectancy provided the medical care is adequate. However, possible complications are:

- recurrent haemoptysis (common);
- pneumonia (common);
- pleurisy and empyema;

- abscess formation (in lung/cerebrum) (rare);
- emphysema (rare);
- respiratory failure;
- right ventricular failure (commonly develops after years of pulmonary sepsis and arterial hypoxaemia if there is widespread bronchiectasis).

MANAGEMENT OF BRONCHIECTASIS

Principles of treatment

The anatomical picture makes very little difference to the treatment of the disease.

- Relieve the obstruction before permanent damage occurs (recognition of either localised obstruction or appropriate treatment for whooping cough or measles).
- Maintain and improve exercise tolerance, as some patients with bronchiectasis become deconditioned owing to fatigue and shortness of breath.
- Control infection. Antibiotics are given prophylactically in all but very mild cases. The dosage of the antibiotics should be altered if an acute infection occurs. Intravenous treatment is indicated for severe infections (Currie 1997). Inhaled (delivered by a nebuliser) or continuous oral therapy may be used for chronic sepsis and more resistant pathogens (e.g. *Staphylococcus aureus* and *P. aeruginosa*).
- Promote good health with a good diet and fresh air.
- Inhaled steroids may be used in order to reduce inflammation and reduce the volume of sputum produced (Elborn et al. 1992).
- Surgery to remove the area of affected lung may be indicated in young patients with localised disease, although there is conflicting evidence regarding the efficacy of surgery when compared to conservative treatment (Corless and Warburton 2000).

PHYSIOTHERAPY FOR BRONCHIECTASIS

Aims of treatment

The principal aims of physiotherapy in bronchiectasis are:

- to promote good general health and maintain or improve exercise tolerance;
- to remove secretions and clear lung fields;
- to teach an appropriate sputum clearance regimen for independent use;
- to educate the patient in the pathology and management of the condition;
- to teach the patient how to fit in home treatment within his or her lifestyle.

Clearing secretions

Postural drainage may be indicated, if tolerated, for patients with excessive bronchial secretions. The position must be accurate for the areas of lung affected. Accuracy is judged by production of sputum and by identification of the affected areas on a chest radiograph. This minimises the danger of secretion overspill into the least affected side, which could cause spread of the disease or pneumonia. Percussion, shaking and vibrations with the ACBT are also necessary and must be accurately applied over the affected area of the lungs.

The patient may be taught the FET. A flutter or PEP valve may be used to facilitate the movement of peripheral mucus plugs and pus into the trachea from where they are cleared by coughing. NIV or IPPB may be used in the breathless patient as an adjunct to reduce the increased work of breathing during physiotherapy. The patient must perform a combination of these treatments 2–3 times daily. It is important to ensure that the patient has disposable sputum pots and polythene or paper bags to dispose of the infected sputum without the risk of reinfection or endangering other members of the family. Should the patient develop a cold or influenza, antibiotics must be readily available together with physiotherapy so that infection and secretions can be cleared promptly.

In order to enhance sputum clearance, nebulisation of sterile water, normal saline, hypertonic saline (care must be taken in case of bronchial hyper-reactivity), β_2 agonists or a bronchodilator may be considered (Bott et al. 2009).

Maintaining exercise tolerance

Mobility of the thorax, good posture and good general health are achieved by the patient performing a daily exercise programme. This comprises general deep breathing, maintenance of good posture and aerobic exercise, such as brisk walking. The patient may also attend pulmonary rehabilitation as this has been shown to be effective in increasing exercise capacity in patients with bronchiectasis (Bradley and Moran 2006). The patient should also be encouraged to partake in sports, such as jogging, walking, cycling, tennis or swimming.

CYSTIC FIBROSIS

Cystic fibrosis (CF) is the most common hereditary disorder transmitted by a recessive gene, estimated to be present in 1 in 25 people in the UK. CF is the most common life-shortening autosomal recessive disorder in the Caucasian population. It is caused by mutations in a single gene on

ABC Definition

Cystic fibrosis (CF) is a life-shortening hereditary disease affecting Caucasians estimated to affect 250,000 persons worldwide. It is a complex, multisystem disease caused by mutations of the CF transmembrane conductance regulator gene (CFTR) (Ratjen and Doring 2003). The mutations affect transport of ion and water transport across the epithelial linings, resulting in thickened secretions in the respiratory, gastrointestinal and reproductive tracts.

the long arm of chromosome 7 that encodes the CF transmembrane conductance regulator (CFTR) (Ratjen and Doring 2003).

Pathology of CF

Mutations in the CFTR gene result in defective chloride transport, which is accompanied by decreased transport of sodium and water in the epithelial cells in the respiratory, hepatobiliary, gastrointestinal and reproductive tracts, and in the pancreas (Quinton 1990). This results in dehydration and, hence, an increase in the viscosity of secretions that are associated with luminal obstruction and scarring of various exocrine ducts (Oppenheimer and Esterly 1975). Other than in the respiratory system, the resultant clinical manifestations include pancreatic insufficiency, diabetes mellitus, azoospermia in affected men and evidence of biochemical liver abnormality in up to 80% of children (Ling et al. 1999).

The primary causes of morbidity and mortality in patients with CF, however, are bronchiectasis and obstructive pulmonary disease; the latter accounts for over 90% of deaths. Infants with CF have persistent endobronchial bacterial infections (Abman et al. 1991) which are associated with an intense inflammatory response that damages the airway and impairs local host defence mechanisms (Konstan and Berger 1993). Continuous inflammation coupled with thickened pulmonary secretions leads to airways obstruction and hyperinflation (Davis et al. 1996). Hyperinflation becomes a marked feature of the disorder leading to altered pulmonary mechanics which cause the inspiratory muscles, particularly the diaphragm, to be foreshortened prior to contraction. In such cases even a small change in breathing pattern (Bellemare and Grassino 1982) or an increase in ventilatory requirement induced by exercise could be enough to induce inspiratory muscle fatigue (Levine and Guillen 1987).

Pulmonary changes

- *Excessive mucus.* There is excess mucus production, especially in the small bronchi and bronchioles. These respiratory passages are structurally normal at birth but become blocked by mucus plugs. Lung disease in CF is also characterised by impaired mucocilary clearance of secretions.
- *Viscid mucus.* The abnormality in the mucous glands results in production of mucus with a reduced water content so that the secretions produced are very viscid and stick to the bronchial walls.
- *Infection.* The accumulated mucus provides a medium for bacterial growth and so the secretions become infected and purulent. This leads to irritation of the bronchial wall tissue, which then becomes inflamed.
- *Bronchiectasis.* Inflammation leads to weakening of the bronchial walls and dilatation occurs as in bronchiectasis.
- *Lack of development of lung tissue.* Mucus and inflammation resulting in airway obliteration inhibits the development of normal lung tissue.

Other pathological changes

Fibrosis of the pancreas causes digestive malfunction and may lead to the development of diabetes. In addition, skeletal muscle atrophy and osteoporosis are features of late disease (Elborn 2007). Intestinal obstruction may occur as a result of gallstones or faecal impaction. In newborn babies there is intestinal obstruction – known as 'meconium ileus' – because there is excess meconium (a greenish black viscid discharge from the bowel of newborn babies) which plugs the small intestine necessitating emergency surgery. Right ventricular hypertrophy occurs because of pulmonary congestion, which develops as fibrosis, and thickening of the pulmonary arterial walls takes place.

Prognosis of CF

With early diagnosis and good management, the life expectancy of patients with CF is increasing and survival may be to the fourth or fifth decade (Elborn et al. 1991). The majority, however, die before 40 years of age from respiratory failure related to pulmonary infection. Indeed, 85% of mortality occurs as a result of lung disease (Flume et al. 2009). In addition to the relentless progression of lung disease, acute exacerbations of chronic infection adversely affect the nutritional status of these patients. During the terminal phase of their life, many patients with CF enter into a vicious cycle of repeated respiratory exacerbations with evidence of deterioration in lung function measurements and declining bodyweight (Elborn et al. 1993). This evidence strongly suggests that the systemic consequences of infection and inflammation are, in part, responsible for weight loss in patients with CF. Chronic infection may also cause anorexia because of physical

factors such as increased mucus production and the anorectic effects of cytokines.

Thus, in patients with CF there may be reduced energy intake, reduced nutrient absorption as a result of maldigestion, and an increase in energy expenditure resulting from abnormal pulmonary function and sepsis (Bell et al. 1996).

Clinical features of CF

Children

At birth, the infant is normal, but symptoms of organ dysfunction can appear soon after. The presenting features vary widely.

- *Meconium ileus*. This may present in approximately 10% of infants and is caused by the abnormally viscid nature of the meconium, causing obstruction of the terminal ileum.
- *Failure to thrive and gain weight*. This results from chronic malnutrition.
- *Cough producing copious, often purulent, sputum*. Recurrent *S. aureus* infections are common and *Pseudomonas* spp. and *Burkholderia capacia* colonise the respiratory tract.
- *Dyspnoea*. This is particularly evident during an exacerbation.
- *Wheezing*. This is caused by airway obstruction as a result of inflammation and bronchospasm.
- *High level of sodium in sweat*. Sweat sodium and chloride concentrations are elevated (sweat sodium >7 mmol/L) in children under 10 years of age. The sweat test is reliable in older children and adults and is a reliable diagnostic sign.
- *Frequent, foul-smelling stools*. This is because of malabsorption and steatorrhoea (fat in the stools) because of secondary dysfunction of the exocrine pancreas.

Adolescents and adults

- *Progressive breathlessness*. This may be associated with infective exacerbations and increasing disease severity.
- *Reduced FEV_1 and deteriorating blood gases*. Pulmonary function tests deteriorate as chronic airways obstruction develops. As the disease progresses, ventilation/perfusion imbalance occurs leading to hypoxaemia and pulmonary hypertension.
- *Continued wheezing and productive cough*. This is associated with purulent sputum from which strains of *Pseudomonas* spp., *Staphylococcus* spp. or *B. capacia* may be cultured.
- *Haemoptysis*. This occurs secondary to bronchiectasis.
- *Chest radiograph*. This will show hyperinflation and bronchial wall thickening, particularly in the upper zones and bronchiectasis.
- *Finger clubbing*. This is associated with bronchiectasis.
- *Puberty delayed*. This may be delayed for both male and female patients. Most women have normal or near-normal fertility, although pregnancy may be inadvisable if pulmonary function is less than 60% of expected.
- *Infertility in males*. This occurs because of blockage of the vas deferens, which is either absent or blocked, although they can produce sperm.
- *Lung function tests* (LFTs). There is reduction of the FEV_1/FVC ratio and the FVC is grossly reduced. The RV will be increased at the expense of the VC because of air trapping and the inability of the expiratory muscles to decrease the volume of the thoracic cavity.
- *Blood gases*. Ventilation/perfusion mismatch is inevitable in CF and leads to a low PaO_2 with or without CO_2 retention. As the disease becomes severe, the arterial $PaCO_2$ may rise and a diffusion abnormality will also be apparent.
- *Auscultation*. There will be inspiratory and expiratory wheeze with added coarse crepitations.
- *X-ray*. No characteristic abnormality is seen in the early stages of the disease. If there is significant airways obstruction there may be signs of chest over-expansion (flattening of the diaphragm) and an enlarged retrosternal airspace.

Complications

- *Haemoptysis*. This is usually mild but frank haemoptysis may occur occasionally.
- *Spontaneous pneumothorax*. This may occur because of rupture of emphysematous bullae.
- *Osteoporosis*. There has been recent recognition of the high prevalence of low bone mineral density leading to osteoporosis and an increased susceptibility to fractures (Haworth et al. 1999; Elborn, 2007).
- *Liver disease*. This usually presents as biliary cirrhosis and may be associated with portal hypertension and oesophageal varices.
- *Diabetes mellitus*. This results from progressive fibrosis damaging the exocrine glands that produce insulin.
- *Deformity*. These patients often develop a barrel chest as a result of hyperinflation with use of accessory muscles of respiration. There may be evidence of a poor posture, including kyphosis and lordosis, and associated musculoskeletal pain.
- *Cor pulmonale*. This may occur in the later stages of the disease.

Social–psychological problems

The disease carries with it some unfortunate social and psychological problems. Coughing and spitting are antisocial, so people, in avoiding the patient, are unwittingly unkind. The parents may feel guilty as they are carrying the gene. They have to spend a lot of time with

the patient, which creates resentment in siblings. The patient, on reaching adolescence, may become resentful of treatment and his/her increasing inability to participate in a full social life. Clearly, this is only a brief mention of the total picture of which the physiotherapist must be aware.

Terminal features

The terminal features include respiratory failure, cyanosis, cor pulmonale and severe nutritional depletion (accelerated loss of lean body mass and fat mass).

MANAGEMENT OF CF

General principles

Paediatric and adult patients with CF should receive care from a specialist CF centre. A low-fat, high-calorie diet is recommended, supplemented with vitamins. In addition to maintaining or improving dietary status, treatment of CF is directed towards the correction of organ dysfunction (Davis et al. 1996), including pancreatic-enzyme replacement and reversal of secondary nutritional and vitamin deficiencies (Ramsey et al. 1992), although the majority of treatment is directed towards the management of abnormalities of pulmonary function. This includes clearance of lower-airway secretions (Zach and Oberwaldner 1989), treatment of persistent pulmonary infections (Turpin and Knowles 1993) and the alleviation of the symptoms of pulmonary dysfunction, especially breathlessness, and with emphasis on the importance of regular exercise.

Owing to abnormalities of pulmonary function, patients with CF ventilate excessively and ineffectively at all work levels compared with subjects with normal lung function (Cerny et al. 1982). This results in loss of functional status because aerobic exercise in CF is limited by both cardiovascular and pulmonary mechanisms. Thus, maintenance of exercise capacity in patients with CF is imperative.

Medications

Antibiotics

Antibiotics are essential and the patient will be prescribed one form or another for life. There is much evidence to suggest that aggressive intravenous antibiotic therapy in children with CF has resulted in a significantly improved survival rate (Turpin and Knowles 1993; Elborn et al. 2000). More recent investigations suggest that the use of prophylactic antibiotics is associated with a reduced requirement for additional courses of oral antibiotics and fewer hospital admissions in the first two years of life, although no effect on infant lung function has been identified (Smyth and Walters 2000). In older patients with CF who are chronically infected with *Pseudomonas* spp., no differences in lung function or mortality rate were identified between 2 groups of 30 patients who were given either elective or symptomatic antibiotic therapy over a 3-year period (Elborn et al. 2000). There is no sound evidence for or against the use of chronic inhaled antibiotics for the treatment of *P. aeruginosa* in this population (Flume et al. 2007).

Bronchodilators

These may be useful when there is airways obstruction which is reversible. During an acute exacerbation, a nebuliser may be used at home.

Oxygen therapy

Oxygen therapy may be appropriate in the terminal stages when there is persistent hypoxaemia. Bubble-through humidification should be avoided because of an increased risk of infection (Bott et al. 2009).

Mucolytic agents

Because lung disease in CF is characterised by impaired mucociliary clearance, recurrent bronchial infections and inflammation, methods that may enhance the removal of retained bronchial secretions may act to lessen the destructive inflammatory process in the airways (Solomon et al. 1996).

Nebulised hypertonic saline has been shown to increase mucociliary clearance immediately after administration. This may have a long-term beneficial effect, although the effect on pulmonary function tests, quality of life and frequency of exacerbations remains unclear (Wark and McDonald 2000). Care must be taken as hypertonic saline may induce bronchospasm. Recombinant human deoxyribonuclease (rhDNase) is currently used to treat pulmonary disease in patients with CF by facilitating protein breakdown in pulmonary secretions, thereby aiding expectoration (Christopher et al. 1999).

PHYSIOTHERAPY IN CF

Key point

Regular exercise and frequent expectoration of sputum for life is an essential part of the treatment of the pulmonary features of CF, and airways clearance is currently recommended as soon as the diagnosis is made. The treatment approach should be adapted to changes in the patient's lifestyle as he/she matures and as the disease progresses.

The aims of physiotherapy are:

- to reduce bronchospasm and to clear the lung fields;
- to encourage activities for maintaining physical fitness/increase exercise tolerance;
- to train postural awareness and relaxation;
- to educate the patient in self-management.

Clearing lung fields

This is the cornerstone of management of patients with CF because the primary causes of morbidity and mortality are bronchiectasis and obstructive pulmonary disease – the latter accounts for over 90% of deaths. It is important that the parents and the rest of the family be involved in the treatment of the child at a young age so that physiotherapy can become an accepted routine.

Chest clearance techniques in the infant include the use of postural drainage, percussion and shaking. The optimal position is usually sitting in the upright position, as the infant will spend much of their time in supine lying. Prior to these techniques it is useful to have an active game with a child so that he or she laughs, producing deeper respiration and then becoming breathless. This is required twice a day, every day, even when the patient is apparently well, as there is some evidence that inflammation and infection exist during infancy (Konstan et al. 1994).

Key point

During exacerbations, or when the child has an upper respiratory tract infection, these treatment sessions may have to be increased up to as much as six times a day.

A baby may be positioned on a pillow on the knee of either the physiotherapist or parent. The physiotherapist has to identify the most effective position for each individual patient and relate the treatment to the home situation. As the child grows, there comes a stage where it may be necessary to position for drainage using cushions if secretions are evident in the lower zones. A tipping frame that supports the patient totally is more comfortable for draining the anterior and lateral segments of the lower lobes, and middle lobe or lingula. Adolescents and adult patients may have blocks made so that their own bed may be tipped.

In addition to postural drainage and manual techniques, the use of a PEP mask and the Flutter device can be used to facilitate the clearance of secretions by enhancing expiratory airflow (Freitag et al. 1989). Infants tend to swallow their secretions so for children under 3 years of age and babies the sputum needs to be cleared by a tissue. Disposal of infected sputum should be discussed with parents, relatives or patient, as it is essential to avoid reinfection.

Recent research has highlighted that there is no statistically significant difference between ACBT, autogenic drainage, PEP and Flutter (in the sitting position) over one year when examining FEV_1, quality of life or body mass index (BMI) (Pryor et al. 2010). Again, NIV and IPPB may be used as an adjunct during chest clearance physiotherapy to minimise fatigue of the respiratory muscles.

Some form of humidification is useful to reduce the viscosity of the mucus and may be applied using a mouthpiece and nebuliser. Ultrasonic nebulisers have been shown to be preferred by CF patients (Thomas et al. 1991). For babies and children, a mask may be necessary. Saline solution may be used as a mucolytic agent. For home use, patients may have an electric compressor with a nebuliser.

As soon as the patient is old enough he/she should be encouraged to become involved in the treatment. The person should also be encouraged to expectorate the secretions and not to swallow the sputum, as this may cause an exacerbation of the abdominal symptoms. During exacerbations of infection, patients may be admitted to hospital where intensive physiotherapy is essential.

A community physiotherapist should visit the patient's home at regular intervals and will become very well known to the involved family. The patient has to attend regular follow-up clinics to be seen by a chest specialist, as well as being taken care of by the GP. It is also essential that the patient be seen regularly by the physiotherapist so that treatment can be updated and problems discussed.

Maintenance of physical fitness/ increasing exercise tolerance

Aerobic fitness in both children and young adults with CF can be improved by aerobic exercise training (Kaplan et al. 1991). Higher levels of aerobic fitness in patients with CF have been shown to be associated with a significantly improved length of survival (Nixon et al. 1992). Exercise has also been shown to decrease breathlessness (O'Neill et al. 1987), improve quality of life (de Jong et al. 1997) and also offers an important contribution to sputum expectoration (Sahl et al. 1989).

Parents of an infant with CF should be encouraged to treat the child as normally as possible so that the child joins in physical activities at school and with friends at weekends. It is helpful for the parents to meet the child's teachers so that they know to encourage the child to participate fully in school life.

Adult patients may benefit from regular swimming or short sessions of jogging, and, if possible, should be encouraged to attend the hospital or clinic when clinically stable to have their baseline exercise capacity measured. This will allow the physiotherapist to recommend a level of exercise to provide an appropriate training effect. It is also important to establish the goals of an exercise programme and to tailor the programme to the patient's level of fitness and disease severity.

Terminal stages

The advanced stages of CF are characterised by repeated exacerbations, reduced mobility and, eventually, respiratory failure. As the blood gases deteriorate and the $PaCO_2$ begins to rise as a result of ventilatory failure, NIPPV may be indicated, particularly if awaiting heart/lung transplantation. This is a distressing time for the patient, the family and the multi-disciplinary team, as the patient becomes very ill and recognises that death is imminent. The family will require a great deal of emotional support from the multi-disciplinary team.

The principal theme is to keep the patient as comfortable as possible, which usually means sedation using morphine or one of its derivatives to relieve the sensation of breathlessness and reduce anxiety. It is inappropriate to discontinue contact with the physiotherapist, even though active treatment is no longer effective. The aim in the terminal stages of the disease is positioning of the patient in high side lying or forward lean sitting to make the person as comfortable as possible and to assist with the clearance of secretions from the upper airways if these become uncomfortable and distressing for the patient. Nasopharangeal suction is not indicated in the terminal stages of the disease.

Surgery

The maintenance of mobility is essential for those patients who are waiting for heart/lung transplantation if this is indicated. Although exercise tolerance at this stage of the illness may be restricted, the physiotherapist should maintain muscle strength and cardiovascular function. Postoperatively, rehabilitation should be extensive in order to maximise function and quality of life.

RESTRICTIVE PULMONARY DISEASES

Pneumonia

ABC Definition

Pneumonia is an acute inflammation of the lung tissue – the alveoli and adjacent airways.

Classification

Pneumonia may be classified in many ways, for example:

- according to its anatomical distribution (e.g. lobar, which is confined to one lobe, or bronchopneumonia, which is a more widespread, patchy infection);
- according to its microbiological cause.

In clinical terms, it may also be defined as:

- according to where the infection is acquired (i.e. in the community or in hospital);
- according to whether the patient is immunocompromised (e.g. by AIDS).

All of these factors may determine the outcome of the disease, the likely causative factors and the clinical features of the disease.

Community-acquired pneumonia

Infection is acquired through the inhalation of droplets containing the specific microorganism and the individual is unable to overcome the infection through the natural pulmonary defence mechanism.

Community-acquired pneumonia is a common pulmonary disease and may be responsible for over 1 million hospital admissions a year in the UK. The microbiological cause does, however, tend to affect different age groups.

- *Streptococcus pneumoniae* pneumonia is the most prevalent community-acquired pneumonia and affects all age groups.
- *Mycoplasma pneumoniae* pneumonia usually occurs in adolescents and young adults.
- Influenza, parainfluenza, measles and adenovirus pneumonias are more common in children and the elderly.
- Chickenpox pneumonia occurs in adults.
- Respiratory syncytial virus (RSV) is an important cause of morbidity and mortality in children under two years of age.
- *Legionella pneumophilia* infection (Legionnaire's disease) may occur in all age groups but is more common in men than women. It thrives in warm water and frequently contaminates badly maintained air-conditioning systems.
- *Haemophilus influenzae* may produce bronchopneumonia in those with pre-existing pulmonary disease (e.g. chronic bronchitis). It is therefore more common in the elderly.
- *Staphylococcus pyogenes, Klebsiella pneumoniae* and *M. pneumoniae* infections are rare in healthy individuals but may commonly complicate viral pneumonia.

Predisposing factors

These are: winter or spring; overcrowding where bacteria and viruses are easily transmitted; alcoholism; smoking (cigarette smoke and alcohol depress ciliary function and phagocytosis); atmospheric pollution; lower socioeconomic groups; pre-existing respiratory disease. The disease may also occur secondary to impaired consciousness and malnutrition.

Pathological changes

The invading organism causes inflammation in the bronchioles and alveoli. The exudate spreads into neighbouring alveoli to provide a medium for rapid spread of bacteria. The alveoli become filled with red blood cells, leucocytes, macrophages and fibrin (red hepatisation), and there is congestion throughout the lobe. The overlying pleural surface is inflamed and a pleural effusion may develop. Resolution occurs when the leucocytes engulf the bacteria and macrophages clear the debris by phagocytosis (grey hepatisation).

In lobular or bronchopneumonia the inflammation is scattered irregularly in the lungs whereas in lobar pneumonia the inflammation is spread throughout but contained within one entire lobe. Without treatment, resolution occurs by liquefaction of the consolidation, which is then expelled by coughing.

Clinical features

The onset may be sudden (lobar pneumonia) or gradual (bronchopneumonia or lobular pneumonia), and is associated with malaise, pyrexia (temperature often >40°C), rigors, vomiting, confusion caused by hypoxaemia (especially in the elderly) and tachycardia.

- *Cough*. This is dry at first but after a few days purulent sputum is produced.
- *Breathlessness*. Blood passing through the affected alveolar membranes is inadequately oxygenated so that the PaO_2 falls. Hyperventilation cannot compensate for this hypoxaemia because blood passing through the normal lung tissue is almost saturated. The inflammation that occurs makes the lung stiff and compliance is reduced with the result that the effort of breathing is increased. Respiration therefore becomes rapid and shallow.
- *Pain*. If inflammation spreads to the pleura there is a sharp pain aggravated by taking a deep breath or by coughing.
- *Radiograph*. Consolidation can be seen as an opacity, especially in lobar pneumonia. There may also be evidence of a pleural effusion.
- *Auscultation*. Bronchial breathing can be heard (especially in lobar pneumonia) because the consolidated lung tissue conducts the sounds of air movement in the trachea. Whispering pectoriloquy and increased vocal resonance can be heard. Wheeze may be evident if bronchospasm is present.

Investigations

- *Haematology*. This may reveal a raised white blood cell count.
- *Biochemistry*. Arterial blood gases should be measured to reveal the extent of arterial hypoxaemia.
- *Microbiology*. Sputum should be sent for Gram staining to identify the causative organism (e.g. *S. pneumoniae*) and to identify which antimicrobial agents are sensitive to the organism.
- *Pleural aspiration for culture*. This should be considered if the pneumonia is complicated by a pleural effusion.

Prognosis

The outcome depends on predisposing factors, the virulence of the bacteria, and the age and general fitness of the patient. Improvement starts within 3–4 days of the patient having antibiotics and within 10 days the sputum should be less in quantity and mucoid in nature – by which time the patient begins to feel better. In an otherwise fit person, the radiograph should be clear in six weeks.

Generally, lobar pneumonia resolves and the patient recovers, particularly in people who are generally fit and are between the ages of 20 and 50 years. Bronchopneumonia is more serious, is often secondary to other problems and may be the terminal illness in patients who are elderly. The disease may be fatal in the very young because the secretions readily block the narrow, underdeveloped airways.

Management

- Antibiotics are given to control infection. Specimens of sputum should be sent for culture and sensitivity as soon as possible to confirm or alter antibiotic therapy.
- Adequate fluids must be taken to ensure fluid balance.
- Analgesics are given to relieve pleuritic pain.
- Oxygen therapy may be necessary and blood gases should be monitored regularly.
- Bed rest at home may be sufficient, but an acutely ill patient should be admitted to hospital.

Complications

Possible complications of pneumonia are:

- spread to other lung areas;
- delayed resolution because of the wrong antibiotic being given, poor compliance with medication or bronchial obstruction (e.g. as a result of carcinoma);
- pleural disease resulting in pleural effusion or empyema – this will require an intercostal tube and drainage (possibly surgical drainage) and antibiotics if an empyema is evident;
- lung abscess – this will cause a swinging pyrexia and will require antibiotics;
- cardiac failure;
- septicaemia;

- pneumococcal meningitis;
- pneumothorax – this is particularly associated with *S. aureus* pneumonia and will require intercostal tube drainage;
- deconditioning caused by malaise.

Physiotherapy in pneumonia

Physiotherapy is indicated when the inflammation has begun to resolve. The aims of treatment are:

- to reduce bronchospasm (if present) and to clear lung fields of secretions;
- to gain full re-expansion of the lungs;
- to regain exercise tolerance and fitness.

Clearing lung fields

Sputum clearance techniques may be indicated if there is evidence of excessive secretions; however, routine chest physiotherapy is not always indicated (Yang et al. 2010). Humidification may be necessary to moisten secretions. The method will vary according to the severity of the illness and may be by nebuliser or IPPB. Clapping, shaking and breathing exercises may all be necessary in a postural drainage position appropriate to the area of the lung affected if there is evidence of excess secretions. Sometimes suction is required for the very ill patient who cannot cough or expectorate. If there is an underlying bronchospasm then a bronchodilator may be given.

Re-expansion of the lungs to improve ventilation

Positioning should be used to increase ventilation to the affected area.

Exercise tolerance and fitness

As soon as possible, the patient should be mobilised and start walking short distances which are progressively increased in length.

Pleurisy

ABC Definition

Pleurisy is a process whereby inflammation occurs on the visceral and parietal pleura which come into direct contact with each other to cause pain.

Aetiology and pathological changes

Pleurisy may also be secondary to tuberculosis or lobar pneumonia. Infection or irritation of the pleura causes inflammation and vascular congestion. A fibrinous exudate is formed within the pleural cavity and the pleural surfaces are roughened. The inflammation may resolve or develop into a pleural effusion (see below), depending upon any underlying conditions. When resolution occurs, fibrin laid down within the exudate tends to form adhesions between the two layers of the pleura.

The causes of pleurisy are:

- viral infection (the most common cause);
- pulmonary infarction;
- bronchial carcinoma;
- pneumonia;
- autoimmune rheumatic diseases (e.g. systemic lupus erythematosus, rheumatoid arthritis).

Clinical features

- *Pleuritic pain*. This is a result of the movement of the inflamed pleural membranes during inspiration. The pain is sharp (knife-like), severe and related to movement of the chest (e.g. deep inspiration or coughing). It is usually well-localised to the area of the chest under which the pleural irritation lies. Irritation of the diaphragmatic pleura, however, causes pain sensation via the phrenic nerve and this is often referred to the tip of the shoulder.
- *Pleural rub*. There is a creaking or grating sound heard through a stethoscope on both inspiration and expiration. It is localised to the affected area. This disappears if an effusion develops.
- *Cough*. Coughing may be present if respiratory infection is the cause.
- *Radiograph*. The diaphragm may be raised on the affected side.
- *Other clinical signs*. Tachycardia and pyrexia may be present depending on associated conditions.

Investigation and treatment

Haematology shows a high white cell count if infection is present. Identification and treatment of any underlying conditions is essential. Analgesics are given to relieve pain, and sedative linctus possibly reduces coughing. Rest is important to allow the inflammation to subside and to minimise the pain.

Physiotherapy in pleurisy

Physiotherapy is usually inappropriate in the early stages. During the recovery stage, however, the aims are:

- to regain full thoracic expansion;
- to regain exercise tolerance;
- to mobilise the thorax.

General deep breathing exercises and early mobility exercises, such as sitting with trunk bending side to side and walking are important to regain mobility of the thorax and thoracic spine, as well as increasing thoracic expansion.

Pleural effusion

ABC Definition

Pleural effusion is an excessive accumulation of fluid in the pleural cavity.

Aetiology

Pleural effusion is often secondary to conditions such as:

- malignancy of the lungs or bronchi;
- pneumonia;
- tuberculosis;
- pulmonary infarction;
- bronchiectasis;
- lung abscess;
- blockage of lymph vessels;
- rupture of blood vessels;
- left ventricular failure.

Pathological changes

Fluid accumulates in the pleural cavity, the composition of which varies according to the underlying cause. The fluid may be reabsorbed naturally or removed by surgical intervention. As the pleural layers come together they may become adherent owing to organisation of fibrin if the fluid contains plasma proteins.

Fluid may accumulate in the pleural cavity as transudate or exudate. *Transudate* occurs when there is an increased pulmonary capillary pressure (as in congestive cardiac failure) or a decreased osmotic pressure (as in hypoproteinaemia associated with malnutrition) across the pleural membrane. *Exudate* occurs when there is inflammation resulting in increased permeability of capillaries and visceral pleura together with impaired lymphatic reabsorption (as in pneumonia or malignancy).

Exudate is cloudy with a high protein content, in contrast to transudate which is clear with a low protein content. Consequently, exudate tends to become consolidated whereas transudate can be reabsorbed if the underlying condition is treated.

Clinical features

- *Breathlessness*. The pressure of fluid reduces lung expansion.
- *Cyanosis*. This may be present in a large effusion.
- *Pyrexia*. This is usually associated with infection.
- *Lethargy*. The person complains of a lack of energy.
- *Pain*. The person complains of pain.
- *Thorax*. Thoracic expansion is restricted on the affected side.

Investigations and treatment

A fluid level can be identified on X-ray. There is a stony dullness on percussion over the fluid. Breath sounds are absent over the effusion (>500 mL of fluid), although bronchial breathing may be heard just above the effusion. Small effusions (220–500 mL) are revealed by chest radiography.

If the fluid does not become reabsorbed naturally, then it should be aspirated (drained surgically). Oxygen therapy may be necessary.

Physiotherapy in pleural effusion

The aims of physiotherapy are:

- to prevent the formation of disabling adhesions between the two layers of pleura;
- to obtain full expansion of the affected lung;
- to increase ventilation of the lungs;
- to increase exercise tolerance following immobility.

The treatment must be modified to take into account any underlying condition. Following aspiration, breathing exercises should be given to encourage localised expansion of the affected side.

If the patient has difficulty in localising the expansion, it may be helpful to lie on the unaffected side over a firm pillow to help stretch the affected side. Breathing exercises may also be practised in this position several times a day. When the patient has regained lung expansion, the treatment programme should be expanded to include mobilisation of the patient and to increase exercise tolerance.

Some malignant pleural effusions may require a pleurodesis – the insertion of a powder such as tetracycline into the pleural cavity

Empyema

ABC Definition

Empyema is a collection of pus in the pleural cavity.

Aetiology

The condition of empyema usually arises secondary to pre-existing lung disease, such as bacterial pneumonia, tuberculosis, lung abscess or bronchiectasis. The most common cause is direct spread of infection into the pleural space in a patient with pneumonia caused by *S. pneumoniae*. It may also arise as a result of a stab wound or as a complication of thoracic surgery.

Pathological changes

Infected material enters the pleural cavity. Both layers of pleura become covered in thick inflammatory exudate

within which fibrous tissue is laid down. As this fibrous tissue contracts it acts as a physical barrier to lung expansion. The pressure of the fibrous tissue on the pus may cause rupture of the pleura and lung tissue and the pus may then be coughed up. Alternatively, an abscess may form. Healing occurs when the pus has been surgically removed or the infection has been overcome by the patient's natural antibodies, assisted by antibiotics. The layers of the pleura come together and adhesion formation may take place, restricting lung movement.

Clinical features

These include:

- pyrexia;
- lassitude and loss of weight;
- tachycardia;
- dyspnoea;
- pleuritic pain severe at first then decreasing in severity;
- diminished thoracic movements.

There may be a history of pneumonia or other associated condition.

Investigations and treatment

On X-ray, the empyema can be seen as a D-shaped shadow, the straight line of the D being on the lung surface. Pleural aspiration or tap will confirm the diagnosis as the sample is often thick and purulent, and may be foul-smelling. Pleural fluid cytology will reveal an exudate with pus cells and organisms.

Antibiotics are given to combat infection. Aspiration through a needle inserted into the cavity may remove sufficient pus to relieve the condition, but continuous underwater drainage may be necessary. Rib resection may be indicated if the effusion is very thick or loculated.

If the condition results in fibrosis of the pleura which severely limits lung expansion, then a rib resection may be performed and the pleura stripped off the lung (decortication).

The prognosis depends on the cause, but untreated infection can make the patient very ill from toxins absorbed into the bloodstream (toxaemia).

Physiotherapy in empyema

The aims are:

- to minimise adhesion formation within the pleura;
- to regain full lung expansion;
- to clear the lung fields;
- to maintain good posture and thoracic mobility;
- to improve exercise tolerance.

If the patient has a chest drainage tube inserted, the physiotherapy is similar to that following a thoracotomy.

Good posture should be encouraged whenever physiotherapy is being given. The tendency is for the patient to protect the affected side, by side-flexing to that side. Therefore, the patient should be taught to take weight evenly on both buttocks, to keep the shoulders level and to practise stretching to the opposite side from the lesion, as well as stretching backwards.

Breathing exercises to expand the lung on the affected side need to be carried out three or four times daily. Postural drainage may be indicated to clear the lungs if secretions are accumulating.

As the patient recovers, general leg, arm and trunk exercises should be taught. Walking should begin as soon as possible with breathing control practised over progressively longer distances, and going down (then up) stairs incorporated. As the patient regains lung expansion, the treatment programme should be expanded to increase exercise tolerance.

Pneumothorax

ABC Definition

Air collects between the visceral and parietal pleura. Air in the pleural space will allow the lung to move away from the chest wall and the lung will partially deflate.

There are two types of pneumothorax, spontaneous (which may be secondary to an underlying disease) and traumatic.

Spontaneous pneumothorax

This can occur at any age but is most common in young men (a ratio of 6 : 1) who are otherwise apparently healthy. It may also be associated with emphysema and chronic bronchitis in men over 50 years of age, result from other underlying disease or be associated with mechanical ventilation. These spontaneous causes may be summarised as:

- airflow limitation caused by asthma or bullous emphysema;
- positive pressure ventilation, particularly with the use of positive end-expiratory pressure (PEEP);
- infections (e.g. Staphylococcal pneumonia, tuberculosis);
- CF;
- Marfan's syndrome.

Traumatic pneumothorax

A traumatic pneumothorax may be caused by:

- penetrating injury to the chest (e.g. by stab or bullet wound);

- non-penetrating injury to the chest wall (e.g. impact of a road traffic accident involving the chest);
- during the insertion of an intravenous (e.g. subclavian) line;
- during surgery to the chest wall;
- during pleural aspiration or biopsy.

When the chest wall remains intact, the condition is termed a *closed pneumothorax*, but if the chest wall is opened following the trauma the term used is *open pneumothorax*. In the presence of an open wound, the emergency treatment is the application of a large dressing pad over the chest wall.

Pathological changes

As air escapes into the pleural cavity and reduces the subatmospheric pressure (i.e. less negative) the lung collapses. The hole in the pleura closes, the air becomes absorbed and the lung gradually re-expands. Sometimes this does not happen and the hole in the pleura becomes like a valve. Air then enters the pleural cavity on inspiration but cannot escape during expiration. The lung remains collapsed and, as air accumulates in the pleural cavity and the pressure increases, there is displacement of the heart together with compression of the other lung and great vessels. This is termed a *tension pneumothorax* and has to be treated as an emergency by needle aspiration and thereafter by insertion of a drain connected to an underwater seal.

Clinical features

The onset is often sudden with severe chest pain and progressive breathlessness. There is diminished chest movement unilaterally and an absence of breath sounds often over the apex of the affected side.

Other clinical features may be related to the underlying pathology (e.g. emphysema). In a patient with known lung disease a pneumothorax should always be considered if the patient becomes more breathless for no apparent reason.

Subcutaneous emphysema may develop at the time of the pleural air leak or following the insertion of an intercostal drain when air may track into the subcutaneous tissues. Subcutaneous air results in a crackling sensation on palpation.

Investigations and treatment

The chest X-ray shows absence of lung markings and the edge of the collapsed lung can be seen. This will confirm the diagnosis. Inspiratory and expiratory radiographs will help define the visceral pleura where there is a small pneumothorax.

A small pneumothorax requires no treatment apart from a few days bed rest until it resolves. A large pneumothorax (i.e. more than 25% of the pleural space is filled with air) is treated by needle aspiration or by an intercostal drain which connects the pleural cavity to a drainage bottle creating an underwater seal. The drain is removed when there are no more bubbles in the drainage bottle – indicating that the pleural cavity is free of air.

Surgery is indicated for a recurrent pneumothorax. Pleurodesis comprises the insertion of a powder into the pleural cavity. This acts as an irritant to the pleural surfaces causing them to adhere to each other. Pleurectomy is the removal of the parietal pleura from the chest wall leaving a raw surface to which the visceral layer sticks. A hole in the visceral pleura may have to be stitched.

Physiotherapy in pneumothorax

A patient who has an underwater drainage system requires expansion breathing exercises to re-expand the lung. Also, full-range shoulder movements are necessary to maintain shoulder, shoulder girdle and thoracic mobility. This treatment is generally given 3–4 times daily until the drain is removed.

Following pleurodesis, expansion breathing exercises are essential to ensure that when the adhesions form between the layers of the pleura the lung is fully expanded. The patient must be taught to practise expansion breathing exercises so that thoracic mobility is maintained, otherwise there may be sharp pleuritic pain if the intrapleural adhesions become too contracted. If the lung does not re-expand within 36 hours then a second operation is required. Physiotherapy after a pleurectomy follows the same principles as for any thoracotomy.

Acute respiratory distress syndrome

A catastrophic event can, either directly or indirectly, cause damage to the pulmonary epithelium or the alveolar capillary membrane. Acute respiratory distress syndrome (ARDS) is, therefore, the respiratory manifestation of a systemic condition which appears within 12–48 hours after the initial triggering event.

ABC Definition

ARDS is a severe and acute form of respiratory failure precipitated by a wide range of catastrophic events, including shock, septicaemia, major trauma, or aspiration or inhalation of noxious substances (Bernard et al. 1994).

Examples of triggering events are: pulmonary aspiration; severe burns with or without inhalation injury; disseminated intravascular coagulation (a coagulation defect caused by clotting abnormalities); cardiopulmonary bypass; severe trauma; massive blood transfusion;

near-drowning; pre-eclampsia; septicaemia; amniotic fluid embolism (substances entering the maternal circulation usually following a vigorous labour); pancreatitis; and fat embolism resulting from the fracture of (long) bones.

Pathological changes

Activated neutrophils are thought to release a number of vasoactive mediators that damage the integrity of the alveolar membrane. As a result of increased endothelial permeability within the alveolar–capillary membrane, fluid moves from the pulmonary capillaries into the gas exchange areas of the lung. This results in alveolar oedema and extravasation of inflammatory cells. The pulmonary oedema is therefore said to be non-cardiogenic because there is normal hydrostatic pressure in the pulmonary vasculature (unlike left ventricular failure when this is raised). As this acute phase progresses, there is increasing congestion in the capillaries. The loss of functioning alveoli results in severe hypoxaemia and respiratory failure.

Clinical features

The defining features of ARDS are:

- severe refractory (resistant to treatment) hypoxaemia;
- the presence of pulmonary oedema with normal hydrostatic pressure in the pulmonary vasculature;
- the appearance of diffuse bilateral pulmonary infiltrates on chest X-ray;
- a falling pulmonary compliance (<50 mL/cmH_2O).

Increasing breathlessness is evident which, left untreated, may lead to acute tachypnoea (>20 breaths per minute). There is evidence of the appearance of diffuse bilateral pulmonary infiltrates on chest X-ray, and widespread wheezes and crackles on auscultation. Despite oxygen, the disease usually progresses to a state of severe respiratory failure, which requires the support of mechanical ventilation. The lungs become progressively stiffer and adequate oxygenation and ventilation becomes more difficult.

Investigations and treatment

On blood gas analysis the PaO_2 is reduced to critical levels. If this is not corrected the $PaCO_2$ may begin to rise.

Any underlying pathological cause is treated. Adequate ventilatory support will be necessary, which may include high inspired oxygen, IPPV and the application of PEEP to restore adequate function by allowing for the recruitment of hypoventilated alveoli. This will result in an improved PaO_2. High levels of oxygen may be used initially to reduce a dangerous hypoxaemia, but as oxygen is toxic at high concentrations (causing further damage to the alveolar membrane) this should be reduced to a level that will give an adequate PaO_2. Although the application of PEEP in this condition has been standard practice for over two decades, over-distension of lung tissue may result in baro- and volu-trauma with the development of pneumothorax and subcutaneous emphysema (Montgomery et al. 1985).

Complications

Nosocominal pneumonitis is a common complication in patients with ARDS on prolonged mechanical ventilation and is directly related to oropharyngeal colonisation of Gram-negative bacilli – the stomach being one of the possible reservoirs of these microorganisms (Driks et al. 1987). The loss of mucosal integrity and clearance mechanisms are predisposing factors which lead to secondary infection and may contribute to worsening gas exchange.

Hence, adequate removal of retained bronchial secretions by chest physiotherapy techniques is an integral part of the management of these patients.

Prognosis

A substantial number of studies have now confirmed that the primary cause of death in patients with ARDS is not the inability to oxygenate arterial blood adequately but rather the result of the development of multiple organ dysfunction and failure (MOF) caused by poor tissue oxygen extraction and altered tissue bloodflow (Montgomery et al. 1985; Fowler and Goldman 1990). These manifestations are associated with a high mortality rate. Approximately 30–60% of patients with ARDS die, despite increasing awareness of the mechanisms of acute injury and the introduction of novel forms of therapy and support (Bernard et al. 1994).

Physiotherapy in ARDS

The aims of physiotherapy are:

- removal of retained secretions if these are present;
- passive/active movements in ventilated patients and ambulation as soon as this is feasible.

Chest physiotherapy involves four principal manoeuvers: positioning to enhance removal of secretions and to improve gas exchange; manual hyperinflation; endotracheal suctioning; and manual techniques (which include shakings and vibrations).

Passive and active exercises need to be performed regularly while the patient's mobility remains restricted during the critical stages of their disease, in order to maintain the mobility of joints and the extensibility of the soft tissues (e.g. the muscles, tendons and ligaments). When possible, the patient should be mobilised.

Fibrosing alveolitis

ABC Definition

In fibrosing alveolitis the alveolar walls become thickened, with an increase in type II pneumocytes and macrophages. As the disease progresses the alveolar walls fibrose and fibrosis spreads to the lung parenchyma.

The aetiology of fibrosing alveolitis is unknown (in most cases), although in some cases there is evidence of a pre-existing specific disease. These may be autoimmune (e.g. rheumatoid arthritis, systemic sclerosis) or gastrointestinal (e.g. chronic active hepatitis, ulcerative colitis).

Clinical features

- *Dyspnoea*. There is an insidious onset of breathlessness often accompanied either by a dry unproductive cough or with little clear sputum.
- *Auscultation*. A typical feature is mid-to-late inspiratory crackles, which are said to be 'metallic' in nature.
- *Finger clubbing*. As the disease progresses most patients develop gross finger clubbing.
- *Cyanosis*. This is caused by impaired gas exchange.

Investigations and treatment

Blood gases reveal a progressive hypoxaemia with hypercapnia evident late in the disease. The chest radiograph will first show a ground-glass appearance, mainly in the lower zones; as the disease progresses, this becomes discrete and nodular. Transbronchial or open lung biopsy shows interstitial and alveolar fibrosis. Lung function tests reveal a restrictive deficit with reduced gas transfer factor.

Chronic forms of fibrosing alveolitis are treated with high doses of oral corticosteroids, although the value of these has been recently investigated and shown to be largely ineffective. Other immunosuppressive drugs (e.g. cyclophosphamide) are sometimes used if the alveolitis is associated with an autoimmune disease. Long-term oxygen therapy (LTOT) should be considered to correct hypoxaemia which leads to pulmonary hypertension and the development of cor pulmonale. Heart–lung transplantation may be considered in younger patients.

The median survival time of patients with fibrosing alveolitis is less than five years, although some patients may live for much longer. In general, the earlier the onset of the disease, the worse the prognosis.

Physiotherapy in fibrosing alveolitis

Physiotherapy should be directed at teaching breathing control and maintaining exercise tolerance as the patient becomes deconditioned as a result of fatigue and shortness of breath.

OTHER PULMONARY DISEASES

Lung abscess

ABC Definition

A lung abscess is the localised formation of pus, usually surrounded by a fibrous capsule, within lung tissue.

Antibiotics and improved anaesthesia have reduced the incidence of lung abscess and the condition now tends to occur secondary to bronchial carcinoma, particularly in patients who are over 40 years.

Aetiology

A variety of bacteria may enter the lungs by one of the following routes:

- through the air passages because of bronchopneumonia or following inhalation of a foreign body;
- through the open chest wall following a wound from a knife stab or bullet;
- from the bloodstream;
- bronchial carcinoma – an abscess forms where secretions accumulate distal to the tumour.

Pathological changes

The invading organisms cause inflammation of the lung tissue. At the centre of the area there is necrosis of lung tissue with liquefaction and suppuration. The area becomes distended and fibroblasts lay down fibrous tissue around the area until there is complete encapsulation. The capsule contracts and the abscess bursts, resulting in the production of foul-smelling sputum. Sometimes the pus drains into the pleura, causing empyema, and if drainage spills into adjacent lung tissue there is a danger of bronchiectasis. Toxins from the pus can be absorbed into the bloodstream and there is then a danger of septicaemia. Healing occurs with the formation of a fibrous scar.

Clinical features

There is malaise, fever, cough and dyspnoea. The cough is irritable and unproductive at first but later it is productive of foul-smelling sputum accompanied by a bad taste in the mouth. The cough may be painful if the pleura are

inflamed. Haemoptysis and halitosis are further features. Finger clubbing may become evident if the abscess becomes chronic.

Investigations and treatment

Blood analysis may reveal an increased white cell count. Cultures of sputum or lung aspirate will reveal the organism. A chest X-ray will show an area of cavitation within the lung tissue, which may contain an air-fluid level.

Most lung abscesses will respond to large intravenous doses of antibiotics to which the organism is sensitive. Drainage of the abscess may be necessary via needle aspiration or a thoracotomy. If there is an endobronchial obstruction caused by a foreign body, removal is essential.

Physiotherapy for a lung abscess

The site of the abscess is ascertained on the radiograph and the patient is positioned accurately for 10–15 minutes every 4 hours. Shaking is applied to the chest wall and breathing exercises are taught to regain breath control after coughing. Deep inspiration should not be encouraged because the increase in negative pressure may move the pus through healthy lung tissue.

It is important to adjust the patient's position to obtain maximum effective drainage and to ensure that precautions are taken to avoid any danger of cross-infection.

Pulmonary tuberculosis

ABC Definition

In pulmonary tuberculosis (TB) the bacillus *Mycobacterium tuberculosis* causes irritation of the mucous lining in the bronchioles or alveoli, and inflammatory changes take place.

In 1882, Robert Koch isolated the tubercle bacillus, of which there are two types: one human and one bovine. Since then, the disease has been controlled by inoculation, mass radiography, drugs and pasteurised milk; however, TB is on the rise again, particularly in areas with a high incidence of HIV/AIDS and poverty.

! Key point

The lungs are not the only tissues to be affected by TB. Types of TB other than pulmonary are: acute miliary (the blood is affected, with spread of the disease to spleen, liver, kidneys, meninges and lymph nodes); surgical TB; bone or joint TB; lupus vulgaris (the skin is affected); and TB adenoma (the lymph nodes are affected).

Aetiology and prevalence

Mycobacterium tuberculosis is spread by droplets during coughing and sneezing, so that a person can be infected from a patient's sputum. The bacillus may be restrained by the immune system, but never destroyed and may lay dormant for a number of years. It may then be reactivated in 5–10% of carriers.

TB is common worldwide and its incidence has increased in the UK in recent years. The disease is still very common in Africa and Asia. In Europe, the age group most commonly affected is middle age, but it also often occurs in elderly men. In the UK, the disease occurs mainly in the immigrant population from Ireland, Asia and the West Indies.

Predisposing factors are the environment, poor hygiene, overcrowding, lower socioeconomic groups, malnutrition, smoking and alcoholism. Other factors are diseases such as HIV/AIDS, diabetes mellitus, congenital heart disorder, leukaemia, Hodgkin's disease, long-term corticosteroids or immunosuppressive drugs.

Pathology

The bacilli are ingested by leucocytes and then absorbed by macrophages. More leucocytes form a barrier around this collection of cells and the complete mass is known as a 'tuberculous follicle'. The centre of the area undergoes necrosis and becomes soft and cheesy in consistency, the process being known as 'caseation'. This material may be moved into a bronchus and coughed up leaving a cavity behind. Fibroblasts lay down a capsule around the tubercle in which calcium salts become deposited and healing takes place.

Cavity formation and calcification are the features of TB, with the calcified lesion remaining a potential source of infection. The bacillus may be reactivated and cause *post-primary pulmonary TB*. The danger then is that the disease may spread to other areas of the lungs including the pleura and through the bloodstream to other parts of the body.

Clinical features

These are:

- malaise, lassitude and irritability;
- loss of appetite and loss of weight;
- pyrexia and tachycardia;
- night sweats;
- productive cough – the bacillus can be cultured from the sputum;
- haemoptysis;
- diminished respiratory movements with possibly some dyspnoea;
- pain if there is pleural involvement.

Investigations

The chest X-ray shows cavity formation and calcification. In children these clinical features may be present to a mild

degree and the disease can pass undetected. Other investigations are as follows.

- *Haematology*. The full blood count may show anaemia.
- *Immunology*. The Mantoux test is usually strongly positive in post-primary pulmonary TB, but is frequently negative in miliary TB.
- *Microbiology*. Sputum culture will show tubercle bacilli after 4–5 weeks of the primary infection. Bacilli may be cultured from the bone marrow in patients with miliary TB.
- *Diagnostic imaging*. In post-primary TB the chest X-ray may demonstrate a pleural effusion or pneumonia. A soft spreading apical shadowing is strongly suggestive of TB. There is widespread shadowing (i.e. small nodules 2–3 cm diameter) in miliary TB.

Prevention

Vaccination with BCG (Bacille Calmette–Guérin) greatly reduces the incidence of the disease and is offered to schoolchildren in the United Kingdom. The vaccination may be offered to people who might have contact with a patient who has active TB, such as relatives, friends, teachers, doctors, nurses and physiotherapists. Pasteurisation of milk prevents transmission of the tubercle to humans from cows.

Treatment

Drug therapy, together with rest, is the treatment for curing TB. Anti-TB drugs used are rifampicin, isoniazid, ethambutol and para-aminosalicylic acid; these must be taken every day for up to 18 months. The antibiotic streptomycin may also be prescribed. Multiple drug regimens are used in the treatment of resistant strains. Uncomplicated pulmonary TB is treated with a relatively short course (i.e. 6–9 months). If other organs are involved a longer course of treatment may be necessary (e.g. 18 months for bone disease). Multidrug resistant TB (MDR-TB) and extensively drug-resistant TB (XDR-TB) is becoming an increasing problem as a result of poorly administered drug regimes, or interrupted or inadequate therapy.

Surgery is appropriate only in a very small proportion of patients. If a patient has a resistant tubercle a lobectomy may be performed, but the patient must still be on a drug regimen. The prognosis is good if the patient is not immunosuppressed.

Physiotherapy in pulmonary tuberculosis

Once the patient is ambulant, a graded programme of exercises may be required. If it is necessary to give breathing exercises, the physiotherapist should stand behind the patient to avoid droplet infection as the patient coughs. Sputum must be disposed of very carefully so that cross-infection is prevented.

Bronchial and lung tumours

Key point

Tumours may be benign or malignant. The majority are malignant growths which may be primary or secondary.

Tumours arising within the lung (bronchial carcinomas) usually originate within the bronchi, while those that spread from other primary sites (e.g. breast, gastrointestinal tract) tend to develop in the lung tissue or the pleura.

In the UK, there are approximately 35,000 deaths from carcinoma of the bronchus each year. Men are more commonly affected than women, although the incidence in women is increasing.

Causative factors

People who smoke tobacco have a much greater risk of developing a malignant tumour than those who do not. The risk depends upon the number of cigarettes smoked, the age of starting to smoke and the timespan of smoking. The concept of pack-years is described at the beginning of this chapter.

The disease is more prevalent in urban dwellers than in rural dwellers. There is also evidence that exposure to carcinogens, either at work or leisure, can result in the development of the disease. Working with radioactive materials, nickel, uranium, chromates or industrial asbestos is associated with an increased risk of bronchial carcinoma.

Pathology

The majority of tumours originate in the large bronchi and spread by direct invasion of the lung, chest wall and mediastinal structures. The tumour grows to occlude the lumen of the bronchus and then atelectasis distal to the growth will occur. There are various types (Table 6.5).

Clinical features

Seventy per cent of patients present with local symptoms. The onset is insidious and the clinical features may present in a variety of ways.

- *Cough*. This is the most common feature and is often ignored by the patient who may associate it with smoking. Initially, the cough is dry and irritating but may become productive if infection occurs in accumulated secretions.

Table 6.5 Histology of bronchial tumours

Histology	Proportion of bronchial cancers (%)	Characteristics
Squamous cell	50	Locally invasive, cavitation sometimes occurs
Oat/small cell	25	Small lung primary, rapidly dividing, metastasise early
Large cell	12	Intermediate between squamous and oat/small cell
Adenocarcinoma	12	Slowly growing, metastasises late, often peripheral lung tumours
Miscellaneous	1	For example, alveolar oat cell carcinoma

- *Haemoptysis*. There are recurrent small spots of blood in the sputum.
- *Dyspnoea*. This is highly variable and may be severe when there is pulmonary collapse or pleural effusion.
- *Pain*. Dull, deep-seated pain is common but it may be pleuritic in nature or intercostal when there is rib disease.
- *Malaise and weight loss*. These are associated with late stages of the disease.
- *Secondary concomitant disease*. Pneumonia or lung abscess may arise as a result of a tumour.
- *Hoarseness of the voice*. This is a result of left recurrent laryngeal nerve involvement by tumour of the left hilum.
- *Stridor*. This is a result of narrowing of the trachea or main bronchus.
- *Facial swelling*. This is a result of superior vena caval obstruction following invasion of the mediastinum.
- *Arm and shoulder pain*. These are caused by tumour at the apex of the lung (Pancoast tumour) invading the brachial plexus.

Metastases

Metastases are common in patients with bronchial carcinoma and may include the following.

- *Cerebral metastases*. These may cause stroke, headaches and epilepsy.
- *Bone metastases*. The patient may present with spinal cord compression, pathological fracture and bone pain.
- *Liver metastases*. The patient may present with jaundice and hepatomegaly (an enlarged liver).

Non-metastatic presentations include finger clubbing, and neuromuscular and endocrine abnormalities.

Investigations

- *Chest radiograph*. This is essential for any patient presenting with haemoptysis and will demonstrate over 90% of lung tumours. Small tumours or those close to the hilum may be missed.
- *CT scanning*. This may be used to identify smaller lesions. It may also be used to assess suitability for surgery by demonstrating metastatic spread.
- *Histopathology*. Sputum culture may have evidence of tumour cells. Three (early morning) samples should be obtained.
- *Bronchoscopy*. This is used to obtain tissue samples and may also be used to assess operability.
- *Percutaneous needle biopsy*. This may be useful for the histological assessment of a peripheral tumour.
- *Pleural aspiration and biopsy*. These may be used for the patient who presents with a pleural effusion.

Treatment

The appropriate treatment may be surgery, chemotherapy and/or radiotherapy. Essentially, drug therapy is to relieve symptoms and includes analgesics, antibiotics and anti-emetics.

- *Surgery*. This involves removing the lobe or lung. It is possible only while the tumour remains localised and in the absence of metastases. Stenting is another option for localised disease.
- *Chemotherapy*. Cytotoxic drugs are used with increasing regularity. Results are mixed but anaplastic tumours tend to respond to this type of treatment.
- *Radiotherapy*. This is used symptomatically particularly to relieve pain and obstruction.
- *Laser phototherapy*. This can be used to treat persistent localised disease.

The prognosis depends upon the type of tumour, but the overall length of survival is around one year. Surgery can prolong life in some patients. In the terminal stages attention should be paid to the patient's general well-being and mental state. Some patients benefit from hospice care and adequate opiate analgesia is essential for pain.

Physiotherapy for bronchial and lung tumours

Physiotherapy may be related to three aspects of management of the disease.

- Pre- and postoperative physiotherapy is essential for patients who have a lobectomy or pneumonectomy.
- During and after radiotherapy, when the tumour begins to decrease in size, the patient will begin to expectorate sputum. Positioning and the ACBT should be used for sputum clearance. Percussion and vigorous shaking should not be used as there is a danger of pathological fractures in ribs or vertebrae in which metastases may be developing. Nor should shaking them be used in the presence of haemoptysis.
- During the terminal stage of the disease, where accumulation of secretions is causing distress, modified postural drainage and vibrations with breathing exercises may help to make the patient more comfortable. If coughing is ineffective, suction may have to be used. An active daily programme which fits the patient's requirements may need to be devised, in which case the physiotherapist works in close collaboration with the healthcare team.

RESPIRATORY FAILURE

ABC Definition

Respiratory failure denotes reduction of function of the lungs as a result of lung disease or a skeletal or neuromuscular disorder resulting in pump failure. It is defined in terms of the gas tensions (pressures) in the arterial blood (Roussos and Koutsoukou, 2003).

Normal arterial oxygen and carbon dioxide pressures (PaO_2 and $PaCO_2$) are 13.0 kPa (97 mmHg) and 6.1 kPa (46 mmHg) respectively. There are two types of respiratory failure.

- *Type 1*: A PaO_2 of less than 8.0 kPa (60 mmHg) is associated with a $PaCO_2$ which is either normal or below 6.7 kPa (50 mmHg).
- *Type 2*: A PaO_2 of less than 8.0 kPa (60 mmHg) is associated with a $PaCO_2$ raised above 6.7 kPa (50 mmHg).

Causes of type 1 respiratory failure

Lung disease results in hypoventilation of the alveoli leading to a ventilation/perfusion mismatch. The blood supply may be normal but there is inadequate oxygen uptake from the affected alveoli. There may be disruption in blood supply to oxygenated alveoli, or shunt, resulting in obstruction of passage of oxygen from the lungs into the blood in the affected area. Diseases associated with this type are early chronic bronchitis and emphysema, pneumonia, asthma, acute pulmonary oedema, pulmonary embolism, pulmonary fibrosis and ARDS.

Causes of type 2 respiratory failure

Because of failure of the skeletal or neuromuscular components of the respiratory system there is loss of the pump mechanism essential for ventilation of the lungs as a whole. Therefore, there is a reduced tidal volume or a reduced respiratory rate, leading to a rise in $PaCO_2$ and a fall in PaO_2. Disorders associated with this type are:

- respiratory muscle fatigue secondary to acute exacerbation of lung disease, such as advanced chronic bronchitis and emphysema;
- status asthmaticus;
- direct denervation to respiratory muscles which may occur in cervical cord injuries;
- neuromuscular disorders affecting the nerves innervating muscles of respiration or the muscles themselves, e.g. muscular dystrophy, myasthenia gravis and polyneuropathies;
- mechanical disorders affecting the thorax, such as kyphoscoliosis or a crush injury to the chest;
- central respiratory drive dysfunction, e.g. head injuries.

Clinical features

- *Type 1 owing to hypoxaemia*: There may be dyspnoea, restlessness, confusion, central cyanosis, tachycardia, renal failure, pulmonary hypertension.
- *Type 2 owing to hypercapnia*: There may be flapping tremor of the hands, confusion, headache, warm peripheries, tachycardia. Dyspnoea occurs initially but the person may become drowsy and comatose if $PaCO_2$ is allowed to rise.

Treatment

! Key point

The diagnosis cannot be accurate until arterial blood gases have been measured. Treatment must be directed towards treating the underlying cause.

In type 1 respiratory failure the main problem is the hypoxaemia, so it is important to raise the PaO_2 by giving oxygen therapy, which should be given in sufficient amounts to correct the hypoxaemia.

In type 2 respiratory failure the hypoxia needs to be reversed, but the hypercapnia also needs to be addressed. Support via invasive or noninvasive ventilation may be indicated in order to increase tidal volumes and 'blow off' excess CO_2.

Physiotherapy in respiratory failure

Type 1 failure

It is vital to clear the lung fields of secretions – if present. If the patient is spontaneously breathing, positioning and ACBT can be used with manual techniques to loosen secretions. If the patient is too weak to cough, a cough insufflator-exsufflator may be used to enhance the cough; however, suction may have to be used. If bronchospasm is evident, a bronchodilator (e.g. salbutamol) may be given in combination with oxygen therapy. Continuous positive airway pressure may be required to increase the functional residual capacity (FRC) and optimise gas exchange. If severe, invasive mechanical ventilation may be indicated. A bronchodilator may be administered through the ventilator and shakings and vibrations should be performed with manual hyperinflation. Suction will be via the endotracheal tube. All treatment is monitored by regular peripheral oxygen saturation measurement or blood gas analysis.

Type 2 failure

Again, it is necessary to raise the PaO_2 and this is achieved by oxygen therapy using a Ventimask delivering 24% oxygen. If this is not sufficient to raise the PaO_2 the Ventimask may be changed to one delivering 28% oxygen, provided the $PaCO_2$ is not rising further. If the $PaCO_2$ starts to rise this is indicative of hypoventilation and worsening of type 2 respiratory failure, usually because the patient is becoming exhausted. NIPPV may be indicated, which will augment tidal volume and increase the FRC. If hypercapnia is severe and NIV is contra-indicated the patient may require invasive mechanical ventilation. Physiotherapy follows similar principles to that for type 1 failure.

Chronic hypercapnia/acute-on-chronic type 2 respiratory failure

This is seen most frequently in patients with COPD. The pH will be within normal limits (owing to metabolic compensation); however, the $PaCO_2$ will be raised above normal. In type 2 respiratory failure there is a danger of reducing the respiratory drive which is normally dependent on the anoxic state of the blood stimulating the chemoreceptors in the carotid and aortic arteries. If too much supplemental O_2 is provided the danger is that the patient's respiration slows, or stops, and the $PaCO_2$ rises, resulting in confusion and coma. A Ventimask giving 24% or 28% inspired oxygen may be applied (see the section on oxygen therapy in COPD).

A patient with chronic hypercapnia may present with acute-on-chronic respiratory failure with a worsening pH and rising $PaCO_2$. Signs and symptoms may include worsening dyspnoea, increasing confusion and respiratory arrest. These patients should be treated the same way as a patient presenting with acute type 2 respiratory failure.

Respiratory failure in neuromuscular disease

Respiratory failure and dysfunction are common in many chronic neurodegenerative conditions, for example multiple sclerosis (Gosselink et al. 1999) and Huntington's disease (Sorensen and Fenger 1992). As these diseases progress the patient may lose his/her ability to cough effectively. Physiotherapy management should be aimed at the removal of retained secretions. This should focus on increasing lung volumes in order to create flow rates sufficient to mobilise and expectorate secretions from the airways. This may be achieved by use of NIV or by a cough-assist device. When expiratory flows remain insufficient, manually-assisted cough techniques can be used to augment patients' cough.

SLEEP AND BREATHING

It is important to consider what happens to breathing during sleep, as problems with breathing may initially become apparent during sleep before progressing to development of daytime respiratory failure.

Normally, during non-rapid eye movement (REM) sleep ventilation is decreased compared with ventilation when awake. During REM sleep breathing becomes irregular with episodes of severe hypoventilation. General muscle tone reduces, including a reduction in tone to the muscles that dilate the pharynx resulting in narrowing of the pharynx and an increase in upper airway resistance. Ventilatory responses to hypoxia and hypercapnia are reduced during REM sleep and in non-REM sleep to a lesser degree. The response of respiratory muscles to signals from the respiratory centre in the brain is reduced during sleep; however, the diaphragm is less affected than the accessory muscles. In normal people there is no clinically significant deterioration in arterial blood gas values; however, profound hypoxaemia may occur in patients with respiratory insufficiency which may eventually result in daytime respiratory failure (McNicholas 2000; Gay 2004).

REFERENCES

Abman, S.H., Ogle, J.W., Harbeck, R.J., et al., 1991. Early bacteriologic, immunologic, and clinical courses of young infants with cystic fibrosis identified by neonatal screening. J Paediatr 119, 211–217.

Adinoff, A.D., Hollester, J.R., 1983. Steroid-induced fractures and bone loss in patients with asthma. N Engl J Med 309, 265–268.

Amado, M.C., Portnoy, J.M., 2006. Recent advances in asthma management. Mo Med 103 (1), 60–64.

Andersen, Z.J., Hvidberg, M., Jensen, S.S., et al., 2010. Chronic obstructive pulmonary disease and long-term exposure to traffic-related air pollution: A cohort study. Am J Respir Crit Care Med 24, 247–253.

Armstrong, P., Wilson, A.G., Dee, P., et al., 1995. Imaging of Diseases of the Chest. Year Book Medical, Chicago.

Baena-Cagnani, C.E., Badellino, H.A., 2010. Diagnosis of allergy and asthma in childhood. Curr Allergy Asthma Rep 30 (6), 213–219.

Bastin, A.J., Starling, L., Ahmed, R., et al., 2010. High prevalence of undiagnosed and severe chronic obstructive pulmonary disease at first hospital admission with acute exacerbation. Chron Respir Dis 7 (2), 91–97.

Bell, S.C., Saunders, M.J., Elborn, J.S., et al., 1996. Resting energy expenditure and oxygen cost of breathing in patients with cystic fibrosis. Thorax 51, 126–131.

Bellemare, F., Grassino, A., 1982. Effect of pressure and timing of contraction on human diaphragmatic fatigue. J Appl Physiol 53, 1190–1195.

Bernard, G., Artiglas, A., Bringham, K., et al., 1994. Report on the American–European consensus conference on ARDS: definitions, mechanisms, relevant outcomes and clinical trial coordination. Inten Care Med 20, 225–232.

Bernard, S., LeBlanc, P., Whittom, F., et al., 1998. Peripheral muscle weakness in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 158, 629–634.

Bolton, C.E., Ionescu, A., Shiels, K., et al., 2004. Associated loss of fat-free mass and bone mineral density in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 170, 1286–1293.

Bone, R.C., 1996. Goals of asthma management: A step-care approach. Chest 4, 1056–1065.

Bott, J., Blumenthal, S., Buxton, M., et al., 2009. Guidelines for the physiotherapy management of the adult, medical, spontaneously breathing patient. Thorax 64, i1–i52.

Bradley, J., Moran, F., 2006. Pulmonary rehabilitation improves exercise tolerance in patients with bronchiectasis. Aust J Physiotherapy 52 (1), 65.

Braman, S.S., 2006. Chronic cough due to chronic bronchitis: ACCP evidence-based clinical practice guidelines. Chest 129 (Suppl. 1), 104S–1115.

BTS (British Thoracic Society), 2001. Pulmonary rehabilitation. Thorax 56 (11), 827–834.

BTS (British Thoracic Society), 2006a. The burden of lung disease: A statistical report from the British Thoracic Society, http://www.brit-thoracic.org.uk/Portals/0/Library/BTS%20Publications/burden_of_lung_disease.pdf; accessed October 2012.

BTS (British Thoracic Society), 2006b. Clinical component for the home oxygen service in England and Wales, http://www.brit-thoracic.org.uk/Portals/0/Clinical%20Information/Home%20Oxygen%20Service/clinical%20adultoxygenjan06.pdf; accessed October 2012.

BTS (British Thoracic Society), 2008. The BTS guidelines on the management of asthma. Thorax 63, v4–iv121.

Brochard, L., Manceebo, J., Wysocki, M., 1995. Non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. N Engl J Med 333, 817–822.

Brocklebank, D., Ram, F., Wright, J., et al., 2001. Comparison of the effectiveness of inhaler devices in asthma and chronic obstructive airway disease: A systematic review of the literature. Health Techn Assess 5 (26), 1–149.

Burr, M.L., Wat, D., Evans, C., et al., 2006. British Thoracic Society Research Committee Asthma prevelance in 1973, 1988 and 2003. Thorax 61 (4), 296–299.

Cecins, N.M., Jenkins, S.C., Pengelley, J., et al., 1999. The active cycle of breathing technique – to tip or not to tip? Respir Med 93, 660–665.

Cerny, F., Pullano, T., Gerd, J., et al., 1982. Cardiorespiratory adaptations to exercise in cystic fibrosis. Am Rev Respir Dis 126, 217–220.

Chang, S.C., Chang, H.I., Shiao, G.M., et al., 1993. Effect of body position on gas exchange in patients with unilateral central airways lesions. Down with the good lung? Chest 103, 787–791.

Christopher, F., Chase, D., Stein, K., et al., 1999. RhDNase therapy for the treatment of cystic fibrosis patients with mild to moderate lung disease. J Clin Pharm Ther 24, 415–426.

Clanton, T.L., Diaz, P.T., 1995. Focus on ventilatory muscle training: clinical assessment of the respiratory muscles. Phys Ther 75, 983–995.

Clarke, S.W., 1991. Chronic bronchitis in the 1990s. Respiration 58 (Suppl.), 43–46.

Cochrane, G.M., Clark, C.J., 1990. Benefits and problems of a physical training programme for asthmatic patients. Thorax 45 (5), 345–351.

Corda, L., Bertella, E., Pini, L., et al., 2006. Diagnostic flow chart for targeted detection of alpha 1–antitripsin deficiency. Respir Med 100 (3), 463–470.

Corless, J.A., Warburton, C.J., 2000. Surgery vs non-surgical treatment for bronchiectasis. Cochrane Database Syst Rev: CD002180.

Corraco, W.M., Braman, S.S., Erwin, R.S., 1979. Chronic cough as the sole presenting manifestation of bronchial asthma. N Engl J Med 300, 633–637.

Crossley, D.J., McGuire, G.P., Barrow, P.M., et al., 1997. Influence of inspired oxygen concentration on deadspace, respiratory drive, and

$PaCO_2$ in intubated patients with chronic obstructive pulmonary disease. Crit Care Med 25, 1522–1526.

Currie, D.C., 1997. Nebulisers for bronchiectasis. Thorax 52 (Suppl. 2), S72–S74.

Davies, D.S., 1973. Pharmokinetics of inhaled substances. Postgrad Med J 51 (Suppl. 7), 69–75.

Davis, P.B., Drumm, M., Konstan, M.W., 1996. Cystic fibrosis (state of the art). Am J Respir Crit Care Med 154, 1229–1256.

Decramer, M., 1989. Effects of hyperinflation on the respiratory muscles. Eur Respir J 2, 299–302.

Decramer, M., De Benedetto, F., Del Ponte, A., et al., 2005. Systemic effects of COPD. Respir Med (Suppl B), S3–10.

Decramer, M., Janssens, W., Miravitlles, M., 2012. Chronic obstructive pulmonary disease. Lancet 379 (9823), 1341–1351.

de Jong, W., Kaptein, A.A., Van der Schands, C.P., et al., 1997. Quality of life in patients with cystic fibrosis. Paediatr Pulmonol 23 (2), 95–100.

DH (Department of Health), 1997. Health of the Nation Briefing Pack. DH, Leeds.

Donahoe, M., Rogers, R.M., Wilson, D.O., et al., 1989. Oxygen consumption of the respiratory muscles in normal and malnourished patients with chronic obstructive pulmonary disease. Am Rev Respir Dis 140, 385–391.

Dransfield, M.T., Davis, J.J., Gerald, L.B., et al., 2006. Racial and gender differences in susceptibility to tobacco smoke among patients with chronic obstructive pulmonary disease. Respir Med 100 (6), 1110–1116.

Driks, M., Craven, D., Celli, C., 1987. Nosocomial pneumonia in intubated patients given sucralfale as compared with antacids or histamine type 2 blockers: the role of gastric colonisation. N Engl J Med 317, 1376–1382.

Elborn, S.J., 2007. How can we prevent multisystem complications of cystic fibrosis. Semin Respir Crit Care Med 28 (3), 303–311.

Elborn, J.S., Shale, D.J., Britton, J.R., 1991. Cystic fibrosis: current survival and population estimates to the year 2000. Thorax 46, 881–885.

Elborn, J.S., Johnston, B., Allen, F., 1992. Inhaled steroids in patients with bronchiectasis. Respir Med 86, 121–124.

Elborn, J.S., Cordon, S.M., Shale, D.J., 1993. Inflammatory responses prior to death in cystic fibrosis. Respir Med 87, 603–607.

Elborn, J.S., Prescott, R.J., Stack, B.H., 2000. Elective versus symptomatic antibiotic treatment in cystic fibrosis patients with chronic *Pseudomonas* infection of the lungs. Thorax 55, 355–358.

Fauroux, B., Hart, N., Luo, Y.M., et al., 2003. Measurement of diaphragm loading during pressure support ventilation. Intensive Care Med 29, 1960–1966.

Fleetham, J.A., Bradley, C.A., Kryger, M.H., et al., 1980. The effect of low flow oxygen therapy on the chemical control of ventilation in patients with hypoxemic COPD. Am Rev Respir Dis 122, 833–840.

Flume, P.A., O'Sullivan, B.P., Robinson, K.A., et al., 2007. Cystic fibrosis pulmonary guidelines. Chronic medications for maintenance of lung health. Am J Respir Crit Care Med 176, 957–969.

Flume, P.A., Mogayzel, P.J., Robinson, K.A., et al., 2009. Cystic fibrosis guidelines. Treatment of pulmonary exacerbations. Am J Respir Crit Care Med 180, 802–808.

Folgering, H., von Herwaarden, C., 1994. Exercise limitations in patients with pulmonary diseases. Int J Sports Med 15 (3), 107–111.

Fowler, A., Goldman, M., 1990. Adult respiratory distress syndrome: prognosis after onset. Am Rev Respir Dis 132, 472–478.

Freitag, L., Bremme, J., Schroer, M., 1989. High frequency oscillation for respiratory physiotherapy. Br J Anaesth 63, 44S–46S.

Gay, P.C., 2004. Chronic obstructive pulmonary disease and sleep. Respir Care 49 (1), 39–51.

Geddes, E.L., O'Brien, K., Reid, W.D., et al., 2008. Inspiratory muscle training in adults with chronic obstructive pulmonary disease: An update of a systematic review. Respir Med 102 (12), 1715–1729.

GOLD (Global Initiative for Obstructive Lung Disease), 2011. Global strategy for the diagnosis, management and prevention of COPD, http://www.goldcopd.org., accessed October 2012.

Gosselink, R., Kovacs, L., Decramer, M., 1999. Respiratory muscle involvement in multiple sclerosis. Eur Respir J 13 (2), 449–454.

Green, M., Moxham, J., 1993. Respiratory muscles in health and disease. In: Barnes, P. (Ed.), Respiratory Medicine: Recent Advances. Butterworth-Heinemann, Oxford, pp. 252–275.

Gross, N.J., 1995. Airway inflammation in COPD: reality or myth? Chest 107 (Suppl. 5), S1–24.

Guell, R., Resqueti, V., Sangenis, M., et al., 2006. Impact of pulmonary rehabilitation on psychosocial morbidity in patients with severe COPD. Chest 129 (4), 899–904.

Hargreave, F.E., Dolovich, J., Newhouse, M.T. (Eds.), 1990. The assessment and treatment of asthma: A conference report. J Allergy Clin Immunol 85, 1098–2011.

Hasegawa, M., Nasuhara, Y., Onodera, Y., et al., 2006. Airflow limitation and airways dimensions in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 173 (12), 1309–1315.

Haworth, C.S., Selby, P.L., Webb, A.K., 1999. Low bone mineral density in adults with cystic fibrosis. Thorax 54, 961–967.

Hogg, J.C., 2004. Pathophysiology of airflow limitation in chronic obstructive pulmonary disease. Lancet 364, 709–721.

Israel, E., Rubin, P., Kemp, J.P., et al., 1993. The effect of inhibition of 5-lipoxygenase by Ziluteton in mild-to-moderate asthma. Ann Intern Med 119, 1059–1066.

Jeffery, P.K., 2000. Comparison of the structural and inflammatory features of COPD and asthma. Giles F. Filley Lecture. Chest 117 (90051), 251S–260S.

Johnson, J.L., Campbell, A.C., Bowers, M., et al., 2007. Understanding the social consequences of chronic obstructive pulmonary disease: The effects of stigma and gender. Proc Am Thorac Soc 4 (8), 680–682.

Kaplan, T.A., ZeBranek, J.D., McKey, R.M., 1991. Use of exercise in the management of cystic fibrosis. Paediatr Pulmonol 10, 205–207.

Kesten, S., Chapman, K.R., 1993. Physician perceptions and management of COPD. Chest 104, 254–258.

Kikuchi, Y., Okabe, S., Tamura, G., et al., 1994. Chemosensitivity and perceptions of dyspnoea in patients with a history of near-fatal asthma. N Engl J Med 330, 1329–1334.

Konstan, M.W., Berger, M., 1993. Infection and inflammation of the lung in cystic fibrosis. In: Davis, P.B. (Ed.), Lung Biology in Health and Disease, vol. 64: Cystic Fibrosis. Marcel Dekker, New York, pp. 219–276.

Konstan, M.W., Hillard, K.A., Norvell, T.M., 1994. Bronchoalveolar lavage findings in cystic fibrosis patients with stable, clinically mild lung disease suggest ongoing infection and inflammation. Am J Respir Crit Care Med 150, 448–454.

Kurmi, O.P., Semple, S., Simkhada, P., et al., 2010. COPD and chronic bronchitis risk of indoor air pollution from solid fuel: A systematic review and meta-analysis. Thorax 65 (3), 221–228.

Lacasse, Y., Wong, E., Guyatt, G.H., et al., 1996. Meta-analysis of respiratory rehabilitation in chronic obstructive pulmonary disease. Lancet 348, 1115–1119.

Levine, S., Guillen, M., 1987. Diaphragmatic pressure waveform can predict EMG signs of diaphragmatic fatigue. J Appl Physiol 62, 1681–1689.

Lightowler, J.V., Wedzicha, J.A., Elliott, M.W., et al., 2003. Non-invasive positive pressure ventilation to treat respiratory failure resulting from exacerbations of chronic obstructive pulmonary disease: Cochrane systematic review and analysis. BMJ 326, 185–189.

Ling, S.C., Wilkinson, J.D., Hollman, A.S., et al., 1999. The evolution of liver disease in cystic fibrosis. Arch Dis Child 81, 129–132.

Loring, S.H., Garcia-Jacques, M., Malhotra, A., 2009. Pulmonary characteristics in COPD and mechanisms of increased work of breathing. J Appl Physiol 107 (1), 309–314.

Maltais, F., LeBlanc, P., Jobin, J., et al., 1997. Intensity of training and physiologic adaptation in patients with chronic obstructive pulmonary disease. Am J Respir Crit Care Med 155 (2), 555–561.

McGarvey, L.P., John, M., Anderson, J.A., et al., 2007. Ascertainment of cause-specific mortality in COPD: operations of the TORCH Clinical Endpoint Committee. Thorax 62 (5), 411–415.

McNicholas, W.T., 2000. Impact of sleep in COPD. Chest 117, 48S–53S.

Meecham-Jones, D.J., Paul, E.A., Jones, P.W., et al., 1995. Nasal pressure support ventilation plus oxygen compared with oxygen therapy alone in hypercapnic COPD. Am J Respir Crit Care Med 152, 538–544.

Montgomery, A., Stager, M., Carrico, C., et al., 1985. Causes of mortality in patients with adult respiratory distress syndrome. Am Rev Respir Dis 132, 485–489.

Moxham, J., 1990. Respiratory muscle fatigue: mechanisms, evaluation and therapy. Br J Anaesth 65, 43–53.

Murciano, D., Auclair, M.H., Pariente, R., et al., 1989. A randomised controlled trial of theophylline in patients with severe chronic obstructive pulmonary disease. N Engl J Med 320, 1521–1525.

National Asthma Campaign, 2002. Starting as we mean to go on. An audit of children's asthma in the UK. Asthma J 8, special supplement.

Nava, S., Hill, N., 2009. Non-invasive ventilation in acute respiratory failure. Lancet 374, 250–259.

Newman, S.P., Pavia, D., Clark, S.W., 1981. How should a pressurised beta-adrenergic bronchodilator be inhaled? Eur J Respir Dis 62, 3–21.

Newman, S.P., Pavia, D., Garland, N., et al., 1982. Effects of various inhalation modes on the deposition of radioactive pressurized aerosols. Eur J Respir Dis 63 (Suppl.), 57–65.

NICE (National Institute for Clinical Excellence) Guideline, 2010. CG101 Chronic obstructive pulmonary disease (update), http://www.nice.org.uk/nicemedia/live/13029/49397/49397.pdf; accessed 26 January 2010.

Nicholson, K.G., Kent, J., Ireland, D.C., 1993. Respiratory viruses and exacerbations of asthma in adults. BMJ 307, 982–986.

Nixon, P.A., Orenstein, D.M., Kelsey, S.F., et al., 1992. The prognostic value of exercise testing in patients with cystic fibrosis. N Engl J Med 327, 1785–1788.

O'Callahan, C., Barry, P., 1997. Spacer devices in the treatment of asthma. Br Med J 314, 1061–1062.

O'Neill, P.A., Dodd, M., Phillips, B., 1987. Regular exercise and reduction of breathlessness in cystic fibrosis. Br J Dis Chest 81, 62–66.

Oppenheimer, E.H., Esterly, J.R., 1975. Pathology of cystic fibrosis: review of the literature and comparison with 146 autopsied cases. Persp Paediatr Pathol 2, 241–248.

Patel, I.S., Vlahos, I., Wilkinson, T.M.A., et al., 2004. Bronchiectasis, exacerbation indices, and inflammation in chronic obstructive pulmonary disease. Am J Respir Crit Care Med 170 (4), 400–407.

Patessio, A., Iolo, F., Donner, C.F., 1993. Exercise prescription. In: Casaburi, R., Petty, T.L. (Eds.), The Principles and Practice of Pulmonary Rehabilitation. WB Saunders, Philadelphia.

Pearson, M.G., Calverley, P.M.A., 1995. Clinical and laboratory assessment. In: Pride, N.B. (Eds.), Calverley PMA. Chronic Obstructive Pulmonary Disease. Chapman & Hall, London.

Pearson, M.G., Spence, D.P.S., Ryland, I., et al., 1993. Value of pulsus paradoxus in assessing acute severe asthma. BMJ 307, 659.

Peters, S.P., Ferguson, G., Deniz, Y., et al., 2006. Uncontrolled asthma: A review of the prevalence, disease burden and options for treatment. Respir Med 100 (7), 1139–1151.

Polkey, M.I., Green, M., Moxham, J., 1995. Measurement of respiratory muscle strength. Thorax 50, 1131–1135.

Poole, P., Black, P., 2010. Mucolytic agents for chronic bronchitis or chronic obstructive pulmonary disease. Cochrane Database Systematic Rev: CD001287.

Porsbjerg, C., von Linstow, M.L., Ulric, C.S., et al., 2006. Risk factors for

onset of asthma: A 12-year prospective follow-up study. Chest 129 (2), 309–316.

Prior, J.G., Cochrane, G.M., 1980. Home monitoring of peak expiratory flow rate using a mini-Wright peak flow meter in diagnosis of asthma. J R Soc Med 73, 1329–1334.

Prior, J.G., Webber, B.A., 1979. An evaluation of the forced expiration technique as an adjunct to postural drainage. Physiotherapy 65, 304–307.

Pryor, J.A., Tannenbaum, E., Scott, S.F., et al., 2010. Beyond postural drainage and percussion: Airway clearance in people with cystic fibrosis. J Cyst Fibros 9, 187–192.

Quinton, P.M., 1990. Cystic fibrosis: A disease of electrolyte transport. FASEB J 4, 2709–2717.

Ramsey, B.W., Farrell, P.M., Pencharz, P., 1992. Nutritional assessment and management in cystic fibrosis: A consensus report. Am J Clin Nutr 55, 108–116.

Ratjen, F., Doring, G., 2003. Cystic fibrosis. Lancet 361 (9358), 681–689.

Rees, P.J., Clark, T.J.H., Moren, F., 1982. The importance of particle size in response to inhaled bronchodilators. Eur J Respir Dis 119 (Suppl.), 73–78.

Richards, G.N., Kolbe, J., Fenwick, J., et al., 1993. Demographic characteristics of patients with severe life threatening asthma: Comparison with asthma deaths. Thorax 48 (11), 1105–1109.

Rosen, M.J., 2006. Chronic cough due to bronchiectasis: ACCP evidence-based clinical practice guidelines Chest 129 (1), 122S–131S.

Roussos, C., Koutsoukou, A., 2003. Respiratory failure. Eur Respir J 22 (Suppl.)47, 3S–14S.

Roussos, C., Zakynthinos, S., 1996. Fatigue of the respiratory muscles. Intensive Care Med 22, 134–155.

Ryter, S.W., Lee, S.J., Choi, A.M., 2010. Autophagy in cigarette smoke-induced chronic obstructive pulmonary disease. Expert Rev Respir Med 4 (5), 573–584.

Sahl, W., Bilton, D., Dodd, M., et al., 1989. Effect of exercise and physiotherapy in aiding sputum expectoration in adults with cystic fibrosis. Thorax 44, 1006–1008.

Senn, O., Russi, E.W., Imboden, M., et al., 2005. Alpha 1-Antitrypsin deficiency and lung disease: risk modification by occupational and environmental inhalants. Eur Resp J 26 (5), 909–917.

Seymour, J.M., Ward, K., Sidhu, P., et al., 2009. Ultrasound measurement of rectus femoris cross-sectional area and the relationship to quadriceps strength in chronic obstructive pulmonary disease. Thorax 64 (5), 418–423.

Shiley, K.T., Lautenbach, E., Lee, I., 2010. The use of antimicrobial agents after diagnosis of viral respiratory tract infections in hospitalized adults: Antibiotics or anxiolytics? Infect Control Hosp Epidemiol 31 (11), 1177–1183.

Shoemark, A., Ozerovitch, L., Wilson, R., 2007. Aetiology in adult patients with bronchiectasis. Respir Med 101 (6), 1163–1170.

Silverman, E.K., Weiss, S.T., Drazen, J.M., et al., 2000. Gender-related differences in severe, early-onset chronic obstructive pulmonary disease. Am J Crit Care Med 162 (6), 2152–2158.

Sinden, N.J., Stockley, R.A., 2010. Systemic inflammation and comorbidity in COPD: A result of 'overspill' of inflammatory mediators from the lungs? Review of the evidence. Thorax 65 (10), 930–936.

Sinuff, T., Cook, D., Randall, J., et al., 2000. Noninvasive positive-pressure ventilation: A utilisation review of use in a teaching hospital. Can Med Assoc J 163, 969–973.

Smyth, A., Walters, S., 2000. Prophylactic antibiotics for cystic fibrosis. Cochrane Database Syst Rev CD0011912.

Soares, S., Costa, I., Neves, A.L., et al., 2010. Characterisation of a population at increased risk of COPD. Rev Port Pnumol 16, 237–252.

Solomon, C., Christian, D., Welch, B., et al., 1996. Cellular pulmonary tract and pulmonary function responses to exercise and serial sputum inductions. Am J Respir Crit Care Med 153, A713.

Sorensen, S., Fenger, K., 1992. Causes of death in patients with Huntington's disease and in unaffected first degree relatives. J Med Genet 29 (12), 911–914.

Sørheim, I.C., Johannessen, A., Guslsvik, A., et al., 2010. Gender differences in COPD: Are women more susceptible to smoking effects than men? Thorax 65 (6), 480–485.

Stavngaard, T., Shaker, S.B., Dirksen, A., 2006. Quantitative assessment of emphysema distribution in smokers and patients with alpha 1–antitripsin deficiency. Respir Med 100 (1), 94–100.

Svanberg, L., 1957. Influence of posture on lung volumes, ventilation and circulation in normals. Scand J Clin Lab Invest 9 (Suppl. 25), 1–195.

Tashkin, D.P., Ashutosh, K., Bleeker, E.R., 1986. Comparison of the anticholinergic bronchodilator ipratropium bromide with metaproterenol in chronic obstructive pulmonary disease: A 90-day multicenter study. Am J Med 81 (Suppl. 5a), 81–90.

Thomas, S.H., O'Doherty, M.J., Graham, A., et al., 1991. Pulmonary deposition of nebulised amiloride in cystic fibrosis: Comparison of two nebulisers. Thorax 46, 717–721.

Thurlbeck, W.M., 1976. Chronic Airflow Obstruction in Lung Disease. WB Saunders, Philadelphia.

Turpin, S.V., Knowles, M.R., 1993. Treatment of pulmonary disease in patients with cystic fibrosis. In: Davis, P.B. (Ed.), Lung Biology in Health and Disease, vol. 64: Cystic Fibrosis. Marcel Dekker, New York, pp. 236–243.

Vender, R.L., 1994. Chronic hypoxic pulmonary hypertension. Chest 106, 236–243.

Wark, P.A., McDonald, V., 2000. Nebulised hypertonic saline for cystic fibrosis. Cochrane Database System Rev: CD001506.

Webber, B.A., Hofmeyr, J.L., Morgan, M.D.L., et al., 1986. Effects of postural drainage, incorporating the forced expiration technique on pulmonary function in cystic fibrosis. Br J Dis Chest 80, 353–359.

West, J.B., 2008. Pulmonary Pathophysiology: The Essentials, seventh ed. Wolters Kluwer/

Lippincott Williams & Wilkins, Philadelphia.

WHO (World Health Organization), 2004. The global burden of disease: 2004 update. WHO Press, Geneva.

Wilson, G.E., Baldwin, A.L., Walshaw, M.J., 1995. A comparison of traditional chest physiotherapy with the active cycle of breathing in patients with chronic suppurative lung disease. Eur Respir J 8, 1715.

Yang, M., Yuping, Y., Wang, B.Y., et al., 2010. Chest physiotherapy for pneumonia in adults. Cochrane Database System Rev: CD006338.

Yelin, E., Katz, P., Balmes, J., et al., 2006. Work life of persons with asthma, rhinitis, and COPD: A study using a national, population-based sample. J Occup Med Toxicol 1 (1), 2.

Zach, M.S., Oberwaldner, B., 1989. Chest physiotherapy: the mechanical approach to antiinfective therapy in cystic fibrosis. Infection 15, 381–384.

Chapter 7

Adult spontaneous and conventional mechanical ventilation

Sue Pieri–Davies, Helen Carruthers, with a Contribution from Melanie Reardon

INTRODUCTION

This chapter focusses on the ventilatory aspects of the patient in respiratory failure. A basic understanding of all major systems is essential when considering the physiotherapeutic requirements of the ventilated adult, as the initial assessment necessitates identification of the underlying cause. The aim of intervention will then either be correction where possible and/or alleviation of respiratory symptoms, such as breathlessness, re-inflation, reduction in the work of breathing and sputum retention.

The detailed undergraduate training in anatomy and physiology places the physiotherapist in an excellent position to manage the respiratory and rehabilitation needs of such complex patients. However, in order to enable an applied approach to the assessment (incorporating problem-solving) findings and required management, as is the requirement in the emergency on-call duty setting, it is essential to understand the basics of respiratory mechanics in the spontaneously breathing individual.

This section, focussed on conventional mechanical ventilation, should be read in conjunction with Chapters 6 and 8. It is beyond the scope of a single chapter to detail all appropriate systems, the numerous conditions and causes of ventilatory failure, the concept and uses of non-invasive ventilation, and the more specialised area of domiciliary ventilation. A more detailed knowledge of these subjects can be obtained through further private study of the recommended texts and websites provided at the end of this chapter.

SPONTANEOUS VENTILATION

The basics of respiratory mechanics in the spontaneously breathing individual must be understood, as the application/weaning of mechanical ventilatory support/control will have a direct impact upon respiratory capacity. An ability to recognise and interpret deviations from the normal is of prime importance in maximising and maintaining efficient spontaneous respiratory function, particularly during an episode of critical illness.

The respiratory muscles

The respiratory muscles consist of two main groups: the primaries (consisting mostly of the diaphragm and intercostals) and the accessories (comprising mostly of the scaleni, abdominals and sternocleidomastoids). It is also important to remember the effects upon the oxygenation status and the balance of the blood pH with increasing metabolic demands should the recruitment of other additional muscles, for example the facial muscles with pursed-lip breathing, and the shoulder girdle and arm fixators, be required during times of respiratory distress/dysfunction. The primaries' main role is that of ventilation. The accessories have other functions, but are recruited to facilitate ventilation when required. Normally, the respiratory muscles have both ventilatory and non-ventilatory motor functions, for example the diaphragm acts as the primary respiratory muscle, responsible for generating approximately 60–70% of the tidal volume while also being responsible for raising intra-abdominal pressure for postural stabilisation of the torso, parturition and micturition. Such considerations must be appreciated by the therapist: when the respiratory muscles are required for both motor and ventilatory functions, their ability to assist ventilation is reduced. This is of particular importance when ventilatory support has recently been reduced and motor activity is being encouraged during daytime hours, i.e. during the weaning and rehabilitative phases of recovery. Upper limb strengthening exercises may be a primary aim at this stage so increasing ventilatory support overnight may be appropriate (Table 7.1).

Table 7.1 Comparison of skeletal muscle fibre types

Characteristics	Type I	Type IIa	Type IIb
Contractile			
Contraction velocity	Slow	Fast	Fast
Myosin adenosine triphosphatase	Slow	Fast	Fast
Twitch duration	Long	Short	Short
Calcium ion sequestration	Slow	Rapid	Rapid
Metabolic			
Capillaries	Abundant	Intermediate	Sparse
Glycolitic capacity	Low	Intermediate	High
Oxidative capacity	High	High	Low
Glycogen content	Low	Intermediate	High
Myoglobin content	High	Intermediate	Low
Fibre diameter	Small	Intermediate	Large
Motor unit size	Small	Intermediate	Large
Recruitment order	Early	Intermediate	Late

(Reproduced from Schauf et al. (1990), with permission.)

The respiratory muscles share the common features of other skeletal muscles and consist of a mixture of fibre types (Johnson et al. 1973). The proportions of fibre types and the metabolic constituents (e.g. capillaries; glycolytic and oxidative capacities; and time of recruitment in contraction) determine a muscle's strength and endurance properties (Schauf et al. 1990). Type I fibres are important for endurance (slow twitch, high oxidative capacity are recruited first and are most resistant to fatigue). Type IIa fibres have a higher oxidative capacity, fast twitch and produce an intermediate level of force, and so are relatively resistant to fatigue, while Type IIb fibres have a low oxidative capacity, fast twitch, produce the greatest force on activation, are the last to be recruited for motor efforts and are easily fatigued when used repeatedly. Greater knowledge of muscle physiology is required if the aim is to train the respiratory muscles, rather than rest them (via ventilatory support). Muscle training may be an appropriate physiotherapy intervention to facilitate the weaning episode of the prolonged ventilatory supported individual, where muscle wasting and disuse atrophy are evident, though more research is required in this area.

Respiratory mechanics and airflow

Contraction of the respiratory muscles affects the overall motion of the chest wall, for example in the upright position, on inspiration, the diaphragm moves downwards on contraction while the abdomen moves out. Synchronicity is achieved when the rib cage and the abdomen move together, increasing in diameter during inspiration and decreasing in diameter during expiration. In the supine position, most movements are abdominal with little movement of the rib cage. Body position in both respiratory mechanics and ventilation to perfusion matching is of great importance, as respiratory muscle dysfunction alone, for example fatigue or weakness, can lead to dyspnoea, hypoventilation, hypercapnoea, reduced oxygenation of body tissues, respiratory failure, metabolic acidosis and, ultimately, death.

Normal resting ventilation is a tri-cyclical activity consisting of the inward flow of air (inspiration), the outward flow (exhalation) and the rest phase, which constitutes the zero-flow status. During the inspiratory and expiratory cycles, a volume of air moves in and out of the lungs. These changes occur as a result of pressure gradients between the airway opening (or mouth) and the alveoli. Prior to the beginning of inspiration, the pressures at the airway opening and in the alveoli are equivalent. As there is no pressure gradient, there is no air movement – this is known as the resting period of the respiratory cycle, where air neither enters nor leaves the lungs (see Figure 7.1).

As inspiration begins the respiratory muscles contract, causing an upward and outward movement of the chest

wall (the bucket and pump handle effects) and the diaphragm descends (see Figure 7.2). This is known as the active phase of the breathing cycle and demands effort (termed the work of breathing). The changes in thoracic dimensions create a drop in the alveolar pressure; the pressure gradient between the airway opening and the alveoli results in an inward movement of air. The volume change that occurs is called the tidal volume and is, on average, 500 mL. (Joint guidelines by the British Thoracic Society and Association of Respiratory Technicians and Physiologists are available for a detailed underpinning of spirometric values and tests. Also downloadable from the internet are the American and European thoracic society recommendations for spirometry).

The opposing forces to ventilation

The work of breathing derives from the two resistive forces of the lungs and chest wall, i.e. the elastic (see Figure 7.3) and frictional forces. Forces within the respiratory system that oppose inflation of the lung and therefore ventilation can be grouped into two categories:

- the elastic opposition to expansion of the lungs;
- the frictional opposition or resistance to air movement.

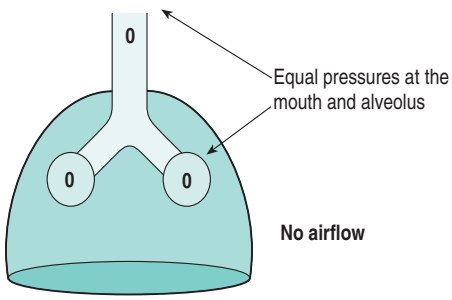


Figure 7.1 Spontaneous breathing: rest phase.

The pressure change that is generated on inspiration must be sufficient to overcome such forces. The effort required and the resulting volume change is termed the 'work of breathing'. Normally, the work of breathing is minimal (healthy lungs). A pressure gradient of 2–5 cmH_2O is typically needed to move the average tidal volume.

The elastic forces are encountered as a result of both the lungs and chest wall being 'elastic' structures, i.e. they resist changes in shape. When they have been inflated or

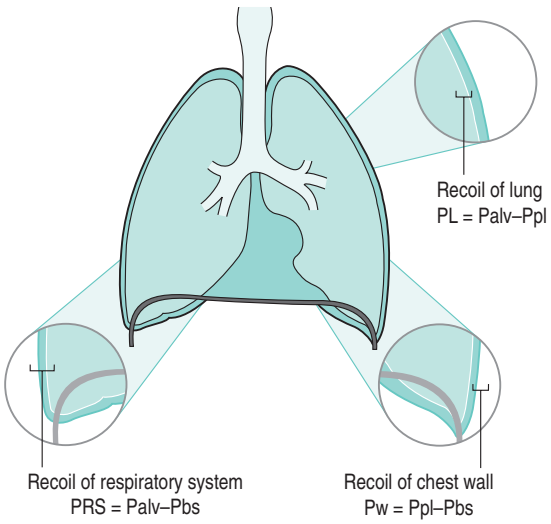


Figure 7.3 The elastic forces of the lung and chest wall. Palv = alveolar pressure (pressure within the alveoli); Pbs = pressure at the body surface; Pl = transpulmonary pressure (pressure difference across the lung); Ppl = pleural pressure; PRS = pressure with the respiratory system; Pw = pleural pressure minus pressure at the body surface (= elastic recoil pressure of the chest wall). *(Courtesy of the Johns Hopkins University website.)*

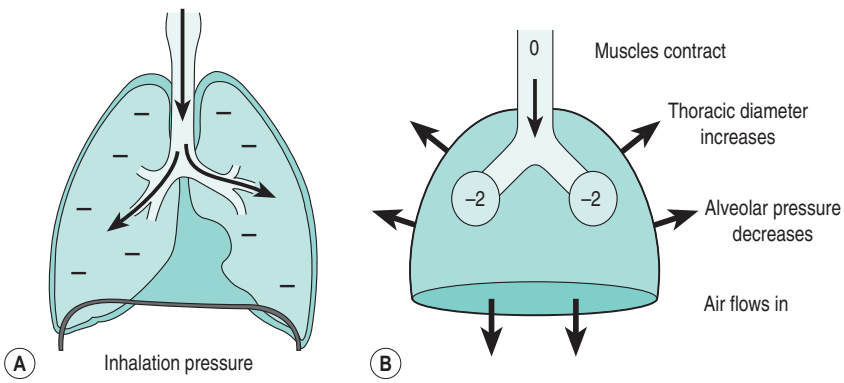


Figure 7.2 Spontaneous breathing: inspiratory phase. *(Courtesy of the Johns Hopkins University website.)*

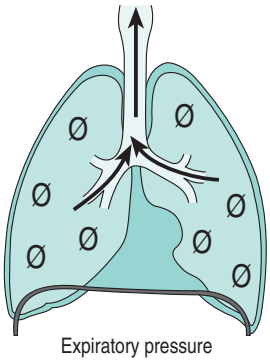


Figure 7.4 Expiratory pressures. *(Courtesy of the Johns Hopkins University website.)*

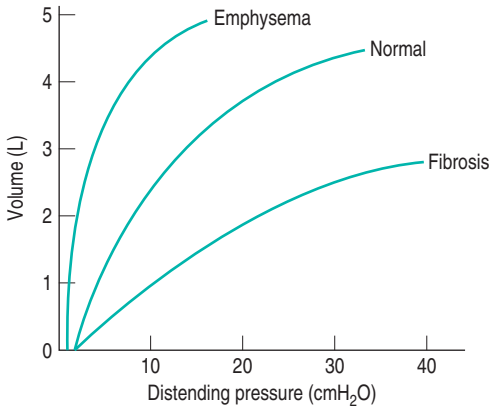


Figure 7.5 Lung compliance curves.

deflated, they tend to return to the same resting/starting position of equilibrium once the driving force has been removed. The lungs naturally want to collapse and the chest wall naturally to expand. Thus, each exerts a pull on the other. In the absence of other forces (e.g. muscles) a position is reached in which the opposing forces are balanced.

Owing to these opposing forces (of lung and chest wall), the intrathoracic pressure is negative (sub-atmospheric). To inflate the lungs an extra force must be applied (by the muscles) and intrathoracic pressure falls lower.

Expiration is mostly passive as a result of the elastic forces returning the lungs and chest wall to a balanced position (Figure 7.4). It can, however, be active, for example forced breathing and coughing, where the expiratory muscles assist the elastic forces (resulting in a more rapid expiratory rate of flow and faster lung deflation).

The inspiratory muscles perform mechanical work through upsetting the balance of the elastic forces. Hence, the harder and faster the respiratory effort is, the more 'elastic' work is required. A certain amount of pressure is required to stretch the lungs to a certain volume. The normal value for elastance is around 10 cmH_2O/L. However, in disease states, the lungs become stiffer and the same pressure change may result in a smaller volume change, i.e. the elastance of the lung is higher. Pneumonia, acute respiratory distress syndrome (ARDS) and pulmonary oedema are common lung conditions affecting elastance. Others include fibrotic lung disease, pleural effusion, kyphoscoliosis and obesity.

Compliance

Compliance (demonstrated by the pressure volume curve) is a measure of the distensibility of the lungs or ease with which the lungs inflate (i.e. the reciprocal of elastance). It is measured as the change of volume in the lungs in response to a change in pressure. Normal lung and chest wall compliance is ~1L per kPa (100 mL per cmH_2O). It can be seen in Figure 7.5 that lung compliance is lowered at high and low lung volumes. Hence, it is favourable for the patient to sit on the steep slope portion of the curve (it is easier to inflate a partially opened lung than a collapsed lung).

The frictional forces

The second group of opposing forces encountered in ventilation is the frictional forces. Impedance to air movement through the airways is called airways resistance. A small, but measurable, amount of work must be done to maintain the flow. Frictional forces are therefore dependent upon the speed with which air moves through the airway. Resistance is defined as the ratio of the pressure change responsible for air movement and the rate of flow. A normal value for resistance is around 2.5 $cmH_2O/L/sec$. Factors affecting airways resistance include the size of the airway, its shape and calibre (Figure 7.5). Different diseases affect such properties, thus altering airway resistance, for example chronic obstructive pulmonary disease (COPD) is the most frequently encountered lung disease that increases airways resistance.

With the presence of lung disease, the opposing forces of ventilation are increased. To sufficiently ventilate the lungs, larger patient efforts may be necessary to generate the pressure change needed to overcome the increased elastance or resistance (see Figure 7.6). Sustaining large inspiratory efforts may lead to excessive workloads and eventually respiratory fatigue and failure.

When the work of breathing is excessive because of an increase in the elastic or resistive forces present, respiratory muscle weakness from fatigue may develop. The strength of the muscles will be inadequate to support normal levels of ventilation and muscle pump effectiveness is diminished resulting in inadequate ventilation and an increase in the arterial carbon dioxide level ($PaCO_2$) causing respiratory acidosis. Hypoventilation or respiratory muscle fatigue may also lead to severe hypoxia. Under these

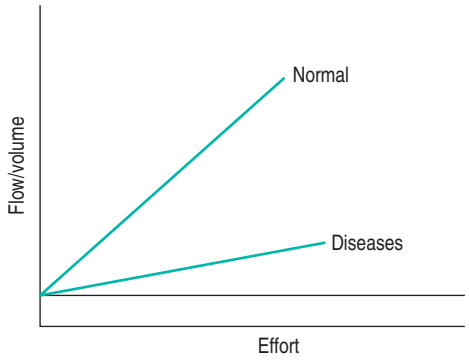


Figure 7.6 The relationship between patient effort and flow/volume.

conditions, ventilatory support to unload the respiratory muscles and improve ventilation is indicated.

Body positioning, pharmacological management and oxygen therapy are of prime importance to reduce the opposing forces of respiration and maximise ventilation prior to, and during, the instigation of mechanical support.

RESPIRATORY FAILURE

Respiratory failure occurs when there is an imbalance of ventilatory requirements to neurocardiorespiratory capacity. It is a common medical condition that occurs when the lungs fail to oxygenate arterial blood adequately (type I respiratory or lung failure) and/or the muscle pump fails to prevent undue CO_2 retention (type II ventilatory or pump failure). While no absolute diagnostic values for arterial oxygen (PaO_2) and $PaCO_2$ have been defined, values generally quoted are a PaO_2 of less than 8.0 kPa and a $PaCO_2$ of greater than 6.0 kPa (Roussos and Koutsoukou 2003).

Arterial blood gas tensions must be measured to make the diagnosis of respiratory failure, as many of the accompanying signs and symptoms, for example breathlessness, dyspnoea, cyanosis, agitation and the use of accessory muscles, are not diagnostic of the condition. Oximeters that estimate arterial oxygen saturation (SaO_2), either from the finger or earlobe, are useful tools for indicating hypoxaemia (SaO_2 <90%), assessing severity and monitoring the patient's condition. Accordingly, all respiratory compromised patients should have oximetry checked. It is important to note, however, that this may be falsely reassuring in the patient receiving supplemental oxygen, as alveolar hypoventilation is not detected. Unconscious patients should, therefore, have arterial blood gas analysis at initial assessment to exclude respiratory failure either as a cause or consequence of neurological depression.

Table 7.2 The typical clinical features of type I respiratory failure

Hypoxia
Hypocarbia
Tachypnoea
Small tidal volumes

Acute hypoxaemic (type I) respiratory failure

This is caused by intrinsic lung disease that interferes with oxygen transfer in the lung. Hypoxaemia results from increased right-to-left shunts (e.g. pneumonia, alveolar collapse, oedema and consolidation) or, more significantly, ventilation perfusion (V/Q) mismatch, for example pulmonary parenchymal disease, or a combination of the two. Where functional residual capacity (FRC) is reduced, airway closure is present throughout the respiratory cycle as tidal exchange occurs below closing volume. This results in an increased number of under-ventilated lung units (placing the patient lower down on the compliance curve).

The increased dead space (air in the airways which does not directly contribute to gas exchange) results initially in an increase in total ventilation to maintain a normal $PaCO_2$. This is because in areas of V/Q inequality, the raised arterial PCO_2 resulting from decreased CO_2 excretion in the under-ventilated alveoli stimulates the respiratory centre. Relative hyperventilation may ensue in response to severe hypoxaemia with a resultant drop in $PaCO_2$ to below normal range. (The degree of hypoxaemia is restricted by constriction of the blood vessels supplying under-ventilated alveoli, i.e. hypoxic pulmonary vasoconstriction.)

The mechanical disadvantage of a reduced FRC results from the consequential reduction of lung compliance and increased resistance, resulting in a greater work of breathing with a higher metabolic cost. The resultant clinical picture is that of rapid shallow breathing, which, in turn, further increases both oxygen consumption and carbon dioxide volume for excretion (Table 7.2).

Ventilatory (type II) respiratory failure

This is caused by failure of the respiratory pump (consisting of the respiratory muscles, chest wall, higher centres and nerves) where the amount of CO_2 excreted is less than that produced by metabolism. With respiratory pump failure, even with normal lung pathology, the arterial $PaCO_2$ is raised, with an inevitable fall in alveolar oxygen tension and hypoxaemia (alveolar hypoventilation). Hypoventilation results from a reduced respiratory effort, an inability to overcome resistive ventilatory forces

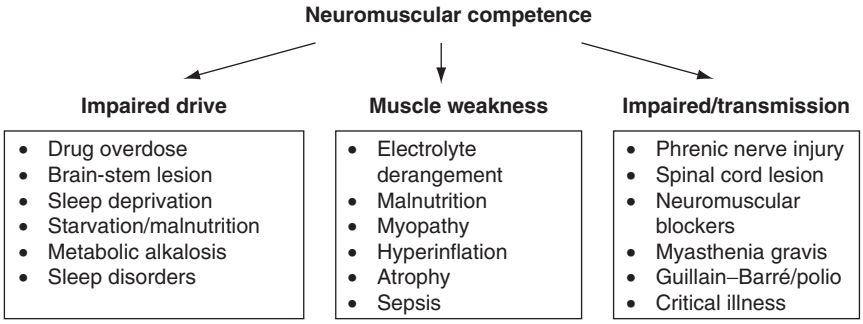


Figure 7.7 Neuromuscular competence.

Table 7.3 The typical clinical features of absolute type II ventilatory failure

Hypercarbia
Hypoxia
Reduced respiratory rate and or tidal volumes
Mixed respiratory failure

Table 7.4 Causes of acute alveolar hypoventilation

1. Chest wall, pleural and airway diseases
   - Loading (obesity)
   - Deformity (pectus/kyposcoliosis)
   - Upper airway obstruction (trachea/larynx)
   - Generalised disease (asthma/bronchitis/emphysema/cystic fibrosis)
2. Neuromuscular diseases and transmission
   - Spinal cord (injury/tumour)
   - Anterior horn cell (polio)
   - Peripheral neuropathy (Guillain-Barré)
   - Neuromuscular junction (Myasthenia gravis)
   - Muscle cell (dystrophies/myopathies)
   - Amyotrophic lateral sclerosis
3. Reduced central drive
   - Drugs (sedatives, pain killers)
   - Central nervous system disease (stroke, trauma)
4. Other
   - Sepsis
   - Shock

(Reproduced from Roussos and Koutsoukou (2003), with permission.)

or an inability to compensate for extra deadspace and/or CO_2 production. The respiratory muscle pump is as vital as the heart, failing in the same way and for the same reasons, but a large respiratory reserve is present and function may be markedly impaired without ventilatory failure accompaniment (Table 7.3).

It is worth noting at this point that a mixed picture of the two types of respiratory failure is frequently seen. While acute hypoxaemic failure may initially be present, some cases result in exhaustion. The patient is unable to compensate for the increased dead space, resulting in a raised $PaCO_2$ tension and mixed respiratory failure. Where individuals are unable to cough effectively or sigh, the risk of alveolar collapse, secretion retention and secondary infection is further increased. Others may be at risk of aspiration, for example unconscious or bulbar palsy, causing further damage to the lungs and further worsening ventilatory function.

Pathways to respiratory failure

The causes of respiratory failure are too numerous to detail but can be divided into two main categories:

- a reduction in neuromuscular competence resulting in the inability to generate adequate respiratory muscle force, for example reduced central drive, spinal injury, trauma, muscle disease (Figure 7.7);
- an increase in respiratory workload caused by raised ventilatory impedance, for example obstructive and restrictive lung defects.

Respiratory muscle dysfunction occurs as a result of various factors. This can be demonstrated by considering COPD, where respiratory muscle function is profoundly affected as a result of the increased work of breathing and the increased ventilatory load. The increased work of breathing arises from the pathological changes resulting in raised airway resistance and hyperinflation. At rest, the minute volume of these patients is higher than that of healthy subjects. As a result, the actual cost of breathing in terms of oxygen consumption is markedly increased and the accessory muscles of respiration are recruited.

Hyperinflation also has adverse effects upon the respiratory muscles. The increased lung volumes in COPD are thought to be compensated for by the lowering of the

diaphragm or expansion of the rib cage. These changes result in the muscles functioning at a disadvantaged position on the length-tension curve, i.e. the diaphragm is at its optimal length for providing the maximum contractile force when it is in its resting domed position.

As lung volume increases, the inspiratory muscles shorten and their ability to generate a negative force on inspiration is reduced. Hence, the inspiratory effort required to obtain the same tidal volume is greater. A completely flattened diaphragm is incapable of generating any useful pressure and on contraction causes in-drawing of the lower rib cage (Hoover's sign), effectively functioning as an expiratory muscle (see intrinsic positive end-expiratory pressure (PEEPi) below).

Respiratory muscle fatigue is an important precursor to respiratory failure. Factors affecting the endurance of the respiratory muscles include energy stores/nutrition, blood substrate concentration, arterial oxygen content, efficiency, mean inspiratory flow, minute ventilation, inspiratory duty cycle and maximum inspiratory pressure.

When fatigue of the respiratory muscles occurs, rest, not exercise, is indicated. However, a delicate balance between the two must be achieved as there is some evidence that if total rest is applied (through the application of full mechanical ventilation) disuse atrophy may occur causing weaning problems.

Increases in both elastic and resistive loads, as present with lung disease, will therefore increase the work of breathing and can lead to respiratory muscle fatigue and weakness, as described previously. When fatigue ensues, hypoventilation will result and weakened muscles will be unable to overcome the opposing forces of respiration to maintain adequate ventilation. Unless ventilatory support is provided to 'unload' the system, respiratory failure results.

The hypoxic drive concept

One of the causes of CO_2 retention in respiratory failure is the use of inappropriate levels of supplemental oxygen. This relates to only a small, but important, group of patients in whom the main ventilatory drive is hypoxaemia. Some patients with COPD develop severe hypoxaemia with some CO_2 retention. Owing to the degenerative pathological changes within the lungs, this is maintained over long periods of time and, while not referred to as respiratory failure, an increased work of breathing is usually encountered. Arterial pH is usually at the lower end of the normal range as renal compensation for the raised arterial CO_2 occurs with time and bicarbonate is retained (compensated respiratory acidosis). The cerebrospinal fluid also has a normal pH because of the raised bicarbonate levels and the main ventilatory drive now arises from the hypoxaemia (despite the raised arterial PCO_2).

The subsequent administration of high inspired oxygen fractions in such cases may pose a potentially lethal clinical scenario as the hypoxic drive will be abolished while the detrimental effects of the underlying lung condition and increased work of breathing continue. The result is gross respiratory depression with the arterial $PaCO_2$ climbing and arterial blood pH falling to extreme levels if left unnoticed or is misinterpreted in the clinical setting.

MECHANICAL VENTILATION

The goals of ventilatory support are clearly defined. They are to:

- improve alveolar ventilation;
- decrease the work of breathing;
- improve gas exchange.

Mechanical ventilatory support alone will not change the opposing forces of ventilation. The objective of mechanical ventilatory support is to reduce the impact of those forces until the abnormality resolves.

A potted history

Ventilation has a long history of development. There is some descriptive evidence of the artificial ventilation of animals via a reed or cane tube as early as the 1500s (Vesalius 1543). Perhaps the first patient reports date back to the 1700s when physicians used ironmongers' bellows to force air into their patients' lungs (Royal Humane Society 1774). Ventilation truly began in the 1830s, with negative pressure devices being the main ventilatory supports (Woollam 1976). The switch to positive pressure ventilation (PPV) did not occur until around 1952 when the ability to intubate and tracheostomise patients was established during the polio outbreak in the 1950s. Invasive ventilatory support using positive airway pressure soon became the main stem treatment, initially using volume-cycled ventilators (Figure 7.8).

Ventilatory support is usually given with the assistance of a mechanical device or ventilator. This can be done in one of two ways.

1. Invasively: the upper airway is bypassed using an endotracheal tube (ETT) or tracheostomy.
2. Non-invasively: the patient is given ventilatory support via the upper airway (not featured in this chapter).

In both methods air is mechanically driven through the patient's lungs to assist alveolar ventilation.

Invasive/conventional ventilation

Conventional (invasive) ventilation and the methods of cycling used have undergone different stages of development. Initially, volume ventilation was the norm, but 'ridged lung' problems were encountered, along with

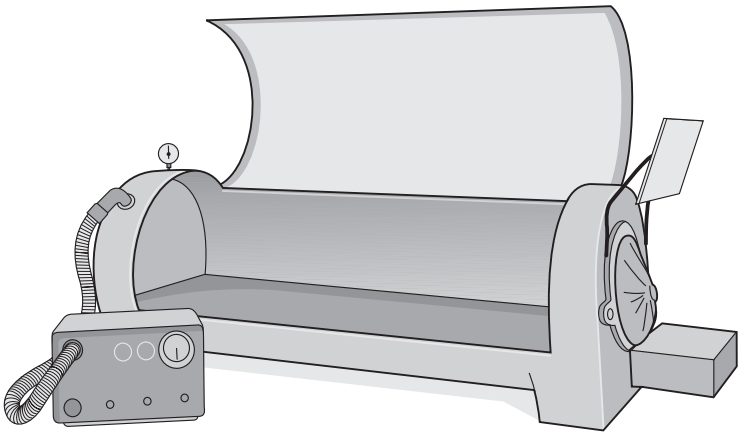


Figure 7.8 Porta lung ventilator. *(Courtesy of www.nemc.org/RespCare/portalng.htm.)*

patient and machine asynchrony (hence the term 'the patient is fighting the ventilator'). There are two primary methods of cycling used: volume and pressure controls.

Volume controlled ventilation

Volume controlled ventilation requires that a predetermined tidal volume be delivered to the lungs. A positive pressure is created at the airway, which continues to increase until the specified volume is achieved. Other controls to be set on a volume ventilator include the number of breaths per minute the patient will receive and the rate of flow at which the volume is delivered to the lungs. A machine delivering a *fixed* rate of flow for a specified time will guarantee a specified tidal volume but is actually *time-cycled*. Where flow measuring devices are sited at the Y connector, a true volume-cycled machine will compensate for leaks upstream of the connector where time-cycled will not (Sykes and Young 1999).

Volume ventilation meets the goals of ventilatory support in that it *guarantees* a specified amount of ventilation. This is appropriate today in the operating theatre setting. The disadvantages of volume controlled ventilation result from 'setting' (and, hence, enforcing) the variables of normal breathing – tidal volume (V_t), respiratory rate (RR) and the I : E ratio (i.e. the amount of time spent in inspiration versus expiration). The usual I : E ratio is 1 : 2 as inspiration is active, while expiration is largely passive and thus needs longer for air to empty from the alveolar units. If expiration is too short, air trapping will occur and PEEPi results (thus increasing the risk of lung trauma owing to excessive pressure).

With such controlled cycling, the patient must adapt to the ventilator rather than the ventilator adapting to the patient. This may cause dys-synchronisation between machine and patient, sometimes further increasing a patient's work of breathing, so indicating the need for sedation ± drug-induced paralysis until the disease process is resolved. In some instances, the patient may stop interacting with the ventilator. With disuse over time, the respiratory muscles atrophy and problems with weaning through muscle weakness and an inability to endure the requirements of normal resting breathing in an already compromised patient may be encountered. Ventilating pressures may be high leading to lung damage, and overventilation may occur if ventilatory requirements are overestimated.

Pressure controlled ventilation

Pressure controlled ventilation is frequently used to guarantee a maximum inflation pressure and reduce the risks of barotrauma (see complications). This mode can be used only when a pressure generator delivers increasing pressure throughout inspiration or with a flow generator. Minute ventilation (i.e. RR × V_t) cannot be guaranteed as with volume cycled ventilation (Sykes and Young 1999), as a pressure limited breath is delivered at a set rate. The volume generated is determined by the preset pressure but is also under the influence of the time spent in inspiration, the compliance of the lung and chest wall and airways resistance. Hence, hypoventilation and hypoxaemia may result with inadequate settings. However, the decelerating flow (as airway pressure rises and alveolar volume increases, the rate of flow slows) and maintenance of airway pressure over time, are more likely to inflate the more difficult non-compliant lung units.

Ventilation modes

Both volume and pressure control are cycles, rather than modes, of ventilation. As such, these controlled methods are significantly different to the spontaneous breathing cycle where the inspiratory flow and rate are of the individual's own choosing. In the 1970s and 1980s assisted modes that allowed spontaneous respiratory efforts were

introduced, and assist control and synchronised intermittent mandatory ventilation modes were developed for both volume and pressure controlled mechanisms. In turn, this led to the more sophisticated triggering and interactivity microprocessor developments of today's ventilators, resulting in full patient interaction. The ventilator was now becoming an interactive weaning device. However, concern regarding ventilator-induced damage became prevalent throughout the 1990s (Tobin et al. 2001) and protective strategies of alveolar recruitment using PEEP and lower tidal volumes to maintain a higher mean airway pressure and limiting plateau pressure emerged (ARDS Network 2000). Such protection strategies were detailed by MacIntyre in 2005.

Numerous modes (including dual modes enabling the combination of pressure limited with volume guaranteed ventilation) have since been developed with more sophisticated valves and control features, for example rise time (the speed with which the preset pressure/volume is reached), tube compensation as an automatic feature and active exhalation valves to name but a few. The aim of these is to enable improved patient comfort, synchrony and interaction, while allowing greater reductions in the risks of associated ventilatory lung trauma.

Pressure supported ventilation

In the last 10 years, pressure supported ventilation, as opposed to pressure controlled ventilation, has become the mode of choice as it offers better synchrony for the patient and the delivered flow can be more easily changed to suit differing lung and chest wall compliance. In this mode, a preset pressure acts as the limit while the patient is assisted but remains in control of all respiratory variables. Pressure support serves to reduce the work of breathing in inspiration and results in a larger tidal volume than a spontaneous breath alone by pushing the patient up the compliance curve. Where FRC is reduced, PEEP/continuous positive airways pressure (CPAP) can also be applied, which serves to restore the resting FRC back to the normal volume.

Advanced pressure modes

Airway pressure release ventilation (APRV), bilevel ventilation and proportional assisted ventilation (PAV) are advanced pressure modes. APRV mode is usually reserved for severe ARDS (where hypoxaemia is a major problem despite a high inspired oxygen fraction) and allows ventilator cycling between two different set CPAP levels (high and low) with the higher level providing the baseline pressure. The pressure is then intermittently 'released' to the lower CPAP level to enable CO_2 elimination. Bilevel ventilation is the same as APRV but allows spontaneous breathing at either CPAP level, with the spontaneous breaths being supported by either tube compensation or pressure support. The mode is well-tolerated and sedation is not usually necessary. This single mode can be used to provide full support through to facilitating weaning.

PAV is a form of ventilation that is fundamentally different from volume and pressure ventilation. In PAV mode, the ventilator generates a pressure change to cause airflow and move volume into the patient's lungs. Unlike conventional ventilation the pressure is generated in proportion to the patient's own inspiratory efforts. PAV tracks and responds to changes in the patient's breathing pattern/efforts and augments ventilation on a breath by breath basis. This means the patient has total control of breathing – their natural effort is simply amplified. The pressure delivered corresponds to changes in elastance, resistance and flow demand.

The development and efficacy of newer strategies concerning neurologically adjusted ventilatory assist modes are under consideration in the UK. This technology is based on a concept that couples the diaphragm and the mechanical ventilator by capture of the electrical signal from active diaphragmatic work and feedback to the ventilator for control of the assist portion required, as dictated by the patient's own physiological (neural) demand.

The complications of mechanical ventilation

Significant pulmonary and non-pulmonary complications may arise at any time as a direct consequence of intubation and mechanical ventilation, some of which are life-threatening. Intubation itself may cause upper airway trauma, loss of teeth, lacerations or trauma (including perforations) to the pharynx, vocal cords or trachea, and inadvertent intubation of the oesophagus or right main bronchus. Longer-term intubation may result in sinusitis, necrosis/stenosis of the trachea and glottic oedema. Retained pulmonary secretions are common in the ventilated patient as the cough reflex is impaired, mucociliary transport is reduced and mucus production increased (Plevak and Ward 1997).

Pulmonary complications

Pulmonary complications that commonly occur are atelectasis, infection and alveolar over-distension (causing the life-threatening problems of hypotension, and barotrauma or volutrauma (also referred to as atelectrauma)).

Atelectasis

Atelectasis is a common problem in the ventilated individual. Causative factors include aspiration, hypoventilation, mucus plugging and secretion retention. Intubation of the right main bronchus may cause over distension of the right lung (which may be reflected by high inflation pressures), while the left lung becomes atelectatic. A loss

of lung volume, as found with atelectasis and lobar/total lung collapse, will be evident on chest radiograph as a shift toward the affected side with increased opacity and diaphragmatic elevation or tenting.

Aspiration (a particular risk at the time of intubation) is most likely to affect the right lung in the supine position owing to the anatomy of the right main bronchus as it branches from the trachea (more vertically than the left). Correct positioning of the ETT and its patency should be checked along with the auscultation of breath sounds on a regular basis. The correctly placed tube should be appropriately secured and the weight of the circuit supported to prevent loss of alignment (and subsequent misplacement/accidental extubation, or erosion and trauma) within the trachea.

Ventilator-induced lung injury

This consists of barotrauma and volutrauma caused by over-distension of the alveolar units. *Barotrauma* is the rupture of alveoli resulting in the tracking of air to the pleura (pneumothorax) or mediastinum (pneumomediastinum). Predisposing risk factors include large tidal volumes and high peak (pressure in the main airways) and plateau (pressure in the small airways) pressures, although predisposing lung pathology may be a better indication of risk. There is usually a gradient between the peak and plateau pressures. However, ventilating pressures below 45 mmHg and 35 mmHg respectively are recommended. The I : E ratio is an important consideration in COPD sufferers requiring ventilatory support owing to the increased risk of PEEPi development in the presence of increased airways resistance. Manipulation of parameters affecting the I : E ratio may, therefore, be required, such as increasing the inspiratory flow rate or reducing the ventilatory rate.

Volutrauma (the over-distension of alveoli) has led to the development of lung protection strategies more prevalent today of lower tidal volumes (6–8 mL/kg). When delivering PPV, air takes the path of least resistance. Thus, normal or relatively normal alveoli are ventilated more easily than collapsed or consolidated lung units and the potential for over-distension (resulting in an increased risk of activating an inflammatory reaction of the normal areas) is established. MacIntyre (2005) emphasises that the forces associated with the repeated opening and closing of alveoli through tidal gas exchanges is linked to worsening inflammatory cascades, so those with acute lung injury or ARDS are recommended protective strategies of PEEP with low volumes and a higher plateau airway pressure to avoid such forces, with a speedier wean from ventilation through adherence to respiratory therapy-led weaning protocols (Ely et al. 1996).

In the mechanically-ventilated patient, a positive pressure (CPAP/PEEP) may be applied at the end of expiration (Figure 7.9) in order to recruit the dependent alveoli. In addition to moving the patient onto the more favourable steep portion of the lung compliance (pressure/volume) curve (a mechanically advantageous position for ventilation), this splinting effect on the airway also enables a longer period of time for oxygen to diffuse across the alveolar-capillary membrane, thus improving the oxygenation status of the patient.

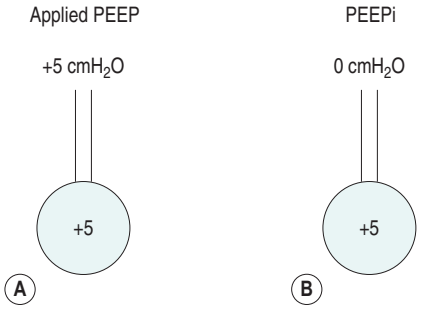


Figure 7.9 Diagram to demonstrate PEEP and $PEEP_i$. (a) Applied PEEP. (b) PEEPi.

PEEPi

PEEPi (Figure 7.9) may develop automatically within the airway, for example because of chronic lung diseases, inappropriate settings of applied ventilatory support and clinical dynamic hyperinflation states, such as status asthmaticus. PEEPi occurs when the expiration phase is inadequate in duration for the lung to fully empty prior to the next inspiratory breath (regardless of type). As such, a proportion of each subsequent tidal volume may be retained and if undetected, result ultimately in barotrauma, volutrauma, hypotension, patient-machine dyssynchrony and even death.

Oxygen toxicity

Oxygen toxicity (owing to the production of oxygen free radicals) is associated with higher maintenance levels of inspired oxygen fractions and duration of use. It results in complications such as absorption atelectasis, hypercarbia, tracheobronchitis and diffuse alveolar damage. The lowest level of fractional inspired oxygen (FiO_2) required while maintaining adequate oxygenation is recommended, with downward titrations encouraged as soon as possible. The exact level thought to be responsible is as yet unknown, although damage has been reported with maintenance levels of 50%. Again, the application of PEEP may help with the oxygenation status of the individual and the lowering of inspired oxygen fractions if the pressure is haemodynamically tolerated.

Ventilator associated pneumonia

Ventilator associated pneumonia (VAP), a life-threatening complication (with reported mortality rates of up to 50%) (Rea-Neto et al. 2008), poses a significant risk and is reported to occur in 13–35% of mechanically-ventilated

patients. Early onset pneumonia (within 48–72 hours of intubation) may be a result of aspiration at the time of intubation. Most are caused by antibiotic-sensitive organisms and flora of the upper airway, such as *Staphylococcus aureus*, *Haemophilus influenzae* and *Streptococcus pneumoniae*. Infections after 72 hours are thought to be caused by antibiotic-resistant pathogens, for examople *Pseudamonas aeruginosa* (prevalent in percutaneous tracheostomised patients, as shown by Rello et al. 2003) and acinetobacter species. Often precursors to VAP are bacterial colonisation of the aerodigestive tract and aspiration of contaminated secretions into the lower airways (Kollef 2002; Byrd et al. 2006). VAP should be suspected if there is evidence of pyrexia, radiological changes and purulent pulmonary secretions. Cultures obtained from pulmonary aspirate may not reliably differentiate between pneumonia and tracheal bacterial colonisation. Secretions obtained via fibreoptic bronchoscopy may be useful, but this technique poses the risk of PEEPi development and poor tidal volumes in the ventilated patient (Lawson et al. 2000). (Preparation and ventilatory alterations may be required prior to this procedure, e.g. the ETT internal diameter should be at least 2 mm greater than the external diameter of the bronchoscope.) Of note in the longer-term ventilated patient, Baram et al. (2005) found that stable patients with no clinical signs of pneumonia carry a higher burden of bacteria that is usually greater than the commonly accepted threshold diagnostic of VAP. Georges et al. (2000) also recommend that those requiring tracheotomy with evidence of bronchial colonisation and pyrexia should have the procedure delayed to prevent the risk of acquiring early PAV. Predisposing risk factors for the acquisition of VAP include surgical procedures (thoracic and upper abdominal), nasogastric tube use, supine position, reintubation, previous antibiotics, chronic lung disease, and use of histamine 2 receptor antagonists and antacids (Celis et al. 1988; Torres et al. 1990). There is recognition that bacterial colonisation of the upper airway may cause contamination of the lower airway despite the use of a tracheal cuff (Feldman et al. 1999) and that there is usually bacterial colonisation in tracheostomised patients on longer-term ventilatory support (Lusardi et al. 2003).

The literature favours the use of initial broad-spectrum antibiotics until the sensitivities of the causative agents are identified. These will usually be tailored to local organisms and antimicrobial resistance. The aspiration of subglottic secretions is also important in reducing the risk of upper tract colonisation and vigilant cuff management should be adopted to clear secretions pooling there (Girou et al. 2004). Gastric distension should be avoided and a semi-recumbent position used (Girou et al. 2004) to reduce the risk of aspiration (Orozco-Levi et al. 1995). Avoidance of unnecessary antibiotic administration and rotation of such medication may also be useful in prevention tactics.

Non-pulmonary complications

These include venous thromboembolism (with predisposing risk factors of trauma, immobilisation, obesity, cardiac failure and underlying malignancy) requiring prophylaxis, for example anticoagulation and elastic stockings; various gastrointestinal problems (Mutlu et al, 2001) (predisposing factors include burns, coagulopathy and head injury) owing to stress ulceration requiring gastric protection strategies; pressure sores which may lead to sepsis or osteomyelitis; neuromuscular weakness and atrophy; and other organ failure.

The cardiovascular system is also inevitably affected in some way with the use of mechanical ventilation. The application of PPV may cause a reduction in preload, stroke volume and cardiac output, which may then reduce renal perfusion and, ultimately, renal function with fluid accumulation. Maintenance of such positive thoracic pressure can also cause a reduction in the return of blood from the head resulting in a raised intracranial pressure and delirium and agitation, making the already complex multi-factorial management even more difficult.

THE ROLE OF THE RESPIRATORY PHYSIOTHERAPIST

This role in intensive care is considered by many to be an integral part of the overall management of the acutely ill patient, although evidence is scant and varied, with further controlled research being required in many areas. The purpose and effectiveness of this area of physiotherapy has been questioned by Templeton and Palazzo (2007), but the lack of detail over the clinical decisions made and the physiotherapy management during this randomised controlled trial has affected the transferability of the results. The trial did question the routine respiratory physiotherapy management of the ventilated patient, although it was controversial because of the quality of the trial, but the importance of emergency on-call care was recognised and not denied to patients. This trial may question the approach to physiotherapy respiratory care and therapists should be seen to treat only when needed from the thorough respiratory assessment rather than routine care.

In future years, the challenge for physiotherapists on intensive care units will be to combine the vital respiratory care needed to the rehabilitation needs of the patient within critical care. Recent guidance from the National Institute of Health and Clinical Excellence (NICE) has highlighted this requirement. For a physiotherapist on an intensive care unit, various techniques will be used which will range from being a generic skill (i.e. exercises and mobilisation) to a specialised respiratory skill (i.e. manual hyperinflation). The specialist treatment strategies to the critical care patient will be discussed in detail in this chapter.

Pulmonary interventions

Interventions should be based upon assessment findings and must include at least an ability to problem-solve some basic issues with the ventilatory equipment, for example factors affecting patients' co-ordination of breathing with the ventilator (Tobin et al. 2001), tube patency, development of dynamic hyperinflation and intrinsic PEEP, and discuss possible corrective measures with the multi-disciplinary team; and an understanding of the ventilator alarms and their significance. In order to do this, a sound knowledge (the background to which has been provided earlier in this chapter) of respiratory mechanics is essential. If disconnection from the ventilator for any reason is required, the physiotherapist must be able to safely reinstate ventilatory support and check all mechanical and physiological parameters. This should not be assumed to be the responsibility of the accompanying nurse, while competence with managing the respiratory equipment and monitors should be gained prior to the first emergency on-call duty.

Suction

Aspiration of pulmonary secretions via the artificial airway is used for secretion clearance only when the patient is unable to do this independently. It should never be used 'routinely' or prophylactically, as research demonstrates undesired effects, for example tracheobronchial trauma (Judson and Sahn 1994), hypoxaemia and destabilisation of haemodynamics (Paratz 1992), although these are limited with the use of pre-oxygenation, reassurance and sedation, if required. To avoid unnecessary suction passes, Guglielminotti et al. (2000) explored the bedside detection of retained secretions in the ventilated patient. They recommend that a sawtooth pattern, as seen on the ventilator's flow-volume loop, and/or respiratory sounds over the trachea are good indicators (especially when there is a combined presence) for the need for tracheal suction in the ventilated patient, thus enabling performance of the technique on an 'as needed' basis.

Many units now use closed suction catheters for a ventilated patient, which takes away the need for a sterile open procedure for suction. This is aimed at reducing the risk of introducing new microorganisms to the airways but, interestingly, Siempos et al. (2008) found no differences in the incidence of VAP when comparing the two methods of suction. Despite these results, closed suction may be preferred in units because of the ease of technique, but the physiotherapist must be aware of both in cases of emergency respiratory management.

Positioning

The use of specific body positioning in the intensive therapy unit is aimed at optimising oxygen transport via improving ventilation to perfusion (V/Q) matching, promoting mucociliary clearance, improving aeration through increased lung volumes and reducing the work of breathing. It has been suggested that frequent re-positioning may decrease the incidence of VAP (Clini and Ambrosino 2005) and this has resulted in the introduction of technology to assist this. Continuous turning beds have been studied and despite suggestions that they assist sputum clearance (Davis et al. 2001), they were found not to affect the patient's length of stay in the intensive care unit or days requiring ventilation (Goldhill et al. 2007).

When considering the positioning of the patient it must be remembered that the semi-recumbent position at 45° has been found to reduce the incidence of acquired pneumonia by preventing the risk of gastric reflux (Alexiou et al. 2009). Also, upright positioning may be required for weaning non-tetraplegic patients. Respiratory mechanics are severely altered in the cervical and upper thoracic complete spinal cord-damaged patient, and the supine position should be adopted with no more than a 10° tilt.

Manual hyperinflation

Manual hyperinflation, which has still not been overtly described owing to the many variations in technique used, requires the patient to be disconnected from the ventilator and attached to a manual circuit for the purpose of hyperventilating the lungs in order to reinflate atelectatic lung units (via collateral ventilatory channels). The potential to both hypoventilate and over distend the lungs exists with this modality and it is recommended that a simple pressure gauge be used in circuit to minimise the risk of barotraumas (Figure 7.10). Perform a slow deep inspiration, ensuring the pressure does not exceed 40–50 cmH_2O (Denehy 1999). It has previously been described as '…a technique that uses a manual bag circuit to deliver a breath of 1.5 times greater than that being delivered by the ventilator, where the ventilator is recording a tidal volume of up to 700 mls' (Clapham et al. 1995, McCarren and Chow 1998).

Evidence is inconsistent concerning the efficacy of this technique owing to variables in studies. However, manual hyperinflation is thought to be particularly useful in

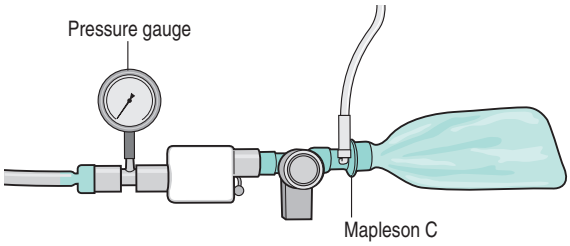


Figure 7.10 Diagram to show integration of a simple pressure gauge in circuit.

reinflating atelectatic areas of lung and thus improving pulmonary compliance (Clarke et al. 1999; Clini and Ambrosino 2005) and oxygenation (Blattner et al. 2008), providing a more efficient breathing pattern. It has been found to reduce airway resistance (Choi and Jones 2005) and facilitate sputum clearance (Savian et al. 2006). This technique is traditionally seen as a key physiotherapeutic technique, but should only be used if indicated by the respiratory assessment.

Despite the advantages of this intervention, it must be remembered that rises in intracranial pressure and significant drops in blood pressure have been reported during the technique, as airway pressure, the amount of PEEP applied, flow rates and the FiO_2 appear to be very variable (Jones et al. 1992; Glass et al. 1993; Denehy 1999).

The technique used should ensure that the weight of the tubing and valve is supported to avoid tracheal tube displacement throughout the episode of intervention. It should be performed ideally using a two-handed technique (especially if the hands are small), rotating the hand at the reverse of the bag at a rate that matches the baseline minute ventilation of the patient (Clapham et al. 1995). The therapist should observe the chest and listen to feedback from the therapist performing the chest wall vibrations to gauge the lung expansion obtained. The peak inflation volume is held for a couple of seconds to obtain a plateau, and then, if utilised, begin vibrations. The bag is then released to allow rapid expiration. Manual vibrations should be stopped midway through the expiration phase to avoid collapsing the airways; however, full expiration must occur prior to the next manual hyperinflation. The techniques should be repeated 5–6 times or as clinically indicated. Baseline parameters should be monitored throughout the process and changes noted. Sudden alterations to the cardiovascular stability of the patient should result in discontinuation of the process, and effects documented alongside the recovery time and any adverse effects. On completion of the intervention the patient should be returned back to the pre-intervention ventilation regime and observed for cardiorespiratory stability. Outcomes of treatment and the future plan should be clearly recorded. Note: synchronisation with the conscious spontaneously breathing patient is essential to the success of this procedure.

Because the intervention involves the disconnection of the ventilator circuit and a potential loss of pressure and de-recruitment of alveoli, some therapists have begun to deliver hyperinflations via the ventilator. Savian et al. (2006) compared ventilator hyperinflation with manual hyperinflation and found increased lung compliance with the ventilator technique. If hyperinflation via the ventilator is to be attempted, agreement with the multiprofessional team must be agreed and parameters returned to pre-technique levels following completion.

The clearance of oropharyngeal secretions should be undertaken on a daily basis to remove stagnant pooled secretions from around the cuffed tube. It is most effectively done with continuous manual pressurisation of the lower airway to expel secretions as soon as the cuff is deflated to avoid bacterial contamination of the lower respiratory tract. Upon cuff reinflation, the cuff volumes used should be in accordance with findings from occasional cuff pressure checks to avoid tracheal pressure damage, and the volume of air required to adequately and safely seal the cuff, should be documented.

Manual techniques

Manual techniques are often used in conjunction with manual hyperventilation, such as shaking and vibrations to facilitate mucus clearance (Denehy 1999). Few well-designed studies have been performed in this specific area and a more recent animal study (Unoki et al. 2005) is inappropriately misleading in its recommendations regarding the use of external rib cage compression in the ventilated patient. However, care should be taken to ensure that compressions to the chest wall are terminated at mid expiratory lung volumes to avoid collapsing the airways and reducing FRC.

Percussion has been shown to have detrimental effects, for example arrhythmias and reduced lung compliance in the critically ill individual (Hammon et al. 1992; Jones et al. 1992). Despite this, Davis et al. (2001) discovered that percussion did aid sputum clearance in the copiously-productive ventilated patient when investigating the effectiveness of continuously turning beds. The use of vibrations in the intensive therapy unit to facilitate reaeration of atelectatic areas is not supported radiographically (Stiller et al. 1996).

Other considerations

While some aspects and combinations of physiotherapy interventions have been considered in light of available evidence, other inputs may also form part of the respiratory therapist's role such as that of leading on weaning protocols (the transfer of the work of breathing from the ventilator back to the patient). As mentioned previously, faster weaning episodes may be expected with the implementation of respiratory therapy-led protocols (Marelich et al. 2000; MacIntyre 2005).

REHABILITATION FOR THE CRITICAL CARE POPULATION

This area of physiotherapy practice is gaining greater emphasis within the critical care environment. There have been more studies exploring what is involved, who should receive rehabilitation and the safety of this treatment, with findings considered below. Additionally, recent guidance

from NICE, and the appropriateness and effectiveness of this area of practice are explored.

Who needs critical care rehabilitation?

This is a very good question and one that is not very easily answered without having knowledge of the patient's case history. The physiotherapist will have detailed knowledge of their own case load. When assessing and treating the critical care patient, weakness and rehabilitation needs must not be forgotten following their critical care stay, as there may be a deleterious impact for the individual upon their level of dependency and quality of life post-discharge.

The reported prevalence of problems in returning to function varies, but the incidence remains surprising. De Jonghe et al. (2000) identified that 25.3% of patients requiring ventilation for longer than seven days had some form of critical care acquired paresis. The quality of life and function of patients 1 year after a critical care stay requiring ventilation for 48 hours or more showed that 54% had restrictions in daily functioning, with walking and mobility being the most commonly affected (van der Schaaf et al. 2009a), and 69% were restricted in performing daily activities, with only 50% having returned to work (van der Schaaf et al. 2009b). Thus, a high proportion of patients will need some form of rehabilitation during or following their intensive care unit stay.

However, when to introduce rehabilitation must also be considered. A number of studies have looked at when rehabilitation can begin, depending on neurological, cardiac and respiratory criteria, and have experienced minimal problems as a consequence (Stiller and Phillips 2004; Bailey et al. 2007). Stiller and Phillips (2003) recommended to aim for less than 50% of maximal heart rate during rehabilitating and a PaO_2/FiO_2 ratio of more than 300 before commencing rehabilitation. Alternatively, Bailey et al. (2007) found that adverse events only occurred in 0.96% of instances using the neurological (response to stimuli), respiratory (FiO_2 above 0.6 and PEEP below 10 cmH_2O) and circulatory (absence of orthostatic and catecholamine drips) assessment criteria.

The effects of weaning the patient from mechanical ventilation must be remembered when deciding whether a patient is ready for rehabilitative treatment. Rehabilitation may not be successful when patients have an increased demand on the respiratory muscles with decreased respiratory support. It is suggested that rehabilitation treatments should take place during periods of higher respiratory support (Stiller and Phillips 2003).

What is critical care rehabilitation?

Again this is not an easy question to answer as the evidence for rehabilitation and what this should consist of is scarce. Patients can suffer psychological and physical problems both during and following an intensive care unit admission. A fundamental component of physiotherapy practice within the intensive care unit is physical rehabilitation but the structure of this varies widely. Treatment may start with positioning and passive movements in the unresponsive patient and progress to assisted, active and resisted exercises when patient cooperation and progress is obtained. The aims of intervention are those of maintaining or improving joint range of motion, soft tissue length, muscle strength and function, and decreasing the risk of thromboembolism.

There are a wide variety of techniques employed and frequency of use within the literature. A study by Stiller et al. (2004) described techniques such as lying to sitting, sitting to standing, transfers and walking as components of their global management. Thomas et al. (2009) included passive rehabilitation, which included hoist and slide transfers to a chair. Although this included minimal patient participation, they concluded that these activities had an important role in the rehabilitation of critically ill patients. The active rehabilitation included active assisted and active muscle strengthening exercises along with those techniques described by Stiller et al. (2004).

Perme and Chandrashekar (2009) described a four-phase rehabilitation programme with criteria for progressing onto the next phase. The programme encompassed progressive mobilisation and walking with the progression based on a patient's functional capability and ability to tolerate the prescribed activity. Schweickert et al. (2009) used passive movements in unresponsive patients, moving onto active assisted and active exercises when patients became more aware of treatment. Treatment could then be advanced to include bed mobility and sitting, progressing onto activities of daily living. They found this strategy for whole body rehabilitation during interruptions of sedation in the earliest days of critical illness was safe and well tolerated.

Guidance and evidence base

The effects of rehabilitation in the critical care setting have been investigated but with limited detail concerning the rehabilitation involved, therefore findings have limited use within clinical practice. Rehabilitation has been found to significantly improve exercise tolerance testing (Nava 1998), strength (Zanotti et al. 2003; Chiang et al. 2006) and inspiratory pressures (Nava 1998; Chiang et al. 2006) in patients during and following mechanical ventilation; however, the lack of detail of the rehabilitation strategies used make these findings less applicable to the clinical setting. Schweickert et al. (2009) discovered that rehabilitation during daily sedation stops for patients requiring mechanical ventilation for more than 72 hours, and improved return to independent functional status for 59% of patients in the intervention group, compared with 35%

of patients in the control group receiving routine care. Although the control group treatment was not clear, the rehabilitation strategies involved active assisted exercises in supine and progressed to walking when appropriate. These strategies could be applied to physiotherapy practice in this population.

NICE have recommended that rehabilitation starts as early as possible for patients at risk and therefore supports the need of rehabilitation within the critical care setting (NICE 2009). The guideline development group also recommended that it was good practice 'to provide individualised, structured rehabilitation at each key stage of the patients' rehabilitation pathway' for those patients with a recognised need at assessment. Therefore, these guidelines not only support a detailed and structured assessment of rehabilitation needs, but also provision of rehabilitation within the critical setting and all the way through to post-discharge care.

There is limited evidence looking at the effect of an outpatient physiotherapy-led exercise programme for patients following discharge from the intensive care unit. McWilliams et al. (2009) assessed the impact of an outpatient-led rehabilitation programme on exercise capacity and anxiety and depression scores in a cohort of adult intensive care survivors. Although this was a small study and the patients were not randomised, the results showed improvement in both these parameters. Van der Schaaf et al. (2009a) led a cross sectional study to look at restrictions in daily functioning and thereby identify prognostic factors for critically ill patients one year after discharge from the intensive care unit. They concluded that patients who stayed in the intensive care unit for at least two days are a potential target population for rehabilitation medicine, but as known prognostic factors had only limited predictive value for the development of functional restrictions, further work is necessary in this area to establish which groups would benefit.

FURTHER READING

AARC (American Association for Respiratory Care) Clinical Practice Guidelines (various), www.rcjournal.com/cpgs/index.cfm.

Heffner, J.E., 2005. Organization of care for people in long-term artificial ventilation: Management of the chronically ventilated patient with a tracheostomy. Chron Respir Dis 2, 151–161.

Hinds, C.J., Watson, J.D., 1995. Intensive Care: A Concise Textbook, second ed. WB Saunders, Philadelphia.

MacIntyre, N.R., Cook, D.J., Ely Jr., E.W., et al., 2001. Evidence-based guidelines for weaning and discontinuing ventilatory support: a collective task force facilitated by the American College of Chest Physicians; the American Association for Respiratory Care; and the American College of Critical Care Medicine. Chest 47, 69–90.

MacIntyre, N.R., Epstein, S.K., Carson, S., et al., 2005. Management of patients requiring prolonged mechanical ventilation. Report of a NAMDRC Consensus Conference. Chest 128 (6), 3937–3954.

Seaton, A., Leitch, A.G., Seaton, D., 2000. Crofton and Douglas's Respiratory Diseases, fifth ed. Blackwell, Oxford.

Thomas, A.J., 2009. Exercise intervention in the critical care unit – what is the evidence? Phys Ther Rev 14 (1), 50–59.

Tobin, M.J., 1988. Respiratory muscles in disease. Clin Chest Med 9 (2), 263–286.

Tobin, M.J., Jubran, A., Laghi F., 2001. Patient-ventilator interaction. Am J Respir Crit Care Med 163 (5), 1059–1063.

REFERENCES

Alexiou, V.G., Ierodiakonou, V., Dimopoulos, G., et al., 2009. Impact of patient position on the incidence of ventilator-associated pneumonia: A meta-analysis of randomized controlled trials. J Crit Care 24, 515–522.

ARDS Network, 2000. Ventilation with lower tidal volumes as compared with traditional tidal volumes for lung injury and the ARDS. N Engl J Med 342, 1301–1308.

Bailey, P.R., Thomsen, G.E.M., Spuhler, V.J.R., et al., 2007. Early activity is feasible and safe in respiratory failure patients. Crit Care Med 35 (1), 139–145.

Baram, D., Hulse, G., Palmer, L.B., 2005. Stable patients receiving prolonged mechanical ventilation have a higher alveolar burden of bacteria. Chest 127 (4), 1353–1357.

Blattner, C., Guaragna, J.C., Saadi, E., 2008. Oxygenation and static compliance is improved immediately after early manual hyperinflation following myocardial revascularisation: a randomised controlled trial. Aust J Physiother 54, 173–178.

Byrd Jr., R.P., Eggleston, K.L., Takubo, T., et al., 2006. Mechanical

Ventilation, http://emedicine.medscape.com/article/304068-overview, accessed October 2012.

Celis, R., Torres, A., Gatell, J., et al., 1988. Nosocomial pneumonia: a multivariate analysis of risk and prognosis. Chest 93, 318–324.

Chiang, L.L., Wang, L.Y., Wu, C.P., et al., 2006. Effects of physical training on functional status in patients with prolonged mechanical ventilation. Phys Ther 86 (9), 1271–1281.

Choi, J.S.-P., Jones, A.Y.-M., 2005. Effects of manual hyperinflation and suctioning on respiratory mechanics in mechanically ventilated patients with ventilator-associated pneumonia. Aust J Physiother 51, 25–30.

Clapham, L., Harrison, J., Raybould, T., 1995. A multi-disciplinary audit of manual hyperinflation technique (sigh breath) in neurosurgical intensive care unit. Intensive Crit Care Nurs 11 (5), 265–271.

Clarke, R.C.N., Kelly, B.E., Convery, P.N., et al., 1999. Ventilatory characteristics in mechanically ventilated patients during manual hyperinflation for chest physiotherapy. Anaesthesia 54, 936–940.

Clini, E., Ambrosino, N., 2005. Early physiotherapy in the respiratory intensive care unit. Respir Med 99, 1096–1104.

Davis Jr., K., Johannigman, J.A., Campbell, R.S., et al., 2001. The acute effects of body position strategies and respiratory therapy in paralyzed patients with acute lung injury. Crit Care 5 (2), 81–87.

De Jonghe, B., Sharshar, T., Lefaucheur, J.P., et al., 2000. Paresis acquired in the Intensive Care unit: A prospective multicenter study. JAMA 288 (22), 2859–2867.

Denehy, L., 1999. The use of manual hyperinflation in airway clearance. Eur Resp J 14, 958–965.

Ely, E.W., Baker, A.M., Dunagan, D.P., et al., 1996. Effect on the duration of mechanical ventilation of identifying patients capable of breathing spontaneously. N Engl J Med 335, 1864–1869.

Feldman, A., Kassel, M., Cantrell, J., et al., 1999. The presence and sequence and endotracheal tube colonization in patients undergoing mechanical ventilation. Eur Resp J 13, 546–551.

Georges, H., Leroy, O., Alfandari, S., et al., 2000. Predisposing factors for nosocomial pneumonia in patients receiving mechanical ventilation and requiring tracheotomy. Chest 118 (3), 767–774.

Girou, E., Buu-Hoi, A., Stephan, F., et al., 2004. Airway colonisation in long-term mechanically ventilated patients. Effect of semi-recumbent position and continous subglottic suctioning. Intensive Care Med 30, 225–233.

Glass, C., Grap, M.J., Corley, M.C., et al., 1993. Nurses ability to achieve hyperinflation with a manual resuscitation bag during endotracheal suctioning. Heart Lung 22, 158–165.

Goldhill, D.R., Imhoff, M., McLean, B., et al., 2007. Rotational bed therapy to prevent and treat respiratory complications: a review and meta-analysis. Am J Crit Care 16, 50–62.

Guglielminotti, J., Alzieu, M., Maury, E., et al., 2000. Bedside detection of retained tracheobronchial secretions in patients receiving mechanical ventilation. Chest 118 (4), 1095–1099.

Hammon, W.E., Connors, A.F., McCaffree D.R., 1992. Cardiac arrythmias during postural drainage and chest percussion of critically ill patients. Chest 102, 1836–1841.

Johnson, M.A., Polgar, J., Weightman, D., 1973. Data on the distribution of fibre types in 31 human muscles. An autopsy study. J Neurol Sci 18, 111–129.

Jones, A.Y.M., Hutchinson, R.C., Oh, T.E., 1992. Effects of bagging and percussion on static lung compliance of the respiratory system. Physiotherapy 78, 661–666.

Judson, M.A., Sahn, M.A., 1994. Mobilisation of secretions in ICU patients. Resp Care 39, 213–226.

Kollef, M., 2002. Respiratory failure: complications of mechanical ventilation. ACP Medicine Online, *http://www.medscape.com/viewarticle/534431_print*.

Lawson, R.W., Peters, J.I., Shelledy, D.C., 2000. Effects of fibreoptic bronchoscopy during mechanical ventilation in a lung model. Chest 118, 824–831.

Lusardi, M., Capelli, A., De Stephano, A., et al., 2003. Lower respiratory tract infections in chronic obstructive pulmonary disease outpatients with tracheostomy and persistent colonization by *P. aeruginosa*. Respir Med 97, 1205–1210.

MacIntyre, N.R., 2005. Current issues in mechanical ventilation for respiratory failure. Chest 128 (5), 561S–567S.

Marelich, G.P., Murn, S., Battistella, F., et al., 2000. Protocol weaning off mechanical ventilation in medical and surgical patients by respiratory care practitioners and nurses: Effect on weaning time and incidence of ventilator-associated pneumonia. Chest 188, 459–467.

McCarren, B., Chow, C.M., 1998. Description of manual hyperinflation in intubated patients with atelectasis. Physiother Theory Pract 14 (4), 199–210.

McWilliams, D.J., Atkinson, D., Carter, A., et al., 2009. Feasibility and impact of a structured, exercise-based rehabilitation programme for intensive care survivors. Physiother Theory Prac 25 (8), 566–571.

Mutlu, G., Mutlu, E.A., Factor, P., 2001. GI complications in patients receiving mechanical ventilation. Chest 119 (4), 1222–1241.

Nava, S., 1998. Rehabilitation of patients admitted to respiratory intensive care. Arch Phys Med Rehabil 79 (7), 849–854.

NICE (National Institute for Clinical Excellence), 2009. Rehabilitation after Critical Illness; www.nice.org.uk/CG83, accessed October 2012.

Orozco-Levi, M., Torres, A., Ferrer, M., et al., 1995. Semirecumbent position protects from pulmonary aspiration but not completely from gastroesophageal reflux in mechanically ventilated patients. Am J Respir Crit Care Med 152, 1387–1390.

Paratz, J., 1992. Haemodynamic stability of the ventilated intensive care patient: a review. Aust J Physiother 38, 167–172.

Perme, C., Chandrashekar, R., 2009. Early mobility and walking program for patients in intensive care units:

Creating a standard of care. Am J Crit Care 18 (3), 212–221.

Plevak, D.J., Ward, J.J., 1997. Airway management. In: Burton, G.G., Hodgkin, J.E., Ward, J.J. (Eds.), Respiratory Care: A Guide to Clinical Practice, fourth ed. Lippincott, Philadelphia, pp. 555–609.

Rea-Neto, A., Youssef, N.C., Tuche, F., et al., 2008. Diagnosis of ventilator-associated pneumonia: a systematic review of the literature. Crit Care 12 (2), R56.

Rello, J., Lorente, C., Diaz, E., et al., 2003. Incidence etiology and outcome of nosocomial pneumonia in ICU patients requiring percutaneous tracheostomy for mechanical ventilation. Chest 124 (6), 2239–2243.

Roussos, C., Koutsoukou, A., 2003. Respiratory failure. Eur Respir J 22 (Suppl. 47), 3S–14S.

Savian, C., Paratz, J., Davies, A., 2006. Comparison of the effectiveness of manual and ventilator hyperinflation at different levels of positive end-expiratory pressure in artificially ventilated and intubated intensive care patients. Heart Lung 35, 334–341.

Schauf, C., Moffett, S., Moffett, D., 1990. Human Physiology: Foundations & Frontiers. Times Mirror, Mosby College Pub.

Schweickert, W.D., Pohlman, M.C., Pohlman, A.S., et al., 2009. Early physical and occupational therapy in mechanically ventilated, critically ill patients: a randomised controlled trial. Lancet 373, 1874–1882.

Siempos, I.I., Vardakas, K.Z., Falagas, M.E., 2008. Closed tracheal suction systems for prevention of ventilatory-associated pneumonia, Br J Anaesth 100 (3), 299–306.

Stiller, K., Jenkins S., Grant, R., et al., 1996. Acute lobar atelectasis:a comparison of five physiotherapy regimens. Physiother Theory Prac 12, 197–209.

Stiller, K., Phillips, A., 2003. Safety aspects of mobilising acutely ill inpatients. Physiother Theory Prac 19, 239–257.

Stiller, K., Phillips, A., Lambert, P., 2004. The safety of mobilization and its effect on haemodynamic and respiratory status of intensive care patients. Physiother Theory Prac 20, 175–185.

Sykes, K., Young, D., 1999. Respiratory Support in Intensive Care, second ed. Blackwell Publishing, Oxford.

Templeton, M., Palazzo, M., 2007. Chest physiotherapy prolongs duration of ventilation in the critically ill ventilated for more than 28 hours. Intensive Care Med 33 (11), 1938–1945.

Thomas, A.J., Wright, K., Mill, L.M., 2009. The incidence of physiotherapy and rehabilitation activities within a general Intensive Care Unit. J Assoc Chart Physiother Respir Care 41, 3–8.

Tobin, M.J., 2001. Advances in mechanical ventilation. N Engl J Med 344, 1986–1996.

Tobin, M.J., Jubran, A., Laghi, F., 2001. Patient-ventilator interaction. Am J Respir Crit Care Med 163 (5), 1059–1063.

Torres, A., Aznar, R., Gatell, J.M., et al., 1990. Incidence, risk and prognosis factors of nosocomial pneumonia in mechanically ventilated patients. Am Rev Resp Dis 142, 523–528.

Unoki, T., Kawasaki, Y., Mizutani, T., et al., 2005. Effects of external rib-cage compression on oxygenation, ventilation, and airway secretion removal in patients receiving mechanical ventilation. Resp Care 50 (11), 1430–1437.

van der Schaaf, M., Beelan, A., Dongelmans, D.A., et al., 2009a. Functional status after Intensive Care: A challenge for rehabilitation professionals to improve outcome. J Rehabil Med 41, 360–366.

van der Schaaf, M., Beelan, A., Donglemans, D.A., et al., 2009b. Poor functional recovery after a critical illness: A longitudinal study. J Rehabil Med 41, 1041–1048.

Vesalius, A., 1543. De Humani Corporis Fabrica Libri Septem.

Woolam, C.H.M., 1976. The development of apparatus for intermittent negative pressure respiration. Anaesthesia 31 (4), 537–547.

Zanotti, E., Felicetti, G., Maini, M., et al., 2003. Peripheral muscle strength training in bed-bound patients with COPD receiving mechanical ventilation. Chest 124, 292–296.

Chapter 8

Cardiac rehabilitation

Sushma Sanghvi

CARDIAC REHABILITATION

Physiotherapists are valuable members of the multidisciplinary cardiac rehabilitation team. This chapter provides important key information about the research evidence, exercise prescription and planning across the four phases of cardiac rehabilitation. For in depth information about planning and delivering exercise rehabilitation for special conditions such as patients with heart failure, implanted cardioverter-defibrillators and cardiac transplantation, the reader is advised to refer to the guidelines and texts listed at the end of the chapter.

BACKGROUND

According to the World Health Organization (WHO), an estimated 17 million people die of cardiovascular disease (CVD) every year of whom 7.2 million die of coronary heart disease (CHD) and 5.7 million die of stroke. CVD is responsible for 10% of disability adjusted life years (DALYs) lost in low- and middle-income countries and 18% in high-income countries.

According to 2008 statistical data from the British Heart Foundation (BHF), CHD causes around 88,000 deaths in the UK every year. It is also the most common cause of premature death (death before the age of 75) in the UK. Eighteen per cent of premature deaths in men and 9% of premature deaths in women are from CHD. Nearly all deaths from CHD are a result of myocardial infarction (MI, heart attack). Around 124,000 people in the UK suffer a MI every year. There are 28,000 new cases of angina and 27,000 new cases of heart failure every year in the UK.

Over the last two decades of the twentieth century in the UK, there was a decline in the death rates from CHD. In a study by Unal et al. (2004), the authors concluded that 58% of CHD mortality decline in the 1980s and 1990s was owing to a reduction in major risk factors, primarily *smoking*. The remaining 42% of the decline in mortality was explained by treatments, including secondary prevention.

There remains considerable variation in the death rates across the UK. Deaths from CHD are highest in Scotland and the north of England. While deaths from CHD have declined overall, the difference between the most deprived groups and the least deprived groups remains high (5 : 1). South Asians living in the UK (Indians, Bangladeshis, Pakistanis and Sri Lankans) have a higher premature death rate from CHD than average. The death rate is 46% higher in South Asian men and 51% in South Asian women.

The overall burden of CVD is now far greater as a result of more people surviving cardiac illnesses and living much longer than before.

While genetic factors play a part, 80–90% of people dying of CHD have one or more major risk factors influenced by lifestyle. Physical activity, obesity, smoking and diabetes are major risk factors for CHD.

Many people find making significant lifestyle changes difficult, for example, people may be addicted to nicotine. If someone recently admitted to hospital with CHD needs to increase the amount of physical activity they undertake regularly, not only do they need to be well motivated but they and their families need to be confident that the exercise is safe.

Like all major illnesses, CHD has major physical, psychological and behavioural impacts on patients and their families. For some, the psychological consequences can be persistent and disabling. They can also be a barrier to making the lifestyle changes necessary to reduce the subsequent cardiac risk. For example, people with CHD can be afraid to take exercise or participate fully in their daily activities for fear of damaging their heart. After admission to the hospital, maybe following a MI or for coronary

revascularisation, the advice and treatment provision in primary care may not always be sufficient. Many people require more intensive help to understand their illness and treatment to attain the lifestyle changes and to regain their confidence so that they can enjoy the best possible physical, mental and emotional health, and return to as normal a life as possible.

WHAT IS CARDIAC REHABILITATION?

The WHO defines cardiac rehabilitation as:

> *The rehabilitation of cardiac patients is the sum of activities required to influence favourably the underlying cause of the disease, as well as the best possible physical, mental and social conditions, so that they may, by their own efforts, preserve or resume when lost, as normal a place as possible in the community. Rehabilitation cannot be regarded as an isolated form of therapy, but must be integrated with the whole treatment, of which it forms only one facet.*
>
> (WHO 1993)

Cardiac rehabilitation is a comprehensive intervention that offers education, exercise and psychosocial support for patients with CHD and their families and is delivered by many specialist health professionals. Cardiac rehabilitation can promote recovery, enable patients to achieve and maintain better health, and reduce the risk of death in people who have heart disease.

ABC Definition

Cardiac rehabilitation is the process by which patients with cardiac disease, in partnership with a multidisciplinary team of health professionals, are encouraged and supported to achieve and maintain optimal physical and psychosocial health.

BACPR (2002)

Research evidence for cardiac rehabilitation

When well provided and when people are offered comprehensive and tailored help with lifestyle modification involving exercise training, education and psychological input, cardiac rehabilitation can make a substantial difference in reducing mortality by as much as 20–25% over three years.

Table 8.1 Cochrane review 2004: meta-analyses of 8440 myocardial infarction, revascularisation and ischaemic heart disease patients

Exercise-only rehabilitation	27% reduction all cause mortality 31% reduction in cardiac mortality
Comprehensive rehabilitation	13% reduction all cause mortality 26% reduction cardiac mortality

The Cochrane Review 2004 (Jolliffe et al. 2004) (Table 8.1) established the importance of exercise-based cardiac rehabilitation. Cardiac mortality was reduced by 31% in the exercise-only cardiac rehabilitation and by 26% in comprehensive cardiac rehabilitation groups.

The research has been focussed around phase III of rehabilitation and in patients after MI and revascularisation.

Many studies still include only low risk, male, Caucasian, middle-aged MI patients and enroll only a small number of women, the elderly and ethnic minorities. Other cardiac patient groups, such as those following cardiac surgery, heart failure or heart transplantation, are excluded, thereby limiting the generalisability of the results.

Systematic reviews for chronic heart failure have demonstrated that exercise-based cardiac rehabilitation reduces mortality, increases the quality of life and that exercise is safe in this group of patients.

Evidence for physical activity and exercise

The WHO estimates that around 6% of all disease burden and around 30% of CHD burden to be caused by physical inactivity (WHO 2010). Physical activity levels are low in the UK. The Health Survey for England data (2008) show that only 39% of men and 29% of women meet the government guidelines of 30 minutes of moderate physical activity five or more times a week. The proportion of both men and women who met the recommendations decreased with age. Therefore, structured exercise as a therapeutic intervention is essential to the cardiac rehabilitation programme.

Evidence for education and psychosocial interventions

Psychological outcomes have been less well studied than the physical and functional effects of exercise training, and less well documented. It is likely that many of the psychological benefits are attributable to group activities, peer support and access to professional advice. There is difficulty in measuring outcomes for these interventions. Questionnaires have been used widely to measure quality of life and health status. Both generic (e.g. Short Form 36 (SF36)) or disease-specific (e.g. Quality

of life after MI (QLMI)) have been used. Short-term benefits have been observed in studies using disease-specific questionnaires.

Patient groups in cardiac rehabilitation

Typically, patients following an acute MI and coronary artery by-pass graft (CABG) surgery have been referred for cardiac rehabilitation. The National Service Framework recommends that cardiac rehabilitation should be available to people manifesting CHD in various forms. Many more groups are now included in both comprehensive and exercise-based rehabilitation.

Post-revascularisation

The number of patients receiving percutaneous transluminous coronary angioplasty (PTCA) and stenting are increasing. Education for lifestyle modification and exercise training is proven to be beneficial on physiological and psychosocial risk factors.

Stable angina

Cardiac rehabilitation improves the management of symptoms and exercise training assists in raising the angina threshold so patients are able to do more before they experience angina.

Chronic heart failure

With the advances in the management of CHD, the number of patients presenting with chronic heart failure are increasing. Exercise-based cardiac rehabilitation is beneficial in improving exercise capacity, reduction of symptoms and improving quality of life. Patients with mild-to-moderate heart failure show the largest improvements.

Special needs groups

An important drawback in most research is the lack of female and elderly patients, and patients' ethnic backgrounds are rarely reported. These under-represented groups need special attention.

Women

Incidence of CHD tends to be higher in men; however, this difference decreases with increasing age. According to the BHF statistics 2010 (BHF 2010), every year 44,000 women in the UK have a MI. Uptake of cardiac rehabilitation among women is low. When women attend cardiac rehabilitation programmes, the outcomes are as good as, or better than, for men. Their need may be greater as they suffer greater loss of function in relation to return to work, activity and sexuality, and experience high levels of anxiety and depression. More gender-specific information, individualised and flexible programmes, and suitable environment are required to address the specific needs of this group.

Older adults

Over half of all MIs occur in people over the age of 70 years and this is going to rise further as the number of older people in the total population increases. Disability rates are very high in these patient populations, particularly in women and in patients with angina or chronic heart failure. The presence of depression is also a determinant of poor physical functioning. Cardiac rehabilitation has been demonstrated to be safe and to improve aerobic capacity and muscle strength in older adults. Elderly people can derive similar benefits from a comprehensive menu-based cardiac rehabilitation programme. Issues of access, transport, timings and flexible programmes need to be addressed to meet the requirement of this patient population.

Ethnic groups

The incidence of CHD is much higher in some ethnic communities (e.g. South Asians). It has been suggested that people from ethnic minorities are less likely to be referred and join cardiac rehabilitation programmes. While planning strategies for rehabilitation for ethnic groups, their heterogeneity and cultural and linguistic needs must be acknowledged. When a behavioural change is required, it is crucial that the message is clearly understood. Knowledge of the cultural influences on physical activity and dietary practices would be beneficial to the patient. Similarly, awareness of health education material in appropriate languages can enhance the quality of service. It will help to involve health professionals from similar cultural backgrounds to develop and evaluate progress.

A variety of settings appropriate to the targeted communities can be used, for example community centres, temples, mosques, churches, health centres, etc. Involvement of the family and the younger generation is vital.

Other groups

For these groups, individualised assessment and risk stratification is essential.

Cardiac transplant

This group is relatively small. There is some evidence that exercise-based cardiac rehabilitation improves exercise tolerance in this group of patients.

Valve surgery

Supervised exercise training in comprehensive cardiac rehabilitation is beneficial in improving functional capacity, reducing symptoms and improving the quality of life in this patient group.

Congenital heart disease

This group includes young people and children. Supervised exercises improve exercise capacity and psychological function in this patient group.

Implanted cardioverter-defibrillators

The number of patients with implanted cardioverter-defibrillators in a cardiac rehabilitation programme may be small; however, comprehensive cardiac rehabilitation is safe and improves exercise ability and psychological well-being significantly.

Provision in the UK and cost-effectiveness

The overall level of provision of cardiac rehabilitation programmes in the UK has increased rapidly in the last 20 years. Current data from the National Audit of Cardiac Rehabilitation reveal the number of programmes at 395. The national service framework for CHD has advocated the use of disease registers in primary care to provide long-term follow up of patients with CHD and has set standards and milestones for secondary prevention.

There are huge variations in programme types, duration, frequency and intensity of exercise training. Many centres are delivering the service in primary care, and menu-based programmes are provided by a multi-disciplinary team.

A UK estimate suggests a cost of £6900 per Quality Adjusted Life Years (QALY) and a cost per life year gained of £15,700 three years after cardiac rehabilitation. This offers good value compared with many other treatments currently provided by the National Health Service (NHS).

COMPONENTS OF CARDIAC REHABILITATION

- Risk factor assessment and modification.
- Education.
- Exercise.
- Psychosocial support.

Operation and delivery

It transpires that cardiac rehabilitation is really a continuum of care from the time the patient is admitted until discharge and extends to outpatient care, as well as long-term follow-up in the community. Patient care is shared with the cardiology team, of which cardiac rehabilitation forms an essential part. Cardiology management includes patient assessment and risk stratification (predicting the likelihood of recurrence of cardiac events and disease prognosis). The patient also undergoes diagnostic tests and drug therapy, and may need revascularisation, such as angioplasty or a bypass grafting as appropriate.

Traditionally, cardiac rehabilitation is divided into four phases, progressing from the acute hospital admission stage to long-term maintenance of lifestyle change.

Phase I: inpatient period.
Phase II: early post-discharge period.
Phase III: supervised outpatient programme, including structured exercise.
Phase IV: long-term follow-up/maintenance in primary care.

Phase I: Inpatient period

Cardiac rehabilitation is offered as soon as it is practical as an integral part of care to someone who is admitted (or who is planned to be admitted) to hospital with CHD. It includes:

- assessment of physical, psychological and social needs;
- negotiation of a written informal plan for meeting these identified needs;
- initial advice on lifestyle, e.g. smoking cessation, physical activity (including sexual activity), diet, alcohol consumption and employment;
- mobilisation;
- education about prescribed medication, its benefits and possible side effects;
- involvement of relevant informal carer;
- provision of locally written information about cardiac rehabilitation;
- discharge planning.

Phase II: Early post-discharge period

This service shows variation in different regions and can vary from as little as a telephone helpline to group sessions or individual appointments (Figure 8.1). The National Service Framework recommendation includes:

- comprehensive assessment of cardiac risk, including physical, psychological and social needs for cardiac rehabilitation, and a review of the initial plan to meet these needs;
- provision of lifestyle advice and psychological interventions according to the agreed plan by the multi-disciplinary team;
- maintaining involvement of relevant informal carer;
- home visits, if appropriate.

Figure 8.1 Assessment with cardiac nurse.

Figure 8.2 Physiotherapist measuring the height to assess body mass index.

Phase III: Supervised outpatient programme, including structured exercise

Traditionally, this phase is well set up in the form of an outpatient hospital-based programme, although more services are now shifted into primary care. It includes:

- risk stratification and identification of the high-, medium- and low-risk patient for exercise;
- individualised progressive exercise prescription and supervised exercise sessions which vary from 4–12 weeks in different regions;
- re-evaluation of risk factors for CHD and health promotion advice and education (Figure 8.2).
- psychosocial interventions, such as stress management, counselling and vocational guidance.

Phase IV: Long-term follow-up/maintenance in primary care

This phase is now well set up in many districts, with emphasis on provision of exercise sessions available in community or leisure centres. Specialist training to exercise CHD patients in the community is available to the exercise instructors by the British Association for Cardiovascular Prevention and Rehabilitation (BACPR).

Phase IV includes:

- long-term maintenance of individual goals;
- professional monitoring of clinical status and follow-up of general progress;
- ongoing psychosocial support and support groups.

Cardiac rehabilitation team

The cardiac rehabilitation package individualised for each patient requires expertise and skills from a multidisciplinary collaborative team of professionals (Figure 8.4). The team includes a cardiologist and staff from nursing, physiotherapy, dietetics, pharmacy, occupational therapy and psychology with training in cardiac rehabilitation. Continuation of care in the community involves the primary healthcare team that is the general practitioner and cardiac nurse, phase IV exercise specialist and a link from a local cardiac patient support group (Figure 8.3).

The role of the physiotherapist

Physiotherapists have the knowledge, assessment skills and clinical reasoning, combined with evidence-based approach to treatment, to undertake the rehabilitation management of patients with multi-pathology problems. Physiotherapists are also trained to run group sessions and classes. Hence, the role of the physiotherapist within the

multi-disciplinary team should focus on exercise prescription, training and education in phases I–III. The modification in exercise prescription needs to be discussed with medical and nursing team members. In a group setting where exercise is delivered to CHD patients, teamwork and liaison with other team members who are aware of patients' clinical and psychosocial issues is essential.

Figure 8.3 Dietician addressing a group of patients attending phase III cardiac rehabilitation programme.

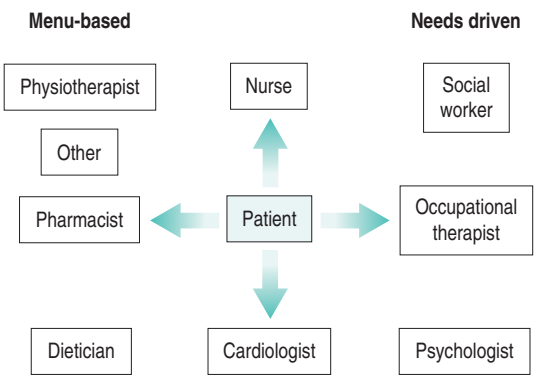


Figure 8.4 The cardiac rehabilitation team.

Professional development

The Association of Chartered Physiotherapists in Cardiac Rehabilitation (ACPICR) recommends that physiotherapists wishing to specialise in this area should refer to the ACPICR competences for the exercise component of phase III cardiac rehabilitation and the Skills for Health: Coronary Heart Disease document (ACPICR 2008). They should consider undertaking professional development in exercise physiology and exercise prescription in cardiovascular disease. Use of clinical and cardiac networks to share experiences, for example interactive CSP (www.csp.org.uk), is recommended.

BENEFITS OF EXERCISE TRAINING

ABC Definition

The term 'exercise training' applies to a programme of repeated exercises undertaken at a guided or prescribed intensity and frequency over a period of time, usually several weeks. It is based upon aerobic exercise designed to improve physical performance at both maximal and submaximal levels. It may be of low, moderate or high intensity, and may also include resistance training.

Research confirms that exercise training improves physical performance (exercise tolerance, muscular strength and symptoms), psychological functioning (anxiety, depression and well-being), and social adaptation and functioning in cardiac patients (Tables 8.2 and 8.4). It shows a reduction in mortality, morbidity, recurrent events and hospital readmissions. It is also found to have a positive impact on patients' physical ability to exercise (Table 8.3). Therefore, exercise training as a therapeutic intervention is central to the cardiac rehabilitation programme.

Table 8.2 Physiological benefits of exercise training in chronic heart disease patients (risk factors modification)

Reduction in systolic and diastolic blood pressures
Reduction in % body fat, increase in lean body mass
Reduction in fibrinogen levels and platelet aggregation
Increase in high-density lipoprotein, reduction in triglycerides
Increased insulin sensitivity, improved glucose-insulin dynamics

Table 8.3 Symptomatic benefits of exercise training in chronic heart disease patients

Reduced risk of arrhythmias
Increased ischaemic threshold
Increased angina threshold
Improved coronary perfusion
Reduction in ST wave changes
Reduced angina episodes/shortness of breath

Table 8.4 Psychosocial benefits of exercise in chronic heart disease patients

Reduction in anxiety and depression
Improved sleep patterns
Improved sense of well-being
Restoration of self-confidence
Reduction in illness behaviour
Improved social communication
Return to daily activities/hobbies
Resumption of sex life
Return to work/vocation
Compliance with other risk factors

PHYSIOLOGICAL ADAPTATIONS TO EXERCISE TRAINING IN HEALTHY INDIVIDUALS AND CORONARY HEART DISEASE PATIENTS

In healthy individuals physiological adaptations to aerobic exercise training are central (cardiac) and peripheral (skeletal muscle and vascular).

Adaptations at submaximal level of aerobic exercise

Adaptations at the submaximal level of aerobic exercise are reduction in heart rate (HR) owing to a decrease in sympathetic activity and increase in parasympathetic activity (vagal tone). The stroke volume increases owing to greater left ventricular filling and an increase in left ventricular mass. A decrease in the resting HR and blood pressure (BP) implies reduced myocardial oxygen demand. Also, the period of diastole is increased allowing greater time for blood to flow into the coronary circulation.

Cardiac output [CO = HR (heart rate) × SV (stroke volume)] must always match metabolic demand, but does so with reduced HR and increased stroke volume. Systolic BP (SBP) decreases and there is redistribution of blood flow to trained skeletal muscle and other tissues. Circulating catecholamines decrease and the arterio-venous oxygen difference increases.

Central changes as a result of aerobic exercise training

- Increased stroke volume.
- Increased left ventricular mass.
- Increased chamber size.
- Increased total blood volume.
- Decreased total peripheral resistance during maximal exercise.

Peripheral changes as a result of aerobic exercise training

- Increased arterio-venous oxygen difference.
- Increased number and size of mitochondria.
- Increased oxidative enzyme activity.
- Improved capillarisation.
- Increased myoglobin.

Key point

As a result of aerobic training there are functional and structural changes in skeletal muscle, the heart and the circulation. These changes improve the circulatory system's capacity to transport oxygen to the working muscles (central changes) and the capacity of skeletal muscle to extract and use oxygen (peripheral changes).

Increase in $VO_{2\ max}$

Oxygen consumption (VO_2) is expressed either in absolute terms as litres per minute ($L.min^{-1}$) or relative to body weight as mL per kg per minute ($mL.kg^{-1}.min^{-1}$). $VO_{2\ max}$ is the highest rate of oxygen consumption attainable during maximal exercise. Aerobic training increases $VO_{2\ max}$.

In relative terms, an individual walking at 4 miles per hour uses 17.5 mL of oxygen per kg of bodyweight per minute. In absolute terms, a man weighing 70 kg will use 1225 mL or 1.2 L per minute.

Key point

The significance of increased $VO_{2\ max}$ is a reduction in physiological stress evoked by submaximal activity and *not* the ability to perform maximal bouts of exercise.

In CHD patients, the increase in $VO_{2\ max}$ is predominantly a result of peripheral adaptations. Central changes are associated with long periods of high intensity training. Although central changes have been shown with high intensity training in CHD patients in some studies, in the

Key point

Aerobic exercise training at moderate intensity confers benefits to CHD patients by *peripheral adaptations*.

conventional cardiac rehabilitation programmes this regime is not suitable. Physical performance improvements are better seen in patients with low exercise tolerance.

ASSESSMENT FOR EXERCISE PRESCRIPTION

A thorough assessment is essential in order to plan an individualised and safe exercise prescription for cardiac patients and should include the following:

- a detailed history of the present condition and clinical presentation;
- previous levels of activity and exercise;
- physical limitations and disabilities;
- signs and symptoms;
- risk factor assessment/profile;
- screening for relative contraindications;
- risk stratification;
- functional capacity test;
- psychosocial assessment: objectives, beliefs, knowledge, interests, ethnicity;
- patient goals and expectations.

Contraindications to exercise

- Unstable angina.
- Resting SBP >200 mmHg or resting diastolic BP (DBP) >110 mmHg.
- Orthostatic BP drop >20 mmHg with symptoms.
- Critical aortic stenosis.
- Acute systemic illness or fever.
- Uncontrolled atrial or ventricular arrhythmias.
- Uncontrolled sinus tachycardia.
- Uncompensated chronic heart failure.
- Third degree heart block.
- Active pericarditis or myocarditis.
- Recent embolism.
- Thrombophlebitis.
- Resting ST segment displacement >2 mm on electrocardiograph (ECG).
- Uncontrolled diabetes (resting blood glucose >400 mg/dL).

EXERCISE PRESCRIPTION: THE FITT PRINCIPLE

To develop an individual exercise training programme, factors known as the FITT principles are considered.

- **F**requency
- **I**ntensity
- **T**ime (duration)
- **T**ype of exercise

Frequency, time and type or modes of exercise are explained later in the chapter with activities in different phases of cardiac rehabilitation.

Intensity of exercise

The risk of developing arrhythmias or adverse events such as an acute MI is increased in cardiac patients with vigorous activity. Low-to-moderate intensity exercise training can produce beneficial changes in functional capacity, cardiac function, coronary risk factors, psychosocial well-being and possibly improve survival in patients with CHD. For patients with low functional capacity, frequent and short duration exercise stimulus incorporated throughout the day may be advisable.

Intensity is prescribed and monitored by several methods which can be used independently or in combination with one another.

Heart rate

Each individual patient should have his/her training HR calculated based on thorough assessment and risk stratification. The training intensities for most patients range between 60% and 75% of the maximum HR for the majority of the population group. The more complex patient will require lower intensities (40–50%); hence, appropriate adjustments to these calculations will be required.

In ideal circumstances, when available, the training HR is obtained from a maximum or symptom limited exercise ECG test (exercise tolerance test (ETT)). The training HR should be set at 60–75% of maximal HR or 20 beats below the HR at which the symptoms appeared, and should be monitored throughout the exercise session. However, ETT information is not always available to the cardiac rehabilitation team and other methods for determining the training intensity are used frequently.

Using HR in isolation as a measure of exercise intensity has a number of limitations; hence, other methods of monitoring intensity should be used in addition. This includes the use of validated rating of perceived exertion scale (RPE) and direct clinical observation for signs of exertion.

Heart rate can remain one of the appropriate intensity markers, even when patients are influenced by

chronotrophic medication, such as beta-blockers. In this instance, the resting HR and the maximal HR are reduced by 20–40 beats per minute and the target HR can be recalculated on this basis.

Age-adjusted predicted maximum heart rate formula

This formula uses a predicted maximum heart rate based on age (220 – age) and, as such, can have an error margin of as much as ± 10 beats per minute.

A percentage of this predicted maximum is selected based on the assessment findings.

EG Example

- Patient A (low risk and uncomplicated) is 70 years of age.
- Maximum age predicted HR = 220 – 70 = 150.
  60–75% of predicted maximum HR:
  0.60 × 150 = 90
  0.75 × 150 = 112.
- Thus, training heart rate = 90 – 112 beats per minute.

Karvonen formula (heart rate reserve)

This formula assumes access to a true observed maximum HR. This may be gained, for example, by an ECG tolerance test. This formula is advantageous in that it accounts for the individual's resting HR. A percentage of this is selected based on the assessment findings, noting that 50–70% of HR reserve (HRR) is equivalent to 60–75% of maximum HR.

EG Example

- Patient B (low risk and uncomplicated) has a resting HR of 65 beats per minute (bpm) and achieves a maximum HR of 160 bpm during an ECG exercise test. The intensity of training following assessment has been set at 50–70% of HRR.
- Calculation of HRR = 160 – 65 = 95.
- Selection of % of HRR:
  50% of HRR = 0.50 × 95 = 47.5;
  70% of HRR = 0.70 × 95 = 66.5.
- Add resting HR:
  47.5 + 65 = 112.5;
  66.5 + 65 = 131.5.
- Thus, training heart rate = 112–131 bpm.

HRR is calculated thus:

- HRR = maximum HR – resting HR;
- training intensity is selected and calculated, i.e. 50–70% HRR;
- resting HR is added to HRR percentage.

Rating of perceived exertion

Patients need to develop the ability to perceive their exertion while exercising. In other words, they should feel the physical sensation of how hard they are working so that they know the safe limits to which they can exert themselves. In the early stages of rehabilitation, the physiotherapist assists using the HR monitoring method and by setting the exercise circuit at a specific work rate and, most importantly, by observation of the patients' response to exercise. On the 6–20 Borg rating of perceived exertion (RPE) scale (Table 8.5), a rating of 12–13 (or 3–4 on the CR-10 scale; Table 8.6) corresponds to 60% $VO_{2\,max}$ or 60% of HRR. A rating of 15 (or 6–7 on the CR-10 scale) corresponds to 75% of $VO_{2\,max}$ or HRR.

In moderate submaximal exercise muscular sensations and breathlessness relate very closely to the exercise stimulus and so the RPE scale is advised in cardiac rehabilitation. The CR-10 scale was developed to focus more on

Table 8.5 The Borg RPE scale (Borg 1998)

6	
7	Very, very light
8	
9	Very light
10	
11	Fairly light
12	
13	Somewhat hard
14	
15	Hard
16	
17	Very hard
18	
19	Very, very hard
20	

Table 8.6 The Borg category ratio (CR-10) scale (Borg 1998)

0	Nothing at all	No '1'
0.3		
0.5	Very, very weak	Just noticeable
0.7		
1	Very weak	
1.5		
2	Weak	Light
2.5		
3	Moderate	
4		
5	Strong	Heavy
6		
7	Very strong	
8		
9		
10	Extremely strong	'Strongest 1'
11		
≤		
•	Absolute maximum	Highest possible

rating individual sensations of strain, exertion or pain. Thus, if pain, breathlessness or localised muscle fatigue is the dominant sensation the CR-10 scale should be used.

Clinical note

Heart rate monitoring with RPE and observation of the patient during the exercise is an effective way of monitoring exercise intensity.

Metabolic equivalent

Metabolic equivalent (MET) relates to the rate of the body's oxygen uptake for a given activity as a multiple of resting VO_2. On average, an individual utilises 3.5 mL of O_2 per kg of body weight per minute ($ml.kg^{-1}.min^{-1}$). Therefore, one MET equals a VO_2 of 3.5 $ml.kg^{-1}.min^{-1}$. MET value is assigned to an activity by measuring the VO_2 for that activity.

Table 8.7 Energy costs of leisure activities

Activity	METs (min.)	METs (max.)
Cycling		
5 mph	2	3
10 mph	5	6
13 mph	8	9
Dancing		
(ballroom)	4	5
(aerobic)	6	9
Skipping		
<80/min	8	10
120–140/min	11	11
Swimming		
(breast stroke)	8	9
(freestyle)	9	10
Tennis	4	9
Walking		
1 mph	1	3
3 mph	3	3.5
3.5 mph	3.5	4
4 mph	5	6

METs = metabolic equivalent; mph = miles per hour.

The MET values are reported on the ETT. This information is useful for the cardiac rehabilitation physiotherapist for prescribing intensity, as well as to identify the functional capacity of the patient. For example, a patient with a peak capacity of seven METs cannot be prescribed skipping (8–10 METs). An individual's exercises can be prescribed and regulated by choice of activities according to the MET values for them (see Table 8.5). (Please also refer to functional capacity described later in the chapter.)

If an individual walking at 3 miles per hour reports his/her exertion as 12–13 on the RPE scale (12–13 RPE corresponds to 60% of $VO_{2\ max}$), then planning activities of comparable MET value in his/her exercise prescription will provide him/her with the appropriate training stimulus.

The MET values are estimated and an individual may be working slightly above or below the estimated MET value for a particular task. The variability depends on the complexity of the task. Table 8.7 gives the minimal and maximal activity values. Walking is a complex activity

which requires balance and use of arm and trunk movements, and, hence, there is variation in the MET value.

Frequency

Exercise training 2–3 times weekly (e.g. two supervised classes and one home circuit) for phase III is recommended. Exercising 2–3 times per week for a minimum of 8 weeks produces physiological and psychosocial adaptations. However, it should be remembered that to gain the optimum benefits patients will need ongoing exposure to exercise. Phase III should be seen as the beginning of these changes and after completion patients should be referred to phase IV for continuation and progression. The ultimate aim is to promote life-long adherence to the individual's exercise behaviour.

Time

The conditioning phase (aerobic exercise training) in a phase III cardiac rehabilitation programme should last between 20 and 30 minutes. This should be in addition to the warm-up and cool-down.

Type

Training activity needs to be aerobic and can be delivered in many ways. Initially, endurance training is desirable for CHD patients. Within an individual prescription, incorporating a variety of exercise types will optimise peripheral adaptation, reduce the likelihood of overuse injuries and will enhance motivation and compliance.

RISK STRATIFICATION

Exercise-based cardiac rehabilitation is associated with a reduction in coronary mortality and morbidity. As a result of novel treatment approaches now available to CHD patients and because of the improvement in the management of cardiac patients, the type and number of patients referred for cardiac rehabilitation is increasing.

The risk of adverse cardiac events during exercise is small. In supervised exercise programmes, the risk of exercise-related cardiac events is also small. However, it is essential that the cardiac rehabilitation team responsible for delivering the exercise programme recognise the likelihood of exercise related incidents and ensure that all reasonable and necessary steps are taken to deliver safe and effective exercise prescription to the patients.

Increased myocardial demands of vigorous exercise can precipitate arrhythmias. Risk is increased with extensive cardiac damage, residual ischaemia and ventricular arrhythmias on exercise. All patients should be assessed and risk stratified prior to recruitment to the exercise component of cardiac rehabilitation.

Clinical note

The term risk stratification for the exercise professional in cardiac rehabilitation implies thorough evaluation of the patient to assess the degree of risk of further cardiac events associated with exercise. This allows the exercise professional to guide patient management for exercise prescription, monitoring and progression appropriately.

The information required for assessment should include the following:

- diagnosis and the site and size of infarct or surgery details, as appropriate;
- current cardiac status;
- results of investigations, e.g. ECG exercise test, ECG report, angiogram;
- current medication;
- recovery and activity levels since discharge, symptoms;
- past medical history, including musculoskeletal, respiratory and neurological problems;
- CHD risk factors;
- psychosocial status.

Classification

The patients are risk stratified into low-, medium- and high-risk groups depending on their current cardiac status. This includes the extent of myocardial damage, previous history of MI, complications and associated signs and symptoms. The main risk to patients attending the exercise component of cardiac rehabilitation is ventricular fibrillation. Extensive myocardial damage, residual ischaemia, significant ECG changes, ST segment depression or arrhythmias on exercise are the key factors when predicting the risk.

The risk classification given below is based on the guidelines from the American Association of CardioVascular and Pulmonary Rehabilitation (AACVPR 2006).

High risk

The patient is classified at high risk when *any one* of the risk factors are present:

- decreased left ventricular function (ejection fraction <40%);
- complex arrhythmias at rest or appearing or increasing during exercise testing and recovery;
- presence of angina or other significant symptoms, such as unusual shortness of breath or dizziness at low levels of exertion (<5 METS) or during recovery;
- high levels of silent ischaemia (ST segment depression ≥ 2 mm from baseline) during exercise testing or recovery;

- abnormal haemodynamics with exercise (especially decrease in SBP during exercise or recovery – severe post-exercise hypotension);
- MI or revascularisation procedure complicated by congestive heart failure, cardiogenic shock and complex arrhythmias;
- survivor of cardiac arrest or sudden death;
- clinically significant depression.

Moderate risk

The patient is classified as moderate risk when he/she can meet neither the high nor the low risk criteria.

- Moderately impaired left ventricular function (ejection fraction 40–49%).
- Presence of angina or other significant symptoms such as unusual shortness of breath or dizziness occurring only at high levels of exertion (≥7 METS).
- Mild-to-moderate level of silent ischaemia (ST segment depression ≤2 mm from baseline) during exercise testing or recovery.
- Functional capacity <5 METS.

Low risk

The patient is classified as low risk when *each* of the risk factors listed below are present:

- no left ventricular dysfunction (ejection fraction >50%);
- no resting or exercise-induced complex arrhythmias;
- absence of angina or other significant symptoms, such as unusual shortness of breath or dizziness during exercise testing and recovery;
- uncomplicated MI, CABG, PTCA;
- normal haemodynamics with exercise testing and recovery;
- functional capacity ≥7 METS;
- absence of clinical depression.

Functional capacity

Functional capacity is a strong and independent risk factor of all-cause and cardiovascular mortality, and the one that can be improved by training. A low functional capacity of less than six METs indicates a high mortality group and functional capacity of greater than ten METs indicates excellent survival, regardless of occlusive coronary disease or left ventricular function.

Functional exercise testing

The most widely recognised measure of cardiopulmonary fitness is the aerobic capacity or maximal oxygen consumption ($VO_{2\ max}$). This variable is defined as the highest rate of oxygen transport and use that can be achieved at maximal physical exertion. Thus, it is an expression of the functional health of the combined cardiovascular, pulmonary and skeletal muscle systems. It may be used to prescribe an appropriate training intensity in rehabilitation programmes and to identify improvements in endurance fitness. Determination of VO_2 peak during cardiopulmonary exercise testing provides an objective and reproducible assessment of functional capacity in patients with cardiac disease. In clinical practice, $VO_{2\ peak}$ is predicted or estimated from the treadmill speed and per cent grade, and expressed as METs. Thus, ETT can produce an estimated MET value to assess the patient's response to exercise, to guide risk stratification and exercise prescription. It would also serve as an objective outcome measure of the impact of exercise programme on functional capacity. An ETT is strongly recommended 3–6 weeks post-event.

ETT can give the following information:

- HRs and exercise level at peak exercise;
- symptoms and/or ECG changes;
- RPE;
- BP response to exercise;
- MET level achieved at training HRs (e.g. at 60–75% of HR max).

MET values can also be estimated from submaximal protocols recommended for assessing functional capacity. These are externally paced field exercise tests, such as the step test, shuttle walk test and cycle ergometry.

The Chester step test is a submaximal multi-stage test lasting ten minutes with a choice of four step heights.

The shuttle walking test (SWT) is a low cost, simple alternative to exercise testing that informs the rehabilitation team on a suitable exercise programme and appropriate training HR, and allows assessment of progress during cardiac rehabilitation. The limitation of SWT is that it is not suitable for people with higher baseline fitness level. Also, it may not be sensitive to change demonstrating improvements in functional capacity in the older cardiac population with coexisting pathologies by incremental walking. Thus a variety of outcome measures may be required for the patient population of cardiac rehabilitation.

The information obtained from the risk classification is used to determine baseline fitness level, exercise prescription, exercise progression, staff–patient ratio, and whether the site of the exercise programme is supervised or unsupervised and based in the hospital or community.

EXERCISE PROGRAMMING

Patients should participate in an induction prior to undertaking the Phase III exercise component of cardiac rehabilitation.

Patients should not take part if they present with:

- fever and acute systemic illness;
- unresolved unstable angina;
- resting systolic blood pressure >200, diastolic blood pressure >110;
- significant unexpected drop in blood pressure;
- tachycardia >100;
- new symptoms of shortness of breath, palpitations, dizziness or lethargy;
- recent embolism;
- thrombophlebitis;
- uncontrolled diabetes (should be assessed with local protocol and on a case-by-case basis);
- severe respiratory, orthopaedic or metabolic condition that would limit the exercise ability.

Warm-up

Strenuous exercise without previous warm-up can produce ischaemic ST segment changes and arrhythmias even in healthy adults.

> **Key point**
>
> For older adults and the cardiac patients, warm-up must be more gradual than for an apparently healthy population.

The warm-up is the preparatory phase of the exercise session. A well planned and effectively carried out warm-up will improve the exercise performance and optimises the safety and effectiveness of the exercise session. For cardiac patients, warm-ups should be of at least 15 minutes duration. This prepares the cardiovascular system for the exercise activity. It allows a gradual increase in myocardial blood supply by vasodilation of the coronary arteries and achieves a gradual rise in aortic pressure. This reduces the risk of provoking ischaemia and arrhythmias. It also prepares the mind by focussing the participants' attention on the activity ahead.

The warm-up consists of pulse-raising exercises, mobilising major joints and stretching – specific warm-up movements.

Pulse-raising exercises

These include rhythmic movements, initially of lower limbs (e.g. walking forwards and back, stepping, side-stepping, step backs, etc.) gradually increasing the HR and blood flow of the active muscles.

Mobilising major joints and stretching

The major joints are mobilised by taking each of them through a normal range of movement (e.g. the shoulders are raised and lowered, circled backwards and forwards, and the lumbar and thoracic spine mobilised by bending sideways and turning). This ensures that the joints are well lubricated and blood flow to the structures surrounding the joints increases, allowing full range of movement. Stretching the large muscles will assist mobilisation.

Muscles which are prone to adaptive shortening owing to cardiac surgery or as a result of the ageing process should be stretched for about ten seconds. While holding a stretch it is important to keep the rest of the body moving to maintain the pulse rise and to avoid pooling of blood in the lower extremities (venous pooling).

Specific movements

Specific exercises that mimic the movements of prescribed activity at low intensity levels will assist the preparation for conditioning phase by activating the neuromuscular pathways (e.g. alternate legs to side before side-stepping) (Figures 8.5 and Figure 8.6).

Figure 8.5 A group of phase III patients doing 'warm-up' stretches.

Figure 8.6 Warm-up involving specific movements.

Key point

It is recommended that the patients should achieve a HR within 20 bpm of the training HR or RPE of 10–11.

Cardiovascular conditioning

This component includes aerobic exercise training which produces the beneficial physiological effects for the healthy and cardiac population. Cardiovascular (CV) training depends on the patients' functional capacity and his/her activity levels as determined in the assessment. Functional capacity may be low for some sedentary patients.

The exercise programme should be designed to produce a training effect which is achieved through varying the frequency, duration, intensity and type/mode of exercise. The principal goal is to improve the duration and efficiency of exercise and then progress the intensity.

CV conditioning can be executed by continuous or interval training approach.

- *Continuous training* is an aerobic activity performed at a constant submaximal intensity which is prescribed and monitored. For example, brisk walking, cycling, stepping up and down on a step machine or bench, and walking/running on the treadmill (Figure 8.7).
- *Interval training* consists of bouts of aerobic exercise that are interspersed with periods of lower intensity work. In the cardiac patients – particularly the elderly or those with low functional capacity – a greater amount of work is achieved with the interval training approach than with continuous training. It is also less daunting and encourages compliance. Lower intensity exercises in the interval training are also referred to as 'active recovery'.

The CV conditioning period should be for 20–30 minutes. Circuit training is popular as it can be designed with or without equipment. 'Active recovery' stations increase the endurance of specific muscle groups, for example triceps, pectorals, trapezius.

Individualisation of the CV component is achieved by varying:

- the duration at CV station;
- the intensity;
- the period of rest between stations;
- the overall duration of conditioning.

Exercises performed in the recumbent position should be avoided during the CV conditioning phase because some older adults may experience difficulty in getting up and down. Immediately after vigorous activity, venous return will increase on lying down and will increase myocardial workload. There is also an increased risk of orthostatic hypotension. Floor work when indicated (e.g. relaxation exercise and stretching) should be carried out after a cool-down period when the cardiovascular system has recovered.

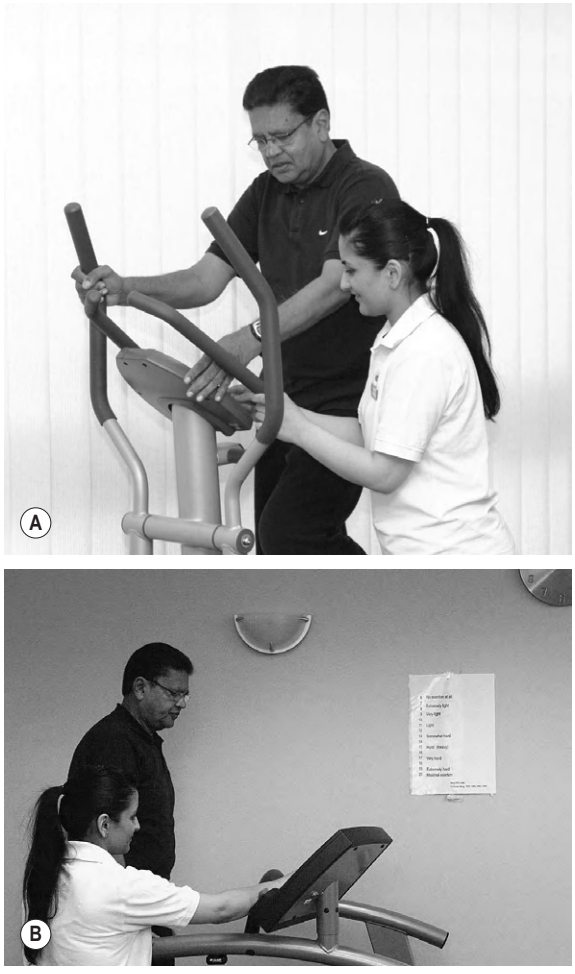


Figure 8.7 Physiotherapist assisting the patient in setting the intensity for exercise training on treadmill and cross trainer.

Key point

In supervised exercise programmes, interval circuit training is better suited to cardiac patients, at least in the initial period. The duration of activity is increased first before increasing the intensity.

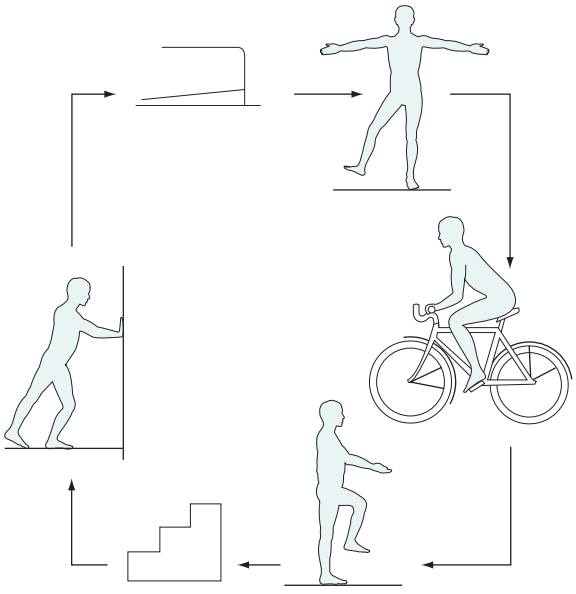


Figure 8.8 An example of a simple circuit set up with cardiovascular and active recovery stations for a beginner in phase III.

Table 8.8 An example of the circuit design

High intensity (CV) stations	Active recovery stations	CV alternative
1. Knee raises	2. Bicep curls	Shuttle walk/ jog
3. Treadmill (walking on incline)	4. Lateral arm raises	
5. Step-ups	6. Upright rows	
7. Alternate side-taps	8. Forward press with theraband	
9. Bike	10. Wall press	

CV = cardiovascular.

Class management

The control of the circuits needs to be carefully considered (Figure 8.8; Table 8.8). One member of staff for five patients is recommended.

- Beginner: 1 minute CV and 1 minute active recovery (AR).
- Intermediate: 1 minute CV, 30 seconds AR and 30 seconds CV alternative.
- Advanced: 1 minute CV and 1 minute CV alternative.

In this circuit, patients go round the circuit twice. The instructor calls at 30 seconds.

The beginner achieves 10 minutes of work, patients at intermediate level achieve 15 minutes of CV work and the advanced-level patients achieve 20 minutes of CV work (continuous training).

Cool-down

This consists of pulse-lowering exercises, large muscle group stretching and joint mobilisation at a slower pace, with movements of steadily reducing intensity. Its aim is to return the cardio-respiratory system to near pre-exercise levels within 10–15 minutes. It is essentially the reverse of warm-up. A minimum period of ten minutes is recommended for cool-down at the end of CV conditioning.

Following the sustained aerobic exercise training there is an increased risk of venous pooling. This may also be coupled with side effects of medication and can cause hypotension. Cooling-down reduces the risk of hypotension, elevated HRs and arrhythmias.

Post-exercise supervision of 15–30 minutes is recommended. In many programmes the education or relaxation session follows the exercise, giving the opportunity for the supervision of patients.

Progression

This is achieved by increasing the duration, frequency or intensity of training in order to maintain the training stimulus. Ideally, serial exercise testing is used to modify prescription. If this is not available then HRs and perceived exertion at reference workloads can be compared and the information used to increase any of the three variables or a combination of them. Progression over a long period is aimed towards a more continuous training approach. Exercise progression will be highly variable between individuals with CHD depending on the severity of disease, coexisting pathology, patient motivation and compliance.

Resistance training

Health-related physical fitness includes cardiovascular (aerobic) fitness, muscle strength, endurance and flexibility, and body composition (lean–fat ratio). Muscle strength is the ability of a muscle to produce a maximum force at a given velocity of movement. Muscle endurance is the ability of a muscle to perform repeated muscle contractions against a submaximal resistance. Resistance training increases lean muscle mass and maintains basal metabolic rate when combined with aerobic training, thus aiding in weight management. By improving muscle strength and balance it can reduce the risk of falling. Positive effects on bone density are well known with resistance training.

Many activities of daily living, for example carrying shopping bags and doing house-work, require upper body strength. Cardiac rehabilitation professionals often come across patients who are fearful of lifting or carrying out resistance-based activities. Resistance training is associated with an increase in arterial BP which increases myocardial workload. Owing to these concerns, traditionally, aerobic exercise training has been the main focus in the cardiac rehabilitation programmes. However, recent research recommends resistance training as a part of a supervised exercise programme in cardiac patients. Haemodynamic and cardiovascular responses to resistance training are similar in CHD patients and normal subjects. Because of the increased diastolic pressure, myocardial perfusion may be enhanced. It is now generally agreed that low- and medium-risk cardiac patients can commence resistance exercise after completion of an aerobic exercise programme for 4–6 weeks: two sets of 8–10 exercises involving major muscle groups performed a minimum of twice per week. Currently in the UK, resistance training is not included in cardiac rehabilitation programmes with high-risk patients.

Key point

Resistance training, principally planned to build up muscular endurance, is associated with maintenance of strength and can be performed safely by patients.

Contraindications to resistance training are:

- abnormal haemodynamic responses to exercise;
- ischaemic changes during graded exercise testing;
- poor left ventricular function;
- uncontrolled hypertension or arrhythmias;
- exercise capacity of less than six METs.

EXERCISE CONSIDERATIONS FOR SPECIAL POPULATIONS

Heart failure

Heart failure is distinguished by the inability of the heart to pump enough blood (and therefore deliver adequate oxygen) to the metabolising tissues. A patient with heart failure presents with symptoms of breathlessness and fatigue at rest and swelling of the ankles. The New York Heart Association (NYHA) has classified the stages of heart failure based on the severity of symptoms (Table 8.9). The prognosis for heart failure is poor, with 50% of patients dying within four years and 50% of those diagnosed with severe heart failure dying within a year.

Heart failure patients are classified as 'high risk' group according to the AACVPR stratification criteria. Exercise prescription and training for this group demands rigorous assessment and monitoring. An appropriate safe environment and system needs to be in place to deliver exercise training. Patients need to be stable – any change in the clinical status may mean exercise is contraindicated. Based on risk stratification, increased staff–patient ratio and close monitoring of the symptoms of breathlessness (Borg CR-10 scale may be desirable), HR, BP pre- and post-exercise should be implemented. Interval training of 1–6 minutes of work/activity (40–60% functional capacity, 11–13 RPE) followed by rest is recommended.

Contraindications to exercise include:

- uncompensated heart failure;
- uncontrolled oedema;
- uncontrolled arrhythmias;
- symptoms at rest or with minimal exertion (class IV);
- unstable angina;
- resting sinus tachycardia (>120 bpm);
- hypotension (SBP <90 mmhg);
- hypokalaemia (serum K <3.0 mEq/L).

Table 8.9 The New York Heart Association functional classification: The stages of heart failure

Class	Patient symptoms
Class I (mild)	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation or dyspnoea (shortness of breath)
Class II (mild)	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation or dyspnoea
Class III (moderate)	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation or dyspnoea.
Class IV (severe)	Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased

Key point

Exercise-based cardiac rehabilitation intervention is safe and effective in stable chronic heart failure patients. Improvement of functional capacity, decreased symptoms ('Improved' NYHA class) and quality of life is reported, and are primarily a result of peripheral adaptations.

Older adults

Many patients referred to cardiac rehabilitation are over 50 years of age. The changes associated with ageing need to be accounted for when prescribing exercise. There is 1% loss of $VO_{2\ max}$ per year from the age of 25 years. Thus, functional capacity can be reduced depending on activity levels. Maximal HR declines with age. Lung elasticity and chest wall expansion decrease with age. Bone density is reduced by about 20% by the age of 65 years in women and by 10–15% by the age of 70 years in men. Muscle function declines by approximately 25% by the age of 65 years, as does joint flexibility and range of movement. Lean body mass reduces and body fat increases.

Motor skills, balance, reaction times and motor coordination decline with age. One third of people over the age of 65 years fall at least once a year. In addition, there may be hearing problems. All these can contribute to anxiety and diminished confidence to exercise. The exercise class atmosphere should be social, welcoming, relaxed and non-threatening. Patients need to feel comfortable.

Exercise prescription

Extended warm-up, and slow and controlled transition between movements should be encouraged. Precautions about extremes of weather should be taken.

- *Frequency*: 2–3 sessions per week.
- *Intensity*: lower end of prescription range (60–75% HR max.), RPE Borg 11–14, more gradual progression.
- *Type*: endurance training of longer duration, moderate intensity, resistance training to be introduced later with 40–60% of 1 repetition maximum (RM), maximum 8–10 repetitions, 1–3 sets, minimum of twice a week.
- *Time*: The conditioning period should be 20–30 minutes.

Hypertension

British Hypertension Society Guidelines (2004) advise antihypertensive therapy at different thresholds as follows:

- individuals not at high risk of CHD/atherosclerotic disease are classified to be hypertensive and treated at BP of: 160/100 mmHg;
- individuals with CHD/other atherosclerotic disease are classified to be hypertensive and treated at BP of: 140–149/90–99 mmHg;
- individuals with diabetes are classified to be hypertensive and treated at BP of: ≥140/ ≥90 mmHg.

Exercise prescription

- *Frequency*: 3–5 times per week.
- *Intensity*: reduced to 50–75% maximum HR.
- *Type*: lower resistance/higher repetitions; avoid over-gripping equipment; avoid valsalva manoeuvre and isometric work.
- *Time*: increase duration at moderate intensity.

Exercise is contraindicated when SBP is >180 mmHg or DBP is >100 mmHg. Antihypertensive medication may lead to hypotension. Post-exercise extended active recovery with constant feet movement is required to ensure venous return.

Diabetes

Diabetes is a group of diseases marked by high levels of blood glucose resulting from defects in insulin production, insulin action or both.

Diabetes must be stabilised following events such as MI and bypass graft surgery. Diabetic patients may not experience pain and can have silent ischaemia. Close supervision during the session is required.

FITT principles apply for exercise prescription. Other considerations:

- monitor blood sugar before and after exercise;
- insulin may need to be reduced on exercise days;
- exercise should be avoided when insulin is at its peak effect; insulin uptake is increased if injection is into an exercising limb;
- when new to exercise it is advisable to have other people around;
- autonomic neuropathy may alter HR and BP response;
- retinopathy;
- silent ischaemia – monitor for overexertion.

Peripheral vascular disease

Peripheral vascular disease (PVD) is also known as atherosclerosis, poor circulation or hardening of the arteries. It presents with intermittent claudication and ischaemic pain on exertion that diminishes with rest. It most commonly affects the legs ('angina' of the legs). In severe cases, symptoms include cold, painful feet.

Patients need to be reassured as they suffer from a lack of confidence to exercise beyond the point of pain.

Exercise prescription

- *Frequency*: increased frequency; short bouts of exercise often better tolerated than continuous.
- *Intensity*: increased duration before intensity; use peripheral vascular disease (PVD) scales of discomfort.
- *Type*: walking/weight-bearing.
- *Time*: daily exercise/graduated increase in duration.

Non-weight-bearing activities, for example cycling, can be used to achieve prescribed cardiovascular dose and improved compliance.

Obesity

Traditionally, a person has been considered to be obese if they are more than 20% over their ideal weight. That ideal weight must take into account the person's height, age, sex and build. Obesity has been more precisely defined by the National Institutes of Health as a body mass index (BMI) of 30 and above.

BMI is a measure of body fat based on height and weight that applies to both adult men and women. It is weight (kgs) divided by height (m^2). As the BMI describes the body weight relative to height, it correlates strongly (in adults) with the total body fat content.

BMI categories:

- underweight: <18.5;
- normal weight: 18.5–24.9;
- overweight: 25–29.9;
- obesity: BMI of 30 or greater.

Obesity is also measured by measuring thickness of skin folds. Central obesity is measured by waist–hip ratio (>0.95 in males, >0.85 in females).

Exercise prescription

- *Frequency*: 3–5 times per week.
- *Intensity*: reduce to 50–75% HR maximum.
- *Type*: combine cardiovascular work and muscular strength and endurance (MSE) work to reduce fat weight and increase lean tissue; avoid high impact work and stress on joints; provide alternatives and, if necessary, avoid supine positions, which can restrict breathing.
- *Time*: increase duration and frequency as able.

Osteoarthritis and rheumatoid arthritis

Osteoarthritis (OA) is a degenerative arthritis caused by wear and tear. It affects discrete joints. Rheumatoid arthritis (RA) is a systemic illness characterised by inflammation and can affect multiple joints.

Both conditions are marked by inflammation, pain and restricted movement. In RA there may be periods of exacerbation and remission. In advanced arthritis there may be joint deformity.

Exercise prescription

- *Frequency*: 3–5 times per week (but rest during RA exacerbations).
- *Intensity*: 60–75/80% HR maximum.
- *Type*: mobility/strength work for range of movement and joint stability; low impact work to avoid stress on joints; non-weight-bearing if limited by pain to achieve cardiovascular prescription with minimal discomfort; postural alignment.
- *Time*: 20–30 minutes.

Respiratory conditions

This may include asthma, chronic bronchitis and emphysema. The presentation is different in each condition, although the common symptoms are breathlessness and increased work of breathing, with or without excessive sputum production.

Exercise prescription

- *Frequency*: 3–5 times per week.
- *Intensity*: based on RPE; assessment on SWT.
- *Type*: endurance work; activities of daily living-based; lower limb activity.
- *Time*: interval training of short duration; increased duration/frequency as able.

EXERCISE PRESCRIPTION AND DELIVERY ACROSS FOUR PHASES OF CARDIAC REHABILITATION

Phase I: Inpatient period

Activities for cardiac patients following acute MI or CABG do not typically exceed 2–3 METS in the early stages. These include general mobility exercises and activities of daily living, such as standing, walking, dressing and personal hygiene. Before discharge, the patient is advised on the progression of physical activity levels. Guidance on convalescence activities over the first 3–4 weeks, written advice on 'do's and don't' activities should be provided and, usually, an incremental walking programme is also given (Table 8.10).

Phase II: Immediate post-discharge period

This period is usually between two and six weeks, and the follow-up varies depending on local protocols. Often this period is frightening for the patient and their family who may feel isolated after the close supervision and support in the hospital.

Although progression is individualised for each patient, the following guide serves as a basis for prescription. The patient should be advised on the signs and symptoms of overexertion, chest pain management, timing of exercise (40–50 minutes after a meal) and to avoid temperature extremes.

Phase III: Supervised outpatient programme, including structured exercise

Structured exercise training during this phase is delivered in either the hospital or the community. Access to

Table 8.10 Incremental walking programme

Week	Borg CR-10 scale	Duration (min.)	Distance (yards)	Frequency (per day)
1	2–3	5	200	1–2
2	2–3	10	400–500	2
3	2–3	15	500–750	2
4	3	20	750–1250	1–2
5	3	25–30	1250–1750	1–2
6	3	30–40	1750–3000	1–2

Table 8.11 Structuring exercise sessions

Staff–patient ratio
Room size
Temperature 65–72°F
Humidity close to 65%
Staff training
Emergency drills

Table 8.12 Outcome measures assessment guideline

Functional capacity	Graded exercise test (SWT, CST)
Return to vocation	Work modification
Smoking cessation	Self-reported
Managed BP	Regular average BP recordings
Stress management	HAD scale, self-reported rating
Quality of Life	Measure of multiple domains of quality of life
Lipids	Lipid profile
Weight control	Height, weight, BMI, waist–hip ratio

BMI = body mass index; BP = blood pressure; CST = Chester step test; HAD = hospital anxiety and depression scale; SWT = shuttle walk test.
(Adapted from American Association of Cardiovascular and Pulmonary Rehabilitation 1995)

emergency facilities should be available. It lasts for 6–12 weeks in most centres. The sessions should be delivered by professional staff with training in cardiology, exercise prescription and emergency procedures. Consideration should be given to the staff–patient ratio (one member of staff per five patients is recommended in the UK guidelines).

The cardiac rehabilitation team delivering exercise sessions should all be trained in basic life support; preferably, one member of the team delivering the exercise session should be advanced life support trained. There should be an access available for automated defibrillator (AED) and the team trained to use it. All staff should have regular practice with emergency drills. Local protocols for health and safety should be followed at all times. The criteria for structuring exercise sessions is highlighted in Table 8.11. An outcome measures assessment guideline is shown in Table 8.12.

Phase IV: Long-term follow-up/ maintenance in primary care

Patients are transferred to phase IV when medically stable and psychologically adjusted.

They should have reached their exercise goals. Patients should demonstrate the ability to exercise safely based on an individual exercise prescription and recognise warning signs and symptoms to take appropriate action (i.e. stop or reduce exercise level, take glyceryl trinitrate).

Exclusion criteria for phase IV:

- unstable angina;
- testing SBP >180 mmHg, DBP >100 mmHg;
- significant drop in BP;
- uncontrolled tachycardia;
- unstable or acute heart failure;
- febrile illness.

Discharge planning

Following completion of phase III cardiac rehabilitation, all patients' individual long-term exercise plans are agreed and arrangements are made for transfer to phase IV. The patient is referred to the primary care service for monitoring of the risk factors. A patient may require further assessment, for example a patient experiencing residual ischaemia. In such cases, appropriate cardiology referral

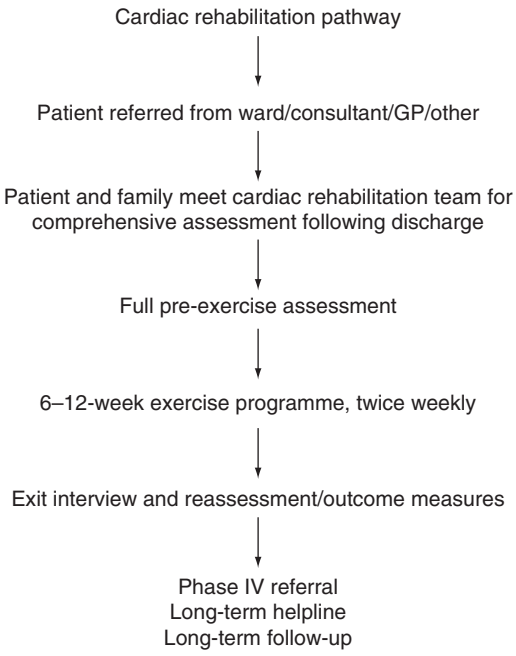


Figure 8.9 The cardiac rehabilitation pathway.

and implications on exercise prescription are noted and explained to the patient. All patients should be given information about long-term helpline and local cardiac support groups (Figure 8.9).

CONCLUSION

Comprehensive cardiac rehabilitation is a cost-effective intervention for patients with cardiac disease. Exercise-based cardiac rehabilitation confers several benefits, including improvement in functional capacity and secondary prevention. Exercise consultation is a vital intervention and should be available and incorporated throughout all phases of cardiac rehabilitation. Exercise intervention is a behavioural change and the rehabilitation team should utilise counselling skills and deploy strategies in order to promote long-term adherence to exercise and physical activity.

Physiotherapists have the knowledge, assessment skills and clinical reasoning, combined with an evidence-based approach, to deliver rehabilitation to patients with multi-pathology problems. Thus, physiotherapists have a key role in the physical activity component of all phases of cardiac rehabilitation.

Weblinks

www.cardiacrehabilitation.org.uk
www.bcs.com
www.acpicr.com
www.aacvpr.org
www.ic.nhs.uk
www.heartstats.org
www.bhf.org.uk
www.csp.org.uk
www.dh.gov.uk
www.sign.ac.uk
www.bhfactive.org.uk
www.sahf.org.uk
www.ash.org.uk
www.bhsoc.org
www.diabetes.org.uk

FURTHER READING

ACPICR (Association of Chartered Physiotherapists in Cardiac Rehabilitation), 2009. Standards for Physical Activity and Exercise in Cardiac Population. ACPICR, http://www.acpicr.com/publications

American College of Sports Medicine, 2006. ACSM's Guidelines for Exercise Testing and Prescription, seventh ed. Lippincott, Williams and Wilkins, Baltimore.

Austin, J., Williams, R., Ross, L., et al., 2005. Randomized control trial of cardiac rehabilitation in elderly patients with heart failure. Eur J Heart Fail 7 (3), 411–417.

BACPR (British Association for Cardiovascular Prevention and Rehabilitation), 2012. Standards and Core Components for Cardiovascular Prevention and Rehabilitation 2012, second ed.; http://www.bacpr.com/resources/46C_BACPR_Standards_and_Core_Components_2012.pdf, accessed October 2012.

Bethell, H.J., Turner, S.C., Evans, J.A., et al., 2001. Cardiac rehabilitation in the United Kingdom. How complete is the provision? J Cardiopulm Rehabil 21 (2), 111–115.

BHF (British Heart Foundation), 2010. Coronary Heart Disease Statistics, 2010; http://www.bhf.org.uk/idoc.ashx?docid=9ef69170-3edf-4fbb-a202-a93955c1283d&version=-1, accessed October 2012.

Bjarnason-Wehrens, B., Mayer-Berger, W., Meister, E.R., et al., 2004. Recommendations for resistance exercise in cardiac rehabilitation. Recommendations of the German Federation for Cardiovascular Prevention and Rehabilitation. Eur J Cardiovasc Prev Rehabil 11 (4), 352–361.

Dalal, H.M., Evans, P.H., 2003. Achieving national service framework standards for cardiac rehabilitation and secondary prevention. BMJ 326, 481–484.

De Bono, D.P., 1998. Models of cardiac rehabilitation: Multidisciplinary rehabilitation is worthwhile, but how is it best delivered? BMJ 316 (7141), 1329–1330.

DH (Department of Health), 2000. National Service Framework for Coronary Heart Disease. DH, London.

DuBach, P., Myers, J., Dziekan, G., et al., 1997. Effect of exercise training on myocardial remodelling in patients with reduced left ventricular function after myocardial infarction. Circulation 95, 2060–2067.

European Heart Failure Training Group, 1998. Experience from controlled trials of physical training in chronic heart failure. Protocol and patient factors in effectiveness in the improvement in exercise tolerance. Eur Heart J 19, 466–475.

ExTraMATCH Collaborative, 2004. Exercise training meta-analysis of trials in patients with chronic heart failure. BMJ 328, 189–192.

Fitchet, A., Doherty, P.J., Bundy, C., et al., 2003. Comprehensive cardiac rehabilitation programme for implantable cardioverter-defibrillator patients: a randomised controlled trial. Heart 89 (2), 155–160.

Franklin, B.A., Gordon, S., Timmins, G.C., 1992. Amount of exercise necessary for the patient with coronary artery disease. Am J Cardiol 69, 1426–1432.

Goble, A.J., Worcester M.U.C., 1999. Best practice guidelines for cardiac rehabilitation and secondary prevention: A synopsis. Heart Research Centre Melbourne, on behalf of Department of Human Services Victoria, Melbourne.

Haskell, W.L., 1994. The efficacy and safety of exercise programs in cardiac rehabilitation. Med Sci Sports Exerc 26, 815–823.

Hunt, S.A., Abraham, W.T., Chin, M.H., et al., 2005. ACC/AHA guideline update for the diagnosis and management of chronic heart failure in the adult: a report of the American College of Cardiology/American Heart Association Task Force on Practical Guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure). J Am Coll Cardiol 46 (9), e1–82.

Jolliffe, J.A., Rees, K., Taylor, R.S., et al., 2004. Exercise-based rehabilitation for coronary heart disease. Cochrane Database Syst Rev 1, http://www.cochrane.org.

Kobashigawa, J.A., Leaf, D.A., Lee, N., et al., 1999. A controlled trial of exercise rehabilitation after heart transplantation. N Engl J Med 340 (4), 272–277.

McArdle, W.D., Katch, F.I., Katch, V.L., 2001. Exercise Physiology: Energy, Nutrition and Human performance, fifth ed. Lippincott Williams & Wilkins, Baltimore.

National Service Framework, 2000. Coronary Heart Disease. Modern standards and service models, http://www.doh.gov.uk/nsf/coronary.htm.

NHS Centre for Reviews and Dissemination, 1998. Effective Health Care Bulletin: Cardiac Rehabilitation. University of York, York.

Nieuwland, W., Berkhuysen, M.A., Van Veldhuisen, D.J., et al., 2000. Differential effects of high-frequency versus low-frequency exercise training in rehabilitation of patients with coronary artery disease. J Am Coll Cardiol 36, 202–207.

Oldridge, N.B., 1998. Comprehensive cardiac rehabilitation: is it cost-effective? Eur Heart J 19 (Suppl.O), 42–50.

Pollock, M.L., Gaesser, G.A., Butcher, J.D., et al., 1998. American College of Sports Medicine Position Stand. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. Med Sci Sport Exerc 30 (6), 975–991.

Rees, K., Taylor, R.S., Singh, S., et al., 2004. Exercise based rehabilitation for heart failure. Cochrane Database Syst Rev (3):CD003331.

SIGN (Scottish Intercollegiate Guidelines Network), 2002. Cardiac Rehabilitation, no. 57. SIGN, Edinburgh.

Singh, S.J., Morgan, M.C.D.L., Scott, S., et al., 1992. Development of a shuttle walking test of disability in patients with chronic airways obstruction. Thorax 47, 1019–1024.

Smart, N., Marwick, T.H., 2004. Exercise training for patients with heart failure: a systematic review of factors that improve mortality and morbidity. Am J Med 116 (10), 693–706.

Stewart, K.J., Badenhop, D., Brubaker, P.H., et al., 2003. Cardiac rehabilitation following percutaneous revascularization, heart transplant, heart valve surgery, and for chronic heart failure. Chest 123, 2104–2111.

The Criteria Committee of the New York Heart Association, 1994. Nomenclature and Criteria for Diagnosis of the Diseases of the Heart and Great Vessels, ninth ed. Little Brown & Co., London, pp. 253–256.

The European Society of Cardiology, 2001. Working Group Report: Recommendations for exercise testing in chronic heart failure patients. Eur Heart J 22(1), 37–45.

Thow, M., 2006. Exercise Leadership in Cardiac Rehabilitation. An Evidence-Based Approach. John Wiley & Sons, Glasgow.

Weiner, D.A., Ryan, T.J., McCabe, C.H., 1987. Value of exercise testing in determining the risk classification and the response to coronary artery bypass grafting in three-vessel coronary artery disease: a report from the Coronary Artery Surgery Study (CASS) registry. Am J Cardio l60, 262–266.

WHO (World Health Organization), 2007. The Atlas of Heart Disease and Stroke, http://www.who.int/cardiovascular_diseases/resources/atlas/en/

Williams, M.A., 1994. Exercise Testing and Training in the Elderly Cardiac Patient. Current Issues in Cardiac Rehabilitation Series. Human Kinetics, Champaign, IL.

Williams, B., Poulter, N.R., Brown, M.J., et al., 2004. British Hypertension Society guidelines for hypertension management (BHS-IV): summary. BMJ 328 (7440), 634.

Wood, D., Durrington, P.N., Poulter, N., et al., 1998. Joint British Guidelines on prevention of coronary heart disease in clinical practice. Heart 80 (Suppl. 2), 1–29.

REFERENCES

AACVPR (American Association of Cardiovascular and Pulmonary Rehabilitation), 2006. Guidelines for Cardiac Rehabilitation and Secondary Prevention Programs, fifth ed. Human Kinetics, Champaign, IL.

ACPICR (Association of Chartered Physiotherapists in Cardiac Rehabilitation), 2008. Competences for the Exercise Component of Cardiac Rehabilitation, http://www.acpicr.com/publications.

BACPR (British Association for Cardiovascular Prevention and Rehabilitation), 2002. Standards for Cardiac Rehabilitation 2002; http://www.bacpr.com/pages/default.asp.

BHF (British Heart Foundation), 2010. Coronary heart disease statistics 2010; http://www.bhf.org.uk/idoc.ashx?docid=9ef69170-3edf-4fbb-a202-a93955c1283d&version=-1, accessed October 2012.

BHS (British Hypertension Society), 2004. British Hypertension Society Guidelines IV, 2004, http://www.bhsoc.org/resources/latest-guidelines/, accessed October 2012.

Borg, G.A.V., 1998. Borg's Perceived Exertion and Pain Scales. Human Kinetics, Champaign, IL.

Jolliffe, J.A., Rees, K., Taylor, R.S., et al., 2004. Exercise-based rehabilitation for coronary heart disease. Cochrane Database Syst Rev 1,http://www.cochrane.org.

NHS Information Centre, 2008. Health Survey for England 2008: Physical activity and fitness. NHS Information Center for health and social care; http://www.ic.nhs.uk/pubs/hse08physicalactivity, accessed October 2012.

Unal, B., Crtichley, J.A., Capewell S., 2004. Explaining the decline in coronary heart disease mortality in England and Wales between 1980 and 2000. Circulation 109, 1101–1107.

WHO (World Health Organization), 1993. Needs and Action Priorities in Cardiac Rehabilitation and Secondary Prevention in Patients with Coronary Heart Disease. WHO, Geneva.

WHO (World Health Organization), 2010. Global recommendations on physical activity for health; http://www.who.int/dietphysicalactivity/publications/9789241599979/en/index.html, accessed October 2012.

Chapter 9

Physiotherapy in thoracic surgery

Anne Dyson and Kelly L. Youd

ANATOMY OF THE THORAX

The skeleton of the thorax is an osteocartilagenous framework within which lie the principal organs of respiration, the heart, major blood vessels, and the oesophagus. It is conical in shape, narrow apically, broad at its base and longer posteriorly. The bony structure consists of 12 thoracic vertebrae, 12 pairs of ribs and the sternum (Figure 9.1).

The musculature of the thoracic cage is in two layers. The outer layer consists of latissimus dorsi and trapezius, the inner layer of the rhomboids and serratus anterior muscles. Anteriorly, the chest wall is covered by pectoralis major and minor. The intercostal muscles run obliquely between the ribs. The diaphragm forms the lower border of the thorax. It is convex upwards showing two cupolae, the right being slightly higher than the left. It is made up of muscle fibres peripherally and is tendinous centrally.

The lungs

The two lungs are basically very similar (Figure 9.2). The right lung is made up of three lobes and the left of two lobes. The lingular segment of the left lung corresponds to the middle lobe on the right. Each lobe is divided into segments.

The thoracic cage is lined by the pleura. There are two layers, the parietal and visceral, which are continuous with each other and enclose the pleural space. The parietal pleura is the outer layer and lines the thoracic cavity. The visceral pleura covers the surface of the lung, entering into the fissures and covering the interlobar surfaces. The two layers are lubricated by a thin layer of pleural fluid lying within the pleural space, which, in healthy individuals, contains no other structure.

The oesophagus

The oesophagus is a muscular tube stretching from the pharynx to the stomach. It is composed of mucosa and circular and longitudinal muscle layers. The oesophagus enters the stomach below the diaphragm at approximately the level of the eleventh thoracic vertebra.

THORACIC SURGERY

Indications for surgery

Tumour

The most common reason for pulmonary and oesophageal resection is a malignant tumour (carcinoma). A small percentage of tumours can be benign.

Lung cancer is the most common cause of cancer in the world and the second most common form in the UK, second to breast cancer. In 2009 there were 41,500 new cases of lung cancer diagnosed in the UK. The male to female ratio is 4:3.

Oesophageal cancer is the ninth most common form of cancer in the UK. There were 8,161 new cases diagnosed in the UK in 2009 (Cancer Research UK).

Lung cancers are classified into two main categories (NICE 2005):

- small-cell: 20% of all cases;
- non-small-cell: 80% of all cases including squamous cell carcinoma, adenocarcinoma and large cell carcinoma.

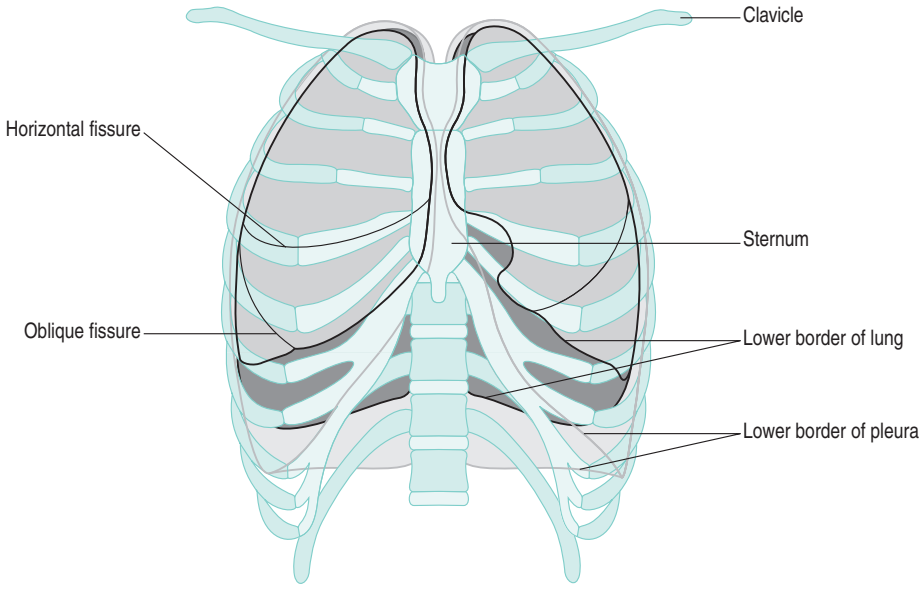


Figure 9.1 Anatomy of the thorax. *(Reproduced from Jacob 2001, with permission.)*

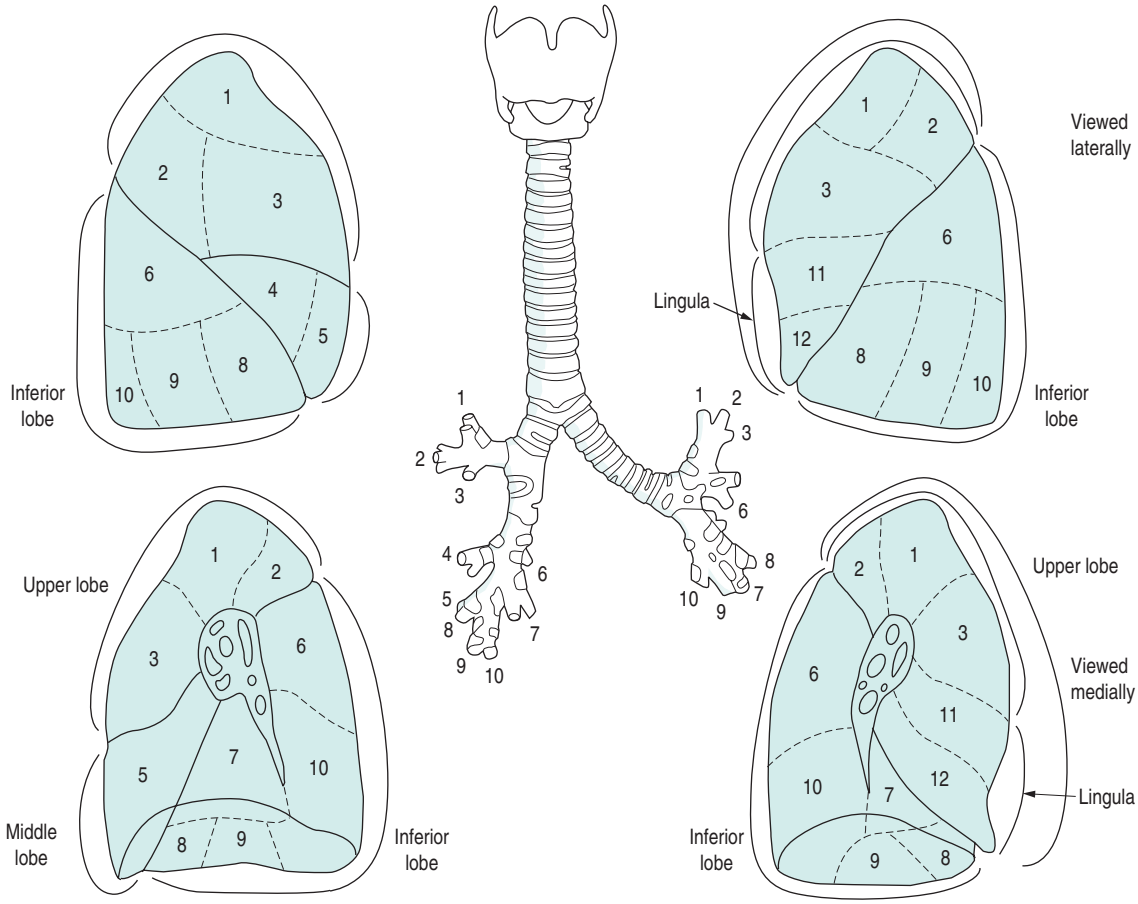


Figure 9.2 Anatomy of the lungs. Bronchopulmonary segments: 1 = apical, 2 = posterior, 3 = anterior, 4 = lateral, 5 = medial, 6 = apical basal, 7 = medial basal, 8 = anterior basal, 9 = lateral basal, 10 = posterior basal, 11 = superior, 12 = inferior. *(Reproduced from Palastanga et al. 2002, with permission.)*

Non-small-cell tumours are treated by resection if possible, if the tumour can be safely removed with clear margins and if metastatic disease is not in evidence. Small-cell cancer is virtually always widespread at diagnosis, so surgery is usually not an option.

Malignant tumours of the oesophagus are generally adenocarcinoma, especially in the lower end. They may have arisen in the cardia of the stomach and spread proximally. In the middle and upper oesophagus, squamous carcinomas predominate.

Benign tumours of the oesophagus and lungs are rare.

Pneumothorax

This is a collection of air in the pleural cavity. It usually occurs spontaneously and is caused by rupture of the visceral pleura of an otherwise healthy lung. This is more common in men than women and more usual in those under 40 years of age.

Patients with chronic obstructive pulmonary disease (COPD) can rupture a bulla resulting in a pneumothorax. Other, much rarer, causes include tumour, abscess and tuberculosis (TB). Traumatic pneumothoraces can occur with blunt trauma to the chest wall, such as following a car accident or heavy fall, or from a penetrating chest wound, i.e. a stab or gunshot wound. Iatrogenic (medical in origin) pneumothoraces can occur following intravenous line insertion, after pacemaker insertion or in ventilated patients on high levels of positive end expiratory pressure (PEEP).

Empyema

Empyema is a collection of pus in the pleural cavity. The cause is commonly pneumonia, lung carcinoma or abscess, bronchiectasis or, more rarely, TB. It can occur in patients with septicaemia or osteomyelitis of the spine or ribs. Most empyemas are located basally but they can occur between two lobes.

Bronchiectasis

Bronchiectasis is a chronic lung condition in which abnormal dilatation of the bronchi occurs associated with obstruction and infection. Patients present with excessive production of purulent secretions, which become chronically infected. Bronchiectasis is generally managed medically with a physiotherapy regime and antibiotics. In some severe cases where the condition is localised to one area of the lung, lobectomy can offer some relief of symptoms.

Oesophageal perforation

Trauma and perforation to the oesophagus may result from the accidental swallowing of a foreign body (such as a dental plate). The oesophagus can rupture in cases of severe vomiting, especially if the patient tries to suppress the vomiting action. Iatrogenic perforation can occur following oesophagoscopy or surgery associated with the pharynx.

Pre-operative investigations

Patients are assessed pre-operatively in order to establish the nature of the lesion and whether they are fit for surgery. The following investigations are commonly done.

Chest X-ray

A standard chest X-ray will be done on all patients to establish pre-operative lung status.

Computerised tomography scan

In patients with cancer a computerised tomography (CT) scan is done universally. The scan will locate the lesion accurately and show if there is invasion into surrounding structures, which determines operability. The presence of metastases in distant organs is a contraindication to surgery.

Positron emission tomography scan

In lung cancer, positron emission tomography (PET) scans are sometimes used to look for cancer in the lymph nodes in the centre of the chest or to show whether the cancer has spread to other areas. This assists with decision-making regarding adjuvant radiotherapy and surgical intervention.

Bronchoscopy/oesophagoscopy

This will establish the site of the lesion and allow biopsy or bronchial washings to be sent for histology. It can be carried out under sedation or general anaesthesia.

Pulmonary function tests

Pulmonary function tests will help the surgeon decide whether the patient can withstand lung resection. It will also provide the anaesthetist with valuable information to assess suitability for general anaesthesia.

Arterial blood gases

Arterial blood gases may be analysed routinely at some hospitals or on high-risk patients, such as those with a pre-existing lung condition.

Types of thoracic incision

Posterolateral thoracotomy

This incision is most commonly used for operations on the lung (Figure 9.3). It is a curved incision that starts at

Figure 9.3 The incision for a posterolateral thoracotomy.

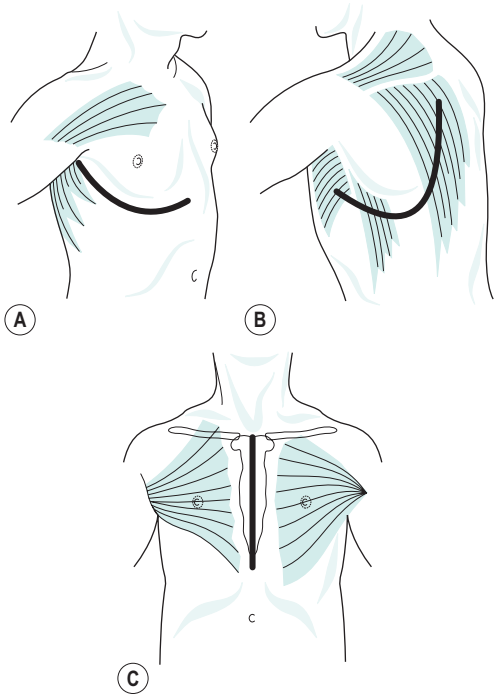


Figure 9.4 Sternotomy. Common incisions. (a) Anterolateral thoracotomy; (b) posterolateral thoracotomy; (c) median sternotomy.

the level of the third thoracic vertebra and follows the vertebral border of the scapula and the line of the rib extending forward to the anterior angle or costal margin. An incision through the bed of the fifth or sixth rib is used for pneumonectomy or lobectomy.

The muscles involved are the trapezius, latissimus dorsi, rhomboids, serratus anterior and the corresponding intercostal. A small piece of rib, approximately 1 cm, may be removed to allow easier retraction and avoid a painful fracture.

Anterolateral thoracotomy

This incision is used primarily for cardiac surgery but can be used to perform pleurectomy.

The incision starts at the level of the fifth costal cartilage. At the sternal edge it follows the rib line below the breast to the posterior axillary line. The muscles cut are pectoralis major and minor, serratus anterior and the corresponding intercostal (Figure 9.4).

Median sternotomy

This incision is used for lung volume reduction surgery and bilateral pleurectomy. It is a vertical incision that involves splitting the sternum.

The incision extends from just above the suprasternal notch to a point about 3 cm below the xiphisternum. No muscle is cut except the aponeuroses of pectoralis major (see Figure 9.4).

Left thoraco-laparotomy

This incision is used for surgery on the lower oesophagus and stomach. The thoracotomy incision follows the curve of the seventh rib and extends anteriorly over the costal margin towards the umbilicus. The muscles involved are lattisimus dorsi, serratus anterior, the corresponding intercostal and the abdominal muscles.

If an oesophageal tumour is involving the middle third of the oesophagus, surgical access may be easier through a right thoracotomy and a separate abdominal incision. If the tumour is in the upper third, then a cervical incision will also be required.

Video-assisted thoracoscopic incisions

This technique aims to carry out conventional thoracic operations through several very small (1–2 cm) incisions as opposed to a posterolateral thoracotomy. Instead of the surgeon seeing inside the patient directly, an endoscope with video camera attachment is introduced into the chest through one of the small incisions – the surgeon sees the image produced on television monitors in theatre. Specialised instruments are inserted via the other incisions so the operation can be completed. Advantages are reduced pain and less impact on respiratory mechanics in the postoperative period, and much smaller scars.

OPERATIONS ON THE LUNG

Figure 9.5 shows resection margins in lung surgery.

Pneumonectomy

Extrapericardial pneumonectomy is carried out for tumours involving a main bronchus. The whole lung is removed and the resulting cavity will fill with protein-rich fluid and fibrin over a period of weeks.

Lateral shift of the mediastinum, upward shift of the diaphragm and reduction of the intercostal spacing on the operated side reduce the size of the cavity.

Intrapericardial pneumonectomy is a more radical procedure involving the removal of part of the pericardium. This is required when the tumour growth involves the pericardium.

Lobectomy

This means removal of a complete lobe with its lobar bronchus. On the right side, two lobes can be removed together – the upper and middle or middle and lower. Removal of the upper lobe on the right, known as a 'sleeve resection' can sometimes include a section of right main bronchus.

Segmental resection

A segment of a lobe along with its segmental artery and bronchus are removed.

Wedge resection

This is a small local resection of lung tissue.

Lung volume-reduction surgery

Lung volume-reduction surgery is a procedure designed to improve respiratory function in patients with severe bullous emphysema. These patients present with hyperinflated lungs and a flattened diaphragm. By excising the bullous tissue and shaping the remaining lung, expansion of the healthy lung and doming of the diaphragm can be achieved. This will result in improved respiratory mechanics and symptomatic relief of dyspnoea (breathlessness). Patients should undergo a period of pulmonary rehabilitation pre-operatively to maximise their respiratory function.

Complications of pulmonary surgery

The major complications of pulmonary surgery are listed in Table 9.1.

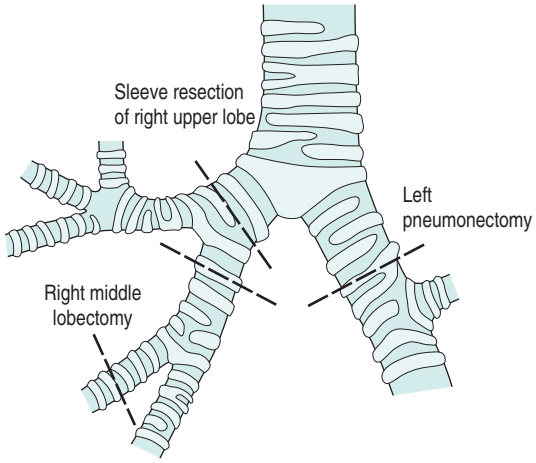


Figure 9.5 Resection margins in lung surgery.

Table 9.1 Complications of pulmonary surgery

Respiratory
- Sputum retention ± infection
- Atelectasis/lobar collapse
- Persistent air leak/pneumothorax
- Bronchopleural fistula (breakdown of the bronchus from which the lung tissue has been resected, more likely to occur following pneumonectomy and generally occurs about 8–10 days after surgery)
- Pleural effusion
- Surgical emphysema
- Respiratory failure

Circulatory
- Haemorrhage
- Cardiac arrhythmia: atrial fibrillation will occur in approximately 30% of lung resection patients
- Deep vein thrombosis
- Pulmonary embolus
- Myocardial infarction

Wound
- Infection
- Chronic wound pain
- Failure to heal

Neurological
- Stroke
- Recurrent laryngeal nerve (RLN) damage (the RLN supplies the vocal chords and trauma during surgery will impair the patients' ability to cough)
- Phrenic nerve damage, resulting in paralysis of the hemi-diaphragm

Loss of joint range
- Loss of shoulder range on operated side
- Postural changes

OPERATIONS ON THE PLEURA

Pleurectomy

Recurrent pneumothoraces will require surgical treatment. In young patients this is usually on the second or third occasion. In a small number of patients, bilateral pleurectomy will be required. In the older patient presenting with pneumothorax as a complication of COPD, surgery may be required on the first occasion.

The procedure involves removing the parietal layer of pleura from the chest wall in the area adjacent to the lung injury. This leaves a raw area to which the lung becomes adherent and thus unable to 'collapse' again. At the same time any bullous lung tissue can be either ligated or excised.

Decortication

Decortication is carried out following chronic empyema. The procedure involves the removal of the thickened, fibrous layer of visceral pleura from the surface of the lung. This allows the lung to re-expand into the space previously occupied by the empyema.

OPERATIONS ON THE OESOPHAGUS

Oesophageal resection

Tumours in the lower third of the oesophagus are resected via a left thoracolaparotomy. The upper third of the stomach is removed and the oesophagus from about 10 cm above the tumour margins. The remaining stomach is passed through the hiatus of the diaphragm into the posterior pleural cavity and a circular anastamosis created between the distal oesophagus and tip of the gastric tube.

Tumours in the middle third are more easily dealt with via a right thoracotomy and separate laparotomy. This is known as an Ivor–Lewis procedure. The stomach is passed into the right pleural cavity and the anastamosis constructed above the level of the aortic arch.

Tumours of the upper third will require resection of virtually all the oesophagus and the anastamosis will be made via an incision in the neck. The stomach is placed as with the previous procedures.

Repair of oesophageal perforations

Oesophageal perforations are treated surgically by direct repair. The site of the perforation will decide the nature of the operation. Some perforations can be treated conservatively and allow natural healing of the perforation without operative intervention.

Complications of oesophageal surgery

All the complications of pulmonary surgery apply to oesophageal operations. In addition, there may be chylothorax, a pleural effusion resulting from the severing of the thoracic chyle duct. The effusion when drained will appear milky and on testing contain fat-staining globules. Occasionally, surgical repair will be required, but usually an intercostal drain (see below) will suffice until the leak stops.

There may also be anastamosis breakdown, usually owing to variable degree of gastric tubes necrosis.

INTERCOSTAL DRAINS

Key point

When thoracotomy has been performed and the pleura opened, it is necessary to insert chest drains. These will commonly be referred to as intercostal drains.

Most patients will require two intercostal drains, one sited in the apex of the pleural cavity to drain air and allow the lung to re-expand, and a second drain in the basal area to drain postoperative bleeding. Patients undergoing pneumonectomy need only a single drain.

The drainage tubes are introduced through stab incisions, in an intercostal space below the level of the thoracotomy and positioned within the chest before closure. They are secured with a purse-string suture which will allow a tight seal to be achieved while the drain is *in situ* and on its removal. The apical drain is generally sited anteriorly and the basal posteriorly. It is always wise to check this in the operation notes.

The drainage tube passes from inside the pleural cavity down to a bottle containing sterile water and attaches to a tube that continues to below the level of the water (Figure 9.6). Above the water level is a second tube that is open to the atmosphere. This maintains atmospheric pressure within the bottle. This is known as 'an underwater sealed drain' and provides a simple, one-way valve allowing air and blood to drain from the pleural cavity. Suction can be applied to the short tube to reduce the pressure in the bottle to below atmospheric and therefore encourage the evacuation of air and blood.

On free drainage the fluid level in the tubing will rise and fall. On inspiration the negative pressure in the thorax pulls water up the tube and on expiration there is less negative pressure in the thorax and the water level falls. If the level ceases to swing then the lung is either fully

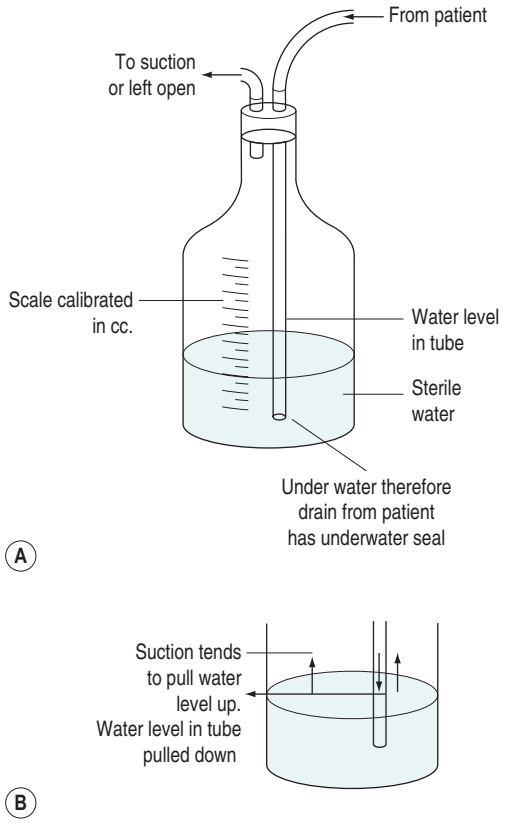


Figure 9.6 Effects of suction on water level: (a) the drainage bottle; (b) the effect of suction on water level; (c) the effect of the patient's breathing.

expanded or the drain is blocked. There will be no swing if the drain is connected to suction.

Drainage bottles must be kept below the level of insertion to prevent the siphoning of fluid back into the pleural cavity (Figure 9.7).

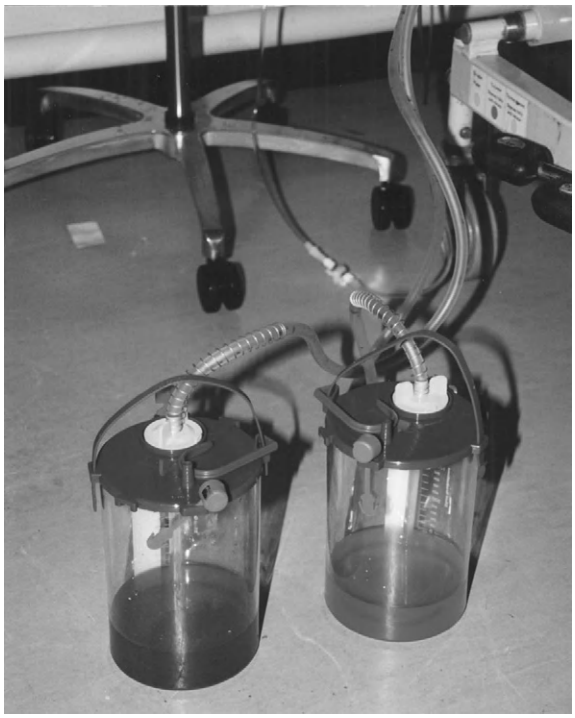


Figure 9.7 Underwater seal drainage in use.

Amount and type of drainage

The amount of drainage is measured on a calibrated scale on the side of the bottle. Initially, this will be bloodstained but progress to serous fluid and then stop. Air drainage can be seen as bubbles in the water, especially after coughing. Persistent air leaks are the result of a hole in the lung tissue or at the resection site. Bubbling may continue for many days and an apical drain cannot be removed until this stops. The basal drain will be taken out 24–48 hours postoperatively and the apical drain after 48–72 hours unless there is continued drainage.

PAIN CONTROL IN THORACIC SURGERY

For all the happiness man can gain is not in pleasure but rest from pain.

John Dryden (1631–1700)

Postoperative pain relief is not solely for the relief of an unpleasant sensation. Disturbances of pulmonary function are common after any form of intrathoracic

operation. A decrease in functional residual capacity (FRC) with minimal change in closing volume leads to atelectasis (Sabanathan et al. 1990). Patients also experience an inability to cough effectively, thus becoming prone to sputum retention leading to infection and arterial hypoxaemia (Ali et al. 1974). Pain from the incision site and drains can be severe for up to three days (Kaplan et al. 1975) and abnormal patterns of breathing owing to pain will only worsen these problems.

Good postoperative pain control is essential in order to carry out effective physiotherapy. This can be delivered in several ways: epidural anaesthesia, paravertebral block, patient-controlled analgesia (PCA), transcutaneous nerve stimulation (TENS) and oral analgesia are the most commonly used.

Epidural anaesthesia

An epidural provides delivery of a local anaesthetic agent, such as bupivicaine, and an opiate, such as fentanyl, directly into the small space just outside the dura mater – the 'epidural space' (Figure 9.8). The local agent will provide dermatomal relief over the incision site and the opiate a more central effect.

Figure 9.8 An epidural used to provide pain relief after surgery.

Epidurals can provide profound analgesia in considerably smaller doses of opiate drug than if used systemically (Chaney 1995). This will minimise the unwanted side effect of respiratory depression commonly seen in opiate use. Lui et al. (1995) demonstrated improved analgesia with physiotherapy in thoracotomy patients using bupivicaine epidurals. An epidural will be inserted by an anaesthetist before the operation begins.

Paravertebral block

If it is not possible to insert an epidural, continuous delivery of a local anaesthetic agent can be achieved using a paravertebral catheter positioned in the paravertebral groove. This can provide safe and effective pain relief after thoracotomy (Inderbitzi et al. 1992). The catheter will be sited by the surgeon prior to closure of the chest.

Patient-controlled analgesia

PCA allows the administration of small doses of intravenous opioids on demand by the patient. The patient must be awake, co-operative and have had adequate instruction pre-operatively on how to use the system. The dose delivered is dependent upon patient weight. A 'lock-out' interval is set to allow time for the opiate to work; this also prevents overdosing.

Transcutaneous nerve stimulation

TENS can be useful if it is initiated postoperatively to relieve referred shoulder pain. The phrenic nerve supplies the diaphragm and if irritated during surgery patients can experience ipsilateral referred shoulder pain (Scawn et al. 2001). TENS can also be of benefit in patients with persistent wound pain when an epidural or paravertebral has been removed.

Oral analgesia

Epidurals, paravertebrals and PCAs will continue, on average, for 72 hours, but analgesia will still be required for many days. The pain experience is individual and oral analgesia required will vary from patient to patient. Simple analgesia, such as paracetamol or ibuprofen, may be adequate, but some patients require stronger medication, such as dihydrocodeine or diclofenac. Oral medication can be prescribed on a regular or an 'as required' (p.r.n.) basis.

Most hospitals will have a specialist nurse for pain control. The nurse will be very helpful in the care of patients with severe pain that is difficult to control on standard analgesia.

THE PHYSIOTHERAPIST AND THORACIC SURGERY

Key point

Chest physiotherapy has a place in the prevention, as well as the treatment of postoperative pulmonary complications.

Pre-operative care

The provision of pre-operative chest physiotherapy is not routine, but it has been shown to be of benefit in high-risk patients. For example, Nagasaki et al. (1982) demonstrated that pre-operative physiotherapy for elderly patients and those with COPD reduced postoperative pulmonary morbidity.

Patients with pre-existing COPD are prone to increased bronchial secretions (Massard and Wihlm 1998) and may require chest clearance prior to surgery. Physiotherapy may be requested by the patient's medical team following bronchoscopic findings (i.e. sputum retention).

The pre-operative care may vary from simple education in postoperative techniques to more intensive chest clearance.

Postoperative care

Postoperative complications commonly present as a restrictive pattern with reduced inspiratory capacity, reduced vital capacity (VC) and reduced FRC (Craig 1981). There are changes in defence mechanisms owing to anaesthesia and reduced cough effort (Scuderi and Olsen 1989) that can lead to retention of secretions.

Postoperative physiotherapy aims to minimise the risk of non-infectious and infectious pulmonary complications (Scuderi and Olsen 1989), the most common being atelectasis and pneumonia. Other common problems are loss of joint range in the shoulder on the incision side and reduced mobility. Therefore, the main aims of physiotherapy are:

- patient education;
- maximisation of lung volume;

Key point

Communication is of great importance in the successful treatment of thoracic patients. The physiotherapist must communicate with nursing and medical staff in order to monitor the patient's progress. Medical notes and chest X-rays should be monitored on a daily basis. Effective communication skills will result in improved patient compliance and make treatment more effective.

Key point

The provision of chest physiotherapy after thoracic surgery is fairly routine in the UK, even though there has been little specific research on the subject.

- prevention of sputum retention;
- sputum clearance;
- maintenance of shoulder range of movement;
- early mobilisation.

Patient assessment

The initial assessment of the patient leads to identification of specific problems. Without an accurate assessment an appropriate treatment plan cannot be initiated (Pryor and Webber 1998). Re-assessment is then an ongoing process to judge the effectiveness of treatment, to identify new problems and to modify a treatment plan. Table 9.2 lists what the initial patient assessment should include.

Following assessment, the problems identified will commonly include:

Table 9.2 The initial patient assessment notes

Database: obtained from medical notes
- Pre-operative information: pulmonary function tests and arterial blood gases
- Surgical procedure and incision
- Concise relevant history of present condition
- Relevant past medical history including previous surgery
- Social history
- Drug history, specific note of respiratory medicines, e.g. inhalers

Subjective: information the patient tells you
- Ask open-ended questions: How do you feel?
- Ask about pain control: Can the person cough?

Objective: information based on examination of the patient and tests carried out
- Cardiovascular status (CVS): blood pressure, heart rate and rhythm
- Oxygen delivery system and FIV1
- Blood gases or O_2 saturation
- Respiratory rate
- Chest X-ray
- Method of pain control
- Number and type of drains
- Auscultation
- Ability to cough
- Range of movement of shoulder on incision side

- reduced lung volume;
- retention of secretions;
- increased work of breathing;
- poor breathing control/pattern;
- ineffective cough;
- pain.

MODALITIES OF PHYSIOTHERAPY

From the initial assessment and problem identification a treatment plan can be formulated.

The amount of chest physiotherapy required will vary from patient to patient. The patient's individual requirements will primarily dictate how often and for how long treatment is needed. Consultant preference and hospital protocols may also influence this (Stiller and Munday 1992).

Key point

A particular treatment modality can be used to address more than one problem. For example, the active cycle of breathing technique (ACBT – see below) will be effective in treating reduced lung volume and sputum retention.

Breathing exercises

The active cycle of breathing technique (ACBT) used in sitting may be sufficient to maintain effective airway clearance (Pryor and Webber 1998). ACBT consists of cycles of breathing control and thoracic expansion exercises followed by the forced expiratory technique (FET) (see Figure 9.9). The thoracic expansion exercises can be combined with inspiratory hold and vibrations. In patients with reduced breath sounds, atelectasis and/or sputum retention positioning in conjunction with ACBT may be indicated.

The whole cycle should be repeated 2–3 times or until the patient becomes non-productive. In early postoperative patients, fatigue may be an issue and treatment should be terminated at this point.

The thoracic expansions should be slow deep breaths in through the nose and sigh out through the mouth. The end-inspiratory hold can improve air flow to poorly ventilated regions (Hough 2001); the breath hold should be encouraged at the height of the inspiratory effort for 2–3 seconds.

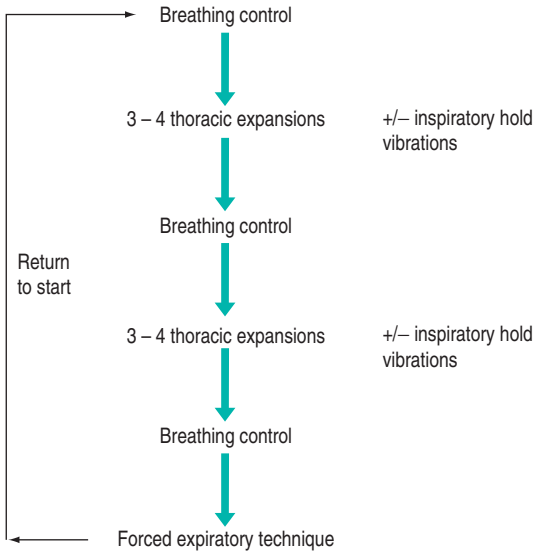


Figure 9.9 The active cycle of breathing technique (ACBT). *(Adapted from Pryor and Webber 1998.)*

Patients should be encouraged to carry out at least two full cycles every waking hour in order to maintain improvements gained in lung function.

Forced expiration

The FET is used to help in the clearance of excess bronchial secretions.

The forced expiratory technique

The technique was defined by Webber and Pryor in 1979. FET is one or two forced expirations from mid-lung to low-lung volumes (Partridge et al. 1989).

An effective FET should sound like a forced sigh. It is dependent on:

- mouth open;
- glottis open;
- abdominal wall contracted;
- chest wall contracted.

Crackles may be heard if secretions are present.

FET performed to low lung volumes will aid removal of secretions peripherally situated. High lung volumes will clear secretions from proximal airways (Pryor and Webber 1998).

Supported cough

A cough is created by forced expiration against a closed glottis. This causes a rise in intrathoracic pressure. As the

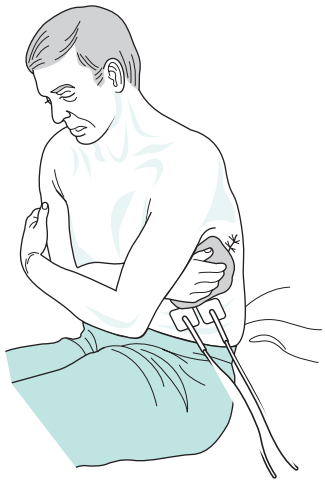


Figure 9.10 Coughing: patient supporting wound.

glottis opens there is rapid, outward airflow and shearing of secretions from the airway walls.

Improved coughing and FET can be achieved if the wound is supported (Figure 9.10). This can be done by the physiotherapist during treatment sessions or by the patient. The arm on the unoperated side is placed across the front of the thorax and over the incision and drain sites. Firm overpressure is applied during coughing/FET. A towel, folded lengthways, passed around the back of the patient and pulled across the front of the thorax can also be useful to support coughing.

Positioning

Key point

The major function of positioning postoperatively is to improve FRC. A good sitting position either in or out of bed, as opposed to slumped in the bed, can achieve this (Jenkins et al. 1988).

There are recognised positions for segmental drainage (Thoracic Society 1950) and these may be utilised if there is a segmental or lobar-specific problem. It is much more likely, however, that modified positions only will be required, especially in view of the changed anatomy of the area.

Positioning can also be used to improve gas exchange. Improvement in oxygenation can be achieved in side-lying with the affected lung uppermost; the ventilation/perfusion match is improved, resulting in increased oxygen uptake (Winslow et al. 1990).

Pneumonectomy patients should *not* be positioned on their unoperated side. This can result in bronchopleural fistula owing to space fluid washing over the bronchial stump. Patients undergoing intrapericardial pneumonectomy should be treated in sitting for the first four days unless advised otherwise by the medical team.

Early mobilisation

Mobilisation should commence as soon as is safely possible as functional residual capacity is maximally improved in standing (Jenkins et al. 1988). Dull and Dull (1983) proposed that early mobilisation in uncomplicated patients could render breathing exercises unnecessary. Patients must be cardiovascularly stable and not requiring high concentrations of oxygen before mobilisation can begin. If intercostal drains are on suction, mobility will be restricted to standing and spot-marching at the bedside. Some anaesthetic departments restrict mobility when an epidural is *in situ* owing to the risk of profound hypotension on mobilising. Hospital protocols should be noted.

Shoulder exercises

The shoulder on the operated side should be checked for range of movement. The patient should practise elevation and abduction of the shoulder at least three times a day. Auto-assisted exercises may be necessary to begin with. Any limitation of range should be more formally assessed and treated.

Adjuncts to physiotherapy

Physiotherapy is a 'hands on' practice. There are several adjuncts that can be used to augment the basic breathing exercise regimen.

Incentive spirometer

Incentive spirometry is a feedback system to encourage patients to take a deep breath and produce a sustained maximal inspiration in order to open atelectatic areas of lung (Su et al. 1991) (Figures 9.11 and 9.12). It is cheap to provide, non-invasive and, when taught well, needs minimal supervision.

Bastin et al. (1997) deduced that deterioration in incentive spirometer performance could be used as a warning of pulmonary deterioration.

Mini-tracheostomy

Sputum retention is a frequent complication in patients recovering from thoracic surgery (Busch et al. 1994). It can be a result of sputum tenacity or a weak ineffective cough. Mini-tracheostomy can be an invaluable tool in

Figure 9.11 The incentive spirometer.

Figure 9.12 The incentive spirometer in use.

the postoperative patient to aid removal of secretions in conjunction with a physiotherapy regimen.

Quidaciolu et al. (1994) concluded that mini-tracheostomy was safe and effective in reducing respiratory morbidity in high-risk patients following pulmonary surgery.

Heated humidification

Pulmonary secretions can become tenacious following surgery. This may be a result of anaesthesia, infection or dehydration – especially in oesophageal patients who are 'nil by mouth'. Improving humidification to the airways by heating the oxygen/air delivery can help in mucus clearance.

Continuous positive airways pressure

Patients with poor arterial blood gases and reduced lung volume can be supported by the use of continuous positive airways pressure (CPAP). It can be used continuously or intermittently. When used continuously it must be humidified.

CPAP is effective in improving FRC and arterial oxygen in patients with acute respiratory failure. It can also reduce the work of breathing (Keilty and Bott 1992). Care must be taken in thoracic patients because of the anastamosis and medical opinion should be sought.

Intermittent positive pressure breathing

There is little literature to support the use of intermittent positive pressure breathing (IPPB), but with good teaching and in the right patient it can be effective. It is of particular use in patients who have loss of lung volume and are tiring. It works to improve lung volume and reduces the work of breathing.

Extreme care must be used when considering IPPB on lung resection patients as the anastamosis may be vulnerable to positive pressure. The physiotherapist should discuss the use of IPPB with the patient's medical team.

DISCHARGE

Patients will reach discharge from treatment at varying points in their recovery. A low-risk patient with no post-operative complications may only need 3–4 days of physiotherapy. High-risk patients and those experiencing pulmonary complications will need considerably more. FRC and VC can be regained even in lung resection patients. The time for full recovery of FRC is about two weeks and for VC it can be in excess of three weeks (Craig 1981). Once discharged, the patient should be advised to continue with regular breathing exercises and gradually increase their mobility.

ACKNOWLEDGEMENTS

The authors and editor would like to thank Mr Richard Page (consultant thoracic surgeon), Jenny Chauveau (acute pain nurse specialist), Mary Kilcoyne (physiotherapist) and all physiotherapy staff at Liverpool Heart and Chest Hospital NHS Foundation Trust and Jo Sharp (lecturer) at the University of Liverpool.

REFERENCES

Ali, J., Weisel, R.D., Layug, A.B., et al., 1974. Consequences of postoperative alterations in respiratory mechanics. Am J Surg 128, 376–382.

Bastin, R., Moraine, J-J., Bardocsky, G., et al., 1997. Incentive spirometry performance: a reliable indicator of pulmonary function in the early postoperative period after lobectomy? Chest 111, 559–563.

Busch, E., Verazin, G., Antkowiak, V.G., et al., 1994. Pulmonary complications in patients undergoing thoracotomy for lung carcinoma. Chest 105, 760–766.

Cancer Research UK, 2012. Cancerstats – Key Facts: Oesophageal Cancer; http://www.cancerresearchuk.org/cancer-info/cancerstats/keyfacts/oesophageal-cancer/, accessed October 2012.

Cancer Research UK, 2012. Cancerstats – Key Facts: Lung Cancer and Smoking; http://www.cancerresearchuk.org/cancer-info/cancerstats/keyfacts/lung-cancer/

Chaney, M.A., 1995. Side-effects of intrathecal and epidural opioids. Can J Anaesth 42, 891–903.

Craig, D., 1981. Postoperative recovery of pulmonary function. Anaesth Analg 60 (1), 46–52.

Dull, J.L., Dull, W.L., 1983. Are maximal inspiratory breathing exercises or incentive spirometry better than early mobilisation after cardiopulmonary bypass? Phys Ther 63, 655–659.

Hough, A., 2001. Physiotherapy in Respiratory Care, third ed. Nelson Thornes, London.

Inderbitzi, R., Fleveckiger, K., Ris, H.B., 1992. Pain relief and respiratory mechanics during continuous intrapleural bupivicaine administration after thoracotomy. Thorac Cardiovasc Surg 40 (2), 87–89.

Jacob, S., 2001. Atlas of Human Anatomy. Churchill Livingstone, Edinburgh.

Jenkins, S.C., Soutar, S.A., Moxham, J., 1988. The effects of posture on lung volumes in normal subjects and in pre- and post-coronary artery surgery. Physiotherapy 74, 492–496.

Kaplan, J.A., Miller, E.D., Gallagher, E.G., 1975. Postoperative analgesia for thoracotomy patients. Anaesth Analg 54, 773.

Keilty, S.E., Bott, J., 1992. Continuous positive airways pressure. Physiotherapy 78 (2), 90–92.

Lui, S., Angel, J.M., Owens, B.D., et al., 1995. Effects of epidural bupivicaine after thoracotomy. Reg Anaesth 20, 303–310.

Massard, G., Wihlm, J.M., 1998. Postoperative atelectasis. Chest Surg Clin N Am 8, 281–290.

Nagasaki, F., Flehinger, B.J., Martini, N., 1982. Complications of surgery in the treatment of carcinoma of the lung. Chest 82 (1), 25–29.

NICE (National Institute for Clinical Evidence), 2005. Lung Cancer: The Diagnosis and Treatment of Lung Cancer – Clinical Guideline 24. NICE, London.

Palastanga, M., Field, D., Soames, R., 2002. Anatomy and Human Movement, fourth ed. Butterworth-Heinemann, Oxford.

Partridge, C., Pryor, J., Webber, B., 1989. Characteristics of the forced expiratory technique. Physiotherapy 75 (3), 193–194.

Pryor, J.A., Webber, B.A., 1998. Physiotherapy for Respiratory and Cardiac Problems. Churchill Livingstone, Edinburgh.

Quidaciolu, F., Gausone, F., Pastorino, G., et al., 1994. Use of mini-tracheostomy in high risk pulmonary resection surgery: results of a comparative study. Minerva Chir 49, 315–318.

Sabanathan, S., Eng, J., Mearns, A.J., 1990. Alterations in respiratory mechanics following thoracotomy. J R Coll Surg Edinb 35 (3), 144–150.

Scawn, N.D., Pennefather, S.H., Soorae, A., et al., 2001. Ipsilateral shoulder pain after thoracotomy with epidural and the influence of phrenic nerve infiltration with lidocaine. Anesth Analg 93, 260–264.

Scuderi, J., Olsen, G.N., 1989. Respiratory therapy in the management of post operative complications. Respiratory Care 34, 281–290.

Stiller, K.R., Munday, R.M., 1992. Chest physiotherapy for the surgical patient. Br J Surg 79, 745–749.

Su, M., Chiang, C.D., Huang, W.L., et al., 1991. A new device of incentive spirometry. Zhonghua Yi Xue Za Zhi (Taipei) 48 (4), 274–277.

Thoracic Society, 1950. The nomenclature of broncho-pulmonary anatomy. Thorax 5, 222–228.

Winslow, E.H., Clark, A.P., White, K.M., et al., 1990. Effects of a lateral turn on mixed venous oxygen saturation and heart rate. Heart Lung 19, 551–561.

Chapter 10

Changing relationships for promoting health

Sally French and John Swain

INTRODUCTION

Our focus for this chapter is 'changing relationships'. The chapter is divided into three sections. The first, 'between people', focusses on the dynamics of relationships within therapy practice, at the interpersonal level, and also broadens the discussion to the group level, looking at questions of culture. The second section, 'context of relationships', considers the social context of relationships, particularly inequalities in health and healthcare, and the various ideologies and viewpoints that prevail in therapy practice. Finally, in 'changing relationships', we look towards the possibilities for changing the dynamics of relationships, focussing first on the notion of partnership and then on health promotion itself. We both work in the field of disability studies and will draw examples from our own work and that of others in this field. Thus, the chapter is particularly orientated towards disability and therapy with disabled people. The processes we are examining, however, are general and, overall, we hope to demonstrate the interrelationships between the interpersonal, personal, cultural and societal in the dynamics of changing relationships to improve and promote health.

Labelling people

Various terms are used to refer to disabled people in relation to services. All are being challenged by disabled people themselves.

- Patient: this term clearly labels people in medical terms and is derived from 'pati', meaning 'suffer'.
- Client: this refers to a person using the services of a professional (lawyer, architect, medical practitioner, etc.). In recent times, the term 'customer' has also been used.
- Service user: this is, perhaps, the most neutral term, although it still labels people according to their relationship with services.

BETWEEN PEOPLE

Communication

The provision of physiotherapy can be thought of as a form of communication and the whole process as one of communication between the client and the physiotherapist, between the physiotherapist and colleagues (including other physiotherapists, doctors, etc.) and between the physiotherapist and members of the client's family. The purpose of this section, then, is to examine interpersonal communication in the particular context of physiotherapy practice.

There are a number of important aspects to a social model of communication. Interpersonal communication is viewed as an interplay between people in which participants are both active agents, affecting the interplay, and reactive agents, affected by the interplay. There is no simple one-to-one correspondence between acts of communication and the meaning expressed. The context is all important. Silence, for instance, can 'say' more than words, but has no meaning outside the particular interpersonal and social context. It could be taken to mean the person is experiencing boredom, fear, respect, deep mutual understanding, passion, hatred – the list is potentially endless. Even signals that usually have a well defined meaning, such as raising the hand to signal 'stop', depend on the context of the two-way flow. If accompanied by a smile,

MODELS OF COMMUNICATION

Linear models

Linear models of communication seem to accord with common sense, as communication is viewed as a process of sending and receiving messages. A popular communication model that is used in the literature is as follows.

1. Sender (self).
2. Encoder (converting thought into message).
3. Channel (verbal, non-verbal, both verbal and non-verbal).
4. Decoder (interpretation of message).
5. Receiver (other/others).
   (Rungapadiachy 1999:195)

Social models

A social view of communication posits a dynamic model in the sense that communication is seen as a transaction constructed between people.

Social models concentrate more on the communication process itself, rather than the notion of message sending (Pearce 1994). From this perspective, improving communication is not a simple matter of improving physiotherapists' skills of expressing and listening to information. Communication is 'meaning-making' rather than 'information processing' and meanings are constructed between people. Communication is both based on and generates our perceptions, descriptions and understandings of the world or, more specifically, physiotherapy.

for instance, the raising of the hand could be meant as, and understood to be, a joke. Furthermore, this model is holistic. Meaning-making is constituted through the organisation and system of a conversation with the whole being greater than the sum of the parts. Physiotherapy–client communication is not just a means for undertaking physiotherapy practice: the participants are creating physiotherapy practice through their communication.

Barriers to communication from the viewpoint of clients

- Inequality of professional–patient relationships.
- Attitudinal barriers.
- Barriers to access to services.
- Barriers to access to information.
- Lack of concern on the part of professionals with emotional issues.
- Lack of disabled professionals.

This takes us into clients' experiences of services, concentrating on barriers to communication, recognising that any analysis of barriers to communication needs to identify the complexities of the communication process itself and the diversity of clients' experiences (Figure 10.1). Such an analysis also needs to recognise the inequality of professional–client relationships. Professional dominance can be seen in assessment procedures where, for example, the professional's observations are viewed as 'objective' whereas the patient's perceptions are viewed as 'subjective', and where pseudo-scientific language serves to mystify and confuse service users (French 1993). Because of the specialisation of the various professional groups, definitions of need tend to be narrow, their scope being dictated by specialised knowledge and interests. The needs of disabled people, however, tend to be multifaceted. As Marsh and Fisher (1992: 50) point out:

> *If the process of assessment becomes one of professional discovery of 'need', rather than a*

Figure 10.1 Barriers to communication, a means of oppressing women, or a symbol of personal, cultural and religious identity?

negotiation of problems, then users tend to feel hemmed in by the definitions used to describe their circumstances and trapped by the choices they are faced with.

Attitudinal barriers are commonly referred to by disabled clients. Boazman (1999: 18–19), for instance, had mixed responses from health professionals when she became aphasic following a brain hemorrhage:

Their responses towards me varied greatly, some showed great compassion, while others showed complete indifference. I had no way of communicating the fact that I was a bright, intelligent, whole human being. That is what hurt the most.

Similar mixed experiences were reported by people with aphasia interviewed by Parr and Byng (1998: 74). One person, talking of doctors, said:

… when you can't communicate they treat you like a kid and that is just so frustrating … A handful of doctors were just awful. You just wanted to say, 'Do you know what this is like?'

Pound and Hewitt (2004) write of the equation of communication disability with 'having nothing to say' and 'being stupid' which, they believe, illustrates the 'Does he take sugar?' syndrome (Figure 10.2). In another, small-scale study involving people with speech impairments, it was found that most difficulties were encountered within medical services. Doctors' and dentists' receptionists were singled out for particular criticism (Knight et al. 2002: 19). Mary, one participant, recalled:

My most embarrassing incidents have been with my doctor's and dentist's receptionists. I have had more trouble with them than with any other group. They were impatient and rude when I tried to make appointments, and would talk to my carer when I was trying to ask questions.

Figure 10.2 Does she take sugar?

Another participant described his encounters with hospital consultants:

They have excluded my carer from any discussion despite me indicating that I preferred to have my carer lip-read to avoid having to use my oesophageal voice.

Another common complaint of the research participants in this study was the means of access to services that generally depended on the telephone.

People with speech and language impairments are often compelled to wait long periods of time for the communication equipment they need. A survey conducted by *Scope* (Ford 2000: 29) found that nearly a fifth of people waited for more than a year. Professionals may also have control of when the equipment can be used. One of the research participants said:

Physiotherapists at school have recommended the Delta Talker be removed from situ during travelling because of possible safety problems. They also used to request removal of the talker at meal times.

Deaf people have complained about the insistence of professionals that they use speech rather than sign language. A deaf person interviewed by Corker (1996: 92) states:

I hated learning speech – hated it – I felt so stupid having to repeat the s,s,s ….. I was asking myself 'Why do I have to keep going over and over it, I don't understand what it all means' ….. It was just so stupid, a waste of time when I could have been learning more important things.

In interviews conducted by French (2004a: 99), two participants spoke of their experiences of occupational therapy:

I've often thought about OTs [occupational therapists] in rehab, if only they could think about the context from which their patients came. I was received as head of department of a girls' comprehensive school, head of physical education, and this OT said to me, 'Now you've really got to learn to type because that's what you'll be doing.' She negated the whole context of my professional life – I was just a patient.

It was a case of being treated like a patient. I felt like my feelings were being ignored, that they were just going through a routine and they would give me exercises to do which I couldn't understand the purpose of because they didn't explain. I had enough speech to ask, but I didn't ask, because I didn't have the confidence to ask.

Information can also be given in an insensitive way as Joan (interviewed by French et al. (1997: 37)) explained:

When I came back for the negatives, oh it was terrible. He lifted them up to the light and he said to the nurse 'Macula degeneration in both eyes, sign a BDS form' or whatever it is. Then he turned to me and he said 'There's nothing we can do about it …… You'll always be able to see sideways but you've got no central vision' …. So I came home feeling very upset about it.

Being unable to access information is a problem faced in all areas of life by visually impaired people, with potentially hazardous consequences of unreadable notices and loss of privacy when documents are unreadable by the intended recipient. Vale (2001) reports that appointment letters continue to be sent out in standard-size print, even by many hospital eye clinics, and only one third of National Health Service (NHS) hospitals offer general patient information in large print.

It is important to recognise that social divisions, for example gender, age and ethnicity, intersect disability and also produce communication barriers in some instances. Summarising the evidence from several studies of the experiences of disabled people from ethnic minority communities, Butt and Mirza (1996: 94) state:

The fact that major surveys of the experience of disability persist in hardly mentioning the experience of black disabled people should not deter us from appreciating the messages that emerge from existing work. Racism, sexism and disablism intermingle to amplify the need for supportive social care. However these same factors sometimes mean that black disabled people and their carers get a less than adequate service.

In their study of young black disabled people's experiences and views, Bignall and Butt (2004: 49) conclude:

Our interviews revealed that most of these young people did not have the relevant information to help them achieve independence. Hardly any knew of new provisions, such as Direct Payments, which would help with independent living. Most people did not know where to get help or information they wanted, for example, to move into their own place or go to university.

Begum (1996) takes institutionalised discrimination as her basis for analysing difficulties in the relationship between disabled women and their general practitioners (GPs). She explored physical, communication and attitudinal barriers, and found that they deny opportunities to women with impairments and can impede access to the services they require. Disabled women, for instance, often find that information is withheld from them. One of her respondents explained that she had not been told that multiple sclerosis had been diagnosed, yet her husband had been told two years before she was informed. It also seems that the flow of information from disabled people to GPs is liable to distortion and failure. This is, at least in part, owing to GPs' responses to impairment. One respondent in the research said: 'Sometimes I find that a GP – particularly one who is only here for a short time and fairly new – is more interested in my sight problem, or my child's sight problem, than in what I've come to ask about' (Begum 1996: 83–84). Age can also be a factor that distorts communication. Olwen (interviewed by French et al. (1997; 35)) talking about the attitude of professionals to her loss of sight said: 'Even though I'm older they were "…at your age what can you expect?". You know they talk to you like that.'

Disabled professionals stand in an interesting position in an analysis of communication between professionals and disabled people. It can both be argued that the barriers to communication have discriminated against disabled people wishing to be service providers and also that the acceptance of more disabled people into the professions would be a significant factor in developing inclusive communication. A visually impaired physiotherapist interviewed by French (2001: 140) spoke of poor communication with her colleagues:

I'm a registered blind person but they haven't got a clue … If I stay in one building I'm fine, it's only when I go over to G … that I get really lost … and one of the physios says, 'So you're not talking today!' because I've walked right passed them … and I've worked with them for years; oh dear … I just say 'I didn't see you' but they don't seem to learn.

Other health professionals have spoken of the advantages they have when communicating with ill and disabled clients (French 1988: 178). A disabled doctor explains:

Very many people have told me they can talk to me because I know what it feels like to have an illness. Once you get over that hump of being accepted for training then you can use your disability.

Cultural differences

ABC Definition

Culture is a general term for the symbolic and learned aspects of human society. A set of basic assumptions – shared solutions to universal problems of external adaptation (how to survive) and internal integration (how to stay together) – which have evolved over time and are handed down from one generation to the next.

Here, our focus is on inequality of power, locating culture within its socioeconomic, political and historical context. One layer of power is provided by the wider social relationships in which everyone in health and social care – service users and service providers – are embedded. In this broader social context, there are significant and constantly changing differences in power between people belonging to different groups embedded in different cultures. Some groups have greater power, resources, status and better health. Others can face discrimination – including members of ethnic minority groups, disabled and older people – in a variety of areas, including access to required services.

ABC Definition

Institutionalised discrimination is unfair or unequal treatment of individuals or groups that is built into institutional organisations and can result from the majority simply adhering unthinkingly to the existing organisational rules or social norms.

Language, in terms of cultural issues, is often seen as the main barrier to effective service provision. It is, therefore, assumed that an adequate supply of leaflets in appropriate languages and interpreters will solve the problem. However, communication consists of more than language skills and literacy. Research by Banton and Hirsch (2000: 32) suggests that even among UK-born English-speaking Asians, there is considerable lack of knowledge of what services are on offer. They state:

Communication problems are identified in all work in this area. Such problems are partly to do with language differences, but also arise from the separate lives led by different ethnic groups in our society and the consequent unlikely coincidence of communications about services arising through informal contacts.

Perhaps the most consistent recommendation from research has been the necessity for the direct involvement of disabled clients, including black disabled clients, in the planning of services (Butt and Box 1997). Again, this needs to be understood within the context of multiple discrimination. Concluding their study with young deaf Asian people and their families, Jones et al. (2001: 68) state:

… identities are not closely tied to single issues and young people and their families simultaneously held on to different identity claims. To this extent, it is not a question of forsaking one claim for another and choosing, for instance, 'deafness' over 'ethnicity', but to negotiate the space to be deaf and other things as well. It is only through addressing these tensions that services will adequately respond to the needs of Asian deaf people and their families.

In 1991, Hill drew attention to the extremes of oppression faced by black disabled people. She stated that the cumulative effect of discrimination is such that black disabled people are 'the most socially, economically and educationally deprived and oppressed members of society' (1991: 6).

Studies of the families of Asian people with learning difficulties also provide evidence of high levels of poverty, with 69% of families having no full-time wage earner and half of the families being on income support (Nadirshaw 1997). Significant language barriers were found in the same study with 95% of carers being born outside the UK and only a minority able to speak or write English.

There are dangers, however, in such statistics. Firstly, they can feed presumptions and stereotyping which belie diversity. In her study of Asian parents, for instance, Shah found that 'the majority of parents had a good command of English and, for some, English was their first language' (1998: 186). She also cites language barriers as an example of preconceived notions of discrimination experienced by Asian families. Secondly, there is a danger of oversimplifying language barriers. Language engages us in freedom of expression, release of emotion, developing cultural identity and sharing values. A common language, therefore, is no guarantee of shared understanding. At the attitudinal level of institutional discrimination, there is a lack of understanding among the majority population concerning the life style, social customs and religious practices of people from ethnic minority groups (Atkin et al. 2004). Discrimination has sometimes been denied and

rationalised through myths that, for instance, black families prefer 'to look after their own'.

Comparable analyses of the experiences of the interaction of other social divisions, for example disabled and old, disabled and female, disabled and gay or lesbian, and disabled and working-class, indicate that there are parallels. For instance, in the most extensive research into the views and experiences of disabled lesbians and bisexual women, there was evidence that they felt marginalised by lesbian and gay groups: '…many disabled lesbian and bisexual women have experienced alienation rather than nurturing and support from the lesbian and gay community' (Gillespie-Sells et al. 1998: 57).

Another, more recent, addition to the list of groups with fragmented identities whose interests are not fully taken into account by single issue movements is disabled refugees and asylum seekers who 'constitute one of the most disadvantaged groups within our society' (Roberts 2000: 945). Disabled refugees and asylum seekers are 'lost in the system' because both '…the disability movement and the refugee community focus their attention … on issues affecting the majority of their populations and fail to engage adequately with issues which affect a small minority' (Roberts 2000: 944).

CONTEXT OF RELATIONSHIPS

Inequalities in health

To the busy physiotherapist with many patients to treat and assess, it may seem that illness, impairment and accidents 'just happen' and that whether somebody has a fractured hip, a chest infection or a stroke, is largely a matter of chance. All the major research reports over the years, however, demonstrate that mortality, morbidity and life expectancy are strongly correlated with socioeconomic class with those in the lower social classes being at a considerable disadvantage (Townsend and Davidson 1982; Whitehead 1988; House of Commons Health Committee 2009). Although the UK has become healthier and wealthier over the years, health inequalities persist; in fact, the gap between the richest and poorest sectors of society has widened (Asthana and Halliday 2006; Wiles 2008).

ABC Definition

Socioeconomic status is the structural position in society of an individual or a group compared with other individuals and groups. It is a central term in the social sciences and a subject of considerable controversy. In official statistics, people are generally classified on the basic of their occupation and income.

Certain groups within society such as old people, people from ethnic minorities and disabled people are also disadvantaged partly because of their over-representation in the lower socio-economic groups (Power and Kuh 2006). There is also something of a north/south divide with people in the north of the UK having more ill health than those in the south, although there is much variation (Naidoo and Wills 2008).

This section of the chapter will examine the meaning of health inequalities. In order to do this it is necessary to consider what is meant by 'health'.

ABC Definition

Definitions of health differ between individuals, cultural groups and social classes. One view is that health is the absence of disease or illness. From a broader perspective, health includes the physical, psychological, behavioural, social and spiritual aspects of a state of well-being and recognises the importance to individuals of realising aspirations and needs.

In 1946 the World Health Organization (WHO) defined health as, 'A state of complete physical, psychological and social well being and not merely the absence of disease or infirmity' (WHO 1946, cited in Ewles and Simnett (2003: 6)). Although this definition moved the concept of health away from a biological and towards a more holistic understanding, it was criticised for being idealistic and unrealistic, and for failing to recognise that people define their health in a variety of ways based on their knowledge, values and expectations, and whether or not they can fulfil roles of importance to them (Jones 2000a). In 1984, the WHO (WHO 1984 cited in Ewles and Simnett (2003:7)) redefined health as:

> *… the extent to which an individual or group is able, on the one hand, to realise aspirations and satisfy needs; and, on the other hand, to change or cope with the environment. Health is, therefore, seen as a resource for everyday life, not the objective of living; it is a positive concept emphasising social and personal resources as well as physical capacities.*

This holistic definition of health is reflected in the book *Meeting the Health Needs of People Who Have a Learning Disability* (Thompson and Pickering 2001) which contains chapters on self-concept, meaningful occupation and life transitions. It can be argued that unless we feel good about ourselves and have meaning in our lives, such as going to work, raising a family, learning new skills, visiting friends, helping others or pursuing hobbies and interests, we

cannot be fully healthy. Having a sense of control over our lives and being connected to the people around us are also important for health and well-being (Naidoo and Wills 2008; Wiles 2008).

There are many influences on all aspects of our health. Dahlgren and Whitehead (1995) depict these as layers piled on top of each other. At the bottom of the pile are biological factors over which we have limited, or no, control. These include our sex and age, and the genes we inherit from our parents. Many diseases become more common as we grow older (e.g. cancer and cardiovascular disease), some diseases are specific to men or women (e.g. prostate and ovarian cancer), while others are genetic or congenital in origin (e.g. cystic fibrosis and congenital heart disease). When the NHS was established in 1948 it concentrated on this biological layer. It was hoped that improvements in health would eliminate health inequalities but although the overall health of the population has gradually improved (mainly through improved living conditions) the gap between the social classes has widened (Asthana and Halliday 2006; Wiles 2008).

The second layer focusses on our personal behaviour. This includes whether or not we smoke cigarettes or eat too much, the amount of exercise we take and how well we attend to our health needs in the broadest sense. Most policy initiatives from government have focussed on this layer, where attempts have been made to change peoples' behaviour in order to improve their health, for example anti-smoking campaigns (Jones 2000b; DH 2004; Wiles 2008). This emphasis on personal behaviour has, however, been criticised. Asthana and Halliday state that '…the government's strategy suggests an implicit assumption that health inequalities can be reduced without changing overall levels of inequality' (2006: 98). There is also a denial of the ways in which the social setting affects our behaviour and reduces our control.

The next layer concerns social and community influences. The people around us, including family members, neighbours, colleagues and friends, can influence our health by giving meaning to our lives and providing assistance and support in times of illness, difficulty and stress. Conversely, these people can have a detrimental effect on our health by neglect, abuse or failing to take account of our needs. Organisations such as the church and self-help groups may also be important (Asthana and Halliday 2006). Berkman and Melchior (2004) point out that social networks provide opportunities for support, access, social engagement and economic advancement, allowing individuals to participate in work, community and family life. Social networks can, however, also lead to discrimination, hostility and exclusion.

Living and working conditions comprise the next layer of influence. It is well known, for instance, that the type of house in which we live and our environment at work can affect our health. Work pressure or noisy neighbours may cause depression and anxiety that can lead to physical ill health (Leon and Walt 2001), and physical hazards, such as dampness, poor architectural design and dangerous work practices, can cause disease and injury (Siegnal and Theorel 2006). Living in deprived neighbourhoods also increases the risk of ill health and mortality, regardless of the individual's personal situation (Steptoe 2006; Wiles 2008). Much of the legislation passed by the Victorians improved people's health by tackling problems at this level (Brunton 2004). Various factory, housing and sanitation Acts, for example, reduced the incidence of serious diseases, such as tuberculosis (TB) and typhoid, as well as improving the quality of people's lives generally. Le Fanu (1999) claims that there was a 92% decline in TB before the introduction of curative drugs. Similar evidence has been put forward by McKeown (1984) who notes that many life threatening and disabling diseases, such as poliomyelitis and diphtheria, had radically declined before the introduction of inoculation. Naidoo and Wills (2008) conclude that medicine has had only a marginal influence on health and mortality rates, although Hubley and Copeman (2008) believe that the underutilisation of health services, especially preventative services, is a contributing factor to the ill health of the poorer sectors of society.

The outermost layer affecting our health concerns general socioeconomic, cultural and environmental conditions. This includes the economic state of the country, the level of employment, the tax system, the degree of environmental pollution and our attitudes, for example towards women, old people, ethnic minorities and disabled people. Increasingly, these factors have taken on an international dimension as globalisation accelerates (Pryke 2009). It is at this level that government can be particularly influential by implementing policy and passing legislation to bring about wide social change, for example seat belt legislation (Figure 10.3), banning smoking in public places and equality legislation, such as

Figure 10.3 Seat belt legislation – influence of government policy.

the Sex Discrimination Act (1975), the Disability Discrimination Act (1995) and the Human Rights Act (1998).

It is clear that these levels all interact and influence each other. If the economic state of the country is favourable, for example, people are likely to have more disposable income which may improve their health by allowing them to buy good quality food and housing of a better standard, engage in leisure pursuits, give their children more opportunities and enjoy relaxing holidays to reduce stress. Similarly, if a person is attempting to give up drugs, success is more likely if community support is strong and if government is willing to act by establishing and financing supportive policies.

Despite the various influences on our health, the evidence suggests overwhelmingly that broad social factors concerning housing, income, educational level, employment and social integration are more important than our individual behaviour or medical practice and advances (Ewles and Simmett 2003). People of the lowest socioeconomic status are at far higher risk, not only of physical illness and early death, but also of accidents, premature births, mental illness and suicide. Smith and Goldblatt (2004) report that of the 66 major causes of death, 62 are more prevalent in the lowest two social classes. It is also the case that men in professional occupations live, on average, seven years longer than men in manual occupations and that the children of manual workers are twice as likely to die before the age of 15 years than the children of professional workers (Naidoo and Wills 2008). In addition, Hargreaves (2007) reports that in 2002–4 infant mortality was 19% higher among manual workers than professional workers, and that the gap had steadily widened. Wiles states that '…across the lifespan there are inequalities in people's health that follow from their economic position. Poor people are more likely to be in poor health and to die at an earlier age' (2008: 52).

It is important to realise, however, that the health status of a country does not equate to its wealth, but rather to how fairly the wealth is distributed (Eberstadt and Satel 2004). Asthana and Halliday (2006) note that longevity rises in societies which are more equal and socially cohesive, especially when infectious diseases have been controlled. They contend that psychosocial stress is related to feelings of relative disadvantage and subordinate status which, in turn, can lead to physical and mental ill health.

It can be disconcerting for healthcare professionals to realise that there is no obvious correlation between healthcare and health status in any population; indeed, the health service has sometimes been referred to as an 'ill health' service as it tends to respond when the damage has been done. This is not to imply, however, that inequalities in health and healthcare facilities should be tolerated. Healthcare should be distributed fairly and in accordance with need. There is evidence, for example, that the uptake of preventative services, such as birth control and screening, is low among poor people (Hubley and Copeman 2008). This is because of a range of factors that were summed up by Tudor Hart (1971) in his notion of the 'inverse care' law.

The inverse care law

The inverse care law states that those who are most at risk of acquiring illness and disease are least likely to receive medical and social services. This is because of a wide variety of factors relating to social inequalities and the availability of services.

People with low incomes find it harder to access healthcare services because of social isolation and lack of facilities, such as a car. It is also the case that the areas in which they live tend to have poor facilities and that health professionals tend to give them less time and attention than people who are perceived to be culturally similar to themselves (Naidoo and Wills 2008).

There are still many people in the UK who do not fully benefit from the facilities of the NHS. People from ethnic minorities are not well served (Atkin et al 2004) nor are people with learning difficulties (Mencap 2004; DH 2009). As noted in the section on culture, this is a result of a variety of factors, including poor and inadequate communication, racism, disablism and lack of cultural sensitivity.

Different models

ABC Definition

Disability can be defined from an individual model or a social model. The individual model is dominant and assumes that the difficulties faced by disabled people are a direct result of their individual impairments or lack or loss of functioning. The social model of disability recognises the social origin of disability in a society geared by, and for, non-disabled people. The disadvantages and restrictions, often referred to as barriers, permeate every aspect of the physical and social environment. Disability can, therefore, be defined as a form of social oppression.

In this section of the chapter we will examine two central models of disability – the individual model and the social model – to illustrate the ways in which underlying ideas and concepts can shape physiotherapy practice and wider medical and social policy. A model can be defined as a conceptual framework for understanding causal relationships. It usually lies within the framework of a broader

theory (Brown 2009). Within every society there are competing models of disability, with some being more dominant than others at different times (Oliver 2004; Wilder 2006). In earlier centuries, for example, models of disability were based upon religion (Stiker 1997; Whalley Hammell 2006). Although often in conflict, models of disability may gradually influence and modify each other. The models put forward by powerful groups within society, such as the medical profession, tend to dominate the models of less powerful groups, such as disabled people themselves (Russell 1998; French and Swain 2008).

It is essential to explore these models of disability, for attitudes and behaviour towards disabled people, policy, professional practice, and the running of institutions, including hospitals and rehabilitation centres, are based, at least in part, upon them. As Oliver (1993: 61) states:

> *The 'lack of fit' between able-bodied and disabled people's definitions is more than just a semantic quibble for it has important implications both for the provision of services and the ability to control one's life.*

Even the ways in which single words are defined can shape both policy and practice. The word 'independence' is an example.

ABC Definition

The predominant meaning of independence is the ability to do things for oneself. This definition has, however, been challenged by disabled people who view independence in terms of self-determination, control, and managing and organising any assistance that is required. Some cultures have a collectivist orientation and do not value independence as much as others. In a very real sense we are all dependent on each other for our survival so nobody is independent.

Health professionals tend to define independence as 'doing things for yourself', whereas disabled people define it as having control of your life. Ryan and Holman state that '…independence is not necessarily about what you can do for yourself, but rather about what others can do for you, in ways that you want it done' (1998: 19).

The individual model of disability

The most widespread view of disability at the present time, at least in the Western world, is based upon the assumption that the difficulties disabled people experience are a direct result of their individual physical, sensory or intellectual impairments (French 2004b; Oliver and Sapey; 2006; Whalley Hammell 2006). Thus, the blind person who falls down a hole in the pavement does so because he or she cannot see it, and the person with a motor impairment fails to get into the building because of his or her inability to walk. Problems are thus viewed as residing *within* the individual. The individual model of disability is deeply ingrained and 'taken as given' in the medical, psychological and sociological literature. Even in the literature on the sociology of health and illness, disability as a social problem is rarely acknowledged (Barnes and Mercer 1996; Swain et al. 2003; Thomas 2007).

The medical model can be regarded as a subcategory of the overarching individual model of disability where disability is conceived as part of the disease process, abnormality and individual tragedy – something that happens to unfortunate individuals on a more or less random basis. In turn, treatment is based upon the idea that the problem resides within the individual and must be overcome by the individual's own efforts. Disabled people have, for example, been critical of the countless hours they have spent attempting to learn to walk or talk at the expense of their education and leisure, and the way their lives have been dominated by medicine. especially when they were young (Sutherland 1981; Oliver 1996; Swain and French 2008). Mason and Rieser (1992: 82) state:

> *For young people the disadvantages of medical treatment need to be weighted against the possible advantages. Children are not usually asked if they want speech therapy, physiotherapy, orthopaedic surgery, hospitalisation, drugs or cumbersome and ugly 'aids and appliances'. We are not asked whether we want to be put on daily regimes or programmes which use hours of precious play-time. All these things are just imposed on us with the assumption that we share our parents' or therapists' desire for us to be more 'normal' at all costs. We are not even consulted as adults as to whether we think those things had been necessary or useful.*

None of these arguments imply that considering the medical or individual needs of disabled individuals is wrong; the argument is that the individual model has tended to view disability *only* in those terms, focussing almost exclusively on attempts to modify people's impairments and return them or approximate them to 'normal'. The effect of the physical, attitudinal and social environment on disabled people has been largely ignored or regarded as relatively fixed, which has maintained the status quo and kept disabled people in their disadvantaged state within society (Oliver and Sapey 2006). Thus, the onus is on disabled people to adapt to a disabling environment (Swain et al. 2004). This is something that disabled people are increasingly joining forces to challenge. As Oliver (1996: 44) states:

The disability movement throughout the world is rejecting approaches based upon the restoration of normality and insisting on approaches based upon the celebration of difference.

ABC Definition

Normality is a dominant, shared expectation of behaviour and personal characteristics that defines what is considered culturally desirable and appropriate.

Individualistic definitions of disability certainly have the potential to do serious harm. The medicalisation of learning disability, whereby people were institutionalised and abused, is one example (Ryan and Thomas 1987; Potts and Fido 1991; Atkinson et al. 2000; Goble 2008). Other examples are the practice of oralism, where deaf children were prevented from using sign language and punished for using it (Humphries and Gordon 1992; Dimmock 1993; Corker 1996) and 'sight-saving' schools where visually impaired children were prevented from using their sight and, in consequence, were denied a full education (French 2005). All of these policies and practices were rooted in an individual model of disability.

The social model of disability

The social model of disability is often referred to as the 'barriers approach' where disability is viewed not in terms of the individual's impairment, but in terms of environmental, structural and attitudinal barriers that impinge upon the lives of disabled people and which have the potential to impede their inclusion and progress in many areas of life, including employment, education and leisure, unless they are minimised or removed (Oliver 1996). The social model of disability has arisen from the thinking, writings and growing cultural identity of disabled people themselves (Swain et al. 2004).

The following definition of impairment and disability is that of the Union of the Physically Impaired Against Segregation (UPIAS), which was an early radical group of the Disabled People's Movement. Its major importance is that it breaks the link between impairment and disability (UPIAS 1976: 14):

- *impairment*: lacking part or all of a limb, or having a defective limb, organ or mechanism of the body.
- *disability*: the disadvantage or restriction of activity caused by a contemporary social organisation which takes no or little account of people who have physical impairments and thus excludes them from participation in the mainstream of social activities.

Physical disability is therefore a particular form of social oppression.

The word 'physical' is now frequently removed from this definition so as to include people with learning difficulties and users of the mental health system (French and Swain 2008). These, and similar, definitions break the connection between impairment and disability, which are viewed as separate entities with no causal link. This is similar to the distinction made between sex (a biological entity) and gender (a social entity) in the women's movement. In recent years, however, it has been recognised that the body is more than a biological entity. Just as height, weight, age and physique have social and cultural dimensions and consequences, so too does impairment (Hughes 2004).

The WHO's *International Classification of Impairment, Disability and Handicap* (ICIDH) (1980) and the revised version (ICIDH-2) (2000) have been rejected by the Disabled People's Movement because, despite taking social and environmental factors into account, the meaning of disability is still underpinned by the medical model and the causal link between impairment and disability remains intact (Hurst 2000; Pfeiffer 2000).

Disability is viewed within the social model in terms of barriers (French 2004b). There are three types of barriers, which all interact:

- *Structural barriers*: these refer to the underlying norms, mores and ideologies of organisations and institutions which are based on judgements of 'normality' and which are sustained by hierarchies of power.
- *Environmental barriers*: these refer to physical barriers within the environment, for example steps, holes in the pavement and lack of resources for disabled people, for example lack of Braille and lack of sign language interpreters. It also refers to the ways things are done, which may exclude disabled people, e.g. the way meetings are conducted and the time allowed for tasks.

Figure 10.4 Whose problem?

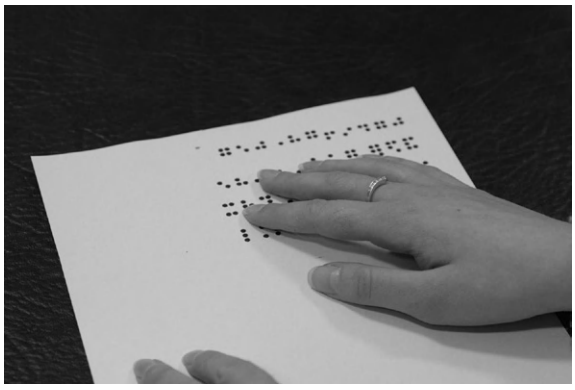


Figure 10.5 Reading Braille.

Figure 10.6 Out of order.

- *Attitudinal barriers*: these refer to the adverse attitudes and behaviour of people towards disabled people.

It can be seen that the social model of disability locates disability not within the individual disabled person, but within society. Thus, the person who uses a wheelchair is not disabled by paralysis but by building design, lack of lifts, rigid work practices, and the attitudes and behaviour of others (Figure 10.4). Similarly, the visually impaired person is not disabled by lack of sight, but by lack of Braille and large print, cluttered pavements and stereotypical ideas about blindness (Figure 10.5). Finkelstein (1981, 1998) has argued that non-disabled people would be equally disabled if the environment were not designed with their needs in mind, for example if the height of doorways only accommodated wheelchair users (Figure 10.6). Human beings fashion the world to suit their own capabilities and limitations, and disabled people are wanting nothing more than that.

CHANGING RELATIONSHIPS

Partnership and user involvement

ABC Definition

Looked at broadly, partnership refers to organisations or individuals working together or acting jointly.

In recent policy developments partnership has been a dominant concept signifying the attainment of greater equality in professional–client relations generally. At a policy level, partnership and collaborative working is considered to be a 'good' thing. Governments of all persuasions have placed emphasis on the need for different agencies to work together to provide more 'seamless' and 'joined-up' service provision by moving towards more integrated health and social care provision (DH 2000a).

Partnership-associated terms

- Inter-agency collaboration.
- Joint working.
- Multi-professional practice.
- Service user involvement.
- Client-centred practice.

Partnership in terms of policy, practice and provision has become a buzz word, widely accepted as an imperative in the development of services for disabled people (and other service users). Defining the concept of partnership, however, is difficult because partnership means different things to different groups of people. It is associated with numerous other terms such as 'participation' and 'empowerment', and also encompasses different relationships.

1. Partnership refers to the relationship between professionals and professional organisations. Associated terms include 'inter-agency collaboration', 'joint working' and 'multi-professional practice'.
2. Partnership refers to the relationship between service users and the service system. A key term here is 'service user involvement'. This is, again, mandated within policy. For instance, one of the Department of Health's (DH) six medium-term priorities was that health authorities should (DH 1995: 9):

… give greater voice and emphasis to users of NHS services and their carers in their own care, the development and definition of standards set

for NHS services locally and the development of NHS policy both locally and nationally.

3. Though much less recognised, the term 'partnership' can be applied to relationships between disabled people and other service users, to include partnerships within and between organisations of disabled people. Barnes and Mercer (2006: 91–92) state:

   The gap between disabled people's expectations and their actual involvement in the statutory and voluntary sectors has reinforced claims from disabled people's organisations that the move to a more equal and democratic society demands a bottom-up approach to politics and policy-making. Its potential is realised by the emergence of active user-led organisations.

4. The term 'partnership' also refers to the more immediate relationship between a professional and a service user. Again, this can encompass different terms such as 'client-centred practice'. Sumsion (2005: 100) defines this as follows:

   In the UK client centred occupational therapy is a partnership between the client and the therapist that empowers the client to engage in functional performance and fulfil their occupational roles in a variety of environments. The clients participate actively in negotiating goals that are given priority and are at the centre of assessment, intervention and evaluation.

In terms of more detailed breakdowns of the meaning of the term partnership, Reynolds (2004) suggests that a partnership approach to healthcare makes the following assumptions:

- both professionals and service users are regarded as bringing strengths or resources to the therapeutic process and the therapeutic relationship;
- both professionals and service users are part of the team that will openly share information and make decisions about the ways forward in therapy;
- the relationship is respectful and affirmative, rather than shaped by dependency, submissiveness or power struggles;
- the relationship is based on adult strategies of communication, rather than infantalising, manipulation or stereotyping, with both partners having a respected voice in the interaction;
- the service user is motivated to share responsibility with the therapist for the therapy process and outcome;
- the purpose of the partnership is to promote the service user's self-actualisation, development and quality of life rather than pressurising specifically for better compliance with treatment.

Partnership in therapy practice could include the service users' voices in the following decision making processes (Thompson 1998: 213):

- identifying problems to be tackled, issues to be addressed, goals to be achieved;
- deciding what steps are to be taken and who needs to do what;
- undertaking the necessary work through collaboration and consultation;
- reviewing progress and agreeing any changes that need to be made to the agreed course of action;
- bringing the work to a close if and when necessary;
- evaluating the work done, highlighting strengths, weaknesses and lessons to be learned.

ABC Definition

Inclusive communication is the valuing and celebration of differences and empowerment through the power of communication. The following factors are central:

- participation;
- accessible communication;
- diversity and flexibility;
- human relations;
- inclusive language.

The term 'inclusion' has been seen by many as a process of social change, rather than a particular state (Ballard 1999), and this can be seen to apply equally to communication and relationships. To develop this notion of an inclusive communication environment, we shall conclude this section by tentatively offering some general principles based on our previous discussion.

Participation

Priority needs to be given to the participation of service users in the planning and evaluation of changing policy, provision and practice in developing inclusive communication. The onus is on service providers to face the challenges of enabling true participation of service users in decision-making processes, recognising that service users wish to participate in different ways. These include the democratic representation of the views of organisations of service users. Participation also includes as wide a consultation process as possible. Service users often continue to be treated as passively dependent on the expertise of others yet control has become increasingly central to social change for service users. As a

disabled man interviewed by French (2004a: 106), with a great deal of experience of therapy treatment, states:

> *Users should have more power. Until you give users real power, real control we'll get nowhere … there's an awful lot of people with a lot of vested interests. The more we shout about rights the more people get afraid. I'd like to see therapy training following the social model rather than the medical model. The only way to do it is to get much more input from disabled people into the training.*

The relationship between disabled people and health professionals has never been an easy one, for it is an unequal relationship with the professional holding most of the power. Traditionally, the professional worker has defined, planned and delivered the services while the disabled person has been a passive recipient with little, if any, opportunity to exercise control (French and Swain 2001). Disabled people's definitions of the problems they face, and the appropriate solutions to such problems, have generally been given insufficient weight, thereby seriously hampering the rehabilitation process – if there is no consensus, little real progress can be made.

While physiotherapists may feel constrained by their working role, there usually remains some degree of flexibility in which they can work in partnership with disabled people and other patients and clients. For example, they can share power by sharing 'expert' knowledge and information, and they can encourage patients and clients to participate actively in the writing of reports and case files so that their voices are heard and their viewpoints represented in official documents.

Awareness of disability by all those who work in physiotherapy departments can be encouraged and developed through disability equality training run by skilled disabled people. Disability equality training is (French 1996: 121):

> *… primarily about changing the meaning of disability from individual tragedy to social oppression; it emphasises the politics of disability, the social and physical barriers that disabled people face, and the links with other oppressed groups.*

Another way in which physiotherapists can work in partnership is by recognising and acknowledging the disabled person's expertise in relation to the meaning and experience of being disabled and their particular impairment. This means encouraging disabled people to exercise choice of services appropriate to their desired lifestyles. Physiotherapists can work in partnership with disabled people by regarding themselves as a resource (expertise, information, advocacy) so that disabled people can work towards achieving their own goals. This would include clarifying the goals to which the disabled person aspires, identifying the barriers that may prevent the realisation of those goals and working towards removing the barriers.

From the perspective of the social model of disability, professional power can be used to highlight the shortfall in resources for disabled people, to ensure that the voices of disabled people are heard and responded to, and to encourage and support disabled people to assert themselves so that their expertise about disability is at the centre of the development of services and support. It is important that physiotherapists, and all other staff in physiotherapy departments, make a conscious decision to heighten their awareness of disability as an area of inquiry.

Accessible communication

The issues around language and ethnicity are complex, but there are many examples of good practice. The Sandwell Integrated Language and Communication Service (SILCS) in the West Midlands, for instance, involves a range of local health organisations – health authorities, NHS Direct, primary care groups, local authorities and voluntary agencies – working together to provide a pooled resource for spoken, written and telephone translation and interpreting, as well as sign language interpreters (Douglas et al. 2004). Accessibility of communication, however, needs management beyond such a resource, including training for staff on using interpreters. The provision of written information in a range of languages must ensure that translations meet the information needs of black and ethnic minority communities and are culturally relevant. There are, of course, many social factors within the diversity of the needs of people from black and ethnic minority communities. As Dominelli (1997: 107) argues, for instance, '…translation services should be publicly funded and provide interpreters matched to clients' ethnic grouping, language, religion, class and gender'.

Much is known about the accessibility of information based on the views expressed by disabled people. Clark (2002) offers wide-ranging recommendations which cover such things as alternative formats, for example, large print, Braille, accessible websites, videotape and British Sign Language, and suggestions for plain written language, layout, typeface and font size. For some people, particularly those with communication disabilities, the issue of time can be crucial to an inclusive communication environment. For people with communication disabilities a slower tempo can be the only accessible pace to ensure understanding. A participant within the research by Knight et al. (2002: 17) explains:

> *I would rather repeat myself ten times than have someone finish a sentence for me. This is*

why I won't use a communication aid. I prefer to speak for myself and I would rather repeat myself several times than have someone say they understood me when they did not.

Along similar lines, Pound and Hewitt (2004) emphasise that access in meetings with people with communication impairments requires attention to their length and timing.

Diversity and flexibility

Responding to diversity and being flexible are complex issues as Douglas et al. (2004: 74) point out:

Interpreting is extremely complex in that interpreters must ensure that the patient or client easily understands the language they use. Again other factors, such as class, region, religion and geography, may impinge on the process of interpreting and communication – such that just speaking the same language may not necessarily mean the same understanding will follow.

A disabled client interviewed by French (2004a: 103) provides the foundation for this by questioning the focus on 'normality', rather than being flexible and taking the client's perspective into account:

What concerns me most of all is this focus on trying to make me 'normal'. I get that from all the therapists. I get a lot of referrals of 'this may help' and 'that may help'. They had a massive case conference before the adaptations – it was a case of 'how normal can we make her first? Are the adaptations necessary?'

The lists of recommendations for communication access, as produced by Clark (2002) and others, clearly challenge the imperatives of 'normality' and emphasise the diversity of communication styles and formats (Figure 10.7). Nevertheless, there are diverse needs even within specific groups of people with impairments, which, again, puts the emphasis on listening to individual people and giving them control. Sally French, as a person with a visual impairment, has found, for example, that she is often presented with large print even though it is the depth, font and colour contrast that are more important to her.

Human relations

Communication is constructed and embedded in relationships between people. The notion of personal relationships can be seen as irrevocably intertwined with communication. Communication is a means of expressing a relationship; it constitutes the initiation, maintenance and ending of a relationship; and it is the medium and substance through which the relationship is defined and given meaning. A disabled client offered advice to therapists on the basis of her experience (French 2004a: 103):

Forget you're a therapist – just be yourself. I don't mean forget all your training – but be yourself. Don't be afraid of showing the real you because that's what makes people respond, when they're ill they respond more easily if the therapist is being real.

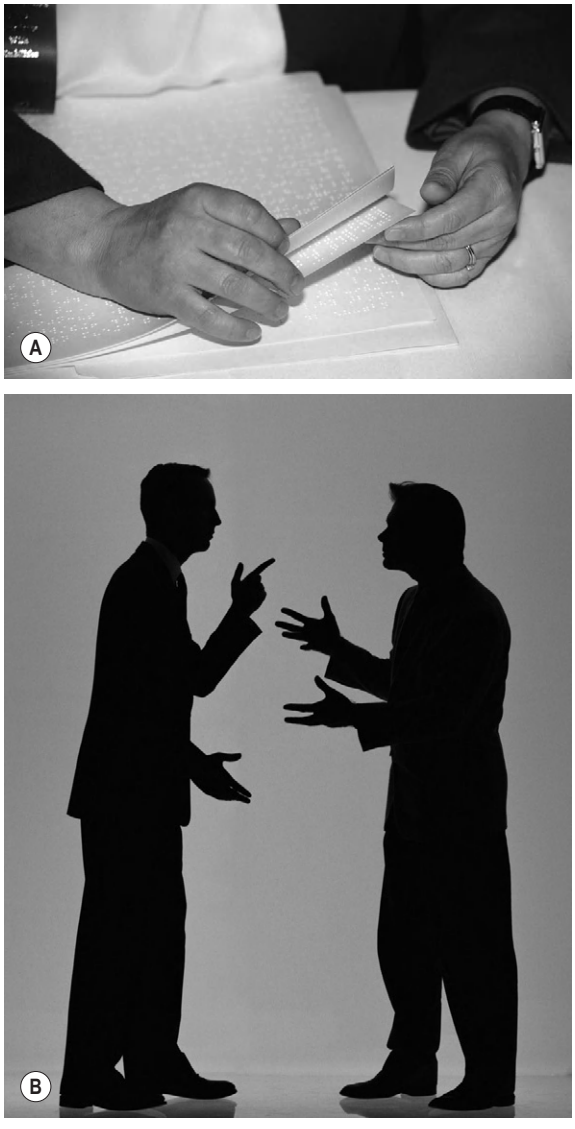


Figure 10.7 There are many ways to communicate.

Use of inclusive language

Inclusive language reflects the idea that language controls or constructs thinking. Sexism, ageism, homophobia, racism and disablism are framed within the very language we use. This has been characterised and degraded by some people as 'political correctness' (PC), often with reference to examples seen as trivial or fatuous (e.g. being criticised for offering black or white coffee). Use of language, however, is not simply about the legitimacy of words or phrases – what we are allowed to say or not say. As Thompson (1998) explains, language is a powerful vehicle within interactions between health and social care professionals and clients. He identifies a number of key issues:

- *jargon:* the use of specialised language, creating barriers and mystification, and reinforcing power differences;
- *stereotypes*: terms used to refer to people that reinforce presumptions, e.g. disabled people as 'sufferers';
- *stigma*: terms that are derogatory and insulting, e.g. 'mentally handicapped';
- *exclusion*: terms that exclude, overlook or marginalise certain groups, e.g. the term 'Christian name';
- *depersonalisation*: terms that are reductionist and dehumanising, e,g, 'the elderly', 'the disabled' and even 'CPs' (to denote people with cerebral palsy).

In this light, questions of the use of language go well beyond listing acceptable and unacceptable words to examining ways of thinking that rationalise, legitimise and underline unequal therapist–client power relations.

ABC Definition

Service user involvement is a general term that covers service user consultation and collaboration in service policy making, planning, delivery and evaluation. It can involve:

- giving users information about what others have decided;
- consulting users;
- making joint decisions with users;
- users doing things for themselves and taking control.

In terms of partnership encompassed within the notion of service user involvement, the literature suggests that methods of involving users of services can take many forms. For instance, Brown (2000) lists the following methods with particular reference to residential care for disabled people:

- residents' committees;
- user panels;
- customer surveys;
- suggestion boxes;
- involvement in management committees;
- involvement in forums and working parties;
- focus groups;
- public meetings.

It is crucial not to rely heavily on any one method as none is perfect and a variety are needed to reach all disabled people.

The following principles were derived from the evaluation of user involvement in a disabled people's organisation (Swain et al. 2005). They do not rely on any particular method, but focus on general principles to provide both the foundations for development and a framework for monitoring change through user involvement.

- There is a clear and absolute requirement that the effective development of user involvement must be generated by and controlled by service users themselves.
- The development of user involvement needs to be seen as embedded in all decision-making within the organisation, including financial and management decision-making at all levels (local, regional and national).
- User involvement is embedded in service users' lives. It is part of defining the quality of life for service users. Quality of life is determined within the say that people have over their lives, from the day-to-day decisions over basic needs (sleep, eating, toilet, etc.) to the control over their own finances and over the support they receive.
- There is no existing model of user involvement that has been developed that can, or should be, adopted for general usage. Any attempt to do so is more likely to be retrograde than enhance user involvement.
- There is no body of concern that can be seen as 'user involvement' that is separate or isolated from all decision-making structures and processes within an organisation – finance, management, etc.
- User involvement in policy-making is crucial. Firstly, service users should be involved in the writing of policy, rather than being simply consulted about drafts of policy statements. Secondly, the least restrictive possible policies and practices arising from legislation need to be implemented. Thirdly, in adopting the least restrictive response policy-making should, as far as possible, be made at a local level, with full user involvement.
- Approaches to user involvement also need to be open, flexible and individual (or client-centred).
- Effective communication is fundamental to user involvement. This includes increased support, communication workers and use of communication equipment at an individual level. It also includes a creative and flexible approach to group meetings, including video conferencing, to open opportunities.

Looking towards inclusion, Bewley and Glendinning (1994) note a heavy reliance on formal meetings in service user involvement and point out that in order to reach black disabled people, people living in rural areas and other marginalised groups, such as travellers and people with learning difficulties, a community development approach, working with local networks, needs to be adopted on a sustained basis.

Turning to the standpoint of disabled people, Beresford et al. (1997) note that organisations of disabled people have influenced mainstream services by collaborating on projects and by training professionals in disability issues. Some disabled people feel that this collaboration is strategically important to ensure their involvement in community care, though others are more cautious fearing that professionals will become too dominant. In their research into self-organised user groups of social and healthcare services, Barnes et al. (1999: 24) found that the representation of disabled people within key decision-making forums was seen as both an end in itself and a means for achieving other objectives. For instance, one participant stated:

> *I think the patients' council works well because it does work on the principle that it's not one or two of our service users … sitting on the edge of a meeting or whatever, it is managers, senior managers coming to meetings of service users to be accountable and really they have to account on the spot.*

A very important component of user involvement has been the development of services that are run and controlled by disabled people for disabled people. Drake (1996: 190) states that:

> *… disabled people have been increasingly active not only in policy debates but also in producing self-governed groups and projects such as CILs [Centres for Independent (or Integrated) Living] which have proved a cogent and powerful alternative to the traditional gamut of projects like day centres and social clubs.*

Centres for Independent (or Integrated) Living (CILs) provide a range of services that are designed to meet the needs of disabled people as they define them. These include the provision of information, advice and associated support services; training in the employment of personal assistants; repair services; peer counselling; independent advocacy; and disability equality training. Mercer (2004: 179) believes that:

> *There is a broad consensus that user-led organisations offer a distinctive approach to service provision. This encompasses adherence to a social model, democratic accountability, promoting independent/integrated living through wider user choice and control and including all disabled people.*

From her review of the literature on user involvement Carr (2004: 12) states:

> *Evaluation of user-led organisations for disabled people has shown that there was overwhelming agreement that user-led organisations were far more responsive to disabled people's support needs both in terms of what was on offer and how it was delivered, with peer support a major consideration.*

It is not surprising, then, that Thompson (2001) argues that CILs can act as a model for service provision. If we return to the four relationships or types of partnerships outlined above, they can be viewed as inter-related. The partnership between disabled people themselves is a key to shifting the power relations between service providers and service users.

Disability studies (the social, political and cultural analysis of disability) have barely touched the curriculum of health professionals, including physiotherapists, but with the growing impact of the social model of disability and changes such as the implementation of the Disability Discrimination Act (1995), physiotherapists will need to extend their viewpoint beyond the individual model of disability and to work with disabled people as equal partners. This may mean moving away from the traditional approach of 'helping' and 'treating' towards a much broader brief that involves joining forces with disabled people and using their professional power, in collaboration and partnership with disabled people, to dismantle every aspect of disablism and to further the fight for full citizenship for all disabled people.

Health promotion

The profession of physiotherapy, along with other healthcare professions, has taken a largely biomedical approach to patient and client care (Hubley and Copeman 2008). This is strongly reflected in physiotherapy literature and research, and the undergraduate curriculum (French and Swain 2005). Over the years, however, education in the social sciences has been included and more physiotherapists now work in the community rather than hospitals, reflecting changes in NHS structure and policy (DH 2000b). Physiotherapy has, until recent times, been under

ABC Definition

Health promotion is a broad range of activities that aim to improve health. These include health education, such as teaching correct lifting techniques; environmental measures, such as the control of pollution; preventative medicine, such as cervical screening; fiscal measures, such as financial benefits; and legal measures, such as seat belt legislation.

the control of doctors, and physiotherapy practice, with its biomedical orientation, has reflected this control. Naidoo and Wills (2008) point out that medicine has focussed on the individual and that this has been perpetuated by the greater power of medical professionals when compared with those who work in health promotion. They also note that few resources are allocated to health promoting practices. A consideration of social, political, cultural and economic factors that may impact on people's health and well-being has been slow to develop in physiotherapy education, and the social sciences remain marginalised and focussed largely on micro issues, such as interpersonal communication.

This section of the chapter aims to uncover the meaning of health promotion in physiotherapy practice. Health promotion is a complex and contested concept which is defined in many ways (Scriven 2005). This is not surprising given that, as noted above, the notion of health itself has a wide range of meanings from the physiological to the philosophical and spiritual. Tones and Tilford (2001: 2) state that health promotion:

> *… means different things to different people. Since health itself is a multi-dimensional notion – open to multiple interpretations – it is unsurprising that the definition of health promotion is itself problematic.*

The development of health promotion stretches back to the nineteenth century. The 1848 Public Health Act for England and Wales, for instance, required local authorities to provide sewage disposal systems and a clean water supply. Ewles and Simnett (2003) trace the development of health promotion throughout the twentieth century. In the first half of the century the main concern was 'public health' which saw, for example, the clearance of the slums, the building of new towns and legislation aimed at reducing environmental pollution. Between 1950 and 1970 the emphasis changed to health education where people were given information designed to persuade or coerce them to change their 'unhealthy' behaviours as a way of preventing disease. School children, for example, were provided with information about a balanced diet in order to prevent diseases such as diabetes and coronary heart disease in later life. Women were given pre- and postnatal advice and instruction by physiotherapists, including pelvic floor exercises, in order to reduce complications such as stress incontinence after birth. People were urged to take part in screening programmes and to have their children inoculated against diseases such as poliomyelitis and diphtheria which can lead to impairment or death.

ABC Definition

Victim blaming is a tendency to blame the person who is experiencing ill health or other difficulties. A heavy smoker, for example, may be blamed for contracting lung cancer. Victim blaming denies the influence of wide social, economic, cultural and political factors on people's behaviour, over which they may have little control. Behaviour cannot be separated from the social context in which it takes place.

Health education, however, came under criticism on the grounds that it leads to a 'victim blaming' approach (Naidoo and Wills 2008).

As noted in the discussion on health inequalities, people in deprived circumstances have the fewest choices, including those concerning their health. It may, for example, be more difficult or impossible for them to find the time to exercise regularly, to attend health-screening and health education programmes, or to afford a balanced diet. Furthermore, environmental factors, such as noise, pollution and poor housing impact on their physical and mental health to a greater extent than their more affluent peers. Tones and Tilford (2001: 7) state:

> *… a focus on individual sins of omission and commission characterised the approach of preventive medicine and health education for the better part of the 20th century. Increasingly, however, explanations of the determinants of health and disease is shifting away from this narrow orientation on individual behaviours and beginning to emphasise the importance of environmental factors.*

Behaviours labelled unhealthy may be rational strategies to reduce stress caused by wider social and economic factors. Thus, a single mother who lives in a high-rise flat in a run down part of town may smoke or over-eat in order to cope with her difficult situation. Trying to prevent her from smoking or over-eating may be both

counter-productive and damaging because, for her, the emotional gains outweigh the physical costs. Hubley and Copeman (2008) speak of a 'culture of poverty' where poor and marginalised people develop coping mechanisms for their own psychological and emotional survival which may be dysfunctional with regard to their physical health.

As Nadoo and Wills (2008: 151) state:

> *There is a limit to a person's capacity to adapt. For example those living on low incomes will be stretched by coping with poverty and its uncertainties. Having to make changes to their health behaviour may be too much to expect for people whose lives are already problematic.*

Focussing on individual behaviour also has the effect of deflecting interest from wider social, economic and political issues. As Ewles and Simnett (2003: 41) state:

> *We cannot assume that individual behaviour is the primary cause of ill health … There is a danger that focussing on the individual detracts attention from the more significant (and, of course, politically sensitive) determinants of health, such as the social and economic factors of racism, relative deprivation, poverty, housing and unemployment.*

Focussing on people's responsibility for their own health can easily serve to legitimate inactivity from both professionals and government.

From the 1980s what is sometimes known as the 'new public health' emerged. This focusses on both public health and health education and aims to involve people and their communities (Nutbeam and Harris 1999). It encompasses traditional healthcare, personal social services, preventative healthcare, community-based work (e.g. the establishment of self-help groups, pressure groups, community facilities and activities), organisational development (e.g. equal opportunity policies and measures to reduce stress), public policies (e.g. education, housing and transport), environmental policies (e.g. smoke-free zones and noise control) and legislative change (e.g. the labelling of food and the control of advertising). Broad programmes operate at multiple levels and address a wide range of health related problems that can be present in a population. It requires communication and co-operation among many different agencies, as well as 'joined up' policy (Nadoo and Wills 2008). In addition, increasing globalisation creates new challenges as health promotion moves beyond national boundaries. International bodies such as the WHO and the World Bank, for instance, are at the forefront of many international health projects.

The term 'health promotion' refers to strategies that not only attempt to prevent ill-health and disease in its broadest sense, but also to improve quality of life and well-being. This is in contrast to the biomedical approach where the emphasis is on finding a cure or managing a condition once it has occurred. Most definitions of health promotion emphasise the need to empower people to have more choice and control over those aspects of their lives which affect their health including the communities in which they live. There is a recognition that medicine and professional practice have had little effect on health and that health can be improved more successfully by investing in the physical and social fabric of society and reducing inequalities (Ewles and Simnett 2003). Hubley and Copeman (2008) argue that health services need to be reshaped away from medicalisation and towards the empowerment of patients and clients.

Naidoo and Wills (2008) see health promotion as encompassing disease prevention, health education and health information, public health reform and community development. They view the work of those involved in health promotion as emphasising a focus on health not illness, the empowerment of clients, recognising that health is multi-dimensional and acknowledging the influence of factors external to the individual which impinge upon their health. When attempting to deal with a health issue such as smoking, for example, a wide range of health-promoting activities are necessary, such as health education, developing assertiveness skills, community development, such as organising a 'healthy eating' campaign, and structural adjustments, such as legislation to ban cigarette advertising and smoking in public places. They emphasise that a diverse range of information and theories are needed in the practice of health promotion drawn from epidemiology, demography and the social sciences, as well as information about particular community needs and priorities. Ewles and Simmett (2003) list the following approaches to health promotion which are not, necessarily, mutually exclusive.

- *The medical approach*. This approach values preventative measures (such as screening and inoculation) and patient compliance.
- *The behavioural change approach*. This approach seeks to change people's attitudes and behaviour in ways defined by the professional.
- *The educational approach*. This approach gives people information but their values and choices are respected. The role of the professional is to help people to gain the skills to make well-informed decisions and to offer their help and support.
- *The client-centred approach*. This approach identifies what patients and clients want to know and what actions, if any, they want to take. Self-empowerment is central and the professional acts as a facilitator.

- *The social change approach*. This approach focusses overtly on political issues and on changing society, rather than changing individuals.

It would seem from the literature and from first-hand accounts from physiotherapists (see French and Swain 2005) that physiotherapy practice reflects most strongly the first three approaches listed above. In the *Curriculum Framework for Qualifying Programmes in Physiotherapy* (2002), for instance, the Chartered Society of Physiotherapy define health promotion in terms of providing advice and health education to patients, carers, support workers and other healthcare professionals. Talking of clients with learning difficulties, however, Standing (1999), a physiotherapist, moves towards the client-centred approach. She advocates working in partnership with patients and clients in order to fulfil their unique goals and aspirations and, as a consequence, to improve their health and well-being.

In 1996 the first international conference on health promotion was held. It led to the Ottawa Charter for Health Promotion which proposed the following five areas for action:

- building a public health policy;
- creating supportive environments;
- developing personal skills;
- strengthening communities;
- re-orientating health services.

(Adapted from Naidoo and Wills 2008.)

Physiotherapists (and other health professionals) are rarely involved in the social change approach advocated here. Many sociologists and disabled people have criticised this stance which, they claim, not only maintains the status quo but, through the power of professionals, defines the very meaning of illness, disability and mental distress (Naidoo and Wills 2008). Davis (2004), a disabled activist, for example, is fervent in his criticism of health professionals' lack of interest and involvement in the disability rights agenda. Similar critiques of medical professionals and healthcare services have been voiced over many years by sociologists such as Friedson (1970), Illich (1976) and McKnight (1995), and feminists such as Doyal (1995). Naidoo and Wills urge health professionals to become more involved in political activity to combat the social and economical determinants of ill-health. They state (2008: 110–111) that:

> *Accepting the status quo is not an apolitical position but a deeply political one … part of the task of health promoters is to uncover and hold up to scrutiny their values and beliefs.*

Health promotion is a broad and complex concept which demands of physiotherapists a comprehensive understanding of the meaning of health and well-being within individual lives, within the communities in which people live and within the world. Physiotherapists and other health professionals are playing their part in many aspects of health promotion (see Scriven 2005), but mostly in the areas of preventative medicine and health

Figure 10.8 What does this have to do with health?

education. Central to moving towards more health-promoting practice is shifting the emphasis from medical and behavioural change approaches towards more client-centred and social change approaches. This shift would involve a broadening of the physiotherapist's role and working in partnership with those engaged in health promotion outside 'traditional' health and social services, such as architects, community workers and political campaigners. This would involve many skills, not least those of communicating with a wide range of people from diverse disciplines. As Scriven (2005) points out, this is no easy task within the NHS where resources are restricted and where curing disease takes precedent over promoting health. A holistic approach towards health and well-being challenges a medicalised view of individuals and extends thinking and activity beyond the individual to the broad social, economic, cultural and political context locally, nationally and internationally (Figure 10.8).

CONCLUSION

In this chapter we have examined relationships within physiotherapy practice and have discussed how these relationships might change to encompass current reasoning, knowledge, policy and legislation. As front line workers in the NHS it is vital that physiotherapists understand the meaning of health, illness and disability from the perspective of patients and clients, and that the physiotherapy service is run flexibly and imaginatively so that people who may benefit from it can use it with ease and convenience and in ways that facilitate their aspirations and goals. Improving health, in its broadest sense, is not just about medicine, but necessitates involvement in areas such as housing, employment, education, leisure, outreach services and community regeneration. It also demands working *with* rather than *on* or *for* people.

ACKNOWLEDGEMENTS

With a special thank you to Lauren and Iris.

REFERENCES

Asthana, S., Halliday, J., 2006. What Works in Tackling Health Inequalities? Polity Press, Bristol.

Atkin, K., French, S., Vernon, A., 2004. Health care for people from ethnic minority groups. In: French, S., Sim, J. (Eds.), Physiotherapy: A Psychosocial Approach, third ed. Butterworth-Heinemann, Oxford.

Atkinson, D., McCarthy, M., Walmsley, J., et al. (Eds.), 2000. Good Times, Bad Times: Women with Learning Difficulties Telling their Stories. British Institute of Learning Disability, Kidderminster.

Ballard, K., 1999. Concluding thoughts. In: Ballard, K. (Ed.), Inclusive Education: International Voices on Disability and Justice. Falmer, London.

Banton, M., Hirsch, M., 2000. Double Invisibility: Report on Research into the Needs of Black Disabled People in Coventry. Warwickshire County Council, Warwickshire.

Barnes, C., Mercer, G. (Eds.), 1996. Exploring the Divide: Illness and Disability. The Disability Press, Leeds.

Barnes, C., Mercer, G., 2006. Independent Futures: Creating User-Led Disability Services in a Disabling Society. The Policy Press, Bristol.

Barnes, M., Harrison, S., Mort, M., et al., 1999. Unequal Partners: User Group and Community Care. The Policy Press, Bristol.

Begum, N., 1996. Doctor, doctor… disabled women's experiences of general practitioners. In: Morris, J. (Ed.), Encounters with Strangers: Feminism and Disability. The Women's Press, London.

Beresford, P., Croft, S., Evans, C., et al., 1997. Quality in personal social services: The developing role of user involvement in the UK. In: Evans, A., Haverinen, K., Leichsering, K., et al. (Eds.), Developing Quality in Personal Social Services. Ashgate, Aldershot.

Berkman, L.F., Melchior, M., 2006. The shape of things to come: How social policy impacts social integration and family structure to produce population health. In: Siegrist, J., Marmot, M., (Eds.), Social Inequalities in Health: New Evidence and Policy Implications. Oxford University Press, Oxford.

Bewley, C., Glendinning, C., 1994. Involving Disabled People in Community Care Planning. Joseph Rowntree Foundation, York.

Bignall, T., Butt, J., 2000. Between Ambition and Achievement: Young Black Disabled People's Views and Experiences of Independence and Independent Living. Policy Press, Bristol.

Boazman, S., 1999. Inside aphasia. In: Corker, M., French, S., (Eds.), Disability Discourse. Open University Press, Buckingham.

Brown, H., 2000. Challenges from Service Users. In: Brechin, A., Brown, H., Ely, M.A. (Eds.), Critical Practice in Health and Social Care. Sage, London.

Brown, V., 2009. Rubbish society: affluence, waste and values. In: Taylor, S., Hitchliffe, S., Clark, J., et al. (Eds.), Making Social Lives. The Open University, Milton Keynes.

Brunton, D., 2004. Dealing with disease in populations: public health, 1830–1880. In: Brunton, D. (Ed.), Medicine Transformed: Health, Disease and Society in Europe 1800–1930. Manchester University Press, Manchester.

Butt, J., Box, L., 1997. Supportive Services, Effective Strategies: The Views of Black-Led Organisations and Social Care Agencies on the Future of Social Care for Black Communities. Race Equality Unit, London.

Butt, J., Mirza, K., 1996. Social Care and Black Communities. Race Equality Unit, London.

Carr, S., 2004. Has Service User Involvement Made a Difference to Social Care Services? Social Institute for Excellence, London.

Clark, L., 2002. Liverpool Central Primary Care Trust Accessible Health Information: Project Report, www.leeds.ac.uk/disability-studies.

Corker, M., 1996. Deaf Transitions: Images and Origins of Deaf Families, Deaf Communities and Deaf Identities. Jessica Kingsley, London.

CSP (Chartered Society of Physiotherapy), 2002. Curriculum Framework for Qualifying Programmes in Physiotherapy. CSP, London.

Dahlgren, G., Whitehead, M., 1995. Policies and strategies to promote social equity in health. In: Benzeval, M., Judge, K., Whitehead, M. (Eds.), Tackling Inequalities in Health: An Agenda for Action. Kings Fund, London.

Davis, K., 2004. The crafting of good clients. In: Swain, J., French, S., Barnes, C., and Thomas, C., 2004. Disabling Barriers – Enabling Environments, second ed. Sage, London.

DH (Department of Health), 1995. Priorities and Planning Guidance for the NHS: 1996/97. NHS Executive, London.

DH (Department of Health), 2000a. The Health and Social Care Bill. Her Majesty's Stationery Office, London.

DH (Department of Health), 2000b. The NHS Plan: A Plan for Investment, A Plan for Reform. Her Majesty's Stationery Office, London.

DH (Department of Health), 2004. Choosing Health: Making Healthy Choices Easier. Her Majesty's Stationery Office, London.

DH (Department of Health), 2009. The NHS Constitution: The NHS Belongs to us All. DH, London.

Dimmock, A.F., 1993. Cruel Legacy: An Introduction to the Record of Deaf People in History. Scottish Workshop Publications, Edinburgh.

Dominelli, L., 1997. Anti-Racist Social Work, second ed. Macmillan Press, Houndmills.

Douglas, J., Komaromy, C., Robb, M., 2004. Diversity and Difference in Communication, Unit 6 K205. The Open University, Milton Keynes.

Doyal, L., 1995. What Makes Women Sick: Gender and the Political Economy of Health. Macmillan, Basingstoke.

Drake, R.F., 1996. Understanding Disability Policies. Macmillan, Basingstoke.

Eberstadt, N., Satel, S., 2004. Health and the Income Inequality Hypothesis. The AEI Press, Washington.

Ewles, L., Simnett, I., 2003. Promoting Health: A Practical Guide, fifth ed. Bailliere Tindall, London.

Finkelstein, V., 1981. To deny or not to deny disability. In: Brechin, A., Liddiard, P., Swain, J. (Eds.), Handicap in a Social World. Hodder and Stoughton, Sevenoaks.

Finkelstein, V., 1998. Emancipating disability studies. In: Shakespeare, T. (Ed.), The Disability Reader: Social Science Perspectives. Cassell, London.

Ford, J., 2000. Speak For Yourself. Scope, London.

French, S., 1988. Experiences of disabled health and caring professionals. Sociol Health Illness 10 (2), 170–188.

French, S., 1993. Setting a record straight. In: Swain, J., Finkelstein, V., French, S., et al. (Eds.), Disabling Barriers – Enabling Environments. Sage, London.

French, S., 1996. Simulation exercises in disability awareness training: a critique. In: Hales, G. (Ed.), Beyond Disability: Towards an Enabling Society. Sage, London.

French, S., 2001. Disabled People and Employment: A Study of the Working Lives of Visually Impaired Physiotherapists Ashgate: Aldershot.

French, S., 2004a. Enabling relationships in therapy practice. In: Swain, J., Clark, J., Parry, K., et al. (Eds.), Enabling Relationships in Health and Social Care: A Guide for Therapists. Butterworth-Heinemann, Oxford.

French, S., 2004b. Defining disability: implications for physiotherapy practice. In: French, S., Sim, J. (Eds.), Physiotherapy: A Psychosocial Approach, third ed. Butterworth-Heinemann, Oxford.

French, S., 2005. Don't look! The history of education for partially sighted children. Br Jl Visual Impairment 23 (3), 108–113.

French, S., Swain, J., 2001. The relationship between disabled people and health and welfare professionals. In: Albrecht, G., Seelman, K.D., Bury, M. (Eds.), Handbook of Disability Studies. Sage, London.

French, S., Swain, J., 2005. The culture and context for promoting health through physiotherapy practice. In: Scriven, A. (Ed.), Health Promoting Practice: The Contribution of Nurses and Allied Health Professionals. Palgrave Macmillan, Basingstoke.

French, S., Swain, J., 2008. Understanding Disability: A Guide for Health Professionals. Elsevier, Oxford.

French, S., Gillman, M., Swain, J., 1997. Working with Visually Disabled People: Bridging Theory and Practice. Venture Press, Birmingham.

Friedson, E., 1970. Profession of Medicine: A Study of the Sociology of Applied Knowledge. Harper and Row, New York.

Gillespie-Sells, K., Hill, M., Robbins, B., 1998. She Dances to Different Drums: Research into Disabled Women's Sexuality. King's Fund, London.

Goble, C., 2008. Institutional abuse. In: Swain, J., French, S. (Eds.), Disability On Equal Terms. Sage, London.

Hargreaves, S., 2007. Gaps between UK social groups in infant mortality are widening. BMJ 384 (7589), 335.

House of Commons Health Committee, 2009. Health Inequalities. The Stationery Office, London.

Hill, M., 1991. Race and disability. In: The Open University (Ed.), Disability – Identity, Sexuality and Relationships: Readings, K665Y course. The Open University, Milton Keynes.

Hubley, J., Copeman, J., 2008. Practical Health Promotion. Polity Press, Cambridge.

Hughes, B., 2004. The disappearing body. In: Swain, J., French, S., Barnes, C., et al. (Eds.), Disabling Barriers – Enabling Environments. Sage, London.

Humphries, S., Gordon, P., 1992. Out of Sight: The Experience of Disability 1900–1950. Northcote House, Plymouth.

Hurst, R., 2000. To revise or not to revise. Disability Society 15, 1083–1087.

Illich, I., 1976. Limits to Medicine: Medical Nemesis, the Expropriation of Health. Penguin, Harmondsworth.

Jones, L., 2000a. Promoting health: everybody's business? In: Katz, J., Peberdy, A., Douglas, J. (Eds.), Promoting Health: Knowledge and Practice, second ed. Palgrave, Basingstoke.

Jones, L., 2000b. Behavioural and Environmental Influences on Health. In: Katz, J., Peberdy, A., Douglas, J. (Eds.), Promoting Health: Knowledge and Practice, second ed. Palgrave, Basingstoke.

Jones, L., Atkin, K., Ahmad, W.I.U., 2001. Supporting Asian deaf young people and their families: The role of professionals and services. Disability Society 16 (1), 51–70.

Knight, B., Sked, A., Garrill, J., 2002. Breaking the Silence: Identification of the Communication and Support Needs of Adults with Speech Disabilities in Newcastle. CENTRIS, Newcastle.

Le Fanu, J., 1999. The Rise and Fall of Modern Medicine. Little, Brown and Company, London.

Leon, D., Walt, G., 2001. Poverty, inequality and health in international perspective: A divided world? In: Leon, D., Walt, G. (Eds.), Poverty, Inequality and Health: An International Perspective. Oxford University Press, Oxford.

Marsh, P., Fisher, M., 1992. Good Intentions: Developing Partnership in Social Services. Joseph Rowntree Foundation, London.

Mason, M., Rieser, R., 1992. The Limits of 'Medicine'. In: Rieser, R., Mason, M. (Eds.), Disability Equality in the Classroom: A Human Rights Issue, second ed. Disability Equality in Education, London.

McKeown, T., 1984. The medical contribution. In: Black, N., Boswell, D., Gray, A., et al. (Eds.), Health and Disease: A Reader. Open University Press, Buckingham.

McKnight, J., 1995. The Careless Society. Basic Books, London.

Mencap, 2004. Treat Me Right: Better Healthcare for People with a Learning Disability. Mencap, London.

Mercer, G., 2004. User-led organisations: facilitating independent living. In: Swain, J., French, S., Barnes, C., et al. (Eds.), Disabling Barriers – Enabling Environments, second ed. Sage, London.

Nadirshaw, Z., 1997. Cultural issues. In: O'Hara, J., Sperlinger, A. (Eds.), Adults with Learning Difficulties. John Wiley and Sons, London.

Naidoo, J., Wills, J., 2008. Foundations of Health Promotion. Elsevier, Oxford.

Nutbeam, D., Harris, E., 1999. Theory in a Nutshell: A Guide to Health Promotion Theory. McGraw-Hill Book Company, London.

Oliver, M., 1993. Re-defining disability: a challenge to research. In: Swain, J., Finkelstein, V., French, S., et al (Eds.), Disabling Barriers – Enabling Environments. Sage Publications, London.

Oliver, M., 1996. Understanding Disability: From Theory to Practice. Macmillan, London.

Oliver, M., 2004. If I had a hammer: the social model in action. In: Swain, J., French, S., Barnes, C., et al. (Eds.), Disabling Barriers – Enabling Environments, second ed. Sage, London.

Oliver, M., Sapey, B., 2006. Social Work with Disabled People, third ed. Macmillan, London.

Parr, S., Byng, S., 1997. Talking about Aphasia. Open University Press, Buckingham.

Pearce, W.B., 1994. Interpersonal Communication: Making Social Worlds. Harper Colins, New York.

Pfeiffer, D., 2000. The devils are in the details: the ICIDH2 and the disability movement. Disability Society 15, 1079–1082.

Potts, M., Fido, R., 1991. A Fit Person to be Removed: Personal Accounts of Life in a Mental Deficiency Institution. Northcote House, Plymouth.

Pound, C., Hewitt, A., 2004. Communication barriers: building access and identity. In: Swain, J., Barnes, C., French, S., et al. (Eds.), Disabling Barriers – Enabling Environments. Sage, London.

Power, C., Kuh, D., 2006. Life course development of unequal health. In: Siegrist, J., Marmot, M. (Eds.), Social Inequalities in Health: New Evidence and Policy Implications. Oxford University Press, Oxford.

Pryke, M., 2009. Living in a common world. In: Bromley, S., Clarke, J., Hinchliffe, S., et al. (Eds.), Exploring Social Lives. The Open University, Milton Keynes.

Reynolds, F., 2004. The professional context. In: Swain, J., Clark, J., Parry, K., et al. (Eds.), Enabling Relationships in Health and Social Care: A Guide for Therapists. Butterworth-Heinemann, Oxford.

Roberts, K., 2000. Lost in the system: Disabled refugees and asylum seekers in Britain. Disability Society 15 (6), 943–948.

Rungapadiachy, D.V., 1999. Interpersonal Communication and Psychology for Health Care Professionals: Theory and Practice. Butterworth-Heinemann, Oxford.

Russell, M., 1998. Beyond Ramps: Disability at the End of the Social Contract. Common Courage Press, Monroe.

Ryan, J., Thomas, F., 1987. The Politics of Mental Handicap, second ed. Free Association Books, London.

Ryan, T., Holman, A., 1998. Able and Willing: Supporting People with Learning Difficulties to Use Direct Payments. Values into Action, London.

Scriven, A., 2005. Health promoting practice: a context and overview. In: Scriven, A. (Ed.), Health Promoting

Practice: The Contribution of Nurses and Allied Health Professionals. Palgrave Macmillan, Basingstoke.

Shah, R., 1998. 'He's our child and we shall always love him' – Mental handicap: the parents' response. In: Allott, M., Robb, M. (Eds.), Understanding Health and Social Care: An Introductory Reader. Sage, London.

Siegnal, J., Theorell, T., 2006. Socio-economic position and health: the role of work and employment. In Siegrist, J., Marmot, M. (Eds.), Social Inequalities in Health: New Evidence and Policy Implications. Oxford University Press, Oxford.

Smith, B., Goldblatt, D., 2004. Whose health is it anyway? In: Hitchliffe, S., Woodward, K. (Eds.), The Natural and the Social: Uncertainty, Risk, Change, second ed. Routledge, London.

Standing, S., 1999. The practice of working in partnership. In: Swain, J., French, S. (Eds.), Therapy and Learning Difficulties: Advocacy, Partnership and Participation. Butterworth-Heinemann, Oxford.

Steptoe, A., 2006. Psychological processes linking socio-economic position with health. In: Siegrist, J., Marmot, M. (Eds.), Social Inequalities in Health: New Evidence and Policy Implications. Oxford University Press, Oxford.

Stiker, H., 1997. A History of Disability. University of Michigan Press, Ann Arbor.

Sumsion, T., 2005. Promoting health through client centred occupational therapy practice. In: Scriven, A. (Ed.), Health Promoting Practice: The Contribution of Nurses and Allied Health Professionals. Palgrave Macmillan, Basingstoke.

Sutherland, A.T., 1981. Disabled We Stand. Souvenir Press, London.

Swain, J., 2004. Interpersonal communication. In French, S., Sim, J. (Eds.), Physiotherapy: A Psychosocial Approach, third ed. Butterworth-Heinemann, Oxford.

Swain, J., French, S., 2004. Understanding inequality and power. In: Swain, J., Clark, J., French, S., et al. (Eds.), Enabling Relationships in Health and Social Care: A Guide for Therapists. Butterworth-Heinemann, Oxford.

Swain, J., French, S. (Eds.), 2008. Disability On Equal Terms. Sage, London.

Swain, J., French, S., Cameron, C., 2003. Controversial Issues in a Disabling Society. Open University Press, Buckingham.

Swain, J., French, S., Barnes, C., et al., 2004. Disabling Barriers – Enabling Environments, second ed. Sage, London.

Swain, J., Thirlaway, C., French, S., 2005. Independent evaluation: Developing User Involvement. Leonard Cheshire, London.

Thomas, C., 2007. Sociologies of Disability and Illness: Contested Ideas in Disability Studies and Medical Sociology. Palgrave, Houndmills.

Thompson, N., 1998. Promoting Equality. Macmillan Press, Basingstoke.

Thompson, N., 2001. Anti-Discrimination Practice, third ed. Palgrave, Houndmills.

Thompson, J., Pickering, S. (Eds.), 2001. Meeting the Health Needs of People who have a Learning Disability. Bailliere Tindall, London.

Tones, K., Tilford, S., 2001. Health Promotion: Effectiveness, Efficiency and Equity, third ed. Nelson Thornes Ltd, Cheltenham.

Townsend, P., Davidson, N. (Eds.), 1982. Inequalities in Health: the Black Report. Penguin, Harmondsworth.

Tudar Hart, J., 1971. The inverse care law. Lancet 29, 405–412.

Union of the Physically Impaired Against Segregation, 1976. Fundamental Principles of Disability. Union of the Physically Impaired Against Segregation, London.

Vale, D., 2001. Improving Lives: Priorities in Health Social Care for Blind and Partially Sighted People. On behalf of the Improving Lives Coalition by the Royal National Institute for the Blind, London.

Whalley Hammell, K., 2006. Perspectives on Disability and Rehabilitation: Contesting Assumptions; Challenging Practice. Elsevier, Oxford.

Whitehead, M., 1988. The Health Divide. Penguin Books, Harmondsworth.

Wilder, E.I., 2006. Wheeling and Dealing: Living with Spinal Cord Injury. Wanderbilt University Press, Nashville.

Wiles, F., 2008. Diverse Communities and Resources for Care, Open University course K101 Understanding Health and Social Care, Block 3. The Open University, Milton Keynes.

Chapter 11

Musculoskeletal assessment

Lynne Gaskell

INTRODUCTION

Students are often in awe of qualified clinicians who assess and make complex clinical reasoning decisions in real time with apparently little effort. Becoming competent in patient assessment, like most things in life, takes practice, refinement and reflection, and it looks easy when performed by an expert. The ability to effectively examine and assess patients is an essential skill for physiotherapists to possess. This chapter introduces some important principles of musculoskeletal assessment. It provides an illustrated guide to many of the important techniques and tests that are valuable tools in the arsenal of the chartered physiotherapist. Furthermore, it provides some assessment templates for specific joints of the body. The objectives of this chapter are:

- to identify the appropriate questions to include in a subjective musculoskeletal assessment;
- to discuss the use of regional and special questions for particular joints;
- to explain the use of appropriate subjective and objective markers;
- to explain the use of specific and regional tests at particular joints;
- to recognise the need for continuous reassessment.

This chapter includes templates for assessment of the lumbar spine (including a biopsychosocial assessment), the cervical spine, the shoulder, the hip, the knee, the ankle and the foot.

GENERAL ISSUES

Since 1977, chartered physiotherapists in the UK have been able to work as autonomous practitioners, making treatment decisions independently of other medical professionals. This professional autonomy makes the profession stimulating and exciting, but with it comes a great deal of responsibility. Upon qualifying, physiotherapists are legally responsible for their actions and treatments. Increasing numbers of physiotherapists now work in the primary care setting and this trend is likely to continue. Allowing patients direct access to physiotherapists could relieve other medical practitioners of considerable workload.

Recent years have seen the introduction of extended-scope practitioners, clinical specialist and consultant physiotherapy posts. Physiotherapists in these roles are assessing patients usually referred by general practitioners (GPs) who would otherwise have been examined by a consultant orthopaedic surgeon. These practitioners are required to possess excellent assessment skills, a wide experience of different clinical conditions and pathologies, and to be able to recognise the appropriate course of action for that particular patient. Audits of these interventions have been encouraging; Gardiner and Turner (2002) found that the extended-scope practitioners showed more consistency between clinical diagnosis and arthroscopic findings in the knee than did their medical counterparts. In the present climate, these posts, along with the newly established consultant physiotherapist role, are likely to expand and in doing so will deservedly raise the profile of the physiotherapy profession.

When should physiotherapists assess patients?

- On first patient contact, it is essential to perform an initial assessment to determine the patient's problems and to establish a treatment plan.
- During the treatment, assessment is particularly appropriate while performing treatments such as

mobilisations and exercises when the patient's signs and symptoms may vary quite rapidly. Be aware of any improvement or deterioration in the patient's condition as and when it occurs.
- Following each treatment, the patient should be reassessed using subjective and objective markers in order to judge the efficacy of the physiotherapy intervention. Assessment is the keystone of effective treatment without which successes and failures lose all of their value as learning experiences. Subjective and objective markers are explained later in this chapter.
- At the beginning of each new treatment, assessment should determine the lasting effects of treatment or the effects that other activities may have had on the patient's signs and symptoms. In reassessing the effect of a treatment, it is essential to evaluate progress from the perspective of the patient, as well as from the physical findings.

Format of the assessment

- *Listen* – history and background.
- *Look* – observation.
- *Test* – individual structures (range of movement, strength).
- *Record* – an accurate account of findings.
- *Assess* – and remember to involve the patient.

Aims of the subjective assessment

The aims of subjective assessment are to gather all relevant information about the site, nature, behaviour and onset of symptoms, and past treatments. Review the patient's general health, any past investigations, medication and social history. This should lead to a formulation of the next step of physical tests.

ABC Definition

Symptoms are what the person complains of (e.g. 'my knee hurts'). Signs are what can be measured or tested (e.g. the patient has a positive patellar tap test).

Aims of the objective assessment

The objective assessment aims to seek abnormalities of function, using active, passive, resisted, neurological and special tests of all the tissues involved. This may be guided by the history. However, it is important to conduct all tests objectively and equally, and not attempt to bias the findings in an attempt to make the hypothesis fit.

Objective examination is concerned with performing and recording objective signs. It aims to:

- reproduce all or parts of the patient's symptoms;
- determine the pattern, quality, range, resistance and pain response for each movement;
- identify factors that have predisposed or arisen from the disorder;
- obtain signs on which to reassess the effectiveness of treatment by producing reassessment 'asterisks' or 'markers' (Jull, 1994).

SUBJECTIVE ASSESSMENT

Initial questioning

Subjective assessment needs to include the name, address and telephone number of the patient, and the patient's hospital number, if appropriate. Both the age and the date of birth of the patient should be recorded. The medical referrer's name and practice should also be recorded for correspondence, discharge letters, etc.

It is also essential for the physiotherapist to obtain sufficient details of the patient's employment. Is the patient currently working? If not, determine the reasons for this. Is it because the person is unable to cope with the physical demands of the job? Do heavy lifting, repetitive movements or inappropriate sustained postures increase the symptoms? These factors may be precursors of poor posture and muscle imbalance, which may accentuate degenerative disease and increase symptoms. However, it is equally important to recognise that withdrawing from normal activities of daily life can result in deconditioning of musculoskeletal structures that may lead to degenerative disease and an increase in symptoms (Waddell 1992; Frost et al. 1998).

Identify the patient's hobbies or interests. Is she/he able to participate in a sport if desired? If not, determine the reasons. Identify the length of time the patient has been off work or has been unable to participate in physical activities. Evaluate the progression of symptoms. If the person has not been participating in physical activities, and if no improvement has occurred it may be appropriate to advise a return to light training in order to prevent devitalisation of tissues and fear avoidance issues.

Present condition

Area of the symptoms

It is useful to record the area of the pain by using a body chart, because this affords a quick visual reference (Maitland 2001). The patient may complain of more than

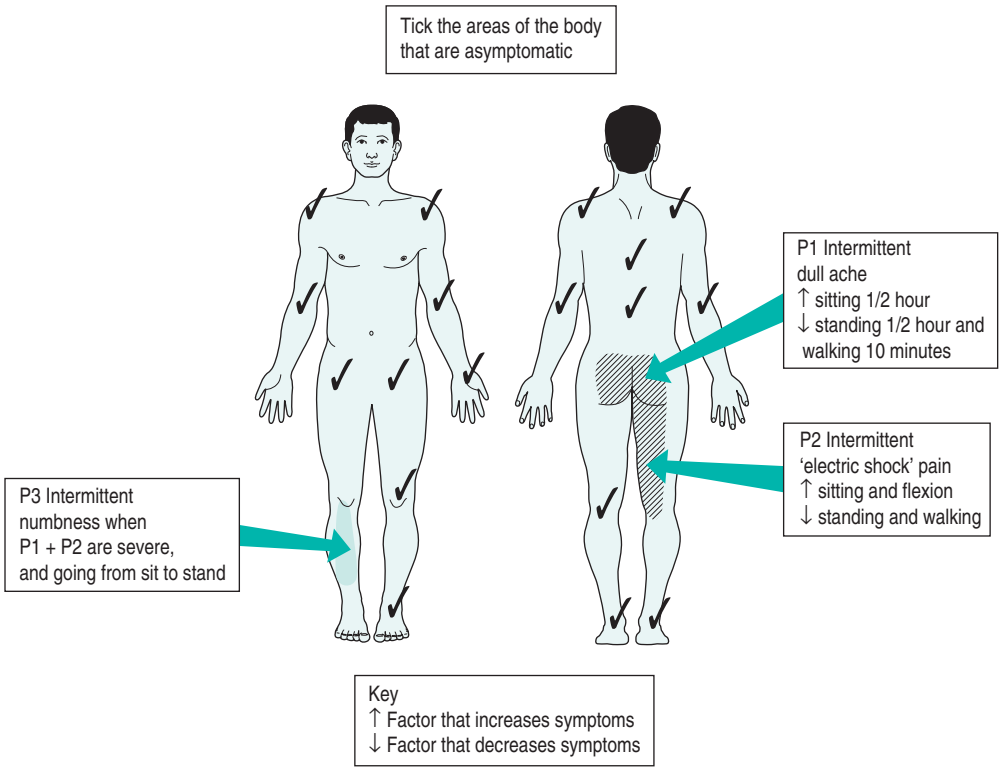


Figure 11.1 Typical body chart. In this example the patient's details have been recorded.

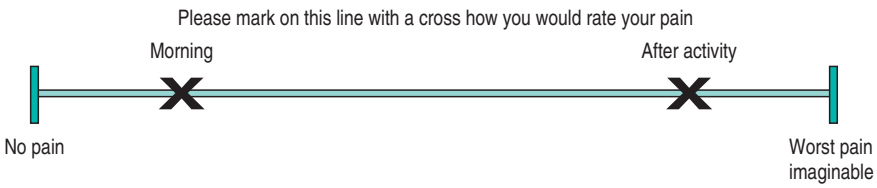


Figure 11.2 The visual analogue scale (VAS).

one symptom, so the symptoms may be recorded or referred to individually as P1 and P2 and so on. Areas of anaesthesia or paraesthesia may be recorded differently on the pain chart – they may be represented as areas of dots in order to distinguish them from areas of pain (Figure 11.1).

Severity of the symptoms

The severity of the pain may be measured on a visual analogue scale (VAS) (Figure 11.2) or on a numerical scale of 0–10 to quantify the pain, where 0 stands for no pain at all and 10 is perceived by the patient as the worst pain imaginable. The mark on a VAS can then be measured and recorded for future comparisons using a ruler. Although these measures are not wholly objective, they do allow changes to be monitored as the treatment progresses.

Duration of the symptoms

Establish whether the pain and symptoms are intermittent or constant. Is the pain present all of the time or does it come and go depending on activities or time of day?

Aggravating and easing factors

Positional factors

Most musculoskeletal pain is mechanical in origin and is therefore made better or worse by adopting particular positions or postures that either stretch or compress the structure that is giving rise to the pain. Moreover, aggravating and easing movements may provide the physiotherapist with a clue as to the structure that is causing the pain. Various body or limb positions place different structures

on stretch or compression and the resultant deformation produces an increase in severity of the pain. The aggravating and easing factors can be recorded on the pain chart, as in Figure 11.1. It is also necessary to record the length of time that engaging in aggravating activities produces an increase in symptoms or, alternatively, takes to settle down. This indicates the irritability of the patient's condition.

Time factors

It is useful to record the behaviour of signs and symptoms over a 24-hour period – the diurnal pattern. Do the symptoms keep the patient awake or awaken the person regularly during the night? Is this because of a particular sleeping posture or to other, unrelated factors? On arising, how are the symptoms for the first hour or so of the day and, moreover, do the symptoms vary from the morning to the afternoon and into the evening? Does this follow a particular pattern? This information can be included on the body chart.

Be careful not to confuse time of day with the performance of particular activities that the patient may undertake at that time. Certain pathologies tend to be more painful at characteristic times of the day. For example, chronic osteoarthritic changes are characteristically painful and stiff initially on arising from sleep, intervertebral disc related pain is often more painful on arising owing to the disc imbibing water during sleep and thus exerting pressure on pain-sensitive structures. Prolonged morning pain and stiffness, which improves only minimally with movement, suggests an inflammatory process (Magee 1992).

Determining the SIN factors

Once the severity of the symptoms and the aggravating and easing factors have been noted, it is then possible to determine the SIN factor of the condition: severity/irritability/nature. SIN factors are used to guide the length and firmness of the objective assessment and subsequent treatment.

Severity

This can be quantified by the VAS, numerical scale or other valid pain questionnaire. It can be recorded as high (pain score of around 7–10), moderate (score around 4–6) or low (score around 1–3).

Irritability

This is the time that the person has to perform the activity to increase the pain and, conversely, how long it takes before the pain settles to its former intensity. It can be measured as either high (the aggravating factor causes the pain to increase very quickly or instantly and then the pain takes a long time to settle back), moderate (the aggravating factor takes longer to increase the symptom) or low (the aggravating factor can be performed for a long time before exacerbating the patient's symptoms and then on stopping the activity the symptoms subside rapidly). An example of low irritablity would be that the knee pain is aggravated after jogging for one hour and then subsides after one minute of rest.

Nature

It is possible to hypothesise the nature of the condition following the subjective history, i.e. whether the patient's condition has a predominantly inflammatory, traumatic, degenerative or mechanical cause.

History of the present condition

Insidious onset

Insidious onset means that the patient's symptoms appear without any obvious cause. An example of this would be a degenerative condition such as osteoarthritis. These types of conditions often begin with a small amount of stiffness and pain, which is characterised by exacerbation and remission but is, nonetheless, progressive.

Traumatic onset

Can the onset of symptoms be related to a particular injury? Identify if there was a definite cause for the patient's symptoms. The mechanism of injury may be indicative of the structures damaged. For example, a valgus strain of the knee may stretch the medial collateral ligament of the knee, whereas forced rotation of the knee joint when in a semi-flexed weight-bearing position may tear the menisci.

Progression of the condition

Are the patient's symptoms getting better or worse? Acute soft-tissue injuries normally undergo a period of inflammation and repair, and symptoms may subside rapidly within a few days or weeks. However, progressive arthritic diseases may have a history of exacerbation and remissions with a general increase in the severity or frequency of their symptoms, as the disease progresses.

Progression of the condition may indicate how quickly the patient's symptoms will subside.

Chronicity or age of the condition

How long has the patient experienced the symptoms? Is the condition acute or chronic? If the injury is chronic or has not resolved completely, it may indicate a number of different causes, such as mechanical instability from a ligament disruption, functional instability because of weakened muscles, loss of proprioception (and therefore the

loss of an inherently protective reflex mechanism at the joint) or malalignment. Furthermore, it may be developing into a degenerative condition. The physiotherapist should identify the following:

1. Is this the first episode?
2. Is it recurrent?
3. Is it getting better or worse?

Previous treatments

Has the patient received any treatment for this condition in the past and, if so, was it effective? Was the improvement partial or total, and did it provide permanent or temporary relief? If the treatment has been effective in the past it may well help again. Be careful not to repeat unsuccessful interventions as they are unlikely to be therapeutic.

Investigations

Record the results of any investigations that the patient has undergone. Case notes, radiographic films and reports can be ordered and read, as patients may not always be a reliable source of the results of their investigations.

X-rays, MRI scans, CAT scans and bone scans

Scans are now commonly used to aid the diagnosis of musculoskeletal disorders. X-rays are useful in that they show the degree or extent of arthritis present at a joint. They are also useful in determining the extent of osteomyelitis (bone infection) and some malignancies and osteoporosis. Moreover, they are valuable following trauma to identify fractures or dislocations. Be aware, however, that there is a poor correlation between X-rays and spinal symptoms for non-specific low back and neck pain. What is identified as pathological on these tests may not always be the structure responsible for the patient's signs and symptoms. Routine X-rays are not helpful in non-specific degenerative spinal disease (CSAG 1994).

Computerised axial tomography (CAT) scans may be used to identify the precise level and extent of disc prolapse and subsequent nerve impingement prior to discectomy. Magnetic resonance imaging (MRI) may be used to identify ligamentous and muscular injuries, particularly in athletes, as well as discogenic prolapse. Bone scans are sensitive to 'hot spots' or areas of inflammation present in bone and may detect malignancy or diseases such as ankylosing spondylitis, some fractures and infection sites.

Blood tests

These are used extensively for the confirmation of the diagnosis of particular diseases such as rheumatoid arthritis, ankylosing spondylitis, osteomyelitis and malignancy.

Other investigations

The patient may be undergoing investigations for other pathologies that could possibly relate to the musculoskeletal condition. These should be noted and recorded.

Past medical history

Determine whether or not the patient is suffering, or has suffered, any major operations or illnesses. These may affect the vitality of the tissues and be a contraindication to particular treatments. Examples are respiratory or cardiac disease, diabetes, rheumatoid arthritis and epilepsy.

The prolonged use of oral steroid medication should be noted, as this affects bone density and produces a tendency towards bruising. This is commonly found in patients suffering chronic respiratory diseases, inflammatory bowel diseases or rheumatoid arthritis. Always identify cases of unexplained weight loss and general debility.

Clinical note

Unexplained weight loss, general debility or the patient looking generally unwell – unremitting pain that is unrelieved by changing position or medication – may suggest a non-mechanical basis for the pain. Feeling unwell or tired is common in neoplastic disease affecting the spine (O'Conner and Curner 1992). In these cases, malignant disease should be suspected and the patient should be referred back immediately to the referring GP or consultant with a full report of your findings. Physiotherapy management may well be contraindicated in this situation and may be wasting valuable time for the patient.

Medication

Record the type and dosage of medication prescribed for, or taken by, the patient. Commonly prescribed drugs for use in musculoskeletal conditions are:

- analgesics (painkillers), e.g. paracetamol and co-codamol;
- non-steroidal anti-inflammatory drugs (NSAIDs), e.g. ibuprofen;
- skeletal muscle relaxants, e.g. diazepam and baclofen.

Medications being taken should alert you to pathologies that the patient may have forgotten to inform you about. For example, a person may tell you that she/he has no significant medical history, but then later in the assessment say that she/he is currently taking anticoagulation therapy for a recent deep vein thrombosis!

OBJECTIVE ASSESSMENT

Following the subjective assessment it is important to highlight the main findings and determine the SIN factor. A hypothesis may be reached as to the cause of the patient's symptoms and the testing procedures are performed in order to support or refute the physiotherapist's hypothesis.

General observation

Observe the person's gait and general demeanour on entering the department.

Local observation

Note any localised swelling at the joint. This may be measured with a tape measure around the joint or limb circumference. Note any asymmetry of joint contours, redness of the overlying skin suggesting local inflammation, atrophy and asymmetry of musculature, deformity, and malalignment of the joint or joints. Compare one joint closely with the other side whenever possible.

Posture

Observe any asymmetry of posture in standing, walking and sitting. Poor posture is frequently a precursor to muscle imbalance, selective tightness and weakness through over- or under-use of specific muscles. The result of prolonged poor postural habits may lead to an acceleration of certain pathologies such as adhesive capsulitis, shoulder impingement syndrome, spinal pain and arthritis. Poor posture is frequently the cause of aches and pains and may be correctable in the early stages and improved in later stages. Correction may prevent recurrence or acceleration of specific pathologies.

Palpation

Palpate for the following:

- tenderness;
- heat (use the back of your hand – it is more sensitive to heat changes);
- swelling;
- muscle spasm.

Assessment of movement

Active movements

These are movements performed by the patient's voluntary muscular effort.

Passive movements

These are movements performed by an external source, such as the physiotherapist or a pulley system. There are two types of passive movements.

- Physiological movements are movements that could be performed actively by the patient (e.g. flexion of the knee or abduction of the shoulder joint).
- Accessory movements cannot be performed actively by the patient (e.g. they incorporate glide, roll or spin movements that occur in combination as part of normal physiological movements). An example of an accessory movement is an anterior–posterior glide at the knee joint.

Resisted movements

These are performed against the resistance of the physiotherapist or weights by the patient's own effort.

Clinical note

Passive, active and resisted movements are used in the assessment and in the treatment of musculoskeletal disorders. Specific examples of these are included later in the individual joint assessments formats.

Assessment of range of movement

Measurement of joint range using a goniometer

Active movement may be assessed by the use of a goniometer (Figure 11.3) or, alternatively, by visual estimation. It

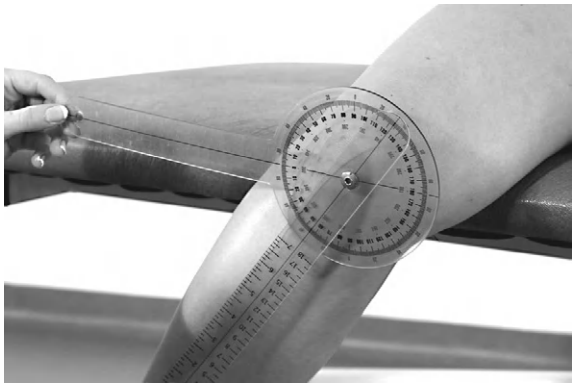


Figure 11.3 Using the goniometer to measure hip joint medial rotation with the hip in 90 degrees of flexion.

is measured in degrees and it is useful to practise using the goniometer by measuring the hip, knee and ankle joints in various positions. Either the 360° or 180° universal goniometers may be used. Ensure adequate stabilisation of adjacent joints prior to taking the measurements and locate the appropriate anatomical landmarks as accurately as possible. For details on specific joint measurements using the goniometer, refer to the appropriate joint assessment. Physiological and accessory passive movements are measured in terms of the above and by the end-feel respectively.

Differentiation tests

If a lesion is situated within a non-contractile structure such as ligament, then both the active and passive movements will be painful and/or restricted in the same direction. For example, both the active and passive movement of inversion will produce pain in the case of a sprained lateral ankle ligament. However, if a lesion is within a contractile tissue such as a muscle, then the active and passive movements will be painful and/or restricted in opposite directions (Cyriax 1982). For example, a ruptured quadriceps muscle will be painful on passive knee flexion (stretch) and resisted knee extension (contraction).

Clinical note

Remember that it is insufficient to measure only the range of movement occurring. The quality of movement should also be observed, along with limiting factors to the movement. Is it the pain, muscle spasm, weakness or stiffness that is limiting the movement? This is determined by noting the differences between active, passive and resisted movements.

End-feel

During passive movements, the end-feel is noted. Different joints and different pathologies have different end-feels. The quality of the resistance felt at the end of range has been categorised by Cyriax (1982). For example:

- bony block to movement or a hard feel is characteristic of arthritic joints;
- an empty feel, or no resistance offered at the end of range, may be a result of severe pain associated with infection, active inflammation or a tumour;
- a springy block is characterised by a rebound feel at the end of range and is associated with a torn meniscus blocking knee extension;
- spasm is experienced as a sudden, relatively hard feel associated with muscle guarding;
- a capsular feel shows a hardish arrest of movement.

Assessment of muscle strength

Symptoms arising from resisted contractions

The Oxford scale is relatively quick and easy to use, and is used widely in clinical practice. However, it is not very objective, functional or sensitive to change as the movements resisted are concentric contractions and the spaces between the grades are not linear. Nevertheless, it provides a guide to muscle strength and is somewhat sensitive to change.

The Oxford classification

0 = no contraction at all

1 = flicker of contraction only, movement of the joint does not occur

2 = movement is possible only with gravity counterbalanced

3 = movement against gravity is possible

4 = movement against resistance is possible

5 = normal functional movement is possible

Measurements using isokinetic machines

Objective measurements of strength throughout different joint angles and at different velocities are made more accurately using isokinetic machines, such as Cybex or Kin-Kom. These machines are particularly valuable in rehabilitative regimens such as anterior cruciate rehabilitation programmes and can determine the strength ratio of the quadriceps to the hamstrings, or the ratios of the operated versus the non-operated leg. Objective markers such as percentages of strength ratios or ratios of operated versus non-operated leg may be used in setting discharge protocols. Isokinetic machines have been found to be reliable and valid in measuring muscle torque, muscle velocity and the angular position of joints (Mayhew et al. 1994). However, they are limited in their use, and Wojtys et al. (1996) suggest that agility and functional exercises may be more beneficial than isokinetic machines in the strengthening of muscle.

Clinical note

Tests of specific structures are performed in order to reproduce the patient's symptoms or signs, i.e. to reproduce the comparable sign. Differentiation tests are useful to distinguish between two or more structures that are suspected to be the source of the symptoms.

Differentiation tests of muscles and tendons

These are contractile structures and are therefore tested by performing a contraction against resistance. A pain response and/or apparent weakness may indicate a strain of the muscle at any particular point of the range of movement. Full range should be checked as the muscle may be weak only at a particular point in the range. Muscle length may also be tested, particularly those muscles that are prone to become tight and then lose their extensibility. Muscles that pass over two joints and have mobiliser characteristics are particularly prone to tightness. Examples of these are the hamstrings, rectus femoris, gastrocnemius and psoas major. The length of the muscle is tested by passively moving the appropriate joints. The stretch is compared with the other side to determine reproduction of pain and/or restriction of movement.

Passive insufficiency of muscles

This occurs with muscles that act over two joints (Figure 11.4a). The muscle cannot stretch maximally across both joints at the same time. For example, the hamstrings may limit the flexion of the hip when the knee joint is in extension as they are maximally stretched in this position. However, if the knee is flexed passively, then the hip will be able to flex further as the stretch on the hamstrings has been reduced.

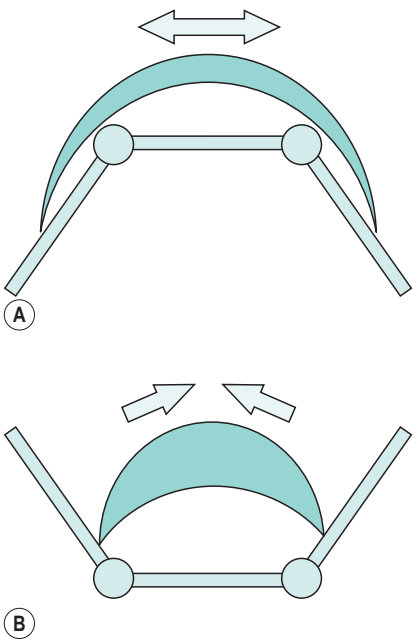


Figure 11.4 (a) Passive insufficiency: the muscle cannot simultaneously stretch maximally across two joints. (b) Active insufficiency. The muscle cannot simultaneously contract maximally across two joints.

Active insufficiency of muscles

This, too, occurs with muscles that act over two joints (Figure 11.4b). The muscle cannot contract maximally across both joints at the same time. An example is the finger flexors. If you are to make a strong fist, you may notice that the wrist is in a neutral or an extended position when you do this action. Now, if you attempt to actively flex your wrist joint whilst keeping your fingers flexed, you will find that the strength of the grip is greatly diminished. This is because the wrist and finger flexors are unable to shorten any further and so the fingers begin to extend or lose grip strength.

Differentiation tests of ligaments

Ligaments are non-contractile structures and are tested by putting the structure on stretch. Examples are a valgus strain of the knee to stretch the medial collateral ligament of the knee or passive inversion of the subtalar joint to stretch the lateral ligament of the ankle. A positive test would be a pain response or observation or feel of any excessive movement of the joint when compared with the other side.

Differentiation tests of bursae

Bursae are sacs of synovial fluid. Inflammation of these (bursitis) results in tenderness and/or heat on palpation. The tenderness is often very localised to the site of the inflamed bursa.

Differentiation tests of menisci

The history and mechanism of injury combined with anterior joint tenderness and the inability to passively hyperextend the knee are useful diagnostic markers of meniscal injury. Rotation on a semi-flexed weight-bearing knee is a common cause of injury.

A history of locking, whereby the joint momentarily locks and is unable to actively or passively release itself from the position, is also common. Objectively, the knee joint is unable to fully flex/hyperextend passively.

Characteristics of degenerative joint disease

Signs and symptoms may include:

- pain that increases on weight-bearing activities (standing and walking, walking downstairs particularly);
- insidious onset of symptoms followed by progressive periods of relapses and remissions;
- pain and stiffness in the morning;
- stiffness following periods of inactivity;
- pain and stiffness that arise after unaccustomed periods of activity;

- bony deformity (e.g. characteristic varus deformity may follow from collapse of the medial compartmental joint space);
- reduction of the joint space observed on X-ray, with bony outgrowths or osteophytes.

Writing up the assessment

It is imperative to record the assessment immediately following the physical testing. Patient notes should be completed on the day of the assessment for legal reasons.

Ensure that your assessment findings are clear and concise, and that they highlight the main points (it may be useful to include one subjective and one objective marker). Formulate a problem list in agreement with the patient. Agree and record SMART goals (specific, measurable, achievable, realistic, timed) with the patient. Use the problem-orientated medical records (POMR) system.

Remember, if you have insufficient time to conduct a full and thorough assessment you can always continue with this when the patient attends for his/her subsequent appointment.

Spinal assessments

THE LUMBAR SPINE

Posture

Normal alignment

Posteriorly, the shoulders, waist creases, posterior superior iliac spines, gluteal creases and knee creases should be horizontal (Figure 11.5). The spine should appear to be vertical. There should be no rotation, side flexion, scoliosis (lateral curvature) or shift (lateral deviation). Laterally, you should observe a normal lordosis in the lumbar spine. *Anteriorly*, the anterior superior iliac spines should be horizontal.

Common deviations from normal posture (refer to Figure 11.6)

- *Creases* in the posterior aspect of the trunk and, particularly, adjacent to the spine may indicate areas

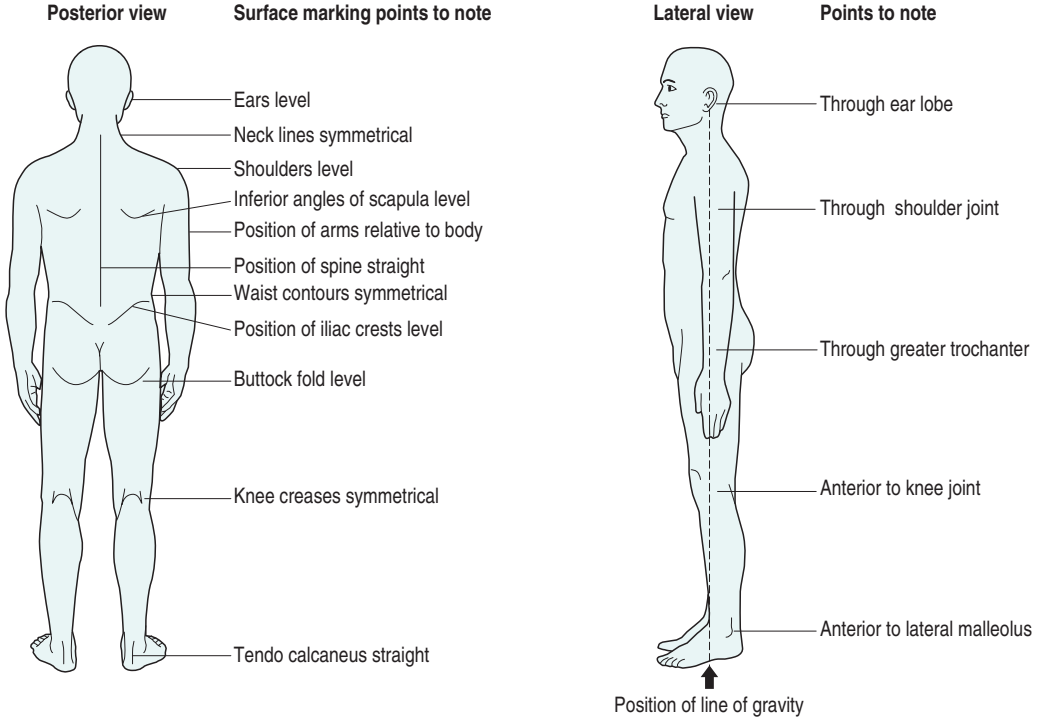


Figure 11.5 Examination of the spine.

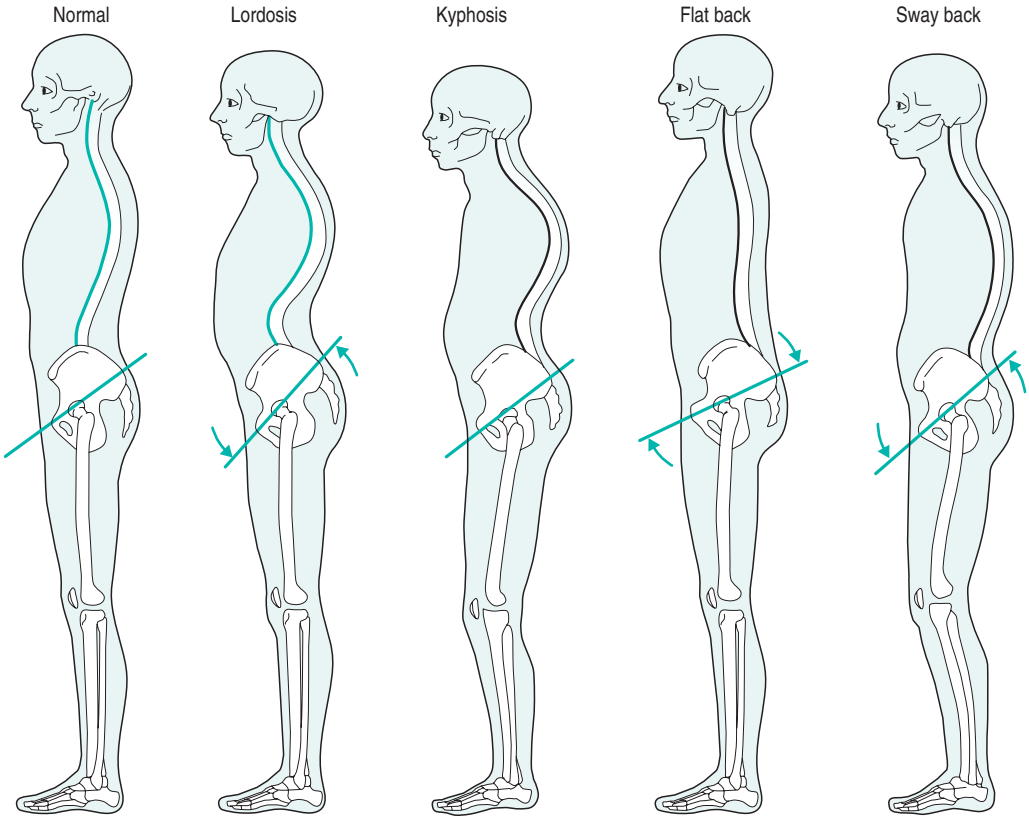


Figure 11.6 Abnormal postural curves of the spine.

of hypermobility or instability of that motion segment.

- *Sway back* comprises hyperextension of the hips, an anterior pelvic tilt and anterior displacement of the pelvis.
- *Flat back* consists of a posterior pelvic tilt and a flattening of the lumbar lordosis, extension of the hip joints, flexion of the upper thoracic spine and straightening of the lower thoracic spine.
- *Kypholordosis* consists of a forward-poking chin posture, elevation and protraction of the shoulders, rotation and abduction of the scapulae, an increased thoracic kyphosis, anterior rotation of the pelvis and an increased lumbar lordosis.
- *Shifted posture* (lateral shift) commonly arises from disc herniation or acute irritation of a facet joint. The shift is thought to result from the body finding a position of ease whereby the shoulders are displaced laterally in relation to the pelvis. Most commonly the shift occurs away from the painful side (Figure 11.7).

Movements

Assess not only the range of movement occurring and the pain response, but also localised areas of give and restriction occurring at specific motion segments.

Active movements

Flexion

Flexion should result in a smooth curve. Segmental areas of give or restriction appear as hinging (segmental hypermobility). Lack of movement in the lumbar spine may be compensated by flexion at the hips or thoracic spine flexion. The gross movement may be measured as fingertip-to-floor distance with a tape measure. Note any limitation of movement, lateral deviation and pain response (Figure 11.8).

Extension

Observe extension in relation to areas of give or restriction. Observe for hinging at specific motion segments indicating areas of hypermobility. This may appear as horizontal

Figure 11.7 Lateral shift.

lines appearing across the hypermobile segment. Note any limitation of movement and pain response.

Side flexion (Figure 11.9)

Normal movement should be observed as a smooth curve. Areas of give or restriction will be observed as hinging (segments of hypermobility) or plane lines (areas of hypomobility). Compare with the other side for symmetry. Note any 'coupling' of movements, i.e. the trunk may flex or rotate to compensate for restriction of side flexion.

Passive physiological intervertebral movements (PPIVMs)

These can be used to confirm any restriction of motion seen on active movement tests and to detect restriction of movement not discovered by the active movement tests. PPIVMs also detect segmental hypermobility (Magarey 1988; Maitland 2001).

Overpressure

If the plane movements have full range and are pain-free, then overpressure applied slowly and with care can be

Figure 11.8 A faulty lumbar flexion pattern. All movements occur at the hip joint while the lumbar spine remains in an extended position.

administered. At the end of the available range the physiotherapist may apply a small oscillatory movement to assess the quality and end-feel of the movement. Also, the range of further movement should be noted, as well as the pain response.

Repeated movements

Repeating movements several times may alter the quality and range of the movement and may give rise to latent pain. McKenzie (1981) advocates the use of repeating flexion and extension in both standing and lying to determine the movement that may centralise the patient's symptoms (Figure 11.10). According to Palmer and Epler (1998), progressive worsening of pain on repeated movements indicates a disc derangement – the pain either becoming more intense or spreading more distally. Centralisation of symptoms means that the referred pain becomes more proximal, i.e. pain experienced at the medial aspect of the shin may centralise to the buttock. Thus, the exercise is believed to be reducing the patient's symptoms and the disc derangement.

Combined movements

According to Edwards (1992): 'Although the use of combining movements is not always necessary – adequate

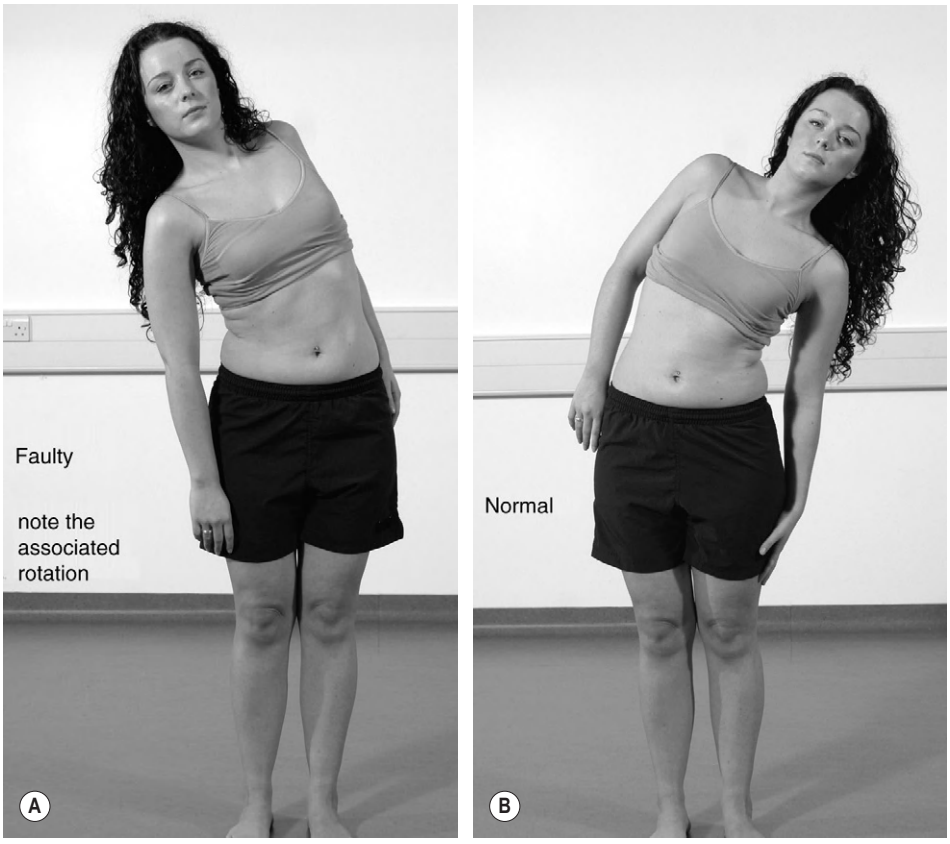


Figure 11.9 Side flexion (a) to the right, and (b) to the left. Note the difference, and the pain response.

Figure 11.10 Repeated extension in prone.

results being obtained by standard examination procedures – there are times when they are helpful. Often, with the more difficult mechanical problems, their use is essential.'

For example, lumbar spine extension may be performed and, while maintaining that extension, side flexion may be added. Symptoms are likely to vary with the addition of a second movement and this may indicate whether or not there is a regular or irregular stretch component to the signs and symptoms. For example, if a disc prolapse is aggravated by flexion, it would be reasonable to hypothesise that the addition of contralateral side flexion would also further increase the symptoms because both of the movements are stretching the posterior component of the disc and posterior longitudinal ligament. Combining ipsilateral side flexion to flexion would be expected to lead to a reduction in the patient's symptoms as the ipsilateral side flexion is reducing the stretch component.

Differentiation between the hip and lumbar spine as a source of symptoms

The hip joint may give rise to pain in the buttock or groin. In order to differentiate between pain arising as a result of spinal or hip pathology it is important that the therapist discounts the hip joint as a possible source of symptoms. With the patient supine, full flexion, medial and lateral rotation is performed actively and passively at the hip

Figure 11.11 Compression testing of the posterior sacroiliac ligaments.

Figure 11.12 The Faber or four test. Normal range of movement.

joint. These are the movements commonly painful or restricted by degenerative joint conditions such as osteoarthritis. If these movements are pain-free and full-range then it is unlikely that the hip is a source of symptoms. Compare both sides.

Assessing the sacroiliac joint

Sitting flexion (Piedello's sign)

The seated patient is asked to flex forwards. The physiotherapist palpates the sacral dimples bilaterally. Both sacral dimples should move equally in a *cephalad direction* (i.e. towards the head). (This tests the movement of the sacrum on the ilium.) Excessive rising of one side indicates hypomobility at that sacroiliac joint.

Standing flexion (stork test)

With the patient standing, the physiotherapist locates the sacral dimples (level of S2) and places the other hand centrally at the sacrum. The patient is instructed to stand on one leg while flexing the non-weight-bearing hip and knee. The sacral dimple on the non-weight-bearing side should appear to move *caudally* (towards the floor) by approximately 1 cm as the ilium rotates posteriorly. Hypomobility is observed if the dimple does not move distally in relation to the sacrum.

Compression tests

Posterior ligaments

These test the integrity of the *posterior* sacroiliac ligaments. The patient lies supine and the hip is passively flexed towards the ipsilateral shoulder (Figure 11.11). A downward thrust is applied along the line of the femur. Observe for pain response, clunk and difference in end-feel between both sides. The test is repeated for (oblique) hip flexion towards the contralateral shoulder and (transverse) hip flexion towards the contralateral hip.

Anterior ligaments – Faber test

Flexion plus abduction plus external rotation (the 'Faber' test) tests the integrity of the anterior sacroiliac ligaments. The test is also described as the 'four test' because of the position of the patient's limb, a combination of flexion, abduction and external rotation. The physiotherapist pushes the leg downward, just proximal to the knee joint while stabilising the opposite hip with the other hand. A normal finding would be to lower the leg to the level of the opposite leg. Observe for pain response or limitation of movement (Figure 11.12).

Neurological testing

Compression or traction of spinal nerve roots by disc trespass and/or osteophytes may give rise to referred pain, paraesthesia and anaesthesia, and also give positive neurological signs. Neurological signs should be carefully monitored as deterioration may indicate worsening pathology.

Dermatomes

A dermatome is an area of skin supplied by a particular spinal nerve. Dermatomes may exhibit sensory changes for light touch and pin prick. Test each dermatome individually, on the unaffected and then the affected side.

Myotomes

A myotome is a muscle supplied by a particular nerve root level. These are assessed by performing isometric resisted tests of the myotomes L1–S1 in middle range, held for approximately three seconds. Test the unaffected side, then the affected: LI–L2 for the hip flexors (see Figure 11.13), L3–L4 for knee extensors (see Figure 11.14), L4 for foot dorsiflexors and invertors, L5 for extension of the big toe, S1 for plantar flexion (see Figure 11.15) and knee flexion, S2 for knee flexion and toe standing, and S3–S4 for muscles of the pelvic floor and the bladder.

Figure 11.13 Resisted hip flexion to test myotome L1/2.

Figure 11.14 Resisted knee extension to test myotome L3.

Reflexes

- Test the non-affected first then affected side. Note: dull reflexes may indicate lower motor neurone dysfunction. Brisk reflexes may indicate an upper motor neurone dysfunction.
- L3 corresponds to the quadriceps. The patient sits with the knee flexed and the therapist hits the patellar tendon just below the patella (Figure 11.16).
- S1 corresponds to the plantarflexors. Dorsiflex the ankle and strike the Achilles tendon. Observe and feel for plantar flexion at the ankle (Figure 11.17).

Clinical note

The Babinski reflex (or plantar response) is an abnormal response and occurs when a blunt object is drawn up the lateral aspect of the sole of the foot. Normally, the great toe (big toe) flexes. Abnormally, the great toe extends indicating upper motor neurone damage. Note that this primitive reflex is seen in the newborn but disappears with time.

Adverse mechanical tension

Passive neck flexion

The patient is supine. The physiotherapist flexes the patient's neck passively. Observe for any low back pain response, which may suggest disc pathology.

Straight leg raise (SLR)

This is also known as Lasegue's test. The patient is supine. The physiotherapist lifts the patient's leg while maintaining extension of the knee (Figure 11.18). An abnormal

Figure 11.15 Toe standing (plantar flexion) to test myotome S1.

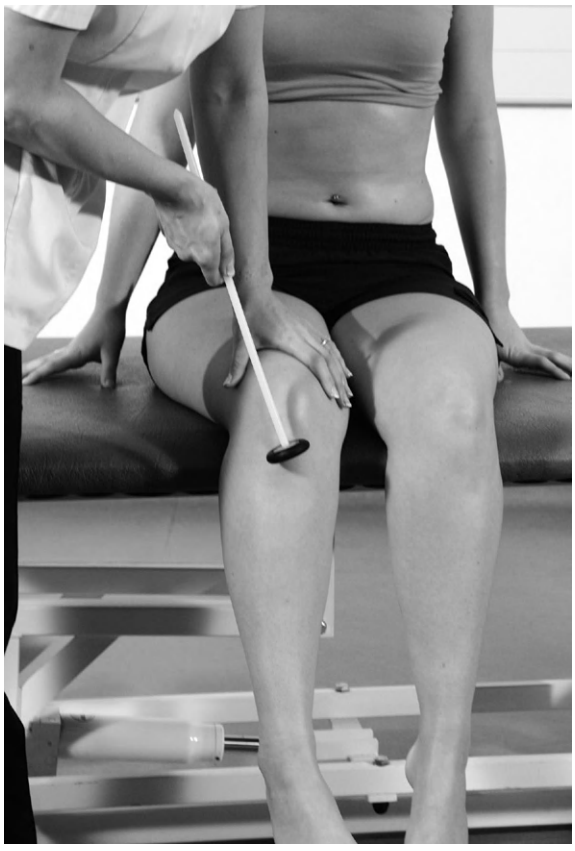


Figure 11.16 The quadriceps reflex (L2, L3, L4).

Figure 11.17 The Achilles tendon reflex (S1, S2).

Figure 11.18 SLR test adding dorsiflexion of the ankle joint.

Figure 11.19 Femoral nerve test.

finding is back pain or sciatic pain. The sciatic nerve is on full stretch at approximately 70 degrees of flexion, so a positive sign of sciatic nerve involvement occurs before this point (Palmer and Epler 1998). Any pain response and range of movement is noted and comparison made with the other side. Factors such as hip adduction and medial rotation further sensitise the sciatic nerve; dorsiflexion of the ankle will sensitise the tibial portion of the sciatic nerve; plantar flexion and inversion will sensitise the peroneal portion of the nerve.

Prone knee bend (femoral nerve stretch)

The patient lies prone and the physiotherapist flexes the person's knee and then extends the hip (Figure 11.19). Pain in the back or distribution of the femoral nerve indicates femoral nerve irritation or reduced mobility. Comparison is made with the other side.

Slump test

This tests the mobility of the dura mater. The patient sits with thighs fully supported with hands clasped behind the back. The patient is instructed to slump the shoulders towards the groin (Figure 11.20). The physiotherapist applies gentle overpressure to this trunk flexion. The patient adds cervical flexion, which is maintained by the therapist. The patient then performs unilateral active knee extension and active ankle dorsiflexion. The physiotherapist should not force the movement. The non-affected side should be assessed first.

Any symptoms are noted at the particular part in range. If the dura mater is tethered, symptoms will increase as each component is added to the slump test. The patient is instructed to extend the head – a reduction in symptoms on cervical extension is a positive finding, indicating abnormal neurodynamics.

Testing for lumbopelvic stability

Stability of the lumbar spine is necessary to protect the lumbopelvic region from the everyday demands of posture and load changes (Panjabi 1992). It is essential for pain-free normal activity (Jull et al. 1993) and should always be assessed.

With the patient in crook lying with the hips at 45 degrees flexion, he/she is instructed to maintain a neutral spine (it may be useful to tell the patient to maintain such a lordosis that an army of ants could just crawl through!). The person then performs an abdominal in-drawing by contracting the transversus abdominis muscle while attempting to maintain the spine in neutral. To challenge the transversus abdominis and multifidus stabilising muscles (and consequently the spinal position), the patient adds the leg load by alternately lifting the heels from the floor and sliding out the leg while maintaining a neutral spine position. The maintenance of a neutral spine posture can be assessed by using a biofeedback device. An inability to maintain the spine in neutral will result in the lumbar spine extending as the leg is lifted. The intra-abdominal pressure mechanism is controlled primarily by the diaphragm and transversus abdominis which provides a stiffening effect on the lumbar spine (Hodges and Richardson 1997).

Figure 11.20 Slump test with single knee extension.

Palpation

Soft-tissue thickening over the articular pillar at one or more spinal levels is a common finding in cases of degenerative disease of the lumbar spine, as is hard bony thickening and prominence over the apophyseal joints. Note any general tightness or localised thickening of muscular tissue or ligamentous tissue. In general, the older the soft-tissue changes, the tougher they are; the more recent, the softer they are. However, a thickened or stiff area is not necessarily painful or the source of a patient's symptoms (Maitland 2001).

Accessory spinal movements

The physiotherapist applies central posteroanterior pressures on the spinous processes and unilateral (one-sided) pressure over the articular pillar (Figure 11.21), noting areas of hyper- and hypomobility. Record any

Figure 11.21 Unilateral pressures applied to the lumbar spine over the articular pillar. Note the pressure is applied through the physiotherapist's pisiform bone.

pain experienced by the patient and the corresponding spinal level.

Biopsychosocial assessment (lumbar spine)

Although historically there has been no change in the pathology or prevalence of low back pain (LBP), disability owing to non-specific LBP has increased dramatically in modern Western societies. Medical interventions for chronic LBP using a limited biomedical approach have been relatively unsuccessful and the cost to National Health Service (NHS) physiotherapy services has been estimated to be £151 million per annum (Maniadakis and Gray 2000). LBP has been described as a twentieth-century healthcare disaster (Waddell 1992). In 80% of cases of LBP no significant pathology is ever found, despite considerable disability. Recent guidelines based on systematic reviews of contemporary literature have recommended a biopsychosocial approach in the assessment and treatment of LBP (CSAG 1994; RCGP 1999; NICE 2009).

CASE STUDY

LUMBAR SPINE

A 30-year-old labourer was referred for physiotherapy following a lifting injury at work. He complained of left-sided low back and medial shin pain, and intermittent paraesthesia affecting his left great toe. The pain was aggravated by flexion and eased by standing and walking. On examination he had a marked shift to the right. Flexion was reduced to fingertips to knees and his left SLR was reduced to 50 degrees.

Owing to the rapid onset of symptoms associated with a lifting injury in a flexed posture, and the pain being aggravated by flexion and eased by extension activities, the injury was hypothesised to be discogenic. Clinical trials suggest that the most usual sources of low back pain are the intervertebral disc, the zygapophyseal (facet) joint and the sacroiliac joint (Maitland 2001).

The patient was treated by rotations to the right (as demonstrated on another patient in Figure 11.22), which centralised his pain. His shift was manually corrected on the first visit. He was prescribed repeated extension exercises in prone, as advocated by McKenzie (1985), to do at home every two hours. By the third visit his pain had centralised to left low back pain and his SLR was 80 degrees. He was then treated by unilateral mobilisations on the left at grade 4. This alleviated his symptoms and he regained full range of all movements.

Prior to discharge, he was given a programme of abdominal and multifidus exercises. He was also given postural and ergonomic advice prior to his return to work. The multifidus and the transverse abdominus muscles have been found to be primarily responsible for imparting local stability to the lumbar spine in the joint's neutral zone (Panjabi 1992; Goel et al. 1993; Wilke et al. 1995; Hodges and Richardson 1996).

Figure 11.22 Right-sided lumbar rotation, used to treat left-sided back and leg pain.

Fundamental differences between acute and chronic pain

It is not satisfactory to view chronic pain simply as acute pain that has persisted for a long period. Acute and chronic pain are different clinical entities. We now acknowledge that there are many different mechanisms and processes involved in the genesis of chronic pain states which are not relevant in acute pain. The diversity of these elements has led to the use of a biopsychosocial approach to indicate that there are biological, social and psychological factors relevant within an individual that can be either causing or maintaining the chronic pain state

Acute pain is basically a protective mechanism to reduce the possibility of increasing the injury. It is usually self-limiting, and lasts until the tissues are healed. It is usually associated with an increased sympathetic nervous activity. This may be associated with feelings of anxiety, panic, nausea, etc. and may be observed during traumatic injuries to the bones and soft tissues.

Chronic pain is detrimental because it lasts long after the injury has healed. Tonic self-sustaining neural loops are set up to perpetuate the pain. Decreases in sympathetic activity may cause depression and apathy. Chronic pain outlasts the normal time of healing and has no recognisable end-point (Grichnik and Ferrante 1991).

Predictors of chronic incapacity

Can disability from low back pain be predicted? Psychosocial researchers in the last decade have found common psychosocial and social traits in people who have developed chronic disability owing to LBP. Many subjects with

chronic LBP have been reported to have a psychological profile that predisposes them to develop chronic pain (Burton et al. 1995; Carrageen 2001). Additionally, people aged between 50 and 60 years are more likely to become disabled because of LBP (Burton et al. 1995).

Further major predictors are listed here.

- People who have unrealistic beliefs about their pain and the nature of their disease (Waddell 1992).
- People whose occupation involves heavy manual work and sustained postures.
- People with a previous history of sickness absence.
- People who seek multiple investigations and treatments (Waddell 1992; Harding and Watson 2000).
- People with low educational achievement or low-status occupations (Cats-Baril and Frymoyer 1991; Frymoyer 1992).
- People who have pending compensation issues (Tait and Chibnall 2001).
- People with fear-avoidance beliefs, that a fear of activity may be more disabling than the original injury (Vlaeyen 1995; Fritz et al. 2001).
- People who exhibit 'illness behaviour', which may include attention seeking, grimacing, catastrophising about their problems or LBP, inappropriate coping strategies, excessive use of splints, braces, walking aids, over-reliance on the NHS, and passive rather than active treatment modalities.

In view of the above factors, it is necessary to screen patients with LBP in order to attempt to reduce the likelihood of chronic disability. It is important to note that a patient's general physical fitness may be a poor predictor of chronic incapacity (Deyo and Weinstein 2001). The identification of the patients at risk of progression to chronicity (failure to respond to treatment) is by means of psychosocial questionnaires, because clinical variables contribute practically nothing to our predictive ability (Burton et al. 1995). The psychosocial traits concerned (coping strategies, depressive tendencies, inappropriate beliefs about pain and activity) are present in the acute phase – they are not just the result of persistent symptoms (Burton et al. 1995).

Management guidelines for LBP (CSAG 1994; RCGP 1999)

In the early management of acute LBP, analgesia with NSAIDs should be administered immediately following an acute onset. Manipulative therapy is advised in the early stages, and active exercise and physical activity should be encouraged. Bed rest is ineffective as treatment for back pain, but is acceptable in moderation in the acute situation for 1–2 days.

Encourage physical activity and an early return to work and sport whenever possible. Practise psychosocial management: educate the patient about the importance of good postural habits and activities of daily living. Challenge the patient who has unrealistic beliefs about the LBP condition and prognosis.

Biopsychosocial assessment

The biopsychosocial assessment differs from the physical assessment in that it incorporates psychological and social issues in more depth. This gives the physiotherapist a good overview of the patient's circumstances, his or her overall mood state, beliefs, attributes and thoughts about the problem, about therapy and about the future.

Psychological factors

A previous history of anxiety and depression, general attitudes and expectations is noted. The patient's perceived level of control over the pain is also assessed with particular regard to the use of active or passive coping strategies (these are often referred to as 'internalised' or 'externalised' locus of control).

Social factors

Identify social areas, including work issues, pending compensation, a history of injury, sickness benefits, and daily functioning, as these may affect the outcome.

Physical examination

A body chart and physical examination may or may not be conducted, as the physiotherapist deems necessary. However, if red flags are noted then a neurological examination is indicated. Assessments may include functional tests such as:

- the distance that can be walked in five minutes;
- the number of times the person can stand from sitting in one minute;
- the number of times the person can step up and down in one minute.

Flags

'Yellow flags' are psychosocial factors that include a previous history of anxiety and depression, impending compensation, absence from work, sickness benefit, invalidity benefit, passivity, and high levels of dependency and poor coping skills. 'Red flags' are clinical features that should alert the therapist to the possibility of severe pathology. They include bladder and bowel malfunction, saddle anaesthesia, bilateral paraesthesia, neurological signs, unexplained weight loss, a past history of carcinoma, general debility and fever.

Outcome measures questionnaires

The following questionnaires and tools are validated, reliable and sensitive. They can be used prior to, and following, intervention to determine efficacy.

- The Oswestry Disability Index (Fairbank et al. 1980).
- The VAS.
- The Present Pain Index.
- The short-form McGill Pain Questionnaire (Melzack 1987).
- The Hospital Anxiety and Depression Questionnaire (Zigmond and Snaith 1983).
- The locus of control questionnaire (FABQ).

Treatment of biopsychosocial aspects of LBP disability

Many studies have reported decreases in pain levels and disability following intensive back rehabilitation programmes combining exercise and cognitive therapy (e.g. Frost et al. 1998; Guzman et al. 2001). The aims of chronic spinal rehabilitation programmes are to:

- reduce the patient's pain, if possible, or enable the patient to cope more effectively with the pain;
- reduce the patient's disability;
- encourage, when possible, a return to work and hobbies to promote better physical functioning (by challenging the unhelpful belief that pain always equates to harm);
- encourage an active, patient-centred approach to LBP management.

Van Korff and Saunders (1996), in a survey of LBP sufferers in the USA, found that patients wanted to understand the following four things.

1. The likely course of their back problem.
2. How to manage the pain.
3. How to return to normal activities of daily living.
4. How to minimise recurrences of back pain.

Example of the content of a back rehabilitation programme

- Pre-intervention questionnaire and physical tests for outcome measures.
- Circuit training, including aerobic and strengthening regimes, with emphasis on postural control, spinal stability, back extensors and deep abdominal musculature.
- Patient-centred discussions, seminars on anatomy, pathology, medication, self help measures, posture and exercise, etc.
- Relaxation workshops.
- Post-programme questionnaire and physical tests.
- Follow-up questionnaires at 1 and 12 months to determine how they are managing their LBP.

THE CERVICAL SPINE

Following on from the subjective assessment, the physiotherapist should highlight the main findings and formulate a hypothesis regarding the clinical diagnosis. The SIN factors will determine the vigour of the examination. The physiotherapist will attempt to find the patient's comparable sign by means of movement or palpation.

Clinical note

Patients presenting with disorders of the cervical spine may also complain of headaches, dizziness, nausea and vertigo. Record this in your assessment. These may be symptoms of vertebrobasilar insufficiency (VBI).

Posture

Note the symmetry of the head on the neck, and the neck relative to the thorax. The chin should be at 90 degrees to the anterior aspect of the neck. There should be no obvious horizontal skin creases posteriorly. A plumbline from the tragus of the ear should fall behind the clavicle.

Assess the cervical lordosis. A decreased lordosis predisposes the vertebral bodies and discs to bear more weight. An increased lordosis increases the compressive loads on the zygapophyseal (facet) joints and posterior elements. Observe for muscle hypertrophy, hypotrophy, spasm, tightness or general asymmetry.

An acute wry neck (torticollis) presents as a combination of flexion and rotation or side flexion away from the painful side. Patients with chronic pathology often have a poking chin posture which consists of excessive upper and middle cervical extension and lower cervical/cervicothoracic flexion. This results from a weakness of the deep cervical flexors and overactivity of sternocleido-mastoid and levator scapulae muscles (Figure 11.23).

Note that cervical posture is influenced by lumbar posture and, hence, the poking chin posture is exaggerated by lumbar and thoracic flexion (Figure 11.23). Cervical and shoulder posture should, therefore, be viewed in both the sitting and standing postures.

The shoulders should, ideally, be level, but this is often not the case because of handedness. For example, in a right-handed person the right shoulder is often held slightly lower than the left.

Movements

It is important to not only assess the range of movement occurring in the cervical region but also the quality of that

Figure 11.23 (a) Poking-chin posture, exaggerated by thoracic and lumbar flexion. (b) Correction of cervical posture by maintaining the lumbar spine in a neutral position.

movement. Note, in particular, the motion segments where the movement is occurring. Hinging may be observed, which indicates areas of hypermobility or instability. Conversely, areas of hypomobility or stiffness are observed as areas of plane or straight lines.

Key point

For consistency and reliability of reassessment, the same order of active movements should be carried out each time.

Active movements

Flexion

The movement should be performed to either the patient's pain or the limit of movement. During flexion the cervical lordosis should be obliterated and the spine appears to be flexed or neutral. The spinous process of C7 should be the most prominent – C6 and T1 less so. The chin should approximate the chest. Common faulty patterns are the upper cervical spine remaining in extension or chin poke. Loss of range, areas of give and restriction should be noted, as well as the pain response, muscle spasm and crepitus.

Extension

The entire cervical spine should extend, and the face should be almost parallel to the ceiling. A vertical line should be observed from the chin to sternum. Common faulty movement patterns include a loss of lower cervical extension and the head does not move posteriorly to the shoulders. Furthermore, excessive hyperextension of the upper and mid cervical spine may occur earlier on in the movement and the chin pokes forward.

Figure 11.24 (a) Normal right cervical rotation. (b) Faulty position: right side flexion limited and completed with contralateral rotation. (c) Faulty position: extension with chin poke, upper cervical extension, lower cervical spine remaining flexed.

Side flexion

Often this movement is the most restricted in degenerative spinal pathologies. Tightness in the contralateral sternocleidomastoid and trapezius may be observed. Common faulty patterns include coupling with rotation owing to tightness in anterior flexor musculature. Observe range, pain response and areas of give or restriction. Compare the sides for symmetry.

Right and left rotation

Observe the range of movement available and the patient's pain response, muscle spasm and crepitus. Common faulty patterns include coupled movements with side flexion and the eyes not moving in a purely horizontal plane (Figure 11.24). Compare the right and the left sides.

Overpressures repeated and combined movements

If the plane movements are full range and pain-free, then overpressure may be applied. At the end of the available range the physiotherapist may apply a small oscillatory movement to feel the quality and end-feel of the movement, and the range of further movement. The pain response is also noted. Combined movements may be examined in an attempt to reproduce the patient's pain or restriction of movement. The patient should if possible perform repeated movements, as this may alter the quality and range of the movement and may give rise to latent pain (McKenzie 1990).

The shoulder complex

Observe full shoulder elevation through flexion and abduction for the shoulders bilaterally, because during the last few degrees of elevation the thoracic spine extends to allow for full shoulder elevation. A stiff kyphotic thoracic spine will limit shoulder elevation.

Passive physiological intervertebral movements (PPIVMs)

PPIVMs may be used to confirm any restriction of motion seen on active movement and to detect restriction of movement not discovered by the active movements. They are also used to detect segmental hypermobility (Magarey 1988; Maitland 2001).

Vertebral artery testing

See the online version of the textbook for a detailed account of how to perform these tests.

Key point

Vertebrobasilar insufficiency (VBI) is a contraindication to cervical manipulation and high–grade mobilisations, particularly rotations, extensions or longitudinal distractions and traction.

Differentiation test to determine between vestibular and VBI symptoms

The patient stands and rotates the cervical spine to the right and left. A note is taken of symptoms such as dizziness or nausea. Either vertebrobasilar artery insufficiency or the vestibular apparatus could cause symptoms produced as a result of this manoeuvre. In order to differentiate between these two structures, the physiotherapist fixes the patient's head and the patient keeps the feet static and facing forwards. The patient then rotates his or her body to the right and to the left while maintaining the head in a static position.

If symptoms such as dizziness or light-headedness are produced with this manoeuvre then they are a result of vertebrobasilar pathology, because the head remains still (and the vestibular apparatus will be unaffected) and the cervical spine is rotating affecting the artery.

Vertebrobasilar testing (Maitland 2001)

This test is performed in both sitting and supine positions.

1. Sustained rotation for ten seconds is performed to each side. Note any symptoms.
2. Sustained extension for ten seconds is performed. If the patient is asymptomatic then:
3. Combined rotation and extension to each side is sustained for ten seconds. Note any symptoms. A positive test would be to induce feelings of dizziness and nausea.

Key point

If the patient has grossly restricted range of movement then the test for VBI is not valid.

Figure 11.25 (a) Resisted shoulder abduction (C5) myotome. (b) Assessment of C6 myotome in resisted biceps.

Neurological testing

Dermatomes

Test for normal sensation, the cutaneous area supplied by a single posterior root of each spinal segment, light touch with the dorsal aspect of the hand or cotton wool and pinprick sensation for each dermatome, C1 to T1.

Myotomes

Isometric testing of the muscles supplied by a spinal segment in mid range for a few seconds is performed at each level from C1 to T1 (Figure 11.25). Weakness may indicate a lower motor lesion from a prolapsed disc or another space-occupying lesion.

Reflexes

Test for normal reflexes: biceps (C5–C6), triceps and brachioradialis (C7). Compare one side with the other. Note brisk reflexes which may be indicative of an upper motor neurone lesion and dull reflexes which may be indicative of a lower motor neurone lesion.

- C5–C6 correspond to biceps brachii. The person's arm should be semi-flexed at the elbow with the forearm pronated. Place your thumb or finger firmly on the biceps tendon and hit your finger with the hammer (Figure 11.26).
- C6–C8 correspond to triceps. Support the person's upper arm and let the forearm hang free. Hit the triceps tendon above the elbow (Figure 11.27).

Figure 11.26 The biceps reflex (C5, C6).

Figure 11.27 The triceps reflex (C6, C7).

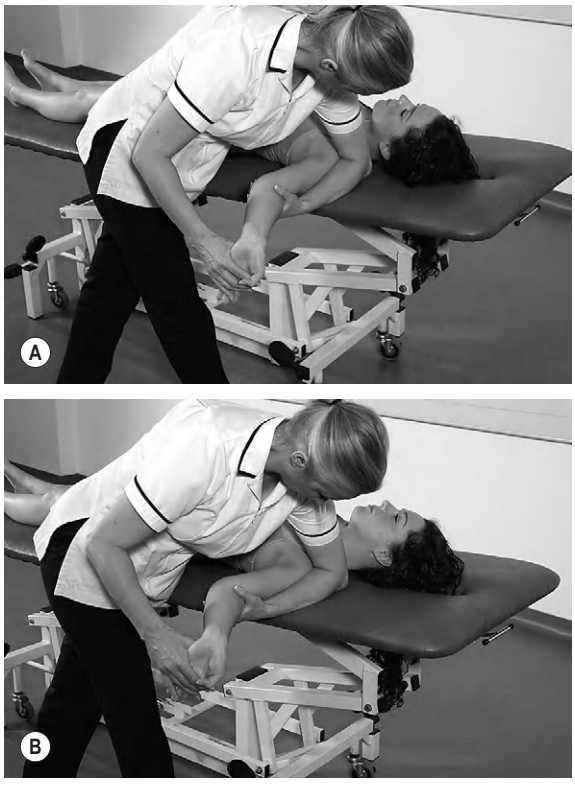


Figure 11.28 (a) Upper limb tension test (ULTT). (b) ULTT with contralateral right side flexion of the cervical spine.

Mechanical tension tests

The upper limb tension test (ULTT) is referred to as the SLR test of the cervical spine. This test mobilises the brachial plexus and particularly biases the median nerve to determine the degree to which neural tissue is responsible for producing the patient's symptoms. Certain movements of the arm, shoulder, elbow, wrist and hand, and, similarly, the neck and the lower limb, can cause neural movement in the cervical spine. These tests are so important that all physiotherapists should know and use them (Butler 1991).

The physiotherapist depresses the patient's shoulder, then adds in 90 degrees abduction, 90 degrees lateral rotation of the shoulder, elbow extension, forearm supination, and wrist and finger extension to the supine patient (Figure 11.28a). Sensitising manoeuvres such as ipsilateral (same side) or contralateral (opposite side) cervical rotation and side flexion are added (Figure 11.28b). Symptoms of pain, paraesthesia and restriction are noted and compared with the other side. Common findings will be reduced range or the reproduction of symptoms on the affected side.

Palpation

Palpate the soft tissues, noting the positions of vertebrae and myofascial trigger points (localised irritable spots within skeletal muscle). These trigger points produce local pain in a referred pattern and often accompany chronic musculoskeletal disorders. Palpation of a hypersensitive nodule of muscle fibres of harder than normal consistency is the physical finding typically associated with trigger points (Alvarez and Rockwell 2002).

Observe for local or referred pain, thickening of structures or stiffness. Remember that anomalies of the bifid spinous processes of the cervical vertebrae and differences in their spacing are not uncommon and may not be clinically significant (Maitland 2001).

Soft-tissue changes, including sub-occipital thickening and shortening in the extensors and prominence, and thickening of the articular pillar of C2–C3 facet joints, are common in degenerative disorders. Soft-tissue changes around the cervicothoracic junction are also commonly found and may be referred to as a Dowager's hump.

Bony anomalies

Osteophytes may be palpable at the C2–C3 facet joints in patients with pre-existing spinal pathology (Maitland 2001). Approximation of the spinous processes of C6–C7 is also a common feature.

Accessory spinal movements

With the patient prone, central pressure on the spinous processes C2–T6 and unilateral pressures on the articular pillars C2–T6 is applied by the physiotherapist, noting levels of stiffness, pain response, muscle spasm and areas of hypermobility (Figure 11.29).

Figure 11.29 Accessory movements: (a) central posteroanterior pressure on spinous process; (b) central posteroanterior with thumbs superimposed; (c) unilateral pressure on the left articular pillar.

CASE STUDY

CERVICAL SPINE

A 50-year-old woman with a 2-year history of central neck and occasionally bilateral shoulder pain (4 on the VAS) was referred for physiotherapy. On examination she had a marked protracted cervical spine (poking chin posture) which could, however, be corrected by the patient on demand. Her cervical range of movement was approximately two-thirds on all movements. Neurological testing was normal. Palpation revealed stiffness at levels C5 and C6 to central posteroanterior pressures over the spinous processes.

The patient was treated with grade III posteroanterior central pressures (as in Figure 11.29) and was given postural correction exercises. Priority one cervical neutral shin slides against a wall. Following three treatment sessions her pain was reduced to 1 on the VAS and her range of movement was almost full. She was given deep flexor exercises on the fourth visit, along with exercises for normal range of movement. On the fifth visit she was asymptomatic and was discharged to continue with the exercises at home.

Both manipulations and mobilisations aim to reduce pain and increase the joint range of motion for spinal conditions (Johnson and Rogers 2000; Maitland 2001). Studies have shown that cervical mobilisation produces a hypoalgesic (pain-relieving) effect and can decrease visual analogue scores (Sterling et al. 2001). The majority of studies conclude that spinal mobilisations have a positive short-term effect on pain (Coulter 1996; Bronfort et al. 2001). Koes et al. (1992) observed an improvement in physical functioning when compared with other physiotherapy modalities, placebo or GP involvement. It could be argued that by improving pain in the short term the patient will return to normal activities more quickly and thus avoid the potential for deconditioning and chronicity.

Muscle imbalance procedures were instituted into the woman's programme to improve the patient's postural awareness, to increase the strength of the deep cervical flexors and to stretch out the tight sub-occipital extensors. According to Heimeyer et al. (1990) it is important for the therapist to differentiate between people who have protracted chin posture but who can voluntarily correct it and those who cannot. For the person in this case study, the posture was correctable, so the role of the physiotherapist as advisor was paramount. Repeated sessions of electrotherapy, for example, would have been inappropriate or would have provided only a short-term solution to the pain. Treatment should be geared towards behaviour modifications – the change of bad postural habits. In this case, the combination of manipulative therapy, corrective exercises and advice was employed with success.

Peripheral joint assessments

THE SHOULDER JOINT

Key point

The patient should be suitably undressed to view the cervical spine, thoracic spine, shoulder girdles, shoulders and both arms.

Posture

It is important to assess the posture of the cervical and thoracic spine because a scoliosis, kyphosis or poking chin posture will affect the mechanics of the shoulder by altering the plane of the glenohumeral joint. The spinal and shoulder complex postures should be observed with the patient in both sitting and standing positions.

Posterior alignment

The shoulders should ideally be level, but for a right-handed person the right shoulder is often held lower than the left and vice versa. Elevation of the shoulder girdle may be a result of tightness or over-activity in the levator scapulae or the upper fibres of trapezius, and lengthening or weakness in the lower fibres of trapezius.

Observe the symmetry of the scapulae. They should lie flat against the thoracic wall and the medial borders should lie approximately 50–75 mm lateral to the spine. Winging of the scapulae is observed when the whole length of the medial border of the scapula is displaced laterally and posteriorly from the wall of the thorax. This may result from weakness in the serratus anterior muscle or a lesion of the long thoracic nerve. Pseudo-winging of the scapulae occurs when the inferior angle of the scapula is displaced from the thoracic wall.

Observe the soft-tissue contours of the shoulder for symmetry, and areas of atrophy and hypertrophy. The acromion processes should be horizontal to, or slightly higher than, the point at the root of the scapula. If the root of the scapula is higher this indicates tightness or over-activity of the levator scapulae and rhomboid musculature, which causes a downward rotation of the glenoid fossa. This may be a precursor to impingement syndromes and rotator cuff pathologies. Moreover, the levator scapulae may cause anterior shear on the cervical spine and give rise to cervical and scapula pain.

Anterior alignment

Note any irregularities of the clavicle, sternoclavicular and acromioclavicular joints resulting from previous fractures or dislocations. Note the soft-tissue contours regarding symmetry atrophy and hypertrophy, particularly in the deltoid, upper trapezius and sternocleido-mastoid muscles.

Lateral alignment

Note the relative positions of the humeral head: no more than one-third of the humeral head should lie anteriorly to the acromion process. Excessive forward translation may result from tightness in the pectoral muscles and elongation of the posterior shoulder capsule. The patient's arms should lie comfortably at the side with the thumbs facing almost forwards. Excessive medial rotation of the shoulders will result in the thumbs facing inwards towards the body. Excessive protraction of the shoulders with an increased thoracic kyphosis and tightness in the pectoral muscles is a common faulty posture.

Palpation

Palpate the local skin temperature, noting any increase suggestive of underlying inflammation. Palpate the acromioclavicular and sternoclavicular joints, observing for pain or tenderness. Palpate the supraspinatus and infraspinatus tendons for tenderness associated with tendonitis, calcification and strain. Palpate the upper trapezius and levator scapulae for tenderness and trigger points. These hyperirritable areas within the muscle and connective tissue are thought to be caused by a secondary tissue response to disc or joint disorders (Hubbard and Berkhoff 1993). They are painful to compression and may cause referred pain.

Muscle length tests

Levator scapulae

With the patient supine, flex and lateral flex the cervical spine away to resistance and add ipsilateral rotation (rotation to the same side as side flexion). Depress the shoulder girdle and compare range and pain response on both sides.

Pectoralis minor

With the patient supine, the lateral border of the spine of the scapula should be within 25 mm of the plinth. If pectoralis minor is shortened, the lateral border of the spine of the scapula is more than 25 mm from the plinth since the shoulder girdle is protracted.

Movements

Clinical note

As the cervical and thoracic spines may refer pain to the shoulder and scapula areas, full active movements and accessory movements of these areas should be assessed. Note any increase or referral of pain around the shoulder or scapula areas. Over-pressure may be used if the movements are pain-free. Furthermore, the ULTT may be performed to rule out referral of pain from neural structures. Refer to the objective assessment of the cervical spine for descriptions of these techniques.

Active movements

Full active movements of the shoulder girdle and joint are performed, noting any restriction, asymmetry and pain response.

Shoulder girdle movements

Assess shoulder girdle elevation, depression, protraction and retraction, observing for pain asymmetry and crepitus.

Shoulder joint flexion

Observe flexion through to elevation and return of movement, assessing the scapulohumeral rhythm. Normal should be in the ratio of 2 : 1 (humerus : scapula). Reversed scapulohumeral rhythm occurs in conditions causing

Figure 11.30 Reversed scapulohumeral rhythm of the right shoulder.

restriction of the glenohumeral joint, such as adhesive capsulitis ('frozen shoulder').

Shoulder joint abduction

Observe abduction through to elevation and return (Figure 11.30), again noting the scapulohumeral rhythm.

Key point

A painful arc of movement is observed in patients suffering from impingement syndromes, whereby the superior aspects of the rotator cuff, biceps tendon and bursae are impinged by repetitive overarm activities. Pain is experienced between 90 and 130 degrees of abduction.

Impingement may be caused by loss of scapular stability. Faulty patterns of scapula motion include early rotation and elevation of the scapula (reversed scapulohumeral rhythm). This may implicate weakness in the stabilisers (e.g. lower fibres of trapezius, rhomboids and serratus anterior) or shortness and over-activity in the upper trapezius and levator scapulae.

Impingement may also be caused by weakness or inhibition of the rotator cuff muscles that produces a superior translation of the humeral head (i.e. subscapularis, teres minor and lower infraspinatus). There may also be late timing of lateral rotation during abduction which may cause impingement.

Tightness of pectoralis minor can cause increased protraction of the scapula which decreases the subacromial space.

Repetition of the movement may induce an element of fatigue and abnormal movements may derive from that. A juddering movement of the scapula on return from elevation implicates poor eccentric control.

Differentiation. If abduction reproduces the person's pain, then differentiation between the glenohumeral and subacromial structures may be required.

1. If the movement is repeated and compression applied to the glenohumeral joint causes an increase in symptoms, then the glenohumeral joint is implicated.
2. If the movement is repeated and a longitudinal force in a cephalad direction is applied (increasing compression on the subacromial structures), with an increase in pain, the subacromial structures are implicated.

Failure to initiate or maintain abduction when placed passively into abduction is a sign of rotator cuff rupture and the patient should be referred to a consultant for a repair/further investigations.

Shoulder joint rotation

Test medial and lateral rotations, both beside the trunk and at 90 degrees of abduction. Note the pain response and limitation of movement.

Shoulder joint horizontal flexion and extension (scarf-test)

Pain on these movements implicates the acromioclavicular joints as the source of pain or restriction.

Clinical note (Figure 11.31)

The medial kinetic rotation test assesses movement dysfunction and impingement and instability risk at the shoulder.

The patient is supine with the arm at 90 degrees of abduction and off the plinth. The therapist stands above the patient's right shoulder facing his/her feet and places his/her left index finger on the patient's humeral head and the left middle finger on the coracoid process. She/he passively medially rotates the patient's shoulder using the forearm to 70 degrees.

In an ideal situation, or negative test, the therapist's fingers on the left hand should not move while rotating the shoulder joint medially to 70 degrees.

Early movement of the humeral head (index finger) suggests an anterior instability of the glenohumeral joint.

Early movement of the coracoid (middle finger) suggests scapula instability and a potential impingement risk at the glenohumeral joint.

Shoulder joint extension

Compare both sides for range and pain response.

Shoulder functional movements

Functional movements, such as the hand behind the back (HBB), and the hand behind the neck (HBN), should also be assessed. These movements are grossly restricted in patients with adhesive capsulitis.

Other shoulder joint abnormalities

Sporting activities that give rise to symptoms, such as the late cocking stage of throwing a ball overhead, should also be assessed to determine faulty mechanics.

Passive movements

All movements performed actively can be repeated passively, noting the differences in range. Observe the differences in end-feel and compare these with the unaffected side.

Figure 11.31 Medial kinetic rotation test.

Key point

Note that the capsular pattern for the glenohumeral joint is limitation of lateral rotation, abduction and medial rotation (Cyriax 1982).

Accessory movements

Acromioclavicular and sternoclavicular joints

Test anteroposterior (AP) and posteroanterior (PA) draw, and caudal glide.

Glenohumeral joint

Test anteroposterior (AP) and posteroanterior (PA) draw, caudal and cephalad glide, and lateral distraction (Figure 11.32).

Further tests (Maitland 2001)

The following tests should be performed only in shoulders of low irritability and when no comparable sign has been found. They stress a number of different structures around the shoulder and are therefore not diagnostic:

- locking test of the shoulder;
- quadrant test of the shoulder (Figure 11.33).

Resisted muscle testing

This provides a guide of strength ratios and pain response. Test: abduction and flexion at around 30–60 degrees; internal and external rotation beside the trunk (Figure 11.34) and at 90 degrees of elevation in the plane of the scapula; and resisted muscle testing in positions of function and/or pain.

Muscle length tests

It may be useful to test the length of muscles that are prone to shortness – latissimus dorsi, pectoralis major and minor, upper fibres of trapezius, levator scapulae and sternocleido-mastoid.

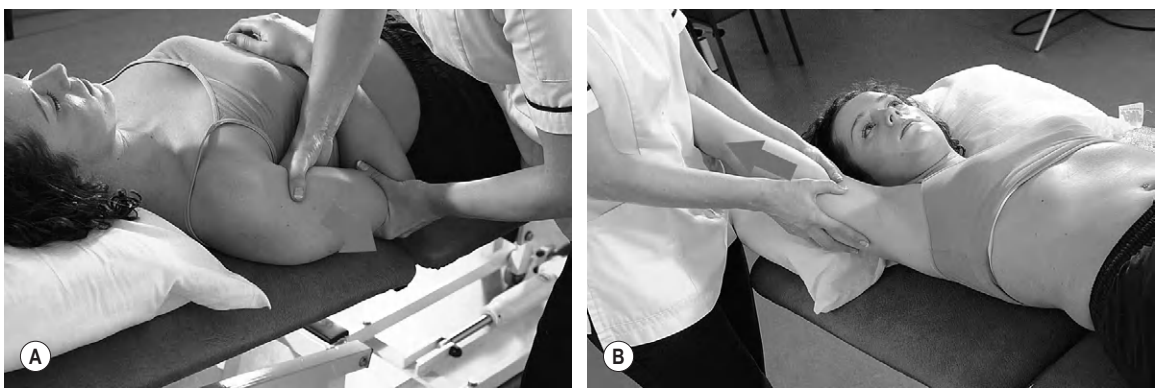


Figure 11.32 (a) Lateral glide of the glenohumeral joint. (b) Caudal glide of the shoulder in elevation.

Figure 11.33 Quadrant test of the shoulder.

Figure 11.34 (a) Resisted lateral rotation at the shoulder. (b) Resisted medial rotation at the shoulder.

Acromioclavicular joint compression and distraction

End-of-range overpressure into horizontal flexion compresses the acromioclavicular joint and may give rise to pain arising from this joint. Acromioclavicular distraction tests the instability of the acromioclavicular joint by applying a downward traction on the arm while palpating the joint line. Reproduction of pain or palpable separation of the joint line is a positive test.

Figure 11.35 Anterior draw of the glenohumeral joint in abduction with stabilisation of the shoulder girdle.

Tests for shoulder instability

Anterior draw/translation (Lachmann's of the shoulder)

This is performed in supine with the patient's arm at around 30 degrees of abduction, 45 degrees of lateral rotation and slight flexion. The physiotherapist grasps the humeral head with one hand and the medial hand is used to stabilise the shoulder girdle. The lateral hand applies the anterior translation force in the same way as the anterior draw test of the knee. Laxity of the joint (excessive anterior translation) is a positive sign (Figure 11.35).

Posterior draw test

This is performed in supine with the patient's glenohumeral joint at the edge of the examination couch in abduction not exceeding 90 degrees. Posterior pressure is applied on the anterior aspect of the humeral head. Excessive movement compared with the other side is a positive sign.

Inferior draw (sulcus) test (Figure 11.36)

This is performed in sitting or supine, arms by the sides. The physiotherapist exerts a strong downward traction force on the arm, grasping the head of the humerus with both hands, to the limit of movement, pain or apprehension while monitoring the superior contour of the shoulder joint. Excessive inferior glide or a significant depression or sulcus distal to the acromion is a positive sign.

Figure 11.36 Inferior draw (sulcus) test. *Courtesy of Nina Dean and Gemma Dickinson.*

Figure 11.38 Palpation of the supraspinatus tendon. The patient's hand is behind the back.

Figure 11.37 The empty-can test position. Resistance to abduction is applied by the therapist.

Medial kinetic rotation test

The shoulder is positioned and supported in 90 degrees abduction and passively medially rotated to 70 degrees. The therapists index and middle fingers are placed on the corocoid process and anterior humeral head. Anterior translation of the corocoid before 70 degrees rotation may suggest scapula instability whilst anterior translation of the humeral head before 70 degrees rotation may indicate glenohumeral instability.

Impingement test

Supraspinatus (empty-can test)

This is performed in sitting or standing with 90 degrees of abduction bilaterally, full available medial rotation and 30 degrees of horizontal flexion (Figure 11.37). Supraspinatus is the main support for the suspended arm in this position. The physiotherapist resists abduction of the shoulder. Pain on resistance is a positive test for a lesion of the supraspinatus muscle or tendon. Following the objective assessment, record your findings clearly and asterisk objective findings.

Test yourself

Match these five scenarios to the likely pathology.

1. Reduced range of movement particularly on active and passive rotations and abduction.
2. Painful arc of movement between 90 and 120 degrees.
3. Inability to actively abduct the arm away from the body and maintain the position when the arm is placed there passively.
4. Pain and weakness on resisted elbow and shoulder flexion.
5. Excessive movement on passive anterior, posterior and sulcus draw tests of the shoulder.

Answers

(1) Frozen shoulder (adhesive capsulitis). (2) Impingement of supraspinatus under the acromion. (3) Rupture of the rotator cuff musculature. (4) Ruptured biceps brachii muscle. (5) Global instability.

THE HIP JOINT

Gait

Observe the person's gait from the front, back and side. Assess the patient with and without a walking aid, as

Key point

The patient should be suitably undressed to view the hip, pelvis and spine. Note that the hip joint is too deep to observe an effusion or palpate the joint line.

deemed appropriate. Ask the patient to walk forwards and backwards, while observing:

- stride length symmetry;
- the time spent on the single leg support phase on each leg;
- corresponding factors of pain, stiffness and/or weakness during the cycle.

Posture

Standing

With the patient standing, view from the front, rear and sides. Note:

1. Pelvic tilting: a line joining the two anterior sacroiliac joints should be horizontal (the same applies posteriorly).
2. The relative levels of the posterior superior iliac spine to the ipsilateral anterior superior iliac spine viewed from the side: differences may be suggestive of sacroiliac rotatory asymmetry.
3. Rotational deformity of the hips: this may be observed as in-toeing or out-toeing.
4. Leg length discrepancy: this may be observed by the differences in the horizontal levels of the gluteal and knee creases.
5. Scoliosis of the lumbar spine: this may be structural or a compensation for a leg length discrepancy.
6. Inequality of weight distribution: the patient may reduce the amount of weight borne on the painful side.
7. Increased lumbar lordosis: this may suggest a fixed flexion deformity of the hip(s).
8. Bruising in the abdominal or groin area: this is suggestive of a sportsman's hernia.
9. Muscle wasting: wasting, particularly of the quadriceps and gluteal muscles, is common and may appear as hollowing posteriorly or laterally at the buttocks.

Supine

With the patient supine, note the following.

1. Leg rotation, through observing the relative positions of the patella and/or the feet.
2. Pelvic rotation.
3. Leg length discrepancy, through observing the relative position of the medial malleoli or heels.

Leg length discrepancy

Apparent leg length discrepancy is measured from the xiphoid of the sternum to the tip of the medial malleolus using a tape measure (compare with the other leg). *True leg length discrepancy* is measured – using a tape measure – from the anterior superior iliac spine to the tip of the medial malleolus. A difference in leg length of up to 1–2 cm is considered normal by some clinicians. If there is a leg length difference, determine if it is the length of the thigh (hip to knee) or leg (knee to ankle).

Key point

From the movement of supine to sitting, one leg may appear to be longer in supine and shorter in sitting. This is caused by anterior rotation of the innominate bone on the affected side and is a sacroiliac joint dysfunction.

Muscle length assessments

The Thomas test

This test determines the presence of a fixed flexion deformity at the hip. With the patient supine, the hip is fully passively flexed, and the lumbar lordosis is obliterated. If the contralateral (opposite) hip rises off the bed, this indicates a fixed flexion deformity of that hip. This may be owing to tightness or restriction in the capsule, iliopsoas or rectus femoris.

To differentiate between the iliopsoas and rectus femoris as the source of restriction, the patient's knee is passively extended (Figure 11.39). If this results in the patient's hip dropping down into less flexion, then the restriction is in the rectus femoris muscle because by extending the knee an element of stretch has been removed. If the hip is unaffected and remains, in the same degree of flexion, independently of the knee extension, then the restriction is in the iliopsoas muscle. This is measured and recorded.

The length of the following muscles may be tested as they are prone to shortening: quadratus lumborum, tensor fascia lata and the hamstrings.

Modified Ober's test (iliotibial band)

With the patient in side-lying and the uppermost hip fully laterally rotated and the knee joint in unlocked extension, the uppermost leg should drop (adduct) to the plinth (Figure 11.40). A tight iliotibial band would result in the leg not being able to adduct to the plinth.

Piriformis test

With the patient supine, or side-lying, with hip at 90 degrees flexion, adduct maximally to resistance and externally rotate. (Note piriformis is a medial rotator in flexion.) Pain in the buttock or in the distribution of the sciatic

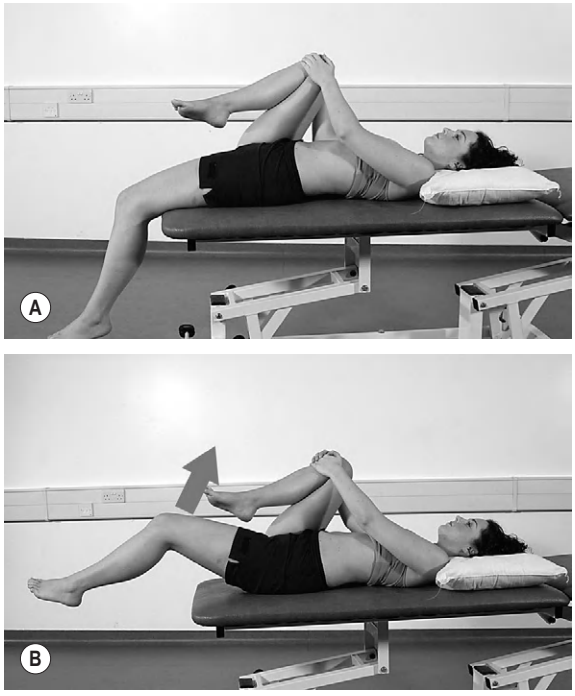


Figure 11.39 The Thomas test: (a) normal; (b) abnormal.

Figure 11.40 Modified Ober's test.

nerve may signify compression of the sciatic nerve by the piriformis muscle (Figure 11.41).

Hamstrings

With the patient sitting with spine in neutral and the hip at 90 degrees, the person should be able to extend the knee to within 10 degrees of full extension (Figure 11.42).

Quadratus lumborum

Test side flexion against a wall without associated flexion or rotations. Compare the two sides. A shortened

Figure 11.41 Piriformis stretch. Note that, in the flexed position, piriformis becomes a hip medial rotator.

Figure 11.42 Assessment of hamstring length. Note that the lumbar spine remains in neutral. This picture shows normal hamstring length.

quadratus lumborum will result in limitation of contralateral side flexion.

Movements

Allow the patient to functionally demonstrate his or her aggravating movement in order to determine the likely structures implicated in producing the symptoms.

Lumbar spine differentiation

The lumbar spine may give rise to referred pain in the region of the hip or groin so it is important to exclude the lumbar spine as a possible cause of symptoms arising at the hip joint. Flexion, extension and bilateral side flexion should be observed actively in standing. Loss of range of motion and pain response should be noted, particularly if these movements reproduce the patient's hip pain or the patient's comparable sign. If the movements are pain-free

and full range, then overpressure may be applied to observe whether this reproduces the patient's symptoms. Accessory movements of the lumbar spine, femoral nerve stretch and SLR should also be screened.

Trendelenberg test

A positive Trendelenburg test demonstrates that the hip abductors are not functioning owing to weakness or pain inhibition, and are unable to perform their role of stabilising the pelvis on the weight-bearing leg. To perform the test the patient stands on the unaffected leg and flexes the other knee to a right-angle. The pelvis should remain level or tilt up slightly on the non-weight-bearing side. The patient then stands on the affected leg and flexes the knee of the other leg. If the pelvis drops on the non-weight-bearing side this signifies a positive Trendelenberg test (Figures 11.43 and 11.44).

Palpation

- Palpate the head of the femur lateral to the femoral artery.
- Rotate the hip passively to elicit crepitus or tenderness at the joint.
- Palpate the psoas major and adductor longus tendons to localise strains and contractures of these structures.
- Palpate the greater trochanter of the femur for tenderness associated with bursitis. Palpate the ischial tuberosity for suspected hamstring strains.
- Tenderness located over the anterior superior iliac spine may indicate a strain of the sartorius muscle or contusion of the spine following contact sports. This is referred to as a 'hip-pointer'.
- Palpate the lower abdominal musculature for suspected inguinal or sports hernias.

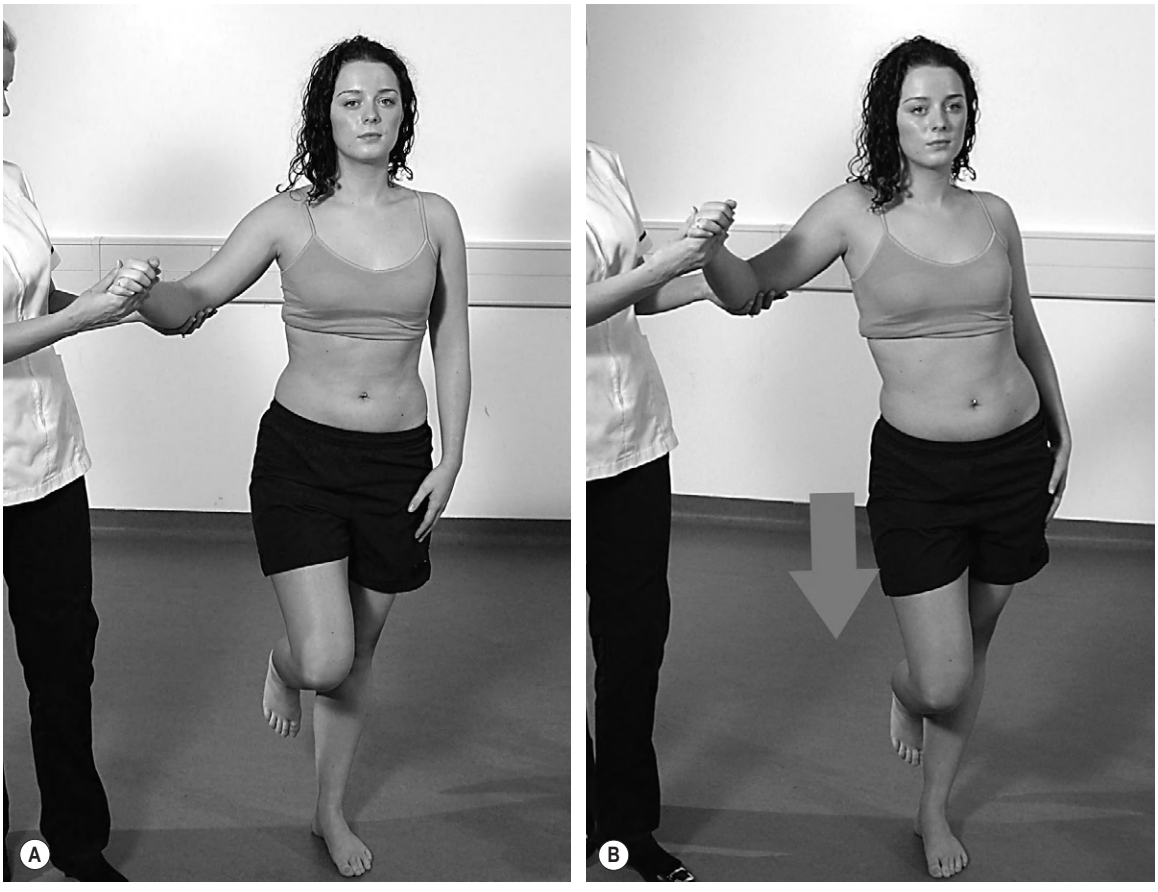


Figure 11.43 Trendelenburg test of right hip abductors: (a) normal; (b) abnormal or positive sign.

Active movements

Key point

Note pain, crepitus and/or limitation of movement. Apply overpressure to the movement if it is pain-free to see whether this reproduces the symptoms not elicited on other movements. Measure both the normal and affected hip for comparison.

Hip flexion/extension

The patient is supine or side lying. The axis of the goniometer is placed directly over the greater trochanter of the femur. The static arm should be parallel to the patient's trunk. The dynamic arm should be placed parallel to the femur. Note loss of range or pain response.

Hip abduction/adduction

The patient is supine. The axis of the goniometer is placed over the anterior superior iliac spine. The static arm should be in line between the left and right anterior superior iliac spine. The dynamic arm should be parallel to the long axis of the femur. Note loss of range or pain response.

Hip rotation

Hip rotation can be easily measured with the patient sitting. Note that the hip joints are approximately 90 degrees flexed (Figure 11.3). The axis of the goniometer is placed at the mid-point of the patella. The static arm is perpendicular to the floor. The dynamic arm should be parallel to the anterior midline of patella. If active movements are full range and pain free then gentle overpressure can be applied noting any reproduction of symptoms.

The normal ranges of movement at the hip joint should be approximately:

- 0–120 degrees for unilateral flexion, 0–150 for bilateral flexion;
- 10–15 degrees for extension;
- 0–40 degrees for abduction;
- 0–25 degrees for adduction;
- 0–35 degrees for medial rotation;
- 0–45 degrees for lateral rotation (Figure 11.45).

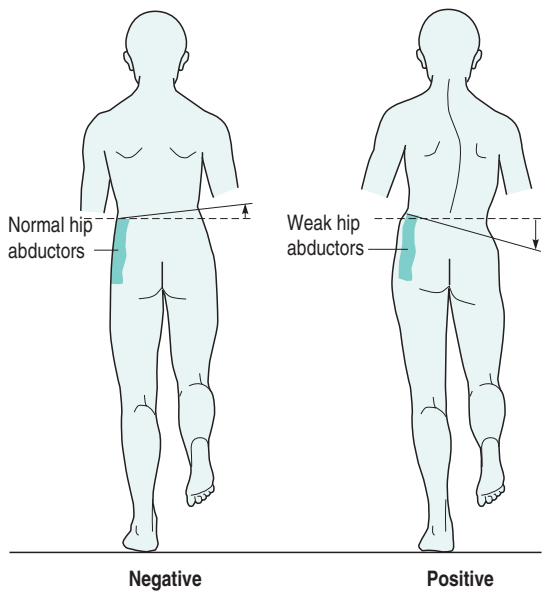


Figure 11.44 Schematic of Trendelburg's sign.

Passive movements

Flexion, extension, abduction, adduction, internal and external rotation should be performed passively by the therapist (Figure 11.46). Note any differences between active and passive ranges and identify reasons for this.

Muscle strength testing

Test the muscles both isometrically and isotonically through range to detect weakness at any particular point in the range. Compare with the opposite side. Pain inhibits muscle contraction and it is therefore important to differentiate between true weakness and pain-induced inhibition.

With the patient side-lying, test the strength of the hip abductors and adductors (weakness of adductors is a common finding in recurrent groin strains). With the patient supine, test the strength of the hip adductors, flexors and medial/lateral rotators (Figure 11.45).

Figure 11.45 Resisted lateral rotation of the hip in flexion.

Figure 11.46 Passive hip and knee flexion.

Figure 11.47 Compression applied through the hip joint.

Accessory movements

Test:

- cephalad longitudinal accessory movement (Figure 11.47);
- caudad longitudinal accessory movement;
- lateral transverse (joint distraction).

Flexion/adduction combination and the hip quadrant test may be performed on non-irritable hips when all plane movements are clear with overpressure (Figure 11.48; Maitland 2001).

Neural tests

Neural tests may be performed if the symptoms produced at the hip appear to be originating from neural or spinal structures:

- femoral nerve stretch test (prone knee bend);
- sciatic nerve stretch test (straight leg raise);
- slump test.

For descriptions of these tests refer to the objective assessment of the lumbar spine.

Figure 11.48 Quadrant test of the hip (flexion and adduction).

Functional tests

Observe the patient performing activities that reproduce their pain. If appropriate, assess activities such as hop, squats, walking forwards, backwards, sideways, etc.

Key point

On completion of the assessment, specific objective signs that reproduce the patient's symptoms should be marked with an asterisk (*) or highlighted. This is commonly referred to as a 'comparable sign' and needs to be reassessed at each treatment to determine the effectiveness of the physiotherapy intervention. Record your findings clearly.

Test yourself

Match these five scenarios to the likely pathology.

1. Local tenderness at the ischial tuberosity and pain on resisted knee flexion.
2. Pain in the groin on coughing, resisted adduction sit-ups and weight-bearing.
3. A three-year history of pain and stiffness particularly on medial rotation.
4. Local tenderness and heat palpated in the area of the greater trochanter having an insidious onset.
5. Increased or exaggerated lumbar lordosis and a positive Thomas test.

Answers

(1) Hamstring strain. (2) Sportsman's hernia.
(3) Osteoarthritic (OA) hip. (4) Trochanteric bursitis.
(5) Tightness in the hip flexors (iliopsoas.)

THE KNEE JOINT

Key point

The patient should be suitably undressed to view the hip, knee and ankle joints.

Gait

Observe the person's gait as they walk forwards and backwards. Make particular note of the equality of stride length, dwell time on each leg, reluctance to bear weight and any pain responses.

Clinical note

The hip joint may refer pain to the knee. To differentiate between the hip and knee joints as a source of symptoms, the hip joint is tested by observing full flexion of the hip joint and then medial and lateral rotation in 90-degree flexion passively. Observe for any pain response or limitation of movement compared with the other side. If the movements are full range and pain-free, it is unlikely that the hip joint is the source of pain (Figure 11.45).

Knee examination

Posture with patient standing

Observe any deformities such as genu varum, genu valgus or genu recurvatum (Figure 11.49). Note any evidence of muscle atrophy, particularly evident in the vastus medialis muscle. Observe the relative positions and size of the patellae. 'Patella alta' is the term used to describe a small high riding patella.

Note any foot, ankle or subtalar deformity, such as foot pronation, which will cause medial rotation of the tibia and, hence, affect the mechanics of the knee joint.

Swelling and discoloration

Swelling that extends beyond the joint capsule may suggest an infection or a major ligamentous injury, and the suprapatellar pouch will appear distended.

Bruising may suggest trauma to superficial tissues or ligaments. Redness of the skin suggests an underlying inflammation. Palpate the temperature around the knee joint with the back of the hand: heat is indicative of an underlying inflammatory disorder.

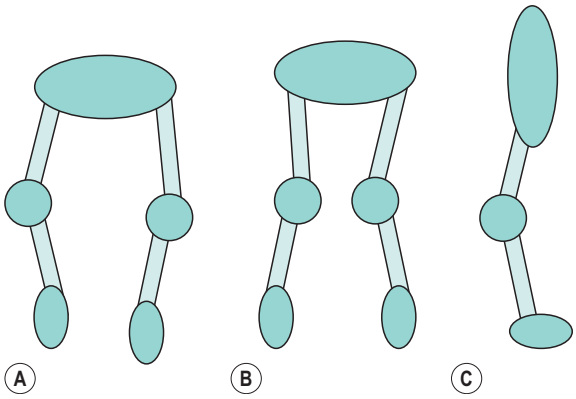


Figure 11.49 Leg posture deformities: genu varum (bow legs); (b) genu valgus (knock-knees); (c) genu recurvatum (hyperextending knees).

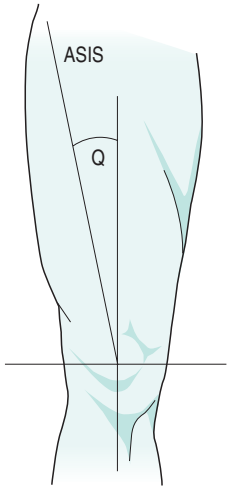


Figure 11.50 Measuring the Q angle.

Clinical note

On your patient, draw a straight line across the middle of the patella. From the centre of this, draw a straight line going downwards through the centre of the tibial tuberosity and another going upwards towards the anterior superior iliac spine (Figure 11.50). Normal values are approximately 12 degrees for males and 15 degrees for females. An increase in the Q angle is a predisposing factor in anterior knee pain and lateral dislocation of the patella.

Clinical note

An effusion (swelling confined within the joint capsule) will appear to obliterate the natural hollows at the sides of the patella. The synovial membrane of the knee is expansive and extends the width of 3–4 fingers above the superior aspect of the patella.

Observe scar tissue that may be indicative of previous surgery or trauma.

Loss of muscle bulk

Observe loss of bulk in the quadriceps muscles, particularly in the vastus medialis which atrophies earlier than vastus lateralis following trauma, degenerative diseases and pain episodes. Measure the circumference of both thighs at 5, 8, 15 and 23 cm above the upper pole of the patella with a tape measure to obtain an objective marker (Magee 1992).

Ask the patient to perform a static quadriceps contraction. Palpate the tone, compare left with right sides of the musculature. Inability to actively extend the knee may result from rupture of the quadriceps tendon or quadriceps weakness, patella fractures, rupture of the patellar ligament, or avulsion of the tibial tubercle. Note any loss of tone in the anterior and posterior tibial muscles and, again, measure, if appropriate, at specific recorded distances below the patella.

Patellar tap

Patellar tap is a simple test to determine the presence of an effusion at the knee joint. It is performed with the patient supine. Any excess fluid is squeezed out of the suprapatellar pouch by sliding the index finger and thumb from 15 cm above the knee to the level of the upper border of the patella (Figure 11.51). Then, place the tips of the thumb and three fingers of the free hand squarely on the patella and jerk it quickly downwards. A 'click' sound indicates the presence of effusion. The test will, however, be negative if the effusion is gross and tense, such as with a haemarthrosis of the knee (blood within the joint) following an anterior cruciate rupture.

Figure 11.51 Patellar tap test.

Fluid displacement test

This is performed as above, by squeezing excess fluid out of the suprapatellar pouch and then stroking the medial side of the knee joint to displace any excess fluid in the main joint cavity to the lateral side of the joint. Repeat this procedure by stroking the lateral side of the joint. Any excess fluid will be seen to move across the joint and distend the medial side of the knee.

Tenderness at the knee (tibiofemoral joint)

Identify the joint line clearly by flexing the knee and observing for hollows at the sides of the patella ligament – these lie over the joint line.

1. Tenderness at the joint line is common in meniscal and fat pad injuries.
2. Tenderness along the line of the collateral ligaments of the knee joint is common at the site of a lesion following a tear, particularly at the upper and lower attachments, and at the ligament's midpoint. Associated bruising and oedema may also be a feature of acute injuries.
3. Tenderness at the tibial tubercle – in children and adolescents, tenderness and hypertrophy of the tibial tubercle prominence – is associated with Osgood Schlatter's disease. Tenderness is also found following acute avulsion injuries of the patella ligament and its tibial attachment.
4. Tenderness and swelling in the popliteal fossa may indicate the presence of a Baker's cyst. This condition is associated with degenerative changes or rheumatoid arthritis involving the knee joint.
5. Tenderness at the adductor tubercle may indicate strain in the adductor magnus muscle.
6. Femoral condyle tenderness may indicate the presence of osteochondritis dessicans.

Patellofemoral joint assessment

A knee assessment should include assessment of both the tibiofemoral and patellofemoral joint. Observe the position of the patella and compare both sides.

- Determination of a high or small patella (patella alta) is made by calculating the ratio of the length of the patellar tendon to the longest diagonal length of the patella. The normal value for this ratio is 1.02 ± 20% (Simmons and

Cameron 1992). Patella alta is a predisposing factor in anterior knee pain and recurrent dislocation of the patella.

- Observe any tilting, lateral glide and rotation of the patella during a quadriceps contraction. Compare this with the other side.
- McConnell (1996) described a 'critical test' for the patellofemoral joint. Resisted inner-range quadriceps contraction is performed with the patient sitting at various degrees of knee flexion to determine whether this reproduces the patient's symptoms. Compare both sides (Figure 11.52).
- The McConnell critical test may be repeated with the patella taped in the corrected position. This will determine whether the taping is effective and should be incorporated into the treatment programme. Taping is believed to enhance activation and earlier timing of vastus medialis in quadriceps contractions and thus restore patellar tracking to normal.
- Observe any excessive pronation of the feet which may increase the Q angle (Figure 11.50).
- Test for tightness in the following structures: lateral retinaculum, iliotibial band, hamstrings and calves. Tightness of the above structures will increase dorsiflexion and therefore pronation of the foot and ankle during the gait cycle. All of this will increase the Q angle (Olerud and Berg 1984).
- Perform passive accessory movements to test the mobility and pain response of the patella in all directions. Observe pain, laxity or muscle spasm.
- Perform Clarke's test. The patient is asked to contract the quadriceps while the patella is pressed firmly down against the femur. Pain is produced in conditions such as chondromalacia or osteoarthritis affecting the patellofemoral joint.

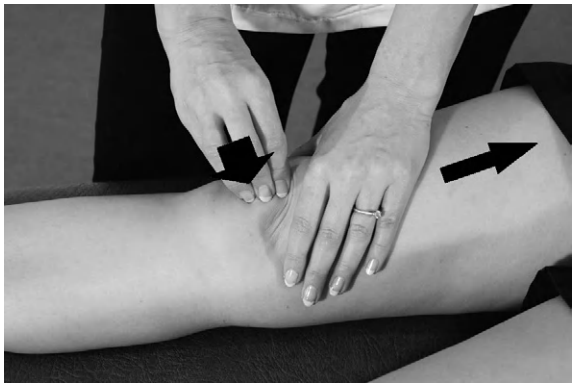


Figure 11.52 Critical test for patellofemoral pain. The test works because of the different contact areas of the patella against the femoral condyles in varying degrees of knee flexion.

Movements

Active movements

The patient is in half lying. Measure the active range of flexion and extension on each leg. The normal range of movement at the knee joint is approximately *minus* 5 degrees to 135 degrees of flexion. Note limitations of pain, stiffness or spasm. Overpressure the movement if full active movement is pain-free.

The axis of the goniometer should be positioned over the lateral femoral condyle. The static arm should be parallel with the long axis of the femur towards the greater trochanter. The dynamic arm should be positioned parallel to the long axis of the fibula and lateral malleolus (Figure 11.53). Hyperextension is present if the knee extends beyond 0 degrees (i.e. when the tibia and femur are in line).

Failure to hyperextend or lock out the knee fully may be a sign of a meniscal tear that is blocking the movement of the joint. Moreover, a springy end-feel may be indicative of a bucket-handle tear of the meniscus. A rigid block to extension is common in arthritic conditions affecting the knee.

Passive movements

Check the range of extension and flexion passively. If there is a difference in active and passive range determine reasons for this.

Valgus stress test (medial collateral ligament of the knee)

With the patient supine, the physiotherapist applies a valgus force to the knee joint (i.e. the femur is pushed medially, and the leg pulled laterally) while the joint is held in extension (Figure 11.54a). A positive sign is

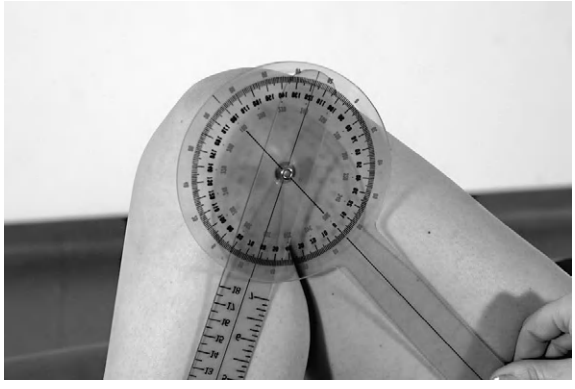


Figure 11.53 Measuring knee flexion using a 360-degree goniometer.

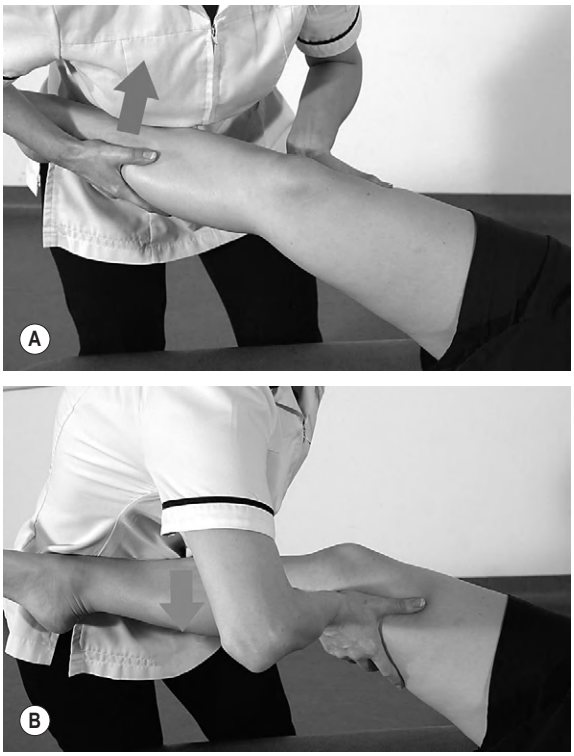


Figure 11.54 Stress tests: (a) valgus; (b) varus.

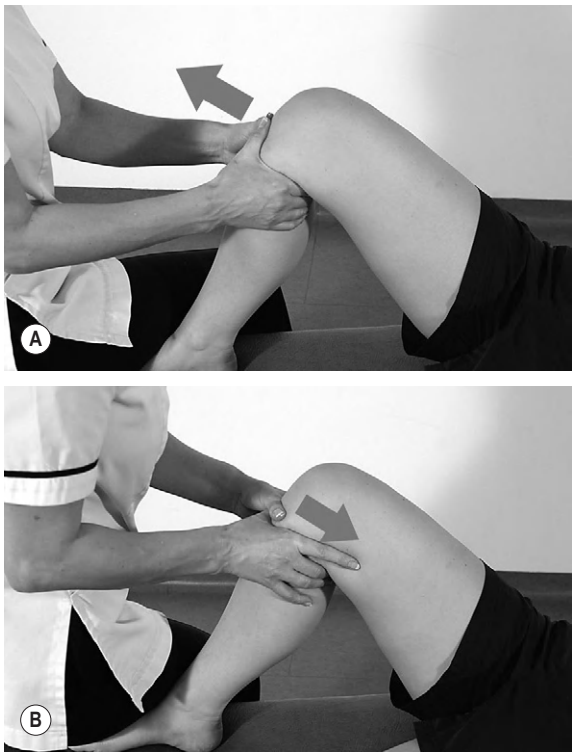


Figure 11.55 Draw tests: (a) anterior; (b) posterior.

observed as excessive opening up on the medial side of the joint. With the knee held in extension, a positive sign suggests major ligamentous injury involving the medial collateral, posterior cruciate and potentially the anterior cruciate. The test is performed again with the knee in 20–30 degrees of flexion.

Varus stress test (lateral collateral ligament of the knee)

With the patient supine, the physiotherapist applies a varus force to the knee joint (i.e. the femur is pushed laterally, and the leg pulled medially) while the joint is held in extension (Figure 11.54b). A positive sign is observed as excessive opening up on the lateral side of the joint. As with the valgus stress test, with the knee held in extension a positive sign suggests major ligamentous injury involving the lateral collateral, posterior cruciate and, potentially, the anterior cruciate. The test is performed again with the knee in 20–30 degrees of flexion.

Anterior draw test (anterior cruciate ligament)

With the patient crook lying, the physiotherapist sits on the patient's foot to stabilise the leg and grasps around the proximal tibia and tibial tuberosity and pulls the tibia forwards (Figure 11.55a). A positive sign is elicited by excessive translation of the tibia anteriorly (the normal translation is approximately 6 mm). Translation of 15 mm confirms rupture. Compare this with the other side. This test also stresses the posterior joint capsule, the medial collateral ligament and the iliotibial band (Magee 1992).

Clinical note

Figure 11.56 illustrates the positive posteroanterior draw following rupture of the anterior cruciate ligament.

Posterior draw test (posterior cruciate ligament)

With the patient crook lying, the physiotherapist sits on the patient's foot to stabilise the leg and grasps around the anterior aspect of the proximal tibia and pushes the tibia backwards (Figure 11.55b). A positive sign is elicited by excessive translation of the tibia posteriorly. Compare this with the other side. This test also stresses the arcuate-popliteus complex, posterior oblique ligament and anterior cruciate ligament (Magee 1992).

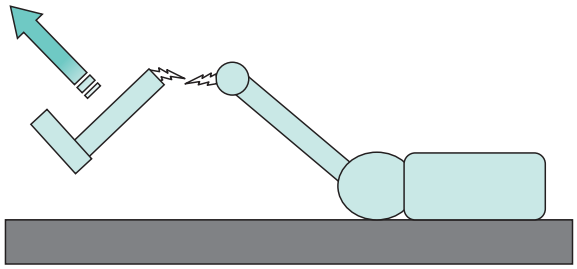


Figure 11.56 Schematic of positive posteroanterior draw following rupture of the anterior cruciate ligament.

Figure 11.57 Lachman's test.

Clinical note

A 'sag sign' is observed with the patient in crook lying, whereby the tibia is posteriorly displaced in relation to the femur. Posterior displacement may give the false impression that the patient has a rupture of the anterior cruciate ligament, as when an anterior draw test is performed a considerable amount of movement is noted. This is a result of the tibia returning to its normal position, however.

Lachman's test (modified anterior draw test)

The patient is supine with the knee resting over the physiotherapist's thigh at around 20–30 degrees of flexion (Figure 11.57). The physiotherapist grasps around the medial proximal aspect of the tibia with the right hand. The lateral aspect of the patient's femur is stabilised by the therapist's left hand. Anterior and posterior translation of the tibia is produced by the physiotherapist's right hand. This tests the anterior cruciate, the posterior oblique ligament and the arcuate-popliteus complex (Magee 1992). The Lachman test has been shown to be sensitive for the diagnosis of anterior cruciate injury (Kim and Kim 1995).

The pivot shift test

This is a test for anterolateral instability of the knee joint. With the foot in medial rotation and the knee in 30 degrees of flexion, a valgus stress is applied to the knee while simultaneously extending it. A 'clunk' indicates a positive test and suggests anterior cruciate ligament pathology (McRae 1999).

Key point

Peripheral tears of the menisci can now be sutured arthroscopically. Many authorities believe that McMurray's tests described below may be of limited value (Evans et al. 1993).

McMurray's medial and lateral meniscus tests

The physiotherapist palpates the medial aspect of the joint line, and passively flexes and then laterally rotates the tibia, so that the posterior part of the medial meniscus is rotated with the tibia. The joint is then moved back from a fully flexed position to 90 degrees of flexion to test for the posterior part of the meniscus. A positive test occurs if pain is elicited, or a snap or click of the joint will occur if the meniscus is torn. The test is then repeated for the lateral meniscus by medially rotating the tibia.

Note that the examiner may be able to detect clicking or snapping sounds when performing this test, as there are various structures in the knee joint that can produce these signs. It is thus easy for this test to produce a false-positive result (Palmer and Epler 1998).

Apley's compression/distraction test (for differentiation between meniscus and ligament)

The patient is prone with the knee flexed at right-angles. The physiotherapist medially and laterally rotates the tibia while applying a distraction force through the knee joint. The test is repeated by applying a compressive force through the knee joint. If the patient's symptoms are worse on compression then the symptoms are likely to be arising from a meniscal injury. Conversely, if they are worse on distraction then they are likely to be arising from a ligamentous injury.

Proprioception

Proprioception is tested with the patient standing on the unaffected leg and then on the affected leg while maintaining balance. Progressive adaptations may include standing on one leg with the eyes closed, standing on a wobble board, catching and throwing a ball, etc.

Accessory movements

Patellofemoral joint

- Medial, lateral, cephalad and caudad glides.

- Medial and lateral rotation.
- Compression and distraction.

Superior tibiofibular joint

- Anteroposterior and posteroanterior glides.
- Compression.

Tibiofemoral joint

- Anteroposterior and posteroanterior glides.
- Medial and transverse glides.

Quadrant tests

These are performed on non-irritable knees when plane movements are pain-free.

- Flexion/adduction quadrant.
- Flexion/abduction quadrant.
- Extension/adduction quadrant.
- Extension/abduction quadrant.

Following the objective assessment record your findings clearly and asterisk objective markers.

Test yourself

Match these five scenarios to the likely pathology.

1. The knee is stiff and painful for about half an hour in the morning, aches at the end of the day and has been like that for a long time.
2. The knee locks and has to be jiggled around to unlock it.
3. Since a tackle last week, the knee keeps giving way and becomes very swollen.
4. The knee is very red and swollen. The person also feels feverish and generally unwell.
5. When the person walks downstairs he feels pain behind his kneecap.
6. There is pain on the inside of the knee and it hurts doing sideways movements.

Answers

(1) Osteoarthritis. (2) Torn meniscus. (3) Ruptured anterior cruciate. (4) Infective arthritis. (5) Chondromalacia patella. (6) Torn medial collateral ligament.

THE ANKLE AND FOOT

Gait

Observe the patient's gait, both barefoot and with shoes. Ask the patient to walk backwards and forwards. Assess the normal heel-to-toe pattern and stride length, rhythm, the posture of the longitudinal arch and weight-bearing on both feet. Note any pain, stiffness and weakness. Inspect the patient's footwear for areas of uneven or greatest wear.

Key point

The patient should be suitably undressed to view the legs, ankles and feet.

Foot and ankle examination

Pulses (leg circulation)

Palpate the posterior tibial and dorsalis pedis pulses to establish the state of the distal circulation. Circulation is often poor in patients suffering from peripheral vascular disease or diabetes. Compare both sides.

Oedema

Note any oedema, suggesting a systemic rather than a local cause for the patient's symptoms. This may indicate heart failure or excessive water retention. Bruising is suggestive of muscle or ligament injury. This is commonly situated on the lateral aspect of the foot beneath the lateral malleolus, following lateral ligament tears.

General condition

Note the skin texture, colour and nail condition, which identifies the state of the peripheral circulation.

Temperature

Feel for any increase in temperature around the joint and compare with the opposite foot. A foot with impaired arterial circulation is colder than normal and may appear cyanosed (blue); conversely a warm foot may be indicative of an inflammatory response, for example following an injury or associated with conditions such as rheumatoid arthritis.

Tenderness

- Tenderness localised over and just proximal to the malleoli often occurs following a fracture.
- Tenderness and pain in the area distal and inferior to the lateral ligaments is common following inversion sprains. The anterior talofibular ligament is the most commonly injured as the ligament is most often torn in the combined position of inversion and plantar flexion. This is the loose packed position and one in which the anterior band of the lateral ligament is particularly placed on stretch.

Clinical note

A *pronated* foot has the appearance of rolling in on the medial side with bulging of the navicular bone medially. Additionally, the longitudinal arch appears flattened. A *supinated* foot has the appearance of rolling outwards with the inner border raised.

- Tenderness along the line of the long flexor tendons and/or the peroneal tendons may indicate the presence of tenosynovitis. This may be accompanied by local thickening.
- Tenderness at the articular surface of the talus is common in osteoarthritic conditions.
- Tenderness at the heel is found in conditions such as calcaneal exostosis (bony spurs), tendocalcaneal bursitis and plantar fasciitis.
- Diffuse tenderness under the metatarsal heads may be a sign of Morton's neuroma. This is a condition characterised by inflammation and pain around the third and fourth digital nerves. Pain is reproduced on squeezing the medial and lateral sides of the forefoot together.
- Diffuse tenderness and swelling on both the plantar and dorsal surfaces of the forefoot is a common finding in rheumatoid arthritis.
- Tenderness on the mid posterior aspect of the calcaneus may be a sign of a calcaneus bursitis.
- Tenderness along the Achilles tendon may be a sign of a sprain or tendonitis in the Achilles tendon.

Alignments

Observe the posture of the heel relative to the leg. The heel and lower leg should be parallel and the calcaneus should rest squarely on the ground. Note any postural misalignment such as excessive supination or pronation.

Excessive pronation may cause posteromedial shin splints, plantar fasciitis, hallux valgus or Achilles tendonitis. Excessive supination may cause anterolateral shin splints, dropped first ray (metatarsal) or plantar fasciitis.

Note whether the heel is inverted or everted. Posteriorly, the Achilles tendon and the calcaneus should be vertically aligned. *Calcaneal varus* is observed as the calcaneus being inverted relative to the leg; *calcaneal valgus* is observed if the calcaneus is everted relative to the leg.

Is the foot splayed or flattened? This may be owing to weakness of the intrinsic muscles and subsequent flattening of the longitudinal arch. Observe the posture of the medial arch and assess its height in comparison with the other.

Note any wastage of the calf musculature. Compare both sides. Measure the circumference of the leg with a tape measure at specified points below the patella.

The leg and hindfoot

With the patient prone, the physiotherapist bisects the calcaneus by drawing a vertical line through the posterior aspect of the calcaneus, then bisects the lower leg by drawing a vertical line on the posterior aspect of the lower third, and places the subtalar joint in a neutral position. If the lines are parallel there is correct alignment of the leg and hindfoot (Figure 11.58b). *Rear foot varus* is observed as the calcaneus appearing to invert relative to the leg (Figure 11.58a); *rear foot valgus* is observed as the calcaneus appearing to evert relative to the leg.

Key point

Complex foot misalignments may require referral to a podiatrist.

The hindfoot and forefoot

As above, observe the position of the whole foot. Correct alignment is observed if the hindfoot and forefoot are in line and perpendicular to the floor (Figure 11.58). *Forefoot*

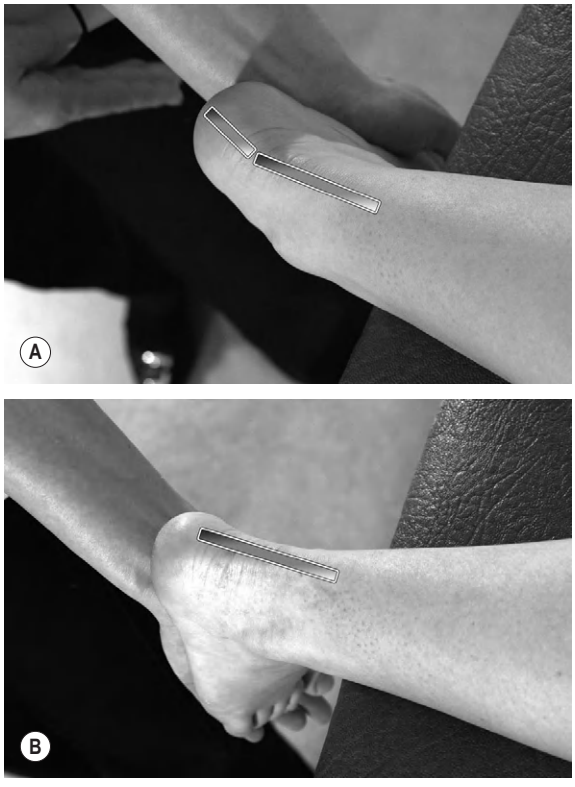


Figure 11.58 Alignment of the right leg and hindfoot: (a) calcaneal varus; (b) normal.

varus is observed if the first toe is superior to the lateral toes. *Forefoot valgus* is observed if the fifth toe is superior to the medial toes.

The toes

Look for:

- clawing (hyperextension of the metatarsophalangeal joints and flexion of the other phalanges);
- mallet toe (flexion of the distal interphalangeal joints);
- hammer toe (hyperextension of the metatarsophalangeal and flexion of the proximal interphalangeal joints);
- hallux valgus (lateral deviation of the first interphalangeal joint);
- hallux rigidus (stiffness of the first interphalangeal joint).

Functional activities

Assess the patient's ability to stand on heels, toes, and the inner and outer borders of the feet. Test the patient's proprioception. This may be performed with the patient standing on one leg or on a wobble board, with the eyes open and then closed. To make this more difficult the patient can catch and throw a ball while trying to maintain balance. Balance on the unaffected leg is assessed and then compared with the affected side. Also, when appropriate, test the patient's ability to hop, squat and jump, noting any stiffness or pain response.

Movements

Active movements

Ankle joint

The patient is in supine or sitting. Measure plantar and dorsiflexion. Movement occurring at the tarsal joints is easily mistaken for movement at the ankle, and vice versa. Note pain or stiffness compared with the other side. Overpressure the movement if it is full range and pain-free.

The axis of the goniometer is placed 15 mm inferior to the lateral malleolus. The static arm should be parallel to the fibula. The dynamic arm should be parallel to the long axis of the fifth metatarsal (Figure 11.59).

- Normal range of plantar flexion is approximately 50–55 degrees.
- Normal range of dorsiflexion is approximately 10–15 degrees.

Note that the knee needs to be in slight flexion if full dorsiflexion is to be achieved. This takes the stretch off the gastrocnemius.

Subtalar and midtarsal joints

In the normal foot, 80% inversion and eversion occurs at the subtalar joint. Most of the remainder occurs at the midtarsal joints, with a little at the tarsometatarsal joints. Determine the range by percentage of the abnormal to the normal. Test:

- combined inversion and adduction (supination);
- combined eversion and abduction (pronation).

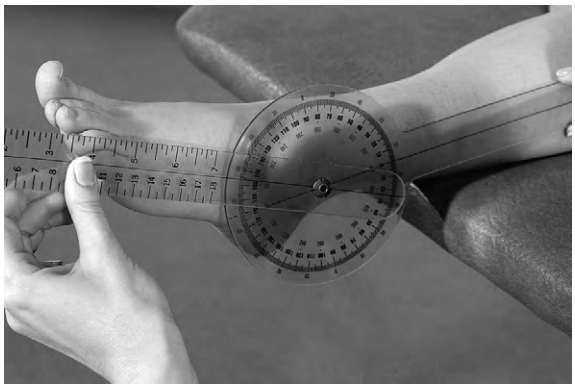


Figure 11.59 Measurement of ankle plantar flexion/dorsiflexion.

ABC Definition

Inversion is the movement whereby the soles of the feet face inwards towards one another. Eversion is the movement whereby they face outwards.

Toe movements

Observe flexion and extension at all the toes and compare with the opposite side.

Passive movements

All the movements measured actively can be measured and tested passively. Note any difference between active and passive ranges, and identify possible reasons for discrepancies.

Muscle strength

The dorsiflexors, plantarflexors, invertors and evertors are tested by isometric and isotonic resisted movements. Assess any weakness or pain elicited at any part of the range.

Ligament tests

Lateral ligament stress test. The patient lies supine and the physiotherapist grasps the heel and passively inverts the foot, feeling for any opening at the lateral side of the foot (Figure 11.60). A positive test may reveal increased inversion movement, a sulcus dimple on the lateral side of the foot or a pain response.

Figure 11.60 Passive inversion of the plantar-flexed foot to test the anterior talofibular ligament.

Figure 11.61 Anteroposterior glide at the inferior tibiofibular joint.

To differentiate between the three bands of the lateral ligaments, the test should be performed in:

- plantar flexion and inversion to strain the anterior band;
- inversion only to strain the calcaneofibular band;
- a combination of dorsiflexion and inversion to strain the posterior band.

Medial ligament stress test. The patient lies supine and the physiotherapist grasps the heel and passively everts the foot. A positive test may reveal increased movement of eversion compared with the other side and/or may elicit a pain response.

Anterior draw test at the ankle. This detects the integrity and stability of the anterior talofibular and calcaneofibular components of the lateral ankle ligaments. The patient lies supine. The physiotherapist stabilises the distal leg, grasps around the talus and pulls it forwards. A positive test reveals an anterior displacement of the talus in the mortise of the lower end of the tibia and fibula, and suggests major lateral ligament disruption. Observe for laxity, an audible 'clunk', or the presence of a lateral suction dimple.

Accessory movements (Maitland 2001)

Inferior tibiofibular joint

Perform posteroanterior and anteroposterior glides (Figure 11.61).

Ankle joint

Perform:

- posteroanterior and anteroposterior glides;
- longitudinal movement cephalad and caudad;
- medial and lateral rotation.

Subtalar joint

Perform medial and lateral glides.

Figure 11.62 Thompson's test of the right calf. Slight plantar flexion of the ankle suggests an intact Achilles tendon.

Test yourself

Match these five scenarios to the likely pathology.

1. One ankle keeps giving way and feels unstable. There is poor proprioception on one leg.
2. There is pain in the plantar aspect of the heel on weight-bearing or toe extension.
3. There is pain under the medial malleolus that increases on resisted inversion.
4. There is longstanding, insidious pain and stiffness in the ankle that increases on weight-bearing.
5. The patient has a history of an inversion strain combined with swelling and bruising under the lateral malleolus.

Metatarsophalangeal and interphalangeal joints

Perform:

- posteroanterior and anteroposterior glides;
- rotations and distractions.

Thompson's squeeze test

This tests the integrity of the gastrocnemius/soleus Achilles tendon complex. With the patient prone, the physiotherapist squeezes the calf firmly just distal to its maximum circumference (Figure 11.62). If the tendon is intact, the foot will plantar-flex. A positive test will occur if the tendon or muscle is ruptured and the ankle will not plantar flex. A palpable gap in the tendon or muscle belly may sometimes be observed if the tendon is ruptured. Following on from the objective assessment write up your findings clearly and asterisk an objective marker.

Answers

(1) Lengthened lateral ligaments. (2) Plantar fasciitis. (3) Tendonitis of tibialis posterior. (4) Osteoarthritis of the ankle. (5) Lateral ligament sprain.

REFERENCES

Alvarez, D.J., Rockwell, P.G., 2002. Trigger points: diagnosis and management. Am Fam Phys 65 (4), 653–660.

Bronfort, G., Evans, R., Nelson, B., et al., 2001. A randomized clinical trial of exercise and spinal manipulation for patients with chronic neck pain. Spine 26 (7), 788–799.

Burton, K., Tillotson, M., Main, C., et al., 1995. Psychological predictors of outcome in acute and chronic low back trouble. Spine 20 (6), 722–728.

Butler, D.S., 1991. Mobilisation of the Nervous System. Churchill Livingstone, Edinburgh.

Carrageen, E.J., 2001. Psychological and functional profiles in select subjects with low back pain. Spine 1 (3), 198–204.

Cats-Baril, W., Frymoyer, J., 1991. Identifying patients at risk of becoming disabled because of low back pain: the Vermont Rehabilitation Engineering Centre Predictive Model. Spine 16 (6), 605–607.

Coulter, I., 1996. Manipulation and mobilization of the cervical spine: results of a literature survey and consensus panel. J Musculoskel Pain 4 (4), 113–123.

CSAG (Clinical Standards Advisory Group), 1994. Back Pain Management Guidelines. Her Majesty's Stationery Office, London.

Cyriax, J., 1982. Textbook of Orthopaedic Medicine: Diagnosis of Soft Tissue Lesions, eighth ed. Ballière Tindall, London.

Deyo, R.A., Weinstein, J.N., 2001. Low back pain. N Engl J Med 344, 363–370.

Edwards, B.E., 1992. Manual of Combined Movements. Churchill Livingstone, Edinburgh.

Evans, P.J., Bell, G.D., Frank, C., 1993. Prospective evaluation of the McMurray test. Am J Sports Med 21 (4), 604–608.

Fairbank, J., Davies, J., Coupar, J., et al., 1980. Oswestry Low Back Pain Disability questionnaire. Physiotherapy 66, 271–273.

Fritz, J.M., George, S.Z., Delitto, A., 2001. The role of fear-avoidance beliefs in acute low back pain: relationships with current and future disability and work status. Pain 94 (5), 7–15.

Frost, H., Lamb, S., Klaber, E., et al., 1998. A fitness programme for patients with chronic low back pain: 2-year follow-up of a randomised controlled trial. Pain 75, 273–279.

Frymoyer, J.W., 1992. Predicting disability from low back pain. Clin Orthopaed Rel Res 221, 101–109.

Gardiner, J., Turner, P., 2002. Accuracy of clinical diagnosis of internal derangement of the knee by extended scope physiotherapists and orthopaedic doctors: retrospective audit. Physiotherapy 88 (3), 153–157.

Goel, V.K., Kong, W., Hans, J.S., et al., 1993. A combined finite element and optimisation investigation of the lumbar spine with and without muscles. Spine 18, 1531–1541.

Grichnik, K.P., Ferrante, F.M., 1991. The difference between acute and chronic pain. Mt Sinai J Med 58 (3), 217–220.

Guzman, J., Esmail, R., Karjalainen, K., et al., 2001. Multidisciplinary rehabilitation for chronic low back pain: systematic review. BMJ 322, 1511–1516.

Harding, V., Watson, P., 2000. Increasing activity and improving function in chronic pain management. Physiotherapy 86 (12), 619–630.

Heimeyer, K., Lutz, R., Menninger, H., 1990. Dependence of tender points upon posture: a key to the understanding of fibromyalgia syndrome. J Man Med 5, 169–174.

Hodges, P.W., Richardson, C.A., 1996. Inefficient muscular stabilisation of the lumbar spine associated with low back pain: a motor control evaluation of transverse abdominus. Spine 21 (22), 2640–2650.

Hodges, P.W., Richardson, C.A., 1997. Contraction of the abdominal muscles associated with movement of the lower limb. Phys Ther 77, 132–144.

Hubbard, D.R., Berkhoff, G.M., 1993. Myofascial trigger points show spontaneous needle EMG activity. Spine 18, 1803–1807.

Johnson, D., Rogers, M., 2000. Spinal manipulation. Phys Ther 80 (8), 820–823.

Jull, G.A., 1994. Examination of the articular system. In: Boyling, J.,

Palastanga, N. (Eds.), Grieve's Modern Manual Therapy, second ed. Churchill Livingstone, Edinburgh, pp. 511.

Jull, J.A., Richardson, C.A., Topperberg, R., et al., 1993. Towards a measurement of active muscle control for lumbar stabilisation. Aust J Physiother 39, 187–193

Kim, S.J., Kim, H.K., 1995. Reliability of the anterior drawer test, the pivot shift test, and the Lachman test. Clin Orthop 317, 237–242.

Koes, B.W., Bouter, L.M., Van Maeren, H., et al., 1992. A blinded randomised clinical trial of manual therapy and physiotherapy for chronic back and neck complaints: physical outcome measures. J Manip Physiol Therap 15 (1), 16–23.

McConnell, J., 1996. Management of patellofemoral problems. Man Therap 1, 60–66.

McKenzie, R.A., 1981. The Lumbar Spine: Mechanical Diagnosis and Therapy. Spinal Publications, New Zealand.

McKenzie, R.A., 1985. Treat Your Own Back. Spinal Publications, New Zealand.

McKenzie, R.A., 1990. The Cervical and Thoracic Spine: Mechanical Diagnosis and Therapy. Spinal Publications, New Zealand.

McRae, R., 1999. Pocketbook of Orthopaedics and Fractures. Churchill Livingstone, Edinburgh.

Magarey, M.E., 1988. Examination of the cervical and thoracic spine. In: Grant, R. (Ed.), Physical Therapy of the Cervical and Thoracic Spine. Churchill Livingstone, New York, pp. 81–109.

Magee, D.J., 1992. Orthopaedic Physical Assessment, second ed. WB Saunders, Philadelphia.

Maitland, G., 2001. Maitland's Vertebral Manipulation, sixth ed. Butterworth-Heinemann, Oxford.

Maniadakis, N., Gray, A., 2000. The economic burden of back pain in the UK. Pain 84, 95–103.

Mayhew, T.P., Rothstein, J.M., Finucane, S.D., et al., 1994. Performance characteristics of the Kin–Com dynamometer. Phys Therap 74 (11), 56–63.

Melzack, R., 1987. The short-form McGill Pain Questionnaire. Pain 30 (2), 191–197.

NICE (National Institute for Health and Clinical Excellence), 2009. Clinical guideline 88. Low Back Pain. www.nice.org..uk/CG88, accessed October 2012.

O'Conner, M.I., Curner, B.L., 1992. Metastatic diseases of the spine. Orthopaedics 15, 611–620.

Olerud, C., Berg, P., 1984. The variation of the Q angle with different positions of the foot. Clin Orthop 191, 162–165.

Palmer, M.L., Epler, M.E., 1998. Fundamentals of Musculoskeletal Techniques, second ed. Lippincott, Philadelphia.

Panjabi, M.M., 1992. The stabilising system of the spine. 1: Function, dysfunction, adaptation and enhancement and 2: Neutral zone and instability hypothesis. J Spinal Dis 5 (4), 383–389.

RCGP (Royal College of General Practitioners), 1999. Clinical Guidelines for the Management of Low Back Pain. RCGP, London.

Simmons, E., Cameron, J.C., 1992. Patella alta and recurrent dislocation of the patella. Clin Orthop 274, 265–269.

Sterling, M., Jull, G., Wright, A., 2001. Cervical mobilisation: concurrent effects on pain, sympathetic nervous system activity and motor activity. Man Therap 6 (2), 72–81.

Tait, R.C., Chibnall, J.T., 2001. Work injury management of refractory low back pain: relations with ethnicity, legal representation and diagnosis. Pain 91 (1/2), 47–56.

Vlaeyen, J.W., Kole-Snijders, A.M., Boeren, R.G., et al., 1995. Fear of movement/(re)injury in chronic low back pain and its relation to behavioral performance. Pain 62 (3), 363–372.

Von Korff, M., Saunders, K., 1996. The course of back pain in primary care. Spine 21, 2833–2837.

Waddell, G., 1992. Biopsychosocial analysis of low back pain. Baillière's Clin Rheumatol 6 (3), 523–555.

Wilke, H.J., Wolf, S., Claes, L.E., et al., 1995. Stability increase of the lumbar spine with different muscle groups: a biomechanical in-vitro study. Spine 20, 192–198.

Wojtys, E.M., Huston, L.J., Taylor, P.D., 1996. Neuromuscular adaptations in isokinetic, isotonic and agility training programmes. Am J Sports Med 24, 187–192.

Zigmond, A.S., Snaith, R.P., 1983. The Hospital Anxiety and Depression Scale. Acta Psychiatr Scand 67 (6), 361–370.

Chapter 12

The physiotherapy management of inflammation, healing and repair

Janette Grey and Gillian Rawlinson

INTRODUCTION

This chapter will introduce the reader to the processes involved in inflammation and tissue healing and repair, as well as discussing physiotherapeutic interventions that may be used to facilitate this process. The healing and repair process that occurs in response to tissue injury can, in broad terms, be described as a continuum of events that comprises of four stages: bleeding, inflammation, proliferation and remodelling.

These stages are not mutually exclusive and will overlap considerably, depending on the nature of the injury and the individual. However, for the purpose of this chapter, these four stages will be considered in sequence as underpinning a return to normal homeostasis and to normal function.

Clinical note

The inflammatory and tissue repair processes are fundamental to many pathologies and injuries that are treated by physiotherapists. The treatment of the inflammation itself may be directly affected by physiotherapy modalities or may need to be treated primarily through drug and medical intervention. For example, the inflammation of the bronchioles in an acute exacerbation of asthma would be managed medically with corticosteroids.

Although inflammation and tissue repair are not exclusive to the musculoskeletal system it is in this system that the physiotherapist most closely works to influence these processes. This chapter will, therefore, focus on the sequence of events following soft tissue injury and its physiotherapeutic management. The aims of the chapter are as follows:

- to identify the aims of physiotherapy and how they change throughout the continuum of tissue healing and repair;
- to provide the reader with specific physiotherapy examples of managing inflammation and repair;
- to develop the reader's ability to clinically reason the use of core physiotherapy modalities in the management of tissue healing and repair;
- to identify where research evidence underpins practice.

In order to clinically reason an appropriate physiotherapeutic approach to treatment for an individual's problem, many factors need to be considered. The physiotherapist needs to understand the anatomical and histological make-up of the different tissues, as well as the normal processes of inflammation, tissue healing and repair. It is also vital that the physiotherapist has excellent assessment and clinical reasoning skills in order to correctly identify the tissues involved and the underlying pathology. The clinical reasoning process also requires the physiotherapist to consider psychological and social factors, which will undoubtedly influence an individual's recovery and rehabilitation.

These factors may significantly alter the approach a clinician takes and maybe even exclude certain treatment modalities that would normally be considered. It is this individualised, holistic and evidence-based approach that makes the physiotherapist an invaluable contributor to the patient's optimal recovery from disease or injury. The clinical reasoning process is illustrated in Figure 12.1.

Two case studies will be presented to illustrate the suggested application of physiotherapy interventions discussed in relation to the healing and repair processes.

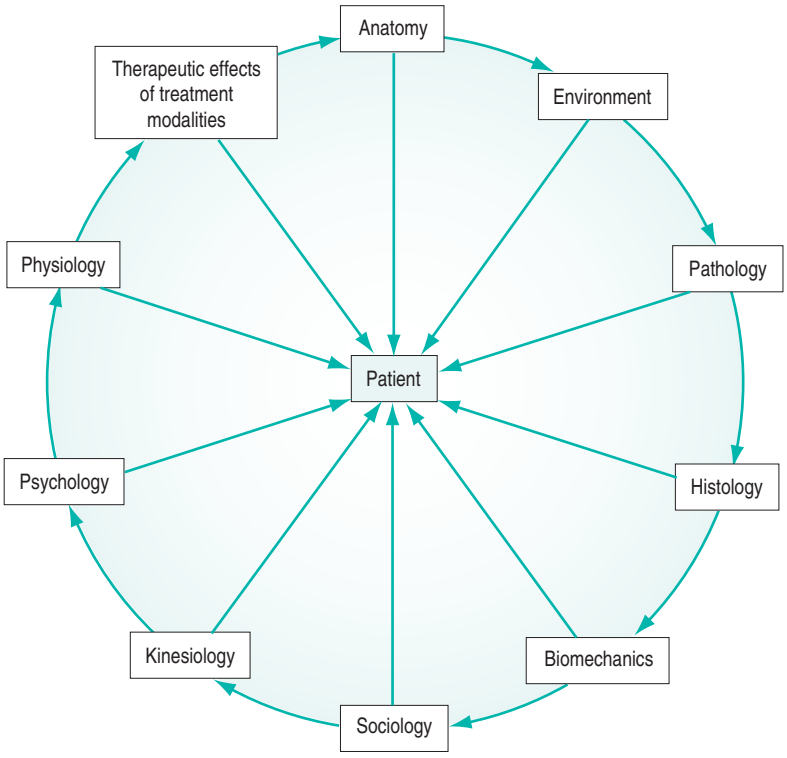


Figure 12.1 Factors influencing the clinical reasoning process for the management of patients' tissue healing and repair.

THE CONTINUUM OF TISSUE HEALING AND REPAIR

When any soft tissue is injured, be it through trauma, injury, overuse or surgery, a natural sequence of events follows in order to repair the damaged tissue and restore homeostasis and normal function.

This process starts with a short period of tissue bleeding owing to the disruption of small blood vessels and capillaries. Immediately following this period of bleeding a complex cascade of biochemical events proceed, triggering an inflammatory reaction. The inflammatory process initiates the proliferation of new tissue cells, which eventually remodel with the aim of restoring normal tissue function. This sequence of events is illustrated in Figure 12.2.

This sequence of events does not take on exact timescales and it is useful to think of this as a continuum whereby each stage of healing will overlap with the next depending on many factors, such as the severity and nature of the injury, the patient's age and tissue type and what the individual does or not do in terms of movement,

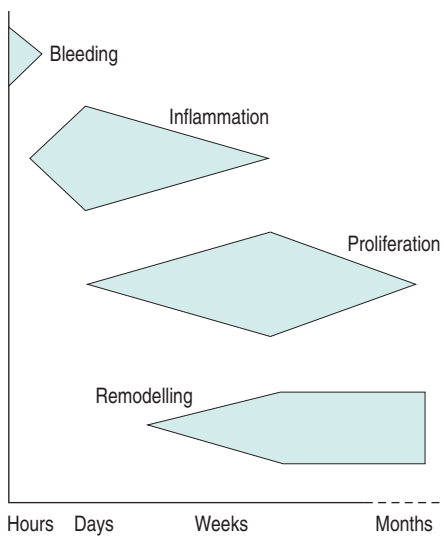


Figure 12.2 Phases of tissue repair. *Reproduced with permission from Watson (2004) with permission.*

activity and intervention. In many cases these healing processes occur without problems; however, several factors can cause this process to be delayed or exaggerated, leading to less than optimal tissue structure, pain and ultimately reduced function.

For many years physiotherapists have utilised a wide range of treatment modalities and interventions with the intention of promoting healing and repair. The main aim of using any physiotherapeutic modality or intervention should be to facilitate and progress tissue through this normal healing and repair continuum, thus facilitating early recovery and return to maximum function.

Soft tissue injury

When injury or trauma occurs producing forces necessary to damage soft tissues it is important to consider what happens to these tissues at a cellular level. The secondary injury model described by Merrick (2002) considers there to be two stages following tissue injury, namely the primary and secondary injury response:

- the primary injury is considered to be the damage to cells caused by the direct injury mechanism, be that a crush, contusion or strain force. The cells damaged by this mechanical force may have their cell membranes disrupted causing a loss in homeostasis and subsequent cell death. Many types of tissue may be involved, including ligament, tendon, muscle, nerve and connective tissues;
- secondary injury is thought to be caused by physiological responses to the initial primary injury. This occurs to tissues at the periphery of the injury to those cells not damaged by the primary injury. It is hypothesised that this damage may occur by two means. These are hypoxic (or maybe better described as ischaemic) and enzymatic mechanisms.

Ischaemic mechanisms

There are several proposed causes of ischaemia (reduced blood flow) to the damaged tissue, including haemorrhaging from blood vessels, increased blood viscosity secondary to inflammatory responses and increased extravascular pressure secondary to oedema. This reduced, or absent, blood supply will deprive cells which may have survived the primary injury of vital oxygen causing them to rely on the anaerobic energy systems which only have a short timescale (possibly up to six hours). If the ischaemia prolongs, many of these cells will subsequently die.

Enzymatic mechanisms

The lysosomes of those cells destroyed or damaged in the primary stage will produce enzymes that may damage and destroy neighbouring cells. This often occurs as a result of damage to the cell membrane, which causes the cells to swell and eventually die.

Phases 1 and 2 of tissue healing and repair

PHASE 1: BLEEDING (0–10 HOURS)

This is a relatively short phase that will depend upon the initial injury type, the tissues damaged and the severity of injury. If there has been an injury to soft tissues some degree of bleeding will occur. When capillaries and small blood vessels sustain primary injury, blood will escape into surrounding tissues and, depending on the location, may gradually track distally as a result of gravity. The type of tissue involved and the type of injury will determine the degree of bleeding in terms of amount and duration. If very vascular tissue is damaged (e.g. muscle) a larger amount of bleeding will occur in comparison with less vascular tissues, for example ligament and tendon. The bleeding phase may only last a period of a few minutes or hours, but in large muscle contusion injuries, for example, bleeding may continue to a small degree for up to 24 hours (Watson 2004).

PHASE 2: INFLAMMATION (0–4 DAYS)

Inflammation is a complex biochemical and cellular process which is still not fully understood. It can be triggered by many factors other than injury, such as infection and pathology.

Clinical note

When assessing bleeding it is also worth considering the location of the injury, as this will affect where the bleeding distributes. For example, with a tear of the anterior cruciate ligament the bleeding will be intracapsular in the knee and is unlikely to present as obvious bleeding and bruising with discolouration but as a tense swelling which, if aspirated, could be identified as blood causing a haemarthrosis.

Following an injury or pathological process there is an immediate inflammatory response. Scott et al. (2004) describe inflammation as a complex process that can be viewed at four levels: clinical, physiological, cellular and molecular, and suggest that it is the application of clinical reasoning skills that allows us to define which level is relevant in any given situation. Discussion of the complex biochemical sequences involved in the inflammatory process are beyond the scope of this chapter – the reader is referred to the wealth of books which discuss these biochemical and pathological aspects in more detail.

The inflammatory response has been reported to last for several days, or even weeks, but usually peaks around 2–3 days post-injury. During this inflammatory phase there may well be several clinical features evident. These include redness, swelling, pain and loss of function. During inflammation there is primarily a vascular and cellular response. The vascular response is a result of chemical inflammatory mediators and also a neural effect on the arterioles. There is an initial vasoconstriction that lasts only a period of seconds, followed by a more prolonged vasodilation response. There is also an increase in the permeability of the capillary walls allowing migration of large plasma proteins into the interstitial space. This alters the osmotic pressure in the tissue and exudate will gather in the interstitial space causing swelling. As cells migrate across the vessel wall into the interstitial fluid this will become cellular exudate. This exudate will contain mainly neutrophils initially and then lymphocytes and monocytes as the inflammatory process progresses (Mutsaers et al. 1997).

The cellular response is mediated and maintained by chemical mediators, which results in altered cellular activity. These processes will eventually aid the removal of any microorgansims and damaged tissue debris. Pain is caused by both irritation of damaged nerve endings from these chemical mediators and pressure on nocioceptors from the increase in exudate.

Often, clinically, this inflammatory phase is seen as a hindrance to the repair process and interventions that have an anti-inflammatory action are often employed with the aim of halting and slowing this process. It should be noted, however, that inflammation is, in fact, a normal response to injury and should be facilitated in order for the patient to progress through the healing and repair continuum.

Occasionally, individuals will develop a prolonged or exaggerated inflammatory response that fails to resolve within the normal timeframes. The mechanisms behind the development of chronic inflammation are not yet fully understood and are beyond the scope of this chapter.

Clinical note

There is some debate as to whether non-steroidal anti-inflammatory drug (NSAID) therapy should be used in soft tissue injury as it may slow down this phase of tissue healing, thus slowing overall recovery. Alternatively, the suggestion is that if it controls the inflammatory response and prevents it becoming exaggerated or prolonged this may, in fact, facilitate progression through the healing continuum.

PHYSIOTHERAPY INTERVENTIONS IN PHASES 1 AND 2 (0–72 HOURS POST-INJURY)

When considering the physiotherapy approaches that we can employ at these early stages of tissue injury, we must consider the physiological processes occurring and the aims and physiological responses to treatment.

When treating any patient with an acute injury, for example a ligament sprain or a muscle contusion, the aims of treatment must be decided in specific relation to the individual, in order to plan treatment accordingly.

General physiotherapy aims of early phase management (phases 1 and 2)

The aims are:

- to reduce pain;
- to limit and reduce inflammatory exudates;
- to reduce metabolic demands of tissue;
- to protect newly damaged tissue from further injury;
- to protect the newly-forming tissue from disruption;
- to promote new tissue growth and fibre realignment;
- to maintain general levels of cardiovascular and musculoskeletal fitness/activity.

The PRICE principles

PRICE is a mnemonic for the principal interventions commonly used in the immediate early stages following tissue injury. PRICE stands for :

- protection;
- rest;
- ice;
- compression;
- elevation.

These interventions together are applied in principle to address the seven aims of early phase tissue injury and healing management. These interventions are discussed below. The reader is also referred to the guidelines written in conjunction with the Association of Chartered Physiotherapists in Sports Medicine (ACPSM), on the immediate

management of soft tissue injury using the PRICE principles (Bleakley et al. 2010).

Protection

When soft tissue injury has just occurred it is important to protect tissues from further damage, both chemical and mechanical (secondary), and also to protect newly forming collagen fibrils in the following days. Measures to prevent tissues from this further mechanical damage are generally described as protective modalities and may include treatments such as strapping, use of crutches, slings and braces, and modification of exercises and movements.

Strapping

There are various techniques of strapping that can be applied in the early stages to protect injured areas from further damage while allowing the individual to continue with other activities and exercise. When deciding on the use of a strapping technique it is important to consider the structure(s) damaged and the movement(s) which are likely to stress the damaged area. Strapping can be applied in a manner to allow maximal movement while protecting from the movement(s) likely to cause further damage. Again, this emphasises why it is essential that the physiotherapist has a detailed knowledge of anatomy and human movement when treating injured and healing tissues.

It is important to remember that any strapping applied in these early stages must be able to accommodate a change in size or circumference because of swelling, to prevent compromise of the circulatory system. Often compression bandaging (as discussed below) will alone restrict movement and may be sufficient to protect from unwanted movement. Alternatively, additional specific strapping may be applied to reinforce and prevent specific movement patterns.

Clinical note

If someone has injured their anterior talofibular ligament in their ankle it will be necessary in these early stages to protect this structure from stresses into inversion and plantar flexion while allowing eversion and dorsiflexion movements. This can be achieved through accurate strapping.

Strapping needs to be reapplied regularly and also needs to be monitored regularly to ensure its safety and effectiveness. The physiotherapist will need to consider if the patient is able to reapply strapping independently or not, which may influence its use if the patient has limited contact with the physiotherapist.

The patient must also understand the limitations of the strapping. Some individuals may feel able to continue with all activities while wearing strapping when, in fact, this could be detrimental to the injured and surrounding tissues.

Other methods of protecting injured and healing tissues

The main factors affecting the physiotherapist's decisions to use a protective device are the individual's needs, the extent and nature of the tissue injury and the location of the damage. Devices such as slings may serve the purpose of elevating the limb while also having a protective role.

Clinical note

In the case of a patient having had a fasciectomy for release of Dupuytren's contracture, a splint is required immediately postoperatively to put the newly cut tissues under stress and encourage lengthening in order to prevent newly forming tissue from re-tightening, thus causing continued reduced function of the hand.

Walking aids

Walking aids will need to be provided if gait is altered. Also, partial weight-bearing may be advised in the early phases to protect damaged tissues, which will be stressed excessively during weight-bearing. Remember that the main aim of prescribing a walking aid is to promote a normal gait pattern within the limits of any restricted weight-bearing. This is essential to allow normal movement patterns of adjacent joints and body segments and to minimise any secondary problems.

Rest

Rest, in this context, usually refers to some form of relative rest in terms of general movement and activity to reduce metabolic demands and, hence, further secondary chemical damage to tissues. Rest from specific activities (relative rest) will also go some way towards protecting damaged and newly forming tissue.

However, it must be remembered that excessive unloading of the structures and prolonged rest can do harm to the patient so must be balanced carefully with appropriate activity (Bleakley et al. 2010). Therefore, the physiotherapist needs to carefully balance these protection and rest aims with those of promoting movement in order to maintain normal function of adjacent joints and structures.

In the very early stages (0–48 hours) when the tissues are still likely to be bleeding and the inflammatory process will be underway, the patient should be encouraged to rest

the injured area fully to prevent increased bleeding and inflammatory response.

As this very early phase ends (around 48 hours), very gentle movement is needed to help improve circulation and removal of waste products, and to provide the necessary stresses for the correct alignment and orientation of newly forming tissue fibres. Both local and systemic exercise can assist in this process through changes in haemodynamics and lymphatic function (Bleakley et al. 2010).

This trade off between rest and activity can often form conflicting advice for the patient – the physiotherapist needs to ensure that advice regarding movement and activity is clearly understood by the patient for maximum benefit.

Again, it is worth remembering at this stage that effective physiotherapy practice is underpinned by firm clinical reasoning where individuals' physical, social and psychological needs, their tissue damage and stage of healing are all considered in light of considered best practice in that treatment area.

Clinical note

It is essential with both elite and recreational athletes that they are advised and managed in terms of maintaining cardiovascular fitness and musculoskeletal fitness. This may require the individual to do a reduced or non-weight-bearing activity/programme such as exercise in water or reduced impact activity, or activities that do not involve the affected area. Remember, athletes may be eager to restart activity prematurely and the physiotherapist must take an active role in ensuring the patient fully understands the implications of all activities on the likely outcome of their injury/problem.

Cryotherapy (ice therapy)

Cryotherapy is defined as the use of cold and cooling agents used for therapeutic benefits, and has long been considered an important part of early tissue injury management. There are various methods of cooling tissues including the application of crushed ice, ice/gel packs, cold compressive devices and ice submersion. The comparative effects of these have not been fully investigated and the method of application is still mainly determined by the nature of the injury, equipment variability and therapist preference. The use of cryotherapy has not been fully investigated in terms of scientific research and much of physiotherapy practice in this area is based upon experiential evidence.

The primary reason for applying ice in the immediate early injury management is to cool the affected tissues, hence reducing the metabolic demands of the neighbouring cells. This should enable more cells to survive the ischaemic phase, thus minimising secondary tissue damage. It is suggested that to maximise the therapeutic effects of cryotherapy, an optimal tissue temperature reduction of 10–15° is required (MacAuley 2001). If the application of cryotherapy can reduce the number of cells damaged overall, the healing and repair process will be quicker, hence speeding up return to function.

Traditionally, the application of cryotherapy may have been thought to induce vasoconstriction of the small blood vessels, thus reducing blood supply to the area and hence causing increased ischaemia to the tissues and further secondary damage as initially described by Knight (1989). Merrick (2002) suggests that the secondary injury model described above now better describes the effects of cryotherapy. It must be noted, however, that there is very little conclusive evidence that investigates how cryotherapy affects the metabolic processes and whether it can influence inflammation (Bleakley et al. 2010).

The duration, application and frequency of cooling to achieve maximum therapeutic benefits has not yet been determined scientifically, yet these factors will greatly affect the degree of tissue cooling. Not all modes of cooling are equally effective; however, crushed ice provides effective cooling and is probably the safest (Bleakley et al. 2010). Subcutaneous fat covering will insulate deeper tissues and thus limit cooling significantly. Therefore, some areas may require slightly longer cooling times. Other important points are that the cold application should cover the whole area of injured tissue and a damp towel should be placed between the cooling agent and the skin to avoid skin and tissue damage. Using a thick, dry protective layer is likely to significantly reduce the cooling effects. It is also worth noting that research suggests that deep tissue temperature stays the same or continues to cool for up to 10 minutes following removal of an ice pack (Bleakley et al. 2010).

The use of cryotherapy is widespread in practice and it is generally accepted that it is a very safe and easy-to-use modality, making it very popular. However, it is very important to remember that direct cold application can cause what are commonly described as 'ice burns'. This may be described as superficial frostbite and the symptoms are similar to a thermal burn with pain, redness, swelling and blistering. It has been highlighted recently that ice burns are probably under-reported and are much more common than was previously thought. *Remember to exercise caution when applying cryotherapy and reassess the patient regularly.* If the patient has reduced sensation, or nerve injury is suspected, extreme caution must be used and it is advisable to avoid cryotherapy until this has been fully assessed.

Clinical note

When deciding on the dosage of cryotherapy application always consider:

- the size of the injury;
- the depth of the tissues injured and subcutaneous fat depth.

Always check sensation and monitor the skin regularly for evidence of ice burns. Placing a damp towel between the ice pack and skin should avoid damage, as should the use of a cold compression device.

Compression

Compression of the affected tissue and adjacent areas can also be used in the early phases to reduce exudate, protect tissues and possibly reduce pain.

The theory behind the application of compression is that the hydrostatic pressure of the interstitial fluid is raised, thus pushing fluid back into the lymph vessels and capillaries, and reducing the amount of fluid that can seep out into surrounding tissues (Rucinski et al. 1991). External compression through the application of an elastic wrap can stop bleeding, inhibit seepage into underlying tissue spaces and help disperse excess fluid (Thorsson et al. 1997).

Compression can be applied using a tubular bandage or some form of elasticated bandage strapping which may be adhesive or non-adhesive. It is vital that any product used is elasticated in order to accommodate changes in size or circumference of the body part without compromising circulation.

Compression can be used in direct conjunction with cryotherapy in the form of an ice compression device, for example Cryocuff, which allows simultaneous cooling and compression of tissues. If this method is not used there is generally some trade-off in terms of using cryotherapy and compression. It is not usually possible to sufficiently cool tissues through a compression bandage; therefore, intermittent use of cooling methods may be applied between compression.

Unfortunately, there are very few studies which look specifically at the effects of applying compression alone in acute soft tissue injury and, again, much of contemporary practice is based upon experiential evidence and consensus opinion (Bleakley et al. 2010).

Elevation

Another treatment method aimed at reducing bleeding, swelling and, thus, pain in acutely injured soft tissues is elevation. Some studies have suggested that elevating the injured limb above the level of the heart reduces interarterial pressure and enhances draining of extravascular fluid away from the area; however, scientific evidence supporting this is minimal.

Clinical note

When applying compression you will need to consider the following points (Kerr et al. 1999).

1. Apply compression as soon as possible after injury.
2. Always apply compression from distally to proximally.
3. Where possible, apply the compression a minimum of six inches above and below the affected area.
4. Always follow the manufacturer's instructions where available.
5. Do not apply compressive materials with the material at full stretch.
6. Ensure consistent overlap (half to two-thirds) of previous turn of compressive material.
7. Apply turns in a spiral fashion, *never* in a circumferential pattern.
8. Use protective padding, such as gauze, underwrap, foam, etc., over vulnerable areas such as superficial tendons and bony prominences.
9. Do not apply elasticated leggings in the lying position or in association with elevation.
10. Remove and reapply if the pressure appears not to be uniform or if the patient complains of discomfort. Otherwise, reapply within 24 hours.
11. Continue compression for first 48 hours when not lying down.
12. Always check the distal areas following compression to check for diminished circulation, i.e. colour changes or cold.

It is common practice to combine cryotherapy and compression with elevation in order to minimise swelling and gathering of interstitial fluid. However, continuously elevating the limb above the level of the heart is often impractical (especially if it is the lower limb) and may even compromise vascular supply to the body part if combined with compression. It may be more appropriate to advise the patient to elevate the affected limb intermittently during the day when possible and to remove compression at this time.

In the early stages a large amount of swelling is often evident if an individual with lower limb injury (particularly distal injury), stands and walks for a long time with the injured part in a dependent position. It would seem logical that if patients are advised to minimise the amount of standing and walking they do, and elevate the limb from the earliest point, this may reduce the accumulation of fluid which can quite quickly become thickened and fibrous owing to its high protein content. When an individual does stand and walk around then the use of compression would seem sensible to continue to minimise the accumulation of exudate in

the surrounding tissues. One practical point to remember when advising elevation is to support the limb adequately during elevation through the use of pillows or a sling.

Again, evidence underpinning the use of elevation to promote healing and recovery is lacking and research often shows that improvements in swelling are short-lived and swelling often recurs when resuming gravity-dependent positions (Bleakley et al. 2010).

First-line management

Increasingly in modern physiotherapy practice the physiotherapist will be the first line of medical assessment, management and advice. This may occur, for example, in the sporting environment, in private practice or via direct access services. These roles require the physiotherapist to have excellent assessment skills and experience, and the understanding of differential diagnoses and indications for onward referral. Several guidelines are in place to aid clinicians in managing and diagnosing injuries, and using investigations such as radiology. Physiotherapists should be aware of these guidelines and implement them appropriately in order to promote best practice. For example, physiotherapists working in these settings should be familiar with the Ottawa guidelines for ankle injuries (Bachmann et al. 2003), which guide clinicians on when to X-ray the ankle to rule out fracture.

Applying the PRICE principles in practice

Having discussed the elements of the PRICE principles it is evident that there are sometimes conflicting demands and that it is often not possible or, indeed, appropriate to employ all five principles simultaneously. As discussed previously, the research evidence underpinning these interventions is insufficient and much of everyday practice is based upon historical approaches and consensus opinion (Bleakley et al. 2010). It is therefore important to consider this and the physiological processes occurring in the tissues when rationalising a treatment plan for an individual patient. It is also important to make realistic and reasoned treatment decisions considering all biopsychosocial factors. This can be illustrated through the use of the case studies in this chapter.

Severity of injury and progression through the healing continuum

The speed of progress and recovery of those who have sustained soft tissue injury will vary depending on a large number of factors. These include the type, severity and location of injury, the individual's tissue type and response to injury, and many extrinsic factors, such as their exposure to appropriate treatment and advice in the immediate stages, and any other psychosocial factors. For example, a minimal grade I ligament injury effectively managed in the early stages may move very quickly through the continuum of tissue injury to repair and remodelling within a few weeks, whereas major tissue injury induced through trauma or surgery may require a much longer process. Those individuals not having sought, or followed, advice and treatment in the early stages are more likely to develop problems such as chronic swelling, loss of range of movement and function in injured and adjacent structures, and may even have compromised the formation of appropriate new tissue.

In view of this it is desirable that individuals seek early advice from an appropriately trained professional and that they fully understand the advice and treatment approaches taken. Often, many of the treatment modalities discussed so far can be implemented by a patient independently at home and the frequency and accuracy by which they do this will have a large impact on their recovery and progress. It should, therefore, be ensured that physiotherapy intervention is focussed on establishing that the patient fully understands the advice and treatment approaches that will be implemented at home, especially if the frequency of physiotherapy contact will be limited. If the patient does not fully understand the advice given and instructions regarding their activities at home then any benefit gained from a physiotherapy treatment session will be undermined by the patient's behaviour in the intervening period.

When deciding how long to continue to apply these principles consideration of the severity and size of the injured area is required. Despite common descriptions of grades of muscle and ligament injury being used, it is impossible to determine this accurately without radiological investigation such as ultrasound imaging. This is an area of increasing research; however, its clinical relevance in the management of muscle injuries is still under debate. Without the aid of valid and reliable imaging it would seem sensible to use the patient's level of pain and possible gentle palpation to indicate the size of injury as indicators of when to progress rehabilitation.

As discussed earlier, it is often suggested to consider beginning early movement of the injured part at around 72 hours post-injury as initial pain is reducing, and progressing to gradually achieve full range of movement of the affected tissues before moving on to increase normal muscle power, strength, timing and proprioceptive control in light of the patient's individual needs and abilities. In the early stages, acute pain is often a sign of further tissue injury and bleeding; thus, activity and movement of the injured area should be discouraged. However, as the continuum progresses, pain is often secondary to problems such as joint stiffness and lack of strength and control, and needs to be respected but not seen as a barrier to progressing rehabilitation. This will be discussed in detail in the following sections.

Phases 3 and 4: proliferation and remodelling

This section of the chapter deals specifically with the management of patients in the later stage of the healing and repair process. That is, beyond the initial bleeding and inflammation stages (the first 1–3 days following injury). Watson (2004) refers to these stages as the stages of proliferation and remodelling. The physiotherapeutic management of patients in these stages of the healing and repair process is still driven by the utilisation of physiotherapeutic measures which compliment, support and encourage these normal processes and therefore promote and facilitate repair. The underpinning reasoning behind physiotherapy in these stages of healing relates to the plastic properties of human tissue.

PLASTICITY IN HUMAN TISSUE

Plasticity refers to a quality associated with being plastic, malleable, and capable of being shaped or formed (OED 2007). Although the term plasticity tends to be most frequently applied in the field of neurology it is important to recognise that it is a characteristic of all human tissue. Human tissues have the capacity to adapt to the nature and extent of the forces applied to them. For example, if we consider the practice of lifting weights. Over a period of time, within a progressive lifting programme, an individual will be able to progressively lift larger weights. The skeletal muscles and tendons loaded by lifting weights adapt over time as the individual progressively lifts more weight. These adaptations affect all components of the musculotendinous apparatus. The contractile component of the muscle gets larger and stronger (Kraemer and Ratamess, 2004) and capable of producing increased force.

In parallel with this process the non-contractile components get correspondingly stronger and thicker to accommodate the extra loading (Benjamin 2002). In addition, adaptations occur at the interface between the tendon and the bone so that the bony prominences also develop to withstand the increased force exerted. Consider an individual who suddenly lifts a weight or engages in an activity where the force generated with the musculotendinous apparatus exceeds that which the muscle, tendon or bone can withstand. In this situation the tissue fails to withstand the force applied and injury occurs. For example, excessive load generated in the quadriceps muscle may lead to muscle damage, patellar tendon damage or avulsion fracture of the tibial tubercle.

Human tissues undergo reversal of these adaptations during the periods of immobilisation or reduced activity which occur frequently following trauma. In these situations the effect of immobilisation causes the reverse of the processes identified above. This reduces the ability of the damaged tissues to withstand loading. Physiotherapeutic management of these patients focusses on trying to restore the affected tissues' capacity to withstand normal loading therefore enabling the individual to return to normal function. It is important to remember that these soft tissue adaptation processes occur in both the injured and the uninjured tissues.

Physiotherapeutic approaches are therefore fundamentally aimed at capitalising on this plastic property of human tissue so that therapy works alongside, and enhances, the normal healing and repair processes.

Clinical note

A patient who sustains a severe second degree injury to the lateral ligament complex at the ankle and who subsequently has to non-weight-bear will experience soft tissue changes throughout the non-weight-bearing leg, not just in the tissues directly affected by the trauma.

FACTORS INFLUENCING THE RATE OF HEALING IN THE STAGES OF PROLIFERATION AND REMODELLING

An important consideration is that the processes of tissue proliferation and remodelling take place over significant periods of time (Watson 2004). The period of time for the completion of these pathophysiological processes can run into years and is dependent on a number of factors including:

- severity of initial trauma, e.g. a severe second-degree ligament sprain of the lateral ligament complex at the ankle will have a more prolonged proliferation and remodelling period than a first degree ligament sprain affecting the same structure;
- early management where the approach taken has laid the necessary foundations for proliferation and repair, e.g. an appropriate approach has been taken to rest/protect traumatised tissues (see earlier sections in this chapter). This has the potential to reduce the onset of chronic inflammation where an agent is responsible for the ongoing irritation of an injury and therefore perpetuates the period of the stages of bleeding and inflammation;
- tissue vascularity, e.g. skeletal muscle, which is highly vascular, has more potential for repair than a relatively avascular tendon;

- age, e.g. a similar grade of injury is likely to take longer in the repair process in an older individual than one who is younger (Myer 2000);
- nutrition – adequate nutrition (also related to blood flow) is required for healing to take place;
- medication, e.g. NSAIDs and steroidal drugs slow down proliferation and remodelling processes;
- temperature;
- biochemical factors;
- appropriate loading of healing tissue during rehabilitation.

It is very important that the physiotherapist has a depth of understanding of the pathophysiological processes involved in proliferation and remodelling, as well as the clinical reasoning skills required to make the appropriate professional decisions. In addition to this knowledge, the ability to apply this knowledge of the clinical effects of a number of physiotherapeutic treatment modalities, as well as the psychological and sociological theory in order that treatments are effective, is also required. In this section, short case studies will be used to illustrate the main points.

Evans (1980) simplified these processes in his 'graphs of hours', 'graphs of days' and 'graphs of months'. Although dated, this timeframe serves as a useful timeline against which to make judgements about physiotherapy approaches. An understanding of the timeframes involved in this continuum enables the physiotherapist to coordinate his/her approach to treatment and capitalise on the normal healing processes while ensuring that physiotherapy intervention does not induce further trauma and therefore delay healing and repair.

PHASE 3: TISSUE PROLIFERATION (FIBROUS REPAIR) (1–10+ DAYS POST-INJURY)

Pathophysiology

Cellular processes during this stage include:

- ongoing phagocytosis;
- angiogenesis (formation of new blood vessels);
- proliferation of fibroblasts;
- production of collagen fibres (initially these are produced in an unordered and random fashion);
- absorption of inflammatory exudate.

As the initial processes of inflammation begin to lessen, the inflammatory exudate begins to be absorbed. This leads to a decrease in swelling. Pain is reduced as a result. An increase in swelling during this stage is usually indicative of a more severe injury. Alternatively, this may be a result of inappropriate early management, for example a patient who has sustained a severe second-degree sprain of the lateral ligament complex at the ankle joint returns to full weight-bearing activity before the injured tissue strength has developed to a point where it is capable of withstanding the forces that walking demands of it. Tissue proliferation begins to occur as early as 24 hours after trauma. This proliferation begins the stage of fibrous repair, which usually involves the production of scar (collagen) material. The nature of the tissue, which is produced in the proliferation phase, is dependent on which elements of the tissues are damaged initially. If the cellular framework (satellite cells) is not damaged then regeneration of these cells may be possible. If the cellular framework (satellite cells) is destroyed then the tissue is replaced by scar tissue. In addition, if the cells damaged have no capacity to regenerate because they are deemed to be permanent cells, for example nerve cell bodies and cardiac muscle, then scarring definitely occurs. Scar tissue is a non-differentiated form of fibrous tissue that may develop some properties of the original tissue but will not function in the same way as the normal tissue would. By 2–3 weeks after the injury the majority of the scar tissue is in place. After this time, the rate of proliferation diminishes but may continue for several months following the injury. There is no hard and fast rule about exactly when this occurs, as the timeframe will vary in accordance with the factors that influence repair identified at the beginning of this section, for example it will occur earlier in vascular tissues.

Around 3–4 days following trauma the process of cell death will be complete and this means that the physiotherapist can assess the injury more objectively. At this stage the risk of aggravating/increasing the trauma or identifying false-positive findings because the patient's pain is masking the real symptoms is reduced. It is at this point that the physiotherapist can begin to use therapeutic techniques that promote an increase in circulation and tissue temperature without fear of inducing further tissue damage or further bleeding.

GENERAL PHYSIOTHERAPY AIMS IN THE TISSUE PROLIFERATION STAGE – PHASE 3

At this stage in the repair process the aims of physiotherapy may include the following:

- decreasing pain;
- decreasing swelling;
- decreasing local temperature;
- preventing further trauma;
- protecting new tissue;
- increasing range of movement;
- maintaining/increasing muscle strength/timing and control;

- preventing soft tissue adaptation in non-injured tissues;
- improving function.

The above represents an approach to a treatment that is impairment focussed and biomedical in approach. It is essential that this generic approach be modified to reflect the individual nuances of each patient and their individual and specific psychosocial needs. For example, increasing range of movement and improving function could be joined into one function-specific, patient-centred aim, for example 'to able to tie own shoelaces'.

Physiotherapy in the tissue proliferation stage (1–10+ days post-injury)

In this tissue proliferation stage physiotherapeutic treatment approaches are aimed at the achievement of the goals as identified above by influencing the pathophysiological processes involved in this stage of healing and repair. While approaches to physiotherapy to address the first four aims have largely been covered in the earlier parts of this chapter when considering the management of the bleeding and inflammation stages of the healing and repair continuum these are still worthy of a mention here. In order to reduce pain the injury may require a degree of ongoing protection, such as limiting movement to that which is pain free. However, the degree of protection will slowly be reduced throughout this stage.

Use of cryotherapy at this stage

Cryotherapy can continue to be utilised if the area injured continues to have an elevated temperature. However, it is likely that this will probably only be required at this stage in more severe injuries. Often at this stage the physiotherapist might choose to apply cryotherapy at the conclusion of a treatment session. The clinical reasoning underpinning this is that during this stage the potential to cause minor bleeding and inflammation as a result of treatment is high owing to the frail and vulnerable nature of the newly developing tissue. Cryotherapy applied at the end of treatment can potentially minimise the risk of this occurring.

Prescribing activity and exercise in the proliferative stage

During this proliferative stage the main input by the physiotherapist is to encourage a graded return to exercise and activity. This is achieved through the prescription of a specific exercise programme tailored to each individual patient's needs. This type of approach that involves active involvement of the patient is valuable as it promotes patient empowerment and prevents the patient from taking a passive approach to treatment.

The aims of increasing range of movement, maintaining/increasing muscle strength/timing/control, preventing soft tissue adaptation in non-injured tissues and improving function are all best achieved through application of graded exercise and activity. Inevitably, function will be improved to some extent by activity to increase range, increase muscle strength and prevent soft tissue adaptation. However, it is important that functional activities that are specifically pertinent to the individual are targeted specifically within an individual's treatment programme as early as possible, for example increasing ankle range of movement is unlikely to produce a direct improvement in walking performance without a specific rehabilitation activity aimed at improving walking. The primary emphasis of activity and exercise in this proliferative phase is to increase range of joint movement.

Grading the level of activity

Ensuring that any activity undertaken by the patient early in the proliferative stage is completed within pain-free limits provides a mechanism to protect newly developing tissues and prevent further secondary damage to those tissues initially traumatised at the time of the injury. It is sensible throughout this proliferative stage of healing and repair to use the patient pain report during activity as an indicator of appropriate levels of activity and exercise. As a rule, any activity or exercise prescribed in the proliferative phase that provokes pain, which could be classified with a high SIN (pain severity, irritability and nature) factor should be avoided.

From around the second day post-injury, fibroblasts are increasingly prevalent around the site of the trauma. These are the precursors to the formation of new fibrous tissue that will enable the healing process. In the proliferative phase, it is inappropriate to do aggressive activities or exercises of the areas directly affected by the trauma, as this will only serve to further damage the newly developing tissue. The use of gentle, active movements controlled by the patient within the pain-free range is appropriate at this stage – they will stimulate fibroblast activity and promote the production of collagen tissue. This is an example of the plasticity described earlier in the chapter.

Beyond the fourth day post-injury, proliferation of new, developed collagen tissue begins to accelerate thereby strengthening the healing of the injured tissue. As a result of this, increasingly greater ranges of pain-free movement can be expected. The physiotherapist needs to encourage their patients to practise these movements frequently throughout the day. Taking the movements to their pain-free limit will begin to progressively load the newly developing tissues and will facilitate an inbuilt progression of activity because as the pain eases progressively

Clinical note

Caution is needed with some injuries in the proliferative stage so that the movements produced do not promote calcification within the associated soft tissues, for example following quadriceps contusion, so called 'dead leg' (haematoma of the thigh). In dead leg, the quadriceps muscle is crushed against the femur by a direct blow. This injury commonly causes damage to the periosteum of the femur, which causes osteoblasts to move into the overlying soft tissues (quadriceps). Overly aggressive early movement can promote osteoblastic activity within the muscle, which may develop into myositis ossificans.

A large observed difference in uninjured–injured knee range immediately post-injury and being unable to play on following injury are both key early indicators of myositis ossificans developing as a likely complication (Alonson et al. 2000).

more range of movement will be achievable. This will promote the further formation and alignment of collagen and hence promote repair and the development of a functional scar (Watson 2004). However, care must continue to be taken, as during this phase any excessive loading still has the potential to damage the newly-formed tissue thereby provoking further injury and delaying the repair process.

Range of movement exercises will contribute to the maintenance of muscle strength and promote tissue-loading, but during this stage it is normal practice to limit the amount of overt strengthening/timing/control exercise recommended to the patient until full, non-weight-bearing pain-free active and passive ranges of movement are achieved. The use of isometric contractions involving structures not directly affected by the injury can be commenced early in the process.

Clinical note

A patient with a severe second-degree sprain of the lateral complex of the ankle can safely exercise the knee and thigh area during the proliferative stage. The degree to which the muscles of the thigh can be loaded may be influenced by the ability of the patient to weight-bear.

Other available physiotherapeutic techniques suitable at this stage include reciprocal inhibition. This can be useful to increase range of movement and increase tissue length (Waddington 1976). Reciprocal inhibition is the physiological phenomena which occurs when a skeletal muscle (antagonist) contracts and the opposing muscle (antagonist) relaxes. These techniques can be useful when trying to encourage elongation of the musculotendinous apparatus. Their main use is when muscle tension or muscle spasm is identified as the reason for limited range of motion.

Very gentle passive static stretching of affected tissues can begin at this stage as long as precautions are taken not to be over zealous. Again, the patient's pain report is the main indicator of the amount of stretch that it is safe to apply. Ballistic stretching would not be advocated at this stage, as the potential to overstretch the newly forming tissues and reinjure while performing this technique is high.

Clinical note

A person with a grade 2 hamstring injury that occurred six days ago is likely to be at the stage where some encouragement of increasing length in the hamstrings is desirable to load the newly forming collagen tissue in order to promote further proliferation and repair. In clinical practice this is often difficult owing to protective muscle spasm in the affected hamstring group. Reciprocal inhibition whereby active contraction/shortening of the quadriceps group is used to induce relaxation and lengthening in the hamstrings can be used. This can be achieved by placing the individual on their unaffected side, fixing the hip of the affected leg in 40 degrees of flexion (thereby ensuring minimal stretch on the hamstrings over the hip joint), moving the knee into extension until slight stretch in the hamstrings is felt and then providing isometric resistance to the quadriceps. As this resistance is applied to the quadriceps, the opposing muscle group (hamstrings) relaxes reciprocally and allows further elongation. This provides a load to the newly forming fibrous tissue and therefore facilitates repair. The skill in this technique is in ensuring that the lengthening achieved in the hamstrings is slow and controlled. In addition, undue pain in the hamstring group should not be provoked by this technique.

As the tissues continue to proliferate, more fibrous tissue is laid down and the healing tissues begin to get stronger. During this time corresponding increases in the forces applied to the tissues can be made by increasing the force of stretch applied, increasing the range of movement produced and increasing the number of repetitions of any activity.

As a progressively greater load is applied to newly forming fibrous tissue it begins to remodel in response to the increasing forces applied. In addition, it is proposed that the haphazard arrangement of collagen laid down in the early healing stages begins to take on a more organised pattern as load is applied to it. The collagen fibres begin to align themselves along the lines of the stress. It is important, therefore, that the stresses applied to newly forming

tissue are directed along the functional lines of stress normally required by that tissue. This requires the physiotherapist to apply their anatomical knowledge in an effective way.

From about the sixth day post-injury onwards there are increasing amounts of fibrous tissue laid down and, correspondingly, increasing levels of activity can be promoted. Tissues should be stressed progressively. It is considered to be both safe and advantageous to warm-up the affected tissues prior to activity – this has the effect of reducing the amount of load required to stress the tissues (compared with cold tissue) and therefore the tissue can be stressed more safely.

Electrotherapy in tissue healing and repair

The use of electrotherapy in the healing and repair processes has long been discussed and, again, there is varied evidence to support or discount the use of modalities such as ultrasound (US), pulsed electromagnetic energy and laser therapy in facilitating these processes. Electrotherapy as a topic is very large and this chapter does not claim to fully address all the literature and physiology surrounding it. The reader is referred to the electrotherapy chapter and other texts and literature for further information and discussion of these topics.

The most common electrotherapy modalities that appear to have the potential to have a direct action on the tissue repair process are US, pulsed shortwave diathermy (PSWD) and laser therapy. There is substantial evidence to support the notion that these modalities all have the capacity to influence the normal physiological processes of tissue repair, particularly the early inflammatory stage; however, clinical trials that demonstrate their efficacy in the clinical situation are lacking.

Watson (2006) describes a simple model of electrotherapy (Figure 12.3). This model summarises that the machine-generated energy in one form or another is delivered to the tissue. This energy is then absorbed by the tissue (to varying degrees depending on type), which then results in a change in one or more physiological events. It is the result of the energy being absorbed by the tissues resulting in a physiological shift, which is referred to as the therapeutic effect.

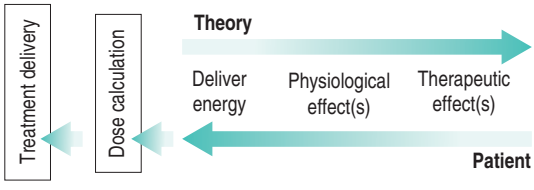


Figure 12.3 A simple model of electrotherapy. *Reproduced from Watson (2006) with permission.*

Choosing when and how to use electrotherapy?

Like any modality such as manual therapy, exercise or drug therapy there is an optimal time to apply it and appropriate dosage or intensity.

Watson (2006) suggests that the clinical reasoning process behind the use of electrotherapy can be illustrated by working through a reversal of the model shown (Figure 12.3).

Starting with the patient and knowing their identified physiotherapy problems and stage of healing and repair, the clinician can now work out what physiological effect is required and on what type of tissue. Once this is known the appropriate modality can be chosen based on the best available evidence. It must be remembered that if there is no electrotherapy modality suitable to achieve the desired result then electrotherapy has no place in the management of this condition at this particular time (Watson 2006).

Research suggests that it is the dosage applied and the specific tissue type that appears to most greatly influence this physiological shift. Studies have shown that different modalities applied at different dosages can have very different physiological effects. There does not yet seem to be an optimal dosage identified for any of these modalities for specific tissue types, thus making it difficult for the clinician to fully justify their choice in dosage.

In the proliferation stage it may be appropriate to continue to apply electrotherapeutic modalities, again ensuring that this is only used when an appropriate therapeutic window for the modality selected exists. During this phase of tissue proliferation the processes of phagocytosis, fibroblast and myofibroblast proliferation, and protein synthesis are prevalent. These processes can be therapeutically enhanced by the use of US, laser or PSWD. All of these modalities have the potential if selected appropriately (i.e. related to their specific tissue absorption properties), applied at the appropriate time and at an appropriate dose to exert a pro-proliferative effect whereby collagen synthesis is enhanced. This enables a speeding up of the proliferative phase and can therefore facilitate the rehabilitation process.

PHASE 4: TISSUE REMODELLING (10 DAYS + POST-INJURY)

Pathophysiology

Key processes involved in the phase of tissue remodelling are:

- ongoing fibroblast activity and collagen production (usually peaking 2–3 weeks following injury);
- absorption of older fibrous tissue;

- deposition of new fibrous tissue;
- scar tissue contraction;
- type III collagen fibres being replaced by type I collagen fibres.

You must remember that the different phases of the healing and repair process do not occur in perfect sequence. The phase of tissue remodelling and soft tissue contraction overlaps significantly with the earlier proliferation phase (see Figure 12.2). Remember that the peak of fibrous tissue repair occurs 2–3 weeks after the injury and the whole healing process is a dynamic one as older fibrous tissue is removed and new scar tissue is laid down. The continued application of physical stress to the developing tissues is one of the most significant factors influencing this phase of the repair process. The newly laid down fibrous tissue becomes 'more organised', being arranged less randomly and beginning to orientate itself along the lines of stress, thus enabling them to function more normally. This is the beginning of the remodelling phase which can continue for months and, possibly, for more than a year. It is clear, therefore, that this process continues long after the noticeable healing process and definitely beyond the time in which the patient would be in direct contact with the physiotherapist within the active rehabilitation process. This means that it is important that therapy, usually in the form of activity and exercise, continues long after an individual has been discharged from the ongoing care of the physiotherapist. Some evidence shows that even at this stage remodelling is still ongoing and tissues have not returned to their full functional capacity. Research in anterior cruciate ligament repair in monkeys indicated that the repaired anterior cruciate ligament reached 56%, 76% and 96% of its normal tensile strength 6 weeks, 12 weeks and 24 weeks after repair respectively. While this extent of remodelling may be acceptable in some members of the population it may not be so in others.

A key feature of the remodelling phase is soft tissue contraction. This is a process by which the newly formed tissues begin to physically shorten. This process has the potential for the healing tissues to begin to restrict and limit mobility and range of movement in the affected tissue. This process of soft tissue contraction begins to occur around the third week post-injury and continues long into the remodelling phase. As this process continues long after a patient would normally be discharged by the therapist, it is a key influence on the nature of the ongoing prescription of activity and exercise given to patients following discharge.

This phase of remodelling process takes place following the proliferative phase and is referred to as the chronic stage in some texts. Within this chapter, this phase is seen as a natural progression following the proliferative stage in an uncomplicated process of proliferation and remodelling.

Clinical note

The potential for tissue reinjury during the process of soft tissue contraction is clear. Failure to address this and plan activity and exercises strategies to avoid it is a potential key influence on reinjury patterns.

General physiotherapy aims in the remodelling stage (phase 4)

The aims of physiotherapy in the remodelling stage may include any, or all, of the following:

- promoting collagen growth and fibre/tissue realignment;
- increasing the range of movement:
  - active;
  - passive;
  - accessory;
- increasing muscle strength/control/timing;
- preventing soft tissue adaptation in injured and non-injured tissues;
- maximising function.

Physiotherapy in the remodelling phase (Phase 4)

These aims can be related to the ongoing pathophysiological processes involved. Fibroblastic activity increases and new collagen fibres continue to be laid down. This process peaks around 2–3 weeks post-injury, continues through to 4–6 weeks following injury and is clearly extended beyond this timeframe in more severe injuries. Ongoing attention to progressive activities started in days 3–10 post-injury is required. The purpose of the physiotherapeutic treatment is to promote movement and mobility of the injured structures/tissues.

Preventing tissue contraction and adhesion formation

Beginning around the third week post-injury the healing scar tissue begins to undergo a process known as soft tissue contraction. This means that the physical length of the tissue actually begins to get shorter. Consider this in the case of a hamstring injury as described above. At the same time that you are working with the patient therapeutically to lengthen the hamstrings the remodelling tissue begins to cause the healing scar tissues to shorten. This is a process that can go on for a considerable period of time after the injury. At this stage, it is normal for the patient to experience some discomfort during end-range stretching activity.

Clinical note

In practice this means that end of range stretching should commence on the third week following injury and should continue until the scar tissue contracture process is completed. The reality of this is that this is a very long-term process which may need to continue for as long as the remodelling process goes on. As indicated earlier this may be in excess of a year.

Contractures can sometimes become permanent. In these cases it becomes impossible to stretch the scar tissue. This is known as fixed contracture. Fixed contractures develop when the normal connective tissues (which are elastic) are replaced by fibrous tissue (which is inelastic). This means that the tissue will not stretch and this prevents normal movement. Contractures can occur in any tissue. The key to preventing the development of fixed contractures lies in ongoing therapeutic exercise and activity aimed at ensuring that as the scar tissue forms it remains a mobile, functionally-able structure. At the end of the remodelling stage the injured soft tissue will be repaired by scar tissue. The desired outcome is that this final scar is able to be a functional replacement for the tissues initially injured.

A potential problem during the remodelling phase is the formation of soft tissue adhesions. This is where newly forming fibrous tissue has been produced between adjacent layers of tissue such that these layers become bound together. These adhesions, when formed, can severely limit movement and soft tissue function. Normal movement requires that adjacent layers of tissue are able to move independently, for example a tendon moves over a bony surface or beneath a fascial band such as retinacula while the bone/retinacula stays still. As the formation of fibrous tissue during the proliferation phase is completely random, the potential for the development of adhesions is real. Movement in the early stages of healing and repair helps to ensure that the newly forming tissues do not create these adhesions. In some healing processes, where long periods of immobility are part of the patient management, their formation is probably inevitable. Soft tissue adhesions are normally easily palpable – tissues feel thickened and immobile to soft tissue palpation and mobilisation.

Clinical note

It is worth considering what happens in the soft issue repair processes following fracture, for example a patient sustaining a fracture of the distal radius is normally immobilised within 6–8 hours of the fracture in a plaster cast, which remains in place for a minimum of four weeks. During this time the normal soft tissue repair phases of bleeding, inflammation, proliferation and some remodelling all occur while the arm is immobilised. During this time, the tissues are not subject to any of the beneficial loading described in this chapter. When the plaster is removed, improving mobility and range of movement in the tissues can take significant periods of time.

Passive and accessory mobilisation techniques

Other techniques aimed at restoring full working/functional length in tissues include passive and accessory movement techniques. The application of these demands the use of an in-depth knowledge of human anatomy. This means that the physiotherapist can select the appropriate techniques to attempt to influence a specific structure.

Activity and exercise continue to play a key part in the physiotherapy management. A shift towards an activity and exercise programme placing more emphasis on improving muscle strength/timing/control is normally seen at this stage. It is important to consider all the ways in which the tissues in question are loaded during normal functional activity. This enables the physiotherapy programme to be tailored appropriately to an individual's need.

Muscle tissue will need to be able to withstand load from active and passive forces, from concentric, eccentric and isometric contractions at varying points within its range. It may need to work in an open-chain or closed-chain environment; it may need to function as an agonist, antagonist, synergist or fixator; it may need to generate strength, power or endurance. All of these factors must be considered when developing patient-specific exercise and activity programmes in the remodelling phase. Only when all of these things are considered and incorporated effectively into an exercise and activity programme is the potential for rehabilitation of individuals back to their full and maximum functional capacity realised.

Clinical note

Returning a patient to full road-running function following a second degree sprain of the hamstring muscles requires full range of motion in the joints of the lumbar spine, hip and knee, appropriate muscle control of the leg musculature (a combination of concentric, eccentric and isometric contractions), the ability to run at different speeds on different surfaces (including inclines) and to varying distances. All of these aspects must be covered in the final rehabilitation programme if the individual is to reach their maximum potential.

The context of an individual's normal functional activity is paramount to effective physiotherapy practice, whether it be playing a 90-minute game of football in the premiership or getting into and out of a car. The key skill that the physiotherapist utilises here is that of movement analysis. By analysing the demands of the activities that an individual aspires to be involved in, the physiotherapist is able to plan rehabilitation to meet individuals' needs with the aim of maximising each individual's capacity. Too often, in the author's opinion, the lowest common denominator of functional ability is accepted. This becomes the norm and physiotherapeutic intervention fails to enable individuals to achieve their full potential.

Tissue remodelling is a prolonged process which is especially pertinent in young patients who are remodelling and growing at the same time. In this client group, remodelling periods are often prolonged beyond the normal periods for adult members of the population.

Clinical note

Returning a patient to playing rugby following a dislocated shoulder requires an analysis not only of the demands of rugby *per se* but also an analysis of the specific position that a player may take on the field. They need to be able to throw and catch while running, to be able to run with the ball, and to tackle and be tackled. In addition, they will need to be able to withstand a direct fall onto the affected arm.

Clinical note

A window cleaner returning to work after a second degree sprain of the lateral ligament complex of the ankle requires full range of motion in the joints of the ankle complex and foot, appropriate muscle control of lower leg musculature (a combination of concentric, eccentric and isometric contractions), and the ability to walk at different speeds on different surfaces and to varying distances. He must also be able to balance on a limited base of support, at a variety of heights, for significant time periods while cleaning windows. Finally, he will probably need to be able to land steadily on his foot when jumping from a height. All of these aspects must be covered in the final rehabilitation programme if the individual is to reach their maximum potential.

Clinical note

It should be acknowledged that while this chapter identifies the key influences that physiotherapy could have on the process of tissue healing and repair, in cases of severe and extensive soft tissue trauma a degree of permanent loss of function of the tissues affected is inevitable. This loss of function within the tissues may lead to a degree of long-term disability for the individual affected.

CASE STUDY

AN INJURED HAMSTRING

Joe is a 25-year-old university student who plays football on a weekly basis. He injured his right hamstring while playing football yesterday. He describes a sudden onset of pain in the posterior thigh while he was running. He was unable to play on and hobbled off the pitch. There was no first-aid advice available. He spoke to a friend who recommended he went to see a local physiotherapist. Joe has self-prescribed ibuprofen and paracetamol. He is otherwise well and has had no previous injuries.

Joe attends the physiotherapy clinic the next day and limps into the clinic, weight-bearing only through his toes on the affected leg.

Table 12.1 Case study: An injured hamstring

Treatment aim	Treatment intervention
To protect newly damaged tissue from further damage To reduce metabolic demands of tissue To prevent and reduce swelling	Cryotherapy: apply ice pack or ice compression device to injury site (20 mins minimum duration regularly through day) Compression application of elasticated strapping (as described above) Elevate affected limb for intermittent periods. Beware of combination with prolonged extensive compression
To reduce pain To protect newly forming tissue from disruption To prevent soft tissue adaptation in non-injured tissues To promote collagen growth and fibre realignment To increase and restore normal joint range of movement (passive, active and accessory)	Use of crutches initially to protect injured area, encourage weight-bearing as pain allows Pain at the early stage should be seen as a protective mechanism to protect and prevent further damage; encourage movements of hip and knee within pain limits Anything that provokes pain at anything other than a minimal level should be discouraged initially Increase passive and active movements of the hip and knee, and then combine movement of both joints to increase stretch and load on tissues
To maintain and increase muscle strength, timing and control To increase proprioception of affected lower limb To increase tensile strength of new collagen tissue To restore and encourage optimal function in relation to patient's needs	Progress active and dynamic activity looking at lower limb and pelvis as a whole. Increase activity/football-specific tasks to improve proprioception, timing and neuromuscular control

CASE STUDY

AN INJURED ANKLE

Margaret is a 55-year-old hotel receptionist who slipped while coming down the stairs this morning sustaining an inversion injury to her left ankle. She is unable to weight-bear and has severe pain in her foot and ankle, and therefore attended the accident and emergency (A&E) department at her local hospital. X-ray revealed no bony injury. She is provided with crutches and taught to non-weight-bear. She is given an appointment to see the physiotherapist in A&E that afternoon.

Table 12.2 Case study: An injured ankle

Treatment aim	Treatment intervention
To protect newly damaged tissue from further damage To reduce metabolic demands of tissue To prevent and reduce swelling	Cryotherapy: apply ice pack or ice compression device to ankle/foot (of about 15 min minimum duration regularly through day). Monitor for reactions. Remember tissues are superficial and minimal subcutaneous fat Compression: application of elasticated strapping, particularly when patient is mobilising and not elevating distal limb (as described above) Elevate affected limb for intermittent periods. Beware of combination with prolonged extensive compression Advise regarding minimising walking and standing in first 5 days to minimise accumulation of swelling
To reduce pain To protect newly forming tissue from disruption To prevent soft tissue adaptation in non-injured tissues To promote collagen growth and fibre realignment To increase and restore normal joint range of movement (passive, active and accessory)	Teach safe use of crutches initially to protect injured ankle with weight-bearing as pain allows Pain at the early stage should be seen as a protective mechanism to protect and prevent further damage Encourage movements of knee, ankle and foot within pain limits Anything that provokes pain at anything other than a minimal level should be discouraged initially Encourage normal gait pattern without use of crutches as pain and acute stage ends Increase passive, and active movements of the knee, ankle and foot, and then increase stretch and load on tissues Consider using accessory joint mobilisations to maintain/increase range of movement at the distal tibiofibular, talocrural, subtalar and mid-tarsal joints
To maintain and increase muscle strength, timing and control To increase proprioception To increase tensile strength of new collagen tissue To restore and encourage optimal function in relation to patient's needs	Progress active and dynamic activity looking at lower limb and pelvis as whole. Increase activity/specific tasks to increase proprioception, timing and neuromuscular control of lower limb, particularly around the foot and ankle Consider occupation and return to work. Can job be adapted initially to allow early return to work or is time off appropriate. Consider all patient's needs in terms of sport and activities

REFERENCES

Alonson, A., Hekeik, P., Adams, R., 2000. Predicting recovery time from the initial assessment of a quadriceps contusion injury. Aust J Physiother 46 (3), 167–177.

Bachmann, L.M., Kolb, E., Koller, M.T., et al., 2003. Accuracy of Ottawa ankle rules to exclude fractures of the ankle and mid-foot: systematic review. BMJ 326, 417–425.

Benjamin, M., 2002. Tendons are dynamic structures that respond to changes in exercise levels. Scand J Med Sci Sports 12 (2), 63–64.

Bleakley, C., Glasgow, P., Phillips, N., et al., 2010. Management of acute soft tissue injury using Protection, Rest, Ice Compression and Elevation: Recommendations from the Association of Chartered Physiotherapists in Sports and Exercise Medicine (ACPSM). ACPSM, London.

Evans, P., 1980. The healing process at cellular level. Physiotherapy 66 (8), 256–259.

Kerr, K.M., Daily, L., Booth, L., 1999. Guidelines for the management of soft tissue (musculoskeletal) injury with protection, rest, ice, compression and elevation (PRICE) the first 64 hours. Chartered Society of Physiotherapy, London.

Knight, K.L., 1989. Cryotherapy in sports injury management. Int Perspect Physiother 4, 163–185.

Kraemer, W.J., Ratamess, N.A., 2004. Fundamentals of resistance training: progression and exercise prescription. Med Sci Sports Exerc 36 (4), 674–688.

MacAuley, D., 2001. Ice therapy: How good is the evidence? Int J Sports Med 22, 379–384.

Merrick, M., 2002. Secondary injury after musculoskeletal trauma: A review and update. J Athletic Train 37 (2), 209–217.

Mutsaers, S.E., Bishop, J., McGrouther, G., et al., 1997. Mechanisms of tissue repair: from wound healing to fibrosis. Int J Biochem Cell Biol 29 (1), 5–17.

Myer, A.H., 2000. The effects of aging on wound healing [wound care and seating]. Topics Geriatr Rehab 16 (2), 1–10.

Oxford English Dictionary, 2007, http://dictionary.oed.com.

Rucinski, T.J., Hooker, D.N., Prentice, W.E., et al., 1991. The effects of intermittant compression on oedema in post-acute ankle sprains. J Orthop Sports Phys Ther 14 (2), 65–69.

Scott, A., Khan, K., Roberts, C., et al., 2004. What do we mean by the term 'inflammation'? A contemporary basic science update for sports medicine. Br J Sports Med 38 (3), 372–380.

Thorsson, O., Lilja, B., Nilsson, O., et al., 1997. Immediate external compression in the management of an acute muscle injury. Scand J Med Sci Sports 7, 182–190.

Waddington, P.J., 1976. PNF Techniques. In: Hollis, M., Fletcher-Cook, P. (Eds.), Practical Exercise Therapy. Blackwell Science, Oxford.

Watson, T., 2004. Soft tissue wound healing review. http://www.electrotherapy.org.

Watson, T., 2006. Electrotherapy and tissue repair. Sportex Med 29, 7–13.

Chapter 13

Exercise in rehabilitation

Duncan Mason

INTRODUCTION

Exercise is one of the cornerstones of rehabilitation, widely used by many types of health professionals to manage an even wider range of medical conditions. It could be defined as 'using voluntary muscle activity produced by the integration of higher centres, cardiovascular, pulmonary and neuro-musculoskeletal components to rehabilitate these systems.' The aims and reported effects of exercise are also numerous. Throughout this chapter we will see an evidence-based overview and rationale behind some of the more commonly encountered types of exercise.

Exercise is frequently used to enhance the recovery rate of all components of the movement control system, namely the musculoskeletal and central nervous system (CNS), to increase the range of motion, increase muscle strength, develop a higher level of proprioceptive feedback and develop an overall improvement in sensorimotor control. However, the list of benefits does not end here; exercise is used in rehabilitation of the cardiovascular and pulmonary systems (Rees et al. 2004), which are required to provide the raw fuels for the physiological processes involved in muscle contraction. Also, exercise has been shown to have beneficial effects on the higher centres, reducing the effects of some mental illnesses (Galper et al. 2006). This explains its invaluable use in this area of healthcare.

Exercise can be delivered in a variety of ways: in the clinical setting or by teaching home exercises; as an individual or in a group setting; on land or in water. All approaches need to be considered to select the most appropriate for the individual requirements of each patient. The way in which the exercise is taught or delivered to an individual is also important in the ultimate success of this modality. This, too, will be discussed in the course of this chapter.

To summarise, exercise is a widely used treatment modality, which has been shown to be effective in the management of many medical conditions when appropriately selected and applied.

Glossary of terms

Delayed-onset muscle soreness (DOMS) A dull, or frequently more severe, aching sensation that follows unaccustomed muscular exertion. It is often associated with athletic activity. Eccentric exercise activity is said to provoke symptoms more readily owing to microtrauma – minor damage as a result of strenuous exercise. Soreness usually peaks 24–48 hours after exercise (Cheung et al. 2003)

Kinetic chain exercise Closed kinetic chain is an exercise performed where the distal segment of the limb is fixed (e.g. knee squats with feet on the floor). Open kinetic chain is an exercise where the distal segment of the limb is able to move freely in space (e.g. a biceps curl with a free weight)

Muscle contracture The adaptive shortening of muscle or other soft tissues that cross a joint, which results in limitation of range of motion (Kisner and Colby 1996)

Muscle spasm A persistent muscle contraction that cannot be voluntarily released

Muscle spasticity A condition associated with hyperactivity of the stretch reflexes and tendon stretch receptors, due to loss of inhibitory influences on the alpha-motor neurones on the motor unit (Mense et al. 2001)

Muscle stiffness (also sometimes referred to as muscle tension) An increase in background resistance to passive movement of soft tissues over a joint. This is often necessary to enhance function and is not necessarily an undesirable occurrence

Muscle tone The resting activity level or tension of a muscle, clinically determined as resistance to passive movement or to deformation

Myalgia Pain felt within a muscle

Proprioception The specialised variation of the sensory modality of touch that encompasses the sensation of joint movement (kinaesthesia) and joint position (joint position sense) (Lephart et al. 1997)

Stretching Any therapeutic manoeuvre designed to lengthen pathologically shortened soft-tissue structures and thereby increase range of motion (Kisner and Colby 1996).

STRENGTHENING EXERCISES

Introduction

Strengthening exercises are aimed at increasing the torque-producing capacity or endurance of a specific muscle or muscle group.

Adequate muscle strength is necessary to perform many activities of daily living whether it is to achieve self-care, occupational tasks or elite athletic performance. As a result of many pathologies muscle strength can be lost, whether directly caused by a trauma resulting in a disruption in the motor control mechanism or indirectly, for example as a result of pain inhibition or disuse atrophy. Pathological disruption to the nerve supply will also directly affect motor unit recruitment and therefore strength.

An evaluation of muscle strength is usually performed as part of a patient's objective examination. This should assist the therapist's clinical reasoning and enable them to rationalise an appropriate point to start strengthening rehabilitation from.

As a strengthening exercise programme is undertaken physiological changes occur within the muscle to increase its capacity to produce torque and sustain muscle contractions. This allows exercises to be progressed, in turn, to further overload and strengthen the muscle. Overload is an important component in an effective strengthening programme.

Therapists commonly employ several types of strengthening exercise

- free active;
- isometric, concentric, eccentric;
- open/closed kinetic chain;
- resisted.

These will be discussed in further detail in the remainder of this section and will be used depending on the strength requirements of the individual.

Other factors such as the number of repetitions, how often they are performed, i.e. number of sets, frequency per day and the load applied are also important factors dependent on the intended outcomes.

Measurement of muscle strength

Key point

It is important for a physiotherapist to measure muscle strength objectively when assessing an individual, to obtain a baseline level from which future improvements (or lack of them) can be gauged. This allows the therapist to devise an individual exercise plan and to evaluate the effectiveness of a prescribed exercise regimen.

Muscle strength can be evaluated in a number of ways: manually, functionally or mechanically.

The Oxford scale

The Oxford scale has been devised to manually assess muscle strength and is widely used by physiotherapists. According to the Oxford scale, muscle strength is graded 0 to 5. Table 13.1 summarises the grades.

There are limitations to the usefulness of the Oxford scale. These include:

- a lack of functional relevance;
- non-linearity (the difference between grades 3 and 4 is not necessarily the same as the difference between grades 4 and 5);

Table 13.1 The Oxford scale

Grade	Muscle contraction
0	No contraction
1	Flicker of a contraction
2	Full-range active movement with gravity eliminated (counterbalanced)
3	Full-range active movement against gravity
4	Full-range active movement against light resistance
5	Normal function/full-range against strong resistance

- a patient's variability with time (alternating between grades owing to fatigue, for instance);
- a degree of subjectivity between assessors;
- assessment of muscles acting only concentrically;
- the difficulty of applying the scale to all cases in clinical practice (so that strength is rarely evaluated throughout full range as many individuals seen by physiotherapists do not possess full range in the first place).

Owing to these shortcomings, modified versions of the Oxford scale are commonly seen in clinical practice.

Functional tools

Functional tools or scales can be used to evaluate strength and can be related to a specific activity or to one of its components. These tools are commonly employed when rehabilitating sportsmen back to competition, for example the triple hop test, designed to identify functional, unilateral lower limb differences. Sport-specific activities can be monitored by a physiotherapist with a working knowledge of the demands of a particular sport, its training and competitive requirements.

Isokinetic assessment

Isokinetic assessment has been used with increasing frequency since its inception in the 1970s. It involves the use of computerised evaluation of movement when exercising at a preset angular velocity on the isokinetic equipment (Figure 13.1).

This means that the subject can push as hard or as little as desired and the machine will move only at the preset velocity. It is therefore the resistance provided by the machine that varies.

The use of isokinetics has functional relevance as it can evaluate both eccentric and concentric activity through range.

Figure 13.1 A typical isokinetic machine.

Key point

Isokinetic machines are used for treatment, as well as assessment. They produce objective, reproducible and quantifiable data, and therefore have obvious advantages over other methods used in evaluating strength in the progression and monitoring of rehabilitation of strength deficits.

Drawbacks

The drawbacks of isokinetics relate to its function, as natural human movements rarely occur at fixed velocities. Also, the machine operates on a fixed axis of movement, which does not replicate the instantaneous axis of movement found in most normal joints. The equipment can also be time-consuming to set up and not all practitioners will have access to it.

Additional limitations have been acknowledged by Lieber (1992). These include the time required to recruit muscle fibres (50–200 ms), making this period of data obtained unusable. Another drawback is the limb striking the testing bar at the end of the movement, although some isokinetic units employ a damping mechanism to prevent this.

Key point

While isokinetics can give the practitioner a good idea of any underlying deficiencies in the musculoskeletal system, there is no single tool to evaluate strength that is both totally functional and quantifiable.

Strength training

Benefits

Strength training can include free active exercise or resistance training, when the body must overcome this resistance to produce the movement. This may be simply by use of body weight or free weights, or by the use of other equipment such as exercise machines in a gymnasium. Resistance training can also be used to train submaximal and endurance work. All aspects of resistance training can be incorporated into various rehabilitation programmes. Some of the reported physiological benefits of resistance training are:

- increased cross-sectional area of muscle;
- increased muscle fibre size;
- increased or maintained bone density;
- increased tensile strength of tendons and ligaments;
- decreased heart rate.

In order for a muscle or muscle group to develop sufficient strength gains it must be loaded progressively, otherwise strength improvements will be limited. This factor can often be overlooked when rehabilitating an individual and failure to progress exercise may result in a lack of improvement on the part of the patient.

Initial improvement in strength, when measured objectively, may be rapid without noticeable changes in physical characteristics. This is a result of enhanced neuromuscular coordination and utilisation of previously redundant motor units. More motor units are recruited within a given muscle and a stronger contraction of the muscle is therefore produced. This neural adaptation occurs before other physical and physiological changes that result from resistance training. It can be said, therefore, that there are two mechanisms that we aim to employ when applying strengthening exercises: firstly, the physiological adaptations occurring in the muscles, which take 6–8 weeks; and, secondly, the neural adaptations to facilitate more motor units within a muscle, which happens a lot sooner.

Table 13.2 Types of muscle contraction

Type of contraction	Characteristics
Isometric	Muscle maintains same length throughout the contraction
Concentric	Muscle contracts from a relatively lengthened position to a shorter position Performed either against gravity or against load/resistance Outer range to inner range
Eccentric	Muscle contracts from a relatively shortened position to a longer position Performed either with gravity or a controlled release of a load/resistance Inner range to outer range

Further study

To gain deeper knowledge on the physiological processes involved in producing muscle contraction and the adaptations that occur, it is necessary to study your recommended physiology text. Study the sliding filament theory and formation of actin and myosin cross bridges.

Free active exercise

These strengthening exercises are performed when the only external resistance that is to be overcome is the resistance provided by the weight of the body part. These exercises are used when the individual is at grade two or three, or below, on the Oxford scale for the targeted muscle group. Certain strategies are employed when dealing with an individual with this level of disability. Exercising the muscle in its middle range is the most effective part of the range for facilitating muscle activity, followed by inner range and then outer range. In middle range there is optimum length for formation of actin and myosin cross bridges; in outer range there is less capacity than inner range, although having more overlap does not allow optimal cross bridge formation. The use of overflow can also facilitate muscle activity, for instance when applying a force at another muscle in the region which normally works functionally with the targeted muscle or by working the corresponding muscle on the other side of the body.

Muscle contractions can also be enhanced by use of afferent input to the CNS, touching the corresponding dermatome (area of skin sharing the same nerve root innervation). Vocal encouragement, visual feedback and the use of biofeedback units, such as electromyography, are all useful in facilitating activity in redundant motor units. The type of muscle contraction used can also have a bearing on the effectiveness of the exercise.

Muscle contractions

Muscles can contract in three different ways: concentrically, eccentrically and isometrically. The characteristics of these different types of contractions are summarised in Table 13.2.

Key point

Sometimes the terminology *isotonic shortening* and *isotonic lengthening* may be seen instead of concentric and eccentric contraction. These terms are best avoided as they imply one contraction is an opposite of the other, whereas, physiologically, their characteristics vary greatly.

Individual muscles often exhibit more than one type of contraction at a time. Consider the muscle work occurring in the hamstring muscles in Figure 13.2.

The proximal part of the muscle is lengthening (controlling hip flexion) and the distal part is shortening, controlling tibial movement.

The features of eccentric exercise when compared with concentric exercise are, on the positive side, that it is mechanically more efficient and metabolically more efficient, but, on the negative side, it is less resistant to fatigue and may result in DOMS micro-trauma inflammation between sarcomeres. See Table 13.3 for further examples of eccentric activity.

Figure 13.2 Concurrent dual contraction during a squat.

Table 13.3 The functions of eccentric muscle work

Function	Example
Deceleration of a limb part	Kicking a football: the hamstrings act to decelerate hip flexion and knee extension
Force absorption	Landing from a jump: the quadriceps contract absorbing some of the ground reaction force thus reducing the joint reaction forces
Controlling a movement against gravity	Sitting down in a chair: gluteus maximus and the upper part of the hamstrings are controlling hip flexion

DOMS is a dull aching sensation that follows unaccustomed muscular exertion. It is a key characteristic occurring following eccentric muscle activity. DOMS should be differentiated from other types of muscle soreness that occur during, or soon after, exercise owing to metabolic factors, such as the build up of lactate.

DOMS is typically felt most acutely 48 hours after eccentric exercise has been completed (Howell et al. 1993; Rodenberg et al. 1993; Leger and Milner 2001). This commonly occurs in certain muscles of individuals undertaking a particular activity infrequently that has quite a high eccentric component. Examples include fell running (quadriceps in the downhill component) or playing squash (gluteus maximus when reaching for a low shot).

DOMS is effectively the occurrence of local microtrauma within the muscle. A key site for this inflammation is between adjacent sarcomeres or within the Z bands. Evidence for this inflammatory reaction can be found in increased levels of creatine kinase (CK) in the blood following exercise. This enzyme is released into the bloodstream following a muscular injury. Occasionally, the high CK levels found following eccentric exercise can confuse the clinical picture of a patient in whom CK levels may be used as a means of informing clinical diagnosis (Gralton et al. 1998). Saxton et al. (1995) have also reported a decrease in proprioceptive function with high-level eccentric exercise.

Despite many suggestions of dietary, medicinal, massage and exercise remedies there is conflicting evidence as to how best to enhance recovery from DOMS (Bennett et al. 2005; Close et al. 2005; Racette et al. 2005; Rahnama et al. 2005).

Treatment with eccentric exercise

Eccentric exercise has been identified as a key treatment technique when rehabilitating tendon injuries, such as in the tendo-achilles. Stanish et al. (1985) proposed treatment protocols with eccentric exercise involving alteration in both load and speed.

Key point

It is important to acknowledge that tendons function by storing elastic energy via elastic deformation and recoil. It may be that improved conditioning from isometric and eccentric exercise to the muscles (e.g. gastrocnemius and soleus) to which they are attached may also be a factor in studies reporting the positive benefits of eccentric exercises in tendon rehabilitation.

The benefits of using eccentric exercise programmes for tendon injuries are thought to include:

- enhancing muscle strength to improve movement efficiency in the musculo-tendinous complex;
- increasing the tensile strength of the tendon;
- stressing healing tissue in a functional manner to influence collagen deposition;
- influencing the organisation of collagen in a structure.

Knowledge of the types of contraction whether eccentric, concentric or isometric, is required when planning strengthening exercises. Eccentric contractions are capable of producing more torque than isometric, which, in turn, are stronger than concentric contractions. Isometric exercises are particularly useful when an area is immobilised, for instance when a joint is immobilised in Plaster of Paris, provided the contraction does not further damage soft tissues or destabilise fracture sites as active movement would be more likely to. It is useful to apply eccentric and concentric exercises in a functional context during rehabilitation. The functions of eccentric exercise being the

Figure 13.3 Eccentric training of the hamstrings.

deceleration of limb part, for example hamstrings braking knee extension when kicking a football, the absorption of force, for example when landing from a jump, or controlling a movement with the recoil of resistance or with gravity, for example during sit to stand (Figure 13.3).

Open-chain and closed-chain kinetic strengthening

The emphasis on closed kinetic chain exercises has developed since the onset of accelerated protocols for anterior cruciate ligament (ACL) rehabilitation and rehabilitation of anterior knee pain pathologies. A closed kinetic chain exercise occurs when the distal part of the limb (upper or lower) is fixed on a firm surface. Squatting is a commonly quoted example of a closed chain kinetic exercise, but performing a leg press exercise with the feet in contact with a metal footplate is also an example. Contemporary post-operative management of ACL reconstruction currently involves a combination of open and closed kinetic chain exercise; this reflects the mixture of open and closed kinetic functions in the knee and the lower limb as a whole in everyday functional activities.

The following are some of the proposed benefits of closed chain exercises in the lower limb:

- the shear force acting at the knee joint is reduced compared with the last 30 degrees of open chain extension (Wilk et al. 1996);
- they encourage more functional movement patterns;
- they stimulate co-contraction of the hamstrings, helping to reduce anterior tibial translation (important in ACL rehabilitation);
- they increase shoulder stability in the upper limb by stimulating co-contraction of surrounding muscles of the glenohumeral joint and scapula-thoracic articulation.

Closed kinetic chain exercises are not just a valuable part of lower limb rehabilitation, they are also widely used to good effect in the upper limb, particularly when addressing scapular or glenohumeral stability problems.

Resisted exercise

Resisted exercise is a more progressive form of strengthening exercise used in the more advanced stages of rehabilitation to achieve grade 5 on the Oxford scale. It is also an essential component of the training regimes of athletes aimed at enhancing performance and preventing the onset of injuries. As the name suggests, it is an activity during which an external resistance must be overcome to complete the exercise. This external resistance could be provided by the therapist during treatment, but, more commonly, is provided by equipment. The conventional means for this is by application of weight training with the use of free weights or weight training equipment as found in gymnasia. However, other equipment, such as medicine balls, elastic tubing or wrist and ankle weights, can be equally effective if used selectively. As larger weights are applied in this type of training it is essential to ensure that the patient has been progressively advanced to this stage of rehabilitation and must be closely supervised during the exercise, particularly whenever the loads are progressed. This type of training can be used to promote power or endurance in muscles and will be discussed in more detail in the following sections.

Types of muscle fibre

There are three main types of muscle fibre, usually called I, IIa and IIb, but newer subdivisions are now being described, such as Ic, IIc and IIab (Scott et al. 2001). Individual muscles are composed from all types, but have different proportions of each. Some of the differences in the physiological make-up of these muscle fibres are illustrated in Table 13.4.

The exact proportion of different fibre types is not consistent between muscles or between individuals. These characteristics are generally thought to be partly genetically determined and are part of the reason for the natural selection that sees individuals excel in different sports or play in different positions within a team. They are also influenced by training applied to the muscle (Scott et al. 2001). However, some general points can be made.

- Postural muscles such as the soleus are involved in maintaining position rather than dynamic activity and therefore have a higher number of type I fibres.
- Muscles that may be involved in more dynamic activity, such as the gastrocnemius, will have proportionately greater numbers of fast twitch fibres – types IIa and IIb.
- With age, the number and size of type II fibres decreases (Rogers and Evans 1993), making tasks that require a quick burst of strength more difficult. This should be an important consideration when rehabilitating elderly patients.

Table 13.4 Some characteristics of the muscle fibre types

	Type I	Type IIa	Type IIb
Fitness component	Cardiorespiratory fitness	Muscular endurance	Strength/power
Energy system utilised	Aerobic	Anaerobic	Anaerobic
Contraction speed	Slow	Fast	Fast
Numbers of mitochondria	Many	Moderate	Low
Resistance to fatigue	High	Moderate	Low

- There is evidence that muscle fibres can convert from one fibre type to another. This occurs between types IIa and IIb (Scott et al. 2001) in particular and is known as *plasticity*.

Number of repetitions

The number of repetitions performed of a particular exercise, along with load applied determines the type of muscle training effect. Resistance training includes pure strength work, as well as endurance work and, as a consequence, the number of repetitions will be based on the required outcome.

There is little clear evidence on the number of repetitions that should be used, but there are many protocols that can be used or adapted. A group of repetitions is known as a *set*, with three or four sets of an exercise usually being performed. This allows physiological recovery of the muscle to occur and delays the effects of fatigue.

Instructing a patient to perform ten repetitions may be the most appropriate number to prescribe for the particular weight and exercise, but there needs to be a method of determining the weight required for these repetitions. It is important not to lose sight of the purpose of resistance training (particularly strength training) – one of progressive overload to increase muscle strength and improve function. Progressive overload will not occur with repetitions of a weight that is too light and, as a consequence, recovery will be slower.

The majority of protocols that are in existence for strength training are based upon what are known as the 'one repetition maximum' (1RM) and the 'ten repetition maximum' (10RM). The 1RM is the weight that can be lifted once and only once in an exercise, with further completed effort being prevented by fatigue (Cahill et al. 1997). Determining the 1RM therefore prevents a challenge in the clinical environment and perhaps explains the sometimes arbitrary nature of repetitions given. The 10 RM is calculated similarly, i.e. the weight that can be lifted ten times. Once the 1RM or 10RM are established, weight training protocols can be calculated using percentages of this figure.

Key point

Sets consisting of ten repetitions are commonplace within exercise prescription. However, this is often just an arbitrary number. Avoid giving 'sets of ten' without any underlying rationale. It is often better when prescribing and teaching an exercise to observe for signs of fatigue and substitution to give a number of repetitions in a set. It may happen to be ten, but more often than not this will not be the case.

The number of repetitions prescribed is determined by the objective of the exercise. If the aim is to increase power then relatively few repetitions will be required, with long rests between sets, to achieve gains provided a high load is applied. Muscles with a higher proportion of type IIa and IIb muscle fibres with a mobiliser function will respond to this type of regime. If the aim is to increase endurance then lower loads will be required, but longer holds and higher frequencies of repetitions will be necessary for suitable gains. These exercises will be suitable for muscles with stability or postural function and a relatively higher proportion of type I fibres.

Further study

For more detail on exact protocols consult a strength and conditioning text.

Resistance training in different populations

The elderly

Physical strength decreases with age. Some of the reported reasons for this decline are the following:

- a decrease in the number of actual muscle fibres;
- muscle fibre atrophy;
- impaired excitation–contraction coupling;
- an inability or decreased ability to recruit type II motor units (Rogers and Evans 1993).

It is important to appreciate these long-term declines when addressing rehabilitation programmes with this section of the population. The issues outlined above related to progressive overload are just as significant but can be forgotten. An increase in basic strength may mean improved performance in functional tasks.

Children and adolescents

There is currently no evidence to suggest that resistance training in children is harmful, provided it is well supervised. The vast majority of adverse incidents have been related to poor technique and/or unsupervised activity. There have been no reports of growth plate fractures in studies related to youth strength training programmes.

The American Academy of Pediatrics has produced a series of recommendations in this area that stress supervision and numbers of repetitions between the age of 8 and 15 years. Practising technique with no load is also highlighted.

From a clinical viewpoint it is important to consider appropriate resistance training programmes when rehabilitating children and adolescents, adhering to the same principles of progressive overload.

MOBILISING EXERCISES

Introduction

These are exercises aimed at moving a targeted anatomical structure a precise amount in order to gain a treatment effect.

Soft tissue extensibility is a prerequisite for normal functioning. Unfortunately, following injury, inflammation, prolonged abnormal postures and other factors, such as exercise history, age and gender, this extensibility can be lost.

Therapists regularly encounter people who have well-established limitation of movement and need to be able to recognise this presentation and act accordingly to restore the length of the soft tissues involved. It is also important that the therapist is able to identify the muscles and soft tissues that are most prone to shorten and lose extensibility. The following sections of this chapter discuss the key concepts in the use of mobilising exercises in management of soft tissue conditions.

Mobilising exercises are a fundamental component of the rehabilitation process as they enhance normal tissue healing and are necessary to load the soft tissues progressively. Ultimately, the resultant scar tissue is stronger (Melis et al. 2002) and this enables it to more effectively withstand the stresses and strains it encounters during normal functional use (Hunter 1998).

Mobilising exercises can be used to maintain or increase range of movement, when restricted, for which the causes are numerous. These causes include:

- contractures of the joint capsule;
- adhesions within the soft tissues;
- muscle spasm or tightness;
- neural sensitivity and inhibition caused by pain.

Physiotherapists often use mobilising exercises in conjunction with other treatment modalities such as passive movements, heat, electrotherapy and soft-tissue techniques, depending on the presenting symptoms of the client.

For the purposes of this chapter, exercises are divided into the following classes:

- passive;
- active-assisted;
- auto-assisted;
- active;
- stretching (including hold/relax).

Classes of mobilising exercise

Passive exercises

Passive exercises can be defined as exercises by which movement is produced entirely by an external force with the absence of voluntary muscle activity on behalf of the patient. This external force may be supplied by the therapist (as is the case with passive movements) or by a machine. For example, continuous passive motion (CPM) units might be used following total knee arthroplasty or other knee surgical procedures, or in stroke rehabilitation (Lynch et al. 2005) to enhance recovery of the shoulder.

Passive exercises are typically employed in the early stages of rehabilitation after the onset of trauma, provided that affected structures are stable enough to sustain movement without vulnerability to further injury, and provided that the exercise is not unduly painful. They may also be used to maintain range of movement in soft tissues during periods of joint inactivity. They are commonly utilised in conjunction with stretching exercises to further increase the ranges achieved.

Active-assisted exercises

These are exercises in which the movement is produced *in part* by an external force, but is completed by use of voluntary muscle contraction. These exercises are of obvious value when strengthening a weakened muscle, but with the assistance given by the external force they can also be used to increase range of movement while allowing the individual to maintain control of the exercise.

Another important factor to be considered is *gravity*. If the exercise is performed with assistance from gravity, this may increase its effect on mobilising the targeted structure (Figure 13.4).

Figure 13.4 (a–c) Active-assisted shoulder elevation demonstrating the use of gravity and the other arm (via a pole) to gain range of movement.

Equipment

All manner of equipment is used to facilitate active-assisted exercises. Common examples are pulleys, slings and pole exercises for shoulders, re-education boards for knees and elbows, as well as many other external adjuncts. The physiotherapist should be as innovative as required.

Auto-assisted exercises

These exercises can be either passive or active-assisted, as described above, and occur when the external force is applied by the individual rather than by the physiotherapist. This may be through use of exercise equipment, for example shoulder mobilisation using auto-assisted pulleys.

Active exercises

Also known as *free active exercises*, these are activities in which the movement is produced solely by use of the individual's voluntary muscle action. They can be used as either strengthening for grade 2 and above on the Oxford scale (see previous) or to mobilise structures – as is the case with dynamic stretching exercises.

Key point

With any exercise the correct choice of starting position is important. For example, for a mobilising exercise aimed at increasing range of movement, gravity may well need to be counterbalanced or the body part positioned so that gravity assists the movement. If an exercise is performed against gravity it will have more emphasis towards strengthening.

A clinically useful feature of active exercises is that they can be performed without the use of equipment, so they can be practised anywhere and can easily form the basis of a home exercise programme.

Stretching exercises

If performed appropriately, stretching exercises may be a simple, yet very effective, form of treatment. For example, in the elderly a loss of hip extension during walking implies the presence of functionally significant hip flexor tightness (Kerrigan et al. 2001) and predisposes individuals to falls and subsequent femoral neck fractures. Overcoming hip tightness with specific stretching exercises as a simple intervention shows some improvement in walking performance and possibly fall prevention in the elderly (Kerrigan et al. 2003).

Stretching is commonly employed within the athletic population. Increasing the range of motion available at a joint has obvious advantages in increasing function and performance. It is suggested that regular stretching improves force, jump height and speed, although there is no evidence that it improves running economy (Shrier 2004). It has also been suggested that it contributes to injury prevention, although research does not suggest that stretching reduces risk of injury or reduces the effects of DOMS at present (Herbert and Gabriel 2002).

Key point

Stretching exercises are normally used to mobilise neural and muscle tissue to the limits of the available range. There are many forms of stretching exercise and techniques commonly used in combination, therefore a consensus as to their effects is difficult to ascertain within the published evidence. Stretching will result in a temporarily increased change in length but prior to sporting activity, as part of a warm-up, it may be more appropriate to use a technique that activates muscles, as stretching may result in a reduction of muscle tone.

Stretching exercises are performed with the target structure moving towards a lengthened position. The stretching exercise will involve further movement in this direction so as to further lengthen the structure. Collagen fibres realign

rapidly as a result of stretching forces and become aligned (and therefore stronger) in the direction of the stretching force (Melis et al. 2002). The limiting factors to further movement, such as the degree of pain experienced, will govern the extent to which any further movement is possible. Stretches are commonly used to increase range of movement by mobilising restrictions within soft tissue (e.g. scar tissue) and are specifically used in the lengthening of tight muscles.

The time at which stretching is commenced after an injury needs careful consideration. After any soft tissue injury, the length of the immobilisation depends on the grade of injury and must be optimised so that the scar can withstand the longitudinal forces operating on it without re-rupture. Mobilisation of soft tissues by stretching will aid reabsorption of the connective tissue scar and recapillarisation of the damaged area (Kujala et al. 1997).

Stretching may also be used as a preventative measure, for example to prevent joint contractures. However, while the primary intervention for the treatment and prevention of contracture is to regularly stretch the soft tissues, and the rationale behind this intervention appears sound, the effectiveness of stretching has not been verified with well-designed clinical trials on subjects who have sustained soft tissue injury (Shrier and Gossall 2000). One such trial (Malliarapolous et al. 2004) looked at the frequency of stretching protocols in athletes with hamstring injuries and found a significant difference in rate of recovery when the protocol was applied three times daily compared with once a day. However, a lack of control group limits our insight into the effectiveness of this modality and a lack of longer-term surveillance limits insight into its effect on reinjury rates.

Stretches may be applied in one of two ways: either dynamically or statically (Table 13.5).

Table 13.5 Stretching exercises: A summary

	Dynamic stretch	Static stretch
Feature of stretch technique	Faster, rhythmic, higher velocity, motor control, functional	Slow, controlled, emphasis on postural awareness, bodily alignment
Duration of stretch	Repetitive, progressive	Sustained 30-second hold
Stage employed	End-stage rehabilitation Training motor patterning	Early, middle and end-stage rehabilitation
Client types	Sportsmen, active persons	All

Dynamic stretching

This involves gaining range by an active movement and should not be confused with ballistic stretching (see below) which involves the use of repetitive, bouncing, dynamic, rhythmic movements performed at higher velocities. Dynamic stretching involves progressively increasing the range through successive movements until the end of range is reached.

Dynamic stretching is especially useful when dealing with more advanced sports-related rehabilitation problems. The exercises enhance dynamic function and neuromuscular control through repetition and practice, thereby training motor patterning and enhancing the movement memory. If the patient is suitable, this form of stretching can be highly effective to mobilise soft tissues and enhance motor control.

Note on ballistic stretching

Ballistic stretching uses repeated, successive movements and *momentum* to gain range. The drawbacks to using this type of exercise with most individuals encountered in the hospital setting are that patients are typically not conditioned to use ballistic stretching effectively or *without sustaining further injury*.

During ballistic stretching, the stretch reflex is normally initiated to resist the change in muscle length and to protect the muscle from injury. This occurs as a result of stimulation of the non-contractile elements of muscle spindles, which send afferent information about the length of muscles to spinal cord level. In turn, this causes stimulation of the extrafusal fibres via the alpha motor neurone, resulting in a muscle contraction.

It is suggested should this muscle contraction coincide with the next ballistic movement, a muscle unconditioned to cope with that stress may become injured. As a result, patients are normally instructed not to 'bounce' while performing their stretching routines.

Static stretching

As the name suggests, this involves maintaining a position for a sustained period to gain the desired effect – it is widely agreed that an effective time to hold a static stretch is at least 30 seconds (Bandy et al. 1997). This gives time for the muscle spindle to adapt to the new length and will also result in an increase in functional length of visco-elastic structures, such as muscles, albeit temporary (Magnusson et al. 1998).

Static stretching is a controlled, slow movement with emphasis on correct bodily alignment. Static stretching protocols are commonly used and, for example, have been shown to be effective in terms of improving flexibility of muscle (Chan et al. 2001). An element of fine motor control and postural awareness is important during static stretching exercises and this can be enhanced by the use of feedback and correction from the therapist, as well as

Key point

Correction of this type of exercise is frequently required to ensure an effective stretch is produced in the targeted tissue.

mirrors. Exercise forms such as pilates, Tai Chi and yoga employ many of these principles and can be used effectively within a patient's exercise programme.

Therapeutic stretching or strengthening exercises are successful only if the target muscle is properly isolated (Gluck and Liebenson 1997), particularly when the tissue concerned crosses more than one body segment. Problems are often encountered when stretching two joint muscles. For example, when stretching the hamstrings, pelvic alignment needs to be controlled, as well as considering hip flexion and knee extension components, to stretch effectively.

The *order* in which the components are added can also influence the effect, depending on which area of the muscle's structure is to be targeted. For example, if it is required to target the distal portion of rectus femoris, one may wish to employ a stretch, which extends the hip then flexes the knee, while proximally the components may be added alternatively (Figure 13.5).

It is also important to consider the functional anatomy of the area being targeted, particularly when considering the addition of rotational components to stretching exercises. Using the hamstrings as an example, the medial hamstrings (semimembranosus and semitendinosus) would require a component of lateral rotation to stretch them effectively. To effectively stretch biceps femoris an element of medial rotation would be required owing to its ability to act as a lateral rotator of the knee.

Spinal position

Many upper and lower limb muscles at the scapular and pelvis have their origins from the axial skeleton, so spinal position can influence limb position. Consequently, throughout many stretching exercises it is important to consider any lumbo-pelvic, scapulothoracic or spinal movement to ensure that it is controlled throughout the exercise. Where possible there should be no spinal movement, ensuring a 'neutral' posture is maintained throughout. The 'neutral' position being the position of optimal alignment and stability (Richardson et al. 2004). For example, in the lumbar spine this would equate to a slight curve in the lumbar lordosis convex anteriorly and in the cervical spine, again, a slight cervical lordosis with a right angle between the mandible and anterior neck. The spine would then be maintained in this position for the duration of the stretch to prevent unwanted spinal movement and potential limitation of treatment effectiveness.

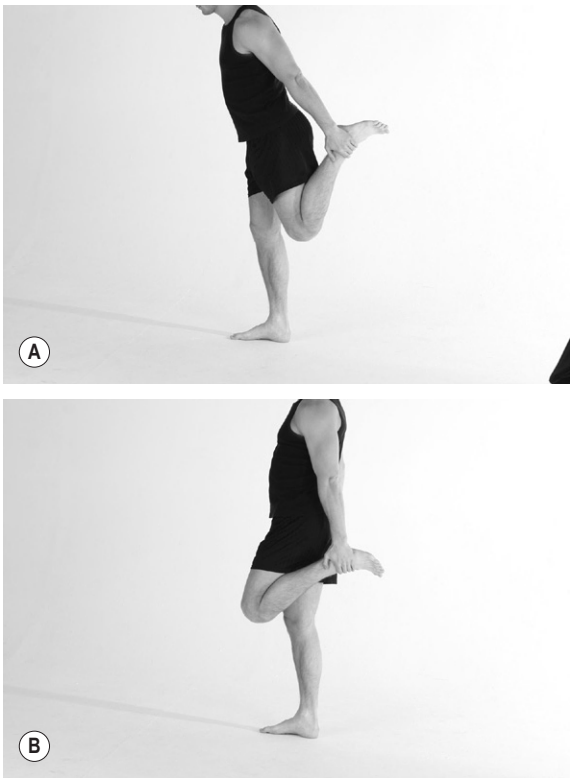


Figure 13.5 Demonstrating the order effect of adding components to a stretch of rectus femoris: hip extension then knee flexion (a); knee flexion then hip extension (b). Note that the lumbar spine should remain in neutral throughout.

Teaching stretching exercises: A practical guide

Before performing the stretch:

- ensure that your assessment has not identified any contraindications to stretching;
- ensure that there is a logical, reasoned basis for your stretching programme, e.g. if there is a bony block to movement caused by osteophytes, stretching is not appropriate (refer to Chapter 11 on 'end-feel');
- explain how and why they are performing the stretch to ensure maximum compliance and benefit;
- explain to the patient what they should experience during the treatment, i.e. how far to stretch, degree of discomfort to expect;
- consider how the stretch might be made more comfortable prior to stretching (e.g. use of a hot pack or hydrotherapy).

During the stretch:

- maintain spinal position in neutral throughout, if appropriate;
- patient to perform stretch across all joints at the same time for two or more joint muscles;

- make the stretch slow and sustained – do not bounce;
- the patient should experience a pulling sensation, not pain;
- hold the position for at least 30 seconds;
- if tension releases, take the movement a little further;
- release slowly.

After the stretch:

- warn the patient what feelings to expect following the stretch;
- remember that once movement has been regained, active muscle control throughout that range will be needed, as well as some form of maintaining the stretch in the long term.

Contraindications to stretching

These are some of the contraindications to stretching:

- bony block or end-feel to movement on passive assessment of the joint in question;
- recent or unstable fractures;
- acute soft tissue injuries;
- the presence of infection or haematoma in the tissues;
- after some surgical repairs and other procedures, such as skin grafting and tendon repair;
- patient refusal.

Key point

Although static stretching is widely believed to cause an increase in a muscle's functional length, recent investigations suggest otherwise. It has been suggested by Magnusson et al. (1998) that the visco-elastic properties of muscles remain unaltered in the long term following stretching and that it is the muscle's *tolerance* to stretch that is increased. Such details, along with further developments in the area, need to be considered when prescribing stretching exercises.

Hold/relax techniques

Proprioceptive neuromuscular facilitation (PNF), also commonly referred to as hold relax and contract relax, can have effective results when trying to mobilise muscles. PNF stretching techniques may produce greater increases in range of motion than passive, ballistic or static stretching methods (Spernoga et al. 2001). PNF is a form of treatment devised to manually rehabilitate movement in specific patterns using a number of physiological principles to enhance its effectiveness.

A core principle of PNF is that after a muscle has contracted maximally it will then relax maximally. This principle can be used when using exercises to mobilise muscles which are in a shortened position. The patient is asked to contract the tight muscle strongly and hold the contraction isometrically for around 10 seconds, then relax. Following a short period of 2–3 seconds the physiotherapist then applies a stretch to the muscle, which is maintained for 30 seconds. Following a period of recovery this sequence is repeated.

Another useful principle used in PNF is that of *reciprocal inhibition*, which states that when a muscle (the agonist) contracts maximally, its opposite counterpart (antagonist) will relax maximally. This can be used by asking the patient to maximally contract the agonist to the muscle to be mobilised followed by application of a stretch.

This principle of reciprocal inhibition can also prove useful and is worth considering during static stretching exercises where a contraction of the muscle's antagonist can act to relax and lengthen it.

PROGRESSION OF EXERCISE

An exercise programme without planned progression will quickly become ineffective. It is essential to review the exercise programme regularly and revise it to match the patient's status. There are various ways to progress exercises, including:

- changing the starting position;
- changing the length of the lever;
- changing the speed at which the exercise is performed;
- altering the range through which the movement is performed;
- applying resistance.

The starting position

Changing the starting position may change the base of support and may affect the difficulty of an exercise. Reducing the base of support will normally have the effect of advancing the difficulty of the exercise from a neuromuscular control perspective and vice versa. For example, performing an exercise while standing on one leg will require more hip abductor control than when standing on two legs.

A change in starting position can also change a body segment's relationship to gravity, which will change the nature of an exercise. An exercise performed against gravity will require concentric muscle work and eccentric work to return to the starting position – it will, therefore, be a strengthening exercise.

With more grossly weakened muscles (Oxford scale 2 and below), exercises performed in a gravity-counterbalanced position will be more effective. Exercises

performed in this position will also be more effective in mobilising structures.

If an exercise is performed with the assistance of gravity it may have a strong mobilising effect upon reaching the end of the available range of movement owing to the weight of the body part. An example of this is performing squats from a standing starting position to increase the range of knee flexion, using the effects of gravity and body-weight to increase knee flexion (Figure 13.6) and ankle dorsiflexion (Figure 13.7). Another example is shoulder flexion in lying and standing (Figures 13.8–13.10).

Length of the lever

Changing the length of the lever will affect the forces applied to the body during exercise (see Figures 13.11 and 13.12).

A lengthened lever will result in a higher force being exerted at the fulcrum of the movement, so more muscle work will be needed to produce or control the movement.

Figure 13.6 Static stretching of the right hip adductors.

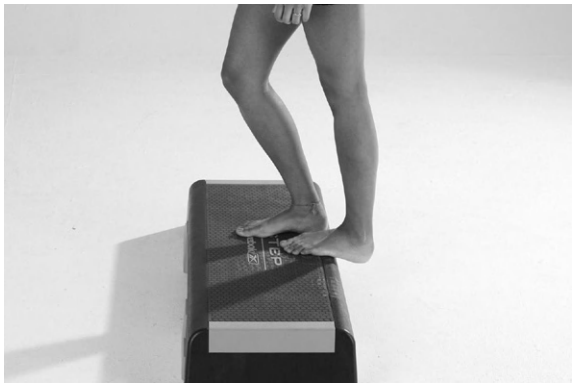


Figure 13.7 Using gravity to mobilise ankle dorsiflexion.

Figure 13.8 Shoulder flexion in lying.

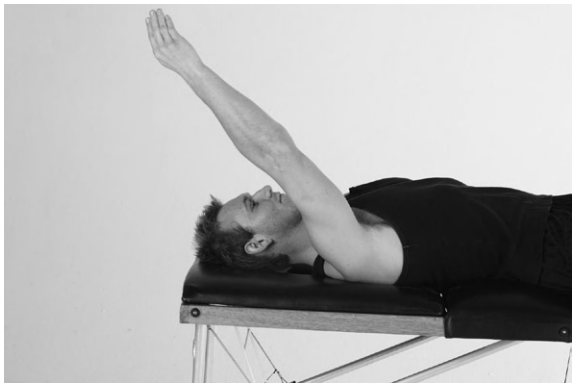


Figure 13.9 Shoulder flexion in lying.

Figure 13.10 Shoulder flexion in standing. Gravity resists the movement throughout the range in standing even though the movement is the same as in Figure 13.9.

Figure 13.11 Shoulder abduction with a shortened lever.

Figure 13.12 Shoulder abduction with a long lever. The muscle work of shoulder abduction is greatly increased by increasing the lever length.

Figure 13.13 A resistance exercise to strengthen shoulder extensors.

This will also result in a higher force applied to structures during mobilising exercises.

Levers may be shortened or lengthened by flexing or extending joints, most commonly the elbow or knee joints. They may also be lengthened by holding or attaching an object to the end of a limb, such as holding a weight at arm's length.

The positioning of the application of resistance along the lever will also affect the muscle work required. Resistance applied distally will require more effort and will reduce when the same resistance is applied more proximally. In mobilising exercises a resistance applied distally on the lever will have a greater mobilising effect than resistance applied proximally.

Speed of movement

A change in the speed at which a movement is performed will change the nature of an exercise. When altering the speed, the physiotherapist must be clear as to the desired effect. An exercise performed slowly requires a great deal of precision and postural control. An exercise performed at higher velocities will produce a greater mobilising effect at the end of range, but the client will require adequate neuromuscular control to perform this without risking further injury.

Range of movement

A change in the range through which an exercise is performed will alter its difficulty. Muscles are at their strongest in middle range and weakest in outer range (a more lengthened position). This is discussed in more detail in the context of muscle strengthening exercises.

Resistance to movement

The final way to progress an exercise is to apply resistance to strengthen a muscle (see Figure 13.13). Resistance can be applied in a number of ways. These are related to the desired effect – whether the aim is to produce a change in power or in endurance, as discussed.

DEALING WITH MOVEMENT DYSFUNCTION

When dealing with movement dysfunction we are interested in the kinetic chain, a series of successive joints or segments, and the way each segment's movement and orientation influences the rest. Movement dysfunction can give rise to acute muscle strain (Emery 1999) and overuse injuries owing to abnormal stresses caused by abnormal movement patterning. When patients present with symptoms resulting from movement dysfunction exercise is

used to rehabilitate them. Sahrman (2002) states that either maintenance or restoration of precise control of each segment is necessary to manage pain of musculoskeletal origin. To learn more about dealing with movement dysfunction and muscle imbalance refer to Chapter 14.

REHABILITATION OF SENSORIMOTOR CONTROL

Exercises aimed at accessing the sensorimotor control loop to develop and enhance movement control

Introduction

Normal proprioception is essential to everyday functioning, whether it involves simply placing a cup on a table or running around a football field. Definitions of proprioception have been the cause of some debate within the literature, as proprioception is only a part of the process of sensorimotor control. Therefore, some authors include the motor response, as well as the initial sensory input within their definition.

ABC Definition

Proprioception is 'specialised variation of the sensory modality of touch and encompasses the sensations of joint movement (kinaesthesia) and joint position (joint position sense)' (Lephart and Henry 1995).

The above definition implies that proprioception is very much an *afferent* (sensory) response. However, motor response has also been incorporated into other definitions of proprioception (Jerosch and Prymka 1996) and proprioception is an example of afferent input entering the CNS (Figure 13.14).

Physiotherapists are concerned with the normal functioning of this afferent response, but more importantly it is the efferent (motor) response to this sensory input that physiotherapists are most involved with from a rehabilitation point of view.

Loss of proprioception is a commonly encountered clinical problem and disturbs the continual feedback of accurate afferent information, and therefore has a knock-on effect on motor control. The key areas where proprioceptive deficits have been identified through research are presented in Table 13.6. The first column in the table illustrates the importance of being aware of the potential scope of proprioceptive loss and how important

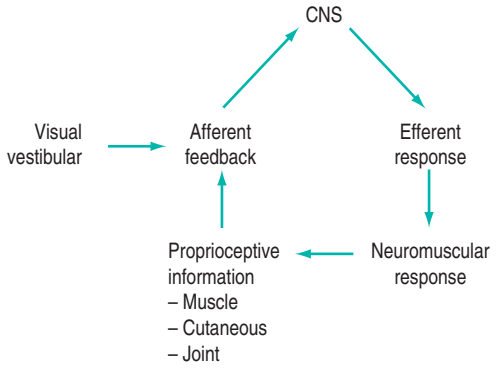


Figure 13.14 Diagrammatic representation of the mechanism of sensorimotor control.

Table 13.6 Conditions commonly exhibiting reduced proprioception

Proprioceptive deficits identified in	Clinical example
Osteoarthritis	Particularly joint degeneration in the lower limb, the knee encountered most commonly
Ageing	Any elderly patient
Immobility	Patients on bed rest or immobilised owing to illness or trauma
Trauma	Anterior cruciate ligament injuries, glenohumeral dislocation and instabilities, lateral ligament injuries of the ankle

! Key point

Proprioception could also play a vital role in prevention of injury, as well as in reducing symptoms of pathology. Caraffa et al. (1996) found a sevenfold reduction in anterior cruciate ligament injuries in a group of 300 semi-professional and amateur footballers who undertook a proprioceptive training programme when compared with a matched control group.

appropriate sensorimotor rehabilitation is with a large percentage of patients. It is primarily the muscle spindles that play a significant role in providing afferent proprioceptive feedback during movement (Proske 2005). It is the articular receptors that provide more information in outer

Table 13.7 Examples of receptors and their functions

Receptor	Type	Function
Ruffini end organs	Slowly adapting	Monitoring position of the limb in space
Pacinian corpuscles	Slowly adapting	Detection of acceleration and deceleration or sudden mechanoreceptor deformation
Muscle spindles	Rapidly adapting	Changes in muscle length
Golgi tendon organs	Slowly adapting	Changes in muscle tension

range when the structures in which they are embedded are put on stretch (Jerosch and Thorwesten 1998). It has also been hypothesised that proprioceptive deficiency could, in fact, precipitate degenerative joint changes (Barrett et al. 1991).

The mechanism of proprioception

There are many *receptors* within joints, muscles and skin that continually convey information to the CNS. Using this information we continually make subconscious and conscious modifications to how we move, allowing us to carry out normal functional activities. Each receptor supplies a different type of information, for example joint pressure, joint acceleration/deceleration and joint velocity. When the receptors are stimulated through movement or other forces, they act as transducers and convert this mechanical deformation into an electrical sensory impulse (Barrack et al. 1994). This sensory impulse then passes on to the CNS, triggering the appropriate motor response.

When walking on uneven ground, for example, the muscles of the lower limb continually have to adjust in order to keep the body upright and prevent a fall. The peronei and anterior tibialis, for example, will be continually controlling the movement of the foot and ankle, responding to constant positional changes the gluteals will also be performing a similar function at the hip joint. This example illustrates the nature of proprioception as a continuous process occurring at different levels of the CNS at the same time.

When rehabilitating sensori-motor control we need to take the task from the conscious into the sub conscious level to function normally. In early stages of rehabilitation we need to re-educate sensory and motor pathways, and movement tends to be very deliberate but as the patient begins to improve, we can start bombarding the CNS with other sensory information and motor tasks to achieve this subconscious level of control. For example, once the patient is able to balance on a wobble board we can then give them other tasks to do such as throwing a ball while maintaining balance.

Receptors, as well as having different functions, also have different properties. For example they may react at different speeds and can be rapidly or slowly adapting (Barrack et al. 1994; Borsa et al. 1994). The impulse from a rapidly adapting receptor drops off quickly, whereas that from a slowly adapting receptor fires for longer periods. Some examples are presented in Table 13.7.

Importantly, many joint receptors are also only active at the beginning and ends of range. Therefore, as a joint moves through a range of movement it is reliant on other receptors to keep the CNS informed of its activity. Particularly important in maintaining this function are the muscle spindles (Figure 13.15).

The large volume of proprioceptive information entering the CNS is utilised at three different levels.

- *Spinal level*. Reflex contractions occurring at this level contribute to reflex stability within a joint, helping to reduce the risk of injury from sudden forces acting on the joint.
- *Brainstem*. This is the part of the brain which receives input from the vestibular centres in the eyes and ears and helps to control balance.
- *Motor cortex, cerebellum and basal ganglia*. Responsible for control of complex movement patterns.

Instability

The word 'instability' is frequently encountered when dealing with patients who display reduced proprioception. Instability can effectively be divided into two types.

- *Structural (mechanical) instability*. Disruption of the passive control system (articular and ligamentous) of a joint results in instability. This is the type of instability detected by manual testing, for example valgus stress testing at the knee to test the medial collateral ligament or an anterior drawer to test for shoulder instability.
- *Functional instability*. Passive control systems are intact with no laxity detected through manual testing. The patient will, however, complain of symptoms such as 'giving way' (lower limb) or 'pain' and 'dysfunction' (upper limb). The problem is one of poor neuromuscular control and was initially described by Freeman et al. (1965) with reference to ankle inversion injuries.

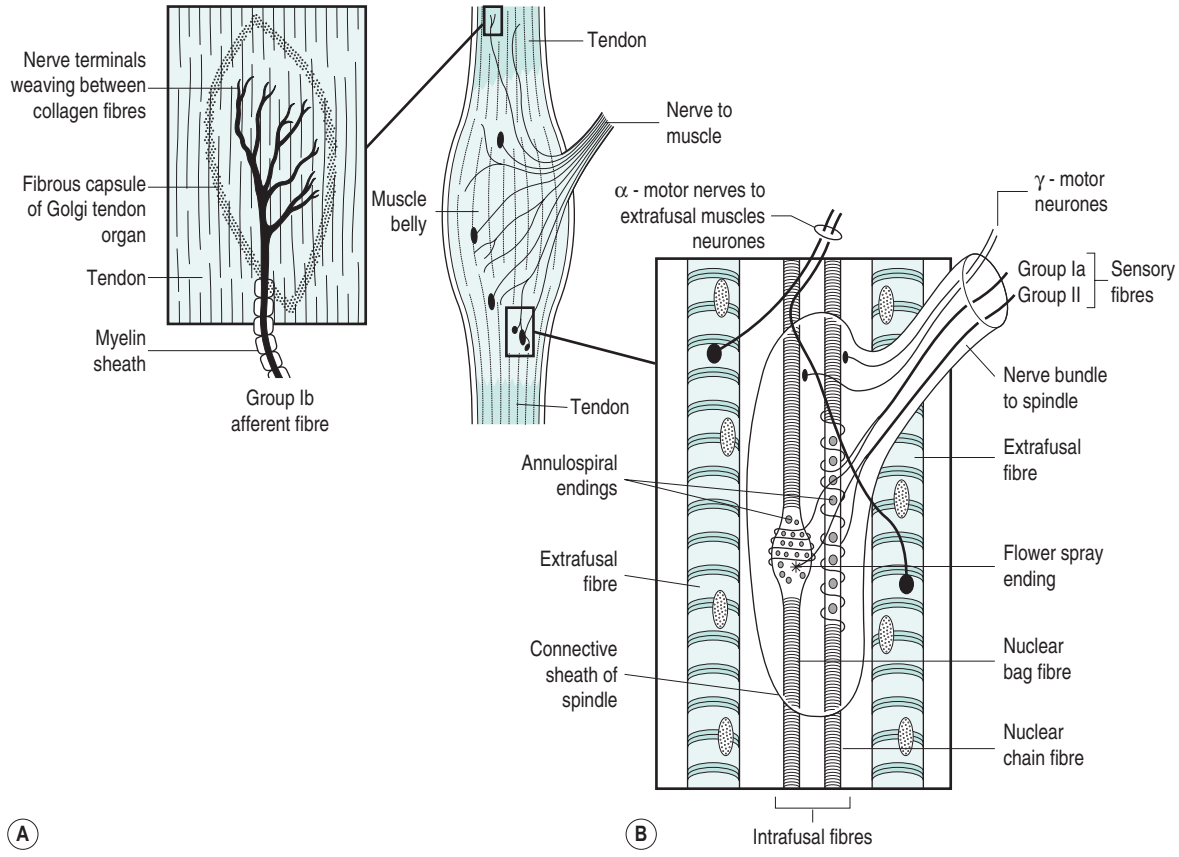


Figure 13.15 (a) The Golgi tendon organ and (b) the muscle spindle.

The above definitions illustrate the fact that joint stability is multifactoral and relies significantly on dynamic, as well as passive, control (Waddington and Shepherd 1996). This is a key point and has major implications for the rehabilitation process. Functional instability is, however, the type of instability most commonly encountered by physiotherapists. Work on ankle injuries has shown that the majority of patients suffer from functional instability only (Richie 2001). The potential causes of functional instability are presented in Table 13.8.

Correcting functional instability is the primary aim during functional rehabilitation (Lephart et al. 1997) and it is particularly important before undertaking rapid dynamic activities, such as sudden deceleration, which are seen in many sports, but, just as importantly, may also be seen in many occupations.

It is possible in some situations to reduce or even alleviate the symptoms of structural instability through appropriate neuromuscular training. More often a referral for surgery may be necessary to correct the structural defect. If functional instability is not addressed, chronic injury syndromes can develop.

Key point

Individuals suffering from structural instability are also likely to develop functional instability – a breakdown in the sensorimotor control loop results in lack of muscular control around the joint. Those suffering from functional instability can eventually develop structural instability owing to abnormal movement patterns placing excessive loads on the body's tissues.

Assessing proprioception

Assessment of proprioception takes several forms, many of which rely on complex equipment, such as force plates or isokinetic dynamometers. Force plates can measure postural sway and, therefore, adaptive responses to movement tasks. Isokinetic dynamometers can measure the threshold of detection of passive movement, for example in the shoulder complex the blindfolded subject needs to indicate when they sense movement in the glenohumeral joint. This value can be compared with the unaffected side.

Table 13.8 Potential causes of functional instability within a joint

Cause	Rationale	Reference
Articular de-afferentation	Articular mechanoreceptors are damaged, reducing the afferent impulse to the CNS, resulting in a decreased motor response	Freeman et al. (1965)
Differentiation	Trauma can result in direct damage to the motor supply, resulting in a decreased muscular response to perturbation	Wilkerson and Nitz (1994)
Neurogenic inflammation	Thought to be related to joint inflammation and inflammatory mediators directly affecting the motor endplate and therefore the motor response	Wilkerson and Nitz (1994)
Capsular distension	Joint effusion following trauma can cause muscle inhibition, leading to instability	Wilkerson and Nitz (1994)

Similarly, subjects can be asked to position the affected limb in the identical position as the opposite one or asked to reproduce a movement on the same side, again measured on the isokinetic dynamometer. There is, however, currently a lack of validated tests that can be used easily within the clinical setting.

REHABILITATION OF SENSORIMOTOR CONTROL OF THE LIMBS

Issues

The key aims of rehabilitation to sensorimotor control are:

- to provide early afferent input to a joint – this will limit further damage and reduce further losses through inactivity;
- to restore reflex stability – this is the coordinated, co-contraction of all stabilising muscles around a segment when called upon to move;
- to restore normal neuromuscular coordination – this is the correct sequencing of muscle activity necessary to perform normal movement;
- to enhance the neuromuscular response to facilitate control through the sensorimotor control loop.

Early commencement of proprioception can sometimes be neglected in rehabilitation. In the case of the lower limb, when partial weight-bearing at least is sometimes incorrectly seen as a prerequisite of proprioceptive training. Wobble board work in sitting is an exercise that can be started early and progressed (Figure 13.16). Sensorimotor control exercises need to be functional and are usually a combination of open and closed kinetic chain exercises. Closed kinetic chain exercises are useful in that they gain co-contraction of stabiliser muscles and hence segmental control, and in the fact that they give high quality proprioceptive feedback as the fixed distal segment gives a reference point for the CNS to work from.

Figure 13.16 Wobble board work in sitting.

There is a wide variety of equipment available to facilitate rehabilitation of the sensorimotor system. There are numerous types of balance boards, starting with simple unidirectional rocker boards to swiss balls. Wobble boards are a good way of bombarding the CNS with afferent input in the early stages of rehabilitation, but they are not a functional piece of equipment and it is important to progress from these types of apparatus as required. Other

equipment, such as low friction surfaces, can be useful acquisitions to enhancing dynamic functional stability. It should also be borne in mind that this equipment could be used just as effectively in the upper limbs as in the lower ones. The only limit to devising proprioceptive exercises should be your imagination.

There are several means available to the physiotherapist to progress proprioceptive exercises. These include:

- removing visual stimulus;
- altering the base of support;
- increasing weight-bearing;
- increasing speed of an activity;
- making an exercise more complex, e.g. adding perturbation (an unexpected external force, e.g. a push).

Visual input provides additional afferent input to the CNS. Without it any given exercise becomes more difficult to perform as the subject will have to rely more heavily on proprioceptive information, even with wobble board activities in sitting.

An example of altering the base of support is illustrated in Figure 13.17. This activity progressively requires greater neuromuscular control and strength to maintain a given position.

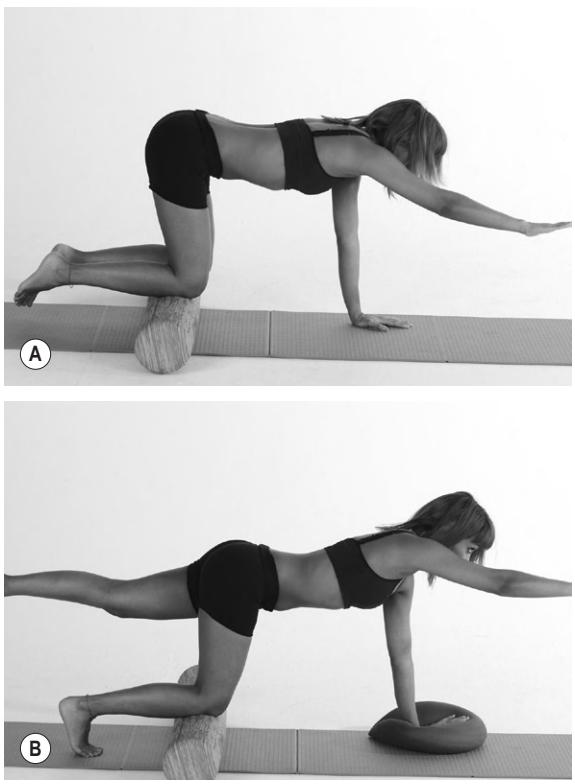


Figure 13.17 (a, b) Altering the base of support with motor control exercises.

Key point

It is important during rehabilitation to place the joint(s) in situations that encourage the necessary reflex stability required to meet the functional demands of the individual. Stability work should therefore be progressed to place joints in more challenging and functional positions.

The lower limb

Proprioceptive exercises in the lower limb in the early stages of rehabilitation are progressions towards full weight-bearing. In the latter stages, more dynamic stop–start activities that require aspects of eccentric control with acceleration, deceleration and strong reflex stability around the ankle and knee can be added. As detailed above, they can be progressed in several ways (see section on progression of exercise).

Figure 13.18 illustrates a typical exercise for the lower limb. It is important to remember that this is not a particularly functional exercise as the limb remains static, so

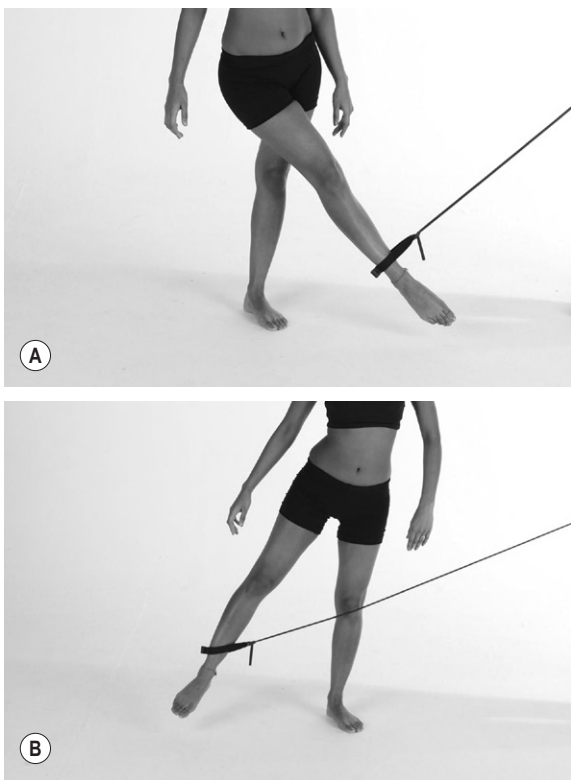


Figure 13.18 Maintain contraction during lower limb movement.

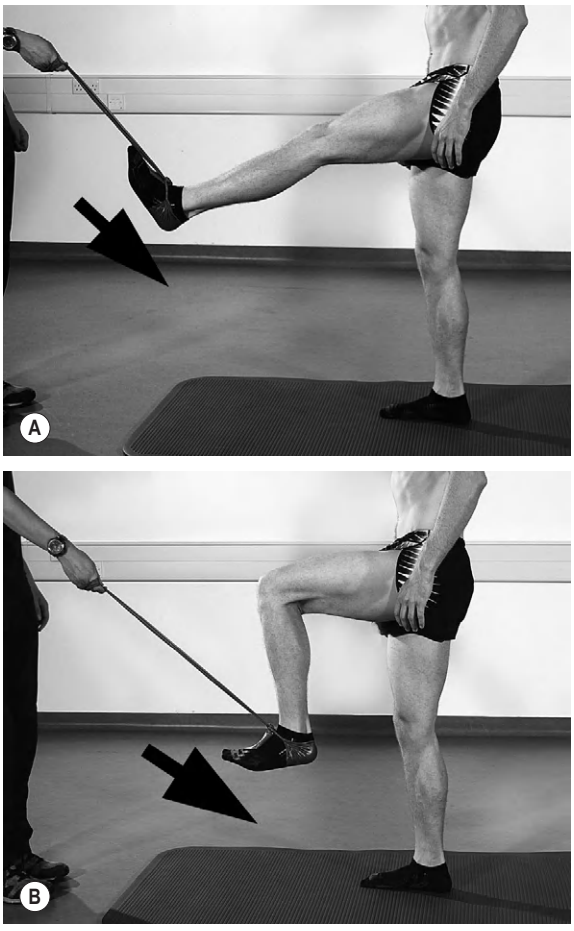


Figure 13.19 Eccentric control of hip extension using rubber tubing. The speed of the exercise and resistance of the tubing can be changed to suit the aims of the exercise and increase specificity.

will require progression by the end-point of rehabilitation. The more dynamic activities outlined above can be described as proprioceptive as they stress the joints in challenging positions and encourage the reflex actions that are required.

The upper limb

Key point

The objective of proprioceptive rehabilitation is to 'enhance cognitive appreciation of the respective joint relative to position and motion, and to enhance muscular stabilisation of the joint in the absence of structural restraints' (Borsa et al. 1994).

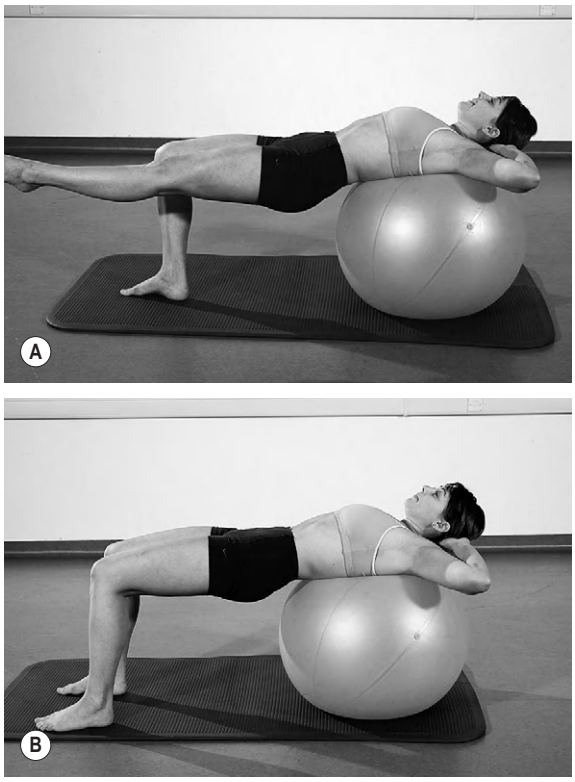


Figure 13.20 Example of lower limb motor control exercises.

The above quotation refers to rehabilitation of unstable shoulders and perhaps highlights the potential differences in the nature of pathologies between the upper and lower limb. The shoulder is inherently more unstable than the joints of the lower limb and is therefore much more vulnerable to mechanical, as well as functional, instability. Consequently, a comprehensive assessment needs to be undertaken when rehabilitating the shoulder to ascertain the exact cause of the problem.

Owing to this potential level of instability, close-chain exercises are often a useful starting point, as they help to encourage joint stability through co-contraction of the surrounding muscles induced by the axial compression (Davies and Dickoff-Hoffman 1993). Variations of the four-point kneeling position are commonly used to achieve this (Figure 13.17). These positions are also useful in rehabilitating scapula stability, which will also be affected with (or be a cause of) many shoulder problems.

When rehabilitating older patients or those with a poorer physical condition, four-point kneeling may not be an appropriate position and variations of closed-chain exercises can be used. These can include resting the hand on a wobble board in sitting or resting the hand against a

wall or table to allow a degree of weight-bearing and then moving the shoulder.

The reflex stabilisations can also be used and are aimed at eliciting the correct motor response at the appropriate speed and in the most functional position. These consist of a sustained isometric contraction in a specific range in the joint's motion with the scapula in the correct position. These particular exercises and variations of them could be used with an athlete who would require shoulder stability in a vulnerable position, for example a rugby player when making a tackle.

Once a degree of control has been restored, more functional exercises can be taught. These may be occupation- or sports-specific to ensure the patient can withstand the stresses they encounter during their lives.

Rehabilitating the sensorimotor system requires adequate strength and endurance within the muscle. Many of the exercises illustrated can also be viewed as strengthening exercises. Although much of the preceding discussion has emphasised sensorimotor rehabilitation in isolation, the need for an accompanying strength and endurance programme is paramount. Indeed, muscular fatigue has been shown to induce proprioceptive deficits (Voight et al. 1996).

There is no definitive way to rehabilitate the sensorimotor system and issues of progression depend on functional need, pain, swelling and an appropriate strength base.

REHABILITATION OF SENSORIMOTOR CONTROL OF THE SPINE

The spine needs an effective neuromuscular control system and also benefits from sensorimotor control rehabilitation. The term 'core stability' is used with reference to back rehabilitation – 'core' referring to the trunk as the central control point of all movement. Research has shown that the muscles that stabilise the spine can become defacilitated and wasted (Hides et al. 1996) and have delayed recruitment during functional movements (Hodges and Richardson 1996). Loss of control of the core is hypothesised to lead to loss of segmental stability owing to loss of core stability mechanisms. These mechanisms include increasing of intra-abdominal pressure (Cresswell et al. 1992) and a stabilising effect through tensioning the thoracolumbar fascia (Hodges and Richardson 1997). If core stability is impaired it could have a detrimental effect on many other anatomical structures along the kinetic chain. This may be proximally in the pelvic and shoulder girdles, or more distally in the limbs. Dynamic postural control requires reflex stabilisation of the trunk musculature that controls core stability. Specific transversus abdominus and multifidus re-education is required, and progressed into dynamic movements and then function. The emphasis on maintaining a neutral spinal position during exercise is necessary (Panjabi 1992), thus reducing shear forces around the spinal segments and facilitating stabiliser activity. Swiss-ball exercises are commonly used to help restore this dynamic control (Figures 13.21 and 13.22).

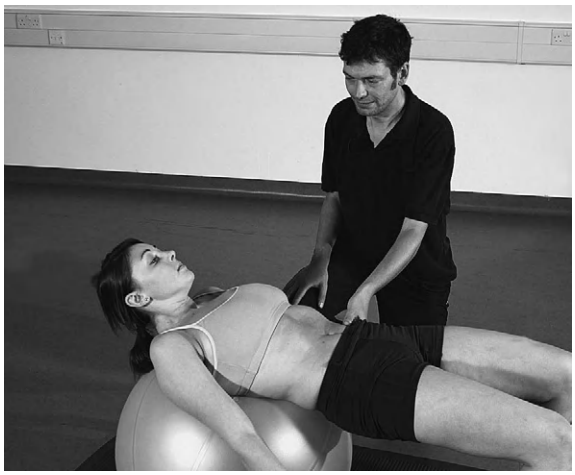


Figure 13.21 Swiss ball exercise designed to develop neuromuscular control around the trunk.

Figure 13.22 Swiss ball exercise aiming to integrate trunk and hip control.

Key point

Panjabi (1992) uses the phrase 'clinical instability' when referring to a decrease in the effectiveness of the passive and dynamic stabilisers within the spine.

Conscious appreciation of spinal position and associated muscle activity has been aided by the use of equipment such as the Stabiliser pressure biofeedback device (Chattanooga, Australia). This is a simple device consisting of an air-filled chamber and an attached dial that monitors pressure changes. Patients are instructed to perform exercises while maintaining a constant pressure reading or altering the pressure through exercise. (Figure 13.23).

Pain is often the major concern in these patients and a neuromuscular training programme will aim to decrease or alleviate pain. The mechanisms of spinal pain are, however, varied and often poorly understood.

Core stability should not be confused with core strength, as is commonly the case. Core stability refers to a level of low threshold activity in the trunk stabiliser muscles, which results in functionally stable sensorimotor control of the trunk. Core strength refers to the effective production of torque in the trunk muscles and does not relate to finer levels of sensorimotor control. To read further on core stability see Chapter 14.

Figure 13.23 Pressure Biofeedback maintain correct degree of deep abdominal contraction.

Figure 13.24 Pole rotation resisted by rubber tubing to enhance trunk control.

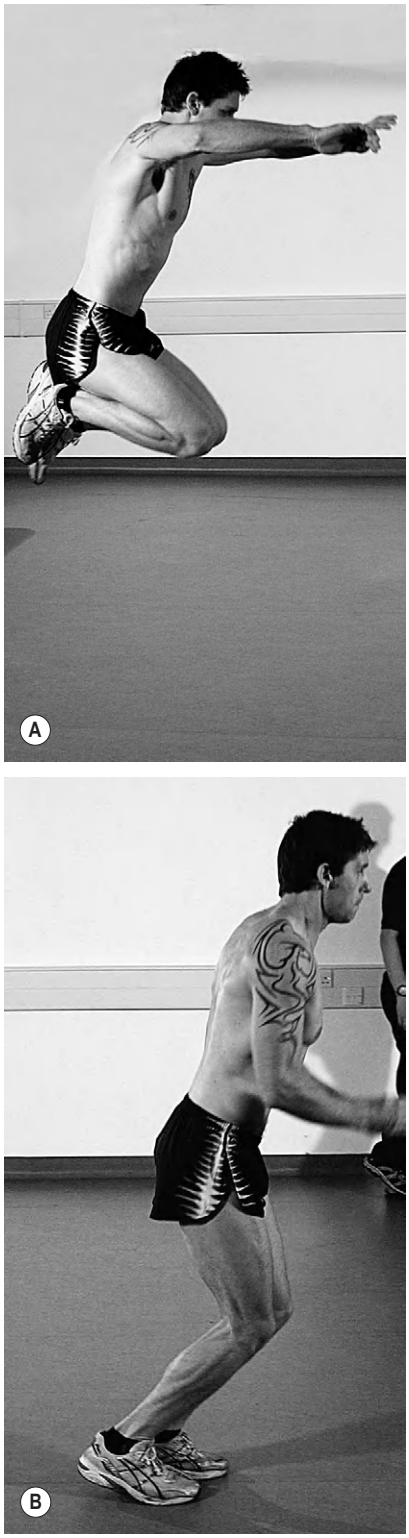


Figure 13.25 (a, b) A plyometric drill for the lower limb.

PLYOMETRIC EXERCISES

Plyometric exercises are normally incorporated in the later stages of a rehabilitation programme and are aimed predominantly at those individuals who require more dynamic neuromuscular control and force generation as part of their normal functional activities, for example athletes. Plyometrics are also used as part of athletes' training programmes, as they have been found to enhance performance (Sinnett et al. 2001, Swanik et al. 2002).

Plyometric exercises utilise the stretch-shortening cycle to produce an enhanced concentric contraction. A plyometric activity consists of a rapid eccentric contraction followed by an immediate concentric contraction. Typically, these exercises are performed in the lower limb and involve various jumping-type activities. They can also be performed in the upper limb. Successful performance involves a rapid change from the eccentric to the concentric contraction. The time between the eccentric and concentric contractions is known as the *amortisation phase* – the quicker this phase the more powerful the muscular concentric contraction.

Consider the jumping activity shown in Figure 13.25.

As the feet hit the floor the knees flex and result in a stretch in what is known as the series elastic component. The series elastic component consists of three structures:

- Z lines;
- myosin hinges;
- Sharpey's fibres (located at the site of tendon insertion into bone).

Acting together, the series elastic components behave like an elastic band that is stretched quickly – it stores up energy that can be suddenly released. This energy is added to the stretch reflex that occurs in the quadriceps on landing and, together with the following concentric contraction, combines to provide a more powerful contraction. This type of activity is utilised in sport particularly in the running gait cycle.

Plyometrics are used in the upper limb and certainly have functional applications. Medicine-ball drills are often incorporated into upper-limb exercises, as are activities with rubber tubing. Medicine ball work can involve paired throwing activities or use of a trampoline (Figure 13.27).

The stated benefits of plyometric exercise are:

- increased power of muscular contraction;
- enhanced neuromuscular coordination.

Before deciding to use plyometric exercises in the clinical situation, this checklist should be considered:

- Is the person free of pain?
- Is there an absence of recurrent swelling?
- Does the person demonstrate a sufficient level of stability?

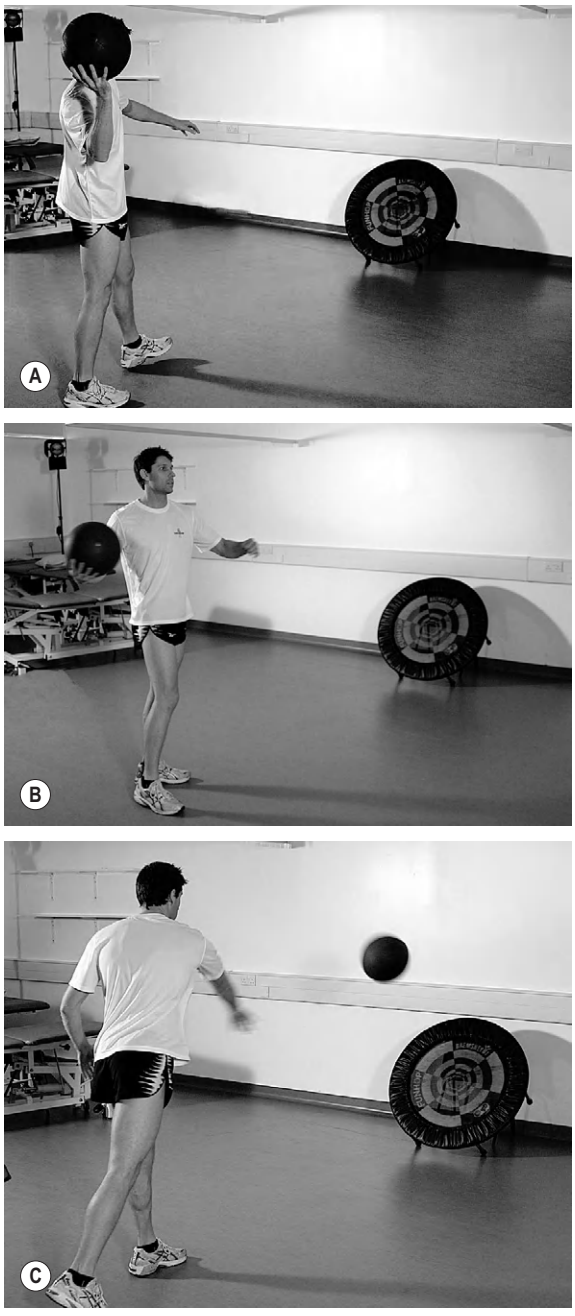


Figure 13.26 Plyometric drill using a trampette.

Despite the emphasis of plyometrics being on advanced rehabilitation, it can also play a role in improving the neuromuscular response following injury.

Latency of contraction is commonly seen in muscles following an injury, for example the peronei in an ankle sprain. Using rubber tubing, the principles of plyometrics

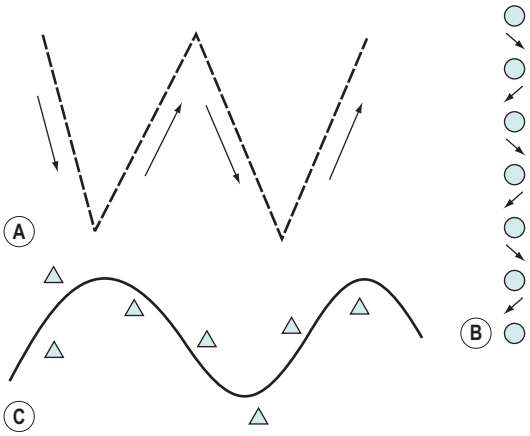


Figure 13.27 Example of a timed circuit.

can be used to work on improving the neuromuscular response and strengthening the peronei at the same time. The patient takes the strain on the rubber tubing, holding the foot in an everted position. The patient is then instructed to release the tension on the rubber and then immediately evert the foot again. The condition of the ankle needs to be carefully assessed before commencing this type of exercise, in the same way as the inclusion criteria are checked for weight-bearing exercises. Similar activities could be used in the upper limb for shoulder medial rotators (Swanik et al. 2002).

There is little information on the number of repetitions that should be completed with plyometric exercises. Foot contacts have been suggested as an effective way of measuring the intensity of activity (Tippet and Voight 1995) in lower limb exercise, with a beginner counting 75–100 foot contacts and progressing up to 200–250 of low-to-moderate intensity. No values have been stated for the upper limb. Common sense should also prevail in monitoring performance, looking out for loss of quality of performance, obvious fatigue and an increase in length of the amortisation phase, etc.

Plyometric exercises should be performed no more than 2–3 times per week. The eccentric component is likely to cause a degree of DOMS and the muscle will need adequate time to recover.

FUNCTIONAL TESTING AND REHABILITATION

All rehabilitation should be geared towards a return to normal functional activity. Towards the later stages of rehabilitation, the functional aspects are, perhaps, of greatest importance.

Rehabilitation should not be measured as complete solely by outcomes such as bilateral symmetry of strength and range of movement, or an individual's pain scores, but on their return to full function – whatever that might be. The physiotherapist needs to have a clear insight into the specific vocational, occupational, sporting and functional requirements of an individual to make sure the person is rehabilitated to the correct level. Such demands may involve working with large loads, working in challenging environments or playing professional sport. The challenge for the physiotherapist is to have an insight into that person's requirements and devise a rehabilitation programme to meet those specific needs.

Functional testing is an extension of functional rehabilitation and plays a major role in determining an individual's ability to return to pre-injury levels. This can take the form of one particular drill that encompasses several of the most difficult aspects of the individual's occupation or sport. For example, a soccer player needs to be able to run quickly, accelerate and decelerate, change direction and jump. A circuit could be set up to test any number of skills. This circuit could be timed over a number of sessions to determine improvement or may simply be used as a one-off test to determine a return to full training. Tests will be specific to their requirements, for example in sport they may want to test strength, endurance or agility depending on the specific nature of the sport or even be position-/role-specific.

Functional tests can be used as a measure of progress and are commonly employed as a requirement for return to work in physically challenging occupations or a return to play in sport. A functional test will establish a baseline to measure future improvement, identify risk factors predisposing to injury or reinjury and provide motivation to the individual as it is a measurable outcome. When assessing functional tests, the individual's ability to be able to respond to and tolerate functional demands is observed. The way the body part contributes to the body's overall movement and sensorimotor performance is also assessed.

There are several validated and widely used functional performance tools in existence but it may be necessary to design a functional test. If this is the case careful planning is required. The first consideration is to identify the appropriate task, whether it is of a strength, endurance, motor control or sports-specific nature. It will then need to be decided in what environment the task needs to be performed in – replicating functional environments enhances specificity. Once the tasks are established it is necessary to plan how they will be measured and what equipment will be required for this, for example video, surface electromyography or a stopwatch. Once the planning has been completed all that remains is to perform the test, collect, analyse and interpret the data. Analysis should consider performance of all phases of the whole task, for example video analysis should include observation of starting

Key point

When devising functional tests it is useful to apply the SMART principle: specific, measurable, achievable, realistic, timed. In this way, they can be developed as a valuable objective measure. There is a large scope for development of such tests, but it is also important that once such tests are devised they are seen to be reliable and valid.

position, preparatory position, approach, execution of the task and termination of activity, for example landing. Once the task has been analysed all that remains is interpretation and analysis on how the individual has shown improvement and if this is sufficient to withstand the rigors of return to competition or work.

Functional rehabilitation allows a more objective measure of fitness to return to work. However, unless working in a specialist sporting environment or occupational centre such as an army rehabilitation centre, being able to completely duplicate a particular working environment as described above may not always be possible. In this situation, the physiotherapist needs to be inventive and reproduce the tasks to be undertaken in the working environment as closely as possible, using any equipment or apparatus that may be available. This can include benches, wall bars, air cushions, rubber tubing, rucksacks and sports-specific equipment.

Functional testing does not always need to be used as a measure for determining return to full function; instead, it may be used as a baseline measure of functional ability. Examples of this include the various timed and distance hop tests which are used to test ACL-deficient knees. Tippet and Voight (1995) suggest the following times when functional testing is undertaken within a sports environment:

- pre-season training;
- during rehabilitation;
- immediately after injury.

Although the examples highlighted above involve quite dynamic activities, functional testing and rehabilitation can be applied to all populations. An example of functional testing in the elderly population is the sit-to-stand test (Figure 13.28) or a walk of fixed distance. This can be either timed or judged on the number of repetitions.

Functional testing also provides psychological, as well as physical benefits, not only in confirming progress but improving confidence levels. It must be remembered that

Figure 13.28 The sit-to-stand functional test. An example of a simple functional test requiring no equipment.

there is no substitute for 'live' conditions, such as performing in front of a hostile crowd, and functional testing can only go so far in preparing individuals for full function.

GROUP EXERCISE

Group exercise sessions are used widely, especially with more recent moves towards physiotherapists working in primary care as part of their educational or advisory role (Crook et al. 1998). Group work can take the form of exercise classes in a gym, in a hydrotherapy pool or as part of an educational programme during which patients not only exercise but are also informed about the nature of their condition (Figure 13.29).

Educational programmes are commonly employed with the more longstanding pathologies and they encourage patients to take responsibility for the continued management of their condition (e.g. back education programmes or osteoarthritis knee schools).

There are both advantages and disadvantages to group work (Table 13.9).

Benefits and drawbacks

Provided that members of a group are carefully selected there are several advantages to treating patients in groups. Exercising with patients who have experienced similar problems can provide peer support, encouragement, reassurance and camaraderie. It is also more economically effective with the therapist able to supervise several people at once.

The disadvantages of group work are that certain individuals may not respond to the group environment. In particular, they may be embarrassed or dislike the interaction. Many clients will respond better to individual attention from their therapist, particularly in the early stages of rehabilitation; this is not possible in group work.

Figure 13.29 Group exercises in a pilates class.

If groups are used solely as a way of treating large numbers of patients with diverse pathologies they will prove ineffective. This factor, along with a poorly organised treatment session, could lead to demotivation. There need to be clear objectives and goals for each class, with defined inclusion criteria.

Finally, it is important that participants in the group do not become too competitive, thereby reducing the effectiveness of the exercise and also risking further injury.

Planning group work

Careful planning is necessary if group work is to be effective. The aims of the group session must be clearly planned and stated so that selection criteria for participants can be agreed. This will ensure the session is both safe and effective. Factors such as age, gender, psychological status, the stage in rehabilitation, past medical history and general fitness need to be considered. The facilities and equipment that are available need to be appropriate for the activities planned. Important considerations are the space available and the layout and temperature of the room or gym, not only to provide a suitable environment but also a safe place to exercise.

Table 13.9 Advantages and disadvantages of group exercises

Advantages	Disadvantages
Competitive element can be useful in increasing a person's performance	Difficult to pitch the exercises at a level suitable for all group members
A variety of exercises is possible	Temptation to put inappropriate individuals in the group to save time and relieve overburdened staff
Group exercise can be fun if properly organised	Difficult to monitor all of the people all of the time
Helps the individual to feel less isolated if meeting people with similar problems	Difficult to progress all the members of the group appropriately
Provides a good opportunity for the physiotherapist to educate and inform the group about the condition	Competitive element may be counterproductive or dangerous
Specialist groups such as ankylosing spondylitis or cardiac rehabilitation groups provide social support	Some people do not respond well in a group situation

Format of a group session

The format of a group session should include:

- patient assessment for suitability for group exercise (at an individual appointment);
- a warm-up session;
- a main exercise session;
- a cool-down period.

A warm-up is commonly used to start a session to improve circulation and increase body tissue temperature, therefore physically, as well as mentally, preparing the participants for exercise. Warm-up exercise may also limit the build-up of metabolites and subsequent acidosis during intense exercise (Kato et al. 2000). It is also interesting to note that proprioception has been found to be significantly more sensitive after warm-up (Bartlett and Warren 2002).

Following the warm-up come the main exercises. It is also important to allow time for a cooling-down, particularly following vigorous exercise, to assist with removal of lactic acid and waste products of metabolism.

Circuit training

Circuit training is often used in group work. Circuit training involves an exercise programme in which exercises are performed in successive stations with either a predetermined number of repetitions or for a set duration. Timed rest follows each exercise period prior to moving to the next station.

The exercises forming the circuit could be chosen from any of the aforementioned exercise types. The exercises included as part of the circuit must each form part of the client's overall rehabilitation goals. There will also usually be a cardiovascular element to circuit training and it is commonly used for cardiac or pulmonary rehabilitation programmes.

The order of exercises in a circuit needs to be planned from a safety aspect so that group members do not endanger each other. Exercises also need to be ordered to achieve optimum effectiveness. For example two exercises targeted to achieve similar effects should not be placed adjacently on a circuit as that might cause excessive fatigue of the muscle groups involved unless this is the desired effect.

Key point

During group work the physiotherapist facilitates the session by giving feedback and motivation to all participants to ensure that they benefit fully from the session. It is also important that the participants have a home exercise programme to ensure their progress is continued at home.

HYDROTHERAPY

Hydrotherapy involves exercising in water. The same principles of exercising on dry land discussed in this chapter are generally true when exercising in water. It is normal to have clients performing individually-devised exercises concurrently under supervision to gain the advantages of group activity and maximal use of this often expensive resource without the drawbacks of a class setting. However, this still requires careful planning.

Hydrotherapy is used in the treatment of a wide range of conditions to enhance cardiovascular fitness, to mobilise, to strengthen, to co-ordinate movement and to regain function of the neuro-musculoskeletal system. Many hospital departments have purpose-built heated hydrotherapy pools. The warm environment may allow the muscles to work more effectively owing to a rise in temperature and of the relaxation of any muscle spasm. It will also have a pain-relieving effect. Conditions commonly managed by hydrotherapy are wide ranging, from rheumatological conditions such as rheumatoid arthritis and ankylosing spondylitis to trauma cases and neurological conditions.

Contraindications and precautions are important when considering patients for any exercise programme and more so with hydrotherapy owing to potential emergency evacuation situations. This is mainly because of the warm environment in which they are exercising and the dangers of slipping or drowning. Contraindications include the presence of certain medical conditions such as recent, or severe, neurological conditions (including uncontrolled epilepsy), certain cardiovascular problems and kidney failure. Hydrotherapy is also contraindicated with debilitating disease and the presence of infections which may be exacerbated or risk transmission to other patients.

The starting positions used to exercise in the pool will be different from those commonly used on dry land. Patients commonly exercise in standing or sitting in the pool or perform more dynamic exercises such as walking or swimming, but they can also be treated while floated in lying. Floats are placed around the neck and waist to support the patient, allowing the patient to exercise freely. Floats may also be placed around the limbs.

The factors that need to be considered, other than starting position, that change the nature of the exercise are those of buoyancy, turbulence and streamlining.

Buoyancy

This results from the relative density of the body, or body part, and the higher density of water. Buoyancy results in an apparent loss of weight of the object when placed in the water and it may be used to either assist or resist movement. Buoyancy will be of particular advantage in

reducing the effect of gravity on the body, particularly on load-bearing joints such as lower back, hips and knees. Buoyancy may increase the function or the range of movement that is possible, for example hip and knee flexion in standing. It may also be utilised to increase range of movement as a mobilising exercise, an effect that can be further enhanced by the use of a float or inflatable wrist or ankle bands, for example to mobilise shoulder flexion with a wrist float in sitting.

Buoyancy can also be used in strengthening exercises. *Buoyancy-resisted* exercise involves pushing against buoyancy. The effect can be increased by again adding floats, for example hip extension while floated in supine against an ankle float.

Key point

The greater the buoyancy of the float the greater the mobilising effect of the exercise. These types of exercise are classed as *buoyancy-assisted*.

As the exercises are progressed, the inflatable bands can be further inflated or, alternatively, the position of the float on the lever can be adapted therefore changing the buoyancy effect on the limb, i.e. the effect will be increased with a distal float placement. If buoyancy is to be counterbalanced the patient will need to exercise along the pool surface.

Turbulence

As limbs move through water they meet resistance and turbulence is created, resulting in the production of currents. Turbulence results in an area of low pressure behind the moving body or body part. Faster movements will produce more turbulence. These currents may act to make movement more difficult and so this principle is of value when progressing an exercise, for example if exercises are performed rapidly more turbulence will be created and will prove more difficult to move though. Also, therapist-created turbulence can be used in re-education of movement, particularly in the weight-bearing muscles of the lower limb. The client is subjected to turbulence during standing and must maintain their position. This can prove very difficult and can be quite an advanced exercise.

Streamlining

This refers to the surface area of the body part exposed to the water when moving through it. The simplest example is the orientation of the upper limb during exercise. If the hand moves with the palm facing the resistance of the water more effort is required than when the limb is rotated so that the ulnar border leads. Moving from streamlined to non-streamlined positions can be used to progressively strengthen. This principle can be further progressed by the use of hand-held bats or by placing flippers on the feet.

Further study

For information on specific treatment techniques such as Bad Ragaz and Halliwick, further reading or study is required.

EXERCISE PRESCRIPTION AND COMPLIANCE

In this chapter we have considered many types of exercise for varying populations and conditions. For prescription of exercise to be effective as a treatment modality, selection of the programme needs to be appropriate, as does how the exercises are delivered. The way in which exercises are taught will enhance their effectiveness by promoting compliance and accurate performance.

It is good practice when teaching exercise, as with any treatment intervention, to gain informed consent. Not only does this conform to legal requirements but also it serves to educate the patient on their condition presenting a rationale behind the treatment approach. Education can only enhance compliance with the exercises (Lee and Yoo 2004), as can active encouragement (Prasad and Cerny 2002). When teaching the exercises it may help to demonstrate to the patient the intended exercise before giving detailed instruction and feedback on performance. There is no point in allowing an exercise to be performed incorrectly as it will not effectively achieve its intended goal. It may benefit the patient to be given a clear, written copy of their exercises, using lay terms; however, this should be a personalised exercise programme. All too often patients are given pre-printed sheets with little thought to individual requirements. Written instructions should also include information on how to perform the exercise, for example speed, the number of repetitions, sets and duration of recovery, and also frequency (how many times a day). It is important for the therapist to closely observe the performance of exercise to detect for signs of substitution or fatigue. Substitution strategies may present with movement dysfunction and it is necessary to inhibit overactive muscles to correct abnormal recruitment patterns so as not to reinforce dysfunction. Fatigue will also affect performance so once early signs of fatigue are noted, it is best to allow the muscles to rest and recover prior to performing further sets.

Feedback is necessary, but should be given in a way that does not reduce the effectiveness of your treatment causing

demotivation. Feedback should also be used positively to reinforce good performance. A positive outlook, high quality education and constructive feedback will all serve to improve motivation and, in turn, a patient that is highly motivated will be more likely to comply with their exercise programme.

As a final point it is also good practice to give a patient a home exercise programme, to review it on each consultation and progress it as the patient's improvement allows. This will give good returns in rehabilitation rates as the patient can actively participate regularly in the recovery and maintenance of their condition.

Key point

For exercise to be successful ensure:

- that patients have clear instructions;
- that patients receive feedback on performance;
- that patients have been educated on the reasons behind their exercise;
- to give regular constructive feedback and encouragement.

ACKNOWLEDGEMENTS

With thanks to Sean Kilmurray, University of Central Lancashire, for his original contribution and Paul Cieplak (MSc advancing physiotherapy student, University of Salford) and Sante Saleh (BSc Honours physiotherapy student, University of Salford).

FURTHER READING

Crook, P., Stott, R., Rose, M., et al., 1998. Adherence to group exercise: physiotherapist-led experimental programmes. Physiotherapy 84, 366–372.

Jerosch, J., Prymka, M., 1996. Proprioception and joint stability. Knee Surg Sports Traum Arthrosc 4, 171–179.

Kisner, C., Colby, L.A., 1996. Therapeutic Exercise: Foundations and Techniques, third ed. FA Davies, Philadelphia.

Lieber, R.L., 1992. Skeletal Muscle Structure and Function: Implications for Rehabilitation and Sports Medicine. Williams & Wilkins, Baltimore.

Mense, S., Simons, D.G., Russell, J. (Eds.), 2001. Muscle Pain: Understanding its Nature, Diagnosis and Treatment. Lippincott Williams & Wilkins, Philadelphia.

Richardson, C., Hodges, P.W., Hides, J., 2004. Therapeutic Exercise for Lumbopelvic Stabilization. A Motor Control Approach for the Treatment and Prevention of Low Back Pain, second ed. Churchill Livingstone, London.

Richie, D.H., 2001. Functional instability of the ankle and the role of neuromuscular control: a comprehensive review. J Foot Ankle Surg 40, 240–251.

Tippet, S.R., Voight, M.L., 1995. Functional Testing in Functional Progressions for Sport Rehabilitation. Human Kinetics, Champaign, IL.

REFERENCES

Bandy, W.D., Irion, J.M., Briggler, M., 1997. The effect of time and frequency of static stretching on flexibility of the hamstring muscles. Phys Ther 77, 1090–1096.

Barrack, R.L., Lund, P.J., Skinner, H.B., 1994. Knee joint proprioception revisited. J Sport Rehabil 3, 18–42.

Barrett, D.S., Cobb, A.G., Bentley, G., 1991. Joint proprioception in normal, osteoarthritic and replaced knees. J Bone Joint Surg 73B (1), 53.

Bartlett, M.J., Warren, P.J., 2002. Effect of warming up on knee proprioception before sporting activity. Br J Sports Med 36 (2), 132–134.

Bennett, M., Best, T.M., Babul, S., et al., 2005. Hyperbaric oxygen therapy for delayed onset muscle soreness and closed soft tissue injury. Cochrane Database Syst Rev (4) CD004713.

Borsa, P.A., Lephart, S.M., Kocher, M.S., et al., 1994. Functional assessment and rehabilitation of shoulder proprioception for glenohumeral instability. J Sport Rehabil 3, 84–104.

Cahill, B.R., Misner, J.E., Boileau, R.A., 1997. The clinical importance of the anaerobic energy system and its importance in human performance. Am J Sports Med 25, 863–872.

Caraffa, A., Cerulli, G., Projetti, M., et al., 1996. Prevention of anterior cruciate ligament injuries in soccer: a prospective controlled study of proprioceptive training. Knee Surg Sports Traum Arthrosc 4, 19–21.

Chan, S.P., Hong, Y., Robinson, P.D., 2001. Flexibility and passive resistance of the hamstrings of young adults using two different static stretching protocols. Scand J Med Sci Sports 11 (2), 81–86.

Cheung, K., Hume, P., Maxwell, L., 2003. Delayed onset muscle soreness: treatment strategies and performance factors. Sports Med 33 (2), 145–164.

Close, G.L., Ashton, T., Cable, T., et al., 2005. Effects of dietary carbohydrate on delayed onset muscle soreness and reactive oxygen species after contraction induced muscle damage. Br J Sports Med 39 (12), 948–953.

Cresswell, A.G., Grundstrom, H., Thorstensson, A., 1992. Observations on intra-abdominal pressure and patterns of abdominal intra-muscular activity in man. Acta Physiol Scand 144 (4), 409–418.

Crook, P., Stott, R., Rose, M., et al., 1998. Adherence to group exercise: physiotherapist-led experimental programmes. Physiotherapy 84, 366–372.

Davies, G.J., Dickoff-Hoffman, S., 1993. Neuromuscular testing and rehabilitation of the shoulder complex. J Orthopaed Sports Phys Ther 18, 449–458.

Emery, C.A., 1999. Does decreased muscle strength cause acute muscle strain injury in sport? A systematic review of the evidence. Phys Ther Rev 4 (3), 141–151.

Freeman, M.A.R., Dean, M.R.E., Hanhan, I.W.F., 1965. The aetiology and prevention of functional instability of the foot. J Bone Joint Surg 47B, 678–685.

Galper, D.I., Trivedi, M.H., Barlow, C.E., et al., 2006. Inverse association between physical inactivity and mental health in men and women. Med Sci Sports Exerc 38 (1), 173–178.

Gluck, N.I., Liebenson, C.S., 1997. Clinical implications of paradoxical muscle function in muscle stretching or strengthening. J Bodywork Mov Ther 1, 219–222.

Gralton, E., Wildgoose, J., Donovan, W.M., et al., 1998. Eccentric exercise and neuroleptic malignant syndrome. Lancet 352, 1114.

Herbert, R.D., Gabriel, M., 2002. Effects of pre- and post-exercise stretching on muscle soreness, risk of injury and athletic performance: a systematic review. Br Med J 2002;325 (7362), 468–472.

Hides, J.A., Richardson, C.A., Jull, G.A., 1996. Multifidus recovery is not automatic following resolution of acute first episode low back pain. Spine 21, 2763–2769.

Hodges, P.W., Richardson, C.A., 1996. Inefficient muscular stabilization of the lumbar spine associated with low back pain: a motor control evaluation of transversus abdominis. Spine 21, 2640–2650.

Hodges, P.W., Richardson, C.A., 1997. Feedforward contraction of transversus abdominis is not influenced by the direction of arm movement. Exp Brain Res 114 (2), 362–370.

Howell, J.N., Chlebourn, G., Conaster, R., 1993. Muscle stiffness, strength loss, swelling and soreness following eccentric induced exercise in humans. J Physiology 464, 183–196.

Hunter, G., 1998. Specific soft tissue mobilization in the management of soft tissue dysfunction. Manual Ther 3 (1), 2–11.

Jerosch, J., Prymka, M., 1996. Proprioception and joint stability. Knee Surg Sports Traum Arthrosc 4, 171–179.

Jerosch, J., Thorwesten, L., 1998. Proprioceptive abilities of patients with post-traumatic instability of the glenohumeral joint. Zeitschrift fur Orthopadie und Ihre Grenzgebiete 136 (3), 230–237.

Kato, Y., Ikata, T., Takai, H., et al., 2000. Effects of specific warm-up at various intensities on energy metabolism during subsequent exercise. J Sports Med Phys Fitness 40 (2), 126–130.

Kerrigan, D.C., Lee, L.W., Collins, J.J., et al., 2001. Reduced hip extension during walking: healthy elderly and fallers versus young adults. Arch Phys Med Rehab 82, 26–30.

Kerrigan, D.C., Xenopoulos-Oddsson, A., Sullivan, M.J., et al., 2003. Effect of a hip flexor-stretching program on gait in the elderly. Arch Phys Med Rehab 84 (1), 1–6.

Kisner, C., Colby, L.A., 1996. Therapeutic Exercise: Foundations and Techniques, third ed. FA Davies, Philadelphia.

Kujala, U.M., Orava, S., Järvinen, M., 1997. Hamstring injuries: current trends in treatment and prevention. Sports Med 23, 397–404.

Lee, S.J., Yoo, J.S., 2004. The effects of a physical activity reinforcement program on exercise compliance, depression, and anxiety in continuous ambulatory peritoneal dialysis patients. Daehan Ganho Haghoeji 34 (3), 440–448.

Leger, A.B., Milner, T.E., 2001. Muscle function at the wrist after eccentric exercise. Med Sci Sports Exer 33, 612–620.

Lephart, S.M., Henry, T.J., 1995. Functional rehabilitation for the upper and lower extremity. Orthoped Clin N Am 26, 579–592.

Lephart, S.M., Pincivero, D.M., Giraldo, J.L., et al., 1997. The role of proprioception in the management and rehabilitation of athletic injuries. Am J Sports Med 25, 130–137.

Lieber, R.L., 1992. Skeletal Muscle Structure and Function: Implications for Rehabilitation and Sports Medicine. Williams & Wilkins, Baltimore.

Lynch, D., Ferraro, M., Krol, J., et al., 2005. Continuous passive motion improves shoulder joint integrity following stroke. Clin Rehab 19 (6), 594–599.

Magnusson, S.P., 1998. Passive properties of human skeletal muscle during stretch manoeuvres. Scand J Med Sci Sports 8 (2), 65–77.

Malliaropoulos, N., Papalexandris, S., Papalada, A., et al., 2004. The role of stretching in rehabilitation of hamstring injuries: 80 athletes follow-up. Med Sci Sport Exerc 36 (5), 756–759.

Melis, P., Noorlander, M.L., van der Horst, C.M., et al., 2002. Rapid alignment of collagen fibres in the dermis of undermined and not undermined skin stretched with a skin-stretching device. Plast Reconstr Surg 109, 674–680.

Mense, S., Simons, D.G., Russell, J. (Eds.), 2001. Muscle Pain: Understanding its Nature, Diagnosis and Treatment. Lippincott Williams & Wilkins, Philadelphia.

Panjabi, M.M., 1992. The stabilising system of the spine. Part II Neutral zone and stability hypothesis. J Spinal Dis 5, 390–397.

Prasad, S.A., Cerny, F.J., 2002. Factors that influence adherence to exercise

and their effectiveness: application to cystic fibrosis. Pediatr Pulmonol 34 (1), 66–72.

Proske, U., 2005. What is the role of muscle receptors in proprioception? Muscle Nerve 31 (6), 780–787.

Racette, R., Peronnet, F., Massicotte, D., et al., 2005. Metabolic response to prolonged cycling with (13) C-glucose ingestion following downhill running. Eur J Appl Physiol 93 (5–6), 598–605.

Rahnama, N., Rahmani-Nia, F., Ebrahim, K., 2005. The isolated and combined effects of selected physical activity and ibuprofen on delayed-onset muscle soreness. Randomized Controlled Trial. J Sports Sci 23 (8), 843–850.

Rees, K., Taylor, R.S., Singh, S., et al., 2004. Exercise based rehabilitation for heart failure. Cochrane Database Syst Rev 2004 (3) CD003331.

Richardson, C., Hodges, P.W., Hides, J., 2004. Therapeutic Exercise for Lumbopelvic Stabilization. A Motor Control Approach for the Treatment and Prevention of Low Back Pain, second ed. Churchill Livingstone, London.

Richie, D.H., 2001. Functional instability of the ankle and the role of neuromuscular control: a comprehensive review. J Foot Ankle Surg 40, 240–251.

Rodenberg, J.B., Bär, P.R., De Boer, R.W., 1993. Relations between muscle soreness and biochemical and functional outcomes of eccentric exercise. J Appl Physiol 74, 2976–2983.

Rogers, M.A., Evans, W.J., 1993. Changes in skeletal muscle with aging: effects of exercise training. Exer Sports Sci Rev 21, 65–102.

Sahrman, S., 2002. Diagnosis and Treatment of Movement Impairment Syndromes. Mosby Inc., St Louis, MO.

Saxton, J.M., Clarkson, P.M., James, R., et al., 1995. Neuromuscular dysfunction following eccentric exercise. Med Sci Sports Exer 27, 1185–1193.

Scott, W., Stevens, J., Binder-Macleod, S.A., 2001. Human skeletal muscle fibre type classifications. Phys Ther 81 (11), 1810–1816.

Shrier, I., 2004. Does stretching improve performance? A systematic and critical review of the literature. Clin J Sport Med 14 (5), 267–273.

Shrier, I., Gossal, K., 2000. Myths and truths of stretching. Physician Sports Med 28 (8), 57–63.

Sinnett, A.M., Berg, K., Latin, R.W., et al., 2001. The relationship between field tests of anaerobic power and 10-km run performance. J Strength Condition Res 15 (4), 405–412.

Spernoga, S.G., Uhl, T.L., Arnold, B.L., et al., 2001. Duration of maintained hamstring flexibility after a one-time, modified hold–relax stretching protocol. J Athlet Train 36 (1), 44–48.

Stanish, W.D., Curwin, S., Rubinovich, M., 1985. Tendinitis: the analysis and treatment for running. Clin Sports Med 4 (44), 593–609.

Swanik, K.A., Lephart, S.M., Swanik, C.B., et al., 2002. The effects of shoulder plyometric training on proprioception and selected muscle performance characteristics. J Shoulder Elbow Surg 11 (6), 579–586.

Tippet, S.R., Voight, M.L., 1995. Functional Testing in Functional Progressions for Sport Rehabilitation. Human Kinetics, Champaign, IL.

Voight, M.L., Hardin, J.A., Blackburn, T.A., et al., 1996. The effects of muscle fatigue on and the relationship of arm dominance to shoulder proprioception. J Orthop Sports Phys Ther 23, 348–352.

Waddington, G.S., Shepherd, R.B., 1996. Ankle injury in sports: role of motor control systems and implications for prevention and rehabilitation. Phys Ther Rev 1, 79–87.

Wilk, K.E., Escamilla, R.F., Fleisig, G.S., et al., 1996. A comparison of tibiofemoral joint forces and electromyographic activity during open and closed kinetic chain exercises. Am J Sport Med 24 (4), 518–527.

Wilkerson, G.B., Nitz, A.J., 1994. Dynamic ankle instability: mechanical and neuromuscular interrelationships. J Sport Rehabil 3, 43–57.

Chapter 14

Muscle imbalance

Alan Chamberlain, Wendy Munro and Alec Rickard

INTRODUCTION

The concept of muscle imbalance (and balance) is not new. This is evidenced through over 60 years of texts involving muscle testing and function (Kendall et al. 2005) and has evolved over the most recent of those years through work from the likes of Janda (1983), Sahrmann (1987), Richardson (1992), Jull et al. (1999) and others. Clearly, alterations in muscle balance cannot be considered in isolation, but must be considered as part of movement control, where the differentiation of the factors leading to movement dysfunction is essential and part of the holistic assessment and management of individuals with musculoskeletal pain.

This chapter concentrates on the adaptation of muscle in relation to posture and movement patterns, and the relationship to musculoskeletal pain syndromes. The following knowledge and understanding of the reader will be assumed:

- the micro- and macro-anatomical structure and physiology of muscle and associated tissue;
- micro- and macrostructure related to function;
- macro-anatomy or gross anatomy of muscles and muscle groups, along with their relationships to each other and other tissue structures, such as bones, joints, connective tissue, viscera, neural and vascular tissue; in other words, what lies deep, superficial, superior, inferior, medial and lateral to the structure being considered.

In order to contain this subject within the limits of a manageable chapter, only the more commonly affected – and/or those deemed clinically important – muscle groups will be discussed, utilising select key clinical examples. However, the principles described in this chapter should be transferrable to any region in the body. In addition, when considering cause, effect and recovery, the Specific Adaptation to Imposed Demand (SAID) principle should be applied, as originally proposed by Selye (1951). Example case scenarios will be illustrated using the subjective, objective, analysis, plan and evaluation (SOAPE) process, familiar to physiotherapists from a plethora of texts and institutions (Baxter 2003).

Clinical note

The assessment of muscle *imbalance* and management of musculoskeletal disorders with muscle *balance* techniques is just *one* approach amongst the plethora of physiotherapeutic assessment and management skills. It can be described as a method to rehabilitate patients presenting with movement dysfunction and its associated problems. Analysis of the subjective and objective assessment of the patient is key to identifying this as an appropriate modality for rehabilitation through hypothesis testing, clinical and critical reasoning, and evaluation of practice.

What is muscle imbalance?

As physiotherapists, we need to be able to differentiate between what is considered a 'normal' (or 'expected') range of motion (ROM) and that which is deemed 'abnormal' – either hypo- or hypermobility – along with which structures may produce or restrict the range of motion. It must be noted that there may also be an underlying predisposition – or *pathology* – for hypo-/hypermobility, as in the case of connective tissue conditions, such as ankylosing spondylitis and hypermobility syndrome (see Figure 14.1) (as distinct from a joint demonstrating hypermobility), or neurological conditions, such as multiple sclerosis. However, it is outwith the scope of this chapter to discuss these specific pathological causes.

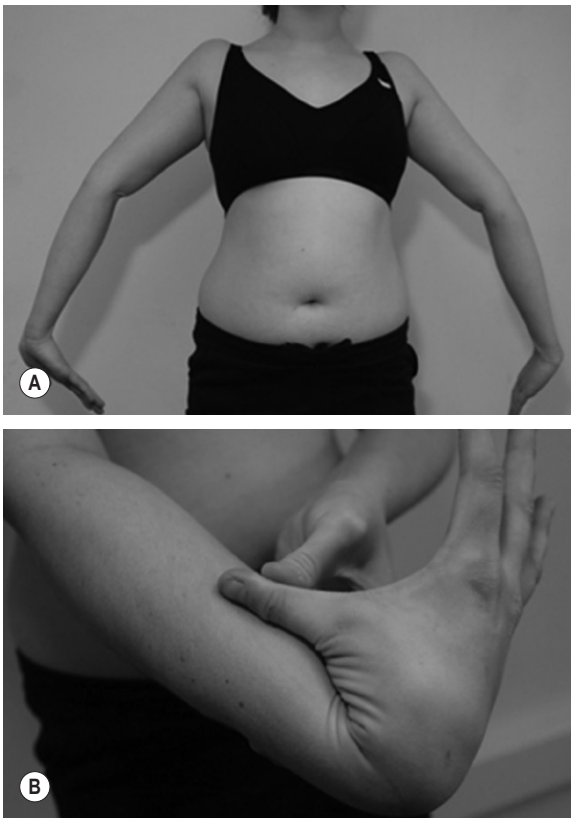


Figure 14.1 (a, b) Classical hypermobility syndrome.

ABC Definition

Richardson (1992) described muscle imbalance as a specific problem of movement dysfunction, concerned with inadequate control and co-ordination of muscles for the protection of joints and surrounding structures.

Muscle imbalance can be either 'passive' or 'active'; passive being identified by muscle length and strength being either less or more than the ideal, and active being identified when one of a synergistic pair of muscles predominates during the movement (Sahrmann 1987). The resultant functional and structural changes in the muscle appear to be reversible, suggesting that exercise to facilitate muscle length changes will be useful in the management of movement dysfunction (Gossman et al. 1982).

Perhaps it is worth reviewing the general characteristics, function and roles of muscles first. Muscles can act as prime movers, or *agonists*, if they are primarily responsible for producing movement. Their direct opposites are *antagonists*, which do not normally oppose movement, but have the potential to do so if they are not inhibited; for example, when flexing the elbow, biceps brachii is the agonist whilst the triceps is the antagonist (relaxed, via reciprocal inhibition). Co-contraction of muscles is possible, which can assist movement and/or stabilise. When this occurs to assist the movement produced by the agonist, those muscles are said to be working as *synergists* (see the example of the scapular force couple in the shoulder section). When muscles contract to stabilise a joint and control the position of the origin of an agonist, they are known as *fixators* (or *stabilisers*, i.e. in the case of elbow flexion as above, the fixators would be those that stabilise, or fixate, the shoulder, such as the rotator cuff).

These descriptions are all well and good, but, unfortunately, prove rather inadequate when it comes to explaining more dynamic, complex movements. Therefore, further classification of the role of muscles has been attempted by various authors according to their anatomy, architecture, fibre type and function, as summarised in Table 14.1. To simplify, some muscles exhibit a tendency to be more stabilising (i.e. multifidius in the spine) or mobilising in their function (i.e. erector spinae), although there are also some that demonstrate characteristics of both and can 'multitask' their role depending on the demands placed upon them at the time. Examples of the latter include the vasti muscles of the quadriceps and soleus. Throughout this chapter, muscles will be discussed in relation to their functional role as local and global stabilisers, or global mobilisers.

Problems may occur when, for example, a predominantly stabilising muscle is injured or dysfunctional, leading to instability and altered joint biomechanics, increased strain on structures, such as joint surfaces, and pain. Muscles with a predominantly mobilising role can be used as temporary stabilisers, but as their anatomy and physiology does not lend themselves to this, this may lead to overuse, fatigue and pain. It has been suggested that myofascial trigger points (taut, irritable bands within muscles resulting from overuse) may develop as a result (Simons et al. 1999; Fernández-de-las-Peñas et al. 2006) and which can perpetuate the muscle imbalance through further pain and resultant inhibition.

Therefore, in short, if muscle imbalance is present and the muscles are unable to fulfil their role correctly, this can be through either being unable to stabilise or induce/permit movement because one or more are either too *weakened* and/or *lengthened*, or too *over-active* and/or *shortened*.

Stability

Additionally, the term 'stability', particularly '*core stability*', has also become synonymous with approaches to dealing with muscle imbalance of the spine, especially in the lumbar spine. Confusion can arise with this term, particularly when used very specifically by one group of

Table 14.1 Summary of the different classifications of muscle roles

Janda (1994), Jull and Janda (1987)	Bergmark (1989)	Richardson (1992)	Comerford and Mottram (2001)
Phasic	**Local muscles**	**Stability synergists**	**Local stabilisers**
Tend to lengthen Weak Uni-articular	Deepest muscle layer that originates and inserts segmentally Control and maintain neutral Respond to changes in posture and to changes in low extrinsic load Independent of the direction of load or movement and appear to be biased for low load activity	Single joint muscle Aligned to oppose gravity Approximates the joint Deep Extensive aponeuroses Slow twitch (type I) muscle fibres Control low force levels for long periods	Increased muscle stiffness to control segmental motion Controls the neutral joint position Contraction = no or minimal length change so does not produce movement Activity is independent of direction of movement Continuous activity throughout movement Proprioceptive input through joint position, range and rate of movement
Postural	**Global muscles**	**Mobility synergists**	**Global stabiliser**
Tend to tighten Bi-articular One-third stronger Trigger points Lower irritability threshold	Superficial or outer layer of muscles lacking segmental vertebral insertions Insert or originate on the thorax or pelvis Respond to changes in the line of action and the magnitude of high extrinsic load Large torque producing muscles biased for range of movement	Fast twitch muscle fibres (type II) Suited to production of high speed movement Span two joints Subject to high force values	Generates force to control range of motion Contraction = eccentric length change – control throughout range especially inner range and outer range Low load deceleration of momentum (especially axial plane:rotation) Activity is direction dependent
			Global mobilisers
			Generates torque to produce range of motion Contraction = concentric length change – concentric production of movement Concentric acceleration of movement (especially sagittal plane:flexion/extension) Shock absorption of load Activity is direction dependent Non-continuous activity (on:off phasic pattern)

Adapted from Comerford and Mottram (2001) and Norris (1995).

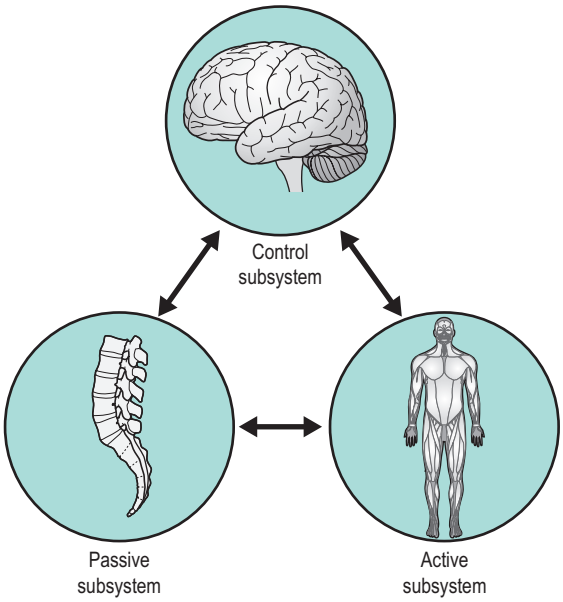


Figure 14.2 A model of stability. *Adapted from Panjabi (2003).*

professionals to be more than merely a descriptive state; when is someone or something stable or unstable, what makes someone or something stable/unstable? For example, an orthopaedic surgeon may well be focussing on the bony congruency or ligamentous support at a spinal segment and any trauma or pathology associated with this (as in spondylolithesis) as the cause of instability. Conversely, a physiotherapist, unless assessment suggests otherwise, may be concerned with the quality of the spinal movement, any associated/compensatory movements and the support of the muscles to stabilise the movement. Therefore, the stability of the spine, in this example, is dependent on a number of factors. Figure 14.2 demonstrates a basic model of stability that considers the interplay of the principal systems involved (Panjabi 1992a, 1992b, 2003).

Panjabi (1992a, 1992b, 2003) additionally discusses control of the neutral zone: the area (or zone) around the segment's neutral position where there is little resistance offered by the passive restraints in response to a small range of displacement (see Figure 14.3). Although originally related to the spine, this model can be applied equally to the appendicular, as well as the axial skeleton, when considering the interplay of the subsystems. This control of the neutral zone can be disturbed locally by the dysfunction of recruitment and motor control of the deep segmental stabilisers, and globally by an imbalance between the mono-articular stabilising muscles and the bi-articular mobilising muscles (Comerford and Mottram 2001). It is hypothesised that an increase in the size of the

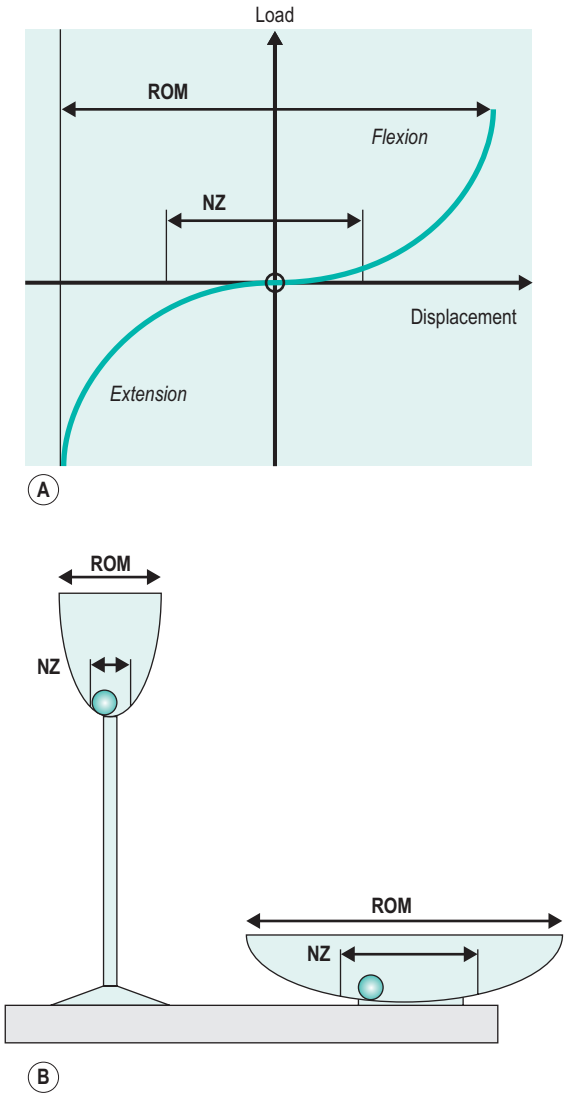


Figure 14.3 Load–displacement curve. (a) The effect of flexion and extension loads on a spinal segment, exhibiting a nonlinear load displacement curve. The neutral zone (NZ) parameters represent laxity of the spinal segment around the neutral position. (b) A graphic analogue of the load-displacement curve where the extension curve from (a) is just inverted. A deep champagne glass and a shallow bowl represent a more and a less stable spine respectively, as the champagne glass has a smaller NZ, greater load (in this case, titling the glass) is required to cause the ball to displace/move. *Reproduced from Panjabi (2003), with permission.*

neutral zone (therefore decreased 'stability' or increased 'instability', if you like) correlates with pain and dysfunction (and possibly could be caused by either); correspondingly, decreasing the neutral zone may decrease pain and dysfunction (Panjabi 2003). However, it must be noted that the clinical reality is rarely this straightforward and is usually multifactorial (see Chapter 17)!

NEUROPHYSIOLOGICAL COMPONENTS OF MUSCLE BALANCE

To understand the concept of muscle imbalance and the implications of the alteration in muscle function, the neurophysiological components of muscle balance will be discussed first.

Motor control

Stabilisation requires automatic recruitment of the muscles surrounding the joint. This requires appropriate co-activation of muscles, the application of appropriate levels of force for the task in hand and appropriate timing of the muscles (Richardson 1992). Normally, the mono-articular stabilisers should activate earlier than the multi-articular mobiliser synergists – a *feed-forward* mechanism. This co-ordinated activity involves sensory strategies through feedback from the joint and ligament afferents, and muscle spindle activity, along with biomechanical, motor processing strategies and learned behaviour that anticipates change (Comerford and Mottram 2001).

Motor units (the motor neurone and the muscle fibres it innervates) contain the same type of fibre so that there are, pragmatically, two main types: slow (tonic, type I fibres) motor units and fast (phasic, type II fibres) motor units. Therefore, slow motor units have a slow speed of contraction, low contraction force and are fatigue resistant compared with fast motor units. Slow motor units are recruited primarily at low loads of less than 25% of maximum voluntary contraction (MVC) and fast motor units are recruited at higher loads (Gibbons and Comerford 2001). Therefore, the recruitment of slow motor units, and use of muscles with high proportions of these is necessary for optimal stabilisation. When mobiliser muscles, which contain high proportions of fast motor units are utilised as compensation for stabiliser dysfunction, degradation of the fine control results along with fatigue and potentially pain and spasm if then over-used.

However, biased recruitment of shortened muscles can be a problem. It is suggested that short muscles are recruited more easily than their lengthened synergists (Sahrmann 1987). This may be owing, mechanically, to the increased overlap of actin and myosin (as in the sliding filament theory, below) leading to an increase in the intrinsic 'stiffness' of the muscle and readiness for activation, and owing, neurologically, to the increased excitability of the alpha motor neurone pool facilitated by the increased tension and activity of the muscle spindle afferents (Comerford and Mottram 2001).

There is evidence to suggest that the reduction of proprioceptive input during sustained low-load contractions from the muscle spindle changes the recruitment order of the motor neurones and the normal dominance of the local stabilisers (Grimby and Hannerz 1976, cited in Comerford and Mottram 2001). Janda (as discussed by Comerford and Mottram (2001)), identified and quantified recruitment and sequencing differences between synergistic muscle groups in functional movement. From this, he was able to identify a consistent recruitment pattern in asymptomatic individuals and also characteristic abnormal patterns in symptomatic individuals. Similar evidence has been identified by several authors in relation to individuals with impingement symptoms of the shoulder (Ludewig and Cook 2000; Cools et al. 2003), cervicogenic headaches (Jull et al. 1999) or patellofemoral pain (McConnell 1996).

Muscle length adaptations

Several theories exist relating to the relationship between muscle length and strength (Sahrmann 1987), and are listed below.

Stretch weakness

It is suggested that stretch weakness occurs when a muscle is maintained in an elongated position beyond its neutral physiological rest position but not beyond the normal range of muscle length (Gossman et al. 1982). Examples of this may be weakness of the dorsiflexors in a bedridden patient when held in a lengthened position owing to the weight of the bed clothes holding the foot in a plantar flexed position. Equally, prolonged postural strain, such as drooping shoulders with elongation of the middle and lower trapezii, can lead to stretch weakness.

Positional weakness and length associated changes

When a muscle is immobilised in a lengthened or shortened position, the muscle adapts in order to either increase or decrease the number of sarcomeres respectively. This change in number allows for a change in sarcomere length to one that gives maximum overlap of actin and myosin for optimum cross bridge formation and the ability of the muscle to achieve maximum tension in the immobilised position (Williams and Goldspink 1978). These adaptations appear to differ with age, occurring primarily in the tendon in young people rather than muscle (Gossman et al. 1982), such that immobilisation in a lengthened

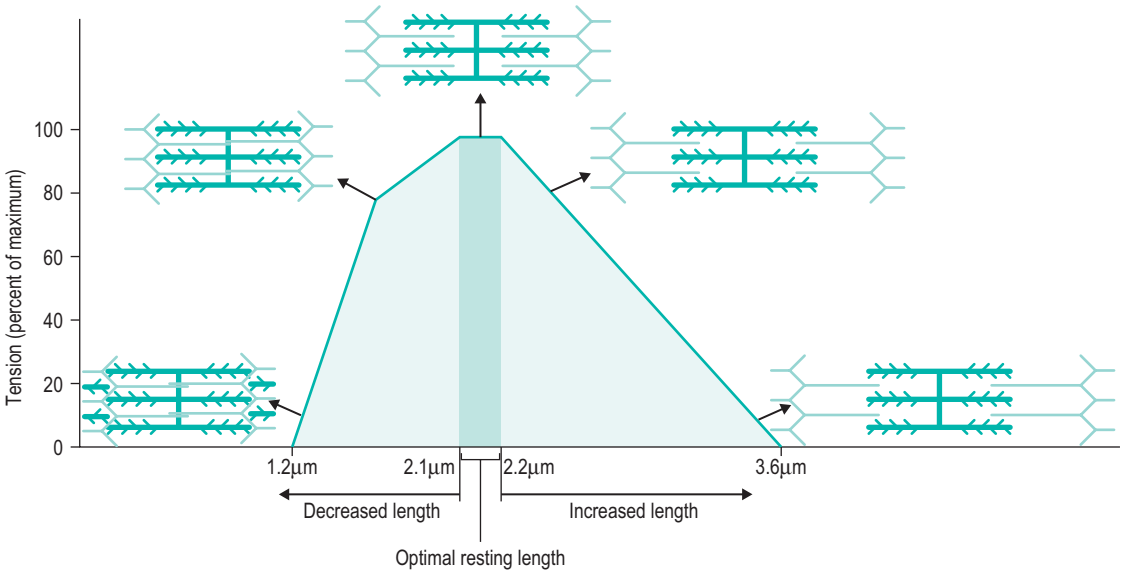


Figure 14.4 The sliding filament theory and length tension curve.

position leads to elongated tendon and shorter muscle belly with a reduction in the number of sarcomeres in the muscle belly.

Clinical note

Clinical implications of these adaptations are that muscles will test *stronger* in their 'new' position, but will test *weaker* in their normal physiological resting position. It has been shown that once immobilisation of a muscle in an altered position is discontinued, the muscles should regain their 'normal' sarcomere number and length (Tabary et al. 1972).

Williams and Goldspink (1984) also demonstrated changes in the connective tissue of the muscle when a muscle was immobilised in a shortened position. It is generally thought that muscle has a parallel elastic component (primarily from the perimyseum, but also the epimyseum) to the active contractile element. This parallel elastic component is thought to distribute forces associated with the passive stretch of the muscle and maintain the relative position of the fibres. In a muscle immobilised in a shortened position the increase in connective tissue is likely to result in an increase in muscle stiffness and reduced elasticity of the muscle.

Similar changes may occur when a muscle works at a shorter length (Williams and Goldspink 1984) and it should be noted that normal, transient changes in muscle length, as part of movement and positioning, will affect the tension that can be developed, as demonstrated by the sliding filament theory and the muscle length tension curve (Figure 14.4).

In addition, where a muscle is bi-/multi-articulate, then active and passive insufficiency can occur. Active insufficiency is the inability of a bi-/multi-articulate muscle to generate enough tension in a shortened position, where the position of one joint affects the tension of the muscle at the other. A classic example is the hamstrings: if you stand with your knees extended and your hips flexed (i.e. in a bow), the hamstrings are able to extend your hips/pelvis to raise your body upright, but if you do the same starting with your knees flexed, your hamstrings are disadvantaged and you rely on your gluteus maximus. The same occurs with the finger flexors if you try to flex the fingers to make a fist with your wrist fully flexed (although here you also encounter *passive* insufficiency), whereby the wrist extensors are fully lengthened across two or more joints (the wrist, metacarpalpharangeal and interpharangeal joints).

Relative flexibility and relative stiffness

'Relative stiffness' as described by White and Sahrmann (1994) in one segment will require flexibility in an adjacent area to gain functional movement. This relative stiffness can occur as discussed above in a shortened muscle, but also when the passive structures around the joint (capsule and ligament) or the joint surfaces themselves become restricted in their movement. An example of this

might be an adhesive capsulitis of the glenohumeral joint; restriction of the capsule around the joint causes limitation of abduction resulting in hypermobility of the scapulo-thoracic joint to gain movement and hence lengthening of the stabilising muscles of the scapula on the chest wall. Similarly, 'relative flexibility' (White and Sahrmann 1994) can occur where mono-articular muscles are unable to adequately shorten, so are lengthened, strained or weak and allow excessive motion at the joint they act upon. This increase in mobility has obvious implications on initiating pathology. This relative stiffness and flexibility gives rise to the concept of 'give and restriction', as discussed by Comerford and Mottram (2001).

EXAMINATION AND ASSESSMENT PRINCIPLES

The examination and assessment process is required to determine whether muscle imbalance is a causal factor of the patient's symptoms. Recognition of postures that deviate from the expected normal (see Kendall et al. 2005) and of physiological movement dysfunction patterns (as detailed in Table 14.2) is essential to this.

It is also necessary to identify whether the abnormal postures and movement patterns are the primary or secondary source of the patient's symptoms (see 'Clinical note' in the cervical spine section below). Secondary stresses can give rise to symptomatology in any part of the kinetic chain affected by the alteration of posture and physiological movement further along the chain. The signs and symptoms of other anatomical regional areas will need to be addressed, but causal primary problems must always be recognised and addressed, as well as those that have developed secondarily.

Treatment, intervention and management principles

As well as the theory described in this chapter, it must be noted that there are other factors to consider in order to determine the most effective and efficient intervention, such as psychosocial influences, age and comorbidities. Consideration also needs to be given to the patient's expectations. Are the function and demands they expect to place upon themselves compatible with attaining/maintaining 'muscle balance'?

The physiological parameters already discussed should inform your treatment decisions. Table 14.3 provides an overview of management principles.

In practice, there is an additional consideration and experiential, anecdotal phenomenon, which is important to consider for the life-busy (perceived or actual) patient who is at risk of non-compliance of rehabilitation if the

Table 14.2 Key assessment principles

Subjective examination
Check past medical history for pathological/contributory causes of observed posture Exclude red flags Consider subjective history and how occupation/functional activities relate to the observable posture Prioritise objective assessment taking into account SIN factor (severity/irritability/nature)
Objective examination
Postural assessment
Assess global posture – gauge what is their 'normal' and what has contributed to, or resulted from, their presenting problem Check for neutral joint position in the joint(s) relevant to the individual's problem Develop a hypothesis of weakened and/or lengthened and overactive and/or shortened muscles
Observe physiological movement
Observe the quality and measure active/passive physiological range of movement • Differences owing to influence of soft tissues? Assess end-feel • Firm/springy = owing to soft tissue tightness? • Empty = owing to hypermobility? Note any symmetry/asymmetry and any deviations away from the normal path of movement
Test muscle strength and length
Test weakened/lengthened muscles to see if they can generate and maintain tension in their shortened position Test length of shortened muscles
Identify compensations
Substitution or dominance of the global stabiliser or mobiliser muscles where there is dysfunction of the local stabilisers → e.g. where the neutral position of the scapula cannot be maintained on the chest wall there may be substitution or dominance by the rhomboids, levator scapula, pectoralis minor or latissimus dorsi → or where there is a weakness of transversus abdominis producing an anterior tilted pelvis and a secondary increased lumbar lordosis or vice versa Where there are shortened muscles leading to hypomobility, observe where the movement occurs to achieve function. (e.g. individual with a shortened hamstring when sitting and asked to extend the knee may compensate by flexing the lumbar spine) *NB.* Check you have fully assessed the available length and strength. Could apparent restrictions or weakness be a result of the testing position itself?

Table 14.3 Overview of management principles

1. Isolated retraining
   Recruitment of appropriate stabilisers
   Low/no load (may need facilitation, auto-assistance, etc.)
   Low repetition
   Concentrate on correct movement with no compensation
2. Strengthening
   Low-load, low effort for stabilisers
   → 25% MVC
   Increasing repetitions and/or load gradually whilst maintaining correct movement
   → endurance versus power
   → avoiding compensatory strategies, especially recruitment of mobilisers
3. Lengthening
   Lengthen tight structures once 'stabilised'
   Consider dynamic versus static
4. Education and advice (but may also be commenced early on)
   Avoid/minimise postures which lengthen weakened structures
   Avoid/minimise activities that facilitate dominant/overactive muscles
   Advise on alternative postures/activities
   → Consider appropriate equipment/aids, i.e. seating, lumbar roll, etc.

programme is too time-consuming. Well-intended programmes have been known to be overloaded and discontinued if it becomes too much and so it may be prudent to withhold some lower priority rehabilitation for a later date and introduce as proven necessary.

The Lumbo-pelvic region

The lumbo-pelvic region has seen a significant amount of research and debate as to the role of the muscles in relation to lower back pain, which should not be surprising considering how common lower back pain is. However, it remains a complicated area and so it is beyond the scope of this chapter to consider all muscles and potential permutations of imbalance. As transversus abdominis (TrA) has received considerable interest both in the literature and clinical environment, this will be the focus as a template approach for the physiotherapist to consider in practice. Table 14.4 lists the main muscles acting on the lumbo-pelvis with their respective primary functional role.

The concept of 'core stability' and muscular stabilisation of the lumbar spine has derived from Panjabi's (1992a, 1992b, 2003) model of the three subsystems described previously. With reference to the passive subsystem, the lumbar spine appears to be at a disadvantage, 'sandwiched' between the inherently more stable thoracic spine (via the rib cage) and pelvis (see Figure 14.5). Therefore, whilst the lumbar vertebrae and intervertebral discs are the largest and strongest in the spine, further support and control is required, but, as you will see in Figure 14.5, TrA, along with the multifidus, diaphragm and pelvic floor, appear to form a 'box' (or more aptly, a corset). The diaphragm and pelvic floor appear to contribute mainly through increasing intra-abdominal pressure and restricting the movement of the viscera (Richardson et al. 2004).

Table 14.4 The main lumbo-pelvic mobiliser and stabiliser muscles

Muscle	Primary muscle role
Transversus abdominis	Local stabiliser
Multifidus	Local stabiliser (deep intersegmental fibres) Global stabiliser (superficial fibres)
Rectus abdominis	Mobiliser
External oblique	Global stabiliser Mobiliser
Internal oblique	Local stabiliser Mobiliser
Erector spinae	Mobiliser

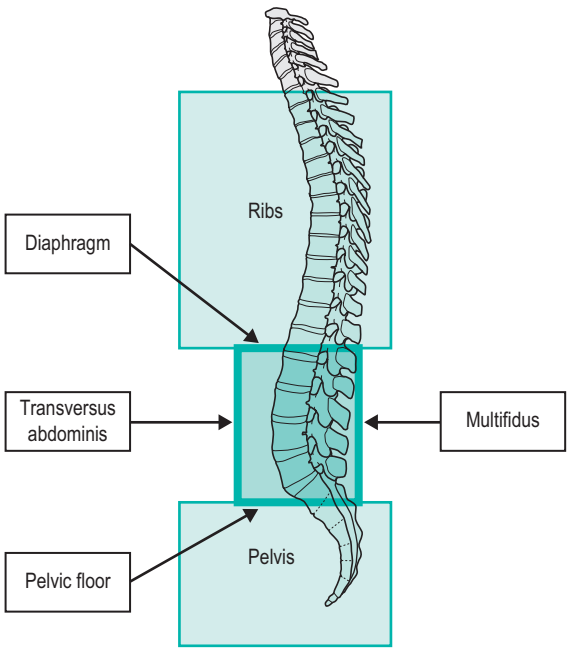


Figure 14.5 The 'core' stabilisers.

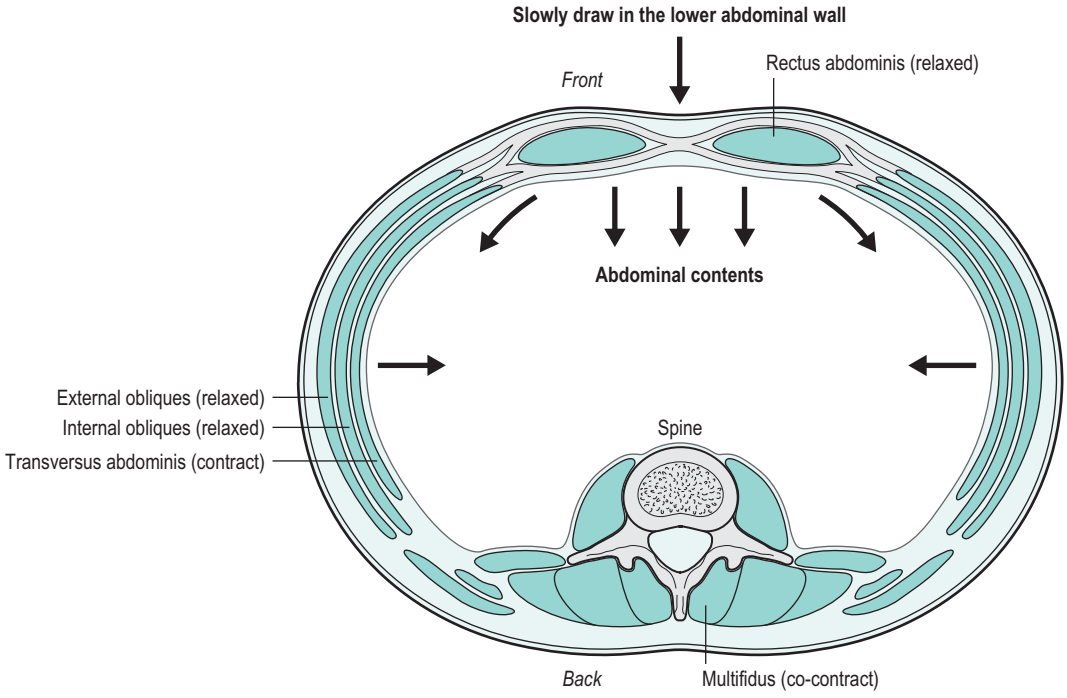


Figure 14.6 Transversus abdominis and multifidus.

Transversus abdominis (TrA)

TrA is the deepest of the abdominal muscles (Figure 14.6). The direction of the muscle fibres runs horizontally medially from the lateral attachments of the internal surface of the six lowest ribs, the thoracolumbar fascia, internal surface of the iliac crest and the lateral third of the inguinal ligament to the sheath of rectus abdominis (Ombregt et al. 2003; Standring 2005). It can be seen from the attachments how contraction can mechanically increase intra-abdominal pressure and thus stabilise the lumbar spine, although it does not act alone, as co-contraction with multifidus is also thought to occur (Richardson et al. 2002, 2004). When considering this action, it is easy to understand how dysfunction may lead to pathological signs and symptoms, and subjects with low back pain have been shown to demonstrate delay and inhibition of contraction compared with asymptomatic subjects (Richardson et al. 2002, 2004).

It is widely accepted that TrA provides support for the abdominal contents and it has been demonstrated through research that it provides indirect stability for the lumbosacral spine and sacroiliac joints (Richardson et al. 2002, 2004). The associated levers of the pelvis and spine arising from these joints must be remembered together, with further consideration when relating TrA structure to its function and the stabilising of the pelvic postero-anterior lever in neutral; in other words, maintaining an antero-posteriorly neutral pelvic level.

Most of the literature explains how the pelvis is stabilised in the neutral position by the indirect stabilising role on the lumbo-sacral and sacroiliac joints, but it can be argued that the pelvis is stabilised in neutral on the spine and prevented from anteriorly tilting directly anatomically through the attachment to the pubis and indirectly via rectus abdominis.

Mechanically, transversus abdominis may have a direct role in maintaining pelvic neutral, producing a cephalic force on the anterior pelvis owing to the lower fibres of the muscle curving inferiorly and medially to attach to the pubic crest and pectineal line, along with the aponeurosis of internal oblique (Standring 2005). Further to this, if, on contraction, it places tension on the rectus abdominis, which in turn attaches to the costal cartilages and ribs superiorly and pubis inferiorly, it can be mechanically argued that transversus abdominis has an indirect role in stabilising and maintaining the pelvis in neutral relative to the spine. This, therefore, helps prevent anterior tilt of the pelvis. An anterior tilted pelvis can therefore present as a sign of transversus abdominis weakness and, from clinical experience, has been supported or accompanied by poor palpable contraction of the muscle. The same anterior tilted pelvis may be the causal link for increasing the lumbar lordosis and therefore be a source for causing mechanical back pain, for example through increased facet joint irritation from the resultant increased compression force production.

It is not only weakness that can lead to an anterior tilted pelvis. The lower fibres of TrA and all the fibres of rectus abdominis may adaptively lengthen over time, such as during pregnancy in women, for example. Also, through prolonged, repeated and sustained static posture in sitting there can be an over-compensatory posture with an anterior tilted pelvis and hyper-lordosis of the lumbar spine in an attempt to maintain a normal lumbar lordosis. This would be considered to be the adverse effects of the SAID principle (Selye 1951). In other words, unwanted adaptation to adverse imposed demand, such as lengthening of the abdominal muscle fibres and the anterior longitudinal ligament of the spine.

In the case of the former example, intervention would require, following confirmation through examination and analysis, recovery of the optimal length, contractibility and strength of the TrA muscle fibres; however, in the case of the latter, it would also require prevention in recurrence through changing postural habits. It has to be borne in mind that the above presentation may be asymptomatic initially and what transpires can be any other number of signs and symptoms of other secondary or tertiary problems, such as 'knock-on' cervico-thoracic or shoulder problems, but it is beyond the scope of this chapter to list or discuss all the potential problems that can arise from an anterior tilted pelvis through muscle imbalance.

Before considering further theory and research, the physiotherapist must also understand that it has also been argued in the literature (Richardson et al. 2002, 2004) that TrA can become non-functional as an inhibitory response to problems, such as mechanical low back pain. It is considered here that this may not be the order of events in all mechanical low back pain and it is difficult to research the order of events, as it would be necessary to have otherwise healthy subjects induce mechanical low back pain in order to determine if this is the point TrA *switches off*. Nevertheless, the physiotherapist can only treat what presents and in the presence of a non-functioning TrA the challenge is to rehabilitate the muscle.

As described above, the SOAPE process will be used for further illustration.

Subjective examination

The subjective reporting of pain may be evident and may be region specific over the lumbar spine with possible radiation into the glutei and lower limb, unilaterally or bilaterally.

Symptomology may occur in secondary areas both proximally and distally affecting the thoracic and cervical spine and upper and lower quadrants owing to the altered alignment of the lumbar spine posture and its knock-on effect on posture elsewhere. Signs and symptoms will need to be addressed but, equally, the other causations of an anteriorly tilted pelvis or associated and secondary lumbo-pelvic-hip complex problems would need investigating and correcting as necessary (see some examples in the non-exhaustive list in Table 14.5).

Other subjective history may include continence problems if the pelvic floor muscles are weak in addition to

Table 14.5 Other contributors to the development of an anterior tilted pelvis and additional associated/secondary problems

Muscle	Problem	Additional associated/secondary problems
Iliopsoas	Too short/passive insufficiency causing anterior tilted pelvis	Increased lumbar lordosis and lumbar spinal dysfunctional restriction of lumbar flexion range of movement (ROM) Hip lateral rotational dysfunctional restriction of ROM, and shortening of internal rotators and adductors of the hip Hip extension dysfunctional restriction of ROM Shortening of other hip flexor, rectus femoris Shortening of other muscles of quadriceps femoris Medial knee joint stresses
Quadratus lumborum	Too short/passive insufficiency causing an increased lumbar lordosis and an anterior tilted pelvis	All of the above Plus, unilateral imbalance may cause rotation/torsion or lateral pelvic tilt
Multifidus	Weakness/lengthening or over-activity/shortening	Although acknowledged as segmental stabilisers, collectively they can contribute to the maintenance of abnormal posture if dysfunctional, such as hyperlordosis and therefore predispose to anterior tilt

NB: It should also be noted that muscles involving the lower limb, which also act on the pelvis need consideration, such as rectus femoris and the hamstrings (see Kendall et al. (2005) and others regarding postures such as the pelvic-crossed syndrome).

TrA owing to their proximity and potential involvement in lumbo-pelvic stability (see Figure 14.5). Specific correcting postures may be recognised as relieving factors of subjective pain, such as lumbar flexion. If neural structures are irritated owing to narrowing of the intravertebral foramina, paraesthesia, anaesthesia and pain radiating in the dermatomal distribution may be complained of.

Objective examination

On observation

In standing: Lateral view

Anterior tilted pelvis may be seen. Other observations may also include some or all of the following superiorly: increased lumbar lordosis; increased thoracic kyphosis; protracted cervical spine; protracted shoulder girdle. An increased lumbar lordosis and/or an anterior tilted pelvis may be less visible to the observer if the former is masked by protective lumbar paravertebral muscle spasm and the latter by adipose tissue. The latter may equally be coupled with a clear observation of a low toned abdominal wall.

Measurements

It is possible to compare the levels of the anterior superior iliac spine (ASIS) and posterior superior iliac spine (PSIS) by palpating them simultaneously, but achieving an angle measurement with a goniometer using these two bony points is either difficult (reducing validity and reliability) or not possible for all somatotypes. There can be a normal slight anterior tilted pelvis, but it is whether it is excessive or not and whether it is a result of weakness of TrA that is important.

High criterion-validity using a universal goniometer has been demonstrated when measuring knee joint movement, but there is sufficient evidence to ensure content validity using this method to measure range of movement (Clarkson 2000). Joint range of movement (ROM) can be reliably measured using a universal goniometer when the same physiotherapist uses the same measurement tool and a standard test position and protocol (Clarkson and Gilewich 1989, cited in Walsh et al. 1998; Clarkson 2000). If there is intra-tester reliability and validity for joint ROM measurement, the same can only be assumed in the measurement of other ROM. Accepting universal goniometer validity and reliability in practice, one of us developed a measurement technique that involves identifying the tangent of the normal iliac crest (line AB, Figure 14.7) and the ASIS (point *d*). With the fulcrum of the goniometer being placed perpendicular to the tangent (point *c*), the stationary arm of the universal goniometer is aligned with line AB, maintaining it in the horizontal plane, and the adjustable arm is aligned with point *d*. It sometimes requires the operator to move the goniometer back and forth posteriorly and anteriorly until the operator is satisfied the fulcrum is aligned with point *c* and the arms of the goniometer are aligned with their respective points.

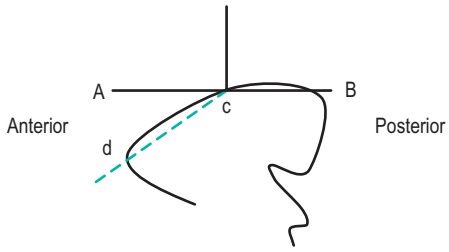


Figure 14.7 Lateral view of normal pelvis.

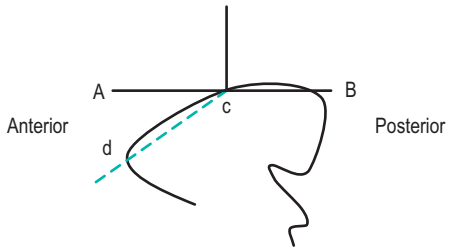


Figure 14.8 Lateral view of a pre-test anterior tilted pelvis.

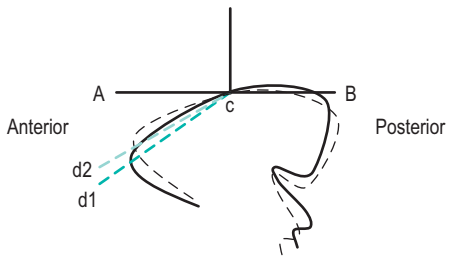


Figure 14.9 Lateral view of an anterior tilted pelvis during the test.

It is accepted that the universal goniometer fulcrum is usually aligned with the fulcrum of movement in peripheral joints and that the fulcrum of movement of the pelvis does not occur at point *c* and, in reality, occurs at the lumbo-sacral spine. However, provided the test position and protocol is followed as described, it is reasonable to use in clinical practice but it remains subject to validity and reliability studies before it can be used in research. If the pelvis is anteriorly tilted, the tangent of the iliac crest would be equally tilted and it would therefore still require the operant to align the stationary arm with a horizontal line at the true apex of the iliac crest, which will be situated inferiorly to the norm.

From experience in clinical practice, the normative angle aĉd is 10°–20° depending on the size of the subject being measured. An excessive angle beyond these values would help confirm and quantify the observed and palpated anterior tilted pelvis. It is this same angle that contributes to monitoring progress of intervention and helps to form one outcome measure in the presence of a weak or non-functioning TrA (Figures 14.8 and 14.9). Using intentional

cognitive contraction of the pelvic floor muscles is considered to be one of the most effective methods of achieving isolation of contraction of TrA (Richardson et al. 2004). It is supported from clinical practice experience and provides additional palpable and measurable feedback. If the physiotherapist palpates the lower abdomen just superior to the mid-point of the inguinal ligament, prior to measuring movement, and asks the subject being examined to contract the muscles that would prevent opening the bowels and/or flow of urine, this should help the understanding of contracting the pelvic floor alone and prevent contraction and mobilisation of the pelvis by the other abdominal muscles. If the action of TrA is closely linked to that of the diaphragm and pelvic floor (Richardson et al. 2004), it seems reasonable to consider contracting what is hopefully a stronger muscle group in the subject in order to synergically elicit a contraction in the weaker or non-functioning muscle. Therefore, ensure the subject is fully relaxed in standing, palpate the lower abdomen and on giving the instruction to contract, palpate for activate muscle contraction. If the correct instruction has been given and the other abdominal muscles, such as the more superficial internal oblique, do not contract and a palpable contraction is palpated, it can be assumed to be that of TrA. This can be tested in the absence of TrA weakness on others and oneself. Examine in the same way as described and a strong contraction will be felt with the ASIS of the pelvis either not moving or moving minimally in a superior or cephalad direction.

The highly admirable and enlightening work of Richardson et al. (2002, 2004) has involved innovative use of equipment and measurement techniques, such as Doppler imaging of vibrations, electromyographic recordings and real-time ultrasound imaging in order to study and demonstrate the function of TrA. Research and clinical practice inform each other, but do not always share the same methodologies for a variety of reasons such as cost, availability and clinical pragmatism.

One alternative, low-technology technique includes the use of a universal goniometer and a sphygmomanometer – a pressure biofeedback unit (PBU) – with the cuff being placed under the lumbar spine in either supine or crook lying, and is described by Prentice (2004), citing a plethora of authors, which the reader is referred to. Functionally, however, these tests clearly do not involve TrA in isolation from other abdominal muscles if active and cognitive flattening of the lumbar lordosis is achieved and maintained, as the abdominal mobilisers have been brought into action. Many patients seen by physiotherapists may not be able to achieve the ability to conduct the tests outlined by Prentice (2004) because of other mechanical and pathological problems, but they can be used for the more able subjects and for those requiring more advanced rehabilitation.

Activation of TrA, independent of the global muscles, is advocated for examination and exercise techniques in low back pain (Richardson et al. 2002), but the physiotherapist would need to reason whether the anterior tilted pelvis and weak or non-functioning TrA is primary or secondary in nature, and address the primary problem in order to prevent recurrence of the situation. It is beyond the scope of this chapter to include all primary and other reasons for developing mechanical low back pain, an anterior tilted pelvis and a weak or non-functioning TrA muscle, and it remains for the physiotherapist to ask the right questions, about the right potential causations or formulate the right hypotheses and progressively address these with the right interventions that are safe, efficient and effective.

Clinical note

Programmes to rehabilitate and stabilise the lumbo-pelvic area, using predominantly TrA, have demonstrated equivocal efficacy (Goldby et al. 2006; Critchley et al. 2007), but they do have some support within clinical guidelines (Mercer et al. 2006; Savigny et al. 2009).

Analysis

As discussed above, an anterior tilted pelvis caused by a weak or non-functioning TrA may cause low back pain through, for example, increased facet joint compressive and/or frictional forces. Equally and conversely, the same muscle can be reflex-inhibited and develop weakness owing to low back pain.

In a very weak contraction, the ASIS of the pelvis may still not move, but this will be in the presence of an excessive aĉd angle. In a partially weak TrA, the ASIS will move superiorly or in a cephalad direction, either minimally or sufficiently to recognise a difference in the aĉd angle in a relaxed position in comparison with that of the contraction occurring on pelvic floor contraction. It is these two aĉd angles that need recording for detecting and monitoring the outcome of intervention. The value of the two angles may equal each other in the presence of a very weak TrA and a difference may occur as the muscle strengthens. The value of the two angles may be observed to converge again as it strengthens further and the relaxed aĉd angle may be seen to normalise towards the normative value. When the two values of relaxation and contraction are seen to converge within the normative value range, the TrA can be considered to be functioning in its stabilising role. If the mobilising abdominal muscles are in need of rehabilitation and TrA has been demonstrated to be functioning in its stabilising role as described, it should be safe to progress rehabilitation to include these muscles without causing other adverse mechanical stresses.

It must be noted that attempts to intentionally contract muscle and cognitively mobilise the pelvis actively will

induce mobilising muscle contraction and mask TrA contraction. The technique of inducing contraction of TrA as described above can be utilised for early rehabilitation; this will be described later.

Plan

It is important for the physiotherapist to always use accurate prescription in rehabilitation, based upon the evidence base from practice and research within literature or theory, or guidelines, in the absence of research and evidence base.

Technique

The core stabilising muscles are primarily type I slow-twitch muscle fibres that best respond to time (Prentice 2004), including TrA, and suitable exercise should be low load (i.e. 25% MVC), and longer hold periods (Mason 2008). Four levels of core stabilisation training programmes have been identified for athletes: level 1, stabilisation; level 2, stabilisation and strength; level 3, integrated stabilisation strength; and level 4, explosive stabilisation (Prentice 2004). If the athlete commences at a level where they maintain stability and optimal neuromuscular control and are progressed through the levels when they have achieved mastery of the previous level (Prentice 2004), it seems reasonable to follow the same programme and progression criteria for those patients requiring rehabilitation of TrA, albeit if most of these commence at level 1 and do not achieve beyond level 2.

Table 14.6 adopts the examples offered by Prentice (2004) for level 1. If the 'DRIFT' (duration, repetition, intensity, frequency, technique) acronym does not suit, alternative, but similar, factors known as the FITT (frequency, intensity, time (duration) and type of exercise) principles can be adopted instead (Sanghvi 2008) and may be easier to remember.

In practice and as discussed earlier, an alternative to the draw-in manoeuvre is pelvic floor contraction. Patients may find this easier to maintain when they progress to crunches/partial sit-ups, including more functional activity such as squats or lunge exercises. The same applies to level 2 exercises made more difficult by conducting, for example, sagittal and diagonal crunches/partial sit-ups on a stability ball or functional proprioceptive neuromuscular facilitation (PNF) pattern exercises on the same. The reader is referred to other texts such as Prentice (2004) for more advanced levels of core stability rehabilitation.

Table 14.6 Level 1 transversus abdominis rehabilitation programme – adapted from Prentice (2004)

Duration	The duration of a contraction should be calculated and checked to see if the same can be repeatedly achieved with a few seconds rest between each contraction. If necessary, slightly reduce the length of contraction time in order to afford greater repetitions and progress time as soon as possible
Repetition	The number of repetitions should be high, but may be determined by the actual contraction time ability. If contraction of only a few seconds can be achieved, these should be undertaken on a repetition rate of 10–12 repetitions
Intensity	Intensity or resistance should be low and the technique offered is sufficient in supine lying, standing or prone lying i.e. gravity-assisted, eliminated and resisted, respectively
Frequency	If the time period for available contraction is calculated to be low, e.g. 10–20 s, then hourly contractions would seem reasonable with frequency possibly reducing as contraction time ability increases, e.g. 60 s contraction every 2–4 h
Technique	In supine lying: Do: Abdominal draw-in manoeuvre. Progression can include sagittal, then diagonal crunches/partial sit ups in crook lying

Evaluation

Evaluation is through reanalysis of the subjective and objective examination markers such as pain, function, lumbo-pelvic posture/position, measured comparative pelvic levels and any other secondary related signs of muscle imbalance. Evaluation determines whether intervention is being effective by comparing the subjective and objective markers and whether to continue or modify the intervention. Progression criteria and progression techniques should be as indicated in the plan above.

THE CERVICAL SPINE REGION

The cervical spine, as well as bearing the weight of the head, is designed for mobility – principally for the positioning of the eyes' line of vision – and therefore requires movements with a high degree of precision. Subsequently, the cervical muscles (in particular those of the upper

cervical spine) are highly innervated (Middleditch and Oliver 2005). The suboccipital muscles, in particular, have a high concentration of muscle spindles (Peck et al. 1984) and act as 'proprioceptive monitors' (Fernández-de-las-Peñas et al. 2007). This presents its own potential problems. As with elsewhere in the body, there is usually a trade-off between mobility and stability, but high motor precision requires highly *balanced* motor function. The increased innervation, essential for the motor control, not only concerns motor output (descending pathways), but also necessitates increased sensory information (ascending pathways) for feedback purposes, etc. but may then make the head and neck more sensitive to the perception of pain.

Other key differences in the anatomy of the cervical spine compared with the lumbar spine, such as smaller vertebral bodies and thinner intervertebral discs, allow for greater mobility, but at the potential cost of stability (Panjabi's passive subsystem (Panjabi 2003)). Therefore, the role of the musculature (the active subsystem) is vitally important as it contributes significantly to the stability of the cervical spine.

Muscles

Movements of the cervical spine are complex as they are not single joint articulations and can also be differentiated into upper cervical or lower cervical movements, with the head being able to articulate on the neck. The assessment of the *quality* of the movement is pertinent. Table 14.7 outlines the key muscles associated with movements of the cervical spine and Table 14.8 highlights those muscles that predominantly act as stabilisers and mobilisers.

Deep cervical flexors

In the cervical spine, the deep cervical flexors (see Figure 14.10) – also referred to as the deep neck flexors – have been the subject of much research and clinical focus, similar to TrA in the lumbar spine (Jull et al. 1999, 2004, 2008). While named 'flexors', their role also includes stabilising the position of the head and upper cervical spine during prolonged postures and repeated movements, aided by their close approximation to the spine and predominantly parallel fibre direction. Their function has been shown to be impaired in people with cervicogenic headaches, whiplash associated disorder (WAD) and insidious neck pain (Jull et al. 1999, 2004), with delay in activation/inhibition, weakness and fatigue demonstrated. The mechanisms for this dysfunction include pain inhibition and altered postural and movement behaviour. The multifidus are less prominent in the cervical spine compared with the lumbar spine and, therefore, it appears less emphasis is placed on their stabilising role and contribution to pain and pathology in the literature.

Table 14.7 Cervical spine movements and their key muscles

Movement	Muscles
Flexion	Sternocleidomastoid (working bilaterally) Draws head anteriorly (protracts) Scalenus anterior (working bilaterally) Deep cervical flexors Longus colli (working bilaterally) Longus capitis Flexes head on neck/upper cervical Rectus capitis anterior Flexes head on neck; stabilise C0–C1
Extension	Levator scapulae (working bilaterally) Splenius cervicis (working bilaterally) Trapezius (working bilaterally) Upper fibres Splenius capitis (working bilaterally) Erector spinae (working bilaterally) Rectus capitis posterior minor Extend head on neck; stablise C0–C1; proprioceptive Rectus capitis posterior major Extend head on neck; stablise C0–C1; proprioceptive Obliquus capitis superior Extend head on neck; stablise C0–C1
Protraction	Sternocleidomastoid (working bilaterally) Longus capitis
Retraction	Deep cervical flexors Trapezius
Rotation	Semispinalis cervicis Multifidus/rotatores Scalenus anterior Splenius cervicis Sternocleidomastoid Splenius capitis Obliquus capitis inferior Rectus capitis posterior major
Lateral flexion	Scalenus anterior Scalenus medius Scalenus posterior Splenius cervicis Levator scapulae Sternocleidomastoid

Table 14.8 Cervical spine mobilisers and stabilisers

Local stabilisers	Global stabilisers	Global mobilisers
Anterior: Longus capitis Rectus capitis anterior Posterior: Rectus capitis posterior minor Rectus capitis posterior major Obliquus capitis superior Deep multifidus	Anterior: Longus colli Posterior: Superficial multifidus Spinalis Semispinalis	Scalenes Sternocleidomastoid hyoids Levator scapulae Upper trapezius Suboccipital extensors (Ligamentum nuchae – although not a muscle, it is significant to note as it can become tight)

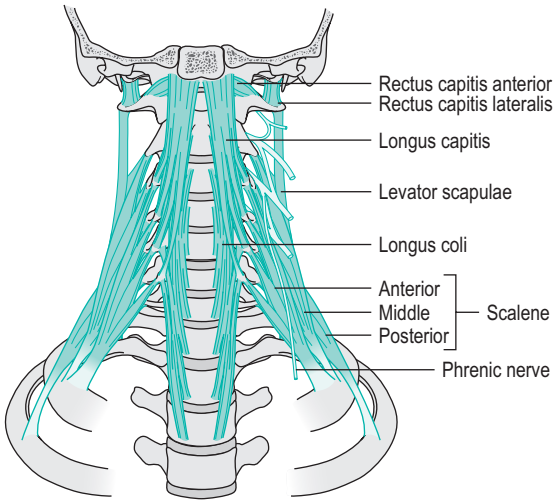


Figure 14.10 Anatomy of deep cervical flexors.

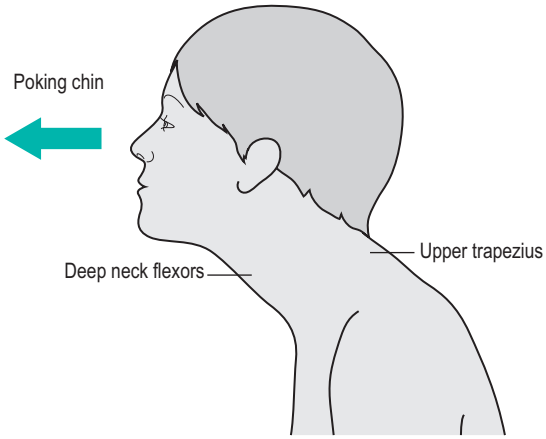


Figure 14.11 Forward head posture demonstrating the poking chin and muscle balance changes.

Posture

Forward head posture (FHP) – or 'poking chin' – has been associated with conditions of the head and neck, such as headaches (Watson and Trott 1993; Fernández-de-las-Peñas et al. 2007) and WAD (Nilsson and Söderlund 2005), but may also be associated with poor sitting and working conditions. The weakened and/or lengthened deep cervical flexors struggle to maintain an upright posture against overactive and/or shortened posterior extensors structures, such as the trapezius (see Figure 14.11). However, the involvement of the suboccipital muscles is disputed as, while it has been reported they can possess painful myofascial trigger points in individuals with FHP and chronic tension-type headaches (Fernández-de-las-Peñas et al. 2006), atrophy has also been demonstrated in some chronic neck pain subjects (Hallgren et al. 1994). Of course, posture needs to be assessed globally and in context of the presenting problem, taking into account the wide range of what is considered 'normal'.

Clinical note

This may be the proverbial 'chicken and egg' situation: Which came first?

- Pain inhibition, weakness and fatigue of the deep cervical flexors, following trauma or pathology, for example, results in FHP.

Or

- FHP is adopted either in an attempt to gain pain relief following trauma/pathology or owing to activities, such as prolonged poor sitting posture in response to work demands or video game playing. This may then result in pain inhibition, weakness and fatigue of the deep cervical flexors.

Or

- FHP is pre-existing, for example, owing to lumbar spine problems or pathology.

Table 14.9 Patient example of cervical muscle imbalance using the SOAPE framework

Subjective	Objective	Analysis	Plan	Evaluation
Pain posterior neck and headache occipital area. Worse after day at work (using computer)	Forward head posture ↓ Cervical range of movement (ROM), especially flexion and rotation C-CFT = unable to hold for 10 s	Weak deep cervical flexors Tight trapezius	Re-train deep cervical flexors (± PBU) Gentle stretch trapezius (end-range flexion) Postural/ergonomic advice	Re-assess C-CFT and ROM, then progress exercises

FHP may also be associated with shortening and overactivity of sternocleidomastoid and the scalenes (particularly the anterior). The superficial cervical flexors are thought to compensate for the poor motor control and have been show to be used more by those with WAD or insidious neck pain than in asymptomatic subjects (Jull et al. 2004). As the superficial flexors are preferentially biased to being mobilisers rather than stabilisers it appears that they are more susceptible to fatigue and pain when over-worked (Watson and Trott 1993), adding to the general irritability in the neck and, potentially, the development of myofascial trigger points (Roth et al. 2007).

Assessment and treatment

The well-documented cranio-cervical flexion test (C-CFT) has been developed and validated to assess the impairment of the deep cervical flexors (Jull et al. 1999, 2004, 2008). This involves flexing (as in nodding) the head on the neck so that the chin is gently tucked in. A PBU is used in order to focus on low-load endurance and provide a more objective measure, while compensatory strategies, such as the use of sternocleidomastoid and platysma (the 'shaving' muscle), are noted but discouraged. The same exercise performed as a training regime using the PBU with a starting pressure of 20 mmHg and held for 10 seconds for 10 repetitions, then increasing in 2 mmHg increments up to 30 mmHg, has demonstrated a reduction in headache and neck pain in subjects with cervicogenic headaches (Jull et al. 2002) and has been recommended in the management of WAD (Moore et al. 2005). The author's clinical experience has demonstrated that the deep cervical flexors can be assessed and treated in a similar manner even without a PBU, only using careful instruction, observation and motivation. Table 14.9 illustrates an example patient's problem documented using the SOAPE framework.

THE SHOULDER COMPLEX

The shoulder complex is made up of four key articulations: the sternoclavicular, acromioclavicular, scapulo-thoracic and glenohumeral joints. In addition to this, within the kinetic chain, it is important to consider the cervical and thoracic spinal joints when assessing movement dysfunction in this area. Considering Panjabi's (2003) model, the passive subsystem providing stability to the glenohumeral joint consists of the glenohumeral ligaments, the glenoid labrum, passive muscle stiffness of the rotator cuff muscles as they insert into the capsule and negative intra-articular pressure within the joint (Hess 2000). Likewise, the bony strut of the clavicle with its capsular and ligamentous attachments at the acromioclavicular and sternoclavicular joints provides stability for the scapula (Kibler and Sciascia 2010). Examination and assessment of these structures, as indicated by the subjective history of the patient, is necessary as part of the routine musculoskeletal assessment.

As the focus of this chapter is muscle imbalance, an overview of movement at the shoulder complex and the key muscles providing static and dynamic stability will be discussed as part of the active subsystem referred to by Panjabi (2003).

Scapulo-humeral rhythm

Despite controversy in the literature over the ratio of scapula rhythm (Freedman and Munro 1966; Doody et al. 1970; Poppen and Walker 1976; Bagg and Forrest 1988; McClure et al. 2001), it is commonly reported, in accordance with Inman (1944), that the scapulo-humeral rhythm has a ratio of 2:1. Inman (1944) states that the scapula seeks a precise position of stability during the first 30°–60° of movement (the setting phase) followed by a constant relationship of humeral to scapular motion of 2:1, whereby between 30° and 170° for every 15° of motion, 10° occurs at the glenohumeral joint and 5° at the scapula-thoracic joint. This movement is accompanied by clavicular movements at the sternoclavicular joint in the early part of range and at the acromioclavicular joint both early and late in the range of elevation.

Functionally, the scapula provides a stable base for the muscle attachments to the glenohumeral joint and serves to maintain the correct length tension relationship of these muscles (rotator cuff, teres major, deltoid,

coraco-brachialis, long head of biceps). Dynamically, its function is to upwardly rotate in order to orientate the glenoid fossa in an upward forward and lateral position and to tilt posteriorly and externally rotate (Kibler and Sciascia 2010). These movements allow the clearance of the acromion from the moving arm and maintenance of the glenoid as a congruent socket for the moving arm. This allows greater movement of the upper limb and elevates the acromion such that there is adequate space in the subacromial and coraco-acromial arches to avoid impingement of soft tissue structures within these spaces.

At the glenohumeral joint, the stabilising muscles maintain and allow fine control of movement of the humerus on the glenoid. Slight lateral rotation of the humeral head is initiated in the early phase of abduction. This should continue throughout the range as the scapula upwardly rotates (Comerford and Kinetic Control 2001). The lateral rotation of the humerus on elevation of the arm allows for greater space between the greater tubercle and the acromion.

Integral to this co-ordination of movement between the scapulo-thoracic and glenohumeral joints at the shoulder complex is motor control, as discussed earlier.

Muscles acting on the scapulo-thoracic joint

The muscles of trapezius and serratus anterior form the proximal force couple and in their stability role maintain the scapula in a stable position on the chest wall. In dynamic stability, the key muscles which control upward rotation of the scapula are the upper and lower fibres of trapezius and serratus anterior (Figure 14.12). Additionally, the serratus anterior helps produce posterior tilt and external rotation of the scapula while stabilising the medial border and inferior angle to prevent scapula winging (Kibler and Sciascia 2010).

Table 14.10 indicates the muscles acting on the scapulo-thoracic joint, their agonist actions and their primary roles as mobilisers or stabilisers. As discussed earlier, the stabiliser muscles are expected to be recruited earlier than the mobilisers (Sahrmann 1987; Richardson 1992; Comerford and Mottram 2001). Muscle imbalance at the scapulo-thoracic joint can be seen by the dominance of the mobiliser muscles where the stabilisers are weak or slower to be recruited. The rhomboids, levator scapulae and pectoralis minor muscles, although they have a minor

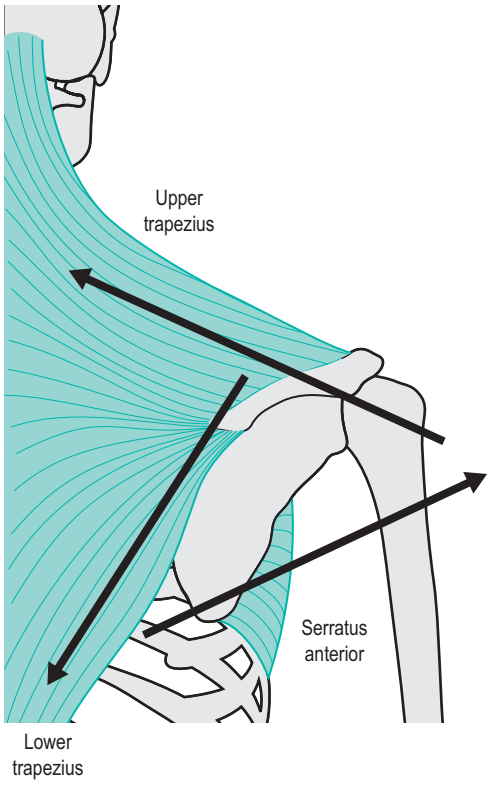


Figure 14.12 The key muscles controlling rotation of the scapula, which act as a 'force couple'.

Table 14.10 Scapulo-thoracic joint muscles

Action	Muscles	Primary muscle role
Elevation	Upper trapezius	Local/global stabiliser
	Levator scapula	Global mobiliser
Depression	Lower trapezius	Local stabiliser
Adduction	Middle trapezius	Local stabiliser
	Rhomboids	Global mobiliser
Abduction	Serratus anterior	Global stabiliser
	Pectoralis minor	Global mobiliser
Upward rotation	Serratus anterior	Local/global stabilisers
	Trapezius	
Downward rotation	Rhomboids	Global mobilisers
	Levator scapulae	
	Pectoralis minor	

stability role of the scapula on the chest wall, primarily act as mobilisers of the scapula. Because of the synergistic actions of rhomboids and levator scapulae with trapezius in relation to adduction and elevation, they can become dominant when trapezius is weak on these activities and therefore can limit upward rotation with their antagonist role as downward rotators. Likewise, pectoralis minor can become dominant and shortened in its synergist activity of abduction of the scapula with serratus anterior leading to an increase in its activity of downward rotation of the scapula when serratus anterior is weak. This will limit upward rotation. It must not be forgotten that latissimus dorsi through its attachment onto the inferior angle of the scapula may also have a downward rotation action on the scapula.

This lack of upward rotation, as indicated earlier, leads to narrowing of the subacromial and coraco-acromial arches and, hence, the risk of impingement. Ludewig and Cook (2000) and Cools et al. (2003) have identified alterations of activation sequencing patterns and strength of the scapula stabilising muscles in patients with impingement.

Muscles acting on the glenohumeral joint

Table 14.11 indicates the key roles of the muscles acting on the gleno humeral joint

The forces of the rotator cuff (supraspinatus, subscapularis, infraspinatus and teres minor) provide compression and depression to counteract the upward pull of deltoid and therefore maintain the humeral head on the glenoid cavity (i.e. in its neutral zone). This relationship continues between the rotator cuff and deltoid throughout elevation of the arm (Figure 14.13). Should there be any weakness in the rotator cuff such that the balance of forces becomes unequal, this can result in a greater pull from deltoid such that the humeral head moves superiorly under the acromion leading to impingement.

At the gleno humeral joint, the global mobilisers (pectoralis major and latissimus dorsi) can be recruited early and become dominant when the local stabiliser

Table 14.11 Key roles of the muscles acting on the glenohumeral joint

Local stabilisers	Global stabilisers	Global mobilisers
Supraspinatus Infraspinatus Teres minor Subscapularis	Teres major Deltoid Coracobrachialis Long head biceps Some fibres of subscapularis, infraspinatus and teres minor	Latissimus dorsi Teres major Pectoralis major Teres minor Infraspinatus Short head biceps Long head triceps

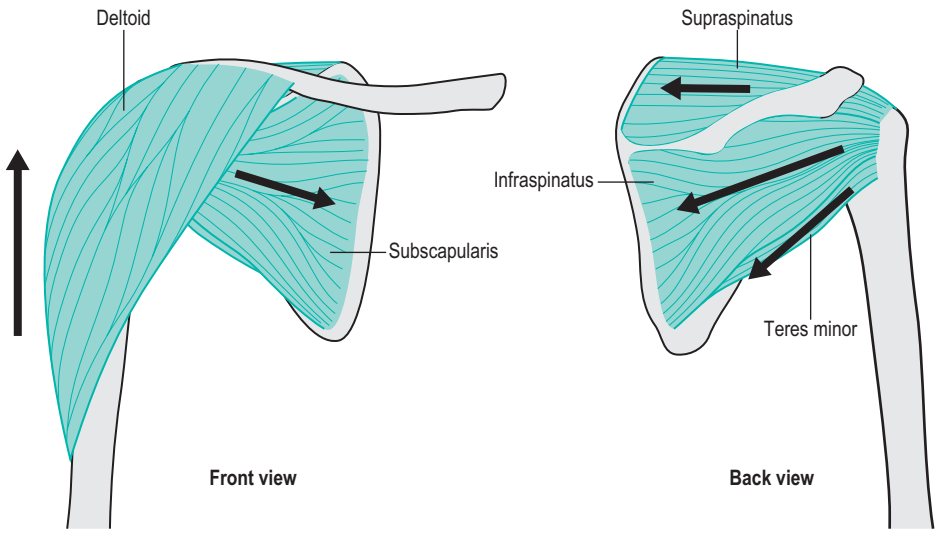


Figure 14.13 Action of the rotator cuff and deltoid.

(subscapularis) is weak. Although infraspinatus and teres minor's key role is stability as part of the rotator cuff, there can be a tendency for these muscles to take on a mobilising role and be seen to lack extensibility, which can lead to anterior translation of the humeral head (Comerford and Kinetic Control 2001) and glenohumeral internal rotation deficit (GIRD). Rotator cuff weakness may be demonstrated for many reasons, for example actual injury, disuse atrophy, inhibition owing to pain or decreased muscle activation patterns owing to lack of a stable base (Kibler and Sciascia 2010). It is therefore essential that a complete musculoskeletal assessment is carried out to identify whether weakness is a result of muscle imbalance or some other cause.

A simplification of the muscles commonly identified in muscle imbalance of the shoulder complex is presented in Table 14.12.

Clinical presentations of the shoulder complex

The following provides a guide with regard to typical findings relating to muscle imbalance around the shoulder complex and its link with impingement. Further reading is suggested to gain a full understanding of the assessment and treatment principles relating to muscle imbalance for this region. An evidence-based approach to the diagnosis assessment and physiotherapeutic management of shoulder impingement syndrome is provided by Hanchard et al. (2004).

Table 14.12 Commonly affected muscles of the shoulder complex

Joint	'Goodies'	'Baddies'
	Muscles expected to initiate and control 'normal' scapula movement or maintain the humeral head centred on the glenoid	Muscles which take over when 'goodies' become long and weak
Scapulo-humeral	Upper and lower trapezius Serratus anterior	Levator scapulae Rhomboid minor and major Pectoralis minor
Gleno-humeral	Supraspinatus Subscapularis Teres minor Infraspinatus	Latissimus dorsi Teres major Pectoralis major Deltoid

Causes of shoulder pain

The link between impingement symptoms and shoulder instability is well established (Cools et al. 2007; Hanchard et al. 2004). Excessive humeral head translation can lead to narrowing of the subacromial or coracoacromial spaces. Identification of functional problems that lead to impingement, i.e. dysfunction of the position of the humeral head on the glenoid or scapula dyskinesis (Cools et al. 2003; Kibler and Sciascia 2010), is essential to the choice of muscle balance techniques for treatment. Signs of primary impingement (a structural narrowing of the subacromial space, for example acromioclavicular arthropathy, type I acromion, swelling of the soft tissue in the subacromial space (Cools et al. 2003)) or structural instability of the glenohumeral joint (Type I and Type II Stanmore classification (Lewis et al. 2004)) must be ruled out.

The SOAPE process (Baxter 2003) is used to discuss common clinical presentations.

Subjective

A person with impingement symptoms can present with:

- pain C5 dermatome;
- painful arc;
- pain aggravated by repetitive overhead activity.

Objective and analysis

Typical movement faults identified are:

- scapula – see Tables 14.13 and 14.14, and Figure 14.14;
- glenohumeral joint – see Table 14.15.

Figure 14.14 Pseudo-winging of the scapula.

Table 14.13 On concentric movement of arm elevation

Objective signs	Analysis
Early movement of scapula where scapula elevates rather than upwardly rotates on abduction	Early recruitment/ overactivity of upper fibres of trapezius and/ or levator scapula
Excessive scapula elevation or protraction on flexion	Weakness of lower fibres of trapezius such that the pull of upper fibres of trapezius and serratus anterior is not counterbalanced
At end-range, scapula inferior angle does not move around to mid axillary line	Shortening/overactivity of downward rotation of the scapula or latissimus dorsi
Downward rather than upward rotation of scapula	Weakness of upward rotators and shortening/early recruitment of downward rotators
Winging of the scapula	Whole medial border – weakness of serratus anterior Psuedo-winging (see Figure 14.14), i.e. inferior angle only – shortening of pectoralis minor

Table 14.14 Eccentric movement

Objective signs	Analysis
Excessive downward rotation of scapula seen as scapula drops on lowering at 90° flexion or 60° abduction	Early recruitment of downward rotators in an attempt to substitute for weak serratus anterior and trapezius during eccentric muscle activity
Winging of the scapula	Weakness of serratus anterior and lower fibres of trapezius resulting in poor scapula stability (Figure 14.15)

Table 14.15 Glenohumeral joint

Objective signs	Analysis
Excessive inferior translation of humerus noted as deep posterior acromial dimple and bulge of humeral head in axilla	Weakness of supraspinatus in providing caudal glide at the humeral head to maintain humeral head centred on the glenoid
Excessive medial rotation on flexion	Overactivity/shortening of latissimus dorsi and/or pectoralis major
Absent or late lateral rotation on abduction	Late recruitment of infraspinatus and/or teres minor

Figure 14.15 Poor scapular control on eccentric lowering of the arm.

Plan (Treatment)

Once a hypothesis has been reached regarding the likely muscle dysfunction, treatment commences with monitoring the patient's symptoms on facilitating the neutral position of the affected joint with low load activity to recruit the weakened muscle groups, for example stabilisation of the scapula on the chest wall. Once this is achieved, the individual is challenged to control the scapula position on movement, for example flexion or lateral rotation (Figures 14.15, 14.16 and 14.17).

All exercises must be pain-free and are required to be performed without fatigue of the muscles being facilitated or substitution by the synergists. Functional movements relating to the patient's activities are encouraged to facilitate compliance. Studies to identify recruitment patterns of the scapular muscles and the optimal shoulder exercises used to facilitate scapular control have been carried out by Kibler et al. (2008), Cools et al. (2003, 2007) and De May et al. (2009).

Figure 14.16 Scapula stabilisation is encouraged on active forward flexion of the arm aiming to avoid elevation of the shoulder girdle and to facilitate normal timing of the upward rotation of the scapula.

Figure 14.17 Scapula neutral has been facilitated on the left and the individual is encouraged to maintain this position on active lateral rotation of the left arm. Also note the increased skin creases in the thoraco-lumbar region demonstrating a 'give' in this region, possibly owing to poor spinal postural control.

Once pain-free, functional movement is achieved any residual muscle tightness is addressed with stretching techniques specific to the muscle affected, making sure that the joints remain in their neutral zone. Postural correction is facilitated and encouraged throughout treatment.

Evaluation

There is an extensive body of literature relating to kinematics of the shoulder complex using complex and expensive motion analysis systems to evaluate movement (Imtiyaz and Kelly 2001; Borstad and Ludewig 2002; Lin et al. 2005) and it is still growing. Monitoring of scapula position and humeral head position in the clinical situation tends to be by visual observation and can be difficult to measure objectively. Video analysis can assist with this process and provide visual feedback to the patient to facilitate normal movement. Evaluation of reduction of pain measured by the numerical rating scale or visual analogue scale on correction of movement dysfunction is the most commonly used outcome measure in the clinical situation.

THE KNEE

There is an inherent imbalance between the major muscle groups of the lower limb that affect both the hip and, in particular, the knee, as the quadriceps are usually stronger than the hamstrings, as to be expected by their greater size. The ratio of the strength of the hamstrings to the strength of the quadriceps can be expressed as the H:Q ratio; the normal range being 50–80% (Rosene et al. 2001; Norris 2004). This, along with their increased eccentric contraction workload, can explain why the hamstrings are more commonly injured in sports, for example. Furthermore, this 'natural' imbalance can be increased by training regimes that favour the quadriceps, such as squats and the leg press.

Vastus medialis oblique

Within the quadriceps group and specifically affecting the patellofemoral joint, there exists another potential imbalance. The vastus lateralis (VL) stabilise and control the lateral glide (or often termed 'tracking') of the patella, while the medial glide is controlled by the vastus medialis, specifically the oblique fibres (vastus medialis oblique (VMO)). These oblique fibres have been reported to be orientated approximately 50° medially compared with less than 20° for the 'longus' fibres (the so-called vastus medialis longus (VML)). This apparent difference has caused debate as to whether VMO is anatomically, as well as functionally, distinct from VML, as it is suggested that VML extends the knee instead (Smith et al. 2009). VMO activity is thought to be most critical during the final 20° extension/first 20° flexion (see Figure 14.18), as it ensures optimal seating of the patella within the trochlear.

As it has also been demonstrated that small changes in VMO activity can have significant effects on patellar position, there has been much interest in assessing and treating the VMO. VMO has been reported as being far more easily inhibited than VL in response to pain, whereas VMO normally exhibits faster reflex response times than VL, such that patellofemoral pain may become a secondary feature of other knee trauma or pathology (McConnell 1996).

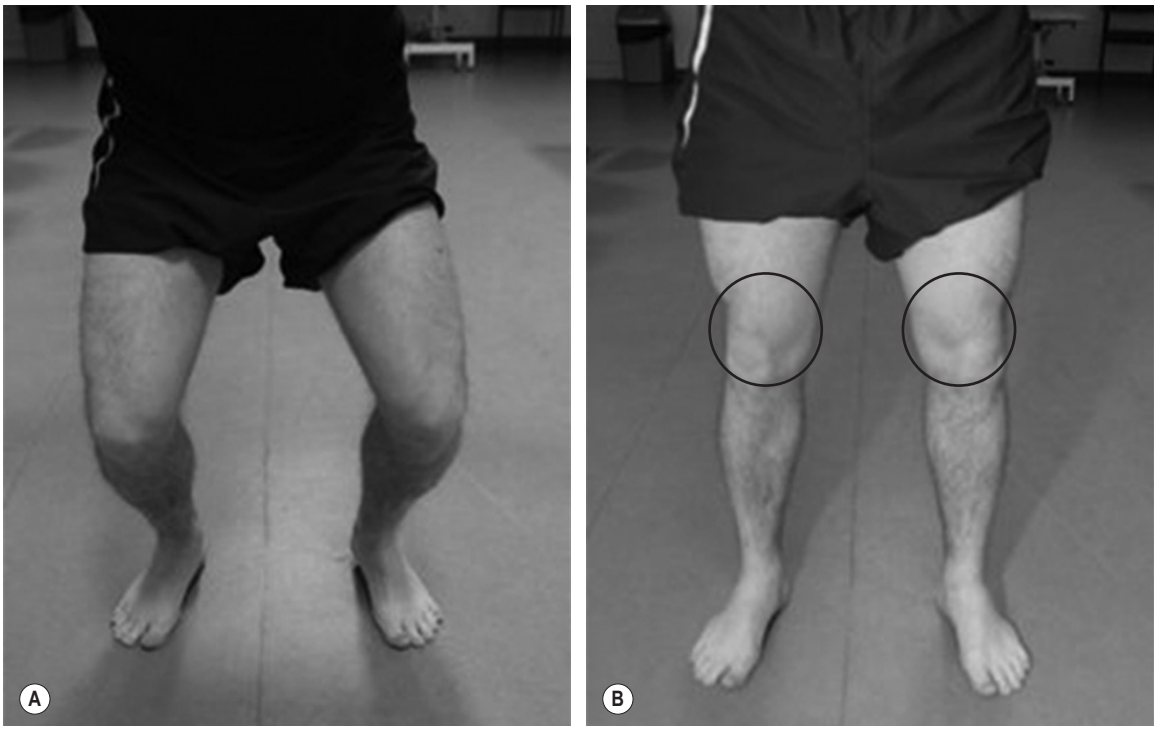


Figure 14.18 (a, b) Vastus medialis oblique (highlighted) working during extension.

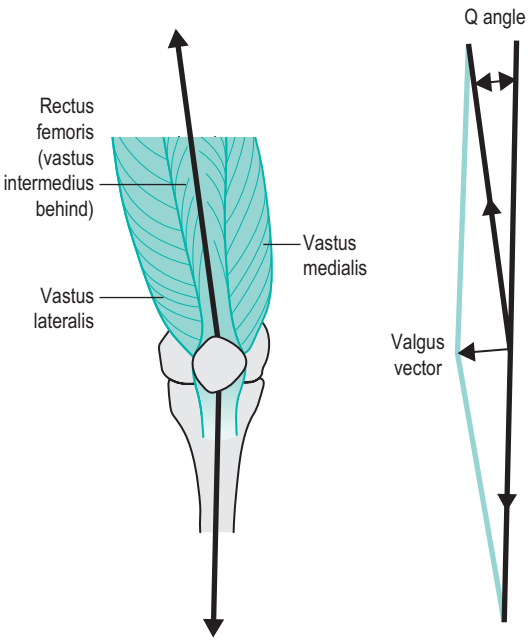


Figure 14.19 Illustration of the pull of the quadriceps and the Q angle.

Assessment

However, the stability of the patella is not without influence from other factors. Owing to the direction of action of the quadriceps (represented by a line from the ASIS to the centre of the patella, or longitudinal axis of the femur) relative to the direction of pull from their attachment on the tibia via the patella tendon (represented by a line along the longitudinal axis of the tibia) – termed the Q angle (see Figure 14.19) – there is a resultant valgus vector. The Q angle is usually described as being approximately 15° for men and approximately 20° for women. An increased angle is thought to be a risk factor for patellofemoral pain (McConnell 1996; Powers 2003). This, combined with the presence of the iliotibial band (ITB) – which in itself can become tight – means that the VMO, as the sole medial stabiliser, can be at a significant disadvantage.

The quadriceps are also commonly prone to tightness (Mason 2008) and consideration needs to be given to rectus femoris as it also crosses the hip. Figure 14.20 illustrates the resultant compressive biomechanical forces exerted on the patellofemoral joint when the knee is flexed and this is increased if the quadriceps are too tight or too short (passively insufficient). In addition to

Table 14.16 Patient example of vastus medialus oblique (VMO) muscle imbalance using the SOAPE framework

Subjective	Objective	Analysis	Plan	Evaluation
Left anterior knee pain Pain on stairs, descent worse	Full range of movement but pain end-range Pain on small knee bend test Left VMO atrophy Tight left iliotibial band (ITB) Reduced left patella medial glide	Increased lateral tracking from VMO/ITB imbalance	Patella taping – medial glide VMO squat ITB stretch	Retest small knee bend with tape on (painfree = positive result) Retest Ober's Progress squat to single-leg, then stepping

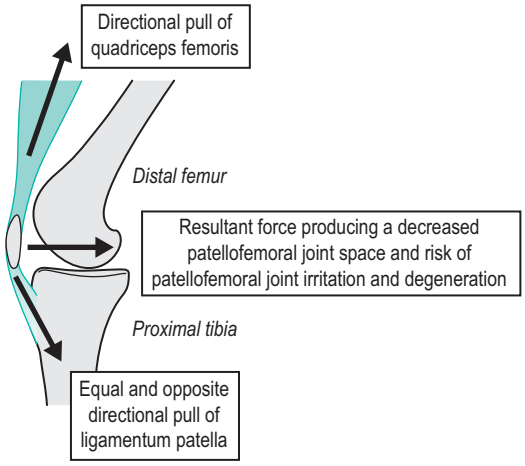


Figure 14.20 Resolution of forces; the resultant force producing prospective patellofemoral problems caused by a passively insufficient rectus femoris.

this compression, quadriceps tightness may lead to the patella sitting higher in relation to the femoral trochlear (patella alta) and therefore affect its engagement and smooth movement. Subjectively, the patient may complain of anterior knee pain and, objectively, there will be evidence of shortened muscles in a passive prone lying knee bend and possibly on accessory movements of the patellofemoral joint with potential patella malalignment present. The Thomas test may be a useful assessment tool to assess the involvement of rectus femoris in relation to iliopsoas, as well as observing medio-lateral deviation (i.e. tight adductors or lateral structures such as ITB, which can be then assessed using Ober's test).

Treatment/management

VMO rehabilitation exercises, such as end-range squats, have long been recommended (McConnell 1996) and there has been some positive research supporting their use (Cowan et al. 2002; Crossley et al. 2002), although, overall, evidence for the effectiveness of exercise therapy for patellofemoral pain syndrome has been described as limited (Heintjes et al. 2003).

Adjuncts to VMO training include ITB/lateral retinaculum stretching and patellar taping, which has been claimed to improve lateral tracking of the patella and the proprioceptive stimulation of the VMO, although the evidence for its efficacy and mechanism has been disputed (Clark et al. 2000; Tobin and Robinson 2000; Hinman et al. 2003; Christou 2004).

In addition, the effect of other, more distal influences (e.g. foot posture) or proximal influences, such as including the gluteus medius' contribution to maintaining lower limb posture and knee stability (Cowan et al. 2002), need to be addressed. Table 14.16 illustrates an example patient's problem, documented using the SOAPE framework.

Clinical note

As in most areas of physiotherapy (if not healthcare in general), myths and controversy surround the subject and interventions can become fads and accepted *en masse* as a panacea; muscle imbalance (and '*core stability*' – defined later) are, perhaps, the epitome of this (see Lederman 2007; Bee 2010). While physiotherapy may appear an 'art form' in application of practice, it is the science that has to be evaluated and a physiotherapist ignores either at his/her peril.

Unfortunately this is usually propagated by an imbalance in the literature itself and, perhaps, its subsequent evaluation. Clinical observations and early research indicated that certain patient populations exhibited changes in muscle function or balance (i.e. those with low back pain had TrA dysfunction compared with asymptomatic subjects). This seemed to suggest that this could be reversed/addressed and various interventions (i.e. exercises) were proposed, many of which were adopted by clinicians without substantial evidence of their efficacy. Even now, the evidence supporting interventions addressing muscle balance is still lacking.

ACKNOWLEDGEMENTS

We are very fortunate and appreciative of having had the opportunity to work closely with colleagues over the years who have entered great informal and non-threatening clinical discussion and debate. One physiotherapist who particularly engaged in this was Jane Brazendale (clinical lead physiotherapist in women's health). She was the first to openly discuss and approve ideas regarding the TrA/pelvic level measurement in the early years of the last decade. We have all been developed by our patients, education and our colleagues, and we thank our colleagues for their healthy contributions to peer development. Alec would also like to thank Sìne Rickard, his wife and physiotherapist, who provided much needed support while heavily pregnant.

REFERENCES

Bagg, S.D., Forrest, W.I., 1988. A biomechanical analysis of scapular rotation during arm abduction in the scapular plane. Am Phys Med Rehabil 67, 238–245.

Baxter, R.E., 2003. Pocket Guide to Musculoskeletal Assessment, second ed. Elsevier, Philadelphia.

Bee, P., 2010. The core stability myth. The Times http://www.thetimes.co.uk/tto/health/diet-fitness/article2679975.ece, accessed August 2010.

Bergmark, A., 1989. Stability of the lumbar spine: A study in mechanical engineering. Acta Orthopedica Scandinavica Suppl, 1–54.

Borstad, J.D., Ludewig, P.M., 2002. Comparison of scapular kinematics between elevation and lowering of the arm in the scapular plane. Clinical Biomechanics 17 (9–10), 650–659.

Christou, E.A., 2004. Patellar taping increases vastus medialis oblique activity in the presence of patellofemoral pain. J Electromyogr Kinesiolo 14 (4), 495–504.

Clark, D.I., Downing, N., Mitchell, J., et al., 2000. Physiotherapy for anterior knee pain: a randomised controlled trial. Ann Rheum Dis 59, 700–704.

Clarkson, H.M., Gilewich, G.B., 1989. Musculoskeletal Assessment: Joint Range of Motion and Manual Muscle Strength. Lippincott, Williams and Wilkins, Baltimore.

Clarkson, H., 2000. Musculoskeletal Assessment: Joint Range of Motion and Manual Muscle Strength, second ed. Lippincott, Williams and Wilkins, Philadelphia.

Comerford, M., Kinetic Control, 2001. Kinetic Control: Dynamic Balance of the Sensory Motor System, Dynamic Stability and Muscle Imbalance of the Cervical Spine and Shoulder Girdle. Course Handbook. Kinetic Control Ltd, Southampton.

Comerford, M.J., Mottram, S.L., 2001. Movement and stability dysfunction – contemporary developments. Man Ther 6 (1), 15–26.

Cools, A.M., Witvrouw, E.E., Declercq, G.A., et al., 2003. Scapular muscle recruitment patterns: trapezius muscle latency with and without impingement symptoms. Am J Sports Med 31, 542–549.

Cools, A.M., Dewitte, V., Lanszweert, F., et al., 2007. Rehabilitation of scapular muscle balance: Which exercises to prescribe? Am J Sport Med 35 (10), 1744–1751.

Cowan, S.M., Bennell, K.L., Crossley, K.M., et al., 2002. Physical therapy treatment alters the recruitment of the vasti in patellofemoral pain syndrome. Med Sci Sport Exerc 34 (12), 1879–1885.

Critchle, D.J., Ratcliffe, J., Noonan, S., et al., 2007. Effectiveness and cost-effectiveness of three types of physiotherapy used to reduce chronic low back pain disability: A pragmatic randomized trial with economic evaluation. Spine, 32 (14), 1474–1481.

Crossley, K., Bennell, K., Green, S., et al., 2002. Physical therapy for patellofemoral pain: a randomized, double-blinded, placebo-controlled trial. Am J Sports Med 30, 857–865.

De May, K., Cagnie, B., Van de Velde, A., et al., 2009. Trapezius muscle timing during selected shoulder rehabilitation exercises. J Orthopaed Sport Phys Ther 39 (10), 743–752.

Doody, S.C., Freedman, L., Waterland, J.C., 1970. Shoulder movements during abduction in the scapular plane. Arch Phys Med Rehab 1, 595–604.

Fernández-de-las-Peñas, C., Alonso-Blanco, C., Cuadrado, M.L., et al., 2006. Trigger points in the suboccipital muscles and forward head posture in tension-type headache. Headache 46, 454–460.

Fernández-de-las-Peñas, C., Pérez-de-Heredia, C., Molero-Sánchez, M.A., et al, 2007. Performance of the craniocervical flexion test, forward head posture, and headache clinical parameters in patients with chronic tension-type headache: a pilot study. J Orthopaed Sport Phys Ther 37 (2), 33–39.

Freedman, L., Munro, R.R., 1966. Abduction of the arm in the scapular plane: Scapular and glenohumeral movements. J Bone Joint Surg Am 48 (8), 1503–1510.

Gibbons, S.G.T., Comerford, M.J., 2001. Strength versus stability: Part 1: Concept and terms. Orthopaed Div Rev March/April, 21–27.

Goldby, L.J., Moore, A.P., Doust, J., et al., 2006. A randomised controlled trial investigating the efficiencey of musculoskeletal physiotherapy on chronic low back back disorder. Spine, 31 (10), 1083–1093.

Gossman, M.R., Sahrmann, S.A., Rose, S.J., 1982. Review of length associated changes in muscle. Phys Ther 62 (12), 1799–1808.

Grimby, L., Hannerz, J., 1976. Disturbances in voluntary recruitment order of low and high frequency motor units on blockades of proprioceptive afferent activity. Acta Physiol Scand 96, 207–216.

Hallgren, R.C., Greenman, P.E., Rechtien, J.J., 1994. Atrophy of suboccipital muscles in patients with chronic pain: a pilot study. J Am Osteopath Assoc 94, 1032–1038.

Hanchard, N., Cummins, J., Jeffries C., 2004. Evidence based clinical guidelines for the diagnosis, assessment and physiotherapy management of shoulder impingement syndrome. Chartered Society of Physiotherapy, London.

Heintjes, E.M., Berger, M., Bierma-Zeinstra, S.M.A., et al., 2003. Exercise therapy for patellofemoral pain syndrome. Cochrane Database Syst Rev (4), CD003472.

Hess, S.A., 2000. Functional stability of the glenohumeral joint. Man Ther 5 (2), 63–71.

Hinman, R.S., Crossley, K.M., McConnell, J., et al., 2003. Efficacy of knee tape in the management of osteoarthritis of the knee: blinded randomised controlled trial. BMJ 327, 135–138.

Imtiyaz, T.S., Kelly, C., 2001. Movement analysis of asymptomatic normal shoulders: A preliminary study. J Shoulder Elbow Surgery 10 (6), 580–584

Inman, V.T., Saunders, J.B., Abbott, L.C., 1944. Observations on the function of the shoulder joint. J Bone Joint Surg 26, 1–30.

Janda, V., 1983. On the concept of postural muscles and posture in man. Aust J Physiother 29 (3), 83–84.

Janda, V.L., 1994. Muscles and motor control in cervicogenic disorders: assessment and management. In: Grant, R. (Ed.), Physical Therapy of the Cervical and Thoracic Spine, second ed. Churchill Livingstone, Edinburgh, pp. 195–216.

Jull, G.A., Janda, V., 1987. Muscles and motor control in low back pain. In: Twomey, L.T. (Ed.), Physical Therapy of the Low Back. Churchill Livingstone, Edinburgh.

Jull, G., Barrett, C., Magee, R., et al., 1999. Further clinical clarification of the muscle dysfunction in cervical headache. Cephalalgia 19, 179–185.

Jull, G., Trott, P., Potter, H., et al., 2002. A randomized controlled trial of exercise and manipulative therapy for cervicogenic headache. Spine 27 (17), 1835–1843.

Jull, G., Kristjansson, E., Dall'Alba, P., 2004. Impairment in the cervical flexors: a comparison of whiplash and insidious onset neck pain patients. Man Ther 9, 89–94.

Jull, G.A., O'Leary, S.P., Falla, D.L., 2008. Clinical assessment of the deep cervical flexor muscles: The craniocervical flexion test. J Manipulative Physiol Ther 31 (7), 525–533.

Kendall, F.P., McCreary, E.K., Provance, P.G., et al., 2005. Muscles Testing and Function with Posture and Pain, fifth ed. Lippincott, Williams and Wilkins, Baltimore.

Kibler, W.B., Sciascia, A., 2010. Current concepts: scapular dyskinesis. Br J Sport Med 44, 300–305.

Kibler, W.B., Sciascia, A.D., Uhl, T.L., et al., 2008. Electromyographic analysis of specific exercises for scapular control in early phases of shoulder rehabilitation. Am J Sport Med 36, 1789–1798.

Lederman, E., 2007. The myth of core stability. PPA News 23, 8–20, http://www.ppaonline.co.uk/download/Myth_of_Core_Stability_PPA.pdf.

Lewis, A., Kitamura, T., Bayley, J.I.L., 2004. The classification of shoulder instability: new light through old windows! Current Orthopaedics 18, 97–108.

Lin, J., Hanton, W.P., Olsen, S.L., et al., 2005. Functional activity characteristics of individuals with shoulder dysfunction. J Electromyography Kinesiol 15, 576–586.

Ludewig, P.M., Cook, T.M., 2000. Alterations in shoulder kinematics and associated muscle activity in people with symptoms of shoulder impingement. Phys Ther 80, 276–291.

Mason, D., 2008. Exercise in rehabilitation. In: Porter, S. (Ed.), Tidy's Physiotherapy, fourteenth ed. Churchill Livingston, Edinburgh.

McClure, P.W., Michener, L.A., Sennett, B.J., et al., 2001. Direct 3-dimensional measurement of scapula kinematics during dynamic movements in vivo. J Shoulder Elbow Surg 10, 269–271.

McConnell J., 1996. Management of patellofemoral problems. Man Therapy 1, 60–66.

Mercer, C., Jackson, A., Hettinga, D., et al. 2006. Clinical Guidelines for the Physiotherapy Management of Persistent Low Back Pain, Part 1: Exercise. Chartered Society of Physiotherapy, London.

Middleditch, A., Oliver, J., 2005. Functional Anatomy of the Spine, second ed. Butterworth-Heinemann, Edinburgh.

Moore, A., Jackson, A., Jordan, J., et al., 2005. Clinical Guidelines for the Physiotherapy Management of Whiplash-Associated Disorder. Chartered Society of Physiotherapy, London.

Nilsson, B-M., Söderlund, A., 2005. Head posture in patients with whiplash-associated disorders and the measurement method's reliability – A comparison to healthy subjects. Adv Physiother 7 (1), 13–19.

Norris, C.M., 1995. Spinal stabilisation 4. Muscle imbalance and the low back. Physiotherapy 81 (3), 127–138.

Norris, C.M., 2004. Sports Injuries: Diagnosis and Management, third ed. Butterworth-Heinemann, Oxford.

Ombregt, L., Bisschop P., ter Veer, H.J., 2003. A System of Orthopaedic Medicine, second ed. Churchill Livingstone, Philadelphia.

Panjabi, M.M., 1992a. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. J Spinal Disord 5 (4), 383–389.

Panjabi M.M., 1992b. The stabilising system of the spine. Part II. Neutral zone and instability hypothesis. J Spinal Disord 5 (4), 390–397.

Panjabi, M.M., 2003. Clinical spinal instability and low back pain. J Electromyogr Kinesiol 13 (4), 371–379.

Peck, D., Buxton, D.F., Nitz, A., 1984. A comparison of spindle concentrations in large and small muscles acting in parallel combinations. J Morphol 180, 243–252.

Poppen, N.K., Walker, P.S., 1976. Normal and abnormal motion of the shoulder. J Bone Joint Surg Am 58, 195–201.

Powers, C.M., 2003. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: a theoretical perspective. J Orthop Sports Phys Ther 33, 639–646.

Prentice, W., 2004. Rehabilitation Techniques for Sports Medicine and Athletic Training, fourth ed. McGraw-Hill, New York.

Richardson, C.A., 1992. Muscle Imbalance: Principles of Treatment and Assessment. In: Proc New Zealand Soc Physiotherapists Challenges Conference, Christchurch.

Richardson, C.A., Snijders, C.J., Hides, J.A., et al., 2002. The relation between the transversus abdominis muscles, sacroiliac joint mechanics and low back pain. Spine 29 (4), 399–405.

Richardson, C.A., Hodges, P., Hides, J.A., 2004. Therapeutic Exercise for Spinal Segmental Stabilisation in Low Back Pain: Scientific Basis and Clinical Approach, second ed. Churchill Livingstone, Edinburgh.

Rosene, J.M., Fogarty, T.D., Mahaffey, B.L., 2001. Isokinetic hamstrings: Quadriceps ratios in intercollegiate athletes. J Athl Train 36 (4), 378–383.

Roth, J.K., Roth, R.S., Weintraub, J.R., et al., 2007. Cervicogenic headache caused by myofascial trigger points in the sternocleidomastoid: a case report. Cephalalgia 27 (4), 375–380.

Sahrmann, S.A., 1987. Posture and Movement Imbalance: Faulty Lumbar-Pelvic Alignment and Associated Musculoskeletal Pain Syndromes, Postgraduate Advances in Physical Therapy. Forum Medicum Inc., Berryville.

Sanghvi, S., 2008. Cardiac and Pulmonary Rehabilitation. In: Porter, S. (Ed.), Tidy's Physiotherapy, fourteenth ed. Churchill Livingstone, Edinburgh.

Savigny, P., Kuntze, S., Watson, P., et al., 2009. Low Back Pain: Early Management of Persistent Non-Specific Low Back Pain. National Collaborating Centre for Primary Care and Royal College of General Practitioners, London. (www.nice.org.uk)

Selye, H., 1951. General-Adaptation-Syndrome. Annu Rev Med 2, 327–342.

Simons, D.G., Travell, J.G., Simons, L.S., 1999. Travell and Simons' Myofascial Pain and Dysfunction: Upper Half of Body. Lippincott Williams & Wilkins, Baltimore.

Smith, T.O., Nichols, R., Harle, D., et al., 2009. Do the vastus medialis obliquus and vastus medialis longus really exist? A systematic review. Clin Anat 22, 183–199.

Standring, S. (Ed.), 2005. Gray's Anatomy: The Anatomical Basis for Clinical Practice, thirty-ninth ed. Churchill Livingstone, Edinburgh.

Tabary, J.C., Tabary, C., Tardieu, C., et al., 1972. Physiological and structural changes in the cat soleus muscle due to immobilization at different lengths by plaster casts. J Physiol 224, 231–244.

Tobin, S., Robinson, G., 2000. The effect of McConnell's vastus lateralis inhibition taping technique on vastus lateralis and vastus medialis obliquus activity. Physiotherapy 86 (4), 173–183.

Walsh, M., Woodhouse, L.J., Thomas, S.G., et al., 1998. Physical impairments and functional limitations: A comparison of individuals 1 year after total knee arthroplasty with control subjects. Phys Ther 78 (3), 248–258.

Watson, D.H., Trott, P.H., 1993. Cervical headache: an investigation of natural head posture and upper cervical flexor muscle performance. Cephalalgia 13, 272–284.

White SG, Sahrmann, SA, 1994. A movement system balance approach to management of musculoskeletal pain. In: Twomey, L.T., Taylor, J.R. (Eds), Physical Therapy of the Cervical and Thoracic Spine. Churchill Livingstone, New York.

Williams, P.E., Goldspink, G., 1978. Changes in sarcomere length and physiological properties in immobilized muscle. J Anat 127 (3), 459–468.

Williams, P.E., Goldspink, G., 1984. Connective tissue changes in immobilized muscle. J Anat 138, 343–350.

Chapter 15

Biomechanics

Jim Richards, Ambreen Chohan and Renuka Erande

INTRODUCTION

This chapter will take you through an introduction to clinical gait analysis, definitions and detailed descriptions of the movement and force patterns found during walking, and the mathematical basis of how joint movement, muscle forces and power may be calculated. More information about clinical biomechanics is available in the textbook *Biomechanics in Clinic and Research: An Interactive Teaching and Learning Course* (Richards 2008).

Clinical gait analysis

In 1953, Saunders and co-workers referred to the major determinants in normal gait and applied these to the assessment of pathological gait. Inman (1966, 1967) and Murray (1967) both published detailed analyses on the kinematics and conservation of energy during human locomotion, and these are resources still frequently referred to. Inman et al. (1981) later published *Human Walking*, a comprehensive textbook on human locomotion.

Brand and Crowninshield (1981) highlighted the distinction between the use of biomechanical techniques to 'diagnose' or 'evaluate' clinical problems. The authors stated: 'Evaluate, in contrast to diagnose, means to place a value on something. Many medical tests are of this variety and instead of distinguishing diseases, help determine the severity of the disease or evaluate one parameter of the disease. Biomechanical tests at present are of this variety'. Brand and Crowninshield also gave a guide of six requirements for tools used in patient evaluation:

1. The measured parameter(s) must correlate well with the patient's functional capacity
2. The measured parameter must not be directly observable and semi-quantifiable by the physician or therapist
3. The measured parameters must clearly distinguish between normal and abnormal
4. The measurement technique must not significantly alter the performance of the evaluated activity
5. The measurement must be accurate and reproducible
6. The results must be communicated in a form which is readily identifiable.

Brand and Crowninshield stated: 'It is clear to us that most methods of assessing gait do not meet all of these criteria. We believe that it is for this reason that they are not widely used.'

Advances in biomechanical assessment in the last 30 years have been considerable. The description of normal gait in terms of movement and forces about joints is now commonplace. The relationship between normal gait patterns and normal function is also well supported in both scientific articles and textbooks (Bruckner 1998; Rose and Gamble 2005; Perry 2010, Levine et al. 2012). This allows deviations in gait patterns to be studied in relation to changes in function in subjects with particular pathologies. It is possible for a clinician or physician to subjectively study gait, but the value and repeatability of this type of assessment is questionable owing to poor inter- and intra-tester reliability (Pomeroy et al. 2003). It is impossible for one individual to study simultaneously, by observation alone, the movement pattern of all the main joints involved during an activity like walking. Studying movement patterns using objective motion analysis allows information to be gathered simultaneously with known accuracy and reliability. In this way, changes in movement patterns owing to intervention by physical therapists and surgeons may be assessed unequivocally.

Most motion analysis systems now report on the joint kinematics of the recorded individual and also contain the mean for normal data on the same graph, allowing a direct comparison of the individual's movement pattern in relation to a predefined normal. Such information is also available in *Clinical Gait Analysis: Theory and Practice* (Kirtley 2005).

Patrick (1991) reviewed the use of movement analysis laboratory investigations in assisting decision-making for the physician and clinician. Patrick concluded that the reasons for the use of such facilities not being widespread were owing to:

- the time of analysis being considerable;
- bioengineers designing systems and presenting results for researchers and not clinicians;
- a lack of understanding by physicians and clinicians of applied mechanics and its relevance to assessment of treatment outcome.

A common argument against movement analysis laboratories has been cost. The cost of movement analysis equipment and its potential use in the clinical setting has been reported (Bell et al. 1996). Indeed, a broader question could be put to any clinical assessment or treatment that requires the use of technology. One example of this is the relative cost of radiography to movement analysis equipment, which, in comparison, is modest. Gage (1994) claimed that gait analysis costs are comparable with magnetic resonance imaging (MRI) or computed axial tomography (CAT) scans. Gage also stated that the use of movement analysis, as a detailed form of assessment, may have wider cost benefits and improve clinical services more than first realised.

Bell et al. (1995) highlighted the use of a holistic approach to motion analysis, which included muscle performance, joint range of motion, kinematic and kinetic parameters of gait. This holistic approach may be applied to many pathologies to give a detailed assessment of pathology and the subsequent effects of treatment.

Many of the techniques of collection and analysing human locomotion have been applied to clinical practice. This has led to a more detailed clinical assessment of therapeutic and surgical intervention, which is becoming increasingly important in the age of evidence-based practice.

KINEMATICS

The gait cycle

Kinematics is the study of the movement of the body and body segments with no reference to the forces which may be acting. For instance, during normal walking there is an obvious division in the length of time that the foot is in contact with the ground and the period when it is not. These are known as the 'stance phase' (approximately 60% of the gait cycle) and the 'swing phase' (approximately 40% of the gait cycle) respectively.

- The *stance phase* can be subdivided into: heel strike, foot flat, mid-stance, heel off and toe off.
- The *swing phase* can be subdivided into: early swing, mid-swing and late swing.

Spatial and temporal parameters of gait

The simplest way to look at the kinematics during walking is by studying foot positions (spatial parameters) and times (temporal parameters).

Spatial parameters

We can display spatial parameters of foot contact during gait as a series of footprints (Figure 15.1). These can also be defined as follows:

- *step length* – the distance between two consecutive heel strikes;
- *stride length* – the distance between two consecutive heel strikes by the same leg;
- *foot angle or angle of gait* – the angle of foot orientation away from the line of progression;
- *base width or base of gait* – the medial lateral distance between the centre of each heel during gait.

Temporal parameters

We can display temporal parameters of heel strike and toe off pictorially (Figure 15.1). These can also be defined as follows:

- *step time* – the time between two consecutive heel strikes;
- *stride time* – the time between two consecutive heel strikes by the same leg, one complete gait cycle;
- *single support* – the time over which the body is supported by only one leg;
- *double support* – the time over which the body is supported by both legs;
- *swing time* – the time taken for the leg to swing through while the body is in single support on the other leg;
- *total support* – the total time a foot is in contact with the ground during one complete gait cycle.

Two other parameters may easily be calculated using this information: *cadence* and *velocity*. The cadence is the number of steps taken in a given time, usually steps per minute.

- $\text{Cadence (steps/min)} = \dfrac{Number\ of\ steps}{Time\ (min)}$

Velocity may be calculated by:

- $\text{Velocity (m/s)} = \dfrac{step\ length\ (m) \times cadence\left(\dfrac{steps}{min}\right)}{60\ (number\ of\ seconds\ in\ one\ minute)}$

Symmetry can also be assessed by dividing the value of a parameter found for the left over that of the right:

- $\text{Symmetry of step length} = \dfrac{step\ length\ for\ left}{step\ length\ for\ right}$

These parameters, although simple, can be a very useful means of outcome assessment. It must be noted, however, that these may not always be appropriate for some more complex pathological gait patterns (Wall et al. 1987). For example, the features of *Parkinsonian gait* can include a reduction in stride length and velocity, and an increase

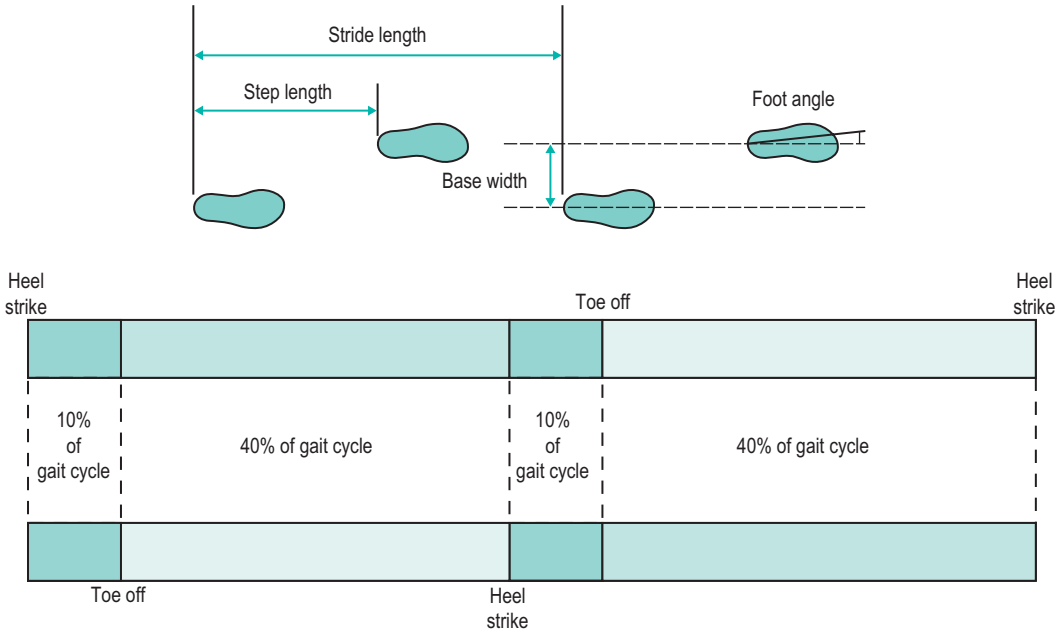


Figure 15.1 Spatial and temporal parameters.

in base width. However, this results in a characteristic *shuffling gait* which makes it hard to determine the events of heel strike and toe off. For further information on Parkinson's disease, please refer to Chapter 24.

ANALYSIS OF JOINT MOVEMENT DURING GAIT

Human walking allows a smooth and efficient progression of the body's centre of mass (Inman 1967). To achieve this there are a number of different movements of the joints in the lower limb. The correct functioning of the movement patterns of these joints allows a smooth and energy efficient progression of the body. The relationship between the movements of the joints of the lower limb is critical. If there is any deviation in the coordination of these patterns the energy cost of walking may increase and the shock absorption at impact and propulsion may not be as effective.

How to find segment angles and joint angles

Segment angles are defined as the angle of body segments away from the vertical axis (Figure 15.2). Segment angles can be calculated by knowing the co-ordinates of the proximal and distal ends of a body segment in a particular plane. The angle can then be found using trigonometry.

A *joint angle* is defined as the angle between the line of the proximal and distal segments of a joint. Figure 15.3 shows the hip, knee and ankle joint angles (α,β,γ). The

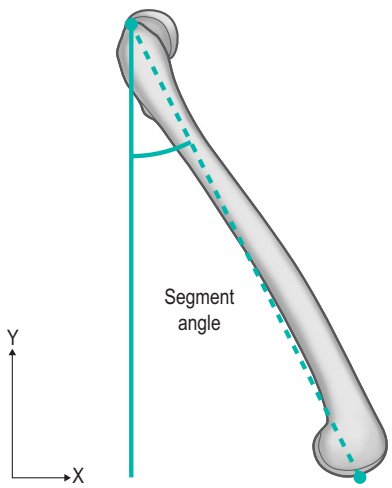


Figure 15.2 The segment angle.

ankle joint angle is defined by the foot with respect to a line 90 degrees to the tibia, with dorsiflexion defined as a positive angle and plantar flexion as a negative angle. The knee joint angle is defined by the long axis of the tibia with respect to the long axis of the femur, with full extension defined as zero degrees and movement into flexion being positive. The hip joint angle is defined by the long axis of the femur with respect to the pelvis, with flexion defined as positive and extension negative.

Joint angles which are commonly reported include: ankle plantar-dorsiflexion, foot rotation, knee flexion/extension, knee valgus/varus, knee rotation, hip flexion/

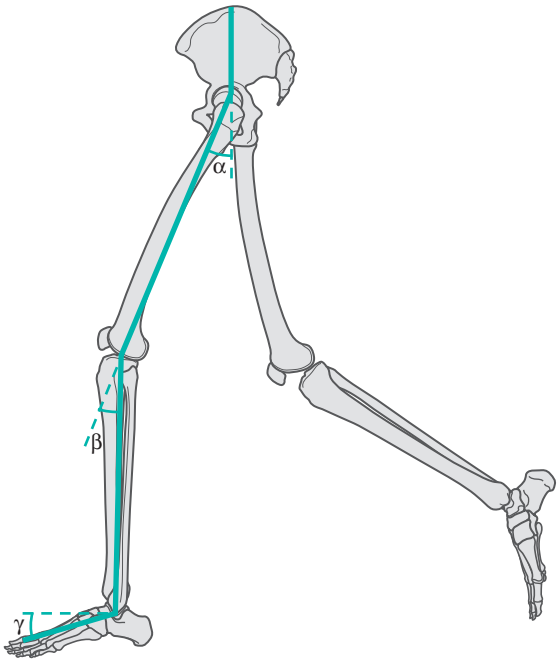


Figure 15.3 Joint angle definitions.

extension, hip abduction/adduction, hip rotation, pelvic tilt, pelvic obliquity and pelvic rotation. Although all these movement patterns are of interest, only major movement patterns of the lower limb are covered within this chapter.

Motion of the ankle joint

The movement of the ankle joint is of great importance as it allows shock absorption at heel strike, progression of the body forwards during the stance phase and is vital in the 'push off' or propulsive stage immediately before the toe leaves the ground. During the swing phase the motion of the ankle joint allows foot clearance, which can be lacking in some pathological gait patterns and is generally known as 'drop foot'.

The range of motion that occurs in walking varies between 20 degrees and 40 degrees, with an average value of 30 degrees. However, this does not tell us how the motion of the ankle varies throughout gait. During gait the ankle has four phases of motion (Figure 15.4).

Phase 1

At initial contact, or heel strike, the ankle joint is in a neutral position; it then plantar flexes to between 3 and 5 degrees until foot flat has been achieved. This is sometimes referred to as 'first rocker' or 'first segment', which refers to the foot pivoting about the heel or calcaneus. During this period the dorsiflexor muscles in the anterior

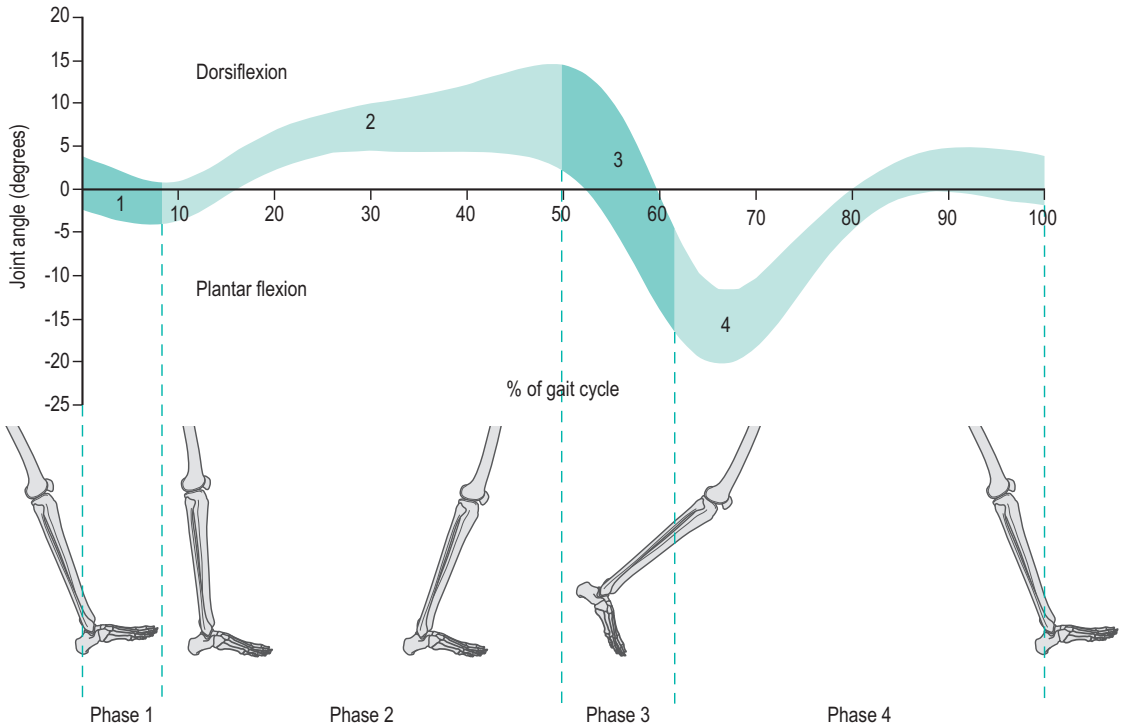


Figure 15.4 Ankle movement through the gait cycle.

compartment of the foot and ankle are acting eccentrically, controlling the plantar flexion of the foot. This gives the effect of a shock absorber and aids smooth weight acceptance to the lower limb.

Phase 2

At the position of foot flat the ankle then begins to dorsiflex. The foot becomes stationary and the tibia becomes the moving segment, with dorsiflexion reaching a maximum of 10 degrees as the tibia moves over the ankle joint. The time from foot flat to heel lift is referred to as 'second rocker' or 'second segment', which refers to the pivot of the motion now being at the ankle joint with the foot firmly planted on the ground. During this time the plantar flexor muscles are acting eccentrically to control the movement of the tibia forwards.

Phase 3

The heel then begins to lift at the beginning of double support, causing a rapid ankle plantar flexion reaching an average value of 20 degrees at the end of the stance phase at toe off. The ankle plantar flexes at a rate of 250 degrees per second. This rapid movement is associated with power production. During this propulsive phase of the gait cycle the plantar flexor muscles in the posterior compartment of the foot and ankle contract concentrically, pushing the foot into plantar flexion and propelling the body forwards. This is referred to as 'third rocker' or 'third segment' as the pivot point is now under the metatarsal heads.

Phase 4

During the swing phase the ankle rapidly dorsiflexes (150 degrees per second) to allow the clearance of the foot from the ground. A neutral position (0 degrees) is reached by mid-swing, which is maintained during the rest of the swing phase until the second heel strike. This is referred to as the 'fourth segment'. It has been recorded that there is sometimes a 3–5 degree dorsiflexion during the swing phase. During this phase the ankle dorsiflexors concentrically contract to provide foot clearance from the ground and prepare for the next foot strike.

Motion of the knee joint

During gait the knee joint moves in the sagittal, transverse and coronal planes. However, the majority of the motion of the knee joint is in the sagittal plane, which involves the flexion and extension of the knee joint. The flexion and extension of the knee joint is cyclic and varies between 0 and 70 degrees, although there is some variation in the exact amount of peak flexion occurring. These differences may be related to differences in walking speed, subject individuality and the landmarks selected to designate limb segment alignments. There are five phases (Figure 15.5).

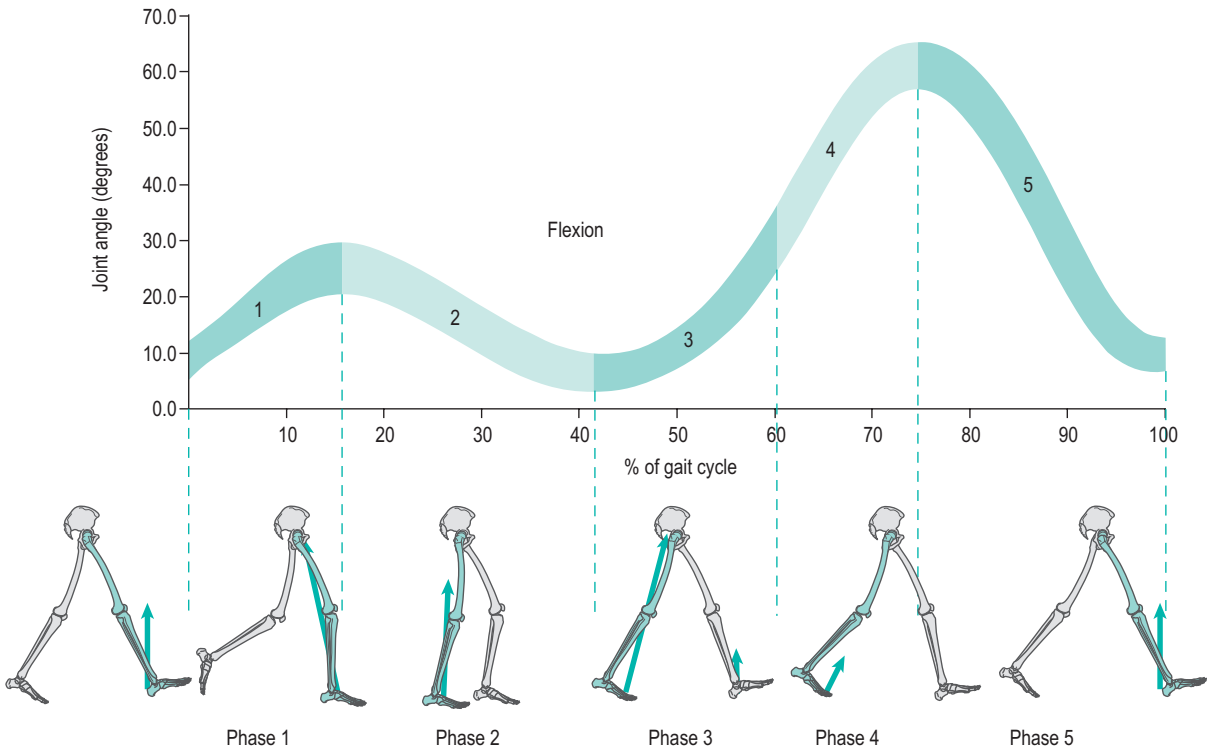


Figure 15.5 Knee movement through the gait cycle.

Heel strike

At heel strike, or initial contact, the knee should be flexed (Figure 15.5). However, people's knee posture can vary between slight hyperextension (–2 degrees) to 10 degrees of flexion, with a mean value of 5 degrees.

Phase 1

After the initial contact there is a flexion of the knee joint to about 20 degrees when the knee is flexed under maximum weight-bearing load. The knee joint flexes to absorb the loading at a rate of 150–200 degrees per second. This occurs at the same time the ankle joint plantar flexes, with a net effect to act as a shock absorber during the loading of the lower limb. During this time the knee extensors are acting eccentrically.

Phase 2

After this first peak of knee flexion the knee joint extends at a rate of 80–100 degrees per second to almost full extension. This is concerned with a smooth movement of the body over the stance limb.

Phase 3

The knee then begins its second flexion phase, which coincides with the heel lift. During this, the lower limb is in the propulsive phase of the gait cycle. The knee then continues to flex in preparation for swing phase, sometimes referred to as pre-swing.

Phase 4

Toe off occurs when the knee flexion is approximately 40 degrees, at which time the knee is flexing at a rate of 300–350 degrees per second. This flexion, coupled with the ankle dorsiflexion, allows the toe to clear the ground. During initial-to-mid-swing the knee continues to flex to a maximum of 65–70 degrees.

Phase 5

During late swing, the knee undergoes a rapid extension, 350–400 degrees per second to prepare for the second heel strike.

Motion of the hip joint in the sagittal plane

During walking the leg flexes forward at the hip joint to take a step and then extends until push off. This motion forms an arc starting at initial contact and finishing at toe off.

The motion of the hip joint is relatively simple (Figure 15.6). Maximum hip flexion occurs during terminal swing

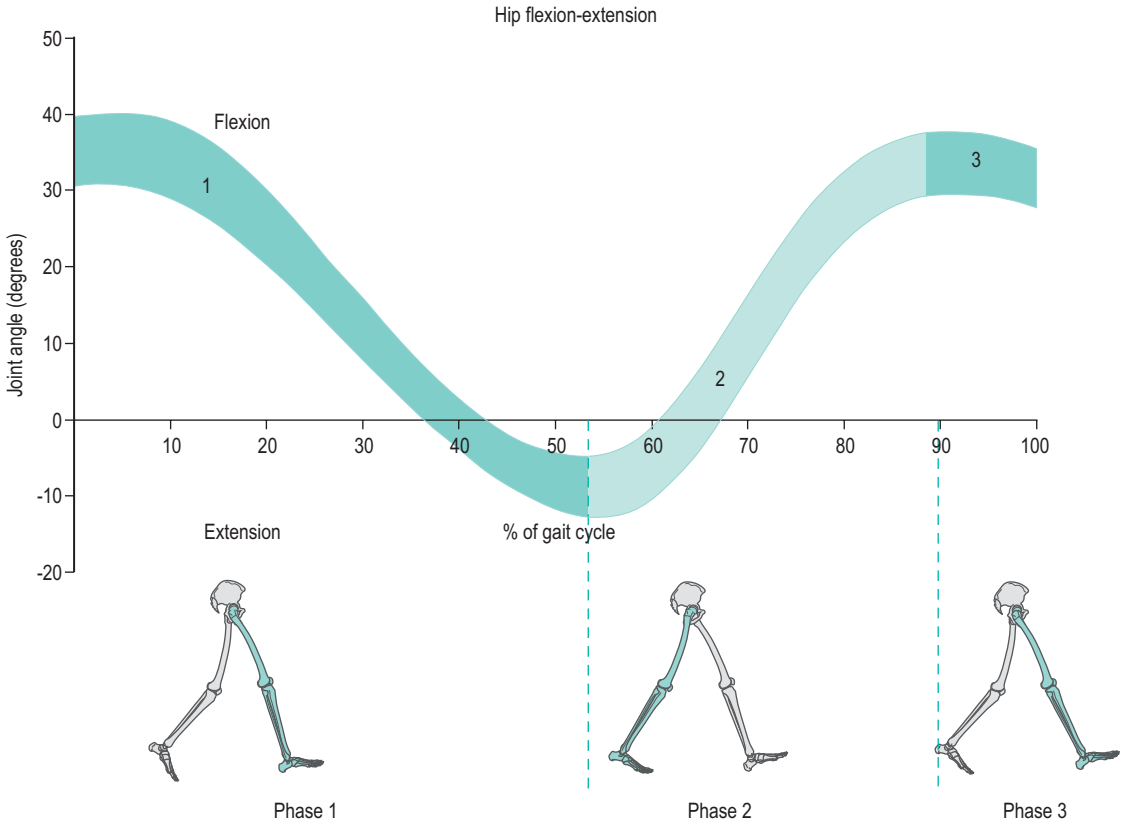


Figure 15.6 Hip movement through the gait cycle.

(phase 3), this is followed by a slight extension before initial contact, (phase 1). After initial contact (phase 1) the hip then extends as the body moves over the limb at a rate of 150 degrees per second. Maximum hip extension occurs just after opposite foot strike (phase 2), weight is then transferred to the forward limb and the trailing limb begins to flex at the hip. This is the pre-swing period. The toe leaves the ground at 60% of the gait cycle and the hip flexes rapidly at a rate of 200 degrees per second. This can be seen from the slope of the angle against time plot below, to progress the limb forward and prepare for the next initial contact (phase 1).

Motion of the pelvis in the coronal plane (pelvic obliquity)

During the early stance phase the contralateral (swing) side of the pelvis drops downward in the coronal plane (Figure 15.7). In order to achieve foot clearance the knee on the contralateral side undergoes rapid flexion. In normal gait the peak pelvic obliquity (drop down) occurs just after opposite toe off, which corresponds to the early stance phase on the weight-bearing limb.

Pelvic obliquity serves three purposes: to aid shock absorption, to allow limb length adjustments and to reduce the vertical excursions of the body (improving efficiency).

To illustrate these points we will consider the gait of an above-knee amputee. In above-knee amputees the pelvic obliquity does not always follow the normal pattern owing to loss of the normal knee joint control. A common strategy is that of hitching up the contralateral side of the pelvis to ensure foot clearance. In this way pelvic obliquity can be used to shorten the effective limb length when required. However, this increases energy expenditure as it increases the vertical excursion of the body.

Motion of the pelvis in the transverse plane (pelvic rotation)

During normal level walking the pelvis rotates about a vertical axis alternately to the left and to the right (Figure 15.8). This rotation is usually about four degrees on each

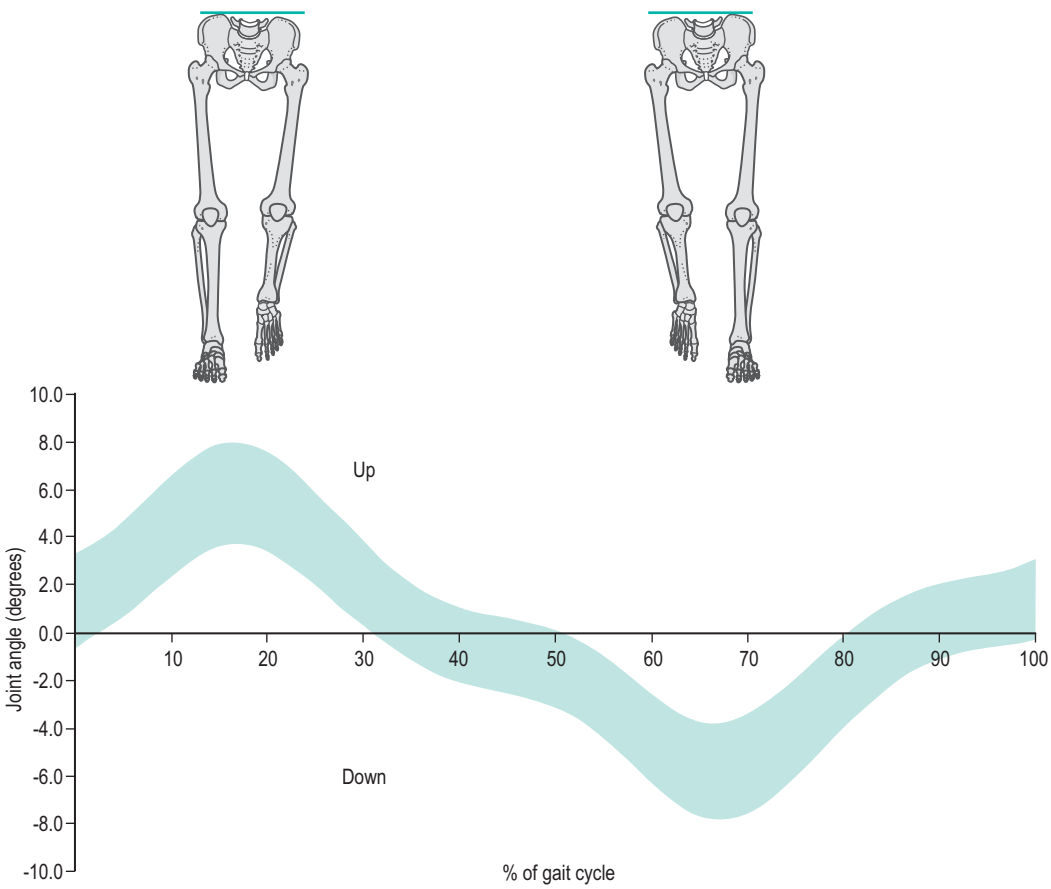


Figure 15.7 Pelvic obliquity.

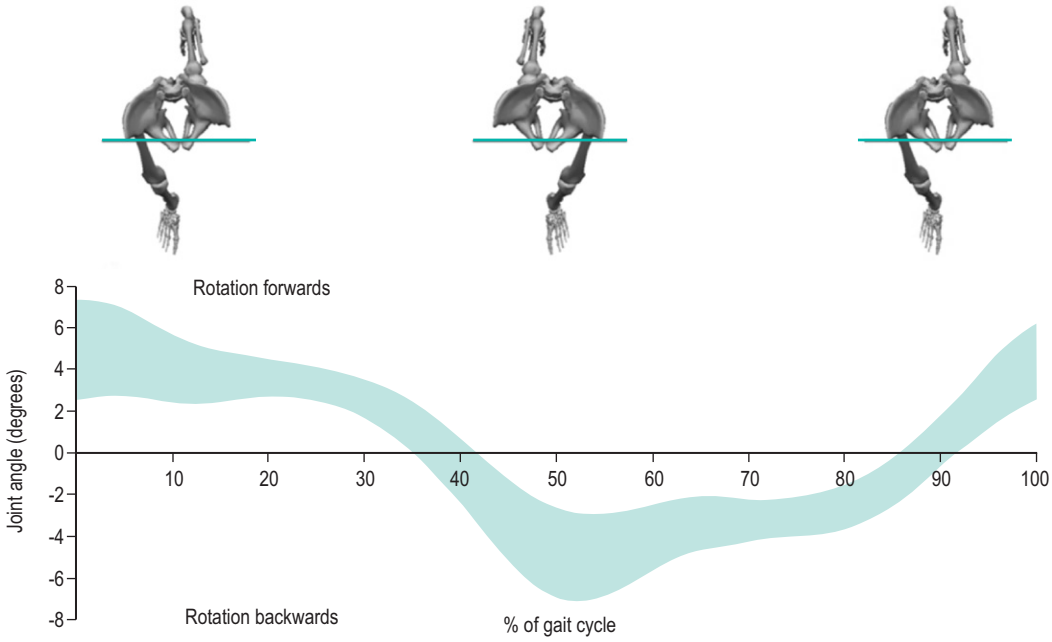


Figure 15.8 Pelvic rotation.

side of this central axis – the peak internal rotation occurring at foot strike and the maximal external rotation at opposite foot strike. This rotation effectively lengthens the limb by increasing the step length and prevents excessive drop of the body, making the walking pattern more efficient. Pelvic rotation also has the effect of smoothing the vertical excursion of the body and reducing the impact at foot strike.

HOW TO FIND LINEAR DISPLACEMENT, VELOCITY AND ACCELERATION

Linear displacement

Linear displacement refers to the movement of an object a particular distance in a particular direction. Displacement may be found by multiplying average velocity by time. The average velocity may be found by adding the initial and final velocities and dividing by two:

$$\text{average velocity} = \frac{(\textit{initial velocity} + \textit{final velocity})}{2}$$

Linear velocity

Linear velocity is the rate of change of displacement, i.e. the distance covered in a particular time. This is the speed of movement in any particular direction or anatomical plane:

$$\text{velocity} = \frac{\textit{change in displacement}}{\textit{time}}$$

Linear acceleration

Acceleration is the rate of change of velocity, i.e. the change in velocity over a given time:

$$\text{acceleration} = \text{change in velocity/time.}$$

KINEMATICS OF A REACHING TASK

The above equations can be used to examine the quality of movement of different tasks. We will consider linear control by evaluating what information may be gained from the study of the movement of the hand forwards during a reaching task, such as reaching to pick up a cup, in a subject who is pain- and pathology-free and a patient who has a painful unstable shoulder. The motion of the upper limb during reaching can be examined by studying the displacement, velocity and acceleration graphs. All of these are derived from the same displacement data; however, they all yield significantly different information which may be used to help us to describe functional aspects of the task. The process of finding velocity from displacement and acceleration from velocity is called *differentiation*.

Linear displacement of the hand during reaching with and without shoulder dysfunction

This graph is drawn from knowing how the linear position of the hand varies over time. Figure 15.9 shows the hand starting at a position zero and moving forwards in a reaching motion. The gradient of the curve indicates the velocity at which the hand is moving throughout the task. Figure 15.10a shows an individual who is pain- and pathology-free and Figure 15.10b shows an individual with a painful, unstable shoulder. Both show a similar pattern; however, the individual with the painful unstable shoulder appears to have a less smooth pattern of movement. We are, however, limited in the measurements we may take from linear displacement graphs; at best we can find the time taken to complete the task and the distance reached with very little information about the performance and quality of the movement during the task.

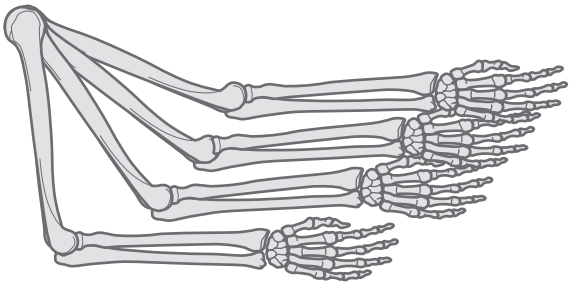


Figure 15.9 Motion of the upper limb during reaching.

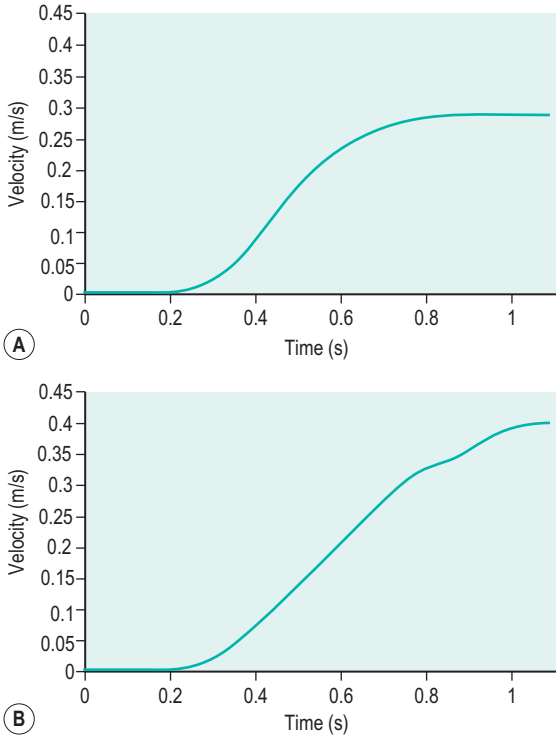


Figure 15.10 Displacement versus time of a reaching task: (a) pain- and pathology-free; (b) with a shoulder dysfunction.

Linear velocity of the hand during reaching with and without shoulder dysfunction

The velocity graph is found by measuring the change in the linear displacement over each successive time interval. The linear velocity graph for the hand of the individual who is pain- and pathology-free shows a bell-shaped curve (Figure 15.11a). Initially, the velocity of the hand is zero; the hand then accelerates to its maximum velocity at approximately the mid-point of the reaching movement. The hand then decelerates as it gets closer to its target; this takes slightly longer than the acceleration phase to ensure control and accuracy of hand positioning. The individual with a painful unstable shoulder (Figure 15.11b) shows a marked difference when considering the linear velocity graph. It shows a rapid acceleration then a continuously varying velocity which is followed by a decrease then an increase in velocity indicating either an unstable or painful part of the movement. The peak velocity may be measured from this graph indicating the level of performance of the task. The unsmooth nature of the pattern gives us a further insight to the control of the task but we cannot measure this directly from the linear velocity graph.

Linear acceleration of the hand during reaching with and without shoulder dysfunction

The acceleration graph is found by measuring the change in the linear velocity over each successive time interval. The individual who is pain- and pathology-free (Figure 15.12a) shows an initial acceleration peak early in the movement. The acceleration then decreases to zero as the hand reaches its maximum velocity. The hand then goes into a deceleration phase as it reaches its target. The peak deceleration is lower than the acceleration phase, and the deceleration phase lasts for a longer period of time, as shown with the velocity curve; again, this is to ensure controlled accuracy of positioning the hand at the target. The individual with a painful unstable shoulder (Figure 15.12b) shows a marked difference when considering the linear acceleration graph – it shows a rapidly changing graph indicating a lack of smooth controlled movement with no clear acceleration and deceleration period. This lack of smoothness tells us important information about

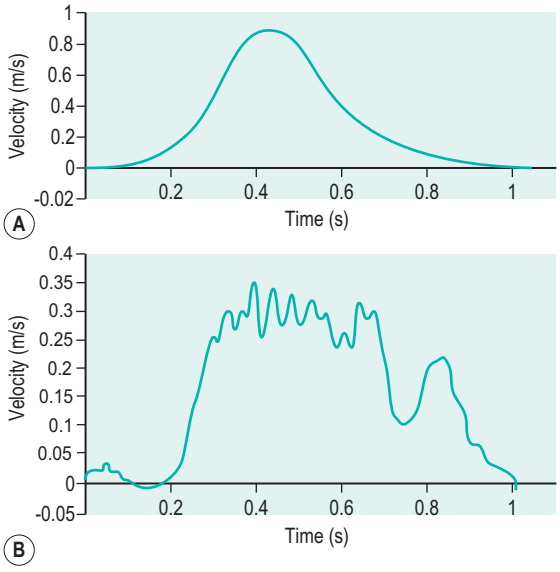


Figure 15.11 Velocity versus time of a reaching task: (a) pain- and pathology-free; (b) with shoulder dysfunction.

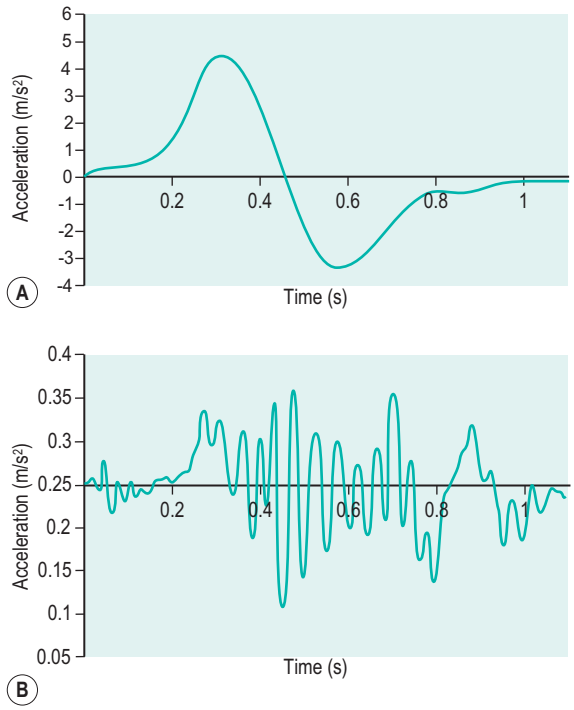


Figure 15.12 Acceleration versus time of a reaching task: (a) pain- and pathology-free; (b) with shoulder dysfunction.

a lack of control which could be owing to poor articulation at the shoulder or poor neuromotor control.

It is important to reiterate that the information presented for displacement, velocity and acceleration comes from the same original data from the cumulative displacement and time. It is also important to realise that each of these graphs tells us different information about the amount of movement, the performance of movement and the control of movement during the task. Movement control strategies may vary depending on various factors such as task, object, individual or cognitive constraints.

HOW TO FIND ANGULAR DISPLACEMENT, VELOCITY AND ACCELERATION

Angular displacement

Angular displacement is given the symbol (θ). This refers to the movement of an object through an angle. Angular displacement can be measured in two ways, either in degrees or in radians. There are 360 degrees in a full circle, or 2π radians. Pi (π) is the ratio of the circumference of a circle to its diameter (a ratio that is for all circles) and is very close to 3.1416, so 1 radian is about 57.3 degrees.

Angular velocity

Angular velocity is the rate of change of angular displacement or the rate at which an angle is covered in a particular time. This is referred to as the angular velocity and is given the symbol (ω). Angular velocity can be expressed in degrees per second or radians per second.

Angular acceleration

Angular acceleration is the rate of change of angular velocity and is given the symbol (α). As with linear acceleration, this relates to the muscles overcoming inertial forces to either start or stop movement, therefore muscle forces can either cause an angular acceleration or deceleration of a joint. Angular acceleration can be written in degree/s^2 or radians/s^2.

KINEMATICS OF THE KNEE DURING WALKING

As with the linear movement of the hand during reaching tasks we can use the angular equations of motion to examine the quality of movement of joints during different tasks. We will now consider the movement of the knee joint in a subject who is pain- and pathology-free and a patient who has medial compartment knee osteoarthritis.

Again, all of these graphs are derived from the same angular displacement data; however, they all yield different information to help us describe the functional performance of the knee.

Knee angular displacement of normal knee function and medial compartment knee osteoarthritis during walking

During normal walking the motion of the knee joint in the sagittal plane varies between 0 and 70 degrees (Figure 15.13a), although there is some variation in the exact amount of peak flexion occurring. At heel strike, or initial contact, the knee is flexed. After the initial contact there is a controlled increase in knee flexion to about 20 degrees when the knee is flexed under maximum weight-bearing load. During this time the knee extensors are acting eccentrically. After this first peak of knee flexion the knee joint extends this in relation to a smooth, eccentrically controlled movement of the body over the stance limb. The knee undergoes a rapid flexion in preparation for swing phase, sometimes referred to as pre-swing. Toe off marks the start of swing phase which allows the toe to clear the ground. During initial-to-mid-swing the knee continues to flex to a maximum of 65–70°. During late swing, the knee undergoes a rapid extension to prepare for the second heel strike.

In the patient with medial compartment knee osteoarthritis (Figure 15.13b), the amount of knee flexion attained during stance phase is significantly lower than that of normal, which could indicate poor eccentric control or a pain avoidance mechanism. The knee then re-extends but not to the same amount as normal – again indicating pain avoidance or reduced control. Swing phase appears to follow a normal pattern of movement but with a reduced amount of knee flexion at mid-swing.

The comparison of the angle against time graphs allows us to identify the positions of the joint at different stages during walking. From this we can examine the range of movement during the different parts of the task; however, we cannot take any direct measures of performance and control of movement from these graphs.

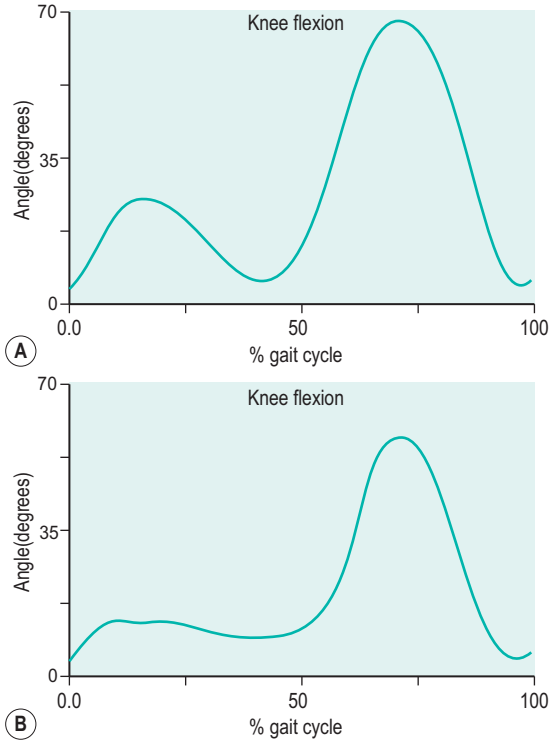


Figure 15.13 Knee angular displacement during walking. (a) An adult who is pain- and pathology-free. (b) A patient with medial compartment knee osteoarthritis.

Knee angular velocity of normal knee function and a patient with medial compartment knee osteoarthritis during walking

The velocity graph is found by measuring the change in the angular displacement over each successive time interval, allowing us to show the speed of movement into flexion or extension during walking. This tells us more about how the movement is achieved. A flexing angular velocity is defined as being positive and extension angular velocity as negative. Such graphs have been used to determine the performance and control of joints, and have been used to determine functional deficits in different pathologies.

During normal walking (Figure 15.14a) the knee flexes to approximately 200 degrees per second during loading, which gives a measure of the eccentric control of the knee during loading. The knee then extends at a rate of approximately 100 degrees per second, which shows a smooth, controlled movement over the stance limb as the body moves forwards. During swing phase the knee flexes and extends with an angular velocity of approximately 400 degrees per second.

In the patient with medial compartment knee osteoarthritis (Figure 15.14b) the knee flexes with a reduced angular velocity, therefore the eccentric speed and the control of the knee during loading is reduced. The knee extension velocity is markedly reduced indicating a substantial deficit in the control, or pain avoidance when moving over the stance limb. Interestingly, during swing phase the control of the knee flexion extension appears close to that of normal suggesting that the knee can move freely when not under load.

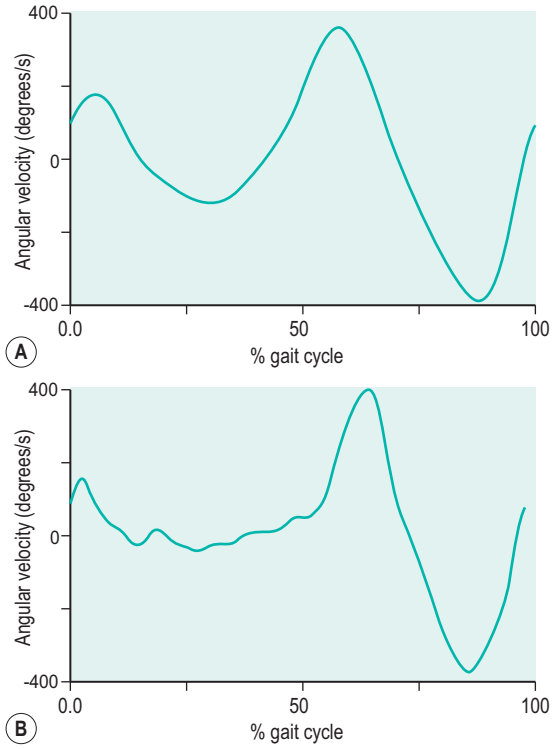


Figure 15.14 Knee angular velocity during walking. (a) An adult who is pain- and pathology-free. (b) A patient with medial compartment knee osteoarthritis.

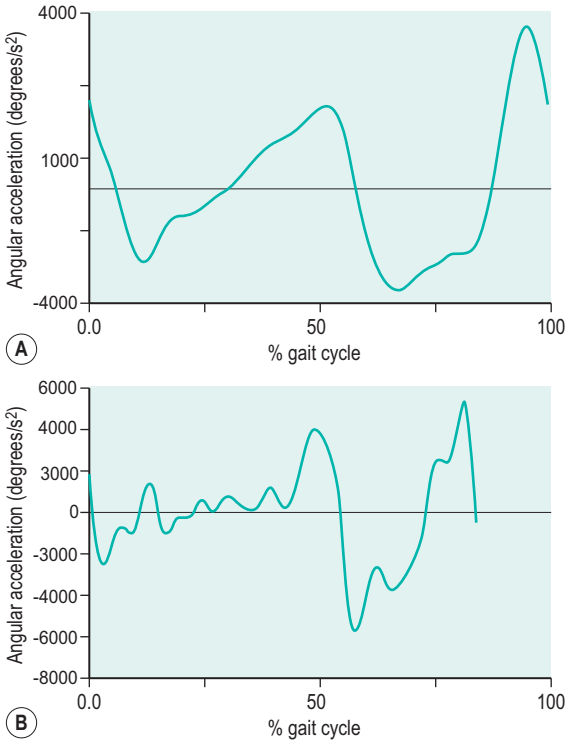


Figure 15.15 Knee angular acceleration during walking (a) An adult who is pain- and pathology-free. (b) A patient with medial compartment knee osteoarthritis.

The comparison of the angular velocity allows us to take measurements that relate to the eccentric and concentric control of the joint. Therefore, we are not just looking at what joint angle is attained at different points during walking but how the joint is controlled between these points.

Knee angular acceleration of normal knee function and a patient with medial compartment knee osteoarthritis during walking

The acceleration graph is found by measuring the change in the angular velocity over each successive time interval, which allows us to determine the smoothness and control of movement into flexion or extension during walking. This also yields important information necessary for the calculation of joint moments, particularly during swing phase. Acceleration into flexion is referred to as positive and a deceleration is negative.

During normal walking (Figure 15.15a) the knee shows a steady pattern of acceleration and deceleration during stance and swing phase. The absence of any rapid changes in acceleration shows that the movement is both smooth and controlled.

In the patient with medial compartment knee osteoarthritis (Figure 15.15b) the knee shows several points with rapid changes in acceleration during mid-stance, in particular, as the body moves over the stance limb. This reflects a deficit in the smoothness and control during this period. It is also interesting to note a deficit in smooth movement during swing phase, which had previously gone undetected, indicating that this movement is not as smooth as we previously thought.

METHODS OF MOVEMENT ANALYSIS

Methods used for movement analysis vary enormously, and are widely dependent on clinical conditions, skills, available facilities and the purpose of the assessment. We will now consider several methods that can be used to collect objective data for a variety of different pathological conditions.

Common clinical tools

Often in clinical settings, joint angles are assessed simply using a hand-held goniometer. There are several types of goniometer, all giving a crude, but useful, measure of angles and range of motion. Clinically, the goniometer allows a quick and useful assessment of static angles. However, these devices are of little use when measuring angles dynamically during different movement tasks. Further information on clinical measurements using instrumentation such as the goniometer are available in the handbook *A Physiotherapist's Guide to Clinical Measurement* (Fox and Day 2009).

Spatial parameters can be measured in a variety of simple ways, including putting ink pads on the soles of the subject's shoes and walking on paper (Rafferty and Bell 1995), and using marker pens attached to shoes (Gerny 1983). Although very cheap, these systems can require awkward and time-consuming analysis. Temporal parameters can be measured by timing how long it takes an individual to walk a set distance and counting the number of steps it took to cover that distance. However, this will only give average velocity and cadence, and will give no value to the symmetry of these parameters. This technique is extremely susceptible to human error.

Walk mat systems

In the last two decades of the twentieth century advances in computer technology led to the development of a number of instrumented walk mat systems. These allow fast collection of temporal and spatial gait data. Using a computer also allows easier, less time-consuming analysis. These systems include apparatus for step length measurement (Durie and Farley 1980), a system for monitoring the position and time of foot contact during walking (Arenson et al. 1983), using a measuring walkway (Hirokawa and Matsumura 1987), a microcomputer-based system (Crouse et al. 1987) and a walk mat system (Al-Majali et al. 1993). Walk mat systems that do not require any modifications to the footwear are now commercially available. These offer far less interference with the gait cycle. One such system is the GAITRite™ system, which uses pressure sensor arrays to determine foot position (Figure 15.16).

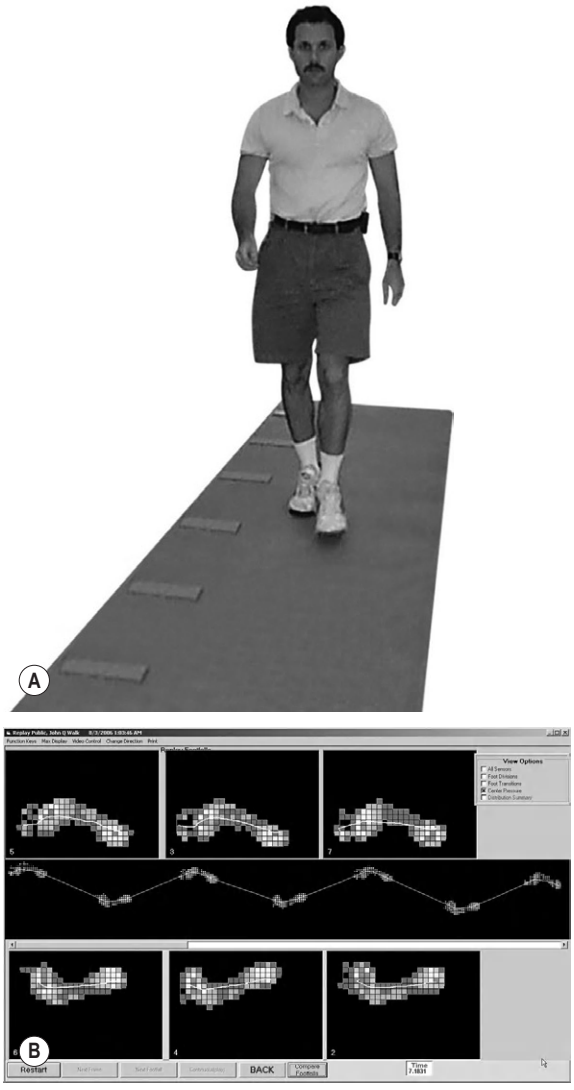


Figure 15.16 GAITRite™ system and typical output from GAITRite™.

Movement analysis systems

In the late nineteenth century the first motion picture cameras recorded patterns of locomotion in both humans and animals. In 1877, Muybridge demonstrated, using photographs, that when a horse is moving at a fast trot there is a moment when all of the animal's hooves are off the ground and in 1887 published *Animal Locomotion*. Muybridge later used 24 cameras to study the movement patterns of a running man and in 1901 published *The Human Figure in Motion*. Marey, a French physiologist, used a 'photographic rifle' to photograph movement of animals in 1873, and in 1882 and 1885 to record displacements in human gait to produce the first stick figure of a runner.

In the second half of the twentieth century many systems capable of automated and semi-automated computer-aided motion analysis were developed. One of the first systems to become commercially available was the Ariel Performance Analysis System, which required the operator to manually identify the location of joint centres or passive markers placed on the body used for each frame. Since then, the problems of automatic marker

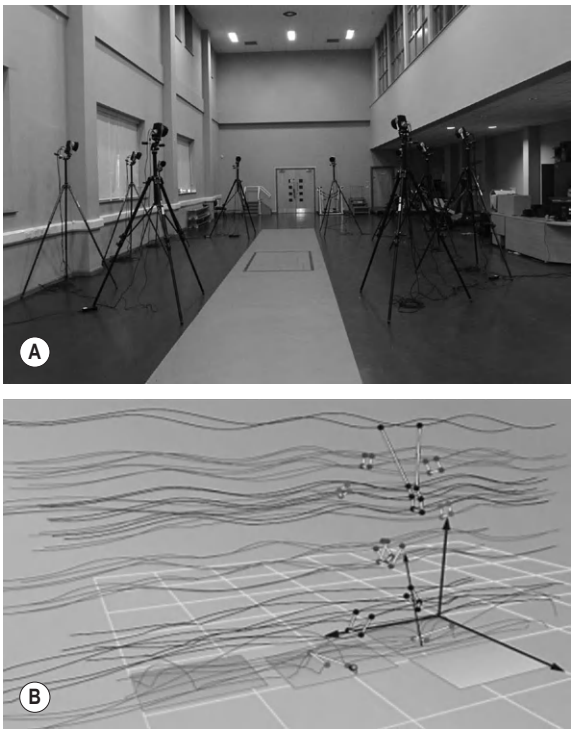


Figure 15.17 (a) UCLan's Movement Analysis Laboratory in the Allied Health Research Unit. (b) Automatic digitising using Qualisys Track Manager (QTM).

identification have been at the forefront of the development of computer-aided motion analysis. In 1974, SELSPOT became commercially available, which allowed automatic tracking of active light-emitting diode (LED) markers; later Optotrak™ and CODA™ used a similar technique. VICON, a camera based system, became commercially available in 1982. Since then, many systems based on television-camera technology have been developed, including the Motion Analysis Corporation system, Elite, and Oqus by Qualisys™ (Figure 15.17).

More recent advances in movement analysis have seen the development of three-dimensional motion capture suits, such as the XSens MVN™ system. This system is a camera-free motion system with a full body configuration of inertial motion trackers. Such configurations allow increased flexibility as they do not require a laboratory for data collection.

UNDERSTANDING FORCES

Forces

Forces make things move, stop things moving or make things change shape. They can either push or pull. Force is a *vector*, which simply means it has both direction and magnitude. All forces thus have two characteristics, magnitude and direction, and both need to be stated in order to describe the force fully. A good place to start is with the laws formulated by Sir Isaac Newton. In 1687 Newton published three simple laws, which together enshrine the fundamental principles of mechanics.

Newton's first law

If an object is at rest it will stay at rest. If it is moving with a constant speed in a straight line it will continue to do so, as long as no external force acts on it. In other words, if an object is not experiencing the action of an external force it will either keep moving or not move at all (Figure 15.18). This law expresses the concept of *inertia*. The inertia of a body can be described as being its reluctance to start moving or stop moving once it has started.

Why not perpetual motion?

Why does a car slow down when rolling on a flat road? The answer is that there are a number of external forces that need to be considered, such as wind resistance and friction in the bearings of the wheels and axle. This means we have to be careful to consider *all* the forces that are acting on an object in order to find out how it is going to move.

Newton's second law

The rate of change of velocity is directly proportional to the applied external force acting on the body and takes place in the direction of the force (Figure 15.19). Therefore, forces can either cause acceleration or deceleration of an object. *Acceleration* is usually defined as being positive and *deceleration* as being negative.

If F is the applied force in Newton (N), m is the mass of the body (kg) and a is the acceleration of the body (m/s^2), then $F = ma$. Therefore, 1 N is that force which produces an acceleration of 1 m/s^2 when it acts on a mass of 1 kg.

Key point

In the SI system of units, forces are measured in Newtons (N).

Newton's third law

If the box shown in Figure 15.20 exerts a force on the table top (*action*), then the table will exert an equal and

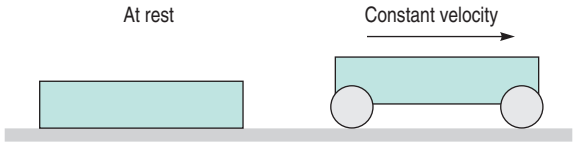


Figure 15.18 Newton's first law.

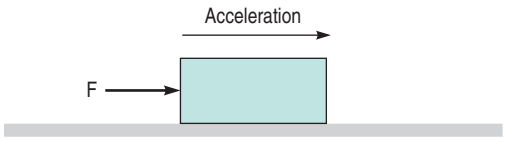


Figure 15.19 Newton's second law.

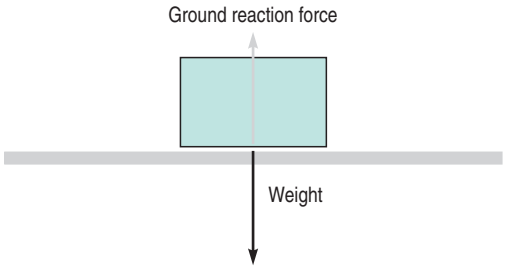


Figure 15.20 Newton's third law.

opposite force on the box (*reaction*). This does not mean the forces cancel each other out as they act on two different objects.

The action of a force on the ground receives an equal and opposite reaction force. This is known as a *ground reaction force* (GRF).

MASS AND WEIGHT

Mass

Mass is the amount of matter an object contains. This will not change unless the physical properties of the object are changed, wherever the object is moved.

Weight

Weight is a force. This depends on both the mass of the object and the acceleration acting on it. Weight is often interpreted as being the force acting beneath our feet, i.e. scales measure this force, although they never use the correct units (they should be Newton).

Therefore, a good way to lose weight is to stand in a lift and press the down button. You will lose weight (the ground reaction force will reduce) as the lift accelerates downwards. Unfortunately, when the lift comes to a stop you will gain weight again as the lift decelerates downwards.

Another example of the difference between mass and weight is to consider astronauts. When they are in space they are weightless, but this does not mean they have gone on an amazing diet, only that there is no acceleration acting on them. Therefore, weight-watchers should really be called mass-watchers.

Acceleration owing to gravity

Wherever you are on Earth there is acceleration owing to gravity acting on you. This does vary a small amount but the value is generally close to 9.81 m/s^2. For the purposes of rough calculations this is often rounded up to 10 m/s^2. However, to get the best possible accuracy 9.81 m/s^2 should be used.

Static equilibrium

The concept of static equilibrium is of great importance in biomechanics as it allows us to calculate forces that are unknown.

Newton's first law tells us that there is no resultant force acting if the body is at rest, i.e. the forces balance. Therefore, if an object is at rest, the sum of the forces on the object, in any direction, must be zero. So, when we resolve in a horizontal and vertical direction the resultant force must be zero.

Free-body analysis

Free-body analysis is a technique of looking at and simplifying a problem. The example below considers someone pulling a box of weight 40 N along the ground with a piece of string with a force of 10 N at an angle of 30 degrees to the horizontal (Figure 15.21a). We now break down what force must be acting on the box and form a simplified picture of just the box. The forces acting are the tension in the string, the frictional force, the weight of the box and the ground reaction force (Figure 15.21b).

Worked example

With reference to Figure 15.22, find the horizontal acceleration and the ground reaction force.

Horizontal forces
Horizontal force in string = 10 cos 30° = 8.66
Therefore 8.66 – 2.66 = mass × acceleration
6 = 4 × a
1.5 m/s = a.

Vertical force
Vertical force in string = 10 sin 30 = 5
Ground Reaction Force + 5 – 40 = 0
Ground Reaction Force = 40 – 5 = 35 N.

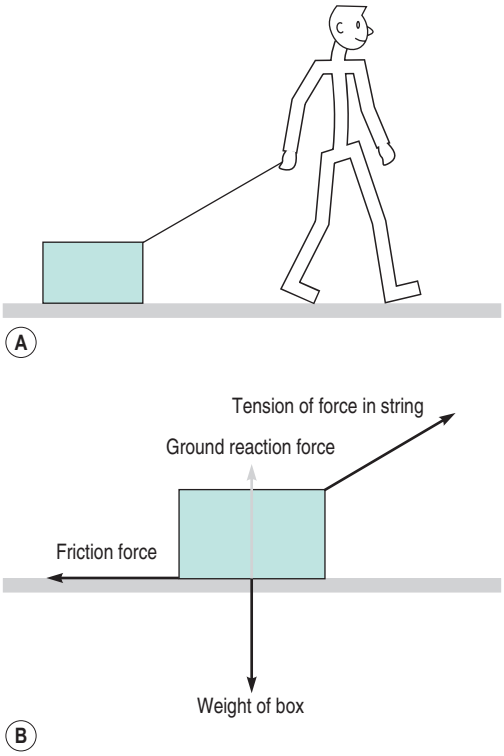


Figure 15.21 Free-body analysis.

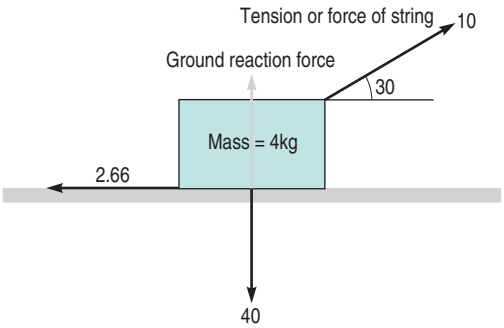


Figure 15.22 Worked example of Newton's laws.

HOW FORCES ACT ON THE BODY

Ground reaction forces

A ground reaction force is the force that acts on a body as a result of the body resting on the ground or hitting the ground. The study of forces is often referred to as kinetics.

If someone stands on a floor without moving, the person is exerting a force (the person's weight) on the floor, but the floor exerts an equal and opposite reaction force on the person. That is an example of the simplest ground reaction force, but it never happens as easily as that with human balancing because of sway.

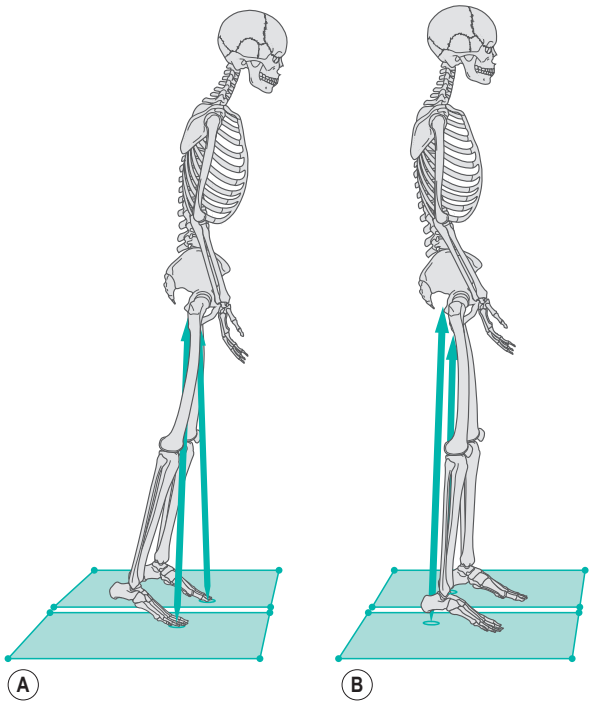


Figure 15.23 Postural sway in the anterior-posterior direction.

When standing we have a natural postural sway backwards and forwards (anterior posterior, Figure 15.23) and from side to side (medial lateral, Figure 15.24). As we sway there are horizontal forces acting, as well as the vertical force (Figure 15.25). The *centre of pressure* (CoP) is the point at which the force is acting beneath the feet. During postural sway the centre of pressure moves forwards, backwards and side to side between the two feet (Figure 15.26).

Ground reaction forces during the gait cycle

Vertical force component

A ground reaction force is made up of three forces acting in three directions at the centre of pressure: vertical, anterior–posterior and mediolateral. The vertical component of the ground reaction force can be split into four sections (refer to Figure 15.27).

Heel strike to first peak

Heel strike to first peak is where the foot strikes the ground and the body decelerates downwards, and transfers the loading from the back foot to the front foot during initial

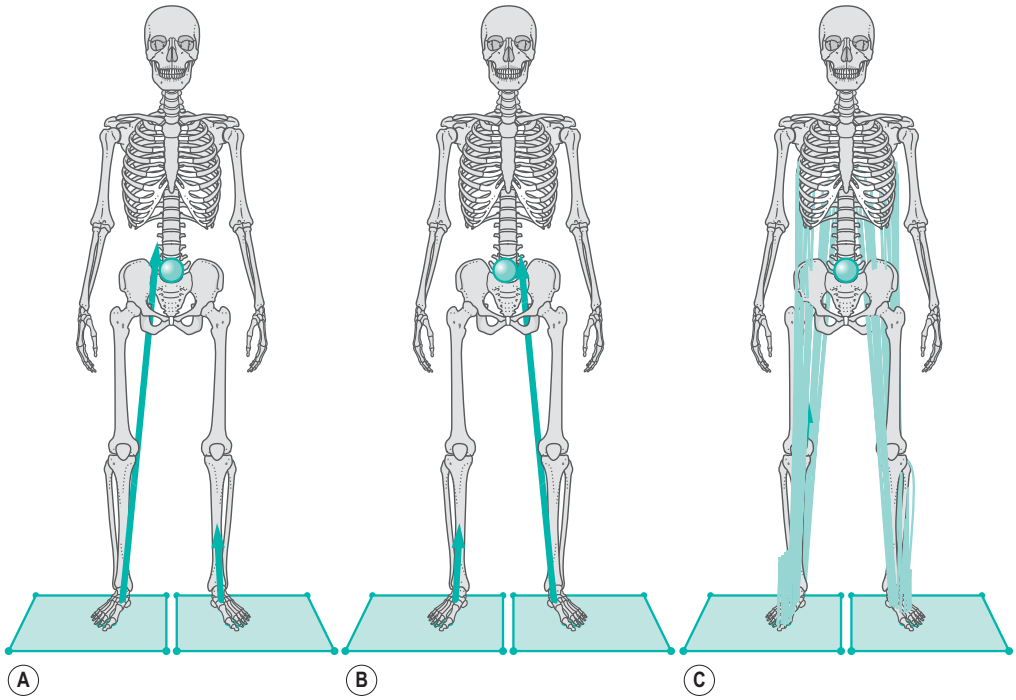


Figure 15.24 Postural sway in the medial lateral direction.

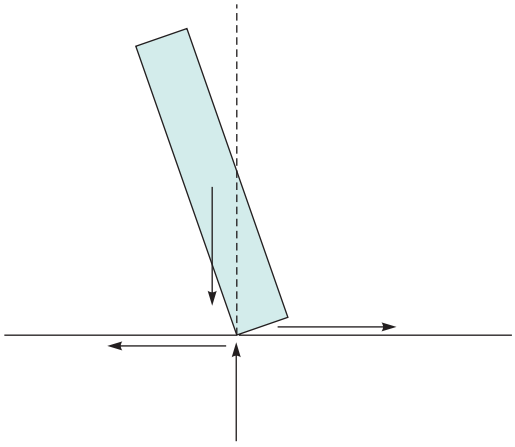


Figure 15.25 Postural sway during balance.

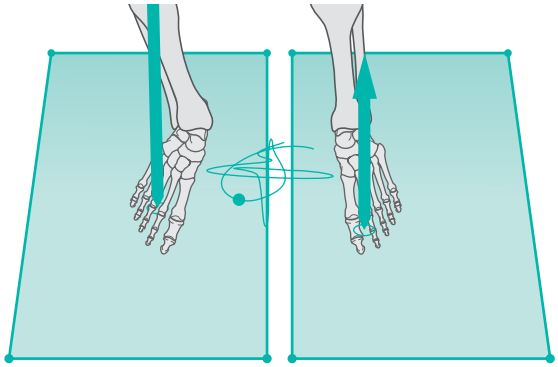


Figure 15.26 Movement of the centre of pressure during postural sway.

double support. The first peak should be in the order of 1.2 times the person's bodyweight.

First peak to trough

Here, in first peak to trough, the knee extends, raising the body. As the body approaches its highest point it is slowing down (decelerating the body) in its upward motion. This reduces the vertical ground reaction force. This has the same effect as going over a hump-backed bridge in a car: as you reach the top of the hump you feel very light because the contact force between you and the seat is reduced. The trough should be in the order of 0.7 times the person's bodyweight.

Trough to second peak

During trough to second peak the centre of mass now falls as the heel lifts and the foot is pushed down and back into the ground by the action of muscles in the posterior

compartment of the ankle joint. Both the deceleration downward and propulsion from the foot–ankle complex cause the second peak. The second peak should be in the order of 1.2 times the person's bodyweight.

Second peak to toe off

During second peak to toe off the foot is unloaded as the load is transferred to the opposite foot.

Anterior–posterior force component

The anterior–posterior component may also be split into four sections (Figure 15.28).

Clawback

Clawback is an initial anterior force that is not always present during walking. This is caused by the swinging limb hitting the ground with a backwards velocity thus causing an anterior reaction force as it decelerates. Clawback is often exaggerated during marching as the swing limb is driven back to meet the ground.

Heel strike to posterior peak

After the initial clawback (if present) the heel is in contact with the ground and the body decelerates, causing a posterior shear force. Imagine you are walking on a thick carpet, loading your front foot, and suddenly you are transported to an ice rink – your leg would slide forwards because the frictional force between the ice and your foot is very low, whereas the carpet can provide a much larger posterior reaction force that stops your leg from slipping forwards. The posterior peak should be in the order of 0.2 times the person's bodyweight.

Posterior peak to crossover

The posterior component reduces as the body begins to move over the stance limb, reducing the horizontal component of the resultant ground reaction force.

Crossover to anterior peak

During crossover to anterior peak the heel lifts and the foot is pushed down and back into the ground by the action of muscles in the posterior compartment of the ankle joint. This has the effect of producing an anterior component of the ground reaction force, which propels the body forwards. The anterior peak should be in the order of 0.2 times the person's bodyweight.

Anterior peak to toe off

Anterior peak to toe off is the period of terminal double support where the force is being transferred to the front foot. The anterior force therefore reduces.

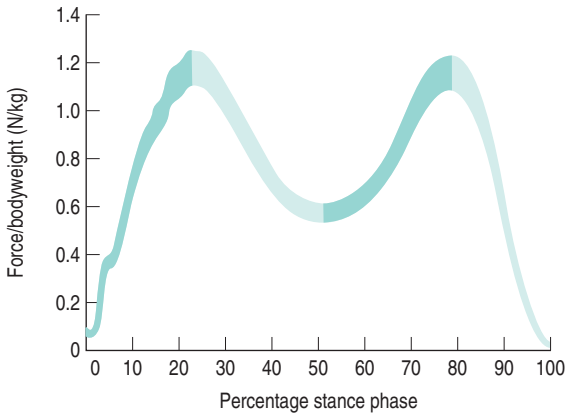


Figure 15.27 Vertical force during normal walking.

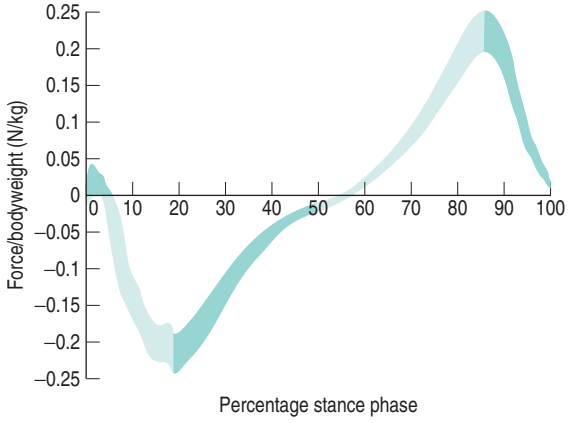


Figure 15.28 Force in the anterior–posterior direction during normal walking.

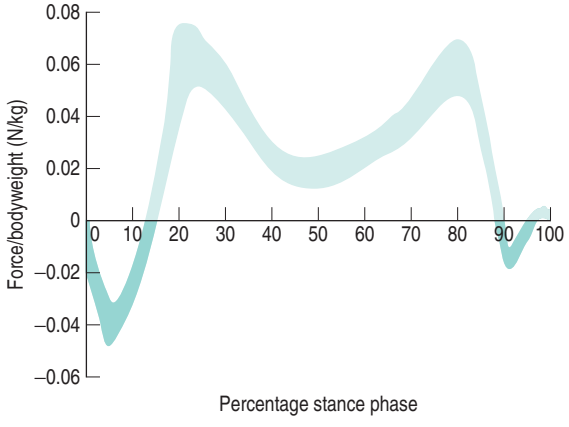


Figure 15.29 Force in the mediolateral direction during normal walking.

Mediolateral force component

The mediolateral component may be split into two main sections (Figure 15.29). Initially, at heel strike, there is a lateral thrust during loading; during this time the foot is working as a mobile adaptor. After the initial loading, the forces push in a medial direction as the body moves over the stance limb. Small lateral forces are often seen during the final push-off stage.

The mediolateral forces are the most variable of the three components and can easily be affected by footwear and foot orthotics. Normal maximum medial force is between 0.05 and 0.1 times the person's bodyweight. The maximum lateral force should be less than the maximum medial force.

How to study the measurements taken from these graphs

For each of these measurements the percentage difference can be studied between the left and right sides, and between the subject tested and non-pathological data. This will not only identify what differences are present in the walking patterns, but also how big the differences are.

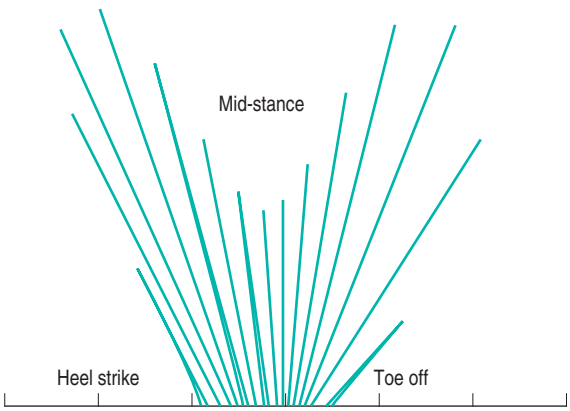


Figure 15.30 Pedotti diagram during normal walking.

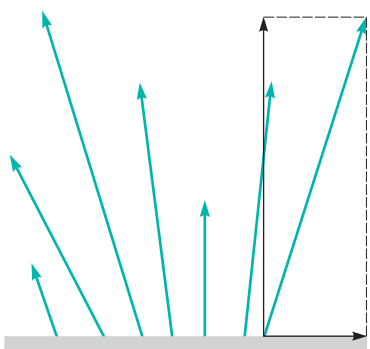


Figure 15.31 Vertical, horizontal and resultant ground reaction forces heel off.

Pedotti diagrams

The interaction of the vertical and anterior–posterior forces described above may be shown with a Pedotti diagram. This shows the magnitude of the resultant ground reaction force. Pedotti diagrams rely on the information provided by force platforms. To construct a Pedotti diagram we need to know the vertical and horizontal forces and the positions of the forces beneath the foot in the plane of interest for each moment in time.

As a subject walks, the forces during the stance phase move forward from under the heel to the toe, so the position of the force moves from posterior to anterior. As we have seen in the previous section, the vertical and horizontal ground reaction forces are continually changing during the stance phase, so changing the direction and magnitude of the resultant ground reaction force (Figure 15.30).

Figure 15.31 shows the vertical, horizontal and resultant ground reaction force components being drawn at heel off. The centre of pressure has moved forwards from the heel to the forefoot and the new vertical, horizontal and resultant ground reaction force components are drawn from the new position. This process is repeated throughout the stance phase, producing a butterfly-like diagram.

Test yourself

Use the data in Table 15.1 to plot a Pedotti diagram on graph paper. You should get something similar to that shown in Figure 15.32.

Table 15.1 Data for the self-assessment task

Position (m)	Anterior–posterior force (N)	Vertical force (N)
0	–8	66
0.04	–155	678
0.08	–180	878
0.12	–54	622
0.16	–13	457
0.2	26	564
0.24	106	889
0.28	180	626
0.32	10	16

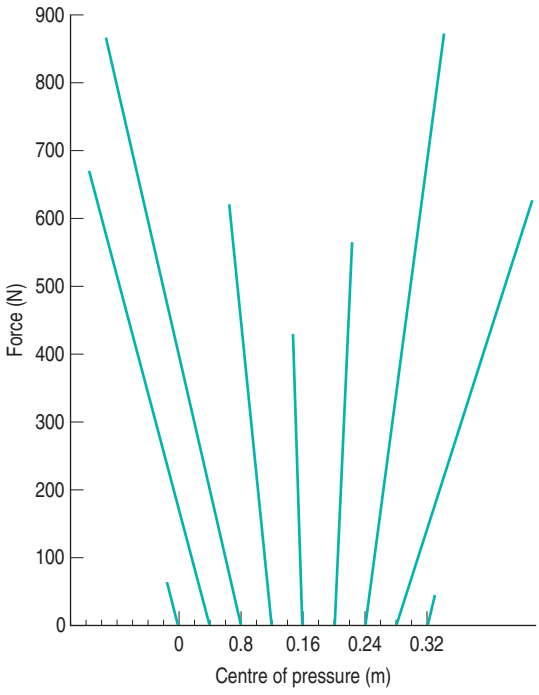


Figure 15.32 Graph from the 'Test yourself' task.

METHODS OF FORCE ANALYSIS

Video vector generators

The video vector generator is a piece of equipment that combines the information from a force platform with a video image. The resultant force can be superimposed on top of the video information, giving a picture of the action of the ground reaction forces (Figure 15.33). This uses the same information as displayed in the Pedotti diagram, but also allows the action of the forces to be seen with respect to the joints of the lower limb giving a visualisation of the joint moments. This information may be used to identify pathologies and monitor gross changes due to treatment. A moment (M) is defined as the magnitude of force F (how hard you push) multiplied by the perpendicular distance d of the force away from the pivot (Figure 15.34).

Force platforms

Force platforms are devices that measure and record ground reaction forces and their point of application at the centre of pressure (Figure 15.35).

To find the total force acting in an anterior–posterior direction, for example, all the anterior–posterior forces measured by the load cells will be added to give the total force in that direction.

Force platforms are considered as a basic, but fundamentally important, tool for gait analysis. The first force measurements date back as far as the late nineteenth century, when Marey used a wooden frame on rubber supports. Elftman (1938) used a similar method with a platform on springs. However, it was not until the advancement of computers and electronic technology that the readings could be accurately measured. In 1965, Peterson and co-workers developed one of the first strain-gauge force platforms. A plethora of publications now exists on the applications of such devices in both clinical research and sports.

Since 1965, forces platforms have undergone considerable development by three internationally accepted manufacturers: Kistler Instruments, AMTI and the Bertec Corporation. Advances have been in the form of making the platforms more accurate, increasing sensitivity and improving portability (Figure 15.36).

Pressure systems

Pressure systems are subtly different to force platforms as they are only able to measure vertical forces. However, pressure systems are able to measure the centre of pressure and the pressure patterns beneath the foot (Figure 15.37), which may be more clinically relevant for some patient groups e.g. diabetics with a risk of foot ulceration. Pressure systems use an array of load sensors, which usually range from 1 to 4 sensors per cm^2 and vary in size from approximately 40 cm × 36 cm (Figure 15.38) to 2 m × 0.4 m, although mats with 15.5 sensors per cm^2 are now commercially available.

Further developments in the study of foot pressure have lead to more portable in-shoe pressure analysis systems which allow the analysis in the clinical setting. One example of this is Tekscan's F-Scan system (Figure 15.39).

UNDERSTANDING MOMENTS AND FORCES

When a force acts on a body some distance away from a pivot point, a turning effect is set up. Consider opening and closing a door whereby you are creating sufficient force to turn the door on its hinges. The force required to do this action multiplied by the distance away from the hinges is referred to as the *moment* (also sometimes called torque).

Now consider the see-saw in Figure 15.40a. It is clear that the see-saw will not balance. However, if the 1000 N force is moved so it is half the distance away from the pivot

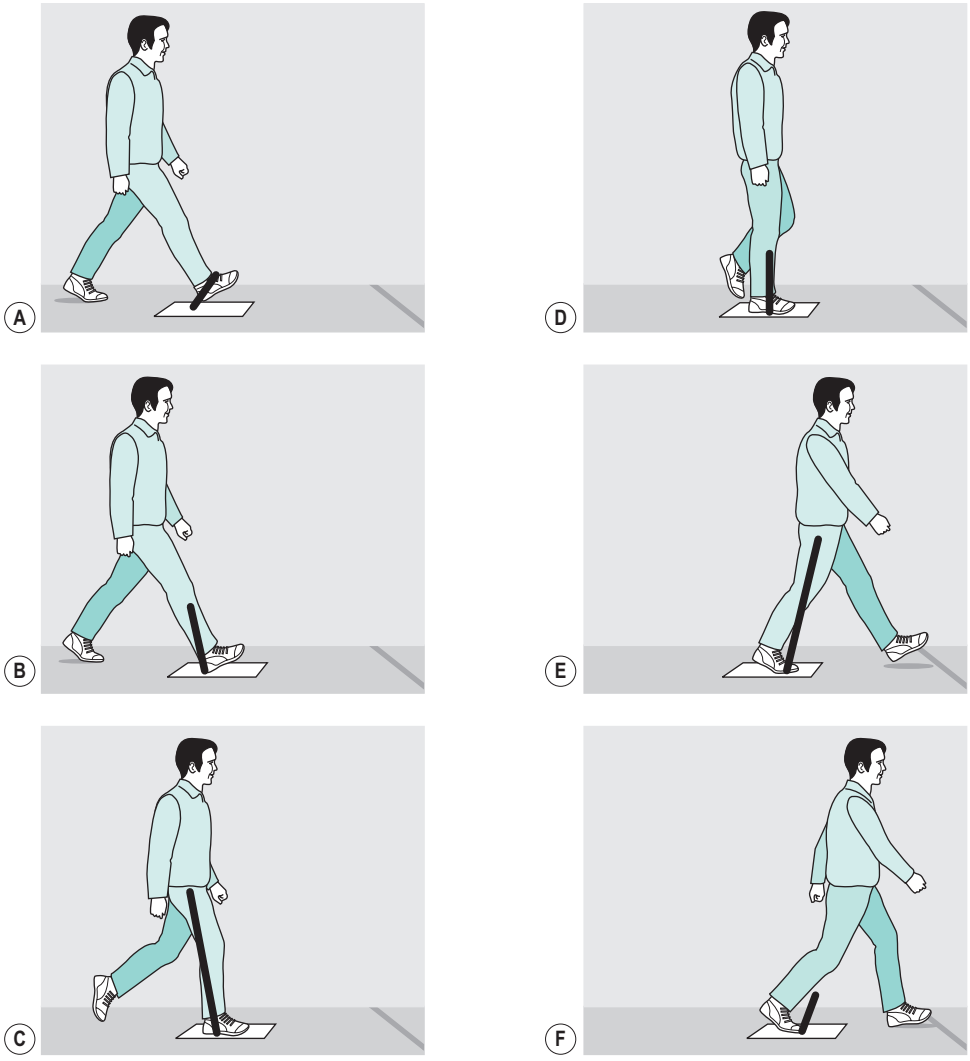


Figure 15.33 Output of video vector generator showing the ground reaction force at: (a) heel strike; (b) just after heel strike; (c) at flat foot; (d) at mid-stance; (e) at heel off; (f) just before toe off.

(point of rotation) and the 500 N force is moved twice the distance as in Figure 15.40b, it will balance.

To solve problems with moments we have to consider the action of each force in turn. To do this we consider if it will rotate the object (in this case a see-saw) in a clockwise or anticlockwise direction. If it is in a clockwise direction it is considered to be in a positive direction and if it is anticlockwise it is considered to be in a negative direction (these are arbitrary choices). Therefore, the 1000 N force will try and turn the see-saw clockwise and the 500 N force will try and turn the see-saw anticlockwise.

If the see-saw balances then the sum of the clockwise turning effects and anticlockwise turning effects must be zero. If we say that anticlockwise moments are negative and clockwise ones are positive, then:

$$\text{Sum of the Moments} = -(1000 \times 1) + (500 \times 2)$$

$$\text{Sum of the Moments} = -(1000) + (1000) = 0$$

i.e. If the sum of the moments on the see-saw is zero then the see-saw will balance.

Although there seems to be little effect when we consider the see-saw vertically, we have a weight on each side giving a total weight (force) of 500 + 1000 = 1500 N downwards. From Newton's third law we know that there must be an equal and opposite reaction force at the pivot of 1500 N acting up on to the pivot of the see-saw (Figure 15.40c).

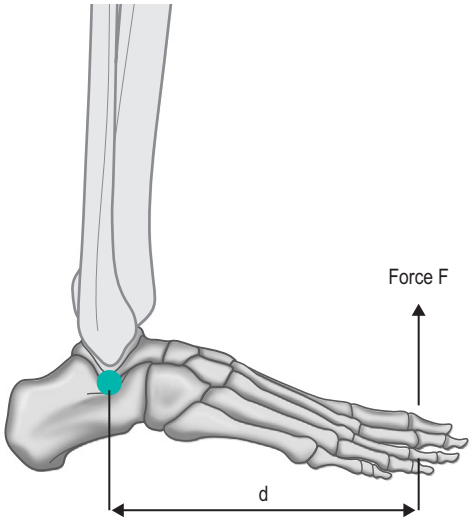


Figure 15.34 The moment about the ankle joint.

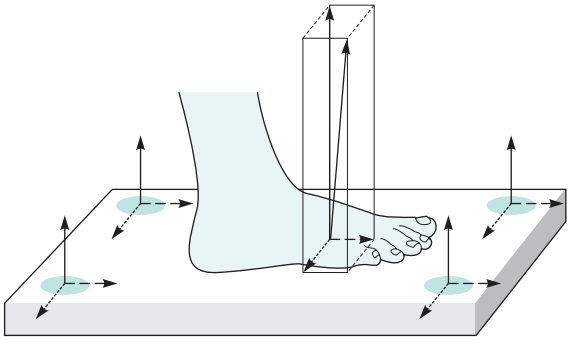


Figure 15.35 A force platform.

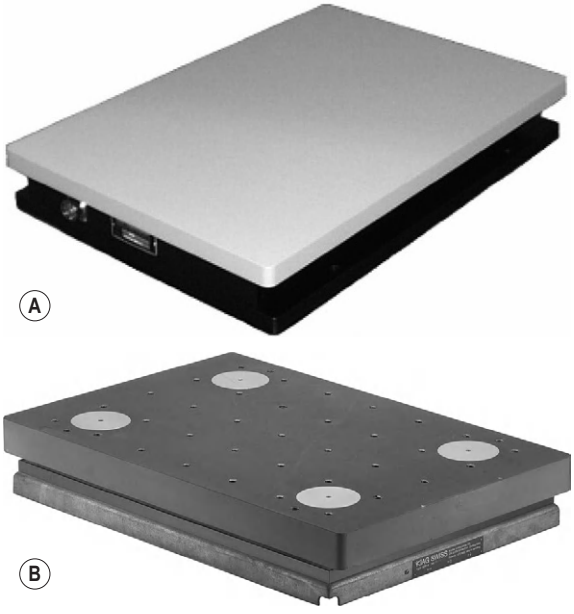


Figure 15.36 AMTI and Kistler Force platforms.

Figure 15.37 Pressure patterns beneath the foot.

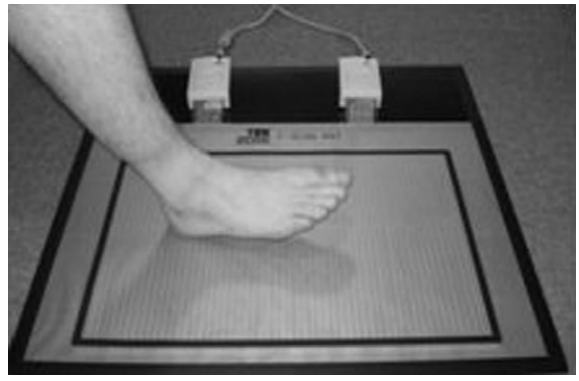


Figure 15.38 Pressure Mat System (FMat Tekscan Inc., South Boston, MA, USA).

Figure 15.39 In-shoe Pressure Measuring System (F-Scan, Tekscan Inc., South Boston, MA, USA).

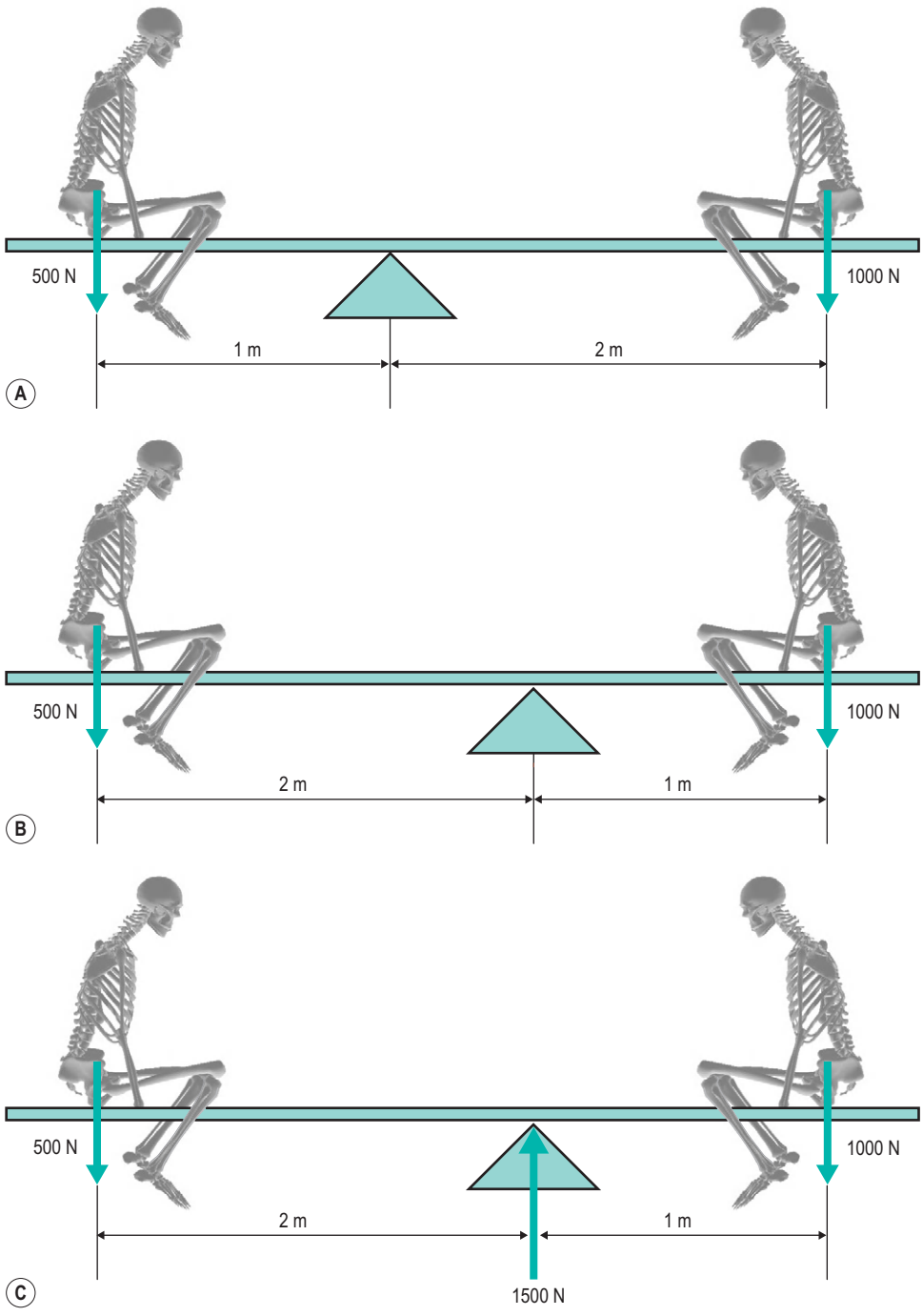


Figure 15.40 (a) Unbalanced see-saw. (b) Balanced see-saw. (c) Pivot reaction force.

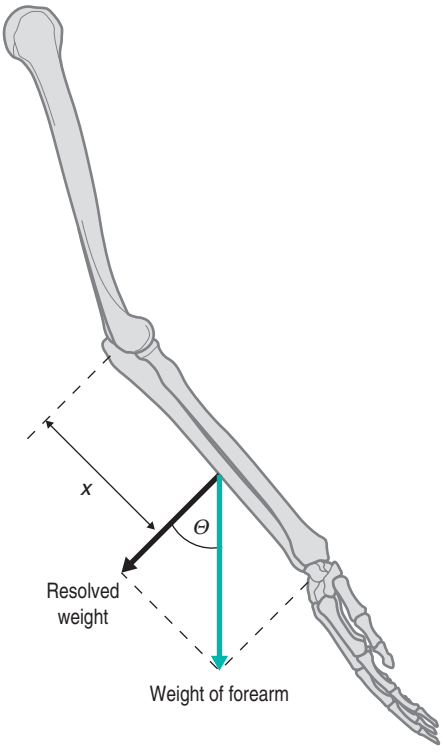


Figure 15.41 Moments in the upper limb using method 1.

HOW FORCES AND MOMENTS ACT ON THE BODY

Forces and moments acting on the body can be found in the same way as finding moments acting on a see-saw, as in the preceding section. Therefore, all we need to know is the forces acting about a joint and the distances at which they act from the joint. This is often referred to as inverse dynamics.

However, forces seldom act at 90 degrees to body segments. Therefore, we invariably need to *resolve* the forces first. There are two methods we can use:

- method 1 – the component of force at 90 degrees to the body segment;
- method 2 – the horizontal and vertical components of the force relative to the ground.

Student's note

This author's preference is to use the component of force at 90 degrees to the body segment *for upper limb and upper body* (trunk) problems, and the horizontal and vertical components of the force relative to the ground for *lower limb* problems which involve ground reaction forces.

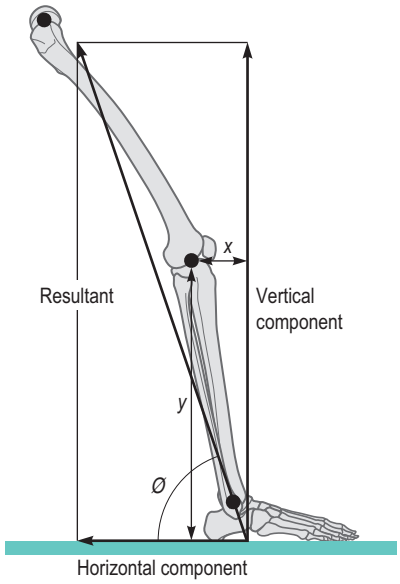


Figure 15.42 Moments in the lower limb using method 2.

How to find moments in the upper limb

Figure 15.41 shows the weight of the forearm acting straight down. This will produce an extending moment about the elbow.

Resolving the force

For this we will use method 1 as the weight of the forearm is not acting perpendicular to the forearm so we cannot use it to find the moment. However, if we find the component of the weight acting perpendicular to the forearm then we can.

The component acting perpendicular to the forearm = weight of the forearm × cos Θ.

Moments about the elbow

The component of the weight is acting a perpendicular distance of x away from the elbow. Therefore:

$$\text{Moment about the elbow} = \text{weight of the forearm} \times \cos\Theta \times x.$$

How to find moments in the lower limb

Figure 15.42 shows the resultant ground reaction force seen at foot flat in a normal subject. The resultant force can be broken up into two separate components, one in the vertical direction and the other in the horizontal direction.

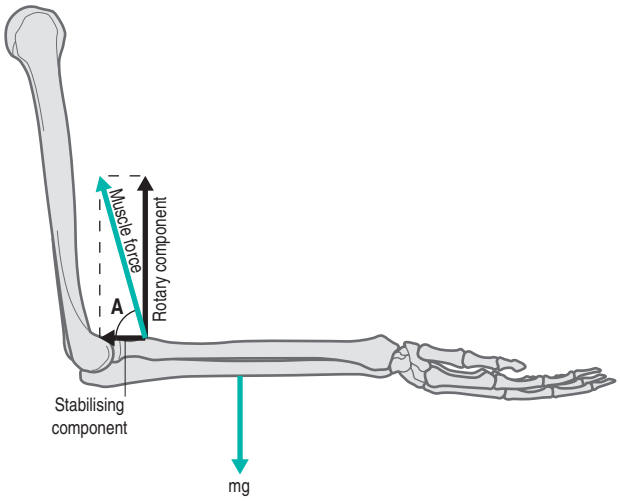


Figure 15.43 Components of muscle force.

If we consider the moments about the knee using method 2, we can find out the effects of both the vertical and horizontal components of the resultant ground reaction force about the knee.

Resolving the forces

$$\text{Vertical component} = \text{Resultant} \times \sin\Theta$$

$$\text{Horizontal component} = \text{Resultant} \times \cos\Theta.$$

Moments about the knee

To find the moment about the knee we are going to consider each of these forces separately.

- The horizontal force (resultant × cos Θ) is passing a perpendicular distance y below the knee. This force will try to turn the knee in a clockwise direction.
- The vertical force (resultant x sin Θ) is passing a perpendicular distance x in front of the knee. This force will try to turn the knee in an anticlockwise direction.

If we say all moments going clockwise are positive and those going anticlockwise are negative, then moments about the knee may be written as:

$$\text{Moment about the knee} = \text{Resultant} \times \cos\Theta \times y - \text{Resultant} \times \sin\Theta \times x.$$

How to find muscle and joint forces

Students without a mathematics background should not become too worried, as this section uses exactly the same principles dealt with in previous ones.

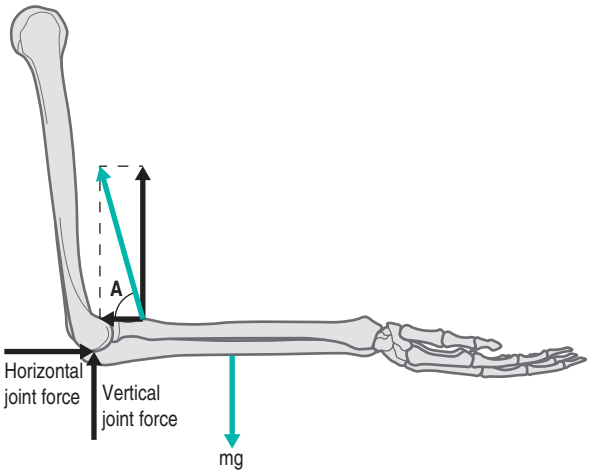


Figure 15.44 Components of joint force.

Muscle forces may be considered in the same way as balancing forces on a see-saw and joint forces may be considered in the same way as the force at the pivot in the middle of the see-saw.

Muscle forces

One way to calculate the moment owing to a force inclined at an angle is to break the force up into two perpendicular components. This is usually necessary in biomechanics calculations where the muscle acts at an angle to a body segment, as in the example in Figure 15.43. The muscle force has components along and perpendicular to the axis of the arm.

- The *rotary component* is the force that tries to turn the body segment around the proximal joint (e.g. flexing or extending the elbow joint) and balances the external moments acting on the body segment.

$$\text{Rotary component} = \text{muscle force} \times \sin A$$

- The *stabilising component* is the force that acts along the body segment (e.g. the forearm) forcing into, or pulling out of, the joint.

$$\text{Stabilising component} = \text{muscle force} \times \cos A$$

The stabilising component acts through the pivot and has zero moment; the rotary component will produce a moment about the proximal joint.

Joint forces

To find the joint force we first need to think back to the see-saw, where the force at the pivot was equal to the sum of the two forces acting downwards. We shall adopt the same technique here (Figure 15.44).

Firstly, we need to find all the forces acting in a vertical direction (including the vertical component of the muscle force). The only force we will not know is the vertical force at the joint. The sum of all these forces must be zero.

Secondly, we need to find all the forces acting in a horizontal direction (including the horizontal component of the muscle force). The only force we will not know is the horizontal force at the joint. The sum of all these forces must also be zero.

Worked example 1: How to find muscle and joint forces in the upper limb

Consider the turning moments about the elbow joint while holding a weight or, more correctly, a mass of 5 kg (Figure 15.45). The 5 kg mass will produce a force, or weight, of 49.05 N and the forearm and hand will produce a force, or weight, of 25 N. If we know the distance d_1 from the elbow joint to the centre of mass of the forearm and hand is 0.15 m, and the distance d_2 from the elbow joint to the 'weight' is 0.4 m then we can find the moments about the elbow.

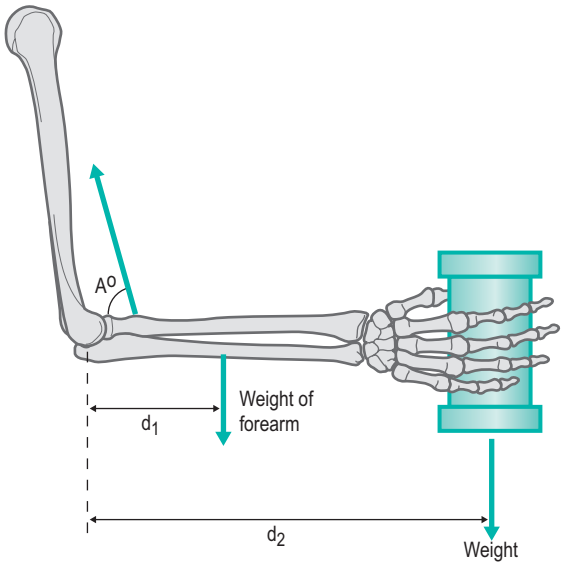


Figure 15.45 Muscle and joint forces in the upper limb for worked example 1.

Finding the moment about the elbow joint

Moment = force × perpendicular distance.

Therefore:

Moments about the elbow joint
= (weight of forearm × d_1)
+ (weight × d_2)
Moments about the elbow joint
= (25 × 0.15) + (49.05 × 0.4)
Moments about the elbow joint = 3.75 + 19.62
= 23.37 Nm.

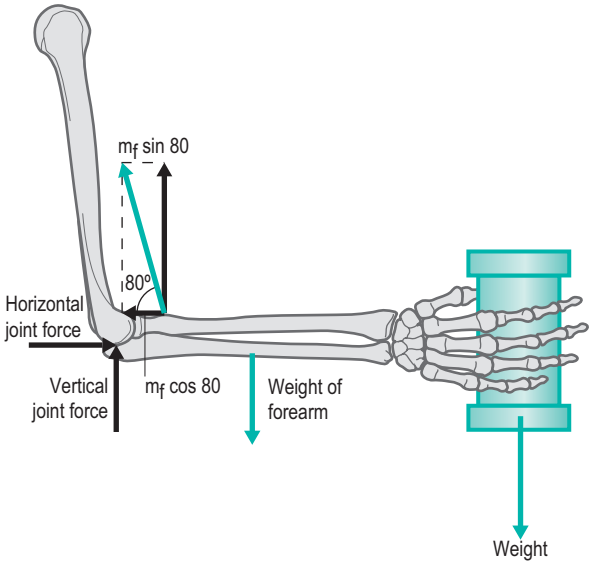


Figure 15.46 Resolving the horizontal and vertical forces.

Finding the force in the muscle

Assume that the muscle is inclined to the forearm at 80 degrees (angle *A*) and the muscle insertion point is 0.04 m away from the elbow joint (Figure 15.46).

The muscle must provide an equal and opposite moment to support the weight of the arm and the weight. However, the muscle is inclined to the forearm so the muscle force needs to be resolved so that it is perpendicular to the distance from the elbow joint.

If the muscle force is given the symbol m_f so the vertical component (or rotary component) of it will be $m_f \sin 80°$. As the muscle produces an equal and opposite moment, the clockwise component must equal the anticlockwise component, i.e. the muscle must provide an equal and opposite moment to:

Moments about the elbow joint = 23.37 Nm

Therefore:

$$m_f \times \sin 80° \times 0.04 = 23.37$$
$$m_f = 23.37/(\sin 80° \times 0.04)$$
$$m_f = 593.3\ \text{N}$$

Therefore, the force in the biceps when holding a 5 kg mass is 593.3 N. To put this into context this is the body weight of someone with a mass of 60.5 kg. This highlights that the amount of force on the muscles and tendons is often considerable greater than we would, perhaps, imagine.

Finding the joint force

Vertical forces

In the problem above the vertical forces include: $m_f \sin 80°$ (584.3 N), weight (49.05 N), weight of the forearm (25 N) and the vertical joint force (Figure 15.46).

If the force is acting up we will call it positive, if it is acting down we will call it negative. Therefore:

$$m_f \sin 80° - \text{weight} - \text{weight of the forearm} - \text{vertical joint force} = 0$$

$$584.3 - 49.05 - 25 - \text{vertical joint force} = 0$$

$$584.3 - 49.05 - 25 = \text{vertical joint force}$$

$$510.25 \text{ N} = \text{vertical joint force}$$

Horizontal forces

The horizontal forces in this problem are easier; the only forces which will have a horizontal component are the muscle force and the joint force. If the force is acting to the right we will call it positive, if it is acting to the left we will call it negative.

$$\text{Horizontal joint force} - m_f \cos 80° = 0$$

$$\text{Horizontal joint force} - 593.3 \cos 80° = 0$$

$$\text{Horizontal joint force} = 593.3 \cos 80°$$

$$\text{Horizontal force} = 103 \text{ N}$$

Resultant joint force

There is one last step. What we want to find is the total effect on the joint, or the resultant joint force. This can be found simply using Pythagoras:

$$510.25^2 + 103^2 = R^2$$

$$520.5 = R = \text{resultant joint force}$$

As with the muscle forces the joint and ligament forces are also greater than we would imagine. This also helps to explain why ligaments and bone, which are immensely strong, are often damaged, and why it is also very important to consider these forces during rehabilitation to avoid overloading these structures.

Worked example 2: How to find moments in the lower limb

Figure 15.47 shows the ground reaction force acting at heel strike. The point of application of the ground reaction

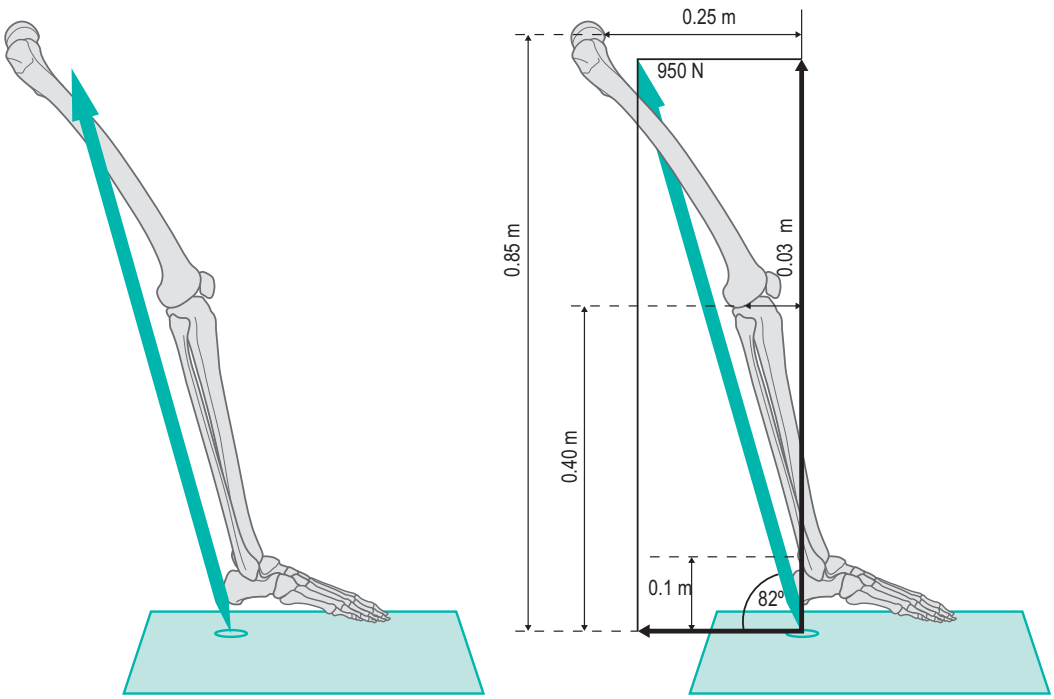


Figure 15.47 Joint moments in the lower limb during walking.

force, the position of the ankle, knee and hip joints are known. To determine the effect on the lower limb we need to calculate the moments produced by the ground reaction force about (i) the ankle joint, (ii) the knee joint and (iii) the hip joint. The ground reaction force, 950 N is acting at 82 degrees to the horizontal.

Resolving

$$\text{Horizontal ground reaction force} = 950 \times \cos 82$$

$$\text{Vertical ground reaction force} = 950 \times \sin 82$$

$$\text{Horizontal ground reaction force} = 132.2 \text{ N}$$

$$\text{Vertical ground reaction force} = 940.75 \text{ N}$$

Once the vertical and horizontal forces have been found we can now consider the action of each component about each joint separately.

Moments about the ankle (Figure 15.48)

The vertical component of the ground reaction force acts straight through the joint; therefore, this will not produce a moment. The horizontal component acts to the right and below the ankle.

$$\text{Moment about the ankle} = 940.75 \times 0 + 132.2 \times 0.1$$
$$= 13.2 \text{ Nm}$$

Moments about the knee (Figure 15.49)

The vertical component of the ground reaction force acts in front of the knee joint. The horizontal component acts to the right and below the knee.

$$\text{Moment about the knee} = -940.75 \times 0.03 + 132.2 \times 0.4$$
$$= 24.66 \text{ Nm}$$

Moments about the hip (Figure 15.50)

The vertical component of the ground reaction force acts in front of the hip joint. The horizontal component acts to the right and below the hip.

$$M_{hip} = -940.75 \times 0.25 + 132.2 \times 0.85 = -122.82 \text{ Nm}$$

What are the effects of these moments on the muscles?

- The moment about the ankle joint is a plantar flexing moment; therefore, the muscles in the anterior compartment of the ankle joint must be active (dorsiflexors).
- The moment about the knee joint is a flexing moment; therefore, the muscles in the anterior compartment of the knee joint must be active (knee extensors).

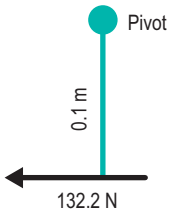


Figure 15.48 Moments about the ankle.

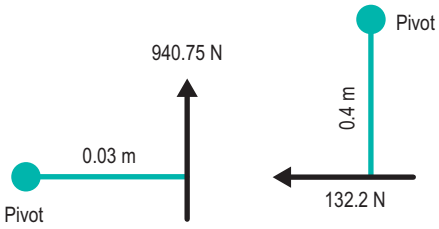


Figure 15.49 Moments about the knee.

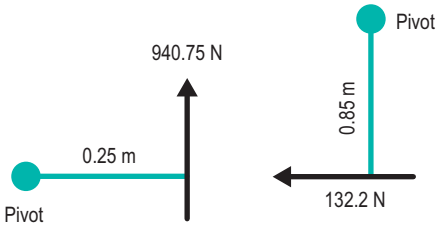


Figure 15.50 Moments about the hip.

- The moment about the hip joint is a flexing moment; therefore, the muscles in the posterior compartment of the hip joint must be active (hip extensors).

Worked example 3: How to find muscle and joint forces on the base of the spine

A person lifts a weight of 600 N, as shown in Figure 15.51.

- Mass of trunk = 52.5 kg
- Mass of head = 8.5 kg
- Mass of upper arms = 5.8 kg
- Mass of forearm = 3.4 kg.

(i) If the centre of mass of the trunk, arms and head is a horizontal distance of 0.2 m from L5–S1 (the base of the spine) and the weights being lifted are a horizontal distance of 0.42 m from L5–S1, find the moment about L5–S1.

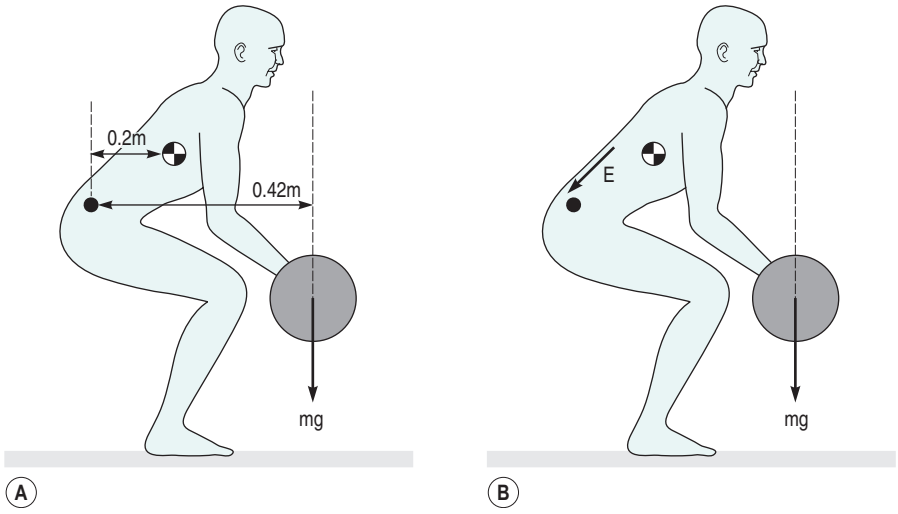


Figure 15.51 Muscle and joint forces on the spine.

(ii) If this moment were supported entirely by the muscle E acting 0.07 m away from L5–S1, what would be the force in the muscle?
(iii) Find the resultant force at L5–S1 if the force *E* is acting at 40 degrees to the vertical.

Solution

(i) $M = 600 \times 0.42 + 702 \times 0.2 = 392.4$ Nm.
(ii) 392.4 Nm = E × 0.07
392.4 / 0.07 = *E*
5606 N = *E*
(iii) Force *E* is acting at an angle to vertical and horizontal, so we find the vertical and horizontal component by resolving:
$E_v = 5606 \times \cos 40 = 4294$ N
$E_h = 5606 \times \sin 40 = 3603$ N
Total force vertical = 4294 + 600 + 702 = 5596 N
Total force horizontal = 3603 N
Resultant = sq root ($5596^2 + 3603^2$)
Resultant = 6655 N.

MOMENTS ABOUT THE ANKLE KNEE AND HIP JOINTS DURING NORMAL WALKING

We have previously considered the movement and ground reaction forces during the gait cycle. We will now consider the effects of the position of the ground reaction force in relation to the ankle, knee and hip joints.

Typical ankle moments during normal gait

At heel strike the ground reaction force passes very close to the ankle joint centre, therefore producing a very small moment. In some cases this will be behind the ankle joint giving rise to a plantar flexion moment. After heel strike the ground reaction force moves in front of the ankle joint producing a dorsiflexion moment. This increases as the force moves under the metatarsal heads and the force increases during push off (Figure 15.52).

Typical knee moments during normal gait

At heel strike the ground reaction force initially passes anterior to the knee joint, giving rise to an extension moment. The ground reaction force then passes quickly behind the knee joint causing a flexion moment. After mid-stance the force passes in front of the knee again until toe off. During swing phase the knee also has significant moments owing to the acceleration and deceleration of the foot and tibia (Figure 15.53).

Typical hip moments during normal gait

At heel strike the ground reaction force passes quite far anterior to the hip joint producing a peak flexion moment. After heel strike the ground reaction force still passes anterior to the hip; however, the distance from the force to the hip reduces. After mid-stance the force passes posterior to

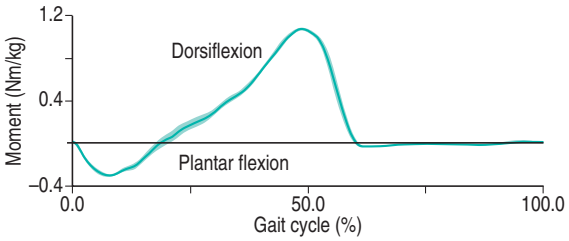


Figure 15.52 Typical ankle moments during normal gait.

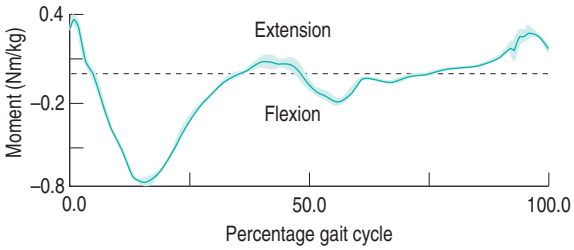


Figure 15.53 Typical sagittal knee moments during normal gait.

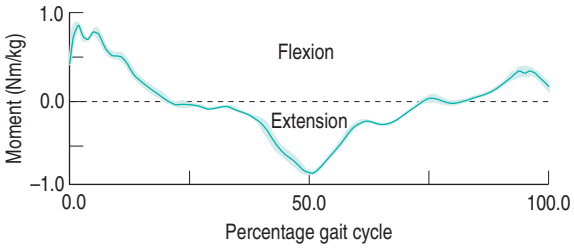


Figure 15.54 Typical sagittal hip moments during normal gait.

the hip giving rise to an extension moment. As with the knee there are significant moments during swing phase owing to the acceleration and deceleration of the lower limb (Figure 15.54).

HOW TO FIND LINEAR WORK, ENERGY AND POWER

Linear work

Work is a product of a force applied to a body and the displacement of the body in the direction of the applied force (Figure 15.55).

$$\text{Work} = \text{force} \times \text{displacement}\ (W = Fs).$$

Linear work does not refer to the muscular or mental effort. Work is basically a force overcoming a resistance and moving an object through a distance. If, for example, an object is lifted from the floor to the top of a table, work is done in overcoming the downward force owing to gravity. However, if a constantly acting force does not produce motion, no work is performed.

Key point

- Holding a book steadily at arm's length does not involve any work, irrespective of the effort required, because there is no movement of the applied force.
- The units of work are newton × metres or *joules* (J).

Linear power

Power is the *rate* of performing work or transferring energy. Therefore, power measures how quickly the workis done.

Suppose a person pushes a box from one end of the room to the other in ten seconds, then pushes the box back to its original position in five seconds. In each trip across the room, the force applied and the distance the box is moved is the same, so the work done in each case is the same. But the second time the box is pushed across the room, the person has to produce more power than in the first trip because the same amount of work is done in five seconds rather than ten.

$$\text{Power} = \text{work done/time taken}$$

Key point

The units of power are joules per second (J/s) or *watts* (W).

Linear energy

While work is done on a body, there is a transfer of energy to the body, and so work can be said to be 'energy in transit'.

Key point

Energy has the same units as work (*joules*) as the work done produces a change in energy.

Energy is the capacity of matter to perform work as the result of its motion or its position in relation to forces acting on it. Energy related to position is known as *potential energy* and energy associated with motion is known as *kinetic energy*. A swinging pendulum has maximum potential energy at the terminal points; at all intermediate positions it has both kinetic and potential energy in varying proportions.

Conservation of energy

Energy can be transformed but it cannot be created or destroyed. In the process of transformation either kinetic or potential energy may be lost or gained, but the sum total of the two always remains the same.

Potential energy

This is stored energy possessed by a system as a result of the relative positions of the components of that system. For example, if a ball is held above the ground, the system comprising the ball and the earth has a certain amount of potential energy; lifting the ball higher increases the amount of potential energy the system possesses. This is expressed mathematically as $PE = mgh$ (mass x gravity × height).

Work is needed to lift the ball up, giving the system potential energy. It takes effort to lift a ball off the ground. The amount of potential energy a system possesses is equal to the work done on the system.

Potential energy also can be transformed into other forms of energy. For example, when a ball is held above the ground and released, the potential energy is transformed into kinetic energy.

Kinetic energy

This is energy possessed by an object resulting from the motion of that object. The magnitude of the kinetic energy depends on both the mass and the speed of the object according to the equation $KE = \frac{1}{2}mv^2$.

HOW TO FIND ANGULAR WORK AND POWER

Angular work

$$\text{Work} = \text{force} \times \text{distance moved}$$

The distance the force is moved is not in a straight line as with linear work, but in an arc. To find the angular work the length of the arc must first be found.

The length of the arc is affected by the radius of the arc and the angle moved through.

$$\text{Length of an arc} = \text{radius} \times \text{angular displacement}$$
$$\text{Length of an arc} = r\Theta.$$

Note: the angular displacement must be measured in radians (rads):

$$1 \text{ radian} = 57.3°$$
$$\text{Work} = Fs$$
$$\text{Angular work} = Fr\Theta.$$

However:

$$\text{Force} \times r = \text{moment (M)}$$

Therefore:

$$\text{Work} = \text{moment } (M) \times \Theta$$

Angular power

$$\text{Power} = \frac{\text{Work done}}{\text{Time taken}}$$
$$\text{Power} = \frac{\text{Force} \times \Theta \times r}{t}$$

or

$$\text{Power} = \frac{\text{Moment } (M) \times \text{t}}{\text{Time taken}}$$
$$\text{Power} = \frac{M \times \Theta}{t}$$

However:

$$\Theta/t = \omega \text{ (angular velocity)}$$

so:

$$\text{Power} = M\omega$$

ω is in Radian/s (rad/s)

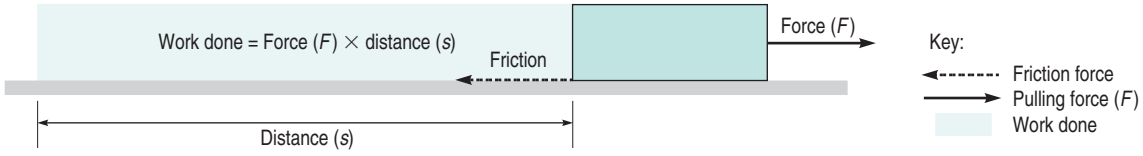


Figure 15.55 Linear work.

JOINT POWER DURING NORMAL WALKING

The results below are found by combining the joint moments (M) and joint angular velocities (ω). Positive values show power generation and negative values show power absorption. It is also sometimes beneficial to consider where the muscle action is either concentric or eccentric. Concentric activity is associated with power generation, whereas eccentric activity is associated with power absorption. However, these graphs can be hard to interpret if you consider whether the moments are trying to flex or extend, and what movements are occurring at each time. The examples below consider the power absorption and generation of the ankle, knee and hip joints during walking. Power absorption (eccentric activity) is shown as negative and power generation (concentric activity) is positive.

Ankle power

If the ankle has a dorsiflexion velocity and the moment is dorsiflexing about the ankle joint then the posterior muscles must be working eccentrically and absorbing power. If the ankle has a plantar flexion velocity and a dorsiflexion moment then the posterior muscles must be working concentrically and therefore generating power.

At heel strike there is a plantar flexion moment and angular velocity, therefore at heel strike there is eccentric power absorption from the dorsiflexors. This is followed by an eccentric power absorption by the plantar flexors as the body moves forwards over the foot. During push off there is a dorsiflexion moment and a plantar flexion velocity, therefore power is generated by concentric activity of the plantar flexors (Figure 15.56).

Knee power

At heel strike the knee shows power generation owing to the ground reaction force being in front of the knee therefore producing an extension moment while the knee is flexing. This initial power generation or concentric contraction of the hamstrings ensures that the knee does, indeed, flex at heel strike, rather than moving into a hyperextended position. After this point the ground reaction force falls behind the knee creating a flexion moment while the knee is flexing, therefore the quadriceps will be working eccentrically to act as a shock absorber. The knee then shows power generation at approximately 20% of the gait cycle. At this point, the ground reaction force is behind the knee and this therefore relates to the quadriceps acting concentrically, pulling the femur over the tibia. As the knee extends the ground reaction force passes through the knee joint producing on moment and therefore little, or no, power is generated or absorbed. It is interesting to note the involvement of the knee during push off at 50–60% of the gait cycle. During this time the ground reaction force falls behind the knee creating a flexion moment but at this time the knee is flexing and therefore power is being absorbed, not generated. The knee, therefore, has little or no involvement in the power production during push off (Figure 15.57).

Hip power

At heel strike the hip shows power absorption at heel strike owing to the hip having a small period of flexion velocity coupled with a flexion moment. The hip then extends to start to move the body over the stance limb while the moment is still trying to flex the hip; this is achieved by power generation by the hip extensors. At approximately 25% of the gait cycle the moment passes behind the hip changing from a flexion moment to an extension moment; however, the hip is still extending. This relates to power absorption, or eccentric control, of the hip flexors as the body moves over the stance limb. After 50% of the gait cycle the hip reaches its maximum extended position. After this point there is a rapid power generation during push off. This power generation is a result of the ground reaction force creating an extension moment while the hip changes from a flexing angular velocity to an extending angular velocity and therefore contributes to power production during push off (Figure 15.58).

STRENGTH TESTING AND TRAINING

So far in this chapter we have been looking at moments, muscle forces and joint forces. However, one way in which we often talk about muscle and joint performance is *strength*. But what exactly *is* strength? The dictionary tells us that strength is the capacity for exertion or endurance or the power to resist force. However, a better way of thinking about *muscle* strength is the amount of force a particular muscle or muscle group can produce. When evaluating muscle strength, however, the measures taken do not directly measure the actual strength of the muscle or muscle group. What is usually recorded is the effective moment being produced by the muscle. This is because muscle forces are hard to measure as we require information about the position of muscle, position of the body segment, muscle insertion points and the line of pull of the muscle – all of which will be constantly changing during dynamic activities.

Most measures taken in the clinical setting do not go as far as to estimate actual muscle forces. However, there are a number of methods of indirect evaluation. Indirect evaluation of the force produced by a muscle can,

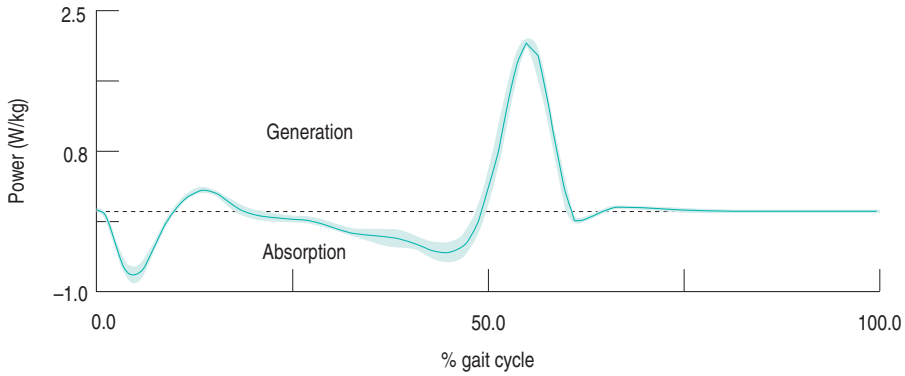


Figure 15.56 Typical ankle power during normal gait.

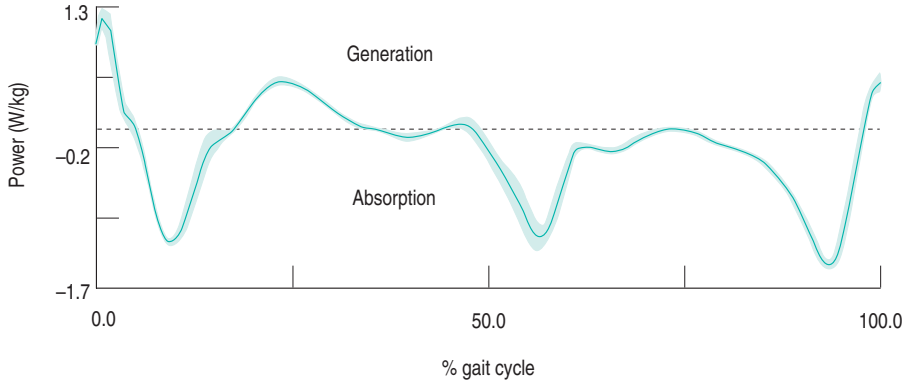


Figure 15.57 Typical knee power during normal gait.

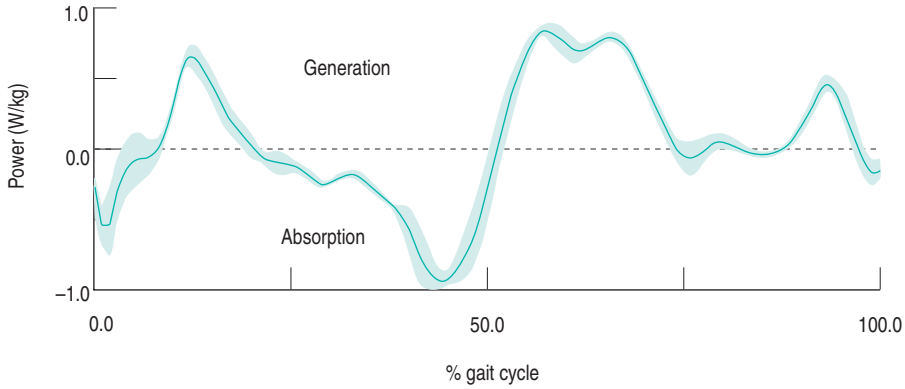


Figure 15.58 Typical hip power during normal gait.

however, be influenced by a number of factors. The following section will consider the different permutations of these factors when considering upper limb muscle strength.

These factors include:

- the body segment inclination;
- the position and size of the applied load;
- muscle insertion points;
- angle of muscle pull;
- type of contraction;
- speed of contraction.

Changing the effective moment caused by the body segment inclination

The inclination of body segments can have a very large effect on joint moments. The effect of the weight of the forearm in the three positions shown is very different (Figure 15.59). The maximum moment about the elbow is when the forearm is level, when the forearm is inclined either up or down the moment reduces, and when the forearm is vertical there will be no moment about the elbow at all as the entire weight of the segment will be acting through the joint. It is also important to note the direction of the 'stabilising' component that acts along the forearm. When the forearm is angled down the component acting along the forearm will try to pull the forearm away from the upper arm, whereas with the forearm angled up the forearm is pushed into the upper arm. This will have the effect of reducing and increasing the joint force at the elbow respectively.

The position and size of the applied load

The position and size of the load applied has an important effect on the moment about the elbow, and will, in turn, have a significant effect on the muscle and joint forces. When assessing muscle strength both these factors should be measured and taken into account. If you position a load at the end of a subject's arm to see if he/she can support it, the moment will depend on the size of the load and the subject's limb length (Figure 15.60).

Both the size of the load and the subject's limb length need to be considered when assessing an individual's muscle performance or strength. For example, if two individuals of different heights and therefore different tibial (shank) lengths conducted the same leg raise activity with the same loads the shorter of the two would, in fact, use less muscle force (strength) to lift the same load, assuming the muscle insertion points were not significantly different.

Muscle insertion points

Different muscles will have different insertion points. The position of these insertion points will have a large effect on the muscle force required to support a given turning moment. Two examples are shown in Figure 15.61. In Figure 15.61a the muscle insertion point is close to the elbow joint, while in Figure 15.61b the muscle insertion point is much further away. For a particular load there will be a larger force in the muscle if its insertion point is close to the joint. Conversely, if there is a maximum force that a muscle group can cope with then larger loads will be

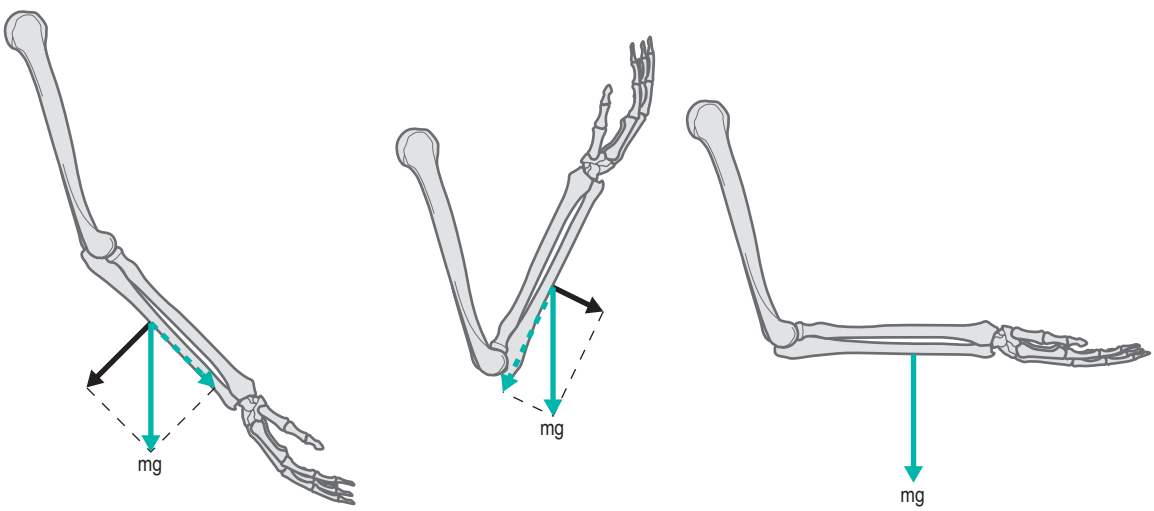


Figure 15.59 Effective moment caused by the weight of the limb.

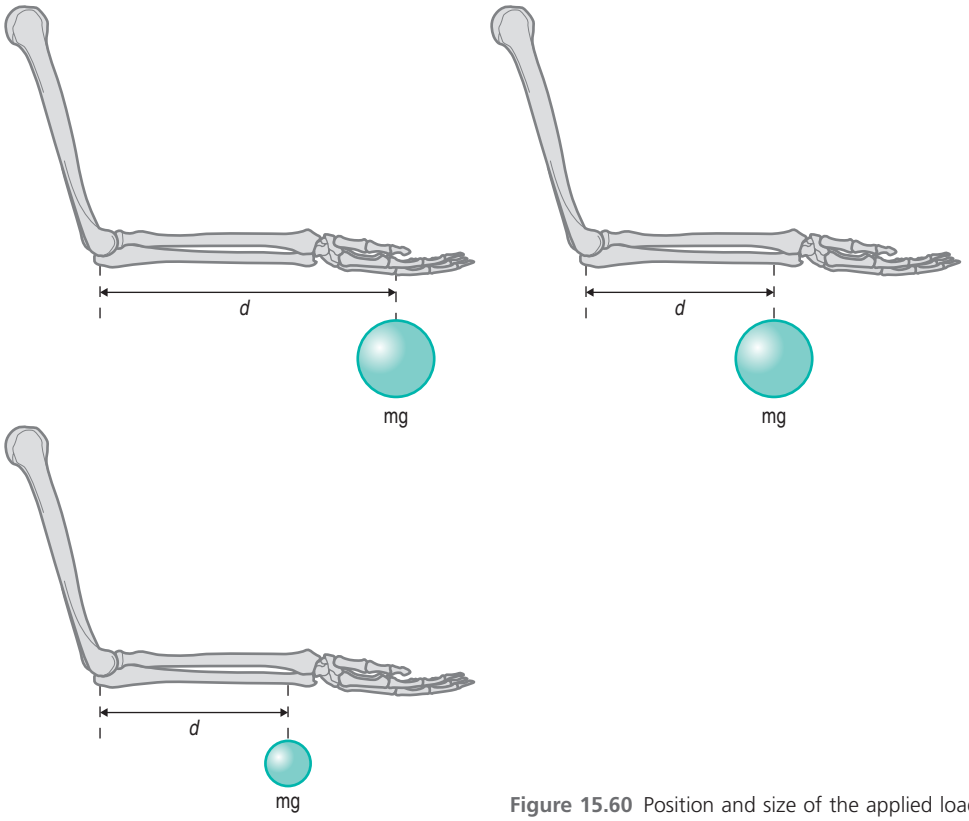
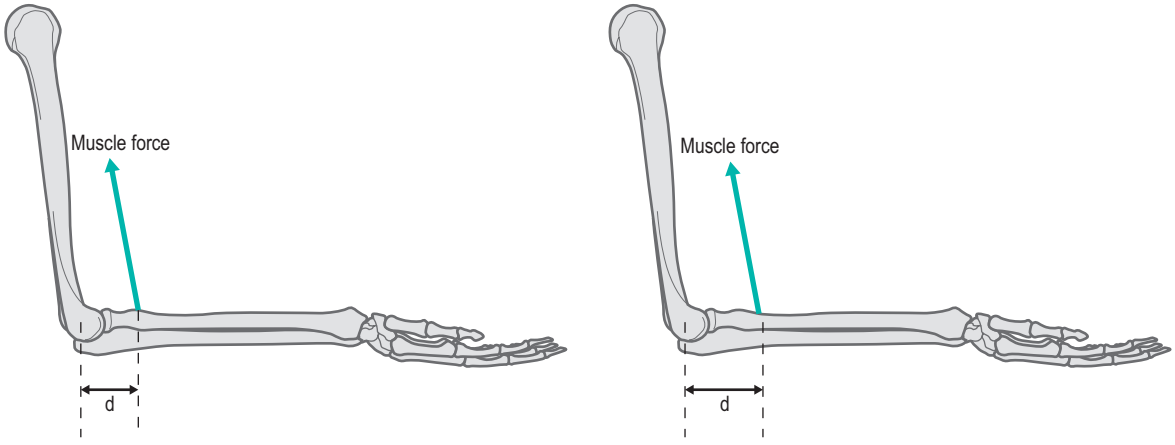


Figure 15.60 Position and size of the applied load.



a) Muscle insertion point close

b) Muscle insertion point further

Figure 15.61 Position and size of the applied load to the joint.

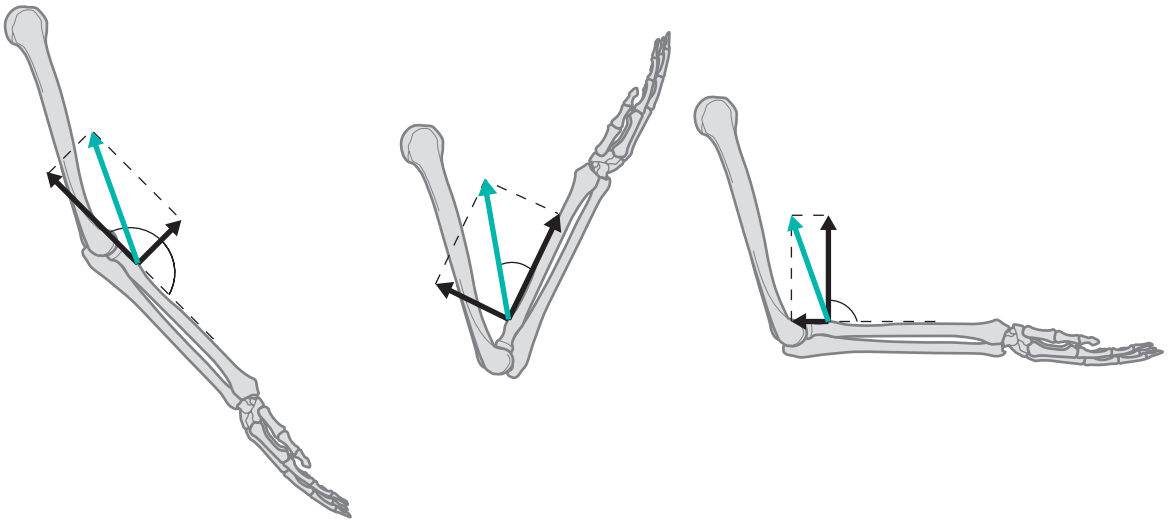


Figure 15.62 Effect of the angle of muscle pull.

able to be carried with the insertion point further away from the joint.

This leads us to an interesting point when we consider weight lifters. Is a weight lifter able to lift the larger load because he or she can support larger muscle forces or is this owing to a difference in the muscle insertion points? If this is the case are we actually assessing something different from *strength* (the force in the muscle) with the task?

The effect of the angle of muscle pull

As the body segment moves relative to the ground, so does the angle of the muscle relative to the body segment. Consider different inclinations of the body segment, but instead of thinking about the moment owing to the weight, think about the line of action of the force in relation to the forearm. The maximum moment that the muscle can produce is when the elbow is at 90 degrees as this makes an approximately 90 degree angle between the muscle and the body segment, and therefore produces the greatest rotary component from the muscle force. As the elbow joint is moved away from this position, either flexed or extended, the moment that the muscle can produce is reduced as the rotary component of the muscle force acting at 90 degrees to the forearm is reduced. When the forearm is vertical with the elbow fully extended the muscle would find it much harder to produce a moment as the rotary component will be at its smallest. It is also interesting to note the direction of the 'stabilising' component of the muscle force when the elbow is flexed. This appears to be pulling the forearm away from the joint and will not provide a compressive stabilising force into the joint; however, in this position the rotary component will be providing a compressive force into the joint. In reality, we will also have a co-contraction from the extensor muscles to stabilise the elbow joint (Figure 15.62).

Type of muscle contraction

The type of muscle contraction affects the resistance that can be controlled, held or overcome. The three types of muscle contraction are isometric, concentric and eccentric. Isometric contractions are stabilising contractions where the muscle length remains virtually constant. Concentric contractions are where the muscle shortens during the activity. These are generally the weakest muscle contractions, requiring more motor unit recruitment than isometric and eccentric for a particular load. Eccentric contractions are where the muscle lengthens during the activity. These are generally the strongest muscle contractions, requiring less recruitment than isometric and concentric for a particular load.

The effect of the speed of contraction

There are three ways of classifying speed during exercises: isotonic, isokinetic and isometric. Isotonic is when a constant load is applied but the angular velocity of the movement may change, allowing an infinite variation in the rate of contraction of a muscle. Although this is closest to real life muscle and joint function the changing in speed continually affects the amount of force that a muscle can produce and makes the exact muscle function quite hard

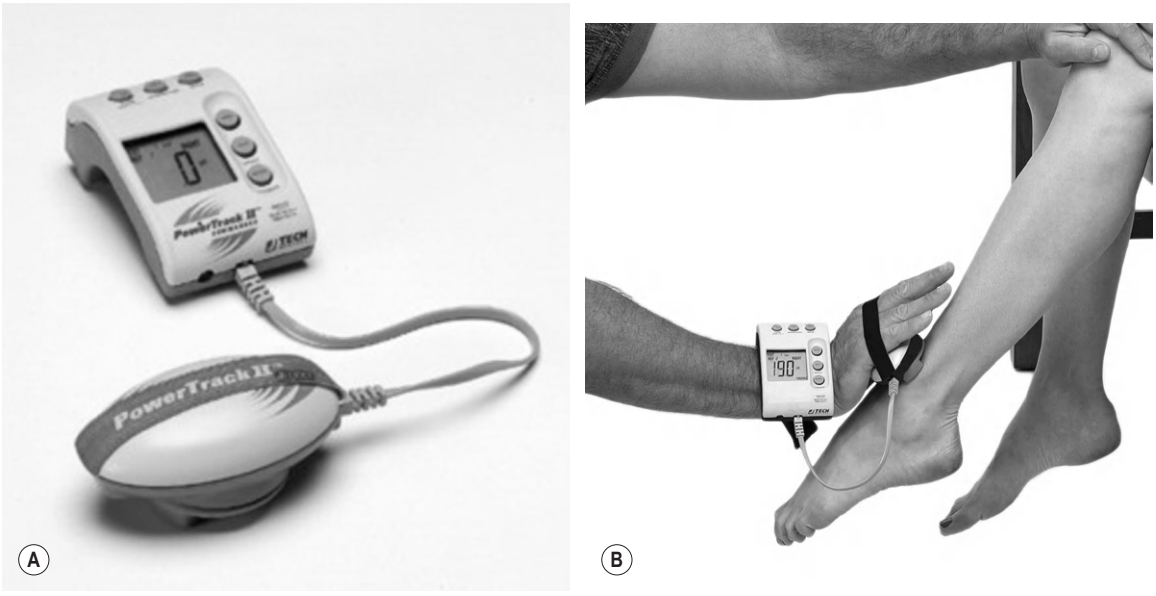


Figure 15.63 JTech Commander Powertrack II.

to assess. Isokinetic is when the velocity or angular velocity of the movement is kept constant, but the load may be varied. This setting of the speed of working helps improve our assessment of muscle performance, but the speed or velocity of the joints are being restricted to only one set speed at any one time. Isometric relates to the force varying but the joint is held in a static position, therefore muscle length remains the same as no movement occurs. This tells us what static moment may be supported; however, it does not necessarily relate to the moments that can be produced or supported dynamically.

Methods of objective assessment

One method of improving the measurements taken in clinical assessment is using a handheld dynamometer. One example of a dynamometer is the Jtech medical Commander Powertrack II, which is a force gauge that has been used to assess the muscle strength component of the constant score to evaluate shoulder function (Figure 15.63). It does have the drawback of assessing isometrics and not dynamic tasks, and care must still be taken in the position of the device on the body segments to ensure repeatable and useful measurements.

Another method is using an isokinetics dynamometer, which allows for a standardised assessment by controlling, or pre-setting the angular velocity and measuring the resistance that can be produced by an individual (Figure 15.64). In controlling the angular velocity and measuring the resistance, the muscle power produced becomes very easy to find. Isokinetics also allow concentric, eccentric

Figure 15.64 Isokinetic machine.

and isometric moments (commonly referred to as torque in isokinetics), and concentric and eccentric power to be found separately. Many isokinetic machines are also capable of isotonic assessment, isotonic referring to constant load, or torque, throughout the range of motion. Isotonic testing is a simulating of free weights, although isokinetic machines also have the ability to allow for the

weight of the segment through the range of motion being tested therefore giving a true representation of the torque and power provided by the muscles.

CONCLUSION

This chapter highlights some of the background theory and methods necessary for the analysis and interpretation of the assessment of movement, forces, moments, strength and power. This chapter should help the reader to understand the movement patterns of individuals who are pain- and pathology-free, and to become familiar with some of the techniques used in the clinical research literature. All these techniques may be applied to the assessment of pathologies and conditions which affect movement which can enable objective assessment and monitoring of recovery through treatment and rehabilitation programmes.

REFERENCES

Arenson, J.S., Ishai, G., Bar, A., 1983. A system for monitoring the position and time of feet contact during walking. J Med Eng Technol 7 (6), 280–284.

Al-Majali, M., Solomonidis, S.E., Spence, W., et al., 1993. Design specification of a walk mat system for the measurement of temporal and distance parameters of gait. Gait Posture 1, 119–120.

Bell, F., Ghasemi, M., Rafferty, D., et al., 1995. An holistic approach to gait analysis: Glasgow Caledonian University's CRC. Gait Posture 3, 185.

Bell, F., Shaw, L., Rafferty, D., et al., 1996. Movement analysis technology in clinical practice. Phys Ther Rev 1, 13–22.

Brand, R.A., Crowninshield, R.D., 1981. Comment on criteria for patient evaluation tools. J Biomech 14 (9), 655.

Bruckner, J., 1998. The Gait Workbook: A Practical Guide to Clinical Gait Analysis. SLACK Inc., Thorofare, NJ.

Crouse, J., Wall, J.C., Marble, A.E., 1987. Measurement of temporal and spatial parameters of gait using a microcomputer based system. J Biomed Eng 9 (1), 64–68.

Durie, N.D., Farley, R.L., 1980. An apparatus for step length measurement. J Biomed Eng 2 (1), 38–40.

Elftman, H., 1938. Forces and energy changes in the leg during walking. Am J Physiol 125, 339–356.

Fox, J., Day, R., 2009. A Physiotherapist's Guide to Clinical Measurement. Elsevier, Oxford.

Gage, J.R., 1994. The role of gait analysis in the treatment of cerebral palsy. J Pediatr Orthoped 4, 701–702.

Gerny, K., 1983. A clinical method of quantitative gait analysis. Phys Ther 63, 1125–1126.

Hirokawa, S., Matsumura, K., 1987. Gait analysis using a measuring walkway for temporal and distance factors. Med Biol Eng Comput 25, 577–582.

Inman, V.T., 1966. Human locomotion. Can Med Assoc J 94, 1047–1054.

Inman, V.T., 1967. Conservation of energy in ambulation. Arch Phys Med Rehab 48, 484–488.

Inman, V.T., Ralston, H.J., Todd, F., 1981. Human Walking. Williams & Wilkins, Baltimore.

Kirtley, C., 2005. Clinical Gait Analysis: Theory and Practice. Churchill Livingstone, Edinburgh.

Levine, D., Richards, J., Whittle, M.W., 2012. Whittle's Gait Analysis, fifth ed. Churchill Livingstone, London.

Marey, E.J., 1873. Animal Mechanism: a Treatise on Terrestrial and Aerial Locomotion. Appleton, New York [republished as vol. XI of the International Scientific Series].

Murray, M.P., 1967. Gait as a total pattern of movement. Am J Phys Med 40, 290–333.

Muybridge, E., 1887. Animal locomotion. In: Brown, L.S. (Ed.), 1957, Animals in Motion. Dover, New York.

Muybridge, E., 1901. The Human Figure in Motion. Chapman & Hall, London.

Patrick, J., 1991. Gait laboratory investigations to assist decision making. Br J Hosp Med 45, 35–37.

Perry, J., 2010. Gait Analysis: Normal and Pathological Function. SLACK Inc., Thorofare, NJ.

Peterson, W.A., Brookhart, J.M., Stone, S.A., 1965. A strain-gage platform for force measurements. J Appl Physiol 20, 1095–1097.

Pomeroy, V.M., Pramanik, A., Sykes, L., et al., 2003. Agreement between physiotherapists on quality of movement rated via videotape. Clin Rehabil 17 (3), 264–272.

Rafferty, D., Bell, F., 1995. Gait analysis – a semiautomated approach. Gait Posture 3 (3), 184.

Richards, J., 2008. Biomechanics in Clinic and Research: An Interactive Teaching and Learning Course. Churchill Livingstone, London.

Rose, J., Gamble, J.G., 2005. Human Walking. Williams & Wilkins, Baltimore.

Saunders, J.B.D.M., Inman, V.T., Eberhart, H.S., 1953. The major determinants in normal and pathological gait. J Bone Joint Surg 35A, 543–558.

Wall, J.C., Charteris, J., Turnbull, G., 1987. Two steps equals one stride equals what? Clin Biomech 2, 119–125.

Chapter 16

Sports management

Hilary Pape

INTRODUCTION

This chapter is aimed at the physiotherapist wishing to work in the field of sports. It sets out to highlight the key skills and knowledge base needed. Traditionally, physiotherapists wanting to work in sports medicine would consolidate the theory and practical skills learned in university, under the supervision of senior clinical staff, before embarking on a sporting career. For many recent graduates this has become impossible owing to the paucity of traditional National Health Service (NHS) jobs. This chapter aims to give inexperienced physiotherapists the necessary background knowledge to work confidently and safely at pitch, court or track side, and the rehabilitation setting. For the purposes of brevity the term 'pitch-side' will be used throughout to refer to any incident occurring acutely in the competition or training setting.

Sports medicine is a broad, complex branch of healthcare encompassing several disciplines (Anderson 2003). The physiotherapist working in sports needs a detailed knowledge of the anatomy and physiology of neuromusculoskeletal systems, an understanding of other systems, such as the cardiovascular and respiratory systems, and the body's response to exercise. It is vital to understand the principles of first aid and acute injury management. A working knowledge of the relevant sport, including the psychological and physiological demands of that sport, the mechanism of commonly sustained injuries and the rules and regulations, is important. The legal considerations, standards, and duty of care and negligence cannot be ignored.

It should be noted that there are many excellent texts centred around sports medicine and first aid. When sourcing literature take care to ensure the guidance and advice, particularly around legalities, is pertinent to the country of application.

The author's experience has been developed principally in the sport of rugby league, therefore many of the examples given are taken from this sport.

QUALIFICATIONS, SKILLS AND RELEVANT CONTINUING PROFESSIONAL DEVELOPMENT

It could be argued that the physiotherapist working pitch-side is principally a first aider. First aid is a skill in itself. It has been defined as the assistance given to a casualty immediately following injury or illness to maintain life and/or prevent the problem getting worse until the casualty can receive medical attention.

The Health and Safety (First-Aid) Regulations (1981) require employers to provide adequate and appropriate equipment, facilities and personnel to enable first aid to be given to employees if they are injured or become ill at work (HSE 2009). The Chartered Society of Physiotherapy (CSP) service standard 16.2 (CSP 2005) states that all physiotherapy staff involved in providing physiotherapy services should receive training in the following:

- fire procedures;
- resuscitation;
- moving and handling;
- dealing with violence and aggression;
- infection control.

However, the legislation around first aid on pitch-side is unclear. The Resuscitation and Emergency Management Onfield (REMO) course (see Table 16.1), based in England, was established in 2001 when the British Olympic Association (BOA) and the United Kingdom Association of Doctors in Sport commissioned the development of an immediate medical care and resuscitation course tailored specifically for doctors and physiotherapists working in sport. The REMO course is now a compulsory qualification for UK doctors working in sports and exercise medicine (Johnson 2010). Advanced Resuscitation and Emergency Aid (AREA) was commissioned by the Football Association (FA) in 2008 and has now become a mandatory qualification for all medical staff working pitch-side in the Premier League (Johnson 2010). The FA

Table 16.1 Where to get help

The Association of Chartered Physiotherapists In Sports Medicine (ACPSM)	http://www.acpsm.org
Sports Massage Association	http://www.thesma.org/
Chartered physiotherapists interested in massage and soft tissue therapies	http://www.csp.org.uk/professional-networks/cpmastt
British Association of Sports Exercise Medicine (BASEM)	http://www.basem.co.uk/article.php?id=331&catid=43
Sport England	http://www.sportengland.org/
English Institute of Sport	http://www.eis2win.co.uk/pages/default.aspx
UK Sport	http://www.uksport.gov.uk/
The Physician and Sports Medicine	http://www.physsportsmed.com/
CogState Sport	http://www.cogstate.com/go/sport/sport-world
100% Me	http://www.ukad.org.uk/pages/100me
World Anti-Doping Agency WADA	http://www.wada-ama.org/
'Green Book' on vaccines by Department of Health	http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_079917
Therapeutic Use Exemptions	http://www.uksport.gov.uk/pages/therapeutic_use_exemptions/ http://www.wada-ama.org/en/Science-Medicine/TUE/
Health and Safety Executive approved first aid courses	http://www.hse.gov.uk/firstaid/training.htm
The National Sports First Aid Course	http://www.sportsmedicinecentre.org/scottish_football.cfm?curpageid=746
Resuscitation Council	http://www.resus.org.uk/siteindx.htm
Resuscitation and Emergency Management Onfield	http://www.remosports.com/

also suggests a hierarchy of medical care where there must be a doctor and Health and Care Professions Council (HCPC)-registered physiotherapist present, including at grassroots levels where, as an acceptable minimum standard, there should be a person in attendance at every match and training session who is available to deliver emergency and first aid (FA 2010). It is advisable then, that any health professional working in this area does not solely rely on their physiotherapy training, but also obtains a first aid qualification. Many sports' governing bodies recommend their own specific first aid courses. The FA provide the FA Emergency Aid Training Certificate and FA/1st4Sport First Aid for Sport Certificate (FA 2010). If working at a professional level in rugby league you are required to hold the Immediate Management on Field of Play (IMMOF) qualification. The Rugby Football League is currently working with the FA on a new first aid policy and training programme for use in amateur rugby league and football.

By legal definition, correct first aid is that which is approved by the voluntary aid societies for publication in their manuals where this is used in training of a first aider (Dunbar 2006). Therefore, it is recommended that at amateur level at least, a basic first aid course should be undertaken. The National Sports First Aid course (see Table 16.1), developed and delivered by the Faculty of Sports and Exercise Medicine, is recommended by the Association of Chartered Physiotherapists in Sports Medicine.

Knowledge and skills needed

Physiotherapists aiming to work in sport should consider the following:

- a working knowledge of the chosen sport;
- continuing professional development (CPD) in the field of emergency medicine and sports medicine/physiotherapy relevant to the role at the club;
- legal responsibilities;
- appropriate medical insurance, (check with the CSP for individual cover);
- a working knowledge of World Anti-Doping Agency (WADA) regulations (see Table 16.1);
- a working knowledge of concussion management.

Knowledge of sports

A working knowledge of the sport you are working in is not essential but gives you invaluable insight which will prevent mistakes being made that may impact on the players, as well as on your ability to do your job. Think about common mechanisms of injury (MOI) and gain an understanding of the forces involved in your sport. This can be invaluable if you do see the injury as it happens. It helps to understand the physical needs and demands on the athletes – this can affect how busy you are likely to be on match day and what kit you always carry with you. In contact sports, such as rugby or American football, fractures, dislocations and lacerations are more common than in swimming, for example. Aim to have a good understanding of the rules and bylaws of the sport – in sports such as Judo you are not allowed to enter the mat or touch a player in competition until the umpire has given you permission or the player is disqualified. In many sports, such as rugby league, you are given unconditional access to the field of play in order to attend injuries. Knowledge of the substitution rules can assist you to work with the players and coaches to best serve everybody's interests.

CPD

As with any area of clinical practice, practitioners must maintain their CPD. In the sporting setting this may include first aid training, which may extend to advanced trauma management and advanced life support, to advanced management of musculoskeletal injuries and acute injury, to medico-legal training, etc. Professional sporting requirements generally require a minimum of five years post-qualification and Masters level postgraduate training. At the time of writing this chapter, the preparations for the 2012 Olympic Games are in full swing. Physiotherapists wanting to participate in events such as the Olympic Games, the Commonwealth and World University Games, or to work with elite athletes need to be HCPC-registered physiotherapists and preferably members of the CSP, hold a degree or graduate diploma in physiotherapy, have a minimum of five years post-registration experience and usually at least two years experience in training and competitive environments. Previous experience of working at major games and multi-sport experience, and active involvement with British Universities and Colleges Sports Association (BUCS) for student sport, is not usually essential but considered an advantage.

Applicants are usually asked to provide proof of their own CPD in the sports medicine fields, including evidence of regular cardiopulmonary resuscitation (CPR) training. A massage therapy qualification from a recognised Sports Massage Association (SMA) course and a diploma or MSc in Sports Physiotherapy is often considered an advantage. It is recommended that physiotherapists join appropriate special interest groups. For those working in sport the Association of Chartered Physiotherapists in Sports Medicine (ACPSM) provides an excellent support service, a well-structured CPD programme, mentoring, a quarterly journal, conferences and advanced notice of employment and voluntary positions in sport. Evidence of working towards, or having achieved, ACPSM silver level CPD standards are usually regarded favourably when applying to work at this level. It is worth mentioning at this point that many of these positions are voluntary or only provide an honorarium, with applicants expected to fund themselves and find their own accommodation. Applicants are usually required to commit to a minimum period of two weeks service when working for the host city at a major games such as the Olympic Games in London in 2012. However, working as part of Team England or Team GB, or for BUCS at a World University Games, all accommodation, travel and food would be paid for.

Physiotherapists wanting to work in sport should consider CPD in the following fields:

- Pre-participation Physical Evaluation (PPE);
- advanced first aid and trauma management including:
  - concussion management (see later discussion);
  - on-field recognition of injuries;
  - fracture and dislocation management;
  - wound management;
  - assessment of return to play protocols;
  - thermoregulation;
  - hydration/nutrition.

Legal responsibilities

When contracted by a team or athlete as the physiotherapist or first aider you have assumed a duty of care (Dunbar 2006). As long as you work within your scope of practice and practise first aid skills in accordance with accepted first aid practice, it is unlikely that a civil action for alleged negligence would succeed (Dunbar 2006). It is worth noting that if you are 'employed' as opposed to working as a volunteer, the club may be liable in any litigation proceedings.

It is important that physiotherapists working with children (those under the age of 18) obtain an enhanced Criminal Records Bureau (CRB) check (free for those in voluntary positions) from the club at which you will work. If you are being remunerated for your work you may have to pay for your own CRB check. The majority of sport national governing bodies (NGB) in the UK have embraced child protection and safeguarding policies and further embedded them at club level through accreditation schemes such as Clubmark (Sport England 2011). It is advised that you check with your club in the first instance, but if you do require further support and information contact the NGB directly. According to Dunbar (2006), provision for sport is subject to Health and Safety Executive (HSE) regulation and, as such, physiotherapists working in sport should comply with the relevant safety

and reporting procedures. Serious accidents occurring to athletes and/or first aiders are regarded as 'notifiable'.

Medical insurance

Membership of the CSP will normally provide sufficient professional and public liability insurance cover to work in amateur sport. However, if diversifying into working with elite athletes the limit of liability may not provide enough cover. If working with racing animals, such as horses and greyhounds, then it is advisable to contact the CSP for further advice as the scope of activities insured excludes animal or veterinary physiotherapy (CSP 2010).

Doping, and WADA and TUEs

The WADA promotes, coordinates and monitors doping in sport. The WADA's responsibilities in science and medicine include, among others, scientific research, the prohibited list, the accreditation of anti-doping laboratories and therapeutic use exemptions (TUEs). Athletes may have illnesses or conditions that require them to take medications. If the medication an athlete is required to take happens to be on the prohibited list, a TUE may give that athlete the authorisation to take the needed medicine. The purpose of the International Standard for Therapeutic Use Exemptions (ISTUE) is to ensure that the process of granting TUEs is harmonised across sports and countries (WADA 2010).

The rules and regulations surrounding drug use prescribed or otherwise are complicated and may seem draconian. The team doctor would normally deal with TUEs at the elite level. Physiotherapists working in high level sports should be extremely careful 'prescribing' any 'medication' (Taylor 2008). If in doubt, athletes are advised not to use anything no matter how innocuous it may seem. More information can be found on the UK Sports, WADA and the '100% ME' websites (see Table 16.1).

Concussion management

Concussion or traumatic brain injury (TBI) is common in sports. About 90% are mild in nature and are referred to as mild TBIs (mTBI) but obviously all TBIs have the potential to develop serious complications (Solomon et al. 2006). The following discussion provides an overview only and should be supported by further reading and attendance on trauma management courses.

There appears to be no clear definition of concussion. However, the Concussion in Sport (CIS) Group at the International Conference on Concussion in Sport in Prague (2004) offered the complex definition below (McRory et al. 2005). This CIS definition represents a consensus of opinion from experts in the sports medicine field and is recommended as the most current (Solomon et al. 2006):

> *Sports concussion is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. Several common features that incorporate clinical, pathologic and biomechanical injury constructs that may be utilised in defining the nature of a concussive head injury include:*
>
> 1. *Concussion may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an 'impulsive' force transmitted to the head.*
> 2. *Concussion typically results in the rapid onset of short lived impairment of neurologic function that resolves spontaneously.*
> 3. *Concussion may result in neuropathological changes but the acute clinical symptoms largely reflect a functional disturbance rather than structural injury.*
> 4. *Concussion results in a graded set of clinical syndromes that may or may not involve loss of consciousness. Resolution of the clinical and cognitive symptoms typically follows a sequential course.*
> 5. *Concussion is typically associated with grossly normal structural neuro imaging studies.*

The review also advised that in many cases post-concussive symptoms may be prolonged or persistent (McRory et al. 2005).

Clinical note

The signs and symptoms of concussion are varied and include:

- confusion;
- amnesia;
- headache;
- dizziness;
- ringing in the ears;
- nausea or vomiting;
- slurred speech;
- perseveration;
- fatigue;
- memory or concentration problems;
- sensitivity to light and noise;
- sleep disturbances;
- irritability;
- depression.

In children and other patients who find it difficult to communicate take note of listlessness, tiring easily, a change in eating or sleeping patterns, lack of interest in

toys and loss of balance or unsteady walking. Not all of the above symptoms may be present, and may develop at different stages. Loss of consciousness (LOC) has traditionally been viewed as the most significant symptom in the diagnosis and determination of severity of concussion. LOC requires immediate medical attention to ensure a patent airway and avoid cardio-respiratory failure until such is ruled out or the casualty regains consciousness. However, it is now thought that retrograde (before) or antegrade (after) amnesia is of more significance. Should LOC occur post-traumatic incident (as opposed to sudden collapse or syncope) it is advisable to assume spinal injury until such time as this can be ruled out. The casualty should be managed appropriately (log rolling, etc.) unless the airway is compromised. Again, the reader is advised to complete first aid and trauma management training to develop the necessary skills to manage the unconscious casualty.

Assessment of concussion involves recognising the above symptoms, which may be difficult if the symptoms are mild. There are several pitch-side assessment strategies and neuropsychological tools that can be used to assess concussion. Many of these tests, to some degree, rely on a pre-season baseline test having being performed, for example Post-Concussion Scale – Revised (PCS-R) or Head Injury Scale (HIS) (Solomon et al. 2006). The tests are then rerun post-suspected concussion and the scores compared. Maddock's Questions are commonly used to assess players in the field of play. Developed in 1995 by Dr D.L. Maddocks and colleagues these are a set of standardised questions that can be adapted to particular sports and are a qualitative measure of the neuropsychological state of a player having sustained a head injury. Questions include:

- Which field are we at?
- Which team are we playing today?
- Who is your opponent at present?
- Which half/period is it?
- How far into the half is it?
- Which side scored the last touchdown/goal/point?
- Which team did we play last week?
- Did we win last week?

The athlete's ability to answer the questions, even if the answer is correct, should alert the first aider to the possibility of concussion.

CogState Sport is a well-recognised computerised concussion test and management system for use by professional, elite and amateur athletes all over the world. CogState Sport is proven sensitive to mild cognitive changes and helps guide medical decisions about return to activity and rehabilitation. Athletes take a baseline test before the season begins (or when uninjured). Testing computers do not need to be connected to the internet for testing. Once the tests are finished, they are submitted, via the internet, to CogState, using a password-protected data transfer system. A report will then be sent back by email as to whether the baseline test was satisfactory or whether the athlete needs to take another test. Disadvantages include time and the need for equipment to carry out the baseline tests. There are recognised paper-based and sideline tests available which can be performed as a baseline test preseason then used pitch-side to monitor a player's progress. The CogState Sport website is worth a visit (see Table 16.1) and provides invaluable advice, information and guidance on concussion and its management for anyone working pitch-side. While preseason testing occurs routinely in professional and elite sports, it is unlikely to occur at amateur level. The principal reasons for this may be lack of facilities and staff. It is likely that the physiotherapist working at amateur level will be the sole medical practitioner. It is difficult to monitor players carrying out the neuropsychological tests while trying to keep an eye on the game in process. In addition, in professional and elite sports you have a captive audience in that these measures are a necessary and recognised part of competing at high level sports and often form part of contractual agreements of playing for a club. In the amateur setting, athletes may not recognise the importance of baseline testing and therefore refuse to participate. It is worth pursuing, however, as we attempt to raise standards of care in sports medicine.

As with the definition of concussion, there is little consensus on when concussed players can return to play. Medical treatment for concussion comprises principally of rest (McRory 2001a). The aim of concussion management, therefore, is to prevent the athlete returning to sport before their brain has recovered. Many sports governing bodies will provide their own guidelines on return to play – particularly at professional level. Usually, this decision will be the responsibility of the club doctor. At amateur level where the onus may fall on the physiotherapist the main consensus seems to be that no player should be allowed to return to the field of play until all the symptoms of concussion have resolved. This may take several days (McRory et al. 2005; Solomon et al. 2006). It should be remembered that there is some evidence that TBI is cumulative and much of the research shows that a player's risk of sustaining a concussion increases once they have already had one (Solomon et al. 2006). Players sustaining repeated head injuries throughout a season may suffer moderate cognitive changes and there is some evidence to suggest there is a relationship between repeated concussion and Alzheimer's disease and depression. Where there is consensus within concussion management it is that any child sustaining a suspected concussion must be referred to hospital.

Second impact syndrome

Second impact syndrome (SIS) has been defined as a syndrome occurring when an athlete sustains a second head injury before the symptoms of the initial injury have

resolved. It is thought to be a disruption in the autoregulation of the brain leading to oedema and an increase in intracranial pressure causing herniation of the brain and, ultimately, death (Solomon et al. 2006). Although SIS is well documented in the literature, Dr Paul McRory, a renowned neurologist specialising in sport and concussion, has questioned the existence of SIS. McRory (2001b) suggests the evidence for such SIS is not compelling and that SIS is more likely to be a clinical condition representing diffuse cerebral swelling. He suggests that clinicians abandon the term 'second impact syndrome' and refer to the syndrome as 'diffuse cerebral swelling'. Despite the lack of a clear consensus, most authors agree that no player should be allowed to return to play until all symptoms have resolved (Solomon et al. 2006).

Summary

Anyone working pitch-side should aim to increase their awareness of the recognition, assessment and management of concussion. Most basic first aid courses will address concussion but more advanced trauma management courses will give a more comprehensive knowledge base. Readers are directed to Solomon et al. (2006) and the CogState Sport website for further information on the recognition, assessment and management of concussion.

ROLE OF THE PHYSIOTHERAPIST IN SPORT

It is worth considering what the role of the physiotherapist is in preparation for competition/games and pitch-side. If you are in a position to be offering rehabilitation for players in between competition you will be likely working in a similar situation to that of musculoskeletal outpatients taking a detailed subjective and objective history, designing an appropriate management plan with the aim of facilitating players return to play. At pitch-side and on competition day the role is more complex. Physiotherapists working for professional clubs are likely to be part of a multi-disciplinary team of what is commonly known as 'backroom staff'. This team will include the coach plus assistants. There may be a dedicated kit man and statistics recorder and team doctor. This means you will have a team to rely on and help you out with carrying equipment and, possibly, match preparation. In the amateur setting this is less likely and experience has demonstrated a need to think outside your normal scope of practice. You may find you take on an almost parental role and become responsible for remembering sub suits for players sitting on the bench at pitch-side when the weather turns cold, sun cream when its hot and stud keys, etc.

In the professional game you are likely to have a clearly defined role limited to pre-match preparation, for example strapping and pitch-side first aid. In the amateur setting you may find yourself being asked to deal with spectators and their families, and even the match officials. You will need to make the decision about how to handle such requests but it is worth remembering that your first responsibility is to the players. If you are being asked to deal with an incident off pitch, inform the referees/umpires and get play stopped.

Key preseason considerations

As well as the skills and knowledge discussed earlier there are some key preseason considerations to be aware of. Before embarking on pitch-side it is advisable to consider your own health and protection. Ensure you have received the appropriate vaccinations and immunisations as for any healthcare worker, for example tetanus and hepatitis B. Pitch-side first aiders should aim to adopt the same standard precautions that all healthcare workers do. Table 16.1 contains a website address for the Green Book on vaccines produced by the Department of Health which gives the most up-to-date advice. However, you can visit your general practitioner (GP) for further advice. All therapists working pitch-side or otherwise are referred to the epic2 guidelines for preventing healthcare-associated infections (Pratt et al. 2007); these are available from the Department of Health website at: www.doh.gov.uk/HAI and the Hospital Infection Society website at: www.his.org.uk. These contain standard principles for preventing infections and should be adopted by all healthcare workers coming into contact with patients' blood or bodily fluids. They are a set of principles designed to minimise exposure to and transmission of a wide variety of microorganisms. On pitch-side ensure you wear suitable personal protective equipment, for example gloves or face protection depending on the sport you are working with and the risks involved (e.g. bodily fluids – blood, sweat, saliva). In cold weather you are likely to be well covered but be aware in warm weather or indoor sports that you may have exposed skin and are therefore at risk of splashes and inoculation injuries. You may choose to wear goggles and protective clothing of some kind depending on the sport you are working with and the risks involved.

If dealing with children check that your enhanced CRB and first aid certificates are current.

You may need or desire to carry out preseason baseline preparation checks on athletes. As discussed in the section on concussion, this may not be appropriate or be difficult to implement. However, it is good practice if it can be done. Aim to get to know all the members of your squad and gain a basic medical history so that you know who is asthmatic or who has allergies or other pre-existing conditions. Ensure you know who to contact in case of emergencies, etc. You may need to perform an inventory of what equipment is available to you and decide if you need to order more supplies. What equipment you may need is discussed later in the chapter.

Consider your own match-day clothing. Do you need studded boots or waterproofs, including trousers? You may get some team kit supplied, although it may not fit. You may be expected to travel and work in a different kit from what you wear post-match, especially when working at a professional level.

Sort out your lines of communication – this is often a problem, particularly in the amateur setting. Club staff often forget that the physiotherapist needs to know what time everybody is meeting and what time the team bus is leaving, etc. Obtain as many telephone numbers and email addresses as possible, including coaching staff, players and other backroom staff.

Finally, you will hopefully be getting paid for your services. Be warned that pay, particularly at amateur level, is not usually high. You will generally find at a professional level you will be salaried but you are often expected to work long, unsociable hours. Most competitive sports occur at weekends and evenings: you will be expected to be there. In amateur sports, clubs will generally ask you what you want to be paid but are rarely able to pay the same hourly rate afforded to NHS staff. When negotiating your pay ensure the club recognises that when playing away fixtures, you should be paid from the minute the team bus sets off to when it arrives back and not just for the period you are pitch-side. It is usual, however, for clubs to agree a set fee for games and a set fee for rehabilitation nights. Some sports have more expendable income than others. You will need to use your discretion and be realistic, but try not to sell yourself or your profession short.

Key pre-match considerations

These are particularly important for away games/competitions. You need to ensure you take everything with you and that you are prepared for almost every eventuality. Much of this comes with experience, but experience dictates if you do not have it with you you'll need it!

Make sure you have discussed with the coaching staff how long you are likely to need for pre-match preparation. Most teams/athletes will require a warm-up. All these factors need consideration, along with travel time, to ensure the departure time is early enough.

If travelling away, ensure the medical kit is on the team bus. This means if the bus is caught up in traffic congestion you can begin to strap on the bus. Obviously, if using scissors wait until the bus is stationary!

Pitch-side

Pitch-side equipment

The following is a possible list of contents for a pitch-side medical bag. It does not represent an exhaustive list and is taken from a collection of sources and personal experience. The contents of your medical kit may be dictated by the sport(s) you are involved in and may continue to be revised and updated in response to incidents that occur.

- Crepe bandages/Tubigrip.
- Semi-compressed chiropody felt – useful for all sorts of things, including as an adjunct to compression bandaging.
- Elastic adhesive bandage (EAB) (various sizes).
- Zinc oxide tape (inelastic) (various sizes).
- Disposable nitrile gloves – if working in hot weather carry a size larger than normal as they will be easier to put on and take off.
- Gauze swabs for mopping up blood, cleaning wounds. Consider disposal carefully.
- Plasters (non-allergenic/waterproof).
- Sterile, non-adherent wound dressings (various sizes).
- Nasal plugs.
- Triangular bandages.
- Sterile eye packs/physiological saline for irrigation.
- Milk sachets/pots for storing lost teeth.
- Petroleum jelly.
- Massage mediums.
- Nasal decongestants-beware TUE regulations.
- Scissors (safety type).
- Nail clippers.
- Blanket – wool/space (foil).
- Small towel.
- Safety pins.
- Splints.
- Neck collar – various sizes – these need to be applied correctly. Ensure you have appropriate training.
- A pocket mask or resuscitation mask/bag-mask-valve/airway (only to be used by trained individuals).
- Water bottle.
- Ice/cryotherapy equipment.
- Mobile phone.
- Stretcher/spinal board – spinal boards are only safe to use if you have a competent team who are well practised in spinal boarding. You may be as well using manual immobilisation and wait for paramedics in suspected spinal injury.
- Insulation tape.
- TBCo (Friar's balsam) – this is excellent stuff and is used as pre-strapping/steristrips to ensure adherence of tape. Avoid contact with eyes and mucous membranes.
- Cotton buds.
- Paracetamol.
- Toilet roll.
- List of players/contact details.
- Advice sheets for head injuries, etc.

PRINCIPLES OF FIRST AID

The following discussion is an overview only. It is not intended to replace attendance on a first aid or trauma

management course but highlights the main principles of pitch-side first aid.

Key points

The key concept is to maintain life and/or prevent the injury getting worse. Your skills as a physiotherapist, for example your anatomical and physiological knowledge, are invaluable, particularly in the rehabilitation setting but essentially at pitch-side you will be required to apply the principles of first aid:

- *assess* the situation quickly and safely and summon appropriate help;
- *protect* the casualty and others from danger;
- *identify* (where possible) the nature of the problem;
- *prioritise* the casualties;
- give *early* and *appropriate* treatment.

Pitch-side assessment is different from a normal musculoskeletal assessment in that the aim is less about obtaining a diagnosis and more about determining whether a player needs to be removed from the field of play. Obviously, if a player had sustained a TBI they may have lost consciousness and their airway may be compromised necessitating on-field emergency management. A limb injury may initially seem serious to you and the player if you rely solely on pain as a guide. When you arrive at the scene do not be in a hurry to act. Unless you suspect airway compromise stop and ask the player what has happened. Do not assume anything: ask them where it hurts. You would be surprised how many players will be clutching a non-injured limb. If you did not see the mechanisms of injury, try and ascertain it to give you an idea of the forces involved. These questions also have the advantage of allowing the player time to calm down and acute pain time to settle. Many ligament tests will be difficult at the scene owing to pain and muscle spasm. If the rules allow it, remove the player from the field and assess at pitch-side so you are not under pressure from referees or umpires. It is often more appropriate to apply the PRICEM (protection, rest, ice, compression, elevation, mobilisation/movement) principles (see below), then assess after a period of time to allow the initial symptoms to settle. It is not unknown for players to be removed from the field and then to find their injury was not as serious as first thought. Put some thought into how to remove a player from the field. If they can, let them get up and hop/limp off under their own steam or with help. Protect you own back, use other players or bystanders if you are a small physiotherapist dealing with a large player. If you suspect serious fractures, such as femoral or spinal, you may need to have the player stretchered off. In the professional setting there should be a trained team (possibly including the physiotherapist) to do this. At amateur level you may be on your own. It is not advisable to spinal board a player without a practised team and appropriate training. In these cases immobilise manually and call an ambulance, no matter how long this takes. If you suspect a limb fracture you may choose to immobilise and send the player to accident and emergency. It is a good idea to familiarise yourself with the OTTAWA rules. These are a set of well documented guidelines to decide if a patient with foot, ankle or knee pain should be offered X-rays to diagnose a possible fracture. Many unnecessary X-rays are taken, which can be costly, time-consuming and pose a possible health risk. If you suspect soft tissue injury you may only be able to ice, elevate and immobilise until after the match, especially if you are working solo.

Practicalities of injury management

PRICEM and the principles of soft tissue injury management in relation to inflammation and healing are covered in Chapter 12. However, it is worth discussing some of the practical applications of these principles at this point.

For more details on PRICEM readers are directed to Chapter 12 and the PRICEM guidelines produced by the ACPSM (see Table 16.1) for further reading.

Protection

It is a good idea to keep a selection of braces and crutches to offer local protection and rest. This avoids the need for adhesive strapping which players may remove, hence losing the benefit. Although not usually a problem in professional sports, at amateur level where finances are tight, you may have to be inventive. It is usual to dispose of knee braces, etc. given to NHS patients. You could consider keeping them and washing and re-using them on other players (unless they are soiled enough to pose a significant infection risk). Many athletes require strapping every match. Use of semi-rigid braces which can be used again can save a lot of money. Wash them in as hot a wash as possible after each match as sweat and dirt will degrade them.

Rest

Experience shows that many injured players will keep 'testing' the injury constantly, breaking fragile fibrin bonds as they try and repair the injury. Using crutches/braces as above, encourages the athletes to stop and rest.

Ice

The evidence base suggests that wet ice (e.g. a wet towel ice bag) applied to soft tissue injuries is most effective (Bleakley and MacAuley 2007). Immersion is useful for non-uniform areas such as hands. However, while

efficacious, these methods may be impractical in certain settings. If injured athletes are about to embark on a long coach trip home from an away game it might be prudent to use sealed methods of cryotherapy, such as Cryocuff™ to avoid spills and wet seats. Although these systems do not appear to cool the tissues as effectively, they do add some compression. Recent evidence suggests that compression and ice may be more effective in limiting oedema than ice alone (Bleakley and MacAuley 2007) and that ice is more important in reducing the effects of secondary cell death owing to tissue hypoxia (Merrick 2002).

Compression

The evidence for the efficacy of crepe bandages versus Tubigrip in the prevention of oedema is not yet conclusive. Until there is a consensus the author recommends Tubigrip. It is easier to apply safely and patients can take it off and re apply as necessary. Tubigrip can also be used for protection. Several sizes will be required.

Elevation

Achieving elevation at pitch-side and when travelling home can prove a logistical challenge. Consider using kit bags, spare clothing, support staff and even supporters to support lower limbs. The author has a large medical kit bag containing a small folding stool. Upper limbs can be held by the uninjured arm or fold the player's shirt up over the injured arm to achieve moderate elevation rather than applying complicated triangular bandage slings. If higher level elevation is required (for example to stop palmer bleeding) lie the player down where possible and use a bystander to maintain the injured limb's position while applying compression.

Mobilisation/movement

Be aware of the inflammatory processes, tissue repair and healing (review Chapter 12). Give appropriate advice to the players about suitable levels of activity before you see them in the rehabilitation setting. If this is not possible give appropriate advice about PRICEM self-management. Suggest the player visits their GP for referral to physiotherapy and to obtain a sick note if necessary. Keep a list of local private physiotherapists that you can refer your players to. Many private practitioners will offer reduced rates to clubs if they are guaranteed a 'regular supply' of patients.

Summary

Pitch-side physiotherapy is principally a first aid role. Ensure you attend a recognised first aid course to supplement your physiotherapy training. Consider attending an advanced trauma management course. This will give you more confidence in dealing with serious situations and injuries.

ROLE OF THE TEAM DOCTOR

At this point it is appropriate to consider the role of the doctor in sports. This will largely depend on the setting in which you are working. In the amateur setting the doctor is likely to be a volunteer – possibly a spectator, supporter or player willing to help out. However, in the professional setting it can become a little more regimented but may include:

- medical care of players;
- pitch-side management of injuries in conjunction with the physiotherapist. The doctor will usually take a back seat until needed;
- secondary referral of injured players to specialist services;
- ensuring the medical needs of the players are met through monitoring and evaluation, including prophylactic medical screening/pre-participation screening;
- TUEs – see earlier discussion;
- post-concussion return to play tests;
- identification, sourcing and distribution of medical equipment;
- oxygen and emergency drug box care;
- the care of the away team;
- keeping up-to-date medical records;
- liaising with other medical staff relevant to the players' welfare for international or representative duty;
- providing in-service/CPD training for the medical team;
- working within the guidelines of medical care and confidentiality as currently set out by the BOA;
- working closely with coaching and management staff.

It should be noted that the team doctor will *not* be responsible for crowd cover. Separate medical cover for the crowd should be provided by the venue.

Summary

The author summarises the role of the physiotherapist in team sports as six 'knows':

1. *Know your sport*. Aim to research common injury patterns and mechanisms of injury. Understand how player positions may lead to predisposition to certain injury patterns. Familiarise yourself with the rules and bylaws of your sport.
2. *Know your players*. Get to know your athletes – their age, medical history and any predisposing factors to injury. Learn whether they suffer from repeat injuries, such as ankle sprains. You may be able to influence this. Get to know their superstitions. While this may not seem important, an awareness and

tolerance of their superstitions, such as not wanting to be strapped until the last minute or will not wear yellow insulation tape, can make for smooth running of pre-match preparations.

3. *Know your coaching staff*. This is particularly important. If you can gain the confidence of the coaching staff they will support you in dealing with difficult players and support you when you ask for equipment. Learn their particular ways of working. Have they had experience of working with a HCPC-registered physiotherapist before? Experience suggests many coaches are used to volunteer 'sponge men'. Coaching staff may not understand your refusal to allow a player back on the pitch after a TBI, even when they appear to have recovered. One suggestion is to put together a manual or handbook for the players and coaching staff explaining your management of common injuries. The team can read this in their own time and concentrate on your rationale without the distractions and concerns of match day. Discuss how long is needed for pre-match warm-up. Explain you cannot effectively strap a sweating player so you need enough time for pre-match strapping before the warm-up.
4. *Know the match officials*. Again, this might not seem important but if you remain professional and helpful to match officials it can only benefit your team and will ensure they support you in any major incidents.
5. *Know your equipment/facilities*. It is important never to assume anything when travelling to an away fixture. Check travel and weather reports, particularly if you are making your own way to a fixture. Check what equipment you will be provided with in the away changing rooms. You may not be provided with a treatment couch and may always choose to take your own portable couch. It is a good idea to take an ice box pre-filled with ice in case the club you are visiting does not have any, or enough. Many amateur clubs provide few, if any, facilities for mixed gender changing. Female physiotherapists may find they have to get changed after the game in the ladies toilets. Carry wet wipes so you can freshen up. On arrival check where the nearest accident and emergency facilities are, and access to the pitch for an ambulance. Basically, be prepared for nothing and everything!
6. *Know your stuff*. This refers to your CPD, first aid training combined with your physiotherapy training. The CSP and HCPC standards mean that working, even in the amateur sports setting, means you need to practise evidence-based medicine. Regular CPD in any form will give you the confidence and the knowledge to practise safely and competently in an environment where you may be the sole medical practitioner.

Working in sport is a rewarding experience. It tends to be slightly more relaxed than traditional NHS environments. It can give the physiotherapist scope to work independently and devise individual ways of working. It can give rise to many opportunities, such as representing your city, county or country, travel and making new friends and colleagues. Ensure you maintain the professional standards that you signed up for when you obtained your licence to practise and you will find the sporting environment one of the most rewarding experiences of your professional career.

ACKNOWLEDGEMENTS

The author would like to acknowledge and thank Stephen Fairhurst, safeguarding consultant; Lynn Booth, MSc, MCSP, Clinical Lead - Physical Therapies Service, London Organising Committee of the Olympic Games and Paralympic Games and Andrea Denton RGN, MA, MSc, infection prevention and control nurse.

FURTHER READING

Andreasen, J.O., Andreasen, F.M. (Eds.), 2003. Traumatic Dental Injuries: A Manual. Blackwell Munksgaard, Oxford.

Bahr, R., Mæhlum, S., 2004. Clinical Guide To Sports Injuries. Human Kinetics, Leeds.

Boxill, J., 2003. Sports Ethics: An Anthology. Blackwell Publishing, Oxford.

Cerny, F.J., Burton, H.W., 2001. Exercise Physiology for Health Care Professionals. Human Kinetics, Leeds.

Eustace, S.J., Johnston, C., O'Byrne, J., et al., 2007. Sports Injuries: Examination, Imaging and Management. Churchill Livingstone, Edinburgh.

Fleck, S.J., Kraemer, W.J., 2004. Designing Resistance Training

Programs, third ed. Human Kinetics, Leeds.
Gardiner, S., James, M., O'Leary, J., et al., 2006. Sports Law. Cavendish, London.
Hodgetts, T.J., Turner, L. (Eds.), 2008. Trauma Rules 2 – Incorporating Military Trauma Rules, second ed. BMJ Publishing Group, London.
Houghlum, P., 2005. Therapeutic Exercise for Musculoskeletal Injuries, second ed. Human Kinetics, Leeds, pp. 3–63.
Kraemer, W.J., Häkkinen, K. (Eds.), 2000. Handbook of Sports Medicine and Science: Strength Training for Sport. An IOC Medical Commission. Blackwell Science, Oxford.
Landry, G.L., Bernhardt, D.T., 2003. Essentials of Primary Care Sports Medicine. Human Kinetics, Leeds.
Morgan, W.J. (Ed.), 2007. Ethics in Sport. Human Kinetics, Leeds.
Spengler, J.O., Connaughton, D.P., Pittman, A.T., 2006. Risk Management in Sport and Recreation. Human Kinetics, Leeds.

REFERENCES

Anderson, M.K., 2003. Fundamentals of Sports Injury Management, second ed. Lippincott Williams & Wilkins, London.
Bleakley, C., MacAuley, D., 2007. What is the role of ice in soft tissue injury management? In: MacAuley, D., Best, T. (Eds.), Evidence-based Sports Medicine, second ed. BMJ Books, London.
CSP (Chartered Society of Physiotherapy), 2005. Service Standards of Physiotherapy Practice. CSP, London.
CSP (Chartered Society of Physiotherapy), 2010. Members' professional and public liability insurance (PLI) scheme policy document. CSP, http://www.csp.org.uk/director/members/practice/professionalliabilityinsurance.cfm; accessed 6 January 2011.
Dunbar, J. (Ed.), 2006. National Sports First Aid Course Handbook, third ed. Hampden – The National Stadium Sports Clinic, Glasgow.
FA (Football Association), 2010. Rules of The Association and Laws of the Game Season 2010–2011. FA, London.
HSE (Health and Safety Executive), 2009. First Aid at Work: The Health and Safety (First-Aid) Regulations 1981 http://www.hse.gov.uk/pubns/priced/l74.pdf, accessed January 2011.
Johnson, P., 2010. Doctors in Sport. J Med Dental Defence Union Scotland, Autumn, 14–15.
McRory, P., 2001a. New treatments for concussion: The next millennium beckons. Clin J Sport Med 11, 190–193.
McRory, P., 2001b. Does second impact syndrome exist? Clin J Sport Med 11 (3), 144–149.
McRory, P., Johnston, K., Meeuwisse, W., et al., 2005. Summary and Agreement Statement of the 2nd International Conference on Concussion in Sport, Prague 2004. Clin J Sport Med 15, 2.
Merrick, M.A., 2002. Secondary injury after musculoskeletal trauma: A review and update. J Athl Train 37 (2), 209–217.
Pratt, R.J., Pellowe, C.M., Wilson, J.A., et al., 2007. Epic 2: National evidence-based guidelines for preventing healthcare-associated infections in NHS hospitals in England. J Hosp Infect 65, S1–S64.
Solomon, G.S., Johnston, K.M., Lovell, M.R., 2006. The Heads-Up on Sport Concussion. Human Kinetics, Leeds.
Sport England, 2011. Available at: http://www.clubmark.org.uk/; accessed 6 January 2011.
Taylor, L., 2008. Feature: Revised TUE Rulings Explained, http://www.uksport.gov.uk/news/feature_revised_tue_rulings_explained/; accessed 3 December 2010.
WADA (World Anti-Doping Agency), 2010. Therapeutic Use Exemptions, http://www.wada-ama.org/en/Science-Medicine/TUE/; accessed 7 January 2011.

Chapter 17

Pain

Lester Jones, G. Lorimer Moseley and Catherine Carus (Case study development)

INTRODUCTION

Pain is a common and normal human experience. Pain helps us to learn to adopt protective behaviour when we are threatened and safe behaviour when our body has been injured. Pain usually seems like a reasonably predictable experience. In a normal state, receptors in the tissues of the body respond at reasonably predictable thresholds of stimulation. When they do respond, they initiate action potentials that travel along peripheral neurones into the spinal cord. Neurotransmitters released from these neurones often activate secondary neurones, which send action potentials up the spinal cord to the brain. The brain then evaluates this information. Often, pain is perceived in the tissues that were stimulated. This might seem simple, but it is not.

ABC Definition

The International Study for the Association of Pain defines pain as '…an unpleasant, sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage' (Merskey and Bogduk 1994).

There are several key parts of this definition.

- Firstly, although the nociceptive system is critical for detecting dangerous stimuli and alerting the brain, pain is not simply the transmission of noxious sensory information (nociception). Rather, pain has potentially profound cognitive and emotional influences, just like other perceptual states (even vision – think of your favourite visual illusion and notice that what you see is not necessarily an accurate reflection of the light that is hitting the light receptors on your retina).
- Secondly, pain is not a measure of tissue damage, which means that tissue can be injured but not painful, and painful but not injured.
- Thirdly, pain is about threat to body tissue, not about a particular mode of sensory input. This sets pain apart from other information that is sent from our tissues to our brain: the so-called somatic senses. It is this threat-specific quality that makes pain critical for protection and preservation of the body: pain seems to be the conscious component of a complex defence system.
- Finally, there are many unconscious components of this defence system, all of which can influence each other and influence pain. Thus, pain is a fundamentally conscious process, which is preceded and accompanied by a range of responses, most of which are not conscious. According to this model of pain, when someone is in pain, we can be sure that their brain is concluding that tissue is in danger and that they should take some sort of action to get the tissues out of danger (Wall 1999; Butler and Moseley 2003).

THE PHYSIOLOGY OF PAIN

This section will discuss the physiology of pain under three categories:

- activation of the nociceptive system;
- sensitisation of the nociception/pain system;
- pain modulation via psychological and social influences.

Within each category, we will separate peripheral from spinal and brain mechanisms, when it is appropriate to do so.

Activation of the nociceptive system

Peripheral transmission

The peripheral nervous system is well studied but not completely understood. Many types of neurones in the periphery are thought to contribute to nociception (Meyer et al. 2006). The most important of these neurones are probably Aδ and C fibres – conventionally called nociceptors – although many Aδ and C fibres also respond to non-noxious inputs (Craig 2002).

For the sake of clarity, we will classify Aδ and C fibres as *primary nociceptors* (see Table 17.1).

Nociceptors are found in most tissues of the body and can be considered to have the principal role of detecting dangerous thermal, mechanical or chemical stimuli. Studies have attempted to identify activation thresholds for different noxious stimuli (the activation threshold is the lowest intensity of a stimulus that produces an action potential in the nociceptor). While there is variability between individuals, there seems to be a predictable range of activation thresholds for primary nociceptors in healthy pain-free individuals.

Thresholds of unsensitised nociceptors

- *Mechanical* – two classes:
  - mechanically sensitive afferents: early response (e.g. to pinch);
  - mechanically insensitive afferents: > 600 kPa (see Meyer et al. 2006);
- *Thermal*:
  - heat: 41–49°C (Tillman et al. 1995);
  - cold: 6.7°C (hand)–11.8°C (forearm); 0°C (Kelly et al. 2005);
- *Chemical* (concentrations that have elicited pain):
  - acetylcholine 1% solution (Schmelz et al. 2003);
  - capsaicin 0.1% solution (Schmelz et al. 2003).

Table 17.1 Comparison of characteristics of Aδ and C fibres. Characteristics referred to by the letter 'a' are physiological, while 'b' describes consequences for the nature of pain.

Characteristic	Aδ	C
1. Conduction speed*	Fast (4–36m/s)	Slow (0.4–2m/s)
2a. Accommodation (adaptation)	Fast	Slow
2b. Pain duration	Brief	Long
3a. Receptive field	Small	Large
3b. Localisation	Precise	Diffuse
4. Sensory quality	Sharp, pricking	Aching, dull, burning
5. CNS response	Reflex, analysis	Emotional, suffering

*Because Aδ fibres conduct more quickly, the pain that is evoked usually commences sooner than it would if only C fibres were activated.
(Reproduced from van Griensven (2005); see also Craig (2002) and Meyer et al. (2006).)

Spinal transmission

Primary nociceptors terminate in the dorsal horn of the spinal cord. The dorsal horn consists of numerous layers, which are defined according to the projections and structural and functional properties of their neurones. The majority of nociceptors terminate in laminae I (Aδ fibres usually terminate here), II (C fibres usually terminate here) and V (Aδ, C and Aβ fibres all terminate here). Lamina II neurones project to other laminae and can be excitatory or inhibitory. Because peripheral input onto lamina I fibres is almost exclusively nociceptive (Aδ), the neurones that project from here are called *nociceptive-specific* second order neurones. Because peripheral input onto lamina V second-order neurones includes Aδ, C and Aβ fibres, lamina V second-order neurones tend to respond over a wide range of stimulus inputs, which is why they are called *wide-dynamic range neurones*.

Transmission from the dorsal horn to the brain

There are five ascending pathways for nociceptive information. The largest is the *spinothalamic tract*, which includes neurones that originate in laminae I and V. Other pathways include the spinoreticular tract, spinomesencephalic, cervicothalamic and spinohypothalamic. Importantly, there is not a single nociceptive pathway, nor is there a single target location (a 'pain centre') in the brain. Rather, there are many sites within the central nervous system (CNS) at which nociceptive input is processed and modulated.

Brain transmission

The brain is defined here as that part of the CNS that lies above the spinal cord. There are several important characteristics of how nociception is handled by the brain.

Parallel processing

Many brain areas are involved in pain. Most often, the thalamus, insula, primary (S1) and secondary (S2) somatosensory and prefrontal cortices are involved. These areas are called the pain matrix (Apkarian et al. 2005) (Figure 17.1). However, these areas are never the only areas involved and they are not always all involved – the pattern of brain activation varies greatly between, and within, individuals.

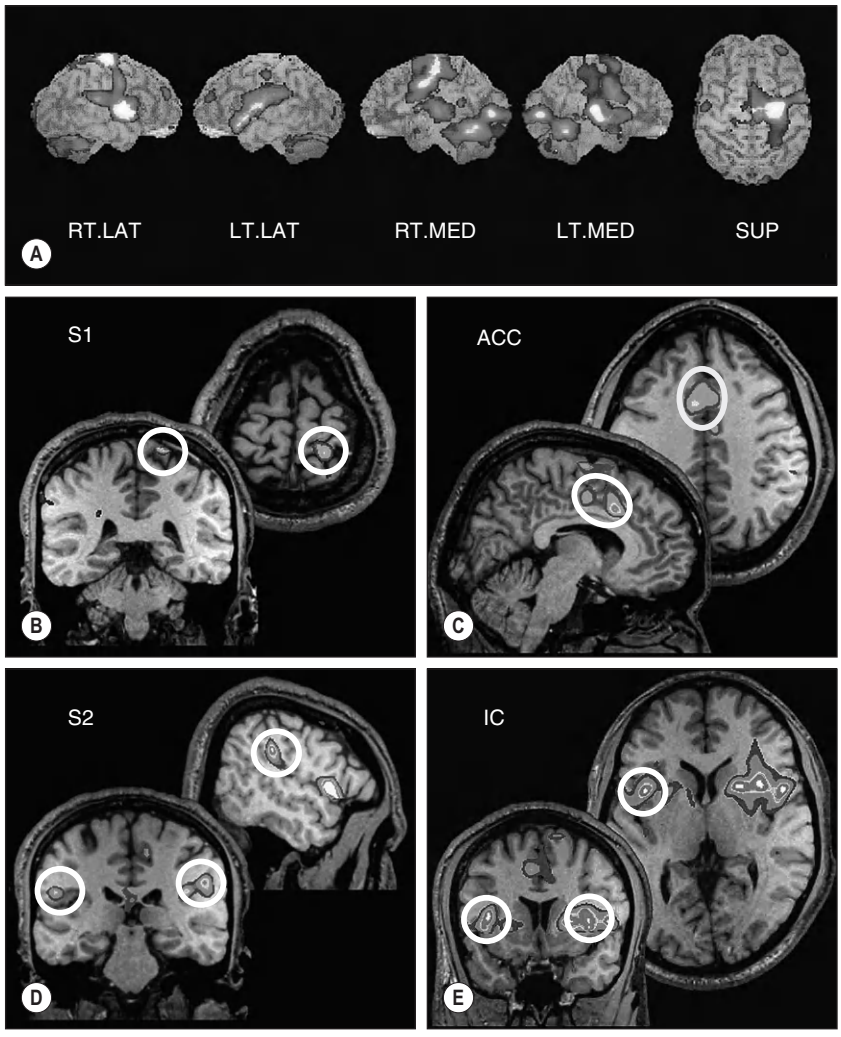


Figure 17.1 Pain evoked activation in the human brain. (a) Surface-rendered image obtained using positron emission tomograms (PET). It shows areas of the brain that are more active during painful thermal stimulation than during non-painful thermal stimulation. (b–e) Images obtained using functional magnetic resonance image (fMRI) of a similar stimulus. This time, cross sectional views are shown so that we can see activation of primary and secondary somatosensory cortices (S1 and S2), anterior cingulated and insular cortices (ACC and IC). Because the areas shown in each image are more active during pain than during non-painful heat, we take them to be important in producing pain. *(Part (a) reproduced from Casey et al. (2001); (b–e) adapted from Bushnell et al. (1999) with permission.)*

Different aspects of pain seem to involve different brain areas

In an attempt to clarify the potential roles of different brain areas in pain, some authors conceptualise two systems. The medial nociceptive system includes the medial thalamic nuclei, anterior cingulate and dorsolateral prefrontal cortices. It is described as slow and only broadly somatotopic, which means it does not have the capacity to code in detail the location in the body at which the stimulus occurred. Activity in this system has been proposed to subserve the affective-emotional dimensions of pain, i.e. the unpleasantness of pain rather than the intensity and quality. This aspect of pain may have value to the person experiencing pain by triggering behaviour that demands other people's attention (Goubert et al. 2009).

The lateral nociceptive system *includes* the lateral thalamic nuclei and the primary (S1) and secondary (S2) somatosensory cortices. It is described as fast and is highly somatotopic, which means it is able to code, in great detail, the tissue location at which the stimulus occurred. Activity in this system is proposed to subserve the sensory-discriminative aspects of pain, i.e. the intensity of the pain and the sensory characteristics of the stimulus (e.g. warm, sharp, deep, superficial). The intensity and unpleasantness of pain can be independently manipulated (e.g. Moseley 2007a).

Multiple inputs: The neuromatrix theory

Melzack (1990) proposed the neuromatrix theory as a way of conceptualising how myriad inputs and factors affect pain and nociception (Figure 17.2). Inputs and factors in that theory include previous learning and past experiences, immune and endocrine states and responses, activity in the autonomic nervous system, and information coming from nociceptors and other sensory receptors. The theory suggests that pain will occur when all these elements are evaluated and a specific network of neurones in the brain is activated. Melzack calls each particular network of neurones a neurosignature (or 'neurotag' (described by Butler and Moseley (2003)), which is analogous to the representation concept used in cognitive neuroscience literature (see Damasio (2000) for a review of representation theory). Remember that this is a theory and that the brain physiology underpinning pain and consciousness is not well understood.

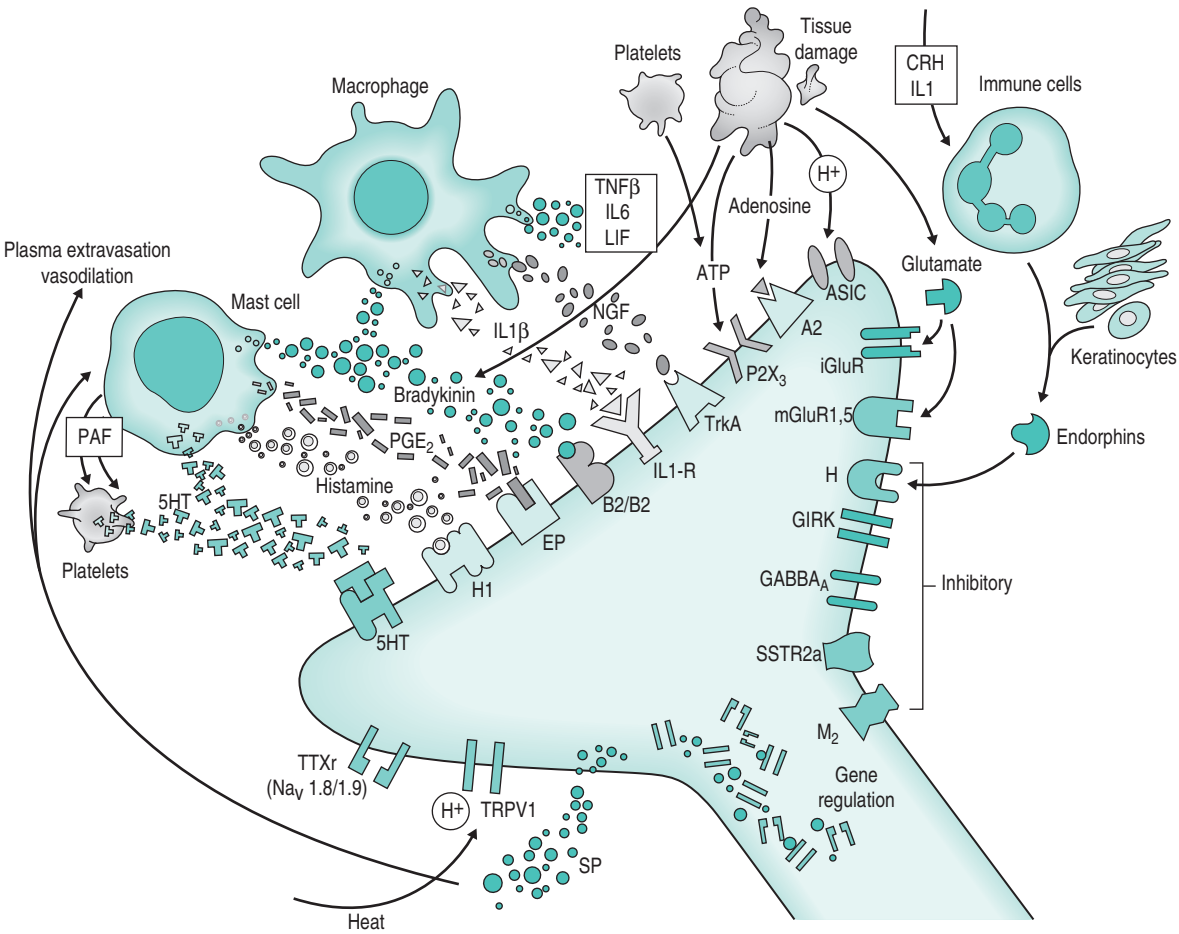


Figure 17.2 Potential peripheral mediators of peripheral sensitisation after inflammation. The point of this figure is not so that the reader can remember everything that happens, but to illustrate that even peripheral sensitisation, which occurs almost every time we injure tissue, is extremely complex. The interested reader should refer to the cited text because to discuss all of these processes here is beyond the scope of this chapter. *(Artwork by Ian Suk, Johns Hopkins University, adapted from Woolf and Costigan (1999).)*

Pain is of the brain

Both Melzack's 'Neuromatrix Model' and the International Association for the Study of Pain (IASP) definition of pain highlight the need to use a biopsychosocial approach in evaluating and treating a person's pain. They also reinforce the concept of pain being created by the brain as a result of multiple influences, in particular the detection or perception of danger to the body's tissues. Tissue damage and the resultant nociception is just one of these influences. As is suggested by the IASP definition, tissue damage is neither sufficient nor necessary for pain.

Sensitisation of the nociception/pain system

The nociceptive system is dynamic. If the system becomes more sensitive than usual, it often results in hyperalgesia (things that hurt now, hurt more) and allodynia (things that didn't hurt now do).

ABC Definitions

Hyperalgesia is an increased response to a stimulus which is normally painful. Allodynia is pain caused by a stimulus that does not normally provoke pain

(Merskey and Bogduk 1994).

Hyperalgesia and allodynia might be expected and seem intuitively sensible after recent damage to tissue, but they are also a predominant feature of persistent pain. It is unlikely that the same processes are involved in hyperalgesia and allodynia in acute post-injury pain, and hyperalgesia and allodynia in chronic pain.

Peripheral mechanisms

Peripheral mechanisms that increase the pain evoked by a standardised stimulus include the presence of inflammatory mediators (including proinflammatory cytokines, bradykinin, prostaglandins), decreased circulation (which increases the local concentration of H^+ ions), the presence of immune mediators and activation of certain genes (Figure 17.2) (see Meyer et al. (2006) for an exhaustive review of peripheral mechanisms of modulation). Inflammatory mediators are released by tissue damage and by activation of nociceptors (neurogenic inflammation). This sensitisation, driven by peripheral mechanisms, is called peripheral sensitisation and the hyperalgesia that follows is called primary hyperalgesia.

Spinal cord mechanisms

Second order nociceptors can also become sensitised. The N-methyl-D-aspartate (NMDA) receptors in the dorsal horn respond to persistent or intense stimulation by opening channels to allow greater post-synaptic activity. This means that the same input from the periphery will result in greater activation of the second order nociceptor, which means that more messages of danger to body tissue will be sent to the brain.

Dorsal horn mechanism

NMDA receptors are important in central sensitisation. They are normally blocked by magnesium, but prolonged nociceptive activity and release of peptides can remove the block and allow glutamate to bind to the receptor. The resultant activation of voltage-sensitive channels allows an influx of calcium into the second order neurone. This process can lead to a change in the NMDA receptor so that the magnesium block then becomes less effective, which means that, next time, the sensitisation will happen more quickly.

There are other mechanisms in the dorsal horn that can sensitise the nociceptive system. Wide-dynamic-range neurones in the laminae of the dorsal horn provide an interaction of the processing for nociception and other sensory information. This may be important in how people describe their pain, sometimes referred to as the 'quality' of pain. It also provides a potential mechanism for further sensitivity of the nociceptive system, whereby the responses to normally non-painful stimuli, such as touch, are now painful. This may be because the wide-dynamic-range neurones synapse with sensitised neurones. Another mechanism of sensitisation involves the sprouting of neurones across laminae. Such sprouting may link a non-nociceptive peripheral nerve fibre (e.g. Aβ fibre) with an interneurone that would normally respond to noxious stimulation. This is probably more likely to occur after peripheral nerve injury or death. Butler and Moseley's (2003) *Explain Pain* discusses these mechanisms in an accessible, but reasonably detailed, way.

Brain mechanisms

Supraspinal mechanisms that enhance the sensitivity of the CNS include influences, mediated by the rostroventral medulla, on the inhibitory transmission affecting the spinal cord. Current research is looking not only at descending modulation, but also on intracortical modulatory processes. Imaging studies have associated pain processing with activity in multiple brain areas, commonly including the thalamus, anterior and posterior cingulate cortex, other areas of the limbic system and the medial prefrontal cortex (Bushnell and Apkarian 2006; Schweinhardt et al. 2008; Wiech et al. 2008). The

attributed function of these brain areas supports a role in the cognitive modulation of pain (see below).

Neuropathic pain

The pain associated with nerve damage is known as neuropathic pain and may include descriptions such as 'burning' or 'electric shock'. CNS damage, such as stroke, can result in centrally-mediated neuropathic pain that reduces the inhibition of the nociceptive system. Peripherally, damage to an axon or the myelin covering of a nerve, can lead to spontaneous transmission of impulses from the damaged area or the dorsal root ganglion (DRG). This may also be driven, in part, by loss of inhibition, specifically through alterations of chloride homeostasis affecting gamma amino-butyric acid (GABA)-mediated inhibition (see review of mechanisms in De Koninck (2009) and also Sandkühler (2009)). Such damage will usually result in hypersensitivity to mechanical input. This sensitivity forms the basis for tests such as Tinel's sign – sometimes considered as an indicator of neuropathic pain.

There is mounting evidence that interactions between the immune system, the endocrine system and the autonomic nervous system are important in all types of pain, including neuropathic pain (Watkins and Maier 2000).

Pain modulation via psychological and social influences

Anecdotal evidence that somatic, psychological and social factors modulate pain is substantial – sport- and war-related stories are common (see Butler and Moseley (2003) for several examples). However, numerous experimental findings corroborate the anecdotal evidence (see Fields et al. (2006) for a review of CNS mechanisms of modulation and Poleshuck and Green (2008) for a review of the impact of socioeconomic disadvantage and pain).

By far the greatest research efforts in this area have been directed towards the cognitive modulation of pain. Experiments that manipulate the psychological context of a noxious stimulus often demonstrate clear effects on pain, although the direction of these effects is not always consistent.

Attention

A large amount of literature concerns the effect of attention on pain and of pain on attention, for example Asmundson et al. (1997), Crombez et al. (2004), Eccleston and Crombez (2005), Naveteur et al. (2005), McCracken (2007), Lautenbacher et al. (2010), Verhoeven et al. (2010).

Despite the wealth of data, consensus is lacking: some data suggest that attending to pain amplifies it and attending away from pain nullifies it; others suggest the opposite. This may be explained by the existence of different modes of selective attention (see Legrain et al. (2009) for review). Most likely, the effect depends on the coping style of the individual and the wider context of the experiment or situation.

Anxiety

Anxiety also seems to have variable effects on pain. Some reports link increased anxiety to increased pain during clinical procedures (Schupp et al. 2005; Klages et al. 2006) and during experimentally-induced pain (Tang and Gibson 2005), but other reports suggest no effect (Arntz et al. 1990; Arntz et al. 1994). Relevant reviews conclude that the influence of anxiety on pain is probably largely dependent on attention (Arntz et al. 1994; Ploghaus et al. 2003).

Expectation

Expectation also seems to have variable effects on pain. As a general rule, expectation of a noxious stimulus increases pain if the cue signals a more intense or more damaging stimulus (Fields 2000; Sawamoto et al. 2000; Keltner et al. 2006; Moseley 2007a; Moseley and Arntz, 2007) and decreases pain if the cue signals a less intense or less damaging stimulus (Pollo et al. 2001; Benedetti et al. 2003). There are several informative reviews on the influence of expectation on pain (Fields 2000; Wager 2005).

The common denominator of the effect of attention, anxiety and expectation on pain seems to be the underlying evaluative context, or *meaning*, of the pain. That is demonstrated by the consistent effect that some cognitive states seem to have on pain. For example, catastrophic interpretations of pain are associated with higher pain ratings in both clinical and experimental studies (see Haythornwaite (2009)). Believing pain to be an accurate indicator of the state of the tissues is associated with higher pain ratings (Moseley et al. 2004), whereas believing that the nervous system amplifies noxious input in chronic pain states increases pain threshold during straight leg raise (Moseley 2004).

Social context

The social context of a noxious stimulus also affects the pain it evokes. For example, when men have blood taken by a woman, it hurts less than when it is taken by another man (Levine and De Simone 1991). The effects are variable but, again, seem to be underpinned by the underlying evaluative context or meaning (see Butler and Moseley (2003) for a review of pain-related data and Moerman (2002) for exhaustive coverage of the role of meaning in medicine and health-related interactions).

To review the very large amount of literature on somatic, psychological and social influences on pain is beyond the scope of this chapter. However, it is appropriate, and clinically meaningful, to reiterate the theme that emerges from that literature: the influences on pain perception, tolerance and report are variable and seem to depend on the evaluative context of the noxious input.

ASSESSMENT AND MEASUREMENT OF PAIN

Pain is an essentially personal experience, which means measurement of pain relies on the person in pain communicating their experience. Therefore, any measure of pain, including report of pain, is really a measure of pain behaviour. Clinicians and researchers make a judgement according to how well they think the measure of pain behaviour might reflect pain, but, ultimately, this relies on assumptions that as yet cannot be verified. This limitation also applies to physiological measures, such as brain imaging.

Earlier in this chapter, we argued that pain depends on many modulating factors and can be expressed in many ways. Moreover, we argued that pain is one output of the brain that serves to protect the tissues. Those arguments mean that assessment and measurement of people in pain encompasses more than measurement of their pain. One framework that is useful in assessment of people in pain is the World Health Organization International Classification of Functioning, Disability and Health (WHO ICF).

The WHO international classification of functioning, disability and health (WHO ICF)

This section uses the WHO ICF as a framework for assessment and treatment planning of patients in pain. This framework is advocated by the IASP (Wittink and Carr 2008). The WHO ICF focusses on how a person is functioning and evaluates outcomes using the person's actual performance in the real-life environment. This makes it especially useful for those patients where pain relief is not the only, and possibly not the most important, objective.

The WHO ICF has three components: the body (function and structure); activities and participation; and contextual factors (e.g. environmental factors that might influence function). Each component is classified at a level of severity (none, mild, moderate, severe, complete) and interactions between components are evaluated (WHO 2002). As it relates to people with pain, the three components of the ICF broadly mirror the components of the biopsychosocial model, which considers tissue-based, psychological and social influences on pain.

The interview

Most therapeutic processes will start with an interview. The interview aims to capture information about the person in terms of the ICF. Physiotherapy interviews have conventionally focussed on symptoms: location, intensity, quality and temporal patterns, and signs. This information is very important, but it is not sufficient. According to the ICF this information relates to *the body*, the biopsychosocial model's equivalent of the tissues. However, we advocate, as do other reviews in this area (e.g. Gifford et al. 2006) that the interview must also provide information about activities and participation and contextual factors (i.e. the 'psychosocial'), and how these issues may interact with pain and the state of the tissues (Goldingay 2006a, 2006b). The aim of the interview then is to identify factors from across biopsychosocial domains which activate or sensitise the nociceptive or pain system (see Table 17.2).

Measures and scales

Self-reporting measures

Assessing pain is a key aspect of the assessment process. Self-reporting measures are considered the gold standard in assessing pain intensity, location, quality and temporal variation.

ABC Definitions

Quality of pain refers to characteristics, such as aching, burning, stabbing and cramping, that a patient attributes to his/her pain.

Temporal aspects of pain commonly refers to 24-hour behaviour of pain but might also relate to other influences, such as menstrual cycle or seasonal changes.

Visual analogue scales (VAS) and numerical rating scales (NRS) are most common and useful. A visual analogue scale consists of a horizontal line, usually 100 mm long. At each end is a term of reference for the patient, called an *anchor*. The left anchor is usually 'No pain' and the right 'worst pain' or 'worst pain imaginable'. The patient marks a point on the line in answer to a question about their pain and the distance from the mark to the left anchor is used as a measure of their pain. A NRS uses numbers instead of a line, such that 0 = 'no pain' and 10 = 'worst pain'. The VAS is probably more sensitive to change, less vulnerable to perseveration (remembering what you said last time and responding the same way) and more difficult to measure. The NRS is easier to use clinically and is probably sufficiently sensitive to detect clinically meaningful

Table 17.2 Structured interview for biopsychosocial assessment

Area of examination	Information gained
Orientation	Nature and location of symptoms; patient's story from onset to present, expectations of physiotherapy, questions and concerns about problem
Previous intervention	Investigations and understanding, treatment effects, advice received, causal beliefs
Medical history	Comorbidity and effect on function, special questions (red flag screening), medication
Effects on function and participation	Current social and employment situation, typical day, effects on work, restrictions on activity, assistance required, aids and adaptations, downtime, sleep
Coping strategies	Current coping strategies: active and passive, perceived consequences of change (e.g. increasing activity, exercise, pacing)
Socio-economic	Effect on finances, benefits, medico-legal
Effect on family	Beliefs, responses, nature of support provided
Emotion	Nature and extent, effect on motivation
Pre-examination	Body chart, behaviour of symptoms

Reproduced from Goldingay (2006b), with kind permission of S. Goldingay and the CNS Press.

changes [a clinically meaningful change is usually considered to be about two points on a ten-point scale (McQuay et al. (1997))].

Several tools assess the quality of pain. A simple tool is a VAS, with anchors reflecting the unpleasantness of pain. The most widely used is the short form of the McGill Pain Questionnaire (MPQ) (Melzack 1975a). The MPQ lists a variety of words that are grouped as being about the sensory-discriminative aspect of pain (e.g. sharp, burning, intense), the affective aspect of pain (e.g. punishing) or its evaluative context (e.g. annoying). There are many other measures that emphasise different aspects of pain.

Behavioural measures

Although self-reporting measures are behavioural measures, they are generally considered as a separate category. Non-self-reporting behavioural measures are useful as a corroborator of self-reporting measures, when the patient is unable to use the measure (e.g. children) or when the clinician considers the patient's report to be dubious. Behavioural measures include observation (by a covert or overt observer) and performance (e.g. functional, endurance, speed or load tests). Full review of behavioural measures is beyond the scope of this chapter, but the interested reader is referred to the *Handbook of Pain Assessment* (Turk and Melzack 2001) and *Manage your Pain* (Nicholas et al. 2007) for examples.

Measures of the impact of pain

Critical to the ICF framework, and to the biopsychosocial model, is evaluation of other factors that might be modulating pain or the behavioural response to pain, or both. Numerous measures have been devised and tested. The following is a brief account of the most widely used and documented.

Measuring potential impact of beliefs and thoughts

The importance of evaluation of the threat to body tissue has been mentioned earlier. Inherent to this evaluation is fear of movement and (re)injury. A large amount of research has investigated the role of pain-related fear avoidance behaviour. There are many specific findings that relate fear and catastrophic thought processes to pain intensity, disability and self-efficacy but the *principle* can be summarised thus: people who have heightened concerns about pain, its cause and its consequences, often have a sense of helplessness and adopt a passive coping style. As a result, they have a lower criterion for a movement or activity to be considered potentially painful or potentially injurious. They therefore avoid those movements and activities – 'fear-avoidance'. Avoidance of activity leads to disuse and hampers healing and recovery, leaving the person in a vicious cycle of progressive depression, disuse and disability (Figure 17.3). According to the fear-avoidance model, most people do not follow this path because they have a more accurate and appropriate level of concern and thus do not tend to avoid movement and activity (Vlaeyen and Linton, 2000). See Vlaeyen and Linton (2000) for a review of fear-avoidance beliefs and pain and Sullivan et al. (2001) for a review of catastrophic thought processes and pain.

Fear avoidance beliefs can be gauged via the interview, but can be quantified via the fear avoidance beliefs questionnaire (Waddell et al. 1993) or the TAMPA scale of kinesiphobia ('fear of movement') (Miller et al. 1991). Data for both tools are available, from a range

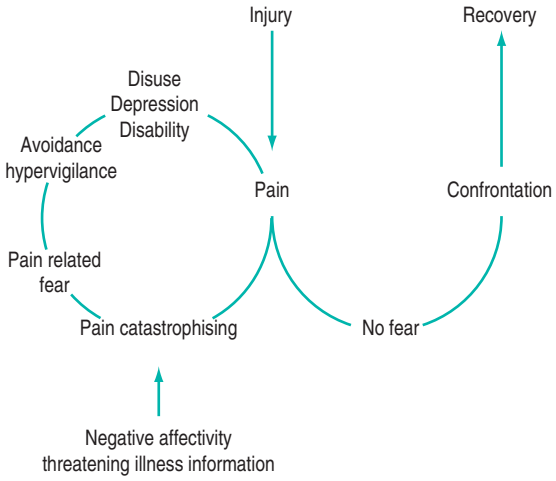


Figure 17.3 The fear-avoidance model.

of clinical and non-clinical populations. Catastrophic thought processes can also be gauged via the interview, but can be quantified via the pain catastrophising scale (Sullivan et al. 1995), which also has a large amount of data available. Other measures that are psychometrically robust and widely used in research, but are less popular clinically, include the survey of pain attitudes (Jensen et al. 1987), the pain beliefs and perceptions inventory (Williams et al. 1994), and the pain anxiety symptoms scale (McCracken et al. 1992).

What about the clinician's thoughts and beliefs?

There is some evidence that the clinician's thoughts and beliefs about pain modulate the effect of their treatment. For example, researchers undertook a placebo-controlled, double blind study of fentanyl (a powerful analgesic) during wisdom teeth removal (Gracely et al. 1985). Although all patients had the same likelihood of receiving fentanyl, the researchers told some of the dentists that the patient would not receive fentanyl. This was a lie. When the clinician thought that the patient might get fentanyl (which was true), pain dropped by two points after the placebo injection. When the clinician thought that the patient could not get fentanyl (which was a lie), pain increased by five points after the placebo injection. This difference (~7 points) was owing to the belief of the dentist!

Other studies have shown that the fear-avoidance beliefs of people with back pain are similar to those of their rheumatologists (Poiraudeau et al. 2006) and that rheumatologists' fear avoidance beliefs affect the decisions they make about their patients' management (Coudeyre et al. 2006).

Self-efficacy

Self-efficacy is the belief an individual has about their ability to perform a task using their current resources. Because self-efficacy depends on appraisal of the demands of a particular task and of the individual's own capacity to meet them, self-efficacy depends on evaluative factors about pain and injury. Self-efficacy is therefore affected by inappropriate or inaccurate appraisals. However, self-efficacy can be assessed as a separate construct that is sensitive to treatment and is closely related to function (Nicholas et al. 1991). For more information on self-efficacy as it relates to pain assessment and management see Nicholas (2007).

Red, yellow, blue and black flags

Red flags are those signs and symptoms that should alert the physiotherapist to seek other specialist opinion. A useful resource for patients with back pain is Greenhalgh and Selfe's *Red Flags*, which includes a focussed discussion (Greenhalgh and Selfe 2006).

The 'yellow' flags are signs, symptoms, beliefs and attitudes that are thought to increase the risk of acute pain developing into a disabling chronic pain condition. This approach was devised to assess for risk of pain-related disability in people with acute (recently occurred) back pain. Increasingly, it is being used in people who report pain in other anatomical locations. The purpose of assessing in this manner is to identify patients who have unrealistic and unhelpful beliefs about their pain and prognosis (Kendall et al. 1998). While there are standardised questionnaires available, carefully selected questions can also be used effectively during interview (Watson and Kendall 2000).

As an adjunct to yellow flags, a more comprehensive consideration of occupational factors can be achieved by considering assessment of 'blue' and 'black' flags. 'Blue' flags would be indicated if a person has work-related concerns about how he/she as an individual might be impeded in returning to work. For example, there may be a perception that the managers do not want this person to return to work. 'Black' flags are indicated if work processes and policy (which influence all workers) impede the person returning to work. An example of this would be where an employer does not have any occupational health processes in place, such as workplace assessment or return to work plans.

Measures of functional limitation and disability

There are a large number of self-report tools that aim to measure the functional impact of a person's pain. A large proportion of those tools concern back pain [e.g. the Roland Morris disability questionnaire (Roland and

Morris (1983)) and the Oswestry questionnaire (Fisher and Johnston (1997))] or neck pain (e.g. the neck disability index (Vernon and Mior (1991)). These tools are widely used and a large amount of data exists from patient and non-patient populations. There are also disability questionnaires that are not anatomically focussed, for example the task-specific tools whereby the patient selects a task or activity that they are unable to perform because of pain and rates their ability to perform that task over the course of treatment. An advantage of such a tool is that it can be used with different populations. A disadvantage is that it makes direct comparison between treatments or between patients difficult.

A recent review explored a range of pain assessment instruments with the purpose of identifying people at risk of developing persistent pain (Grimmer-Somers et al. 2009). The review identified 16 assessment tools suitable for use in primary health settings. This is one of many attempts to identify assessment strategies that screen those at risk of developing long-term pain and disability. Early identification of 'modifiable obstacles to recovery' (Watson et al. 2010) has been an underachieved aim, despite the development of clinical evidence-based guidelines. Concepts of 'acute' and 'chronic' pain may be unhelpful here, as multi-dimensional pain assessment may not occur for several weeks post-injury. To ensure early risk identification, pain assessment should always aim to be multi-dimensional, even in instances of acute injury with frank tissue damage.

It should also be noted that novel treatment techniques (see later) drawn from revolutionary knowledge about the plasticity of the brain also demand novel assessment techniques. Harman (2000) wrote about the impact of plasticity on assessment and treatment of pain more than a decade ago and more recently Flor and Moseley and colleagues have assessed for asymmetries of brain representations to guide their interventions (Moseley 2007b; Flor 2009; Moseley and Wiech 2009, 2010; Moseley et al. 2009). While it is impractical to be scanning the brain of people in clinical practice, more practical assessment techniques using computer or card-based images may be indicators of the central processing asymmetries.

MANAGEMENT OF PAIN

European guidelines for the management of low back pain, based on the best available research evidence, have been published. Included are guidelines for people with pain of recent onset, for people with pain that is persistent and for people with pain that is threatening to lead to ongoing disability (van Tulder et al. 2002; Airaksinen et al. 2004). Elements of these guidelines will be presented below (see Airaksinen et al. (2006) for application in physiotherapy). It is important to recognise that these are specific to low back pain, but in the absence of guidelines they may also provide a basis for the management of other painful conditions.

Promoting optimal function with reference to WHO ICF

By using the ICF framework, the range of factors influencing a person's function will be considered as treatment targets. For example, the physiotherapist may recognise that a person with back pain has restricted movement. If, when considering restriction in *activities and participation*, it is also identified that the person is afraid of walking up the front stairs at home because they do not have a rail, the focus of treatment might change to incorporate this context. Thus, the clinician must reason as to the most achievable goal: addressing the restriction in movement may resolve the fear about the stairs. Alternatively, the person may need a handrail. Optimally, the physiotherapist assists the person in becoming confident to climb stairs without a rail. This will lead to generalisation to confidence in other environments. The important issue is that focussing on body structure and function alone will not guarantee that function is restored, and using the ICF prompts clinicians to explore and resolve other barriers to rehabilitation.

Clinical note

Warning: You can make things worse.

These behaviours of the clinician have been linked to worse outcomes (Kouyanou et al. 1998):

- over-investigation;
- over-treatment;
- unhelpful treatment;
- disconfirmation of pain;
- unhelpful advice.

Deactivating/desensitising the nociceptive system

While many traditional approaches to pain support peripheral treatment to the musculoskeletal system and a focus on deactivating pain systems there is an increased need to consider treatments that promote desensitising mechanisms. Indeed, it may be that many so-called musculoskeletal treatments, such as manipulation and mobilisation of joints, may be better described as neurological treatments. Assessing the sensitisation (i.e. via assessment of state and structure) of the nervous system then becomes

the starting point for making sense of a person's pain and for identifying the best intervention (Jones 2007). In cases of persistent pain, innovative treatment approaches have exploited knowledge of sensitivity and brain activity for best results. The following discussion will lead from interventions where the local site of injury is believed to be the target, to more complex interventions where the effects of treatment are likely to be diverse.

Addressing peripheral mechanisms of nociception

Removing the noxious stimulus is often the primary motivation of the person with pain and where this is not possible it becomes the clinician's role. As such, reducing high threshold mechanical, chemical or thermal stimuli should deactivate the primary afferent activity. However, the consequences of cell damage establish at the least, peripheral sensitisation as a normal and necessary sequel to injury. With the nervous system in this state, normally subthreshold stimuli may now trigger nociceptor activity. For example, normally innocuous biomechanical deformation may now lead to pain; indeed, this is the basis for using palpation to detect tissue damage.

Inflammation is the main cause of peripheral sensitisation, so limiting inflammation should help desensitise the nociceptive system. Normally, the body's response to injury, which includes motor, immune and vascular responses as outlined earlier, limits the period of inflammation. In addition, the conventional response to inflammation has been to initially cool the area, compress, elevate and immobilise it (rest, ice, compression and elevation (RICE)) and as healing progresses engage in a gradual increase in movement and activity.

There are also pharmacological means to reduce or limit inflammation. The broad class of drugs that are most often used to reduce inflammation are aptly called 'anti-inflammatories', although there are three important considerations. Firstly, the clinician must evaluate the relative importance of inflammation in initiating the healing process. This depends on sound knowledge of biological mechanisms associated with healing, nociception and pain. Secondly, the site of analgesic effect of anti-inflammatories has not been established – it is unlikely that their only effect is at the tissues. Thirdly, even if their main effect is at the tissues, they cannot be confined to the target area because they are administered systemically (e.g. orally, by injection) or transported remotely in the circulation (e.g. topical agents). This is why most drugs have unwanted side effects. Thus, advice about any drug should not be made without appropriate expertise and medico-legal jurisdiction.

An important method by which inflammation and its effects are normally limited is movement. Movement promotes blood flow, oxygen supply, waste product removal and appropriate forces to guide healing. One common barrier to movement is the conscious decision of the person to not move – a decision enhanced by the increased nervous system sensitivity. Such decisions depend on the person's evaluation of the importance and risks of doing so, and this is where the clinician can be key to normal recovery – effectively removing the barriers to abnormal recovery. Of course, strategies that enhance a person's motivation to move should be considered in light of a sound understanding of tissue healing and within a clinical reasoning framework. This includes determining the stage of healing and therefore the relative vulnerability of affected tissues – at times this is counter to the pain report of the patient. We will revisit motivation later when we focus on central mechanisms.

Awareness of analgesics

In some places the physiotherapist has a prescribing role. However, it is more common for physiotherapists to liaise with other healthcare professionals to ensure effective pain relief is available and administered to the patient. Different analgesic drugs act differently, stay in the bloodstream for different durations and have different side effects. It is important that physiotherapists know about the analgesics which are prescribed to the patients with whom they work.

Addressing spinal mechanisms of nociception

According to the 'Gate Control Theory', interactions of primary afferent and second order neurones in lamina II can modulate the transmission from nociceptors (Melzack and Wall 1965). While the theory is more than 40 years old, and the understanding of neurophysiology has developed further, the basic premise that sensory information is received and modified in the dorsal horn by competing stimulation and descending information holds true.

Thus, there are two broad mechanisms that can desensitise spinal nociceptive neurones: shifting the balance of descending modulation toward inhibition and providing novel peripheral input that activates Aβ neurones. The former can be promoted by reducing the perceived threat and perceived vulnerability, i.e. make the person feel safe. For example, listen to them, make them comfortable (as possible), speak to them nicely and respect them. The latter can be utilised in varying degrees of sophistication, from 'rubbing it better' to transcutaneous electrical nerve stimulation (TENS), which was developed as a direct application of the gate control theory (Melzack 1975b). There is good evidence that TENS reduces acute pain, but not chronic pain (McQuay et al. 1997).

Elements of other comforting treatments, such as therapeutic ultrasound, heat therapy and massage therapy, may also contribute to spinal desensitisation by making the person feel safe and by providing novel peripheral input. This highlights that benefits seen with many treatments can potentially be attributed to diverse mechanisms, including those associated with placebo. Where these benefits do not clearly align with the theory supporting the treatment modality, they are referred to as non-specific treatment effects and perhaps reflect complex brain mechanisms. It is the clinician's responsibility to critically analyse the benefits of a treatment modality and recognise which elements are influencing the observed outcomes. Systematic reviews and clinical guidelines can be instructive in this analysis (Ottawa Panel 2004; French et al. 2006; Furlan et al. 2008).

Addressing brain mechanisms of nociception and pain

The mechanisms by which nociception and pain can be decreased by the brain are not well understood. However, the *principle,* which depends on the conceptualisation of pain as the conscious correlate of the implicit perception of threat to body tissues (see Moseley (2007a) for a review) is well understood. We argued earlier that pain depends on evaluative factors, which means that any input that alters the brain's evaluation of threat to body tissue should modulate pain. In the same way that evaluative factors should change pain, they should also change descending modulation of nociceptive circuits and, therefore, the state of the nervous system, i.e. its sensitivity. Comprehensive coverage of the strategies that are advocated within the literature is beyond the scope of this chapter, but it is appropriate to mention some of them.

Cognitive-behavioural therapy

Cognitive-behavioural therapy probably began as a combination of cognitive therapy and behavioural therapy. Cognitive therapy focusses on identifying, challenging and replacing cognitions (thoughts, beliefs and emotional processes) that might be contributing to the patient's pain or functional limitations, perhaps via fear or avoidance of movement and re-injury, or simply by elevating the perception of threat to body tissue. Once identified, thoughts and beliefs can be challenged by pointing the patient to material, experiences or situations that contradict that belief. Ultimately, unhelpful thoughts and beliefs need to be replaced by more accurate or helpful ones.

Behavioural therapy was first applied to management of people in pain several decades ago (Fordyce 1970, 1973). In that work, operant conditioning principles guided treatment such that behaviours consistent with decreased pain or disability were reinforced and those consistent with increased pain or disability were not.

Modern cognitive-behavioural pain management is far wider in its application than cognitive and behavioural therapy combined. There are many strategies that are incorporated into a cognitive-behavioural pain management approach. Cognitive-behavioural principles within the context of physiotherapy practice are comprehensively presented in Nicholas and Tonkin (2004).

Clinical note

The psychological construct of self-efficacy is likely to be important. Self-efficacy is the belief someone has about his/her ability to perform a task using current resources. Physiotherapists tend to be good at promoting activities that enhance self-efficacy, although they may not always recognise it. Education, verbal encouragement, promoting mastery of a task and creating of groups of similar people for activity all have the potential to improve self-efficacy.

Explaining pain biology

Intensive education about the biology of pain is one way in which unhelpful cognitions about pain, injury and activity can be challenged and replaced. Explaining pain biology in detail has been shown to change beliefs and attitudes about pain, movement and activity (Moseley et al. 2004), increase pain threshold during relevant tasks (Moseley 2004) and, combined with movement-based physiotherapy, decrease pain and disability in the long term (Moseley 2002, 2003). By educating someone effectively, the person is given a new perspective on which to base thoughtful action. This involves acceptance by the person that the information he/she had before has been superseded by the newly presented information. Patients are given new concepts that include neural sensitivity and central processing to replace old ones about tissue vulnerability. The material that is used to explain pain is presented in the aptly named book *Explain Pain* (Butler and Moseley 2003).

Motivation

Strategies that are used to enhance motivation include reassurance and education (Butler and Moseley 2003; Moseley et al. 2004), training diaries and overt monitoring of self-initiated rehabilitation (Moseley 2006a), challenging unhelpful cognitions and offering new ones (effectively cognitive therapy principles), careful and graded exposure to movements or activities of which the patient is fearful (Vlaeyen and Linton, 2000) and reinforcement of helpful behavioural responses (effectively, behavioural therapy principles (Nicholas and Sharp 1997)).

Clinical note

Motivation is sometimes used to describe the opposite to laziness. However, it should be considered as a term that describes the outcome of a person's evaluation of influences on the decision to act or not. A challenge for the clinician is to help resolve barriers to action and enhance resources. Together, these might swing the balance and enhance a person's motivation.

Relaxation

Relaxation is best performed at times when a person is still in control of his/her pain but feels that it is worsening. Often, it is targeting the emotional response to pain or the expectation of pain. As such, it is important to ensure patients have a strategy that can be used at such times, preferably without interruption to any task that they might be doing. Physiotherapists often prefer muscular hold-relax techniques but teaching more meditative techniques is perhaps more valuable, as it can be applied in many different contexts and environments.

Clinical note

Relaxation should be seen as a skill that needs to be practised. A useful technique is described by Nicholas (2007). First, there is a focus on breathing and 'loosening' up with exhalation. Second, the person is encouraged to imagine the tension leaving the muscles with each exhalation. The third step involves linking the relaxation to a word the person says to him-/herself (such as 'relax') and an image of a peaceful scene. The word and the image may facilitate further relaxation, but through repetition they will also become triggers to relaxation (via classical conditioning). A person is then able to use the word or the image to quickly bring on a relaxed state.

Pacing

Pacing is essential for people who have persistent pain and find it difficult to plan ahead because they are unsure of how their pain might affect activity. An example is where a person is highly active on days when the pain is mild, but inactive on days when the pain increases. Sometimes called the 'boom or bust cycle', it prevents a person from attending regular employment, as well as interfering with planning of social activities.

Resolution of the 'boom or bust cycle' can be achieved by pacing activity to a level that the person knows they can achieve, despite pain. When pain is increased the person should attempt to use strategies, such as thought challenging and relaxation, to increase activity to the planned level. When the pain is mild, the person must resist increasing activity above the planned level.

Clinical note

It is useful when giving exercise advice to a person with persistent pain or a sensitised nervous system to consider the use of exercise quotas; that is, to use a pre-determined duration or number of repetitions as a limit to activity, rather than using pain or discomfort as the guide. This will reinforce the message that activity is beneficial and that pain is not a sign of ongoing damage.

Setting quotas requires establishing a baseline of tolerance for the exercise or activity. The first quota should be below this (80% of baseline) to ensure the person is confident of achieving it. Then the person should work at gradually increasing activity past the baseline towards a more desirable level. It is essential that the person plans the next quota in advance. Initially, this may be just the day before, then as he/she becomes more confident in his/her activity levels, he/she can plan the week in advance.

Setbacks may occur and sometimes quotas may not be achieved. In such cases, the person should be encouraged to use active coping strategies to try to improve performance. If this fails the quota may need to be slightly decreased. It is important to assist the person with their self-motivation by providing successful experiences and it should also be clear to the person that the activity is the focus not the pain (Nicholas and Tonkin 2004).

When a person first comes to physiotherapy, the individual will have a sense of the level of activity they can achieve. That sense may be exaggerated or, more likely, under-estimated. A more realistic level may only be achieved after education, successful behavioural experiments and reconceptualisation. Optimum levels of activity will be achieved when the skills of thought challenging, relaxation and pacing are well developed and consistently applied.

Graded activity versus graded exposure

Physiotherapists will often employ a graded activity programme to help someone return to full function. The assumed benefits of this are generally considered to be physical, i.e. the person is getting gradually stronger or more flexible and is therefore able to do more. Another

benefit is confidence or self-efficacy. Graded activity can enhance self-efficacy by allowing the person to achieve successful performance at increasingly more difficult levels. Assessment of performance should always include these influences; changes in performance should not just be considered in terms of strength and range of movement.

Further to this is the role of fear or phobias on confidence and on performance. As previously mentioned, fear avoidance behaviour can lead to severe disability. An effective psychological strategy that has been employed to reduce fear is graded exposure. This involves first finding a baseline that does not elicit fear and then gradual exposure to increasingly more fearful experiences that have been identified and ranked by the patient. If someone is fearful of pain on movement then the experiences often will involve physical activities. For example, someone with complex regional pain syndrome in his/her right upper limb may start with imagining the hand being touched by someone else. The person may then progress to stroking a rough texture, to shaking hands with someone, to lifting a bag full of shopping.

By integrating graded exposure and graded activity, the physiotherapist is likely to help the person to reach physical goals that relate to strength, range of movement and function, as well as psychological goals that relate to decreasing fear and increasing self-efficacy, participation and function.

Targeting cortical representations of the body

Emerging data suggest that there may be another strategy by which supraspinal mechanisms of nociception/pain can be desensitised. That strategy relates to the one way in which the brain changes when pain persists – alterations in the representation of the affected part in primary sensory cortex (S1) (see Flor et al. (2006) and Moseley (2006b)). The mechanisms by which this occurs may be activity-dependent, but may also relate to inhibition of non-noxious sensory input at the thalamus (Rommel et al. 1999). That S1 representation changes cause pain is argued (Harris 1999) but not empirically demonstrated (Moseley and Gandevia 2005). Notwithstanding, it is clear that normalisation of S1 representation is associated with reduced pain (Flor 2003; Maihofner et al. 2004; Pleger et al. 2005), that tactile acuity relates to pain and to S1 representation changes (Flor 2003), and that training tactile discrimination increases tactile acuity and reduces pain – at least in some patient groups (e.g. phantom limb pain (Flor et al. 2001) and complex regional pain syndrome (Moseley and Wiech 2009). The role of normalising S1 representation is yet to be established; however, it seems reasonable to suggest that a role may emerge (see Flor (2009) for further reading).

Clinical note

There is a large amount of data that shows (i) you can improve tactile acuity via training; (ii) that when you improve tactile acuity, you do so by altering the firing properties of neurones in the brain; and (iii) improving tactile acuity is therefore associated with altered representation of the trained body part in the brain.

There seem to be several important aspects to training tactile acuity.

- Sensory stimulation: the body part in question must be stimulated.
- Attention: training is more effective if the subject attends to the input. A useful way to ensure that this occurs is to provide a task that requires that they attend. This is what Flor and colleagues did (Flor et al. 2001) and what Moseley and colleagues did (Moseley and Wiech 2009). In those studies, patients were required to discriminate between stimuli of different types and at different locations.
- Gaze direction: training is better if the subject directs his/her head toward the body part being stimulated.
- Visual input: training is better if the subject can see the body part being stimulated.

LIMITATIONS AND OPPORTUNITIES

This chapter attempts a monumental task – to synthesise volumes of pain research from basic and clinical sciences into a digestible package appropriate for clinical physiotherapy practice. The complexity of the human experience, which is exemplified in the human experience of pain, means that this chapter can only provide an introduction. We considered it most important that the reader conceptualises pain in a manner that is consistent with what is known about human biology. Entire books have been written about the peripheral nociceptor and entire libraries could be filled with books on the spinal cord and brain, so we have attempted to convey what we think is sufficient evidence for adopting a certain way of thinking about pain. We think the following points are justified on the basis of the available research and can be considered the key 'take-home messages'.

- The science shows clearly that pain depends on the brain's appraisal of threat to body tissue. Nociception is a very important informant in this regard, but it is neither sufficient nor necessary for pain.
- Many factors from sensory, psychological and social domains modulate pain.
- The effect of these factors can be understood via the principle that pain is the conscious correlate of the implicitly perceived threat to body tissues.

- It is possible to identify and estimate the impact of various factors on pain by questioning the patient carefully in the interview and by using self-report questionnaires.
- Once issues have been identified, they should be addressed in management.
- Physiotherapists have a wide range of skills and resources with which to deactivate and desensitise the nociception and pain systems at various levels – tissue, spinal and brain.
- This chapter is a starting place. We have referred the reader to more comprehensive sources of information.

When we evaluate the large amount of research in pain sciences and management, two things seem clear. Firstly, the field is progressing rapidly. With a better understanding of the complexity of pain we are encountering new opportunities for pain management. Secondly, physiotherapists are ideally trained and resourced to be intimately involved with these new developments – physiotherapists, perhaps more than any other professional group, can access every domain that contributes to pain, from the tissues to the society.

CASE STUDY: INFLAMMATORY PHASE (IMMEDIATE POST-INJURY TO 2–5 DAYS)

Introduction

Daniel slipped and fell stepping off an icy footpath on his way home from university. He hurt his ankle and, with the help of his friends, attended the hospital emergency department. A decision was made to X-ray the ankle and, as no bony injury was detected, a diagnosis of a grade 2 ankle sprain of anterior talofibular ligament (ATFL) was made. They provided him with crutches, fitted an elasticised tubular bandage and made an appointment for physiotherapy.

The physiology of pain

Activation of the nociceptive system

As Daniel's ankle was forced into inversion the high threshold mechanical force would have triggered nociceptor activity. As his body parts were deranged and uncontrolled as he slipped and fell, the potential threat to tissues would be enhanced and reflected in the central processing of the mechanical stimuli. Once injury to the tissue occurred, substances would be present in the tissues that directly trigger the nociceptors (chemical stimulus).

Sensitisation of the nociceptive/pain system

Along with the inflammation response, sensitising substances promote transmission of nociceptive information, enhancing normally painful stimulation (peripheral sensitisation/primary hyperalgesia) and, possibly, via spinal mechanisms, contributing to increased sensitivity to areas adjacent to the site of injury (central sensitisation/secondary hyperalgesia) and even creating areas that have a painful response to stimuli that is normally not painful, for example touch (allodynia).

Pain modulation via psychological and social influences

Daniel was obviously anxious enough about his ankle to seek immediate medical advice – with assistance from his concerned friends – so it could be assumed attention to the injured part and the social context may promote enhancement of his pain experience. Although as stated in the body of the chapter there is some contention about the role of attention. The medical staff's decision to refer to X-ray may have also provoked some anxiety, although the result of no bony injury should have been reassuring.

Physiotherapy appointment (day 2 post-injury)

Assessment and measurement of pain

From the initial interview (refer to Table 17.2)

- Daniel is 19 years old with no previous history of lower limb problems (personal factors).
- He tells you that his ankle swelled up 'like a balloon' and he experienced pain on the lower lateral border of fibula and was unable to weight-bear for more than four steps.
- He currently complains of pain on the lateral aspect of his foot and ankle (hyperalgesia) which worsens with movement, weight-bearing and when the quilt on his bed touches it at night (allodynia) (body function).
- He is unable to wear normal footwear because of the swelling (activity/participation; body structure).
- He lives with his parents, who are now going to have to drive him around, and is currently unable to undertake his daily routine, work part-time in a bar or play football for the university team (activity/participation).
- He tells you he is already 'fed up', that he can't do anything and is keen to 'get back to normal as soon as possible'. He is particularly anxious about losing wages from his part-time job, as he is unable to

stand for long periods of time (personal/ environmental factors).
- The hospital medical staff prescribed anti-inflammatory medication and encouraged the use of a recommended daily dose of paracetamol. He reports that this has been mostly effective.
- His VAS for pain intensity measures 40 mm for the last 24 hours and is currently 55 mm. His last analgesia was taken two hours ago. When he tries to weight-bear he describes the pain as much worse, measuring 95 mm on the VAS.
- His VAS for pain unpleasantness for the last 24 hours measures 65 mm and is currently 65 mm. The difference in this measure compared with the pain intensity measure may reflect that there is an emotional influence on his pain, perhaps related to his frustration about the situation.

In this, and subsequent appointments, it is important to ensure Daniel does not have any co-pathology that might have predicated the fall or tissue damage (see red flags) or any unhelpful beliefs or expectations about his condition (see yellow flags/ blue and black flags).

You undertake full physical examination and your findings include:

- non-weight-bearing on affected lower limb;
- significant swelling of lateral ankle extending into anterolateral foot;
- local bruising around lateral malleolus;
- movement is limited by pain and swelling, especially inversion;
- tenderness is present with palpation of lateral ankle, especially the area corresponding to ATFL.

Your observation is consistent with a grade 2 ATFL ankle sprain (Brukner and Khan 2006).

Management of pain

The aims of treatment of Daniel's pain are to:

- educate about his injury, pain and physiotherapy, i.e. establish 'helpful' beliefs/expectations;
- control the inflammatory process (RICE and non-steroidal anti-inflammatories (NSAIDs)), i.e. reduce concentration of sensitising chemicals;
- control and reduce swelling, i.e. the increase in sensitivity, the mechanical force of distension or compression of tissues caused by swelling may be triggering nociception;
- encourage early protected movement, including weight-bearing, i.e. to promote self-efficacy, reinforce pre-existing synaptic activity and mobilise tissues/ swelling.

In the acute stage, pain is a reasonable guide to safe activity. This is because the increased sensitivity (hyperalgesia) means that transmission of nociceptors is initiated well before the risk of tissue re-injury. It should be noted, however, that analgesics can confound this.

CASE STUDY: THE REMODELLING PHASE (FROM THREE WEEKS TO TWO+ YEARS)

Introduction

Sally hurt her back while mopping the floor at work four months ago. She couldn't go to work the next day and made an appointment with the local doctor. The doctor gave her some NSAIDs and told her to rest for three weeks. After three weeks, she went back to see the doctor as she was no better; he gave her stronger analgesics and sent her for physiotherapy.

There was a six-week waiting list to see the physiotherapist so Sally continued to rest as she didn't know what to do in the meantime.

When Sally saw the physiotherapist she was given several simple exercises.

The exercises made her back hurt more. Sally associated this with re-injury/ further damage so she did not keep her next physiotherapy appointment.

The doctor has told her that if she doesn't go to the physiotherapy appointments she isn't going to get better. Reluctantly, she returned to physiotherapy.

The physiology of pain

Activation of the nociceptive system

It could be that four months ago, there was a significant mechanical stimulus that triggered transmission in nociceptors and possibly a local inflammatory reaction. However, as she is now reporting pain with even simple exercises, it can be expected that the sensitisation of the system is the important factor here. Chemical stimuli, especially changes to tissue pH, may trigger nociceptors where movement is restricted, affecting blood flow and nutrition.

Sensitisation of the nociceptive/pain system

Presuming that some tissue damage occurred during the mopping incident – although tissue damage is not required for someone to feel pain – then the inflammation process would now be well and truly complete. Unless of course, there has been a re-injury, or a comorbidity such as diabetes, that prolongs tissue healing. If there is no inflammation then there are no sensitising chemicals and no swelling is likely. Enhanced spinal processing of nociceptor and somatosensory information may still be occurring

owing to descending influences (see below). Also, prolonged afferent activity may promote transmission at spinal NMDA receptors – this may be in the form of protective muscle spasm.

Pain modulation via psychological and social influences

Sally has been unable to work for four months and could be expected to be feeling quite helpless. It can be expected that she doesn't trust her back and may feel it is at risk of injury. She may also avoid movement activities (kinesiophobic) because she is anxious about the pain. Her parents may be inadvertently reinforcing this avoidance if, through their assistance, they are reinforcing the 'helpless' message (reinforcing ineffective behaviours). According to the concept of pain promoted in this chapter, Sally's sense of vulnerability is likely to reduce the inhibition of nociceptor transmission and promote mechanisms – via spinal and supra-spinal processes related to attention, expectation and mood – that reduce the activation threshold of the nociceptive system. As such, her pain is still 'real' pain but is likely to have less obvious peripheral triggers. Altered cortical representations may also have established across this time of relative inactivity.

Physiotherapy appointment (four months post-injury)

Assessment and measurement of pain

From the initial interview (refer to Table 17.2)

- Sally is 28 years old and a single mother with two young sons, aged five and seven years (personal factors).
- Owing to her pain, Sally has stopped all housework and her parents are having to do all of the shopping and care for the children, especially after school (activity/participation).
- Sally finds that resting from her normal activities was making her pain easier but now she is feeling weak, easily short of breath and generally very stiff and still experiencing pain (body structure/ function).
- She finds when her pain is less she does much more activity but then needs two days rest to recover (body function; activity/participation).
- She tearfully tells you that she is worried about her future, she is unable to meet the rent payments, she has lost her job and is only sleeping 3–4 hours per night (personal/ environmental factors; body function).
- She is unclear about what is happening in her back and why the pain is going on so long. She asks you if you think a scan would help as she thinks she isn't being taken seriously by her doctor (personal factors).
- Her VAS for pain intensity measures 60 mm for the last 24 hours and is currently 65 mm. Her last analgesic was taken two hours ago. She describes her worst pain when she bends forwards, measuring 100 mm on VAS.
- Her VAS for pain unpleasantness for the last 24 hours measures 80 mm and is currently 75 mm. Sally's higher rating for the unpleasantness of pain may relate to the affective component of pain and reflect beliefs about persisting or worsening tissue damage, or might be related to depressed mood.

As part of the assessment process, it is crucial to ensure Sally does not have any co-pathology. This includes sinister pathologies (see red flags). The information provided from the interview indicates evidence of factors that inhibit recovery and return to work (see yellow flags/ blue and black flags).

You undertake full physical examination and your findings include:

- posterior pelvic tilt with flattened lumbar spine;
- tightness in hip flexors/abdominals/latissimus dorsi;
- slow and rigid gait for age;
- movement of upper and lower limbs is limited by pain in back;
- tenderness on palpation generally and especially lumbar spine;
- high body mass index.

Management of pain

The aims of treatment/management of Sally's pain are :

- to educate about her injury, the phases of healing, pain and physiotherapy, i.e. establish 'helpful' beliefs/expectations;
- to encourage effective use of medication on a 'time-linked' basis;
- to encourage movement, including bending, i.e. to promote self-efficacy, redress synaptic activity and normalise mapping of brain representations and control;
- to incorporate graded motor imagery and tactile discrimination techniques as appropriate;
- to practise strategies for upgrading and predicting consistent levels of activity, such as quota-setting, pacing, challenging of unhelpful beliefs, relaxation/ distraction;
- employment of self-applied modalities, such as soft tissue stretching, heat and TENS, as supported by best research evidence, with advice to avoid dependence;
- vocational training.

It is not reasonable to use pain as a guide during the remodelling phase in this case because the healing process has achieved tissue integrity – movement is safe and the increased sensitivity (hyperalgesia) means that pain is felt at low, ineffective activity levels. Pain still needs to be respected. Sally should be made to feel *safe* at every opportunity, while encouraging her to set her own goals and challenges for improvement in activity level, despite pain. It is essential that progressions in her level and type of activity should be to a level and within a timeframe that she feels comfortable with.

ACKNOWLEDGEMENTS

GLM is supported by the National Health and Medical Research Council of Australia, ID 579010.

REFERENCES

Airaksinen, O., Brox, J., Cedraschi, C., 2004. European guidelines for the management of chronic non-specific low back pain. European Commision, Research Directorate General, Brussels.

Airaksinen, O., Brox, J.I., Cedraschi, C., et al., 2006. Chapter 4 – European guidelines for the management of chronic nonspecific low back pain. Eur Spine J 15, S192–S300.

Apkarian, A.V., Bushnell, M.C., Treede, R-D., et al., 2005. Human brain mechanisms of pain perception and regulation in health and disease. Eur J Pain 9 (4), 463–484.

Arntz, A., Vaneck, M., Heijmans, M., 1990. Predictions of dental pain – the fear of any expected evil is worse than the evil itself. Behav Res Ther 28 (1), 29–41.

Arntz, A., Dreessen, L., De Jong, P., 1994. The influence of anxiety on pain: Attentional and attributional mediators. Pain 56 (3), 307–314.

Asmundson, G.J., Kuperos, J.L., Norton, G.R., 1997. Do patients with chronic pain selectively attend to pain-related information?: Preliminary evidence for the mediating role of fear. Pain 72 (1–2), 27–32.

Benedetti, F., Pollo, A., Lopiano, L., et al., 2003. Conscious expectation and unconscious conditioning in analgesic, motor, and hormonal placebo/nocebo responses. J Neurosci 23 (10), 4315–4323.

Brukner, P., Khan, K., 2006. Clinical Sports Medicine. McGraw-Hill Medicine, Sydney.

Bushnell, M.C., Apkarian, A.V., 2006. Representation of pain in the brain. In: McMahon, M. (Ed.), Melzack and Wall's Textbook of Pain, fifth ed. Churchill Livingstone, Edinburgh.

Bushnell, M.C., Duncan, G.H., Hofbauer, R.K., et al., 1999. Pain perception: Is there a role for primary somatosensory cortex? Proc Natl Acad Sci USA 96, 7705–7709.

Butler, D., Moseley, G.L., 2003. Explain Pain. NOI Group Publishing, Adelaide.

Casey, K.L., Morrow, T.J., Lorenz, J., et al., 2001. Temporal and spatial dynamics of human forebrain activity during heat pain: Analysis by positron emission tomography. Journal of Neurophysiology 85 (2), 951–959.

Coudeyre, E., Rannou, F., Tubach, F., et al., 2006. General practitioners' fear-avoidance beliefs influence their management of patients with low back pain. Pain 124 (3), 330–337.

Craig, A.D., 2002. How do you feel? Interoception: the sense of the physiological condition of the body. Nat Rev Neurosci 3 (8), 655–666.

Crombez, G., Eccleston, C., Van den Broeck, A., et al., 2004. Hypervigilance to pain in fibromyalgia: the mediating role of pain intensity and catastrophic thinking about pain. Clin J Pain 20 (2), 98–102.

Damasio, A.R., 2000. Descartes' Error: Emotion, Reason, and the Human Brain. Quill, New York.

De Koninck, Y., 2009. Signaling events and ionic mechansims disrupting spinal inhibition in neuropathic pain. In: Castro-Lopes, J. (Ed.), Current Topics in Pain: 12th World Congress on Pain, IASP Press: Seattle, pp. 139–164.

Eccleston, C., Crombez, G., 2005. Attention and pain: merging behavioural and neuroscience investigations. Pain 113 (1–2), 7–8.

Fields, H.L., 2000. Pain modulation: expectation, opioid analgesia and virtual pain. Prog Brain Res 122, 245–253.

Fields, H., Basbaum, A., Heinricher, M., 2006. CNS mechanisms of pain modulation. In: McMahon, S.B., Koltzenburg, M. (Eds.), Textbook of Pain, Elsevier, London, pp. 125–143.

Fisher, K., Johnston, M., 1997. Validation of the Oswestry Low Back Pain Disability Questionnaire, its sensitivity as a measure of change following treatment and its relationship with other aspects of the chronic pain experience. Physiother Theory Pract 13 (1), 67–80.

Flor, H., 2003. Cortical reorganisation and chronic pain: Implications for rehabilitation. J Rehabil Med 35, 66–72.

Flor, H., 2009. Extinction of pain memories: Importance for the treatment of chronic pain. In: Castro-Lopes, J. (Ed.), Current Topics in Pain: 12th World Congress, IASP Press, Seattle, pp. 221–244.

Flor, H., Denke, C., Schaefer, M., et al., 2001. Effect of sensory discrimination training on cortical reorganisation and phantom limb pain. Lancet 357 (9270), 1763–1764.

Flor, H, Nikolajsen, L, Jensen, TS, 2006. Phantom limb pain: a case of maladaptive CNS plasticity? Nat Rev Neurosci 7 (11), 873–881.

Fordyce, W.E., 1970. Operant conditioning as a treatment method in management of selected chronic pain problems. Northwest Med 69 (8), 580–581.

Fordyce, W.E., 1973. An operant conditioning method for managing chronic pain. Postgrad Med 53 (6), 123–128.

French, S.D., Cameron, M., Walker, B.F., et al., 2006. A Cochrane review of superficial heat or cold for low back pain. Spine 31 (9), 998–1006.

Furlan, A.D., Imamura, M., Dryden, T., et al., 2008. Massage for low-back pain. Cochrane Database Syst Rev (4), CD001929.

Gifford, L., Thacker, M., Jones, M, 2006. Physiotherapy and pain. In: McMahon, S.B., Koltzenburg, M. (Eds.), Textbook of Pain, Elsevier, London, pp. 603–618.

Goldingay, S., 2006a. Communication and assessment: The skills of information gathering. In: Gifford, L. (Ed.), Topical Issues in Pain, vol. 5. CNS Press, Falmouth.

Goldingay, S., 2006b. Communication and assessment: What are the issues for physiotherapists? In: Gifford, L. (Ed.), Topical Issues in Pain, vol. 5. CNS Press, Falmouth.

Goubert, L., Vervoort, T., Crombez, G., 2009. Pain demands attention from others: the approach/avoidance paradox. Pain 143 (1–2), 5–6.

Gracely, R.H., Dubner, R., Deeter, W.R., et al., 1985. Clinicians' expectations influence placebo analgesia. Lancet 1 (8419), 43.

Greenhalgh, S., Selfe, J., 2006. Red Flags. A Guide to Identifying Serious Pathology of the Spine. Churchill Livingstone Elsevier, Oxford.

Grimmer-Somers, K., Vipond, N., Kumar, S., et al., 2009. A review and critique of assessment instruments for patients with persistent pain. J Pain Res 2, 21–47.

Harman, K., 2000. Neuroplasticity and the development of persistent pain. Physiother Can 52 (1), 64–71.

Harris, A.J., 1999. Cortical origin of pathological pain. Lancet 354 (9188), 1464–1466.

Haythornwaite, J.A., 2009. It's a belief. It's an appraisal. It's coping. No, it's catastrophising. In: Castro-Lopes, J. (Ed.), Current Topics in Pain: 12th World Congress on Pain, IASP Press, Seattle, pp. 271–287.

Jensen, M.P., Karoly, P., Huger, R., 1987. The development and preliminary validation of an instrument to assess patients' attitudes toward pain. J Psychosom Res 31 (3), 393–400.

Jones, L., 2007. An introduction to current concepts of pain. In: Partridge, C. (Ed.), Recent Advances in Physiotherapy. Wiley-Blackwell, London, pp 135–141.

Kelly, K.G., Cook, T., Backonja, M-M., 2005. Pain ratings at the thresholds are necessary for interpretation of quantitative sensory testing. Muscle Nerve 32 (2), 179–184.

Keltner, J.R., Furst, A., Fan, C., et al., 2006. Isolating the modulatory effect of expectation on pain transmission: A functional magnetic resonance imaging study. J Neurosci 26 (16), 4437–4443.

Kendall, N.A.S., Linton, S.J., Main, C., 1998. Psychosocial yellow flags for acute low back pain: 'yellow flags' as an analogue to 'red flags'. Eur J Pain 2 (1), 87–89.

Klages, U., Kianifard, S., Ulusoy, O., et al., 2006. Anxiety sensitivity as predictor of pain in patients undergoing restorative dental procedures. Community Dent Oral Epidemiol 34 (2), 139–145.

Kouyanou, K., Pither, C.E., Rabe-Hesketh, S., et al., 1998. A comparative study of iatrogenesis, medication abuse, and psychiatric morbidity in chronic pain patients with and without medically explained symptoms. Pain 76 (3), 417–426.

Lautenbacher, S., Huber, C., Schofer, D., et al., 2010. Attentional and emotional mechanisms related to pain as predictors of chronic postoperative pain: a comparison with other psychological and physiological predictors. Pain 151 (3), 722–731.

Legrain, V., Damme, S.V., Eccleston, C., et al., 2009. A neurocognitive model of attention to pain: behavioral and neuroimaging evidence. Pain 144 (3), 230–232.

Levine, F.M., De Simone, L.L., 1991. The effects of experimenter gender on pain report in male and female subjects. Pain 44 (1), 69–72.

Maihofner, C., Handwerker, H.O., Neundorfer, B., et al., 2004. Cortical reorganization during recovery from complex regional pain syndrome. Neurology 63 (4), 693–701.

McCracken, L.M., 2007. A contextual analysis of attention to chronic pain: what the patient does with their pain might be more important than their awareness or vigilance alone. J Pain 8 (3), 230–236.

McCracken, L.M., Zayfert, C., Gross, R.T., 1992. The Pain Anxiety Symptoms Scale: development and validation of a scale to measure fear of pain. Pain 50, 67–73.

McQuay, H.J., Moore, R.A., Eccleston, C., et al., 1997. Systematic review of outpatient services for chronic pain control. Health Technol Assess 1 (6), i–iv, 1–135.

Melzack, R., 1975a. The McGill Pain Questionnaire: major properties and scoring methods. Pain 1 (3), 277–299.

Melzack, R., 1975b. Prolonged relief of pain by brief, intense transcutaneous somatic stimulation. Pain 1 (4), 357–373.

Melzack, R., 1990. Phantom limbs and the concept of a neuromatrix. Trend Neurosci 13 (3), 88–92.

Melzack R., Wall P.D., 1965. Pain mechanisms: a new theory. Science 150 (699), 971–979.

Merskey, H., Bogduk, N., 1994. Classification of Chronic Pain, second ed. IASP Press: Seattle.

Meyer, R, Ringkamp, M., Campbell, J.N., et al., 2006. Peripheral mechanisms of cutaneous nociception. In: McMahon, S.B., Koltzenburg, M. (Eds.), Textbook of Pain, fifth ed. Elsevier, London, pp. 3–35.

Miller, R., Kori, S., Todd, D., 1991. The Tampa scale for kinesiphobia. Unpublished report, Tampa, FL.

Moerman, D., 2002. Meaning, Medicine and the 'Placebo Effect'.

Cambridge University Press, Cambridge.

Moseley, G.L., 2002. Combined physiotherapy and education is effective for chronic low back pain. A randomised controlled trial. Aust J Physioth 48, 297–302.

Moseley, G.L., 2003. Joining forces – combining cognition-targeted motor control training with group or individual pain physiology education: a successful treatment for chronic low back pain. J Man Manip Therap 11, 88–94.

Moseley, G.L., 2004. Evidence for a direct relationship between cognitive and physical change during an education intervention in people with chronic low back pain. Eur J Pain 8 (1), 39–45.

Moseley, G.L., 2006a. Do training diaries affect and reflect adherence to home programs? Arthritis Rheum 55 (4), 662–664.

Moseley, G.L., 2006b. Making sense of S1 mania – are things really that simple? In: Gifford, L. (Ed.), Topical Issues in Pain, vol. 5. CNS Press, Falmouth, pp. 321–340.

Moseley, G.L., 2007a. Reconceptualising pain according to its underlying biology. Phys Ther Rev 12, 169–178.

Moseley, G.L., 2007b. Using visual illusion to reduce at-level neuropathic pain in paraplegia. Pain 130 (3), 294–298.

Moseley, G.L., Gandevia, S.C., 2005. Sensory-motor incongruence and reports of 'pain'. Rheumatology 44 (9), 1083–1085.

Moseley, G.L., Arntz, A., 2007. The context of a noxious stimulus affects the pain it evokes. Pain 133, 64–71.

Moseley, G.L., Wiech, K., 2009. The effect of tactile discrimination training is enhanced when patients watch the reflected image of their unaffected limb during training. Pain 144 (3), 314–319.

Moseley, G.L., Nicholas, M.K., Hodges, P.W., 2004. A randomized controlled trial of intensive neurophysiology education in chronic low back pain. Clin J Pain 20 (5), 324–330.

Moseley, G.L., Gallace, A., Spence, C., 2009. Space-based, but not arm-based, shift in tactile processing in complex regional pain syndrome and its relationship to cooling of the affected limb. Brain 132 (Pt 11), 3142–3151.

Naveteur, J., Mars, F., Crombez, G., 2005. The effect of eye orientation on slowly increasing pain. Eur J Pain 9 (1), 79–85.

Nicholas, M.K., 2007. The pain self-efficacy questionnaire: Taking pain into account. Eur J Pain 11 (2), 153–163.

Nicholas, M., Sharp, T.J., 1997. Cognitive-behavioral programs: theory and application. Int Anesthesiol Clin 35 (2), 155–170.

Nicholas, M.K., Tonkin, L., 2004. Application of cognitive-behavioural principles to activity-based pain management programs. In: Refshauge, K., Gaff, E. (Eds.), Musculoskeletal Physiotherapy: Clinical Science and Evidence Based Practice. Elsevier, Oxford, pp. 277–293.

Nicholas, M.K., Wilson, P.H., Goyen, J., 1991. Operant-behavioural and cognitive-behavioural treatment for chronic low back pain. Behav Res Ther 29 (3), 225–238.

Nicholas, M.K., Siddal, P., Tonkin, L., et al., 2007. Manage Your Pain. ABC Books, Sydney.

Ottawa Panel, 2004. Ottawa Panel Evidence-Based Clinical Practice Guidelines for Electrotherapy and Thermotherapy Interventions in the Management of Rheumatoid Arthritis in Adults. Phys Ther 84(11), 1016.

Pleger, B., Tegenthoff, M., Ragert, P., et al., 2005. Sensorimotor returning in complex regional pain syndrome parallels pain reduction. Ann Neurol 57 (3), 425–429.

Ploghaus, A., Becerra, L., Borras, C., et al., 2003. Neural circuitry underlying pain modulation: expectation, hypnosis, placebo. Trend Cogn Sci 7 (5), 197–200.

Poiraudeau, S., Rannou, F., Baron, G., et al., 2006. Fear-avoidance beliefs about back pain in patients with subacute low back pain. Pain 124 (3), 305–311.

Poleshuck, E.L., Green, C.R., 2008. Socioeconomic disadvantage and pain. Pain 136 (3), 235–238.

Pollo, A., Amanzio, M., Arslanian, A., et al., 2001. Response expectancies in placebo analgesia and their clinical relevance. Pain 93 (1), 77–84.

Roland, M., Morris, R., 1983. A study of the natural history of back pain. Part I: Development of a reliable and sensitive measure of disability in low-back pain. Spine 8 (2), 141–144.

Rommel, O., Gehling, M., Dertwinkel, R., et al., 1999. Hemisensory impairment in patients with complex regional pain syndrome. Pain 80 (1–2), 95–101.

Sandkühler, J., 2009. The role of inhibition in the generation and amplification of pain. In: Castro-Lopes, J. (Ed.), Current Topics in Pain: 12th World Congress on Pain. IASP Press, Seattle, pp. 53–72.

Sawamoto, N., Honda, M., Okada, T., et al., 2000. Expectation of pain enhances responses to nonpainful somatosensory stimulation in the anterior cingulate cortex and parietal operculum/posterior insula: an event-related functional magnetic resonance imaging study. J Neurosci 20 (19), 7438–7445.

Schmelz, M., Schmidt, R., Weidner, C., et al., 2003. Chemical response pattern of different classes of C-nociceptors to pruritogens and algogens. J Neurophysiol 89 (5), 2441–2448.

Schupp, C.J., Berbaum, K., Berbaum, M., et al., 2005. Pain and anxiety during interventional radiologic procedures: Effect of patients' state anxiety at baseline and modulation by nonpharmacologic analgesia adjuncts. J Vasc Intervent Radiol 16 (12), 1585–1592.

Schweinhardt, P., Kalk, N., Wartolowska, K., et al., 2008. Investigation into the neural correlates of emotional augmentation of clinical pain. Neuroimage 40 (2), 759–766.

Sullivan, M.J.L., Bishop, S.R., Pivik, J., 1995. The pain catastrophizing scale: development and validation. Psycholog Assoc 7 (4), 524–532.

Sullivan, M.J.L., Thorn, B., Haythornthwaite, J.A., et al., 2001. Theoretical perspectives on the relation between catastrophizing and pain. Clin J Pain 17 (1), 52–64.

Tang, J., Gibson, S.J., 2005. A psychophysical evaluation of the

relationship between trait anxiety, pain perception, and induced state anxiety. J Pain 6 (9), 612–619.

Tillman, D.B., Treede, R.D., Meyer, R.A., et al., 1995. Response of C fibre nociceptors in the anaesthetized monkey to heat stimuli: estimates of receptor depth and threshold. J Physiol 485 (Pt 3), 753–765.

Turk, D.C., Melzack, R. (Eds.), 2001. Handbook of Pain Assessment, second ed. Guildford Press, New York.

van Griensven, H., 2005. Pain in Practice: Theory and Treatment Strategies for Manual Therapists. Elsevier, London.

van Tulder, M., Kovacs, F., Muller, G., et al., 2002. European guidelines for the management of low back pain. Acta Orthopaed Scand 73, 20–25.

Verhoeven, K., Crombez, G., Eccleston, C., et al., 2010. The role of motivation in distracting attention away from pain: an experimental study. Pain 149 (2), 229–234.

Vernon, H., Mior, S., 1991. The Neck Disability Index: a study of reliability and validity. J Manipulative Physiol Ther 14 (7), 409–415.

Vlaeyen, J.W., Linton, S.J., 2000. Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. Pain 85 (3), 317–332.

Waddell, G., Newton, M., Henderson, I., et al., 1993. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. Pain 52 (2), 157–168.

Wager, T.D., 2005. Expectations and anxiety as mediators of placebo effects in pain. Pain 115 (3), 225–226.

Wall, P.D., 1999. Pain: The Science of Suffering. Weidenfeld & Nicolson, London.

Watkins, L., Maier, S., 2000. The pain of being sick: implications of immune-to-brain communication for understanding pain. Ann Rev Psychol 51, 29–57.

Watson, P., Kendall, N.A.S., 2000. Assessing psychosocial yellow flags. In: Gifford, L. (Ed.), Topical Issues in Pain, vol. 2. CNS Press, Falmouth.

Watson, P., Main, C., Smeets, R.J.E.M., 2010. Basics, management, and treatment of low back pain. In: Mogil, J (Ed.), Pain 2010. An Updated Review. Refresher Course Syllabus. IASP Press, Seattle, pp. 361–380.

WHO (World Health Organization), 2002. Towards a common language for functioning, disability and health, http://www.who.int/classifications/icf/training/icfbeginnersguide.pdf; accessed September 2012.

Wiech, K., Ploner, M., Tracey, I., 2008. Neurocognitive aspects of pain perception. Trends Cogn Sci 12 (8), 306–313.

Williams, D.A., Robinson, M.E., Geisser, M.E., 1994. Pain beliefs: Assessment and utility. Pain 59 (1), 71–78.

Wittink, H., Carr, D.B., 2008. Outcomes and effective pain treatment. Pain: Clin Updates 16 (1).

Woolf, C.J., Costigan, M., 1999. Transcriptional and posttranslational plasticity and the generation of inflammatory pain. Proc Natl Acad Sci USA 96(14), 7723–7730.

Chapter 18

Acupuncture in physiotherapy

Andrew Bannan

A BRIEF HISTORY OF ACUPUNCTURE

While it is widely assumed that acupuncture originated in China, some evidence of its existence in central Europe was discovered in 1991 when a frozen corpse, subsequently named Oetsi the iceman and estimated to be the remains of a 5000-year-old male, was found with tattoos largely consistent with acupuncture points (Dorfer et al. 1999). Furthermore, the location of these points appeared consistent with an acupuncture prescription for the treatment of the lower back and abdominal conditions which study of the remains would suggest this man suffered from (Moser et al. 1999). In China, the discovery of sharpened bones and stones (known in Chinese as *bian* or *bian shi*) dating to the Neolithic period, or 8000 years ago, has been suggested to be evidence of the practice of a form of acupuncture, but it is more likely that these sharpened tools were, at least initially, used for blood-letting and the drainage of abcesses (Ma 1992; Basser 1999; Unschuld 1985). It is thought that acupuncture practice may well have had origins in the once near-universal practice of medical blood-letting which began in Ancient Greece and was used to remove what was diagnosed as stagnant blood, a description sometimes used in traditional Chinese medicine to describe the state of qi flow in ill health which may be beneficially affected by needling.

Regardless of its specific origins, the classic text *Huang Di Neijing* (translated as *Inner Classic of Huang Di)* introduced the practical and theoretical foundations of what subsequently became known as acupuncture (Ramey and Buell 2004). Commonly referred to as *The Yellow Emperor's Classic of Internal Medicine,* this text is judged to be approximately 2000 years old and is presented as a series of questions and answers between the then Emperor, Huang Di and his physician Chi-Po (Ma 1992; Kaptchuk 2002; White and Ernst 2004; Baldry 2005). Over the centuries acupuncture developed and was used alongside herbal medicine, diet, massage and the use of heat therapies, including the burning of moxa or moxibustion in China (Ma 1992). A sixteenth century acupuncture specialist cast bronze figurines to assist in the location of acupuncture points (Ma 1992). It was not until 1601 that the publication of *The Great Compendium of Acupuncture and Moxibustion* by Yang Ji-Zhou formulated what we now recognise as acupuncture practice detailing 361 points (Kaplan 1997). Throughout the early development of acupuncture it is important to note that traditional Chinese medicine approaches and treatment philosophies were based upon subjective perceptions and in the absence of medical dissection or surgical exploration (Basser 1999; Unschuld 1985; White and Ernst 2004).

The practice of acupuncture was adopted by Japan and Korea in the sixth century and by Vietnam between the eighth and tenth centuries (Baldry 2005). France led the way in Europe, adopting the practice of acupuncture after Jesuit missionaries returned with reports of its practice in the sixteenth century. In 1683, the first medical treatise to appear in the West on 'acupuncture' (a term coined by its author from the Latin *acus* = needle and *pungere* = to prick) was written by the physician Willem Ten Rhijne (Bivins 2000; Baldry 2005). Throughout the eighteenth century, in both Europe and America, doctors practised acupuncture, formulating a Western rationale based on the scientific discoveries of their day (Kaptchuk 2000).

Ironically, this was at a time when acupuncture use was falling out of favour in China (Baldry 2005). By the mid-nineteenth century acupuncture had become unfashionable and politically incorrect in the west, and in China its use was restricted to rural areas following a decree by the Daoguang Emperor citing it as an impediment to medical advancement (Ernst and White 1999). This decree effectively meant that acupuncture was no longer officially recognised by The Imperial Medical Institute and was, along with other forms of traditional Chinese medicine, banned in 1929 in favour of modern scientific medicine (Ma 1992).

Acupuncture in the twentieth century

In 1949 with Chairman Mao Tse-Tung as head of the communist revolution and the establishment of The People's Republic of China, acupuncture, along with other forms of traditional Chinese medicine experienced a revival. This resurgence in traditional Chinese medicine was largely for political reasons and linked to the Cultural Revolution in China (Unschuld 1985). In fact, it was at this time that the existing divergent forms of therapeutic intervention in China were consolidated into what we now refer to as traditional Chinese medicine (Birch and Kaptchuk 1999). Alongside this, research into acupuncture was promoted in China which later led to Han's work into the effects acupuncture may have on neurotransmitter release (Han and Terenius 1982). Owing to China's political isolation this renewed enthusiasm for traditional Chinese medicine went unnoticed by the West until the early 1970s.

In preparation for Richard Nixon's 1972 visit, Henry Kissinger visited China in 1971. Among his press entourage was a man who was surgically treated for acute appendicitis while in China and received postoperative acupuncture for abdominal pains (Reston 1971). His report of this care in the New York Times was the catalyst for a rush of interest by US physicians in seeing the surgical benefits of Chinese acupuncture first hand (Diamond 1971). Perhaps the most enduring and controversial of these first-hand experiences was when Dr Isadore Rosenfeld observed open heart surgery in a young female patient ostensibly using acupuncture points in her ear (auricular acupuncture) with electrical stimulation (electro-acupuncture) in place of standard anaesthetics. It now seems clear that what Dr Rosenfeld observed was, at best, an exaggeration of the analgesic affects of auricular electro-acupuncture and, at worst, and more likely, an elaborate hoax with much of the analgesic effect being attributable to low doses of midazolam, droperidol and fentanyl. Nonetheless, this and the experiences of other visiting physicians, galvanised interest in acupuncture analgesia among the medical community at the time. Unfortunately, reports of these experiences were presented more as sensationalist journalistic pieces than objective academic works and much of the hype regarding the potential benefits of acupuncture this created was not well founded. Indeed, a recent systematic review by Lee and Ernst (2005) concluded that the evidence for acupuncture analgesia in surgery was inconclusive.

Figure 18.1 Model of traditional acupuncture points.

ACUPUNCTURE FROM A TRADITIONAL CHINESE MEDICINE PERSPECTIVE

Acupuncture is an intervention involving the insertion and manipulation of fine needles into the body to achieve a therapeutic effect. From a traditional Chinese medicine perspective the anatomical sites at which these needles are inserted are specified and these points are located along channels (*mai* in Chinese) that have come to be known as meridians in the West (in part owing to their similarity to geographical meridian lines). It is worth noting that ancient texts demonstrate that these meridians were developed from clinical experience with moxibustion and not acupuncture (Harper 1998). There are 12 principle meridians with both superficial and deep representations, and a number of so-called extraordinary meridians. Again, from a traditional Chinese medicine perspective, these meridians are believed to have specific effects on the physiology of body organ systems and are named accordingly, for example Lung (LU), Heart (HT), Pericardium (PC), Stomach (ST), Large Intestine (LI), Small Intestine (SI) and Bladder (BL) meridians (Figures 18.1 and 18.2).

The insertion of acupuncture needles and their stimulation either manually or via electrical stimulation is believed to effect the flow of qi, pronounced 'chi', and commonly translated as vital energy, life force or spirit, while a literal translation of the Chinese character for qi is 'vapors rising from food' (Basser 1999). It is believed in traditional Chinese medicine that by effecting qi flow along the meridians a person's overall health and well-being can be beneficially influenced. In order to

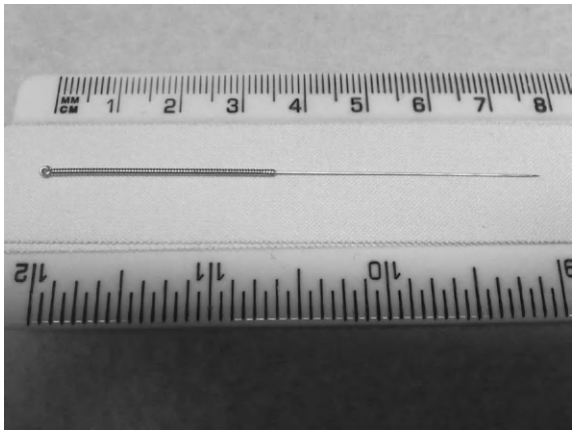


Figure 18.2 Typical acupuncture needle.

effectively produce this therapeutic effect it is commonly believed that needling stimulation is required to produce a specific sensation know in Chinese as de qi and described as a deep heavy aching sensation which may propagate along the needled meridian.

Diagnosis in traditional Chinese medicine conceptually views the health status of an individual as a microcosm of nature and thus could be viewed as a human meteorological report (Kaptchuk 2002). Linked to this are the concepts of five fundamental universal elements and that of balancing a person's yin and yang with those of nature, terms originally denoting the shaded and sunny aspect of a hill respectively. These terms of yin and yang are also used to classify acupuncture meridians, the former being on the inner aspect of a limb and the latter on the outer aspect.

In traditional Chinese medicine, the body is viewed as being comprised of functional systems or *zang-fu* in Chinese. Though not directly associated, each system is named according to an organ. The *zang* systems are linked with the solid, *yin* organs, for instance the kidneys, while the *fu* systems are linked with the hollow *yang* organs, for instance the stomach.

The individual acupuncture points are named and numbered according to the meridian along which they are located, for example LI4, ST36. Points are located on an individual's body in relation to tendons, muscles and bony points, and a system of proportional measurements using the 'cun' as its base measurement which is the width of that individual's interphalangeal joint of the thumb (Cheng 1987). Palpation is also viewed as being of great importance in locating points to needle. Historically, and in keeping with the traditional Chinese medicine view of health being related to a numerical and holistic paradigm, 365 points were described to reflect the days of the year without any further objective basis (Lun 1975). In addition to the overall effects which needling these points can have on an individual's health and organ function, in traditional Chinese medicine specific acupuncture points are anecdotally believed to have specific effects on certain conditions, for example PC6 for nausea and vomiting, GB37 for conditions related to vision and BL60 for lower back pain (Maciocia 2005). These points are commonly referred to as 'empirical' points.

In addition to these classical meridian points, needle insertion can be directed locally to the symptomatic area or into points of maximal tenderness referred to as ah-shi (translated as 'oh yes'/'that's it') points. Interestingly, the Chinese character used to denote an acupuncture point can also mean 'hole' suggesting that acupuncture points may be viewed as points of access to structures deeper in the body (Langevin and Yandow 2002).

WESTERN MEDICAL ACUPUNCTURE

In 2000, the British Medical Association recommended the integration of acupuncture into practice (Silvert 2000). On the face of it, Western medical acupuncture may appear very similar to traditional Chinese medicine acupuncture. Needles are inserted often both local to the symptomatic region of the body and more distally along the arm or leg, often with points in the hand or foot. Once the needles are in place they are typically manipulated by hand or by electrical stimulation over the course of a treatment lasting anywhere from 5–30 minutes. The points are typically named using traditional Chinese medicine terminology and often empirical points are added to the points used. The key differences between Western and traditional Chinese medicine acupuncture is in the patient assessment, expectation and in the intention behind the treatment itself. While a traditional Chinese medicine approach would typically assess a patient's 'qi balance and flow' by taking a history and using observation of the tongue, eyes and specific palpation of the pulse, a Western approach would seek to make a clinical diagnosis based on pathology using history and clinical assessment findings. In terms of the treatment itself, a traditional Chinese medicine practitioner would seek to alter qi movement and flow by needling, whereas a Western practitioner would attempt to stimulate specific neurochemical and both connective and contractile tissue responses by needling. In addition, a traditional Chinese medicine practitioner would chose points according to meridians with a consideration of the effects this may have on organ system physiology. Although Western acupuncture may still use classic traditional Chinese medicine nomenclature to specify the points used often no consideration is given to the potential effects on organ systems, for instance in the case of using points along the bladder meridian to treat lower back pain. Instead, the local, segmental, extra-segmental and central neurological effects are what concern a Western acupuncture practitioner.

Prevalence of acupuncture use

Acupuncture use by general practitioners (GPs) in the UK is increasing and it is now likely to be the most prevalent of all complementary therapies. Thomas et al. (2001) reported that in 1998 approximately 7% of English adults had received acupuncture treatment at some point. Within National Health Service (NHS) chronic pain clinics, acupuncture is estimated to be offered in 84% of cases (Woollam and Jackson 1998). According to the Acupuncture Association of Chartered Physiotherapists (AACP) there are approximately 5000 AACP-registered physiotherapists now working in the NHS and in private practice in the UK.

Safety of acupuncture

As with any physiologically active and invasive therapeutic intervention, considerations of not only the effectiveness but also of the safety of acupuncture are necessary. The mere fact that acupuncture has been practised for centuries does not provide sufficient evidence of its safety. Historically, autoclave procedures were employed for sterilising needles and it was not until reports of acupuncture-related cross infection, such as hepatitis B (Kent et al. 1988) coupled in no small way with the outbreak of AIDS in the 1980s, that single-use needles were advocated. It is clear that reports of adverse events and more serious complications attributed to acupuncture during the 1970s and 1980s highlight the improvements in acupuncture safety that have been made in the past three decades. Further reports of acupuncture-associated hepatitis B transmission were, however, reported in the 1990s (Rosted 1997; White 1999). The latter of these reports not only recommended the use of single-use, disposable needles, but also the vaccination of all acupuncture practitioners against hepatitis B. During the 1990s it was proposed that a theoretical risk of transmitting variant Creutzfeldt-Jacob Disease existed even with autoclaved needles, further supporting the use of sterile single-use needles.

In 1999, Peuker et al. reported on the importance of acupuncture practitioners having a sound knowledge of surface and deep anatomy, at least to the depth of needling which can vary from a few millimetres to several centimetres. In this report, potential sites of traumatic lesions by needling are divided into those affecting the thoracic viscera, abdominal viscera, peripheral and central nervous system (CNS) structures. In the thoracic region attention is drawn to the presence of a sternal foramen in a small, but not insignificant, proportion of the population level with the fourth intercostal space. Needling perpendicularly through this foramen if present when using the acupuncture point referred to as conception vessel 17 (CV17) carries the risk of causing a cardiac tamponade.

The other potential complication in the thoracic region is injury to the lungs or pleura through needling. Needling in the parasternal region or along the midclavicular line to a depth of 10–20 mm has been shown to be sufficient to reach the lungs in cadaveric study. Numerous cases of pneumothorax induced by acupuncture have been reported (White 2004). Points which have resulted in injury to these structures include Stomach 11 and 12 (ST11, ST12). Caution should be exercised when needling Stomach 13 (ST13), Lung 2 (LU2) and Kidney 27 (KID27). Kidney (KID22–KID27), Stomach (ST12–ST18) and Bladder (BL41–BL54) points should all be needled bearing in mind the depth of the lung at these sites. As far as the abdominal viscera are concerned, few reports of complications have been published apart from rare lesions of the intestine and bladder, plus a report of a foreign body in a kidney identified as an acupuncture needle.

Peripheral nerve injuries may occur in cases of patients with atypical median nerve courses at the wrist. Points to note in this instance are Pericardium 6 and 7 (PC6, PC7) and Lung 8 (LU8). In the lower limb a number of points are located over peripheral nerve pathways. For instance, Bladder 39 (BL39) and potentially Gall Bladder 34 (GB34), the common peroneal nerve and Bladder 40 (BL40), and the tibial nerve and popliteal vessels.

Four reports of blood vessel injury are briefly detailed by Peuker et al. (1999). These involved a partially thrombosed pseudoaneurysm adjacent to the costocervical artery (Fujiwara et al. 1994), a false aneurysm of the popliteal artery following deep needling of Bladder 40 (BL40) (Lord and Schwartz 1996), a deep vein thrombophlebitis in the upper calf (Blanchard 1991) and anterior compartment syndrome in the upper calf in a patient on anticoagulant therapy (Smith et al. 1986).

A series of articles by Peuker and Cummings (2003a, 2003b, 2003c) expanded upon this work of examining anatomy as it pertains to acupuncture. These three articles divided the body into three sections: the head and neck, the chest, back and abdomen, and the upper and lower limbs, and detailed the pertinent neuromusculoskeletal anatomy to guide acupuncturists in targeting the intended target and avoiding neurovascular targets.

A review by Ernst and Sherman (2003) concluded that in almost all cases poor practice and lack of adherence to recommended hygiene procedures was responsible for serious adverse reactions to acupuncture. This fact obviously supports the continued regulation of acupuncture practitioners by organisations such as the British Medical Acupuncture Society (BMAS) and the AACP in the UK.

ACUPUNCTURE RESEARCH IN THE TWENTIETH CENTURY

Throughout the 1970s much of the medical and scientific community attributed the benefits of acupuncture to the placebo effect. However, this belief failed to account for the benefits of acupuncture in young children and in

animals. In order to satisfy standards of Western scientific rigor and understandable scepticism regarding acupuncture and its effects, clinical research was required. Western evidence-based medical research into acupuncture has since largely dismissed traditional Chinese medicine concepts of qi and meridians. In their place neurochemical and anatomical models have been proposed based on the best evidence from research studies. At the extreme this Western scientific approach is typified by Felix Man's assertion in the 1970s that 'Acupuncture points and meridians in the traditional sense, do not exist' (Mann 2004).

Principally, this research investigated the analgesic effects of acupuncture. Initially, the best theory to explain acupuncture analgesia was the gate control theory of pain proposed by Melzack and Wall in the 1960s (Melzack and Wall 1965). The fundamental basis of this model was that a counter stimulus could effectively supersede the noxious stimulus at the spinal cord level thus suppressing the nociceptive pathway.

During the late 1970s extensive research was performed by Bruce Pomeranz, alone and with others. Pomeranz used research evidence to outline a sequence of neurochemical events following the insertion of an acupuncture needle. At spinal cord level the acupuncture stimulus was believed to stimulate the release of dynorphin and encephalin leading to the attenuation of noxious transmission. At mid-brain level a further release of encephalin affected a descending inhibitory pathway back to the spinal cord. Finally, Pomeranz proposed that at the level of the hypothalamus the stimulation of release of beta-endorphin into the mid-brain by the arcuate nucleus and pituitary re-enforced this descending inhibition (Pomeranz and Chui 1976; Pomeranz et al. 1977; Pomeranz 1978). Subsequent research focussed on the potential effects acupuncture may have on the release of opioids, such as endorphin. One of the most significant and seminal of these pieces of research was carried out in Beijing and looked into the neurochemistry of acupuncture analgesia (Han and Terenius 1982). This study found that acupuncture could affect the release of neurotransmitters, especially opioid peptides, which could explain its analgesic effects.

Throughout the 1970s and 1980s frequent but ultimately inconclusive attempts were also made to find anatomical correlates for acupuncture points. These studies pointed to the endings of a variety of sensory nerves (Ciczek et al. 1985), areas of dense neuro-vascularity (Bossy 1984) and motor end-plates (Liu et al. 1975; Gunn et al. 1976; Dung 1984) as potential anatomical target sites for needling. Another line of research looked at the physiology of acupuncture points. On many occasions skin conductance has been evidenced to be higher at acupuncture points when compared with adjacent skin (Reichmanis et al. 1976; Comunetti et al. 1995). Unfortunately, inadequate statistical testing, control and subject numbers have adversely affected the quality of these studies.

Brain imaging acupuncture research

With the advent of positron emission tomography (PET) scanning and, in particular, functional magnetic resonance imagery (fMRI) in the 1990s, there was a new method of investigating the cortical effects of needling in acupuncture. For instance, it was possible to study the effects of needling on activity in areas of the brain believed to be associated with the processing and perception of pain. While such research took many forms and investigated acupuncture from a variety of different perspectives, perhaps the most talked about study was published in 1998 and sought to correlate brain activity with an empirical point associated with visual disorders (Cho et al. 1998). This study focussed on an acupuncture point located on the lateral aspect of the foot referred to as Bladder 67 (BL67) which, according to traditional Chinese medicine theory, is an empirical point used in the treatment of eye disorders (Kaptchuk 2000; Stux et al. 2003). The authors observed fMRI activity in the brain stimulated by needling BL67 and fMRI activity stimulated by visual light stimulation. A correlation was found between the specific areas of cortical activation leading the authors to claim that this supported traditional Chinese medicine theory regarding this empirical point. However, a retraction was published in 2006 by the authors of the original article aside from three: J.P. Jones, J.B. Park and H.J. Park. The other five authors stated that they 'no longer agree with the results' of the original article and conclude from research carried out in the intervening years that there is no research evidence to support point specificity in acupuncture.

By the turn of the century, research had revealed evidence that acupuncture had a role in the regulation of a variety of physiological functions, protection against infection and as an analgesic (WHO 1999). More specific study revealed that needle manipulation at the point known as Large Intestine 4, or LI4, could modulate activity as seen on fMRI in the limbic system of the brain and in subcortical regions (Hui et al. 2000). This finding could go some way towards explaining some of the anecdotally reported multisystem clinical effects of acupuncture treatment.

The World Health Organization's (WHO) review of acupuncture evidence in 1999, which limited its scope to randomised controlled and controlled clinical trials with adequate subject numbers, advocated the use of acupuncture for pain relief, especially in chronic cases. This is because for chronic pain states, acupuncture has been shown to be equally effective as morphine but without the potential side effects. Of note is the similarity between conditions where acupuncture is advocated and those where an element of central sensitisation is suggested, such as osteoarthritis, some forms of headaches, fibromyalgia and musculoskeletal disorders with generalised pain and hypersensitivity, such as chronic lower back pain.

Acupuncture use is supported in the WHO report for the management of headaches and a variety of musculoskeletal conditions such as neck pain or cervical spondylitis (David et al. 1998), fibromyalgia (Deluze et al. 1992), common extensor tendinopathy of the elbow (Haker and Lundeberg 1990; Molsberger and Hille 1994), knee osteoarthritis (Berman et al. 1999) and chronic lower back pain (Lehmann et al. 1986).

In the case of rheumatoid arthritis the WHO report cites evidence supporting the benefits of acupuncture treatment to address the pain, inflammation and immune system components of this auto-immune condition (WHO 1999). Furthermore, and in summary, the report presents some convincing evidence supporting the use of acupuncture in the treatment of traumatic and postoperative pain (Jiao 1991), as an adjunct in stroke rehabilitation (Johansson et al. 1993) in reducing exercise-induced asthma (Fung et al. 1986), as an anti-emetic (Vickers, 1996), and in some gynaecological and obstetric conditions (Helms 1987; Chen 1997).

Of note is the fact that this WHO report included reports published in Russian, Chinese, Japanese, German, Danish and Indonesian, and loosely defined acupuncture as a treatment to include standard needling, electro- and auricular acupuncture, moxibustion and, in some research, included the use of cupping. It is worth pointing out that research published in Chinese was second only to that published in English and this fact does deserve consideration when interpreting the findings owing to potential culturally driven variations in treatment expectation.

Acupuncture research since the turn of the twenty-first century

Perhaps the most significant development in acupuncture research since the turn of the century was a series of observational and controlled studies, now commonly referred to as the GERAC trials. These trials were instigated by insurance companies across Germany in response to the increasing expense of claims where acupuncture was part of the treatment. These trials led to the publication of studies involving much larger subject numbers and investigated the use of acupuncture in the treatment of migraines (Linde et al. 2005), tension type headache (Melchart et al. 2005), chronic neck pain (Willich et al. 2006), chronic low back pain (Brinkhaus et al. 2006), and osteoarthritis of the hip and knee (Witt et al. 2006).

The fundamental outcome of these trials was that German insurance companies were sufficiently convinced by the evidence for acupuncture in the treatment of knee osteoarthritis and chronic lower back pain that they would fund it as part of treatment. In the case of the other conditions investigated, the insurance companies were not convinced that acupuncture provided sufficient benefits over and above usual care or sham acupuncture procedures. The National Institute for Health and Clinical Excellence 2009 recommendations for treating lower back pain also supported the use of acupuncture treatment.

Interestingly, a Cochrane Review by Linde et al. (2009) of acupuncture for tension type headaches stated that 'acupuncture could be a valuable non-pharmacological tool in patients with frequent episodic or chronic tension-type headaches' in light of research published since 2006, such as Endres et al. (2007) and Jena et al. (2008). The Cochrane Review into acupuncture for prevention of migraines by Linde et al. (2009) concluded that on the basis of recent research that 'Acupuncture should be considered a treatment option for patients willing to undergo this treatment.' Benefits of acupuncture were seen to be as good as pharmacological treatment with fewer side effects; however, no statistically significant difference was noted between the effects of 'true' versus 'sham' acupuncture procedures. This furthers the debate as to whether depth of needling and location of needling, for instance, are of importance over and above the benefits of intent and expectation.

A 2010 review into acupuncture for peripheral osteoarthritis (including osteoarthritis of the hip, knee and hands) concluded that while the benefits of acupuncture versus sham procedures were statistically significant they were not, however, felt to be clinically significant. This finding may well be a result, somewhat ironically perhaps, of larger subject numbers involved in research studies. When compared with waiting list care the benefits of acupuncture were found to be both statistically and clinically significant. However, in all cases it was felt that the benefits of acupuncture could reasonably be attributed to intention and/or expectation – in short a placebo effect (Manheimer et al. 2010).

As far as more recent evidence for acupuncture treatment of neck pain is concerned, a 2007 review by Trinh et al. suggests that moderate evidence exists to support the use of acupuncture use for treating chronic neck pain (Trinh et al. 2007). Among other areas of recent research interest are upper limb, lower back and pelvic pain in pregnancy, chronic knee pain, myofascial pain and fibromyalgia.

Unfortunately, all too often acupuncture research has lacked sufficient methodological rigor, adequate subject blinding or sufficient subject numbers to adequately investigate this multifaceted intervention. Much of the research into acupuncture has been dogged by a lack of an adequate placebo with which to compare 'true acupuncture'. No clear agreement exists as to what is an adequate dose of acupuncture. Trials have used 'minimal needling', non-acupuncture point needling or sham acupuncture needling, and 'placebo needling' using retractable non-penetrating needles to attempt to remedy this situation (Streitberger and Kleinhenz 1998; White et al. 2003). However, the near universal result is that these supposed control or placebo conditions have, at least in terms

of statistical evaluation, essentially matched the 'true acupuncture' in terms of its effects. Therefore, in acupuncture research, as in medical trials, the placebo condition remains a clinically active intervention. This has led sceptics to brand acupuncture as being no better than a placebo and advocates to claim that the control conditions used were therapeutically active interventions. A key consideration that can be overlooked here is the fact that a placebo effect, which can be dismissed, is, nonetheless, a real treatment effect. As in pain science, assuming we have consigned Cartesian dualism to history, this means that the so-called control conditions used in research are clinically active and produce effects on the brain (Campbell 2009).

What now appears clear is that even superficial pressure via sham needling as part of a therapeutic intervention can have significant effects on the brain's limbic system underlying the importance of a patient's expectation and beliefs in any treatment response (Pariente et al. 2005). This concept and, more specifically, the role that C tactile fibres may play in the outcome of all manual therapy is worth bearing in mind in both research and clinical settings (Campbell 2006a). It is accepted that portions of the limbic system of the brain form part of the pain neuromatrix but also are commonly referred to as the emotion and reward centre. Following on from this there appears to be a suggestion that superficial needling may be more effective in conditions such as headaches and migraines with a large affective component versus conditions with largely sensory components, such as knee arthritis (Lund and Lundberg 2006).

Despite the clear difficulties associated with constructing sound clinical trials to assess the benefits of acupuncture, some review articles have recently supported its use in three disparate conditions. A meta-analysis by Manheimer et al. (2005) supported the use of acupuncture in treating lower back pain; Ezzo et al. (2005) supported acupuncture use in the treatment of chemotherapy-induced nausea and vomiting; and White et al. (2007) found evidence that acupuncture was more effective than placebo in treating chronic knee pain.

The current Western scientific understanding of acupuncture

So what do we now understand is happening from a neurochemical point of view when acupuncture is used as a treatment? Biomedical acupuncture theory recognises that acupuncture modulates central, peripheral and autonomic nervous system activity (Wu et al. 1999). Chemical mediators in the CNS have been implicated in the mechanism of acupuncture since a study by the Research Group of Acupuncture Anaesthesia in Peking Medical College (1974) took cerebrospinal fluid of rabbits treated with acupuncture and effected increased pain thresholds in rabbits by injecting this into their cerebral ventricles. Early research into acupuncture mechanisms revealed that stimulation of local nerves was at least partly responsible for its effects. Han and Terenius (1982) noted that acupuncture analgesia was blocked by procaine infiltration of points and Wang et al. (1985) demonstrated that acupuncture needling stimulated nerve action potentials. It has also been shown that it is not possible to elicit acupuncture analgesia in paraplegic or hemiplegic patients which underlines the importance of intact afferent pathways (Han and Terenius 1982).

It has been shown more recently that stimulation of small diameter type II and III myelinated sensory nerve fibres in muscles may have a role in the initiation of acupuncture analgesia. Interestingly, the needling sensation known as 'de qi' has been shown to occur in tandem with action potentials typical of those propagated by and along these type II and III fibres.

As far as the potential anti-inflammatory effects of acupuncture are concerned, beta-endorphin and adrenocorticotropic hormone release into the circulation may produce systemic effects such as changes in T-lymphocyte levels plus natural killer cells, and an increase in the production of anti-inflammatory corticosteroids by the adrenal glands respectively (Takeshige et al. 1992; Sato et al. 1997; Lundeberg 1999).

What consistently appears in the scientific literature are studies linking the effects of acupuncture to modulation of activity in the limbic system of the brain. This may mean that as far as anti-nociceptive effects, acupuncture alters pain perception using a pathway common to placebo treatments, hypnosis and, potentially, manipulations (Campbell 2006b). The limbic system plays a leading role in maintaining homeostasis in the body (Craig 2002, 2003). This fact, coupled with the fact that acupuncture modulates activity within the limbic system, may explain acupuncture's ability to stimulate or inhibit metabolic processes as required for a return to a healthy state.

Recent study has identified that the local analgesic and potentially the anti-inflammatory effects of acupuncture are mediated by adenosine A1 receptors (Goldman et al. 2010).

> *Somatic sensory stimulation activates impulse relays in the CNS, which then alter processing and output.*
>
> Robinson (2007)

It should be noted that fear and anxiety states consistent with sympathetic arousal may reduce acupuncture parasympathetic actions.

Western correlates of traditional Chinese medicine paradigms

The search for anatomical and physiological correlates of acupuncture points and meridians has produced many

plausible theories but has largely been inconclusive. One of the more convincing theories was that an overlap existed between acupuncture points and myofascial trigger points. However, despite the specific criteria for what constitutes a trigger point, some difference of opinion remains as to whether trigger points are muscular or fascial entities. Kawakita and Okada (2006) describe both acupuncture points and trigger points as 'sensitised polymodal receptors' which may be treated with a variety of interventions. A number of authors have proposed a potential correlation between connective tissue planes and acupuncture meridians (Larson 1990; Oschman 1996; Ho and Knight 1998; Langevin and Yandow 2002; Dorsher 2009; Fung 2009). For instance, Fung (2009) explored the connective tissue interstitial fluid system, mechanotransduction and mast cell degranulation, while Ho and Knight (1998) investigated the liquid crystalline collagen fibres of connective tissue in relation to acupuncture meridians. This line of work may fit well with acupuncture points as portals to deeper tissues are often referred to as fascia and connective membranes in historical texts (Matsumoto and Birch 1988).

The work of Helen Langevin has provided further experimental evidence to support the potential relationships between connective tissue and acupuncture mechanisms. An article by Langevin and Yandow (2002) demonstrated an 80% overlap between acupuncture point locations in the upper limb and inter or intramuscular septa. This has led to the proposal that needling at acupuncture points targets interstitial loose connective tissue and not muscles, and that this tissue forms a continuous dynamic signalling network responsible for the effects of acupuncture. This theory echoes the recent reconceptualisation of functional anatomy and work into myofascial meridians and tensegrity model in the field of manual therapies by Thomas Myers (2009).

Scientific study of the biomechanical response to needling has demonstrated that the measured force required to extract needles (indicative of needle grasp which can accompany eliciting a 'de qi' response) was greater at acupuncture points than non-acupuncture points (Langevin et al. 2001). In addition, Langevin et al. (2001) and Langevin et al. (2002) demonstrated evidence that the needle grasp was a response not by muscular tissue but involving connective tissues, which may modulate mechanosensitive gene expression.

What of the manipulation of the needle once it is *in situ*? The work of Langevin et al. (2004, 2006, 2007) investigated connective tissue responses to needling. Findings included a winding specific to loose areolar tissue around the needle and remodelling of fibroblasts in the connective tissue in response to needle rotation and linear manipulation. The next stage in this research was to explore the therapeutic value of stimulating deep connective tissues in this way, outwith the effects on sensory nerves, blood vessels and immune cells. There is a suggestion from research by Yu et al. (2009) that deep connective tissue winding is necessary to stimulate the afferent fibres necessary for effecting central descending inhibition. This, however, may be at odds with the consistent finding that so-called 'sham' or placebo acupuncture has effects not statistically significantly different from 'true' acupuncture.

CONDITIONS THAT MAY BE TREATED – FROM THE AACP SITE

A large range of conditions can be treated successfully by acupuncture within physiotherapy. Some are shown in Box 18.1.

Clinical implications

Unfortunately the very nature of much of the research evidence available means that individual effects and variations are watered down and lost in the statistical analysis. It is this heterogeneity that clinicians must grapple with in their daily practice and for which research all too often offers limited guidance. This is likely to be the reason why there is a lack of agreement among acupuncture practitioners in their treatments.

For instance, what constitutes an adequate dose of acupuncture in practice? Consideration needs to be given to the patient's present state from a psychological, neurological, immunological and endocrine perspective (White et al. 2008). This is similar to the considerations that may be given before prescribing rehabilitative exercises to a patient. It is also important to acknowledge the priming effect of patient expectation and belief on the reward centres of the brain. If we consider the importance of this then what clinicians say to patients as a preamble to the

Box 18.1

- Acute injuries
- Sports injuries
- Whiplash
- Back pain
- Neck pain
- Headaches
- Stress-related illnesses
- Chronic injuries
- Osteoarthritis
- Rheumatoid arthritis
- Joint pain
- Women's health
- Hormone imbalances
- Asthma
- Bronchitis
- Strokes
- Multiple sclerosis
- Migraine
- Irritable bowel
- Skin conditions
- Eczema
- Allergies
- Breathing difficulties
- Hay fever
- Chronic fatigue syndrome (ME)
- Bladder and bowel dysfunctions

use of acupuncture as a treatment and their clinical intentions may influence treatment outcomes (Pariente et al. 2005). This may be of most importance when the patient's condition has a dominance of affective versus sensory components.

Variables that need consideration from a practical treatment perspective include the number of needles used, the location and depth of needling and the nature of needle stimulation or manipulation. In addition, the duration and frequency of treatment needs to be individually tailored to each patient. A degree of active, but safe, experimentation is necessary in order to investigate each patient's dose–response relationship, again in much the same way one would assess a patient's exercise or aerobic capacity. From here, as with all clinical practice, reasoning and outcome assessment needs to be regular and patient-centred.

What can assist the clinician in treatment selection is to consider the treatment goals based on their clinical diagnosis. For instance, are you looking for effects peripherally, at spinal cord level or centrally? Consideration needs to be given to the type of pain, whether it is nociceptive, neurogenic or centrally evoked, as well as the stage of tissue repair. For instance, in acute pain a segmental effect may be required but may best be elicited by needling points within a common dermatome, myotome or sclerotome if trigger point needling is likely to be irritable and poorly tolerated.

Peripheral effects you may wish to produce include an increase in substance P, histamine and calcitonin gene related peptide (CGRP) leading to local vasodilation and modulation of the local immune response. This can be provoked by a treatment lasting from 10–20 minutes. For spinal effects the use of the relevant bladder points or huato points is suggested in order to attenuate the nociceptive input at dorsal horn level. Finally, for supraspinal effects such as stimulation of the arcuate nucleus and hypothalamus to envoke descending inhibition, peripheral points such as LI4 and LIV3 are suggested owing to their large representation on the somatosensory cortex. In this case the treatment time may be extended from 20–40 minutes and involve more aggressive needle manipulation. This may additionally produce changes in the autonomic system output and endocrine systems (Bradnum 2007). It is worth considering here that research by Fang et al. (2004) did not find evidence that the nature of needle manipulation significantly affected the neuronal response.

What of the needling response commonly known as de qi which has been synonymous with treatment effect (MacPherson et al. 2001; Kong et al. 2007)? De qi is accepted to be a feeling of spreading heavy soreness and distension during needling manipulation (Wu et al. 1999). Studies have revealed that producing a de qi response produces a pattern of reduced activity in the limbic and cerebellar brain regions that is distinct from that produced by either minimal needling or painful needling (Hui et al. 2005; Asghar et al. 2009). From a clinical standpoint, then, the consistency of needling sensation may well be a determinant of successful treatment with acupuncture. This may be especially the case if the condition being treated has a dominant affective component and treatment is targeting the pain neuromatrix in the brain. Furthermore, the treatment of painful conditions such as lower back pain with a segmental approach may differ from that of non-painful conditions, such as nausea with PC6 where greater point specificity may be justified (Lewith et al. 2005) (Figure 18.3).

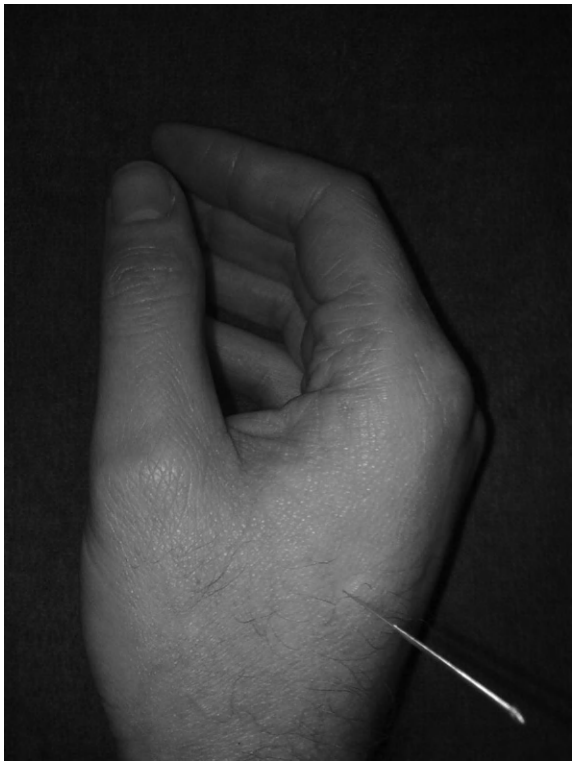


Figure 18.3 Acupuncture needle at the Large Intestine 4 (LI4) point *in situ*.

ACKNOWLEDGEMENTS

The author would like to acknowledge the assistance he received from Dr Val Hopwood (course director for the MSc in Acupuncture at Coventry University) at the first draft stage of the chapter.

REFERENCES

Asghar, A.U.R., Green, G., Lythgoe, M.F., et al., 2009. Acupuncture needling sensation: The neural correlates of deqi using fMRI. Brain Res 1315, 111–118.

Baldry, P.E., 2005. Acupuncture, trigger points and musculoskeletal pain, third ed. Churchill Livingstone, Edinburgh.

Basser, S., 1999. Acupuncture: A history. Sci Rev Altern Med 3, 34–41.

Berman, B.M., Singh, B.B., Lao, L., et al., 1999. A randomized trial of acupuncture as an adjunctive therapy in osteoarthritis of the knee. Rheumatology 38 (4), 346–354.

Birch, S., Kaptchuk, T., 1999. History, nature and current practice of acupuncture: an East Asian perspective. In: Ernst, E., White, A. (Eds.), Acupuncture: A Scientific Appraisal. Butterworth-Heinemann, Oxford, pp. 11–30.

Bivens, R.E., 2000. Acupuncture, Expertise and Cross-Cultural Medicine. Palgrave, Manchester.

Blanchard, B., 1991. Deep vein thrombophlebitis after acupuncture. Ann Intern Med 115, 748.

Bossy, J., 1984. Morphological data concerning the acupuncture points and channel network. Acupunct Electrother Res 9, 79–106.

Bradnum, L., 2007. A physiological underpinning for treatment progression of Western acupuncture. J Acupuncture Assoc Chartered Physiother Autumn 25–33.

Brinkhaus, B., Witt, C.M., Jena, S., et al., 2006. Acupuncture in patients with chronic low back pain: A randomized controlled trial. Arch Intern Med 166, 450–457.

Campbell, A., 2006a. Role of C tactile fibres in touch and emotion – clinical and research relevance to acupuncture. Acupunct Med 24 (4), 169–171.

Campbell, A., 2006b. Point specificity of acupuncture in the light of recent clinical and imaging studies. Acupunct Med 24 (3), 118–122.

Campbell, A., 2009. Hidden assumptions and the placebo effect. Acupunct Med 27 (2), 68–69.

Chen, B.Y., 1997. Acupuncture normalized dysfunction of hypothalamic-pituitary-ovarian axis. Acupuncture Electrother Res 22, 97–108.

Cheng, X., 1987. Chinese Acupuncture and Moxibustion. Foreign Language Press, Beijing.

Cho, Z.H., Chung, S.C., Jones, J.P., et al., 1998. New findings of the correlation between acupoints and corresponding brain cortices using functional MRI. PNAS 95, 2670–2673.

Ciczek, L.S.W., Szopinski, J., Skrzypulec, V., 1985. Investigations of morphological structures of acupuncture points and meridians. J Trad Chin Med 5, 289–292.

Comunetti, A., Laage, S., Schiessl, N., et al., 1995. Characterisation of human skin conductance at acupuncture points. Cell Mol Life Sci 51 (4), 328–331.

Craig, A.D., 2002. How do you feel? Interoception: the sense of the physiological condition of the body. Nat Rev Neurosci 3, 655–666.

Craig, A.D., 2003. Interoception: the sense of the physiological condition of the body. Curr Opin Neurobiol 13, 500–505.

David, J., Modi, S., Aluko, A., et al., 1998. Chronic neck pain: A comparison of acupuncture treatment and physiotherapy. Br J Rheumatol 37, 1118–1122.

Deluze, C., Bosia, L., Zirbs, A., et al., 1992. Electroacupuncture in fibromyalgia: Result of a controlled trial. BMJ 305, 1249–1252.

Diamond, E.G., 1971. Acupuncture anesthesia. Western medicine and Chinese traditional medicine. J Am Med Assoc 218, 1558–1563.

Dorfer, L., Moser, M., Bahr, F., et al., 1999. A medical report from the stone age? Lancet 354, 1023–1025.

Dorsher, P.T., 2009. Myofascial referred pain data provide physiologic evidence of acupuncture meridians. J Pain 10 (7), 723–731.

Dung, H.C., 1984. Anatomical features contributing to the formation of acupuncture points. Am J Acupunct 12, 139–143.

Endres, H.G., Böwing, G., Diener, H.C., et al., 2007. Acupuncture for tension-type headache: A multicentre, sham-controlled, patient- and observer-blinded, randomised trial. J Headache Pain 8 (5), 306–314.

Ernst, E., White, A., 1999. Acupuncture: A Scientific Appraisal. Butterworth-Heinemann, Oxford.

Ernst, E., Sherman, K.J., 2003. Is acupuncture a risk factor for hepatitis? Systematic review of epidemiological studies. J Gastroenterol Hepatol 18, 1231–1236.

Ezzo, J., Vickers, A., Richardson, M.A., et al., 2005. Acupuncture point stimulation for chemotherapy induced nausea and vomiting. J Clin Oncol 23 (28), 7188–7198.

Fang, J.L., Krings, T., Weidemann, J., Meister, I.G., Thron, A., 2004. Functional MRI in healthy subjects during acupuncture: Different effects of needle rotation in real and false acupoints. Neuroradiology 46 (5), 359–362.

Fujiwara, T., Tanohata, K., Nagase, M., 1994. Pseudoaneurysm caused by acupuncture: A rare complication. Am J Radiol 162, 731.

Fung, K.P., Wun Chow, O.K., So, S.Y., 1986. Attenuation of exercise-induced asthma by acupuncture. Lancet 2, 1419–1422.

Fung, P.C., 2009. Probing the mystery of Chinese medicine meridian channels with special emphasis on the connective tissue interstitial fluid system, mechanotransduction, cells durotaxis and mast cell degranulation. Chinese Med 4, 10.

Goldman, N., Chen, M., Fujita, T., et al., 2010. Adenosine A1 receptors mediate local anti-nociceptive effects of acupuncture. Nat Neurosci 13 (7), 883–888.

Gunn, C.C., Ditchburn, F.G., King, M.H., et al., 1976. Acupuncture loci: A proposal for their classification according to their relationship to known neural structures. Am J Chin Med 4, 183–195.

Haker, E., Lundeberg, T., 1990. Acupuncture treatment in epicondylalgia: A comparison study

of two acupuncture techniques. Clin J Pain 6, 221–226.

Han, J., Terenius, L., 1982. Neurochemical basis of acupuncture analgesia. Annu Rev Pharmacol Toxicol 22, 193–220.

Harper, D., 1998. Early Chinese Medical Literature. The Mawangdui Medical Manuscripts, first ed. Keegan Paul International, London.

Helms, J.M., 1987. Acupuncture for the management of primary dysmenorrhea. Obstet Gynecol 69, 51–56.

Ho, M.W., Knight, D.P., 1998. The acupuncture system and the liquid crystalline collagen fibers of the connective tissues. Am J Chin Med 26, 251–263.

Hui, K.K.S., Liu, J., Makris, N., et al., 2000. Acupuncture modulates the limbic system and subcortical gray structures of the human brain: Evidence from fMRI studies in normal subjects. Hum Brain Mapp 9, 13–25.

Hui, K.K., Liu, J., Marina, O., et al., 2005. The integrated response of the human cerebro-cerebellar and limbic systems to acupuncture stimulation at ST 36 as evidenced by fMRI. Neuroimage 27, 479–496.

Jena, S., Witt, C.M., Brinkhaus, B., et al., 2008. Acupuncture in patients with headache. Cephalalgia 28 (9), 969–979.

Jiao, Y., 1991. Acupuncture analgesia in treating sprain of limbs. Acupuncture Res 11(3–4), 253–254.

Johansson, K., Lindgren, I., Widner, H., et al., 1993. Can sensory stimulation improve the functional outcome in stroke patients? Neurology 43 (11), 2189.

Kaplan, G., 1997. A brief history of acupuncture's journey to the West. J Altern Complement Med 3, 5.

Kaptchuk, T.D., 2000. The Web that has no Weaver. Understanding Chinese Medicine. McGraw-Hill, New York.

Kaptchuk, T.D., 2002. Acupuncture: Theory, Efficacy, and Practice. Ann Intern Med 136, 374–383.

Kawakita, K., Okada, K., 2006. Mechanisms of action of acupuncture for chronic pain relief – polymodal receptors are the key candidates. Acupunct Med 24(Suppl.), S58–S66.

Kent, G.P., Brondum, J., Keenlyside, R.A., et al., 1988. A large outbreak of acupuncture-associated hepatitis B. Am J Epidemiol 127 (3), 591–598.

Kong, J., Gollub, R., Huang, T., et al., 2007. Acupuncture de qi, from qualitative history to quantitative measurement. J Altern Complement Med 13, 1059–1070.

Langevin, H.M., Yandow, J.A., 2002. Relationship of acupuncture points and meridians to connective tissue planes. Anat Rec 269 (6), 257–265.

Langevin, H.M., Churchill, D.L., Fox, J.R., et al., 2001. Biomechanical response to acupuncture needling in humans. J Appl Physiol 91 (6), 2471–2478.

Langevin, H.M., Churchill, D.L., Wu, J., et al., 2002. Evidence of connective tissue involvement in acupuncture. FASEB J 16, 872–874.

Langevin, H.M., Konofagou, E.E., Badger, G.J., 2004. Tissue displacements during acupuncture using ultrasound elastography techniques. Ultrasound Med Biol 30, 1173–1183.

Langevin, H.M., Bouffard, N.A., Badger, G.J., et al., 2006. Subcutaneous tissue fibroblast cytoskeletal remodeling induced by acupuncture: Evidence for a mechanotransduction-based mechanism. J Cell Physiol 207, 767–774.

Langevin, H.M., Bouffard, N.A., Churchill, D.L., et al., 2007. Connective tissue fibroblast response to acupuncture: Dose-dependent effect of bi-directional needle rotation. J Alt Complement Med 13 (3), 355–360.

Larson, D., 1990. The role of connective tissue as the physical medium for the conduction of healing energy in acupuncture and rolfing. Am J Acu 18, 251–266.

Lee, H., Ernst, E., 2005. Acupuncture analgesia during surgery: A systematic review. Pain 114, 511–517.

Lehmann, T.R., Russell, D.W., Spratt, K.F., et al., 1986. Efficacy of electroacupuncture and TENS in the rehabilitation of chronic low back pain patients. Pain 26, 277–290.

Lewith, G.T., White, P.J., Pariente, J., 2005. Investigating acupuncture using brain imaging techniques: The current state of play. Evid Based Complement Alternat Med 2 (3), 315–319.

Linde, K., Streng, A., Jürgens, S., et al., 2005. Acupuncture for patients with migraine: A randomised controlled trial. JAMA 293, 2118–2125.

Linde, K., Allais, G., Brinkhaus, B., et al., 2009. Acupuncture for tension type headache. Cochrane Database Syst Rev 21 (1), CD007587.

Liu, K.Y., Varela, M., Oswald, R., 1975. The correspondence between some motor points and acupuncture loci. Am J Chin Med 3, 347–358.

Lord, R., Schwartz, P., 1996. False aneurysm of the popliteal artery complicating acupuncture. Aust N Z J Surg 66, 645–647.

Lun, L., 1975. Acupuncture develops in the struggle between the Confucian thinking and the legalist thinking. Sci Sin 18 (5), 581–590.

Lund, I., Lundberg, T., 2006. Are minimal, superficial or sham acupuncture procedures acceptable as inert placebo controls? Acupunct Med 24 (1), 13–15.

Lundeberg, T., 1999. Effects of sensory stimulation (acupuncture) on circulatory and immune systems. In: Ernst, E., White, A. (Eds.), Acupuncture: A Scientific Appraisal. Butterworth-Heineman, Oxford, pp. 93–106.

Ma, K.W., 1992. The roots and development of Chinese acupuncture: from prehistory to early 20th century. Acupunct Med 10(Suppl.), 92–99.

Maciocia, G., 2005. The Foundations of Chinese Medicine: A Comprehensive Text for Acupuncturists and Herbalists, second ed. Churchill Livingstone, New York.

MacPherson, H., Thomas, K., Walters, S., et al., 2001. A prospective study of adverse effects and treatment reactions following 34,000 consultations with professional acupuncturists. Acupunct Med 19, 93–102.

Manheimer, E., White, A., Berman, B., et al., 2005. Meta-analysis: Acupuncture for low back pain. Ann Int Med 142 (8), 6510–6663.

Manheimer, E., Cheng, K., Linde, K., et al., 2010. Acupuncture for

peripheral joint osteoarthritis. Cochrane Database Syst Rev 1, CD001977.

Mann, F., 2004. Reinventing Acupuncture. A New Concept of Ancient Medicine. Elsevier Butterworth-Heinemann, Oxford.

Matsumoto, K., Birch, S., 1988. Hara Diagnosis: Reflections of the Sea. Paradigm Publications, Brookline.

Melchart, D., Streng, A., Hoppe, A., et al., 2005. Acupuncture in patients with tension-type headache: Randomised controlled trial. BMJ 331, 376–382.

Melzack, R., Wall, P.D., 1965. Pain mechanism: A new theory. Science 150, 971–979.

Molsberger, A., Hille, E., 1994. The analgesic effect of acupuncture in chronic tennis elbow pain. Br J Rheumatol 33 (12), 1162–1165.

Moser, M., Dorfer, L., Spindler, K., et al., 1999. Are Ötzi's tattoos acupuncture? Skin markings on the Tyrolean Iceman may have been treatment for his ills. Discover Archaeol 1, 16–17.

Myers, T.W., 2009. Anatomy Trains. Myofascial Meridians for Manual and Movement Therapists, second ed. Churchill Livingstone Elsevier, Oxford.

Oschman, J.L., 1996. Energy Review. J Bodywork Movement Therapies 1 (1), 34–39

Pariente, J., White, P., Frackowiak, R.S.J., et al., 2005. Expectancy and belief modulate the neuronal substrates of pain treated by acupuncture. Neuroimage 25, 1161–1167.

Peuker, E.T., Cummings, M., 2003a. Anatomy for the acupuncturist – Facts & fiction 1: The head and neck region. Acupunct Med 21, 2–8.

Peuker, E.T., Cummings,.M., 2003b. Anatomy for the acupuncturist – Facts & fiction 2: The chest, abdomen, and back. Acupunct Med 21, 72–79.

Peuker, E.T., Cummings, M., 2003c. Anatomy for the acupuncturist – Facts & fiction 3: Upper & lower extremity. Acupunct Med 2003 21, 112–132.

Peuker, E.T., White, A., Ernst, E., et al., 1999. Traumatic complications of acupuncture. Therapists need to know human anatomy. Arch Fam Med 8, 553–558.

Pomeranz, B., 1978. Do endorphins mediate acupuncture analgesia? Adv Biochem Psychopharmacol 18, 351–359.

Pomeranz, B., Chui, D., 1976. Naloxine blockade of acupuncture analgesia: Endorphin implicated. Life Sci 19, 1757–1762.

Pomeranz, B., Cheng, R., Law, P., 1977. Acupuncture reduces electrophysiological and behavioural responses to noxious stimuli: Pituitary is implicated. Exp Neurol 54, 172–178.

Ramey, D., Buell, P.D., 2004. A true history of acupuncture. Focus Altern Complement Ther 9, 269–273.

Reichmanis, M., Marino, A.A., Becker, R.O., 1976. DC skin conductance variation at acupuncture loci. Am J Chin Med 4 (1), 69–72.

Research Group of Acupuncture Anaesthesia, Peking Medical College, 1974. The role of some neurotransmitters of the brain in finger-acupuncture analgesia. Sci Sin 17 (1), 2–30.

Reston, J, 1971. Now about my operation in Peking. New York Times 1, 6.

Robinson, N.G., 2007. Veterinary acupuncture. An ancient tradition for modern times. Altern Complement Ther 13 (5), 259–265.

Rosted, P., 1997. Adverse reactions after acupuncture: A review. Crit Rev Phys Rehabil Med 9 (3&4), 245–264.

Sato, A., Sato, Y., Schmidt, R., 1997. The Impact of Somatosensory Input on Autonomic Functions. Springer-Verlag, Berlin.

Silvert, M., 2000. Acupuncture wins BMA approval. BMJ 321, 11.

Smith, D., Walczyk, M., Campbell, S., 1986. Acupuncture needle-induced compartment syndrome. West J Med 144, 478–479.

Streitberger, K., Kleinhenz, J., 1998. Introducing a placebo needle into acupuncture research. Lancet 352 (9125), 364–365.

Stux, G., Berman, B., Pomeranz, B., 2003. Basics of Acupuncture, fifth ed. Springer, New York.

Takeshige, C., Nakamura, A., Asamoto, S., et al., 1992. Positive feedback action of pituitary beta-endorphin on acupuncture analgesia afferent pathway. Brain Res Bull 29, 37–44.

Ten Rhijne, W., 1683. Dissertatio de arthritide: Mantissa schematic de acupunctura et orationes tres it chymiæ et botaniæ antiquitate et dignitate, de physionomia et de monstris. The Hague, Leipzig, London.

Thomas, K.J., Nicholl, J.P., Coleman, P., 2001. Use and expenditure on complementary medicine in England: A population based survey. Complement Ther Med 9 (1), 2–11.

Trinh, K., Graham, N., Gross, A., et al., 2007. Acupuncture for neck disorders. Spine 32 (2), 236–243.

Unschuld, P.U., 1985. Medicine in China: A History of Ideas. University of California, Berkeley.

Vickers, A.J., 1996. Can acupuncture have specific effects on health? A systematic review of acupuncture antiemesis trials. J Royal Soc Med 89 (6), 303–311.

Wang, K.M., Yao, S.M., Xian, Y.L., et al., 1985. A study on the receptive field of acupoints and the relationship between characteristics of needling sensation and groups of afferent fibres. Sci Sin 28, 963–971.

White, A., 1999. Hepatitis-B outbreak from acupuncture. Acupunct Med 17 (2), 149.

White, A., 2004. A cumulative review of the range and incidence of significant adverse events associated with acupuncture. Acupunct Med 22, 122–133.

White, A., Ernst, E., 2004. A brief history of acupuncture. Rheumatology 43, 662–663.

White, A., Lewith, G., Hopwood, V., et al., 2003. The placebo needle, is it a valid and convincing placebo for use in acupuncture trials? A randomised, single blind, cross-over pilot trial. Pain 106, 401–409.

White, A., Foster, N.E., Cummings, M., et al., 2007. Acupuncture treatment for chronic knee pain: A systematic review. Rheumatology 46 (3), 384–390.

White, A., Cummings, M., Barlas, P., et al., 2008. Defining an adequate dose of acupuncture using a neurophysiological approach – A narrative review of the literature. Acupunct Med 26 (2), 111–120.

WHO (World Health Organization), 1999. Acupuncture: Review and Analysis of Reports on Controlled Clinical Trials. WHO, Geneva.

Willich, S.N., Reinhold, T., Selim, D., et al., 2006. Cost-effectiveness of acupuncture treatment in patients with chronic neck pain. Pain 125, 107–113.

Witt, C.M., Jena, S., Brinkhaus, B., et al., 2006. Acupuncture in patients with osteoarthritis of the knee or hip – A randomized, controlled trial with an additional nonrandomized arm. Arthritis Rheum 54 (11), 3485–3493.

Woolan, C.H.M., Jackson, A.O., 1998. Acupuncture in the management of chronic pain. Anaesthesia 53 (6), 593–595.

Wu, M.T., Hsieh, J.C., Xiong, J., et al., 1999. Central nervous pathway for acupuncture stimulation: Localisation of processing with functional MR imaging of the brain – Preliminary experience. Radiology 212, 133–141.

Yu, X., Ding, G., Huang, H, et al., 2009. Role of collagen fibers in acupuncture analgesia therapy on rats. Connect Tissue Res 50, 110–120.

Chapter 19

Electrotherapy

Tim Watson

INTRODUCTION

Electrotherapy has been a component of physiotherapy practice since the early days of the profession. Modern electrotherapy use deserves to be evidence-based and the modalities used judiciously. When used appropriately, electrotherapy modalities have a demonstrable capacity to achieve significant benefit. Used unwisely, they will either do no good at all or, worse still, aggravate the clinical condition. In addition to the delivery skills of each modality, there is a critical skill in making the appropriate clinical decision as to which modality to use and when.

It is commonly argued that electrotherapy has little, or no, value in modern physiotherapy practice, and that it lacks an evidence base and so should be 'left out' of the treatment options. It is not uncommon to find whole clinics or departments that have decided not to deliver electrotherapy any longer on this premise. As you will see from the information in this chapter (which is not a full review of the evidence – just a brief summary of it), there is a substantial evidence base behind these modalities and if we were to deliver 'evidence-based therapy', then electrotherapy *would* be included – not for everything – but certainly for a significant range of musculoskeletal and associated presentations.

With regard the mechanisms by which each modality achieves its effects, it is important to realise that it is not the modality *per se* which brings about the therapeutic benefit. The applied energy stimulates or induces a physiological response. It is the physiological response which, in turn, brings about the therapeutic effects. The key to the application of electrotherapy is the relationship between these concepts. The therapist working with an electrotherapy modality is using, or manipulating, the physiological changes in order to achieve the desired effect. It may seem to be a pedantic argument, but it is a critical point. The outcome of the therapy is achieved through physiological manipulation and this concept applies to the application of all electrotherapy modalities.

A further very important concept is that the electrotherapy intervention is only a component of the overall treatment package. It is rarely appropriate for a patient to receive electrotherapy in isolation. It is most effectively combined with a range of manual therapies, exercise, advice and education. The elements of the treatment package need to be complementary. Careful construction of treatment programmes will enable the most effective outcome. It may be that the electrotherapy components are only utilised for the first few sessions or, indeed, only for the sessions later in a series. Electrotherapy is not an essential component for all patients and should only be used when and where appropriate.

Electrotherapy versus electrophysical agents

There is a general shift to move away from the term 'electrotherapy' toward a more encompassing term of 'electrophysical agents' (EPAs). This is largely to be welcomed in that electrotherapy, in the strictest sense of the term, would only apply to those modalities which involved the delivery of electrical, and possibly also electromagnetic, energies (e.g. transcutaneous electrical nerve stimulation (TENS), interferential therapy (IFT)). Ultrasound, light, vibration and various heat therapies, for example, would not fit this more narrow definition. As a term, EPA is certainly more inclusive and thus a more accurate reflection of the wide range of modalities employed in physiotherapy (Watson 2010). A general shift away from the term electrotherapy and towards a more common use of EPAs is anticipated over the next few years. The term electrotherapy in the context of this chapter will be used in its broad, inclusive sense rather than as a strict definition of the energy applied.

Scope

The aim of this chapter is to enable the reader to identify the key issues in electrotherapy, using commonly employed modalities as examples. It does not purport to fully examine or explain the evidence for every modality, and there are many 'modalities' that will hardly be mentioned. This does not mean that they are unimportant or worthless: it is a realistic reflection of the complexity of modern electrotherapy practice and the limits of what can be achieved in a single chapter. Further details are available in the standard texts (e.g. Robertson et al. 2006; Watson 2008b; Belanger 2010) and reference will be made in this chapter to further useful research articles and resources. In terms of clinical practice, the most widely used modalities in the UK are ultrasound, IFT and TENS, with pulsed shortwave with laser and neuromuscular electrical stimulation (NMES) following close behind. Microcurrent therapy, shockwave therapy, low intensity pulsed ultrasound (LIPUS) and some new radio frequency (RF) applications have been added to this chapter as evidenced and emerging interventions.

MODEL OF ELECTROTHERAPY

Electrotherapy modalities follow a very straightforward model that is presented below. The model (Figure 19.1) identifies that the delivery of energy from a machine or device is the starting point of the intervention. The energy delivery to the tissues results in a change in one or more physiological events – some of which are very specific while others are multifaceted or more general. The capacity of the applied energy to influence physiological events is key to the process. The physiological shift that results from the energy delivery is used in practice to generate what is commonly referred to as therapeutic effects.

The clinical application of the model is best achieved by what appears to be a reversal of this process. Starting with the patient and their problems, identified from the clinical assessment, the treatment priorities can be established and the rationale for the treatment determined. Having established the therapeutic target (or aim), move one step back through the model and identify which physiological events/processes need to be activated or stimulated in order to achieve the outcome. Once the required physiological changes have been identified, moving one step further back will enable a modality decision to be made. This is based on the existing evidence relating to which modalities stimulate which physiological effects. If there is no electrotherapy modality that is capable of achieving the intended effects, then electrotherapy would have no rational use in the management of this particular patient. The effects of electrotherapy appear to be modality dependent. This is a critical decision, in that each modality has a limited subset of effects which are fundamentally different from another modality. It is certainly not the case that some modalities are universally 'better' than others – it is the case that some modalities are more effective at achieving particular therapeutic effects.

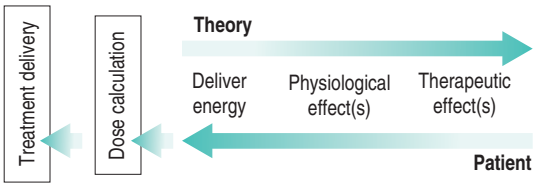


Figure 19.1 A generic model of electrotherapy (electrophysical agents).

Having identified the modality that is best able to achieve the effects required, the next clinical stage is to make a 'dose' selection. Not only is it critical to apply the right modality, but it needs to be applied at the appropriate 'dose' in order for maximal benefit to be achieved. There is a substantial and growing body of evidence that the same modality can be applied at different doses and the results will be different (Watson 2010). An example might be the use of ultrasound energy. Applied at a low 'therapeutic' dose, it can stimulate tissue repair and healing. Applied at a much higher dose (high intensity focussed ultrasound (HIFU)) it can be used to ablate tumour tissue. The energy form is the same, but by varying the applied 'dose' the outcome is clearly different.

One might argue that this is an extreme example, which in some ways it is, but the point is that the effects of the therapy are both modality- and dose-dependent. There are 'therapeutic windows' in electrotherapy (as there are in almost all therapeutic interventions) and in order to achieve the 'best' outcome, it is essential to get as close to this window as one possibly can.

This fundamental model used to explain electrotherapy could be applied to many interventions, including drug therapy, manual therapy and exercise therapy. All involve the use of an intervention in order to achieve a physiological shift or change. It is this change that is the therapeutic tool. The treatment is just a tool to stimulate the physiological change and electrotherapy is therefore little different from manual therapy or any other intervention except that a 'machine' is employed as an initiator of the physiological shift. It is a tool that when applied at the right time at the right dose and for the right reason has the capacity to be beneficial. Applied inappropriately, it is not at all surprising that is has the capacity to achieve nothing or, in fact, to make things worse. The skilful practitioner uses the available evidence combined with experience to make the best possible decision, taking into account the psychosocial and holistic components of the problem – it is not a simple reductionist solution.

THERAPEUTIC WINDOWS

Windows of opportunity are topical in many areas of therapy and medical practice, and are not a new phenomenon. It has long been recognised that the 'amount' of a treatment is a critical parameter. This is no less true for electrotherapy than for other interventions. There are literally hundreds of research articles that illustrate that the same modality applied at a different 'dose' will produce a different outcome.

Given the research evidence, there appear to be several aspects to this issue. Using a very straightforward model, there is substantial evidence, for example, that there is an *amplitude* or *strength* window. An energy delivered at a particular amplitude has a beneficial effect while the same energy at a lower amplitude may have no demonstrable effect (too low) or a tissue destruction effect (too high in therapy terms). Laser therapy offers an obvious example: one level will produce a distinct cellular response while a higher dose can be considered to be destructive. Karu (1987) demonstrated and reported these principles related to laser energy and the research produced since have served to reinforce the concept (e.g. Vinck et al. 2003). Further examples of amplitude windows can be seen easily in the work of Hill et al. (2002), Reher et al. (2002), Miller and Gies (1998), Cleary (1987) and Pereira et al. (2002), and have been more extensively reviewed in Watson (2010).

Along similar lines, 'frequency windows' are also apparent. A modality applied at a specific frequency or pulsing regime might have a measurable benefit, while the same modality applied using a different pulsing profile may not appear to achieve equivalent results. Examples can be found in many articles, including Young and Dyson (1990a), Young and Dyson (1990b) and Sontag (2000).

Electrical stimulation frequency windows have been proposed and there is clinical and laboratory evidence to suggest that there are frequency-dependent responses in clinical practice. TENS applied at frequency X appears to have a different outcome to TENS applied at frequency Y in an equivalent patient population. Studies by Sluka et al. (2006), Han et al. (1991) and Palmer et al. (1999) illustrate the point.

Assuming that there are likely to be more than two variables to the real world model, some complex further work needs to be invoked. There is almost certainly an *energy* or *time-based* window (e.g. Hill et al. 2002) and then another factor based on treatment frequency (number of sessions a week or treatment intervals). Work continues to identify the more and less critical parameters for each modality across a range of clinical presentations and a more detailed review of these concepts can be found in Watson (2010).

ELECTROTHERAPY MODALITY GROUPING

No matter which classification of the numerous electrotherapy modalities is adopted, it can easily be criticised. The groupings used in Figure 19.2 are one way of looking at the scope of electrotherapy, although it is not presented as the 'right' model.

The division into three main subgroups – electrical stimulation, thermal modalities and non-thermal modalities – is the theme that will be followed in this chapter. The *electrical stimulation modalities* have a common mode of action in that their primary effect will be on nerve (and, in some circumstances, muscle) tissue. Commonly employed forms of electrical stimulation include TENS, IFT, various forms of muscle stimulation (e.g. NMES, functional electrical stimulation (FES)) and many others which will not be considered in any detail in this chapter. Microcurrent therapy is 'different' in that its primary mode of action is to influence tissue repair rather than stimulate nerve, and thereby falls into an overlap zone between categories.

The *thermal modalities* group includes various forms of heating that have been used for many years in therapy, including infrared therapy, conductive heating, wax therapy, hot packs and the 'deeper' heating modalities – shortwave diathermy (SWD), other RF applications and microwave diathermy (MWD). The use of the heating modalities has diminished in clinical practice over recent years and much as some of the interventions employed in the past may lack evidence, there are compelling reasons to keep heat therapies in the clinical repertoire and there are areas where the use of heat-based therapies is likely to re-emerge as a strongly evidence-based intervention. Examples of modern heat therapy applications include Michlovitz et al. (2004), Usuba et al. (2006), Mayer et al. (2006), and Leung and Cheung (2008).

The *non-thermal modalities* are grouped together on the basis that if delivered at sufficiently high levels, any of these modalities *could* produce a significant or even destructive heating effect in the tissues. If they are delivered at sufficiently low dose, they are considered to be 'non-thermal' in their primary effect. This is somewhat misleading in that any energy delivered to the tissues which is subsequently absorbed will achieve a heating effect. The non-thermal label is derived from the fact that there is no gross thermal change and the patient is not able to perceive a thermal effect. More properly, these should possibly be referred to as *microthermal* or *subthermal* modalities. Ultrasound, pulsed shortwave therapy (PSWT) and laser therapy fall most obviously into this group, and various forms of magnetic therapy which are gaining ground in the literature would also be best suited to this area, as would some of the developing low-power RF

Electrical Stimulation Agents/Modalities	Thermal Agents/ Modalities	Non-Thermal Agents/ Modalities
Transcutaneous Electrical Nerve Stimulation (TENS)	Infrared Irradiation (IRR)	[Pulsed] Ultrasound
Interferential Therapy (IFT)	Shortwave Diathermy (SWD)	Low Intensity Pulsed Ultrasound (LIPUS)
Neuromuscular Electrical Stimulation (NMES)	Microwave Diathermy (MWD)	[Pulsed] Shortwave Therapy (PSWT)
Functional Electrical Stimulation (FES)	Other RF Therapies	[Pulsed] Laser Therapy (LLLT/LILT)
Russian Stimulation	**Contact Heating** *Hydrocollator Packs* *Wax Therapy* *Balneotherapy (water/spa/whirlpool)* *Mud (and associated) Therapy*	Low Intensity RF Applications
Diadynamic Therapy	Fluidotherapy	**Magnetic Therapies** *Pulsed Magnetic Therapy* *Static Magnetic Therapy* *Induced Magnetic Therapy*
Iontophoresis	Ultrasound	Microcurrent Therapies
H Wave Therapy: Action Potential Stimulation (APS)	Laser	
Rebox Therapy	Cryotherapy/Cold/ Ice/Immersion	
High Voltage Pulsed Galvanic Stimulation (HVPGS)		
Microcurrent Therapy		

Figure 19.2 Electrotherapy (EPA) modalities classification.

applications. The current clinical use of these modalities is primarily directed at enhancing the process of healing and tissue repair. Ultrasound presents a dilemma in that some practitioners use it as a modality which is employed with the deliberate intention of heating the tissues, although the evidence would support its 'non-thermal' use over and above its thermal application. In the context of this chapter, microcurrent therapy will be included in the electrical stimulation section (though it could fall into either group) and shockwave therapy will be included in the non-thermal group in that it is an energy delivery which is not intended as a thermal agent and is not an electrical stimulation modality – thus illustrating that any classification system fails at some point!

ELECTRICAL STIMULATION MODALITIES

General principles of electrical stimulation

The general principles of electrical stimulation (ES) in the context of commonly employed electrotherapy modalities focusses on the use of electrical currents (usually in the form of discrete pulses) to initiate action potentials in nerves. This would be true for modalities like TENS, NMES in its various forms and interferential therapy (though this modality does not employ discrete pulses). There are other forms of intervention which employ alternative mechanisms – one of which is iontophoresis (which uses a direct or pulsed direct current to enhance the delivery of a chemical substance or drug through the skin) and microcurrent-type therapies which are delivered at a level that is insufficient to stimulate a nerve action potential but which do appear to have an effect on the repair responses in wounds and damaged tissues.

Nerve action potentials

Assuming that the majority of ES modalities work by means of nerve activation, a brief examination of how this is achieved would be beneficial. A nerve in its resting state is said to be '*polarised*'. When an action potential is transmitted along a nerve, the membrane at the point of the action potential is momentarily *depolarised* before returning to its normal state (*repolarisation*). Essentially, the employment of an electrical current or pulse is as a means of initiating an action potential along the course of the nerve. Once the action potential has been initiated by this exogenous (external to the body) signal, then it will continue along the nerve (whether sensory or motor) in the normal fashion: the electrical stimulator is simply an initiator of the activity. If the nerve is stimulated ten times a second, then there will be ten action potentials a second

initiated. If stimulated 100 times a second, predictably, it will fire 100 times a second. There are some constraints to this relationship based on refractory periods and threshold potentials which are beyond the scope of this chapter but are usefully reviewed in most standard electrotherapy texts (Robertson et al. 2006; Watson 2008b).

The nerve being stimulated is largely unable to differentiate between different types of electrical stimulation. It is simply responding to an external stimulus and will fire accordingly. The main difference between stimulators is that they are set to have an optimal effect on particular nerve types such that a TENS machine will be the most efficient device to use to achieve stimulation of a sensory nerve and a NMES device will be optimal in stimulating motor nerves. TENS will, of course, stimulate both sensory and motor nerves; however, it is more efficient in having an effect on the sensory nerves. The same would be true for a NMES stimulator – the effect is not exclusively a motor one, it is just that the motor effect is dominant.

Bearing these principles in mind, the commonly employed ES modalities will be considered in brief with their primary clinical applications described. There are many alternative and additional widespread uses of these modalities that are evidenced, but are beyond the scope of this overview chapter. More comprehensive considerations can be found in the references supplied, as well as in the major texts.

Transcutaneous electrical nerve stimulation (TENS)

TENS is a method of electrical stimulation which aims primarily to provide a degree of pain relief (symptomatic) by specifically exciting sensory nerves and thereby stimulating either the pain gate mechanism and/or the opioid system (Walsh 1997; Sluka and Walsh 2003; Johnson 2008). Strictly speaking, any form of electrical stimulation applied with surface electrodes that stimulates nerves can be referred to as TENS but in clinical practice the term is most commonly employed in the context identified above.

The different methods of applying TENS relate to these different physiological mechanisms. Success is not guaranteed with TENS. The percentage of patients who obtain pain relief will vary, but would typically be in the region of ≥70% for acute-type pains and ≥60% for more chronic pains. Both of these are significantly 'better' than the placebo effect.

The technique is non-invasive and has few side effects when compared with drug therapy. Modern TENS devices may be analogue (Figure 19.3a,b) or digital (Figure 19.3c,d) in design. Although their outputs are essentially the same, their control systems differ. TENS is normally included in the multimodal stimulators (Figure 19.3f). Most TENS applications are now made using self-adhesive, pre-gelled electrodes (Figure 19.3e) which have several advantages, including a lower allergy incidence, a reduced cross-infection risk, easier application and lower overall cost, although some practitioners retain the older, impregnated carbon rubber electrode systems. Specialist TENS variations include 'maternity' TENS (Figure 19.3g), which have a simple to operate 'boost' function.

Machine parameters

The main parameters (or settings) on a TENS machine are those that are influential in terms of sensory nerve stimulation, which is the primary aim of the modality. The location of these controls on typical analogue and digital TENS machines is illustrated in Figure 19.4.

The current intensity (A) (strength) will typically be in the range of 0–80 mA, though some machines may provide higher outputs. Although this is a small current, it is sufficient because the primary target for the therapy is the sensory nerves and so long as sufficient current is passed through the tissues to depolarise these nerves, the modality can be effective.

The pulse rate (B) will normally be variable from about 1 or 2 pulses per second (pps) up to 200 pps or more. To be clinically effective, it is suggested that the TENS machine should cover a range of 2–150 Hz.

In addition to the stimulation rate, the duration (or pulse width) of each pulse (C) may be varied from about 40 to 250 microseconds (μs). Recent evidence would suggest that this is possibly a less important control than the intensity and frequency. These are short duration pulses which are effective because sensory nerves have a relatively low threshold and will respond well to short duration, rapidly changing pulses. There is generally no need to apply a prolonged pulse in order to force the nerve to depolarise.

Most modern machines will offer a BURST mode (D) in which the pulses will be delivered in bursts or 'trains', usually at a rate of 2–3 bursts per second. Finally, a modulation mode (E) may be available which employs a method of making the pulse output less regular and therefore minimising the accommodation effects which are often encountered with this type of stimulation.

Machines most commonly offer a dual channel output, i.e. two pairs of electrodes can be stimulated simultaneously. In some circumstances this can be a distinct advantage, though it is interesting that most patients and therapists tend to use just a single channel application.

The pulses delivered by TENS stimulators vary between manufacturers, but tend to be asymmetrical biphasic modified square wave pulses. By employing biphasic pulses it means that there is usually no net direct current component, thus minimising any skin reactions owing to the build up of electrolytes under the electrodes.

Figure 19.3 TENS machines and electrodes: (a) analogue TENS (Body Clock); (b) analogue TENS (Chatanooga); (c) digital TENS (TensCare); (d) digital TENS (Natures Gate); (e) self-adhesive electrodes (TensCare); (f) multimodal stimulator including TENS (Enraf); (g) obstetric/maternity TENS (Body Clock).

Mechanism of action

The type of stimulation delivered by the TENS unit aims to excite (stimulate) the sensory nerves and, by so doing, activate specific natural pain relief mechanisms. For convenience, if one considers that there are two primary pain relief mechanisms which can be activated – the pain gate mechanism and the endogenous opioid system – the variation in stimulation parameters used to activate these two systems will be briefly considered.

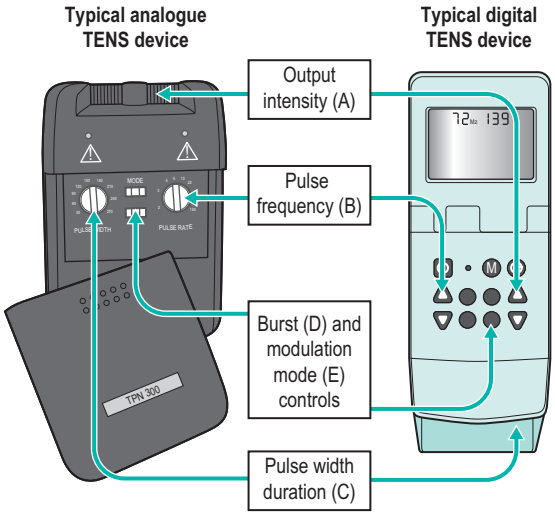


Figure 19.4 Typical analogue and digital TENS machine controls.

Pain relief by means of the pain gate mechanism primarily involves activation (excitation) of the Aβ sensory fibres, thus reducing the transmission of the noxious stimulus from the 'c' fibres through the spinal cord and on to the higher centres. The Aβ fibres appear to respond preferentially when stimulated at a relatively high rate (in the order of 80 or 90–130 Hz). It is difficult to find support for the concept that there is a single frequency that works best for every patient, but this range appears to cover the majority of individuals (Walsh 1997). This TENS mode is delivered with high frequency (traditional/normal) TENS.

An alternative approach is to stimulate the Aδ fibres which respond preferentially to a much lower rate of stimulation (in the order of 2–5 Hz), which will activate the opioid mechanisms and provide pain relief by causing the release of an endogenous opiate (encephalin) in the spinal cord which will reduce the activation of the noxious sensory pathways (Han et al. 1991; Walsh 1997; Sluka et al. 2006). This TENS mode is delivered with low-frequency (acupuncture (AcuTENS)) TENS.

A third possibility is to stimulate both nerve types at the same time by employing a burst mode stimulation. In this instance, the higher frequency stimulation output (typically at about 100 Hz) is interrupted (or burst) at the rate of about 2–3 bursts per second. When the machine is 'on', it will deliver pulses at the 100 Hz rate, thereby activating the Aβ fibres and the pain gate mechanism, but by virtue of the rate of the burst, each burst will produce excitation in the Aδ fibres, therefore stimulating the opioid mechanisms. For some patients this is by far the most effective approach to pain relief, though as a sensation, numerous patients find it less acceptable than the other forms of TENS.

Traditional TENS (hi-TENS, normal TENS)

Traditional TENS usually uses stimulation at a relatively high frequency (80 or 90–130 Hz) and employs a relatively narrow pulse width (often used at about 100 μs though, as identified above, there is less support for manipulation of the pulse width in the current research literature and the use of a fixed pulse duration of around 200 μs may be the most efficient). The stimulation is delivered at 'normal' intensity (see below). Thirty minutes is probably the minimally effective time, but it can be delivered for as long as needed. The main pain relief is achieved during the stimulation, with a limited 'carry over' effect, i.e. pain relief after the machine has been switched off (Chesterton et al. 2002).

Acupuncture TENS (lo-TENS, AcuTENS)

When using AcuTENS, TENS is used at a lower stimulation frequency (2–5 Hz) with longer duration pulses (200–250 μs). The intensity employed will usually need to be greater than with the traditional TENS – a definite, strong sensation but still one that is not painful (see below). A minimally useful stimulation of 30 minutes should be delivered. It takes some time for the opioid levels to build up with this type of TENS and hence the onset of pain relief may be slower than with the traditional mode. Once sufficient opioid has been released, however, it will keep on working after cessation of the stimulation. Many patients find that stimulation at this low frequency at intervals throughout the day is an effective strategy. The 'carry over' effect may last for several hours in the clinical setting, though timeframes of rather more limited duration have been demonstrated by Chesterton et al. (2002).

Brief intense TENS

Brief intense TENS is a mode that can be employed to achieve rapid pain relief, but some patients may find the strength of the stimulation too intense and will not tolerate it for sufficient duration to make the treatment worthwhile. The pulse frequency applied is high (in the 90–130 Hz band) and the pulse width is also high (≥200 μs). The current is delivered at, or close to, the tolerance level for the patient such that they would not want the machine turned up any higher. In this way, the energy delivery to the patients is relatively high when compared with the other approaches. It is suggested that 15–30 minutes at this stimulation level is the most that would normally be used. Pain relief onset is rapid and marked if the patient can cope with the stimulation intensity (Walsh 1997; Sluka and Walsh 2003).

Burst mode TENS

As described above, the machine is set to deliver traditional TENS, but the burst mode is switched in, therefore

interrupting the stimulation outflow at rate of 2–3 bursts/second. The stimulation intensity will need to be relatively high – not as high as the brief intense TENS but similar to that applied in AcuTENS.

Frequency selection

With all of the above mode guides, it is probably inappropriate to identify very specific frequencies that need to be applied to achieve a particular effect. If there was a single frequency that worked for everybody, it would be much easier, but the research does not support this concept. The patient (or the therapist) needs to identify the most effective frequency for their pain and manipulation of the stimulation frequency dial or button is the best way to achieve this. Patients who are told to leave the dials alone are less likely to achieve optimal effects. Frequency ranges within which the 'ideal' is likely to be found are those identified above. Some TENS devices do not enable control of specific pulse rate and duration settings, but instead offer 'automatic' programmes that effectively deliver some of each of the effective modes. This can be considered advantageous (that the patient has less to worry about on the machine) but others consider it a broad brush approach and the amount of time spent at the optimal setting for the patient is minimal; hence, it may, in fact, be ineffective. There is currently no published evidence that supports the 'bit of everything' approach in the clinical environment.

Stimulation intensity

It is not possible to describe the treatment current strength in terms of how many (milli)amps should be applied. The most effective intensity management appears to be related to what the patient feels during the stimulation and this may vary from session to session, though will tend to be fairly consistent for any individual patient. As a general guide, it appears to be effective to go for a 'definitely there but not painful' level for the normal (high) TENS and a 'strong but not painful' level for the acupuncture (low) mode (Sluka and Walsh 2003). Figure 19.5 illustrates these settings on a 'subjective' scale. Some evidence (e.g. Bjordal et al. 2003; Aarskog et al. 2007) suggests that stronger stimulation might be more effective for clinical pain states.

Electrode placement

In order to get the maximal benefit from the modality, target the stimulus at the appropriate spinal cord level (appropriate to the pain). Placing the electrodes either side of the lesion – or painful areas – is the most common mechanism employed to achieve this. There are many alternatives that have been researched and found to be effective – most of which are based on the appropriate nerve root level/spinal cord segment:

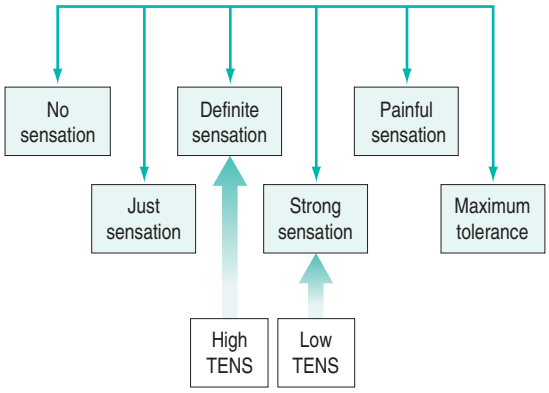


Figure 19.5 Stimulus strength for TENS clinical application.

- stimulation of appropriate nerve root(s);
- stimulate the peripheral nerve (proximal to the pain);
- stimulate motor point (innervated by an appropriate nerve root level);
- stimulate trigger point(s) or acupuncture point(s);
- stimulate the appropriate dermatome, myotome or sclerotome.

It is beyond the scope of this chapter to detail specific electrode combinations for specific clinical problems and, in any case, would probably be inappropriate to do so. The TENS literature covers electrode placement in some detail and the interested reader is referred to useful specific texts in this context (Walsh 1997; Johnson 2008).

If the pain source is vague, diffuse or particularly extensive both channels can be employed simultaneously. A two-channel application can also be effective for the management of a local plus a referred pain combination, with one channel used for each component. Most standard machines do not allow different stimulation parameters to be set for each channel, though there are some devices that will allow this possibility, for example channel A on low frequency, opioid setting and channel B on higher frequency pain gate setting.

Numerous systematic and Cochrane Reviews have been published relating to the application of TENS for several different clinical pain groups (e.g. Rutjes et al. 2009; Walsh et al. 2009). Many of these come to an 'inconclusive' conclusion, though this may be related to dose-related issues (therapeutic windows) (Johnson and Martinson 2007; Watson 2010) and methodological limitations of the research rather than the failure of TENS to have a significant effect.

Interferential therapy (IFT)

The basic principle of IFT is to utilise the strong physiological effects of low frequency (≅<250 pps) electrical

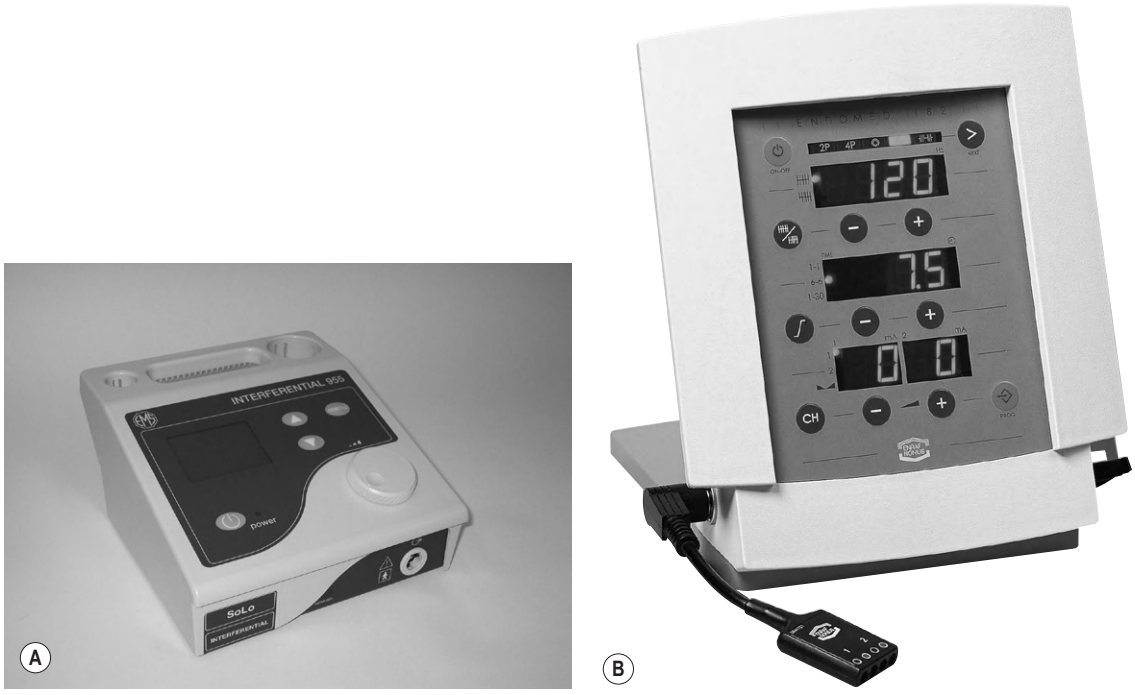


Figure 19.6 Mains-powered interferential devices. (a) IFT device (EMS Physio); (b) IFT available on a general electrical stimulation device (Enraf).

stimulation of muscle and nerve tissues without the associated pain encountered with low frequency stimulation (Watson 2000; Palmer and Martin 2002).

IFT is delivered using either dedicated main s-powered interferential devices (Figure 19.6), portable (battery-powered) devices (Figure 19.7) or multimode units that include IFT stimulation among several other treatment modes (Figure 19.8).

To produce low frequency effects at sufficient intensity at depth, patients may experience considerable discomfort in the superficial tissues (i.e. the skin). This is a result of the compound impedance of the skin being inversely proportional to the frequency of the stimulation. The result of applying this higher frequency is that it will pass more easily through the skin, requiring less electrical energy input to reach the deeper tissues and giving rise to less discomfort.

The effects of tissue stimulation with these 'medium frequency' currents (medium frequency in electromedical terms is usually considered to be 1 kHz–100 kHz) is not fully understood and while it is likely to have an effect, little detail is currently known though it is assumed not to directly stimulate nerve. Ward (2009) recently reviewed the key issues with medium frequency currents.

Interferential therapy utilises two of these medium frequency currents, passed through the tissues simultaneously, where they are set up so that their paths cross and

Figure 19.7 Portable (battery-powered) IFT device (TensCare).

Figure 19.8 Examples of multimode units that include IFT: (a) Gymna; (b) Chatanooga; (c) Uniphy.

they literally interfere with each other. This interference gives rise to an interference (beat frequency) which has the characteristics of low frequency stimulation. In effect the interference mimics a low frequency stimulation in the cross over (interference) zone.

The exact frequency of the resultant interference (or beat frequency) can be controlled by the input frequencies. If, for example, one current was at 4000 Hz and its companion current at 3900 Hz, the resultant beat frequency would be at 100 Hz carried on a medium frequency 3950 Hz amplitude modulated current (Figure 19.9).

By careful manipulation of the input currents it is possible to achieve any beat frequency that is needed clinically. Modern machines usually offer beat frequencies of 1–150 Hz, though some offer a choice of up to 250 Hz or more. Some machines offer a range of 'carrier' frequencies, i.e. other than 4000 Hz. The evidence would suggest that the higher the carrier frequency, the less discomfort will be experienced by the patient and therefore where there is a choice, the higher carrier frequency should be employed.

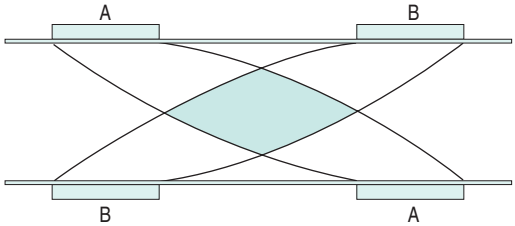


Figure 19.9 Basic principles of interferential current production.

The use of two-pole IFT stimulation is made possible by electronic manipulation of the currents – the interference occurs within the machine instead of in the tissues. There is no known physiological difference between the effects of IFT produced with two- or four-electrode systems; in fact, the pre-modulated currents can be considered superior in clinical effectiveness terms (e.g. Ozcan et al. 2004). The key difference is that with a four-pole application the

interference is generated in the tissues and with a two-pole treatment, the current is 'pre-modulated', i.e. the interference is generated within the machine unit.

Whichever generation system is employed, the treatment effect is generated from low frequency stimulation, primarily involving the peripheral nerves. Low frequency nerve stimulation is physiologically effective (as with TENS and NMES) and this is the key to IFT intervention.

Frequency sweep

Nerves will accommodate to a constant signal and a sweep (or gradually changing frequency) is often used to overcome this problem. The principle of using the sweep is that the machine is set to automatically vary the effective stimulation frequency using either pre-set or user-set sweep ranges. The sweep range employed should be appropriate to the desired physiological effects (see below). It has been repeatedly demonstrated that 'wide' sweep ranges are ineffective in the clinical environment. The clinical advantage of the sweep treatment application, beyond that of minimising the accommodation effects, are that a range of treatment frequencies can be automatically applied (Watson 2000).

Clinical note

Care needs to be taken when setting the sweep on a machine in that with some devices the user sets the actual base and top frequencies (e.g. 90 and 130 Hz) and with other machines the user sets the base frequency and then how much needs to be added for the sweep (e.g. 90 and 40 Hz).

The pattern of the sweep makes a significant difference to the stimulation received by the patient. Most machines offer several sweep patterns, though there is very limited 'evidence' to justify some of these options. In the classic 'triangular' sweep pattern, the machine gradually changes from the base to the top frequency, usually over a time period of six seconds, though some machines also offer one- or three-second options. In the example illustrated (Figure 19.10), the machine is set to sweep from 90 to 130 Hz, employing a triangular sweep pattern. All frequencies between the base and top frequencies are delivered in equal proportion.

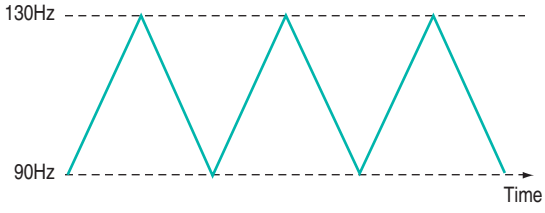


Figure 19.10 90–130 Hz triangular frequency sweep pattern with IFT.

Other patterns of sweep can be produced on many machines, for example a rectangular (or step-like) sweep. This produces a very different stimulation pattern in that the base and top frequencies are set, but the machine then 'switches' between these two specific frequencies rather than gradually changing from one to the other. Figure 19.11 illustrates the effect of setting a 90–130 Hz rectangular sweep.

There is a clear difference between these examples, even though the same 'numbers' are set: one will deliver a full range of stimulation frequencies between the set frequency levels and the other will switch from one frequency to the other. There are numerous other variations on this theme and the 'trapeziodal' sweep (Figure 19.12) is effectively a combination of these two.

The only sweep pattern for which 'evidence' appears to exist is the triangular sweep. The others are perfectly safe to use, but whether they are clinically effective or not remains to be shown.

Physiological effects and clinical applications

It has been suggested that IFT works in a 'special way' because it is 'interferential' as opposed to 'normal' stimulation. The evidence for this special effect is lacking and it is most likely that IFT is just another means by which peripheral nerves can be stimulated. Many regard it as more acceptable than other forms of electrical stimulation as it generates less (skin) discomfort (e.g Shanahan et al. 2006).

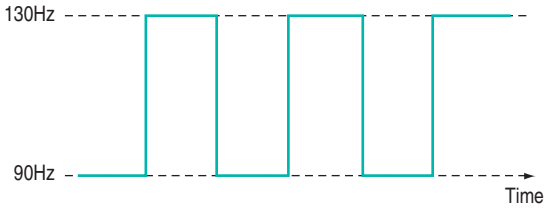


Figure 19.11 90–130 Hz rectangular frequency sweep pattern with IFT.

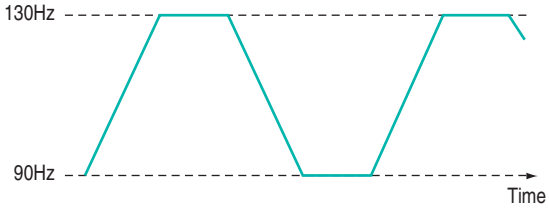


Figure 19.12 90–130 Hz trapezoidal frequency sweep pattern with IFT.

The clinical application of IFT therapy is based on peripheral nerve stimulation (frequency) data, though it is important to note that much of this information has been generated from research with other modalities, and its transfer to IFT is assumed, rather than proven. There is a lack of IFT-specific research compared with other modalities (e.g. TENS, NMES).

The are four main clinical applications for which IFT appears to be used:

- pain relief;
- muscle stimulation;
- increased local blood flow;
- reduction of oedema.

In addition, claims are made for its role in stimulating healing and repair, though they are not specifically covered in this section. As IFT acts primarily on nerve, the strongest effects are likely to be those which are a direct result of such stimulation (i.e. pain relief and muscle stimulation). The other effects are more likely to be secondary consequences of these.

Pain relief

Electrical stimulation for pain relief has widespread clinical use, though the direct research evidence for the use of IFT in this role is limited. Logically one could use the higher frequencies (90–130 Hz) to stimulate the pain gate mechanisms and thereby mask the pain symptoms. Alternatively, stimulation with lower frequencies (2–5 Hz) can be used to activate the opioid mechanisms, again providing a degree of relief. These two different modes of action can be explained physiologically and will have different latent periods and varying duration of effect (same as for TENS). It remains possible that pain relief may be achieved by stimulation of the reticular formation at frequencies of 10–25 Hz or by blocking C fibre transmission at >50 Hz. Although both of these latter mechanisms have been proposed with IFT, neither have been categorically demonstrated (Palmer and Martin 2008).

A good number of studies (e.g. Johnson and Tabasam 2003; Hurley et al. 2004; McManus et al. 2006; Jorge et al. 2006; Walker et al. 2006; Lau et al. 2008; Fuentes et al. 2010) provide substantive evidence for a pain relief effect of IFT.

Muscle stimulation

Stimulation of the motor nerves can be achieved with a wide range of frequencies. Clearly, stimulation at low frequency (e.g. 1 Hz) will result in a series of twitches, while stimulation at 50 Hz will result in a tetanic contraction. There is limited evidence at present for the 'strengthening' effect of IFT (evidence does exist for some other forms of electrical stimulation), though the article by Bircan et al. (2002) suggests that it might be a possibility. On the basis of the current evidence, the contraction brought about by IFT is no 'better' than would be achieved by active exercise, though there are clinical circumstances where assisted contraction is beneficial. For example, to assist the patient to appreciate the muscle work required (similar to surged Faradism used previously, but much less uncomfortable). For patients who cannot generate useful voluntary contraction, IFT may be beneficial as it would be for those who, for whatever reason, find active exercise difficult. There is no evidence that has demonstrated a significant benefit of IFT over active exercise. The choice of treatment parameters will depend on the desired effect. The most effective motor nerve stimulation range with IFT appears to lie between approximately 10 and 20 Hz (maybe between 10 and 25 Hz).

Caution should be exercised when employing IFT as a means to generate clinical levels of muscle contraction in that the muscle will continue to work for the duration of the stimulation period (assuming sufficient current strength is applied). It is possible to continue to stimulate the muscle beyond its point of fatigue and short stimulation periods with adequate rest is probably a preferable option. Some IFT devices are capable of generating a 'surged' stimulation mode which might be advantageous in that fatigue would be minimised – this surged intervention would be similar to, but more comfortable than, Faradism.

Blood flow

There is very little, if any, quality evidence demonstrating a direct effect of IFT on local blood flow changes. Most of the work that has been done involves laboratory experimentation on animals or asymptomatic subjects, and most blood flow measurements are superficial, i.e. skin blood flow. Whether IFT is actually capable of generating a change (increase) in blood flow at depth remains questionable. The elegant study by Noble et al. (2000) demonstrated vascular changes at 10–20 Hz, though they were unable to identify clearly the mechanism for this change. The stimulation was applied via suction electrodes and the outcome could, therefore, be a result of the suction rather than the electrical stimulation, though this is largely negated by virtue of the fact that other stimulation frequencies were also delivered with the suction electrodes without significant flow changes. The most likely mechanism therefore is via muscle stimulation effects (IFT causing muscle contraction which brings about a local metabolic and thus vascular change). The possibility that the IFT is acting as an inhibitor of sympathetic activity remains a theoretical possibility rather than an established mechanism.

Based on current available evidence, the most likely option for IFT use as a means to increase local blood flow remains via the muscle stimulation mode; thus, the 10–20 or 10–25 Hz frequency sweep appears to be preferable.

Figure 19.13 IFT rubber and sponge pad electrode system (Gymna).

Figure 19.14 IFT Vacuum electrode system (Gymna).

Oedema

IFT has been claimed to be effective as a treatment to promote the reabsorption of oedema in the tissues. Again, the published, as opposed to the anecdotal, evidence is very limited in this respect and the physiological mechanism by which it could be achieved as a direct effect of IFT remains to be established. The preferable clinical option in the light of the available evidence is to use the IFT to bring about local muscle contraction(s) which, combined with the local vascular changes, could be effective in encouraging the reabsorption of tissue fluid. The use of suction electrodes may be beneficial, but also remains unproven in this respect.

A study by Jarit et al. (2003) demonstrated a change in oedema following knee surgery in an IFT group; however, a study by Christie and Willoughby (1990) failed to demonstrate a significant benefit on ankle oedema following fracture and surgery. The treatment parameters employed are unlikely to be effective given the information now available. If IFT has a capacity to influence oedema, the current evidence and physiological knowledge would suggest that a combination of pain relief (allowing more movement), muscle stimulation (above) and enhanced local blood flow (above) is the most likely combination to be effective and thus 10–20 Hz stimulation around the largest local muscle group is probably the most effective approach.

Treatment parameters

Stimulation can be applied using several different electrode systems (Figures 19.13 and 19.14). Pad electrodes and sponge covers are commonly employed; electroconductive gel is an effective alternative. The sponges should be thoroughly wet to ensure even current distribution. Self-adhesive pad electrodes are also available (similar to the newer TENS electrodes) and in the view of many practitioners make IFT application easier. The suction electrode application method has been in use for several years and while it is useful, especially for larger body areas like the shoulder girdle, trunk, hip and knee, it does not appear to provide any therapeutic advantage over pad electrodes. Care should be taken with regard to the maintenance of electrodes, electrode covers and associated infection risks (Lambert et al. 2000).

Electrode positioning should ensure adequate coverage of the area for stimulation. In some circumstances, a bipolar method is preferable if a longitudinal zone requires stimulation rather than an isolated tissue area. Placement of the electrodes should be such that a cross-over effect is achieved in the desired area (when using the four-pole application).

Treatment times vary widely according to the usual clinical parameters of acute/chronic conditions and the type of physiological effect desired. In acute conditions, shorter treatment times of ten minutes may be sufficient to achieve the effect. In other circumstances, it may be necessary to stimulate the tissues for 20–30 minutes. It is suggested that short treatment times are adopted initially, especially with acute cases in the event of symptom exacerbation. These can be progressed if the aim has not been achieved and no untoward side effects have been produced. There is no research evidence to support the continuous progression of a treatment dose in order to increase or maintain its effect.

Muscle stimulation modalities

Historically, motor nerve stimulation in order to generate muscle contraction was a widely employed modality, most commonly in the form of Faradism. More recently, this intervention has fallen somewhat 'out of favour'. There are, in fact, still circumstances where it could be of significant benefit, but in clinical practice using IFT (preferably in a surged mode) is probably a more effective means to the same end.

There has been a significant increase in the use of portable or mains-powered muscle stimulators which are effectively the modern replacement for Faradism. Figure 19.15

Figure 19.15 (a–c) Battery-powered (portable) muscle stimulators, all of which will deliver muscle-stimulating currents: (a) NMES stimulator (TensCare); (b) NMES stimulator (Body Clock); (c) IntelliSTIM (Natures Gate); (d) multimodal mains-powered device – part of MediLink (EMS Physio); (e) stimulator with dedicated knee sleeve electrode system (BMR Neurotech).

illustrates examples of dedicated battery-powered and multimodal mains-powered devices.

There is a growing range of chronic (meaning 'not short-term') electrical stimulation devices that are aimed at stimulating the motor nerve and hence bringing about muscle activity/contraction. This form of therapy goes by several names, but the most commonly applied are neuromuscular electrical stimulation (NMES), chronic NMES and neuromuscular stimulation (NMS). NMES is probably the preferred generic term.

It is suggested, with a growing body of evidence, that gains in strength, endurance capacity and function can be achieved with these types of stimulation. Much of the early work has been conducted in laboratory studies and also with athletes rather than typical patient groups. There are recent articles, however, that have demonstrated significant clinical benefit with patient groups, including strengthening of peripheral musculature (Talbot et al. 2003; Callaghan and Oldham 2004; Stevens et al. 2004; Lyons et al. 2005), work with shoulder problems in stroke patients (Chantraine et al. 1999; Ada and Foongchomcheay 2002), chronic obstructive pulmonary disease (COPD)/cardiac patients (Neder et al. 2002; Zanotti et al. 2003; Vivodtzev et al. 2006) and various forms of incontinence (Indrekvam et al. 2001; Amaro et al. 2003; Barroso et al. 2004). Useful reviews of this field of intervention are included in McDonaugh (2008), Robertson et al. (2006) and Lake (1992).

A further whole branch of neuromuscular-based stimulation is the so called functional electrical stimulation (FES). In this branch of electrotherapy, the explicit intent of the stimulation is to achieve controlled muscle activation in order to facilitate some aspect of functional activity. The range of applications is growing swiftly, especially with the recent advances in computer-controlled stimulators. One of the early, and most successful areas, is the dropped foot stimulator (Taylor et al. 1999; Sheffler et al. 2006) which assists patients – most often following a stroke – to achieve ankle dorsiflexion during the swing phase of gait utilising stimulation of the anterior tibial nerve. Modern devices incorporate a range of foot switches or pressure detectors which enable stimulation to be active at exactly the right part of the gait cycle (Figure 19.16). Other forms of FES include standing and gross walking activity with paraplegic patients which is also making significant gains with computerised control systems.

Microcurrent therapy

Microcurrent therapy is becoming increasingly employed in the clinical environment as a new and emerging modality (though, in fact, it is neither new nor emerging – it has strong published evidence dating back several decades).

It is interesting in that it appears to break all the classification 'rules' that were identified earlier in the chapter. It is not delivered with the intention of stimulating nerves, but rather plays to the bioelectric environment of the tissues, and therefore has a primary effect in terms of tissue repair (Watson 2006a; Poltawski and Watson 2009). The general characteristics of this type of therapy are that they utilise a direct current (pulsed or continuous) delivered at a very low amplitude (literally in the microamperage (millionths of an Amp) range) which is usually subsensory from the patient perspective. This type of therapy has already been shown to be effective in several clinical areas – most notably fracture repair (Simonis et al. 2003; Ciombor and Aaron 2005) and healing of open wounds (Watson 1996; Evans et al. 2001; Watson 2008a) though soft tissue repair research is now evolving and showing strong future potential. The use of this therapy in tissue injury treatment has been reviewed recently (Poltawski and Watson 2009).

Figure 19.16 Odstock dropped foot stimulator. Salisbury ODFS111 stimulator, innersoles, footswitch, electrode pads and leads. The stimulator is a single channel device designed primarily for gait assistance. *(Courtesy of the Department of Clinical Science and Engineering, Salisbury District Hospital, UK.)*

Microcurrent therapy devices appear to be most effectively employed when used for hours a day (rather than minutes a week in the clinic) and therefore home-based therapy with small, inexpensive, portable devices (Figure 19.17) is a likely way forward. Microcurrent therapy can, of course, be delivered using mains-powered and multimodal devices (e.g. Chatanooga Intelect and Gymna 400) and it has yet to be clearly determined whether the small portable battery-powered home-based machines or the clinic-based ones will become the most widely employed.

Other forms of electrical stimulation

There are probably more 'new' electrical stimulation-type devices that come out in a year than in any other area of electrotherapy. Many of them are actually based on one of the three main areas identified in previous sections (TENS, IFT or NMES), though there are, of course, machines that do, indeed, deliver a 'different' form of stimulation and others that are simply a variation on a theme or a combination of themes.

Figure 19.17 Microcurrent therapy battery-powered, portable devices. (a) Alpha Stim microcurrent stimulator; (b) Tendon Works microcurrent stimulator (Synapse); (c) Elexoma microcurrent device.

In addition, combination therapy involves the simultaneous delivery of ultrasound and IFT, thereby achieving the effects of both modalities, though there is no evidence that any additional effects will occur by using this combination approach. Ultrasound is sometimes combined with other electrical stimulation modes (e.g. diadynamic currents, TENS).

It is not possible in a general chapter such as this to identify all the possible combinations and variations. The interested reader might try investigating the major texts in the area (Robertson et al. 2006; Watson 2008b; Belanger 2010). Try the current literature (though many of these 'new' devices are yet to have specific research published with regard to their efficacy) or utilise web-based resources (though as with any web-based material, one needs to be both critical and selective with regards the information accepted).

THERMAL MODALITIES

Introduction

Thermal-based treatments have been popular in the past and have somewhat lost favour in more recent times, being considered old fashioned and ineffective. There is little doubt that the application of heat to the body tissues generates significant physiological effects (Lehmann 1982; Michlovitz 1996; Watson 2008b; Belanger 2010), and applied with good reason and utilising the appropriate forms of thermal energy (contact or induction methods) is capable of being useful in therapy.

Principles of thermal treatments

The general principles of heat transfer to the body are well covered in the standard texts, and only a brief summary will be included here. Essentially, thermal energy can be applied using contact methods (hydrocollator or hot packs, wax therapy), by using radiant heat sources (infrared) or by applying electromagnetic energy which is absorbed in the tissues resulting in heat generation (e.g. shortwave, other RF and microwave therapies).

Some modalities (e.g. laser and ultrasound) can generate heating effects if applied at sufficient energy levels, but these modalities appear to be more effective in the clinical environment when applied at 'non-thermal' levels and so are considered in the next section.

Therapeutic effects of heating therapies

The effects of tissue heating can be effectively described in general terms rather than repeating the same information in subsequent sessions. These effects are described and

evidenced in much more detail in specialist texts (Lehmann 1982; Michlovitz 1996; Watson 2008b), but are summarised below for quick reference. Those effects listed are considered to be local rather than systemic effects. Systemic effects are usually minor in relation to the therapeutic application of heat and are detailed in the same sources as identified above.

- Increased metabolic activity of cells/tissues.
- Increased local blood flow (volume).
- Increased collagen extensibility.
- Reduction in local muscle tone.
- Reduced pain perception.
- (Possible) increased rate of tissue healing (almost certainly a secondary effect related to increased metabolic rate and local blood flow changes).

Infrared radiation

Infrared is one of the least used heat modalities in modern practice. The radiation is delivered using luminous (combined infrared and visible red radiation) or non-luminous (infrared only) sources, but whichever one is employed, the effective penetration depth of the electromagnetic energy is a matter of 1 or 1.5 mm at best and, hence, is a very superficial form of heating. It is argued that even though the heat is only generated in the superficial tissues, the effect in terms of pain relief is significant, as many sensory nerve endings are found in these tissues and, when stimulated, they can be used to achieve pain relief by means of the pain gate mechanism. Furthermore, the heating of superficial tissues gives rise to local increases in blood flow which can, in turn, provide reduction in pain by increasing the absorption of inflammatory metabolites, decreasing local muscle spasm, encouraging the reabsorption of oedema and possibly increasing tissue repair by means of metabolic stimulation. Whether these effects are of sufficient magnitude to be of clinical value remains controversial, though there is little doubt that the local application of such superficial heat does give rise to (symptomatic) pain relief and the patients often report a benefit as a result (Kitchen and Partridge 1991; Watson 2008b).

Wax therapy

In a similar way to infrared therapy, wax therapy has become far less widely used than in the past, with the exception of some specialist applications, most notably in hand therapy and peripheral nerve lesion rehabilitation (e.g. Ayling and Marks 2000; Brosseau et al. 2002).

In principle, the melted wax is applied to the part to be treated either using a 'dunk' technique in which the hand or foot, for example, is repeatedly dunked into a bath of moulten wax at around 45–50°C. Each layer of wax being allowed to cool slightly before the next is applied. Alternatively, the wax can be applied using a bandage application or ladled on to the part to be treated.

As the wax solidifies from its melted state, thermal energy is released (latent heat) which is subsequently transferred to the superficial tissues. Tissue temperatures can be raised by a useful 3–4°C to a depth of possibly up to 2 cm, therefore providing a 'deeper' treatment effect than infrared therapy, for example.

In addition to the direct heating effect, it is argued that the deeper tissues will also heat up as a result of the conduction of thermal energy from the superficial tissues. The applied wax also contains a variety of oils which have beneficial effects in terms of improving skin condition and facilitating exercise and massage therapies that usually follow the wax treatment.

It is suggested that a similar heating level can be achieved in the tissues using a hot pack (following section) or hot water immersion, but many patients find that the application of therapeutic wax has perceived benefits over and above the heat part of the treatment, and will certainly prefer it as a form of therapy to other forms of heating – this appears to be especially true for patients with long-term or chronic conditions, low grade, chronic inflammatory conditions and non-acute degenerative joint problems.

Further details are available in most standard electrotherapy or thermal therapy texts (Lehmann 1982; Michlovitz 1996; Robertson et al. 2006; Belanger 2010).

Hot packs

Hot packs used in therapy go by several names, but are most commonly known as hydrocollator packs or simply hot packs. This type of heat application is sometimes referred to as 'moist' heat in that the pack is heated by immersion in hot (70°C) water to bring the pack up to therapeutic temperature. The pack is wrapped, typically in towelling prior to placing against the skin of the patient. The skin surface temperature of the wrapped pack will be significantly lower than the 70°C owing to the insulating nature of the towels, which are essential as these packs should never be applied directly to the skin surface.

The tissue temperature rises associated with hot pack therapy are of sufficient magnitude to achieve changes in the 'therapeutic range' (Lehmann 1982), and will produce sufficient heating to be of clinical value down to a depth of at least 2–3 cm.

Treatment durations of 15–20 minutes appear to be clinically effective and the rise in tissue temperature will achieve effects in common with other thermal modalities.

Shortwave and microwave diathermy

Both continuous shortwave diathermy (SWD) and certainly microwave diathermy (MWD) have shown a

significant reduction in clinical popularity in recent years (Pope et al. 1995). Recent surveys have shown (e.g. Shah et al. 2007; Chipchase et al. 2009) that almost no departments in Australia or the UK routinely use microwave diathermy, and most departments and practices do not even have the machines available. Continuous shortwave, once a highly utilised modality, has also undergone a reduction in clinical use, though many departments do still have the equipment available (Al Mandeel and Watson 2006).

Both modalities bring about tissue heating by means of the introduction of electromagnetic energy into the tissues, and tissue temperature rises occur as a result of increased molecular activity – it is an indirect form of heating (as opposed to the conductive type heating described previously).

SWD utilises a high frequency (around 27 MHz) of electromagnetic energy which is applied either using capacitor plate-type electrodes or a monode/drum applicator. Although attached to the same treatment unit, each of these electrode types brings about heating in a slightly different way and, importantly, there is a fundamental difference in the type of tissues in which the heat is produced.

The interested reader is referred to the detail in electrotherapy texts but, in summary, the capacitor plate-type application results in high frequency energy being delivered to the tissues (electrostatic). Dissipation of this energy is primarily in the tissues of high impedance, thus a very high proportion of the heating effect is generated in the skin, fat and other 'insulating' superficial tissues. The previous claims that this was a deep form of heating is questionable and in terms of current clinical application its demise is probably appropriate.

The alternative mode of application employs the monode or drum applicator (as per PSWT). The energy delivered from this type of applicator is effectively an electromagnetic (as opposed to electrostatic) field, oscillating at high frequency. The delivered energy is primarily absorbed in the tissues of low impedance (such as muscle, nerve, tissue where there is significant water content) and it is here that the most significant heating effects are achieved.

It is suggested that when applying continuous shortwave in the clinical environment, with the deliberate intention of heating the tissues, the monode or drum applicator should routinely be used unless the primary tissue in which the heating is desired is the skin or superficial fat layers.

Other radio frequency (RF) therapies

There has been a recent upsurge in the use of various RF energy applications at frequencies other than those classically employed for shortwave and microwave treatments. Delivery of these energies either via a capacitive coupling or resistive coupling system appears to generate significant tissue heating and these effects appear to be achieved at some reasonable depth. The anecdotal and early experimental evidence is generally supportive but, as yet, there are no substantive comparative trials (i.e. comparing the efficacy of these new applications with existing thermal treatments) to enable a clinical effectiveness judgement to be made. Work is in progress and it may transpire that RF applications at frequencies other than those historically employed in therapy are more effective, and a shift in clinical delivery systems may therefore be justified. The machines that deliver the RF energy at relative high doses can also be employed in 'low dose' mode, achieving clinical effects more in line with the 'non-thermal' modalities described in the next section. Establishing whether the high or low energy modes are more effective than other therapies in these sections is ongoing. An examples of RF treatment equipment is illustrated in Figure 19.18.

Figure 19.18 Example of a radio frequency treatment device (Indiba).

NON-THERMAL MODALITIES

Introduction

There has been a significant debate in recent years with regard to the potential for various electrotherapy modalities to have their primary mode of action by means of 'non-thermal' effects. There is a growing body of evidence that supports the contention that it is not necessary to provide sufficient energy to actually 'heat' the tissues in order to generate therapeutic benefit. Modalities such as ultrasound, shortwave, RF, laser and microwave therapies, when applied at sufficiently low intensity, appear to bring about significant clinical effects without generating perceptible thermal change. This is a difficult area as when any energy is delivered to the body and absorbed in sufficient quantity, there will be some thermal effect. What is generally meant by the clinical term 'non-thermal' is that neither the patient nor the therapist are able to detect any significant (gross) temperature change.

Various authors have argued that the therapeutic benefits of generating tissue 'stimulation' without overt heating is advantageous in many circumstances (e.g. Watson 2000; Watson 2006a; Kitchen and Dyson 2008). This is not to denigrate the therapeutic benefits of heating as a therapeutic tool, but rather to add a non-thermal aspect. Clearly, all the modalities identified in this section *could* generate significant heating if applied at sufficient levels. Their application as described here is in the clinically non-thermal mode.

In this section, ultrasound (traditional and LIPUS), PSWT and laser therapies will constitute the main content. Microcurrent therapy, as identified in the introductory section, could fit in this section or in the electrical stimulation section. For the purposes of this chapter, it has been included in the electrical stimulation group. The emerging RF applications (see the 'Thermal modalities' section) could also be included in this section, but until there is sufficient new evidence their effects will be confined to the thermal group.

Therapeutic ultrasound

Ultrasound therapy is one of the most commonly employed electrophysical treatments in many countries. Both mains-powered and battery-powered treatment units are available and, additionally, it is reasonably common to find ultrasound as one modality offered on multimodal electrotherapy machines (Figure 19.19).

Ultrasound is a form of mechanical (vibration) energy, and vibration at increasing frequencies is known as sound energy. The normal human sound range is from 16 Hz to something approaching 15–20,000 Hz. Beyond this upper limit, the mechanical vibration is known as ultrasound. The frequencies used in therapy are typically between 1.0 and 3.0 MHz (1 MHz = 1 million cycles per second) and is clearly beyond the human sound detection range. Some therapy devices offer application frequencies at other rates, for example 1.5 MHz, 0.75 MHz and longwave ultrasound devices that operate in the kilohertz range (e.g. 48 kHz and 150 kHz). The effect of longwave ultrasound is beyond the scope of this section, which will concentrate on 'traditional' (MHz) ultrasound.

Sound waves are longitudinal waves consisting of zones of compression and rarefaction. Particles of a material, when exposed to a sound wave will oscillate about a fixed point. Clearly, any increase in the molecular vibration in the tissue can result in heat generation and ultrasound can be used to produce thermal changes in the tissues, though current usage in therapy does not focus on this phenomenon (Nussbaum 1997; ter Haar 1999; Baker et al. 2001; Watson and Young 2008). In addition to thermal changes, the vibration of the tissues has effects which are considered to be 'non-thermal' in nature, although, as with other modalities, there must be a thermal component – however small. As the ultrasound

Figure 19.19 Examples of clinical ultrasound therapy machines: (a) mains-powered ultrasound machine (EMS Physio); (b) mains/battery ultrasound machine (Chatanooga); (c) multimodal device which includes ultrasound (Enraf).

wave passes through a material (the tissues), the energy levels within the wave will diminish as energy is transferred to the material.

Ultrasound transmission through the tissues

All materials (tissues) will present impedance to the passage of sound waves. The specific impedance of a tissue will be determined by its density and elasticity. In order for the optimal transmission of energy from one medium to another, the impedance of the two media needs to be as similar as possible. The greater the difference in impedance at a boundary, the greater the reflection that will occur and, therefore, the smaller the amount of energy that will be transferred.

The difference in impedance is greatest for the steel/air interface which is the first one that the ultrasound has to overcome in order to reach the tissues. To minimise this difference, a suitable coupling medium has to be utilised. If even a small air gap exists between the transducer and the skin, the proportion of ultrasound that will be reflected approaches 99.998% which, in effect, means that there will be no transmission.

The coupling media used in this context include water, various oils, creams and gels. Ideally, the coupling medium should be fluid so as to fill all available spaces, relatively viscous so that it stays in place, have an impedance appropriate to the media it connects and should allow transmission of ultrasound with minimal absorption, attenuation or disturbance. At the present time, gel-based media appear to be preferable to oils and creams. Water is a good media and can be used as an alternative but clearly it fails to meet the above criteria in terms of its viscosity. There is no realistic (clinical) difference between the gels in common clinical use (Poltawski and Watson 2007).

Absorption and attenuation

The absorption of ultrasound energy follows an exponential pattern, i.e. more energy is absorbed in the superficial tissues than in the deep tissues. In order for energy to have an effect it must be absorbed. At some point this must be considered in relation to the ultrasound dosages applied to achieve certain effects. Because the absorption (penetration) is exponential, there is (in theory) no point at which all the energy has been absorbed, but there is certainly a point at which the ultrasound energy levels are not sufficient to produce a therapeutic effect. As the ultrasound beam penetrates further into the tissues, a greater proportion of the energy will have been absorbed and therefore there is less energy available to achieve therapeutic effects. The half value depth is often quoted in relation to ultrasound and it represents the depth in the tissues at which half the surface energy remains available.

As it is difficult, if not impossible, to know the thickness of each specific tissue layer in an individual patient, average half-value depths are employed for each frequency. The 'average' half-value depths of commonly employed ultrasound frequencies are: 3 MHz = 2.0 cm and 1 MHz = 4.0 cm. Table 19.1 provides an estimate of the ultrasound energy remaining at various tissue depths relative to the skin surface intensity.

Table 19.1 Ultrasound intensity at various tissue depths based on average half-value data

Depth (cm)	3 MHz	1 MHz
2	50%	
4	25%	50%
6		
8		25%

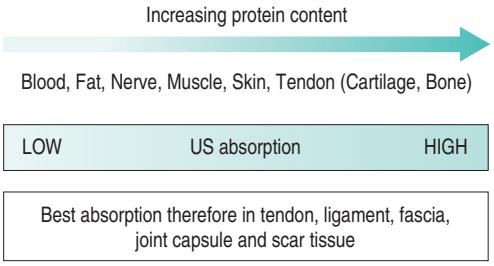


Figure 19.20 Ultrasound absorption in different tissues.

To achieve a particular ultrasound intensity at depth, account must be taken of the proportion of energy which has been absorbed by the tissues in the more superficial layers. The table gives an approximate reduction in energy levels with typical tissues at two commonly used frequencies. More detailed information and absorption tables are available at www.electrotherapy.org and in Watson (2002).

As the penetration (or transmission) of ultrasound is not the same in each tissue type, it is clear that some tissues are capable of greater absorption of ultrasound than others. Generally, the tissues with the higher protein content will absorb ultrasound to a greater extent, thus tissues with high water content and low protein content absorb little of the ultrasound energy (e.g. blood and fat), while those with a lower water content and a higher protein content will absorb ultrasound far more efficiently. It has been suggested that tissues can therefore be ranked according to their tissue absorption (Figure 19.20).

Although cartilage and bone are at the upper end of this scale, the problems associated with wave reflection mean that the majority of ultrasound energy striking the surface of either of these tissues is likely to be reflected. The best absorbing tissues in terms of clinical practice are those with high collagen content: ligament, tendon, fascia, joint capsule and scar tissue (Frizzell and Dunn 1982;

Table 19.2 Pulse ratios and equivalent duty cycle percentage for clinical ultrasound machines

Mode	Pulse ratio	Duty cycle
Continuous	N/A	100%
Pulsed	1 : 1	50%
	1 : 2	33%
	1 : 3	25%
	1 : 4	20%
	1 : 9	10%

Nussbaum 1998; ter Haar 1999; Watson 2000; Watson 2008c; Watson and Young 2008).

The application of therapeutic ultrasound to tissues with a low energy absorption capacity is likely to be less effective than the application of the same energy to a more highly absorbing material. Recent evidence of the ineffectiveness of such an intervention can be found in Wilkin et al. (2004), while application in tissue that is a better absorber will result in a more effective intervention (e.g. Leung et al. 2004; Sparrow et al. 2005).

Pulsed ultrasound

Most machines offer the facility for pulsed ultrasound output. Typical pulse formats are 1 : 1 and 1 : 4, although others are available. In 1 : 1 mode, the machine offers an output for 2 ms followed by 2 ms rest. In 1 : 4 mode, the 2 ms output is followed by an 8 ms rest period. The effects of pulsed ultrasound are well documented (Watson 2008c; Watson and Young 2008) and this type of output is preferable, especially in the treatment of the more acute lesions. The duty cycle (% of time during which the machine gives an output) will be 50% for the 1 : 1 mode and 20% for the 1 : 4 mode. Table 19.2 illustrates the equivalent pulse ratios and duty cycles (as a percentage).

Clinical uses of ultrasound therapy

One of the therapeutic effects for which ultrasound has been used is in relation to tissue repair/healing. It is suggested that the application of ultrasound to injured tissues will, among other things, speed the rate of healing and enhance the quality of the repair, which will be the focus of this section.

The therapeutic effects of ultrasound are generally divided into *thermal* and *non-thermal* groups.

Thermal effects and uses

In thermal mode, ultrasound will be most effective in heating the dense collagenous tissues and will require a relatively high intensity, preferably in continuous mode, to achieve this effect.

Many articles have concentrated on the thermal effectiveness of ultrasound and, much as it can be used effectively in this way when an appropriate dose is selected (continuous mode >0.5 W cm^{-2}), the focus of this chapter will be on the non-thermal effects. Both Nussbaum (1998) and ter Haar (1999) have provided some useful review material with regard to the thermal effects of ultrasound. Comparative studies on the thermal effects of ultrasound have been reported by several authors (e.g. Draper et al. 1993; Draper and Ricard 1995; Draper et al. 1995; Meakins and Watson 2006) with some interesting, and potentially useful, results.

It is too simplistic to assume that a particular treatment application will either have thermal or non-thermal effects. It is almost inevitable that both will occur, but, furthermore, it is reasonable to argue that the dominant effect will be influenced by treatment parameters, especially the mode of application, i.e. pulsed or continuous. Baker et al. (2001) have argued the scientific basis for this issue coherently.

Non-thermal effects and uses

The non-thermal effects of ultrasound are now attributed primarily to a combination of *cavitation* and *acoustic streaming* (Williams et al. 1987; ter Haar 1999; Baker et al. 2001; Watson 2008c). There appears to be little by way of convincing evidence to support the notion of *micromassage*, although it does sound rather appealing. Details of these primary physical effects of ultrasound are detailed in appropriate texts and papers (Robertson et al. 2006; Watson and Young 2008).

The result of the combined effects of stable cavitation and acoustic streaming is that the cell membrane becomes 'excited' (up-regulates), thus increasing the activity levels of the whole cell. The ultrasound energy acts as a trigger for this process, but it is the increased cellular activity which is in effect responsible for the therapeutic benefits of the modality (Figure 19.21) (Dinno 1989; Watson 2000, 2008c; Leung et al. 2004; Watson and Young 2008).

Ultrasound application in relation to tissue repair

The process of tissue repair is a complex series of cascaded, chemically-mediated events that lead to the production of scar tissue that constitutes an effective material to restore the continuity of the damaged tissue. The process is more complex than will be described here and there are numerous review articles, including Watson (2003, 2006a).

Inflammation

During the inflammatory phase, ultrasound has a stimulating effect on the mast cells, platelets, white cells with

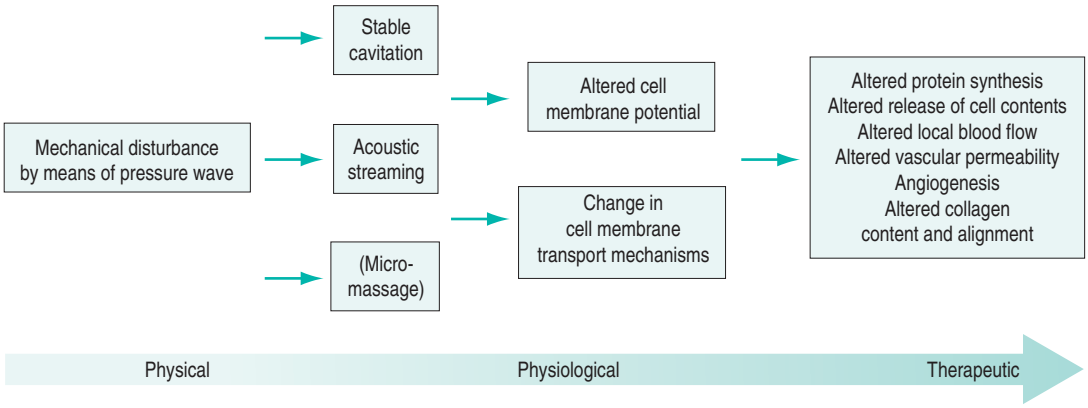


Figure 19.21 Proposed mechanisims of ultrasound action.

phagocytic roles and the macrophages (Maxwell 1992; Nussbaum 1997; ter Haar 1999). For example, the application of ultrasound induces the degranulation of mast cells, causing the release of arachidonic acid (which itself is a precursor for the synthesis of prostaglandins and leukotriene), which act as inflammatory mediators (Mortimer and Dyson 1988; Nussbaum 1997; Leung et al. 2004). By increasing the activity of these cells, the overall influence of therapeutic ultrasound is certainly pro-inflammatory rather than anti-inflammatory. The benefit of this mode of action is not to 'increase' the inflammatory response as such (though if applied with too great an intensity at this stage, it is a possible outcome (Ciccone et al. 1991)), but rather to act as an 'inflammatory optimiser'. The inflammatory response is essential to the effective repair of tissue and the more efficiently the process can complete, the more effectively the tissue can progress to the next phase (proliferation). Ultrasound is effective at promoting the normality of the inflammatory events and, as such, has a therapeutic value in promoting the overall repair events (ter Haar 1999). A further benefit is that the inflammatory, chemically-mediated events are associated with stimulation of the proliferative phase and, hence, the promotion of the inflammatory phase also acts as a promoter of repair.

Employed at an appropriate treatment dose, with optimal treatment parameters (intensity, pulsing and time), the benefit of ultrasound is to make as efficient as possible the earliest repair phase and, thus, have a promotional effect on the whole healing cascade. For tissues in which there is an inflammatory reaction, but in which there is no 'repair' to be achieved, the benefit of ultrasound is to promote the normal resolution of the inflammatory events, and hence resolve the 'problem' This will be most effectively achieved in the tissues that preferentially absorb ultrasound, i.e. the dense collagenous tissues (Watson 2006a, 2008c).

Proliferation

During the proliferative phase, ultrasound also has a stimulative effect (cellular up-regulation), though the primary active targets are now the fibroblasts, endothelial cells and myofibroblasts (Dyson and Smalley 1983; Mortimer and Dyson 1988; Young and Dyson 1990a, 1990b; Maxwell 1992; Nussbaum 1997, 1998; Ramirez et al. 1997). These are all cells that are normally active during scar production, and ultrasound is therefore pro-proliferative in the same way that it is pro-inflammatory – it does not change the normal proliferative phase, but maximises its efficiency – producing the required scar tissue in an optimal fashion. Harvey et al. (1975) demonstrated that low dose, pulsed ultrasound increases protein synthesis, and several research groups have demonstrated enhanced fibroplasia and collagen synthesis (Enwemeka 1989; Turner et al. 1989; Enwemeka et al. 1990; Huys et al. 1993; Ramirez et al. 1997). Recent work has identified the critical role of numerous growth factors in relation to tissue repair and some accumulating evidence has identified that therapeutic ultrasound has a positive role to play in this context (e.g. Reher et al. 2002).

Remodelling

During the remodelling phase of repair, the somewhat generic scar that is produced in the initial stages is refined such that it adopts functional characteristics of the tissue that it is repairing. This is achieved by a number of processes, but is mainly related to the orientation of the collagen fibres in the developing scar and also to the change in collagen type, from predominantly type III collagen to a more dominant type I collagen (Watson 2006b).

The application of therapeutic ultrasound can influence the remodelling of the scar tissue in that it appears to be capable of enhancing the appropriate orientation of the newly formed collagen fibres and also to the collagen

profile change from mainly type III to a more dominant type I construction, thus increasing tensile strength and enhancing scar mobility (Nussbaum 1998; Wang 1998). Ultrasound applied to tissues enhances the functional capacity of the scar tissues (Huys et al. 1993; Nussbaum 1998; Yeung et al. 2006). The role of ultrasound in this phase may also have the capacity to influence collagen fibre orientation as demonstrated in an elegant study by Byl et al. (1996).

The application of ultrasound during the inflammatory, proliferative and repair phases is not of value because it changes the normal sequence of events, but because it has the capacity to stimulate or enhance these normal events and thus increase the efficiency of the repair phases (ter Haar 1999; Watson 2006a, 2008c). It would appear that if a tissue is repairing in a compromised or inhibited fashion, the application of therapeutic ultrasound at an appropriate dose will enhance this activity. If the tissue is healing 'normally', the application will optimise the process and thus enable the tissue to reach its endpoint faster than would otherwise be the case. The effective application of ultrasound to achieve these aims is dose-dependent.

Treatment doses

It appears to be important to make an accurate dose selection when applying therapeutic ultrasound. There are numerous research articles which have demonstrated no therapeutic/clinical value when using the modality, and part of the reason for this would appear to be that there is a dose-dependency in terms of therapeutic outcome (reviewed in Watson 2010) though others have argued against this proposition (Robertson 2002).

The detail of making dose selections is beyond the remit of this chapter. Essentially, it involves the variation of frequency, pulsing ratio (duty cycle), intensity and time. Details of calculation methods have been published (e.g. Watson 2002) and are also available at various web resources and manufacturers' sites (e.g. www.electrotherapy.org).

Low intensity pulsed ultrasound (LIPUS) and fracture healing

There has recently been a strong range of research articles that have identified the potential for low intensity ultrasound to promote tissue repair, most strongly evidenced in relation to fractures, including both 'normal', delayed/non-union and post-surgery clinical scenarios.

While it is beyond the remit of this chapter to present a full analysis of this increasingly popular treatment, the essentials will be covered and some key references presented. It is suggested that while this may not yet constitute a 'normal' physiotherapy treatment, it is anticipated that it is likely to become so within the near future.

Figure 19.22 Example of LIPUS device. Exogen (Smith and Nephew).

Numerous recent articles have identified the benefits of using therapeutic ultrasound for both normally healing (fresh) fractures and delayed union or non-union situations. A systematic review and meta-analysis (Busse et al. 2002) has carefully considered the evidence in respect to the effect of LIPUS on the time to fracture healing. They conclude that the evidence from randomised trials where the data could be pooled (3 studies, 158 fractures) that the time to fracture healing was significantly reduced in the ultrasound-treated groups than in the control groups and the mean difference in healing time was 64 days. The systematic review (Griffin et al. 2008) evaluating LIPUS for fresh fractures considers seven randomised controlled trials and two meta-analyses, suggesting that this body of evidence is supportive. Warden et al. (2000) published a review article and concluded from animal and human studies that the use of LIPUS could accelerate the rate of fracture repair by a factor of up to 1.6.

The units utilised for this work deliver a very low intensity (0.03 W cm^{-2} or 30 mW/cm^{-2}) at 1.5 MHz pulsed at a ratio of 1:4 at 1 kHz, applied for 20 minutes daily. An examples of a currently available specialist LIPUS device is illustrated in Figure 19.22. The intensity of this application is considerably lower than the lowest intensity which is deliverable by the majority of current therapy ultrasound machines (normally 0.1 W cm^{-2}) and although Warden et al. (Warden et al. 1999) have tried to replicate this using standard therapy devices, it has only been evaluated using an animal model to date and human trials with physiotherapy ultrasound machines have yet to be reported in the published literature. The key issue, in addition to the very low intensity, is that these specific devices have a particularly low beam uniformity (BNR) and thus are considered to be safe to apply with a stationary treatment head, unlike conventional physiotherapy ultrasound machines. This could be an important factor given the 20 minutes treatment duration on a daily basis, with a

static treatment applicator and, most commonly, with the patient using the device at home rather than attending for therapy.

Heckman et al. (1994) demonstrated a 38% reduction in the healing time for tibial fractures using a LIPUS device, while Kristiansen et al. (1997) demonstrated a 30% acceleration in healing for fractures of the radius. Jensen (1998) identifies the beneficial effects of ultrasound in the treatment (as opposed to the diagnosis) of stress fractures with an overall success rate of 96%. Mayr et al. (2000) report a series of outcomes when using LIPUS for patients with delayed unions (n = 951) and non-unions (n = 366). The overall success rate for the delayed unions was 91% for the delayed unions and 86% for the non-unions.

The authors undertook an interesting stratified analysis of their patients, and identified that those who were using non-steroidal anti-inflammatory drugs (NSAIDs), calcium channel-blockers or steroids had a less favourable outcome – a finding that could be considered to be consistent with several research publications that have tried to identify the mechanism by which the ultrasound could bring about fracture healing acceleration and other wider research concerning the adverse influence of NSAIDs on tissue repair (e.g. Evans and Butcher 2004; Tsai et al. 2004).

Of the reviews specific to LIPUS treatment of delayed union and non-union, Romano et al. (2009) identify a success rate for LIPUS of between 70% and 93%, which is impressive when compared with the 'typical' rates after surgery and bone graft. Rutten et al. (2007) reviewed tibial non-unions treated in the Netherlands with LIPUS and provide evidence for a 73% healing rate, significantly better than that achieved in non-LIPUS groups. Claes and Willie (2007) review the reviews and, in addition to an evaluation of the clinical effectiveness and uses, also try to make some sense of the wide range of proposed mechanisms by which this intervention achieves its effects. Further reviews are offered by Hadjiargyrou et al. (1998), Tsunoda (2003), Malizos et al. (2006), and Pounder and Harrison (2008), who identify a success rate of some 80% in Japan.

The use of such low doses has been shown to result in non-significant increases in tissue temperature. Using higher ultrasound doses could have an adverse effect on the fracture healing process (Chang et al. 2002) and LIPUS is considered to be effective and safe for this patient group. A review by Jingushi et al. (2007) usefully includes a commentary on the potential mechanisms for this effect, while Della Rocca (2009) also reviews the literature in this field, including the potential mechanisms of effect, though much of the work considered relates to animal and laboratory experimentation. The mechanisms by which therapeutic ultrasound can be effective for fracture repair includes nitric oxide pathways and prostaglandin (PGE_2) (Warden et al. 2001; Reher et al. 2002). This too would be consistent with other proposed mechanisms of ultrasound action (ter Haar 1999) and the relationship between the use of NSAIDs and tissue repair following injury.

Tis et al. (2002) and Sakurakichi et al. (2004) have evaluated the use of ultrasound as a component of treatment (in an animal model) during distraction osteogenesis; both demonstrated significant benefits. Cook et al. (2001) have demonstrated similar benefits following spinal fusion surgery and Tanzer et al. (2001) have shown that the use of ultrasound in combination with porous intramedullary implants is also beneficial.

LIPUS, therefore, is a well-evidenced and effective therapy intervention for fresh fractures, delayed unions and non-unions, and as an adjunctive treatment post-surgery. The treatment parameters are well defined and it appears to be most commonly employed as a home-based therapy on the basis that this is the most cost effective way to deliver the treatment. It is anticipated that therapists will become more involved in the organisation, delivery and monitoring of this treatment package as it becomes more widespread. It remains possible that future generations of ultrasound machines in clinics may have the facility to deliver this effective treatment dose so that it can be used as a clinic option if home-based therapy is impractical or unrealistic as an option. There have been suggestions, but little evidence to date, that the LIPUS approach may also be beneficial for musculoskeletal and soft tissue lesions. While there is anecdotal evidence, there is little as yet in the published work to provide unequivocal support, although this field may develop as swiftly as fracture research has over the last 10–15 years.

Pulsed shortwave therapy (PSWT)

PSWT is a widely used modality in the UK (Al Mandeel and Watson 2006), though it is often called pulsed electromagnetic energy (PEME) which is less than fully appropriate in that many modalities come under the heading of PEME – PSWT being only one of them – and the use of the term is best avoided. The older term 'pulsed shortwave diathermy' is not really appropriate either in that the modality is not primarily employed as a diathermy (literally 'through heating').

PSWT employs the same operating frequency as traditional SWD, i.e. 27.12 MHz. The output from the machine is pulsed such that the 'on' time is considerably shorter than the 'off' time, thus the mean power delivered to the patient is relatively low, even though the peak power (i.e. during the on pulses) can be quite high (typically around 150–200 W peak power with modern machines – examples of which are illustrated in Figure 19.23.

The control offered by the machine will enable the user to vary (a) the mean power delivered to the patient and (b) the pulsing parameters governing the mode of delivery of the energy. It would seem from current research that the mean power is probably the most

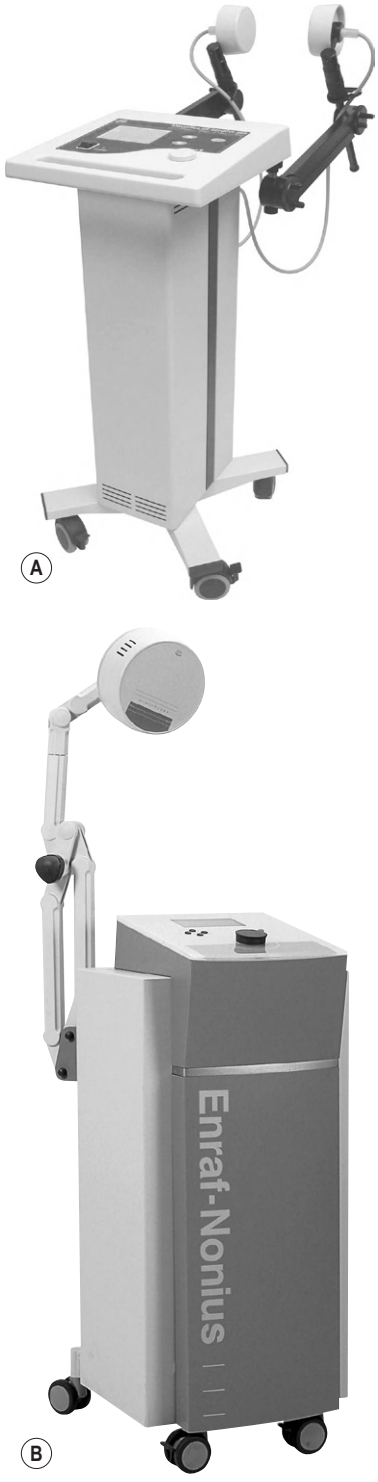


Figure 19.23 Examples of pulsed shortwave therapy devices: (a) Megapulse system (EMS Physio); (b) Curapuls system (Enraf).

important parameter (e.g. Hill et al. 2002; Al Mandeel and Watson 2008).

When using pulsed shortwave in the clinical environment, there are two 'modes' of application, and the information in this section relates to application using the 'monode' or 'drum' electrode rather than the capacitor plate delivery system which lacks sufficient supportive evidence.

Main machine parameters

Pulse repetition rate (Hz or pps)

The pulse repetition rate controls the number of pulses of shortwave energy that are delivered to the patient in a second. The units are often denoted in Hertz (Hz), although pulses per second (pps) is probably a more appropriate term. Most, if not all, machines offer a pre-set range of pulse repetition options with widely varying options. There is no evidence that the actual value of the pulse rate is critical, but the ability to deliver greater or fewer pulses per second does enable the user to influence the mean power (see below).

Pulse duration (width)

The pulse duration (often called the pulse width) refers to the duration, in microseconds, of each individual pulse of shortwave energy. Most, but not all machines, allow the therapist to adjust the pulse duration using a range of pre-set options. There is no evidence that the actual duration is a critical measure – it is simply a means of influencing mean power delivery.

Power output of the machine

The combination of having the facility to determine both the repetition rate of the pulses and the duration of each pulse enables the user to influence the mean power delivered to the patient.

The peak power (which can sometimes, but not always, be controlled by the operator) is typically around 150–200 W in modern machines. This is the 'strength' of the shortwave whilst the pulse is *on*, i.e. energy is delivered to the patient. The *mean power* takes account of the fact that there are *on* and *off* phases – the energy delivery is intermittent – and thereby describes the average power output rather than the power output at any one moment in time (which might be maximum or zero).

The relationship between the pulse parameters and the power levels are illustrated in Figure 19.24.

Mean power

The applied *mean power* appears to be the critical dose parameter in the clinical environment. Each different type of pulsed shortwave machine will have a 'mean power' table associated with it, and it is from this table that the therapist is able to establish the settings required to deliver

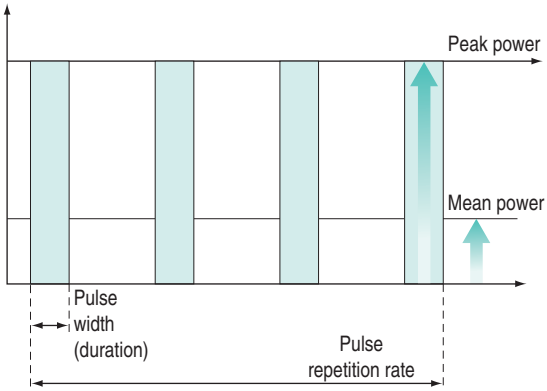


Figure 19.24 The relationship between pulse parameters and power levels with pulsed shortwave therapy.

a mean power of XX Watts. If, for example, it is intended to deliver a mean power of 5 W with a particular machine, the therapist should consult the table specific for that machine to determine exactly which combination of pulse repetition rate and pulse duration will provide the required mean power. It is important to note that these tables are not transferable between machines. The settings made on machine A and machine B to achieve the same mean power may well be different. Getting the mean power levels 'correct' appears to be more important than any other individual parameter.

Tissue heating

With respect to the effects of pulsed shortwave, there is an element of tissue heating which occurs during the 'on' pulse, but this is dissipated during the prolonged 'off' phase and, therefore, it is possible to give treatment with no *net* increase in tissue temperature. In Figure 19.25, part (a) demonstrates no accumulation of either thermal or non-thermal effects. In part (b), the pulses are sufficiently close to generate an accumulative non-thermal effect and in part (c) there is an accumulation of both thermal and non-thermal effects. The settings applied on the machine will determine which of these is achieved in a particular treatment. The 'non-thermal' effects of the modality are generally thought to be of greater significance. They appear to accumulate during the treatment time and have a significant effect after a latent period, possibly in the order of 6–8 hours. It is suggested (Hayne 1984) that the energy levels required to produce such an effect in humans are low.

Research has been conducted over several years relating to the thermal nature of PSWT. It was unclear just what power levels were required to bring about real tissue heating and, in fact, there has been some opinion that PSWT is a non-thermal modality. Research has demonstrated (e.g. Bricknell and Watson 1995) that PSWT does have a thermal component and that real tissue heating can occur under different treatment settings. This is important in that if the modality is to be applied in circumstances where the heating would be inappropriate or contraindicated, it is essential to know the power/energy levels where the thermal effects begin. This research shows that a measurable heating effect can be demonstrated at power levels over 5 Watts, though on average, it will become apparent at some 11 Watts mean power. More recent work (Seiger and Draper 2006) suggests that it may still be safe to apply higher mean power levels than previously thought, even with metal in the tissues. A clinical trial using PSWT at 48 Watts mean power in the tissues resulted in clinical benefit and no adverse outcomes, even when there was metal in the tissues.

If a 'non-thermal' treatment is the intended outcome of the treatment, it is essential that the mean power applied remains below a level that is likely to induce significant heating effects, and at present, this is taken as being at 5 Watts mean power. If a thermal effect is an intentional outcome of the intervention, then it may be perfectly appropriate to deliver power levels in excess of 5 Watts, but, if doing so, the therapist must ensure that the precautions are taken as for any other thermal intervention.

Effects of PSWT

Effects of PSWT can be divided into two basic types – those of the electric field and those of the magnetic field. There appears to be almost no literature/research concerning the effects of pulsing the electric field (using condenser plate electrodes) and all the research identified here is concerned with the therapeutic effects of the magnetic field (monode-type electrode).

The primary effects of the pulsed magnetic field appear to be at the cell membrane level and are concerned with the transport of ions across the membrane. Some interesting publications have strongly supported the 'non-thermal' effects at cell membrane level (Cleary 1996; Luben 1996).

Normal cell membranes exhibit a potential difference owing to the relative concentration differences of various ions on either side of the membrane (reviewed in Charman 1990). It is argued that the application of energy that is absorbed at the cell membrane level results in cellular 'up-regulation' and thereby increases levels of cellular activity – this is not a change in role, but effectively an increase in work rate (Al Mandeel and Watson 2008).

Cells involved in the inflammatory process demonstrate a reduced cell membrane potential and consequently, the cell function is disturbed. The altered potential affects ion transport across the membrane, and the resulting ionic imbalance alters cellular osmotic pressures. The application of PSWT to cells affected in this way is claimed to restore the cell membrane potential to their 'normal' values and also restores normal membrane

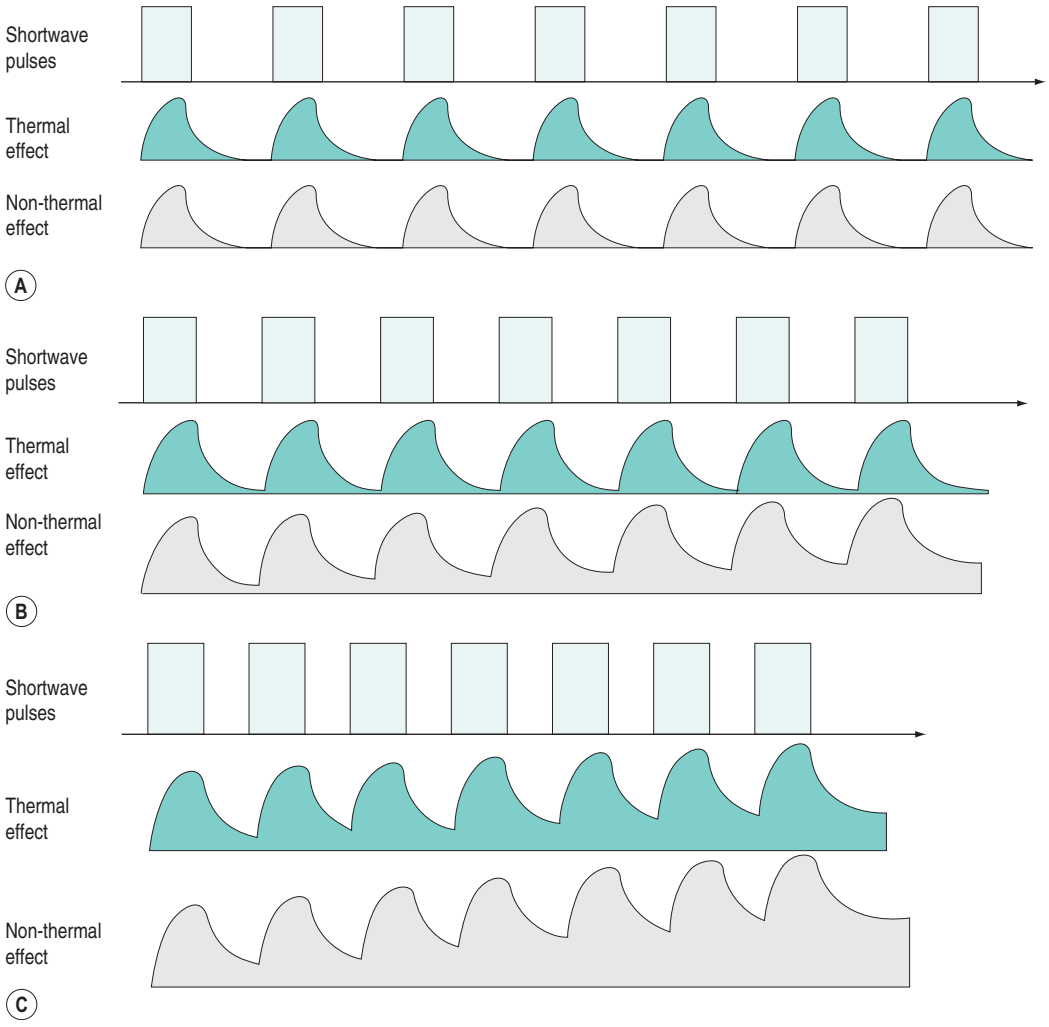


Figure 19.25 (a–c) Effect of varying the pulse parameters on the accumulated heat generated in the tissues. (a) Pulses at sufficient 'distance' – no accumulative effect. (b) Pulses closer together – accumulating non-thermal effect, no thermal accumulation. (c) Pulses closer still – accumulation of thermal and non-thermal effects.

transport and ionic balance. The mechanism by which this effect is brought about has not yet been established, but the two theories suggest that this is either a direct ionic transport mechanism or an activation of various pumps (sodium/potassium) by the pulsed energy. Evidence (Luben 1996) supports the contention that the energy is absorbed in the membrane and that via a mechanism of signal transduction, stimulates or enhances intracellular effects.

There appears to be a strong similarity in the mechanism of effect of ultrasound, laser and pulsed shortwave – all three modalities appear to have their primary effect at cell membrane level, with the resulting 'up-regulation' of cellular behaviour being the key to the therapeutic effects.

PSWT: Clinical effects

The clinical effects of PSWT are related primarily to the inflammatory and repair phases in musculoskeletal/soft tissues. Goldin et al. (1981) lists the effects of the modality (following research in soft tissue repair following skin graft application). Other articles have similarly identified the list of effects, which are most easily reviewed in the standard electrotherapy texts (Robertson et al. 2006; Watson 2006a, 2008c).

Suggested treatment doses

In light of current research, it is suggested that the minimum energy required to achieve a therapeutic effect should be utilised. Specific detailed research (clinical and laboratory) is essential for further validation of the treatment which is currently criticised for being unfounded. There is also a strong argument that the concentration of electromagnetic energy is likely to be critical, and it may be in the near future that PSWT doses are described more in terms of mean power concentration (W cm^{-2}) rather than just Watts. This would be in keeping with ultrasound and laser therapies.

The general guide below is based on both clinical and research evidence wherever possible. Further details can be found in specific research articles, standard texts and on the www.electrotherapy.org website.

Acute conditions

Apply a *mean power* of less than 3 Watts. The more acute the lesion presentation, the lower the delivered mean power (i.e. 3 Watts is maximal for this group). Using shorter duration (narrower) pulses and higher repetition rate may be beneficial and the typical treatment time is between 10 and 15 minutes.

Sub-acute conditions

Apply a *mean power* of between 2 and 5 Watts and as the condition becomes less acute, longer duration (wider) pulses appear to be preferable. Treatment time is typically in the region of 15 minutes.

Chronic conditions

Application of a mean power greater than 5 Watts is usually required in order to achieve a reasonable tissue response. As noted previously, when such mean powers are employed, it is important to remember that the 'thermal' effect of pulsed shortwave becomes apparent at power levels greater than 5 Watts, and therefore appropriate thermal precautions need to be taken. Pulses of longer duration are probably of benefit if there is a choice, and treatment times are typically between 15 and 20 minutes.

Some therapists 'worry' about using 'high' doses with PSWT, but even the higher doses on modern machines deliver considerably less energy than those used in the past without ill effect. Much as low doses appear to be very effective, especially for the more acute conditions, doses up to 48 Watts mean power are safe, evidenced and clinically effective in some (chronic) circumstances.

Laser therapy/low level laser therapy/low intensity laser therapy

The term 'laser' is an acronym for the light amplification by stimulated emission of radiation. In simple, yet realistic, terms, the laser can be considered to be a form of light amplifier – it provides enhancement of particular properties of light energy. Examples of commonly encountered laser therapy devices are illustrated in Figure 19.26.

Figure 19.26 Typical laser therapy devices: (a) THOR; (b) EMS Physio; (c) Gymna.

Laser light will behave according to the basic laws of light in that it travels in straight lines at a constant velocity in space. It can be transmitted, reflected, refracted and absorbed. It can be placed within the electromagnetic spectrum according to its wavelength/frequency, which

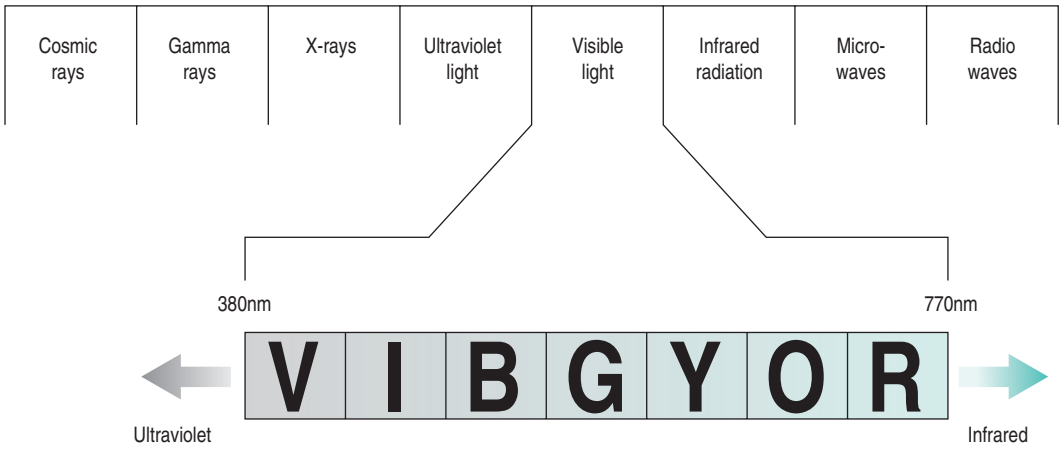


Figure 19.27 Light energy as a part of the electromagnetic spectrum.

will vary according to the particular generator under consideration (Figure 19.27).

There are several aspects of laser light which are deemed to be special and are often referred to in the literature (Figure 19.28). These include *monochromacity*, *coherence* and *polarisation*. There remains some doubt as to exactly how essential these particular aspects of laser light are in relation to the therapeutic application of this energy form. Monochromacity is probably the most important factor, as many of the therapeutic effects have been noted in various trials with light which is non-coherent. Additionally, it is thought that the polarisation is soon lost within the tissues and may therefore be less important than was thought at first.

Therapy lasers have several common characteristics which are summarised below.

Terms

Therapy lasers tend to fall into a particular category of laser light known as 3A or 3B. More recently, the terms low level laser therapy (LLLT) and low intensity laser therapy (LILT) have been adopted. Ohshiro and Calderhead (1988) suggest that LLLT involves treatment with a dose that causes no detectable temperature rise in the treated tissues and no macroscopically visible change in tissue structure – essentially, the energy can cause an increase in temperature and a change in tissue structure, but that is not the intention with therapy laser which is applied at levels below that needed to achieve these more overt effects (compared with surgical laser).

Parameters

Most LLLT apparatus generates light in the red visible and near infrared bands of the electromagnetic spectrum (Figure 19.27), with typical wavelengths of 600–1000 nm.

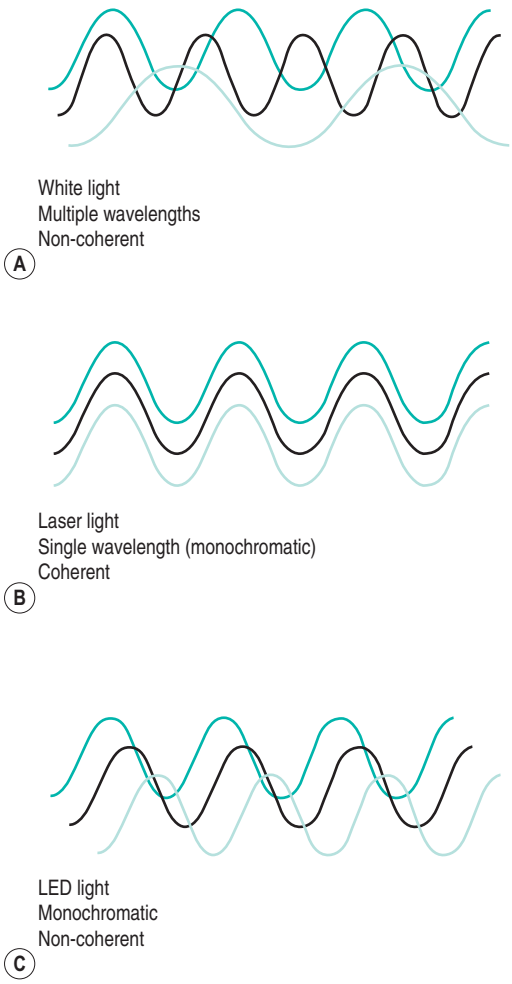


Figure 19.28 Characteristics of various light forms: (a) 'normal' white light; (b) true laser light; (c) LED-type laser light.

Figure 19.29 Laser therapy applicators: (a) cluster probes (Thor); (b) single and cluster probes (EMS Physio).

The mean power of such devices is generally low (1–100 mW), though the peak power may be much higher than this.

The treatment device may be a single emitter or a cluster of several emitters (Figure 19.29), though it is common for most emitters in a cluster to be non-laser-type devices. The beam from single probes is usually narrow (Ø1 mm to 6 or 7 mm) at the source. A cluster probe will usually incorporate both higher and lower power emitters of different wavelengths.

The output may be continuous or pulsed, with narrow pulse widths (in the nano- or microsecond ranges) and a wide variety of pulse repetition rates from 2 Hz up to several thousand Hz. It is difficult to identify the evidence for the use of pulsing from the research literature, though it would appear to be a general trend (anecdotal) that the lower pulsing rates are more effective in the acute conditions while higher pulse rates work better in more chronic conditions.

Many texts make reference to the types of generators according to the gas contained within the tube, but in therapy applications most modern apparatus utilise laser diodes, most commonly made of gallium aluminium arsenide (GaAlAs) in which the ratio of the constituents determines the wavelength of the output or alternatively, laser light emitting diodes (LEDs) of various kinds.

Light absorption in the tissues

As with any form of energy used in electrotherapy, the energy must be absorbed by the tissues in order to have some effect. The absorption of light energy within the tissues is a complex issue, but, generally, the shorter wavelengths (ultraviolet and shorter visible) are primarily absorbed in the epidermis by the pigments, amino and nucleic acids. The longer infrared radiation wavelengths (>1300 nm) appear to be rapidly absorbed by water and therefore have limited penetration into the tissues. The bands between (i.e. 600–1000 nm) are capable of penetration beyond the superficial epidermis and are, in part at least, available for absorption by other biological tissues.

When applied to the body tissues, LLLT delivers energy at a level sufficient to disturb local electron orbits and result in the generation of heat, initiate chemical change, disrupt molecular bonds and produce free radicals. These are considered to be the primary mechanisms by which LLLT achieves its physiological, and therefore therapeutic, effects and the primary target is effectively the cell membrane (see below).

Although much of the applied laser light is absorbed in the superficial tissues, it is proposed that deeper or more distant effects can be achieved, probably as a secondary consequence via some chemical mediator or second messenger systems, though there is limited evidence to fully support this contention.

The actual penetration of LLLT at common wavelengths is a widely debated point and it is common to find widely varying values cited in the literature. It is often claimed that because laser light is monochromatic, polarised and coherent it is capable of greater penetration than 'normal' (or non-coherent) light. This should give penetration depths of 3–7 mm for visible red light and some 30–40 mm (at best) for infrared radiation laser light. The fact that the polarisation appears to be lost in the tissues, as is much, if not all, of the coherence, will result in a shallower penetration. King (1990) cites a penetration depth for 630 nm light to be 1–2 mm, while at 800–900 nm penetration depths of 2–4 mm are expected. (Penetration depth in this context refers to the depth of the tissues to which 37% of the light at the surface is able to penetrate.) A very small percentage of the light energy available at the surface will be available at 10–15 mm into the tissues.

Laser–tissue interaction

Photobioactivation is a commonly used phrase in connection with LILT. It means the stimulation of various

biological events using light energy but without significant temperature changes. Much, if not all, of the cited work on therapeutic lasers consider these photobioactivation effects. Some authors have proposed that there are other terms which are preferable, including *photobiostimulation* and *photobiomodulation*. It provides for a great semantic argument, but assume at this point that these terms are generally interchangeable.

Many of the early ideas of photobioactivation were proposed by Karu who reported and demonstrated several key factors. She notes in her 1987 article that some biomolecules change their activity in response to irradiation with low intensity visible light, but that these molecules do not appear to absorb the light directly. The cell membrane appears to be the primary absorber of the energy which then generates intracellular effects by means of a second messenger/cascade-type response. The magnitude of the photoresponse was deemed to be determined at least in part by the state of the cells/tissues prior to irradiation, summarised in a beautifully simple statement that 'starving cells are more photosensitive than well fed ones'. The laser light irradiation of the tissues is seen then as a trigger for the alteration of cell metabolic processes via a process of photosignal transduction. The often cited Arndt-Schults Law supports this proposal.

The following list of physiological and cellular level effects is compiled from several reviews and research papers (e.g. Baxter 2002, 2008; Tuner and Hode 2004) and does not claim to be complete or guaranteed for the *in vivo* situation. It does, however, illustrate the range and scope of photobioactivation effects:

- altered cell proliferation;
- altered cell motility;
- activation of phagocytes;
- stimulation of immune responses;
- increased cellular metabolism;
- stimulation of macrophages;
- stimulation of mast cell degranulation;
- activation and proliferation of fibroblasts;
- alteration of cell membrane potentials;
- stimulation of angiogenesis;
- alteration of action potentials;
- altered prostaglandin production;
- altered endogenous opioid production.

Treatment doses

Most research groups and many manufacturers recommend that the dose delivered to a patient during a treatment session should be based on the *energy density* rather than the power or other measure of dose. Energy density is measured in units of Joules per square centimetre (J/cm^2). One of the most significant inhibitors to the more widespread adoption of laser therapy in the clinical environment relates to the difficulty in getting these 'effective' laser doses to work on a particular machine. Few devices enable the practitioner to set the dose in J/cm^2. Some will provide Joules, some Watts and some $Watts/cm^{-2}$. It is currently argued that Joules (i.e. energy) may, in fact, be the most critical parameter rather than energy density. The debate is not yet resolved. Energy density will be used here, mainly because the published research almost exclusively cites it and, therefore, it may be of more use when it comes to trying to replicate an evidence-based treatment dose.

Some machines offer 'on board' calculations of this dose, while other machines require the operator to make some calculations based on known machine parameters:

- output power (Watts);
- irradiation area (cm^2);
- time (seconds);
- if pulsed – pulse width, frequency and power settings.

$$\text{Energy density (J/cm}^2\text{)} = \frac{\text{Total amount of energy (J)}}{\text{Irradiation area (cm}^2\text{)}}$$

$$\text{Total energy (J)} = \text{Average power (Watts)} \times \text{time (sec)}$$

$$\text{Average power (Watts) (pulsed output only)} = \text{Peak power (W)} \times \text{frequency (Hz)} \times \text{pulse duration (sec)}$$

There are various alternative methods for calculating these doses, but those cited above offer a reasonably simple method should one be needed.

Most authorities suggest that the energy density per treatment session should generally fall in the range of 0.1–12.0 J/cm^2 although there are some recommendations which go up to 30 J/cm^2. Again, as a generality, lower doses should be applied to the more acute lesions which would appear to be more energy sensitive.

Clinical applications

Recent research, both laboratory-based and clinical trials, is found to concentrate on a few key areas. Most dominant among these are wound healing, inflammatory arthropathies, soft tissue injury and the relief of pain (which includes laser acupuncture). There is supportive research for the clinical use of LLLT in these and other circumstances, but, as with many treatment modalities, the evidence remains somewhat controversial at the present time.

Open wounds

There is a growing body of evidence in this context, with some mixed results but, on the whole, they are positive outcome trials. There are useful sections in Baxter (1994, 2008), and Tuner and Hode (2004). A general summary might conclude that a treatment programme could be: treat to the floor of the ulcer/pressure sore/wound, preferably using a cluster probe to cover the area, typically up to 2 J/cm^2. Also treat the margin/periphery normally using

a single probe with a dose typically up to 4 J/cm^2. Care will need to be taken to ensure infection control policies are maintained, especially with laser treatment applicators being placed in contact with wounds.

Inflammatory arthropathies

There have been several trials involving the use of LILT and various inflammatory problems in joints. As with the wound work, there are mixed results, but the general trend appears to be supportive. The Ottawa Panel (Ottawa-Panel 2004) review was supportive of laser therapy in rheumatoid arthritis.

Soft tissue injury

There is a fairly widespread use of LILT in a variety of soft tissue treatments. Some results are excellent and others poor. It is possible that the weak results relate to incorrect doses or possibly considering the use of laser therapy for injuries that are simply beyond the reach of the energy delivered. Tuner and Hode (2004) describe multiple examples of effective (and less effective) soft tissue treatments with LILT and identify some of the key research in this area. Further material is also included in the Baxter (2008) review.

Pain

It has been broadly assumed (until recently) that the effect of laser therapy with regards to pain relief was primarily a secondary effect of dealing with the inflammatory state. While this may well be true (to some extent at least), there is growing evidence that laser therapy can have a more direct effect of nerve conduction characteristics and hence may result in reduced pain as a more direct effect of the therapy (e.g. Vinck et al. 2005).

The therapeutic use of lasers has increased slowly in the general practice of therapy, though there are specialist areas where it is strongly advocated, for example wound healing. One of the key issues limiting the more widespread uptake of the modality is the apparent difficulty in deciding on an effective clinical dose and then setting the machine to deliver such a dose. It is hoped that with increases in technology, this will become an easier task and, thus, the clinical application of laser therapy is likely to increase in the short- to medium-term future.

The last of the 'non-thermal' modalities that will be briefly included is shockwave therapy, which is emerging in both practice and research terms. It is difficult to know where it should 'fit' in the categorisation of electrophysical agents. It employs a mechanical energy (like ultrasound), is applied at high energy (like the thermal modalities) but is not used as a heating method, so it is included here under the non-thermal applications. At some point there might be a need for a 'new' mechanical energy group (shockwave, whole body vibration, deep oscillation therapy), but that would leave ultrasound either in the 'wrong' group or dissociated from its therapeutic partners.

Shockwave therapy

Shockwave therapy in the context of therapy practice is a relatively recent development and, like LIPUS and microcurrent therapies, is rapidly gaining ground both in research and in practice terms. This section only aims to provide a brief overview of this treatment approach. At the present time, it remains unclear who the dominant users of this treatment will be – specialist technicians, orthopaedic surgeons or physiotherapists.

Shock waves were initially employed as a non-invasive treatment for kidney stones (from the early 1970s, with treatment proper starting in the 1980s) and it has become a first-line intervention for such conditions. In the process of the animal model experimentation associated with this work, it was identified that shock waves could have an (adverse) effect on bone. This led to a series of experimental investigations looking at the effect of shock waves on bone, cartilage and associated soft tissues (tendon, ligament, fascia) resulting in what is now becoming an intervention of increasing popularity, most especially for the recalcitrant lesions of these tissues.

The treatment goes by several names, the most popular being *shockwave therapy* or *extracorporeal shockwave therapy*, though, as ever, there are several variations, often linked to the names of particular machines and systems. Obvious examples of shock waves are the sonic boom from an aircraft, thunder or the sound following an explosion. A very readable but succinct history of the development of shock waves for medical applications can be found in Thiel (2001).

Basically, there are three different ways to produce the 'shock wave', which, without getting too technical are: spark discharge/piezoelectric, electromagnetic and pneumatic (ballistic). The wave that is generated will vary in its energy content and will also have different penetration characteristics in human tissue. The pneumatic (ballistic) generators produce a gently divergent wave which appears to have the better options in 'therapy' (the other generators normally generate a focussed beam, mainly for non-therapy applications).

A clinically useful shock wave is effectively a controlled explosion (Ogden et al. 2001) and when it enters the tissues, it will be reflected, refracted, transmitted and dissipated like any other energy form. The energy content of the wave will vary and the propagation of the wave will vary with tissue type. Just like an ultrasound wave, the shock wave consists of a high pressure phase followed by a low pressure (or relaxation) phase. When a shock wave reaches a 'boundary', some of the energy will be reflected and some transmitted.

The characteristics of a clinical shock wave are (Ogden et al. 2001; Speed 2004):

- peak pressure (typically 35–120 MPa);
- fast pressure rise (usually <10 nanoseconds);
- short duration (usually about 10 microseconds);
- narrow effective beam (2–8 mm diameter).

Broadly speaking, there are high and low power applications. The high power treatments (>0.6 mJ/mm^2) are 'one off' and generate a fair amount of pain such that some form of local anaesthetic is usually needed for the patient to be able to tolerate the treatment. The more common approach in therapy is to use several (usually 3–5) applications at low power (up to 0.08 mJ/mm^2 – no anaesthetic required) with typically 1000–1500 shocks delivered per session.

The pressure wave causes direct (linked to the high pressure part of the cycle) and indirect effects (associated with the subsequent low pressure part of the cycle during which cavitation will occur – as with therapeutic ultrasound). The collapse of these cavitations (bubbles) is, in part at least, responsible for the efficacy of the therapy.

As the shock wave travels through a medium and comes to an interface, part of the wave will be reflected and part transmitted. The dissipation of the energy at the interface is almost certainly responsible for the generation of the physical, physiological and thus the therapeutic effects. While the detailed mechanisms of how these are achieved remains elusive, there is a growing body of evidence that supports the use of the therapy in several different clinical areas.

In the therapy arena, the best evidence appears to be with the chronic tendinopathies (e.g. Rompe et al. 2008, 2009) in which case shock wave therapy was shown to be effective in isolation, but when combined with an eccentric loading programme, better results were achieved. There is also clinical evidence for fracture healing problems (delayed or non-union) and numerous other soft tissue clinical problems, especially those concerning ligament and fascia. Useful reviews can be found in Chung and Wiley (2002), Wilbert (2002), Mouzopoulos et al. (2007), Rompe and Maffulli (2007) and van Leeuwen et al. (2009).

Examples of current therapy-type shockwave machines and applications are illustrated in Figure 19.30.

As identified in the introductory comments, shock wave is not yet a 'normal' therapy intervention, but it is likely

Figure 19.30 Examples of therapy-based shockwave machines and application: (A) Swiss DolorClast (Spectrum Technology); (B) Shock Master (Gymna); (C) Lateral elbow shockwave therapy (Spectrum Technology).

to move into common practice based on the rapidly growing body of supportive evidence. Therapists applying this modality at low power over several sessions appears to be the most likely way forward and the early clinical results are encouraging.

SUMMARY

In summary, electrotherapy has an evidence-based role in physiotherapy practice. It has been the subject of considerable debate over recent years, but the evidence base identifying what it is (and what it is not) capable of achieving is substantial. This chapter has attempted to summarise the key modalities in use in the current clinical environment, identified some emerging modalities and has alluded to others. The gradual transition from the term 'electrotherapy' to a more encompassing term 'electrophysical agents' is inevitable and to be welcomed.

Each modality will have a range of specific physiological effects and these can be employed in turn to bring about therapeutic effects. Modalities have some commonality in terms of their modes of action and the modalities have been grouped together in this chapter on that basis – electrical stimulation modalities, thermal modalities and 'non-thermal' modalities.

By employing an evidence-based clinical decision-making model, it is possible to make rational and evidenced therapeutic decisions with regard to which modality (if any) to use and when. The details of dose-based decisions have been largely omitted from this text, but making the appropriate decision with regard to which modality to employ in particular circumstances is the first, and probably the critical, choice. Once the most appropriate modality has been identified, the specific treatment 'dose' needs to be identified. Research articles and standard electrotherapy texts are available in addition to online resources to facilitate these detailed decisions.

The overuse of electrotherapy in the past has resulted in the demise of this area of practice. This chapter has attempted to identify the key issues for commonly available modalities, to illustrate their potential clinical applications based on the available evidence and to identify detailed sources for further information.

Weblinks

Alpha Stim: www.themicrocurrentsite.co.uk
Body Clock Health Care Ltd: www.bodyclock.co.uk
Chatanooga (part of the DJO Group): www.chattgroup.com/
Elexoma: www.elexoma.com/
EMS Physio Limited: www.emsphysio.co.uk
Enraf Nonius: www.enraf-nonius.com
Gymna Uniphy: www.gymna-uniphy.com/
Indiba: www.indiba.com
Natures Gate: www.naturesgate-uk.com/
Neurotech: www.neurotech.co.uk/
Smith and Nephew (Exogen): global.smith-nephew.com
Spectrum Technology: www.spectrumtechnologyuk.com
Synapse Tendonworks: www.tendonworks.com/index.html
TensCare Ltd: www.tenscare.co.uk
THOR International: www.thorlaser.com/

REFERENCES

Aarskog, R., Johnson, M.I., Demmink, J.H., et al., 2007. Is mechanical pain threshold after transcutaneous electrical nerve stimulation (TENS) increased locally and unilaterally? A randomized placebo-controlled trial in healthy subjects. Physiother Res Int 12 (4), 251–263.

Ada, L., Foongchomcheay, A., 2002. Efficacy of electrical stimulation in preventing or reducing subluxation of the shoulder after stroke: a meta-analysis. Aust J Physiother 48 (4), 257–267.

Al Mandeel, M., Watson, T., 2006. An evaluative audit of patient records in electrotherapy with specific reference to pulsed short wave therapy (PSWT). Int J Ther Rehab 13 (9), 414–419.

Al Mandeel, M., Watson, T., 2008. Pulsed and continuous shortwave therapy. In: Watson, T. (Ed.), Electrotherapy: Evidence Based Practice. Churchill Livingstone/Elsevier, Edinburgh, pp. 137–160.

Amaro, J.L., Oliveira Gameiro, M.O., Padovani, C.R., 2003. Treatment of urinary stress incontinence by intravaginal electrical stimulation and pelvic floor physiotherapy. Int Urogynecol J Pelvic Floor Dysfunct 14 (3), 204–208.

Ayling, J., Marks, R., 2000. Efficacy of paraffin wax baths for rheumatoid arthritic hands. Physiotherapy 86 (4), 190–201.

Baker, K.G., Robertson, V.J., Duck, F.A., 2001. A review of therapeutic ultrasound: biophysical effects. Phys Ther 81 (7), 1351–1358.

Barroso, J.C., Ramos, J.G., Martins-Costa, S., et al., 2004. Transvaginal electrical stimulation in the treatment of urinary incontinence. BJU Int 93 (3), 319–323.

Baxter, D., 1994. Therapeutic Lasers: Theory & Practice. Churchill Livingstone, Edinburgh.

Baxter, D., 2002. Low intensity laser therapy. In: Kitchen, S. (Ed.), Electrotherapy: Evidence Based Practice. Elsevier, Oxford.

Baxter, D., 2008. Low intensity laser therapy. In: Watson, T. (Ed.), Electrotherapy: Evidence Based Practice. Elsevier/Churchill Livingstone, Edinburgh, pp. 161–178.

Belanger, A.-Y., 2010. Therapeutic Electrophysical Agents: Evidence Behind Practice. Lippincott Williams and Wilkins, Philadelphia.

Bircan, C., Senocak, O., Peker, O., et al., 2002. Efficacy of two forms of electrical stimulation in increasing quadriceps strength: a randomized controlled trial. Clin Rehabil 16 (2), 194–199.

Bjordal, J.M., Johnson, M.I., Ljunggreen, A.E., 2003. Transcutaneous electrical nerve stimulation (TENS) can reduce postoperative analgesic consumption. A meta-analysis with assessment of optimal treatment parameters for postoperative pain. Eur J Pain 7 (2), 181–188.

Bricknell, R., Watson, T., 1995. The thermal effects of pulsed shortwave therapy. Br J Therapy Rehab 2 (8), 430–434.

Brosseau, L., Robinson, V., Pelland, L., et al., 2002. Efficacy of thermotherapy for rheumatoid arthritis: a meta analysis. Phys Ther Rev 7, 5–15.

Busse, J.W., Bhandari, M., Kulkarni, A.V., et al., 2002. The effect of low-intensity pulsed ultrasound therapy on time to fracture healing: a meta-analysis. CMAJ 166 (4), 437–441.

Byl, N.N., Hill Toulouse, L., Sitton, P., et al., 1996. Effects of ultrasound on the orientation of fibroblasts: an in-vitro study. Eur J Phys Med Rehabil 6 (6), 180–184.

Callaghan, M.J., Oldham, J.A., 2004. Electric muscle stimulation of the quadriceps in the treatment of patellofemoral pain. Arch Phys Med Rehabil 85 (6), 956–962.

Chang, W.H., Sun, J.S., Chang, S.P., et al., 2002. Study of thermal effects of ultrasound stimulation on fracture healing. Bioelectromagnetics 23 (4), 256–263.

Chantraine, A., Baribeault, A., Uebelhart, D., et al., 1999. Shoulder pain and dysfunction in hemiplegia: effects of functional electrical stimulation. Arch Phys Med Rehabil 80 (3), 328–331.

Charman, R.A., 1990. Bioelectricity and electrotherapy – towards a new paradigm? Part 2: Cellular reception and emission of electromagnetic signals. Physiotherapy 76 (9), 509–516.

Chesterton, L.S., Barlas, P., Foster, N.E., et al., 2002. Sensory stimulation (TENS): effects of parameter manipulation on mechanical pain thresholds in healthy human subjects. Pain 99, 253–262.

Chipchase, L.S., Williams, M.T., Robertson, V.J., 2009. A national study of the availability and use of electrophysical agents by Australian physiotherapists. Physiother Theory Pract 25 (4), 279–296.

Christie, A.D., Willoughby, G.L., 1990. The effect of interferential therapy on swelling following open reduction and internal fixation of ankle fractures. Physiother Theory Pract 6, 3–7.

Chung, B., Wiley, J.P., 2002. Extracorporeal shockwave therapy: a review. Sports Med 32 (13), 851–865.

Ciccone, C.D., Leggin, B.G., Callamaro, J.J., 1991. Effects of ultrasound and trolamine salicylate phonophoresis on delayed-onset muscle soreness. Phys Ther 71 (9), 666–675.

Ciombor, D.M., Aaron, R.K., 2005. The role of electrical stimulation in bone repair. Foot Ankle Clin 10 (4), 579–593.

Claes, L., Willie, B., 2007. The enhancement of bone regeneration by ultrasound. Prog Biophys Mol Biol 93 (1–3), 384–398.

Cleary, S.F., 1987. Cellular effects of electromagnetic radiation. IEEE Eng Med Biol 6 (1), 26–30.

Cleary, S.F., 1996. In vitro studies of the effects of nonthermal radiofrequency and microwave radiation. In: Non-thermal Effects of RF Electromagnetic Fields. ICNIRP, Munich.

Cook, S.D., Salkeld, S.L., Patron, L.P., et al., 2001. Low-intensity pulsed ultrasound improves spinal fusion. Spine J 1, 246–254.

Della Rocca, G.J., 2009. The science of ultrasound therapy for fracture healing. Indian J Orthop 43 (2), 121–126.

Dinno, M., 1989. The significance of membrane changes in the safe and effective use of therapeutic and diagnostic ultrasound. Phys Med Biol 34, 1543.

Draper, D.O., Ricard, M.D., 1995. Rate of temperature decay in human muscle following 3 MHz ultrasound: the stretching window revealed. J Athl Train 30 (4), 304–307.

Draper, D., Sunderland, S., Kirkendall, D., et al., 1993. A comparison of temperature rise in human calf muscle following applications of underwater and topical gel ultrasound. JOSPT 17, 247–251.

Draper, D.O., Schulties, S., Sorvisto, P., et al., 1995. Temperature changes in deep muscles of humans during ice and ultrasound therapies: an in vivo study. J Orthopaed Sport Phys Ther 21 (3), 153–157.

Dyson, M., Smalley, D., 1983. Effects of ultrasound on wound contraction. In: Millner, R., Rosenfeld, E., Cobet, U. (Eds.), Ultrasound Interactions in Biology & Medicine. Plenum Press, New York, pp. 151–158.

Enwemeka, C.S., 1989. The effects of therapeutic ultrasound on tendon healing. A biomechanical study. Am J Phys Med Rehabil 68 (6), 283–287.

Enwemeka, C.S., Rodriguez, O., Mendosa, S., 1990. The biomechanical effects of low-intensity ultrasound on healing tendons. Ultrasound Med Biol 16 (8), 801–807.

Evans, C.E., Butcher, C., 2004. The influence on human osteoblasts in vitro of non-steroidal anti-inflammatory drugs which act on different cyclooxygenase enzymes. J Bone Joint Surg Br 86 (3), 444–449.

Evans, R.D., Foltz, D., Foltz, K., 2001. Electrical stimulation with bone and wound healing. Clin Podiatr Med Surg 18 (1), 79–95.

Frizzell, L.A., Dunn, F., 1982. Biophysics of ultrasound. In: Lehmann, J. (Ed.), Therapeutic Heat and Cold. Williams & Wilkins, Baltimore.

Fuentes, J.P., Armijo Olivo, S., Magee, D.J., et al., 2010. Effectiveness of interferential current therapy in the management of musculoskeletal pain: A systematic review and meta-analysis. Phys Ther 90 (9), 1219–1238.

Goldin, J.H., Broadbent, N., Nancurrow, J., et al., 1981. The effects of Diapulse on the healing of wounds: A double blind randomised controlled trial in man. Br J Plastic Surgery 34, 267–270.

Griffin, X.L., Costello, I., Costa, M.L., 2008. The role of low intensity pulsed ultrasound therapy in the management of acute fractures: a systematic review. J Trauma 65 (6), 1446–1452.

Hadjiargyrou, M., McLeod, K., Ryaby, J.P., et al., 1998. Enhancement of fracture healing by low intensity ultrasound. Clin Orthop 355 (Suppl.), S216–S229.

Han, J.S., Chen, X.H., Sun, S.L., et al., 1991. Effect of low- and high-frequency TENS on Met-enkephalin-Arg-Phe and dynorphin A immunoreactivity in human lumbar CSF. Pain 47 (3), 295–298.

Harvey, W., Dyson, M., Pond, J.B., et al., 1975. The stimulation of protein synthesis in human fibroblasts by therapeutic ultrasound. Rheumatol Rehabil 14, 237.

Hayne, C.R., 1984. Pulsed high frequency energy – its place in physiotherapy. Physiotherapy 70, 459–464.

Heckman, J.D., Ryaby, J.P., McCabe, J., et al., 1994. Acceleration of tibial fracture-healing by non-invasive, low-intensity pulsed ultrasound. J Bone Joint Surg Am 76 (1), 26–34.

Hill, J., Lewis, M., Mills, P., et al., 2002. Pulsed short-wave diathermy effects on human fibroblast proliferation. Arch-Phys-Med-Rehabil 83 (6), 832–836.

Hurley, D.A., McDonough, S.M., Dempster, M., et al., 2004. A randomized clinical trial of manipulative therapy and interferential therapy for acute low back pain. Spine 29 (20), 2207–2216.

Huys, S., Gan, B.S., Sherebrin, M., 1993. Comparison of effects of early and late ultrasound treatment on tendon healing in the chicken limb. J Hand Ther 6, 58–59.

Indrekvam, S., Fosse, O.A., Hunskaar, S., 2001. A Norwegian national cohort of 3198 women treated with home-managed electrical stimulation for urinary incontinence—demography and medical history. Scand J Urol Nephrol 35 (1), 26–31.

Jarit, G.J., Mohr, K.J., Waller, R., et al., 2003. The effects of home interferential therapy on post-operative pain, edema, and range of motion of the knee. Clin J Sport Med 13 (1), 16–20.

Jensen, J.E., 1998. Stress fracture in the world class athlete: a case study. Med Sci Sports Exerc 30 (6), 783–787.

Jingushi, S., Mizuno, K., Matsushita, T., et al., 2007. Low-intensity pulsed ultrasound treatment for postoperative delayed union or nonunion of long bone fractures. J Orthop Sci 12 (1), 35–41.

Johnson, M.I., 2008. Transcutaneous electrical nerve stimulation (TENS). In: Watson, T. (Ed.), Electrotherapy: Evidence Based Practice. Elsevier/Churchill Livingstone, Edinburgh, pp. 253–296.

Johnson, M.I., Tabasam, G., 2003. An investigation into the analgesic effects of different frequencies of the amplitude-modulated wave of interferential current therapy on cold-induced pain in normal subjects. Arch Phys Med Rehabil 84 (9), 1387–1394.

Johnson, M., Martinson, M., 2007. Efficacy of electrical nerve stimulation for chronic musculoskeletal pain: a meta-analysis of randomized controlled trials. Pain 130 (1–2), 157–165.

Jorge, S., Parada, C.A., Ferreira, S.H., et al., 2006. Interferential therapy produces antinociception during application in various models of inflammatory pain. Phys Ther 86 (6), 800–808.

Karu, T.I., 1987. Photobiological fundamentals of low power laser therapy. J Quantum Electr 23 (10), 1703–1717.

King, P.R., 1990. Low level laser therapy; A review. Physiother Theory Prac 6, 127–138.

Kitchen, S.S., Partridge, C.J., 1991. Infra-red therapy. Physiotherapy 77 (4), 249–254.

Kitchen, S.S., Dyson, M., 2008. Low energy treatments: non-thermal or microthermal. In: Watson, T. (Ed.), Electrotherapy: Evidence Based Practice. Elsevier/Churchill Livingstone, Edinburgh, pp. 115–122.

Kristiansen, T.K., Ryaby, J.P., McCabe, J., et al., 1997. Accelerated healing of distal radial fractures with the use of specific, low-intensity ultrasound. A multicenter, prospective, randomized, double-blind, placebo-controlled study. J Bone Joint Surg Am 79 (7), 961–973.

Lake, D.A., 1992. Neuromuscular electrical stimulation: An overview and its application in the treatment of sports injuries. Sport Med 13 (5), 320–336.

Lambert, I., Tebbs, S.E., Hill, D., et al., 2000. Interferential therapy machines as possible vehicles for cross-infection. J Hosp Infect 44 (1), 59–64.

Lau, P.M., Chow, D.H., Pope, M.H., 2008. Early physiotherapy intervention in an Accident and Emergency Department reduces pain and improves satisfaction for patients with acute low back pain: a randomised trial. Aust J Physiother 54 (4), 243–249.

Lehmann, J., 1982. Therapeutic Heat and Cold. Williams & Wilkins, Baltimore.

Leung, M.S., Cheung, G.L., 2008. Effects of deep and superficial heating in the management of frozen shoulder. J Rehabil Med 40 (2), 145–150.

Leung, K.S., Cheung, W.H., Zhang, C., et al., 2004. Low intensity pulsed ultrasound stimulates osteogenic activity of human periosteal cells. Clin Orthop Rel Res 418, 253–259.

Luben, R.A., 1996. Effects of microwave radiation on signal transduction processes of cells in vitro. In: Non-thermal Effects of RF Electromagnetic Fields. ICNIRP, Munich.

Lyons, C.L., Robb, J.B., Irrgang, J.J., et al., 2005. Differences in quadriceps femoris muscle torque when using a clinical electrical stimulator versus

a portable electrical stimulator. Phys Ther 85 (1), 44–51.

McDonaugh, S., 2008. Neuromuscular and muscular electrical stimulation. In: Watson, T. (Ed.), Electrotherapy: Evidence Based Practice. Elsevier/ Churchill Livingstone, Edinburgh, pp. 231–252.

McManus, F.J., Ward, A.R., Robertson, V.J., 2006. The analgesic effects of interferential therapy on two experimental pain models: cold and mechanically induced pain. Physiotherapy 92 (2), 95–102.

Malizos, K.N., Hantes, M.E., Protopappas, V., et al., 2006. Low-intensity pulsed ultrasound for bone healing: an overview. Injury 37 (Suppl. 1), S56–S62.

Maxwell, L., 1992. Therapeutic ultrasound: Its effects on the cellular and molecular mechanisms of inflammation and repair. Physiotherapy 78 (6), 421–426.

Mayer, J.M., Mooney, V., Matheson, L.N., et al., 2006. Continuous low-level heat wrap therapy for the prevention and early phase treatment of delayed-onset muscle soreness of the low back: a randomized controlled trial. Arch Phys Med Rehabil 87 (10), 1310–1317.

Mayr, E., Frankel, V., Ruter, A., 2000. Ultrasound–an alternative healing method for nonunions? Arch Orthop Trauma Surg 120 (1–2), 1–8.

Meakins, A., Watson, T., 2006. Longwave ultrasound and conductive heating increase functional ankle mobility in asymptomatic subjects. Physical Therapy in Sport 7, 74–80.

Michlovitz, S., 1996. Thermal Agents in Rehabilitation. FA Davis Co., Philadelphia.

Michlovitz, S., Hun, L., Erasala, G.N., et al., 2004. Continuous low-level heat wrap therapy is effective for treating wrist pain. Arch Phys Med Rehabil 85 (9), 1409–1416.

Miller, D.L., Gies, R.A., 1998. The interaction of ultrasonic heating and cavitation in vascular bioeffects on mouse intestine. Ultrasound Med Biol 24 (1), 123–128.

Mortimer, A.J., Dyson, M., 1988. The effect of therapeutic ultrasound on calcium uptake in fibroblasts. Ultrasound Med Biol 14 (6), 499–506.

Mouzopoulos, G., Stamatakos, M., Mouzopoulos, D., et al., 2007. Extracorporeal shock wave treatment for shoulder calcific tendonitis: a systematic review. Skeletal Radiol.36 (9), 803–811.

Neder, J.A., Sword, D., Ward, S.A., et al., 2002. Home based neuromuscular electrical stimulation as a new rehabilitative strategy for severely disabled patients with chronic obstructive pulmonary disease (COPD). Thorax 57 (4), 333–337.

Noble, J.G., Henderson, G., Cramp, A.F., et al., 2000. The effect of interferential therapy upon cutaneous blood flow in humans. Clin Physiol 20 (1), 2–7.

Nussbaum, E.L., 1997. Ultrasound: to heat or not to heat – that is the question. Phys Ther Rev 2, 59–72.

Nussbaum, E., 1998. The influence of ultrasound on healing tissues. J Hand Ther 11 (2), 140–147.

Ogden, J., Alvarez, R., Levitt, R., et al., 2001. Shock wave therapy (Orthotripsy) in musculoskeletal disorders. Clin Orthopaed Rel Res 387, 22–40.

Oshiro, T., Calderhead, R., 1988. Low Level Laser Therapy: A practical introduction. Wiley-Blackwell.

Ottawa-Panel, 2004. Ottawa panel evidence-based clinical practice guidelines for electrotherapy and thermotherapy interventions in the management of rheumatoid arthritis in adults. Phys Ther 84 (11), 1016–1043.

Ozcan, J., Ward, A.R., Robertson, V.J., 2004. A comparison of true and premodulated interferential currents. Arch Phys Med Rehabil 85 (3), 409–415.

Palmer, S., Martin, D., 2002. Interferential Current for Pain Control. Electrotherapy: Evidence Based Practice. Elsevier, Oxford.

Palmer, S., Martin, D., 2008. Interferential Current. In: Watson, T. (Ed.), Electrotherapy: Evidence Based Practice. Elsevier/Churchill Livingstone, Edinburgh.

Palmer, S.T., Martin, D.J., Steedman, W.M., et al., 1999. Alteration of interferential current and transcutaneous electrical nerve stimulation frequency: effects on nerve excitation. Arch Phys Med Rehabil 80 (9), 1065–1071.

Pereira, A.N., Eduardo Cde, P., Matson, E., et al., 2002. Effect of low-power laser irradiation on cell growth and procollagen synthesis of cultured fibroblasts. Lasers Surg Med 31 (4), 263–267.

Poltawski, L., Watson, T., 2007. Relative transmissivity of ultrasound coupling agents commonly used by therapists in the UK. Ultrasound Med Biol 33 (1), 120–128.

Poltawski, L., Watson, T., 2009. Bioelectricity and microcurrent therapy for tissue healing – a narrative review. Phys Ther Rev 14 (2), 104–114.

Pope, G.D., Mockett, S.P., Wright, J.P., 1995. A survey of electrotherapeutic modalities: ownership and use in the NHS in England. Physiotherapy 81 (2), 82–91.

Pounder, N.M., Harrison, A.J., 2008. Low intensity pulsed ultrasound for fracture healing: a review of the clinical evidence and the associated biological mechanism of action. Ultrasonics 48 (4), 330–338.

Ramirez, A., Schwane, J.A., McFarland, C., et al., 1997. The effect of ultrasound on collagen synthesis and fibroblast proliferation in vitro. Med Sci Sports Exerc 29, 326–332.

Reher, P., Harris, M., Whiteman, M., et al., 2002. Ultrasound stimulates nitric oxide and prostaglandin E2 production by human osteoblasts. Bone 31 (1), 236–241.

Robertson, V.J., 2002. Dosage and treatment response in randomized clinical trials of therapeutic ultrasound. Phys Ther Sport 3 (3), 124–133.

Robertson, V.J., Ward, A., Low, J., et al., 2006. Electrotherapy Explained: Principles and Practice. Elsevier, Oxford.

Romano, C.L., Romano, D., Logoluso, N., 2009. Low-intensity pulsed ultrasound for the treatment of bone delayed union or nonunion: a review. Ultrasound Med Biol 35 (4), 529–536.

Rompe, J.D., Maffulli, N., 2007. Repetitive shock wave therapy for lateral elbow tendinopathy (tennis elbow): a systematic and qualitative analysis. Br Med Bull 83, 1–24.

Rompe, J.D., Furia, J., Maffulli, N., 2008. Eccentric loading compared with shock wave treatment for chronic insertional achilles tendinopathy. A randomized, controlled trial. J Bone Joint Surg Am 90 (1), 52–61.

Rompe, J.D.,. Furia, J., Maffulli, N., 2009. Eccentric loading versus eccentric loading plus shock-wave treatment for midportion achilles tendinopathy: a randomized controlled trial. Am J Sports Med 37 (3), 463–470.

Rutjes, A.W., Nuesch, E., Sterchi, R., et al., 2009. Transcutaneous electrostimulation for osteoarthritis of the knee. Cochrane Database Syst Rev (4), CD002823.

Rutten, S., Nolte, P.A., Guit, G.L., et al., 2007. Use of low-intensity pulsed ultrasound for posttraumatic nonunions of the tibia: a review of patients treated in the Netherlands. J Trauma 62 (4), 902–908.

Sakurakichi, K., Tsuchiya, H., Uehara, K., et al., 2004. Effects of timing of low-intensity pulsed ultrasound on distraction osteogenesis. J Orthop Res 22, 395–403.

Seiger, C., Draper, D.O., 2006. Use of pulsed shortwave diathermy and joint mobilization to increase ankle range of motion in the presence of surgical implanted metal: A case series. J Orthop Sports Phys Ther 36 (9), 669–677.

Shah, S., Farrow, A., Esnouf, A., 2007. Availability and use of electrotherapy devices: A survey. Int J Ther Rehabil 14 (6), 260–264.

Shanahan, C., Ward, A.R., Robertson, V.J., 2006. Comparison of the analgesic efficacy of interferential therapy and transcutaneous electrical nerve stimulation. Physiotherapy 92 (4), 247–253.

Sheffler, L.R., Hennessey, M.T., Naples, G.G., et al., 2006. Peroneal nerve stimulation versus an ankle foot orthosis for correction of footdrop in stroke: impact on functional ambulation. Neurorehabil Neural Repair 20 (3), 355–360.

Simonis, R.B., Parnell, E.J., Ray, P.S., et al., 2003. Electrical treatment of tibial non-union: a prospective, randomised, double-blind trial. Injury 34 (5), 357–362.

Sluka, K.A., Walsh, D., 2003. Transcutaneous electrical nerve stimulation: basic science mechanisms and clinical effectiveness. J Pain 4 (3), 109–121.

Sluka, K.A., Lisi, T.L., Westlund, K.N., 2006. Increased release of serotonin in the spinal cord during low, but not high, frequency transcutaneous electric nerve stimulation in rats with joint inflammation. Arch Phys Med Rehabil 87 (8), 1137–1140.

Sontag, W., 2000. Modulation of cytokine production by interferential current in differentiated HL-60 cells. Bioelectromagnetics 21 (3), 238–244.

Sparrow, K.J., Finucane, S.D., Owen, J.R., et al., 2005. The effects of low-intensity ultrasound on medial collateral ligament healing in the rabbit model. Am J Sports Med 33 (7), 1048–1056.

Speed, C.A., 2004. Extracorporeal shock-wave therapy in the management of chronic soft-tissue conditions. J Bone Joint Surg 86 (2), 165–171.

Stevens, J.E., Mizner, R.L., Snyder-Mackler, L., 2004. Neuromuscular electrical stimulation for quadriceps muscle strengthening after bilateral total knee arthroplasty: a case series. J Orthop Sports Phys Ther 34 (1), 21–29.

Talbot, L.A., Gaines, J.M., Ling, S.M., et al., 2003. A home-based protocol of electrical muscle stimulation for quadriceps muscle strength in older adults with osteoarthritis of the knee. J Rheumatol 30 (7), 1571–1578.

Tanzer, M., Kantor, S., Bobyn, J.D., 2001. Enhancement of bone growth into porous intramedullary implant using non-invasive low intensity ultrasound. J Orthop Res 19 (2), 195–199.

Taylor, P., Burridge, J., Dunkerley, A., et al., 1999. Clinical audit of 5 years provision of the Odstock dropped foot stimulator. Artif Organs 23 (5), 440–442.

ter Haar, G., 1999. Therapeutic ultrasound. Eur J Ultrasound 9, 3–9.

Thiel, M., 2001. Application of shock waves in medicine. Clin Orthop Relat Res 387, 18–21.

Tis, J.E., Meffert, R.H., Inoue, N., et al., 2002. The effect of low intensity pulsed ultrasound applied to rabbit tibiae during the consolidation phase of distraction osteogenesis. J Orthop Res 20 (4), 793–800.

Tsai, W.C., Tang, F.T., Hsu, C.C., et al., 2004. Ibuprofen inhibition of tendon cell proliferation and upregulation of the cyclin kinase inhibitor p21CIP1. J Orthop Res 22 (3), 586–591.

Tsunoda, M., 2003. Treatment of non-union and delayed-union by low intensity pulsed ultrasound. Clin Calcium 13 (10), 1293–1296.

Tuner, J., Hode, L., 2004. The Laser Therapy Handbook. Prima Books, Grangesberg.

Turner, S., Powell, E., Ng, C., 1989. The effect of ultrasound on the healing of repaired cockerel tendon: is collagen cross-linkage a factor? J Hand Surg 14B, 428–433.

Usuba, M., Miyanaga, Y., Miyakawa, S., et al., 2006. Effect of heat in increasing the range of knee motion after the development of a joint contracture: an experiment with an animal model. Arch Phys Med Rehabil 87 (2), 247–253.

van Leeuwen, M.T., Zwerver, J., van den Akker-Scheek, I., 2009. Extracorporeal shockwave therapy for patellar tendinopathy: a review of the literature. Br J Sports Med 43 (3), 163–168.

Vinck, E.M., Cagnie, B.J., Cornelissen, M.J., et al., 2003. Increased fibroblast proliferation induced by light emitting diode and low power laser irradiation. Lasers Med Sci 18 (2), 95–99.

Vinck, E., Coorevits, P., Cagnie, B., et al., 2005. Evidence of changes in sural nerve conduction mediated by light emitting diode irradiation. Lasers Med Sci 20 (1), 35–40.

Vivodtzev, I., Pepin, J.L., Vottero, G., et al., 2006. Improvement in quadriceps strength and dyspnea in daily tasks after 1 month of electrical stimulation in severely deconditioned and malnourished COPD. Chest 129 (6), 1540–1548.

Walker, U.A., Uhl, M., Weiner, S.M., et al., 2006. Analgesic and disease modifying effects of interferential current in psoriatic arthritis. Rheumatol Int 26 (10), 904–907.

Walsh, D., 1997. TENS: Clinical Applications and Related Theory. Churchill Livingstone, Edinburgh.

Walsh, D.M., Howe, T.E., Johnson, M.I., et al., 2009. Transcutaneous electrical nerve stimulation for acute pain. Cochrane Database Syst Rev (2), CD006142.

Wang, E.D., 1998. Tendon repair. J Hand Ther 11 (2), 105–110.

Ward, A.R., 2009. Electrical stimulation using kilohertz-frequency alternating current. Phys Ther 89 (2), 181–190.

Warden, S., Bennell, K., McMeeken, J.M., et al., 1999. Can conventional therapeutic ultrasound units be used to accelerate fracture repair? Phys Ther Rev 4, 117–126.

Warden, S.J., Bennell, K.L., McMeeken, J.M., et al., 2000. Acceleration of fresh fracture repair using the sonic accelerated fracture healing system (SAFHS): a review. Calcif Tissue Int 66 (2), 157–163.

Warden, S.J., Favaloro, J.M., Bennell, K.L., et al., 2001. Low-intensity pulsed ultrasound stimulates a bone-forming response in UMR-106 cells. Biochem Biophys Res Commun 286 (3), 443–450.

Watson, T., 1996. Electrical stimulation for wound healing. Phys Ther Rev 1 (2), 89–103.

Watson, T., 2000. The role of electrotherapy in contemporary physiotherapy practice. Man Ther 5 (3), 132–141.

Watson, T., 2002. Ultrasound dose calculations. In Touch 101, 14–17.

Watson, T., 2003. Soft tissue healing. In Touch 104, 2–9.

Watson, T., 2006a. Electrotherapy and tissue repair. Sportex Med 29, 7–13.

Watson, T., 2006b. Tissue repair: The current state of the art. Sportex Med 28, 8–12.

Watson, T., 2008a. Electrical stimulation for enhanced wound healing. In: Watson, T. (Ed.), Electrotherapy: Evidence Based Practice. Churchill Livingstone/Elsevier, Edinburgh, pp. 329–346.

Watson, T. (Ed.), 2008b. Electrotherapy: Evidence Based Practice. Churchill Livingstone/Elsevier, Edinburgh.

Watson, T., 2008c. Ultrasound in contemporary physiotherapy practice. Ultrasonics 48, 321–329.

Watson, T., 2010. Narrative review: Key concepts with electrophysical agents. Phys Ther Rev 15 (4), 351–359.

Watson, T., Young, S., 2008. Therapeutic Ultrasound. In: Watson, T. (Ed.), Electrotherapy: Evidence Based Practice. Churchill Livingstone/Elsevier, Edinburgh.

Wilbert, D.M., 2002. A comparative review of extracorporeal shock wave generation. BJU Int 90, 507–511.

Wilkin, L.D., Merrick, M.A., Kirby, T.E., et al., 2004. Influence of therapeutic ultrasound on skeletal muscle regeneration following blunt contusion. Int J Sports Med 25 (1), 73–77.

Williams, A., McHale, J., Bowditch, M., et al., 1987. Effects of MHz ultrasound on electrical pain threshold perception in humans. Ultrasound Med Biol 13, 249–258.

Yeung, C.K., Guo, X., Ng, Y.F., 2006. Pulsed ultrasound treatment accelerates the repair of Achilles tendon rupture in rats. J Orthop Res 24 (2), 193–201.

Young, S.R., Dyson, M., 1990a. The effect of therapeutic ultrasound on angiogenesis. Ultrasound Med Biol 16 (3), 261–269.

Young, S.R., Dyson, M., 1990b. Effect of therapeutic ultrasound on the healing of full-thickness excised skin lesions. Ultrasonics 28 (3), 175–180.

Zanotti, E., Felicetti, G., Maini, M., et al., 2003. Peripheral muscle strength training in bed-bound patients with COPD receiving mechanical ventilation: effect of electrical stimulation. Chest 124 (1), 292–296.

Chapter 20

Physiotherapy for people with major amputation

Carolyn A. Hale

INTRODUCTION

Rehabilitation following amputation is the responsibility of the multi-disciplinary team (MDT), with the patient focussed in the centre. Working with a specialist team will produce the best outcome for the individual who has undergone life-changing amputation surgery (Pernot 1997). The physiotherapist is a key member of this team, involved at all stages of the process from the pre-operative phase, through amputation and discharge home, to prosthetic training and during life thereafter.

This chapter provides an overview of the physiotherapy management of people with amputation, outlining the role and importance of the physiotherapist's intervention. Readers will need to refer to the appropriate texts for details of specific treatment modalities and knowledge of causative factors, surgical techniques, prosthetic componentry and associated equipment. A list of reading material and resources will help you source this information, as will many of the other chapters in this book. This chapter will predominantly address the management of adult lower limb amputation.

Amputation is performed for life threatening disease, pain or unsalvageable injury, where all other treatments have failed. The goal is to preserve life and improve function and general health. Having a limb amputated is a life-changing event with potentially devastating consequences to every aspect of living, not just physical functioning. Becoming disabled in this way can affect a person's personality: affecting body image and relationships, and sometimes threatening the family unit. If amputation occurs when someone is of a young age it can affect earning potential and ambition, and, in most cases, can result in the person having to move home to more suitable accommodation. For many, meeting these challenges is the incentive that drives the determination to overcome their physical disability to achieve a good quality of life.

CAUSES AND LEVELS OF AMPUTATION

According to the Limbless Statistics (currently replacing the National Amputee Statistical Database, or NASDAB, and managed by UNIPOD), over 72% of lower limb amputations performed in the UK are as a result of vascular deficiencies, such as peripheral arterial disease and/or diabetes (Van de Ven and Engstrom 1999), whereby 50% of these people have diabetes. In fact, this is the case for the majority of lower limb amputations performed in the Western world (Ebskov 1999). Most patients in this category will be over the age of 65 years and may have other comorbidities associated with the ageing process and vascular dysfunction, such as arthritis and cardiac disease (Fyfe 1992, Ham and McCreadie 1992). As most vascular pathologies are progressive in nature, the patient may have undergone earlier intervention in the form of arterial bypass operations (to relieve blockages and narrowing in arteries) or toe amputations (as a result of tissue death) prior to a major amputation. When planning a treatment regime, these concurrent pathologies and history must be accounted for.

The next most common cause of amputation is trauma, where an individual has, perhaps, been involved in a road traffic accident or industrial incident. Amputation may be the only option if the injury to bone, soft tissue, blood vessels and/or nerves cannot be treated and made viable. Amputation may be at the time of the accident or, in some cases, days, months or even years afterwards, when the injured part becomes a burden.

Trauma is the major cause of upper limb amputation, which is often a result of a work-related accident. This population of patients are usually therefore young and of working age. Overall, however, people with amputation owing to trauma remain a very small population in comparison with those with vascular issues.

Table 20.1 Causes of amputation in the developed world (Van De Ven 1999)

Developed world cause	Relative percentage (%)
Lower limb	
Peripheral arterial disease (25–50% of which also have diabetes mellitus)	85–90
Trauma	9
Tumour	4
Congenital deficiency	3
Infection	1
Upper limb	
Trauma	29
Disease	30
Congenital deficiency	15
Tumour	26

Table 20.2 Levels of amputation (Van de Ven 1999)

Lower limb	Upper limb
Hemipelvectomy	Forequarter
Hip disarticulation	Shoulder disarticulation
Transfemoral*	Transhumeral*
Supracondylar, transcondylar and Gritti-Stokes	Elbow disarticulation
Knee disarticulation	Transradial*
Transtibial*	Wrist disarticulation
Symes	Transmetacarpal
Chopart/Lisfranc	Interphalangeal
Transmetatarsal	
Interphalangeal	

*Denotes the most commonly seen levels in clinical practice.

Key point

The MDT consists of surgeons, nurses, physiotherapists, occupational therapists, counsellors and psychologists, prosthetists and orthotists, wheelchair therapists, rehabilitation doctors, podiatrists and chiropodists, social workers and homecare agencies. Specialist regional units, sometimes known as Disablement Services Centres (DSCs) are the usual hub of management. Services are provided by primary, secondary and tertiary establishments, and the patient will meet numerous clinicians throughout their rehabilitation. The physiotherapist has a key role, often providing the constant link between all the professionals.

Historically, the field of prosthetic limbs develops significantly at times of war owing to the increased demand for such services by the young war-injured. Injuries caused by bomb blasts are particularly challenging for the MDT owing to the extensive nature of injury.

Other reasons for major amputation of an arm or leg are tumours, infection and congenital deficiencies. Children born with limb absences or deformity do not always have an amputation; however, their limb dysfunction is often managed like an amputation. In general, it is fair to say that people born with full or partial limb absence cope better mentally and physically with their impairment than someone with acquired limb amputation – particularly those with upper limb involvement who tend to be less reliant on a prosthesis.

The decision regarding amputation level is determined by the need to remove all non-viable tissue while creating a healed, pain-free, functional and potentially prosthetically suitable residuum (stump), also called residual limb. Incidentally, many patients do not like their leg being referred to as a 'stump' and appropriate references should be found. Where possible, when an amputation is a planned event, the physiotherapist should be involved in the amputation level decision as their assessment findings can predict postoperative functional ability and therefore the likely rehabilitation outcome. Tables 20.1 and 20.2 list the causes of amputation seen in the developed world and the recognised levels of surgical amputation.

THE PSYCHOSOCIAL IMPACT OF AMPUTATION

The physiotherapist, as for all team members, must have an understanding of the psychological implications associated with having a limb amputated. This will help in building a rapport with the patient, aiding agreed goal-planning and facilitating a motivating rehabilitation regime for the individual.

Loss of a body part and consequent change to body image can potentially lead to loss of confidence; loss of function; loss of a lifestyle; loss of role, income and status; and loss of independence and control. Having an amputation can result in people feeling vulnerable, worthless and isolated. People will be affected by each of these aspects to differing degrees and their ability to accept their new situation will also vary greatly. The normal reactions to grief and bereavement are well documented (Kubler-Ross 1969; Parkes 1972, 1975; Campling 1981); for some patients the reaction is transient and

minor, while for others it is profound, disabling and longer lasting (Bradway et al. 1984; Butler et al. 1992; Krueger 1984).

Key point

Models of grief and bereavement offer frameworks for understanding grief associated with loss. It must be recognised that grief affects people in different ways and does not always manifest itself in a set pattern to a set timeframe. The physiotherapist must be sensitive to patients' emotional needs at all stages of the rehabilitation process.

Anyone who has an amputation needs to be given time to adjust. They need to be given accurate information about their rehabilitation programme and realistic ideas of what they can expect. Amputation affects the whole family and loved ones should be included in any rehabilitation process (Caron 1989). A successful outcome in restoring independence and self-worth is dependent on adjustment and acceptance by the individual and their close support network. People with disability often lack choice and other people make decisions on their behalf (O'Shea and Kennelly 1996). This has the impact of denying them a role in society. The physiotherapist must take time to talk with their patient, to understand their fears and their hopes, to recognise barriers to progress and work together to set goals. Recovering after an amputation is not just about functional recovery, for example being able to 'walk' or to make a cup of tea.

PAIN AND PAIN RELIEF

There are three potential types of pain following amputation.

Residual limb pain

Amputation surgery creates tissue disruption and trauma. This produces a natural inflammatory response resulting in oedema (swelling). Refer to Chapter 12 for details. The oedema results in pressure on already injured nerve endings, causing pain. This pain is usually managed postoperatively with analgesics and possibly epidurals. Managing the oedema itself will also help with pain relief. The physiotherapist can use limb elevation, elastic compression such as Tubifast, intermittent compression and exercise to improve the circulation thereby promoting the healing process, reducing swelling and thus pain (see Figure 20.1). Normal primary healing takes around three weeks. Any delays in healing can result in greater scar tissue which can become adherent to the underlying bone and therefore be painful on skin movement, especially when under pressure. Massage and ultrasound can be helpful at relieving this type of scar pain.

Figure 20.1 Elevation with a wheelchair stump board and compression garment.

Following amputation, when a cut nerve heals there is a growth of nerve cells at the distal end. As the dissected nerve has no neural sheath to grow into it forms a bundle called a neuroma. The surgeon endeavours to bury major nerve endings within soft tissue, as they can be very sensitive. If the neuroma is superficial, direct pressure from wearing a prosthesis can create pain. Sometimes ultrasound therapy can ease symptoms.

Later on, use of a prosthesis can cause pain in the residual limb owing to sheer friction or excessive pressure. This can damage skin, which can result in bruising, skin breakdown, infected hair follicles and blisters. The physiotherapist must ensure that the user has put it on correctly and ensure a correct fit. Liaison with the prosthetist is essential (see the section related to prostheses further on in the chapter). Skin hygiene and skin care is also very important.

Phantom limb pain and sensations

Phantom limb is described as sensation experienced in the missing limb part (phantom limb sensation (PLS)) and it can, in many cases, be experienced as pain (phantom limb pain (PLP)). It is well documented and symptoms are well recognised, if not poorly understood (Fraser et al. 2001). It is a feature that can impact significantly on the life of a patient (Hill et al 1995; Weiss and Lindell 1996; Williams and Deaton 1997). Experiencing a phantom limb can be alarming to people and they need to be reassured that this is normal, as 70% of people with amputation experience PLP (Butler and Moseley 2008).

There are two reasons why someone may still perceive the amputated body part. Firstly, the brain has an area of tissue dedicated to that body part (the homunculus map) which expects sensory information. This area of the brain is obviously not removed during limb amputation and thus still tries to process information. As a result, it can acknowledge sensory input from the adjacent brain tissue. When the sensory feedback is painful, this can be more troublesome for the individual as it is perceived as real pain.

A second reason to perceive pain is owing to the surgical cut of the nerve causing damage and inflammation. In addition, at the time of injury or disease, the nerve tissue may have been crushed or starved of a vascular supply, also resulting in painful symptoms.

Types of phantom pain described are: 'burning', 'electric', 'shooting', 'twisting', 'cramping', 'crushing' and 'sharp'. PLP can be intermittent or constant, and can be felt in any part of the removed limb. This can take a long time to settle down and in a few cases never resolves. It can seem worse when the individual is stressed or unwell, throughout a lifetime. It is important that the physiotherapist assesses pain carefully to determine its cause and allay patient fears that something is wrong. Effective pain relieving modalities for phantom include: transcutaneous electrical nerve stimulation (TENS), acupuncture, relaxation, massage, exercise, compression and analgesia. Chapter 17 ('Pain') will be useful to consult. Alternative methods can include reflexology, counselling and hypnotherapy.

Key point

Phantom pains are often exacerbated by stressful life events, such as illness, changing house or job, or divorce and bereavement.

PLP should not be confused with PLS, which are sensations in the missing limb that are not painful. These are often sensations of the limb as it was before amputation, often in normal orientation, but sometimes in a strange position. People have described such sensations as 'foot facing backwards', 'tight shoes', 'itching' and 'pins and needles,' as well as feeling that their hand or foot is now at the end of the residual limb, known as the telescoping effect. These sensations can be equally distressing and as distracting as phantom pain.

Secondary pain

Another significant area of pain can be the secondary pain caused by the stress to the remaining musculoskeletal system, owing to the asymmetry of the posture caused by amputation and the use of a prosthesis. It is well known that lower limb prosthetic users are prone to early onset osteoarthritis, particularly in the lower back and in the hip and knee on the remaining side (Ehde et al. 2001; Norvell et al. 2005) Your skills in the assessment and management of musculoskeletal problems will be vital.

THE ROLE OF THE PHYSIOTHERAPIST FOLLOWING LOWER LIMB AMPUTATION

Physiotherapy involves the continuous assessment of patients' goals, needs and abilities in order to set realistic and agreed treatment plans. The physiotherapist will rely on all their skills for treating a number of conditions.

The physiotherapist must re-educate movement patterns to optimise independent function for activities of daily life such as self-care, wheelchair and prosthetic use, and normal occupation, while managing all influencing factors.

Physiotherapy aims

Rehabilitation following lower limb amputation is a continual process, involving all aspects of a person's life: physical, mental, emotional and socioeconomic. For a successful physiotherapeutic outcome all these aspects must be addressed.

The final goal should be optimal independence, with or without a prosthesis, to return to as normal a life as possible. Ideally, the person should achieve:

- independent self-caring;
- independent indoor mobility;
- independent outdoor mobility;
- being able to get in/out of a car or other transport;
- a return to leisure/hobbies/work/society.

Considerations

The following are reasons why some patients have difficulties achieving goals:

- poor residual limb condition, e.g. adherent scar tissue, unhealed, bulbous shape, failed myodesis, neuroma, bony prominences, pain, hypersensitivity, poor vascularity, short leverage, skin frailty;
- concurrent pathologies leading to an inability to learn, reduced range of motion, reduced strength and stamina, pain, poor balance, poor dexterity, socket intolerance;
- social and environmental difficulties, e.g. living alone, unsuitable accommodation for wheelchairs or prostheses, poor access to accommodation, unhealthy lifestyle, dominant carers;
- lack of motivation – fear, fatigue, emotional barriers to achieving success;
- inappropriate equipment, e.g. poor prosthetic socket fit or alignment, a too big or too small wheelchair, incorrect prescriptions;
- lack of specialist rehabilitation services.

The inability to learn new skills is probably the largest determining factor to successful outcome. Sometimes this can be influenced by physiotherapy intervention but not in every case.

Patients must be involved in co-ordinating all aspects of their treatment-planning, goal-setting and monitoring as self-responsibility and self-management are the foundations of rehabilitation (Watson 1996).

PHYSIOTHERAPY ASSESSMENT

Assessment

A thorough subjective and objective assessment will ensure accurate and realistic goal planning. Table 20.3 outlines the content of both a subjective and objective

Table 20.3 Recommended content of assessment following amputation

Subjective assessment	
Present complaint	Level of amputation and cause, time since amputation. Current symptoms
History of present complaint	Ulcers, gangrene, bypass surgery, intermittent claudication, rest pain, sympathectomy, embolectomy, heparinisation, stages of amputation. Accident details and orthopaedic history, salvage operations, oncology treatment
Past medical history	Related: diabetes, myocardial infarction, cerebrovascular accident, angina, renal status, eyesight and neuropathy, concurrent injuries Other: respiratory disorders, osteoarthritis, rheumatoid arthritis, major surgery, old injuries, hearing and sight, depression, epilepsy, low back or peripheral joint pain
Social history	Home environment, cohabiters, family support and dependants, home care package, district nurse, access (ramps), doorways, bathroom upstairs, rails/stair lift, employment, school and education, hobbies, finances, compensation claim status (related to trauma), driving, smoking and lifestyle, attitude to exercise and fitness
Drug history	Drugs that may affect rehabilitation programme
Mobility	Pre-amputation – distance covered, walking aids used, stairs, outdoors, wheelchair, exercise and sports, limiting factors Post-amputation – as above plus transfers and bed mobility Type of wheelchair, walking aids and prosthesis
Function	Self-care, domestic tasks, shopping, laundry, carrying objects, picking up objects, stairs, steps/kerbs, slopes, energy expenditure, limiting factors, compensations, safety, falls, aids used
Prosthetic rehabilitation history	Current and previous prostheses: prescription, delivery date, socket fit, ability to don/doff, maintenance, pattern of use (how long, daily, how far) and reasons for no prosthesis. Current and previous physiotherapy intervention
Psychological status	Attitudes, emotions, depression, goals and aspirations
Objective assessment	
Residual limb	Wound, scar, healing status, dressings, oedema, pain (visual analogue scale 0–10*), sensation, colour, temperature, compression therapy, reflexes, joint range, contractures, weakness, strength, co-ordination
Remaining limb	Vascularity, strength, joint range, scars, wounds, risk level, temperature, ulcers, footwear, colour, pulse, pain, oedema, numbness, joint dysfunction and adaptations, sensation, reflexes, motor control
Trunk and balance	Sitting, standing, pain, trunk range and control of movement, alignment, posture and core stability, abdominal strength, compensation reactions (see Figure 20.2 – postural alignment changes following amputation)
Gait/function with prosthesis	Gait pattern, deviations and possible causes, speed, indoor and outdoor use, varying surfaces, exercise tolerance

*0 denotes no pain and 10 denotes worst pain imaginable.

assessment. It is important during the assessment that the physiotherapist gains an understanding of what the patient has been through, their current situation and their goals for the future. This needs to be in terms of their physical and psychosocial well-being. The objective assessment needs to carefully assess any musculoskeletal or neurological dysfunction and current movement control.

Following amputation, the skeletal system makes compensations for the imbalance caused by the missing anatomy or the restrictions caused by the prosthesis. Joint alignment and soft tissues adapt to new prolonged postures. Muscles can start to work inefficiently and in an uncoordinated manner, affecting a person's ability to move safely (Comerford et al. 2005). See Figure 20.2 to see the likely postural shift in someone with a lower limb amputation. The head centralises over the remaining heel and the foot has rotated outwards for increased stability. In some cases, such postural changes can result in pain. The altered biomechanics can result in increased energy expenditure and loss of confidence when moving. Restoring midline alignment aids the most efficient muscle recruitment.

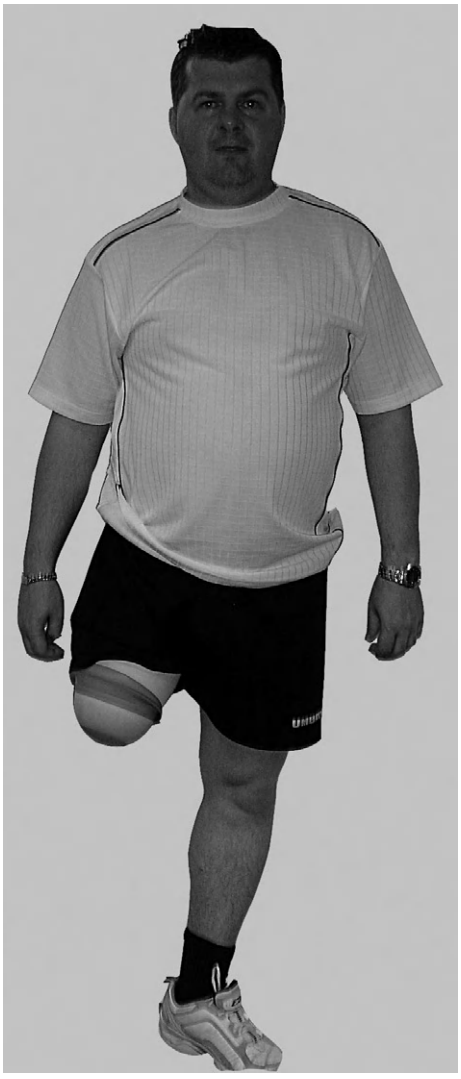


Figure 20.2 Common postural changes following amputation.

Problem list

Based on findings from assessment, the problem list is formulated with agreed and realistic goals, for both the short and the long term.

Treatment plan

A treatment plan is drawn up in order to achieve the desired goals based on the assessment findings.

> **Key point**
>
> Frequent reassessment will guide any revision of goals. Physiotherapy treatment is concerned with teaching people new skills. It is a partnership, not related to 'doing to the patient' but related to the patient learning to do things for themselves.

STAGES OF PHYSIOTHERAPY MANAGEMENT

The physiotherapist acts as part of the MDT and is involved at all stages of the amputation management, as summarised in Table 20.4. It is important that the physiotherapist liaises with all members of the team at the different stages of the rehabilitation process in order to gain best outcome for their patient and have common agreed goals.

Pre-operative stage

In cases of planned surgery it should always be possible for the physiotherapist to assess the patient before their amputation. The physiotherapy assessment should be holistic, assessing sensory and motor performance, including respiratory status and function, while addressing current and pre-morbid levels of independence and psychological well-being.

As part of the MDT, the physiotherapist should be involved in the decision as to what level to amputate and in the preparation of the patient for surgery (Cutson and Bougiorni 1996). Exercises and wheelchair or crutch use can be taught at this stage if the patient is not in too

Table 20.4 Summary of stages of physiotherapy management

Stage of management	Aspects
Pre-operative	Physical, functional and psychological assessment prior to surgery to inform decision of level to amputate Provision of pre-operative exercises in preparation for postoperative period Provision of information about rehabilitation process Wheelchair and mobility practice
Postoperative	Physical and functional assessment Address pain and oedema control Practise transfers and independent function Practise use of walking aids and wheelchairs Promote wound healing, pressure care and good nutritional intake Provision of exercises for strengthening, stretching and balance in order to aid independent mobility Provide education and information for patient and carers Liaise with MDT within the hospital and community Refer to appropriate agencies for discharge
Pre-prosthetic	Prosthetic preparation: compression and shaping of the residual limb using elasticated stocking, elevation, exercise and early walking aids Improve cardiovascular fitness Provide education and information for patient and carers Liaise with MDT within the hospital and community Refer to appropriate agencies for prosthetic provision
Prosthetic	Physical, psychological and functional assessment Provision of exercises for strengthening, stretching, stability and balance Gait re-education to minimise gait deviations, achieve safe mobility and promote energy efficient function Continue pain control measures Progress walking aids and skills with the prosthesis Practise activities of daily living (dressing, cooking, walking outdoors, shopping) as part of MDT Information regarding care of the residual and remaining limbs and prosthesis
Lifelong	Maintenance exercises Further intervention when the situation changes Review appointments to reassess Liaise with community agencies Facilitate reintegration to normal living Act as long-term resource for patients, families and carers

much pain. As the quality of the amputation surgery can influence final rehabilitation outcome, the surgeon is an integral member of the team.

Both the patient and their family will benefit from information about the rehabilitation process and what to expect (Yetzer et al. 1994). At this time it is helpful to spend time with the patient, reassuring them and building a rapport. This will be advantageous in the postoperative stage.

Postoperative stage

Once the person has had their amputation, they are usually brought back to the main ward. Only if there are comorbidities or complications are they transferred to high-dependency or intensive care units.

Ongoing physiotherapy assessment needs to take place at all stages. At many operating hospitals a care pathway is in place which will outline how the immediate postoperative phase should be managed. The physiotherapist can be guided by this, applying it to the individual patient's needs. An example of such a pathway can be found in Table 20.5.

Early mobility is important to prevent weakening, stiffness, loss of balance and confidence, especially in the elderly. It is common practice to get out of bed on day two after amputation. This immediate re-education of posture and balance provides a big psychological boost.

Table 20.5 Example care pathway for early rehabilitation following lower limb amputation

Day 1: In bed
Respiratory care, pain control exercises for bed mobility and joint range

Day 2: Sit out in chair
Range of movement and strength for all limbs and trunk
Balance training
Transfer practice and wheelchair provision
Oedema control

Day 3: Independence
Continue exercises for strength, range and balance
Pain and oedema control
Functional exercises, i.e. bridging and supported standing, wheelchair, personal care
Posture correction

Days 4–7: Discharge planning
Plan home visit with occupational therapist
Monitor oedema control
Continue exercise programme and mobility
Ensure appropriate clothing for rehabilitation

Days 7–14: Prosthetic assessment
Attend gym environment, individual and group exercise sessions
Assess and practise with early walking aid, i.e. pneumatic post-amputation mobility (PPAM) aid
Prepare for prosthetic rehabilitation: stump shape, balance, proprioception and strength
Refer to regional prosthetic centre
Discharge or transfer to rehabilitation ward or intermediate care

Days 14–28: Develop appropriate plan
Progress rehabilitation programme: prosthetic or non-prosthetic

Weeks 4–6: Outpatient or community
Cast for prosthesis
Start prosthetic training
Ensure reintegration into home environment

Controlling residual limb oedema is vital to postoperative progress. A reduction in swelling will reduce pain, improve compliance, aid healing and prevent delays to rehabilitation. Compression, exercise and positioning in elevation will aid in oedema control.

Key point

Residual limb shape is important for making a prosthetic socket and can be determined by using a Juzo compression garment (www.juzousa.com), which controls the oedema. The correct sizing of such a garment is essential and should be measured by a suitably qualified practitioner (Lambert and Johnson 1995).

In the early stage following amputation a person will be very keen to learn to be independent. Practising use of a wheelchair, and transferring to and from it safely are important activities. Most people with unilateral amputation will stand on their remaining limb to pivot into the chair positioned at 90 degrees, with or without the armrest being removed. Protective footwear is necessary for this transfer technique. Sliding boards are a useful tool where patients are too weak to lift their own body weight or where their remaining limb is not suitable. Figure 20.3 demonstrates a forwards/backwards transfer suitable for someone with bilateral lower limb amputations. Crutches or frames are suitable for those with excellent balance and upper limb strength, although this raises the risk of falls and residual limb oedema owing to the dependent position while mobilising.

As an integral member of the team and using a holistic approach the physiotherapist should also play their part in pressure-management of vulnerable tissues at this time of relative immobility and encourage adequate nutrition to aid postoperative recovery.

An essential role of the physiotherapist at this stage is to encourage normal movement patterns and prevent joint contracture. Reduced joint range can be painful, delaying rehabilitation and, in severe cases, can prevent prosthetic fitment. The physiotherapist educates the patient in exercises for joint stretching and posture care. The common contractures in lower limb amputation affect both the hip and knee joints and are associated with pain and prolonged sitting positions:

- *transtibial level* – contracture of the knee into a flexed position. Active knee extension should be encouraged, with passive stretching, and positioning provided by elevation on the bed or stump board, as shown in Figure 20.1;
- *transfemoral level* – contracture of the hip into a flexed position. There may also be a contracture into abduction, where the hip flexors and abductors are unopposed by weakened extensors and adductors. Active exercise of the weak muscles and positioning will help to address this imbalance.

Hip flexion contractures can also commonly occur in transtibial amputations. Figure 20.4 shows how a small stool can be used to strengthen groups of muscles, creating better balance between opposing muscles, therefore reducing the incidence of joint contractures.

Re-educating postural balance and control will contribute to safe mobility, whether with a wheelchair, crutches or prosthesis. A gym ball is a great adjunct for developing a person's core stability and control, as Figure 20.5 demonstrates.

Reassuring patients and repeating information will allay fears and help them to concentrate on their rehabilitation programme. Liaison with colleagues and discharge agencies is necessary to facilitate smooth progress. Continuing rehabilitation into the community will

Figure 20.3 A forwards/backwards transfer suitable for someone with bilateral lower limb amputations.

Figure 20.4 Combined muscle strengthening to control joint range.

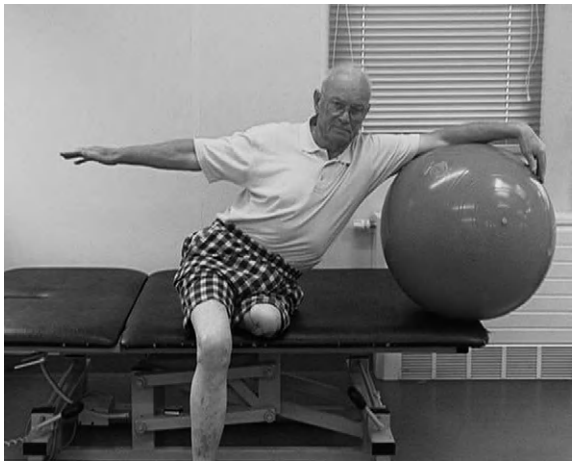


Figure 20.5 Use of a gym ball to aid trunk rehabilitation.

encourage adaptation to new skills of independence aided by the familiar surroundings (Park et al. 1994).

Pre-prosthetic stage

Once it has been decided that someone has the potential ability to use a prosthesis, the pre-prosthetic preparation should start. The person may still be an inpatient, at home following discharge or they may be at an intermediate care facility. It is worth mentioning that almost half of people undergoing amputation in the UK are not suitable for prosthetic prescription owing to comorbidities, such as respiratory or cardiac incapacity. Exercises should be progressed to challenge the posture and balance in a more upright position. Use of early walking aids, equipment that offers bipedal gait without a prosthesis, promotes oedema reduction and wound healing, and provides an insight for patients and therapists as to how well someone will progress. Over 80% of UK physiotherapy departments have early walking aids called pneumatic post-amputation mobility (PPAM) aids (www.ortho-europe.com; Lein 1992), whereby the patient's residual limb is surrounded by an inflatable bag within a metal frame by which they can carry some weight on the amputated side. The bags are inflated to 40 mmHg in a non-weight-bearing position, which is known to be a pressure that will not cause tissue damage when applied in a constant manner. Walking with a PPAM aid should be partial weight-bearing and always initiated within the parallel bars. In vascular cases this should be introduced at day 7–10 post-operation, and is suitable for transtibial, knee disarticulation and long transfemoral levels of amputation (see Figure 20.6). The physiotherapist should always follow the manufacturers' instructions and national guidelines (see *Further Reading*).

A second type of early walking aid is a Femurett (manufactured by Össur) which is designed for use with transfemoral amputations. Össur have produced a training guide and video in collaboration with the British Association of Chartered Physiotherapists in Amputee Rehabilitation (BACPAR). This type of walking aid can be used in full weight-bearing and is commonly found at the regional limb centres.

Prosthetic stage

Being able to use a prosthesis can allow a person to enjoy a greater quality of life, possibly being rid of years of pain and poor function from a diseased or injured limb, and being able to return to work and leisure activities. At this stage of rehabilitation an intensive gait re-education programme is necessary. Early prosthetic fitting and rehabilitation are vital to successful physical, emotional and psychological recovery in the short and long term (Bradway et al. 1984).

Using a prosthesis well is about learning a new motor skill, similar to learning to play the piano, play tennis or

Figure 20.6 Use of PPAM aid within parallel bars.

ride a bicycle. The prosthetic user needs to move with the equipment. It takes hours of practice to be, and remain, competent. Natural ability influences the final outcome to some degree; however, effective training by a physiotherapist makes a huge impact.

The safe environment of the parallel bars is provided initially for walking, progressing from here so as not to encourage dependence on the bars in terms of partial weight-bearing or fear of walking beyond this environment. Tension increases fatigue so patients are always advised what is happening and what is expected from them. All movement is observed carefully to assess what is not normal, why that may be and whether it can be improved.

An essential aspect to using a prosthesis is being able to fully weight-bear through the socket. In addition, the user must develop their proprioception to feel contact with the ground through the socket, in order to confidently move over the prosthetic foot. Appropriate motor output relies on sensory input. Learning to recognise stimuli at low thresholds forms effective proprioception, leading to normal recruitment of muscle activity and balance reactions. A comfortable socket is vital to achieving full loading and control. Exercises should be used to facilitate correct muscle recruitment, in particular to create pelvic excursion and to stabilise body segments, especially at the hip on the amputated side. Figure 20.7 shows how stepping onto a block with the remaining leg can encourage

Figure 20.7 Exercise for controlling weight-bearing and weight transference.

weight-bearing and weight transfer over the prosthesis, using hip abductors and extensors. Figure 20.8 shows how the physiotherapist can facilitate rotation of the pelvis around the longitudinal axis necessary for forward progression. Being able to stabilise the body while standing on the prosthesis is necessary also for safe, confident movement and can be encouraged using dynamic surfaces, such as a wobble cushion (see Figure 20.9). The Nintendo Wii Fit has become a useful adjunct to the physiotherapist's tool kit, as balance exercises can help someone to normalise their posture and control body movements through fun, visual feedback. Guidelines are available.

During prosthetic training, the physiotherapist is responsible for teaching the user how to care for their residual and remaining limbs. This is particularly important for people with diabetes, whose lack of foot sensation puts them at great risk of a second amputation. How to care for the prosthesis, understanding how the

Figure 20.8 Facilitation of pelvic rotation.

components function and how to don the socket properly is key to successful use. Prevention of pressure sores will ensure continued use of the prosthesis. The physiotherapist must also make their prosthetic rehabilitation programme functional and orientated to the individual's lifestyle. Where appropriate, getting on and off the floor, using transport and walking outdoors over uneven ground, slopes, stairs and kerbs should be included, as well as tasks around the home.

Key point

Specialist prosthetic training is essential to the success of the rehabilitation programme. Readers are directed to numerous texts containing specific knowledge of prosthetic products, the normal gait cycle and gait training techniques.

Figure 20.9 Hip stabilisation exercises.

For some people having a prosthesis can be more of a hindrance than a help. Learning to use a prosthesis requires strength, co-ordination, cardiovascular fitness, motivation and cognitive ability. Prosthetic abandonment is common. At long-term post-amputation evaluation approximately half of transtibial and less than a third of transfemoral amputees use their prosthesis full-time (De Luccia et al. 1992; Davies and Datta 2003). Wheelchair independence is a more appropriate option for many elderly amputees. Rehabilitation in a wheelchair must also address independence in activities of everyday living and social integration. There are many seated exercises, which can be effective and fun. Working on physical strength, agility and posture aids transferring skills and also prevents secondary pain.

Lifelong stage

The physiotherapist will be involved in the long-term management of a person with an amputation. Maintenance exercises should be prescribed and monitored to minimise secondary dysfunctions. Review appointments to assess movement patterns can promote independence and continued use of the prosthesis.

As people progress through life their needs will change, facilitating the further input of the physiotherapist. Examples of such events are when the prosthetic prescription changes, return to work and/or sport, issues related to the ageing process or change in health status and problems occurring as a result of postural compensations. People with lower limb amputation commonly develop a loss of control of hip and knee extension on the prosthetic side, poor control of trunk and hip rotation and hip abduction, and excessive use of spinal extension.

Regular reassessments will highlight when the physiotherapist needs to refer on to the other professionals, and a working link should be maintained with key MDT members.

Falls

Following amputation, people are at a greater risk of falling. It is reported that 54% of people with amputation fall each year (Miller et al. 2001). It is an important part of the physiotherapy management to address this. The risk factors of falling should be assessed for. These include vision, home hazards, confidence, walking aids, medication, alcohol intake, cardiovascular and neurological status, and the prosthesis. Managing falls in the older population is well documented and can be applied to this client group. Any treatment regime for people with amputation must include strengthening the anti-gravity muscles, balance training and how to get off the floor. For those who cannot get from the floor by themselves, they need to learn a strategy of what to do in the event of a fall. Any persistent unexplained falls must be investigated further. There is a BACPAR guideline to further inform you.

PROSTHESES

The prosthetist is primarily responsible for deciding on the type of prosthesis to be provided and its manufacture. The rehabilitation doctor, physiotherapist and occupational therapist are also key in deciding what functions are most appropriate and necessary for the potential user.

A prosthesis comprises: a bespoke socket, a form of suspension, joint/s and interjoint segments, a foot or hand/terminal device and a cosmetic cover. Examples of transtibial and transfemoral prostheses without cosmetic covers can be seen in Figures 20.10 and 20.11. The socket must contain the remaining musculoskeletal tissues (muscle, fascia and bone) and transmit the internal and external forces involved in movement to the ground. A comfortable and well-fitting socket is critical to a successful prosthesis. Weight should be loaded on weight-tolerant tissues and off-loaded in areas of pressure intolerance, such as bony structures and nerves. Peak pressures and friction will cause tissue damage and make the user reluctant to wear the prosthesis. Teaching the user to don the prosthesis correctly cannot be stressed enough.

The mechanics of the prosthesis can cause the user to move in a certain manner. This can be very disconcerting and it is the role of the physiotherapist to teach the user to control the prosthesis and not be controlled by it. The physiotherapist must be familiar with different facets of prosthetic components and their provision in order to teach best use. Close liaison with the prosthetist will ensure an optimal outcome.

Figure 20.10 Example of a transtibial prosthesis.

PROSTHETIC GAIT DEVIATIONS

A sound understanding of normal gait will allow the physiotherapist to analyse prosthetic gait and diagnose any deviations. Chapter 15 ('Biomechanics') will provide the required grounding. Prosthetic gait abnormalities are caused by the user's movement patterns or the prosthesis. Often, it is the combination of both. The most common gait deviations are described in brief below. This list is not exhaustive.

Transtibial level deviations

- Excessive knee flexion during stance owing to weak quadriceps and gluteals, fixed flexion at hip or knee, excessive dorsiflexion at prosthetic ankle and short prosthetic foot lever.
- Insufficient knee flexion during stance owing to poor hip and knee control, socket discomfort and excessive plantar flexion at prosthetic ankle.
- Lateral bending of the trunk to prosthetic side during stance owing to socket discomfort, lack of hip stability and a prosthesis being too short.

Figure 20.11 Example of a transfemoral prosthesis.

Transfemoral level deviations

- Abducted prosthesis, a wide base of support throughout stance and swing owing to abduction contracture, weak hip stabilisers, socket discomfort, a prosthesis being too long, insufficient lateral socket wall support and poor prosthetic knee control.
- Lateral bending of the trunk to prosthetic side during stance owing to abduction contracture, short residual limb, poor hip stability, insufficient lateral socket wall support and too short a prosthesis.
- Circumduction, abduction through prosthetic swing only owing to weak adductors, too long a prosthesis, inadequate suspension, unstable prosthetic knee and poor prosthetic knee control.
- Vaulting, rising into plantar flexion through stance on sound side owing to short residual limb, poor prosthetic swing control, a prosthesis being too long, inadequate suspension and too slow a gait.
- Excessive lumbar lordosis during prosthetic stance owing to hip flexion contracture, weak abdominals and hip extensors, unstable prosthetic knee and insufficient socket flexion.

General observed deviations are uneven step length, uneven timing and uneven arm swing. Using a prosthesis requires energy expenditure in excess of normal walking with two intact limbs. This can be very tiring. To compensate, users will walk more slowly (Waters and Mulroy 2004; Hagberg et al. 2007).

The physiotherapist plays a pivotal role in training and can influence the following causes of deviation: fear; weakness; poor range of movement; poor weight-bearing; pain; poor fit/donning; incorrect use; poor movement patterns from incorrect muscle recruitment; fatigue; inappropriate walking aids or their use; poor balance; change in footwear; and bad habit/technique.

Prosthetic length and alignment issues are best identified and resolved with the physiotherapist and prosthetist working together as a team. With respect to energy levels and proprioception, it is far easier to rehabilitate with a transtibial level amputation than a transfemoral level as greater mobility can be achieved.

OUTCOME MEASURES

A physiotherapist should always have a benchmark by which to measure whether the physiotherapy intervention has been successful or not. This can be levels of pain, range of joint motion or levels of function. Rehabilitation after amputation is complex and multifaceted and measuring the outcome can be problematic. There are a number of outcome tools for disability that can be applied to amputees. Described here are the tools validated specifically for prosthetic rehabilitation.

- *SIGAM Algorithm* (based on Harold Wood scale) offers a simple, valid and reliable means of measuring mobility in lower limb amputees. SIGAM can be used throughout the stages of rehabilitation to identify changes in mobility, making it useful for both new and established amputees (Ryall et al. 2003).
- *Locomotor Capabilities Index* (LCI) was designed to trace a comprehensive profile of locomotor capabilities of the lower limb amputee with the prosthesis and to evaluate the level of independence while performing these activities. The LCI is part of the prosthetic profile of the amputee (PPA) (Grisé and Gauthier-Gagnon 1993; Gauthier-Gagnon and Grisé 1998).

- *Trinity Amputee Prosthetic Evaluation Scale* (TAPES) is a questionnaire designed to investigate different aspects of having an artificial limb (Gallagher and MacLachlan 2004).
- *Functional Measure of the Amputee* (FMA) is a questionnaire that was developed from the PPA questionnaire, to collect long-term functional and prosthetic use information following discharge. It contains the LCI (Callaghan et al. 2002).
- *Activities-Specific Balance Confidence Score* (ABC UK) is a self-completed questionnaire estimate (0–100%) of how confident they are at certain activities, such as picking things up off the floor, walking on a slippery surface, reaching overhead and climbing stairs (Powell and Myers 1995).
- *The Amputee Mobility Predictor* (AMP Pro) scores components of walking in order to predict outcome and guide the rehabilitation programme (Gailey et al. 2002).
- Simple video recordings can be taken at stages of rehabilitation as an aide mémoire and a tool for comparing change.

BACPAR have produced a tool kit of suitable outcome measures which may be considered.

SPECIAL CONSIDERATIONS

Children with amputation and limb absence

Children with acquired amputation or congenital deficiency represent a highly specialised area in the field of prosthetic rehabilitation, as the numbers are relatively small. Much of the physiotherapist's time will be spent reassuring, informing and teaching the parents (who may be feeling very distressed and guilty about their child). A child cannot be forced into using a prosthesis and many manage very adequately in life without one, especially at upper limb levels. Adaptation throughout their life is dependent on acceptance of the deformity by those around them, such as family, friends and teachers. Bipedal and bimanual activities should be encouraged as early as possible, to facilitate normal child development. Acquired amputations pose different challenges than birth defects. Each person and their family is unique.

When involved in training a child, the therapist must make sessions fun and challenging. With upper limb involvement, it is often the role of the occupational therapist to teach prosthetic use, whereby the physiotherapist may manage any movement problems.

Trauma and tumour amputation

People who undergo amputation for accidental injury are often of working age. Amputation can be the result of a road traffic accident, an industrial injury, a domestic accident or gunshot wounds. They can usually compensate well and adapt physically to amputation but it can be a huge psychological blow. Rehabilitation plans must address their emotional needs, as many suffer from post-traumatic stress disorder following horrific experiences. As this patient group is young and active they have high expectations for recovery and challenge the MDT to meet their activity demands. Many need to return to work and sport (see Figure 20.12).

Figure 20.12 Running with transfemoral prosthesis.

Patients involved in accidents can claim compensation and may purchase their prosthetic rehabilitation from private clinics. A claim can take many years to settle – a trying time for the amputee.

Those who have an amputation for cancerous tumours often face arduous treatment for the cancer at the same time as recovering from their amputation. Rehabilitation needs to be quick in cases where life expectancy is shortened. An understanding of emotional status is imperative, as coupled with the trauma of limb loss, the individual has the life-threatening condition of malignancy to deal with. It may be that ongoing cancer treatment will override any prosthetic rehabilitation.

Upper limb amputation

Amputation at this level is most frequently seen in young adults and the working population, where exposure to trauma is more likely. It tends to affect men more than women. The second distinct group of patients requiring upper limb amputations is those with congenital limb absence. In these cases, amputation is not always indicated and a prosthetic socket can be fitted to the limb remnant.

Traditionally, upper limb prosthetic training is undertaken by an occupational therapist. If the amputation is on the dominant side, occupational therapy intervention

Figure 20.13 Transradial prosthesis at the fitting stage.

must be instrumental in promoting a change in hand dominance. The physiotherapist's role relates more to exercising for strength, range and co-ordination, managing oedema and offering pain-relieving modalities. Phantom pain is as common in upper limb amputation as lower limb, where people describe feeling their hand in the last position prior to the traumatic event. Secondary dysfunctions are common owing to overuse of the remaining arm and because of poor movement and stability around the affected shoulder girdle. Long-term posture exercises for the residuum, remaining limb and trunk are important.

Arms and hands are used in social interaction, as tools of expression and embrace. They are essential for providing comfort and for intimate self-care. An upper limb amputation can be very difficult to adjust to. People with upper limb amputations desire effective cosmetic replacements, as well as functional tools. Usually, more than one prosthesis is provided to meet these separate goals. Figure 20.13 shows a transradial prosthesis prior to completion. Some upper limb prostheses use body power to move prosthetic joints, where others can use myoelectric signals, sensed by electrodes within the socket, for hand control. Skin-care and pressure-management within the prosthetic socket, owing to gravity dependence rather than loading as in the lower limb cases, requires equal care and attention.

Complex cases

Due to the progressive nature of vascular disease, a third to a half of people with amputations of this origin will undergo a second amputation of their remaining leg within a few years. Bilateral amputation is difficult at any age and requires considerable environmental adaptations when the person is older and often wheelchair dependent.

People presenting with multiple amputations including extensive orthopaedic or neurological involvement pose a real challenge to the physiotherapist. All the therapist's skills are required to teach independence in activities of personal care and daily living. The whole MDT will need to work together to address all aspects. Causes of multiple limb loss are burns, meningococcal septicaemia, accidents and combat. Often rehabilitation programmes must be modified to accommodate associated skin damage and muscle loss. Energy levels for ambulation are immense, and sometimes, prosthetically prohibitive. The physiotherapist must work hard at core strength and at stamina levels. Rehabilitation using multiple prostheses can take months if not years and the physiotherapist must remain enthusiastic, focussed and motivating.

Walking with two lower limb prostheses at transfemoral level of amputation can use more than 300% more energy than if walking with their own two feet. Prosthetically, it is good practice that someone starts on short rocker pylons and stubbies to develop effective walking patterns. These are prosthetic devices comprising sockets on simple end devices that are short in stature to lower the centre of gravity and ease balance. Once control, endurance and function are reached, long articulating definitive prostheses can be tried.

Key point

Amputation is a lifelong, life-changing event. The physiotherapist plays a key role in optimising a person's independence and their reintegration into normal life experiences.

Weblinks

Amputee Running: www.amputee-running.com.
BACPAR: www.bacpar.org.uk
ISPO: www.ispo.org.uk
BAPO: www.bapo.org.uk
Scottish Physiotherapy Amputee Research Group (SPARG)/National Centre for Training in Prosthetics and Orthotics (NCTEPO), University of Strathclyde, Glasgow: www.strath.ac.uk/prosthetics
Limbless Association: www.limbless-association.org
Limb Loss Information Centre: www.limblossinformationcentre.com

FURTHER READING

BACPAR (British Association of Chartered Physiotherapists in Amputee Rehabilitation), 2009 Össur Guide to the use of the Nintendo WiiFit in Lower Limb Prosthetic Users. BACPAR, Leeds; http://www.wiihabilitation.co.uk/files/wii_fit_bro_uk_traffic_light_version.pdf; and, Protocol for use of the Nintendo WiiFIT balance board with lower limb prosthetic users in the physiotherapy department; http://www.wiihabilitation.co.uk/amputees.shtml, accessed October 2012.

BACPAR (British Association of Chartered Physiotherapists in Amputee Rehabilitation), 2008. Guidelines for the Prevention of Falls in Lower Limb Amputees. BACPAR, Leeds; http://www.csp.org.uk/sites/files/csp/secure/falls_prevention_lowerlimb_amputees.pdf; accessed October 2012.

BACPAR (British Association of Chartered Physiotherapists in Amputee Rehabilitation), 2010. Toolbox of Outcome Measures. BACPAR, Leeds; http://www.bacpar.csp.org.uk/publications; members only access.

BACPAR (British Association of Chartered Physiotherapists in Amputee Rehabilitation), 2010. Risks to the contra-lateral foot of unilateral lower limb amputees: A therapit's guide to identification and management. BACPAR, Leeds; http://www.bacpar.csp.org.uk/publications; accessed October 2012.

Broomhead, P., Dawes, D., Hale, C., et al., 2003. Evidence Based Clinical Guidelines for the Physiotherapy Management of Adults with Lower Limb Prostheses. Chartered Society of Physiotherapy, London.

Broomhead, P., Dawes, D., Hale, C., et al., 2006. Clinical Guidelines for the Pre and Post Operative Physiotherapy Management of Adults with Lower Limb Amputation. Chartered Society of Physiotherapy, London.

Butler, D.S., Lorimer Moseley, G., 2008. Explain Pain. Noigroup Publications, Australia.

Disability Information Trust, 1994. Employment and the Workplace. Disability Information Trust, Oxford.

Disability Information Trust, 1996. Sport and Leisure – Equipment for Disabled People. Disability Information Trust, Oxford.

Disability Information Trust, 1998. Powered Wheelchairs and Scooters – A Practical Guide. Disability Information Trust, Oxford.

Disability Information Trust, 1999. Outdoor Transport. Disability Information Trust, Oxford.

Divers, C., Scott, H., 2005. A Physiotherapist's Guide to Prosthetic Knee Mechanisms and Gait Training Principles. SPARG (Scottish Physiotherapy Amputee Research Group), Scotland.

Ham, R., Cotton, L., 1991. Limb Amputation. Chapman & Hall, London.

Ham, R., Barsby, P., Lumley, C., et al., 1995. Amputee Rehabilitation – A Handbook. Lumbley Associates, York.

Howells, C., 2009. The Amputee Coach. Global Publishing Group, Australia.

Johnson, S., Turner, A., Foster, M., 2001. Occupational Therapy and Physical Dysfunction: Principles, Skills and Practice, fifth ed. Churchill Livingstone, Oxford.

Lusardi, M.M., Nielson, C.C., 2000. Orthotic and Prosthetics in Rehabilitation. Butterworth-Heinemann, Oxford.

Mensch, E., Ellis, P.M., 1987. Physical Therapy Management of Lower Extremity Amputations. Heinemann Physiotherapy, London.

PIRPAG (Physiotherapy Inter Regional Prosthetic Audit Group), 2005. Physiotherapy exercises for lower limb amputation, http://www.interactivecsp.org.uk/uploads/documents/PIRPAGAdviceSheetforPhysiotherapists.pdf; members only access.

PIRPAG (Physiotherapy Inter Regional Prosthetic Audit Group), 2005. Physiotherapy exercises for lower limb amputation, http://www.interactivecsp.org.uk/uploads/documents/PIRPAGExercisesTransfemoral.pdf; members only access.

PIRPAG (Physiotherapy Inter Regional Prosthetic Audit Group), 2005. Physiotherapy exercises for lower limb amputation, http://www.interactivecsp.org.uk/uploads/documents/PIRPAGExercisesTranstibial.pdf; members only access.

Rose, J., Gamble, J., 1992. Human Walking, second ed. Williams & Wilkins, Oswestry.

Smith, D.G., Michaels, J.W., Bowker, J.H., 2004. Atlas of Amputations and Limb Deficiencies: Surgical, Prosthetic, and Rehabilitation Principles, third ed. American Academy of Orthopaedic Surgeons, Rosemount, IL.

UNIPOD (United National Institute for Prosthetics & Orthotics Development). Limbless Statistics Report – 2006/07. UNIPOD, Salford; http://www.limbless-statistics.org/, accessed October 2012.

Van de Ven, C., Engstrom, B., 1999. Therapy for Amputees 1999, third ed. Churchill Livingstone, London.

Whittle, M.W., 1991. Gait Analysis – An Introduction. Butterworth-Heinemann, Oxford.

Winchell, E., 1995. Coping with Limb Loss: A Practical Guide to Living with Amputation for you and your Family. Avery Publishing Group, New York.

REFERENCES

Bradway, J.K., Malone, J.M., Racy, J., et al., 1984. Psychological adaptation to amputation: an overview. Orthot Prosthet 38, 46–50.

Butler, D.S., Moseley, G.L., 2008. Explain Pain. Noigroup Publications, Australia.

Butler, D.J., Turkal, N.W., Seidl, J.J., 1992. Amputation: preoperative psychological preparation. J Am Board Fam Pract 5, 69–73.

Callaghan, B.G., Sockalingam, S., Treweek, S.P., et al., 2002. A post-discharge functional outcome measure for lower limb amputees: test-retest reliability with trans-tibial amputees. Prosthet Orthot Int 26, 113–119.

Campling, J., 1981. Images of Ourselves: Women with Disabilities Talking. Routledge, London.

Caron, C., 1989. Study on predictive factors for adjustment to chronic heamodialysis: a literature review and discussion of future directions. Can J Psychiatr 34 (7), 654–661.

Comerford, M.J., Mottram, S.L., Gibbons, S.G.T., 2005. Understanding Movement and Function. Kinetic Control, Shropshire.

Cutson, T.M., Bougiorni, D.R., 1996. Rehabilitation of the older lower limb amputee: a brief review. J Am Geriatr Soc 44, 1388–1393.

Davies, B., Datta, D., 2003. Mobility outcome following unilateral lower limb amputation. Pros Orthot Int 27, 186–190.

De Luccia, N., De Souza Pinto, M.A.G., Guedes, J.P.B., et al., 1992. Rehabilitation after amputation for vascular disease: a follow up study. Pros Orthot Int 16, 124–128.

Ebskov, B., 1999. Relative mortality and long term survival for the non-diabetic lower limb amputee with vascular insufficiency. Prosthet Orthot Int 23, 209–216.

Ehde, D.M., Smith, D.G., Czerniecki, J.M. et al., 2001. Back pain as a secondary disability in persons with lower limb amputations. Arch Phys Med Rehabil 82, 731–734.

Fraser, C.M., Halligan, P.W., Robertson, I.H., et al., 2001. Characterising phantom limb phenomena in upper limb amputees. Pros Orthot Int 25, 235–242.

Fyfe, N.C.M., 1992. Assessment of the rehabilitation of amputees. Curr Pract Surg 4, 90–98.

Gailey, R.S., Roach, K.E., Applegate, E.B., et al., 2002. The amputee mobility predictor: an instrument to assess determinants of the lower-limb amputee's ability to ambulate. Arch Phys Med Rehabil 83 (5), 613–627.

Gallagher, P., MacLachlan, M., 2004. The trinity amputation and prosthesis experience scales and quality of life in people with lower limb amputation. Arch Phys Med Rehabil 85, 730–736.

Gauthier-Gagnon, C., Grisé, M., Lepage, Y., 1998. The locomotor capabilities index: content validity. J Rehabil Outcome Measure 2 (4), 40–46.

Grisé, M.C., Gauthier-Gagnon, C., 1993. Prosthetic profile of people with lower extremity amputation: conception and design of a follow up questionnaire. Arch Phys Med Rehabil 74, 862–870.

Hagberg, K., Haggstrom, E., Branemark, R., 2007. Physiological cost index (PCI) and walking performance in individuals with transfemoral prostheses compared to healthy controls. Disabil Rehabil 29 (8), 643–649.

Ham, R., McCreadie, M., 1992. Rehabilitation of elderly patients in the United Kingdom following lower limb amputation. Top Geriatri Rehabil 8 (1), 64–71.

Hill, A., Niven, C.A., Knussen, C., 1995. The role of coping in adjustment to phantom limb pain. Pain 62, 79–86.

Krueger, D.W., 1984. Rehabilitation Psychology – A Comprehensive Textbook. Aspen Publications, Maryland.

Kubler-Ross, E., 1969. On death and dying. Macmillan, London.

Lambert, A., Johnson, J., 1995. Stump shrinkers: a survey of their use. Physiotherapy 81, 234–236.

Lein, S., 1992. How are physiotherapists using the Vessa Pneumatic Post Amputation Mobility Aid? Physiotherapy 78 (5), 318–322.

Miller, W.C., Deathe, A.B., Speechley, M., et al., 2001. The influence of falling, fear of falling, and balance confidence on prosthetic mobility and social activity among individuals with a lower extremity amputation. Arch Phys Med Rehabil 82 (9), 1238–1244.

Norvell, D.C., Czerniecki, J.M., Reiber, G.E., et al., 2005. The prevalence of knee pain and symptoms knee osteoarthritis among veteran traumatic amputees and non amputees. Arch Phys Med Rehab 86 (3), 487–493.

O'Shea, E., Kennelly, B., 1996. The economics of independent living: efficiency, equity and ethics. Int J Rehabil Res 19 (1), 13–26.

Park, S., Fisher, A.G., Velozo, C.A., 1994. Using the assessment of motor and process skills to compare occupational performance between clinic and home settings. Am J Occup Ther 48 (8), 697–709.

Parkes, C.M., 1972. Bereavement Studies of Grief in Adult Life. Tavistock, London.

Parkes, C.M., 1975. Psychological transition comparison between reactions of loss of limb to loss of spouse. Br J Psychol 127, 204–210.

Pernot, H.F., 1997. Daily functioning of the lower extremity amputee: an overview of the literature. Clin Rehabil 11 (2), 93–106.

Powell, L.E., Myers, A.M., 1995. The Activities-specific Balance Confidence (ABC) Scale. J Gerontol Med Sci 50 (1), M28–M34.

Ryall, N.H., Eyres, S.B., Neumann, V.C., et al., 2003. The SIGAM mobility grades: a new population – specific measure for lower limb amputees. Disabil Rehabil 25 (15), 833–844.

Van de Ven, C., Engstrom, B., 1999. Therapy for Amputees, third ed. Churchill Livingstone, London.

Waters, R.L., Mulroy, S.J., 2004. Energy expenditure of walking in individuals with lower limb

amputations. In: Atlas of Amputations and Limb Deficiencies: Surgical, Prosthetic, and Rehabilitation Principles. American Academy of Orthopaedic Surgeons, Rosemount, IL.

Watson, G., 1996. Neuromusculoskeletal Physiotherapy: encouraging self-management. Physiotherapy 82 (6), 352–357.

Weiss, S.A., Lindell, B., 1996. Phantom limb pain and etiology of amputation in unilateral lower extremity amputees. J Pain Symptom Manage 11 (11), 3–7.

Williams, A.M., Deaton, S.B., 1997. Phantom pain, elusive yet real. Rehabil Nurs 22 (2), 73–77.

Yetzer, E.A., Kauffman, K., Sopp F, et al., 1994. Development of a patient education programme for new amputees. Rehabil Nurs 19 (6), 355–358.

Chapter 21

Massage

Joan M. Watt

INTRODUCTION

Massage is the starting point of the physiotherapy profession. Swedish massage was the basis for the strokes and principles used by the first members of the Chartered Society of Physiotherapists. There is evidence of massage being used in many ancient civilisations. The word massage may derive from the Arabic 'mass', meaning to press, or the Greek word 'massien' meaning to knead.

ABC Definition

Massage is an age-old process that involves stimulation of the tissues by rhythmically applying both stretching and pressure (Watt 1999). Holey and Cook (2003) define therapeutic massage as '…the manipulation of the soft tissues of the body by a trained therapist as a component of a holistic therapeutic intervention.'

PREPARATION

Preparation encompasses the treatment room, couch, self, patient and contact medium.

Treatment room

The treatment room needs to be kept at a comfortable temperature to ensure the patient is not subjected to chills or draughts. It must also afford the necessary privacy without contravening the patient's rights. There must be a treatment couch, small arm table and chair.

Couch

In the clinical setting most treatment couches/plinths can have the height adjusted to suit the individual therapist. To get a good height for performing massage, stand side-on to the couch, rest your hand with fingers loosely flexed at the metacarpal phalangeal joints and elbow not quite fully extended. Adjust height of couch until you can comfortably rest your hand as above. This takes time, trial and error to gain the optimum position (Figure 21.1). The couch should have a fresh clean cover and also a paper couch roll, pillows, a face hole or face pillow, and blanket/towels to cover the patient.

Self preparation

Stance

It is very important to ensure that your stance allows you to reach all the parts to be massaged in a specific stroke without having to keep altering position. Stand in lunge position to perform long strokes, such as effleurage and stroking (Figure 21.2). To reach across the patient assume a similar position facing the couch (Figure 21.3). Do not stand still – let your whole body go with the stroke and always keep your pelvis tucked and knees slightly bent to protect your back. Ensure your shoulders are relaxed, comfortable and not hunched.

Clothing

Clothing should be loose enough to allow unrestricted movement whilst looking neat and professional. Hair should not be allowed to come into contact with the patient. If hair is long enough to touch the collar it should be tied back.

Figure 21.1 Setting the plinth to the correct height.

Figure 21.2 Lunge stance.

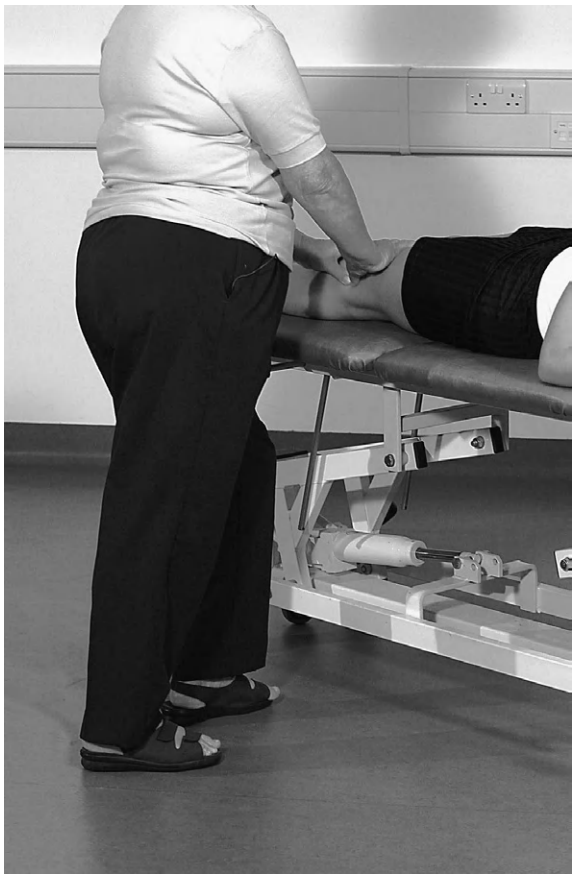


Figure 21.3 Reaching the patient.

Hands

Hands are your most important tool when using massage. They must be clean, smooth and have skin that is well nourished by using hand creams regularly. Nails should be free from polish and cut short, so they do not show above the fleshy finger pad. Only a very thin wedding ring can be worn, provided the patient does not experience any irritation from it. All other rings, bracelets, bangles and watch must be removed. It is also important to practise exercises to improve the ranges of movement of your hands. Hollis (2009) advocates full abduction/extension of the thumb to give a wide grasp of an octave span, and full flexion and extension of the wrists or at least 80 degrees of each movement, plus full pronation and supination of the radio-ulnar joints.

Exercises

1. Make a fist and then spread fingers and thumbs out as far as possible; hold for 5–10 seconds.
2. Place the finger tips of one hand in contact with finger tips of other and press so that thumbs and little fingers are as widely spaced as possible.
3. Push two, three and then four fingers between two adjacent fingers of the other hand. Repeat in each space of both hands.
4. Place palms together with fingers and thumbs in contact and elbows bent at chest level. Slowly turn hands to point fingers to ground. Return to start position.
5. Place the backs of the hands together, hold arms out straight and bend elbows up towards chin to flex wrists.
6. Clasp the hands with wrists crossed and elbows straight. Bend the elbows to bring hands up to the chin and straighten the elbows out. Keep hands firmly clasped at all times. Do not force – go only as far as is possible (Figure 21.4a–c).

Practice rhythm

Sit with a pillow on your knee with your hands open and relaxed. Try to clap as rhythmically as possible. Repeat with the ulnar borders of hands, clenched fists and ulnar borders of fists (Figure 21.5).

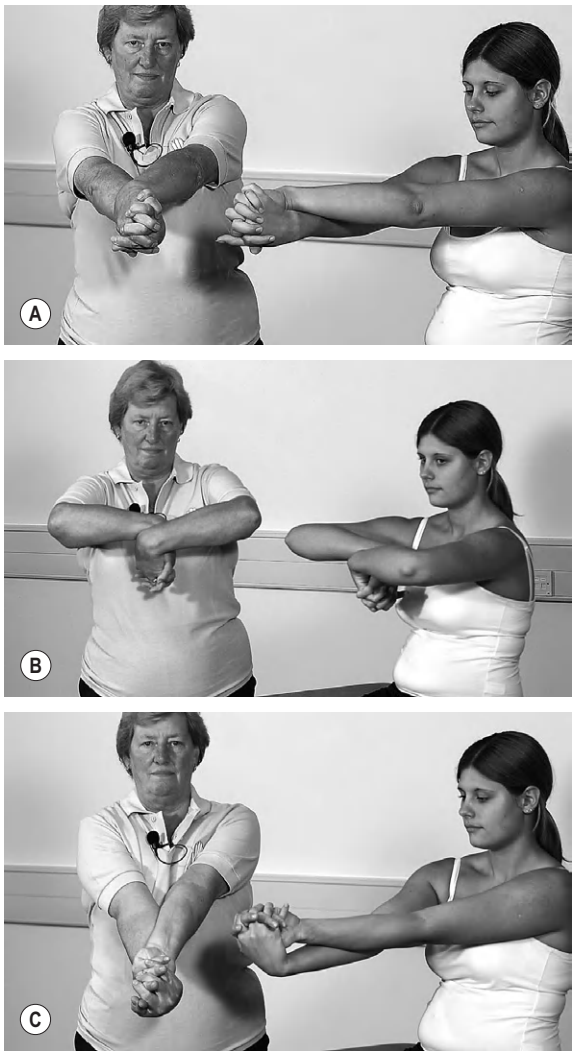


Figure 21.4 (a–c) Pre-massage exercises.

Figure 21.5 Practising your rhythm.

Figure 21.6 Practising strokes.

Practice strokes

Sit with a pillow on your knees and grasp the outer edge of pillow with lightly clenched hands. Alternately push and pull with your hands (Figure 21.6).

Palpation

The sense of touch and palpation skills must be highly developed in the person performing massage. It requires practice to attune and improve our sense of touch and therefore palpation skills. Start by feeling the differing textures of various types of material. Sit with a pillow on your knees and feel the pillow slip between fingers and thumb, then do the same with a towel (Figure 21.7a). Once you are happy you can easily tell one type of fabric from another repeat the exercise with your eyes closed (Figure 21.7b). Use your own body to identify anatomical landmarks and accustom your hand to the feel of differing tissues. Start by feeling the point of your elbow first with thumb tip, then each finger separately and together, and then with the palm of your hand. Repeat with each hand in turn; close your eyes and focus on the message you receive from your hands. Try the same routine on the calf, shin, anterior aspect of the thigh, forearm, neck, shoulders and abdomen.

Figure 21.7 (a, b) Assessing textures.

Your sense of touch can be improved by many different methods. Put a selection of different round objects of various size and texture into a bag and then try to identify each by touching only. As this becomes easier choose smaller articles of more closely related textures and time how long it takes to identify correctly.

It is also important to learn the depth of contact. Too light can be uncomfortable and ticklish and is frequently referred to as 'skin polishing'. Too-heavy firm pressure may well produce trauma and damage tissue. Practise depth again on your own skin – try to move your hand across your leg as lightly as possible and then increase depth until you feel it is producing pain. Try out on a friend or colleague and agree a score rate from, say, 1–5 of very light to heavy. You make your mind up what depth/number you are contacting then ask your model for their answer. Always listen to what the model tells you and alter to get to the score they think it is.

Patient preparation

It is important that the patient is fully informed of exactly what the massage session will involve. Consent is dealt with in 'Legal aspects' and must always be in place before any massage starts.

It is vital to request the patient to remove the specific pieces of clothing required to permit the treatment. Always explain the reasons behind these requests. The patient's decency and modesty has to be respected at all times.

Neither the physiotherapist nor patient should ever feel uncomfortable, threatened or embarrassed by the manipulations being used. Always tell the patient exactly what you are doing and why. Explain why strokes have to be taken right up into the lymph drainage areas and make sure they are content to have this done.

Draping or covering the patient has to be properly carried out. Use shorts, towels of various sizes, towelling robes and togas (Figure 21.8). Patient's own clothing is also very useful. Again, explain why you want the towel tucked in and wherever possible get the patient to do this for themselves.

Never remove any article of the patient's clothing without asking their specific permission to do so. Remember it is possible to apply some forms of massage through one layer of clothing or over a thin cloth or towel.

Coupling media

Many and varied products are used as coupling media in massage. Massage oils, creams, gels and powders, and

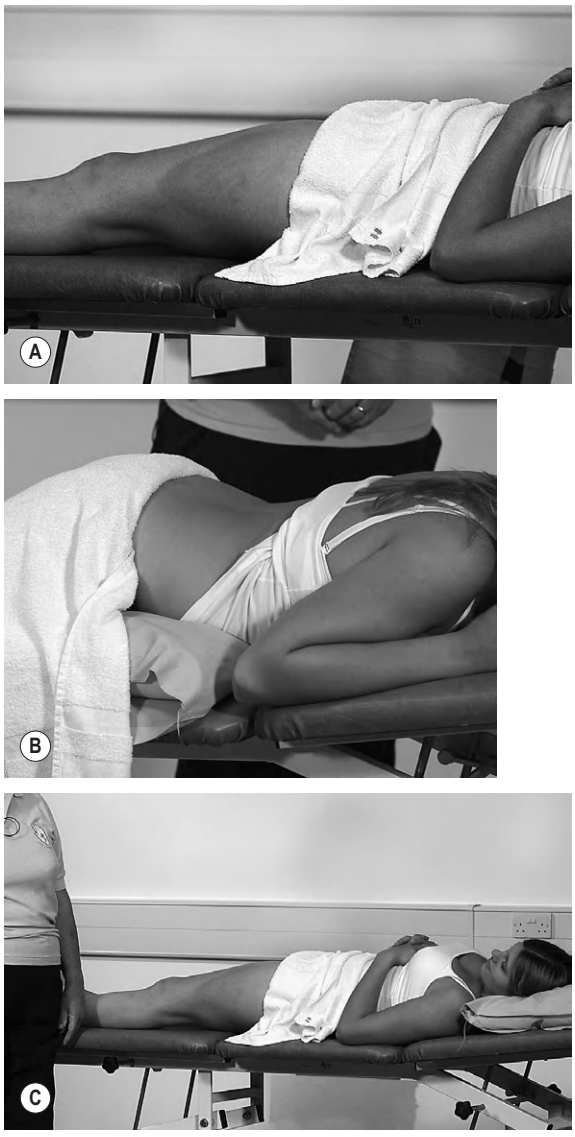


Figure 21.8 (a–c) Covering the patient.

aromatherapy products are readily available. The first and most important step is to ensure that the lubricant selected is acceptable to both patient and masseur, and there are no contraindications to its use.

It is essential that there is full information on all the contents of the chosen product. This is vital if the recipient or masseur has any allergies. Many massage oils/creams use nut oils as a base and, obviously, this should never be applied in cases of nut allergy. Very hairy skin can be irritated and damaged by the use of powder or too little lubricant during massage. Powder should never be applied to hot sweaty skin as it clogs pores and tends to form hard lumps on the surface.

Ice massage can be extremely useful but great care must be taken to ensure the skin is not damaged by the effect of the ice. Always ensure the ice is kept moving, regularly check skin colour and reaction. Stop if the patient reports feeling uncomfortable or cooling too fast.

Apply oils directly to your hands and do not use too much. Always make sure the whole area to be treated is clean before starting and also clean thoroughly at the end of massage.

Water alone or soapy water can be used with good effect as a lubricant and the addition of a small amount of oil can be beneficial when dealing with dry, scaly skin.

Never apply any heating agent/cream when you are treating any area where there has been tissue damage and healing. A hot cream may produce too much superficial increase in skin temperature and create further damage.

LEGAL ASPECTS

It is mandatory to acknowledge and implement all pertinent legal requirements before, during and after using massage. The practitioner must explain the reasons for applying massage and explain exactly what is involved. Written informed consent must be obtained – to apply massage without such consent can be construed as assault. Throughout the whole treatment both patient and physiotherapist must be totally comfortable and at ease with the methodology. Keep inquiring if the patient is happy and content with the proceedings.

All child protection, chaperone and local laws must be obeyed. Remember to keep accurate, legible, dated and signed treatment notes. Don't just write 'massage': give a short description of techniques used and list outcomes.

CONTRAINDICATIONS

In some conditions massage can exacerbate problems and can be dangerous. Always have a diagnosis, carry out an assessment and be clear on the aims of the treatment.

1. Skin infections of viral, fungal or bacterial origin are contraindications to massage. Any type of skin infection in the area to be massaged or involving the hands of the physiotherapist would preclude the application of massage.
2. Open wounds should not be massaged. Apart from being painful, massage can damage the healing tissue and restart bleeding and may cause infection.
3. Circulatory problems can be adversely affected by massage which increases blood flow and manipulates the blood vessels. Bleeding disorders, such as haemophilia, arteriosclerosis, haemorrhage, thrombosis and artificial blood vessels, are all

contraindications to massage. Remember deep vein thrombosis is a total contraindication to massage.

4. Recent injury, if massaged too early in the healing process, will cause further damage. Massage at that stage will cause trauma to the fragile healing tissue leading to delay in repair, further bleeding and resultant excess scar tissue. It is recommended that massage should not be started until 48 hours after injury. Always ensure bleeding has stopped. Proceed with great care and use very gentle strokes initially.
5. Tumours can be mechanically stimulated by massage which speeds up metabolism and so spreads the tumour. Do not massage directly over or close to a tumour. Remember massage can be very effective and prove an invaluable aid to cancer victims and in palliative care.
6. Acute inflammation is contraindicated for massage as the inflammatory process can be increased by the effects produced.
7. Myositis ossificans should not be massaged directly. This can produce increased formation of bony cells and further damage the soft tissues surrounding the site. Vigorous deep massage has been known to separate small bony particles from the site of ossification.
8. Diabetes – although not a total contraindication to massage, great care must be used when treating diabetics with massage. It is usual for the peripheral circulation to be affected in diabetes and as a result blood vessels and skin can be easily damaged. Apply massage with great care.
9. Alteration of skin sensation can be a contraindication to the use of massage.

Massage may be used in certain circumstances, provided the experienced practitioner has an accurate diagnosis and knows the extent and reason for the changes. Loss of sensation, heightened sensation, presence of tingling or neurological skin alteration all contraindicate massage.

TECHNIQUES

Massage manipulations are grouped into various headings. The three listed are the main components of basic massage.

1. Stroking manipulations.
2. Pressure or petrissage manipulations.
3. Percussive or tapotement manipulations.

Stroking manipulations

There are two strokes in this group:

- stroking and
- effleurage.

Figure 21.9 (a, b) Stroking.

Stroking

This is, as the name suggests, a stroking movement, traditionally performed from distal to proximal in the direction of the lymph drainage (Figure 21.9a). It can also be performed in the opposite direction or in a cross over method.

This manipulation can be applied using pads of fingers, thumbs, one, two or alternate hands (Figure 21.9b). A specific type of stroking is 'Thousand Hands', as first described by Hollis (2009). In this case, one hand is used to perform a short stroke and then the second hand performs the same movement overlapping the first. This is a fairly light touch, but must not be so light as to irritate or tickle, and can be applied in fast or slow strokes.

Effects of stroking

- Gets the patient used to your touch.
- Allows assessment of the state of the skin and tissues of the area to be treated.
- Improves sensory analgesia.

Figure 21.10 (a, b) Effleurage.

- Slow stroking will relax and sedate and decrease muscle tone.
- Faster strokes will stimulate superficial blood flow, accelerate lymph drainage.
- Used at start and end of session.

Effleurage

The meaning of this word is to stroke; in massage it is a deeper form of stroking (Figure 21.10a). As such, it can be applied as described for stroking and also be reinforced with one hand over the other, or using lightly clenched fist (Figure 21.10b) or forearm. Because this manipulation goes deeper it can be graded. Grade 1 is sufficient to influence flow in superficial vessels; grade 2 affects deeper vessels; and grade 3 applies to reinforced effleurage, with one hand on top of the other. Effleurage can be applied in the direction of the lymph glands, or opposite, in all sorts of patterns from a Figure 7 or 8, to a circular or T shape.

Effects of effleurage

- Assists lymphatic and venous return.
- Assists interchange of tissue fluid.
- Assists removal of waste product and chemical irritants.
- Passively stretches muscle fibres.
- Restores mobility at tissue interfaces.
- Light strokes decrease muscle tone.
- Deep strokes increase muscle tone.
- Used at start, end and inbetween other manipulations.

Clinical note

The listed contraindications apply for stroking and effleurage. Also ensure enough pressure and contact to prevent tickly sensations and apply sufficient lubricant to allow smooth movement over the skin at all depths.

Pressure manipulations or petrissage

The petrissage group is made up of five categories: kneading, picking up, wringing, rolling and shaking.

Kneading

The tissues are compressed against the underlying structures during this stroke. The kneading action is performed in a circular movement either from proximal to distal or distal to proximal (Figure 21.11a,b). Whole hand, one, both or alternate, finger and thumb tips, or finger and thumb pads may be used to perform this manipulation. The stroke can also be superimposed or reinforced by placing one hand or other fingers on top of the contact. The action is to perform a circular movement with pressure on the upward part for about 25% of the circumference. Contact must be maintained for the rest of the circle and only be lifted to move on to the next circle.

Kneading is graded into three grades: grade 1 is sufficient to influence superficial vessels and compress superficial tissues on underlying structures; grade 2 affects deeper tissue drainage and will compress deep tissue on underlying structures; and grade 3 is applied to superimposed or reinforced strokes and may be as much as can be tolerated by the patient without producing tissue damage.

Effects of kneading

- Stimulates venous and lymphatic flow.
- Increases mobility of fibrous tissues.
- Helps interchange of tissue fluids.
- Helps prepare soft tissue for exercise.
- Helps removal of waste products.
- Increases length and strength of connective tissues.

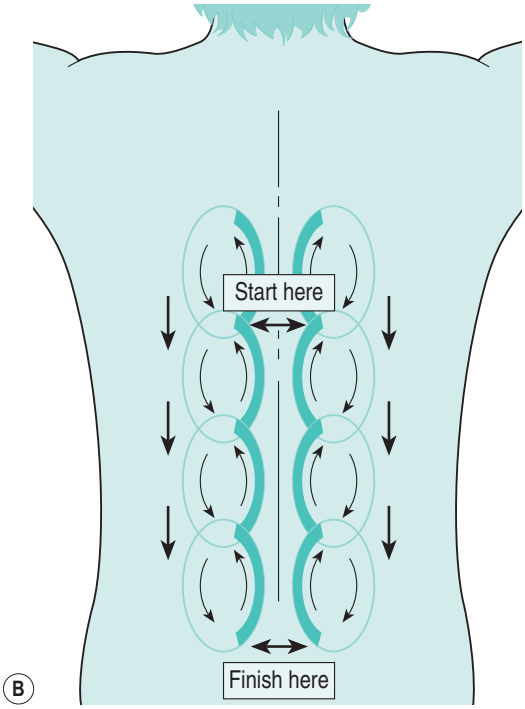


Figure 21.11 (a, b) Kneading.

Figure 21.12 Picking up: single-handed C. (a) Hand position; (b) on the patient.

Figure 21.13 Picking up: single-handed V. (a) Hand position; (b) on the patient.

- Provokes somato-visceral reflex effects.
- Restores mobility between tissue interfaces.

Clinical note

The listed contraindications above apply to kneading. Be careful not to compress too heavily. Take care not to nip tissue by squeezing too hard.

Picking up

The tissues are compressed against underlying structures, then 'picked up', lifted, squeezed and released. The manipulation can be single-handed C-shape (Figure 21.12a), double-handed alternate C-shape, single-handed V-shape (Figure 21.13) or double-handed V-shape. For C-shape, the thumb is held away from the palm so that the web between it and the first finger form a C-shape (Figure 21.12b). V-shape is when the web forms a letter V (Fig 21.13).

Grading for picking up is the same as kneading with grade 3 only being used in the double-handed manipulation and will only be tolerated over very muscular areas.

Effects of picking up

- Exactly the same as for kneading.
- Particularly good for mobilising soft tissues.
- Use after effleurage and kneading.

Clinical note

The contraindications as listed above apply to picking up. Be sure there is no recent soft tissue trauma. Be aware that if skin contact is lost during this manipulation a pinching action will result.

Wringing

In wringing the tissues are compressed against underlying structures, then one hand pulls towards the physiotherapist and the other hand pushes away. Fingers and thumb can be used in wringing with the tissues being compressed between them. The tissue is then lifted and pulled towards you with one hand and pushed away from you with the other. Full hands are also used in this stroke (Figure 21.14). There are only two grades for wringing: 1 and 2. Grade 1 is usually applied to finger strokes only with grade

Figure 21.14 Wringing.

Figure 21.15 Rolling.

2 using the whole hand. There is no grade 3 as it would be too painful to tolerate.

Effects of wringing

These are as stated for kneading. Wringing is particularly good for separating superficial and deep adherent tissue.

Clinical note

The usual contraindications apply for wringing. Be aware of tissue damage if used on recently injured tissue. Grasping tissue too tightly will produce a nipping effect.

Rolling

In this manipulation both hands are used. Contact is made with both palms and the thumbs are held away from the fingers; the tips of the thumbs are close to, or touch, each other (Figure 21.15). The fingers pull the tissue towards the thumbs and then the thumbs squeeze and lift to push the tissue away.

There are two types of rolling – skin rolling and muscle rolling. In muscle rolling, the roll action is not so marked and the action is more push with fingers and pull back with the thumbs. Skin rolling is grade 1 and muscle rolling is grade 2. Again, there is no grade 3 because it would be too painful.

Figure 21.16 Shaking.

Effects of rolling

- These are as stated for kneading.
- Rolling also mobilises scar tissue.
- When performed slowly it has a stretch effect on the tissues being manipulated.

Clinical note

The usual contraindications apply for rolling. Take care not to nip tissues on applying action. Do not be too vigorous when working with recent scar tissue.

Shaking

In this manipulation muscle or more superficial tissue can be shaken from side to side. The tip of the thumb and tips of one or more fingers are used when treating small areas. The full length of thumb and all fingers are used in larger areas (Figure 21.16). In a very large area, such as the buttocks or thighs, the whole flat hand can be placed on the area and after applying gentle compression shaken. In the final type of shaking – whole limb shaking – the leg or arm is lifted, gently supported and then shaken. Grade 1 shaking is used to describe thumb and finger tip shaking; grade 2 applies to full thumb and fingers and also to flat hand; grade 3 is whole limb shaking.

Effects of shaking

- This manipulation produces a feeling of invigoration and stimulation.
- Increases tissue mobility.
- Assists in breaking down tissue adhesions.
- Stimulates lymphatic and venous flow.
- Helps prepare soft tissues for stretch and exercise.

Figure 21.17 Hacking.

Figure 21.18 Clapping.

Clinical note

The usual contraindications apply to shaking with specific care being taken to ensure the tissues are not nipped when using fingers and thumb. It is important in whole limb shaking that the patient is relaxed and there is no 'kick back' produced in any joint or any feeling of strain by the patient.

Percussion or tapotement manipulations

In this category of massage strokes there are five main types: hacking, clapping, beating, pounding and vibration. In some cases, the last mentioned stroke could be applied to the petrissage group but that tends to be in the more specific massage for sport and not the general concept.

Hacking

To perform this manipulation the arms are held abducted with the elbows bent. The area to be massaged is then hit by the medial border of the hands and/or fingers (Figure 21.17). The action is produced by pronation and supination of the radio-ulnar joints. It is important that the practitioner's shoulders and hands are relaxed and not held in any tension. The rhythm of all percussion strokes should be practised to ensure it is consistent.

Grade 1 uses only the medial borders of the fingers; grade 2 uses medial borders of hands and fingers; grade 3 uses the medial borders of hands and fingers more deeply and slowly.

Effects of hacking

- Stimulates local circulation.
- Stimulates muscle tone.
- Gives generalised feeling of stimulation.
- Provokes muscle and tendon reflexes.
- Light strokes effect superficial tissue.
- Deeper strokes aid the evacuation of the lungs.

Clinical note

All the usual contraindications apply to hacking. When using deep technique it is good practice to cover the area with a thin cloth to prevent any skin irritation.

Clapping

To perform this stroke the hands are held loosely cupped and the area is struck by the palmar aspect of the hands and fingers (Figure 21.18). The action is produced by alternately flexing and extending the wrist joints.

Grade 1 clapping is very superficial, often called skin clapping. This is used at a fairly fast pace with minimal contact. Grade 2 is deeper, slower and firmer, and grade 3 clapping is very firm and may involve elbow, as well as wrist, action.

Effects of clapping

These are the same as listed for hacking with the exception of provoking tendon or muscle reflex.

Clinical note

All the usual contraindications apply to clapping. Always ensure the hands are relaxed or the patient will find the contact produces pain. In grade 3 clapping, cover the area with a light cloth.

Beating

This form of tapotement involves the use of lightly clenched fists to hit the area. The action involved is wrist

flexion and extension with the finger/palmar area making contact with the treatment area (Figure 21.19).

Grade 1 beating is performed at a fast rate, with fairly light contact. Grade 2 is slower with firmer contact and grade 3 is very deep and the rate can be varied as required.

Effects of beating

The effects are identical to clapping.

Clinical note

The usual contraindications apply to beating. Again, ensure that hands are relaxed during the stroke. Always make sure you cover tissue with light cloth with grade 3.

Pounding

In this stroke the hands are held in lightly clasped fists with the thumbs resting against the first fingers. The action is to pronate and supinate the radio-ulnar joints and make contact with the patient using the ulnar border of the hand (Figure 21.20).

Grade 1 of this stroke applies to fast rate, light contact. Grade 2 is used for slower rate with firmer contact and grade 3 is deeper, with varying rates.

Effects of pounding

These are exactly the same as listed for clapping.

Clinical note

The same precautions should be applied as for clapping.

Vibrations

In this manipulation the tissues are pressed and moved up and down, or away from and towards the manipulator and then released. At the same time small oscillations of the whole arm produce a trembling effect. A single hand or both hands (Figure 21.21) may be used, and hands should be held slightly in flexion.

Grade 1 applies to light rapid movements. Grade 2 is firmer and slower with more tissue movement, and grade 3 is as firm a pressure as can be tolerated and in a very slow action.

Effects of vibrations

- Stimulates muscle tone.
- Stimulates local circulation.
- Provides feeling of well-being.
- Aids peristalsis.

Figure 21.19 Beating.

Figure 21.20 Pounding.

Figure 21.21 Vibrations.

Clinical note

The usual contraindications apply to vibrations. Hands must make firm enough contact to not produce ticklish sensation.

OTHER TECHNIQUES

Massage is still evolving and, as a result, there are newer types of manipulations being added and with the increase in good scientific research we are able to understand what these can do. We shall consider a few here, but this is in no way a comprehensive list. As long as massage is a well-used, practised and evaluated skill it will develop and new ideas will be added.

In this section we will look at myofascial release, frictions, trigger pointing and acupressure.

Myofascial release

As the name suggests, this type of manipulation is used to produce change in the myofascia, both superficial and deep. Myofascia is continuous throughout the body and is made up of collagen fibres. Superficial fascia underlies the skin and deep fascia covers, separates and protects skeletal muscle. The functions of the fascia are to form, support, provide boundaries, guide, mould, compartmentalise and control.

Problems associated with the fascia which respond to the myofascial massage manipulations are:

- adhesion formation;
- restricted or prevented movement;
- alterations to the internal muscle structure;
- fibrositis;
- tissue contraction and scarring.

Strokes used in myofascial release are myofascial spread, fascial lift and roll, and myofascial mobilisation.

Myofascial spread

This manipulation is basically a stretch technique. Finger tips or hands in a line or crossed can be used. The hands/fingers are laid on the tissue and pressed in until a slight resistance is felt. The hands are then drawn apart evenly until the tissue will go no further. This position is held until the resistance gives and the hands slide further apart (Figure 21.22). The movement is slow, steady and sustained.

Grade 1 applies to release of superficial fascia and grade 2 deals with deep fascia.

Effects of myofascial spread

- Makes fascia more fluid.
- Releases collagen bonds.
- Lengthens fascial layers.
- Restores mobility.
- Decreases effects of adhesions and scars.

Figure 21.22 Myofascial spread.

Clinical note

All the general contraindications apply to the use of myofascial techniques. Caution should be used when dealing with flaccid paralysis, lax or unstable joints. Beware some advanced arthritic joints are held by fascial splinting.

Fascial lift and roll

This technique is very similar to skin and muscle rolling but is aimed at the fascia. Both hands are placed over the area, the tissue is lifted until a very slight resistance is felt below the skin level. The tissue is then lifted slowly and pressed by the thumbs towards the fingers (Figure 21.23).

Grade 1 only applies as only used on superficial fascia.

Effects of fascial lift and roll

- Releases adherent areas in the superficial fascia.

Clinical note

Precautions are exactly as for myofascial spread. Never force the tissue.

Myofascial mobilisation

To perform this stroke the fingers, knuckles, palms or forearm are placed on the surface, pressure is applied and the myofascia is moved against the underlying structures. The action can be forwards and backwards or circular in pattern (Figure 21.24).

Grade 1 is light and is used for the superficial fascia. Grade 2 is deeper and mobilises the deeper fascia.

Figure 21.23 (a, b) Fascial lift and roll.

Figure 21.24 Myofascial mobilisation.

Effects of myofascial mobilisation

- Frees myofascia which is adhering to itself or underlying tissues.
- Re-establishes the ability of tissues to slide freely over each other.

Clinical note

Precautions are as for myofascial spread. Myofascial mobilisation should not produce pain.

Frictions

This massage technique was developed by Dr James Cyriax to treat soft tissue lesions (Cyriax 1993). This is a localised technique applied at the injury point and aims to give a stretching across the fibres to separate them and restore mobility.

To perform this stroke the finger tips or thumbs are applied either alone or reinforced by the adjacent finger. The action is then to move across the tissue (transverse friction (Fig 21.25)) or perform a circular pattern (circular friction).

Grade 1 does not apply to this technique as friction is aimed at deeper structures; grade 2 is sufficient to affect deep tissue and cause compression; grade 3 applies to reinforced and most transverse frictions, and may produce pain before causing numbing.

Effects of frictions

- Restores tissue mobility.
- Stimulates local circulation.
- Aids the resolution of inflammation.
- Reduces pain as a counter irritant effect.
- Stretches fibrous tissue.

Clinical note

All the usual contraindications apply. Friction must be given to exact site of damage. Skin must move with the finger/thumb tips. Use little, or no, lubricant. Warn patient treatment can cause pain until numbing takes place – usually about two minutes. Always apply within the patient's tolerance.

Trigger pointing and acupressure

Trigger points in the myofascia were first identified by Travell and Simons (1992). These points are bands within a muscle which have become ischaemic and fibrous because they have been held in excess spasm or tone for some time. The cause can be postural or mechanical and may be affected by metabolic, nutritional or other factors.

The trigger points are easily identified by palpation. Pressure produces pain and this can be in the particular pattern of referral as described by Travell and Simons.

Figure 21.25 Deep transverse friction to the gastrocnemius musculotendinous junction.

Figure 21.26 Trigger pointing.

Types of trigger point

There are various types of trigger point and it is important to address all when treating:

- active – produces pain at rest;
- latent – only pain on palpation;
- primary – response to trauma, acute or chronic overload;
- key – activates or neutralises satellites;
- satellite – activated by key, situated in area of referral or antagonist or synergist;
- general – near centre of muscle fibre;
- attachment – in tendon or aponeurosis.

To apply trigger pointing technique a deep stroking is performed by the tips of finger or thumbs. Direct pressure may be applied to the specific point by thumb, finger tip or elbow (Figure 21.26). Hold for 8–12 seconds, treat the area of referred pain and always stretch after trigger pointing. When full range has been restored the trigger point will no longer be active.

There is no grade 1. Grade 2 applies to the stroking technique and grade 3 is as much as the patient can tolerate.

Effects of trigger pointing

- Gives pain relief.
- Reduces muscle spasm.
- Helps restore normal muscle tone.
- Allows full stretch of tissue.

Clinical note

The usual contraindications apply to trigger pointing. It must always be applied within the patient's tolerance.

Acupressure

Acupressure is very similar to trigger pointing, where direct pressure is applied. In this technique the pressure is applied to the points on the acupuncture meridian lines with the aim of producing effects on the tissue without using needles.

Effects of acupressure

- Pain relief by release of endorphins.
- Stimulates local circulation.
- Stimulates lymphatic flow.

Clinical note

Contraindications as mentioned before apply to acupressure. It should not be used on patients with heart disease or visceral conditions. The therapist must have full knowledge of acupressure points and an understanding of energy points and the philosophy behind the oriental belief.

SPECIFIC USAGE OF MASSAGE

Tension headaches

Tension headaches respond well to treatment with massage techniques. Start off with effleurage to identify particular tension spots and accustom them to touch. Address the mid-scapular, trapezius and levator scapulae areas with trigger pointing (Figure 21.27a), then treat the base of the skull with pointing and gentle thumb/finger circular kneading (Figure 21.27b). The length of time

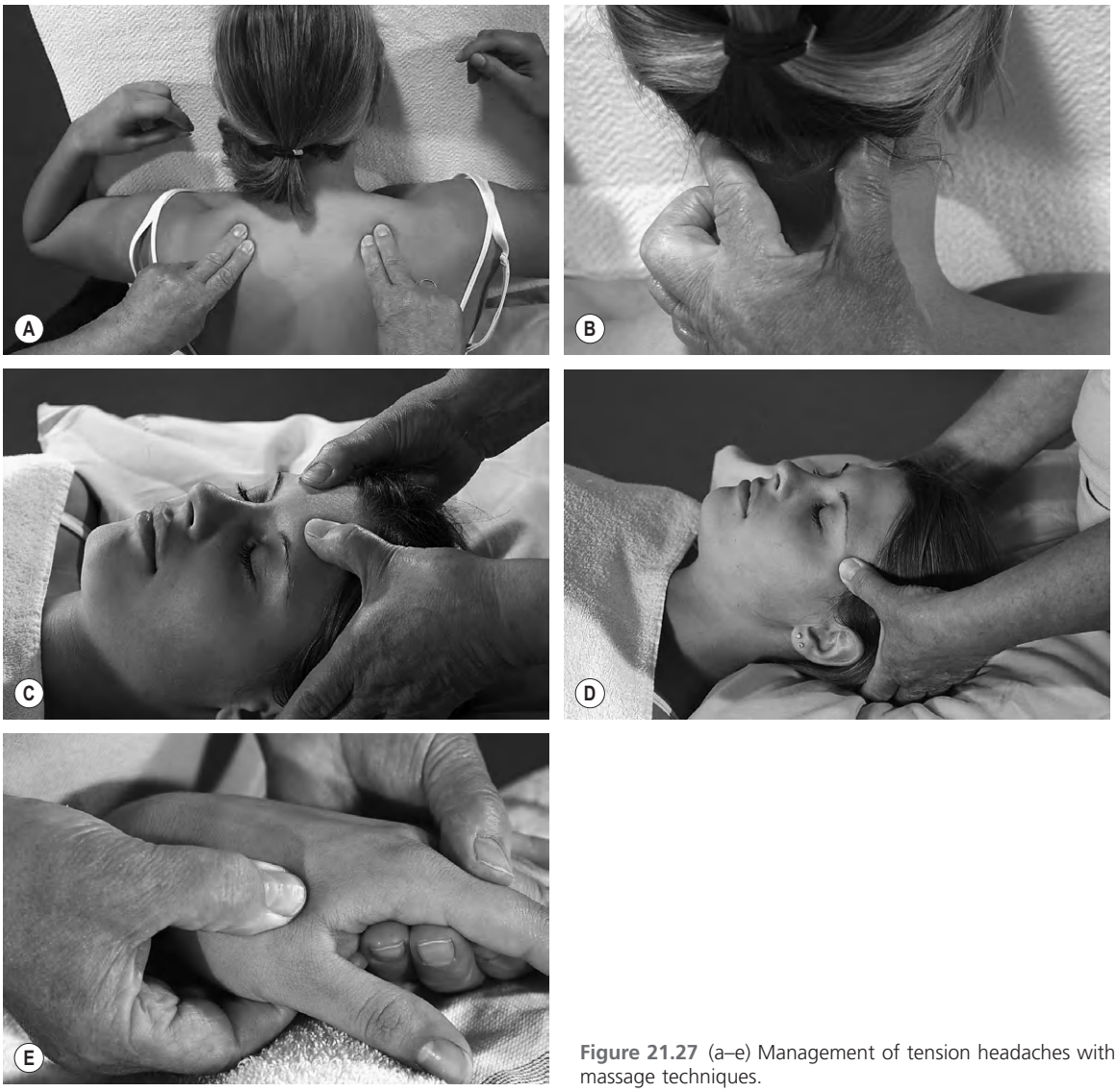


Figure 21.27 (a–e) Management of tension headaches with massage techniques.

Figure 21.28 (a, b) Frictions for lateral epicondylitis (tennis elbow).

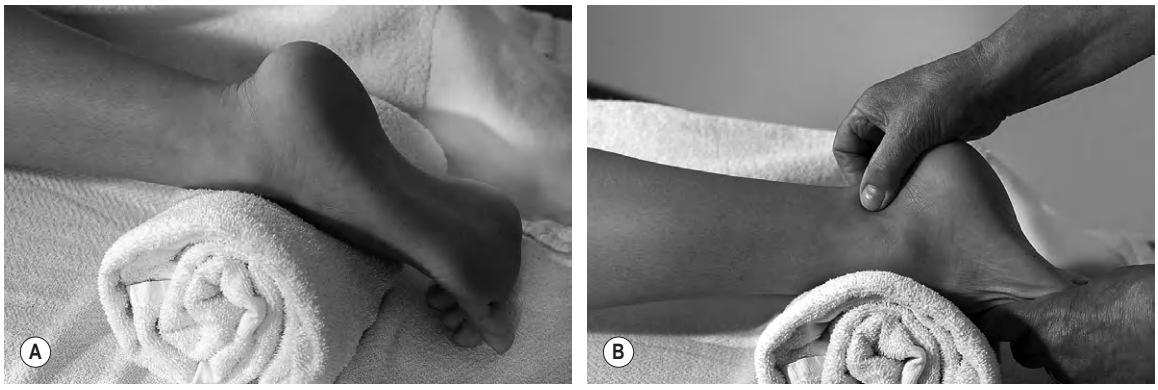


Figure 21.29 (a, b) Frictions for Achilles tendonitis.

for relaxation to be felt will vary from 4–5 minutes to 10 minutes. When the tension releases return to the use of effleurage for the whole upper posterior shoulder area and then turn the patient into supine. In this position start with the two acupressure areas immediately above the inner eyebrows (Figure 21.27c), trigger for 20 seconds on, then 20 seconds off. Four times is usually sufficient. Then, move to the two points at the side of the temple (Figure 21.27d) and repeat the same routine; finally, go to the point in the posterior web of each thumb (Figure 21.27e) and use the 20 seconds on and off again for four repetitions. It is also good to teach the patient to administer these trigger point routines on themselves.

Specific frictions for tennis elbow

Identify the centre of the pain by asking the patient to extend the wrist against resistance. Once located, start with circular friction to accustom the patient to the touch and gradually deepen (Figure 21.28a). Then, change to transverse friction (Figure 21.28b), warn the patient they will experience pain and ensure you are working within their tolerance. After a maximum of two minutes the spot should become numb and the patient may well say the pain has gone. Teach them home-stretching exercises and explain that ice massage may also be of some help. The treatment can be repeated as often as the patient can tolerate but the deep transverse friction may preclude further intervention for 3–4 days.

Friction of tendo-Achilles

Frictions are used very successfully for the treatment of Achilles tendonitis. Start with the foot resting over a rolled-up towel (Figure 21.29a). Isolate the tendon and ensure you are clear of the bursa – use a circular pattern alternating between finger and thumb. Increase depth as the patient can tolerate, but, again, always work within the patient's tolerance (Figure 21.29b). Follow with full-range passive stretch, then assisted stretch and finally into active stretch and eccentric exercises.

Massage for lymphoedema

Reduction of post-mastectomy lymphoedema can be helped by the careful administration of massage. The patient should be seated with the arm in elevation (Figure 21.30a). Start by clearing the distal areas first with short, light effleurage strokes (Figure 21.30b). As you begin to approach the axilla the patient may well tense up as this area can be extremely tender and sensitive. Explain what you are doing and start at the posterior aspect of the upper arm again, using short, no greater than grade 1, effleurage (Figure 21.30c). Slowly work to the lateral, anterior and, finally, medial aspects. Finish with full-arm effleurage deeper distally and easing as you near the drainage areas (Figure 21.30d).

The next steps in massage – how to build on and enhance basic massage skills

More and more the title massage is being changed and the skills described are being referred to as soft tissue therapy. This new title encompasses many additional modalities such as soft tissue release, deep tissue massage, connective tissue massage, aromatherapy, Shiatsu and the Bowen technique. In specifically-applied massage, such as sports massage, techniques including proprioceptive neuromuscular facilitation (PNF) and muscle energy (MET), neuromuscular technique and strain–counter strain are frequently taught. Some of those names refer to the same or very similar applications and this makes it difficult to investigate and get useful research results. There are, however, an increasing number of good research studies available and the use of all massage techniques is increasing, despite it being a one-to-one, time-consuming treatment.

Figure 21.30 (a–d) Massage for lymphoedema.

FINAL THOUGHTS

- We still do not fully or scientifically understand the total effects of touch.
- Be prepared and aware that by releasing, relaxing and easing soft tissue, the practitioner may also cause emotional release. It is not unusual for patients to cry or feel very drowsy after treatment.
- Always have permission to touch and a signed informed consent form. Remember, failure to do so can be construed as assault or even battery.
- Explain exactly which areas you will touch and why.
- Never proceed if you or the patient feel uncomfortable or embarrassed with the procedure.
- Respect the patient's privacy and expose only the areas required. Use any draping so that your hands cannot disappear under it during the performance of any techniques.
- Request the patient's permission to remove or undo any clothing.
- Get the patient to tuck in towels or other drapes themselves.
- Remember to respect all legalities, including child protection and chaperonage.
- Regularly ask if the patient is comfortable and happy with the massage.
- Protect your own back and hands.

FURTHER READING

Andrade, C.-K., Clifford, P., 2000. Outcome Based Massage. Lippincott Williams and Wilkins, Philadelphia.

Benjamin, B., Sohnen-Moe, C., 2004. The Ethics of Touch. Sohnen-Moe Associates, Tucson.

Dryden, T., Moyer, C.A., 2012. Massage Therapy: Integrated Research and Practice. Human Kinetics, Champaign, IL.

Jarmey, C., Tindall, J., 2006. Acupressure for Common Ailments. Gaia Books, London.

Neil-Asher, S., 2005. The Concise Book of Trigger Points. Lotus Publishing, Chichester.

Hollis, M., Jones, E., 2009. Massage for Therapists: A Guide to Soft Tissue Therapy, third ed. Wiley-Blackwell, Oxford.

Johnson, J., 2009. Soft Tissue Release. Human Kinetics, Champaign, IL.

Johnson, J., 2011. Deep Tissue Massage. Human Kinetics, Champaign, IL.

REFERENCES

Cyriax, J., 1993. Text Book of Orthopaedic Medicine, Vol. 2. Balliere and Tindall, London.

Holey, E., Cook, E., 2003. Evidence Based Therapeutic Massage. Churchill Livingstone, Edinburgh.

Hollis, M., Jones, E., 2009. Massage for Therapists: A Guide to Soft Tissue Therapy. Wiley-Blackwell, Oxford.

Travell, J., Simons, D., 1992. Myofascial Pain and Dysfunction, Lippincott Williams and Wilkins, Philadelphia.

Watt, J., 1999. Massage for Sport. Crowood Press, Ramsbury.

Chapter 22

An introduction to fractures

Helen Alsop and the Executive Committee of the Association of Orthopaedic Chartered Physiotherapists

This chapter looks at some basic facts and concepts about fractures but should not be seen as a definitive guide to fracture management. Suggested further reading is included at the end of the chapter.

DEFINITION AND CLASSIFICATIONS

ABC Definition

A fracture is an interruption in the continuity of bone. The terms fracture and break mean the same thing in medicine. The symbol # (hash) represents a fracture.

Classification of fractures

There are numerous different ways of classifying fractures and this will vary depending on country, hospital or consultant preference. Although it is helpful to categorise common fracture types and mechanisms, any bone may break in a variety of ways, so no two fractures will be exactly alike. Obviously, the type of fracture will affect the initial management and treatment.

Fractures may be classified as *open* or *closed* (Figure 22.1). Open or *compound* types of fracture occur when the bone-end or some other object has pierced the skin. These fractures are an additional cause for concern because of the possibility of the introduction of microorganisms, leading to bone infection (osteomyelitis). With closed fractures the skin remains intact. Another common classification includes *displaced* or *un-displaced*.

Figure 22.2 shows some further classifications. *Spiral* fractures commonly occur from a twisting injury. A direct blow could give a *transverse* or *oblique* fracture depending on the angle of the force and whether the limb is fixed or moving at the time of the trauma. Longitudinal forces tend to result in *compression* or *crush* fractures. In some cases there are a number of fragments of bone and this is termed a *'comminuted'* fracture (not to be confused with *'compound'*). Loose fragments of bone are known as 'butterfly fragments'.

A *greenstick* fracture is a type of fracture sustained by young children whose bones are still relatively malleable; therefore, fractures are more likely to present as an

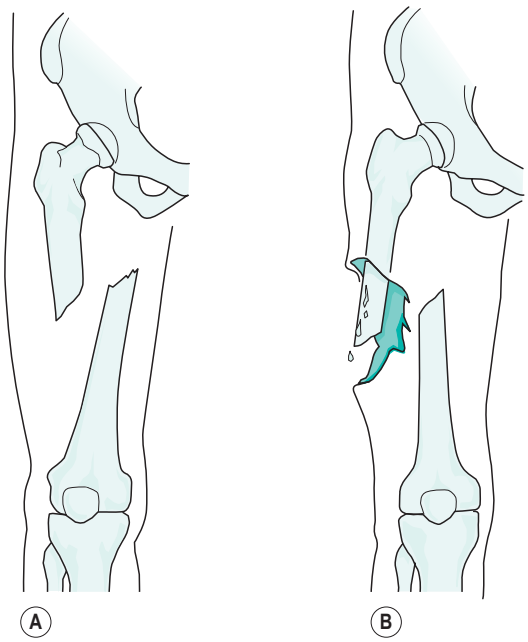


Figure 22.1 (a) Closed fracture. (b) Open or compound fracture. *(Adapted from Dandy and Edwards 1998, with permission.)*

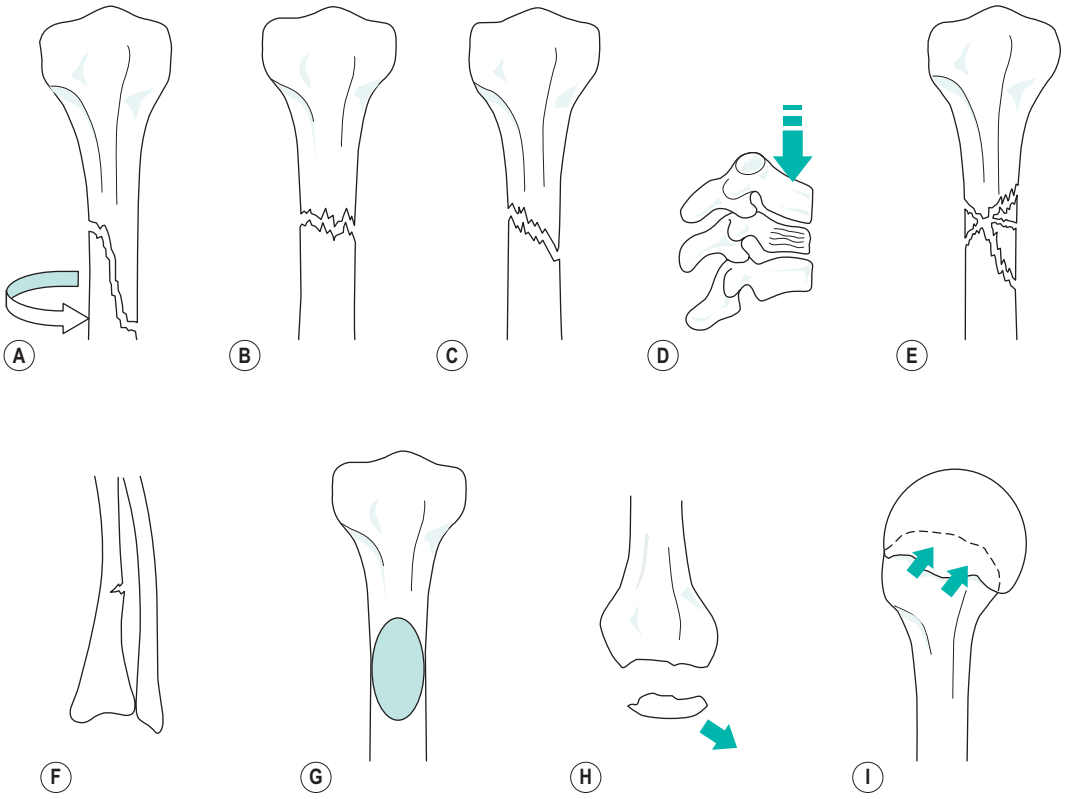


Figure 22.2 Classification: (a) spiral; (b) transverse; (c) oblique; (d) compression; (e) comminuted; (f) greenstick; (g) pathological; (h) avulsion; (i) impacted.

incomplete fracture – a greenstick fracture. (The analogy is attempting to break a green twig, which will bend and split but not snap.) *Avulsion* fractures occur when a bit of bone is pulled off owing to its attachment to soft tissues (e.g. ligaments). Impacted fractures are generally compressed and therefore more stable.

THE CAUSES OF FRACTURES

Trauma

Most fractures are a result of some form of injury. This might be a *direct* blow, a fall from a height or a weight falling onto a part of the body. Other fractures may be caused by *indirect* trauma, such as falling on an outstretched hand leading to the transmission of force up the arm causing a fracture of the clavicle. *Twisting* forces may result in fractures of the tibia and fibula, for example during soccer or skiing when the weight of the body rotates on a fixed foot. *Stress* or *fatigue* fractures are caused by repeated minor trauma, which can occur after walking or running long distances, and often affect the foot metatarsals.

Pathological fractures

These occur as the result of a disease that weakens the composition of the bone itself, making it liable to fracture as the result of a relatively trivial injury. There are a number of such diseases but those most commonly seen clinically are osteoporosis, Paget's disease, carcinoma, osteomyelitis or osteogenesis imperfecta (brittle bone disease).

CLINICAL FEATURES OF FRACTURES

Clinical features vary depending on the cause and nature of the injury, and range from unconsciousness to the patient being able to use the limb, although complaining of pain – such as following fatigue fractures and some impacted or crack fractures. Most will be diagnosed by

X-ray. Some fractures, for example fractures of the scaphoid bone, are sometimes not detected upon initial X-ray and can be misdiagnosed as wrist sprain. The clinical features of fractures are summarised below.

Pain

This may be immediate from the local inflammatory reaction and trauma, but the cause may not be obvious in some cases. There will be marked tenderness around the site of the fracture. Once reduced, a fracture is remarkably painless.

Deformity

This is noticeable when there is displacement of the bone fragments. Some fractures exhibit classical deformities, for example the 'dinner fork' deformity which occurs following a Colles' fracture of the distal radius, caused by displacement of the distal fragment; and also shortening of the leg with a fractured neck of femur.

Oedema

This is localised immediately after the injury and becomes more extensive with time. It may be necessary to apply a temporary cast or splint and then reapply the plaster once the swelling has subsided.

Muscle spasm

Muscle spasm is an attempt by the body to stop things from moving. It often affects powerful muscle groups, such as the quadriceps, and may cause displacement or overriding of the bone ends. Traction may be needed to counteract this.

Abnormal movement/crepitus

There may be grating between the broken ends of the bone. Do not deliberately attempt to elicit this, though, because that might result in further damage.

Loss of function

This may be complete following severe fractures but some activity may be possible when the injury is less severe, such as stress, impacted or crack fractures. Sometimes normal function can be regained very quickly with appropriate assessment, advice and treatment, whereas in other cases there are a number of problems for which more intensive treatment may be required. Modern orthopaedics is now geared towards early mobilisation with minimal surgical trauma, and physiotherapy needs to complement this.

Shock

Hypovolaemic shock is a possibility following fractures. A fractured shaft of femur may haemorrhage as much as three pints (1.7 litres).

Limitation of joint movement

Joint mobility can be affected by many factors: adhesion formation, tight muscles, pain, spasm, fear, mechanical obstruction or swelling. Movement may also be limited because of weak muscles, in which case it will be possible to move the joint passively through total range. If the fracture involves the articular surface of the joint this may also cause limitation of movement and future cartilage degeneration. For this reason, certain fractures are now treated aggressively with almost immediate movement (aggressive in this context meaning soon, not rough).

Muscle atrophy

There will be a loss of strength in disused muscle groups.

FRACTURE HEALING

Healing of compact bone

Bone has the incredible ability to replace itself with new bone, not scar tissue. Healing starts within seconds of a fracture being sustained and will still be ongoing years later – this makes ascribing a healing timescale difficult.

Wolff's law states that bone responds to the stresses that are imposed upon it by rearranging its internal architecture to best withstand the stresses. In other words, bone is laid down where it is needed and absorbed where it is not. It is important to understand this concept when dealing with people who have sustained fractures. Bone is a living tissue, not the brittle, chalky specimens that students may be familiar with. It is continually in a dynamic equilibrium of growth and reabsorption. Figure 22.3 shows the process of fracture healing in compact bone taken through five stages.

Haematoma

As a result of the tearing of blood vessels within seconds of the injury, a haematoma forms at the fracture site. Very small portions of bone immediately adjacent to the fracture die and are gradually absorbed.

Periosteal and endosteal proliferation

There is a proliferation of cells from the deep surface of the periosteum adjacent to the fracture site. These cells are precursors of the osteoblasts and form around each

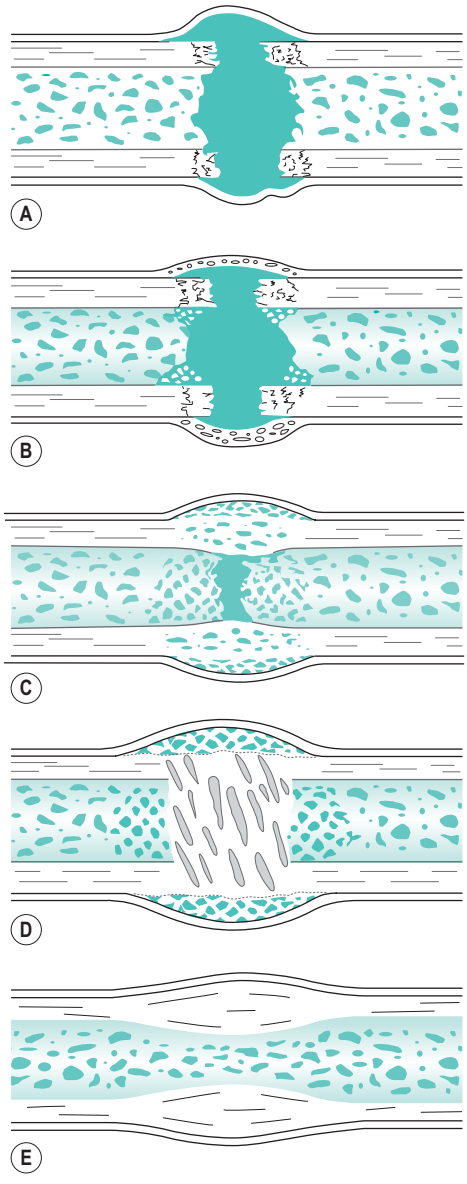


Figure 22.3 Fracture healing: (a) haematoma; (b) periosteal and endosteal proliferation; (c) callus formation; (d) consolidation; (e) remodelling.

fragment of bone. At the same time cells proliferate from the endosteum in each fragment and this tissue gradually forms a bridge between the bone ends. During this stage the haematoma is gradually reabsorbed.

Callus formation

The proliferating cells mature as osteoblasts or, in some instances, as chondroblasts. The chondroblasts form cartilage and are found in varying amounts at a fracture site. Osteoblasts lay down an intercellular matrix of collagen and polysaccharide which then becomes impregnated with calcium salts forming the immature bone called callus or woven bone. This is visible on X-ray and gives evidence that healing is taking place.

Consolidation

Osteoblastic activity results in the change of primary callus to bone, which has a lamellar structure, and at the end of this stage union is complete. New bone forms a thickened mass at the fracture site and obliterates the medullary cavity. The amount of this new bone varies for a number of reasons. It tends to be more extensive if there has been a large haematoma or it has been impossible to obtain exact apposition of the bone fragments.

Remodelling

The lamellar structure is changed and the bone adapts by strengthening along the lines of stress imposed upon it. The surplus bone formed during healing is gradually removed and eventually the bone structure appears very similar to the original. In children, healing is usually very good and it is difficult to see the fracture site on a radiograph. In adults there may be a permanent area of thickening, which might be felt or seen, in a superficial bone.

Healing of cancellous bone

This follows a different pattern from that described above. As with compact bone, a haematoma will form, but as there is no medullary cavity, the second stage differs. Cancellous bone has a greater area of contact between the fragments of bone and penetration of the bone-forming tissue is facilitated by the open arrangement of trabeculae as it grows out from both fragments. Osteogenic cells lay down intercellular matrix, which calcifies to form woven bone. The process of remodelling then continues to form the cancellous bone.

When is a fracture healed?

One of the most common questions asked by patients is 'When will my fracture be healed?' Unfortunately, the answer to this question is not always straightforward and depends upon many factors, including the type of bone fractured, the type of fracture sustained, the age of the person, the treatment undergone and the nutritional status of the person.

The current mainstay for evaluating when a fracture is healed is still based upon a combination of clinical judgement, X-ray evaluation and historical knowledge on specific fracture behaviours. A fracture is considered to be clinically healed based upon the combination of physical findings and symptoms over time.

The following suggest complete healing:

- absence of pain on weight-bearing, lifting or movement;
- no tenderness on palpation at the fracture site;
- blurring or disappearance of the fracture line on X-ray;
- full or near full functional ability (Hoppenfeld and Urthy 1999).

Time for a fracture to unite

The time it takes for a fracture to unite depends on a number of factors.

- *Type of bone*. Cancellous bone heals more quickly than compact bone. Healing of long bones depends on their size so that bones of the upper limb unite earlier (3–12 weeks) than do those of the lower limb (12–18 weeks).
- *Revascularisation* of devitalised bone and soft tissues adjacent to the fracture site.
- The *mechanical environment* of the fracture (Marsh and Li 1999).
- *Classification of the fracture*. It is easier to obtain good apposition of bone ends with some fractures than with others. This may depend on the initial position of the fragments before reduction and the effect of muscle pull on the fragments.
- *Blood supply*. Adequate blood supply is essential for normal healing to take place. Certain fractures can be notoriously slow to heal (e.g. fractures of the lower third of tibia). This part of the bone has a poor blood supply owing to the fact that under normal circumstances it does not require one as there is little muscle bulk here, therefore little demand for nutrients and oxygen.
- *Fixation*. Adequate fixation prevents impairment of the blood supply which may be caused by movement of the fragments. It also maintains the reduction thus preventing deformity and consequent loss of function. Interestingly, if a fracture is rigidly immobilised, the stimulus for callus to form is lost, so a small amount of movement at a fracture site actually encourages fracture healing.
- *Age*. Union of a fracture is quicker in children and consolidation may occur at between 4 and 6 weeks. Age makes little difference to union in adults unless there is accompanying pathology.
- It has been suggested that certain *drugs* such as non-steroidal anti-inflammatory drugs may interfere with fracture healing; however, evidence remains inconclusive (Bandolier 2004).
- *Smoking*. There are increased rates of delayed union and non-union in people who smoke who have sustained open tibial fractures (Adams et al. 2001).
- *Ultrasound*. Recent work has suggested that low-intensity ultrasound may accelerate fracture healing (Azuma et al. 2001).

COMPLICATIONS OF FRACTURES

Critical blood disorders

Pulmonary embolism and *deep vein thrombosis* are two possible complications of a fracture. *Shock* may be caused by hypovolaemia or loss of blood. Femoral shaft fractures may bleed as much as three pints (1.7 litres) and pelvic fractures may lose six pints (22.4 litres). Clinical signs of this are tachycardia (rapid heart rate), pallor from reduced peripheral perfusion, hypoxia (decreased oxygen saturation), confusion, and a state of semi-consciousness.

Infection and *tetanus* are threats, especially following open or compound fractures. Most people are now immunised against tetanus or given booster tetanus injections if they have a large open wound. Bone infection (osteomyelitis) can be stubborn to respond to treatment.

Fat embolism (acute respiratory distress syndrome)

If a person sustains multiple fractures of large bones or crushing injuries, or if large amounts of marrow become exposed, there may be leakage of microscopic fat globules into the circulatory system. These may become trapped in the lungs. Symptoms include respiratory distress, shortness of breath, drowsiness, a decrease in saturation of oxygen levels and petechiae (tiny haemorrhages which appear on the chest). Acute respiratory distress syndrome (ARDS) is potentially fatal.

Skin plaster sores

Reassure the patient that large amounts of dry flaky skin following removal of plaster is normal. Reddened areas or sores caused by plaster or splints must be reported to the relevant team member.

Muscle damage and atrophy

Muscle fibres may be torn, crushed or ruptured as a result of the injury and this will cause additional bleeding and swelling. Tendons may be severed, particularly in the case of open fractures, or sometimes there may be a rupture following a fracture. Surgical intervention is usually necessary to repair a rupture.

Compartment syndrome

If muscles become damaged or inflamed at the time of injury, and intramuscular pressure builds up with no

means of release, death (necrosis) of the tissues from ischaemia (lack of blood supply) may result. It is defined as the condition in which high pressure within a closed fascial sheath reduces capillary blood perfusion below the level necessary for tissue viability. Compartment syndrome is seen most commonly in the anterior tibial muscles or forearm muscles.

Clinical signs of a limb with compartment syndrome are the five P's:

- pale;
- painful;
- pulseless;
- paraesthesia;
- paralysed.

Treatment revolves primarily around accurate diagnosis. Check colour, sensation and movement after any injury or surgery, elevate and cool the limb. Surgical decompression (fasciotomy) may be necessary as an emergency procedure.

Avascular necrosis

Bone receives its blood supply by the soft tissue structures attached to it or by intra-osseous vessels. In certain instances one part of the bone is very dependent on the intra-osseous (within the bone) vessels for its blood supply and if this is interrupted because of a fracture, avascular necrosis may occur (part of the fractured bone may die). It can occur in fractures of the neck of femur leading to avascular necrosis of the head, and in fractures of the scaphoid bone where the proximal pole may be affected. This may be a cause of non-union of the fracture and as the fragment usually includes an articular surface it can lead to osteoarthritis.

Problems with union

Delayed union may occur if the gap between the bone ends is too big, the blood supply is poor (lower third of the tibia), the area is infected or if internal fixation is used (this sometimes removes the stimulus for callus formation).

There may be distinct pathological changes and radiological evidence of *non-union*. There appears to be no callus formation and the fractured ends of bone become dense and the outline clear-cut. The gap between the bone fragments may be filled with fibrous tissue and form a pseudo-arthrosis. The lower third of the tibia has notoriously poor healing capabilities, even occasionally in the young and healthy.

A fracture may heal in a less than perfect position – *malunion*. Overlapping of the fragments could lead to shortening and this would affect function. Angulation or rotation of the fragments may impair function because of the resulting altered biomechanics.

Growth disturbance

In younger people there may be growth disturbance if the fracture includes the epiphysis (growth plate).

Complex regional pain syndrome I (CRPS I)

The term *complex regional pain syndrome* (also sometimes known as Sudeck's atrophy, reflex sympathetic dystrophy (RSD), algodystrophy or causalgia) is now being used to describe these pathological states. Fortunately it is a rare complication. Patients complain of severe pain at rest, as well as on movement, out of proportion to the initial injury. The limb is swollen and the skin appears shiny and discoloured, and feels cold. In extreme cases this may lead to the limb becoming exquisitely tender and discoloured. Osteoporosis and permanent contractures may follow.

Management is difficult. When the lower limb is involved, weight-bearing is encouraged with the help of physiotherapy and pain control aids, such as transcutaneous electrical nerve stimulation (TENS). However, this is minor therapy compared with the use of other treatments such as sympathetic nerve blocks (Viel et al. 1999), vasodilator drugs (e.g. guanethedine) and local analgesia. All of them have variable results. Recovery is slow and may take several months.

Intra-articular fractures

Fractures involving the articular cartilage predispose the joint to osteoarthrosis in the future (e.g. fractures of the tibial plateau). This is owing to the area of roughness that inevitably results after a fracture and also because the immobilisation of the fracture results in cartilage death (see below). For the latter reason, some fractures are now treated aggressively by physiotherapists from an early stage.

Another problem with intra-articular fractures is that if callus is attempting to form within a joint cavity, it is constantly being washed away by synovial fluid.

Visceral injuries

A fractured pelvis may damage the bladder or urethra. A fractured rib may cause a pneumothorax. A skull fracture may cause brain injury. These are just three examples.

Adhesions

These may be within the joint (intra-articular) or around the joint (peri-articular). Adhesions are the price paid for immobilising a fracture. Intra-articular adhesions may occur when the fracture extends into the joint surface and there is a haemarthrosis, or bleeding within a joint cavity. If this is not absorbed, fibrous adhesions may form within

the synovial membrane. Peri-articular adhesions may occur if oedema is not reduced and is allowed to organise in the surrounding tissues. This leads to adhesion formation between tissues such as the capsule and ligaments, and results in joint stiffness, which is less of a problem now that new techniques of fixation allowing early mobilisation have been developed.

Capsular adhesions are common, for example in the capsule of the shoulder joint which possesses dependent folds on its inferior aspect to permit the huge range of motion at this joint. These may stick together after fracture or injury causing limitation of movement.

Injury to large vessels

If a large artery is occluded in such a position as to cut off the blood supply to the limb, this may lead to gangrene or, if there is partial occlusion, an ischaemic contracture may develop. These injuries must be dealt with as an emergency by the surgical team.

Thrombosis of veins may occur in the area of the fracture. This presents as a sudden development of a cramp-like pain in the part, by an increase of swelling and by marked tenderness along the line of the vein. Anything that appears to be abnormal in the circulatory system must be reported to the surgeon immediately. Blood vessels may sustain damage, for example following supracondylar humeral fractures the brachial artery may be damaged.

Nerve injury

Certain fractures can lead to nerve palsy (e.g. radial nerve injury in mid-shaft fractures or the common peroneal nerve when a plaster is applied too tightly to the lower limb). Functional bracing (e.g. ankle foot orthosis or AFO) can be helpful while the nerve recovers. Sometimes bracing has to be used long-term if the nerve does not recover.

Oedema

Oedema may be apparent below the level of the plaster and it is often necessary to elevate the limb, exercise the fingers or toes not encased in plaster, and perform isometric contractions of the muscles within the cast in an attempt to encourage muscle pump activity (Tschakovsky et al. 1996; Sheriff and Van Bibber 1998). Once the plaster has been removed, atrophied muscles may not provide an adequate muscle pump on the veins, in which case swelling may reappear especially after activity or non-elevation.

PRINCIPLES OF FRACTURE MANAGEMENT

Once a fracture has been diagnosed, the most suitable treatment must be decided upon. This should be the minimum possible intervention that will safely and effectively provide the right environment for healing of the fracture. Interestingly, nature has devised a system by which a slight amount of movement at a fracture site is useful in stimulating callus formation so there is a balance to be made between immobilising a fracture but allowing enough movement to stimulate callus formation and healing (Figure 22.4; Cornell and Lane 1992).

This is a common dilemma in orthopaedics. In the same way that there is no recipe for the physiotherapy treatment of a fracture, there is no single recipe for the surgical management of fractures. This 'see-saw' will be referred to in the case study presented later in this chapter.

Reduction

Reduction means to realign into the normal position or as near to the normal anatomical position as possible (Figure 22.5). Reduction of a fracture may be either open or closed. *Closed reduction* means that no surgical intervention is used with the fracture being manipulated by hand under local or general anaesthesia. *Open reduction* means that the area has been surgically opened and reduced.

Reduction may not always be necessary, even when there is some displacement. For example, fractures of the clavicle may heal with a bump which may be a problem only in the cosmetic sense; function is the most important end-point.

However, when there is poor alignment of the fragments or the relative positions of the joints above and below the fracture are lost as a result of angulation or rotation of the bone ends, or if there is loss of leg length, then accurate

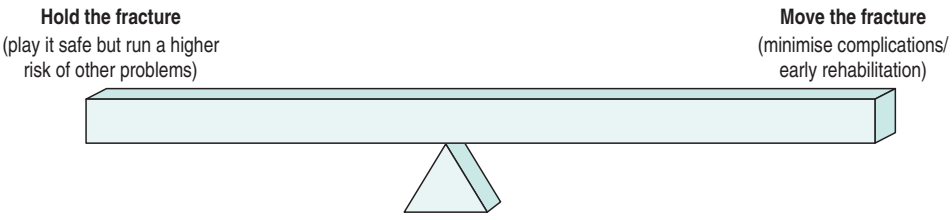


Figure 22.4 The mobilisation see-saw.

Figure 22.5 Reduction and healing of a fractured shaft of tibia showing the developing callus formation. *(With thanks to Martin George, Superintendent Radiographer; Sue Evans, Senior Radiographer; Lynn Porter, Sonographer; Southport and Ormskirk Hospitals, UK.)*

anatomical reduction is necessary. X-rays are used to ascertain the exact position of the fragments before and after reduction. Real-time X-rays can now be taken using image intensifiers so that the surgeon can more accurately reduce. Improvements in computed tomography (CT) and magnetic resonance imaging (MRI) scanning mean that complex fractures can be studied in great detail preoperatively, which assists the planning of surgery.

Immobilisation

The objectives of immobilising a fracture are:

- to maintain the reduction;
- to provide the optimal healing environment for the fracture;
- to relieve pain.

In some fractures where there is no likelihood of displacement, fixation may not be necessary or minimal fixation will suffice, for example buddy strapping for some finger fractures (Figure 22.6).

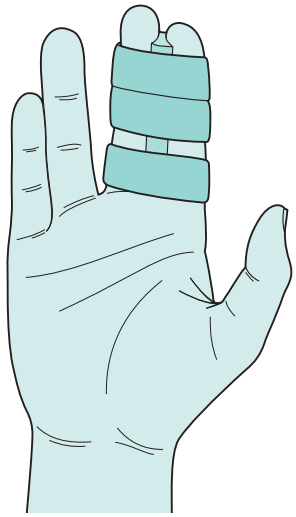


Figure 22.6 Double buddy strapping applied to both sound and damaged fingers to assist movement of the latter.

Common methods of fracture immobilisation

Plaster of Paris

This is a plaster-impregnated bandage that can be moulded to the part when wet, which sets in time. The standard method of external splinting is still plaster of Paris (Figure 22.7).

Synthetic materials are now used for splinting some fractures because of their light weight and waterproof qualities. Custom-made lightweight thermoplastics can be

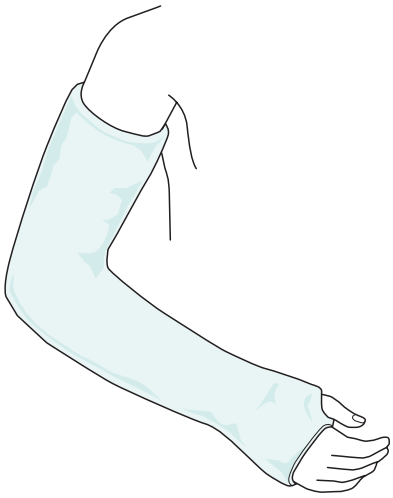


Figure 22.7 Plaster of Paris immobilisation.

moulded to the limb and remoulded if swelling or atrophy cause changes in the limb contour. Some synthetic casting materials, however, are less malleable and cannot be moulded as effectively as plaster of Paris and they can occasionally cause allergies.

A plaster saw is needed to remove a cast. This special tool has an oscillating blade that will cut through the hard cast without damaging the skin.

The advantages and disadvantages of using plaster of Paris are listed in Table 22.1.

Table 22.1 The advantages and disadvantages of plaster of Paris

Advantages	Disadvantages
• No surgery or its complications • No infection risk • Quick to apply • Rapid patient discharge • Cheap, relatively easy to apply with training • New lightweight casts are an alternative • Radio translucent (bones can be X-rayed through the cast) • May absorb fluids or bleeding. The extent of bleeding can be traced on the cast itself and monitored daily • Can be moulded for several minutes before hardening	• It may not be possible to reduce the fracture correctly or maintain reduction • May require surgery at a later date • Plaster needs removal/ or windowing (removal of a piece of the cast) to inspect the skin • May need removal in case of increased swelling or reapplication once swelling has subsided • Bad odour if it gets wet • Heavy • May crack • May rub the skin and cause sores

Clinical note

Medical advice should be sought if any of the following occur to a limb that is in a plaster of Paris or similar splint:

- pale or blue coloration of the skin on the injured part;
- numbness, tingling or throbbing of the injured part;
- inability to move the fingers or toes;
- excessive pain in the injured part;
- swelling, bulging or puffiness around the edges of the cast;
- a foul smell from under the cast;
- if it becomes loose and slides around.

Functional bracing (cast bracing)

It has been found unnecessary to fix some fractures as rigidly as was thought necessary in the past. An example of this is cast (or functional) bracing. Functional braces have hinges to allow movement (see the case study towards the end of the chapter and Figure 22.24).

The soft tissues of the limb squeeze against the inside of the brace and, in conjunction with the use of a heel cup, permit weight to be taken through the substance of the brace. This has reduced many of the problems that were seen as a direct result of prolonged immobilisation. Another benefit of allowing movement of joints, provided that it does not unduly stress the fracture site, is that it may promote union by improving the area's blood supply.

Internal fixation

Surgical intervention by applying a plate and screws to the fracture is known as *open reduction and internal fixation*, often abbreviated to ORIF (Figure 22.8).

Advantages of ORIF

It permits a detailed inspection and accurate surgical assessment of the site of injury and procedure to be undertaken.

Disadvantages of ORIF

- Surgery inevitably causes additional trauma and potential exposure to microorganisms.
- It can convert a closed fracture into an open fracture.
- It requires surgery with all its sequelae and potential complications. Ironically, rigid fixation may remove the stimulus for callus formation. The implants are usually left in unless they cause problems e.g. irritation, infection or protrusion. In the young, they will be removed if they will affect growth of the bone.

Figure 22.8 Fracture of the tibia/fibula fixed by plate and screws.

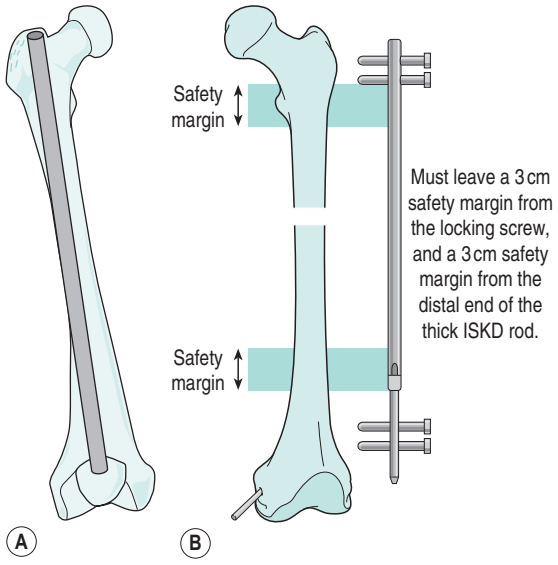


Figure 22.9 Intramedullary nailing. (a) ISKD nail. (b) Intramedullary skeletal kinetic distractor (ISKD) *(With thanks to Dania Sorio, Customer Service International, Orthofix Srl and Grit Soboll, Communications Manager, Orthofix Srl, www.orthofix.it.)*

Intramedullary nailing

Here, a hollow metal rod is introduced at one end of a long bone, travels down the medullary canal and may be locked with screws distally and proximally (Figure 22.9). The proximal aspect of the nail is threaded and this permits a tool to be threaded onto the nail at a later date for its removal.

Intramedullary nailing for fractures of long bones has revolutionised the management of many fractures, which, historically, would have been managed by prolonged bed rest. The trauma is less than with open techniques and results in a shorter hospital stay, more rapid patient mobilisation and rehabilitation with minimal risk of complications associated with immobility. The implant, rather than the bone, may take stresses and strains, and, for this reason, the surgeon may choose to remove the locking screws at a later stage. This permits the nail to move slightly and cause compaction of bone ends (dynamisation). It allows the bone to once again take its normal stresses and strains and adapt in accordance with Wolff's law. The endosteal proliferation that occurs as part of the normal fracture healing process may be lost with certain types of internal fixation. Fractures of the shaft of tibia and humerus may also be nailed in this way.

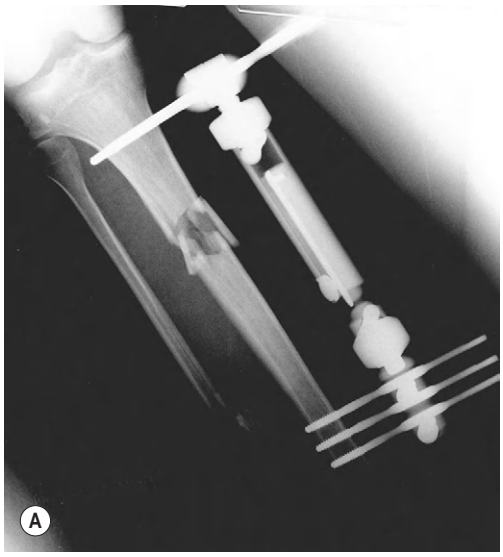
External fixation

Figure 22.10 shows fixation of a fractured tibia using an external fixator. Pins or wires are driven into the fragments and held by a piece of apparatus on the outside of the body. Figure 22.11 shows an external fixator for a comminuted intra-articular fracture of the distal radius. Figure 22.12 shows an external fixator for an unstable pelvic fracture.

The advantages and disadvantages of external fixation are listed in Table 22.2.

The intramedullary skeletal kinetic distractor

The intramedullary skeletal kinetic distractor (ISKD) is a two-part metal rod that can be used for tibial or femoral lengthening. An osteotomy is performed to allow distraction to take place. The device can only rotate in one direction and requires the patient to perform a rotational movement to cause lengthening. A hand-held monitor that contains a magnetic sensor is able to detect a small magnet sealed inside the ISKD. As the ISKD lengthens, the magnet rotates through 360 degrees. The monitor detects these changes and is able to record measurements so that lengthening can be very accurately controlled. Patients take measurements regularly throughout the day. The duration of lengthening depends on how much length needs to be achieved. The average desired rate is usually 1 mm per day. Patients are generally non-weight-bearing during the distraction phase.



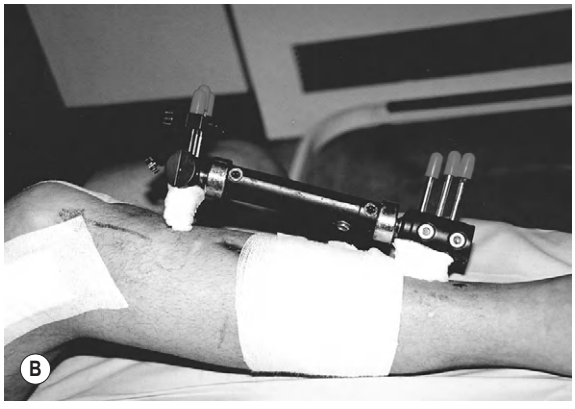


Figure 22.10 Fixation of a fractured tibia using an external fixator. Pins or wires are driven into the fragments and held by a piece of apparatus on the outside of the body, as shown in the external view. *(Reproduced by kind permission of Mr R.F. Adam.)*

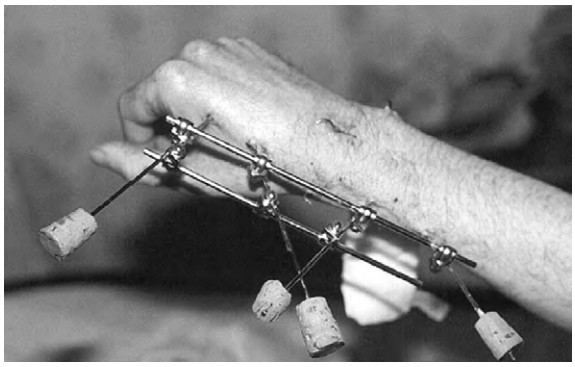


Figure 22.11 Fixation of a comminuted intra-articular fracture of the distal radius. *(Photo reproduced by kind permission of Ms Fiona Cobbold ©.)*

Figure 22.12 External fixator for an unstable pelvic fracture.

The Ilizarov method

The Ilizarov method of fracture fixation originated in Russia in the 1940s. It incorporates an axial system of wires or pins fitted through the bone and connected to a circular ring. It has proved successful in cases of non-union (Schwartsman et al. 1990). This method also incorporates the principle of 'distraction osteogenesis', and can be used in the restoration of large skeletal defects, limb lengthening and the correction of skeletal deformities (Figure 22.13).

Figure 22.13 Ilizarov fixation for fractured humerus and scapula.

Table 22.2 Advantages and disadvantages of external fixation

Advantages	Disadvantages
• Minimal disruption to the fracture site • Enables inspection of the wound and fracture • Can be adjusted with minimal trauma • Can be used for limb lengthening procedures • Can be used to pin multiple fragments (e.g. comminuted fractures) • Allows preservation of tissues in open or compound fractures, de-gloving injuries or burns	• Infection risk at pin sites • Needs meticulous wound care • Cosmetically ugly • Functional impairment (e.g. adjacent joints may be restricted or soft tissues pierced by fixator) • Anaesthetic risk and its associated complications • Patient will need several days in hospital • Stresses taken by implant, so decreased stimulus for callus formation • Heavy
Statement	**Consequence for the physiotherapist**
No two orthopaedic patients are alike	Do not ask for a 'treatment recipe'. Your approach should be flexible and dynamic and will change as a result of many factors
No two assessments are alike	Learn the basic assessment framework but tailor your assessment slightly to each individual
No two treatment courses are alike. Patients do not always do what the textbook says!	Keep an open mind, recognise when a treatment is not working and change or modify it
No single assessment can predict the outcome of the problem	Experienced physiotherapists are able to 'assess as they treat'. This means that the patient is continually receiving the most appropriate attention and the situation is dynamic. Treatment goals may need modification and should not be totally inflexible

Skeletal traction

In skeletal traction a Steinman pin is inserted through the bone and a weight system attached to allow localised, effective traction. Common sites for this are the tibial plateau or the calcaneum. Pin sites must be kept clean and free of infection. These pins are usually tolerated well and are not as painful as they appear.

Traction

Figure 22.14 shows an example of skin traction. The traction force is applied through the skin instead of through the bone.

Traction is the application of a pulling force to a part of the body; it may be either a direct or an indirect pull. Traction is less common on the orthopaedic ward nowadays, although it still has its place. Uses include:

- restoring bone or limb length if it has been reduced by fracture or disease;
- maintaining correct limb length and overcoming muscle spasm, which may be the cause of limb shortening after a fracture;
- to correct deformity in a joint;
- to reduce a dislocated joint;
- to immobilise a joint;
- to relieve pain pre-operatively;
- to promote rest and healing postoperatively.

PHYSIOTHERAPY AND FRACTURES

General issues

The physiotherapist's role is to identify the cause of the problem and to select the appropriate procedure to alleviate or eliminate the cause of the loss of movement – 'the right tool for the right job'. For example, there is little point in using accessory joint mobilisations if muscle spasm is the limiting factor and a hot pack would not be appropriate if there were a bony block to movement.

Assessment of each individual will dictate the rehabilitation programme – there is no standard 'recipe' for the

Figure 22.14 Skin traction – this woman sustained a fractured shaft of femur and, owing to her general health, surgery was not indicated. She was managed conservatively. *(With thanks to K. McGregor.)*

treatment of fractures. Any exercises given to a person must be realistic, attainable, adaptable, functional and memorable, as patients often become confused about their exercises.

Most orthopaedic units have in place standardised protocols/guidelines and postoperative care plans for particular surgical or orthopaedic interventions, and the physiotherapist should adhere to these. Providing the best possible treatment to orthopaedic patients will take many years of practice, reflection and fine-tuning of clinical reasoning skills.

Clinical reasoning is not an abstract concept, it is applying common sense to your knowledge base. Your knowledge will improve every time you assess a patient and evolves with experience. The following section gives pointers about assessing patients who have sustained fractures.

How physiotherapists assess and treat fractures depends very much on the time elapsed since the fracture, and the stage of rehabilitation at which they are performing the assessment.

For example, students are often dismayed on their clinical placement to find that they are unable to perform a complete assessment of a patient who has just been put into plaster of Paris. Their treatment plan may now consist of:

- attaining safe non-weight-bearing on elbow crutches;
- negotiating stairs safely;
- planning for safe discharge home;
- advising on isometric exercises.

Initial patient assessment

An assessment is essential if you are to plan out a safe and appropriate treatment. Assess a fracture like any other condition, but be aware of any specific instructions or limitations. For example, is the patient allowed to fully or partially bear weight? Take time and think about what you are doing. If you omit something, make a note and remember to follow it up next time. Your assessment will vary considerably depending on the clinical setting you are in.

The problem-oriented medical record

The problem-oriented medical record (POMR) system is based on a data collection system that incorporates the acronym SOAP:

- *S*ubjective – any information given to you by the patient: allergies, past medical history, past surgical history, family history, social history (living arrangements, social conditions, employment, medication), review of systems;
- *O*bjective – all information obtained through observation or testing, e.g. range of joint movement, muscle strength;
- *A*nalysis – a listing of problems based on what you know from a review of subjective and objective data. For patients with multiple problems, number and list each problem consecutively, with the most important listed first;
- *P*lan – as the name implies this refers to the plan of forthcoming treatment.

Key point

Documentation is developing and many hospitals now use multi-disciplinary integrated care pathways (ICPs) or electronic health records (EHRs). The documentation of your clinical reasoning and adherence to both your local and Chartered Society of Physiotherapy Core Standards of Practice is paramount.

The subjective assessment

Basic background information to record

- Occupation.
- Drug history.
- X-rays/scans/other tests.
- Family history.
- Date of next clinic appointment.
- Specific surgical instructions.

History of present condition

Include the date of onset, mode of onset, course and treatment to date. Note specific instructions, for example partial weight-bearing for the next three weeks. If the patient does not know his or her own postoperative instructions, do not guess. Speak to the other team members to establish treatment parameters.

Previous medical history

Are there any warning signs or findings that might affect your treatment options?

For example:

- a person with internal fixation in place would not be considered for certain electrotherapy treatments;
- a patient with advanced osteoporosis would not be considered for high-impact gym work;
- diabetic patients may have neurovascular complications that impact on the skin and healing;
- patients with a history of chronic obstructive pulmonary disease (COPD) may have limited mobility and exercise tolerance.

Social history

Do not underestimate the importance of asking the following questions:

- Is the patient living alone?
- Does the person need to go up stairs?
- Is the person losing money through not working?
- What are the person's hobbies?
- Does the person care for sick relatives?
- What does that person need to be able to do to be 'normal'?

The most effective physiotherapists are able to listen to what the patient tells them and incorporate this into the treatment plan. Do not ask leading or multiple questions, but keep your questioning on track and relevant and do not lose sight of why you are there. Set long- and short-term goals as you would with any other patient, but be prepared to adapt them if necessary.

Pain

It is not sufficient to ask merely whether or not the patient has pain. Ask the person about the location, type, duration and radiation of the pain. Is it related to time of day or certain activities, and does it have alleviating or aggravating factors? Visual analogue scales may be used as an attempt to quantify pain (see Chapter 11). Remember that dull aching of the fracture site may be considered normal, especially after activity. This may signify bone remodelling. Sharp pain of a prolonged nature is more cause for concern – hence the need to understand the physiology underlying healing.

The objective assessment

From the subjective assessment you should be able to devise a plan for your objective testing. The symptoms will act as a guide to the structures that will be examined: articular, muscular, vascular or neural. The painful areas should all be assessed, including areas other than the fracture site with associated soft tissue injuries. The tests used will be adapted to the stage in the healing process and the individual. By remembering the fundamental principles of fracture treatment (reduction, immobilisation and preservation of function), the assessment can be completed effectively and without damage. With an unstable fracture, for example, muscle contraction across the site could cause further damage. Fixators immobilise a fracture but are not as stable as union and should be treated with care.

Convenient sub headings for your assessment are: look, feel and move (Table 22.3).

Look

The observational skills you develop over time will be very important. A physiotherapist is often the first to pull back the bed covers to find the overnight changes. The changes in swelling, bruising, deformity, spasm, oedema and atrophy will indicate the progression of the pathology. Leg length discrepancies, posture alignment and gait patterns can be seen and then converted into objective measurements.

Table 22.3 Objective examination

Look	Feel	Move
Swelling Spasm Deformity Bruising Oedema Atrophy	Swelling Heat Sensation Tenderness on palpation Spasm	Active first Then passive Then overpressure (care – this may be inadvisable depending upon the stage of fracture healing) What is the quality and amount of movement and what is the end-feel?

Feel

The palpation of swelling, pulses, heat, spasm and tenderness may be the main substance of your objective assessment in the acute unstable fracture. They are also essential for the detection of complications. Sensation changes will indicate the extent of your neural testing.

Move

This section is used to design your exercise programme for the preservation of function. A patient will be fearful of movement and will rely on you to indicate what, and how, to get moving. Therefore, it is essential to test joints above and below the site to establish for the patient the limitations of their condition. Local to the acute unstable fracture passive or assisted active movements would be less pain and harmful. However, in later stages of the pathology, active, passive and then overpressure testing is required. What is the quality and amount of movement and what is the end-feel? In answering these questions you will be able to diagnose the nature of the damage.

The stage of the fracture healing will dictate the extent of strength testing. Muscles that do not cross the fracture site but move the limb should be assessed. Their role as an agonist, antagonist, synergist or fixator will influence the choice of test and the maintenance exercises prescribed.

Functional movement tests are essential to tailor your treatment to the individual; their abilities may be influenced by their injury, age or comorbidities. By measuring their ability to move about the bed, to get in and out of bed, to maintain weight-bearing status and do stairs will indicate discharge suitability. In the later stages the tests may relate more to occupational or leisure needs.

The assessment allows you to pull together the subjective and objective findings to form a clinical diagnosis. You may need to discuss this with your supervisor initially to help clarify your reasoning, aid in goal-setting and the treatment planning. To complete the planning stage it is necessary to inform the patient of your findings and agree the treatment plan.

Setting goals for orthopaedic patients

Without goals we have nothing to measure our performance against. This simple summary should help you plan goals for patients. When you set goals for any patient (orthopaedic or otherwise) the goals need to be SMART:

- *S*pecific
- *M*easurable
- *A*chievable
- *R*ealistic
- *T*imely.

Typical examples of orthopaedic goals possessing all the SMART characteristics are:

1. Goal – Mr X will be able to safely negotiate stairs, partial weight-bearing with two elbow crutches in four days' time.
2. Goal – Mrs Y will have attained 50 degrees of active knee flexion by one week from today.
3. Goal – Mrs Z will be able to transfer safely from bed to chair within two days.

Test yourself

Each of these goals fails to achieve one or more of the SMART criteria. Why?

1. Mr X will be totally pain free within one day of sustaining his fractured femur, tibia and humerus.
2. Mr X will be able to walk in eight months' time.
3. Mr X will be much better in one week.
4. Mr X will mobilise full weight-bearing on the unstable fracture within one week.
5. Mr X will have more knee flexion within one week.

Answers

(1) Not realistic. It is extremely unlikely that Mr X will be totally pain-free one day after three such major fractures. (2) Not timely. The end-point of this goal is too far in the future. (3) Not specific – what does 'much better' mean? (4) Not achievable. The orthopaedic protocol does not permit this. (5) Not measurable and not specific – what does 'more' mean?

General points

Think about how you will realistically progress your treatment and how you will measure any progression (e.g. grip strength, isokinetic machine, goniometry). Work as part of the multi-disciplinary team (MDT) and think about who else needs to have input into the case, but, at the same time, do not forget your own role. Be aware of potential complications, reassure and encourage your patient – who is the most important member of the MDT. Make the patient responsible for his or her own recovery – a partner in fact. Home exercises should be clear, practical and monitored. Reassess progress as necessary. Are you attaining your goals? If not, change or modify your goals or your treatment. Before you discharge the patient remember that a normal limb needs:

- full active movement;
- accessory movement;

- full strength;
- full function;
- anything else specific to that patient.

Does the patient need follow-up appointments or domiciliary physiotherapy? The physiotherapist is a member of the MDT. The membership and relative roles of this team change according to the nature of the injury and the stage of treatment but the physiotherapist must liaise and work with the other members throughout the rehabilitation period. Initially, if the patient is in hospital the members of the team will include: the patient, medical staff, nurses, occupational therapist, pharmacists, radiographers, district nurse and corresponding domiciliary staff.

COMMONLY ENCOUNTERED FRACTURES AND SOME PRINCIPLES OF MANAGEMENT

Fractures of the upper limb

Fractures of the clavicle and scapula

Scapular fractures are not particularly common and usually occur as a result of direct trauma. The clavicle often fractures following a fall on to the side or as a result of a fall on an outstretched hand. The fracture is usually in the middle or the junction of the outer and middle thirds of the bone. The pull of sternocleidomastoid muscle can cause displacement.

These fractures are usually immobilised by a brace, a sling, or a collar and cuff. Complications include a restricted range of movement in the shoulder girdle or shoulder joint, as the two work together, and associated muscle weakness.

Fractures of the proximal humerus

Fractures of the proximal humerus may be classified using the Neer classification:

- Group 1 – minimal displacement;
- Group 2 – anatomical neck fracture with less than 1 cm displacement;
- Group 3 – displaced or angulated surgical neck;
- Group 4 – displaced fracture of greater tuberosity;
- Group 5 – fractures of the lesser tuberosity;
- Group 6 – fracture dislocations.

Fractures of the surgical neck of humerus

These usually occur in elderly people as the result of a fall on the outstretched hand. There may or may not be displacement of the fragments, but in a large number of cases the fragments are impacted. This means that one bone fragment has been driven into the other, often stabilising the fracture at the time of injury. Displaced fractures, and particularly those occurring in the elderly, are not usually reduced for a number of reasons:

- lack of good alignment does not affect union;
- it is preferable to avoid surgery in the elderly unless essential;
- early movement is important to avoid a stiff shoulder.

Fractures of the shaft of the humerus

These fractures usually occur in the middle third of the bone and may be a result of direct or indirect trauma. *Direct trauma* may give rise to transverse or oblique fractures and sometimes present as comminuted fractures. Displacement may result owing to muscle pull, and if the fracture is below the insertion of deltoid the upper fragment will be abducted. *Indirect trauma* tends to give a rotational force resulting in a spiral fracture.

In stable fractures the fixation can be minimal and consist of a sling alone or with a posterior slab from below the shoulder to the wrist with the elbow at 90°. This allows the weight of the arm to maintain reduction. If the fracture needs sturdier fixation, intramedullary nailing is possible or a complete plaster from the shoulder to the wrist or hand may be applied.

Because the fracture usually occurs in the middle part of the shaft, the radial nerve may be affected as it winds through the radial groove. As the radial nerve supplies the wrist and forearm extensor muscles, a wrist drop may result. The injury may compress the radial nerve and cause a neuropraxia, or if it is stretched it may result in axonotmesis. Normally, these will recover spontaneously although an axonotmesis will take longer as degeneration of the nerve has occurred within the sheath. Patients are usually given a wrist splint, to support the wrist in extension, while the nerve is healing. In an open fracture the radial nerve may be severed resulting in a neurotmesis; this will require surgical suturing.

Delayed union or non-union can be complications but they are not very common.

Fractures of the condyles of the humerus

These fractures are common in children following a fall. A supracondylar fracture is the most common type (Figure 22.15). After reduction the arm may be immobilised in one of the following ways depending on the type of fracture:

- plaster with the elbow at approximately 90 degrees or a little more and extending from below the shoulder down to the wrist or hand (the plaster should be cut so that it is possible to feel the radial pulse at the wrist);
- a posterior slab plus a collar and cuff;
- a collar and cuff.

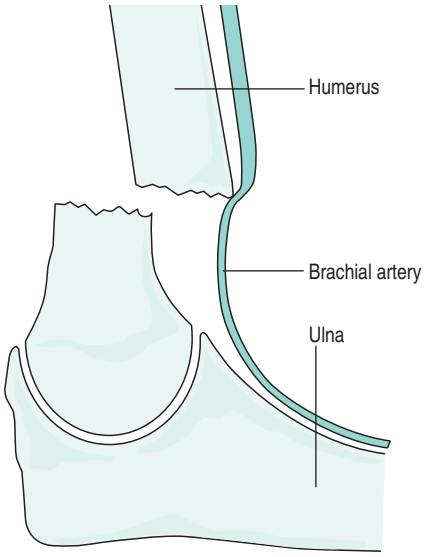


Figure 22.15 Supracondylar fracture of the humerus.

Some fractures of the condyles may extend onto the articular surfaces and thereby cause additional problems. One of the most serious complications that can occur is damage to the brachial artery, which could be severed or contused owing to its close proximity to the fracture site. Therefore, circulation must be monitored. Impairment of the circulation requires emergency treatment, as occlusion can lead to irreversible ischaemic effects within a few hours. If the circulation is not restored, Volkmann's ischaemic contracture may develop. This affects the flexor muscles of the forearm. Muscle tissue is replaced by fibrous tissue which contracts and produces flexion of the wrist and fingers. The skin and nerves will also be affected by the diminished blood supply.

Another problem following elbow fractures is post-traumatic ossification – sometimes known as myositis ossificans. If there is a severe injury, the periosteum may be torn from the bone resulting in bleeding and the formation of a haematoma. Osteoblasts invade this blood clot and new (ectopic) bone forms. This can also occur as the result of forced extension of the elbow. First indications that this is developing may be pain and loss of movement. The elbow should be rested in a sling or collar and cuff for about three weeks to allow the haematoma to be absorbed. If this does not occur and bone is formed it may be necessary to remove the bone tissue surgically. If deformity develops at the elbow – such as a cubitus valgus – this may cause a stretch on the ulnar nerve, which may require surgical intervention with a transposition of the nerve from the posterior to the anterior aspect of the elbow. Fractures that extend onto the articular surfaces and cause disruption of the joint may cause a permanently stiff elbow, lead to the development of osteoarthritis or both.

Fractures of the radius

Radial head fractures

Management of radial head fractures ranges from no immobilisation at all in undisplaced fractures, to screw fixation, excision or replacement arthroplasty.

With fractures of the radius and/or ulna, both bones may be fractured as a result of direct or indirect violence such as a fall on the outstretched hand. The resulting displacement may be difficult to correct and, in some instances, may require open reduction. Accurate anatomical reduction is very important because loss of the normal relationship between the two bones may result in impairment of pronation and supination – a very important component of hand function. In children, the damage may not be so severe and they may sustain a greenstick fracture with minor angulation which normally will heal without any complications. Fracture of the ulna with radial head subluxation is called a *Monteggia fracture*, while fracture of the radius and subluxation of the lower end of ulna is called a *Galeazzi fracture*.

Fractures of the distal radius

Fractures of the distal radius are described by their angulation (dorsal or volar), whether they are intra- or extra-articular and the degree of comminution. They are common, particularly in the elderly, as a consequence of osteoporosis (Figure 22.16a, b, c).

These fractures are reduced and immobilised with a complete plaster cast, or backslab, from just below the elbow to the hand, ending just above the proximal crease on the palm. The position of the wrist and whether or not there is a complete plaster will depend on the pattern of the displacement. If there is gross swelling it may be necessary to use a plaster back slab and then a complete plaster when the swelling has reduced. Fixation is usually maintained for 4–6 weeks.

These fractures used to be known as Colles' (dorsal angulation) and Smith's fractures (volar angulation).

Fractures of the proximal radius are less common and tend to occur in younger people following either a direct blow or a fall on the outstretched hand which causes a fracture through the head of the radius. Fractures of the ulna alone are not as common as those of the radius.

There are a number of complications that can occur with fractures of the lower end of the radius, although these are rare considering the numbers of fractures dealt with in fracture clinics. Complications can include:

- loss of shoulder movement if it is injured when the patient falls or as a consequence of wearing a sling or collar and cuff;

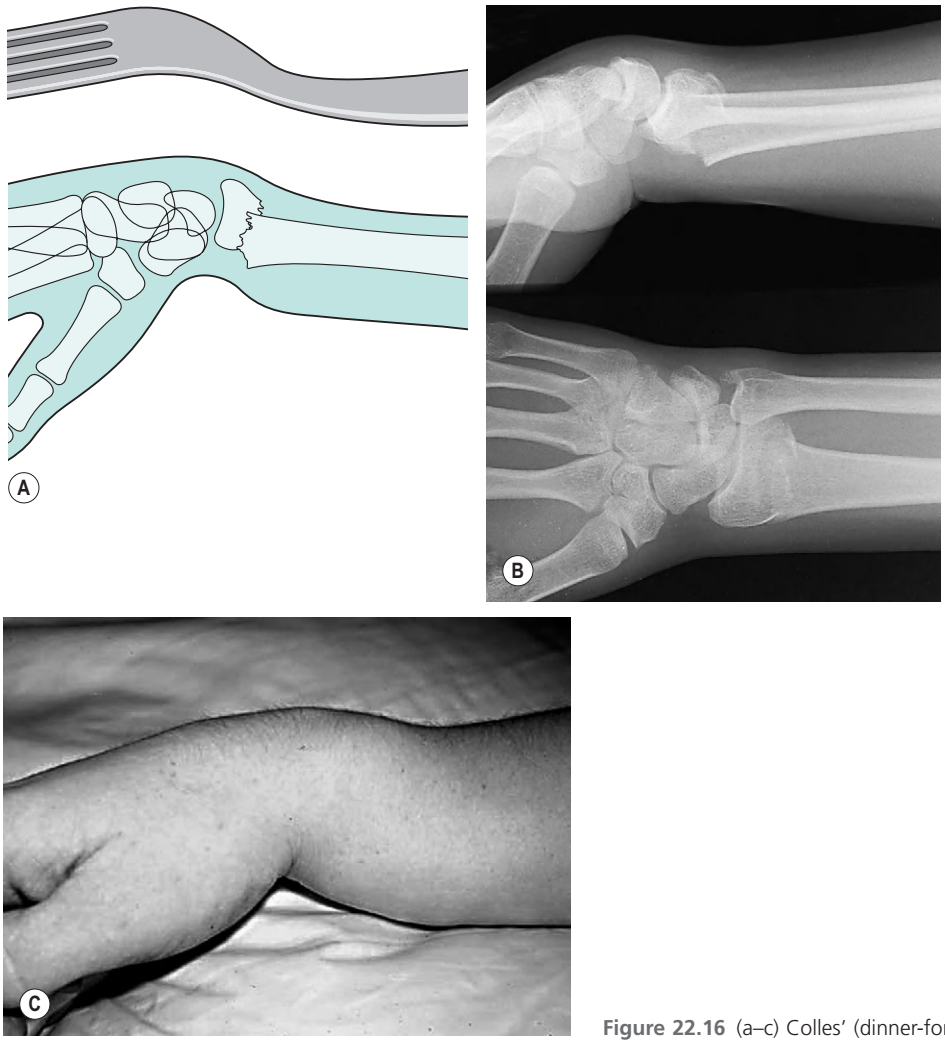


Figure 22.16 (a–c) Colles' (dinner-fork) fracture.

- rupture of the extensor pollicis longus, occurring 4–8 weeks after the fracture;
- Sudeck's atrophy;
- median nerve neuritis if displacement causes stretching or compression of the nerve.

Fracture of the scaphoid

This fracture tends to occur in young adults as the result of falls on the outstretched hand. It may be overlooked either because the person considers it to be a strain, or the fracture may not be visible on the initial X-ray. Healing is often slow in this fracture and, in some instances, there may be non-union. In the latter case the arm is usually placed in a so-called 'scaphoid plaster' as a precaution and X-rayed again after a couple of weeks. If these fractures are accurately diagnosed within one week followed by plaster immobilisation, non-union can be prevented (Roolker et al. 1999).

If the fracture occurs through the waist of the bone the blood supply to the proximal part of the bone will be impaired and avascular necrosis may develop. Long-term complications include the development of osteoarthrosis.

Fractures of the phalanges or metacarpals

Accurate anatomical reduction and fixation is essential, but it is also important to keep the period of immobilisation as short as possible if a good functional result is to

be obtained. The position of the fixation will vary depending on which phalanx or phalanges are fractured and on the subsequent stability of the reduced fracture. This can be very important in relation to regaining function of the hand, and the team has to decide on the priorities in each case. The optimal splinting position for the hand ensures that the metacarpophalangeal (MCP) joints are held in 90 degrees of flexion with the interphalangeal (IP) joints in neutral. This ensures that the MCP collateral ligament length is maintained preventing contractures and loss of range of motion and function.

In stable fractures, a buddy strap splint may be used which fixes the injured finger to the adjacent finger and gives some support while encouraging some movement.

Bennett's fracture

This is a fracture dislocation affecting the carpometacarpal joint of the thumb.

Fractures of the lower limb

ABC Definition

The patient's weight-bearing status is dependent on many factors, including the type of fracture, quality of bone, age of the patient and the orthopaedic management undertaken.

Fractures of the pelvis

The pelvis may be thought of as a ring; like a ring it will often break in two places at once. An isolated fracture is not, as a rule, serious unless it is complicated by damage to the internal organs. The same is true of double, or even multiple fractures, provided that there is no fracture or dislocation in the iliac segment. However, if there are two or more fractures or dislocations with at least one in each segment, then the displacement may be considerable.

The majority of pelvic fractures are caused by direct violence, falls or following crushing injuries. In major pelvic trauma the blood loss sustained by the patient at the time of the injury or from damage to the internal organs may be a life-threatening complication. When the pelvic ring is severely disrupted then rapid reduction and fixation is necessary. If it is possible to reduce the displacement manually then fixation may be by means of a plaster spica, but otherwise another method of external fixation may be used by placing pins through the iliac bones and fixing to a transverse bar. Skeletal traction may be used for certain pelvic fractures. Complications may include injuries to the bladder or urethra and possibly to other tissues within the pelvis.

More common fractures include *pubic ramus* fractures (secondary to osteoporosis) which are managed conservatively with analgesia and gradual mobilisation. These fractures can be very painful as the hip adductors have their origin in this area, so walking is, understandably, painful.

The skill as a physiotherapist here is to gain the confidence and respect of the patient and gradually mobilise the person to recovery. It also highlights the need for teamwork when co-ordinating analgesia with mobilisation.

Avulsion fractures occasionally occur in the pelvis at the anterior iliac spines, more specifically at the attachment of rectus femoris and sartorius. They are caused by forcible contraction of the muscle, pulling off the tip of the bone.

Fracture of the neck of the femur

This is probably the most common and most significant fracture in terms of morbidity, mortality and socioeconomic impact in developed countries (Reginster et al. 1999). Mortality after fracture is high: Schurch et al. (1996) found that the one-year death rate was as high as 23.8% following a fractured neck of femur (Figure 22.17).

Femoral neck fractures should be described/classified by its location, for example basicervical and whether it is intracapsular or extracapsular, as this has a bearing on the surgical fixation chosen (Figure 22.18). Classifications are:

- subcapital;
- transcervical;
- basicervical;
- intertrochanteric;
- subtrochanteric.

They are also referred to as Garden's classification; however, this terminology is not as current as it was but may still be present in the literature.

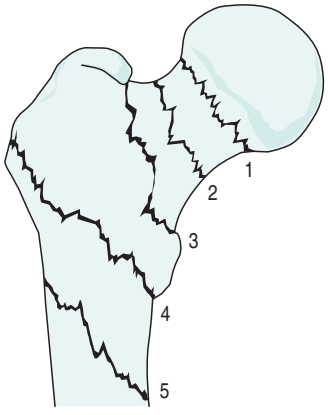


Figure 22.17 The Garden classification of femoral neck fractures: (1) subcapital; (2) transcervical; (3) basicervical; (4) intertrochanteric; (5) subtrochanteric.

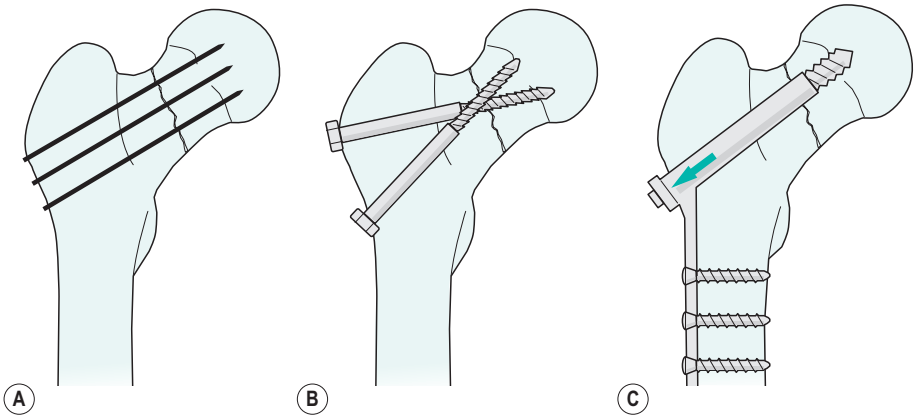


Figure 22.18 Methods of fixation for femoral neck fractures: (a) k wire; (b) cannulated screw; (c) dynamic hip screw (DHS). *(Reproduced from Dandy and Edwards (1998), with permission.)*

Femoral neck fractures are extremely common in the elderly, often following falls, and most orthopaedic units will have a number of these fractures at any one time. The architecture of the bone may have been so weakened that patients say that they 'heard a crack' before they hit the ground. In other words the fracture caused the fall, not the fall the fracture. Osteoporosis is often referred to as the silent epidemic as it may not present any clinical signs until fracture.

The resulting fracture is usually displaced with lateral rotation of the femoral shaft so that the leg will be laterally rotated and shortened in comparison with the other limb. Occasionally, the fragments are impacted in slight abduction and the patient may be able to get up and walk after the injury. Displaced fractures will need operative fixation. For intracapsular fractures the usual method is to excise the head and perform total hip replacement arthroplasty, although a hemiarthroplasty is still used in some patients (Figure 22.19). This is the method of choice for displaced fractures because of the dangers of avascular necrosis and because of the benefits of early mobilisation, which is so important in the frail. For more active older people who have fewer comorbidities, orthopaedic surgeons are increasingly choosing to fix the fracture with a total hip replacement.

For extracapsular fractures, where the blood supply is not impaired, a compression screw plate called a 'dynamic hip screw' is used. This allows dynamic movement at the fracture site which stimulates healing – more so when patients weight-bear through the affected limb. Subtrochanteric fractures are routinely fixed with a proximal femoral nail. Minimally displaced (e.g. Garden type 1) fractures may be managed by cannulated screw fixation. Both the last two procedures are likely to involve a weight-bearing restriction for the patient on the affected side until the bone becomes stronger.

Figure 22.19 Thompson's hemiarthroplasty.

Complications

The blood supply to the femoral head is predominantly via a periarticular anastomosis (Palastanga et al. 1998). Avascular necrosis (death of part of the bone owing to lack of blood supply) can occur as the blood supply to the head of the femur may be impaired following a fractured neck of femur (Figure 22.20).

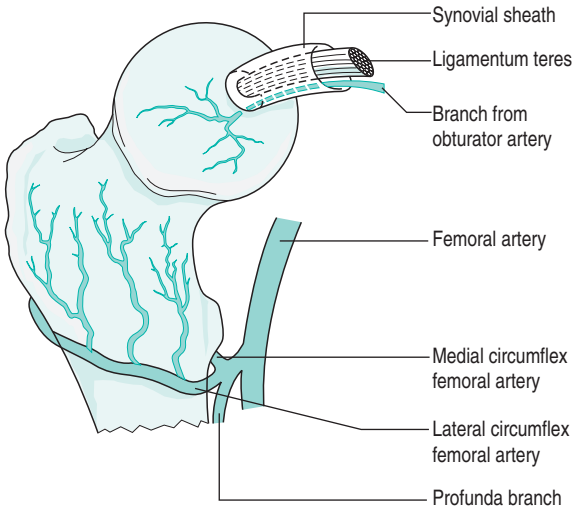


Figure 22.20 Arterial supply to the hip. *(Reproduced from Palastanga et al. (2002), with permission.)*

Fractures of the shaft of the femur

These fractures are usually the result of severe violence and may occur at any part of the shaft, and may be of any type – transverse, oblique, spiral – and may be comminuted. Usually, there is marked displacement with overlap of the fragments, which could lead to limb shortening if it is not corrected. Angulation may occur depending on the injury and on powerful muscle spasm pulling the fragment in the direction of the attached muscles.

For children under the age of three years 'gallows' traction may be used. A modern, more acceptable alternative to traction is the use of an intramedullary nailing (Figure 22.9) which can be performed by the closed technique. The nail is passed through the greater trochanter and down the medullary canal of a long bone and through the fracture site. This is preferable to the previously favoured method of prolonged traction or open reduction, as there is less risk of infection and the complications of bed rest and immobility.

If the fracture is an open fracture there is a risk of infection. Delayed union or non-union is occasionally a complication of this injury, as is malunion. If the overlap of the fragments is not reduced or there is redisplacement, this can occur with consequent shortening of the femur. When the fracture has been fixed internally with an intramedullary nail, mobilisation can occur more rapidly. More comminuted femoral fractures could be fixed with external fixation (e.g. an Ilizarov frame) at the specialist centres.

Fractures around the knee

These include fractures of the tibial condyles, the patella and the femoral condyles.

Injury to the tibial condyles may comprise either a comminuted compression or a depressed plateau fracture. In the former, reduction is not usually attempted and early mobilisation is encouraged. Depressed plateau fractures require reduction to try to achieve an anatomically correct articular surface. In complex tibial fractures, surgery is often undertaken to prepare the area for a subsequent total knee replacement where onset of traumatic osteoarthritis is anticipated. Constant passive motion may be used immediately after the fracture or after surgery to preserve synovial sweep and maintain articular cartilage nutrition.

Fractures of the femoral condyles are not very common but a supracondylar fracture occurs more frequently.

Complications

- A stiff knee could occur as the result of adhesions or because of disruption of the articular surfaces in fractures of the tibial condyles or patella.
- Secondary osteoarthritis may occur as a late complication following disruption of the articular surfaces.
- Genu valgum may develop following depression of the lateral tibial condyle.
- Haemarthrosis may be a problem after fractures of the tibial condyles. This may need to be aspirated (drained) and bandaged, but the swelling that occurs as a result of a synovitis will gradually absorb. If other soft tissue structures are damaged there may be further swelling, in which case the limb should be elevated to assist drainage.

Fracture of the patella

This can be caused by a direct blow on the knee or a sudden violent contraction of the quadriceps resulting in an avulsion fracture. The former tends to cause a crack or comminuted fracture whereas the latter may produce a transverse fracture. Internal fixation may be required if there is separation of the fragments.

Fractures of the tibia and fibula

These fractures are common and occur at all ages as a result of direct or indirect violence. Often, they are open fractures either because of the direct violence or because the anterior tibia is very close to the surface and the fragments may extrude through the skin.

Direct violence, commonly owing to road traffic accidents or soccer, is likely to give an oblique or transverse fracture. It may be comminuted and further complicated by soft tissue damage. Fractures caused by a rotatory force, such as may occur in skiing, are usually spiral and the fractures of the two bones are at different levels.

Fixation will depend on the type of fracture and the amount of soft-tissue damage. A functional brace with a

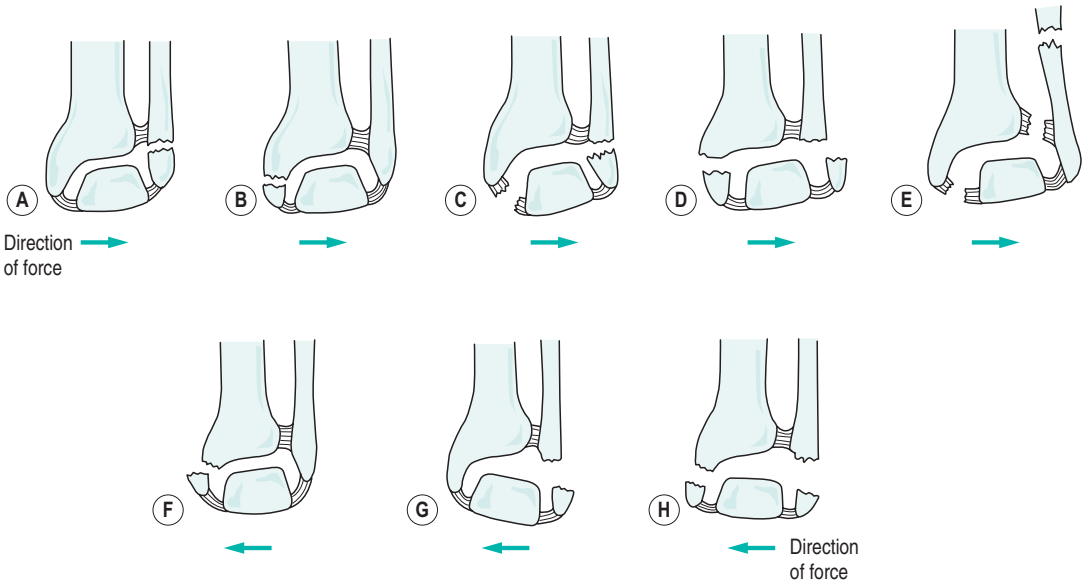


Figure 22.21 (a–e) Fractures resulting from an abduction/lateral rotation force: (a) fracture of lateral malleolus; (b) avulsion fracture of medial malleolus; (c) fracture of lateral malleolus, rupture of medial ligament and lateral shift of talus; (d) fracture of lateral and medial malleoli and lateral shift of talus; (e) tibio-fibular diastasis – rupture of tibio-fibular and medial ligaments, fracture of shaft of fibula and lateral shift of talus. (f–h) show fractures resulting from an adduction force: (f) fracture of medial malleolus; (g) avulsion fracture of lateral malleolus; (h) fractures of lateral and medial malleoli plus medial shift of talus.

hinge at the ankle may be used and this has the advantage of allowing more movement and a better walking pattern. If there is a lot of soft tissue damage and consequent swelling, a split plaster may be applied and the leg elevated on a Braun frame. This is replaced with a plaster once the swelling has subsided.

Another method that is used to immobilise this fracture is external fixation. Sometimes internal fixation is used with either an intramedullary nail or plate and screws. Common practice now is to commence active movement in elevation immediately after surgery and delay the application of a plaster until movements are sufficiently regained.

Complications

Fractures of the tibia or fibula alone are not very common. Infection is a possible complication as many of the fractures are open. Antibiotics are used to help avoid this and careful wound care is important. The tibia can be the site of a stress fracture owing to repeated minor trauma, probably associated with sport.

Vascular impairment can occur owing to damage to a blood vessel or a plaster cast that is too tight. Great care must be taken by all concerned with the management of these patients to monitor for any signs of circulatory deficiency. Fractures can be very slow to heal (delayed union) or there may be a non-union owing to poor blood supply in the lower third of the tibia. Compartment syndrome is another common complication.

Fractures around the ankle

Figure 22.21 shows fractures resulting from an abduction/lateral rotation force or an adduction force. Complications include limitation of movement in the ankle joint and foot resulting from peri-articular and intra-articular adhesions or from disruption of the articular surfaces. The latter may also lead to the later development of secondary osteoarthritis.

For fractures without displacement, a below-knee walking plaster may be applied for 3–6 weeks. When there is displacement it is important for the surgeon to try to ensure that reduction establishes the normal anatomical relationship at the ankle joint. If reduction cannot be attained by manipulation and plaster immobilisation it may be necessary to have an ORIF and use a screw (or screws) to maintain a good position of the fragments, followed by immobilisation in a below-knee plaster. The modern trend is towards immediate postoperative active exercise (with adequate analgesia), then subsequent application of a cast once good movement (particularly dorsiflexion) is attained. A Tri-malleolar fracture involves fractures of both the medial and lateral malleoli, as well as fracture of the posterior tibial malleolis. Tri-malleolar fractures are generally unstable.

Fractures of the foot

Fractures of the calcaneum usually occur as the result of a fall from a height on to the feet, fracturing the calcaneum in one or sometimes both feet. It may well be accompanied by a fracture of one of the lower thoracic or upper lumbar vertebrae. (Take note if a patient with a fractured calcaneum complains of back pain.) Calcaneal fractures can be extremely painful and carry a poor functional prognosis if inversion and eversion are not regained, as their movements are essential for normal function of the foot in such activities as adapting to uneven surfaces. These may be managed either conservatively or by open reduction and internal fixation.

The emphasis of physiotherapy management is on the reduction of the oedema and mobilisation. Once the patient is allowed to bear weight it is important to re-educate gait, as well as concentrating on strengthening muscles and regaining range of movement in the ankle and foot. It may not be possible to regain any movement at the mid-tarsal joints and the patient will have to learn to adapt to this loss of movement. The arches of the foot may have flattened and this could be the result of weak muscles, deformity of the foot or both. In the former case the muscles can be strengthened, but if the latter is the case the arches will not reform: the patient may continue to have persistent pain and tenderness for a long time after the fracture has healed and it is difficult to relieve.

Generally speaking, it is advisable to commence early active mobilisation in elevation. Cryotherapy/TENS, flowtron boot and patient-controlled analgesia (PCA) have all been used with success. In later stages, accessory mobilisations to the affected joints may be appropriate.

The phalanges and metatarsals are most likely to be fractured by a heavy object falling on the foot. This will also cause soft-tissue damage and, consequently, swelling is likely to be severe. These fractures do not, as a rule, require reduction or immobilisation. However, a below-knee walking plaster is usually applied for fractures of the metatarsals to relieve pain and enable the patient to walk. If swelling is severe the patient will need to rest in bed with the leg elevated for a few days.

Another type of fracture that occurs in the metatarsals is a stress fracture – often known as a 'March' fracture. It is caused by repeated minor trauma that may arise from prolonged walking, particularly on hard surfaces, and usually in someone who is unaccustomed to walking long distances. It is usually a crack fracture affecting the shaft or neck of the second or third metatarsal. No fixation is required but a walking plaster may be applied if the pain is severe.

Complications

Complications include stiffness, particularly if there has been disruption of the articular surfaces of the subtalar joint. Secondary osteoarthritis may develop later as a result of the disruption of the joint surfaces.

Spinal fractures

These are not dealt with in this text.

CRYOTHERAPY

This is regularly used in the orthopaedic setting and is covered in Chapter 12.

Continuous passive motion (CPM)

Use of a CPM machine is variable in the orthopaedic setting (Figure 22.22a, b).

Like any other modality in medicine, it has its place and its limitations. Salter (1993) has made an interesting study of the uses of CPM.

BENEFITS OF CPM

- There is maintenance of synovial sweep and thus hyaline articular cartilage nutrition. This is useful after certain intra-articular fractures (e.g. tibial plateau).
- Regular rhythmical motion can act as an analgesic, can stimulate circulation and may assist in the reduction of swelling.
- CPM has been used following anterior cruciate reconstruction, particularly following patellar tendon graft. It is possible that this encourages more rapid revascularisation and therefore strength of the donor graft.
- It is possible to increase the flexion/extension in a controlled manner that is immediately obvious to the patient and can assist in giving the patient a goal to strive for.
- Some units have counters so that the healthcare team can tell exactly for how long the patient has been using the unit.
- CPM units are now available for shoulder, wrist and other joints.
- CPM units may now be used in the patient's home.

Continued

DISADVANTAGES OF CPM

- It is passive and, therefore, by definition will not build muscle strength. Some patients mistakenly neglect active exercises in the belief that they no longer need to undertake them. It is the responsibility of the physiotherapist to ensure that this situation does not occur.
- Some patients are distressed by the appearance of the unit and feel threatened. Most units have a panic button so the patient can stop the unit for rest, meals or toiletting.
- The units can be bulky and expensive.
- If incorrectly positioned they can cause pressure problems and be uncomfortable.
- They pose an infection risk if not properly cleaned and policies for their use followed.

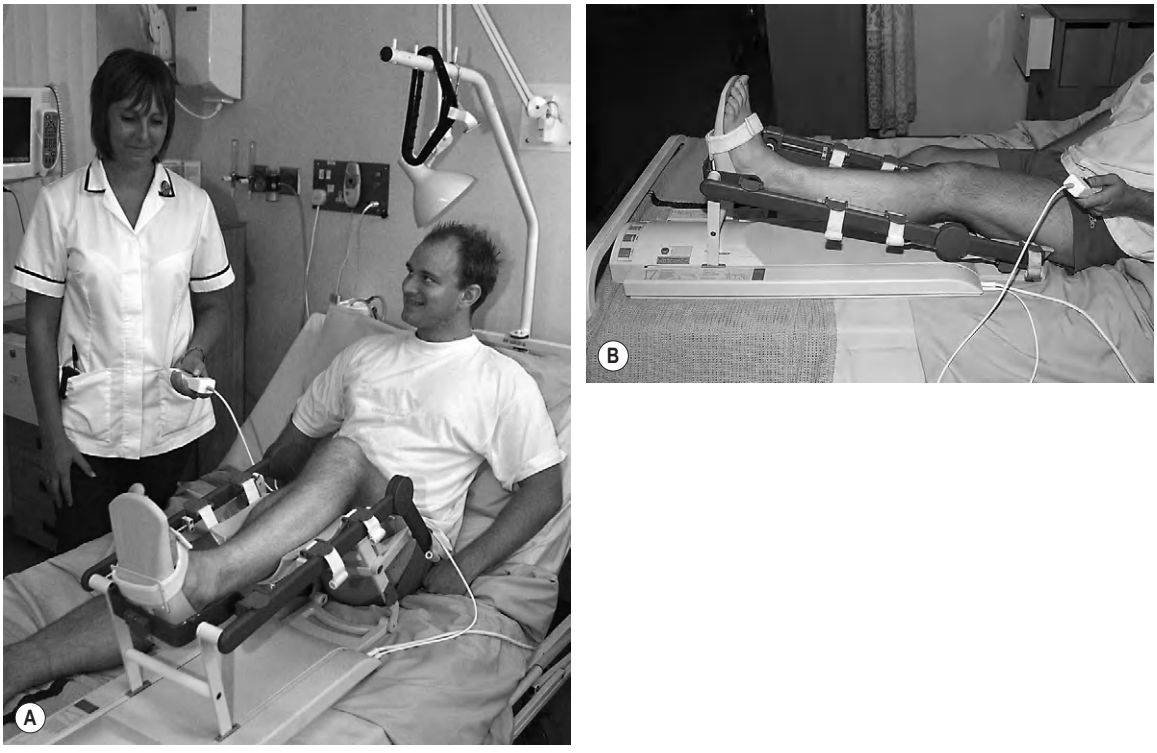


Figure 22.22 A demonstration of a continuous passive motion (CPM) machine. *(With thanks to Joanne Kenyon.)*

CASE STUDY: FRACTURED SHAFT OF THE FEMUR

Note that the material in this section does *not* represent a recipe for the management of all fractures of the femur.

BACKGROUND

Ms Jones is a 25-year-old law student who was involved in a road traffic accident. She was driving and as a result of a head-on collision the dashboard was pushed backwards into her knees. She sustained a fractured shaft of the left femur (Figure 22.23a) and was treated by intramedullary fixation (Figure 22.23b). Ms Smith spent ten days in hospital and has now come to the outpatient physiotherapy department. She did not sustain any other injuries.

ON EXAMINATION

Ms Jones is partial weight-bearing and is mobilising with two elbow crutches and wearing a functional (cast) brace. She is independently mobile, but needs help to remove her training shoe and sock. The brace was custom-made by the occupational therapists (Figure 22.24) and it is unlocked to permit 0–90 degrees of knee flexion. The physiotherapist may unlock the brace by a further 10 degrees each week. The patient must wear the brace when walking but is permitted to remove it to perform knee flexion exercises and to take a shower.

She is extremely anxious about what is going to happen to her and confused about how much or how little she should be doing. The only exercise she can remember is the straight leg raise (SLR). She is articulate, co-operative and keen to take advice. She cannot currently attain heel strike and she states that this is because it causes her calf to be painful (deep vein thrombosis has been excluded). She has been told to keep moving and, as a result, she is walking long distances every day. Her leg aches badly at bedtime and she is worried that the fracture has 'moved', although X-rays show that it has not displaced since the time of surgery.

OBJECTIVE EXAMINATION

There are two incisions: one proximal to the greater trochanter, one lateral to the knee joint. These have healed. The patient has a reddened area over the lateral malleolus, which is a result of the brace rubbing her skin.

Knee joint

The range of movement of the knee joint with the brace removed is shown in Table 22.4.

Patello-femoral joint

All accessory movements are reduced by approximately 50% on the left side.

Ankles

There is full active range plantar flexion in both ankles. Terminal dorsiflexion of the affected side causes calf 'tightness' when the left knee is in the fully extended

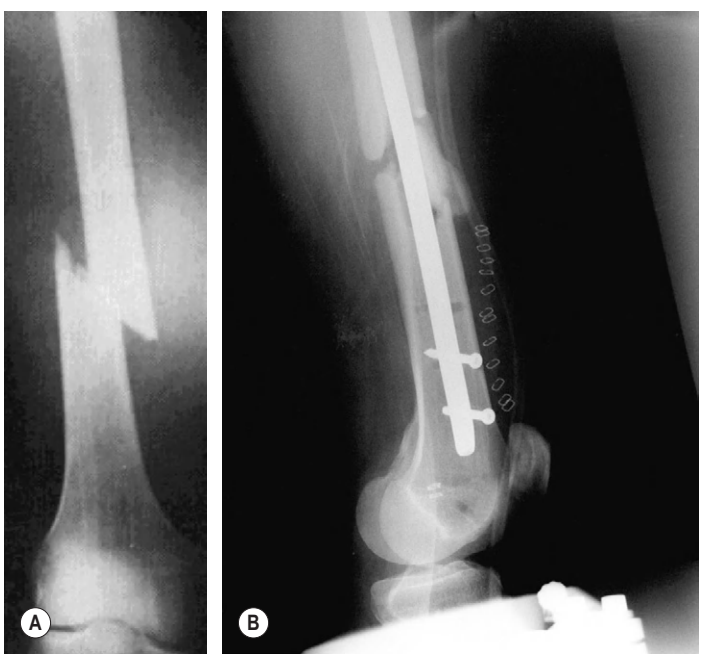


Figure 22.23 (a) The femoral fracture. Note that spasm in the quadriceps has resulted in overriding of the fracture fragments. (b) Note the locking screws and skin staples visible on X-ray.

Continued

Table 22.4 Features of the patient's knee joint

	Left knee			Right knee		
	*Passive**	*Active*†	*Over-pressure*§	*Passive*	*Active*	*Over-pressure*
Flexion	0–60	0–40	–	130	130	–
Extension	Full extension possible but causes posterior knee discomfort			Full 0-degree extension painless		
Lateral rotation	Full			Full		
Internal rotation	Full			Full		

*Limited by apprehension. †Limited by aching at the fracture site and quadriceps spasm. §Not tested in view of fracture status and patient apprehension. The units are degrees.

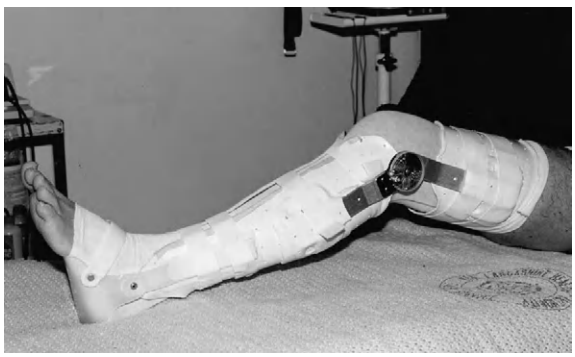


Figure 22.24 The functional brace. The heel cup is essential for correct off-loading of weight from the fracture. *(Courtesy of Mike Somervell and Julie Butler.)*

Table 22.5 Features of the patient's muscle strength (Oxford grade)

	L	R
Quadriceps	4	5
Hamstrings	4	5
Tibialis anterior	4	5
Gastrocnemius	4	5

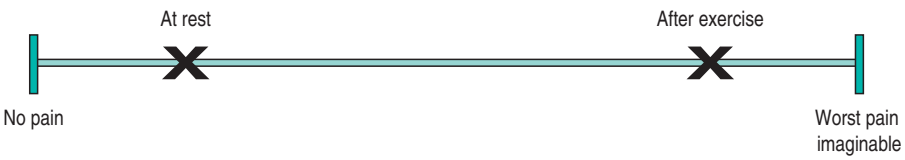


Figure 22.25 The patient's pain scale.

position. The range of plantar and dorsiflexion is full, however. Small joints of the foot, intertarsal and tarso-metatarsal, have some loss of accessory movement of the subtalar and intertarsal joints of the affected side. There is some oedema of the ankle. Capillary filling is poor.

Muscle strength

(Oxford grade – left:right) (See Table 22.5).

Pain

Figure 22.25 shows the pain recorded using a visual analogue scale.

Limb girth

See Table 22.6.

SHORT-TERM PROBLEMS

Do not underestimate the impact of apprehension and confusion. If a person is nervous, frightened or confused, whatever you plan to tell, teach or ask of them, will be adversely influenced. Do not think only of the fracture – take a holistic approach. Physiotherapists are not technicians, they are also educators. It is perfectly valid to undertake with her a teaching session on fracture healing

Continued

Table 22.6 The patient's limb girth

Limb girth	10 cm above patella apex	20 cm above patella apex
Left leg	48 cm	44 cm
Right leg	56 cm	52 cm

and to discuss your aims of treatment. This might seem time-consuming but consider your aims of treatment and your role as her physiotherapist. The quality of your communication and subsequent discussion of your management plan will influence her eventual health outcome. There is now strong research evidence suggesting a positive correlation between effective professional–patient communication and improved patient health outcomes.

The sore caused by the brace is a high priority. The physiotherapist needs to refer the patient to the occupational therapist for adjustment of the brace. If the sore develops to the point where she cannot tolerate the brace, your ability to progress her treatment will be adversely affected.

Pain and aching at the end of the day is normal – your goal here is to explain *why* the pain is happening and that it signifies bone healing. Do not assume that your patient will automatically know this. Some patients equate pain with 'no pain no gain' and see it as a positive factor, whereas others will be frightened by pain. In this person's case it seems likely that too much walking is being undertaken; she needs advice on smaller, more frequent walks with attainable goals. For example, she could be told to walk the length of the home every hour. Knee flexion may be painful, in which case your role as her physiotherapist is to identify the specific cause and minimise or reduce it prior to active exercise. This might consist of asking the patient to take her analgesia one hour before commencing her exercises, or the application of TENS or hydrotherapy as a supplement to exercise.

Her current exercise regimen needs to be modified. She should be advised to go for regular, short walks and gradually build up the length of time she walks for. She needs exercises that will help strengthen her leg. These should include quadriceps exercises, exercises for the hamstrings, hip extensors and abductors, gastrocnemius and anterior tibial muscles, and should be worked within the permitted limits of the fracture rehabilitation. She should only be given a few exercises at a time to encourage compliance and help her grow in confidence. These must be taught and explained clearly, written down if possible, and the rationale behind them explained to the patient. Patient compliance with exercise is, in general, poor (Campbell et al. 2001) and the more you can do to make home exercises simple and practical, the more success you will have in educating and rehabilitating your patient. The patient should be advised to stop or reduce the exercises if they are causing pain and seek further advice.

Loss of knee flexion owing to callus may be impeding soft tissue mobility. Pain, fear or muscle spasm might also be causes of limited mobility; as she has more passive than active range, something other than the joint is limiting her movement. Your role is to identify the cause and treat it accordingly. If there are no inflammatory signs, a heat pack or massage may relieve spasm. The brace limits flexion to 90 degrees so there is no reason why you should not aim for 90 degrees at this time.

With regard to the oedema, an inefficient muscle pump is a likely cause. She is not attaining heel strike but is walking great distances; these will both exacerbate dependent oedema. Little and often is the key, with elevation during periods of rest, and a graduated increase of walking distance. Massage might be used to relieve oedema but can be time-consuming.

This person has a loss of limb girth on the affected side. This will be a result of disuse atrophy. Decreased limb girth is not as important as muscle function but may be distressing to the patient; reassure her that function will come first, then bulk. While it is not a true reflection of quadriceps girth or strength, it does give the person something specific to aim for.

Loss of accessory movement needs to be addressed in the treatment plan, for example to the small joints of the foot and mid-tarsal joints. The loss of accessory motion at the patello-femoral joint should also be addressed; the function of this joint cannot, and should not, be isolated from the function of the knee joint itself. It is relatively easy to identify immediate problems, but unfortunately this alone is insufficient; you also need to be aware of potential or longer-term complications.

POTENTIAL AND LONG-TERM PROBLEMS: THE BIGGER PICTURE

- Adaptive shortening of the Achilles tendon leading to permanent soft tissue contracture may occur if her gait is not corrected as a consequence of her not attaining heel strike. She currently does not have any soft-tissue contracture as her measurements are full.
- Encourage her to walk shorter distances with a normal gait.
- Check that her pain is being controlled adequately so that she is not afraid to attain heel strike.
- Explain this to the patient so that she can take an active role in her own rehabilitation.
- Make sure that a potential problem does not become a real one.
- Fixed flexion deformity of the knee joint is also a possibility if the importance of attaining full knee extension is not explained to the patient.

Continued

- Remember that the hip joint is also involved and should not be forgotten when planning rehabilitation and providing exercises for the patient.
- Fibrous adhesions may occur if oedema is allowed to organise.
- Retro-patellar adhesions may form as a result of immobility of the knee's expansive synovial membrane and might become a cause of stiffness. The likelihood of this happening might be reduced by isometric contractions of the quadriceps and articularis genus muscle whose task it is to retract the synovium of the knee joint (Ahmad 1975).
- Degeneration of hyaline articular cartilage occurs if a joint is not subjected to movement. The patient should be encouraged to undertake general mobility and exercise little and often.
- Generalised osteopaenia, or a reduction in bone mineral density, may result if weight is not placed through the limb. The see-saw needs careful consideration here – see earlier in this chapter.

TREATMENT PLAN

- The patient must become effectively involved in the management of her condition.
- Commence partial weight-bearing gait and progress within protocol guidelines.
- Re-educate normal gait, i.e. heel strike.
- Mobility of all unaffected joints must be maintained during the period of immobilisation.
- Regain movement and function to normal. This is person-specific and full function might not be attained until removal of the intramedullary nail in one or two years' time.
- Draw up a thorough and fully understood home exercise programme which can be monitored and progressed as needed. It is better to teach two exercises well and have the patient thoroughly understand them than teach ten exercises that are too complex and confusing to the patient.
- Strengthen appropriate muscles dependent upon your assessment findings. This may include exercises for shoulders and the upper limb, as long use of elbow crutches can cause pain in the wrists and shoulders. This should not be overlooked.
- Progress to full weight-bearing within rehabilitation guidelines; check any protocols and specific guidelines.

OVERALL PROGRESS AND DISCHARGE

Study the simplified graph in Figure 22.26. If we plot a hypothetical 'improvement versus time' graph, the student might *hope* for this pattern. ('Improvement' may mean different things to different people. For purposes of clarity it has been shown as a single item.) If this were the case, recovery could always be predicted, all patients could use the same treatment regimen, one assessment would be sufficient, a discharge date could be predicted many weeks in advance and undergraduate training would take about a month! Unfortunately, this is not the case.

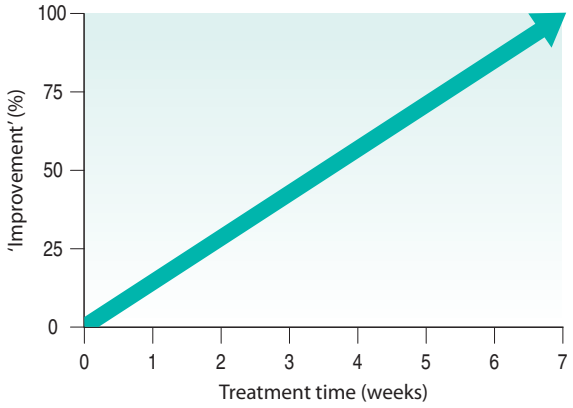


Figure 22.26 A hypothetical improvement chart.

It is more likely that you will see a pattern of peaks, troughs and plateaus, with a general trend towards recovery. The overall shape of the real improvement curve displays the gradual attainment of a plateau in improvement.

This pattern of recovery is common: the patient may gain ten degrees of knee flexion per week for the first three weeks of treatment, then improvements will slow to the point where it takes a further three weeks to attain an additional five degrees. This presents the physiotherapist with a constantly changeable situation – which is one of the reasons why the profession is so stimulating. It also poses the problem of when to discharge the patient. The final decision must be a mutually agreed decision between physiotherapist and patient, and it is at this point that previously-agreed goals are essential. It may be the case that 95% 'improvement' is the maximum possible improvement attainable while the intramedullary nail remains *in situ*. For example, there may be some residual discomfort on running or squatting. Experienced physiotherapists will be able to integrate their knowledge of anatomy, physiology and biomechanics and explain to the patient that now is the time for discharge and that the further 5% will occur following implant removal. Without a sound prior assessment and formulation of goals, appropriate patient discharge may be jeopardised, resulting in discharge that is too early or treatments that become repetitive, non-adaptive and inappropriate. The advantage of physiotherapists being autonomous practitioners is that with a little thought and a sound initial assessment one can avoid the situation where the patient is seen 'twice a week for eight weeks' with no change in treatment or progression. The patient and physiotherapist 'own' the process.

ACKNOWLEDGEMENTS

With thanks to Grit Soboll, Communications Manager Orthofix Srl; Dania Sorio, Customer Service International Orthofix Srl; and the Association of Orthopaedic Chartered Physiotherapists Executive Committee.

FURTHER READING

Association of Orthopaedic Chartered Physiotherapists. Physiotherapy Guidelines of Good Practice in Orthopaedics, www.aocp.co.uk.

Atkinson, K., Coutts, F., Hassenkamp, A.M., 1999. Physiotherapy in Orthopaedics – A Problem Solving Approach. Churchill Livingstone, Edinburgh.

Barker, K.L., Lamb, S.E., Simpson, A.H., 2004. Functional recovery in patients with nonunion treated with the Ilizarov technique. J Bone Joint Surg Br 860B, 81–85.

Barry, S., Wallace, L., Lamb, S., 2003. Cryotherapy after total knee replacement: A survey of current practice. Physiother Res Int 8 (3), 111–120.

Bruckner, P., Bennell, K., 1997. Stress fractures in female athletes: diagnosis, management and rehabilitation. Sports Med 24 (6), 419–429.

Duckworth, T., 1995. Lecture Notes on Orthopaedics and Fractures. Blackwell Science, Oxford.

McRae, M., 1999. Pocketbook of Orthopaedics and Fractures. Churchill Livingstone, Edinburgh.

Stewart, M.A., 1995. Effective physician–patient communication and health outcomes: a review. CMAJ 152 (9), 1423–1433.

Tidswell, M., 1998. Orthopaedic Physiotherapy. Mosby, New York.

Scottish Intercollegiate Guidelines Network (SIGN), 1997. Management of Elderly People with Fractured Hip. A National Clinical Guideline. SIGN, Edinburgh.

REFERENCES

Adams, C.I., Keating, J.F., Court-Brown, C.M., 2001. Cigarette smoking and open tibial fractures. Injury 32 (1), 61–65.

Ahmad, I., 1975. Articular muscle of the knee – articularis genus. Bull Hosp Joint Dis 36 (1), 58–60.

Azuma, Y., Ito, M., Harada, Y., et al., 2001. Low-intensity pulsed ultrasound accelerates rat femoral fracture healing by acting on the various cellular reactions in the fracture callus. J Bone Miner Res 16 (4), 671–680.

Bandolier Evidence Based Healthcare, 2004. NSAIDS, Coxibs, Smoking and Bone, www.jr2.ox.ac.uk/bandolier/booth/painpag/wisdom/NSAIbone.html.

Campbell, R., Evans, M., Tucker, M. et al., 2001. Why don't patients do their exercises? Understanding non-compliance with physiotherapy in patients with osteoarthritis of the knee. J Epidemiol Commun Health 55 (2), 132–138.

Cornell, C.N., Lane, J.M., 1992. Newest factors in fracture healing. Clin Orthop 277, 293–311.

Dandy, D.J., Edwards, D.J., 1998. Essential Orthopaedics and Trauma, third ed. Churchill Livingstone, New York, p. 94.

Hoppenfeld, S., Urthy, V.L., 1999. Treatment and Rehabilitation of Fractures. Lippincott Williams and Wilkins, Baltimore.

Marsh, D.R., Li, G., 1999. The biology of fracture healing: optimising outcome. BMJ 55, 856–869.

Palastanga, N., Field, D., Soames, R., 1998. Anatomy and Human Movement: Structure and Function, third ed. Butterworth-Heinemann, Oxford.

Palastanga, N., Field, D., Soames, R., 2002. Anatomy and Human Movement, fourth ed. Butterworth-Heinemann, Oxford, pp. 3–20.

Reginster, J.Y., Gillet, P., Ben Sedrine, W., et al., 1999. Direct costs of hip fractures in patients over 60 years of age in Belgium. Pharmacoeconomics 15 (5), 507–514.

Roolker, W., Maas, M., Broekhuizen, A.H., 1999. Diagnosis and treatment of scaphoid fractures: can non-union be prevented? Arch Orthop Trauma Surg 119 (7–8), 428–431.

Salter, R.B., 1993. Continuous Passive Motion (CPM) – a Biological Concept for the Healing and Regeneration of Articular Cartilage, Ligaments and Tendons. Lippincott, Williams and Wilkins, Baltimore.

Schurch, M.A., Rizzoli, R., Mermillod, B., et al., 1996. A prospective study of socioeconomic aspects of fracture of the proximal femur. J Bone Miner Res 11 (12), 1935–1942.

Schwartsman, V., Choi, S.H., Schwartsman, R., 1990. Tibial nonunions: treatment tactics with the Ilizarov method. Orthop Clin N Am 21 (4), 639–653.

Sheriff, D.D., Van Bibber, R., 1998. Flow-generating capability of the isolated skeletal muscle pump. Am J Physiol 274 (5:2), H1502–1508.

Tschakovsky, M.E., Shoemaker, J.K., Hughson, R.L., 1996. Vasodilation and muscle pump contribution to immediate exercise hyperemia. Am J Physiol 271 (4:2), H1679–1701.

Viel, E., Ripart, J., Pelissier, J., et al., 1999. Management of reflex sympathetic dystrophy. Ann Med Interne (Paris) 150 (3), 205–210.

Chapter 23

Joint arthroplasty

Ann Price

INTRODUCTION

There is no 'typical' patient who is appropriate for a joint arthroplasty. As with all modern medicine, a decision has to be made which balances the risks of surgery against the potential improvements. Patient age per se is no longer an acceptable clinical decision-making tool (Brander et al. 1997). Generally, the surgical team promote conservative treatments and optimisation of health until pain or disability is severe enough to cause a significant impact on the person's quality of life, where surgery would make things significantly better or prevent a major deterioration.

Thus, successful modern joint replacements are based on appropriate patient selection, selection of the appropriate implant, specific surgical technique and expertise, and multi-disciplinary patient preparation and rehabilitation. Nonetheless an artificial joint is not as efficient as its organic counterpart: it does not repair itself and does not absorb the stresses and strains of daily life as an organic joint can.

UPPER LIMB ARTHROPLASTY

There are prostheses available for every joint in the upper limb. Elbow, wrist and finger arthroplasties are performed mainly for patients with rheumatoid arthritis, although metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints are now being developed for patients with osteoarthritis (OA) and following trauma. The carpometacarpal joint of the thumb is the most common joint replacement for OA. There are also prostheses available to replace the ulnar head and the radial head; these are normally used for reconstruction following difficult forearm and elbow fractures.

SHOULDER ARTHROPLASTY

Initially developed for the reconstruction of severe proximal humerus fractures, proximal humeral arthroplasty was also used for people suffering from OA, with surprisingly good results. In 1973, Neer redesigned the humeral component and added a glenoid to make the first unconstrained total shoulder arthroplasty. The basis of the design was to produce as near to an anatomical replacement as possible – the principle followed in most modern prostheses (Neer et al. 1982).

There are many factors influencing the outcome of shoulder arthroplasty (Iannotti and Williams 1998; see Table 23.1) that physiotherapists need to know about so that realistic rehabilitation goals can be set. Therefore, good communication with the surgical team is very important.

Primary OA is the indication for total shoulder arthroplasty from which the best results can be expected. The result is dependent on the severity of the degenerative changes that have taken place prior to surgery. For patients who have primary OA without gross soft-tissue damage or loss of bone, a near-normal range of movement and strength can be expected: patients who start off with rotator cuff disease or glenoid erosion should have less high expectations.

For total shoulder arthroplasty, the most common surgical approach is known as the 'deltopectoral approach'. The incision passes between the deltoid and pectoralis major, and access to the shoulder joint is via the subscapularis muscle and the anterior part of the capsule. Thus, the subscapularis muscle is the only active structure which will need to be protected in the early postoperative period.

Table 23.2 shows a typical postoperative protocol; however, these will vary depending upon the surgeon's

Table 23.1 Factors affecting outcome of problematic shoulder reconstruction

Pathology
Rotator cuff disease
Glenoid erosion bone loss
Humeral bone loss
Bone loss
Bone density
Surgical technique
Prosthetic placement
Prosthetic–cement–bone interface
Soft-tissue balancing
Prosthetic design
Size selection
Glenoid
Humeral head
Humeral stem
Offsets
Material properties
Rehabilitation programme
Range of motion
Strength
Stability

Table 23.2 Example of postoperative routine following primary total shoulder arthroplasty for osteoarthritis

Day 1
Usual postoperative check, maintenance of range of movement in hand and wrist
Day 2
Assisted active flexion and abduction, first by physiotherapist followed by teaching patient-assisted active of same movements using pulleys and exercise stick
Patient is taught to do exercises x 4 daily
Day 3 to 3 weeks
Continue with exercises as above and add extension, internal rotation and lateral rotation to 30 degrees, with the arm by the side. Abduction should always be with the arm in neutral as the combined movement of abduction and lateral rotation should not be attempted until 3 weeks post-operation
The patient can progress from assisted active to active movement as able. Once the person is comfortable he or she may begin to use the arm for self-care activities within the limits of pain and strength
3 weeks to 6 weeks
The patient should now have discarded the sling completely during the day and should be progressing from assisted active to active movement. The physiotherapist will be showing appropriate ways to do this
Active external rotation can be progressed beyond 30 degrees but stretching should still be avoided
6 weeks to 3 months
Progressive strengthening exercises in all ranges should be encouraged according to the patient's individual capabilities. Rotator cuff strengthening should be emphasised, using a progressive system such as Theraband
3 months onwards
Many patients are discharged from treatment but should be encouraged to continue with strengthening exercises until optimum function has been achieved

preference, any intra-operative changes to the procedure, patient factors and their response. As always, the postoperative regimen must be agreed between the surgeon and the physiotherapy team.

People with rheumatoid arthritis (RA) who undergo shoulder arthroplasty are likely to have a number of the adverse pathological factors (Table 23.1). The expected results will depend on how many, and how severe, they are, so the results are generally not as good as in OA patients. The surgical approach and basic postoperative management are the same.

In advanced disease (either OA or RA type) it is not always possible to insert a glenoid component – it is not possible to attach the glenoid component securely enough if there is gross bone loss around the glenoid fossa. Also, if there is a lack of rotator cuff function, the humeral head will 'rock' the glenoid component, causing loosening. The problem of glenoid fixation is one of the ongoing dilemmas in shoulder arthroplasty (Figure 23.1 and Figure 23.2).

Following a complex fracture it is normally a hemiarthroplasty that is performed, as the glenoid is usually intact. The operation can be performed either as the primary treatment for the fracture or later as a secondary procedure (the results are better if it is performed in the acute phase). Frequently, the tuberosities are disrupted giving the surgeon the additional challenge of restoring normal anatomical alignment and cuff attachment with secure fixation, thus allowing early rehabilitation. The physiotherapy programme should be individualised, taking into account any soft tissue trauma that will have occurred at the time of injury.

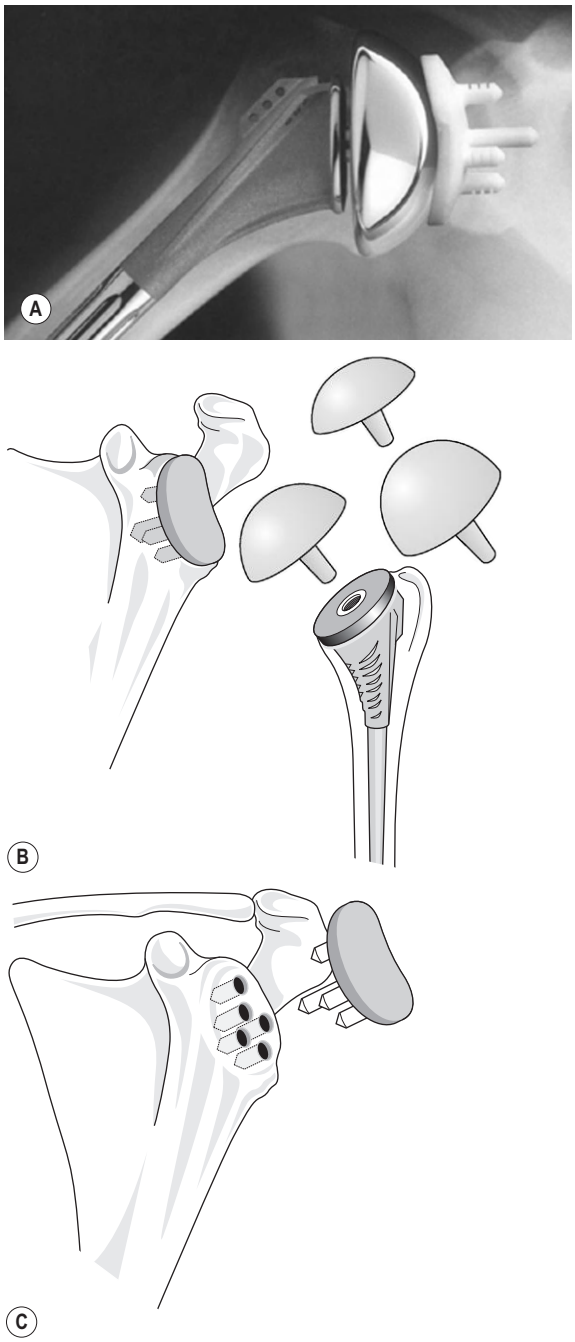


Figure 23.1 (a) The global shoulder implant. (b) Sketch to demonstrate the different heights of humeral head available. (c) Sketch to show how the glenoid component is attached to the glenoid fossa. *(Reproduced by kind permission of De Puy Orthopaedics Inc., IN, USA.)*

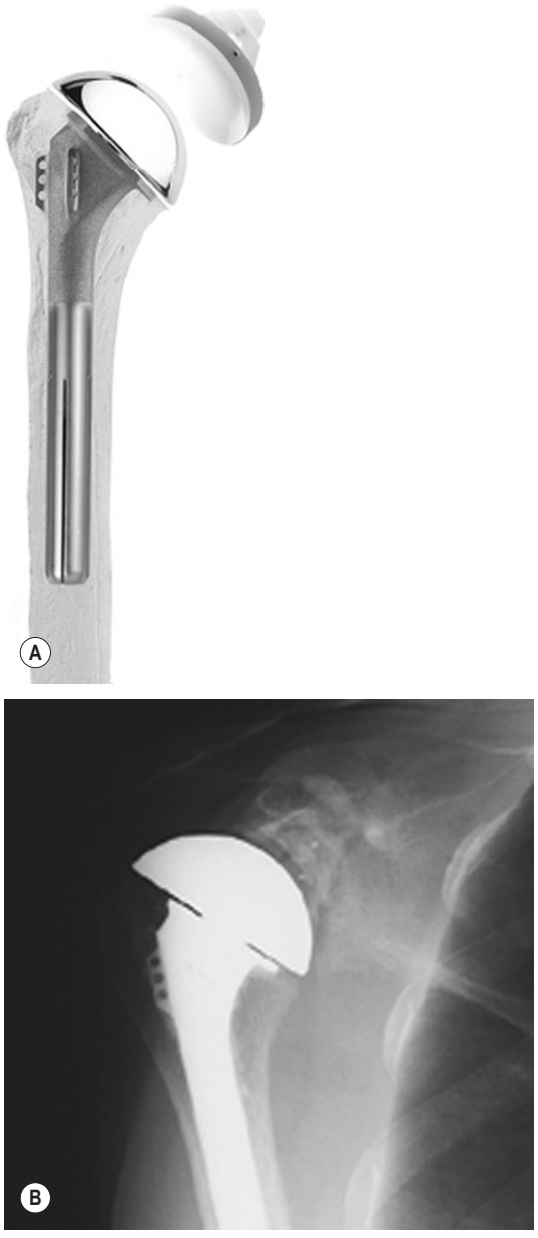


Figure 23.2 Shoulder replacement showing humeral and glenoid components and the appearance on X-ray. *(Reproduced by kind permission of Adam C. Gaines.)*

TOTAL ELBOW ARTHROPLASTY

Elbow joint replacement is a more recent and much less common operation. Although the number performed remains small, it is regarded as a well-established surgical procedure. It is performed usually within specialist orthopaedic centres for patients with marked degenerative changes predominantly from RA, but also from OA or trauma. Usually, all conservative management strategies have failed leaving the patient with significant pain, disability and a poor quality of life.

The joint consists of two metal stems cemented into the humerus and ulna, joined by a metal and plastic hinge. The postoperative regime depends on the surgeon's preference; some patients are splinted for a few weeks prior to mobilisation. Normal function usually starts to resume after 12 weeks. There will always be a carrying-weight restriction on people with a total elbow arthroplasty.

Figure 23.3 The hands of a person with rheumatoid arthritis showing the common deformities of ulnar drift and subluxation of the metacarpophalangeal joints.

THE HAND

RA can affect any joint in the body, but it is particularly devastating to the complex collection of joints and intricate soft tissues that make up the hand (Figure 23.3). Note in Figure 23.3 the ulnar deviation of the fingers and the more functionally debilitating deformity of the volar subluxation of the metacarpal heads that robs the flexor tendons of a proportion of their power, thus weakening grip. The multi-disciplinary team (MDT) within specialist hand units assess, surgically treat and rehabilitate patients to regain hand function.

Key point

A principal role of the upper limb is to allow for maximum use of the hand. Without a functioning hand the rest of the upper limb becomes mainly a component of balance.

The most common surgical procedure is the replacement of the MCP joint by a silastic flexible hinge (Figure 23.4), developed by Swanson. A flexible implant arthroplasty is different in principle from other joint arthroplasties in that the implant acts as an inert flexible spacer, which is quickly surrounded by a layer of synovial tissue. The new tissue remains in contact with the implant and surrounding this a stronger capsule develops.

Figure 23.4 Insertion of a Swanson silastic metacarpophalangeal flexible implant. *(Reproduced courtesy of Wright Medical Inc.)*

As well as excising the destroyed joint surfaces and inserting the flexible hinge, the surgeon must release and rebalance the soft tissues crossing the joint if function is to be restored.

Key point

Swanson devised an equation to explain the process: bone resection + implant + encapsulation = new joint (Swanson et al. 1978).

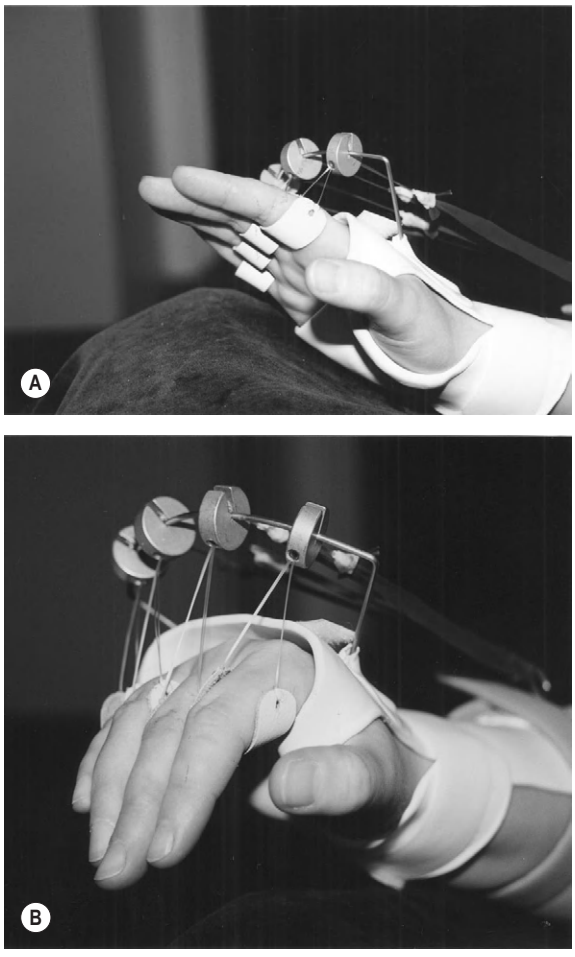


Figure 23.5 (a) Dynamic extension splint (outrigger) in the resting position. (b) Flexion at the MCP joints against the resistance of the elastic bands.

Figure 23.6 Splint to protect against recurrence of ulnar drift. Protection is particularly important in strong gripping activities.

Postoperatively, the short-term aims are:

- to achieve a functional arc of movement;
- to protect soft tissue repairs;
- to prevent further deformity at the affected joint and the development of secondary deformities/ pathological change in the related musculoskeletal system;
- to strengthen weak muscles;
- to re-educate function;
- to teach specific joint protection techniques.

These aims are achieved by early controlled movement, which stimulates the formation of a strong, but flexible, capsule around the implant. The most common way of controlling the movement is by the use of a dynamic extension splint (DES), often referred to as an 'outrigger' (Figure 23.5), although in recent years static splinting regimes have also been developed (Figure 23.6).

In the longer term, functional aims are achieved by strengthening exercises, re-education of pulp–pulp pinch and education in joint protection techniques.

When treating RA patients following surgery, always remember that they have a systemic disease and postoperative regimens often need to be individually tailored.

Other upper limb arthroplasties are shown in Figures 23.7–23.10, which show photographs of the radial head, wrist and finger, and trapezium arthoplasty photographs and X-rays.

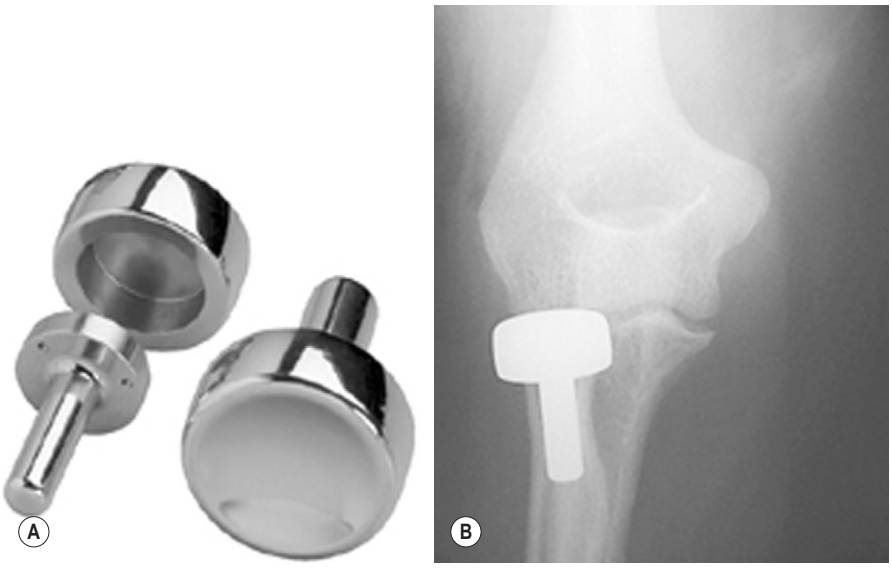


Figure 23.7 Radial head arthroplasty and the appearance on X-ray. *(Reproduced by kind permission of Adam C. Gaines, Wright Medical Technology, TN, USA.)*

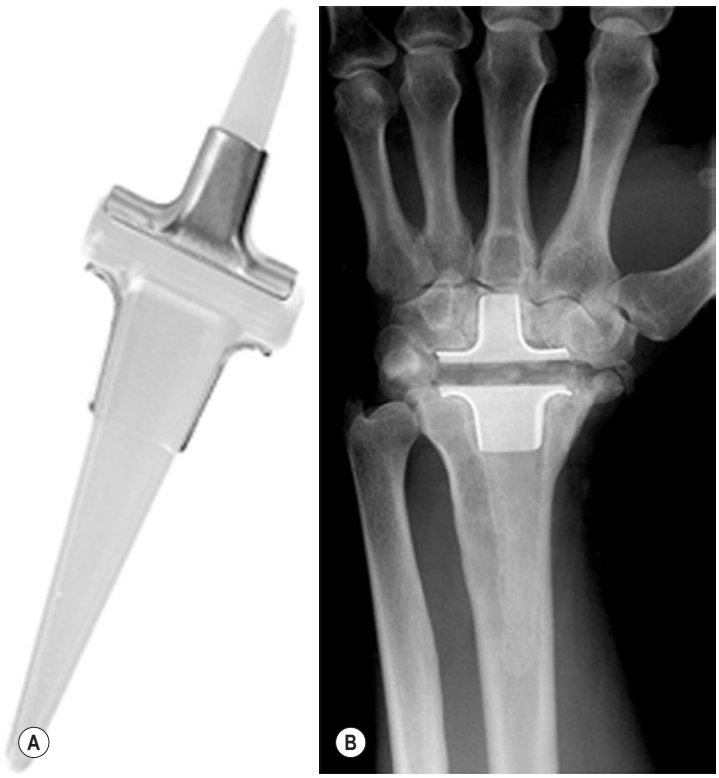


Figure 23.8 Wrist joint arthroplasty and the appearance on X-ray. *(Reproduced by kind permission of Adam C. Gaines, Wright Medical Technology, TN, USA.)*

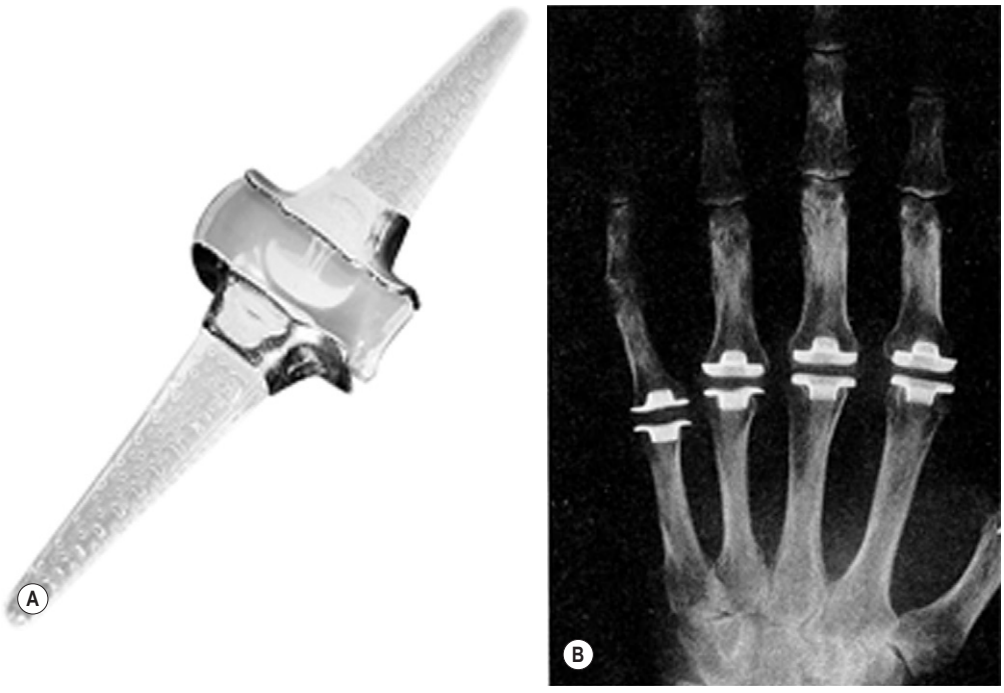


Figure 23.9 Swanson finger replacement and the appearance on X-ray. *(Reproduced by kind permission of Adam C. Gaines, Wright Medical Technology, TN, USA.)*

Figure 23.10 Trapezium replacement. *(Reproduced by kind permission of Adam C. Gaines, Wright Medical Technology, TN, USA.)*

LOWER LIMB ARTHROPLASTY

The aims of lower limb joint replacement are:

- to relieve pain;
- to improve the range of motion at the joint;
- to improve the functional ability of the individual;
- to improve the person's quality of life;
- to prevent further deformity.

Key point

Lower limb joint replacement began in the 1950s. Sir John Charnley refined and researched the unconstrained low-friction hip arthroplasty that today is the gold standard against which all other joint arthroplasty is compared. Nowadays, the National Joint Registry for England and Wales monitors the performance of hip, knee and ankle replacements (www.njrcentre.org.uk).

Hip joint replacement is a major, yet commonplace, orthopaedic procedure. The main pathologies that lead to replacement surgery are OA, RA, congenital dislocation of the hip, trauma (acute or longstanding), avascular necrosis of the femoral head and infection. Revision surgery with bone grafting is becoming more commonplace (both for early and late failure) and specialist centres offer ever more complex procedures including complex hip reconstruction and the use of custom made implants.

These pathologies and their respective drug therapies can lead to varying bone structure that the surgeon has to allow for at operation. For example, in the rheumatoid joint the bone tends to be 'soft', whereas if the patient has had previous trauma or surgery (e.g. femur or upper femoral osteotomy) then the bone is more 'hard'. Thus,

within the generalisation that tends to occur with a known procedure with a standardised rehabilitation protocol, individualised care is essential to achieve optimum success for the patient.

Modern implants aim, as far as possible, to replicate the surfaces of the joint to be replaced. Experience has shown that failure to fully understand joint motion and to engineer components capable of tolerating the complex interplay of forces generated by muscle pull (a three-dimensional effect), ligaments, acceleration, deceleration, weight and gravity will result in implant failure, pain and disability. There has also been much research into materials that are biologically inert, hard-wearing and with minimum coefficient of friction. Today, this has led to the use of components manufactured from metal alloys, high-density polyethylene and ceramics. Fixation of the components within the bone is, again, a complex issue, but most success is through pressurised cementing techniques or the use of hydroxyapatite-coated implants that enable a fibro-osseous fixation to develop as part of the bone healing process.

Although a very small percent age of patients will fail within the first year from recurrent dislocation or infection, an increasing number of patients are now requiring revision surgery owing to implant failure from wear over time. The expected life of a joint replacement should be 10–15 years, with many hip replacements lasting longer than 20 years. The main factors determining implant life are surgical skill, bodyweight, lifestyle demands and levels of high-impact stresses through the joint.

Primary modular metal and plastic hip replacements are still very commonplace (separate metal alloy stem, neck and ball and polyethylene socket) (Figure 23.11), although with the new materials available surgeons now have more choice, enabling a more customised implant. A variety of techniques are also used to surgically approach the hip that can impact on early rehabilitation goals. The relatively recent development of the metal on metal hip resurfacing procedure has proved popular with the younger, active patient, although the British Orthopaedic Association is currently advising patients to have regular follow-up as there has been a small, but significant, proportion of patients developing a reaction to potential microscopic metal debris accumulation.

Many objective assessments have been devised to assist with patient selection, particularly for the hip and knee, in order to quantify the severity of a patient's problem and general practitioners (GPs)/extended-scope practitioners may use a score threshold for referral to an orthopaedic surgeon. Until then, most patients will have had drug therapy, minor procedures where indicated (particularly in the knee) and physiotherapy, including self-management programmes. Apart from therapeutic arthroscopy, joint replacement (sometimes partial replacement in the knee) is the only option that will allow a return to an improved quality of life that is, on the whole, pain-free with good function. Ankle replacement is less common and very dependent upon accurate assessment by the surgeon, particularly of the hind foot, and may require additional pre-replacement surgery.

Factors taken into account by the surgeon when listing a patient for surgery include:

- severity of disease – onset, progress, other joints affected, investigations;
- pain and sleep disturbance;
- disability – effect on work and lifestyle;

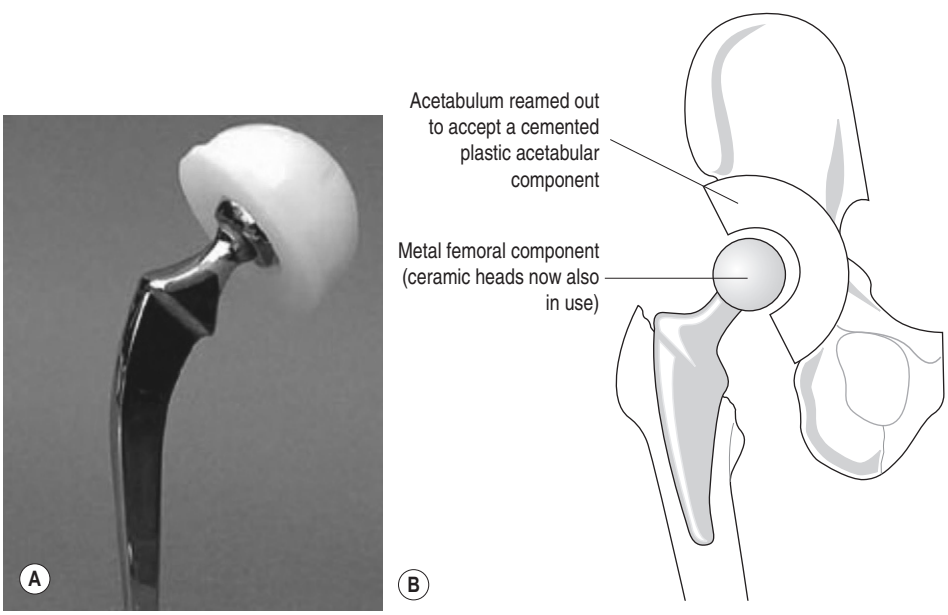


Figure 23.11 Total hip replacement. *(Reproduced by kind permission of Medical Models Ltd, UK.)*

- age – given the life expectancy of the implants;
- weight – keeping the stresses through the implant as low as possible;
- emotional stability – outcome less successful after a personal shock or bereavement;
- cognitive function.

Once listed for joint replacement, any underlying medical condition must be optimised prior to surgery via the pre-operative clinic. The anaesthetist usually reviews patients with significant medical history, decides the level of anaesthetic risk and, if possible, plans appropriate anaesthesia with the patient. General anaesthesia is less common – spinal anaesthesia with effective pain management enables more rapid mobilisation and helps to decrease the risk of deep vein thrombosis (DVT).

Pre-operatively the physiotherapist, along with the occupational therapist and the nurse, should prepare the patient for the procedure and help set their expectations for discharge and recuperation – commonly through information and education groups, although an individual session may be required by patients with complex needs.

Complications of joint replacement in the short-term are likely to be secondary to having a surgical intervention: DVT, pulmonary embolism, wound infection, heart dysfunction, paralytic ileus and bleeding. Specific early orthopaedic complications include dislocation, deep infection, neuropraxia and haematoma.

In the long-term the complications include scar sensitivity, dislocation, joint infection and implant loosening.

TOTAL HIP REPLACEMENT

The hip is the largest and deepest joint in the body. It takes the form of a multiaxial spheroidal joint with three degrees of freedom of movement with high levels of congruency (stability and surface area for stress transmission) and extensive range of movement.

The implant

Where a small femoral head is used the procedure is known as the 'low-friction arthroplasty' (LFA), as designed by Sir John Charnley.

The hip joint can be approached through a variety of incisions: commonly a posterior approach requiring detachment of gluteus medius and minimus or a lateral approach requiring osteotomy of the greater trochanter. The femoral neck is then divided, the joint dislocated and the head removed. The femoral canal and acetabulum are reamed down to fresh, bleeding bone and prepared for component implantation. Cavity size depends on the fixation technique. Component fixation is achieved either by (1) using pressurised cement (that can be available impregnated with antibiotics) to fix both socket and femoral components or (2) hammering the components into place. The hip is trialled for stability and leg length before definitive placement and closure. The gluteal tendons are reattached to the greater trochanter in the posterior approach and the greater trochanter reattached using a specialised wiring technique in the lateral approach (Figure 23.12). Soft tissues are closed in layers. Drains may or may not be used and dressings are applied. The patient is roused and transferred to the recovery ward. Once stable the patient returns to the ward. Pain relief is vital for early rehabilitation and safe swift discharge – to the care of a hospital at home team where available – at day 2–4.

These differences usually dictate rehabilitation protocols with the osteotomy usually requiring protection for a longer period. Always follow the surgeon's protocol unless informed otherwise or there is a change in the patient's condition.

Complications of hip replacement

All surgery is not without potential complications. Hip replacement surgery is major surgery and the general major risks apply. Specifically in hip replacement complications can be intra-operative, early postoperative and late. During surgery there is risk of nerve and blood vessel damage. Early postoperative complications include dislocation (3.9%), embolism (0.9%) and deep infection (0.2%). See Table 23.3 for early complications and preventative strategies. Late complications, i.e. after six months, include aseptic loosening (4.3%), recurrent dislocation (1.1%) and deep infection (0.3%).

Key point

It is vital for the physiotherapist to understand the procedure that the person is about to undergo, and the likely routine to be followed. Watching the operation at least once is useful.

Figure 23.12 X-ray of a hip replacement.

Table 23.3 Complications of hip arthroplasty

Complication	Caused by	Solution
Anaesthetic risk (chest, heart)	Anaesthesia type	Careful preoperative anaesthetic assessment Appropriate physiotherapy assessment and treatment as required
Infection	Open surgery	Laminar flow theatres, exhaust suits, prophylactic antibiotics post-operation [24 hrs]
Dislocation	Difficult surgery, poor surgical technique, complex case, inherently unstable, e.g. if patient has had a cerebrovascular accident (CVA), the risk of subsequent dislocation may be higher owing to decreased muscular stability at the hip joint	Abduction wedge, no adduction/flex beyond 90 degrees, special restraining device attached to socket may be used in cases of previous dislocation
DVT/PE	Pelvic surgery, immobility Damage to the blood vessels during surgery	Prophylactic anticoagulants – sub cut or oral, early mobilisation, anti embolic stockings
Anaemia	Blood loss during/post surgery	Transfuse if haemoglobin (Hb) below 8 or patient symptomatic (check hospital protocol)
Swollen ankle	Ineffective muscle pump	Reassure, walk little and often, frequent rest in bed, ankle exercises, compression stockings may help
Arm/hand pain	Crutch walking	Time
Neck pain	Neck trauma due to neck being held in an extended position during the intubation [GA]	Time
Stiffness after immobility	Inflammatory exudate builds at rest	Reassure, walk little and often

The immediate postoperative period

The patient will have an abduction pillow in place to protect the new joint. Physiotherapy includes active exercises for feet and ankles, isometric hip and knee contractions of all muscle groups, chest physiotherapy as necessary, advice on positioning and mobility in the bed.

As soon as the patient is assessed as ready to mobilise, i.e. is orientated, has satisfactory observations, pain is controlled and motor control is established (for those patients with epidural/peripheral nerve blocks), the patient is assisted out of bed initially by two staff and instructed in movement techniques that prevent excess hip flexion, adduction and medial rotation, and safe mobilisation – usually partial weight-bearing using an appropriate aid (a walking frame or a pair of elbow crutches).

Initially, the patient will stand out of bed and take a few steps with crutches or another appropriate aid (usually partial weight-bearing). Walking distance is progressed daily until the patient is walking independently with crutches or a frame.

Routines vary, but patients usually commence sitting at 1–2 days (no adduction or flexion beyond 90 degrees), with stair practice before discharge if needed. Usually, patients remain on crutches for six weeks, at which time they progress to sticks, then full weight-bearing.

'Dos' and 'don'ts' must be stressed to the patient (Table 23.4) and an educational booklet provided as a reminder. It is also important that occupational therapists and other members of the MDT see the patient.

Suggested rehabilitation protocol following total hip replacement

Key point

All activity should be documented to the Chartered Society of Physiotherapist's (CSP) documentation standards. MDT care pathways can also be completed – these aid effective communication between all staff, ensure all goals are met and allow for ease of service audit.

Table 23.4 Dos and don'ts following total hip replacement

Follow your hospital instructions but generally

Don't

- Do not pick up any objects from the floor or reach into low cupboards. Use a reaching aid if possible.
- Do not cross your legs.
- Do not overbend your hip.
- Do not sit on low chairs, low stools, or low toilets.
- Avoid sitting on chairs without arms. You may need to use the arms of the chair to help you to rise.
- When sitting, standing or lying down, try to keep your foot pointing forwards, not rolling inwards.
- Move to the edge of the chair before you stand up (keep your operated leg fairly straight at the knee).
- Do not lie either side for 6 weeks.

Do

- Take your time when you get home.
- Rotate your activities, i.e. lie for short periods if you are very tired, walk little and often, sit for short periods.
- Set yourself small achievable targets with your walking, using your crutches as instructed.
- Pace yourself.
- Remember that you will get good days and bad days.
- It may be useful for you to keep a progress diary so that you can reflect back on your progress.

Day 1

There should be assessment by the physiotherapist of neurovasculomotor and respiratory systems. Active exercises for circulation and static exercises for muscle tone around the hip can be started, along with deep-breathing exercises. Notes and X-rays should be studied as soon as available. Note the surgical approach and any non-standard procedure. Mobilisation may commence, according to the surgeon's preference.

Day 2

Functional rehabilitation continues towards achievement of safe, independent mobility with walking aids. The physiotherapist assists and ensures hip protection at all times.

Day 3 onwards

Mobility is progressed on a daily basis. A stair assessment should be completed if possible. Advice is given prior to discharge concerning expected levels of activity until clinic review. No other specific exercises are required during this period. Regular and gradually extended walking practice is necessary in/outdoors. Ensure the emphasis is on hip protection and recovery (effective capsular scarring). There are three simple, golden rules:

- do not overbend the hip (flex beyond a right-angle);
- do not cross the leg over the midline (adduct beyond neutral);
- do not twist in either standing or sitting.

These rules apply for six weeks in all cases. If the trochanter is to be protected then the rules apply for longer and it is generally accepted that the patient can return to normal activities at three months.

After discharge

If the soft-tissue approach was used the patient may be issued with sticks and advised to gradually increase weight-bearing over the next six weeks. If the transtrochanteric approach was used then the site of bony union must be protected for 12 weeks.

A long-term rule is to avoid high-impact activities, as sudden high pressures cause the most wear to the plastic.

The patient may telephone with queries following discharge but must contact the GP or attend the accident and emergency department with any sudden significant pain or swelling.

Further physiotherapy/exercise may be required following clinic review and the appropriate referral can be made. Some centres offer outpatient rehabilitation programmes.

Rehabilitation following revision surgery

There is frequently augmentation of the acetabular and femoral bone required using bone grafting techniques and, in some cases, the use of screws and mesh to hold the graft in place. The graft acts as scaffolding for migrating osteocytes which then lay down new bone and incorporate the graft.

Depending on the reason for revision (commonly wear and loosening, fracture, infection, dislocation) and the complexity of the procedure, the physiotherapy as outlined above is modified appropriately in the light of the surgeon's instructions.

TOTAL KNEE REPLACEMENT

The knee is a complex joint whose stability is dependent on extra-articular ligaments, fascia and muscular control. Knee problems usually present with gross biomechanical deformity requiring soft tissue release at operation in order to regain biomechanical alignment and normal balanced soft tissue lengths.

The implant

The unconstrained implant uses metal-alloy components over the distal femur and proximal tibia. A high-molecular-weight (high-density) polyethylene bearing is inserted between . The patella may be resurfaced with a metal-alloy articulating button if necessary.

In the case of a severely deformed joint and, more commonly, in revision procedures, physical constraint to movement can be achieved by use of long-stemmed linked implants. The risk of failure secondary to its inability to move freely is less likely in the patient with low functional demand or is an accepted risk when surgical options are compromised.

The operation

Commonly, a tourniquet is applied, the leg is exsanguinated and the tourniquet tightened for a timed period. With the knee in flexion the surgical approach is, on the whole, by anterior skin incision followed by medial parapatellar incision through quadriceps expansion. The patella is reflected laterally and the joint exposed. Very rarely (and seen more in revision surgery) the tibial tuberosity is sawn off with a large piece of anterior tibial bone to allow improved access to the joint. Bone cuts and preparation followed by component trial is carried out. The femoral component is held in place by two short pegs into each condyle and the tibial component by a large single peg into the tibia. The pressurised cementing technique as described previously for the hip replacement operation can be used or uncemeted coated components hammered into the bone. Closure of the joint is in layers. The patient may or may not have drains inserted. A wool and crêpe bandage is applied to control oedema and a backsplint may be applied to maintain the knee in extension until quadriceps function is regained (Figure 23.13).

Suggested rehabilitation protocol following total knee replacement

The general postoperative physiotherapy is as previously described for hip replacement, except that the emphasis is on regaining quadriceps control. If the patient has had a femoral block for pain relief, active quadriceps function may take up to 24 hours to return. Mobilisation and the start of the exercise programme may commence on days 1–2. The patient starts to walk with two crutches and is full or partial weight-bearing for six weeks. Transfer on to sticks is possible at any point during this time also. Exercises are prescribed according to the surgeon's routine and the physiotherapeutic assessment of the patient. The splint and bandage must be removed prior to physiotherapy. It is vital that exercises be practised very regularly – little and often, almost hourly at first, progressing to several times

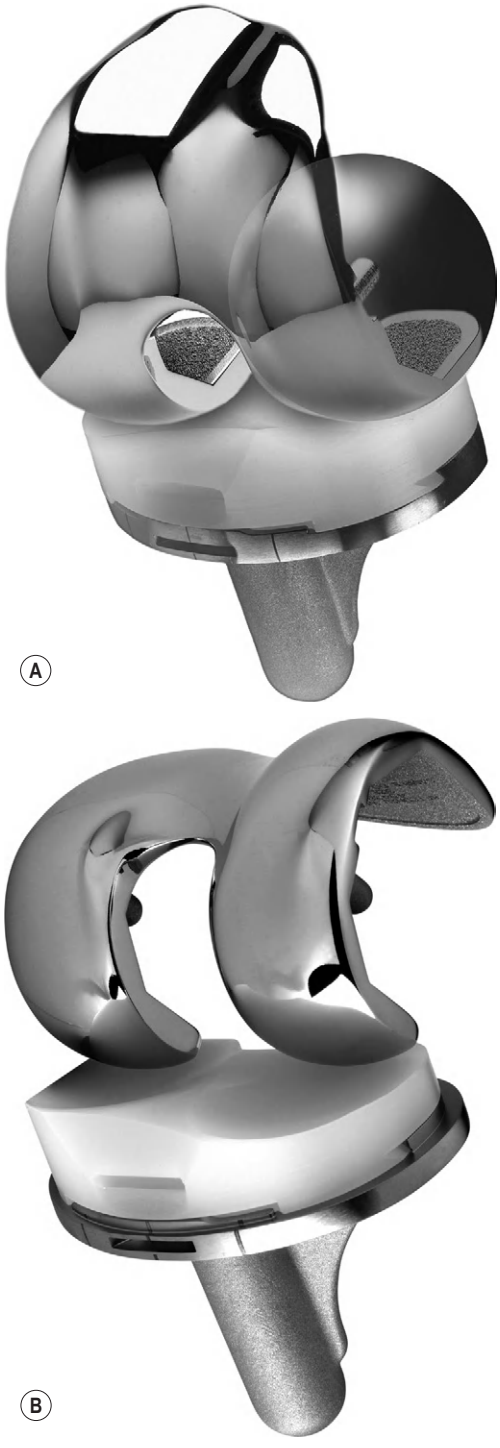


Figure 23.13a,b Total knee replacements. *(Reproduced courtesy of Wright Medical Technology, Inc.)*

per day of differing exercises. It is paramount that quadriceps function be regained and therefore the control and protection of the joint established using simple closed-chain quadriceps and slow stretch knee flexion exercises. It is important to provide the patient with a written exercise programme tailored to the individual's needs that can be adjusted and reviewed as progress continues. Flexion is usually hampered by pain, fear, bruising and swelling. Physiotherapy should therefore include help in the control of these symptoms by reassurance, coordination with analgesia, cryotherapy and careful assisted flexion. The patient can be instructed in the safe application of ice at home. Do not forget that full extension is a vital component of knee joint function.

Length of hospital stay is usually 3–4 days, by which time the patient should be functionally independent and progressing with rehabilitation. Ideally, the range of movement should be 0–90 degrees with active knee control. On discharge most patients are referred for outpatient physiotherapy to review progress, continue with assisted exercises or start more advanced rehabilitation.

TOTAL ANKLE REPLACEMENT

Total ankle replacement is the 'youngest' of the lower limb joint replacements. Attempts were made in the 1970s but were unsuccessful. There has now been success with a semi-constrained implant; however, this success will depend on the health of the subtalar joint below it. Sometimes (particularly in people with RA), the diseased subtalar joint is fused prior to the total ankle replacement to give the optimum outcome. Also, in this anatomical region the integrity of the circulation is vital and great care is taken in the selection of a patient in whom both circulation and skin can withstand the stress of operation and support the demands of the healing process.

The articular surfaces of the tibia and the talus are replaced with metal alloy implants. The talar implant is centrally ridged forward to back to accommodate the sulcus on the inferior surface of the polyethylene bearing inserted between them (Figure 23.14). This allows for flexion and extension and decreases the risk of malleolar fracture by medial slip of the bearing. The surgical approach to the joint is via a 10-cm anterior ankle incision. Care is taken to avoid the anterior tibial neurovascular bundle. The bones are cut and prepared as previously described. The implant is usually uncemented; the components being hammered into place. The tissues are closed in layers and compression dressing applied. The patient is roused and nursed as described previously and the limb is maintained in elevation.

Figure 23.14 Ankle joint replacement. *(Reproduced by kind permission of De Puy Orthopaedics Inc., IN, USA.)*

Suggested rehabilitation protocol following total ankle replacement

Postoperative physiotherapy is as previously described for hip and knee replacements, except that routine ankle exercises are avoided and toe exercises substituted. It may be necessary for the physiotherapist to ensure dorsiflexion to a right-angle (plantigrade) and, where this is difficult, to show the patient an appropriate stretch exercise with a strap.

Mobilisation is usually on day 2 or 3 when the swelling is controlled and the compression dressings removed, according to the surgeon's instructions. A protective knee-length plastic boot that can be removed for wound inspection can be fitted and the patient mobilised to partial weight-bearing with elbow crutches for functional requirements only (the leg is rested in elevation otherwise). Techniques to manage stairs/steps/ramps are taught.

The length of stay is usually 3–4 days. The patient is reviewed in the clinic at 2–3 weeks for removal of sutures and the plastic boot. Exercises can be started at this point to facilitate rehabilitation, including Achilles tendon stretches, muscle strengthening and balance function. Specific mobilisation of the ankle is avoided.

FURTHER READING

Atkinson, K., Coutts, F., Hassenkamp, A.-M., 1999. Physiotherapy in Orthopaedics: a Problem Solving Approach. Churchill Livingstone, Edinburgh.

Birch, A., Gwillian, L., 2000. Rheumatoid arthritis and its effects on the hand. In: Salter, M., Cheshire, L. (Eds.), Hand Therapy Principle and Practice. Butterworth-Heinemann, Oxford.

Simmen, B.R., Allieu, Y., Luch, A., et al., (Eds.), 2001. Hand Arthroplasties. Martin Dunitz, London.

Skirven, T.M., et al., 2011. Rehabilitation of the hand and upper extremity, 2-Volume set, sixth ed. Mosby, Philadelphia.

REFERENCES

Brander, V.A., Malhotra, S., Jet, J., et al., 1997. Outcome of hip and knee arthroplasty in persons aged 80 years and older. Clin Orthop 345, 67–78.

Iannotti, J.P., Williams, G.R., 1998. Total shoulder arthroplasty: factors influencing prosthetic design. Ortho Clinics N Am 29, 377–391.

Neer, C.S., Watson, K.C., Stanto, F.J., 1982. Recent experience in total shoulder replacement. J Bone Joint Surg 64A, 319–337.

Swanson, A.B., Swanson G de, G., Leonard, J., 1978. Postoperative rehabilitation program in flexible implant arthroplasty of the digits. In: Hunter, J.M., Schneider, L.H., Mackin, E.J., et al., (Eds.), Rehabilitation of the Hand. CV Mosby, St Louis, pp. 477–481.

Swanson, A.B., Swanson G de, G., DeHeer, D.H., 2000. Small joint implant arthroplasty: 38 years of research experience. In: Simmen, B.R., Allieu, Y., Luch, A., et al., (Eds.), Hand Arthroplasties. Martin Dunitz, London.

Chapter 24

Physiotherapy management of Parkinson's and of older people

Bhanu Ramaswamy and Paula McCandless

INTRODUCTION

Physiotherapists working with older adults and people diagnosed with Parkinson's are required to have knowledge of core physiotherapy areas. Effective practice depends on your ability to apply the knowledge appropriately in context of the individual's physical, psychological and sociocultural status and need.

In this chapter, physiotherapy management of Parkinson's and of older adults will be explored together; however, Parkinson's should *not be* regarded as a condition affecting only the older population. Dealing with either population highlights vastly different needs and solutions, yet the situations also offer fundamentally similar challenges. These include working within an interdisciplinary environment; utilising skills that deal with complex bio-psycho-social factors; applying knowledge in managing a process of progression (usually deterioration); and relating changes to the ageing process, to a neurological presentation, or to both. For these reasons, the two topics have been integrated into one chapter.

Information has been integrated through the chapter where evidence and practice can be transferred between the two groups. If something is of particular relevance to either the older adult population or to Parkinson's, this has been specified.

Finally, as the book aims to assist your development of physiotherapy practice, the chapter focusses more on the practical aspects of assessment and management than on demography and pathology. All issues have signposts to easily accessible information for you to pursue.

Key point

Irrespective of a person's condition or age, physiotherapy has a role to play in enabling both health and well-being-related lifestyle choices. This may be through education, through the restoration of function, maximising the potential of the person or by minimising the impact of decline for people with a deteriorating condition.

ABOUT AGEING

Ageing describes the process of growing old, with normal ageing being considered a state that occurs without disease. Worldwide populations are growing as human life expectancy increases. The proportionate increase of older adult numbers within the populations has been named 'The Greying of the Nations'. In light of this issue, over the last 20 years, various countries have supported research to investigate their older populations so that they might estimate and provide the resources required to maintain as healthy a population as possible.

On average, the 'older adult' label spans a 20–30 year period with an arbitrary division into three ages of 'old':

- between 60 and 75 years = young old;
- between 75 and 85 years = old;
- those older than 85 years are considered the frail older population.

Table 24.1 Percentage of people aged over 60 years admitted to Northern General Hospital, Sheffield

Speciality Description (% >60 years)	1999	2001	2003	2005	2007	2009
Cardiology	57	59.0	60.8	60.4	61.9	62.1
Cardiothoracics	57	58.9	62.4	66.3	59.5	68.8
General/chest medicine	60	64.5	65.2	75.4	67.7	56.2
Heart transplant	27	28.0	35.4	55.8	53.3	10.0
Nephrology	42	43.3	50.1	56.5	59.8	63.8
Orthopaedics	40	41.4	44.5	45.2	45.6	45.9
Plastic surgery	27	31.3	30.3	31.6	29.4	35.4
Spinal injuries	18	21.9	23.8	21.3	22.1	26.0
Vascular surgery	61	64.4	59.0	51.6	53.9	60.5
Average	**37.54**	**37.49**	**40.1**	**41.11**	**43.12**	**46.88**

The variations between the populations in each age band require different approaches when considering the physical, psychological and social needs of people within the groups (DH 2001).

Thinking that the ageing population is only of concern to physiotherapists specialising in older people is unrealistic. The percentage of people aged above 60 years admitted to clinical specialities in a Sheffield hospital over a ten-year period (Table 24.1) illustrates a pattern of overall increase in the numbers of older people. It is important that these people receive the same level of service and care as anyone else, irrespective of age.

Recommended reading

- National Statistics Online at: http://www.statistics.gov.uk/. This site provides information such as population demographics across the UK, changes in lifestyle choices, health and life expectancy.
- The National Service Framework for Older People (DH 2001). The Standards agreed for this 10 year programme of action ensure fair, high quality, integrated services for older people; support independence and promote good health, respect, dignity and fairness (http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_4003066).
- Reports on *Global Action on Ageing* at: www.globalaging.org.
- The United Nations *Programme on Ageing* at: http://www.un.org/esa/socdev/ageing/index.html.

ABOUT PARKINSON'S

Parkinson's is a chronic, progressive neurodegenerative disorder of the central nervous system; it is the most common disorder of the basal ganglia and the second most common neurodegenerative disorder in the UK after Alzheimer's disease (Jones and Playfer 2004). The incidence increases with age and has no currently known cure.

The condition was described first as 'the shaking palsy' in 1817 by Dr James Parkinson, whose clinical description identified both the main motor symptoms and non-motor symptoms (Tables 24.2 and 24.3; adapted from Jankovic 2008: 368). The main area of basal ganglia dysfunction appears to stem from the loss of dopamine-producing cells in the substantia nigra, causing difficulty in the initiation of movement (internal cueing mechanism).

Symptoms differ from person to person and in the same person over time. The presentation of these varied symptoms has led people in the UK affected by Parkinson's (those diagnosed, their families and their carers) to campaign against the use of the term 'disease'.

Tremor

The typical Parkinson's rest tremor occurs at 4–6 Hz. It begins unilaterally, described in the hand as a 'pill rolling tremor' (resembling a pill being rolled between the thumb and fingertips) and can occur in the feet, jaw, chin and lips. It is distinct from 'essential tremor' and varies during the course of the condition and within the Parkinson's population.

Table 24.2 Primary motor symptoms (TRAP) or cardinal signs of Parkinson's

*T*remor (rest)
*R*igidity
*A*kinesia (including bradykinesia)
*P*ostural instability (loss of postural reflexes)

Clinical note

Parkinson's is a neurodegenerative disorder characterised by tremor, bradykinesia and postural instability (leaving them at greater risk of falls than the general population), and is associated with a number of non-motor features, including cognitive impairment, neuropsychiatric signs and symptoms, dysautomnia and sleep disorder (NICE 2006; Chaudhuri et al. 2007).

From the point of view of a physiotherapist, the progressive symptoms of Parkinson's can affect the quality of life of the individual, their carers and/or their relatives. Increasing impairment and disability results in a reduced participation in societal roles. Speech impairment can be particularly burdensome, increasing social isolation; physical activity levels are reduced and cardiac symptoms, such as heart rate variability and sympathetic denervation of the heart, affecting the response to mobility and exercise.

Table 24.3 Secondary motor and non-motor symptoms

Secondary motor symptoms of Parkinson's	Freezing, shuffling gait, festination Dysarthria (motor speech disorder) Hypomimia (reduced facial expression) Dysphagia (difficulty swallowing) Dystonia (movement disorder-spasms) Sialorrhea (excessive secretion of saliva) Glabellar reflexes (blinking on tapping of the forehead) Micrographia (small handwriting)
Non-motor symptoms of Parkinson's	Autonomic dysfunction, cognitive neuro-behavioural abnormalities. Includes dementia, depression, psychosis, impulse control disorder (Weintraub 2010) Sleep disorders, sexual problems Anosmia (lack of smell) Gastrointestinal dysfunction Constipation Urinary incontinence Weight problems Sensory abnormalities (paraesthesia, pain)

(Adapted from Jankovic 2008.)

Rigidity

A form of hyper-tonicity causing stiffness or inflexibility of the muscles from loss of normal reciprocal inhibition – is thought to be a result of excessive excitation of alpha motor neurones that are normally inhibited by the basal ganglia. You may observe rigidity as reduced arm swing during walking or reduced heel strike owing to rigidity in the calf. Rigidity can cause pain and cramping, impoverished movement and added internal resistance when undertaking exercise. It is described as:

- lead pipe rigidity – consistent resistance to movement throughout the full joint range;
- cog-wheel rigidity – 'jerky', inconsistent resistance to movement throughout joint range, occurring distally (wrist and ankle) if an existing tremor interferes with movement.

Akinesias

There are several forms of akinesias:

- *Bradykinesia* (slowness in movement performance or loss of spontaneity of movement) – is the most characteristic feature of Parkinson's, although the pathophysiology has not been fully identified. It is thought to result from a reduction in normal motor cortex activity facilitated by a reduction in dopaminergic function (dopamine deficiency levels). The slowness may be demonstrated through difficulty with planning, initiating, executing and performing sequential and simultaneous tasks, and loss of fine motor control. If there is a swallowing impairment, presentations of bradykinesia might include drooling, loss of spontaneous movements and gesturing.
- *Hypokinesia* – a decrease in amplitude may, for example, account for lack of arm swing during walking, loss of facial expression and reduced blinking.

These forms of akinesia cause reduction in movement range and velocity, making automatic and repetitive movements difficult.

- *Dyskinesias* – are abnormal, involuntary movements and often are a common side effect of medication. They can become disabling, adding to the risk of falls and injury.

Postural instability

Postural instability equates to the loss of postural reflexes essential for normal movement and alongside freezing of

gait, is a major cause of falls and hip fractures in those with Parkinson's; it usually manifests in the later stages of the condition. The 'pull test' of the Unified Parkinson's Disease Rating Scale (UPDRS) or 'retropulsion test' is used to assess this clinical feature by pulling the person being tested backwards or forwards by their shoulders (or hips), and noting their response to displacement. An abnormal response (positive postural instability) is identified if no postural correction is observed or more than two steps backwards are taken.

Combinations of *TRAP* (tremor, rigidity, akinesia, postural instability) can lead to secondary motor symptoms. A classic one is the 'Simian' posture where the spinal curvature results in a flattened lumbar lordosis, rounded shoulders and a poking chin (hyperextended cervical spine). The consequent forward flexed upper body results in the 'ape-like' posture. Examples of secondary motor and common non-motor symptoms are tabulated above (Table 24.3).

Diagnosis

Currently, no reliable test exists for Parkinson's so diagnosis is determined through the history and clinical signs of at least two of the four cardinal motor symptoms described earlier using the UK Brain Bank Criteria and investigations (scans) (NICE 2006).

Parkinson's is the most common presentation within a collection of motor system disorders known as 'Parkinsonism' (Table 24.4). The various disorders can be identified using positron emission tomography (PET) and structural magnetic resonance imaging (MRI) scanning. They are also referred to as 'Parkinson's Plus' as they involve a wider area of the nervous system than idiopathic Parkinson's (SIGN 2010: 11–13).

Medication is less effective or may have to be withdrawn for people with Parkinson's Plus syndromes, and the course and presentation differs from those with 'idiopathic' Parkinson's described in this chapter.

Clinical note

To minimise misdiagnosis and incorrect treatment regimes, a person with Parkinson's should be seen by a specialist multi-disciplinary team, with the opportunity for review by a specialist neurologist or geriatrician, and referral to a Parkinson's Nurse Specialist.

Aetiology and epidemiology

There are approximately 120,000 people with Parkinson's in the UK and an estimated 6.3 million worldwide. The average age of onset is 60–65 years. The term 'idiopathic' is used when there is no known cause of the Parkinson's, as no known single, universal causative factor has yet been identified. Several theories exist – the most common of which proposes Parkinson's to result from a variety of pathological processes in addition to influencing factors, such as the environment, the ageing process and genetics.

Table 24.4 Parkinsonism or 'Parkinson's Plus' syndromes

Primary neurodegenerative disorder	Progressive supranuclear palsy (PSP) Multiple systems atrophy (MSA) Cortico-basal degeneration (CBD)
Secondary neurodegenerative disorder	Multiple cerebral infarction Drugs and environmental toxins Trauma from boxing

Neuropathology

The 'ascending hypothesis' of idiopathic Parkinson's conceptualises a pathological progression from the lower brainstem (pre-clinical stage) to the midbrain (substantia nigra clinical stage) and cortex (end-stage) (Braak et al. 2003; Yanagisawa 2006). Extra-pyramidal system dysfunction leads to the loss of the internal 'go' setting, essential for movement initiation that becomes evident after the pre-clinical stage.

Pathologically, Parkinson's is characterised by a reduction of neuromelanin cells in the brainstem, primarily dopamine neurones in the substantia nigra, but also in the corpus striatum (caudate nucleus and putamen). People with Parkinson's will have lost over 70% of dopamine-producing cells before motor signs (TRAP) are visible. This is thought to cause an inability to direct and control movements (rigidity and loss of inhibition of tremor), while increasing inhibition to the thalamus (bradykinesia). A lack of inhibition mainly of the reticulospinal and vestibulospinal pathways results in excessive contraction of postural muscles. The imbalance of inhibition and excitation result in the classical clinical features of Parkinson's.

Classification

Parkinson's is classed in various ways. The most common method is by severity according to the (modified) Hoehn and Yahr scale (1967), categorising people along a continuum from asymmetry at Stage 1 to Stage 5 palliation. Physiotherapy referrals usually occur when signs of poor balance develop and possibly a fall from Stage 3 onwards.

MacMahon and Thomas (1998) have provided a clinical staging classification, allowing for periods of deterioration during illness, but regaining the ability on recovery. Details of both these can be read in the *Parkinson's Professional Guide* (PDS 2007).

In light of evolving information regarding the pre-clinical development of Parkinson's, Stern et al. (2012) have proposed the following phases to reflect the progress of the condition (Table 24.5).

A cluster analysis of presentation at diagnosis has provided detail according to a person's age, cognitive state (presence of dementia or not) and symptom dominance (van Rooden et al. 2010). The four subtypes of Parkinson's are identified below.

- Early disease onset (25%) – longest duration until death, delay before falls or cognitive decline.
- Tremor dominant (31%) – same life expectancy, falls history and hallucinations as non-tremor dominant.
- Non-tremor dominant (36%) – strong association with cognitive impairment (Lewy body pathology).
- Rapid disease progression without dementia (8%) – often tremulous onset in older people, early depression, midline symptoms, increased mentation, freezing and activities of daily living (ADL) sub-scores according to Part I and II of the UPDRS.

Table 24.5 Phases of development in Parkinson's (Stern et al. 2012)

Phase 1	Pre-clinical PD	PD-specific pathology assumed to be present, supported by molecular or imaging markers, no clinical signs and symptoms
Phase 2	Pre-motor PD	Presence of early non-motor signs and symptoms due to extranigral PD pathology
Phase 3	Motor PD	PD pathology involves substantia nigra leading to nigrostriatal dopamine deficiency sufficient to cause classic motor manifestations followed by later non-motor features due to extension of the pathology

The importance of such classifications for physiotherapists is evident when decision-making about progression and intervention have to be considered, and equipment and support networks put into place.

Recommended reading

For more details about the demographics of Parkinson's, of diagnostic processes and of neuroanatomy, read the following or look up information on the following websites:

- the National Institute for Health and Clinical Excellence (NICE) Guidelines 35 – Parkinson's disease: Diagnosis and management in primary and secondary care (2006) (http://www.nice.org.uk/nicemedia/live/10984/30088/30088.pdf);
- The Parkinson's UK website (http://www.parkinsons.org.uk/default.aspx). This site will also give you a current *Glossary of useful terms;* utilising this will aid your knowledge and understanding of choices for the management of people affected by Parkinson's;
- The European Parkinson's Disease Association website (http://www.epda.eu.com/).

The International Classification of Functioning, Disability and Health

The International Classification of Functioning, Disability and Health (ICF) (WHO 2001) offers a model for consideration of multiple impairments, relating them to domains that guide assessment, goal-setting and treatment-planning (Figure 24.1).

Impairments often impact on activities of daily living and how the person can participate in societal roles in later life (Izaks and Westendorp 2003), and the ICF framework considers issues in context. For example, environmental (physical and attitudinal) and personal (medication, support) factors which can act as barriers or facilitators in the analysis and subsequent planning of

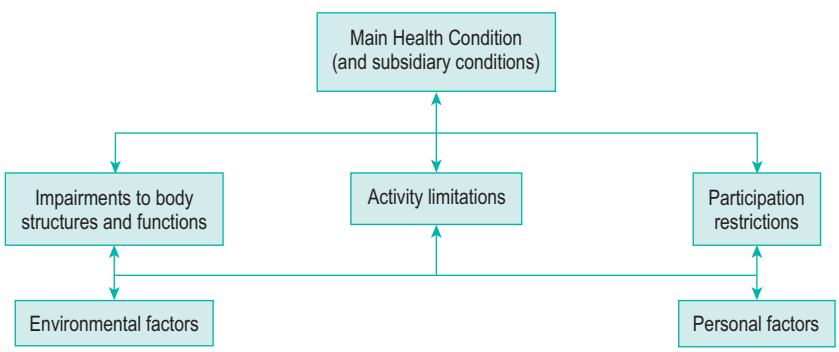


Figure 24.1 Interactions between the components of the International Classification of Functioning (ICF). *Permission to reproduce granted by the World Health Organization.*

appropriate intervention. Consideration of the target level (whether an impairment, activity or participation issue) ensures that therapeutic management meets the current status and needs of the individual. An appreciation of how people live with ongoing (a set of) conditions using such a bio-psychosocial and partnership approach to practice is key to achieving person-centred management.

ASSESSMENT

Prior to physical assessment, consider the following:

- *Consent and confidentiality*. People within these groups (older adults and people with Parkinson's) may fall into the category of 'vulnerable adults' and, as circumstances alter over time, their needs and support systems will change. Consent may have to be elicited differently if, for example, speech deteriorates in a person with Parkinson's or if an older person has a stroke and verbal communication is no longer reliable. Sometimes tensions arise between the *individual's rights* and the *professional or organisation's responsibility* to that individual or the well-being of the family. Examples include dealing with confused individuals at risk of injury who insist on returning home or a struggling carer whose health is at risk yet refuses the help of strangers in their house. As long as the person has capacity to understand risks and take responsibility for them, the choice is theirs.
- *Ethics and law* need considering, especially relating to end-of-life decisions or when people have reduced mental capacity requiring full discussion with family and team (Dimond 2009).
- *Confidentiality*. Awareness of suspected elder abuse or a more serious underlying medical problem is important. Ensure that issues to be discussed outside of the immediate ward/home environment receive the person's consent first and only then reveal information relevant to the consultation (Whiddett et al. 2006).
- *Recognising patterns of health and illness*. The relationship between age and disease lies along a continuum making it difficult to distinguish between them (Izaks and Westendorp 2003). For example, postural changes, stiffness and restricted activity are so often seen as a part of ageing that the rigidity and bradykinesia of idiopathic Parkinson's is missed.

Recommended reading

- The Mental Capacity Act (2005) with amendments to both the Capacity and Mental Health Act (2009) (http://webarchive.nationalarchives.gov.uk/+/www.dh.gov.uk/en/SocialCare/Deliveringadultsocialcare/).
- MentalCapacity/MentalCapacityAct2005/index.htm puts the needs and wishes of a person who lacks capacity at the centre of any decision-making process. Particularly relevant to discharge planning if conflict arises between the interdisciplinary team's judgement and the individual's choice.

Clinical note

Active or inactive pathological processes will alter over time, impacting on the individual's lifestyle. An awareness of pathological and normal physiological changes will assist your assessments and help you decide on best management.

It is easy to overlook how people are accepting and adapting to the challenges they bring. Although our priority is normally to deal with the physical disability, remember to consider the possible psychological consequences; the person can be referred to colleagues to ensure a holistic approach to person-centred care is maintained.

Recommended viewing for Parkinson's

Look up the following documents to help you formulate in-depth ideas about the condition:

1. The Royal Dutch Society for Physical Therapy *Guidelines for Physical Therapy in Patients with Parkinson's Disease* (Keus et al. 2004). It provides the best available evidence for use in clinical practice and can offer standardised guidance to physiotherapists (https://www.kngfrichtlijnen.nl/downloads/Parkinsons_disease.pdf).
2. The complete adapted version of the Dutch Quick Reference Cards (Ramaswamy et al. 2009) (http://www.parkinsons.org.uk/advice/publications/professionals/quick_reference_cards.aspx).
3. Information from the Association of Physiotherapists in Parkinson's Disease Europe (APPDE) can be accessed at: http://www.appde.eu/.
4. Information about cues and strategies to assist movement can be found on the RESCUE site (www.rescueproject.org). It is informed by multi-centre research trials undertaken to look into the impact of cues and physiotherapy.
5. *A Professional's Guide*, published by the Parkinson's UK team in 2007, has a chapter dedicated to physiotherapy assessment and intervention (http://www.parkinsons.org.uk/advice/publications/professionals.aspx).

Clinical note

AGILE is the clinical interest group of the Chartered Society of Physiotherapy (CSP) and is comprised of physiotherapists who work with older people.

The fundamental principles on which physiotherapy with older people is based are:

- disability is generally regarded as being a result of a pathological process or injury, not prima facie 'old age';
- the effects of biological ageing reduce the efficiency of the body's systems but, throughout life, optimum function is maintained in each individual by continuing to use these systems to their maximum capacity;
- physiotherapists have a key role in enabling older people to use a number of the body's systems fully to enhance mobility and independence;
- when neither improvement nor even maintenance of functional mobility is a reasonable goal, physiotherapists can contribute to helping older people to remain comfortable and pain-free;
- prevention of the development of problems in later life is through health promotion (CSP 2005; AGILE 2008).

Also be aware that people living with chronic illness continually shift their perspective over time between one of illness and one of wellness (Paterson 2001). Intervention for these variations requires the skill of a different professional (health or social) depending on the manifestation of symptoms.

The assessments and principles of therapy should follow suggested national protocols and guidance. Assessment of older people differs from younger people only in the differences in the body that occur with age; for people with Parkinson's, it is more the fact that symptoms may present differently over the course of one day.

A comprehensive subjective and objective assessment provides clarity of the clinical problem, ensuring the information acquired enables the right decisions to be made regarding the subsequent physical assessment. Access to notes prior to assessment (electronic or hard copy) will provide greater insight to the previous medical, and current drug and social histories.

Recommended reading for issues regarding older people

- *Myths about ageing*. There are many 'myths of ageing'. The WHO published a booklet for the International Year of Older Persons in 1999 to shatter these myths (http://whqlibdoc.who.int/hq/1999/who_hsc_ahe_99.1.pdf).
- *Demystifying the myths of ageing.* Another WHO booklet demonstrates how problems associated with old age can be positively tackled (http://www.euro.who.int/_data/assets/pdf_file/0006/98277/E91885.pdf).
- Visit the AGILE website (www.agile.csp.org.uk) for information about physiotherapy with older people, and to download a copy of standards and principles of assessment and intervention.

Clinical note

An informal consensus for AGILE (Ramaswamy and Jones 2005) provided four recommendations for physiotherapists working with people with Parkinson's. Physiotherapists should:

- locate the aim of the episode of care as being either (1) maintenance of the individual's current movement capability; (2) management of complex problems; or (3) palliative care;
- ensure their baseline assessment comprises a comprehensive set of data against which to monitor change with disease stage;
- use appropriate outcome measurement to evaluate the impact of a specific intervention;
- relate treatment strategies to problems identified at assessment.

Bear these in mind when you consider the assessment and management of Parkinson's.

Assessment should be:

- undertaken at different points in the day (medication cycle) if possible to capture variations in presentation. This would reflect a 'best' and 'worse' episode, highlighting functional difficulties experienced by the individual;
- meaningful and functional, allowing identification and monitoring of rehabilitation priorities;
- person-centred, focussing on perceived functional difficulty and tailoring reassessment and intervention accordingly.

Box 24.1 Subjective assessment questions

Include:

- history of presenting condition;
- past medical and surgical history;
- current medications, including over the counter medicines, e.g. herbal remedies;
- social history;
- what they perceive as the main issue(s);
- what they know about their condition(s), i.e. do they have literature about the condition;
- previous experience of this issue and how they dealt with it; if new, what they perceive would be the best outcome;
- impact on functional ability and lifestyle alterations;
- expectations from physiotherapy.

Subjective assessment

Appropriate questioning allows the therapist to focus on the main presenting problem(s) (Box 24.1). Depending on their own experiences and those of people they know, a person may make out they can do better or are worse than you know them to be. For example, if a person is falling a lot at home and during a hospital admission are afraid a decision they cannot cope at home will be made, they may make out they *could* and *are* doing a lot for themselves at home. Work with colleagues (e.g. occupational therapists or nurses) to review if the information has been corroborated by friends and family. Alternatively, if someone has fallen and is afraid of returning home, they may seem reluctant to progress in therapy. Although challenging, analysis of such behaviour assists decision-making for a supportive/empathetic or firm tactic, or both.

Establishing agreed goals can be difficult where someone is unclear of their expectations from physiotherapy; insufficient understanding of their condition will limit expectations. A classic example on asking what the individual hopes to achieve by the end of their therapy is 'I want to walk better'. Your questioning should probe into how far they need to go indoors and out of doors, whether they need a mobility device or if they need assistance – only then can you agree realistic goals and set objective measures to gauge the outcome of your intervention.

Objective assessment

This confirms the subjective history and the referral diagnosis, and identifies issues that require referral for further investigation. Consider the assessment setting(s). For example, if an older person attends an 'osteoarthritis knee' class, they may improve in objective markers (joint range, strength) used by the physiotherapist but this does not indicate their coping ability at home; consider if they need a domiciliary physiotherapy referral to assess them at home.

Depending on the reason for referral, the objective assessment itself includes specific joint, nerve and muscle tests to assess the quality of these structures. It is especially important to assess the person's functional ability and responses to tests of general flexibility, strength, power and endurance. Balance, co-ordination and function are recognised as the physical manifestations that affect independence (Guralnik et al. 2001).

Many older people make remarkable, and sometimes unexpected, recoveries from severe mental or physical impairment. This process may be slower in the frail older population and in those with Parkinson's. With age comes multiple-pathology, the most common conditions of which are recorded in Table 24.6. An idea of the pathology of these conditions enables you to review what is 'normal', as well as what intervention you might realistically offer.

Table 24.6 Common conditions of older adults

Category	Examples
Orthopaedic conditions	Osteoporosis and osteomalacia, fractures – especially femoral and humeral necks, Colles' and vertebral fracture, Paget's disease, osteoarthritis and rheumatoid arthritis
Neurological conditions	Cerebrovascular disease, Parkinson's, neuropathies and other such neurological conditions
General medical conditions	Diabetes, falls, diverticulitis *and* irritable bowel syndrome, carcinomas, incontinence, urinary tract infections, hernia – especially hiatus, renal failure and thyroid disorders
Cardiorespiratory conditions	Ischaemic heart disease, congestive cardiac failure, pneumonias, chronic obstructive pulmonary diseases
Psychological conditions	Depression (whether a stand-alone condition or secondary to a disabling condition), dementia, acute confusion, anxiety/fear of falling

Drug history

Older people are often put on medication and left without review until they reach toxic levels. Those with Parkinson's

Recommended reading

Common ageing changes and thoughts as to how these might influence a physiotherapist's clinical decisions can be found in the AGILE booklet or on the *Physiopedia* site. *Physiopedia* is a resource aimed at international collaboration, contribution and sharing of knowledge and evidence for rehabilitation professionals throughout the world using wiki technology. Access it at: http://www.physio-pedia.com/index.php5?title=Main_Page and look for the 'Older People' tab.

Also look at the 'Physiotherapy' chapter of the *Professional's Guidance to Parkinson's* as it includes more detailed information about the impact of neuroanatomical changes in Parkinson's.

progress to a combination of several tablets (polypharmacy), increasing the risk of drug interactions, adverse reactions and non-compliance (Milton et al. 2008). Reactions can be vague and non-specific, such as confusion, constipation, hypotension and falls.

Medicines may be hard to swallow, but may be available in a different form if necessary, including via a percutaneous endoscopic gastrostomy (PEG) for people in the late stages of Parkinson's. Also, if a bottle is hard to open or if memory is a problem, different containers can aid dispensing and concordance with medication.

Non-pharmacological management is becoming increasingly advocated. For example, a sense of loneliness and low mood is natural following bereavement or diagnosis of a progressive condition such as Parkinson's, and a social network or counselling would help a person to cope rather than sedatives and anti-depression medication.

An issue specific to Parkinson's medication is the 'on-off' phenomenon which dramatically affects the quality of life for the individual. It refers to fluctuations in motor function with the ability to move more freely in an 'on' phase when drugs are working well, to reduced ability to function when drug action is reduced creating an 'off state'.

Clinical note

As Parkinson's progresses, a combination of several tablets is prescribed. In the older person, a dopamine-replacement drug, for example Madopar or Sinemet, is the main choice. Because of the increasing number of side effects and impact on the motor system over time with this category of drugs, i.e. dyskinesia or 'on/off' phenomenon, younger people diagnosed with Parkinson's are more likely to be prescribed a dopamine agonist as first-line therapy, for example Ropinerole or Rotigotine. There are also classes of drugs called mono-amine oxidase inhibitor type B (MAOI-B), for example Selegiline or Rasagiline, and catechol-o-methyltransferase (COMT) inhibitors, for example Entacapone or Tolcapone. While these work in different ways, they basically make levodopa last longer or reduce the amount required.

For more information about the drugs prescribed for Parkinson's visit http://www.parkinsons.org.uk/about_parkinsons/treating_parkinsons/drugs.aspx.

Pain

Pain is a common issue for older people, as well as those with Parkinson's. Serious complaints, however, are fewer than expected (Kumar and Allcock 2008) owing, in part, to the fact that some older people mistakenly think that pain is unavoidable so do not report it. In pathology that causes intense pain in younger people (e.g. angina or fractures), there may be so little discomfort or the pain may present in such a way that diagnosis is delayed – sometimes with fatal consequences. Pain is often incorrectly recognised and treated in people with dementia; in Parkinson's, it may be present differently according to whether it is as a result of injury or if it is manifested through a non-motor symptom, for example sensory symptoms of pain and paraesthesia (Mitra et al. 2008). In these cases, it is better to work pragmatically, looking at the person's functional ability and management of pain instead of looking at the restrictions it may cause.

Specific issues to assess in both the older population and in those with Parkinson's are the mobility-related problems which increase over time. High percentages of people over 50 years of age already self-report limitation in physical function related to difficulty with mobility and with basic activity of daily life (self-care) (DH 2004; Banks et al. 2010). Nearly half of people with Parkinson's want to do activities outside of their home, such as socialising, visiting relatives or pursuing hobbies, but cannot; common reasons why not were feeling too unwell, feeling too tired, lack of public toilets, problems getting around the streets, difficulties with transport and problems with access to buildings (PDS 2008).

Recommended reading

- The English Longitudinal Study of Ageing (ELSA) surveys people aged 50 and over about their lives in England. Publications can be accessed at: http://www.natcen.ac.uk/elsa/faq/about.htm.
- Assessment of mobility is important and a useful website on motor control illustrating brain connectivity is: http://thebrain.mcgill.ca/flash/d/d_06/d_06_cr/d_06_cr_mou/d_06_cr_mou.html#2.

It is mainly through the basal ganglia and thalamic connections that well-learnt, long and complex movement sequences are controlled. The component nuclei and the effect of the main neurotransmitter for the system, dopamine, enable the co-ordination of the following actions:

- pre-movement planning and preparation;
- initiation of movement;
- sequencing and timing of movement;
- maintaining cortically-selected movement amplitude, i.e. the frontal cortex is involved in the choice of movement, after which the basal ganglia takes over;
- habit-building;
- allowing the shifting of motor and cognitive sets (Morris et al. 2010).

Clinical note

For Parkinson's-specific assessments, see *Quick Reference Cards* (QRC) (UK) 1 and 2 (Ramaswamy et al. 2009). *QRC1: History Taking* and *QRC2: Physical Assessment* suggest a baseline of evidence-based themes of questioning and assessment.

Transfers

This refers to movement from one stationary point to another, such as rising from a chair or bed, getting in and out of a car, or up off the floor. With age, the lower spine becomes less flexible; the combination of limited joint range with slowed movement sequences affects momentum so, on standing, the person's centre of gravity is too far back in relation to their feet and they fall backwards (Morris 2000).

People with Parkinson's mention bed mobility and transfers as difficult tasks, especially turning to get comfortable, or getting into and out of bed. This occurs with a progressive decrease in spinal rotation from bradykinesia. Rigidity, the loss of automatic movement and a fear of rolling off the bed is worse if the person has dementia or is easily confused. As Parkinson's progresses, transfers involving a turn become more problematic. To turn 180 degrees, the feet must have good ground clearance; stability is needed through the legs to the trunk, plus continuity of movement and good posture assist in transition of weight in the upright posture. A fit, older person may be able to turn 180 degrees in two to three steps, while an older person without Parkinson's may do it in five steps or fewer (Simpson et al. 2002). Someone with progressing Parkinson's will take over four steps and will tend to favour turning in one direction over another; some people may freeze when simply thinking about turning (Stack et al. 2006). *Transfers and bed mobility* can be assessed as part of the Lindop Parkinson's Assessment Scale (LPAS) (Pearson et al. 2009), and in the Modified Parkinson's Assessment Scale (MPAS) (Keus et al. 2009).

Clinical note

Because of the high risk of falls, both older people and those with Parkinson's need to be assessed for their ability to rise from the floor (Keus et al. 2004).

Posture (including range of joint movement)

Posture becomes increasingly flexed – especially in people with Parkinson's (described earlier) who might develop an unstable posture that brings them up onto their forefoot, affecting all aspects of mobility in the later stages of the condition. In both populations, stiff trunk muscles reduce the range of movement, limiting trunk rotation and muscular extensor activity (Laughton et al. 2003). In Parkinson's, leg muscle strength, particularly of the knee and hip extensors has been proven to be weaker than healthy controls (Inkster et al. 2003). Trunk stiffness affects the counter-rotational ability of the thorax on the pelvis until they rotate in the same direction. Again, this is generally worse in people with Parkinson's unless the older person has a spinal condition, such as osteoporosis or ankylosing spondylitis. The onset of flexion is often insidious, leaving people unaware it is occurring until you take a photograph and show a 'normal' versus 'good' posture.

Clinical note

Use your assessment to distinguish whether the postural imbalance is permanent, through a fixed shortening, or whether subsequent intervention may be able to correct or improve it. Also, review the seating position, as people may sit for prolonged periods leaning to one side, or in a very slumped position.

Balance

Balance is of concern to both the older and the Parkinson's populations as it is a major contributor to falls. In Parkinson's, a person's ability to balance is typically affected in Hoehn and Yahr stage III and people will report increasing difficulty in dual or multi-tasking. This affects many aspects of daily life, such as walking and talking, or walking while carrying bags (Rochester 2004). The effect of muscle disuse and stiffness (rigidity in Parkinson's and bradykinesia), functional range of movement, strength and

Table 24.7 Common gait changes in older people and in people with Parkinson's

Changes	Comments
Slower pace	From decreases in hip amplitude and speed of movement. Average speed of 0.75 m/s can decrease to below 0.4 m/s – less if a person with Parkinson's freezes
Reduced step and stride length	Asymmetry of Parkinson's will affect one leg more than another or a condition such as stroke, arthritis or orthopaedic surgery
Increased double stance time	This phase is increased from 11% to 25% of the gait cycle
Increased flexion in posture	Affects hip extensors and calf muscle recruitment; worsens once a person begins using a mobility device
Reduced arm swing and body movement	Affects momentum and counterbalance action. More noticeable in people with Parkinson's. Also affected when a person begins using a mobility device
Decreased foot clearance	Toe clearance during the swing phase decreases as a result of reduced active hip flexion and calf work. Can result in tripping
Shuffle in later stages and festination	May be Parkinson's but may be higher level gait disorder from another vascular cause. A 'festinating gait' describes the short, quickening gait pattern, when the person with Parkinson's is flexed forward and on their forefoot, pulling the centre of gravity out of the base of support so they lose control of forward motion and often fall forward
Freezing or initiation problems	Assess whether initiation problems (where the person cannot set off), freezing (which occurs during the course of an activity, resulting in the person coming to a standstill mid-movement) or termination difficulties (where the person is unable to moderate their reactions to stop while walking) are experienced

posture make it very difficult for muscles to react as quickly as normal to rapid changes in body position. This provides a cause of postural instability which, in turn, impairs righting and equilibrium reactions, increasing the likelihood of falling (see later section).

Functional gait (including freezing, and indoor and outdoor mobility)

Walking is slowed as people become older and in those with Parkinson's. Some aspects, such as freezing during gait or festination are particular to Parkinson's, while other features, for example small steps, are common to all (Table 24.7).

There are a number of different ways of describing the Parkinson's gait. The stooped posture with hip and knee flexion, small shuffling steps, lack of trunk rotation, heel strike and reduced arm swing is well documented (Knuttson 1972; Murray et al. 1978), but, at times, could just as easily describe the gait of an older person with severe arthritis or post-stroke. Distinguish the components of movement that are causing the decreased efficiency of gait (Table 24.8).

Freezing of gait (FoG) is a form of akinesia (loss of movement) known as 'motor block', a 'sudden transient inability to move' (Jankovic 2008), whereby the feet appear glued to the floor. It is a major cause of falls and one of the most disabling symptoms of Parkinson's, with the incidence reported to be greater than 50% in people who have had Parkinson's for more than five years (Giladi 2001). It predominantly affects the limbs while walking, but has also been identified in the upper

Table 24.8 Gait disturbances in advanced Parkinson's

Parkinson's features	Gait disturbances
Hypo-/bradykinesia	Shorter steps, slower, less arm swing, festination
Rigidity (with abnormal posture)	Joint motion, flexed posture
Disturbed postural response	Fear of falling, hesitated gait, festination
Disturbed automatic motor tasks	Start hesitation, freezing of gait
Disturbed autonomic function	Weakness, light-headed unsteadiness
Involuntary movements	Dystonia or dyskinesias

limbs and during speech. There are five subtypes of freezing: hesitation at start, turn, in tight quarters, at destination and open space.

It is known that frequency and duration of FoG increases with disease severity and duration, but as it occurs at all stages of Parkinson's, it should be assessed accordingly. Identification of FoG can lead to the timely implementation of appropriate treatment and management with the aim to optimise function for those with freezing difficulty.

Gait initiation is a major cause of falls with significant differences in gait initiation between young and elderly populations, normal elders and those with Parkinson's. A research study investigating this comparison of normal elders with those with Parkinson's and gait initiation difficulties when utilising different cueing devices is currently underway (McCandless et al. 2011).

General mobility

Teaching mobility may sometimes mean testing safety with a wheelchair, including transfers.

Falls

A fall is defined as: 'An unexpected event in which the participant comes to rest on the ground, floor or lower level' (Lamb et al. 2005).

Key point

Falls and bone health are a major cause of disability in both populations, with a high risk of accidental home deaths resulting from injury. Many interventions provided through physiotherapy, or physiotherapy as part of a team, have been proven to modify the risk and help to prevent future falls.

At least 35% of those over the age of 65 years fall every year, with records as high as 50–60% in the Parkinson's population (Bloem 2001; DH 2009). The annual incidence of falls in individuals recorded with dementia was 40–60% in 1998 (Shaw and Kenny 1998).

The costs of a fall include health and social service costs, for example from treatment of injury or formal care during recovery. Perhaps the most important cost is the psychological toll it takes on the person, especially if they are prone to falling (DH 2009).

In frail older people and those with later stage Parkinson's, a fall can result in hip fracture, a requirement to accept long-term nursing care or even death.

The cause of falls is multi-factorial, for example ageing, pathology, inactivity and environmental factors, with over 200 risk factors identified (Lord et al. 2007), and, as such, the assessment process should also be multi-factorial (Box 24.2).

Box 24.2 Recommended areas for identification and assessment as the leading causes of falls

- Alteration to gait, balance and mobility, or muscle weakness.
- Older person's perception of functional ability and fear relating to falling.
- Visual impairment.
- Cognitive impairment.
- Alterations on a neurological examination.
- Urinary incontinence.
- Presence of home hazards.
- Cardiovascular pathology.
- Medications prescribed.

Cognitive impairment is an independent risk factor for falls. Nevertheless, older persons with cognitive impairment have been excluded from most of the successful falls-prevention randomised controlled trials in the community setting. Wraith and Criddle (2008) demonstrated both a reduction in falls in people with dementia in a carer-directed home exercise programme and also that these programmes maintained quality of life for people with dementia over 12 months.

Clinical note

Physiotherapists can assess falls factors using the DAME mnemonic:

D – drugs and alcohol

A – age-related physiological changes

M – medical (includes psychological, as well as physical factors)

E – environmental

Not all falls can be explained. Falls may be *intrinsic* in nature, for example postural hypotension, visual defects and a person's cognitive state, or *extrinsic* in nature, for example medication, footwear or the environment. Falls amongst people younger than 75 years are more likely to be a result of extrinsic factors than those aged 75 and over.

Posing a question correctly will determine the content and accuracy of response. For falls and near misses (an indicator of falls risk), the following wording is recommended: 'In the past month, have you had any fall, including a slip or trip in which you lost your balance and landed on the floor or ground or lower level?' (Lamb et al. 2005).

Functional ability may be assessed subjectively, for example question how a person manages personal and

domestic activities of daily living, and also observe how the person stands up from a chair; assess the person's ability to multi-task, for example walk and carry objects, walk and talk (as poor walking responses in a dual-task setting are a possible prognostic value for multiple falls) (Faulkener et al. 2007).

For people who have fallen, when assessing specific injuries that might have been sustained during a fall, consider AGILE's four identified aims (Goodwin and Briggs 2012):

1. To prevent falls;
2. To improve the older person's ability to withstand threats to their balance;
3. To prevent the consequences of a long lie;
4. To optimise confidence and reduce fear of falling.

In Parkinson's, falling when a person is walking is most common in a forward direction (Bloem et al. 2004) although people commonly fall backwards or take several attempts to generate sufficient forward momentum when getting up from a chair or the bed. A large proportion of falls also happen when a person with Parkinson's is turning, owing to a combination of effects, including slow response to the eye-head co-ordination, weight-transfer and freezing. Specific evidence relating to falls and physical capacity is still very limited and warrants further research (Rochester 2010). It is noteworthy that if mobility improves with balance, gait training and cueing, fall risk can increase owing to new found mobility and increased fall risks.

Recommended reading

Read the NICE Guideline on *Assessment and Prevention of Falls in Older People CG21* (2004) (http://guidance.nice.org.uk/CG21) and the Parkinson's UK information section *Falls and Parkinson's* (http://www.parkinsons.org.uk/advice/publications/motor_and_non-motor_symptoms/falls_and_parkinsons.aspx).

Mental health

A decline in mental health can hinder and limit a person's participation and progression with physiotherapy. Physiotherapy for mental health problems varies depending on the reason for the condition and the stage it is at; however, there are recommended good practice points for us to follow (Box 24.3). Some physiotherapists specialise in this clinical field post-qualification.

When engaging with people with dementia in mobility-related activities Oddy (2004) states that consideration should be given to providing frequent reminders; utilising clear and short instructions; using familiar words or cues; knowing a person's likes and dislikes; offering reassurance; checking medication for hypotension and medication side effects that may affect balance; and checking footwear is safe.

Dealing with someone who is anxious or confused will develop skills in empathy and in patience, as well as your basic listening and counselling skills. This may also be either with the individual or a stressed family member, giving insight and an understanding of their environment and behaviour.

Box 24.3 Good practice points when working with people with mental health problems

The physical treatment may involve:

- a thorough assessment of ability, with involvement from other MDT members;
- maintenance of range of movement (especially in the later stages when immobility increases) and work on promoting mobility;
- improvement of balance, to minimise the risks of falling, as mobility declines;
- treatment of any specific injuries sustained;
- provision of information to help reduce anxieties and fears, and assistance in carrying out a specific programme designed for that individual;
- teaching of positioning and manual handling in the later stages of some of the disorders.

INTERVENTION

Physiotherapy assessment and treatment focusses primarily on physical issues. In these two populations, in addition to reviewing overall function, do not ignore specific manual techniques, as the mechanical changes to posture and joints will require appropriate mobilisation and exercise.

The difference in approach is often during clinical reasoning where you have to piece together implications of multiple pathology and complexity of the social context in addition to physical presentation.

Goals

As with any individual, the goals agreed should be directed towards management and, if appropriate, condition improvement. Your assessment of pre-referral ability compared with current ability will help identify the person's rehabilitation potential. You will need to consider which presenting feature relates to de-skilling, deconditioning, pathology or ageing, and, therefore, which are reversible and manageable. All the while, consider the

realism of the person's expectations, their safety and the support they might get.

If in a hospital or community team setting, decision-making occurs with consultation of the individual, their family and health and social service teams. When planning discharges, or after a home visit, goals may have to be reviewed.

Clinical note

Good practice issues to consider when agreeing goals and planning for discharge are:

- that you understand the meaning of the illness to the person or relative so you can agree on appropriate interventions and goals. You can put it in context, i.e. how important is the illness and its treatment compared with other social/psychological/physical problems?;
- that you are aware of the individual's style of coping with illness/disability, e.g. avoidance, emotion-focussed, problem-solving;
- knowing their mood and cognitive status (depression, anxiety, confusion), as it will affect learning, participation and carry over;
- knowing if there are hidden implications to that person in terms of difficulty in performing exercises you have asked of them and their energy requirement, the time it takes, if there is pain or discomfort, if there are actual or perceived risks, and visibility of progress for motivation. Are there any financial implications? The person may feel that these far outweigh any benefits of exercise.

Measuring outcome

Outcome measurement is both a requirement of competent physiotherapy practice (CSP 2005) and a government requirement (DH 2010). It can be difficult to distil the physiotherapy-specific input in populations requiring team involvement as the outcome is dependent on input from the whole team. For example, the physiotherapist and occupational therapist will measure different aspects in the person's function and the nurse may measure overall change to life quality. What gets measured (usually pre-to-post intervention, episode or unit of care) depends on your definition of 'the outcome of interest', on who wants the data and for what purpose. For example, a service manager or commissioner will want a measure of the cost to deliver a service as opposed to a therapist measuring change following intervention.

If an assessment form is used as a measuring tool, for example the Elderly Mobility Scale (Smith 1994), still assess the specific impairments underlying the individual's needs and also choose the correct measuring tool to measure the outcome of intervention. There are many measures you can choose depending on factors such as pathology or function. Table 24.9 lists the most commonly used tools; full details and references for these can be looked up on any good database, including the CSP outcome measure website, or in a book specifically dealing with outcome measurement.

Although tools are usually chosen on their properties of validity, sensitivity and specificity to the person's need, not all situations have such measures.

Specific intervention for people with Parkinson's

Physiotherapy has now been proven of benefit to people with Parkinson's (Tomlinson et al. 2012). In the earlier stages of Parkinson's, physiotherapy may only incorporate education and advice to enable maintenance of fitness levels and to minimise progression. The physiotherapist may also be involved at this stage in prescribing specific exercises to the individual so they can regain movement, prevent falls, maximise respiratory function or reduce pain.

As the condition progresses, intervention moves from maintenance to promotion of independence by using a wider network of support, whether asking family to assist, or requesting formal social services intervention. Finally, in the palliative stages, input becomes necessary to relieve pain and pressure areas, treating symptoms as they occur (Keus et al. 2004; Ramaswamy and Jones 2005).

General intervention in the form of physical activity and exercise

Definition

Physical activity is considered to be 'any bodily movement produced by skeletal muscles that results in energy expenditure', while *exercise* is defined as: 'Leisure time physical activity which is planned and structured, and repetitive bodily movement undertaken to improve or maintain one or more components of physical fitness' (Caspersen et al. 1985).

Physical activity is an umbrella term for many activities, including exercise, sport, dancing, gardening and walking. Regular physical activity is essential for physical and mental well-being across all age groups and conditions. There are very few physical and mental conditions that regular exercise does not help to prevent, or reduce the risk of developing or improving symptoms.

Table 24.9 Commonly used outcome measures

Purpose	Common Parkinson's examples	Common older people examples
Global measures	Barthel scale; the functional independence measure (FIM) or the therapy outcome measures (TOMs) done by the multi-/ interdisciplinary team	
Stage or age specific measures	Hoehn and Yahr disease staging	Elderly Mobility Scale
Disease or condition specific scales	Unified Parkinson's Disease Rating Scale (old and new versions); Parkinson's Activity Scale (disease-specific)	Scales for Parkinson's disease, arthritis, stroke, pain, cognitive state, etc.
Function specific tools – may look at particular aspects of function, e.g. balance, turning, gait, etc.	Rivermead Index; timed get up and go; multiple tasks test – postural control Turning with: standing start 180 degree turn test; turning in bed; turning step count (on spot and during turning) Balance: Berg balance scale; functional reach (FR) Postural stability: retropulsion test Gait: 10-metre walk; freezing of gait (First) step length	Timed get up and go test; Tinetti evaluation (performance oriented assessment measure) of balance and gait Turning: 180 degree turn test Balance: Berg balance scale, FR Gait: 10-metre walk; 6-minute walk
Patient-specific measures or patient reported outcome measure (PROM)	Treatment evaluation by the Le Roux method (TELER); Goal attainment scoring (GAS) Utility of cueing devices for Parkinson's (McCandless et al. 2010)	TELER and GAS
Emotional status	Carer Strain Index	
Quality of life scales	PDQ-39, SF 36	SF 36, WHO – quality of life for older people
Self-assessment scales	EQ5D (perceived health state); Falls Efficacy Scale	

It is known that physical activity is reduced in the older population and in those with long-term conditions whose lives become more sedentary (Guralnik et al. 2001; Banks et al. 2010). Preferred exercise or sport should be continued as long as possible alongside education on the benefits of exercise. Older adults engaged in regular physical activity demonstrate improved balance, strength, coordination and motor control, flexibility and endurance.

Newer technology, for example the Nintendo-Wii, can improve well-being in older adults, at home and in residential care, and are useful for socialising and intergenerational fun.

Reduced physical activity levels can occur as a result of:

- falls and injury – if the person has Parkinson's, this might be from bradykinetic-rigid syndromes or freezing (Bloem et al. 2004);
- increasing rate of disease complications and disability (comorbidity) in addition to motor impairment alone, in more advanced disease stage or with age (strength and function);
- cardiopulmonary decline and the resultant secondary complications.

Key point

As physiotherapists, we can influence people of all ages to increase their physical activity levels and educate them to improve their general state of health and well-being.

As with the older population, the fields of cellular biology and animal-model research are increasing evidence of exercise benefits at central nervous system level showing increased learning, and neuroplastic increases in dendritic spine density and synapse number. The importance for people with Parkinson's is the additional possibility that this may serve as a neuroprotective function, minimising progression of the course of the condition (Fisher et al. 2008; Petzinger 2010); this emerging evidence is starting to influence clinical practice.

Physiotherapists should set a progressive exercise regime, ideally to be completed regularly in the home environment. The use of a structured exercise programme in picture booklet format for illustration and guidance can be particularly helpful (Pang et al. 2006).

For people with Parkinson's, components in the exercise programme should include:

- exercising – large amplitude movements (amplitude training) emphasising rotation and extension in sitting, and lying and standing for trunk and limbs;
- facial, speech and breathing exercises should also be included;
- targeted training for gait, transfers, balance, resistance and flexibility exercises;
- postural awareness (relaxation).

Exercise or targeted training should be undertaken at the peak dose of the medication cycle (Keus et al. 2004).

Important considerations *specific to the older adult population* include:

- that the intensity of aerobic activity takes into account the older adult's aerobic fitness;
- activities that maintain or increase flexibility;
- balance exercises for older adults at risk of falls;
- that older adults that have medical conditions or disabilities that may affect their capacity to be physically active should seek advice from a doctor.

Examples of exercise include treadmill training leading to an improvement in gait and balance, and mood and cognitive function. Treadmill training can improve gait parameters (Fisher et al. 2008), lower limb tasks and increase muscle size (hypertrophy).

There are functional and behavioural benefits in response to different forms of exercise and part of a physiotherapist's role is to identify any barriers and enablers to gait initiation, physical activity and exercise. Recommended exercise includes Tai Chi, boxing, Nintendo Wii Fit and tango dancing. For people with Parkinson's, Argentine tango dancing utilises music as the auditory cue and corresponding dance steps as the movement strategy which, if done for one hour, improves both gait and balance.

More detailed guidance for exercise testing (aerobic, endurance, strength, flexibility, neuromuscular and functional) and exercise programming (aerobic, endurance, strength, flexibility, functional) is given by the American College of Sports Medicine (ACSM 2009). It is important to note the variability of symptoms and response to exercise owing to autonomic dysfunction, such as altered heart rate, blood pressure and response to heat, alongside effects of muscular rigidity raising heart rate and oxygen consumption.

Recommended reading

Look at information (with video links) to the SPRING conference of 2009 about evidence for the positive effect of exercise on Parkinson's symptoms (*http://spring.parkinsons.org.uk/images/stories/ExerConf/ExConfBooklet.pdf*).

Physical activity and exercise for falls

Of all the above interventions, for physiotherapists, exercise (for strength and balance) has been shown to have the most effective outcomes in falls-management, with a reduction in falls rates, including high falls-risk inpatients on subacute wards (Sherrington et al. 2008). As the majority of people who attend such programmes relapse into old ways by six months, the physiotherapist should attempt to reinforce and encourage a change in behaviour, eliciting ideas of what might keep the person motivated both during the course of the programme and into the future, in addition to the physical perspective. This is becoming the focus of health promotion and multidisciplinary intervention has been proven to be of most effect for fallers (NICE 2004).

The research by Sherrington et al. (2008) demonstrates that clinicians have to ensure that:

- the balance training is highly challenging, individualised and progressive;
- exercise should be done at least twice a week and for a minimum duration of six months;
- walking should only be prescribed in addition to a high intensity/high dose programme.

Balance impairment and falls are a frequent disabling consequence of Parkinson's (Morris et al. 2010). Freezing and falls significantly impact their everyday lives, with an increased risk of comorbidities and admission to nursing homes. As freezing may be a predictor of falls, the inclusion of freezing in falls assessment is needed, for example the New Freezing of Gait Questionnaire (N-FOGQ).

Freezing of gait (FoG) is one of the key problems for the Postural Instability and Gait Disorder (PIGD) group. Cognitive and motor-loading during dual tasking, for example walking and talking, increases the odds of FoG episodes and falling.

Principles of physiotherapy practice and Parkinson's (Morris et al. 2010)

Evidence-based research and guidelines can be distilled into a wide range of contemporary physiotherapy-based treatments; however, they can be usefully categorised as follows:

- motor skill learning in skill acquisition;
- teaching people strategies to enable them to move more easily;
- treatment of secondary problems, such as cardiac de-conditioning and muscle weakness;
- educational and health promotion to reduce the risk of falls, increase physical activity levels and, thus, influence social participation.

The above treatment and management strategies are targeted at addressing every level of the ICF: impairment,

activity limitation and participation, and interventions must be tailored to ensure individual's needs are met.

It is useful, as part of the assessment, to categorise patients into early-, mid- and late-stage Parkinson's in order to tailor the treatment approach accordingly. Exercise, cueing and movement strategies are three key areas of intervention for people with Parkinson's. These can be combined for best effect.

- *Early-stage*: motor skill learning.
- *Mid-stage*: resistance training; compensatory.
- *Late-stage*: secondary sequalae; dance.

In addition to the 'prescribing' of exercise for people with Parkinson's, the other area of evidence for the effectiveness of physiotherapy is in maintaining and accessing automatic movement patterns through the choice of appropriate cues and cueing strategies (Lim et al. 2005; Rochester 2005; Nieuwboer et al 2007). Cueing can be effective in the short term within the home environment; however, longer-term effects are yet unknown (Nieuwboer et al. 2007). Similarly, the laser-cane (a walking stick with a laser light as a visual cue) was proved to be an effective cueing device at overcoming freezing during gait initiation compared with auditory cues and vibratory cues in the laboratory, but its effectiveness in the home and outdoors (short- or long-term) has yet to be evaluated (McCandless et al. 2010).

Table 24.10 Commonly used cues

Cues	Examples
Visual	Strips of card on the floor can assist step length and initiation problems Strategically placed cue cards with a keyword to activate movement Laser cane Laser-walker
Auditory	Metronomes, a musical beat or voice can help gait initiation and maintenance
Proprioceptive	Rocking side-to-side taking a step backwards can help overcome freezing; trial with functional electrical stimulation (FES)
Internal cueing self-instruction Cognitive	Memorising separate parts of the movement and rehearsing them mentally Taking a *big* step; walking fast; counting aloud while walking; visualisation of parts of a movement sequence; visualisation of a set step length and adjusting walking to step with that size

Table 24.10 provides some of the common types of cues recommended and the evidence base for these.

To help process some of the information from this chapter, we have provided a case study to highlight the goals, aims and examples of treatment plans based on guidance for physiotherapists working with Parkinson's. Read through the case study and guidelines while considering the following:

- ICF (impairment, activity, participation);
- stage of the condition (anatomical location of progressive pathology: lower brainstem, middle brainstem or cortical involvement = dementia);
- person-centred goals;
- principles of management at early-, mid- and late-stage;
- the role of physiotherapy – assessment, treatment, 'coach', advisor, exercise prescriber;
- relevant outcome measures.

Consider QRC 2 in particular and which key elements of assessment you would include if the person in the case study was not early diagnosis, but older and at mid- or late-stage. Finally, go one step further and consider how your assessment and input would differ if it were an older person with dementia (cortical involvement).

CASE STUDY: EARLY STAGE NEW DIAGNOSIS – HOEHN AND YAHR 1

Katherine is 40 years old, is married with two pre-teenage children and works full-time as a secretary for a large department store. She has just been diagnosed with Parkinson's. She is anxious her employer remains unaware and is keen to work as long as possible. She presents with mild right (dominant) hand and complains of backache after sitting at her desk. Her voice has become quieter making telephone conversations problematic on occasion. Katherine enjoys spending time with the children and drives them to athletics and after-school swimming club. She has noticed both her legs feeling stiffer than normal and has stopped attending ballroom dancing with her husband. Katherine occasionally loses her balance (trips and overbalances forward) while walking her golden retriever on uneven ground. She would like to try playing golf but lacks confidence.

GOAL: MANAGEMENT/TREATMENT (TABLE 24.11)

1. Increase aerobic capacity in and outside the work environment.
2. Retain current employment and independence as long as possible.
3. Take regular meaningful activity/exercise/sport individually and with the whole family at least once a week.

Table 24.11 Recommended treatment goals and strategies to manage Parkinson's

Stimulation of activities	Goal (for early phase) QRC 3	Strategy QRC 4
Transfers	Prevention of inactivity Motor skill acquisition	Warm-up and stretch prior to tango dancing with husband
Body posture	Increase postural awareness and reduce back pain at work	Postural exercises at work for endurance and flexibility in spinal extensors (neck and back); limit time in flexed sitting position. Ergonomic assessment at work Lumbar roll at work and while driving
Reaching and grasping	Maintain hand dexterity, reaching/grasping	Practise throwing/catching balls while walking the dog or playing Frisbee with the children
Balance	Identify balance problems (linked to fear of falling while dog-walking)	Training for 5 km school charity 'fun run' will increase strength in lower limbs (important for prevention of falls)
Gait	Improve gait speed using Nordic walking poles. Simple cardiac monitor	'Fast walking' (power walking)/Nordic walking with group at lunchtime twice a week
Prevention	75–80% target heart rate 5–8 mph Average energy expenditure 4.3 metabolic equivalents (METS) (Fisher et al. 2008)	Use of intense exercise to act as neuroprotective agent
Inactivity	Preserve or improve physical condition Prevent cardiac deconditioning See American College of Sports Medicine (ACSM) Guidelines 2009 For exercise prescription in Parkinson's disease Research local golf facilities and charitable organisations	Minimum 30 min exercise 5 times a week Train for 5 km fun run while children at athletics after school – aerobic exercise Use heart rate monitor, warm-up cool-down, include stretches for hamstrings and gastrocnemius Provide information on keeping active through sports swimming once a week with family, ballroom dancing (with husband for one hour a week) Find a 'golf buddy' to accompany Katherine, consider organising a golf group for people with Parkinson's once a week. Match medication timing to tee-off schedule
Falls	Prevent fear of falling while out dog-walking on hills/uneven ground	Walk the dog on an area where the dog can run freely (without a lead), thus reducing risk of being pulled forward/off-balance by the dog on the lead Strengthen lower limb muscles using a gym membership or home exercise programme

CONCLUSION

As mentioned at the start of the chapter, Parkinson's can be *very* different clinically to the management of older adults. As with any population, a physiotherapist learns to explore clinical problems through subjective and objective assessment, adhering to a patient-centred approach with 'shared decision making' (DH 2010) and implementation of best practice utilising evidence-based guidelines, such as those for Parkinson's (Keus et al. 2004).

In general terms, it is useful to consider aspects of both Parkinson's and of older adult conditions as being similar to an iceberg; the ice above the water comprises the physical alterations we are referred to assess in these populations, whereas the larger expanse of ice below the water comprises non-motor problems. These latter problems are often hidden or unrecognised, yet an understanding of the

Recommended reading

Carry out a literature search for authors such as Diana Jones, Meg Morris, Alice Nieuwboer, Lynn Rochester and Nir Giladi. They have written extensively on physiotherapy-relevant articles reviewing the mechanisms of gait in people with Parkinson's, mainly describing people with bradykinetic-rigid symptoms or those with freezing. Few articles research dyskinesia, where the gait pattern can be so erratic that assessment is difficult, and the treatment plan follows alleviation of current symptoms.

impact of such symptoms for the individual, relatives and carers is vital. They are a key to facilitating identification of person-centred goals as targets for treatment.

The guidance on assessment in the chapter should help you highlight the person's clinical need. The toolkit of interventions provided will enable the achievement of agreed goals, and the information on physical activity linked with accurate measurement of progress utilising appropriate outcomes will help you to promote a good holistic intervention.

We hope you will also use the recommended reading and websites to read more around the issues and to develop a broader understanding of these subjects.

ACKNOWLEDGEMENTS

A big thank you to all the therapists, patients and people we know who agreed to be photographed for this publication, and from whom we are still learning. Bhanu would like to thank all of her AGILE colleagues: Sal Foulkes, Vicki Goodwin, Diana Jones, Fiona Lindop, Penelope Reynolds, Amy Sheppard and Lynn Whyke for ideas, opinions, ears and patience during this undertaking. To members of the Parkinson's UK (Sheffield Branch) Classes and Committee for unfailing support, and finally to my family, especially my father, who set the bar for my high practice standards, and my physiotherapist husband Michael Heys for mobilising my back and neck when I could tear him away from the rugby league and football. Paula would like to thank UCLAN student physiotherapist Ryan Pope, a student research intern and CSP representative who presented a Parkinson's UK-funded project on the effectiveness of cueing devices in Montana 2010. Finally, thanks to UCLAN International physiotherapy student Shiva Mauree, a physiotherapy research intern who provided photos for this edition.

REFERENCES

ACSM (American College of Sports Medicine), 2009. ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities, third ed. Human Kinetics, Leeds.

AGILE, 2008. Core standards of physiotherapy practice. Service standards of physiotherapy practice. http://www.csp.org.uk/sites/files/csp/secure/agile_standards_2008_final_version.pdf; accessed August 2012.

Banks, J., Lessof, C., Nazroo, J., et al., 2010. The 2008 English Longitudinal Study of Ageing (Wave 4). Financial circumstances, health and well-being of the older population in England. The Institute for Fiscal Studies, London; http://www.ifs.org.uk/elsa/report10/elsa_w4-1.pdf; accessed August 2012.

Bloem, B., 2001. Prospective assessment of falls in Parkinson's disease. J Neurol 248, 950–958.

Bloem, B.R., Hausdorff, J.M., Visser, J.E., et al., 2004. Falls and freezing of gait in Parkinson's disease: a review of two interconnected, episodic phenomena. Movement Disord 19 (8), 871–884.

Braak, H., Del Tredici, K., Rüb, U., et al., 2003. Staging of brain pathology related to sporadic Parkinson's disease. Neurobiol Aging 24, 197–211.

Caspersen, C.J., Powell, K.E., Christensen, G.M., 1985. Physical activity, exercise, and physical fitness: definitions and distinctions for health-related research. Public Health Rep 100, 126–131.

Chaudhuri, K.R., Martinez-Martin, P., Brown, R.G., et al., 2007. The metric properties of a novel non-motor symptoms scale for Parkinson's disease: results from an international pilot study. Movement Disord 22 (13), 1901–1911.

CSP (Chartered Society of Physiotherapy), 2005. Core Standards of Physiotherapy Practice. CSP, London.

DH (Department of Health), 2001. National Service Framework for Older People. Her Majesty's Stationery Office, London.

DH (Department of Health), 2004. Health Surveys for England; http://www.dh.gov.uk/en/Publicationsandstatistics/PublishedSurvey/HealthSurveyForEngland/index.htm.

DH (Department of Health), 2009. Prevention Package for Older People Resources. Her Majesty's Stationery Office, London.

DH (Department of Health), 2010. Equity and Excellence; Liberating the NHS. Her Majesty's Stationery Office, London.

Dimond, B., 2009. Legal Aspects of Physiotherapy, second ed. Blackwell Publishing, Oxford.

Faulkener, K., Redfern, M., Cauley, J., 2007. Multi-tasking: Association between poorer performance and a history of recurrent falls: A brief report. J Am Geriatr Soc 55 (4), 570–576.

Fisher, B., Wu, A., Salem, G., 2008. The effect of exercise training in improving motor performance and cortico-motor excitability in people with early Parkinson's Disease. Arch Phys Med Rehabil 89, 1221–1229.

Giladi, N., 2001. Freezing of gait. Clinical overview. Adv Neurol 87, 191–197.

Goodwin, V., Briggs, L., 2012. Guidelines for the Physiotherapy management of older people at risk of falling. AGILE Falls Guidelines Working Group; http://www.csp.org.uk/sites/files/csp/secure/agile_falls_guidelines_update_2012.pdf, accessed October 2012.

Guralnik, J., Ferruci, L., Balfour, J., et al., 2001. Progressive versus catastrophic loss of the ability to walk. Implications for the prevention of mobility loss. J Am Geriatr Soc 49, 1463–1470.

Hoehn, M., Yahr, M., 1967. Parkinsonism: onset, progression and mortality. J Neurol 17 (5), 427–442.

Inkster, L., Eng, J., MacIntyre, D., et al., 2003. Leg muscle strength is reduced in Parkinson's disease and relates to the ability to rise from a chair. Movement Disord 18 (2), 157–162.

Izaks, G., Westendorp, R., 2003. Ill or just old? Towards a conceptual framework of the relation between ageing and disease. BMC Geriatrics 3, 7.

Jankovic, J., 2008. Parkinson's disease. Clinical features and diagnosis. J Neurol Neurosurg Psychiatr 79, 368–376.

Jones, D., Playfer, J., 2004. Parkinson's disease. In: Stokes, M. (Ed.), Physical Management in Neurological Rehabilitation. Elsevier, London, pp. 203–219.

Keus, S.H.J., Hendriks, H.J.M., Bloem, B.R., et al., 2004. Clinical practice guidelines for physical therapy for people with Parkinson's disease. Dutch J Physiother 114 (Suppl. 3), 1–94.

Keus, S., Nieuwboer, A., Bloem, B., et al., 2009. Clinimetric analyses of the Modified Parkinson Activity Scale. Parkinsonism Relat Disord 15 (4); 263–269.

Knuttson, E., 1972. An analysis of parkinsonian gait. Brain 95, 475–486.

Kumar, A., Allcock, N., 2008. Pain in Older People: Reflections and Experiences from an Older Person's Perspective. Help the Aged, London, http://www.britishpainsociety.org/book_pain_in_older_age_ID7826.pdf; accessed August 2012.

Lamb, S., Jørstad-Stein, E.C., Hauer, K., Becker, C., 2005. Development of a common outcome data set for fall injury prevention trials: The Prevention of Falls Network Europe consensus. J Am Geriatr Soc 53 (9), 1618–1622.

Laughton, C., Slavin, M., Katdare, K., et al., 2003. Aging, muscle activity, and balance control: physiologic changes associated with balance impairment. Gait Posture 18 (2), 101–108.

Lim, I., van Wegen, E., de Goede, C., et al., 2005. Effects of external rhythmical cueing on gait in patients with Parkinson's disease: A systematic review. Clin Rehabil 19 (7), 695–713.

Lord, S., Sherrington, C., Menz, H., et al., 2007. Falls in Older People – Risk Factors and Strategies for Prevention, second ed. Cambridge University Press, Cambridge.

MacMahon, D., Thomas, S., 1998. Practical approach to quality of life in Parkinson's disease: the nurse's role. J Neurol 245, S19–S22.

McCandless, P., Evans, B., Robinson, N., et al., 2010. The effectiveness of different cueing devices for people with Parkinson's disease and gait initiation difficulties. World Movement Disord 25 (3), S683.

McCandless, P., Evans, B., Churchill, A., et al., 2011. The effectiveness and acceptability of different cueing devices for people with Parkinson's Disease and gait initiation difficulties. Physiotherapy, 97(1): eS18–eS1415.

Milton, J., Hill-Smith, I., Jackson, S., 2008. Prescribing for older people. BMJ 33 (6), 606–609.

Mitra, T., Nauda, Y., Martinez, P., 2008. The non-declaration of non motor symptoms of Parkinson's disease to healthcare professionals. An international survey using the NMSQuest. 6th International Congress on Mental Dysfunctions and Other Non-motor Features in Parkinson's Disease and Related Disorders. Dresden: POII: 161.

Morris, M., 2000. Movement disorders in people with PD: a model for physical therapy. Phys Ther 80, 578–595.

Morris, M., Martin, C., Schenkman, M., 2010. Striding out with Parkinson disease: Evidence-based physical therapy for gait disorders. Phys Ther 90, 280–288.

Murray, P., Sepic, S.B., Gardner, G.M., et al., 1978. Walking patterns of men with Parkinson's. Am J Phys Med 57 (6), 278–294.

NICE (National Institute for Health and Clinical Excellence), 2004. CG21: Falls: The Assessment and Prevention of Falls in Older People. NICE, London.

NICE (National Institute for Health and Clinical Excellence), 2006. CG35. Parkinson's Disease: Diagnosis and Management in Primary and Secondary Care. NICE, London.

Nieuwboer, A., Kwakkel, G., Rochester, L., 2007. Cueing training in the home improves gait-related mobility in Parkinson's disease: The RESCUE-trial. J Neurol Neurosurg Psychiatry 78 (2), 134–140.

Oddy, R., 2004. Promoting success in mobility for residents with dementia. Nurs Resident Care 693, 124–127.

Pang, M.Y.C., Harris, J.E., Eng, J.J., 2006. A community-based upper-extremity group exercise program improves motor function and performance of functional activities in chronic stroke: a randomized controlled trial. Arch Phys Med Rehabil 87, 1–9.

Paterson, B., 2001. The shifting perspectives model of chronic illness. J Nurs Scholar 33 (1), 21–26.

PDS (Parkinson's Disease Society), 2007. Professional's Guide to Parkinson's. PDS, London.

PDS (Parkinson's Disease Society), 2008. Life with Parkinson's Today

– Room for Improvement. Results of the UK's Largest Ever Survey of People with Parkinson's and Carers. PDS, London.

Pearson, M., Lindop, F., Mockett, S., et al., 2009. Validity and inter-rater reliability of the Lindop Parkinson's Disease Mobility Assessment: A preliminary study. Physiotherapy 95, 127–134.

Petzinger, G., 2010. Exercise and Parkinson's disease: How to make it work for you. Movement Disord 25 (3), S599.

Ramaswamy, B., Jones, D., 2005. Making links between evidence and practice: The UK experience with physiotherapy for Parkinson's disease. Agility 2, 3–7.

Ramaswamy, B., Jones, D., Goodwin, V., et al., 2009. Quick Reference Cards (UK) and Guidance Notes for Physiotherapists Working with People with Parkinson's Disease. Parkinson's Disease Society, London.

Rochester, L., 2004. Attending to the task: Interference effects of functional tasks on walking in Parkinson's disease and the roles of cognition, depression, fatigue and balance. Arch Phys Med Rehabil 85 (10), 1578–1585.

Rochester, L., 2005. The effect of rhythmical cues on walking during a simple and dual functional motor task in a complex environment in people with Parkinson's disease. Arch Phys Med Rehabil 86, 999–1006.

Rochester, L., 2010. Physical therapy and Parkinson's disease: Evidence-based assessment of physical therapy. Movement Disord 25 (3), S600.

Shaw, F., Kenny, R.A., 1998. Can falls in patients with dementia be prevented? Age Ageing 27, 7–9.

Sherrington, C., Whitney, J., Lord, S., et al., 2008. Effective exercise for the prevention of falls: A systematic review. J Am Geriatr Soc 56 (12), 2234–2243.

SIGN (Scottish Intercollegiate Guidelines Network), 2010. Diagnosis and pharmacological management of Parkinson's disease. A national clinical guideline. Edinburgh; http://www.sign.ac.uk/pdf/sign113.pdf; accessed August 2012.

Simpson, J.M., Worsfold, C., Reilly, E., et al., 2002. A standard procedure for using TURN180: testing dynamic postural stability among elderly people. Physiotherapy 88 (6), 342–353.

Smith, R., 1994. Validation and reliability of the Elderly Mobility Scale. Physiotherapy 80 (11), 744–747.

Stack, E., Ashburn, A., Jupp, K., 2006. Turning strategies demonstrated by people with Parkinson's disease during an everyday activity. Parkinsonism Relat Disord 12, 87–92.

Stern, M., Lang, A., Poewe, W., 2012. Toward a redefinition of Parkinson's disease. Movement Disorders 27 (1), 54–60.

Tomlinson, C., Patel, S., Meek Herd, C., et al., 2012. Physiotherapy intervention in Parkinson's disease: Systematic review and meta-analysis. BMJ 2012, 345, e5004; http://www.bmj.com/content/345/bmj.e5004, accessed October 2012.

Van Rooden, S., Heiser, W., Kok, J., et al., 2010. The identification of Parkinson's disease subtypes using cluster analysis: A systematic review. Movement Disord 25 (8), 969–978.

Weintraub, D., Koester, J., Potenza, M., et al., 2010. Impulse control disorders in Parkinson Disease: A cross-sectional study of 3090 patients. Arch Neurol 67 (5), 589–595.

Whiddett, R., Hunter, I., Engelbrecht, J., et al., 2006. Patients' attitude towards sharing their health information. Int J Health Informatics 75 (7), 530–541.

WHO (World Health Organization), 2001. International Classification of Functioning, Disability and Health. World Health Organization, Geneva.

Wraith, M., Criddle, A., 2008. Reduction in falls found for people with dementia from carer-directed home exercise program. Alzheimers Dementia 4 (Suppl. 4), T448–449.

Yanagisawa, N., 2006. Natural history of Parkinson's disease: From dopamine to multiple system involvement. Parkinsonism Relate Disord 12, S40–S46.

Chapter 25

Neurodynamics

Monika Lohkamp, Lee Herrington and Katie Small

INTRODUCTION

In 1864, Lasegue first described the straight leg raise test (SLR) as an assessment for lower back problems with nerve involvement (Butler 1991). This concept was further developed by Goddard and Reid in 1965 who described the SLR as movement of the sciatic nerve. Also, in the 1960s Alf Breig investigated the biomechanics of the nervous system in more detail and showed that nerves move independently from other tissues. This formed the basis of the concept of 'neurodynamics'. Earlier terms for this concept were 'neural tension' (Breig 1978) or 'adverse mechanical tension of the nervous system' (Butler 1989). However, the actual term 'neurodynamics' was first introduced by Gifford in 1989. The concept of neurodynamics has been further developed by Grieve, Butler, Maitland and Shacklock over the last 30 years. Nowadays, although neurodynamics remains a relatively new concept within musculoskeletal clinical assessments, it is becoming more widely used and accepted as an important aspect for injury assessment and treatment.

Mechanics of the peripheral nervous system

In general terms, the nervous system can be divided into the central nervous system (CNS; brain and spinal cord), peripheral nervous system (all nerves after leaving the nerve roots within the spinal vertebrae) and autonomic nervous system (sympathetic and parasympathetic division) (Michael-Titus et al. 2007). The central and peripheral nervous system are closely connected through meninges (Butler 1991). The mechanical and physiological properties of peripheral nerves are crucial in their ability to function well.

The peripheral nerves are not only involved in conducting impulses and chemical traffic, but are also considered as dynamic tissue in the same way as muscles and joints. Along with the CNS, the peripheral nerves are designed to function while stretching and sliding (longitudinal and transverse motion) as a result of a wide variety and range of body movements (Shacklock 1995). When nerves are sliding they always slide towards the joint where movement takes place (Coppieters and Butler 2008). During movements of the body, nerves can undergo extremes of elongation, for example the spinal canal is 5–9 cm longer in flexion than extension, thus leading to an increased tensile load on the spinal cord (Breig 1978; Louis 1981 cited in Butler and Gifford 1989). Another example is the median nerve which has to adapt to a nerve bed nearly 20% longer if the shoulder is 90 degrees abducted, and the wrist and elbow are moved from flexion to extension (Millesi et al. 1986).

The nervous tissues are able to tolerate large tensile and compressive forces during movements associated with daily living and sporting activities. Nerves are strong structures owing to the surrounding connective tissue sheath helping them withstand large compressive and tensile forces (Shacklock 1995). If nerves are loaded by movement they react by moving and absorbing some of the pressure. During normal movement, as the nerve elongates the perineurium tightens, intraneural pressure increases and intrafasicular capillaries stop flowing. This occurs at 8% elongation, with intraneural microcirculation ceasing completely at 15% elongation (Ogata and Naito 1986). As the nervous system is dynamic, forces that cause a temporary disruption are easily tolerated. However, if the load becomes too excessive or persists, the maintenance of the increased local pressure to the nerve can cause local tissue ischaemia and injury.

Pathophysiology of the nervous system

Injuries involving nervous structures can either be caused by direct or indirect mechanics. A direct injury can occur from over-stretching or irritation from a repetitious

activity, whereas an indirect injury can result from bleeding owing to a muscle tear through which the nerve passes. Either of these types of injury can cause extraneural dysfunction (affecting movements outside of the nerve; relative to the nerve) or intraneural dysfunction (affecting movements within the nerve (Butler (1989)). These two forms of dysfunction are not exclusive of one another and sometimes extraneural dysfunction can actually lead to intraneural dysfunction. Both, however, will be discussed separately in further detail.

Extraneural dysfunction

The term 'extraneural' refers to anything outside of the nerve. This refers to the 'mechanical interface', which is any structure surrounding the nerves and lying between them and other surrounding structures (Butler and Gifford 1989). The nervous system is surrounded by, and passes through or around, various structures, including muscle, bone, tendon, blood vessels and intervertebral discs. These help contain the nervous system and allow for its movement, so as the nerve shortens, elongates or twists during daily movement so does the mechanical interface (Shacklock 1995). The smooth and normal movement of the nervous system and mechanical interface as it interacts with the musculoskeletal system is important for injury prevention. The consequence of extraneural pathology is to subject the nerve to increased tensile, frictional or compressive load. Examples of vertebral extraneural pathology are the narrowing of the intervertebral foramina (e.g. through osteophytes) or disc protrusions irritating a nerve root. In the peripheral nervous system, examples of extraneural pathology include the sciatic nerve lying in a bed of blood from a hamstring tear, tight fascial bands across nerves and a nerve as it passes through an oedematous tunnel (tarsal tunnel, carpal tunnel).

During movements, nerves take their lead from the movement of joints and muscles, needing to maintain a dynamic relationship with all non-neural tissue. For example, they must slide through tunnels of muscle and fascia, and be compressed against various structures, such as bone. Some interfacing structures make the nervous system more vulnerable to injury like, for example, tunnels (carpal tunnel, cubital tunnel, tarsal tunnel), hard interfaces (nerve on bone, passing through fascia), proximity to the surface or when nerves are fixed to the interfacing structures (e.g. common peroneal nerve at the fibular head). With abnormalities in these interfacing tissues, such as callus development, formation of bands of scar tissue, pressure from haematoma or enlarging tumour or ganglia, peripheral nerve neurodynamics and, therefore, neurophysiology may be adversely affected.

Mechanical interface dysfunctions occur when the forces exerted on the nervous system by the interfacing motion segment are abnormal or undesirable. The pressure exerted could be intermittent or sustained. These can be further divided into either 'opening' or 'closing' dysfunctions. This principle can be used throughout different neurodynamic and musculoskeletal assessments. These dysfunctions can be caused by, for example, incorrect posture, anatomical abnormalities or undesirable technique/movement.

Key point

A closing dysfunction means an alteration in the closing mechanism of the movement complex around the nervous system. You can have either too much closing (i.e. compression) or reduced closing.

Opening dysfunction involves an abnormality in the opening mechanism of the movement complex located adjacent to the nervous system. You can either have increased opening or reduced opening function (Shacklock 2005).

Intraneural dysfunction

Intraneural dysfunction refers to within the nerve itself. Entrapment of a nerve by surrounding tissues or with an intraneural fibrosis (scarring within the nerve structures) can cause pathological changes within the nerve (Butler and Gifford 1989). This may lead to a loss of range of movement or alteration in elasticity of the nerve and, as a result, enhance the normal mechano-sensitivity of the nerve during movement and thus provoke pain and reactive muscle spasm to prevent elongation.

Intraneural pathology can cause damage to the tissue of the nervous system itself, either to the conducting tissue in the form of demyelination, neuroma or hypoxia, or to the surrounding connective tissue, for example scarring of the epineurium. The possibility exists that a fibrosed nerve can cause a friction fibrosis elsewhere in the system. Tension has been taken up at one site in the nervous system thereby compromising the amount of available 'slack' elsewhere in the system. An example is a fibrotic length of common peroneal nerve at the head of fibular that may lead to abnormal deep peroneal nerve/interface relationships at the ankle. This may contribute to altered proprioception input from the ankle and in combination with less slack in the nervous system, influence the neurological functioning of the ankle joint, perhaps predisposing to recurrent ankle ligament sprains.

Mechano-sensitivity

The term mechano-sentitivity refers to how easily a nerve is activated following application of mechanical force (Shacklock 2005). There are a variety of causes for mechano-sensitivity; however, perhaps the most common factor is owing to pressure. This can either be applied directly or indirectly, as will be discussed.

Direct mechanical pressure can be applied to the nerve by any tissue infringing the space occupied by the nerve (e.g. 'bulging disc' or poorly fitted athletic knee brace). For example, compression of a disc hernia reduces blood flow in the nerve by 70% (Kobayashi et al. 2003). When loading persists, as it could from sustained un-physiological postures or maintained local pressures, damage may ensue as a result of local tissue ischaemia.

Indirect pressure is a more powerful cause of compressive load and therefore mechano-sensitivity, and is caused by immediate swelling in surrounding tissues that have been damaged. This inflammatory response also results in the release of chemical substances (histamines and enzymes) which create a chemical irritation, increasing pain, and can potentially lead to the laying down of scar tissue in and around the nerve with long-lasting negative effects on the mobility of the nerve.

Double and multiple crush syndrome

Double (DCS) or multiple crush syndrome (MCS) refers to a condition where proximal compression or elongation of a nerve decreases the nerve's ability to withstand compression or elongation at a distal site (Upton and McComas 1973). This type of condition is caused by changes to the nerve's axoplasmic flow. Axoplasmic flow is the term used to describe the transport mechanism of cell components in the nerve to their functional site where they help to maintain tissue health. Therefore, any pressure which is directly applied to the nerve or in the surrounding tissue following injury may cause slowing down of the axoplasmic flow and cause the nerve to 'become sick'. If pressure is applied to two (double crush) or multiple areas (multiple crush), the nerve will be considerably more prone to injury.

Axoplasmic flow relies on a mechanically unimpeded nervous system and an uninterrupted blood flow. Changes in the flow characteristics of axoplasm could cause insidious attrition to both target tissues and neurones, and impairment in the ultrastructure of the neurone. This enables previously resistible compressive or tensile forces to cause damage, usually at the vulnerable areas of the nervous system outlined previously. For example, patients with carpal tunnel syndrome are more likely to develop a compression lesion of the ulnar nerve at Guyon's canal and those suffering from carpal tunnel syndrome frequently have bilateral symptoms.

The original DCS described neural injuries at the neck which then predisposed to carpal tunnel syndrome (Upton and McComas 1973). Alternatively, reversed DCS occurs where a distal neural injury (e.g. at the wrist) predisposes to a proximal injury, perhaps at the shoulder. MCS occurs where following initial neural injury a patient experiences multiple and related areas of symptoms. Clinical presentation of DCS or MCS may often be widespread and unfamiliar in nature. However, patients may complain of a string of injuries or multiple areas of localised pain which have common pathophysiologies that represent a minor form of DCS or MCS.

The concept of DCS or MCS therefore provides an excellent reason for clinicians to evaluate along the nerve tract when suspicious of a neurogenic lesion. This should always be considered a possibility whenever an athlete reports multiple areas of pain, for instance a distance runner who complains of tibialis anterior and buttock pain. If diagnosis is later confirmed as DCS or MCS, treatment should also be considered for all the sites involved. For the example of the distance runner, treatment of the piriformis muscle may be needed to alleviate neural symptoms at the foot by altering a site of pressure contributing to altered axoplasmic flow to the foot.

ASSESSMENT PRINCIPLES

Indications

The main purpose of neurodynamic testing is to load the tested neural structures to help gain an understanding of their mechanical function in relation to load and their state of sensitivity to that load. There is no set *indication* of when neurodynamic testing should be performed; however, some believe that it should be performed if the client presents with any form of neurological symptoms, such as pins and needles, tingling, shooting or burning pain, unexplained strength loss, paraesthesia or numbness. Alternatively, neurodynamic testing may be indicated for any overuse injury or reinjury, or if the client has sustained an injury to the vertebral region. However, in theory, neurodynamic testing could be considered for any musculoskeletal injury as nerves are closely connected to other structures (e.g. muscles, tendons and ligaments).

As well as indications, there are also contraindications and precautions to neurodynamic testing. *Contraindications* represent any kind of factor or condition that increases the risk or danger to cause serious harm to the client by carrying out neurodynamic testing. Therefore, if any contraindications are present the neurodynamic tests should *not* be carried out. *Precautions* are factors that you should be aware of and take additional care of when carrying out and interpreting neurodynamic tests, although this does not mean that you cannot carry out the tests. Common contraindications and precautions to neurodynamic testing are presented in Table 25.1 (Butler 1989).

Structural differentiation

Neurodynamic tests involve assessing the mobility of the nervous system as it interacts with the musculoskeletal

Table 25.1 Contraindications and precautions to neurodynamic testing

Contraindications	Precautions	
Recent onset/worsening of neurological signs	Spinal stenosis or spondolythesis	Dizziness (vertebrobasilar insufficiency)
Cauda Equina lesions	Disc protrusion	Severe, unremitting pain
Cancer of nervous system/vertebral column	Presence of neurological signs	Frank cord injury
Acute inflammatory infection	Worsening disorder	Circulatory disturbances
Osteoporosis resulting in abnormal interfacing tissues	Irritability of nervous system	General health problems (diabetes, AIDS, multiple sclerosis)
Injury to spinal cord (tethered cord)		

system. However, this can create a problem when trying to distinguish whether reproductions of the client's pain/symptoms during a test is as a result of the movement by nervous or musculoskeletal structures. Structural differentiation emphasises movement of the neural tissues as opposed to the musculoskeletal tissues, therefore helping to identify the damaged structure – whether neural or musculoskeletal (Butler 2000). For example, when performing the slump test by extending the knee it would cause movement, and potential tension, of both nervous (sciatic nerve) and musculoskeletal (hamstrings) tissues. To help distinguish which structure was the cause of the client's pain/symptoms, we could get the client to release the cervical flexion. This structurally differentiating manoeuvre is unlikely to alter the strain on the lower limb musculoskeletal structures (hamstrings); however, it will change the tension on the nervous system. Therefore, if this manoeuvre resulted in reducing the client's pain/symptoms, we could propose that their injury was related to neurological, rather than musculoskeletal, damage.

Sensitising manoeuvres

While structural differentiation is a key element for helping identify neurogenic problems, for patients with a high level of function less severe problems may remain hidden. Therefore, in order to make the test more specific to identify the problem we can add sensitising manoeuvres. This is where the normal neurodynamic technique is modified slightly to increase the test sensitivity so that it becomes more specific to the patient's problem (Keneally et al. 1988). The easiest and most common way of sensitising tests is to put extra strain, or stretch, on the nerve by elongating it. A simple example of this would be to add cervical flexion when performing a SLR. Another sensitising manoeuvre would be to contract a muscle close to the nerve pathway around the affected area during the neurodynamic test, such as contracting piriformis muscle during SLR.

Key point

Once an individual joint position is achieved make sure this is maintained while other joints are moved during the test.

Gain constant verbal and visual feedback from the patient during testing.

Regularly re-test and monitor patient's symptoms during assessment and treatment and adapt as appropriate.

Clinical note

General rules when carrying out neurodynamic assessment:

- explain to the patient what you are doing and what they may feel during the test
- always carry out the test slowly
- do not only listen to the verbal feedback from the patient but also feel the resistance during the assessment
- do not hold the end position longer than you need to. Once the end position is reached, release it and ask the patient what they felt. In the worst case the patient may not remember and you may have to repeat the test
- always compare sides, testing the injured/affected side last
- always ask about the nature and location of the test responses

However, these manoeuvres cause a greater risk of pain to the patient. Therefore, sensitising manoeuvres are only recommended when previous standard neurodynamic tests have not revealed enough information to make a clear diagnosis and/or it is unlikely to cause severe pain/reproduction of symptoms, and the patient has no persistent or worsening neurological problems.

It is vital during neurodynamic assessment that the tests are performed correctly by the clinician as ill-performed tests can result in different responses by the client, therefore impairing accurate diagnosis of the injury. When correctly performing neurodynamic tests, it is important to carefully observe how the patient responds at all times, the available range of movement and the resistance felt by the clinician. The following points are to be considered during the neurodynamic test:

- the quality of the movement (e.g. is the movement smooth and not jerky);
- the range of movement (similarity bi-laterally);
- resistance through range and at end-of-range (here, the clinician may feel tightness that stops or impairs the movement);
- pain and symptoms through range (this may or may not be specific to reproducing their pain/symptoms).

Clinical note

Unlike other musculoskeletal tests the neurodynamic tests do not always have a clear positive or negative, and findings must be reflected upon in light of the bigger patient picture.

INTERPRETATION OF FINDINGS

Early clinicians were taught to determine whether a test was 'positive' or 'negative'. However, when performing neurodynamic tests it is highly likely that there is no clear-cut positive or negative answer. The findings from the neurodynamic examination must be clinically reasoned using all the information gained throughout the complete clinical assessment to make an informed decision of how next to proceed with treatment relevant to the individual patient's problem. With this in mind, the patient's response to the neurodynamic testing can be divided into four general categories, which are listed below.

- *Optimal* – this is where the neural system behaves exactly the way it should, allows full range of movement and does not produce any symptoms or discomfort to the patient, even when high loads and stress are applied.
- *Suboptimal* – the neural system here behaves less well, allows slightly reduced range of movement and may result in producing some symptoms in the patient, although not necessarily until a high enough load is applied to provoke the system.
- *Normal* – here, the patient is likely to have some symptoms during the testing; however, the function of the neural system (i.e. range of movement) is considered to be within 'normal' values (although these are not clearly established) or similar on both sides.
- *Abnormal* – this is where the function of the neural system is obviously outside the normal range and the patient may have significant symptoms. This can be caused by pathology or abnormalities in the mechanics and physiology of the nervous tissues. Therefore, as abnormalities do not always produce symptoms, the 'abnormal' response can be further divided into:
  - *relevant* – this would be a direct link whereby the test has clearly caused the patient's problem;
  - *irrelevant* – no causal link to the patient's problem.

Neurodynamic test responses can be summarised in an algorithm as shown in Figure 25.1.

Normal neurogenic response

A normal neurogenic response is one where there is a positive response to a structural differentiating manoeuvre that is a change in range of motion or symptoms. This change is very similar on both sides and does not provoke symptoms.

Abnormal neurogenic response

An abnormal neurogenic response is one where there is a positive response to a structural differentiating manoeuvre that is a change in range of motion. This change is very different between both sides and the test may or may not provoke symptoms. If the test provokes symptoms it is regarded as a *relevant abnormal neurogenic response*, i.e. there is a structurally differentiated positive response which is asymmetrical and provokes symptoms. The alternate to this is an irrelevant abnormal neurogenic response. Here, there is a structurally differentiated positive response which is asymmetrical but does not provoke the patient's symptoms. There may, however, be a causal link between this finding and the patient's symptoms.

The test is *positive* when the patient gets an abnormal neurogenic response. This is indicated by a change in range of motion with structural differentiation as compared with the non-affected side. Alongside the change in range of movement with structural differentiation, the test can also demonstrate symptom reproduction which is altered by structural differentiation (relevant abnormal

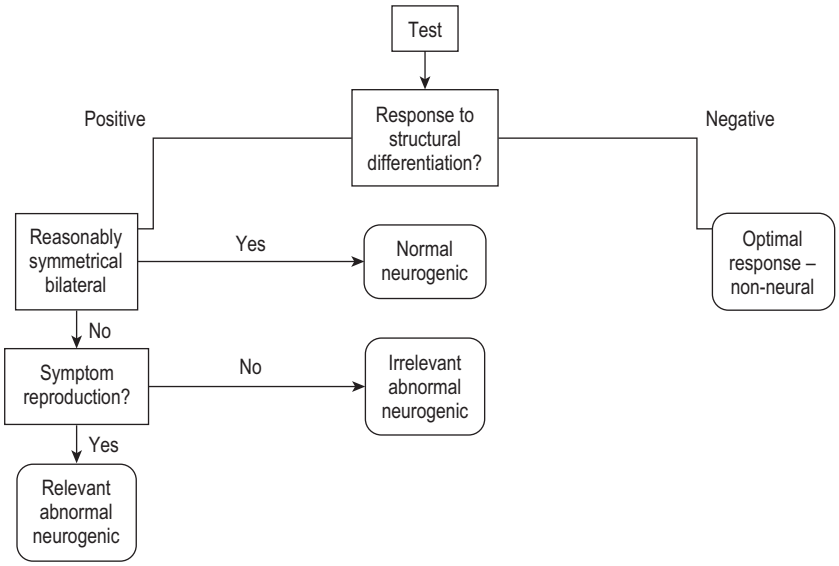


Figure 25.1 Algorithm to interpret neurodynamic response to testing.

neurogenic response) or might not demonstrate symptom reproduction (irrelevant abnormal neurogenic response).

If you find an abnormal neurogenic response during the test you only know that there is a neurodynamic dysfunction (increased mechano-sensitivity) somewhere along the nerve but you are not yet able to tell exactly what the cause for the dysfunction is and where exactly it is. The subjective history may give you some indications as to where the side of pathology may be.

What is causing increased mechano-sensitivity? It can be decreased neural tissue mobility, inflammation of the innervated tissue or mechanical interface dysfunction. You can always assume there is a problem with neural tissue mobility if there is mechano-sensitivity. We need to find out if the mechanical interface contributes to this problem and/or the degree of inflammation within the innervated tissue. Tissue inflammation can be identified by the typical signs of inflammation (hot, red, swollen) and the loss of function related to recent injury. To identify the side of possible mechanical interface dysfunction further assessment is needed but it is not within the scope of this chapter to discuss this in detail.

Clinical note

- *Sequencing* – depending on the injury you might want to change the sequence of movements during the tests.

GENERAL TESTS

In the following sections we describe the indications, techniques and structural differentiation for the main neurodynamic tests.

Passive neck flexion

Indications

- Headaches.
- Pain in the upper limbs.
- Pain in the neck and shoulder area.

Technique

Start with the patient lying supine. Stand behind the patient and place one hand under the neck and the other hand on the forehead. Alternatively, have both hands under the head (Figure 25.2). Move the head in flexion.

Structural differentiation

For structural differentiation a second therapist is needed for assistance. Once you have positioned the head in maximum neck flexion, the second therapist carries out a SLR as described later.

Slump test

Indications

- Pain and altered sensation related to nerve roots L4–S3.
- Muscle dysfunction (weakness or overactivity) in myotomes supplied by these nerve roots (foot dorsiflexion, extension hallux, eversion of the foot, contraction buttock, knee flexion, toe standing).
- Pain in the area of the sensory supply of the sciatic, tibial or peroneal nerve.
- Muscle dysfunction (weakness/overactivity) of the muscles supplied by the sciatic nerve (semitendinosus, semimembranosus, biceps femoris, hamstring part of adductor magnus), tibial nerve (gastrocnemius, soleus, plantaris, popliteus, tibialis posterior, flexor digitorum longus, flexor hallucis longus) or peroneal nerve (peroneus longus and brevis, extensor digitorum longus, tibialis anterior, extensor hallucis longus, peoneus tertius, extensor digitorum brevis).
- Symptoms related to sitting, driving, sprinting, hurdling, kicking and bending.
- Pathologies such as lumbar spine pathologies, sacroiliac joint pathologies, hamstring and calf muscle strains, Achilles, peroneal and tibial tendon injuries, ankle ligament sprains and plantar fasciitis.

Technique

Start the test with the patient sitting on the edge of a plinth with their spine in a neutral position, their sacrum vertical and their hands behind their back. Then, get the patient to slump forward so that their thoracic and lumbar spine is flexed but still with their head looking up and the sacrum vertical (neutral). Next, ask the patient to flex their neck so that they bring their chin to their chest. Place your hand over the top of their head and your elbow on the thoracic spine to maintain this position (Figure 25.3). Now, ask the patient to actively extend their knee until the end of range (Figure 25.4), and then dorsiflex their ankle also to end-of-range or to when they indicate a reproduction of pain/symptoms (Figure 25.5). Finally, remove your hand from the patient's head and ask them to look up (Figure 25.6). This is your structural differentiation manoeuvre. If neural tissue is involved the symptoms are changing (either reducing or increasing).

Sensitising manoeuvre

You can sensitise the test by getting the patient to put their foot into plantar flexion and inversion instead of dorsiflexion (sensitises for the common peroneal nerve).

Figure 25.2 Hand positioning during passive neck flexion.

Figure 25.3 Neck and thoracic flexion during slump.

Figure 25.4 Knee extension during slump.

Figure 25.5 Ankle dorsiflexion during slump.

Figure 25.6 Structural differentiation for slump.

LOWER LIMB NEURODYNAMIC TESTS (LLNTs)

Straight leg raise test (SLR)

Indications

- Pain and altered sensation related to nerve roots L4–S3.
- Muscle dysfunction (weakness or overactivity) in myotomes supplied by these nerve roots (foot dorsiflexion, extension hallux, eversion of the foot, contraction buttock, knee flexion, toe standing).
- Pain in the area of the sensory supply of the sciatic, tibial or peroneal nerve.
- Muscle dysfunction (weakness/overactivity) of the muscles supplied by the sciatic nerve (semi-tendinosus, semi-membranosus, biceps femoris, hamstring part of adductor magnus), tibial nerve (gastrocnemius, soleus, plantaris, popliteus, tibialis posterior, flexor digitorum longus, flexor hallucis longus) or peroneal nerve (peroneus longus and brevis, extensor digitorum longus, tibialis anterior, extensor hallucis longus, peoneus tertius, extensor digitorum brevis).
- Symptoms related to sitting, driving, sprinting, hurdling, kicking and bending.
- Pathologies such as lumbar spine pathologies, sacroiliac joint pathologies, hamstring and calf muscle strains, Achilles, peroneal and tibial tendon injuries, ankle ligament sprains and plantar fasciitis.

Generally, the slump test is used because it puts greater mechanical load on neural tissues and so allows for greater sensitivity. SLR might be chosen over the slump test if tissue is perceived to be highly irritable and sensitive to load.

Technique

Start the test with the patient lying supine. Stand to the side of the patient you are about to perform the test on, facing towards their head. Place one hand just above their knee and the other under their Achilles tendon with your palm over their foot (Figure 25.7). Perform the test by lifting their leg, bringing their hip into flexion while maintaining the extended knee until the end-of-range or until the patient indicates a reproduction of pain/symptoms (Figure 25.8).

Structural differentiation

Add structural differentiation by getting the patient to side flex their trunk to the contralateral side (Figure 25.9).

Sensitising manoeuvre

You can sensitise the test by adding plantar flexion and inversion of the foot (sensitises for the common peroneal nerve) or adding ankle dorsiflexion (sensitises for the tibial nerve).

Modifications of SLR: peroneal, sural and tibial nerve test

Indications

- Pain and altered sensation related to nerve roots L4–S3.

Figure 25.7 Hand positioning during straight leg raise.

Figure 25.9 Structural differentiation for straight leg raise.

Figure 25.8 Straight leg raise.

Figure 25.10 Foot position peroneal neurodynamic test.

- Muscle dysfunction (weakness or overactivity) in myotomes supplied by these nerve roots (foot dorsiflexion, extension hallux, eversion of the foot, contraction buttock, knee flexion, toe standing).
- Pain in the area of the sensory supply of the tibial, sural or peroneal nerve.
- Muscle dysfunction (weakness/overactivity) of the muscles supplied by the tibial nerve (gastrocnemius, soleus, plantaris, popliteus, tibialis posterior, flexor digitorum longus, flexor hallucis longus) or peroneal nerve (peroneus longus and brevis, extensor digitorum longus, tibialis anterior, extensor hallucis longus, peoneus tertius, extensor digitorum brevis).
- Symptoms related to sitting, driving, sprinting, hurdling, kicking and bending.
- Pathologies such as lumbar spine pathologies, sacroiliac joint pathologies, hamstring and calf muscle strains, Achilles, peroneal and tibial tendon injuries, lateral ankle ligament sprains and plantar fasciitis.

Technique

Peroneal

Start the test with the patient lying supine. Stand to the side of the patient you are about to perform the test on, facing towards their feet and place one hand just above their knee. Reach with the other hand under their foot, placing your fingers on their toes to bring the foot into plantar flexion and inversion (Figure 25.10). Perform the test by lifting their leg, bringing their hip into flexion while maintaining the extended knee and plantar flexed/inverted foot until the end-of-range or the patient indicates a reproduction of pain/symptoms.

Sural

Start the test with the patient lying supine. Stand to the side of the patient you are about to perform the test on, facing towards their feet and place one hand just above their knee. Place the other hand around their foot and bring the foot into dorsiflexion, inversion position.

Perform the test by lifting their leg, bringing their hip into flexion while maintaining the extended knee and dorsiflexed/inverted foot until the end-of-range or the patient indicates a reproduction of pain/symptoms.

Tibial

Start the test with the patient lying supine. Stand to the side of the patient you are about to perform the test on, facing towards their feet and place one hand just above their knee. Place the other hand around their foot and bring the foot into dorsiflexion, eversion position. Perform the test by lifting their leg, bringing their hip into flexion while maintaining the extended knee and dorsiflexed/everted foot until the end-of-range or the patient indicates a reproduction of pain/symptoms.

Structural differentiation for peroneal, sural and tibial neurodynamic test

Add structural differentiation by getting the patient to side flex their trunk to the contralateral side.

Prone knee bend

Indications

- Pain and altered sensation related to nerve roots L2–L4.
- Muscle dysfunction (weakness or overactivity) in myotomes supplied by these nerve roots (hip flexion, knee extension, foot dorsiflexion).
- Pain in the area of the sensory supply of the femoral nerve.
- Muscle dysfunction (weakness/overactivity) of the muscles supplied by the femoral nerve (iliacus, sartorius, quadriceps femoris).
- Symptoms related to iliac crest area pain, groin pain, hip pain (anterior), thigh pain and knee pain.
- Pathologies such as higher lumbar spine pathologies, hip pathologies, groin and quadriceps muscle strains and anterior knee pain.

Technique

Start the test with the patient lying on their front (referred to as 'prone'). Place one hand under their thigh, just above the knee or on the sacrum, and the other hand around their ankle (Figure 25.11). Then, bring their knee into flexion until end-of-range or the patient indicates a reproduction of pain/symptoms (Figure 25.12).

Structural differentiation

Add structural differentiation by getting them to side flex their trunk to the contralateral side (Figures 25.13 and 25.14).

Figure 25.11 Starting position prone knee bend.

Figure 25.12 End position prone knee bend.

UPPER LIMB NEURODYNAMIC TESTS (ULNTs)

Median neurodynamic test 1 (MNT1 or ULNT1)

Indications

- Pain and altered sensation related to nerve roots C5–T1.
- Muscle dysfunction (weakness or overactivity) in myotomes supplied by these nerve roots (shoulder abduction, elbow flexion, elbow extension, finger flexion, finger abduction and adduction, thumb extension).

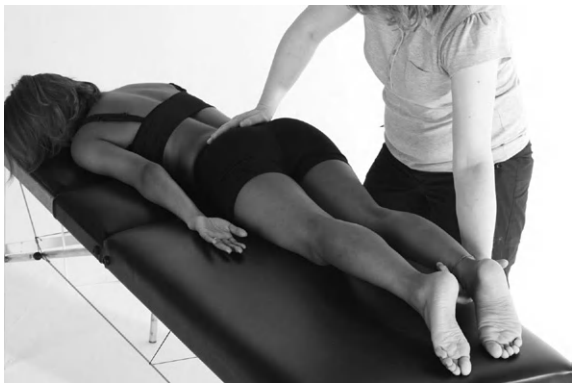


Figure 25.13 Starting position prone knee bend with structural differentiation.

Figure 25.15 Starting position median neurodynamic test 1.

Figure 25.14 End position prone knee bend with structural differentiation.

Figure 25.16 Shoulder abduction during median neurodynamic test 1.

- Pain or altered sensation in the area of the sensory supply of the median nerve.
- Muscle dysfunction (weakness/overactivity) of the muscles supplied by the median nerve (pronator teres, flexor carpi radialis, palmaris longus, flexor digitorum superficialis, abductor pollicis brevis, flexor pollicis brevis, opponens pollicis, flexor pollicis longus).
- Symptoms related to throwing or reaching.
- Pathologies such as neck pathologies, whiplash, shoulder dislocations/subluxations, golfer's elbow (medial epicondylitis), pronator tunnel related pathology and carpal tunnel syndrome.

Technique

Facing the supine lying patient, place your elbow superior to their shoulder and gripping the lateral side of their elbow with the hand of the same arm. With the other hand take hold of the patient's wrist and hand to place the wrist and fingers in full extension. Using your elbow, which is placed over their shoulder, depress their scapula to a neutral position by pulling it downwards towards their feet (caudad direction) (Figure 25.15). Now, take their shoulder into 90 degrees abduction and bring their forearm into supination (Figure 25.16). Next, place the shoulder in lateral rotation (Figure 25.17) and complete the test by extending their elbow until the end of range or they indicate a reproduction of pain/symptoms (Figure 25.18).

Structural differentiation

There are two different ways of carrying out structural differentiation to MNT1. The first technique requires you to carry out MNT1 as described above. When the end position in terms of elbow extension is reached, ask the patient to side flex the neck to the contralateral side (Figure 25.19). If neural tissue is involved, you may expect an increase or a decrease in the intensity of the symptoms

Figure 25.17 Shoulder lateral rotation during median neurodynamic test 1.

Figure 25.18 End position median neurodynamic test 1.

Figure 25.19 Structural differentiation for median neurodynamic test 1.

and more resistance. If there is no neural tissue involvement, the symptoms will not change.

The other way is to place the head in contralateral lateral flexion prior to carrying out MNT1 as described above. Once the end position is reached ask the patient to bring the head back into a neutral position. If there is neural tissue involvement the intensity of the symptoms and resistance may reduce or increase and you may be able to move the elbow a bit more in extension. If there is no neural tissue involvement you will not find any change.

Median neurodynamic test 2 (MNT2 or ULNT2A)

Indications

- Pain and altered sensation related to nerve roots C5–T1.
- Muscle dysfunction (weakness or overactivity) in myotomes supplied by these nerve roots (shoulder abduction, elbow flexion, elbow extension, finger flexion, finger abduction and adduction, thumb extension).
- Pain in the area of the sensory supply of the median nerve.
- Muscle dysfunction (weakness/overactivity) of the muscles supplied by the median nerve (pronator teres, flexor carpi radialis, palmaris longus, flexor digitorum superficialis, abductor pollicis brevis, flexor pollicis brevis, opponens pollicis, flexor pollicis longus).
- Symptoms related to underarm throwing, reaching behind, falling onto outstretched hand and pulling objects (dog-walking).
- Pathologies such as neck pathologies, shoulder dislocations/subluxations, golfer's elbow (medial epicondylitis), pronator tunnel related pathology and carpal tunnel syndrome.

Technique

With the patient lying supine and slightly obliquely on the bed, stand at the top corner of the bed, depress their scapula by leaning your hip against their shoulder, adding slight pressure. Place one hand under their elbow (flexed to 90 degrees) and take the arm into slight abduction. With the other, hold their hand so that you take their forearm into supination while extending their wrist and fingers (Figure 25.20). Next, fully extend their elbow and laterally rotate their shoulder (Figure 25.21). Finally, abduct and extend their shoulder until the end of range (Figure 25.22).

Structural differentiation

Structural differentiation can be carried out in the same way as for MNT1.

Figure 25.20 Starting position median neurodynamic test 2.

Figure 25.21 Mid-position median neurodynamic test 2.

Figure 25.22 End position median neurodynamic test 2.

Ulnar neurodynamic test (UNT or ULNT3)

Indications

- Pain and altered sensation related to nerve roots C8–T1.
- Muscle dysfunction (weakness or overactivity) in myotomes supplied by these nerve roots (finger flexion, thumb extension, finger abduction and adduction).
- Pain in the area of the sensory supply of the ulnar nerve.
- Muscle dysfunction (weakness/overactivity) of the muscles supplied by the ulnar nerve (flexor carpi ulnaris, flexor digitorum profundus, palmaris brevis, abductor, flexor and opponens digiti minimi, medial two lumbricales, palmar and dorsal interossei, adductor pollicis).
- Symptoms related to overarm throwing, reaching overhead and breast stroke swimming.
- Pathologies such as neck pathologies, shoulder dislocations/subluxations, climber's elbow and golfer's elbow (medial epicondylitis).

Technique

Start the test with the patient lying supine, place your hand on the scapula. Begin the test by pulling their scapula downwards, in the direction of their feet, to cause it to depress. Then, slightly abduct the patient's shoulder (to around 30 degrees) and flex their elbow to 90 degrees. Next, bring the patient's forearm into pronation while maximally extending their wrist and fingers (Figure 25.23). Now, laterally rotate their shoulder to 90 degrees and abduct it (Figure 25.24). Finally, maximally flex their elbow and further abduct their shoulder so that you bring their hand towards their ear until the end of range or they indicate a reproduction of pain/symptoms (Figure 25.25). It is important not to let the shoulder elevate during this test.

Structural differentiation

Structural differentiation can be carried out in the same way as for MNT1 and MNT2 by applying contralateral lateral flexion (Figure 25.26).

Radial neurodynamic test (RNT or ULNT2)

Indications

- Pain and altered sensation related to nerve roots C5–T1.
- Muscle dysfunction (weakness or overactivity) in myotomes supplied by these nerve roots (shoulder

Figure 25.23 Starting position ulnar neurodynamic test.

Figure 25.26 Structural differentiation for ulnar neurodynamic test.

Figure 25.24 Mid-position ulnar neurodynamic test.

Figure 25.25 End-position ulnar neurodynamic test.

abduction, elbow flexion, elbow extension, finger flexion, finger abduction and adduction and thumb extension).

- Pain in the area of the sensory supply of the radial nerve.
- Muscle dysfunction (weakness/overactivity) of the muscles supplied by the radial nerve (triceps, anconeus, brachialis, brachioradialis, extensor carpi radialis longus, supinator, extensor carpi radialis brevis, estensor digitorum, extensor digiti minimi, extensor carpi ulnaris, extensor pollicis longus and brevis, extensor indicis, abductor pollicis longus).
- Symptoms related to throwing (follow through), tennis backhand, swimming (breast stroke, front crawl and butterfly) and golf.
- Pathologies such as neck pathologies, shoulder dislocations/subluxations and tennis elbow (lateral epicondylitis).

Technique

With the patient in the same position as for MNT2, lying supine obliquely across the plinth, stand at the top corner of the bed and depress their scapula by leaning your hip against their shoulder adding slight pressure. Then, abduct their shoulder slightly and, holding their hand, take their forearm into pronation while flexing their wrist and fingers (Figure 25.27). Next, fully extend their elbow and medially rotate their shoulder (Figure 25.28). Finally, abduct and extend their shoulder until the end of range (Figure 25.29).

Structural differentiation

Structural differentiation can be carried out in the same way as for the median (MNT 1 and MNT 2) and ulnar neurodynamic test.

Figure 25.27 Starting position radial neurodynamic test.

Figure 25.28 Mid-position radial neurodynamic test.

Figure 25.29 End position radial neurodynamic test.

TREATMENT PRINCIPLES

General rules when carrying out neurodynamic treatment

- Always carry out the movements slowly;
- After one set of treatments, reassess;
- Do not continue with the same treatment if symptoms or signs worsen on reassessment.

Basically, there are three different treatment approaches once a neurodynamic dysfunction has been identified: neural tissue mobility, inflammation of the innervated tissue or mechanical interface dysfunction. The optimum treatment obviously links to what you find during the assessment.

This section will focus on treating neural tissue mobility. In brief, inflammation of the innervated tissue can be treated by the usual modalities to reduce inflammation, for example ice and anti-inflammatory drugs, but of greatest impact will be to 'offload' the nerve and rest it. Offloading means to avoid positions where the nerve is elongated or interface tissue is in a position where it does not compress the nerve. The mechanical interface treatment applies where peripheral nerves pass through numerous interfaces (e.g. muscles, intervertebral foramen, tunnels), each of which can influence the mechano-sensitivity of the nerve. These different structures should be treated appropriately with, for example massage, stretching and mobilisation techniques. It is beyond the scope of this chapter to discuss the treatment of these interfaces.

Clinical note

- *Interface treatment* – neurodynamic treatment does not only involve mobilisation of the nerve. The interfaces the nerves pass through directly influence nerve mobility; therefore, treating neurodynamic dysfunctions also has to involve the treatment of the interfaces the nerves pass through and relate to.

Neural tissue mobility

To have an optimum function, nerves need to be able to move freely within the nerve bed, as well as in relation to the surrounding tissues. If the movement is impaired, circulation is also frequently compromised. One technique to increase sliding mobility, as well as circulation, is a so-called 'slider'.

To carry out sliders, nerves need to be brought into a position where the slack is taken up, meaning that some

tension needs to be applied to the nerve before starting the sliding movement. The sliding movement should be carried out in mid-range, avoiding positions where the nerve is put into too much tension. The sliding movement can be carried out in one joint (one-ended slider) or in two joints at the same time (two-ended slider). The general rule is that to start a treatment the joint furthest away from the area of the injury is moved. This means, for example, if somebody has a shoulder injury, your slider treatment would start with moving the fingers or wrist.

For the two-ended sliders, you are moving two joints at the same time. One joint will be in the position where the nerve is elongated and the other joint is placed in the position where the nerve is shortened. During movement, the first joint changes into a position where the nerve is shortened and the other joint into a nerve-lengthening position.

Tensioners aim to decrease the mechano-sensitivity of a nerve. This means getting the nerve used to mechanical tension without reacting to it and to increase the visco-elastic properties of the nerve. This involves bringing the nerve into an elongated position (end-range movement), holding this position for a short period of time and then completely releasing the tension. Similar to sliders there are one- and two-ended tensioners, with one-ended tensioners being when load is applied to one end of the nerve bed and two-ended tensioners being when the two ends of the nerve are taken away from each other, simultaneously elongating the nerve from two ends. If tensioners are evoking symptoms such as numbness, tingling, pins and needles, carry out sliders to increase circulation and to reduce these symptoms.

A general rule for slider and tensioner treatment is that if the injury is in a joint, you do not move this joint but leave it in a neutral position that is comfortable for the client. If the injury is in a muscle, you can move the joints either side of the muscle as slider and tensioner treatment.

The number of repetitions and sets of sliders and tensioners depends on the client response to the treatment and how acute the injury is. Always ensure you are retesting after the first set of treatment to evaluate. Adapt your treatment according to the client's reaction.

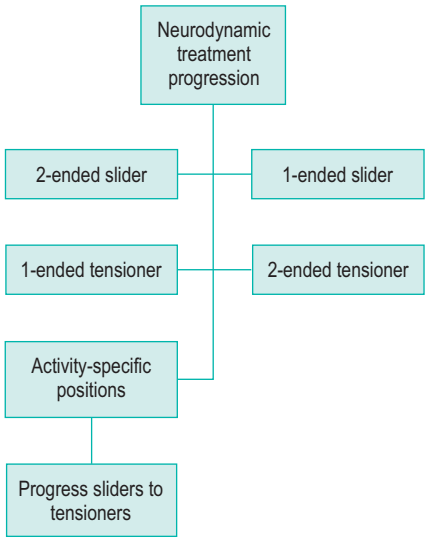


Figure 25.30 Progression of treatment.

Progression of treatment neural dysfunction

To progress treatment the following steps can be taken:

- offloading position;
- move from two-ended sliders through to one-ended sliders into one-ended tensioners to two-ended tensioners;
- increase the range of movement during sliders or tensioners in relation to the relevant treatment barrier;
- increase the number of repetitions;
- increase the number of sets;
- move joint/s closer to the area of the injury;
- carry out neural mobilisation techniques in sport or functionally-related positions (Figure 25.30).

Key point

Caution: assessment and treatment can irritate the patient. The patient must be aware of the potential for flare-ups and how to deal with them (offloading position, etc.).

CASE STUDIES

TYPICAL SCENARIOS WHEN NEURODYNAMIC TESTING AND TREATMENT IS UNDERTAKEN

CASE STUDY 1

Patient presents reporting tingling and sometimes pins and needles at the antero-lateral lower leg. This started after playing netball without any trauma. Slump, SLR and peroneal test were positive; tibial test was negative. Treatment: 3 × 10 two-ended sliders of peroneal nerve showed some immediate improvement. Continued with 3 × 10 one-ended sliders in combination with interface treatment of the peronei muscles and the problem was resolved.

CASE STUDY 2

Patient presents following a hamstring 'strain' playing football. He has pain on resisted knee flexion and local tenderness in the lateral hamstring on palpation. SLR showed reduced range and was painful; structural differentiation was positive. After 3 × 20 two-ended sliders, SLR had full strength with slight ache on resisted knee flexion; SLR range significantly improved.

CASE STUDY 3

Patient presents with a tennis elbow which is not responding to conventional treatment of ultrasound, deep transverse friction massage and stretching. Radial neurodynamic test was positive on structural differentiation. Treatment of 2 × 10 one-ended sliders and 2 x 10 tensioners was successful and radial neurodynamic test was negative.

REFERENCES

Breig, A., 1978. Adverse Mechanical Tension in the Central Nervous System. Almqvist and Wiksell, Stockholm.

Butler, D.S., 1989. Adverse mechanical tension in the nervous system. A model for assessment and treatment. Aust J Physiother 35 (4), 225–238.

Butler, D.S., 1991. Mobilisation of the Nervous System. Churchill Livingstone, Edinburgh.

Butler, D.S., 2000. The Sensitive Nervous System. Noigroup Publications, Australia.

Butler, D.S., Gifford, L., 1989. The concept of adverse mechanical tension in the nervous system. Physiotherapy 75 (11), 622–629.

Coppieters, M.W., Butler, D.S., 2008. Do 'sliders' slide and 'tensioners' tension? An analysis of neurodynamic techniques and considerations regarding their application. Man Ther 13 (3), 213–221.

Keneally, M., Rubenach, H., Elvey, R., 1988. The upper limb tension test: The SLR of the arm. In: Grant, R. (Ed.), Physical Therapy of the Cervical and Thoracic Spine. Churchill Livingstone, New York, pp. 167–194.

Kobayashi, S., Shizu, N., Suzuki, Y., et al., 2003. Changes in nerve root motion and intraradicular blood flow during an intraoperative straight-leg-raising test. Spine 28 (13), 1427–1434.

Michael-Titus, A., Revest, P., Shortland, P., 2007. The Nervous System. Basic Science and Clinical Conditions. Churchill Livingstone, Edinburgh.

Millesi, H., Zoech, G., Reihsner, R., 1986. Mechanical properties of peripheral nerves. Clin Orthop Relat Res 314, 76–83.

Ogata, K., Naito, M., 1986. Blood flow of peripheral nerve effects of dissection, stretching and compression. J Hand Surg 11 (1), 10–14.

Shacklock, M., 1995. Neurodynamics. Physiotherapy 81 (1), 9–16.

Shacklock, M., 2005. Clinical Neurodynamics. Elsevier, Edinburgh.

Upton, A.R., McComas, A.J., 1973. The double crush in nerve entrapment syndromes. Lancet 2 (7852), 359–362.

Chapter 26

Neurological physiotherapy

Christine Smith, Anita Watson and Louise Connell

INTRODUCTION

Neurology is a specialised field that deals with disorders of the nervous system. There are a vast number of neurological conditions that can affect the central nervous system (CNS – brain and spinal cord), the peripheral nervous system and the autonomic nervous system. The challenges facing physiotherapists working in the clinical field of neurology are many and varied. The complex nature of the human nervous system and the broad range of neurological conditions found in clinical practice place heavy demands on physiotherapists. The onset of a neurological condition as a result of disease or trauma has a devastating effect not only on the patient but also on their families. It is essential that any approach to management encompasses the needs of all parties. Many neurological conditions are progressive and longstanding, and result in some element of residual impairment. There are numerous challenges in the rehabilitation process from early diagnosis and treatment through to discharge. The longstanding nature of many neurological conditions means that professionals are involved in the management and rehabilitation of patients over a number of years.

An emerging evidence base surrounding the issues of neurological rehabilitation means that the role of the physiotherapist in treating and managing neurological conditions is developing. Current research identifies the importance of task-specific, strength and repetitive training. Alongside this the use of novel interventions, such as functional electrical stimulation (FES) and constraint-induced movement therapy (CiMT) are advocated. Research has shown that no one treatment approach is superior to any other; instead, practitioners need to select the most appropriate intervention based on the available evidence base. Neurological damage can result in the disruption of normal physical, psychological, cognitive and social functions, which reinforces the need for a collaborative and co-ordinated approach from a wide range of rehabilitation professionals. It is vital that a truly holistic approach to the management and treatment of patients and their families be adopted, with clinical reasoning and problem-solving at its centre. Practitioners need to understand both the patients' and carers' perspective. Research has shown that therapists' and patients' goals often differ. As such, measurement of outcome is an important aspect of neurological rehabilitation. The chapter provides a section on outcome measurement and goal-setting in relation to the International Classification of Function (ICF) framework set out by the World Health Organization (WHO) in 2001. The most appropriate measures have been selected based on their psychometric properties. Readers should refer to Tyson et al. (2008) for further information.

This chapter begins by outlining the principal causes of neurological damage and then describes types of movement disorders and the most frequently encountered clinical features. The clinical features have been grouped together into categories to aid cross reference within the assessment process. Physiotherapy assessment and the main evidence-based interventions are then discussed in relation to present-day clinical practice. A new section on promising interventions that use rehabilitation technologies has been included to demonstrate their growing use in healthcare practice. The chapter concludes with an overview of the more commonly known neurological conditions, namely stroke, multiple sclerosis, motor neurone disease and traumatic brain injury (Figure 26.1). These sections are not intended to be exhaustive and readers can consult the lists of further recommended reading at the end of the chapter.

Further reading

Although neurological rehabilitation remains a new science in comparison with some other subject areas there is now an emerging evidence base for the types of interventions practitioners should be considering. Readers are directed to Shumway-Cook and Woollacott (2007).

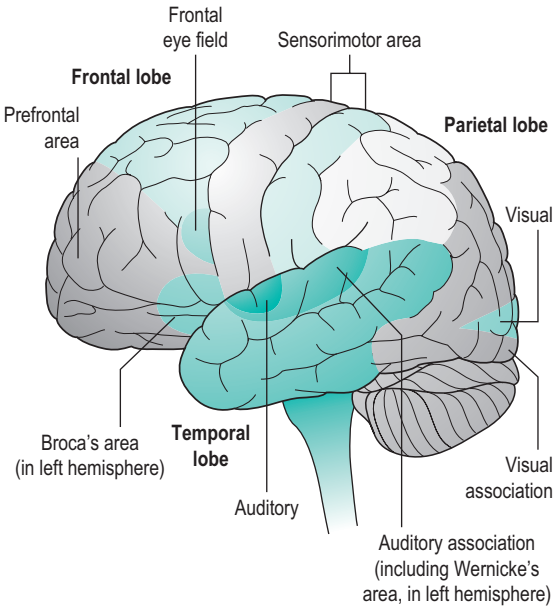


Figure 26.1 Brain.

Background knowledge

Readers are advised to refer to appropriate literature for details of the anatomy of the CNS in relation to its function. A thorough understanding of the structure and function of the CNS and the neural control of movement will be needed to effectively manage patients with complex neurological conditions.

Glossary

Towards the end of this chapter is a glossary of some of the terms used within neurology.

THE FUNDAMENTALS OF CNS DAMAGE

Principal causes of neurological damage

The most commonly occurring neurological conditions are those that occur as a result of problems within the circulatory system, namely stroke (see later section). Other causes of neurological damage include trauma, as in head injury or spinal injury, where direct or indirect trauma results in temporary inflammation processes occurring and, therefore, repairable or permanent damage (if there has been destruction of nerve tissue). Diseases can affect the nervous system and some seem to have an affinity for a particular part of the system. Examples of diseases that can affect the nervous system are meningitis, syphilis, and (less commonly) tuberculosis and poliomyelitis. Diseases of unknown origin that affect the CNS include multiple sclerosis and motor neurone which are discussed later in the chapter.

Congenital defects, such as spina bifida, inherited conditions, such as Huntington's chorea, spinocerebellar ataxia, vitamin B deficiency and neoplasms, and toxic substance poisoning, such as lead, arsenic and mercury, can all also have a detrimental effect on the nervous system. However, these are less common occurrences and therefore will not be covered within this chapter. Peripheral neuropathies and fibromyalgia are also not covered.

Clinical features of damage to the CNS

Movement disorders

The result of abnormal tone, altered motor control and impaired sensory input will lead to what is known as a movement disorder. Day-to-day functions, such as walking, dressing and eating, become difficult to perform owing to movements becoming un-coordinated, uncontrollable and involuntary. Common movement disorders are listed below and can be seen with conditions such as stroke, multiple sclerosis and Parkinson's (see Chapter 24).

Ataxia

Movements are un-coordinated and jerky. Functional activities like walking look unsteady and arm movements are difficult to control leading to overshooting or undershooting of the hand to a target. Ataxia is found as a result of damage to the cerebellum, sensory and vestibular systems.

Dystonia (previously known as athetosis)

Movements produced are writhing, slow and lead to abnormal sustained postures being adopted. Generalised dystonia may produce gross movements of the arms and legs. Focal dystonias usually involve the eyes, neck or upper limbs. They are thought to occur as a consequence of damage to the basal ganglia. There may also be disruption in facial and tongue movements. It is thought that these movements may be owing to a disturbance in reciprocal inhibition.

Chorea

Movements are jerky but tend to occur more randomly throughout the body. The absence of sustained abnormal posturing distinguishes this condition from dystonia.

Ballismus

Movements are large and sudden, and can affect one side of the body (hemiballismus).

Tremor

Movements are fine, rapidly oscillating and unwanted. Tremors are often classified in relation to the circumstances in which they occur, such as an intention tremor which is found when a movement of a limb is made towards a target. Many other tremors are known but will not be covered in this chapter.

Bradykinesia

Movements are slow. This disorder is commonly seen in Parkinson's (see Chapter 24).

Impairments

The signs and symptoms of damage or disease to the CNS are commonly known as clinical features. These clinical features are also described as *impairments* when using the ICF classification of functioning, disability and health (ICFDH – see later section). The site and severity of disruption to the CNS will determine which impairments are seen. However, because the CNS is integrative, damage to one part can result in a disruption of function of other parts. This means that the site of damage alone is not necessarily a predictor of the impairments the patient will present with. For the purpose of classification, *impairments* will be considered under the headings of motor impairments, sensory impairments, visual impairments, congnitive impairments, behavioural changes, perceptual disturbances, auditory disturbances, communication disturbances/swallowing, fatigue, bladder/bowel incontinence and automatic disturbances.

Motor impairments

Damage to neural motor pathways or motor cortex areas, and direct muscle damage will lead to a loss or limitation in muscle function.

- *Muscle weakness and/or atrophy* – a reduction in muscle strength of a single muscle or groups of muscles. Weakness can be specific to a side, such as hemiplegia, or be generalised as a result of a person no longer performing physical activities (deconditioned). Atrophy describes muscle loss owing to lack of use.
- *Muscle tone* – this is the 'state of readiness' of an individual's muscles at rest. It is vital for maintaining posture. The continuous small adjustments of the muscle sensory receptors (muscle spindles and Golgi tendon organs (GTOs) prepare the muscles for movement). Muscle tone can become increased (hypertonic) or decreased (hypotonic) depending on the amount of information being sent to the muscle receptors. This means that damage to the CNS can result in altered muscle tone.
- *Spasticity* – is 'a motor disorder, characterised by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyper-excitability of the stretch reflex as one component of the upper motor neurone (UMN) syndrome' (Lance 1980).
- *Hypotonia* – is low muscle tone. Muscle receptors are unprepared for movement resulting in resistance-free movement and weakness of muscles.
- *Rigidity* – is an increase in muscle tone which leads to a resistance in passive movement. There are two types commonly found in Parkinson's:
  - lead pipe rigidity manifests as a uniform resistance to movement throughout the range of movement;
  - cogwheel rigidity presents as an intermittent on/off resistance throughout the range of movement, making the movements jerky.

Sensory impairments

Damage to the sensory receptors, sensory cortex areas or pathways carrying sensory information, such as touch, joint position and pain, will result in the following:

- *altered cutaneous sensation* – the skin sensation can become diminished (anaesthesia), absent (parasthesia) or more sensitive (hyperaesthesia) to inputs such as light and deep touch, pressure, vibration and two-point discrimination;
- *altered proprioception* – the muscle sensory receptors (muscle spindles and GTOs) are unable to provide information about a joint's position. This means that an individual may be unable to recognise what position a body part is in;
- *pain* – damage to nerve endings and/or pain pathways, as well as pain caused by secondary complications, such as malalignment of joints (owing to swelling or poor positioning of soft tissue structures) will result in pain.

Visual impairments

Damage to the visual cortex area, optic nerve and motor pathways controlling muscle function of the ocular system will result in the following:

- *hemianopia* – loss of vision in half of the visual field of each eye;
- *diplopia* – double vision;
- *nystagmus* – un-coordination of the eye muscles leading to fast or slow alternating movements of the eye when asked to move them or hold them on a target.

Cognitive impairments

Damage to the parietal lobe can lead to loss in the ability to process, learn and remember information:

- attention;
- memory;
- learning;
- emotional changes.

Damage to the limbic system can lead to the following situations:

- emotional lability;
- depression;
- euphoria.

Behavioural changes

Damage to the frontal lobe can lead to the following behaviour issues:

- agitated states;
- disinhibition;
- reduced motivation.

Perceptual disturbances

Perception is the process of acquiring, interpreting, selecting and organising sensory information. Damage to areas within the brain that co-ordinate sensory and motor information can lead to the following:

- figure–ground differentiation;
- spatial awareness;
- inattention or neglect;
- disturbances in constructional abilities.

Apraxia is a disorder of planning. The individual has the ability to perform a movement or talk about what the task will involve but is unable to perform the task in the correct sequence.

Cognitive, emotional, behavioural and perceptual impairments will all affect the success of an intervention. An individual's ability to concentrate, remember, understand and be motivated will determine the effectiveness of a treatment. It is important that these impairments are assessed by the multi-disciplinary team (MDT) to ensure that each healthcare profession can manage these aspects within their treatments.

Auditory disturbances

Deafness directly related to neurological conditions is relatively uncommon in neurological patients. When it occurs it is usually a result of trauma to the structures of the auditory system.

Communication disturbances/swallowing

Damage to Broca's area within the brain and motor components that produce muscle movement can lead to the following:

- *dysarthria* – a difficulty in articulating words owing to muscle weakness or lack of co-ordination producing slurred speech;
- *dysphasia* (expressive and receptive) – the processing part of speech is affected. Individuals can have difficulty saying what they want (expressive) or understanding what the word means (receptive).

Fatigue

A sense of overwhelming tiredness that is not fully understood. This is a common feature for multiple sclerosis patients, who struggle to perform functional activities owing to fatigue and not physical weakness. Fatigue should not be confused with poor endurance. Endurance relates to an individual's ability to maintain a period of aerobic exercise. A person with poor endurance is likely to have limited cardiovascular fitness rather than be fatigued.

Bladder/bowel incontinence

Incontinence can be a result of damage to the control centres of the brain for bodily functions (see autonomic disturbances) but also damage to motor pathways producing muscle control.

Autonomic disturbances

Autonomic dysfunctions may be a result of damage affecting autonomic areas or pathways. Following spinal cord injury, some patients can present with autonomic dysreflexia. This is a sympathetic nervous system discharge producing hypertension, bradycardia, sweating, skin vasoconstriction, headache, pilo-erection and capillary dilation. This response is often triggered by impaired bladder or bowel function, or other noxious stimulus.

ASSESSMENT OF NEUROLOGICAL PATIENTS

As in all areas of physiotherapy, assessment is an ongoing process that is an integral part of treatment. Assessment of neurological patients is complex, and a particular diagnosis can result in very different presentations. Many members of the healthcare team will carry out assessments. Wherever possible, joint assessments with sharing of information will be carried out. Often, the initial assessment occurs over a number of sessions, because carrying out a full assessment in one attempt can frequently be too tiring for the patient. Gathering information over a longer period of time invariably gives a much more accurate picture of the patient's strengths and difficulties.

Why do we do assessments?

- To identify the patient's problems.
- To establish or define the patient's level of impairment and activity and participation limitations.

- To establish baseline measurements of the patient's impairments, activity and participation limitations, and goals.
- To inform treatment choices.

Assessments generally include a subjective assessment (questioning) and an objective assessment (clinical examination). In addition, the use of standardised outcome measures is recommended as a way of promoting evidence-based practice and is part of good clinical practice.

Subjective assessment

Obtaining a thorough subjective history is very important. This can come from a variety of sources: the medical notes, nursing notes, the patient, relatives and carers. Initial interviews can be important, not just for gathering information, but also because this is where establishing a rapport with the patient and carers begins.

Physiotherapists need to draw on all aspects of their communication skills to allow the patient and family to be at ease. Increasingly, in appropriate cases, initial interviews take place in the patient's home. This not only ensures the person is more relaxed, but allows assessment of the patient's functional environment. Later, when home programmes are being devised, knowledge of the patient's home can be invaluable.

When assessing patients with communication disorders, it is important to find alternative or supplementary methods of communicating with the patient. Collaboration with the speech and language therapist is invaluable at such times.

While interviewing the patient, important clues can be observed with regard to physical abilities, communication, cognition, emotion, hearing, vision and attention span. It should also be noted how much the patient changes position, attends to posture and so on. Assessment thus begins the minute contact is made with the patient.

The assessment should include both background history and also components specific to physiotherapy. Background history should include demographic details, past medical history, diagnosis, history of present condition, present condition, medication, social factors (housing, family, lifestyle, care circumstances) and details of the involvement of other healthcare professionals.

Objective assessment

The main purpose of the clinical examination is to gather information about the patient's movement disorder and level of functional ability (Freeman 2002). Initially, observe the patient and try to draw conclusions about what they are able to do unaided. It is important to remember that at no point must the patient be put at risk. It is part of the physiotherapist's role to 'risk assess' what is reasonable to allow the patient to attempt independently and what they need help with in order to be safe. Objective assessment should include assessment of active and passive range of movement and strength. This will guide observation of posture and movement analysis in sitting, lying to sitting, sit-to-stand, transferring from bed to chair, standing and walking, etc. The exact activities observed and/or assisted will depend on the level of ability of the patient. Once the patient has been given the opportunity to demonstrate what they can do unaided, assistance can then be given to ascertain what the person can achieve with help. This is where assessment and treatment begin to merge into one and the same thing. The assessment begins when initial contact is made. For instance, observing the person's gait when they walk into the room often gives a more accurate picture of functional gait pattern than when the person is asked to walk while being watched.

International Classification of Functioning, Disability and Health

The WHO provides a useful framework and common terminology for describing and measuring the consequences of disease and the impact that rehabilitation may have on them (Edwards 2002). This is the *International Classification of Functioning, Disability and Health*, known more commonly as ICF, which provides a standard language and framework for the description of health and health-related states. This describes changes in body function and structure (impairment), what a person with a health condition can do (their level of ability), as well as what they actually do in their usual environment (their level of participation).

- 'Body function' considers the loss or abnormality of body structure, or of a physiological or psychological function.
- 'Activity' refers to the nature and extent of functioning at the level of the person. It may be limited in nature, duration or quality. It considers if a person can do a task and how well they do it.
- 'Participation' refers to the nature and extent of a person's involvement in life situations in relation to impairment, activities, health conditions and contextual factors.

Research by the Greater Manchester Outcome Measures Project team identified the domains that physiotherapists need to measure during clinical assessment in neurological patients using the ICF as a framework. (Tyson et al. 2008). They suggested neurological assessments should include assessment of:

Impairments (problems in body functions or structures)

- ataxia/co-ordination
- balance impairment: postural sway
- oedema
- muscle tone/spasticity

- pain
- personal fatigue
- posture impairment: alignment, weight distribution
- range of movement/contracture
- sensation and proprioception
- subluxation
- walking impairment: speed, endurance, step length, cadence, etc.
- weakness

Activities (ability an individual has in executing activities)

- balance activity – sitting, standing, dynamic, supported, assisted
- mobility activity – bed mobility, sit-to-stand, transfers, walking, stairs/steps, etc.
- upper limb activity – grips/grasps, dexterity, activities of everyday life
- walking activity – in/outdoor, assistance, use of equipment, independence.

Ultimately, physiotherapy aims to improve an individual's ability to undertake activities to improve function, participation and quality of life.

OUTCOME MEASURES

Objective measurement of function is core within rehabilitation, with both clinical guidelines and professional bodies such as the Chartered Society of Physiotherapy (CSP) explicitly stating that standardised outcome measures should be used. Therefore, part of the objective assessment should include use of standardised outcome measures, which provide baseline information on individual's ability and can be repeated to evaluate effectiveness of treatment. In the current climate of limited resources and financial cuts, it is vital that physiotherapists undertake robust, standardised measures to enable evaluation of the effectiveness of their treatment at an individual and service level. It is no longer acceptable to state physiotherapy treatment is effective – we need to be able to provide evidence to justify this.

Measurement implies the quantification of data in either absolute or relative terms. Determining the effectiveness of an intervention by measuring its effect on an outcome provides the basis for evidence-based healthcare (Edwards 2002). Outcome measures take the guesswork and subjectivity out of evaluation and can assist the physiotherapist in proving clinical effectiveness.

Outcome measures also provide a method of communication. The importance of a language of universal use among clinicians must be promoted. The focus on multidisciplinary care and the blurring of traditional professional boundaries requires, at the very least, a system of measurement that can be understood by and utilised by the whole interdisciplinary team.

CSP core standards of physiotherapy practice (2005) standard 6

Taking account of the patient's problems, a published, *standardised, valid, reliable and responsive* outcome measure is used to evaluate change in the patient's health status.

The outcome measure should be:

- applicable to the clinical setting;
- relevant to the patient's problems;
- related to the aims of treatment;
- standardised, valid, reliable and responsive measure;
- used to evaluate change against the aims of treatment.

Physiotherapists should:

- have the necessary skill and experience to administer and interpret;
- ensure the instruction manual is followed to administer and score.

There are a wide range of outcome measures available and care is required when deciding which outcome measure to use and when. The general principles to follow when developing and/or considering the use of outcome measures are:

- deciding what needs to be measured;
- selecting the appropriate outcome measure;
- does it measure what you want it to measure (reliability, validity and sensitivity)?;
- ease of use on a day-to-day basis (clinical utility).

For a discussion of outcome measurement theory and properties in rehabilitation, we refer the reader to Finch et al. (2002).

CSP website

The CSP has provided a summary of the outcome measures currently available, which have relevance to physiotherapy care interventions. It can be found on the CSP website at www.csp.org.uk.

Outcome measures should be sensitive enough to allow for measurement of changes over time. The use of a suitable outcome measure for all physiotherapy interventions is an essential part of the treatment and management process, and the clinical area of neurology is no exception.

Outcome measures in context

Physiotherapy outcome measures should be considered in the wider context of rehabilitation. Rehabilitation can be considered a problem-solving and educational process aimed at reducing disability and enhancing function in people who are affected by disease (Wade 1992). Rehabilitation principles are based upon the enhancement of

activity by restoring skills and capabilities through functional retraining and environmental adaptation. Rehabilitation promotes independence and aims to facilitate the fullest potential physically, psychologically, socially and vocationally for a patient. Rehabilitation involves the recovery or improvement of function, as well as prevention of disability and the maintenance of a social role.

Irrespective of the approach taken towards rehabilitation, the ability to quantify the function is the key to successful treatment. This process involves assessment, treatment-planning, goal-setting and evaluation of outcome. The WHO ICF classification and the process of rehabilitation together provide the context for which outcome measurement is used in physiotherapy.

Table 26.1 provides a summary of the outcome measures commonly used within neurology. It is not meant to be comprehensive and the reader is directed to the 'Further reading' list at the end of the chapter.

Table 26.1 Outcome measures frequently used within the clinical setting

Dimension	Outcome measure	References
Impairments		
Muscle strength	Hand-held dynanometer Medical Research Council (MRC) grades	Bohannon et al. (1995) MRC (1976)
Range of motion	Goniometry	Norkin and White (1975)
Tone	Modified Ashworth Scale	Bohannon and Smith (1987)
Sensation	Nottingham Sensory Assessment	Lincoln et al. (1998)
Fatigue	Fatigue Severity Scale	Petajan et al. (1996)
Activity		
Global activity		
Generic	Barthel Index Functional Independence Measure	Mahoney and Barthel (1965) Granger et al. (1993)
Disease-specific	Motor Assessment Scale for Stroke	Carr et al. (1985)
Focal activity		
Gait	Ten-metre timed walking test Rivermead Mobility Index	Wade (1992) Collen et al. (1991)
Mobility	Rivermead Motor Assessment	Lincoln and Leadbitter (1979)
Balance	Brunel Balance Assessment Berg Balance Scale Functional Reach Test Timed get-up-and-go test	Tyson & DeSouza (2004) Berg et al. (1989) Duncan et al. (1990) Podsialo and Richardson (1991)
Upper-limb function	Box and Block Test Nine-hole Peg Test Action Research Arm Test	Mathiowetz et al. (1985) Mathiowetz et al. (1985) Crow et al. (1989)
Participation	London Handicap Scale Environmental Status Scale	Harwood et al. (1997) Mellerup et al. (1981)
Quality of life		
Generic	Thirty-six-item Short Form Health Survey Nottingham Health Profile	Ware et al. (1993) Hunt et al. (1981)
Disease-specific	Thirty-nine-item Parkinson's Disease Questionnaire Functional Assessment of Multiple Sclerosis	Peto et al. (1995) Cella et al. (1996)

Goal-setting

ABC Definition

Goal-setting refers to the identification and agreement of targets that the patient, therapist and team will work towards over a specified period of time (Wade 1999a, 1999b).

Once the assessment is complete, key problems need to be identified by using clinical reasoning processes. Goals of treatment, with appropriate timescales, can be formulated with the patient and the family. Goals need to be negotiated and discussed with all parties, including the rehabilitation team, so that there is a clear understanding of what is involved in achieving them. Failure to go through this process can lead to frustration and misunderstanding for all parties, particularly the patient and the family.

The planning of goals is necessary to ensure that the rehabilitation effort is as effective and efficient as possible (Elsworth et al. 1999). Outcome is better if the goals involve the patient, are challenging and are set at different levels.

The evidence relating to goal-setting is limited, but there is a general trend towards the inclusion of goal-setting in the rehabilitation process (Wade 1999c). With the emphasis on patient-centred care and inclusion of the patient in the decision-making processes, a formal process of goal planning will help to improve the co-ordination and co-operation of all those people involved. Co-operative goal setting makes the process of rehabilitation more patient-focussed and helps to motivate the patient through the long period of rehabilitation and beyond. In other words the effects of treatment will be long-lasting and continue to be evident when treatment has ceased.

Good rehabilitation practice should involve SMART goals (specific, measurable, achievable, realistic, time-framed).

1. Set meaningful and challenging but achievable goals.
2. Involve the patient and carers.
3. Include short- and long-term goals.
4. Set goals both at a team and an individual professional level.

It is common practice to set goals for the long, medium and short term. In summary, the terms commonly used to document goals are:

- long-term goals = aims;
- medium-term goals = objectives;
- short-term goals = targets.

Wade (1999a) defines these terms as given in Table 26.2.

In summary, goal-setting allows for the alignment of patient and professional goals. It is a method of ensuring that the rehabilitation team focusses on the needs of an individual patient and can help to motivate patients. It should lead to an overall improvement in treatment effectiveness and provide a method of measuring the effectiveness of treatment interventions (McGrath and Adams 1999; Edwards 2002).

Table 26.2 Aims, objectives and targets

	Describes a state
Aim	Is for the patient and family Is in terms of a social role or functioning or well-being
Objective	Is set within the medium term Involves direction of change as much as achieving a specific state Is framed in terms of patient behaviour and environment
Target	Is set within the short term Is specific and often involves only one named person/profession May be set at any level or in terms of the rehabilitation process

Adapted from Wade (1999b).

List of problems and goals

Now the assessment is complete, a problem and goals list needs to be formulated. It is important to bring the problems identified as a physiotherapist together with the patient's/carer's perceived problems. Problems and goals need to be agreed with the patient and family. At the end of the day it is important to remember that the goals should be the patient's, not the physiotherapist's (Table 26.3).

INTERVENTIONS

Task-specific practice

Ultimately, the majority of treatment aims to increase ability at a task or activity, and, consequently, one approach to treatment is simply to practise the task itself. Task-specific practice – repeated practice of tasks similar to those commonly performed in daily life – is a component of current approaches to stroke rehabilitation. In the past the focus of treatment was on reducing impairments in the expectation that activities would naturally improve.

Systematic reviews of treatment interventions suggest that participants benefit from exercise programmes in

Table 26.3 Problems and goals

Problem list (with date set in order of priority)	Reason for problems	Agreed goals (in order of priority)	Review date	Treatment plan	Date goal achieved, signature and job title. If not achieved state reason why

which functional tasks are directly trained, with less benefit if the intervention is impairment-focussed (Van Peppen 2004). Examples of task-specific practice include practising sit-to-stand, walking and reaching activities, and are often mixed with other components, including strengthening and treadmill training.

How the intervention might work

Many aspects of rehabilitation involve repetition of movement. Repeated motor practice has been hypothesised to reduce muscle weakness and spasticity (Feys 1998, Nuyens 2002), and to form the physiological basis of motor learning (Butefisch 1995), while sensorimotor coupling contributes to the adaptation and recovery of neuronal pathways (Bruce and Dobkin 2004). Active cognitive involvement, functional relevance and knowledge of performance are hypothesised to enhance learning (Carr and Shepherd 1987).

Evidence

Most research has been undertaken on patients with stroke. A Cochrane review, acknowledged as the best single source of evidence about the effects of healthcare interventions, has been undertaken on repetitive task training for improving functional ability after stroke (French et al. 2007). This showed repetitive task training resulted in modest improvement in lower limb function, but not upper limb function. Training may be sufficient to impact on daily living function. However, there is no evidence that improvements are sustained once training has ended. An evidence-based review of upper limb interventions found repetitive task-specific training may improve arm function (EBRSR 2009).

UK stroke guidelines

Emerging evidence suggests that giving the patients the opportunity to practise functional activities (task-specific training) may be important in improving outcomes. The UK clinical guidelines (Intercollegiate Stroke Working Party 2008) for stroke state:

Task-specific training should be used to improve activities of daily living and mobility:

- *Standing up and sitting down*
- *Gait speed and gait endurance*

Exercise

Exercise (as covered in Chapter 13) includes stretching, strengthening and aerobic types of activities. If performed regularly with enough repetitions and appropriate duration these activities can develop into an exercise programme that will improve an individual's range of movement, muscle strength and physical fitness.

Current literature reviewing the effectiveness of these types of exercise now shows that strengthening exercises are beneficial to many types of neurological patients in the acute, rehabilitation and community settings (Weiss et al. 2000; Romberg et al. 2004). A Cochrane review suggested that aerobic exercise to develop physical fitness in stroke patients can improve their walking speed, tolerance and level of mobility (Saunders et al. 2009).

Any activity that seeks to enhance or maintain a function or movement can be classed as exercise. Task-specific training is one such form of exercise that has been proven to improve function in neurological rehabilitation (see above).

In summary, exercise should be considered a key part of the rehabilitation programme in the acute, rehabilitation and community settings. The exact number of repetitions, intensity, how often and content are currently being looked into. The reader should review the emerging evidence base to identify what will work best.

Treadmill training

The recovery of walking is important to function. It influences the discharge destination and package of community support an individual receives when leaving a rehabilitation environment. Physiotherapists concentrate a large amount of time on regaining safe mobility, be it independent or with an assistive device. The importance of repetitive practice (as highlighted by task-specific training and strengthening) has forced therapists to look at alternative methods for achieving increased intensity and duration of walking practice.

The use of walking aids allows body weight to be supported but leads to an altered gait pattern, which can be tiring to maintain. Hoisting equipment allows a patient's body weight to be partially supported while walking and a more normal gait pattern to be adopted. However, a large space is required to walk a patient with this type of equipment so intensity, distance and duration are low. The introduction of a treadmill, which provides a constant flat moving surface, gives patients the chance to walk greater distances without moving around a room and provides

Figure 26.2 Treadmill.

safety with the use of a partial body weight support (PBWS). This type of walking practice can reduce the number of therapists needed to walk a patient (Figure 26.2).

A recent review by Ada et al. (2010) shows that mechanically-assisted walking with body weight support systems is more effective than overground methods of gait rehabilitation and does not have any detrimental effects on an individual's walking speed or capacity. Again, the reader is advised to look at the current published literature available for treadmill training.

Novel interventions

Within the current health and economic climate, providing adequate rehabilitation services is becoming increasingly challenging (RCP 2004). Arguably, as a result of this, there has been a growing recognition that different approaches, such as constraint-induced movement therapy (CiMT), are needed to complement, or sometimes replace, existing approaches.

Constraint-induced movement therapy

CiMT is an approach proposed by Edward Taub in the 1980s as a means of treating the upper limb following a stroke. This form of therapy involves restricting the use of the unaffected limb (often by wearing a glove) while targeting the affected limb with intensive (up to six hours per day), task-orientated, functional retraining (shaping). This combined approach appears to prevent the learned non-use phenomenon so often seen in stroke patients by using a restraint on the unaffected hand combined with an intensive practice schedule designed to facilitate neuroplastic changes in the CNS. Over the last few years this approach has been gaining in popularity and in 2008 a high-quality randomised controlled trial of over 200 patients (Wolf et al. 2008, on the EXCITE trial) demonstrated that the patients who received CiMT achieved substantial improvements in the functional use of their affected arm and quality of life, even two years after treatment had stopped. In spite of the promising results from these studies, there remains a problem with adoption of this approach into mainstream clinical practice.

In addition to this there has been a growing recognition that technology could play a significant role in the rehabilitation programmes of the future. Virtual reality, robotics and functional electrical stimulation (FES) are three emerging rehabilitation interventions that have been incorporated into rehabilitation programmes.

Virtual reality

Virtual reality (VR) is a computer-based, interactive, multi-sensory simulation environment. The main types of VR are immersive and non-immersive. In immersive VR systems, the user feels as though they are actually present in the computer-generated world. Non-immersive VR systems still require the user to interact with the environment but provide a lesser feeling of 'presence' within the virtual world. Supporters of VR feel that it provides a safe environment for users to learn or relearn skills that can be fun and help to motivate the user (Rizzo and Kim 2005). VR systems fulfil many of the requirements for sensory-motor relearning by being flexible enough to allow the task to be tailored to meet the needs of the client and afford the achievement of a variety of rehabilitation goals (Page 2003). In addition to this, another vital component for sensory-motor relearning is provided, namely feedback on performance of the task to both the client and the therapist (Adamovich et al. 2009).

An extensive review of English-language scientific literature was carried out by Henderson et al. (2007) on the use of immersive and non-immersive VR in rehabilitation of the upper limb post-stroke in acute, sub-acute and chronic stroke, adult patients. The review concluded that the current evidence on the effectiveness of VR in the rehabilitation of upper limb in clients post-stroke is limited but sufficiently encouraging to justify further research in this area (Figure 26.3).

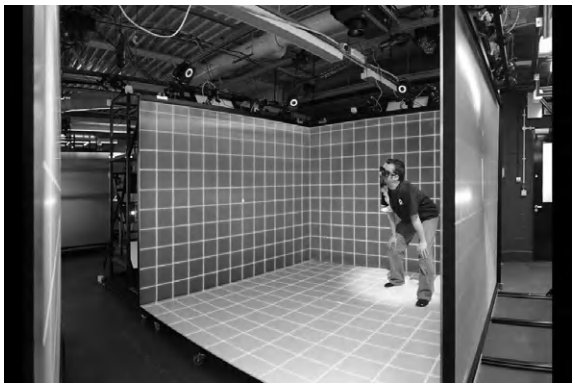


Figure 26.3 Repositioning of objects in an immersive virtual environment (CAVE). *Reproduced with permission of the Centre for Virtual Environments, University of Salford, UK.*

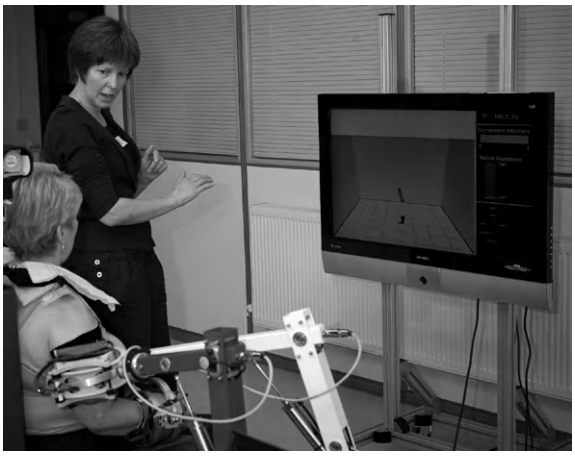


Figure 26.4 iPAM: A physiotherapist instructing the patient on how to use the stroke rehabilitation robotic system for arm exercises. *Reproduced with permission of iPAM team, Leeds University, UK.*

Robotics

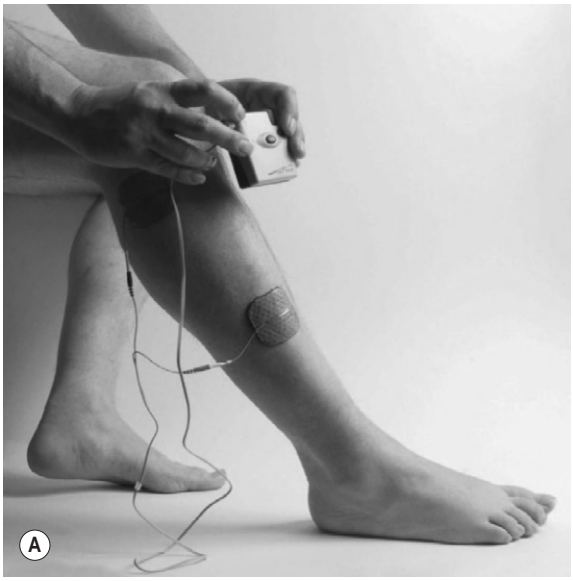
Robotic devices have been developed to try to offer an alternative intervention for treatment of upper limb post-stroke, especially for patients with little, or no, use in their affected arm. One of the main drivers for robotics has been the need to deliver a large number of repetitive movements in order to facilitate recovery. Rehabilitation in the early stages of stroke requires a high percentage of therapist's time and is labour intensive. Two examples of these robotic devices are MIT-MANUS (or the In-Motion system) and GENTLE, which are single point of contact robotic designs. More recently, a dual point of contact robotic system, iPAM, has been developed to more closely mimic the manner in which a therapist handles the upper limb (Jackson et al. 2007). There is some evidence to suggest that robotic-aided therapy could be more effective than conventional therapy (Prange et al. 2006); however, once again, in spite of these promising findings, the uptake of robotic therapy remains in its infancy owing to the high cost of equipment and its lack of usability in the home environment (Figure 26.4).

Functional electrical stimulation (FES)

One intervention that is showing significant promise for the restoration of function is FES. FES is 'the use of Neuromuscular Electrical Stimulation to activate paralysed muscles in a precise sequence and magnitude so as to directly accomplish functional tasks' (Sheffler and Chae 2007). The most established application for FES is in the treatment of 'drop foot' post-stroke. In 2009, the National Institute for Health and Clinical Excellence (NICE) concluded that the evidence base for FES in drop foot was adequate to support its use. The NICE Specialist Advisers reported that the key efficacy outcomes of FES included improved gait, reduction in effort when walking, reduction in pain and discomfort, reduction in falls, return to work and other quality of life outcomes. FES stimulates the common peroneal nerve via surface stimulation or implantable electrodes. In turn, this creates muscle activation of the anterior tibial and peronei muscles resulting in a lifting of the foot to achieve toe clearance and aiding more consistent foot placement on the ground. A small but growing number of patients elect to have electrodes implanted into the epineurium of the peroneal nerve as an alternative to using surface stimulation. In both methods, the electrodes are linked to a stimulator unit either by leads or by using wireless technology. A pressure sensor switch under the foot determines when stimulation occurs (Figure 26.5).

The use of FES for the upper limb is also gathering a more convincing evidence base. Recent studies by Popovic et al. (2005) and by the Salisbury group (Mann et al. 2005) suggest that it is possible to achieve clinically significant results if the FES treatment is based on the voluntary initiation of functional movements.

Unsurprisingly, approaches that challenge more long standing treatment approaches, such as the Bobath Concept, will take time to become embedded in the minds and hearts of practitioners. It is important to stress that these novel interventions need to be seen as an adjunct to the therapeutic process rather than one that replaces it. Both patients and practitioners need to be persuaded that the evidence base for their implementation is strong and that it provides an additional dimension to the 'toolbox' of practitioners. These interventions show the potential to free up valuable therapist time and provide a situation where patients can access rehabilitation interventions in order to practise functional movements at their own pace.



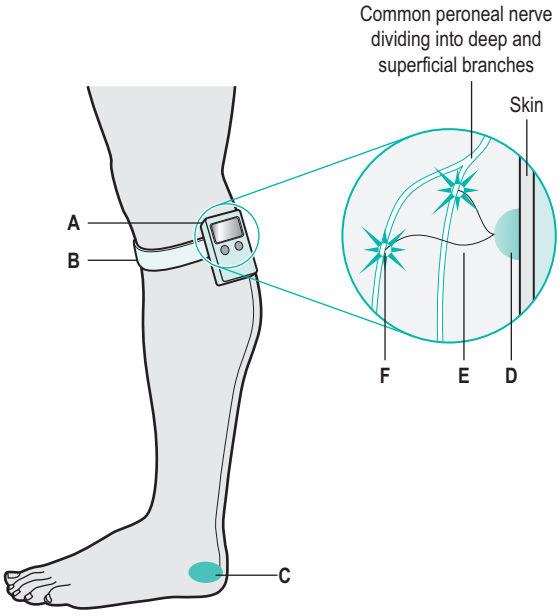


Figure 26.5 (a) Functional electrical stimulation (FES) device to assist with foot clearance; (b) diagram showing nerve distribution; (A) control unit, (B) strap, (C) foot switch under heel, (D) electrode, (E) electrical current, (F) cell body (motor neuron). *Reproduced with permission of the National Clinical FES Centre, Salisbury, UK.*

Patients have expressed the view that they wish to be able to access rehabilitation without the presence of a therapist (McNair et al. 2005). Such opportunities enhance rather than undermine practitioner approaches and should be viewed as an opportunity rather than as a threat.

NEUROLOGICAL CONDITIONS

Stroke

The word *stroke* is used to refer to a clinical syndrome of presumed vascular origin typified by rapidly developing signs of focal or global disturbance of cerebral functions lasting more than 24 hours or leading to death. Stroke is the third most common cause of mortality and the leading cause of long-term disability worldwide (Mackay and Mensah 2004), with over 900,000 people living in England who have had a stroke (DH 2005). It affects between 174 and 216 people per 100,000 population in the UK each year (Mant et al. 2004), and accounts for 11% of all deaths in England and Wales. The risk of recurrent stroke within five years of a first stroke is between 30% and 43% (Mant et al. 2004).

The conditions are traditionally referred to as stroke, transient ischaemic attack and subarachnoid haemorrhage, and are briefly described below.

Transient ischaemic attack (TIA) is a clinical syndrome characterised by an acute loss of focal cerebral or ocular function with symptoms lasting less than 24 hours. It is thought to be a result of inadequate cerebral or ocular blood supply as a result of low blood flow, thrombosis or embolism associated with diseases of the blood vessels, heart or blood (Hankey and Warlow 1994). Transient ischaemic attacks affect 35 people per 100,000 of the population each year and are associated with a very high risk of stroke in the first month of the event and up to one year afterwards (Rothwell et al. 2007). Some people have a mild residual deficit, which persists for days or weeks. The National Clinical Guidelines for Stroke (Intercollegiate Stroke Working Party 2008) state that it is important for people who suffer a TIA to commence a daily dose of 300 mg aspirin immediately. This helps to reduce the risk of a further TIA or stroke.

Subarachnoid haemorrhage (SAH) is a haemorrhage from a cerebral blood vessel, aneurysm or vascular malformation into the subarachnoid space, i.e. the space surrounding the brain where blood vessels lie between the arachnoid and pial layers. SAH affects 6–12 people in each 100,000 of the population per year and constitutes about 6% of incident first strokes. Clinically, the acute presentation is usually different from the presentation of other strokes, specifically because it presents with sudden onset of severe headache and non-focal neurological symptoms, which may include loss of consciousness.

Risk factors

Strokes can occur at any age but are more common in the elderly. There are several risk factors associated with cerebrovascular disease (see Table 26.4).

A stroke has a major impact on a person's life. It can lead to long-term restriction in functional activities and necessitate long-term care. Patients who have suffered a stroke (cerebrovascular accident) present healthcare professionals with a variety of complex physical, psychological and social problems. The sudden loss of any capacity causes severe stress not only to the patient but also to the family. The resultant neurological deficit can have a devastating outcome.

The signs and symptoms associated with a stroke are dependent upon the size and location of the lesion. A stroke usually results in hemiplegia (paralysis of one side of the body). This hemiplegia is contralateral (opposite) to the side of the brain in which the lesion occurs. An interruption of blood flow to the brain leaves the patient with a focal loss of function of varying severity. The most common deficit is a motor deficit. Other neurological deficits can include:

- visual;
- perceptual;
- sensory;
- communication;
- swallowing.

Pathology

Strokes are non-progressive in nature and are caused by ischaemia or haemorrhage. A small number of strokes are caused by congenital abnormalities of the blood vessels and can result in spontaneous intercranial haemorrhage.

Table 26.4 Risk factors for stroke

Major risk factors	Minor risk factors
Hypertension	Contraceptive pill
Raised cholesterol	Excessive alcohol consumption
Atherosclerosis	Physical inactivity
Diabetes mellitus	Obesity
Cardiac disease	
Smoking	

Adapted from Weiner and Goetz (1994).

These defects of the blood vessels are known as arteriovenous malformations (AVMs). They are liable to subsequent bleeding and surgical intervention is the treatment of choice (Stokes 1998; Fawcus 2000).

Ischaemic stroke

Ischaemic stroke occurs when an embolus (a migrating clot) or thrombus (a fixed clot) lodges itself in a blood vessel, obstructing the blood flow to the area distal to the blockage. This causes an abrupt interruption to blood flow and leads rapidly to cell death and focal neurological deficit. The thrombus is usually a result of atherosclerosis and is often associated with hypertension, diabetes mellitus, and coronary or peripheral vascular disease. The symptoms of ischaemia develop over a few minutes.

The area affected by the stroke will depend upon the distribution of the artery and the degree of anastomosis by other cerebral arteries. The addition of oedema at the site of the lesion will add to the focal deficit. Once the oedema has reduced the residual deficit will become apparent.

Haemorrhagic stroke

A haemorrhagic stroke occurs when there is a rupture of a blood vessel into the brain tissue. This can occur as a result of hypertension that causes lipohyalinosis to occur in the small arteries of the brain causing micro-aneurysms to form, which then rupture. The onset is dramatic, with severe headache, vomiting and loss of consciousness.

Subarachnoid haemorrhage

This occurs when there is bleeding into the subarachnoid space following the rupture of a berry aneurysm near the circle of Willis. There is a sudden intense headache associated with vomiting and neck stiffness. Loss of consciousness may occur. Ten per cent of people will die within a few hours and 40% within two weeks (Stokes 1998). Surgical intervention is the best hope of recovery, followed by intensive rehabilitation.

Clinical features

The clinical manifestations of occlusion of the cerebral arteries are shown in Table 26.5.

Further reading

The range and severity of clinical features is extremely varied. The recommended reading at the end of the chapter provides readers with texts that deal with these signs more comprehensively.

Table 26.5 Arteries involved in cerebral vascular occlusion

Artery	Clinical manifestation of occlusion
Middle cerebral artery (supplies most of the convexity of the cerebral hemispheres)	Dense contralateral hemiplegia Contralateral homonymous hemianopia Cortical type of sensory loss Speech problems in left hemisphere lesions, with neglect of contralateral side Lesions in the right hemisphere result in parietal damage, visuospatial disturbances and left-sided neglect
Posterior cerebral artery	Visual disturbance Contralateral homonymous field defect Memory disturbance and contralateral sensory loss
Anterior cerebral artery	Contralateral monoplegia Cortical sensory loss Sometimes behavioural abnormalities associated with frontal lobe damage

Adapted from Stokes (1998).

The clinical features depend on the size and severity of the lesion. Many of the clinical features of damage to the CNS can be manifested (see clinical features section). Generally, there will be a mixture of these features. The list is not exhaustive and it is beyond the scope of this text to cover every possibility. No two patients are alike.

Management

Time

The nature of the problems faced changes over time. The National Clinical Guidelines (Intercollegiate Stroke Working Party 2008) consider:

- prevention;
- acute phase (0–7 days), usually dominated by medical diagnosis and treatments;
- sub-acute or recovery phase (1–26 weeks), usually dominated by rehabilitation;
- long-term (over six months), usually dominated by support and care.

There are three stages of management: medical management, rehabilitation and prevention (Fawcus 2000). The general increase in chronic disease has led to an increased importance of health promotion strategies.

Medical management

Any person who arrives at hospital with an acute onset neurological syndrome with persisting symptoms and signs (i.e. potential stroke) needs full diagnosis to separate out acute cerebrovascular causes from other causes. It is important to establish the type of stroke that has occurred because the medical management will depend on whether it is an ischaemic, haemorrhagic or a subarachnoid stroke. The National Clinical Guidelines for Stroke (Intercollegiate Stroke Working Party 2008) outlines the recommended treatments and management.

The diagnosis of stroke is primarily dependent upon the clinical presentation and confirmed by a brain scan. It is important to diagnose as quickly as possible so immediate actions can be taken to reverse, or at least limit, brain damage. The National Stroke Strategy states: 'Patients requiring urgent brain imaging are scanned in the next scan slot within usual working hours, and within 60 minutes of request out-of-hours with skilled radiological and clinical interpretation being available 24 hours a day.'

Ten to 15% of patients following stroke are suitable for thrombolysis treatment. Thrombolysis means the breaking up of blood clots. Thrombolysis or 'clot-busting' medicine called tissue plasminogen activator (tPA) can dissolve the blood clot following acute ischaemic stroke and significantly improves outcome.

Other medical management usually consists of the treatment of any underlying pathology (e.g. hypertension) and the prevention of secondary complications.

In the UK there are recommendations for the type of service provision stroke sufferers can expect. Research suggests that stroke patients do better when treated in a specialised stroke unit that has a co-ordinated multidisciplinary stroke team (Fawcus 2000; Edwards 2002).

Physiotherapy management

The physiotherapy-specific national guidelines for stroke (RCP and CSP 2008) contain the recommendations from the National Clinical Guidelines that have direct implications for physiotherapists and aim to provide ready access to the latest guidance. They should be viewed as a framework to guide clinical decisions rather than as rigid rules. The guidelines are continually updated in the light of new evidence. Examples of the guidance are given in Boxes 26.1 and 26.2.

Multiple sclerosis

Introduction

Multiple (disseminated) sclerosis is a progressively degenerative disease of the CNS of unknown cause, whose pathological trademarks are inflammation and demyelination. The presenting clinical features can be very varied, resulting in a complex combination of physical, psychological and cognitive problems. This highly variable presentation

Box 26.1 Rehabilitation treatment approach

All members of a stroke service should:

- use an agreed consistent approach for each problem faced by a patient, ensuring the patient is given the same advice and taught the same technique to ameliorate or overcome it;
- give as much opportunity as possible for a patient to practise repeatedly and in different settings any tasks or activities that are affected;
- work within their own knowledge, skills, competence and limits in handling patients, and using equipment, being taught safe and appropriate ways to move and handle specific patients if necessary.

Box 26.2 Rehabilitation treatment quantity (intensity of therapy)

- Patients should undergo as much therapy appropriate to their needs as they are willing and able to tolerate. In the early stages they should receive a minimum of 45 minutes daily of any therapy that is required.
- The team should promote the practice of skills gained in therapy into the patient's daily routine in a consistent manner and patients should be enabled and encouraged to practise that activity as much as possible.
- Therapy assistants may facilitate practice but should work under the guidance of a qualified therapist.

(and often unpredictable condition) poses a major challenge to therapists if they are to assist individuals in managing the condition as effectively as possible.

The prevalence of multiple sclerosis varies worldwide, with the lowest among populations living nearest to the equator. The condition appears to be most common in temperate climates. People who emigrate before the age of 15 years exhibit the rate of incidence of their adopted country (Dean and Kurtzke 1971). These facts seem to suggest that there may be an environmental trigger that allows the disease to develop through a genetic or immunological susceptibility in children below the age of about 15 years that is no longer present in adults.

Approximately 15% of individuals with multiple sclerosis have an affected relative. This risk rises to 1:50 for offspring and 1:20 for siblings of affected persons (Sadovnick et al. 1988).

Another possible contributing factor to the onset of multiple sclerosis could be gender. Multiple sclerosis is more common in women than men, with a ratio of approximately 2:1. Race appears to have an influence, with black and Asian populations having a lower incidence. Other suggested factors are diet and socioeconomic status, with the higher the standard of living the higher the risk of developing multiple sclerosis.

Key point

In the UK, an estimated 80,000 people have been diagnosed with multiple sclerosis. This makes it the second highest cause of neurological disability in young adults. Approximately 1 per 800–1000 of the population is affected, with the average onset of the condition being at 30 years of age (Sadovnick and Ebers 1993). The range of onset is extremely broad, from 10 years to 59 years.

Pathology

Acute stage

Breach of the blood-brain barrier is one of the first significant events to occur at this stage. T-lymphocytes, in particular, are targeted against the vascular wall. They are responsible for the secretion of cytokines, which then recruit other cells including macrophages. The lymphocyte makes a hole in the endothelium and enters the CNS. This results in a perivascular inflammatory lesion that leads to tissue damage, particularly of myelin.

This inflammatory stage can vary in its duration from days to a month. The intensity and duration of the attack will determine the overall extent of the damage. This phase is associated with vasogenic oedema that ultimately resolves over a matter of weeks, with repair of the blood-brain barrier. A residual fibrous scar is then left on the myelin sheath, termed a *plaque* or *sclerosis*.

Later stages

In the early stages of the condition, demyelination is not the main cause of any symptoms but rather the inflammation itself. However, as the condition progresses, repeated onset of the attacks results in more permanent damage. Over a period of time the CNS combats this damage by a number of compensatory mechanisms, but eventually the amount it can compensate for the deficits is superseded by the structural damage. Permanent deficits are the final outcome of this run of events.

Forms of multiple sclerosis and diagnosis

About 45% of sufferers initially present with a *relapsing–remitting* form of the condition (RRMS). An acute flare-up, lasting from a few days to a few weeks, is followed by a period of remission where no symptoms are displayed. The periods of relapse and remission can vary in themselves, with an acute relapse being followed by a period of remission lasting weeks to months.

About 40% of the people who initially presented with RRMS will go on to develop a secondary stage of progression with or without superimposed relapses – known as *secondary progressive multiple sclerosis* (SPMS). The person appears to exhibit a steady deterioration, without any noticeable acute periods to account for this.

The third main form of the condition, known as *primary progressive multiple sclerosis* (PPMS), presents as a steadily deteriorating condition from the onset, with no identifiable relapses or remissions. The rate of deterioration can be fairly rapid in some cases. About 10–15% of cases are in this category.

Two further categories need to be included for completeness: *benign* (10–20% of cases), with no significant disability owing to multiple sclerosis 10 years after onset, and *malignant* (Marburg's disease).

In spite of the increasing reliance on magnetic resonance imaging (MRI) as a diagnostic tool, clinical evaluation continues to be the main method of diagnosing multiple sclerosis. Other investigations used are evoked potentials and, less commonly, analysis of cerebrospinal fluid by way of a lumbar puncture. The most widely used diagnostic criteria are those of Poser et al. (1983).

Clinical features

Any part of the CNS may be affected, so, unsurprisingly, symptoms vary tremendously according to which part of the CNS is affected. Some of the most common clinical features will be described here, but readers may need to refer to other texts for more detailed information (see also www.msif.org).

Vision

The optic nerve, cervical cord, brainstem and cerebellar peduncles are most commonly affected. Frequently at onset the first feature is of optic neuritis of varying severity. Pain is felt behind the affected eye with some element of visual disturbance occurring. In 58% of cases acuity is affected without pain. However, overall more than 90% of cases recover the majority of their visual acuity. Later on, other symptoms affecting eye movement are dysmetria, nystagmus, internuclear ophthalmoplegia (slowing of adduction) and occular flutter. Diplopia might be experienced.

Sensation

Sensory symptoms are common early on in the course of the condition. Numbness, pins and needles, tightness around a limb and other more unusual sensations, such as of water running down a limb, are the most frequently experienced. Later on, joint proprioception is also commonly affected.

Motor function

Motor symptoms are more common than sensory symptoms and are usually more disabling in the long run. Changes in muscle tone, weakness, tremor, poor co-ordination and ataxia are all impairments that often result in difficulties with movement. Neurological conditions where the white matter is affected often result in considerable spasticity. Spasticity, most commonly in the legs, may be present with or without accompanying weakness. Other than cerebellar ataxia, spasticity is frequently the most disabling feature in people with multiple sclerosis. Individuals may report spasms, sometimes at night, which can be painful. Cerebellar ataxia can result in arm movements being un-coordinated (intention tremor), a wide-based gait pattern and speech disturbances. The reader is asked to refer to the 'Clinical features' section for more specific details on each of the above symptoms.

Swallowing

Individuals entering the later stages of multiple sclerosis may experience swallowing difficulties (dysphagia). An under-estimation of the degree of swallowing difficulties may result in bronchopneumonia and can ultimately, therefore, be potentially life-threatening. Swallowing is a highly complex motor skill that requires careful assessment by a speech and language therapist, dietician and radiographer. Videofluoroscopy using a modified barium swallow is an essential investigation if comprehensive assessment of dysphagia is to occur. Assessment of the individual's posture during swallowing should not be overlooked. Ultimately, if severe difficulties persist with oral feeding then other alternatives, such as a gastrostomy, may need to be considered.

Bladder and bowel

Bladder disturbances in multiple sclerosis are very common and often take the form of either detrusor hyperreflexia or detruso-sphincter dyssernygia. Urgency, frequency, nocturnal disturbances and urge incontinence are the various types of bladder presentations. People often report that social trips have to be strategically planned around where the nearest toilet might be. The former type of bladder disturbance rarely occurs without the presence of spasticity in the legs. The second type of bladder disturbance (detruso-sphincter dyssernygia) presents as delay or inability to void, frequency, urgency and an inability to fully empty the bladder. This may result in urinary retention and the susceptibility to urinary tract infections.

Treatment is often successful in the form of muscle relaxants, anticholinergic drugs and self-catheterisation.

Constipation is the main bowel problem and is relatively straightforward to treat.

Key point

One of the most common and usually most disabling symptoms is that of fatigue. Fatigue in its truest sense, for people with multiple sclerosis, is usually described as 'a deterioration in performance with continuing effort' (Barnes 2000).

Pain and fatigue

People with multiple sclerosis tend to be more susceptible to pain syndromes such as trigeminal neuralgia,

myelopathies and musculoskeletal-type pain. The latter is usually as a result of poor posture and poor alignment of joints – sometimes through soft tissue adaptations and over stressed joints.

As the day progresses, any activities involving physical effort are often increasingly difficult to carry out. Fatigue is a significant feature of most people with multiple sclerosis. This is an important consideration when it comes to planning physiotherapy treatment interventions.

Cognition

Cognitive difficulties, such as disturbances in functional memory, reduced information processing speeds and impaired intellectual function, are more common in people with multiple sclerosis than is widely recognised. Cognitive impairments in multiple sclerosis may contribute significantly to any reduction in functional capability. Although these types of impairments are more common in people with longstanding multiple sclerosis, they have also been shown to exist at a time when there are relatively few physical symptoms (Van den Burg et al. 1987).

Management

Medical management

Key point

Medical management can be divided into interventions that may influence the condition directly and those that help to manage the symptoms.

In acute relapses, rest and steroid therapy are the mainstay of medical management. In spite of the widespread use of steroids in multiple sclerosis there remains little consensus in the UK over the most appropriate dose and mode of administration. Steroid therapy is most commonly administered in the form of 1 g intravenous methylprednisolone for 3–5 days with or without a reducing dose of steroids. The most common oral regimen is prednisolone 60 mg daily, reducing to 0 over 1–3 weeks. Low-dose oral steroids are generally better tolerated, but evidence of efficacy is less robust.

Steroids have been shown to shorten the recovery time during relapses, although they do not seem to have an overall effect on the long-term progress of the condition. Longer-term use of steroids is less well supported in the literature and any potential benefits must be weighed against the potentially harmful side effects of steroid therapy.

Interferone β has now unequivocally been proven to be of benefit to some people with multiple sclerosis by reducing the number of relapses that occur in RRMS, thereby delaying progression of the condition.

Many other types of medical intervention are utilised to manage the consequences of the disease process, such as antispasmodic medication to reduce the effects of spasticity and referral for further investigations depending upon the presenting symptoms. For example, an ultrasound scan of the bladder may be needed to establish incomplete voiding of the bladder.

Physiotherapy management

The key to any successful intervention is a comprehensive and ongoing assessment of the person's difficulties and needs. A co-ordinated interdisciplinary approach is also crucial if the condition is to be managed effectively (Ko Ko 1999). Rehabilitation programmes must be tailored to meet the needs of the individual and carers, who should be at the centre of the goal-setting approach.

Individuals with multiple sclerosis often feel their needs are best served by services that allow direct access to them at the time of need. They also often feel they benefit most from a consistent approach on an ongoing basis that allows continuity of management rather than episodes of care (Robinson et al. 1996).

The main aims of any physiotherapy intervention must be to keep the individual as functionally independent as possible. In order to do this, often in the face of potential increasing loss of function, it is vitally important that the physiotherapist works with the person on a psychological, as well as a physical, level. Timing of interventions is crucial and is one of the main reasons for the need for ongoing monitoring of this group of patients. The importance of early contact with patients cannot be over stated. This allows time for an effective rapport to be established which will become invaluable as the disease progresses. Even in the early stages, the more aware the patient is of their condition, the more effective the management is likely to be. Physiotherapists need to be proactive in the area of patient education by consulting with the evidence base in conjunction with an informed knowledge of the condition, to ensure this component of the management programme is as effective as possible. Establishing and encouraging early self-management with the patient will pay dividends in the long run.

Additional goals of a physiotherapy programme are to minimise abnormalities of muscle tone, preserve the integrity of the musculoskeletal system by preventing secondary problems (Thompson 1998), improve posture and give advice on postural management, and to facilitate the use of efficient functional movement patterns, including re-education of gait.

Motor neurone disease

Introduction

Motor neurone disease (MND) is characterised by progressive degeneration of motor neurones:

- anterior horn cells in the spinal cord, resulting in lower motor neurone lesions (LMN);

- corticospinal tract cells, resulting in upper motor neurone lesions (UMN);
- motor nuclei in the brain stem, resulting in both upper and lower motor lesions.

Key point

'Motor neurone disease' is, in fact, a global term mainly used in the UK and Australia. In other parts of the world the condition is often referred to as 'amyotrophic lateral sclerosis' (ALS).

Although MND is primarily a disease of the motor neurones, there may be occasional involvement of other areas of the CNS. The autonomic nervous system, sensory nerves, lower sacral segments of the spinal cord and the three cranial nerves that control movement of the eyes are usually unaffected.

The aetiology of the condition is unknown, although 5% of people have a familial form (Figlewicz and Rouleau 1994). It is a condition that usually affects people in later life, starting at between 50 and 70 years, with a marginally higher occurrence in males. The cause and pathogenesis of the motor neurone degeneration in ALS appears to be a complex and multifactorial process. More recently it has been hypothesised that altered RNA processing may be involved in the disease pathogenesis (Van Damme and Robberecht 2009).

Key point

Precise figures for incidence and prevalence are not known. The incidence is thought to be 2 per 100,000 per year, while the prevalence is estimated to be 7 people per 100,000 of the population. The approximate number of people with MND in the UK is 5000 (MND Association 2000).

Clinical features and diagnosis

The onset of MND is usually insidious and the exact presentation depends upon the areas of the CNS affected. Where there is lower motor neurone degeneration, the main features are weakness, muscle wasting and fasciculation of the nerves undergoing degeneration. Degeneration of upper motor neurones usually results in spasticity with accompanying weakness and muscle wasting.

Forms of MND

The following is a broad categorisation of the forms of MND, but, in reality, there is often considerable overlap between the forms.

Amyotrophic lateral sclerosis (ALS)

This is the most common form of the condition (65%). It is more common in males and involves both upper and lower motor neurones. It is characterised by spasticity, muscle weakness, hyperactive reflexes, with possible bulbar signs of dysarthria, dysphagia and emotional lability. The hands tend to be affected initially with the person reporting clumsiness, with evidence of thenar eminence wasting and the shoulder also often being affected early on. With bulbar involvement, speech, swallowing and the tongue are affected. The average survival rate is 2–5 years.

Progressive bulbar palsy

This affects 25% of people with MND, with slightly older people and women being more commonly affected. Both upper and lower neurones may be involved. Dysphagia and dysarthria are characteristic of this form of the condition owing to lower motor neurone damage causing nasal speech, regurgitation of fluids via the nose, tongue atrophy, pharyngeal weakness and fasciculation. Life expectancy is usually between six months and three years.

Progressive muscular atrophy

This affects approximately 7.5% of people with MND when there is predominantly lower motor neurone involvement. Males are more affected than females at a ratio of muscle wasting and weakness, with weight loss and fasciculation the main presenting features. Mental deterioration and dementia are found in fewer than 5% of people (Tandan 1994). Life expectancy is usually more than five years.

Primary lateral sclerosis

This is a rare form of the condition that affects mainly the upper motor neurones. It is characterised by spastic quadraperisis, pseudobulbar symptoms, spastic dysarthria and hyperreflexia. A survival rate of 20 years or more can be expected.

Diagnosis

As with many other progressive diseases, no one specific test exists for the diagnosis of MND. Investigations often include imaging (MRI), myelogram (electromyography), blood tests and clinical investigations to exclude the possibility of other conditions such as syringomyelia or cervical spondylosis (Swash and Schwartz 1995).

Management

Medical management

Medical and healthcare professionals have a significant role to play in the management of this condition owing to its complex and potentially rapidly deteriorating course. Owing to the high number of professionals involved in the person's care, it is vitally important that interventions

be co-ordinated in some way. As in many cases where a high number of professionals from various agencies are involved, individuals with MND and their families often report that one of the most frustrating aspects of their management is the lack of co-ordination of service provision. In spite of the logic for a MDT approach to the management of people with MND, a recent Cochrane review (Ng et al. 2009) concluded that the evidence for multidisciplinary care in the treatment of people with MND was poor owing to there being an absence of randomised controlled trials. It was highlighted that further research is needed into the most appropriate study designs, outcome measures, care-givers' needs and evaluation of optimal settings for treatment together with more research into the type, intensity and cost-effectiveness of interventions.

The family and their long-term needs must be considered. It is vitally important that the person with MND maintains as much control over his/ her life as possible. Timely interventions, speedy responses, advice and support, ongoing assessment and access to equipment and services are the main needs for this group of people.

Rilutek

In 1996 a drug called Rilutek, whose active ingredient is riluzole, became the first licensed drug treatment available for people with MND. It is not a cure for the condition but has been reported to extend life by several months (NICE 2001).

Physiotherapy management

Assessment of the person's needs on an ongoing basis is taken as a prerequisite before any treatment interventions. Again, a MDT assessment and approach is necessary for this group of people.

In the early stages, information on MND and support agencies, advice on appropriate exercise programmes, support for the person with MND and the family, and providing a point of contact will be the main physiotherapy interventions.

As the condition progresses – and this will vary from person to person – physiotherapy interventions will take the form of advice on exercise programmes and how best to incorporate these into everyday activities. This will ensure that the movement is as functional as possible. Active-assisted exercises may be required if the person needs assistance to move. The main aim at this stage is to keep the person as independent as possible for as long as possible. To this end, assistive devices may be needed and, again, the physiotherapist may need to liaise with the occupational therapist, orthotist and rehabilitation engineers in the case of environmental control systems.

As movement becomes more difficult through weakness or spasticity, passive movements are indicated to prevent secondary impairments of the musculoskeletal system. It is vital that carers be involved in this process, if they so wish, to assist in the process of maintenance. A large part of the physiotherapist's role will be teaching and advising carers and other members of the team. One aspect of advice from the physiotherapist might be how to move and position the client in the most appropriate manner. The client is likely to be increasingly reliant on other forms of mobility during this stage of the condition and, as such, will need advice on the type of wheelchair (often powered) required.

Non-invasive ventilation to aid breathing has been purported to increase the survival rate and increase, or at least maintain, the quality of life of people with MND. However, the evidence base is still fairly limited with only two randomised controlled trials having been carried out to date (Radunovic et al. 2009). This finding was based on individuals with better bulbar function.

With individuals with dysphagia it is important that the physiotherapist works closely with the speech and language therapist and nursing staff to ensure optimum positioning for a safe swallow. Monitoring of respiratory function will also be necessary for this group of clients.

Terminal stage

In the terminal stage of the disease the person may be cared for at home or admitted to a hospice. The main aims of treatment are management of any prevailing symptoms, ensuring the person is as comfortable as possible, providing psychosocial support, advising on potentially life-prolonging treatments and end-of-life care. It is of vital importance that both practical and emotional support is forthcoming for the family and carers. Deterioration is often rapid, with the most common cause of death being respiratory tract infection leading to respiratory failure.

Brain injury

Introduction

Brain injury and head injury tend to be used interchangeably within the literature. An acquired brain injury (ABI) refers to any brain injury caused by the following:

- trauma;
- vascular accident;
- cerebral anoxia, e.g. as a result of cardiac arrest;
- toxic or metabolic conditions, e.g. hypoglycaemia;
- infection, e.g. meningitis.

The brain injury may be direct as a result of physical injury to the brain or indirect if a situation arises where cerebral perfusion pressure reaches a critical low and regulatory mechanisms are lost leading to ischaemic damage (Mendlow et al. 1983). Alternatively, intracranial causes can be the result of damage, such as acute traumatic haematomas, raised intracranial pressure (ICP) and infection.

Table 26.6 Classification of brain injury

Classification	Score on Glasgow Coma Scale (GCS)	Duration of coma	Length of post-traumatic amnesia
Mild	13–15	<20 min	<1 hour
Moderate	9–12	<6 hours	1–24 hours
Severe	3–8	>6 hours	>24 hours

Traumatic brain injury (TBI) describes a head injury resulting from a traumatic event. TBI is a subset of ABI with the other subset being non-traumatic events. In addition to the damage which leads to the TBI there are often associated injuries, such as limb fractures and internal organ damage. Skull fractures may be simple or compound, undisplaced or depressed.

Any brain injury can be classified according to the Glasgow Coma Scale (GCS) duration of coma, by the length of post-traumatic amnesia (PTA) or by results from a computerised tomography (CT) scan (Table 26.6) (Department of Veterans Affairs 2008). PTA is defined as 'the length of time between injury and the restoration of continuous day-to-day memory' (Russell and Smith 1961).

Key point

- Each year an estimated 1 million people attend hospital A&E in the UK following head injury. Many more head injuries go unreported and are not assessed by medical professionals.
- Of these, around 135,000 people are admitted to hospital each year as a consequence of brain injury.
- It is estimated that across the UK there are around 500,000 people (aged 16–74) living with long term disabilities as a result of TBI. The DH document National Service Framework for Long Term Conditions (2005) provides some guidance on how to manage these people.
- Approximately 85% of traumatic brain injuries are classified as minor, 10% as moderate and 5% as severe.
- Men are two to three times more likely to have a TBI than women. This increases to five times more likely in the 15–29 age range.
- Road traffic accidents and sporting injuries are the other most common cause of TBI, but alcohol consumption and recreational drug use may be contributing factors.

Adapted from Headway, The Brain Injury Association (Key feacts and statistics: Traumatic brain injury); www.headway.org.uk.

Pathology

The most common causes of TBI are direct blows to the head, deceleration and rotational forces. *Contrecoup lesions* can occur as a result of falls or direct blows to the head where there may be contusions at the site of impact along with contusions at the opposite side to the impact. Often, the frontal lobes are most prone to damage owing to their proximity to the orbital ridges. Vascular damage usually occurs from subarachnoid bleeding with intracerebral and subdural haematomas appearing at the time of injury.

Potential problems following TBI

Post-traumatic epilepsy

This can be present in up to 40% of cases, depending on the severity of brain injury. 'Anticonvulsants may be prescribed during the first 7 days following TBI for the prevention of early seizures' (RCP and BSRM 2003: 33). Medication more commonly prescribed includes carbamazepine and valproic acid owing to their reduced side effects in comparison with phenobarbital and phenytoin. Many anticonvulsants can produce significant side effects.

Post-traumatic hydrocephalus

This refers to enlargement of the ventricular system and generally is non-obstructive. If hydrocephalus is diagnosed, ventricular shunting is the usual treatment.

Neuroendocrine and autonomic disorders

These types of problem include hypertension, hypothalamic–pituitary disorders and hyperthermia. Hypertension can occur as a new condition in 10–15% of patients but is usually transient and can be managed effectively with beta-blockers.

Cranial neuropathies

These are a common occurrence following TBI, with cranial nerves I and III and, less commonly, IV, VI and VIII being affected.

Gastrointestinal and nutritional needs

Nutritional needs in the acute stages of TBI are reported to increase by an estimated 25%. Long-term outcomes

have been more favourable owing to the nutritional needs of patients being met (Young et al. 1987). Alternative methods of feeding may need to be considered in the early stages to achieve this aim. Problems with dysphagia occur in approximately 25% of patients and this might also be another reason why alternative methods of feeding may need to be adopted.

Orthopaedic and musculoskeletal complications

Fractures are a common feature in TBI, with occult fractures (where there is no evidence of fracture on initial X-ray) being amongst the most serious problems. Peripheral nerve injuries are frequently under-diagnosed and heterotopic ossification occurs in 76% of cases.

Continence

Urinary incontinence following TBI is very common for a number of reasons but is primarily a result of disinhibition.

Sexual dysfunction

The most common occurrence is oligomenorrhea, i.e. infrequent or light menstruation. Other complications include impotence, altered libido along with difficulties created by any behavioural changes.

Motor function

Disturbances in the CNS can lead to hypertonicity, contractures and disordered movement. Hypertonicity can predispose patients to adaptive muscle shortening (contractures). Timely and effective management of hypertonicity can ultimately avoid the need for surgical intervention. Movement disorders encountered might be rigidity, tremors, ataxia, akathisia (feeling of inner restlessness and the patient feels unable to sit still) and dystonias, including chorea.

Sensation

Disturbances in sensory function can present in the form of diminished or absent cutaneous sensation (paraesthesia/anaesthesia), or various agnosias such as sensory neglect. Certain sensory disturbances can be addressed to some extent within the rehabilitation programme. However, long-standing sensory disturbances tend to persist.

Cognition and behavioural impairments

Altered cognition can be the most troublesome aspect of a patient's rehabilitation and long-term social reintegration. Commonly occurring features include disturbances in level of arousal, speed of processing of information, memory, abstract reasoning and flexibility, self-awareness, distractability and limited attention span. Behavioural deficits and psychosocial adjustment after TBI include depression, poor social awareness, agitation, aggressive behaviour and difficulties in initiating activities (Wood 1990; Prigatano 1992).

Management

Medical management

The NICE Clinical Guidelines CG56 published in September 2007 provide information on triage, assessment, investigation and early management of head injury in infants, children and adults. Assessment by a trained healthcare professional in determining the nature and severity of the head injury must be done. A CT scan, skull X-ray and continual GCS monitoring all need to be carried out. In some cases, intracranial monitoring will be necessary to ensure no further damage or deterioration in the patient's condition occurs. Certain patients will require neurosurgical intervention, which cannot be covered in any detail within this text (reference to Black and Rossitch (1995) is suggested for further information). Additionally, any other injuries, such as fractures, contusions and haematomas, should be dealt with.

In summary, the overall goals in the acute setting are:

- gaining medical stability;
- clearing of post-traumatic amnesia;
- reduction of behavioural and physical dependence.

If the patient does not require a high level of medical input, a less medically acute setting with intensive therapy services may be more appropriate.

Physiotherapy management

Early stages

Within the intensive care environment the role of the physiotherapist is to maintain respiratory function (see Chapter 7) and reduce soft tissue complications owing to prolonged bed positioning. All interventions during this stage need to be carefully balanced against the risk of raising the intracranial pressure (Ada et al. 1990).

As with many complex neurological conditions, patients are best managed by an experienced interdisciplinary rehabilitation team using a patient-centred, goal-orientated approach. Patients should be in an environment that is conducive to intensive rehabilitation. It is increasingly common for patients with complex disabilities to be managed using a key worker or case manager approach in order to provide continuity of the services delivered more effectively.

Patients can present with a wide variety of impairments, depending on the site of the damage. Patients with severe physical impairments early on in their rehabilitation will benefit from intensive rehabilitation (Hellweg and Johannes 2008). However, by far an over-riding residual problem is one of cognitive impairment, including disruption of executive functions. These types of impairments and the resultant psychosocial implications can lead to significant limitations in social interactions and can be the

main barriers to individuals returning to independent living (Oddy et al. 1978; Oddy and Humphrey 1980; Brooks and McKinlay 1983; Lezak 1986).

The effectiveness of physiotherapy rehabilitation is currently under review (Teasell et al. 2007; Hellweg and Johannes 2008; Holmberg and Lindmark 2008). It is now recognised that intensive early rehabilitation leads to better functional outcomes but the exact nature of these interventions is unspecified. Only task-specific rehabilitation shows strong evidence for improving outcomes and is recommended from the literature. Most of the research surrounding the rehabilitation of ABI and TBI consists of single case studies and low-powered randomised controlled trials. Even though these studies show some success of individual interventions, the lack of quality means that more research is needed to clearly identify what physiotherapy interventions work best.

Later stage – after months or years

The cognitive abilities and behavioural presentation of individuals with TBI will have an impact upon their level of function. It is vital, therefore, that these elements be assessed and taken into account when agreeing goals for interventions. Historically, physiotherapists have failed to take sufficient account of these factors, and subsequent physiotherapy interventions have, perhaps, been less effective than they might have been. Access to a neuropsychologist or cognitive occupational therapist, who can assess and advise on the most effective way to deal with these factors when planning treatment, is invaluable.

Glossary

Agnosia Loss of knowledge or inability to perceive objects

Anaesthesia Absence of sensation

Ataxia Loss of co-ordination affecting functional movement

Babinski sign Abnormal response of the plantar reflex (great toe turns upwards on testing)

Bradykinesia Slowness of movement

Clonus Succession of intermittent muscular relaxation and contraction usually resulting from a sustained stretch

Diplopia Double vision

Dysarthria In-coordination of speech

Dysphagia Difficulty in swallowing

Dysphasia Disruption of expressive (produce) and/or receptive (understand) speech

Dyspraxia Inability to execute volitional purposeful movements

Dystonia Involuntary movement characterised by twisting and repetitive movement

Flaccidity Absence of muscle tone

Hemianopea Loss of visual field in one half of each eye

Hemiplegia Paralysis of one side of the body

Hypertonicity Increased muscle tone

Hypotonicity Decreased muscle tone

Nystagmus Involuntary rhythmic oscillation of one or both eyes

Paraesthesia Disruption of sensation causing abnormal sensation

Ptosis Drooped eyelid

Rigidity Stiffness of neurological origin, increased resistance to stretch throughout the range

Tone The active resistance of muscle to stretch

Tremor Fine type of involuntary movement (several types seen in neurological dysfunction)

ACKNOWLEDGEMENTS

The authors would like to acknowledge Professor Bippin Bhakta and Sophie Makower (Charterhouse Rehabilitation Technologies Laboratory, University of Leeds), Paul Taylor (National Clinical FES Centre, Salisbury), Irina Sanders, and Professor David Roberts (Centre for Virtual Environment, University of Salford) for kindly providing photographs for the rehabilitation technology section in this chapter.

FURTHER READING

General

Brincat, C.A., 1999. Managed care and rehabilitation: Adopting a true team approach. Top Stroke Rehabil 6 (2), 62–65.

Carr, J.H., Shepherd, R.B., 1998. Neurological Rehabilitation: Optimizing Motor Performance. Butterworth-Heinemann, London.

Edwards, S., 2002. Neurological Physiotherapy: A Problem-Solving Approach, second ed. Churchill Livingstone, London.

Greenwood, R., Barnes, M.P., Mcmillan, T.M., et al. (Eds.), 1993. Neurological Rehabilitation. Churchill Livingstone, Edinburgh.

Partridge, C. (Ed.), 2002. Neurological Physiotherapy: Bases of Evidence for Practice. Whurr, London.

Reinkensmeyer, D., Lum, P.S., Winters, J., et al., 2002. Emerging technologies for improving access

to movement therapy following neurological injury. In: Emerging and Accessible Telecommunications: Information and Healthcare Technologies. IEEE Press, New York.

Stokes, M., 1998. Neurological Physiotherapy. Mosby International, New York.

Stroke

Alexander, H., Bugge, C., Hagen, S., 2001. What is the association between the different components of stroke rehabilitation and health outcomes? Clin Rehabil 15, 207–215.

Ashburn, A., 1997. Physical recovery following stroke. Physiotherapy 83, 480–490.

Bosworth, H., Horner, R., Edwards, L., et al., 2000. Depression and other determinants of values placed on current health state by stroke patients: evidence from VAS Acute Stroke (VAS+) study. Stroke 31, 2603–2609.

RCP (Royal College of Physicians) Intercollegiate Stroke Working Party, 2000. National Clinical Guidelines for Stroke. RCP, London.

RCP (Royal College of Physicians) Intercollegiate Stroke Working Party, 2012. National Clinical Guidelines for Stroke, fourth ed. RCP, London.

Head injury

Campbell, M., 2000. Rehabilitation for Traumatic Brain Injury: Physical Therapy Practice in Context. Churchill Livingstone, London.

REFERENCES

Ada, L., Canning, C., Paratz, J., 1990. Care of the unconscious head-injured patient. In: Ada, L., Canning, C. (Eds.), Key Issues in Neurological Physiotherapy. Butterworth-Heinemann, London, p. 249.

Ada, L., Dean, C.M., Morris, M.E., et al., 2010. Randomized trial of treadmill walking with body weight support to establish walking in subacute stroke: the MOBILISE trial. Stroke 41 (6), 1237–1242.

Adamovich, S.V., Fluet, G.G., Tunik, E., et al., 2009. Sensorimotor training in virtual reality: A review. NeuroRehabilitation 25 (1), 29.

Barnes, D., 2000. Multiple Sclerosis: Questions and Answers. Merit Publishing International, Hampshire.

Berg, K., Wood-Dauphinee, S., Williams, J.I., et al., 1989. Measuring balance in the elderly: preliminary development of an instrument. Physiother Canada 41 (6), 304–331.

Black, P., Rossitch, E., 1995. Neurosurgery: An Introductory Text. Oxford University Press, Oxford.

Bohannon, R.W., Smith, M.B., 1987. Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther 67, 206–207.

Bohannon, R.W., Cassidy, D., Walsh, S., 1995. Trunk muscle strength is impaired multidirectionally after stroke. Clin Rehabil 9, 47–51.

Brooks, D.N., McKinlay, W.W., 1983. Personality and behavioural change after severe blunt head injury: A relative's view. J Neurol Neurosurg Psychiat 46, 336–344.

Bruce, H., Dobkin, M.D., 2004. Activity-dependent learning contributes to motor recovery. Annals of Neurology 44 (2), 158–160.

Butefisch, C., Hummelsheim, H., Mauritz, K.H., 1995. Repetitive training of isolated movements improves the outcome of motor rehabilitation of the centrally paretic hand. J Neurol Sci 130, 59–68.

Carr, J.H., Shepherd, R.B., Nordholm, L., et al., 1985. Investigation of a new motor assessment scale for stroke patients. Phys Ther 65, 175–180.

Carr, J., Shepherd, R., 1987. A Motor Relearning Programme for Stroke, second ed. William Heinemann, London.

Cella, D.F., Dineen, K., Arnason, B., et al., 1996. Validation of the functioning assessment of multiple sclerosis quality of life instrument. Neurology 47, 129–139.

Collen, F.M., Wade, D.T., Robb, G.F., et al., 1991. The Rivermead Mobility Index: A further development of the Rivermead Motor Assessment. Int Disabil Studies 1991; 13 (2), 50–54.

Crow, J.L., Lincoln, N.B., Nouri, F.M., et al., 1989. The effectiveness of EMG feedback in the treatment of arm function after stroke. Int Disabil Studies 11, 155–160.

Dean, G., Kurtzke, J.F., 1971. On the risk of multiple sclerosis according to the age at immigration to South Africa. BMJ 3, 725–729.

Department of Veterans Affairs, 2008. Schedule for Rating Disabilities. Evaluation of Residuals of Traumatic Brain Injury (TBI). US Government Printing Office, Washington. Volume 73, No. 185.

DH (Department of Health), 2005. National Service Framework for Long Term Conditions; http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/dh_4105369.pdf, accessed, October 2012.

DH (Department of Health), 2005. Reducing Brain Damage: Faster access to better stroke care. National Audit Office, London.

Duncan, P.W., Weiner, D.K., Chandler, J., et al., 1990. Functional reach: A new clinical measure of balance. J Gerontol 45, 192–197.

EBRSR (Evidence-Based Review of Stroke Rehabilitation, 2009. Upper Extremity Interventions. EBRSR, Ontario, Canada; http://www.ebrsr.com/reviews_details.php?31, accessed October 2012.

Edwards, S. (Ed.), 2002. Neurological Physiotherapy: A Problem-Solving Approach, second ed. Churchill Livingstone, London.

Elsworth, J.D., Marks, J.A., McGrath, J.R., et al., 1999. An audit of goal planning in rehabilitation. Top Stroke Rehabil 6 (2), 51–61.

Fawcus, R. (Ed.), 2000. Stroke Rehabilitation: A Collaborative Approach. Blackwell Scientific, Oxford.

Figlewicz, D.A., Rouleau, G.A., 1994. Familial disease. In: Williams, A.C. (Ed.), Motor Neurone Disease. Chapman & Hall, London, pp. 427–450.

Finch, E., Brooks, D., Stratford, P.W., et al., 2002. Physical Rehabilitations Outcome Measures. A Guide to Enhanced Clinical Decision-Making, second ed. Canadian Physiotherapy Association, Toronto.

Freeman, J.A., 2002. Assessment, outcome measurement and goal setting in physiotherapy practice. In: Edwards, S. (Ed.), Neurological Physiotherapy, second ed. Churchill Livingstone, London, pp. 21–34.

French, B., Thomas, L.H., Leathley, M.J., et al., 2007. Repetitive task training for improving functional ability after stroke. Cochrane Database Syst Rev (4), CD006073.

Feys, H.M., DeWeerdt, W.J., Selz, B.E., et al., 1998. Effect of a therapeutic intervention for the hemiplegic upper limb in the acute phase after stroke: A single-blind, randomized, controlled multicenter trial. Stroke 29 (4), 785–792.

Granger, C.V., Cotter, A.C., Hamilton, B.B., et al., 1993. Functional assessment scales: A study of persons after stroke. Arch Phys Med Rehabil 74, 133–138.

Hall, K.M., Cope, D.N., 1995. The benefit of rehabilitation in traumatic brain injury: A literature review. J Head Trauma Rehabil 10 (5), 1–13.

Hankey, G.J., Warlow, C.P., 1994. Transient Ischaemic Attacks of the Brain and Eye. WB Saunders–Baillière Tindall, London.

Harwood, R.H., Gompertz, P., Pound, P., et al., 1997. Determinants of handicap 1 and 3 years after a stroke. Disabil Rehabil 19 (5), 205–211.

Headway, The Brain Injury Association, 2009. Key feacts and statistics: Traumatic brain injury; https://www.headway.org.uk/key-facts-and-statistics.aspx.

Hellweg, S., Johannes, S., 2008. Physiotherapy after traumatic brain injury: A systematic review of the literature. Brain Injury 22 (5), 365–373.

Henderson, A., Korner-Bitensky, N., Levin, M., 2007. Virtual reality in stroke rehabilitation: A systematic review of its effectiveness for upper limb motor recovery. Top Stroke Rehabil 14, 52–61.

Holmberg, T.S., Lindmark, B., 2008. How do physiotherapists treat patients with traumatic brain injury? Advances in Physiotherapy 10 (3), 138–145.

Hunt, S.M., McKenna, S.P., Williams, J., 1981. Reliability of a population survey tool for measuring perceived health problems: A study of patients with osteoarthritis. J Epidemiol Commun Health 35, 297–300.

Intercollegiate Stroke Working Party, 2008. National Clinical Guideline for Stroke, third ed. Royal College of Physicians, London.

Jackson, A.E., Holt, R.J., Culmer, P.R., et al., 2007. Dual robot system for upper limb rehabilitation after stroke: The design process. Proceedings Institute of Mechanical Engineers Part C 221 (7), 845–857.

Ko Ko, C., 1999. Effectiveness of rehabilitation for multiple sclerosis. Clin Rehabil 13 (Suppl. 1), 33–41.

Lance, J.W., 1980. Symposium synopsis. In: Feldman, R.G., Young, R.R., Koella, W.P., (Eds.), Spasticity: Disordered control. Yearbook Medical, Chicago, pp. 485–494.

Lezak, M.D., 1986. Psychological implications of traumatic brain damage for the patient's family. Rehabil Psychol 31, 241–250.

Lincoln, N.B., Jackson, J.M., Adams, S.A., 1998. Reliability and revision of the Nottingham Sensory Assessment for stroke patients. Physiotherapy 84, 358–365.

Lincoln, N.B., Leadbitter, D., 1979. Assessment of motor function in stroke patients. Physiotherapy 65, 48–51.

Mackay, J., Mensah, G., 2004. The Atlas of Heart Disease and Stroke. WHO, Geneva.

McNair, B., Islam, N., Eccleston, C., et al., 2005. EPSRC Smart Rehabilitation: Technological applications for use in the home by people who have had a stroke and their primary carers. Report for EQUAL project SMART rehabilitation engineering team; http://research.shu.ac.uk/lab4living/smart-rehabilitation, accessed October 2012.

Mahoney, F.I., Barthel, D.W., 1965. Functional evaluation: The Barthel Index. Maryland State Med J 14, 61–65.

Mann, G.E., Burridge, J.H., Malone, L.J., et al., 2005. A pilot study to investigate the effects of electrical stimulation on recovery of hand function and sensation in subacute stroke patients. Neuromodulation 8 (3), 193–202.

Mant, J., Wade, D., Winner, S., 2004. Health care needs assessment: stroke. In: Stevens, A., et al., (Eds.), 2004, Health Care Needs Assessment: The Epidemiologically Based Needs Assessment Reviews, second ed. Radcliffe Medical Press, Oxford.

Mathiowetz, V., Weber, K., Kashman, N., et al., 1985. Adult norms for the 9-hole peg test of finger dexterity. Occup Ther J Res 5, 24–37.

McGrath, J.R., Adams, L., 1999. Patient-centered goal planning: A systematic psychological therapy? Top Stroke Rehabil 6 (2), 43–50.

Mellerup, E., Fog, T., Raun, N., et al., 1981. The socio-economic scale. Acta Neurolog Scand 64, 130–138.

Mendlow, A.D., Teasdale, G.M., Teasdale, E., et al., 1983. Cerebral blood flow and intracranial pressure in head injured patients. In: Ishii, S., Nagai, H., Brock, M. (Eds.), Intracranial Pressure. Springer-Verlag, Berlin, pp. 495–500.

MND Association (Motor Neurone Disease Associaton), 2000. A Patient and Carer Centred Approach for Health and Social Professionals: Motor Neurone Disease Resource File. MND Association, Northampton.

MRC (Medical Research Council), 1976. Aids to the Examination of the Peripheral Nervous System. Her Majesty's Stationery Office, London.

Ng, L., Khan, F., Mathers, S., 2009. Multidisciplinary care for adults with amyotrophic lateral sclerosis or motor neuron disease. Cochrane Database of Systematic Reviews 2009 (4).

NICE (National Institute for Health and Clinical Excellence), 2001. Technology Appraisal: Guidance on the Use of Riluzole for the Treatment of Motor Neurone Disease. Appraisal no. 20. NICE, London.

Norkin, C.C., White, D.J., 1975. Measurement of Joint Motion: A Guide to Goniometry. FA Davies, Philadelphia.

Nuyens, G.E., De Weerdt, W.J., Spaepen, A.J. Jr, et al., 2002. Reduction of spastic hypertonia during repeated passive knee movements in stroke patients. Arch Phys Med Rehabil 83 (7), 930–935.

Oddy, M., Humphrey, M., 1980. Social recovery during the year following severe head injury. J Neurol Neurosurg Psychiat 43, 798–802.

Oddy, M., Humphrey, M., Uttley, D., 1978. Subjective impairment and social recovery after closed head injury. J Neurol Neurosurg Psychiat 41, 611–616.

Page, S.J., 2003. Intensity versus task-specificity after stroke: how important is intensity? Am J Phys Med Rehabil 82 (9), 730–732.

Petajan, J.H., Gappmaier, E., White, A.T., et al., 1996. Impact of aerobic training on fitness and quality of life in multiple sclerosis. Ann Neurol 39, 432–441.

Peto, V., Jenkinson, C., Fitzpatrick, R., et al., 1995. The development and validation of a short measure of functioning and wellbeing for individuals with Parkinson's disease. Qual Life Res 4, 241–248.

Podsialo, D., Richardson, S., 1991. The timed 'up and go': A test of basic functional mobility for frail elderly persons. J Am Geriatr Soc 39, 142–148.

Popovic, M.R., Thrasher, T.A., Zivanovic, V., et al., 2005. Neuroprosthesis for retraining reaching and grasping functions in severe hemiplegic patients. Neuromodulation 8 (1), 58–72; http://www.toronto-fes.ca/publications/Popovic_Thrasher_05.pdf, accessed, October 2012.

Poser, C.M., Paty, D.W., Scheinberg, L.C., et al., 1983. New diagnostic criteria for multiple sclerosis: Guidelines for research protocols. Ann Neurol 13, 227–231.

Prange, G.B., Jannink, M., Groothuis-Oudshoorn, C., et al., 2006. Systematic review of the effect of robot-aided therapy on recovery of the hemiparetic arm after stroke. JRRD 43 (2), 171–184; http://www.isa.umh.es/vr2/euron08/doc/Jane%20Burridge/Prange%20JRRD%202006.pdf, accessed October 2012.

Prigatano, G.P., 1992. Personality disturbances associated with traumatic brain injury. J Consult Clin Psychol 60, 360–368.

Radunovic, A., Annane, D., Jewitt, K., Mustfa, N., 2009. Mechanical ventilation for amyotrophic lateral sclerosis/motor neuron disease. Cochrane Database Syst Rev 7 (4).

RCP (Royal College of Physicians) Intercollegiate Stroke Working Party, 2004. National Clinical Guidelines for Stroke, second ed. RCP, London; http://www.sorcan.ca/Resources/General/NCG.pdf, accessed October 2012.

RCP (Royal College of Physicians), BSRM (British Society of Rehabilitation Medicine), 2003. Rehabilitation Following Acquired Brain Injury: National Clinical Guidelines. RCP, BSRM, London; http://bookshop.rcplondon.ac.uk/contents/43986815-4109-4d28-8ce5-ad647dbdbd38.pdf, accessed October 2012.

RCP (Royal College of Physicians), CSP (Chartered Society of Physiotherapy) Intercollegiate Stroke Working Party, 2008. Physiotherapy Concise Guide for Stroke. 2008; http://bookshop.rcplondon.ac.uk/contents/3756b29c-1001-4db1-97a6-dcd220970fd6.pdf, accessed October 2012.

Rizzo, A., Kim, G.J., 2005. A SWOT analysis of the field of virtual reality rehabilitation and therapy. Presence 14 (2), 119–146; http://www.usc.edu/projects/rehab/private/docs/advisors/rizzo/8_rizzo_a_swot_analysis.pdf, accessed October 2012.

Romberg, A., Virtanen, A., Ruutiainen, J., et al., 2004. Effects of a 6-month exercise program on patients with multiple sclerosis. A randomized study. Neurology 63, 2034–2038; http://www.direct-ms.org/pdf/GeneralInfoMS/ExerciseMSStudy.pdf, accessed October 2012.

Rothwell, P.M., Giles, M.F., Chandratheva, A., et al., 2007. Effect of urgent treatment of transient ischaemic attack and minor stroke on early recurrent stroke (EXPRESS study). Lancet 370 (9596), 1432–1442.

Robinson, I., Hunter, M., Neilson, S., et al., 1996. A Dispatch from the Front Line: The Views of People with Multiple Sclerosis about their Needs. A Qualitative Approach. Brunel MS Research Unit, London.

Russell, W.R., Smith, A., 1961. Post-traumatic amnesia in closed head injury. Arch Neurol 5, 4–17.

Sadovnick, A.D., Baird, P.A., Ward, R.H., 1988. Multiple sclerosis: Updated risks for relatives. Am J Med Genet 29, 533–541.

Sadovnick, A.D., Ebers, G.C., 1993. Epidemiology of multiple sclerosis: A critical review. Can J Neurol Sci 20, 17–29.

Saunders, D.H., Greig, C.A., Young, A., et al., 2009. Physical fitness training for stroke patients. An updated review. Cochrane Database of Systematic Reviews, e160–161; http://stroke.ahajournals.org/content/41/3/e160, accessed October 2012.

Sheffler, L.R., Chae, J., 2007. Neuromuscular electrical stimulation in neurorehabilitation. Muscle Nerve 35 (5), 562–590.

Shumway-Cook, A., Woollacott, M.H., 2007. Motor Control: Translating Research into Clinical Practice. Lippincott Williams and Willkins, Philadelphia, USA.

Stokes, M., 1998. Neurological Physiotherapy. Mosby International, New York.

Swash, M., Schwartz, M.S., 1995. Motor neurone disease: the clinical syndrome. In: Leigh, P.N., Swash, M. (Eds.), Motor Neurone Disease: Biology and Management. Springer-Verlag, London, pp. 1–17.

Tandan, R., 1994. Clinical features and differential diagnosis of classical motor neurone disease. In: Williams, A.C. (Ed.), Motor Neurone Disease. Chapman & Hall, London, pp. 3–27.

Teasell, R., Bayona, N., Marshall, S., et al., 2007. A systematic review of the rehabilitation of moderate to

severe acquired brain injuries. Brain Injury 21 (2), 107–112.

Thompson, A., 1998. Symptomatic treatment in multiple sclerosis. Curr Opin Neurol 11, 305–309.

Tyson, S.F., DeSouza, L.H., 2004. Development of the Brunel Balance Assessment: A new measure of balance disability post stroke. Clin Rehabil 18, 801–810.

Tyson, S., Watson, A., Moss, S., et al., 2008. Development of a framework for the evidence-based choice of outcome measures in neurological physiotherapy. Disabil Rehabil 30 (2), 142–149.

Van Damme, P., Robberecht, W., 2009. Recent advances in motor neuron disease. Curr Opin Neurol 22 (5), 486–492.

Van den Burg, W., Van Zomeren, A.H., Minderhoud, J.M., et al., 1987. Cognitive impairment in patients with multiple sclerosis and mild physical disability. Arch Neurol 44, 494–501.

Van Peppen, R.P., Kwakkel, G., Wood-Dauphinee, S., et al., 2004. The impact of physical therapy on functional outcomes after stroke: What's the evidence? Clin Rehabil 18 (8), 833–862.

Wade, D.T., 1992. Measurement in Neurological Rehabilitation. Oxford University Press, Oxford.

Wade, D.T., 1999a. Goal planning in stroke rehabilitation: how? Top Stroke Rehabil 6 (2), 16–36.

Wade, D.T., 1999b. Goal planning in stroke rehabilitation: what? Top Stroke Rehabil 6 (2), 8–15.

Wade, D.T., 1999c. Goal planning in stroke rehabilitation: Evidence. Top Stroke Rehabil 6 (2), 37–42.

Ware, J.E., Snow, K.K., Kosinski, M., et al., 1993. SF-36 Health Survey: Manual and Interpretation Guide. Health Institute, New England Medical Center, Boston, MA.

Weiner, W.J., Goetz, C.G., 1994. Neurology for the Non-neurologist, third ed. Lippincott, Philadelphia.

Weiss, A., Suzuki, T., Bean, J., et al., 2000. High intensity strength training improves strength and functional performace after stroke. Am J Phys Med Rehabil 79 (4), 369–376.

Wood, R.L.I., 1990. Neurobehavioural paradigm for brain injury rehabilitation. In: Wood, R.L. (Ed.), Neurobehavioural sequelae of traumatic brain injury. Taylor & Francis, New York, pp. 3–17.

Wolf, S.L., Winstein, C.J., Miller, J.P., et al., 2008. Retention of upper limb function in stroke survivors who have received constraint-induced movement therapy: The EXCITE randomised trial. Lancet Neurol 7 (1), 33–40.

Young, B., Ott, L., Twyman, D., et al., 1987. The effect of nutritional support on outcome from severe head injury. J Neurosurg 67, 668–676.

Chapter 27

Physiotherapy in women's health

Gill Brook, Tamsin Brooks, Yvonne Coldron, Ruth Hawkes, Judith Lee, Melanie Lewis, Jacquelyne Todd, Kathleen Vits and Liz Whitney

INTRODUCTION

This chapter offers an overview of the role of the physiotherapist in women's health. Some of the expert contributors are members of the Association of Chartered Physiotherapists in Women's Health (ACPWH), a professional network group of the Chartered Society of Physiotherapy (CSP). Originally known as the Obstetric Physiotherapists Association, it was formed in 1948 and is one of the oldest such organisations.

Weblink

Association of Chartered Physiotherapists in Women's Health (ACPWH): http://acpwh.csp.org.uk/

The ACPWH forms a representative body that can be consulted, and will act in the professional interest of physiotherapists working in women's health and in the specific field of continence (including men). It encourages and provides means by which physiotherapists may improve specialist therapeutic skills and understanding of the speciality; it also publishes a journal and hosts an annual conference. In order to nurture interest in this field among student physiotherapists there is a student award, which provides funding to attend the annual conference.

There is a lack of evidence for some aspects of women's health physiotherapy and, to encourage research, support may be offered in the form of a small bursary. There is, in addition, a research officer who works closely with the CSP and other relevant funding bodies to set the future agenda for research and the ACPWH produces a comprehensive selection of leaflets for the public and health professionals.

The women's health physiotherapist is a vital member of the multiprofessional team and ACPWH maintains strong ties with the Royal College of Midwives, the Association for Continence Advice and the Bladder and Bowel Foundation. It communicates with other organisations with a similar interest (e.g. the charities Wellbeing of Women and the National Childbirth Trust).

Since 1999, the ACPWH has been a member of the International Organization of Physical Therapists in Women's Health (IOPTWH), an official subgroup of the World Confederation for Physical Therapy (WCPT) whose mission is to improve healthcare for women internationally through facilitation and promotion of best practice women's health physical therapy. In 2012, its membership was 23 countries.

Weblink

International Organization of Physical Therapists in Women's Health (IOPTWH): www.ioptwh.org

ANATOMY AND PHYSIOLOGY

A physiotherapist needs knowledge of the pelvic anatomy and the physiological changes relevant to women's health.

Bones and joints of the pelvis

The pelvic girdle protects and supports the pelvic contents, provides muscle attachment and facilitates the transfer of weight from trunk to legs in standing, and to the ischial tuberosities in sitting. The joints (Figure 27.1) are supported by some of the strongest ligaments in the body, which may become more lax in pregnancy leading to

increased joint mobility and less efficient load transfer through the pelvis.

The pelvic outlet at the base of the pelvis is narrower in transverse diameter when compared with the pelvic inlet; it comprises the pubic arch, ischial spines, sacrotuberous ligaments and coccyx (Haslam 2004a).

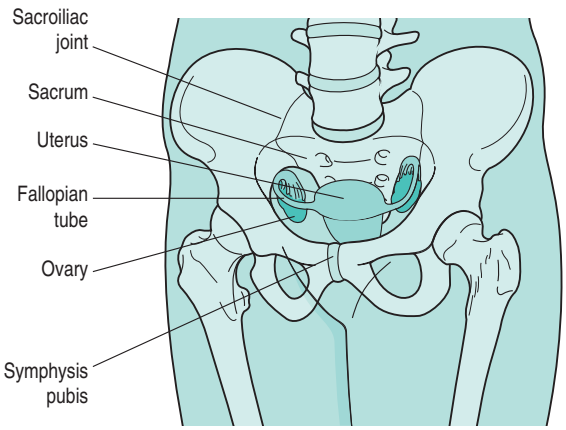


Figure 27.1 True and false pelvis: female reproductive organs.

Muscles

Four pairs of abdominal muscles combine to form the anterior and lateral abdominal wall, and may be termed the abdominal corset. Transversus abdominis (TrA), whose fibres run transversely, lies deep to the internal abdominal oblique (IO) and external abdominal oblique (EO), with the rectus abdominis (RA) central, anterior and superficial (Figure 27.2). IO, EO and TrA insert into an aponeurosis joining in the midline at the linea alba.

The deep abdominal muscles, together with the pelvic floor muscles, multifidus and diaphragm, can be considered as a complete unit and may be termed the lumbopelvic cylinder. This provides support for the abdominal contents and maintains intra-abdominal pressure (Cresswell et al. 1992). At low level muscle activity force is exerted on the thoracolumbar fascia and studies suggest that this contributes to lumbopelvic stability, crucial to pain-free resting posture and normal function (Richardson et al. 1999). Motor control of the muscles of the lumbopelvic cylinder is also significant in maintaining continence and controlling respiration.

Optimal function of these muscles depends on timing of recruitment, endurance, strength and co-ordination.

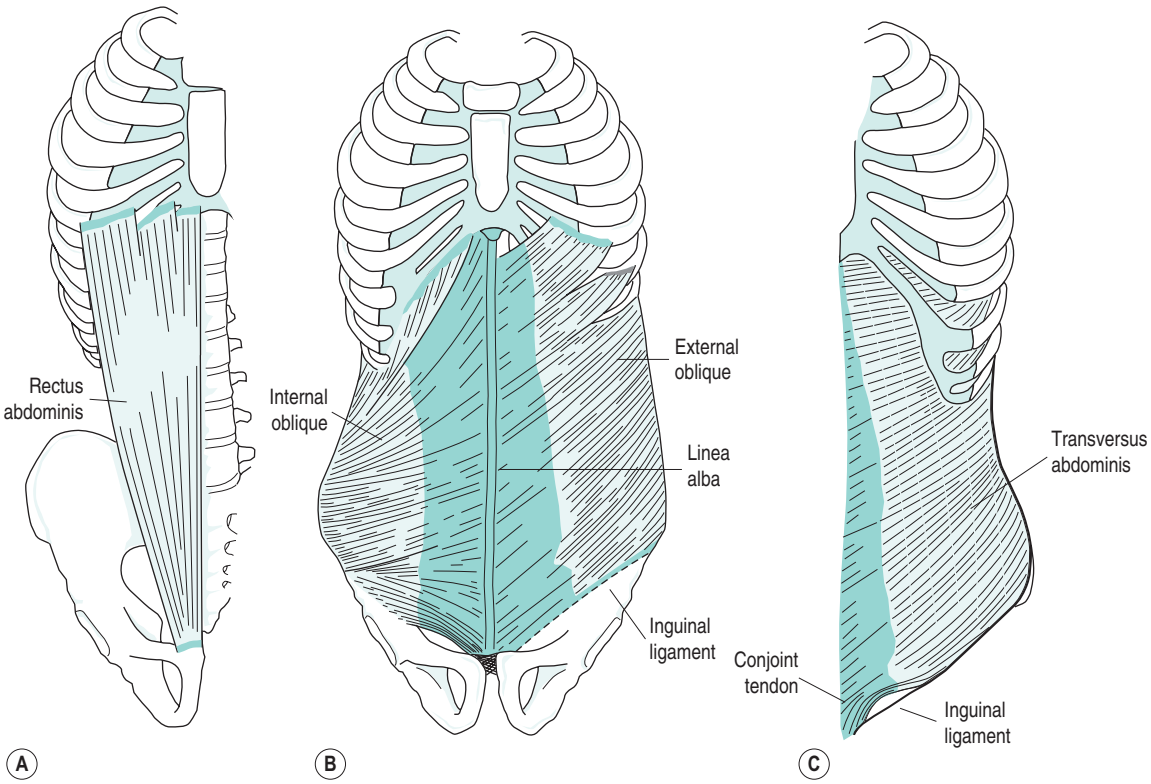


Figure 27.2 (a) Right rectus abdominis. (b) Right internal oblique and left external oblique muscles. (c) Left transversus abdominis. *(Reproduced from Palastanga et al. (2002) with permission.)*

This can be adversely affected by pain, postural malalignment, deficient nerve supply or fascial detachment.

The main function of RA is lumbar spine flexion (Sapsford et al. 2001), while the obliques, interlaced diagonally in midline deep to the recti, produce side-flexion and rotation of the spine.

Co-ordinated action of the abdominal muscles increases intra-abdominal pressure which facilitates expulsive actions – defaecation, micturition and parturition when the pelvic floor is relaxed – and coughing, sneezing or vomiting if the diaphragm is relaxed.

In later pregnancy, the growing uterus stretches the abdominal muscles and may cause RA to be separated in midline by several finger widths (Sapsford et al. 1998). This inter-recti distance – diastasis rectus abdominis muscle (DRAM) – can persist postnatally (Barton 2004; Coldron et al. 2008).

The pelvic floor is a fascial and muscular sheet forming the inferior boundary of the abdominopelvic cavity (Figure 27.3). It supports the pelvic and abdominal contents and comprises four layers. The deepest (viscerofascial) is important for both organ support and muscle attachment. The next part of the supportive mechanism and assisting with the sphincteric control of the bladder and bowel is formed by the levator ani muscles – pubococcygeus (pubovisceral muscle), puborectalis, ileococcygeus and ischiococcygeus. The co-coordinated action of levator ani, in the presence of intact fascia, generates a rise in intra-abdominal pressure to maintain organ support, and urinary and faecal continence. Contraction of the pelvic floor muscles (PFM) results in a cranio-ventral movement that has been observed by real-time ultrasound (Lovegrove Jones et al. 2009). The next layer is a dense triangular membrane lying anteriorly – the perineal membrane. It is of importance for connection of the urethra, vagina and perineal body to the ischiopubic rami (De Lancey 2001). The most superficial external genital muscles – ischiocavernosus, bulbocavernosus and the transverse perineal muscles – have a sexual function in assisting a woman to achieve orgasm.

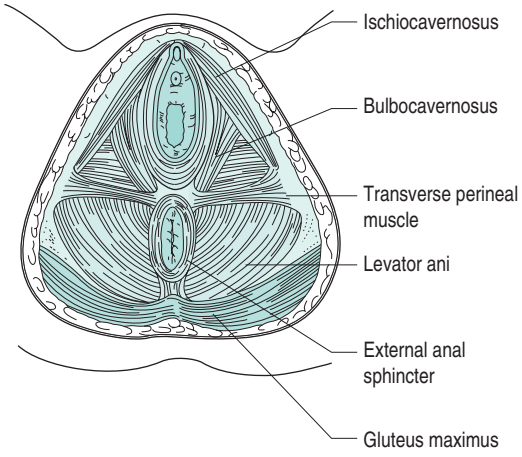


Figure 27.3 Pelvic floor muscles.

It has always been believed that the pudendal nerve, from sacral nerve root S2–4, supplies all of the pelvic floor muscles. However, Barber et al. (2002) suggested that although the pudendal nerve supplies the urethral sphincter and the external genital muscles, levator ani has a distinctive separate nerve supply from S3–5 which has been named the 'nerve to levator ani'. This theory is under some dispute.

Pregnancy and childbirth are major contributory factors to pelvic floor muscle dysfunction (Freeman 2002). Women who suffer urinary incontinence in pregnancy are twice as likely to be symptomatic 15 years after childbirth (Dolan et al. 2003). Vaginal delivery also increases the likelihood of future urinary and faecal disorders, but caesarean section may not be fully protective (MacArthur et al. 2006). It is also thought that there is a possible genetic factor related to collagen (Hannestad et al. 2004).

Organs of reproduction

The uterus – a hollow, pear-shaped organ – comprises the fundus, body, isthmus and cervix, and weighs about 50 g in its normal state (Haslam 2004a). Together with the ovaries, it is suspended in connective tissue and peritoneum inside the true pelvis (Figure 27.1). The broad ligament (a double fold of peritoneum) extends from the lateral walls of the uterus to the sidewalls of the pelvis, dividing the pelvic cavity into two compartments – the anterior (containing the bladder) and the posterior (containing the rectum). The ovaries lie in the broad ligament on each side of the uterus. Behind and slightly above are the trumpet-shaped open ends of the contractile fallopian tubes. Their tentacle-like fringes (fimbriae) catch the ovum when it erupts from the ovary at ovulation. The free ovum is propelled along the fallopian tube to the uterus by a peristaltic wave and the cilia lining the tube walls. On fertilisation, the ovum is embedded in the endometrium lining the uterus. If fertilisation has not occurred, the lining is shed (menstruation).

The uterus has a remarkable capacity to grow as the fetus develops during pregnancy, able to accommodate the baby (in 1 litre of amniotic fluid within a membranous sac) and placenta, which attaches to the uterine wall.

The physiology of pregnancy

Following fertilisation of the ovum, the first sign of pregnancy is amenorrhea (cessation of menstruation). As pregnancy progresses, the uterus grows, its muscle fibres lengthening and thickening, and weight increasing from 50 g to 1000 g at term. By 12 weeks it has enlarged to become an abdominal organ. Gestational dates can be determined by the level of the uterus, which continues to

rise until the later weeks of pregnancy. Co-ordinated contractions of the uterus may be felt by women from about 20 weeks (Braxton Hicks contractions). These contractions assist in blood flow through the placental site and in development of the lower uterine segment. The cervix gradually increases in size with an increase in collagen content, hypertrophy of external muscle fibres and increase in vascularity (McNabb 1997) and becomes slightly softer in consistency. A mucous plug acts as a barrier to infection.

The disc-shaped placenta grows during pregnancy with the major responsibility of maintaining the fetal circulation in order to facilitate blood gas exchange, nutrition and excretion, and to be a barrier to noxious substances. It also produces progestogens and oestrogens.

The fetus grows and develops within the amniotic sac and is nurtured by the placenta via the umbilical cord. Its heartbeat can be detected at about 10 weeks with ultrasonic equipment and movements may be felt by primigravidae (women pregnant for the first time) at 18–20 weeks, whereas multigravidae (women who have experienced a previous pregnancy) may notice this at 16–18 weeks.

Pregnancy is governed and controlled by hormones, which affect various systems. Some are of particular relevance to the physiotherapist:

- progesterone decreases smooth muscle tone, initiates sensitivity to carbon dioxide in the respiratory centre, and causes an increase in maternal temperature, breast development, and storage of fat deposits for milk production;
- oestrogen influences uterine and breast growth and development, prepares prime receptor sites (e.g. pelvic joints) for relaxin, and causes increased water retention. Increased vaginal glycogen predisposes pregnant women to thrush;
- in target areas relaxin replaces collagen with a modified form of greater pliability and extensibility. It may have a softening effect on connective tissue (pelvic floor and abdominal fascia), increasing extensibility in those structures. It also inhibits myometrial activity up to 28 weeks gestation.

On average, pregnancy lasts 40 weeks and is divided into three trimesters, each of three months in duration. Physiological and anatomical changes occur owing to hormonal changes and weight gain. These factors will influence the physiotherapy management of musculoskeletal problems in pregnancy.

Within this text attention will be paid to the changes and complications of most relevance to the physiotherapist dealing with women during pregnancy and the postnatal period. Further reading is advised (see the end of the chapter).

MUSCULOSKELETAL CHANGES DURING PREGNANCY

Postural changes

Kypho-lordotic, sway back and flat back have been described as ideal postures (Kendall et al. 2005). The overall equilibrium of the spine and pelvis alters as pregnancy progresses but there is still confusion as to the exact nature of any associated postural adaptation. With weight gain, increased blood volume and ventral growth of the fetus, the centre of gravity no longer falls over the feet and women may need to lean backwards to gain equilibrium (Abitol 1997) resulting in disorganisation of spinal curves. Reported postures include a reduction in lumbar lordosis (Moore et al. 1990), an increase in both lumbar lordosis and thoracic kyphosis (Bullock-Saxton 1991) or a flattening of the thoracolumbar spinal curve (Gilleard et al. 2002). These differences imply a large individual variation with no 'normal' posture in pregnancy and antenatal lumbopelvic posture may be an accentuation of normal pre-pregnancy posture. There will be compensatory changes to posture in the thoracic and cervical spines, and this combined with the extra weight of the breasts may result in posterior displacement of the shoulders and thoracic spine, and increase of the cervical lordosis. Postural changes may still be present 12 weeks postnatally (Bullock-Saxton 1991).

Articular and connective tissue changes

Altered levels of relaxin, oestrogen and progesterone during pregnancy result in an alteration to collagen metabolism and increased connective tissue pliability and extensibility. Therefore, ligamentous tissues are predisposed to laxity with resultant reduced passive joint stability. The symphysis pubis and sacroiliac joints are particularly affected to allow for birth of the baby. Ligamentous laxity may continue for six months postpartum (Foti et al. 2000).

Biomechanical changes of the spinal and pelvic joints may involve an increase in sacral promontory, an increase in lumbosacral angle, a forward rotatory movement of the innominate bones, and downward and forward rotation of the symphysis pubis (Golightly 1982). The normal pubic symphyseal gap of 4–5 mm shows an average increase of 3 mm during pregnancy (Abramson et al. 1934). Pelvic joint loosening begins around 10 weeks, with maximum loosening near term. Joints should return to normal at 4–12 weeks postpartum (Snow and Neubert 1997). The sacrococcygeal joints also loosen. By the last trimester, the hip abductors and extensors, and the ankle plantarflexors increase their net power during gait

(Foti et al. 2000) and there is an increase in load on the hip joints of 2.8 times the normal value when standing and working in front of a worktop (Paul et al. 1996).

As the uterus rises in the abdomen the rib cage is forced laterally and the diameter of the chest may increase by 10–15 cm (Polden and Mantle 1990).

Neuromuscular changes

Abdominal and pelvic muscles contribute to spinal and pelvic stability via active tension exerted on the passive ligamentous and fascial stability structures. During pregnancy the enlarged uterus results in elongation of the abdominal muscles and separation of the linea alba (Boissonnault and Blaschak 1988). Passive joint instability (as seen in pregnancy) alters afferent input from joint mechanoreceptors and probably affects motor neurone recruitment. A decrease in muscle stiffness and thus active stability of joints may result from alteration of muscle spindle regulation (Bullock-Saxton 1999) and this is applicable particularly to muscles around the pelvic girdle. These changes may lead to poor recruitment of the muscles responsible for pelvic girdle stability (particularly gluteus medius and maximus) and result in decreased tension of these muscles during walking, perhaps resulting in pelvic girdle pain (PGP).

Rectus abdominis

As pregnancy progresses, rectus abdominis (RA) elongates (Gilleard and Brown 1996) and becomes wider and thinner (Coldron et al. 2008). In many women the disproportion between width and thickness remains at 12 months postpartum (Coldron et al. 2008). Although the consequences of this are unknown, the strength of gross muscle flexion can be impaired at 24 weeks postpartum (Potter et al. 1997b).

The linea alba becomes wider and thinner via hormonally-mediated change as the fetus grows anteriorly, with the two bellies of RA curving round the abdominal wall (Haslam 2004b). In nulliparous women the interrecti distance (IRD) at the umbilicus is approximately 11 mm (± 3.62) whereas the divarication of the linea alba during the third trimester and immediately postpartum may vary widely between women, with an average gap of 42 mm (± 20.28) at the umbilicus (Coldron et al. 2008). The linea alba can split and become a diastasis with herniation of the abdominal contents. Diagnostic criteria of a pathological diastasis at the umbilical level in people under 45 years has been defined by Rath et al. (1996) as an IRD of >27 mm. The IRD at the umbilical level has been shown to resolve to about 22 mm (± 9.44) by 8 weeks postpartum, when it reaches a plateau, though in a minority of women a diastasis may persist (Coldron et al. 2008).

Lateral abdominal muscles

There is very little information on the effect of pregnancy on the three lateral abdominal muscles (transversus abdominis (TrA), internal (IO) and external (EO) obliques). However, research on normal subjects has identified the importance of TrA as the prime stabiliser of the trunk (Hodges 1999). Decreased stabilisation of the pelvis happens in late pregnancy and poor stability persists at eight weeks postpartum (Gilleard and Brown 1996). In one study, EO appeared to atrophy during pregnancy but hypertrophy in the postnatal period to become thicker than that of nulliparous controls (Coldron 2006). Furthermore, the thickness of the middle fibres of IO and TrA was significantly greater in day 1 postpartum women than that of controls, but by eight weeks postpartum there was no difference between the two groups. Parity, exercise and a history of lumbopelvic pain had no bearing on the results. The relevance of these findings is unknown but it may be that IO and TrA had an increase in stability function during pregnancy from load-bearing with the weight of the fetus.

Pelvic floor muscles

During pregnancy there is stretching to the pelvic floor and trauma/tearing during labour and vaginal delivery. It is now thought that the function and recruitment of TrA and the pelvic floor musculature are closely associated, with voluntary activity in the deep abdominal muscles resulting in increased pelvic floor muscle activity (Sapsford and Hodges 2001). There is an association between PFM dysfunction and pregnancy-related lumbopelvic pain (Pool-Goudzwaard et al. 2005).

LABOUR, BIRTH AND THE PUERPERIUM

ABC Definition

Labour is defined as the process by which the products of conception are expelled from the uterus after the 24th week of pregnancy.

Labour

Labour can be described through a series of physiological events which culminate in the delivery of the neonate between 37 and 42 weeks of gestation. The specific trigger initiating onset of labour remains unknown, although it is understood that certain fetal and maternal hormonal interactions and mechanical factors play a part. Increased

uterine contractility causes the taking up (effacement) of the ripened cervix and subsequent opening (dilatation) in the primigravid (first pregnancy) woman.

Stages of labour

Labour continues to be described in three stages, though this theory has been challenged in recent years (Winter and Cameron 2006).

First stage

The first stage of labour is from the commencement of regular uterine contractions effecting dilatation (opening) of the cervix, culminating when the cervix is fully dilated, allowing the passage of the fetus into the birth canal. It can be further subdivided into the latent phase (early labour), where contractions are short and irregular, and the active phase (established labour), where contractions become intense and regular (Chapman and Charles 2009). Contractions (tightening) and retraction (shortening) of myometrial muscle fibres increase in length, strength and frequency as labour progresses. The mucous plug (show) is expelled as the cervix opens and the membrane sac (amnion and chorion) often spontaneously ruptures, allowing amniotic fluid to drain.

Various changes can be observed in women adapting to the intensity of contractions. Expenditure of energy increases the need for hydration and food, although as labour progresses appetite is often suppressed. Women may appear hot, flushed and agitated as intense contractions may cause pain, fear and distress. Endorphin release in response to pain provides an analgesic and euphoric bolster, which may cause some women to appear calm, quiet and withdrawn. Many women find relief through their own instinctive behaviour, such as mobility, change of position and posture (De Jonge and Lagro-Jansen 2004), warm baths or compresses, massage, relaxation and distraction techniques.

As the cervix nears full dilatation further changes can be observed in the woman as she enters the 'transition' into second stage. The intensity of contractions increases, exacerbating pain and stress, although the contractions may be less frequent. The woman is exhausted and often expresses defeat. She may appear agitated and overwhelmed by the effort of labour, or conversely calm and removed. Increased vocalisation, spontaneous shaking, rapid movement of the legs, nausea and vomiting may be seen, and she may express an urge to bear down or push.

Second stage

The second stage of labour is the expulsive stage culminating in the birth. Commonly defined as commencing from full dilatation of the cervix which was thought to herald the urge to push, it has been suggested (Long 2006) that second stage labour commences 'when the presenting part has passed through the cervix and is below the ischial spines'. When the presenting part distends the genital tract and pelvic floor a surge of oxytocin is released, known as the fetal ejection or Ferguson reflex, whereby strong expulsive contractions facilitate the birth. Throughout the second stage women should be encouraged to instinctively bear down as the urge occurs with a contraction, adopting positions which increase the pelvic outlet (Gupta et al. 2004). Prolonged breath-holding and overzealous pushing should be avoided, as this interferes with placental perfusion and may compromise the fetus. The environment should feel safe and non-threatening, with minimal intrusion from staff, and the midwife should be confident in her skill to support normal labour (Downe 2010).

Observations

Maternal well-being and progress in labour is determined by observation of the physiological and psychological changes which occur in response to labour.

Pain relief

Kabeyama and Miyoshi (2001) demonstrated that self-control was the most important factor in a satisfactory childbirth experience, and women who experienced labour as a challenge, utilising their own resources through breathing and relaxation, had better outcomes. Management of pain relief should therefore take a woman-centred approach utilising the whole spectrum of pain management options.

Non-pharmacological approaches to pain management, such as position and posture, play an important part in labour and most women will naturally adopt positions which enhance labour, if they are provided the space and freedom to do so. Warm baths, compresses, massage, breathing, relaxation, music, dance and distraction techniques can also be used to great advantage in labour (Hamilton 2010). Complementary therapies, such as hypnotherapy, aromatherapy, acupuncture and reflexology, should also be considered utilising suitably qualified practitioners.

Transcutaneous electrical nerve stimulation (TENS) can be used in labour. A low-frequency high-intensity current of 2–10 Hz is thought to increase the production of endorphins and encephalins. This is applied throughout labour and a high-frequency low-intensity current at 100–200 Hz which activates the pain-gate mechanism (Melzack and Wall 1982) is used during the more intense contractions. The current is introduced via four electrodes placed over the nerve roots to the uterus (T10–L2) and the pelvic floor and perineum (S2–S4). The two channels enable individual control of each pair of electrodes.

TENS is easy to apply, is non-invasive and has no known side effects for mother or baby. It allows mobility and affords the woman some control of her analgesia. It cannot be used in water and may, on occasion, interfere

with the cardiograph tracings. Recent evidence demonstrates that a reduction in the demand for pethidine and other pain relief was found when TENS was employed (Poole 2007).

Inhaled analgesia as a premixed combination of 50% oxygen and 50% nitrous oxide (Entonox) inhaled via a mouthpiece is self-administered. Some women experience a feeling of loss of control or drunkenness, nausea and vomiting; however, the effects of Entonox wear off quickly when inhalation is stopped.

Opiates, such as pethidine, diamorphine and meptazinol, may be used to relieve pain in labour. Pethidine is historically the most common drug of choice in the UK. Easily administered by intramuscular injection these drugs have similar pain-relieving properties, although their benefits in labour have been challenged (Collis 2000) owing to the episodic nature of contractions and the unpleasant side effects of nausea, vomiting, drowsiness, amnesia, feeling of loss of control and depression of the respiratory centre, especially in the neonate. Neonatal drowsiness also interferes with the establishment of breastfeeding.

Epidural will provide a pain-free labour provided it is sited and working correctly. A regional anaesthetic is introduced into the lumbar epidural space by an experienced obstetric anaesthetist. A combination of local anaesthetic (bupivicaine) and opioid (fentanyl) is used to provide adequate analgesic effect with minimal impact on mobility and blood pressure. Continual infusion of drugs as opposed to a bolus dose has been found to reduce the risk of instrumental delivery (COMET 2001). Possible side effects include dural puncture and possible headache, loss of bladder sensation requiring catheterisation, risk of instrumental delivery, infection, backache (possibly as a result of poor posture or localised bruising), total spinal block and respiratory arrest, neurological sequelae (rare).

Perineal trauma

During delivery the perineum stretches to accommodate the fetus and can remain intact, or perineal trauma may occur in the form of grazes and tears. Tears are classified according to the extent of tissue damage – first degree tear involves the skin of the fourchette and second degree also includes the superficial perineal muscles. These tears are usually repaired by the midwife. Third and fourth degree tears involve the anal sphincter and rectal mucosa respectively and require surgical intervention.

Episiotomy

Occasionally, an incision through the perineal tissues may be required but should not be undertaken lightly and always with consent. Two main indications for episiotomy have been identified (Carroli and Belizan 2009): to enlarge the vulval outlet in order to minimise severe trauma to the vagina and perineum, in particular where instrumental delivery is required; and in cases of fetal distress to expedite delivery. Local anaesthetic is infiltrated and a mediolateral incision is made to prevent damage to the Bartholins gland and reduce the risk of extension of the incision into the anal margin (Downe 2010).

Third stage of labour

The third stage is defined as the period from the birth of the baby to complete expulsion of placenta and membranes, and control of haemorrhage from the placental site. It can be managed physiologically or actively with the use of drugs. The placenta and membranes are examined for completeness and occasionally part, or all, of the placenta and/or membranes may be retained, requiring anaesthetic and manual removal.

Caesarean section

Caesarean section is an operative procedure carried out under regional or, more rarely, general anaesthesia. Regional anaesthesia ensures that the woman can participate in the birth and recovers more quickly with less risk of complications.

The fetus, placenta and membranes are delivered through a transverse incision on the bikini line of the abdominal wall and through the lower uterine segment. On rare occasions a vertical or classical incision may be performed; this is associated with increased morbidity.

Assisted birth

Assisted vaginal birth by vacuum extraction or forceps is a widely practised intervention used to expedite delivery where there is delay in the second stage, maternal exhaustion or fetal distress. It is more commonly used among women who choose epidural as a form of pain relief (Anim-Somuah et al. 2005).

Vacuum extraction is the most common choice owing to ease of use and increased safety. However, similar levels of complications occur as with forceps delivery and vacuum extraction is less efficient at achieving vaginal delivery. A suction cup (Ventouse/Kiwi) is placed onto the fetal head, and traction and maternal effort are applied during uterine contraction in order to deliver the fetus.

Forceps delivery involves separately placing two spoon-shaped metal blades (which lock into place) on either side of the fetal head. Slow, steady traction and maternal effort is applied to deliver the fetus. Forceps may also be used to protect the head of the preterm fetus or the after coming head in the breech (Hamilton 2010). Adequate analgesia must be provided prior to assisted birth.

Induction

Induction of labour is an intervention to initiate labour where there is disadvantage to the continuation of

pregnancy. The mother may also request induction for social reasons (NICE 2008a). Indications for induction of labour include prolonged pregnancy (exceeding 42 completed weeks), maternal factors (hypertension, diabetes), or pre-labour rupture of membranes, which may lead to infection risk. Fetal factors include growth restriction, macrosomia (large baby), fetal anomaly or fetal death.

Different approaches to induction can be used individually or collectively:

- *stretch and sweep* – a digital vaginal examination to stretch the cervix, sweeping the membranes and separating them from the uterine wall;
- *prostaglandin* – administered vaginally to ripen the cervix, which may initiate labour;
- *artificial rupture of the membranes* once the cervix has started to dilate;
- *intravenous oxytocin infusion* may also be required.

Puerperium

Puerperium is defined as the six-week period following childbirth during which the woman's body returns almost to its pre-pregnancy state:

- *lactation* is initiated and established or suppressed;
- the uterus involutes, returning almost to the pre-pregnant size and position;
- the *placental site* begins to heal and the lochia (blood loss) diminishes to a creamy white discharge;
- *perineal trauma* and *abdominal wounds* begin to heal.

Many women require a considerably longer time to adjust both physiologically and psychologically to the complex life-changing event of childbirth. High expectations with regard to regaining pre-pregnancy shape and lifestyle are not always reflected in reality and can lead to low self-esteem and feelings of inadequacy. Talking and sharing worries both with the health professional and other mothers can be beneficial, and women should be made aware of local groups (Byrom et al. 2010).

PHYSIOTHERAPY IN THE CHILDBEARING YEAR

Women's health physiotherapists work as part of the multi-disciplinary team (MDT) caring for pregnant women. Contact with pregnant women may be in the community, at a health centre, at a leisure centre or in the physiotherapy department.

The role may include:

- education of pregnant women for pregnancy, labour and beyond (see section on antenatal classes);
- advice on exercise (see the section on exercise and pregnancy);
- identification, assessment and treatment of musculoskeletal problems (dealt with in this section).

Pelvic floor dysfunction

During pregnancy approximately one in three women experiences stress urinary incontinence (Francis 1960; Stanton et al. 1980; Viktrup and Lose 2001) and pregnancy and birth can have an adverse effect on the area through perineal trauma, muscle, connective tissue and pudendal nerve damage (Snooks et al. 1984; Allen et al. 1990). Pelvic floor muscle exercises (PFME) during pregnancy are effective in reducing urinary incontinence in pregnancy and the immediate postnatal period (Mørkved 2007) and the National Institute for Health and Clinical Excellence (NICE 2006) recommends PFME for every woman during her first pregnancy (see the section on urogenital dysfunction). During pregnancy, physiotherapists may consider it prudent to limit their intervention to advice.

Pregnancy-related lumbar spine and pelvic girdle pain

Lumbopelvic pain is common during pregnancy with a prevalence described variously as ranging from 50% to 70% (Mantle et al. 1977; Fast et al. 1987; Berg et al. 1988; Ostgaard and Andersson 1991; Wu et al. 2004; Mogren and Pohjanen 2005; Gutke et al. 2006). It may be of spinal and/or pelvic girdle origin (Ostgaard et al. 1996; Stuge et al. 2003; Wu et al. 2004; Vleeming et al. 2004; Mogren and Pohjanen 2005; Gutke et al. 2006). Pain of spinal origin is normally referred to as low back pain (LBP). Pregnancy-related PGP is a global term that encompasses symphysis pubis dysfunction, diastasis symphysis pubis and sacroiliac joint pain.

Lumbopelvic pain is often regarded as 'a normal part of pregnancy' but, without appropriate treatment, a minor episode may develop into a chronic problem. A third of women report severe back pain that interferes with daily life and compromises their ability to work (Ostgaard and Andersson 1991; Mens et al. 1996). Most backache resolves quickly postpartum, but may continue for 18 months (Ostgaard and Andersson 1992) or present postpartum for the first time (Russell and Reynolds 1997). Some patients experience a relapse around menstruation and in a subsequent pregnancy (Mens et al. 1996).

The anatomical origins of pregnancy-related lumbopelvic pain vary and are difficult to determine and diagnose (Nilsson-Wikmar et al. 1999). Common conditions include unilateral sacroiliac dysfunction, symphysis pubis dysfunction, minor lumbar disc herniation, lumbar zygapophyseal joint problems, thoracic spine pain and coccydinia. Women describe pain variously as occurring in the

low back, sacral, posterior thigh and leg, anterior thigh, pubic, groin and hip areas. These may occur simultaneously or separately, antenatally, during delivery or postnatally (Heiberg and Aarseth 1997). There is often associated cervical, thoracic or coccygeal pain. Sciatic pain is common and may be of lumbar origin or from sacroiliac joint involvement because the L5 and S1 components of the lumbosacral plexus run immediately anterior to the sacroiliac joints. Several studies have differentiated between pregnancy-related LBP and PGP (Ostgaard et al. 1994, 1996; Ostgaard 1997; Noren et al. 2002; Vleeming et al. 2004; Bastiaanssen et al. 2005; Gutke et al. 2006). It is important that both the lumbar spine and pelvic girdle are examined to determine the origin of symptoms and plan appropriate management.

Causes of lumbopelvic pain appear multi-factorial and may include postural adaptations, fatigue, increased joint mobility, increased collagen volume causing pressure on pain-sensitive structures, weight gain and pressure from the growing fetus (Haslam 2004b). The main risk factors are a history of previous lumbopelvic pain and/or previous trauma to the pelvis and possibly a high workload and multiparity (Berg et al. 1988; Ostgaard and Andersson 1991; Kristiansson et al. 1996a; Larsen et al. 1999; Vleeming et al. 2004). Others may include pelvic girdle pain in a previous pregnancy (Larsen et al. 1999), poor workplace ergonomics and awkward working conditions (Larsen et al. 1999), abdominal sagittal and transverse diameters and a naturally large lumbar lordosis (Ostgaard et al. 1993), and also a decreased fitness level before pregnancy (Ostgaard et al. 1993). Factors not associated with PGP include contraceptive pill use, time interval since last pregnancy, height, smoking, age and breastfeeding (Berg et al. 1988; Kristiansson et al. 1996b; Larsen et al. 1999).

Reported musculoskeletal factors contributing to pregnancy-related PGP include the pelvic girdle joints moving asymmetrically (Damen et al. 2001), symphyseal laxity (Björklund et al. 1999) and ligamentous strain and muscle weakness (Mens et al. 1996). PGP is probably caused by a combination of these factors plus altered activity in the spinal (Sihvonen et al. 1998), abdominal, pelvic girdle, hip (Pool-Goudzwaard et al. 1998) and pelvic floor muscles (Pool-Goudzwaard et al. 2005) leading to abnormal pelvic girdle biomechanics and stability. However, a small number of women might have non-biomechanical but hormonally-induced pain in the pelvic girdle.

Symphysis pubis dysfunction

Symphysis pubis dysfunction (SPD) relates to pain in the region of the symphysis pubis joint whereas diastasis symphysis pubis (DSP) is a true separation of the symphysis pubis joint confirmed radiologically. The definition of DSP is symphyseal separation of more than 10 mm and vertical shift of more than 5 mm (Hagen 1974). The amount of symphyseal separation does not always correlate with symptoms (Snow and Neubert 1997) and not all symptomatic patients have an increased gap. SPD or DSP may occur antenatally, during delivery or postnatally, and might cause severe social difficulties (Fry 1999). Trauma to the symphysis pubis may occur during a difficult delivery where forceful and excessive abduction of the thighs is necessary (Capiello and Oliver 1995; Gherman et al. 1998; Heath and Gherman 1999; Kharrazi et al. 1997; Albert et al. 2001).

Diagnosis of PGP

Pain distribution may be in the groin, medial and anterior thighs, perineum, coccyx, and one or both sacroiliac joints (Fry 1999; Coldron 2005). Severity and irritability vary from mild to severe and may differ day-to-day. Common physical signs are pain on thigh abduction, turning in bed, lifting a light weight, getting up from a chair and using stairs, a shuffling or waddling gait, severe symphyseal tenderness and an inability to weight-bear unilaterally (Fry 1999; Hansen et al. 1999). Self-reported pain locations in the pelvis, a positive posterior pain provocation test and a sum of other provocation tests (compression/distraction; Patrick-Faber test; palpation of the symphysis pubis; palpation of the long dorsal ligament) were significantly associated with disability and pain intensity in late pregnancy (Robinson et al. 2010). Furthermore, distress was significantly associated with disability. The active straight leg raise (ASLR), fear-avoidance beliefs and the number of pain sites were not associated with pain and disability. Minor trauma, such as stepping down from a kerb, may cause severe symphysis pubis pain. A forward rotation and oblique slip of the innominate caused by overactivity in the adductor muscles of the thigh may contribute to SPD (Röst 1999). With poor use of the glutei and lack of force closure of the pelvis, disruption of the self-locking mechanism of the pelvis may occur.

Non-musculoskeletal causes of PGP

Proposed non-musculoskeletal factors for PGP include increased hyaluronidase (Schwartz et al. 1985), oral contraceptives (Wreje et al. 1997) – although this is disputed by Björklund et al. (2000b) – and genetic susceptibility (MacLennan and MacLennan 1997). The role of relaxin in production of pregnancy-related lumbopelvic pain is controversial with some evidence suggesting an association between relaxin and PGP (MacLennan et al. 1986b; Kristiansson et al. 1996b) while other evidence shows none (Albert et al. 1997; Björklund et al. 2000a). Relaxin levels are at their highest during labour but fall to almost non-pregnant levels by three days postpartum (MacLennan et al. 1986a). As many women experience postpartum lumbopelvic pain, other reasons apart from serum relaxin levels probably contribute.

Conclusive association between new-onset postpartum backache and epidural analgesia has not been demonstrated (Breen et al. 1994; Russell et al. 1996; MacArthur et al. 1997; Howell et al. 2002; Loughnan et al. 2002) though its masking effect on pain may lead to women adopting unsuitable positions in labour (MacArthur et al. 1990).

Management of lumbopelvic pain and dysfunction

It is important to acknowledge that pregnancy-related lumbopelvic pain is a common, recognised condition, which is better managed with prompt identification, assessment and appropriate treatment. If left untreated it may last more than two years (Albert et al. 2001).

Advice, posture, education and general exercise

Antenatal education on posture and back pain by a physiotherapist has been shown to reduce back and pelvic pain, reduce sick leave and continue to benefit women in the postnatal period (Noren et al. 1997). Antenatal advice includes adopting comfortable resting positions; moving out of bed, a chair or the car; postures in walking and standing; and correct lifting and handling. In addition, postnatal advice includes positions for breastfeeding, nappy changing, bathing and handling the growing baby.

Woman may be advised to (ACPWH 2010a):

- remain active within the limits of pain;
- accept offers of help and involve partner, family and friends in daily chores;
- rest when needed;
- avoid standing on one leg;
- consider alternative sleeping positions;
- explore alternative ways to climb stairs;
- plan her day;
- avoid activities that involve asymmetrical positions of the pelvis;
- consider alternative positions for intercourse;
- organise hospital appointments for the same day if possible;
- avoid activities which make the pain worse (e.g. vacuuming, pushing a supermarket trolley, lifting heavy weights).

Liaison with midwives is essential. Women should be aware of the masking effect of epidural and spinal anaesthesia in relation to excessive abduction of hips during labour and delivery. If possible, they should adopt the most comfortable position during labour (for example left-side-lying, or kneeling upright with support). They should be discouraged from placing their feet on attendants' hips and care should be taken if lithotomy is required (ACPWH 2010a). Suturing should take place in the most comfortable position for the mother.

Treatment of articular dysfunctions/ movement restriction of the spine and pelvic girdle

There is some evidence that pregnancy-related lumbopelvic pain may respond to manual therapy (Daly et al. 1991; Diakow et al. 1991; McIntyre and Broadhurst 1996), and correct assessment of the spine and pelvic girdle is imperative to enable treatment to be targeted at the correct structures. Providing severity and irritability has been properly assessed, many manual therapy techniques in use in the non-pregnant population can be used and adapted for women with pregnancy-related lumbopelvic pain. Many can be undertaken with the woman in side lying.

Management depends upon whether there is movement limitation caused by a true articular restriction, or the joint is held in an abnormal position by imbalance or altered recruitment of the muscles (Lee and Vleeming 2000). Lee (1999) recommended that articular restrictions be treated first with mobilisation or manipulation techniques followed by treatment of myofascial structures using muscle energy techniques (MET). Other techniques for treating myofascial dysfunctions include trigger points, strain/counterstrain, positional release, soft tissue manipulation techniques or taping to offload overactive muscles. Unilateral or bilateral muscles that may be overactive with PGP are the hip adductors, psoas, piriformis, erector spinae and quadratus lumborum. Tightness may be palpated in the posterior fibres of the pubococcygeus.

With SPD, special attention should be paid to overactive pelvic adductors, underactive abductors, unilateral displacement of one innominate bone (Röst 1999) and poor pelvic girdle and spinal stabilising muscles.

The use of a sacroiliac/trochanteric belt for sacroiliac and symphysis pubis instability both ante- and postnatally may stabilise the pelvic girdle joints (Damen et al. 2002; Mens et al. 2006) and substitute the work of the internal oblique muscle (Snijders et al. 1998) but its effect on pain is equivocal (Depledge et al. 2005). However, a belt should be tried if ASLR test is positive. A large tubular bandage for the abdomen or a maternity belt may give added support. In the most severe cases crutches or a wheelchair may be required.

Muscle re-education

Abdominal, spinal and pelvic girdle muscle motor control needs to be retrained to stabilise an unstable spinal segment or pelvic girdle joint. Specific spinal and pelvic girdle stabilising programmes are effective in reducing pain antenatally (Elden et al. 2005) and postnatally (Stuge et al. 2004a, 2004b, 2006).

Rehabilitation exercises antenatally and postnatally should concentrate initially on correct recruitment and strength training of both pelvic girdle stabilising muscles (gluteus medius and maximus) and core lumbopelvic stabilising muscles (transversus abdominis, lumbar

multifidus, pelvic floor muscles). Poor recruitment and decreased strength of hip adductors and flexors have been shown to be a factor in pregnancy-related PGP (Mens et al. 2002), and altered recruitment of gluteus maximus has been shown in patients with sacroiliac joint (SIJ) pain. No studies of gluteus medius in pregnancy-related PGP have been undertaken, but the waddling gait of these patients is a common sign and is probably a result of poor gluteus medius control. It is proposed that the main muscles to be targeted in women with PGP are the trunk core stability muscles, as described above, plus the hip abductors, adductors, flexors and extensors. Consideration of the balance between adductors and abductors should be given owing to their function in providing stability across the symphysis pubis (Lee 1999). Exercise for global stabilising muscles, such as the oblique abdominals, erector spinae, latissimus dorsi and iliopsoas should follow as pain and physical ability allows. The role of RA in lower abdominal strength and support should not be ignored as Coldron et al. (2008) found that characteristics of RA shape and size had not returned to normal values by 12 months post-partum. A thinner, wider and longer RA has implications for strength and fascial support, and may cause decreased stiffness of the anterior abdominal wall and predispose to a mechanical disadvantage. Exercises that target the return of normal RA width, thickness (strength) and length without loading and compressing the lumbar spine are required.

Rehabilitation exercises need to be functional, as many women cannot regularly attend a physiotherapy department owing to family commitments. During pregnancy women should be encouraged to retain their cardiovascular fitness where possible and, if necessary, increase it postpartum.

Management of diastasis rectus abdominis

The consequences of a persistent diastasis rectus abdominis (DRA) are not known fully but a relationship between DRA and diagnoses of stress urinary incontinence, faecal incontinence and pelvic organ prolapse has been shown (Spitznagle et al. 2007). It is also possible that the mechanical advantage of the two RA bellies is compromised and thus could affect muscle strength and potential development of lumbopelvic pain. The IRD should resolve to approximately 20 mm by 8 weeks postpartum (Coldron et al. 2008) which approximates to 1–2 finger widths. However, in some women the IRD does not return to normal values by 12 months postpartum (Coldron et al. 2008) and, together with the changes to RA, anterior abdominal wall stiffness is likely to be compromised. Physiotherapists should examine the postnatal gap between the recti at the level of the umbilicus and assess the IRD. If there appears to be a persistent DRA (>3 finger widths) advice regarding exercise should be given, including initial training of the deep abdominal muscles (TrA) (Sheppard 1996; Potter et al. 1997a), gentle exercises to strengthen RA while avoiding strong trunk curling exercises to prevent herniation of abdominal contents. In a small study, the occurrence and size of DRA in pregnant women was found to be much greater in non-exercising pregnant women than in those who exercised (Chiarello 2005) supporting the view that good fitness pre-pregnancy is of benefit to the woman.

Pain management

There is increasing evidence that acupuncture is effective in relieving antenatal lumbopelvic pain (Wedenberg et al. 2000; Ternov et al. 2001, Guerreiro da Silva et al. 2004; Kvorning et al. 2004; Elden et al. 2005; Lund et al. 2006) and recent evidence suggests that acupuncture has no observable adverse effects on the pregnancy, mother, delivery or the fetus/neonate (Elden et al. 2008). Some experts advise that it is avoided in the first trimester (Forrester 2003) and acupuncture points that have been traditionally associated with abortion (miscarriage) should also be avoided (Betts 2003; West 2008), though this latter point is controversial as no adverse effects have been reported.

The use of TENS antenatally for pain relief is controversial owing to concerns about fetal malformations and spontaneous abortion, but no negative effects have been reported from the use of TENS during any stage of pregnancy (ACPWH 2007). Therefore, its use in pregnancy for lumbopelvic pain could be justified provided the usual precautions and contraindications are observed, the current density is kept low, large electrodes are used, electrodes are not placed over the abdomen and acupuncture points thought to induce labour are avoided. Although the evidence for pain relieving effects of TENS is equivocal (Khadilkar et al. 2008) it poses less risk than most analgesic medicines, so could be tried.

Rib pain

Pain along the anterior surface of the lower ribs (possibly related to pressure from the ascending uterus, and commonly called 'rib flare') may be accompanied by thoracic spine and lateral chest pain. This may be relieved by side flexion manoeuvres away from the pain and manual therapy techniques.

Nerve compression syndromes

Fluid retention may occur during the third trimester, which can lead to a variety of nerve compression syndromes. These include carpal tunnel syndrome (CTS), brachial plexus compression, meralgia paraesthetica (compression of the lateral cutaneous nerve of the thigh as it passes under the inguinal ligament, presenting as tingling and burning in the outer thigh) and posterior

tibial nerve compression. These entrapments normally resolve postpartum.

Wrist splints and ice are useful for CTS. Postural advice can be used for brachial plexus compression. Ice and elevation may help posterior tibial nerve compression (Polden and Mantle 1990).

EXERCISE AND PREGNANCY

General issues

Physiological, emotional, social and psychological issues influence physical fitness in pregnancy. Physiotherapists must be sensitive towards these, and be aware of other issues, such as language, ethnic cultures, equal opportunities and women with special needs. The therapist's approach to pregnant women should be holistic, flexible, individual, and – where available – evidence-based.

Many women exercise regularly and wish to continue during pregnancy. Some women take up exercise for the first time, and exercise should be encouraged during pregnancy (RCOG 2006). Special guidelines for safe activity and exercise in pregnancy exist for women with weight-management problems, diabetes and pre-eclampsia (CMACE/RCOG 2010; NICE 2010a). Health professionals should use any appropriate opportunity to provide information about the health benefits of losing weight before becoming pregnant to women with a body mass index (BMI) ≥30. This should include information on the increased health risks their weight poses to themselves and would pose to their unborn child.

Research suggests that mild-to-moderate exercise is beneficial to healthy pregnant women (Clapp 2000) and is not harmful to the fetus (Clapp et al. 2000; Riemann and Kanstrup Hansen 2000; ACOG 2002; Arena and Maffulli 2002). Moderate intensity is defined as being able to talk easily, while increasing the heart rate to a maximum of 140 beats per minute. Choice of exercise must be influenced by the physiological changes which will occur (Artal and O'Toole 2003). For example, plasma volume increases before red cell volume, leading to a decreased ability to provide oxygen in response to demand. Also, increased demand causes raised respiratory rates, cardiac output values increase during pregnancy for the same activity (over the non-pregnant woman) and there is a loss of cardiac reserve.

Benefits and contraindications

Potential *benefits* of exercise include:

- maintenance of cardiovascular fitness;
- maintenance of healthy weight range;
- improvement of body awareness, posture, co-ordination and balance;
- improvement in circulation;
- increase in endurance and stamina;
- provision of social interaction with exercise, enhancing feelings of social and emotional well-being;
- possible reduction in problems during labour and delivery;
- potentially shorter labour;
- possible prevention of gestational diabetes;
- reduction in minor complaints of pregnancy;
- more rapid postnatal recovery.

Contraindications to exercise include:

- cardiovascular, respiratory, renal or thyroid disease;
- diabetes (type 1, if poorly controlled);
- history of miscarriage, premature labour, fetal growth restriction, cervical incompetence;
- hypertension, vaginal bleeding, reduced fetal movement, anaemia, breech presentation, placenta praevia (ACOG 2002).

Advice

The advice given to regular and non-regular exercisers will differ.

Regular exercisers

- Consult your doctor or midwife before beginning exercise.
- Exercise at a moderate level most days for 30 minutes or more (Hefferman 2000; RCOG 2006).
- Discontinue contact sports and activities which carry a high risk of falling or abdominal trauma. Avoid scuba diving (RCOG 2006).
- Self-regulate both the level of intensity and duration of exercise, aiming to keep core temperature below 38°C.
- Aim for low impact activity.
- Wear suitably supportive footwear to reduce musculoskeletal stresses.
- Maintain adequate fluid intake to prevent dehydration, and avoid exercise during hot and humid weather, or with pyrexia.
- Warm-up and cool-down for at least five minutes.
- Do not use developmental stretching (because of the effects of relaxin).
- Seek professional advice on specific exercises (e.g. for the pelvic floor muscles).
- Avoid ballistic exercise, low squats, crossover steps and rapid changes of direction.
- Do not exercise in supine after 16 weeks gestation, to avoid aortocaval compression.
- Eat to appetite, without calorific restriction.
- Work towards cross-training to avoid over-training, and stop exercise before fatigue sets in.

Non-regular exercisers

In addition to the above, women not used to regular exercise should be advised:

- not to start an exercise programme until >13 weeks gestation;
- to consider beginning with non-weight-bearing exercises, such as aquanatal classes;
- to progress from simple and basic levels of exercise, increasing exercise tolerance gradually, under the supervision of a suitably qualified professional.

When to stop

All women should stop exercising immediately and seek advice from a doctor if they experience:

- abdominal pain;
- per vaginum (from the vagina) bleeding;
- shortness of breath, dizziness, faintness, persistent severe headache, palpitations or tachycardia;
- PGP, which may also lead to difficulty in walking.

Most women will naturally reduce the amount of exercise they take during pregnancy as their weight increases, and they fatigue and become breathless more rapidly.

Types of exercise

General categories

- *Leisure sports.* Provided the contraindications have been noted and the woman is familiar with her chosen activity, it is safe to continue pursuing leisure sports. These may include brisk walking, swimming, jogging, hiking, rowing and dancing. The pace adopted should be sufficient to cause aerobic changes. If PGP is a problem, avoid the kicking motion of the legs during breast stroke swimming.
- *Low impact aerobics* (or equivalent classes). The emphasis is on maintaining fitness levels.
- *Pilates or yoga* (modified for pregnancy) cater for the non-aerobic elements of fitness – flexibility, control of breathing and relaxation.
- *Back care classes.* Core stability exercises may be taught, sometimes using a Swiss ball.
- *Gym work.* The pregnant woman may have access to a static bicycle, treadmill or cross-trainer, all of which encourage aerobic activity. Technique is especially important when strength training. Women should use light weights, with submaximal lifts, aiming to use both upper and lower body muscle groups in a variety of exercises. Weights, sets and repetitions should be decreased further as pregnancy progresses (Avery et al. 1999)

Aquanatal classes

Pregnant women may find exercise and relaxation in water enjoyable and beneficial, largely because of the feeling of weightlessness and the reduced jarring of the joints. It has been suggested that women submerged to the xiphisternum will experience only 28% of their body weight (Harrison and Bulstrode 1987). Women in aquanatal classes may get relief from aches and pains, feel they have more energy after and sleep better. Another important benefit is the absence of post-exercise muscle soreness because, during immersion, all muscle work is concentric (Newham 1988). Other significant advantages are less obvious to the woman: exercise in water helps to tone the respiratory muscles; the leg movements of swimming and exercise in water aid venous return; and the diuretic effect of immersion is helpful to pregnant women troubled by fluid-retention, as immersion for 20–40 minutes results in a loss of 300–400 ml of fluid (Katz et al. 1991).

Women must be screened for contraindications and for any musculoskeletal problem of which the teacher should be aware (ACPWH 2010c). The water temperature should be between 28 and 32°C (ACPWH 2010c).

Exercises can be amended for someone with a musculoskeletal problem. Women with PGP may be more comfortable if they modify their breast stroke leg movements and take small, rather than wide, steps sideways. All women should take short backward steps to avoid an increase in lumbar lordosis. Exercises that overstretch the already compromised abdominal muscles should be excluded. Conversely, squatting, which is difficult on land and thought by some to be damaging, is safe in water as virtually no weight goes through the knee joints.

The aim of the class is to help maintain, not improve, a woman's level of fitness. Exercises must be safe and carefully chosen, and each included for a reason. Water exercises should be considered in their own right and not taken unchanged from exercise classes on land (Evans 2002). It is necessary to warm-up in the water before starting aerobic and strengthening exercises. Buoyancy can be used to assist movement and to make the exercises easier, or to resist movement and allow muscle-strengthening. Pelvic floor muscle exercises should be included before, during or at the end of the class. Good posture should be taught at the beginning of the class and participants reminded to maintain it throughout the class. If the water is warm enough, a relaxation session is an excellent way to end the class.

ANTENATAL CLASSES

It is recommended that all pregnant women receive appropriate antenatal advice and information (NICE 2008b), including access to parenting education, and preparation

for birth classes (DH 2004). A physiotherapist has been identified as a member of the MDT (DH 2004).

Midwives, physiotherapists and other healthcare professionals typically work together to provide antenatal education in the form of classes and group activity. The sessions take many formats, and vary in number and timing. They should provide high-quality advice and information at a convenient time in a location that is accessible, and in a format that is appropriate to the needs of the woman and anyone who might accompany her. Historically, women's health physiotherapists participated in several classes, but within recent years this has been decreased in many places (if not stopped). Therefore, for many, an additional challenge is to include the most pertinent information within a limited time, in particular topics that relate to the unique knowledge and skills of physiotherapists.

'Early bird' groups may be held in early pregnancy; however, historically, the majority of women attend classes in the third trimester. Classes may be for first-time mothers or for women who have had previous pregnancies; commonly they include a mixture of both. There may be specific classes, for example for twin pregnancies, for teenagers and for non-English-speaking women.

The physiotherapist must choose the class content to suit her/his audience and might include:

- advice on safe exercise;
- back and pelvic girdle care;
- pelvic floor and abdominal exercises;
- activities of daily living and work;
- management of pregnancy-related musculoskeletal dysfunctions;
- coping strategies for labour, e.g. relaxation, positions of comfort, breathing awareness;
- advice on early postnatal exercises and return to fitness.

Other sections of this chapter (e.g. 'Labour', 'Birth and the puerperium', 'Physiotherapy in the childbearing year', 'Exercise and pregnancy', 'Urogenital dysfunction') provide more information on what might be included in antenatal classes.

POSTNATAL PHYSIOTHERAPY

The role of the physiotherapist in the days, weeks and sometimes even months following the birth includes advice for new mothers on how to regain and perhaps improve their former level of fitness through appropriate exercise and education. Also included are the assessment and treatment of specific physical problems, emotional support and health education. Although it might be considered the ideal for every woman to be advised by a women's health physiotherapist postnatally, this is becoming increasingly uncommon. Many women will not be seen by a physiotherapist, but should be given appropriate advice by a midwife and high-quality written information.

Inpatient postnatal physiotherapy, if offered, may include the following:

- *caesarean section* – early mobility, bed exercises if indicated, treatment of respiratory complications, pelvic floor muscle and abdominal exercises;
- *perineal trauma* – physiotherapeutic interventions might include advice, exercises, ice or appropriate electrotherapy;
- *incontinence* – training in PFM exercises by a physiotherapist postnatally, incorporating strategies to improve compliance, has been shown to reduce the incidence and severity of urinary incontinence (Chiarelli and Cockburn 2002), and the effect of postnatal pelvic floor muscle training on the prevention and treatment of stress urinary incontinence has been shown to endure one year after delivery (Mørkved and Bø 2000). Persistent pelvic floor dysfunction should be treated (see section on urogenital dysfunction). Incontinence of stool can also occur and is more prevalent among women who experienced a third-degree (partial or complete disruption of the anal sphincter) or fourth-degree tear (complete disruption of the external and internal anal sphincter and epithelium) during delivery (Eason et al. 2002; see section on anorectal dysfunction).
- *musculoskeletal problems* – see the earlier section on musculoskeletal problems in the childbearing year.

Postnatal groups

Physiotherapists may also participate in hospital- or community-based postnatal groups offering advice and appropriate exercise. Stress-free physical activity is associated with a reduced likelihood of developing postnatal depression (Koltyn and Schultes 1997) and may help women return to pre-pregnancy levels of fitness.

UROGENITAL DYSFUNCTION

Problems relating to the female urinary and genital tracts are common and often complex. The problems encountered most frequently by physiotherapists are bladder dysfunction and pelvic organ prolapse.

Bladder dysfunction

Most common is urinary incontinence, which may occur at any time in a woman's life but rises in incidence with age. A study of women aged 50–74 found that some

leakage of urine was reported by 47% and regularly by 31% (Holtedahl and Hunskaar 1998); other authors report similar findings. Although there are several types of urinary incontinence, this chapter will concentrate on those most commonly treated by the women's health physiotherapist.

Stress urinary incontinence (SUI) is defined as the complaint of involuntary leakage on effort or exertion, or on sneezing or coughing (ICS 2002). This is the most common type of incontinence and may coexist with overactive bladder. The urethra has to remain closed and sealed, except during voluntary bladder emptying. Continence is maintained by urethral closure pressure, which must remain higher than detrusor (bladder muscle) pressure, both at rest and on physical exertion. Physical effort causes a sudden rise in intra-abdominal pressure, which is transmitted to the bladder. When pressure in the bladder rises above that of the urethra, there will be an involuntary escape of urine. The pelvic floor muscles (PFMs) contribute significantly to the continence mechanism, providing about one-third of urethral closure pressure (Raz and Kaufman 1977; Rud et al. 1980).

Urgency urinary incontinence (UUI) is involuntary leakage accompanied by, or immediately preceded by, urgency; that is, a sudden compelling desire to pass urine, which is difficult to defer (ICS 2002). This form of leakage occurs when the detrusor contracts inappropriately as the bladder fills (overactive bladder). Urinary urgency, frequency and nocturia (waking to pass urine during the night) can exist without leakage, and may be deemed troublesome by women. Although UUI may be idiopathic, the symptoms can have a neurological cause, for example multiple sclerosis (see below).

Mixed urinary incontinence describes a mixture of SUI and UUI.

Neurogenic detrusor overactivity describes bladder dysfunction of neurological origin (ICS 2002). Women commonly report symptoms as described above under urgency urinary incontinence.

Pelvic organ prolapse

Pelvic organ prolapse (POP) is the descent of one or more of the anterior/posterior vaginal wall; apex of the vagina (cervix, uterus); or vault (cuff) after hysterectomy (ICS 2002). It may be further categorised from stage 1 (least) to stage 4 (most) severe. Symptoms may include the feeling of a lump ('something coming down') or heaviness in the vagina, and urinary or bowel symptoms may coexist.

POP occurs when the fibromuscular supports of the pelvic organs fail. Studies suggest that 50% of parous women (those who have had children) have some loss of PFM support (Bø 2006) which might contribute to POP, though not all will be symptomatic, with a reported incidence of 4–12.2% (Tegerstedt et al. 2005). Severity increases with age and number of vaginal births (Rortveit et al. 2007).

Factors contributing to urogenital dysfunction

It is widely accepted that urogenital problems are associated with vaginal delivery (Wilson et al. 1996; Toozs-Hobson 1998). For many women, childbirth is probably the most significant factor contributing to the development of symptoms. Allen et al. (1990) suggest that a woman's first vaginal delivery causes muscle, fascial and nerve damage, and it is likely that further damage will occur with future deliveries. Chiarelli and Campbell (1997) suggest that forceps delivery increases this risk. There are other reported risk factors: pregnancy itself, straining at stool, heavy lifting, inappropriate exercise, chronic cough, obesity, pelvic surgery, hormonal status and ageing.

Risk factors for POP include symptoms during pregnancy, a maternal history of POP and heavy physical work (Slieker-ten Hove et al. 2008). The same author reports risk factors identified in earlier studies, including previous hysterectomy, surgery for urinary incontinence and/or POP, education, family history, lung disease, smoking, BMI, hormone replacement therapy, race and medication. Brækken et al. (2009) also found a link with pelvic floor muscle function.

Physiotherapy

The role of physiotherapy in the management of urogenital dysfunction is well established and largely evidence-based. The National Institute for Health and Clinical Excellence (NICE 2006) states that the first-line treatment for women with stress or mixed urinary incontinence should, following digital assessment of the muscle contraction, be a trial of supervised pelvic floor muscle training (PFMT) for at least three months. Without digital examination (i.e. verbal or written instructions) many women may not be contracting the muscles correctly (Bump et al. 1991). PFMT has been shown to be effective for women with stress (Dumoulin and Hay-Smith 2010; Imamura et al. 2010) and all types (Dumoulin and Hay-Smith 2010) of urinary incontinence. It appears to be most effective when intensive (Imamura et al. 2010). Brækken et al. (2010) showed that PFMT can be an effective treatment for POP, without adverse effects.

Pelvic floor muscle training

It has been suggested that PFMT is effective for women with stress urinary incontinence by building up long-lasting muscle volume which provides structural support (Bø 2004). In addition, women are advised to contract

their PFMs before and during coughing, sneezing, etc., which has been shown to reduce leakage in women with stress urinary incontinence (Miller et al. 2008). Although a PFM contraction is normally accompanied by co-contraction of transversus abdominis (TrA) there is, to date, insufficient evidence for the use of TrA training instead of, or in addition to, PFMT for women with urinary incontinence (Bø et al. 2009).

Many widely differing exercise protocols are described in the literature. Bø (2007) advises close to maximum contractions, building up to 8–12 three times a day, and weekly supervised training. The ACPWH (2009) suggests both long and short squeezes, three times a day.

Teaching PFM exercises

It is important that physiotherapists can teach a pelvic floor contraction, particularly when they do not have the opportunity to examine a woman to check her technique (e.g. antenatal classes). The following instructions have been suggested (ACPWH 2009):

1. Imagine you are trying to stop yourself from passing urine and breaking wind
2. Squeeze tightly inside your vagina
3. Lift and squeeze.

It has been suggested that imagery may help women contract correctly (Bø and Mørkved 2007):

1. A lift or elevator – closing the doors (squeeze) and moving up (lift)
2. Eating spaghetti
3. Action of a vacuum cleaner.

Biofeedback

Biofeedback may be via electromyography, a pressure sensor or real-time ultrasound. The woman receives immediate visual information regarding her pelvic floor activity and may be able to modify/increase her effort accordingly. It has been suggested that PFMT with biofeedback is an effective treatment for women with stress urinary incontinence (Imamura et al. 2010). Vaginal cones also constitute a form of biofeedback and are used by some physiotherapists as an adjunct to PFMT. Recent studies (Castro et al. 2008; Gameiro et al. 2010) suggest that they are no more effective that PFMT.

Electrical stimulation

Electrical stimulation (ES) has been suggested as a treatment for stress urinary incontinence (Laycock 2003) or overactive bladder (Yamanishi et al. 2000). Goode et al. (2003) showed that the addition of ES to a comprehensive behavioural programme for women with SUI did not increase the effectiveness of treatment. If used, ES may be applied in a physiotherapy department, or used at home.

Behavioural modification

Physiotherapy interventions normally include advice, and the management of urogenital dysfunction is no exception. Women with SUI might be advised on weight-loss, adjustment of fluid intake, smoking cessation, exercise modification and regularisation of bowel habit (Imamura et al. 2010). For POP, advice might include avoidance of heavy physical work, lung disease and smoking, which have been suggested as risk factors for the condition (Slieker-ten Hove et al. 2008).

Men

Bothersome lower urinary tract symptoms (LUTS) are present in up to 30% of men aged over 65 years and supervised pelvic floor muscle training is recommended for men with stress urinary incontinence caused by prostatectomy (NICE 2010b). Men receiving physiotherapist-guided PFMT following radical prostatectomy have been shown to experience reduced urinary incontinence one year after surgery, compared with patients who exercised on their own (Overgård et al. 2008).

ANORECTAL DYSFUNCTION

Physiotherapists might also treat anorectal dysfunctions such as faecal incontinence and constipation.

Faecal incontinence

Involuntary loss of flatus or stool may affect 11–15% of the population (Macmillan et al. 2004), being most prevalent in older people in care. It can have a negative impact on quality of life (Madoff et al. 2004). Causes are similar to those listed under urogenital dysfunction – the most common being obstetric trauma. Leakage is described as passive (patient not aware) or urge (patient is aware of a need to defaecate, but is unable to control it). Arguably, of greatest significance is the consistency of stool. If liquid or very soft, it is far more difficult to contain. Ideally, the referring doctor will have addressed this issue.

Sphincters

The internal anal sphincter comprises smooth muscle, and provides 80–85% of resting pressure. The external anal sphincter (EAS) is under voluntary control and contracts in response to rectal distension to allow the individual to defer defaecation until an appropriate time. Puborectalis, part of the levator ani, also contributes to the continence mechanism, by forming an acute angle at the top of the anal canal (anorectal angle) to prevent the escape of rectal contents.

Physiotherapy

NICE (2007) recommends specialist management for sufferers whose symptoms are not relieved by simple advice. This management includes PFM training, biofeedback and electrical stimulation, all within the scope of the specialist physiotherapist. A recent review (Norton et al. 2010) recommends that further research is undertaken on these interventions to prove their effectiveness.

Constipation

Constipation has been reported to affect between 2% and 34% of adults, and a literature review (D'Hoore and Penninckx 2003) has suggested that obstructed defaecation (see below) specifically affects 7%. Constipation may be defined as infrequent bowel motions (fewer than three times a week) or the need to strain at defaecation. It may be subdivided into slow-transit and obstructed defaecation.

Slow-transit constipation may be idiopathic, but can be a result of the avoidance or postponement of defaecation by an individual, among other causes. It has been claimed (Gattuso and Kamm 1993) that up to 50% of patients with severe idiopathic constipation may have a history of childhood bereavement or of emotional, sexual or physical abuse.

Obstructed defaecation is an inability to evacuate the rectal contents and the cause may be anatomical or functional. The latter may be a failure to relax puborectalis (pelvic floor dyssynergia) or the EAS, or a lack of pelvic floor support (and resulting descent of the perineum) during defaecation (Markwell and Sapsford 1995). Causes are many, including obstetric trauma, pelvic surgery and prolonged straining. As with slow transit, researchers have suggested an association with sexual abuse (Leroi et al. 1996). Obstructed defaecation may be found in combination with slow transit (Hutchinson et al. 1993).

Physiotherapy

The aim of physiotherapy is to teach effective defaecation without straining (Markwell and Sapsford 1995) and should include correct positioning on the toilet, relaxation of puborectalis and the EAS, and optimum abdominal muscle action with an expulsive effort. Biofeedback might also be used and reviews of the literature (Bassotti et al. 2004; Chiarelli 2008) concluded that, although controlled trials were few and open to criticism, patients with obstructed defaecation owing to pelvic floor dyssynergia may benefit from biofeedback. Further research was recommended. If there is evidence of perineal descent or a rectocele, women might be advised to offer manual support to the perineum or digital support to the posterior vaginal wall, respectively, to facilitate defaecation. As there is an increase in colonic transit following meals, patients can be encouraged to attempt defaecation at these times. Abdominal massage may also be used and has been shown to decrease symptoms of constipation when used alongside medication (Lämås et al. 2009).

GYNAECOLOGICAL SURGERY

Gynaecological surgery may be necessary for many reasons, including the removal of benign or malignant tumours, urogynaecological conditions (if they fail to respond to physiotherapy) and the treatment of problems related to fertility or ectopic pregnancy. The physiotherapist should be aware of the indication for surgery.

Physiotherapy

The physiotherapist may be part of the multi-disciplinary team providing pre-operative advice and intervention postoperatively to ensure safe discharge from hospital and health education for life.

The enhanced recovery after surgery (ERAS) programme enables patients to recover from surgery and leave hospital sooner by minimising the stress responses on the body during surgery (DH 2010). Pre-operative intervention ensures that the patient is in the best possible condition for surgery and aware of self-help strategies postoperatively. Early postoperative rehabilitation will help to prevent venous thrombo-embolism (VTE), chest infection and digestive problems. ERAS recommends that rehabilitation services are available seven days a week.

Pre-operative care

Ergonomic advice for home and work, sports and leisure, postural care, abdominal and pelvic floor exercise and relaxation should be provided, preferably pre-operatively, and, ideally, before admission to hospital. This offers patients an opportunity to organise changes at home and inform employers how long they may be away from their usual work. Groups are good use of the physiotherapist's time and offer a forum for peer support and discussion. It is important to provide good written information (ACPWH 2010b; RCOG 2010).

Physiotherapists should know each woman's history, to be aware of any relevant information (e.g. diagnosis of malignancy). Special attention should be given to the psychosocial and psychosexual effects of gynaecological surgery on the patient and her family. Referral to other professional groups, for example a Macmillan nurse or a social worker, may be appropriate.

The physiotherapist might include:

- assessment of risk factors (e.g. respiratory conditions, VTE risks, impaired mobility, current medication, etc.) (NICE 2010c);

- identification of urogenital or anorectal dysfunction;
- identification of musculoskeletal problems;
- exercises and strategies to promote postoperative mobility and comfort;
- postoperative recovery plan, both before and after discharge from hospital;
- relaxation.

Postoperative care

The presence of an intravenous infusion, urinary catheter (suprapubic or urethral), patient-controlled analgesia and vaginal pack should be noted. The physiotherapist must be aware of the findings at surgery, and their potential physical and psychological impact on the patient. There may be concerns about relationships or resuming sexual activity, and referral to the relevant professional may be appropriate (Sacco Ezzell 1999). The physiotherapist should assess and advise on:

- respiratory function – especially for those with pre-existing problems;
- circulation – particularly for patients with limited mobility and/or risk of deep vein thrombosis;
- mobility, comfort and posture – including bed exercises and transfers, if appropriate;
- urination and defaecation – may include advice on position on the toilet and wound support;
- abdominal exercises to help reduce discomfort when moving, and ease and low back pain or flatus;
- pelvic floor muscles (PFMs) – the physiotherapist should confirm with the consultant any local policy for commencement of pelvic floor exercises (Jarvis et al. 2005). Women who have undergone surgery for prolapse and/or incontinence may already have poor connective tissue. They should be given appropriate advice – correct lifting techniques, avoidance of standing for long periods, awareness of the role of the pelvic floor and its interaction with transversus abdominis (Britnell et al. 2005);
- going home – discharge from hospital may be very early following some surgery, though it can be considerably longer. Convalescence is usually for 4–8 weeks, with a gradual increase in physical activity, but heavy lifting should be avoided for at least 6 weeks (RCOG 2010).

PELVIC PAIN

Acute and chronic pain

Pelvic pain in women has many varied sources, diagnoses and outcomes. It can be acute or chronic. Acute pain usually has an easily identifiable cause, such as dyspareunia (pain on sexual intercourse) caused by scar tissue following childbirth. Appropriate treatment will alleviate it.

When pain has lasted more than six months it is termed chronic. Although it may still have a well-defined cause (e.g. endometriosis), it can involve a much more complex interplay between biomechanical, behavioural, emotional and sociocultural influences, all of which need to be taken into account in treatment. Subsequently, clinicians advocate a multi-disciplinary integrated approach (Peters et al. 1991; Steege et al. 1998;). Good quality research is scarce for the multiple treatment modalities advocated (Stones et al. 2005); however, physiotherapy is included in the multi-disciplinary team (Fall et al. 2010).

Clinical note

The annual prevalence of chronic pelvic pain has been estimated at between 24.4 cases per 1000 women (Davies et al. 1992) and 38.3 per 1000 (Zondervan et al. 1999). The economic burden to the National Health Service (NHS) may be £158 million, with indirect costs of a further £24 million (Davies et al. 1992).

Pelvic pain has traditionally been ascribed (from a gynaecological view) to endometriosis, pelvic inflammatory disease, adhesions secondary to infection or surgery, and – more recently – pelvic venous congestion. The most important non-gynaecological cause is irritable bowel syndrome. Other causes include ilio-inguinal nerve entrapment, levator ani syndrome, coccydynia, interstitial cystitis, vulval vestibulitis/vulvodynia and musculoskeletal dysfunction.

Two studies showed that between 28% and 61% of patients never received a diagnosis (Mathias et al. 1996; Zondervan et al. 1999). This is highly significant as sufferers often feel that their pain has not been validated and will not be taken seriously until they have a diagnosis (Grace 1995). This may result in a fruitless round of clinicians.

Physiotherapy

The role of physiotherapy is to decrease pain, increase function and treat existing (and help prevent future) musculoskeletal dysfunction.

Following a detailed musculoskeletal examination, including assessment of the pelvic floor muscles, appropriate treatment modalities will be agreed, according to the needs of the patient and the experience of the clinician involved. Close liaison with other members of the MDT is essential to maximise the benefit of treatments which might include muscle imbalance work, muscle energy techniques, core stability exercises, pelvic floor

muscle rehabilitation, electrical stimulation, soft-tissue mobilisations, joint manipulation, breathing and relaxation techniques, heat/cold therapy, hydrotherapy, biofeedback and alternative therapies.

MENOPAUSE

Menopause is the cessation of menstruation and marks the end of a woman's reproductive years. It has significant implications for a woman's health and quality of life, so is of great relevance to the physiotherapist.

Most women experience the menopause between the ages of 40 and 58 years, with a median age of 52 years (NIH 2005). Therefore, with an average life expectancy of 81.9 years (ONS 2010a) women in the UK can expect to enjoy about 30 years of postmenopausal life.

Biochemical and metabolic changes following menopause may cause distressing symptoms that adversely affect quality of life. The perimenopause describes the period during which ovarian function declines, the menstrual cycle becomes erratic and a woman may experience symptoms of oestrogen depletion which include:

- vasomotor effects – hot flushes and night sweats;
- vaginal dryness which may result in painful intercourse;
- sleep disturbance.

There are other symptoms which have been attributed to the menopause, but which are yet to be substantiated conclusively by research (NIH 2005):

- mood changes – depression, anxiety, irritability;
- cognitive disturbances, e.g. forgetfulness;
- somatic symptoms, e.g. back pain, stiff and painful joints, tiredness;
- urinary incontinence;
- menorrhagia (heavy bleeding);
- sexual dysfunction;
- quality of life – either positive or negative effects.

Oestrogen, or oestrogen combined with progestogen, have been shown to have a positive effect on vasomotor symptoms, but studies have identified risks associated with their use (NIH 2005). Botanical products such as black cohosh and red clover are also taken by some women, but studies have not been the most robust so further research is advised (NIH 2005).

In relation to the physiotherapist, exercise, health education, acupuncture and breathing techniques may confer some benefit (NIH 2005; Roberts 2007).

BREAST CANCER

Breast cancer is the most common cancer affecting women in the UK with an average of 1 in 9 women being diagnosed. In 2007, 45,700 new cases of breast cancer cases were recorded and it causes over 12,000 deaths annually. Worldwide, 1.38 million women were diagnosed with breast cancer in 2008. Until 1999 it was the primary cause of cancer death in women (ONS 2010b).

Risk factors of breast cancer (van den Brandt et al. 2000; Dixon 2001; Clavel-Chapelon 2002; Megdal et al. 2005)

1. *Gender* – predominantly female.
2. *Age* – increases with age.
3. *Hormonal* – uninterrupted menstrual cycles, early menarche/late menopause.
4. *Reproduction* – no children or delay, and lack of breastfeeding.
5. *Exogenous hormones* – contraceptive pill/hormone replacement therapy.
6. *Family or personal history* of breast cancer.
7. *Other* – postmenopausal obesity; lack of physical activity; excess alcohol consumption; exposure to ionising radiation; fatty diet; working long night shifts; and higher socioeconomic status.

Initial diagnosis is made by mammography and ultrasound scan followed by needle aspiration or biopsy to establish the extent and stage of the disease, and its hormonal (oestrogen and progesterone) status. There are different types of breast cancer whose names relate to the position of the lesion, for example invasive ductal breast cancer, invasive lobular breast cancer or two very early types called ductal carcinoma *in situ* (DCIS) and lobular carcinoma in situ (LCIS). There are four stages (Table 27.1) of breast cancer with stages I–III being potentially curable.

The majority of patients will undergo surgery to remove the cancer, although management depends on the stage of the disease. Many women choose breast-sparing surgery (e.g. wide local excision, lumpectomy) which removes the breast lump and surrounding tissue. Scars are normally small and cosmetic appearance of the breast is good, with obvious psychological benefit. If the cancer is large or below the nipple a mastectomy is more appropriate. For some patients primary breast reconstruction is achieved at the same time as mastectomy, although it is usual to delay this until all oncology treatment has been completed. During surgery, axillary lymph nodes are excised to assess whether the cancer has spread beyond the breast. Historically, all the axillary lymph nodes were removed; however, newer techniques of sampling lymph nodes and sentinel node biopsy are now undertaken, which may reduce the future incidence of lymphoedema (Mansel et al. 2006).

Table 27.1 Stages of breast cancer

Stage I	Tumour up to 2 cm No lymph nodes affected No evidence of spread beyond the breast
Stage II	Tumour between 2 cm and 5 cm and/or: lymph nodes in armpit affected no evidence of spread beyond the armpit
Stage III	Tumour more than 5 cm Lymph nodes in armpit affected No evidence of spread beyond the armpit
Stage IV	Tumour of any size Lymph nodes in armpit often affected Cancer has spread to other parts of the body

Adjuvant therapies

Radiotherapy is frequently used, particularly after breast-sparing operations. Its main aim is to irradiate any remaining cancer cells.

Chemotherapy is mainly used for pre-menopausal women and when the cancer has spread to the axillary lymph nodes, or may be given as neo-adjuvant therapy to shrink the tumour facilitating more conservative surgery. Women who have oestrogen-sensitive tumours also receive some form of hormonal therapy, for example Tamoxifen or Arimidex, to block the cancer-promoting effect of oestrogen. Ongoing trials aim to establish the best regimes and therapies for pre- and postmenopausal women (Wishart et al. 2002; NICE 2009).

Physiotherapy

The physiotherapeutic approach to problem solving linked with an anatomical and physiological knowledge of the shoulder complex plays an important role in the rehabilitation of breast cancer patients. Physiotherapists promote independence and a return to normal activities, ultimately improving quality of life. Ward exercise groups can work well postoperatively with patients offering peer support.

Pre-operative care

- Assessment of any existing musculoskeletal shoulder or neck problems that may affect postoperative outcomes.
- Identification of potential risk factors for reduced mobility or lymphoedema.
- Information and advice, including a leaflet on postoperative exercise.
- Advice and strategies to reduce the occurrence of lymphoedema. Ideally, circumferential measurements should be taken as a baseline recording.

Postoperative care

Most patients have a drain for 2–5 days, although patients undergoing sentinel node biopsy and wide local incision may not. Physiotherapists must be aware of the psychological impact of a cancer diagnosis and altered body image that can occur with the loss of a breast.

- *Post-operative exercise* – daily general upper limb mobility exercises should be encouraged. Specific shoulders exercises are taught to prevent stiffness. Limiting shoulder movements below shoulder level for the first week postoperatively may result in a lower incidence of lymphoedema (Todd et al. 2008). An active programme of exercise and physiotherapy in the subsequent post-operative period indicates a greater recovery of shoulder mobility (Schultz et al. 1997; Clodius 2001; Box et al. 2002; Lacomba et al. 2010). Patients require good shoulder mobility to facilitate radiotherapy positioning. Post-radiotherapy patients should be encouraged to continue their shoulder mobility exercises as treatment can cause delayed-onset stiffness.

Pain

Postoperative pain is normally managed with medication. However, some patients suffer chronic pain which may be complex owing to the biomechanical, behavioural, emotional and sociocultural influences. TENS, acupuncture, muscle imbalance treatment, soft-tissue and joint mobilisations, relaxation techniques and alternative or cognitive behaviour therapies can help.

Prevention of lymphoedema (see lymphoedema section)

Patients are encouraged to moisturise the arm and breast area daily with emollient cream and avoid invasive medical interventions such as venepuncture or blood pressure measurement on the affected limb. Normal shoulder mobility and maintaining optimum BMI are also encouraged. Some lymphoedema clinics hold breast cancer prevention group sessions for patients undergoing breast cancer surgery and any axillary intervention.

Cording/axillary web syndrome

Tender, cord-like structures, thought to be a result of lymphangitis or lymphatic thrombosis, may arise from the chest wall to the axilla, sometimes extending down the arm itself. These cords restrict movement and cause significant pain. Gentle soft tissue massage and passive and active physiological movements initiate dispersion.

Scarring

Surgical scars may become adhered to underlying structures, for example fascia, resulting in tightness and discomfort in the axilla and chest wall. Patients are

encouraged to massage scars once healed to improve tissue pliability.

Postural advice

Removal of breast tissue causes a weight imbalance and may result in shoulder protraction. If not corrected, the pectoral muscles will shorten and tighten leading to muscle imbalance and pain.

Numbness

During axillary surgery cutaneous nerves are incised resulting in sensory loss in axilla, lateral chest wall and upper arm. It is not uncommon for patients to experience pain and paraesthesia as sensation recovers.

Return to function

Patients are encouraged to lead an active and normal life after having breast surgery. Care is always needed to prevent lymphoedema but patients should not be discouraged from activities of daily living that could improve quality of life.

LYMPHOEDEMA

ABC Definition

Lymphoedema is a chronic and progressive swelling caused by a low output failure of the lymphatic system, resulting in the development of a high-protein oedema in the tissues (Földi et al. 2003).

Lymphoedema is a form of chronic oedema, a persistent swelling owing to excess accumulation of fluid in the tissues (BLS 2001). It can affect people of all ages and occurs in limb/limbs, head and neck, trunk or genital area, but is more frequently seen in a unilateral upper limb after breast cancer, or bilateral lower limbs. Lymphoedema may not become apparent for some time after initial trauma or surgery and patients have a lifetime risk of developing it. Lymphoedema affects individuals physically, psychologically and socially and has a significant impact on quality of life and ability to undertake normal activities.

Other factors including chronic venous insufficiency, recurrent cellulitis, inflammation, dependency or obesity can result in chronic oedema. Provided there are no indications for medical intervention, a combination of physical therapies may be very beneficial.

Classification of lymphoedema

Primary lymphoedema is a result of either congenital abnormalities or the absence of lymph tissue. It usually presents at puberty, rarely soon after birth and, although less common than secondary lymphoedema, it is often more extreme with disability being present for much of a patient's life (Sitzia et al. 1998). It may be congenital and some forms of genetic mutation result in known subgroups such as Milroy disease and lymphoedema distichiasis. A new system of classification is being developed (Connell et al. 2010).

Secondary lymphoedema occurs owing to anatomical obliteration of part of the lymphatic system as a result of an extrinsic process (e.g. surgery or repeated infections) or as a consequence of functional deficiency (e.g. paralysis) (ISL 2003).

In the Western world, cancer or its treatment is a common cause of secondary lymphoedema. Surgical removal of part of the lymphatic system, or fibrotic changes subsequent to surgery or radiotherapy, result in a partial obstruction in lymphatic drainage and the development of lymphoedema in the affected limb and associated part of the trunk. Onset can occur many years after the initial cancer treatment (Stanton et al. 1996).

Prevalence and incidence of cancer-related lymphoedema

Evidence suggests a crude prevalence for chronic oedema of 1.3 per 1000 (Moffat et al. 2003). Incidence for people with breast cancer-related lymphoedema is estimated at around 25% of the population receiving treatment (Pain and Purushotham 2000). Evolving surgical techniques, such as sentinel lymph node biopsy, may ultimately reduce the incidence in patients not requiring full axillary clearance; however, there is currently little information to substantiate a population reduction (Armer 2005). Evidence around the incidence of lower limb and midline lymphoedema is limited although there are indications that risk of lymphoedema is at least equal to that for breast cancer patients (Keeley 2000).

The number of people in the UK with a non-cancer-related lymphoedema may outnumber those with a cancer-related lymphoedema by a ratio of 3:1 (Moffat et al. 2003). The prevalence of lymphoedema increases with age (5.4 per 1000 over the age of 65 years).

Onset and progression

Onset of lymphoedema is often marked by intermittent swelling and paraesthesia (Piller 1999). Initially, the oedema is soft and pitting and reduces on elevation (Keeley 2000). With time and recurrent skin infections, fibrotic changes occur both in the skin and subcutis, with a progressive swelling and distortion of shape. Skin and tissues become thickened with enhanced skin folds and increased adiposity, and the swelling becomes largely non-pitting. Limb size increases and is sometimes accompanied by a distortion in shape. Movement becomes

restricted as the limb becomes increasingly heavy and uncomfortable.

Lymphoedema sufferers are predisposed to recurrent acute infections, often referred to as cellulitis (Browse et al. 2003). Signs and symptoms of these apparent skin infections include acute pain, swelling, anorexia, fevers, vomiting and rigors.

The psychosocial impact of living with lymphoedema can be profound, resulting in embarrassment, loss of self-esteem and increased feelings of anxiety and depression. Patients also experience impaired physical mobility and pain (Tobin et al. 1993; Woods et al. 1995; Williams et al. 2004).

Although no curative treatment is available, a combination of physical therapies, delivered by a specialist clinician, is used to control and reduce the complications associated with lymphoedema. Ongoing reviews are usually required.

Physiotherapy

Treatment is based around four key areas (Földi et al. 2003; Jenns 2000).

- *Skin care* – daily washing and application of emollient cream, and provision of information and advice on skin care to minimise the risk of cellulitis. Advice includes protection of the skin from cuts, insect bites and burns (e.g. when gardening, sewing or sunbathing). Whenever possible, medical interventions, such as venepuncture or taking blood pressure, should be undertaken on the unaffected side. In lower limb lymphoedema, identification and treatment of fungal infections is paramount.
- *Compression garments/multilayer lymphoedema bandaging (MLLB)*. MLLB is used in combination with lymphatic massage to reverse complications, such as severe swelling or distorted limb shape, prior to the introduction of compression garments (Badger et al. 2000). Although bandaging is used in the initial treatment of some patients with complex needs, compression garments are a mainstay of treatment in many patients to limit and control swelling. In breast cancer, a garment such as a glove, sleeve or combined glove and sleeve may be used depending on the extent of swelling. Garments are usually worn during the day, especially for exercise and when working. Pneumatic compression therapy (e.g. Flowtron) is a method of treatment traditionally used, but there is concern that continued use may increase the risk of midline swelling (Boris and Lasinski 1989; Casley-Smith and Casley-Smith 1997). Newer pneumatic therapy machines based on the principle of manual lymphatic drainage massage are now available and are reducing the risks.
- *Dynamic exercise* is beneficial, as lymphatic drainage is enhanced by the muscle pump effect, particularly when a compression garment is worn. Gentle stretching exercises also help to maintain or improve range of movement and to facilitate good posture (Harris et al. 2001). Excessive exercise can increase the lymphatic load and result in further swelling, so patients are warned to introduce any new activity gradually and with caution.
- *Manual lymphatic drainage (MLD)* is characterised by the pressure and sequence of the technique, designed to stimulate drainage through the functioning lymphatics. This is usually the only method of care available to treat midline oedema (i.e. face, genitals and trunk). Simple lymphatic drainage (SLD) (Williams et al. 2002) is taught to patients.

PSYCHOSEXUAL ISSUES

Throughout this chapter contributors have described a range of life stages, conditions, dysfunctions, pathologies and interventions which have not only a physical, but also a psychological, impact on the women concerned. Many also affect the psychological and emotional aspect of sex – i.e. are psychosexual concerns – of which the physiotherapist should be aware.

Pregnancy and childbirth

Although there is no evidence that sex during pregnancy could be harmful (NICE 2008b), sexual activity may decrease during pregnancy (Pauleta et al. 2010; Serati et al. 2010) and this can continue postnatally for various reasons, the most common being pain (Abdool et al 2009).

Menopause

Vaginal dryness may result in painful intercourse and women of menopausal age may also report sexual dysfunction associated with altered libido, arousal and other aspects of sexuality (NIH 2005).

Gynaecological surgery

Although women are advised that sexual intercourse can normally be resumed 4–6 weeks after surgery (RCOG 2010), some report sexual problems which might relate to loss of desire, lubrication, sensitivity, elasticity, capacity or pain, and which are possibly more prevalent in women who have undergone surgery for malignancy (Aerts et al. 2009).

Breast cancer

Women may suffer poorer body image, anxiety/depression, side effects, such as vaginal irritation and lowered libido,

related to their treatment, affecting their sexuality (Sheppard and Ely 2008).

Urinary incontinence

One third of women who experience urinary incontinence may leak with intercourse, which has a significant impact on their reported quality of life (Pons and Clota 2008).

The above list is by no means exhaustive.

The physiotherapist is one of the few professionals who may include a vaginal examination as part of her/his assessment. This is a very intimate procedure and a time of self-awareness for the patient, which should be approached with sensitivity and care. It may be the occasion when past feelings come to light, for example previous sexual abuse. The physiotherapist may be the first person with whom the woman shares some of her concerns. Psychosexual counselling may not be a physiotherapy core skill, but clinicians in women's health should know what services are available locally and how to access them. In addition, they may wish to access postgraduate training to enhance their skill set.

Weblinks

Association of Chartered Physiotherapists in Women's Health (ACPWH): http://acpwh.csp.org.uk/
International Organization of Physical Therapists in Women's Health (IOPTWH): www.ioptwh.org
Bladder and Bowel Foundation: www.bladderandbowelfoundation.org
Chartered Physiotherapists Promoting Continence: www.cppc.org.uk
International Continence Society: www.icsoffice.org
International Urogynecological Association: www.iuga.org
The British Menopause Society: www.thebms.org.uk
Menopause Matters: www.menopausematters.co.uk
Cancer Research UK: www.cancerresearchuk.org
Breast Cancer Care and Research Fund: www.breastcancercare.org
Institute of Psychosexual Medicine: www.ipm.org.uk

FURTHER READING

Physiotherapy in women's health

Mantle, J., Haslam, J., Barton, S. (Eds.), 2004. Physiotherapy in Obstetrics and Gynaecology, second ed. Butterworth-Heinemann, Oxford.

Irion, J.M., Irion, G.L. (Eds.), 2010. Women's Health in Physical Therapy. Lippincott, Williams & Wilkins, Baltimore.

Sapsford, R., Bullock-Saxton, J., Markwell, S., 1998. Women's Health: A Textbook for Physiotherapists. WB Saunders, London.

Pregnancy and childbirth

Fraser, D., Cooper, M. (Eds.), 2010. Myles Textbook for Midwives, fifteenth ed. Churchill Livingstone, Edinburgh.

Macdonald, S., Magill-Cuerden, J. (Eds.), 2011. Mayes Midwifery: A Textbook for Midwives, fourteenth ed. Baillière Tindall, London.

Antenatal classes, advice and exercises

Brayshaw, E., 2003. Exercises for Pregnancy and Childbirth: A Guide for Educators. Books for Midwives, Oxford.

Nolan, M.L., Foster, J., 2004. Birth and Parenting Skills: New Directions in Antenatal Education. Churchill Livingstone, Edinburgh.

Schott, J., Priest, J., 2002. Leading Antenatal Classes: A Practical Guide. Books for Midwives Press, Oxford.

Pelvic floor muscle dysfunction and rehabilitation

Bø, K., Berghmans, B., Mørkved, S., et al. (Eds.), 2007. Evidence-based Physical Therapy for the Pelvic Floor. Elsevier, Edinburgh.

Haslam, J., Laycock, J. (Eds.), 2008. Therapeutic Management of Incontinence and Pelvic Pain, second ed. Springer-Verlag, London.

Breast cancer and lymphoedema

Mortimer, P., Todd, J., 2007. Lymphoedema: Advice on Self-management and Treatment, third ed. Beaconsfield Publishers Ltd, Beaconsfield.

Psychosexual issues

Schachter, C.L., Stalker, C.A., Teram, E., et al., 2009. Handbook on Sensitive Practice for Health Care Practitioners: Lessons from Adult Survivors of Childhood Sexual Abuse. Public Health Agency of Canada, Ottawa.

REFERENCES

Abdool, Z., Thakar, R., Sultan, A.H., 2009. Postpartum female sexual function: a review. Eur J Obstet Gynecol Reprod Biol (in press) doi:10.1016/j.ejogrb.2009.04.014.

Abitol, M.M., 1997. Quadrupedalism, bipedalism, and human pregnancy. In: Vleeming, A., Mooney, V., Dorman, T. (Eds.), Movement, Stability and Low Back Pain. Churchill Livingstone, Edinburgh.

Abramson, D., Roberts, S.M., Wilson, P.D., 1934. Relaxation of the pelvic joints in pregnancy. Surg Gynaecol Obstet 58, 595–613.

ACOG (American College of Obstetricians and Gynecologists), 2002. Exercise during pregnancy and the postpartum period. Obstet Gynecol 99, 171–173.

ACPWH (Association of Chartered Physiotherapists in Women's Health), 2007. Guidance of the safe use of transcutaneous electrical nerve stimulation (TENS) for musculoskeletal pain during pregnancy. ACPWH, London; http://acpwh.csp.org.uk/publications/acpwh-guidance-safe-use-transcutaneous-electrical-nerve-stimulation-tens-musculo, accessed October 2012.

ACPWH (Association of Chartered Physiotherapists in Women's Health), 2009. ACPWH, London; http://www.csp.org.uk/sites/files/csp/secure/acpwh-pelvicfloor_0.pdf, accessed October 2012.

ACPWH (Association of Chartered Physiotherapists in Women's Health), 2010a. Pregnancy-related pelvic girdle pain. Guidance for health professionals. ACPWH, London; http://www.csp.org.uk/sites/files/csp/secure/acpwh-pelvicfloor_0.pdf, accessed October 2012.

ACPWH (Association of Chartered Physiotherapists in Women's Health), 2010b. Fit for Life. Advice and exercise following gynaecological surgery. ACPWH, London; http://www.csp.org.uk/sites/files/csp/secure/acpwh-pgphp_0.pdf, accessed October 2012.

ACPWH (Association of Chartered Physiotherapists in Women's Health), 2010c. Aquanatal Guidelines: Guidance on antenatal and postnatal exercises in water. ACPWH, London; http://www.csp.org.uk/sites/files/csp/secure/acpwh-aquanatal.pdf, accessed October 2012.

Aerts, L., Enzlin, P., Verhaeghe, J., et al., 2009. Sexual and psychological functioning after pelvic surgery for gynaecological cancer. Eur J Gynaecol Oncol 30 (6), 652–656.

Albert, H., Godskesen, M., Westergaard, J.G., et al., 1997. Circulating levels of relaxin are normal in pregnant women with pelvic pain. Eur J Obstet Gynecol Reprod Biol 74, 19–22.

Albert, H., Godskesen, M., Westergaard, J., 2001. Prognosis in four syndromes of pregnancy-related pelvic pain. Acta Obstet Gynecol Scand 80, 505–510.

Allen, R.E., Hosker, G.L., Smith, A.R., et al., 1990. Pelvic floor damage and childbirth: a neurophysiological study. Br J Obstet Gynaecol 97, 770–779.

Anim-Somuah, M., Smyth, R.M.D., Howell, C.J., 2005. Epidural versus non-epidural or no analgesia in labour. Cochrane Database Syst Rev (4): CD000331.

Arena, B., Maffulli, N., 2002. Exercise in pregnancy: how safe is it? Sports Med Arthrosc 10 (1), 15–22.

Armer, J.M., 2005. The problem of post-breast cancer lymphedema: impact and measurement issues. Cancer Invest 23 (1), 76–83.

Artal, R., O'Toole, M., 2003. Guidelines of the American College of Obstetricians and Gynecologists for exercises during pregnancy and the postpartum period. Br J Sports Med 37, 6–12.

Avery, N.D., Stocking, K.D., Tranmer, J.E., et al., 1999. Foetal responses to maternal strength conditioning exercises in late gestation. Can J Appl Physiol 24 (4), 362–376.

Badger, C.M.A., Peacock, J.L., Mortimer, P.S., 2000. A randomized controlled parallel group clinical trial comparing multilayer bandaging followed by hosiery versus hosiery alone in the treatment of patients with lymphedema of the limb. Cancer 88 (12), 2832–2837.

Barber, M.D., Bremer, R.E., Thor, K.B., et al., 2002. Innervation of the female levator ani muscles. Am J Obstet Gynecol 187, 64–71.

Barton, S., 2004. The postnatal period. In: Mantle, J., Haslam, J., Barton, S. (Eds.), Physiotherapy in Obstetrics and Gynaecology, second ed. Butterworth-Heinemann, London, pp. 205–247.

Bassotti, G., Chistolini, F., Sietchiping-Nzepa, F., et al., 2004. Biofeedback for pelvic floor dysfunction in constipation. BMJ 328 (7436), 393–396.

Bastiaanssen, J.M., de Bie, R.A., Bastiaenen, C.H., et al., 2005. A historical perspective on pregnancy-related low back and/or pelvic girdle pain. Eur J Obstet Gynecol Reprod Biol 120, 3–14.

Berg, G., Hammar, M., Moller-Nielsen, J., 1988. Low back pain during pregnancy. Obstet Gynaecol 71, 71–75.

Betts, D., 2003. The Essential Guide to Acupuncture in Pregnancy and Childbirth. Eastland Press Inc., Seattle.

Björklund, K., Nordström, M.L., Bergström, S., 1999. Sonographic assessment of symphyseal joint distention during pregnancy and post partum with special reference to pelvic pain. Acta Obstet Gynecol Scand 78, 125–130.

Björklund, K., Bergström, S., Nordström, M.L., et al., 2000a. Symphyseal distention in relation to serum relaxin levels and pelvic pain in pregnancy. Acta Obstet Gynecol Scand 79, 269–275.

Björklund, K., Nordström, M.L., Odlind, V., 2000b. Combined oral contraceptives do not increase the risk of back and pelvic pain during pregnancy or after delivery. Acta Obstet Gynecol Scand 79, 979–983.

BLS (British Lymphology Society), 2001. Clinical Definitions, www.lymphoedema.org/bls.

Bø, K., 2004. Pelvic floor muscle training is effective in treatment of female stress urinary incontinence but how does it work? Int Urogynecol J Pelvic Floor Dysfunct 15, 76–84.

Bø, K., 2006. Can pelvic floor muscle training prevent and treat pelvic organ prolapse? Acta Obstet Gynecol Scand 85 (3), 263–268.

Bø, K., 2007. Pelvic floor muscle training for stress urinary incontinence. In: Bø, K., Berghmans, B., Mørkved, S., et al. (Eds.), Evidence-based Physical Therapy for the Pelvic Floor. Elsevier, Edinburgh, pp. 171–187.

Bø, K., Mørkved, S., 2007. Pelvic floor and exercise science. In: Bø, K., Berghmans, B., Mørkved, S., et al. (Eds.), Evidence-based physical therapy for the pelvic floor. Elsevier, Edinburgh, pp. 113–132.

Bø, K., Mørkved, S., Frawley, H., et al., 2009. Evidence for benefit of transversus abdominis training alone or in combination with pelvic floor muscle training to treat female urinary incontinence: a systematic review. Neurourol Urodyn 28 (5), 368–373.

Boissonnault, J.S., Blaschak, M.J., 1988. Incidence of diastasis recti abdominis during the childbearing year. Phys Ther 86, 1082–1086.

Boris, M., Lasinski, B., 1989. The risk of genital oedema after external pump compression for lower limb lymphoedema. Lymphology 31, 15–20.

Box, R.C., Reul-Hirche, H.M., Bullock-Saxton, J.E., et al., 2002. Shoulder movement after breast cancer surgery: results of a randomised controlled study of post operative physiotherapy. Breast Cancer Res Treat 75, 35–50.

Brækken, I.H., Majida, M., Ellström Engh, M., et al., 2009. Pelvic floor function is independently associated with pelvic organ prolapse. BJOG 116 (13), 1706–1714.

Brækken, I.H., Majida, M., Ellström Engh, M., et al., 2010. Can pelvic floor muscle training reverse pelvic organ prolapse and reduce prolapse symptoms? An assessor-blinded, randomized controlled trial. Am J Obstet Gynecol 203 (170), e1–e37.

Breen, T.W., Ransil, B.J., Groves, P.A., et al., 1994. Factors associated with back pain after childbirth. Anesthesiology 81 (1), 29–34.

Britnell, S.J., Cole, J.V., Isherwood, L., et al., 2005. Postural health in women: the role of physiotherapy. Canadian Physiotherapy Association, www.sogc.org.

Browse, N., Burnand, K., Mortimer, P., 2003. Diseases of the Lymphatics. Edward Arnold, London.

Bullock-Saxton, J.E., 1991. Changes in posture associated with pregnancy and the early postnatal period measured in standing. Physiother Theory Pract 7, 103–109.

Bullock-Saxton, J., 1999. Musculoskeletal changes in the perinatal period. In: Sapsford, R., Bullock-Saxton, J., Markwell, S. (Eds.), Women's Health. Harcourt Brace and Company Limited, London, pp. 134–161.

Bump, R., Hurt, W.G., Fantl, A., et al., 1991. Assessment of Kegel pelvic muscle exercise performance after brief verbal instruction. Am J Obstet Gynecol 165, 322–329.

Byrom, S., Edwards, G., Bick, D., 2010. Essential midwifery practice: postnatal care. Wiley-Blackwell, Hoboken, NJ.

Capiello, G.A., Oliver, B.C., 1995. Rupture of symphysis pubis caused by forceful and excessive abduction of the thighs with labor epidural anesthesia. J Fla Med Assoc 82 (4), 261–263.

Carroli, G., Belizan, J., 2009. Episiotomy for vaginal birth. Cochrane Database Syst Rev (1): CD000081.

Casley-Smith, J.R., Casley-Smith, J.R., 1997. Modern Treatment for Lymphoedema, fifth ed. Terrance, Adelaide, pp. 288–301.

Castro, R.A., Arruda, R.M., Zanetti, M.R.D., et al., 2008. Single-blind, randomized controlled trial of pelvic floor muscle training, electrical stimulation, vaginal cones, and no active treatment in the management of stress urinary incontinence. Clinics 63 (4), 465–472.

Chapman, V., Charles, C., 2009. The midwife's labour and birth handbook. Blackwell Publishing, London.

Chiarelli, P., 2008. Systematic review: the management of constipation using physical therapies including biofeedback. Aust NZ Continence J 14 (1), 6–13.

Chiarelli, P., Campbell, E., 1997. Incontinence during pregnancy: prevalence and opportunities for continence promotion. Aust NZ J Obstet Gynaecol 37 (1), 66–73.

Chiarelli, P., Cockburn, J., 2002. Promoting urinary continence in women after delivery: randomised controlled trial. BMJ 324 (7348), 1241–1243.

Chiarello, C., 2005. The effects of an exercise program on diastasis recti abdominis in pregnant women. J Women Health Phys Ther 29 (1), 11–16.

Clapp, J.F., 2000. Exercise during pregnancy: a clinical update. Clin Sports Med 19 (2), 273–286.

Clapp, J.F., Kim, H., Burciu, B., et al., 2000. Beginning regular exercise in early pregnancy: effect on foetoplacental growth. Am J Obstet Gynecol 183, 1484–1488.

Clavel-Chapelon, F., 2002. Differential effects of reproductive factors on the risk of pre- and postmenopausal breast cancer. Results from a large cohort of French women. Br J Cancer 86 (5), 723–727.

Clodius, L., 2001. Minimizing secondary arm lymphedema from axillary dissection. Lymphology 34, 106–110.

CMACE (Centre for maternal and child enquiries), RCOG (Royal College of Obstetricians and Gynaecologists), 2010. Management of women with obesity in pregnancy. CMACE, London.

Coldron, Y., 2005. Mind the gap – symphysis pubis dysfunction revisited. J Assoc Chart Physiother Wom Health 96, 3–15.

Coldron, Y., 2006. Characteristics of rectus abdominis during the first postnatal year. In: Characteristics of Abdominal and Paraspinal Muscles in Postnatal Women. St George's, University of London (unpublished PhD thesis), pp. 185–225.

Coldron, Y., Crothers, E., Haslam, J., et al., 2007. ACPWH guidance of the safe use of transcutaneous electrical nerve stimulation (TENS) for musculoskeletal pain during pregnancy, www.acpwh.org.uk.

Coldron, Y., Stokes, M.J., Cook, K., 2008. Postpartum characteristics of rectus abdominis on ultrasound imaging. Man Ther 13 (2), 112–121.

Collis, R., 2000. Pain and anaesthesia. In: Kean, L., Baker, P., Edelstone, D. (Eds.), Best Practice in Labour Ward Management. Saunders, London.

COMET (Comparative Obstetric Mobile Epidural Trial) Study Group UK, 2001. Effect of low dose mobile versus traditional epidural techniques on mode of delivery: a randomised control trial. Lancet 358, 19–23.

Connell, F., Brice, G., Keeley, V., et al., 2010. A new classification system for primary lymphatic dysplasias based on phenotype. Clinical Genetics 77 (5), 438–452.

Cresswell, A.G., Grundstrom, H., Thortensson, A., 1992. Observations on intra-abdominal pressure and patterns of abdominal intramuscular activity in man. Acta Physiol Scand 144, 409–418.

Daly, J.M., Frame, P.S., Rapoza, P.A., 1991. Sacroiliac subluxation: a common, treatable cause of low-back pain in pregnancy. Fam Pract Res J 11, 149–159.

Damen, L., Buyruk, H.M., Guler-Uysal, F., et al., 2001. Pelvic pain during pregnancy is associated with asymmetric laxity of the sacroiliac joints. Acta Obstet Gynecol Scand 80, 1019–1024.

Damen, L., Spoor, C.W., Snijders, C.J., et al., 2002. Does a pelvic belt influence sacroiliac joint laxity? Clin Biomech 17, 495–498.

Davies, L., Gangar, K.F., Drummond, M., et al., 1992. The economic burden of intractable gynaecological pain. J Obstet Gynaecol 12 (Suppl. 2), S54–S56.

De Jonge, A., Lagro-Jansen, A., 2004. Birthing positions: a qualitative study into the views of women about various birthing positions. J Psychosom Obstet Gynaecol 25, 47–55.

De Lancey, J.L.O., 2001. Anatomy. In: Cardozo, L., Staskin, D. (Eds.), Textbook of Female Urology and Urogynaecology. Isis Medical Media, London, pp. 112–124.

Depledge, J., McNair, P.J., Keal-Smith, C., et al., 2005. Management of symphysis pubis dysfunction during pregnancy using exercise and pelvic support belts. Phys Ther 85, 1290–1300.

DH (Department of Health), 2004. National Service Framework for Children, Young People and Maternity Services. DH, London.

DH (Department of Health), 2010. Delivering Enhanced Recovery: Helping Patients to Get Better Sooner After Surgery. DH, London.

D'Hoore, A., Penninckx, F., 2003. Obstructed defecation. Colorectal Dis 5 (4), 280–287.

Diakow, P.R., Gadsby, T.A., Gadsby, J.B., et al., 1991. Back pain during pregnancy and labor. J Manipulative Physiol Ther 14, 116–118.

Dixon, J.M., 2001. Hormone replacement therapy and the breast. BMJ 323 (7326), 1381–1382.

Dolan, L.M., Hosker, G.L., Mallett, V.T., et al., 2003. Stress incontinence and pelvic floor neurophysiology 15 years after the first delivery. BJOG 110 (12), 1107–1114.

Downe, S., 2010. The transition and the second stage of labour: physiology and the role of the midwife. In: Fraser, D., Cooper, M.M. (Eds.), Textbook for Midwives, fifteenth ed. Churchill Livingstone, Edinburgh.

Dumoulin, C., Hay-Smith, J., 2010. Pelvic floor muscle training versus no treatment, or inactive control treatments, for urinary incontinence in women. Cochrane Database Syst Rev (1): CD005654.

Eason, E., Labrecque, M., Marcoux, S., et al., 2002. Anal incontinence after childbirth. CMAJ 166 (3), 326–330.

Elden, H., Ladfors, L., Olsen, M.F., et al., 2005. Effects of acupuncture and stabilising exercises as adjunct to standard treatment in pregnant women with pelvic girdle pain: randomised single blind controlled trial. BMJ 330, 761.

Elden, H., Ostgaard, H.C., Olsen, M.F., et al., 2008. Treatments of pelvic girdle pain in pregnant women: adverse effects of standard treatment, acupuncture and stabilising exercises on the pregnancy, mother, delivery and the fetus/neonate. BMC Compliment Altern Med 8 (34), 1–13.

Evans, G.M., 2002. Aquanatal exercise. In: Campion, M.R., Pattmann, J. (Eds.), Hydrotherapy: Principles and Practice, second ed. Butterworth-Heinemann, Oxford.

Fall, M., Baranowski, A.P., Elneil, S., et al., 2010. Guidelines on chronic pelvic pain. Euro Urol 57 (1), 35–48.

Fast, A., Shapiro, D., Ducommun, E.J., 1987. Low back pain in pregnancy. Spine 12, 368–371.

Földi, E., Földi, M., Kubik, S. (Eds.), 2003. Textbook of Lymphology. Urban & Fischer, Munich.

Forrester, M., 2003. Low back pain in pregnancy. Acunpunct Med 21, 36–41.

Foti, T., Davids, J.R., Bagley, A., 2000. A biomechanical analysis of gait during pregnancy. J Bone Joint Surg Am 82, 625–632.

Francis, W.J.A., 1960. Disturbances of bladder function in relation to pregnancy. J Obstet Gynaecol Br Emp 67, 353–366.

Freeman, R.M., 2002. The effect of pregnancy on the lower urinary tract and pelvic floor. In: MacLean, A.B., Cardozo, L. (Eds.), Incontinence in Women. RCOG, London, pp. 331–345.

Fry, D., 1999. Perinatal symphysis pubis dysfunction: a review of the literature. J Assoc Chart Physiother Wom Health 85, 11–18.

Gameiro, M.O., Moreira, E.H., Gameiro, F.O., et al., 2010. Vaginal weight cone versus assisted pelvic floor muscle training in the treatment of female urinary incontinence. A prospective, single-blind, randomized trial. Int Urogynecol J 21 (4), 395–399.

Gattuso, J., Kamm, M., 1993. The management of constipation in adults. Aliment Pharmacol Ther 7, 487–500.

Gherman, R.B., Ouzounian, J.G., Incerpi, M.H., et al., 1998. Symphyseal separation and transient femoral neuropathy associated with the McRoberts maneuver. Am J Obstet Gynecol 178 (3), 609–610.

Gilleard, W.L., Brown, J.M.M., 1996. Structure and function of the abdominal muscles during pregnancy and the immediate post birth period. Phys Ther 7, 750–762.

Gilleard, W.L., Crosbie, J., Smith, R., 2002. Static trunk posture in sitting

and standing during pregnancy and early postpartum. Arch Phys Med Rehabil 83, 1739–1744.

Golightly, R., 1982. Pelvic arthropathy in pregnancy and the puerperium. Physiotherapy 68, 216–220.

Goode, P.S., Burgio, K.L., Locher, J.L., et al., 2003. Effect of behavioral training with or without pelvic floor electrical stimulation on stress incontinence in women. JAMA 304 (24), 2667–2759.

Grace, V.M., 1995. Problems of communication, diagnosis, and treatment experienced by women using the New Zealand health services for chronic pelvic pain: a quantitative analysis. Health Care Women Int 16 (6), 521–535.

Guerreiro da Silva, J.B., Nakamura, M.U., Cordeiro, J.A., et al., 2004. Acupuncture for low back pain in pregnancy – a prospective, quasi-randomised, controlled study. Acupunct Med 22, 60–67.

Gupta, J.K., Hofmeyr, G.J., Smyth, R., 2004. Position in the second stage of labour for women without epidural anaesthesia. Cochrane Database Syst Rev (1): CD002006.

Gutke, A., Ostgaard, H.C., Oberg, B., 2006. Pelvic girdle pain and lumbar pain in pregnancy: a cohort study of the consequences in terms of health and functioning. Spine 31, E149–E155.

Hagen, R., 1974. Pelvic girdle relaxation from an orthopaedic point of view. Acta Orthop Scand 45, 550–563.

Hamilton, A., 2010. Assisted births. In: Fraser, D., Cooper, M.M. (Eds.), Textbook for Midwives, fifteenth ed. Churchill Livingstone, Edinburgh.

Hannestad, Y.S., Lie, R.T., Rortveit, G., et al., 2004. Familial risk of urinary incontinence in women: population based cross sectional study. BMJ 329, 889–891.

Hansen, A., Jensen, D.V., Wormslev, M., et al., 1999. Symptom giving pelvic girdle relaxation in pregnancy. II: Symptoms and clinical signs. Acta Obstet Gynecol Scand 78, 111–115.

Harris, S.R., Hugi, M.R., Olivotto, I.A., et al., 2001. Clinical practice guidelines for the care and treatment of breast cancer: 11. Lymphedema. CMAJ 164 (2), 191–199.

Harrison, R., Bulstrode, F., 1987. Percentage weight bearing during partial immersion in a hydrotherapy pool. Physiother Pract 3, 60–63.

Haslam, J., 2004a. Anatomy. In: Mantle, J., Haslam, J., Barton, S. (Eds.), Physiotherapy in Obstetrics and Gynaecology, second ed. Butterworth-Heinemann, London, pp. 1–25.

Haslam, J., 2004b. Physiology of pregnancy. In: Mantle, J., Haslam, J., Barton, S. (Eds.), Physiotherapy in Obstetrics and Gynaecology, second ed. Butterworth-Heinemann, London, pp. 27–52.

Heath, T., Gherman, R.B., 1999. Symphiseal separation, sacroiliac joint dislocation and transient lateral femoral cutaneous neuropathy associated with McRoberts maneuver. A case report. J Reprod Med 44 (10), 902–904.

Hefferman, A.E., 2000. Exercise and pregnancy in primary care. Nurse Pract 25 (3), 42, 49, 53–56.

Heiberg, E., Aarseth, S.P., 1997. Epidemiology of pelvic pain and low back pain in pregnant women. In: Vleeming, A., Mooney, V., Dorman, T., et al. (Eds.), Movement, Stability and Low Back Pain. Churchill Livingstone, Edinburgh, pp. 405–410.

Hodges, P.W., 1999. Is there a role for transversus abdominis in lumbo-pelvic stability? Manual Ther 4 (2), 74–86.

Holtedahl, K., Hunskaar, S., 1998. Prevalence, 1-year incidence and factors associated with urinary incontinence: a population based study of women 50–74 years of age in primary care. Maturitas 28 (3), 205–211.

Howell, C.J., Dean, T., Lucking, L., et al., 2002. Randomised study of long term outcome after epidural versus non-epidural analgesia during labour. BMJ 325, 357.

Hutchinson, R., Notghi, A., Mostafa, A.B., et al., 1993. Audit of transit abnormality in chronic idiopathic constipation. Gut 34 (Suppl. 4), W49.

ICS (International Continence Society), 2002. Standardisation of Terminology in Lower Urinary Tract Function. ICS, London.

Imamura, M., Abrams, P., Bain, C., et al., 2010. Systematic review and economic modelling of the cost-effectiveness of non-surgical treatments for women with stress urinary incontinence. Health Technol Assess 14 (40), 1–506.

ISL (International Society of Lymphology), 2003. The diagnosis and treatment of peripheral lymphedema. Lymphology 36 (2), 84–91.

Jarvis, S.K., Hallam, T.K., Lujic, S., et al., 2005. Peri-operative physiotherapy improves outcomes for women undergoing incontinence and or prolapse surgery: results of a randomised controlled trial. Aust NZ J Obstet Gynaecol 45 (4), 300–303.

Jenns, K., 2000. Management strategies. In: Twycross, R., Jenns, K., Todd, J. (Eds.), Lymphoedema. Radcliffe Medical, Oxford, pp. 98–117.

Kabeyama, K., Miyoshi, M., 2001. Longitudinal study of the intensity of memorised labour pain. Int J Nurs Pract 7, 46–53.

Katz, V.L., McMurray, R.G., Cefalo, R.C., 1991. Aquatic exercise during pregnancy. In: Mittelmark, R.A., Wiswell, R.A., Drinkwater, B.L. (Eds.), Exercise in Pregnancy, second ed. Williams & Wilkins, pp. 271–278.

Keeley, V., 2000. Classification of lymphoedema. In: Twycross, R., Jenns, K., Todd, J. (Eds.), Lymphoedema. Radcliffe Medical, Oxford, pp. 22–44.

Kendall, F.P., McCreary, E.K., Provance, P.G., et al., 2005. Muscles: Testing and Function with Posture and Pain, fifth ed. Lippincott, Williams and Wilkins, Baltimore, MD.

Khadilkar, A., Odebiyi, D.O., Brosseau, L., et al., 2008. Transcutaneous electrical nerve stimulation (TENS) versus placebo for chronic low back pain. Cochrane Database Syst Rev (4): CD003008.

Kharrazi, F.D., Rodgers, W.B., Kennedy, J.G., Lhowe, D.W., 1997. Parturition-induced pelvic dislocation: A report of four cases. J Orthop Trauma 11 (4), 277–281.

Koltyn, K.F., Schultes, S.S., 1997. Psychological effects of an aerobic exercise session and a rest session following pregnancy. J Sports Med Phys Fitness 37 (4), 287–291.

Kristiansson, P., Svardsudd, K., von Schoultz, B., 1996a. Back pain during pregnancy: a prospective study. Spine 21, 702–709.

Kristiansson, P., Svardsudd, K., von Schoultz, B., 1996b. Serum relaxin, symphyseal pain, and back pain during pregnancy. Am J Obstet Gynecol 175, 1342–1347.

Kvorning, N., Holmberg, C., Grennert, L., et al., 2004. Acupuncture relieves pelvic and low-back pain in late pregnancy. Acta Obstet Gynecol Scand 83, 246–250.

Lacomba, M., Sánchez, M., Goñi, A., et al., 2010. Effectiveness of early physiotherapy to prevent lymphoedema after surgery for breast cancer: randomised, single blinded, clinical trial. BMJ 340, b5396.

Lämås, K., Lindholm, L., Stenlund, H., et al., 2009. Effects of abdominal massage in management of constipation – a randomized controlled trial. Int J Nurs Stud 46 (6), 759–767.

Larsen, E.C., Wilken-Jensen, C., Hansen, A., et al., 1999. Symptom giving pelvic girdle relaxation in pregnancy. I: Prevalence and risk factors. Acta Obstet Gynecol Scand 78, 105–110.

Laycock, J., 2003. Clinical Guidelines for the Physiotherapy Management of Females Aged 16–65 with Stress Urinary Incontinence. Chartered Society of Physiotherapy, London.

Lee, D., 1999. Biomechanics of the lumbo-pelvic-hip complex. In: The Pelvic Girdle, second ed. Churchill Livingstone, Edinburgh, pp. 43–72.

Lee, D., Vleeming, A., 2000. Current concepts on pelvic impairment. In: Singer, K.P. (Ed.), Proceedings of the 7th Scientific Conference of the IFOMT in Conjunction with the MPAA International Federation of Orthopaedic Manipulative Therapists. Perth, pp. 465–491.

Leroi, A.M., Bernier, C., Watier, A., et al., 1996. Prevalence of sexual abuse among patients with functional disorders of the lower gastrointestinal tract. Int J Colorectal Dis 10 (4), 200–206.

Long, L., 2006. Redefining the second stage of normal labour could help to promote normal birth. Br J Midwif 14 (2), 104–106.

Loughnan, B.A., Carli, F., Romney, M., et al., 2002. Epidural analgesia and backache: a randomized controlled comparison with intramuscular meperidine for analgesia during labour. Br J Anaesth 89, 466–472.

Lovegrove Jones, R.C., Peng, Q., et al., 2009. Mechanisms of pelvic floor muscle function and the effect on the urethra during a cough. Eur Urol 57, 1101–1110.

Lund, I., Lundeberg, T., Lonnberg, L., et al., 2006. Decrease of pregnant women's pelvic pain after acupuncture: a randomized controlled single-blind study. Acta Obstet Gynecol Scand 85, 12–19.

MacArthur, C., Lewis, M., Knox, B.J., et al., 1990. Epidural anaesthesia and long-term backache after childbirth. BMJ 301, 9–12.

MacArthur, A.J., MacArthur, C., Weeks, S.K., 1997. Is epidural anesthesia in labor associated with chronic low back pain? A prospective cohort study. Anesth Analg 85, 1066–1070.

MacArthur, C., Glazener, C.M., Wilson, P.D., et al., 2006. Persistent urinary incontinence and delivery mode history: a six year longitudinal study. BJOG 113 (2), 218–224.

McIntyre, I.N., Broadhurst, N.A., 1996. Effective treatment of low back pain in pregnancy. Aust Fam Physician 25, S65–S67.

MacLennan, A.H., MacLennan, S.C., 1997. Symptom-giving pelvic girdle relaxation of pregnancy, postnatal pelvic joint syndrome and dysplasia of the hip. Acta Obstet Gynecol Scand 76, 760–764.

MacLennan, A.H., Nicolson, R., Green, R.C., 1986a. Serum relaxin in pregnancy. Lancet 2, 241–243.

MacLennan, A.H., Nicolson, R., Green, R.C. et al., 1986b. Serum relaxin and pelvic pain of pregnancy. Lancet 2, 243–245.

Macmillan, A.K., Merrie, A.E.H., Marshall, R.J., et al., 2004. The prevalence of fecal incontinence in community-dwelling adults: a systematic review of the literature. Dis Colon Rectum 47 (8), 1341–1349.

McNabb, M., 1997. Maternal and foetal physiological responses to pregnancy. In: Sweet, B.R. (Ed.), Mayes Midwifery, twelfth ed. Baillière Tindall, London, pp. 123–147.

Madoff, R., Parker, S.C., Madhulika, G.V., et al., 2004. Faecal incontinence in adults. Lancet 364, 621–632.

Mansel, R.E., Fallowfield, L., Kissin, M., et al., 2006. Randomized multicenter trial of sentinel node biopsy versus standard axillary treatment in operable breast cancer. The ALMANAC trial. J Natl Cancer Inst 98 (9), 599–609.

Mantle, M.J., Greenwood, R.M., Currey, H.L.F., 1977. Backache in pregnancy. Rheumatol Rehabil 16, 95–101.

Markwell, S.J., Sapsford, R.R., 1995. Physiotherapy management of obstructed defaecation. Aust J Physiother 41, 279–283.

Mathias, S.D., Kuppermann, M., Liberman, R.F., et al., 1996. Chronic pelvic pain: prevalence, health-related quality of life, and economic correlates. Obstet Gynecol 87, 321–327.

Megdal, S.P., Kroenke, C.H., Laden, F., et al., 2005. Nightwork and breast cancer risk: a systematic review and meta-analysis. Eur J Cancer 41, 2023–2032.

Melzack, R., Wall, P., 1982. The Challenge of Pain. Penguin, Harmondsworth.

Mens, J.M., Vleeming, A., Stoeckart, R., et al., 1996. Understanding peripartum pelvic pain. Spine 21, 1363–1370.

Mens, J.M., Vleeming, A., Snijders, C.J., et al., 2002. Reliability and validity of hip adduction strength to measure disease severity in posterior pelvic pain since pregnancy. Spine 27, 1674–1679.

Mens, J.M., Damen, L., Snijders, C.J., et al., 2006. The mechanical effect of a pelvic belt in patients with pregnancy-related pelvic pain. Clin Biomech 21, 122–127.

Miller, J.M., Sampselle, C., Ashton-Miller, J., et al., 2008. Clarification and confirmation of the Knack maneuver: the effect of volitional pelvic floor muscle contraction to preempt expected stress incontinence. Int Urogynecol J 19 (6), 773–782.

Moffat, C.J., Franks, P.J., Doherty, D.C., et al., 2003. Lymphoedema: an underestimated health problem. Q J Med 96, 731–738.

Mogren, I.M., Pohjanen, A.I., 2005. Low back pain and pelvic pain

during pregnancy: prevalence and risk factors. Spine 30, 983–991.

Moore, K., Dumas, G.A., Reid, J.G., 1990. Postural changes associated with pregnancy and their relationship with low-back pain. Clin Biomech 5, 169–174.

Mørkved, S., 2007. Evidence for pelvic floor physical therapy for urinary incontinence during pregnancy and after childbirth. In: Bø, K., Berghmans, B., Mørkved, S., et al. (Eds.), Evidence-Based Physical Therapy for the Pelvic Floor. Elsevier, Edinburgh, pp. 317–336.

Mørkved, S., Bø, K., 2000. Effect of postpartum pelvic floor muscle training in prevention and treatment of urinary incontinence: a one year follow up. Br J Obstet Gynaecol 107, 1022–1028.

Newham, D.J., 1988. The consequences of eccentric contraction and their relationship to delayed onset of muscle pain. Eur J Appl Physiol 57, 3353–3359.

NICE (National Institute for Health and Clinical Excellence), 2006. Urinary Incontinence; The Management of Urinary Incontinence in Women. NICE, London.

NICE (National Institute for Health and Clinical Excellence), 2007. Faecal Incontinence; The Management of Faecal Incontinence in Adults. NICE, London.

NICE (National Institute for Health and Clinical Excellence), 2008a. Induction of Labour. NICE, London.

NICE (National Institute for Health and Clinical Excellence), 2008b. Antenatal Care: Routine Care for the Healthy Pregnant Woman. NICE Clinical Guideline 62. NICE, London.

NICE (National Institute for Health and Clinical Excellence), 2009. Early and Locally Advanced Breast Cancer. CG80. NICE, London.

NICE (National Institute for Health and Clinical Excellence), 2010a. Dietary Interventions and Physical Activity for Weight Management in Pregnancy and After Birth. NICE, London.

NICE (National Institute for Health and Clinical Excellence), 2010b. Lower Urinary Tract Symptoms. The Management of Lower Urinary Tract Symptoms in Men. NICE, London.

NICE (National Institute for Health and Clinical Excellence), 2010c. Venous Thromboembolism (VTE) – Reducing the Risk. NICE, London.

NIH (National Institutes of Health), 2005. NIH State-of-the-Science Conference Statement on Management of Menopause-Related Symptoms. NIH Consens State Sci Statements 22 (1), 1–38.

Nilsson-Wikmar, L., Harms-Ringdahl, K., 1999. Back pain in women post-partum is not a unitary concept. Physiother Res Int 4 (3), 201–213.

Noren, L., Ostgaard, S., Nielson, T.F., et al., 1997. Reduction of sick leave for lumbar back and posterior pain in pregnancy. Spine 22, 2157–2160.

Noren, L., Ostgaard, S., Johansson, G. et al., 2002. Lumbar back and posterior pelvic pain during pregnancy: a 3-year follow-up. Eur Spine J 11, 267–271.

Norton, C., Whitehead, W.E., Bliss, D.Z., et al., 2010. Management of fecal incontinence in adults. Neurourol Urodyn 29, 199–206.

ONS (Office for National Statistics), 2010a. Life Expectancy 2007–2009. Stationery Office, London.

ONS (Office for National Statistics), 2010b. Mortality Statistics: Cause. England and Wales. Stationery Office, London.

Ostgaard, H.C., 1997. Lumbar back and posterior pelvic pain in pregnancy. In: Vleeming, A., Mooney, V., Dorman, T., et al. (Eds.), Movement, Stability and Low Back Pain. Churchill Livingstone, Edinburgh, pp. 411–420.

Ostgaard, H.C., Andersson, G.B., 1991. Previous back pain and risk of developing back pain in a future pregnancy. Spine 16, 432–436.

Ostgaard, H.C., Andersson, G.B.J., 1992. Postpartum low back pain. Spine 17, 53–55.

Ostgaard, H.C., Andersson, G.B.J., Schuttz, A.B., et al., 1993. Influence of some biomechanical factors on low back pain in pregnancy. Spine 18, 61–65.

Ostgaard, H.C., Zetherstrom, G., Roos-Hansson, E., et al., 1994. Reduction of back and posterior pelvic pain in pregnancy. Spine 19, 894–900.

Ostgaard, H.C., Roos-Hansson, E., Zetherstrom, G., 1996. Regression of back and posterior pelvic pain after pregnancy. Spine 21, 2777–2780.

Overgård, M., Angelsen, A., Lydersen, S., et al., 2008. Does physiotherapist-guided pelvic floor muscle training reduce urinary incontinence after radical prostatectomy? A randomized controlled trial. Eur Urol 54 (2), 438–448.

Pain, S.J., Purushotham, A.D., 2000. Lymphoedema following surgery for breast cancer. Br J Breast Surg 87, 1128–1141.

Palastanga, N., Field, D., Soames, R., 2002. Anatomy and Human Movement, fourth ed. Butterworth-Heinemann, Oxford.

Paul, J.A., Salle, H., Frings-Dresen, M.H., 1996. Effect of posture on hip joint moment during pregnancy, while performing a standing task. Clin Biomech 11, 111–115.

Pauleta, J.R., Pereira, N.M., Graça, L.M., 2010. Sexuality during pregnancy. J Sex Med 7, 136–142.

Peters, A.A.W., van Dorst, E., Jellis, B., et al., 1991. A randomized clinical trial to compare two different approaches in women with chronic pelvic pain. Obstetr Gynecol 77 (5), 740–744.

Piller, N.B., 1999. Gaining an accurate assessment of the stages of lymphedema subsequent to cancer: the role of objective and subjective information in when to make measurements and their optimal use. Eur J Lymphol 7 (25), 1–9.

Polden, M., Mantle, J., 1990. The postnatal period. In: Polden, M., Mantle, J. (Eds.), Physiotherapy in Obstetrics and Gynaecology. Butterworth-Heinemann, Oxford, pp. 222–280.

Poole, D., 2007. Use of TENS in pain management: part 2. How to use TENS. Nurs Times 103 (980), 28–29.

Pool-Goudzwaard, A.L., Vleeming, A., Stoeckart, R., et al., 1998. Insufficient lumbopelvic stability: a clinical, anatomical and biomechanical approach to 'a-specific' low back pain. Man Ther 3, 12–20.

Pool-Goudzwaard, A.L., Slieker ten Hove, M.C., Vierhout, M.E., et al., 2005. Relations between pregnancy-related low back pain, pelvic floor activity and pelvic floor dysfunction. Int Urogynecol J Pelvic Floor Dysfunct 16, 468–474.

Pons, M.E., Clota, M.P., 2008. Coital urinary incontinence: impact on quality of life as measured by the King's Health Questionnaire. Int Urogynecol J 19 (5), 621–625.

Potter, H.M., Downey, J.L., Jones, S.T., 1997a. Effect of an intense training programme for the deep antero-lateral abdominal muscles on rectus abdominis diastasis: a single case study. In: Proceedings of the 10th Biennial Conference of the Manipulative Physiotherapists Association of Australia (MPAA). Manipulative Physiotherapists Association of Australia, Victoria, pp. 153–155.

Potter, H.M., Randall, H.F., Strauss, G.R., 1997b. Effect of pregnancy and motherhood on trunk muscle strength: an examination of isokinetic trunk strength at 24 weeks postpartum. In: Proceedings of the 10th Biennial Conference of the Manipulative Physiotherapists Association of Australia (MPAA). Manipulative Physiotherapists Association of Australia. Victoria, pp. 151–152.

Rath, A.M., Attali, P., Dumas, J.L., et al., 1996. The abdominal linea alba: an anatomo-radiologic and biomechanical study. Surg Radiol Anat 18, 281–288.

Raz, S., Kaufman, J.J., 1977. Carbon dioxide urethral pressure profile in female incontinence. J Urol 117, 765–769.

RCOG (Royal College of Obstetricians and Gynaecologists), 2006. Exercise in Pregnancy. Statement 4. RCOG, London.

RCOG (Royal College of Obstetricians and Gynaecologists), 2010. Returning to Fitness: Recovering Well. RCOG, London.

Richardson, C., Jull, G., Hodges, P., Hides, J., 1999. Therapeutic Exercise for Spinal Segmental Stabilization in Low Back Pain. Churchill Livingstone, Edinburgh, p. 95.

Riemann, M.K., Kanstrup Hansen, I.L., 2000. Effects on the foetus of exercise in pregnancy (review). Scand J Med Sci Sports 10 (1), 12–19.

Roberts, H., 2007. Managing the menopause. BMJ 334, 736–741.

Robinson, H.S., Veierod, M.B., Mengshoel, A.M., et al., 2010. Pelvic girdle pain – associations between risk factors in early pregnancy and disability or pain intensity in late pregnancy: a prospective cohort study. BMC Musculoskeletal Disorders 11 (91), 1–12.

Rortveit, G., Brown, J.S., Thom, D.H., et al., 2007. Symptomatic pelvic organ prolapse: prevalence and risk factors in a population-base, racially diverse cohort. Obstetr Gynecol 109 (6), 1396–1403.

Röst, C., 1999. Bekkenpijn Tijden En Na De Zwangerschap Een Programma ter Voorkoming van Chronische Bekkeninstabiliteit. Elsevier/De Tijdstroom, Maarssen.

Rud, T., Anderson, K.E., Asmusson, M., et al., 1980. Factors maintaining the intraurethral pressure in women. Invest Urol 17, 343–347.

Russell, R., Reynolds, F., 1997. Back pain, pregnancy and childbirth. BMJ 314, 1062.

Russell, R., Dundas, R., Reynolds, F., 1996. Long term backache after childbirth: prospective search for causative factors. BMJ 312, 1384–1388.

Sacco Ezzell, P., 1999. Managing the effects of gynaecological cancer treatment on quality of life and sexuality. Soc Gynaecol Nurs Oncol 8 (3), 23–26.

Sapsford, R.R., Hodges, P.W., 2001. Contraction of the pelvic floor muscles during abdominal manoeuvres. Arch Phys Med Rehabil 82, 1081–1088.

Sapsford, R.R., Bullock–Saxton, J., Markwell, S., 1998. Women's Health: A Textbook for Physiotherapists. WB Saunders, London.

Sapsford, R.R., Hodges, P.W., Richardson, C.A., et al., 2001. Coactivation of the abdominal muscles and pelvic floor muscles during voluntary exercises. Neurourol Urodyn 20, 31–42.

Schultz, I., Barholm, M., Grondal, S., 1997. Delayed shoulder exercises in reducing seroma frequency after modified radical mastectomy. Ann Surg Oncol 4 (4), 293–297.

Schwartz, Z., Katz, Z., Lancet, M., 1985. Management of puerperal separation of the symphysis pubis. Int J Gynaecol Obstet 23, 125–128.

Serati, M., Salvatore, S., Siesto, G., et al., 2010. Female sexual function during pregnancy and after childbirth. J Sex Med 7 (8), 2782–2790.

Sheppard, S., 1996. The role of transversus abdominis in post partum correction of gross divarication recti. Man Ther 1, 214–216.

Sheppard, L.A., Ely, S., 2008. Breast cancer and sexuality. Breast J 14 (2), 176–181.

Sihvonen, T., Huttunen, M., Makkonen, M., et al., 1998. Functional changes in back muscle activity correlate with pain intensity and prediction of low back pain during pregnancy. Arch Phys Med Rehabil 79, 1210–1212.

Sitzia, J., Woods, M., Hine, P., 1998. Characteristics of new referrals to twenty-seven lymphoedema treatment units. Eur J Cancer Care 7 (4), 255–262.

Slieker-ten Hove, M.C., Pool-Goudwaard, A.L., Eijkemans, M.J.C., et al., 2008. Symptomatic pelvic organ prolapse and possible risk factors in a general population. Am J Obstet Gynecol 200 (2), e1–e7.

Snijders, C.J., Ribbers, M.T.L.M., de Bakker, H.V., et al., 1998. EMG recordings of abdominal and back muscles in various standing postures: validation of a biomechanical model on sacroiliac joint. J Electromyogr Kinesiol 8, 205–214.

Snooks, S.J., Setchell, M., Swash, M., et al., 1984. Injury to innervation of pelvic floor sphincter musculature in childbirth. Lancet 2 (8402), 546–550.

Snow, R.E., Neubert, A.G., 1997. Peripartum pubic symphysis separation: a case series and review of the literature. CME Review 7, 438–443.

Spitznagle, T.M., Leong, F.C., Van Dillen, L.R., 2007. Prevalence of diastasis recti abdominis in a urogynecological patient population. Int Urogynecol J 18, 321–328.

Stanton, A.W.B., Levick, J.R., Mortimer, P.S., 1996. Current puzzles presented by post mastectomy oedema (breast cancer related lymphoedema). Vasc Med 1, 213–225.

Stanton, S.L., Kerr-Wilson, R., Grant Harris, V., 1980. The incidence of urological symptoms in normal pregnancy. Br J Obstet Gynaecol 87, 897–900.

Steege, J.F., Metzger, D.A., Levy, B.S. (Eds.), 1998. Chronic Pelvic Pain: An Integrated Approach. WB Saunders, Philadelphia.

Stones, W., Cheong, Y.C., Howard, F.M., 2005. Interventions for treating chronic pelvic pain in women. Cochrane Database Syst Rev (2): CD000387.

Stuge, B., Hilde, G., Vollestad, N., 2003. Physical therapy for pregnancy-related low back and pelvic pain: a systematic review. Acta Obstet Gynecol Scand 82, 983–990.

Stuge, B., Laerum, E., Kirkesola, G., et al., 2004a. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: a randomized controlled trial. Spine 29, 351–359.

Stuge, B., Veierod, M.B., Laerum, E., et al., 2004b. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: a two year follow-up of a randomized clinical trial. Spine 29, E197–E203.

Stuge, B., Holm, I., Vollestad, N., 2006. To treat or not to treat postpartum pelvic girdle pain with stabilizing exercises? Man Ther 11 (4), 337–343.

Tegerstedt, G., Maehle-Schmidt, M., Nyrén, O., et al., 2005. Prevalence of symptomatic pelvic organ prolapse in Swedish population. Int Urogynecol J Pelvic Floor Dysfunct 16, 497–503.

Ternov, N.K., Grennert, L., Aberg, A., et al., 2001. Acupuncture for lower back and pelvic pain in late pregnancy: a retrospective report on 167 consecutive cases. Pain Med 2, 204–207.

Tobin, M.B., Lacey, H.J., Meyer, L., et al., 1993. The psychological morbidity of breast cancer related arm swelling. Cancer 72, 3248–3252.

Todd, J., Scally, A., Dodwell, D., et al., 2008. A randomised controlled trial of two programmes of shoulder exercise following axillary lymph node dissection for invasive breast cancer. Physiotherapy 94, 265–273.

Toozs-Hobson, P., 1998. Pelvic floor ultrasonography: the current state of ultrasound imaging of the pelvic floor in relation to urogynaecology and childbirth. J Assoc Chart Physiother Wom Health 84, 18–22.

van den Brandt, A., Spiegelman, D., Yaun, S.S., et al., 2000. Pooled analysis of prospective cohort studies on height, weight, and breast cancer risk. Am J Epidemiol 152 (6), 514–527.

Viktrup, L., Lose, G., 2001. The risk of stress incontinence 5 years after first delivery. Am J Obstet Gynecol 185, 82–87.

Vleeming, A., Albert, H., Ostgaard, H.C., et al., 2004. European Guidelines on the Diagnosis and Treatment of Pelvic Girdle Pain, www.backpaineurope.org.

Wedenberg, K., Moen, B., Norling, A., 2000. A prospective randomized study comparing acupuncture with physiotherapy for low-back and pelvic pain in pregnancy. Acta Obstet Gynecol Scand 79, 331–335.

West, Z., 2008. Acupuncture in Pregnancy and Childbirth. Churchill Livingstone, Edinburgh.

Williams, A.F., Vadgama, A., Franks, P.J., et al., 2002. A randomized controlled crossover study of manual lymphatic drainage therapy in women with breast cancer-related lymphoedema. Eur J Cancer Care 11, 254–261.

Williams, A.F., Moffatt, C.J., Franks, P.J., 2004. A phenomenal study of the lived experiences of people with lymphoedema. Int J Palliat Care 10 (6), 279–296.

Wilson, P.D., Herbison, R.M., Herbison, G.P., 1996. Obstetric practice and the prevalence of urinary incontinence three months after delivery. Br J Obstet Gynaecol 103, 154–161.

Winter, C., Cameron, J., 2006. The stages model of labour; deconstructing the myth. Br J Midwif 14 (8), 454–457.

Wishart, G.C., Gaston, M., Poultsidis, A.A., et al., 2002. Hormone receptor status in primary breast cancer – time for a consensus? Eur J Cancer 38 (9), 1201–1203.

Woods, M., Tobin, M.B., Mortimer, P., 1995. The psychosocial morbidity of breast cancer patients with lymphoedema. Cancer Nurs 18 (6), 467–471.

Wreje, U., Isacsson, D., Aberg, H., 1997. Oral contraceptives and back pain in women in a Swedish community. Int J Epidemiol 26, 71–74.

Wu, W.H., Meijer, O.G., Uegaki, K., et al., 2004. Pregnancy-related pelvic girdle pain (PPP), I: Terminology, clinical presentation, and prevalence. Eur Spine J 13 (7), 575–589.

Yamanishi, T., Yasuda, K., Sakakibara, et al., 2000. Randomized, double-blind study of electrical stimulation for urinary incontinence due to detrusor overactivity. Urology 55 (3), 353–357.

Zondervan, K.T., Yudkin, P.L., Vessey, M.P., et al., 1999. Prevalence and incidence of chronic pelvic pain in primary care: evidence from a national general practice database. Br J Obstet Gynaecol 106, 1149–1155.

Dedication

I dedicate this book to the memory of Krish Gooranah, Richard Butcher and Graham Dunn. On behalf of my colleagues at the University of Salford it was our pleasure to have known and taught these fine young men all too briefly; our sadness at their passing is profound.

Tidy's Physiotherapy

Edited by

Stuart B. Porter PhD, BSc (Hons), GradDipPhys, MCSP, FHEA, SRP, CertMHS

Lecturer, University of Salford, Manchester, UK
External Examiner, University of East London, UK
External Examiner, University of Bradford, UK

Edinburgh London New York Oxford Philadelphia St Louis Sydney Toronto 2013

SAUNDERS
ELSEVIER

ISBN 9780702043444

British Library Cataloguing in Publication Data
A catalogue record for this book is available from the British Library

Library of Congress Cataloging in Publication Data
A catalog record for this book is available from the Library of Congress

Notices
Knowledge and best practice in this field are constantly changing. As new research and experience broaden our understanding, changes in research methods, professional practices or medical treatment may become necessary.

Practitioners and researchers must always rely on their own experience and knowledge in evaluating and using any information, methods, compounds or experiments described herein. In using such information or methods they should be mindful of their own safety and the safety of others, including parties for whom they have a professional responsibility.

With respect to any drug or pharmaceutical products identified, readers are advised to check the most current information provided (i) on procedures featured or (ii) by the manufacturer of each product to be administered, to verify the recommended dose or formula, the method and duration of administration, and contraindications. It is the responsibility of practitioners, relying on their own experience and knowledge of their patients, to make diagnoses, to determine dosages and the best treatment for each individual patient, and to take all appropriate safety precautions.

To the fullest extent of the law, neither the Publisher nor the authors, contributors or editors assume any liability for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions or ideas contained in the material herein.

Printed in China

Contributors

Helen Alsop BSc (Hons), MCSP, SRP
Private Practicioner, Sprint Physiotherapy Ltd., London, UK

Andrew Bannan MSc, BSc (Hons), HND, CertEd, MCSP, MHCPC
Lecturer in Physiotherapy, University of Essex; Clinical Specialist Physiotherapist, Bury Physiotherapy; First Team Physiotherapist, Bury St Edmunds Rugby Club; School of Health and Human Sciences, University of Essex, Colchester, UK

Gill Brook MCSP, MSc
Women's Health Physiotherapist, Bradford Teaching Hospitals NHS Foundation Trust; Honorary Lecturer, University of Bradford; Secretary of the International Organization of Physical Therapists in Women's Health, Otley, West Yorkshire, UK

Tamsin Brooks BSc (Hons), MCSP, HPC
Women's Health Physiotherapist, Private Practicioner, Bakewell Physiotherapy Clinic, Bakewell, UK

Helen Carruthers MSc, BSc (Hons), CertHE, MCSP, PGCert
Lecturer in Physiotherapy, Admissions Officer Part-time Programme, University of Salford, Salford, UK

Catherine Carus MSc, GradDipPhys, PGCHEP, MCSP
Physiotherapy Lecturer, Division of Allied Health Professions, University of Bradford, School of Health Studies, Bradford, UK

Alan Chamberlain MA, PG DPE, MCSP, RGN
Fellow of the Higher Education Academy, Senior Lecturer in Physiotherapy, Programme Lead MSc in Advanced Practice in Physiotherapy and MSc Physiotherapy, Faculty of Health and Wellbeing, University of Cumbria, Carlisle/Lancaster, UK

Alison Chambers EdD, MEd, MCSP, FHEA
Dean of Academic Development and Employability, University of Central Lancashire, Preston, UK

Ambreen Chohan PhD, MPhil, BEng (Hons)
RA Clinical Biomechanics, Allied Health Professions, School of Sports, Tourism and the Outdoors, University of Central Lancashire, Preston, UK

Lynn Clouder GradDipPhys, BSc (Hons), MA, PhD, MCSP, FHEA
NTF Director of the Centre for Excellence in Learning Enhancement (CELE), Professor, Faculty of Health and Life Sciences, Coventry University, Coventry, UK

Yvonne Coldron PhD, MSc, MCSP, MMACP
Clinical Specialist Physiotherapist, Croydon Healthcare Services NHS Trust, Croydon, UK

Louise Connell PhD, MCSP
Research Therapist, Division of Physiotherapy Education, University of Nottingham, Nottingham, UK

Lindsey Dugdill BA, MA, MPhil, PhD
Professor of Public Health, College of Health and Social Care, University of Salford, Salford, UK

Anne Dyson MCSP, SRP
Physiotherapy Manager, Liverpool Heart and Chest Hospital NHS Foundation Trust, Liverpool, UK

Stephanie Enright PhD, MPhil, MSc, MCSP, SRP, PGCertHE
Wales College of Medicine, Biology, Life and Health Sciences, Cardiff University, Cardiff, UK

Renuka Erande MSc, BSc, CSP
Stroke Research Practitioner, University College London Hospitals, NHS Foundation Trust, Thames Stroke Research Network, London, UK

Sally French
Freelance Researcher and Writer; Associate Lecturer at the Open University, UK

Lynne Gaskell M (Res) GradDipPhys, MCSP
Lecturer in Physiotherapy, Directorate of Sport, Exercise and Physiotherapy, School of Health, Sport and Rehabilitation Sciences, University of Salford, Salford, UK

Janette Grey GradDipPhys, CertEd HE/FE, MSc, MCSP, HPC registered, FHEA
Professional Lead for Physiotherapy, University of Central Lancashire, Preston, Lancashire, UK

Contributors

Rob Grieve PhD, MSc, MCSP
Senior Lecturer in Physiotherapy, Department of Allied Health Professions, Faculty of Health and Life Sciences, University of the West of England, Bristol, UK

Carolyn A. Hale GradAssocPhys, MSc, HCPC
Chartered Physiotherapist, Clinical Specialist, Pace Rehabilitation, Cheadle, Cheshire, UK

Ruth Hawkes FCSP
Women's Health Physiotherapist, Choir School Physiotherapy, Lincoln, UK

Lee Herrington PhD, MCSP
Senior Lecturer in Sports Rehabilitation, Programme Leader MSc Sports Injury Rehabilitation, Directorate of Sport, Exercise and Physiotherapy, School of Health, Sport and Rehabilitation Sciences, University of Salford, Salford, UK

Linda Hollingworth
Senior Lecturer, Directorate of Sport, Exercise and Physiotherapy, School of Health Sciences, University of Salford, Salford, UK

Lester Jones MScMed (PM), BAppSc (Hons), BBSc, GradDipBehavStdsHlthCare
Lecturer, Department of Physiotherapy, Faculty of Health Sciences, La Trobe University, Melbourne, Australia

Mandy Jones PhD, MSc, MCSP, SRP
Course Director, Physiotherapy School of Health Sciences and Social Care, Brunel University, London, UK

Swati Kale MSc, BSc, PGCHEP, MCSP
Lecturer in Physiotherapy, Admissions Officer for BSc Physiotherapy and MSc Physiotherapy, University of East Anglia, Norwich, UK

Judith Lee MCSP
Clinical Specialist Women's Health Physiotherapist, Nottingham University Hospitals NHS Trust, Nottingham, UK

Melanie Lewis MCSP
Lead Macmillan Lymphoedema Physiotherapy Specialist, Singleton Hospital, Swansea, UK

Monika Lohkamp PhD, MSc, MCSP, FHEA
Lecturer in Sport Rehabilitation, Department of Sport, Health and Exercise Science, University of Hull, Hull, UK

Duncan Mason BSc (Hons), SRP
Lecturer in Physiotherapy, Directorate of Sport, Exercise and Physiotherapy, School of Health, Sport and Rehabilitation Sciences, University of Salford, Salford, UK; Director of Athlete Matters, Manchester Endurance Physiotherapist UK Athletics, UK

Paula McCandless MSc, MCSP, SRP, FHEA
Senior Lecturer in Physiotherapy (Neurology), School of Sport, Tourism and the Outdoors, University of Central Lancashire, Preston, UK

Lorimer Moseley PhD, BAppSc (Hons), FACP
Professor of Clinical Neurosciences and Chair in Physiotherapy, The Sansom Institute for Health Research, University of South Australia, Adelaide, Australia; Senior Research Fellow, Neuroscience Research Australia, Sydney, Australia

Wendy Munro MSc, PGCert HEPR, MCSP
Lecturer in Physiotherapy, Programme Leader Part-time Route, Directorate of Sport, Exercise and Physiotherapy, School of Health, Sport and Rehabilitation Sciences, University of Salford, Salford, UK

Hilary Pape MSc, BSc (Hons), DPodM PCAP
Physiotherapy Lecturer, Division of Allied Health Professions, University of Bradford, Bradford, UK

Sue Pieri-Davies MCSP, MSc, PGCILTH
Consultant AHP and Lead Clinician – Ventilation Services; Lecturer for Cardiff and Edge Hill Universities, NWRSIC Southport District General Hospital, Southport, UK

Sarah Prenton BSc (Hons), PGCert HEPR, MCSP
Lecturer in Physiotherapy, Directorate of Sport, Exercise and Physiotherapy, School of Health Sciences, University of Salford, Salford, UK

Ann Price MSc, MCSP, DipTP, Cert Ed
Advanced Practitioner in Arthroplasty, Wrightington Hospital, Lancashire, UK

Bhanu Ramaswamy MCSP, MSc (First Contact Care), PracCert Non-medical Prescribing, PGCert (Neurological Physiotherapy), GradDipPhys, SRP
Independent Physiotherapy Consultant, Sheffield; Honorary Visiting Fellow at Sheffield Hallam University, Sheffield, UK

Gillian Rawlinson
Senior Lecturer, University of Central Lancashire, Preston, Lancashire, UK

Melanie Reardon MSc GradDipPhys, MCSP, SRP, CertMHS
Senior Physiotherapist, Southport and Ormskikr NHS Trust, Southport, UK

Jim Richards PhD, MSc, BEng (Hons)
Professor of Biomechanics and Research Lead for Allied Health Professions, School of Sports, Tourism and the Outdoors, University of Central Lancashire, Preston, UK

Alec Rickard BSc (Hons), PGCert LTHE, MCSP, FHEA
Lecturer and Admissions Tutor, Physiotherapy, School of Health Professions, Faculty of Health, University of Plymouth Peninsula Allied Health Centre, Plymouth, UK

Sushma Sanghvi MSc, MSc (Ayu), MCSP, MA
Freelance Lecturer and Consultant Physiotherapist, The Sherwood Clinic, Harrow, UK

Fiona M. Schreuder **MSc, BAppSc, PGDip LTHE, MCSP, HPC registered**
Senior Lecturer, Physiotherapy Division, Department of Allied Health Professions and Midwifery, School of Health and Social Work, University of Hertfordshire, Hatfield, UK

Ceri Sedgley
Professional Adviser, Chartered Society of Physiotherapy, London, UK

Katie Small **PhD, BSc (Hons), GSR**
Senior Lecturer in Sports Therapy, Quality Group Sport and Physical Activity, Faculty of Health and Well-being, University of Cumbria, Carlisle, UK

Christine Smith **MSc, GradDipPhys, Cert Ed**
Director, School of Health, Sport and Rehabilitation Sciences, Senior Lecturer in Physiotherapy, University of Salford, Salford, UK

Nicholas T.L. Southorn **BSc (Hons), MSc, MCSP**
Physiotherapy Pain Specialist, PhD (Medicine) Student, Pain Management Solutions Ltd, Nottingham, UK

John Swain **BSc (Hons), MSc, PhD, PGCE**
Professor of Disability and Inclusion, Research and Enterprise Health, Community and Education Studies, Northumbria University, Benton, Newcastle-upon-Tyne, UK

Jacquelyne Todd **MCSP, PhD**
Physiotherapy Consultant in Lymphoedema, Wharfedale Hospital, Otley, West Yorkshire, UK

Kathleen Vits **MCSP, MSc**
Women's Health Physiotherapist Urogynaecology Department, Princess Anne Hospital, Southampton, UK

Anita Watson **BSc (Hons), MCSP, SRP**
Lecturer in Physiotherapy, School of Health, Sport and Rehabilitation Sciences, University of Salford, Salford, UK

Tim Watson **PhD, BSc, MCSP**
Professor, Department of Allied Health Professions and Midwifery, School of Health and Social Work, University of Hertfordshire, Hatfield, UK

Joan M. Watt **MA, MCSP, MSMA**
Hon President CPMaSTT; President Sports Massage Association; Hon Medical Advisor Scottish Commonwealth Games; Lead Physiotherapist British Shooting; Head Physiotherapist to GB Athletics 1984 to 1996.

Liz Whitney **RGN, RM, BSc (Hons), Combined Health Studies, MSc Midwifery, PGDip HE**
Midwifery Lecturer, University of Bradford, Bradford, UK

Jill Wickham **MSc, MCSP, FHEA**
Senior Lecturer in Physiotherapy; Co-ordinator for CPD and Lifelong Learning, Faculty of Health and Life Science, York St John University, York, UK

Kelly L. Youd **BSc (Hons), MCSP, SRP**
Physiotherapy Clinical Lead – Critical Care, Liverpool Heart and Chest Hospital NHS Foundation Trust, Liverpool, UK

Preface

Tidy's Physiotherapy returns for its fifteenth edition and for a third time I have been entrusted with the task of editing it. Once again in this new edition, experts from a wide range of clinical and academic backgrounds have produced chapters that give physiotherapy students a clearly laid out reference guide, whilst at the same time encouraging them to problem-solve and facilitate their learning. With my team of authors, we have maintained the strong core that has become the hallmark of this textbook and added some new chapters, for example, on reflection, leadership, neurodynamics and acupuncture. Each author brings their own professional experience and flavour to the chapters and the final result is a textbook which is palatable and accessible to students and practitioners alike.

As usual my thanks to all my students who teach us so very much.

I would like to give specific thanks to Rita Demetriou-Swanwick, Veronika Watkins and Clive Hewat at Elsevier for their support. The following people have been an invaluable source of opinions and comments: Debra Dunlop, Angie Fairhurst, Natalie Ingham, Kirsty Smith, Basharat Kahn, Evaggelia Moriatis, Daniel Richards, Rebecca Borsbey, Andrew Gerling, Charlotte Kelly, Natasha Barrett, Rachel Sellens, Andrew Lockhart, Laura Jane Cowdell, Louis Platt, Sante Saleh, Paul Cieplak, Martyn Matthews, Suzanne (Binns) McCollum, Gemma Hepburn, Stacey Arthern, Paul Wike, Sviatlana Birukova, Nina Dean, Gemma Dickinson, Jennifer Littler, Sarah Willians, Adam Newall, Rita Ijeomah, Andrew Lee, Alisha Alford, Darragh Sheehy, Adrianna Hynowska, Nicola deJong, Alisha Alford, Rachel Squibbs, Daniel Rix and Alex Long.

For the Southeast Asia edition I am indebted to Kush Yadav BPT, MSc in advancing physiotherapy, Gurpreet Singh BPT MSc in advancing physiotherapy and Suhas Deshmukh BPY MSc in advancing physiotherapy.

Finally, my wife whose love and support spur me on, and my daughters, who may have outgrown my lap but will never outgrow my heart.

Dr Stuart B Porter
PhD, BSc (Hons), GradDipPhys, MCSP, FHEA, SRP, CertMHS
Lecturer in Physiotherapy
Admissions Officer, Full Time Programme
Admissions Officer MSc, Advancing Physiotherapy
Level 1 Manager, Full Time Programme
University of Salford, Manchester
External Examiner, University of Bradford
External Examiner, University of East London

Student's Testimonial

If you want something done – do it yourself. If you want something done well – ask Dr Stuart Porter.

As Tidy's fresh-faced new editor all those years ago, Dr Porter dragged a tired, limping beast of a textbook off the shelf and not only into the twentieth century but also to the top of the best-selling physiotherapy textbook list. To take on such a Herculean job may also be seen as a thankless Sisyphean task. Nonetheless, Dr Porter has achieved that which would take many a lifetime: an engaging, highly useable, professional textbook that manages to straddle several professions, while still being pertinent and user-friendly to all.

The 15th edition continues to act as a transparent and readable reference book for physiotherapy students who often require an anchor in the stormy waters of undergraduate study. Included in this edition are topics as diverse as neurodynamics, biomechanics, pain and pharmacology – all of which are relevant to students from a wide variety of professional backgrounds.

No success is achieved single-handedly and this edition is no exception – teeming, as it does, with a plethora of professionals; indeed, it is their knowledge and their desire to impart this to others that ensures this edition will excel.

Katherine E. Crook MA PGDE BA
Bury, 2012

Index

Page numbers followed by 'f' indicate figures, 't' indicate tables, and 'b' indicate boxes.

A

Aδ fibres, 382, 382t
ABC UK (Activities-Specific Balance Confidence Score), 470
abdominal muscles, female, 606–607
  anatomy/physiology, 606–607
  in pregnancy, 609
    re-education, 614–615
abduction
  hip joint, 239
  prosthesis, transfemoral amputee, 469
  shoulder joint, 232, 321t
    exercises, 232, 286f
  see also FABER test
abductors, hip, assessment, 238–239, 238f
abscess, lung, 117–118
acceleration
  angular *see* angular acceleration
  gravity and, 345
  linear *see* linear acceleration
  Newton's second law and, 344
accessible communication, 195–196
accessory movements
  assessment, 212
    ankle joint, 249–250
    cervical spine, 229
    hip joint, 240
    knee joint, 243, 245–246
    lumbar spine, 222–223
    shoulder joint, 233
  with femoral shaft fractures, loss, 503, 519
  in soft tissue injury rehabilitation, 212–213
accessory respiratory muscles, 129
Accuhaler (Disk), 99
ACE (angiotensin-converting enzyme) inhibitors, 58
acetaminophen, 60–61, 65
Achilles tendon reflex, 220, 221f
Achilles tendonitis, frictions in, 491
aciclovir, 63
acoustic streaming, ultrasound, 437
acromioclavicular joint assessment, 233–234
action
  reflection before (reflection on future), 73, 79f
  reflection during/in, 72b, 73, 79f
  reflection following/on, 72b, 73, 79f
action learning sets, 49–50
action potentials, nerve, 61, 420–421
action–reaction in Newton's third law, 344–345
active cycle of breathing technique
  COPD, 94
  thoracic surgery patient, 178
active exercises
  assisted, 280
  free, 276–278, 281
    in acute respiratory distress syndrome, 116
active insufficiency of muscles, 214
active movements
  assessment, 212
    ankle joint, 248–249
    cervical spine, 226–227
    hip joint, 239
    knee joint, 249
    lumbar spine, 216–217
    shoulder joint, 231–233
active muscle imbalance, 306
active phase of breathing cycle, 130–131
activities (activity), neurological patients, 584
  definition in International Classification of Functioning, 583
  as outcome measure, 585t
activities of daily living
  in cardiac rehabilitation, 162
  impairments impacting on, 543–544
  see also exercise
Activities-Specific Balance Confidence Score (ABC UK), 470
acupressure, 488–489
acupuncture, 403–415
  antenatal, 615
  clinical implications, 410–411
  history, 403–404
  meridian points *see* meridian points
acupuncture TENS, 423
acute respiratory distress syndrome (ARDS), 115–116
  risk with fractures, 499
acyclovir, 63
adduction
  hip joint, 236, 239
  scapulothoracic joint, 321t
adenocarcinoma, lung, 120t
adenosine, 63
adherence (compliance) with exercise prescription, 300–301
adhesions
  intra-/periarticular, fractures leading to, 500–501, 522
  soft tissue, prevention, 266–267
adjunct analgesia drugs, 63
adjuvant therapies in breast cancer, 624
adolescents
  cystic fibrosis, clinical features, 107
  resistance training, 280
adrenaline, 63

advance pressure modes of ventilation, 129–133
advice *see* education
aerobic exercise
  in cardiac rehabilitation, adaptations at submaximal level of, 153–154
  in pregnancy, 617
age
  of condition, determining, 210–211
  fracture union and, 499
  heart rate maximum in exercise training and, 155
  soft tissue healing and, 262
  see also adolescents; children; infants; older people
ageing, 539–540
AGILE (clinical interest group of Chartered Society of Physiotherapy), 539, 545, 545b
  fall prevention, 551
agonist, 62
aims in goal-setting (neurological patient), 586, 586t
air pollution and chronic bronchitis, 85
airflow
  resistance, 132–133
    disorders of *see* obstructive pulmonary disease
  in spontaneous ventilation, 130–131
airway disease *see* obstructive pulmonary disease; respiratory disease; restrictive pulmonary disease
airway pressure, positive *see* positive airways pressure
airway pressure release ventilation, 137
akinesia, Parkinson's, 541–542
alendronate, 63
alfentanil, 63
algodystrophy *see* complex regional pain syndrome
alignment
  ankle/foot, 247–248
  lumbar spine
    deviation, 215–216
    normal, 215
  shoulder, anterior/posterior/lateral, 231
  see also deformities
allergens in asthma, 84, 95–96
  avoidance, 101
Allied Health Professions, 43–45
  interprofessional education and, 27
allodynia, 385, 385b
alveolar hypoventilation, 133–134, 134t
alveolitis, fibrosing, 117
aminoglycosides, 59
aminophylline, 63
amiodarone, 63–64
amitriptyline, 64
amlodipine, 64
amortisation phase, 295
'amplitude' window for electrotherapy, 419
amputation (predominantly lower limb), 457–474
  assessment, 461–462
  causes, 457–458
  complex/multiple, 471
  levels, 458, 458t
  outcome measures, 469–470
  pain and its relief, 459–460
  psychosocial impact, 458–459
  role of physiotherapist, 460–461
  special considerations, 470–471
  stages of physiotherapy, 462–468
Amputee Mobility Predictor (AMP Pro), 470
amyotrophic lateral sclerosis, 596
anaesthesia, 59–61
  epidural *see* epidural analgesia/anaesthesia
  local anaesthetics, 61
anal disorders, 621
  incontinence *see* faeces
analgesia (in general) *see* pain management
analgesic drugs, 59–61, 391, 391b
  adjuncts, 63
  childbirth, 611
  thoracic surgery, 176
anatomy
  acupuncture and knowledge of, 406
  pelvic, women, 605–608
angina, 58
  stable, cardiac rehabilitation, 149
angiotensin-converting enzyme (ACE) inhibitors, 58
angular acceleration, 340
  knee (normal and osteoarthritic) during walking, 342
angular displacement, 340
  knee (normal and osteoarthritic) during walking, 341
angular power, 361
angular velocity, 340
  knee (normal and osteoarthritic) during walking, 341–342
angular work, 361
ankle (joint), 220–222, 221f, 246–250
  angle, 333
  assessment, 246–250
    femoral shaft fractures, 503
  dorsiflexion *see* dorsiflexion
  fractures around, 516
  ligaments *see* ligaments
  moments, 358
    in gait, 359
  motion/movements, 248, 334–335
  power during walking, 362
  proprioception, 248
  replacement, 537
  soft tissue injury (incl. sprains), 246, 257b, 261b, 264b, 270b
    case study, 386
anorectal disorders *see* anal disorders; rectal disorders
antenatal education and classes (incl. exercise), 614, 617–618
anterior draw test
  ankle, 249
  knee, 244
    modified (Lachman's test), 211, 245
  shoulder, 234
antiarrhythmic (antidysrhythmic) drugs, 57
antibiotics, 59
  respiratory infections
    cystic fibrosis, 108
    tuberculosis, 119
    ventilator-associated pneumonia, 111
anticholinergics, COPD, 92
anticipation (expectation) of pain, 386
antidepressants, tricyclic, 61
antidysrhythmic drugs, 57
anti-epileptic drugs, 61
anti-inflammatory drugs, 58–59, 391
  airway inflammation, 58–59
  non-steroidal *see* non-steroidal anti-inflammatory drugs
anti-inflammatory effects of acupuncture, 409
antispasmodics in multiple sclerosis, 595
α_1-antitrypsin deficiency, 86–87
anus *see* anal disorders
anxiety and pain, 386
anxiolytics, 59
Any Qualified Provider, 17–18
ape-like posture, Parkinson's disease, 542
apraxia, 582
aquatic exercises *see* water-based exercises
arm *see* upper limb

arrhythmias (dysrhythmias), drug therapy, 57
arterial blood gases *see* blood gases
arterial disease, peripheral *see* peripheral vascular disease
arthritis (and inflammatory arthropathies)
  degenerative *see* osteoarthritis
  laser therapy, 448
  rheumatoid *see* rheumatoid arthritis
arthroplasty (joint replacement), 525–538
  lower limb, 531–537
    hip *see* hip
    knee, 535–537
  upper limb, 525
articulations *see* joints
Asian people, 187–188
aspirin, 60, 64
  stroke, 590
assessment (patient incl. musculoskeletal tissues), 207–251
  amputee, 461–462
  in cardiac rehabilitation, exercise training prescription, 154
  fractures, 506–509
    case study, 519–520
  general issues, 207–208
  muscle imbalance, 311–317
  neurodynamic, 563–565
  neurological patient, 582–584
    Parkinson's disease, 544–551
  objective *see* objective assessment
  older people, 544–551
  pain *see* pain
  Parkinson's disease, 544–551
  pitch-side, 376
  proprioception *see* proprioception
  reflection as form of, 71–72
  subjective *see* subjective assessment
  thoracic surgery, 177–178
  see also specific musculoskeletal structures
Association of Chartered Physiotherapists in Cardiac Rehabilitation (ACPICR), 152b
Association of Chartered Physiotherapists in Women's Health, 605, 605b
asthma, 95–98
  acute/acute severe attacks, 98
  aetiology and prevalence, 95–96
  clinical features, 96–98
  drug therapy, 58–59
  extrinsic/atopic, 95–97
  intrinsic/chronic, 95, 98
  medical treatment, 98–102
  pathology, 96
  physiotherapy, 102–103
asylum seekers, disabled, 188
ataxia, 580–581
atelectasis, ventilator-associated, 137–138
atenolol, 64
athetosis, 580
athletes *see* sports
atmospheric pollution and chronic bronchitis, 85
atopic (extrinsic) asthma, 95–97
ator(o)vastatin, 64
atropine, 64
attention
  pain and its effects on, 386, 386b, 395
  tactile acuity training, 394b
attitudinal barriers to disability, 185, 187–188, 192–193
audit, clinical, 15
auditory cues in Parkinson's patients, 555t
auditory disturbances, neurological conditions with, 582
auscultation (breathing sounds)
  asthma, 97
  COPD, 90
  cystic fibrosis, 107
  fibrosing alveolitis, 117
  pneumonia, 111
auto-assisted exercises, 281
autonomic nervous system, 561
  brain injury, 598
  neurological conditions causing disturbances, 582
  see also parasympathetic nervous system; sympathetic nervous system
autonomy (personal freedom), 5
  patient, 5
  professional, 2, 5
avascular necrosis (with fractures), 500
  femoral neck fractures, 514
avoidance *see* fear–avoidance beliefs
avulsion fracture, 495
  pelvis, 513
axillary lymph nodes in breast cancer, 623–624
axillary web syndrome (after breast surgery), 624
axoplasmic flow, 563

B

Babinski reflex, 220b
back
  flat, 216
  pain *see* low back pain
  sway, 216
back care classes in pregnancy, 617
back rehabilitation program, 225
bacterial infections, antibiotics *see* antibiotics
Baker's cyst, 242
balance
  amputee
    assessment, 461t
    postoperative, 464
  neurological patients, as outcome measure, 585t
  older people, 548–549
  Parkinson's disease, 548–549, 554
    case study of management, 556t
ball *see* gym ball; medicine ball; Swiss ball
ballismus, 581
ballistic stretching, 282
bandages, compression *see* compression garments
barotrauma, ventilator-induced, 138
basal ganglia, 548
  dysfunction (incl. Parkinson's disease), 540
  in proprioception, 288
BCG vaccination, 119
beating, 485–486
beclomethasone, 64
bed, lower limb amputee rehabilitation in, 464t
behaviour (individual/personal)
  disturbances, 582
    brain injury, 599
  health related to, 189
    changes/improvements in, 199
    ill (unhealthy behaviour), 189, 199–200
  in pain measurement, 388
  professional, codes, 8–9
behavioural interventions (incl. modification/change), 200
  in urogenital dysfunction, 620
  see also cognitive-behavioural therapy
beliefs and their potential impact on pain, 388–389
  clinicians, 389b
bendroflumethiazide, 64
benign multiple sclerosis, 594
Bennett's fracture, 513
benzodiazepines, 59
beta$_2$ (β_2) agonists
  asthma, 98, 100–101
  COPD, 92
biceps brachii reflex testing, 228
bilevel ventilation, 137
bioavailability, 62
biochemical tests, pneumonia, 111

biofeedback
anorectal dysfunction, 621
pressure biofeedback device, 294
in urogenital dysfunction, 620
biological factors affecting health, 189
biomechanics, 331–368
pregnancy-related changes in spinal and pelvic joints, 608–609
respiratory, 130–131
biopsy, bronchial/lung tumour, 120
biopsychosocial assessment
lumbar spine, 223–225
pain patient, 387, 388t
black flags, 389
black people, disabled, 186
discrimination, 187
bladder dysfunction
multiple sclerosis, 595
women, 618–619
see also urinary incontinence
bleeding *see* haemorrhage; healing and repair
blood
coughing up *see* haemoptysis
flow/supply *see* circulation
blood disorders complicating fractures, 499
blood gases (arterial)
asthma, 97
COPD, 90
cystic fibrosis, 94
respiratory failure, 129
thoracic surgery, preoperative, 171
see also carbon dioxide; oxygen
blood pressure, high (hypertension), exercise-based cardiac rehabilitation and, 163
blood tests (previous), records of, 211
see also haematology
blood vessels *see* vasculature
blue bloaters, 91
blue flags, 389
body
cortical representations, targeting in pain management, 394
forces acting on, 346–349, 354–359
function *see* function
moments acting on, 354–359
positioning, *see also* positioning
segment *see* segment angle/inclination
body chart, 208–209
body mass index and exercise-based cardiac rehabilitation, 164
bone
cervical spine anomalies, 229
fracture and its healing *see* fracture
metastases from lung cancer, 120
pelvic, female anatomy, 605–606
bone scans (previous), records of, 211
Borg category ratio, 156t
Borg rating of perceived exertion, 155t
bow-legs (genu varum), 241
bowel problems in multiple sclerosis, 594
see also faeces; gastrointestinal tract
brachioradialis reflex testing, 228
bracing, 503
femoral shaft fracture, 519–521
bradykinesia, 581
Parkinson's, 541
brain (cerebrum; supraspinal structures)
in acupuncture, 409
imaging studies, 407–408
anatomy, 588f
haemorrhage *see* haemorrhage
injury, 597–600
non-traumatic, 597
traumatic, 372–373, 597–600
metastases from lung cancer, 120
pain/nociceptive mechanisms, 382–386
targeting in pain management, 392
proprioception and the, 288
brainstem and proprioception, 288
breast cancer, 623–625
psychosexual issues, 626–627
breath-activated inhalers, 83
breathing
active phase of cycle of, 130–131
with inhalers, technique, 101–102
sleep and, 122
sounds, auscultation *see* auscultation
work of, 131, 134–135
see also expiration; inspiration; ventilation
breathing control (incl. breathing exercises)
asthma, 103
COPD, 94
thoracic surgery patients, 178
see also thorax, expansion exercises
breathlessness/shortness of breath *see* dyspnoea
brief intense TENS, 423
broad ligament in pregnancy, 607
Broca's area, damage, 582
bronchi
irritations in COPD, decreasing, 91–92
secretions *see* secretions
tumours, 119–121
bronchiectasis, 103–105, 171
in cystic fibrosis, 106
surgery, 105, 171
bronchitis, chronic, 85–86
emphysema combined with, 86b
bronchodilators (for bronchospasm control), 58–59
asthma, 58–59, 98
COPD, 92
cystic fibrosis, 108
bronchopneumonia, 111
bronchopulmonary segments, 170f
bronchoscopy (incl. preoperatively) of tumours, 120, 171
bronchospasm control *see* bronchodilators
buddy strapping for finger fractures, 502
bulbar palsy, progressive, 596
buoyancy, 299–300
buprenorphine, 60
burns from ice, 258
bursa, differentiation tests, 214
burst mode TENS, 423–424, 432

C

C fibres, 382, 382t
cadence, 332
Caesarean section, 611
inpatient postnatal physiotherapy after, 618
calcaneal fracture, 517
calcaneal valgus and varus, 247
calcium antagonists, 58
calf squeeze test, 250
callus formation, 498
cancellous bone healing, 498–499
cancer (malignant tumour)
amputation in, 470
breast *see* breast cancer
lung, 119
imaging, 171
lymphoedema in *see* lymphoedema
massage contraindicated, 480
oesophageal, 169, 171
see also metastases
captopril, 64
carbamazepine, 64
carbon dioxide tension, arterial ($PaCO_2$), in respiratory failure, 129
type I failure, 121, 133
type II failure, 121, 133–134
carcinoma, lung, types, 120t
cardiac problems *see* heart
cardiovascular system
conditioning exercises, 160
disease (CVD), COPD-associated risk, 90
see also heart disease
drugs acting on, 57–58

in exercise training, physiological adaptations, 153–154
ventilator-related adverse effects, 139
see also circulation
cardioverter-defibrillator, implantable, 147
care (health)
collaborative social care and, 23–39
duty of, 5–6
inverse, law of, 190b
outcome evaluation *see* outcome measures
Care Quality Commission, 14
carpal tunnel syndrome, 562–563
carpometacarpal joint of thumb, fracture, 513
cartilage (articular), fractures involving, 500
cast bracing *see* functional bracing
causalgia *see* complex regional pain syndrome
cavitation, ultrasound, 437
cell(s)
in inflammatory response, 256
laser therapy effects, 447
pulsed shortwave therapy effects on membrane, 442–443
cellulitis in lymphoedema, 626
central nervous system (CNS), pain/nociceptive mechanisms, 382, 385–386
see also brain; neurological disorders; spinal cord
Centre for the Advancement of Interprofessional Education (CAIPE), 24
centre of mass, 333, 347–348, 356, 358
centre of pressure, 346, 349–350
Centres for Independent (or Integrated) Living, 198
centrilobular/centriacinar emphysema, 86–87, 86b
cerebellar ataxia, 594
cerebellum and proprioception, 288
cerebral artery occlusion, clinical features, 591–592
cerebrovascular accident *see* stroke
cerebrum *see* brain
cervical spine, 225–229, 317–320
assessment, 225–229
case study, 223b
lordosis, 288
flexion *see* flexion
muscle imbalance and the, 317–320
cervix in pregnancy, 607–608
dilatation in labour, 610
Charter for Health Promotion, 201
Chartered Society of Physiotherapy (CSP), 2–3, 6–8, 13–15, 43, 47–48
AGILE (clinical interest group) *see* AGILE
clinical guidelines, 14
education programmes, 9
medical insurance and sports physiotherapy, 372
membership, 8
outcome measures, 15
Physiotherapy Framework, 9
Rules of Professional Conduct, 9
chemical noxious stimuli and nociception, 382
chemotherapy
breast cancer, 624
bronchial/lung cancer, 120
chest *see* thorax *and entries under* thorac-
Chester step test, 158
childbirth, 609–612
pelvic floor muscle dysfunction, 607
psychosexual issues, 626
urogenital dysfunction following, 619
see also delivery; labour
children
asthma aetiology and prevalence, 95–96
concussion, 372–373
cystic fibrosis
clinical features, 107
treatment, 107–109
fractures
greenstick, 495
growth disturbances with, 500–501
proximal radius, 511
limb amputation or absence in, 470
resistance training, 280
see also adolescents; infants and babies
China, acupuncture
20th C., 404
historical use, 403
Chinese medicine, traditional (acupuncture and), 404–405
Western medicine and, 405, 409–410
chlorpromazine, 64
chorea, 580
chronic obstructive pulmonary disease (COPD), 84–85
basic issues, 84–85
clinical features/presentations, 87–91
varying, 91
medical treatment, 91–93
pneumothorax, 171
preoperative care in thoracic surgery, 177
chronicity of condition, determining, 210–211
cimetidine, 64
circuit training
for exercise-based cardiac rehabilitation, 160b
control, 161
design, 161t
for functional testing, 296
in group work, 276
circulation (blood supply/flow)
drugs and, 55–56
interferential therapy local effects, 428
leg, assessment, 211
lung surgery, complications, 173t
massage contraindicated with problems in, 479–480
see also cardiovascular system
clapping, 485
Clarke's test, 243
clavicular fracture, 510
clawback, 348
client
communication barriers from viewpoint of, 184b
in health promotion, 200
use of term, 183
see also patient; service user
clinical audit, 15
clinical effectiveness, 12–13
delivery of clinically-effective services, 18
clinical governance, 9–14
continuing professional development/life-long learning and, 16
definition, 10b
clinical guidelines, 13–14
national, applied locally, 13
clinical interview *see* history-taking
clinical leadership *see* leadership
clinical reasoning, 5, 69–71
fracture management, 507
closed fractures, 495
closed kinetic chain exercises, 273, 278, 290
closed reduction, 501
closing the loop in reflective practice, 75
clothing in massage
patient's, 478
practitioner's, 475
clubbing (of digits)
bronchiectasis, 104
cystic fibrosis, 107
fibrosing alveolitis, 117

coaching (by leaders), 50
coaching staff (sports), 378
Cochrane Review, cardiac rehabilitation, 148, 148t
Code of Professional Values and Behaviour, 8–9
codeine, 60, 64
cognition
  clinical reasoning and, 69
  disturbances, 581–582
    brain injury, 599
    multiple sclerosis, 595
  pain and modulatory effect of, 386
cognitive-behavioural therapy, pain, 392
collateral ligaments
  ankle
    assessment, 248–249
    lateral *see* lateral ligaments
  knee
    assessment, 242–244
    tenderness, 242
colleagues, responsibility to your, 7
Colles' fracture, 497, 511, 512f
colour, knee, assessment, 241–242
combined movements
  cervical spine, 227
  lumbar spine, 217–218
common learning, 26t
communication, 183–187
  accessible, 195–196
  barriers to, from client's viewpoint, 184b
  diversity and flexibility, 196
  inclusive, 194–195, 194b, 197–198
  models, 184b
  neurological conditions affecting, 582
  thoracic patients, 177b
  see also speech problems
community
  care in
    amputee, 464t
    cystic fibrosis, 109
    service delivery, 18
  influences on health, 189
community-acquired pneumonia, 110
compact bone healing, 497–498
compartment syndrome, fractures, 499–500
compensations (in imbalance), 312
  muscle imbalance, 311t
competence/competency, 6–7
  in leadership, developing, 48–50
complaints (patient), 15
complex regional pain syndrome (Sudeck's atrophy; reflex sympathetic dystrophy; algodystrophy; causalgia), 394
  fractures, 500
compliance (lungs), 132
compliance with exercise prescription, 300–301
compression (nerve), and associated syndromes, 561, 563
  in pregnancy, 615–616
compression fractures, 495
compression garments (pressure garments) and bandages
  lymphoedema, 626
  soft tissue injury, 259
  sports injuries, 377
compression tests
  acromioclavicular joint, 234
  knee, 245
  lumbar spine, 219
computed tomography
  chest
    bronchial/lung cancer, 120
    preoperative, 171
  previous, records of, 211
concentric contraction and movement, 276t, 277–278, 362
  arm elevation, 324t
concussion, 372–373
conduct (professional), rules, 9
congenital bronchiectasis, 103
congenital emphysema, 86
congenital heart disease, cardiac rehabilitation, 150
congenital limb absence, 470
connective tissue
  acupuncture and, 409–410
  pregnancy-related changes, 608–609
consciousness
  pain and, 381
  sports player and loss of (LOC), 372–373
consent (informed)
  assessment of older people and Parkinson's patients, 544
  to exercise, 300
  to massage, 478
consolidation, fracture, 498
constipation, 621
  multiple sclerosis, 594
continence problems *see* faeces; urinary incontinence
Continuing Professional Development (CPD), 16
  sports physiotherapy, 371, 378
continuous passive motion, 517b–518b
continuous positive airways pressure, post-thoracic surgery, 180
continuous training in exercise-based cardiac rehabilitation, 160
contract/relax techniques, 284
contractions
  cardiac muscle/myocardial, drugs increasing, 57–58
  skeletal muscle
    concentric *see* concentric contraction
    eccentric *see* eccentric contraction
    interferential therapy in generation of, 428
    Oxford Scale of muscle strength, 213b, 274–275, 274t
    reciprocal inhibition, 264, 284
    resisted, symptoms arising from, 210–211
    speed of, effect, 366–367
    types, 277–278, 366
  uterus
    antenatal, 607–608
    in labour, 610
contractures, 273
  amputated lower limb, 464
  Dupuytren's, 257b
  prevention, 266–267
contrecoup lesions, 598
cool-down, exercise-based cardiac rehabilitation, 161
cooling *see* cryotherapy
COPD *see* chronic obstructive pulmonary disease
coping strategies, pain, 388t
cor pulmonale
  COPD, 88
  cystic fibrosis, 107
cording (after breast surgery), 624
core stability, 294, 306–308, 312
  muscle conferring, 317
Core Standard 6 and neurological patients, 584b
core strength, 294
coronal plane, pelvic motion in, 337
coronary artery disease (ischaemic heart disease; CHD), 147–150
  epidemiology, 147–148
  psychosocial benefits of exercise training in, 153–154
  rehabilitation *see* rehabilitation
  revascularisation *see* revascularisation
  risk factors in, 147
cortical representations of body, targeting in pain management, 394
corticosteroids *see* steroids
cost (economic), interprofessional education, 26t
  see also socioeconomic status
cost-effectiveness (UK)
  cardiac rehabilitation, 150
  delivery of cost-effective services, 18

couch, massage, 475
cough
in asthma, 96
in bronchiectasis, 104
in COPD, 88
in cystic fibrosis, 93, 107
in lung cancer, 119
in pleurisy, 112
in pneumonia, 111
supported (technique), 178–179
see also haemoptysis
coupling media, massage, 478–479
covering the patient for massage, 478
COX *see* cyclo-oxygenase
cranial neuropathies in brain injury, 598
cranio-cervical flexion test, 320
creases, lumbar spine assessment, 215–216
crepitus, fractures, 497
critical care patient, rehabilitation in/after, 141
critical friend, 74–75
critical incident reports, 77
crossover (in gait), 348
cruciate ligaments
anterior
assessment, 244–245
rehabilitation, 278
posterior, assessment, 244
crush fractures, 495
cryotherapy (cooling; ice therapy), 258
compression combined with, 259
elevation combined with, 259–260
fractures, 517
in phase 1 and 2 of healing, 258
in phase 3 of healing, 263
sports injuries, 376–377
CSP *see* Chartered Society of Physiotherapy
cueing strategies, Parkinson's disease, 555, 555t
culture, 187
see also ethnic groups/minority groups
cutaneous flaps/grafts etc. *see* skin
cyanosis
asthma, 97
fibrosing alveolitis, 117
cyclo-oxygenase (COX), 60, 62
inhibitors *see* non-steroidal anti-inflammatory drugs
cystic fibrosis, 105–110
cytochrome P450 enzymes, 57

D

de qi (acupuncture), 404–405, 409–411
dead leg, 264b
deafness with neurological disorders, 582
death, coronary heart disease, 147
decision-making, 69–71
service user involvement, 194–195, 197
disabled people, 198
decortication of pleura, 174
deep venous thrombosis with fractures, 499
defaecation
education, 621
obstructed, 621
post-gynaecological surgery, 622
deformities/malalignments (postural)
foot/ankle, 247
fractures causing, 497
leg, 241, 241f, 247
degenerative joint disease, signs and symptoms, 214–215
see also osteoarthritis
delayed-onset muscle soreness, 273, 276–277
delegation, 16–17
delivery
Caesarean *see* Caesarean section
vaginal (2nd stage of labour), 610
assisted, 611
deltoid, 322
deltopectoral approach to shoulder arthroplasty, 525
dementia, 551
demyelination in multiple sclerosis, 593
deoxyribonuclease in cystic fibrosis, 108
depersonalisation, 197
depolarisation, nerve, 417
dermatome tests
cervical spine, 228
lumbar spine, 219
detrusor disorders
multiple sclerosis, 594
women, 619
diabetes mellitus
in cystic fibrosis, 107
exercise-based cardiac rehabilitation and, 163
massage contraindicated, 479
diamorphine, 60
diary, reflection, 78
diastasis (in pregnancy)
rectus abdominis, 615
symphysis pubis, 613
diathermy, shortwave and microwave, 433–434
diazepam, 64
diclofenac, 64
diet *see* nutrition
differentiation tests, 213
hip joint, 218–219, 236–238, 241b
knee, 214, 245
lumbar spine, 237–238
muscles/tendons, 214–215
shoulder joint, 232
vestibular versus vertebrobasilar insufficiency symptoms, 227–228
see also structural differentiation
digit(s) *see* clubbing; fingers; thumb; toes
digitalis drugs (incl. digoxin), heart failure, 57–58
dihydrocodeine, 60, 64
diplopia, 581
direction-setting, 45
disability, 190–193
awareness of, 195
communication, and the barrier, 185–186, 195
cultural/ethnic dimensions, 187–188
decision-making involving people with, 198
definitions, 190b, 192
equality training, 195
health professionals and their relationship with, 195
inclusion and involvement of people with, 198
labelling people with, 183b
from low back pain
predictors, 223–224
treatment of biopsychosocial aspects, 225
measures, in pain, 389–390
models, 190–193, 195
see also International Classification of Functioning, Disability and Health
discharge (and return home/return to function and active lifestyle)
amputee, planning, 464t
breast surgery, 625
from cardiac rehabilitation, planning, 165–166
cardiac rehabilitation immediately after (=phase II), 150
exercise prescription, 164
femoral shaft fracture, 522
gynaecological surgery, 622
hip arthroplasty rehabilitation after, 535
from intensive care, outpatient physiotherapy-led exercise programme after intensive care, 143
thoracic surgery, 180

discoloration, knee, 241–242
discrimination
  cultural, 187–188
  institutionalised, 186, 187b
Disk/Accuhaler, 99
dislocation
  hip replacement, 508t
  shoulder, 268b
displacement
  angular *see* angular displacement
  of fracture, 495
distraction tests
  acromioclavicular joint, 234
  menisci versus ligaments, 245
distribution of drug, volume of, 63
diuretics, 58
diversity in communication, 196
DNase in cystic fibrosis, 108
doctor in sports, 377–378
dopamine, Parkinson's disease and, 59, 541–542, 548
dopamine agonists, 59
doping in sport, 372
dorsal horn and pain, 382, 385
dorsiflexion, 220–222, 221f, 248, 335–336, 362
  exercises increasing, 285
  in slump test, 567
dosing (drug), 57
double crush syndrome, 563
drainage
  chest (with intercostal drains), 174–175
  manual lymphatic (by massage), lymphoedema, 491, 616
  postural *see* postural drainage
draping the patient for massage, 478
draw tests
  ankle, 249
  knee, 242–244
  shoulder, 234
DRIFT (duration, repetition, intensity, frequency, technique), transversus abdominis rehabilitation, 317, 317t
drug(s) (medication), 53–65
  commonly encountered, 57–61
  delivery systems
    asthma, 99
    COPD, 92–93
  fracture union affected by, 499
  glossary, 63–65
  history-taking, 211
    amputee, 461t
  soft tissue healing affected by, 262
  sports and doping and, 372
  see also pharmacology
drug therapy
  arrhythmias, 57
  asthma, 98–99
    guidelines, 100–101
  COPD, 92
  cystic fibrosis, 108
  inflammation *see* anti-inflammatory drugs
  motor neurone disease, 597b
  multiple sclerosis, 595
  see also medical management *and specific (types of) drugs*
dry power inhalers, 99
dual channel output in TENS, 421
Dupuytren's contracture, 257b
duty of care, 5–6
dynamic exercise, lymphoedema, 626
dynamic splints, metacarpophalangeal joint replacement, 529
dynamic stretching, 282
dynamometer
  handheld, 367
  isokinetic (isokinetic machine), 213, 275, 289–290, 367–368
dysarthria, 582
dyskinesia, Parkinson's, 541
dysphagia *see* swallowing difficulty
dysphasia, 582
dyspnoea (shortness of breath)
  asthma, 96
  bronchial/lung cancer, 120
  bronchiectasis, 104
  COPD, 88
  cystic fibrosis, 107
  fibrosing alveolitis, 117
  MRC scale, 85t
  pneumonia, 111
dysrhythmias *see* arrhythmias
dystonia, 580

E

eccentric contraction/movement/exercise, 276–278, 276t–277t, 300, 362
  arm elevation, 324t
  hip extension, 292f
  treatment with, 277–278
ECG (electrocardiogram), asthma, 97
economic factors *see* cost; socioeconomic status
education (patient information and advice; teaching)
  antenatal, exercise, 614, 616–617
  asthma, 102
  cardiac rehabilitation, evidence for, 148–149
  case studies, 543b
  gynaecological surgery, postoperative pelvic floor muscle exercises, 622
  in health promotion, 189
  interprofessional *see* interprofessional education
  mental capacity, 544b
  muscle imbalance, 312t
  pain
    biology, 392
    lumbopelvic, in pregnancy, 614
  Parkinson's disease, 544b
  prosthetic wearing, 465–467
  stretching exercises, 277–278
education and learning (profession of physiotherapy), 9
educator role in interprofessional education, 30
effectiveness *see* clinical effectiveness; cost-effectiveness
effleurage, 481
effusions
  knee, 209, 242
  pleural, 113
eformeterol, 92
elastic components in series, plyometrics and, 295
elastic opposition to lung expansion, 131–132
elbow (and elbow joint)
  arthroplasty, 528
  fractures, 510–511
  moments, 366
    calculation, 354, 356
elderly *see* older people
electric field in pulsed shortwave therapy, 442
electrical stimulation, 419–432, 420f
  functional (FES), 431, 589–590
  general principles, 420
  nerve, transcutaneous *see* transcutaneous electrical nerve stimulation
  neuromuscular *see* neuromuscular electrical stimulation
  in urogenital dysfunction, 620
electrocardiogram (ECG), asthma, 97
electrodes
  in functional electrical stimulation, 438
  in interferential therapy, 429
  in shortwave diathermy, 434
  in TENS, 421, 422f
    placement, 424
electrophysical agents *see* electrotherapy
electrotherapy (electrophysical agents), 417–455
  grouping of modalities, 419–420
  mechanisms (in general), 417
  model of, 418
  therapeutic windows, 223, 418–419

in tissue healing and repair *see* healing and repair
see also electrical stimulation
elevation of limb
arm
concentric movement, 324t
scapulothoracic joint muscles in, 321t
in injury
to soft tissues, 259–260
sports injuries, 377
residual limb (after amputation), 459, 459f
elimination constant, 62
embolism
fat, with fractures, 499
pulmonary, with fractures, 499
emphysema, 86–87
subcutaneous, 115
employment (occupation; work)
asking questions relating to, 208
blue and black flags and, 389
functional tests in rehabilitation for, 296
health affected by conditions of, 189
empowerment, service user, 192–194, 194b, 200–201
empty can test, 235
empyema, 113–114, 171
end-feel with passive movements, 213
end-range squats, 327
endosteal proliferation, fracture, 497–498
energy, 360
conservation, 361
costs, leisure activities, 156t
kinetic, 361
linear, 360
potential, 360–361
'energy-based' window for electrotherapy, 419
Entonox in labour, 611
environment
amputee difficulties relating to, 460
asthma management, 101
as disability barrier, 192
working, affecting health, 189–190
enzymes
as drug targets, 55f
in soft tissue injury, 255
epicondylitis, lateral (tennis elbow), 491, 577
epidural analgesia/anaesthesia
childbirth, 611
thoracic surgery, 176
epilepsy
drug management, 61
post-traumatic, 598
epinephrine (adrenaline), 63
epiphysis, fractures affecting, 500
episiotomy, 611
equality training, disability, 195
equilibrium, static, 345
equipment for sport events, 375, 378
Equity and Excellence: Liberating the NHS, 27
ethical issues, 5–6
older people and Parkinson's patients, 544
ethnic groups/minority groups
cardiac rehabilitation, 149
culture and, 187–188
eversion, foot, 248, 248b
evidence-based practice (incl. evidence from research), 10–12
neurological patients, rehabilitation, 587
rehabilitation in critical care setting, 142–143
see also research
examination *see* assessment; physical examination
EXCITE trial, 588
exclusion, 197
excretion, drug, 57
exercise (and physical activity), 273–303
abdominal, post-gynaecological surgery, 622
acute respiratory distress syndrome, 116
ankle arthroplasty, 537
breast cancer surgery, postoperative, 624
in cardiac rehabilitation *see* exercise training
compliance *see* compliance
coronary heart disease and levels of, 148
femoral shaft fractures, 520–521
group, 298–299
hand, massage practitioner, 476
hip arthroplasty, 534–535
knee arthroplasty, 532
lymphoedema, 626
mobilising *see* mobilisation
movement dysfunction and value of, 286–287
neurological patients, 587–588
older people, 552–554
in falls prevention, 554
outpatient physiotherapy-led programmes of, after intensive care, 143
in pain management
graded, 393–394
pacing, 393
Parkinson's disease, 552–554
case study of prescription, 556t
in falls prevention, 554
pelvic floor *see* pelvic floor muscles
in pregnancy, 614–618
advice, 614, 616–617
benefits, 616
contraindications, 616
types, 617
prescription, 300–301
in cardiac rehabilitation, 154–157
soft tissue injuries, 263
progression *see* progression
shoulder *see* shoulder
soft tissue injuries, 267
grading level of activity, 263–265
prescription, 263
strengthening *see* strength
thoracic surgery, postoperative, 178–179
see also breathing control; fitness; retraining/re-education; sports; thorax, expansion exercises
exercise tolerance
in bronchiectasis, increasing/maintaining, 105
in COPD
increasing/maintaining, 93
reduced, 90
in cystic fibrosis, increasing, 109
in pneumonia, increasing, 112
test (in cardiac rehabilitation), 154, 156, 158
exercise training in cardiac rehabilitation, 152
contraindications, 154, 159, 162
definition (of exercise training), 152b
prescription, 154–157
assessment for, 154
and delivery across four phases of cardiac rehabilitation, 164–166
programming, 158–162
risk stratification, 157–158
special populations, 162–164
exertion, rating of perceived, 155–156
expectation of pain, 386
expert opinion, 11
expiration, 130, 132
COPD, 88
emphysema, 87
with inhalers, 102
see also forced expiration technique; forced expiratory volume in one second; peak (expiratory) flow; positive end-expiratory pressure

extension
cervical, 226–227, 318t
hip, 239
eccentric control, 292f
knee, 219, 336, 362
osteoarthritic versus normal knee, 341
resisted, 220f
in slump test, 567
lumbar, 216–217
repeated, 218
shoulder, 233
horizontal, 232
extensor strengthening exercises, shoulder, 286f
external fixation, 504
tibial fracture, 516
extracorporeal shockwave therapy, 448–450
extraneural dysfunction, 562
exudates
inflammatory, 262
pleural, 113

F

FABER (flexion plus abduction plus external rotation) test, 219
faeces (stools)
in cystic fibrosis, 107
incontinence (anal/stool incontinence), 582, 620–621
obstetrically-related, 618
see also constipation; defaecation
fall(s)
amputee, 468
older people, 550–551
femoral neck fracture, 513–514
physical activity and exercise in prevention of, 554
Parkinson's patients, 550–551
prevention, 554, 556t
fallopian tubes, 607
Faradism, 429
fascia
lift and roll, 487
muscle *see* myofascia
fasciectomy, Dupuytren's contracture, 257b
fast-twitch (type IIa/IIb) muscle fibres, 130, 278, 279t, 317
fat embolism with fractures, 499
fatigue, 582
multiple sclerosis, 594–595, 594b
respiratory muscle, 132–133, 135
fatigue fracture *see* stress fracture
fear–avoidance beliefs (with pain), reducing impact, 394, 397
feedback
360°, 50
patient, 15
feel *see* palpation
feet *see* foot
females *see* women
femoral nerve stretch (prone knee bend), 221, 570
femur
amputation through, 469f
gait deviations, 469
hip contractures, 464
prostheses *see* prostheses
fracture, 513–515
case study, 519b–522b
condylar, 515
neck, 513–514
shaft, 515, 519b–522b
in knee arthroplasty, prosthesis component, 536
Femurett, 465
fentanyl, 60, 64
ferrous sulphate, 64
fertility problems (male) in cystic fibrosis, 107
fetus, 608
see also childbirth
FEV_1 *see* forced expiratory volume in one second
fibrosing alveolitis, 117
fibrous repair phase of healing *see* healing and repair
fibular fracture, 504f, 515–516
fight or flight response, 56
fingers
arthroplasty, 528–529, 531f
clubbing *see* clubbing
fractures, buddy strapping, 502
see also thumb
first aid, sports, 369–371, 374–377
first pass metabolism, 62
fitness, physical/cardiopulmonary/cardiovascular
athletes, 258b
in exercise training for cardiac rehabilitation, 158
maintenance and improvement
cystic fibrosis, 109
pneumonia, 112
in risk stratification for exercise-based cardiac rehabilitation, 158
FITT principle (frequency/intensity/time/type) in exercise training, 154–157
in special patient groups
arthritis, 164
diabetes, 163
hypertension, 163
obesity, 164
older adults, 163
peripheral vascular disease, 163
respiratory conditions, 164
transversus abdominis rehabilitation, 317
fixation (fracture), 499–500, 503
femoral shaft fracture, 515
tibial/fibular fracture, 515–516
time to union with, 499
flags, 389
low back pain, 224b
flat back, 216
flexibility
in communication, 196
in movements, relative, 310–311
flexion
cervical/neck, 226, 318t
passive, assessment, 220, 566
in slump test, 567
cranio-cervical flexion test, 320
hip joint, 219, 239
contractures, amputees, 464
and knee, 240f
knee, exercises increasing, 285
knee, passive, 243
and hip, 240f
knee, in walking/gait, 241b, 243, 335–336, 362
amputee, abnormalities, 468
osteoarthritic versus normal knee, 341
lumbar, 216
repeated, 218
plantar, 219–220, 220f, 248, 248f–249f, 335
shoulder joint, 231–232
exercises with, 285
horizontal, 232
thoracic, in slump test, 567
see also dorsiflexion; FABER test; lateral flexion; side-flexion; sitting flexion; standing flexion
flexors, cervical/neck, deep, 318
assessment of impairment, 320
fluid displacement test, knee, 242
see also effusion; exudate; oedema; swelling
fluoxetine, 64
foot, 246–250
assessment, 246–250
fractures, 517
Football Association (FA), 369–370
force(s), 344–350
on body, 346–349, 354–359
ground reaction *see* ground reaction forces
horizontal *see* horizontal force

joint *see* joint
methods of analysis, 350
moment of *see* moment
muscle *see* muscle
resolving (in calculation of moments about joints), 354–355, 358–359
understanding, 344–345, 350–351
in ventilation, opposing, 131–132
vertical *see* vertical force
force platforms, 350
forced expiration technique (FET)
bronchiectasis, 105
COPD, 94
thoracic surgery patient, 178
forced expiratory volume in one second (FEV_1)
asthma, 97
COPD, 85
cystic fibrosis, 107
forced vital capacity (FVC)
asthma, 97
positioning improving, 179b
forceps delivery, 611
forefoot examination, 247–248
forward head (poking chin) posture, 225, 226f, 319–320
four-pole interferential therapy, 426–427
'four' test (Faber test), 219
fractures, 495–523
causes, 496
classifications, 495–496
and time to union, 499
clinical features, 496–497
commonly encountered sites of, 510–517
complications, 499–501
see also specific fractures
definition, 495b
healing, 497–499
low-intensity ultrasound therapy and, 440
time for full healing, 498–499
humeral *see* humeral fracture
management, 501–506
case study, 519b–522b
physiotherapy, 506–510
principles, 501–506, 510–517
pathological, 496
traumatic brain injury combined with, 599
France, acupuncture, historical use, 403
free active exercises *see* active exercises
free-body analysis, 345
freezing of gait in Parkinson's disease, 549–550, 554
frequency (stimulation/pulse) in electrotherapy
frequency windows, 419
interferential therapy, 424–426
sweep patterns, 427
TENS, 421, 423
selection, 424
friction massage technique, 488
Achilles tendonitis, 491
tennis elbow, 491
frictional forces opposing airflow, 132–133
function, body (and functional capacity)
amputees, assessment, 461t, 470
in exercise-based cardiac rehabilitation, in risk stratification, 158
fractures and loss of, 497
in neurological patients, 583
measures of, 584–586
older people, in outcome measures, 553t
pain impact on, measurement, 389–390
Parkinson's disease, in outcome measures, 553t
return to *see* discharge
in WHO ICF, 387
promoting optimal function (in pain patient) with reference to, 390
functional bracing (cast brace), 503
femoral shaft fracture, 519–521
functional disability *see* disability
functional electrical stimulation (FES), 431, 589–590
functional instability, 288–289
potential causes, 273
structural instability and, 289b
Functional Measure of the Amputee, 470
functional MRI, acupuncture research, 404
functional tests (incl. movements), 286
ankle/foot, 248
fractures, 509
hip, 240
and rehabilitation, 286
shoulder, 233
functional tools in strength assessment, 275
furosemide, 64

G

G-coupled protein receptor, 62
gabapentin, 64
gait (walking manner), 331–368
amputee
assessment with prosthesis, 461t
deviations, 468–469
analysis, 331–368
development of equipment, 343–344
ankle/foot and, 246, 359
cycle, 332
ground reaction forces during, 346–349
joint moments during, 359–360
joint movements during, 333–338
joint power during, 362
hip joint and, 235–236, 359–360
knee joint and, 241, 359
neurological patient, examination, 583
older people, 549–550
Parkinson's disease, 549–550
freezing of, 549–550, 554
spatial and temporal parameters, 332–333
GAITRite™ system, 343
Galeazzi fracture, 511
older people, 549–550
Parkinson's disease, 549–550
gallow's traction, femoral shaft fracture, 515
Garden classification of femoral neck fracture, 513
gastrointestinal tract
brain injury and, 598–599
in multiple sclerosis, 594
NSAID adverse effects, 60
gate control theory of pain, 391, 407
genitourinary dysfunction, 618–620
see also reproductive tract
GENTLE, 589
genu recurvatum, 241
genu valgum, 241, 515
genu varum, 241
German Acupuncture Trials (GERAC), 408
Gibbs' model of reflection, 80
Glasgow Coma Scale, 598, 598t
glenohumeral joint
assessment, 232–234
muscle acting on (and muscle imbalance and movement faults), 306–308, 321–323
gliclazide, 64
glucocorticoids *see* steroids
glyceryl trinitrate, 58, 64
glycosides, cardiac, 57–58
goals (agreeing and setting)
fractures, 509–510
neurological patient, 586
Parkinson's disease, 546, 551–552, 555, 556t
older people, 546
falls, 551

Golgi tendon organ, 288t, 289f
goniometer, 355–356
  knee, 243
governance *see* clinical governance
graded activity versus graded exposure in pain management, 393–394
gravity
  acceleration and, 345
  active-assisted exercises and, 274
greenstick fractures, 495
grief process, amputation, 459b
ground reaction forces (GFRs), 345–346
  in gait cycle, 359–360, 362
  lower limb moment calculation and, 354–355, 357–358
  methods of showing, 350
groups
  exercise in, 298–299
  in pregnancy
    antenatal, 617–618
    postnatal, 618
growth, fractures affecting, 500–501
guidelines *see* clinical guidelines
gym ball, amputee, 464
gym work in pregnancy, 617
gynaecological disorders, 618–620
  surgery *see* surgery

H

hacking, 485
haematological complications of fractures, 499
haematology (tests)
  pneumonia, 111
  tuberculosis, 119
haematoma, fracture, 497
Haemophilus influenzae pneumonia, 110
haemoptysis
  bronchial/lung cancer, 120
  bronchiectasis, 104
  cystic fibrosis, 93–94
haemorrhage, brain/intracranial
  stroke due to, 591
  subarachnoid, 591–592
half-life, 62
hallux valgus and rigidus, 248
hamstrings
  eccentric training, 277–278
  injury, 264b, 267b, 269b
    neurodynamic assessment and treatment, 577
  length assessment, 236–237
hands
  arthroplasty, 528–529
  fractures, 512–513
  massage practitioner's, 476–478
  reaching task, kinematics, 339–340
head
  forward head (poking chin) posture, 225, 226f, 319–320
  injury, 310–311
    management, 599
    sports, 372–373
    see also brain
headache, acupuncture, 408
healing and repair (tissue - primarily soft tissue), 253–271
  case studies, 253, 269b–270b
  continuum of, 254–255
    injury severity and progression through, 260
  electrotherapy in, 265
    pulsed shortwave therapy, 443
    ultrasound, 437
  fracture *see* fractures
  massage too early in process of, 480
  phase 1 (bleeding - 0-10 hours), 254–255
    physiotherapy interventions, 256–260
  phase 2 *see* inflammation
  phase 3 *see* proliferation stage
  phase 4 *see* remodelling stage
health
  behaviour and *see* behaviour
  definitions, 188, 188b
  inequalities, 188–190, 199
  promotion, 198–202
  women's *see* women
  see also International Classification of Functioning, Disability and Health
Health and Care Professions Council (HCPC - formerly the HPC), 3, 7–8
  interprofessional education and, 27
  reflective practice and, 71
Health and Safety (First-Aid) Regulations (1981), 369
health outcomes of care, evaluation *see* outcome measures
Health Professions Council *see* Health and Care Professions Council
Health Professions Order, 3
health services *see* services
healthcare *see* care
hearing (auditory) disturbances, neurological conditions with, 582
heart
  adverse events during exercise-based cardiac rehabilitation, 157
  drugs acting on, 57–58
  rate, in exercise training, 154–155
  tamponade, acupuncture needling and risk of, 406
  transplantation, 149
  valve surgery, 150
heart disease
  congenital, 150
  coronary *see* coronary artery disease
  older people, 546t
  rehabilitation *see* rehabilitation
heart failure (chronic), 162
  cardiac rehabilitation, 148–149
    exercise-based, 162
  in cor pulmonale, 88, 90f
heat/heating *see* humidification *and entries under* thermal
heel
  lift, 335–336, 347–348
  strike, 334–336, 346–349, 357–360, 362
hemianopia, 581
hemiarthroplasty
  hip, 514
  humeral (proximal) fracture, 526
heparin, 64
hepatic… *see* liver
heroin (diamorphine), 60
hierarchy of evidence, 11
High Quality Care for All: Next Stage Review Final Report, 42–43
High Quality Workforce: Next Stage Review Report, 42–43
hindfoot examination, 247–248
hip (hip joint), 235–240
  angle, 333
  assessment, 218–219, 235–240
  contractures in amputees, 464
  extension *see* extension
  flexion *see* flexion
  fracture, 513–514
  hemiarthroplasty, 514
  knee versus, as source of symptoms, differentiation, 241b
  lumbar spine versus, as source of symptoms, differentiation, 218–219, 237–238
  moments, 358
    in gait, 359–360
  motion in sagittal plane, 336–337
  pain referred to knee, 208
  power during walking, 362
  total replacement (arthroplasty), 531–535
    complications, 533
    revision surgery, 535
histo(patho)logy, lung cancer, 120, 120t, 169–171
history
  acupuncture, 403–404

health promotion, 199
interprofessional education, 24
mechanical ventilation, 135
movement analysis systems, 343–344
profession of physiotherapy, 2–3
history-taking (clinical interview), 210–211
amputee, 461t
fractures, 508
neurological patients, 583
Parkinson's disease, 546–547
older people, 546–547
pain patient, 388t
hi-TENS, 423
hold/relax techniques, 284
home, return to *see* discharge
horizontal flexion and extension of shoulder joint, 232
horizontal forces, 345
knee, 355
upper limb, 357
hormones in pregnancy, 608
hospital
cardiac rehabilitation as in-patient (=phase I), 150, 164
discharge *see* discharge
outpatient *see* outpatient
postnatal physiotherapy as in-patient, 618
hospital-acquired (nosocomial) pneumonia complicating acute respiratory distress syndrome, 116
hot packs, 433
human relations, 196
humeral fracture, 510–511
complex, hemiarthroplasty, 526
condyle, 510–511
neck, 510
proximal, 510
shaft, 510
humidification (incl. heated humidification)
COPD, 94
cystic fibrosis, 109
thoracic surgery patient, 180
hydrocephalus, post-traumatic, 598
hydrocollator packs, 433
hydrocortisone, 64
hydrotherapy *see* water-based exercises
5-hydroxytryptamine, 61
hyperalgesia, 385, 385b
hypercapnia, chronic, 122
hypercapnic (type II) respiratory failure, 121–122, 133–134
hyperinflation
manual, 140–141
pathological, adverse effects, 106–107, 134–135
hypertension, exercise-based cardiac rehabilitation and, 163
hypnotics, 59
hypokinesia, Parkinson's, 541
hypotonia, 581
hypoventilation, alveolar, 133–134, 134t
hypovolaemic shock, fractures, 497
hypoxaemic (type I) respiratory failure, 121–122, 133
hypoxic drive, 135

I

ibuprofen, 53b, 60, 64
ice burns, 258
ice massage, 479
ice therapy *see* cryotherapy
identity, professional, 33–34
iliac spine
anterior superior, 315–316
posterior superior, 315
iliopsoas, 314t
problems, 314t
restriction (versus rectus femoris restriction), 236
iliotibial band, 236
Ilizarov method, 505b
imaging
brain, acupuncture research, 407–408
thoracic surgery, preoperative, 171
see also specific modalities
immobilisation
fracture, 502–506
proprioceptive deficits with, 287t
immotile cilia syndrome, 103
immunisation, tuberculosis, 119
immunology, tuberculosis, 119
impact of pain, measures, 388
impairments, 581, 583–584
definition, 192
as outcome measures, 585t
impingement, shoulder, 232, 232b, 322–323
test, 235
implantable cardioverter-defibrillator, 147
incentive spirometry, 179
inclusion, 194–195, 194b, 197–198
income, low *see* socioeconomic status
incontinence *see* faeces; urinary incontinence
incremental walking programme, 164, 165t
independence
amputees, 464, 464t
definition, 191, 191b
individual *see entries under* person; personal
inertia, 344
infants and babies, cystic fibrosis, 106–107
treatment, 107, 109
infections
fractures and risk of, 499
tibial/fibular, 516
respiratory *see* respiratory infections
skin *see* skin
see also antibiotics
inferior draw (sulcus) test, shoulder, 234
infertility (male) in cystic fibrosis, 107
inflammation, 253–271, 391
acute
massage contraindicated, 480
pulsed shortwave therapy, 443
in soft tissue injury *see subheading below*
ultrasound therapy, 437–439
management, 391
peripheral mediators, 384f, 385
pharmacological management *see* anti-inflammatory drugs
in soft tissue injury (phase 2 of healing and repair), 255–256
case study of ankle sprain, 395–396
physiotherapy interventions, 256–260
see also anti-inflammatory effects
inflammatory arthropathies *see* arthritis
influence, spheres of, 47–48
influenza, 110
information *see* education
informed consent *see* consent
infrared radiation, 433
inhaled analgesia in labour, 611
inhalers
asthma, 99
technique, 101–102
COPD, 92–93
injury, traumatic *see* trauma
In-Motion, 589
inpatient *see* hospital
in-shoe pressure analysis, 350
insidious onset of condition, 210
inspiration, 130–131, 131f
with inhalers, 102
muscles involved, 132
training, 93–94
instability, 288–289
assessment *see* stability
postural, in Parkinson's disease, 541–542, 548
institutionalised discrimination, 186, 187b

instrumental (assisted) vaginal delivery, 611
insulin, 64
insurance (medical) and sports physiotherapy, 372
intensity of stimulation in TENS, 424, 431
intensive care, rehabilitation in/after, 141
intercostal drains, 174–175
interface treatment (neurodynamic), 575
interferential therapy, 424–429
interferon-β, multiple sclerosis, 595
intermittent positive pressure breathing/ventilation
  COPD, 94–95
  thoracic surgery patient, 180
internal fixation, 503
  tibial/fibular fracture, 516
International Classification of Functioning, Disability and Health, 387, 543–544, 583–584
  promoting optimal function with reference to, 390
International Classification of Impairment, Disability and Handicap (1980), 192
international dimensions of interprofessional education, 25–26
International Organization of Physical Therapists in Women's Health, 1–2
interphalangeal joint, foot, assessment, 250
interpreters (language), 195–196
interprofessional education, 23–39
  collaborative working and, 28–29
  definitions, 24, 24b
  history, 24
  international dimensions, 25–26
  key points for success, 29b
  in practice, 29–33
  technology and, 31
  UK context, 26–29
interprofessional networks, 50
interprofessionalism, 33–34
interval training in exercise-based cardiac rehabilitation, 160
intervertebral movements, passive physiological
  cervical spine, 227
  lumbar spine, 218–219
interview
  clinical *see* history-taking
  pain patient, 387
    case studies, 395–397
intestine *see* bowel; faeces; gastrointestinal tract
intra-articular adhesions with fractures, 500–501, 522
intra-articular fractures, 500
intracranial haemorrhage *see* haemorrhage
intramedullary nailing, 504
intraneural dysfunction, 562
invasive ventilation, 135–136
inverse agonist, 62
inverse care law, 190b
inversion, foot, 248, 248b
investigations (previous), recording results, 211
involuntary movements, Parkinson's, 541
ion channels as drug targets, 55f
ionization of drugs, 55, 62
iPAM, 589
irritability of condition, determination, 210
ischaemia
  cerebral *see* ischaemic stroke; transient ischaemic attack
  in soft tissue injury, 255
ischaemic attack, transient, 593
ischaemic stroke, 591
isokinetic assessment (machines/dynamometer), 213, 275, 289–290, 367–368
isometric contraction and forces, 276t, 277–278, 362
isotonic shortening and lengthening, 276b

J

jargon, 197–198
Johns' model of reflection, 80
joints (articulations)
  adhesions in/around, with fractures, 500–501, 522
  angle, definition/calculation, 333–334
  assessment (peripheral/non-spinal), 230–250
  degenerative disease *see* degenerative joint disease; osteoarthritis
  forces (and their calculation), 355–356
    spine (base), 358–359
    upper limb, 356–357
  fractures involving, 500
  inflammatory disease *see* arthritis
  moments, 354–360
    body segment inclination and, 364
    elbow, 354, 356, 366
    lower limb, 354–355, 357–360
  movements *see* mobilisation; movements; range of movements
  pelvic, female
    anatomy, 605–606
    in pregnancy, dysfunction, 614
    in pregnancy, normal changes, 605–606, 608–609
  power during walking, 362
  receptors in proprioception, 288
  replacement *see* arthroplasty
  sensing of position and movement *see* proprioception
  stability *see* instability; stability

K

Kartagener's syndrome, 103
Karvonen formula, 155
kinematics, 332–333
  hand in reaching, 338–340
  knee in walking, 340–342
  shoulder complex, 325
kinetic chain exercises, 273, 290
kinetic energy, 361
kinetics, definition, 346
Klebsiella pneumoniae, 110
kneading, 481–483
knee (joint), 325–327
  angle, 333
  assessment, 241–243
    with femoral shaft fractures, 519
  bend, prone, 221, 570
  contractures in transtibial amputation, 464
  extension *see* extension
  flexion *see* flexion
  fractures around, 515
  moments, 355, 358–359
    in gait, 359
  motions and their analysis (in walking), 335–336, 341
  muscle imbalance and, 325–327
  osteoarthritis
    acupuncture, 408
    kinematics, 341–342
  power during walking, 362
  replacement, 535–537
knock-knees (genu valgum), 241, 515
knowledge
  clinical reasoning and, 69
  of self reflective practice, 77
  specific to profession, possessing, 4–5
  sports physiotherapy, 370–374
Kolb model of reflection, 79
kypholordosis, 216

L

labelling people, 183b
labour, 609–612
  1st stage, 610
  2nd stage *see* delivery
  3rd stage, 611
  definition, 609b
  induction, 611
  onset, 609–610
  see also childbirth
Lachmann's test
  knee joint, 211, 245
  shoulder, 234
lactation, 612
language
  as communication barrier, 187
  inclusive, 197–198
  interpreters, 195–196
lansoprazole, 64
large cell lung cancer, 120t
Lasègue's *see* straight leg raising test
laser phototherapy, bronchial/lung cancer, 120
laser therapy, 444–448
lateral epicondylitis (tennis elbow), 491, 577
lateral flexion in cervical spine, 318t
lateral ligaments (lateral collateral ligaments)
  ankle
    assessment, 248
    injury, 261b, 268b
  knee, assessment, 244
lateral meniscus tests, 245
lateral sclerosis
  amyotrophic, 596
  primary, 596
lateral shift (lumbar spine), 216
law *see* legal issues
LDL cholesterol, drugs lowering levels of, 58
leadership (clinical), 41–52
  case for, 47
  competency development, 48–50
  context, 42–43
  role and service improvement, 45–47
learning cycle, Gibbs', 79f
  see also education and learning
leg *see* lower limb
legal issues (law/legislation/regulations), 7–8
  ethical older people and Parkinson's patients, 544
  massage, 479
  sports management, 369–372
leisure sports *see* sports
length
  leg, discrepancy, 236
  of lever (in exercise), changing, 285–286
  muscles
    adaptations, 309
    increasing (=lengthening) in management of imbalance, 312t
  muscles, tests, 214
    hamstrings, 237f
    hip, 237
    muscle imbalance and, 311t
    shoulder, 231, 233
  step, 332
  stride *see* stride
leukotriene antagonists, asthma, 98
levator scapulae, 321–322
  length test, 231
lever (in exercise), changing length, 285–286
levodopa, 59
life-long care, amputee, 463t, 467–468
life-long learning (LLL), 16
  definition, 16
  emphasis on, 16
lifestyle
  active, return to *see* discharge
  coronary disease risk factor, 147
  older people and Parkinson's disease and impact on, 544b
lift and roll, fascial, 487
ligaments
  ankle
    assessment, 246, 248–249
    injury, 246, 257b, 264b, 268b
  differentiation tests, 214
  knee
    assessment, 243–245
    differentiation tests of menisci and, 214, 245
  lumbar spine, anterior and posterior, assessment, 212, 219
  pelvic, in pregnancy, 608
  see also specific named ligaments
light absorption by tissues in laser therapy, 446
limb(s)
  amputation *see* amputation
  amputees' remaining (non-amputated)
    assessment, 461t
    teaching how to care for, 466–467
  amputees' residual (after amputation)
    assessment, 461t
    contractures, 464
    oedema, 464
    pain, 459
    poor condition, 460
    shape, 464b
    teaching how to care for, 466–467
  children with absence of, 470
  elevation *see* elevation
  proprioceptive exercises, 291–293
  see also compartment syndrome; lower limb; upper limb
limbic system
  acupuncture and, 409
  damage, 582
linear acceleration, 338
  reaching task, 339–340
linear displacement, 338
  reaching task, 339
linear energy, 360
linear models of communication, 184
linear power, 360
linear velocity, 338
  reaching task, 339
linear work, 360
LIPUS (low-intensity pulsed ultrasound) and fracture healing, 439–440
liver
  cystic fibrosis-related disorders, 107
  drug handling, 57
  metastases from lung cancer, 120
living conditions *see* socioeconomic status
load
  excess (overload) in muscle strengthening, 274, 279
  muscle strength evaluation and, 364
lobes of lung, 169
  excision (lobectomy), 173
lobular pneumonia, 111
local anaesthetics, 61
Locomotor Capabilities Index, 469
look *see* observation
loop diuretics, 58
lordosis, 216f
  cervical, assessment, 288
  lumbar, transfemoral amputee, 469
  see also kypholordosis
lo-TENS, 423
low back pain (LBP), 223–225
  acupuncture, 408
  assessment, 223–225
  case study, 396–398
  management guidelines, 224
low-density lipoprotein cholesterol, drugs lowering levels of, 58
low-intensity laser therapy (LILT), 444–448

low-intensity pulsed ultrasound and fracture healing, 439–440
low-level laser therapy (LLLT), 444–448
lower limb (and leg)
  alignment, 247
  amputation *see* amputation
  arthroplasty, 531–537
  circulatory assessment, 246
  fractures, 513–517
  girth, assessment with femoral shaft fractures, 521t
  length discrepancy, 236
  moments about joints in, 354–355, 357–360
  neurodynamic tests, 568–570
  power in joints of, 362
  proprioceptive exercises, 291–292
  walking *see* walking
  see also dead leg; straight leg raising test
lubricant, massage, 478–479
lumbar spine, 215–225
  assessment, 215–225, 237–238
    case study, 223b
  lordosis, transfemoral amputee, 469
  in pregnancy, 608
    pain, 612–613
lumbopelvic region, 312
  pain in pregnancy, 612–615
    management, 614–615
  stability, 312, 314–315, 606
    testing, 222
  see also pelvis
lung(s)
  abscess, 117–118
  acupuncture needle injury to, 406
  anatomy, 169, 170f
  clearing fields *see* secretion
  compliance, 132
  in cystic fibrosis, 106
  disease (in general), opposing forces in, 132
  drugs acting on, 58–59
  emphysematous changes, 87f
  expansion
    elastic opposition to, 131–132
    re-expansion in pneumonia, 112
  interventions by respiratory physiotherapist, 140–141
  obstructive disease *see* obstructive pulmonary disease
  oedema
    acute respiratory distress syndrome, 116
    cor pulmonale, 88
  operations, 173
    complications, 169
    resection (pneumonectomy), 171, 173
  rehabilitation *see* rehabilitation
  restrictive disease *see* restrictive pulmonary disease
  tumours, 119–121, 169–171
    imaging, 171
  ventilator-associated complications, 137–139
  see also respiratory disease
lung function tests
  asthma, 97
  COPD, 83–84
  cystic fibrosis, 107
  thoracic surgery, preoperative, 171
  see also spirometry
lying
  shoulder flexion exercise, 285
  supine, hip examination, 236
  see also prone knee bend
lymph nodes in breast cancer, 624–625
lymphatic massage, lymphoedema, 491, 616
lymphoedema, 625–626
  manual lymphatic drainage (by massage), 491, 616
  women, 625–626
    cancer patients, 625

M

McConnell critical test, 243
McGill Pain Questionnaire, 388
McMurray's medial and lateral meniscus tests, 245
magnetic field in pulsed shortwave therapy, 442
magnetic resonance imaging (MRI)
  functional, acupuncture research, 407
  records of previous MRI, 211
malalignments *see* deformities
males *see* men
malignant multiple sclerosis, 594
malignant tumour *see* cancer
malleolar fracture, 516, 516f
manipulation techniques in massage *see* massage
manual hyperinflation, 140–141
manual lymphatic drainage (massage), lymphoedema, 491, 616
manual techniques/therapy
  lumbopelvic pain in pregnancy, 614
  respiratory (incl. ventilated) patients, 141
march fracture, 517
mass, 345
  centre of, 333, 347–348, 356, 358
  distinction from weight, 345
massage, 475–493
  contraindications, 479–480
    see also specific massage techniques
  legal aspects, 479
  preparations, 475–479
  specific usage, 489–491
    lymphoedema (manual lymphatic drainage massage), 491, 616
  techniques (manipulations), 480–486
    newer/other, 487–489
  see also micromassage
match day
  officials, 378
  pre-match and pitch-side considerations, 375
mechanical instability, 288
mechanical noxious stimuli and nociception, 382
mechanical tension tests
  cervical spine, 229
  lumbar spine, 220–222
mechanics *see* biomechanics
mechanosensitivity, 562–563
  cause of increases, 566
  treatment reducing, 575–576
meconium ileus in cystic fibrosis, 107
medial kinetic rotation test, 208b
medial ligaments (medial collateral ligaments)
  ankle, assessment, 249
  knee, assessment, 243–244
medial meniscus tests, 245
medial nociceptive system, 383
median neurodynamic test (MNT)
  MNT1, 570–572
  MNT2, 572
medical approach to health promotion, 189
medical history, past/previous, 211
  amputee, 461t
  fractures and, 508
  pain patient, 388t
medical insurance and sports physiotherapy, 372
medical management
  brain injury, 599
  motor neurone disease, 596–597
  multiple sclerosis, 595
  stroke, 592
  see also drug therapy
medical model of disability, 191
medical record, problem-oriented, 507
Medical Research Council Dyspnoea Scale, 85t
medication *see* drugs
medicine ball work, 295
membrane (cell), pulsed shortwave therapy effects, 442–443
men (males)
  infertility in cystic fibrosis, 107
  lower urinary tract symptoms, 620

meniscal assessment, 214, 245
menopause, 623
  psychosexual issues, 626
Mental Capacity Act (2005), 544b
mental health problems *see* psychosocial disorders
mental practice of upper limb movement following stroke, 70b
mentoring/mentorship, 50
  reflection and, 74–75
meperidine *see* pethidine
meridian points (acupuncture), 404–407, 409–410
  pressure on, 489
metabolic equivalents and exercise prescription in cardiac rehabilitation, 156, 158
metabolism (drug), 57, 62
  first pass, 62
metacarpal fracture, 512–513
metacarpophalangeal joint
  replacement, 528–529
  splinting, 512–513, 529
meta-cognition and clinical reasoning, 69
metastases (spread of cancer)
  bronchial/lung cancer, 120
  massage and risk of, 480
metatarsal fracture, 517
metatarsalgia, Morton's (Morton's neuroma), 249
metatarsophalangeal joint, assessment, 250
metered-dose inhalers (MDIs), 99
metformin, 64
methadone, 60
methotrexate, 64
metoclopramide, 64
microbiology
  pneumonia, 111
  tuberculosis, 119
microcurrent therapy, 431
micromassage, ultrasound, 437
microthermal modalities of electrotherapy, 419–420
microwave diathermy, 433–434
midtarsal joint assessment, 248
migraine, acupuncture, 408
mini-tracheostomy, 179–180
MIT-MANUS, 589
mobilisation (mobilisation exercises), 280–284
  in cardiac rehabilitation, 159
  classes of exercises, 280–284
  myofascial, 487–488
  neural tissue, 575–576
  postoperative
    amputation, 463
    ankle replacement, 537
    hip replacement, 534
    knee replacement, 536–537
    thoracic surgery, 179
  soft tissue injuries, 267–268
  sports, 377
mobilisation (myofascial), 487–488
mobiliser muscles, 306, 309
  cervical, 319t
  glenohumeral joint, 322t
  lumbopelvic, 312t
  scapulothoracic joint, 321–322
mobility *see* movements
Modernisation Agency (MA), 46–47
moment (torque), 350–351, 354–360
  definition, 350
  joint *see* joint
monochromacity in laser therapy, 445
Monteggia fracture, 511
moral authority (trustworthiness), 6
morphine, 60, 64
mortality (death), coronary heart disease, 147
Morton's neuroma, 247
motions *see* movements
motivation in pain management, 392
motor control of muscle balance, 309
  see also sensorimotor control
motor cortex and proprioception, 288
motor function
  amputee, difficulties in learning new skills, 461
  cardiac rehabilitation in older people and, 163
  disorders/impairments (due to CNS damage), 581
    in brain injury, 599
    in motor neurone disease, 596
    in multiple sclerosis, 594
    in Parkinson's disease, 539–540, 541t, 542, 547
  see also sensorimotor control
motor nerve stimulation in interferential therapy, 428–429
motor neurone disease, 595–597
motor units, 309
movement(s) (motions/mobility)
  accessory *see* accessory movements
  analysis (biomechanical perspective), 331–332
    development of equipment, 343–344
    in gait, 333–338
    methods, 342–344
    specific joints, 334–338
    see also kinematics; kinetics
  assessment, 212
    ankle/foot, 248–250
    fractures, 509
    hip joint, 237–240
    knee joint, 243–246
    neurological patient, 583
    shoulder joint, 231–234
    spine *see subheading below*
  cervical spine, 318t
    assessment, 225–227
  dysfunction, exercises dealing with, 286–287
  in exercise-based cardiac rehabilitation, 159
  falls during *see* falls
  fractures
    abnormal, 497
    assessment, 509
    limitation, 497
  functional *see* functional tests
  inflammation and, 391
  neural tissues
    treatment addressing, 575–576
    versus musculoskeletal tissue, identifying symptoms due to *see* structural differentiation
  Newton's laws, 344–345
  older people, 547–548
    transfers, 548
  in Parkinson's disease, 547–548
    transfers, 548, 556t
    voluntary, dysfunction, 541
  pelvic girdle, restriction in pregnancy, 614
  range of *see* range of movement
  resistance to *see* resistance to movement
  in sensorimotor control rehabilitation, development and enhancement control of, 287–290
  speed of (in exercise), change in, 286
  spine, assessment
    cervical *see subheading above*
    lumbar (in general), 216–219, 237–238
  spine, restriction in pregnancy, 614
  upper limb *see* upper limb
  in walking *see* gait
  see also mobilisation; motor function *and specific movements*
movement disorders, clinical features, 580–581
  see also specific disorders
MRC Dyspnoea Scale, 85t
mucus
  in chronic bronchitis, 86

in cystic fibrosis, excess and viscid, 106
drugs breaking down (mucolytics)
asthma, 98b
COPD, 92
cystic fibrosis, 108
see also secretions
multidisciplinary team *see* team
multifidus, 313, 313f
problems, 314t
multiple crush syndrome, 563
multiple sclerosis, 592–595
muscle, cardiac, drugs increasing contractions, 57–58
muscle, skeletal
abdominal *see* abdominal muscles
agonists, 306
antagonists, 306
atrophy/bulk loss, 581
with fractures, 497, 499, 501
progressive, 596
thigh, 241–242
balance
loss *see* imbalance *(subheading below)*
neurophysiological components, 309–311
classification of roles, 306, 307t
contraction *see* contraction
contractures *see* contractures
in COPD, loss of mass, 90
damage with fractures, 499
delayed-onset soreness, 273, 276–277
differentiation test, 214–215
dysfunction in neurodynamic tests
median neurodynamic test 1 (MNT1), 570–571
median neurodynamic test 2 (MNT2), 572
prone knee bend, 570
radial neurodynamic test, 573–574
slump test, 567
straight leg raising test, 568
straight leg raising test modified, 569
ulnar neurodynamic test, 573
fascia *see* myofascia
forces (and their calculation), 355
spine (base), 358–359
upper limb, 356–357
imbalance, 305–330
assessment/examination, 305b, 311–317
definition, 305–306
insertion points (and relevance to strength evaluation), 364–366
length *see* length
as mobilisers *see* mobiliser muscles
pelvic floor *see* pelvic floor muscles
pull angle (relevance to strength evaluation), 366
reciprocal inhibition, 264, 284
re-education in childbearing year, 614–615
resisted muscle testing *see* resistance to movement
respiratory *see* respiratory muscles
spasm/spasticity *see* spasm; spasticity
as stabilisers *see* stabiliser muscles
stiffness/tension, 274
in Parkinson's disease (=rigidity), 541–542, 581
relative, 310–311
stimulation (electrical), in interferential therapy, 428–431
see also neuromuscular electrical stimulation
strength *see* strength
stretching *see* stretch
synergists, 306, 307t
tightness, muscle groups prone to, 214, 225
tone, 274
disturbances (neurological patient), 581
weakness *see* weakness
see also specific named muscles
muscle fibre types, 130t, 278–279, 279t
in respiratory muscles, 130
muscle spindles, 288t, 289f
and proprioception, 287–288
CNS damage effects, 581
musculoskeletal system (and orthopaedics)
assessment *see* assessment
in brain injury, 599
females, 605–608
pregnancy, 608–609, 618
identifying symptoms due to movement of neural tissue or tissues of *see* structural differentiation
older people's conditions of, 546t
Mycobacterium tuberculosis (tuberculosis), 118–119
Mycoplasma pneumoniae pneumonia, 110
myelin loss/damage in multiple sclerosis, 593
myofascia
mobilisation, 487–488
release, 487
spread, 487
trigger points, 488–489
acupuncture and, 409–410
palpation, 229, 231
myositis ossificans, 511
massage contraindicated, 480
myotome tests
cervical spine, 228
lumbar spine, 219

N

nailing, intramedullary, 504
naloxone, 65
naproxen, 65
National Health Service *see* NHS
National Institute for Health and Clinical Excellence (NICE), 14
critical care rehabilitation, 143
National Service Frameworks, 13
nature of condition, determination, 210
nebulisers, 99
asthma, 99
COPD, 92–93
cystic fibrosis, 108–109
neck
flexion *see* flexion
flexors *see* flexors
pain, acupuncture, 408
rotation *see* rotation
wry, 225
need, commitment to assist those in, 5–6
Neer arthroplasty, 525
Neer classification of proximal humeral fracture, 510
neoplastic disease *see* cancer; tumours
nerve(s)
action potentials, 61, 420–421
compression *see* compression
dysfunction
closing versus opening, 562b
extraneural, 562
intraneural, 562
progression of treatment, 576
electrical stimulation *see* motor nerve stimulation; transcutaneous electrical nerve stimulation
injury/damage, 561–563
with acupuncture needles to, 406
with fractures, 501
pain relating to (neuropathic pain), 386
mechanical forces activating *see* mechanosensitivity

needling (acupuncture) and its effects on, 409
see also neurodynamics; peripheral nervous system *and specific named nerves*
nervous system *see* autonomic nervous system; central nervous system; peripheral nervous system; sympathetic nervous system
networks, professional, 50
neural… *see* nerve; neurodynamics; neurological tests
neurodynamics, 561–577
case studies, 577b
neurodynamic assessment/tests, 563–565
contraindications, 563
general tests, 566–567
indications and precautions, 561, 563
interpreting findings, 565–566
lower limb tests, 568–570
upper limb tests, 570–574
neurodynamic treatment, 575–576
neuroendocrine disorders in brain injury, 598
neurogenic detrusor overactivity, 619
neurogenic response in structural differentiation *see* structural differentiation
neuroimaging, acupuncture research, 407–408
neurological disorders/dysfunction (CNS), 59, 579–604
assessment *see* assessment
bladder dysfunction due to, 619
causes, 580
clinical features, 580–582
glossary of terms, 600
drug therapy, 59
interventions, 586–590
lung surgery-related, 173t
older people, 546t
outcome measures *see* outcome measures
neurological tests (incl. neural tests)
cervical spine, 228–229
hip joint, 240
lumbar spine, 219–223
neuromatrix theory of pain/nociception, 384
neuromuscular competence, reduction leading to respiratory failure, 134
neuromuscular electrical stimulation (NMES), 421, 431
neuromuscular system
disease, respiratory muscle failure, 106, 122, 134t
pregnancy, 609
see also muscles; nerves; proprioceptive neuromuscular facilitation
neuropathic pain, 386
neuropathies, cranial, in brain injury, 598
neuropathology, Parkinson disease, 542
neurophysiological components of muscle balance, 309–311
neurotransmitters, 62
Parkinson's disease, 548
neutral zone concept, 308–309
New York Heart Association functional classification of heart failure, 162, 162t
Newton's laws, 344–345
NHS (National Health Service)
leadership and, 42–43
leadership qualities framework (LQF), 42, 44
long-term vision (in 2010), 17
loss of public confidence, 8, 10
outcomes framework, 15–16
services *see* services
NICE *see* National Institute for Health and Clinical Excellence
Nintendo Wii, 466
older people, 553
nitrous oxide + oxygen (Entonox) in labour, 611
NMDA (N-methyl-D-aspartate) receptors and pain, 385
nociception (nociceptive system), 381
activation, 382–384
case studies, 395–396
targeting in pain management, 390–394
sensitisation *see* sensitisation
non-invasive ventilation, 135
COPD, 92, 94–95
motor neurone disease, 597
non-small cell lung cancer, 169, 171
non-steroidal anti-inflammatory drugs (NSAIDs; cyclo-oxygenase inhibitors), 60–61
adverse/side-effects, 60
tissue repair, 440
COX-2-specific, 60
fracture union time and, 499
in soft tissue injury, 253
non-thermal modalities of electrotherapy, 419–420, 420f, 434–450
normality, 192, 192b, 196
nosocomial pneumonia complicating acute respiratory distress syndrome, 116
numbness after breast surgery, 625
numerical rating scales of pain, 387–388
nutrition/diet
brain injury care and, 598–599
soft tissue healing and, 503
nystagmus, 581

O

oat (small) cell lung cancer, 120t
Ober's test, modified, 236
obesity and exercise-based cardiac rehabilitation, 164
objective(s), in goal-setting (neurological patient), 586, 586t
objective assessment, 212–215
aims, 208
amputee, 463t
fractures, 507–509
femoral shaft, 519–520
muscle imbalance, 311t, 315–317
cervical spine region, 320t
shoulder complex, 323
vastus medialis oblique imbalance, 308–309
muscle strength, 213
neurological patients, 583
Parkinson's disease, 546
older people, 546
see also SOAPE process
oblique fractures, 495
oblique muscles, external and internal (EO/IO), females, 606, 606f
in pregnancy, 609
obliquity, pelvic, 337
observation (look)
fractures, 508
general and local, 212
mother in labour, 610
muscle imbalance, 315–316
neurological patient, 583
obstetrics *see* pregnancy
obstructive pulmonary disease, 83
chronic *see* chronic obstructive pulmonary disease
types, 83
see also specific types
occupation *see* employment
ocular muscle dysfunction, 581
odansetron, 65
oedema (fluid accumulation)
ankle/foot, 246
fractures, 497, 501

interferential therapy, 429
lymphatic *see* lymphoedema
pulmonary *see* lungs
residual limb following amputation, 464
see also swelling
oesophagoscopy, preoperative, 171
oesophagus
anatomy, 169
operations, 174
complications, 174
perforation, 171
repair, 174
tumours, 171–172, 174
oestrogen(s)
in menopause
depletion, 623
in symptom management, 623
in pregnancy, 608
oil, massage, 479
older people/elderly, 539–559
assessment, 544–551
cardiac rehabilitation, 149
exercise-based, 163
falls *see* falls
interventions, 551–555
resistance training, 279–280
omeprazole, 65
one (single) compartment model, 63
one repetition maximum (1RM), 279
onset of condition
insidious, 210
traumatic, 210
open kinetic chain exercises, 273, 278, 290
open reduction, 501
and internal fixation, 503
open wound
fractures with, 495
femoral shaft, 515
laser therapy, 447–448
massage contraindicated, 479
Operating Framework (DoH), 47
opioids and opiates, 59–60
childbirth, 611
patient-controlled with, in thoracic surgery, 176
receptors, 59–60, 62
organisations, leaders and their development in, 48–49
oropharyngeal secretions, removal, 141
orthopaedics *see* musculoskeletal system
osteoarthritis
acupuncture, 408
exercise-based cardiac rehabilitation, 164
knee *see* knee
proprioceptive deficits, 287t
shoulder arthroplasty, 525
postoperative routine, 526t
osteoporosis
COPD and, 90–91
cystic fibrosis and, 94
OTTAWA rules, 376
outcome measures (in interventions/rehabilitation), 15
amputation, 469–470
cardiac rehabilitation, 165t
low back pain, 225
neurological disorders, 584–586
Parkinson's disease, 552, 553t
older people, 552, 553t
outpatient
amputee, 464t
cardiac rehabilitation programme (phase III), 151, 159f, 164–165
physiotherapy-led exercise programmes after intensive care, 143
ovaries, 607
overload in muscle strengthening, 274, 279
overpressures
cervical spine, 227
lumbar spine, 222
Oxford Scale, 213, 213b, 274–275
femoral shaft fractures, 520, 520t
oxygen consumption (VO_2)
aerobic exercise, 153–154
functional exercise testing, 158
oxygen tension, arterial (PaO_2), in respiratory failure, 133
type I failure, 121
type II failure, 121
oxygen therapy
adverse effects/toxicity, 138
asthma, 98–102
COPD, 91–92
cystic fibrosis, 93

P

pacing of activity in pain management, 393
pack-years (exposure to smoking risks), 86b
paediatrics *see* children
pain, 381–401
acute versus chronic, 223
pelvic area in women, 622–623
amputation, 459–460
assessment/measurement, 381–387, 395–397
of aggravating and easing factors, 209–210
of area, 208–209
of duration, 209
with fractures, 508, 520
of severity, 209
breast cancer patients, postoperative, 624
bronchial/lung cancer, 120
cardiac *see* angina
case studies, 395–398
definition, 381, 381b
fractures, 497
assessment, 508, 520
hip (as source of pain)
versus knee, differentiation, 241b
versus lumbar spine, differentiation, 218–219, 237–238
knee, referral from hip, 208
low back and pelvis *see* low back pain; lumbopelvic region; pelvis
neck, acupuncture, 408
in neurodynamic tests, 565
median neurodynamic test 1 (MNT1), 570–571
median neurodynamic test 2 (MNT2), 572
prone knee bend, 570
radial neurodynamic test, 573–574
slump test, 567
straight leg raising test, 568
straight leg raising test modified, 568–569
ulnar neurodynamic test, 573
neurological conditions causing, 581
multiple sclerosis, 594–595
Parkinson's disease, 547–548
older people, 547–548
physiology/biology, 381–387, 395–397
gate control theory, 391, 407
patient education, 392
pleuritic *see* pleurisy
pregnancy-related, 612–615
psychological factors *see* psychological factors
quality, 387b
assessment, 388
sensitisation *see* sensitisation
in sensorimotor rehabilitation of spine, 294
shoulder *see* shoulder
social factors *see* socioeconomic status
temporal aspects, 387b
see also complex regional pain syndrome; headache; tenderness

pain management/relief (analgesia), 390–394
in acupuncture, 404, 407, 409
amputated limb, 459–460
in breast cancer surgery, postoperative, 624
case studies, 396–398
electrotherapy
interferential therapy, 428
laser therapy, 448
TENS *see* transcutaneous electrical nerve stimulation
in pelvic pain in women, 622–623
pharmacological *see* analgesic drugs
in pregnancy, 615
in labour, 610–611
in thoracic surgery, 175–176
see also analgesic drugs
palliative care *see* terminal stages
palpation (feel/touch)
acupuncture points, 405
assessment by, 212
cervical spine, 229
fractures, 509
hip joint, 238
lumbar spine, 222
shoulder joint, 231
in massage, 477–478
panlobular/panacinar emphysema, 86–87, 86b
paracetamol, 60–61, 65
parallel bars, prosthetic-fitted amputee, 466
parasympathetic nervous system, 56
paravertebral block, thoracic surgery, 176
Parkinson disease, 59, 539–559
aetiology and epidemiology, 542
assessment, 544–551
case study, 555b
classification, 542–544
diagnosis, 542
gait, 332–333
interventions, 551–555
neuropathology, 542
symptoms, 540–542
rigidity, 541–542, 581
parkinsonism (Parkinson's plus), 542, 542t
partial agonist, 62
participation
service user, 194–195
in WHO International Classification of Functioning, 583
partner as factor contributing to high quality workforce, 43f
partnership, 193–202
definition, 193–194, 193b
user involvement, 193–202
passive insufficiency of muscles, 214
passive movements/motions
assessment, 212
ankle joint, 248
cervical spine, 227
hip joint, 239
knee joint, 243
lumbar spine, 218–219
neck, 220
shoulder joint, 233
end-feel during, 213
exercises, 280
in acute respiratory distress syndrome, 116
in soft tissue injury rehabilitation, 212–213, 264
see also continuous passive motion
passive muscle imbalance, 306
passive neck flexion, assessment, 220, 566
passive physiological intervertebral movements *see* intervertebral movements
passive physiological intervertebral movements, lumbar spine, 218–219
past medical history *see* medical history
patella
fracture, 515
taping, 327
vastus medialis oblique imbalance and the, 325–327
patellar tap, 242
patellofemoral joint assessment, 242–243, 245–246
femoral shaft fractures, 519
patient
assessment *see* assessment
autonomy, 5
communication *see* communication
complaints by, 15
compliance with exercise prescription, 300–301
education *see* education
feedback, 15
preparation for massage, 478
reflection on interactions with, 77t
reporting of outcome measures, 15
responsibilities to, 6–7
use of term, 1, 183
see also body; client; service user
patient-controlled analgesia, thoracic surgery, 176
payers of services, responsibility to, 7
peak (expiratory) flow in asthma, 97
measurement, 101
pectoral muscles
smaller (pectoralis minor), 321–322
length test, 231
tightness, 232
Pedotti diagrams, 349
peer review, 16
pelvic floor muscles, female
anatomy/physiology, 606–607
exercise/training, 619–620
biofeedback as adjunct to, 620
post-gynaecological surgery, 622
postnatal, 624
in pregnancy and childbirth, 609, 614
dysfunction, 607, 612
exercise, 624
normal changes, 606–607
re-education, 614–615
restricted movement, 614
pelvic girdle (women)
anatomy, 605–606
in pregnancy
pain (PGP), 612–615
restricted movements, 614
pelvic organ prolapse, 619
pelvis
anatomy, women (incl. pregnancy), 605–608
anterior tilted, 313–316, 314t, 315f
fracture, 513
joint *see* joints
motion
prosthetic-wearing amputee, 337–338, 466
in coronal plane (=obliquity), 337
in transverse plane (=rotation), 337–338
pain in area of, 622–623
see also lumbopelvic region
perceptual disturbances, 582
percussion (percussive manipulations - tapotement), 485–486
ventilated patient, 141
percussion note, asthma, 87–88
perineal trauma in labour, 611–612, 618
periosteal proliferation, fracture, 497–498
peripheral nervous system, 561–563
mechanics, 561
in pain, 382, 385
targeting in pain management, 391
pathophysiology, 561–563
see also nerve; neurodynamics
peripheral vascular (arterial) disease
amputation in, 457, 471
exercise-based cardiac rehabilitation and, 163
peroneal neurodynamic test, 568–570

person-centred practice, 5
personal (individual) behaviour *see* behaviour
personal (individual) freedom *see* autonomy
personal (individual) model of disability, 190b, 191–192
personal (individual) qualities and requirements of leaders, 44, 49–50
pethidine (meperidine), 60, 65
  childbirth, 611
petrissage, 481–484
pH, 62
phalangeal fracture
  foot, 517
  hand, 512–513
phantom limb pain and sensations, 459–460
pharmacodynamics, 62–63
pharmacokinetics, 63
pharmacology, 53–65
  basic science, 54–57
  defining, 53
  glossary, 61–65
  see also drugs
photobioactivation effects of laser therapy, 446–447
photobiomodulation effects of laser therapy, 446–447
phototherapy, bronchial/lung cancer, 120
'physical', word removed from definition of disability, 192
physical examination, 224
  ankle/foot, 246–248
  fractures, 508t
  pain, 396–397
    low back, 224
physical exercise *see* exercise; exercise tolerance; fitness
physiological movements, assessment, 212
  intervertebral movements *see* intervertebral movements
  muscle imbalance, 311t
physiology
  pain *see* pain
  women's (incl. pregnancy), 611
physiotherapists, 1–21
  cardiac rehabilitation and, 151–152
  community, cystic fibrosis, 109
  fractures, 506–510
  lower limb amputation and, 460–461
  respiratory, 139–141
  responsibilities (in general), 1–21
  sport and, 374–375
  thoracic surgery and, 177–178
Physiotherapy Framework (CSP), 9
picking up (massage), 483
Piedello's sign, 219
pilates in pregnancy, 617
pink puffers, 91
piriformis test, 236–237
pitch-side considerations, 375
pivot shift test, 245
placebo effect in acupuncture, 406–409
placenta
  expulsion, 611
  growth and function, 608
  healing of site, 612
planning
  amputee treatment, 462
  discharging of patient *see* discharge
  disorder of (apraxia), 582
  fracture management, 507
    case study with femoral shaft fracture, 522
  group exercise, 298
  in muscle imbalance rehabilitation, 317
  in reflective practice, 75–76
  see also SOAPE process
plantar flexion, 219–220, 220f, 248, 248f–249f, 335
plantar response/reflex, 220b
plaster (of Paris) immobilisation, 502–503
  sores, 499
plasticity, tissue, 261
pleura
  acupuncture needle injury to, 406
  anatomy, 169
  aspiration
    and biopsy for tumours, 120
    for culture in pneumonia, 111
  effusions, 113
  operations, 174
  pus collection (empyema), 113–114, 171
  rub, 112
pleurectomy, 174
pleurisy (and associated pain), 112
  bronchial/lung cancer, 120
  pneumonia, 111
plyometric exercises, 295–296
pneumatic post-amputation mobility (PPAM) aids, 465
pneumonectomy, 171, 173
pneumonia (infective), 110–112
  in bronchiectasis, recurrent, 104
  nosocomial, complicating acute respiratory distress syndrome, 116
  ventilator-associated, 138–139
pneumothorax, 114–115, 171
  open versus closed, 115
  spontaneous, 114, 171
    cystic fibrosis, 107, 112
  traumatic, 114–115, 171
  ventilator-induced, 138
poking-chin (forward head) posture, 225, 226f, 319–320
polarisation
  in laser therapy, 445
  nerve, 417
political correctness, 197
political dimensions of interprofessional education, 26t
positional factors affecting symptoms (incl. pain), 209–210
positional weakness (muscle), 309–310
positioning
  joint, sensing *see* proprioception
  patient, 140
    post-thoracic surgery, 179
    respiratory patient, 140
    sputum removal in COPD, 94
  spinal, stretching exercises and, 283
positive airways pressure (positive pressure breathing)
  continuous, post-thoracic surgery, 180
  intermittent *see* intermittent positive pressure breathing
  non-invasive, COPD, 94–95
positive end-expiratory pressure (PEEP), 136–137
  intrinsic (PEEPi), 137–138
positron emission tomography of lung cancer, preoperative, 171
posterior draw test
  knee, 244
  shoulder, 234
postnatal period (puerperium), 612
  advice, 614
postoperative care
  amputation, 463–465, 463t
  breast cancer surgery, 624–625
  gynaecological surgery, 622
  joint replacement
    hip, 534
    metacarpophalangeal, 529
    shoulder, 526t
  thoracic surgery, 177
postpartum period *see* postnatal period
postural drainage
  bronchiectasis, 105
  COPD, 94
  cystic fibrosis, 109
posture (generally but predominantly standing)
  assessment/examination, 212

cervical spine and, 225
hip joint and, 236–237
knee joint and, 241
lumbar spine and, 215–216
muscle imbalance and, 311t
neurological patient, 583
shoulder and, 230–231
in asthma, 96
leg, deformities, 241, 241f
muscles involved in, 307t
muscle fibre types and, 278
older people, 548
Parkinson's disease, 548
case study of management, 556t
instability, 541–542, 548
poking-chin (forward head), 225, 226f, 319–320
postoperative
amputee, 464
breast surgery, 625
in pregnancy
advice, 614
changes, 608
sway *see* sway back; sway backwards and forwards
see also alignment; deformities
potassium-sparing diuretics, 58
potential energy, 360–361
pounding, 486
poverty *see* socioeconomic status
power (mechanical), 360
angular, 361
joint, during walking, 362
linear, 360
power (pulsed shortwave therapy), output, 441
power (service user) and empowerment, 195, 200–201
practice
decision-making, 69–71
evidence-based *see* evidence-based practice
reflection in *see* reflection
scope of, 4–5
social interaction and spheres of influence in, 47–48
standards of *see* standards
student qualification and key elements of preparing for, 4
practice-based learning, 32
practitioner as factor contributing to high quality workforce, 43f
prednisolone, 65
pregabalin, 65
pregnancy, 608–616, 618
anatomy, 605–608
exercise *see* exercise
musculoskeletal changes, 608–609
physiology, 607–608
sex in, 626
see also childbirth; delivery; labour; postnatal period
pre-match considerations, 375
preoperative period (care/procedures/investigations)
amputation, 462–463, 463t
breast cancer, 624
gynaecological surgery, 621–622
hip arthroplasty, 533
thoracic surgery
care, 177
investigations, 171
preseason considerations in sport, 374–375
present condition/complaint, 208–209
amputee, 461t
history of, 210–211
fractures, 508
pressure
on acupuncture meridian points, 489
centre of, 346, 349–350
in-shoe analysis of, 350
massage using, 481–484
pressure biofeedback device/unit, 294, 316
pressure-controlled ventilation, 136
advanced modes, 137
pressure garments and bandages *see* compression garments
pressure-supported ventilation, 137
previous medical history *see* medical history
PRICE (and PRICEM) principles, 256–260
practical application, 260
sports injuries, 376
see also specific components
primary care
cardiac rehabilitation (in long-term follow-up/maintenance), 151
exercise prescription, 165
service delivery, 18
problem(s), listing (in goal-setting with neurological patients), 586
problem-based learning, 32–33
problem-oriented medical record, 507
prodrug, 63
profession(s)
belonging to one, 7–9
characteristics/description, 3–4
education between *see* interprofessional education
history of, 2–3
networks between, 50
responsibility to the, 7
professional(s)
autonomy, 2, 5
characteristics of being one, 5–6
code of values and behaviour, 8–9
identity, 33–34
responsibilities of being one, 3–7
Rules of Conduct (the Rules), 9
professional development
in cardiac rehabilitation, 152b
continuing *see* Continuing Professional Development
professional membership, 8
progesterone in pregnancy, 608
progression
of condition, determining, 210
of exercises, 284–286
in cardiac rehabilitation, 161
proprioceptive exercises, 291
of soft tissue healing after injury, 260
progressive bulbar palsy, 596
progressive multiple sclerosis
primary, 594
secondary, 593
progressive muscular atrophy, 596
proliferation stage (fibrous/phase 3) of repair (1-10 days), 261–268
factors affecting rate, 261–262
pathophysiology, 262
physiotherapy, 262–265
ultrasound therapy and, 438
pronation, foot, 243, 247, 247b
prone knee bend, 221, 570
propofol, 65
proportional assisted ventilation, 137
proprioception, 287–290
assessment, 289–290
ankle, 248
knee, 245
deficiencies (and associated conditions), 287–288, 287t
neurological disorders, 581
definition, 274, 287b
mechanism, 288
muscle balance and, 309
rehabilitation, 287–290
Parkinson's disease, 555t
proprioceptive neuromuscular facilitation, 284
prose, reflection in, 78–79
prostheses (amputees), 465–468
assessment, 461t, 464t
of gait, 461t
gait deviations, 468–469
management before fitting of, 463t, 465

outcome measures, 469–470
physiotherapy management with
after fitting, 465–467
before fitting, 465
transfemoral, 469f
both limbs, 471
transradial, 471f
transtibial, 468f
prostheses (joint - also called implants)
lower limb, 532
hip, 534
knee, 536
upper limb, 525
shoulder, 525, 526t
protection (in PRICE principles), 257
sports injuries, 376
psychological factors, pain, 386
case studies, 395, 397
low back, 224
psychological treatments, pain, 392
psychosexual issues, 626–627
psychosocial disorders (mental health issues)
older people, 546t, 551
postpartum risk, 612
psychosocial impact
amputation, 458–459
in coronary disease of exercise training, 153t
cystic fibrosis, 107–108
exercise training in health and in coronary heart disease, 153–154
lymphoedema, 626
see also biopsychosocial assessment
psychosocial interventions in cardiac rehabilitation, 148–149
pubertal delay in cystic fibrosis, 107
pubic bone
ramus fracture, 513
symphysis *see* symphysis pubis
public health and its promotion, 200
history, 199
puborectalis, 620
pudendal nerve, 607
puerperium *see* postnatal period
pull (muscle), angle of, 366
pull test, Parkinson's disease, 541–542
pulmonary embolism with fractures, 499
pulmonary tissue/organ *see* lung
pulse (arterial)
in asthma, 96
exercise raising, in cardiac rehabilitation, 159
leg, 246
pulse (electrotherapy)
frequency *see* frequency
in pulsed shortwave therapy
duration/width, 441
repetition rate, 441
in TENS, 421, 423–424
pulsed shortwave therapy, 440–444
pulsed ultrasound, 437
low-intensity, and fracture healing, 439–440
pursed-lip breathing, 88, 89f
purulence *see* pus formation
pus, pleural (=empyema), 113–114, 171

Q

Q angle, 241f, 243, 326
qi and acupuncture, 404–405
quadrant tests
hip, 240
knee, 246
shoulder, 233
quadratus lumborum
assessment, 237
problems, 314t
quadriceps, loss of bulk, 242
quadriceps reflex, 220, 221f
qualifications in sports physiotherapy, 369–374
Quality Assurance Agency, 27
quality assurance standards, 9, 14
quality of life as outcome measure in neurological patients, 585t
question(s), initial (in assessment), 208
see also history-taking; interview

R

radial neurodynamic test, 573–574
radiofrequency therapies, 433–434
radiology *see* imaging *and specific modalities*
radionuclide scan of bone, records of previous scans, 211
radiotherapy, 625
breast cancer
adjuvant, 624
care following, 624
bronchial/lung cancer, 105
radius
amputation through, prosthesis, 471f
arthroplasty of head, 530f
fractures, 511–512
distal, 497, 504, 511–512, 512f
see also Colles' fracture; Smith's fracture
head, 511
proximal, 511
ramipril, 65
range of movements
assessment/measurement, 212–213
ankle, 248, 334
hip, 239
knee, 243, 341
exercises increasing, 280–282, 284–286
fractures causing limitation, 497
soft tissue injuries, exercises, 264
reaching task, kinematics, 338–340
reaction *see* action–reaction; ground reaction forces
rearfoot (hindfoot) examination, 247–248
reasoning, clinical *see* clinical reasoning
receptors
agonists, 62
antagonists, 62
as drug targets, 55f
inverse agonist, 62
partial agonist, 62
in proprioception, 287b
reciprocal inhibition (technique), 264, 284
record(s), medical, problem-oriented, 507
rectal disorders, 621
rectus abdominis, females, 606, 606f
in pregnancy, 609
diastasis, 615
re-education, 614–615
rectus femoris
restriction, versus iliopsoas restriction, 236
stretch, 283
red flags, 389
low back pain, 224b
reduction (of fracture), 501–502
open *see* open reduction
re-education *see* retraining/re-education (muscles)
reflection, 6, 11, 16, 50, 67–81
defining, 67–69
as form of assessment, 71–72
in practice, 16, 16b
important principles, 75–78
methods, 78–80
process of, 72–75
rationale, 68–69
requirements, 71
reflex(es)
spinal
cervical, 228
lumbar, 220
proprioception and, 288
stretch, in ballistic stretching, 282
reflex sympathetic dystrophy *see* complex regional pain syndrome

refugees, disabled, 188
registration (with Health Professions Council), 7
regulations *see* legal issues
rehabilitation
  back, programme, 225
  cardiac (in cardiovascular incl. coronary disease), 147–168
    definition, 148–150
    exercise training in *see* exercise
    outcome measures, 165t
    pathway, 166f
    patient groups and special populations, 149–150, 162–164
    phases/components, 150–152, 164–165
    provision and cost-effectiveness in UK, 150
    research evidence, 148–149
    team, 151, 152f
  critical care patient, 141
  exercise in *see* exercise
  functional, 286
  joint replacement
    ankle, 537
    hip, 534–535
    knee, 536–537
  muscle imbalance
    transversus abdominis, 317t
    vastus medialis oblique, 327
  neurological, 586–590
    assessment for *see* assessment
    multiple sclerosis, 593b, 595
    outcome measures *see* outcome measures
    stroke, 592
  outcome measures *see* outcome measures
  pulmonary (in chronic respiratory disease), 93, 102
    asthma, 102
    COPD, 93
    definitions, 93b
  sensorimotor *see* sensorimotor control
relapsing–remitting multiple sclerosis, 593
relationships (between people), 183–205
  changing dynamics, 193–202
  context, 188–193
relaxation
  asthma, 103
    with inhalers, 102
  pain management, 393, 393b
relaxin, 616
remifentanil, 60
remodelling, bone, 498
remodelling stage (phase 4) of repair (10+ days), 261–268
  case study, 396–398
  factors affecting rate, 261–262
  pathophysiology, 265–266
  physiotherapy interventions, 266–268
  ultrasound therapy and, 438–439
renin–angiotensin system, 58
repair *see* healing and repair
repeated movements
  cervical spine, 227
  lumbar spine, 217
repetition number in exercises, 279
  plyometrics, 296
  see also DRIFT
repolarisation, nerve, 417
reproductive (genital) tract
  female, anatomy and physiology, 607
  prolapse, 619
  see also urogenital dysfunction
re-registration, 8
research
  acupuncture, 406–410
  evidence, 10–13
    cardiac rehabilitation, 148–149
resistance to movement
  exercises (resistance training), 278–280
    in cardiac rehabilitation, 161–162
    special patient groups, 279–280
  testing, 212
    shoulder joint, 233
resisted contractions, symptoms arising from, 213
respiratory disease/disorders, 83–127
  chronic, pulmonary rehabilitation *see* rehabilitation
  exercise-based cardiac rehabilitation, 164
  lung surgery-related, 173t
  older people, 546t
  see also lung
respiratory distress syndrome, acute *see* acute respiratory distress syndrome
respiratory failure (ventilatory failure), 121–122, 133–135
  type I, 121–122, 133
  type II, 121–122, 133–134
respiratory failure
  mixed, 134
  pathways to/causes of, 134–135
respiratory infections
  in bronchiectasis, control, 105
  in COPD, control, 91
  in cystic fibrosis, 106, 108
    management, 108
  see also pneumonia; tuberculosis
respiratory mechanics, 130–131
respiratory muscles (ventilatory muscles), 129–131
  accessory, 129
  failure/dysfunction/weakness, causes, 134
    COPD, 132
  fatigue, 132–133, 135
  training, 93–94
respiratory physiotherapist roles, 139–141
respiratory syncytial virus, 110
responsibilities, 1–21
rest
  in PRICE principles, 257–258
    sports injuries, 376
  tremor at, Parkinson's disease, 540
restricted movements of spine and pelvic girdle in pregnancy, 614
restrictive pulmonary disease, 83, 110–117
  types, 83
    see also specific types
resuscitation, sports, 369–370
retraction, cervical spine, 318t
retraining/re-education (muscles)
  in muscle imbalance, 312t
  pregnant women, 614–615
retropatellar adhesions with femoral shaft fractures, 522
retropulsion test, Parkinson's disease, 541–542
revascularisation
  coronary, 158
    cardiac rehabilitation after, 149–150
  fracture site, 499
rheumatoid arthritis
  exercise-based cardiac rehabilitation, 164
  hand arthroplasty, 525
  management
    acupuncture, 408
    laser therapy, 448
rhomboids, 321–322
rhythm, massage practitioner's, practice, 476
rib pain (flare), antenatal, 615
rigidity in Parkinson's disease, 541–542, 581
riluzole, motor neurone disease, 597b
robotics, 589
Rolfe model of reflection, 80
rolling, 484
  fascial lifting and, 487

rotary component of muscle force, 355
rotation
  cervical/neck, 227, 318t
  hip joint, 239
  pelvic, 337–338
    prosthetic-wearing amputee, 466
  shoulder joint/complex, 219, 232b, 321t
  see also FABER test
rotator cuff
  actions/forces, 322
  deficiencies/weakness, 232, 322–323
Rotocaps, 99
Royal Charter, 2–4
Rules of Professional Conduct (the Rules), 9

S

sacroiliac joint assessment, 219
sacroiliac ligaments, assessment, 212, 219
sacroiliac/trochanteric belt (n pregnancy), 614
safe environment for reflection with supervisor or mentor, 75
safety issues, acupuncture, 406
sag sign, 245b
sagittal plane, hip joint movement in, 336–337
SAID (Specific Adaptation to Imposed Demand) principle, 305, 314
salbutamol, 65
  asthma, 98
  COPD, 92
salmeterol, 92
Sandwell Integrated Language and Communication Service (SILCS), 195
saturation kinetics, 63
scaphoid fracture, 512
scapula
  fracture, 510
  movement faults and their management, 306–309, 324t
    winging, 231, 324t
scapulohumeral joint, 323t
scapulohumeral rhythm, 231–232, 232f, 320–321
scapulothoracic joint, muscle acting on (and muscle imbalance), 321–322
scar
  breast surgery-related, 624–625
  contractures and, 267
  formation, 262
scarf test, 232
scintigraphy (radionuclide scans), bone, records of previous scans, 211
scope of practice, 4–5
Scottish Intercollegiate Guidelines Network, 14
second impact syndrome, 373–374
secretions, respiratory/bronchial (incl. sputum)
  as clinical feature
    in bronchiectasis, 104
    in COPD, 88
    in cystic fibrosis, 106
  management (incl. control and clearance of lung fields)
    in asthma, 102–103
    in bronchiectasis, 105
    in COPD, 91, 94
    in cystic fibrosis, 109
    in pneumonia, 112
    postoperative (after thoracic surgery), 178–179
    in respiratory failure, 122
    ventilated patients, 140
  see also mucus; oropharyngeal secretions
see-saw, 350–351
segment (body) angle/inclination
  calculation, 333–334
  forces and moments acting on, 354
  joint moment and, 364
segmental resection of lung, 173
self-efficacy, 389, 392b
self-report pain measures, 387–388
senna, 65
sensing of joint position and movement *see* proprioception
sensitisation of nociceptive/pain system, 385–387
  case studies, 395–397
  management strategies targeting, 390–394
sensitizing manoeuvres (in neurodynamic tests), 564–565
  slump test, 567
  straight leg raising test, 568
sensorimotor control, rehabilitation, 287–290
  limbs, 291–293
  spine, 293–294
  see also motor control
sensory cortex, primary, persistent pain and, 394
sensory disturbances, 581
  brain injury, 599
  massage and, 480
  multiple sclerosis, 594
  phantom limb, 459–460
series elastic component and plyometrics, 295
serotonin (5-HT), 61
serratus anterior, 321–322
service(s)
  clinically and cost-effective, delivery, 18
  evaluation, 14–16
  leadership and
    delivery of services, 45
    improvement of services, 45–47
  in primary and community care, delivery, 18
  quality assurance standards for delivery of, 8
  responsibility to those paying for, 7
  world-class, commissioning of, 45–47
service user, 193–198
  involvement, 193–198
    definition, 197b
  use of term, 1, 183
  see also client; patient
setting direction, 45
setting goals *see* goals
severity
  of injury
    and progression through healing continuum, 260
    and rate of healing in stages of proliferation and remodelling, 261–262
  of symptoms, assessment, 208–209
sexual dysfunction
  brain injury, 599
  psychological issues, 626–627
shaking (massage), 484
shared learning, 24, 26t
shifted posture (and lumbar spine), 216
shock, fractures, 497, 499
shockwave therapy, 448–450
shortwave therapy
  pulsed, 440–444
  shortwave diathermy, 433–434
shoulder (shoulder joint and complex), 227, 230–235, 320–325
  arthroplasty, 525–526
  assessment, 227, 230–235
  clinical presentations, 323–325
  dislocation, 268b
  exercises, 285, 286f
    arthroplasty for osteoarthritis, postoperative, 526t
    thoracic surgery, postoperative, 179
  fractures in area of, 510

impingement *see* impingement
linear displacement of hand during reaching with and without dysfunction of, 339
muscle imbalance and the, 320–325
pain, 69–71, 323
with bronchial/lung tumours, 120
shoulder girdle movements, 231
shuffling gait, 332–333
shuttle walking test, 158
side-flexion
cervical, 227
lumbar, 217–218
SIGAM (Special Interest Group in Amputee Medicine) algorithm, 469
SIGN (Scottish Intercollegiate Guidelines Network), 14
signs (clinical), definition, 208b
simian posture, Parkinson's disease, 542
simulated learning, 32
simvastatin, 65
SIN factor (severity/irritability/nature) determination, 210
single compartment model, 63
sinusitis in bronchiectasis, chronic, 104
sit-to-stand test, 297
sitting flexion, assessment, 219
skeletal muscle *see* muscle, skeletal
skeletal traction, 506b
skills (specific to profession/not shared by others), 16–17
possessing, 4–5
sports physiotherapy, 369–374
skin
care in lymphoedema, 626
infections
lymphoedema and predisposition to, 626
massage and, 479
plaster sores, 499
sensory disturbances *see* sensory disturbances
traction, 500
sleep and breathing, 122
slider treatment (nerves), 575–576
slow-twitch (type I) muscle fibres, 130, 278, 279t, 317
slump test, 222, 567
small cell lung cancer, 120t
SMART goals, 215
functional tests, 297b
neurological patient, 586
orthopaedic patient, 509
Smith's fracture, 511
smoking, 189
behavioural interventions, 189, 200–201
COPD, 84
chronic bronchitis, 85
coronary heart disease, 147
emphysema, 87
exposure to risk, 86b
fracture union and, 499
lung tumours, 119
SOAPE (subjective, objective, analysis, plan and evaluation) process in muscle imbalance, 305, 314
cervical spine and, 320, 320t
fractures, 507
shoulder complex and, 323
vastus medialis oblique imbalance, 308–309
see also individual components
social care and health care, collaborative, 23–39
social change approach in health promotion, 201
social difficulties *see* psychosocial impact
social history
amputee, 461t
fractures, 508
social interaction, 47–48
social learning theory, 35
social models
of communication, 183–184
of disability, 190b, 192–193, 195
socioeconomic (social and/or economic) status
definition, 188b
health inequalities and, 188–190
pain and, 386–387
case studies, 395, 397
low back, 224
see also entries under psychosocial
sodium in sweat, cystic fibrosis, 107
soft tissue
cervical spine area, palpation, 229
injury, 255
ankle *see* ankle
healing and repair *see* healing and repair
laser therapy, 448
severity *see* severity
plasticity, 261
soreness of muscle, delayed-onset, 273, 276–277
sounds of breathing *see* auscultation
spasm, muscle, 273
with fractures, 497
spasticity, muscle, 276b, 581, 594
in multiple sclerosis, drug therapy, 595
spatial parameters
gait, 332
measurement, 343
Special Interest Group in Amputee Medicine (SIGAM) algorithm, 469
special needs groups, cardiac rehabilitation, 149
Specific Adaptation to Imposed Demand (SAID) principle, 305, 314
speech problems, neurological conditions causing, 582
speed
of movement (in exercise), change in, 286
of muscle contractions, effect, 366–367
spheres of influence, 47–48
sphincters, anal, and faecal incontinence, 620
spinal boards, 375–376
spinal cord
pain mechanisms, 382, 385
targeting in pain management, 391–392
spinal muscle in pregnancy, re-education, 614–615
spine
assessment, 215–229
cervical *see* cervical spine
lumbar *see* lumbar spine
muscle imbalance and the, 317–320
muscle and joint forces on base of, calculation, 358–359
in pregnancy, 608–609, 612–613
pain, 612–613
restricted movements, 614
reflexes *see* reflexes
sensorimotor rehabilitation, 293–294
stability, 306–308
in stretching exercises, position, 283
spinothalamic tract and pain transmission, 382
spiral fractures, 495
spirometry
COPD classification, 85t
incentive, 179
splints, metacarpophalangeal joint, 512–513, 529
sports (incl. leisure sports/activities and athletes), 369–379
cardiovascular and musculoskeletal fitness, 258b
energy costs, 156t

functional testing of shoulder joint, 233
in pregnancy, 617
qualifications and skills, 369–374
role of physiotherapist, 374–375
sputum *see* secretions
squamous cell lung carcinoma, 120t
squats, 285, 327
end-range, 327
squeeze test (calf), 250
stabiliser muscles, 306
cervical, 319t
core, 317
glenohumeral joint, 322t
lumbopelvic, 312t
scapulothoracic joint, 321–322
Stabiliser pressure biofeedback device, 294
stabilising component of muscle force, 355
stability
assessment, 306–309
lumbopelvic, 222, 314–315, 606
shoulder, 234
core *see* core stability
exercises involving re-education of spinal and pelvic stability in pregnancy, 614–615
prosthetic-wearing amputee, 466
see also instability
stance, massage practitioner's, 475
stance phase in gait, 499–500, 506
standard(s), 7, 13
quality assurance, 9, 14
see also Core Standards
Standards of Proficiency for Physiotherapists, 7
standing
muscle imbalance, observation, 315
neurological patient observed in and moving to or from, 583
posture in *see* posture
shoulder flexion exercise, 285
on toes (myotome S1 test), 220f
standing flexion sign, 219
Staphylococcus pyogenes, 110
static equilibrium, 345
static splints, metacarpophalangeal joint replacement, 529
static stretching, 282–283, 284b
statins, 58
step
length, 332
time, 332
step test, Chester, 158
stereotypes, 197
sternal foramen, acupuncture needling through, 406
sternoclavicular joint assessment, 233
sternotomy, median, 172
steroids (corticosteroids; glucocorticoids), 58–59
adverse effects, 92
asthma, 58–59, 98
bronchiectasis, 105
COPD, 92
multiple sclerosis, 595
stigma, 197
stools *see* defaecation; faeces
stork test, 219
straight leg raising test (SLR; Lasègue's test), 220–221, 568
historical aspects, 561
indications, 568
modifications, 568–570
structural differentiation, 568
technique, 568
upper limb equivalent (of cervical spine), 229
strapping, 209–210
buddy, finger fractures, 502
streamlining (hydrotherapy), 300
strength (muscle)
assessment/testing, 213, 274–275, 362–368
ankle/foot, 248
fractures, 509, 520, 520t
hip, 239
muscle imbalance and, 311t
upper limb, 362–368
exercises/training/rehabilitation, 274–280, 362–368
amputee, 464
benefits, 275–276
in muscle imbalance, 312t
upper limb, 362–368
in water, 299–300
as outcome measure in neurological patients, 585t
'strength' window for electrotherapy, 419
Streptococcus pneumoniae pneumonia, 110
streptokinase, 65
stress (fatigue) fracture, 496
foot, 517
tibia, 516
stress tests, musculoskeletal
ankle ligaments, 248–249
knee, 243–244
stress urinary incontinence, 619
stretch (and stretching), 281–284
in cardiac rehabilitation, 159
contraindications, 278
definition, 274
prolonged, causing weakness, 309
in soft tissue injury rehabilitation, 264
teaching, 277–278
stretch reflex in ballistic stretching, 282
stride
length, 332
Parkinson's, 549t
time, 332
stroke (cerebrovascular accident), 590–600
clinical features, 591–592
haemorrhagic, 591
ischaemic, 591
management/interventions, 592
UK guidelines, 580
upper limb movements *see* upper limb
virtual reality, 588
pathology, 591
risk factors, 598t
stroking (massage practitioner's), 480–481
practising, 477
structural barriers to disability, 192
structural differentiation manoeuvre, 563
median neurodynamic test 1 (MNT1), 571–572
median neurodynamic test 2 (MNT2), 572
neurogenic response in
abnormal, 565–566
normal, 565
passive neck flexion, 566
peroneal neurodynamic test, 570
prone knee bend, 570
radial neurodynamic test, 574
slump test, 567f
straight leg raising test, 568
sural neurodynamic test, 570
tibial neurodynamic test, 570
ulnar neurodynamic test, 573
structural instability, 288
students, 23–26
interprofessional learning led by, 33
key elements of preparing for practice on qualification, 4
subacromial structures, testing, 232
subarachnoid haemorrhage, 591–592
subcutaneous emphysema, 115
subjective assessment, 208–211
aims, 208
amputee, 461t
fractures, 501, 508
muscle imbalance, 311t, 314–315
cervical spine region, 320t
shoulder complex, 323
vastus medialis oblique, 308–309
neurological patient, 583
Parkinson's disease, 546

older people, 546
see also SOAPE process
subtalar joint assessment, 248–249
subthermal modalities of electrotherapy, 419–420
suction, 140
ventilated patients, 140
COPD, 95
Sudeck's atrophy *see* complex regional pain syndrome
sulcus test, shoulder, 234
supervisor, reflection with, 73–75
supination, foot, 247, 247b
supine lying, hip examination, 236
supracondylar fracture of humerus, 510
supraspinal structures *see* brain
supraspinatus (lesion) test, 235
sural neurodynamic test, 568–570
surgery
breast cancer, 623
bronchial/lung cancer, 120, 169–171
bronchiectasis, 105, 171
cystic fibrosis, 110
gynaecological, 621–622
pyschosexual problems, 626
heart valves, 150
pneumothorax, 171
recurrent, 115
thoracic *see* thoracic surgery
tuberculosis, 119
see also postoperative care; preoperative period *and specific techniques/procedures*
swallowing difficulty (dysphagia), 582
multiple sclerosis, 594
Swanson finger arthroplasty, 528, 531f
sway back (abnormal), 216
sway backwards and forwards (normal), 346
sweat test, cystic fibrosis, 107
swelling
fluid accumulation causing *see* oedema
knee, 241–242
limb elevation with, 259–260
swing phase in gait, 334–336, 341–342, 359–360
time, 332
Swiss ball exercises proprioception, 293
symmetry in gait, 332
sympathetic nervous system, 56
pain and, 223
symphysis pubis in pregnancy, 608–609
diastasis, 613
dysfunction (SPD), 613–614
symptoms
aggravating and easing factors, assessment, 209–210
area of, assessment, 208–209
definition, 208b
duration, assessment, 209
exercise training in chronic heart disease and its beneficial effects on, 153t
hip and lumbar spine as source of, differentiation, 218–219, 237–238
in resisted contractions, 213
severity of, assessment, 208–209
time factors, 210
see also specific disorders
systematic reviews, 11

T

tactile acuity training, 394, 394b
talofibular ligament injury, anterior, 246, 257b
tamoxifen, 65, 624
taping, patella, 327
tapotement *see* percussion
task-specific practice with neurological patients, 586–587
teaching (patient) *see* education
team, multidisciplinary
amputation and, 458b
cardiac rehabilitation, 151, 152f
fractures and, 509–510
reflection on interactions with, 77t
team doctor (sports), 377–378
technology and interprofessional education, 31
temperature, ankle/foot, 246
see also cryotherapy; ice burns; ice massage *and entries under* thermal
temporal parameters *see* timing
ten repetition maximum (10RM), 279
tenderness
ankle/foot, 246–247
knee, 242
tendon(s)
differentiation tests, 214–215
injury, eccentric exercise, 277, 301
tendon reflex, Achilles, 220, 221f
tendonitis, Achilles, frictions in, 491
tennis elbow, 491, 577
TENS *see* transcutaneous electrical nerve stimulation
tension, mechanical *see* mechanical tension tests; muscle
tension headache, acupuncture, 408
tensioner treatment (nerves), 576
terbutaline, 65
asthma, 98
terminal stages (and care incl. palliation)
COPD, 95
cystic fibrosis, 108, 110
motor neurone disease, 597
tetanus risk, fractures, 499
texture assessment in massage, 477
therapeutic use exemptions (TUEs), 370
therapeutic window
drugs, 424
electrotherapy, 223, 418–419
therapy *see* treatment
thermal effects of non-thermal electrotherapy
pulsed shortwave therapy, 442
ultrasound, 437
thermal modalities of electrotherapy, 419, 420f, 432–434
general principles, 432
therapeutic effects, 432–433
see also subthermal modalities
thermal noxious stimuli and nociception, 381
thiazide diuretics, 58
Thomas test, 236
Thompson's hemiarthroplasty, 514f
Thompson's squeeze test, 250
thoracic spine, flexion in slump test, 567
thoracic surgery, 169–181
incisions/approaches, 171–172
indications, 169–171
modalities of physiotherapy, 178–180
pain control, 175–176
physiotherapist and, 177–178
postoperative care, 177
preoperative period *see* preoperative period
thoraco-laparotomy, left, 172
thoracoscopic incisions, video-assisted, 172
thoracotomy
anterolateral, 172
posterolateral, 171–172
thorax (chest)
anatomy, 169
deformed shape
COPD, 88
cystic fibrosis, 107
drains, 174–175
expansion exercises (lung re-expansion)
COPD, 94
pneumonia, 112
pneumothorax, 115

mobility in bronchiectasis, decreasing, 104
X-ray *see* X-ray
thoughts and their potential impact on pain, 388–389
clinicians, 389b
360° feedback, 50
thrombolysis, stroke, 592
thrombosis, venous *see* venous thrombosis
thumb, carpometacarpal joint fracture, 513
tibial fracture, 502f, 504f, 515–516
condylar, 515
fixation, 504
healing
low-intensity pulsed ultrasound, 440
shaft, 502f
tibial level amputation
gait deviations, 468
knee contractures, 464
prosthesis, 468
tibial neurodynamic test, 568–570
tibial prosthetic component in knee arthroplasty, 536
tibiofemoral joint
inferior, accessory movements, 249
superior, accessory movements, 246
tenderness, 242
tibiofibular joint, superior, accessory movements, 246
timing (temporal parameters incl. duration and frequency)
of assessment, 207–208
electrotherapy, 419
in interferential therapy, 429
in pulsed shortwave therapy, pulse duration, 441
in exercise training *see* FITT principle
fracture healing and union, 498–499
gait, 332–333
methods of measuring temporal parameters, 343
of pain, 387b
in reflective practice, 76
stroke management, 592
of symptoms, 209–210
tiredness *see* fatigue
tissue
healing and repair *see* healing and repair
heating in non-thermal electrotherapy *see* thermal effects of non-thermal electrotherapy
in laser therapy
interactions with laser, 446–447
light absorption, 446
pain and threat to, 381
plasticity, 261
shockwave therapy effects, 448
in ultrasound therapy
US absorption and attenuation by, 436–437
US transmission through tissues, 436
see also soft tissue
toe(s)
clubbing *see* clubbing
examination, 248
movements, 248
standing on (myotome S1 test), 220f
toe off, 332, 336, 341, 348
tone *see* muscle
torque *see* moment
torticollis, 225
touching *see* palpation; tactile acuity training
traction, 506
femoral shaft fracture, 515
skeletal, 495
skin, 500
traditional Chinese medicine *see* Chinese medicine
training, formal, reflection on attending, 77t
tramadol, 60, 65
trampette, 295f
transcutaneous electrical nerve stimulation (TENS), 391, 421–424
acupuncture (low TENS), 423
machine parameters, 421
mechanism of action, 422–423
in pregnancy, 610–611
labour, 615
thoracic surgery, 176
transfemoral amputation *see* femur
transfer movements
older people, 548
Parkinson's disease, 548, 556t
transient ischaemic attack, 593
transplants, heart, cardiac rehabilitation, 149
transporters as drug targets, 55f
transradial amputation, prostheses, 471f
transtibial amputation *see* tibial level amputation
transudative effusions, 113
transverse fractures, 495
transverse plane, pelvic motion in, 337–338
transversus abdominis (TrA), 312–317
in pelvic floor muscle training, 619–620
in pregnancy, 609
trapezium replacement, 531f
trapezius, 321–322
trauma (traumatic injury)
acupuncture-induced, 406
ankle (incl. soft tissue), 246, 257b, 261b, 264b, 270b
brain *see* brain
fracture caused by, 496
fracture causing
to blood vessels and nerves, 501
to viscera, 500
hamstring *see* hamstrings
head *see* head
limb amputation following, 457, 470
massage and, 480
onset of condition related to, 210
perineal, in labour, 611–612, 618
pneumothorax, 114–115, 171
proprioception in prevention of, 287b
proprioceptive deficits, 287t
severity *see* severity
soft tissue *see* soft tissue
tendon, eccentric exercise, 277, 301
see also burns; fractures
treadmill training, 587–588
treatment
previous, asking questions about, 211
timing of assessment in relation to, 207–208
tremor, 581
Parkinson's disease, 540, 542
dominant or non-dominant, 543
Trendelenburg test, 238
triceps reflex testing, 228, 229f
tricyclic antidepressants, 61
trigger points *see* myofascia
trimalleolar fracture, 516
Trinity Amputee Prosthetic Evaluation Scale, 470
trunk, amputee
assessment, 461t
lateral bending with prosthesis, 468–469
trust and trustworthiness, 6–7
in reflecting with supervisor or mentor, 74–75
truth in reflection with supervisor or mentor, 75
tuberculosis, 118–119
tumour(s)/neoplastic disease
amputation due to, 470
lung *see* lung

malignant *see* cancer
massage contraindications, 480
oesophageal, 171–172, 174
Turbohaler, 99
turbulence in water, 300
twisting forces, fracture due to, 496
two compartment model, 63
two-pole interferential therapy, 426–427

U

ulnar neurodynamic test, 573
ultrasound therapy, 435–439
clinical uses, 437
fractures, 499
dose, 439
tissue repair and *see* healing and repair
union of fractures
delayed/faulty/failure, 500
factors affecting, 499
upper limb (arm)
amputation, 470–471
causes, 457, 458t, 470
levels, 458t
see also amputation
arthroplasty, 525
fractures, 510–513
function as outcome measure in neurological patients, 585t
joint forces, calculation, 356–357
lower limb (and leg), proprioceptive exercises, 292–293
moments, 354
movements, following stroke
constraint-induced movement therapy, 588
mental practice following stroke, 70b
robotics, 589
muscle
forces, calculation, 356–357
strength testing and training, 362–368
neurodynamic tests, 570–574
pain with bronchial/lung tumours, 120
plyometrics, 295–296
reaching motions, 339
tension test, 229
urge incontinence, 619
urinary incontinence, 580–582
multiple sclerosis, 594
psychosexual issues, 627
traumatic brain injury, 599
women, 618–619
obstetrically-related, 607, 612, 618
urinary tract symptoms (lower) in men, 620
urine, drug excretion in, 57
urogenital dysfunction, 618–620
users of services *see* service user
uterus, 607
contractions *see* contractions

V

vaccination, tuberculosis, 119
vacuum extraction, 611
vaginal delivery *see* delivery
valgus deformities
foot, 247–248
knee (genu valgum), 241, 515
valgus stress test, knee, 243–244
values, professional, code of, 8–9
valves (heart) surgery, 150
varus deformities
foot, 247–248
knee (genu varum), 241
varus stress test, knee, 244
vasculature (blood vessels)
acupuncture needles injury to, 406
amputation due to disease of, 457, 471
with fractures
injury to, 501, 514, 516
supply affecting time to union, 499
in inflammation and injury and its repair, 256, 261
see also cardiovascular system; circulation; peripheral vascular disease; revascularisation
vastus lateralis, 325
vastus medialis oblique and its imbalance, 306–309
vaulting, transfemoral amputee, 469
velocity (in gait), 332
angular *see* angular velocity
linear *see* linear velocity
venous thrombosis risk
with fractures, 510–517
deep, 499
ventilation, 129–145
mechanical/supported/assisted, 135–139
complications, 137–139
COPD, 92, 94–95
goals, 135
historical background, 135
modes, 136–137
motor neurone disease, 597
physiotherapist's role, 139–140
thoracic surgery patient, 180
weaning, 141–142
spontaneous, 129–133
opposing forces, 131–132
see also breathing
ventilatory failure *see* respiratory failure
ventilatory muscles *see* respiratory muscles
verbal reflection, 78
vertebral artery testing, 227–228
vertebrobasilar insufficiency and vestibular symptoms, differentiation test, 227–228, 227b
vertebrobasilar testing, 228
vertical forces, 345
in gait cycle, 346
lower limb (knee), 355
upper limb, 357
vestibular and vertebrobasilar insufficiency symptoms, differentiation test, 227–228
vibrations
in massage, 486
in ultrasound therapy, 435–436
victim-blaming, 199, 199b
video-assisted thoracoscopic incisions, 172
video recordings
amputee rehabilitation, 470
muscle imbalance, 325
video vector generators, 350
virtual reality, 588
visceral injury, fracture causing, 500
visual analogue scale (VAS) of pain, 209, 387–388
case studies, 396–397
visual disturbances, neurological conditions causing, 581
multiple sclerosis, 594
visual input
as cues in Parkinson's patients, 555t
in proprioceptive exercises, 291
volume-controlled ventilation, 136
volume of distribution, 63
volume reduction surgery, lung, 173
voluntary movements in Parkinson's disease, dysfunction, 541
volutrauma, ventilator-induced, 138

W

walk mat systems, 343
walking
aids, 257
amputee, 465
amputee, 466
aids, 465
bilateral prostheses, 471
incremental programme, 164, 165t

lower limb joints in, 359–360
knee kinematics, 340–342
moments, 359–360
power, 362
moments around ankle/knee/hip joints in, 359–360
older people, 549
Parkinson's disease, 549
recovery in treadmill training, 587–588
see also gait
walking tests, shuttle, 158
war, amputations, 458
warfarin, 65
warm-up, exercise-based cardiac rehabilitation, 159
water, as massage medium, 479
water-based/aquatic exercises (hydrotherapy), 299
pregnancy, 617
wax therapy, 433
weakness (muscle), 581
CNS damage causing, 581
positional, 309–310
rotator cuff, 232, 322–323
stretch, 309
weaning from ventilation, 141–142
wedge resection of lung, 173
weight, 345
weight-bearing
amputee, 465–466
hip replacement, 534
weight-lifting and muscle insertion points, 366
weight-transference, amputee, 466
Western medicine, acupuncture in, 405–406, 409–410
wheeze
asthma, 96
COPD, 88
cystic fibrosis, 93, 107
WHO *see* World Health Organization
winging of scapula, 231, 324t
wobble board, 248, 290–291
Wolff's law, 497
women, 605–635
cardiac rehabilitation, 149
pelvic anatomy (incl. pregnancy), 605–608
physiology, 611
pregnancy *see* pregnancy
work (mechanical)
angular, 361
of breathing, 130–131, 134–135
linear, 360
work (occupation) *see* employment
workforce, high-quality, 42–43
World Anti-Doping Regulations Association (WADA), 370, 372
World Confederation (Congress) for Physical Therapy (WCPT), 4
International Organization of Physical Therapists in Women's Health subgroup, 1–2
interprofessional education, 24–25
World Health Organization (WHO)
acupuncture and, 407–408
on cardiovascular disease, 147
health defined by, 188
International Classification of Functioning, Disability and Health *see* International Classification of Functioning, Disability and Health
International Classification of Impairment, Disability and Handicap (1980), 192
wound
lung surgery, complications, 173t
open *see* open wound
wringing (massage), 483–484
wrist
arthroplasty, 530f
fractures in area of, 512
writing
of assessment, 215
methods of reflection, 78–79
wry neck, 225

X

X-ray radiograph
chest
asthma, 87–88
bronchial/lung cancer, 120
bronchiectasis, 104
COPD, 90
cystic fibrosis, 107
pleurisy, 112
pneumonia, 111
preoperative, 171
tuberculosis, 119
previous, records of, 211
xanthene derivatives, COPD, 92

Y

yellow flags, 389
low back pain, 224b
yin and yang, 405
yoga in pregnancy, 617