Muscles, Nerves and Movement

Muscles, Nerves and Movement
in human occupation

Third edition

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and

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Preface to the third edition

This book is written primarily for students entering the paramedical professions, particularly those with a limited scientific background. Clinicians will use the book as a reference source for acquiring a further understanding of the basic movement problems encountered by their clients. This third edition of *Muscles, Nerves and Movement* extends the study of movement in daily activities, established in earlier editions to include the wider domain of human occupation.

Section I describes the structure and function of the basic components of the musculoskeletal system. An introduction to the location, organisation and functions of central and peripheral nervous systems is given, which is developed further in Section III. The terms to describe the movements at joints and the types of muscle work are defined and the mechanical principles related to the stability of the body are outlined.

Section II is a functional approach to the anatomy of movement. The structure and movements of the major joints of the body are described. The position, attachments and nerve supply of the muscles are given, together with their use in named activities of daily living.

Section III augments the knowledge of the nervous system from Section I to formulate an account of the sensory and motor systems in movement control.

Section IV has two new chapters. One chapter explores the multiple factors in the performance of functional movements, and develops a system for the analysis of core body positions and the transitions between them. A framework for the understanding of human occupation forms the basis of the final chapter and several case histories are described to demonstrate the application of the framework to specific clinical problems.

Each chapter begins with a contents list and ends with a summary. The exercises which direct the reader to observe colleagues in the classroom and people in their daily lives have been retained from earlier editions. Some of the clinical note-pads have been revised and expanded.

The first edition was written during many years of experience of teaching and examining at the Liverpool (Barbara Tyldesley) and London (June Grieve) Schools of Occupational Therapy. For this third edition, two lecturers in occupational therapy have been recruited to the team of authors. Linda Gnanasekaran of Brunel University has written and edited Chapter 13 on the Performance of Functional Movements. Ian McMillan of Queen Margaret University College, Edinburgh, devised and developed the framework for the Understanding of Human Occupation in Chapter 14. He also revised the section on the interpretation of pain in Chapter 11. Both Linda and Ian have given valuable advice on the updating of Sections I to III.

Basic anatomy, physiology and neurology are now frequently integrated into modules of study of the theory and practice of occupational therapy. We hope that students will use this book as a reference source at appropriate levels in the course. If the knowledge gained from this book leads a student to ask questions and to seek answers in other reading, then our objectives have been achieved.

Acknowledgements

We very much appreciate the time and energy given by Linda Gnanasekaran and Ian McMillan to this new edition. Their advice and support have made our task easier and we have learnt from them. Jo Creighton has combined her drawing skills with her understanding of movement to produce the figures for Chapter 13, for which we thank her. We are also grateful to clinical occupational therapists Ronnie Bentley, Linda Gwilliam and Louise Hogan who have contributed to the case history exercises. Caroline Connelly has been an enthusiastic and encouraging editor at Blackwell Science.

June Grieve
Barbara Tyldesley

Section I

Introduction to movement

Components of the musculoskeletal and nervous system, movement terminology

- Basic units, structure and function: supporting tissues, muscle and nerves
- Movement terminology
- The central nervous system: the brain and spinal cord
- The peripheral nervous system: cranial and spinal nerves

1

Basic Units, Structure and Function: Supporting Tissues, Muscle and Nerve

The foundation for the study of movement lies in the musculoskeletal and nervous systems. While muscle action is the basis of movement of the skeleton at joints, the nervous system is an essential component of all movement. Nervous tissue sends commands to the muscles for action, and responds to changes in the external and internal stimuli that affect movement during performance. The nervous system regulates the activity in the cardiovascular and respiratory systems to meet the demands of the muscles for the release of energy. The combined activity of muscle and nerve maintains muscle tone. This is the background muscle activity which holds the static positions of the body in preparation for movement.

This chapter covers the basic components of structure that are organised to produce a movable joint, a contractile muscle and nerves that are excitable, with the connective tissues supporting all of these. The three basic functional units are:

- **connective tissues** which provide the framework for stability and support while body parts move in particular directions;
- **skeletal muscle** which changes in length and pulls on bones to produce movements at joints, powered by the energy released in the the muscles;
- **neurones and nerves** forming the nervous system which conducts information between the environmental sensors, the control centres for movement and the muscles. In this way appropriate movement is initiated, executed and regulated.

FRAMEWORK AND SUPPORT: THE CONNECTIVE TISSUES

The overall function of connective tissue is to unite or connect structures in the body, and to give support. Bone is a connective tissue which provides the rigid framework for support. Where bones articulate with each other dense fibrous connective tissue, rich in collagen fibres, surrounds the ends of the bones, allowing movement to occur while maintaining stability. Cartilage, another connective tissue, is also found associated with joints, where it forms a compressible link between two bones, or provides a low-friction surface for smooth movement of one bone on another. Connective tissue attaches muscles to bone, in the form of either a cord (tendon) or a flat sheet (fascia). The connective tissues may be divided into:

- **dense fibrous tissue**
- **cartilage**
- **bone.**

Dense fibrous tissue

Dense fibrous connective tissue unites structures in the body while still allowing movement to occur. It has high tensile strength to resist stretching forces. This connective tissue has few cells and is largely made up of fibres of collagen and elastin that give the tissue great strength. The fibres are produced by fibroblast cells that lie in between the fibres (Fig. 1.1). The toughness of this tissue can be felt when cutting through stewing steak with a blunt knife. The muscle fibres are easily sliced, but the covering of white connective tissue is very tough. Examples of this tissue are as follows:

- The **capsule** surrounding the movable (synovial) joints which binds the bones together (see Fig. 1.7).
- **Ligaments** form strong bands that join bone to bone. Ligaments strengthen the joint capsules in particular directions and limit movement.
- **Tendons** unite the contractile fibres of muscle to bone.

In tendons and ligaments, the collagenous fibres lie in parallel in the direction of greatest stress.

- An **aponeurosis** is a strong flat membrane, with collagen fibres that lie in different directions to form sheets of connective tissue. An aponeuro-

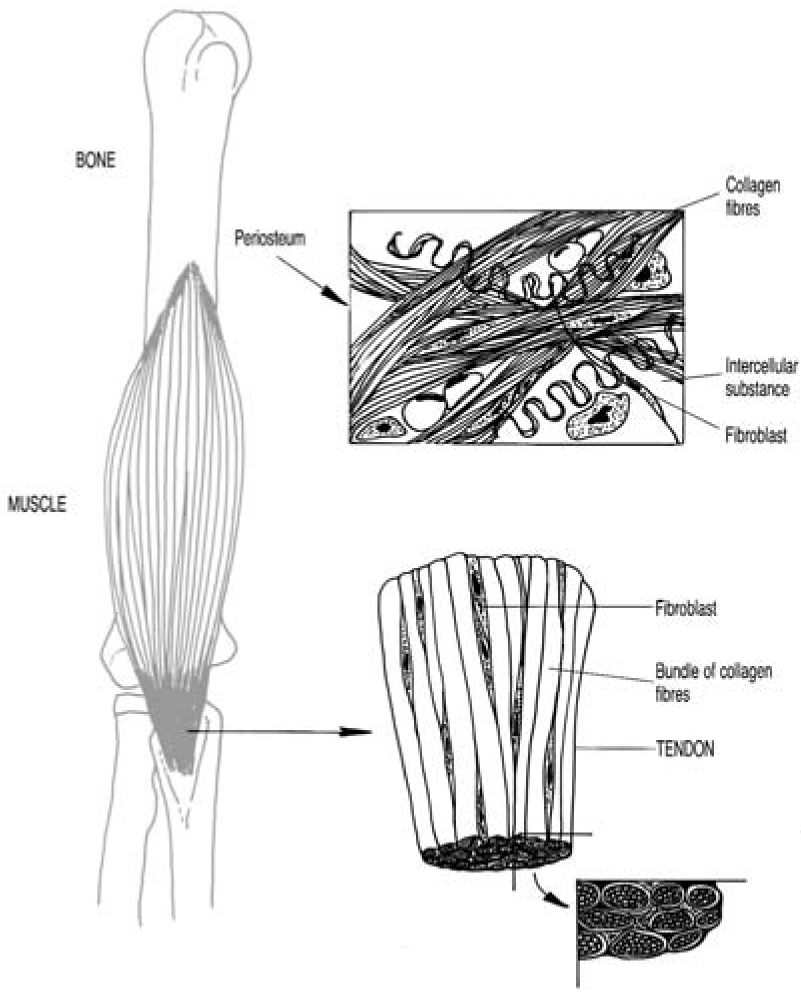

Fig. 1.1 Dense fibrous connective tissue seen covering bone as periosteum, and forming the tendon of a skeletal muscle.

sis can form the attachment of a muscle, such as the oblique abdominal muscles, which meet in the midline of the abdomen (see Chapter 10, Fig. 10.6). In the palm of the hand and the sole of the foot an aponeurosis lies deep to the skin and forms a protective layer for the tendons underneath (see Chapter 8, Fig. 8.21).

- A **retinaculum** is a band of dense fibrous tissue that binds tendons of muscles and prevents bowstring during movement. An example is the flexor retinaculum of the wrist, which holds the tendons of muscles passing into the hand in position (see Chapter 6, Fig. 6.15).
- **Fascia** is a term used for the large areas of dense fibrous tissue that surround the musculature of all the body segments. Fascia is particularly developed in the limbs, where it dips down between the large groups of muscles and attaches to the bone. In some areas, fascia provides a base for the attachment of muscles, for example the thoracolumbar fascia gives attachment to the long muscles of the back (see Chapter 10, Fig. 10.6).
- **Periosteum** is the protective covering of bones. Tendons and ligaments blend with the periosteum around bone (see Fig. 1.36).
- **Dura** is thick fibrous connective tissue protecting the brain and spinal cord (see Chapter 3, Fig. 3.22).

Clinical note-pad 1A: Contracture
Fibrous connective tissue loses its strength and elasticity when there is a loss of movement for any reason over a period of time. When muscles and tendons remain the same length for any length of time, e.g. due to muscle weakness in stroke, or joint pain in rheumatoid arthritis, permanent shortening occurs (contracture) which leads to joint deformity. See also Dupuytren's contracture, Clinical note-pad 6B.

Cartilage

Cartilage is a tissue that can be compressed and has resilience. The cells (chondrocytes) are oval and lie in a ground substance that is not rigid like bone. There is no blood supply to cartilage, so there is a limit to its thickness. The tissue has great resistance to wear, but cannot be repaired when damaged.

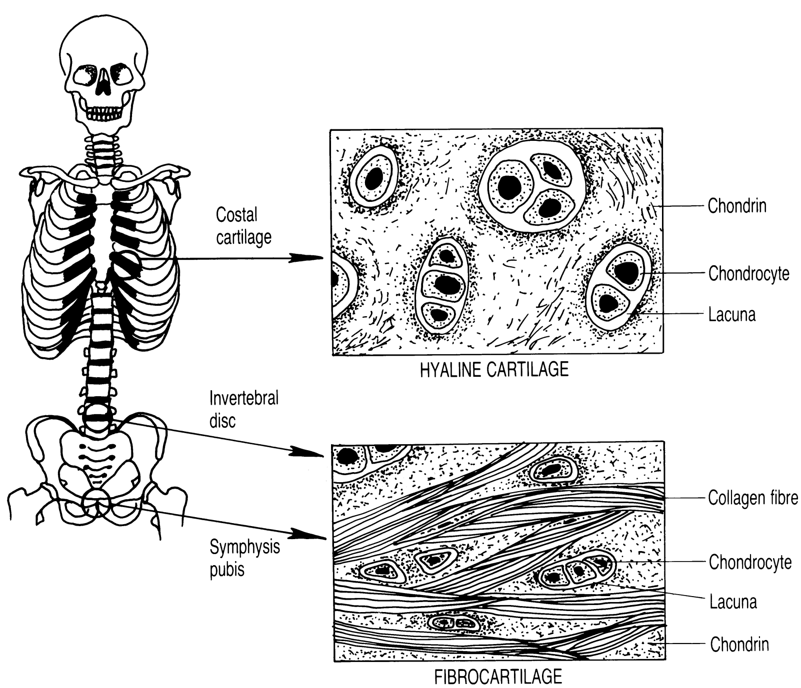

Fig. 1.2 Microscopic structure of hyaline and fibrocartilage, location in the skeleton of the trunk.

Hyaline cartilage is commonly called gristle. It is smooth and glass-like, forming a low-friction covering to the articular surfaces of joints. In the elderly, the articular cartilage tends to become eroded or calcifies, so that joints become stiff. Hyaline cartilage forms the costal cartilages which join the anterior ends of the ribs to the sternum (Fig. 1.2). In the developing foetus, most of the bones are formed in hyaline cartilage. When the cartilaginous model of each bone reaches a critical size for the survival of the cartilage cells, ossification begins.

- *LOOK at some large butcher's bones to see the cartilage covering the joint surfaces at the end. Note that it is bluish and looks like glass.*

Fibrocartilage consists of cartilage cells lying in between densely packed collagen fibres (Fig. 1.2). The fibres give extra strength to the tissue while retaining its resilience. Examples of where fibrocartilage is found are the discs between the bones of the vertebral column, the pubic symphysis joining the two halves of the pelvis anteriorly, and the menisci in the knee joint.

Bone

Bone is the tissue that forms the rigid supports for the body by containing a large proportion of calcium salts (calcium phosphate and carbonate). It must be remembered that bone is a living tissue composed of cells and an abundant blood supply. It has a greater capacity for repair after damage than any other tissue in the body, except for blood. The strength of bone lies in the thin plates (lamellae), composed of collagen fibres with calcium salts deposited in between. The lamellae lie in parallel, held together by fibres, and the bone cells or **osteocytes** are found in between. Each bone cell lies in a small space or lacuna, and connects with other cells and to blood capillaries by fine channels called canaliculi (Fig. 1.3a).

In **compact bone**, the lamellae are laid down in concentric rings around a central canal containing blood vessels. Each system of concentric lamellae (known as a Haversian system or an osteon) lies in a longitudinal direction. Many of these systems are closely packed to form the dense compact bone found in the shaft of long bones (Fig. 1.3b).

In **cancellous** or trabeculate bone, the lamellae form plates arranged in different directions to form a mesh. The plates are known as trabeculae and the spaces in between contain blood capillaries. The bone cells lying in the trabeculae communicate with each other and with the spaces by canaliculi (Fig. 1.3c). The expanded ends of long bones are filled with cancellous bone covered with a thin layer of compact bone. The central cavity of the shaft of long bones contains bone marrow. This organisation of the two types of bone produces a structure with great rigidity without excessive weight (Fig. 1.4). Bone has the capacity to remodel in shape in

Clinical note-pad 1B: Osteoporosis

Osteoporosis is literally a condition of porous bones, largely due to a depletion of calcium from the body. For a number of reasons, calcium loss exceeds calcium absorption from the diet, causing bone mass to decrease excessively. This leads to fractures occurring as a result of normal mechanical stresses upon the skeleton which it would normally withstand. Spontaneous fractures may occur for no apparent reason.

The whole skeleton is affected, but problems manifest most frequently in bones that bear weight or transmit large forces. Hence, the vertebrae may shrink and crumble or fracture. This causes height loss, kyphosis and back pain. A range of motor and sensory problems may occur owing to compression of the spinal nerves or the spinal cord. Fractures the neck of the femur and the wrist are common, often resulting from a fall.

Osteoporosis commonly affects people in the latter half of life, particularly elderly women who are at increased risk because of the sudden drop in oestrogen levels occurring at the menopause, combined with a lower bone mass than men. Other risk factors are poor dietary intake of calcium, vitamin D deficiency, physical inactivity, low body weight, smoking, high alcohol intake and some medications, e.g. long-term corticosteroid use. Osteoporosis is thought to affect one in three women in the UK, and can be a source of major disability.

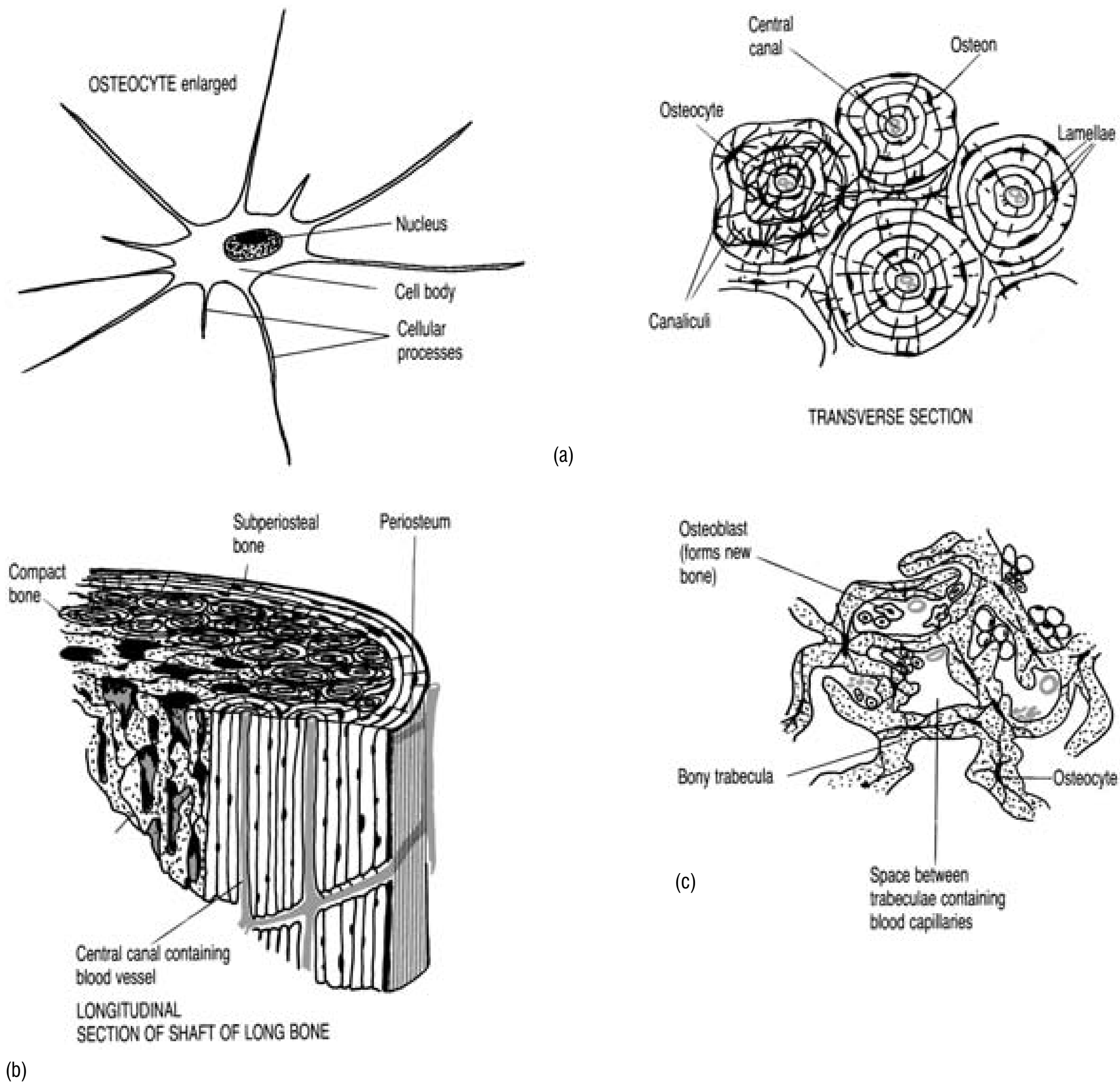

Fig. 1.3 Microscopic structure of bone: (a) an osteocyte (enlarged) and the organisation of osteons in compact bone seen in transverse section; (b) a section of the shaft of long bone; (c) cancellous bone showing trabeculae with osteocytes.

response to the stresses on it, so that the structure lines of the trabeculae at the ends of the bone follow the lines of force on the bone. For example, the lines of trabeculae at the ends of weight-bearing bones, such as the femur, provide maximum strength to support the body weight against gravity. Remodelling of bone is achieved by the activity of bone-forming cells known as osteoblasts, and bone-destroying cells known as osteoclasts; both types of cell are found in bone tissue. The calcium salts of bone are constantly interchanging with calcium ions in the blood, under the influence of hormones (parathormone and thyrocalcitonin). Bone is a living, constantly changing connective tissue that provides a rigid framework on which muscles can exert forces to produce movement.

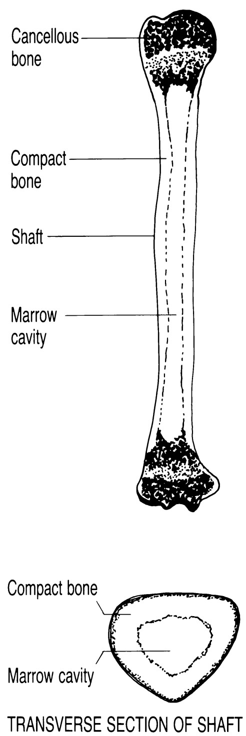

Fig. 1.4 Gross structure of long bone: longitudinal and transverse sections.

- *LOOK at any of the following examples of connective tissue that are available to you:*
  (1) Microscopic slides of dense fibrous tissue, cartilage and bone, noting the arrangement of the cellular and fibre content.
  (2) Dissected material of joints and muscles which include tendons, ligaments, aponeurosis and retinaculum.
  (3) Fresh butcher's bone: note the pink colour (blood supply), and the central cavity in the shaft of long bones.
  (4) Fresh red meat to see fibrous connective tissue around muscle.

ARTICULATIONS

Where the rigid bones of the skeleton meet, connective tissues are organised to bind the bones together and to form joints. It is the joints that allow movement of the segments of the body relative to each other. The joints or articulations between bones can be divided into three types based on the particular connective tissues involved. The three main classes of joint are **fibrous, cartilaginous** and **synovial**.

Fibrous joints

Here, the bones are united by dense fibrous connective tissue.

The **sutures** of the skull are fibrous joints that allow no movement between the bones. The edge of each bone is irregular and interlocks with the adjacent bone, a layer of fibrous tissue linking them (Fig. 1.5a).

A **syndesmosis** is a joint where the bones are joined by a ligament that allows some movement between the bones. A syndesmosis is found between the radius and the ulna (Fig. 1.5b). The interosseous membrane allows movement of the forearm.

A **gomphosis** is a specialised fibrous joint that fixes the teeth in the sockets of the jaw (Fig. 1.5c).

Cartilaginous joints

In these joints the bones are united by cartilage.

A **synchondrosis** or primary cartilaginous joint is a joint where the union is composed of hyaline cartilage. This type of joint is also called primary cartilaginous. The articulation of the first rib with the sternum is by a synchondrosis. During growth of the long bones of the skeleton, there is a synchondrosis between the ends and the shaft of the bone, where temporary cartilage forms the epiphyseal plate. These plates disappear when growth stops and the bone becomes ossified (Fig. 1.6a).

A **symphysis** or secondary cartilaginous joint is a joint where the joint surfaces are covered by a thin layer of hyaline cartilage and united by a disc of fibrocartilage. This type of joint (sometimes called secondary cartilaginous) allows a limited amount of movement between the bones by compression of the cartilage. The bodies of the vertebrae articulate by a disc of fibrocartilage (Fig. 1.6b). Movement between two vertebrae is small, but when all of the intervertebral discs are co pressed in a particular direction, considerable movement of the vertebral column occurs. Little movement occurs at the pubic symphysis, the joint where the right and left halves of the pelvis meet. Movement

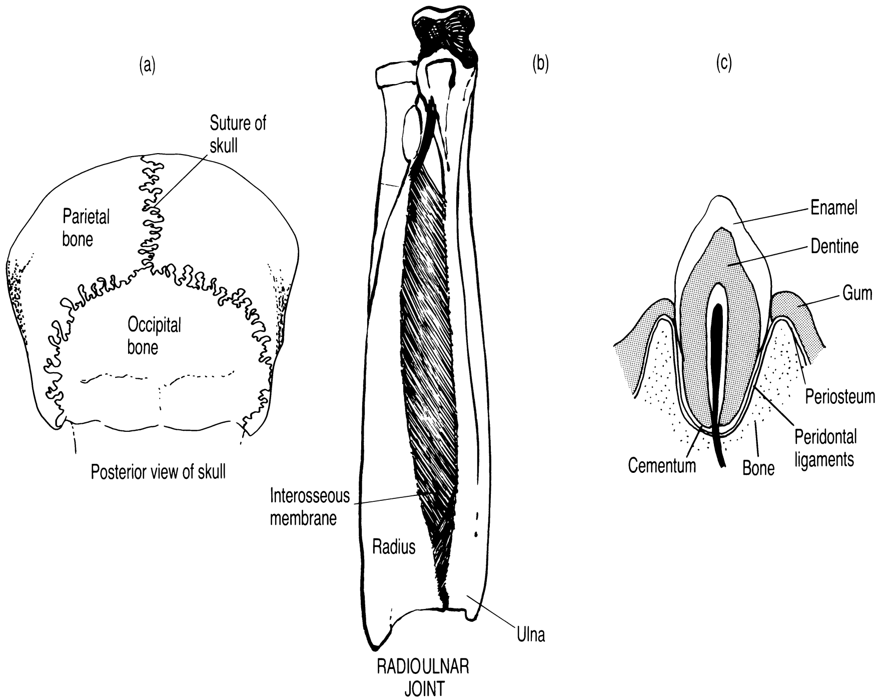

Fig. 1.5 Fibrous joints: (a) suture between bones of the skull; (b) syndesmosis between the radius and ulna; (c) gomphosis: tooth in socket.

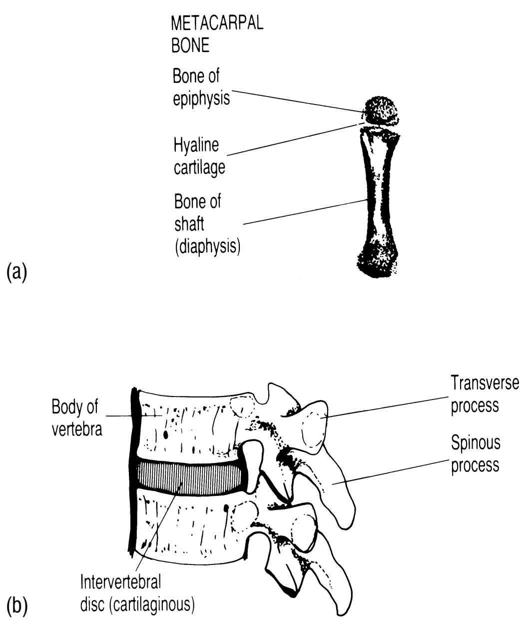

Fig. 1.6 Cartilaginous joints: (a) synchondrosis in a child's metacarpal bone, as seen on X-ray; (b) symphysis between the bodies of two vertebrae.

is probably increased at the pubic symphysis in the late stage of pregnancy and during childbirth, to increase the size of the birth canal.

Synovial joints

Synovial joints are the mobile joints of the body. There is a large number of these joints, which show a variety of form and range of movement. The common features of all of them are shown in the section of a typical synovial joint (Fig. 1.7) and listed as follows.

- **Hyaline cartilage** covers the ends of the two articulating bones, providing a low-friction surface for movement between them.
- A **capsule** of dense fibrous tissue is attached to the articular margins, or some distance away, on each bone. The capsule surrounds the joint like a sleeve.
- There is a **joint cavity** inside the capsule which allows free movement between the bones.
- **Ligaments**, bands or cords of dense fibrous tissue, join the bones. The ligaments may blend

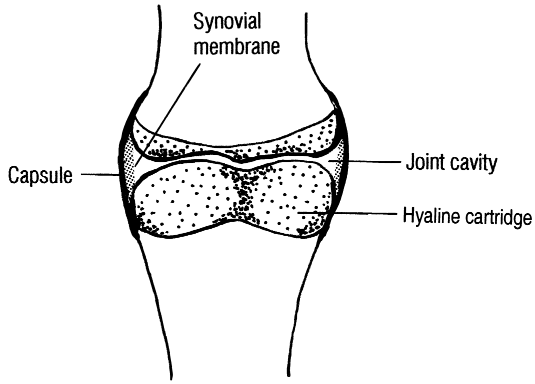

Fig. 1.7 Typical synovial joint.

with the capsule or they are attached to the bones close to the joint.
- A **synovial membrane** lines the joint capsule and all non-articular surfaces inside the joint, i.e. any structure within the joint not covered by hyaline cartilage.

One or more bursae are found associated with some of the synovial joints at a point of friction where a muscle, a tendon or the skin rubs against any bony structures. A bursa is a closed sac of fibrous tissue lined by a synovial membrane and containing synovial fluid. The cavity of the bursa sometimes communicates with the joint cavity. Pads of fat, liquid at body temperature, are also present in some joints. Both structures have a protective function.

All of the large movable joints of the body, for example the shoulder, elbow, wrist, hip, knee and ankle, are synovial joints. The direction and the range of their movements depend on the shape of the articular surfaces and the presence of ligaments and muscles close to the joint. The different types of synovial joint are described in Chapter 2 when the directions of movement at joints are considered.

Clinical note-pad 1C: Osteoarthritis and rheumatoid arthritis

Osteoarthritis is a degenerative disease occurring in the middle aged and elderly. There is a progressive loss of the articular cartilage in the weight-bearing joints, usually the hip and the knees. Bony outgrowths occur and the capsule becomes fibrosed. The joints become stiff and painful.

Rheumatoid arthritis is a systemic disease that can occur at any age (average 40 years) and it is more common in women. The peripheral joints (hands and feet) are affected first, followed by the involvement of other joints. Inflammation of the synovial membrane, bursae and tendon sheaths leads to swelling and pain which is relieved by drugs. Deformity is the result of erosion of articular cartilage, stretching of the capsule and the rupture of tendons.

SKELETAL MUSCLE

Skeletal muscle is attached to the bones of the skeleton and produces movement at joints. The basic unit of skeletal muscles is the **muscle fibre**. Muscle fibres are bound together in bundles to form a whole muscle, which is attached to bones by fibrous connective tissue. When **tension** develops in the muscle, the ends are drawn towards the centre of the muscle. In this case, the muscle is contracting in length and a body part moves. Alternatively, a body part may be moved by gravity and/or by an added weight, for example an object held in the hand. Now the tension developed in the muscle may be used to resist movement and hold the object in one position.

In summary, the tension developed allows a muscle:

- to shorten to produce movement
- to resist movement in response to the force of gravity or an added load.

Furthermore, muscles may develop tension when they are increasing in length. This will be considered in Chapter 2, muscle work.

Both muscle and fibrous connective tissue have elasticity. They can be stretched and return to the original length. The unique function of muscle is the capacity to shorten actively.

- *HOLD a glass of water in the hand. Feel the activity in the muscles above the elbow by palpating them with the other hand. The tension in the muscles is resisting the weight of the forearm and the water.*

- *LIFT the glass to the mouth. Feel the muscle activity in the same muscles as they shorten to lift the glass.*

Structure and form

The structure of a whole muscle is the combination of muscle and connective tissues, which both contribute to the function of the active muscle. In a whole muscle, groups of contractile muscle fibres are bound together by fibrous connective tissue. Each bundle is called a fasciculus. Further coverings of connective tissue bind the fasciculi together and an outer layer surrounds the whole muscle (Fig. 1.8).

The total connective tissue element lying in between the contractile muscle fibres is known as the parallel elastic component. The tension that is built up in muscle when it is activated depends on the tension in the muscle fibres and in the parallel

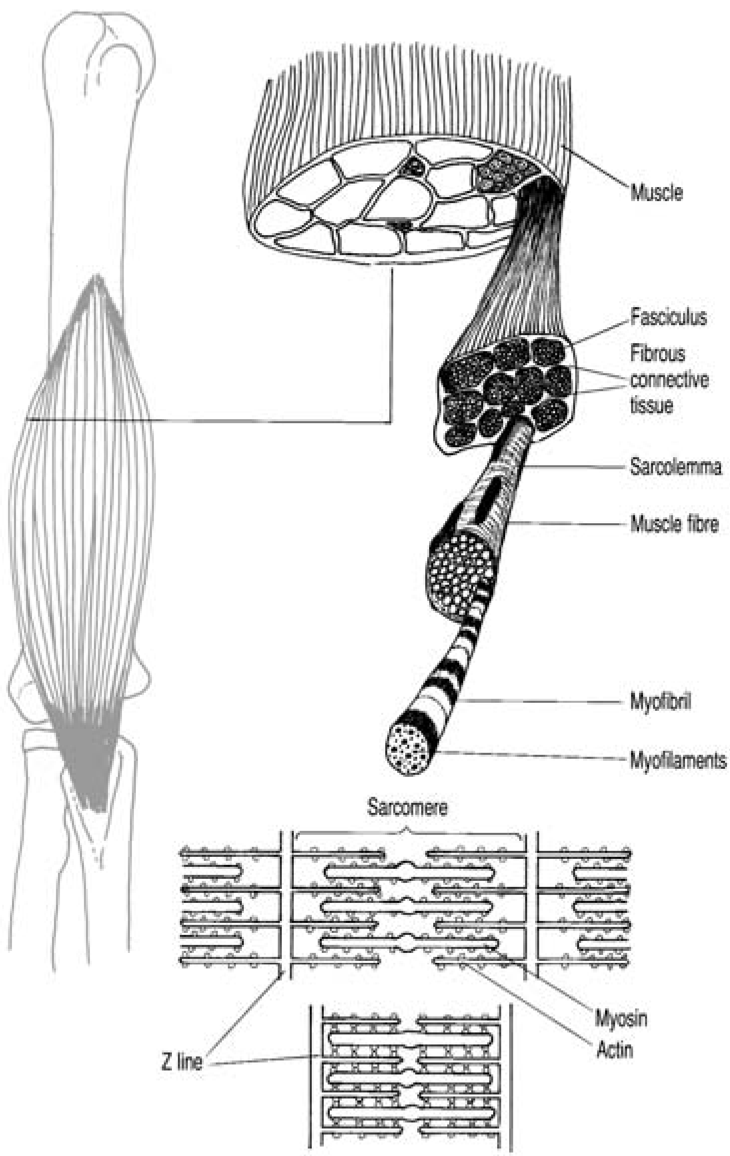

Fig. 1.8 Skeletal muscle: the organisation of muscle fibres into a whole muscle, and a sarcomere in the relaxed and the shortened state (as seen by an electron microscope).

elastic component. The fibrous connective tissue, for example a tendon, which links a whole muscle to bone is known as the series elastic component. The initial tension that builds up in an active muscle tightens the series elastic component and then the muscle can shorten. A model of the elastic and contractile parts of a muscle is shown in Fig. 1.9. If the connective tissue components lose their elasticity, through lack of use in injury or disease, a muscle may go into contracture (see Clinical notepad 1A). Lively splints are used to maintain elasticity and prevent contracture while the muscle recovers.

The individual muscle fibres lie within a muscle in one of the following two ways.

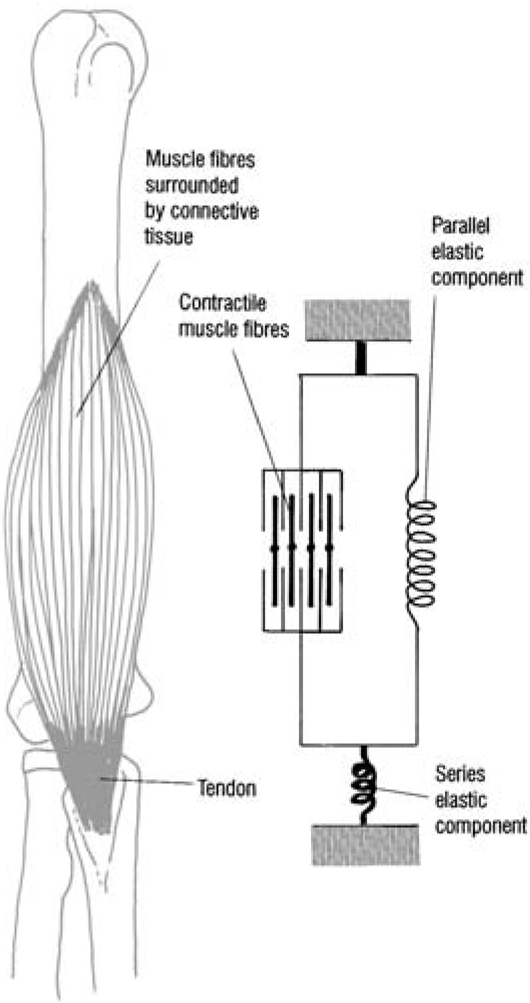

Fig. 1.9 Elastic components of muscle.

- Parallel fibres are seen in strap and fusiform muscles (Fig. 1.10a, b). These muscles have long fibres which are capable of shortening over the entire length of the muscle, but the result is a less powerful muscle.
- Oblique fibres are seen in pennate muscles. The muscle fibres in these muscles cannot shorten to the same extent as parallel fibres. The advantage of this arrangement, however, is that more muscle fibres can be packed into the whole muscle, so that greater power can be achieved.

The muscles with oblique fibres are known as unipennate, bipennate or multipennate, depending on the particular way in which the muscle fibres are arranged (Fig. 1.10c, d). Some of the large muscles of the body combine parallel and oblique arrangements. The deltoid muscle of the shoulder (see Chapter 5, Fig. 5.9) has one group of fibres that are multipennate and two groups that are fusiform, which combines strength to lift the weight of the arm with a wide range of movement. The form of a particular muscle reflects the space available and the demands of range and strength of movement.

Muscles have a limited capacity for repair, although a small area of damage to muscle fibres may regenerate. In more extensive damage, the connective tissue responds by producing more collagen fibres and a scar is formed. An intact nerve and adequate blood supply are essential for muscle function. If these are interrupted the muscle may never recover. Movement can then only be restored by other muscles taking over the functions of the damaged muscles.

Microscopic structure

A muscle fibre can just be seen with the naked eye. Each muscle fibre is an elongated cell with many nuclei surrounded by a strong outer membrane, the sarcolemma.. If one fibre is viewed under a light microscope, the nuclei can be seen close to the membrane around the fibre. The chief constituent of the fibre is several hundreds of myofibrils, strands of protein extending from one end of the fibre to the other (Fig. 1.8). The arrangement of the two main proteins, actin and myosin, that form each myofibril presents a banded appearance. The light and dark bands in adjacent myofibrils coincide, so that the whole muscle fibre is striated.

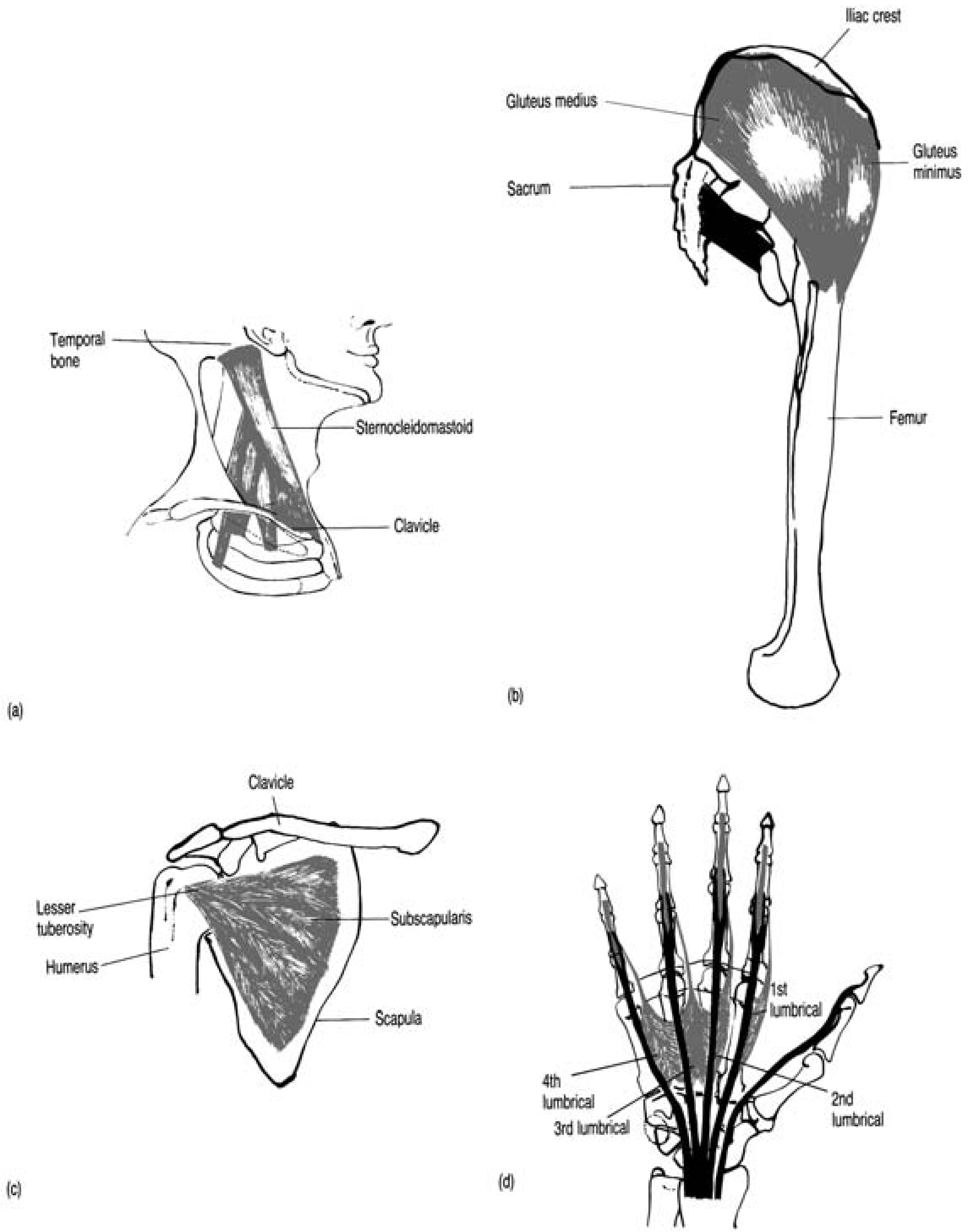

Fig. 1.10 Form of whole muscle: parallel fibres (a) strap and (b) fusiform; oblique fibres (c) multipennate and (d) unipennate and bipennate.

The electron microscope reveals the detail of the cross-striations in each myofibril. A repeating unit, known as the sarcomere, is revealed along the length of the myofibril. Each sarcomere links to the next one at a disc called the Z-line. The thin filaments of actin are attached to the Z-line and project towards the centre of the sarcomere. The thicker myosin filaments lie in between the actin strands. The darkest bands of the myofibril are where the actin and myosin overlap in the sarcomere.

The arrangement of the myosin molecules in the thick myosin filaments forms cross-bridges that link with special sites on the active filaments when the muscle fibre is activated. The result of this linking is to allow the filaments to slide past one another, so that each sarcomere becomes shorter. This, in turn, means that the myofibril is shorter, and since all the myofibrils respond together, the muscle fibre shortens.

- *LOOK at Figure 1.8, starting at the bottom, to identify the details of the structure of a muscle: (1) sarcomeres lie end to end to form a myofibril; (2) myofibrils are packed tightly together inside a muscle fibre; (3) muscle fibres are bound together in a fasciculus; and (4) fasciculi are bound to form a whole muscle.*

In active muscles, the energy required to develop tension is released by chemical reactions. Most of these reactions occur in structures called **mitochondria** (Fig. 1.11). All cells have mitochondria, but they are more abundant in muscle fibres where they lie adjacent to the myofibrils. The breakdown of adenosine triphosphate (ATP) and a 'back-up' phosphocreatine provide a high level of energy output in the muscle. The store of ATP is replenished in the mitochondria using oxygen and glucose brought by the blood in the network of capillaries surrounding muscle fibres (Fig. 1.11). In this way, the muscle fibres have a continuous supply of energy, as long as the supply of oxygen is maintained (aerobic metabolism). Glycogen is another source of energy that is stored in muscle fibres. When there is insufficient oxygen to replenish ATP by oxidative reactions, energy is released from breakdown of glycogen to maintain the ATP levels. This occurs during a short burst of high-level muscle activity.

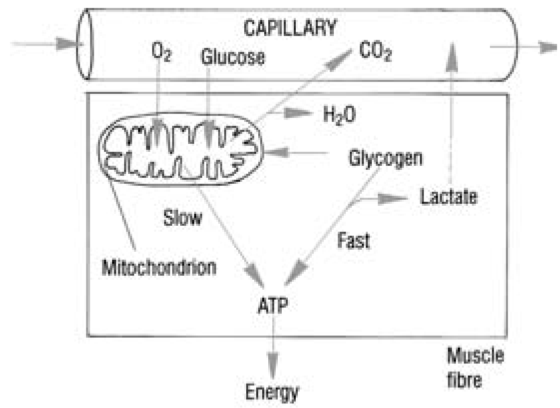

Fig. 1.11 Energy for muscle contraction (simplified). The 'slow' pathway predominates in type I fibres, where ATP is replenished by aerobic reactions to provide the energy for long periods of low-level activity. The 'fast' pathway predominates in type II fibres, where glycogen provides energy without oxygen for short bursts of high-level activity.

Adaptation of muscles to functional use

Not all muscle fibres in one muscle are the same. Two main types have been distinguished.

- **Slow** fibres, known as type I fibres, are red because they contain myoglobin which stores oxygen, like the haemoglobin in the blood, and they are surrounded by many capillaries. Energy supply for the slow fibres (called SO) is mainly from oxidative reactions. The slow fibres respond to stimulation with a slow twitch and they are resistant to fatigue.
- **Fast** fibres, known as type II fibres, are white with no myoglobin and have fewer capillaries per fibre. Energy is derived mainly from the breakdown of glucose and stored glycogen without oxygen. The fast fibres (called FG) respond with a fast twitch, but they are easily fatigued when the glycogen stores are used up.

Slow fibres are adapted for sustained postural activity, while the fast fibres are recruited for rapid intense bursts of activity, for example running, cycling and kitchen tasks such as cutting bread and chopping vegetables.

Skeletal muscle shows a remarkable capacity to adapt its structure to functional use. Both the relative proportion of slow and fast fibres and the number of sarcomeres in the myofibrils can change over time.

Muscle strength and bulk is increased by progressive resistance training programmes using weights or strength-training machines. The added strength is due to an increase in the number and size of the myofibrils, particularly in the fast muscle fibres which hypertrophy most readily. Less increase occurs in the slow fibre type. There is little evidence that similar training programmes can strengthen the muscles of patients with chronic degenerative disorders of the neuromuscular system. Any change may depend on the number of remaining intact fibres. For these patients, improvement in stamina rather than strength will be more useful for daily living in any case. Training for endurance in healthy young adults has the effect of changes in some fast fibres, which become more like slow fibres. The presence of these type IIA or FGO fibres increases the length of time that the muscle can perform movement without fatigue.

Studies of the effects of ageing have shown a progressive decrease in the size of fast fibres with fewer changes in slow fibres. These changes are most likely to be the response to a less active life. Fast fibres can increase in size in elderly people, so that exercise programmes are beneficial when there are no pathological changes present.

Muscles also change the number of sarcomeres in the myofibrils if a muscle is held in a shortened or lengthened position, for example by a plaster cast. Sarcomeres are lost in the shortened position and added in the lengthened position. This is an adaptation to changes in the functional length of the muscle. Any benefit, however, may be overridden by the changes in the muscle which lead to muscle contracture.

Clinical note-pad 1D: Myopathies
Neuromuscular disorders that are myopathic originate in the muscle, and may be inherited or acquired. There is muscle weakness in the proximal muscles, which is slowly progressive with muscle wasting.

Duchenne muscular dystrophy is an inherited myopathy that affects boys only. There is a rapid progression of muscle weakness that begins in childhood.

Acquired myopathy can result from infections, or endocrine disorders, or as a complication of steroid drug treatment.

BASIC UNITS OF THE NERVOUS SYSTEM

The functions of the nervous system in movement are: to conduct motor commands from the brain to the muscles; to regulate the activity in the cardiovascular and respiratory systems which supply the muscles with essential nutrients and oxygen; and to monitor changes in the environment that affect movement.

The properties of **neurones** are:

- excitation: neurones generate impulses in response to stimulation
- conduction of impulses between neurones (in one direction only).

Neurones are organised in networks or centres in the brain and the spinal cord. Activity in one centre is directed to a particular end, for example the location of a specific sensation. The output from one processing centre is then conducted to one or many other centres in a series of operations, for example from motor centres in the brain to the spinal cord. Information can also be conducted in parallel between processing centres.

The properties of neural networks are:

- processing of activity directed to a particular end
- relay of the output of processing to other centres in the nervous system.

This section is primarily concerned with the structure and the activity in the basic units of the nervous system, the neurones. Neural processing in specific centres in the central nervous system will be considered in Section III.

The neurone: excitation and conduction

Each neurone has a **cell body** and numerous processes extending outwards from the cell. The processes are living structures and their membrane is continuous with that of the cell body (Fig. 1.12). (Think of the cell body like a conker with spines projecting out in all directions.) The projections vary in length: short processes are called dendrites, and each neurone has one long process, the **axon**.

The dendrites are adapted to receive signals or impulses and pass them on to the cell body. Some neurones, particularly in the brain, have thousands of complex branching dendrites, so that signals from a large number of other neurones can be received.

The axon is the output end of every neurone. The length of an axon varies from a few millimetres to 1 m. Cell bodies of motor neurones in the spinal cord in the lower back have long axons that extend down the leg to supply the muscles of the foot. The axon may be surrounded by a sheath of **myelin**, a fatty material, which increases the rate at which impulses are conducted down the axon. The myelin is laid down between layers of membrane of Schwann cells that wrap around the axon. Gaps in the myelin occur between successive Schwann cells forming nodes of Ranvier (Fig. 1.12).

At the end of the neurone the axon branches, and each branch is swollen to form a bouton or synaptic knob. The boutons lie near a dendrite or cell body of another neurone. A typical motor neurone may have as many as 10,000 boutons on its surface which originate from other neurones. Axons also terminate on muscle fibres at a neuromuscular junction, on some blood vessels and in glands.

An impulse is a localised change in the membrane of a neurone. When a neurone is excited, the membrane over a small area allows charged particles (ions) to pass across the membrane, a process known as depolarisation, and an impulse is generated. The area of depolarisation then moves to the adjacent area and the impulse travels down the membrane in one direction only. Each impulse is the same size, like a morse code of dots only, but the signals carried can be varied by the rate and pattern of the impulses conducted along the neurone.

A **synapse** is the junction where impulses pass from one neurone to the next. Impulses always travel in one direction at a synapse, i.e. from the axon of one neurone to the dendrites and cell body of the next neurone. This ensures the one-way traffic in the nervous system.

When an impulse arrives at the end of the axon, a chemical is released from the boutons into the gaps between them and the next neurone. The chemical is known as a neurotransmitter and the gap is the synaptic cleft (Fig. 1.12). Each molecule of the neurotransmitter has to match special protein molecules, known as receptor sites, on the next neurone. When the transmitter locks on to the receptor site, the combination triggers the depolarisation of the membrane of the second neurone and impulses are conducted down it. Next, the transmitter substance is broken down by enzymes, taken up again by the boutons, re-formed and stored.

Each neurone has a threshold level of stimulation. The level of excitation reaching a neurone must be sufficient to depolarise the membrane, so that impulses are generated. Some impulses reaching a neurone affect the membrane in such a way that no impulses are propagated, this is known as **inhibition**. The source of inhibitory effects may be the presence of small neurones, the activity of which always produces inhibition, or the release of different transmitter substance from the boutons of the axon. The mechanism of inhibition will be discussed in more detail in Chapter 12.

Various transmitter substances have been identified in the nervous system. These include acetylcholine, adrenaline, dopamine and serotonin. Acetylcholine is the neurotransmitter released at most of the synapses in the pathways involved in movement, and also at the neuromuscular junctions. Drugs that prevent the release of acetylcholine at synapses are used as relaxants for muscles, for example in abdominal surgery.

Clinical note-pad 1E: Multiple sclerosis (MS)
In multiple sclerosis, changes in the myelin sheath around axons result in the formation of plaques, which affects the rate of conduction of nerve impulses. Axons in the central nervous system (brain and spinal cord) are affected, while those in the peripheral nervous system are not. The visual system seems to be most sensitive to plaque formation. Disturbance of both movement and sensation occurs. Fatigue and cognitive impairment are other common clinical features that affect function. The number of plaques and their sites vary between individuals and with time in the same individual, so that the disease sometimes follows a course of relapse and remission. In some patients there is progressive deterioration.

Neuroplasticity

The total number of neurones in the brain decreases after early adult life. Despite this loss, the brain retains its capacity to learn new skills and to use knowledge in different ways. Brain injury destroys neurones, either by direct damage to the neurones or as a result of reduced blood flow to the affected area of the brain. Recovery and rehabilitation may produce a, sometimes remarkable, return of function. These facts suggest that the nervous system has the capacity to be modified and new connections can be made, although the reasons are not entirely understood.

Biochemical changes have been explored as a possible explanation for the plasticity of neurones. During the early development of the basic networks of the brain, protein substances called nerve growth factors (NGFs) are present, but these are absent in the adult brain. Attempts to reintroduce NGFs in patients with degenerative diseases of the nervous system have been largely unsuccessful.

Evidence from animal experiments has demonstrated that structural changes in neurones can occur in a damaged area in some parts of the brain. These changes include new synaptic connections made by undamaged neurones and the sprouting of the axons to form synapses at sites that were previously activated by injured axons. The time when the fibres make new connections coincides with the return of function.

Current knowledge supports some plasticity of neurones in response to learning new skills and after injury. The cell body of each neurone is relatively fixed, but the synaptic connections that it makes with other neurones can be modified.

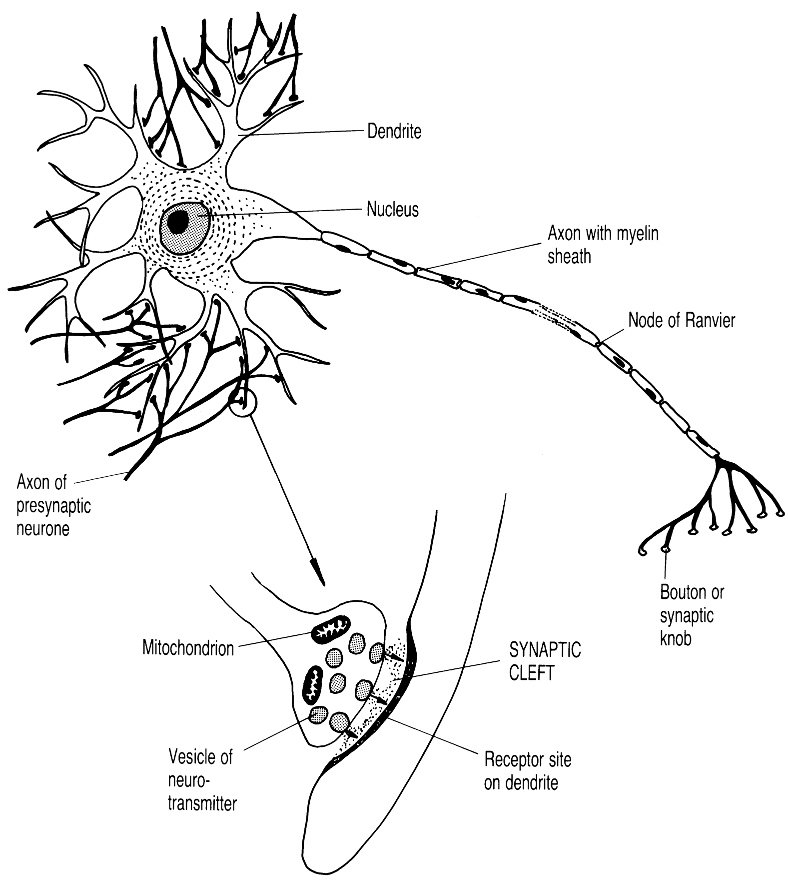

Fig. 1.12 Neurone and synapse; synaptic cleft enlarged.

Motor and sensory neurones

So far, the structure and properties of a typical neurone have been described. Electrochemical changes in the dendrites and cell body result in impulses that are propagated in one direction only, down the axon. In the organisation of the nervous system in development, the cell bodies of neurones lie in the central nervous system (brain and spinal cord) and the axons lie in the peripheral nerves that leave it to be distributed to all parts of the body.

Motor (efferent) neurones carry impulses away from the central nervous system to all parts of the body, or from the brain down to the spinal cord.

Sensory (afferent) neurones develop in a different way. The cell bodies of the sensory neurones are found in ganglia just outside the spinal cord. There are no synaptic junctions on the c ell bodies, and the axon divides into two almost immediately after it leaves the cell. The two branches formed by this division are a long process in a peripheral nerve that ends in a specialised sensory receptor, for example in the skin or a muscle, and a short process that enters the spinal cord and terminates in the central nervous system.

Figure 1.13a shows the arrangement of a typical sensory neurone. It is sometimes called 'pseudo-unipolar', since it has one axon but appears to be bipolar. Compare this with the multipolar motor neurone shown in Figure 1.12. Figure 1.13b shows the position of a sensory neurone in relation to the spinal cord, a spinal nerve and its branches. Note the cell body lying in a ganglion (swelling) and the axon entering the spinal cord. Sensory neurones carry impulses from the body towards the central nervous system, or from the spinal cord up to the brain.

Interneurones are those which lie only in the central nervous system and their axons do not extend into the nerves leaving it.

The motor unit

The motor neurones in the spinal cord, which activate the skeletal muscles, lie in a central H-shaped core of grey matter. These lower motor neurones are found in the anterior (ventral) limb of the grey matter. Neurones that activate a particular group of muscles lie together and form a motor neurone

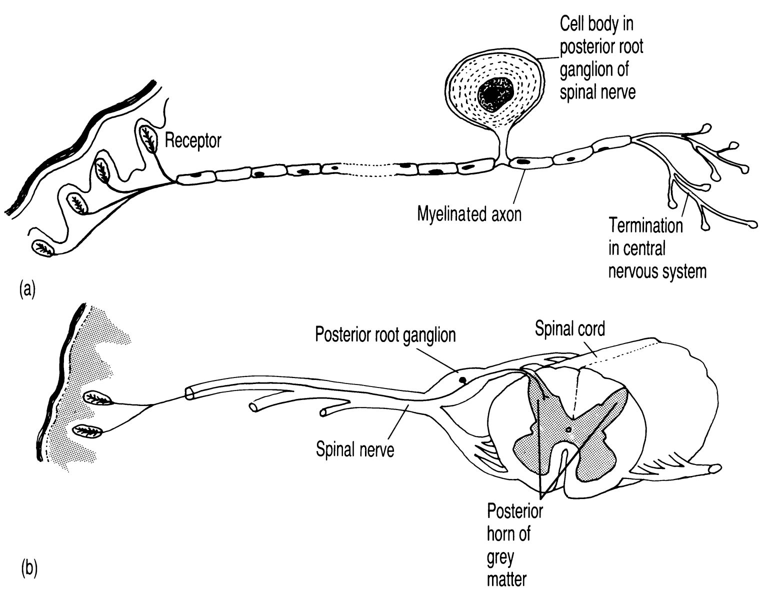

Fig. 1.13 Sensory neurones: (a) typical sensory neurone; (b) the position of a sensory neurone in a spinal nerve and the spinal cord.

Clinical note-pad 1F: Peripheral neuropathies

Neuromuscular disorders that are neurogenic originate in the nerve supply to the muscles, either in the spinal cord, in the nerve roots or in the peripheral nerves (see Chapter 4). Neuropathies of peripheral nerves affect sensory and motor axons, usually commencing distally, and are known as 'glove and stocking'. Muscle weakness and sensory loss occur. Peripheral neuropathy can occur as a complication of diabetes that is not under control.

Guillain–Barré syndrome is an acute peripheral neuropathy that affects motor axons. It usually follows a viral infection, and the resulting motor weakness involves the trunk and proximal limb muscles, mainly in the lower limbs. Recovery is nearly always complete unless there is severe involvement of the respiratory muscles or axonal damage.

pool (Fig. 1.14). The axons of these neurones lie in spinal nerves that branch to form the nerve supplying the muscle. There are fewer motor neurones in the pool than muscle fibres in the muscle, and therefore each neurone must supply a number of muscle fibres.

A **motor unit** consists of one motor neurone in the anterior horn of the spinal cord, its axon and all the muscle fibres innervated by the branches of the axon (Fig. 1.15). The number of muscle fibres in one motor unit depends on the function of the muscle rather than its size. Muscles performing large, strong movements have motor units with a large number of muscle fibres. For example, the large muscle of the calf has approximately 1900 muscle fibres in each motor unit. In muscles that perform fine precision movements, the motor units have a small number of muscle fibres (e.g. up to 100 in the muscles of the hand). The muscle fibres of one motor unit do not necessarily lie together in the muscle, but may be scattered in different fasciculi. The number of motor units that are active in a muscle at any one time determines the level of performance of the muscle.

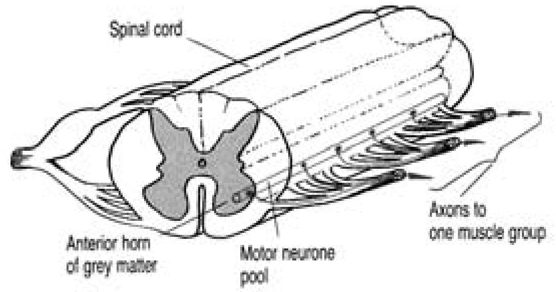

Fig. 1.14 Motor neurone pool in the spinal cord.

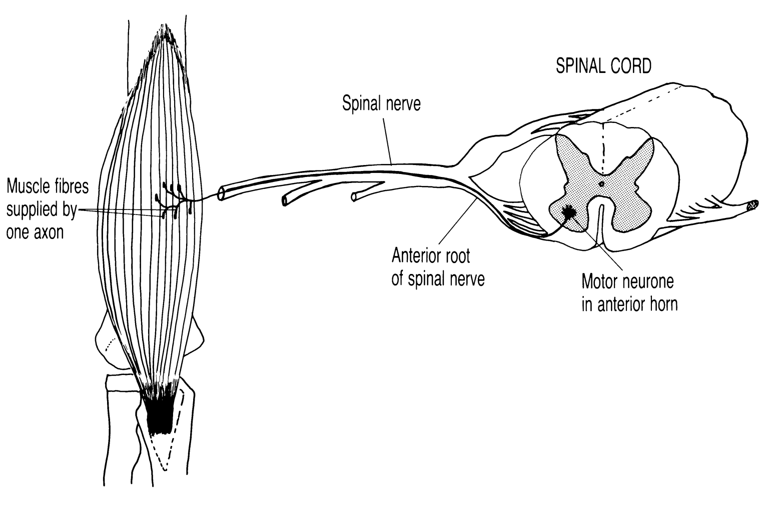

Fig. 1.15 Motor unit.

There are two types of motor unit.

- **Low-threshold** motor units supplying slow type I muscle fibres are involved in the sustained muscle activity that holds the posture of the body. The number of active motor units remains constant, but activity changes between all the low-threshold neurones. The slow type I muscle fibres do not fatigue easily and the activity is maintained over long periods.
- **High-threshold** motor units with large-diameter axons supplying fast type II muscle fibres are involved in fast, active movements, which move the parts of the body from one position to another. These motor units soon fatigue, but they are adapted for fast, strong movements such as running and jumping.

In a strong purposeful movement, such as pushing forwards on a door, the motor units are activated or recruited in a particular order. The slow units are active at the start of the movement and then the fast units become active as the movement reaches its peak.

All muscle activity includes a combination of slow and fast motor units. The slow units contribute more to the background postural activity, while the fast units play a greater part in rapid phasic movements. In manipulative activities the shoulder muscles have sustained postural activity to hold the limb steady, while the hand performs rapid precision movements, such as writing, sewing or using a tool.

Clinical note-pad 1G: Motor neurone disease
This is a progressive disorder of the motor neurones in the spinal cord. Muscle weakness and fatigue of the muscles of the limbs and the trunk occur, which become generalised to affect swallowing and speech. There is no sensory loss. Onset is usually around the age of 40 years, with rapid deterioration over 3–5 years.

Receptors

Receptors are specialised structures that respond to a stimulus and generate nerve impulses in sensory neurones. They are collectively the source of the sensory information that is transmitted into the central nervous system. While there is awareness of some receptor stimulation, a large amount of sensory processing and the resulting response occurs below consciousness. I can feel the pressure of the fingertips on the computer keys as I write and hear the telephone when it rings. At the same time, I am unaware of the receptors in the muscles in the neck and the balance part of the ear responding to changes in the position of the head so that my posture is adjusted to keep my eyes on the keyboard.

A system of receptors that respond to a specific stimulus is known as the **modality** of sensation, for example tactile modality. Several receptors of the same type may give input into one sensory neurone. The area covered by all the receptors activating one sensory axon is called a **receptive field**. There may be overlap in receptive fields, so that stimulation

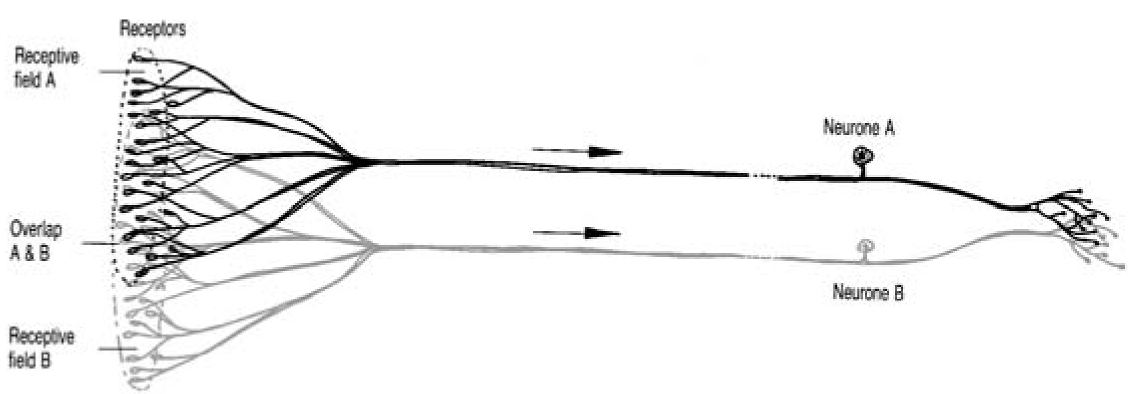

Fig. 1.16 Receptors in the receptive fields of two neurones.