Chapter objectives

After studying this chapter you should be able to:

1. Understand normal bone structure and function including bone remodelling and the different hormones that affect calcium metabolism.

2. Appreciate the aetiopathogenesis of the most common metabolic bone diseases.

3. Understand the epidemiology of osteoporosis and its clinical importance.

4. Assess the risk factors commonly associated with osteoporosis.

5. Describe the essential anatomy of the hip joint relevant to femoral neck fractures and other common diseases affecting the hip.

6. Understand the general principles of management of osteoporosis.

7. Understand the general principles of surgical treatment of hip fractures and their complications.
Introduction

The principal functions of the skeleton are mechanical support, maintenance of calcium homeostasis and haematopoiesis in the bone marrow. These can be disturbed in a variety of conditions encompassed by the general term, metabolic bone disease. Osteoporosis is the commonest metabolic bone disease. It is already an important public health problem in all developed countries and is becoming one in most developing countries. Osteoporosis means skeletal fragility leading to an increased risk of fracture. Hip fractures are the most important type of osteoporotic fracture, both in terms of direct health costs and social effects on the patient. In western countries, up to 1 in 2 women and 1 in 3 men will sustain an osteoporotic fracture during their lifetime. The cost of treating hip fractures alone has been estimated at 750 million pounds in the UK in 1995. In the USA, the total cost for treating all types of fractures (not just hip fractures) was estimated at $14 billion in 1999. Early diagnosis is now possible using precise methods of bone density measurement.

This chapter will review normal bone structure and function as well as the major metabolic bone diseases. Since this topic will be illustrated by a case in which an osteoporotic hip fracture has occurred, the key anatomy of the hip joint will also be reviewed.

Normal skeletal structure and function

Bones are extremely dense connective tissue that, in various shapes, constitutes the skeleton. Although one of the hardest structures in the body, bone maintains a degree of elasticity owing to its structure and composition. Bone is enclosed, except where it is coated with articular cartilage, in a fibrous outer membrane called the periosteum. Periosteum is composed of two layers, an outer fibrous layer and a deeper elastic layer containing osteoblasts that are capable of proliferating rapidly when a fracture occurs, as will be discussed further in Chapter 10. In the interior of the long bones is a cylindrical cavity (called the medullary cavity) filled with bone marrow and lined with a membrane composed of highly vascular tissue called the endosteum.

Types of bone: cortical and cancellous

There are two types of bone: (a) compact or cortical bone and (b) trabecular or cancellous bone. Cortical bone is found principally in the shafts (diaphyses) of long bones. It consists of a number of irregularly
spaced overlapping cylindrical units termed Haversian systems. Each consists of a central Haversian canal-surrounded by concentric lamellae of bony tissue (Fig. 5.2A). Trabecular bone is found principally at the ends of long bones, and in vertebral bodies and flat bones. It is composed of a meshwork of trabeculae within which are intercommunicating spaces (Fig. 5.2B).

The skeleton consists of approximately 80% cortical bone, largely in peripheral bones, and 20% trabecular bone, mainly in the axial skeleton. These amounts vary according to site and relate to the need for mechanical support. While trabecular bone accounts for the minority of total skeletal tissue, it is the site of greater bone turnover because its total surface area is greater than that of cortical bone.

**Blood supply of bone**

Bones are generally richly supplied with blood, via periosteal vessels, vessels that enter close to the articular surfaces and nutrient arteries passing obliquely through the cortex before dividing into longitudinally directed branches. Loss of the arterial supply to parts of a bone results in death of bone tissue, usually called avascular necrosis or osteonecrosis. Certain bones in the body are prone to this complication, usually after injury, including the head of the femur (discussed later in this chapter), the scaphoid bone in the wrist, the navicular in the foot and the tibial plateau. Nutrient arteries to the scaphoid bone are large and numerous at the distal end but become sparse and finer as the proximal pole is approached. Fractures of the scaphoid, especially of the waist or proximal pole, may be associated with inadequate blood supply resulting in necrosis and later secondary osteoarthritis. In the foot, the navicular bone is the last tarsal bone to ossify and its ossification centre may be dependent on a single nutrient artery. Compressive forces on weight bearing are thought to be the cause of avascular necrosis of the ossification centre, which usually presents as a painful limp in a child. This condition is also known as Kohler’s disease.

**Calcium homeostasis and hormonal control**

In addition to its role as a support structure, bone’s other primary function is calcium homeostasis. More than 99.9% of the total body calcium resides in the skeleton. The maintenance of normal serum calcium depends on the interplay of intestinal calcium absorption, renal excretion and skeletal mobilisation or uptake of calcium. Serum calcium represents less than 1% of total body calcium but the serum level is extremely important for maintenance of normal cellular functions. Serum calcium regulates and is regulated by three major hormones: parathyroid hormone (PTH), 1,25-dihydroxyvitamin D and calcitonin (Fig. 5.3). Parathyroid hormone is an 84-amino acid peptide secreted by the four parathyroid glands located adjacent to the thyroid gland in the neck. Calcitonin is a 32-amino acid peptide secreted by the parafollicular cells of the thyroid gland. Vitamin D, from dietary sources (D₃) or synthesised in skin (D₂), is converted to 25-hydroxyvitamin D in the liver and then to 1,25-dihydroxyvitamin D in the kidney.

PTH and 1,25-dihydroxyvitamin D are the major regulators of calcium and bone homeostasis. Although calcitonin can directly inhibit osteoclastic bone resorption, it appears to play a relatively minor role in calcium homeostasis in normal adults. PTH acts on the kidney to increase calcium reabsorption, phosphate excretion and 1,25-dihydroxyvitamin D production. It acts on bone to increase bone resorption. 1,25-dihydroxyvitamin D is a potent stimulator of bone resorption and an even more potent stimulator of intestinal calcium (and phosphate) absorption. It is also necessary for bone mineralisation. Intestinal calcium absorption is probably the most important calcium homeostatic pathway.

A number of feedback loops operate to control the level of serum calcium and the two major calcium homeostatic hormones. A calcium-sensing receptor, identified in parathyroid and kidney cells but also found in other tissues, that senses extracellular calcium plays a critical role in calcium homeostasis. Low serum calcium levels stimulate 1,25-dihydroxyvitamin D synthesis directly through stimulation of PTH release (and synthesis). The physiological response to increasing levels of PTH and 1,25-dihydroxyvitamin D is a gradual rise in serum calcium level. To prevent an elevated level of serum calcium, a second set of feedback loops operate to decrease PTH and 1,25-dihydroxyvitamin D levels. These feedback loops maintain serum calcium within a narrow physiological range. Disturbances in these control mechanisms or over/underproduction of these three major hormones can lead to various clinical states, discussed in more detail below. More recently, a PTH-related peptide (PTHrP) has been identified as playing a role in calcium homeostasis, especially in the fetus and in the growing skeleton.

**Cellular basis of bone remodelling**

The structural components of bone consist of extracellular matrix (largely mineralised), collagen and cells.
BONE STRUCTURE AND FUNCTION IN NORMAL AND DISEASE STATES

Subperiosteal outer circumferential lamellae

Concentric lamellae of osteon (Haversian system)

Marrow meshwork surrounds sinusoids (contains haematopoietic cells, fibroblasts, and fat cells)

Peripheral arteriolar branch of nutrient artery gives rise to capillaries that enter Volkmann's canals of cortical (compact) bone

Central arteriolar branches of nutrient artery

Trabeculae project into central medullary (marrow) cavity

Nutrient artery eventually anastomoses with distal metaphyseal arteries

Vein

Nutrient artery eventually anastomoses with distal metaphyseal arteries

Marrow meshwork surrounds sinusoids (contains haematopoietic cells, fibroblasts, and fat cells)

Capillaries in haversian canals

Capillaries in Volkmann's canals

On cut surfaces (as in sections), trabeculae may appear as discontinuous spicules

Osteoid (unmineralized matrix)

Active osteoblasts produce osteoid

Inactive osteoblasts (lining cells)

Marrow spaces contain haematopoietic cells and fat

Trabeculae

Osteoclasts (in Howship's lacunae)

Osteocytes

Fig. 5.2
Structure of bone: (A) cortical (compact) bone; (B) trabecular bone.
The collagen fibres are of type I, comprise 90% of the total protein in bone and are oriented in a preferential direction giving lamellar bone its structure. Spindle- or plate-shaped crystals of hydroxyapatite \[3\text{Ca}_3(\text{PO}_4)_2\cdot(\text{OH})_2\] are found on the collagen fibres, within them, and in the ground substance. The ground substance is primarily composed of glycoproteins and proteoglycans. These highly anionic complexes have a high ion-binding capacity and are thought to play an important role in the calcification process. Numerous non-collagenous proteins have been identified in bone matrix, such as osteocalcin synthesised by the osteoblasts, but their role is unclear.

The principal cells in bone are the osteoclasts and osteoblasts (including bone-lining cells and osteocytes). Osteoclasts, the cells responsible for resorption of bone, are derived from haematopoietic stem cells. Osteoblasts are derived from local mesenchymal cells. They are the pivotal bone cell, responsible directly for bone formation and indirectly, via paracrine factors, for regulating osteoclastic bone resorption.

Various cytokines control osteoclast recruitment and activity, including interleukin-1β (IL-1β) and IL-6. Recently, a transmembrane protein belonging to the tumour necrosis factor superfamily, called osteoclast differentiating factor (or ODF), has been identified as playing an important role in osteoclast differentiation and activity (Fig. 5.4A). Its receptor is called RANK (receptor activator of NFκB) since, after binding, a transcriptional factor known as NFκB translocates to the nucleus and appears responsible for expression of genes that lead to the osteoclast phenotype. This process is inhibited by a soluble receptor, osteoprotegerin (OPG), which competes for binding of ODF to produce an inactive complex.

Bone is continually undergoing renewal called remodelling (Fig. 5.4B). In the normal adult skeleton, new bone laid down by osteoblasts exactly matches osteoclastic bone resorption, i.e. formation and resorption are closely ‘coupled’. Although there is a lesser amount of trabecular bone than cortical bone in the skeleton, because trabecular bone ‘turns over’ between 3–10 times more rapidly than cortical bone, it is more sensitive to changes in bone resorption and formation. Most bone turnover occurs on bone surfaces, especially at endosteal surfaces. Moreover, the rate of remodelling differs in different locations according to physical loading, proximity to a synovial joint or the presence of haemopoietic rather than fatty tissue in adjacent marrow.

Bone remodelling follows an ordered sequence, referred to as the basic multicellular unit of bone turnover or bone remodelling unit (BMU). In this cycle, bone resorption is initiated by the recruitment of osteoclasts, which act on matrix exposed by proteinases derived from bone lining cells. A resorptive pit (called a Howship’s lacuna) is created by the osteoclasts. Osteoclasts have a convoluted membrane called a ruffled border through which lysosomal enzymes are released into pockets, causing matrix resorption. This resorptive phase is then followed by a bone formation phase where osteoblasts fill the lacuna with osteoid. The latter is subsequently mineralised to form new bone matrix. This cycle of coupling of bone formation and resorption is vital to the maintenance of the integrity of the skeleton. Uncoupling of the remodelling cycle, so that bone resorption or formation are in excess of each other leads to net bone change (gain or loss).
Bones develop by one of two processes, either:
- from a preformed cartilaginous structure (endochondral ossification), or
- de novo at specific sites in the skeleton (intramembranous ossification).

Subsequent skeletal growth involves remodelling of bone. In the growing skeleton, the long bones consist of a diaphysis (or shaft) separated from the ends of the
bone (called the epiphyses) by cartilage. The part of the diaphysis immediately adjacent to the epiphysial cartilage is the site of advancing ossification and is known as the metaphysis. Endochondral ossification is a complex process in which the growth plate cartilage is progressively replaced by bone. The growth plate (physis) and bone front steadily advance away from the bone centre, resulting in progressive elongation of bone. Longitudinal growth continues while the growth plate remains open.

Growth plates start to close after puberty in response to the surge in circulating oestrogen. Several hormones including growth hormone, insulin-like growth factor-1 (IGF-I) and PTHrP play a role in bone growth. With growth throughout early childhood, bone size and mass gradually increase in a linear fashion. Then between the onset of puberty and young adulthood, skeletal mass approximately doubles. Most of the increase in bone mass in early puberty is due to increases in bone size. In cortical bone, both the inner (endocortical) and outer (periosteal) diameters increase, owing to enhanced resorption and apposition on these surfaces respectively. Later in puberty, bone density increases again but this is less related to increase in bone size. Gains in bone mineral density during puberty are dependent on the pubertal stage.

Growth ceases when closure of the growth plate occurs, but bone mass and density may continue to increase beyond this time by a process called consolidation. The maximum skeletal mass achieved is termed the peak bone mass. The age at which this is attained varies in different skeletal sites. For example, forearm peak bone mass in children occurs around age 25. However, in the lumbar spine and femoral neck, peak bone mass may be achieved by age 18.

**The adult skeleton**

In both men and women, bone mineral loss from the skeleton starts from age 40–50, again depending upon the skeletal site. In addition, in women bone loss can be rapid immediately after the menopause. Bone size also contributes to bone strength. Thus men have higher bone mineral density (and a lower fracture risk) than women, because they have bigger bones. In clinical practice, osteoporosis is usually defined in relation to the degree to which bone mineral density is reduced. Bone mineral density is usually expressed as a T score (number of standard deviations from the young normal mean) or Z score (number of standard deviations from the age-matched mean). Osteoporosis is usually defined as a T score below −2.5 (Fig. 5.5).

### Table 5.1

**Biochemical markers of bone turnover**

<table>
<thead>
<tr>
<th>Bone formation</th>
<th>Bone resorption</th>
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</thead>
<tbody>
<tr>
<td><strong>Serum</strong></td>
<td>Tartrate-resistant acid phosphatase</td>
</tr>
<tr>
<td>Osteocalcin</td>
<td>Cross-linked C telopeptide of type 1 collagen</td>
</tr>
<tr>
<td>Bone-specific alkaline phosphatase</td>
<td></td>
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<tr>
<td><strong>Urine</strong></td>
<td>Pyridinolone, deoxypyridinoline</td>
</tr>
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<td>–</td>
<td>N telopeptide of collagen cross-links</td>
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<td></td>
<td>C telopeptide of collagen cross-links</td>
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</table>

*Fig. 5.5* Diagnostic categories based upon bone densitometry according to the number of standard deviations from the young normal mean (a T score of zero). T scores between –1 and –2.5 are called low bone mass or osteopenia. T scores below –2.5 are called osteoporosis.
not matched by a concomitant increase in formation. The bone matrix is normally mineralised but there is simply less bone. In most forms of osteoporosis the loss of bone is not evenly distributed throughout the skeleton. For reasons that are not clear, some struts of trabecular bone are resorbed completely, resulting in a loss of connectivity between adjacent bone plates (Fig. 5.7). This contributes to markedly decreased bone strength and fracture risk. Because the remodelling surface-to-volume ratio of trabecular bone is high, bone loss tends to affect this type of bone, such as that in the spine and hip, to a greater extent.

An imbalance between resorption and formation occurs with ageing but also in several other circumstances. These include:

- when bone is subject to reduced mechanical loading as a result of bed rest or immobilisation
- the presence of reduced sex hormone concentrations such as after the menopause in females
- the presence of excess corticosteroids usually given as treatment for a variety of conditions such as arthritis or asthma.

Loss of bone mineral has no clinical effect itself, unless a fracture occurs. Common sites of fracture due to osteoporosis include the spine (Fig. 5.8), wrist, hip or pelvis after minor trauma, but almost any bone can be affected. Vertebral fractures can manifest as loss of anterior height (wedge fractures), loss of midvertebral height (called codfish vertebrae) or loss of anterior, middle and posterior height (called compression or crush fractures). Vertebral fractures may present with

### Metabolic bone disease

Metabolic bone disease is a loose term that encompasses generalised diseases of bone in which abnormal bone remodelling results in a reduced volume of mineralised bone and/or abnormal bone architecture. These processes in turn give rise to bone pain and usually an increased risk of fracture. The commonest metabolic bone diseases are osteoporosis, osteomalacia, Paget’s disease, hyperparathyroidism, and bone disease associated with renal failure (renal osteodystrophy).

### Osteoporosis

Osteoporosis is characterised by an imbalance in remodelling – a relative increase in resorption that is not matched by a concomitant increase in formation. The bone matrix is normally mineralised but there is simply less bone. In most forms of osteoporosis the loss of bone is not evenly distributed throughout the skeleton. For reasons that are not clear, some struts of trabecular bone are resorbed completely, resulting in a loss of connectivity between adjacent bone plates (Fig. 5.7). This contributes to markedly decreased bone strength and fracture risk. Because the remodelling surface-to-volume ratio of trabecular bone is high, bone loss tends to affect this type of bone, such as that in the spine and hip, to a greater extent.

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### Case note: measurement of bone mineral density

In most subjects with hip fractures, bone mineral density is not routinely measured since low bone mineral density is evident in virtually all elderly subjects. However, Mrs Jones has sustained her hip fracture at a relatively early age, so measurement is appropriate in her case. Her bone mineral density reveals a femoral neck T score of −4.0 and her Z score is −2.3 (Fig. 5.6). Her T score confirms that she has osteoporosis (being below −2.5). However, since her Z score is also considerably below what is expected for her age, secondary causes for osteoporosis or other metabolic bone disease should be sought.

![Fig. 5.6](image.png)

Mrs Jones’ right hip scan measured by dual energy X-ray absorptiometry showing her values plotted against the age-related normal range.
an acute self-limiting episode of back pain or subclinically as height loss and increasing thoracic kyphosis (forward bending of the spine).

**Osteomalacia**

Osteomalacia occurs when there is insufficient calcium and phosphate to mineralise newly formed osteoid. Since bone mineral – hydroxyapatite – gives bone its compressive strength, osteomalacic bones are softer and more liable to bend, become deformed, or fracture. Rickets is essentially the same problem – impaired mineral deposition in bone – when it occurs in children or adolescents. Rickets is only seen before the growth plate disappears. Osteomalacia and rickets usually occur as a result of vitamin D deficiency, such as in institutionalised patients, those with reduced sun exposure and in patients with gut malabsorption or poor nutrition. Rarely, an inability of the kidney to retain phosphate results in phosphate wasting and chronic hypophosphataemia and osteomalacia/rickets (Fig. 5.9).

**Paget’s disease**

Paget’s disease is a condition in which localised areas of bone show markedly increased bone turnover. There is gross disorganisation of newly reformed bone, triggered by overactive osteoclasts. These local areas of increased remodelling may cause deformity such as bowing of a limb or enlargement (Fig. 5.10), bone pain, increased fracture risk and, rarely, neoplastic transformation to osteosarcoma. The X-ray appearance often shows a complex pattern of radiolucency (owing to early osteolysis) and bone expansion and sclerosis (generally later). Paget’s disease is now treated most effectively by drugs called bisphosphonates, which are discussed later in this chapter (p. 84).
Hyperparathyroidism and renal osteodystrophy

Overproduction of parathyroid hormone, usually by a benign tumour (adenoma) of one of the four parathyroid glands, leads to increased bone resorption and elevation of the serum calcium level. Hyperparathyroidism can occur as a discrete condition (primary hyperparathyroidism) or may be secondary to renal failure (owing to decreased production of 1,25-dihydroxyvitamin D by the kidney). In patients with renal failure, the mixed picture of secondary hyperparathyroidism and osteomalacia is commonly called renal osteodystrophy.

Osteoporosis: pathophysiology and risk factors

Fracture risk is directly related to bone mineral density. Bone mineral density at any age is the result of the peak bone mass achieved and subsequent bone loss (postmenopausal and age-related). This section

<table>
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<tr>
<th>Disorder</th>
<th>Ca</th>
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<th>Alkaline phosphatase</th>
<th>PTH</th>
<th>25-OH-vitamin D</th>
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</thead>
<tbody>
<tr>
<td>Primary hyperparathyroidism</td>
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<td>↓</td>
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</tr>
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<tr>
<td>Paget's disease</td>
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<tr>
<td>Osteoporosis</td>
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will discuss factors contributing to bone loss that are considered important in the aetiology of osteoporosis.

**Sex hormone deficiency**

Since Albright first observed that the majority of women with osteoporosis were postmenopausal, sex hormone deficiency, resulting from natural or surgical menopause, has been recognised to cause bone loss. Replacement of oestrogen following the menopause prevents bone loss and fractures. In men, testosterone deficiency is similarly associated with bone loss and can be reversed with testosterone replacement.

Menopausal status is probably the most important risk factor for osteoporosis of all. Women with an early menopause (considered to be present in women who become menopausal before 45 years of age) or having bilateral oophorectomy have lower bone mineral density at all sites and increased risk of subsequent fracture. The earlier the menopause, the greater the risk appears to be.

**Race and genetic influences**

Racial factors influence bone mineral density. For example, blacks, whether in Africa or in the USA, appear to have greater bone density than whites of the same age and sustain fewer related fractures. People of Asian origin often have lower bone densities and often higher fracture rates than whites.

Similarly, it is established from twin and family studies that bone mineral density at both the appen-
Epidemiology of fractured hip

Hip fractures, or fractures of the neck of the femur, are the most serious fractures in older people, at both an individual and population level. Their economic cost is enormous because of the need for hospitalisation, surgery and rehabilitation. Complications are frequent, especially in those with co-morbid conditions (which are common in subjects aged >80 years). 20% of those who fracture their hip die within a year and most survivors never regain their pre-fracture level of physical function. Hip fractures lead to permanent admission to a nursing home in approximately 20% of patients.

The incidence of hip fracture rises dramatically with increasing age in most countries. Hip fractures are more common in women than in men: epidemiological studies suggest a white woman has a 16% lifetime risk of suffering a hip fracture and a white man has a 5% lifetime risk.

Risk factors for falls

The high rate of hip fracture in older people is due not only to their lower bone strength but also to their...
increased risk of falling. Established risk factors for falls and hence hip fracture include impaired balance, muscle weakness and psychotropic medication. Although physical activity levels are related to bone density, the benefits of exercise in the elderly probably relate more to reduced risk of falling than increases in bone strength.

**Essential anatomy of the hip**

The hip joint, like most of the lower limb joints, is a synovial joint. Synovial joints are characterised by the following features:

- articular cartilage covering the bony surfaces
- a joint cavity containing viscous synovial fluid
- a surrounding articular capsule that consists of outer fibrous tissue and an inner lining called a synovial membrane.

The hip joint is also a ball-and-socket joint, formed by the articulation of the rounded head of the femur with the cup-shaped acetabulum of the pelvis. It combines a wide range of motion with great stability. This is possible because of the deep insertion of the head of the femur into the acetabulum, the strong articular capsule and the muscles that pass over the joint and insert at a distance below the head of the femur. These anatomical features provide leverage for the femur and stabilisation for the joint. When a hip fracture occurs, it is usually at one of three sites (Fig. 5.11): either high in the femoral neck (subcapital), across the neck itself (cervical) or in the trochanteric region (pertrochanteric).

The acetabulum is formed by fusion of three pelvic bones: the ischium, ilium and pubis (Fig. 5.12). There is a circular rim of fibrocartilage, which forms the glenoid labrum around the acetabular cavity, the lower portion of which is incomplete, forming the acetabular notch. Blood vessels pass into the joint through a foramen formed by a transverse ligament over this notch. The acetabulum is deepest and strongest superiorly and posteriorly, where it is subject to the greatest strain when a person is in the erect position.

The hip joint has a strong, dense articular capsule. It is attached proximally to the edge of the acetabulum, the glenoid labrum and the transverse ligament passing over the acetabular notch. All of the anterior surface and the medial half of the posterior surface of the femoral neck are intracapsular. The articular capsule is strong and thick over the upper and ante-

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**Osteoporosis box 5**

**Case note: Preoperative management of hip fracture**

After admission to hospital, Mrs Jones is transferred to the Orthopaedic Ward, 24 of whose 30 beds are occupied by elderly subjects recovering from hip fractures. These fractures all occurred following a fall or trivial trauma. Like many older subjects, Mrs Jones is on a large number of drugs (polypharmacy) and her 10 current medications should be critically reviewed to determine if they are all necessary. Her sleeping tablets should be stopped, as these types of medications are commonly associated with falls. Since she also takes diuretics, postural hypotension should be checked for as a cause of her falls. To reduce the risk of morbidity or mortality, Mrs Jones must be assessed by her anaesthetist prior to surgery for co-morbid conditions, and her cardiopulmonary and fluid-electrolyte state should be evaluated. Fluid and electrolyte imbalance is common in an elderly patient taking diuretics and anti-inflammatory drugs and with poor nutrition. Any imbalance must be corrected before surgery.

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**Fig. 5.11**

Anterior view of bones of the hip joint showing three main sites of fracture: high in the femoral neck (subcapital), across the neck itself (cervical) or in the trochanteric region (pertrochanteric).
by other vessels via the femoral neck, these may be damaged in cervical fractures. If the blood supply via the ligamentum teres is insufficient, avascular necrosis of the femoral head may occur.

The iliotibial band is a portion of the fascia lata of the thigh that extends inferiorly from the sacrum, the iliac crest and the ischium, over the greater trochanter of the femur and lateral aspect of the thigh to insert into the lateral condyle of the femur and tibia.

Important structures around the hip joint

Ligaments (Fig. 5.13A)
The fibrous tissue of the articular capsule of any joint usually shows localised thickenings, which form the ligaments of the joint. There are a number of important ligaments around the hip joint. The iliofemoral ligament is the strongest of these. Crossing the front of the capsule, it extends from the ilium to the anterior portion of the base of the neck. Its lower portion divides into two bands, forming an inverted Y shape. It is relaxed in flexion and taut in extension of the thigh and prevents excessive extension of the hip. In the upright position, the iliofemoral ligament stabilises the hip by pulling the femoral head firmly into its socket.

The pubofemoral and ischiocapsular ligaments are weaker than the iliofemoral ligament but help reinforce the posterior portion of the capsule. The ligamentum teres is an intracapsular ligament that loosely attaches the femoral head to the lower portion of the acetabulum and adjacent ligaments. It has little effect on the normal motion or stability of the joint but is a channel for blood vessels to the head of the femur. Although the femoral head’s blood supply is mainly

Fig. 5.12
Lateral view of the right hip of a child showing the three bones that form the acetabulum before fusion occurs.

Fig. 5.13
Anterior view of the right hip joint showing: (A) the main ligaments; (B) the key bursae.
Surgical management of hip fractures

Surgical management followed by early mobilisation is the treatment of choice for hip fractures. Surgical management varies according to the type of fracture and can be broadly divided into two types.

Femoral neck fractures

Undisplaced femoral neck fractures are most commonly fixed with multiple parallel screws or pins. The treatment of displaced femoral neck fractures depends on the patient’s age and activity level: young active patients should undergo open reduction and internal fixation; older, less active patients are usually treated with hip replacement (hemiarthroplasty), either cemented or uncemented. The ultimate goal is to return patients to their pre-fracture level of function by rapid rehabilitation.

Trochanteric fractures

A sliding hip screw is the device most commonly used for fracture stabilisation in both undisplaced...
and displaced intertrochanteric fractures. Fracture stability is dependent on the status of the posteromedial cortex of the femur. The most important aspect of its insertion is secure placement in the femoral head. Although the sliding hip screw allows postoperative fracture impaction, it is essential to obtain an impacted reduction at the time of surgery. If there is a large posteromedial fragment, an attempt should be made to internally fix the fragment with a screw or wire.

Medical management of osteoporotic hip fracture

Hip fractures are particularly common problems in the very elderly. After initial surgical management, diagnostic approaches should be directed at a general medical assessment. Elderly patients often have significant unrecognised multisystem disease that may impair the rehabilitation process. Investigations to exclude different types of underlying metabolic bone
disease should include looking for the possibility of subclinical vitamin D deficiency as a contributing factor to falls (due to muscle weakness) and fracture (due to osteomalacia), especially in institutionalised patients.

Measurement of bone mineral density is the best method for confirming the diagnosis of osteoporosis and is commonly used for monitoring the response to therapy. Bone density is usually measured at two sites, most commonly the spine and hip, using the technique of dual energy X-ray absorptiometry. Bone density can also be assessed by quantitative CT scanning. The ability of bone density to predict fracture is at least as good as cholesterol level to predict heart disease and blood pressure to predict stroke. Bone biopsy may rarely be performed to exclude diseases like osteomalacia. Ultrasound measurements, usually of the heel (calcaneus), can also be used to provide an assessment of bone density and structure. Spinal X-rays may be appropriate to examine for vertebral wedge or compression fractures. These fractures may be associated with pain but often occur silently and result in height loss and increased kyphosis.

Treatment of osteoporosis is aimed at preventing further fractures. It is important to select treatment individually for each patient. Treatment with calcium, vitamin D metabolites, oestrogen, selective oestrogen-receptor modulators, bisphosphonates or calcitonin may be considered.

**Calcium**

Calcium is weakly antiresorptive (i.e. a weak inhibitor of bone resorption) and supplementation may reduce negative calcium balance and so reduce bone resorption, particularly in older age. Controlled trials have demonstrated calcium supplementation can prevent bone loss in postmenopausal women and this has been associated with a modest reduction in fracture risk in longer-term studies. There is also evidence that calcium supplementation augments the effect of oestrogen on bone density.

**Vitamin D**

Since a substantial proportion of institutionalised (or house-bound) elderly may be vitamin D deficient, vitamin D supplementation is recommended in institutionalised or house-bound elderly subjects. Active vitamin D metabolites may be appropriate in patients with known or presumed calcium malabsorption.

**Oestrogen and selective oestrogen-receptor modulators**

Oestrogen replacement therapy is the treatment of first choice in most perimenopausal women. Oestrogen reduces osteoclastogenesis by decreasing production of cytokines such as IL-1 and RANK (Fig. 5.4). Treatment should be given for at least 5 years. Compliance will be enhanced by explaining the risks and benefits, regular monitoring and using preparations that minimise side-effects like bleeding, such as continuous regimens of oestrogen and progestagen.

Controversy exists over whether there may be an increased risk of breast cancer with long-term oestrogen use. This has led to the development of selective oestrogen-receptor modulators, which act to decrease bone resorption, like oestrogen, whilst not stimulating the breast or uterus. Controlled clinical trials have shown modest increases in bone density and significant reductions in vertebral fractures.
**Bisphosphonates**

Bisphosphonates are an effective alternative to oestrogen therapy. Bisphosphonates are potent inhibitors of bone resorption, acting through the inhibition of osteoclast function (Fig. 5.4). Randomised controlled trials have shown that treatment with these agents can significantly increase bone density and reduce further fracture risk by about 50%. The benefits of oestrogen and bisphosphonates may not persist after treatment is stopped.

Exercise programmes are most useful in relation to preventing further falls, even though little effect on bone density may be achieved. Biochemical markers of bone turnover such as bone-specific alkaline phosphatase (a marker of bone formation) or urinary pyridinoline (a marker of bone resorption) may be used to monitor compliance with therapy.

A period of weeks to months in a specialist rehabilitation unit may be necessary to improve coordination and gradually strengthen muscle power. However, functional impairment in activities of daily living because of poor mobility will be present in many of these patients. For example, about 50% of hip fracture survivors are discharged to nursing homes. Rehabilitation will enable many patients to regain independence. Prior to discharge, a home visit by the occupational therapist may be necessary to ensure that the home environment is safe. In addition, a variety of aids may be recommended by the occupational therapist to promote independent living.

**Case note: Postoperative management of hip fracture**

A multidisciplinary team comprising her orthopaedic surgeon, a physician, a physiotherapist, an occupational therapist and a social worker were all involved in Mrs Jones’ postoperative progress. The main goals were re-establishing her independence and early mobilisation to avoid pressure sores and thromboembolism.

As falls were involved in Mrs Jones’ case, a general medical assessment that included her visual function, and neuromuscular and cardiovascular systems was made. Calcium and vitamin D supplements were added to her treatment and she was transferred to a rehabilitation unit on day 8 after her surgery.

**Self-assessment case study**

A 65-year-old woman complains of acute thoracic back pain for 2 weeks. She recalls opening a window at the time of its onset but gives no history of trauma. X-rays reveal a crush fracture of the 10th thoracic vertebra. She describes no fever or weight loss but has been taking low-dose inhaled corticosteroids for asthma. Her menopause was at age 45 but she has never taken hormone replacement therapy because there is a family history of breast cancer.

After studying this chapter you should be able to answer the following questions:

1. What are the two different types of bone and their differences?
2. What are the common clinical risk factors for osteoporotic fracture?
3. What is the appropriate treatment in this patient?

*Answers see page 183*

**Self-assessment questions**

1. What are the two different types of bone and their differences?
2. What are the major hormones regulating calcium metabolism?
3. What is the normal bone remodelling sequence?

*Answers see page 184*

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**Further reading**

Moore K L, Dalley A F 1999 Clinically oriented anatomy, 4th edn. Williams & Wilkins, Baltimore

Favus M J (ed) 1999 Primer on the metabolic bone diseases and disorders of mineral metabolism, 4th edn. Lippincott Williams & Wilkins, Philadelphia