BASIC FACTS IN ORTHOPAEDICS

PATRICK S. H. BROWNE

BASIC FACTS IN ORTHOPAEDICS

PATRICK S. H. BROWNE
MA, BM, BCh, FRCS
Senior Lecturer in Orthopaedics
University of Tasmania

BLACKWELL
SCIENTIFIC PUBLICATIONS
OXFORD LONDON EDINBURGH
BOSTON MELBOURNE

Blackwell Scientific Publications
Editorial offices:
Osney Mead, Oxford, OX2 0EL
8 John Street, London, WC1N 2ES
9 Forrest Road, Edinburgh,
EH1 2QH
52 Beacon Street, Boston
Massachusetts 02108, USA
214 Berkeley Street, Carlton
Victoria 3053, Australia

First published 1981

Typeset by Scottish Studios and Engravers Ltd, Glasgow.
Printed and bound in Great Britain by Billing & Sons Ltd.,
Guildford, Worcester and London

DISTRIBUTORS

USA
Blackwell Mosby Book
Distributors, 11830 Westline
Industrial Drive, St Louis,
Missouri 63141

Canada
Blackwell Mosby Book
Distributors, 120 Melford Drive,
Scarborough, Ontario,
M1B 2X4

Australia
Blackwell Scientific Book
Distributors, 214 Berkeley
Street, Carlton, Victoria 3053

British Library
Cataloguing in Publication Data
Browne, Patrick S. H.
Basic facts in orthopaedics
1. Orthopedia
I. Title
617'.3 RD731
ISBN 0-632-00718-4

Preface

Medical students are taught many things. So much is taught that it is almost beyond the capacity of the average student to learn it all. More intelligent and more intellectual students are being recruited.

The average student has the capacity to become the average doctor—whose qualities of patience, integrity and good-humoured durability appeal to the average patient. The more intelligent and more intellectual doctor may not have these qualities.

The basic facts of orthopaedics are explicable to the average student—the minutiae of orthopaedic practice may not be. There are differences of opinion even among orthopaedic surgeons concerning the minutiae.

This book is written for the average medical student in the hope that it will help him in his struggle to become the average doctor.

I thank all those who have helped with this book. In particular I thank Mrs Hilary Goldsmid for the excellence of her illustrations.

P.S.H.B.

Contents

CHAPTER 1

Adult Bone

Bone is composed of an organic matrix known as osteoid. This consists of collagen fibres embedded in a cementing gel of protein polysaccharide. A mineral known as apatite consisting of calcium and phosphate is deposited on the collagen fibres as needle-shaped crystals.

In adult bone the collagen fibres are aligned to parallel the average compression and tension stresses to which the bone is subject. The apatite crystals are similarly orientated on the collagen fibres.

The strength of bones is dependent both on the normal formation of osteoid and mineral and on this alignment parallel to the average stresses to which the bone is subject.

Long bones in adults consist of tubes of cortical bone. The hollow centre contains marrow and occasional trabeculae of cancellous bone. The ends of bone are expanded towards the articular surface. This expansion is the metaphysis – here the cortex is thinned but arcades of cancellous bone supporting the articular surface are more pronounced (Fig. 1.1). These arcades also parallel the average stress to which the ends of the bone is subject and are aligned to transmit these stresses to the diaphysis (Fig. 1.2).

The shaft of a long bone is ensheathed in a layer of periosteum. The outer portion of this layer is fibrous tissue; the inner portion (cambium) contains primitive mesenchymal cells. The lining of the marrow cavity is known as endosteum and also is a source of primitive mesenchymal cells.

Pluripotent mesenchymal cells are present in the periosteum and the endosteum and also the trabeculae. They can develop into:

1. Osteoblasts which lay down new bone.

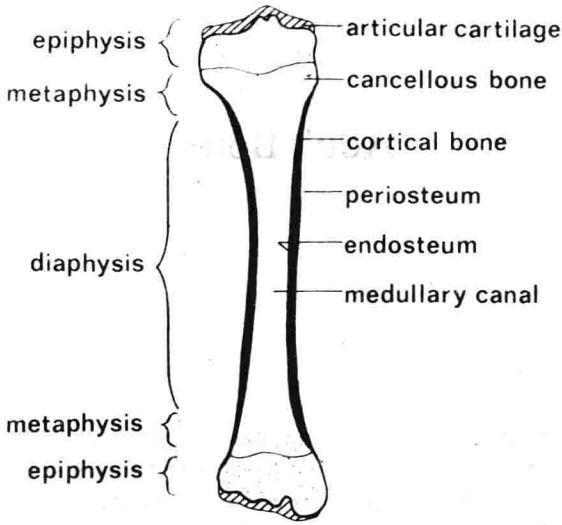

Fig. 1.1 Parts of bone.

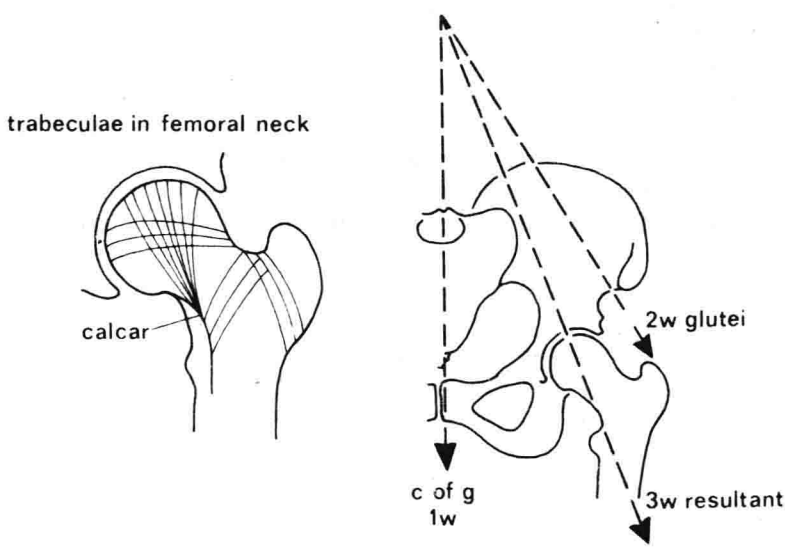

Fig. 1.2 Trabeculae aligned along the lines of stress; w, body weight.

2. Osteoclasts which reabsorb bone.

This activity is stimulated by trauma (fracture) or by infection or tumours which tend to displace the periosteum. These lesions cause a periosteal reaction which are visible on radiographs as new bone.

The cortex of a long bone is made of well-organised compact bone. This bone is organised in a series of Haversian systems (each an osteon) based on a central blood vessel. Osteocytes are embedded in the bone surrounding the blood vessel and are joined to it by minute canaliculi. The osteocytes maintain bone and are associated with its biochemical turnover. The alignment of the lamellae of each osteon is slightly different from its neighbour. This varied alignment adds strength to the bone, particularly as regards tangential stresses (Fig. 1.3).

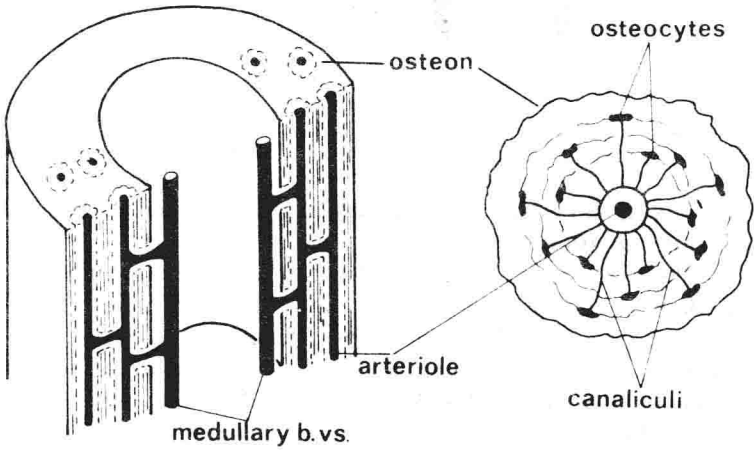

Fig. 1.3 Haversian system.

Cancellous bone is present towards the ends of long bones. The trabeculae are arranged adjacent to blood vessels; they are thinner and less complex than the lamellae of the cortex. The trabeculae are arranged to parallel the average compression and tension stresses to which the ends of bone are subject (Fig. 1.4).

Any lesion which interferes with the normal arrangement of cortical or cancellous bone will tend to weaken it. An example of such a lesion is Paget's disease in which there is no deficiency in the amount of bone. However the internal architecture of bone is

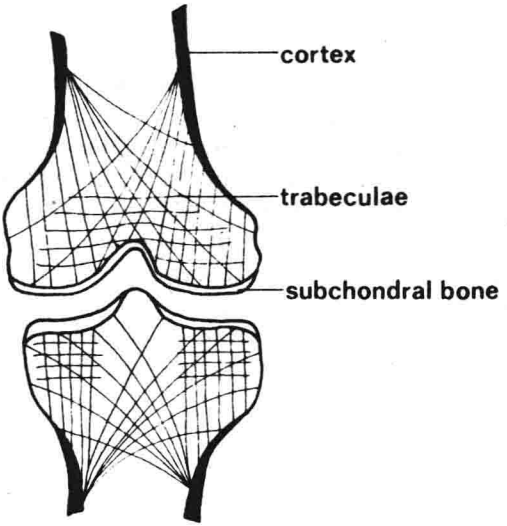

Fig. 1.4 Bone architecture.

disorganised and weakened, so that the long bones become bowed and liable to pathological fracture.

In all people, there is a continuous turnover of bone known as remodelling. It is much more active and rapid in children. Each individual osteon can be removed by osteoclasts and replaced by osteoblasts. This replacement can occur in a slightly different location in response to altered stresses. In old people, and in areas relieved of usual stresses, this replacement process lags behind the remodelling process and osteoporosis results.

The process of remodelling:

1. Permits a micro repair mechanism for wear and tear of minor trauma. It acts as a built-in protection against 'fatigue' (in the metallurgical sense).

2. It permits realignment of lamellae in response to changes of loads.

The blood supply of an adult long bone is:

1. From a central nutrient artery which supplies the marrow endosteum and the inner two-thirds of the cortex of the diaphysis.

2. Vessels from the periosteum which supply the outer one-third of the cortex.

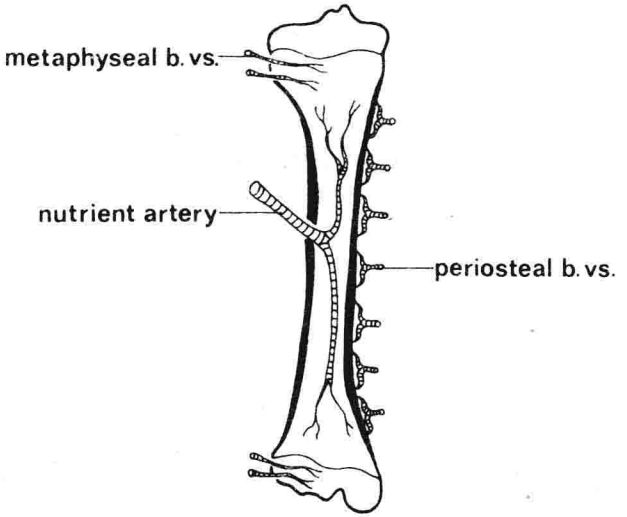

Fig. 1.5 Blood supply to a long bone.

3. Various vessels in the metaphyseal region (Fig. 1.5).

Lesions which disrupt the blood supply to a bone will cause that part of the bone to die.

CHAPTER 2

Calcium Haemostasis

It is necessary to maintain the serum calcium ion concentration at about 10mg per cent (4.7 – 5.2 m/cg/litre) as it is involved in numerous vital functions. Bone forms a reservoir of calcium and phosphate. There is a rapid turnover between bone and the extracellular fluid in order to maintain the serum calcium. Various factors are involved in this homoestatic process.

Parathormone secretion is stimulated by lowering of the serum calcium concentration and inhibited by an increase.

1. It has the function of promoting bone reabsorption and thus raising the serum calcium.
2. It also increases the excretion of phosphate by the kidney.

Vitamin D also affects the homeostasis of calcium.

1. It promotes the absorption of calcium from the intestine.
2. It promotes the activity of parathormone on bone.
3. It promotes bone accretion in patients with Vitamin D deficiency.

Calcitonin from the thyroid lowers serum calcium ion concentration by inhibiting bone reabsorption.

The level of serum calcium is closely linked with that of phosphate. It depends on absorption from the intestine and on reabsorption from the kidney tubules. Phosphate is also excreted and reabsorbed from the kidney tubules. The level of serum calcium may be affected by alimentary and kidney lesions. These lesions will have the greatest effect in children in whom the utilisation and turnover of calcium and phosphate is so much greater.

CHAPTER 3

Epiphyseal Growth Plate

Longitudinal growth of bone in children takes place at the epiphyseal growth plate, which is a plate of cartilage between the bone of the epiphysis and that of the metaphysis in a long bone. Several zones of the plate are described (Fig. 3.1).

1. The zone of germinal and of proliferating cartilage adjacent to the epiphysis. New cartilage cells are developed here and form cartilage matrix.
2. The zone of maturing cartilage where the cells form palisades and hypertrophy.

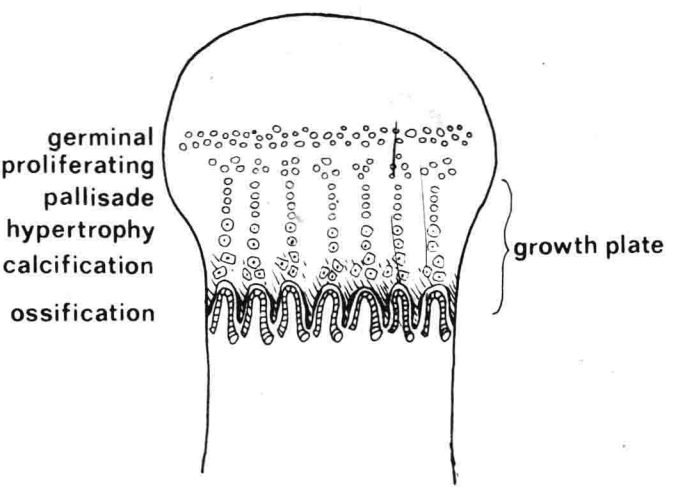

Fig. 3.1 Growth plate.

3. The zone of calcifying cartilage. The matrix becomes impregnated with apatite and the cells degenerate and die.

4. The zone of osteogenesis. The degenerating and dead cells are reabsorbed and are replaced by osteoblasts which secrete osteoid. The osteoid is rapidly mineralised with apatite and primitive bone results.

5. The zone of remodelling. Some of the new bone is reabsorbed as the shaft narrows and more regularly arranged lamellar bone is formed.

The growth plate has blood supply (Fig. 3.2):

1. From vessels to the epiphysis which reaches the germinal zone of cartilage.

2. From vessels to the metaphysis which reaches the area of degenerating cartilage.

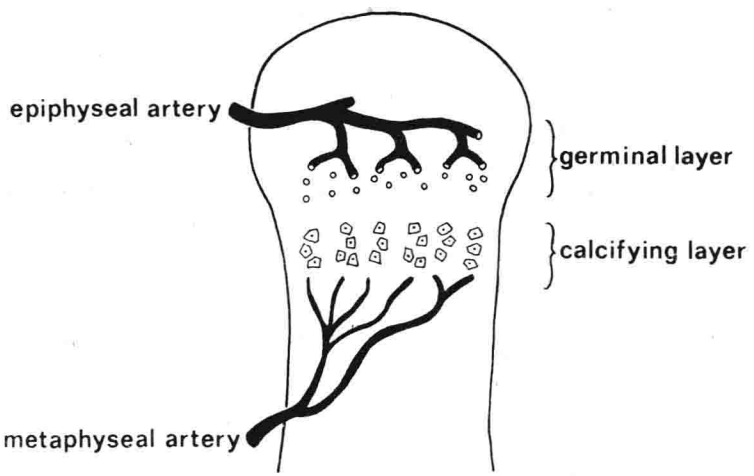

Fig. 3.2 Blood supply to growth plate.

The rest of the growth plate can only receive nutrients by diffusion from these two groups of vessels. Interference with the blood supply damages the growth plate.

The girth of bones depends on appositional growth from the periosteum. Small bones (and the epiphyses of long bones) grow from a spherical growth plate which surrounds the ossific nucleus (in the epiphyses this becomes hemispherical). The shape of the epiphyses and of small bones depends very much on neighbouring

contiguous structures. If the anatomy is distorted (as in club foot) the eventual shape of the bone will be distorted too.

The shape of the bone is also dependent on the stresses to which it is subject during growth. The upper end of the femur is subject to traction from its various muscle attachments. In particular, the abductor muscles (gluteus medius and minimus) and the psoas and adductors are normally in balance. In certain conditions such as meningomyelocele, cerebral palsy and poliomyelitis, the pull of the abductor muscles is relatively weak. As a result, the epiphysis of the greater trochanter does not develop fully and growth of the upper part of the femoral neck is reduced. Coxa valga results (Fig. 3.3).

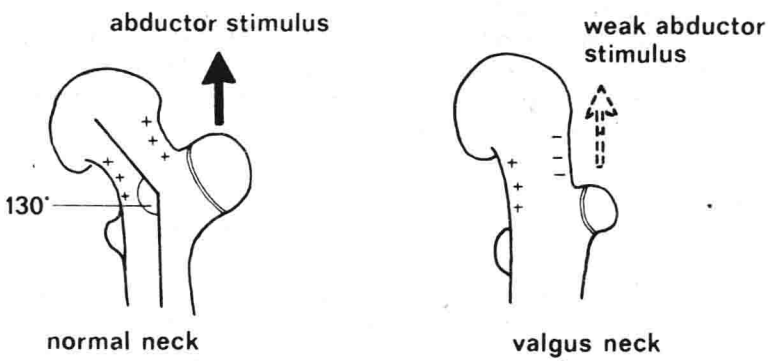

Fig. 3.3 Coxa valga.

The growth plate is affected by:

1. Nutritional factors.
2. Biochemical factors.
3. Hormones.
4. Blood supply.

Nutritional factors

Any generalised nutritional deficiency will be apparent at the growth plate as it is an area of high metabolic activity. Vitamin C is important in collagen synthesis; infantile scurvy is reflected in defects at the epiphyseal growth plate.

Biochemical factors

Calcium and phosphate are deposited as hydroxyapatite in the zone of calcifying cartilage and the zone of osteogenesis in the growth

plate. The condition known as rickets develops from defects of calcium and phosphate metabolism, in the growing child. The formation of cartilage matrix involves the production of chondroitin. Defects in mucopolysaccharide metabolism are reflected in dysfunction of the growth plate and is seen in the group of conditions known as mucopolysaccharidoses.

Hormones

Growth hormone promotes the activity of the growth plate cells; both cartilage formation and bone formation are increased. Excessive activity produces giants; deficiency produces dwarfs. Testosterone and oestrogen will stimulate the growth plate cells and then effect early closure.

Corticosteroids inhibit all cellular activity in the growth plate and will cause stunted growth if used to treat children. Thyroid hormone stimulates the growth plate. Cretinism is characterised by stunted growth and wide epiphyseal plates.

Blood supply

The blood supply is critical to growth plate function. If the epiphyseal vessels are interrupted the germinal layer of chondrocytes will die and growth will cease. If the metaphyseal vessels are interruped the effects vary depending on the severity and length of time of the interruption. Trauma or infection can damage the blood supply to the growth plate.

Generalised interference of growth plate function can produce:

1. Dwarfism.
2. Altered proportions of limbs to trunk.
3. Multiple deformities.
4. Early onset of degenerative joint disease.
5. Associated systemic manifestations.

Interference of function at one specific growth plate produces:

1. Limb length discrepancy.
2. Deformity.
3. Early onset of degenerative change in the adjacent joint.

CHAPTER 4

Leg Length Discrepancy

Many people have a discrepancy in leg lengths of about one centimetre. They can accommodate themselves to this discrepancy with no symptoms and no outward physical signs. Such people require no physical treatment.

However, leg length discrepancy can be severe and cause a limp and symptoms in the lumbar spine due to mechanical derangement. In some patients with congenital abnormalities the limbs may be so short that they will require an extension prosthesis.

CAUSES OF LEG DISCREPANCY

Congenital

There are many causes of congenital leg length discrepancy. Many of these can be classified as dysmelias (which means a failure in development of the limb). An amelia is congenital absence of the limb.

A phocomelia occurs in patients who have a flipper limb, the hand or foot extremity being attached directly onto the axial skeleton. These deformities occurred as a result of the thalidomide tragedy.

A proximal focal femoral defect occurs in various grades of severity. In this condition, the femur is deficient proximally and in the most severe form, only the distal end of the femur is apparently attached to the pelvis.

Infections

Osteomyelitis or septic arthritis in infancy may damage the epiphyseal growth plate and cause incomplete growth. Over the

years, a very significant growth disturbance takes place, and the limb may be very short (particularly if the epiphysis at the lower end of the femur or the upper end of the tibia is involved).

Osteomyelitis in an older child will probably not effect the growth plate. However the blood supply to the growth plate concerned may be actually increased as a result of the infection and this may cause a stimulus of growth. Such children may in fact end up with a slightly longer limb than normal. This increase of length does not usually require treatment.

Trauma

The long bones in children unite readily. If the fracture is reduced end to end the child may finish with the limb slightly longer than it was before. The fracture of the shaft of the femur in a child reduced perfectly may result in the limb being as much as 1.5 cm longer after a year or eighteen months. If malunion is allowed to occur, however, there may well be significant shortening.

Certain fractures will damage the epiphyseal plate. Particularly important are fractures involving the lower end of the femur or the upper end of the tibia. After such fractures in the young child, a very significant shortening can develop on reaching maturity.

Neurological lesions

A paralysed limb will usually be a short limb. Leg length discrepancy occurs particularly in childen with poliomyelitis and the affected limb may be very much shorter than the other. Paraplegic children with meningomyelocele develop very short paralysed limbs. Even children with cerebral palsy are found to have the most affected limb shorter than the other.

Other causes

Arteriovenous fistulae tend to cause increase in leg length. Patients with haemangiomata may have the affected limb longer than normal or shorter than normal. Patients with neurofibromatosis may develop a gigantism of one limb or a portion of a limb.

TREATMENT OF LEG LENGTH DISCREPANCY

Less than one centimetre

Patients with such a discrepancy usually require no treatment and will not have any significant symptoms from the discrepancy.