A SYSTEM OF ORTHOPAEDICS AND FRACTURES

A. Graham Apley

Fourth Edition

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FOURTH EDITION

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PREFACE TO THE FOURTH EDITION

After reprinting the third (1968) edition in 1970 and again in 1972, a fourth could no longer be postponed. This attempts to mirror the important changes in orthopaedics during the last few years. Some subjects, for example spina bifida, bone dystrophies and dysplasias, congenital hip subluxation and Perthes' disease, have been largely rewritten and up-dated. A few new sections have been added dealing with topics such as total hip replacement and its complications, Silastic implants and joint hypermobility. The principles of fracture management have scarcely altered, but changes in detail of technique or in emphasis have been incorporated and every page has been revised. Some old or obsolescent topics which have lost their importance have now been discarded, while others such as tuberculosis and poliomyelitis have been deliberately cut down in size. So the present edition can still be carried around, if not in a white coat pocket, then at least on a robust ward trolley.

The book has indeed emerged just a trifle slimmer than its predecessor, even though I have added some new pictures. In fact the same triple process of revision, addition and pruning was applied to the illustrations as to the text. Of the original 300-odd composites nearly a quarter have been changed; some are quite new, some have been considerably modified and others only slightly. What I have not changed is the basic idea—which seems to me important for teaching purposes—of grouping individual illustrations to enhance their meaning; rather have I tried to extend the technique still further (there are now 1,841 illustrations arranged as 347 composites). As before, each composite aims to paint the picture of a disease process, to display its variants, or to compare different diagnoses and differing treatments. They continue, I hope, to be self-explanatory and self-contained for quick reference and painless revision.

PYRFORD
July, 1973

A. GRAHAM APLEY

PREFACE TO THE THIRD EDITION

I first wrote the outline of this book in 1954. The F.R.C.S. course at Pyrford was then six years old; but as it became more comprehensive the students could either pay attention or scribble notes—they couldn't do both. The only answer was to give them summaries of all the lectures. These were revised and re-typed annually, but as the course grew longer typed notes became unwieldy (and secretaries rebellious) so in 1959 the publishers had to take over.

For the printed version I tried to convert the notes into more readable English, but decided to stick to the original systematic approach. Students seemed to like the idea of a standard pattern of headings for orthopaedics and fractures alike, and welcomed the logic of a constant sequence for describing physical signs; learning to *look*, *feel* and *move* before turning to x-rays is a habit they can profitably carry over from the lecture room (via the examination hall) to the consulting room.

Orthopaedics is so full of exciting developments that new editions are needed fairly frequently, but not every advance is of lasting value, and there is always dead wood that needs cutting out; by a combination of selection and excision I have tried, in successive re-writings, to avoid the increase of bulk which usually goes with advancing age. This present edition is much bigger—not because the text is longer, but because, for the first time, illustrations are included.

Originally I meant to publish a separate pictorial atlas as a companion volume. But does anybody read an atlas? Why not incorporate pictures with text? The difficulty was that I wanted so many. So I made a selection from the illustrations collected during the twenty years of the Pyrford course. I then set about pruning them and combining them into 'composites', each of which would tell its own story. This idea fitted in well with something every teacher knows: that, no matter how good a single illustration may be, it is more effective for teaching when combined with others in meaningful groups. Composites are the natural way of showing stages in a process, of contrasting differing methods of treatment, of highlighting important physical signs, and of summing up differential diagnosis. In all I have grouped 1,802 separate illustrations into 312 composites. The illustrations can be used on their own for quick revision; together with the text I hope they provide a substantial yet concise presentation of orthopaedics and fractures in a single volume.

PYRFORD
January, 1968

A. GRAHAM APLEY

ACKNOWLEDGMENTS

I owe and gratefully acknowledge an immeasurable debt to my teacher George Perkins, whose influence has, I hope, pervaded both my work and my teaching. He has also generously allowed me to use a number of the illustrations from his classic book on orthopaedics; Athlone Press, his publishers, have kindly added their permission.

Among colleagues and friends who have given ungrudging help three must be mentioned especially and I list them alphabetically. Gordon Hadfield who, for the first and second editions, carefully read and criticized the text has, for the third and fourth, devoted much time and talent to helping with the illustrations. Basil Helal's erudition has been a tower of strength, especially with regard to surgery of the hand. Liam Murphy, who originally helped so much with proof-reading and composing legends for illustrations, has this time concentrated his considerable efforts on those sections which have been largely re-written; I hope his delicately witty touch is reflected in the text. George Raine has been meticulous in perusing every word of the text, in proof-reading, in checking references and, in general, being tirelessly helpful.

The difficult and exacting requirements of photography have been carried out with superb skill by Ken Fensom at Pyrford and Mrs. Barry at St. Peter's Hospital. For the new diagrams I am indebted to David Seaton who also prepared, trimmed and assembled the new composites. The publishers have yet again proved unfailingly co-operative.

My colleagues at Pyrford, R. J. Furlong, F. A. Simmonds and G. Hadfield have kindly allowed me to include illustrations of some of their patients. I am grateful also to those from other hospitals who so willingly loaned x-rays or photographs to fill some inevitable gaps. They include the following, but I apologize in advance if, inadvertently, the name of some generous contributor has been omitted.

Chase Farm Hospital, Enfield and Highlands Hospital, Winchmore Hill. Orthopaedic department **Mr. B. Helal**; X-ray departments **Dr. K. Lavers, Dr. L. Pell.**—Figs. 3.1 (a), 3.7 (a, e), 5.4 (a, b), 6.8 (c, d), 6.10 (a), 6.11 (g), 7.1 (a), 7.4 (d), 15.3 (e), 18.5 (b), 20.16 (d), 21.11 (f), 21.15 (a), 24.10 (a, b), 25.24 (c).

Einstein Medical College, New York. **Prof. A. J. Helfet.**—Fig. 25.18 (a, b).

King's College Hospital, London. **Mr. H. L.-C. Woods, Mr. R. C. F. Catterall, Mr R. Q. Crellin.**—Figs. 6.6 (a, b), 23.21 (g)

London Hospital Medical College. **Dr. R. M. Mason.**—Fig. 2.6 (b).

Norfolk and Norwich Hospital. **Mr. R. C. Howard.**—Figs. 11.2 (d), 18.30 (a).

Princess Margaret Rose Hospital, Edinburgh. **Mr. G. E. Fulford.**—Fig. 6.4 (a).

Queen Mary's Hospital for Children, Carshalton. **Mr. T. L. Bowen.**—Fig. 1.7.

Redhill Group of Hospitals. **Mr. P. A. Ring.**—Fig. 18.31 (b).

Royal Adelaide Hospital, S. Australia. **Mr. G. A. Jose.**—Fig. 6.10 (b, c).
Royal Portsmouth Hospital. **Mr. R. A. Denham.**—Figs. 1.5 (d), 6.7, 6.16 (b, c), 7.4 (a, b, c, d), 8.8 (a), 12.2 (b).
Royal Hospital for Sick Children, Aberdeen. **Dr. A. M. Stewart.**—Fig. 8.8 (b, c).
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Royal Victoria Infirmary, Newcastle-upon-Tyne. **Mr. J. K. Stanger.**—Fig. 8.3 (a).
St. Thomas' Hospital, London. Orthopaedic department **Mr. R. J. Furlong, Mr. D. R. Urquhart;** Physical Medicine department **Dr. D. A. H. Yates;** X-ray department **Dr. M. Lea-Thomas;** Photographic department **Mr. G. Brandon.**—Figs. 1.4 (e, f), 3.2 (d), 3.7 (f), 6.8 (e, f), 8.3 (d), 8.4 (a), 17.20 (d, e), 19.18 (e, f), 21.9 (c, d), 21.17 (d), 21.19 (c), 23.3 (c), 25.9 (e), 25.31 (h).
United Bristol Hospitals. **Dr. R. S. Gordon.**—Figs. 2.1 (a, b, c), 2.2, 6.4 (c), 7.1 (c), 7.3 (c), 21.25 (f, g, h).
Westminster Hospital, London. **Mr. H. E. Harding, Mr. D. L. Evans.**—Fig. 25.2 (c).
Winchester Group of Hospitals. **Prof. J. S. Ellis.**—Fig. 23.27 (e).
Wrightington Centre for Hip Surgery, Wigan. **Prof. J. Charnley.**—Figs. 11.2 (c), 18.31 (d).

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Fig. 1.8: Miss R. Wynne-Davies, *Journal of Bone & Joint Surgery*, **52B**, 704.
Fig. 9.8: G. W. N. Eggers, *Journal of Bone & Joint Surgery*, **34A**, 4, 827.
Fig. 15.8: D. A. Bailey, *The Infected Hand.* London; H. K. Lewis.
Fig. 15.9: R. J. Furlong, *Injuries of the Hand.* London; Churchill.
Fig. 21.17 (d): N. L. Browse, M. Lea-Thomas and M. J. Solan; *British Medical Journal* (1967) **4**, 596.
Figs. 24.1–24.12 have also appeared in the *Annals of the Royal College of Surgeons*, **46**, 210.

CONTENTS

FRACTURES AND DISLOCATIONS

CHAPTER 1

DIAGNOSIS IN ORTHOPAEDICS

An orthopaedic disorder does not exist in isolation. It is part of a patient who has a personality, a mind and a body; a job and hobbies; a family and a home. Any of these factors may have an important bearing upon the disorder and its treatment. They will not be considered at length, but are stressed here as they should be at the beginning of any clinical examination. It would also be out of place to discuss in detail the symptoms and signs of general illness in patients with orthopaedic disorders. Their importance is obvious, and in subsequent chapters they take pride of place before the symptoms and signs of local disorder.

Orthopaedics is concerned with disorders of bones, joints, muscles, tendons and nerves. The field is wide, yet limited. When a diagnosis appears elusive it is sometimes helpful to review the pathological entities likely to be encountered. They fall into easily remembered pairs: injury and inflammation; tumour and degeneration; muscle weakness and mechanical derangement; congenital deformity and acquired dystrophy.

LOCAL SYMPTOMS

A thorough history demands patience. Unless the doctor allows the patient to tell his story more or less in his own way, important facts may be missed and the patient may feel justifiably aggrieved.

The common symptoms in orthopaedics fall into three groups. The patient may complain that something *looks* wrong (deformity, shortening, swelling or a lump); that something *feels* wrong (pain, tingling or numbness); or that movement is wrong (limp, weakness, flailness, stiffness or mechanical derangement). Pain, local or referred, is the most common and important symptom.

Although the patient must be allowed to tell his own story, he needs guidance. Of any particular symptom it may be necessary to enquire if the onset was sudden or gradual, or preceded by injury or illness; if it is constant or intermittent, static or increasing, and whether anything makes it better or worse; finally, the occupation and any previous illness or injury may be important.

LOCAL SIGNS

For examination, a patient must be suitably undressed; no mere rolling up of a trouser leg is sufficient. Where one limb is to be examined, the opposite one must be adequately exposed, so that the two may be compared.

EXAMINATION OF A JOINT

GENERAL FEATURES		A brisk general appraisal of the patient is imperative
LOCAL SYMPTOMS		Let the patient tell his story, encouraged by an occasional judicious question
LOCAL SIGNS		A system is the key to accurate diagnosis
Look	Skin Shape Position	At this stage *shortening* is assessed.
Feel	Skin Soft tissues Bones	
Move	Range Muscles Function	
X-ray		Plus other investigations

LOOK

The student, or inexperienced doctor, is inclined to rush in with his hands—a temptation which must be resisted. His motto should be 'look before you feel'. And in looking he must follow a purposeful orderly system; otherwise he will miss vital clues.

Skin — This naturally comes first. Colour changes, abnormal creases, and scars (operative or accidental) often point the way to diagnosis.

Shape — 'Shape' means what it says. A mis-shapen limb may be too fat (think of fluid or a lump), or too thin (think of wasting).

Position — While the position in which a joint is held may vary, if the joint is normal, it 'looks natural'; any deviation from this natural appearance demands investigation. In many joint disorders and in most nerve lesions the limb adopts a characteristic attitude.

FEEL

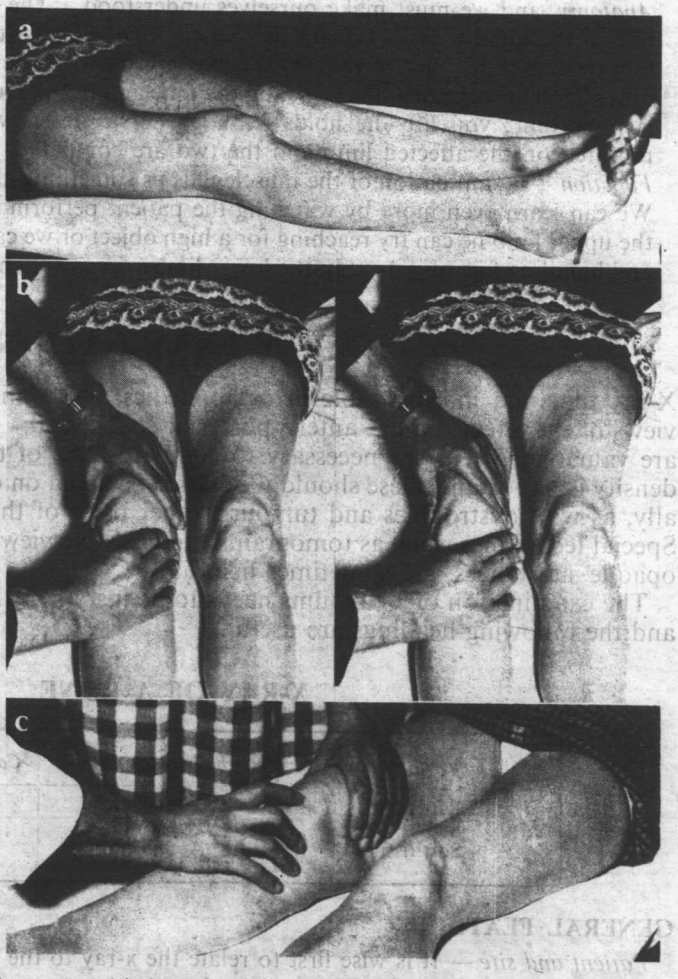
We must feel (as we should have looked) systematically: the good limb then the bad; and the skin before the deep tissues.

Skin — Is the skin warm or cold, moist or dry, rough or smooth? and—equally important—can the patient feel you touching him, or is sensation abnormal?

Soft tissues — Deep to the skin we may encounter tenderness, which is important—in two ways. First, we must avoid hurting the patient; and so we watch his face and not our hands while examining him. Secondly, tenderness is often sharply localized; if so we know immediately the precise anatomical site of the lesion.

With superficial joints we can also feel if the synovial membrane is thickened (by rolling its edge under the fingers) and we can detect excess fluid. Two of the methods of demonstrating fluid are shown in Fig. 1.1.

A soft tissue lump always demands careful examination to determine its size, shape, surface, consistency, edge and attachments.



1.1 FLUID IN THE KNEE
The suprapatellar pouch bulges, filling the hollows (a). It can be felt by cross-fluctuation (b), or by the patellar tap (c).

Bony lumps are discussed on page 14; but the entire bone should always be palpated for tenderness, abnormal thickening or any irregularity of surface.

MOVE

Range — Should we examine active movements, passive movements, or both? In the upper limb and spine we usually find ourselves examining active movements first; in the lower limb, passive. In fact we need to test both varieties only when muscle is torn,

paralysed, or painful; otherwise the two are the same. What matters more is to examine the good limb first, or both limbs simultaneously. We need to know if a particular movement is limited (and by how much), or painful (and at what angle); we must also be on the lookout for increased movement and for abnormal movements.

Muscles — Muscle testing is not as easy as it sounds; few patients have mastered *Gray's Anatomy*, and we must make ourselves understood. The easiest way is exemplified by testing the quadriceps. Lift up the patient's *good* leg (with his knee straight), and ask him to keep it up; then to resist you while you try with one hand to bend his knee—your other hand at the same time is feeling the tone and bulk of his quadriceps. The sequence is important: you lift—he holds—you push—he resists while you feel. This is then repeated on the affected limb and the two are compared.

Function — Examination of the muscle tells us something about the function of the limb. We can learn even more by watching the patient perform certain specific activities. In the upper limb he can try reaching for a high object or we can test him picking up weights and handling fine objects. In the lower limb we can watch him stand, walk, run or hop.

X-RAY EXAMINATION

X-ray films are indispensable in orthopaedics. The minimum requirement is two views of the affected area: antero-posterior and lateral. Occasionally oblique views are valuable. Often it is necessary to compare films of two limbs and, where bone density is important, these should if possible be taken on one x-ray plate. Occasionally, as with dystrophies and tumours, other parts of the body need to be x-rayed. Special techniques, such as tomograms, stereoscopic views and the injection of radio-opaque substances, are sometimes helpful.

The examination of x-ray films needs to be just as methodical as that of a patient, and the following headings are useful.

X-RAY OF A BONE

General features	*The bone as a whole*	*Components of the bone*
Patient	Shape	Periosteum
Site	Density	Cortex
Soft tissues	Architecture	Medulla

GENERAL FEATURES

Patient and site — It is wise first to relate the x-ray to the patient. Some disorders, for example, are age-specific: thus, solitary bone cysts are seen only before skeletal maturity, giant-cell tumours only after it.

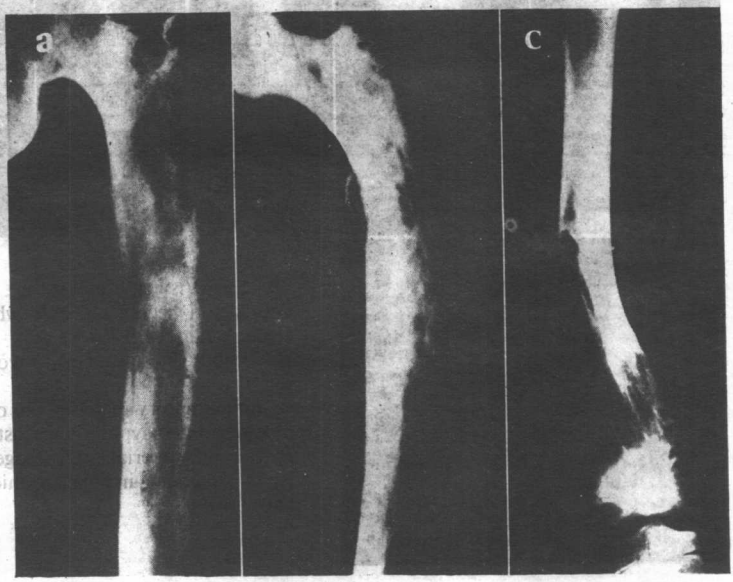
Soft tissues — These merit a separate heading, because they are otherwise liable to be forgotten. Metallic foreign bodies are always strikingly self evident, but even wood or glass may show in suitable films. Loose bodies in a joint are sometimes less obvious, but it is always worth trying to identify their source. Extra-osseous calcification may occur

in a haematoma (myositis ossificans), in a cold abscess, in a tendon (especially supraspinatus), in a tendon sheath (peritendinitis calcarea), and in veins (phleboliths). Occasionally a damaged ligament calcifies and huge masses of calcium may be seen round joints in the rare condition called tumoral calcinosis. If the soft tissues are unduly translucent, think of a lipoma.

THE BONE AS A WHOLE

Shape — This is clearly important. The bone may be too wide, as in Paget's disease; too narrow, as in osteogenesis imperfecta; or it may be bent (page 8).

Density — Bone density must be assessed with caution, for illusions can be created by variations in radiography. Generalized increase of density is seen in marble bones (page 74); generalized decrease in osteoporosis (page 93) and osteomalacia (page 90). More localized changes follow alterations in the amount of blood reaching the bone: thus avascular necrosis following injury or bone infection causes increased density ('dead bone is dense bone'); and rarefaction follows the increased vascularity of a joint chronically inflamed in tuberculosis or rheumatoid arthritis. 'Osteolysis' is the term used when bone disappears for no obvious reason.



1.2 BONE ARCHITECTURE (a) In chronic osteomyelitis the normal architecture is lost and the bone is thickened but straight. (b) Paget's disease looks somewhat similar except that the bone is bent. (c) In fibrous dysplasia the bone contains 'bubbles and stripes': it is bent but not thick.

Architecture — This term is difficult to define, but refers to the general structural appearance of the bone. The three examples in Fig. 1.2 are relatively common and important, although unimportant rarities such as osteopoikilosis and osteopathia striata (page 75) may be more striking.

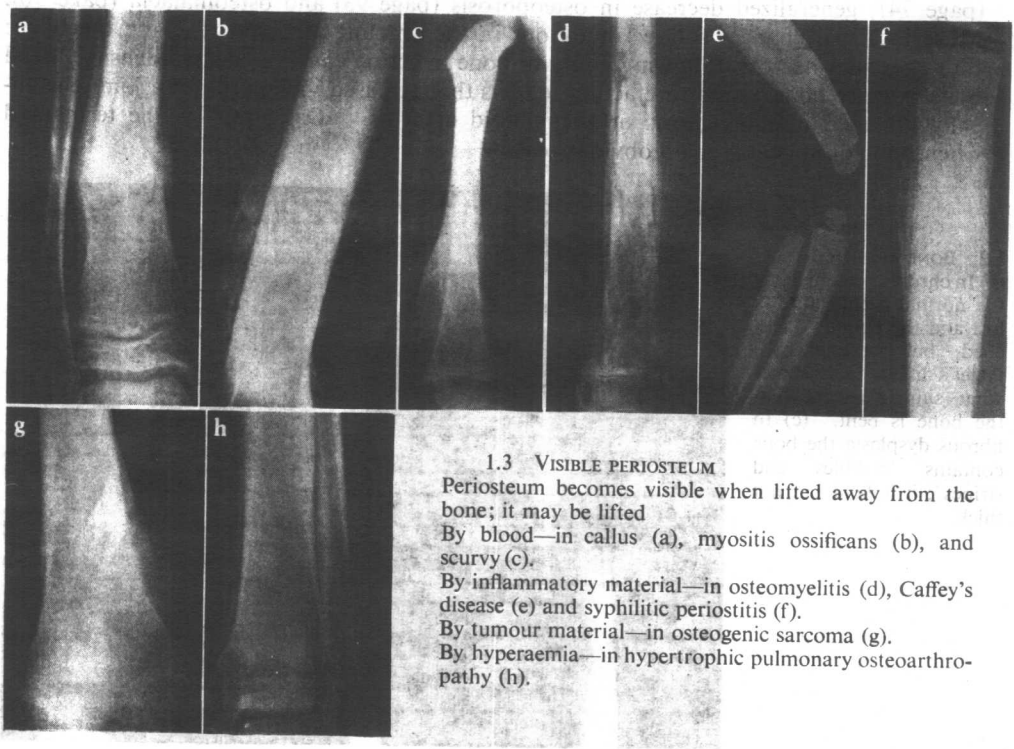
COMPONENTS OF THE BONE

Periosteum — Except in young infants periosteum is not visible on x-ray; but once it is

lifted away from the cortex of the bone calcification occurs (Fig. 1.3). Widespread periosteal changes are seen in syphilis and in Caffey's disease (page 18).

Cortex — The cortex of pipe bone should be uniform in thickness throughout the diaphysis but tapering at both ends. Any alteration is noteworthy. The cortex may look thinner because it has been partly eroded (e.g. by a cyst, tumour or aneurysm); or it may be thickened in conditions such as Paget's disease. Actual perforation of the cortex is more sinister, suggesting malignancy, although in syphilis the appearances can be strikingly similar ('syphilis can mimic anything').

Medulla — The medulla needs to be inspected purposefully for areas of increased or diminished density, which may be single or multiple.



1.3 VISIBLE PERIOSTEUM
Periosteum becomes visible when lifted away from the bone; it may be lifted
By blood—in callus (a), myositis ossificans (b), and scurvy (c).
By inflammatory material—in osteomyelitis (d), Caffey's disease (e) and syphilitic periostitis (f).
By tumour material—in osteogenic sarcoma (g).
By hyperaemia—in hypertrophic pulmonary osteoarthropathy (h).

A single rarefied area — This may be due to one of the following conditions:

Inflammation, for example a Brodie's abscess, which has a sclerosed margin and is often lobulated.

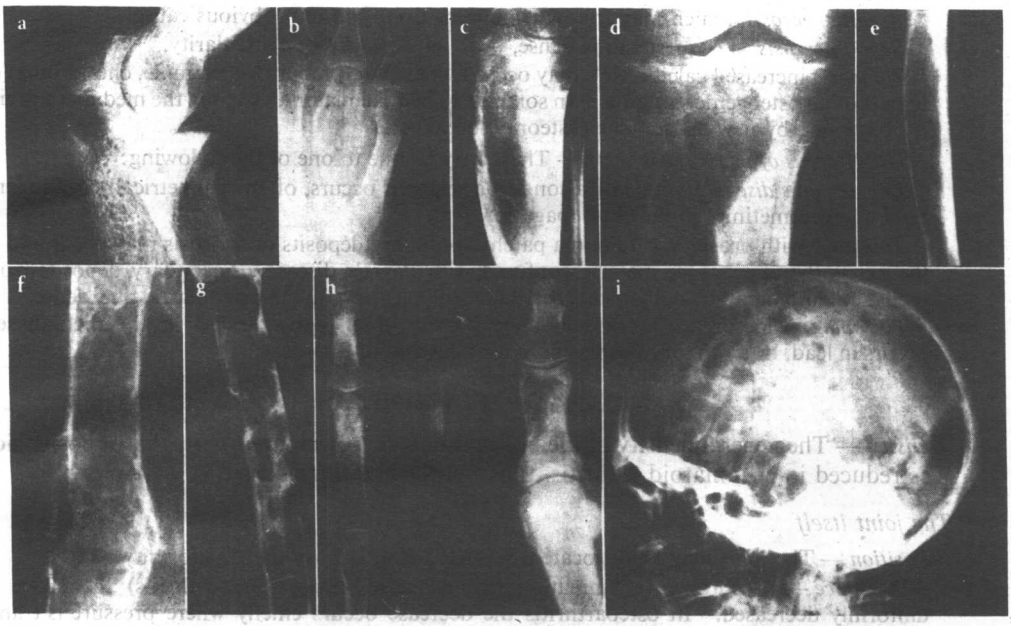
Solitary cyst, which has a well-defined but not sclerosed margin and is situated on the shaft side of an epiphyseal line.

Other cysts occur in association with osteoarthritic joints; they are usually small and situated in the subchondral area. Similar small cysts have been described in adults with no

osteoarthritis; from their contents these have been called 'bone ganglia'. In sarcoidosis also multiple bone cysts may occur, especially in the fingers.

Benign tumour: a chondroma usually shows specks of calcification and occurs in short bones; a giant-cell tumour is often trabeculated and at the very end of a long bone; both have a clearly defined edge.

Malignant tumour: an osteolytic sarcoma has no well-defined edge.



1.4 EXAMPLES OF RARE AREAS IN BONE
Single: Brodie's abscess (a), tuberculous dactylitis (b), solitary cyst (c), giant-cell tumour (d), eosinophilic granuloma (e).
Multiple: Hand-Schüller-Christian disease (f), hydatid disease (g), sarcoidosis in the hand and foot (h), secondary deposits (i).

Multiple rarefied areas — these may be due to one of the following:

Fibrous dysplasia: cysts occur in one or several bones (page 77).

Storage diseases: Gaucher's disease is a familial primary lipoidosis with reticulum cell hyperplasia; the spleen is huge and the liver often enlarged; the cells are also found in bone marrow where x-rays show osteolytic areas.

The secondary lipoidoses constitute a group of disorders which may be stages of a single disease process called histiocytic granulomatosis or histiocytosis 'X'. The group includes: (1) Hand-Schüller-Christian disease: there are multiple deposits in the bones, especially the skull, vertebrae and femora (showing as sharply defined translucent areas on x-ray), in the pituitary gland (causing diabetes insipidus), in the orbit (causing exophthalmos), in the skin and many other soft tissues. (2) Letterer-Siwe disease: this is probably an acute form of (1). It occurs in infants and is rapidly fatal. (3) Eosinophilic granuloma of bone: the deposits may

be single or multiple, but are not numerous and recovery is usual, with or without treatment. Collapse of a vertebra containing such a deposit is thought to be responsible for Calvé's disease.

Malignant disease: In leukaemia the bones may show ill-defined areas of rarefaction; there is also anaemia, enlargement of the spleen, liver and lymph nodes, and often haemorrhages in the skin or gut. In secondary carcinoma and myelomatosis multiple areas of bone rarefaction also occur (*see* Chapter 8).

A single area of increased density — This may be due to one of the following conditions.

Aseptic necrosis, which may follow trauma or occur without obvious cause.

Septic necrosis: a sequestrum is dense, probably because of avascularity.

Tumours: increased calcification may occur in a benign tumour (for example, chondroma) or in part of an osteogenic sarcoma. In some cases of anaemia or leukaemia the medullary cavity is obliterated by new dense bone (osteomyelosclerosis).

Multiple areas of increased density — These may be due to one of the following:

Engelmann's disease: in this rare condition sclerosis occurs, often symmetrically in the long bones, and sometimes in the skull (page 77).

Tumours: with prostatic carcinoma patchy secondary deposits of sclerosis may occur and in the pelvis the appearance may be confused with Paget's disease. Secondary deposits from breast carcinoma are usually osteolytic, but may occasionally be osteoblastic and dense.

Poisoning: widespread increase in bone density, often concentrated in the metaphyses, occurs in lead, bismuth, or phosphorus poisoning and in fluorosis.

X-RAY OF A JOINT

Density — The general density of the bones is noted. Compared with the opposite side it is reduced in rheumatoid arthritis, tuberculosis and after disuse.

The joint itself

Position — The joint may be dislocated, subluxed or in a position of deformity.

Joint space — In chronic inflammation (for example, rheumatoid arthritis) the space is uniformly decreased. In osteoarthritis the decrease occurs chiefly where pressure is transmitted, and there may be lipping or osteophytes at the edges.

The joint space is increased in some varieties of osteochondritis (for example, Perthes' disease) and occasionally when a joint is distended with fluid. Loose bodies may be visible within the joint space, and occasionally menisci are calcified.

Joint line — In chronic arthritis the articular surfaces are irregularly eroded. In osteochondritis dissecans a crater is seen on one convex surface.

The bones — The bones above and below the joint are systematically examined as already described.

SPECIAL PROBLEMS

In this section certain important clinical features are selected for further consideration.

BENT BONES

The long bones are straight or have slight natural curves. If a bone is abnormally bent it must have broken, or been soft at some time, or have grown faultily.