An Introduction to Clinical Rheumatology

Michael Mason
H L F Currey

Second Edition

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Preface

The encouraging reception accorded the first appearance of this book has stimulated us to produce a second edition, incorporating alterations helpfully suggested by both colleagues and reviewers. Almost all the latter regretted the absence of references and, although these were omitted from the first edition for what we thought were good reasons, they are now included. In doing this we have tried to provide a few key references selected because they offer a suitable starting point for pursuing a particular subject in greater depth. We have also borne in mind the desirability that these references should be accessible on the shelves of an undergraduate medical school library.

In order that the text should be kept consistent we have tried to maintain the policy of the first edition by inviting as new contributors only teachers associated with the London Hospital. Thus, Prof. Robert Cohen has written a new chapter on metabolic bone diseases, and Dr Stephen Wolkind, Senior Lecturer in Psychiatry, one on psychological aspects of rheumatism. We are fortunate also in having enlisted the services of Dr Barrie Vernon-Roberts who, as a morbid anatomist with special interest in the rheumatic diseases, has joined The London since the first edition was prepared. He has contributed a chapter dealing with the applied anatomy of joints and with some selected topics in the pathology of rheumatic diseases. This particular arrangement has inevitably led to a degree of overlap between this and some other chapters. However, it is hoped that the cross-references provided will allow readers of the clinical chapters to turn to this pathological section when morbid anatomical details are required. The topics covered in Dr Vernon-Roberts' chapter have been included with this in mind.

Experience with the first edition has encouraged two slight changes in editorial policy. Unexpectedly, more copies of the book were sold outside the British Isles than within, so we have omitted the more parochial material dealing with training and practice specifically in the United Kingdom. In addition, we have become aware that the book is probably

used as much by postgraduates as undergraduates, and we have kept these two groups equally in mind in considering content and style.

So that we did not need to enlarge the book beyond its original length, which seemed to us about right, we have omitted the chapter on Immunology, feeling that this is a subject which can barely be covered, as was attempted, in seven pages, and that the reader should rather turn to one of the excellent primers on this subject which have recently become available. Our continuing awareness of the overlap, interdependence and the need for co-operation with the specialty of Orthopaedic Surgery is reflected in the recruitment of a second orthopaedic surgeon, Mr Brian Roper, who has undertaken the chapter on joint infections.

We have re-examined the content of the book in the light of our desire to provide a balanced account of clinical rheumatology. Here we have had in mind criticisms that the proportionate page allocations did not reflect the relative numbers of patients presenting with the various disorders. Thus it has been suggested that more space should be devoted to conditions such as the painful shoulder and to low back pain. We are certainly well aware of the need to provide the trainee rheumatologist with a practical approach to the diagnosis, understanding and management of these important clinical problems, but the more one explores the literature on these subjects the more impressed does one become with the overall ignorance that exists about them. Much of what has been written about this group of disorders consists of uncritical observations and impressions, often contradictory, which, especially when coupled with dogmatic and empirical statements on treatment, do not stand up to close scrutiny. The very terminology is full of terms hallowed by clinical usage but (often for perfectly good reasons) unsupported by pathological observations. In these circumstances we have felt it right to give a relatively simple account of these particular conditions, pointing out the lack of information that exists, and we have tried to avoid burdening the reader with too many hypotheses and prejudices.

Finally we must record our grateful appreciation of the many kind people who so generously gave us their help when it was needed. In particular we want to thank Mr R. F. Ruddick and his colleagues in the Photographic Department of The London Hospital for preparing the illustrations, Dr John Mathews, Dr Alan Ridley and Dr Michael Swash for their ungrudging help and advice on parts of the text and Mr Stephen Neal of Pitman Medical for his courtesy and patience.

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May 1974

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Contents

	LIST OF CONTRIBUTORS	v
	PREFACE Michael Mason and H. L. F. Currey	vii
1	THE APPLIED ANATOMY OF JOINTS B. Vernon-Roberts	1
2	RHEUMATOID ARTHRITIS Colin G. Barnes and Michael Mason	24
3	JUVENILE CHRONIC POLYARTHRITIS (STILL'S DISEASE) Colin G. Barnes	55
4	'SERONEGATIVE' ARTHROPATHIES Michael Mason	63
	Ankylosing Spondylitis	64
	Psoriatic Arthritis	75
	Reiter's Disease	81
	Colitic Arthritis	91
5	ARTERITIS Colin G. Barnes	96
	Polyarteritis Nodosa	97
	Giant Cell Arteritis	102
	Allergic Vaculitis	105
	Takayasu's Arteritis	106
	Wegener's Granulomatosis	108
6	POLYMYALGIA RHEUMATICA Colin G. Barnes	110
7	PROGRESSIVE SYSTEMIC SCLEROSIS AND POLYMYOSITIS H. L. F. Currey	114
	Progressive Systemic Sclerosis	114
	Polymyositis	121

8 SYSTEMIC LUPUS ERYTHEMATOSUS 132
Graham R. V. Hughes

9 SJÖGREN'S SYNDROME 156
Colin G. Barnes

10 GOUT 162
H. L. F. Currey

11 PYROPHOSPHATE ARTHROPATHY (PSEUDOGOUT, CHONDROCALCINOSIS) 180
H. L. F. Currey

12 BACTERIAL INFECTIONS OF JOINTS 187
B. A. Roper

13 RHEUMATIC FEVER 196
A. D. M. Jackson

14 DEGENERATIVE JOINT DISEASE I: OSTEOARTHROSIS OF PERIPHERAL JOINTS 208
J. D. Jessop

15 DEGENERATIVE JOINT DISEASE II: SPONDYLOSIS AND DISC LESIONS 222
H. L. F. Currey

16 MEDICAL ORTHOPAEDICS 235
H. L. F. Currey
Shoulder Lesions – Tennis and Golfer's Elbow – De Quervain's Syndrome – Carpal Tunnel Syndrome – Trigger Finger – Dupuytren's Contracture – Chondromalacia Patellae – Painful Heel – Metatarsalgia – March Fracture – Virus Myalgia – Tietze's Syndrome

17 METABOLIC BONE DISEASE IN RHEUMATOLOGY 248
R. D. Cohen

18 LESS COMMON RHEUMATIC CONDITIONS 260
Colin G. Barnes
Acromegaly – Anaphylactoid Purpura – Amyloidosis – Aseptic Necrosis Atrial Myxoma – Behçhet's Syndrome – Calcific Periarthritis – Clutton's Joints – Erythema Nodosum – Familial Mediterranean Fever – Haemochromatosis – Haemoglobinopathies – Haemophilia – Hypercholesterolaemic Arthropathy – Hypermobility Syndromes – Hyperparathyroidism – Hypertrophic Pulmonary Osteoarthropathy – Hypogammaglobulinaemia – Hypothyroidism and Thyrotoxicosis – Jaccoud's Syndrome – Leukaemia – Long Leg Syndrome – Neuropathic Joints – Ochronosis – Osteochondritis Dissecans – Pigmented Villo-Nodular Synovitis – Relapsing Polychondritis – Renal Transplantation and Dialysis – Rubella – Sarcoidosis – Serum Sickness –

Synovial Chondromatosis – Synovial Rupture – Synovial Tumours – Whipple's Disease

19 PSYCHIATRIC AND PSYCHOLOGICAL ASPECTS OF RHEUMATIC DISEASES 281
S. N. Wolkind

20 OPERATIVE SURGERY IN THE RHEUMATIC DISEASES 292
M. A. R. Freeman

APPENDIXES

A CLINICAL EXAMINATION OF THE JOINTS 322
H. L. F. Currey

B LABORATORY INVESTIGATIONS 348
Graham R. V. Hughes and H. L. F. Currey
Rheumatoid Factors – LE-Cells – Antinuclear Factor – Uric Acid – Homogentisic Aciduria – Antistreptolysin 'O' – Synovial Fluid – Crystals in Synovial Fluid – Synovial Biopsy – Arthroscopy

1

The Applied Anatomy of Joints

B Vernon-Roberts

SYNOVIAL JOINTS

GENERAL ANATOMICAL FEATURES

The general anatomical features of a typical synovial joint are illustrated in Fig. 1.1.

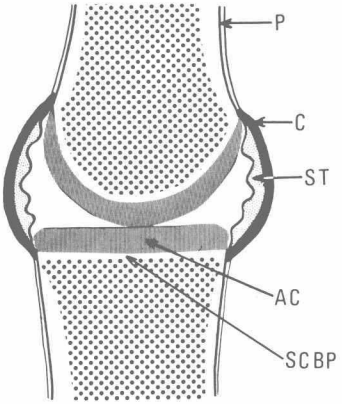

FIGURE 1.1 Diagrammatic representation of typical synovial joint. Shows articular cartilage (*AC*) lying on sub-chondral bone plate (*SCBP*), and non-articular surfaces within joint covered by synovial tissue (*ST*). The whole joint is enclosed by the capsule (*C*) which is continuous with the periosteum (*P*) covering the bone.

Articular Cartilage

The articular surface of each bone is covered by a layer of avascular hyaline cartilage up to 3 mm thick which acts as a resilient cushion and has an undulating surface advantageous for the purpose of joint lubrication. It is fixed to the underlying sub-chondral bone by collagen fibres arranged as a series of arcades so that the superficial fibres are tangential and the deep fibres are vertical. The cartilage is nourished by diffusion from the synovial tissues via the synovial fluid.

Capsule and Ligaments

The joint is enclosed by a fibrous tissue *capsule* attached to the bone near the periphery of the articular cartilage and in continuity with the periosteum. Thickenings of the capsule form named *ligaments* which serve to stabilise and strengthen the joint and to limit certain movements.

Synovial Tissues

The capsule is lined by *synovial tissue* which invests all the non-articular surfaces within the joint. The synovial tissue normally possesses variable numbers of folds and finger-like *synovial villi,* and comprises a thin intimal layer 1–4 cells in depth (Fig. 1.2) which forms the lining of

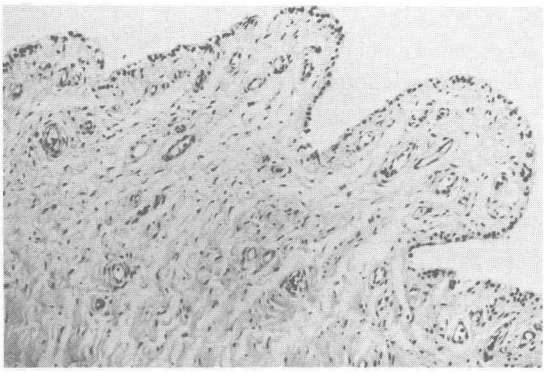

FIGURE 1.2 Section of normal synovial tissue. Shows thin intimal layer and vascular sub-intimal connective tissues.

the non-articular joint surface, and an underlying sub-intimal supporting layer composed of fibro-fatty connective tissue of varying thickness which merges with the capsule. The cells of the intimal layer are divisible into two principal types on the basis of their ultrastructure: the *type A cells* have the appearance of cells engaged in phagocytic activity and may contain a variety of ingested materials, whereas the *type B cells* have the appearance of cells engaged in synthetic activity and are concerned with the production of hyaluronic acid which has important functions in the lubrication of the load-bearing articular cartilage.

Synovial Fluid

Under normal conditions, a small quantity (about 2 ml in the knee joint) of the lubricant *synovial fluid* is present. It is clear, pale yellow, highly viscous, and contains less than 1,000 cells per cu. mm. The majority of cells are small lymphocytes (about 50 per cent) and the remainder are neutrophil polymorphs and mononuclear phagocytes.

Intra-articular Structures

Intra-articular fibro-cartilages completely dividing the joint cavity into separate compartments are present within the sternoclavicular and temporo-mandibular joints where movements in different planes take place in the separate compartments. In the knee joint, incomplete fibro-cartilages (menisci) change their shape to accommodate changes in the profiles of the femoral condyles during flexion and extension of the knee. Fatty pads covered by synovial tissue are present in some joints and occupy spaces where bony surfaces are incongruous.

Blood Supply

Hyaline cartilage is avascular, whereas bone is a richly vascular tissue. The synovial tissues are supplied with blood from the *circulus vasculosus,* a vascular plexus which lies between the capsule and the synovial tissue at their attachment to the epiphyseal line.

Nerve Supply

The synovial tissues are poorly innervated, but the capsule and ligaments have a rich sensory supply, and these tissues are probably the main origin of pain in the rheumatic diseases. The articular cartilage has no nerve supply and is therefore incapable of directly giving rise to pain. Although bone is well innervated, the distribution of pain afferents has not been established.

Stability of Synovial joints

The major factors contributing to the stability of joints are, in decreasing order of importance, muscles, ligaments and bones.

Muscles are the most important factor in maintaining joint stability. Joints having marked incongruity of their bony contours, such as the shoulder and knee, are particularly unstable in the absence of normal muscular function.

Ligaments are composed of relatively inelastic collagenous tissue and are an important factor in maintaining the stability of most joints by guarding against sudden stresses and preventing excessive movement. Muscles are more important in situations of continuous stress such as the support of the arches of the foot; however, in some cases, as with the ilio-femoral ligament of the hip joint, ligaments may dispense with the need for sustained muscular action in maintaining a particular attitude.

Whereas it is clear that bony contours play an important part in maintaining the stability of the ball-and-socket joint of the hip, the mortise joint of the ankle, and the hinge joint of the elbow, they can play little part in stabilising such joints as the shoulder, knee, wrist or digits of the hands and feet.

APPLIED ANATOMY

The progression of changes which occur in synovial joints in the rheumatic diseases are most easily understood by considering, in turn, the changes which occur in the affected joints at the microscopic level, and the macroscopic effects of these changes.

Osteoarthrosis

The earliest changes take place within the articular cartilage, particularly in those areas of articular cartilage exposed to the greatest mechanical stresses during life.

Articular Cartilage. Whatever the initial disturbance which occurs at the sub-cellular and chemical level, there is an early loss of proteoglycan (mucopolysaccharide) ground substance which particularly affects the superficial layers of the articular cartilage. This loss is accompanied by changes in the resilience of the cartilaginous matrix, and in response to normal mechanical stresses the collagen fibres within the superficial layers of cartilage begin to separate. The superficial fibres follow a tangential course and the earliest lesion is therefore horizontal flaking of the articular cartilage (Fig. 1.3).

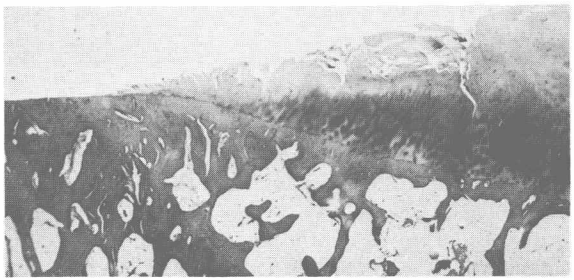

FIGURE 1.3 Articular surface in advanced osteoarthrosis. The cartilage on the right shows early horizontal flaking only, but the cartilage changes are more advanced in the centre of the picture. The full thickness of cartilage has been lost on the extreme left, and the exposed bone is polished (eburnated) and dense.

This splitting process, known as *fibrillation,* progresses to affect the deeper fibres (Fig. 1.3) which are arranged radially and parallel, and extends eventually through the cartilage to reach the underlying subchondral bone. Fibrillated cartilage is thinner than normal due to the necrosis and shedding of the superficial layers, and this results in a closer apposition of the bone surfaces which is radiologically (inaccurately) called 'diminution of joint space'. The process of fibrillation progresses centrifugally, and eventually the full thickness of cartilage is

lost and the underlying bone is exposed in the severely affected central areas (Fig. 1.3).

Bone. During destruction of the cartilage, there is advancing ossification into the deeper layers of the cartilage, and simultaneously new bone is laid down in the sub-chondral region. By the time that the full thickness of cartilage has been lost and the exposed bone comes to act as the weight-bearing surface, the sub-chondral bone is already much increased (Fig. 1.3).

As part of the general remodelling of bone in the area, there is also new bone formation at the margins of the articular surfaces, apparently to compensate for changes in the distribution of mechanical stresses, and this new bone forms the *osteophytes* (Fig. 1.6) which are a radiologically characteristic feature of osteoarthrosis.

At the same time that these changes are taking place, a light infiltrate of chronic inflammatory cells appears in the marrow spaces of the sub-chondral bone. Radiologically visible 'cysts' in the sub-chondral bone may be true cystic spaces sometimes communicating with the joint cavity through a defect in the articular surface (Fig. 1.4), or pseudocysts composed of fibrous tissue and chronic inflammatory cells replacing areas of lost bone.

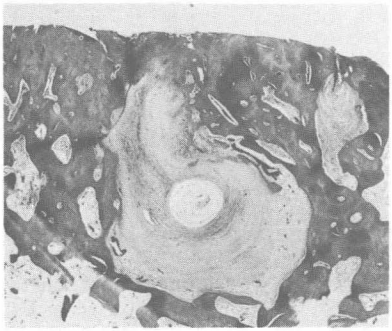

FIGURE 1.4 True cystic space in sub-chondral bone in advanced osteoarthrosis.

Synovial tissues. As a result of the damage to the cartilage with shedding of debris into the joint cavity, the synovial tissues may exhibit moderate villous proliferation and intimal cell hyperplasia (Fig. 1.5), and in long-standing cases there may be a sparse infiltrate of lymphocytes. There is a diffuse increase in collagenised fibrous tissue in the sub-synovial tissues, and the capsule also becomes markedly thickened by fibrous tissue.

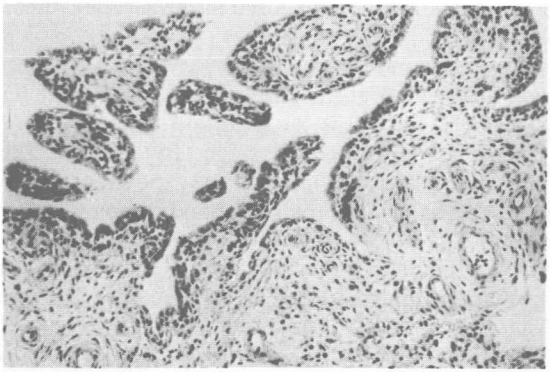

FIGURE 1.5 Synovial tissues in advanced osteoarthrosis. There is moderate intimal cell hyperplasia and villus formation, and the sub-intimal tissues are lightly infiltrated by chronic inflammatory cells. Compare with Figures 1.2 and 1.9.

Synovial fluid. The synovial fluid is usually increased in amount in the more severe grades of osteoarthrosis. Even when a large effusion is present, in the presence of osteoarthrosis uncomplicated by other pathology the fluid is usually clear and viscous, and the total cell count seldom exceeds 1,000 cells per cu. mm. The majority of cells are lymphocytes.

Gross pathological changes. With knowledge of the microscopic changes, it is a simple matter to follow the evolution of changes taking place at the macroscopic level. An arbitrary system of grading the severity of osteoarthrosis may be used, but this grading system in no way represents fixed changes in the progress of the disease.

GRADE 1 Tangential flaking, early fibrillation, shallow pits, blisters or grooves restricted to the superficial cartilage. Osteophytes absent.

GRADE 2 Deep fibrillation and cartilage destruction (but not exposing bone) in pressure areas, and superficial fibrillation elsewhere. No large osteophytes but early lipping of articular margins.

GRADE 3 Total loss of cartilage in one or more areas with exposure of underlying bone. Remaining cartilage exhibits extensive fibrillation. Sclerosis of sub-chondral bone. Marked osteophyte formation. Fibrosis of capsular tissues. Reactive proliferation of synovial tissues.

GRADE 4 Complete loss of articular cartilage from large areas. Exposed bone eburnated (polished) and grooved. Gross

irregularity of articular surfaces. Large osteophytic outgrowths from articular margins. Destruction of intra-articular ligaments. Extensive fibrosis of synovial and capsular tissues. Excess of viscous joint fluid. Deformity due to subluxation (articular surfaces articulate in abnormal position).

Osteoarthrosis at special sites. The *knee* is the joint most commonly and earliest affected by osteoarthrosis, and this is not surprising in view of the high mechanical pressures obtaining between the patella and femoral groove during flexion and extension; thus, osteoarthrosis of the patello-femoral articulation is almost universal after the fifth decade. It is also not surprising that severe osteoarthrosis of the knee is accompanied by loss of joint stability due to deformation of the articular surfaces which leads to subluxation, destruction of the cruciate ligaments, fibrosis of synovial and capsular tissues, and wasting of muscles due to disuse because of pain when moving the affected joint.

Severe osteoarthrosis of the *hip* joint is particularly disabling since large osteophytes form around the acetabular rim, the margin of the head and the intracapsular portion of the neck of the femur (Fig. 1.6). Apposition of these outgrowths limits movement early in the disease. Eventually, the joint may become fixed because of interlocking of the femoral and acetabular osteophytes, but true bony ankylosis rarely occurs in the absence of supervening infection. The femoral head may become flattened (Fig. 1.6) or cone-shaped and the acetabulum is correspondingly shallow or deepened.

The *finger joints,* especially the terminal inter-phalangeal joints, are not uncommonly the site of osteoarthrosis. The marginal osteophytes,

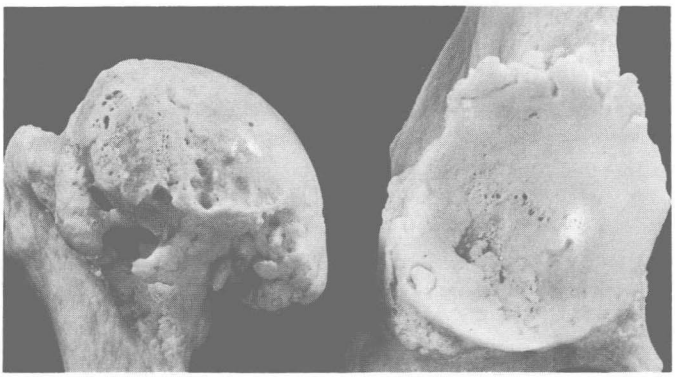

FIGURE 1.6 Severe osteoarthrosis of hip joint. Large osteophytic outgrowths extend from articular margins of femoral head (left) which is flattened and polished, and from margins of acetabulum (right) which is also polished.