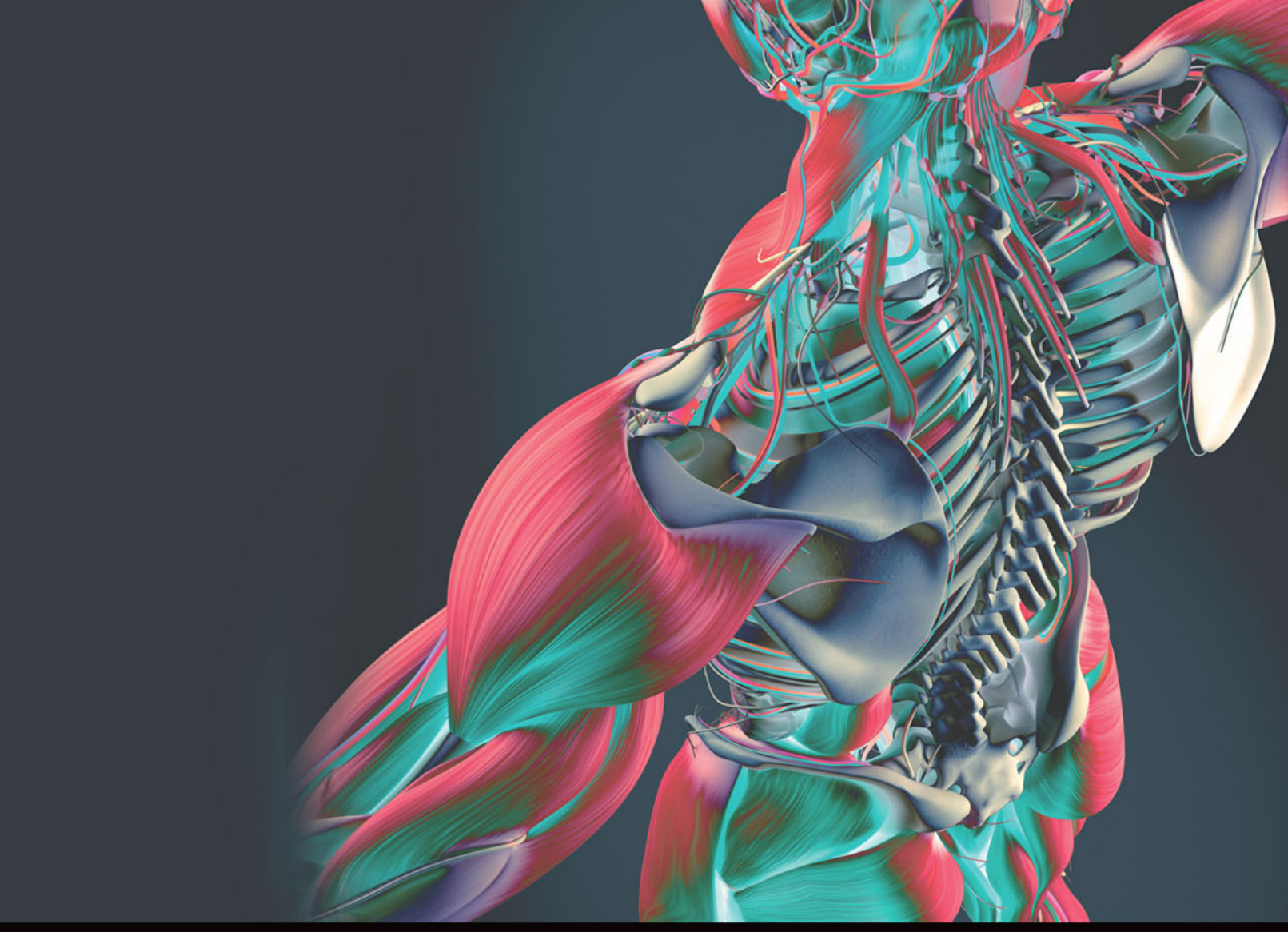

APLEY & SOLOMON'S

System of Orthopaedics and Trauma

Tenth Edition

EDITED BY

Ashley Blom, David Warwick, Michael R. Whitehouse

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CRC Press
Taylor & Francis Group

Apley and Solomon's System of Orthopaedics and Trauma

Alan Graham Apley 1914–1996

Louis Solomon 1928–2014

Inspired teachers, wise mentors and joyful friends

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Apley and Solomon's System of Orthopaedics and Trauma

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DEDICATION

To Louis
from your friends and colleagues on behalf of the thousands of patients
who have benefitted from your lifetime's work

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PREFACE

Orthopaedics in a changing world

Since Alan Apley published the first edition of this book the world has changed considerably and so has the practice of orthopaedic surgery. In 1959, hip replacement was rare and had high failure rates, knee replacement and arthroscopy did not exist and fractures were primarily treated in traction.

The last edition of this book commented on the projected impact of the HIV/AIDS epidemic. The epidemic has largely been brought under control, with effective treatment resulting in normal life expectancy for sufferers. However, in untreated individuals, the incidence of secondary infection such as tuberculosis is high and the prognosis is still dire. It is interesting and encouraging to note that both the National Joint Registry for England and Wales and the Malawian Joint Registry have shown that hip replacement is an effective treatment for patients who have multimorbidity which includes AIDS with no increased risk of early postoperative mortality compared with patients who do not have AIDS.

Over the lifetime of this book many treatments have been invented, extensively used, found to be ineffective or suboptimal and subsequently have declined dramatically in popularity. Examples of this include arthroscopic debridement for knee osteoarthritis, metal-on-metal hip replacement and excision arthroplasty of the distal ulna. It is important that we continue to challenge the efficacy of existing and novel treatments. In a world of increasing global need orthopaedics has to be proven to be efficacious and cost-effective.

Since 1959, the world's population has more than doubled to over 7 billion people and has aged considerably. Life expectancy at birth is now 80 years in Europe and 74 years in Asia. There are still marked disparities – for instance Japan has a life expectancy at birth of 83 years compared to 57 years in South Africa – but these differences are narrowing. It is projected that by 2050 4% of the world's population (but 16% of Japan's population) will be over 80 years of age. Between 2010 and 2050 the proportion of the population aged over 65 years will double in most countries, and it is predicted to increase from 5% to 11% in South Africa, 5% to 13% in India and 17% to 36% in Spain.

Orthopaedics remains as relevant a speciality as ever, treating a large burden of the world's morbidity. However, the nature of care has changed, with a much lower burden of chronic musculoskeletal infections today and a steeply rising incidence of joint replacement for primarily degenerative conditions. The World Health Organization estimates that 10% of men and 18% of women aged over 60 years have symptomatic osteoarthritis. Total knee and total hip replacement are now the second and third commonest elective operative procedures performed in developed countries. For example, in England and Wales, which have a combined population of approximately 55 million people, over 170 000 hip and knee replacements are performed annually. The provision of arthroplasty varies greatly, with 226 knee replacements per 100 000 population performed annually in the United States of America compared to only 3 per 100 000 population in neighbouring Mexico. Increasingly the outcomes of common procedures, such as arthroplasty and fractured neck of femur fixation, are being monitored by national registries in a wide range of countries and healthcare settings. It is heartening that even low-income countries such as Malawi have established implant registries which are providing clinically important data. As the prevalence of infectious diseases declines in low-income countries and people live longer, more health resources will be spent on treating long-term conditions of the elderly such as osteoarthritis.

Accidents and emergencies still represent a major healthcare burden. Over 1.25 million people die worldwide annually as a result of road traffic accidents. The majority of these occur in Asia. Millions more are seriously injured. Injuries from road traffic accidents are the third largest cause of morbidity among adult males. Orthopaedic care remains of paramount importance for effectively and quickly returning patients as closely as possible to their pre-injury state and thereby allowing them to participate fully in society.

The provision of health care and resources varies considerably between countries: Greece has 6.3 doctors per 1000 population, South Africa has 0.8 and India only 0.7. While the number of doctors practising in some countries has remained relatively static, in Australia and the United Kingdom there has been an increase of over 50% in the number of registered doctors in the past decade. Part of this is due to migration of doctors, which may exacerbate shortages in low-income countries. More than 40 000 foreign-trained doctors, including an author of this preface, work in the United Kingdom, nearly half of whom come from India and Pakistan. In Israel, New Zealand, Norway and Ireland over a third of practising doctors are foreign-trained. Movement of doctors between countries promotes the spread of ideas and innovation and improves training. However, there is a natural gravitation of expertise towards countries that offer higher remuneration and better working conditions at the expense of low- and middle-income countries. The United States of America spends $8713 per capita on health care, while China spend $649 and India $215.

With rapidly increasing per capita GDP in countries such as China and India, the demographics of health care will change markedly over the next decade. The relative need to treat infection and injury will hopefully decline, but this will inevitably be coupled with an increase in treatments for longer-term musculoskeletal conditions.

Ashley W. Blom
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Bristol and Southampton, 2017

Data are publically available from the OECD at:
http://www.oecd-ilibrary.org/social-issues-migration-health/health-at-a-glance_19991312#

PREFACE TO THE NINTH EDITION

When Alan Apley produced the first edition of his System of Orthopaedics and Fractures 50 years ago he saw it as an aid to accompany the courses that he conducted for aspiring surgeons who were preparing for the FRCS exams. With characteristic humour, he called the book 'a prophylactic against writer's cramp'. Pictures were unnecessary: if you had any sense (and were quick enough to get on the heavily oversubscribed Apley Course), you would be treated to an unforgettable display of clinical signs by one of the most gifted of teachers.

You also learnt how to elicit those signs by using a methodical clinical approach – the Apley System. The Fellowship exam was heavily weighted towards clinical skills. Miss an important sign or stumble over how to examine a knee or a finger and you could fail outright. What Apley taught you was how to order the steps in physical examination in a way that could be applied to every part of the musculoskeletal system. 'Look, Feel, Move' was the mantra. He liked to say that he had a preference for four-letter words. And always in that order! Deviate from the System by grasping a patient's leg before you look at it minutely, or by testing the movements in a joint before you feel its contours and establish the exact site of tenderness and you risked becoming an unwilling participant in a theatrical comedy.

Much has changed since then. With each new edition the System has been expanded to accommodate new tests and physical manoeuvres developed in the tide of super-specialization. Laboratory investigations have become more important and imaging techniques have advanced out of all recognition. Clinical classifications have sprung up and attempts are now made to find a numerical slot for every imaginable fracture. No medical textbook is complete without its 'basic science' component, and advances are so rapid that changes become necessary within the period of writing a single edition. The present volume is no exception: new bits were still being added right up to the time of proofreading.

For all that, we have retained the familiar structure of the Apley System. As in earlier editions, the book is divided into three sections: General Orthopaedics, covering the main types of musculoskeletal disorder; Regional Orthopaedics, where we engage with these disorders in specific parts of the body; and thirdly Fractures and Joint Injuries. In a major departure from previous editions, we have enlisted the help of colleagues who have particular experience of conditions with which we as principal authors are less familiar. Their contributions are gratefully acknowledged. Even here, though, we have sought their permission to 'edit' their material into the Apley mould so that the book still has the sound and 'feel' of a single authorial voice.

For the second edition of the book, in 1963, Apley added a new chapter: 'The Management of Major Accidents'. Typically frank, he described the current arrangements for dealing with serious accidents as 'woefully inadequate' and offered suggestions based on the government's Interim Report on Accident Services in Great Britain and Ireland (1961). There has been a vast improvement since then and the number of road accident deaths today is half of what it was in the 1960s (Department of Transport statistics). So important is this subject that the relevant section has now been rewritten by two highly experienced Emergency and Intensive Care Physicians and is by far the longest chapter in the present edition.

Elsewhere the text has been brought completely up to date and new pictures have been added. In most cases the illustrations appear as composites – a series of images that tell a story rather than a single 'typical' picture at one moment in the development of some disorder. At the beginning of each Regional chapter, in a run of pictures we show the method of examining that region: where to stand, how to confront the patient and where to place our hands. For the experienced reader this may seem like old hat; but then we have designed this book for orthopaedic surgeons of all ages and all levels of experience. We all have something to learn from each other.

As before, operations are described only in outline, emphasizing the principles that govern the choice of treatment, the indications for surgery, the design of the operation, its known complications and the likely outcome. Technical procedures are learnt in

simulation courses and, ultimately, in the operating theatre. Written instructions can only ever be a guide. Drawings are usually too idealized and 'in theatre' photographs are usually intelligible only to someone who has already performed that operation. Textbooks that grapple with these impediments tend to run to several volumes.

The emphasis throughout is on clinical orthopaedics. We acknowledge the value of a more academic approach that starts with embryology, anatomy, biomechanics, molecular biology, physiology and pathology before introducing any patient to the reader. Instead we have chosen to present these 'basic' subjects in small portions where they are relevant to the clinical disorder under discussion: bone growth and metabolism in the chapter on metabolic bone disease, genetics in the chapter on osteodystrophies, and so forth.

In the preface to the last edition we admitted our doubts about the value of exhaustive lists of references at the end of each chapter. We are even more divided about this now, what with the plethora of 'search engines' that have come to dominate the internet. We can merely bow our heads and say we still have those doubts and have given references only where it seems appropriate to acknowledge where an old idea started or where something new is being said that might at first sight be questioned.

More than ever we are aware that there is a dwindling number of orthopaedic surgeons who grew up in the Apley era, even fewer who experienced his thrilling teaching displays, and fewer still who worked with him. Wherever they are, we trust that they will recognize the Apley flavour in this new edition. Our chief concern, however, is for the new readers who – we hope – will glean something that helps them become the next generation of teachers and mentors.

LS
SN
DJW

ACKNOWLEDGEMENTS

This textbook is an iterative process and for this current edition new authors have been asked to revise and refresh the existing text. The editors and new authors thoroughly acknowledge the contribution of those who have gone before them, much of whose work remains in this updated text.

Chapter 2, *Infection*, contains some material from 'Infection' by Louis Solomon, H. Srinivasan, Surendar Tuli & Shunmugam Govender. The material has been revised and updated by the current author.

Chapter 4, *Crystal deposition disorders*, contains some material from 'Crystal deposition disorders' by Louis Solomon. The material has been revised and updated by the current authors.

Chapter 5, *Osteoarthritis*, contains some material from 'Osteoarthritis and related disorders' by Louis Solomon. The material has been revised and updated by the current authors.

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Chapter 8, *Genetic disorders, skeletal dysplasias and malformations*, contains some material from 'Genetic disorders, skeletal dysplasias and malformations' by Louis Solomon & Deborah Eastwood. The material has been revised and updated by the current authors.

Chapter 9, *Tumours*, contains some material from 'Tumours' by Will Aston, Timothy Briggs & Louis Solomon. The material has been revised and updated by the current authors.

Chapter 10, *Neuromuscular disorders*, contains some material from 'Neuromuscular disorders' by Deborah Eastwood, Thomas Staunton & Louis Solomon. The material has been revised and updated by the current author.

Chapter 11, *Peripheral nerve disorders*, contains some material from 'Peripheral nerve injuries' by David Warwick, H. Srinivasan & Louis Solomon. The material has been revised and updated by the new contributor Michael Fox.

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Chapter 18, *The back*, contains some material from 'The back' by Stephen Eisenstein, Surendar Tuli & Shunmugam Govender. The material has been revised and updated by the current authors.

Chapter 19, *The hip*, contains some material from 'The hip' by Louis Solomon, Reinhold Ganz, Michael Leunig, Fergal Monsell & Ian Learmonth. The material has been revised and updated by the current authors.

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Chapter 26 The wrist Contains some material from 'Injuries of the Forearm and Wrist' by David Warwick with updates from the new contributors Adam Watts, Mike Uglow and Joanna Thomas.

Chapter 28, *Injuries of the spine*, contains some material from 'Injuries of the spine' by Stephen Eisenstein & Wagih El Masry. The material has been revised and updated by the current authors.

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Chapter 31, *Injuries of the knee and leg*, contains some material from 'Injuries of the knee and leg' by Selvadurai Nayagam. The material has been revised and updated by the current author.

LIST OF ABBREVIATIONS USED

AAS	atlantoaxial subluxation
ABC	aneurysmal bone cyst
ABPI	ankle brachial pressure index
ACA	angulation correction axis
ACDF	anterior cervical discectomy and fusion
ACE	angiotensin-converting enzyme
ACEI	angiotensin-converting enzyme inhibitor
ACL	anterior cruciate ligament
ACLR	anterior cruciate ligament reconstruction
ACPA	anti-citrullinated peptide antibodies
ACTH	adrenocorticotropic hormone
ADH	antidiuretic hormone
ADHD	attention deficit hyperactivity disorder
ADHR	autosomal dominant hypophosphataemic rickets
ADI	atlantodental interval
ADL	activity of daily living
AFO	ankle–foot orthosis
AFP	alpha-fetoprotein
AIDP	acute inflammatory demyelinating polyneuropathy
AIDS	acquired immune deficiency syndrome
AJCC	American Joint Committee on Cancer
AL	anterolateral
ALI	acute lung injury
ALIF	anterior lumbar interbody fusion
ALP	alkaline phosphatase
ALS	amyotrophic lateral sclerosis
AM	anteromedial
AMC	arthrogryposis multiplex congenita
ANA	antinuclear antibodies
anti-CCP	anti-cyclic citrullinated peptide antibodies
AO/ASIF	Arbeitsgemeinschaft für Osteosynthesefragen/Association for the Study of Internal Fixation
AP	anteroposterior
APACHE	Acute Physiology and Chronic Health Evaluation (model)
APC	antigen-presenting cell *and* anteroposterior compression (injuries)
ARCO	Association Research Circulation Osseous
ARDS	acute respiratory distress syndrome
ARHR	autosomal recessive hypophosphatemic rickets
ARM	awareness, recognition, management
ARMD	adverse reaction to metal debris
AS	ankylosing spondylitis
ASCT	autologous stem-cell transplantation
ASIS	anterior superior iliac spine
ATFL	anterior talofibular ligament
ATLS	Advanced Trauma Life Support
AUSCAN	Australian–Canadian Hand Osteoarthritis Index
AVN	avascular necrosis
AVPU	aware, verbally responsive, pain responsive, and unresponsive
BAPRAS	British Association of Plastic, Reconstructive and Aesthetic Surgeons
BASICS	British Association for Immediate Care
BCIS	bone cement implantation syndrome
BCP	bicalcium phosphate
BMD	bone mineral density
BMI	body mass index
BMP	bone morphogenetic protein
BOA	British Orthopaedic Association
BOAST	BOA Standards for Trauma
BSA	body surface area
BSR	British Society for Rheumatology
BUN	blood urea nitrogen
BVM	bag–valve–mask
CaSR	calcium-sensing receptor
C-A-T™	Combat Application Tourniquet
CC	cartilage calcification
CCP	cyclic citrullinated peptide
CDH	congenital dislocation of the hip
CDR	cervical disc replacement
4CF	four-corner fusion
CIMT	constraint-induced movement therapy
CKD-MBD	chronic kidney disease mineral bone disorder
CMAP	compound muscle action potential

CMC	carpometacarpal
CMI	cell-mediated immunity
CNS	central nervous system
COC	ceramic on ceramic (THA bearing)
COMP	cartilage oligomeric matrix protein
COP	ceramic on polyethylene (THA bearing)
CORA	centre of rotation of angulation
COX-2	cyclooxygenase-2
CPM	continuous passive motion
CPPD	calcium pyrophosphate dihydrate
CR	cruciate retaining
CRP	C-reactive protein
CRPS	complex regional pain syndrome
CSF	cerebrospinal fluid
CT	computed tomography
CTX	serum type I collagen C-terminal cross-linking telopeptide
CVP	central venous pressure
DDD	degenerative disc disease
DDH	developmental dysplasia of the hip
DIC	disseminated intravascular coagulation
DIP(J)	distal interphalangeal (joint)
DISH	diffuse idiopathic skeletal hyperostosis
DISI	dorsal intercalated segment instability
DLC	*discoligamentous complex*
DLIF	direct lateral interbody fusion
DMARDs	disease-modifying antirheumatic drugs
DMD	Duchenne muscular dystrophy
DNA	deoxyribonucleic acid
DRUJ	distal radioulnar joint
DTH	delayed type hypersensitivity
DVT	deep vein thrombosis
DXA	dual-energy X-ray absorptiometry
ECRB	extensor carpi radialis brevis
ECRL	extensor carpi radialis longus
ECU	extensor carpi ulnaris
EDF	elongation–derotation–flexion
EEG	electroencephalography
eFAST	extended focused assessment sonography in trauma
eGFR	estimated glomerular filtration rate
EMG	electromyography
EMS	emergency medical service
EMT	emergency medical technician
ENL	erythema nodosum leprosum
ENT	ear, nose and throat
EPL	extensor pollicis longus
ESR	erythrocyte sedimentation rate
ETA	estimated time of arrival
$EtCO_2$	end-tidal carbon dioxide
EULAR	European League Against Rheumatism
FAB	foot abduction brace
FABER	Flexion, ABduction, and External Rotation test
FABS	flexion, abduction, supination
FAI	femoroacetabular impingement
FAST	focused assessment sonography in trauma
FBC	full blood count
FDP	flexor digitorum profundus
FDS	flexor digitorum superficialis
FFF-STA	Flat foot associated with a short tendo Achilles
FFO	functional foot orthoses
FGF	fibroblast growth factor
FGFR	fibroblast growth receptor
FHH	familial hypocalciuric hypercalcaemia
FHON	femoral head osteonecrosis
FISH	fluorescence in situ hybridization
FLS	Fracture Liaison Services
fMRI	functional magnetic resonance imaging
FMS	fibromyalgia syndrome
FNCLCC	Federation Nationale des Centres de Lutte Contre le Cancer
FPB	flexor pollicis brevis
FPE	fatal pulmonary embolism
FPL	flexor pollicis longus
GABA	gamma-aminobutryic acid
GAGs	glycosaminoglycans
GCS	Glasgow Coma Scale
GCT	giant cell tumour
GCTTS	giant cell tumour of tendon sheath
GMFCS	gross motor function classification system
GPI	general paralysis of the insane
GGT	gamma-glutamyl transferase
GH	*growth hormone*
GRF	ground reaction force
HA	hydroxyapatite
HEMS	helicopter emergency medical service
HHR	humeral head replacement
HIE	hypoxic–ischaemic encephalopathy
HIV	human immunodeficiency virus
HLA	human leucocyte antigen
HMSN	hereditary motor and sensory neuropathy
HNPP	hereditary neuropathy with liability to pressure palsies
HO	heterotopic ossification
HOOS	Hip Dysfunction and Osteoarthritis Outcome Score
HR	hip resurfacing
HRT	hormone replacement therapy
IASP	International Association for the Study of Pain
ICF	International Classification of Functioning, Disability and Health
ICP	intracerebral pressure
ICS	intercostal space
ICU	intensive care unit

IDH isocitrate dehydrogenase
IFSSH International Federation of Societies for Surgery of the Hand
IGRA interferon-gamma release assay
IL interleukin
IM intramuscular
IMRT intensity-modulated radiotherapy
INR international normalized ratio
IP(J) interphalangeal joint
IRIS immune reconstitution inflammatory syndrome
IRMER Ionising Radiation Medical Exposure Regulations
ISS injury severity score
ITB intrathecal baclofen
IV intervertebral *and* intravenous
IVF *in vitro* fertilization
IVH intraventricular haemorrhage
JIA juvenile idiopathic arthritis
JOAMEQ Japanese Orthopaedic Association Cervical Myelopathy Evaluation Questionnaire
KAFO knee–ankle–foot orthosis
KOOS Knee Dysfunction and Osteoarthritis Outcome Score
LBP lower back pain
LC lateral compression
LCH Langerhans cell histiocytosis
LCL lateral collateral ligament
LCPD Legg–Calvé–Perthes disease
LDH lactate dehydrogenase
LHB long head of biceps
LLD leg length discrepancy
LMA laryngeal mask airway
LMN lower motor neuron
LMWH low molecular weight heparin
MAP mean arterial pressure
MARS metal artifact reduction sequences (MRI)
MB multibacillary
MCL medial collateral ligament
MCP(J) metacarpophalangeal (joint)
M-CSF macrophage colony-stimulating factor
MCV mean corpuscular volume
MDM2 murine double minute-2
MED multiple epiphyseal dysplasia
MEN multiple endocrine neoplasia
MGUS monoclonal gammopathy of undetermined significance
MHC major histocompatibility complex
MIC minimal inhibitory concentration
MIPO minimally invasive percutaneous osteosynthesis
MND motor neuron disease
MO multiple osteochondromas
MODS multiple organ failure or dysfunction syndrome
MOM metal on metal (THA bearing)
MOP metal on polyethylene (THA bearing)
MP migration percentage
MPFL medial patellofemoral ligament
MPM mortality prediction model
MPNST malignant peripheral nerve sheath tumour
MPS mucopolysaccharidoses
MRC Medical Research Council
MRA magnetic resonance arthrography or angiography
MRI magnetic resonance imaging
MRSA methicillin-resistant *Staphylococcus aureus*
MSSA methicillin-sensitive *Staphylococcus aureus*
MTC Major Trauma Centre
MTP(J) metatarsophalangeal (joint)
NARU National Ambulance Resilience Unit
NCIN *National Cancer Intelligence Network*
NCTH non-compressible torso haemorrhage
NCV nerve conduction velocity
NDI Neck Disability Index
NF neurofibromatosis
NIBP non-invasive blood pressure
NICE National Institute for Health and Care Excellence
NOF non-ossifying fibroma
NP nasopharyngeal
NPS Nail–patella syndrome
NSAIDs non-steroidal anti-inflammatory drugs
OA osteoarthritis
OCD osteochondritis dissecans
OFD osteofibrous dysplasia
OI osteogenesis imperfecta
OMT Oberg, Manske and Tonkin (classification)
ONJ osteonecrosis of the jaw
OP oropharyngeal
OPG osteoprotegerin
OPLL ossification of the posterior longitudinal ligament
P1NP serum type I collagen extension propeptide
PA posteroanterior
PACS Picture Archiving and Communication System
PAFC pulmonary artery flotation catheterization
PAO periacetabular osteotomy
PAOP pulmonary artery occlusion pressure
PB paucibacillary
PCA patient-controlled analgesia
PCL posterior cruciate ligament
PCR polymerase chain reaction
PD proton density
PDB Paget's disease of bone

PE pulmonary embolism
PEA pulseless electrical activity
PEEP positive end-expiratory pressure
PET positron emission tomography
PH Pavlik harness
PHEM pre-hospital emergency medicine
Pi inorganic phosphate
PIP(J) proximal interphalangeal (joint)
PJI periprosthetic infection
PL posterolateral
PLC posterior ligamentous complex *and* posterolateral corner
PLL posterior longitudinal ligament
PLRI posterolateral rotatory instability
PM posteromedial
PMMA polymethylmethacrylate
PNS peripheral nervous system
PPE personal protective equipment
PPS post-polio syndrome
pQCT peripheral quantitative computer tomography
PRC proximal row carpectomy
PRICE protection, rest, ice, compression and elevation
PRICER protection, rest, ice, compression, elevation and rehabilitation
PRP platelet rich plasma
PS posterior stabilized
PSA prostate-specific antigen
PsA psoriatic arthritis
PTH parathyroid hormone
PTHrP parathyroid hormone-related peptide
PTS post-thrombotic syndrome
PVL periventricular leucomalacia
PVNS pigmented villonodular synovitis
QCT quantitative computed tomography
QoL quality of life
QUS quantitative ultrasonometry
RA radiographic absorptiometry *and* rheumatoid arthritis
RANKL receptor activator of nuclear factor-$\acute{\kappa}\beta$ ligand
REBOA resuscitative endovascular balloon occlusion of the aorta
RF rheumatoid factor
RGO reciprocating gait orthoses
RICE rest, ice, compression and elevation
RNA ribonucleic acid
RR reversal reaction
RSD reflex sympathetic dystrophy
RSI rapid sequence induction
RTC road traffic crash
SAC space available for spinal cord
SACE serum angiotensin converting enzyme
SAMU Services de l'Aide Medical Urgente
SAPHO synovitis, acne, pustulosis, hyperostosis and osteitis
SAPS simplified acute physiology score
SAS subaxial subluxation
SBC simple bone cyst
SCFE slipped capital femoral epiphysis
SCI spinal cord injury
SCIWORA spinal cord injury without obvious radiographic abnormality
SCM sternocleidomastoid muscle
SDD selective digestive tract decontamination
SDR selective dorsal rhizotomy
SE spin echo
SED spondyloepiphyseal dysplasia
SEMLS single event multi-level surgery
SERM selective oestrogen receptor modulator
SIJ sacroiliac joint
SIRS systemic inflammatory response
SLAP superior labrum, anterior and posterior (tear)
SLE systemic lupus erythematosus
SLIC Subaxial Cervical Spine Injury Classification
SMR standardized mortality ratio
SMUR Services Mobile d'Urgence et de Reamination
SNAP sensory nerve action potential
SNPs single nucleotide polymorphisms
SOFA sequential organ failure assessment
SONK 'spontaneous' osteonecrosis of the knee
SOP standard operating procedure
SPA spondyloarthropathy
SpA spondyloarthritis
SPECT single photon emission computed tomography
SPORT Spine Patient Outcomes Research Trial
STIR short-tau inversion recovery
STT scaphoid–trapezium–trapezoid arthritis *and* soft-tissue tumour
SUA serum uric acid
SUFE slipped upper femoral epiphysis
TAR thrombocytopaenia with absent radius syndrome
TARN Trauma Audit and Research Network
TB tuberculosis
TBI total body involvement
TBS Trabecular Bone Score
^{99m}Tc-HDP technetium(^{99m}Tc)-labelled hydroxymethylene diphosphonate
^{99m}Tc-MDP technetium(^{99m}Tc)-labelled methyl diphosphonate
TDR total disc replacement
TE time to echo
TFCC triangular fibrocartilage complex
THA total hip arthroplasty
TIP terminal interphalangeal (joint)

TISS therapeutic intervention scoring system
TKR total knee replacement
TLIF transforaminal lumbar interbody fusion
TMT tarsometatarsal
TNF tumour necrosis factor
TNM tumour–node–metastasis
TOE transoesophageal echocardiogram
TSF Taylor spatial frame
TSH thyroid-stimulating hormone
TSR total shoulder replacement
TU Trauma Unit
UCP unilateral cerebral palsy
UFD unifacet dislocation
UHMWPE ultra-high molecular weight polyethylene
UICC Union for International Cancer Control
ULT urate-lowering therapy
UMN upper motor neuron
UPS undifferentiated pleomorphic sarcoma
US ultrasound
VAC vacuum-assisted closure
VACTERLS refers to the systems involved and the defects identified: *v*ertebral, *a*nal, *c*ardiac, *t*racheal, *e*sophageal, *r*enal, *l*imb and *s*ingle umbilical artery
VCR vertebral column resection
VCT voluntary counselling and testing
VFA Vertebral Fracture Assessment
VISI volar intercalated segment instability
VMO vastus medialis oblique
VP ventriculoperitoneal
VQ ventilation–perfusion
VS vertical shear *and* vertical subluxation
VTE venous thromboembolism
WALANT wide awake local anaesthetic no tourniquet
WBC white blood cell
WHO World Health Organization
XLH sex-linked hypophosphataemic rickets

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Section 1

General Orthopaedics

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Diagnosis in orthopaedics

1

Louis Solomon & Charles Wakeley

Orthopaedics is concerned with bones, joints, muscles, tendons and nerves – the skeletal system and all that makes it move. Conditions that affect these structures fall into seven easily remembered pairs:

1 Congenital and developmental abnormalities
2 Infection and inflammation
3 Arthritis and rheumatic disorders
4 Metabolic and endocrine disorders
5 Tumours and lesions that mimic them
6 Neurological disorders and muscle weakness
7 Injury and mechanical derangement

Diagnosis in orthopaedics, as in all of medicine, is the identification of disease. It begins from the very first encounter with the patient and is gradually modified and fine-tuned until we have a picture, not only of a pathological process but also of the functional loss and the disability that goes with it. Understanding evolves from the systematic gathering of information from the history, the physical examination, tissue and organ imaging and special investigations. Systematic, but never mechanical; behind the enquiring mind there should also be what D. H. Lawrence has called 'the intelligent heart'. It must never be forgotten that the patient has a unique personality, a job and hobbies, a family and a home; all have a bearing upon, and are in turn affected by, the disorder and its treatment.

HISTORY

'Taking a history' is a misnomer. The patient tells a story; it is we the listeners who construct a history. The story may be maddeningly disorganized; the history has to be systematic. Carefully and patiently compiled, it can be every bit as informative as examination or laboratory tests.

As we record it, certain key words and phrases will inevitably stand out: injury, pain, stiffness, swelling, deformity, instability, weakness, altered sensibility and loss of function or inability to do certain things that were easily accomplished before.

Each symptom is pursued for more detail: we need to know when it began, whether suddenly or gradually, spontaneously or after some specific event; how it has changed or progressed; what makes it worse; what makes it better.

While listening, we consider whether the story fits some pattern that we recognize, for we are already thinking of a diagnosis. Every piece of information should be thought of as part of a larger picture which gradually unfolds in our understanding. The surgeon-philosopher Wilfred Trotter (1870–1939) put it well: 'Disease reveals itself in casual parentheses.'

Symptoms

Pain

Pain is the most common symptom in orthopaedics. It is usually described in metaphors that range from inexpressively bland to unbelievably bizarre – descriptions that tell us more about the patient's state of mind than about the physical disorder. Yet there are clearly differences between the throbbing pain of an abscess and the aching pain of chronic arthritis, between the 'burning pain' of neuralgia and the 'stabbing pain' of a ruptured tendon.

Severity is even more subjective. High and low pain thresholds undoubtedly exist, but pain is as bad as it feels to the patient, and any system of 'pain grading' must take this into account. The main value of estimating severity is in assessing the progress of the disorder or the response to treatment. The commonest method is to invite the patient to mark the severity on an analogue scale of 1–10, with 1 being mild and easily ignored, and 10 being totally unbearable. The problem about this type of grading is that patients who have never experienced very severe pain simply do not know what 8 or 9 or

10 would feel like. The following is suggested as a simpler system:

Grade I (mild) Pain that can easily be ignored
Grade II (moderate) Pain that cannot be ignored, interferes with function and needs attention or treatment from time to time
Grade III (severe) Pain that is present most of the time, demanding constant attention or treatment
Grade IV (excruciating) Totally incapacitating pain

Identifying the site of pain may be equally vague. Yet its precise location is important, and in orthopaedics it is useful to ask the patient to point to – rather than to say – where it hurts. Even then, do not assume that the site of pain is necessarily the site of pathology; 'referred' pain and 'autonomic' pain can be very deceptive.

Referred pain Pain arising in or near the skin is usually localized accurately. Pain arising in deep structures is more diffuse and is sometimes of unexpected distribution; thus, hip disease may manifest with pain in the knee (so might an obturator hernia). This is not because sensory nerves connect the two sites; it is due to inability of the cerebral cortex to differentiate clearly between sensory messages from separate but embryologically related sites. A common example is 'sciatica' – pain at various points in the buttock, thigh and leg, supposedly following the course of the sciatic nerve. Such pain is not necessarily due to pressure on the sciatic nerve or the lumbar nerve roots; it may be 'referred' from any one of a number of structures in the lumbar spine, the pelvis and the posterior capsule of the hip joint. See Figure 1.1.

Autonomic pain We are so accustomed to matching pain with some discrete anatomical structure and its known sensory nerve supply that we are apt to dismiss any pain that does not fit the usual pattern as 'atypical' or 'inappropriate' (i.e. psychologically determined).

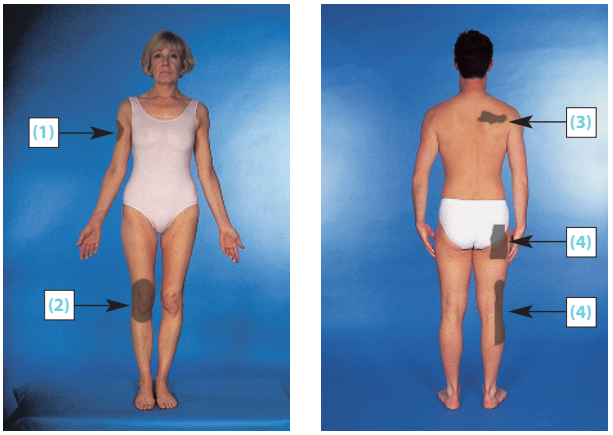


Figure 1.1 Referred pain Common sites of referred pain: (1) from the shoulder; (2) from the hip; (3) from the neck; (4) from the lumbar spine.

But pain can also affect the autonomic nerves that accompany the peripheral blood vessels and this is much more vague, more widespread and often associated with vasomotor and trophic changes. It is poorly understood, often doubted, but nonetheless real.

Stiffness

Stiffness may be generalized (typically in systemic disorders such as rheumatoid arthritis and ankylosing spondylitis) or localized to a particular joint. Patients often have difficulty in distinguishing localized stiffness from painful movement; limitation of movement should never be assumed until verified by examination.

Ask when it occurs: regular early morning stiffness of many joints is one of the cardinal symptoms of rheumatoid arthritis, whereas transient stiffness of one or two joints after periods of inactivity is typical of osteoarthritis.

Locking 'Locking' is the term applied to the sudden inability to complete a particular movement. It suggests a mechanical block – for example, due to a loose body or a torn meniscus becoming trapped between the articular surfaces of the knee. Unfortunately, patients tend to use the term for any painful limitation of movement; much more reliable is a history of 'unlocking', when the offending body slips out of the way.

Swelling

Swelling may be in the soft tissues, the joint or the bone; to the patient they are all the same. It is important to establish whether it followed an injury, whether it appeared rapidly (think of a haematoma or a haemarthrosis) or slowly (due to inflammation, a joint effusion, infection or a tumour), whether it is painful (suggestive of acute inflammation, infection or a tumour), whether it is constant or comes and goes, and whether it is increasing in size.

Deformity

The common deformities are described by patients in terms such as round shoulders, spinal curvature, knock knees, bow legs, pigeon toes and flat feet. Deformity of a single bone or joint is less easily described and the patient may simply declare that the limb is 'crooked'.

Some 'deformities' are merely variations of the normal (e.g. short stature or wide hips); others disappear spontaneously with growth (e.g. flat feet or bandy legs in an infant). However, if the deformity is progressive, or if it affects only one side of the body while the opposite joint or limb is normal, it may be serious (Figure 1.2).

Weakness

Generalized weakness is a feature of all chronic illness, and any prolonged joint dysfunction will inevitably

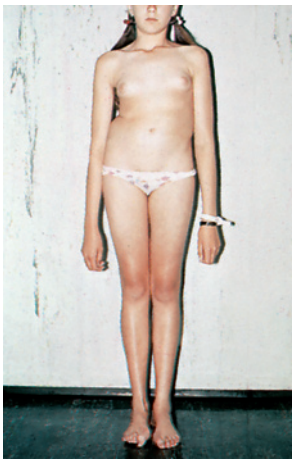



Figure 1.2 Deformity This young girl complained of a prominent right hip; the real deformity was scoliosis.

lead to weakness of the associated muscles. However, pure muscular weakness – especially if it is confined to one limb or to a single muscle group – is more specific and suggests some neurological or muscle disorder. Patients sometimes say that the limb is 'dead' when it is actually weak, and this can be a source of confusion. Questions should be framed to discover precisely which movements are affected, for this may give important clues, if not to the exact diagnosis at least to the site of the lesion.

Instability

The patient may complain that the joint 'gives way' or 'jumps out of place'. If this happens repeatedly, it suggests abnormal joint laxity, capsular or ligamentous deficiency, or some type of internal derangement such as a torn meniscus or a loose body in the joint. If there is a history of injury, its precise nature is important.

Change in sensibility

Tingling or numbness signifies interference with nerve function – pressure from a neighbouring structure (e.g. a prolapsed intervertebral disc), local ischaemia (e.g. nerve entrapment in a fibro-osseous tunnel) or a peripheral neuropathy. It is important to establish its exact distribution; from this we can tell whether the fault lies in a peripheral nerve or in a nerve root. We should also ask what makes it worse or better; a change in posture might be the trigger, thus focusing attention on a particular site.

Loss of function

Functional disability is more than the sum of individual symptoms and its expression depends upon the needs of that particular patient. The patient may say, 'I can't stand for long' rather than 'I have backache'; or 'I can't put my socks on' rather than 'My hip is stiff.' Moreover, what to one patient is merely inconvenient may, to another, be incapacitating. Thus a lawyer or a teacher may readily tolerate a stiff knee provided it is painless, but to a plumber or a parson the same disorder might spell economic or spiritual disaster. One question should elicit the important information: 'What can't you do now that you used to be able to do?'

Past history

Patients often forget to mention previous illnesses or accidents, or they may simply not appreciate their relevance to the present complaint. They should be asked specifically about childhood disorders, periods of incapacity and old injuries. A 'twisted ankle' many years ago may be the clue to the onset of osteoarthritis in what is otherwise an unusual site for this condition. Gastrointestinal disease, which in the patient's mind has nothing to do with bones, may be important in the later development of ankylosing spondylitis or osteoporosis. Similarly, certain rheumatic disorders may be suggested by a history of conjunctivitis, iritis, psoriasis or urogenital disease. Metastatic bone disease may erupt many years after a mastectomy for breast cancer. Patients should also be asked about previous medication: many drugs, and especially corticosteroids, have long-term effects on bone. Alcohol and drug abuse are important, and we must not be afraid to ask about them.

Family history

Patients often wonder (and worry) about inheriting a disease or passing it on to their children. To the doctor, information about musculoskeletal disorders in the patient's family may help with both diagnosis and counselling. When dealing with a suspected case of bone or joint infection, ask about communicable diseases, such as tuberculosis or sexually transmitted disease, in other members of the family.

Social background

No history is complete without enquiry about the patient's background. There are the obvious things such as the level of care and nutrition in children; dietary constraints which may cause specific deficiencies; and, in certain cases, questions about smoking habits, alcohol consumption and drug abuse, all of which call for a special degree of tact and non-judgemental enquiry.

Find out details about the patient's work practices, travel and recreation: could the disorder be due to

a particular repetitive activity in the home, at work or on the sports field? Is the patient subject to any unusual occupational strain? Has he or she travelled to another country where tuberculosis is common?

Finally, it is important to assess the patient's home circumstances and the level of support by family and friends. This will help to answer the question: 'What has the patient lost and what is he or she hoping to regain?'

EXAMINATION

In *A Case of Identity*, Sherlock Holmes has the following conversation with Dr Watson.

Watson: You appeared to read a good deal upon [your client] which was quite invisible to me.

Holmes: Not invisible but unnoticed, Watson.

Some disorders can be diagnosed at a glance: who would mistake the facial appearance of acromegaly or the hand deformities of rheumatoid arthritis for anything else? Nevertheless, even in these cases systematic examination is rewarding: it provides information about the patient's particular disability, as distinct from the clinicopathological diagnosis; it keeps reinforcing good habits; and, never to be forgotten, it lets the patient know that he or she has been thoroughly attended to.

The examination actually begins from the moment we set eyes on the patient. We observe his or her general appearance, posture and gait. Can you spot any distinctive feature: Knock-knees? Spinal curvature? A short limb? A paralysed arm? Does he or she appear to be in pain? Do their movements look natural? Do they walk with a limp, or use a stick? A telltale gait may suggest a painful hip, an unstable knee or a foot-drop. The clues are endless and the game is played by everyone (qualified or lay) at each new encounter throughout life. In the clinical setting the assessment needs to be more focused.

When we proceed to the structured examination, the patient must be suitably undressed; no mere rolling up of a trouser leg is sufficient. If one limb is affected, both must be exposed so that they can be compared.

We examine the good limb (for comparison), then the bad. There is a great temptation to rush in with both hands – a temptation that must be resisted. Only by proceeding in a purposeful, orderly way can we avoid missing important signs.

Alan Apley, who developed and taught the system used here, shied away from using long words where short ones would do the job. (He also used to say, 'I'm neither an inspector nor a manipulator, and I am definitely not a palpator.') Thus the traditional clinical routine, inspection, palpation, manipulation, was replaced by *look*, *feel*, *move*. With time, his teaching has been extended and we now add *test*, to include the special manoeuvres we employ in assessing neurological integrity and complex functional attributes.

Look

Abnormalities are not always obvious at first sight. A systematic, step-by-step process helps to avoid mistakes.

Shape and posture The first things to catch one's attention are the shape and posture of the limb or the body or the entire person who is being examined. Is the patient unusually thin or obese? Does the overall posture look normal? Is the spine straight or unusually curved? Are the shoulders level? Are the limbs normally positioned? It is important to look for deformity in three planes, and always compare the affected part with the normal side. In many joint disorders and in most nerve lesions the limb assumes a characteristic posture. In spinal disorders the entire torso may be deformed. Now look more closely for swelling or wasting – one often enhances the appearance of the other! Or is there a definite lump?

Skin Careful attention is paid to the colour, quality and markings of the skin. Look for bruising, wounds and ulceration. Scars are an informative record of the past – surgical archaeology, so to speak (see Figure 1.3). Colour reflects vascular status or pigmentation – for example, the pallor of ischaemia, the blueness of cyanosis, the redness of inflammation, or the dusky purple of an old bruise. Abnormal creases, unless due to fibrosis, suggest underlying deformity which is not always obvious; tight, shiny skin with no creases is typical of oedema or trophic change.

General survey Attention is initially focused on the symptomatic or most obviously abnormal area, but we

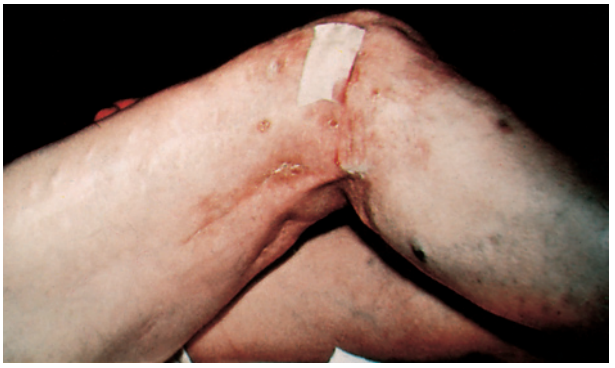


Figure 1.3 Look Scars often give clues to the previous history. The faded scar on this patient's thigh is an old operation wound – internal fixation of a femoral fracture. The other scars are due to postoperative infection; one of the sinuses is still draining.

must also look further afield. The patient complains of the joint that is hurting now, but we may see at a glance that several other joints are affected as well.

Feel

Feeling is exploring, not groping aimlessly. Know your anatomy and you will know where to feel for the landmarks; find the landmarks and you can construct a virtual anatomical picture in your mind's eye.

The skin Is it warm or cold; moist or dry; and is sensation normal?

The soft tissues Can you feel a lump; if so, what are its characteristics? Are the pulses normal?

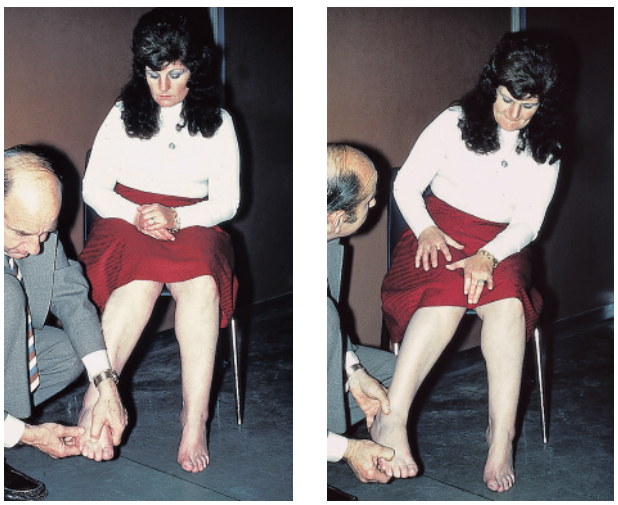
The bones and joints Are the outlines normal? Is the synovium thickened? Is there excessive joint fluid?

Tenderness Once you have a clear idea of the structural features in the affected area, feel gently for tenderness (Figure 1.4). Keep your eyes on the patient's face; a grimace will tell you as much as a grunt. Try to localize any tenderness to a particular structure; if you know precisely *where* the trouble is, you are halfway to knowing *what* it is.

Move

'Movement' covers several different activities: active movement, passive movement, abnormal or unstable movement, and provocative movement (see Figures 1.5 and 1.6).

Active movement Ask the patient to move without your assistance. This will give you an idea of the degree of mobility and whether it is painful or not. Active movement is also used to assess muscle power.



(a) (b)

Figure 1.4 Feeling for tenderness (a) The wrong way – there is no need to look at your fingers, you should know where they are. (b) It is much more informative to look at the patient's face!

Passive movement Here it is the examiner who moves the joint in each anatomical plane. Note whether there is any difference between the range of active and passive movement.

Range of movement is recorded in degrees, starting from zero which, by convention, is the neutral or anatomical position of the joint, and finishing where movement stops, due either to pain or to anatomical limitation. Describing the range of movement is often made to seem difficult. Words such as 'full', 'good', 'limited' and 'poor' are misleading. Always cite the range or span, from start to finish, in degrees. For example, 'knee flexion 0–140 degrees' means that the range of flexion is from zero (the knee absolutely straight) through an arc of 140 degrees (the leg making an acute angle with the thigh). Similarly, 'knee flexion 20–90 degrees' means that flexion begins at 20 degrees (i.e. the joint cannot extend fully) and continues only to 90 degrees.

For accuracy you can measure the range of movement with a goniometer, but with practice you will learn to estimate the angles by eye. Normal ranges of movement are shown in chapters dealing with individual joints. What is important is always to compare the symptomatic with the asymptomatic or normal side.

While testing movement, feel for crepitus. Joint crepitus is usually coarse and fairly diffuse; tenosynovial crepitus is fine and precisely localized to the affected tendon sheath.

Unstable movement This is movement which is inherently unphysiological. You may be able to shift or angulate a joint out of its normal plane of movement, thus demonstrating that the joint is unstable. Such abnormal movement may be obvious (e.g. a wobbly knee); often, though, you have to use special manoeuvres to pick up minor degrees of instability.

Provocative movement One of the most telling clues to diagnosis is reproducing the patient's symptoms by applying a specific, provocative movement. Shoulder pain due to impingement of the subacromial structures may be 'provoked' by moving the joint in a way that is calculated to produce such impingement; the patient recognizes the similarity between this pain and his or her daily symptoms. Likewise, a patient who has had a previous dislocation or subluxation can be vividly reminded of that event by stressing the joint in such a way that it again threatens to dislocate; indeed, merely starting the movement may be so distressing that the patient goes rigid with anxiety at the anticipated result – this is aptly called the *apprehension test.*

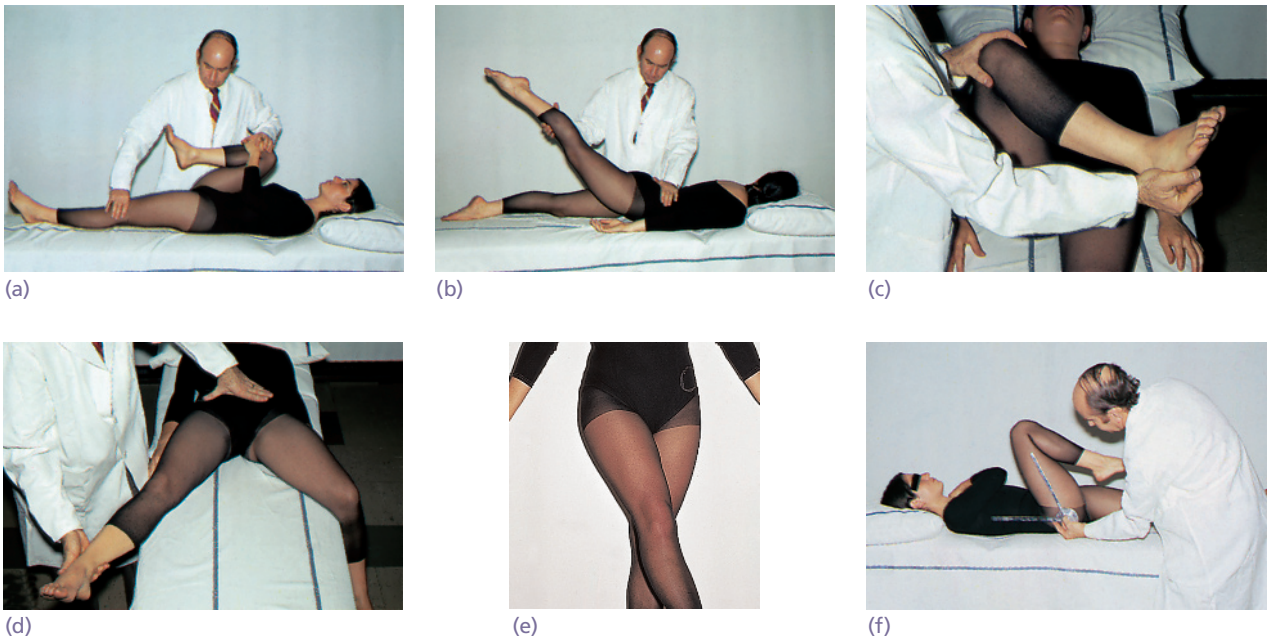


Figure 1.5 Testing for movement (a) Flexion, (b) extension, (c) rotation, (d) abduction, (e) adduction. The range of movement can be estimated by eye or measured accurately using a goniometer (f).

Test

The apprehension test referred to in the previous paragraph is one of several clinical tests that are used to elicit suspected abnormalities: some examples are *Thomas' test* for flexion deformity of the hip, *Trendelenburg's test* for instability of the hip, *McMurray's test* for a torn meniscus of the knee, *Lachman's test* for cruciate ligament instability and various tests for intra-articular fluid. These and others are described in the relevant chapters in Section 2. Tests for muscle tone, motor power, reflexes and various modes of sensibility are part and parcel of neurological examination, which is discussed later in this chapter.

Caveat

We recognize that the sequence set out here may sometimes have to be modified. We may need to 'move' before we 'look': an early scoliotic deformity of the spine often becomes apparent only when the patient bends forwards. The sequence may also have to be altered because a patient is in severe pain or disabled: you would not try to move a limb at all in someone with a suspected fracture when an X-ray can provide the answer. When examining a child, you may have to take your chances with look or feel or move whenever you can!

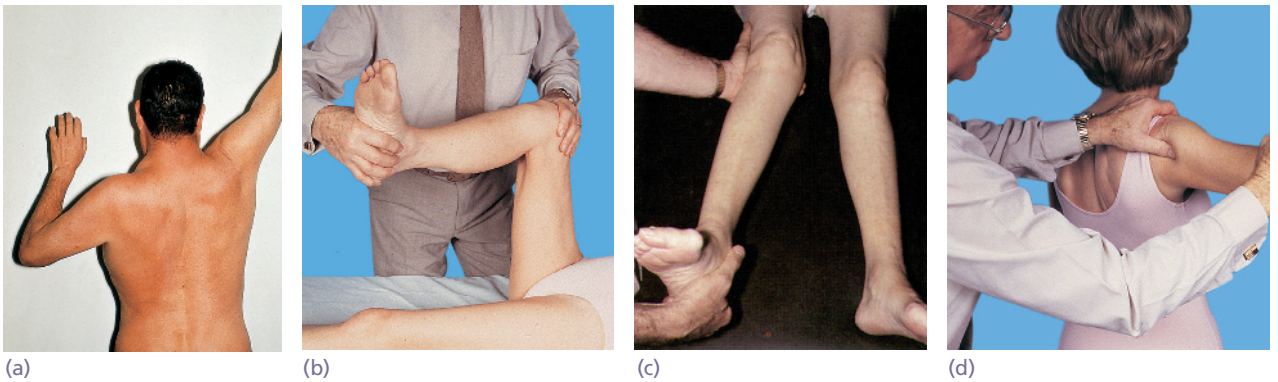


Figure 1.6 Move (a) Active movement – the patient moves the joint. The right shoulder is normal; the left has restricted active movement. (b) Passive movement – the examiner moves the joint. (c) Unstable movement – the joint can be moved across the normal planes of action, in this case demonstrating valgus instability of the right knee. (d) Provocative movement – the examiner moves (or manipulates) the joint so as to provoke the symptoms of impending pain or dislocation. Here he is reproducing the position in which an unstable shoulder is likely to dislocate.

Terminology

Colloquial terms such as front, back, upper, lower, inner aspect, outer aspect, bow legs, knock knees have the advantage of familiarity but are not applicable to every situation. Universally acceptable anatomical definitions are therefore necessary in describing physical attributes.

Bodily surfaces, planes and positions are always described in relation to the **anatomical position** – as if the person were standing erect, facing the viewer, legs together with the knees pointing directly forwards, and arms held by the sides with the palms facing forwards.

The principal **planes** of the body (Figure 1.7) are named **sagittal, coronal and transverse**; they define the direction across which the body (or body part) is viewed in any description. **Sagittal planes**, parallel to each other, pass vertically through the body from front to back; the **midsagittal** or **median plane** divides the body into right and left halves. **Coronal planes** are also orientated vertically, corresponding to a frontal view, at right angles to the sagittal planes; **transverse planes** pass horizontally across the body.

Anterior signifies the frontal aspect and **posterior** the rear aspect of the body or a body part. The terms **ventral** and **dorsal** are also used for the front and the back respectively. Note, though, that the use of these terms is somewhat confusing when it comes to the foot: here the upper surface is called the **dorsum** and the sole is called the **plantar surface**.

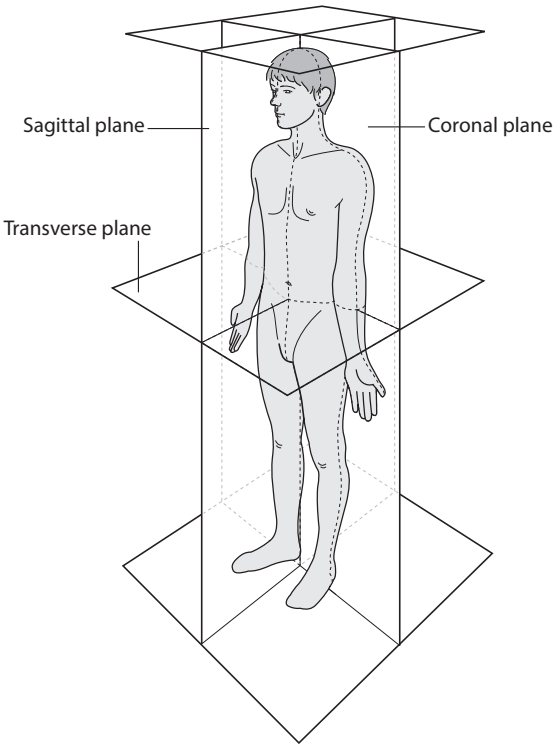


FIgure 1.7 Planes The principal planes of the body, as viewed in the anatomical position: sagittal, coronal and transverse.

Medial means facing towards the median plane or midline of the body, and **lateral** away from the median plane. These terms are usually applied to a limb, the clavicle or one half of the pelvis. Thus the inner aspect of the thigh lies on the medial side of the limb and the outer part of the thigh lies on the lateral side. We could also say that the little finger lies on the medial or **ulnar side** of the hand and the thumb on the lateral or **radial side** of the hand.

Proximal and **distal** are used mainly for parts of the limbs, meaning respectively the upper end and the lower end as they appear in the anatomical position. Thus the knee joint is formed by the distal end of the femur and the proximal end of the tibia.

Axial alignment describes the longitudinal arrangement of adjacent limb segments or parts of a single bone. The knees and elbows, for example, are normally angulated slightly outwards (**valgus**) while the opposite – 'bow legs' – is more correctly described as **varus** (see 'Physical variations and deformities' later in this chapter). Angulation in the middle of a long bone would always be regarded as abnormal.

Rotational alignment refers to the tortile arrangement of segments of a long bone (or an entire limb) around a single longitudinal axis. For example, in the anatomical position the patellae face forwards while the feet are turned slightly outwards; a marked difference in rotational alignment of the two legs is abnormal.

Flexion and extension are joint movements in the sagittal plane, most easily imagined in hinge joints like the knee, elbow and the joints of the fingers and toes. In elbows, knees, wrists and fingers, flexion means bending the joint and extension means straightening it. In shoulders and hips, flexion is movement in an anterior direction and extension is movement posteriorwards. In the ankle, flexion is also called **plantarflexion** (pointing the foot downwards) and extension is called **dorsiflexion** (drawing the foot upwards). Thumb movements are the most complicated and are described in Chapter 16.

Abduction and adduction are movements in the coronal plane, away from or towards the median plane. Not quite for the fingers and toes, though: here abduction and adduction mean away from and towards the longitudinal midline of the hand or foot!

Lateral rotation and medial rotation are twisting movements, outwards and inwards, around a longitudinal axis.

Pronation and supination are also rotatory movements, but the terms are applied only to movements of the forearm and the foot.

Circumduction is a composite movement made up of a rhythmic sequence of all the other movements. It is possible only for ball-and-socket joints such as the hip and shoulder.

Specialized movements such as opposition of the thumb, lateral flexion and rotation of the spine, and inversion or eversion of the foot, will be described in the relevant chapters.

NEUROLOGICAL EXAMINATION

If the symptoms include weakness or incoordination or a change in sensibility, or if they point to any disorder of the neck or back, a complete neurological examination of the related part is mandatory. Once again we follow a systematic routine, first looking at the general appearance, then assessing motor function (muscle tone, power and reflexes) and finally testing for sensory function (both skin sensibility and deep sensibility) (see Table 1.1 and Figure 1.8).

Table 1.1 Nerve root supply and actions of main muscle groups

Muscles/Muscle action	Nerve root supply
Sternomastoids	Spinal accessory C2, 3, 4
Trapezius	Spinal accessory C3, 4
Diaphragm	C3, 4, 5
Deltoid	C5, 6
Supra- and infraspinatus	C5, 6
Serratus anterior	C5, 6, 7
Pectoralis major	C5, 6, 7, 8
Elbow flexion extension	C5, 6 C7
Supination	C5, 6
Pronation	C6
Wrist extension flexion	C6, (7) C7, (8)
Finger extension flexion ab- and adduction	C7 C7, 8, T1 C8, T1
Hip flexion extension adduction abduction	L1, 2, 3 L5, S1 L2, 3, 4 L4, 5, S1
Knee extension flexion	L(2), 3, 4 L5, S1
Ankle dorsiflexion plantarflexion inversion eversion	L4, 5 S1, 2 L4, 5 L5, S1
Toe extension flexion abduction	L5 S1 S1, 2

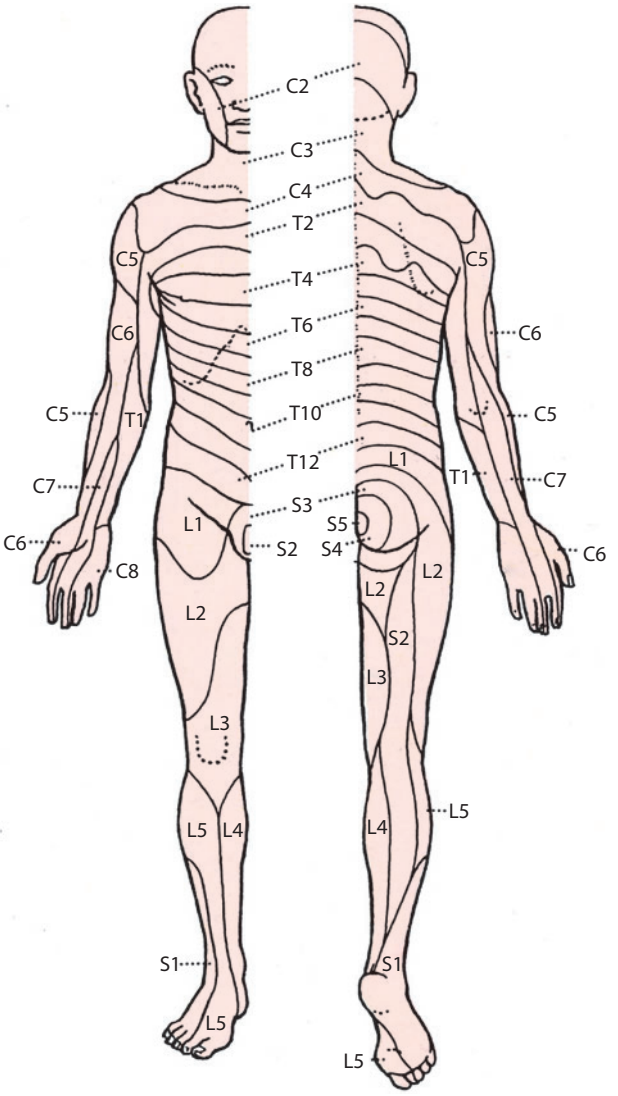


FIgure 1.8 Examination Dermatomes supplied by the spinal nerve roots.

Appearance

Some neurological disorders result in postures that are so characteristic as to be diagnostic at a glance: the claw hand of an ulnar nerve lesion; 'drop wrist' following radial nerve palsy (Figure 1.9); or the 'waiter's tip' deformity of the arm in brachial plexus injury. Usually, however, it is when the patient moves that we can best appreciate the type and extent of motor disorder: the dangling arm following a brachial plexus injury; the flail lower limb of poliomyelitis; the symmetrical paralysis of spinal cord lesions; the characteristic drop-foot gait following sciatic or peroneal nerve damage; and the jerky, 'spastic' movements of cerebral palsy.

Concentrating on the affected part, we look for trophic changes that signify loss of sensibility: the smooth, hairless skin that seems to be stretched too tight; atrophy of the fingertips and the nails; scars

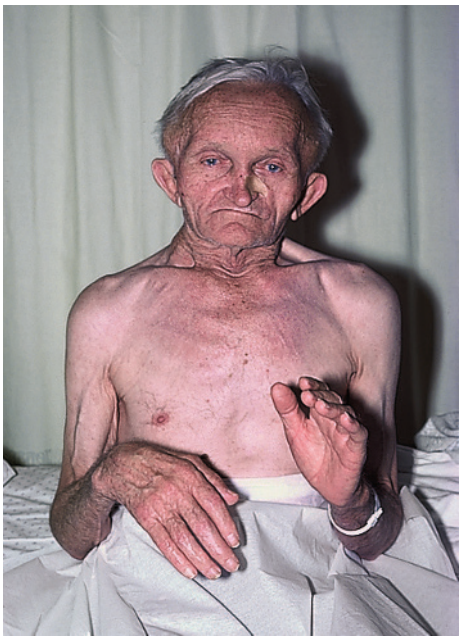


FIgure 1.9 Posture Posture is often diagnostic. This patient's 'drop wrist' – typical of a radial nerve palsy – is due to carcinomatous infiltration of the supraclavicular lymph nodes on the right.

that tell of accidental burns; and ulcers that refuse to heal. Muscle wasting is important: if localized and asymmetrical, it may suggest dysfunction of a specific motor nerve.

Muscle tone

Tone in individual muscle groups is tested by moving the nearby joint to stretch the muscle. Increased tone (spasticity) is characteristic of upper motor neuron disorders such as cerebral palsy and stroke. It must not be confused with rigidity (the 'lead-pipe' or 'cogwheel' effect) which is seen in Parkinson's disease. Decreased tone (flaccidity) is found in lower motor neuron lesions; for example, poliomyelitis. Muscle power is diminished in all three states; it is important to recognize that a 'spastic' muscle may still be weak.

Power

Motor function is tested by having the patient perform movements that are normally activated by specific nerves. We may learn even more about composite movements by asking the patient to perform specific tasks, such as holding a pen, gripping a rod, doing up a button or picking up a pin.

Testing for power is not as easy as it sounds; the difficulty is making ourselves understood. The simplest way is to place the limb in the 'test' position, then ask the patient to hold it there as firmly as possible and resist any attempt to change that position. The normal limb is examined first, then the affected limb, and the two are compared. Finer muscle actions, such as those of the thumb and fingers, may be reproduced by first demonstrating the movement yourself, then testing it in the unaffected limb, and then in the affected one.

Muscle power is usually graded on the Medical Research Council scale:

Grade 0	No movement
Grade 1	Only a flicker of movement
Grade 2	Movement with gravity eliminated
Grade 3	Movement against gravity
Grade 4	Movement against resistance
Grade 5	Normal power

It is important to recognize that muscle weakness may be due to muscle disease rather than nerve disease. In muscle disorders the weakness is usually more widespread and symmetrical, and sensation is normal.

Tendon reflexes

A deep tendon reflex is elicited by rapidly stretching the tendon near its insertion. A sharp tap with the tendon hammer does this well; but all too often this is performed with a flourish and with such force that the finer gradations of response are missed. It is better to employ a series of taps, starting with the most forceful and reducing the force with each successive tap until there is no response. Comparing the two sides in this way, we can pick up fine differences showing that a reflex is 'diminished' rather than 'absent'. In the upper limb we test biceps, triceps and brachioradialis; and in the lower limb the patellar and Achilles tendons.

The tendon reflexes are monosynaptic segmental reflexes; that is, the reflex pathway takes a 'short cut' through the spinal cord at the segmental level. Depression or absence of the reflex signifies interruption of the pathway at the posterior nerve root, the anterior horn cell, the motor nerve root or the peripheral nerve. It is a reliable pointer to the segmental level of dysfunction: thus, a depressed biceps jerk suggests pressure on the fifth or sixth cervical (C5 or C6) nerve roots while a depressed ankle jerk signifies a similar abnormality at the first sacral level (S1). An unusually brisk reflex, on the other hand, is characteristic of an upper motor neuron disorder (e.g. cerebral palsy, a stroke or injury to the spinal cord); the lower motor neuron is released from the normal central inhibition and there is an exaggerated response to tendon stimulation. This may manifest as ankle clonus: a sharp upward jerk on the foot (dorsiflexion) causes a repetitive, 'clonic' movement of the foot; similarly, a sharp downward push on the patella may elicit patellar clonus.

Superficial reflexes

The superficial reflexes are elicited by stroking the skin at various sites to produce a specific muscle contraction; the best known are the abdominal (T7–T12), cremasteric (L1, 2) and anal (S4, 5) reflexes. These are corticospinal (upper motor neuron) reflexes. Absence of the reflex indicates an upper motor neuron lesion (usually in the spinal cord) above that level.

The plantar reflex

Forceful stroking of the sole normally produces flexion of the toes (or no response at all). An extensor response (the big toe extends while the others remain in flexion) is characteristic of upper motor neuron disorders. This is the *Babinski sign* – a type of withdrawal reflex which is present in young infants and normally disappears after the age of 18 months.

Sensibility

Sensibility to touch and to pinprick may be increased (hyperaesthesia) or unpleasant (dysaesthesia) in certain irritative nerve lesions. More often, though, it is diminished (hypoaesthesia) or absent (anaesthesia), signifying pressure on or interruption of a peripheral nerve, a nerve root or the sensory pathways in the spinal cord. The area of sensory change can be mapped out on the skin and compared with the known segmental or dermatomal pattern of innervation. If the abnormality is well defined, it is an easy matter to establish the level of the lesion, even if the precise cause remains unknown.

Brisk percussion along the course of an injured nerve may elicit a tingling sensation in the distal distribution of the nerve (*Tinel's sign*). The point of hypersensitivity marks the site of abnormal nerve sprouting: if it progresses distally at successive visits, this signifies regeneration; if it remains unchanged, this suggests a local neuroma.

Tests for temperature recognition and two-point discrimination (the ability to recognize two touch-points a few millimetres apart) are also used in the assessment of peripheral nerve injuries.

Deep sensibility can be examined in several ways. In the vibration test a sounded tuning fork is placed over a peripheral bony point (e.g. the medial malleolus or the head of the ulna); the patient is asked if he or she can feel the vibrations and to say when they disappear. By comparing the two sides, differences can be noted. Position sense is tested by asking the patient to find certain points on the body with the eyes closed – for example, touching the tip of the nose with the forefinger. The sense of joint posture is tested by grasping the big toe and placing it in different positions of flexion and extension. The patient (whose eyes are closed) is asked to say whether it is 'up' or 'down'. Stereognosis, the ability to recognize shape and texture by feel alone, is tested by giving the patient (again with eyes closed) a variety of familiar objects to hold and asking him or her to name each object.

The pathways for deep sensibility run in the posterior columns of the spinal cord. Disturbances are therefore found in peripheral neuropathies and in spinal cord lesions such as posterior column injuries or tabes dorsalis. The sense of balance is also carried in the posterior columns. This can be tested by asking the patient to stand upright with his or her eyes closed; excessive body sway is abnormal (*Romberg's sign*).

Cortical and cerebellar function

A staggering gait may imply an unstable knee – or a disorder of the spinal cord or cerebellum. If there is no musculoskeletal abnormality to account for the sign, a full examination of the central nervous system will be necessary.

EXAMINING INFANTS AND CHILDREN

Paediatric practice requires special skills. You may have no first-hand account of the symptoms; a baby screaming with pain will tell you very little, and over-anxious parents will probably tell you too much. When examining the child, be flexible. If he or she is moving a particular joint, take your opportunity to examine movement then and there. You will learn much more by adopting methods of play than by applying a rigid system of examination. And leave any test for tenderness until last!

Infants and small children

The baby should be undressed, in a warm room, and placed on the examining couch. Look carefully for birthmarks, deformities and abnormal movements – or absence of movement. If there is no urgency or distress, take time to examine the head and neck, including facial features which may be characteristic of specific dysplastic syndromes. The back and limbs are then examined for abnormalities of position or shape.

Examining for joint movement can be difficult. Active movements can often be stimulated by gently stroking the limb. When testing for passive mobility, be careful to avoid frightening or hurting the child. In the neonate, and throughout the first two years of life, examination of the hips is mandatory, even if the child appears to be normal. This is to avoid missing the subtle signs of developmental dysplasia of the hips (DDH) at the early stage when treatment is most

Table 1.2 Normal developmental milestones

Age	Normal developmental milestone(s)
Newborn	Grasp reflex present Morrow reflex present
3–6 months	Holds head up unsupported
6–9 months	Able to sit up
9–12 months	Crawling Standing up
9–18 months	Walking
18–24 months	Running

effective. It is also important to assess the child's general development by testing for the normal milestones which are expected to appear during the first two years of life (Table 1.2).

Older children

Most children can be examined in the same way as adults, though with different emphasis on particular physical features. Posture and gait are very important; subtle deviations from the norm may herald the appearance of serious abnormalities such as scoliosis or neuromuscular disorders, while more obvious 'deformities' such as knock knees and bow legs may be no more than transient stages in normal development; similarly with mild degrees of 'flat feet' and 'pigeon toes'. More complex variations in posture and gait patterns, when the child sits and walks with the knees turned inwards (medially rotated) or outwards (laterally rotated) are usually due to anteversion or retroversion of the femoral necks, sometimes associated with compensatory rotational 'deformities' of the femora and tibiae. Seldom need anything be done about this; the condition usually improves as the child approaches puberty and only if the gait is very awkward would one consider performing corrective osteotomies of the femora.

PHYSICAL VARIATIONS AND DEFORMITIES

Joint laxity

Children's joints are much more mobile than those of most adults, allowing them to adopt postures that would be impossible for their parents. An unusual degree of joint mobility can also be attained by adults willing to submit to rigorous exercise and practice, as witness the performances of professional dancers and athletes, but in most cases, when the exercises stop, mobility gradually reverts to the normal range.

Persistent generalized joint hypermobility occurs in about 5% of the population and is inherited as a simple Mendelian dominant (Figure 1.10). Those affected

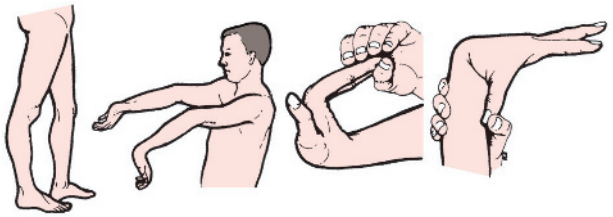


Figure 1.10 Tests for joint hypermobility
Hyperextension of knees and elbows; metacarpophalangeal joints extending to 90 degrees'; thumb able to touch forearm.

describe themselves as being 'double-jointed': they can hyperextend their metacarpophalangeal joints beyond a right angle, hyperextend their elbows and knees and bend over with knees straight to place their hands flat on the ground; some can even 'do the splits' or place their feet behind their neck!

It is doubtful whether these individuals should be considered 'abnormal'. However, epidemiological studies have shown that they do have a greater than usual tendency to recurrent dislocation (e.g. of the shoulder or patella). Some experience recurrent episodes of aching around the larger joints; however, there is no convincing evidence that hypermobility by itself predisposes to osteoarthritis.

Generalized hypermobility is not usually associated with any obvious disease, but severe laxity is a feature of certain rare connective tissue disorders such as Marfan's syndrome, Ehlers–Danlos syndrome, Larsen's disease and osteogenesis imperfecta.

Deformity

The boundary between variations of the normal and physical deformity is blurred. Indeed, in the development of species, what at one point of time might have been seen as a deformity could over the ages have turned out to be so advantageous as to become essential for survival. So too in humans. The word 'deformity' is derived from the Latin for 'misshapen', but the range of 'normal shape' is so wide that variations should not automatically be designated as deformities, and some undoubted 'deformities' are not necessarily pathological; for example, the generally accepted cut-off points for 'abnormal' shortness or tallness are arbitrary and people who in one population might be considered abnormally short or abnormally tall could, in other populations, be seen as quite ordinary. However, if one leg is short and the other long, no one would quibble with the use of the word 'deformity'!

Specific terms are used to describe the 'position' and 'shape' of the bones and joints. Whether, in any particular case, these amount to 'deformity' will be determined by additional factors such as the extent

to which they deviate from the norm, symptoms to which they give rise, the presence or absence of instability and the degree to which they interfere with function.

Varus and valgus It seems pedantic to replace 'bow legs' and 'knock knees' with 'genu varum' and 'genu valgum', but comparable colloquialisms are not available for deformities of the elbow, hip or big toe; and, besides, the formality is justified by the need for clarity and consistency. Varus means that the part distal to the joint in question is displaced towards the median plane, valgus away from it (Figure 1.11).

Kyphosis and lordosis Seen from the side, the normal spine has a series of curves: convex posteriorly in the thoracic region (kyphosis), and convex anteriorly in the cervical and lumbar regions (lordosis). Excessive curvature constitutes kyphotic or lordotic deformity (also sometimes referred to as hyperkyphosis and hyperlordosis). Colloquially speaking, excessive thoracic kyphosis is referred to as 'round-shouldered'.

Scoliosis Seen from behind, the spine is straight. Any curvature in the coronal plane is called scoliosis. The position and direction of the curve are specified by terms such as thoracic scoliosis, lumbar scoliosis, convex to the right, concave to the left, etc.

Postural deformity A postural deformity is one which the patient can, if properly instructed, correct voluntarily: e.g. thoracic 'kyphosis' due to slumped shoulders. Postural deformity may also be caused by temporary muscle spasm.

Structural deformity A deformity which results from a permanent change in anatomical structure cannot be voluntarily corrected. It is important to distinguish postural scoliosis from structural (fixed) scoliosis. The former is non-progressive and benign; the latter is usually progressive and may require treatment.

'Fixed deformity' This term is ambiguous. It seems to mean that a joint is deformed and unable to move but this is not so. It means that one particular movement cannot be completed. Thus the knee may be able to flex fully but not extend fully – at the limit of its extension it is still 'fixed' in a certain amount of flexion. This would be called a 'fixed flexion deformity'.

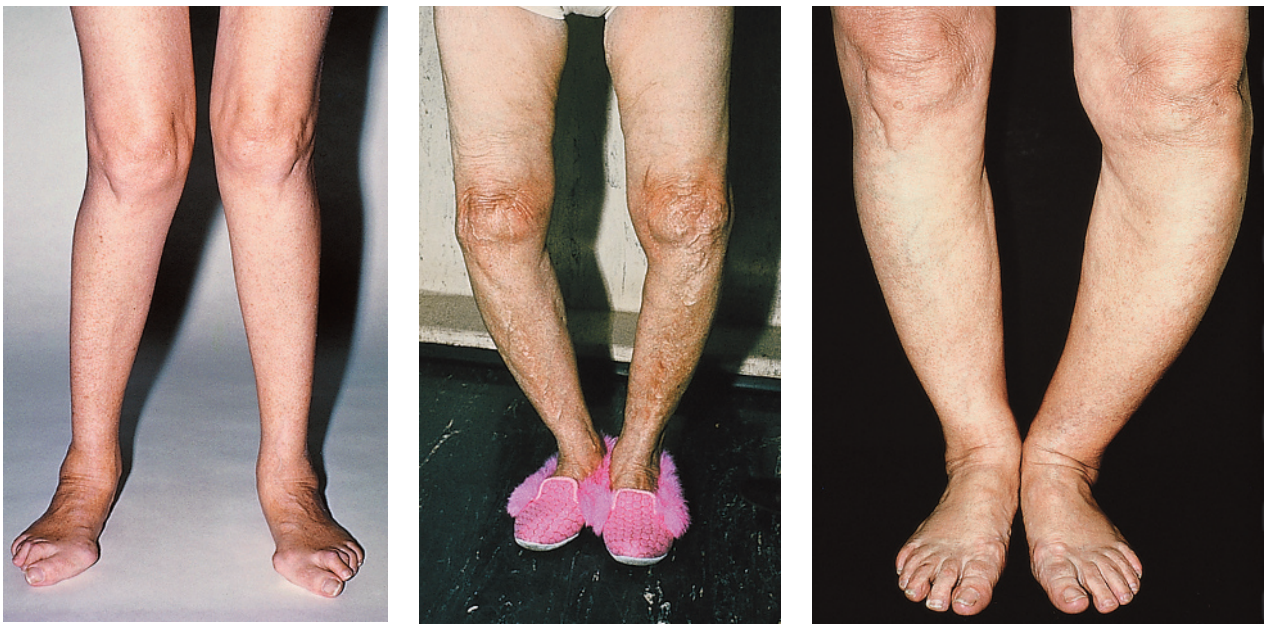
CAUSES OF JOINT DEFORMITY

There are six basic causes of joint deformity.

Contracture of the overlying skin This is seen typically when there is severe scarring across the flexor aspect of a joint, e.g. due to a burn or following surgery.

Contracture of the subcutaneous fascia The classical example is Dupuytren's contracture in the palm of the hand.

Muscle contracture Fibrosis and contracture of muscles that cross a joint will cause a fixed deformity of the joint. This may be due to deep infection or fibrosis following ischaemic necrosis (Volkmann's ischaemic contracture).



(a) (b) (c)

FIgure 1.11 Varus and valgus (a) Valgus knees in a patient with rheumatoid arthritis. The toe joints are also valgus. (b) Varus knees due to osteoarthritis. (c) Another varus knee? No – the deformity here is in the left tibia due to Paget's disease.

Muscle imbalance Unbalanced muscle weakness or spasticity will result in joint deformity which, if not corrected, will eventually become fixed. This is seen most typically in poliomyelitis and cerebral palsy. Tendon rupture, likewise, may cause deformity.

Joint instability Any unstable joint will assume a 'deformed' position when subjected to force.

Joint destruction Trauma, infection or arthritis may destroy the joint and lead to severe deformity.

CAUSES OF BONE DEFORMITY

Bone deformities in small children are usually due to genetic or developmental disorders of cartilage and bone growth; some can be diagnosed *in utero* by special imaging techniques (e.g. achondroplasia); some become apparent when the child starts to walk, or later still during one of the growth spurts (e.g. hereditary multiple exostosis); and some only in early adulthood (e.g. multiple epiphyseal dysplasia). There are a myriad genetic disorders affecting the skeleton, yet any one of these conditions is rare. The least unusual of them are described in Chapter 8.

Acquired deformities in children may be due to fractures involving the physis (growth plate); ask about previous injuries. Other causes include rickets, endocrine disorders, malunited diaphyseal fractures and tumours.

Acquired deformities of bone in adults are usually the result of previous malunited fractures. However, causes such as osteomalacia, bone tumours and Paget's disease should always be considered.

Bony lumps

A bony lump may be due to faulty development, injury, inflammation or a tumour. Although X-ray examination is essential, the clinical features can be highly informative (for example, see Figure 1.12).

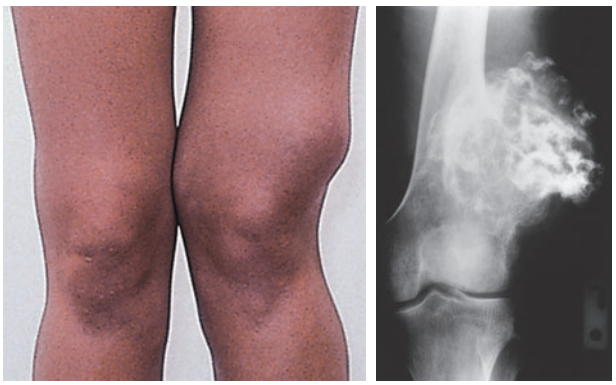


FIgure 1.12 Bony lumps The lump above the left knee is hard, well defined and not increasing in size. The clinical diagnosis of cartilage-capped exostosis (osteochondroma) is confirmed by the X-rays.

Size A large lump attached to bone, or a lump that is getting bigger, is nearly always a tumour.

Site A lump near a joint is most likely to be a tumour (benign or malignant); a lump in the shaft may be fracture callus, inflammatory new bone or a tumour. A benign tumour has a well-defined margin; malignant tumours, inflammatory lumps and callus have a vague edge.

Consistency A benign tumour feels bony and hard; malignant tumours often give the impression that they can be indented.

Tenderness Lumps due to active inflammation, recent callus or a rapidly growing sarcoma are tender.

Multiplicity Multiple bony lumps are uncommon: they occur in hereditary multiple exostosis and in Ollier's disease.

Joint stiffness

The term 'stiffness' covers a variety of limitations. We consider three types of stiffness in particular: (1) all movements absent; (2) all movements limited; (3) one or two movements limited.

All movements absent Surprisingly, although movement is completely blocked, the patient may retain such good function that the restriction goes unnoticed until the joint is examined. Surgical fusion is called 'arthrodesis'; pathological fusion is called 'ankylosis'. Acute suppurative arthritis typically ends in bony ankylosis; tuberculous arthritis heals by fibrosis and causes fibrous ankylosis – not strictly a 'fusion' because there may still be a small jog of movement.

All movements limited After severe injury, movement may be limited as a result of oedema and bruising. Later, adhesions and loss of muscle extensibility may perpetuate the stiffness.

With active inflammation all movements are restricted and painful and the joint is said to be 'irritable'. In acute arthritis spasm may prevent all but a few degrees of movement.

In osteoarthritis the capsule fibroses and movements become increasingly restricted, but pain occurs only at the extremes of motion.

Some movements limited When one particular movement suddenly becomes blocked, the cause is usually mechanical. Thus a torn and displaced meniscus may prevent extension of the knee but not flexion.

Bone deformity may alter the arc of movement, such that it is limited in one direction (loss of abduction in coxa vara is an example) but movement in the opposite direction is full or even increased.

These are all examples of 'fixed deformity'.

DIAGNOSTIC IMAGING

The map is not the territory

Alfred Korzybski

Plain film radiography

Plain film X-ray examination is over 100 years old. Notwithstanding the extraordinary technical advances of the last few decades, it remains the most useful method of diagnostic imaging. Whereas other methods may define an inaccessible anatomical structure more accurately, or may reveal some localized tissue change, the plain film provides information simultaneously on the size, shape, tissue 'density' and bone architecture – characteristics which, taken together, will usually suggest a diagnosis, or at least a range of possible diagnoses.

The radiographic image

X-rays are produced by firing electrons at high speed onto a rotating anode. The resulting beam of X-rays is attenuated by the patient's soft tissues and bones, casting what are effectively 'shadows' which are displayed as images on an appropriately sensitized plate or stored as digital information which is then available to be transferred throughout the local information technology (IT) network. See Figure 1.13.

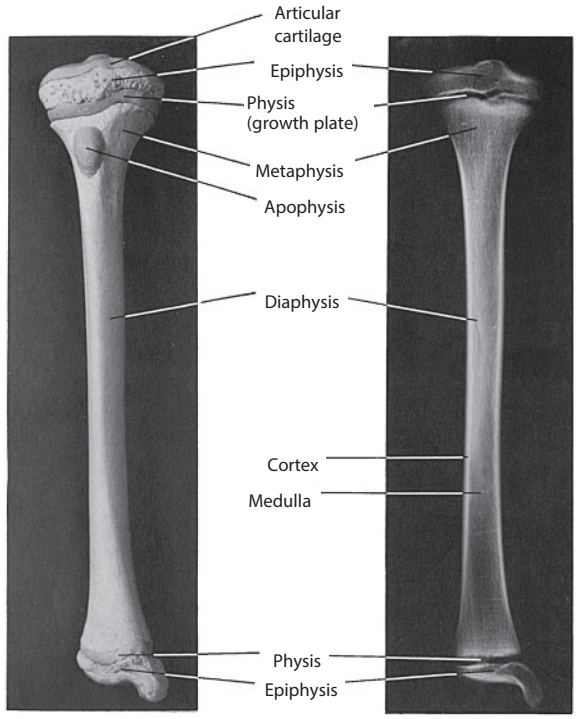


FIgure 1.13 The radiographic image X-ray of an anatomical specimen to show the appearance of various parts of the bone in the X-ray image.

The more dense and impenetrable the tissue, the greater the X-ray attenuation and therefore the more blank, or white, the image that is captured. Thus, a metal implant appears intensely white, bone less so and soft tissues in varying shades of grey depending on their 'density'. Cartilage, which causes little attenuation, appears as a dark area between adjacent bone ends; this 'gap' is usually called the joint space, though of course it is not a space at all, merely a radiolucent zone filled with cartilage. Other 'radiolucent' areas are produced by fluid-filled cysts in bone.

One bone overlying another (e.g. the femoral head inside the acetabular socket) produces superimposed images; any abnormality seen in the resulting combined image could be in either bone, so it is important to obtain several images from different projections in order to separate the anatomical outlines. Similarly, the bright image of a metallic foreign body superimposed upon that of, say, the femoral condyles could mean that the foreign body is in front of, inside or behind the bone. A second projection, at right angles to the first, will give the answer.

Picture Archiving and Communication System (PACS) This is the system whereby all digitally coded images are filed, stored and retrieved to enable the images to be sent to work stations throughout the hospital, to other hospitals or to the Consultant's personal computer.

Radiographic interpretation

Although *radiograph* is the correct word for the plain image which we address, in the present book we have chosen to retain the old-fashioned term 'X-ray', which has become entrenched by long usage. The process of interpreting this image should be as methodical as clinical examination. It is seductively easy to be led astray by some flagrant anomaly; systematic study is the only safeguard. A convenient sequence for examination is: *the patient – the soft tissues – the bones – the joints.*

THE PATIENT

Make sure that the name on the film is that of your patient; mistaken identity is a potent source of error. The clinical details are important; it is surprising how much more you can see on the X-ray when you know the background. Similarly, when requesting an X-ray examination, give the radiologist enough information from the patient's history and the clinical findings to help in guiding his or her thoughts towards the diagnostic possibilities and options. For example, when considering a malignant bone lesion, simply knowing the patient's age may provide an important clue: under the age of 10 it is most likely to be a Ewing's sarcoma; between 10 and 20 years it is more likely to

be an osteosarcoma; and over the age of 50 years it is likely to be a metastatic deposit.

THE SOFT TISSUES

Generalized change Muscle planes are often visible and may reveal wasting or swelling. Bulging outlines around a hip, for example, may suggest a joint effusion; and soft-tissue swelling around interphalangeal joints may be the first radiographic sign of rheumatoid arthritis. Tumours tend to displace fascial planes, whereas infection tends to obliterate them.

Localized change Is there a mass, soft tissue calcification, ossification, gas (from penetrating wound or gas-forming organism) or the presence of a radio-opaque foreign body?

THE BONES

Shape The bones are well enough defined to allow one to check their general anatomy and individual shape (Figure 1.14). For example, for the spine, look at the overall vertebral alignment, then at the disc spaces, and then at each vertebra separately, moving from the body to the pedicles, the facet joints and finally the spinous appendages. For the pelvis, see if the shape is symmetrical with the bones in their normal positions, then look at the sacrum, the two innominate bones, the pubic rami and the ischial tuberosities, then the femoral heads and the upper ends of the femora, always comparing the two sides.

Generalized change Take note of changes in bone 'density' (osteopaenia or osteosclerosis). Is there abnormal trabeculation, as in Paget's disease (Figure 1.15a)? Are there features suggestive of diffuse metastatic infiltration, either sclerotic or lytic? Other polyostotic lesions include fibrous dysplasia, histiocyotis, multiple exostosis and Paget's disease. With aggressive-looking polyostotic lesions, think of metastases (including myeloma and lymphoma) and also multifocal infection. By contrast, most primary tumours are monostotic.

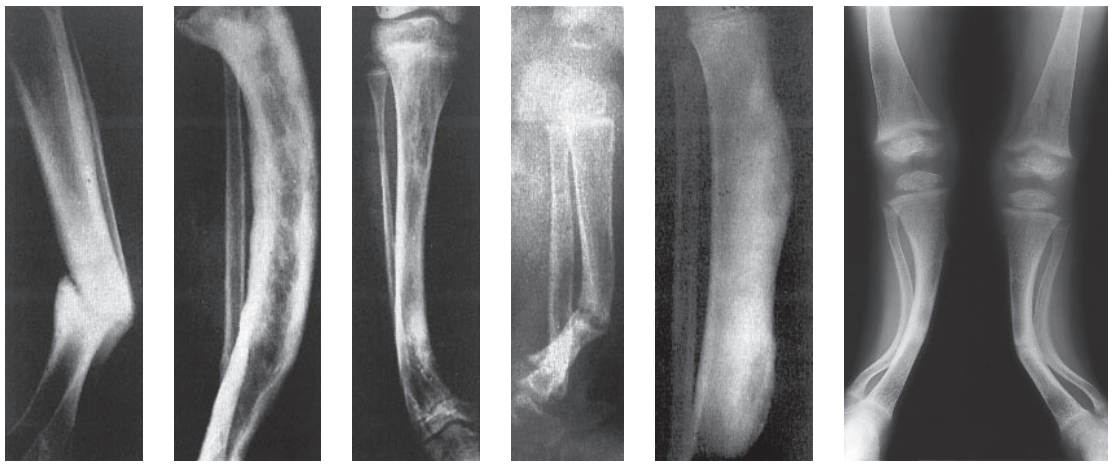
Localized change Focal abnormalities should be approached in the same way as one would conduct a clinical analysis of a soft tissue abnormality. Start describing the abnormality from the centre and move outwards. Determine the lesion's size, site, shape, density and margins, as well as adjacent periosteal changes and any surrounding soft tissue changes. Remember that benign lesions are usually well defined with sclerotic margins (Figure 1.15b) and a smooth periosteal reaction. Ill-defined areas with permeative bone destruction (Figure 1.15c) and irregular or speculated periosteal reactions (Figure 1.15d) suggest an aggressive lesion such as infection or a malignant tumour.

THE JOINTS

The radiographic 'joint' consists of the articulating bones and the 'space' between them.

The 'joint space' The joint space is, of course, illusory; it is occupied by a film of synovial fluid plus radiolucent articular cartilage which varies in thickness from 1 mm or less (the carpal joints) to 6 mm (the knee). It looks much wider in children than in adults because much of the epiphysis is still cartilaginous and therefore radiolucent. Lines of increased density within the radiographic articular 'space' may be due to calcification of the cartilage or menisci (chondrocalcinosis). Loose bodies, if they are radio-opaque, appear as rounded patches overlying the normal structures.

Shape Note the general orientation of the joint and the congruity of the bone ends (actually the subarticular bone plates), if necessary comparing the



(a) (b) (c) (d) (e) (f)

FIgure 1.14 X-rays – bent bones (a) Malunited fracture; (b) Paget's disease; (c) dyschondroplasia; (d) congenital pseudarthrosis; (e) syphilitic sabre tibia; (f) osteogenesis imperfecta.

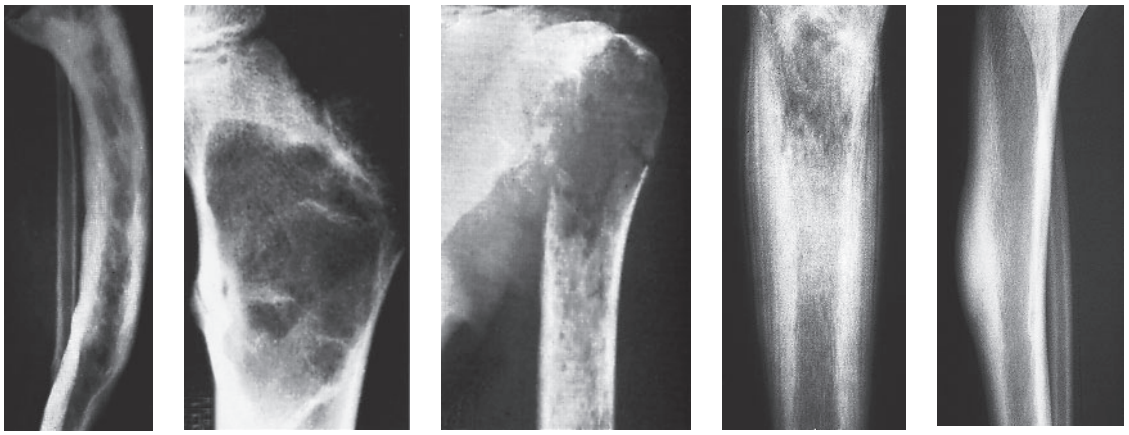


FIgure 1.15 X-rays – important features to look for (a) *General shape and appearance*, in this case the cortices are thickened and the bone is bent (Paget's disease). (b,c) *Interior density*, a vacant area may represent a true cyst (b), or radiolucent material infiltrating the bone, like the metastatic tumour in (c). (d) *Periosteal reaction*, typically seen in healing fractures, bone infection and malignant bone tumours – as in this example of Ewing's sarcoma. Compare this with the smooth periosteal new bone formation shown in (e).

abnormal with the normal opposite side. Then look for narrowing or asymmetry of the joint 'space': narrowing signifies loss of hyaline cartilage and is typical of infection, inflammatory arthropathies and osteoarthritis. Further stages of joint destruction are revealed by irregularity of the radiographically visible bone ends and radiolucent cysts in the subchondral bone. Bony excrescences at the joint margins (osteophytes) are typical of osteoarthritis.

Erosions Look for associated bone erosions. The position of erosions and symmetry help to define various types of arthropathy. In rheumatoid arthritis and psoriasis the erosions are periarticular (at the bare area where the hyaline cartilage covering the joint has ended and the intracapsular bone is exposed to joint fluid). In gout the erosions are further away from the articular surfaces and are described as juxta-articular. Rheumatoid arthritis is classically symmetrical and predominantly involves the metacarpophalangeal and proximal interphalangeal joints in both hands. The erosions in psoriasis are usually more feathery with ill-defined new bone at their margins. Ill-defined erosions suggest active synovitis whereas corticated erosions indicate healing and chronicity.

Diagnostic associations

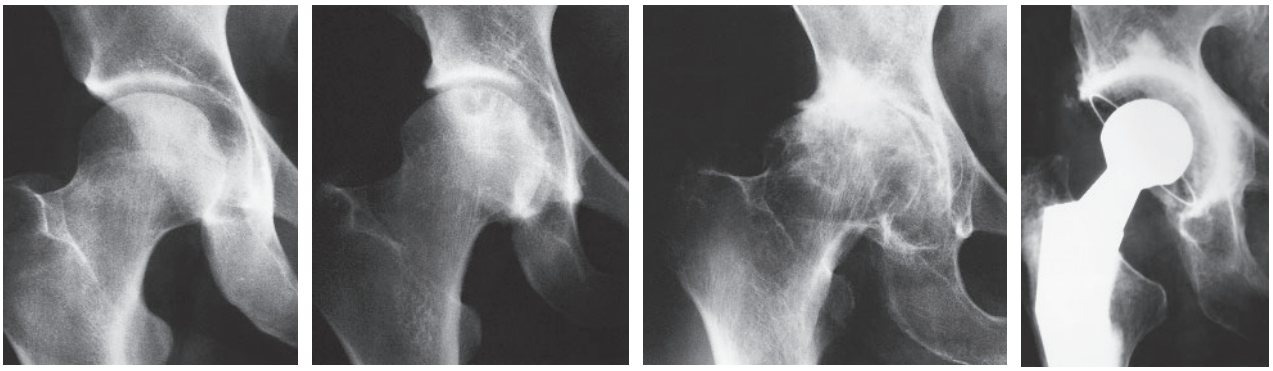
However carefully the individual X-ray features are observed, the diagnosis will not leap ready-made off the X-ray plate. Even a fracture is not always obvious. It is the pattern of abnormalities that counts: if you see one feature that is suggestive, look for others that are commonly associated.

- Narrowing of the joint space + subchondral sclerosis and cysts + osteophytes = osteoarthritis (Figure 1.16).
- Narrowing of the joint space + osteoporosis + periarticular erosions = inflammatory arthritis. Add to this the typical distribution, more or less symmetrically in the proximal joints of both hands, and you must think of rheumatoid arthritis.
- Bone destruction + periosteal new bone formation = infection or malignancy until proven otherwise.
- Remember: the next best investigation is either the previous radiograph or the subsequent follow-up radiograph. Sequential films demonstrate either progression of changes in active pathology or status quo in long-standing conditions.

Limitations of conventional radiography

Conventional radiography involves exposure of the patient to ionizing radiation, which under certain circumstances can lead to radiation-induced cancer. The Ionizing Radiation Medical Exposure Regulations (IRMER) 2000 are embedded in European Law, requiring all clinicians to justify any exposure of the patient to ionizing radiation. It is a criminal offence to breach these regulations. Ionizing radiation can also damage a developing fetus, especially in the first trimester.

As a diagnostic tool, conventional radiography provides poor soft-tissue contrast: for example, it cannot distinguish between muscles, tendons, ligaments and hyaline cartilage. Ultrasound (US), computed tomography (CT) and magnetic resonance imaging (MRI) are now employed to complement plain X-ray examination. However, in parts of the world where these techniques are not available, some modifications of plain radiography still have a useful role.



(a) (b) (c) (d)

Figure 1.16 Plain X-rays of the hip Stages in the development of osteoarthritis (OA). (a) Normal hip: anatomical shape and position, with joint 'space' (articular cartilage) fully preserved. (b) Early OA, showing joint space slightly decreased and a subarticular cyst in the femoral head. (c) Advanced OA: joint space markedly decreased; osteophytes at the joint margin. (d) Hip replacement: the cup is radiolucent but its position is shown by a circumferential wire marker. Note the differing image 'densities': (1) the metal femoral implant; (2) the polyethylene cup (radiolucent); (3) acrylic cement impacted into the adjacent bone.

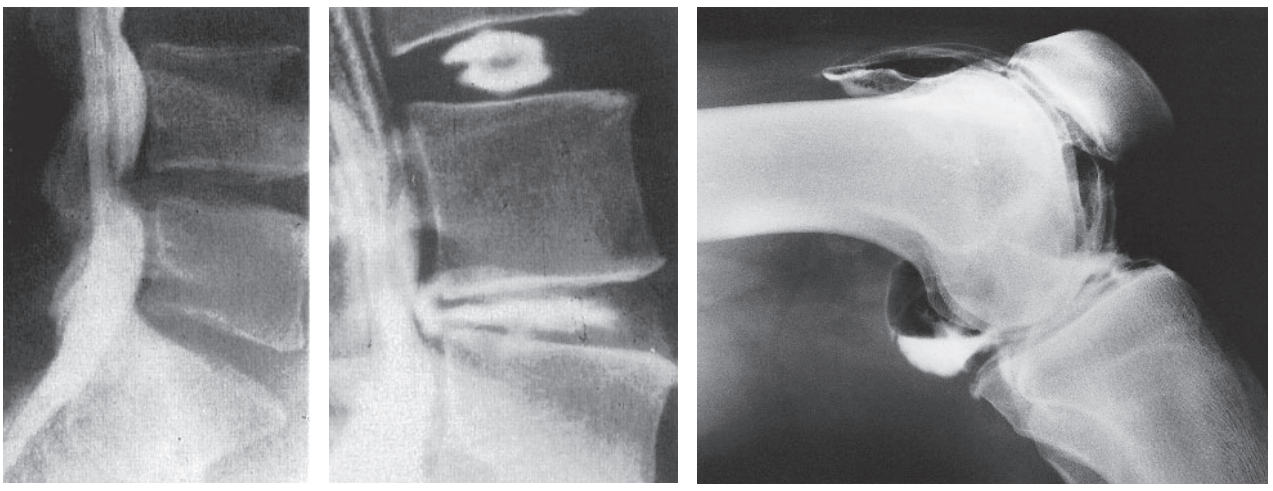
X-RAYS USING CONTRAST MEDIA

Substances that alter X-ray attenuation characteristics can be used to produce images which contrast with those of the normal tissues. The contrast media used in orthopaedics are mostly iodine-based liquids which can be injected into sinuses, joint cavities or the spinal theca (Figure 1.17). Air or gas also can be injected into joints to produce a 'negative image' outlining the joint cavity.

Oily iodides are not absorbed and maintain maximum concentration after injection. However, because they are non-miscible, they do not penetrate well into all the nooks and crannies. They are also tissue irritants, especially if used intrathecally and are now rarely used as they have been shown to cause adhesive arachnoiditis. Ionic, water-soluble iodides permit much more detailed imaging and, although also somewhat irritant and neurotoxic, are rapidly absorbed and excreted.

Sinography

Sinography is the simplest form of contrast radiography. The medium (usually one of the ionic water-soluble compounds) is injected into an open sinus; the film shows the track and whether or not it leads to the underlying bone or joint.



(a) (b) (c)

Figure 1.17 Contrast radiography (a) Myelography shows the outline of the spinal theca. Where facilities are available, myelography has been largely replaced by CT and MRI. (b) Discography is sometimes useful: note the difference between a normal intervertebral disc (upper level) and a degenerate disc (lower level). (c) Contrast arthrography of the knee shows a small popliteal herniation.

Arthrography

Arthrography is a particularly useful form of contrast radiography. Intra-articular loose bodies will produce filling defects in the opaque contrast medium. In the knee, torn menisci, ligament tears and capsular ruptures can be shown. In children's hips, arthrography is a useful method of outlining the cartilaginous (and therefore radiolucent) femoral head. In adults with avascular necrosis of the femoral head, arthrography may show up torn flaps of cartilage. After hip replacement, loosening of a prosthesis may be revealed by seepage of the contrast medium into the cement/bone interface. In the hip, ankle, wrist and shoulder, the injected contrast medium may disclose labral tears or defects in the capsular structures. In the spine, contrast radiography can be used to diagnose disc degeneration (discography) and abnormalities of the small facet joints (facetography).

Myelography

Myelography was used extensively in the past for the diagnosis of disc prolapse and other spinal canal lesions. It has been largely replaced by non-invasive methods such as CT and MRI. However, it still has a place in the investigation of nerve root lesions and as an adjunct to other methods in patients with back pain.

The oily media are no longer used, and even with the ionic water-soluble iodides there is a considerable incidence of complications, such as low-pressure headache (due to the lumbar puncture), muscular spasms or convulsions (due to neurotoxicity, especially if the chemical is allowed to flow above the mid-dorsal region) and arachnoiditis (which is attributed to the hyperosmolality of these compounds in relation to cerebrospinal fluid). Precautions, such as keeping the patient sitting upright after myelography, must be strictly observed.

Metrizamide has low neurotoxicity and at working concentrations it is more or less isotonic with cerebrospinal fluid. It can therefore be used throughout the length of the spinal canal; the nerve roots are also well delineated (radiculography). A bulging disc, an intrathecal tumour or narrowing of the bony canal will produce characteristic distortions of the opaque column in the myelogram.

Plain tomography

Tomography provides an image 'focused' on a selected plane. By moving the tube and the X-ray film in opposite directions around the patient during the exposure, images on either side of the pivotal plane are deliberately blurred out. When several 'cuts' are studied, lesions obscured in conventional X-rays may be revealed. The method is useful for diagnosing segmental bone necrosis and depressed fractures in cancellous bone (e.g. of the vertebral body or the tibial plateau); these defects are often obscured in the plain X-ray by the surrounding intact mass of bone. Small radiolucent lesions, such as osteoid osteomas and bone abscesses, can also be revealed.

A useful procedure in former years, conventional tomography has been largely supplanted by CT and MRI.

Computed tomography (CT)

Like plain tomography, CT produces sectional images through selected tissue planes – but with much greater resolution (Figure 1.18). A further advance over conventional tomography is that the images are trans-axial (like transverse anatomical sections), thus exposing anatomical planes that are never viewed in plain film X-rays. A general (or 'localization') view is obtained, the region of interest is selected and a series of cross-sectional images is produced and digitally recorded. 'Slices' through the larger joints or tissue masses may be 3–5 mm apart; those through the small joints or intervertebral discs have to be much thinner.

New multislice CT scanners provide images of high quality from which multiplanar reconstructions in all three orthogonal planes can be produced. Three-dimensional surface rendered reconstructions and volume rendered reconstructions may help in demonstrating anatomical contours, but fine detail is lost in this process.

Clinical applications

Because CT achieves excellent contrast resolution and spatial localization, it is able to display the size, shape and position of bone and soft-tissue masses in transverse planes. Image acquisition is extremely fast. The technique is therefore ideal for evaluating acute trauma to the head, spine, chest, abdomen and pelvis. It is better than MRI for demonstrating fine bone detail and soft-tissue calcification or ossification.

Computed tomography is also an invaluable tool for assisting with preoperative planning in secondary fracture management. It is routinely used for assessing injuries of the vertebrae, acetabulum, proximal tibial plateau, ankle and foot – indeed complex fractures and fracture-dislocations at any site (Figure 1.19).

It is also useful in the assessment of bone tumour size and spread, even if it is unable to characterize the tumour type. It can be employed for guiding soft-tissue and bone biopsies.