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Mechanisms of CLINICAL SIGNS

DENNIS, BOWEN AND CHO

ELSEVIER

3rd Edition

Mechanisms of CLINICAL SIGNS

The authors would like to dedicate this edition to the people who truly allow clinical examination skills to be taught – our patients.

Allowing a stranger (or more often multiple strangers) to physically examine you – at a time of vulnerability and distress – can be the most confronting part of medical care for many people. Having 'something found' by the person looking, listening and feeling is a situation every patient dreads, and from that point on, physical examination will be repeated throughout their hospital stay by senior staff, other specialties, interns, residents and groups of medical students shuffling around the bedside, often to the point where the person feels that they are no more than their pathology.

As students, the authors relied entirely on the good will of patients, who endured our clumsiness, confusion and nervousness with grace and acknowledgment of the learning process. So many times, the phrase 'you need to learn' was repeated as they gave us consent to practise our evolving skills, for which we remain eternally grateful.

Being allowed to examine a patient is a privilege, not a right. We take this opportunity to remind our readers that in the medical world, both 'patient' and 'sensitivity' have more than one definition.

Mechanisms of CLINICAL SIGNS

3rd Edition

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Elsevier Australia. ACN 001 002 357
(a division of Reed International Books Australia Pty Ltd)
Tower 1, 475 Victoria Avenue, Chatswood, NSW 2067

ISBN: 978-0-7295-4329-3

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National Library of Australia Cataloguing-in-Publication Data

A catalogue record for this
book is available from the
National Library of Australia

Content Strategist: Larissa Norrie
Content Project Manager: Fariha Nadeem
Copy edited by Katie Millar
Proofread by Annabel Adair
Cover by Alice Weston
Index by Innodata Indexing
Typeset by Toppan Best-set Premedia Limited
Printed in China by 1010 Printing International Limited

Last digit is the print number: 9 8 7 6 5 4 3 2 1

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Foreword

We live in a technological age, and the advances in multimodality imaging have revolutionised the practice of medicine. Nonetheless, these should not diminish the value of the human interaction between the patient and the physician. The essential components of optimal clinical practice are a clinical diagnosis based upon a careful history and physical examination, which in turn can drive appropriate sophisticated imaging, but not vice versa – the tail should never wag the dog. These precepts remain as important in the current era as they were previously.

This gem of a book, authored by Dr Mark Dennis, Dr William Bowen and Dr Lucy Cho, is now in its third edition, and is targeted primarily at medical students and physician trainees. The book illustrates key clinical signs on examination, but importantly, discusses the pathophysiology and mechanisms underlining these. There is a plethora of textbooks that teach clinical examination and a wealth of information on the pathogenesis of diseases. This text uniquely sits between these two tools by providing details (when known) of when and why the clinical signs we seek during a physical examination occur. The book is lavishly illustrated through colour photography, online video and audio, and the carefully designed flow diagrams which facilitate an understanding of complex mechanisms. In addition, the book provides helpful summaries and evidence of the utility of physical findings or the lack thereof in specific situations. This edition incorporates a number of additional features in response to reader feedback and includes a thoroughly updated list of references. One of the successful additions to the prior edition was the highlighted section Clinical Pearls, which draws attention to important concepts. Although the book is mainly directed at medical students and physician trainees, I found much in this book to interest the senior clinician in addition to providing a valuable reference to assist them in teaching the next generation of physicians. Readers of this book will use it as a specific reference for particular questions or as a book to be read from cover to cover to optimise their ability to interpret signs and to conduct an insightful physical examination.

This wonderful book has been a core text in medical schools across Australia for many years. It is ready to expand its presence on the global stage. I agree with the statement in the foreword to the second edition that 'this book provides a wonderful resource to those wishing to become more complete physicians'.

Bernard J. Gersh
Professor of Medicine, Mayo Clinic College of Medicine
Consultant in Cardiovascular Diseases and Internal Medicine

Preface 3rd Edition

The authors are very pleased to present the latest edition of this textbook. As with the second edition, refinements have been made in response to comments from clinicians, health workers and medical students far and wide. This edition brings current knowledge up to date with new evidence (where available) and the latest concepts in mechanisms of pathology. Many of these changes have been made due to direct feedback, and we are grateful to all the readers who take time to communicate with us.

The authors recognise clinical examination is changing. More and more there is greater focus on the use of imaging techniques for diagnosis. One example is the addition of point-of-care ultrasound (POCUS) in emergency medicine, an area we hope to integrate into future editions. However, as always, we urge our readers not to dismiss the knowledge and skills required for a thorough physical examination – for which there is truly no replacement.

We hope you find the third edition of *Mechanisms of Clinical Signs* a useful adjunct to your work and training and look forward to bringing you subsequent publications.

Abbreviations

5-HT	5-hydroxytryptamine (serotonin)
ACA	anterior cerebral artery
ACE	angiotensin-converting enzyme
ACL	anterior cruciate ligament
ACTH	adrenocorticotropic hormone
ADP	adenosine diphosphate
ADH	antidiuretic hormone, or vasopressin
AIDS	acquired immune deficiency syndrome
AION	anterior ischaemic optic neuropathy
AN	acanthosis nigricans
AP	anterioposterior
AR	aortic regurgitation
ARDS	acute respiratory distress syndrome
ASD	atrial septal defect
AV	arterio-venous
AV (node)	atrioventricular (node)
AVM	arteriovenous malformation
BMI	body mass index
BPH	benign prostatic hypertrophy
BPPV	benign paroxysmal positional vertigo
CCK	cholecystokinin
CG	ciliary ganglion
CGL	chronic granulocytic leukaemia
CGRP	calcitonin gene-related peptide
CHF	congestive heart failure
CI	confidence interval
CLL	chronic lymphocytic leukaemia
CMC	carpometacarpal
cMOAT	canalicular multispecific organic anion transporter
CMT	Charcot–Marie–Tooth (disease)
CMV	cytomegalovirus
COPD	chronic obstructive pulmonary disease
CNS	central nervous system
CRAO	central retinal artery occlusion
CREST	calcinosis cutis, Raynaud's phenomenon, (o)esophageal dysfunction, sclerodactyly, telangiectasia syndrome
CRH	corticotrophin-releasing hormone
CRVO	central retinal vein occlusion
CS	cavernous sinus
CSA	central sleep apnoea
CSF	cerebrospinal fluid
CT	computerised tomography
CV	cortical veins
CVP	central venous pressure
DAS	dorsal acoustic stria
DHEA-S	dehydroepiandrosterone sulfate
DIP	distal interphalangeal joint
DI	diabetes insipidus
DM	diabetes mellitus
DRE	digital rectal examination
DVT	deep vein thrombosis
EBV	Epstein–Barr virus
EGFR	epidermal growth factor receptor
EMH	extramedullary haematopoiesis
ENAC	epithelial sodium (Na) channel
EOM	extraocular muscle
EW	Edinger–Westphal nucleus
FABER	flexion abduction external rotation
FGFR	fibroblast growth factor receptor
FSH	follicle-stimulating hormone
G6PD	glucose-6-phosphate dehydrogenase
GABA	gamma-aminobutyric acid
GAS	group A streptococcus
GBS	Guillain–Barré syndrome
GH	growth hormone
GI	gastrointestinal
GnRH	gonadotrophin-releasing hormone
GORD	gastro-oesophageal reflux disease
GPe	globus pallidus pars externa
GPi	globus pallidus pars interns

Gs	guanine nucleotide-binding protein that couples to TSH receptor
GV	great vein of Galen
Hb	haemoglobin
HbSC	sickle cell haemoglobin C
hCG	human chorionic gonadotropin
HIV	human immunodeficiency virus
HLA	human leukocyte antigen
HOCM	hypertrophic obstructive cardiomyopathy
HPOA	hypertrophic pulmonary osteoarthropathy
HPV	human papilloma virus
HSV	herpes simplex virus
IAS	intermediate acoustic stria
IBD	inflammatory bowel disease
ICA	internal carotid artery
ICV	internal cerebral vein
IFN	interferon
IGF-1	insulin-like growth factor-1
IJ	internal jugular vein
IL	interleukin
INC	interstitial nucleus of Cajal
INO	internuclear ophthalmoplegia
IO	inferior oblique (muscle or subnucleus)
IR	inferior rectus (muscle or subnucleus)
ISS	inferior sagittal sinus
IVC	inferior vena cava
JVP	jugular venous pressure
LA	left atrial
LBBB	left bundle branch block
LGN	lateral geniculate nucleus
LH	luteinising hormone
LPS	lipopolysaccharides
LR	lateral rectus (muscle)
LR	likelihood ratio
LR	livedo reticularis
LS	lateral sinus
LTB_4	leukotriene B_4
LV	left ventricular
MAOI	monoamine oxidase inhibitor
MCA	middle cerebral artery
MCPJ	metacarpophalangeal joint
MD	muscular dystrophy
MDMA	methylenedioxymethamphetamine (Ecstasy)
MDPK	myotonic dystrophy protein kinase
MEN	multiple endocrine neoplasia
MLF	medial longitudinal fasciculus
MMP	matrix metalloproteinase
MPTP	1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (toxicity)
MR	medial rectus (muscle)
MRF	midbrain reticular formation
MRI	magnetic resonance imaging
mRNA	messenger ribonucleic acid
MSH	melanocyte-stimulating hormone
MTP	metatarsophalangeal
MV	mitral valve
NAA	N-acetyl-L-aspartate
NF-κB	nuclear factor kappa-light-chain-enhancer of activated B cells
NHL	non-Hodgkin lymphoma
NLD	necrobiosis lipodica diabeticorum
NO	nitric oxide
NPV	negative predictive value
OCP	oral contraceptive pill
OSA	obstructive sleep apnoea
PAI-1	plasminogen activator inhibitor-1
PC	posterior commissure
PCA	posterior cerebral artery
PComm	posterior communicating artery
PCOS	polycystic ovarian syndrome
PCP	phencyclidine (toxicity)
PCWP	pulmonary capillary wedge pressure
PDA	patent ductus arteriosus
PDGF	platelet-derived growth factor
PFO	patent foramen ovale
PGE	prostaglandin E
PGI_2	prostaglandin I_2
PGH	prostaglandin H
PICA	posterior inferior cerebellar artery
PIP	proximal interphalangeal joint

PLR	positive likelihood ratio
PND	paroxysmal nocturnal dyspnoea
PPRF	paramedian pontine reticular formation
PPV	positive predictive value
POMC	pro-opiomelanocortin
PR (interval)	measured from the beginning of the P wave to the beginning of the QRS complex
PR	pulmonary regurgitation
PS	petrosal sinus
PSA	prostate-specific antigen
PSP	progressive supranuclear palsy
PTH	parathyroid hormone
PTH-rp	parathyroid hormone-related protein
PTN	pretectal nucleus
RA	rheumatoid arthritis
RA	right atrial
RAA(S)	renin–angiotensin–aldosterone (system)
RANK	receptor activator of nuclear factor kappa
RAPD	relative afferent pupillary defect
RAR	rapidly adapting receptor
RBBB	right bundle branch block
RBC	red blood cell
riMLF	rostral interstitial medial longitudinal fasciculus
RN	red nucleus
RNA	ribonucleic acid
RR	relative risk or risk ratio
RTA	renal tubule acidosis
RV	right ventricular
SA (node)	sinoatrial (node)
SC	superior colliculus
SCA	superior cerebellar arteries
SCC	squamous cell carcinoma
SCFE	slipped capital femoral epiphysis
SLAP	superior labrum anterior posterior
SLE	systemic lupus erythematosus
SNc	substantia nigra pars compacta
SNr	substantia nigra pars reticulate
SO	superior oblique (muscle)
SPS	stiff-person syndrome
SR	superior rectus (muscle or subnucleus)
SS	sigmoid sinus
SS	straight sinus
SSS	superior sagittal sinus
SSRI	selective serotonin reuptake inhibitor
STN	subthalamic nucleus
SVC	superior vena cava
T_3	triiodothyronine (thyroid hormone)
T_4	thyroxine (thyroid hormone)
TB	tuberculosis
TF	tissue factor
TGF-β	transforming growth factor-beta
TH	torcular Herophili
Th-1	helper T cell type 1
TIA	transient ischaemic attack
TNF	tumour necrosis factor
TRH	thyrotrophin-releasing hormone
TS	transverse sinus
TSH	thyroid stimulating hormone
TSHR	thyroid stimulating hormone receptor
TTP	thrombotic thrombocytopenic purpura
URTI	upper respiratory tract infection
V2 (receptor)	arginine vasopressin receptor 2
VAS	ventral acoustic stria
VEGF	vascular endothelial growth factor
VIP	vasoactive intestinal peptide
VL	ventral lateral
VSD	ventricular septal defect
vWF	von Willebrand factor
VZV	varicella zoster virus

Sign Value

Eliciting or identifying a clinical sign is a requisite skill in medicine – however, it is merely the beginning of the story. More importantly, a good clinician understands a sign's predictive value, evidence base and role in diagnostic evaluation. The presence or absence of a clinical sign offers a data point, allowing us to refine the probability of the disease of interest as the differential diagnosis (i.e. the process of risk stratification).

In the *Sign Value* section, the reader will find a brief précis of the evidence base for the given sign, including (where available), sensitivity, specificity, positive or negative predictive values and/or likelihood ratios. With a positive LR (sign is present) or negative LR (sign is absent) value, one can determine the post-test probability of disease, using the following equation (a component of Bayesian Theory):

$$\text{Pre-test probability} \times \text{likelihood ratio} = \text{post-test probability}$$

Example:
A 20-year-old, immunocompetent, male student presents to his local Emergency Department, complaining of severe headache. The junior doctor assessing him notes that he appears toxic, is febrile and has a non-blanching purpuric rash and non-focal neurological signs. The doctor specifically identifies the absence of Kernig's sign. The entire clinical scenario is suspicious for bacterial meningitis due to *Neisseria meningitides*, complicated by meningococcaemia. This patient has a very high pre-test probability of bacterial meningitis. The absence of Kernig's sign (–LR 1.0) does not affect the probability of meningitis being present.

$$\textit{Very high pre-test probability} \times 1.0 = \textit{very high post-test probability}$$

It is critical to understand the predictive value of the presence or absence of a clinical sign. The junior clinician should not be swayed by the absence of Kernig's sign. This patient requires emergent administration of IV antibiotics, lumbar puncture and public health notification. CT imaging prior to lumbar puncture may be considered in certain clinical scenarios.

The sensible clinician will judiciously consider examination findings and/or diagnostic tests and how they affect the probability of the diagnosis and management plan.

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CHAPTER 1

MUSCULOSKELETAL SIGNS

Anterior drawer test

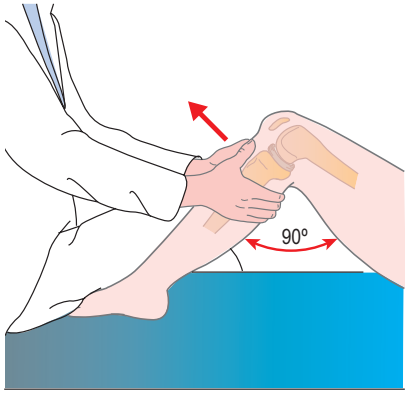


FIGURE 1.1
Anterior drawer test for anterior cruciate ligament injury

Description

With the patient lying supine, the knee at 90° flexion and the foot immobilised by the examiner, the proximal third of the tibia is pulled towards the examiner. In a positive test, there is anterior (forward) movement of the tibia without an abrupt stop.[1]

Condition/s associated with

- Anterior cruciate ligament (ACL) injury

Mechanism/s

The ACL arises from the anterior aspect of the tibial plateau and inserts into the medial aspect of the lateral femoral condyle. It limits anterior movement of the tibia upon the femur. Loss of continuity of the ACL permits inappropriate anterior movement of the tibia and thus knee joint instability.

Sign value

A literature review of six studies reported variable sensitivity of 27–88%, specificity of 91–99%, positive LR of 11.5 and negative LR of 0.5.[2] A literature review by Solomon DH et al. of nine studies reported a sensitivity of 9–93% and specificity of 23–100%.[1]

While a positive anterior drawer sign (+LR 11.5)[2] has been suggested to be strong evidence of ACL injury, the results are not uniform, with another study reporting a +LR 2.0 (sensitivity 83%, specificity 57%, –LR 0.3).[3] A negative anterior drawer sign cannot reliably exclude ACL injury (sensitivity 27–88%; –LR 0.5).[2] When strong clinical suspicion persists, further diagnostic steps are necessary (e.g. interval re-examination, MRI, arthroscopy).

CLINICAL PEARL

Apley's grind test

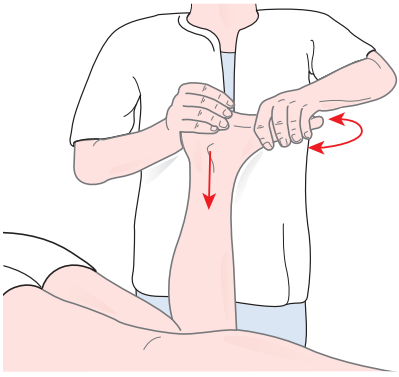


FIGURE 1.2
Apley's grind test

Description

With the patient lying prone and the knee at 90° flexion, the lower leg is passively internally and externally rotated while axial pressure is applied to the lower leg. The test is considered positive if tenderness is elicited.

The process can also be combined with or without distraction. If rotation plus distraction is more painful or there is increased rotation relative to the unaffected side, this is suggestive of a ligamentous lesion. If rotation plus compression is more painful or there is decreased rotation relative to the unaffected side, this is suggestive of meniscal injury.[4]

Condition/s associated with

- Meniscal injury

Mechanism/s

Direct mechanical force upon the injured meniscus elicits tenderness.

Sign value

A review by Hegedus EJ et al. reported a pooled sensitivity of 60.7% and specificity of 70.2% with an odds ratio of 3.4.[5] Significant heterogeneity in the data limits its accuracy. Overall, Apley's grind test has limited diagnostic utility, limited supporting data and, in the acute setting, the manoeuvre produces severe pain.[6]

McMurray's grind test has more robust supporting data.

Apley's scratch test

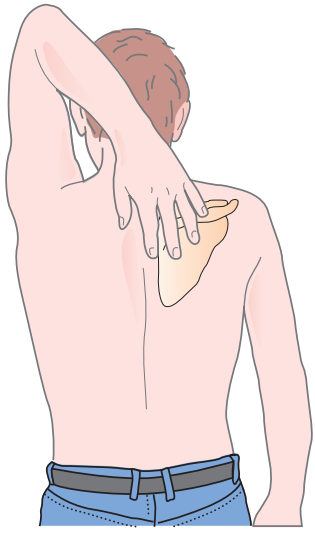


FIGURE 1.3
One of three manoeuvres of Apley's scratch test

Based on Woodward T, Best TM. The painful shoulder: part 1, clinical evaluation. Am Fam Phys *2000; 61(10): 3079–3088.*

Description

Apley's scratch test is a general range of movement assessment of the shoulder joint (i.e. glenohumeral, acromioclavicular, sternoclavicular and scapulothoracic joints). The patient is instructed to touch the unaffected shoulder anteriorly and posteriorly (behind their head), and touch the inferior scapula posteriorly (behind their back). Tenderness and/or limited range of movement while performing these movements is considered an abnormal test.[7]

Condition/s associated with

Common

- Rotator cuff muscle injury
- Labral tear
- Anterior shoulder dislocation
- Bicipital tendonitis
- Adhesive capsulitis (frozen shoulder)
- Acromioclavicular joint injury

Mechanism/s

The shoulder joint is a complex structure. Its components include the humeral head, glenoid fossa, acromion, clavicle, scapula and surrounding soft tissue structures. Under normal circumstances the shoulder joint is capable of a vast range of movement. Apley's scratch test assesses glenohumeral abduction, adduction, flexion, extension, internal rotation and external rotation. Tenderness or limited range of movement suggests injury to one or more components of the shoulder joint.

Sign value

Apley's scratch test is a useful component of the general shoulder exam but has limited utility for a specific diagnosis. The position of the shoulder at which tenderness or limited range of movement occurs should be noted. In the patient with an abnormal Apley's scratch test, further diagnostic manoeuvres should be performed to narrow the differential diagnosis.

Apparent leg length inequality (functional leg length)

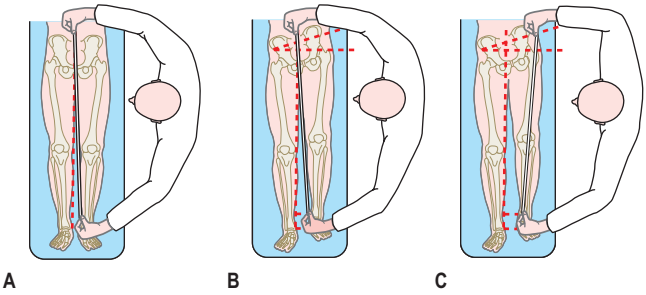


FIGURE 1.4
Measurement of leg lengths
A The apparent leg length is the distance from the umbilicus to the medial malleolus; **B** pelvic rotation causing an apparent leg length discrepancy; **C** the true leg length is the distance from the anterior superior iliac spine to the medial malleolus.

Based on Firestein GS, Budd RC, Harris ED et al., Kelley's Textbook of Rheumatology, *8th edn, Philadelphia: WB Saunders, 2008: Fig 42-24.*

Description

A disparity between the relative distance from the umbilicus to the medial malleolus of each leg.[8] By definition it implies asymmetry of the lower extremities in the absence of a bony abnormality. (See 'True leg length inequality' in this chapter.)

Condition/s associated with

- Altered foot mechanics
- Adaptive shortening of soft tissues
- Joint contractures
- Ligamentous laxity
- Axial malalignments

Mechanism/s

An apparent or functional leg length inequality may occur at any point from the pelvis to the foot.[8]

Ligamentous laxity

The ligaments on one side (e.g. in the hip joint) may be more flexible or longer than their counterparts, making the femur sit lower in the joint capsule.

Joint contracture

A joint contracture impairs full range of movement. If the knee joint is contracted in a flexed position, the length of the affected side will be less than the opposite leg during maximal attempted extension.

Altered foot mechanics

Excessive pronation of the foot eventuates in and/or may be accompanied by a decreased arch height compared to the 'normal' foot, resulting in a functionally shorter limb.[8]

Sign value

The distance (anywhere from 3–22 mm) at which apparent leg length inequality results in a clinically significant effect is controversial.[8] The test should be interpreted in relation to the patient's history and full gait assessment.

Apprehension test

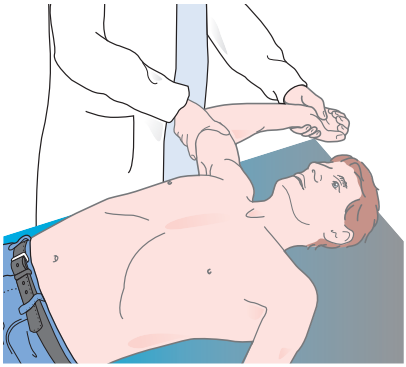


FIGURE 1.5
Apprehension test
The arm is abducted and placed in an externally rotated position. Note the right arm of the examiner is providing anterior traction on the humerus, pulling the posterior part of the humeral head forward. The same test can be done from the back, with the patient sitting up and the examiner pushing forward on the posterior head of the humerus.

Description

The apprehension test is an assessment of glenohumeral joint instability. With the patient sitting or lying supine, the shoulder is placed into 90° abduction, 90° external rotation and 90° elbow flexion. The examiner applies pressure to the posterior aspect of the proximal humerus and attempts to move the humeral head anteriorly (see Figure 1.5). The test is positive if the patient experiences *apprehension* due to impending subluxation or dislocation of the glenohumeral joint.[9]

Condition/s associated with

More common – traumatic

- Recurrent glenohumeral joint subluxation or dislocation
- Rotator cuff muscle injury
- Glenoid labrum injury
- Glenoid defect (e.g. Bankart's fracture)
- Humeral head defect (e.g. Hill–Sachs fracture)

Less common – atraumatic

- Connective tissue disorder: Ehlers–Danlos syndrome, Marfan's syndrome
- Congenital absence of glenoid

Mechanism/s

Glenohumeral joint instability is caused by dysfunction of the bony and/or soft tissue structures that maintain joint stability: glenoid, humeral head, joint capsule, capsuloligamentous or glenohumeral ligaments, labrum, and rotator cuff muscles. The shoulder joint is susceptible to instability due to its inherent mobility and complex soft tissue structures responsible for stability.

In the apprehension test, the joint is placed into a position vulnerable to instability. It is the typical position precipitating traumatic anterior shoulder dislocation. For this reason, a significant number of healthy patients will experience apprehension during this manoeuvre.

Sign value

T'Jonck L et al. reported a sensitivity of 88.0%, specificity of 50%, positive likelihood ratio of 1.8 and negative likelihood ratio of 0.23.[10]

The apprehension test for glenohumeral joint instability is a moderately useful screening test. Based on available data, the test has limited utility to rule in the diagnosis. It is not used in the setting of acute anterior shoulder dislocation.

Apprehension–relocation test (Fowler's sign)

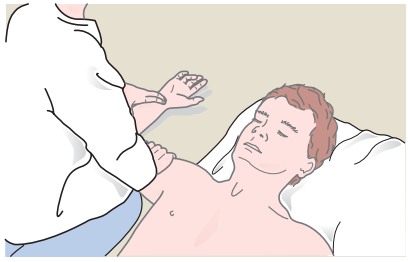


FIGURE 1.6
Apprehension–relocation (Fowler) test
Note that pressure is applied anteriorly to the proximal humerus.

Description

The apprehension–relocation test is an assessment of glenohumeral joint instability. The relocation manoeuvre is typically performed following the apprehension test (see 'Apprehension test'). With the patient sitting or lying supine, the shoulder is placed into 90° abduction, 90° external rotation and 90° elbow flexion. The examiner applies pressure to the anterior aspect of the proximal humerus and attempts to move the humeral head posteriorly. The test is positive if the patient experiences relief of apprehension (i.e. no longer feels impending shoulder dislocation).

Condition/s associated with

- Recurrent glenohumeral joint subluxation or dislocation
- Rotator cuff muscle injury
- Glenoid labrum injury
- Glenoid defect (e.g. Bankart's fracture)
- Humeral head defect (e.g. Hill–Sachs fracture)

Less common – atraumatic

- Connective tissue disorder: Ehlers–Danlos syndrome, Marfan's syndrome
- Congenital absence of glenoid

Mechanism/s

The underlying anatomy and causes of glenohumeral joint instability are outlined under 'Apprehension test' and apply here. In the apprehension–relocation test, symptomatic relief is due to restoration of the normal anatomical relationship of the humeral head in the glenohumeral joint.

Sign value

T'Jonck L et al. reported a sensitivity of 85%, specificity of 87%, positive likelihood ratio of 6.5 and negative likelihood ratio of 0.18.[10] Lo et al. reported sensitivity of 32% and specificity of 100%.[11] Speer KP et al. reported a sensitivity of 68% and specificity of 100%.[12]

The apprehension–relocation test is a useful screening manoeuvre for anterior glenohumeral joint instability. It appears to be more specific than the 'apprehension test' alone.

Bouchard's and Heberden's nodes

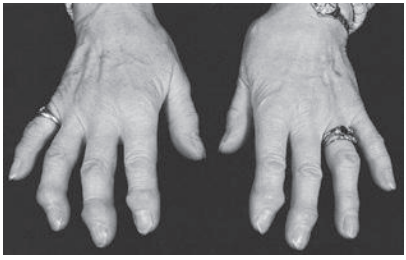


FIGURE 1.7
Prominent Heberden's nodes
Based on Ferri FF, Ferri's Clinical Advisor, *Philadelphia: Elsevier, 2011: Fig 1-223.*

Description

Bouchard's nodes are bony outgrowths or nodules found over the *proximal* interphalangeal joints of the hands.

Heberden's nodes are similar but located over the *distal* interphalangeal joints.

Condition/s associated with

- Osteoarthritis
- Familial

Mechanism/s

A number of studies have implicated *bony osteophyte growth* as the principal cause of Heberden's and Bouchard's nodes.[13] Other contributing factors or theories include:

- genetic predisposition
- endochrondral ossification of hypertrophied cartilage as a result of chronic osteoarthritic changes[14]
- traction spurs growing in tendons in response to excessive tension and repetitive strain.[15]

Sign value

Bouchard's or Heberden's nodes are a classical sign of interphalangeal osteoarthritis[15,16] and are associated with generalised osteoarthritis.[17,18] The presence of Bouchard's and/or Heberden's nodes is predictive of the radiographic changes of osteoarthritis.[19]

Boutonnière deformity

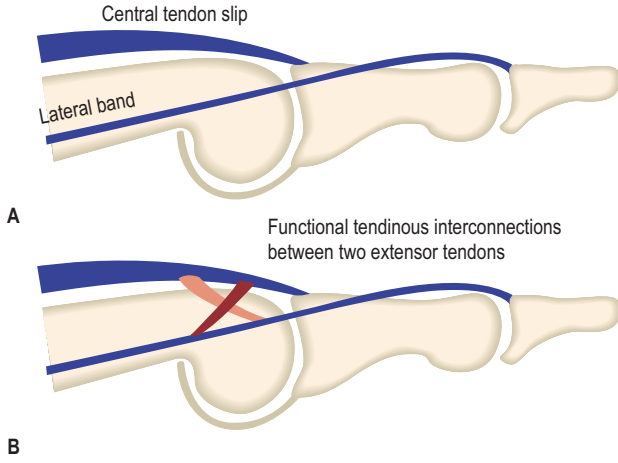


FIGURE 1.8
Digital extensor mechanism
A The proximal interphalangeal joint is extended by the central tendon slip (an extension of the hand's dorsal extensor tendon); **B** the *X* is a functional representation of the fibrous interconnections between the two systems.

Based on DeLee JC, Drez D, Miller MD, DeLee and Drez's Orthopaedic Sports Medicine, *3rd edn, Philadelphia: Saunders, 2009: Fig 20B2-27.*

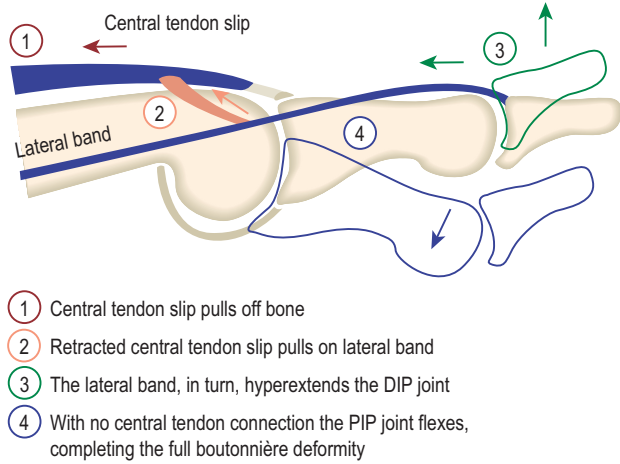


FIGURE 1.9
Pathoanatomy of boutonnière deformity
The sequence is: rupture of the central tendon slip, which then simultaneously pulls on the lateral bands, pulling the DIP joint into hyperextension and the PIP into flexion.

Based on DeLee JC, Drez D, Miller MD, DeLee and Drez's Orthopaedic Sports Medicine, *3rd edn, Philadelphia: Saunders, 2009: Fig 20B2-28.*

Description

Used to describe a deformity of the resting finger in which the proximal interphalangeal (PIP) joint is flexed and the distal interphalangeal (DIP) joint is hyperextended.

Condition/s associated with

- Inflammatory arthropathy (e.g. rheumatoid arthritis)
- Central slip extensor tendon injury

Mechanism/s

Disruption or avulsion of the *central slip extensor tendon* and volar migration of the lateral bands of the extensor tendon mechanism result in PIP flexion and DIP extension. The sign derives its name from the appearance of the central tendon slip, which was thought to resemble a buttonhole, or *boutonnière* in French, when torn.

The central tendon slip attaches to the dorsal aspect of the middle phalanx. Its main function is to maintain PIP extension and stabilise the extensor tendon apparatus. If the central tendon is disrupted or avulsed (torn off the base of the middle phalanx), the actions of the lateral bands and flexor digitorum profundus are unopposed, resulting in resting PIP flexion and DIP hyperextension.

Inflammatory arthropathy (e.g. rheumatoid arthritis)

Pannus in the PIP joint (which may be present in rheumatoid arthritis) can damage the central slip tendon.[20] Chronic inflammation and synovitis of the joint may result in persistent PIP flexion and gradual elongation of the central slip tendon. Subsequent volar migration of the lateral bands results in the characteristic deformity.[21–24]

Trauma

Forced flexion of an extended PIP joint, crush injury or penetrating injury may result in avulsion of the central tendon slip. Typically, the degree of deformity increases in the days following the injury. Acutely, the deformity may be subtle.

Sign value

A boutonnière deformity is classically associated with rheumatoid arthritis occurring in up to 50% of patients with the disease.

In a patient with blunt or penetrating trauma, the presence of a boutonnière deformity should be considered evidence of a central slip extensor tendon injury.

Bulge/wipe/stroke test

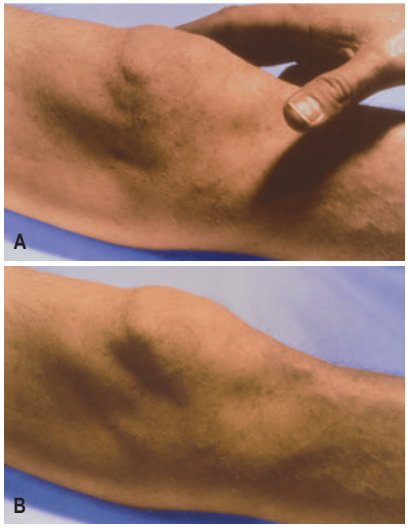


FIGURE 1.10
Demonstration of the bulge test for a small synovial knee effusion
The medial aspect of the knee has been stroked to move the synovial fluid from this area (shaded depressed area in **A**); **B** shows a bulge in the previously depressed area after the lateral aspect of the knee has been tapped.

Based on Firestein GS, Budd RC, Harris ED et al., Kelley's Textbook of Rheumatology*, 8th edn, Philadelphia: WB Saunders, 2008: Figs 35-9A and B.*

Description

The bulge, wipe or stroke test is used to assess for knee joint effusion. With the patient supine and their knee extended, the examiner 'swipes' the medial aspect of the knee joint to displace fluid into the superolateral aspect of the synovial compartment, and then swipes the lateral side looking for a visible fluid shift. The test is positive if the examiner sees a wave of fluid.

Condition/s associated with

Any condition causing a knee effusion.

More common

- Osteoarthritis
- Rheumatoid arthritis
- Haemoarthrosis – trauma, coagulopathy
- Gout
- Infection – septic arthritis, gonococcal arthritis, transient synovitis

Less common

- Pseudogout (calcium pyrophosphate deposition disease)
- Tumour

Mechanism/s

Mechanical manipulation of excess fluid in the synovial joint capsule results in visible fluid shift. The wipe or bulge test displaces synovial fluid from one part of the synovial joint to another, thus suggesting the presence of a joint effusion as the cause of knee swelling.

Sign value

Limited evidence has been gathered on the value of this test as an individual sign. Some authors report that this test may pick up on *as little as 4–8 mL of swelling*.[25] An effusion in the absence of acute traumatic injury or systemic disease is most commonly due to osteoarthritis.[26]

Gogus F et al.[27] reported the wipe test as having a sensitivity of 11–33% and specificity of 66–92% for identifying the presence of a knee effusion. Emphasis should be placed upon identifying a joint effusion in the setting of septic arthritis, an orthopaedic emergency.

Butterfly rash (malar rash)

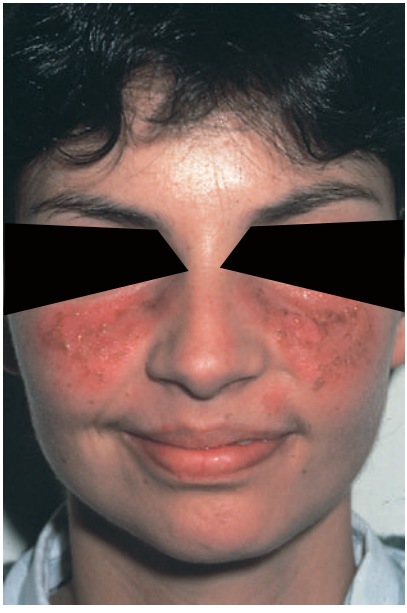


FIGURE 1.11
Malar rash of SLE

Reproduced, with permission, from Goldman L, Ausiello D, Cecil Medicine, *23rd edn, Philadelphia: Saunders, 2007: Fig 287-3.*

Description

A red or purple, macular, mildly scaly rash that is seen over the bridge of the nose and cheeks. The shape of the rash can somewhat resemble a butterfly. The rash spares the nasolabial folds, which helps distinguish it from other rashes (e.g. rosacea). It is also photosensitive.

Condition/s associated with

Common

- Systemic lupus erythematosus (SLE)
- Drug-induced lupus erythematosus
- Dermatomyositis

Mechanism/s

The exact mechanism is unclear. However, like the underlying disorder in SLE, it is thought to result from an autoimmune reaction caused by genetic, environmental and immunological factors.

Factors shown to be involved include:[28]

- A genetic predisposition to ineffective or deficient complement, leading to a failure to clear immune complexes of apoptotic cells, which in turn increases the chance of the development of autoimmunity.
- Sunlight has been shown to damage and/or induce apoptosis of keratinocyte proteins in the epidermis and can stimulate autoantibody production. Sunlight may also increase the chance of keratinocytes being destroyed by complement and antibody-dependent mechanisms.
- Altered cellular and humoral immunity reactions have been seen in studies reviewing cutaneous manifestations of lupus.

It is likely that a combination of these factors leads to immune deposition in the skin, damage, oedema and the characteristic malar rash.

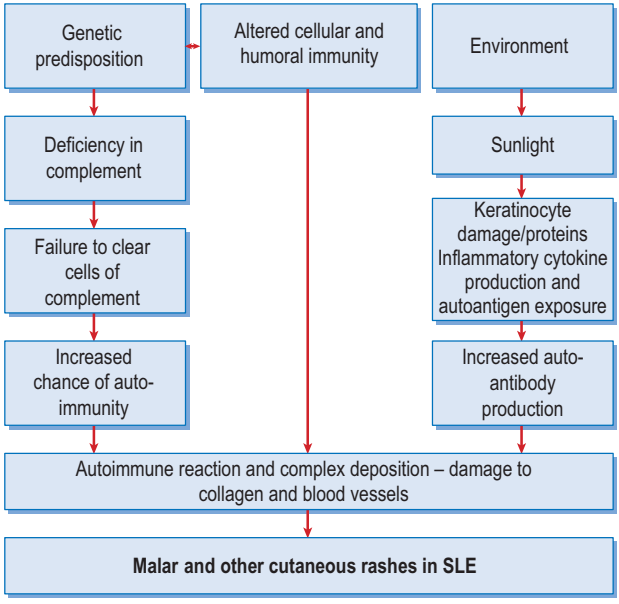


FIGURE 1.12
Mechanism of malar rash

Sign value

The malar rash is seen in approximately 40% of patients with SLE.[28] It has a sensitivity of 57% and specificity of 96% for SLE.[14] Its absence does not exclude the diagnosis.

Calcinosis/calcinosis cutis

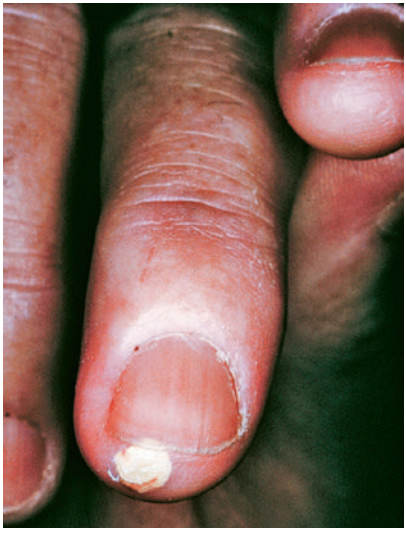


FIGURE 1.13
Calcinosis
Hard, whitish nodules of the digit representing dystrophic calcinosis in this patient with dermatomyositis.

Reproduced, with permission, from James WD, Berger T, Elston D, Andrews' Diseases of the Skin: Clinical Dermatology*, 11th edn, Philadelphia: Saunders, 2011: Fig 26-12.*

Description

Calcinosis refers to the formation or deposition of calcium in soft tissue. *Calcinosis cutis* more specifically refers to calcium deposits in the skin.

Condition/s associated with

Conditions associated with calcinosis may be classified as dystrophic, metastatic, iatrogenic, idiopathic or calciphylaxis.

- Dystrophic calcinosis
  - » Scleroderma
  - » Dermatomyositis
  - » SLE
  - » Systemic sclerosis
  - » Burns
- Metastatic
  - » Due to hypercalcaemia or hyperphosphataemia of any cause
  - » Chronic renal failure – most common
  - » Excess vitamin D
  - » Primary hyperparathyroidism – rare
  - » Paraneoplastic hypercalcaemia
  - » Destructive bone disease (e.g. Paget's disease)
- Iatrogenic
  - » Calcium gluconate injections
  - » Tumour lysis syndrome
- Idiopathic
- Calciphylaxis
  - » End-stage renal disease
  - » Altered calcium metabolism

Mechanism/s

Dystrophic calcinosis

Dystrophic calcinosis occurs when *crystals of calcium phosphate or hydroxyapatite* are deposited in the skin *secondary to inflammation, tissue damage and degeneration.*[30] Calcium and phosphate levels are usually *normal.* Proposed mechanisms include:

- High local levels of alkaline phosphatase break down a pyrophosphate that normally inhibits calcification.[31]
- Tissue breakdown may lead to denatured proteins that bind to phosphate. These phosphate–protein compounds may react with calcium and thus provide a nidus for calcification.[32]

Metastatic calcinosis

Abnormal calcium or phosphate metabolism with high levels of either or both is present. Excess calcium and/or phosphate allows for the formation and precipitation of calcium salts.

In chronic renal failure a number of mechanisms lead to altered phosphate and calcium metabolism:

- Decreased renal excretion of phosphate leads to hyperphosphataemia.
- Hyperphosphataemia results in a compensatory rise in parathyroid hormone (PTH) in an attempt to excrete phosphate. The rise in PTH results in an increase in phosphate absorption from the gut and also mobilises calcium from the bones, resulting in more calcium being available to precipitate with phosphate.
- Vitamin D deficiency owing to renal failure worsens initial hypocalcaemia and, therefore, further stimulates secondary hyperparathyroidism.

Iatrogenic

Intravenous administration of calcium or phosphate may cause local extravasation and precipitation of hydroxyapatite in surrounding tissue. Inflammation of the surrounding tissue secondary to the injection may also cause calcium and protein release, contributing to precipitation.

Idiopathic

Occurs in the absence of tissue injury or systemic metabolic disturbance.

Sign value

There is very limited evidence on this sign and it is rarely seen in isolation. If identified, further investigation is warranted.

Charcot foot

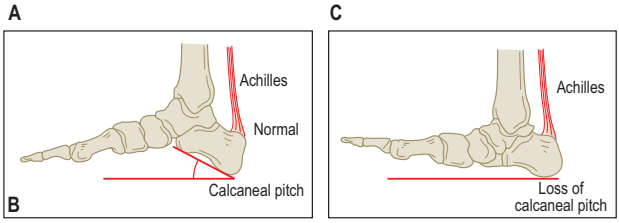


FIGURE 1.14
Charcot foot
A, **B** The classic rocker-bottom Charcot foot, with collapse and then reversal of the longitudinal arch; **C** loss of the normal calcaneal pitch, or angle relative to the floor, in patients with Charcot collapse of the arch.

Reproduced, with permission, from Mann JA, Ross SD, Chou LB, Chapter 9: Foot and ankle surgery. In: Skinner HB, Current Diagnosis & Treatment in Orthopedics, *4th edn, Fig 9-8. Available: http://proxy14.use.hcn.com.au/content.aspx?aID=2321540 [10 Mar 2011].*

Description

A progressive destructive arthropathy of the ankle and foot.[33] In its early stages, it may present as unilateral foot oedema following minor trauma. In advanced disease, significant destruction of bones and joints may occur (particularly in the midfoot), resulting in collapse of the plantar arch and development of 'rocker-bottom foot'.

Condition/s associated with

Conditions resulting in sensory neuropathy:

- Diabetes mellitus – most common
- Syphilis – original description by Charcot

Mechanism/s

In *neurotraumatic theory*, peripheral neuropathy caused by diabetes leads to decreased pain sensation and impaired proprioception. Thus, if an acute injury occurs (e.g. microfracture, subluxation or fracture), the patient feels little or no pain and does not 'guard' the foot when mobilising. This leads to a destructive cycle of continued loading on the injured foot and progressive damage.[34]

Under the *inflammatory theory*, when the same local insult occurs (microfracture, subluxation or fracture), inflammatory cytokines are released, including TNF-α and interleukin-1β. These two cytokines have been shown to increase activation of RANK ligand (RANKL), which in turn increases the transcription factor nuclear factor-κB (NF-κB). The net result of this is *stimulation of the maturation of osteoclasts*, which further eat away at bone. This predisposes the patient to engage in another vicious cycle of further fractures, inflammation, abnormal weight loading and osteolysis.[34]

Regardless of underlying contributing factors, the RANKL/OPG (osteoprotegerin) pathway is thought to be a common denominator.

RANKL is a member of the tumour necrosis factor (TNF) superfamily, and OPG is the competitive protein of RANKL. The process of bone

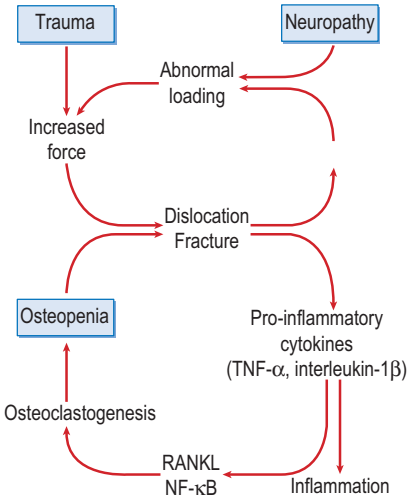


FIGURE 1.15
Simplified inflammatory and neurotraumatic mechanisms of Charcot foot

Based on Jeffcoate WJ, Game F, Cavanagh PR, Lancet *2005; 366: 2058–2061.*

resorption and formation is controlled by the level of RANKL and OPG, and many factors contribute to this pathway.

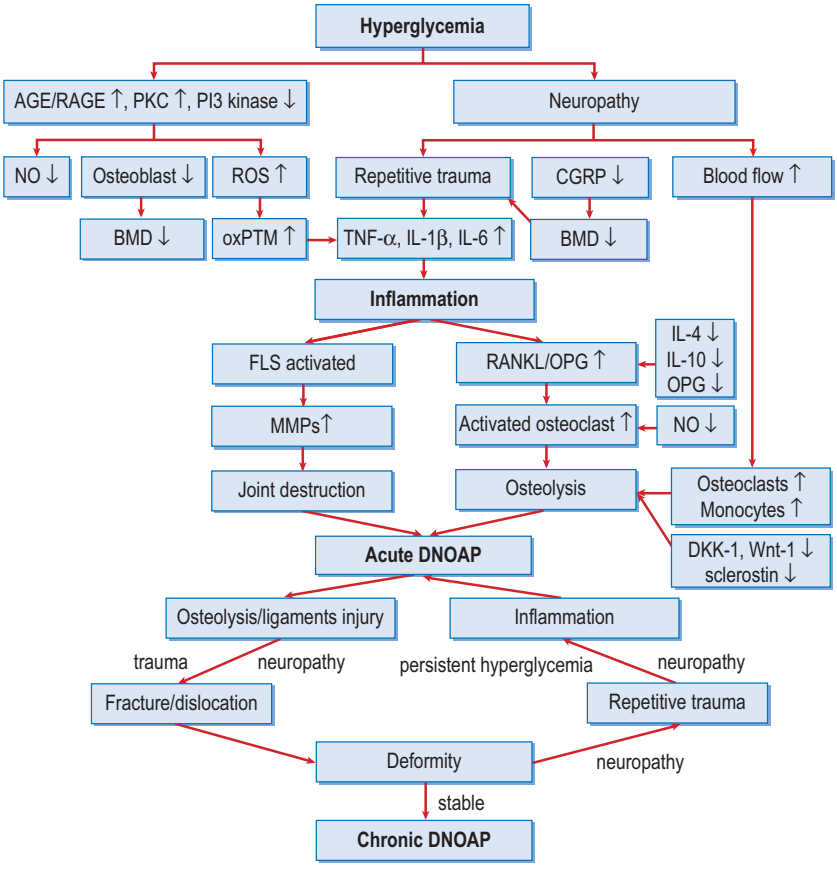
RANKL has been shown to mediate osteolysis in Charcot foot by stimulating osteoclastic differentiation of monocytes/macrophages and triggering the synthesis of nuclear factor-κβ (NF-κβ), spurring maturation.

Hyperglycaemia can increase the level of advanced glycation end products (AGEs), reactive oxygen species (ROS), and oxidised lipids, which may all enhance the expression of RANKL in diabetes.

The pro-inflammatory state increases cytokine expression (TNF-α, IL-1β and IL-6), which may also increase RANKL. Diabetic neuropathy is associated with exhaustion of calcitonin gene-related peptide (CGRP) stores from C and Aδ nerve fibres. The deficiency of CGRP may further accelerate underlying osteopenia due to unrestricted RANKL activity.[35]

Other contributing factors include:

- Sympathetic denervation in distal limbs leads to increased peripheral blood flow – hyperaemia and more inflammation.[36]
- Pre-existing osteopaenia has been seen in both type 1 and type 2 diabetes via a number of mechanisms,[36] and this predisposes the diabetic patient to microfracture.
- Abnormal loading mechanics.
  - » Oxidative stress and the formation of reactive oxidant species (ROS) with local dysregulation of immunoinflammatory processes.
  - » AGE/RAGE pathway – hyperglycaemia generates AGEs. These promote change in both intra- and extracellular proteins, which become defective and lose functionality. Binding of AGE to receptors for AGE (RAGE) accelerates ROS production, activating NF-κB. An increase in AGE-modified collagen has been shown to affect osteoblastic cell differentiation and function in vitro. This may play a role in the pathogenesis of osteopaenia, which is present in patients with poorly controlled diabetes.
  - » PI3 kinase pathway – PI3 kinase normally impedes inflammation via enhanced nitric oxide (NO) production. NO may cause apoptosis of osteoclast progenitors as well as inhibit the resorptive action of mature osteoclasts. The PI3 kinase and NO pathway may be compromised in diabetes.
  - » Altered Wnt/β-catenin pathway (an important bone anabolic pathway).



oxPTM: oxidative post-translational modifications
BMD: bone mineral density
FLS: fibroblast-like synoviocytes
MMPs: matrix metalloproteinases
DNOAP: diabetic neuropathic osteoarthropathy

FIGURE 1.16
Detailed mechanism behind the development of Charcot foot

From Zhao H-M, Diao J-Y, Liang X-J et al. Pathogenesis and potential relative risk factors of diabetic neuropathic osteoarthropathy. Journal of Orthopaedic Surgery and Research *2017; 12: 142.*

» Altered fibroblast-like synoviocytes (FLS).

» OPG genetic polymorphisms.[35]

Sign value

Patients with Charcot foot are at higher risk of diabetic foot ulcers (affecting up to 50% of patients)[37,38] and amputation.[36]

The presence of foot pain, heat and/or swelling in the diabetic patient needs immediate attention including referral to diabetic/high-risk foot clinics or orthopaedic and diabetic specialist services.

Crepitus

Description

Grating, crunching, popping or crackling sounds heard and/or felt over joints during passive range of motion examination.

Condition/s associated with

- Arthropathy
  - » Osteoarthritis
  - » Rheumatoid arthritis
- Trauma
  - » Cartilaginous injury – meniscal injury, labral injury
  - » Ligamentous injury – anterior cruciate ligament
  - » Fracture

General mechanism/s

Crepitus of the joints is caused when *two rough surfaces grind against one another.*

Rheumatoid/osteoarthritis

In both rheumatoid arthritis and osteoarthritis arthritis, degeneration of the articular cartilage of the joint surfaces occurs, creating erosions and irregularity. Two rough surfaces moving against each other produce crepitus.

In rheumatoid arthritis, the autoimmune response and subsequent inflammation, cytokine release and pannus formation cause destruction of cartilage.

In osteoarthritis, repetitive strain with loss of glycosaminoglycans and activation of matrix metalloproteinases (MMPs) is principally responsible for damage.

Sign value

Altman R et al. reported crepitus had a sensitivity of 89%, specificity of 58%, positive likelihood ratio of 3.0 and negative likelihood ratio of 0.2 for predicting osteoarthritis of the knee.[39] Crepitus is common in patients with osteoarthritis. Crepitus alone has limited diagnostic value, due to its presence in other common disease states.

Dropped arm test

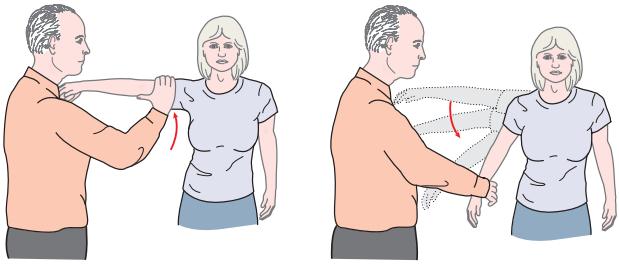


FIGURE 1.17
Dropped arm test

Based on Multimedia Group LLC, Occupation Orthopedics. *Available: http://www.eorthopod.com/eorthopodV2/index.php?ID=7244790ddace6ee8ea5da6f0a57f8b45&disp_type=topic_detail&area=6&topic_id=4357b9903d317fcb3ff32f72b24cb6b6 [28 Feb 2011].*

Description

With the patient upright, the examiner passively moves the patient's arm to 90° of abduction. Then the patient is asked to slowly lower the arm to the anatomical position. A positive test occurs if the patient is unable to perform the action due to pain or if the arm just 'drops' to the side.

Condition/s associated with

- Rotator cuff muscle injury (e.g. supraspinatus muscle)
- Subacromial impingement
- Neurogenic weakness
- Suprascapular nerve palsy
- Axillary nerve palsy
- C5 radiculopathy

Mechanism/s

Abduction of the arm from 0° to 90° is dependent upon the supraspinatus and deltoid muscles. The supraspinatus is responsible for the first 15° of motion. The deltoid muscle is responsible for movement beyond 15°.[40] Therefore, if a rotator cuff tear (e.g. supraspinatus muscle tear) or subacromial impingement is present, the ability of the arm to maintain abduction is impaired.

Sign value

Murrell GAC et al. and Dinnes J et al. reported a sensitivity of 10% and specificity of 98%, and a calculated positive likelihood ratio greater than 10 for rotator muscle tear.[41,42] Park HB et al. reported a sensitivity of 27%, specificity of 88%, positive likelihood ratio of 2.3 and negative likelihood ratio of 0.8 for subacromial impingement.[43]

When positive, the dropped arm test significantly increases the probability of rotator cuff muscle tear (supraspinatus muscle tear) or subacromial impingement. A negative test does not reliably exclude the diagnosis.

Eichhoff's test

FIGURE 1.18
Eichhoff's test

Description

The patient places their thumb within the palm of the examiner's hand, who grasps it tightly. The hand is then abducted towards the ulna by the examiner (see Figure 1.18).

Condition/s associated with

- De Quervain's tenosynovitis

Mechanism/s

De Quervain's tenosynovitis is an inflammatory condition of the contents of the first extensor synovial compartment: the tendons of abductor pollicis longus and extensor pollicis brevis.

Repetitive strain injury or inflammatory disorders cause inflammation, leading to swelling over the radial aspect of the wrist. This narrows the space that the abductor pollicis longus and extensor pollicis brevis pass through on their way to the hand.

This manoeuvre and Finkelstein's test involve generation of a passive distension and shear stress between the tendons and radius on its blunt styloid edge. In essence, the abductor pollicis longus and extensor pollicis brevis tendons are moved into the narrowed compartment and stretched, causing pain.

Sign value

There is limited data on the evidence of Eichhoff's test's diagnostic accuracy in diagnosing de Quervain's tenosynovitis. One small study suggested Eichhoff's test was associated with more false positives than Finkelstein's test.[44]

Finkelstein's test

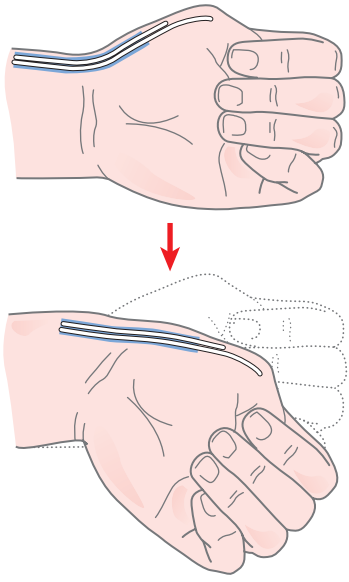


FIGURE 1.19
Finkelstein's test
With the thumb inside the hand, the wrist is ulnarly deviated. Pain indicates a positive test.

Based on Frontera WR, Silver JK, Rizzo Jr TD, Essentials of Physical Medicine and Rehabilitation, *2nd edn, Philadelphia: Saunders, 2008: Fig 24-2.*

Description

The examiner applies force at the patient's thumb metacarpal, placing the wrist into forced ulnar deviation. Tenderness with the manoeuvre at the radial aspect of the wrist (at the abductor pollicis longus tendon or extensor pollicis brevis tendon) is considered a positive test result.

Condition/s associated with

- De Quervain's tenosynovitis

Mechanism/s

De Quervain's tenosynovitis is an inflammatory condition of the contents of the 1st extensor synovial compartment: abductor pollicis longus and extensor pollicis brevis tendons.

Repetitive strain injury or inflammatory disorders cause inflammation that, in turn, causes swelling over the radial aspect of the wrist. This narrows the space through which the abductor pollicis longus and extensor pollicis brevis pass on their way to the hand. When performing this manoeuvre, the abductor pollicis longus and extensor pollicis brevis tendons are moved into the narrowed compartment and stretched, causing pain.[45]

Sign value

There is limited data on the evidence for Finkelstein's test in diagnosing De Quervain's tenosynovitis. De Quervain's tenosynovitis is a clinical diagnosis.

Gottron's papules

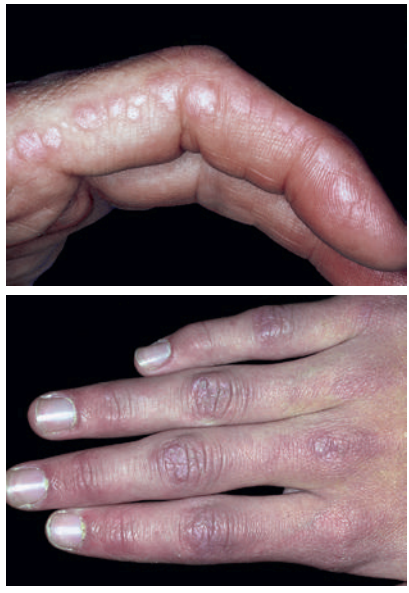


FIGURE 1.20
Gottron's papules
Found over bony prominences: fingers, elbows and knees. The lesions are slightly elevated, violaceous papules with slight scale.

Reproduced, with permission, from Habif TP, Clinical Dermatology, *5th edn, Philadelphia: Mosby, 2009: Figs 17-20, 17-21.*

Description

Violaceous (violet-coloured) papular rash on the dorsal aspect of the interphalangeal joints.[46]

Condition/s associated with

- Dermatomyositis

Mechanism/s

One histological study[47] demonstrated lymphocytic infiltration, epidermal atrophy and vacuoles in the basal layer of the skin, in addition to other findings. The mechanism is unknown.

Sign value

Gottron's papules are said to be pathognomonic for dermatomyositis; however, they are not present in all patients with the disease.[48]

CLINICAL PEARL

Hawkins' impingement test

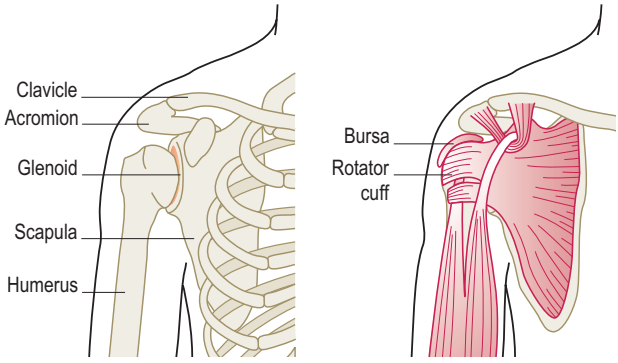


FIGURE 1.21
Hawkins' test anatomy

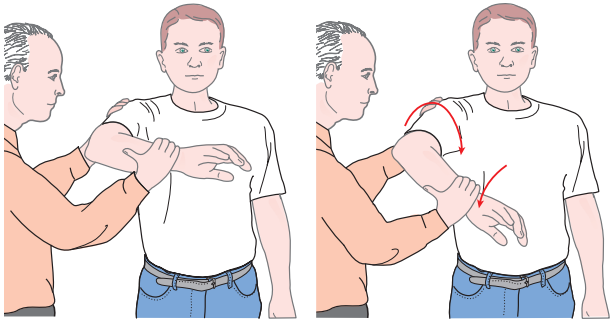


FIGURE 1.22
Hawkins' test

Description

With the patient upright, shoulder and elbow both flexed to 90°, the examiner internally rotates the shoulder joint. The sign is positive if tenderness is elicited (see Figure 1.22).

Condition/s associated with

- Rotator cuff muscle impingement – supraspinatus, teres minor, infraspinatus muscles
- Rotator cuff tendonitis

Mechanism/s

The tendons of the rotator cuff muscles pass through a narrow space between the acromion process of the scapula, bursa and the head of the humerus. Hawkins' impingement test exacerbates narrowing in the coracoacromial space and will worsen pre-existing impingement of the tendons and muscles when present. The position advances the greater tuberosity towards or against the coracoacromial ligament. This manoeuvre will also elicit tenderness when rotator cuff tendonitis

is present, due to mechanical forces or compression on the injured tendon or muscle.[49]

Sign value

Calis M et al. reported a sensitivity of 92% and a specificity of 26–44% for identifying rotator cuff tendonitis.[50] Macdonald PB et al. reported a sensitivity of 83% and a specificity of 51% for NLR of 0.3 for rotator cuff tear.[51]

A pooled analysis of 1029 patients across six studies by Alqunaee M et al.[52] reported a sensitivity of 74% and specificity of only 57%. Given these results, the test is of little value to the examiner, while a negative test has moderate utility. Further physical examination and imaging should be used to confirm the diagnosis.

Heliotrope rash

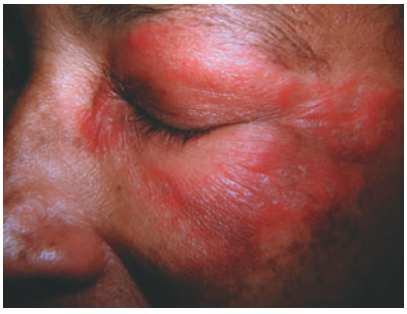


FIGURE 1.23
Heliotrope eruption seen in dermatomyositis

Reproduced, with permission, from Firestein GS, Budd RC, Harris ED et al., Kelley's Textbook of Rheumatology, *8th edn, Philadelphia: WB Saunders, 2008: Fig 47-10.*

Description

Usually described as a macular, confluent, purple or violaceous rash over both eyelids and periorbital tissue. It can present with or without oedema.

Condition/s associated with

- Dermatomyositis
- Paraneoplastic syndrome

Mechanism/s

The mechanism is unknown but thought to be autoimmune in origin. Skin lesions demonstrate perivascular CD4 positive T-cell infiltration in the dermis.[53]

Sign value

Occurring in up to 83% of cases in a specific European region,[54] the heliotrope rash is thought to be pathognomonic of dermatomyositis and should trigger further investigation.

Skin changes associated with dermatomyositis may precede muscle weakness, EMG abnormalities and elevations in creatinine phosphokinase by weeks or months.

CLINICAL PEARL

Kyphosis

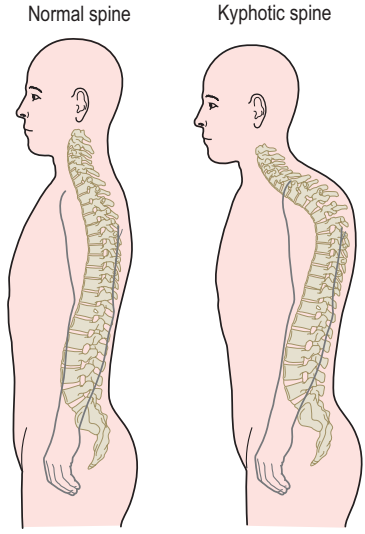


FIGURE 1.24
The normal and kyphotic spines
Note the prominent convexity of the kyphotic spine.

Description

Abnormally pronounced convex curvature of the thoracic spine as seen from the side. Kyphosis may be visible from any direction when severe. Often referred to in elderly females as the 'dowager's hump'.

Condition/s associated with

More common

- Osteoporosis/degenerative joint disease
- Traumatic – vertebral body fracture

Less common

- Ankylosing spondylitis
- Congenital
- Scheuermann kyphosis

Mechanism/s

Narrowing of the anterior aspect of the vertebral body is common in most forms of kyphosis.

Osteoporosis/degenerative joint disease

In degenerative or osteoporotic kyphosis, poor posture, mechanical straining and osteoporosis result in degeneration and/or compression fractures of the vertebrae. There is a relative loss of height of the anterior aspect of the vertebral body, leading to increased thoracic kyphosis.

Congenital kyphosis

Congenital kyphosis results from either a *failure of formation or a failure of segmentation* of the vertebral body elements.[55] In failure of segmentation, the anterior part of the vertebral body fails to separate from the vertebral body below, resulting in anterior fusion of the anterior aspect of the vertebrae. The posterior aspect continues to grow, resulting in kyphosis.[55]

Scheuermann kyphosis

Scheuermann kyphosis is a form of adolescent kyphosis. The mechanism behind Scheuermann kyphosis is multifactorial,[56] including:

- herniation of vertebral disc material into the vertebral body, causing decreased vertebral height and increased pressure anteriorly, leading to abnormal growth and wedging of the vertebrae
- a thickened anterior ligament
- abnormal collagen matrix.

Sign value

Kyphosis in paediatric patients may be suggestive of congenital kyphosis, which can have serious complications and lead to significant disability if left untreated. Acute worsening in the degree of kyphosis in an elderly patient should prompt consideration of pathological fracture.

Lachman's test

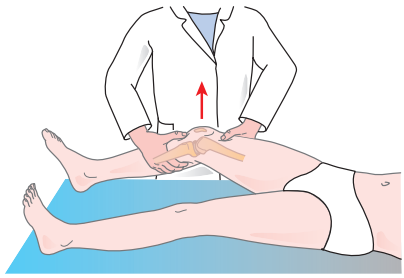


FIGURE 1.25
Lachman's test of the anterior cruciate ligament (ACL)
With 20–30° knee flexion, the tibia is moved forward on the femur to test the integrity of the ACL.

Description

The patient lies supine with the knee at 20–30° flexion. The examiner immobilises the femur just above the knee with one hand and attempts to pull the proximal tibia anteriorly with the other hand; the thumb is placed upon the tibial tuberosity. The test is positive if there is anterior movement of the tibia without an abrupt stop.

Condition/s associated with

- Anterior cruciate ligament (ACL) injury

Mechanism/s

The ACL arises from the anterior aspect of the tibial plateau and inserts into the medial aspect of the lateral femoral condyle. It limits anterior movement of the tibia on the femur. If the ACL is intact, the tibia should not have significant forward movement; if it is ruptured, there will be inappropriate anterior movement of the tibia and knee joint instability.

Sign value

A review by McGee of five studies reported a sensitivity of 48–96%, a specificity of 90–99%, a positive likelihood ratio of 17.0 and a negative likelihood ratio of 0.2.[2]

A positive Lachman's test is strongly predictive of ACL injury (+LR 17.0).[2] In a patient with a high clinical suspicion of ACL injury despite a negative Lachman's test (–LR 0.2),[2] further evaluation is necessary (e.g. interval re-examination, MRI). In general, Lachman's test is considered the better examination manoeuvre for ACL injury when compared with the anterior drawer sign and pivot-shift test.[57] A more recent systematic review of six studies found a sensitivity of 81–89%, specificity of 91–100%, with a +LR of up to 42, but with wide confidence intervals.[58]

CLINICAL PEARL

Livedo reticularis

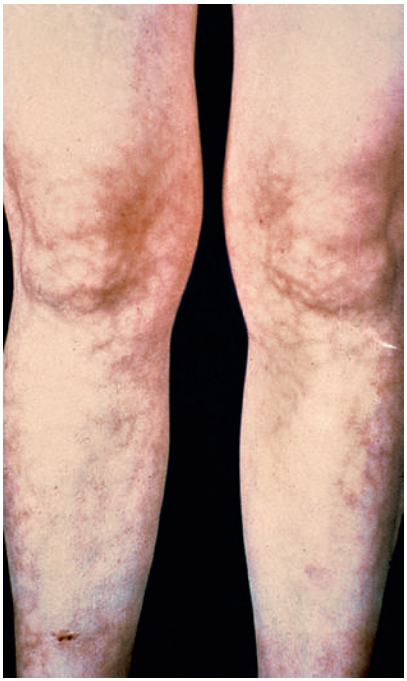


FIGURE 1.26
Livedo reticularis – a net-like pattern, often erythematous or violaceous in colour

Reproduced, with permission, from Floege J et al., Comprehensive Clinical Nephrology, 4th edn, Philadelphia: Saunders, 2010: Fig 64-13.

Description

A macular, bluish/purple discolouration of the skin that has a lacy or net-like appearance.

Condition/s associated with

More common

- Primary or idiopathic livedo reticularis (LR)
- Hypothermia
- Elderly

Less common

- Secondary LR

Present in numerous disorders including:

- Hypercoagulable state
  - » Antiphospholipid syndrome
  - » Cryoglobulinaemia
  - » Multiple myeloma
  - » DVT
- Microangiopathy/microangiopathic haemolytic anaemia (MAHA)
  - » Thrombotic/thrombocytopenic purpura (TTP)
  - » Haemolytic uraemic syndrome
  - » Disseminated intravascular coagulation
- Vasculitis/arteriopathy
  - » Snedden's syndrome
  - » Calciphylaxis
- Connective tissue disorders (e.g. SLE, dermatomyositis)
- Embolisation (e.g. cholesterol embolisation syndrome)
- Drug side effect
  - » Amantadine
  - » Quinine

General mechanism/s

Arterioles arising from the dermis divide to form a capillary bed. These capillaries then drain into the venules of the venous plexus. Livedo reticularis results from *increased visibility of the venules* of the skin. *Venodilatation of superficial venules* and *deoxygenation of blood* in the plexus are two main factors.[59]

In general, venodilatation is caused by altered autonomic nervous system function, circulating factors that cause

CLINICAL PEARL

venodilatation or in response to local hypoxia. Venodilatation results in engorged venules, making them larger and thus easier to see through the skin.

Deoxygenation is principally caused by decreased cutaneous perfusion,[59] which can be the result of decreased arteriolar inflow[60] or decreased venous outflow. These are caused by:

- *decreased arteriolar inflow* – vasospasm due to cold, autonomic nervous system activity, arterial thrombosis or increased blood viscosity
- *decreased venous outflow* – venous thrombosis, increased blood viscosity.

Primary or idiopathic livedo reticularis

LR without the presence of underlying disease or hypothermia is associated with spontaneous arteriolar vasospasm, which decreases oxygenated blood inflow, causing tissue hypoxia and increased deoxygenation of venous blood.[61]

Hypothermia (autonomic nervous system)

The normal physiological response to hypothermia is arteriolar vasospasm. This decreases arteriolar blood flow, local tissue hypoxia and venous plexus dilatation.

Elderly

The previous mechanisms apply to elderly patients, but with the added element of *thinning of the skin* that occurs with old age. This delicate and relatively translucent skin makes it more likely that the venous plexus will be visible.

Anti-phospholipid syndrome

Anti-phospholipid syndrome is associated with arterial and venous thrombosis, resulting in increased tissue hypoxia and venule dilatation (due to venous stasis).

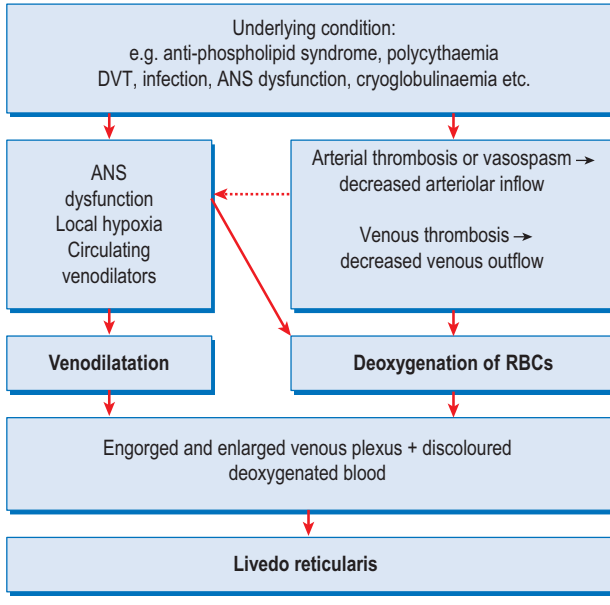


FIGURE 1.27
Mechanism of livedo reticularis