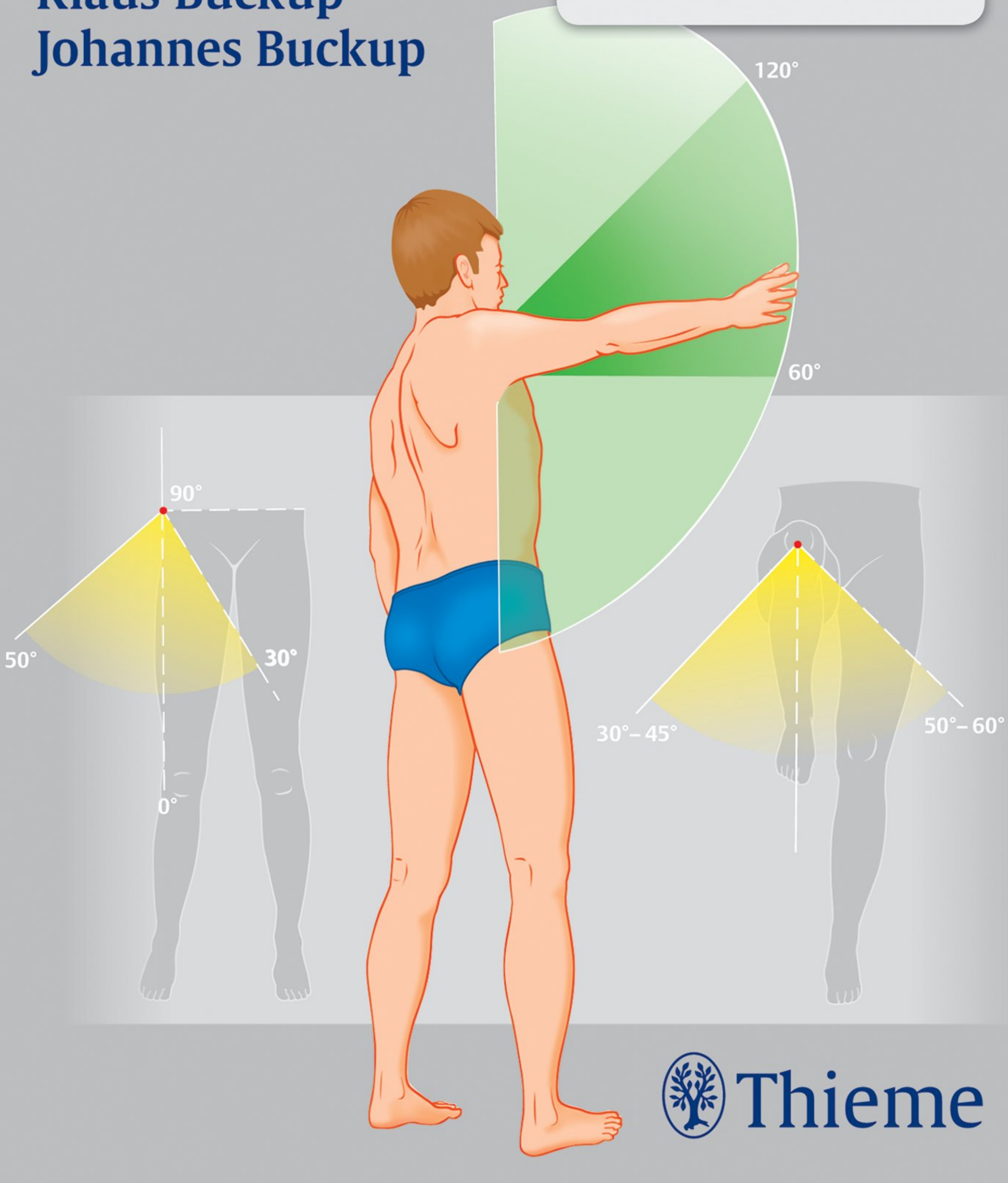
Clinical Tests for the Musculoskeletal System

Examinations—Signs—Phenomena

Third Edition

Klaus Buckup
Johannes Buckup



Thieme

Thieme

Clinical Tests for the Musculoskeletal System

Examinations—Signs—Phenomena

Third Edition

Klaus Buckup, MD †

Johannes Buckup, MD
Division of Trauma and Orthopaedic Surgery
BG Unfallklinik
Frankfurt am Main, Germany

614 illustrations

Thieme
Stuttgart · New York · Delhi · Rio de Janeiro

Library of Congress Cataloging-in-Publication Data is available from the publisher.

This book is an authorized translation of the German edition published and copyrighted 2012 by Georg Thieme Verlag, Stuttgart. Title of the German edition: Klinische Tests an Knochen, Gelenken und Muskeln. Untersuchungen, Zeichen, Phänomene

Translator: Alan Wiser, MD, Ambler, PA, USA
Illustrator: Heike Huebner, Berlin; Barbara Junghaehnel, Dortmund; Detlev Michaelis, Friedrichsdorf, Germany

1st Chinese edition 2010
2nd English edition 2008
2nd French edition 2010
1st Greek edition 2013
3rd Italian edition 2010
4th Polish edition 2014
1st Portuguese edition (Brazil) 2002
2nd Russian edition 2013
4th Spanish edition 2013

Thieme Publishers Stuttgart
Rüdigerstrasse 14, 70469 Stuttgart, Germany
+49 [0]711 8931 421,
customerservice@thieme.de

Thieme Publishers New York
333 Seventh Avenue, New York, NY 10001, USA
+1-800-782-3488,
customerservice@thieme.com

Thieme Publishers Delhi
A-12, Second Floor, Sector-2, Noida-201301
Uttar Pradesh, India
+91 120 45 566 00, customerservice@thieme.in

Thieme Publishers Rio, Thieme Publicações Ltda.
Edifício Rodolpho de Paoli, 25º andar
Av. Nilo Peçanha, 50 – Sala 2508
Rio de Janeiro 20020-906 Brasil
+55 21 3172 2297 / +55 21 3172 1896

Cover design: Thieme Publishing Group
Typesetting by primustype Robert Hurler GmbH, Notzingen, Germany

Printed in China by Asia Pacific Offset, Hong Kong
5 4 3 2 1
ISBN 9783131367938

Also available as an e-book:
eISBN 9783131494931

Important note: Medicine is an ever-changing science undergoing continual development. Research and clinical experience are continually expanding our knowledge, in particular our knowledge of proper treatment and drug therapy. Insofar as this book mentions any dosage or application, readers may rest assured that the authors, editors, and publishers have made every effort to ensure that such references are in accordance with **the state of knowledge at the time of production of the book.**

Nevertheless, this does not involve, imply, or express any guarantee or responsibility on the part of the publishers in respect to any dosage instructions and forms of applications stated in the book. **Every user is requested to examine carefully** the manufacturers' leaflets accompanying each drug and to check, if necessary in consultation with a physician or specialist, whether the dosage schedules mentioned therein or the contraindications stated by the manufacturers differ from the statements made in the present book. Such examination is particularly important with drugs that are either rarely used or have been newly released on the market. Every dosage schedule or every form of application used is entirely at the user's own risk and responsibility. The authors and publishers request every user to report to the publishers any discrepancies or inaccuracies noticed. If errors in this work are found after publication, errata will be posted at www.thieme.com on the product description page.

Some of the product names, patents, and registered designs referred to in this book are in fact registered trademarks or proprietary names even though specific reference to this fact is not always made in the text. Therefore, the appearance of a name without designation as proprietary is not to be construed as a representation by the publisher that it is in the public domain.

FSC
www.fsc.org
MIX
Paper from responsible sources
FSC® C012521

In memory of my father

Contents

Preface

Orthopaedic surgery and trauma surgery have advanced rapidly in the last few years. Given the multiplicity of instrumental investigative methods available, especially the imaging techniques (digital radiography, ultrasound, CT, and MRI), the importance of a good clinical examination is often forgotten, usually to the detriment of the patient.

The experienced musculoskeletal physician, however, is aware of the wide range of interpretations associated with imaging findings. The evaluation of a genuine functional impairment and its significance is not easily achieved without corresponding clinical findings and tests. Thus, clinical examination remains of key importance as the basis for a timely and accurate diagnosis. The popularity of the previous editions of *Clinical Tests for the Musculoskeletal System* and the number of editions of translations into other languages bear witness that this is widely and persistently appreciated. The fourth German edition was awarded the Carl Rabl Prize in 2010.

Nonetheless, anything can be improved upon. When becoming complacent it is not long before one realizes that others are already shifting gear and attempting to pass one by.

All the chapters have been revised for the present edition. New tests are included, and suggestions from readers of previous editions have also been considered.

The basic criteria that an examiner expects from a diagnostic test are precision, reliability, and reproducibility. The probability that the findings of a particular diagnostic test are true and accurate can be influenced by inherent factors of the test itself, such as specificity and sensitivity, and by the clinical situation in which the test is applied. In an extensive review of the literature, I have reviewed most of the tests for their clinical value; but, so as not to overburden the text, I have refrained from giving full details of the reliability and validity of individual tests. For these details, please consult the extensive bibliography at the end of the book.

Each examiner is encouraged to use those tests he or she has found to be clinically effective. Under no circumstances should special tests be used in isolation. They should be viewed as an integral part of the total examination. Some new tests that have been included in this edition are based upon reader feedback, which indicates that these tests have gained in popularity.

In addition to referring to evidence-based, peer-reviewed papers that evaluate the effectiveness of each test, additional comments and reference to other or similar clinical tests are sometimes included. This should help the reader to understand the historical or anatomical background of a test.

As we were working together on the fifth German edition of this book in 2010, my father, Klaus Buckup, the original author of this work, passed away. For his trust, his love, and his support, I am forever grateful.
I would like to thank Dr. Hans Paessler for the great support and assistance, Alan Wiser, MD, for his excellent translation, and Thieme Publishers Stuttgart, especially Ms. Gabriele Kuhn-Giovannini, Ms. Angelika-M. Findgott, Ms. Jo Stead, and Mr. Martin Teichmann for their excellent cooperation.

Johannes Buckup, MD

Contributors

Klaus Buckup, MD †

Johannes Buckup, MD
Division of Trauma and Orthopaedic Surgery
BG Unfallklinik
Frankfurt am Main, Germany

Hans H. Paessler, MD
Professor
Atos Hospital Heidelberg, Germany

Basic Principles

1. The examination must proceed in a systematic manner.
2. Evaluate the patient in terms of gait, posture, etc. as he or she enters the examination room.
3. Start with a winner: “What brings you to see me?”
4. The four important questions:
   - *How* did the pain first start and how does it express itself? (Spontaneous, after trauma, gradual, burning, sticking, etc.)
   - *Where* is the pain localized? (Localized, radiating →disk prolapse, originating in joint or muscle, etc.)
   - *When* do you feel the pain? (Mornings, night →e.g., calcium deposits in the shoulder, evening)
   - *What are you doing* when you get the pain? (After weight bearing, at rest, in certain positions, etc.)
5. Win the trust of the patient by explaining what will be examined, why, and how. Avoid provoking pain any more than necessary. The patient will be grateful.
6. Inspection:
   Do not touch the patient—state first what you see.—William Osler
   - Posture:
     - Shoulder contour.
     - Position of the pelvis.
     - Leg length difference.
     - Gait pattern (normal, unique to the individual, pathologic).
     - Limping (from pain, foreshortening or deformity; intoeing or outtoeing gait).
   - Deformities (axis malalignments, contracture deformities of the extremities or the spine).
   - Contours (areas of swelling, shrinkage, atrophy).
   - Skin (pigmentation, callus formation).
   - Anomalies.
7. Palpation:
   - Tender points.

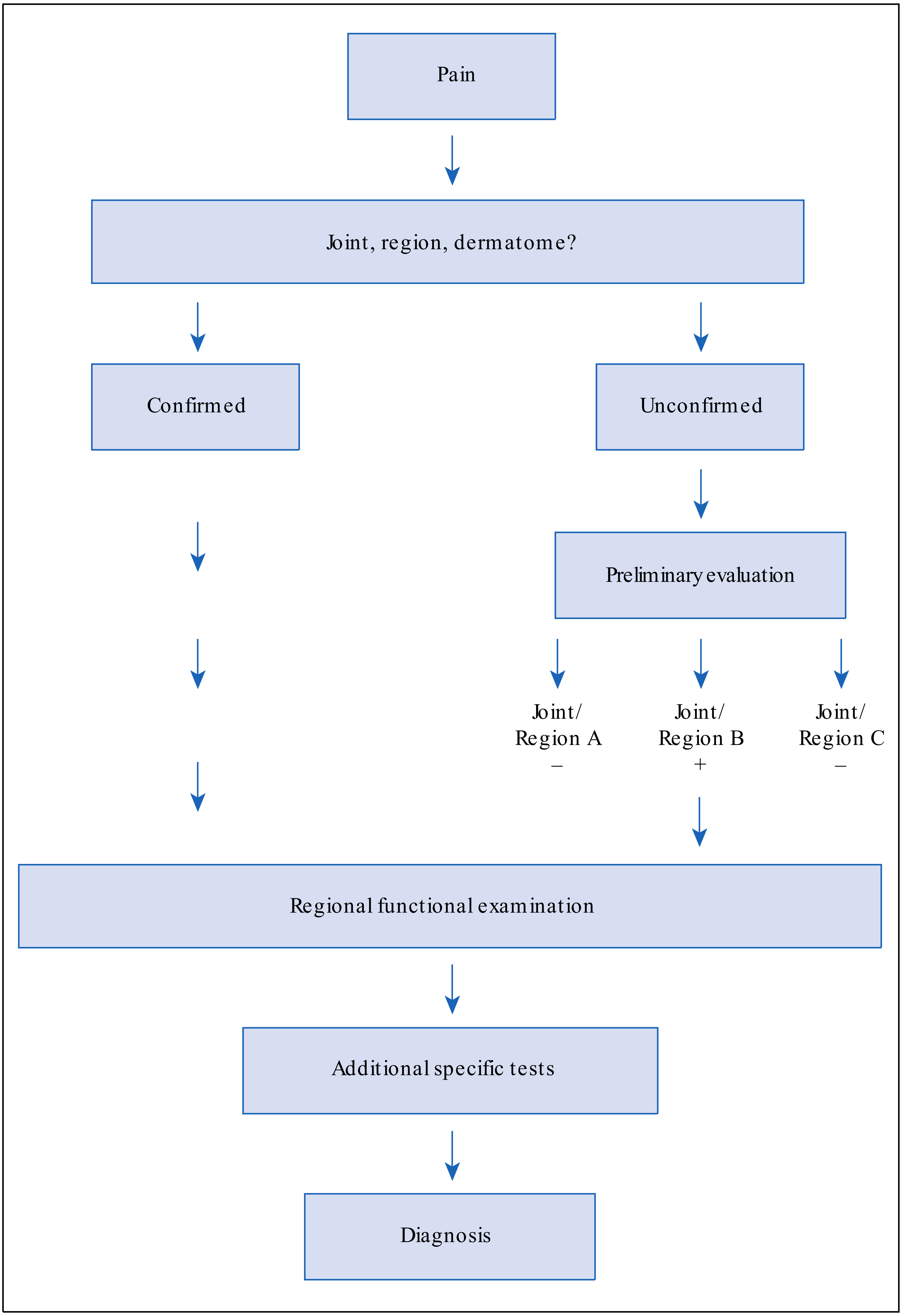
- Skin (temperature, sweat secretion).
- Areas of swelling.
- Effusion (intra-articular or extra-articular).
- Bulging (hard, firm, elastic).
- Crepitus (retropatellar → sign of arthritis).

8. Examination of the joints:
   - Test the healthy (uninvolved) side first. This approach enables an initial insight into the individual joint mobility. The patient experiences what it's like to be examined and gains trust.
   - First check the active range of movement (ROM), then the passive ROM, and lastly, ROM against resistance (document using the neutral-zero method).
   - Check ROM against resistance from a neutral joint position or from a relaxed body position.
   - Test the ROM several times to better differentiate the pain.
   - Test each active (individual) ROM and observe when and with which movement pains occur. For instance, pains occurring with shoulder abduction between 60 and 120° imply subacromial impingement.
   - Check passive (anatomical) range of motion. As a rule, this is somewhat larger than the active ROM. Compare the right and left sides—hypomobile/hypermobile (lax), stable/unstable.
   - Evaluate the "end-feel" of the movement:
     - Bony → restricted mobility caused by osteophyte formation, as of elbow ROM or cervical spine rotation.
     - Elastic → synovium, edema.
     - Capsular (frozen shoulder, for example).
     - Spastic (reflex spasticity after injury).
     - Elastic (pinched meniscus, for example).
   - Test sensation, motor function, mono- and polysynaptic reflexes, and the peripheral pulses.
   - Check the development of muscular strength using active and passive range of motion to evaluate for neurologic deficits. Use the following muscle status survey to quantify the muscular strength of the individual voluntary muscles of an extremity.

Muscle performance scale

Degree	Magnitude	Percentage of normal muscle strength (%)	Clinical neurologic findings
5	Normal	100	Full ROM against strong resistance
4	Good	75	Full ROM against mild resistance
3	Weak	50	Full ROM against gravity
2	Very weak	25	Full ROM without the influence of gravity
1	Negligible	10	Incomplete visible and palpable ROM
0	None	0	Complete paralysis, no contraction

Abbreviation: ROM, range of movement.



From pain to diagnosis.

1 Spine

Differential diagnosis of back pain is often a daunting task given the wide range of possible causes that must be considered. Terms such as "cervical spine syndrome" or "lumbar spine syndrome" are ambiguous as they identify neither the location nor the nature of the disorder.

Once the history has been taken, any examination of the spine should be preceded by a general physical examination. This is required to properly evaluate those changes in the spine that are attributable to causes elsewhere in the body such as in the limbs and muscles. The examination begins with inspection. General body posture is noted, and the position of the shoulders and pelvis (level of the shoulders, symmetry of both shoulder blades, level of the iliac crests, lateral pelvic obliquity), vertical alignment of the spine (any deviation from vertical), and the profile of the back (kyphotic or lordotic deformity, or absence of physiologic kyphosis and/or lordosis) are evaluated. Palpation can detect changes in muscle tone such as contractures or myogelosis and can identify tender areas. The active and passive mobility of the spine as a whole and the mobility of specific segments are then evaluated.

In patients presenting with a spine syndrome, the first step is to identify the location and nature of the disorder. Tissue destruction, inflammation, and severe degenerative changes usually exhibit a characteristic clinical picture with corresponding radiographic and laboratory findings. A number of additional diagnostic modalities can supplement plain-film radiography in cases where further diagnostic studies are indicated to confirm or exclude a tentative diagnosis. The choice of additional imaging modalities depends on the line of inquiry. For example, computed tomography with its higher contrast between bone and soft tissue is more suitable for visualizing changes in bone than is magnetic resonance imaging, whose advantage lies in its high-resolution visualization of soft tissue. Dysfunctional muscular and ligamentous structures render the clinical evaluation of spine syndromes more difficult.

Radiographic and laboratory findings alone are rarely able to provide a conclusive diagnosis in these spinal disorders. This makes manual diagnostic techniques that focus on evaluation of function particularly important. The examiner evaluates changes in the skin (hyperalgesia and characteristics of the paraspinal skin fold, also known as the Kibler fold), painful muscle spasms, painfully restricted mobility with loss of play in the joint, functional impairments with painful minimal mobility, and radicular pain. The examination evaluates each part of the spine as a whole (cervical, thoracic, and lumbar) and each segment individually.

Because every pair of adjacent vertebrae is connected by many ligaments, only limited motion is possible in any one intervertebral joint. However, the sum of all the movements in the many vertebral articulations results in significant mobility in the spinal column and trunk as a whole. This mobility varies considerably between individuals (**Fig. 1.1**). The main motions are flexion and extension in the sagittal plane, lateral bending in the coronal plane, and rotation around the longitudinal axis. The cervical spine exhibits the greatest range of motion. It is both the most highly mobile portion of the spine and the one most susceptible to spinal disorders.

About 50% of flexion and extension occurs between the occiput and C1. The other 50% is distributed evenly along the other cervical vertebrae, especially C5 and C6.

About 50% of rotation occurs between C1 (atlas) and C2 (axis). The other 50% is distributed evenly along the other five cervical vertebrae.

Rotation and lateral bending in the thoracic spine occur primarily in the lower thoracic spine and the thoracolumbar junction. The lumbar spine with its sagittally aligned facet joints primarily allows flexion and extension (forward and backward bending) and lateral bending. The capacity for rotation is less well developed in this portion of the spine.

Neurologic examination can exclude sensory deficits and palsies of the lower extremities. In addition to eliciting intrinsic reflexes, this includes testing for nerve stretching signs.

When examining the spine, the physician must consider the possibility that “back pain” may in fact be referred pain caused by pathology in other areas.

Range of Motion of the Spine (Neutral-Zero Method)

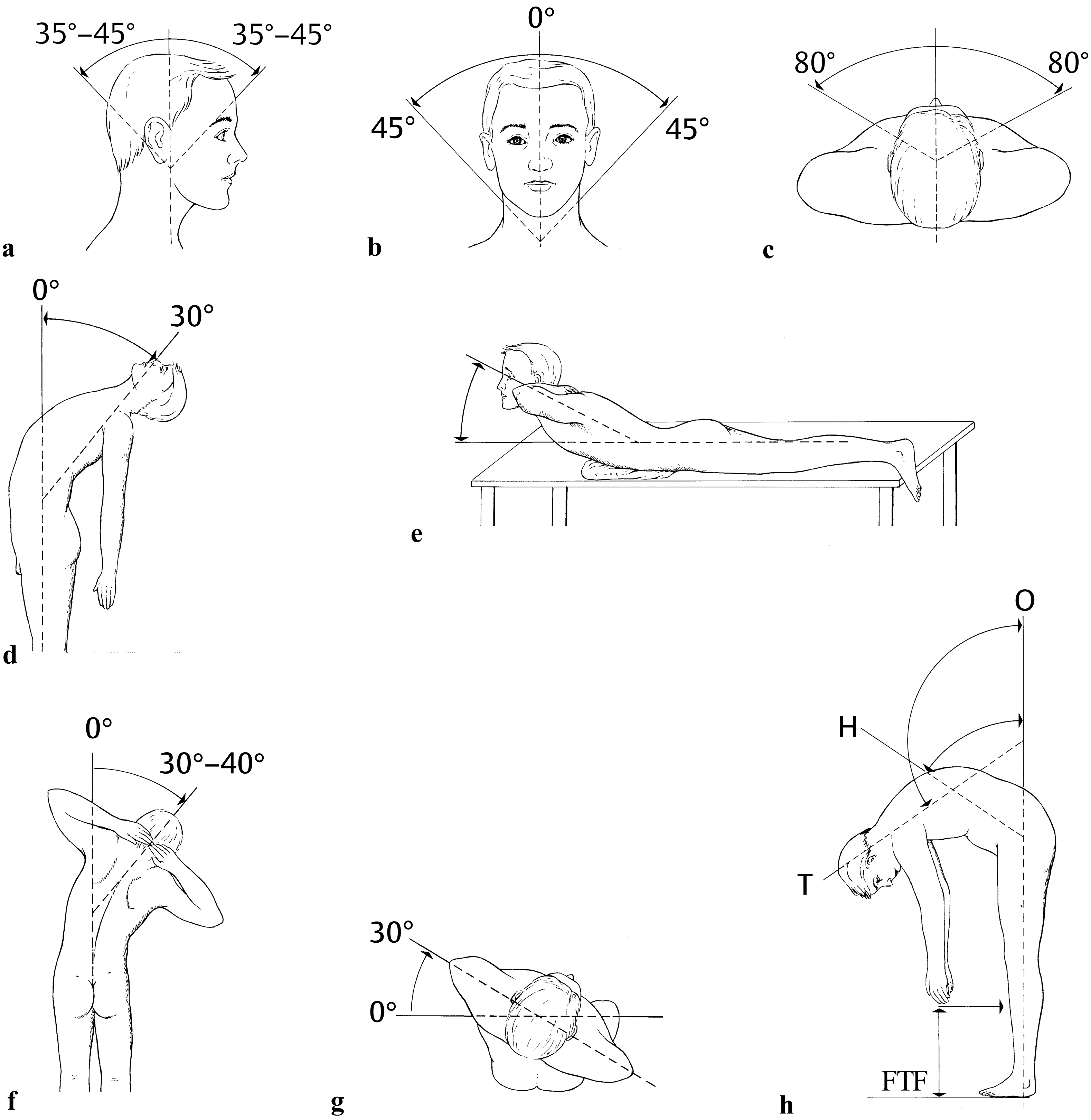


Fig. 1.1 (a) Forward and backward bending (flexion and extension). **(b)** Lateral bending. **(c)** Rotation in middle position 80/0/80°, rotation in flexion 45/0/45° (C0–C1), rotation in extension 60/0/60°. **(d, e)** Backward bending (extension) of the spine: standing (d) and prone (e). **(f)** Lateral bending of the spine. **(g)** Rotation of the trunk. **(h)** Forward bending of entire spine: H, flexion in hip; T, total excursion, FTF, distance between fingertips and floor.

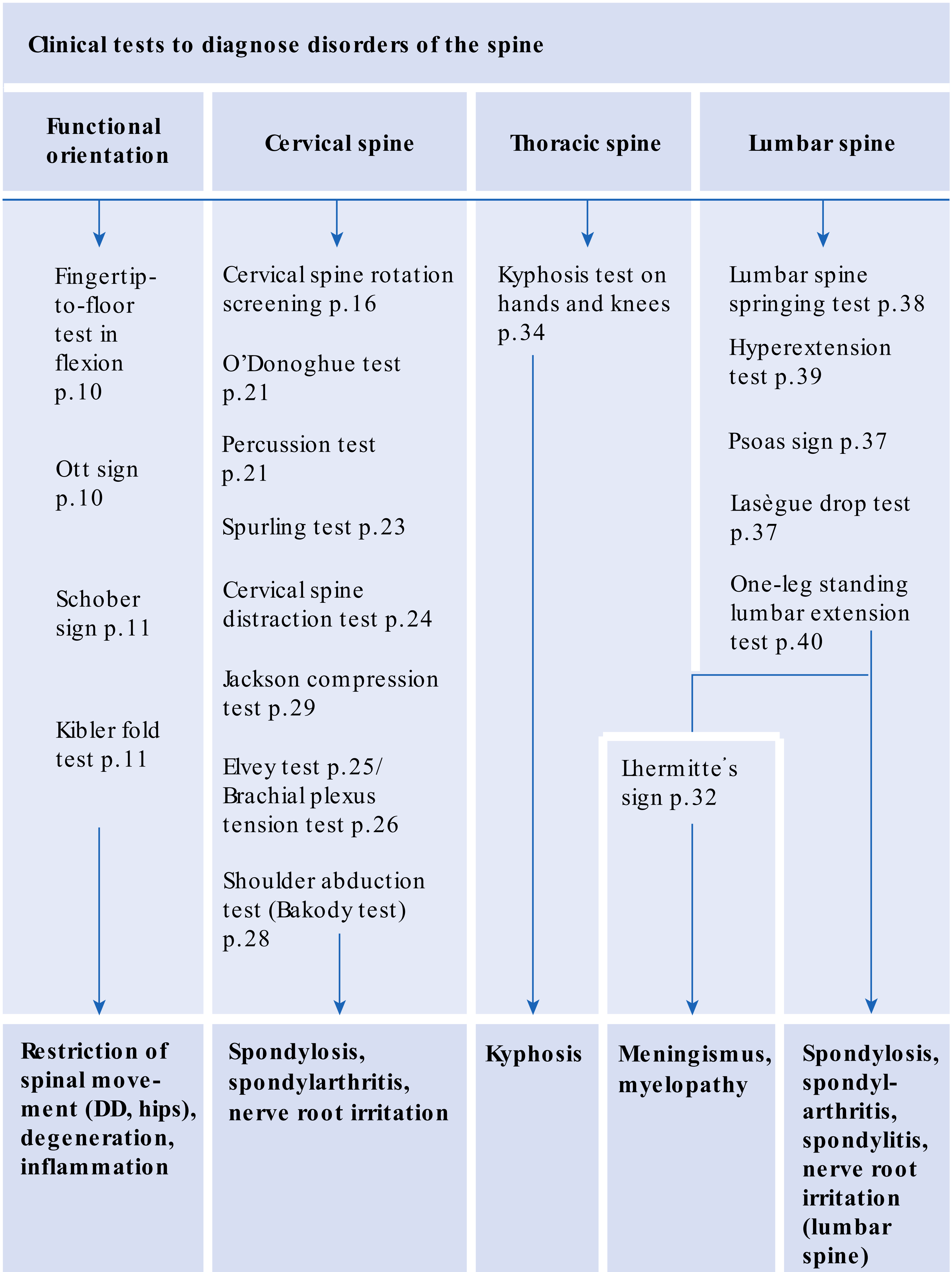
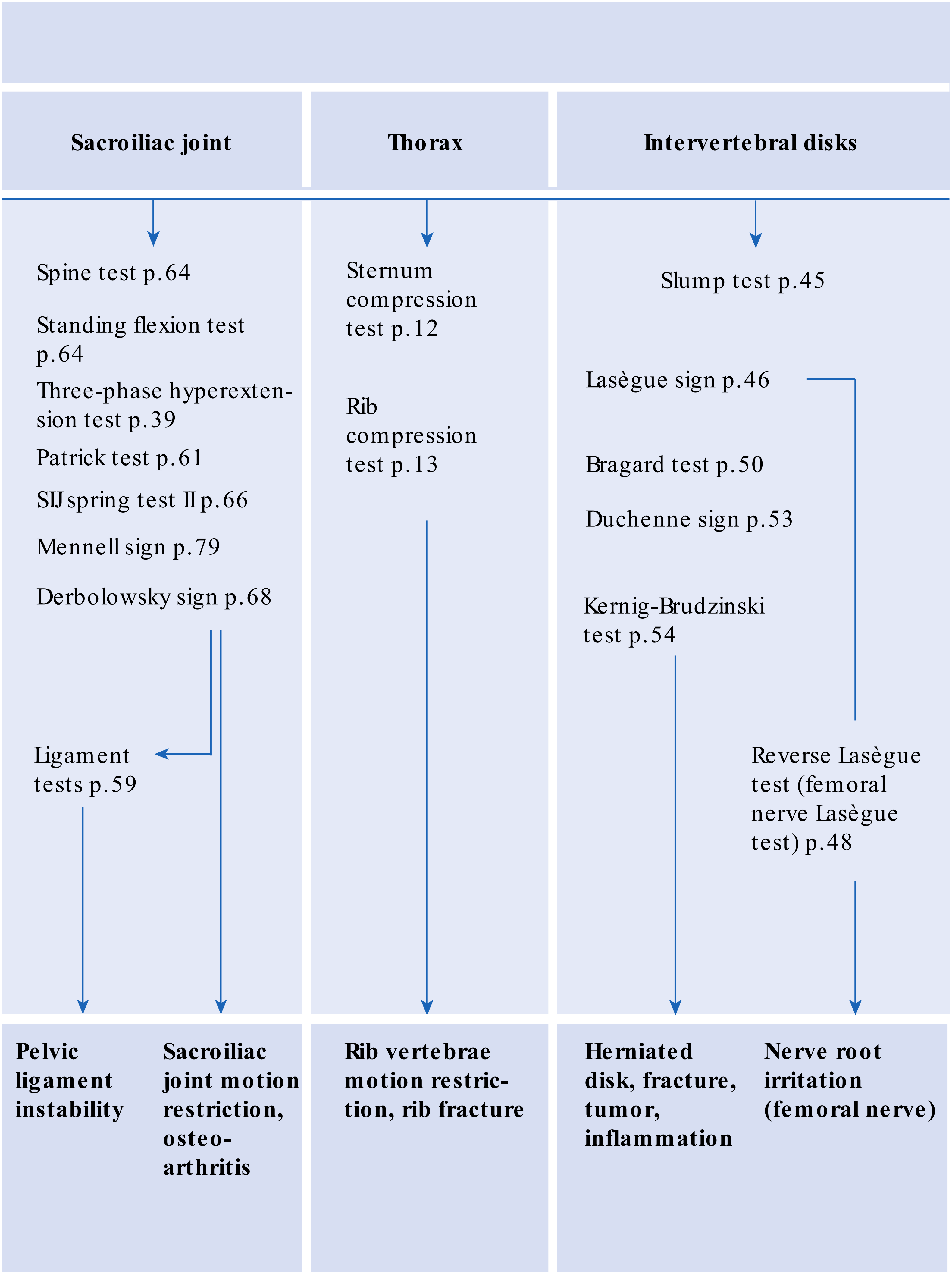


Fig. 1.2 Clinical tests to diagnose disorders of the spine.


Sacroiliac joint
Thorax
Intervertebral disks
Spine test p.64
Standing flexion test p.64
Three-phase hyperextension test p.39
Patrick test p.61
SIJ spring test II p.66
Mennell sign p.79
Derbolowsky sign p.68
Ligament tests p.59
Sternum compression test p.12
Rib compression test p.13
Slump test p.45
Lasègue sign p.46
Bragard test p.50
Duchenne sign p.53
Kernig-Brudzinski test p.54
Reverse Lasègue test (femoral nerve Lasègue test) p.48
Pelvic ligament instability
Sacroiliac joint motion restriction, osteo-arthritis
Rib vertebrae motion restriction, rib fracture
Herniated disk, fracture, tumor, inflammation
Nerve root irritation (femoral nerve)

Fingertip-to-Floor Distance Test in Flexion

Measures the mobility of the entire spine when bending forward (fingertip-to-floor distance in centimeters).

□ **Procedure:** The patient may be standing or seated on the examination table. When the patient bends over with the knees fully extended, both hands should come to rest at approximately the same distance from the feet. The distance between the patient's fingers and the floor is measured, or how far the patient's fingers reach may be recorded (to the knee, midtibia, etc.; **Fig. 1.1h**).

□ **Assessment:** This mobility test assesses a combined motion involving both the hips and the spine. Good mobility in the hips can compensate for stiffening in the spine. In addition to the distance measured, the profile of the flexed spine should also be assessed (uniform kyphosis or fixed kyphosis).

A great distance between the fingertip and floor is therefore a nonspecific sign that is influenced by several factors:

1. Mobility of the lumbar spine.
2. Shortening of the hamstrings.
3. Presence of the Lasègue sign.
4. Hip function.

Clinically, the fingertip-to-floor distance is used to assess the effect of treatment.

Ott Sign

Measures the range of motion of the thoracic spine.

□ **Procedure:** The patient is standing. The examiner marks the C7 spinous process and a point 30 cm inferior to it. This distance increases by 2 to 4 cm in flexion and decreases by 1 to 2 cm in maximum extension (leaning backward).

□ **Assessment:** Degenerative inflammatory processes of the spine restrict spinal mobility and hence the range of motion of the spinous processes.

Schober Sign

Measures the range of motion of the lumbar spine.

□ **Procedure:** The patient is standing. The examiner marks the skin above the S1 spinous process and a point 10 cm superior to it. These skin markings move up to about 15 cm apart in flexion and converge to a distance of 7 to 9 cm in maximum extension (leaning backward).

□ **Assessment:** Degenerative inflammatory processes in the spine restrict spinal mobility and hence the range of motion of the spinous processes.

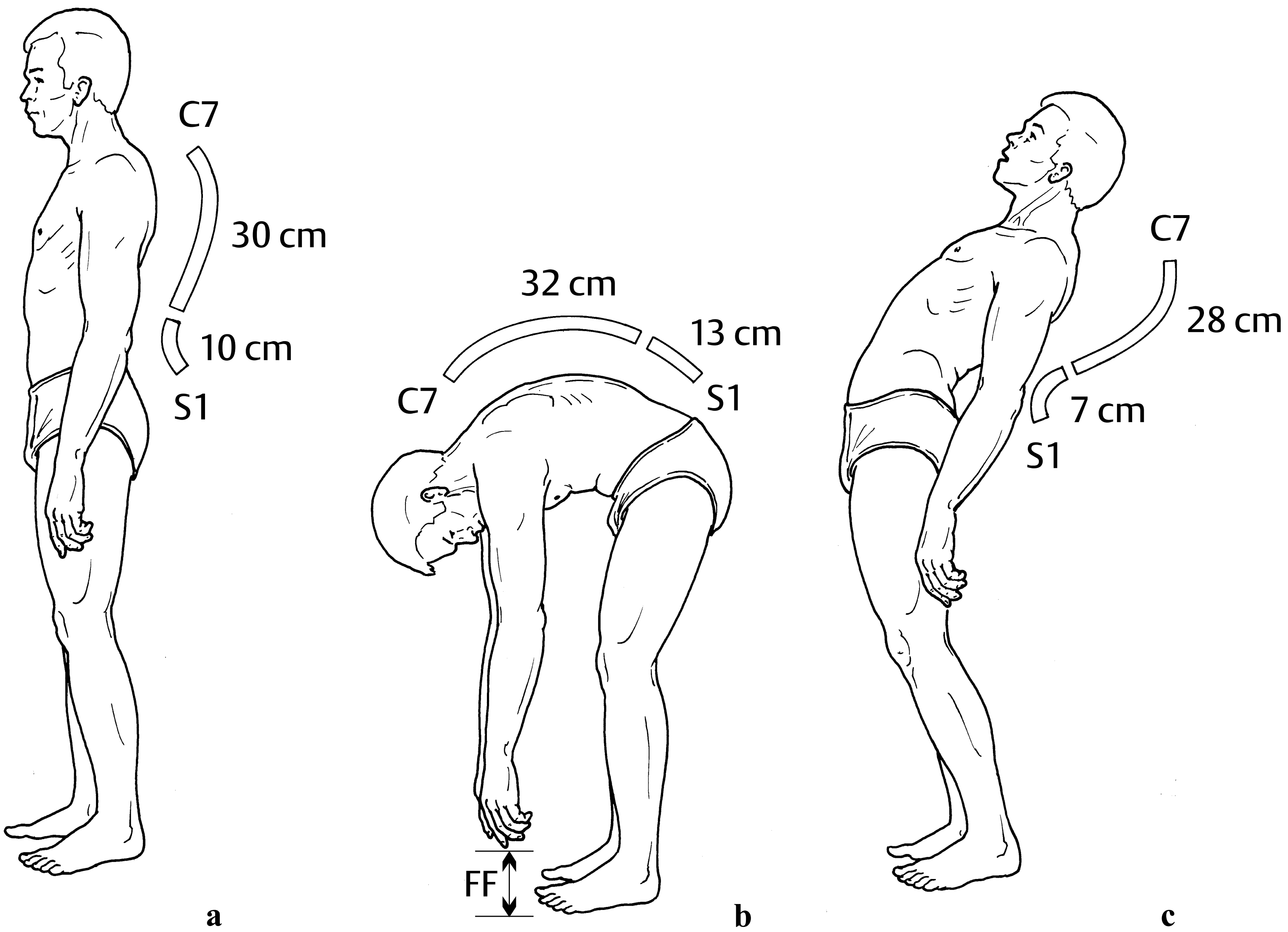


Fig. 1.3 Ott and Schober signs (fingertip-to-floor distance test). **(a)** Upright position. **(b)** Flexion. **(c)** Extension.

Skin-Rolling Test (Kibler Fold Test)

Nonspecific back examination.

□ **Procedure:** The patient lies prone with arms relaxed alongside the trunk. The examiner raises a fold of skin between thumb and forefinger and "rolls" it along the trunk or, on the extremities, perpendicular to the course of the dermatomes.

□ **Assessment:** This test assesses regional variation in how readily the skin can be raised, the consistency of the skin fold (rubbery or edematous), and any lack of mobility in the skin. Palpation can detect regional tension in superficial and deep musculature as well as autonomic dysfunction (such as localized warming or increased sweating). In areas of hypalgesia, the skin is less pliable, is more difficult to raise, and resists rolling. The patient reports pain. Areas of hypalgesia, tensed muscles, and autonomic dysfunction suggest vertebral disorders involving the facet joints or intercostal joints.

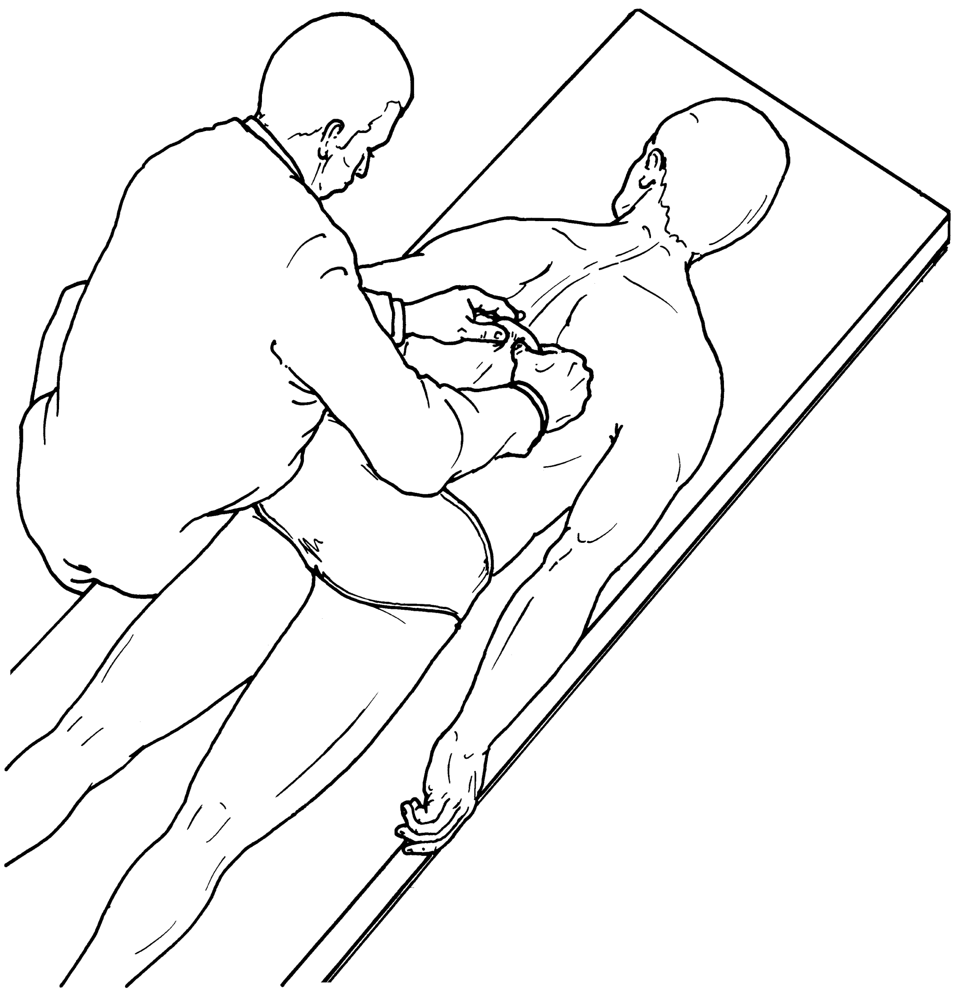


Fig. 1.4 Skin-rolling test (Kibler fold test).

Chest Tests

Sternum Compression Test

Indicates rib fracture.

□ **Procedure:** The patient is supine. The examiner exerts pressure on the sternum with both hands.

□ **Assessment:** Localized pain in the rib cage can be due to a rib fracture.

Pain in the vicinity of the sternum or a vertebra suggests impaired costal or vertebral mobility.

Rib Compression Test

Indicates impaired costovertebral or costosternal mobility or a rib fracture.

▫ **Procedure:** The patient is seated. The examiner stands or crouches behind the patient and places his or her arms around the patient's chest, compressing it sagittally and horizontally.

▫ **Assessment:** Compression of the rib cage increases the movement in the sternocostal and costotransverse joints and in the costovertebral joints. Performing the test in the presence of a motion restriction or other irritation in one of these joints elicits typical localized pain.

Fig. 1.5 Sternum compression test.

Fig. 1.6 Rib compression test.

Pain along the body of a rib or between two ribs suggests a rib fracture or intercostal neuralgia.

Chest Circumference Test

Measures the circumference of the chest at maximum inspiration and expiration.

▫ **Procedure:** The patient is standing or seated with arms hanging relaxed. The difference in chest circumference between maximum inspiration and expiration is measured. The circumference is measured immediately above the convexity of the breast in women, and immediately below the nipples in men.

The difference in chest circumference between maximum inspiration and expiration normally lies between 3.5 and 6 cm.

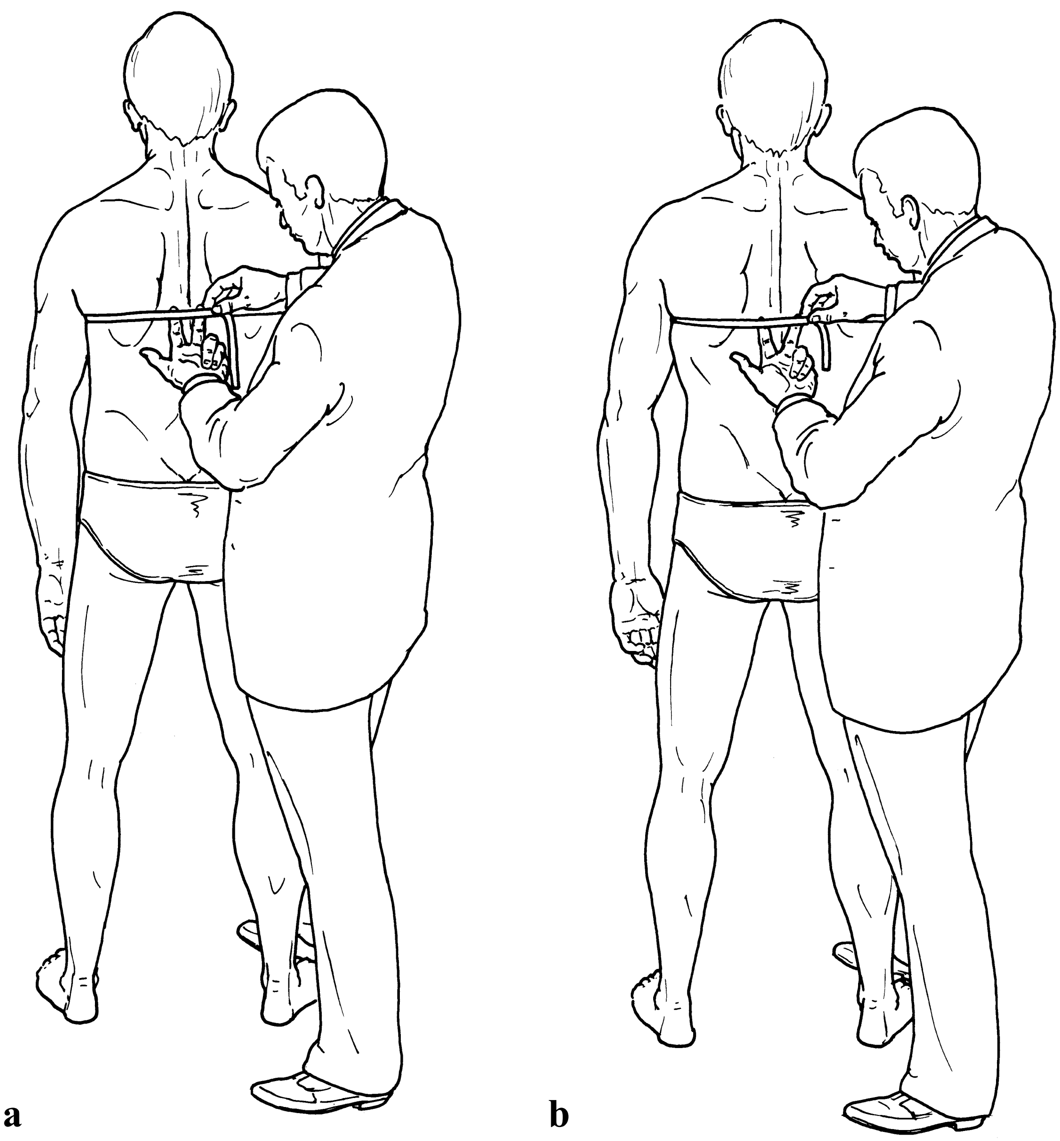
□ **Assessment:** Limited depth of breathing is encountered in ankylosing spondylitis, where the impairment of inspiration and expiration is usually painless. Impaired or painful inspiration and expiration with limited depth of breathing is observed in costal and vertebral dysfunctions (motion restricted), inflammatory or tumorous pleural processes, and pericarditis. Bronchial asthma and emphysema are associated with painless impaired expiration.

Schepelmann Test

For the differential diagnosis of chest pain.

□ **Procedure:** The patient is seated and is asked to bend first to one side, then to the other.

□ **Assessment:** Pain on the concave side is a sign of intercostal neuralgia. Pain on the convex side suggests pleuritis. Rib fractures are painful on any movement of the spine.



a b

Fig. 1.7 Chest circumference test. **(a)** At maximum expiration. **(b)** At maximum inspiration.

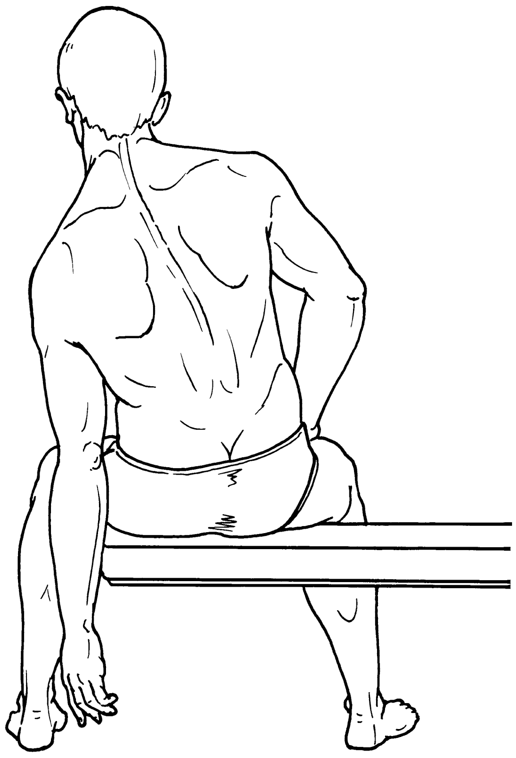


Fig. 1.8 Schepelmann test.

Cervical Spine Tests

The clinical picture of cervical spine disease is based primarily on degenerative changes of the intervertebral disks and the vertebral joints (spondylosis, spondylarthritis, uncovertebral joint arthrosis). Because of these changes, mechanical irritation and compression of adjacent neurogenic and vascular structures develop, resulting in cervical headaches and radicular cervical syndromes. Chronic degenerative changes of the cervical spine can affect the vertebral artery as well, in which case rotation of the head can at times cause compression of the vertebral artery. Dizziness, nausea, visual disturbances, syncope, and nystagmus are typical signs of ipsilateral vertebral artery narrowing or buckling (Barré–Liéou sign). The patient with a positive Barré–Liéou sign is a poor candidate for cervical spine manipulation.

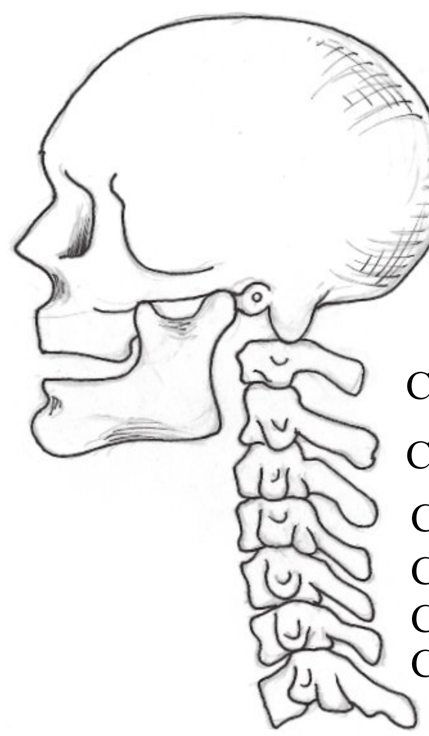


Fig. 1.9 Lateral view of the head and cervical spine.

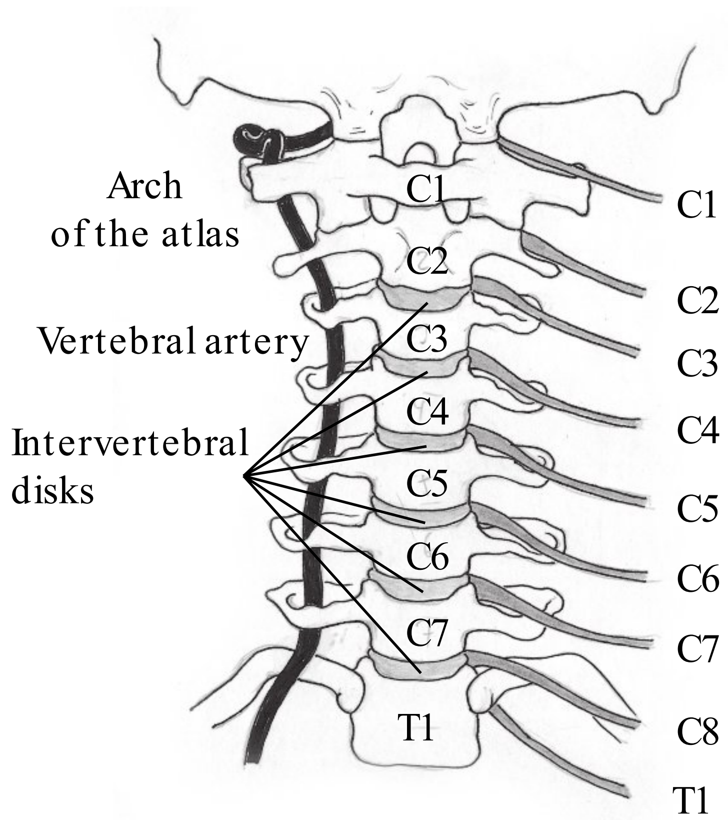


Fig. 1.10 Anterior view of the cervical spine: representation of the nerve root outlets and the course of the vertebral artery.

Screening of Cervical Spine Rotation

The range of movement (ROM) available in the cervical spine is the sum of the ROM between the head and C1 and that between the underlying pairs of vertebrae in the cervical spine. Many factors can influence the ROM of the cervical spine, including the flexibility of the intervertebral disks, the position of the articular processes of the facet joints, and the degree of laxity of the ligaments and the joint capsules. Except in flexion, women have a greater cervical ROM than men. Cervical ROM decreases with age. However, as opposed to other parts of the cervical spine, the range of rotation between C1 and C2 increases with age. The passive cervical ROM with the patient supine is greater than the active or passive ROM while in the upright position. This can be explained by the fact that, in the sitting position, the weight of the head that must be supported against gravity is increased, thus increasing the muscle tension as well. For this reason, passive ROM with overpressure should only be performed in parallel with active ROM testing. If passive ROM with overpressure is normal and pain free, the examiner may test the cervical spine for end-range extension, flexion, lateral flexion, and rotation. These directions of movement allow the examiner to evaluate the musculoligamentous structures of the anterior, posterior, and lateral neck as well as the vertebral artery. Overpressure at end-range ROM can evoke a variety of symptoms:

- Nerve root irritation (radicular symptoms).
- Apophyseal joint irritation (localized pain).
- Involvement of the vertebral artery (dizziness, nausea).

Table 1.1 Clinical diagnostic levels of radicular irritation in the cervical spine region

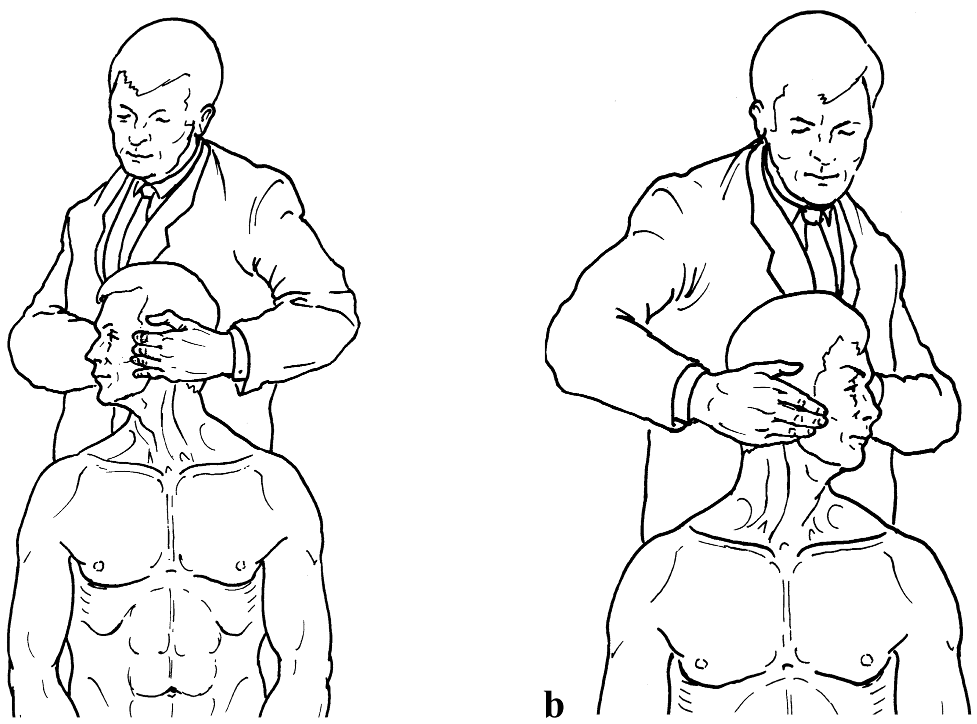
Flexion C1–C2 (cervical plexus)
Lateral flexion C1–C4 (cervical plexus)
Shoulder elevation C5–C7 (long thoracic nerve)
Shoulder abduction C4–C6 (axillary nerve)
Elbow flexion and/or wrist extension C5–C6 (musculocutaneous nerve, radial nerve)
Elbow extension and/or wrist flexion C7–C8 (radial nerve)
Thumb extension and/or ulnar deviation C6–C8 (radial nerve, deep branch of the ulnar nerve)
Abduction and/or adduction of the intrinsic muscles of the hand/wrist C8–T1 (ulnar nerve, deep branch of the ulnar nerve)

□ **Procedure:** The patient is seated and upright. The examiner holds the patient's head with both hands around the parietal region and, with the patient's neck slightly extended, passively rotates the patient's head to one side and then the other from the neutral position.

□ **Assessment:** The range of motion is determined by comparing both sides. The examiner also notes the quality of the end point of motion, which is resilient in normal conditions but hard when functional impairment is present.

Restricted mobility with pain is a sign of segmental dysfunction (arthritis, blockade, inflammation, or muscle shortening). Restricted rotation with a hard end point and pain at the end of the range of motion suggest degenerative changes, predominantly in the middle cervical vertebrae (spondylosis, spondylarthritis, or uncovertebral arthritis).

A soft end point is more probably attributable to shortening of the long extensors of the neck or the longus colli muscle.



a b

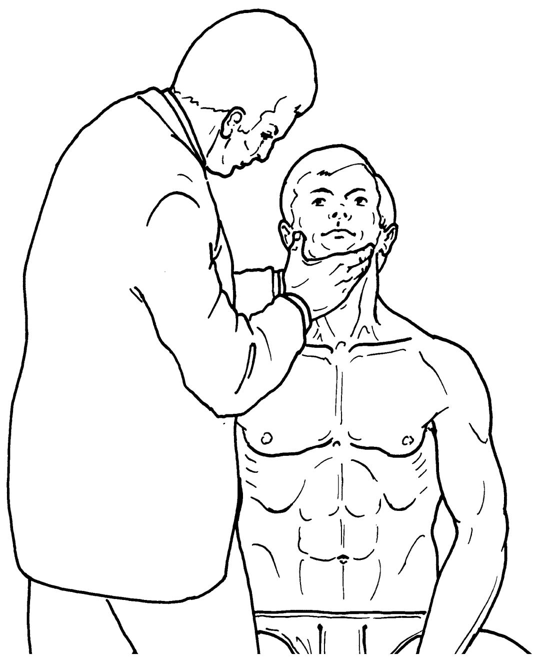
Fig. 1.11 Screening of cervical spine rotation. **(a)** At maximum right rotation. **(b)** At maximum left rotation

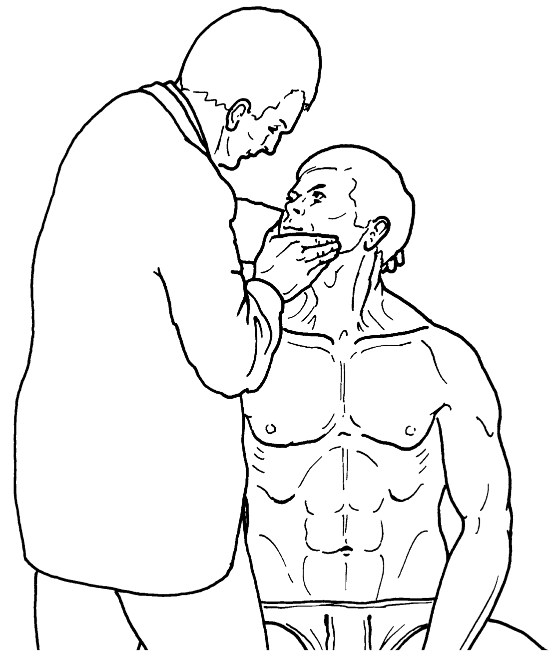
Test of Head Rotation in Maximum Extension

Functional test of the lower cervical spine.

□ **Procedure:** The patient is seated. Holding the back of the patient's head with one hand and the patient's chin with the other, the examiner passively extends the patient's neck (tilts the head backward) and rotates the head to both sides. This motion involves slight lateral bending in the cervical spine.

□ **Assessment:** In maximum extension, the region of the atlantooccipital joint is locked, and rotation takes place largely in the lower segments of the cervical spine and the cervicothoracic junction. Restricted mobility with pain is a sign of segmental dysfunction. The most likely causes include degenerative changes in the middle and lower cervical spine (spondylosis, spondylarthritis, or uncovertebral arthritis). Vertigo suggests compromised vascular supply from the vertebral artery.


a


b

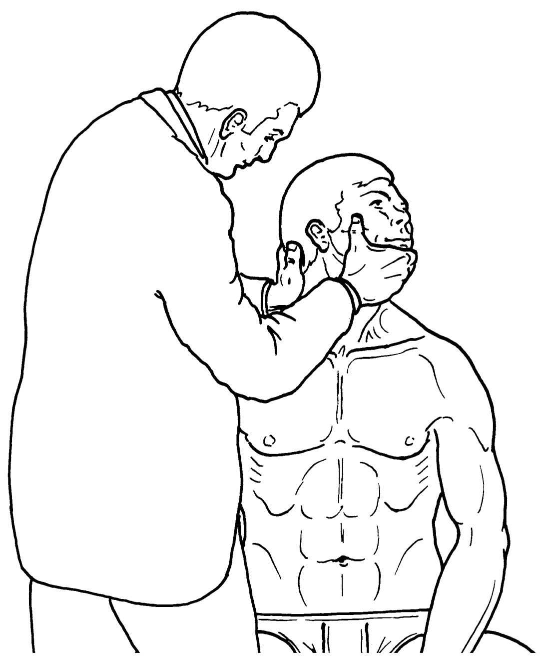

c

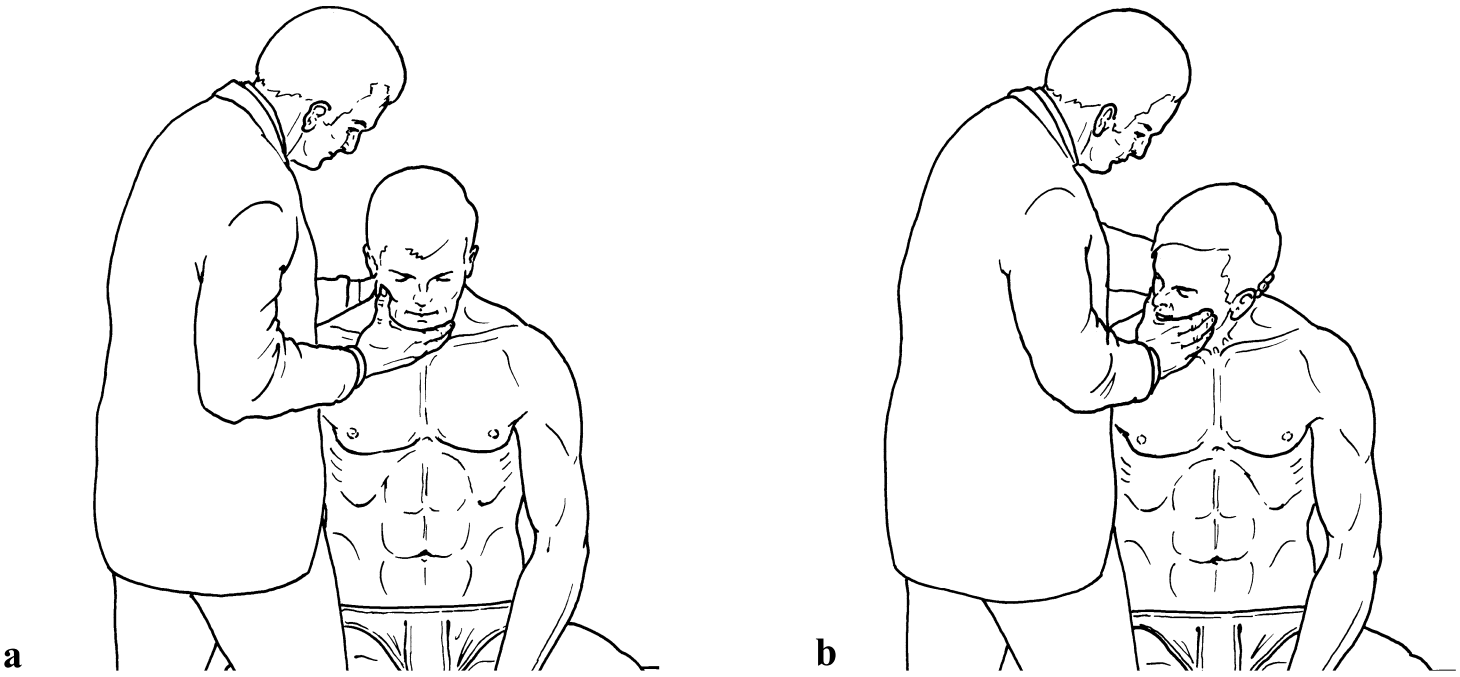
Fig. 1.12 Test of head rotation in maximum extension. **(a)** Backward tilt. **(b)** Right rotation. **(c)** Left rotation.

Test of Head Rotation in Maximum Flexion

Functional test of the upper cervical spine.

■ **Procedure:** The patient is seated. Holding the back of the patient's head with one hand and the patient's chin with the other, the examiner passively flexes the patient's neck (tilts the head forward) and rotates the head to both sides. This motion involves slight lateral bending in the cervical spine.

■ **Assessment:** In maximum flexion, the segments below C2 are locked and rotation largely takes place in the atlantooccipital and atlantoaxial joints. Restricted mobility with pain is a sign of segmental dysfunction. The most likely causes to consider include degenerative causes, instability, and inflammatory changes. Any occurrence of autonomic symptoms such as vertigo require further diagnostic studies.



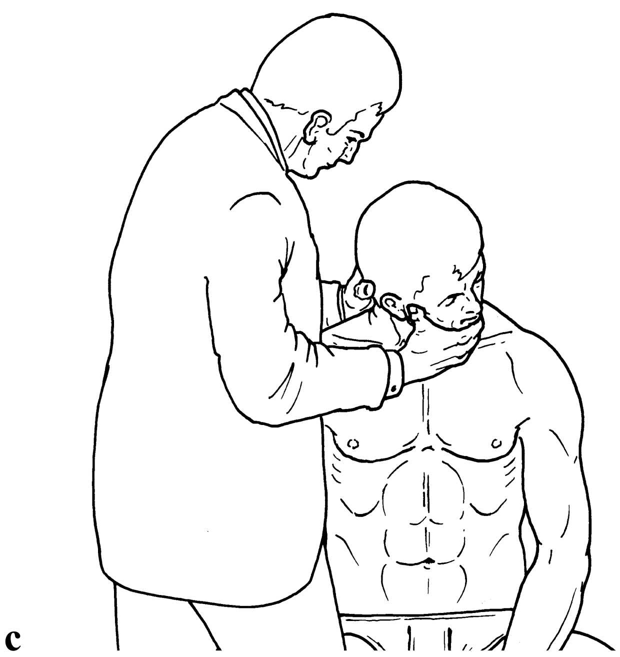


Fig. 1.13 Test of head rotation in maximum flexion. **(a)** Forward tilt. **(b)** Right rotation. **(c)** Left rotation.

Test of Segmental Function in the Cervical Spine

□ **Procedure and Assessment:** For direct diagnostic testing of segmental function in the cervical spine, the examiner stands next to the patient. Placing one hand around the patient's head so the examiner's elbow is in front of the patient's face, the examiner then places the ulnar edge of the same hand with the little finger on the arch of the upper vertebra of the segment to be examined. Segmental mobility is evaluated with the palpating finger of the contralateral hand. Posterior and lateral mobility in the segment can be assessed by applying slight traction with the upper hand. Rotation in the segment can also be evaluated during the same examination.

For diagnostic testing of segmental function in the cervicothoracic junction in flexion and extension, the examiner immobilizes the patient's head with one hand and places the fingers of the other hand on the three adjacent spinous processes. By passively flexing and extending the patient's neck, the examiner can assess the range of motion in the individual segments by observing the excursion of the spinous processes.

Fig. 1.14 Test of segmental function in the cervical spine.

Soto-Hall Test

Nonspecific test of cervical spine function.

□ **Procedure:** The patient is supine and first actively raises his or her head slightly to bring the chin as close as possible to the sternum. The examiner then passively tilts the patient's head forward, at the same time exerting light pressure on the sternum with the other hand.

□ **Assessment:** Pain in the back of the neck when pressure is applied during passive raising of the head suggests a bone or ligament disorder in the cervical spine. Pulling pain occurring when the patient actively raises the neck is primarily due to shortening of the posterior neck musculature.

Percussion Test

□ **Procedure:** With the patient's cervical spine slightly flexed, the examiner taps the spinous processes of all the exposed vertebrae.
□ **Assessment:** Localized nonradicular pain is a sign of a fracture or of muscular or ligamentous functional impairment. Radicular symptoms indicate intervertebral disk pathology with nerve root irritation.

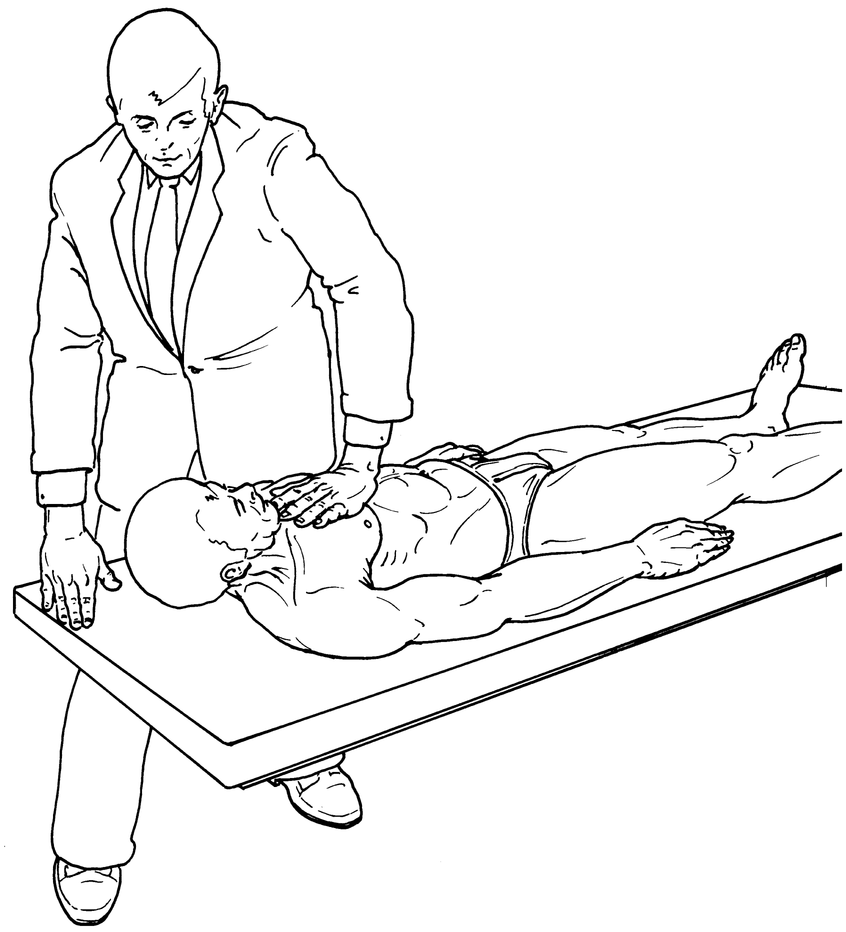


Fig. 1.15 Soto-Hall test.

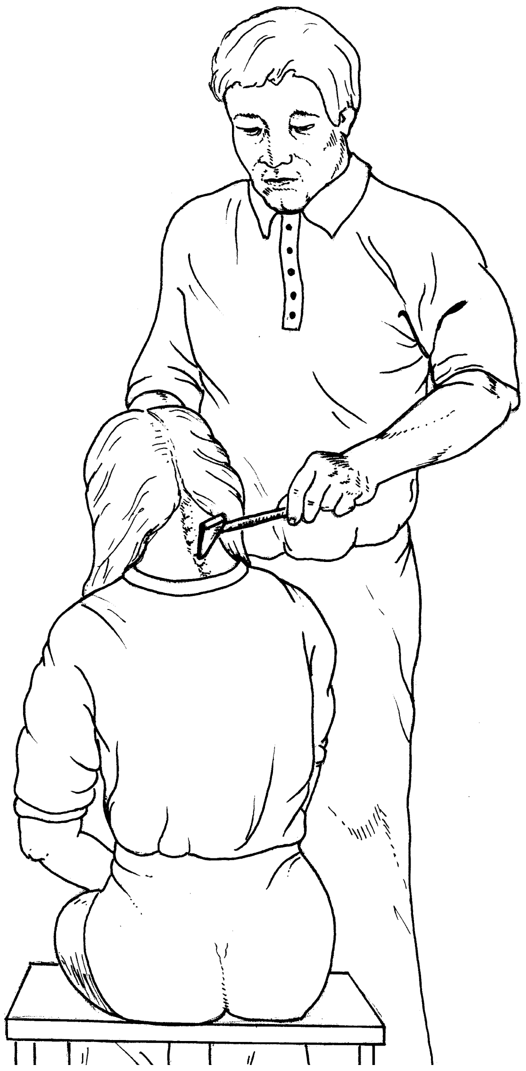


Fig. 1.16 Percussion test.

O'Donoghue Test

Differentiates between ligamentous pain and muscular pain in the back of the neck.
□ **Procedure:** The seated patient's head is passively tilted first to one side and then the other. Then the patient is asked to tilt his or her head to one side against the resistance of the examiner's hand resting on the zygomatic bone and temple.
□ **Assessment:** Occurrence of pain during this active head motion with isometric tensing of the ipsilateral and contralateral paravertebral musculature suggests muscular dysfunction, whereas pain during passive lateral bending of the cervical spine suggests a functional impairment involving ligaments or articular, possibly degenerative processes.

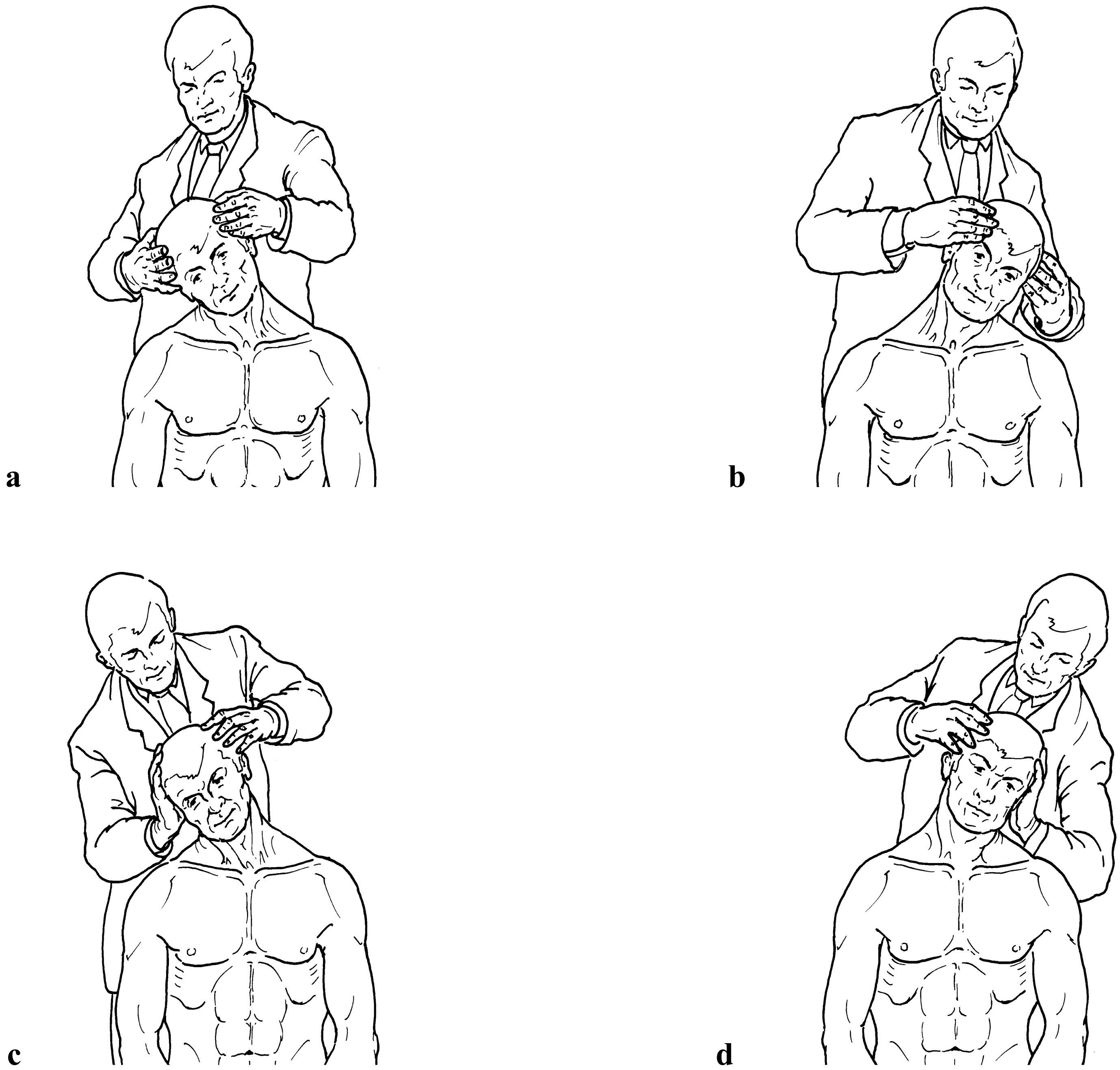


Fig. 1.17 O'Donoghue test. **(a, b)** Passive motion. **(c, d)** Active motion against resistance.

Valsalva Test

- **Procedure:** The patient is seated with the thumb in the mouth and attempts to push the thumb out by blowing out hard.
- **Assessment:** Bearing down in this manner increases the intraspinal pressure, revealing the presence of space-occupying masses such as extruded intervertebral disks, tumors, narrowing due to osteophytes, and soft tissue swelling. This leads to radicular symptoms entirely confined to the respective dermatome or dermatomes. The test should be performed with great caution, because the patient may lose consciousness during or after the test due to potential impaired blood supply to the brain.

Spurling Test

Assesses facet joint pain and nerve root irritation.

□ **Procedure:** While seated, the patient flexes the head and tilts it laterally, first to the unaffected side and then to the affected side. The examiner stands behind the patient with one hand on the patient's head. With the other hand, the examiner lightly taps (compresses) the hand resting on the patient's head. If the patient tolerates this initial step of the test, it is then repeated with the cervical spine extended as well.

Fig. 1.18 Valsalva test.

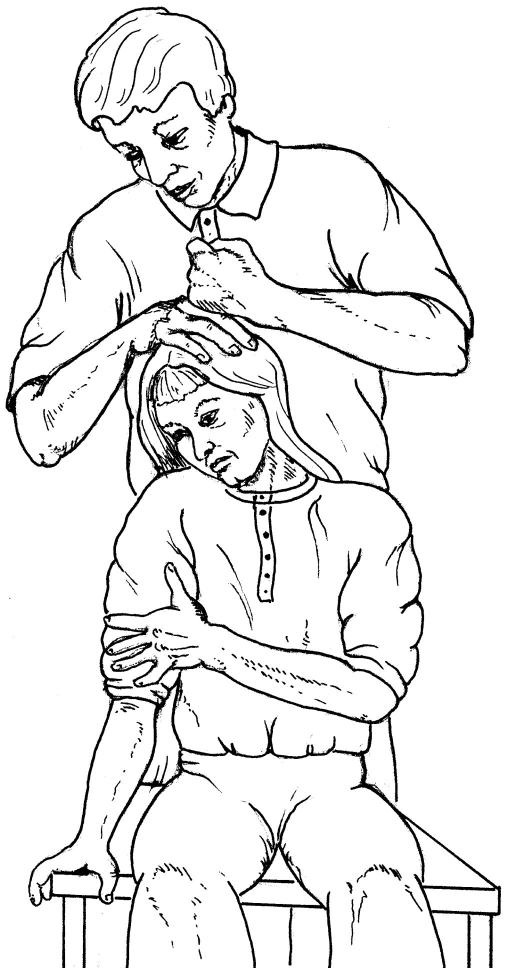


Fig. 1.19 Spurling test.

□ **Assessment:** If pain radiates from the cervical spine down the patient's arm, the test is considered to be positive. Simultaneous extension of the cervical spine narrows the intervertebral foramina by 20 to 30% In conditions such as cervical stenosis, spondylosis, osteophytes, trophic facet joints, or herniated disks, the foramina may already be smaller than normal.

The test is not considered to be positive if there is neck pain only, without radiation into the shoulder or arm. Pain radiating to the arm specific to a certain dermatome suggests nerve root irritation. Pain that is already present will be increased by this movement.

Myalgia and whiplash syndrome can cause pain on the opposite side. This is called a reverse Spurling sign and suggests pain on the side of muscles that have been stretched from muscle strain or functional disturbance with muscle foreshortening.

□ **Note:** Pain on the concave side indicates nerve root irritation or facet joint pathology (Spurling sign).

Pain on the convex side indicates muscle strain (reverse Spurling sign).

The Spurling test is an aggressive cervical compression test, and the patient should be prepared for each step of the examination. The test should not be performed when cervical fracture, dislocation, or instability are suspected.


a


b

Fig. 1.20 Cervical spine distraction test. **(a)** Middle position. **(b)** Rotation.

Cervical Spine Distraction Test

Helps to determine whether pain in the back of the neck, shoulder, and arm is radicular in origin or is due to ligamentous or muscular causes.

□ **Procedure:**

The patient is seated. The examiner grasps the patient's head about the jaw and the back of the head and applies superior axial traction.

□ **Assessment:**

Distraction of the cervical spine reduces the load on the intervertebral disks and exiting nerve roots within the affected levels or segments while producing a gliding motion in the facet joints. Reduction of radicular symptoms, even in passive rotation, when the cervical spine is distracted is a sign of discogenic nerve root irritation. Increased pain during distraction and rotation suggests a functional impairment in the cervical spine due to muscular or ligamentous pathology or articular, possibly degenerative processes.

Elvey Test (Upper Limb Tension Tests)

The nerve roots supplying the upper extremity are provoked by stretching them utilizing position changes of the shoulder, elbow, and wrist.

- **Procedure:**

The patient lies supine. During all the tests the examiner fixes the shoulder in a proximal anterior position with one hand while using the other hand to guide the patient's arm to the set positions. The Elvey tests for the cervical spine are equivalent to the Lasègue test for the lumbar spine. The tests as described by Elvey are divided into four examination segments.

- **Test 1:** The examiner abducts the shoulder to 110°, extends the elbow, supinates the forearm, extends the wrist, and extends the fingers and thumb. Nerve bias: median nerve, anterior interosseous nerve, C5/C6/C7.
- **Test 2:** The examiner places the shoulder at 10° abduction, extends the elbow, supinates the forearm, and extends the wrist, fingers, and thumb. From this position, the shoulder is externally rotated. Nerve bias: median, musculocutaneous, and axillary nerves.

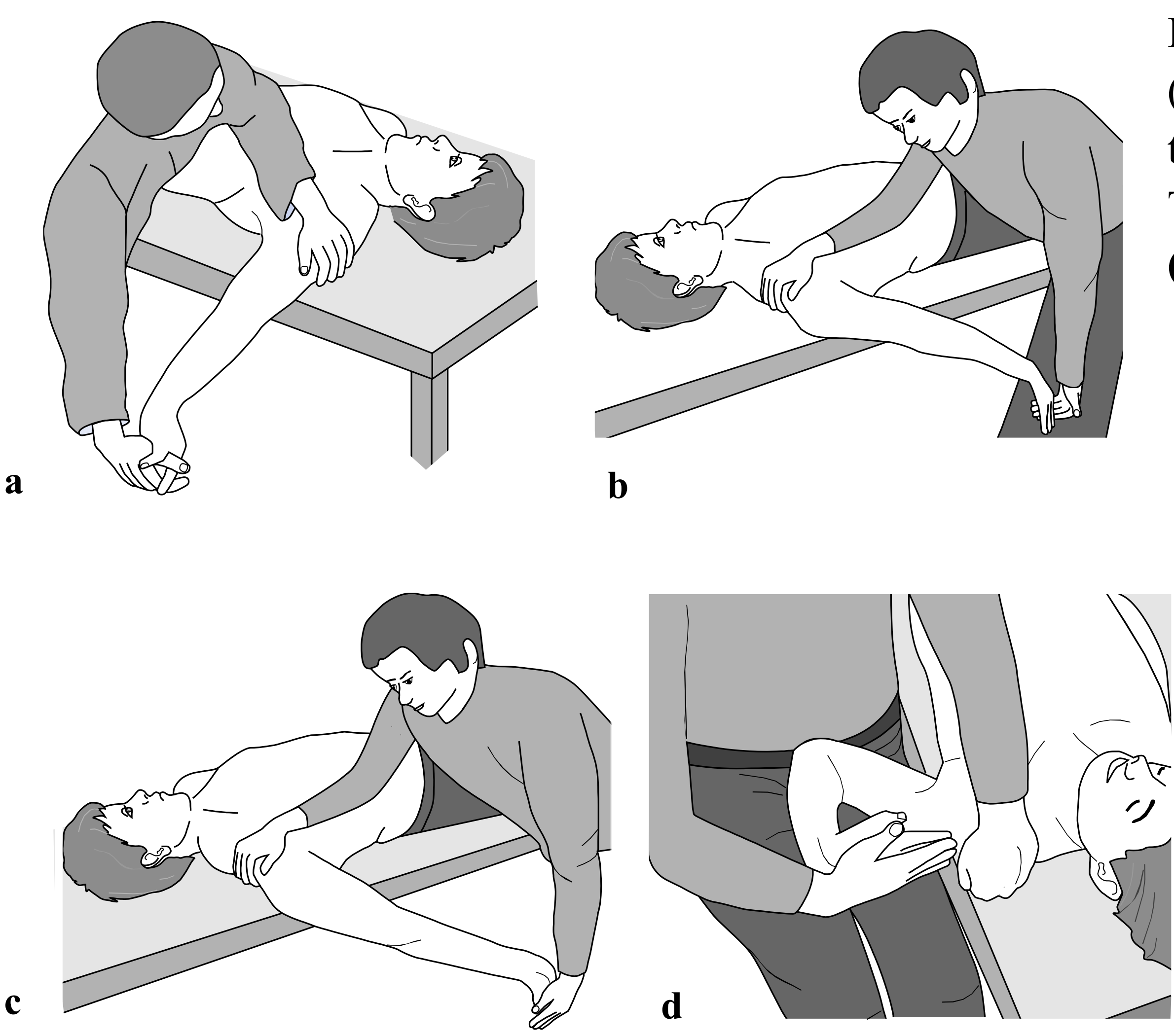


Fig. 1.21 Elvey test (upper limb tension tests). **(a)** Test 1. **(b)** Test 2. **(c)** Test 3. **(d)** Test 4.

□ **Test 3:** Starting position: shoulder abduction 10°, elbow extension, forearm pronation, wrist flexion and ulnar deviation, finger and thumb flexion. The examiner internally rotates the arm. Nerve bias: radial nerve.

□ **Test 4:** The examiner successively abducts the shoulder from 10 to 90° and guides the hand toward the ear with the elbow maximally flexed and the forearm supinated. The wrist is extended and radially abducted, the fingers and thumb extended. The shoulder is externally rotated. Nerve bias: ulnar nerve, C8 and T1 nerve roots.

□ **Assessment:** These tests cause narrowing of the intervertebral foramina. Radicular pains already present are worsened by these movement patterns. The occurrence of localized pains in the cervical spine without radicular symptoms suggests facet irritation. Pain on the convex side of the cervical spine indicates muscle dysfunction (of the sternocleidomastoid muscle, for instance).

□ **Note:** This test not only gives an indication as to the cause of pain but also demonstrates the success of treatment (with manual therapy measures, for example).

Brachial Plexus Tension Test

This is a modification of the Elvey test to assess motor and sensory disturbances using compression of the brachial plexus.

□ **Procedure:** The patient is seated and abducts and externally rotates both arms until pain or paresthesias occur. Then the patient lowers both arms just until the symptoms disappear again. The examiner, standing behind the patient, fixes both upper arms in this position. The patient is now asked to bend the elbows slowly from this position and to fold the hands together behind the head.

□ **Assessment:** The test is positive if the symptoms are reproduced by bending the elbows. This primarily tests the ulnar nerve and the C8 and T1 nerve roots. With increasing flexion (bending the head forward), the symptoms increase.

□ **Note:** As a variation of the test, the patient abducts the arm at the shoulder to 90° and simultaneously maximally flexes the elbow. Then the patient extends the elbow. If this action produces radicular symptoms, the test is considered positive, with the suspicion of a brachial plexus lesion (Bikele sign).

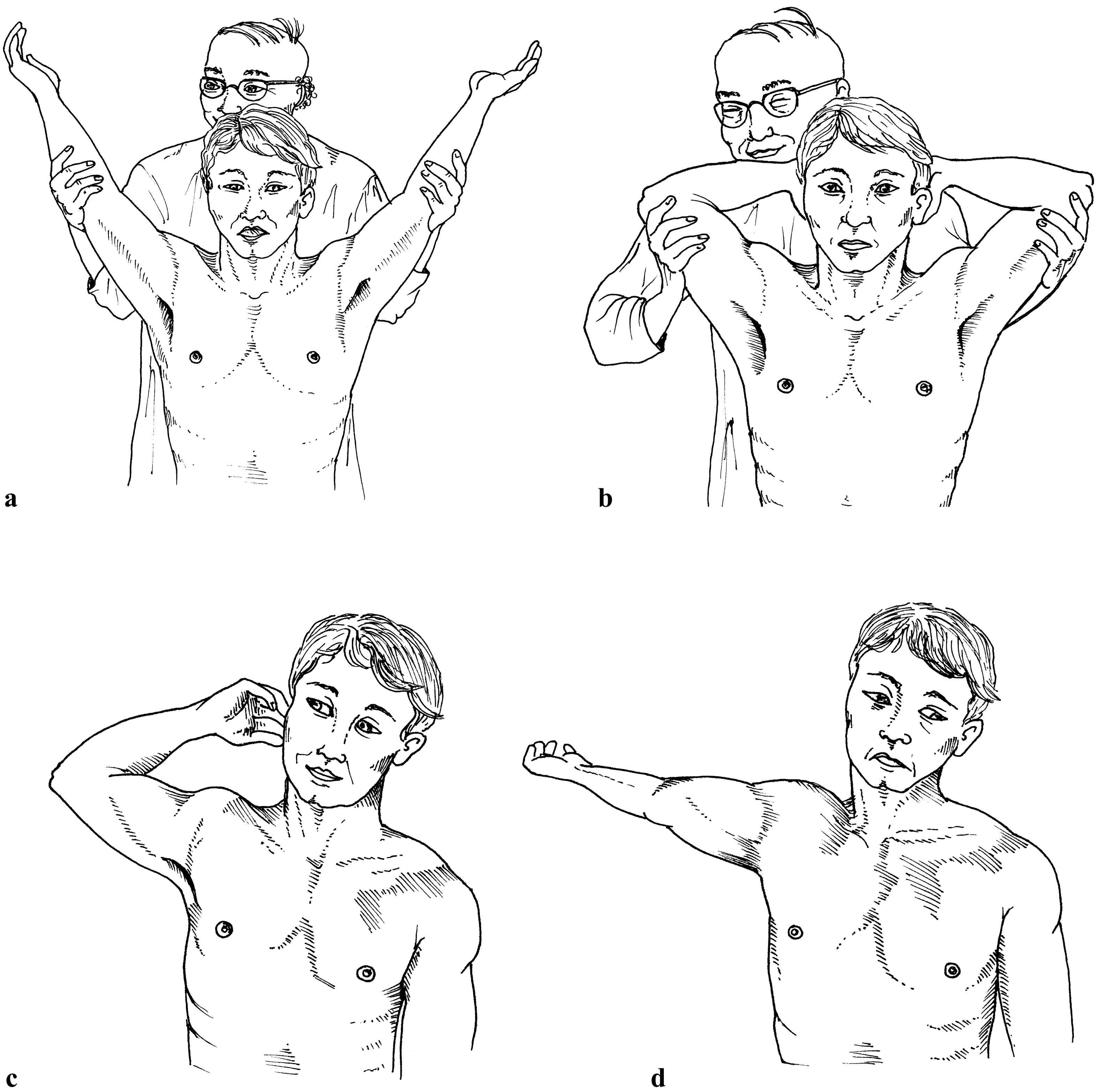
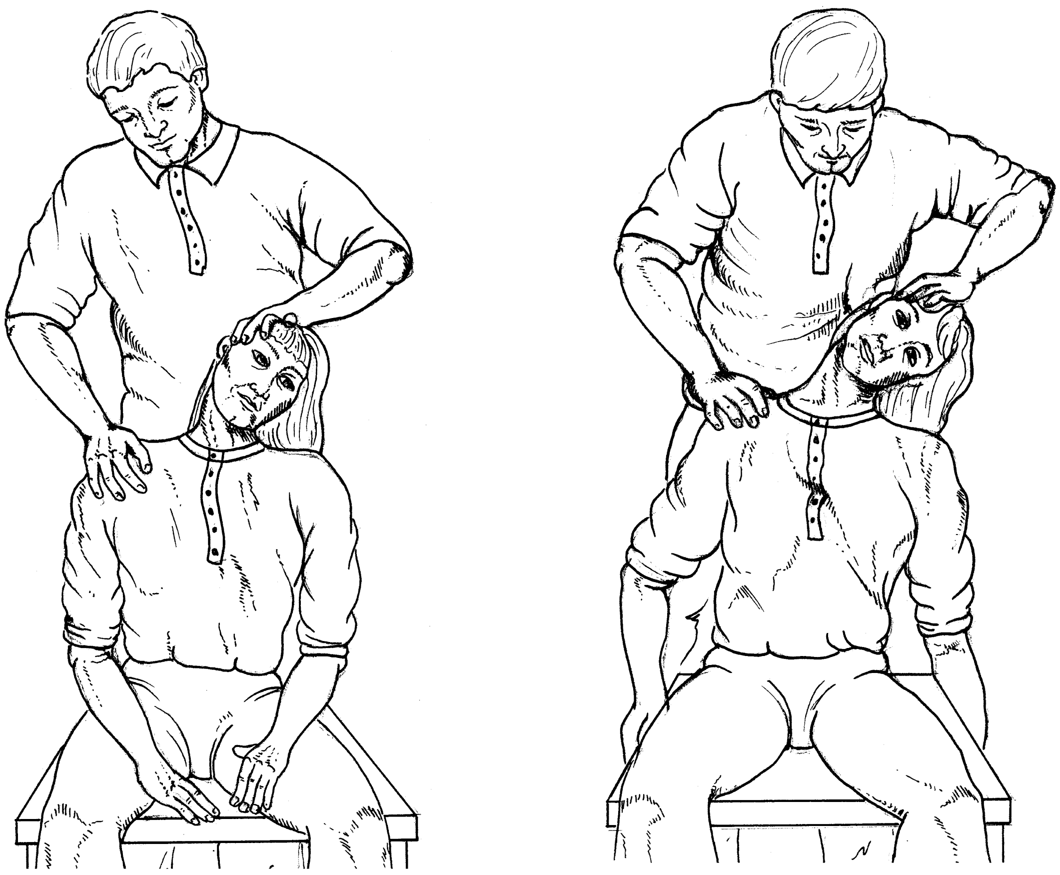


Fig. 1.22 Brachial plexus tension test. **(a)** Abduction and external rotation. **(b)** Bending the elbows and placing the hands behind the head. **(c)** Bikele sign: abduction of the arm to 90° with the elbow fully flexed. **(d)** Bikele sign: extension of the elbow.

Shoulder Press Test

□ **Procedure:** The patient is seated. The examiner presses downward on one shoulder while bending the cervical spine laterally toward the contralateral side. This test is always performed on both sides.

□ **Assessment:** Provocation of radicular symptoms is a sign of adhesion of the dural sac and/or a nerve root. Increase in pain during the test suggests nerve root compression, foraminal narrowing by osteophytes, dural sheath changes, or contracture of the facet joint capsule on the side being tested. Circumscribed pain on the side of the stretched musculature indicates increased muscle tone in the sternocleidomastoid or trapezius. Decreased muscular pain in the side that is not stretched suggests a pulled muscle or a functional impairment involving shortening of the musculature.



a

Fig. 1.23 Shoulder press test. **(a)** Lateral bending. **(b)** Forced lateral bending.

Shoulder Abduction (Bakody) Test

This test is performed for suspicion of C4 or C5 nerve root irritation.

□ **Procedure:** The patient sits or lies supine. Either the examiner or the patient abducts the shoulder, flexes the elbow, and places the palm of the hand on the top of the head.

□ **Assessment:** The test is counted as positive if the symptoms of nerve root irritation improve or resolve. Which roots are affected can be determined by clinical findings (dermatome distribution). The C4–C6 nerve roots are primarily examined in this test.

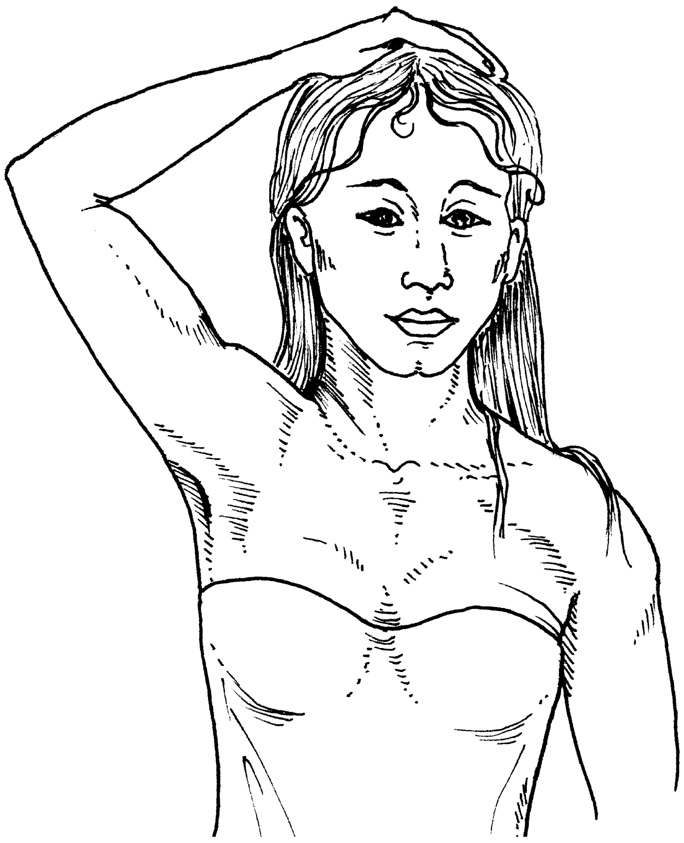


Fig. 1.24 Shoulder abduction test (Bakody test).

This test is also called the Bakody sign. Abduction of the arm decreases the tension of the nerve roots. If the nerve root irritation increases during the course of the above test, one must suspect a thoracic outlet syndrome (due to changes in the scalene muscles or to a cervical rib).

Patients with moderate to severe radicular symptoms usually do not have to be directed into the Bakody sign position, because it also is an antalgic posture that relieves pain.

Jackson Compression Test

- **Procedure:** The patient is seated. The examiner stands behind the patient with his or her hand on the top of the patient's head and passively tilts the head to either side. In maximum lateral bending, the examiner presses down on the head to exert axial pressure on the spine. The head is slightly rotated to the involved side.
- **Assessment:** The axial loading results in increased compression of the intervertebral disks, exiting nerve roots, and facet joints. Pressure on the intervertebral foramina acts on the facet joints to elicit distal pain that does not exactly follow identifiable segmental dermatomes. Presence of nerve root irritation will cause radicular pain symptoms. Local circumscribed pain will be attributable to stretching of the contralateral musculature of the neck.

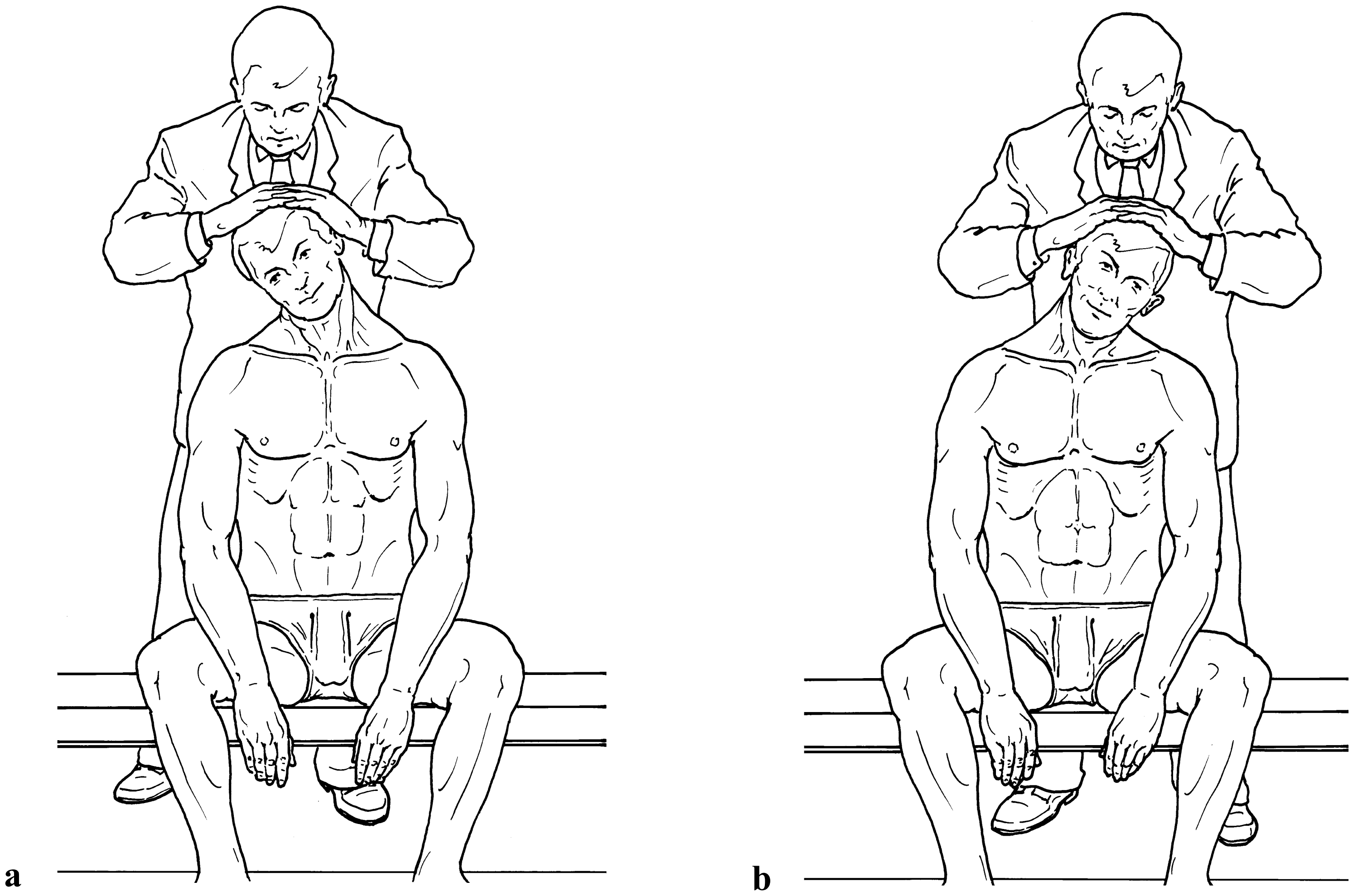


Fig. 1.25 Jackson compression test. **(a)** Right lateral bending. **(b)** Left lateral bending.

Intervertebral Foramina Compression Test

□ **Procedure:** Axial compression is applied to the cervical spine in the neutral (0°) position.

□ **Assessment:** Compression of the intervertebral disks and exiting nerve roots, the facet joints, and/or the intervertebral foramina increases a radicular, strictly segmental pattern of symptoms. The presence of diffuse symptoms that are not clearly specific to any one segment may be regarded as a sign of ligamentous or articular functional impairment (facet joint pathology).

Flexion Compression Test

□ **Procedure:** The patient is seated. The examiner stands behind the patient and passively moves the cervical spine into flexion (tilts the patient's head forward). Then axial compression is applied to the top of the head.

□ **Assessment:** This is a good test of the integrity of the intervertebral disk. In the presence of a posterolateral disk extrusion, this maneuver will press the extruded portion of the disk in a posterior direction, resulting in increasing compression of the nerve root. An increase in radicular symptoms can therefore indicate the presence of a posterolateral disk extrusion.

Fig. 1.26 Intervertebral foramina compression test.

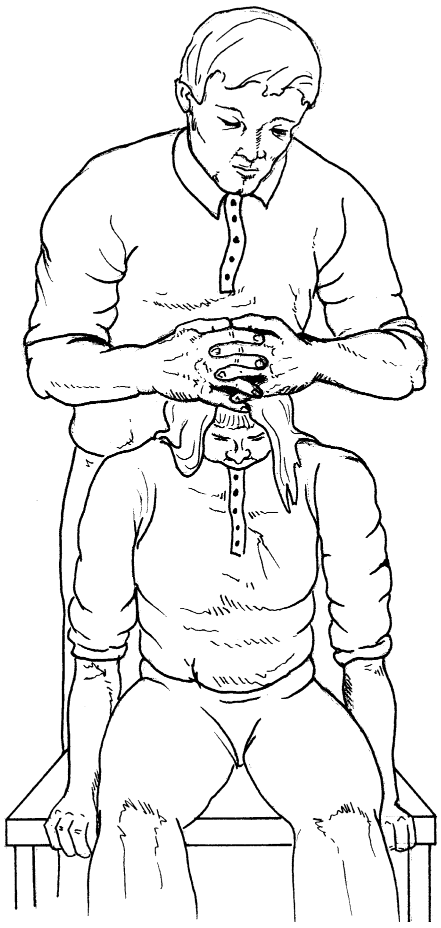


Fig. 1.27 Flexion compression test.

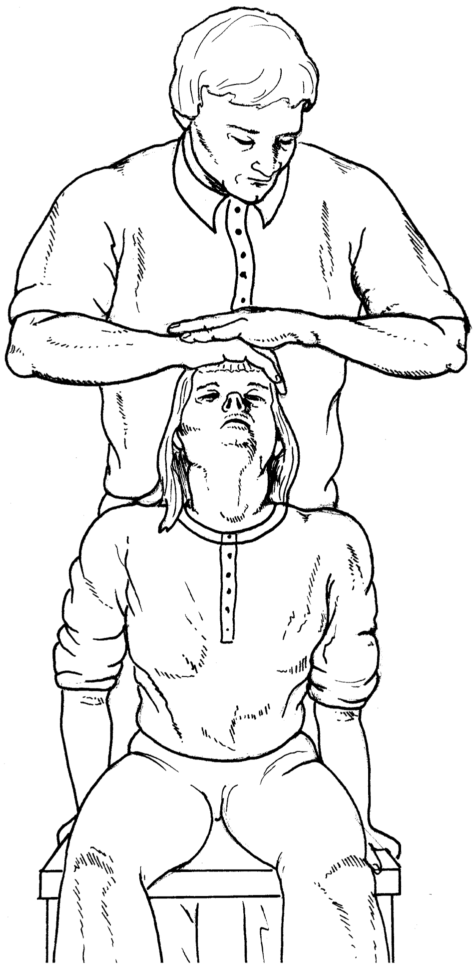


Fig. 1.28 Extension compression test.

The forward tilting of the head usually reduces the load on the facet joints and can reduce pain due to degenerative changes. Increasing pain may indicate an injury to posterior ligamentous structures.

Extension Compression Test

□ **Procedure:** The patient is seated and the examiner stands behind the patient. The cervical spine is extended 30°. The examiner then applies axial compression to the top of the head.
□ **Assessment:** This test assesses the integrity of the intervertebral disk. Where a posterolateral extrusion with an intact anulus fibrosus is present, shifting the pressure on the disks anteriorly will reduce symptoms. Increased pain without radicular symptoms usually indicates an irritation in the facet joints as a result of decreased mobility due to degenerative changes.

Lhermitte Sign

This test differentiates between spinal cord lesions and peripheral nerve root lesions.

□ **Procedure:** The patient sits with outstretched legs on the examination table. The examiner grasps the patient's foot with one hand and places the other on the back of the patient's head. The examiner then simultaneously flexes the outstretched leg at the hip and increasingly flexes the cervical spine.

□ **Assessment:** The test is positive if an acute pain occurs that radiates into the upper or lower extremity. This suggests dural or meningeal irritation of the spinal cord (root irritation) or possibly cervical myelopathy.

If the patient were to actively bend the head toward the breast, then this would be the Soto-Hall test. Maximally flexing the cervical spine places strong tension on the spinal cord.

□ **Note:** A positive Lhermitte sign can indicate stenosis of the cervical spinal canal. The patient describes a sudden, generalized electric shock in the arms and trunk, especially when inclining the head.

Spinal stenosis is usually of bony origin from pronounced spondylosis and spondylarthritis as a result of a degenerative intervertebral disk injury. The symptoms occur insidiously. Early symptoms are abnormal sensations in the hands, gait disorders, and clumsiness of the hands (disturbances of fine motor function, writing and grip).

Spastic, hyperactive deep-tendon reflexes and pyramidal signs are further findings.

In terms of differential diagnosis, the following disorders must be kept in mind:

- Brachial plexus lesion.
- Irritated cervical root syndrome.
- Multiple sclerosis.
- Spinal tumor.
- Amyotrophic lateral sclerosis.

Fig. 1.29 Lhermitte sign.

Thoracic Spine Tests

Pain in the thoracic vertebral spinal segments may arise from the thoracic spine, but also from the chest cavity (viscous perforation, for example). Degenerative, inflammatory, tumorous, and traumatic lesions lead to symptoms in the thoracic spine, often with the participation of neural structures. Nerve root syndromes due to herniated disks are rare. Thoracic spinal stenosis causes symptoms from bilateral sensory dysfunctions right up to major motor malfunction, such as paresis and paraspasticity.

Adams Forward Bend Test

Assesses structural or functional scoliosis.

- **Procedure:** The patient may be seated or standing. The examiner stands behind the patient and asks the patient to bend forward.
- **Assessment:** This test is performed in patients with detectable scoliosis of uncertain etiology or as a screening examination in patients with a family history of scoliotic posture. If the scoliotic posture improves during forward bending, then the condition is a functional scoliosis; where the scoliotic deformity remains with the same projection of the ribs and the lumbar distortion observed in upright posture, the condition is true scoliosis with structural changes.

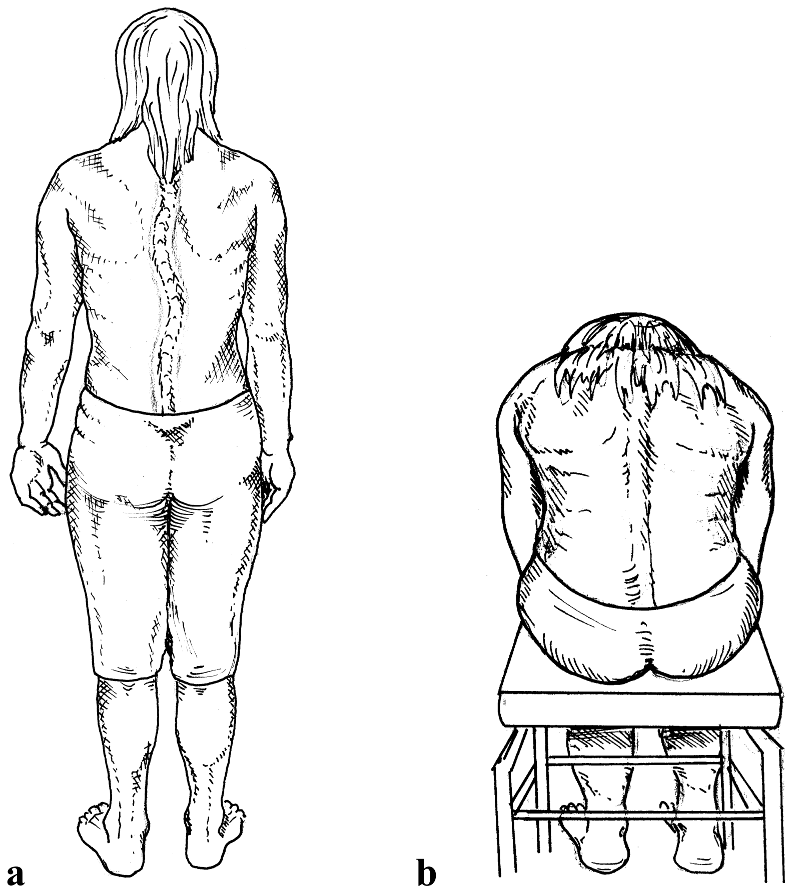


Fig. 1.30 Adams forward bend test. **(a)** Upright posture. **(b)** Forward bending.

Kyphosis Test on Hands and Knees

□ **Procedure:** The patient is asked to kneel and stretch out his or her arms as far forward as possible on the floor.

□ **Assessment:** This posture will correct a flexible kyphotic deformity of the thoracic spine. A kyphotic posture that remains unchanged is a fixed deformity.

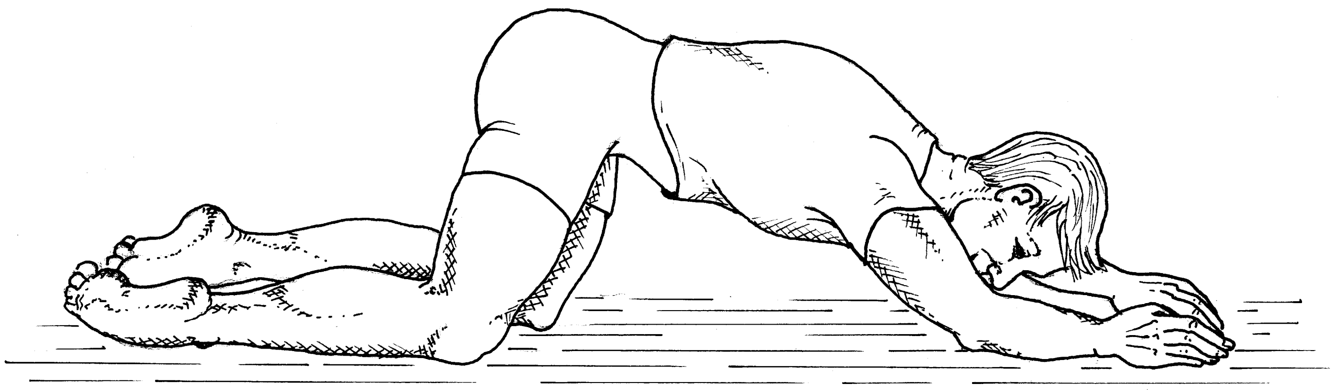
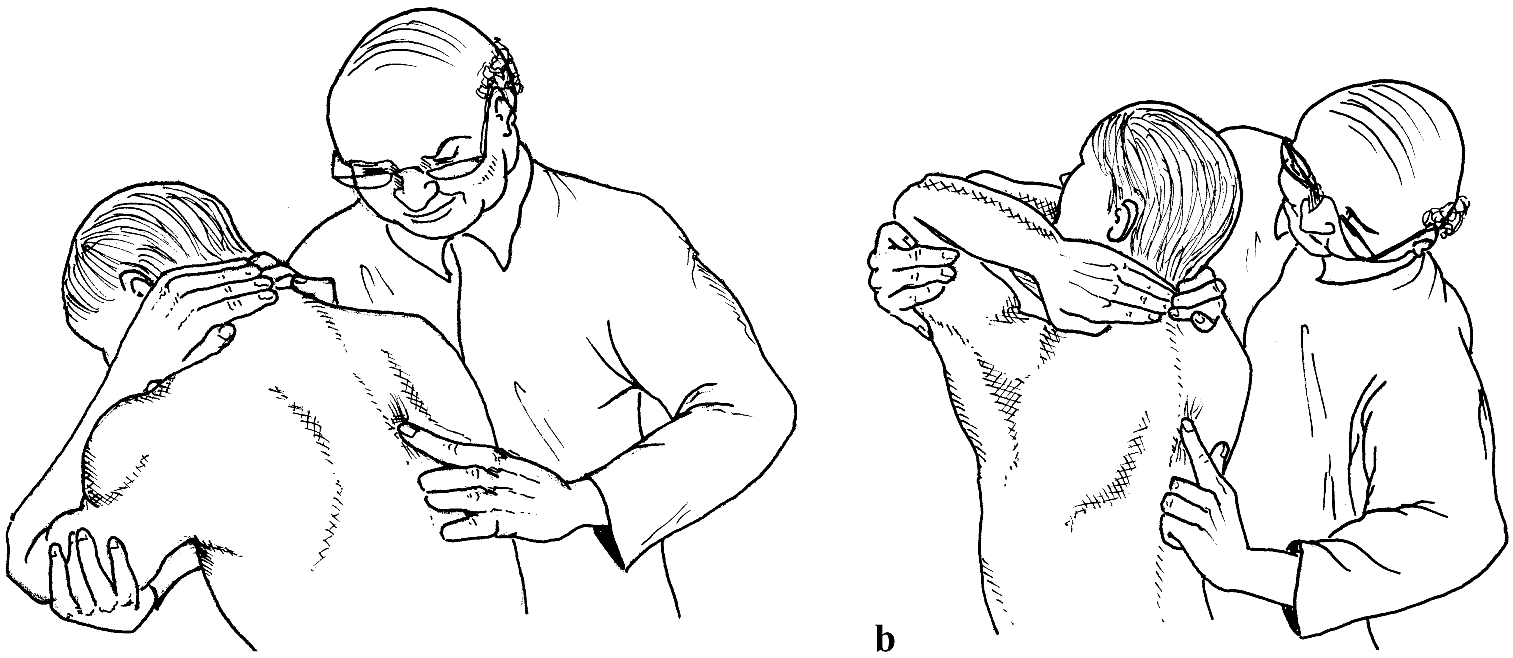


Fig. 1.31 Kyphosis test on hands and knees.

Test of Segmental Function in the Thoracic Spine in Extension and Flexion

□ **Procedure:** The seated patient clasps both hands behind his or her head with the elbows together. The examiner immobilizes the patient's arms in front of the patient with one hand, leaving the examining hand free.

□ **Assessment:** The examiner can detect segmental functional impairments by palpating the individual segments while passively moving the patient's spine into flexion, extension, lateral bending, and rotation. A similar technique can also be used to evaluate segmental function in the lumbar spine.



a **b**

Fig. 1.32 Test of segmental function in the thoracic spine. **(a)** Flexion. **(b)** Extension.

Lumbar Spine Tests

Disorders of the lumbar spine with accompanying neurologic disturbances are usually based on degenerative changes of the intervertebral disk tissue with reactive changes of the facet joints and the superior and inferior end plates of the vertebral bodies. Less often, direct or indirect traumatic damage to the spinal cord is responsible for back pain.

In particular, protrusion or herniation of intervertebral disks causes spinal nerve irritation. Primarily affected are the two lowest intervertebral disk levels: L4/L5 level involving the L5 and less often the L4 nerve roots and the L5/S1 level involving the S1 nerve root.

With increasing age of the spine, progressive degenerative processes cause stenotic areas in the spinal canal with circumscribed, bony-ligamentous narrowing. Clinically, back pain and loading-dependent symptoms in the legs (claudication) occur.

As a rule, the patient has long-term complaints of insidiously progressive back pain that is load dependent, radiates into the legs, and leads to nonspecific symptoms such as fatigue and feelings of heaviness. With further progression, neurologic deficits may develop, including hypesthesia and paresis. Symptoms may become continuous, even at rest, and could progress all the way to a cauda equina syndrome. The distance the patient can walk becomes limited. By relieving the hyperlordosis in a stooped body posture, activities such as cycling or the support from a shopping cart improve the symptoms. In contrast to arterial occlusive disease, the simple act of standing still does not improve symptoms. After a certain distance, the patient has to sit down. Long bike trips are

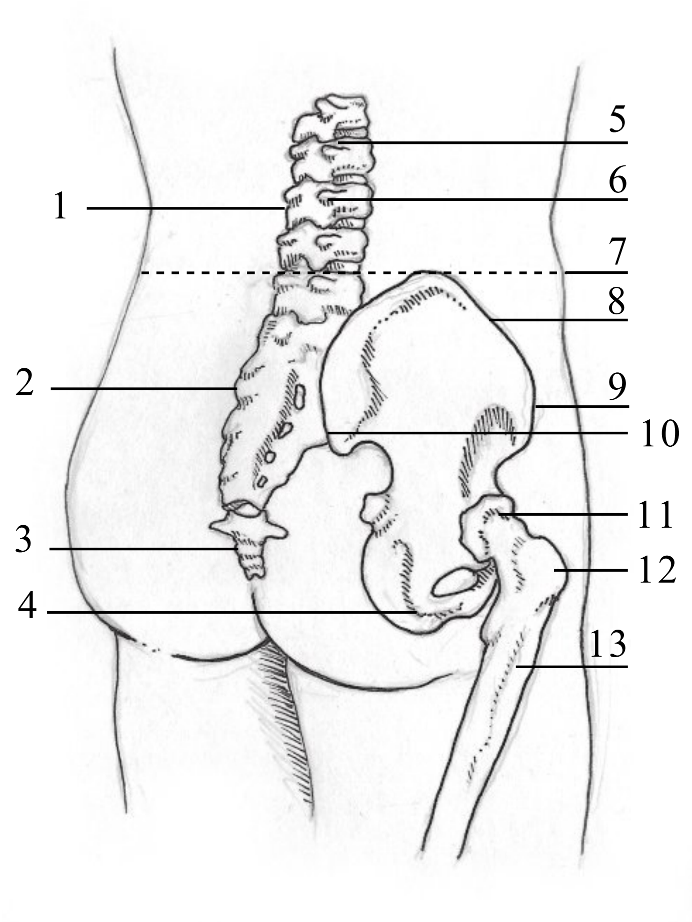


Fig. 1.33 Bony landmarks of the lumbar spine and pelvic region:
1 Spinous process.
2 Sacral crest.
3 Coccyx (tailbone).
4 Ischial tuberosity ("sitting bone").
5 Facet joint.
6 Transverse process.
7 Supracristal line—L4/5 disk space.
8 Iliac crest.
9 Anterior superior iliac spine.
10 Posterior superior iliac spine.
11 Hip joint.
12 Greater trochanter.
13 Femur.

possible. Of course, one must evaluate the status of the peripheral pulses to differentiate between vascular and neurogenic claudication. If claudication is vascular in origin, unlimited cycling is not possible. After walking a certain set distance, the patient does not have to sit down—simply standing still is sufficient, and he or she can then continue walking ("window-shopping disease").

Prone Knee Flexion Test for Lumbar Spine

Differentiates between lumbar and sacroiliac pain.

□ **Procedure:** Prone position. The examiner bends the patient's knee and tries to bring the heel as far as possible toward the buttocks. First the patient should passively give way, but then try to extend the leg against the resistance of the examiner's hand.

□ **Assessment:** During the test, there is a feeling of tension first in the sacroiliac joint, then in the lumbosacral junction, and lastly in the lumbar spine. This test should be performed when there is suspicion of changes in the pelvic ligaments and intervertebral disks. Pain in the sacroiliac joint or in the lumbosacral or lumbar areas without radicular, radiating pain suggests degenerative changes and/or ligament insufficiency. Increase in radicular pain indicates intervertebral disk injury.

Unilateral nerve root pain in the lumbar spine, in the buttock region, and over the posterior thigh suggest L2/L3 nerve root injury.

During this test the femoral nerve will also be stretched. The occurrence of pain and/or paresthesias in the anterior thigh indicates tight quadriceps, quadriceps lesion, or femoral nerve irritation.

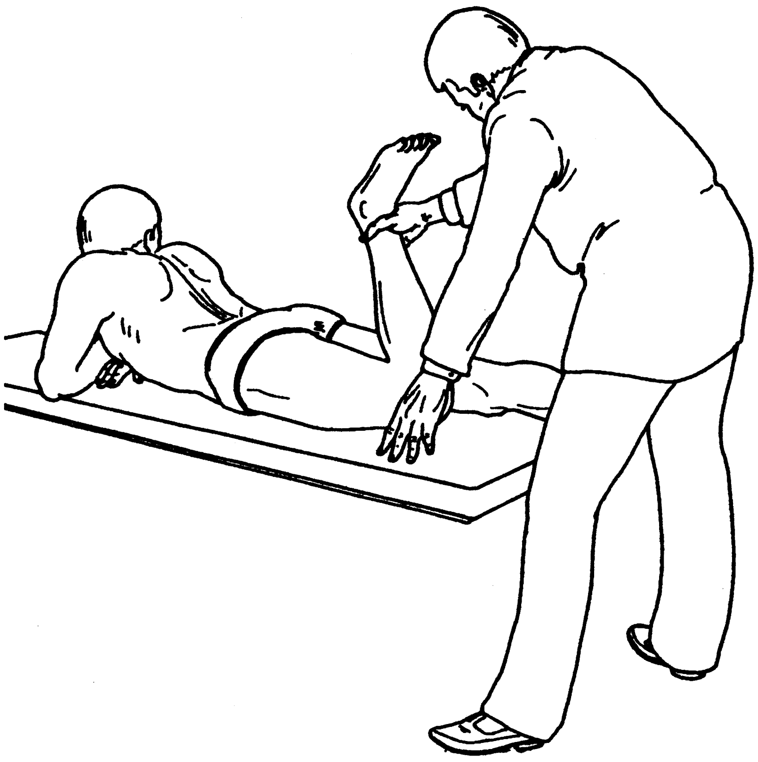


Fig. 1.34 Prone knee flexion test.

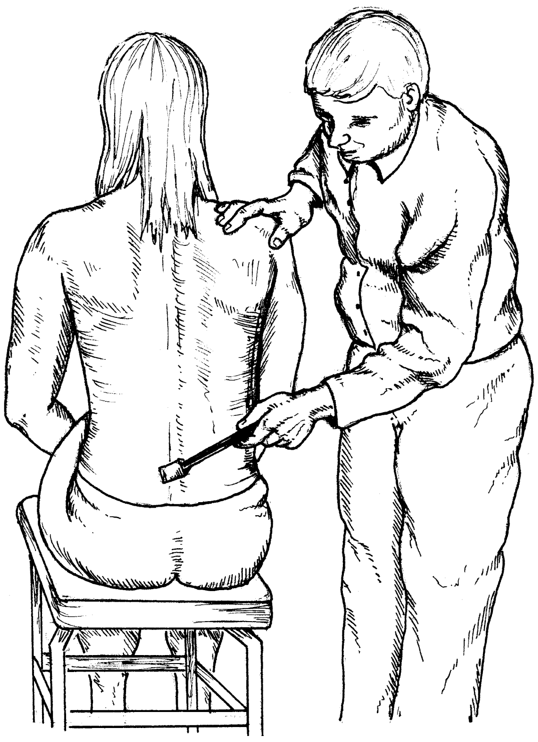


Fig. 1.35 Spinous process tap test.

Spinous Process Tap Test

Indicates lumbar spine syndrome.

□ **Procedure:** The patient is seated with the spine slightly flexed. With a reflex mallet, the examiner taps on the spinous processes of the lumbar spine and on the paraspinal musculature.

□ **Assessment:** Localized pain can indicate irritation of the involved spinal segments as a result of degenerative inflammatory changes. Radicular pain can be a sign of disk pathology.

Psoas Sign

For diagnostic assessment of lumbar pain.

□ **Procedure:** The patient is supine and raises one leg with the knee extended. The examiner presses suddenly on the anterior aspect of the thigh.

□ **Assessment:** This sudden pressure on the distal thigh causes reflexive contraction of the iliopsoas, with traction on the transverse processes of the lumbar spine. Patients will report pain in the presence of disorders in the lumbar spine (spondylarthritis, spondylitis, or disk herniation) or in the sacroiliac joint.

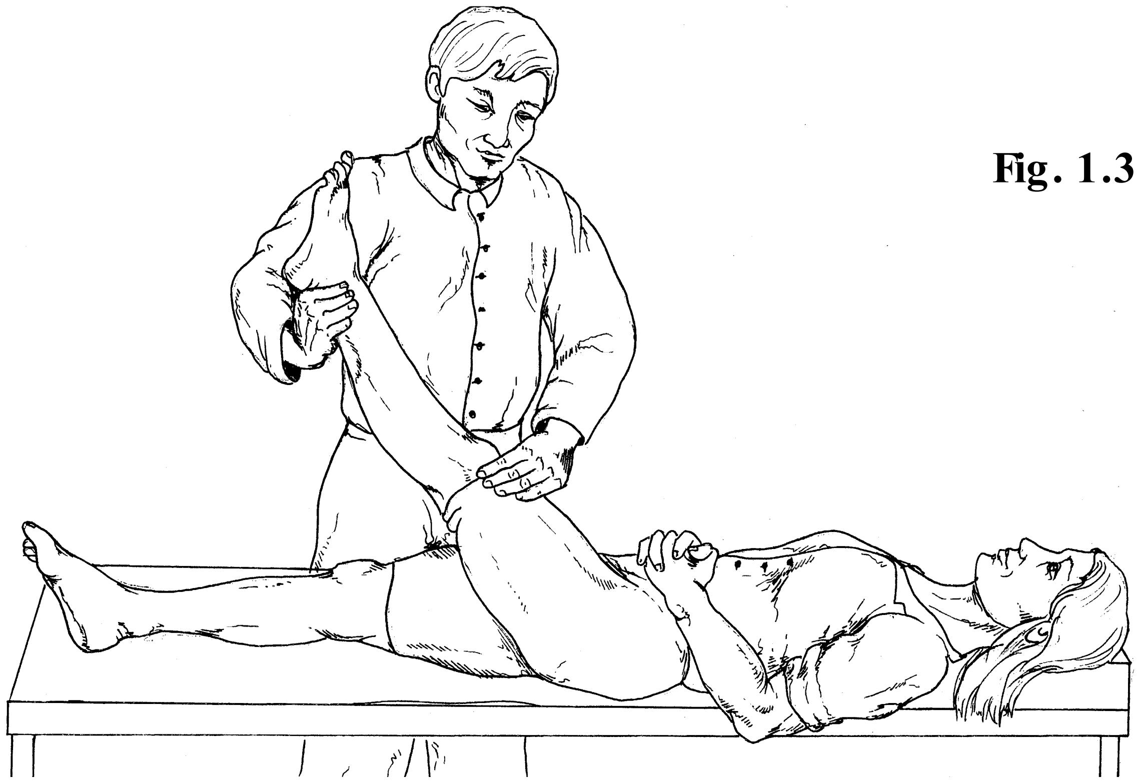


Fig. 1.36 Psoas sign.

Lasègue Drop (Rebound) Test

Differentiates lumbar pain.

□ **Procedure:** The patient is supine. The examiner performs the test on the affected side as in the Lasègue test, raising the leg until the patient begins to feel discomfort. Then the examiner lets go of the leg from this position.

□ **Assessment:** Suddenly and unexpectedly letting go of the leg precipitates reflexive contraction of the muscles of the back and buttocks. It is primarily the iliopsoas that contracts, placing traction on the transverse processes of the lumbar spine. Patients will report pain in the presence of disorders of the lumbar spine (spondylarthritis, spondylitis, or disk herniation) or disorders of the sacroiliac joints (see Psoas Sign, p. 37).

For a differential diagnosis it must be borne in mind that this test can also intensify visceral pain such as that caused by appendicitis.

Lumbar Spine Springing Test

For localization of functional impairments in the lumbar spine.
□ **Procedure:** The patient is prone. The examiner palpates the articular processes or laminae of the vertebrae in question with his or her index and middle fingers. With the ulnar edge of the other hand, which is held perpendicularly over the palpating fingers, the examiner repeatedly presses lightly in a posteroanterior direction. The palpating fingers conduct this light springing pressure to the articular processes or laminae of the vertebrae in question.
□ **Assessment:** Where joint function is intact, the articular processes or laminae will be resilient.

Lack of resiliency or excessive resiliency is a sign of abnormal segmental mobility, in the former case a blockade and in the latter case hypermobility. However, this test is also a provocation test for the posterior longitudinal ligament in particular and will result in an increase in the deep, dull low back pain that is typical of this structure and is difficult to localize.

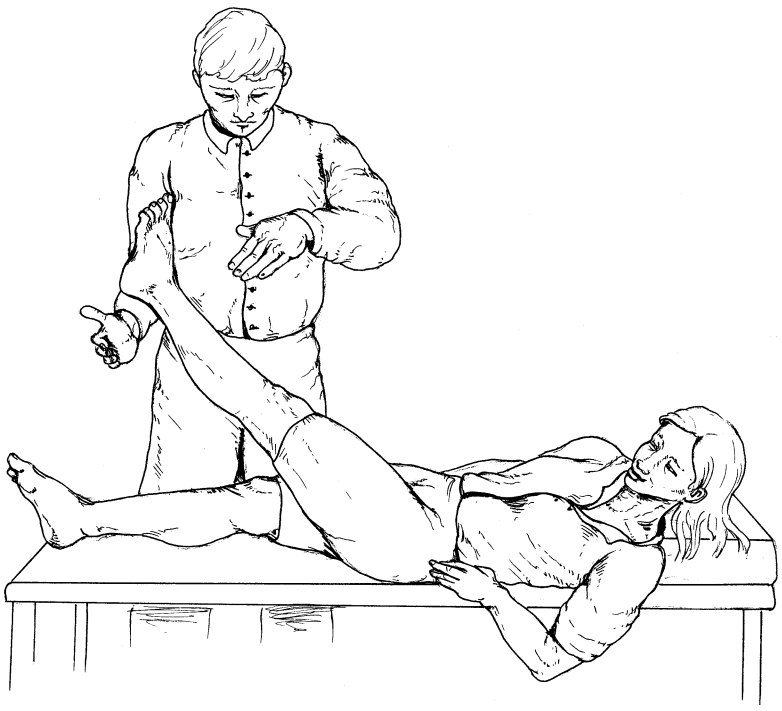


Fig. 1.37 Lasègue straight leg drop test.

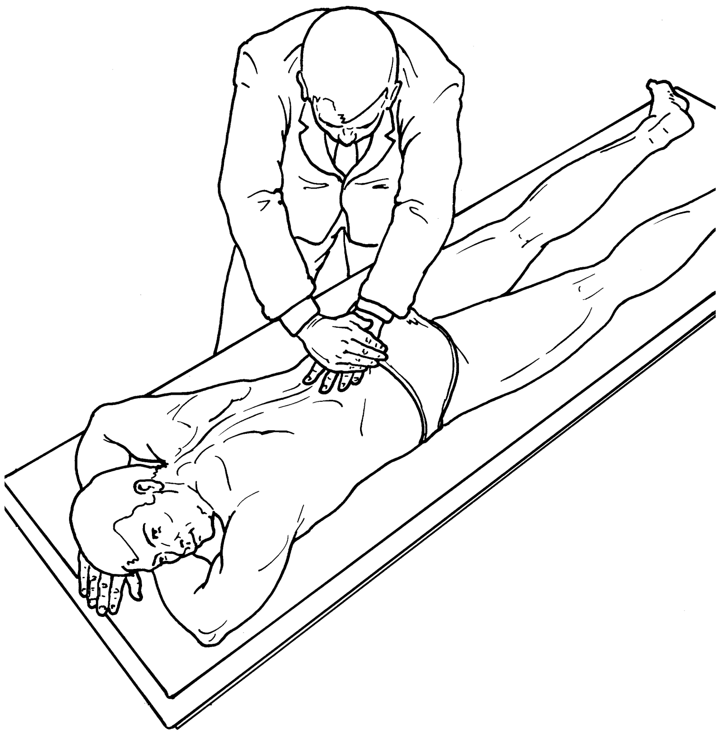


Fig. 1.38 Springing test.

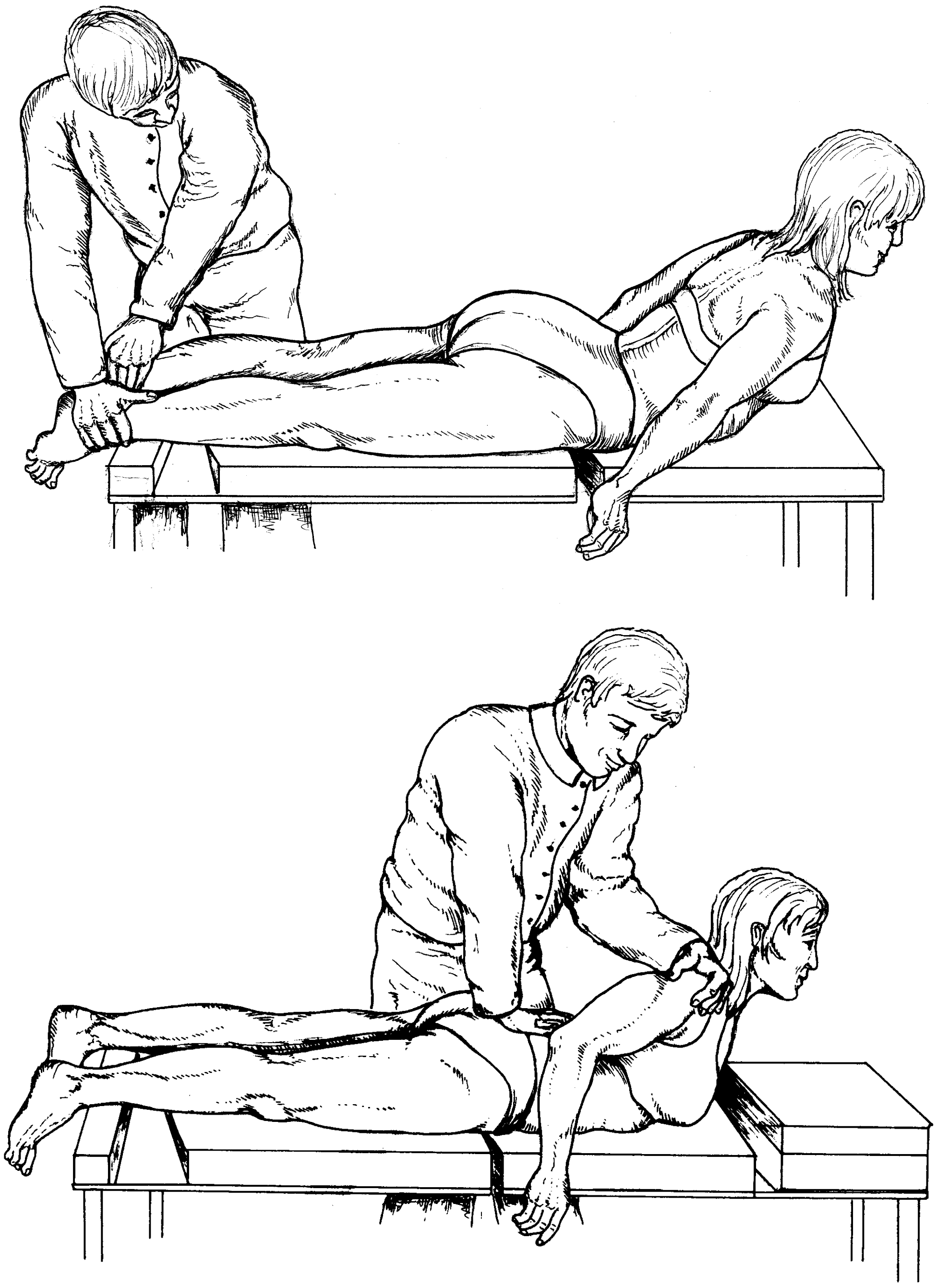
Hyperextension Test

Indicates a lumbar spine syndrome.

□ **Procedure:** The patient is prone. The examiner immobilizes both the patient's legs and asks the patient to raise his or her torso.

In the second phase of the examination, the examiner passively extends the patient's spine and adds a rotational motion. The examiner's other hand rests on the patient's lumbar spine and is used to assess both the mobility in the lumbar spine and the level of the painful site.

□ **Assessment:** Where segmental dysfunction in the lumbar spine is present, active extension of the lumbar spine will elicit or increase pain. The passive extension with an additional rotational motion allows the examiner to assess diminished segmental and/or regional mobility. A hard end point of the range of motion suggests degenerative changes, whereas a soft end point more probably suggests shortening of the longissimus thoracis and iliocostalis lumborum.



a

b

Fig. 1.39 Hyperextension test. **(a)** Active hyperextension. **(b)** Passive hyperextension and rotation.

One-Leg Standing (Stork Standing), Lumbar Extension Test

Evaluation of facet joint dysfunction.

□ **Procedure:** The patient extends the spine while balancing on one leg. The test is repeated with the patient standing on the opposite leg.

□ **Assessment:** The test is positive if back pain occurs. This can be associated with an intra-articular stress fracture in spondylolisthesis. If the stress fracture is unilateral, standing on the leg of the affected side causes more pain. If pain occurs on extension combined with rotation, this is an indicator of possible facet joint pathology on the side to which the spine is rotated.

Fig. 1.40 One-leg standing test.

Supported Forward Bend Test (Belt Test)

Differentiates lumbar pain from iliosacral pain.

□ **Procedure:** The patient is standing. The examiner stands behind the patient and asks the patient to bend forward until lumbosacral pain is felt. The patient then returns to the upright position. The examiner again asks the patient to bend forward. This time the examiner supports the patient's sacrum with his or her thigh and guides the motion by grasping both ilia.

□ **Assessment:** Forward bending requires normal function in the sacroiliac joint and the lumbosacral junction as well as mobility in the individual segments of the lumbar spine. Pain in unguided motion suggests a sacroiliac syndrome; this pain will improve or disappear in guided motion with the pelvis immobilized.

Changes in the lumbar spine will produce pain in forward bending with or without support.

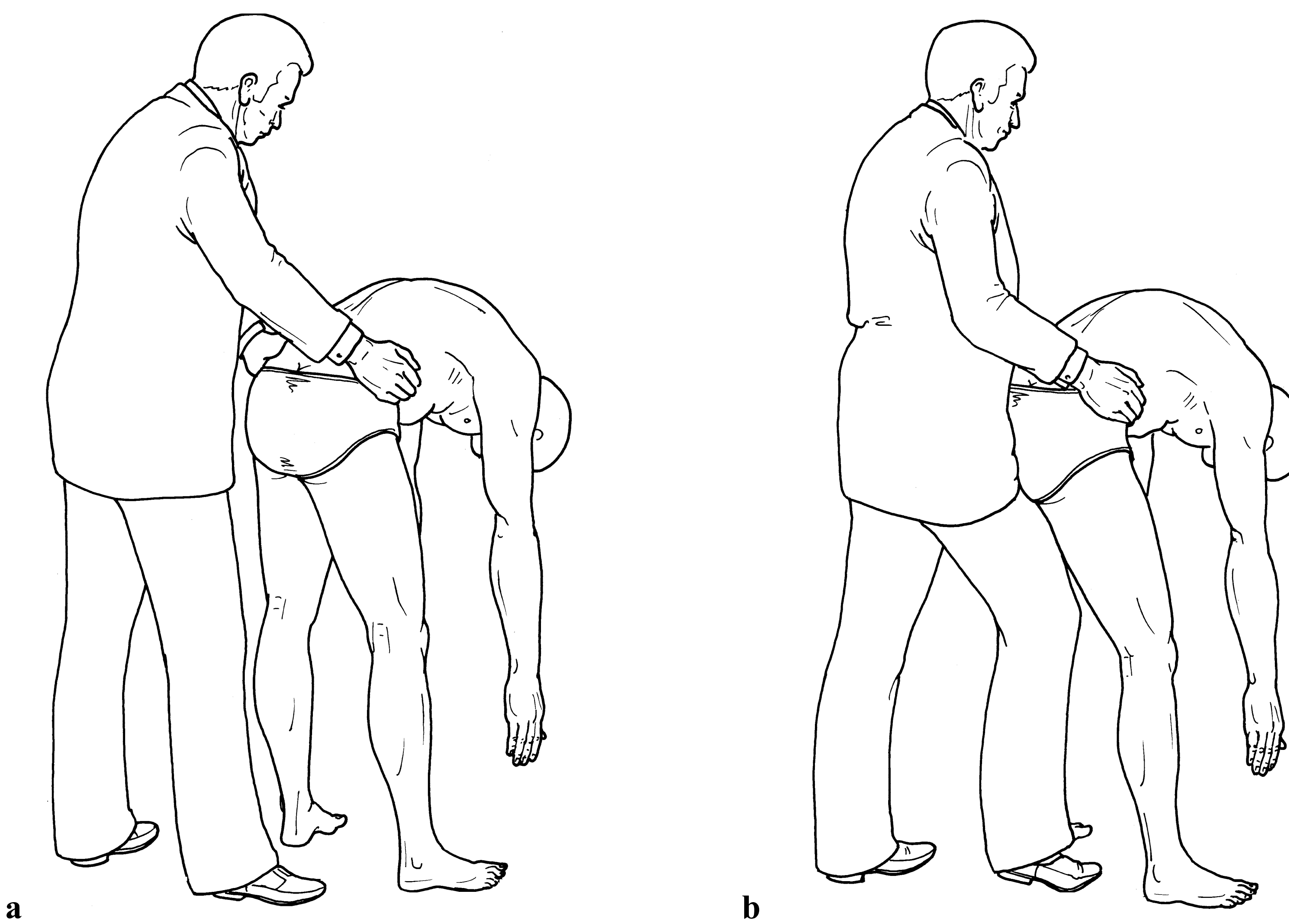


Fig. 1.41 Supported forward bend test. **(a)** Forward bending without support. **(b)** Supported forward bending.

Nerve Root Compression Syndrome

Disk extrusions usually lead to nerve compression syndromes with radicular pain. The pain in the sacrum and leg is often exacerbated by coughing, sneezing, pushing, or even simply walking. Mobility in the spine is severely limited by pain, and there is significant tension in the lumbar musculature. Sensory and motor deficits and impaired reflexes are additional symptoms that occur with nerve root compression.

Often the affected nerve root can be identified by the description of the paresthesia and radiating pain in the dermatome. Extrusions of the fourth and fifth lumbar disks are especially common, while extrusions of the third lumbar disk are less so. Disk extrusions involving the first and second lumbar disks are rare.

The Lasègue sign is usually positive (often even at 20–30°) in compression of the L5–S1 nerve root (typical sciatica). In these cases, even passively raising the normal leg will often elicit or exacerbate pain in the lower back and the affected leg (crossed Lasègue sign). In nerve root compression syndromes from L1 through L4 with involvement of the femoral nerve, the Lasègue sign is usually only slightly positive.

When the femoral nerve is irritated, the reverse Lasègue sign and/or pain from stretching of the femoral nerve can usually be triggered.

Pseudo-radicular pain must be distinguished from genuine radicular pain (sciatica). Pseudo-radicular pain is usually less circumscribed than radicular pain. Facet syndrome (arthritis in the facet joints), sacroiliac joint syndrome, painful spondylolisthesis, stenosis of the spinal canal, and postdiskectomy syndrome are clinical pictures that frequently cause pseudo-radicular pain.

Neurodynamic testing is used to evaluate the mechanical extensibility and/or compression of neural structures of the lumbar spine. These tests include the slump test, the Lasègue test (straight leg raising), the reverse Lasègue test, and the femoral nerve traction test.

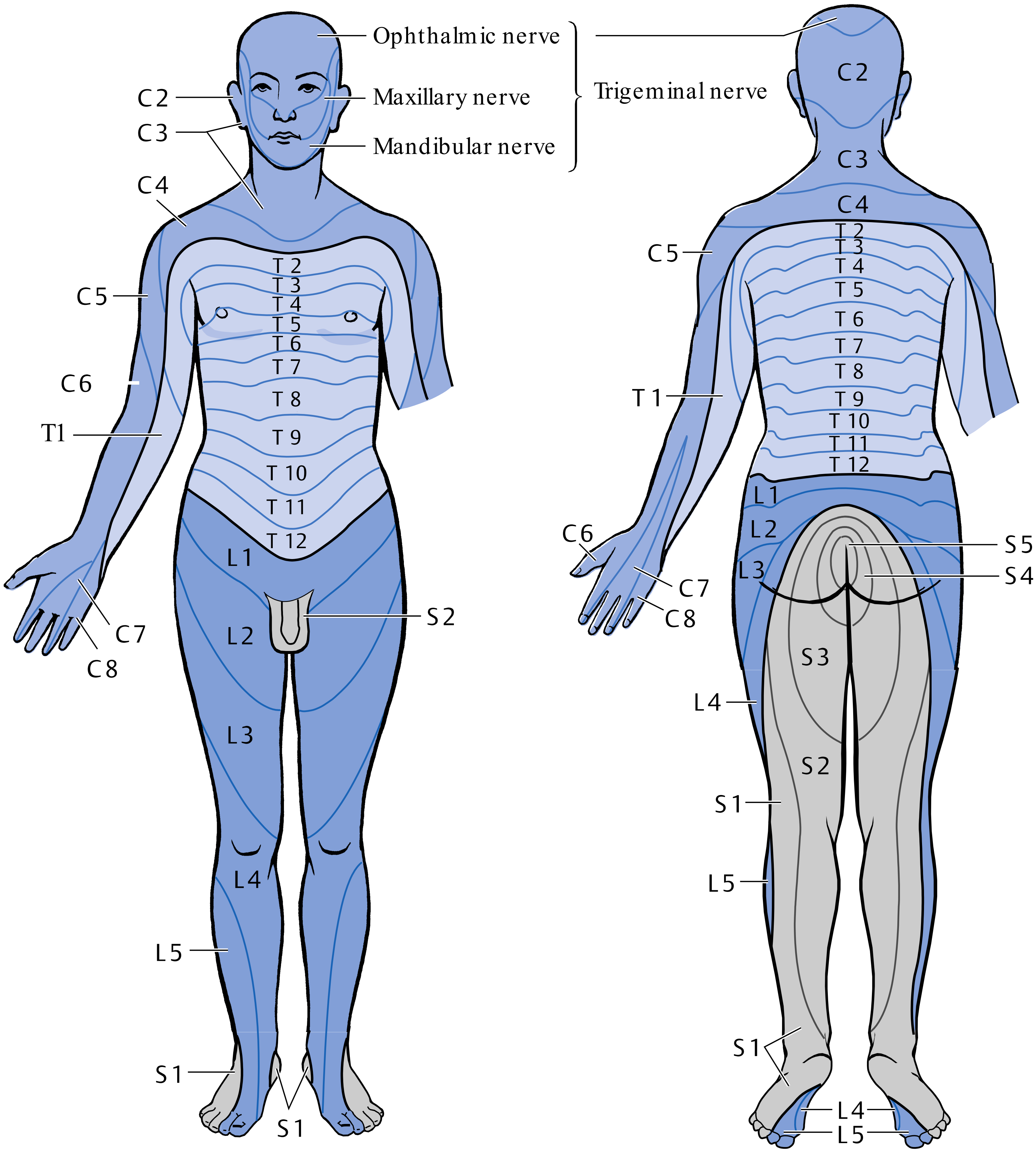


Fig. 1.42 Segmental innervation of the skin (after Hansen and Schliack).

1

Table 1.2 Signs of radicular symptoms

Root	Dermatome		Paralyzed muscles	Impaired reflexes
	Pain	Sensory deficit		
L2 L1–L2 Extraforaminal: L2–L3	Thoracolumbar junction, sacroiliac joint, groin, iliac crest, proximal medial thigh	Groin, proximal anterior and medial thigh	Paresis of the iliopsoas, quadriceps femoris, and adductors (slight)	Cremasteric and patellar reflex weakened
L3 L2–L3 Extraforaminal: L3–L4	Upper lumbar spine, anterior proximal thigh	From the anterior thigh to the medial thigh and distal to the knee	Paresis of the iliopsoas, quadriceps femoris, and adductors (slight)	Absent or weakened patellar reflex
L4 L2–L3 Extraforaminal: L3–L4	Lumbar spine, anterolateral thigh, hip region	From the lateral thigh to the medial lower leg and margin of the foot	Paresis of the quadriceps femoris and tibialis anterior (difficulty walking on heels)	Weakened patellar reflex
L5 L4–L5 Extraforaminal: L5–S1	Lumbar spine, posterior thigh, lateral lower leg, medial foot, groin, hip region	From the lateral lower leg to the medial foot (great toe)	Paresis of the extensor hallucis longus and brevis, extensor digitorum longus and brevis (difficulty walking on heels)	Loss of tibialis posterior reflex (significant only when readily elicited on contralateral side)
S1 L5–S1	Lumbar spine, posterior thigh, posterolateral lower leg, lateral margin of foot, sole of foot, groin, hip region, coccyx	Posterior aspect of the thigh and lower leg, lateral margin of the foot and sole of the foot (little toe)	Paresis of the peroneus muscles and triceps surae (difficulty walking on tiptoes; foot bends laterally)	Weakening or loss of Achilles tendon reflex

Slump Test

□ **Procedure:** The patient sits upright on the examining table with the legs hanging loosely over the edge of the table. The hips are in a neutral position and the hands are placed behind the back. The examination is performed step by step.

First the patient is asked to "slump" the back into thoracic and lumbar flexion while the examiner supports the head to keep it in a neutral position. With one arm, the examiner then applies pressure across the shoulders to maintain increased flexion in the thoracic and lumbar spine. At the same time, the patient is asked to actively flex the cervical spine and head as far as possible. Using the same hand, the examiner then applies pressure to maintain flexion in all three parts of the spine.

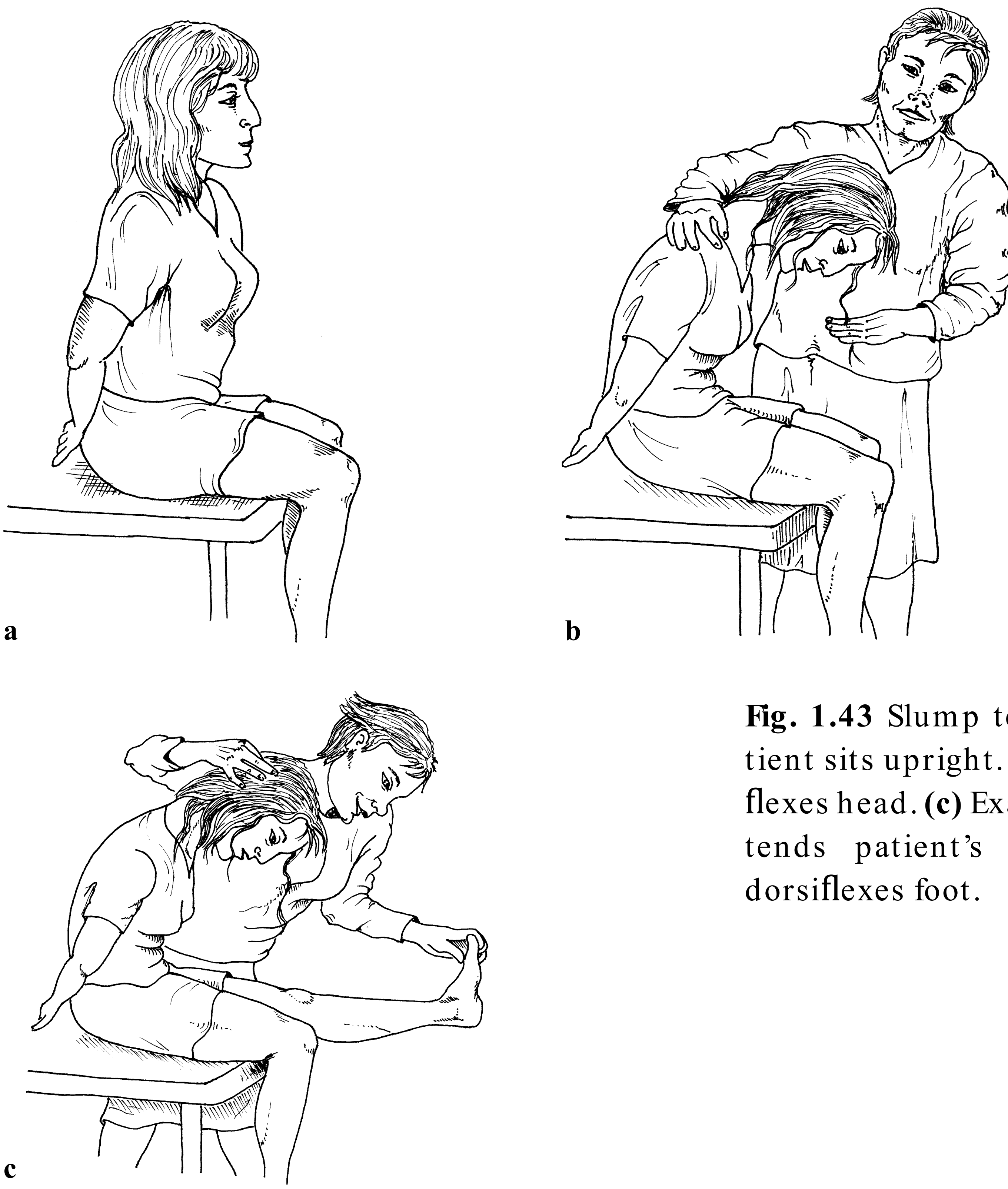


Fig. 1.43 Slump test. **(a)** Patient sits upright. **(b)** Patient flexes head. **(c)** Examiner extends patient's knee and dorsiflexes foot.

With the other hand the examiner holds the patient's foot in maximum dorsiflexion. In this position the patient is asked to actively straighten the knee as much as possible.

The test is then repeated with the other leg and, if possible, with both legs at the same time.

□ **Assessment:** The test can cause impingement or irritation of the dura and/or nerve roots, with pain radiating down into the areas supplied by the sciatic nerve.

If the patient is unable to extend the knee because of pain, the examiner reduces the pressure on the cervical spine and asks the patient to slowly raise the head. If the patient is then able to extend the knee further without pain or with less pain, the test is considered to be positive, indicating that neural structures are affected.

During the course of the test, pains may occur that must be defined in the differential diagnosis. These include facet joint arthritis, tight hamstring muscles and limited knee and ankle mobility.

Lasègue Sign (Straight Leg Raising Test)

Indicates nerve root irritation.

□ **Procedure:** The examiner slowly raises the patient's leg (extended at the knee) until the patient reports pain.

□ **Assessment:** Intense pain in the sacrum and leg suggests nerve root irritation (disk extrusion or tumor). However, a genuine positive Lasègue sign is only present where the pain shoots into the leg explosively along a course corresponding to the motor and sensory distribution of the affected nerve root.

The patient often attempts to avoid the pain by lifting the pelvis on the side being examined.

The angle achieved when lifting the leg is estimated in degrees. This angle gives an indication of severity of the nerve root irritation present (genuine Lasègue sign is at 60° or less).

Sciatica can also be provoked by adducting and internally rotating the leg with the knee flexed. This test is also described as a Bonnet or piriformis sign (adduction and internal rotation of the leg stretches the nerve as it passes through the piriformis).

Increases in sciatic pain on raising the head (Kernig sign) and/or passive dorsiflexion of the great toes (Turyn sign) are further signs of significant sciatic nerve irritation (differential diagnosis should consider meningitis, subarachnoid hemorrhage, and carcinomatous meningitis).

Sacral or lumbar pain that increases only slowly as the leg is raised or pain radiating into the posterior thigh is usually attributable to degenerative joint

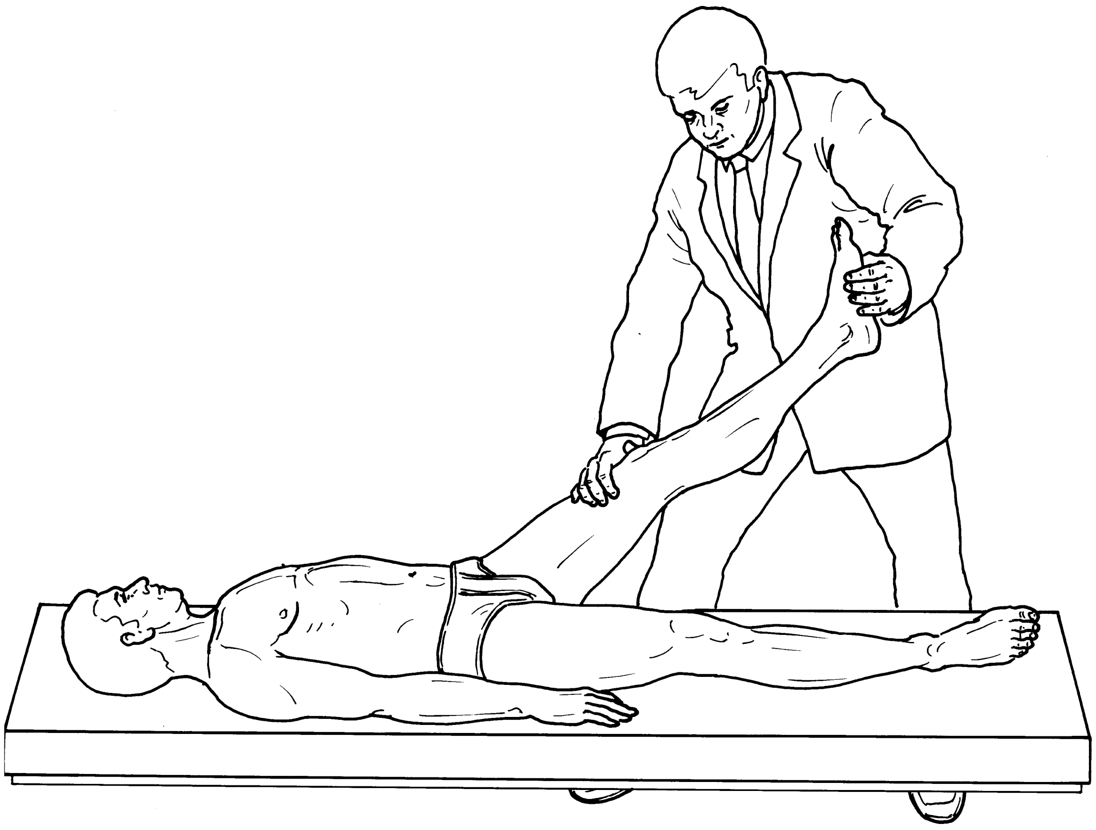


Fig. 1.44 Lasègue sign.

disease (facet syndrome), irritation of the pelvic ligaments (tendinitis), or increased tension or shortening in the hamstrings (indicated by a soft end point, usually also found on the contralateral side). It is important to distinguish this "pseudo-radicular" pain (pseudo-Lasègue sign) from genuine sciatica (true Lasègue sign).

Normally the leg can be raised 15 to 30° before the nerve root is stretched in the intervertebral foramen. Pain occurring at over 60° of flexion suggests joint pain in the lumbar area (e.g., facet joints or sacroiliac joints).

It is important to differentiate whether one is dealing with joint pain, soft tissue pain, or nerve root pain.

If one leg is lifted and pain occurs on the opposite side, it suggests a herniated disk or a tumor. This may be called the crossover sign and usually indicates a rather large medial intervertebral disk protrusion.

Lasègue and crossover signs give an indication of the degree of disk injury. Many patients with large, central disk herniations are candidates for surgery, especially if there are bowel and bladder symptoms.

For patients who have difficulty lying supine, a modified straight leg raising test in the lateral position is possible. The patient lies with the test leg uppermost with the hip and knee flexed to 90°. The lumbosacral spine is normally in a neutral position, but may be slightly flexed or extended as the patient's condition allows. The examiner then slowly extends the patient's knee passively on the affected side. The occurrence of pain or resistance to stretching the knee indicate a positive test.

It can also be impossible to lift the leg at the hip if the patient consciously resists and attempts to press the leg downward against the examiner's hand. Occasionally one will encounter this behavior in experienced patients undergoing examination within the scope of an expert opinion (see Lasègue test with the patient seated).

Reverse Lasègue Test (Femoral Nerve Lasègue Test)

Indicates nerve root irritation.

□ **Procedure:** The patient is prone. The examiner passively raises the leg, which is flexed at the knee.

□ **Assessment:** Hyperextension of the hip with the knee flexed places traction on the femoral nerve. Occurrence of unilateral or bilateral radicular pain in the sacrum or anterior thigh and rarely in the lower leg is a sign of nerve root irritation, such as a herniated disk, in the L3/L4 spinal column segment. This should be differentiated from complaints caused by degenerative hip disease or from a shortened rectus femoris or psoas muscle.

The reverse Lasègue test (femoral nerve Lasègue test) can also be carried out with the patient in the lateral position. The patient lies on the unaffected side with the affected extremity slightly flexed at the hip and knee. The examiner fixes the pelvis with one hand and grasps the patient's knee with the other and guides the hip into a hyperextended position while increasingly flexing the knee.

Radicular pain in the anterior thigh suggests nerve root irritation.

This test evaluates the nerve roots of the midlumbar spine (L2 to L4).

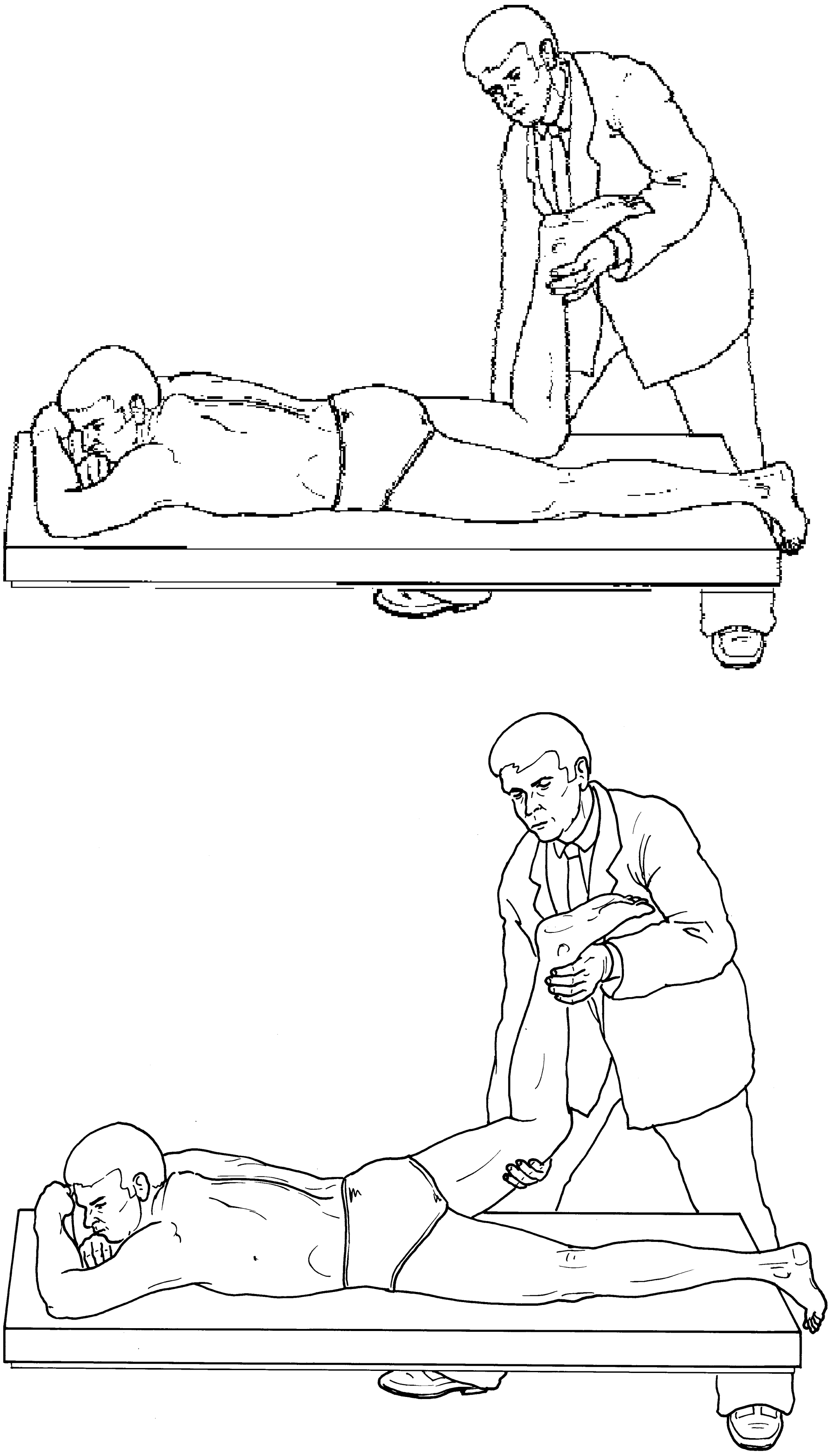
As with the Lasègue test, symptoms may also occur on the opposite side, which is then referred to as a crossed femoral stretching test.

Pain in the groin and hip region radiating along the medial side of the thigh suggests an L3 origin, while pain over the anterior lower leg indicates an L4 problem.

This test is similar to the Ober test used to evaluate a contracted, shortened iliotibial band. A tight iliotibial band does not allow the leg to adduct to any significant degree, and is often pain free. If pain is present, it is localized over the greater trochanter proximally and the lateral condyle distally.

Femoral nerve pain usually has a different history and is normally more distinct and dermatome-related.

□ **Note:** In the case of a disk herniation at the L3/L4 level, there is weakness of the quadriceps muscle combined with an absent or weakened patellar tendon reflex.



a

b

Fig. 1.45 Reverse Lasègue test. **(a)** Starting position. **(b)** Hip hyperextended.

Bonnet Sign (Piriformis Sign)

□ **Procedure:** The patient lies supine with the leg flexed at the hip and knee. The examiner adducts and internally rotates the leg.

□ **Assessment:** The Lasègue sign occurs earlier in this maneuver. The nerve is stretched as it passes through the piriformis, resulting in increased pain.

Lasègue-Moutaud-Martin Sign (Contralateral Lasègue)

Indicates nerve root irritation.

□ **Procedure:** The patient lies supine. The examiner raises the nonpainful leg with the knee extended.

□ **Assessment:** If a herniated disk with nerve root irritation is present, raising the leg on the healthy side can cause sciatica on the affected side by transferring the movement to the affected spinal segment.

Bragard Test

Indicates nerve root compression syndrome, differentiating a genuine Lasègue sign from a pseudo-Lasègue sign.

□ **Procedure:** The patient is supine. The examiner grasps the patient's heel with one hand and anterior aspect of the knee with the other. The examiner slowly raises the patient's leg, which is extended at the knee. At the onset of

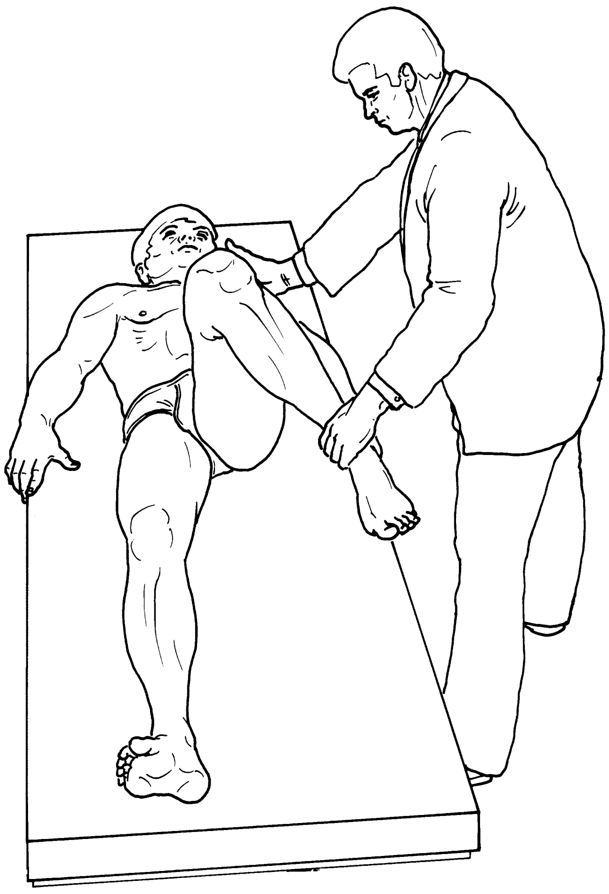


Fig. 1.46 Bonnet sign.

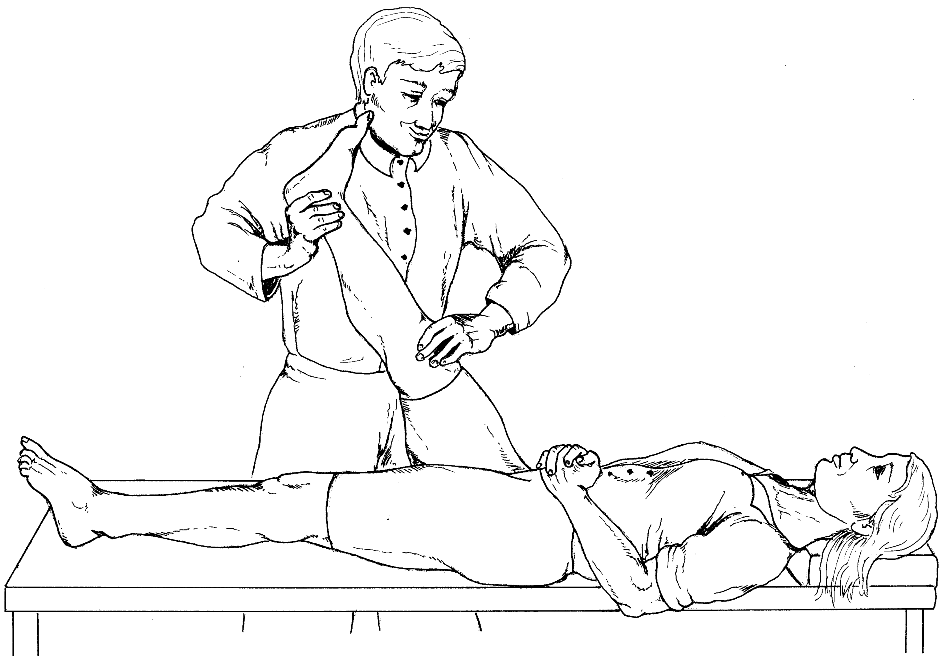
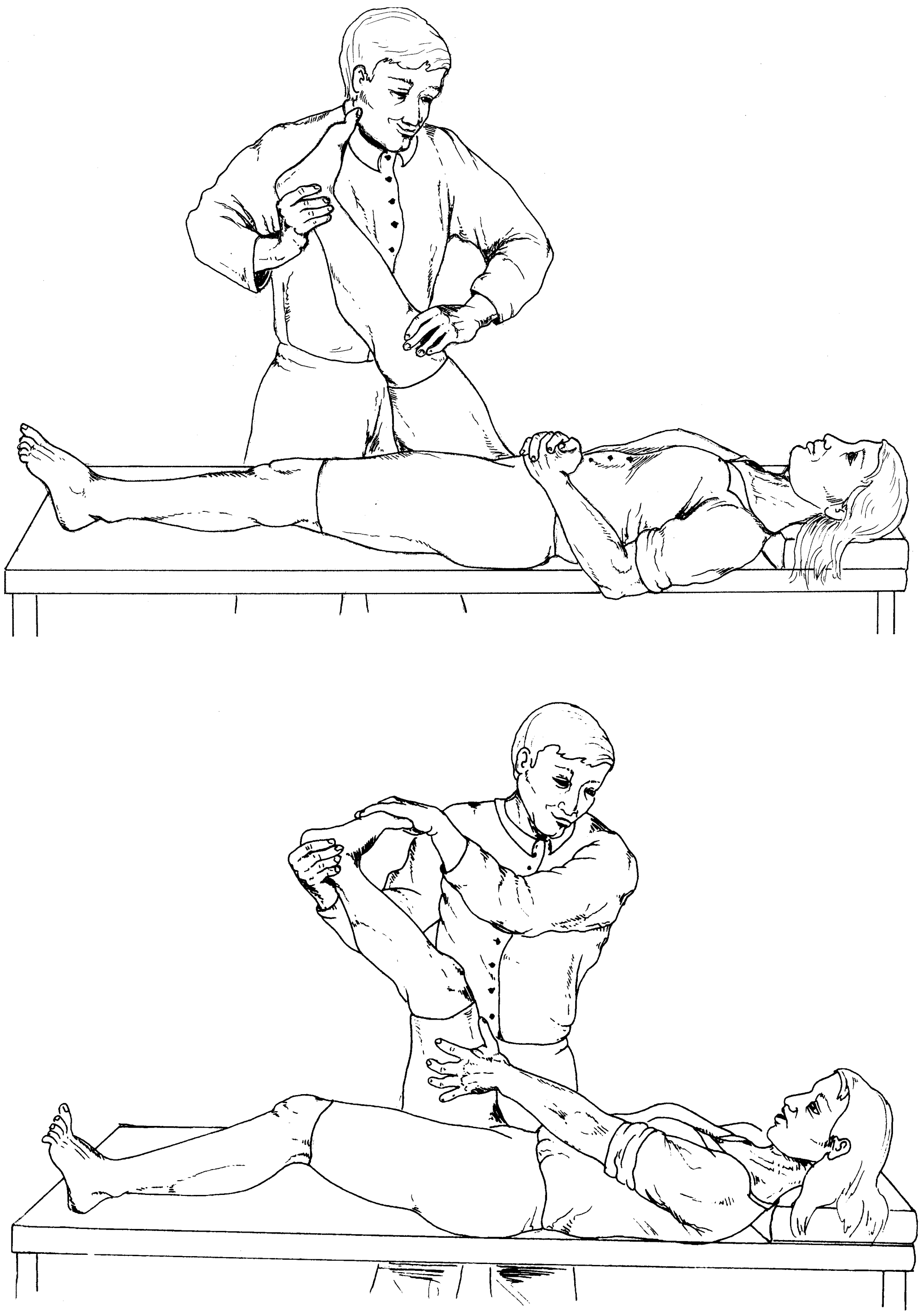


Fig. 1.47 Lasègue–Moutaud–Martin sign.



a

b

Fig. 1.48 Bragard test.
(a) Starting position.
(b) Dorsiflexion of the foot.

the Lasègue sign, the examiner lowers the patient's leg just far enough that the patient no longer feels pain. The examiner then passively moves the patient's foot into extreme dorsiflexion in this position, eliciting the typical pain caused by stretching of the sciatic nerve.

□ **Assessment:** A positive Bragard sign is evidence of nerve root compression, which may lie between L4 and S1.

Dull, nonspecific pain in the posterior thigh radiating into the knee is attributable to stretching of the hamstrings and should not be assessed as a Lasègue sign.

A sensation of tension in the calf may be attributable to thrombosis, thrombophlebitis, or contracture of the gastrocnemius.

The Bragard sign can be used to test whether the patient is malingering. The sign is usually negative in malingerers.

Lasègue Differential Test

Differentiates sciatica from a hip disorder.

□ **Procedure:** The patient is supine. The examiner grasps the patient's heel with one hand and the anterior aspect of the knee with the other. The examiner slowly raises the patient's leg, which is extended at the knee, until the patient feels pain. The examiner then notes the location and nature of the pain and estimates in degrees the maximum pain-free angle that can be achieved when lifting the leg. The test is repeated and the leg is then flexed once the painful angle is reached.

□ **Assessment:** In a patient with sciatic nerve irritation, flexing the knee will significantly reduce symptoms, even to the point that they disappear completely. Where a hip disorder is present, the pain will remain and may even be exacerbated by increasing flexion in the hip.

□ **Note:** Pain in hip disorders is usually located in the groin and only rarely in the posterolateral region of the hip. Only in the case of posterolateral pain may

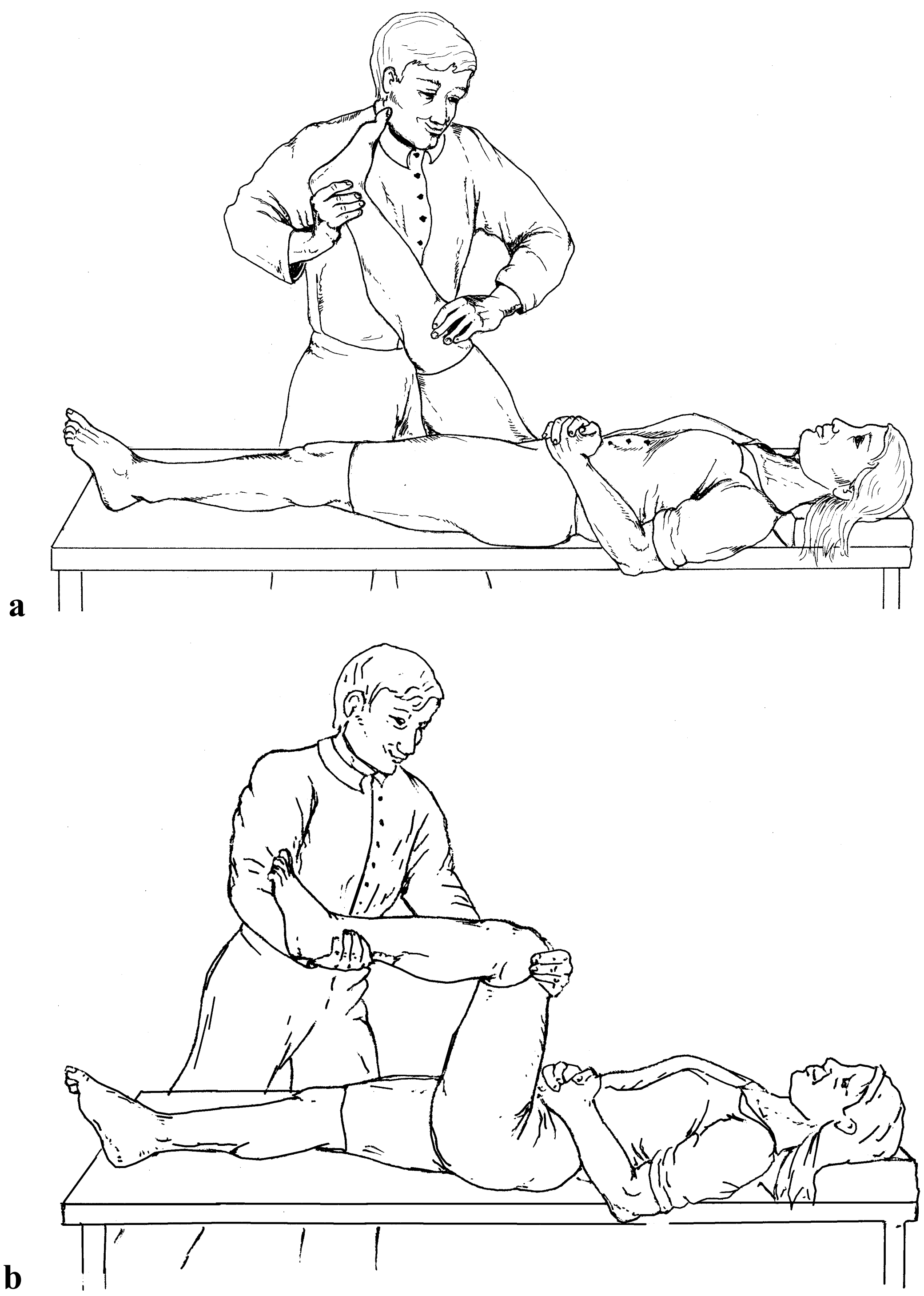


Fig. 1.49 Lasègue differential test. **(a)** Starting position. **(b)** Knee flexed.

it be hard to differentiate between nerve root irritation and pain caused by a hip disorder.

Duchenne Sign

Assesses a nerve root disorder.

□ **Procedure:** The patient is supine. The examiner grasps the patient's heel with one hand and uses one finger of the other hand to press the first metatarsal head toward the dorsum of the foot. From this position, the patient is asked to plantarflex the foot.

□ **Assessment:** In the presence of a disk disorder involving the S1 nerve root, the patient will be unable to resist the finger pressure. Paresis of the peroneus muscles causes supination of the foot due to the action of the anterior and posterior tibial muscles.

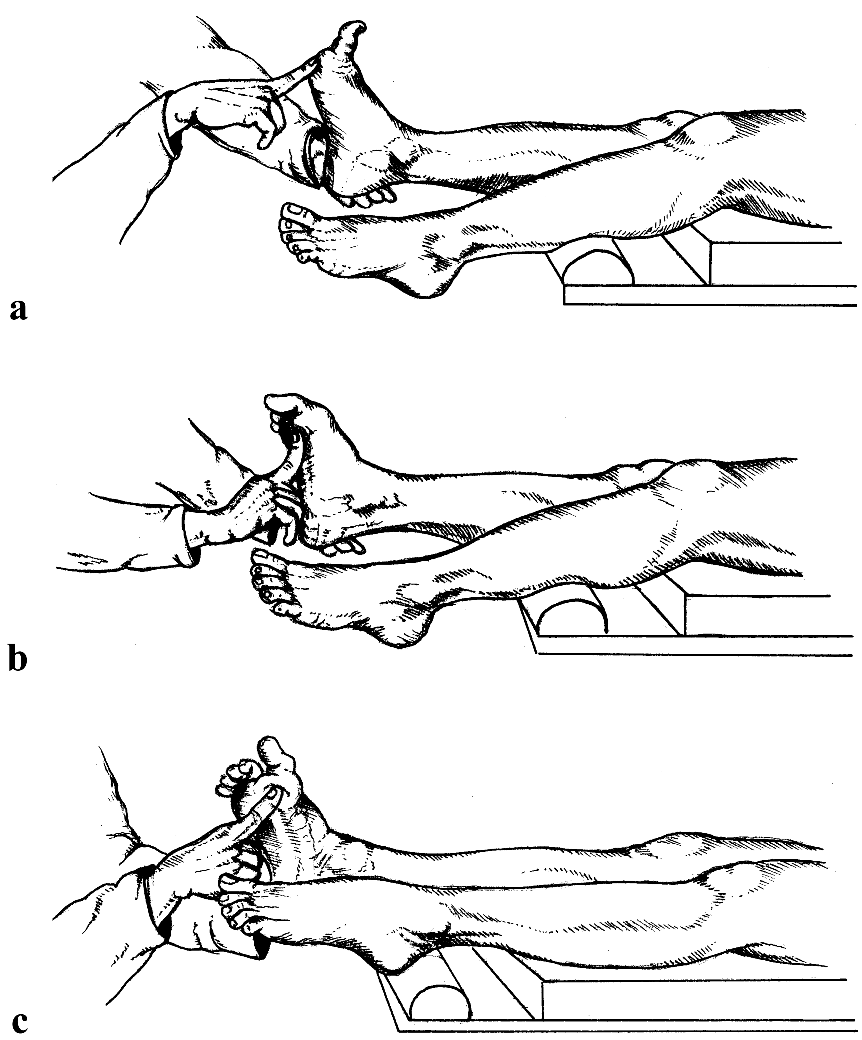


Fig. 1.50 Duchenne sign. **(a)** Starting position. **(b)** Normal. **(c)** Abnormal.

Kernig–Brudzinski Test

Indicates nerve root or dural irritation.

□ **Procedure:** The patient is supine and is asked to flex the hip and knee of one leg. In the first part of the test, the examiner attempts to passively extend the patient's knee. In the second part, the patient is asked to actively extend the knee.

□ **Assessment:** Pain in the spine or radicular pain in the leg occurring during active or passive knee extension suggests nerve root irritation from a disk herniation or from an inflammatory or tumorous process in the spine.

If the test is negative in spite of suspicion of nerve root irritation, the examiner may further increase the tension on the nerve root and dura by flexing the cervical spine passively—the Brudzinski portion of the test.

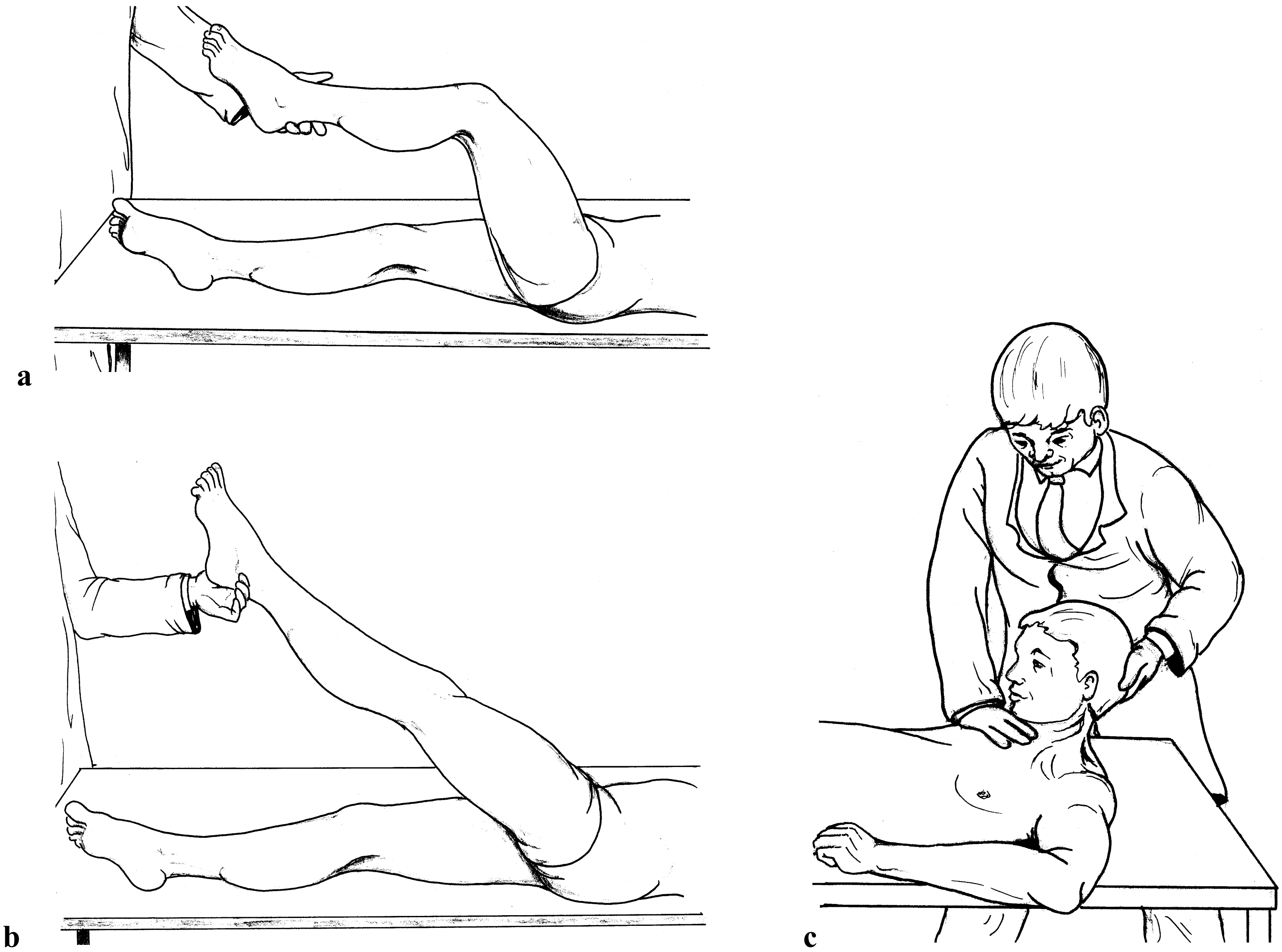


Fig. 1.51 Kernig–Brudzinski test. **(a)** Starting position. **(b)** Knee extended. **(c)** Brudzinski sign.

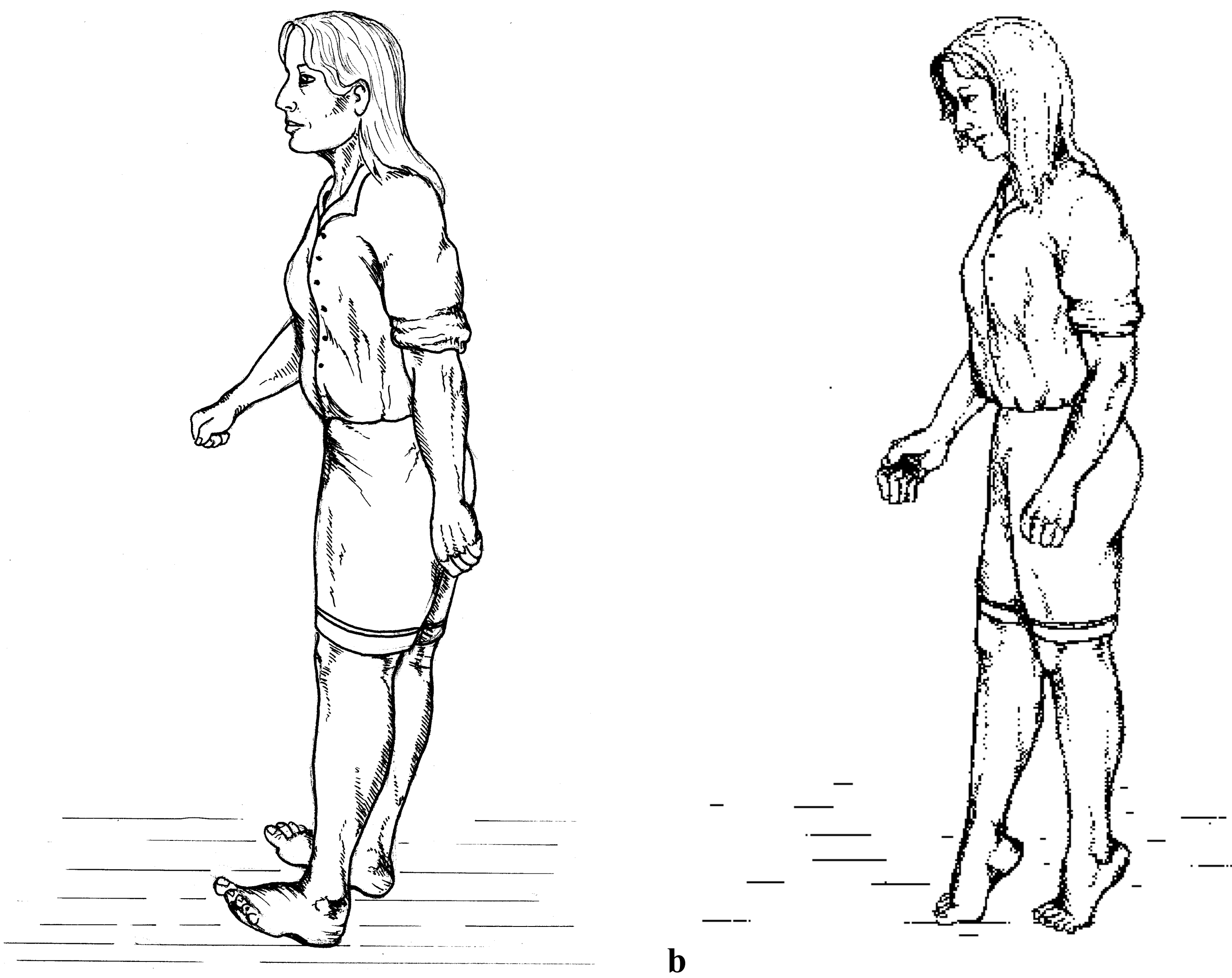
Tiptoe and Heel Walking Test

Identifies and assesses a nerve root disorder in the lumbar spine.

□ **Procedure:** The patient is asked to stand first on his or her heels, then on tiptoe, and then to take a few steps in each of these positions if possible.

□ **Assessment:** Difficulty or inability to stand or walk on tiptoe suggests a lesion of the S1 nerve root; difficulty or inability to stand or walk on the heels suggests a lesion of the L4–L5 nerve root.

□ **Note:** A differential diagnosis must exclude a ruptured Achilles tendon. This injury makes it impossible to stand on tiptoe, especially when standing only on the affected leg.



a **b**

Fig. 1.52 Tiptoe and heel walking test. **(a)** Heel walking. **(b)** Tiptoe walking.

Lasègue Test with the Patient Seated

Indicates nerve root irritation.

□ **Procedure:** The patient sits on the edge of the examining table and is asked to flex his or her hip with the leg extended at the knee.

□ **Assessment:** This test corresponds to the Lasègue sign. When nerve root irritation is present, the patient will avoid the pain by leaning backward and using his or her arms for support. This test can also be used to identify simulated pain. If the patient can readily flex the hip without leaning backward, then a previous positive Lasègue sign must be questioned. The examiner can also perform this test in the same manner as the test for the Lasègue sign by passively flexing the hip with the knee extended.

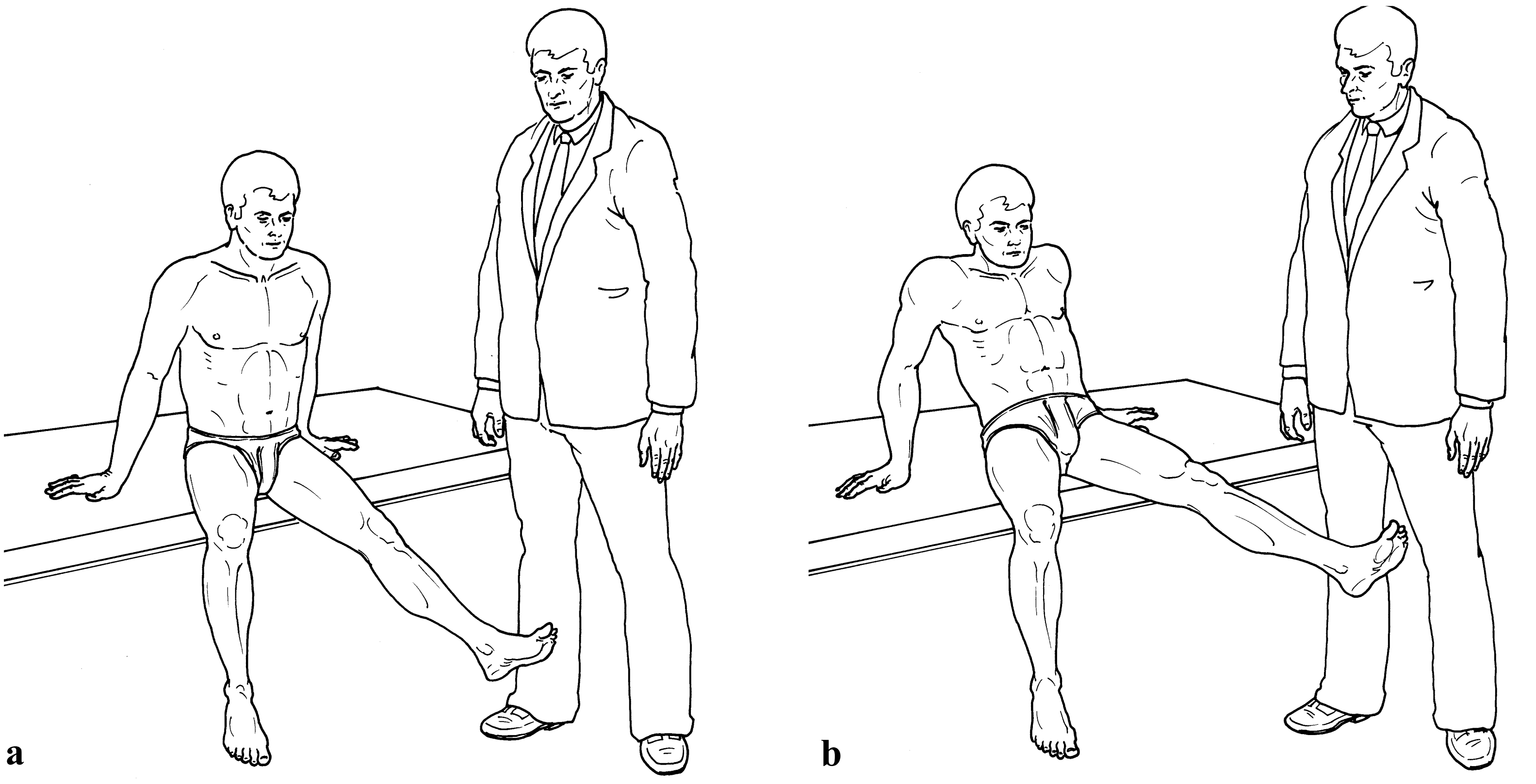


Fig. 1.53 Lasègue sign with the patient seated. **(a)** Beginning hip flexion. **(b)** With increasing hip flexion.

Hoover Test

Evaluation of malingering.

- **Procedure:** The patient lies supine. The examiner places one hand under each calcaneus. The patient is then asked to perform active straight leg raising. The knee is kept straight and the leg is actively lifted off the table.
- **Assessment:** If the patient does not lift the leg or the examiner does not feel pressure under the opposite heel, the patient is probably not really trying or may be malingering. If the lifted limb is weaker, pressure under the normal heel increases, because of the increased effort to lift the weak leg off the examination table. Often patients will report that they cannot raise the leg at all. Both sides have to be compared for differences.

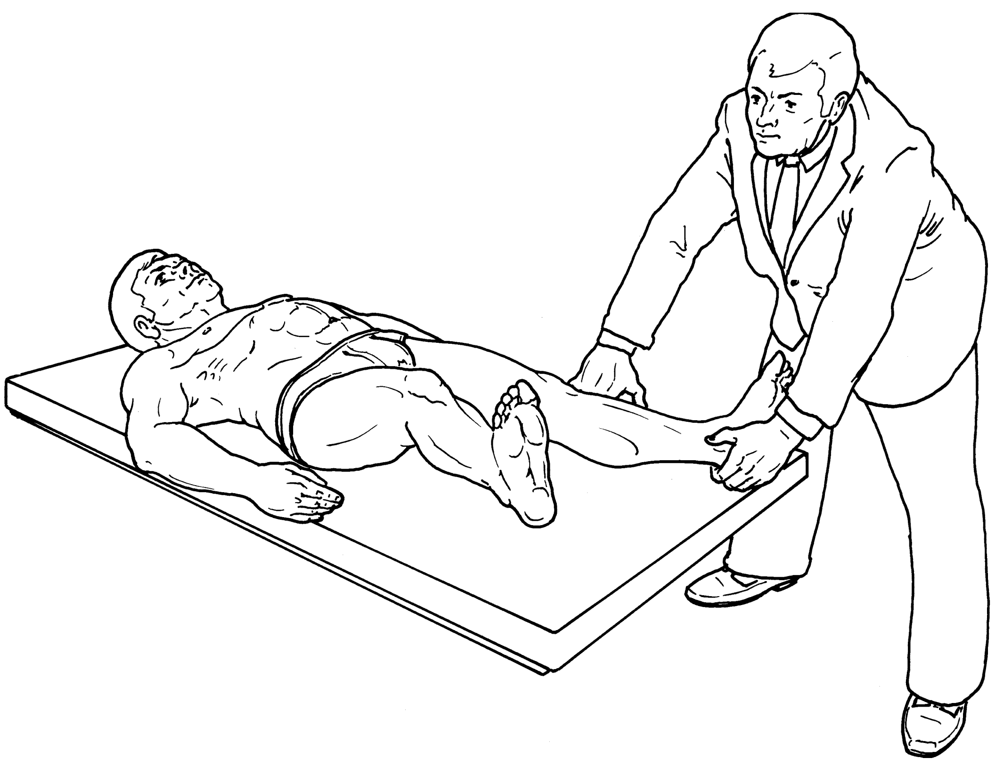


Fig. 1.54 Hoover test.

1

Sacroiliac Joint

The sacrum forms the base of the spine and is connected to the two halves of the pelvis (the ilia) by articulations known as the sacroiliac joints. While these articulations are true joints in the anatomical sense, from a functional standpoint they may be regarded as symphyses: the tight ligaments surrounding the bone and the crescentic shape and uneven contour of the articular surfaces effectively minimize mobility in these joints. In spite of this, compensatory movements between the spine and pelvis can result in significant impairments in this joint that can eventually affect the entire spine and the joints of the lower extremities.

Motion restriction or instability of a sacroiliac joint can develop secondary to trauma, dislocation, or pelvic fractures. However, they may also develop as a result of asymmetric loads on the pelvis or for other reasons. Pain during motion will be felt in the sacroiliac, gluteal, inguinal, and trochanteric regions. Usually it will radiate posteriorly within the S1 dermatome as far as the knee, occasionally producing symptoms resembling sciatica. Often patients will also experience pain in the lower abdomen and groin due to tension in the iliopsoas. Sacroiliac joint symptoms usually manifest themselves as tenderness to palpation and tapping in the parasacral region adjacent to the sacroiliac joints.

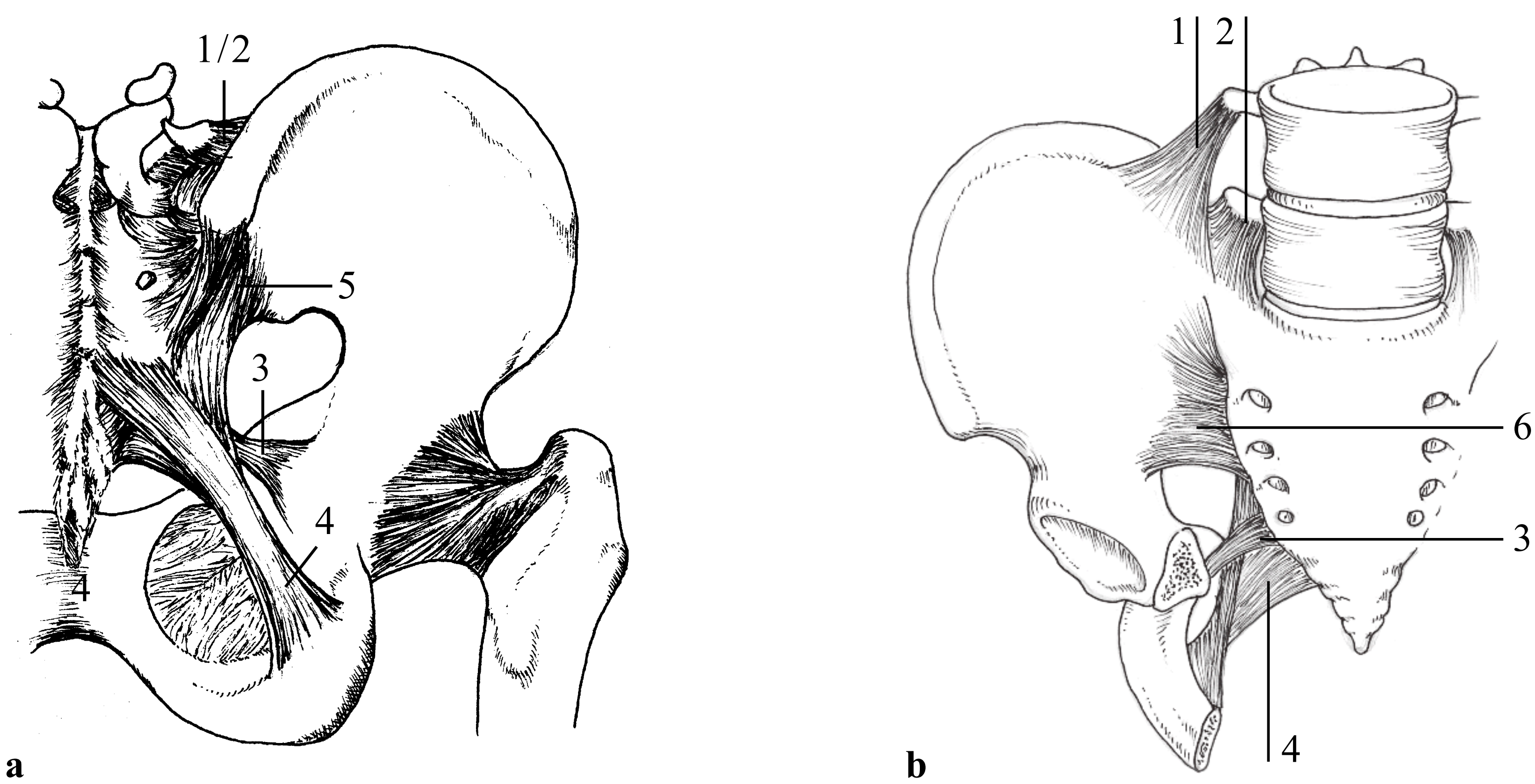


Fig. 1.55 Ligaments of the pelvis. **(a)** Anterior view. **(b)** Posterior view:
1, 2 Iliolumbar ligament.
3 Sacrospinous ligament.
4 Sacrotuberous ligament.
5 Posterior sacroiliac ligament.
6 Anterior sacroiliac ligament.

A number of manipulative tests may be performed on the standing, supine, or prone patient to identify functional impairments in the sacroiliac joints.

□ **Function and provocation tests of the sacroiliac joint:**

1. Mennell sign.
2. Springing test.
3. Mobilization test.
4. Standing flexion test.
5. Variable leg length.
6. Spine test.
7. Patrick test (Fabere test).
8. Three-phase hyperextension test.

Ligament Tests

Functional assessment of the pelvic ligaments.

□ **Procedure:** The patient is prone.

1. To evaluate the iliolumbar ligament, the patient's knee and hip are flexed and the examiner then adducts the leg to the contralateral hip. While executing this maneuver, the examiner presses on the knee to exert axial pressure on the femur.
2. To evaluate the sacrospinous and sacroiliac ligaments, the patient's knee and hip are maximally flexed and the examiner adducts the leg toward the contralateral shoulder. While executing this maneuver, the examiner presses on the knee to exert axial pressure on the femur.
3. To evaluate the sacrotuberous ligament, the patient's knee and hip are maximally flexed and the examiner moves the leg toward the ipsilateral shoulder.

□ **Assessment:** Stretching pain occurring within a few seconds suggests functional shortening and excessive stresses on the ligaments, although it can also occur in a hypermobile or motion-restricted sacroiliac joint.

Pain caused by stretching the iliolumbar ligament is referred to the inguinal region (the differential diagnosis includes a hip disorder). Pain caused by stretching the sacrospinous and sacroiliac ligaments is felt within the S1 dermatome from a point posterolateral to the hip as far as the knee. Sacrotuberous ligament pain radiates into the posterior aspect of the thigh.

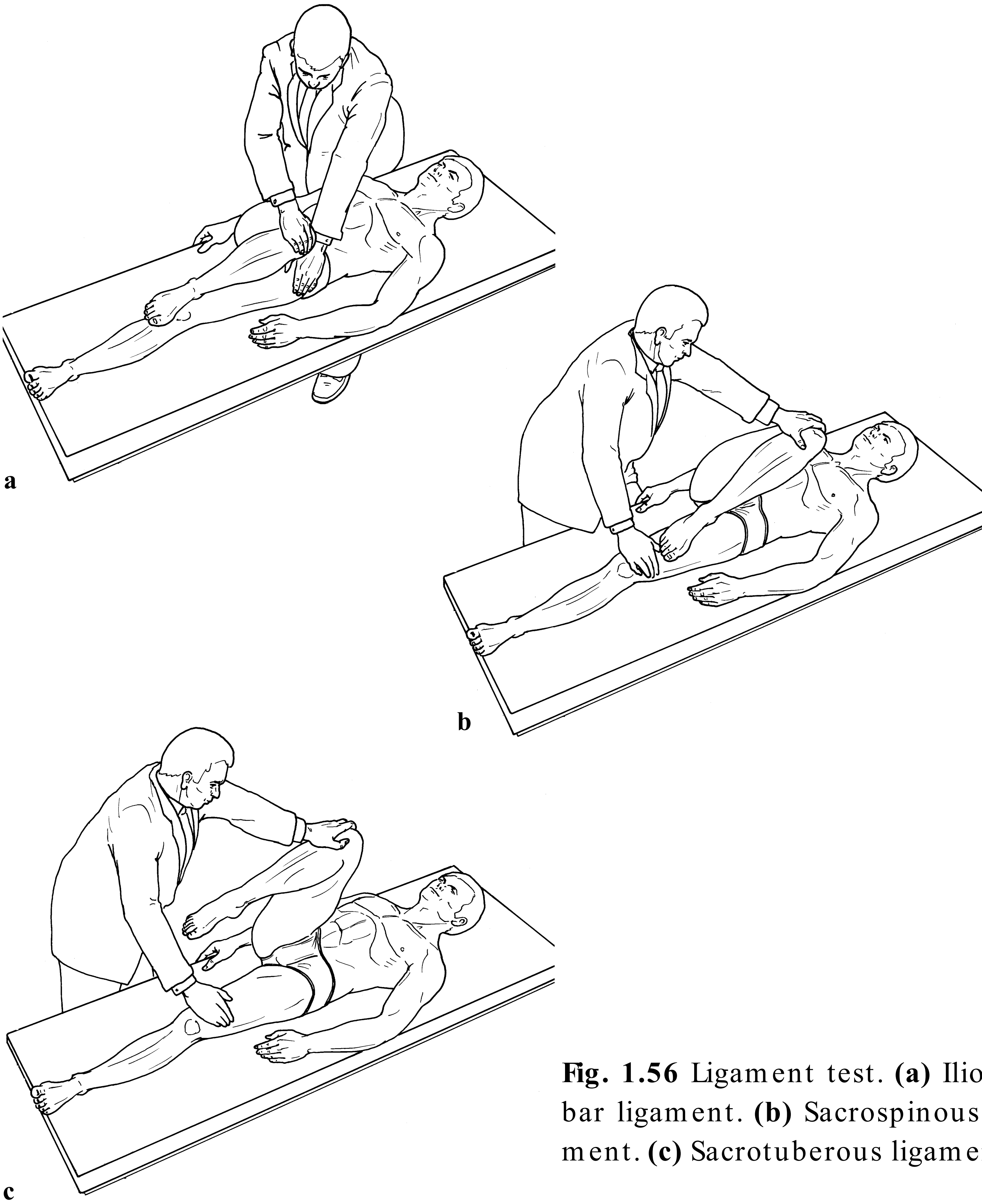


Fig. 1.56 Ligament test. **(a)** Iliolumbar ligament. **(b)** Sacrospinous ligament. **(c)** Sacrotuberous ligament.

Springing Test 2

Assesses facet hypermobility in the sacroiliac joint.

□ **Procedure:** The examiner places the index finger of one hand first on the superior margin of the sacroiliac joint and then on its inferior margin (S1–S3) in such a manner that the fingertip lies on the sacrum and the volar aspect of the distal phalanx lies on the medial margin of the ilium.

The examiner's other hand grasps the index finger and exerts posteroanterior pressure, which the palpating finger transmits to the sacrum.

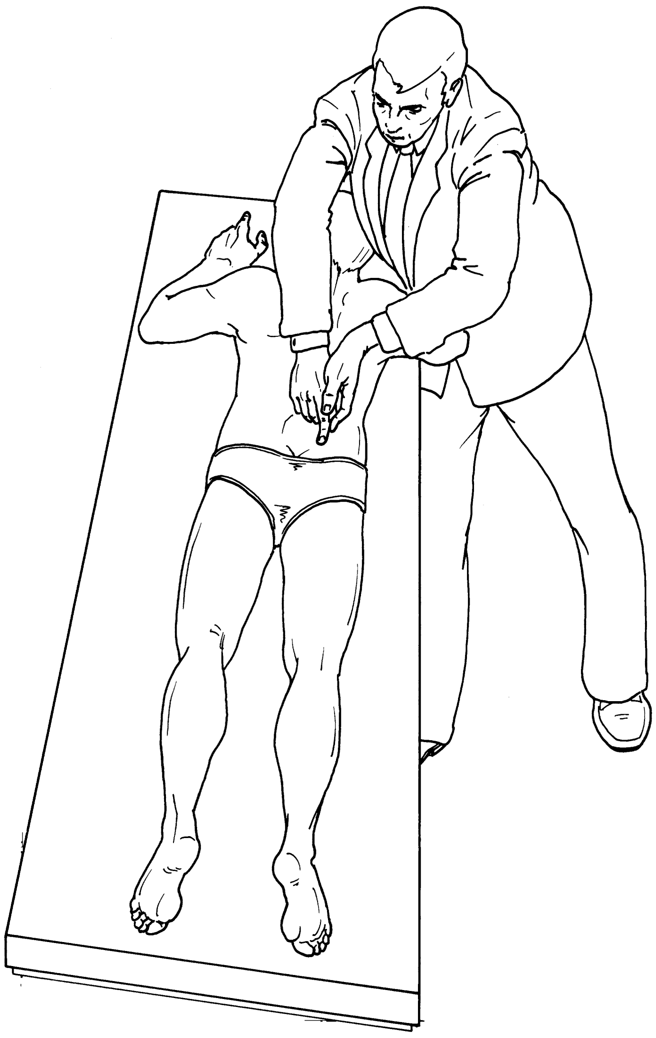


Fig. 1.57 Springing test 2.

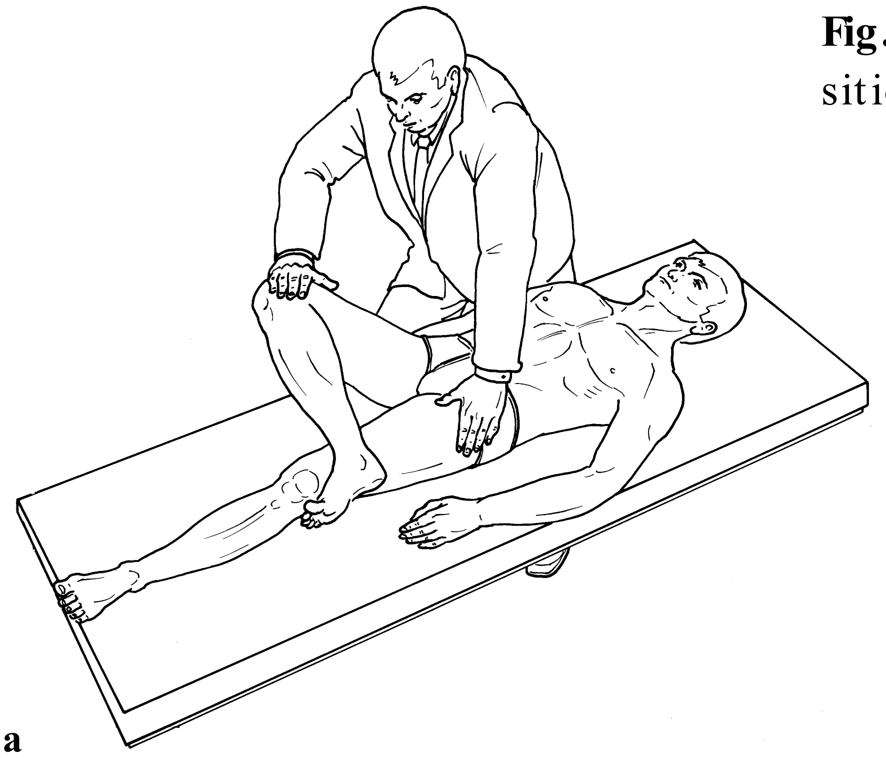
□ **Assessment:** A normal sacroiliac joint will be resilient: palpating pressure will slightly increase the distance between the posterior margin of the ilium and the sacrum. This resiliency is not present in a motion-restricted sacroiliac joint. A relatively long range of motion with a hard end point suggests hypermobility in the sacroiliac joint. Pain during the examination can occur in both a motion-restricted and a strained hypermobile joint (painful hypermobility).

Patrick Test (Fabere Sign)

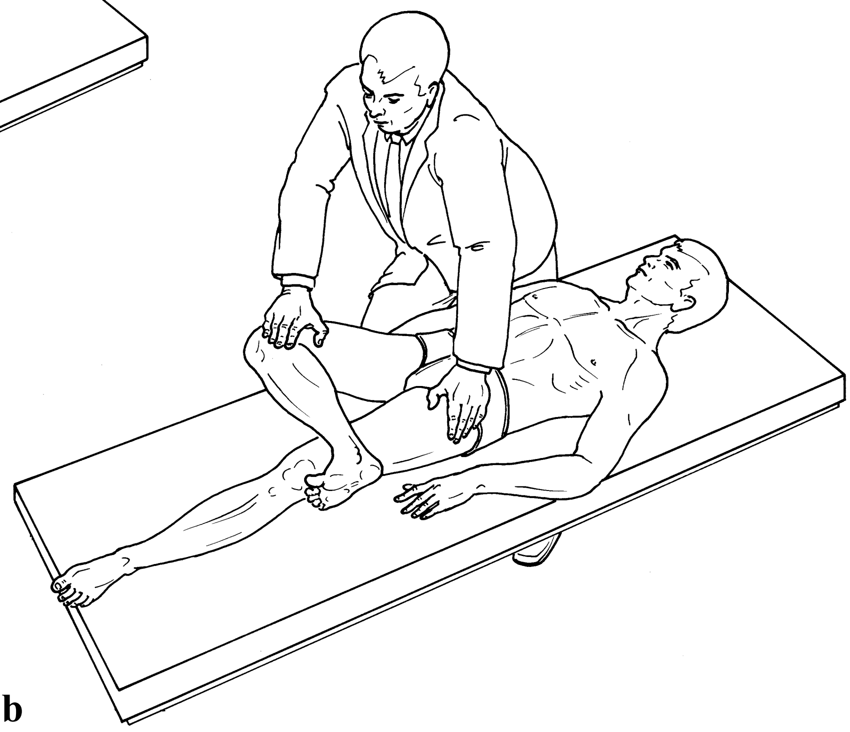
Differentiates hip disorders from disorders of the sacroiliac joints (assessment of adductor tension).

□ **Procedure:** The patient is supine with one leg extended and the other flexed at the knee. The lateral malleolus of the flexed leg lies across the other leg superior to the patella.

The test may also be performed so that the foot of the flexed leg is in contact with the medial aspect of the knee of the contralateral leg. The flexed leg is then allowed to fall into abduction, and from this position the examiner increases the external rotation by increasingly pressing the patient's knee down toward the examining table with one hand. The examiner must immobilize the pelvis on the extended contralateral side to prevent it from moving during the test.



a



b

Fig. 1.58 Patrick test. **(a)** Starting position. **(b)** With pressure applied.

□ **Assessment:** Normally the knee of the abducted leg will almost touch the examining table. Comparative measurements of the distance between the knee and the table on both sides are made. A difference in mobility with painfully restricted motion in hyperabduction suggests the absence of a hip disorder; observation of normal adductors suggests dysfunction in the ipsilateral sacroiliac joint. Hip disorders are excluded by testing range of motion in the hip (especially rotation) and palpating the hip capsule deep in the groin.

Three-Phase Hyperextension Test

□ **Procedure:** The patient is prone. In the first phase of the test, the examiner grasps the patient's extended leg and raises it into hyperextension while immobilizing the pelvis with the other hand.

In the second phase, the examiner immobilizes the patient's sacrum parallel to the sacroiliac joint with the same hand and passively raises the patient's leg into hyperextension. In the third phase, the examiner immobilizes the fifth lumbar vertebra with the heel of one hand while passively guiding the patient's leg into hyperextension with the other hand. By moving the immobilizing hand up the spine, the examiner can also evaluate higher segments of the lumbar spine.

□ **Assessment:** Under normal conditions no pain should occur in any phase of the test. The hip should allow about 10 to 20° of hyperextension. The sacroiliac joint should exhibit slight movement (joint play), and the lumbar spine should allow elastic hyperextension (lordosis) at the lumbosacral junction.

Pain with the ilium immobilized (phase 1) suggests a hip disorder or muscle contracture (rectus femoris and/or psoas). Pain when the sacrum is immobilized suggests motion restriction of the sacroiliac joint or other disorders of this joint, such as ankylosing spondylitis (phase 2, Mennell sign), while pain when the lumbar spine is immobilized suggests a disorder of the lumbosacral junction (vertebral motion restriction or protrusion or extrusion of an intervertebral disk) (phase 3).

□ **Note:** The test for a Mennell sign is identical to the second phase of the three-phase hyperextension test.

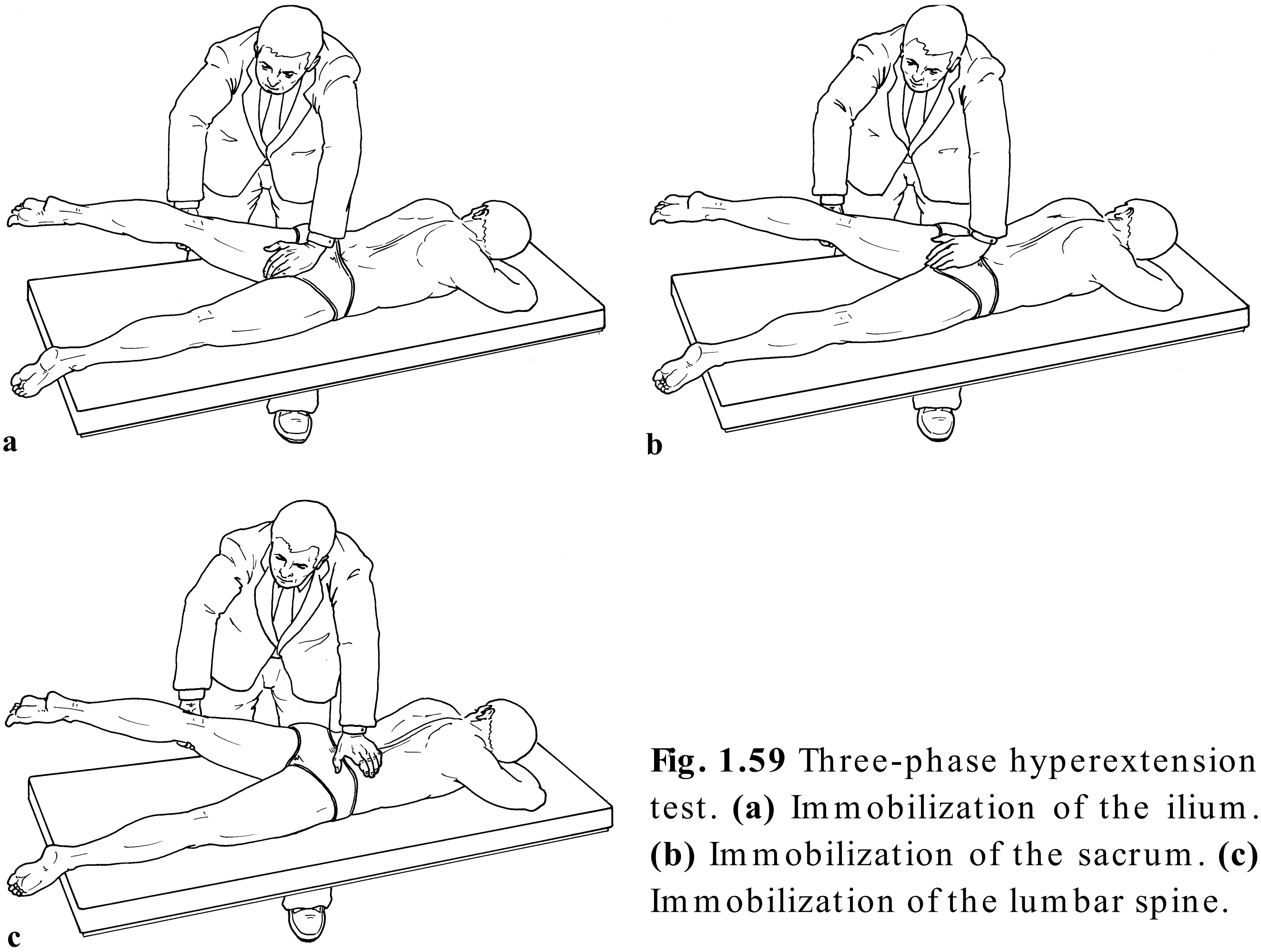


Fig. 1.59 Three-phase hyperextension test. **(a)** Immobilization of the ilium. **(b)** Immobilization of the sacrum. **(c)** Immobilization of the lumbar spine.

Spine Test

Assesses sacroiliac joint function.

□ **Procedure:** The examiner stands behind the standing patient and palpates the posterior superior iliac spine and the median sacral crest (spinous processes of the fused sacral vertebrae) at the same level. The patient is asked to raise the ipsilateral leg and push his or her knee as far forward as possible.

□ **Assessment:** If the sacroiliac joint is not motion-restricted, the ilium will move downward on the side being examined. The posterior superior iliac spine will be seen to shift inferiorly about 0.5 cm or up to at most 2 cm with the movement. This downward shift will not occur if the sacroiliac joint is motion-restricted; in fact, the motion restriction will usually cause the posterior superior iliac spine to move upward (superiorly) as the pelvis tilts in compensation.

Standing Flexion Test

Assesses sacroiliac joint function.

□ **Procedure:** The patient stands with his or her back to the examiner. The examiner's thumbs simultaneously palpate both posterior superior iliac spines. The patient is asked to slowly bend over while keeping both feet in contact with the floor and the knees extended. The examiner observes the position and/or motion of both iliac spines as the patient's torso bends forward.

□ **Assessment:** The sacrum rotates relative to the ilia around a horizontal axis in the sacroiliac joints. This motion is referred to as "nutation."

In normal patients with mobile sacroiliac joints, the two posterior superior iliac spines will be level with each other throughout the range of motion when the patient bends over.

If nutation does not occur in the sacroiliac joint on one side, the posterior superior iliac spine on that side will come to rest farther superior with respect to the sacrum than the spine on the contralateral side.

Where nutation fails to occur or this relative superior advancement is observed, this is usually a sign of a blockade in the ipsilateral sacroiliac joint. Bilateral superior advancement can be simulated by bilateral shortening of the hamstrings.

□ **Note:** When evaluating this superior advancement phenomenon, the examiner must consider or exclude possible asymmetry of the pelvis and hips. Pelvic obliquity due to a difference in leg length should be compensated for by placing shims under the shorter leg.

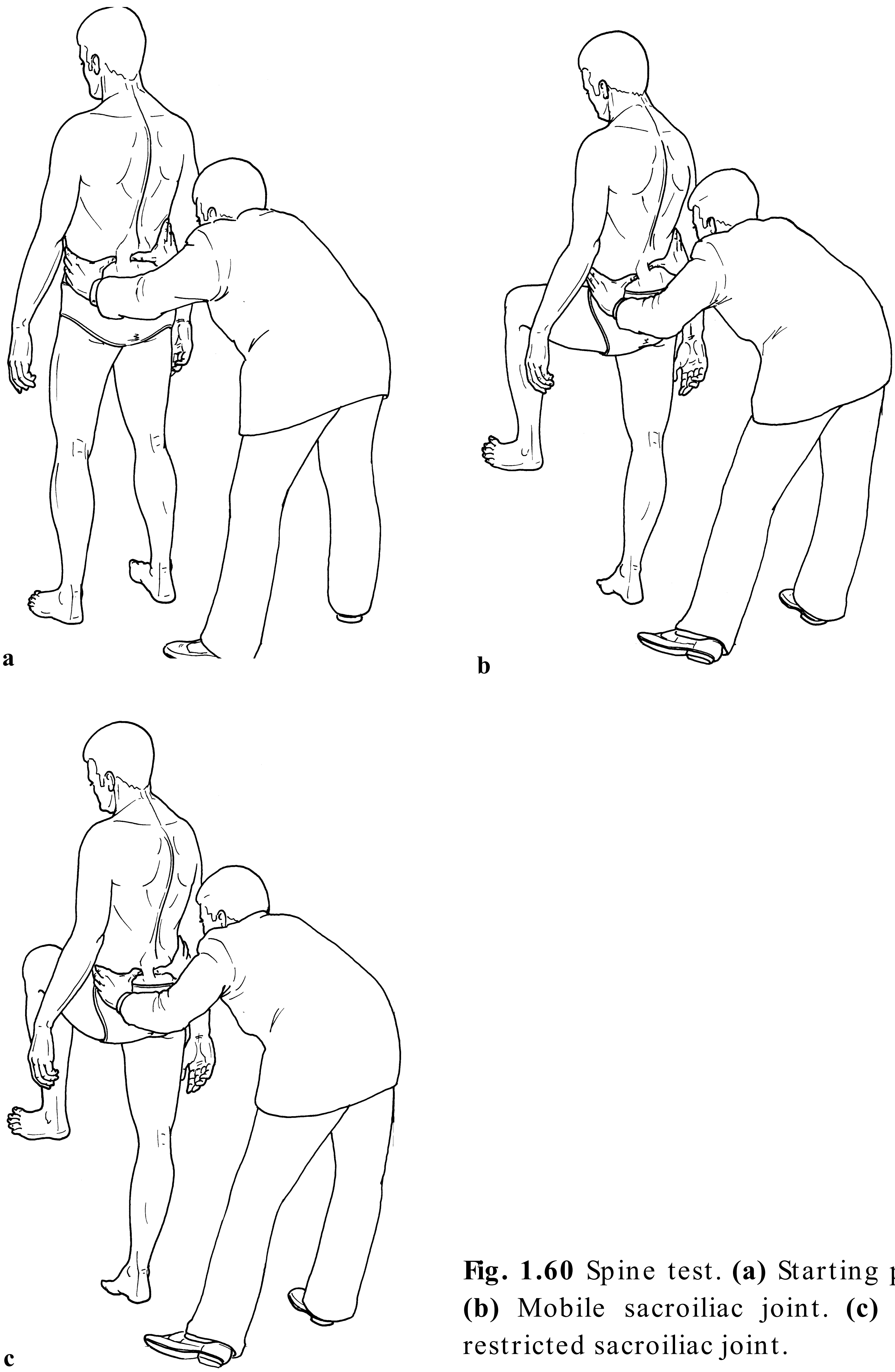


Fig. 1.60 Spine test. **(a)** Starting position. **(b)** Mobile sacroiliac joint. **(c)** Motion-restricted sacroiliac joint.

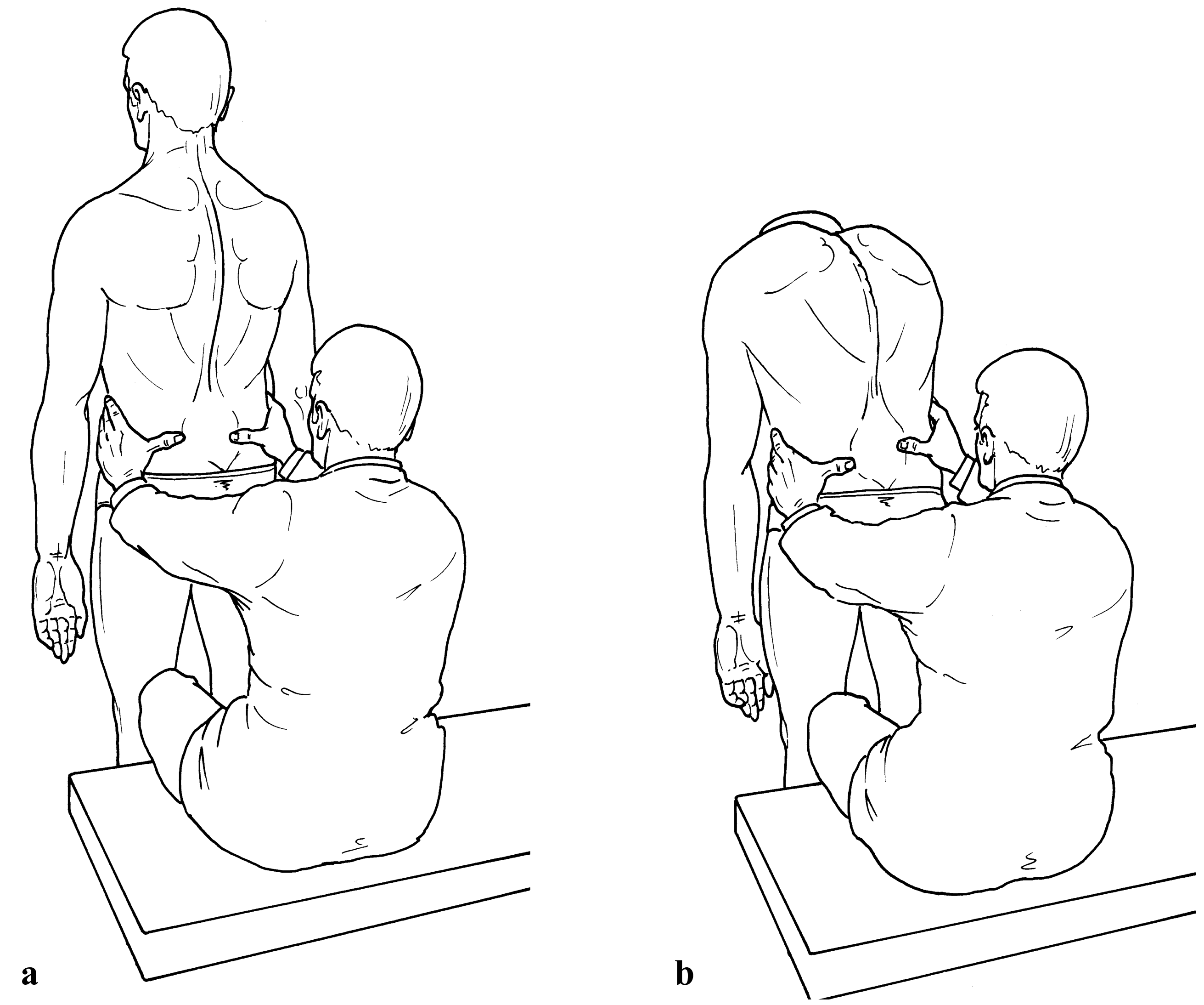


Fig. 1.61 Standing flexion test. **(a)** Starting position. **(b)** Motion-restricted right sacroiliac joint.

Sacroiliac Joint Springing Test 2

□ **Procedure:** To directly test the play in the sacroiliac joint, the patient is placed supine. The leg opposite the examiner is flexed at the knee and hip and adducted toward the examiner until the pelvis begins to follow. The other leg remains extended. Next, the examiner grasps the knee of the adducted leg and palpates the sacroiliac joint with the other hand while exerting resilient axial pressure on the knee.

□ **Assessment:** This maneuver normally produces a springy motion in the sacroiliac joint, which will be palpable as movement between the posterior iliac spine and the sacrum. Lack of joint play is typical of a functional impairment. This springing test is based on the knowledge that the range of motion in an intact joint can be increased by resilient pressure even with the joint at the extreme end of its range of motion. This essentially allows the diagnosis of a functional impairment in any joint by manual manipulation. However, the important thing is to perform the test with initial stress already applied to the joint. This test is recommended to supplement the prone springing tests.

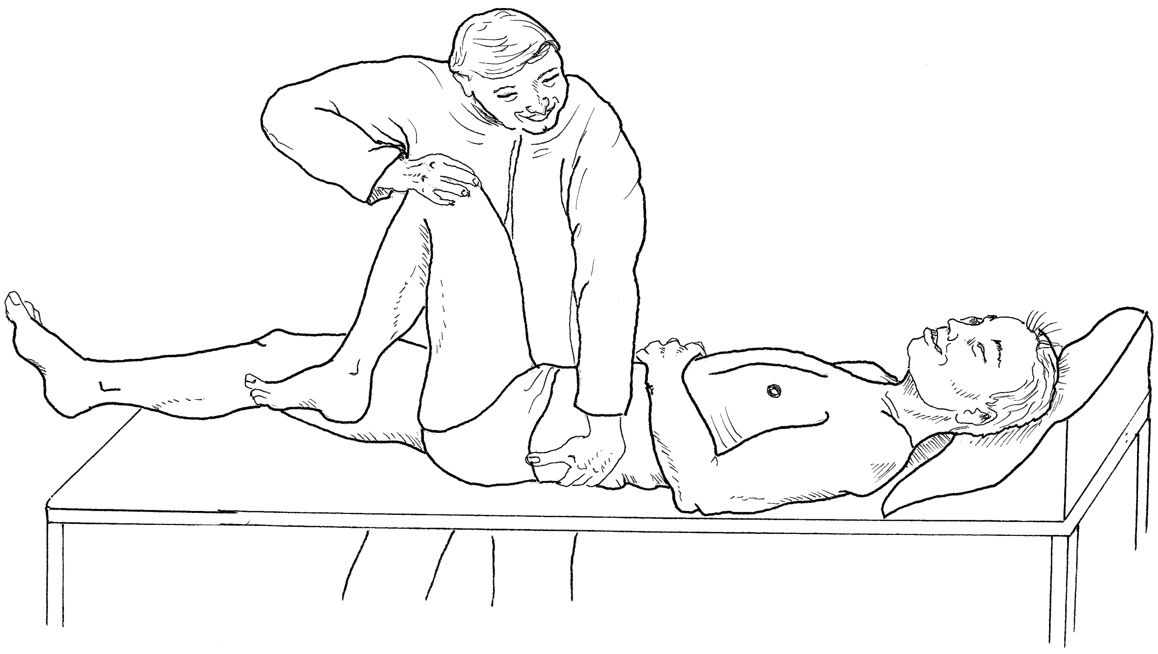


Fig. 1.62 Springing test of the sacroiliac joint.

Sacroiliac Mobilization Test

Assesses sacroiliac joint function.

□ **Procedure:** The patient is prone. The examiner places the fingers of the palpating hand over the sacroiliac joints, i.e., over the posterior sacral ligaments (the sacroiliac joint itself is not accessible to palpation because of its anatomical position). The examiner places the other hand around the anterior iliac wing. With this hand, the examiner performs small shaking and lifting motions in a posterior direction (moving the ilium posteriorly relative to the sacrum).

□ **Assessment:** The palpating fingers over the sacroiliac joint will detect resilient motion in a normal joint, or painfully limited resiliency in the presence of a blockade.

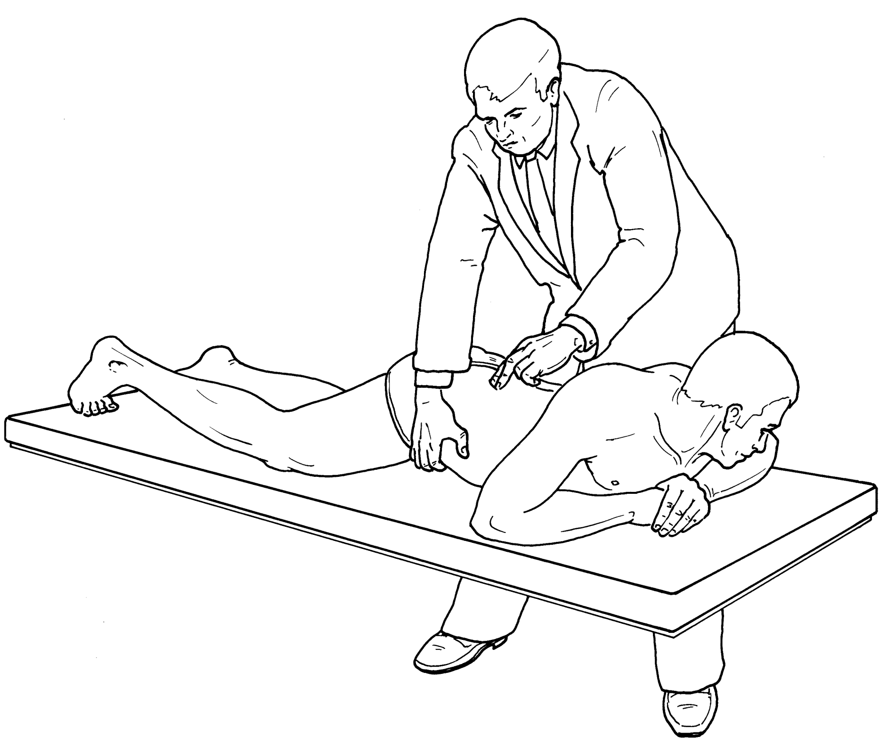


Fig. 1.63 Sacroiliac mobilization test.

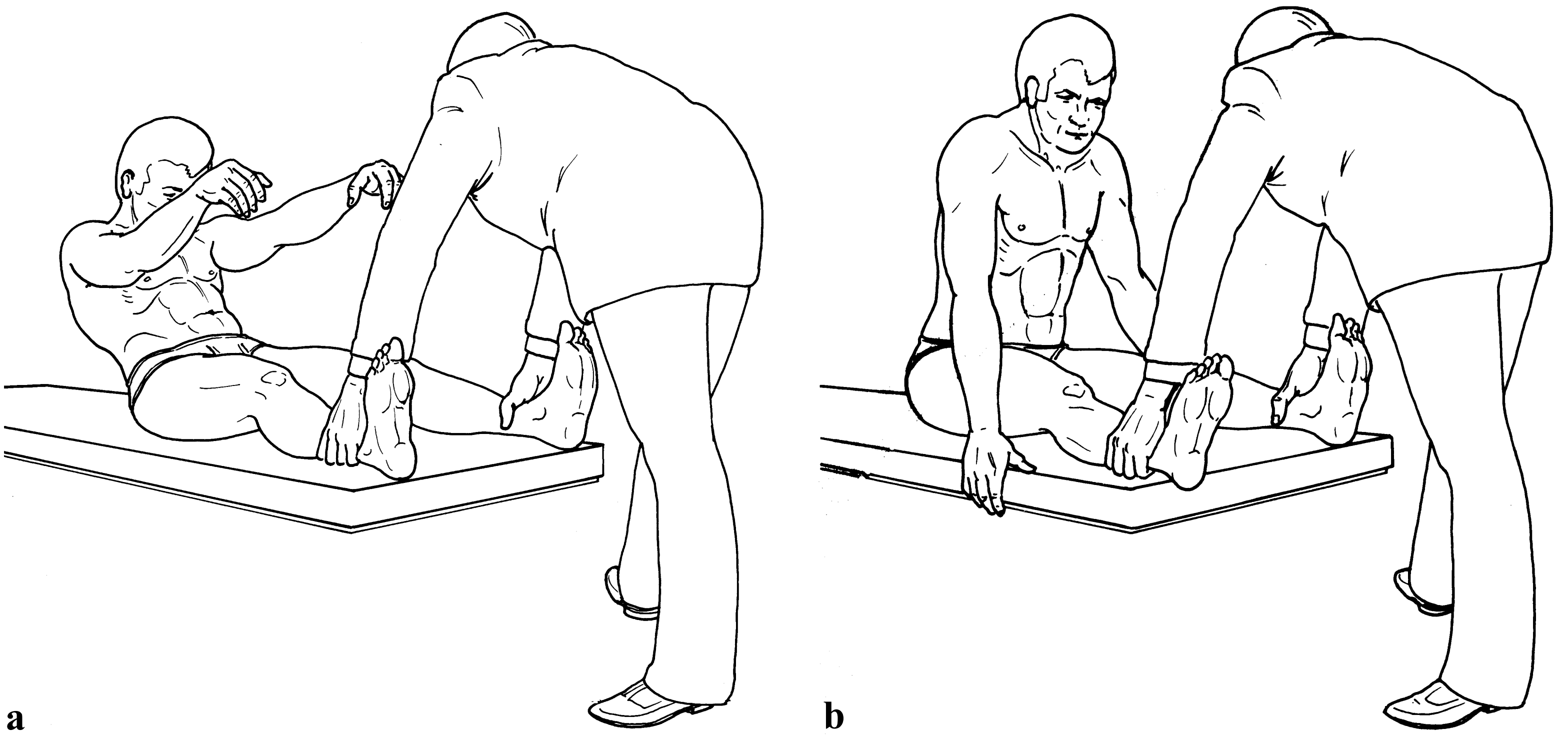


Fig. 1.64 Derbolowsky sign. **(a)** Mobile sacroiliac joint. **(b)** Motion-restricted right sacroiliac joint (causes leg lengthening when the patient sits up).

Derbolowsky Sign

Assesses a leg length difference: an advancement phenomenon with the patient supine.

□ **Procedure:** The patient is supine. The examiner grasps both ankles, palpates the patient's medial malleoli with each thumb, and evaluates the relative level and rotation of the medial malleoli using the positons of the thumbs as reference.

The patient is asked to sit up; either the examiner may help the patient do so, or the patient may use his or her hands for support. The legs should be lifted off the table to prevent interference. Then the level and rotation position of the malleoli are again evaluated. Lastly, the patient is asked to bend the trunk maximally forward to come as close to the extended knees as possible. The test should be carried out several times to prevent false-positive test results due to muscle tension.

□ **Assessment:** Forward advancement in the supine positon suggests pelvic rotation. Where there is a motion restriction in the sacroiliac joint without any play between the sacrum and ilium, the ipsilateral leg will be longer when the patient sits up and apparently shorter or the same length as the other leg when the patient is supine. The examiner measures the difference in the level of the two malleoli, which previously were at the same level.

The differential diagnosis should consider whether something other than a motion restriction in the sacroiliac joint may be causing the variable leg length difference. Possible such causes include shortening of the hamstrings or genuine anatomical leg lengthening or shortening. Pain during the test could suggest loosening of the sacral structure, muscular foreshortening, or neurologic pain from a protruding or herniated disk.

Note: This test is considered to be diagnostically significant if the difference in levels amounts to at least 1 to 2 cm. When one sees larger differences in connection with myalgic pain, then one should consider shortened hamstring muscles as a cause. If the difference is greater than 5 cm combined with symptoms of radicular pain and pelvic rotation with compensatory flexion of the knee, then one should consider vertebral disk dysfunction.

Gaenslen Sign (Second Mennell Sign)

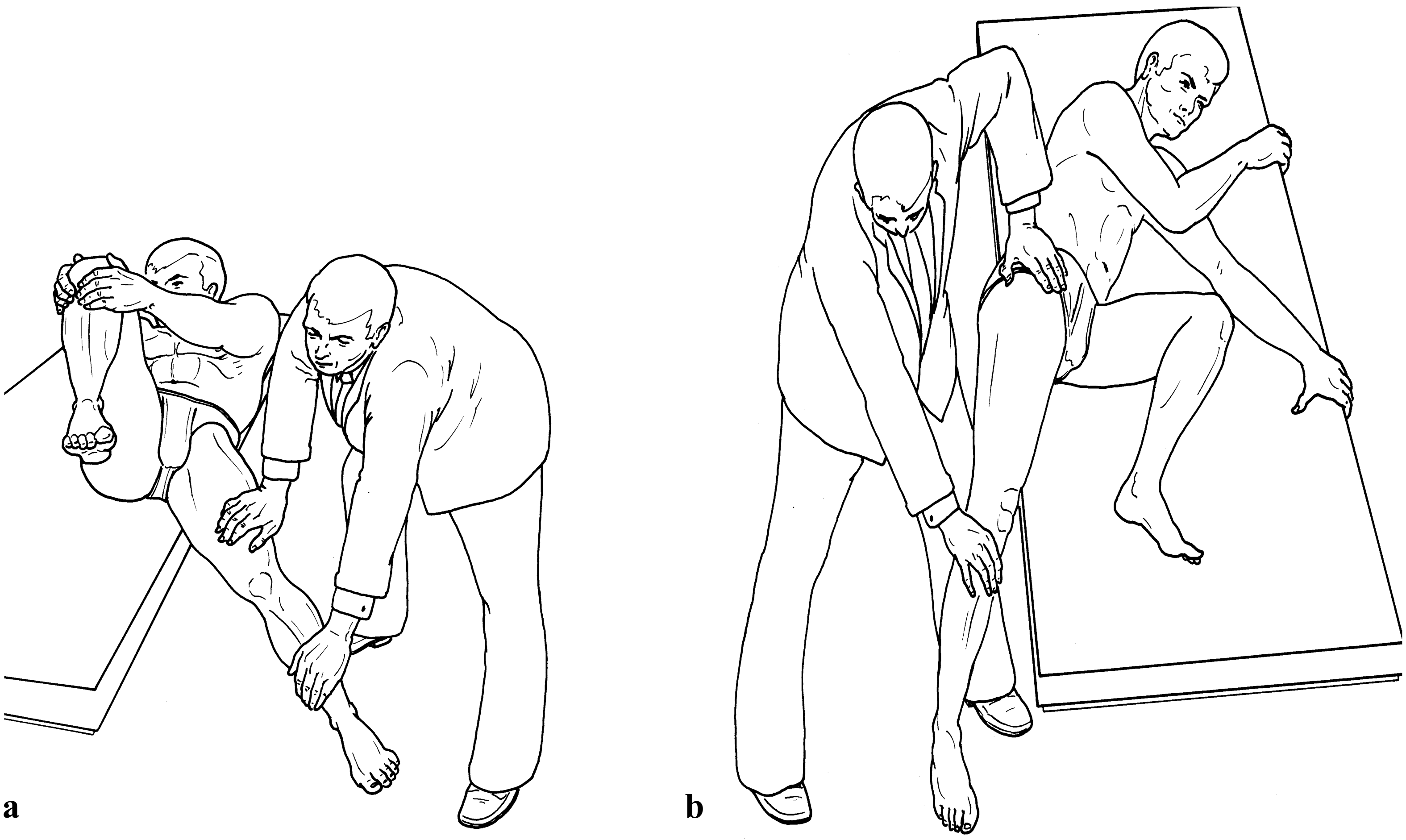
Assesses sacroiliac joint function.

Procedure: The patient is supine with the painful side as close as possible to the edge of the examining table or projecting beyond it. To stabilize this position and immobilize the lumbar spine, the patient flexes the knee and hip of the contralateral leg and draws the leg as close to the torso as possible (Thomas grip). The examiner then passively hyperextends the leg next to the edge of the examining table.

The test may also be performed with the patient in a lateral position. This is done with the patient lying on his or her normal side with that leg flexed at the hip and knee. The examiner then passively hyperextends the other leg (the one not in contact with the table).

Assessment: If there is dysfunction in the sacroiliac joint, hyperextension of the leg will lead to motion in the sacroiliac joint, causing pain or exacerbation of existing pain.

Pain may also be caused by hip pathology or an ipsilateral nerve root lesion.



a b

Fig. 1.65 Gaenslen sign. **(a)** Supine. **(b)** Lateral position.

Iliac Compression Test

Indicates sacroiliac disease.

□ **Procedure:** The patient assumes a lateral position. The examiner places both hands on the ilium of the affected side and exerts downward pressure on the pelvis.

□ **Assessment:** Occurrence of or an increase in pain in the sacroiliac joint adjacent to the examiner's hand suggests a joint disorder (such as motion restriction or inflammation).

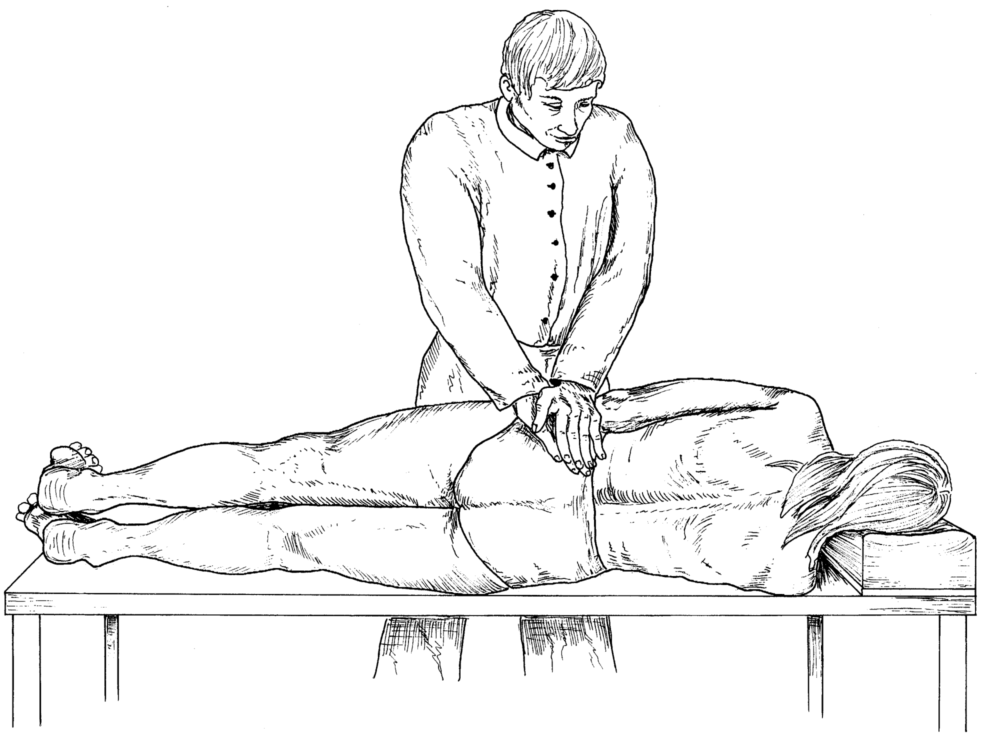


Fig. 1.66 Iliac compression test.

Mennell Sign

Indicates sacroiliac disease.

□ **Procedure:** The patient is prone. When examining the left sacroiliac joint, the examiner immobilizes the patient's sacrum with the left hand while grasping the patient's extended left leg with the right hand and suddenly hyperextending the hip.

The examination may also be performed with the patient in a lateral position. This is done with patient lying on his or her right side and immobilizing that leg, flexed at the hip and knee, with both hands. The examiner stands behind the patient, holding the patient's pelvis with his or her right hand, and then suddenly hyperextends the patient's left hip with the left hand.

□ **Assessment:** Pain in the sacroiliac joint suggests a joint disorder (such as motion restriction or inflammation).

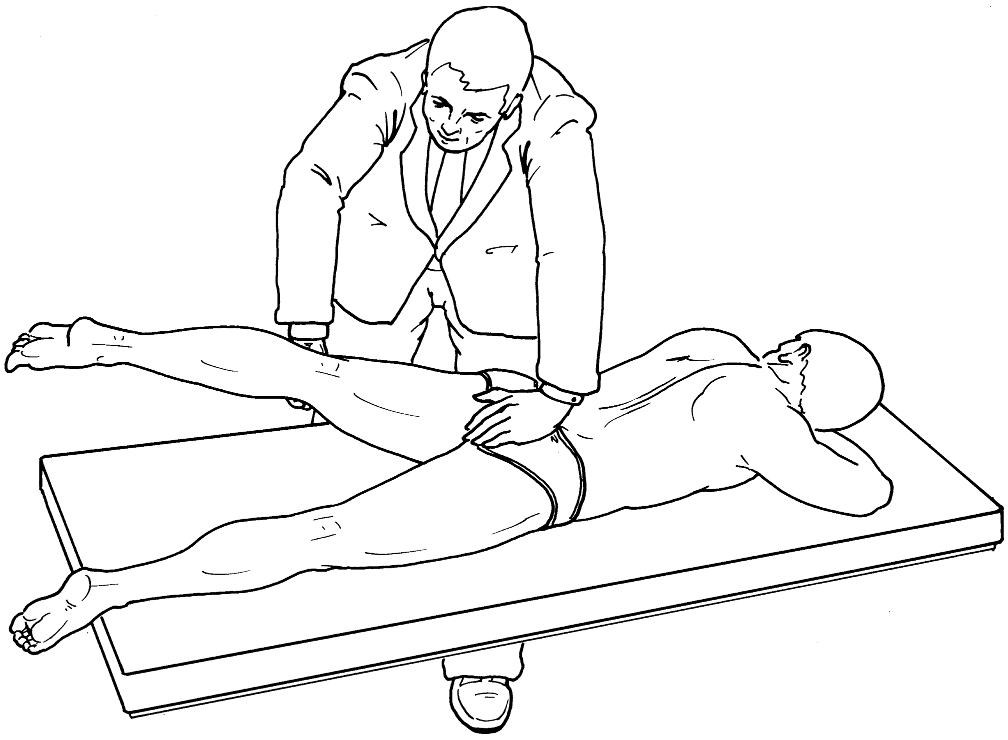


Fig. 1.67 Mennell sign.

Yeoman Test

Assesses sacroiliac pain.

□ **Procedure:** The patient is prone with the knee flexed 90°. The examiner raises the flexed leg off the examining table, hyperextending the hip.

□ **Assessment:** The first part of this test initially places stress on the posterior structures of the sacroiliac joint; later the stress shifts to the anterior portions, primarily affecting the anterior sacroiliac ligaments. Pain in the lumbar spine suggests the presence of pathologic processes at that site. Anterior thigh paresthesia may indicate a femoral nerve stretch.

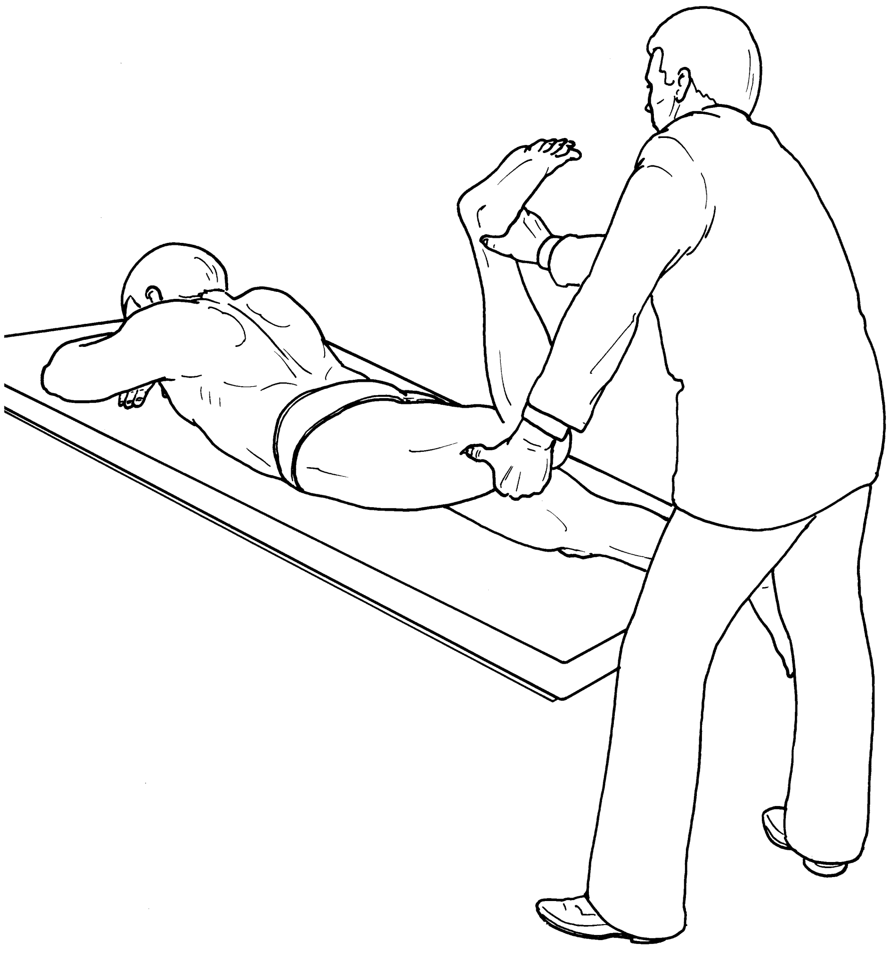


Fig. 1.68 Yeoman test.

Fig. 1.69 Laguerre test.

Laguerre Test

Differentiates hip pain from sacroiliac pain.

□ **Procedure:** The patient is supine. The examiner passively flexes the patient's hip and knee 90°. Then the hip is passively abducted and placed in extreme external rotation.

□ **Assessment:** This maneuver moves the femoral head into the anterior part of the joint capsule of the hip. Pain within the hip suggests degenerative joint disease, hip dysplasia, or contracture of the iliopsoas. Pain felt posteriorly in the sacroiliac joint suggests a disease process at that site.

Sacroiliac Stress Test

Demonstrates involvement of the anterior sacroiliac ligaments in a sacroiliac joint syndrome.

□ **Procedure:** The patient is supine. The examiner exerts anterior pressure on the iliac wings with both hands. By crossing his or her hands, the examiner adds a lateral force vector to the compression. The anteroposterior direction of the compressive load on the pelvis places stress on the posterior portions of the sacroiliac joint, whereas the lateral component places stress on the anterior sacroiliac ligaments.

□ **Assessment:** Deep pain is a sign of strained anterior sacroiliac ligaments on the side of the pain (sacrospinous and sacrotuberous ligaments). Pain in the buttocks can be produced by compression from the examining table or by irritation of the posterior portions of the sacroiliac joint. Determining the precise location of the pain helps to identify its cause.

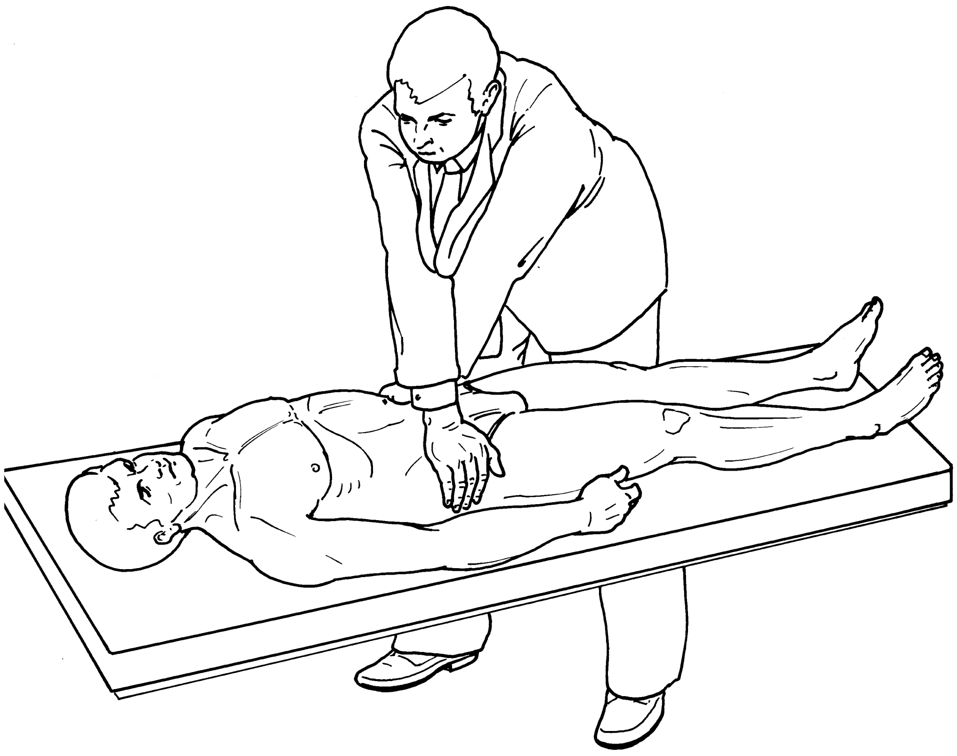


Fig. 1.70 Sacroiliac stress test.

Abduction Stress Test

Indicates a sacroiliac joint syndrome.

□ **Procedure:** The patient is in a lateral position. With the leg that is in contact with the table flexed, the patient attempts to continue to abduct the upper extended leg against the examiner's resistance. This test is normally performed to evaluate insufficiency of the gluteus medius and gluteus minimus muscles.

□ **Assessment:** Increasing pain in the affected sacroiliac joint is a sign of sacroiliac irritation. Patients with hip disorders may also feel increased pain when this test is performed. The location of the pain is suggestive of the type of disorder. A leg that can only be abducted slightly or not at all without causing pain suggests insufficiency of the gluteus minimus.

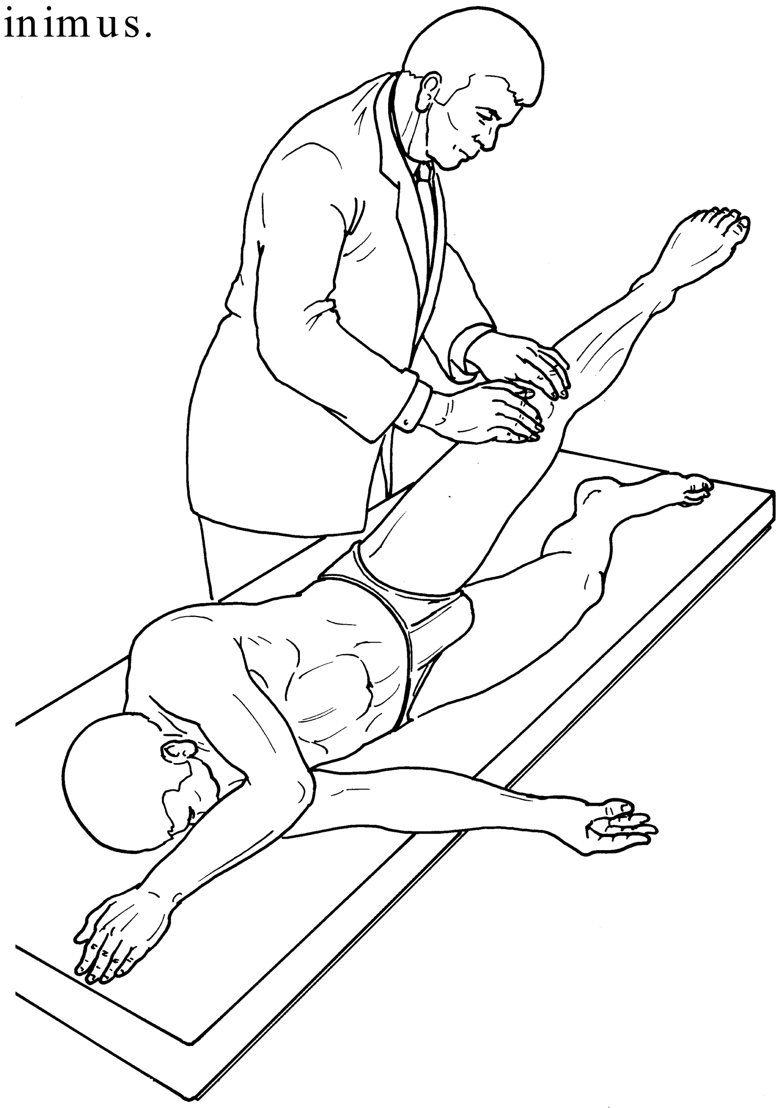


Fig. 1.71 Abduction stress test.

2 Shoulder

The complex anatomy and biomechanics of the shoulder enable what is, for the human body, a very large range of movement. However, this also opens the door to numerous pathologic processes and injuries.

Shoulder complaints are a frequent problem. Contributing factors, among others, include demographic developments with an increase in age-related degenerative processes and injuries from chronic overuse in recreational and competitive activities. Years of occupational, recreational, or household activities involving overhead work lead to excessive stresses and muscle imbalance, as does sitting at an ergonomically unfavorable workplace.

The examiner should use a working outline that he or she has individually designed and structured in performing the clinical diagnostic evaluation of the shoulder. Here, as in any clinical examination, the first step in examining the shoulder is to obtain a thorough history. The many different shoulder disorders may have their causes in acute trauma, local processes due to chronic overuse, age-related degeneration, or systemic disease. In adolescence and early adulthood, shoulder disorders are primarily attributable to trauma or congenital deformities; the most common of these include dislocations and subluxations and their resulting instabilities. With advancing years, degenerative disorders become more prominent; these include impingement syndrome, ruptures of the rotator cuff, and degenerative acromioclavicular joint changes.

Inquiring about occupational stresses and athletic activities provides important information. Jobs involving a lot of overhead work (painting) and sports with similar requirements (basketball, baseball, tennis, swimming, volleyball) often lead to early disorders in the subacromial and intra-articular spaces. These are accompanied by degenerative changes in the acromioclavicular joint. Obtaining a detailed history from an athlete requires knowledge of the motion sequences specific to his or her particular sport. This is crucial to diagnosing patterns of injury specific to that sport.

However, acute symptoms are not always attributable to obvious trauma from an identifiable mechanism of injury. In the presence of preexisting tendon degeneration, a minor injury can lead to rupture of the rotator cuff.

In addition to specific questions about shoulder disorders, the examiner must always be alert to the possibility of diseases of other organ systems. Pain from angina pectoris often radiates into the shoulder and arm, and this referred pain does not invariably occur on the left side. Gallbladder or liver disorders can also cause pain in the right shoulder. Rheumatic polyarthritis and hyperuricemia can manifest first in the shoulder. Patients with diabetes mellitus very often have an associated shoulder affliction that tends to restrict motion in the shoulder.

Observing the patient provides the examiner with an initial overview. Gait and any compensatory contralateral motion of the upper extremities are noted. A patient with a frozen shoulder avoids internal or external rotation and motion above horizontal when undressing. Patients with a ruptured rotator cuff will often ask for help undressing because they lack the strength to abduct the arm. Asymmetry (especially muscle atrophy) is best revealed by comparison with the contralateral side. In comparative inspection of both acromioclavicular joints, the examiner looks for swelling or a step-off resulting from an acromioclavicular joint separation. Distal displacement of the belly of the biceps brachii muscle suggests rupture of the long head of the biceps tendon. Side-to-side asymmetry of the shoulder contour can indicate scapulothoracic imbalance.

After inspection, it is recommended that the examiner inquire about the patient's neurologic orientation and test for it. To determine the vertebral origin of a particular pain, such as in a nerve or nerve root compression syndrome, the examiner should always begin the evaluation of the shoulder with an orientation examination of the cervical spine. The examiner must conduct a thorough neurological examination appropriate to each clinical suspicion. Specific provocation maneuvers such as the Adson test (p.345) and the Gagey's hyperabduction test (p.341) are required to rule out neurovascular compression syndromes.

In terms of palpation, one should test the sternoclavicular joint, clavicle, acromioclavicular joint, coracoid process, intertubercular sulcus, and the greater and lesser tubercles for tenderness. Test the active and passive ROM for flexion/extension, abduction/adduction, and external/internal rotation with the shoulder at 0 and 90° of abduction. Compare left and right sides using the neutral-zero method.

Only after a thorough clinical examination can one form the correct diagnostic indication for possible follow-up diagnostic imaging or injection tests.

Anteroposterior and axial radiographs and special shoulder views are indicated to supplement the clinical examination. These can differentiate bony changes from soft tissue pathology. Ultrasound, MRI, and CT may also be useful in visualizing shoulder disorders.

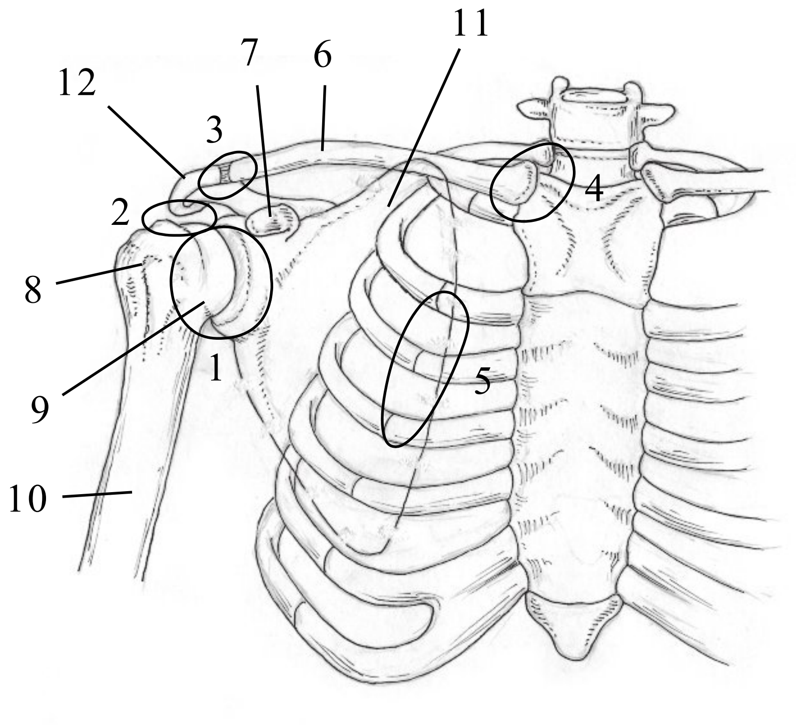


Fig. 2.1 The five regions of the shoulder.
1 Glenohumeral joint.
2 Subacromial "joint" (space between the coracoacromial arch and the head of the humerus including the greater and lesser tubercle and the subacromial bursa).
3 Acromioclavicular joint.
4 Sternoclavicular joint.
5 Scapulothoracic gliding "joint" (scapula glides on the posterior thoracic wall).
6 Clavicle.
7 Coracoid process.
8 Greater tubercle.
9 Head of the humerus.
10 Humerus.
11 Scapula.
12 Acromion.

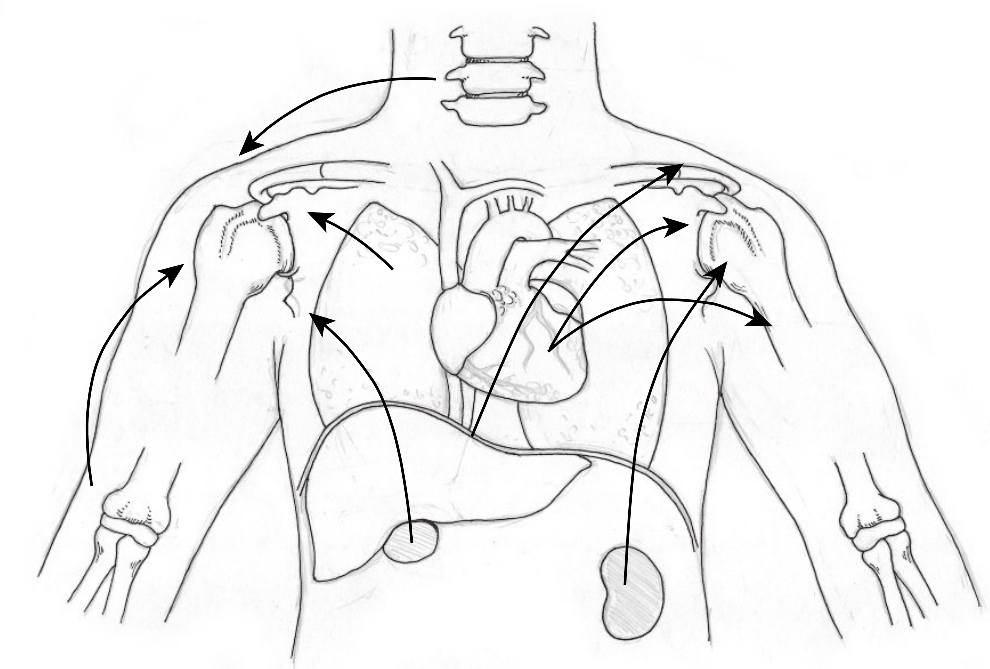


Fig. 2.2 Pain projecting from internal organs and other joints into the shoulder

Range of Motion of the Shoulder (Neutral-Zero Method)

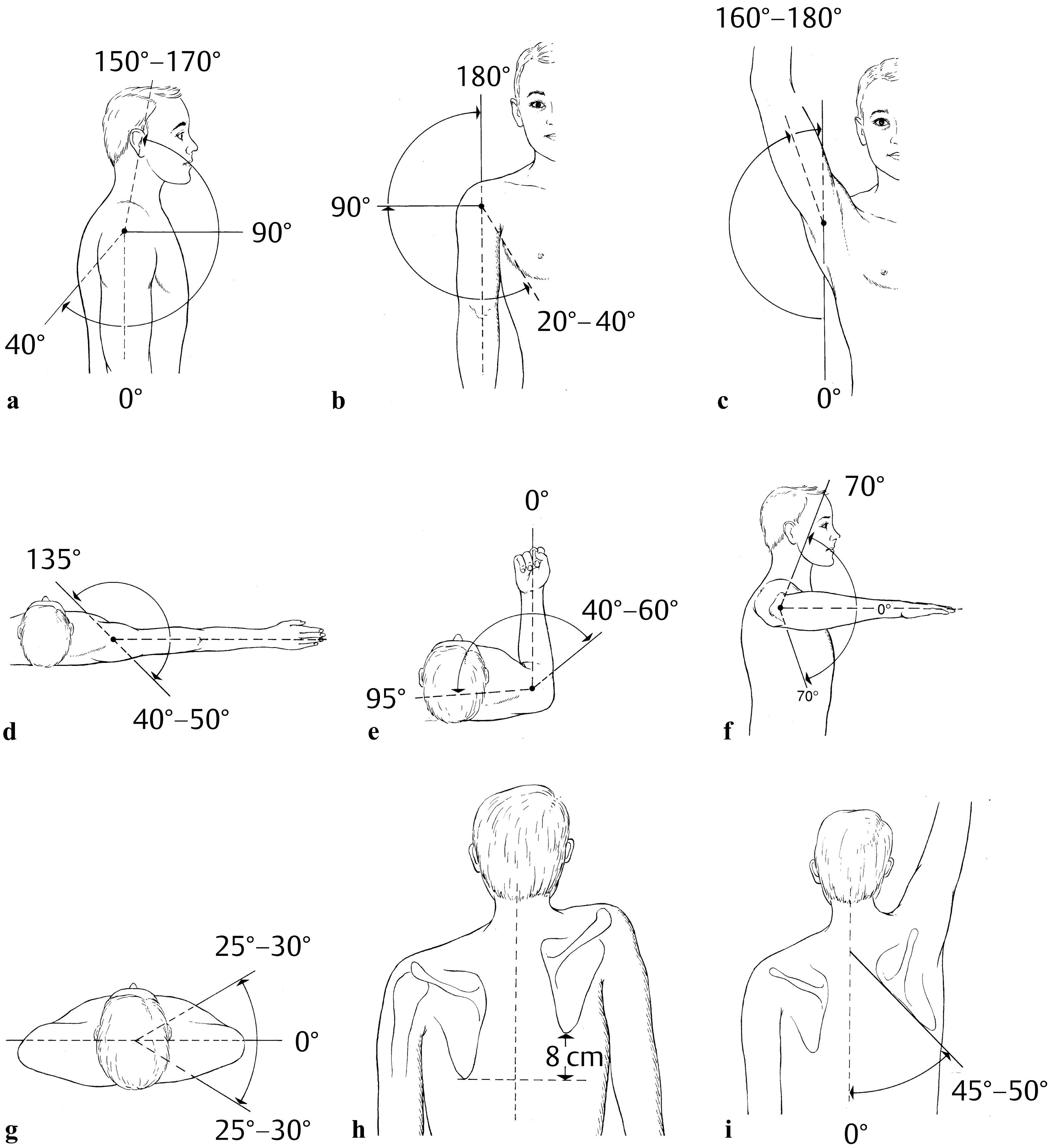


Fig. 2.3 (a) Forward flexion and extension. **(b)** Abduction and adduction. **(c)** Abduction exceeding 90° requires external rotation of the humerus in the glenohumeral joint and rotation of the scapula. **(d)** Horizontal flexion and extension (forward and backward motion of the arm, abducted 90° from the body). **(e, f)** External and internal rotation: with the arm hanging down **(e)** and abducted 90° **(f)**. **(g)** Protraction and retraction of the shoulder. **(h, i)** Scapular elevation and depression **(h)**; scapular rotation relative to the trunk **(i)**.

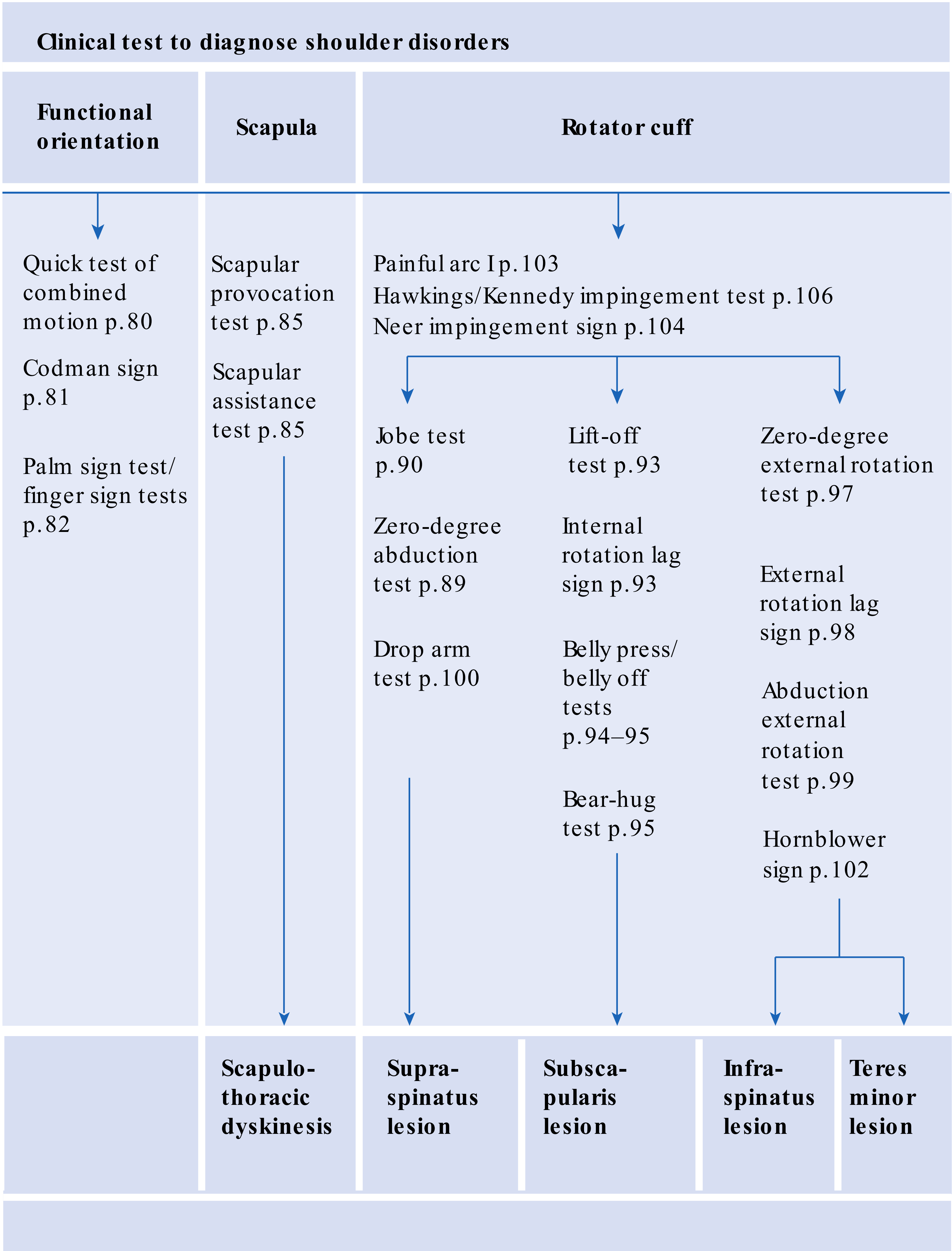
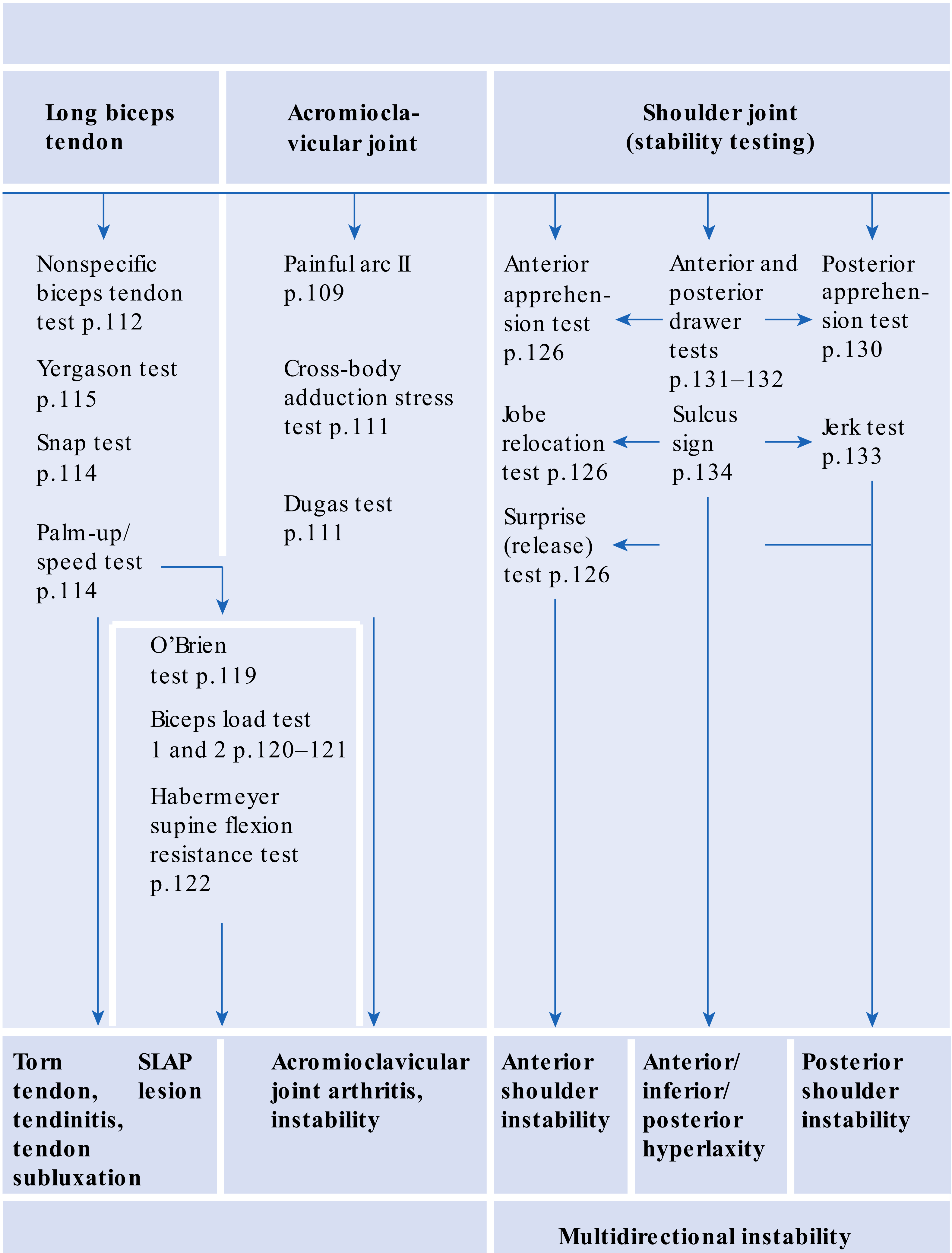


Fig. 2.4 Clinical tests to diagnose shoulder disorders.



SLAP, superior labral anterior and posterior.

Orientation Tests

Quick Test of Combined Motion

□ **Procedure:** A quick test of mobility in the shoulder is to ask the patient to place a hand behind his or her head and touch the contralateral scapula (Apley Scratch Test, see p. 102). In a second movement the patient places the hand behind his or her back, reaching upward from the buttocks to touch the inferior margin of the scapula.

□ **Assessment:** Mobility on one side that is restricted in comparison with the contralateral side is a sign that there is a shoulder disorder. Other tests may then be used to diagnose this disorder in greater detail. Pain over the shoulder usually indicates tendinitis of one of the tendons of the rotator cuff—usually the supraspinatus tendon—adhesive capsulitis, or subacromial bursitis.

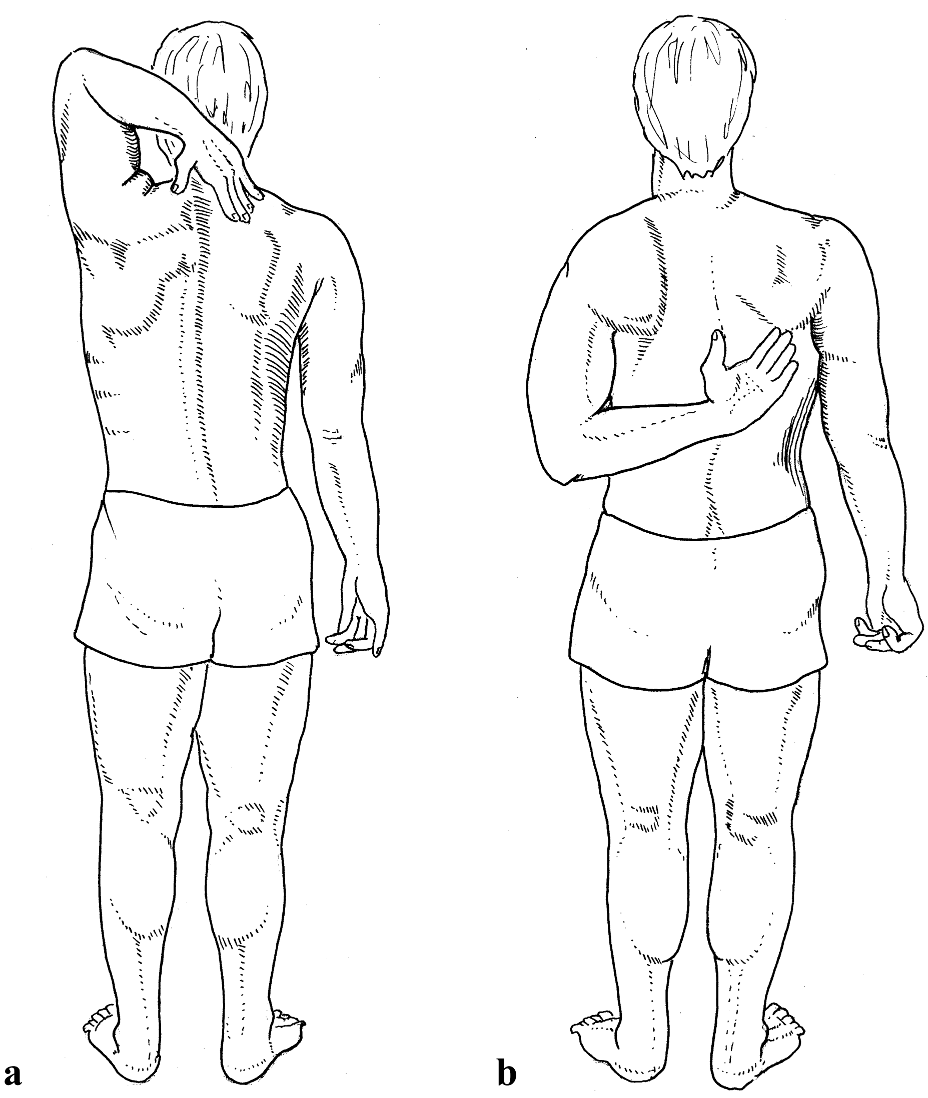


Fig. 2.5 Quick test of combined motion. **(a)** Touching the scapula from behind the neck. **(b)** Touching the scapula from behind the back.

Codman Sign

Tests passive motion in the shoulder.

▫ **Procedure:** The examiner stands behind the patient and places his or her hand on the patient's shoulder so that the thumb immobilizes the patient's scapula slightly below the scapular spine, the index finger rests on the anterior margin of the acromion toward the tip of the coracoid, and the remaining fingers extend anteriorly past the acromion.

The examiner then moves the patient's arm in every direction using the other hand.

▫ **Assessment:** The examiner notes any crepitation in the glenohumeral joint, snapping phenomena (such as dislocations of the long head of the biceps tendon), or restricted motion.

The most important bony pressure points, such as the greater and lesser tubercles of the humerus, coracoid process, and sternoclavicular and acromioclavicular joints, are assessed for tenderness to palpation. Joint stability is also assessed, and pain in the tendons of the rotator cuff is evaluated by palpation.

The range of motion is determined using the neutral-zero method. The active and passive ranges of motion are determined, as are the region of occurrence and specific localization of symptoms. Restricted motion in every direction indicates the presence of a "frozen shoulder."

In the early stages of a rotator cuff tear, only active motion is restricted; passive motion remains normal. A chronic tear or advanced impingement syndrome will exhibit the universally restricted motion of a frozen shoulder.

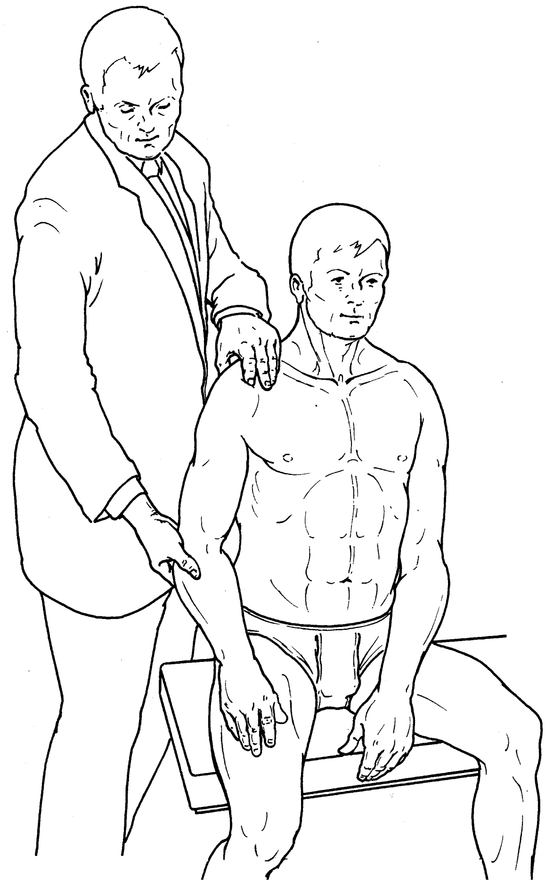


Fig. 2.6 Codman sign.

Palm Sign Test and Finger Sign Test

Typically, shoulder pain begins in the shoulder and radiates into the upper arm. Patients usually describe this pain in two ways. The “palm sign” is typical of glenohumeral and subacromial pain; the patient places the palm of the normal contralateral hand directly under the acromion.

The “finger sign” is typical of pain in the acromioclavicular joint; in this case, the patient places the finger of the normal contralateral hand directly on the affected acromioclavicular joint.

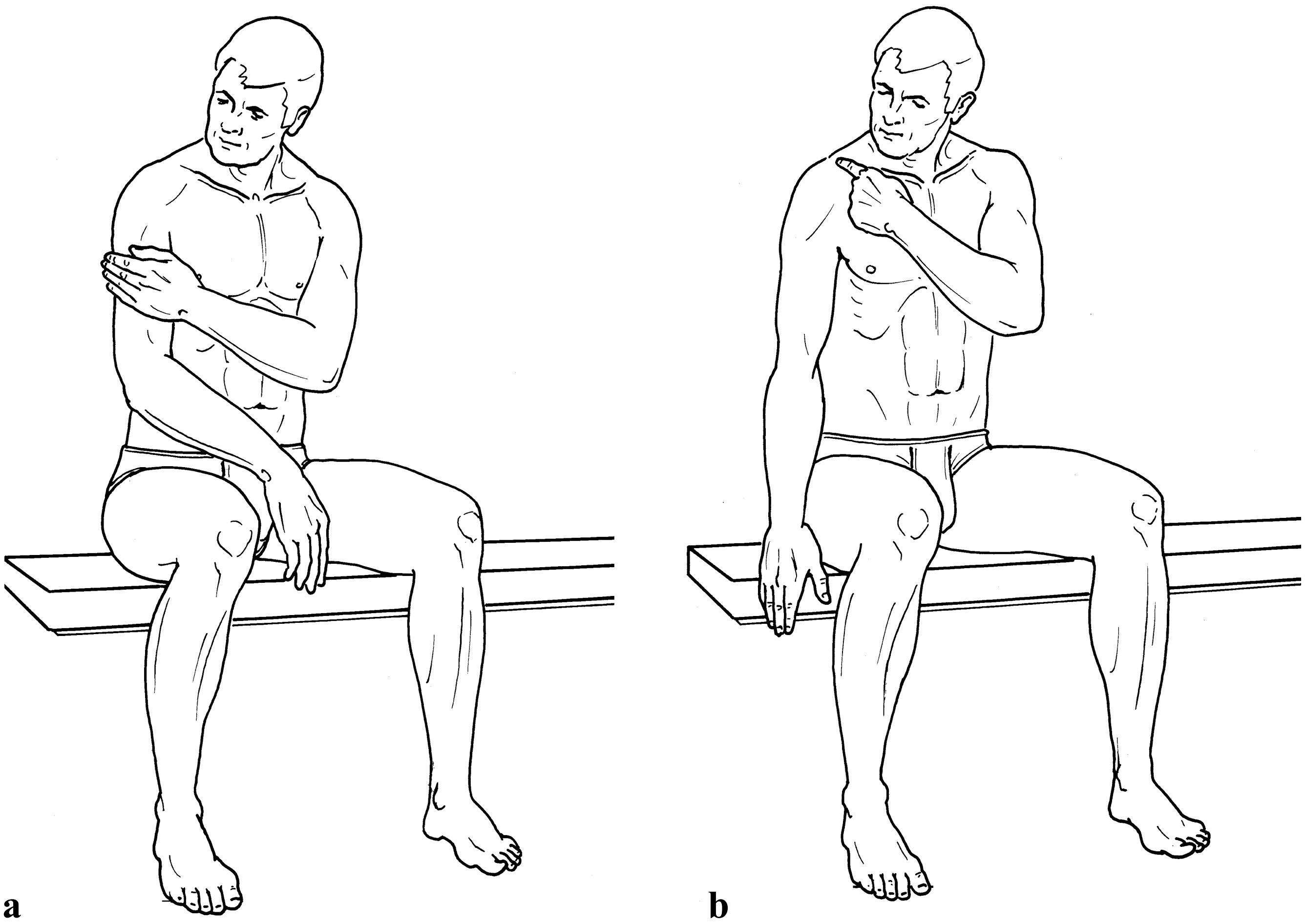


Fig. 2.7 (a) Palm sign test and **(b)** finger sign test.

Bursitis Tests

Bursae

The shoulder contains a series of bursae. Communicating structures include the subscapular and subcoracoid bursae and the subdeltoid bursa with its subacromial extension. Together, they form the "subacromial accessory joint" and ensure smooth motion between the rotator cuff and the acromion and acromioclavicular joint that lie superficial to it. Pathologic processes in the shoulder cause swelling of the bursal walls, thus provoking pain by further narrowing the subacromial space.

Bursitis Sign

Diagnosis of shoulder pains of uncertain etiology.

▫ **Procedure:** The examiner palpates the anterolateral subacromial region with his or her index and middle fingers.

The examiner can expand the subacromial space by passively extending or hyperextending the patient's arm with the other hand and pressing the humeral head forward with the thumb. This also allows palpation of the superior portions of the rotator cuff and its insertions into the greater tubercle of the humerus.

▫ **Assessment:** Localized tenderness to palpation in the subacromial space suggests irritation of the subacromial bursa but can also be a sign of a rotator cuff disorder.

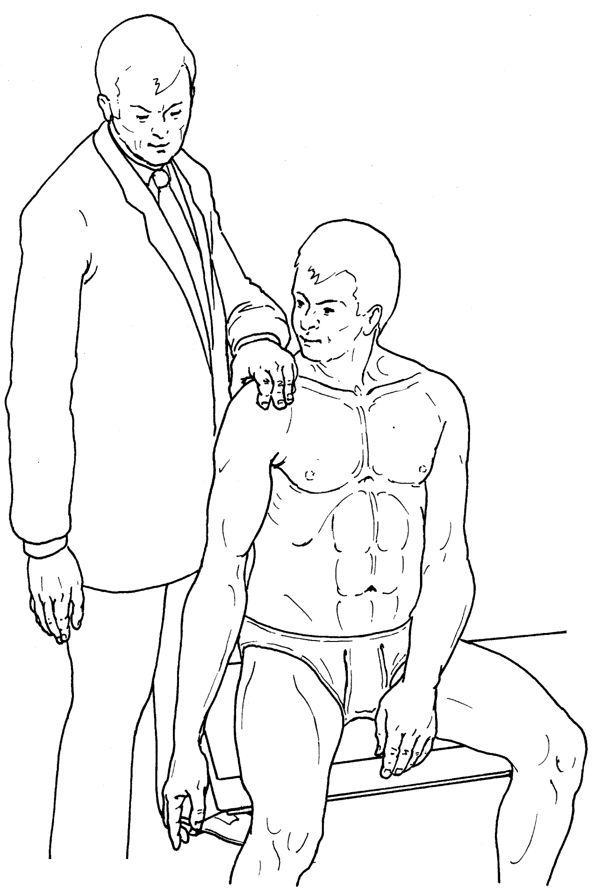


Fig. 2.8 Subacromial bursitis sign.

Dawbarn Test

A sign of subacromial bursitis.

□ **Procedure:** While further abducting the patient's moderately abducted arm with one hand, the examiner palpates the anterolateral subacromial space with the other hand.

The examiner exerts additional focal subacromial pressure while passively abducting the patient's arm up to 90°.

□ **Assessment:** Subacromial pain that decreases with abduction suggests bursitis or rotator cuff lesion. In abduction, the deltoid glides over the margin of the subacromial bursa, reducing the pain.

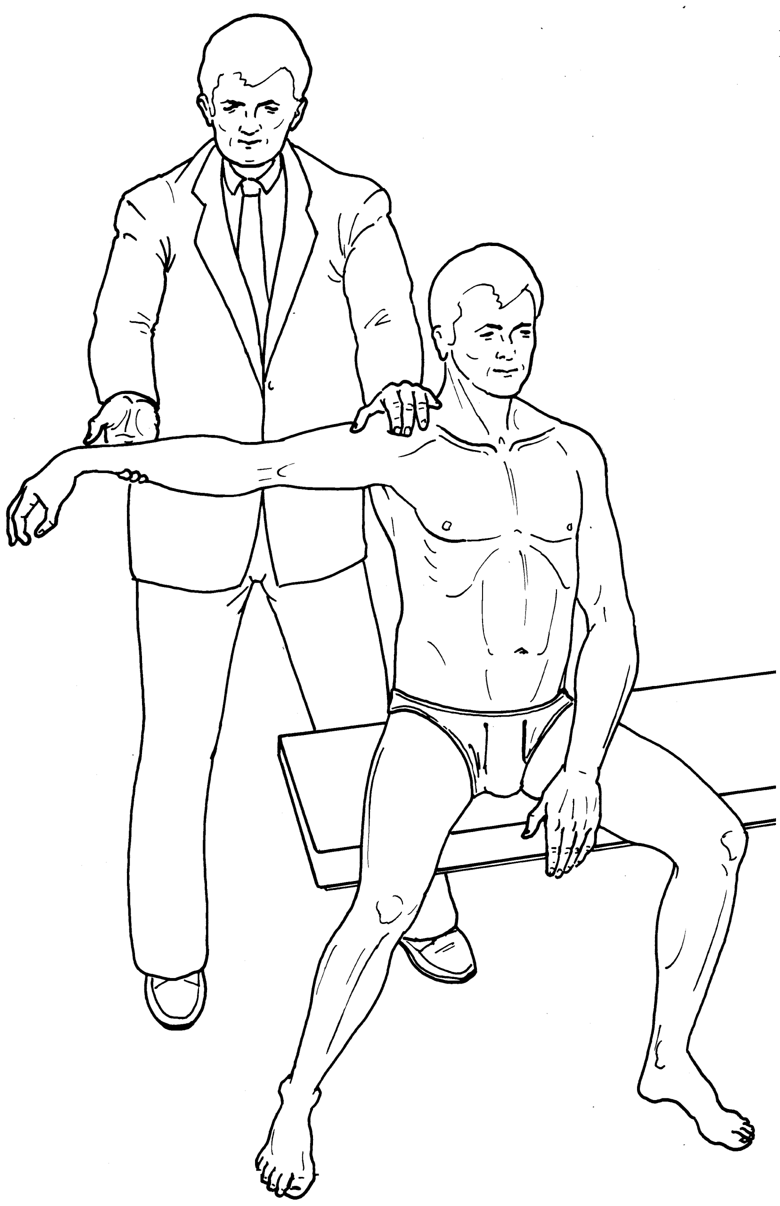


Fig. 2.9 Dawbarn test.

Scapulothoracic Dyskinesis

Disturbances in the scapulothoracic motion sequence usually occur because of muscular imbalance in conjunction with pathology of the glenohumeral and acromioclavicular joints. This can involve dysfunction from pain as a result of various shoulder disorders or from insufficient eccentric control function by the scapula-stabilizing muscles (trapezius, rhomboids, serratus anterior, and levator scapulae).

One can observe this scapular movement disturbance particularly when lowering the internally rotated arms from an elevated position, because this movement places significant stress on the eccentric muscles (**scapular provocation test**).

Kibler differentiates three types of scapular dyskinesis:

- Type I: Prominent inferior angle.
- Type II: Prominent medial margin with posterior tilt.
- Type III: Prominent superior angle.

Scapular Assistance Test

As with the classical impingement syndrome, patients with scapular dyskinesis frequently exhibit a painful arc between 60 and 120°. One can utilize the scapular assistance test to exclude impingement in the subacromial space caused by entrapment or irritation.

Fig. 2.10 Scapular assistance test.

□ **Procedure:** The examiner stands behind the patient. The patient is asked to actively elevate the arm while the examiner uses one hand to stabilize the upper medial border of the scapula (superior angle) and the other to support and rotate the inferior angle in a superolateral direction.
□ **Assessment:** The test is considered positive if there is reduction of pain in the "painful arc" and improved range of motion.

Rotator Cuff

Pain and varying degrees of functional impairment are typically the dominant features in the clinical picture of a rotator cuff lesion.

In the phase of acute pain, it will usually be difficult to obtain sufficient information from the examination to determine whether the shoulder pain is due to calcification, tendinitis, subscapularis syndrome, or a rotator cuff tear. It is even more difficult to distinguish a rotator cuff tear from disorders caused by degenerative tendon changes without rupture. Clinical classification of shoulder pain and muscle weakness becomes easier only once the pain of the acute phase has abated.

Active motion is nearly normal, but reduced overall, in supraspinatus muscle tears involving the anterior superior portion. The loss of active motion is more pronounced in injuries to the posterior portion and most extreme in complete tears. However, this is only an indication; the range of motion does not allow conclusions about the type of lesion.

Pseudo-stiffening of the shoulder must be distinguished from frozen shoulder. Pseudo-stiffening is often caused by advanced but minimally painful osteoarthritis in the sternoclavicular joint. If this change is not considered, one risks mistakenly attributing the decreased range of motion to changes in the glenohumeral joint. A good test to distinguish these two is to watch the patient shrug (elevate the shoulders); a limited range of motion may only be attributed to glenohumeral joint pathology where elevation of the shoulders is normal.

Scapular and thoracic pathology must be excluded in the same manner. A "creaking" shoulder due to bony projections such as scapular or costal osteophytes is less serious than the scapula that becomes fixed in a posterior thoracic defect, such as can occur secondary to thoracoplasty or multiple fractures of adjacent ribs. It is equally important to exclude dysfunction of the shoulder musculature, whether the scapular and thoracic muscles or the glenohumeral musculature. The examiner must be alert to the possibility of serratus muscle palsy, which can be demonstrated by winging of the scapula when one pushes against the patient's outstretched arms. Paralysis of the trapezius must also be excluded. This paralysis limits mobility in the shoulder because the scapula can no longer be immobilized. The ability to elevate the scapula rules out this paralysis, as does the ability to elevate the shoulders (in shrugging).

Even under normal circumstances, there is little space available for the structures that lie beneath the coracoacromial arch. This space is further diminished when the greater tubercle of the humerus moves beneath the acromion in elevation. The supraspinatus muscle is particularly affected by this confinement. The space available for its motion is limited on all sides by the anterior acromion, the coracoacromial ligament, the acromioclavicular joint, and the coracoid process (the supraspinatus outlet).

Impingement syndrome is a painful functional impairment of the shoulder that occurs when the rotator tendons impinge on the anterior margin of the coracoacromial arch and/or the acromioclavicular joint. The rotator cuff and the bursa beneath it can be locally compressed on the anterior margin of the acromion in elevation, and against the coracoid process in internal rotation. A subacromial or subcoracoid impingement syndrome can occur. Impingement lesions can also involve structures other than the rotator cuff that lie in the impingement zone, such as the biceps tendon and the subacromial bursa.

According to Neer, a distinction is made between primary impingement (outlet impingement) and secondary impingement (non-outlet impingement). Primary impingement involves irritation of the supraspinatus as a result of mechanical constriction (in the supraspinatus outlet). Contributing factors may include congenital changes in the shape of the acromion, acquired bone spurs on the anterior margin of the acromion, inferior osteophytes on the acromioclavicular joint, and posttraumatic deformities of the coracoid process, acromion, and greater tubercle of the humerus. Secondary impingement (subacromial syndrome) involves relative constriction of the subacromial space due to the increase in volume of the structures that pass beneath the coracoacromial arch. Thickening of the rotator cuff and bursa (due to calcifications or chronic bursitis) and posttraumatic superior displacement of the greater tubercle of the humerus are the most common causes.

According to Keyl, the failure of the depressor muscles of the humeral head that occurs in a tear of the rotator cuff or the long head of the biceps tendon is the principal cause of secondary impingement. Where a defective rotator cuff is no longer able to counterbalance the superior pull of the deltoid, elevating the shoulder will cause the humeral head to shift upward and produce impingement. The same also applies to shoulder instability, where, especially in multidirectional displacement, the humeral head is pulled against the roof of the joint capsule, producing impingement. Functional constriction can also result where muscular paralysis and weakness prevent involvement of the scapula in the overall elevation of the arm, or where separation of the acromioclavicular joint has eliminated its supporting structures. Finally, one should also

2

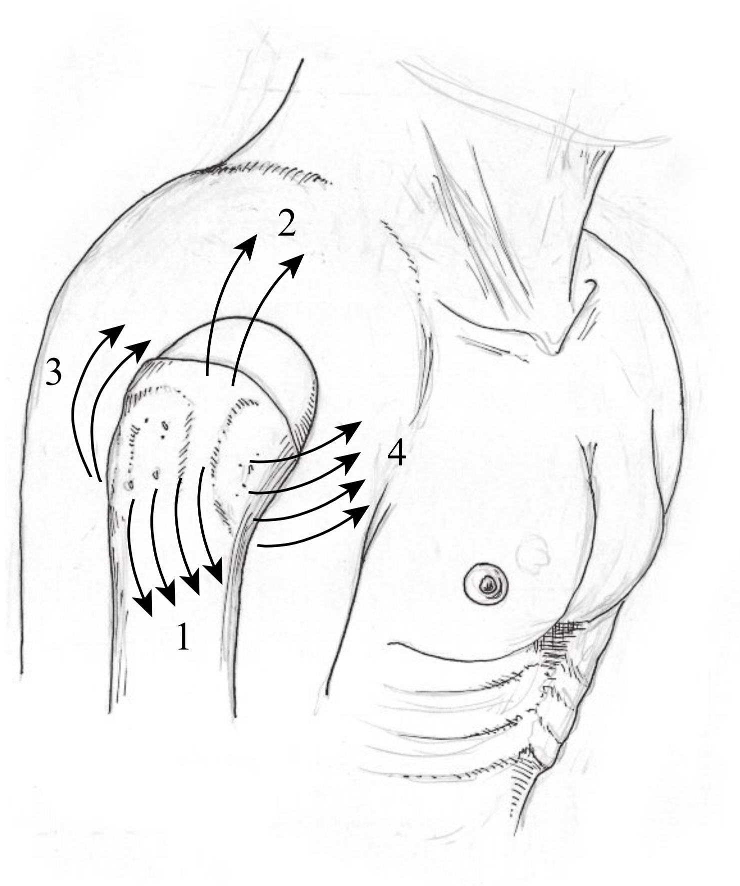


Fig. 2.11 Muscle functions at the shoulder.
1 Adductor muscles (pectoralis major, latissimus dorsi, teres major and teres minor).
2 Abductor muscle (deltoid and supraspinatus).
3 External rotator muscles (deltoid and supraspinatus).
4 Internal rotator muscles (subscapularis, pectoralis major, latissimus dorsi and teres major).

remember the pathogenetic significance of a shrunken posterior capsule. If the humeral head cannot glide far enough posteriorly in flexion, it will be increasingly pressed against the anterior margin of the acromion, resulting in impingement.

The chronic stage of impingement syndrome can involve clinically conspicuous atrophy of the deltoid muscle as well as the supraspinatus and infraspinatus muscles. The tendon insertions on the greater and lesser tubercles of the humerus are often tender to palpation, and mobility in the glenohumeral joint is often limited toward the end of its range of motion. Active elevation is more painful than passive elevation.

If the patient is able to abduct his or her arm against resistance in spite of pain, this suggests degenerative tendon changes rather than a tear. The Neer impingement injection test allows one to clinically distinguish between weakness in abduction due to a rupture and that due to pain. In the presence of a tendon rupture, the weakness in abducting the arm may be expected to remain even after infiltration of the subacromial space with anesthetic has reduced or eliminated pain.

A patient with “pseudoparalysis” is unable to lift the affected arm. This global sign suggests a rotator cuff disorder. Further examinations are then required to identify the damaged tendon. Provocati tests can be very helpful in this regard. External and internal rotation against resistance is evaluated with the shoulder in various positions. Weakness is more probably due to a functional deficit (such as a rupture), whereas pain is more probably attributable to inflammation of the tendon insertions or the adjacent bursae.

Zero-Degree Abduction Test (Starter Test)

Procedure: The patient stands with his or her arms hanging relaxed. The examiner grasps the distal third of each forearm. The patient attempts to abduct the arms against the examiner's resistance.

Assessment: Abduction of the arm is "started" by the supraspinatus and deltoid muscles. Pain and, especially, weakness in abducting and holding the arm strongly suggest a rotator cuff tear.

Eccentricity of the humeral head in the form of superior displacement of the humeral head in a rotator cuff tear causes relative insufficiency of the outer muscles of the shoulder. Small tears that can be functionally compensated for will cause minor loss of function with the same amount of pain. Larger tears are invariably characterized by weakness and loss of function.

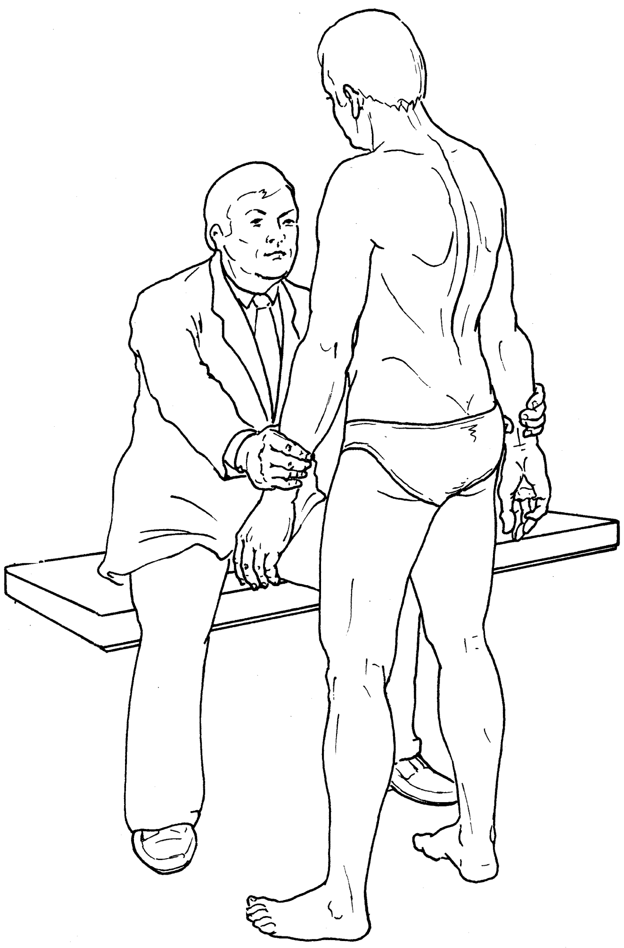


Fig. 2.12 Zero-degree abduction test.

2

Jobe Supraspinatus Test (Empty Can Test)

□ **Procedure:** This test may be performed with the patient standing or seated.

With the elbow extended, the patient's arm is held at 90° of abduction, 30° of horizontal flexion, and in internal or external rotation. The examiner exerts pressure on the upper arm during the abduction and horizontal flexion motion. Using electromyography, this test enables testing of the supraspinatus muscle largely in isolation. It is important to apply pressure gently at first and to increase the pressure only if pain has not been triggered during the course of the test to that point.

□ **Assessment:** When this test elicits severe pain and the patient is unable to hold his or her arm abducted 90° against gravity, this is called a positive **drop arm sign**.

The superior portions of the rotator cuff (supraspinatus) are particularly assessed in internal rotation (with the thumb down as when emptying a can), and the anterior portions in external rotation (thumb points upward—full can).

The test may be repeated at only 45° abduction to further differentiate the findings. Where the impingement component predominates, there will be less pain and more strength where the tendon is still intact. The test can yield false-positive results where pathology of the long head of the biceps tendon is present.

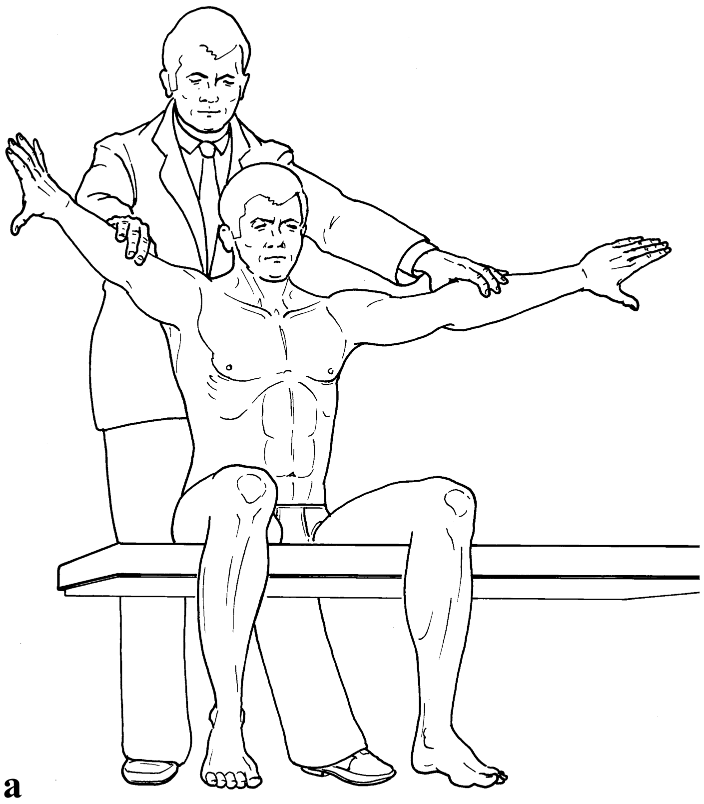
If the test elicits pain and the patient is unable to abduct the arm 90° and hold it against gravity, this indicates a tear of the tendon of the supraspinatus muscle, or damage to the suprascapular nerve.

Strength in the supraspinatus muscle may not be completely diminished until over two-thirds of the tendon is torn.

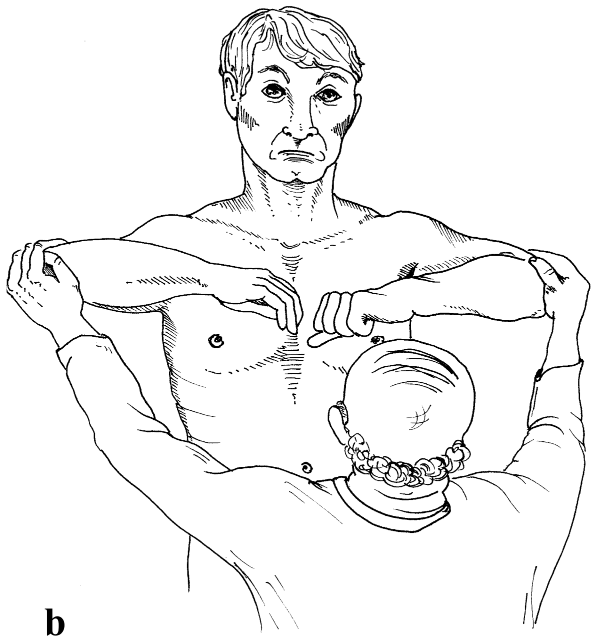
Studies performed by anesthetizing the suprascapular and axillary nerves show that the supraspinatus and deltoid muscles are responsible for abduction of the arm (**Fig. 2.11**). The supraspinatus muscle, along with the other muscles of the rotator cuff, press the head of the humerus into the socket and abduct the arm for the first 20°. Then the deltoid muscle comes into play. Even if the supraspinatus muscle tendon is completely torn, the shoulder is still capable of good range of motion. There is a deficit only at the onset of abduction and then again when the arm reaches 90° and above.

EMG tests show no difference in the EMG activity whether the arm is held in full internal rotation (classic Jobe *empty can* position), with the thumb pointing to the floor, or with the arm in maximum external rotation (*full can* position).

The strength of the supraspinatus muscle can also be tested with the elbows flexed rather than extended. For the patient, this position requires less holding power and less stress, and is therefore also less painful.



a



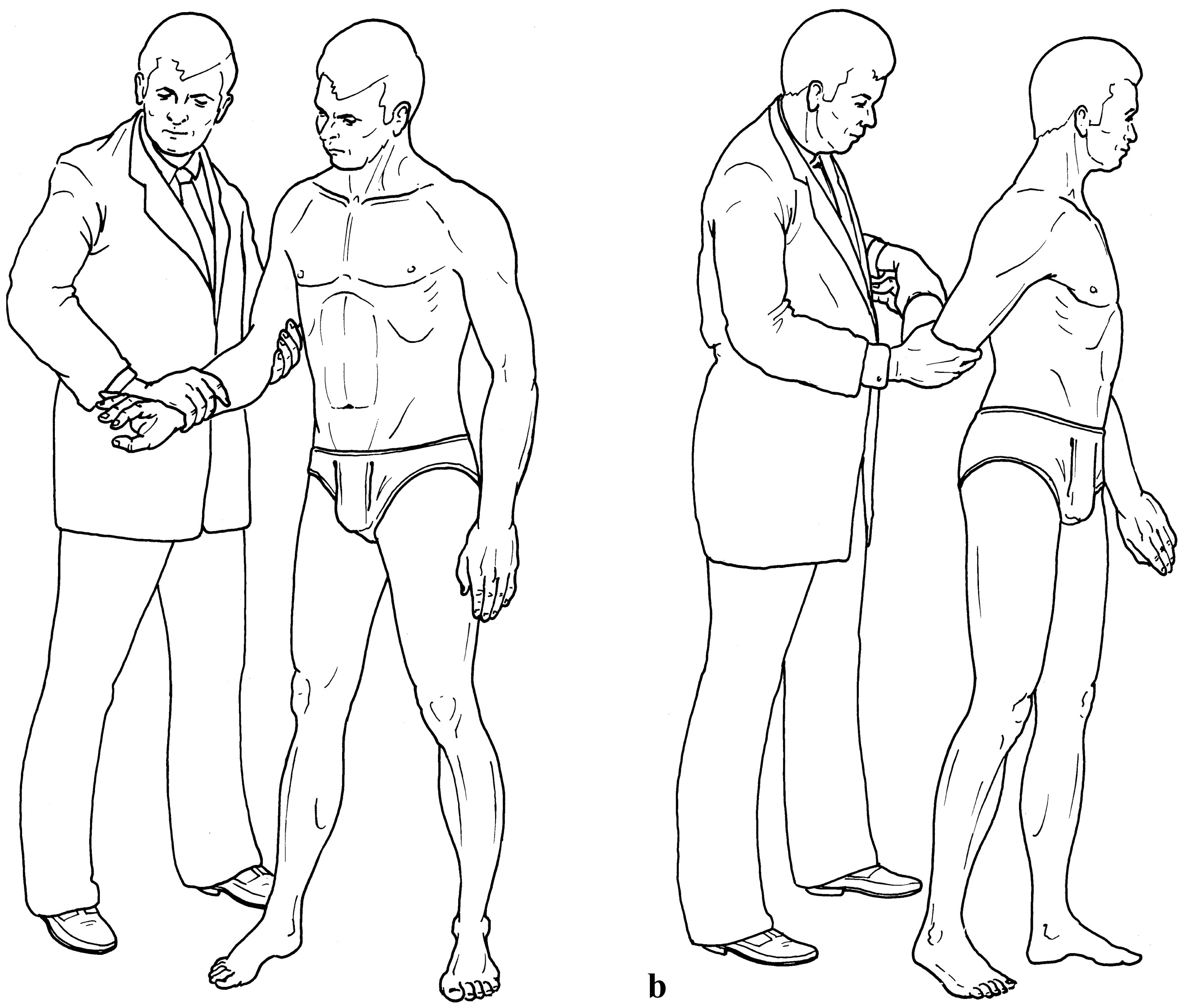
b

Fig. 2.13 (a,b) Jobe supraspinatus test.

Subscapularis Muscle Test

□ **Procedure:** This test is the reverse of the zero-degree external rotation (infraspinatus) test (p. 97). With the patient's elbow alongside but not quite touching the trunk, the examiner comparatively assesses passive external rotation in both arms and active internal rotation of the shoulder against resistance.

□ **Assessment:** An increase in painless passive external rotation and weakness of active internal rotation in comparison with the contralateral side suggests an isolated tear of the subscapularis tendon. A tear of the subscapularis muscle manifests itself as pain and weakness of internal rotation. Reduced strength in conjunction with minimal pain is more likely to represent a tear. When pain is more severe, it is difficult to distinguish between a tear and tendinopathy.



a b

Fig. 2.14 Subscapularis muscle test. **(a)** Passive external rotation. **(b)** Active internal rotation behind the back.

Internal Rotation Lag Sign (IRLS)

□ **Procedure:** The patient stands with his or her back to the examiner, who brings the arm passively into extension and submaximal internal rotation behind the patient's back. One should avoid maximal internal rotation to avoid the elastic-recoil effect of the capsule. Then the patient is asked to hold this position.

□ **Assessment:** If there is a tear of the subscapularis tendon, the patient is unable to actively hold the submaximally internally rotated position. The arm recoils against the back. This test is especially well suited to clinical evaluation of the superior portion of the tendon.

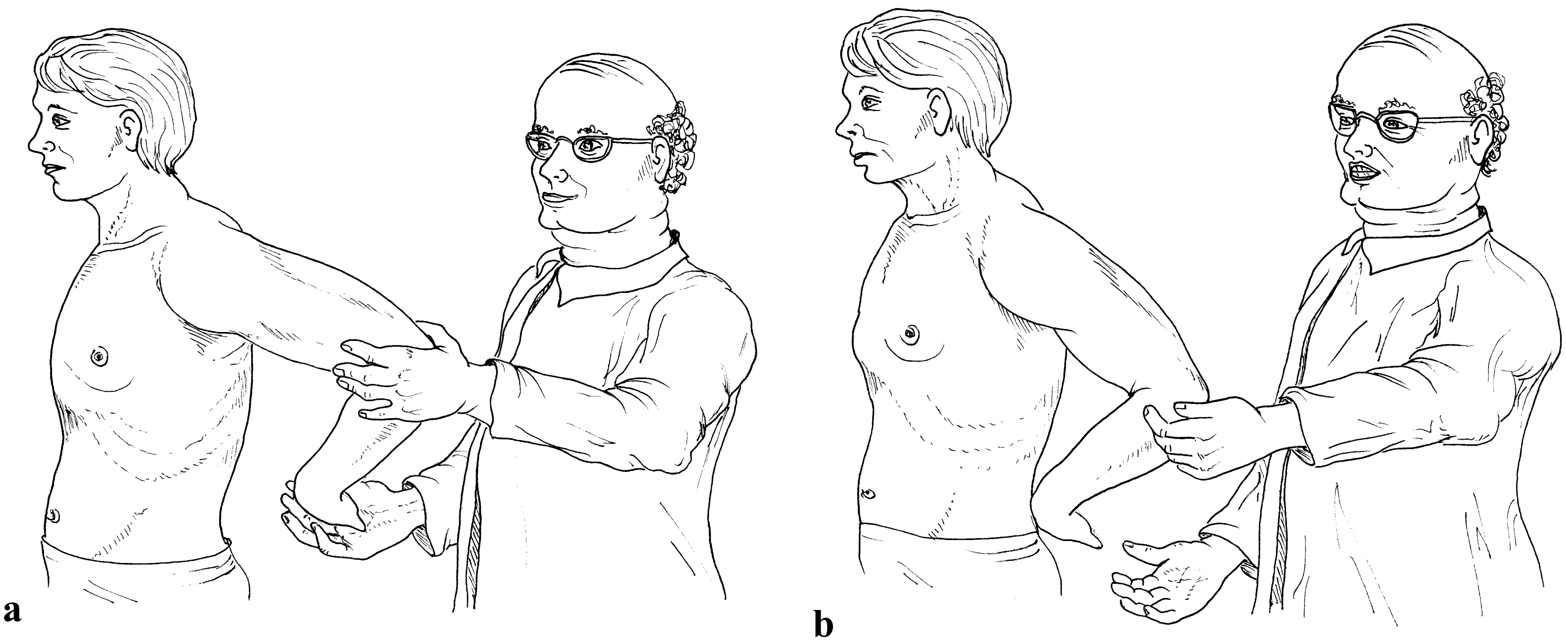


Fig. 2.15 Internal rotation lag sign. **(a)** Arm in submaximal internal rotation. **(b)** Recoil of the arm when there is a tear of the subscapularis muscle.

Gerber Lift-Off Test

□ **Procedure:** With the arm in internal rotation, ask the patient to place the dorsum of the hand against the back. Then ask the patient to lift the hand away from the back. If the patient is able to do this, the examiner should apply an increasing counterpressure against the patient's hand to test the strength of the subscapularis muscle.

□ **Assessment:** If there is a tendon rupture or muscle insufficiency, the patient will be unable to actively lift the hand off the back against the examiner's resistance. Where pain renders maximum internal rotation impossible, the belly press test may be performed.

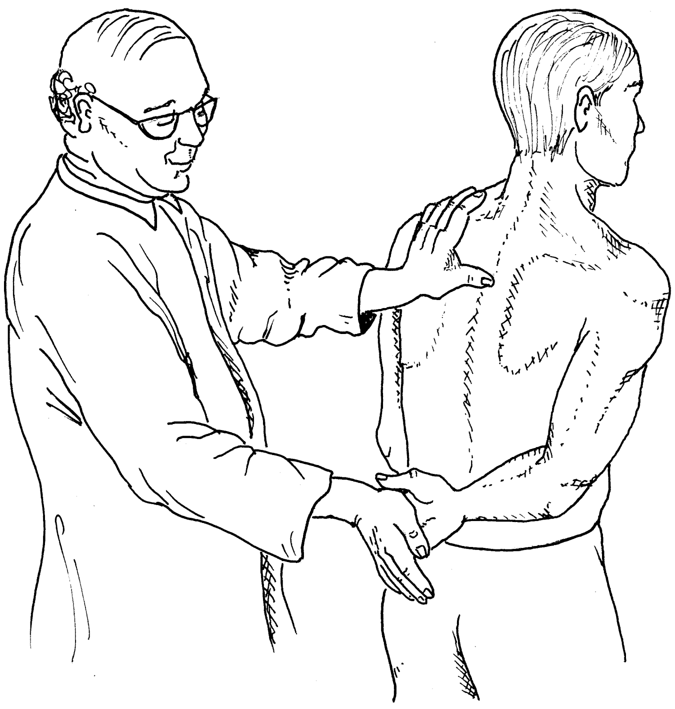


Fig. 2.16 Lift-off test.

Because many patients with biceps subluxations also have partial or full-thickness tears of the subscapularis, a positive lift-off test compels one to think of biceps tendon pathology as well.

Belly Press (Abdominal Compression) Test

- **Procedure:** The patient is standing. The examiners places the patient's forearm on the abdomen with the elbow flexed and the arm maximally internally rotated. The patient is now requested to press the arm forcefully against the abdomen while holding the elbow forward.
- **Assessment:** If there is a tear in the subscapularis tendon, the patient will not be able to hold the hand firmly against the abdomen, and it drifts off. The elbow deviates laterally and posteriorly under the influence of the latissimus dorsi and teres major muscles. Flexion also occurs in the wrist.

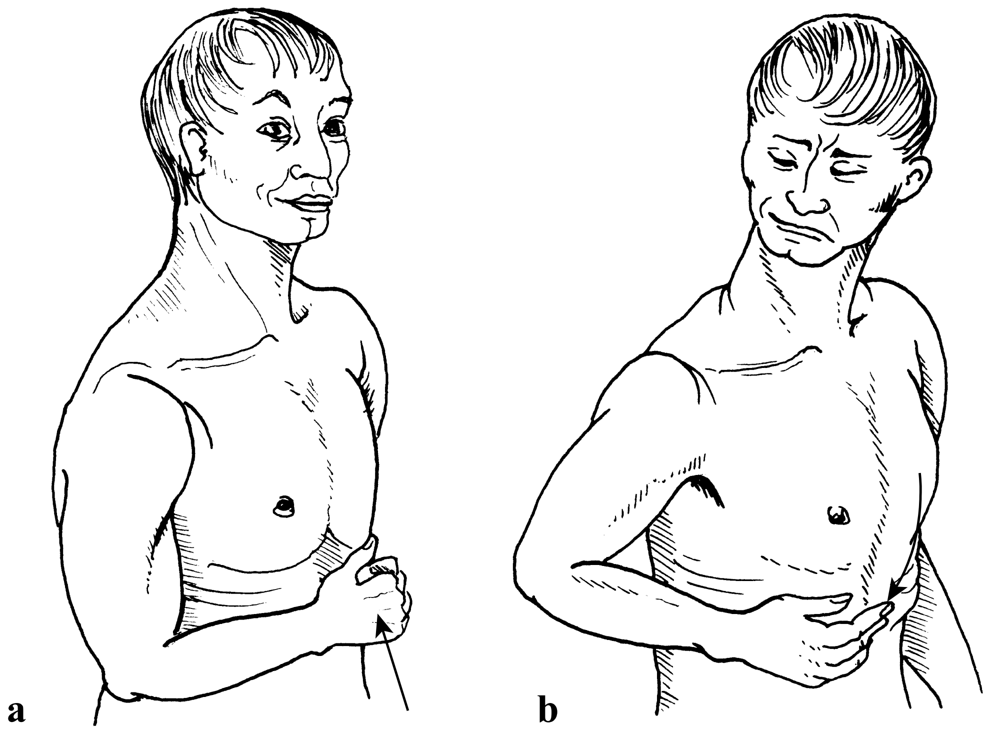


Fig. 2.17 Belly press test (abdominal compression test). **(a)** The forearm lies along the abdomen with the elbow flexed. **(b)** The arm deviates laterally and posteriorly while the wrist is flexed

Belly-Off Test

□ **Procedure:** In this test, the examiner lays the patient's arm passively on the abdomen in a fully flexed and internally rotated position with the wrist extended. The examiner asks the patient to hold this position.

□ **Assessment:** The belly-off test is positive if the patient cannot hold the position, and the hand slips away from the abdomen. In the case of an isolated partial subscapularis tendon tear, this test is usually positive as a result of the preponderance of externally rotating forces.

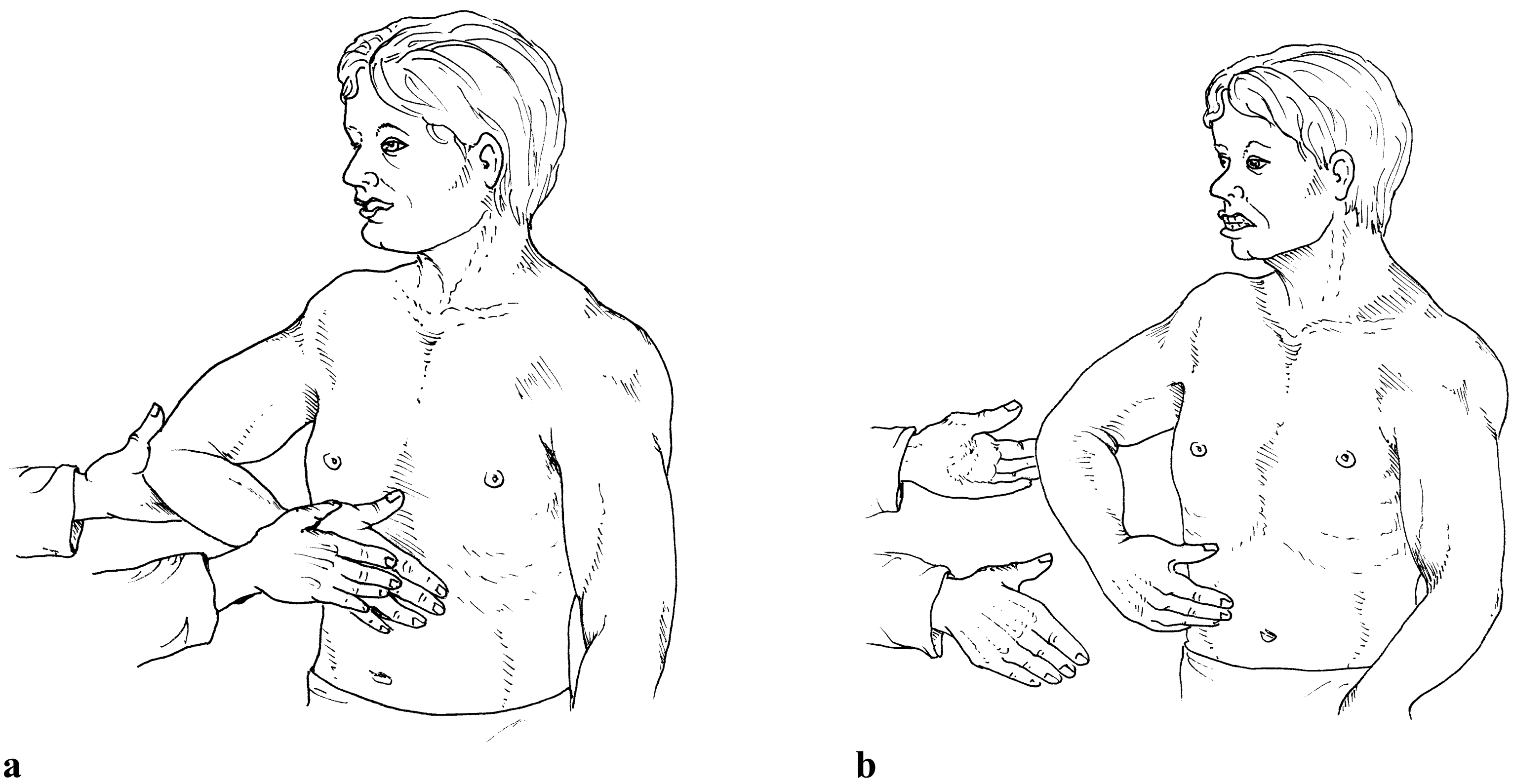


Fig. 2.18 Belly-off test. **(a)** The arm at end-range flexion and internal rotation. **(b)** The patient cannot actively hold the hand against the abdomen.

Bear-Hug Test

□ **Procedure:** The patient places the palm of the hand of the affected side on the opposite shoulder with outstretched fingers and the elbow pointing forward. While the patient tries to hold this position by actively internally rotating, the examiner grasps the patient's wrist and tries to loosen the hand from the shoulder by forcibly externally rotating the arm, pulling at right angles to the forearm.

□ **Assessment:** If there is a tear of the subscapularis tendon, especially of the upper part, the patient is unable to maintain the hold on the opposite shoulder. Pain and muscle weakness in comparison with the other side suggests muscle dysfunction.

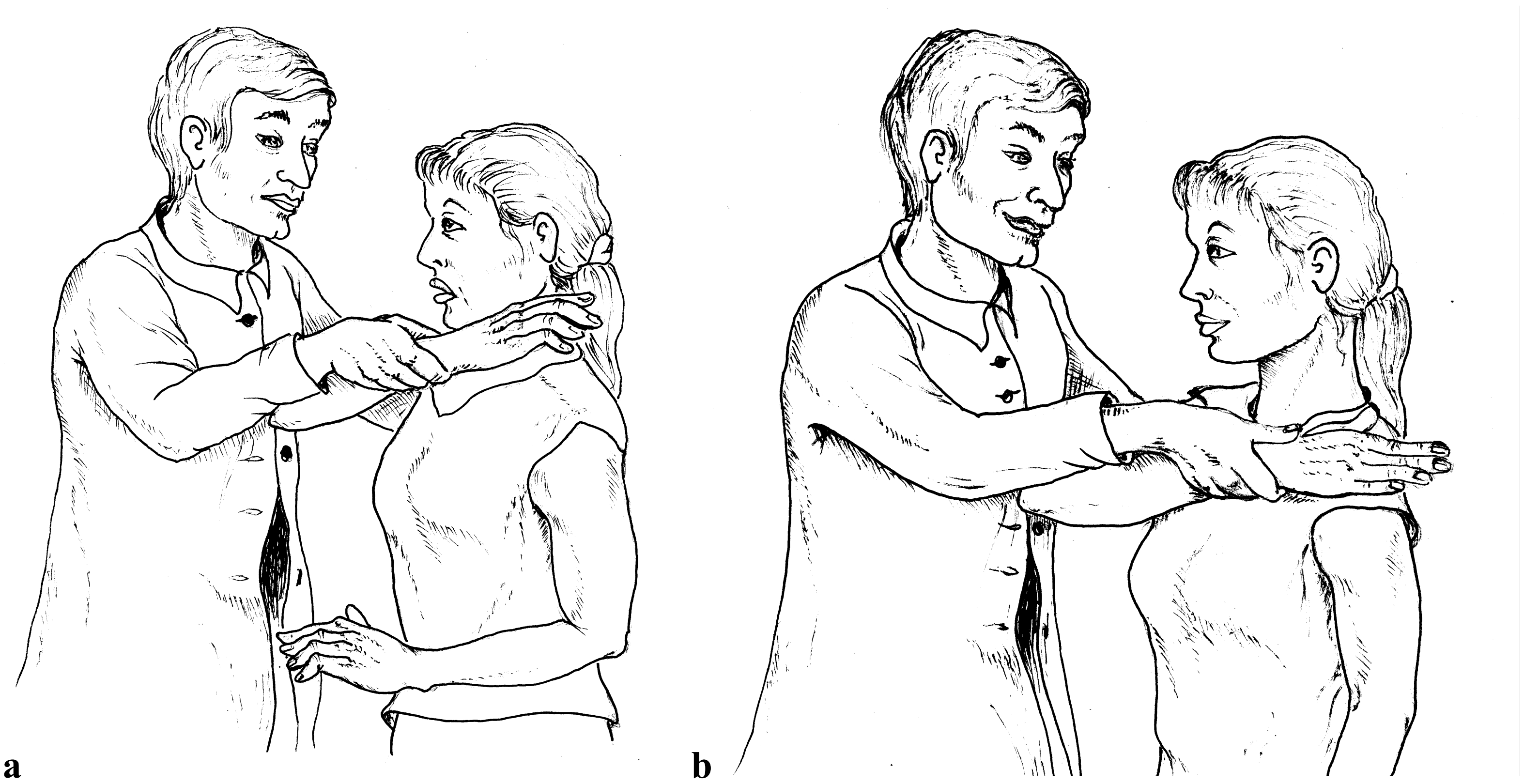


Fig. 2.19 Bear hug test. **(a)** The hand on the side of the affected shoulder brought to the opposite shoulder. **(b)** The patient cannot hold the hand against the externally rotating force of the examiner.

Napoleon Sign

□ **Procedure:** The examiner instructs the patient to press both palms forcefully against the abdomen with the wrists in an extended position and the elbows flexed. At the same time, the patient is asked to hold the elbows as far forward as possible.

□ **Assessment:** This movement sequence normally produces an anterior motion of the elbows due to tension in the subscapular musculature. In patients with a rupture in the subscapularis tendon, the position of the arm remains unchanged. Some patients also exhibit increased passive external rotation. The wrist tends to flex as well; Burkhart and Thereny developed a classification system based on the degree of flexion. If the wrist is flexed to 90° (a positive result), one assumes that there is a complete rupture of the subscapularis tendon. If there is flexion of 30 to 60° (intermediate result), then there is usually a tear of the upper two-thirds of the tendon.

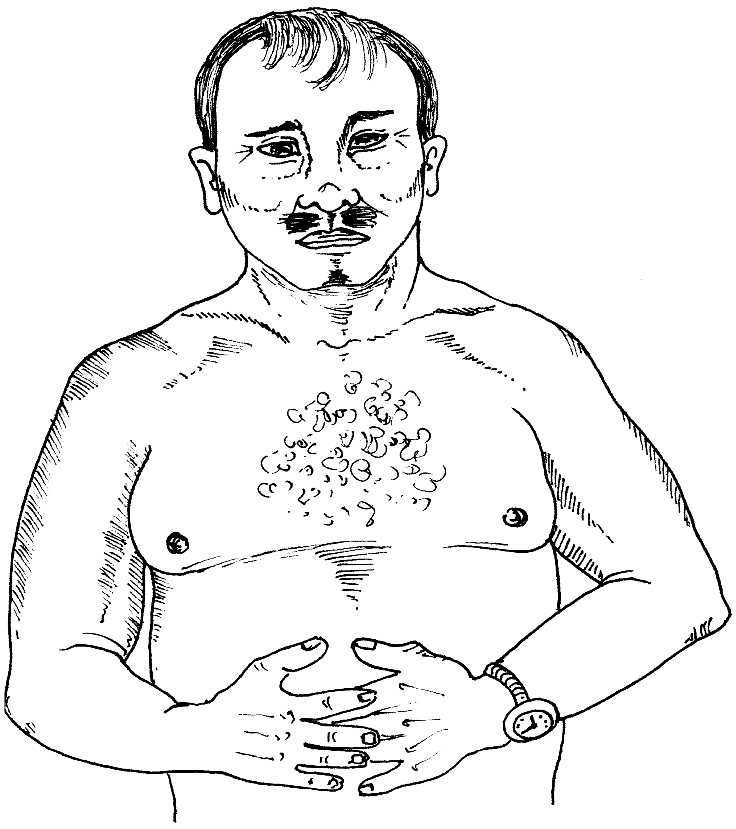


Fig. 2.20 Napoleon sign (positive on the right side).

Zero-Degree External Rotation Test (Infraspinatus Muscle Test)

□ **Procedure:** This test may be performed with the patient seated or standing.

Comparative testing of both sides is best. The patient's arms should hang relaxed with the elbows flexed 90° but not quite touching the trunk. The examiner places his or her palms on the dorsum of each of the patient's hands and then asks the patient to externally rotate both forearms against the resistance of the examiner's hands.

□ **Assessment:** Pain or weakness in external rotation indicates a disorder of the infraspinatus muscle (external rotator). As infraspinatus tears are usually painless, weakness in rotation strongly suggests a tear in this muscle. This test can also be performed with the arm abducted 90° and flexed 30° to eliminate involvement of the deltoid in this motion (see Patte Test, p.99).

The most common etiology for the atrophy of the infraspinatus muscle is a tendon tear or damage to the infraspinatus branch of the suprascapular nerve by compressive lesions (synovial cysts, exostoses) or by traction injuries (overhead athletes such as volleyball players).

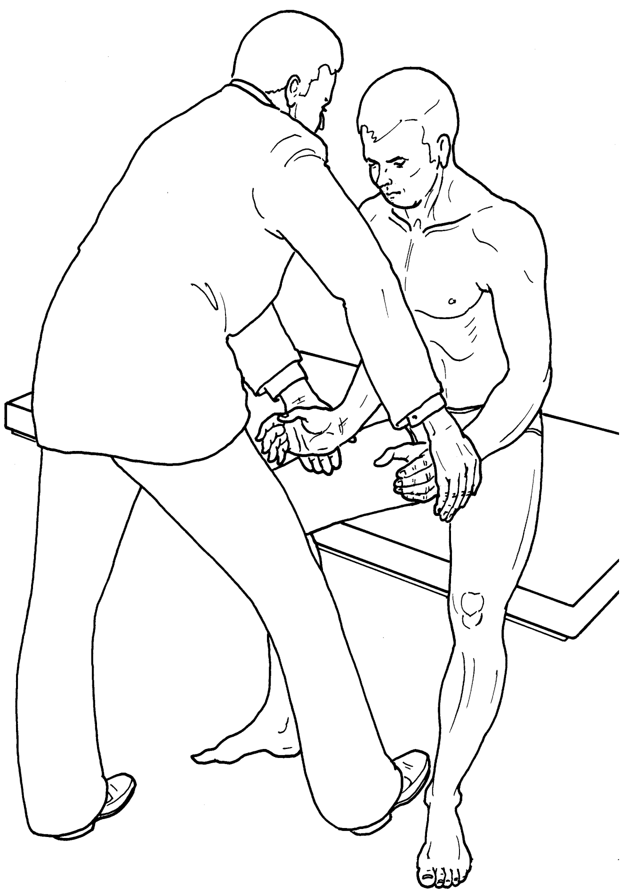


Fig. 2.21 Zero-degree external rotation test (infraspinatus test).

External Rotation Lag Sign (ERLS)

□ **Procedure:** The patient sits with his or her back to the examiner, who holds the patient's arm passively in submaximal external rotation (not maximal external rotation to avoid the effect of the capsule) at 20° abduction with the elbow flexed to 90°. The patient is then asked to hold this externally rotated position.

The same test can be repeated with the arm abducted to 90°, which specifically tests the infraspinatus and teres minor muscles.

□ **Assessment:** If the patient cannot hold the arm in the position into which it was passively placed (submaximal external rotation with slight abduction) and it reverts to the internally rotated position, then this indicates a lesion of the supraspinatus tendon. If the position cannot be held at 90° abduction, the test is highly sensitive and specific for a lesion of the teres minor muscle.

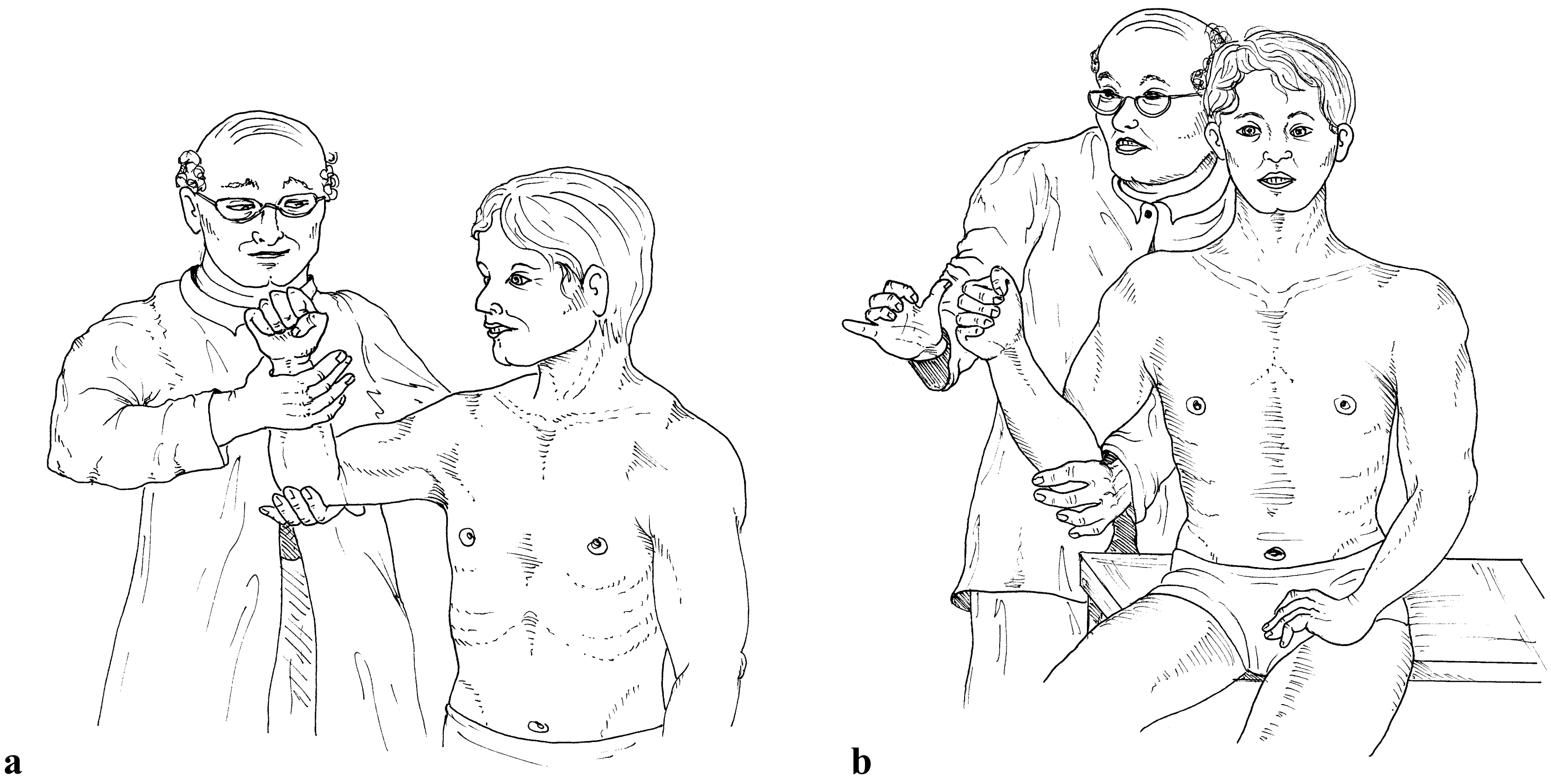


Fig. 2.22 External rotation lag sign. **(a)** The shoulder brought to 20° (or 90°) abduction and submaximal external rotation. **(b)** After release, the arm reverts back into internal rotation.

Abduction External Rotation Test (Patte Test)

□ **Procedure:** The arm is abducted 90° and flexed 30°. This neutralizes the effect of the deltoid in external rotation. The patient attempts to continue to externally rotate the arm against the resistance of the examiner's hand.

□ **Assessment:** The lack of active external rotation in the abducted arm suggests a clinically significant rupture of the infraspinatus tendon.

Performing the test at over 45° of external rotation primarily tests the teres minor.

Fig. 2.23 Abduction external rotation test.

Nonspecific Supraspinatus Muscle Test

□ **Procedure:** The patient is seated with the arm abducted 90° with the examiner's hand resting on the patient's forearm. The examiner then asks the patient to further abduct the arm against the examiner's resistance.
□ **Assessment:** Weakness in further abduction and/or pain indicate pathology of the supraspinatus tendon.
□ **Note:** Further abduction is sometimes also associated with pain in the acromioclavicular joint.

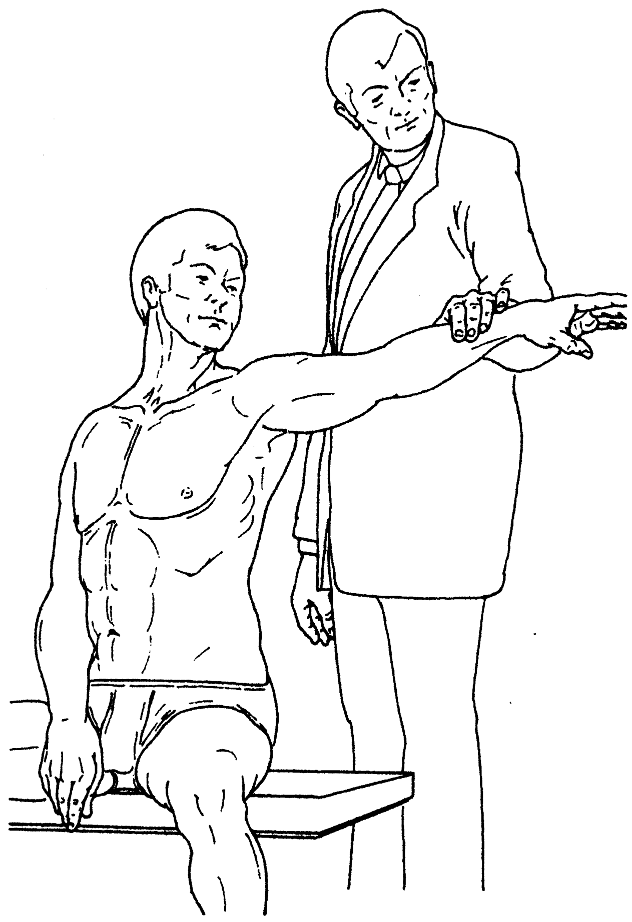


Fig. 2.24 Nonspecific supraspinatus muscle test.

Drop Arm Test

□ **Procedure:** The patient is seated and the extended arm passively abducted 90°. The patient is instructed to hold the arm in this position without support and then slowly lower it.
□ **Assessment:** Weakness in maintaining the position of the arm, with or without pain, or sudden dropping of the arm suggests a rotator cuff lesion. Most often this is due to a defect in the supraspinatus muscle. In pseudoparalysis, the patient will be unable to lift the affected arm. This global sign suggests a rotator cuff disorder.

A positive drop arm test may also have neurologic causes, such as damage to the subscapular nerve, so the patient must be thoroughly examined from a neurologic standpoint.

In lesions of the supraspinatus muscle as a result of chronic degenerative processes, the drop arm test may be falsely negative due to muscular compensation, especially by the deltoid muscle.

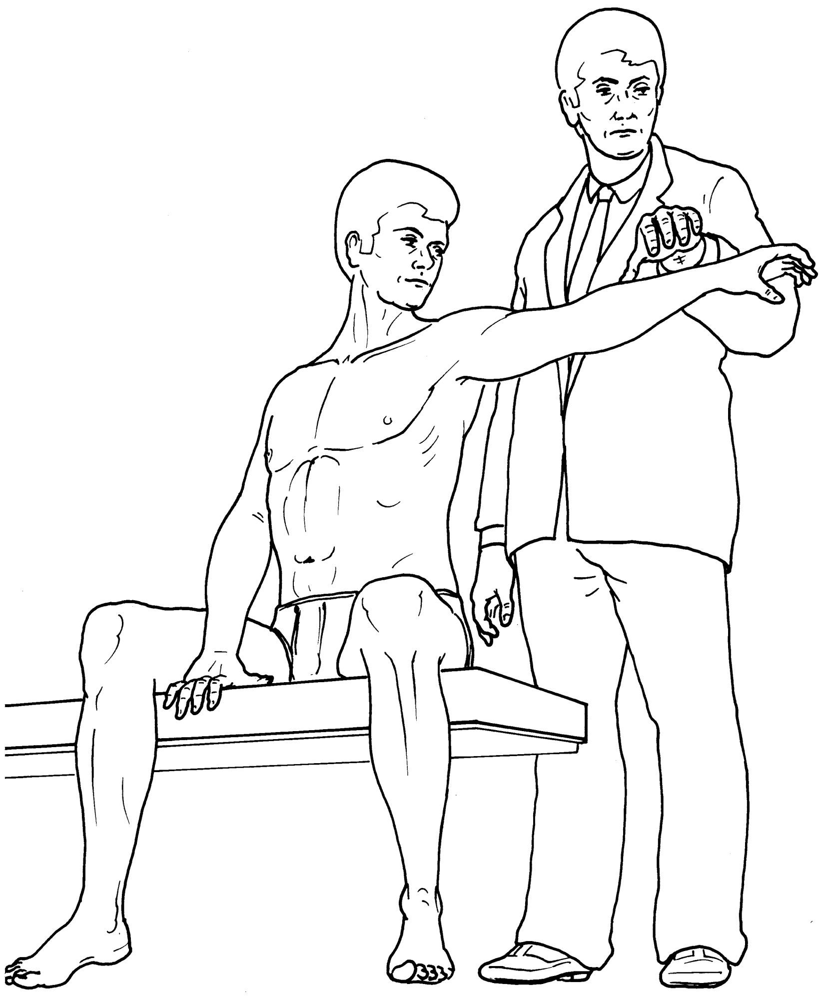


Fig. 2.25 Drop arm test.

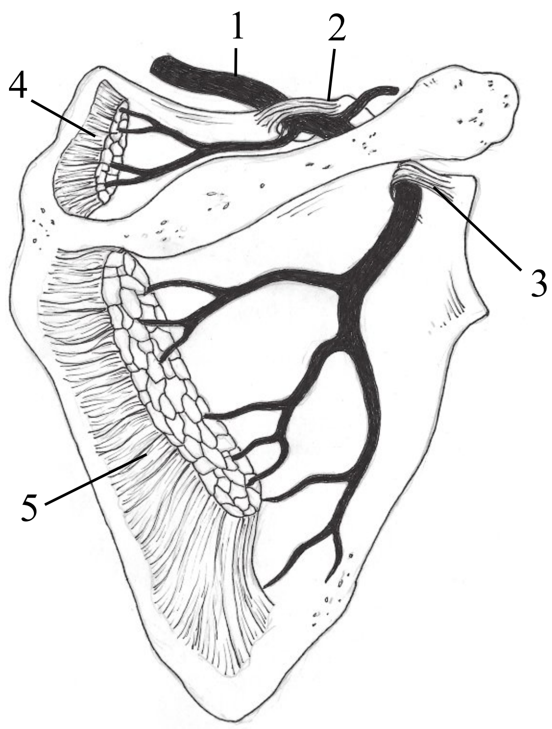


Fig. 2.26 Course and distribution area of the suprascapular nerve.

1 Suprascapular nerve.
2 Superior transverse scapular ligament.
3 Inferior transverse scapular ligament (spinoglenoid ligament).
4 Supraspinatus muscle.
5 Infraspinatus muscle.

Walch/Hornblower Sign

□ **Procedure:** The patient is requested to touch his or her mouth with the affected hand.

□ **Assessment:** Where there is complete insufficiency of both external rotators (infraspinatus and teres minor), the arm will deviate into internal rotation and the patient will have to lift the elbow higher than the hand.

To bring the hand to the mouth, the patient must first elevate the arm to 90°. From this position, the arm falls into internal rotation, now enabling the hand to reach the mouth.

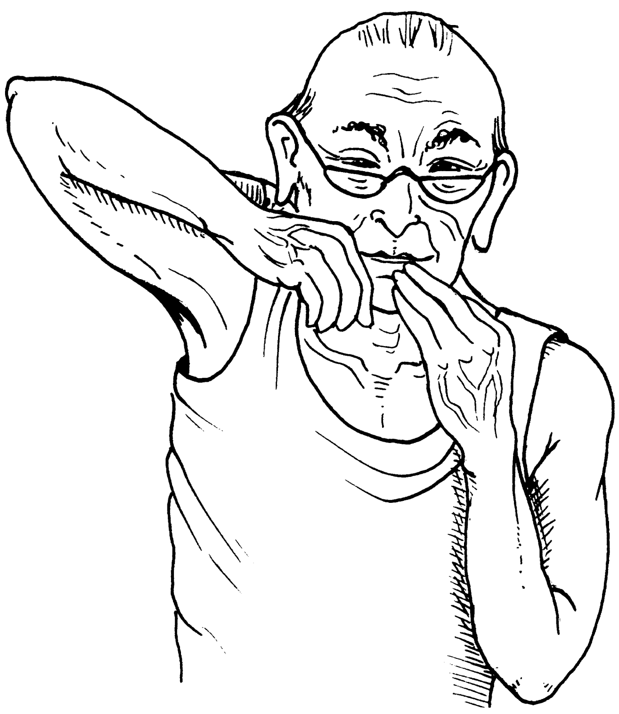


Fig. 2.27 Hornblower sign.

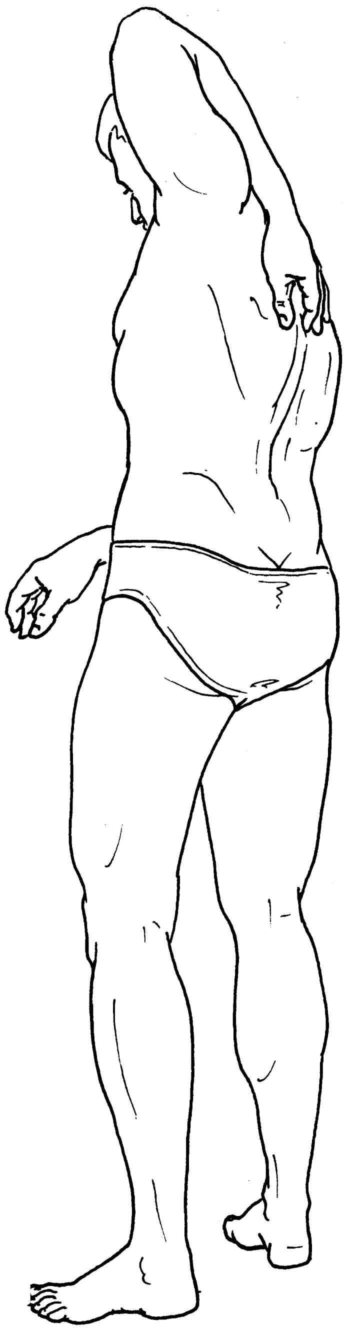


Fig. 2.28 Apley scratch test.

Apley Scratch Test

□ **Procedure:** The patient is asked to touch the contralateral superior part of the medial border of the scapula with the index finger.

□ **Assessment:** Pain elicited in the rotator cuff and failure to reach the scapula because of restricted mobility in external rotation and abduction indicate rotator cuff pathology (most probably involving the supraspinatus). A differential diagnosis should consider osteoarthritis in the glenohumeral and acromioclavicular joints as well as capsular fibrosis.

Painful Arc I

□ **Procedure:** The arm is passively and actively abducted from the rest position alongside the trunk.

□ **Assessment:** Pain occurring in abduction between 60 and 120° (**Fig. 2.29b**) is a sign of a lesion of the supraspinatus tendon, which becomes impinged between the greater tubercle of the humerus and the acromion in this phase of the motion (subacromial impingement). Contrast this with the painful arc in acromioclavicular joint disorders, where the pain only occurs only at 140 to 180° of abduction (p. 190). Patients are usually free of pain above 120°.

In the evaluation of the active and passive ranges of motion, the patient can often avoid the painful arc by externally rotating the arm while abducting it. This increases the clearance between the acromion and the diseased tendinous portion of the rotator cuff, avoiding impingement in the range between 60 and 120°.

In addition to complete or incomplete rotator cuff tears, swelling and inflammation as a result of bursitis, scapulothoracic dyskinesias, abnormalities of the margin of the acromion, and occasionally osteoarthritis in the acromioclavicular joint lead to impingement with a painful arc.

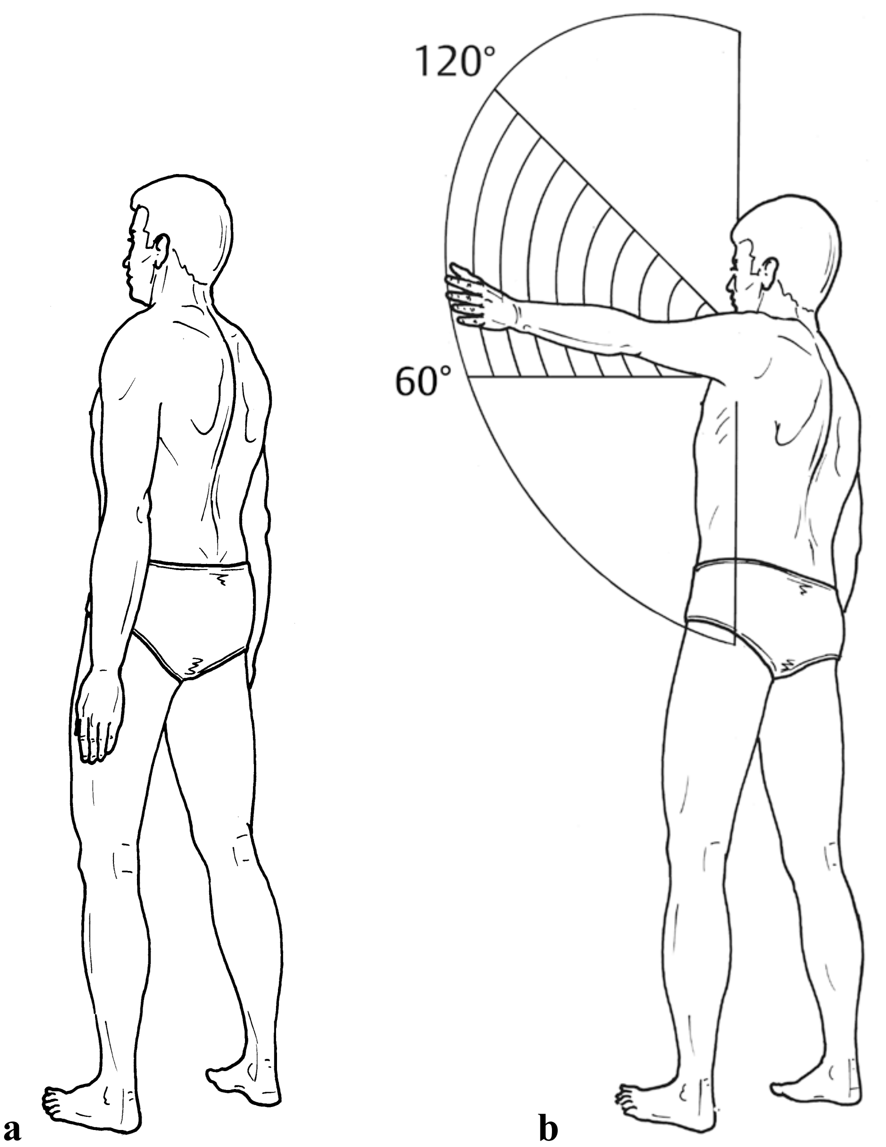


Fig. 2.29 Painful arc I. **(a)** Starting position. **(b)** Painful motion between 60 and 120°.

Neer Impingement Sign

□ **Procedure:** The examiner immobilizes the scapula with one hand while the other hand jerks the patient's arm forward, upward, and sideways (medially) into the scapular plane.

□ **Assessment:** If an impingement syndrome is present, subacromial constriction or impingement of the diseased area against the anterior inferior margin of the acromion will produce severe pain with motion.

This test is nonspecific. A variety of other conditions can cause positive test results, including bursitis, limited shoulder mobility, anterior instability of the shoulder, calcific tendinitis, bone tumors, and rotator cuff tears.

If the test is positive when done with the arm externally rotated, acromioclavicular joint dysfunction may be the cause.

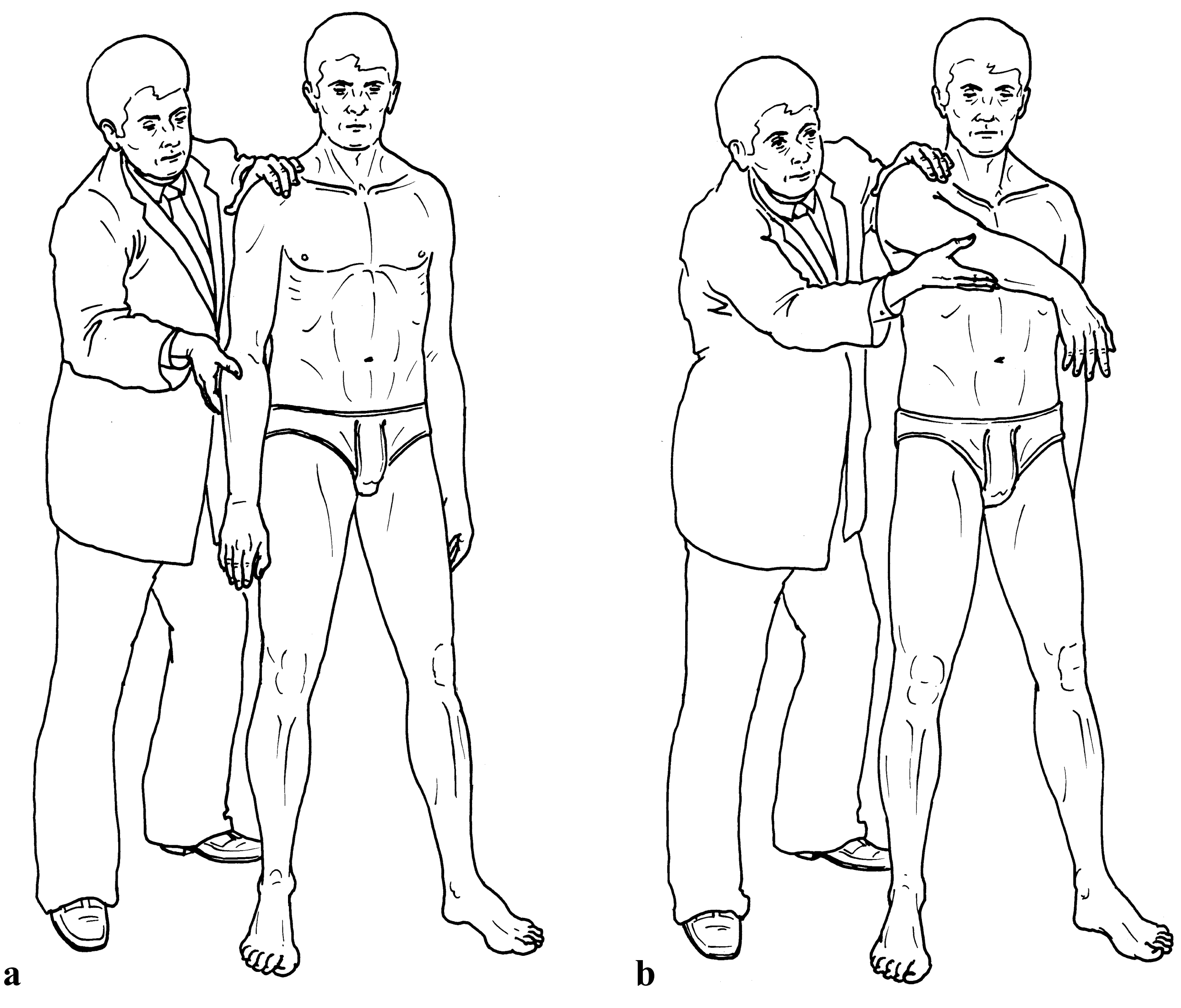


Fig. 2.30 Neer impingement sign. **(a)** Starting position. **(b)** Forcible forward flexion and adduction of the extended arm.

Internal Rotation Resistance Strength Test (IRRST)

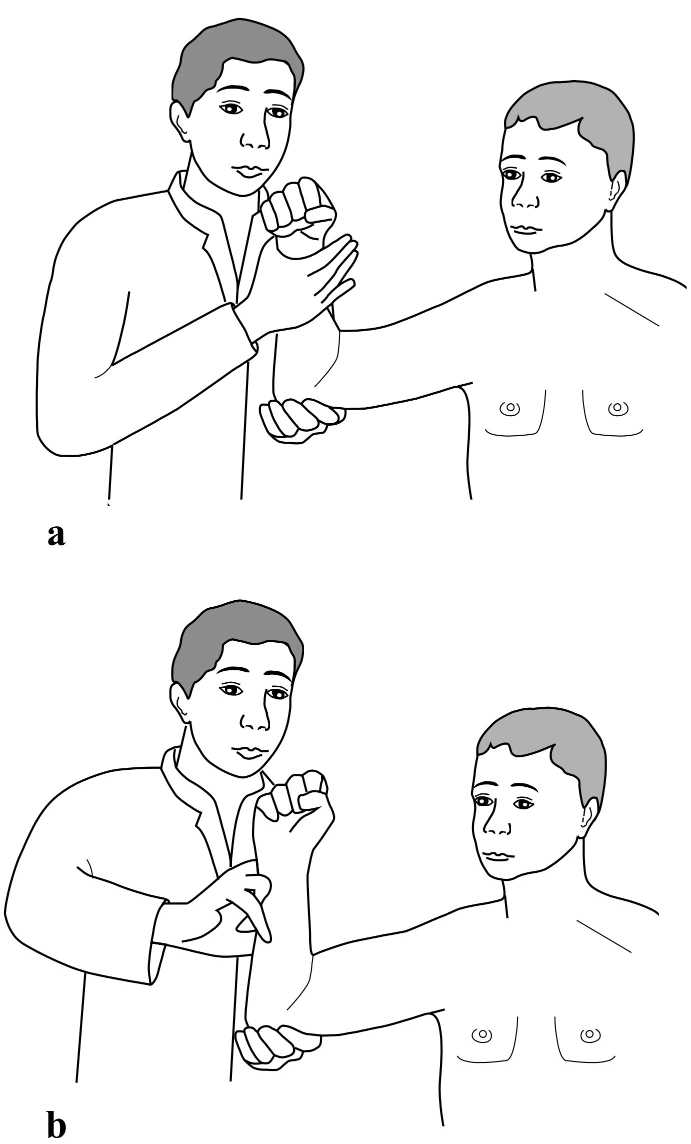
This test is a supplement to a positive impingement test to differentiate an outlet impingement in the classical sense from a non-outlet impingement as a result of intra-articular pathology.

Procedure The examiner stands behind the patient. The patient places his or her arm at 90° abduction and 80° external rotation with the elbow flexed and is then asked to rotate the arm powerfully first internally and then externally against the examiner's resistance.

Assessment: If the patient with a positive impingement test shows a clearly diminished strength of internal rotation in comparison to external rotation, then the test is considered positive, indicating a non-outlet impingement. If the patient shows weakness during external rotation, that indicates a classic outlet impingement instead.

Note: A classic outlet impingement (primary impingement) is defined as a true constriction caused by subacromial spurs or hypertrophic acromioclavicular joint arthritis.

A non-outlet impingement (secondary impingement) is a functional narrowing due to an increase in volume of the subacromial structures, usually inflammatory or posttraumatic in origin.



a

b

Fig. 2.31 Internal rotation resistance strength test (IRRST).

Hawkins-Kennedy Impingement Test

□ **Procedure:** From a position with the arm flexed at 90° and the elbow also bent, the examiner exerts a forcible, passive internal rotation.

□ **Assessment:** Pain suggests tendinitis or calcification in the supraspinatus tendon or secondary impingement, caused by anterior acromial margin changes, for example.

In a positive impingement syndrome, impingement of the greater tubercle or compression of the supraspinatus tendon occurs, causing severe pain on motion. Coracoid impingement is revealed when the arm is further adducted and the supraspinatus tendon also impinges against the coracoid process.

Jobe introduced a variation of the impingement test in which the adducted arm is internally rotated, causing primarily the posterior portion of the supraspinatus tendon to become wedged under the coracoacromial arch.

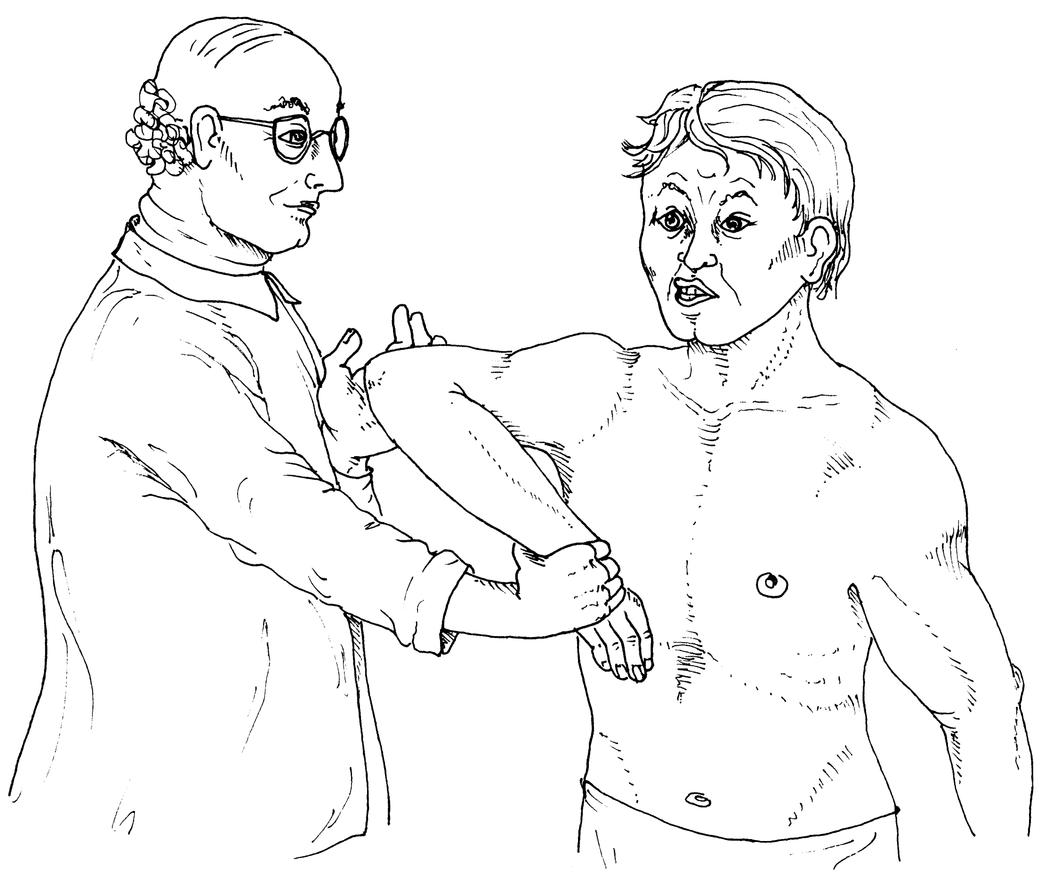


Fig. 2.32 Hawkins-Kennedy impingement test.

Neer Impingement Injection Test

□ **Procedure:** Under sterile conditions, the region beneath the anterior acromion (subacromial space) is infiltrated, preferable intrabursally, with a local anesthetic and a corticosteroid when indicated.

To open the subacromial space, the patient is asked to let the arm relax and hang down loosely. The weight of the arm opens the subacromial space, but only if the patient is sufficiently relaxed. After the injection, the active range of movement and the painful provocation tests are retested and evaluated.

□ **Caution:** After an injection it is necessary to observe the patient for some time to allow one to recognize and treat medication-related cardiovascular reactions in a timely fashion. The patient must be warned that the shoulder can be temporarily quite painful, and movement may be limited by pain after an injection. In addition, inflammatory, nonbacterial changes in the shoulder can develop after an injection, especially with a steroid.

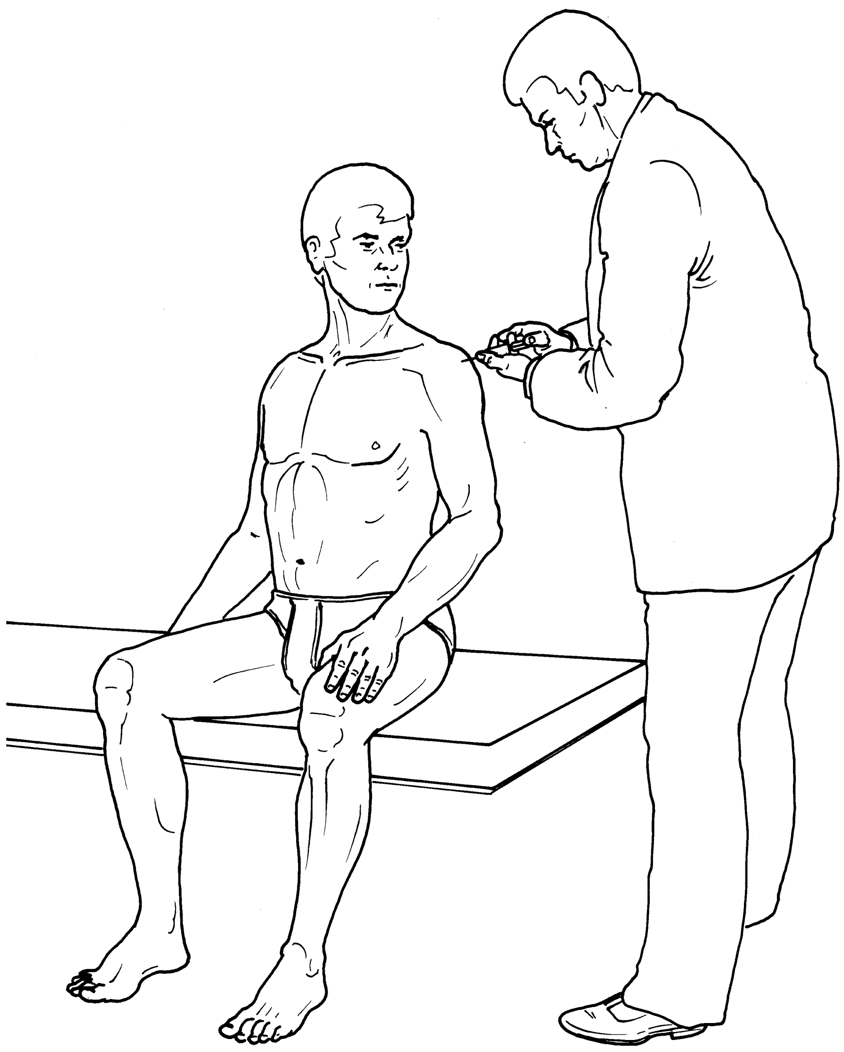


Fig. 2.33 Neer impingement injection test.

Acromioclavicular Joint

The acromial end of the clavicle articulates with the acromion. The acromioclavicular ligament reinforces the capsule of this joint. Another strong ligament joins the scapula and clavicle—the coracoclavicular ligament. It arises from the coracoid process and inserts into the inferior aspect of the clavicle.

One of the most helpful signs of the presence of acromioclavicular joint problems is to compare the two shoulders for asymmetry of the acromioclavicular joints (e.g., asymptomatic arthritis, trauma, tumors, infections, synovial cysts). Osteoarthritis of the acromioclavicular joint can cause pain and lead to further constriction of the subacromial space. In addition to pain with motion and tenderness to palpation over the shoulder, palpation will often reveal thickening of the bony joint margins. The vast majority of degenerative acromioclavicular joints are not symptomatic and do not warrant treatment.

Acromioclavicular joint disease produces findings that can mimic other diagnoses, such as cervical spine disease (which frequently radiates down the trapezius muscle into the superior shoulder), a superior labral anterior and posterior (SLAP) lesion, or a rotator cuff tear.

Acromioclavicular capsular ligament injures are common. Rockwood classified acromioclavicular joint injuries into six grades:

- **Grade 1:** Acromioclavicular joint sprain.
- **Grade 2:** Partial rupture of the acromioclavicular and coracoclavicular ligaments and subluxation in the acromioclavicular joint.
- **Grade 3:** Complete rupture of the acromioclavicular and coracoclavicular ligaments; dislocation of the acromioclavicular joint.
- **Grade 4:** Complete rupture of the acromioclavicular and coracoclavicular ligaments and the deltotrapezial fascia. Dislocation of the acromioclavicular joint; the clavicle is posteriorly displaced into the trapezius muscle.
- **Grade 5:** Complete rupture of the acromioclavicular and coracoclavicular ligaments and the deltotrapezial fascia. Dislocation of the acromioclavicular joint; the clavicle is superiorly displaced by at least twice the width of the clavicle.
- **Grade 6:** Complete rupture of the acromioclavicular and coracoclavicular ligaments and the deltotrapezial fascia. Dislocation of the acromioclavicular joint; the clavicle is inferiorly displaced beneath the coracoid process.

Painful Arc II

□ **Procedure:** The patient's arm is passively and actively abducted from the rest position alongside the trunk.

□ **Assessment:** Pain in the acromioclavicular joint occurs between 140 and 180° of abduction. Increasing abduction leads to increasing compression and torsion in the joint. (In an impingement syndrome or a rotator cuff tear, by comparison, pain symptoms will occur between 70 and 120°).

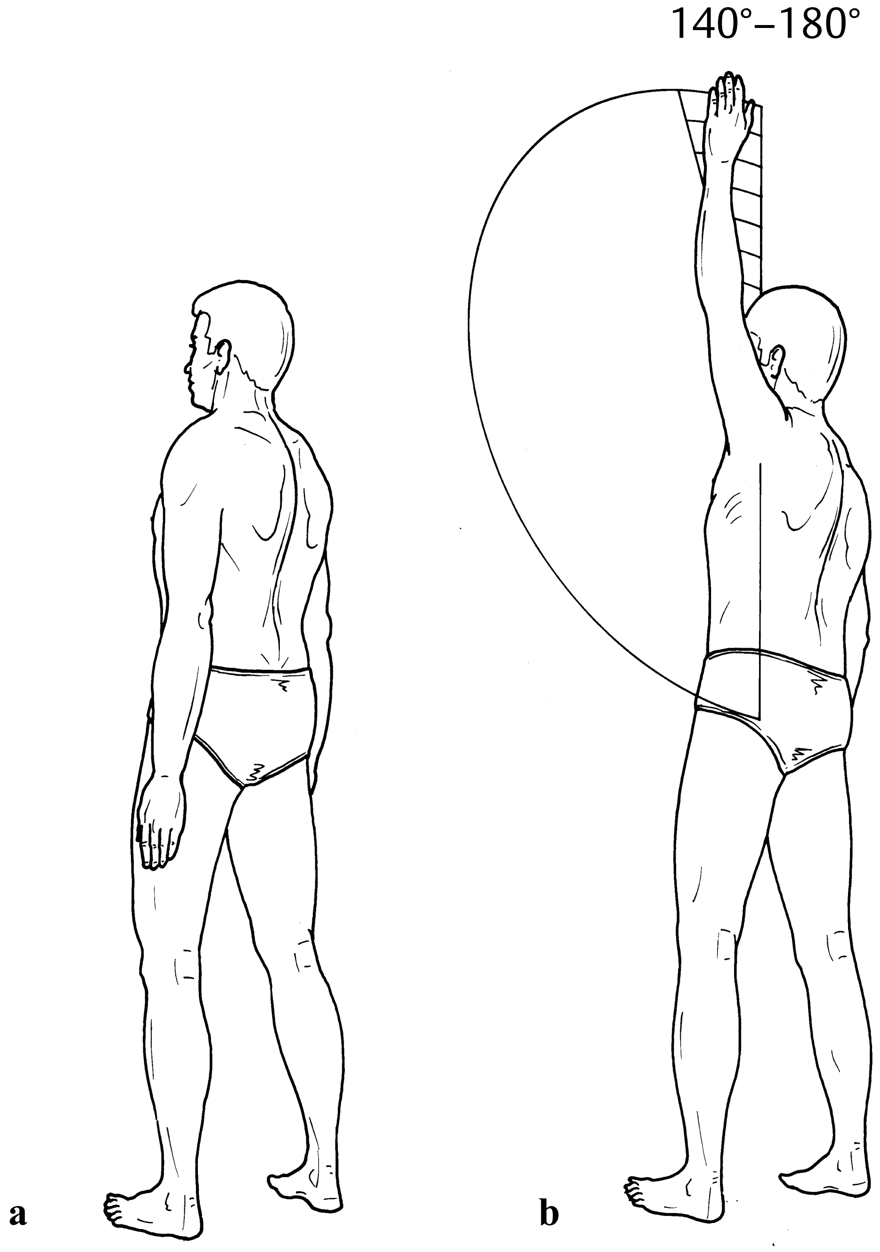


Fig. 2.34 Painful arc II. **(a)** Starting position. **(b)** Pain between 140 and 180° (sign of osteoarthritis in the acromioclavicular joint).

Forced Adduction Test on Hanging Arm

□ **Procedure:** The examiner grasps the upper arm of the affected side with one hand while the other hand rests on the contralateral shoulder and immobilizes the shoulder girdle. Then the examiner forcibly adducts the hanging affected arm behind the patient's back against the patient's resistance.

□ **Assessment:** Pain across the anterior aspect of the shoulder suggests acromioclavicular joint disease or subacromial impingement. (Symptoms that disappear or improve following injection of an anesthetic indicate that the acromioclavicular joint is causing the pain.)

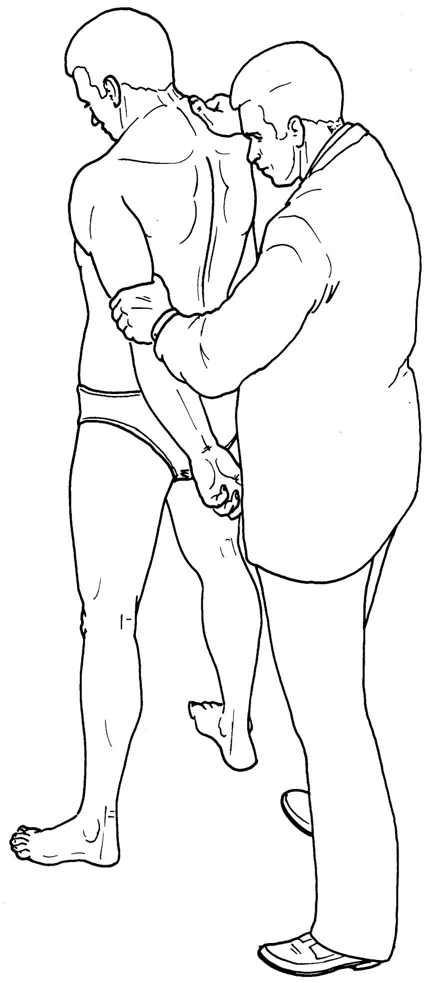


Fig. 2.35 Forced adduction test on hanging arm.

Fig. 2.36 Clavicle mobility test.

Clavicle Mobility Test

□ **Procedure:** The examiner grasps the lateral end of the clavicle between two fingers and moves it in all directions.

□ **Assessment:** Increased mobility of the lateral clavicle with or without pain is a sign of instability in the acromioclavicular joint. In isolated osteoarthritis there will be circumscribed tenderness to palpation and pain with motion. Acromioclavicular joint separation with rupture of the coracoclavicular ligaments will be accompanied by a positive "piano key" sign: the subluxated lateral end of the clavicle displaces proximally with the pull of the cervical musculature and can be pressed inferiorly against elastic resistance.

Dugas Test

□ **Procedure:** The patient is seated or standing and touches the contralateral shoulder with the hand of the 90°-flexed arm of the affected side.
□ **Assessment:** Acromioclavicular joint pain suggests joint disease (osteoarthritis, instability, disk injury, or infection). A differential diagnosis must exclude anterior subacromial impingement, due to the topographic proximity of that region.

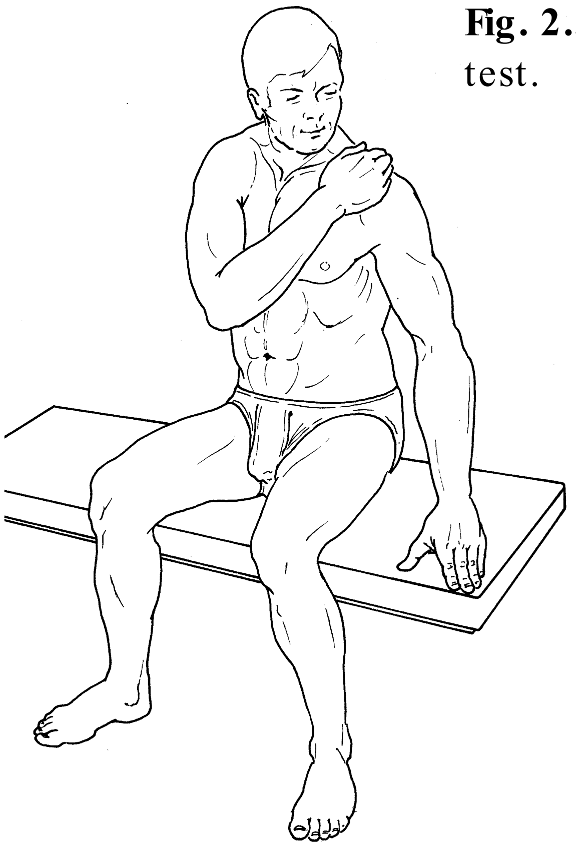


Fig. 2.37 Dugas test.

Fig. 2.38 Cross-body adduction stress test.

Cross-body Adduction Stress Test

□ **Procedure:** The 90°-abducted arm on the affected side is forcibly adducted across the chest toward the normal side.
□ **Assessment:** Pain in the acromioclavicular joint suggests joint pathology, anterior impingement, or suprascapular nerve entrapment syndrome. (Absence of pain after injection of an anesthetic is a sign of joint disease.)
□ **Note:** Dull, deep-seated pain over the superior scapular margin in the supraspinous fossa and posterolaterally on the scapula with radiation into the upper arm can be an indication of compression of the suprascapular nerve under the transverse scapular ligament by distal displacement of the scapula (**Thomson and Kopell horizontal flexion test**).

Acromioclavicular Injection Test

□ **Procedure:** Inject the acromioclavicular joint with an anesthetic such as lidocaine (with a corticosteroid where indicated) using a proximal approach. The injection must be performed under sterile conditions. Large osteophytes, arthritic joints, or a defective meniscus may render the injection into the anatomically narrow acromioclavicular joint space impossible.

□ **Assessment:** If the injection relieves local pain, at least temporarily, this indicates that acromioclavicular pathology is present. To confirm the diagnosis it is recommended, while anesthesia persists, to attempt to reproduce the pain with whichever examination produced the most pain prior to injection, such as the cross-body adduction stress test or painful arc test.

Long Head of the Biceps Tendon

A rupture of the long head of the biceps tendon will appear as a distally displaced protrusion of the muscle belly of the biceps. The close anatomical proximity of the intra-articular portion of the tendon to the coracoacromial arch predisposes it to involvement in degenerative processes in the subacromial space. A rotator cuff tear is often accompanied by a rupture of the long head of the biceps tendon.

Isolated inflammation of the long head of the biceps tendon (bicipital tenosynovitis) is accordingly rare. In younger patients, this may occur as a tennis or throwing injury. Subluxations of the long head of the biceps tendon in the bicipital groove are usually difficult to detect. However, a series of specific tests can be used to diagnose biceps tendon injuries; the typical sign of these injuries is not the distally displaced muscle belly but incomplete contraction and/or "snapping" of the tendon.

Nonspecific Biceps Tendon Test

□ **Procedure:** The patient holds the arm abducted in neutral rotation with the elbow flexed 90°. The examiner immobilizes the patient's elbow with one hand and places the heel of the other hand on the patient's distal forearm. The patient is then asked to externally rotate his or her arm against the resistance of the examiner's hand.

□ **Assessment:** Pain in the bicipital groove or at the insertion of the biceps suggests a tendon disorder.

Pain in the anterolateral aspect of the shoulder is often a sign of a disorder of the rotator cuff, especially the infraspinatus tendon.

Abbott-Saunders Test

Demonstrates subluxation of the long head of the biceps tendon in the bicipital groove.

□ **Procedure:** The patient's arm is externally rotated and abducted about 120° with progressive internal rotation. The examiner slowly lowers the arm from this position. The examiner guides this motion of the patient's arm with one hand while resting the other on the patient's shoulder and palpating the bi-cipital groove with the index and middle fingers.

□ **Assessment:** Pain in the region of the bicipital groove or a palpable or audible snap suggest a disorder of the biceps tendon (subluxation sign). An inflamed bursa (subcoracoid or subscapular bursa) can also occasionally cause snapping.

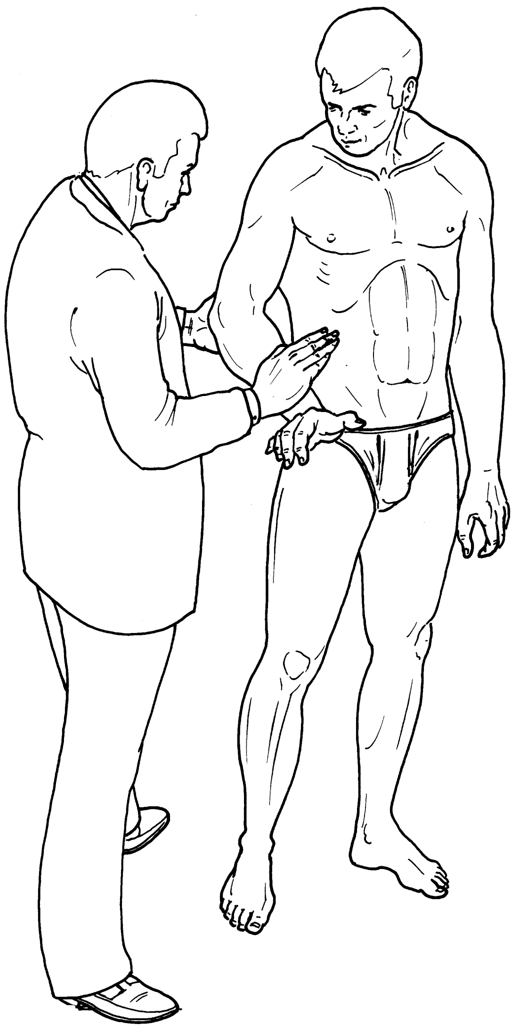


Fig. 2.39 Nonspecific biceps tendon test.

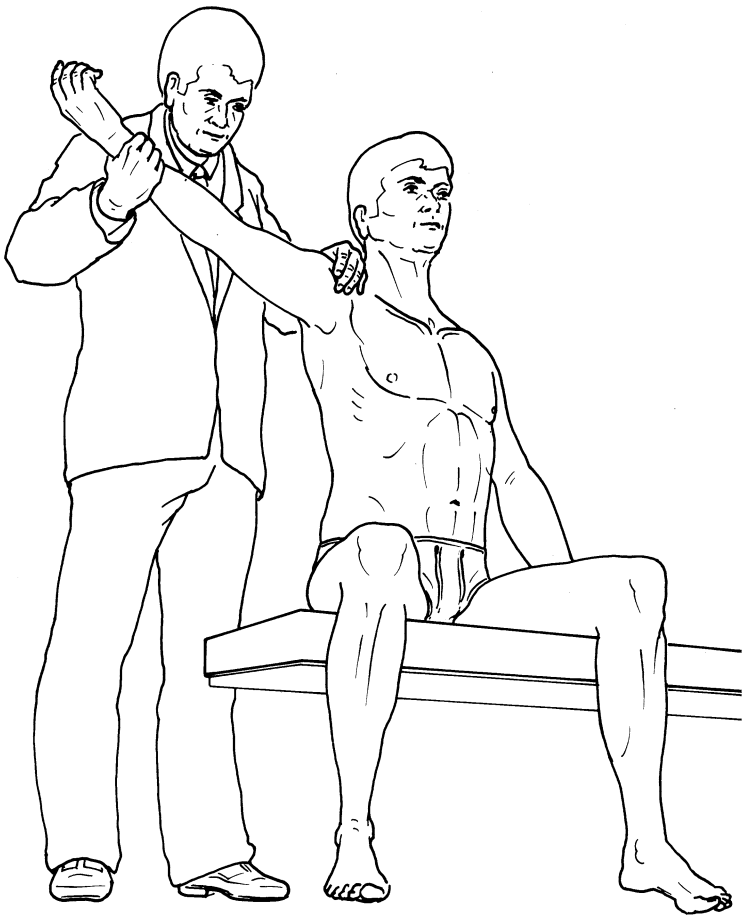


Fig. 2.40 Abbott–Saunders test.

2

Palm-Up Test (Speed Biceps or Straight Arm Test)

□ **Procedure:** The patient's arm is extended in supination at 90° of abduction and 30° of horizontal flexion. The patient attempts to either maintain this position or continue to abduct and pronate the arm against the downward pressure of the examiner's hand.
□ **Assessment:** A side-to-side difference in the strength of abduction with pain in the bicipital groove region suggests pathology in the long head of the biceps such as tenosynovitis or subluxation.

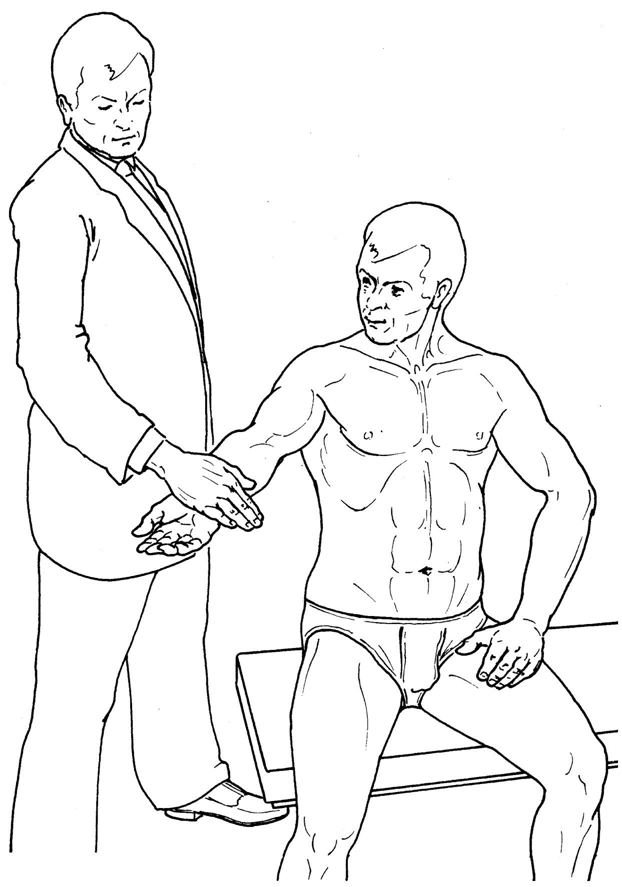


Fig. 2.41 Palm-up test.

Snap Test

Tests for subluxation of the long head of the biceps tendon.
□ **Procedure:** The examiner palpates the bicipital groove with the index and middle finger of one hand. With the other hand, the examiner grasps the wrist of the patient's arm (abducted 80 to 90° and flexed 90° at the elbow) and passively rotates it at the shoulder, first in one direction and then in the other.
□ **Assessment:** Subluxation of the long head of the biceps tendon out of the bicipital groove will be detectable as a palpable snap.

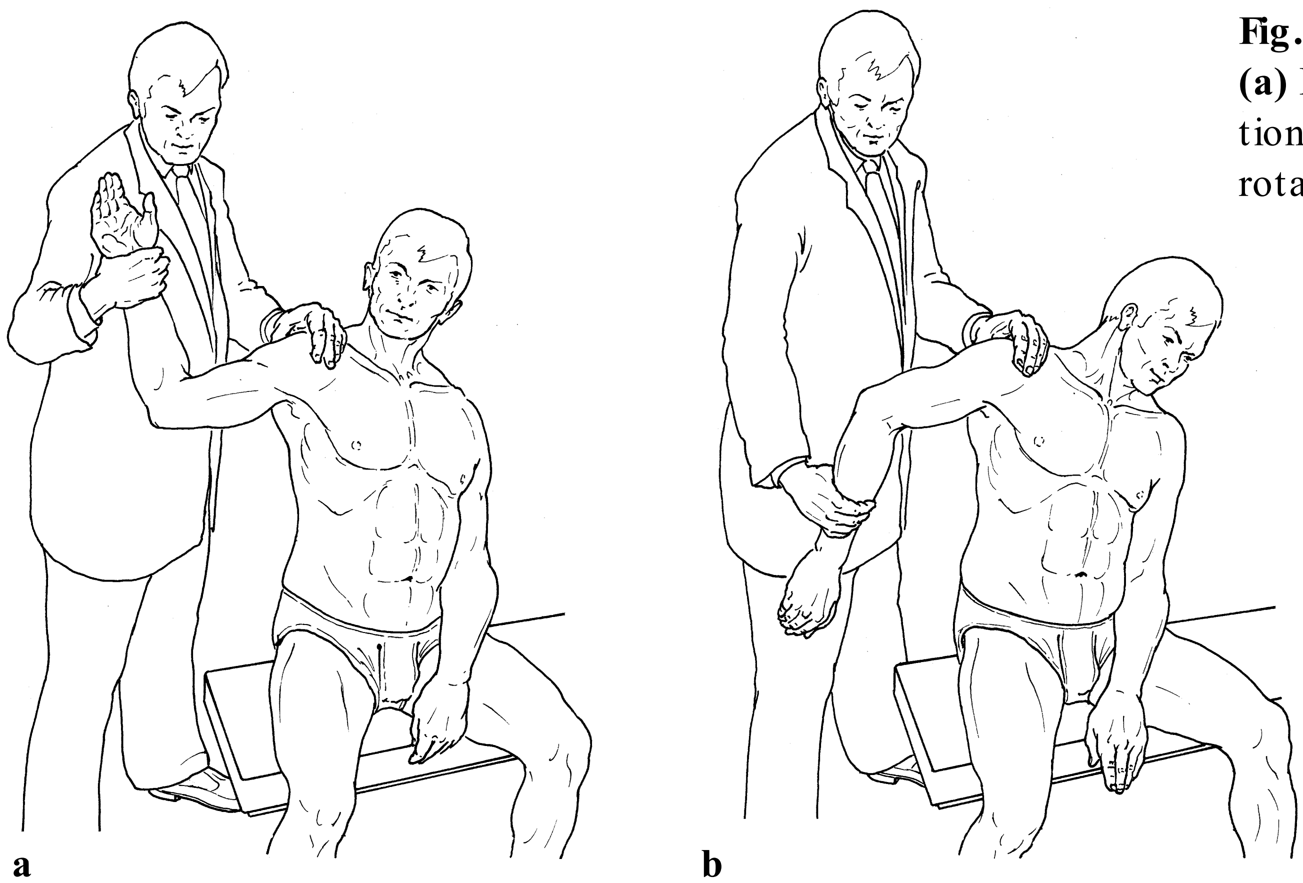


Fig. 2.42 Snap test. **(a)** External rotation. **(b)** Internal rotation.

Yergason Test

Functional test of the long head of the biceps tendon.

□ **Procedure:** The patient's arm is alongside the trunk and flexed 90° at the elbow. One of the examiner's hands stabilizes the elbow while the other hand grasps the patient's forearm as if to shake hands. The patient is asked to supinate the forearm against the examiner's resistance. This places isolated tension on the long head of the biceps tendon.

□ **Assessment:** Pain in the bicipital groove is a sign of a lesion of the biceps tendon, its tendon sheath, or its ligamentous connection via the transverse ligament. The typical provoked pain can be increased by pressing on the tendon in the bicipital groove.

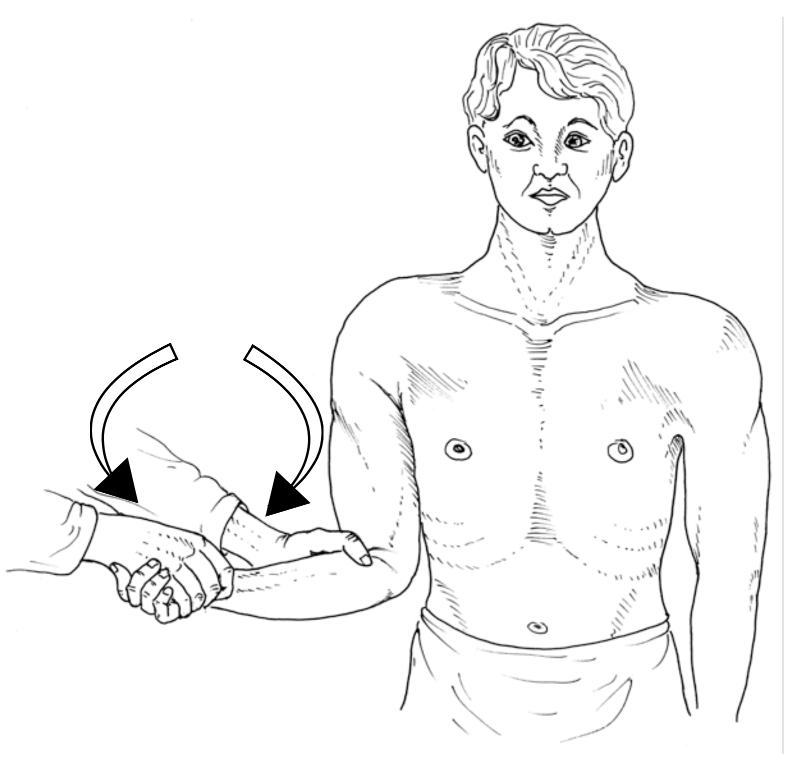


Fig. 2.43 Yergason test.

Hueter Sign

□ **Procedure:** The patient is seated with the arm extended at the elbow and the forearm in supination. The examiner grasps the posterior aspect of the patient's forearm. The patient is then asked to flex the elbow against the resistance of the examiner's hand.

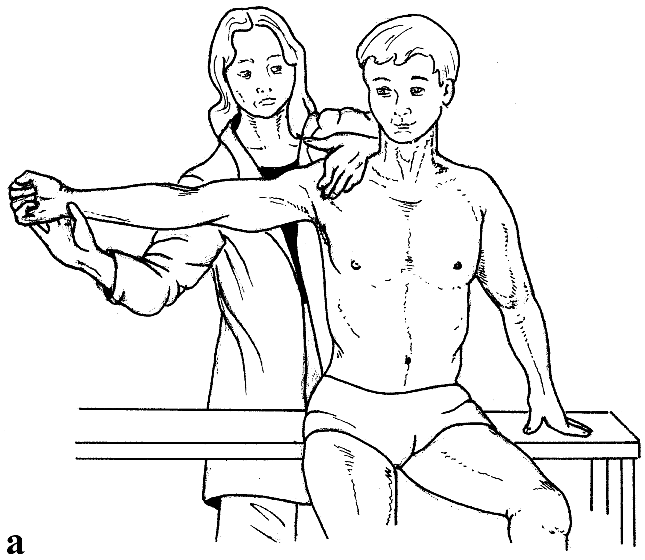
□ **Assessment:** In a rupture of the long head of the biceps tendon, the distally displaced muscle belly can be observed as a "ball" directly proximal to the elbow when the upper arm muscles contract.

Fig. 2.44 Hueter sign.

Transverse Humeral Ligament Test

□ **Procedure:** The patient is seated with the arm abducted 90°, internally rotated, and extended at the elbow. From this position, the examiner externally rotates the arm while palpating the bicipital groove to verify whether the tendon snaps.

□ **Assessment:** In the presence of ligamentous insufficiency, this motion will cause the biceps tendon to spontaneously displace out of the bicipital groove. Pain reported without displacement suggests biceps tendinitis.



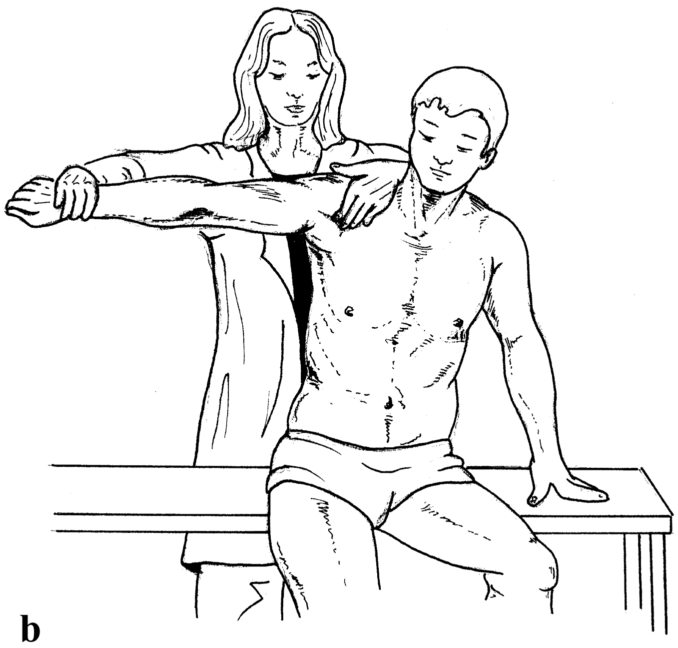


Fig. 2.45 Transverse humeral ligament test. **(a)** Starting position. **(b)** Palpating the biceps tendon in internal rotation.

Ludington Test

Procedure: The patient sits or stands and is asked to place both hands behind the head, interlocking the fingers. In this position both arms are relaxed. The patient is then asked to alternately relax and contract the biceps muscle, while the examiner palpates the long head of the biceps tendon at the same time.

Assessment: In comparing the two sides, if the examiner notes tenderness or subluxation of the tendon, this suggests an unstable tendon, tendinitis, or even a defect of the transverse ligament.

Fig. 2.46 Ludington test.

Lippman Test

2

□ **Procedure:** The patient sits or stands and the examiner holds the arm and flexes it to 90°. The examiner now palpates the biceps tendon at the level of the bicipital groove, about 9 cm distal to the glenohumeral joint, and attempts to move it back and forth.
□ **Assessment:** The test is positive if the patient feels pain along the course of the biceps tendon during this examination.

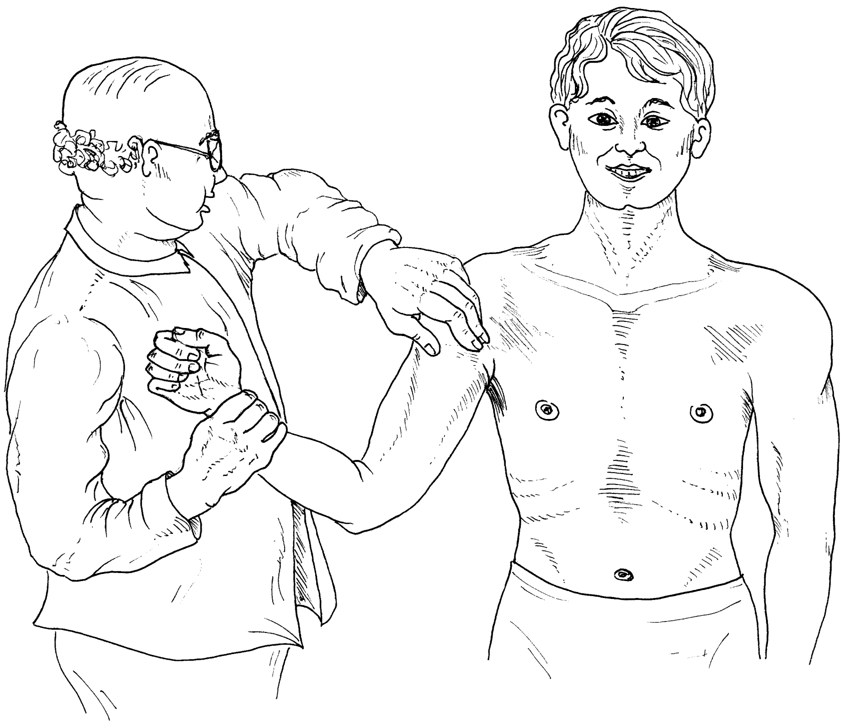


Fig. 2.47 Lippman test.

SLAP Lesions (Superior Labral Anterior/Posterior Lesion)

The labrum is a fibrocartilaginous ring, which, similar to the meniscus, enlarges the joint surfaces and deepens the socket. The intra-articular insertion of the long head of the biceps tendon forms an integral unit with the adjacent superior glenoid labrum. A fall on the extended, slightly flexed, and abducted arm; trauma in external rotation and abduction; microtrauma from repeated throwing motions—all of these can lead to superior labral anterior/posterior (SLAP) lesions.

Associated injuries are common and may include tears of the rotator cuff and Bankart lesions.

Snyder classifies SLAP lesions as follows:

- **Type I:** Degenerative changes of the labral margin without impact on the anchoring of the biceps tendon or the labrum (11%).

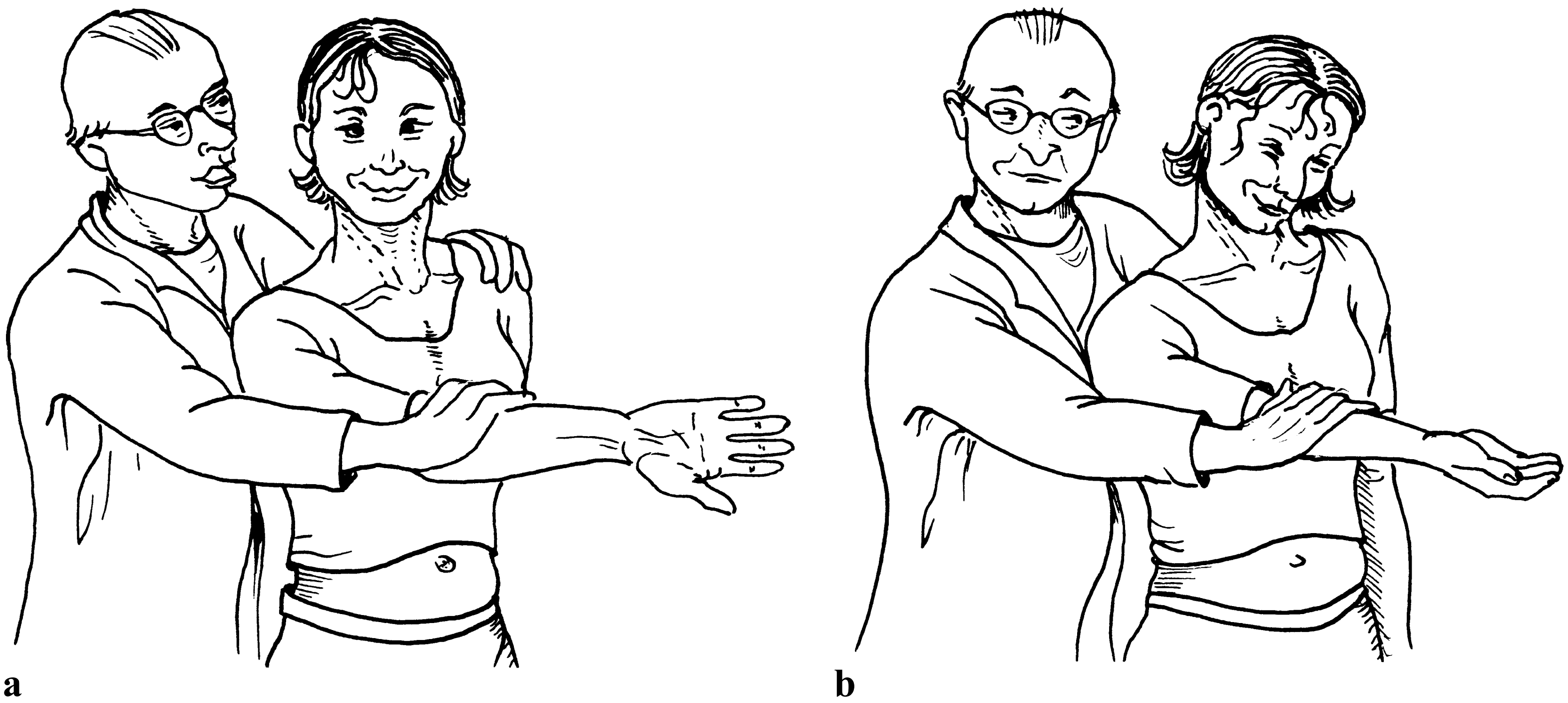
- **Type II:** Avulsion of the biceps tendon from the supraglenoid tubercle (41%). Biceps and labrum are avulsed together.
- **Type III:** Bucket-handle tear of the superior labrum with intact biceps anchor (33%).
- **Type IV:** Bucket-handle tear of the superior labrum involving the biceps anchor (15%).

O'Brien Active Compression Test

Evaluation of a SLAP lesion.

□ **Procedure:** The patient stands with the elbow extended and moves his or her arm into 90° flexion, 10° adduction, and maximum internal rotation (thumbs pointing downward). The examiner attempts to press the arm downward against the patient's resistance. The test is then repeated in maximum external rotation.

□ **Assessment:** The test is positive where the first phase elicits pain that then lessens or disappears in supination (maximum external rotation). It is crucial to inquire about the location of the pain as the O'Brien test can also yield positive results in the presence of acromioclavicular joint disorders. Pain reported within the shoulder suggests a SLAP lesion, whereas pain over the acromioclavicular joint may also be due to osteoarthritis of the acromioclavicular joint.



a b

Fig. 2.48 O'Brien active compression test. **(a)** Elbow extended, arm 10° adducted, 90° flexed, and maximally internally rotated; **(b)** with extended elbow, 90° flexion, and maximal external rotation of the 10° adducted arm.

Stretch Test

□ **Procedure/Assessment:** Passive extension of the shoulder, extension of the elbow, and pronation of the forearm by the examiner leads to pain in the anterior deltoid muscle area along the course of the biceps tendon, as does the active attempt by the patient to reverse this position by supinating the forearm, flexing the elbow, or flexing the shoulder.

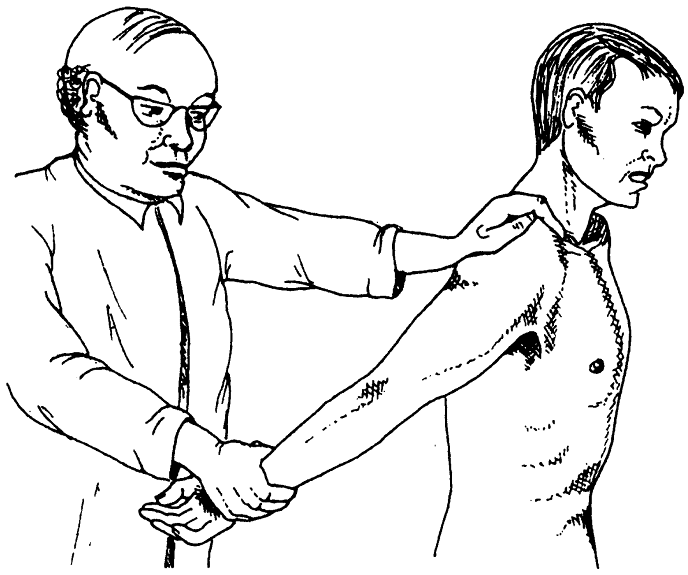


Fig. 2.49 Stretch test.

Biceps Load Test 1

Diagnosis of superior labral tears in patients who have anterior shoulder instability associated with Bankart lesions.

□ **Procedure:** The patient is placed supine with the arm abducted to 90 degrees. The elbow is flexed 90° and the arm is placed in a neutral rotation. The forearm is supinated and then an anterior apprehension maneuver is performed (external rotation of the arm).

If a feeling of instability or anterior shoulder pain occurs, external rotation is stopped. The patient is then asked to actively flex the forearm at the elbow against resistance by bringing the hand toward the face.

During this maneuver, the patient is asked whether the feeling of instability is improved, unchanged, or worsened.

□ **Assessment:** If the pain and feeling of instability improve, there is no indication of a SLAP lesion. If the symptoms are unimproved or even worsened, then there is suspicion of a SLAP lesion. The examiner should note that the forearm should be supinated during the test. Also, the examiner should be at the same level as the patient.

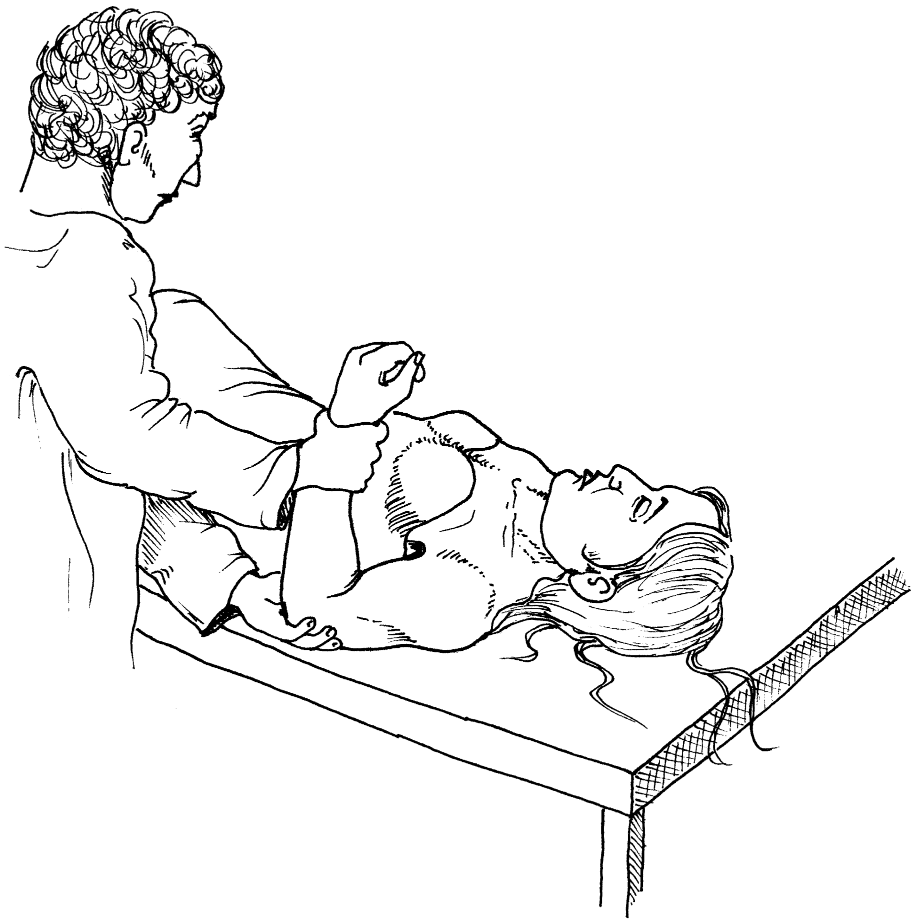


Fig. 2.50 Biceps load test 1.

Biceps Load Test 2

Test for isolated SLAP lesions independent of shoulder instability.

 Procedure: The patient is supine with the arm abducted to 120°. The arm is externally rotated to its maximal extent, the elbow flexed at 90° and the forearm supinated. The patient is then asked to flex the elbow further, trying to bring the hand to the head against the resistance of the examiner's hand.

 Assessment: There is suspicion of a SLAP lesion if the test triggers pain deep in the joint or along the joint line.

Fig. 2.51 Biceps load test 2.

Habermeyer Supine Flexion Resistance Test

Used to evaluate pathology in the area of the upper labrum–biceps tendon complex.

□ **Procedure:** The patient lies supine and elevates the arms maximally over the head so they rest on the examination table with the palms upward. The examiner stands alongside the patient on the side of the shoulder to be examined and holds the patient's arm just below the elbow. The patient is asked to raise the arm up from this position against the resistance of the examiner's hand.

□ **Assessment:** One must assume that a SLAP lesion is present if the patient reports pain deep in the shoulder joint or along the posterior joint line from the strain from the flexion (throwing movement) against resistance.

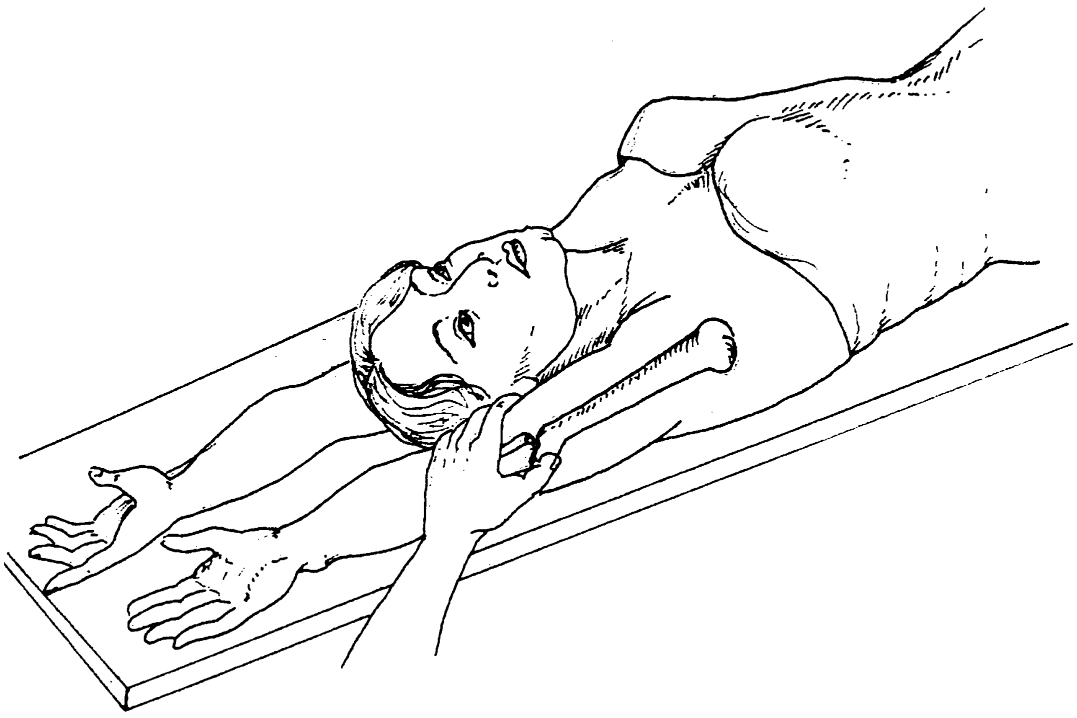


Fig. 2.52 Habermeyer supine flexion resistance test.

Shoulder Instability

The joint capsule of the shoulder may be too loose, leading to instability. Often this is attributable to congenital generalized laxity of the ligaments (hyperlaxity) with increased bilateral multidirectional instability. Chronic shoulder pain may be attributable to an unstable shoulder.

The clinical picture of subluxation in particular is often difficult to diagnose, and patients themselves can usually give only a vague description of their symptoms.

According to Neer, patients with instability invariably have a history of a period of intensive shoulder use (such as competitive sports), an episode of repeated minor trauma (overhead use), or generalized ligament laxity. Both young athletes and inactive persons are affected, men and women alike.

The transition between subluxation and dislocation is continuous. There is no clearly defined point before which a lesion is still a subluxation and beyond which it is considered to be fully dislocated. Patients with voluntary instability represent a separate issue.

The differential diagnosis must specifically consider an impingement syndrome, a rotator cuff tear, osteoarthritis in the acromioclavicular joint, and also a cervical spine syndrome. In cases of doubt, injection of a local anesthetic at the point of maximum pain may be required.

Signs of generalized ligament laxity may include increased mobility in other joints, particularly increased hyperextension in the elbow or retroflexion in the metacarpophalangeal joint of the thumb with the forearm extended.

The use of a variety of relatively specific tests will make it easier for the examiner to arrive at a diagnosis.

Assessment of the range of motion is crucial in patients with suspected shoulder instability. Rotation should be examined in both adduction and 90° abduction. Restricted external rotation in both adduction and abduction will often be the first sign of instability in patients with anterior instability. Flexion and abduction in the scapular plane are not normally restricted.

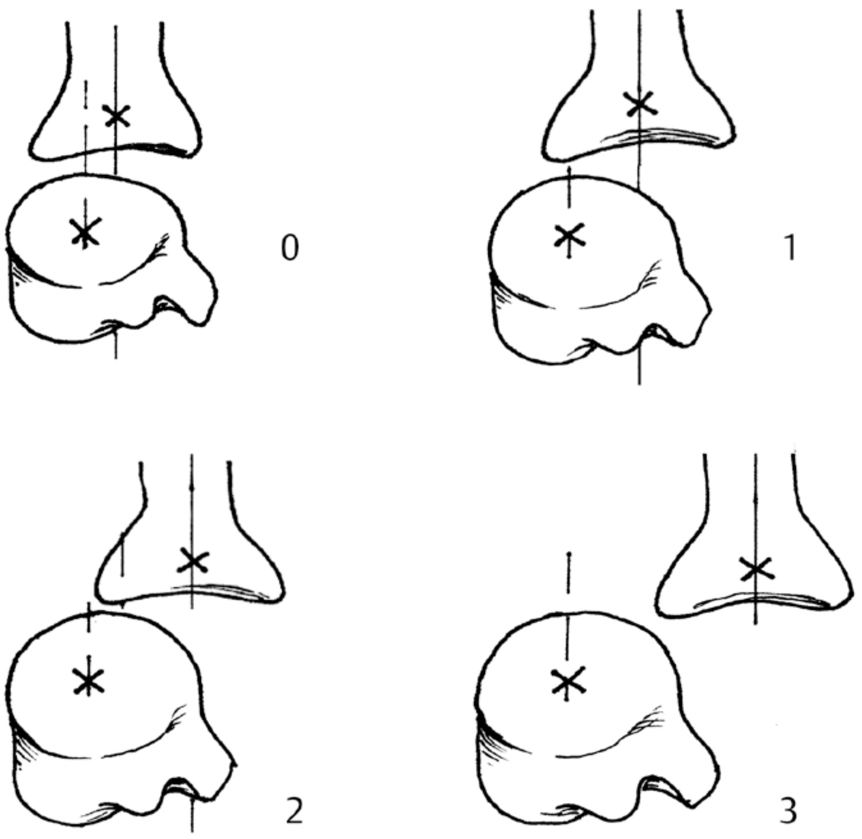


Fig. 2.53 Hawkins classification.

The anterior and posterior drawer tests enable one to determine the degree of instability (translation). Hawkins describes a four-grade classification of the degree of translation (**Fig. 2.53**):

- **Grade 0:** No translation of the humeral head (normal laxity).
- **Grade 1:** Humeral head moves slightly up to the glenoid rim.
- **Grade 2:** Translation of the head over the rim, but spontaneously reduces.
- **Grade 3:** Complete luxation without spontaneous relocation.

It is important to pose two questions to the patient for all the luxation tests:

1. Do you feel the increased shift (translation)?
2. Do you have this feeling in everyday life (in sports or work, for example)?

Gerber's classification (**Table 2.1**), in which six forms of shoulder instability are differentiated, is better suited for everyday clinical use. An expansion of this classification by Gerber and Nyfeller differentiates between statistical (Class A) and dynamic instability (Class B) as well as voluntary luxation (Class C).

The examination must be carried out comparing right and left sides to determine if instability present on examination is clinically symptomatic or just an individual physiologic hyperlaxity without clinical relevance.

Table 2.1 Gerber classification of shoulder instability (1997)

Type	Description
I	Chronic luxation
II	Unidirectional instability without hyperlaxity
III	Unidirectional instability with multidirectional hyperlaxity
IV	Multidirectional instability without hyperlaxity
V	Multidirectional instability with multidirectional hyperlaxity
VI	Unidirectional or multidirectional voluntary luxation

Compression Test

□ **Procedure and assessment:** Passive elevation of the arm to the end of its range of motion with continued application of posterior pressure produces pain as a result of compression of the biceps tendon between the acromion and humeral head.

Evaluation of the range of motion is crucial in patients with suspected shoulder instability. Rotation should be examined in both adduction and 90° abduction. Restricted external rotation in both adduction and abduction will often be the first sign of instability in patients with anterior instability. Flexion and abduction in the scapular plane are not normally restricted.

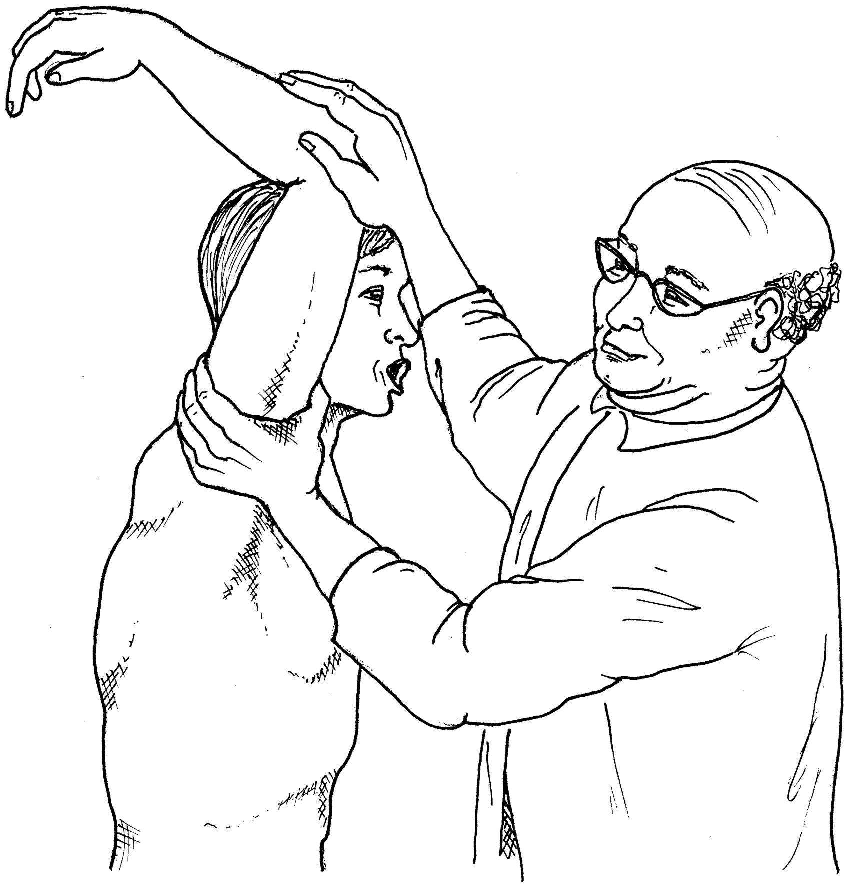


Fig. 2.54 Compression test.

Anterior Apprehension Test

Tests shoulder stability.

□ **Procedure:** The examination begins with the patient seated. The examiner palpates the humeral head through the surrounding soft tissue with one hand and guides the patient's arm with the other hand. The examiner passively abducts the patient's shoulder with the elbow flexed and then brings the shoulder into maximum external rotation, keeping the arm in this position. The test is performed at 60°, 90°, and 120° of abduction to evaluate the superior, medial, and inferior glenohumeral ligaments. With the guiding hand, the examiner presses the humeral head in an anterior and inferior direction.

This test can also be performed in the supine position with improved muscular relaxation. The shoulder lies on the edge of the examining table, which acts as a fulcrum. In this position the apprehension test can be initiated in various external-rotation and abduction positions. The healthy shoulder serves for comparison.

□ **Assessment:** Anterior shoulder pain with reflexive muscle tensing is a sign of an anterior instability syndrome. The patient has apprehension, the fear that the shoulder will dislocate. Prompted by pain, he or she tries to avoid the examiner's movement.

Even without pain, however, the tension of the anterior shoulder musculature (pectoralis) alone may be a sign of instability.

Placing the patient supine improves the specificity of the apprehension test. From the apprehension position, the examiner applies a posterior translational stress to the head of the humerus, thereby leading to a sudden decrease in pain and of the fear of dislocation (the humeral head reduces into the socket, and external rotation can be increased—**Jobe relocation test**).

In a further stage of the apprehension test, releasing the posteriorly directed pressure causes a sudden increase in pain with the apprehension phenomenon (**release test**).

□ **Note:** When the patient complains of sudden stabbing pain with simultaneous or subsequent paralyzing weakness in the affected extremity, this is referred to as the "dead arm sign." It is attributable to the transient compression the subluxated humeral head exerts on the plexus.

It is important to know that at 45° of abduction the test primarily evaluates the medial glenohumeral ligament and the subscapularis tendon. At or above 90° of abduction, the stabilizing effect of the subscapularis is neutralized and the test primarily evaluates the inferior glenohumeral ligament.

The test must be performed slowly and carefully to avoid the danger of causing the humeral head to dislocate.

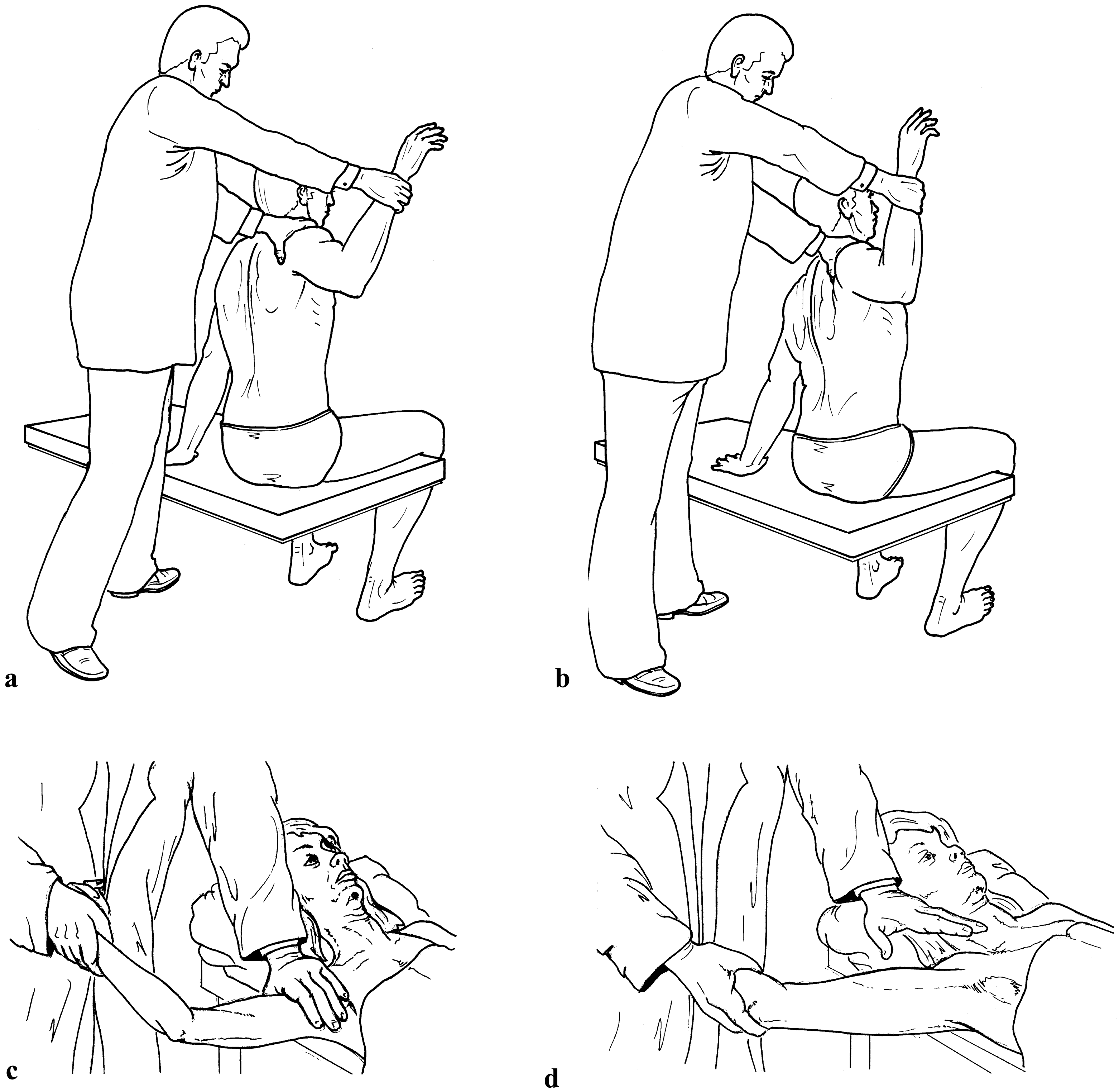


Fig. 2.55 Anterior apprehension test. **(a)** Starting position. **(b)** Test position. **(c)** Supine with posteriorly directed pressure applied to the humeral head (pain relieved); relocation test. **(d)** After releasing the posteriorly directed pressure (pain increases); release test.

Fulcrum Test

□ **Procedure:** The patient lies supine. The arm is abducted and externally rotated, and the elbow is flexed. The examiner exerts pressure from the posterior side to displace the humeral head anteriorly.

Stability should be tested at abductions of 60°, 90°, and 120°.

□ **Assessment:** A patient who has anterior instability expects pain the farther anteriorly the humeral head moves toward potential dislocation over the labrum, and reacts with evasive movement.

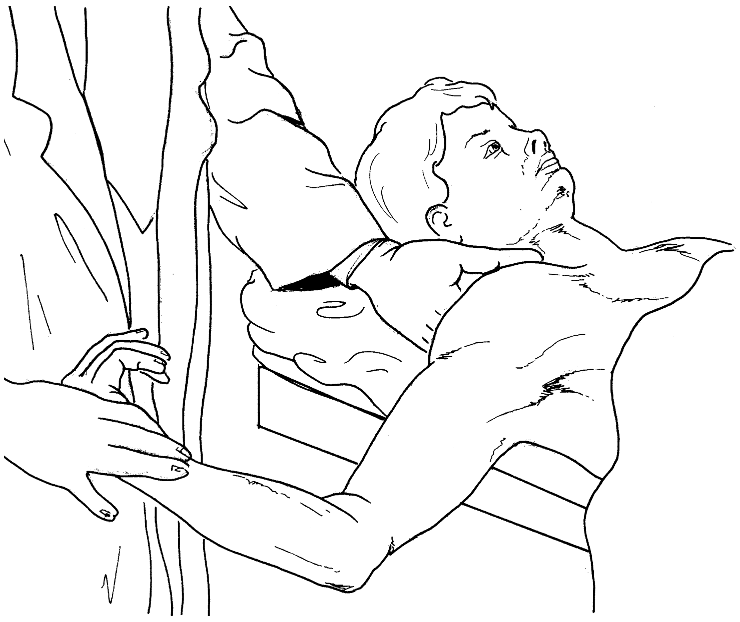


Fig. 2.56 Fulcrum test.

Throwing Test

□ **Procedure and assessment:** In the throwing test, the patient executes a rapid throwing motion against the examiner's resistance. This test can reveal anterior subluxation that occurs during the throwing motion.

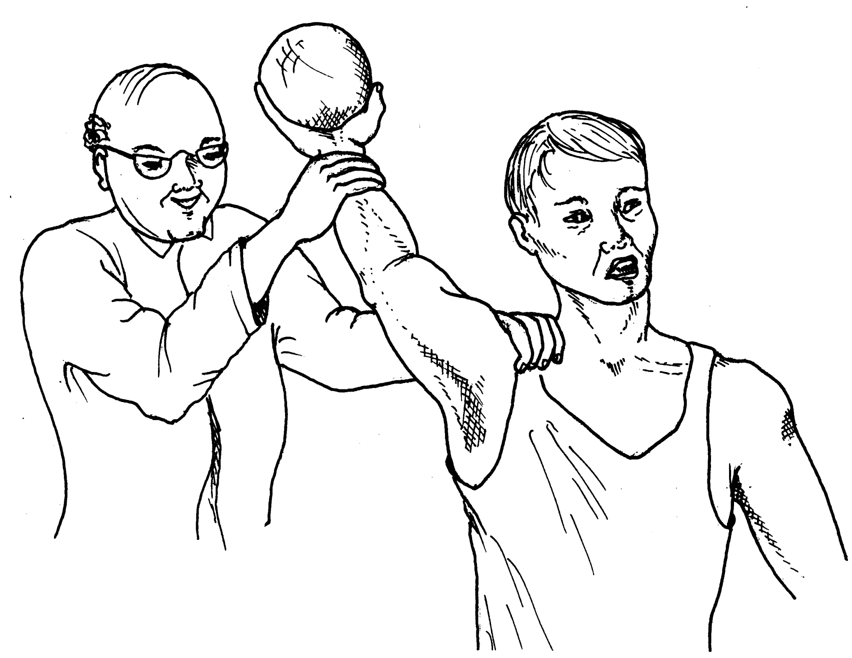


Fig. 2.57 Throwing test.

Leffert Test

□ **Procedure and assessment:** The Leffert test can be used to quantify a drawer phenomenon. Looking downward at the shoulder of the seated patient (craniocaudal view), the examiner displaces the humeral head anteriorly. The anterior displacement of the examiner's index finger in relation to the middle finger shows the degree of anterior translation of the humeral head.

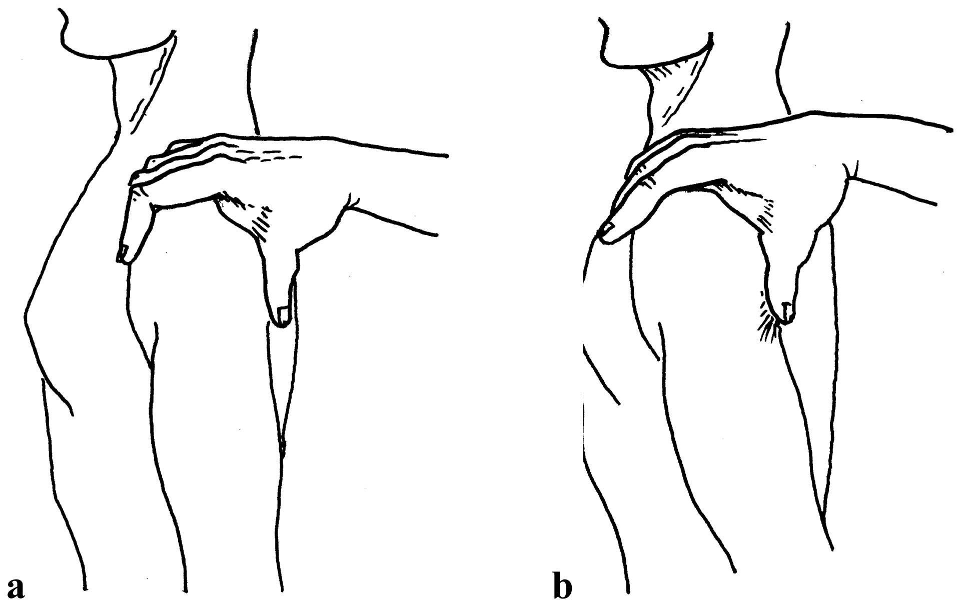


Fig. 2.58 Leffert test. **(a)** Starting position. **(b)** Index finger displaced anteriorly.

Load and Shift Test (Drawer Test)

□ **Procedure:** The patient is seated. The examiner stands behind the patient. To evaluate the right shoulder, the examiner grasps the patient's shoulder with the left hand to stabilize the clavicle and superior margin of the scapula while using the right hand to move the humeral head anteriorly and posteriorly.
□ **Assessment:** Significant anterior or posterior mobility of the humeral head with or without pain suggests instability.

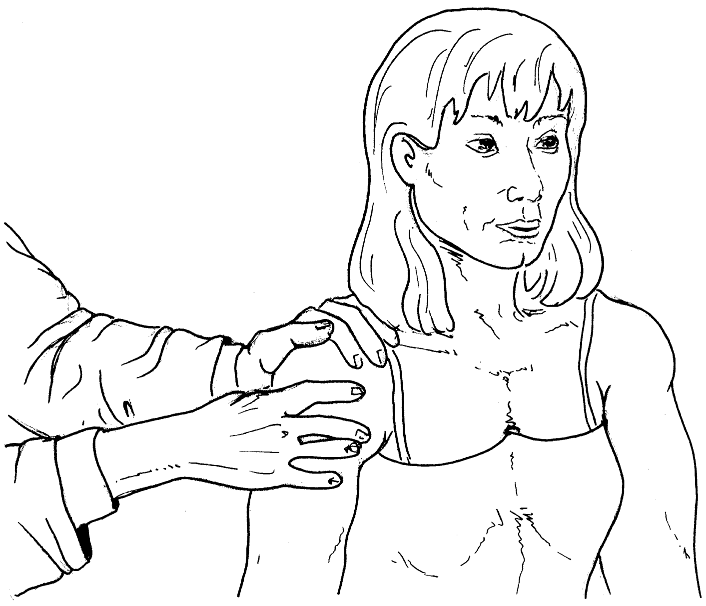


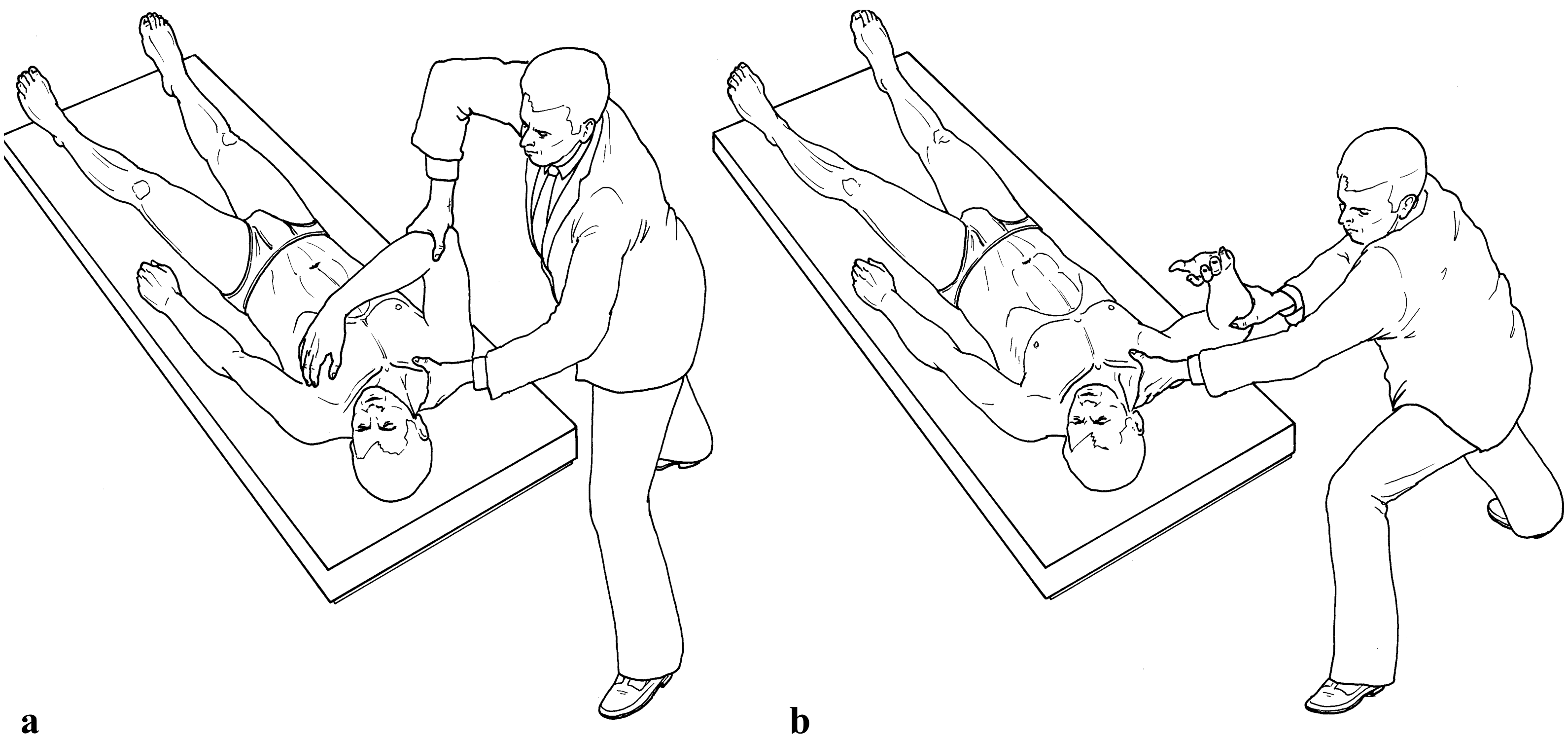
Fig. 2.59 Load and shift test.

Posterior Apprehension Test (Posterior Shift and Load Test)

□ **Procedure:** The examiner places one hand under the supine patient's scapula and grasps the elbow with the other. By pressing the abducted, horizontally flexed, and internally rotated arm posteriorly along the upper arm axis, the examiner attempts to provoke posterior subluxation of the humeral head.

□ **Assessment:** Sufficient laxity in the capsular ligaments will allow posterior subluxation of the humeral head, with associated pain.

Maintaining the axial pressure on the humeral head increasingly abducts and retracts the arm. The previously subluxated head can be reduced again with a readily palpable and audible click. (Caution: This test involves a certain risk of acute dislocation!)



a b

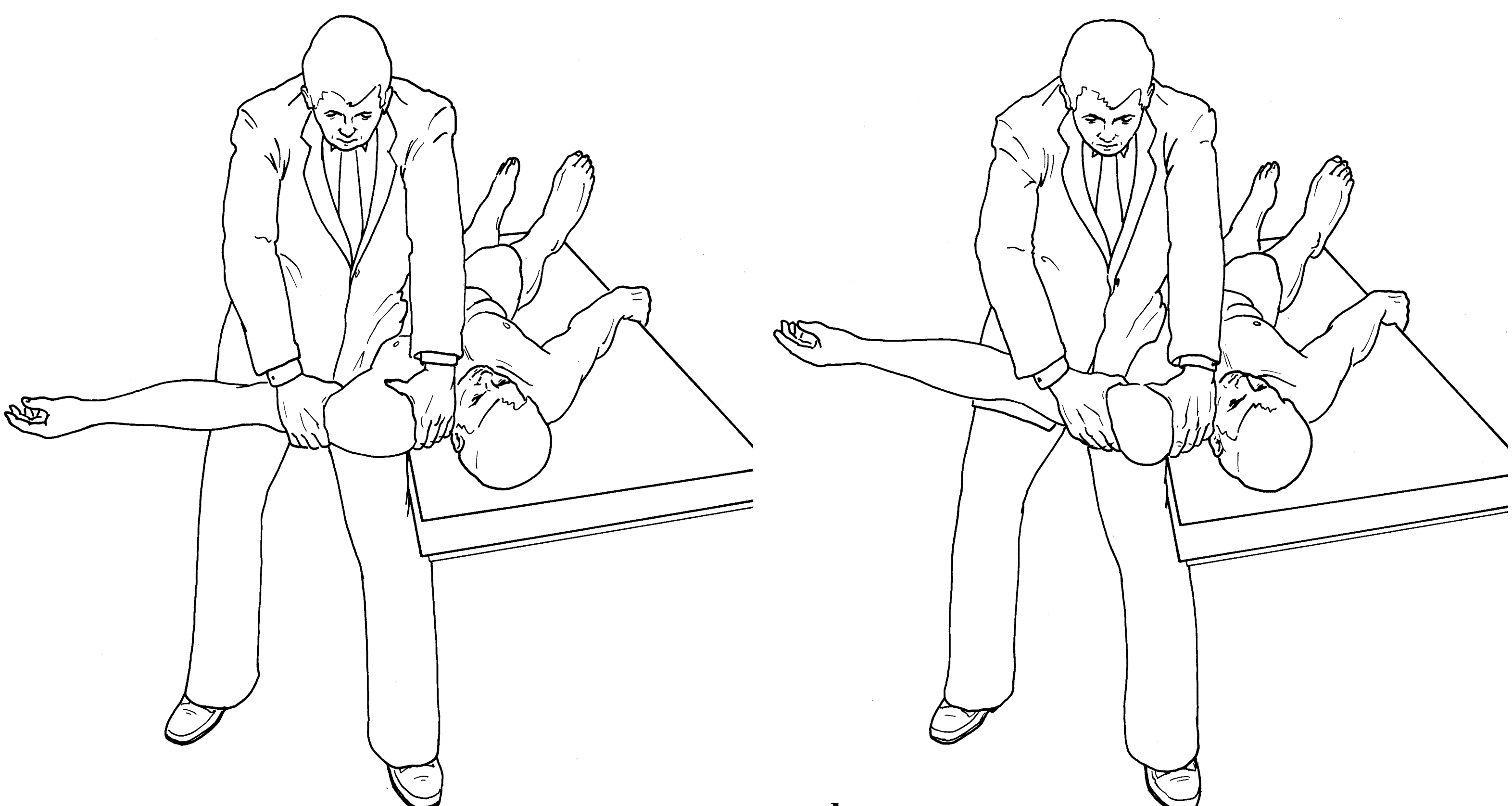
Fig. 2.60 Posterior apprehension test. **(a)** Starting position. **(b)** Reduction maneuver.

Gerber–Ganz Anterior Drawer Test

□ **Procedure:** The patient is supine with the affected shoulder positioned such that it projects slightly past the edge of the examining table. The affected shoulder is held in 80 to 120° of abduction, 0 to 20° of flexion, and 0 to 30° of external rotation as loosely and as painlessly as possible. The examiner immobilizes the scapula with the left hand (with the index and middle fingers on the scapular spine and the thumb on the coracoid). With the right hand, the examiner tightly grasps the patient's proximal upper arm and pulls it anteriorly in a manner similar to the Lachman test for anterior instability in the knee.

□ **Assessment:** The relative motion between the immobilized scapula and the anteriorly displaced humerus is a measure of anterior instability and can be specified in degrees.

Occasional audible clicking with or without pain can indicate an anterior labral defect.



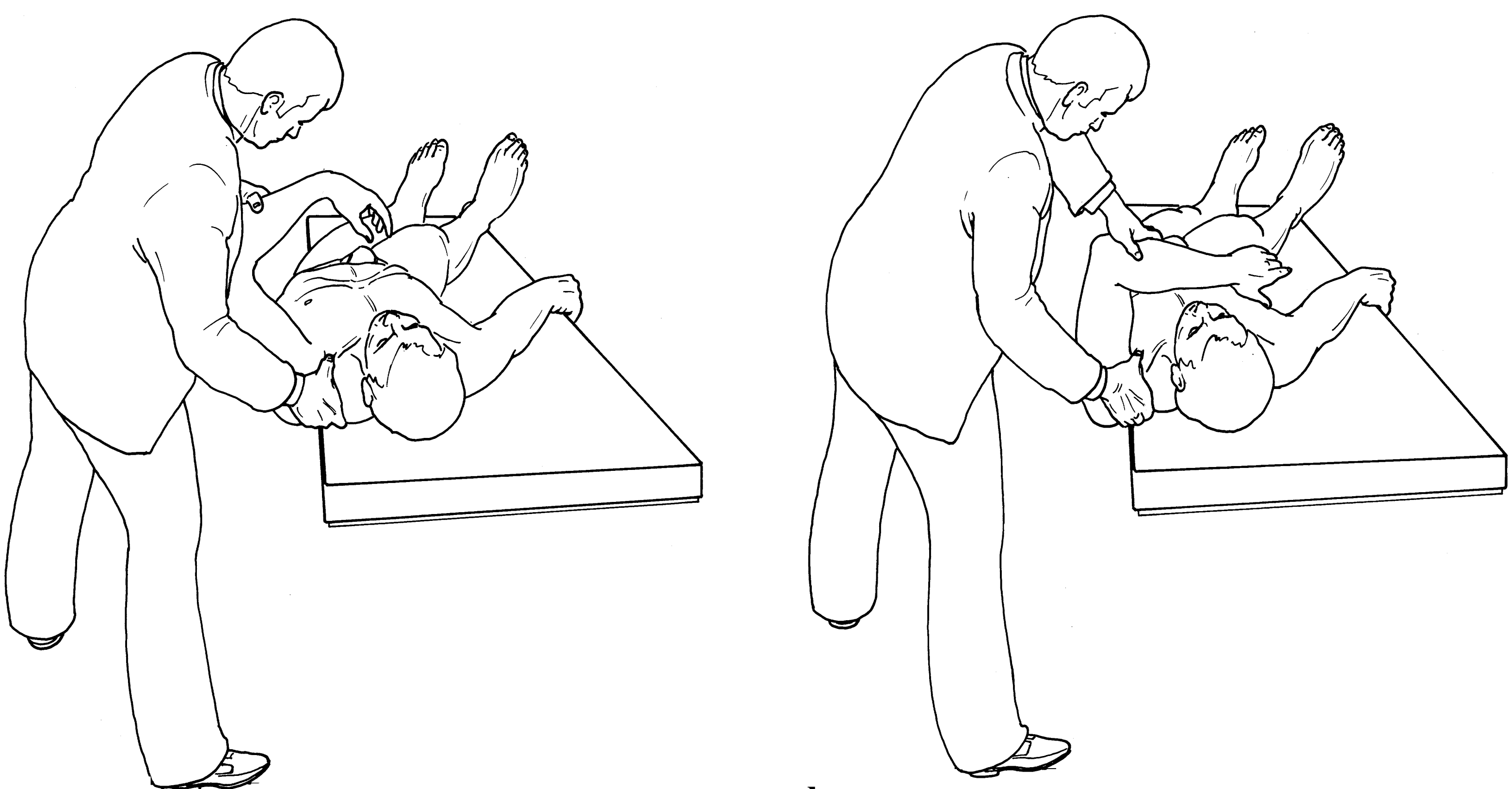
a **b**

Fig. 2.61 Gerber–Ganz anterior drawer test. **(a)** Starting position. **(b)** Dislocation maneuver.

Gerber–Ganz Posterior Drawer Test

□ **Procedure:** The examiner uses one hand to guide the humeral head of the supine patient, placing the thumb on the anterior humeral head and the fingers on the scapular spine, posterior humeral head, and scapular spine and posterior glenoid if necessary. With the other hand the examiner holds the patient's arm in 90° of flexion at about 20 to 30° of horizontal extension. The examiner exerts pressure on the anterior humeral head with the thumb while simultaneously holding the arm in horizontal flexion and applying axial posterior compression in slight internal rotation.

□ **Assessment:** Where there is sufficient laxity in the capsular ligaments, this test will provoke a posterior drawer (subluxation or dislocation of the humeral head). Horizontal extension, slight external rotation of the arm, and additional posteroanterior pressure applied by the finger to the posterior aspect of the humeral head will suffice to reduce the humeral head. The snap that accompanies reduction must be carefully distinguished from anterior subluxation. The important thing is to assess the motion of the humeral head relative to the glenoid fossa by placing the index finger posteriorly around the glenoid and pressing the humeral head in an anteroposterior direction with the thumb.



a b

Fig. 2.62 Gerber–Ganz posterior drawer test. **(a)** Starting position. **(b)** Dislocation maneuver.

The examination may also be performed with the patient seated. With the patient in a relaxed posture bending slightly forward with the arm hanging alongside the trunk, the examiner places his or her thumb on the patient's scapular spine or posterior glenoid and grasps the humeral head anteriorly. Applying rotation and pressure with the fingers will provoke posterior subluxation of the head where there is sufficient laxity in the capsular ligaments.

In posterior instability, the humeral head can be posteriorly displaced by one-half its diameter.

Jerk Test

- **Procedure:** The patient stands or sits. The examiner abducts the affected arm 90° at the shoulder with the elbow flexed 90°. The examiner's other hand stabilizes the shoulder girdle from behind. From this position, the examiner attempts to provoke a posterior drawer sign (subluxation) using increasing internal rotation and adduction with simultaneous axial pressure.
- **Assessment:** If posterior instability is present, the posteriorly oriented thrust along the longitudinal axis of the humerus with slowly increasing adduction leads to posterior subluxation in the glenohumeral joint. Abducting the arm horizontally by 20 to 30° in the same horizontal plane will lead to palpable "snapping" reduction of the humeral head back into the socket with a "jerk" or a "clunk."

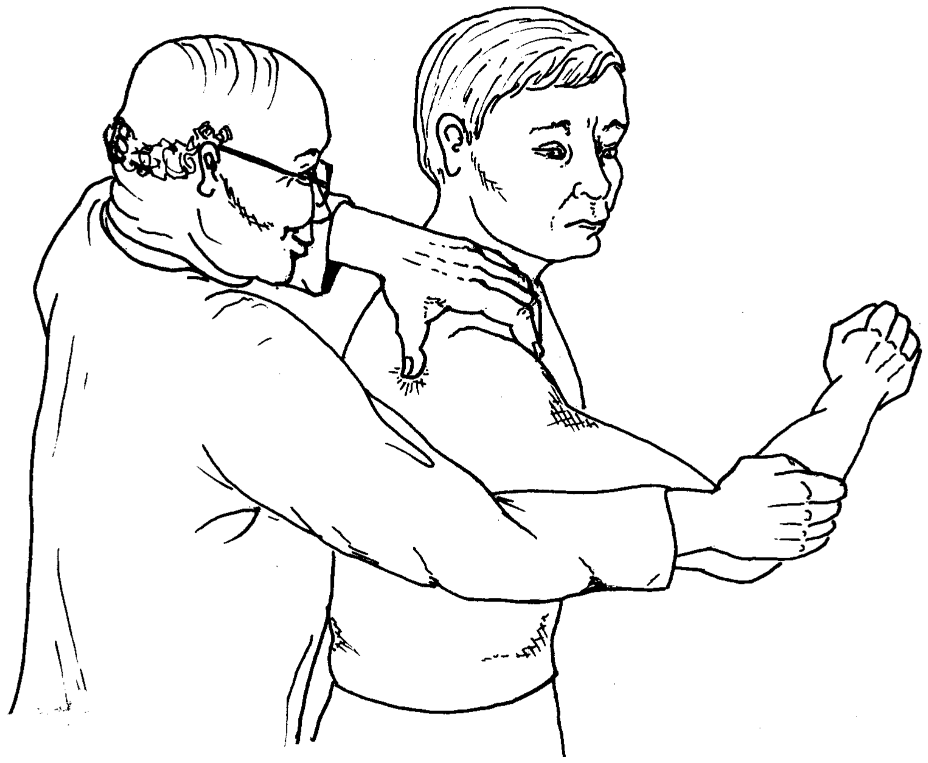


Fig. 2.63 Jerk test.

Fukuda Test

□ **Procedure and assessment:** The Fukuda test elicits a passive posterior drawer sign. The patient is seated with the examiner's thumbs resting on both the patient's scapular spines. The examiner's other fingers rest anterior to the humeral head and exert posterior pressure to trigger a posterior drawer. This test should preferably be performed on both shoulders at the same time to provide a better comparison of the two sides.

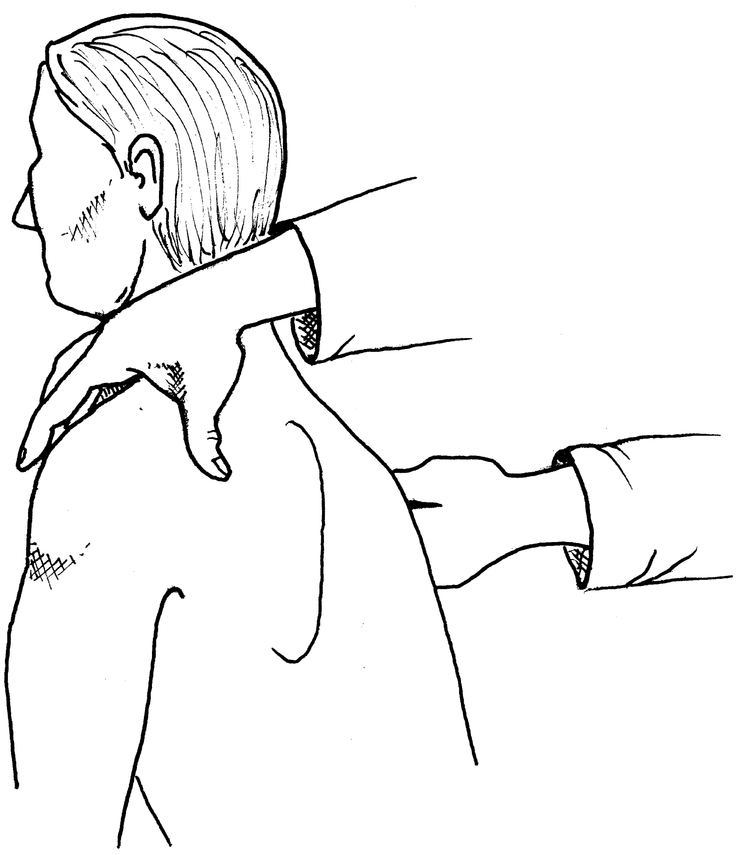


Fig. 2.64 Fukuda test.

Sulcus Sign

Tests for multidirectional instability.

□ **Procedure:** The patient is seated or standing. With one hand, the examiner stabilizes the patient's contralateral shoulder while exerting a distal pull on the patient's relaxed affected arm with the other hand. This is best done by grasping the patient's arm at the elbow, with the elbow slightly flexed.

□ **Assessment:** Instability with distal displacement of the humeral head creates an obvious indentation (sulcus sign) inferior to the acromion.

Gradation in the clinical assessment of the sulcus sign is expressed in millimeters. In reference to the grade of inferior instability, however, there is a large range of physiologic and individual variation. The sulcus sign may be graded by measuring from the inferior margin of the acromion to the humeral head. Grade II/III implies a distance of about 2 to 3 cm. A high-grade sulcus sign (grade III) is a sign of multidirectional instability.

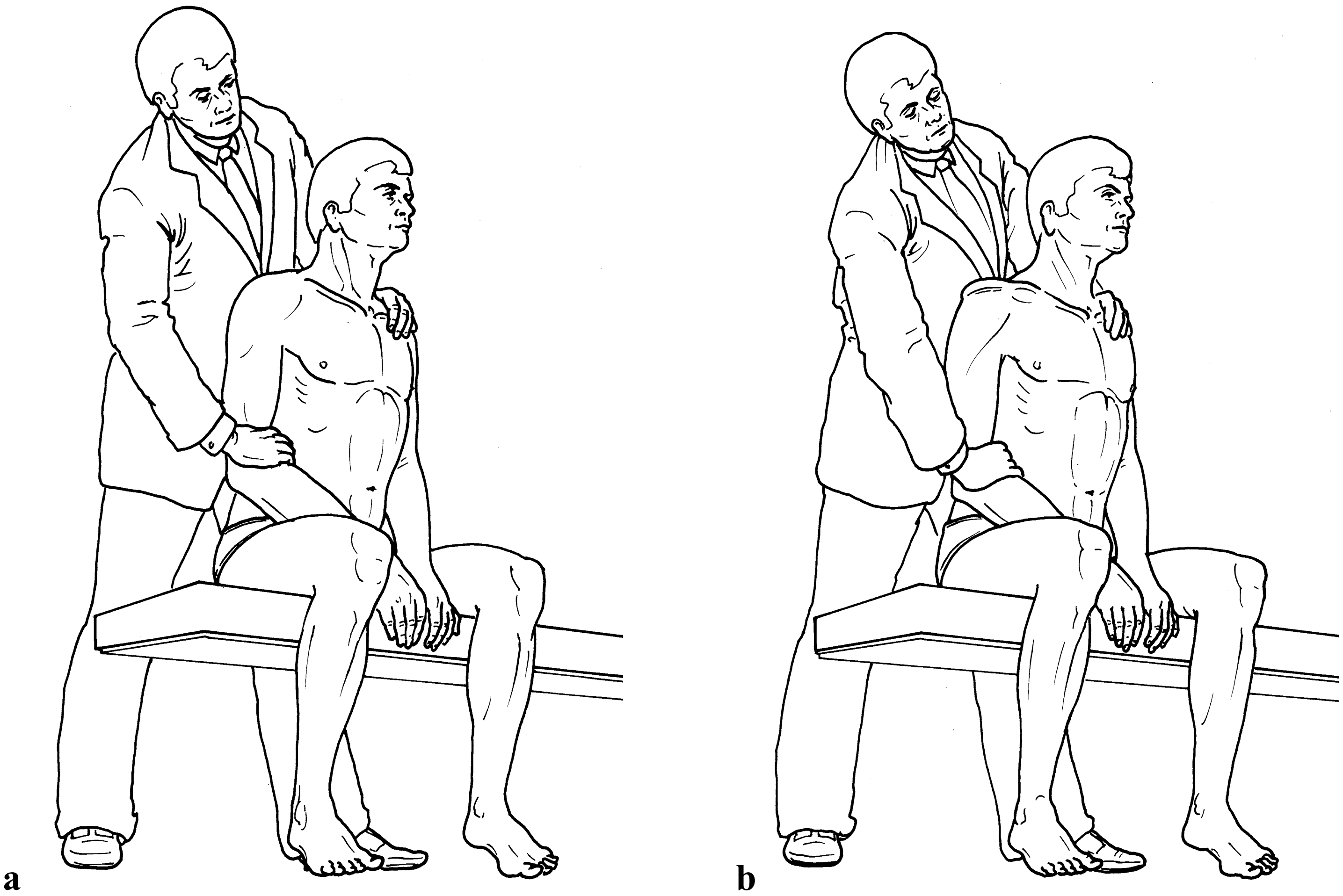


Fig. 2.65 Sulcus sign. (a) Starting position. (b) Sulcus sign with distal distraction of the arm.

The test can also be performed so that the examiner supports the patient's 90°-abducted arm. Applying pressure to the proximal one-third of the upper arm from above can then provoke distal subluxation of the humeral head. This will create a significant step-off beneath the acromion.

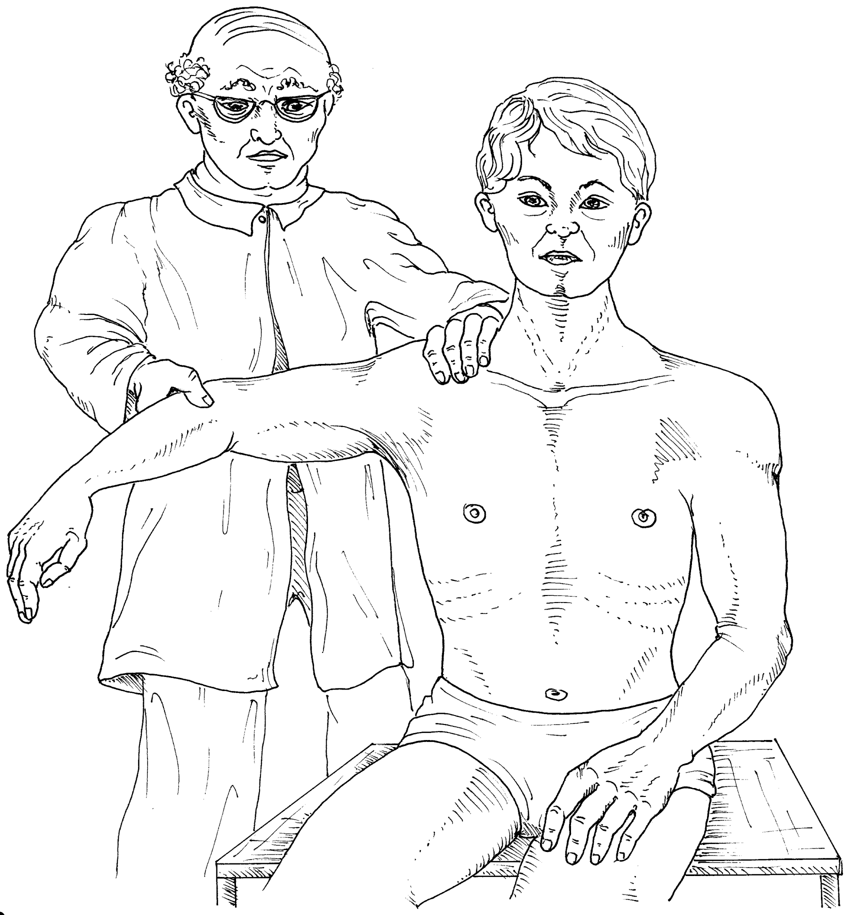
Aside from testing for the sulcus sign in the neutral position, it is recommended to perform the test with the arm externally and internally rotated as well. Increased inferior translation in external rotation suggests elongation of the rotator interval. A positive sulcus sign that occurs with the arm in internal rotation demonstrates laxity of the posterior capsular structures.

Gagey Hyperabduction Test

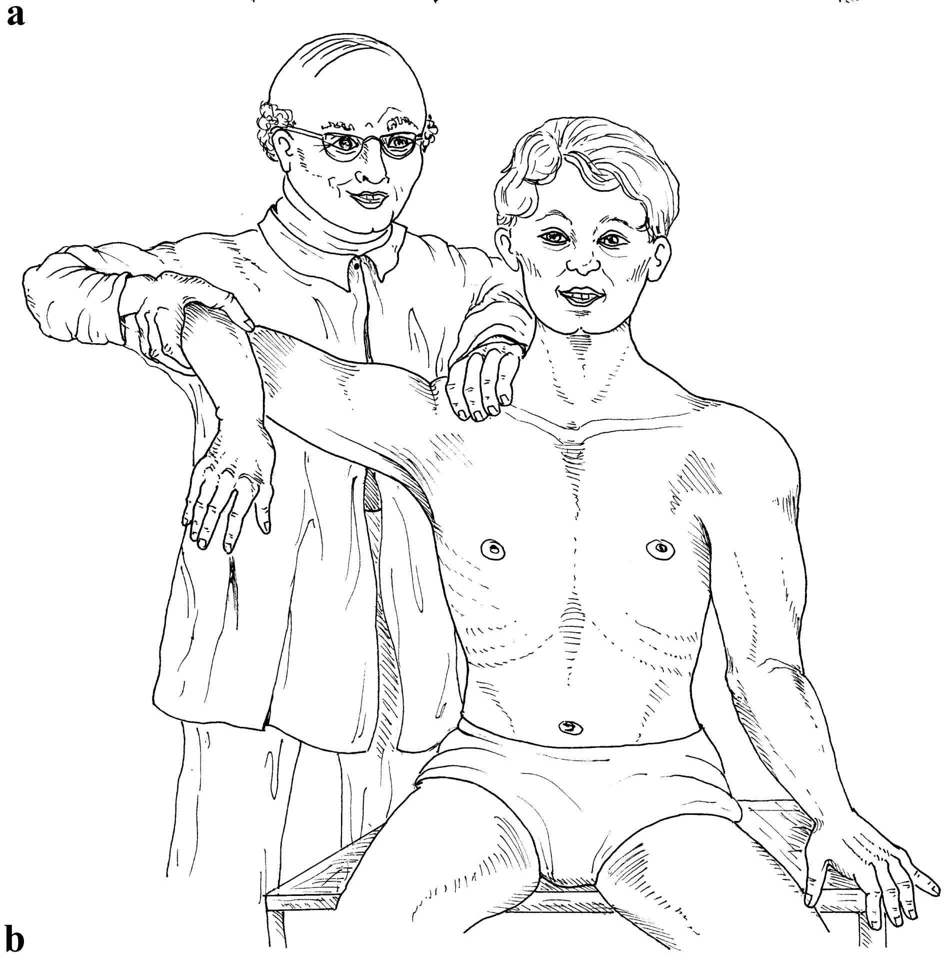
Tests for hyperlaxity of the inferior capsule.

□ **Procedure:** The examiner stands behind the seated patient and immobilizes the scapula with one hand. With the other hand the examiner passively abducts the arm at the glenohumeral joint to the scapular level.

□ **Assessment:** Achieving purely glenohumeral abduction over 105° as opposed to 90° on the other side suggests hyperlaxity of the inferior glenohumeral ligament in particular.



a



b

Fig. 2.66 Gagey hyperabduction test. **(a)** Starting position. **(b)** Increased passive abduction in hyperlaxity.

Rowe Test

□ **Procedure:** The patient stands and bends forward slightly with the arm relaxed. To examine the right shoulder, the examiner grasps the patient's shoulder with the left hand and with the right hand passively moves the patient's arm slightly anteriorly and inferiorly. The examiner then pulls the arms down slightly.

□ **Assessment:** In this position, the examiner can carry out a gentle anteroinferior translation to test shoulder stability.

To test for anterior instability the humeral head is pushed anteriorly with the thumb while the arm is extended 20 to 30° from the vertical position. To test for posterior instability the humeral head is pushed posteriorly with the index and middle fingers while the arm is flexed 20 to 30° from the vertical position.

For inferior instability, more traction is applied to the arm and the sulcus sign is evident.

Fig. 2.67 Rowe test.

3 Elbow

3

Pain in the elbow can have a wide variety of causes. In addition to an accurate clinical examination, there are a series of functional tests to help ensure the correct diagnosis. It is especially important to observe the axis of the outstretched arms. In adults with the forearm supinated, the axis of the ulna is slightly valgus compared to that of the humerus (10-15° in women and 5-10° in men). An angle over 15° indicates cubitus valgus; an angle under 5°, cubitus varus (**Fig. 3.1**). When a joint effusion is present, synovial thickening and osteoarthritis of the joint present as a mild flexion contracture.

Synovial thickening, joint effusions and olecranon bursitis are most clearly seen and palpated posteriorly around the olecranon process.

In arthritis, crepitation may be felt and heard at examination. The patient might describe locking of the elbow in the presence of free intra-articular bodies.

The ulnar and radial collateral ligaments ensure the stability of the elbow. The examiner can detect instabilities easily using appropriate examination

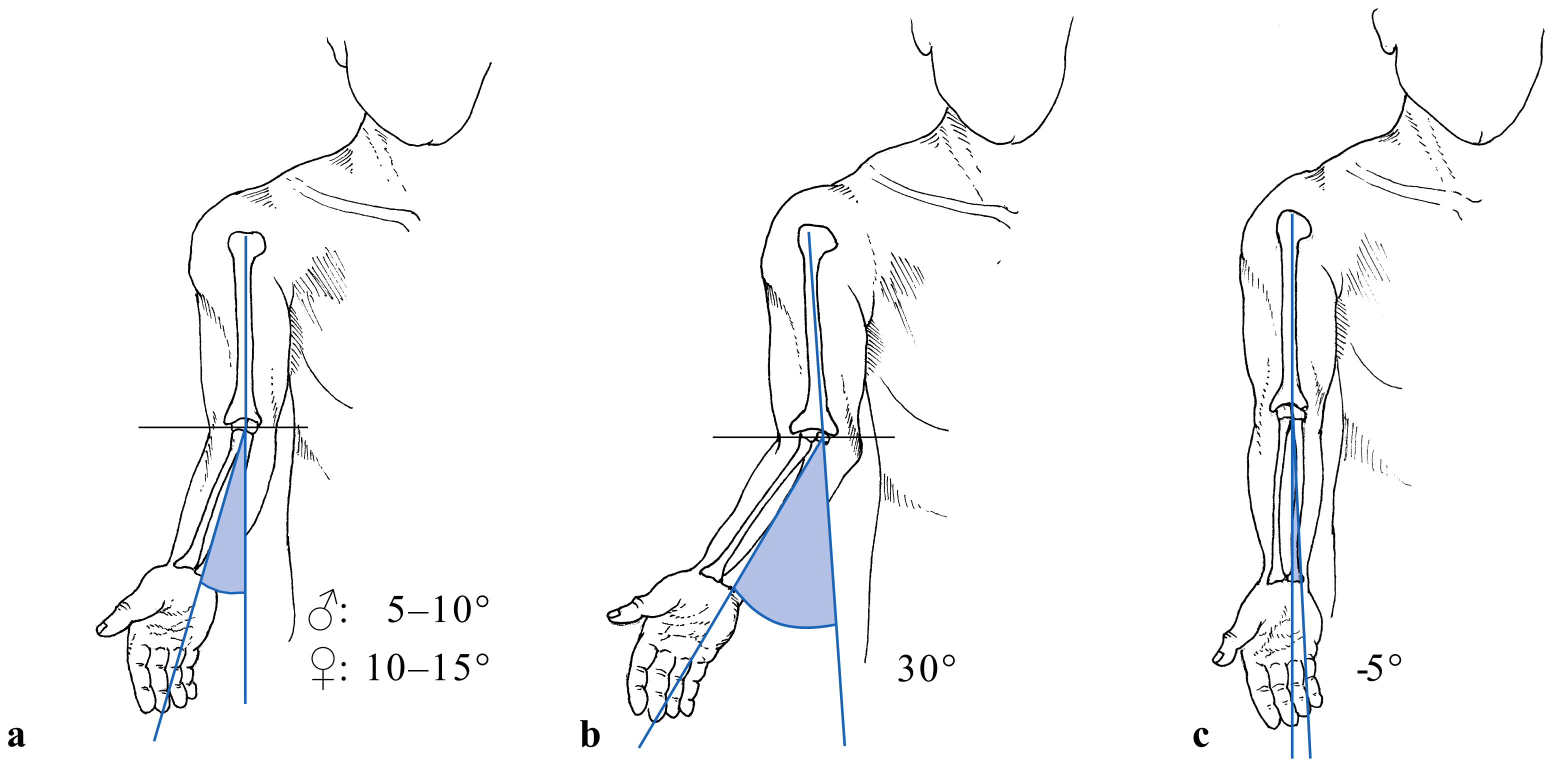


Fig. 3.1 Cubitus varus/valgus. **(a)** Physiologic valgus position: 10 to 15° in women and 5 to 10° in men. **(b)** Extreme cubitus valgus. **(c)** Cubitus varus.

techniques. Swelling, contractures, and painful limitations of movement can have a variety of causes. Osteochondrosis, inflammatory conditions such as rheumatoid arthritis and gout, chondrocalcinosis, tumors, tendinitis, and osteoarthritis are common disorders of the elbow. However, impingement syndromes also originate in the elbow, such as the ulnar nerve sulcus syndrome caused by osteophytes narrowing the groove for the ulnar nerve. Injuries of the medial collateral ligament in adults (see Valgus Stress Test, p. 144) correspond to apophyseal injuries in children and adolescents. Cervical spine syndromes can at times also cause radiating pain in the elbow.

Lateral epicondylitis (tennis elbow) is one of the most common causes for symptoms related to the elbow. Tennis elbow may be caused not only by playing tennis but also by numerous other stressors that cause repeated activation of the wrist extensors. Symptoms localized on the medial epicondyle (medial epicondylitis or golfer's elbow) are less common.

Along with local tenderness, typical findings on examination are circumscribed local pains accompanying passive stretching of the wrist extensors in tennis elbow and the wrist flexors in golfer's elbow as well as pain on tightening the muscles. Pain on the lateral side of the elbow can also be caused by Panner's disease (necrosis of the capitulum of the humerus), osteoarthritis of the humeroradial joint, or compression of the posterior interosseous nerve.

Special examination tests help to distinguish symptoms secondary to epicondylar conditions from other pathologic etiologies.

Range of Motion of the Elbow (Neutral-Zero Method)

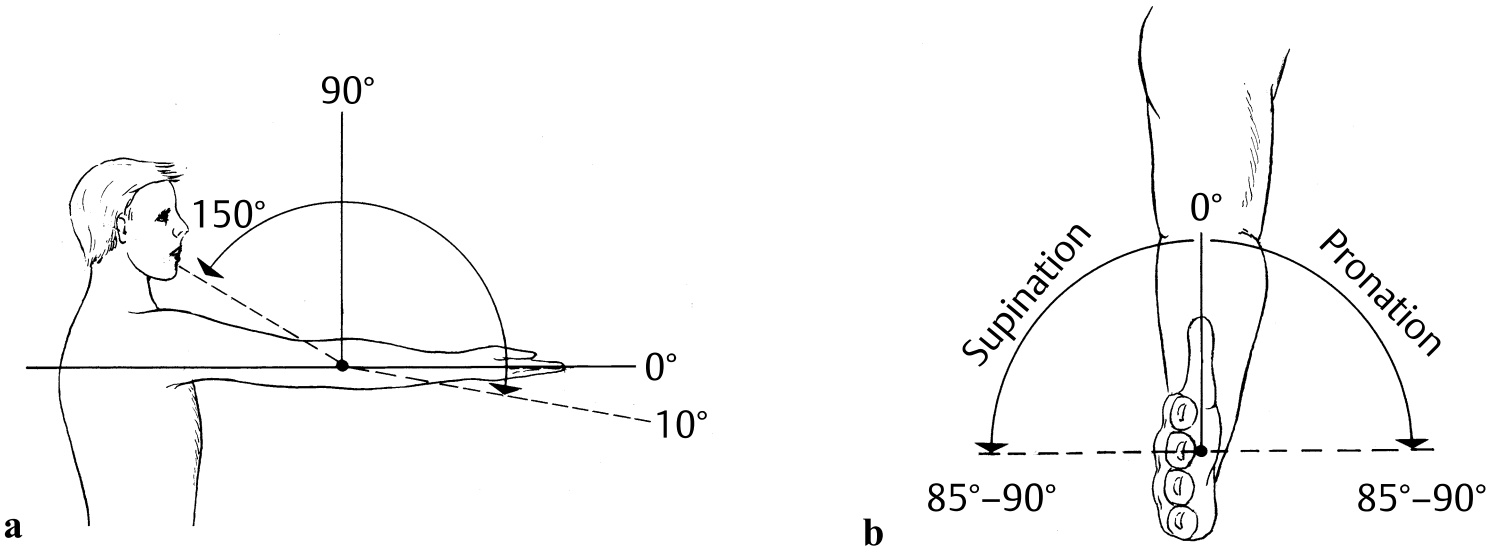


Fig. 3.2. **(a)** Flexion and extension. **(b)** Pronation and supination of the forearm.

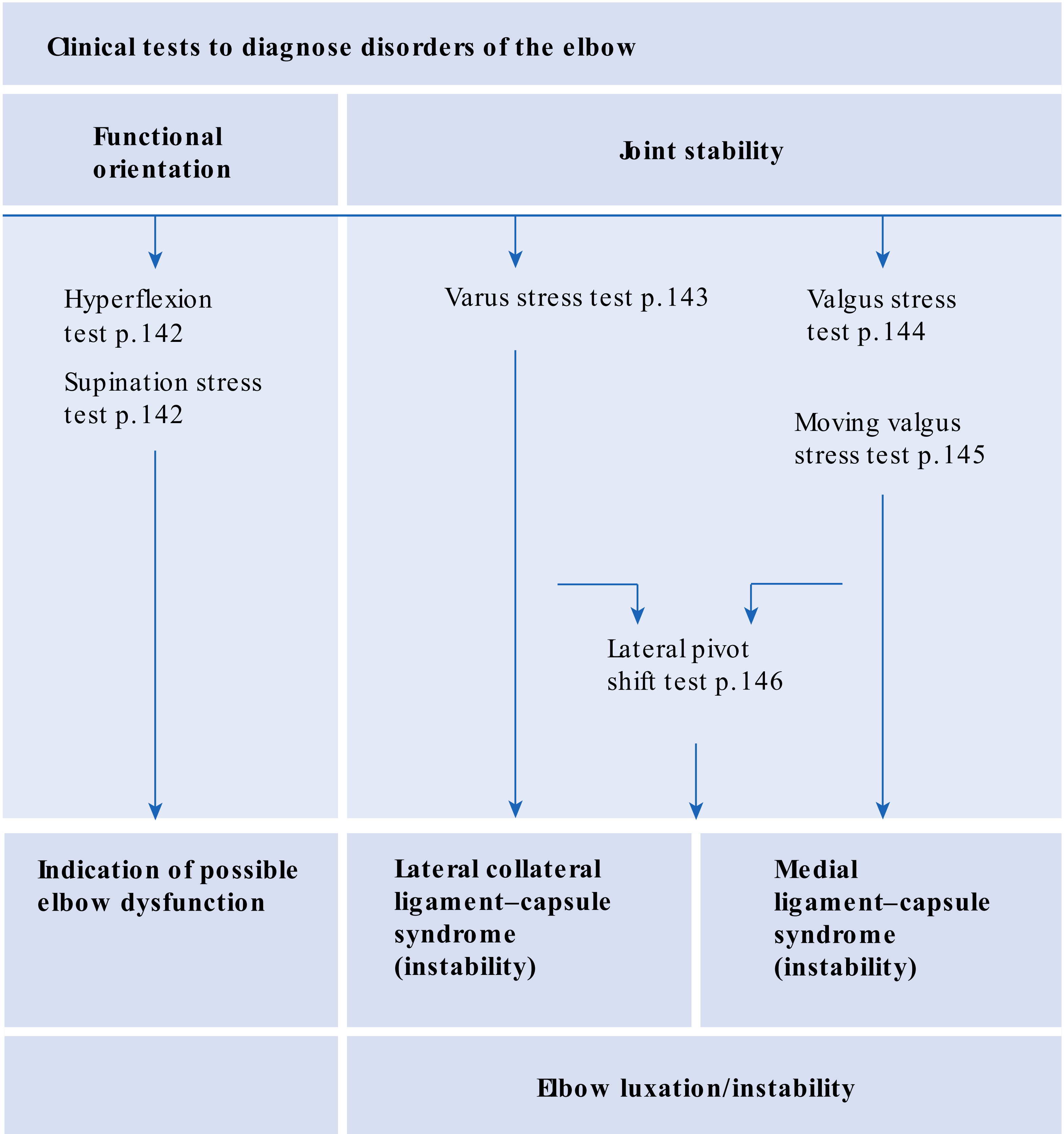


Fig. 3.3 Clinical tests to diagnose disorders of the elbow.

Epicondylitis

- Mill test p. 149
- Cozen test p. 150
- Motion stress test p. 149
- Chair test p. 147
- Bowden test p. 148
- Thomson test p. 148

→ **Lateral epicondylitis (tennis elbow)**

- Reverse Cozen test p. 150
- Golfer's elbow sign p. 151
- Forearm extension test p. 152

→ **Medial epicondylitis (golfer's elbow)**

Nerve compression syndrome

- Tinel test p. 152
- Elbow flexion test p. 153

→ **Cubital tunnel syndrome**

- Supinator compression test p. 154

→ **Radial nerve (deep branch) syndrome**

3

Function Tests

This section describes a series of function tests that will indicate specific lesions in the region of the elbow. Those that provide the most diagnostic information are presented below. They have been divided into four groups based on the particular anatomical structure being tested.

1. General orientation tests.
2. Stability tests.
3. Epicondylitis tests.
4. Compression syndrome tests.

Orientation Tests

Hyperflexion Test

Indicates the presence of an elbow disorder.

Procedure: The patient is seated. The examiner grasps the patient's wrist and maximally flexes the elbow, carefully noting any restricted motion and the location of any pain.

Assessment: Increased or restricted mobility in the joint coupled with pain is a sign of joint damage, muscle contracture, tendinitis, or a sprain. At 70° flexion, the elbow has the greatest capsular volume. An effusion manifests itself primarily in the lateral recess between the tip of the olecranon and the radial head.

Supination Stress Test

For diagnostic assessment of an elbow disorder.

Procedure: The patient is seated. The examiner grasps the patient's forearm with one hand while holding the medial aspect of the elbow with the other. From this position, the examiner forcibly and abruptly supinates the forearm.

Assessment: This test evaluates the integrity of the elbow including the bony and ligamentous structures. Pain or restricted motion suggests joint dysfunction requiring further examination.

Fig. 3.4 Hyperflexion test.

Fig. 3.5 Supination stress test.

Stability Tests

Varus Stress Test

Indicates ligament instability.

□ **Procedure:** The patient sits with the elbow slightly flexed. The examiner stabilizes the upper arm medially with one hand while, with the other hand, adducting the patient's forearm in the elbow joint. This creates varus stress to the lateral collateral ligament (varus instability).

□ **Assessment:** This test checks the stability of the lateral collateral ligament at the elbow. The examiner should note pain and any unusual mobility compared to the uninvolved elbow (see Lateral Pivot Shift Test, p. 146).

Fig. 3.6 Varus stress test.

Valgus Stress Test

Indicates ligament instability.

□ **Procedure:** The patient sits with the elbow slightly flexed. While stabilizing the upper arm laterally with one hand, the examiner abducts the patient's forearm in the elbow joint (valgus stress).

□ **Assessment:** This test checks the stability of the medial collateral ligament at the elbow. The examiner should note pain and any unusual mobility compared to the uninvolved elbow.

Valgus instability occurs posttraumatically (medial collateral ligament injury or fracture of the radial head) or by chronic stress on the medial capsule–ligament apparatus (pitching arm). Injuries to the medial collateral ligament occur in athletes who throw, such as pitchers, European handball players, and javelin throwers. The throwing motion produces valgus and extension stress. Chronic overload can lead to arthritis, ulnar nerve neuritis, and tendinitis at the insertions of the pronator teres muscle and the flexor carpi radialis and ulnaris muscles.

Fig. 3.7 Valgus stress test.

Moving Valgus Stress Test

Indicates instability of the medial capsular ligament apparatus.

□ **Procedure:** The patient stands upright with the shoulder held in 90° abduction. The examiner brings the elbow into maximal flexion under moderate valgus stress until the shoulder has reached its greatest possible external rotation. Maintaining constant valgus pressure, the examiner quickly extends the elbow to about 30° of flexion.

□ **Assessment:** If the test recreates the same pain that the patient complained of during bodily activity, and if the pain is greatest in the "pain zone" between 120 and 70° during extension of the elbow, this suggests with a high degree of sensitivity that there is instability of the medial capsular ligament apparatus.

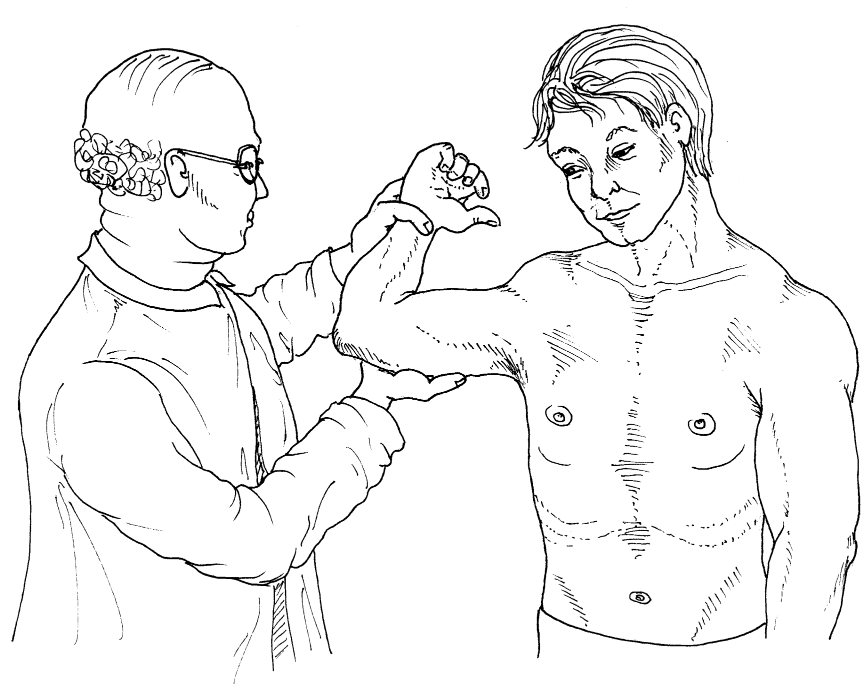


Fig. 3.8 Moving valgus stress test: Under constant valgus pressure, the examiner quickly extends the elbow from maximum flexion to about 30° of flexion.

Lateral Pivot Shift Test (Posterolateral Apprehension Test)

Varus instability of the elbow is acutely due to rupture of the lateral collateral ligament from subluxation of the elbow or from instability due to a lateral collateral ligament injury that has failed to heal properly. Rarely, one sees instability in people who are chronically dependent on walking sticks or in patients in the context of surgery for lateral epicondylitis or surgical repair of a radial head fracture. Posterolateral instability occurs with rupture of the lateral collateral ligament. Because of the anatomical fact that the arm normally has a valgus orientation and load, mild varus instabilities remain unnoticed clinically. Mild valgus instabilities are more likely to be present (see Valgus Stress Test, p. 144).

□ **Procedure:** With the patient supine, the examiner stands at the patient's head and grasps around the patient's wrist with one hand and the slightly flexed elbow with the other. The examiner supinates the forearm and applies a mild valgus force. From this position, the patient's elbow is further flexed and axial pressure is applied to the elbow.

□ **Assessment:** If posterolateral instability is present, a posterolateral subluxation of the elbow occurs at 20 to 30° of flexion, accompanied by pain and an evasive movement by the patient. With continued flexion of the elbow to 40 to 70°, the joint reduces with a palpable and audible "snap."

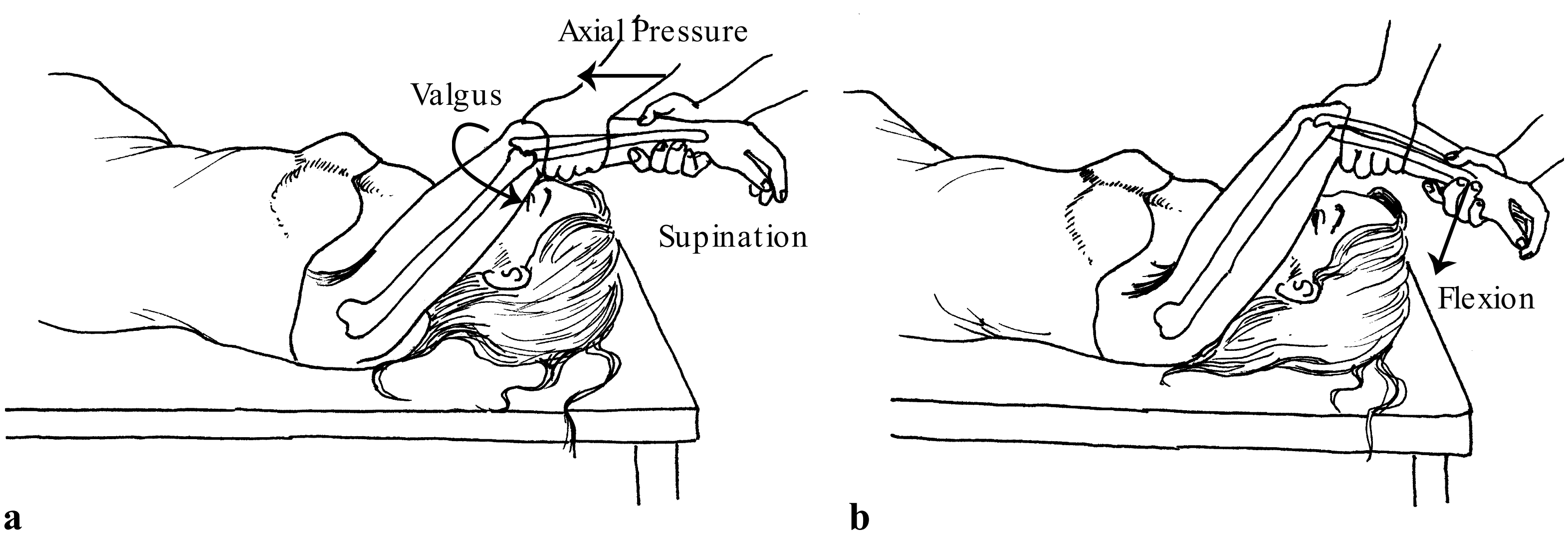


Fig. 3.9 Lateral pivot shift test. **(a)** Subluxation maneuver: flexion of the elbow to 20 to 30°. **(b)** Reduction maneuver: increased flexion of the elbow to 40 to 70°.

Epicondylitis Tests

Chair Test

Indicates lateral epicondylitis.

□ **Procedure:** The patient is requested to lift a chair. The arm should be extended with the forearm pronated.

□ **Assessment:** Occurrence of or increase in pain over the lateral epicondyle and in the extensor tendon origins in the forearm indicates epicondylitis.

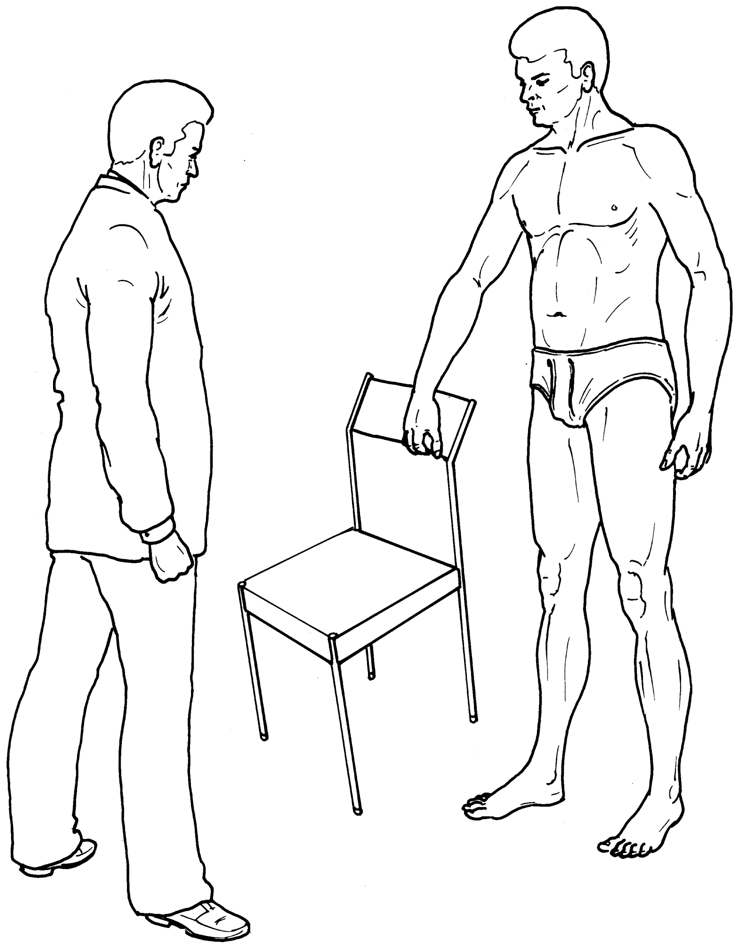


Fig. 3.10 Chair test.

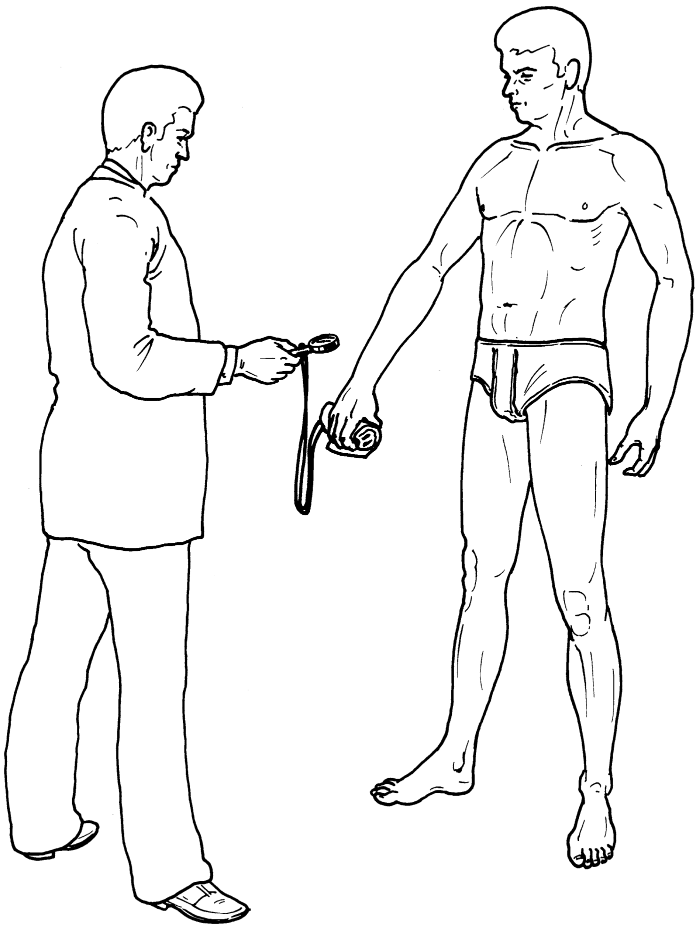


Fig. 3.11 Bowden test.

Bowden Test

Indicates tennis elbow (lateral epicondylitis).

□ **Procedure:** The patient is requested to squeeze a blood-pressure cuff that has been inflated to about 30 mm Hg (about 4.0 kPa) with his or her hand; alternatively, ask the patient to maintain a pressure specified by the examiner by squeezing the cuff.

□ **Assessment:** Occurrence of or increase in pain over the lateral epicondyle and in the extensor tendon origins in the forearm indicates epicondylitis.

Thomson Test (Tennis Elbow Sign)

Indicates lateral epicondylitis.

□ **Procedure:** The patient is requested to make a fist and extend the elbow with the hand in slight dorsiflexion. The examiner immobilizes the dorsal wrist with one hand and grasps the fist with the other hand. The patient is then requested to further extend the fist against the examiner's resistance; or the examiner attempts to press the dorsiflexed fist into flexion against the patient's resistance.

□ **Assessment:** Severe pain over the lateral epicondyle and in the lateral extensor compartment strongly suggests lateral epicondylitis.

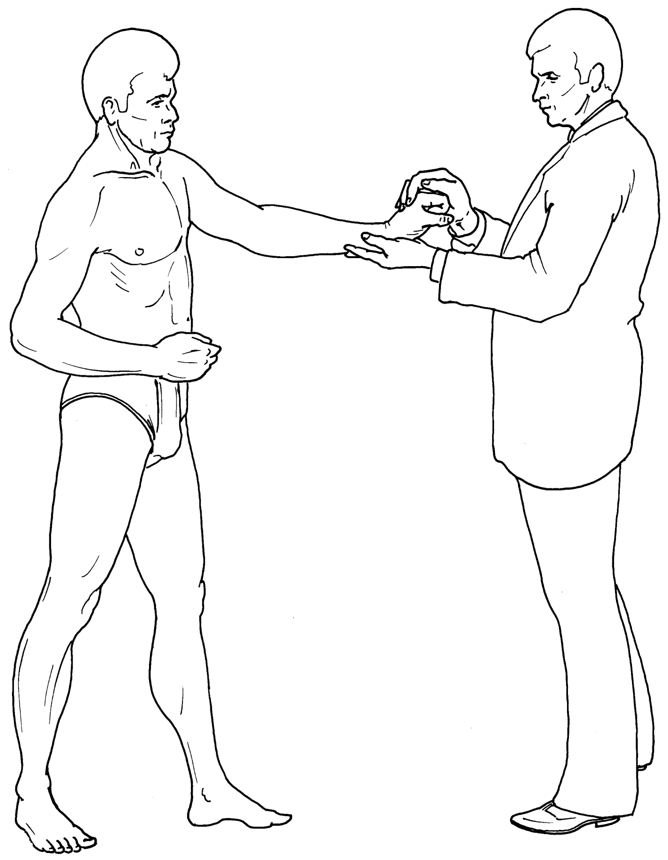


Fig. 3.12 Thomson test.

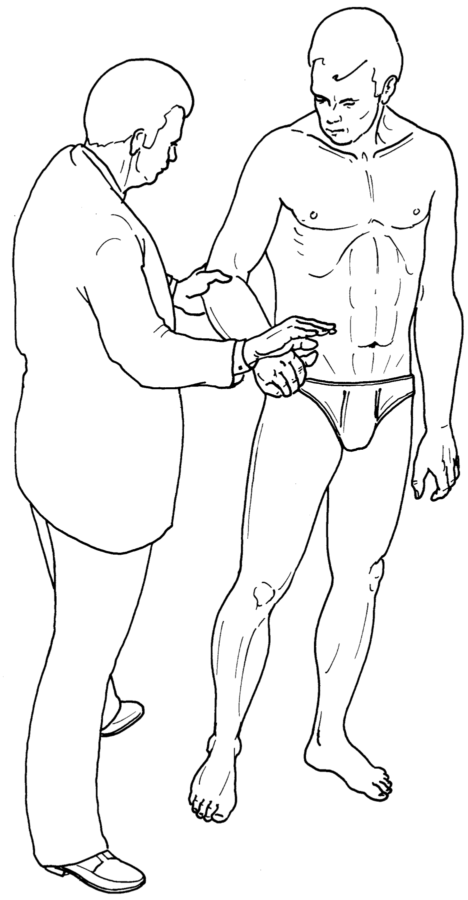


Fig. 3.13 Mill test.

Mill Test

Indicates lateral epicondylitis.

□ **Procedure:** The patient is standing. The arm is slightly pronated with the wrist slightly dorsiflexed and the elbow flexed. With one hand, the examiner grasps the patient's elbow while the other rests on the lateral aspect of the distal forearm or grasps the forearm. The patient is then requested to supinate the forearm against the resistance of the examiner's hand.

□ **Assessment:** Pain over the lateral epicondyle and/or in the lateral extensors suggests epicondylitis.

Motion Stress Test

Indicates lateral epicondylitis.

□ **Procedure:** The patient is seated. The examiner palpates the lateral epicondyle while the patient flexes the wrist and elbow, pronates the forearm, and then extends the elbow again in a continuous motion.

□ **Assessment:** Pronation and wrist flexion place great stresses on the tendons of the forearm muscles that arise from the lateral epicondyle. Occurrence of pain in the lateral epicondyle and/or lateral extensor musculature with these motions suggests epicondylitis. However, pain and paresthesia can also occur as a result of compression of the median nerve, because in this maneuver the action of the pronators can compress the nerve.

a

b

Fig. 3.14 Motion stress test. **(a)** Starting position. **(b)** Extension and pronation.

Cozen Test

Indicates lateral epicondylitis.

□ **Procedure:** The patient is seated for the examination. The examiner immobilizes the elbow with one hand while the other hand lies flat on the dorsum of the patient's fist. The patient is then requested to dorsiflex the wrist against the resistance of the examiner's hand. Alternatively, the examiner may attempt to press the fist, which the patient holds with the wrist firmly extended, into flexion against the patient's resistance.

□ **Assessment:** Localized pain in the lateral epicondyle of the humerus or pain in the lateral extensor compartment suggests epicondylitis.

Fig. 3.15 Cozen test.

Reverse Cozen Test

Indicates medial epicondylitis.

□ **Procedure:** The patient is seated. The examiner palpates the medial epicondyle with one hand while the other hand rests on the wrist of the patient's supinated forearm. The patient attempts to flex the extended hand against the resistance of the examiner's hand on the wrist.

□ **Assessment:** The flexors of the forearm and hand and the pronator teres have their origins on the medial epicondyle. Acute, stabbing pain over the medial epicondyle suggests medial epicondylitis.

With this test, it is particularly important to stabilize the elbow. Otherwise, a forcible avoidance movement or pronation could exacerbate a compression syndrome in the pronator musculature (pronator compartment syndrome).

a b

Fig. 3.16 Reverse Cozen test. **(a)** Starting position. **(b)** Flexion in the wrist against the resistance of the examiner's hand.

Golfer's Elbow Sign

Indicates medial epicondylitis.

□ **Procedure:** The patient flexes the elbow and hand. The examiner grasps the patient's hand and immobilizes the patient's upper arm with the other hand. The patient is then requested to extend the elbow against the resistance of the examiner's hand.

□ **Assessment:** Pain over the medial epicondyle suggests epicondylar pathology (golfer's elbow).

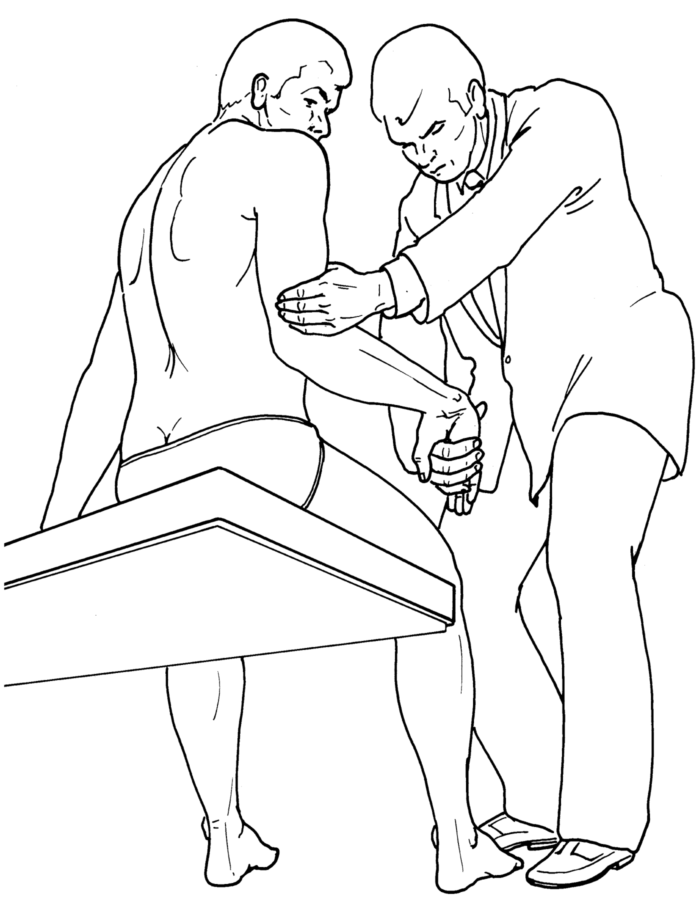


Fig. 3.17 Golfer's elbow sign.

Forearm Extension Test

Indicates medial epicondylitis.

□ **Procedure:** The examiner grasps the patient's distal forearm. The patient then attempts to extend the elbow against the resistance of the examiner's hand.

□ **Assessment:** Pain over the medial epicondyle and over the origins of the forearm flexors suggests epicondylar pathology.

Fig. 3.18 Forearm extension test.

Compression Syndrome Tests

Tinel Test

Sign of cubital tunnel syndrome.

□ **Procedure:** The patient is seated. The examiner grasps the patient's arms and gently taps on the groove for the ulnar nerve with a reflex hammer.

□ **Assessment:** The ulnar nerve courses through a bony groove posterior to the medial epicondyle. Because of its relatively superficial position, compression injuries are common. The most common causes of damage to the ulnar nerve are scar tissue, callus, or exostosis formation following condylar or supracondylar fractures; chronic inflammatory conditions related to rheumatoid arthritis or epicondylitis; and chronic mechanical compression from constantly propping the elbow on hard surfaces. Inherent dysplasia of the bony nerve canal can also be the cause of an ulnar nerve syndrome. Pain elicited by gently tapping the groove for the ulnar nerve suggests chronic compression neuropathy.

With this test, care should be taken not to tap the nerve too hard because a forceful tap will cause pain even in a normal nerve. Note, too, that repeated tapping can injure the nerve.

Elbow Flexion Test

Sign of cubital tunnel syndrome.

Procedure: The patient is seated. The elbow is maximally flexed with the wrist flexed as well. The patient is requested to maintain this position for 5 minutes.

Assessment: The ulnar nerve passes through the cubital tunnel, which is formed by the ulnar collateral ligaments and the flexor carpi ulnaris muscle. Maximum traction is applied to the ulnar nerve in the position described above.

Occurrence of paresthesia along the course of the nerve suggests compressive neuropathy. If the test is positive, the diagnosis should be confirmed by electromyography or nerve conduction velocity measurement.

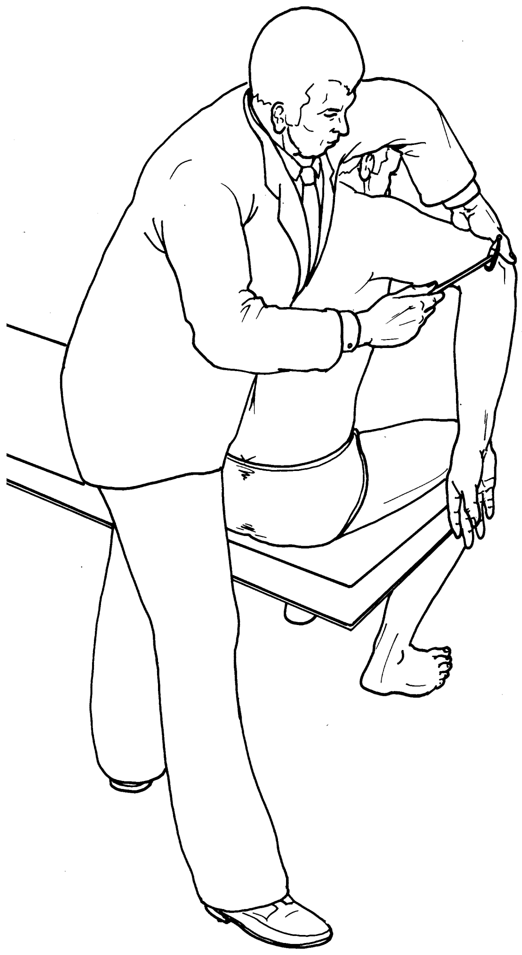


Fig. 3.19 Tinel test.

Fig. 3.20 Elbow flexion test.

3

Supinator Compression Test

Indicates a supinator compartment syndrome (Frohse's syndrome) with damage to the deep branch of the radial nerve.

□ **Procedure:** The patient stands for the test. With one hand, the examiner palpates the groove lateral to the extensor carpi radialis muscle distal to the lateral epicondyle. The examiner's other hand resists the patient's active pronation and supination.

□ **Assessment:** Constant tenderness in the muscle groove or pain in the proximal lateral forearm that increases with pronation and supination suggests compression of the deep branch of the radial nerve within the supinator muscle. (Above the radial head, the radial nerve divides into its two terminal branches. The deep, motor branch of the radial nerve penetrates the supinator muscle to extend from the antecubital fossa to the posterior side of the forearm.)

The point of tenderness lies farther anterior than the point at which pain is felt in typical lateral epicondylitis. The compression neuropathy of the nerve can be caused by proliferation of connective tissue in the muscle, a radial head fracture, or a soft tissue tumor. Direct injury to the nerve can also occur iatrogenically during an intramuscular injection performed to treat lateral epicondylitis. Weakened or absent extension in the metacarpophalangeal joints of the fingers other than the thumb indicates paralysis of the extensor digitorum supplied by the deep branch of the radial nerve.

Fig. 3.21 Supinator compression test.

4 Wrist, Hand, and Fingers

Injuries to and lesions of the hand play a significant role in everyday life and in sports.

Examination of the hand requires good knowledge of the functional anatomy and begins with inspection to detect possible defects and position anomalies. With the hand at rest in a passive position, the wrist is in a neutral position between flexion and extension and the fingers are in slight flexion (the finger flexors are about four times as strong as the finger extensors).

Joint inflammation causes circumscribed swelling over the respective joint, and tenosynovitis manifests itself as swelling and erythema in the skin along the course of the tendon. Swelling of the distal interphalangeal joints with a painful flexion contracture (Heberden nodes) often occurs in postmenopausal women. Chronic inflammatory disorders (rheumatoid arthritis) often first manifest themselves in the metacarpophalangeal and proximal interphalangeal joints.

Ganglia arising from the tendons, tendon sheath, or synovial tissue may be the cause of swelling. Nerve palsy leads to contractures. For example, radial nerve palsy leads to a wrist drop. Median nerve palsy leads to deformity resembling an ape's hand. Ulnar nerve palsy leads to a claw hand deformity in which the proximal phalanges are extended and the middle and distal phalanges are flexed.

When palpating the wrist and hand, the examiner notes the texture and quality of the skin, muscles, and tendon sheaths; evaluates swelling, inflammation, and tumors; and determines the exact localization of pain.

Passive range of motion testing can detect restricted motion (due to osteoarthritis) and instability. Painful disorders of the tendon sheaths can be associated with crepitation along the course of the tendon in both active and passive motion.

Neurologic changes such as muscle atrophy, usually caused by compression neuropathies, exhibit characteristic losses of function that can be evaluated by specific functional tests.

Range of Motion in the Hand (Neutral-Zero Method)

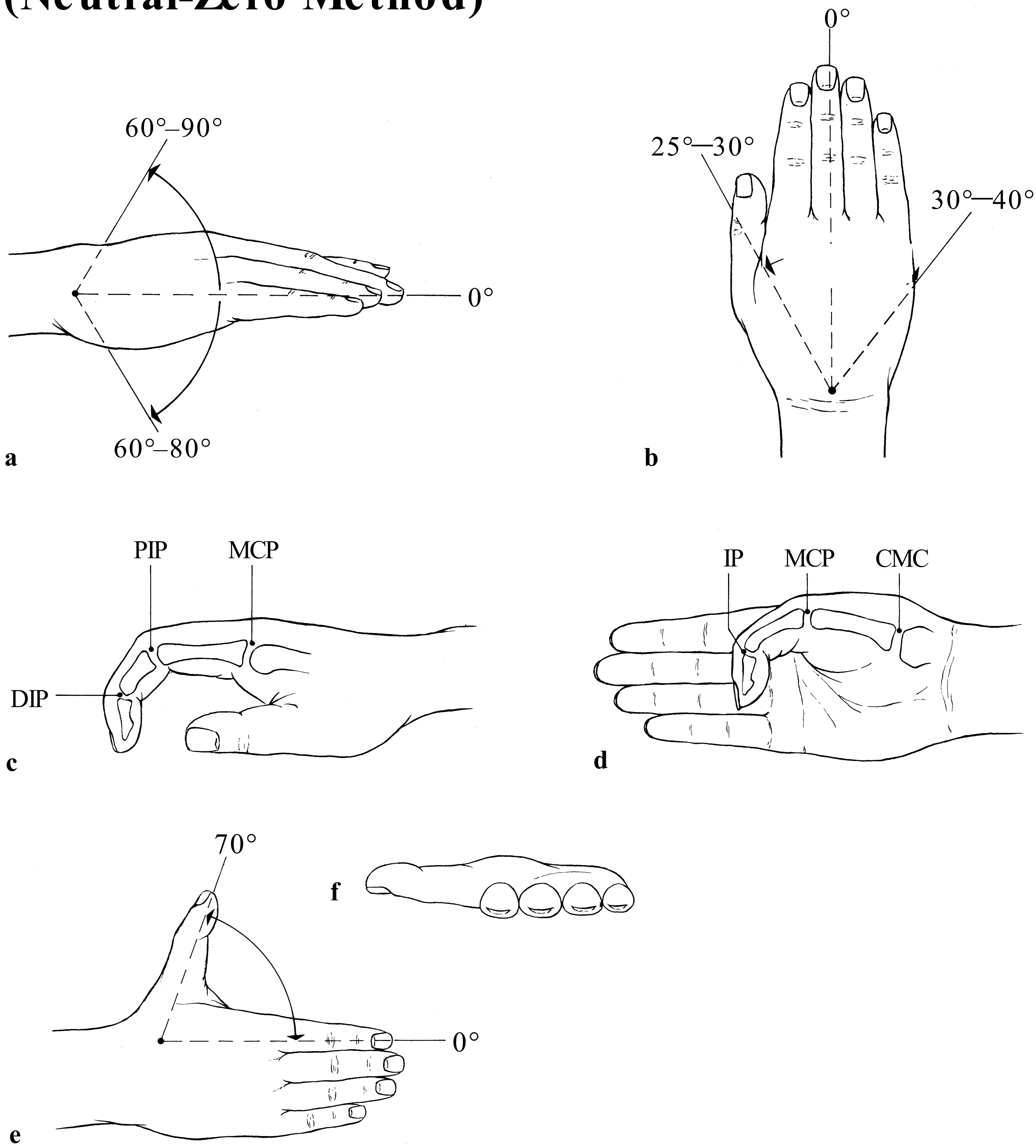
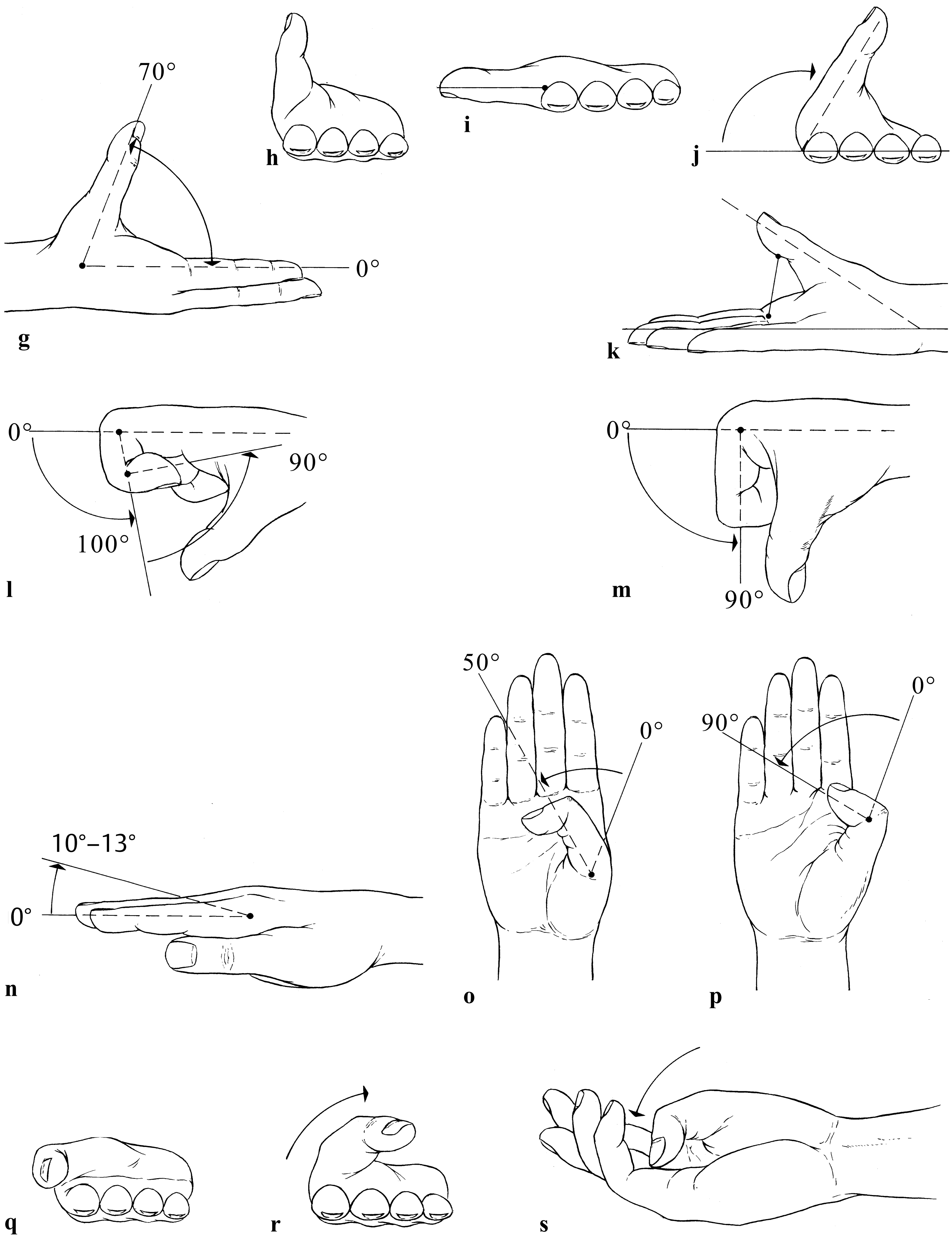


Fig. 4.1. **(a)** Flexion and extension of the wrist including the intercarpal joints. **(b)** Radial and ulnar deviation of the hand. **(c, d)** Designations of the joints of the fingers **(c)** and thumb **(d)**: DIP, distal interphalangeal joint; PIP, proximal interphalangeal joint; MCP, metacarpophalangeal joint; IP, interphalangeal joint (of the thumb); CMC, carpometacarpal joint. **(e, f)** Abduction and adduction of the thumb



in the plane of the palm. **(g, h)** Palmar abduction and adduction of the thumb perpendicular to the plane of the palm. **(i–k)** Circumduction of the extended thumb. **(l, m)** Flexion of the finger joints: DIP and PIP joints **(l)** and MCP joint **(m)**. **(n)** Hyperextension of the MCP joint. **(o, p)** Flexion of the thumb joints: the MCP joint **(o)** and the IP joint **(p)**. **(q–s)** Opposition of the thumb: starting position **(q)**, during motion **(r)**, and in opposition position **(s)**.

Clinical tests to diagnose disorders of the hands, wrists, and fingers

Tenosynovitis		Motor function tests	Contract-tures	Nerve compres-sion syndrome
Hand flexor tendon tests p.163 Muckard test p.161 Finkelstein test p.162 Linburg test p.162	Grind test p.164 (Muckard test) (Finkelstein test)	Pinch grip pp.174, 47 Key grip p.174 Power grip p.175 Chuck grip p.175 Grip strength p.175	Bunnel-Littler test p.165	Radial nerve palsy screening test p.177 Thumb extension test p.178 Supination test p.178
Tenosynovitis (inflammatory, rheumatic)	**Thumb saddle joint arthritis**	**Osteoarthritis, tenosynovitis (inflammatory, rheumatic), instability, nerve lesion**	**Contractures of the intrinsic muscles of the hand (Volkmann's contracture)**	**Radial nerve lesion**

Fig. 4.2 Clinical tests to diagnose disorders of the hands, wrists, and fingers.

Nerve compression syndrome

- Tinel sign p. 179
- Median nerve palsy screening test p. 180
- Ochsner test p. 181
- Carpal tunnel sign p. 181
- Phalen test p. 182
- Nail sign p. 182
- Bottle test p. 183
- Reverse Phalen test p. 184
- Wrist flexion provocation test p. 188
  - → **Median nerve lesion**

- Froment sign p. 185
- Ulnar nerve palsy screening test p. 186
- Intrinsic test p. 186
- O test p. 187
  - → **Ulnar nerve lesion**

Testing wrist stability

- Watson test p. 186
- Scapholunate ballottement test p. 167
  - → **Scapho-lunate instability**
  - → Dorsal capitate displacement apprehension test p. 168
    - → **Capitate instab-ility, arthritis**
- Reagan test p. 169
  - → **Lunotri-quetral instability**
- Stability test for torn MCP ulnar collateral ligament of the thumb p. 170
  - → **Skier's thumb**
- Finger exten-sion (Shuck) test p. 168
  - → **Radiocarpal, midcarpal, or scaphoid instability**

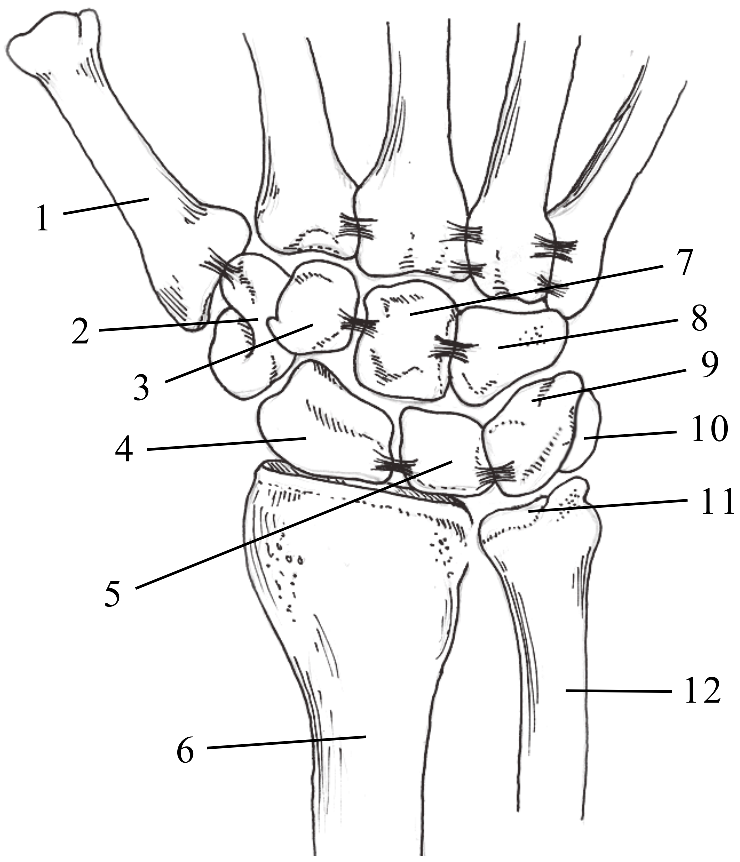


Fig. 4.3 Anatomy of the hand, palmar aspect.
1 First metacarpal.
2 Trapezium.
3 Trapezoid.
4 Scaphoid.
5 Lunate.
6 Radius.
7 Capitate.
8 Hamate.
9 Triquetrum.
10 Pisiform.
11 Articular disk.
12 Ulna.

Function Tests

Tests of the Flexor Tendons of the Hand

Flexor Digitorum Profundus

□ **Procedure:** The examiner places two fingers (index and middle fingers) on the volar aspect of the patient's affected finger so that the finger remains extended in the proximal interphalangeal joint (**Fig. 4.4a**). The patient is then asked to flex only the distal interphalangeal joint of the finger. This examination is repeated for each finger separately.

□ **Assessment:** The flexor digitorum profundus belongs to the deep layer of flexors in the forearm. Its tendons insert into the palmar aspect of the bases of all of the distal phalanges of fingers 2 to 5.

Inability to flex the distal interphalangeal joint is a sign of a torn tendon; painful flexion suggests tenosynovitis.

Differential diagnosis should exclude osteoarthritis of the distal interphalangeal joint (Heberden nodes) with a painful flexion contracture.

Flexor Digitorum Superficialis

□ **Procedure:** The patient is asked to flex the proximal interphalangeal joint of the affected finger while the examiner holds the other fingers in extension to neutralize the effect of the flexor digitorum profundus tendon (**Fig. 4.4b**). The flexor digitorum profundus tendons of the three ulnar fingers share a common muscle belly. Therefore, unrestricted flexion of one finger with the others immobilized in extension requires an intact flexor digitorum superficialis tendon. This examination is performed for each finger separately.

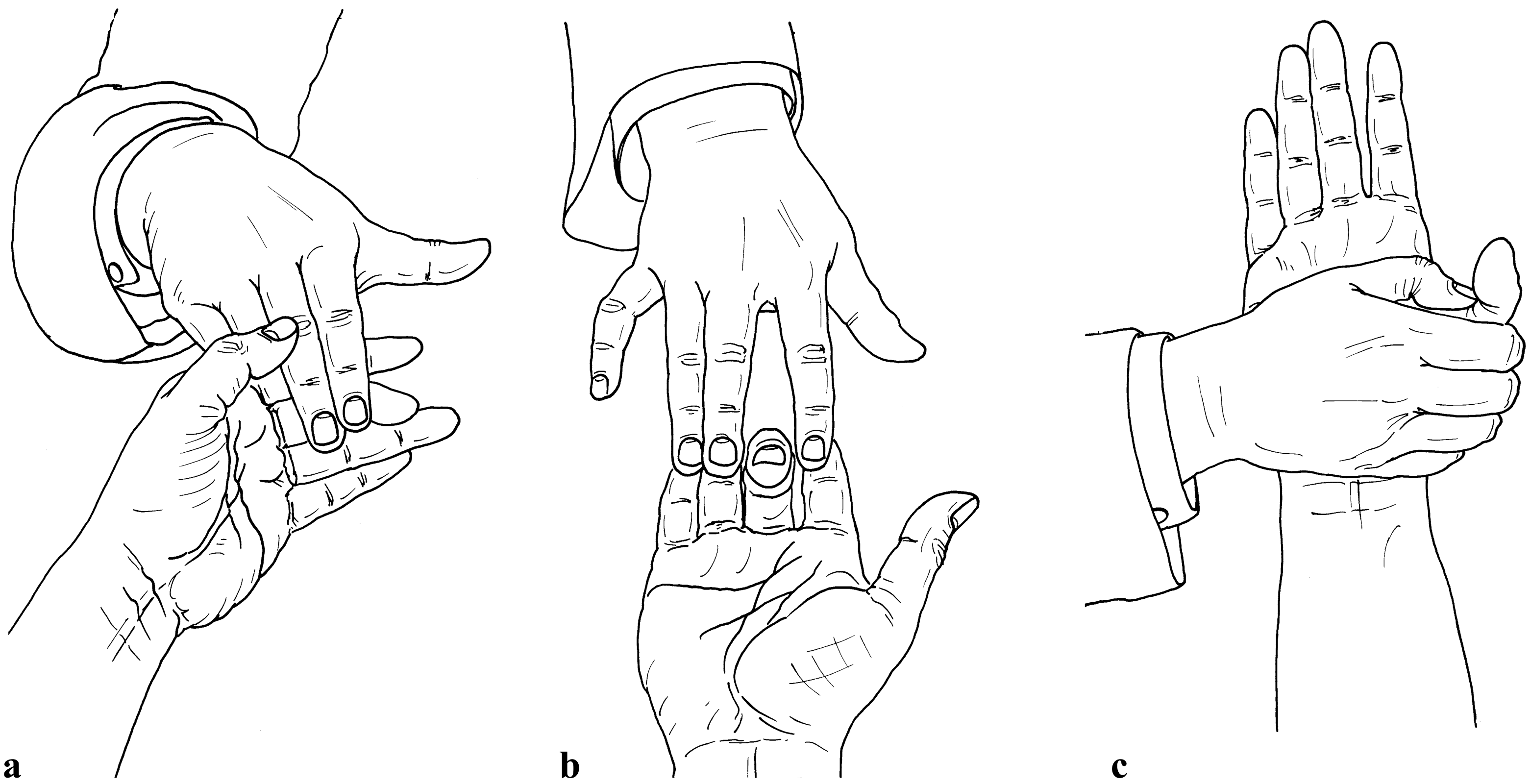
□ **Assessment:** The flexor digitorum superficialis is a broad, strong muscle whose tendons insert into the middle phalanges of the fingers.

Wherever the patient can flex the proximal interphalangeal joint of a finger, the flexor digitorum superficialis tendon is intact. Flexion will not be possible where injuries to the tendon are present. Pain suggests tenosynovitis.

Flexor Pollicis Longus and Extensor Pollicis Longus

□ **Procedure:** The examiner grasps the patient's thumb and immobilizes the metacarpophalangeal joint (**Fig. 4.4c**). Then the patient is asked to flex and extend the phalanx of the thumb. The flexor pollicis longus lies in the deep layer of the flexor muscles; its tendon inserts into the base of the distal phalanx of the thumb.

□ **Assessment:** Impaired flexion and extension in the interphalangeal joint of the thumb suggest injury (torn tendon) or disease (tenosynovitis) of the respective tendon.



a b c

Fig. 4.4 Tests of the hand flexor tendons. **(a)** Flexor digitorum profundus. **(b)** Flexor digitorum superficialis. **(c)** Flexor pollicis longus and extensor pollicis longus.

Muckard Test

For diagnosis of acute or chronic tenosynovitis of the abductor pollicis longus and extensor pollicis brevis tendons (stenosing tenosynovitis or de Quervain's disease).

▯ **Procedure:** The patient "tilts" the hand into ulnar deviation at the wrist with the fingers extended and the thumb adducted.

▯ **Assessment:** Severe pain in the radial styloid radiating into the thumb and forearm suggests tenosynovitis of the abductor pollicis longus and extensor pollicis brevis tendons.

Swelling and tenderness to palpation over the first dorsal compartment will usually be present as well. Abduction of the thumb against resistance is painful.

Tenosynovitis is the result of inflammation of the synovial tissue, which is often caused by overuse or inflammatory rheumatoid disorders. However, blunt trauma can also lead to these disorders.

A differential diagnosis should exclude osteoarthritis of the carpometacarpal joint of the thumb or radial styloiditis.

Finkelstein Test

Indicates stenosing tenosynovitis (de Quervain's disease).

▯ **Procedure:** With the thumb flexed and the other fingers flexed around it, the wrist is moved into ulnar deviation either actively or passively by the examiner.

▯ **Assessment:** Pain and crepitation above the radial styloid suggest nonspecific tenosynovitis of the abductor pollicis longus and the extensor pollicis brevis (see Muckard Test for etiology).

It is important to differentiate stenosing tenosynovitis (de Quervain's disease) from osteoarthritis in the carpometacarpal joint of the thumb. Specific examination of the carpometacarpal joint of the thumb and a radiograph will allow a quick differential diagnosis. The test should also be performed on both sides for comparison.

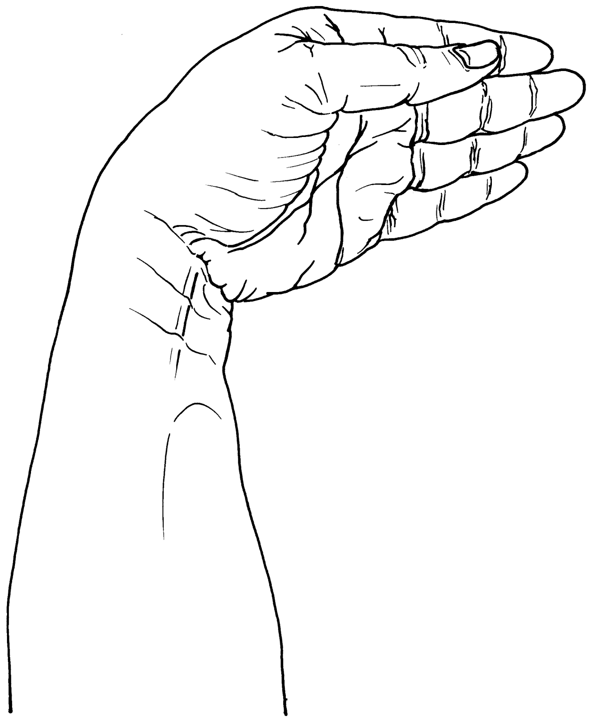


Fig. 4.5 Muckard test.

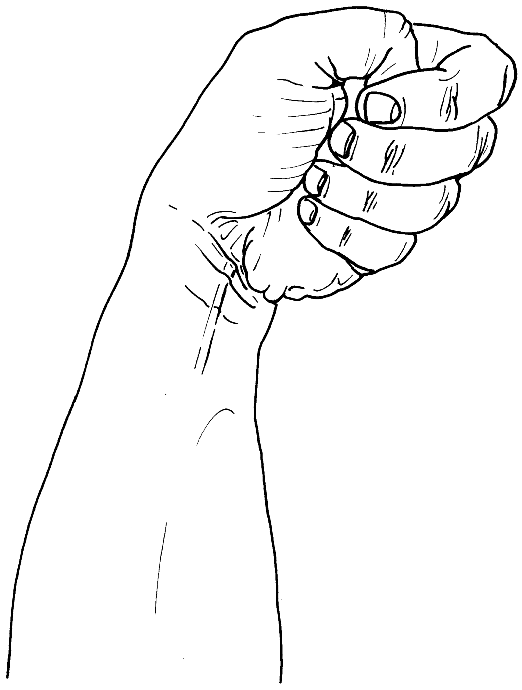


Fig. 4.6 Finkelstein test.

Grind Test

Assessment of osteoarthritis in the carpometacarpal joint of the thumb.

- **Procedure:** The examiner grasps the painful thumb and performs grinding motions while compressing the thumb along its longitudinal axis.
- **Assessment:** Pain reported in the carpometacarpal joint of the thumb is usually due to osteoarthritis in the joint (differential diagnosis includes a Bennett or Rolando fracture). Tenderness to palpation and painful instability are additional signs of wear in the joint. The patient will typically also complain of pain in the carpometacarpal joint of the thumb when opposing the thumb against the resistance of the examiner's hand.

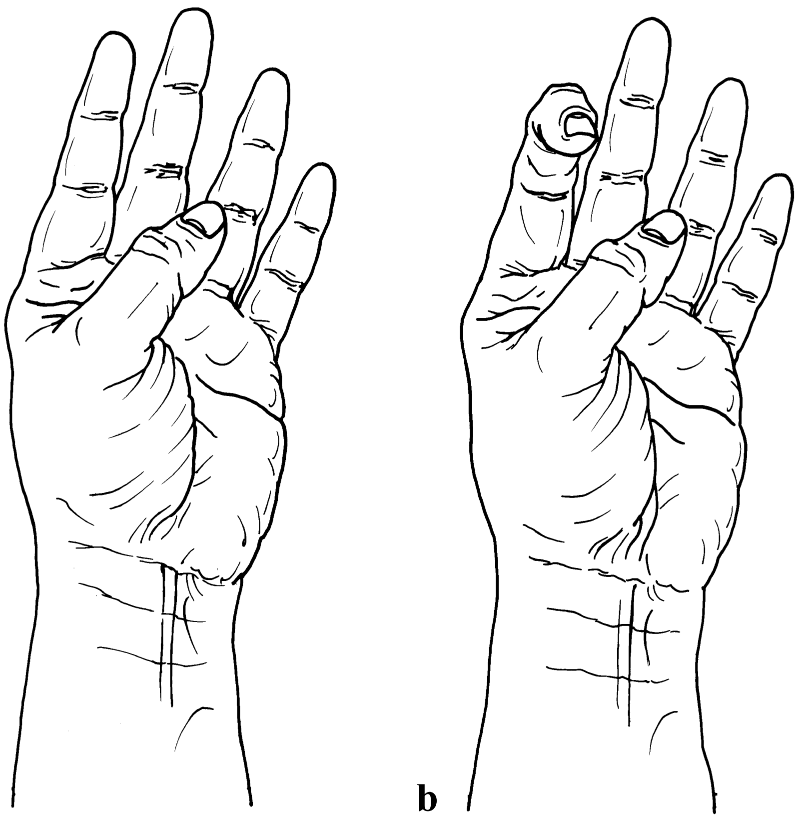
Fig. 4.7 Grind test.

Linburg Test

Indicates a congenital anomaly of the flexor pollicis longus and flexor digitorum profundus tendons that occurs in 10 to 15% of hands.

□ **Procedure:** The patient is asked to bring the thumb against the palm of the hand in a combined flexion and adduction motion with the fingers extended.

□ **Assessment:** In the presence of a congenital ligamentous connection between the flexor pollicis longus tendon and flexor digitorum profundus tendon of the index finger, this combined thumb motion will produce flexion in the distal interphalangeal joint of the index finger.



a b

Fig. 4.8 (a,b) Linburg test.

Bunnell–Littler Test

Assessment of an ischemic contracture in the intrinsic musculature of the hand.

□ **Procedure:** The patient's hand is extended. In the first part of the test, the examiner evaluates passive and active flexion in all three joints of a finger. In the second part, the examiner immobilizes the metacarpophalangeal joint in extension and again evaluates flexion in the proximal and distal interphalangeal joints of the finger.

□ **Assessment:** In the presence of an ischemic contracture of the intrinsic muscles of the hand, the patient will be unable to actively or passively flex or extend the proximal or distal interphalangeal joint when the metacarpophalangeal joint is passively immobilized in extension. This is due to shortening of the interossei. With the wrist actively or passively flexed, active flexion of the proximal and distal interphalangeal joints is possible. Usually the contracture will affect several fingers. The test allows one to distinguish an ischemic contracture from other articular changes such as joint stiffness, tendon adhesions, and tenosynovitis.

Increased pressure in the fascial compartments of the hand produces a typical deformity with slight flexion in the metacarpophalangeal joints, extension in the proximal and distal interphalangeal joints, intensification of the transverse arch of the hand, and adduction of the thumb (intrinsic plus deformity).

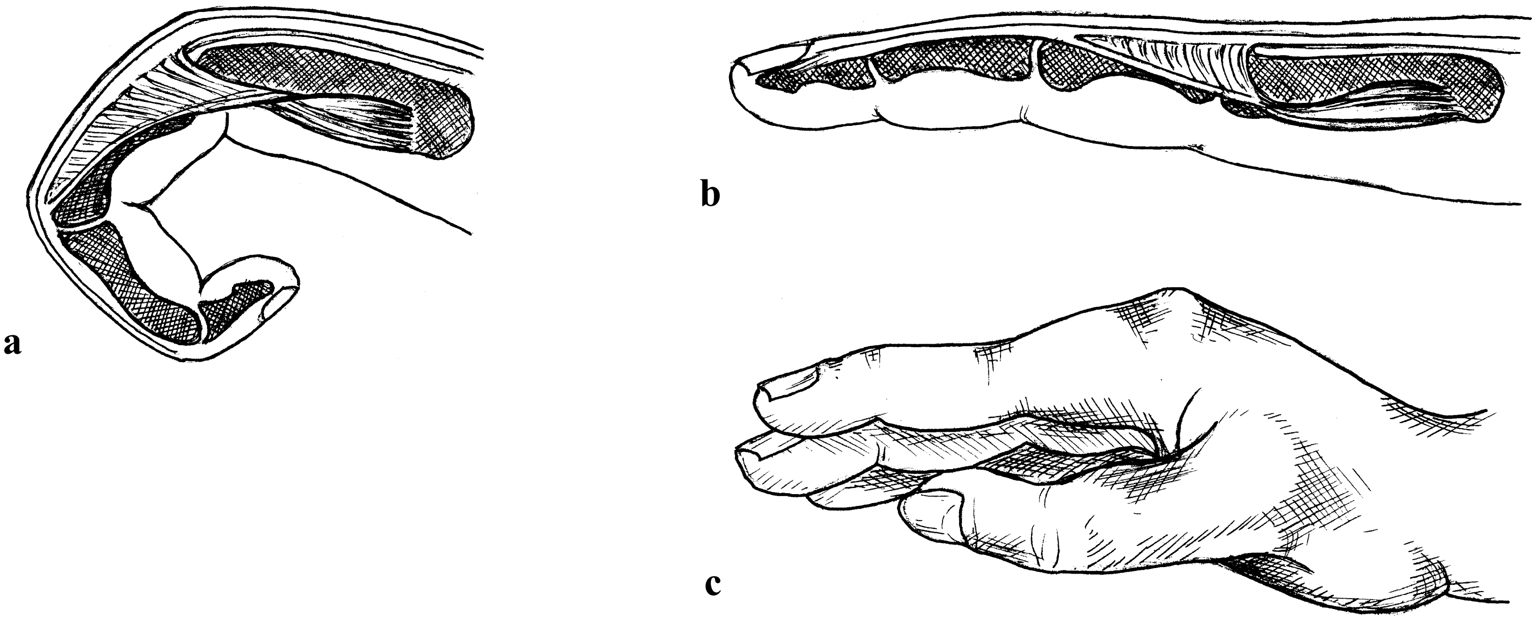


Fig. 4.9 Bunnell–Littler test. **(a)** Active and passive flexion of all finger joints is possible (first part). **(b)** Metacarpophalangeal joint is immobilized in extension; flexion of the proximal and distal interphalangeal joints is not possible (second part). **(c)** Intrinsic plus deformity.

4

Watson Test (Scaphoid Shift Test)

Tests wrist stability.

□ **Procedure:** This test is performed with the patient sitting with the elbow resting on the table. With the patient's wrist in full ulnar deviation, the examiner fixes the scaphoid bone between his or her thumb and index finger, with the thumb pressed against the distal pole of the scaphoid (tubercle) such that the scaphoid is held in extension. The examiner now radially deviates the wrist, which normally would be accompanied by scaphoid flexion but which is now prevented by the thumb's pressure on the scaphoid.

□ **Assessment:** The test is positive when the proximal pole of the scaphoid shifts to the dorsal rim of the scaphoid fossa, subluxates, and bumps against the examiner's index finger. This "snap" is accompanied by pain, demonstrating damage to the scapholunate ligament; however, it does not give any information as to the severity of the lesion.

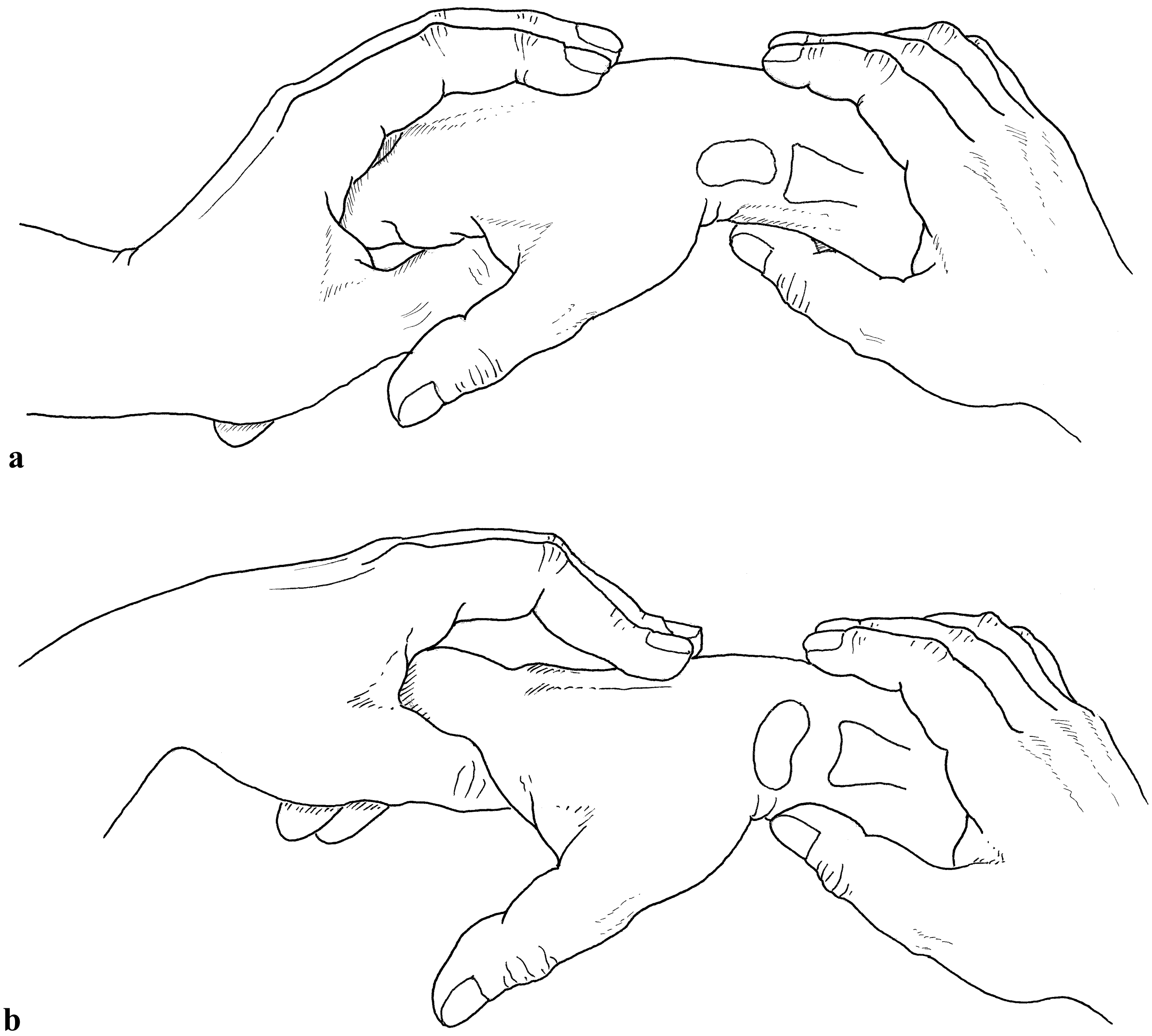


Fig. 4.10 Watson test (scaphoid shift test). **(a)** Wrist in ulnar deviation; immobilization of the scaphoid in extension. **(b)** Radial deviation of the wrist.

Scapholunate Ballottement Test

Tests wrist stability.

□ **Procedure:** The examiner holds the scaphoid and lunate tightly between the thumb and index finger of both hands while moving them relative to each other in a dorsal and a volar direction, respectively.

□ **Assessment:** Instability is present where the resistance of the scapholunate ligament complex to these shear forces is reduced. Painful shear motion indicates a ligament injury. Scapholunate instability occurs often as a result of a fall on the thumb with the forearm pronated and the wrist extended and in ulnar deviation, or as the result of an impact in ball sports. This causes a tear in the ligaments between the scaphoid and lunate. Chronic scapholunate instability can also occur without trauma, for example, secondary to removal of a ganglion or in degenerative disorders. Patients complain of severe tenderness to palpation and pain with motion in the proximal radial wrist, especially when supporting the body with the hands. They also report loss of strength and occasionally describe a snapping sound when moving the wrist into ulnar deviation.

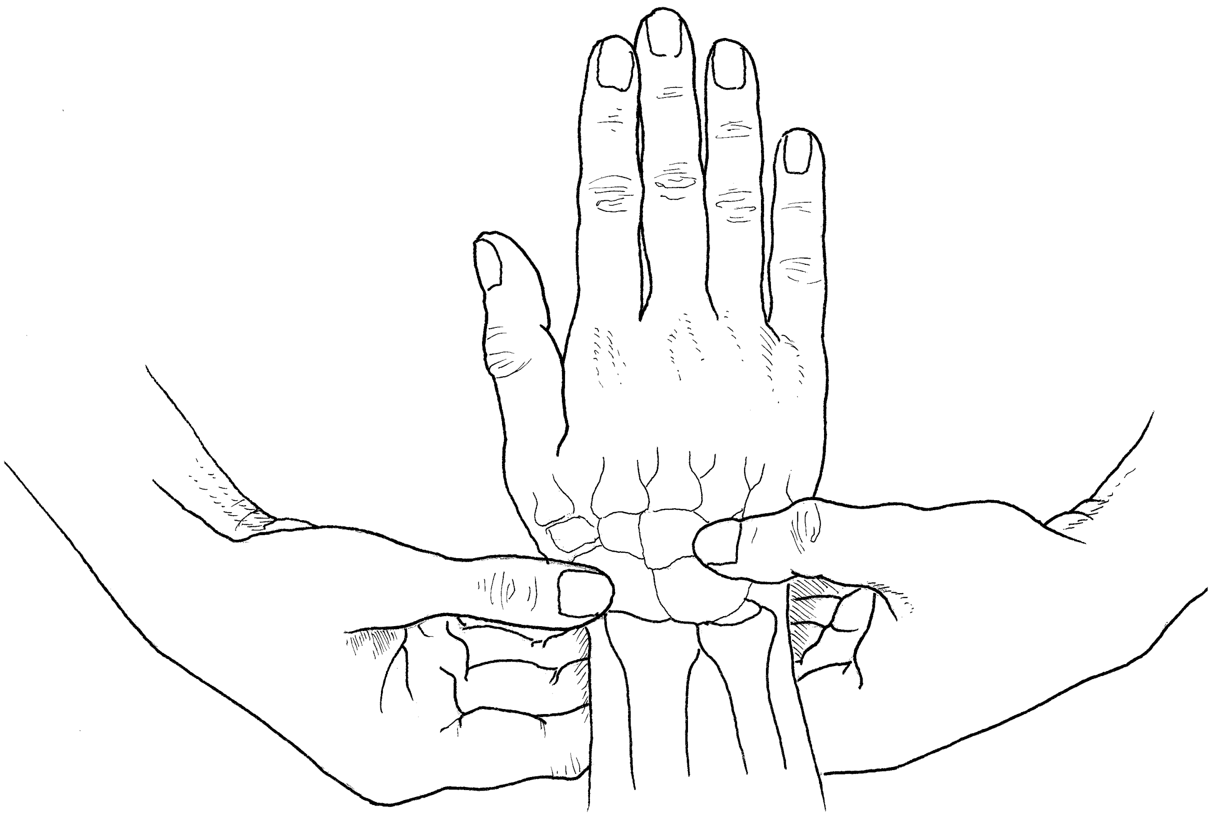


Fig. 4.11 Scapholunate ballottement test.

Finger Extension or "Shuck" Test

□ **Procedure:** The examiner holds the sitting patient's wrist flexed and asks the patient to actively extend the fingers against the resistance of the examiner's other hand, thus stressing the radiocarpal and intercarpal regions.
□ **Assessment:** The test is positive when pain occurs in the wrist, which can indicate radiocarpal or midcarpal instability, scaphoid instability, or necrosis of the lunate bone (Kienböck's disease).

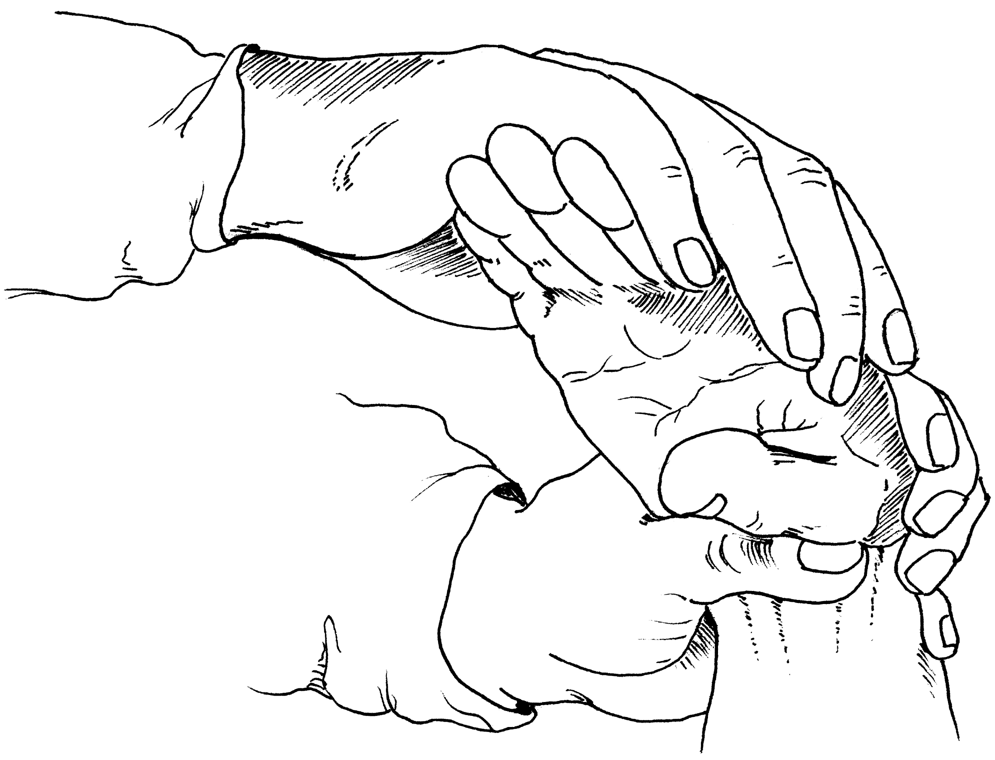


Fig. 4.12 Finger extension or "shuck" test.

Dorsal Capitate Displacement Apprehension Test

Determination of the stability of the capitate bone.
□ **Procedure:** The examiner holds the forearm of the sitting patient with one hand. The thumb of the examiner's other hand is placed over the palmar aspect of the capitate while the examiner's fingers hold the patient's fingers in a neutral position and apply a counterpressure. The examiner then pushes the capitate posteriorly with the thumb.
□ **Assessment:** If the patient complains of pain or tries to avoid the examining hand, instability or osteoarthritis of the capitate bone is implied. In cases of pronounced instability, a "snap" can sometimes be heard and felt.

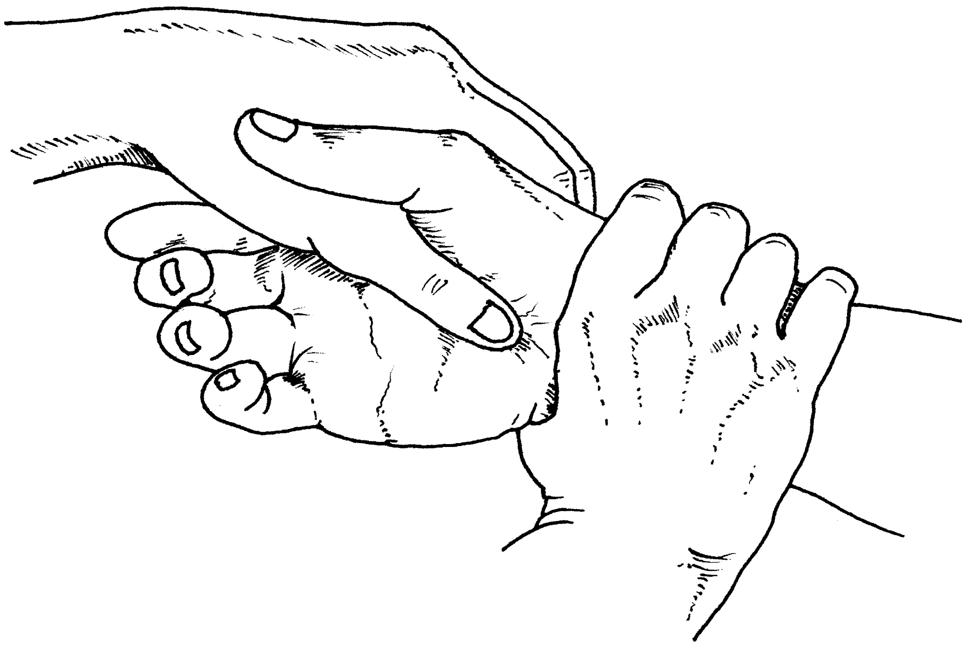


Fig. 4.13 Dorsal capitate displacement apprehension test.

Reagan Test (Lunotriquetral Ballottement Test)

Evaluation of the integrity of the lunotriquetral ligament.

▫ **Procedure:** The examiner grasps the lunate between the thumb and forefinger with one hand and the triquetrum between the fingers of the other hand. With the triquetrum fixed, the examiner then moves the lunate back and forth several times in a dorsopalmar direction.

▫ **Assessment:** In a positive test, this shear motion is painful even if instability cannot always be demonstrated.

Lunotriquetral instability can result from trauma involving hyperpronation or hyperextension. Patients report pain in the wrist. Tenderness to palpation over the triquetrolunate joint and pain with motion can be provoked, but pronation and supination do not cause any pain. The injury does not necessarily involve loss of strength. Patients occasionally describe the instability as a clicking that occurs during wrist motion.

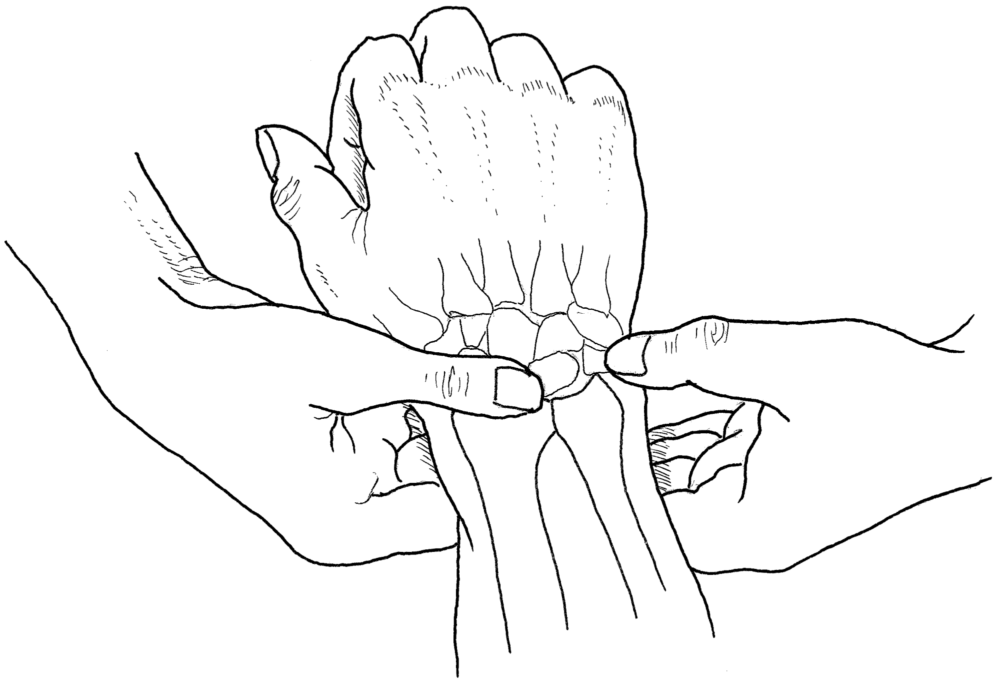


Fig. 4.14 Reagan test (lunotriquetral ballottement test).

Stability Test for a Torn Ulnar Collateral Ligament in the Metacarpophalangeal Joint of the Thumb

□ **Procedure:** The patient flexes the metacarpophalangeal joint of the affected thumb 20 to 30°. The examiner passively moves the thumb into radial deviation.

□ **Assessment:** If the thumb can be abducted, this suggests a tear in the ulnar collateral ligament of the metacarpophalangeal joint of the thumb. Known as gamekeeper's or skier's thumb, this injury is caused by forced radial deviation of the extended thumb in a fall on the hand. Stability is tested with the thumb flexed 20 to 30°. This is done to minimize the action of the accessory collateral ligament, which, if intact, could mask the tear in the collateral ligament in extension. Where the joint can be opened in extension, one may assume that a complex injury to the capsular ligament complex is present.

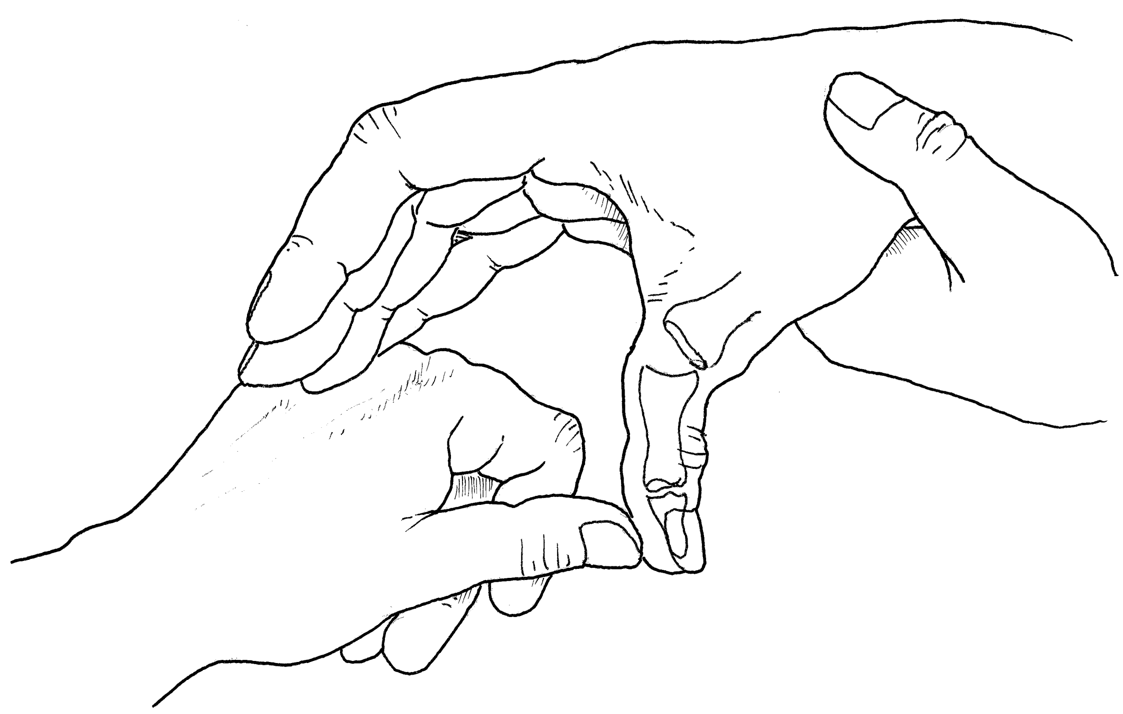


Fig. 4.15 Stability test for a torn ulnar collateral ligament in the metacarpophalangeal joint of the thumb.

Supination Lift Test

Testing for pathology of the articular disk.

- **Procedure:** The patient sits with the elbows flexed at 90° and the forearms supinated. The examiner lays his or her hands, palm down, over the patient's hands. The patient is then asked to push up against the counterpressure of the examiner's hands.
- **Assessment:** Localized pain on the ulnar side of the wrist and difficulty applying force indicate a tear of the articular disk.

The wrist (radiocarpal joint) is a true joint formed by the radius and articular disk on the one side and the proximal row of carpal bones on the other.

Fig. 4.16 Supination lift test.

Compression Neuropathies of the Nerves of the Arm

A number of compression neuropathies and entrapment syndromes can affect the nerves of the arm. Clinical tests can help differentiate between them.

Pronator Teres Syndrome

In the antecubital fossa, the median nerve runs under the bicipital aponeurosis and can become compressed in the pronator canal between the humeral and ulnar heads of the pronator teres muscle.

There are a number of possible causes for a pronator teres syndrome. These include external pressure on the forearm, muscular hypertrophy of the pronator teres from physical training, direct trauma, and pressure from a tumor. Pain, a burning sensation, and sensory deficits in the hand are typical symptoms, as are weakness in thumb opposition and weakness in flexion in the thumb, index finger, and middle finger (hand of benediction deformity). Pronation against resistance exacerbates the symptoms.

Compression Neuropathy of the Ulnar Nerve in Guyon's Canal

The ulnar artery and vein run with the ulnar nerve run through Guyon's canal, which is formed by the flexor retinaculum, pisohamate ligament, and palmar aponeurosis.

Causes of compression of the ulnar nerve include repetitive strain injuries from dorsiflexion of the wrist (as in bicycling), development of a ganglion or lipoma, fracture of the pisiform bone or the hook of the hamate, and as a result of injuries.

Sensory impairments in the ulnar aspect of the ring and little fingers and motor impairments in the hypothenar musculature are typical symptoms of this compression neuropathy.

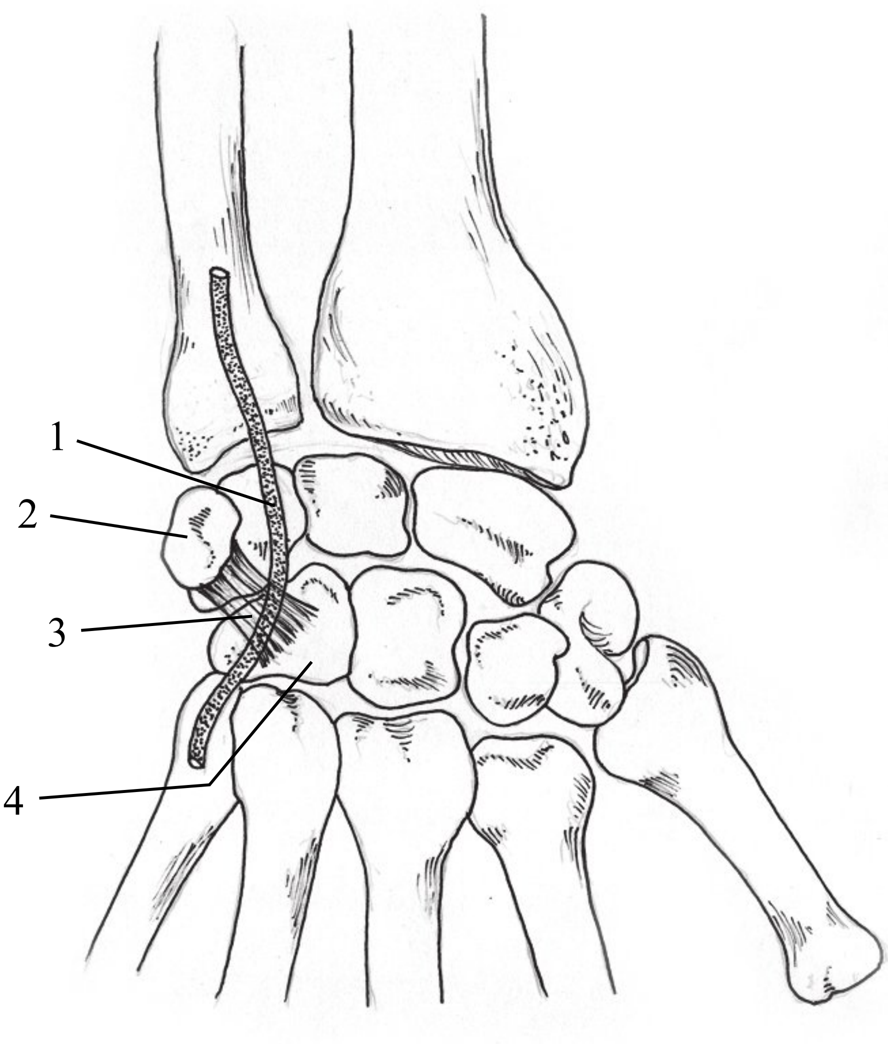


Fig. 4.17 Anatomy of Guyon's canal.
1 Ulnar nerve.
2 Pisiform bone.
3 Pisohamate ligament.
4 Hook of the hamate.

Carpal Tunnel Syndrome (CTS)

Compression of the median nerve can occur in the carpal tunnel. Formed by the carpal bones and the flexor retinaculum (transverse carpal ligament), the carpal tunnel encloses all of the finger flexor tendons and the median nerve. Causes of carpal tunnel syndrome with stenosis from increased pressure within the tunnel include local inflammatory processes such as rheumatoid arthritis, previous fractures of the base of the radius or carpal bones, tenosynovitis of the long tendons, and mechanical overuse in the workplace or in sports activities. Metabolic disorders such as gout, diabetes mellitus, and amyloidosis as well as hormonal changes (pregnancy) can also lead to CTS because of swelling within the tunnel.

Nighttime paresthesia and brachialgia are typical symptoms of nerve compression; patients awaken because of pain in the middle of the night a few hours after falling asleep. Other signs include morning stiffness and persistent sensory and, at a later stage, motor deficits in the region supplied by the median nerve, with atrophy of the thenar musculature and loss of strength when making a fist. Pinching with thumb and fingers is weak or impossible.

Differential diagnosis should consider cervical spinal cord and brachial plexus lesions, pronator teres syndrome, compression neuropathy in Guyon's canal, thoracic outlet syndrome, and osteoarthritis of the first carpometacarpal joint.

Electromyography and measurement of nerve conduction velocity by electroneurography are important studies in diagnosing carpal tunnel syndrome.

Cubital Tunnel Syndrome

The ulnar nerve courses through a bony groove posterior to the medial epicondyle. Because of its relatively superficial position, compression injuries are common. Injury, traction, inflammation, scarring, and chronic compression are the most common causes of damage to the ulnar nerve.

Sensory deficits (numbness in the little finger) and motor deficits in the area supplied by the ulnar nerve are typical findings in the presence of a nerve lesion.

Electromyography and sensory electroneurography can determine the location of the compression neuropathy.

Tests of Motor Function in the Hand

Demonstrate motor and sensory deficits in the presence of nerve lesions.

Testing the Pinch Grip

□ **Procedure:** The patient is asked to pick up a small object between the thumb and the index finger.

□ **Assessment:** Satisfactory performance requires intact sensation. The patient should repeat the test with his or her eyes closed. Unimpaired function of the lumbricals and interossei is essential for this maneuver. At an advanced stage of carpal tunnel syndrome or arthritis of the saddle joint of the thumb, the ability to pinch is diminished or entirely lost.

Testing the Key Grip

□ **Procedure:** The patient is asked to hold a key between the thumb and the side of the index finger in the normal manner.

□ **Assessment:** A sensory deficit on the radial aspect of the index finger, such as can occur in a radial nerve lesion, or arthritis of the saddle joint of the thumb render the key grip impossible.

Testing the Power Grip

□ **Procedure:** The patient is asked to hold onto a pencil with the thumb and fingers while the examiner attempts to pull the pencil away.

If finger flexion is restricted, the test is repeated using an object with a larger diameter.

□ **Assessment:** In the presence of injuries to the median or ulnar nerve, full finger flexion is not possible and strength is limited, causing a positive test.

Testing the Chuck Grip

□ **Procedure:** This precision grip maneuver is evaluated by giving the patient a small ball and having him or her hold on to it tightly.

□ **Assessment:** This maneuver tests the strength of adduction of the thumb and finger flexion, thus evaluating the median and ulnar nerves.

Testing Grip Strength

□ **Procedure:** The examiner pumps a rolled-up blood pressure cuff to 20 mm Hg (26.7 kPa) and then asks the patient to squeeze it as tightly as possible.

□ **Assessment:** Patients with normal hand function should attain a value of 200 mm Hg (about 26.7 kPa) or more. The different strengths of men, women, and children must be taken into account. This test should be performed with each hand for comparative evaluation.

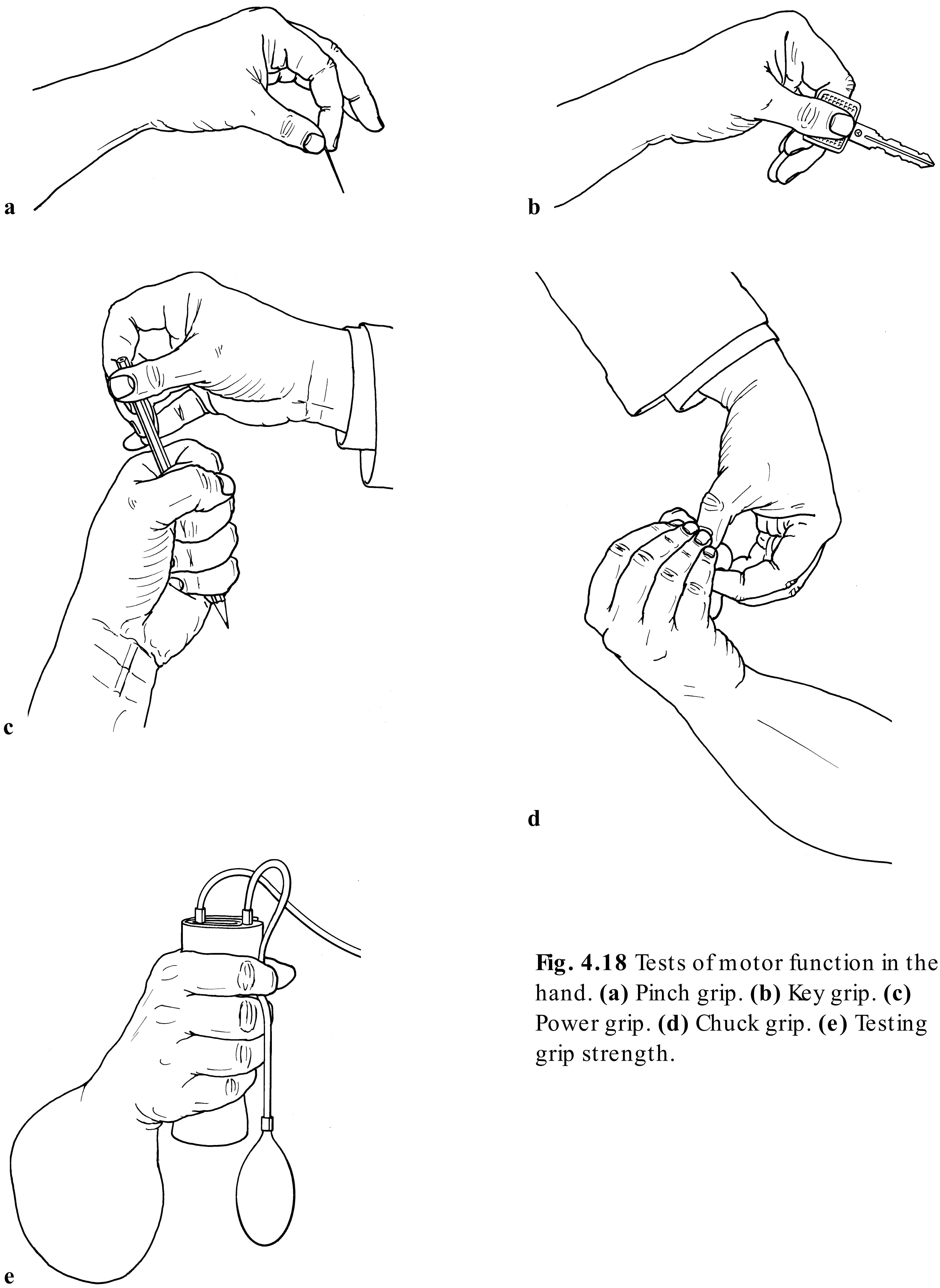


Fig. 4.18 Tests of motor function in the hand. **(a)** Pinch grip. **(b)** Key grip. **(c)** Power grip. **(d)** Chuck grip. **(e)** Testing grip strength.

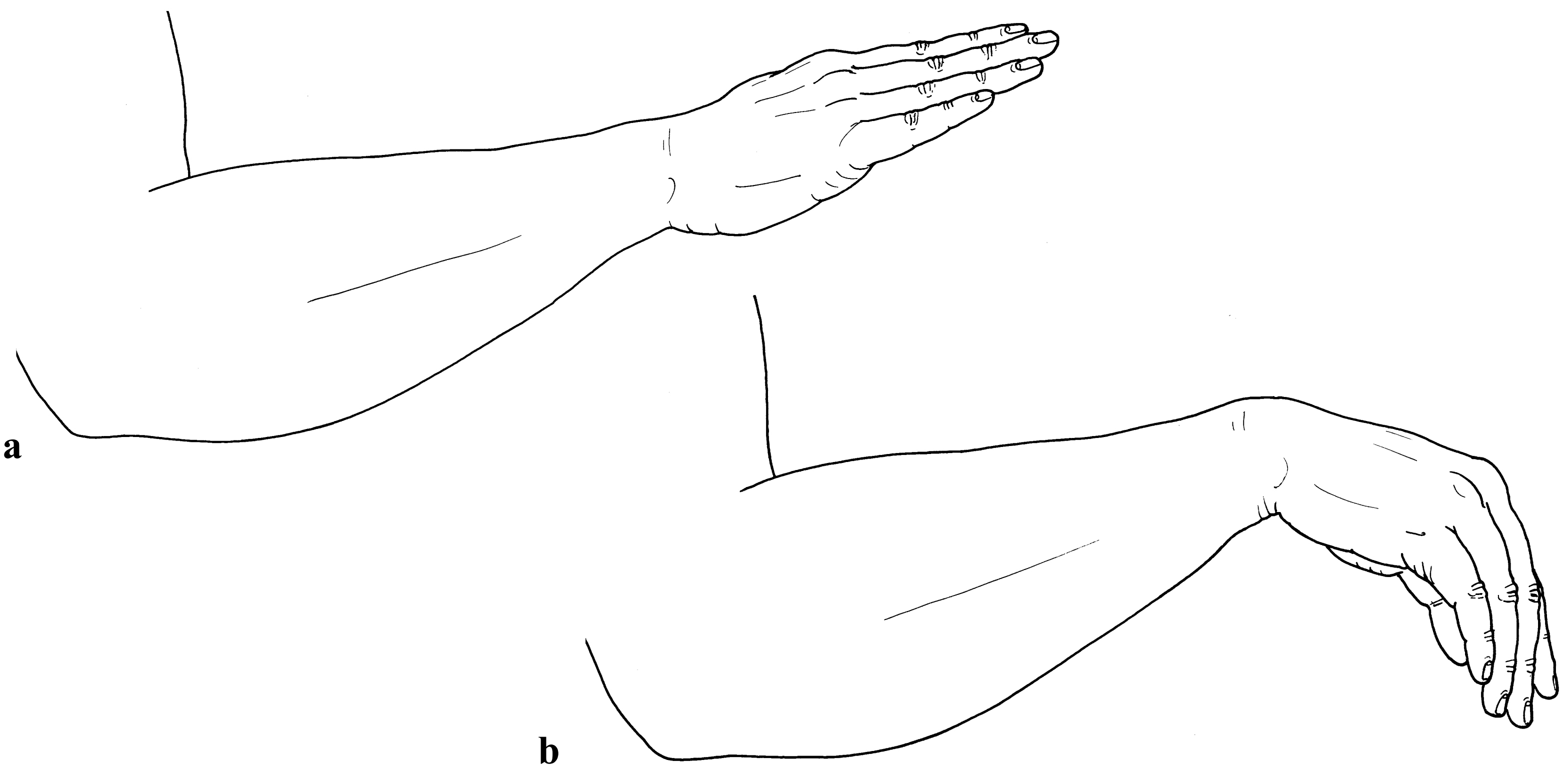
Radial Nerve Palsy Screening Test

Screening method for the assessment of radial nerve palsy.

□ **Procedure:** The patient is asked to extend his or her wrist with the elbow flexed 90°.

□ **Assessment:** In radial nerve palsy affecting the wrist extensors, the patient will be unable to extend the wrist. The hand will hang down in a deformity commonly known as a wrist drop. In a second stage of the test, the patient is asked to abduct the thumb. In radial nerve palsy, the patient will be unable to abduct the thumb because of the paralysis of the abductor pollicis longus muscle and, to a lesser extent, the extensor pollicis brevis muscle.

Radial nerve lesions usually occur secondary to humoral shaft fractures. Another cause is sleep palsy ("Saturday night palsy" or "park-bench palsy") which has a better prognosis.



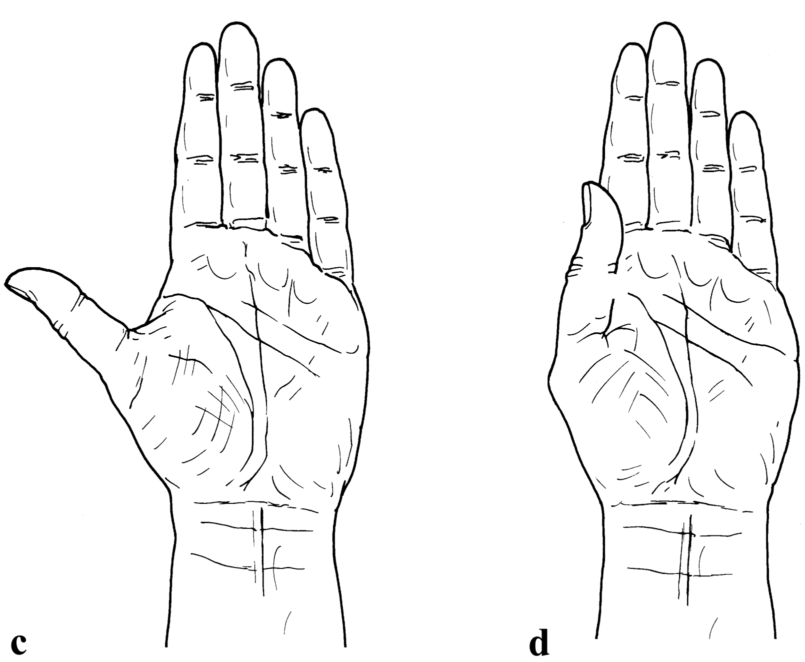


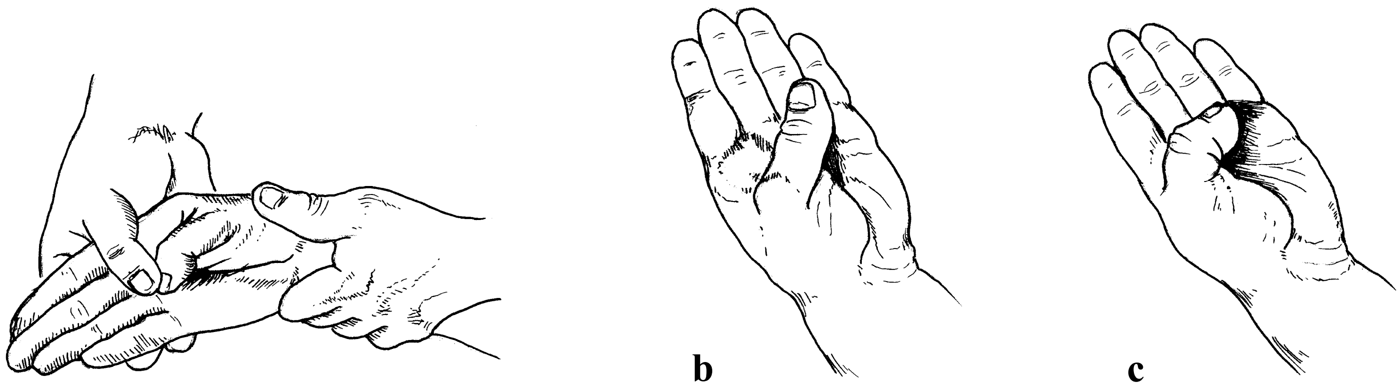
Fig. 4.19 Radial nerve palsy screening test. **(a)** Hand extension. **(b)** Wrist drop. **(c)** Thumb abduction. **(d)** Paralysis.

Thumb Extension Test

Assesses a radial nerve lesion.

Procedure: The patient is seated. The examiner grasps the patient's wrist with one hand and presses the thumb into adduction with the other hand. Then the patient is asked to extend or abduct both the metacarpophalangeal and interphalangeal joints of the thumb.

Assessment: This test requires an intact radial nerve. Where this nerve is damaged, thumb extension will be weakened or impossible as a result of paralysis of the extensor pollicis longus and brevis. In patients with degenerative joint disease or rheumatoid arthritis in the joints of the thumb, this test generally produces pain in addition to demonstrating weakness. Simple nerve palsy without degenerative changes will not produce any joint symptoms.



a b c

Fig. 4.20 Thumb extension test. **(a)** Starting position. **(b)** Normal function. **(c)** Abnormal weakness in thumb extension.

Supination Test

Evaluates a compression neuropathy of the deep branch of the radial nerve.

Procedure: The patient is seated, holding the elbow slightly flexed and the forearm pronated. The elbow is held alongside the trunk to minimize motion in the shoulder. The patient is then asked to supinate his or her forearm, at first normally and then against the examiner's resistance.

Assessment: Weakness or loss of supination of the forearm is a sign of paresis of the supinator muscle, which is supplied by the deep branch of the radial nerve.

Care should be taken not to flex the elbow too much during this test, because the biceps also participates in supination as elbow flexion increases. Despite the fact that both muscles are naturally involved in supination, increasing elbow flexion would lead to false-negative test results, because the biceps has more responsibility for supination when the elbow is significantly flexed, whereas the supinator has far greater influence on supination when the elbow is extended.

A supinator compartment syndrome occurs from hardening of the soft tissue, as a result of trauma such as dislocation of the radial head or Monteggia fracture, and rarely from an intramuscular injection.

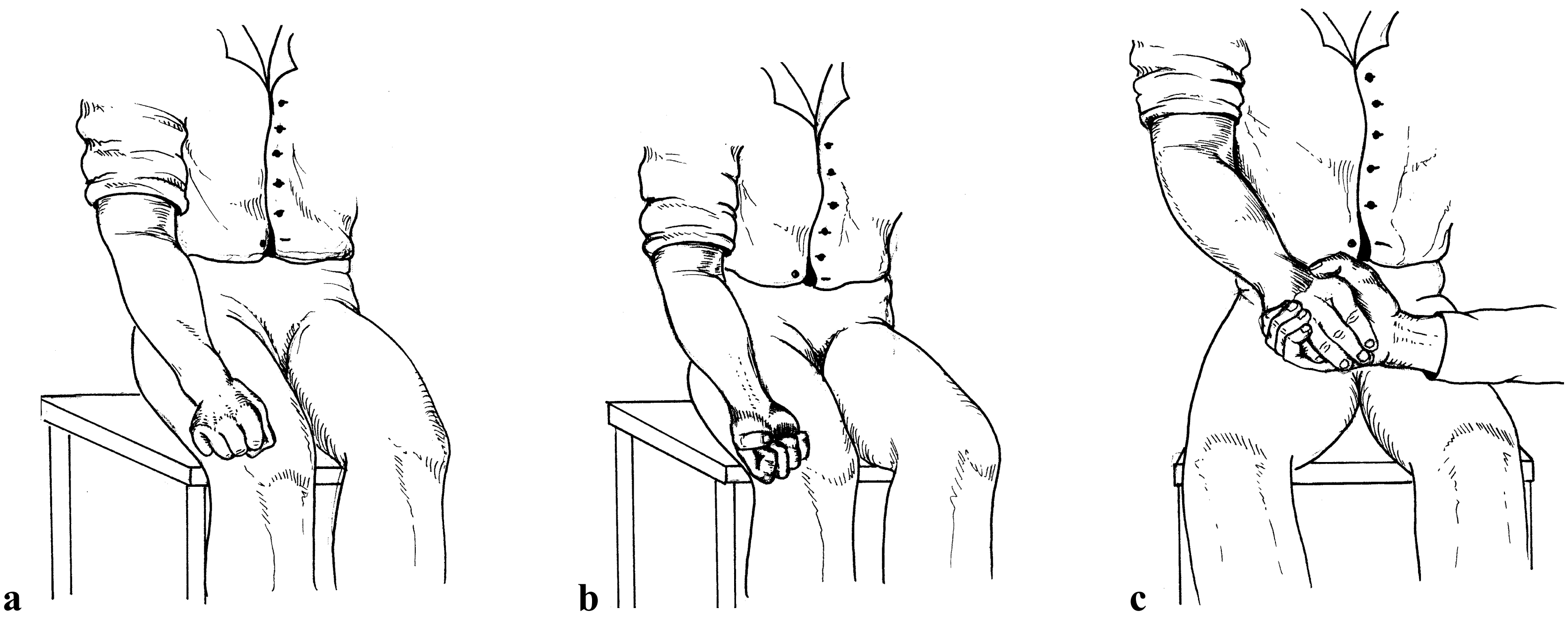


Fig. 4.21 Supination test. **(a)** Starting position. **(b)** Normal supination. **(c)** Supination against resistance.

Tinel Sign

Indicates a median nerve lesion.

□ **Procedure:** The patient's hand is slightly dorsiflexed; the dorsum of the wrist rests on a cushion on the examining table. The examiner taps the median nerve at the level of the wrist crease carefully with a reflex hammer or the index finger.

□ **Assessment:** Paresthesia and pain radiating into the hand and occasionally into the forearm as well are signs of a compression neuropathy of the median

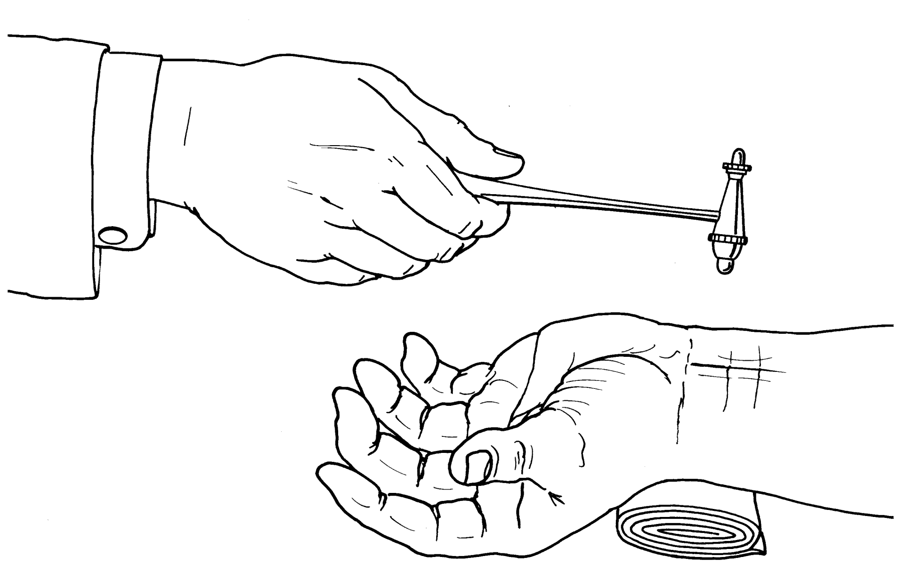


Fig. 4.22 Tinel sign.

nerve (carpal tunnel syndrome). The tingling and paresthesia must be felt distal to the point of the pressure for a positive test. The test is also an indication of the rate of regeneration of the sensory fibers of the median nerve. The test will produce a false-negative result in a chronic compression neuropathy in which nerve conductivity has already been severely reduced.

Median Nerve Palsy Screening Test

A screening test used when median nerve palsy is suspected.

□ **Procedure:** The patient is asked to oppose the tip of the thumb and the tip of the little finger. In the next step, the patient is asked to make a fist. Finally, the patient palmar flexes the hand slightly with the fingers extended.

□ **Assessment:** Paralysis of the opponens pollicis makes it impossible to bring the tips of the thumb and little finger into opposition.

Because of weakness of thumb opposition and flexion in the first three digits, the patient cannot make a fist. This produces a typical deformity (hand of benediction) in which only the ring and little fingers are flexed while the other digits remain extended. Paralysis of the opponens, abductor pollicis brevis, and flexor pollicis brevis muscles coupled with the antagonistic pull of the adductor pollicis causes the thumb to lie in the plane of the fingers. The thumbnail lies in the same plane as the fingernails, creating a deformity resembling an ape's hand, and the patient is unable to oppose the thumb.

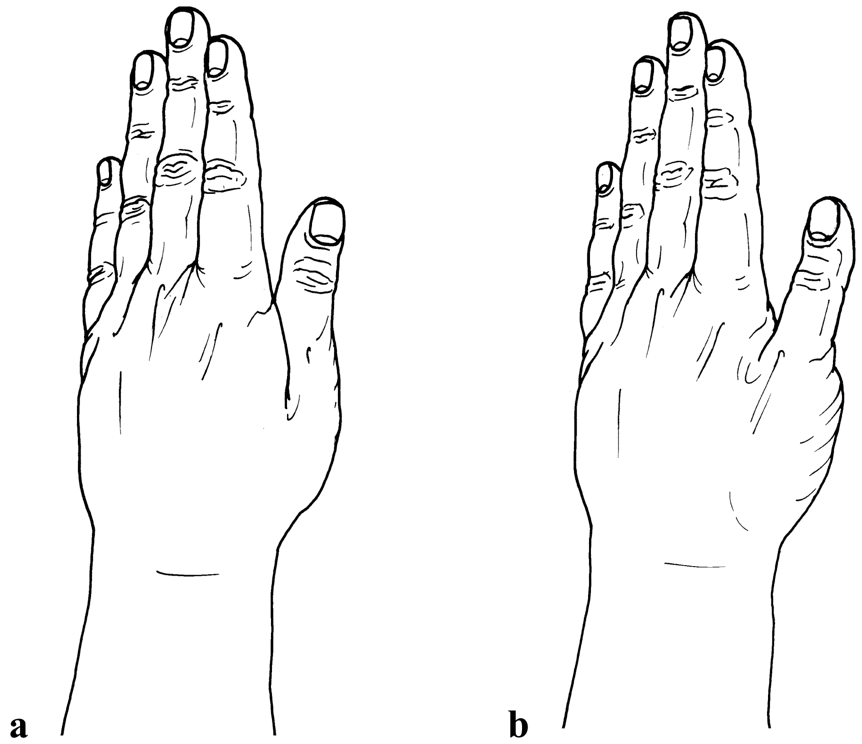


Fig. 4.23 Median nerve palsy screening test. **(a)** Normal position. **(b)** "Ape hand" deformity.

Ochsner Test

Indicates median nerve palsy.

□ **Procedure:** The patient is asked to join his or her hands with the fingers interlocked.

□ **Assessment:** If median nerve palsy is present, the patient will be unable to flex the index and middle fingers due to partial paralysis of the flexor digitorum profundus.

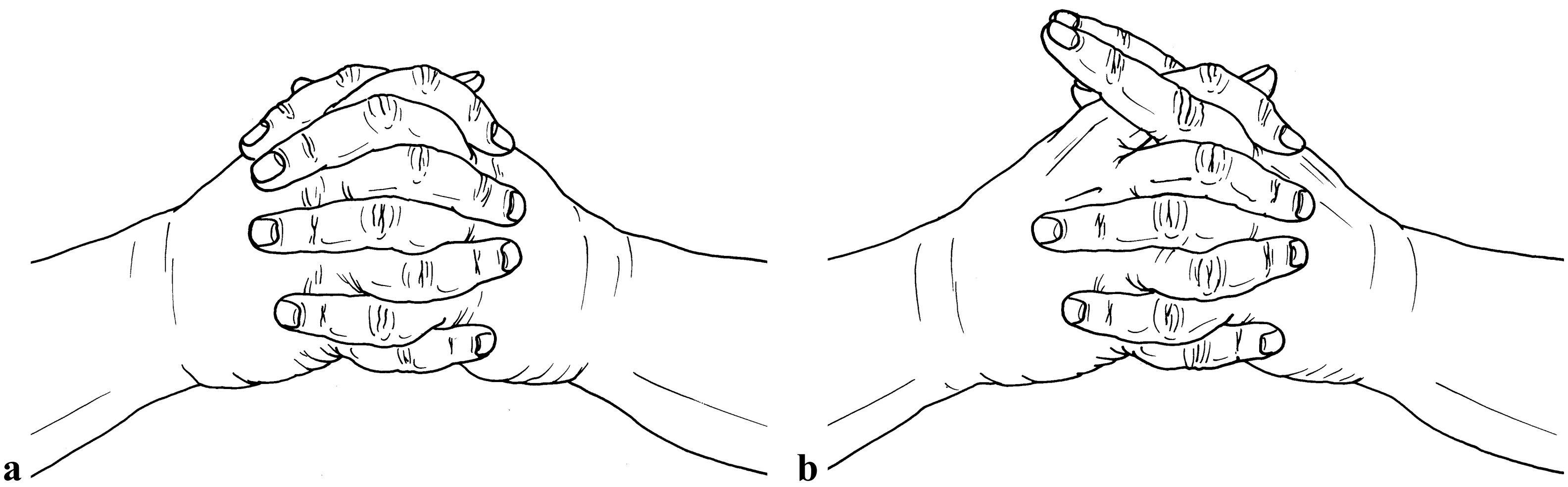


Fig. 4.24 Ochsner test. **(a)** Normal position. **(b)** The index and middle fingers extended due to weakness in the flexors.

Carpal Tunnel Sign

Indicates damage to the median nerve.

□ **Procedure:** The patient is asked to keep his or her wrists completely flexed for 1 to 2 minutes.

□ **Assessment:** Paresthesia that occurs or worsens in the region supplied by the median nerve is a sign of carpal tunnel syndrome.

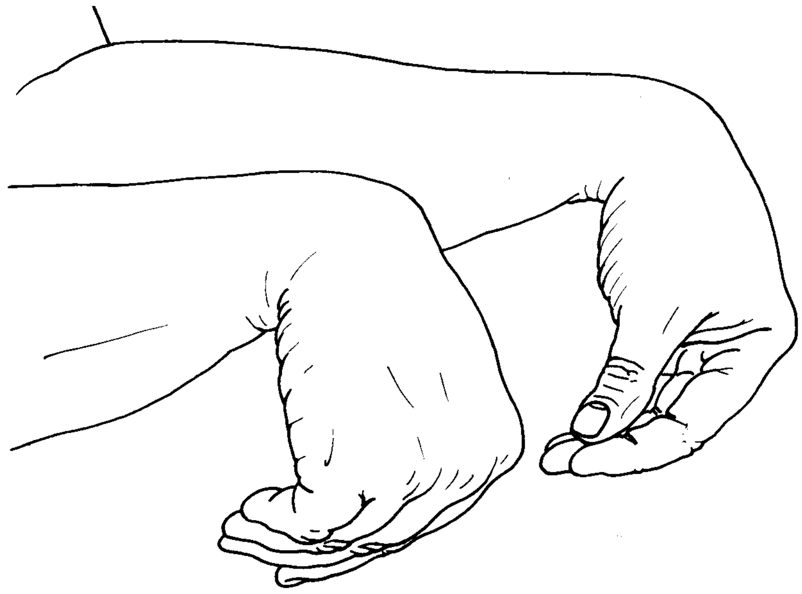


Fig. 4.25 Carpal tunnel sign.

Phalen Test

Indicates damage to the median nerve.

□ **Procedure:** The "wrist flexion sign" is evaluated by having the patient drop his or her hands into palmar flexion and then maintain this position for about 1 to 2 minutes. Pressing the dorsa of the hands together increases pressure in the carpal tunnel.

□ **Assessment:** Pressing the dorsa of the hands together will often lead to paresthesia in the area supplied by the median nerve in normal patients as well, not just in those with carpal tunnel syndrome. Patients with carpal tunnel syndrome will experience worsening of symptoms in the Phalen test. Like the Tinel sign, this test can produce false-negative results in the presence of chronic neuropathy.

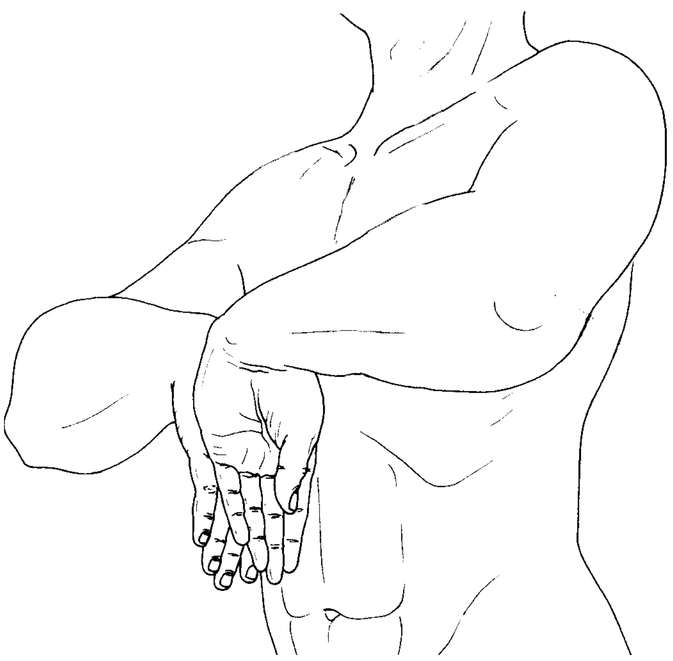


Fig. 4.26 Phalen test.

Nail Sign

Indicates damage to the median nerve.

□ **Procedure:** The patient is asked to touch his or her thumb to the tip of the little finger.

□ **Assessment:** Median nerve palsy will produce paralysis of the opponens pollicis. The thumb cannot be opposed but will only move along an arc in adduction toward the palm.

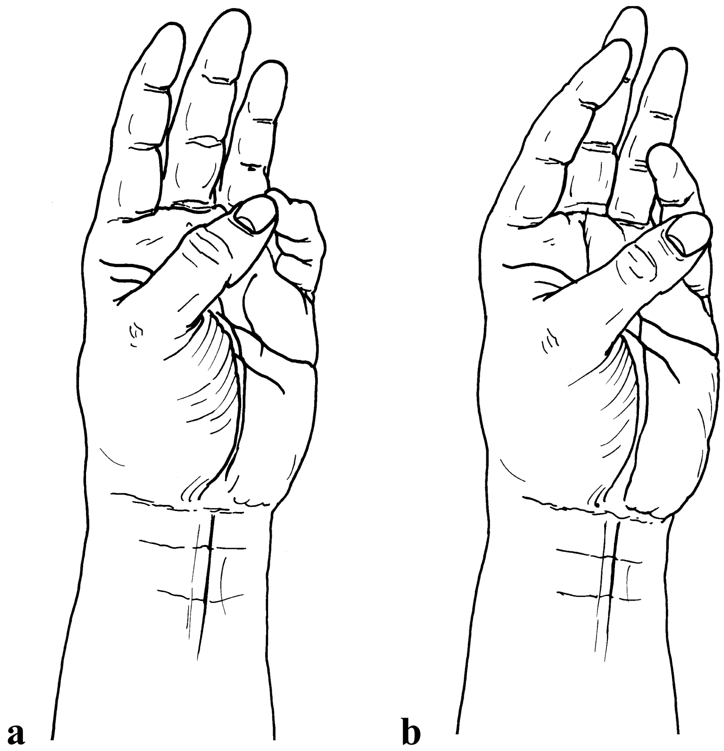


Fig. 4.27 Nail sign. **(a)** Normal. **(b)** Abnormal position due to weakened opposition of the thumb.

Bottle Test

Indicates median nerve palsy.

□ **Procedure:** The patient is asked to grasp a bottle in each hand between the thumb and index finger.

□ **Assessment:** In paralysis of the abductor pollicis brevis, the web between the thumb and index finger will not be in contact with the surface of the bottle. The patient will be unable to hold the bottle between the thumb and index finger in such a way that the hand is in continuous contact with the circumference of the bottle.

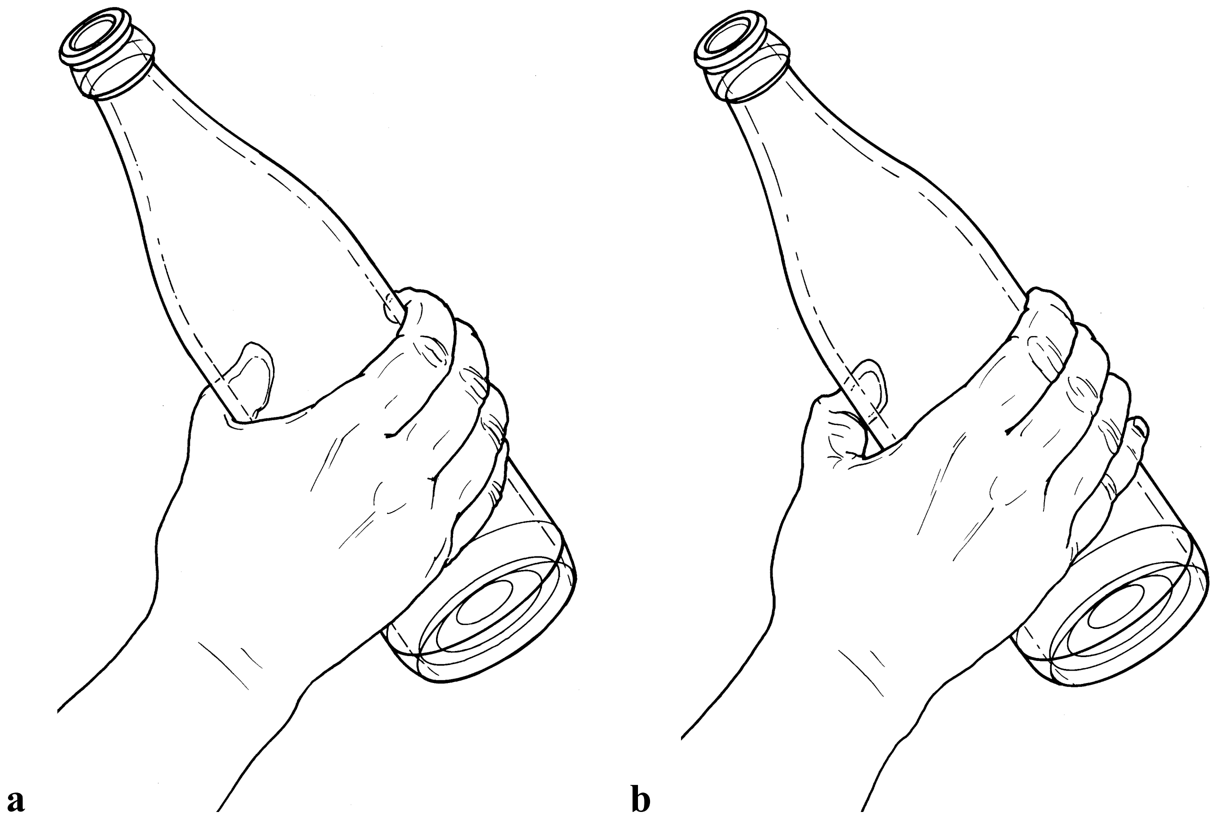


Fig. 4.28 Bottle test. **(a)** Normal. **(b)** Abnormal.

Reverse Phalen Test

Indicates carpal tunnel syndrome.

□ **Procedure:** The seated patient is asked to press both hands together in maximum dorsiflexion and to maintain this position for 1 minute.

□ **Assessment:** This position increases the pressure in the carpal tunnel. Paresthesia in the region supplied by the median nerve is a sign of carpal tunnel syndrome. The reverse Phalen test is less reliable than the Phalen test.

Fig. 4.29 Reverse Phalen test.

Pronation Test

Assessment of pronator teres and pronator quadratus pathology.

□ **Procedure:** The patient is seated with both hands and forearms in supination on the examining table. The examiner asks the patient to pronate his or her forearms, initially normally and then against the resistance of the examiner's hand.

□ **Assessment:** Weakness in active pronation against resistance in one arm as compared with the contralateral side indicates a median nerve lesion. The lesion normally lies at the level of the elbow. In the presence of a median nerve lesion distal to the elbow, the patient may be able to actively pronate the forearm against resistance because the pronator teres is still largely functional.

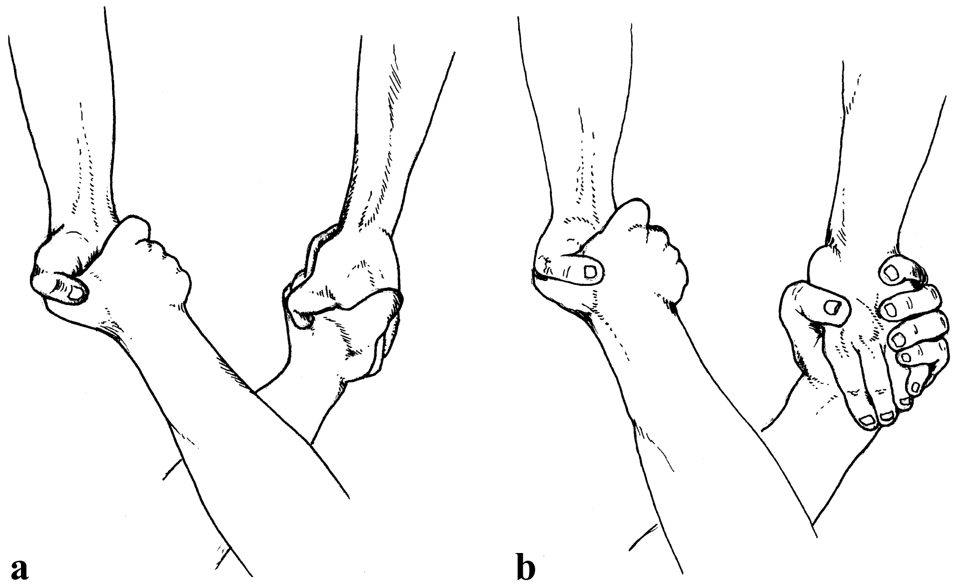


Fig. 4.30 Pronation test. **(a)** Starting position. **(b)** Weakness in pronation of the right arm.

Froment Sign

Indicates a cubital tunnel syndrome.

□ **Procedure:** The patient is asked to hold a piece of paper between the thumb and index finger (pinch mechanism) against either the pull of the patient's contralateral hand or the pull of the examiner's hand. The muscle for this motion is the adductor pollicis, which is supplied by the ulnar nerve.

□ **Assessment:** Where there is weakness or loss of function in this muscle, the interphalangeal joint of the thumb will be flexed due to contraction of the flexor pollicis brevis, which is supplied by the median nerve. There may be additional weakness or paralysis of the interosseous muscles and/or the third and fourth lumbrical muscles resulting in the typical claw hand deformity. Occasional volar hypesthesia on the ring and little fingers is also a characteristic sign.

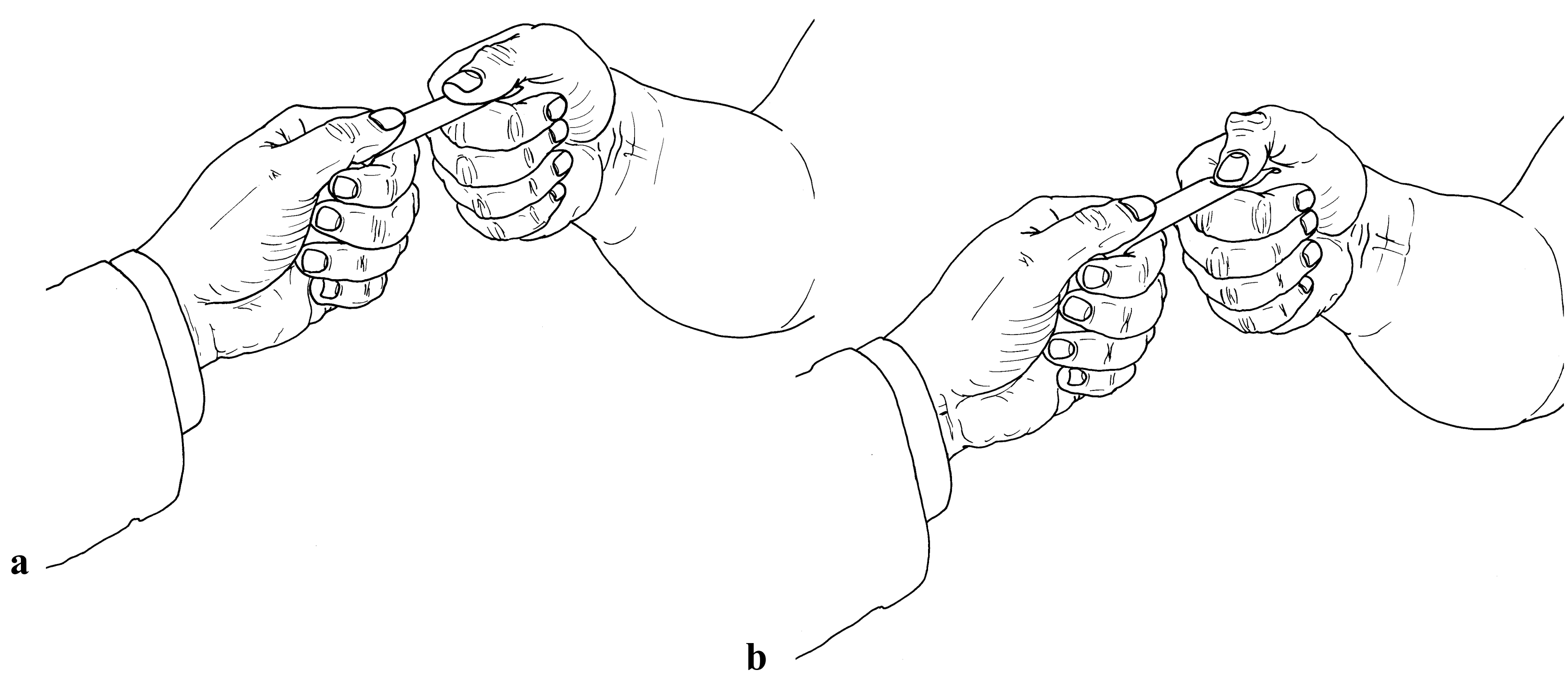


Fig. 4.31 Froment sign. **(a)** Normal. **(b)** Abnormal.

Ulnar Nerve Palsy Screening Test

Indicates ulnar nerve palsy.

□ **Procedure:** The patient is asked to make a fist.

□ **Assessment:** Where the ring and little fingers remain extended, flexion in the metacarpophalangeal and proximal interphalangeal joints of these fingers is not possible. This is a sign of paralysis of the interossei. Patients with a long history of chronic ulnar nerve palsy will exhibit significant muscle atrophy between the fourth and fifth and between the first and second digital rays of the hand.

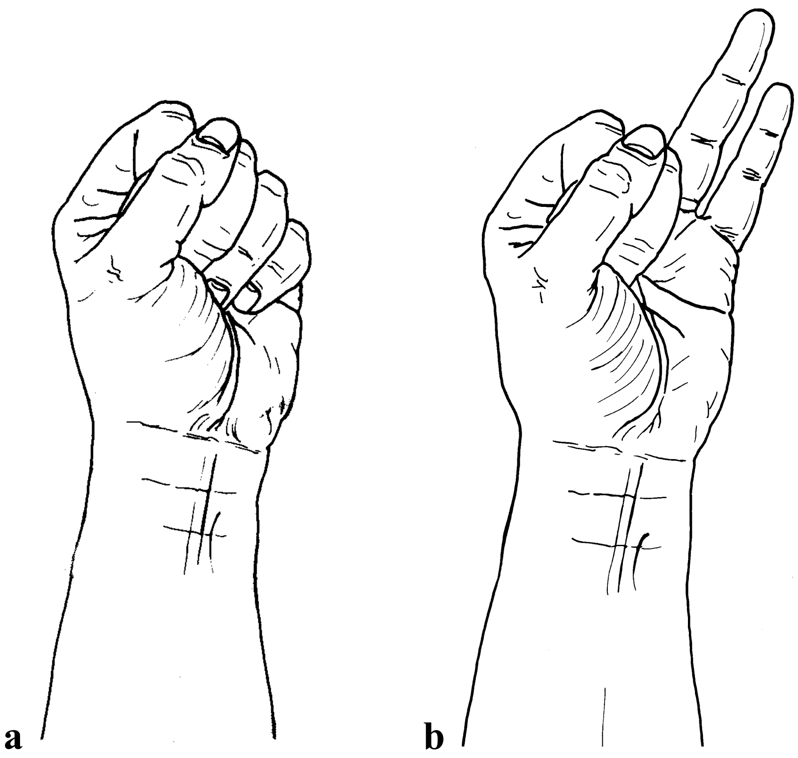


Fig. 4.32 Ulnar nerve palsy screening test. **(a)** Normal. **(b)** Abnormal with loss of flexion in the ring and little fingers.

Intrinsic Test

Indicates compression neuropathy of the ulnar nerve.

□ **Procedure:** The patient is asked to hold a piece of paper between the ring finger and the little finger. The examiner attempts to pull the piece of paper away from the patient.

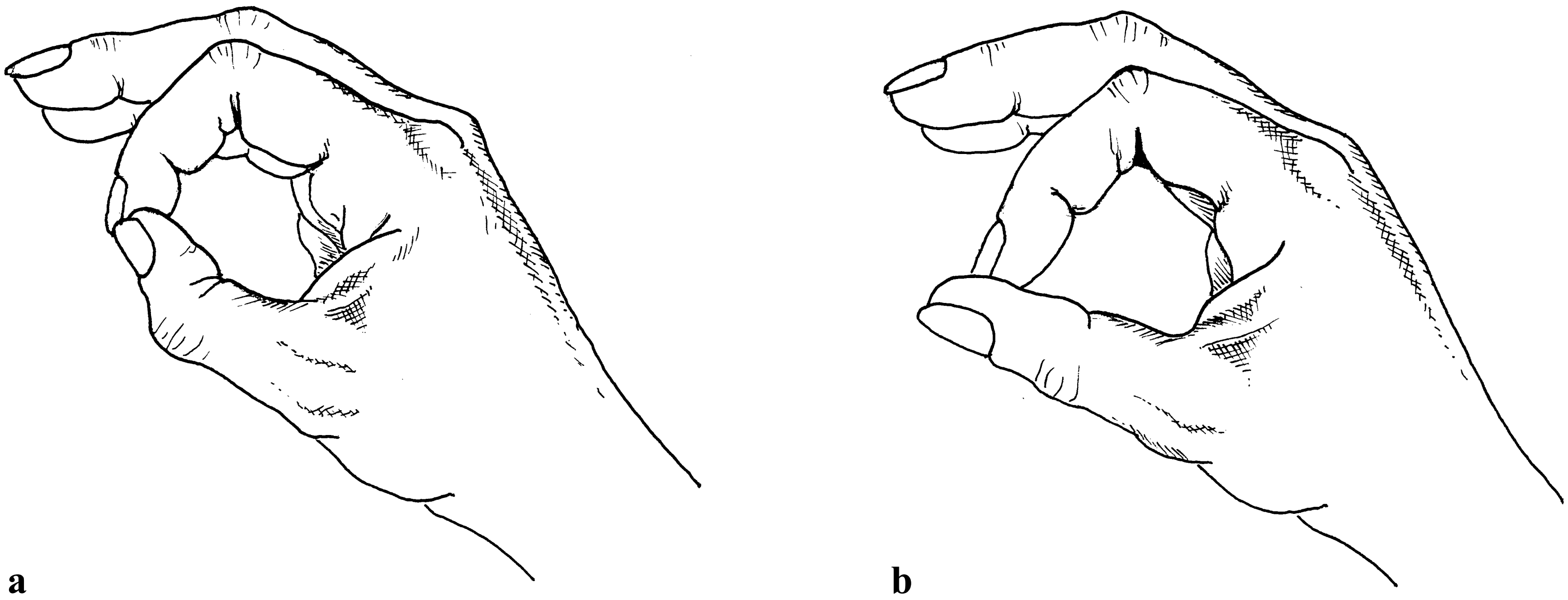
□ **Assessment:** In the presence of ulnar nerve neuropathy, adduction in the little finger will be limited and the patient will be unable to hold on to the paper. The test should be performed on both hands for comparison. Compression neuropathy of the ulnar nerve can occur in the carpal tunnel, in the elbow, and in Guyon's canal in the wrist. A positive Tinel sign and paresthesia on the ring and little fingers are additional signs of compression. Complete ulnar nerve palsy results in loss of function in the intrinsic muscles of the hand. The fingers are then hyperextended in the metacarpophalangeal joints and flexed in the proximal and distal interphalangeal joints.

Fig. 4.33 Intrinsic test.

O Test (Pinch Sign)

□ **Procedure:** The pinch mechanism is a combined motion involving several muscles. Normally the thumb and index finger form the shape of an "O." With normal function in the muscles involved, the examiner will be unable to change the shape of the "O" by pulling with his or her own index finger inserted between the patient's thumb and index finger.

□ **Assessment:** In an anterior interosseous nerve syndrome with paralysis of the flexor digitorum profundus of the index finger and flexor pollicis longus, the thumb and index finger remain extended in the distal interphalangeal joints. The patient is then unable to form a proper "O" with the thumb and index finger.



a b

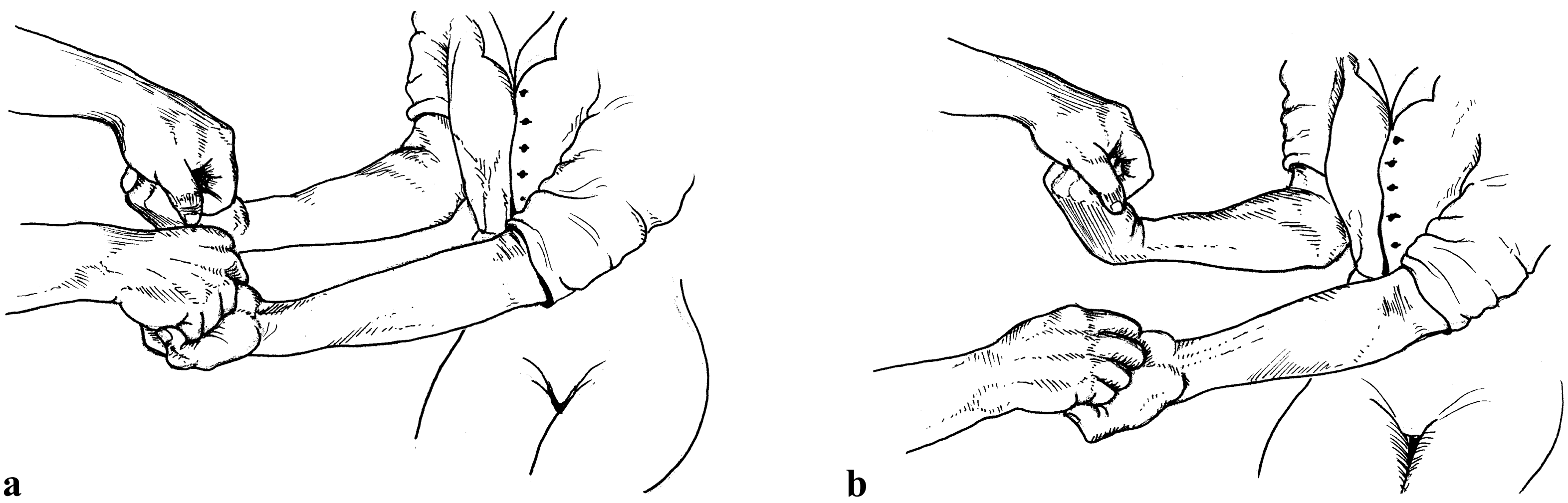
Fig. 4.34 O test. **(a)** Normal. **(b)** Abnormal result with paralysis of the flexor digitorum profundus of the index finger and flexor pollicis longus.

Wrist Flexion Test

Assessment of a distal nerve lesion in the forearm.

□ **Procedure:** The patient is seated with both forearms supinated. The examiner asks the patient to flex his or her wrists, first normally and then against the resistance of the examiner's hands.

□ **Assessment:** Weakness in active flexion against resistance indicates paresis or paralysis of the flexors in the forearm, especially the flexor carpi radialis. Weakness in this motion without resistance is a sign of complete paralysis. Weakness in active flexion against resistance indicates a problem with the median nerve at the level of the elbow or further proximally. Complete inability to flex the wrist against resistance could indicate a lesion involving both the median and ulnar nerves.



a b

Fig. 4.35 Wrist flexion test. **(a)** Normal. **(b)** Abnormal with weakness in active flexion of the left forearm.

5 Hip

Hip pain can have any number of causes. In children and adolescents, it is usually a sign of a serious disorder and therefore always requires a thorough diagnostic work-up.

Patients usually report hip pain in the groin or posterior to the greater trochanter, occasionally radiating into the medial aspect of the thigh as far as the knee. For this reason, especially in children, a hip disorder can be easily misinterpreted as a knee disorder. The differential diagnosis should include disorders of the adductor tendons, lumbar spine, and, especially, the sacroiliac joints.

Many of the hip disorders associated with pain correlate with a certain age group. Frequent causes of pain in the hip include chronic hip dislocations and Legg–Calvé–Perthes disease in children and slipped capital femoral epiphysis in adolescents. Hip dysplasia and osteoarthritis of the hip are the main causes of hip pain in adults.

Untreated or insufficiently treated congenital hip dislocation with persisting acetabular dysplasia is one of the most frequent causes of subsequent degenerative joint disease. Pain on walking, which patients usually describe as groin pain, is often attributable to hip dysplasia.

Aseptic necrosis of the femoral head, injuries, the "normal" aging process, and rheumatic and metabolic disorders are other factors that can lead to degenerative hip disease. The hip joint is surrounded by a strong muscular envelope. Inspection alone will provide only a modest amount of diagnostic information about the condition of the joint. Even a significant joint effusion may escape detection. The position of the legs (flexion contracture of the hip, malrotation, or leg shortening) and the position of the spine (scoliosis or lordosis) are important in evaluating the pelvis; their abnormal positions may actually be caused by a hip disorder and can allow one to draw conclusions about the condition of the hip.

The normal pelvis is tilted anteriorly, producing lordosis in the lumbar spine. Contracture of the hip results in an abnormal position of the legs, pelvis, and back. This is usually more apparent when the patient is standing upright than when lying down. Increased lumbar lordosis can be due to a flexion contracture in the hip; this contracture may be compensated for by an increased anterior tilt of the pelvis and increased lordosis. Actual and apparent leg shortening also significantly influences leg position and gait. When examining leg length, one must consider the possibility of apparent lengthening or shortening due to an abduction or adduction contracture. In the presence of an abduction contracture of the leg at the hip, the patient can only bring his or her legs into parallel alignment by tilting the pelvis. This pushes the normal hip upward, making that leg appear shortened. The adduction contracture has an analogous effect, although in this case the affected leg appears shortened. If the patient does not want to stand with one leg on tiptoe to compensate for the shortening, he or she will have to flex the contralateral knee. This produces an additional flexion in the hip that the patient can compensate for by increasing the anterior tilt of the pelvis.

Abnormal positioning of the pelvis due to hip disorders usually results in changes to the spine in the form of lumbar scoliosis and spinal torsion or a compensatory curvature of the posterior lumbar section of the spine.

Assessment of the patient's gait allows the examiner to identify gait abnormalities due to articular causes (osteoarthritis or inflammation) and/or muscular causes. In Duchenne antalgic gait, the patient attempts to reduce the load on the hip that causes the pain. In a Trendelenburg gait, weakness of the hip abductors, primarily the gluteal musculature, causes the pelvis to dip toward the unaffected side in the stance phase. In a compensatory limp with leg shortening, the upper body is shifted slightly over the leg in the stance phase. Otherwise, the gait is relatively smooth. Arthrodesis of the hip does not produce a true limp in the sense that the pelvis dips in the stance phase. Instead, the increased tilt of the pelvis in the sagittal plane, as it moves from hyperlordosis into lumbar kyphosis, produces femoral anteversion in the swing phase.

Other function tests are of help in the closer assessment of hip disorders and in clarifying their cause and confirming the diagnosis.

Range of Motion of the Hip (Neutral-Zero Method)

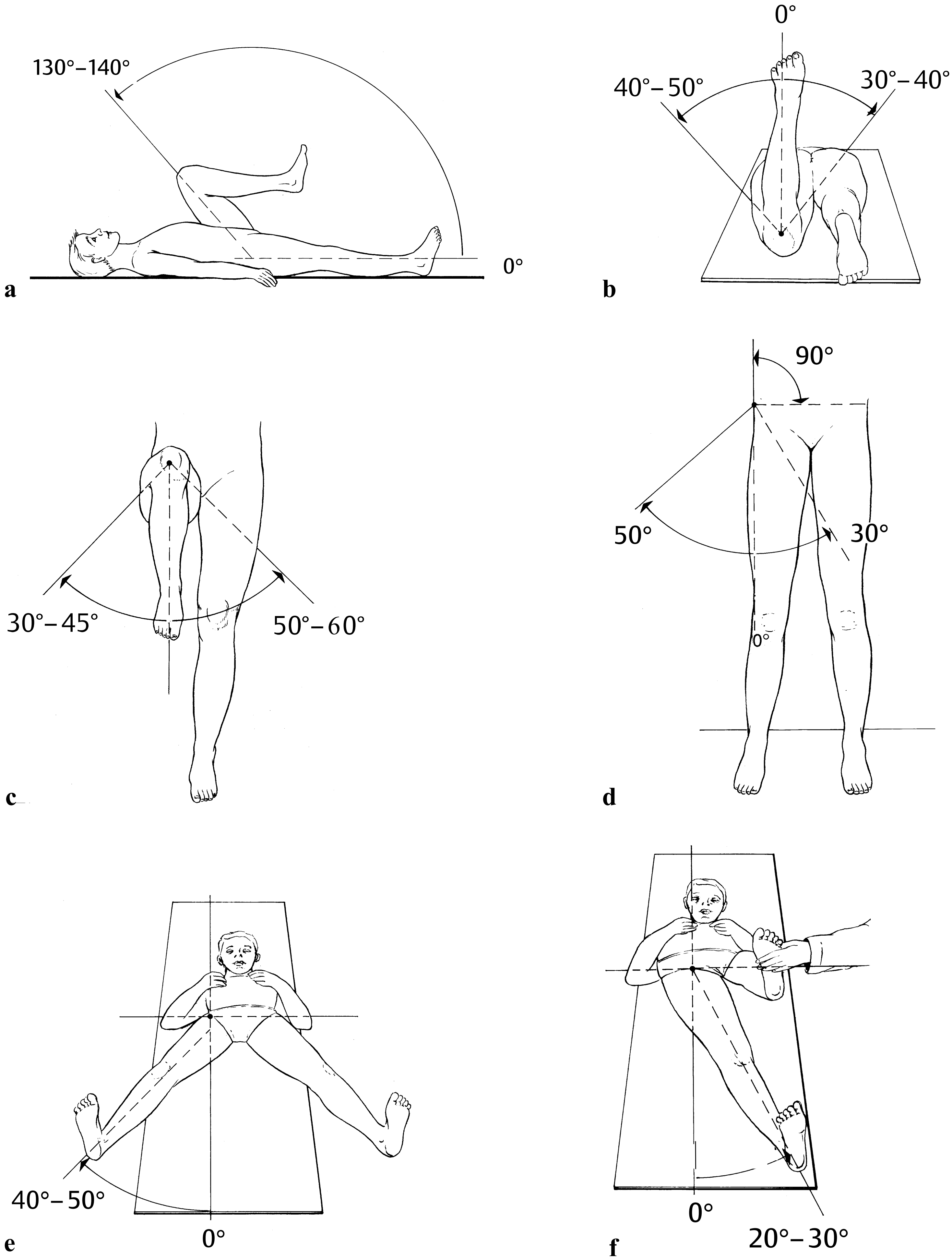


Fig. 5.1 (a) Flexion and extension of the hip, supine (hyperextension 10°). **(b, c)** Internal and external rotation of the hip: prone, with the hip extended **(b)**; supine, with the hip flexed **(c)**. **(d)** Abduction and adduction of the hip. **(e, f)** Abduction and adduction of the hip.

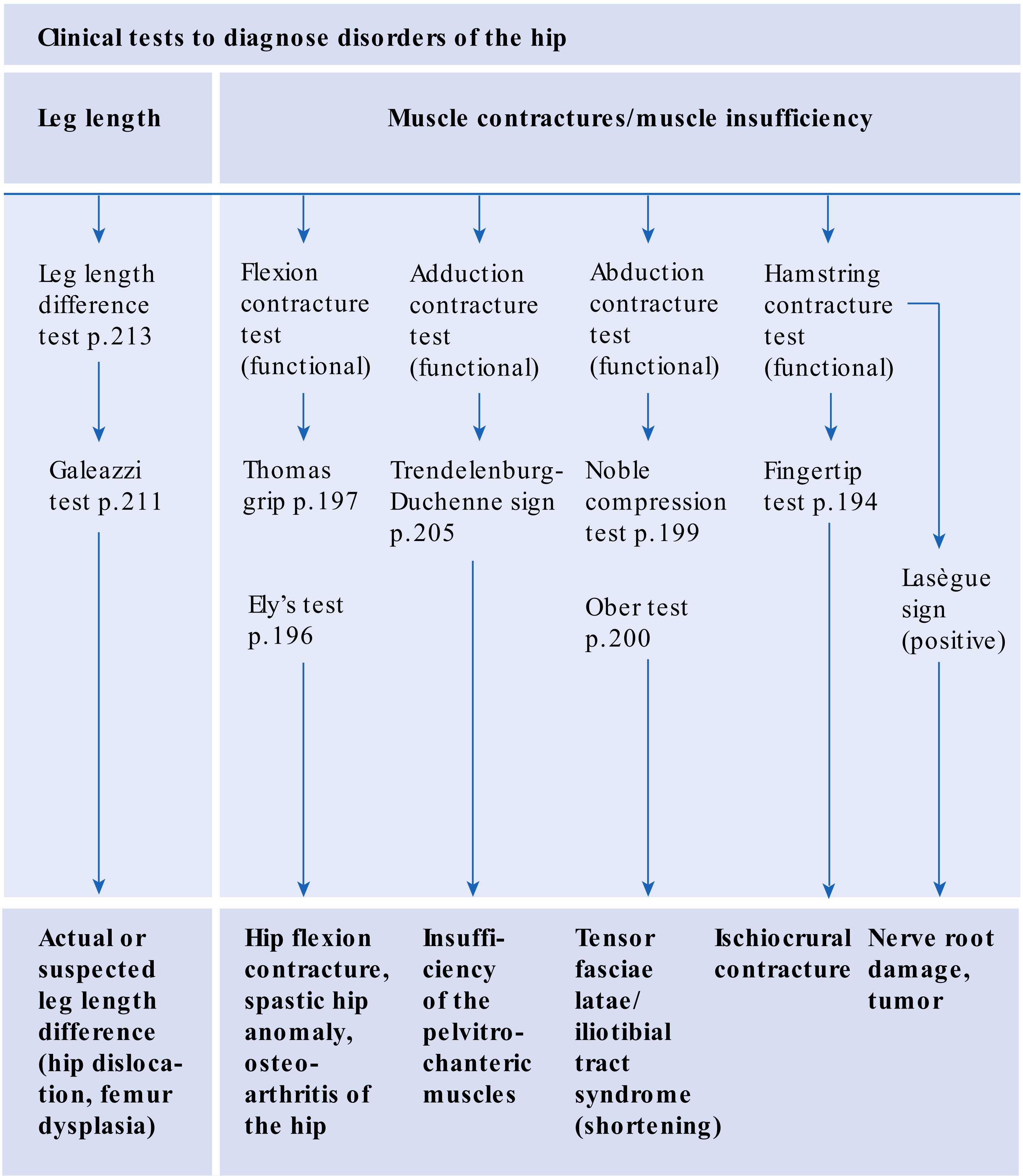
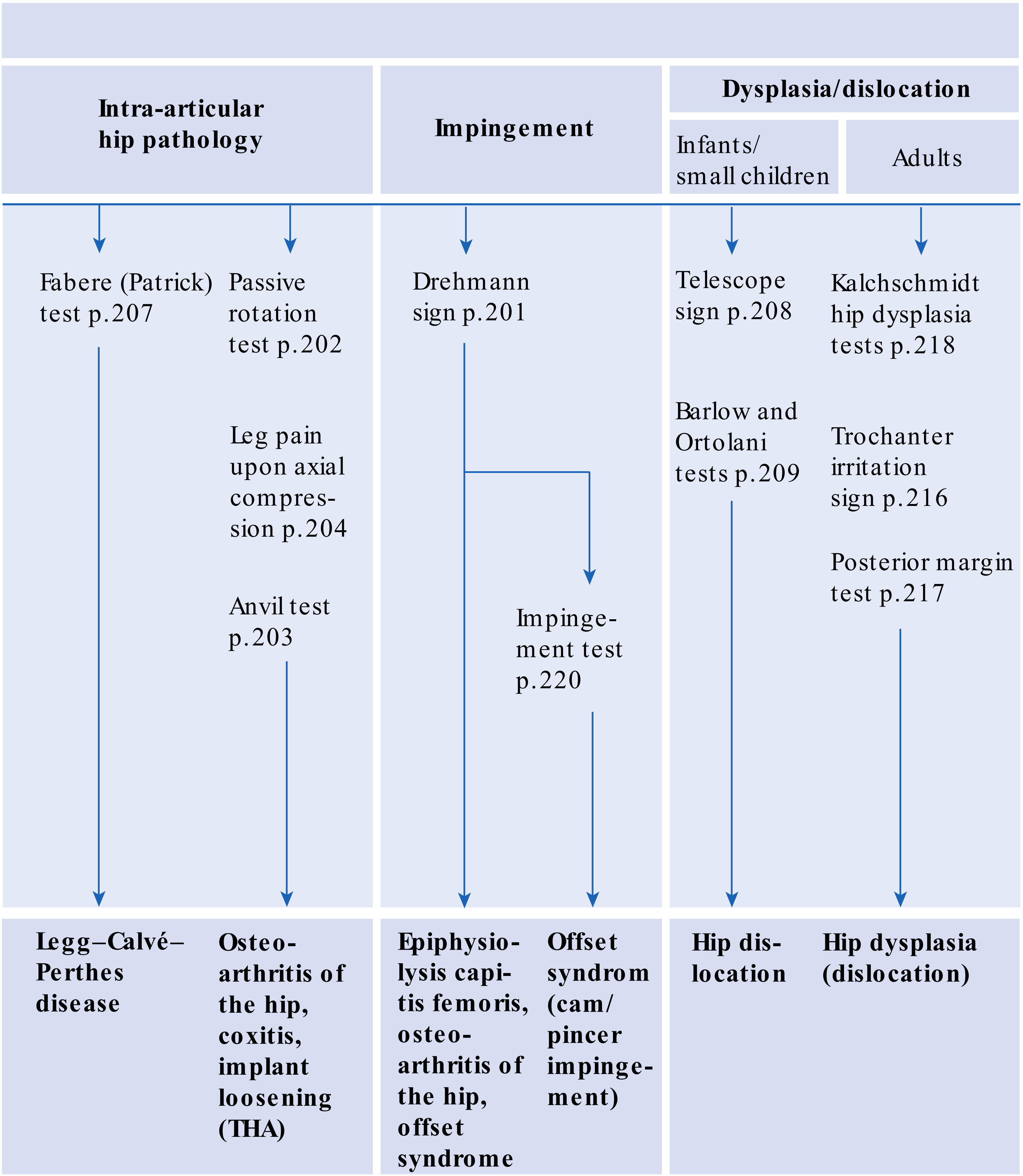


Fig. 5.2 Clinical tests to diagnose disorders of the hip.



THA, total hip arthroplasty

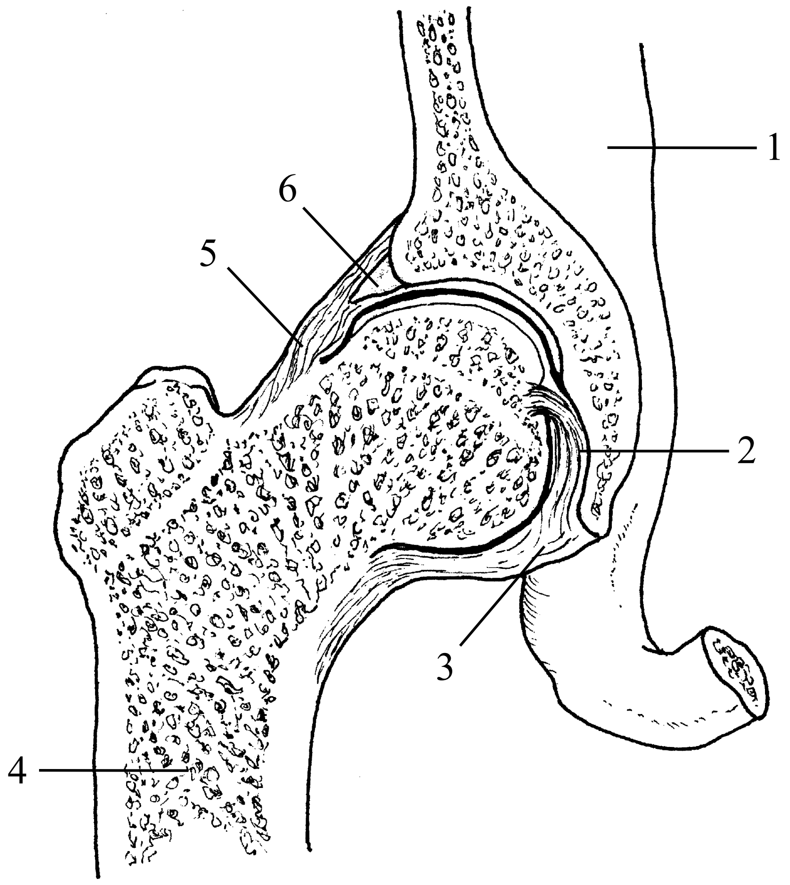


Fig. 5.3 Anatomy of the hip.
1 Ilium.
2 Ligament of the head of the femur.
3 Transverse acetabular ligament.
4 Femur.
5 Joint capsule with the iliofemoral and ischiofemoral ligaments.
6 Acetabular labrum.

Function Tests

Fingertip Test

Assesses contracture of the hamstrings.

□ **Procedure:** The patient is seated, holding one leg (flexed at the hip and knee) close to the trunk with the ipsilateral arm. The other leg remains extended. The patient is requested to touch the toes of the extended leg with the fingertips of the free arm. This test is then repeated on the contralateral side.

□ **Assessment:** In the presence of a hamstring contracture, the patient can only bring the fingertips into the general area of the foot and complains of "pulling" pain in the posterior thigh. The test is positive when there is a difference between the two sides and symptoms are present. Uniform, painless developmental shortening of the hamstrings is common. Restricted motion can result secondary to a spinal disorder or osteoarthritis of the hip.

□ **Note:** Symptoms of nerve root irritation can be excluded by other tests. Shortened hamstrings increase retropatellar pressure and can therefore cause retropatellar symptoms.

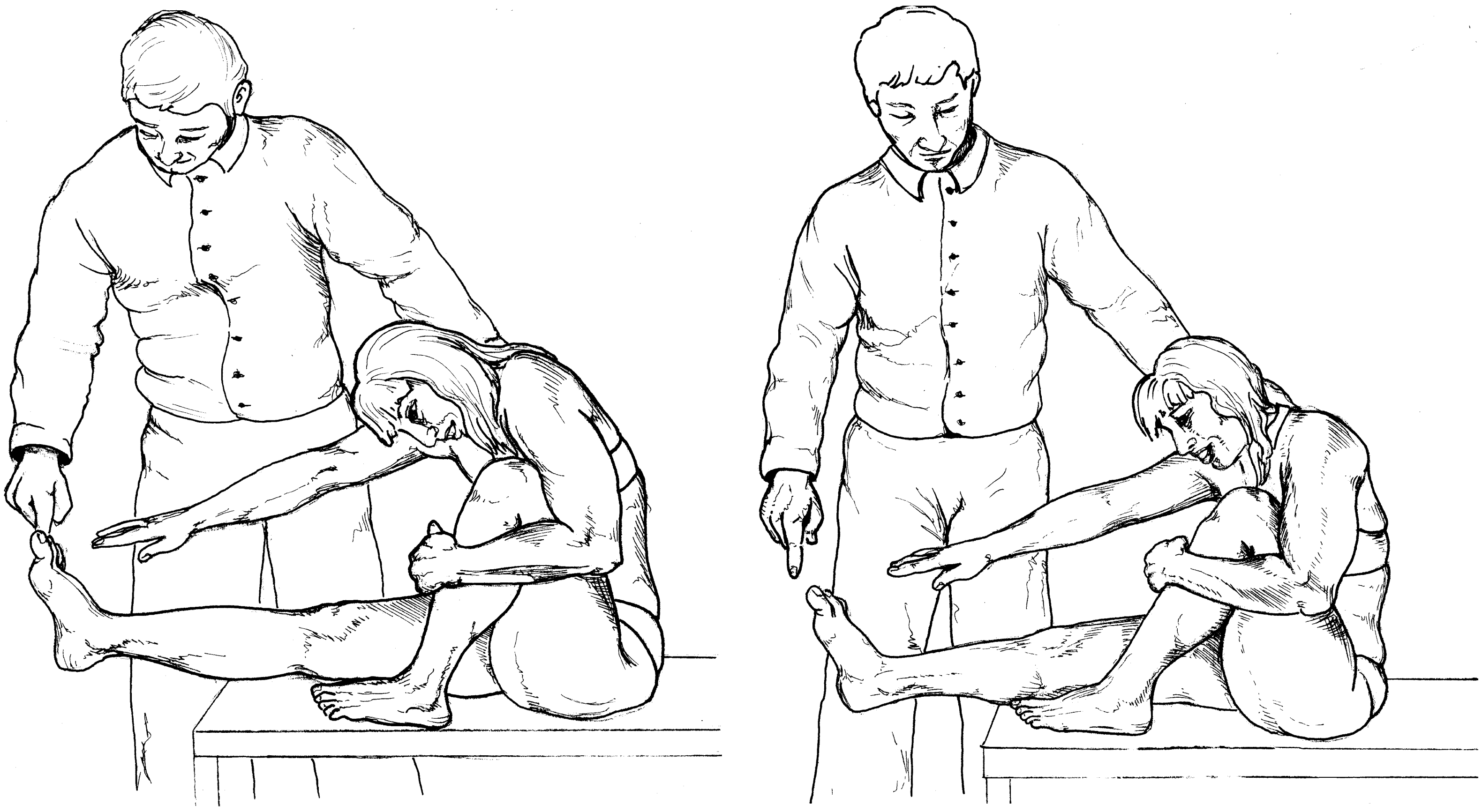


Fig. 5.4 Fingertip test. **(a)** Normal. **(b)** Abnormal with contracture of the hamstrings.

Test for Rectus Femoris Contracture

Assesses contracture of the quadriceps muscle.

□ **Procedure:** The patient is supine with the lower legs hanging over the edge of the examining table. The patient is requested to grasp one knee and pull it up against his or her chest. The examiner notes the angle that the hanging leg assumes. The test is repeated on the contralateral side.

□ **Assessment:** In a contracture of the rectus femoris, drawing one knee closer to the chest will produce flexion in the other leg lying on the table; when this starts to happen will depend on the contracture. The test will also be positive in the presence of a flexion contracture of the hip due to a hip disorder, psoas irritation (psoas abscess), lumbar spine disorder with increased lordosis, and change in pelvic inclination.

□ **Note:** A contracture of the quadriceps increases the retropatellar pressure and may thus be the cause of retropatellar symptoms.

5

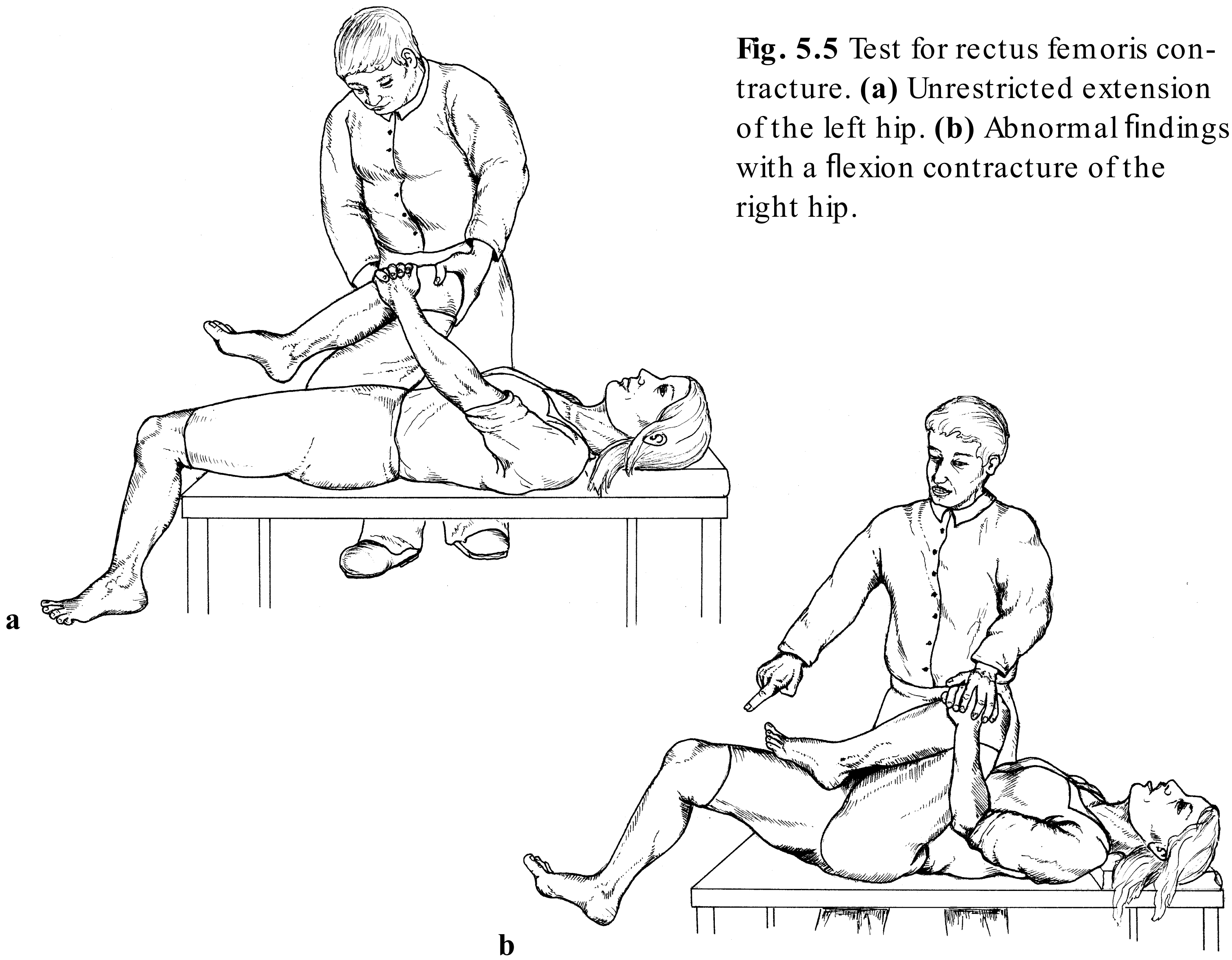


Fig. 5.5 Test for rectus femoris contracture. **(a)** Unrestricted extension of the left hip. **(b)** Abnormal findings with a flexion contracture of the right hip.

Rectus Femoris Stretch Test (Ely's Test)

□ **Procedure:** With the patient prone, the examiner tests the passive flexion of the knee, comparing the two sides.

□ **Assessment:** An increased gap between the heel and the gluteal muscles or the spontaneous flexion of the hip on the same side suggests a functional shortening of the rectus femoris muscle.

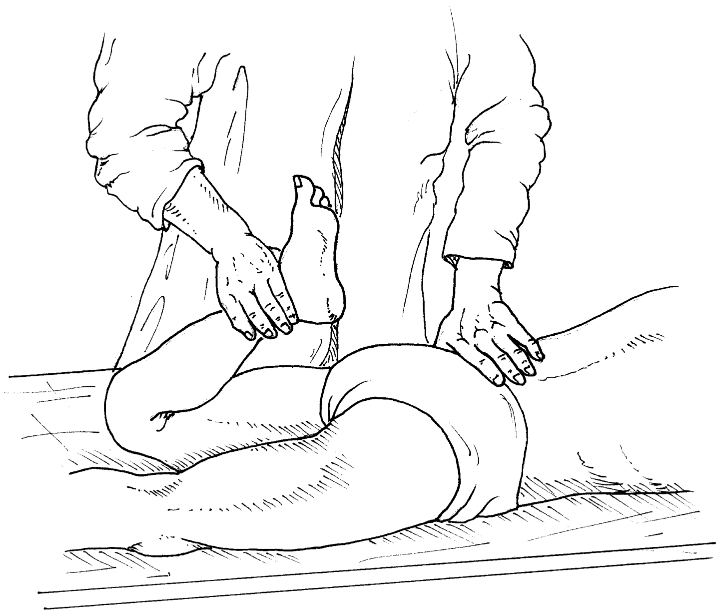


Fig. 5.6 Rectus femoris stretch test (Ely's test).

Hip Extension Test

Assesses flexion contracture of the hip.

□ **Procedure:** The patient is prone with both hips flexed over the edge of the examining table. The leg that is not being examined is held between the examiner's legs, or supported on a chair, or simply allowed to hang down.

With one hand, the examiner immobilizes the patient's pelvis. With the other hand, he or she slowly extends the leg to be examined. The prone position fully compensates for the lumbar lordosis.

□ **Assessment:** The point at which motion in the pelvis begins or the lumbar spine goes into lordosis indicates the end point of hip extension. The angle between the axis of the thigh and the horizontal (the examining table) approximately indicates the flexion contracture in the hip. This test allows good assessment of a flexion contracture, especially in bilateral contractures (such as in spasticity).

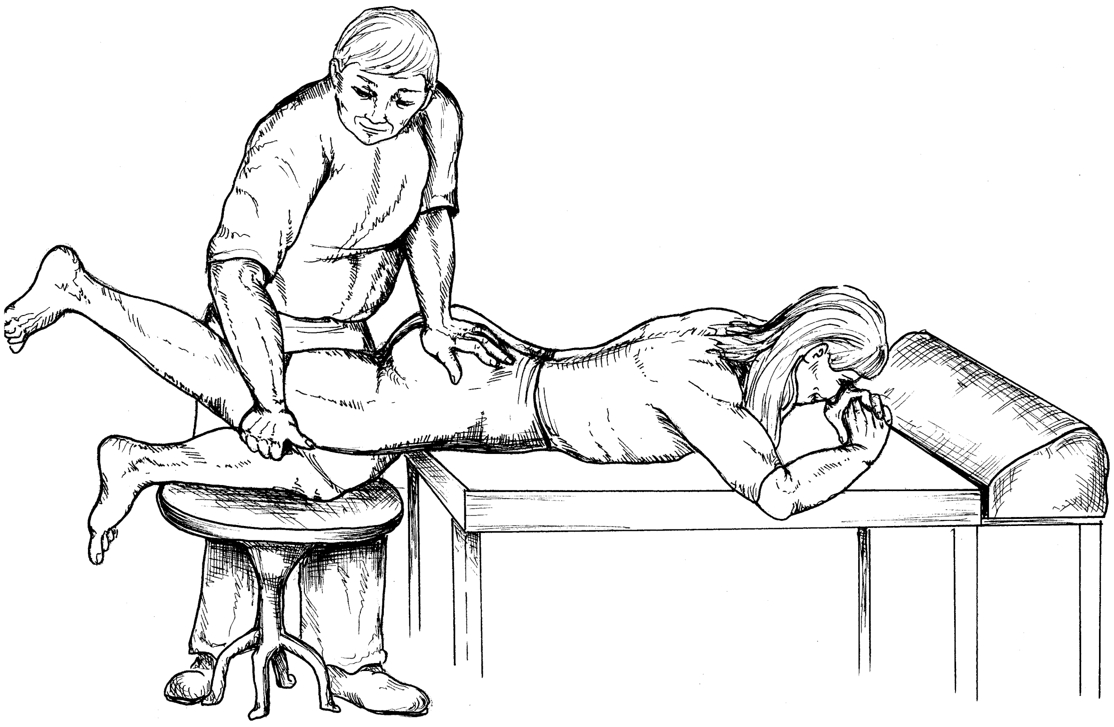


Fig. 5.7 Hip extension test.

Thomas Grip

Assesses flexion contracture at the hip.

□ **Procedure:** The patient is supine. The unaffected, contralateral leg is flexed at the hip until the lumbar lordosis disappears. This is verified by inserting one hand between the patient's lumbar spine and the examining table. With the patient in this position, the examiner immobilizes the pelvis in its normal position. The pelvis should exhibit about 12° of anterior inclination. This is what creates the lumbar lordosis. An increased flexion contracture in the hip can be compensated for by an increase in lumbar lordosis, in which case the patient only appears to assume a normal position.

□ **Assessment:** Extension is only possible up to the neutral position (0°); the thigh lies flat on the surface of the examining table. Further flexion can tilt the pelvis further upright. So long as the leg being examined remains in contact with the examining table, the angle of pelvic tilt achieved corresponds to the maximum hyperextension of the hip.

In a flexion contracture, the hip being examined does not continue to lie extended on the examining table. Instead it moves along with the increasing hip flexion or pelvic tilt, taking on a position of increasing flexion. The flexion contracture can be quantified by measuring the angle that the flexed, affected leg forms with the examining table.

Contractures of the hip occur in osteoarthritis, inflammation, and articular deformities of the hips. They can also cause spinal disorders.

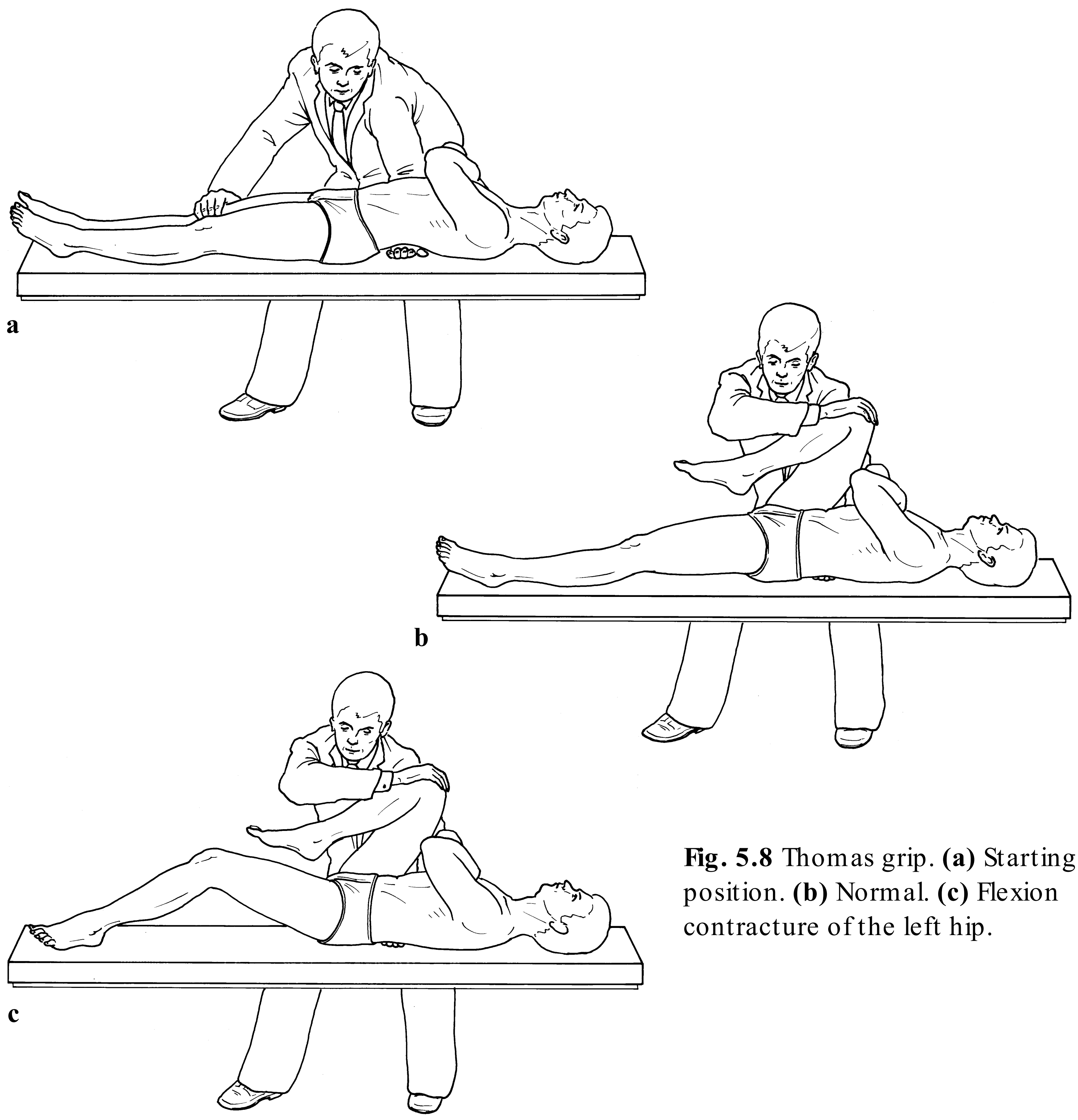


Fig. 5.8 Thomas grip. **(a)** Starting position. **(b)** Normal. **(c)** Flexion contracture of the left hip.

Noble Compression Test

Evaluation of a contracture of the tensor fasciae latae.

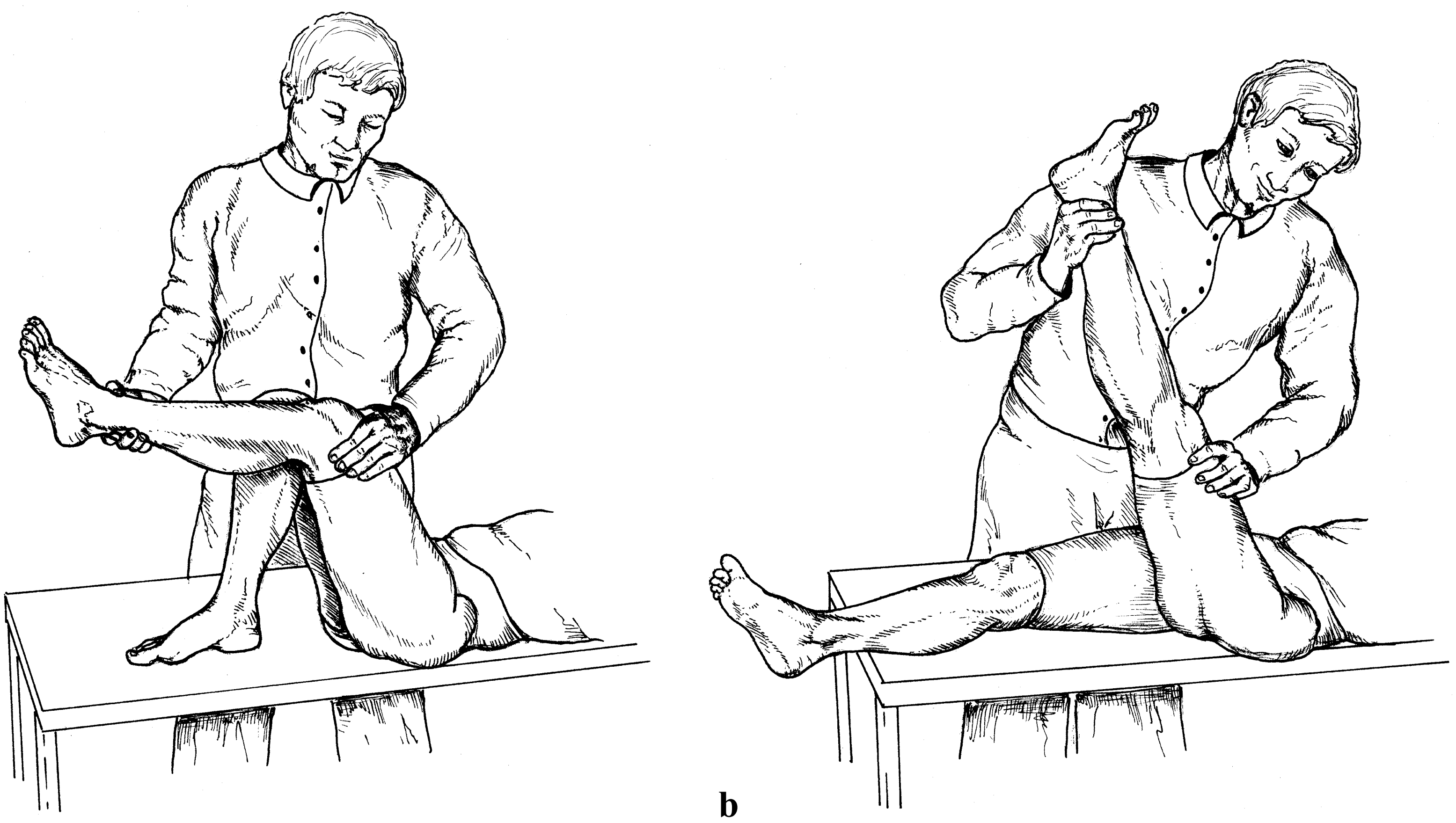
□ **Procedure:** The patient is supine. The examiner passively flexes the patient's knee 90° and the hip approximately 50°. With the fingers of the other hand, the examiner gently presses on the lateral femoral condyle. Maintaining the flexion in the hip and pressure on the lateral femoral condyle, the examiner then increasingly extends the knee passively. Once the knee is in about 40° of flexion, the patient is requested to fully extend the knee.

□ **Assessment:** The tensor fasciae latae arises from the anterolateral margin of the ilium (anterior superior iliac spine). It is an anterior branch of the gluteus medius. Its tendon inserts into the anterior margin of the iliotibial tract, which reinforces the fascia lata of the thigh.

The tensor fasciae latae inserts into the iliotibial tract, which in turn inserts into the tubercle of Gerdy on the proximal tibia. Extending the knee from 30° of flexion places maximum stress on the iliotibial tract.

Pain along the proximal and distal iliotibial tract suggests a contracture of the muscle or of the iliotibial tract itself. Pain in the posterior thigh that occurs with increasing extension is most likely indicative of a contracture of the hamstrings and should not be confused with a contracture of the tensor fasciae latae.

This test is often positive during walking when an iliotibial band friction syndrome is present.



a b

Fig. 5.9 Noble compression test. **(a)** Starting position. **(b)** Extension.

Ober Test

A contracted, nonelastic quadriceps muscle and shortened hamstring muscles cause an increase in the retropatellar pressure. Shortening of the iliotibial tract can lead to chronic pain on the lateral side and over its connection to the lateral patellar retinaculum as well as leading to functional disturbances in the femoropatellar joint.

□ **Procedure:** The patient lies in the lateral position. The leg to be examined is slightly adducted and the hip is slightly hyperextended. If the iliotibial tract is severely shortened, adduction is barely possibly.

The examiner places one hand on the distal iliotibial tract, which allows evaluation of muscle tone. With the forearm, the examiner registers evasive movements of the pelvis while grasping the lower leg near the ankle with the other hand. Tension is placed on the iliotibial tract by pressing the patient's lower leg toward the floor. The knee is then examined in various degrees of flexion.

□ **Assessment:** Increased tone as the knee approaches extension is readily detectable. However, flexing the knee reduces tension in the iliotibial tract as this moves the origin and insertion closer together. Careful palpation may detect fluctuations of the iliotibial tract near its insertion such as can occur in iliotibial tract friction syndrome or bursitis.

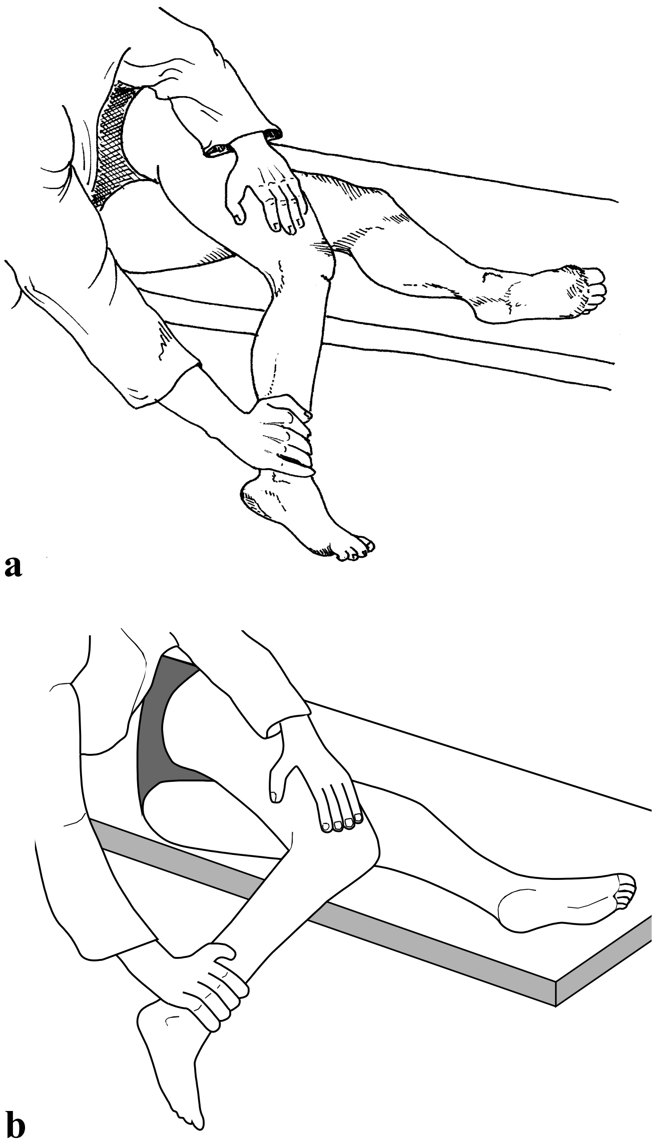
The patient often reports spontaneously that the type of pain is exactly the same as that felt during jogging (see Noble Compression Test, p. 199).

In severe shortening of the iliotibial tract or tensor fasciae latae, pain will also be felt at 30 to 60° of flexion.

□ **Note:** Stretching the iliotibial tract often helps in lateral displacement of the patella with excessive lateral pressure.

Even though the tension in the iliotibial tract is greater when the knee is extended, Ober described the test with flexed knee. In addition, when the knee is bent, the femoral nerve may be stretched during the course of the test. If neurologic symptoms occur, such as paresthesias and/or radiating pain, then there is suspicion of L3–L4 nerve root irritation.

Pain over the greater trochanter suggests trochanteric tendinopathy or bursitis.



a

b

Fig. 5.10 Ober test. **(a)** Starting position. **(b)** Pressing the lower leg toward the floor.

Drehmann Sign

□ **Procedure:** The patient is supine. The examiner grasps the patient's foot and knee and flexes the knee. A hip disorder is present when flexion produces increasing external rotation in the hip. The motion may be painless or it may cause pain.

□ **Assessment:** In adolescents, a positive Drehmann sign occurs primarily in the presence of a slipped capital femoral epiphysis. This causes the thigh to move into increasing compensatory external rotation as the hip is flexed. However, a hip infection, incipient osteoarthritis, or a tumor may also produce positive test results.

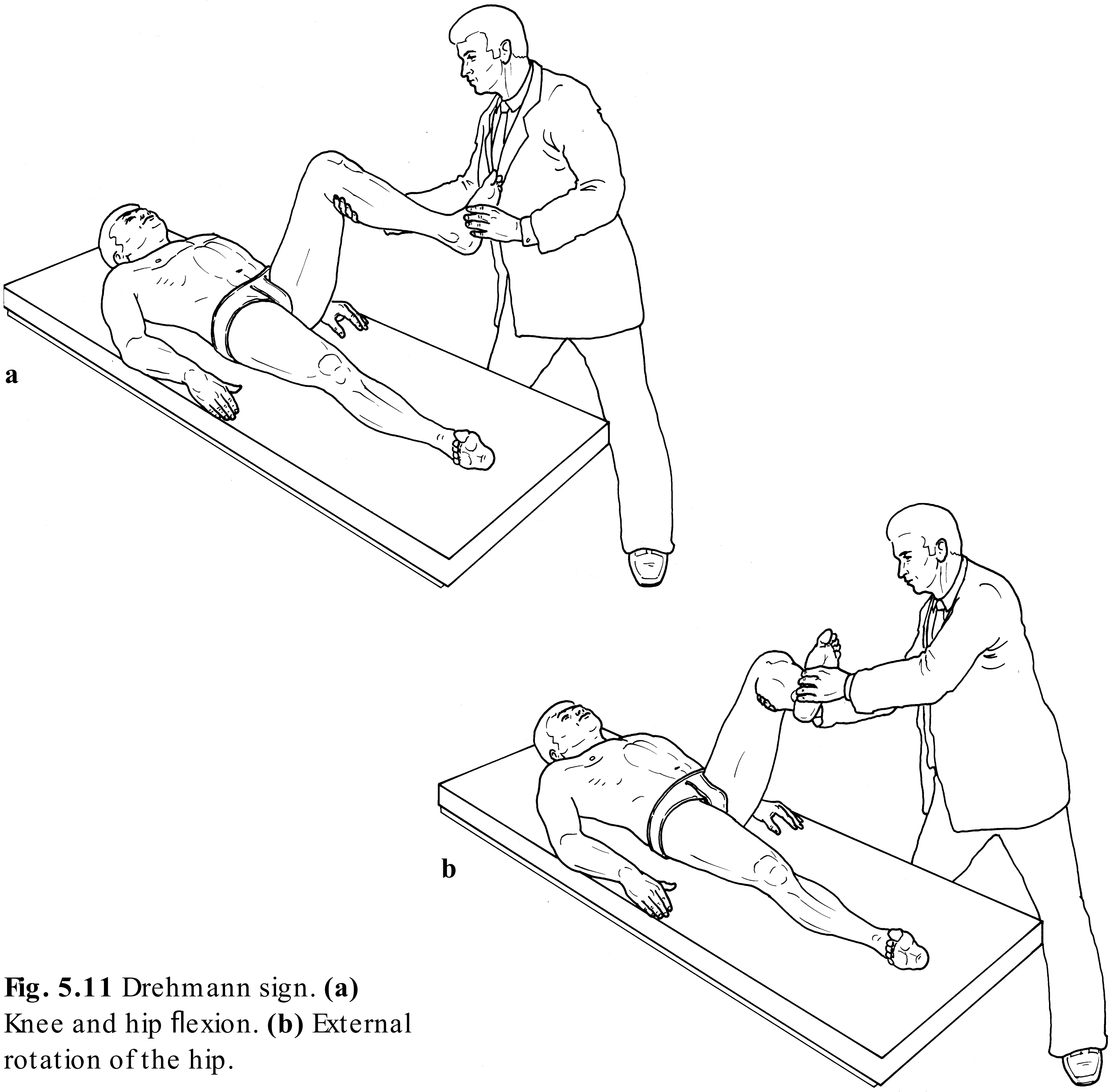


Fig. 5.11 Drehmann sign. **(a)** Knee and hip flexion. **(b)** External rotation of the hip.

Passive Rotation Test (Log Roll Test)

□ **Procedure:** With the patient supine, the examiner places one hand on the thigh and the other on the ankle and gently rolls the leg back and forth, thus alternately internally and externally rotating the hip. During this process, the joint surfaces of the femoral head move within the acetabulum without activating neural or myotendinous structures.

□ **Assessment:** Pain or movement restrictions when compared with the opposite side suggest intra-articular pathology within the hip joint.

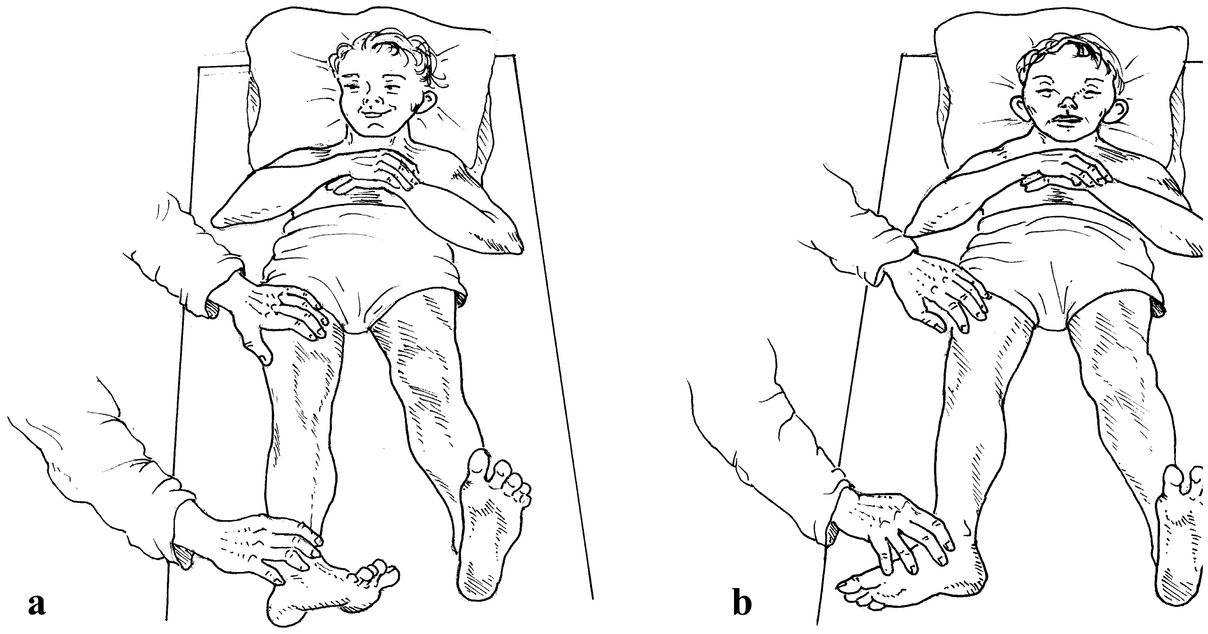


Fig. 5.12 Passive rotation test (log roll test). **(a)** Internal rotation. **(b)** External rotation.

Anvil Test

□ **Procedure:** The patient is supine with legs extended. The examiner raises the extended leg slightly with one hand and hits the heel axially with the fist of the other hand.

□ **Assessment:** The force of the blow is transmitted to the hip. Pain in the groin or in the thigh adjacent to the hip suggests hip disease (such as osteoarthritis or inflammation of the hip) or femoral neck fracture. In total hip arthroplasty patients, it suggests implant loosening (groin pain suggests loosening of the acetabular component, whereas pain in the lateral thigh suggests loosening of the femoral stem).

Symptoms in the lumbar spine occur in intervertebral disk disease or in rheumatoid spine disorders.

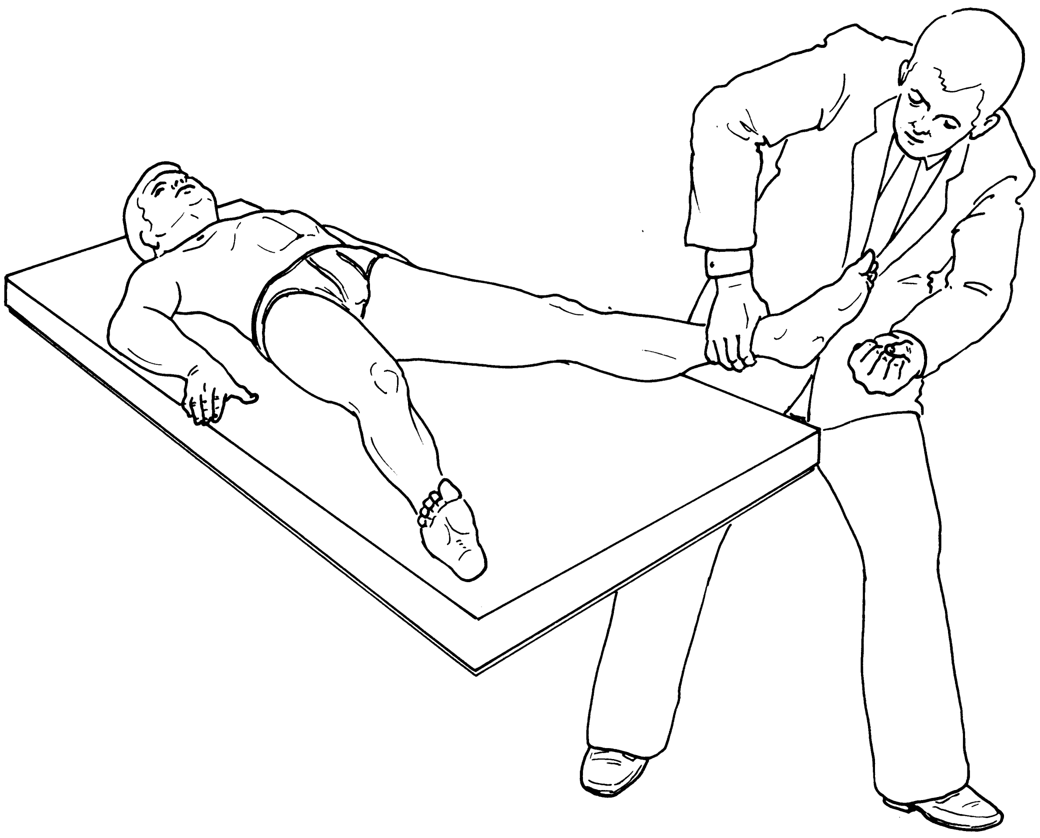


Fig. 5.13 Anvil test.

Leg Pain upon Axial Compression

□ **Procedure:** The patient is supine with one leg extended and the other flexed at the knee and externally rotated at the hip. The lateral malleolus of the flexed leg lies just superior to the patella of the contralateral leg.

The examiner grasps the distal thigh of the flexed leg with both hands and compresses it axially.

□ **Assessment:** This motion compresses the hip joint and the affected side of the pelvis.

Pain in the groin suggests hip disease such as osteoarthritis of the hip. In total hip arthroplasty patients, it suggests implant loosening.

Symptoms in the lumbar spine occur in intervertebral disk disease or in rheumatoid spine disorders.

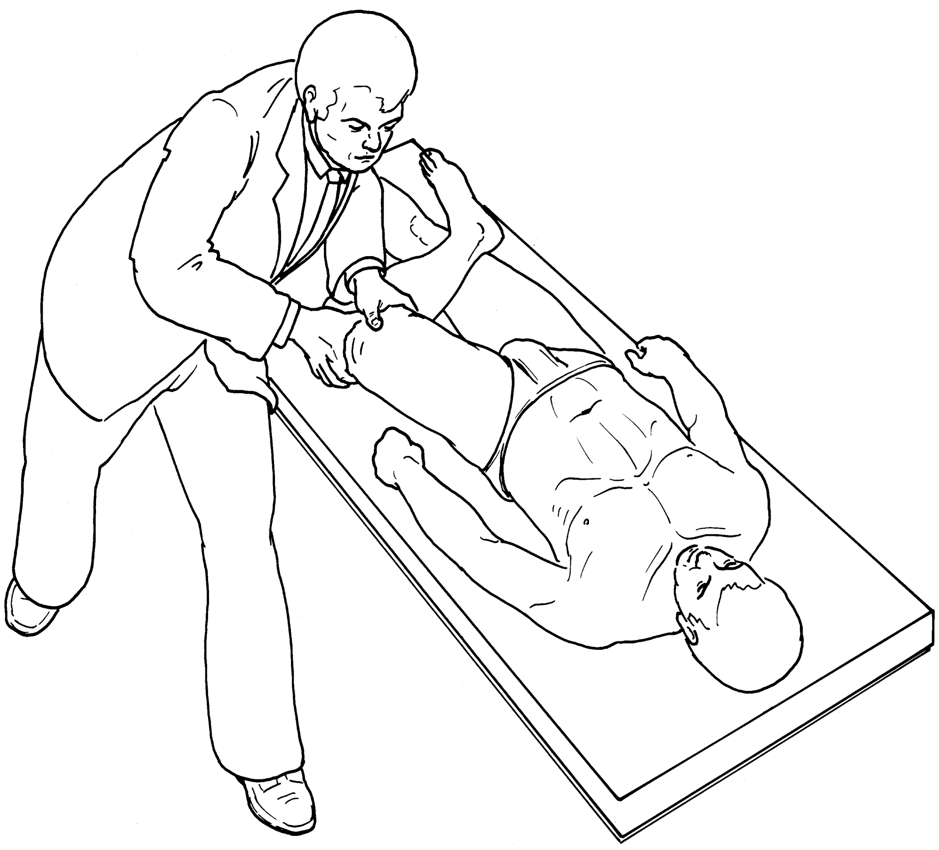


Fig. 5.14 Leg pain upon axial compression.

Piriformis Test

Assessment of a contracture of the piriformis muscle.

□ **Procedure:** The patient lies in the lateral position with the test leg uppermost. The patient flexes the hip of that leg to 60° with the knee flexed. The examiner stabilizes the hip with one hand and applies downward pressure to the knee.

□ **Assessment:** If the piriformis is tight, pain is elicited in the muscle. If the piriformis is pinching the sciatic nerve, pain results in the buttock and the patient may experience sciatica.

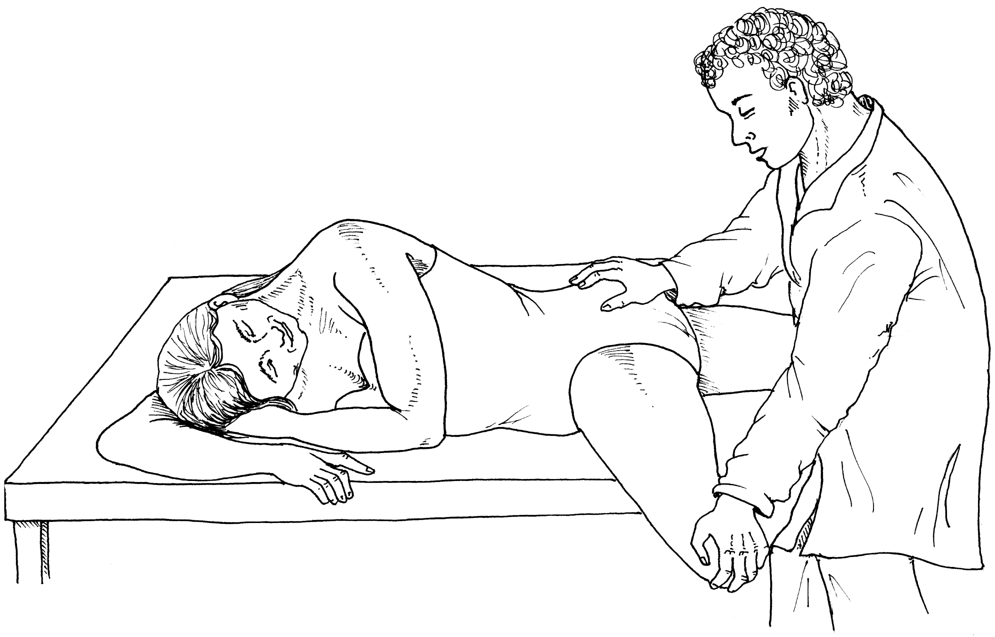


Fig. 5.15 Piriformis test.

In about 15% of the population the sciatic nerve or other branches of the sacral plexuses pass through the piriformis muscle rather than below it. These people are more likely to suffer from this relatively rare piriformis syndrome.

Painful shortening of the piriformis muscle can also cause restriction of internal rotation of the hip.

Trendelenburg Sign/Duchenne Sign

Tests pelvic and trochanteric muscle function.

□ **Procedure:** The examiner stands behind the standing patient. The patient is requested to raise one leg by flexing the knee and hip.

□ **Assessment:** In the single leg stance, the pelvic and trochanteric musculature (gluteus medius and gluteus minimus) on the weight-bearing side contract and elevate the pelvis on the unsupported side, holding it nearly horizontal. This process allows uniform gait. Where the gluteal muscles are compromised (weakened as a result of a hip dislocation, due to paralysis, or following multiple hip operations) with functional deficits, they are no longer able to support the pelvis on the weight-bearing side. The pelvis then drops down on the normal, non–weight-bearing side (positive Trendelenburg sign). The patient will exhibit a typical ducklike waddling gait, especially in a bilateral condition (as in bilateral hip dislocation).

The drop in the pelvis toward the unaffected side also shifts the body's center of gravity in that direction. Patients usually compensate by shifting the body toward the weight-bearing leg (Duchenne sign).

Reasons for insufficiency of the pelvic and trochanteric musculature:

- Genuine weakness (paresis or paralysis).
- Reduced distance between origin and insertion (hip dislocation, high-riding greater trochanter, varus osteotomy, Legg–Calvé–Perthes disease).
- Altered mechanics (shortened femoral neck, increased anteversion).
- Pain.

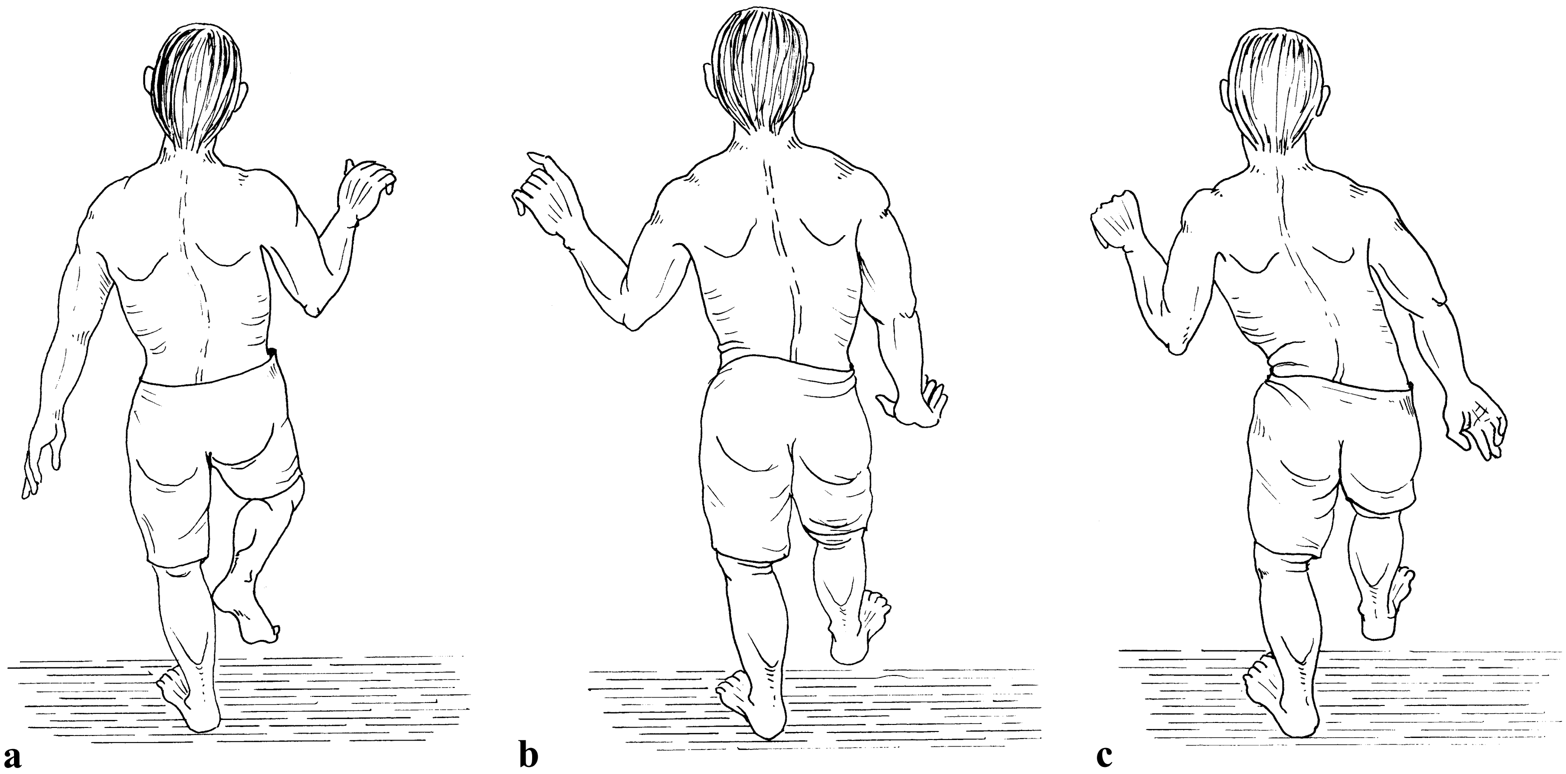


Fig. 5.16 Trendelenburg sign/Duchenne sign. **(a)** Normal hip: patient can lift the pelvis by contracting the pelvic and trochanteric musculature on the weight-bearing side. **(b)** Insufficiency of the pelvic and trochanteric musculature causes the pelvis to dip toward the normal non–weight-bearing side (positive Trendelenburg sign). **(c)** Insufficiency of the pelvic and trochanteric musculature can be partially compensated for by shifting the body's center of gravity toward the weight-bearing leg (Duchenne sign).

Table 5.1 Grading of the Trendelenburg sign (from Hoppenfeld 1982)

Negative	Patient can lift the pelvis on the non–weight-bearing side
Weakly positive	Patient can maintain the position of the pelvis on the non–weight-bearing side but not lift it
Positive	Pelvis on the non–weight-bearing side drops visibly

Fabere Test (Patrick Test) for Legg–Calvé–Perthes Disease

□ **Procedure:** The child is supine with one leg extended and the other flexed at the knee. The lateral malleolus of the flexed leg lies across the other leg superior to the patella. The test may also be performed so that the sole of the foot of the flexed leg is in contact with the medial aspect of the knee of the contralateral leg. The flexed leg is then pressed or allowed to fall further into abduction.
□ **Assessment:** Normally the knee of the abducted leg will almost touch the examining table. The examiner makes comparative measurements of the distance between the knee and the table on both sides. On the side of the positive Patrick sign, motion is impaired, the adductors are tensed, and the patient feels pain when the leg is further abducted past the starting position in limited abduction. Pain in the groin can be a sign of incipient Legg–Calvé–Perthes disease.

Legg–Calvé–Perthes disease is regarded as belonging to the group of aseptic avascular necroses. The disease manifests itself in the epiphysis, metaphysis, and apophysis of the long bones and in the tarsal and carpal bones that ossify within the cartilage. Legg–Calvé–Perthes disease is the most common form of

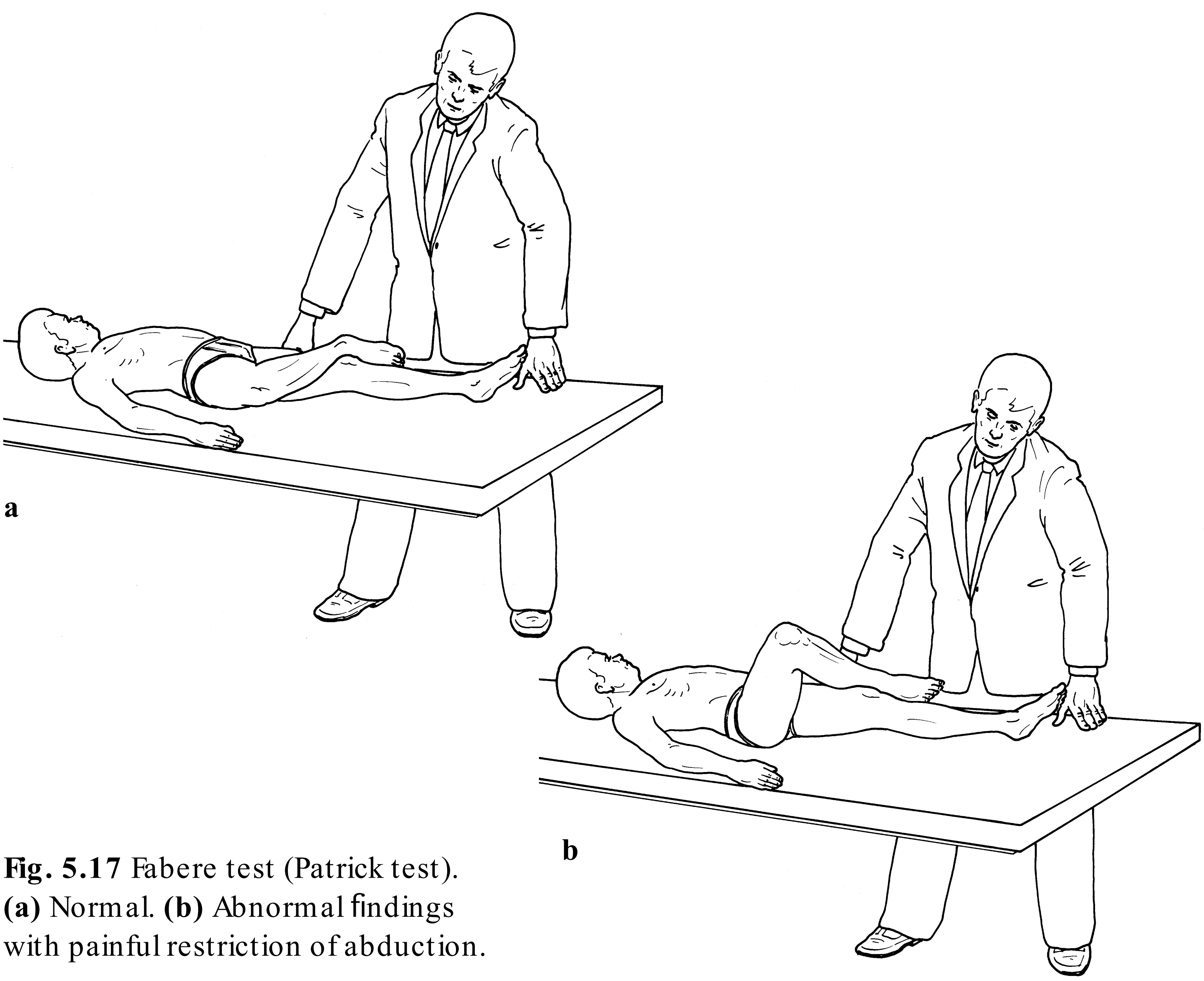


Fig. 5.17 Fabere test (Patrick test). **(a)** Normal. **(b)** Abnormal findings with painful restriction of abduction.

aseptic bone necrosis. It occurs primarily between the ages of 3 and 12 years, with peak occurrence between the ages of 4 and 8 years. In the early stages of the disease, children tire quickly and begin to limp slightly. They complain of slight pain in the hip; occasionally they only complain of knee pain.

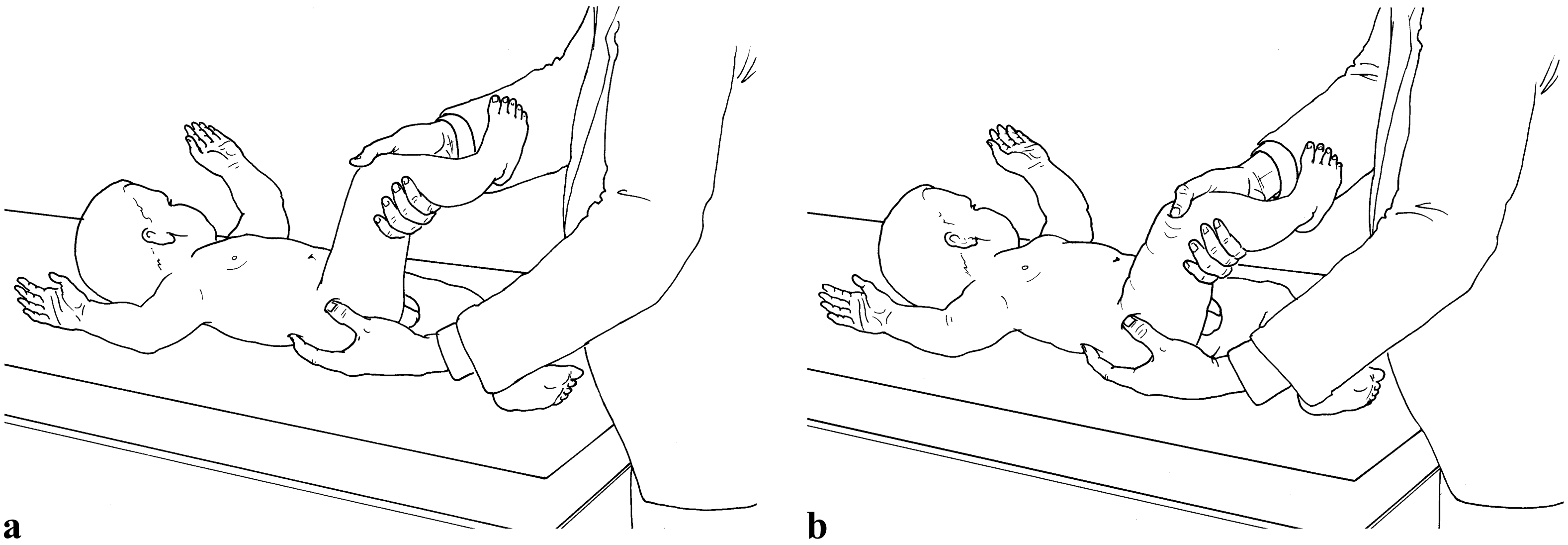
□ **Note:** A positive test result in adults with simultaneous occurrence of movement restriction and painful pressure points suggests hip disease or contracture of the iliopsoas muscle. Within the differential diagnosis, dysfunction of the ipsilateral sacroiliac joint must be ruled out.

Telescope Sign

Indicates congenital hip dislocation.

□ **Procedure:** The examiner grasps the affected leg with one hand and passively flexes the hip and knee. The other hand rests posterolateral to the hip. The examiner palpates the greater trochanter with the thumb of this hand and the motion of the femoral head with the index finger. The hand guiding the leg alternately applies axial compression and traction to the femur.

□ **Assessment:** In a hip dislocation, the leg will appear to shorten or lengthen. The palpating hand follows the motion of the greater trochanter and femoral head into the dislocated position and back to reduction.



a b

Fig. 5.18 Telescope sign. **(a)** Apparent leg "shortening" on axial compression. **(b)** Apparent leg "lengthening" on axial traction.

Barlow and Ortolani Tests

This test assesses hip instability in infants.

□ **Procedure:** With the infant supine, the examiner passively flexes one leg, immobilizing the pelvis. The other hand grasps the knee and thigh of the leg to be examined in such a manner that the index finger and thumb rest inferior to the inguinal fold.

With the thigh initially in extreme adduction, the examiner carefully exerts axial pressure while simultaneously pressing the thigh into abduction from the medial side. The fingers provide controlled resilient resistance to this motion. Instability in the hip will be palpable as the direction of force changes between the fingers and thumb. This is the Barlow dislocation test. In the second phase of the examination, the examiner slowly abducts the thigh while maintaining axial compression. If the femoral head was pushed out of the center of the acetabulum during the first phase (Barlow test), the examiner can now reduce it into the acetabulum with a palpable snap by pressing on the greater trochanter with the fingers. This is known as the Ortolani "click."

This test assesses should be repeated separately for each leg.

□ **Assessment:** The examination detects instability of the hip and also allows one to define the degree of instability present. Tönnis differentiates four grades of instability:

- **I**: Slightly unstable hip without a snap.
- **II**: Dislocatable hip. The hip can be fully or largely reduced by abduction alone (with a snap).
- **III**: Hip that can be dislocated and reduced.
- **IV**: Dislocated hip that cannot be reduced. The acetabulum is empty, and the femoral head can be palpated posteriorly; abduction is severely limited and reduction is not possible.

□ **Note:** A "dry click" without dislocation can often be provoked during the first days of life, but disappears thereafter.

The Barlow and Ortolani test is particularly useful in newborns 2 to 3 weeks old. The **Ludloff–Hohmann test** is an alternative in slightly older children. With the hip flexed and abducted, spontaneous knee flexion will normally occur as a result of the physiologic tension in the hamstrings. A knee that can be fully extended with the hip flexed and abducted suggests an unstable hip.

5

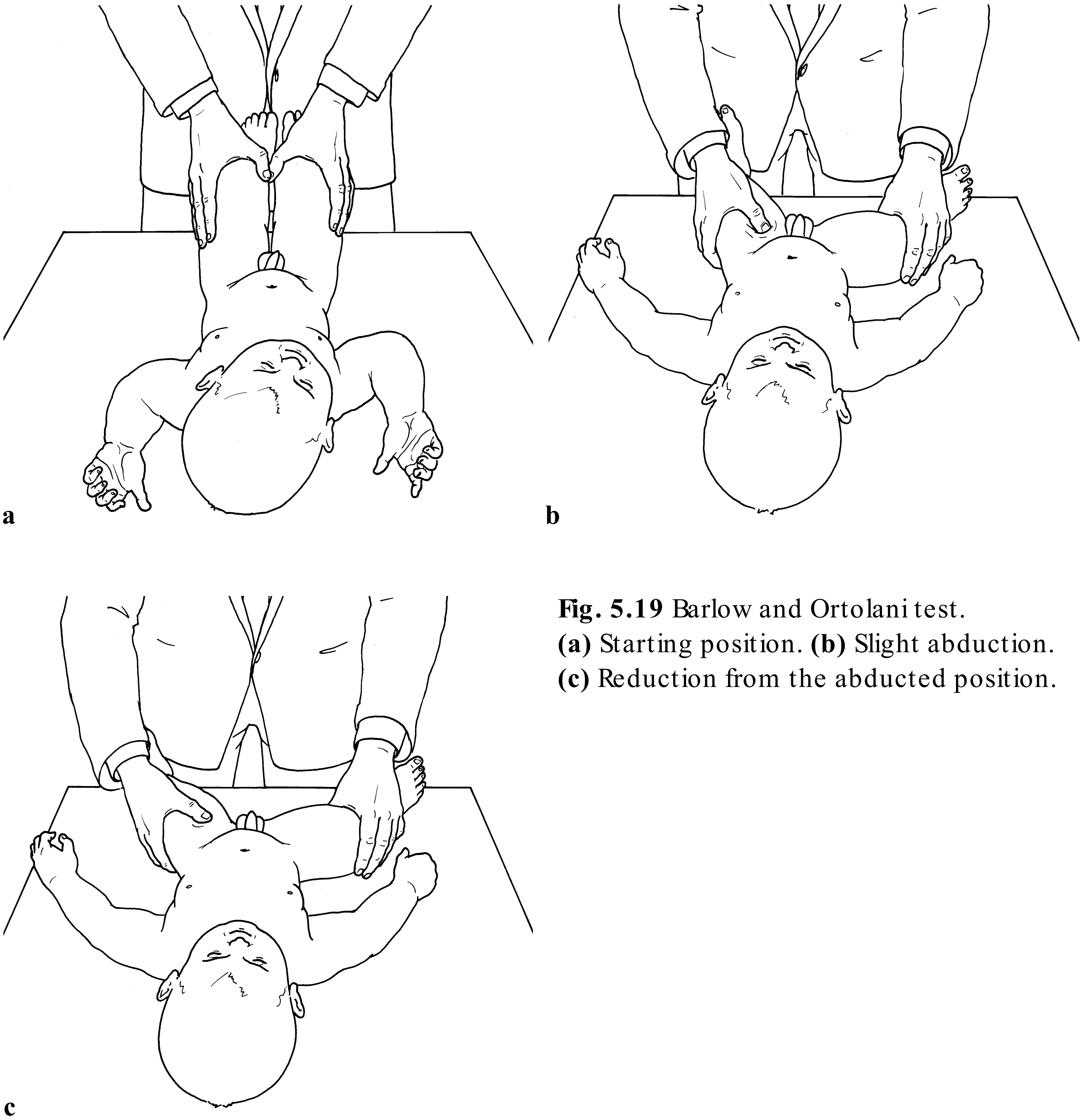


Fig. 5.19 Barlow and Ortolani test.
(a) Starting position. **(b)** Slight abduction.
(c) Reduction from the abducted position.

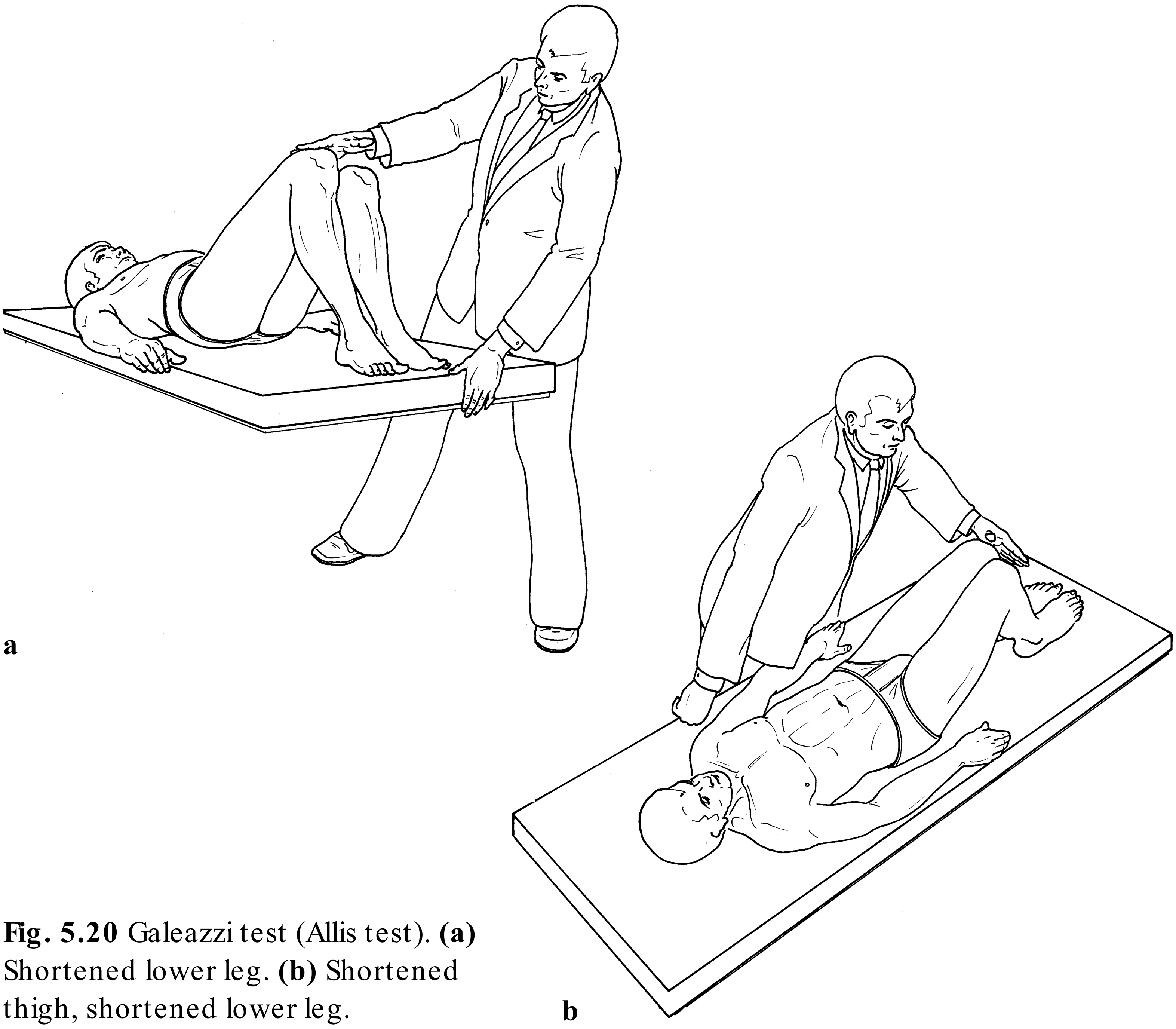
Galeazzi Test (Allis Test)

Assesses leg length difference.

□ **Procedure:** The patient is supine with the knees flexed 90° and the soles of the feet flat on the examining table. The examiner assesses the position of both knees from the end of the table and from the side.

□ **Assessment:** Normally both knees are at the same level. Where one knee is higher than the other, either the tibia of that side is longer or the contralateral tibia is shorter. Where one knee projects farther forward than the other, either that femur is longer or the contralateral femur is shorter. The test for assessment of femur length is indicated as an additional test for evaluating hip dislocation. However, in such a case there is only an apparent difference in length; the femurs are the same length but one thigh appears shorter due to the hip dislocation.

Note that the Galeazzi test will yield a false-negative result in cases of bilateral hip dislocation.



a

b

Fig. 5.20 Galeazzi test (Allis test). **(a)** Shortened lower leg. **(b)** Shortened thigh, shortened lower leg.

Anteversion Test (Craig Test)

□ **Procedure:** The test is carried out in the prone position. With the patient's knee flexed, the examiner uses one hand to rotate the lower leg and the other to palpate the greater trochanter of the femur to ascertain when it extends the farthest laterally.

□ **Assessment:** At the most lateral position of the greater trochanter, the degree of anteversion can be estimated directly on the basis of the angle by which the lower leg deviates from the vertical. In this position, the femoral neck lies in the horizontal plane while the condyles of the knee and the lower leg indicate the anteversion angle.

The precision of this measurement performed by an experienced examiner is comparable to that of radiographic measurement.

At birth, the anteversion angle is approximately 40°; in the adult it adjusts to 8–15°. Increased anteversion leads to knock knees and an intoeing gait.

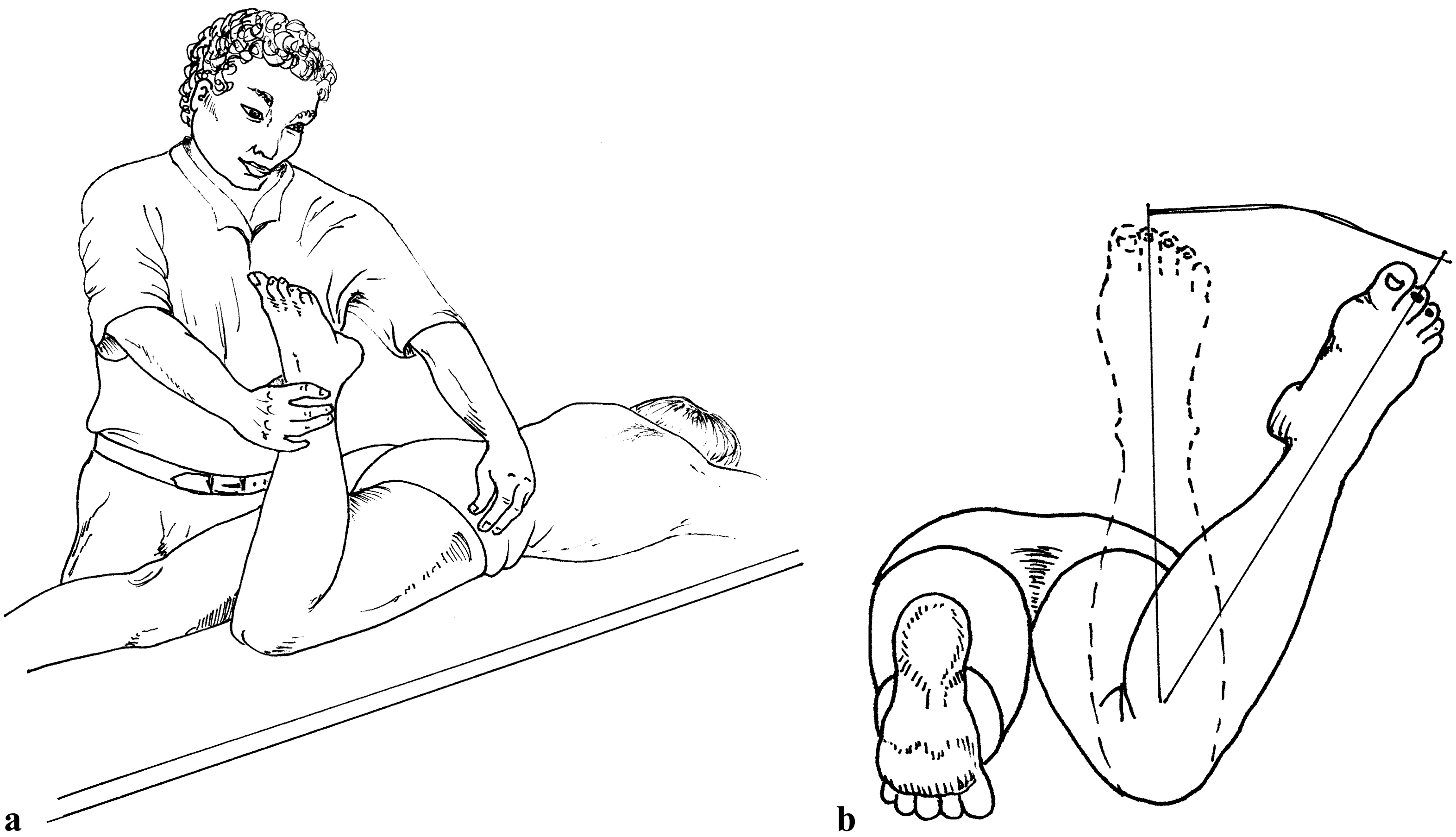


Fig. 5.21 Anteversion test. **(a)** Flexion of the knee to 90°. **(b)** Angle of deviation of the lower leg from the vertical.

Leg Length Difference Test

Assesses actual and functional leg length differences.

Procedure: Measurement of an actual difference in leg length is performed with the patient standing by placing shims of varying thickness (0.5, 1, 2 cm) underneath the shorter leg until the pelvic obliquity is fully compensated.

Assessment: Compensation of the pelvic obliquity is usually readily apparent, especially when the patient bends forward from a standing position. With the pelvis horizontal, the leg length difference corresponds to the total height of the shims placed beneath the foot. Evaluating leg length difference by palpating the iliac crests from behind the patient is often imprecise. Often the iliac wings (iliac crests) will not be at the same level although radiographic findings confirm identical leg length and a normal vertical spine. Asymmetric iliac wings are frequently encountered in conditions such as hip dysplasia. The iliac wing on the dysplastic side is usually smaller. Often only a pelvic radiograph obtained with the patient standing and showing the sacrum and lower lumbar spine will allow one to draw reliable conclusions about the type and severity of the leg length difference.

Where placement of shims cannot compensate the pelvic obliquity, the patient has a fixed deformity of one or more joints or a fixed scoliosis leading to a functional leg length difference. This functional difference occurs as a result of a flexion or adduction contracture in the hip. The pelvis dips toward the normal side; the normal leg appears lengthened and the affected leg shortened.

An abduction contracture in the hip causes a functional leg length difference. The pelvis dips toward the affected side; the normal leg appears shortened and the affected leg lengthened. An actual leg length difference is best evaluated and measured with the patient standing, a functional difference with the patient supine.

Note: One can also use **direct leg-length measurement** to determine leg length difference by using a tape measure to measure the distance between the anterior superior iliac spine and the medial malleolus.

5

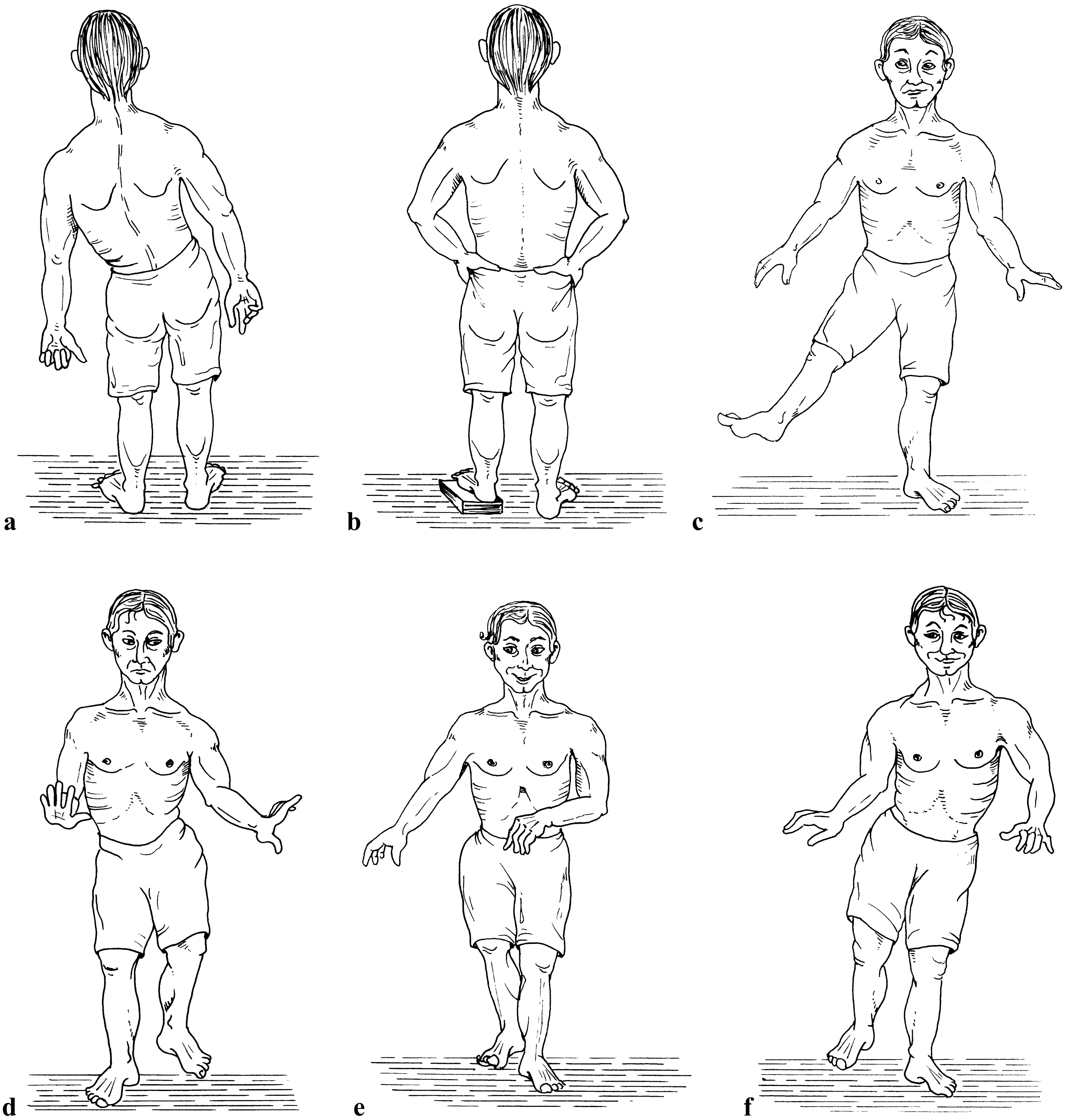

a b c d e f

Fig. 5.22 Leg length difference test. **(a, b)** Actual shortening of the leg: The legs appear equally long with the patient standing. Shortening of the left leg is compensated for by pelvic obliquity and scoliotic posture **(a)**. The pelvic obliquity and scoliotic posture can be eliminated by placing shims under the leg **(b)**. **(c, d)** Functional lengthening of the leg: Abduction contracture on the right side **(c)**. The pelvis dips toward the affected side. The normal leg appears shortened and the affected leg lengthened **(d)**. **(e, f)** Functional shortening of the leg: Adduction contracture on the right side **(e)**. The affected leg appears shortened and the normal leg lengthened **(f)**.

Hip and Lumbar Rigidity in Extension

Indicates spinal cord disease and intervertebral disk pathology in children.

□ **Procedure:** The child is supine. The examiner lifts the child's legs.

□ **Assessment:** Reflexive rigidity that maintains hip extension when the child's legs are lifted is a sign of a spinal cord lesion such as a tumor, compression of the spinal cord as in spondylolisthesis, or nerve root compression as in intervertebral disk extrusion.

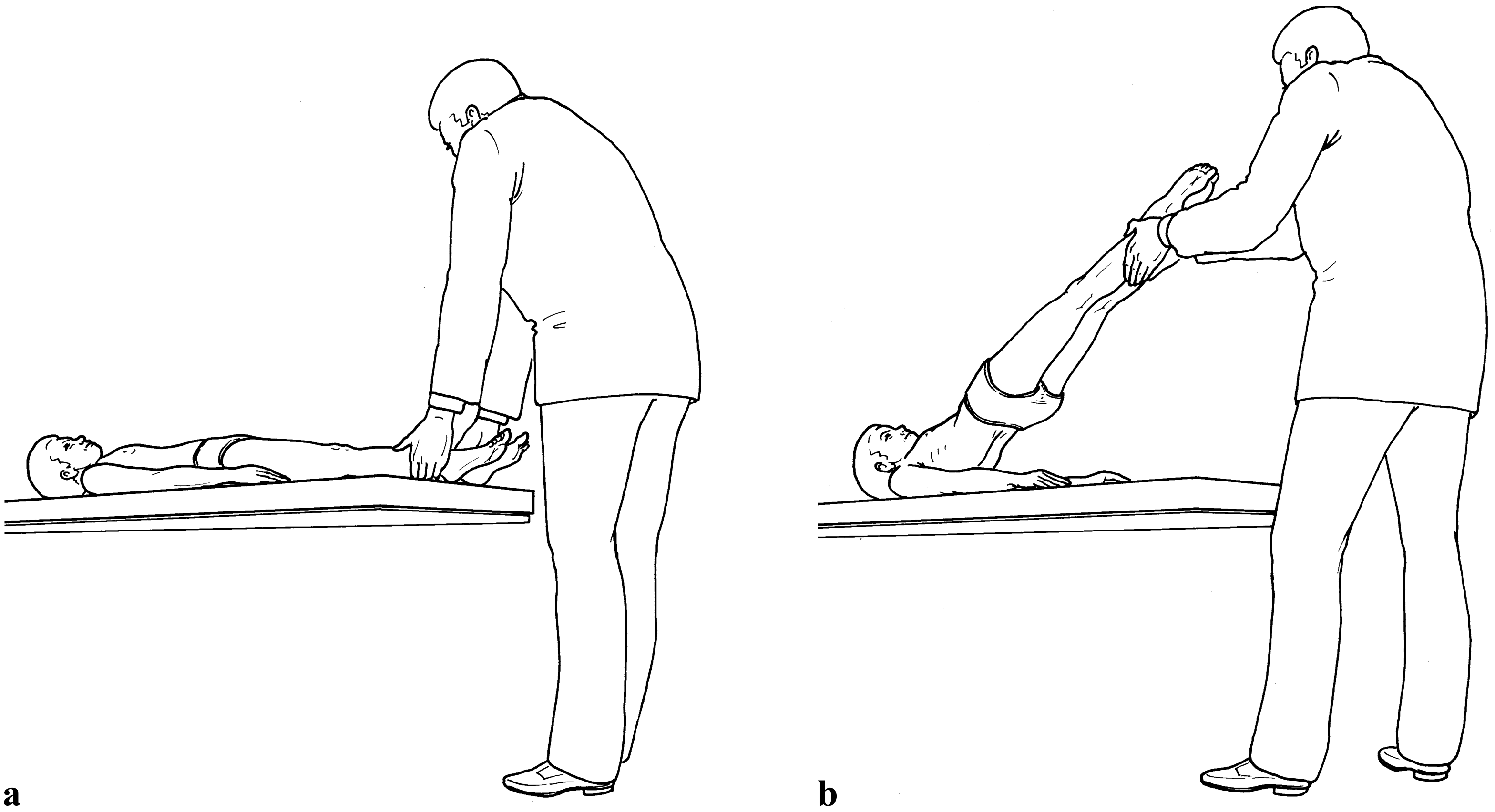


Fig. 5.23 Hip and lumbar rigidity in extension. **(a)** Starting position. **(b)** Abnormal findings.

Trochanter Irritation Sign (Bicycle Test)

A test of abductor function, indicative of hip dysplasia.

□ **Procedure:** The patient lies on the normal side. In this lateral position, the patient performs bicycle pedaling motions in slight abduction.

□ **Assessment:** Pain felt over the greater trochanter and gluteal musculature is indicative of exercise pain in the abductors, which in turn can suggest hip dysplasia. This test is only performed in patients with a history of exercise pain in the abductors. In addition to tenderness over the greater trochanter, weakness of the abductors can also lead to a positive Trendelenburg sign and a characteristic hip-favoring limp to relieve pressure on the hip. An occasional asymptomatic or painful “snapping” of the hip can be evoked by a labral lesion or by the psoas tendon as it slides over the protruding femoral head.

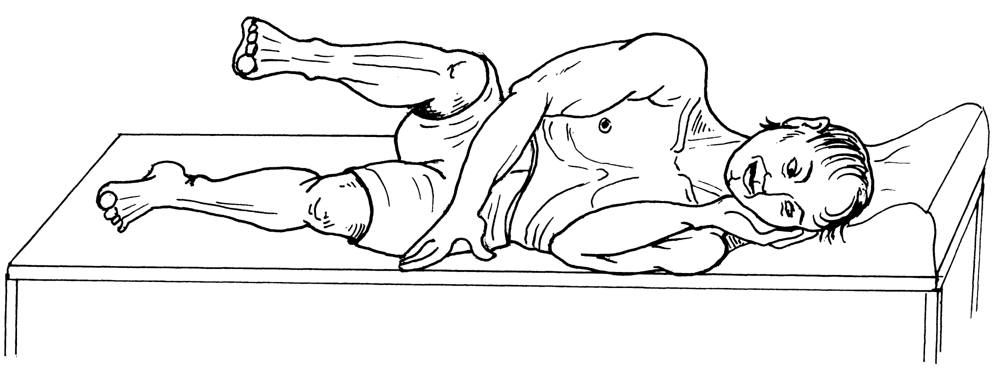


Fig. 5.24 Trochanter irritation sign.

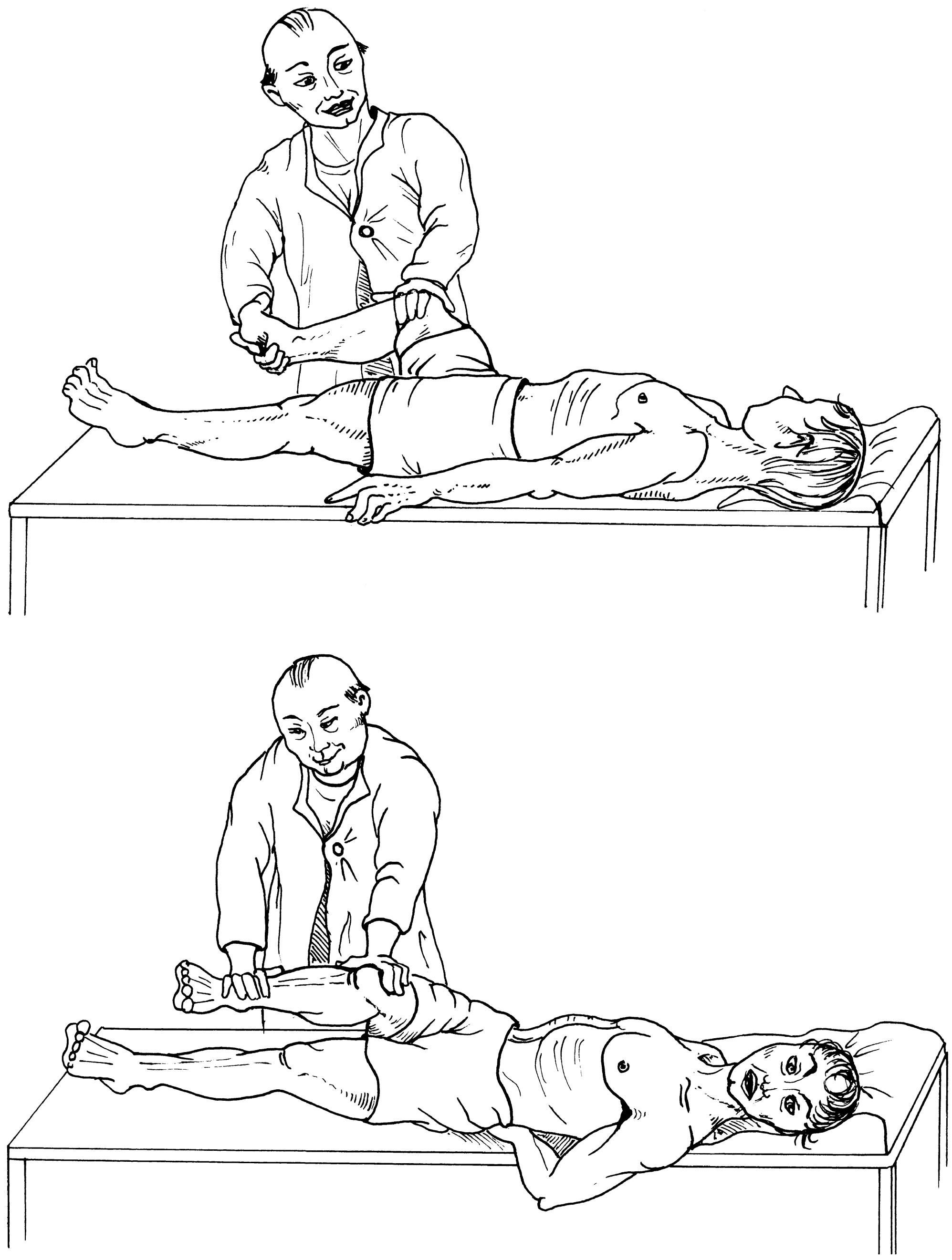
Posterior Margin Test

Indicates a lesion of the posterior acetabular labrum.

□ **Procedure:** With the patient supine, the hip is forcibly flexed, abducted, and externally rotated. Then it is extended in adduction and internal rotation.

□ **Assessment:** In this maneuver, motion of the femoral head places compressive and shear stresses on the posterior capsule–labrum complex. Pain felt in the posterolateral region of the hip is a sign of a posterior capsular and/or labral lesion.

A diagnostic infiltration test (intra-articular injection of 10 mL of local anesthetic agents) can differentiate between intra-articular and extra-articular pain patterns. Where a labral lesion is present, painfully restricted motion should be limited to flexion and rotation and the capsular pattern, and the positive labrum provocation tests should be completely normal or greatly improved immediately after infiltration.



a

b

Fig. 5.25 Posterior margin test. **(a)** Hip is forcibly flexed, abducted, and externally rotated. **(b)** Hip is extended in adduction and internal rotation.

5

Kalchschmidt Hip Dysplasia Tests

Assesses symptoms caused by hip dysplasia.

Most patients with symptoms due to hip dysplasia report pain with weight bearing felt in the groin, in the region of the greater trochanter, or in both areas. However, there are patients who cannot clearly identify the anatomical region of the symptoms and complain of pain in the lower back, buttock, and thigh.

The following tests are helpful where clinical and radiographic evidence suggests painful hip dysplasia:

- **Test 1:** With the patient standing on the painful leg and the examiner guiding the patient's shoulders, the examiner turns the patient's body so that the affected hip is in maximum external rotation (**Fig. 5.26a, b**). Backward bending also hyperextends the hip.

Where symptoms are attributable to hip dysplasia, this posture will cause groin pain. When the patient then bends forward and the hip is brought into internal rotation by the examiner's guiding of the patient's shoulders, the pain disappears.

- **Test 2:** The patient is prone (a sandbag may also be placed under the knee). While pressing on the patient's buttock, the examiner passively flexes the patient's knee 90° and applies increasing resilient pressure to externally rotate the thigh (**Fig. 5.26c**).

Where symptoms are attributable to hip dysplasia, the patient will report pain in the groin region. This test provides useful diagnostic information when both sides are compared, and is easy to perform.

- **Test 3:** The patient is supine. First the examiner palpates the hip beneath the anterior inferior iliac spine. The examiner then uses the hypothenar eminence of the extended arm to place gradually increasing pressure on the femoral head (**Fig. 5.26d**).

Where symptoms are attributable to hip dysplasia, the patient will report pain. This test provides useful diagnostic information, especially when both sides are compared. Often, the examiner will observe that performing the test presses an eccentric, anteriorly displaced femoral head back into the acetabulum.

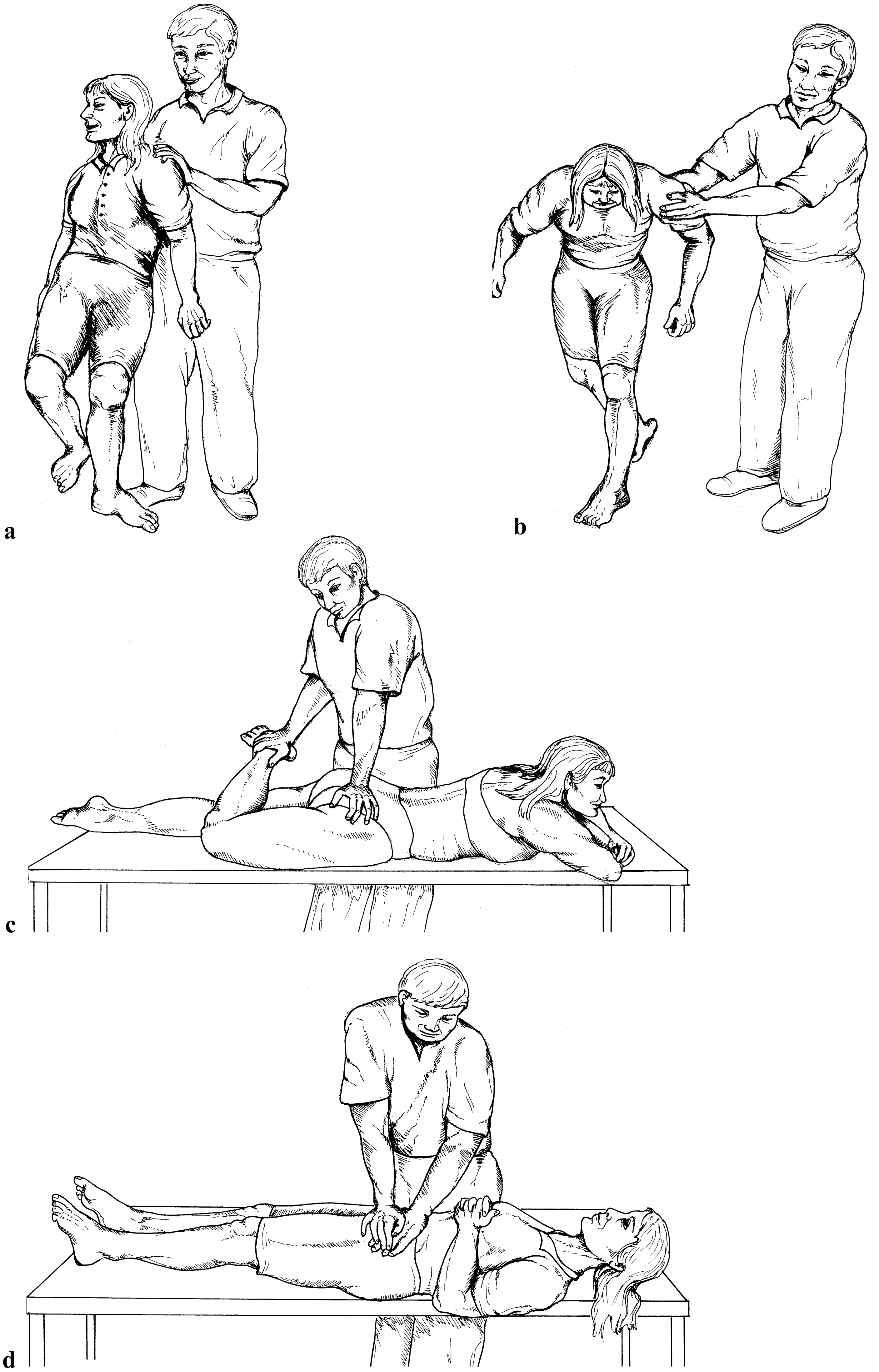


Fig. 5.26 Kalchschmidt hip dysplasia tests. **(a, b)** Test 1. **(c)** Test 2. **(d)** Test 3.

Femoroacetabular Impingement Test

Procedure: The patient lies supine. The examiner grasps the affected leg near the heel with one hand and at the knee with the other and passively flexes the hip and knee. Then the hip is hyperflexed, internally rotated, and adducted.

Assessment: A positive test result is indicated by groin pain with limitation of flexion and internal rotation. The forced movement combining flexion, adduction, and internal rotation brings the femoral neck into contact with the anterolateral acetabular rim. Impingement occurs when bony prominences at the junction of the femoral head and neck (cam impingement) and/or at the anterior rim of the acetabulum (pincer impingement) cause the femoral neck to contact the anterior rim of the acetabulum earlier than normal. The same occurs also in the case of deep acetabular sockets. This impingement causes lesions of the acetabular labrum and joint cartilage, especially in young and physically active individuals, who clinically experience groin pain when sitting and when involved in sports activities.

The symptoms are usually partially or completely relieved by the movement combining flexion and external rotation, during which the femoral neck moves laterally by the anterior acetabular roof without impingement. Restrictions of internal rotation and of flexion occur in multiple other disorders that must be considered in the differential diagnosis, including Legg–Calvé–Perthes disease, epiphysiolysis, and decreased anteversion.

Note: Forced passive hyperextension and external rotation can cause a painful anterior subluxation of the femoral head, in which the femoral head contacts the labrum, which is partially or completely torn (in hip dysplasia). Deep-seated joint pains suggest posteroinferior impingement.

Decreasing the femoral offset (cam impingement) as well as extending the roof can cause structural changes leading to the development of osteoarthritis of the hip.

If a labral lesion is present, forcing the movement combination of hip flexion, abduction, and internal rotation will lead to pain due to contact of the femoral neck with the anterolateral acetabular rim (impingement test).

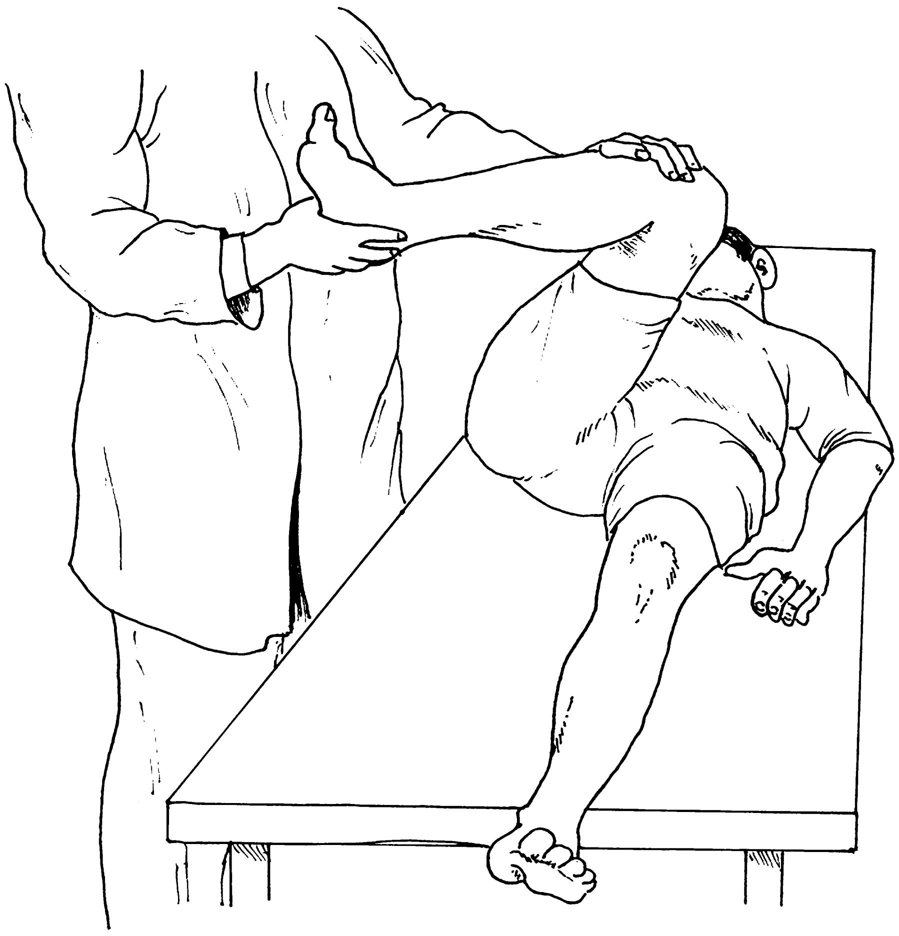


Fig. 5.27 Femoroacetabular impingement test.

6 Knee

Our knowledge of the knee has expanded significantly over the last few decades. New information about anatomy, biomechanics, and pathophysiology has improved the detection and treatment of knee disorders. Injuries to the knee, particularly in conjunction with sporting activities, have become a major focus of interest.

Noninvasive modalities such as ultrasound, computed tomography, and magnetic resonance imaging today allow precise assessment of diseased and injured structures in the knee. Diagnostic arthroscopy has evolved into a surgical method of treatment.

Diagnostic assessment of knee symptoms begins with history-taking and physical examination. Anteroposterior and lateral radiographs of the knee together with an axial view of the patella and trochlear groove are required to detect changes in bony structures right at the start.

It is very important to identify the location and type of pain as well its duration and/or when it occurs (pain with weight bearing, joint blockade, etc.). Inspection and evaluation of axial deviations (genu valgum, genu varum, genu recurvatum, or a flexion deformity), swelling of the knee, and muscle atrophy provide information about the possible causes of joint symptoms. Palpation then allows the examiner to identify diseased joint structures with greater accuracy and assess them in greater detail. Clinical tests of passive and active motion, some of which entail complex motions, also aid in making a diagnosis. Understanding how the accident occurred is important for diagnosing knee injuries. The type and severity of the injury are dependent on the direction, duration, and intensity of the trauma and on the position of the joint at the time of the injury.

Sports injuries and developmental anomalies (axial deviations, malformation of the patella, etc.) are the most common causes of knee complaints in children and young adults. For example, Osgood–Schlatter disease should be suspected when an adolescent engaged in a jumping sport in school athletics complains of pain in the tibial tuberosity. In older adolescents, one should suspect patellar tendinitis ("jumper's knee"). Degenerative damage to the meniscus can lead to sudden meniscus symptoms with impingement without an identifiable causative event even in early adulthood. In older patients, incipient or advanced wear in the joint due to aging processes, posttraumatic conditions, occupational stresses, and congenital or acquired deformities is most often responsible for knee symptoms. Diffuse knee pain occurring in an older patient in the absence of trauma is almost invariably a sign of meniscus degeneration or joint wear. Swelling and a sensation of heat in the knee are normally present as well. Patients with retropatellar arthritis complain of pain on climbing stairs and walking downhill, occasionally accompanied by a feeling of instability. Patients with Baker cysts report pain in the popliteal fossa.

Aside from these characteristic descriptions of pain, any uncharacteristic pain described by the patient should be carefully assessed. The differential diagnosis must include disorders of the adjacent joints. Patients with osteoarthritis of the hip will often report pain radiating into the knee. Changes in the sacroiliac joints or lumbar spine, leg shortening, axial deviations, and ankle deformities can also cause knee symptoms.

Disorders of other organ systems should also be considered when assessing distal neurovascular dysfunction. The knee is affected in 60% of all cases in rheumatoid arthritis. Lyme disease should also be considered as a possible cause of isolated arthritis of the knee. A thorough history and extensive laboratory diagnostic studies are helpful in the differential diagnosis of such knee disorders.

Knee Tests

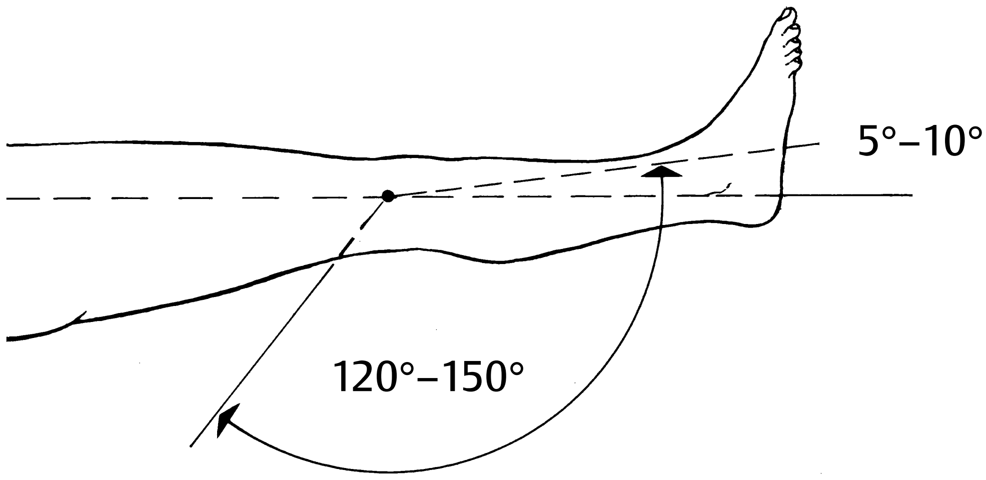


Fig. 6.1 Flexion and extension. Internal and external rotation do not occur in extension. In 90° of knee flexion with the lower leg hanging freely, the knee exhibits a range of motion from 10° of internal rotation to up to 25° of external rotation.

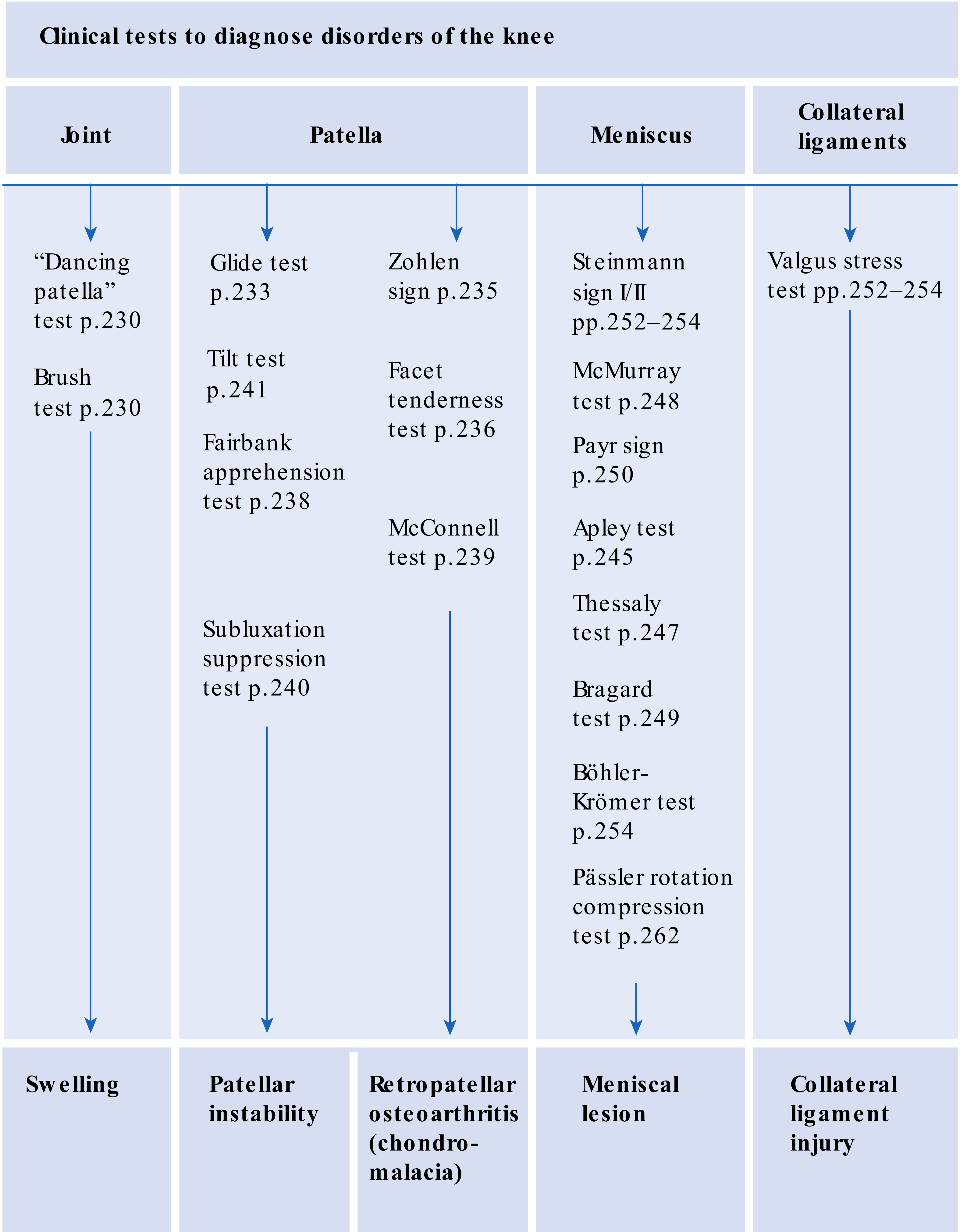


Fig. 6.2 Clinical tests to diagnose disorders of the knee.

Cruciate ligament		Plica	Quadriceps	Condyle
Lachman/stable Lachman test pp.266–269	Posterior drawer test p.282	Mediopattellar plica test p.242	Dreyer test p.242	Wilson test p.263
Active Lachman test p.271	Posterior sag sign p.290	Hughston plica test p.243		
Anterior drawer test p.272	Loomer test p.295			
Pivot shift test p.275	Reversed Jakob pivot shift test p.289			
Jakob graded pivot shift test p.276	Godfrey test p.294			
Martens test p.282				
Anterior cruciate ligament lesion	**Posterior cruciate ligament lesion, posterior rotational instability**	**Plica lesion**	**Tear of the quadriceps tendon**	**Osteo-chondritis dissecans**

6

Muscle Stretch Tests

Testing the condition of the muscles goes hand-in-hand with range of motion testing. It is important in this regard to take note of shortening and contractures in the muscles of the upper and lower leg in addition to defining the individual muscle groups.

Quadriceps Stretch Test

□ **Procedure:** With the patient prone, the examiner passively flexes the knee and presses the heel toward the buttock.

□ **Assessment:** Normally, the heel can be brought all the way to the buttock bilaterally. Increased distance between heel and buttock is indicative of shortening of the quadriceps muscle.

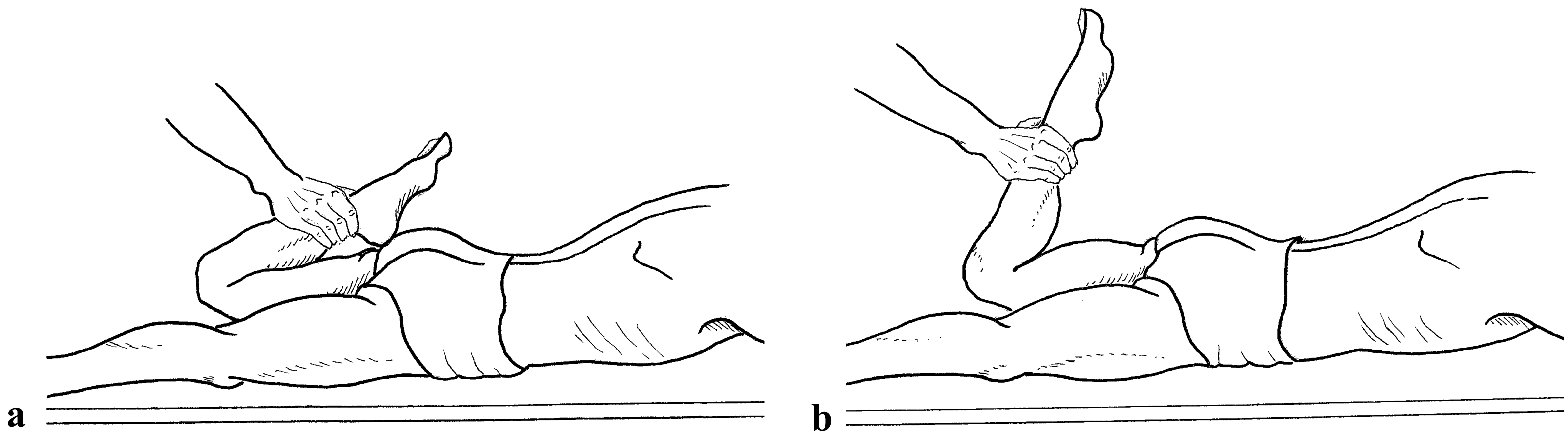


Fig. 6.3 Quadriceps stretch test. **(a)** Heel reaches buttock. **(b)** Restricted range of motion due to shortened quadriceps.

Rectus Femoris Muscle Stretch Test

□ **Procedure:** Testing of the rectus femoris muscle is carried out in the supine position. The patient uses his or her hands to hold the unaffected leg in maximal flexion. The examiner flexes the knee of the affected leg, which is hanging over the end of the examination table.

□ **Assessment:** One can normally attain knee flexion over 90° easily while keeping the hip extended, but shortening of the rectus femoris muscle causes a decrease in knee flexion to less than 90°.

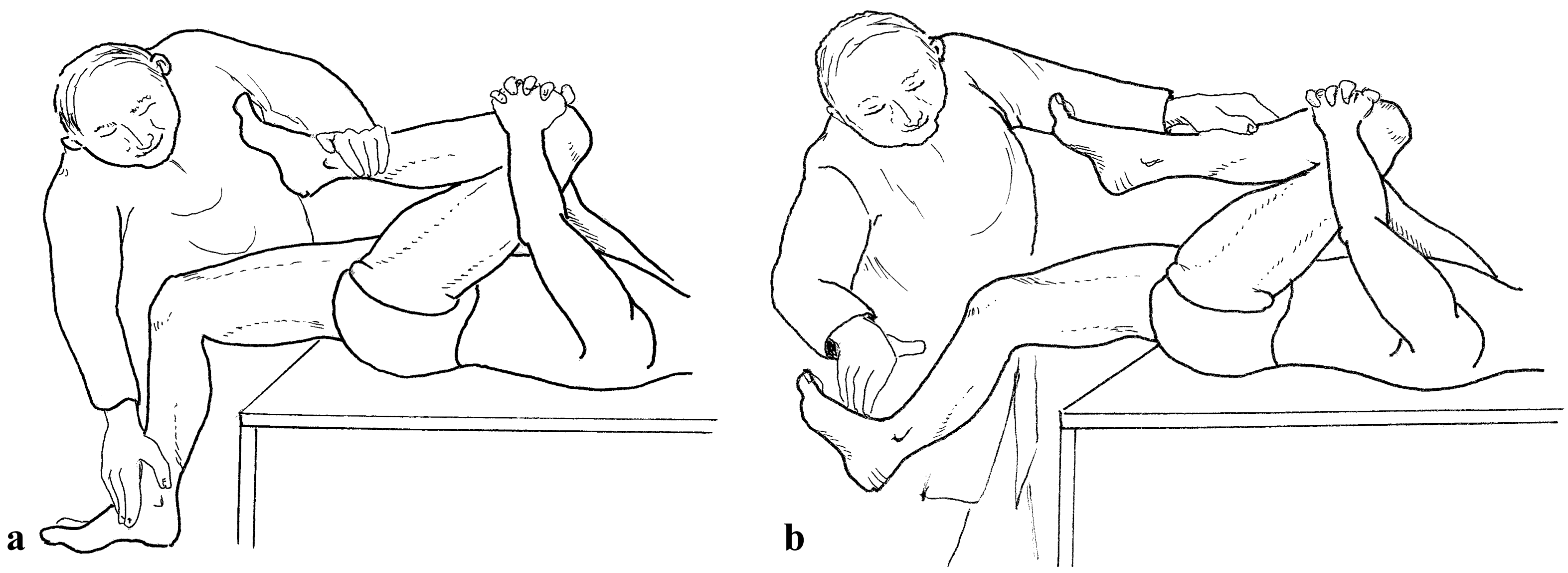


Fig. 6.4 Rectus femoris muscle stretch test. **(a)** 90° knee flexion with extended hip. **(b)** Restricted knee flexion due to shortening of the rectus femoris muscle.

6

Hamstring Muscle Stretch Test

Procedure: The patient is supine for the hamstring muscle test. The examiner raises the extended leg and records the degree of attainable hip flexion with lumbar lordosis eliminated (lumbar spine flat against the table).

Assessment: An angle of less than 90° is considered to be abnormal. When the hamstring muscles are shortened, further hip flexion is only made possible by flexing the knee.

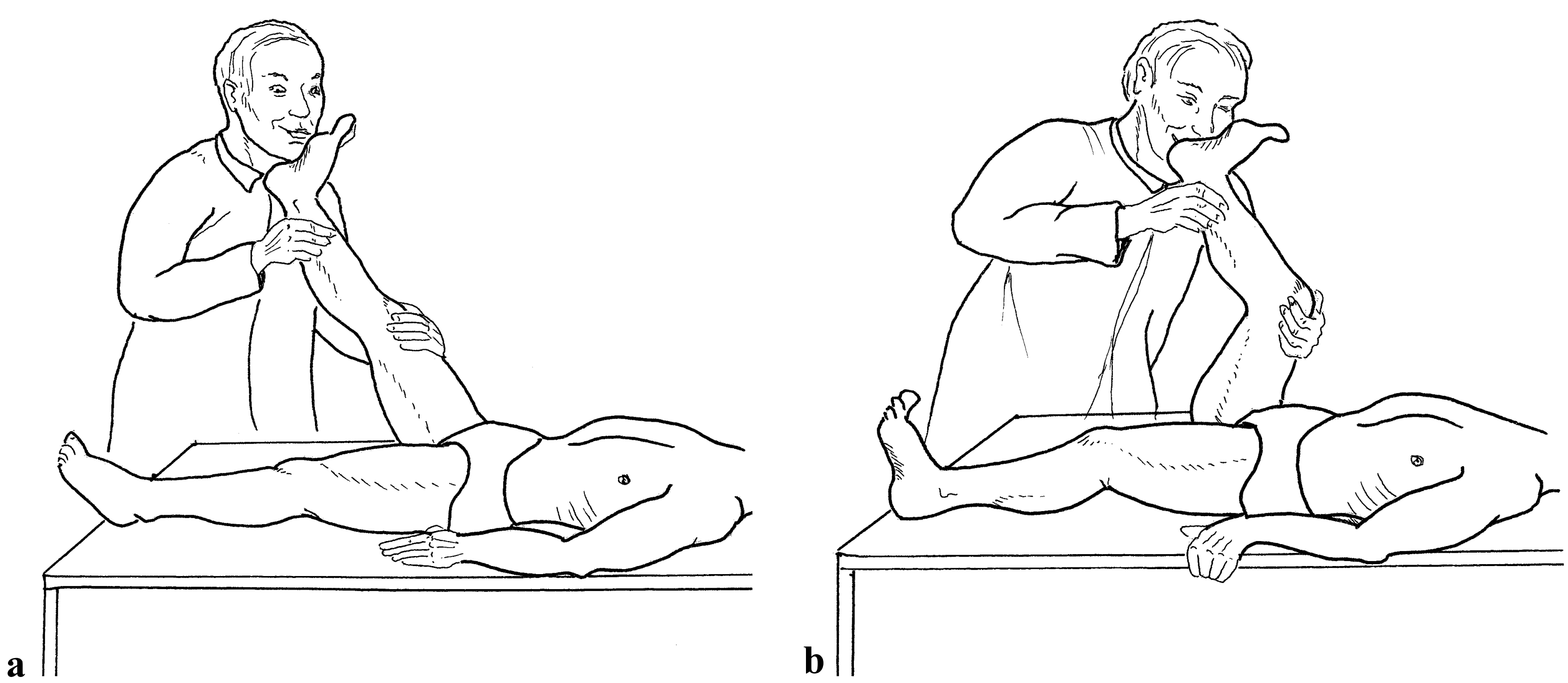


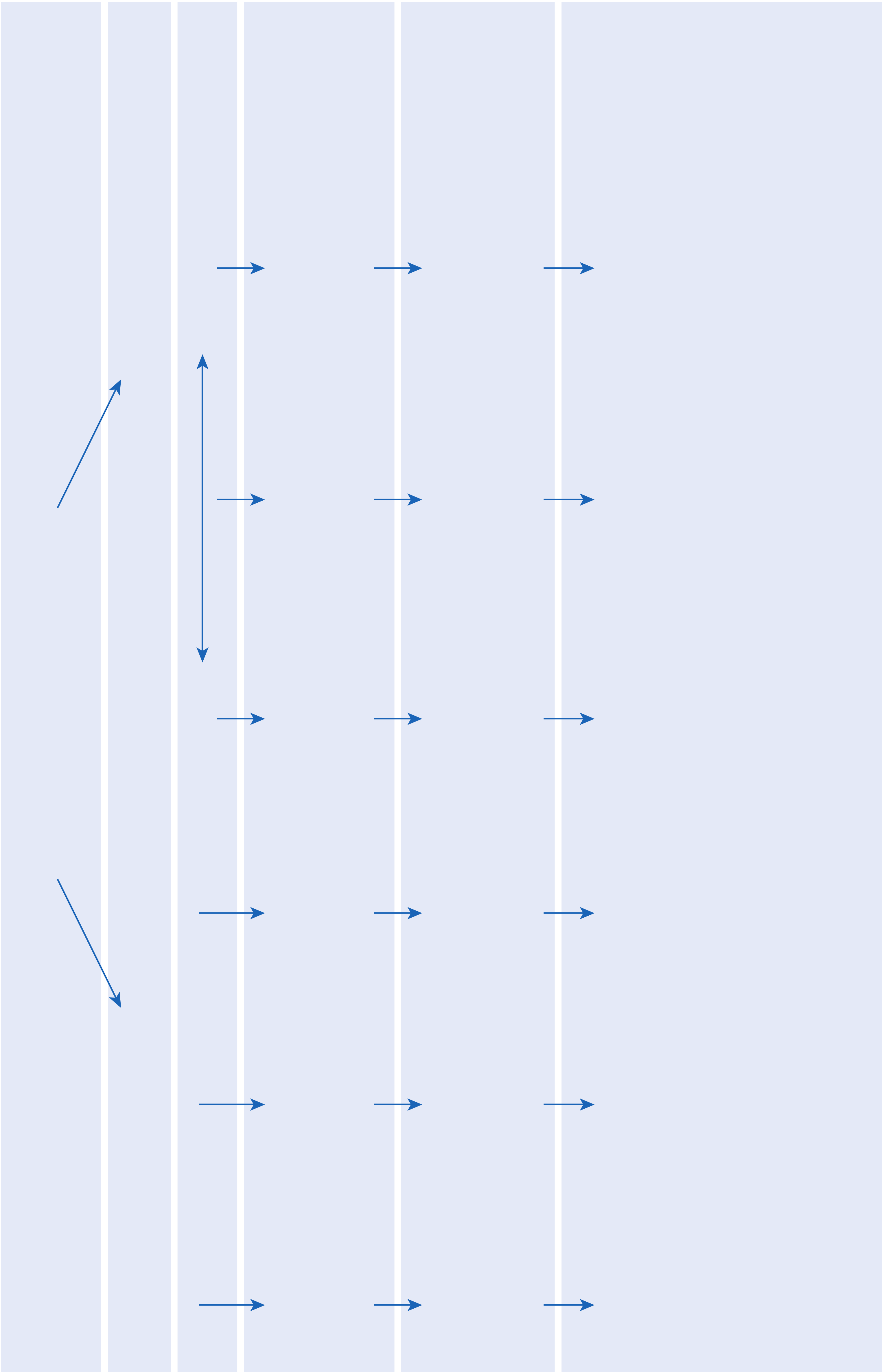
Fig. 6.5 Hamstring muscle stretch test. **(a)** Limited hip flexion due to shortened hamstring muscles. **(b)** 90° hip flexion possible by flexing knee.

Knee Swelling

When assessing swelling, the examiner must determine the type and amount of swelling that is present. The examiner must differentiate between swelling and synovial thickening. Normally the knee contains 2 mL of synovial fluid. A bloody effusion (hemarthrosis) is usually traumatic, caused by a ligament tear (usually of the anterior cruciate ligament), osteochondral fracture, or peripheral meniscus tear. A hemarthrosis usually develops very quickly, within 1 to 2 hours. On palpation it has a “doughy” feeling and is relatively hard to the touch. The overlying skin feels warm.

Nontraumatic synovial effusion caused by chronic joint disease normally takes several hours or even days to develop. The examiner feels a fluctuant joint effusion; the overlying skin is mildly warm to the touch. Synovial effusion usually develops from overuse of the knee and disappears after a few days of inactivity.

In the case of a joint infection, the development of the effusion is delayed. The overlying skin is very hot and red. Palpation demonstrates a large joint effusion that is taut and tender. Movement is clearly limited by pain.



Brush (Stroke, Wipe) Test

For assessing minimal effusion.

□ **Procedure:** The patient lies supine. The examiner uses one hand to "smooth out" the medial side of the knee from distal to proximal. With the other hand the examiner presses the superior recess moving distally from a proximal and lateral position.

□ **Assessment:** Even with a mild joint effusion, a wave of fluid may be felt in the medial, distal part of the joint.

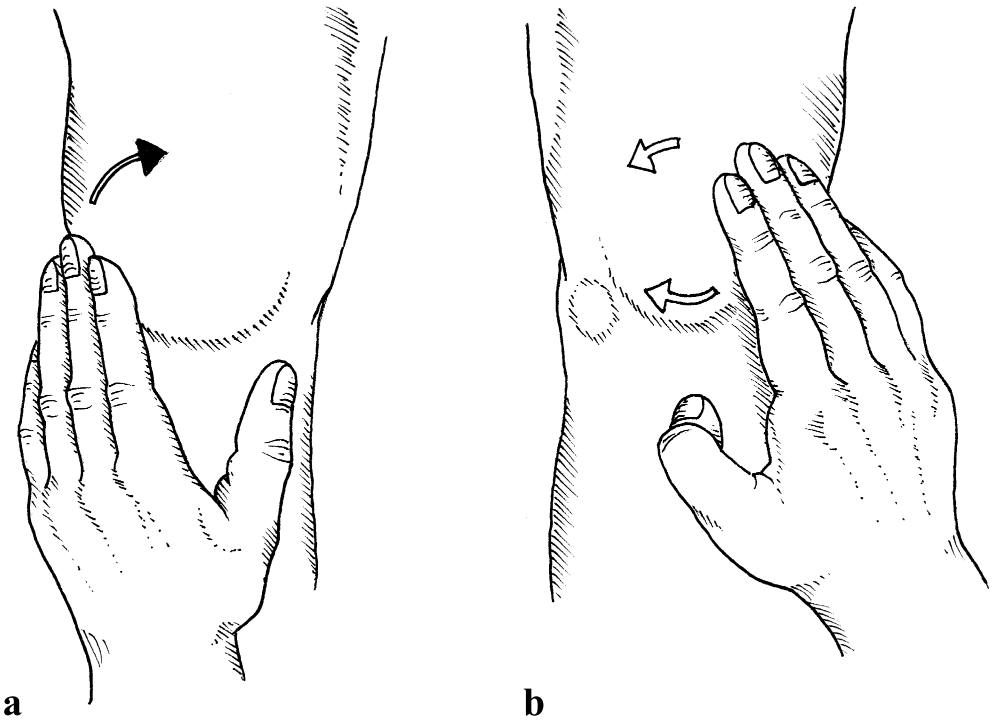

a b

Fig. 6.6 Brush test. **(a)** From distal to proximal. **(b)** From proximal to distal.

"Dancing Patella" Test

Indicates effusion in the knee.

□ **Procedure:** The patient is supine or standing. With one hand, the examiner smoothes the suprapatellar pouch from proximal to distal while pressing the patella against the femur with the other hand or moving it medially and laterally with slight pressure.

□ **Assessment:** Resilient resistance (a dancing patella) is abnormal and suggests effusion in the knee.

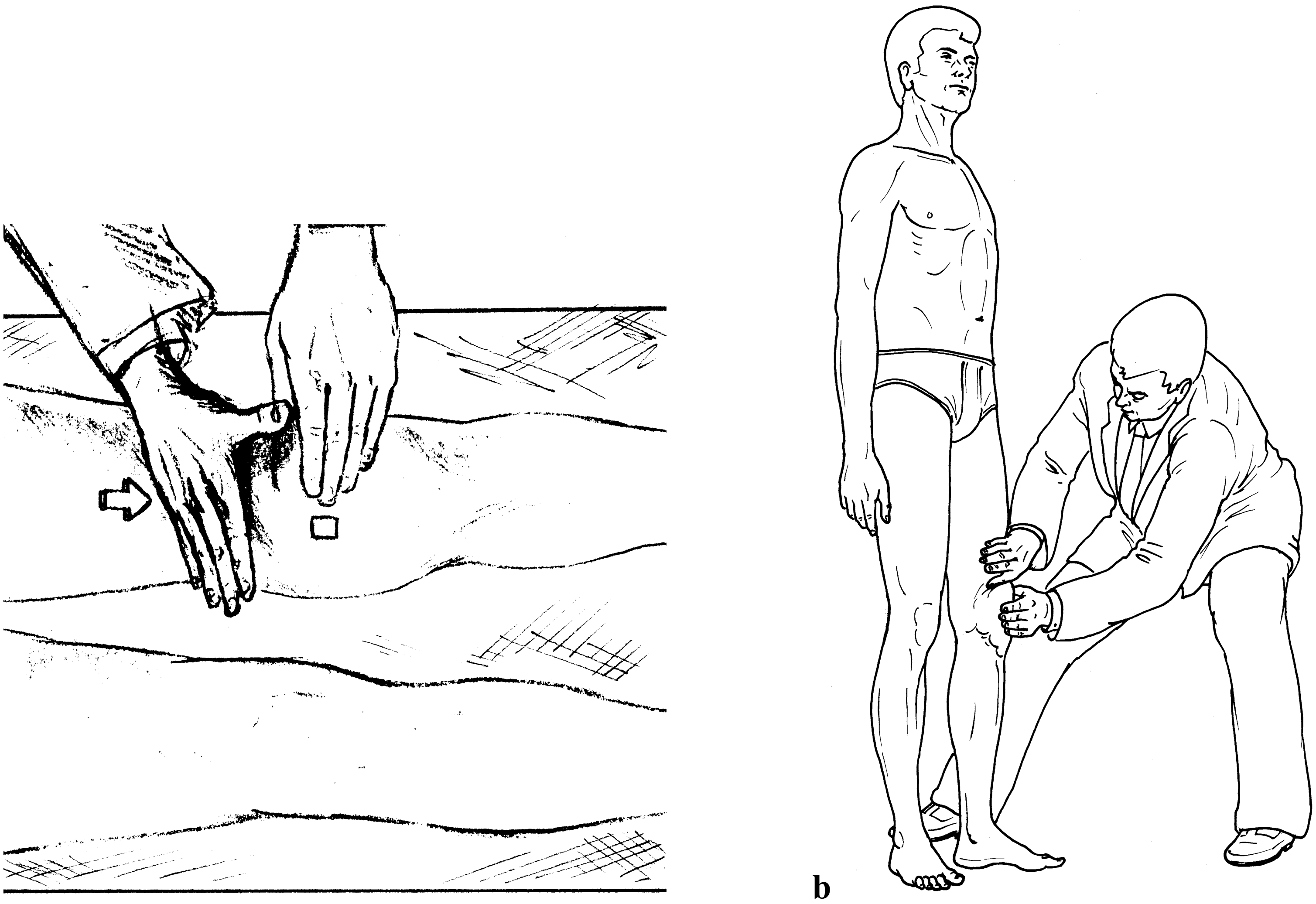


Fig. 6.7 "Dancing patella" test. **(a)** With the patient supine. **(b)** With the patient standing.

Patella

When reporting patellofemoral knee pain, patients usually complain of diffuse pain over the anterior knee. In this context, one speaks of a peripatellar or anterior knee syndrome. As a rule, the pain increases with weight bearing, especially climbing steps. Prolonged sitting makes it worse. Pain that is constantly worse with weight bearing suggests cartilage damage, while symptoms that are initially improved by activity but later worsen are indicative of a muscle or tendon problem, such as tendinopathy of the patellar tendon of the quadriceps muscle or of the iliotibial band.

"Giving way" is also a typical symptom that can occur as a reflex to retropatellar pain.

Meniscal and cruciate ligament disorders and patellar subluxation or dislocation are to be differentiated from this.

Retropatellar rubbing suggests early retropatellar osteoarthritis and is often associated with a mild knee effusion.

Pronounced joint effusion occurs after dislocation with disruption of the medial patellar retinaculum.

Clinical examination begins with a thorough evaluation of the axis of the leg in terms of genu valgum or genu varum, tibial torsion, femoral anteversion, and pronation of the foot. In addition, evaluate the position of the patella, which can occur as patella infera (baja), patella alta, or patellar lateralization.

Marked pronation of the foot is associated with internal rotation of the tibia, which leads to increased stress on the peripatellar soft tissue structures and causes anterior knee pain.

Following that, one should check the mobility of the patella, which is normally positioned slightly laterally when the knee is extended. With increasing flexion, the patella moves medially and tracks into the femoral trochlear groove. In the case of an unstable patella or patella alta, the patella does not enter the trochlear groove until the knee is markedly flexed.

Overstressing the patellofemoral joint with a poorly oriented extensor mechanism (maltracking) leads to anterior and peripatellar knee pain with tendinopathies and retropatellar cartilage changes.

Malalignment of the axis of the leg is associated with an increased Q-angle, which in turn can be a measure of patellofemoral disease.

Q-Angle Test

The Q-angle is defined as the angle between the quadriceps muscle (primarily the rectus femoris) and the patellar tendon. It corresponds to the physiologic valgus angle of the femoral shaft. This creates a lateral pull on the patella. This tendency is a factor in habitual patellar dislocation and in the patellofemoral pain syndrome (anterior knee pain). It also creates problems in total knee arthroplasty.

▸ **Procedure:** A line is drawn from the anterior superior iliac spine to the midpoint of the patella, corresponding to the quadriceps' tensile direction, and from the tibial tubercle to the midpoint of the patella, corresponding to the patellar tendon. The angle formed by the crossing of these two lines is called the Q-angle. The hip and the foot should be placed in a neutral position, because significant internal rotation of the leg and pronation of the foot alter the Q-angle.

▸ **Assessment:** Normally the Q-angle is 13° for men and 18° for women when the knee is straight. Any angle less than 13° may be associated with patellofemoral dysfunction or patella alta. A Q-angle greater than 18° is often associated with subluxing patella, increased femoral anteversion, genu valgum, or increased lateral tibial torsion.

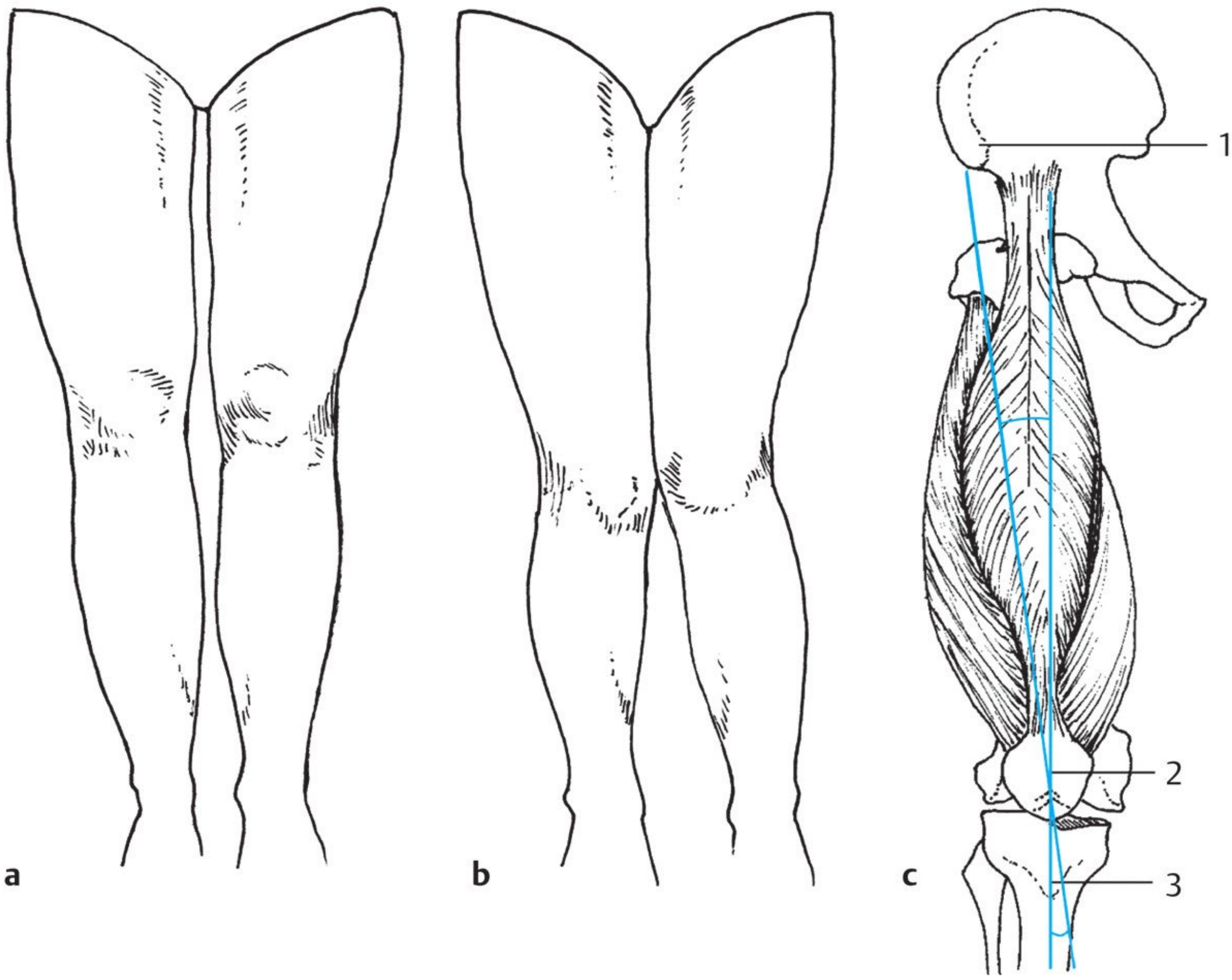


Fig. 6.8 Q-angle test. **(a)** Leg axis normal. **(b)** Left: genu valgum with increased Q-angle. **(c)** Defining the Q-angle:

1 Anterior superior iliac spine.
2 Midpoint of the patella.
3 Tibial tuberosity.

Patellar Mobility Test (Patellar Glide Test)

Procedure: The patient is supine. The examiner stands at the patient's side next to the knee and grasps the proximal half of the patella with the thumb and index finger of one hand and the distal half with the thumb and index finger of the other. For the lateral glide test, the examiner's thumbs push the patella laterally over the lateral femoral condyle and the index fingers resting there. For the medial glide test, the examiner's index fingers push the patella in the opposite direction. In each case, the examiner's index finger or thumb can palpate the projecting posterior surface of the patella. Where increased lateral mobility is suspected, the same test is performed to assess stability with the quadriceps tensed. The patient is asked to lift his or her foot off the examining table. The examiner then notes the resulting motion of the patella. The medial and lateral glide tests provide information about the degree of tension in the medial or lateral retinaculum, respectively. The tests should always be performed comparatively on both knees.

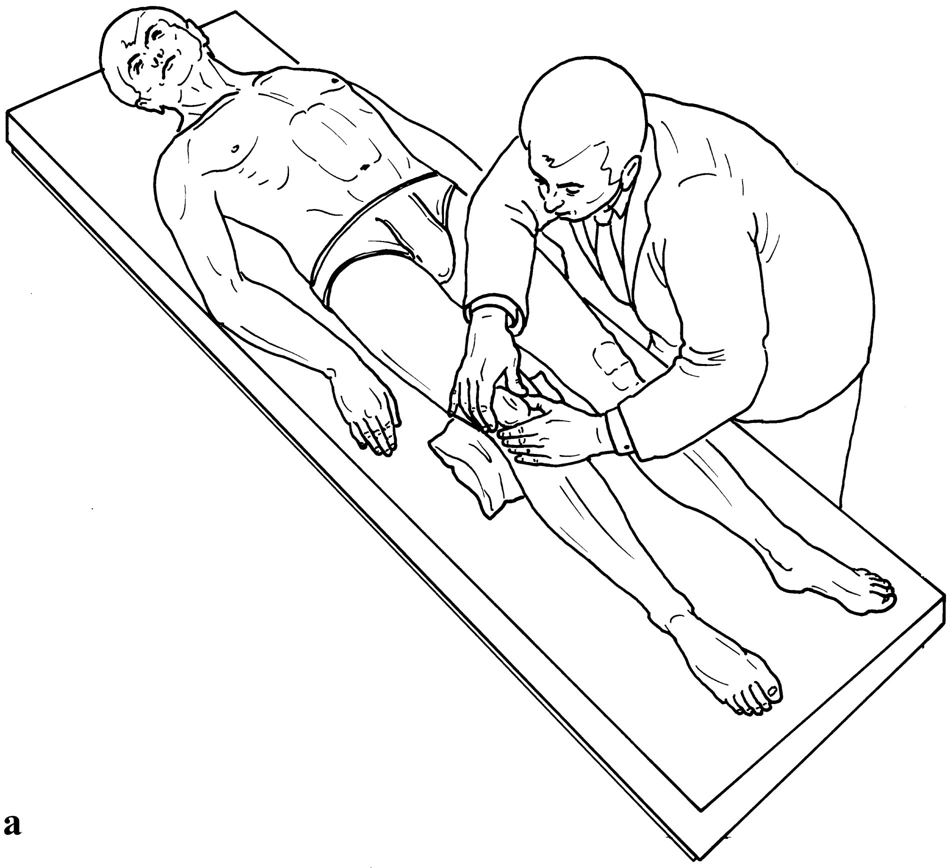
With the hands in the same position, the examiner can also place traction on the patella by lifting it off the condyles.

□ **Assessment:** Normal physiologic findings include painless, symmetrical mobility of both patellae without any crepitation or tendency to dislocate. Increased lateral or medial mobility of the patella suggests laxity of the knee ligaments or habitual patellar subluxation or dislocation.

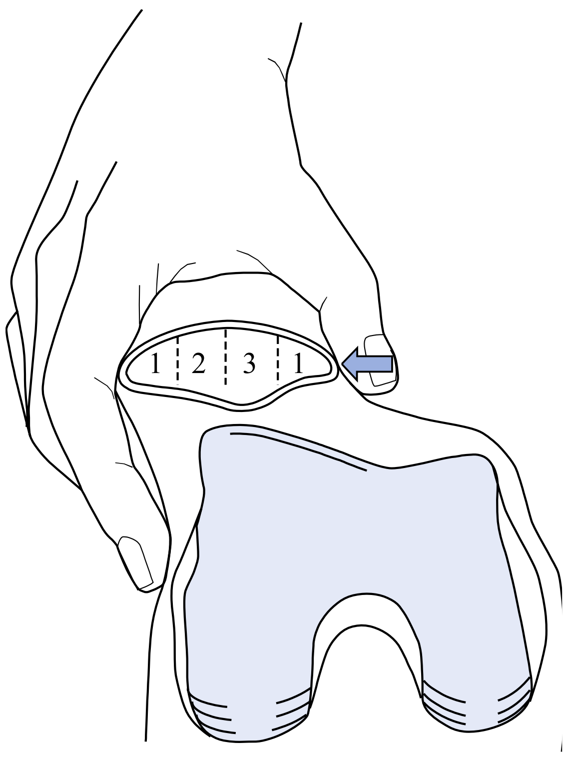
To define patellar mobility, the patella is divided longitudinally into four equal quadrants. A hypomobile patella is defined as one that cannot be mobilized medially or laterally over one quadrant. A hypermobile patella is one that can be moved two quadrants, or one-half of the patellar width, medially or laterally.

Crepitation (retropatellar friction) occurring when the patella is mobilized suggests chondropathy or retropatellar osteoarthritis.

□ **Note:** With the hands in the same position, the examiner can expand the test by moving the patella distally. Decreased distal mobility of the patella suggests shortening of the rectus femoris or patella alta (high-riding patella).



a



b

Fig. 6.9 Patellar mobility test. **(a)** Medial and lateral mobility of the patella. **(b)** Division of the patella into four quadrants.

Zohlen Sign

□ **Procedure:** The patient is supine with the leg extended. The examiner applies medial and lateral pressure to the proximal patella to press it into the trochlear groove and asks the patient to extend the leg further or to tense the quadriceps. In the second phase, the examiner pushes the patella directly downward into the trochlear grove while the patient contracts the quadriceps. This test is called the Clarke sign or the patellar grind test.

□ **Assessment:** The quadriceps exerts a proximal pull on the patella, pressing it tightly against the trochlear groove. This will cause retropatellar and/or peripatellar pain in the presence of retropatellar cartilage damage.

□ **Note:** As this test is often positive in normal patients, one should repeat the procedure several times, increasing the pressure on the patella each time and comparing the results with those of the unaffected side. To better localize the retropatellar damage, the knee should be tested in 30°, 60°, and 90° of flexion as well as in full extension.

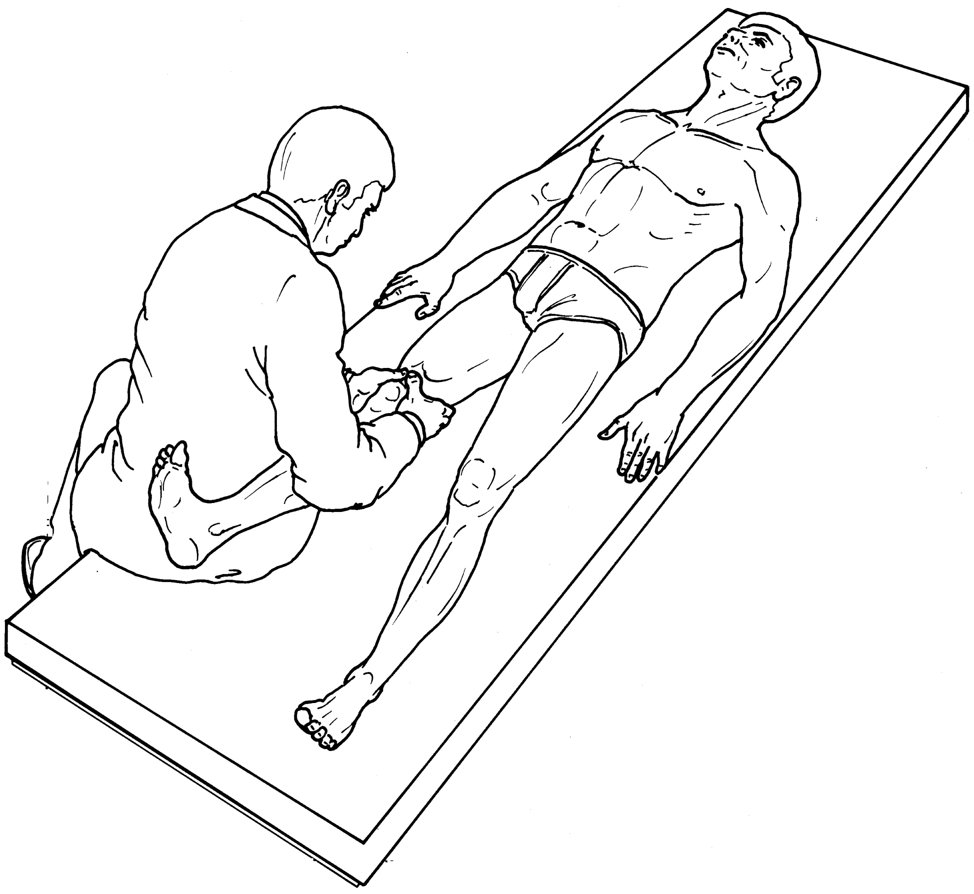


Fig. 6.10 Zohlen sign.

Facet Tenderness Test

□ **Procedure:** The patient is supine with the knee extended. The examiner first elevates the medial margin of the patella with his or her thumbs and palpates the medial facet with a thumb, then elevates the lateral margin with the index fingers and palpates the lateral facet with an index finger. Elevating the patella allows palpation of the retropatellar region, which is important in disorders such as chondromalacia. Tenderness to palpation at the distal pole of the patella can be a sign of patellar tendinitis ("jumper's knee").

□ **Assessment:** Patients with retropatellar osteoarthritis, tendinitis, or synovitis will report pain, especially when the examiner palpates the medial facet.

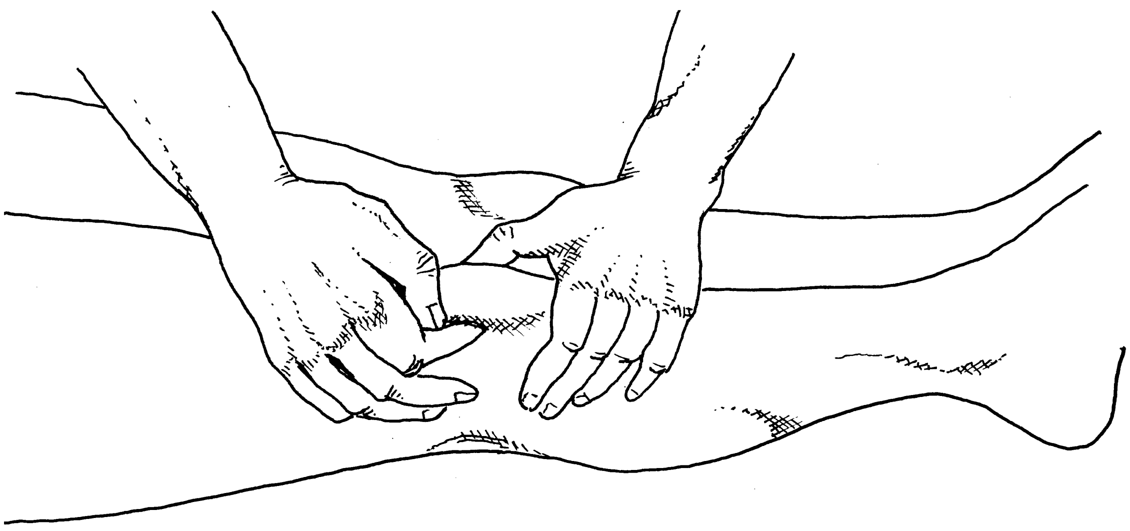


Fig. 6.11 Facet tenderness test.

Crepitation Test

□ **Procedure:** The examiner kneels in front of the patient and asks the patient to crouch down or do a deep knee bend. The examiner listens for sounds posterior to the patella.

□ **Assessment:** Crepitation ("snowball crunch" sound) suggests severe chondromalacia (grades II and III). Painless cracking sounds like those that occur in almost everyone during the first or second deep knee bend have no significance. For this reason, the patient is asked to do several deep knee bends. Usually the insignificant cracking sounds will decrease in intensity. In the absence of any audible retropatellar crepitation, the examiner may safely conclude that no severe retropatellar cartilage damage is present. However, the test results should not be used as a basis for far-reaching therapeutic decisions. They only provide information about the condition of the retropatellar cartilage. The crepitation test will be positive in many patients with normal knees.

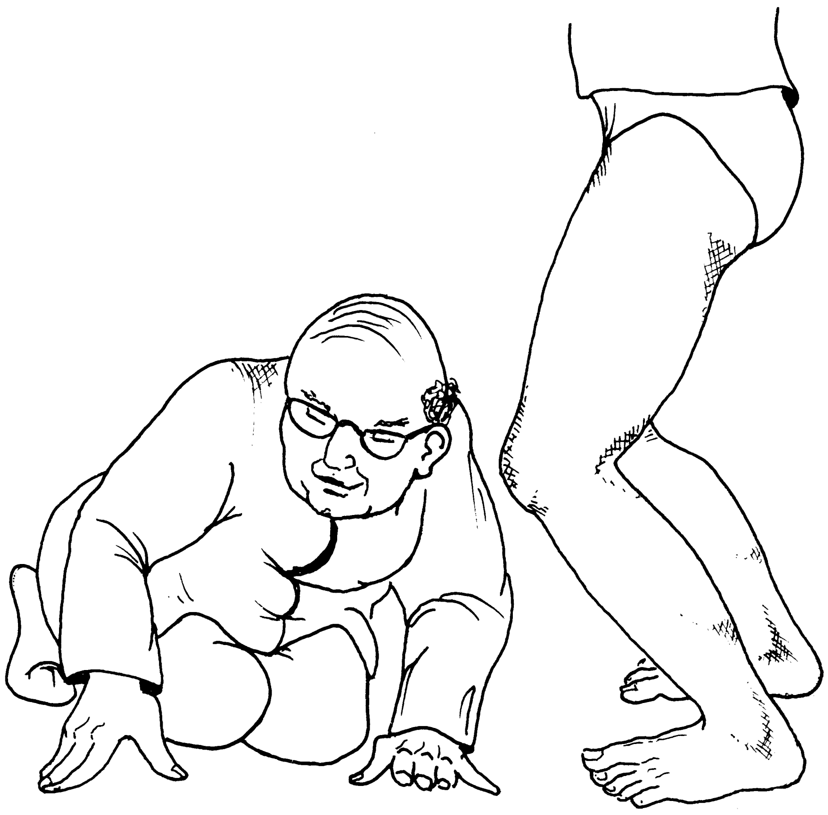
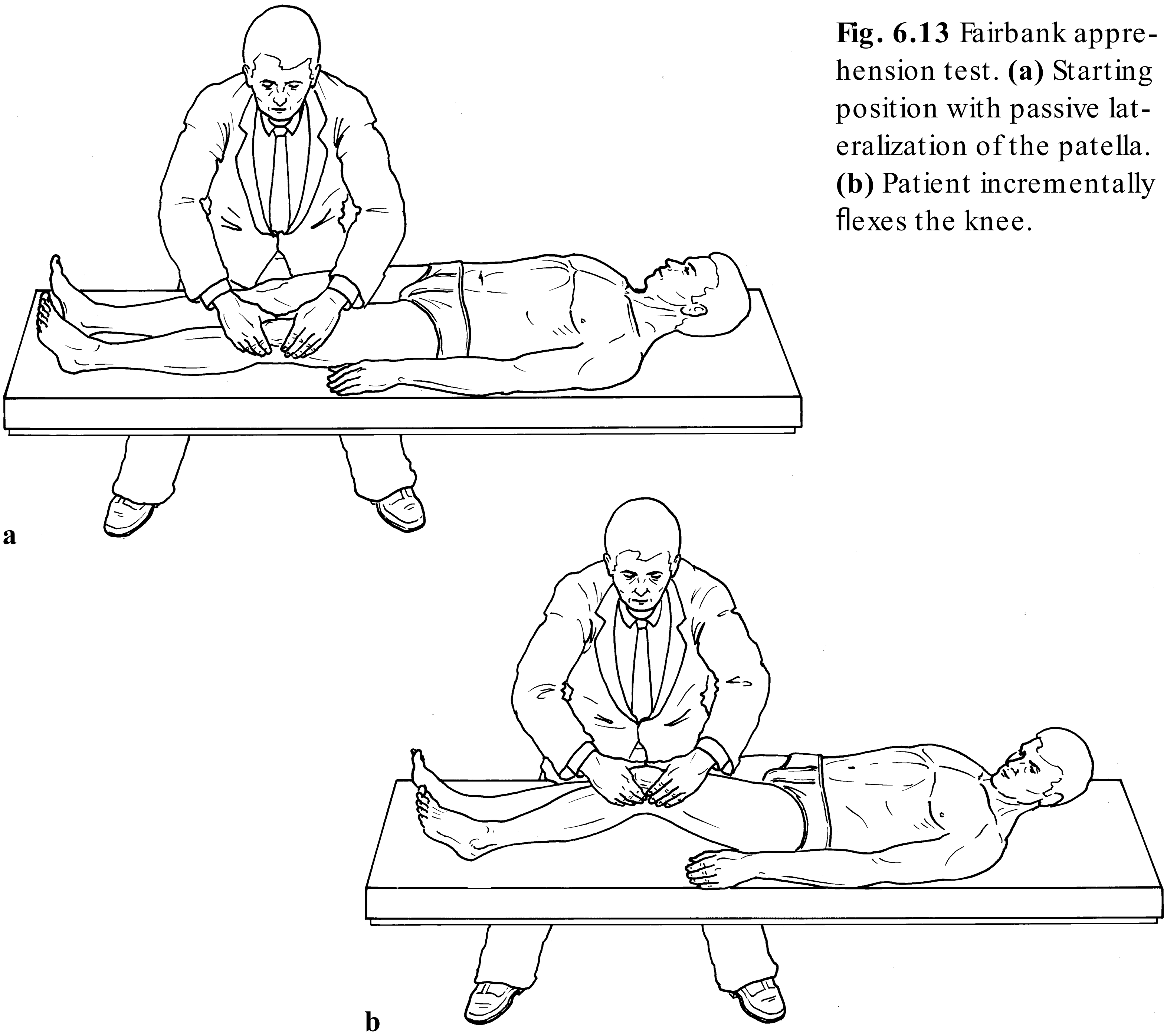


Fig. 6.12 Crepitation test.



a

b

Fig. 6.13 Fairbank apprehension test. **(a)** Starting position with passive lateralization of the patella. **(b)** Patient incrementally flexes the knee.

Fairbank Apprehension Test

□ **Procedure:** The patient is supine with the knee extended and the thigh muscles relaxed. The examiner attempts to simulate a dislocation (in a manner similar to the apprehension test in anterior instability of the shoulder) by placing both thumbs on the medial aspect of the knee and pressing the patella laterally. The patient is then asked to flex the knee.

□ **Assessment:** Where a patellar dislocation has occurred, the patient will report severe pain and will be apprehensive of another dislocation in extension or, at the latest, in flexion.

McConnell Test

□ **Procedure:** The patient is seated with the legs relaxed and hanging over the edge of the table. This test attempts to provoke patellofemoral pain with isometric tensing of the quadriceps. This is done with the knee in various degrees of flexion (0°, 30°, 60°, and 120°). In each position, the examiner immobilizes the patient's lower leg and asks the patient to extend the leg against the examiner's resistance (this requires contraction of the quadriceps).

□ **Assessment:** Where the patient reports pain or a subjective sensation of constriction, the examiner medially displaces the patella with his or her thumb. In a positive test, this maneuver reduces pain. The examination should always be performed comparatively on both knees. Alleviation of pain by medial displacement of the patella is a diagnostic criterion for the presence of retropatellar pain.

□ **Note:** In a positive McConnell test, pain can often be reduced by taping the knee so as to pull the patella medially. This "McConnell tape" bandage includes a lateral-to-medial slip that pulls the patella medially. A small plaster slip running medially from the middle of the patella is applied where a lateral patellar tilt requires correction. If required, a rotational slip extending from the medial knee to the tip of the patella and then to the lateral aspect can be applied to bring the patella into a neutral position. Physical therapy should concentrate on strengthening the vastus medialis and stretching the rectus femoris and iliotibial tract.

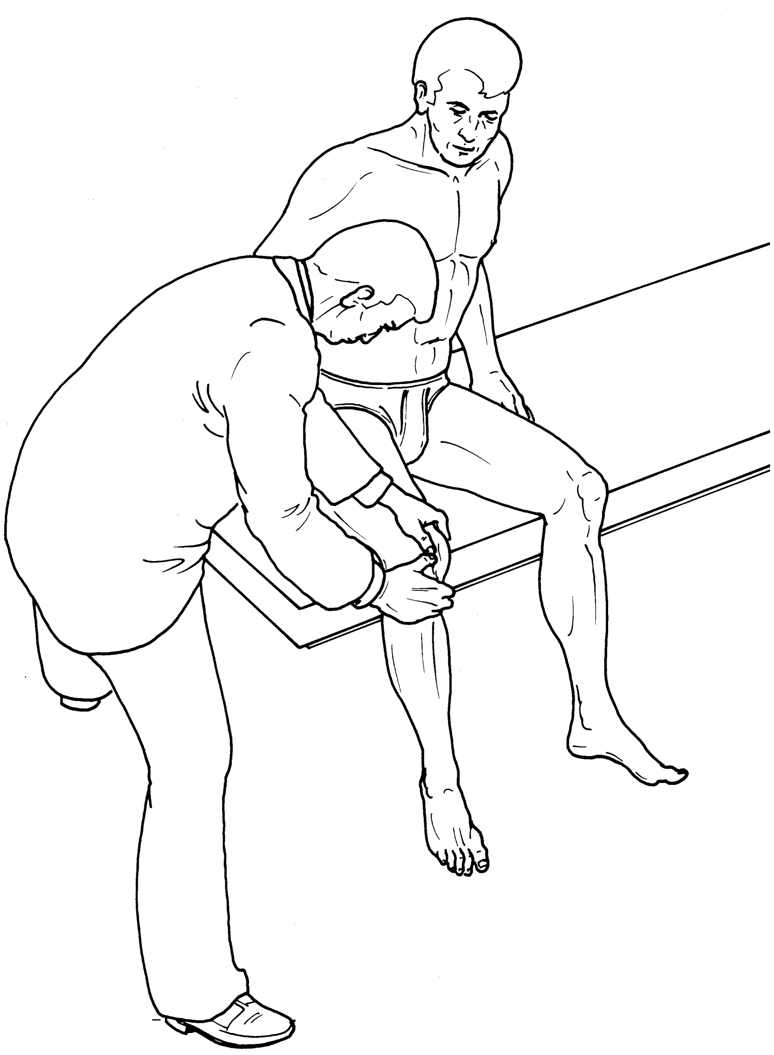


Fig. 6.14 McConnell test.

Subluxation Suppression Test

Demonstrates lateral or medial patellar subluxation.

Lateral Subluxation Suppression Test

□ **Procedure and Assessment:** To demonstrate lateral subluxation, the examiner places his or her thumbs on the proximal half of the lateral patellar facet. The patient is then asked to flex the knee. Either the thumb will be seen to prevent lateral subluxation or the examiner will feel the lateral motion of the patella. Flexing the knee without any attempt to prevent subluxation will lead to lateral patellar subluxation.

Medial Subluxation Suppression Test

□ **Procedure and Assessment:** To demonstrate medial subluxation, the examiner places his or her index fingers on the proximal half of the medial patellar facet. The patient is then asked to flex the knee. The examiner's finger will be seen to prevent medial subluxation. In contrast, flexing the knee without any attempt to prevent subluxation will lead to medial patellar subluxation (this is extremely rare).

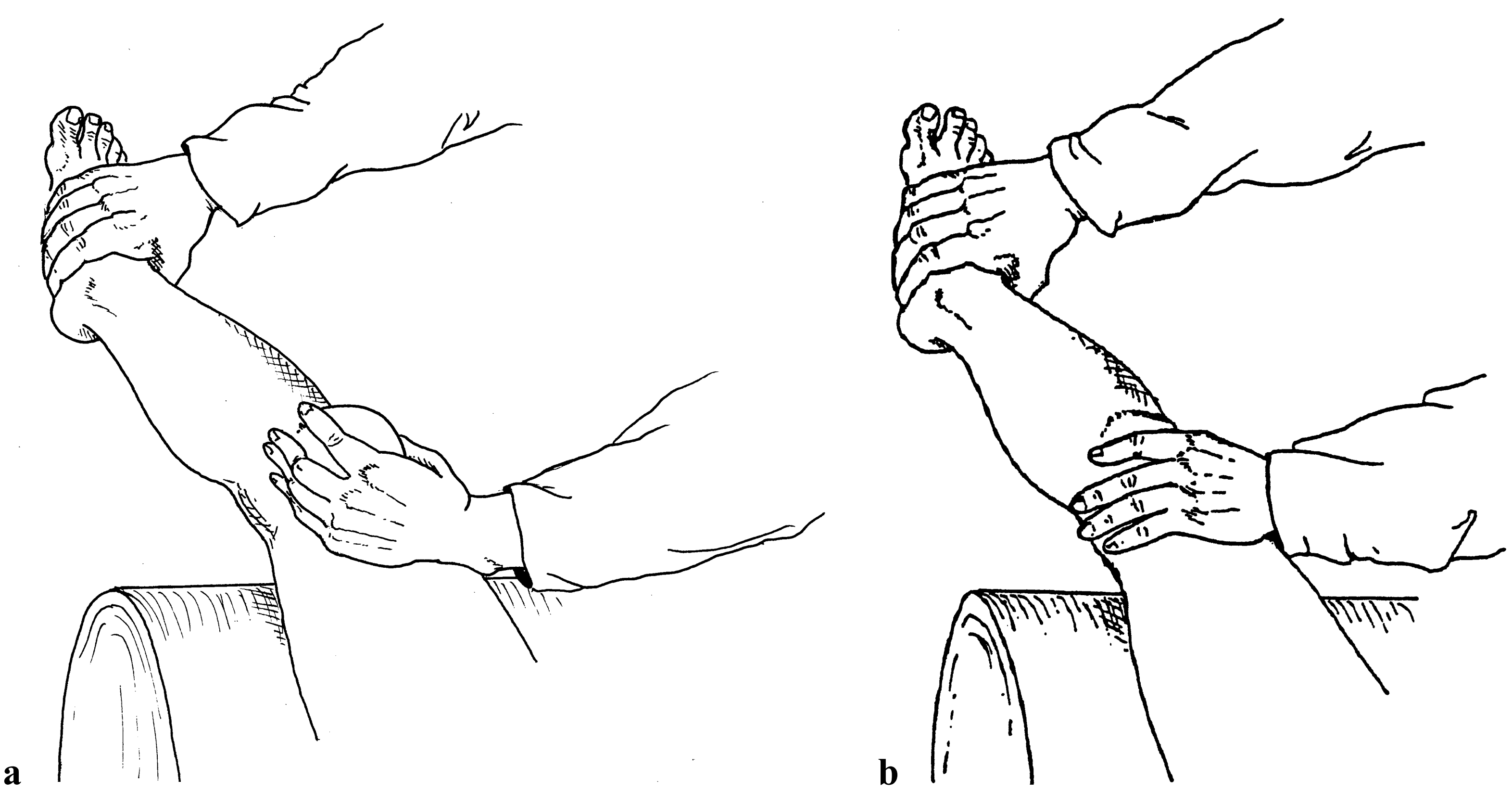


Fig. 6.15 Subluxation suppression test. **(a)** Lateral subluxation test. **(b)** Medial subluxation test.

Tilt Test

□ **Procedure:** The patient is supine. The examiner passively displaces the patella laterally, noting how it behaves during lateral displacement.
□ **Assessment:** Where the lateral retinaculum is very tight due to contracture, the lateral facet will dip toward the femur (negative "abnormal" tilt test). Where there is normal tone in the retinaculum, the patella will remain at roughly the same height with respect to the femur (neutral tilt test). With laxity of the lateral retinaculum and with generalized ligament laxity, the lateral margin of the patella will rise up out of the trochlear groove (positive tilt test).
□ **Note:** The primary purpose of the tilt test is to evaluate tension in the lateral retinaculum. Where the tilt test is neutral or positive, a lateral release to decompress the patellofemoral joint will hardly improve symptoms at all. However, it may be expected to improve symptoms in cases where the tilt test is negative. Patients with a positive tilt test greater than 5° and medial and lateral gliding of the patella exhibit poor results after an isolated lateral release. Dysplasia of the trochlear groove can lead to atypical test results. The tilt test should always be performed comparatively on both knees.

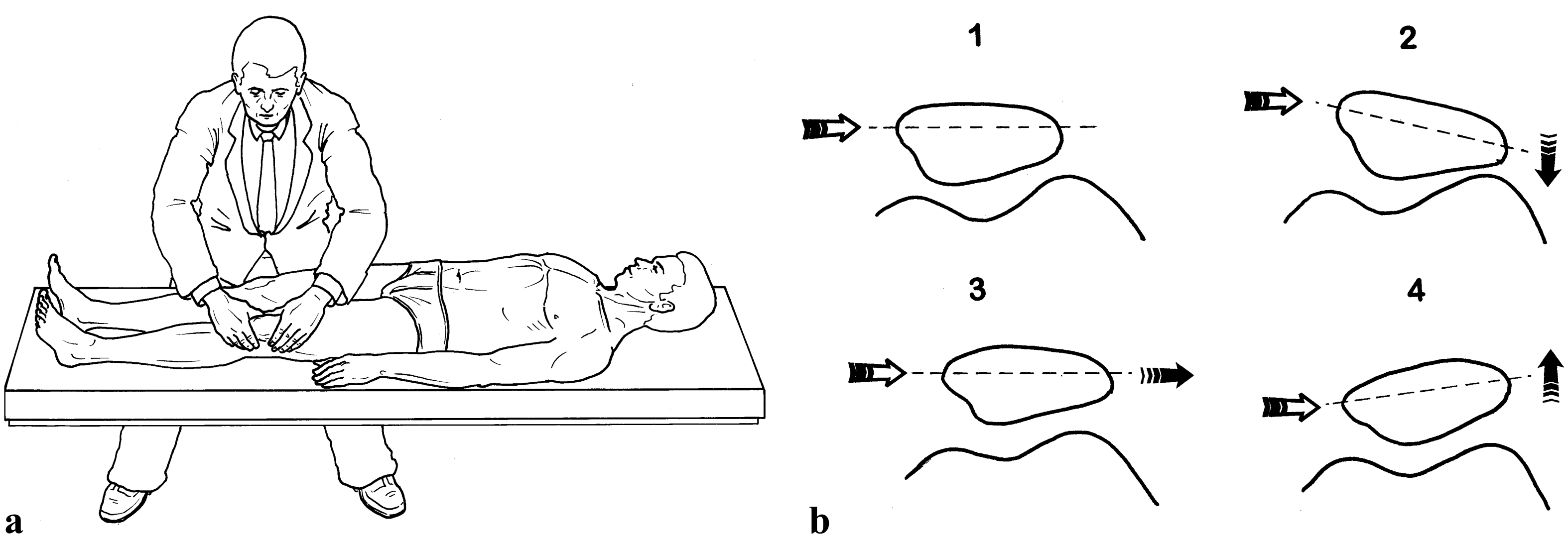


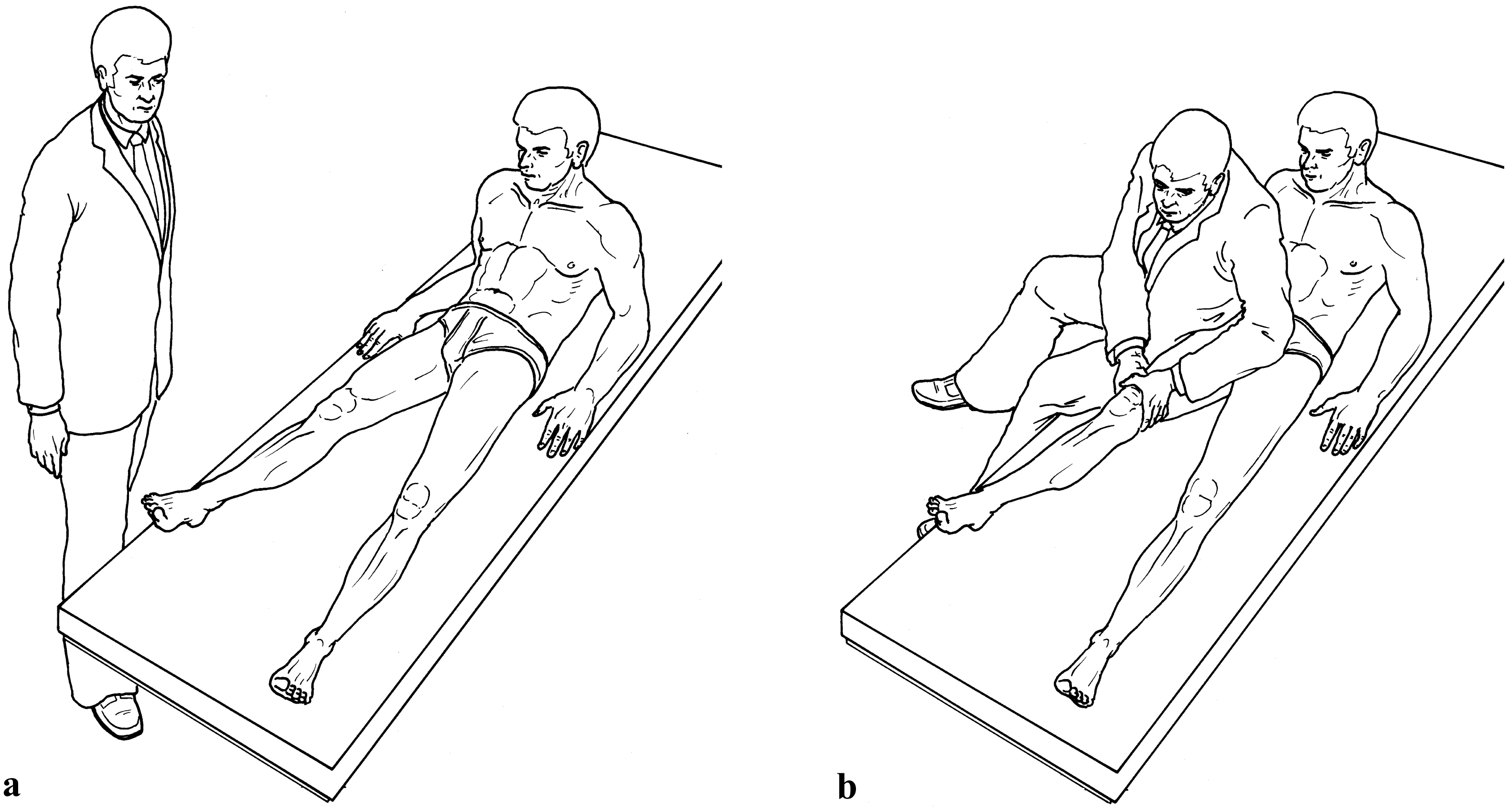
Fig. 6.16 Tilt test. **(a)** Passive lateralization of the patella. **(b)** Starting position (1), negative "abnormal" tilt test (2), neutral test (3), positive test (4).

Dreyer Test

Assesses a quadriceps tendon tear at the superior pole of the patella.

□ **Procedure:** The supine patient is asked to raise the extended leg. If the patient is unable to do so, the examiner stabilizes the quadriceps tendon proximal to the patella and has the patient lift the leg again.

□ **Assessment:** When stabilizing the tendon allows the patient to lift the leg, the examiner should suspect an avulsion of the quadriceps tendon from the patella or a chronic patellar fracture in applicable cases.



a b

Fig. 6.17 Dreyer test. **(a)** Abnormal: patient is unable to lift the leg. **(b)** With the examiner stabilizing the patella.

Mediopatellar Plica Test

□ **Procedure:** The patient lies in the supine position and the examiner flexes the affected knee to 30°.

□ **Assessment:** If the examiner then passively presses the patella medially, the patient complains of typical anteromedial pain if a plica syndrome is present. This pain, indicating a positive test, is caused by pinching of the edge of the plica between the medial femoral condyle and the patellar facet.

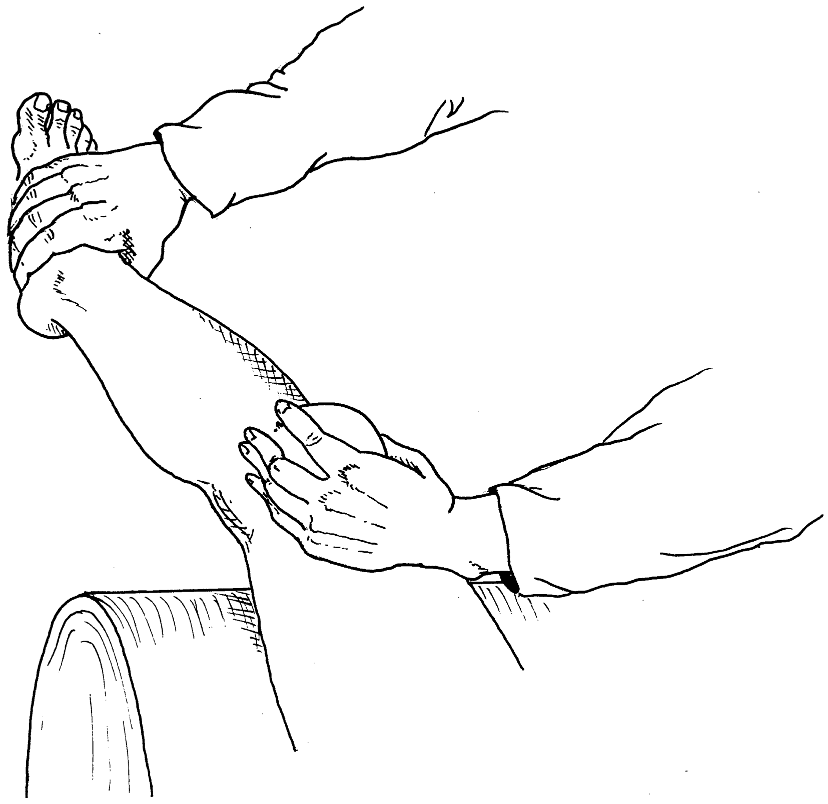


Fig. 6.18 Mediopatellar plica test.

Hughston Plica Test

□ **Procedure:** The patient lies in the supine position and the examiner grasps around the knee with one hand from an anterolateral position and presses the patella medially with the heel of the hand while palpating the medial femoral condyle with the fingers of the same hand. The examiner grasps the patient's heel with the other hand, internally rotates the lower leg and then repeatedly flexes and extends the knee.

□ **Assessment:** A painful audible or palpable "popping" suggests a mediopatellar plica syndrome.

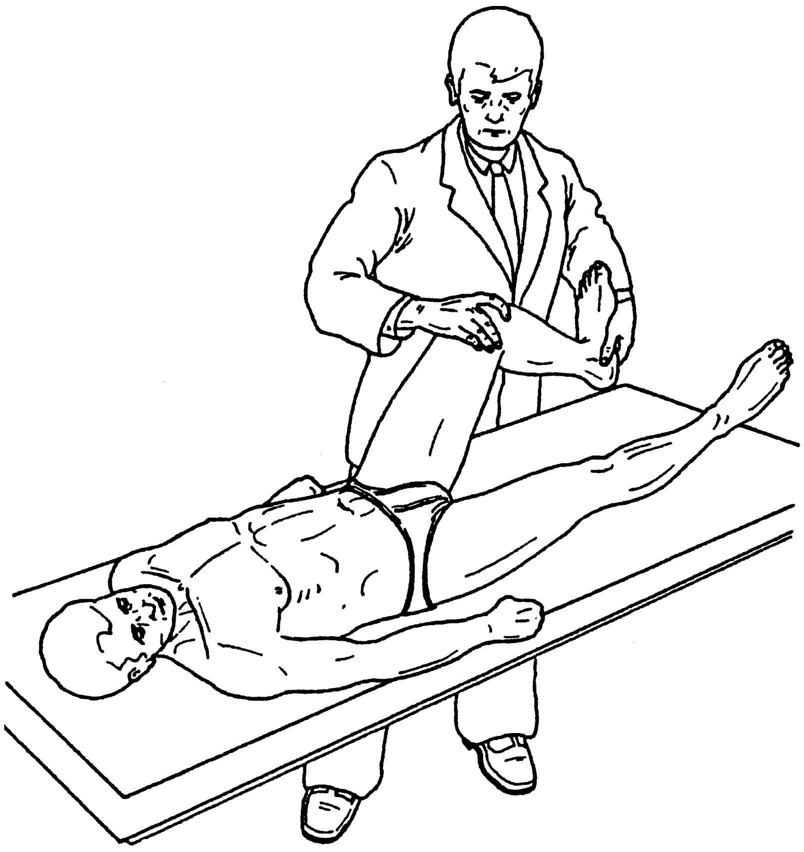


Fig. 6.19 Hughston plica test.

Meniscus

The menisci are important in guiding motion and ensuring stability in the knee. They also transmit and distribute compressive stresses between the femur and tibia. Meniscus injuries include tears or avulsions of the cartilage disks. Anatomical factors predispose the medial meniscus to a far higher incidence of injury than the lateral meniscus.

Meniscus lesions can be degenerative or traumatic in origin. Degenerative meniscus conditions usually first manifest themselves as increasing pain with exercise. Usually a minor injury will suffice to produce a tear in a weakened meniscus. In diagnosing knee injuries, one must always be alert to the possibility of a combined injury involving the collateral and cruciate ligaments in addition to the meniscus injury. Any insufficiently treated ligament injury with instability of the knee can also lead to meniscus damage. The primary symptoms of late sequelae of meniscus injuries include pain with exercise accompanied by occasional impingement symptoms and joint effusions with irritation.

There are a number of diagnostic signs of meniscus damage (see **Table 6.2**).

The function tests are based on pain provocation as a result of compression, traction, or shear forces acting on the meniscus.

An isolated function test will rarely be sufficient to evaluate a meniscus lesion. Usually a combination of various maneuvers is required to confirm the diagnosis.

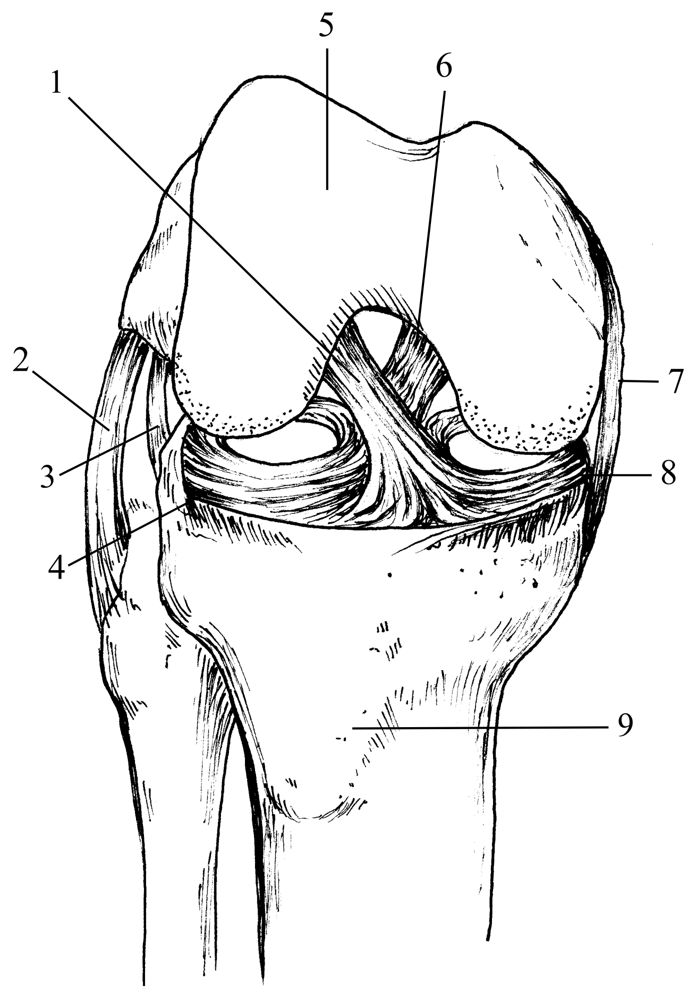


Fig. 6.20 Anterior view of the flexed knee.
1 Anterior cruciate ligament.
2 Lateral collateral ligament.
3 Popliteal ligament.
4 Lateral meniscus.
5 Patellar surface (femoral trochlea).
6 Posterior cruciate ligament.
7 Medial collateral ligament.
8 Medial meniscus.
9 Tibial tuberosity.

Table 6.2 Symptoms and signs of a meniscal lesion

Pain along the joint space
Positive meniscus tests
Loss of flexion and extension
Swelling
Crepitation

Apley Distraction and Compression Test (Grinding Test)

- **Procedure:** The patient lies prone with the affected knee flexed 90°. The examiner immobilizes the patient's thigh with his or her knee. In this position, the examiner rotates the patient's knee while alternately applying axial traction and compression to the lower leg.
- **Assessment:** Pain in the flexed knee occurring during rotation of the lower leg with traction applied suggests injury to the capsular ligaments (positive distraction test). Pain with compression applied suggests a meniscus lesion (positive grinding test).

Snapping phenomena can occur with discoid menisci or meniscal cysts. Pain in internal rotation suggests injury to the lateral meniscus or lateral capsule and/or ligaments; pain in external rotation suggests injury to the medial meniscus or medial capsule and/or ligaments.

The sign cannot be elicited where the capsular ligaments are tight, nor is this possible in an injury to the posterior horn of the lateral meniscus.

Wirth describes a modification of the grinding test (compression test), in which the knee is extended with the lower leg in fixed rotation. Wirth was able to confirm the presence of a meniscus lesion in over 85% of all cases with this modified Apley test.

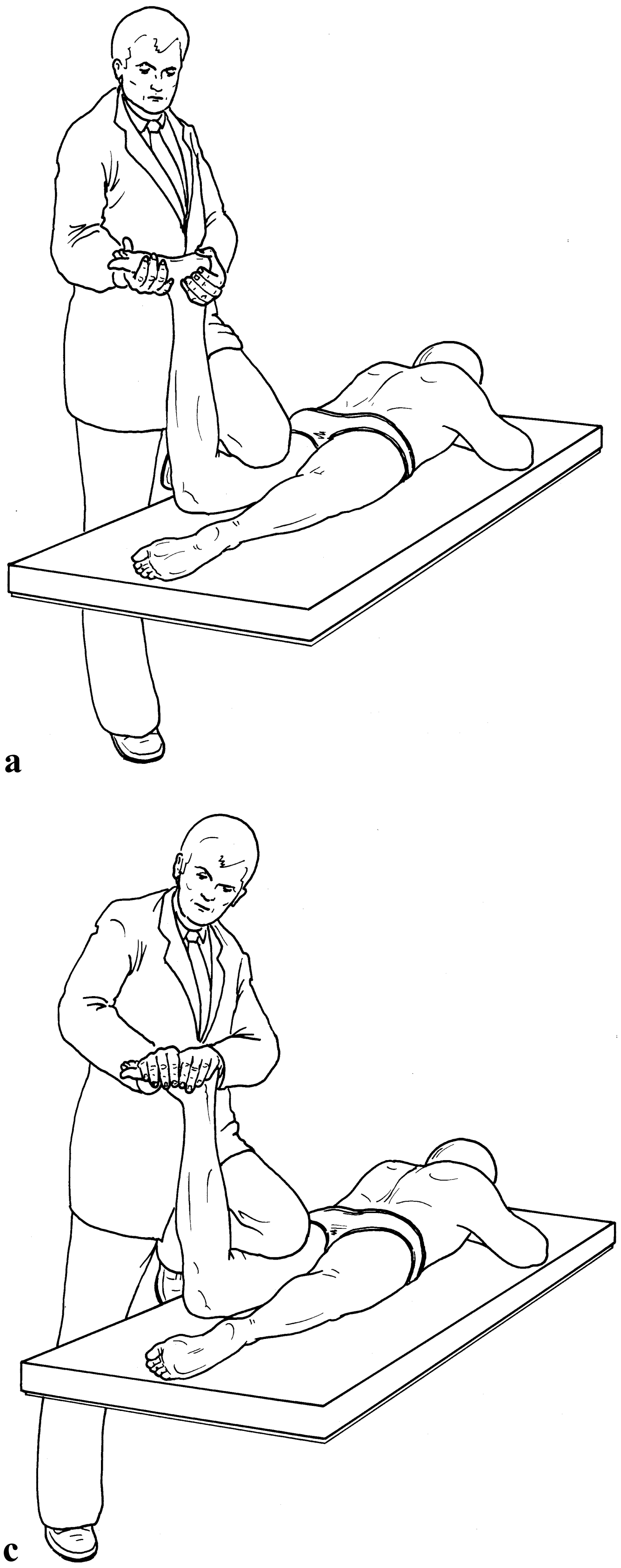


Fig. 6.21 Apley distraction and compression test. **(a)** Distraction and external rotation. **(b)** Distraction and internal rotation. **(c)** Compression and external rotation. **(d)** Compression and internal rotation.

Thessaly Test

□ **Procedure:** The patient first stands barefoot on the healthy leg, supported by the examiner, who holds the patient's outstretched hands. With the knee flexed 5°, the patient is asked to actively rotate the knee and the whole body externally and internally three times around the fixed foot. The procedure is repeated again with 20° of flexion.

The test is then carried out standing on the affected leg.

□ **Assessment:** Pain along the joint space suggests a lesion of the meniscus on the ipsilateral side. "Snapping" or "locking" of the meniscus can also occur and are also signs of meniscal injury. The test has a high degree of diagnostic accuracy when carried out at 20° of flexion.

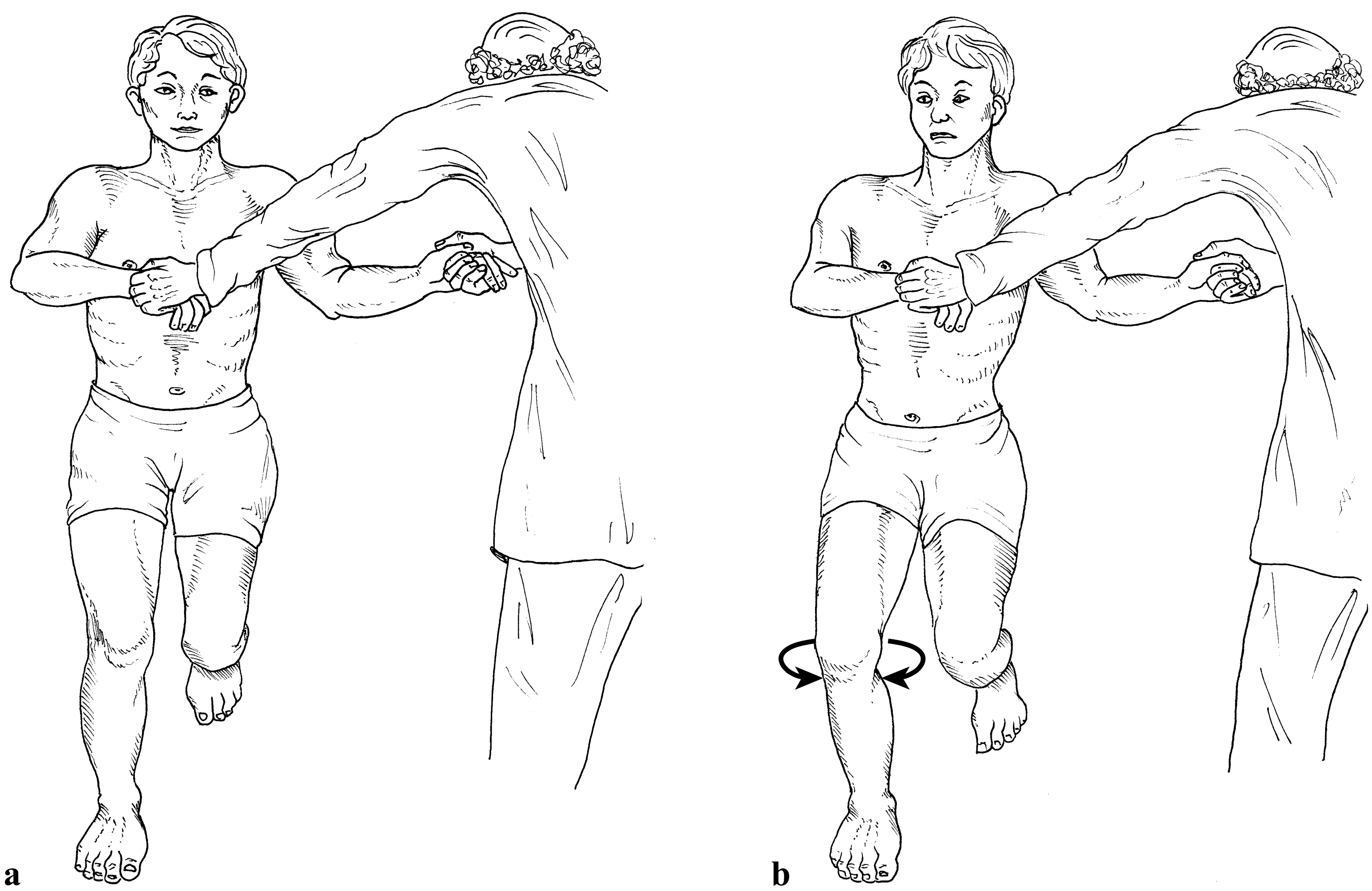


Fig. 6.22 Thessaly test. **(a)** Frontal view in the neutral position. **(b)** Internal and external rotation with 20° of knee flexion.

McMurray Test / Fouche Sign (reversed McMurray Test)

□ **Procedure:** The patient is supine with the knee and hip of the affected leg in maximum flexion. The examiner grasps the patient's knee with one hand and the patient's foot with the other. Holding the patient's lower leg in maximum external (McMurray test) or internal (Fouche sign) rotation, the examiner then passively extends the knee into 90° of flexion.

□ **Assessment:** Pain while extending the knee with the lower leg externally rotated and abducted suggests a medial meniscus lesion; pain in internal rotation suggests an injury to the lateral meniscus. A snapping sound in extreme flexion occurs when a projecting meniscal flap becomes impinged on the posterior horn. Snapping in 90° of flexion suggests an injury in the middle section of the meniscus.

The snapping symptoms can be increased by moving the entire lower leg in a circle (modified McMurray test).

□ **Note:** Continuing the extension as far as the neutral (0°) position corresponds to the Bragard test. This test, when performed by slowly extending the knee with the lower leg in external rotation to test the medial meniscus, is also described as the Fouche sign. The McMurray test is positive in 30% of all children with normal knees. Approximately 1% of the normal population should test positive.

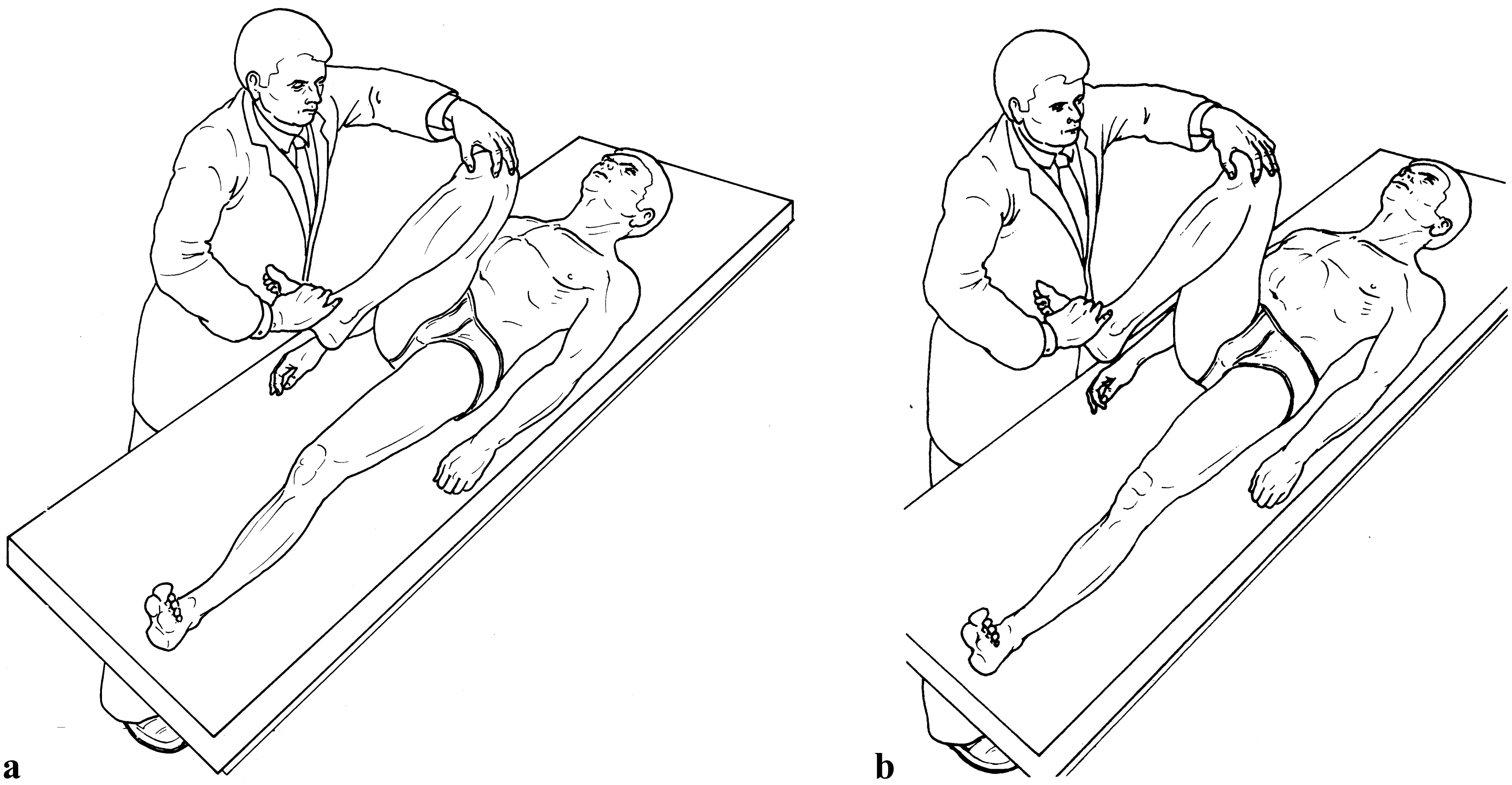


Fig. 6.23 McMurray test. **(a)** In maximum flexion. **(b)** In 90° of flexion.

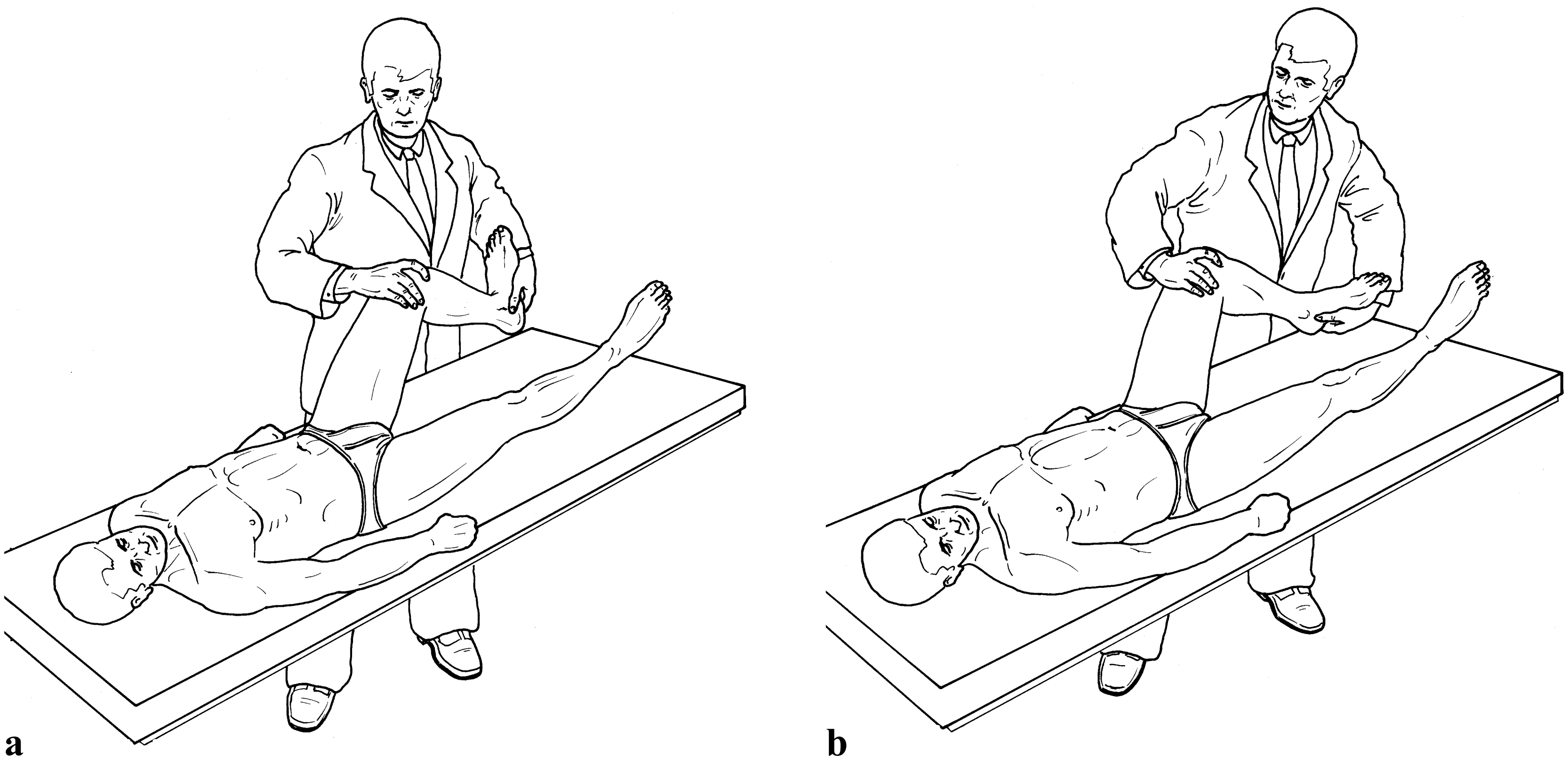
Bragard Test

□ **Procedure:** The patient is supine. With one hand, the examiner grasps the patient's 90°-flexed knee and palpates the lateral and medial joint cavity with the thumb and index finger. With the other hand, the examiner grasps the patient's foot and rotates the patient's lower leg.

□ **Assessment:** Pain felt over the joint cavity indicates a meniscus lesion. In an injury to the medial meniscus, external rotation and extension from a flexed position increases the pain in the medial joint cavity.

With internal rotation and increasing flexion in the knee, the meniscus migrates back into the interior of the joint and is no longer accessible to the examiner's palpating finger. This reduces pain.

When a lateral meniscus lesion is suspected, the examiner palpates the lateral meniscus. This is done while first extending and internally rotating the knee from a position of maximum flexion. Pain in the lateral joint space is a sign of lateral meniscus injury. In external rotation and increasing flexion, the meniscus shifts back into the interior of the joint and is no longer palpable with the finger. This maneuver reduces pain. The diagnosis is more certain if the tenderness to palpation migrates with joint motions. The lateral meniscus, and with it the tenderness to palpation, migrates posteriorly as the knee is internally rotated.



a b

Fig. 6.24 Bragard test. **(a)** Flexion–extension–external rotation (medial meniscal injury). **(b)** Flexion–extension–internal rotation (lateral meniscal injury).

Payr Sign

□ **Procedure:** The patient is seated cross-legged. The examiner exerts intermittent pressure on the affected leg, which is flexed and externally rotated.

□ **Assessment:** Pain in the medial joint cavity suggests meniscus damage (usually a lesion of the posterior horn). Occasionally, patients themselves will be able to provoke snapping. Moving the knee back and forth causes the injured portion of the meniscus to be drawn into the joint and then spring back out with a snap when the joint cavity is distended.

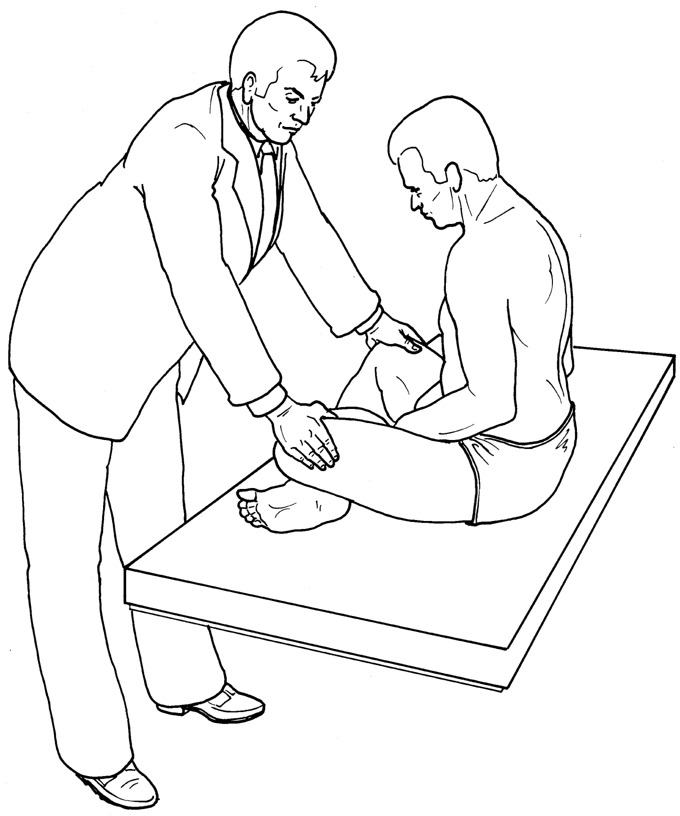
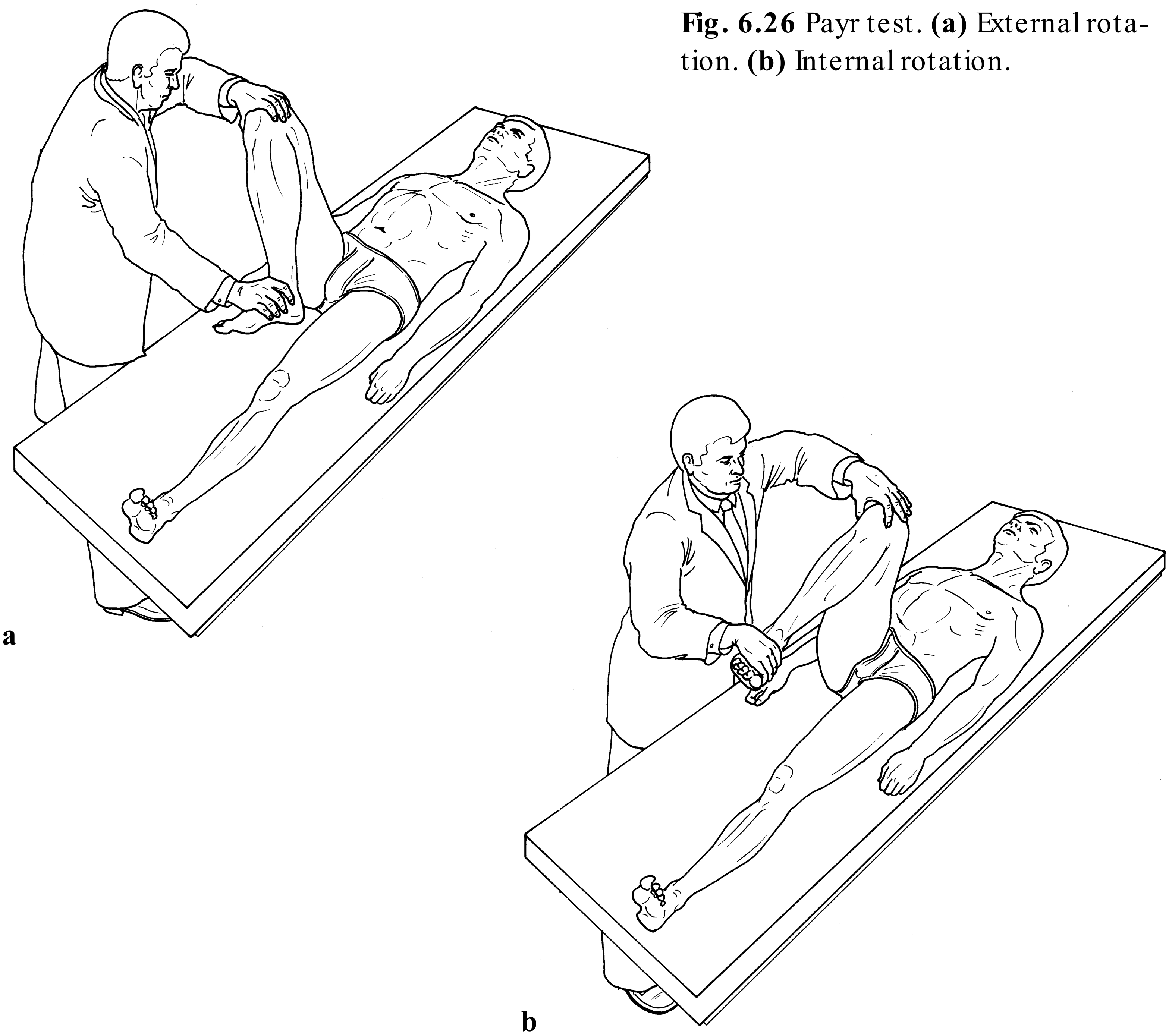


Fig. 6.25 Payr sign.

Payr Test

□ **Procedure:** The patient is supine. The examiner immobilizes the patient's knee with one hand and palpates the lateral and medial joint cavity with the thumb and index finger, respectively. With the other hand, the examiner grasps the patient's ankle. With the knee maximally flexed, the lower leg is externally rotated as far as possible. Then with the knee in slight adduction (varus stress), the leg is flexed further in the direction of the contralateral hip.

□ **Assessment:** Pain in the posterior medial joint cavity suggests damage to the medial meniscus (most often the posterior horn is involved, which is compressed by this maneuver). The posterior horn of the lateral meniscus can be similarly examined with the knee internally rotated and abducted (valgus stress).



a

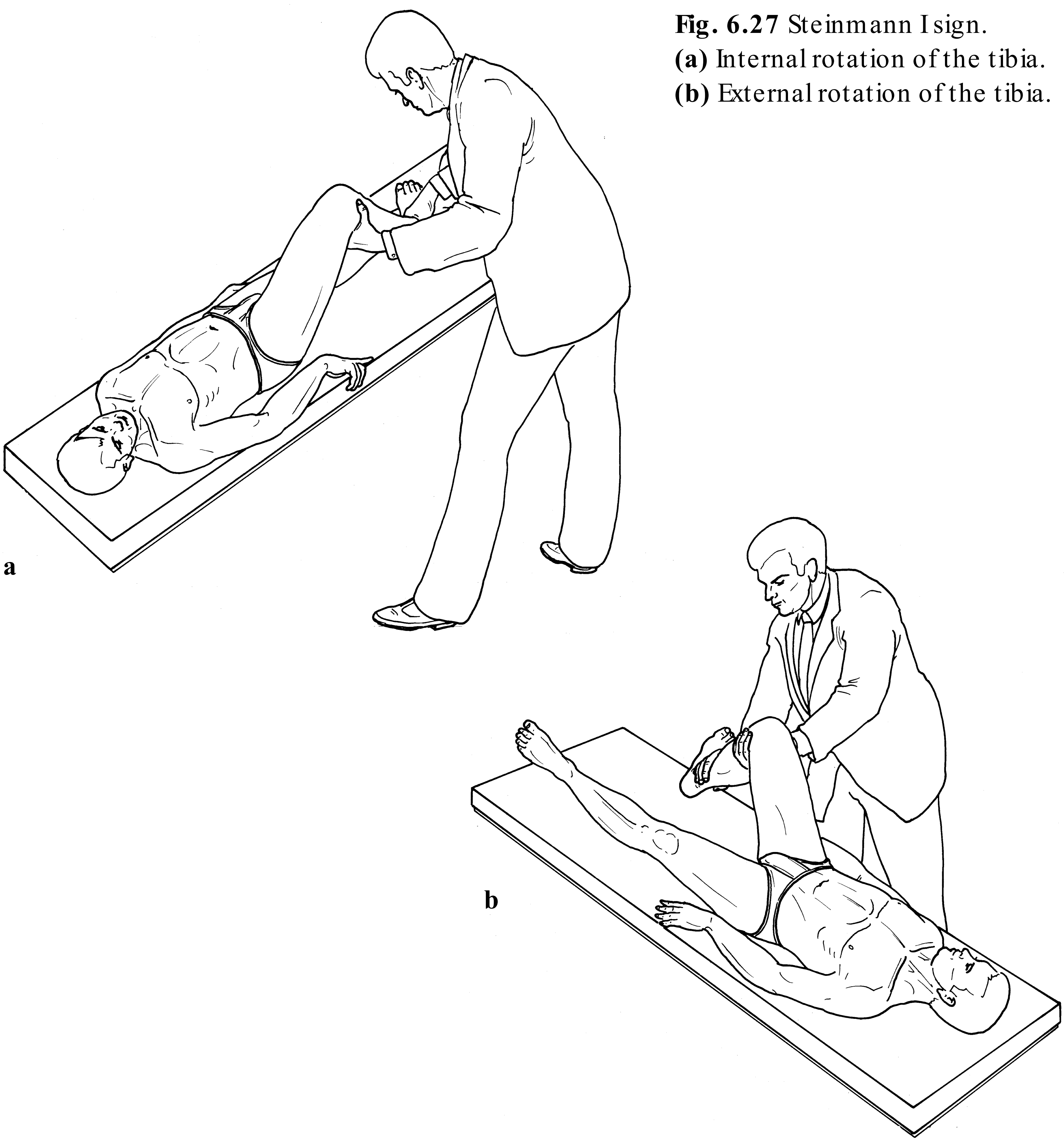
b

Fig. 6.26 Payr test. **(a)** External rotation. **(b)** Internal rotation.

Steinmann I Sign

□ **Procedure:** The patient is supine. The examiner immobilizes the patient's flexed knee with one hand and grasps the lower leg with the other hand. The examiner then forcefully rotates the lower leg in various degrees of knee flexion.

□ **Assessment:** Pain in the medial joint cavity in forced external rotation suggests damage to the medial meniscus; pain in the lateral joint cavity in internal rotation suggests damage to the lateral meniscus. Because the localization of the tear can vary, the test for the Steinmann I sign should be performed with the knee in varying degrees of flexion.



a

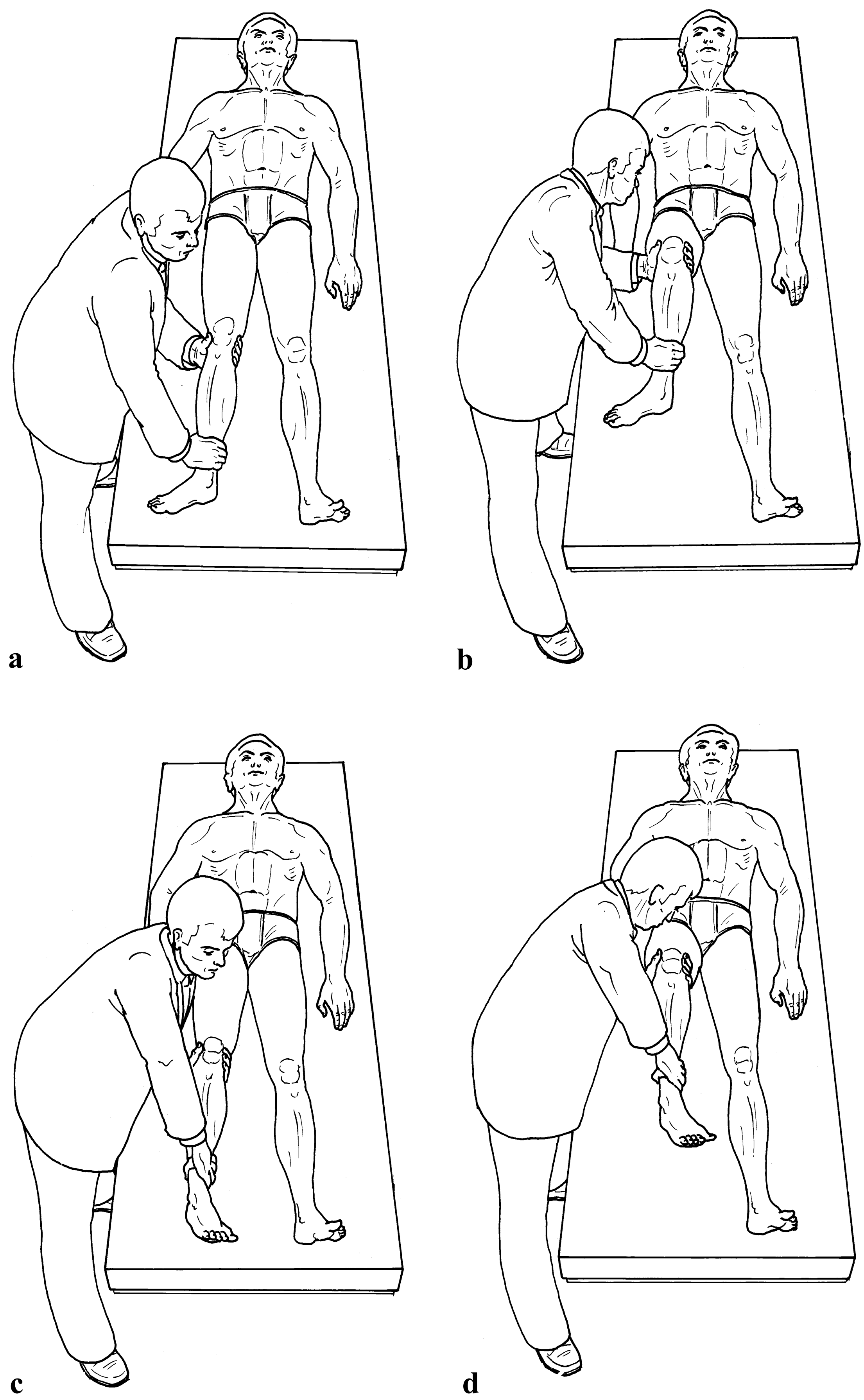
b

Fig. 6.27 Steinmann I sign.
(a) Internal rotation of the tibia.
(b) External rotation of the tibia.

Steinmann II Sign

□ **Procedure:** The patient is supine. The examiner grasps the knee with one hand and palpates the joint cavity. With the other hand, the examiner grasps the lower leg just proximal to the mortise of the ankle. With the patient's thigh immobilized, the examiner places the lower leg first in external rotation, then in internal rotation, in each case alternately flexing and extending the lower leg while applying slight axial compression.

Fig. 6.28 Steinmann II sign. **(a)** Starting position with the lower leg externally rotated. **(b)** Flexion. **(c)** Starting position with the lower leg internally rotated. **(d)** Flexion.



□ **Assessment:** Pain in the medial or lateral joint cavity suggests a meniscus injury. The tenderness to palpation in the joint cavity migrates medially and posteriorly during flexion and slight external rotation of the knee; it then migrates proportionally to the pressure and shear forces placed on the meniscus, posteriorly during knee flexion, and back anteriorly as the knee is extended.
□ **Note:** Although this test can also be used for an injury to the lateral meniscus, its primary purpose is to help evaluate medial meniscus lesions. A differential diagnosis must consider osteoarthritis and lesions of the medial collateral and capsular ligaments.

Böhler–Krömer Test

□ **Procedure:** The patient is supine. The examiner stabilizes the femur from the lateral side with one hand and grasps the medial malleolus with the other. With the lower leg abducted (valgus stress applied), the examiner then passively flexes and extends the knee.

With his or her hands on the patient's lateral malleolus and medial thigh, the examiner grasps the leg and flexes and extends the knee with the lower leg adducted (varus stress applied).

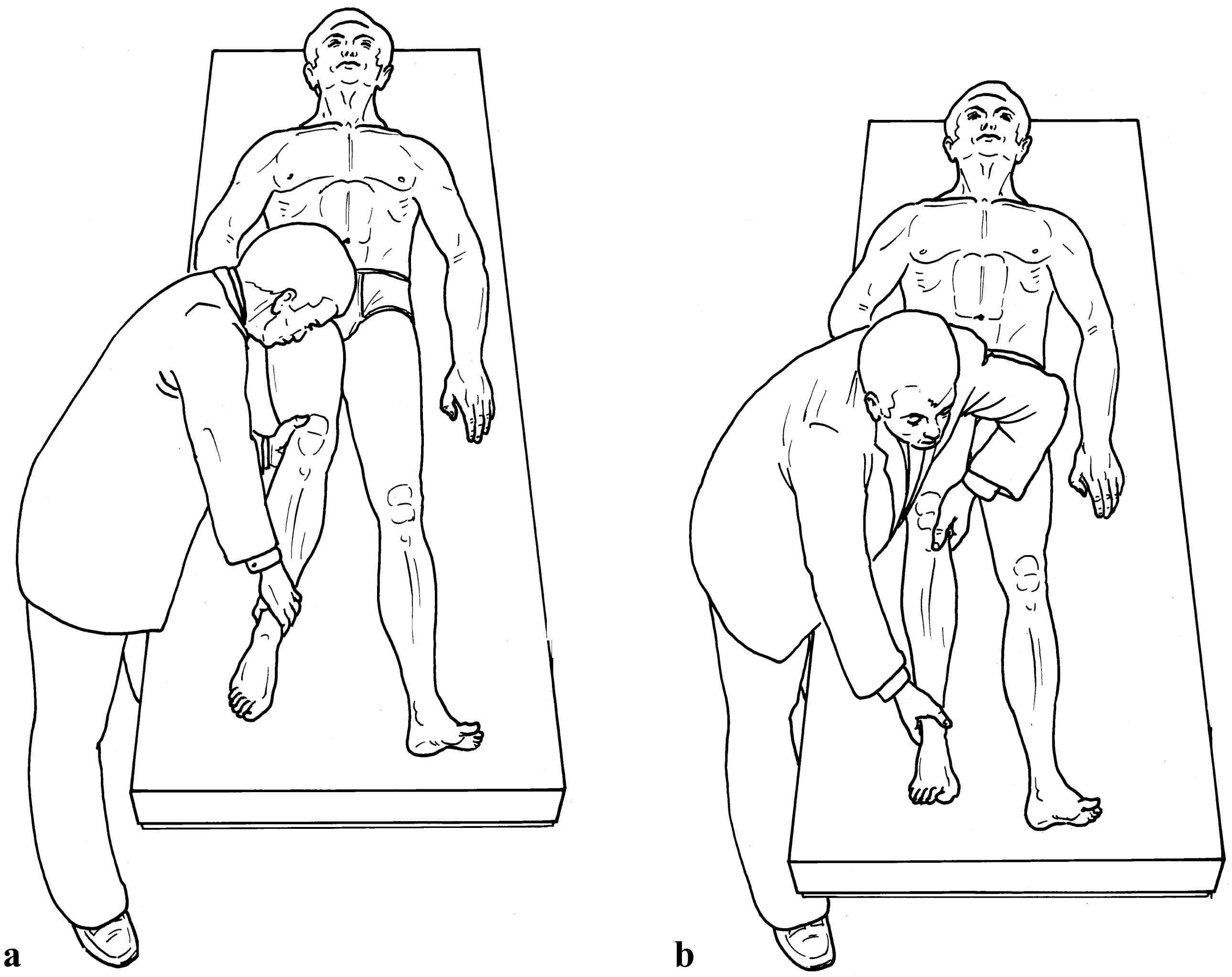


Fig. 6.29 Böhler–Krömer test. **(a)** Lower leg abducted (valgus). **(b)** Lower leg adducted (varus).

Assessment: Flexing and extending the knee with the lower leg alternately adducted and abducted (the Krömer test) alternately increases compression of the medial meniscus and lateral meniscus. Opening the joint cavity compresses the opposite meniscus. Opening the medial cavity creates a valgus stress for testing the lateral meniscus; opening the lateral cavity creates a varus stress for testing the medial meniscus.

Note: The Böhler meniscus tests in the coronal plane (with the knee extended) allow simultaneous assessment of the ligaments of the knee in the side opposite the motion.

Merke Test

Procedure: The patient bears weight on the affected leg with the knee slightly flexed. The examiner immobilizes the foot of the affected leg.

The examiner lifts the patient's contralateral leg slightly and asks the patient to internally and externally rotate the thigh of the affected leg.

The lower leg is rotated as in the Steinmann I sign.

Assessment: Because of the increased axial compression due to the weight of the body, the Merke test usually elicits more severe pain. Pain in the medial compartment during external rotation of the lower leg at the knee suggests a medial meniscal lesion, while pain in the lateral compartment during internal rotation of the lower leg at the knee suggests a lateral meniscal lesion.

The Merke test is occasionally positive in the presence of collateral ligament lesions.

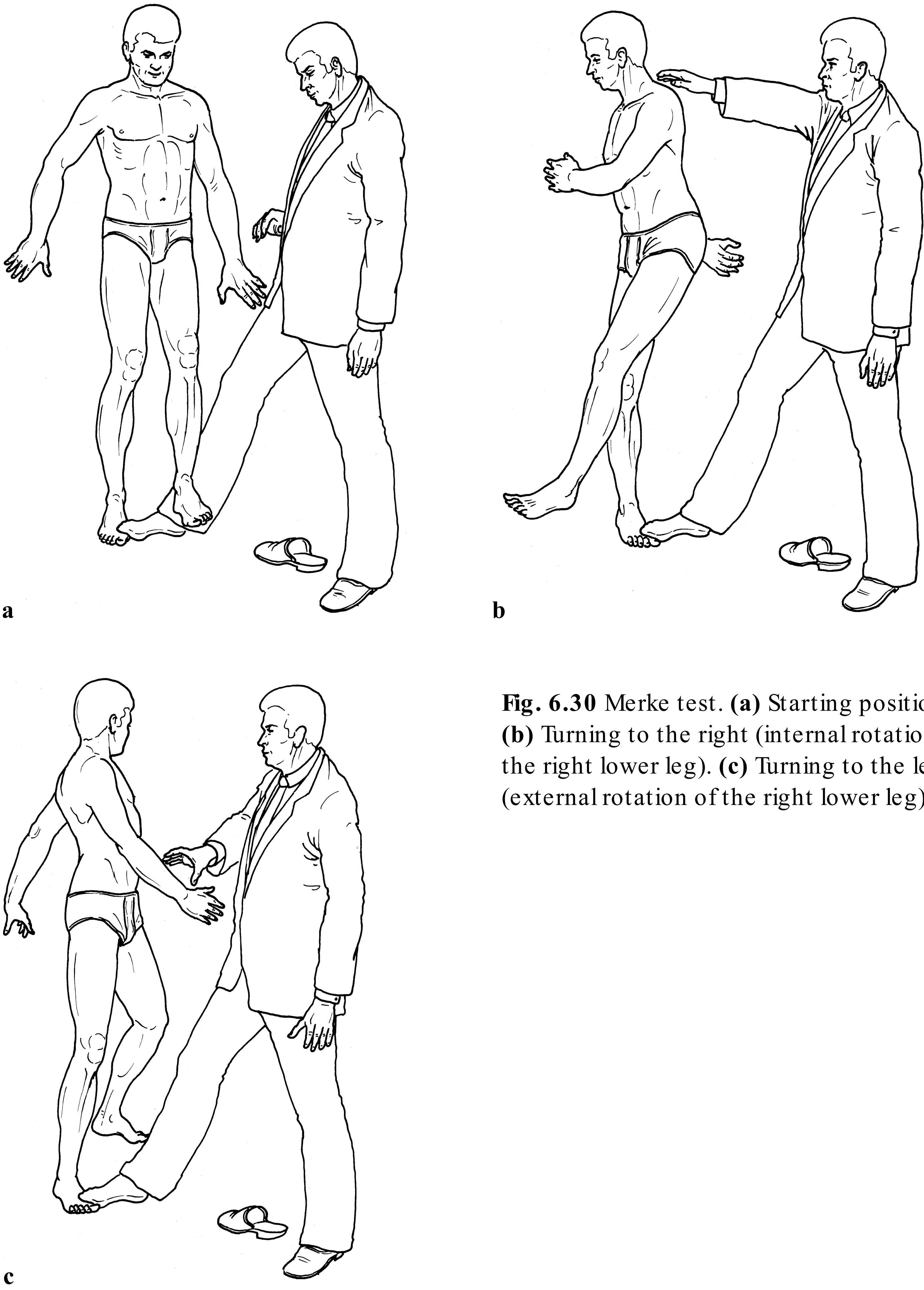


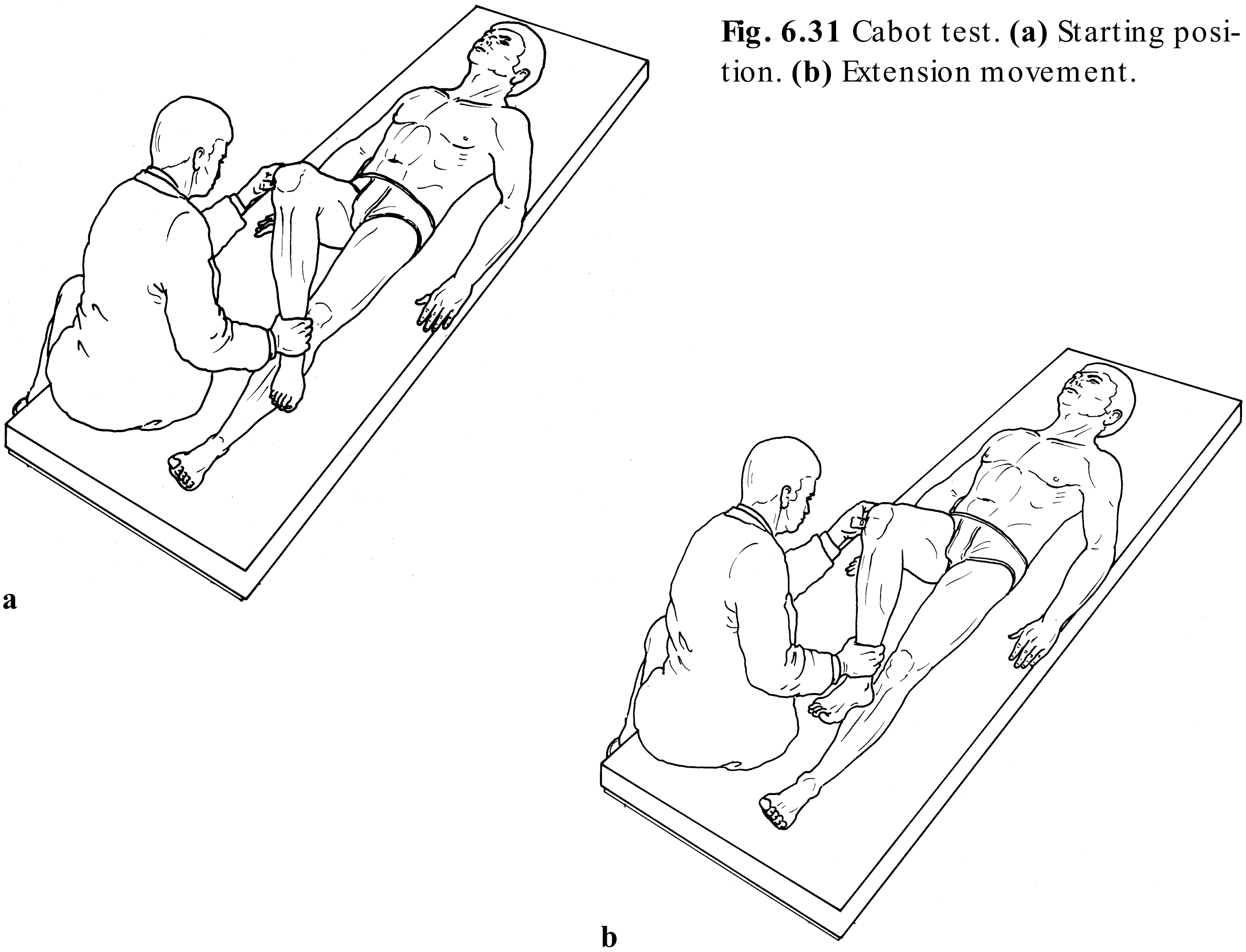
Fig. 6.30 Merke test. **(a)** Starting position. **(b)** Turning to the right (internal rotation of the right lower leg). **(c)** Turning to the left (external rotation of the right lower leg).

Cabot Test

□ **Procedure:** The patient is supine with the affected leg flexed at the knee and placed over the proximal portion of the contralateral lower leg. With one hand, the examiner grasps the patient's knee and palpates the lateral joint cavity with the thumb. With the other hand, the examiner grasps the patient's lower leg slightly proximal to the subtalar joint. The patient is then asked to extend the knee against the resistance of the examiner's hand.

□ **Assessment:** Pain will occur where there is a lesion of the posterior horn of the lateral meniscus. Depending on the severity of the pain, the patient will often be unable to extend the knee farther. The painful point, which is palpable with the thumb, lies primarily in the lateral posterior joint cavity. Occasionally patients will report pain radiating into the popliteal fossa and calf.

□ **Note:** The Cabot test is also described in the literature as the popliteus sign.



a

b

Fig. 6.31 Cabot test. **(a)** Starting position. **(b)** Extension movement.

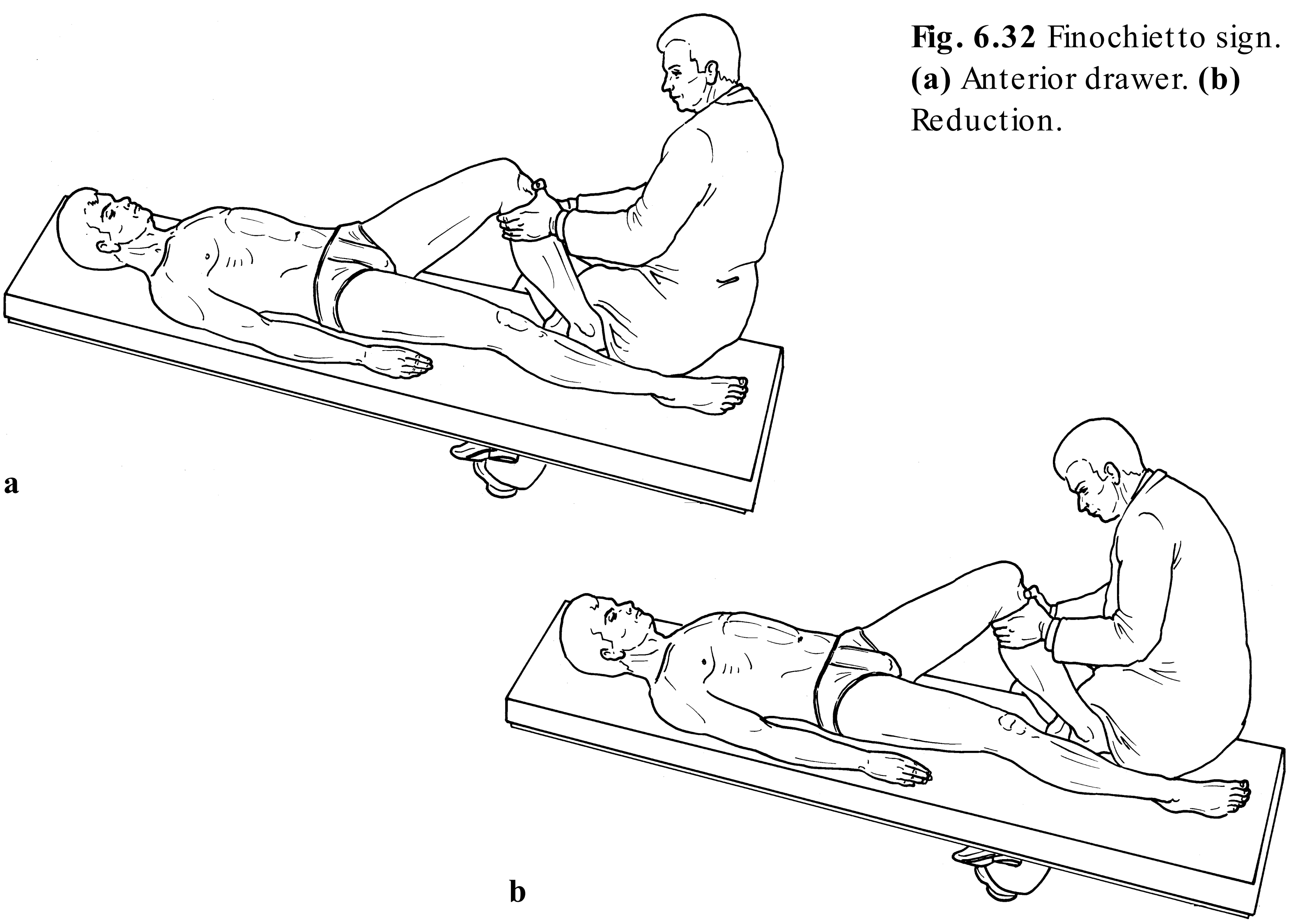
Finochietto Sign

Simultaneously tests anterior cruciate ligament and meniscus injuries.

□ **Procedure:** The patient is supine. The anterior drawer test is performed with the knee flexed 90°.

□ **Assessment:** Where the injury also involves an anterior cruciate ligament tear, the anterior drawer test with the knee flexed 90° will cause anterior displacement of the tibia. The laxity of the knee ligaments causes the femoral condyle to ride up over the posterior horn of the medial meniscus under the stress of the anterior drawer. A positive Finochietto test produces an audible snap and/or a palpable skip. If the tibia is then pressed posteriorly, the femoral condyle will glide back down from the posterior horn of the medial meniscus. Occasionally, reduction of the displaced meniscus will be necessary following a positive Finochietto test. In this case, there is reason to suspect a full posterior separation of the medial meniscus and/or a longitudinal or bucket-handle tear.

□ **Note:** In the setting of anterior cruciate ligament insufficiency, damage to the posterior horn of the medial meniscus or its capsular attachments results from derangement of the rolling and sliding mechanism secondary to a cruciate ligament tear. This produces a shear injury to the posterior horn of the medial meniscus.



a

b

Fig. 6.32 Finochietto sign. **(a)** Anterior drawer. **(b)** Reduction.

Childress Sign

□ **Procedure:** The patient assumes a squatting position, preferably with the buttocks in contact with the heels. The patient is then asked to waddle in this position.

□ **Assessment:** In the presence of an injury to the posterior horn, the patient will notice a painful snapping shortly before maximum flexion or in the early phase of extension. This is caused by impingement of the injured meniscus. Patients in severe pain will usually be unable to assume the squatting position.

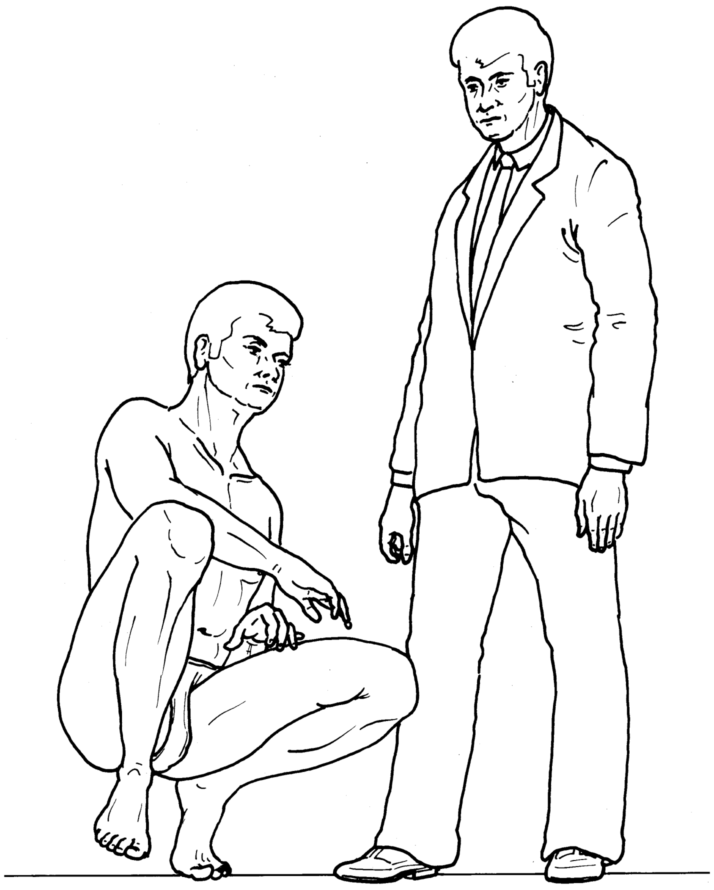


Fig. 6.33 Childress sign.

Turner Sign

In 1931, Turner described a meniscus sign caused by chronic irritation of the infrapatellar branch of the saphenous nerve. A meniscus lesion will often be accompanied by an irregular hyperesthetic area measuring approximately 4 to 5 cm. This area will be located at the level of and slightly proximal to the medial joint cavity on the medial aspect of the knee or along the course of the infrapatellar branch of the saphenous nerve. Thermal and mechanical stimuli (tapping) are used to test the area for local hypersensitivity. According to Zippel, careful examination technique will demonstrate this symptom more often than one would expect. No similar sign is known for injuries to the lateral meniscus.

Anderson Medial and Lateral Compression Test

Procedure: The patient is supine. The examiner grasps the patient's lower leg and immobilizes the foot between his or her own forearm and waist. With the free hand, the examiner palpates the anterior joint cavity. The examiner then flexes the knee to 45° while applying a valgus stress and extends it while applying a varus stress. This produces a circular movement in the knee.

Assessment: A longitudinal or flap tear in the meniscus causes pain and/or friction rub at the level of the joint cavity. Complex tears lead to chronic friction rub. However, the same symptoms can occur with osteoarthritis or secondary to resection of a meniscus. This test involves placing stresses on the knee as it approaches extension and in moderate flexion. Therefore, one can occasionally provoke subluxation as the knee approaches extension, as in a positive pivot shift test with insufficiency of the anterior cruciate ligament.

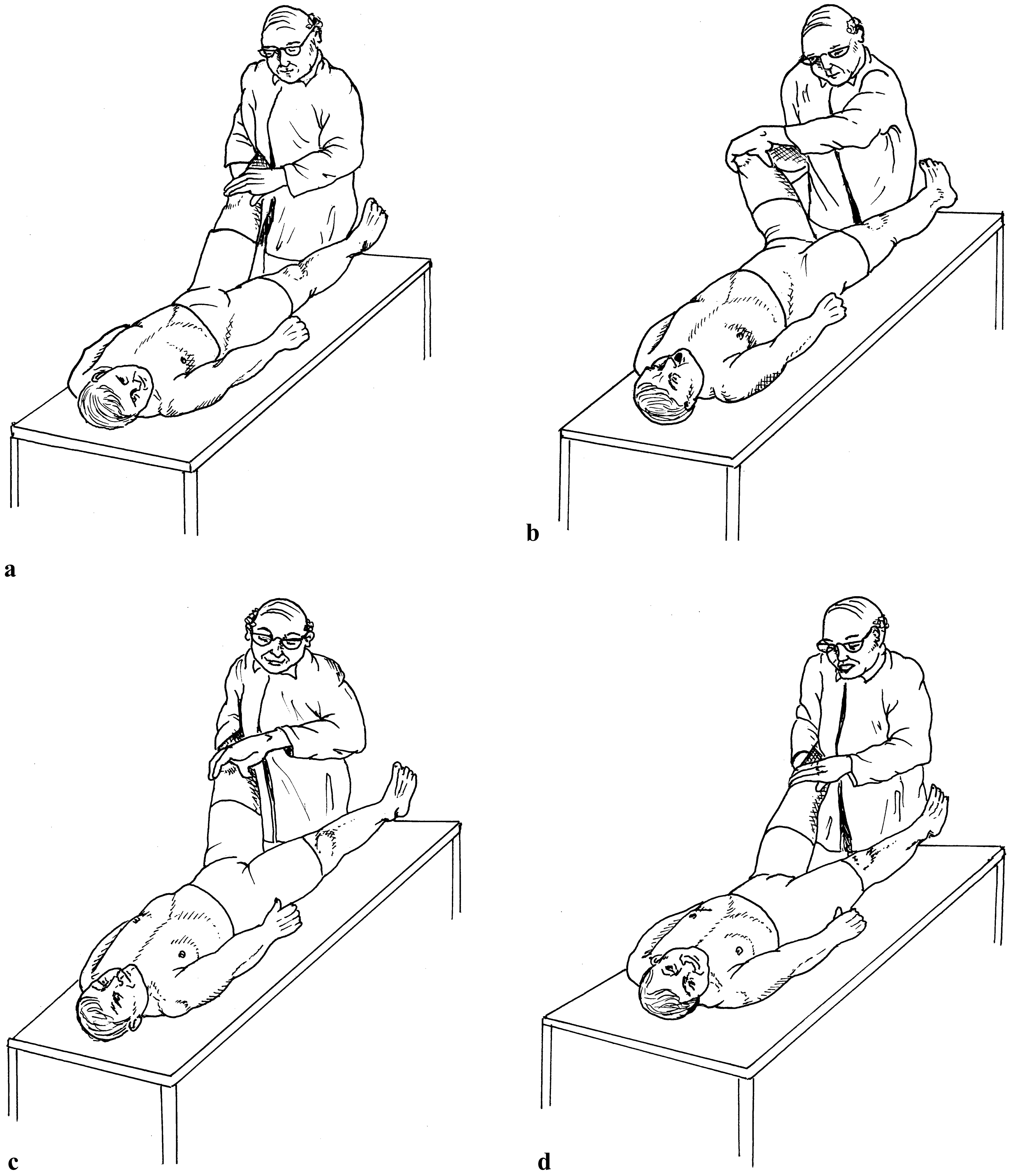


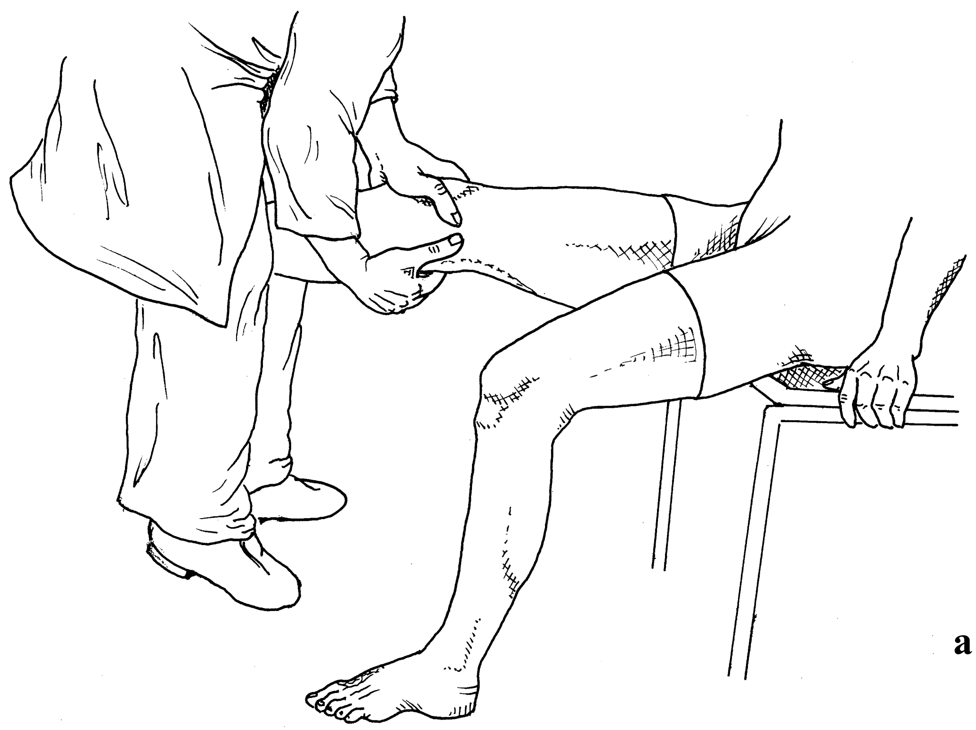
Fig. 6.34 Anderson medial and lateral compression test. **(a)** Starting position. **(b)** Valgus stress during flexion of the knee to 45°. **(c)** Extension of the 45° flexed knee. **(d)** Varus stress during extension of the knee.

Pässler Rotational Compression Test

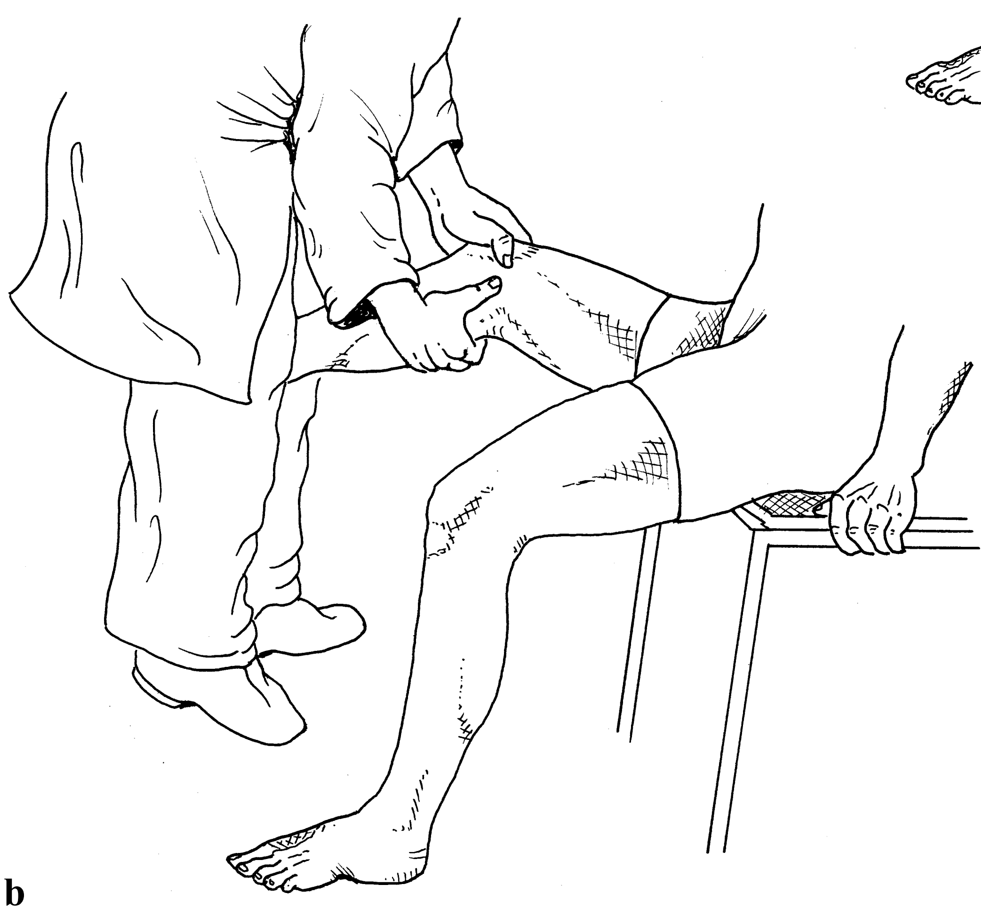
■ **Procedure:** The patient is seated. The examiner immobilizes the foot of the leg to be examined, holding it between his or her own legs slightly proximal to the knees. To evaluate the medial meniscus, the examiner rests both thumbs on the medial joint cavity and moves the patient's knee in a circle in the form of external and internal rotational movements. This causes the knee to move through various degrees of flexion. At the same time, the examiner applies a varus or valgus stress, respectively.

■ **Assessment:** The test is positive when the patient reports pain with the circular motion. It is considered strongly positive when pain can be elicited by the circular motion alone in either the medial joint cavity (suspected medial meniscus lesion) or the lateral joint cavity (suspected lateral meniscus lesion).

Fig. 6.35 Pässler rotational compression test. **(a)** Starting position with examiner's thumbs on medial joint cavity. **(b)** Circular motion of the knee.



a



b

Tschaklin Sign

Quadriceps atrophy is often encountered in chronic meniscus lesions. Atrophy of the vastus medialis in medial meniscus lesions is often associated with compensatory increase in muscle tone in the sartorius, which is known as the Tschaklin sign.

Wilson Test

Indicates osteochondritis dissecans.

- **Procedure:** With the patient in a supine position, the examiner grasps the knee above the patella with one hand and simultaneously palpates the area around the medial joint space. He or she passively flexes the patient's knee to 90°. Then he or she asks the patient to actively extend the knee gradually while maintaining the tibia in internal rotation and to let the examiner know if pain occurs. From this position, the patient is instructed to externally rotate the tibia.
- **Assessment:** In osteochondritis dissecans, joint pain occurs between 20 and 30° from the pressure on the joint and from the palpating fingers. The pain is typically reduced by external rotation of the lower leg. The test is positive if pain experienced during extension with internal tibial rotation is relieved by externally rotating the tibia. The classic site for osteochondritis dissecans and

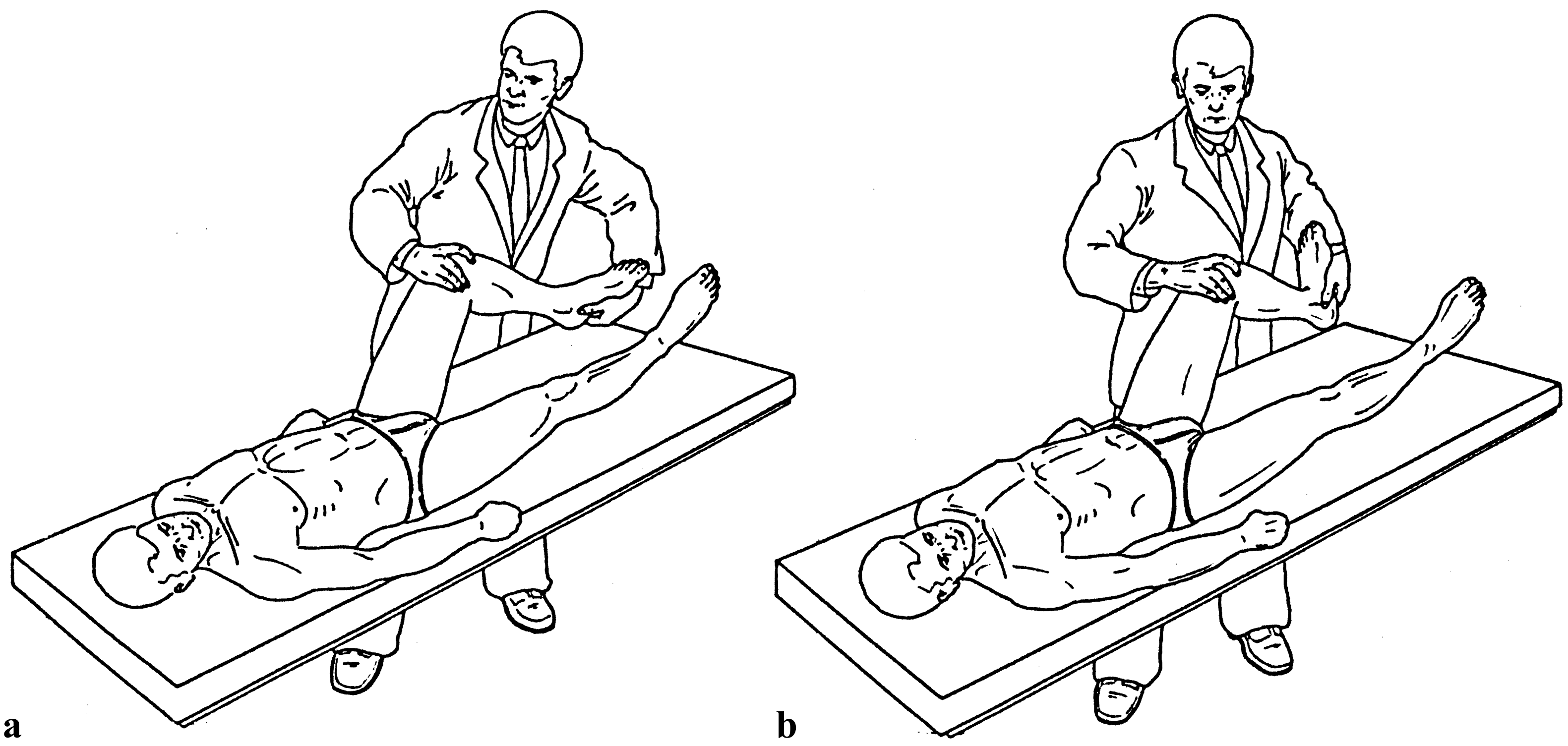


Fig. 6.36 Wilson test. **(a)** Extension in internal rotation. **(b)** External rotation.

the pain that radiates from it is on the medial femoral condyle near the intercondylar fossa.

□ **Note:** Osteochondritis dissecans is an aseptic necrosis that arises in the subchondral bone of the articular surfaces and disrupts the overlying cartilage. In its advanced stages, separation of parts of articular cartilage and underlying bone can occur, creating intra-articular loose bodies (joint mice). Osteochondritis dissecans should always be considered in adolescents presenting with joint effusion and knee pain.

Knee Ligament Stability Tests

The knee is stabilized by the ligaments, menisci, the shape and congruency of the articular surfaces, and the musculature. The ligaments ensure functional congruency by guiding the femur and tibia and limiting the space between them. Ligament injuries lead to functional impairment of the knee with instability. Knee ligament stability tests can help to identify and differentiate these instabilities.

Abnormal directions of motion can be divided into three categories:

1. Direct instability in a single plane.
2. Rotational instability.
3. Combined rotational instability.

Clinical instability is divided into three gradations. Estimated joint opening or drawer of up to 5 mm is defined as 1+ (or +), 5 to 10 mm as 2+ (++), and over 10 mm as 3+ (or +++).

Abduction and Adduction Test (Valgus and Varus Stress Test)

Assesses medial and lateral knee stability.

□ **Procedure:** The patient is supine. The examiner grasps the patient's knee at the tibial head with both hands while palpating the joint cavity. The examiner immobilizes the patient's distal lower leg between his or her own forearm and waist while applying a valgus and varus stress to the knee. The fingers resting on the joint cavity can palpate any opening of the joint.

□ **Assessment:** Lateral stability is assessed in 20° of flexion and in full extension. Full extension prevents lateral opening as long as the posterior capsule and posterior cruciate ligament are intact, even if the medial collateral ligament is torn. In 20° of flexion, the posterior capsule is relaxed. Applying a valgus stress in this position evaluates the medial collateral ligament alone as the primary stabilizer. This allows the examiner to identify the nature of damage to the posteromedial capsular ligaments.

The opposite applies to adduction (varus) stress. In 20° of flexion, the primary lateral stabilizer is the lateral collateral ligament. The anterior cruciate ligament and popliteus tendon act as secondary stabilizers.

When testing lateral stability, the examiner assesses the degree of joint opening and the quality of the end point.

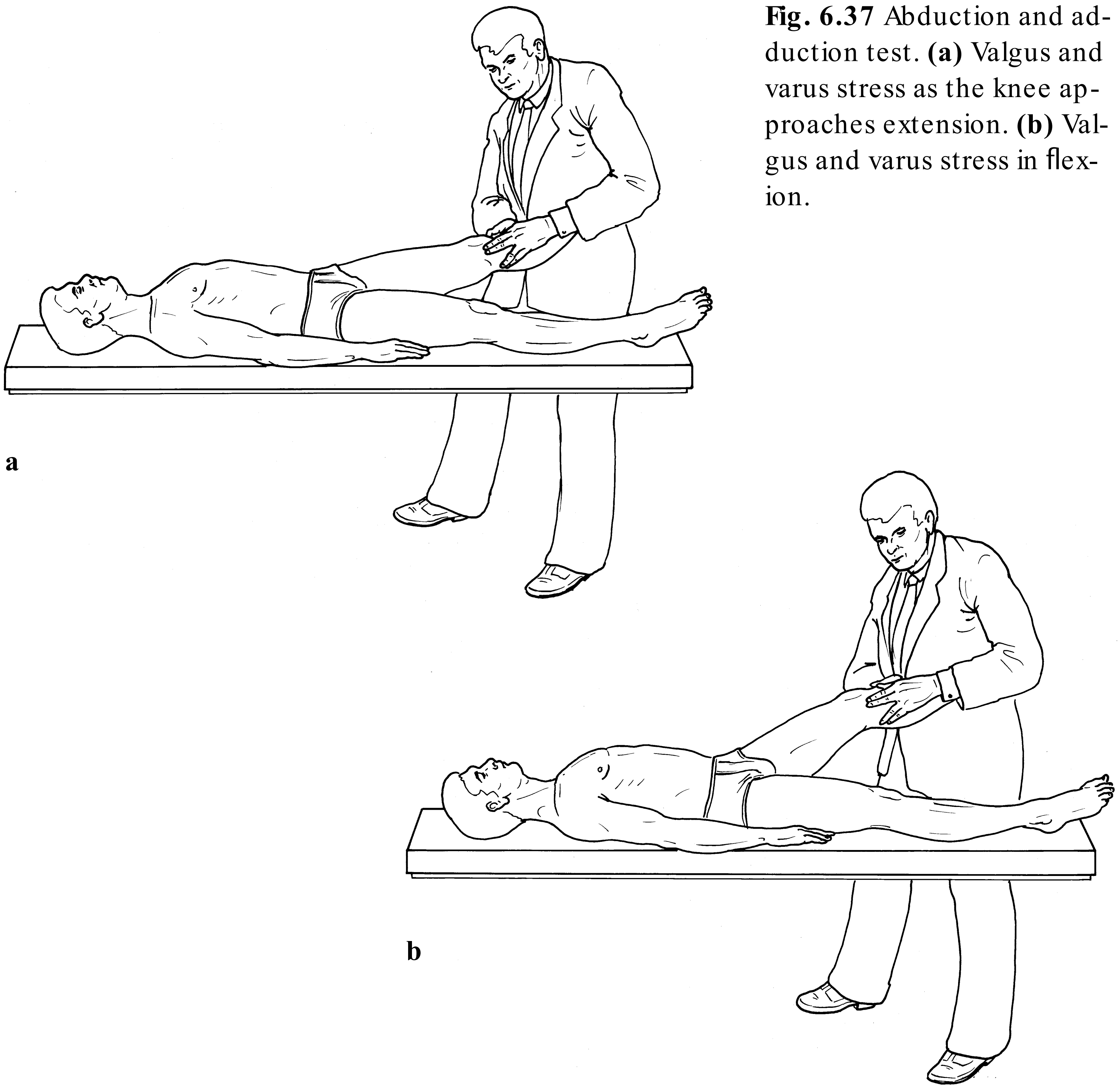


Fig. 6.37 Abduction and adduction test. **(a)** Valgus and varus stress as the knee approaches extension. **(b)** Valgus and varus stress in flexion.

Function Tests to Assess the Anterior Cruciate Ligament

Lachman Test (Noulis Test)

6

□ **Procedure:** The examiner holds the patient's knee between 15 and 30° of flexion. In this position in particular, the stabilizing function of the anterior cruciate ligament is essential in changing direction and braking. Insufficiency of the anterior cruciate ligament is therefore particularly evident in this position of the joint as it approaches extension due to the occurrence of lateral subluxation of the proximal tibia (pivoting).

The tibia should be slightly externally rotated and the anterior tibial translation force (anterior drawer) should be applied from the posteromedial aspect.

□ **Assessment:** The anterior cruciate ligament is damaged when mobility of the tibia with respect to the femur can be demonstrated. The end point of motion must be soft and gradual without a hard stop; any hard stop suggests a degree of stability of the anterior cruciate ligament. A hard end point within 3 mm suggests complete stability of the anterior cruciate, whereas one after 5 mm or more suggests relative stability of the anterior cruciate ligament, such as may be present following an earlier sprain.

Cruciate ligament injury should be suspected where the end point is soft or absent. In the presence of a drawer exceeding 5 mm, comparison with the contralateral knee is helpful in excluding congenital laxity of the articular ligaments.

A positive Lachman test is certain proof of anterior cruciate ligament insufficiency. A false-negative test may occur if the femur is not properly stabilized, if a meniscus lesion or degenerative changes such as osteophytes on the intercondylar eminence block translation, or if the tibia is medially rotated.

□ **Note:** In his dissertation as early as 1875, the Greek physician George Noulis described the test of the cruciate ligaments in a nearly extended position of the knee, exactly the same test known today as the Lachman test, which was not described or so named until 1976.

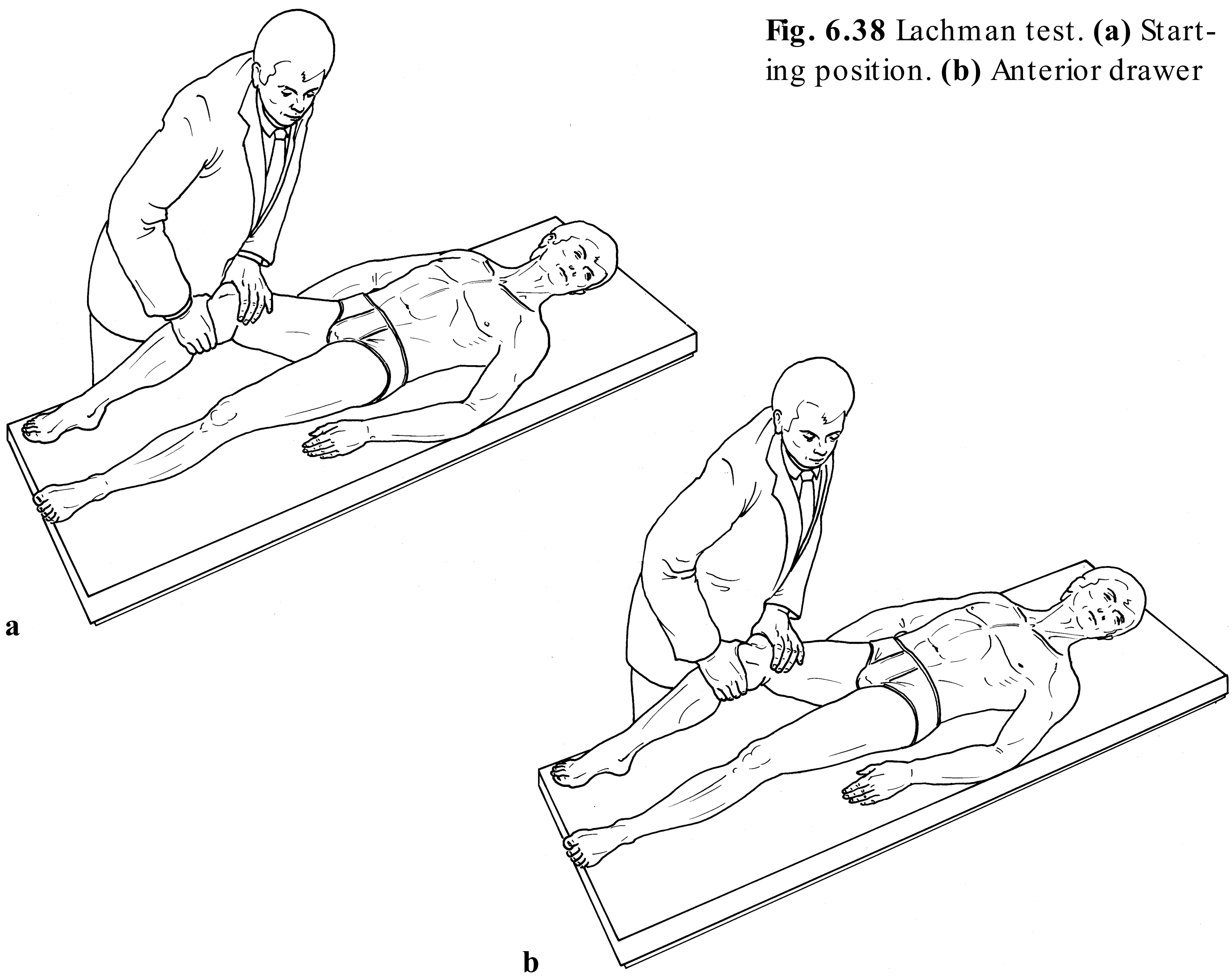


Fig. 6.38 Lachman test. **(a)** Starting position. **(b)** Anterior drawer

6

Prone Lachman Test

□ **Procedure:** The patient is prone. The examiner grasps the lateral aspect of the proximal tibia and immobilizes the patient's leg in his or her own axilla. With the other hand, the examiner grasps the distal femur immediately proximal to the patella to immobilize the thigh. Then the examiner pushes the tibia anteriorly with respect to the femur.

□ **Assessment:** Damage to the cruciate ligament is present where there is demonstrable mobility of the tibia relative to the femur. The motion must have a soft end point. Any hard end point suggests a certain stability of the anterior cruciate. Where this occurs within 3 mm, it suggests complete stability; where it only occurs after 5 mm, it suggests relative stability with previous elongation of the anterior cruciate.

Cruciate ligament injury should be assumed where the end point is soft or absent. In drawer motion exceeding 5 mm, comparison with the contralateral side is helpful in excluding congenital laxity of the articular ligaments.

A positive Lachman test is proof of insufficiency of the anterior cruciate ligament.

□ **Note:** Although the patient is relaxed in the prone position, it is not always easy to assess the quality of the end point.

A hard end point and hemarthrosis suggest an acute partial tear; a hard end point without hemarthrosis suggests a suspected chronic partial tear, elongation, or excessive laxity.

A soft end point and hemarthrosis suggest a complete tear; a soft end point without hemarthrosis suggests a chronic complete tear.

Where the end point is hard, a posterior cruciate lesion must be excluded by testing the spontaneous posterior drawer and applying the active tests.

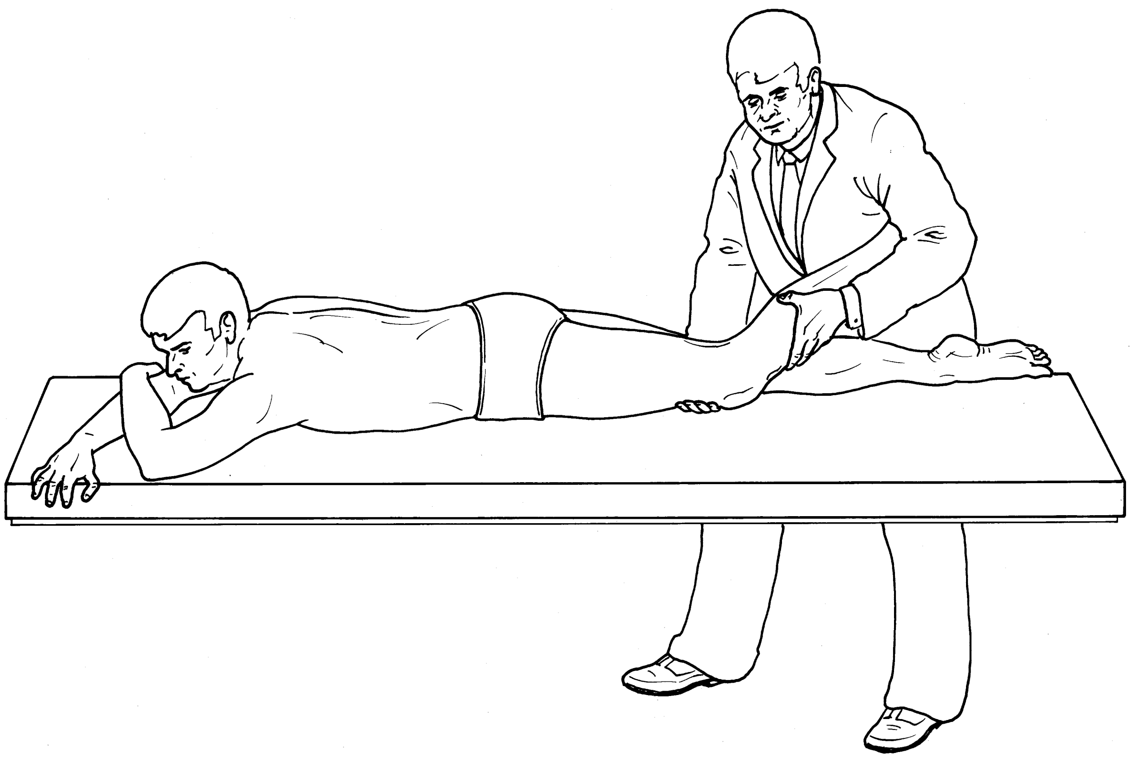


Fig. 6.39 Prone Lachman test.

Stable Lachman Test

A variation of the classic Lachman test.

□ **Procedure:** The patient is supine. The examiner places the patient's thigh over his or her own thigh. This holds the patient's leg in constant flexion that the patient cannot change. With the distal hand, the examiner pulls the tibia anteriorly while the other hand immobilizes the patient's thigh on the examiner's own thigh.

□ **Assessment:** Identical to that of the classic Lachman test.

□ **Note:** The classic Lachman test presents problems not only for examiners with small hands—simultaneously immobilizing the thigh and lower leg can also be difficult for any examiner with an obese or muscular patient. Using one's own thigh as a "workbench" for examining the patient's knee is an easy solution in such cases and one that allows examination even of obese or muscular patients. The character of the end point (hard or soft) is easier to evaluate in this test.

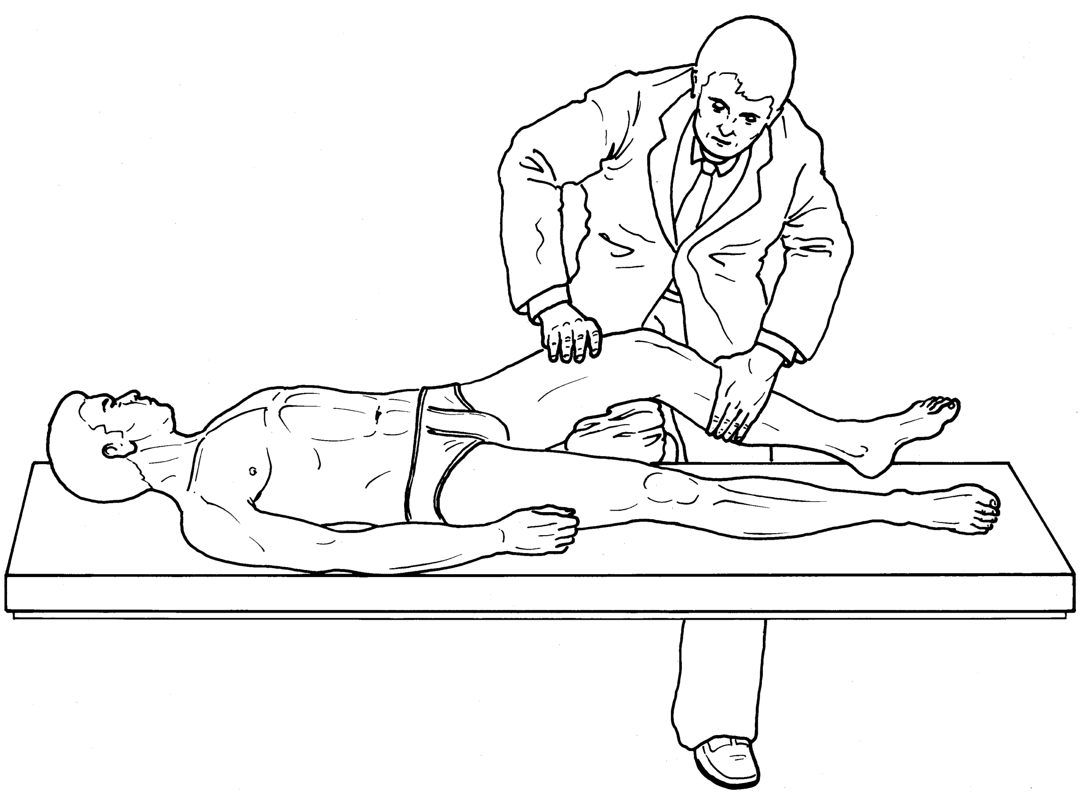


Fig. 6.40 Stable Lachman test.

No-Touch Lachman Test

□ **Procedure:** The patient is supine and grasps the thigh of the affected leg near the knee with both hands and slightly flexes the knee. The patient is then asked to raise the lower leg off the examining table while maintaining flexion in the knee. The examiner observes the position of the tibial tuberosity during this maneuver.

□ **Assessment:** If the ligaments are intact, there will be no change in contour, or only a slight one as the tibial tuberosity moves slightly anteriorly. In an acute injury to the capsular ligaments involving the anterior cruciate and medial collateral ligaments, the examiner will observe a significant anterior displacement of the tibial tuberosity (subluxation of the joint).

□ **Note:** This test often allows one to exclude complex injuries without having to touch the patient.

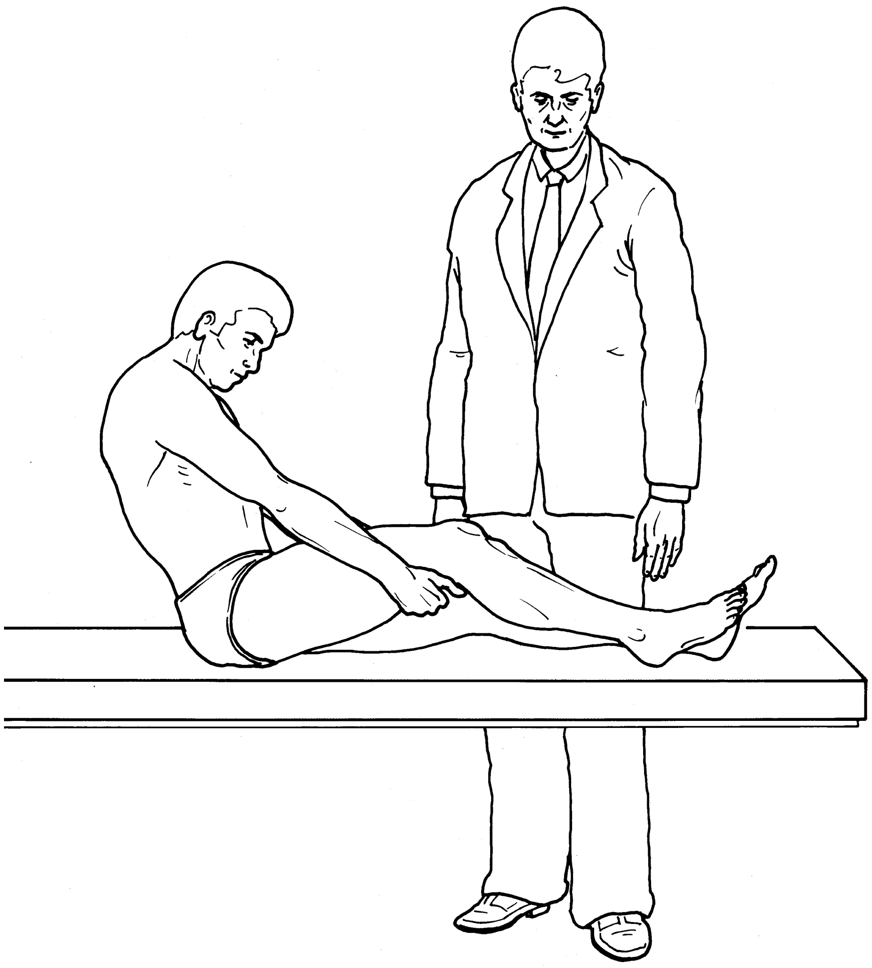


Fig. 6.41 No-touch Lachman test.

Active Lachman Test

□ **Procedure:** The examiner asks the supine patient to extend the leg in such a way as to lift the foot off the examining table. During this maneuver, the examiner keeps his or her eyes on the knee the better to discern the contours of the tibial tuberosity and patellar ligament. The examiner achieves slight passive flexion in the knee by passing one hand beneath the thigh of the patient's affected leg and resting it on the contralateral knee. The effect of the quadriceps is increased by immobilizing the foot on the examining table.
□ **Assessment:** Slight anterior migration of the tibial head will be observed where the anterior cruciate ligament is intact. In a cruciate tear, there will be a significant anterior migration compared with the contralateral side. This is because the anterior cruciate ligament no longer limits the displacement caused by contraction of the quadriceps.
□ **Note:** The physiologic drawer in active motion as the knee approaches extension usually measures 2 to 3 mm. In contrast, tibial displacement of 3 to 6 mm will be observed with an anterior cruciate ligament tear. This test should only be performed after excluding a posterior cruciate ligament injury, in which the tibia would spontaneously displace posteriorly. There, too, contraction of the quadriceps will produce significant anterior displacement of the tibia and with it a false-positive active anterior drawer test.

Contraction of the quadriceps can also cause meniscal impingement where loosening of the posterior attachment of the medial meniscus accompanies the insufficiency of the medial ligaments and anterior cruciate.

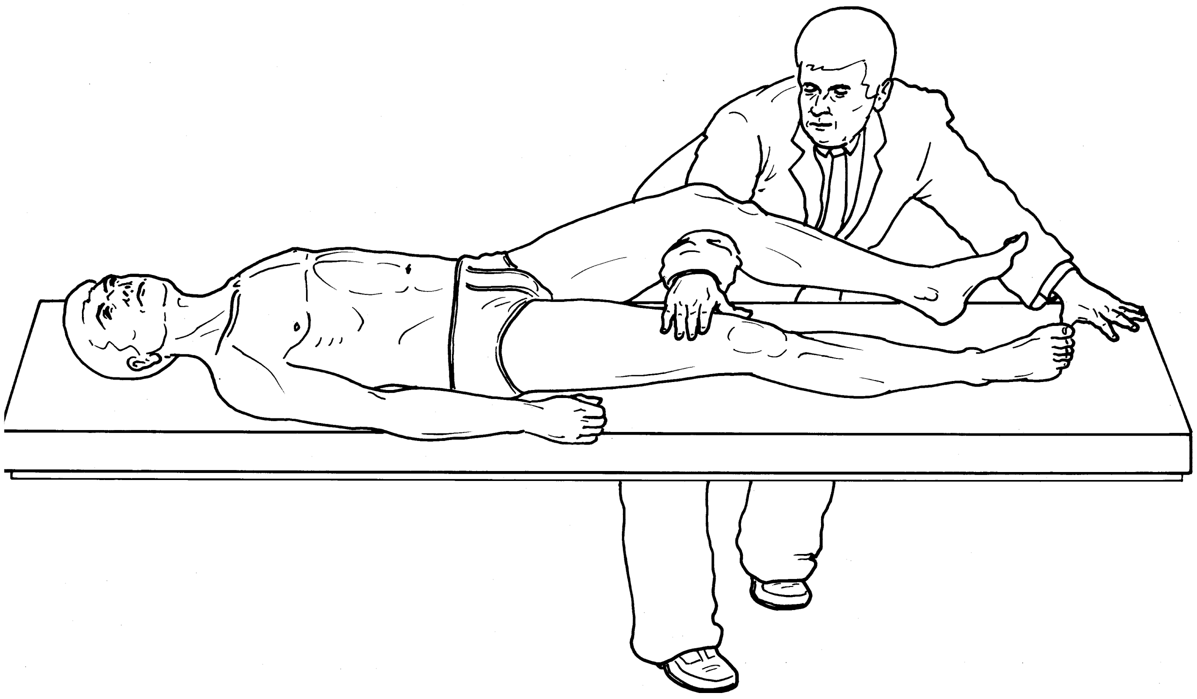


Fig. 6.42 Active Lachman test.

The active Lachman test differs from the traditional Lachman test in that the lower leg can easily be immobilized in various degrees of rotation and the stabilizing effect of the medial and lateral capsular ligaments can be assessed. Generalized anterior instability (involving the anterior cruciate ligament and the medial, posteromedial, lateral, and posterolateral capsular ligaments) will produce significant active anterior tibial displacement in internal and neutral rotation and, especially, in external rotation.

Anterior Drawer Test in 90° Flexion

Passive anterior drawer test to assess the stability of the anterior cruciate ligament.

Procedure: The patient is supine with the hip flexed 45° and the knee flexed 90°. The examiner sits on the edge of the examining table and uses his or her buttock to immobilize the patient's foot in the desired rotational position. The examiner then grasps the tibial head with both hands and pulls it anteriorly with the patient's knee flexors relaxed. The test is performed in a neutral position, with the foot in 15° of external rotation to assess anterior and medial instability, and with the foot in 30° of internal rotation to assess anterior and lateral instability.

Assessment: A visible and palpable anterior drawer (that is, anterior displacement of the tibia with a soft end point) is present in chronic insufficiency of the anterior cruciate ligament.

The anterior drawer test in 90° of flexion is often negative in acute injuries because pain often prevents the patient from achieving this degree of flexion and causes reflexive muscle contraction. Additionally, these are usually combined injuries involving complete or partial ligament tears so that the stress of the drawer test stretches the partially torn medial and lateral structures. The resulting pain produces false-negative test results, giving the appearance of a stable joint.

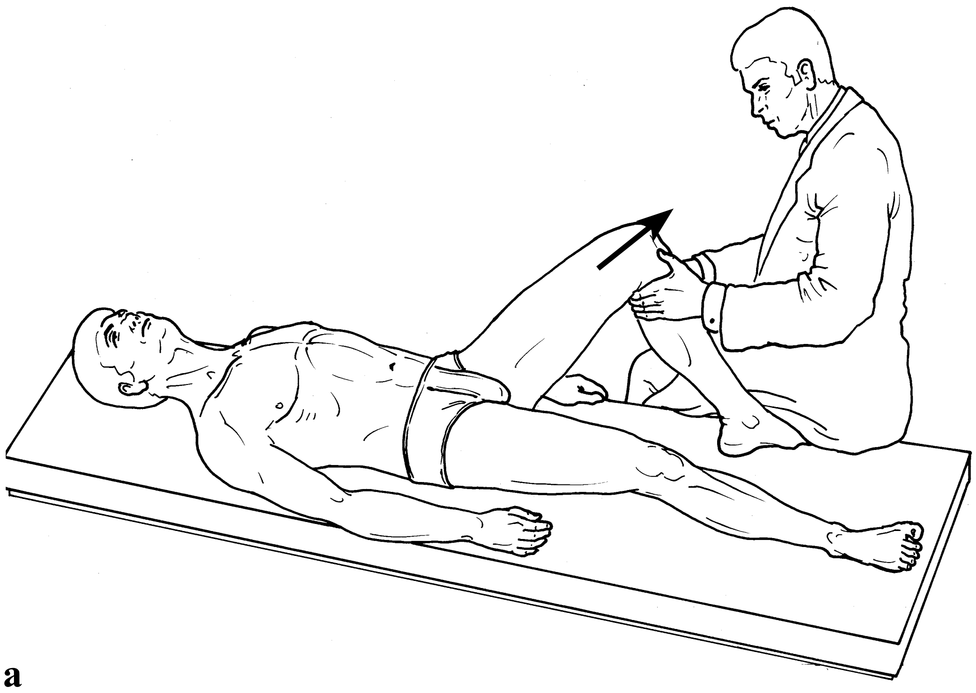
In acute injuries in particular, the test should preferably be performed with the knee in slight flexion (Lachman test). The situation is different in chronic ligament injuries, where the primary symptom is the sensation of instability. In these cases, the test can usually be performed painlessly in 90° of flexion and still provide useful diagnostic information.

Note: As a rule, the anterior drawer is best assessed in neutral rotation. This allows one to demonstrate the greatest degree of displacement. Rotation forces the tibia into a position where the twisting of the peripheral ligaments and capsular structures increases tension in the joint, impairing the mobility of the drawer. Assessment of rotational stability together with assessment of lateral stability in flexion and extension provides information about the complexity of the ligament injury and the stability of the secondary stabilizers.

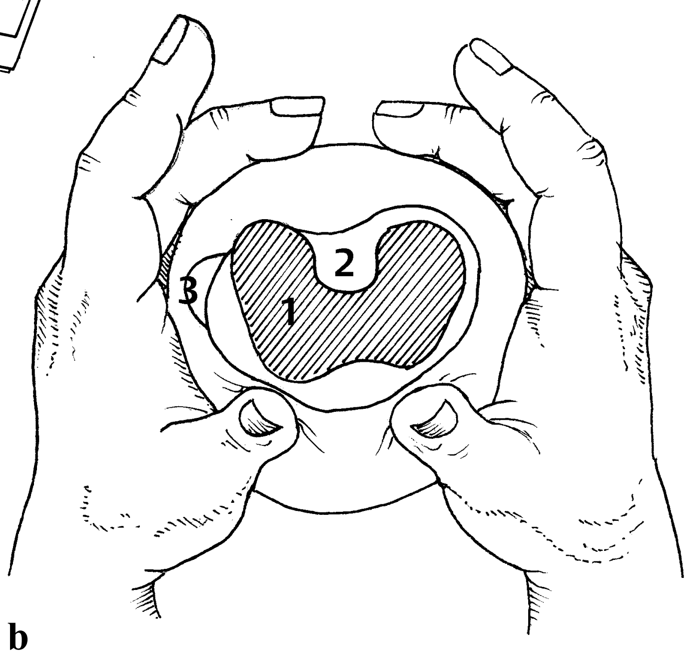
An anterior drawer should not automatically be interpreted as an anterior cruciate ligament tear. On the other hand, a negative drawer test does not necessarily confirm that the anterior cruciate is intact. The proximal portion of the tibia is pulled anteriorly or pushed posteriorly. It can be difficult to determine the exact starting position (the neutral position) from which an anteriorly directed force will produce an anterior drawer. For example, where the examiner exerts an anterior drawer stress in the presence of a posterior cruciate ligament injury in which the tibial head is posteriorly depressed (a spontaneous posterior drawer), it will seem as if an isolated anterior drawer is present. What has actually happened in this case is that the tibia has merely been drawn anteriorly out of its posterior displacement (due to the posterior cruciate tear) and into a neutral position. The anterior cruciate then tenses and limits further anterior displacement of the tibia.

□ **Caution:** An apparent anterior drawer may only be interpreted as a true anterior drawer once the absence of a posterior drawer has been demonstrated.

6



a



b

Fig. 6.43 Anterior drawer test in 90° flexion. **(a)** Starting position in external rotation. **(b)** Interior of knee (superior view):
1 Femur.
2 Tibia.
3 Fibula.

Jakob Maximum Drawer Test

□ **Procedure:** The patient is supine with the knee flexed 50 to 60°. The examiner pushes the tibial head into maximum anterior subluxation with his or her forearm while grasping the patient's contralateral knee with the hand of the same arm. With the other hand, the examiner grasps the tibial head and palpates how far anteriorly the medial or lateral joint cavity is displaced. The patient's lower leg is not immobilized in this test so that rotation is not restricted. This allows maximum tibial displacement.
□ **Assessment:** See anterior drawer test in 90° flexion.

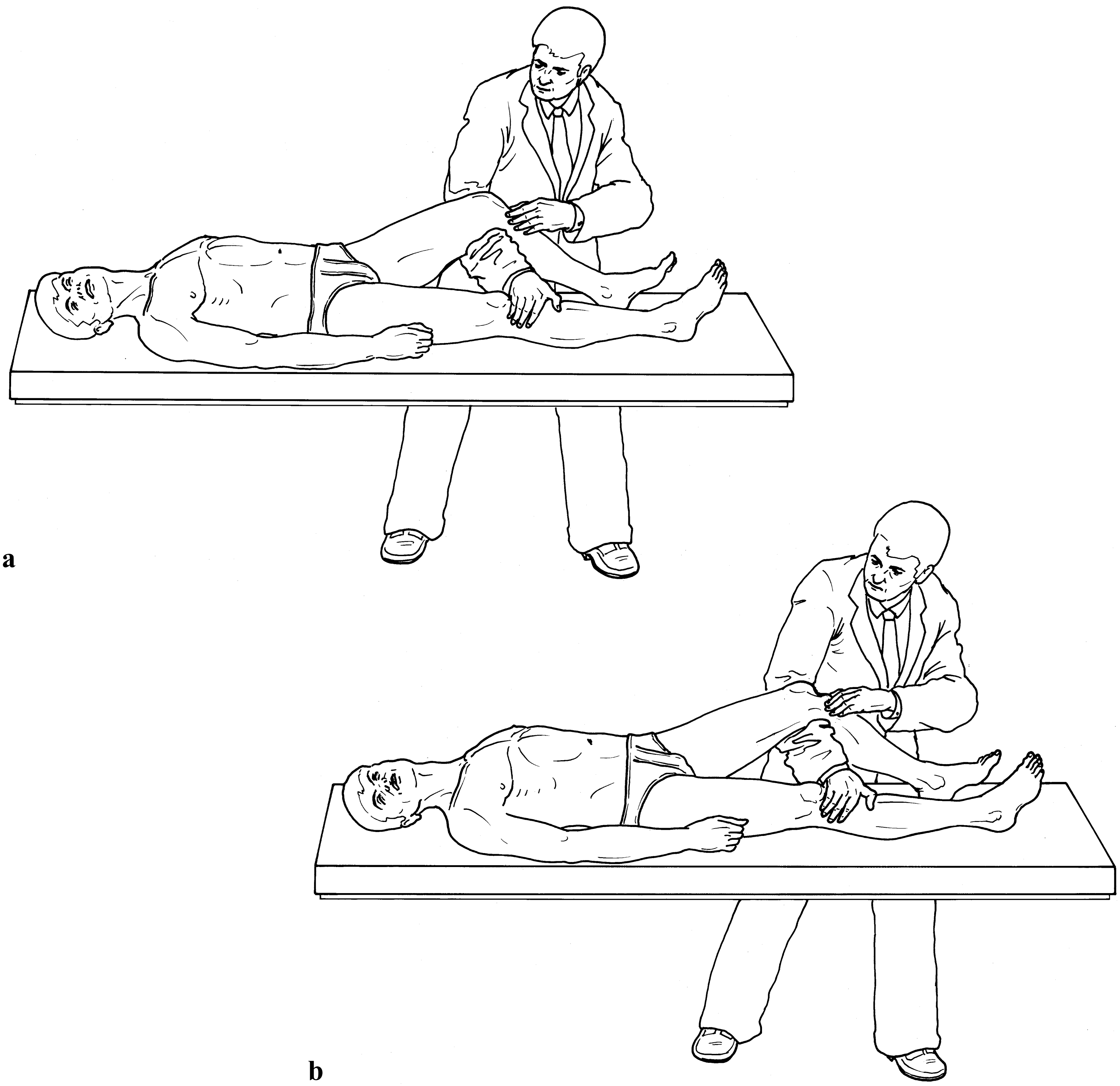


Fig. 6.44 Jakob maximum drawer test. **(a)** Starting position. **(b)** Maximum anterior traction on the tibia.

Pivot Shift Test

□ **Procedure:** The patient is supine. The examiner grasps and immobilizes the lateral femoral condyle with one hand and palpates the proximal tibia or fibula with the thumb. With the other hand, the examiner holds the patient's lower leg in internal rotation and abduction (valgus stress). From this starting position the knee is then moved from extension into flexion.

□ **Assessment:** In the presence of a torn anterior cruciate ligament, the valgus stress will cause the tibia to subluxate anteriorly while the knee is still in extension. The blockade of the knee in anterior subluxation depends on the degree of valgus stress applied; occasionally the sign can be elicited more easily when the examiner immobilizes the patient's leg between his or her own forearm and waist while applying slight axial compression. The knee is then flexed while the same internal rotation and abduction of the lower leg is maintained; this then causes the subluxated tibial head to reduce posteriorly at 20 to 40° of flexion. The pivot shift test is particularly well suited for testing anterolateral instability. The iliotibial tract, which with increasing flexion glides from a position anterior to the lateral epicondyle in extension to a position posterior to the axis of flexion, draws the tibial head posteriorly again. Thus a positive test result is always dependent on an intact iliotibial tract. The degree of reduction and flexion depends on the severity of the anterior subluxation. Reduction occurs earlier when there is only slight anterior translation. The patient usually confirms the diagnosis by reporting that the typical sensation of the knee giving way felt in sports activities can be reproduced in this test.

According to Jakob, a genuine pivot shift phenomenon can partially disappear, despite anterior cruciate ligament insufficiency, under the following conditions:

1. When a complete tear of the medial collateral ligament is present, the valgus opening prevents force concentration in the lateral compartment. Subluxation cannot occur under these circumstances.
2. When the iliotibial tract is traumatically divided, only the subluxation will be observed, not the abrupt reduction.
3. A bucket-handle tear of the medial or lateral meniscus can prevent anterior translation or reduction of the tibia.
4. Increasing osteoarthritis in the lateral compartment with osteophytes can create a concave contour along the once convex lateral tibial plateau.

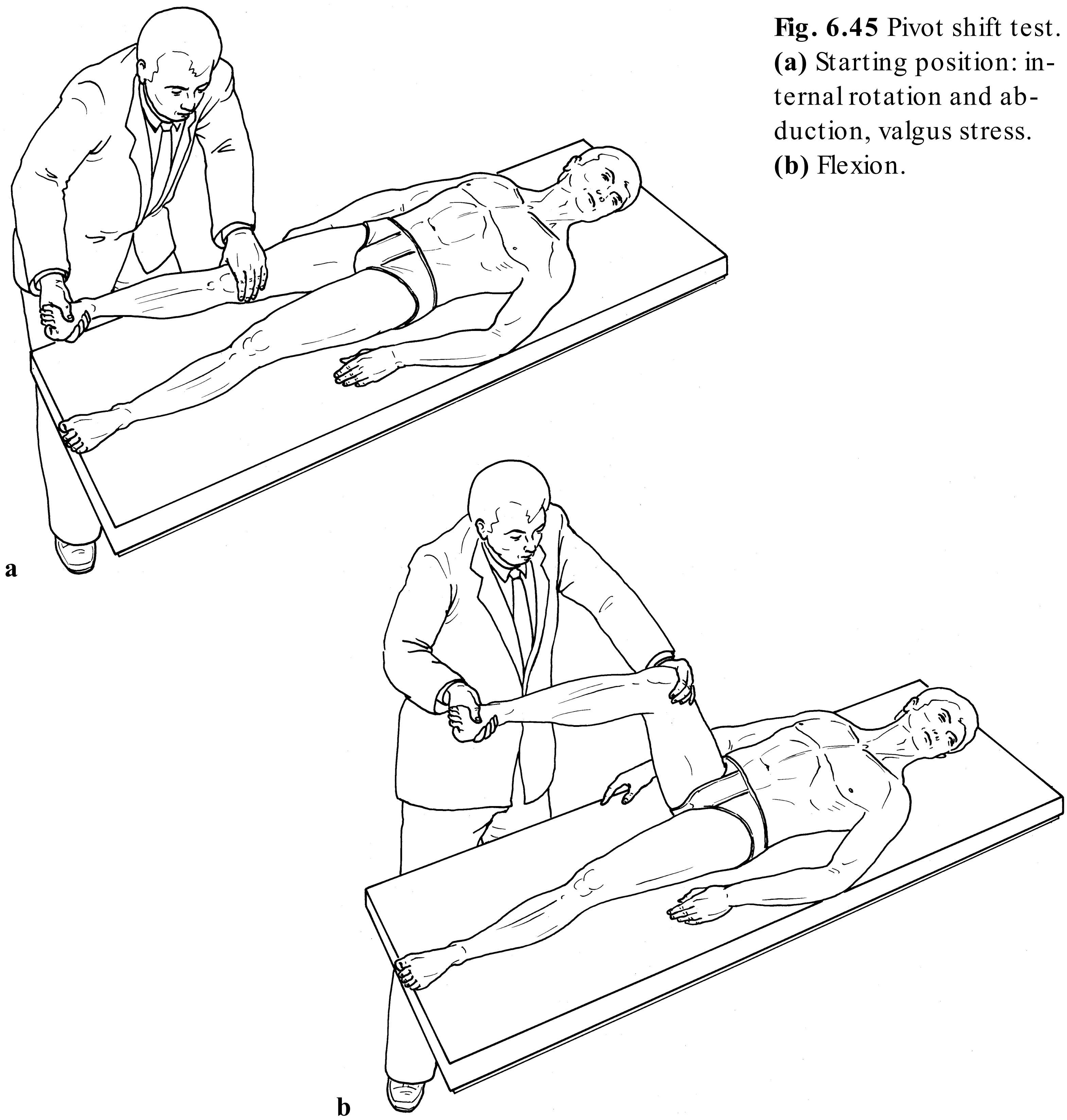


Fig. 6.45 Pivot shift test. **(a)** Starting position: internal rotation and abduction, valgus stress. **(b)** Flexion.

Jakob Graded Pivot Shift Test

Gradation of the pivot shift test allowing for translation and rotation of the tibia.

□ **Procedure:** The procedure is identical to the pivot shift test except that here instability of the knee is assessed with the lower leg not only in internal rotation but also in neutral and external rotation.

□ **Assessment:**

- Pivot shift grade I: The pivot shift test is positive only in maximum internal rotation; it is negative in neutral and external rotation. The subluxation as the knee approaches extension is more palpable than visible to the examiner (slight translation may be apparent).

- Pivot shift grade II: The pivot shift test is positive in internal and neutral rotation; however, it is negative in external rotation. There is visible and palpable translation on the lateral aspect of the joint.
- Pivot shift grade III: The pivot shift test is clearly positive in neutral rotation and particularly conspicuous in external rotation. The sign is less distinct in internal rotation. Pivot shift grade III can only be demonstrated in acute knee injuries where the posteromedial and lateral structures are damaged in addition to the anterior cruciate. In chronic instability, a grade III pivot shift will be detectable in cases where the secondary stabilizers have loosened over time.

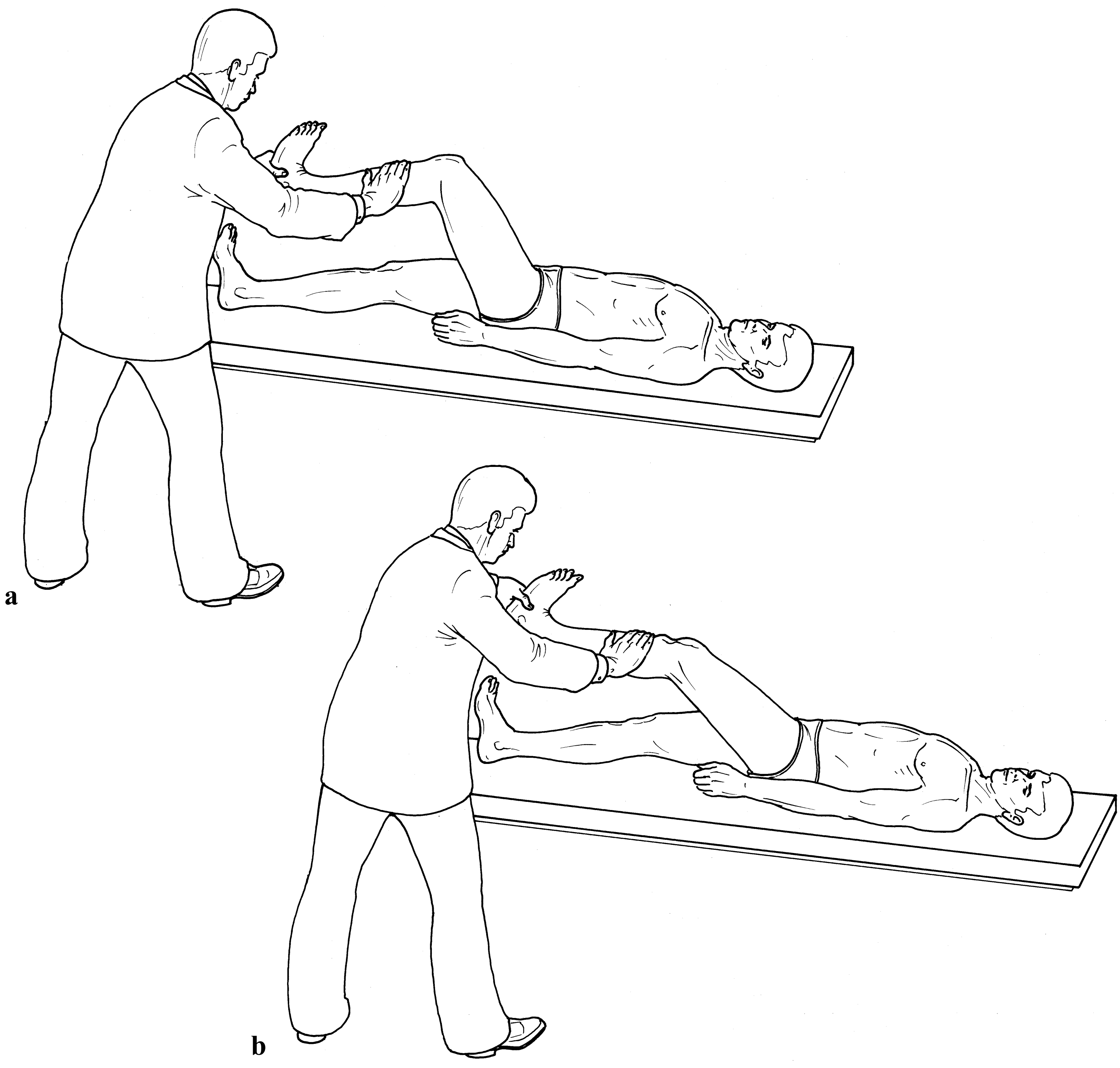


Fig. 6.46 Jakob graded pivot shift test. **(a)** Starting position: flexion and internal rotation of lower leg, valgus stress on the knee. **(b)** Anterior subluxation of the lateral tibial head as the knee approaches extension with lower leg internally rotated and valgus stress on the knee.

□ **Note:** In an anterior cruciate tear, both the medial and lateral portions of the tibia migrate anteriorly under the stress of the anterior drawer.

In an isolated tear of the anterior cruciate ligament, the anterior motion of the lateral portion of the tibia will be more pronounced than that of the medial portion. The anterior motion of the medial portion of the tibial plateau increases relative to that of the lateral portion as the number of injured medial structures increases. Increasing anterior motion of the medial tibial plateau in turn increases the severity of the subluxation and subsequent reduction phenomenon observed by the examiner. This reduction will also be observed to occur at an increasingly high degree of flexion.

Modified Pivot Shift Test

□ **Procedure:** The patient is supine. With one hand, the examiner holds the patient's lower leg in internal rotation while the other hand grasps the tibial head laterally and holds it in a valgus position. In a positive test, this alone will produce anterior subluxation of the lateral tibial head. The rest of the procedure is identical to the pivot shift test. Subsequently flexing the knee while maintaining internal rotation and valgus stress on the lower leg causes posterior reduction of the subluxated tibial head at about 30° of flexion. The test is performed with the femoral head in abduction and adduction and in each case with the lower leg in external and internal rotation.

□ **Assessment:** The iliotibial tract plays an important role in subluxation as the knee approaches extension and in subsequent reduction as flexion increases in the pivot shift test. The initial stress present in the iliotibial tract greatly influences the severity of subluxation. The iliotibial tract is relaxed in hip abduction, whereas it is under tension in hip adduction. In patients with an insufficient anterior cruciate ligament, subluxation is therefore more pronounced in hip abduction than it is in hip adduction,

The iliotibial tract contributes directly and indirectly (passively) to stabilizing the lateral knee. The portion of the iliotibial tract between the fibers of Kaplan and Gerdy's tubercle can be regarded as a passive ligamentlike structure that is placed under tension by the proximal portion of the tract that runs through the thigh. The tension in this passive femorotibial portion of the tract determines the degree of subluxation of the tibial head. Internally rotating the lower leg and adducting the hip tenses the entire iliotibial tract, which increases tension in the ligamentlike portion that spans the knee. This tension will prevent anterior subluxation of the tibial head during the pivot shift test in the presence of a torn anterior cruciate ligament. However, externally rotating the lower leg reduces the tension in the portion of the iliotibial tract that spans the knee, allowing greater anterior subluxation of the tibial head. The degree of subluxation is even greater when the leg is abducted.

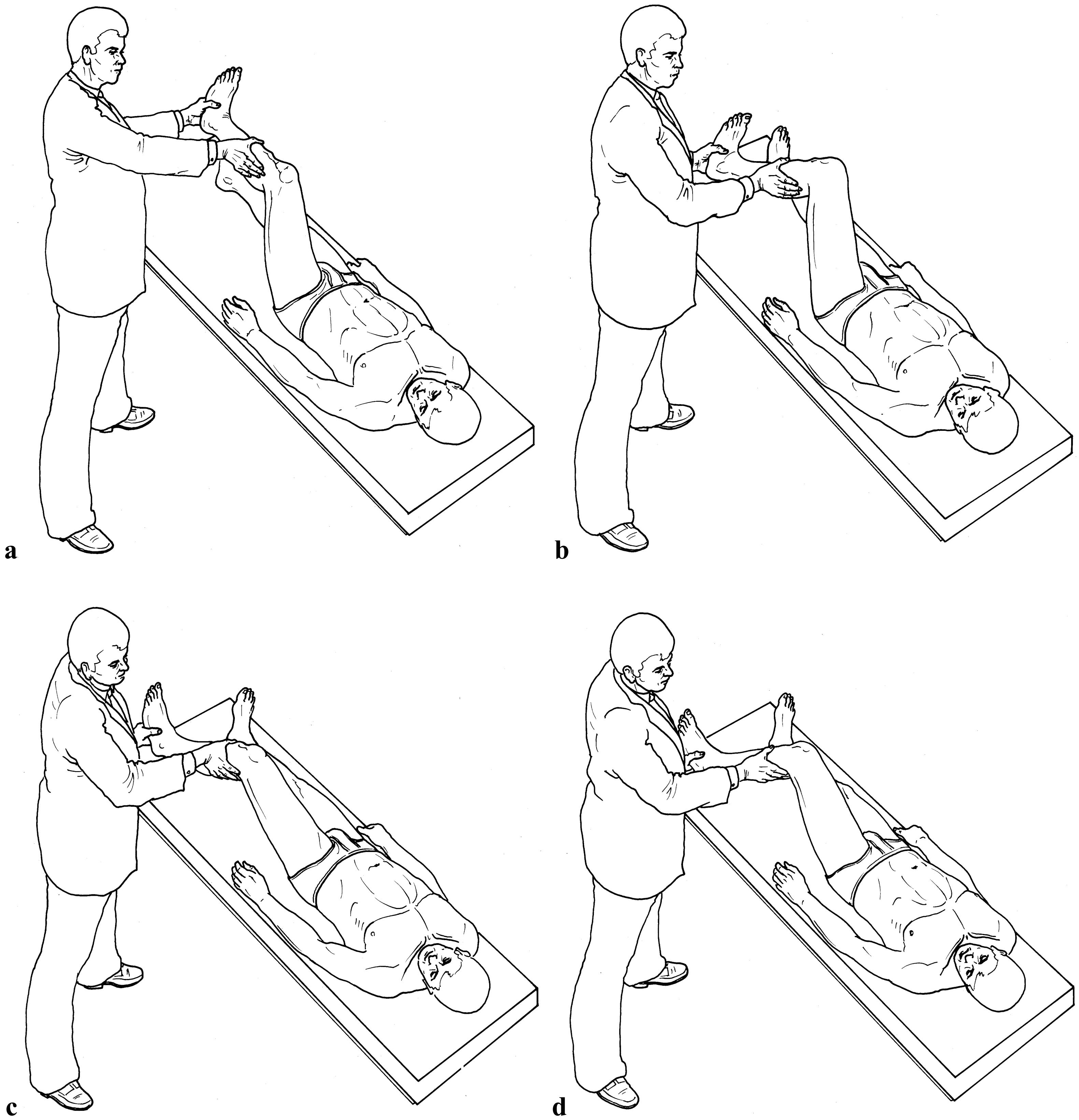


Fig. 6.47 Modified pivot shift test. **(a)** Subluxation during extension of the adducted leg with valgus stress and lower leg internally rotated. **(b)** Reduction during flexion of the leg from the same position. **(c)** Subluxation during extension of the abducted leg with valgus stress on the knee and lower leg externally rotated. **(d)** Reduction during flexion of the leg from the same position.

Medial Shift Test

□ **Procedure:** The examiner immobilizes the patient's lower leg between his or her forearm and waist to evaluate the medial or lateral translation (tibial displacement) as the knee approaches extension. To assess medial translation, the examiner places one hand on the lower leg slightly distal to the medial joint cavity while the other hand rests on the lateral thigh. While applying a valgus stress to the knee via the lower leg, the examiner presses medially with the hand resting on the patient's thigh.

□ **Assessment:** In an anterior cruciate tear, the tibia can be displaced medially until the intercondylar eminence comes in contact with the medial femoral condyle. Because the posterior cruciate ligament courses from medial to lateral, lateral translation of the tibial head will be detectable in the presence of a posterior cruciate tear (positive lateral shift test).

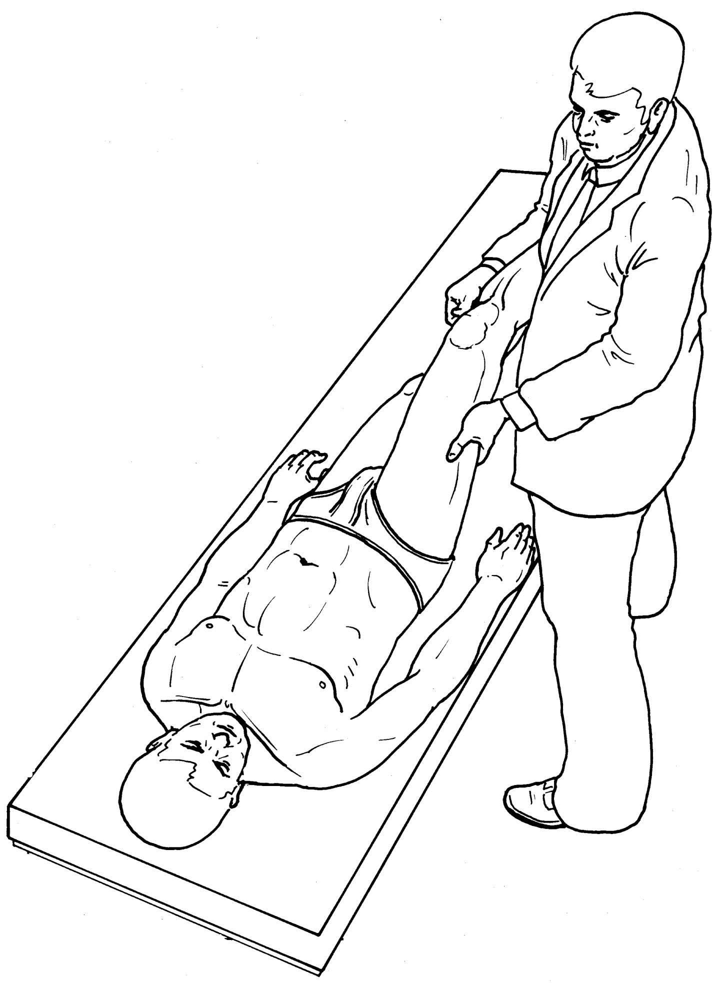


Fig. 6.48 Medial shift test.

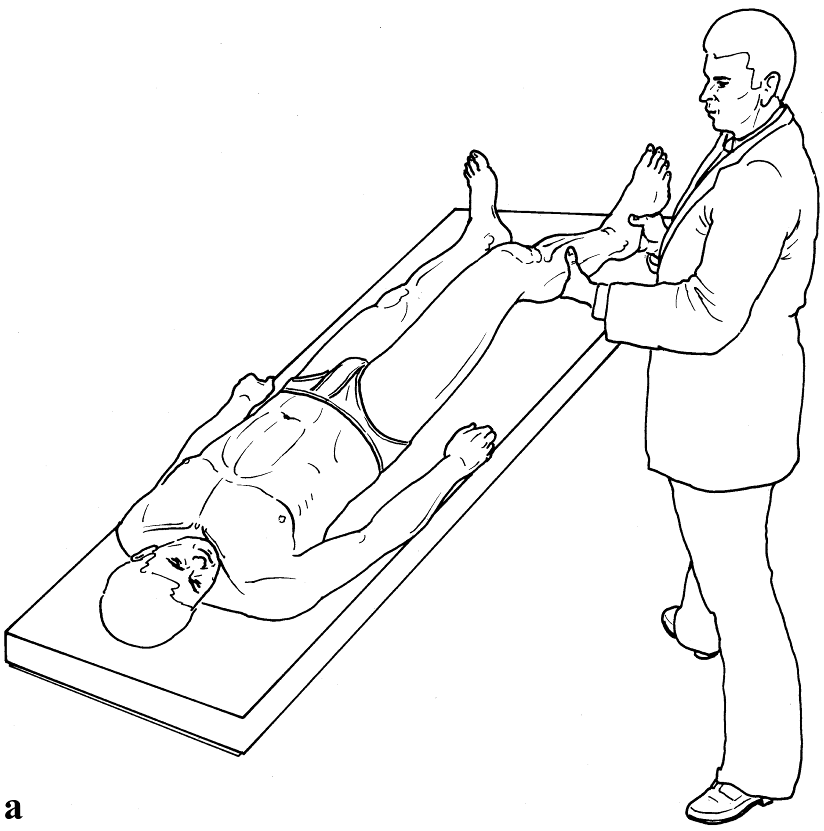
Soft Pivot Shift Test

□ **Procedure:** The patient is supine. The examiner grasps the patient's foot with one hand and the calf with the other. First, the examiner alternately flexes and extends the knee carefully, using these normal everyday motion sequences to alleviate the patient's anxiety and reduce reflexive muscle tension. The patient's hip is abducted, and the foot is held in neutral or external rotation.

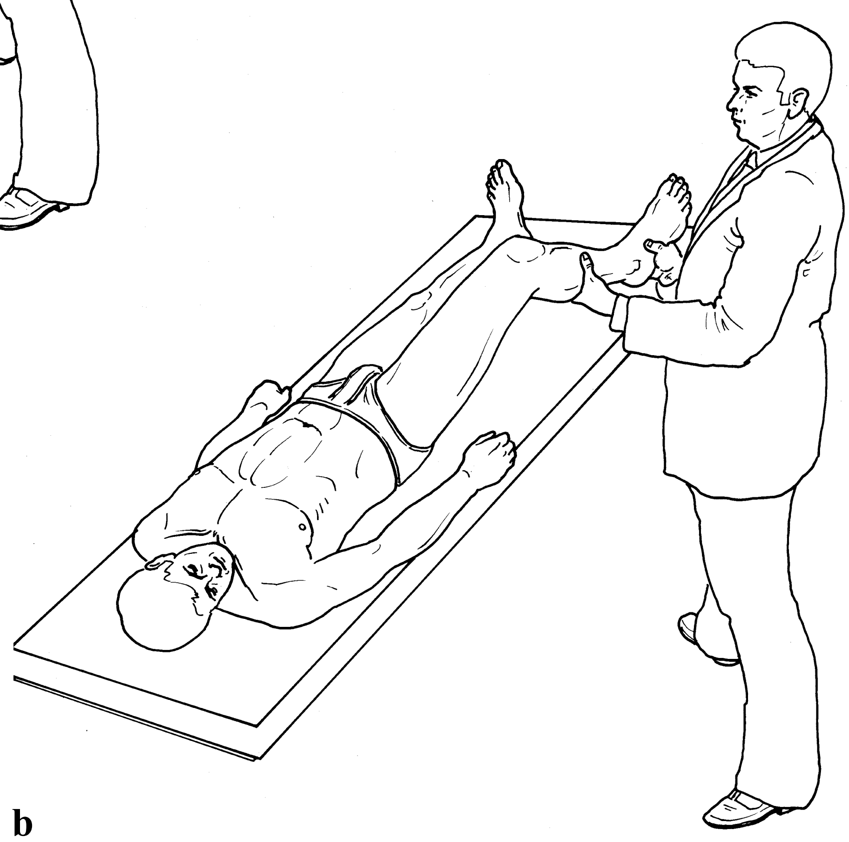
Next, the examiner gently applies axial compression after about 3 to 5 flexion and extension cycles. With the hand resting on the calf, the examiner applies a mild anterior stress.

Assessment: Under axial compression and mild anterior stress, slight subluxation will occur as the knee approaches extension, with reduction occurring as flexion increases. By varying the speed of the flexion and extension cycle, the axial compression, and the anteriorly directed pressure, the examiner can precisely control the intensity of the subluxation and subsequent reduction. In this test, the examiner literally feels his or her way toward the subluxation and reduction.

Note: The soft pivot shift test ensures reduction with minimal pain or even with no pain at all. Carefully performed, this test can be repeated several times without the patient's complaining of pain.



a



b

Fig. 6.49 Soft pivot shift test. **(a)** Subluxation as the knee approaches extension with the lower leg externally rotated while applying axial compression and anterior stress. **(b)** Reduction in flexion while maintaining the axial compression and applying slight valgus stress.

Martens Test

□ **Procedure:** The patient is supine. The examiner stands lateral to the injured leg and immobilizes the patient's calf just distal to the knee with one hand, resting the index finger on the fibula. The patient's lower leg is immobilized between the examiner's forearm and waist while a valgus stress is applied. While pulling the lower leg anteriorly with the same hand, the examiner pushes the distal thigh posteriorly with the other.

□ **Assessment:** The maneuver begins with the knee in a position approaching extension. As flexion is increased from this starting position, the subluxated lateral portion of the tibia will reduce at about 30° of flexion if an anterior cruciate ligament injury is present.

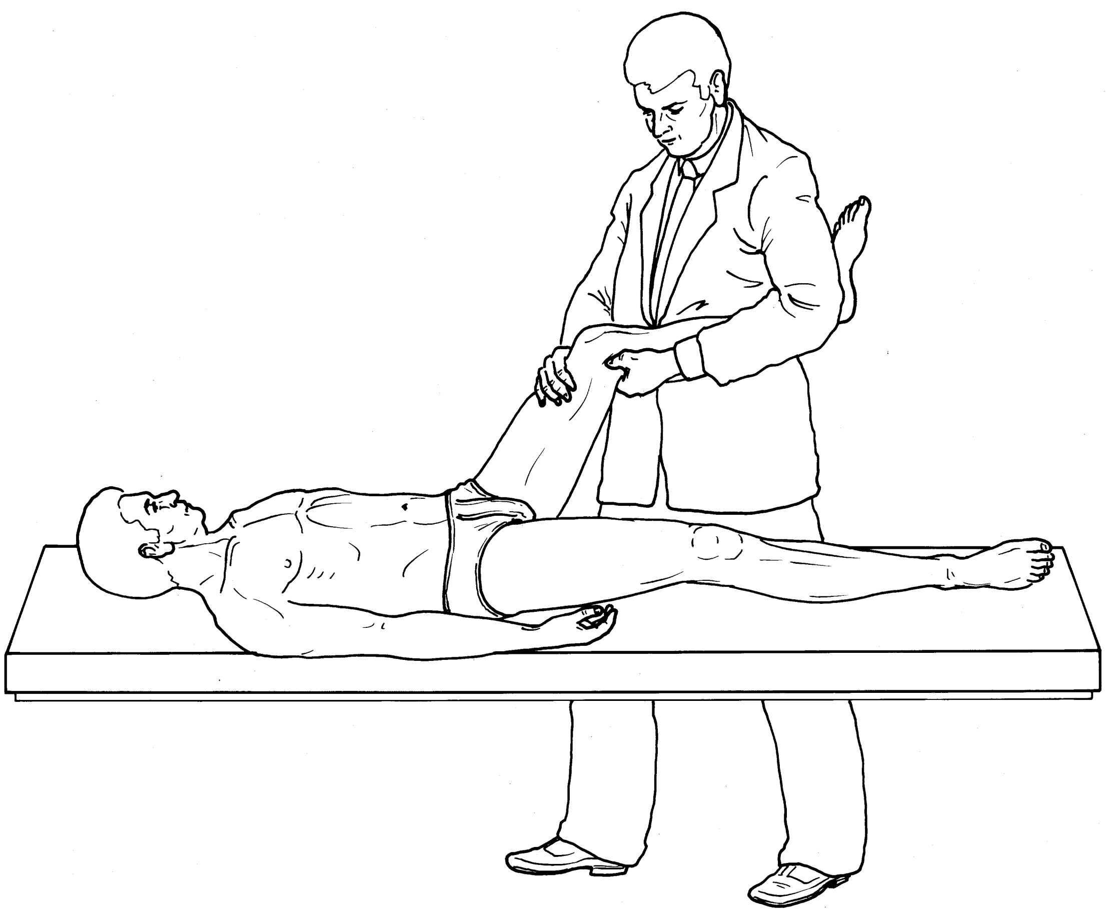


Fig. 6.50 Martens test.

Losee Test

□ **Procedure:** The patient is supine. The examiner grasps the knee laterally with the thumb posterior to the fibular head and the fingers resting on the patella. The other hand grasps the lower leg medially proximal to the ankle. In contrast to the other dynamic subluxation tests, the examiner does not internally rotate the lower leg but instead moves it into slight external rotation.

□ **Assessment:** When the knee is extended from 40 to 50° of flexion, an anterior cruciate ligament injury will lead to visible and palpable anterior subluxation of the lateral portion of the tibial head.

□ **Note:** Because of the external rotation of the tibia at the onset of the test, the Losee test has traditionally occupied a singular position among the dynamic subluxation tests. However, it is important for the examiner not to force this external rotation, but to hold the lower leg in a relaxed way in external rotation with the knee flexed. Extending the knee causes the lateral portion of the tibia to subluxate anteriorly, meaning that the entire lower leg moves into internal rotation. The examiner must not interfere with this relative internal rotation.

Slocum Test

Procedure: The patient lies on the unaffected side with the hip and knee flexed, holding the injured upper leg in slight internal rotation with the foot extended where possible. In this position, the weight of the leg exerts a slight valgus stress. The examiner stands behind the patient and grasps the patient's thigh with one hand and the tibial head with the other, palpating the fibular head with the thumb or index finger.

Assessment: In an injury to the anterior cruciate ligament, the lateral tibial head will subluxate anteriorly with the knee in a position approaching extension. Subsequent flexion will then lead to posterior reduction of the tibial head at about 30° of flexion.

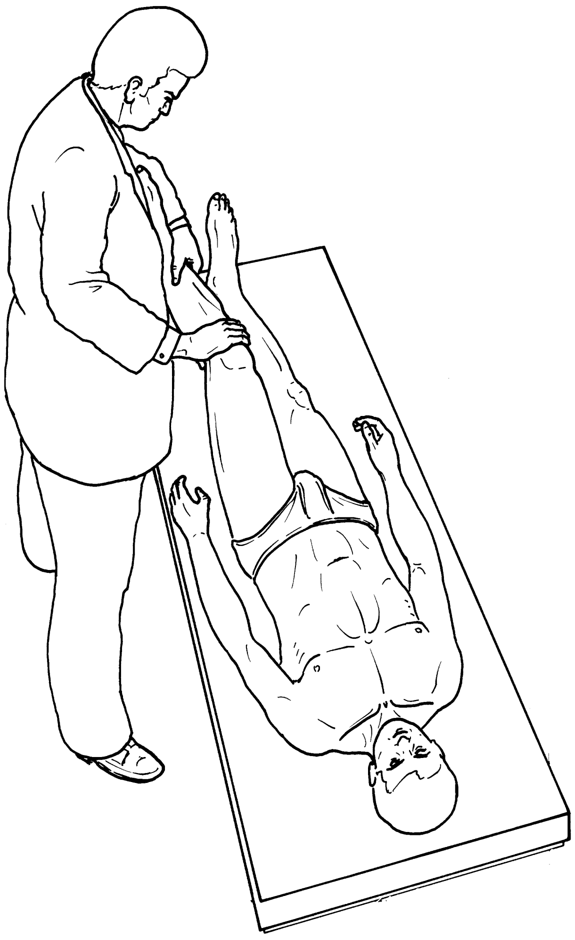


Fig. 6.51 Losee test.

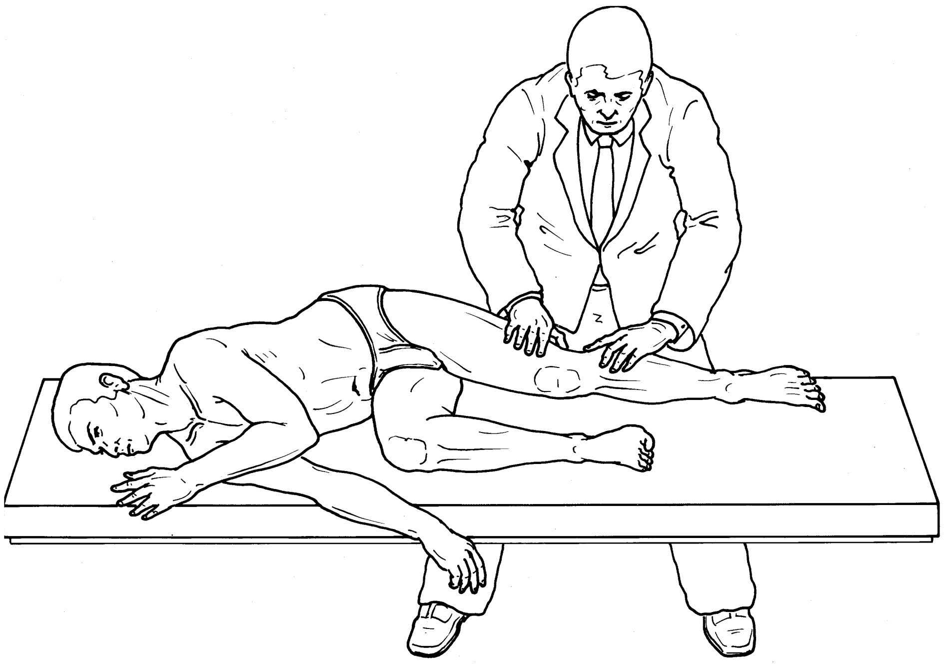


Fig. 6.52 Slocum test.

Arnold Crossover Test

□ **Procedure:** The patient is standing. The examiner immobilizes the foot of the patient's injured leg. The patient then crosses the normal leg over the injured leg, rotating the pelvis and trunk toward the injured side.

□ **Assessment:** The contraction of the quadriceps causes the immobilized leg to reproduce the lateral pivot shift phenomenon. The patient will experience an unpleasant sensation and report that the knee is about to dislocate.

□ **Note:** In muscular patients, this test usually provides more useful diagnostic information than the other dynamic anterior cruciate ligament tests.

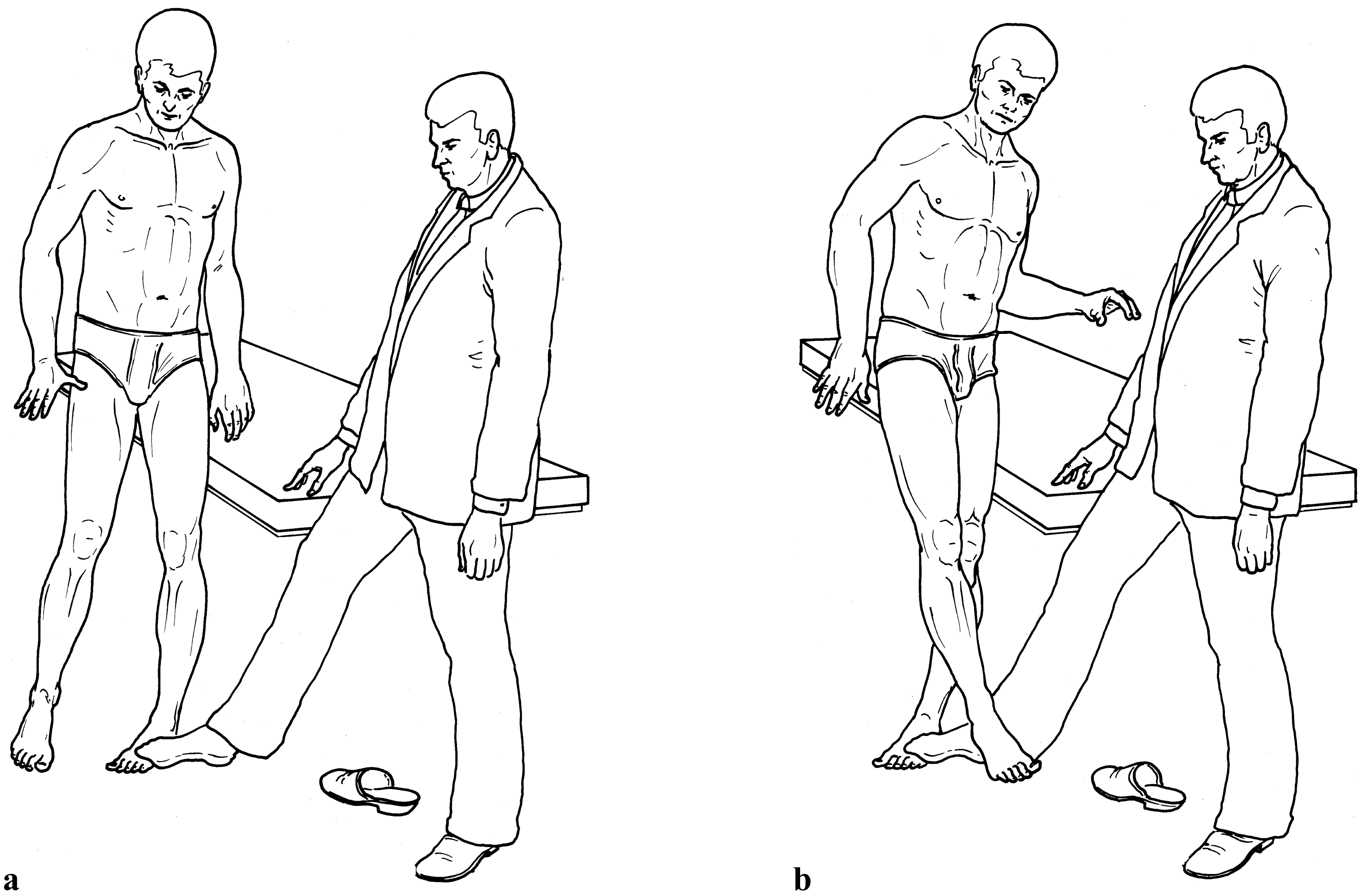


Fig. 6.53 Arnold crossover test. **(a)** Starting position. **(b)** Crossover.

Noyes Test

□ **Procedure:** The patient is supine. The examiner grasps the head of the tibia with both hands and immobilizes the patient's distal lower leg between his or her forearm and waist. With the knee in about 20° of flexion, the examiner elicits a slight anterior drawer motion while simultaneously using the index fingers to evaluate whether the hamstrings are relaxed. The distal femur will drop into external rotation and slightly recede posteriorly (subluxation). The knee is then flexed from this position.

□ **Assessment:** In contrast to other dynamic anterior subluxation tests, it is not the lateral portion of the tibia but the distal femur that is tested for reduction and subluxation relative to the tibial head, which the examiner immobilizes and guides posteriorly. The test is positive when knee flexion results in palpable internal rotation of the distal femur (reduction). This indicates cruciate ligament insufficiency.

□ **Note:** The Noyes test is suitable for assessing cruciate ligament insufficiency in an apprehensive patient who has difficulty relaxing the hamstrings.

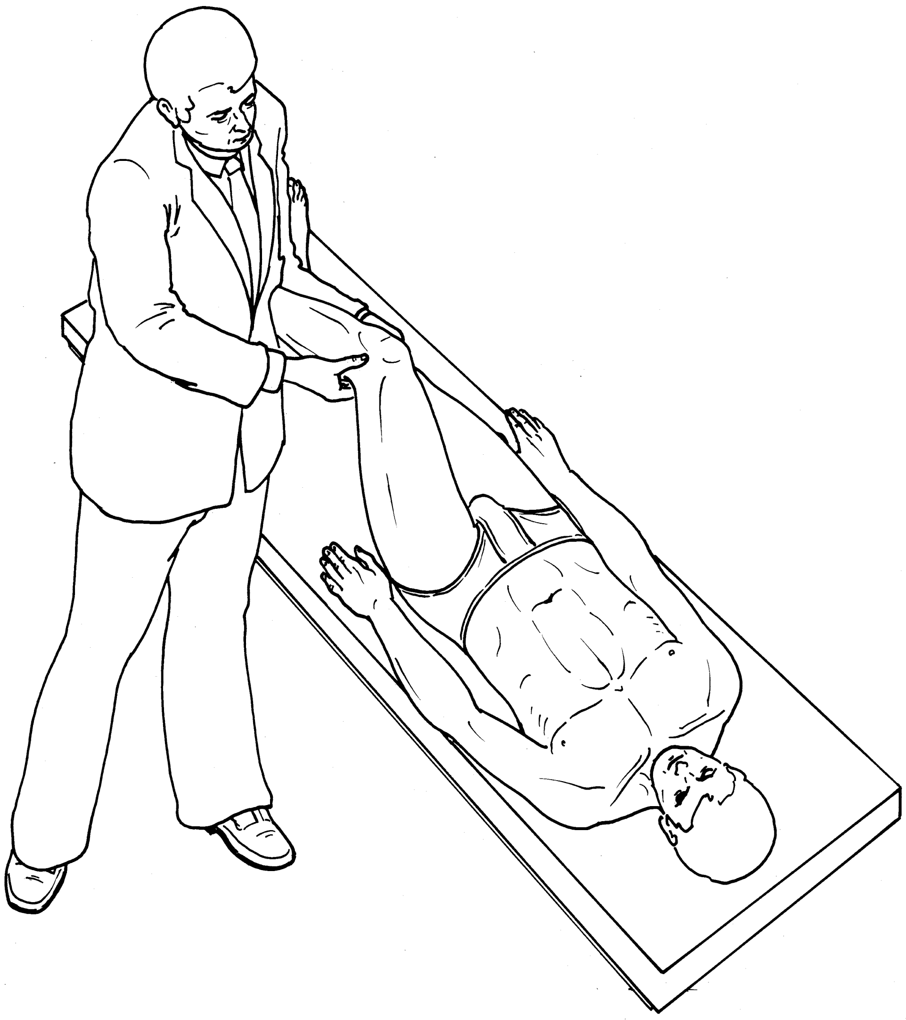


Fig. 6.54 Noyes test.

Jakob Giving Way Test

□ **Procedure:** The patient leans against the wall on the normal side and distributes his or her body weight over both legs. The examiner places one hand proximal and one hand distal to the injured knee and applies a valgus stress while the patient flexes the knee.

□ **Assessment:** The test is positive when anterior subluxation of the tibial head occurs and the patient reports a subjective sensation of the knee "giving way."

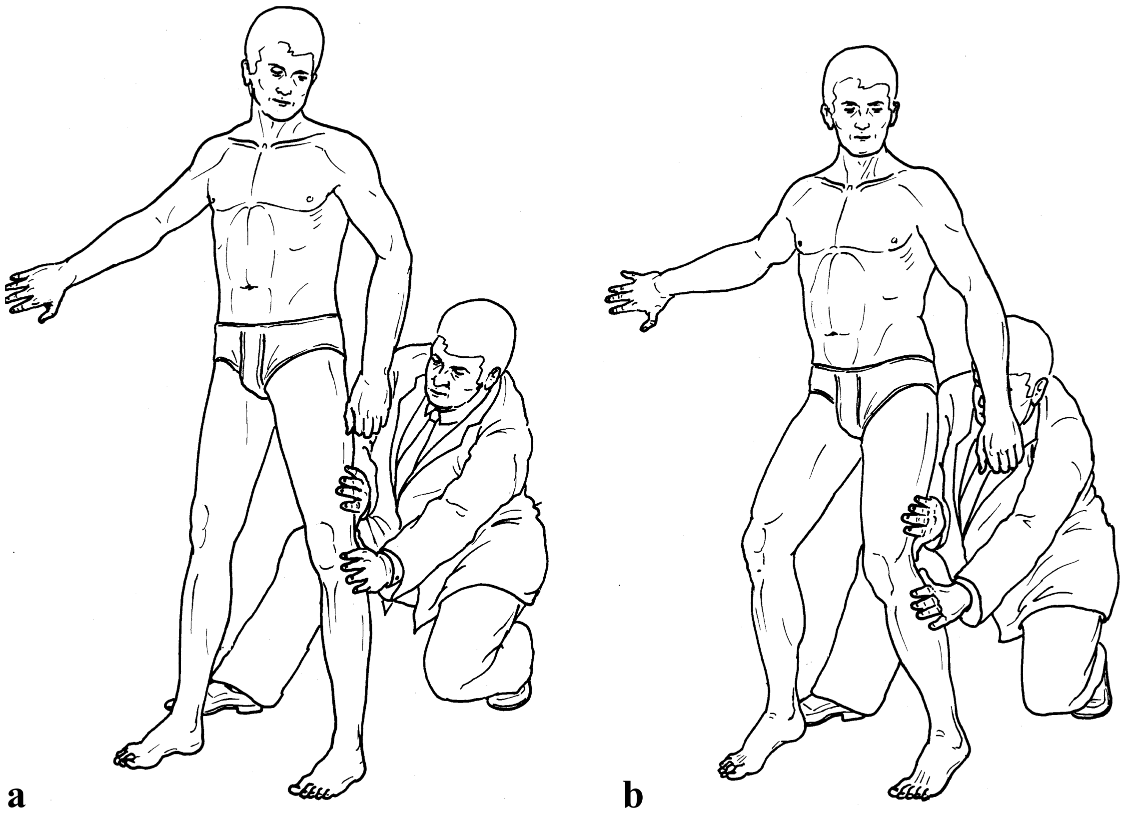


Fig. 6.55 Jakob giving way test. **(a)** Starting position with valgus stress. **(b)** Reduction in flexion while maintaining valgus stress.

Lemaire Test

□ **Procedure:** The patient is supine. The examiner internally rotates the patient's foot with one hand while pressing against the lateral thigh with the other hand, which rests proximal to the lateral femoral condyle. The examiner then carefully extends and flexes the knee.

□ **Assessment:** In an anterior cruciate ligament tear, the examiner will observe anterior subluxation of the lateral tibial head as the knee approaches extension. Spontaneous reduction will then occur at 30 to 50° of flexion.

□ **Note:** This test method was described first by Lemaire and subsequently by Galway and McIntosh; it is often referred to by the latter names.

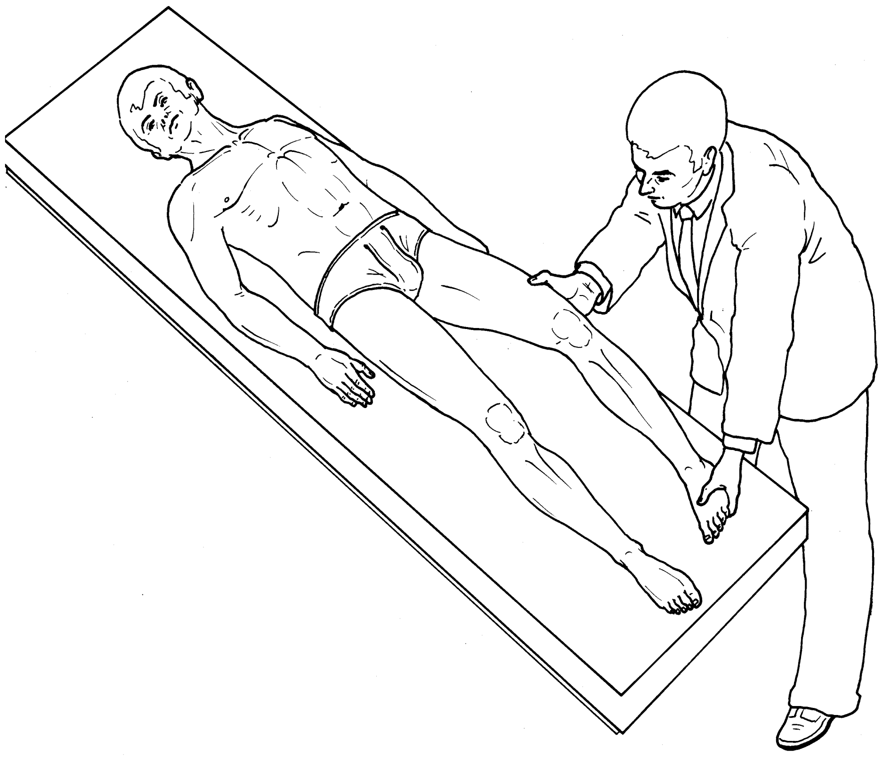


Fig. 6.56 Lemaire test.

Hughston Jerk Test

□ **Procedure:** The patient is supine with the knee flexed 60 to 70°. The examiner grasps the patient's foot with one hand and internally rotates the lower leg while applying a valgus stress with the other hand.

□ **Assessment:** The flexed knee is extended with the tibia in slight internal rotation. In an anterior cruciate ligament tear, the lateral portion of the tibial head will abruptly subluxate anteriorly at about 20° of flexion.

□ **Note:** A positive jerk test indicates that the same structures are injured as indicated by a positive pivot shift maneuver and assesses anterolateral rotatory instability. This test is not as sensitive as the pivot shift test.

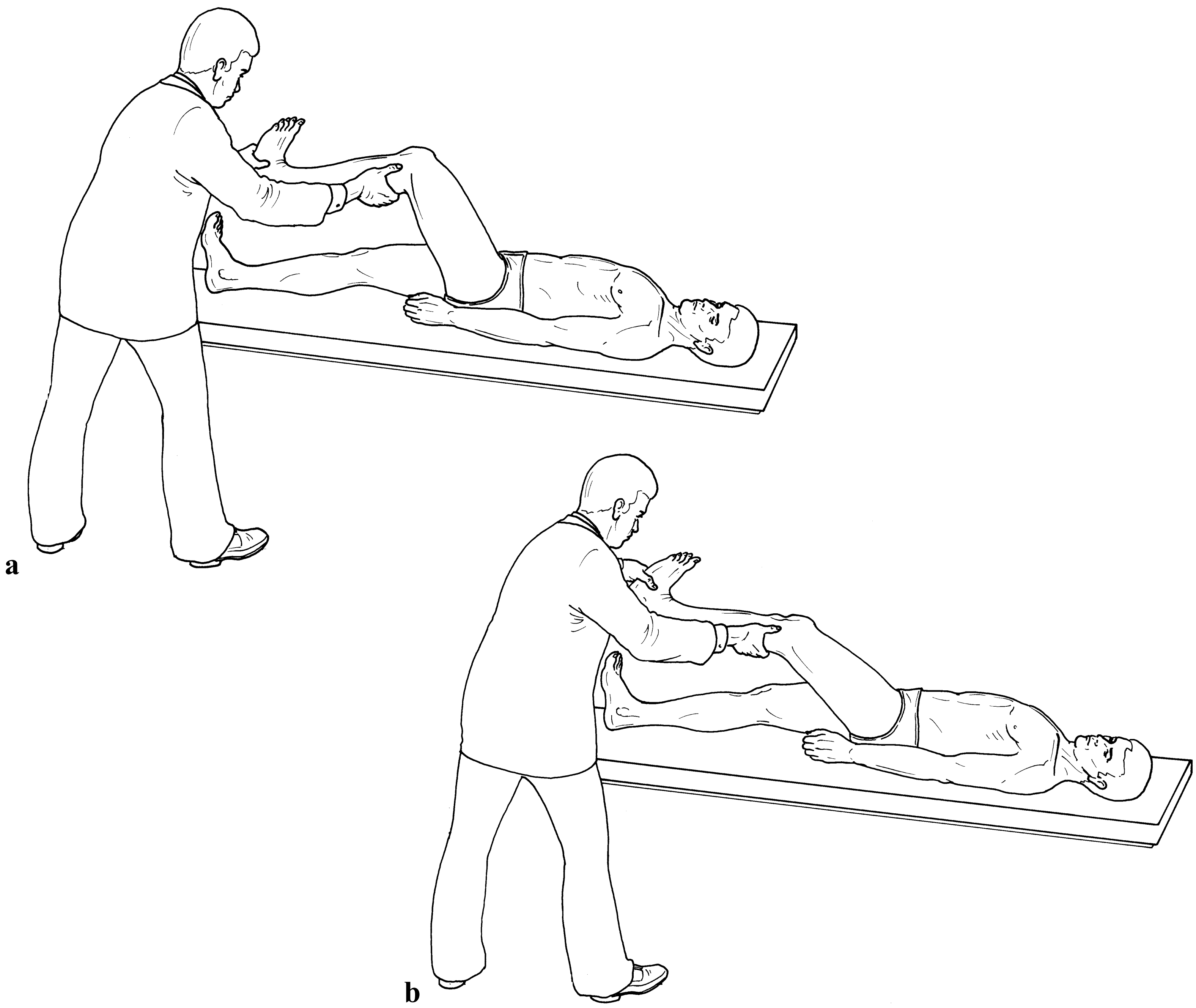


Fig. 6.57 Hughston jerk test. **(a)** Starting position with knee flexed 70°, lower leg internally rotated, and valgus stress applied. **(b)** Anterior subluxation of the lateral tibial head at 20° of flexion with the lower leg internally rotated and valgus stress applied.

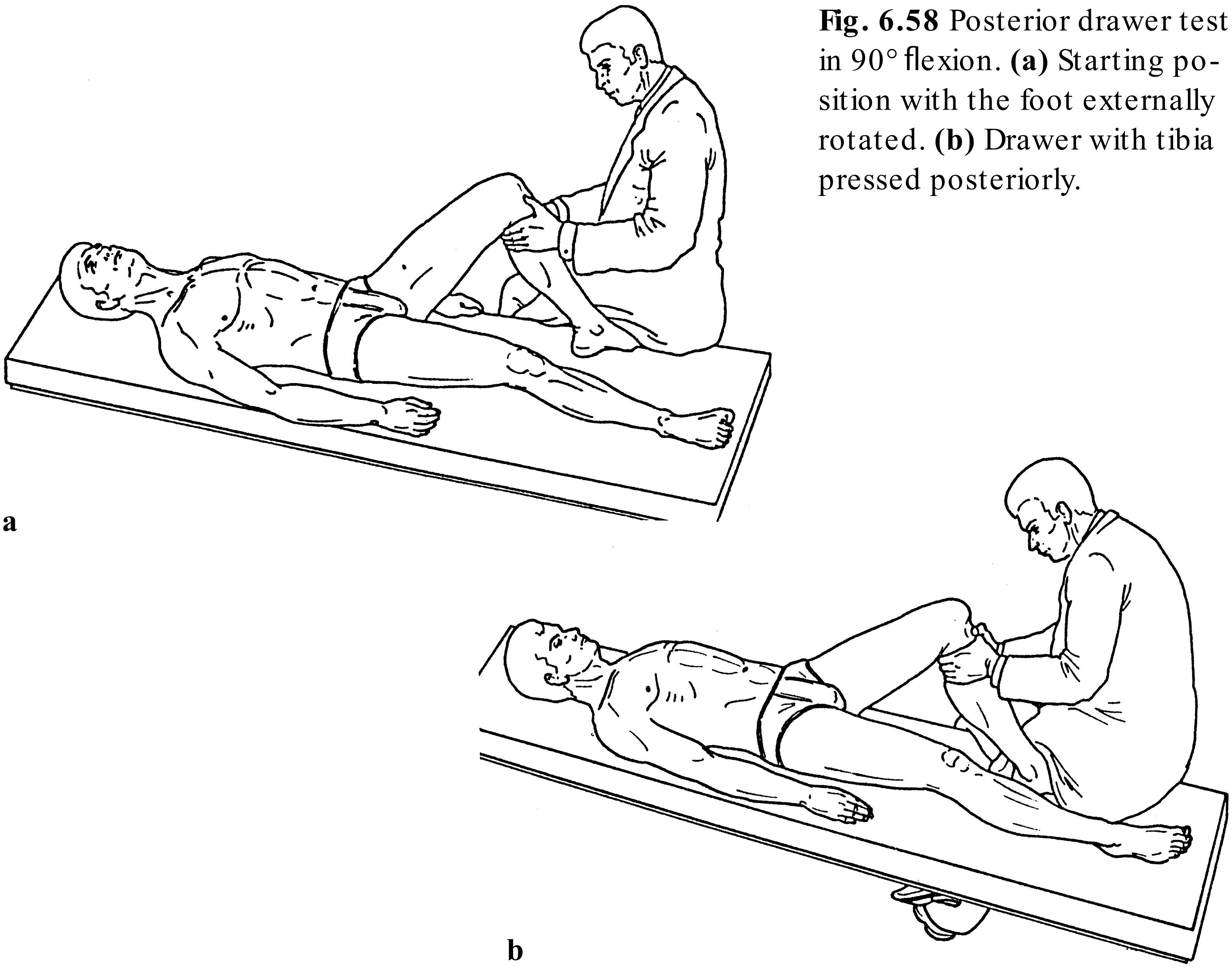
Function Tests to Assess the Posterior Cruciate Ligament

Posterior Drawer Test in 90° Flexion (Posterior Lachman Test)

□ **Procedure:** The posterior drawer test is performed with the knee in flexion and in a position approaching extension. It is similar to the anterior drawer test except that it is used to evaluate posterior translation in neutral, internal, and external rotation of the tibia.

□ **Assessment:** Isolated posterolateral instability exhibits maximum posterior translation with the knee in a position approaching extension. Maximum posterolateral rotation and minimum posterior drawer are observed with the knee in 90° of flexion. In an isolated posterior cruciate ligament injury, maximum posterior translation occurs in flexion, and posterolateral translation will be observed neither in flexion nor with the knee in a position approaching extension.

Where there is combined insufficiency of the posterior cruciate ligament and the posterolateral structures, an increased posterior drawer, external rotation, and lateral opening will be observed in all degrees of flexion.



a

b

Fig. 6.58 Posterior drawer test in 90° flexion. **(a)** Starting position with the foot externally rotated. **(b)** Drawer with tibia pressed posteriorly.

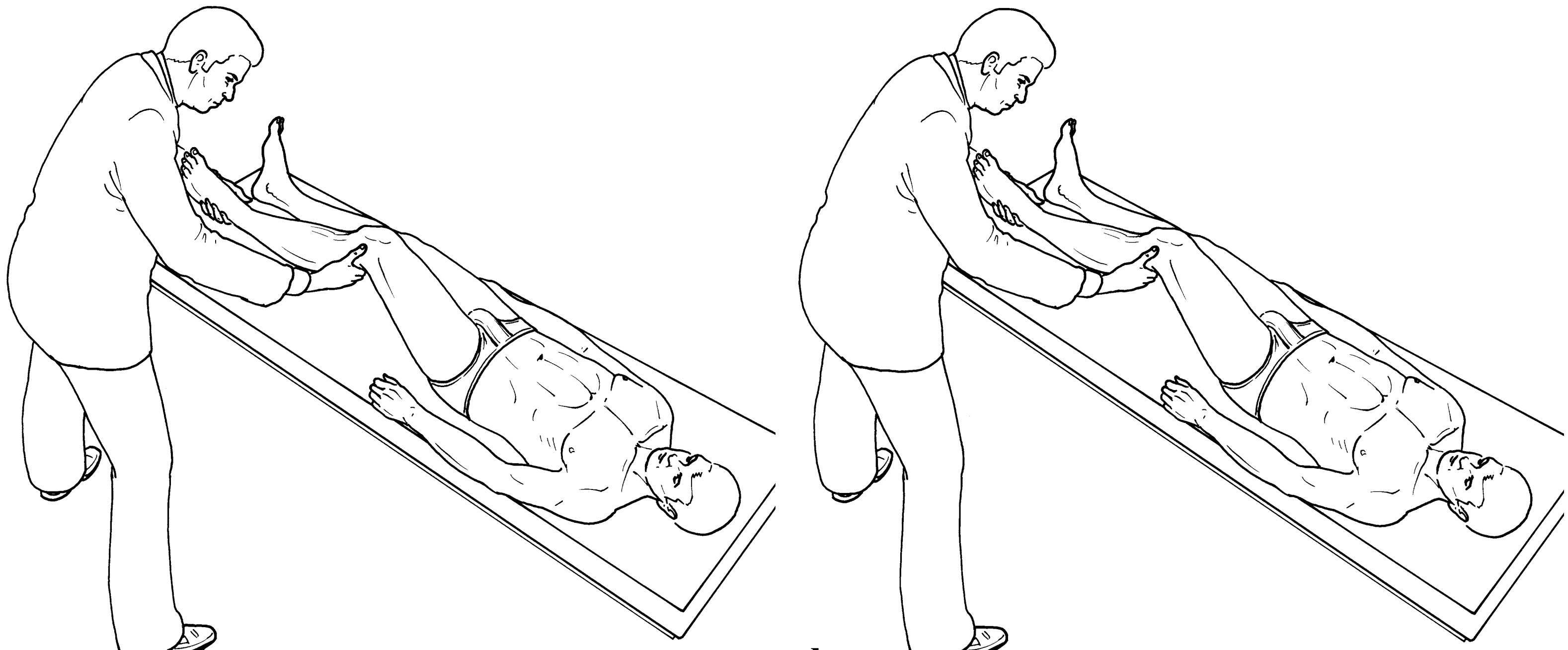
Reversed Jakob Pivot Shift Test

Assesses posterolateral rotational instability.

□ **Procedure:** The patient is supine. The examiner stands on the side of the injured leg. With one hand, the examiner grasps the patient's foot while the other hand supports the lateral aspect of the lower leg at the level of the knee. The thumb of this hand palpates the fibular head and applies valgus pressure. The examiner now flexes the patient's knee 70 to 80°. Externally rotating the foot in this position causes posterior subluxation of the lateral tibial plateau. The examiner then slowly extends the knee while maintaining slight valgus stress.

□ **Assessment:** In the presence of a posterolateral injury with the knee flexed, the tibia follows gravity and drops into posterolateral subluxation. Externally rotating the tibia increases this subluxation. As the knee is then extended and passes through 30 to 20° of flexion, the iliotibial tract begins to act as an extensor and reduces the joint. The posterolateral capsule, the posterior soft tissue envelope of the knee, and the quadriceps also contribute to the reduction.

□ **Note:** This test is the functional counterpart of the dynamic anterior subluxation test. However, it can be positive in patients with increased generalized laxity of the ligaments. This test is only clinically significant when a positive result can be elicited unilaterally and faithfully reproduces the painful subluxation symptoms described by the patient. A positive test primarily suggests a posterolateral capsular ligament injury. Injury to the posterior cruciate ligament is likely in patients with a history of trauma and simultaneous posterolateral instability in the form of a positive posterior drawer when the lower leg is in external rotation.



a b

Fig. 6.59 Reversed Jakob pivot shift test. **(a)** Posterior subluxation of the tibia with knee flexed more than 60°. **(b)** Reduction as the knee approaches extension.

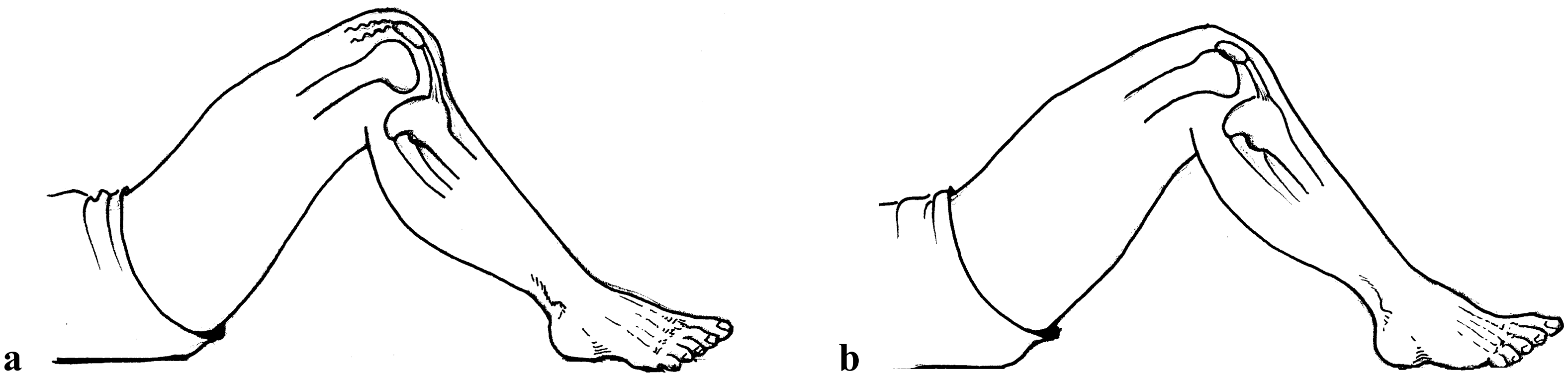
6

Quadriceps Contraction Test

Assesses a posterior cruciate ligament injury.

- **Procedure:** The patient is supine. The injured leg is flexed 90° at the knee and placed in external rotation. The patient is asked to tense the quadriceps and lift the leg off the examining table.
- **Assessment:** In the presence of posterolateral instability, the external rotation of the foot causes posterior subluxation of the lateral tibia relative to the lateral femoral condyle. The examiner observes this as a posterior droop of the lateral tibial plateau. The active quadriceps contraction and increasing knee extension cause the lateral tibial plateau to move anteriorly out of posterior subluxation and into reduction with a sort of reverse pivot shift. The joint reduces at about 30 to 20° of flexion. This test is also called an active reduction test and can usually be demonstrated only in the presence of chronic ligament injuries.

6



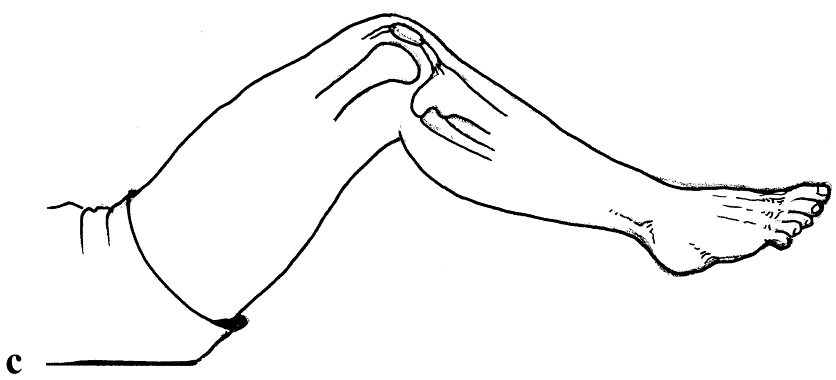


Fig. 6.60 Quadriceps contraction test. **(a)** Subluxation with posterior droop. **(b)** Tensing the quadriceps. **(c)** Active knee extension: reduction position.

Posterior Sag Sign

- **Procedure:** The patient lies in the supine position with the hips flexed at 45° and knees flexed at 90°.
- **Assessment:** Inspecting the silhouettes of both tibial heads from the side reveals that the tibial head in the affected knee appears to “sag.” The rest position of the posterior drawer is influenced by gravity and is a sensitive sign of a posterior cruciate ligament injury.

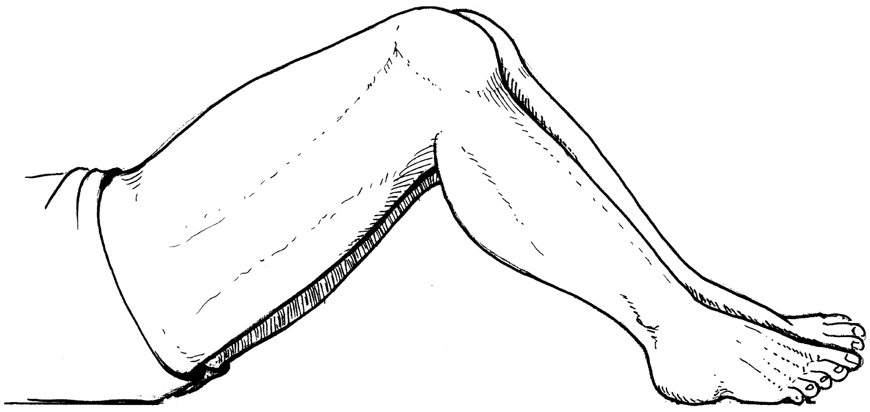


Fig. 6.61 Posterior sag sign.

Soft Posterolateral Drawer Test

□ **Procedure:** The patient sits with the legs relaxed and hanging over the edge of the table. The foot of the affected leg rests lightly on the thigh of the examiner, who crouches in front of the patient. The examiner grasps the tibial head with both hands and presses it posteriorly with the balls of the thumbs.

□ **Assessment:** Posterior translation (drawer motion) of the lateral tibial plateau is a sign of posterolateral instability.

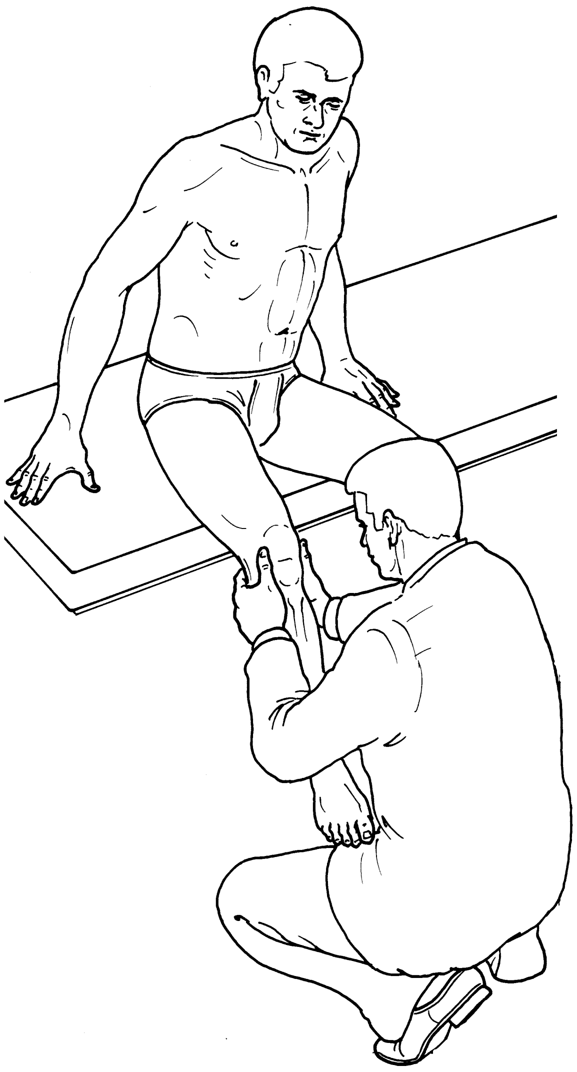
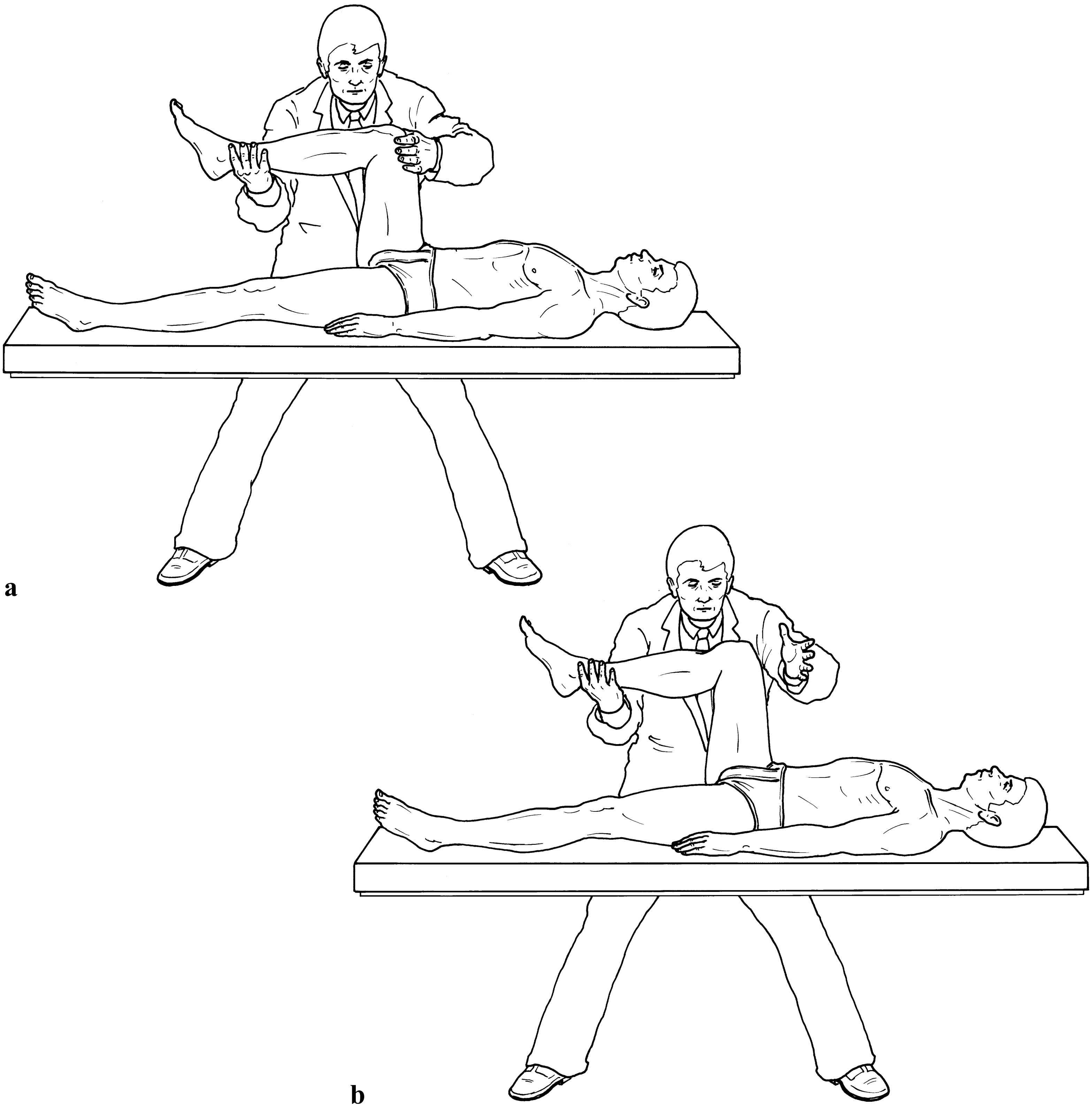


Fig. 6.62 Soft posterolateral drawer test.

Gravity Sign and Genu Recurvatum Test

□ **Procedure:** The patient lies supine with the hip and knee of the affected leg flexed 90°. With one hand, the examiner grasps the patient's lower leg while stabilizing the knee proximal to the patella with the other hand. The examiner then abruptly pulls away the stabilizing hand from the knee.
□ **Assessment:** If the posterior cruciate ligament is torn, the tibia will recede posteriorly (posterior droop).
□ **Note:** In the genu recurvatum test, the extended leg is lifted. A torn posterior cruciate ligament will result in a posterior droop of the tibia.



a

b

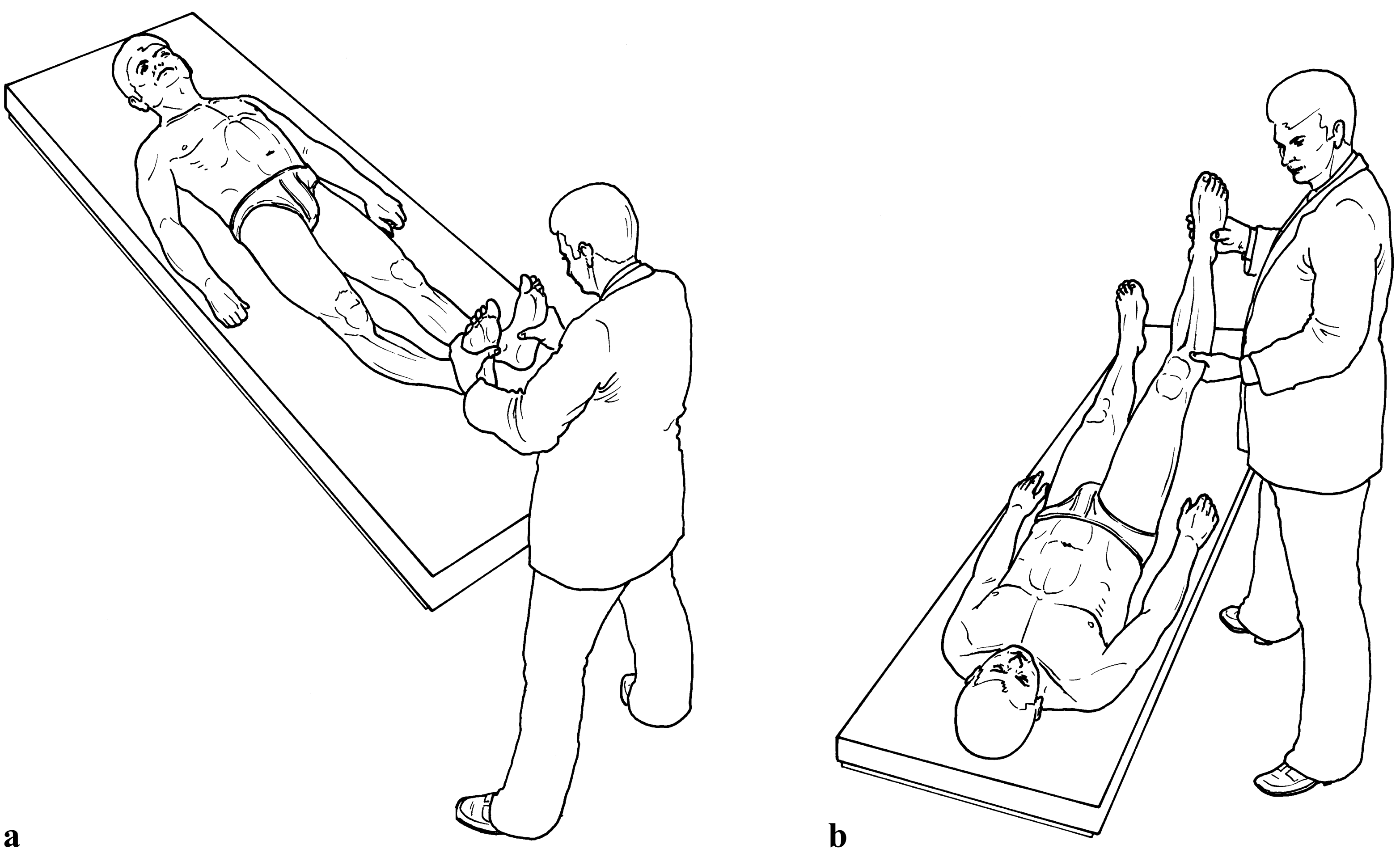
Fig. 6.63 Gravity sign and genu recurvatum test. **(a)** Stabilizing the joint. **(b)** Posterior droop of the tibia after removal of stabilization.

Hughston Test for Genu Recurvatum and External Rotation

□ **Procedure:** The patient lies supine with both quadriceps completely relaxed. The examiner then lifts each forefoot.

□ **Assessment:** In posterolateral instability, this maneuver will produce a hyperextended varus position in the knee with simultaneous external rotation of the tibia.

□ **Note:** To demonstrate the external rotation and genu recurvatum deformity (hyperextension) more clearly, the test may be performed on one leg at a time. This is done by moving the knee from slight flexion into extension. The examiner places one hand on the posterior aspect of the knee to palpate the posterior droop and the slight external rotation of the proximal tibia.



a b

Fig. 6.64 Hughston test for genu recurvatum and external rotation. **(a)** Hyperextended varus position. **(b)** Flexion into extension movement.

Godfrey Test

□ **Procedure:** The patient lies supine with both knees and both hips flexed 90°. The examiner holds the patient's lower legs while pressing the tibial tuberosity of the injured knee posteriorly.

□ **Assessment:** Even in the starting position, the examiner will readily notice the slight posterior droop in the proximal tibia indicative of posterior cruciate ligament insufficiency. Applying pressure to the anterior tibia increases the posterior droop of the lateral tibial plateau.

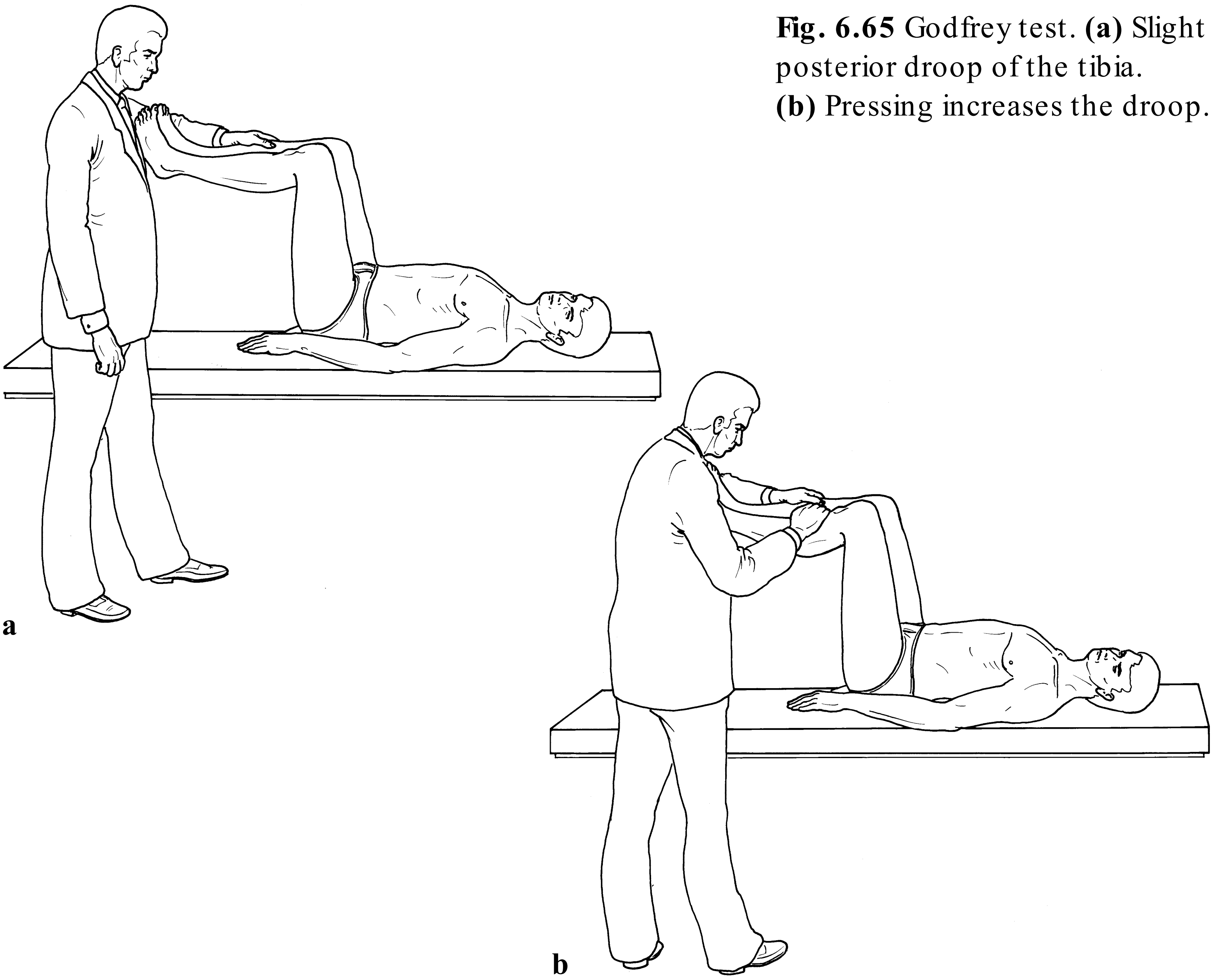


Fig. 6.65 Godfrey test. **(a)** Slight posterior droop of the tibia. **(b)** Pressing increases the droop.

Dynamic Posterior Shift Test

□ **Procedure:** The patient is supine. The examiner passively flexes both the hip and the knee of the affected leg to 90°, holding the knee in neutral rotation. One of the examiner's hands rests on the thigh and acts as a buttress while the examiner slowly extends the knee with the other hand.

□ **Assessment:** Once the knee reaches about 20° of flexion, the examiner will be able to observe and palpate an abrupt movement of the tibial plateau out of posterior subluxation into reduction and external rotation.

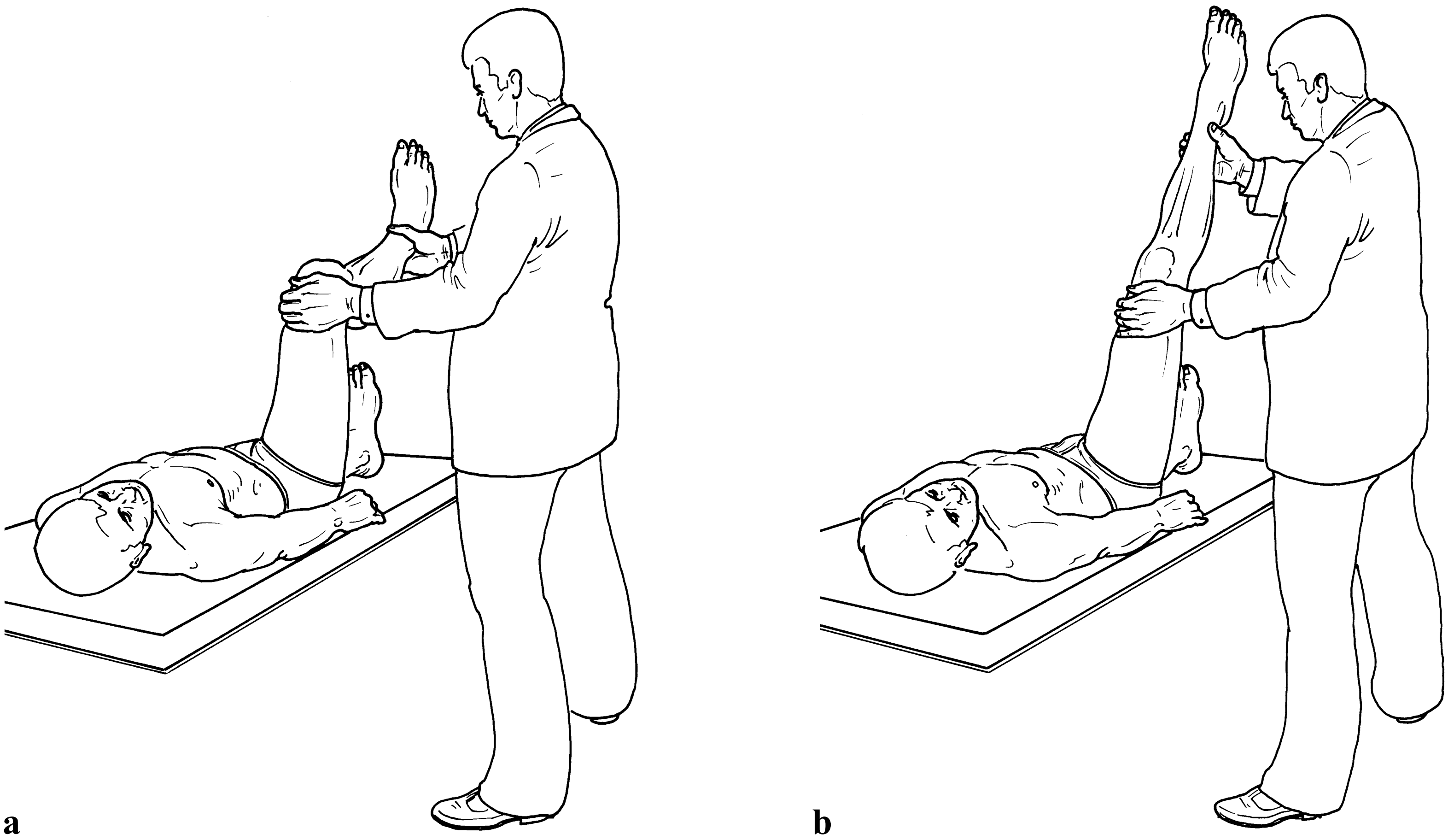


Fig. 6.66 Dynamic posterior shift test. **(a)** Subluxation with hip and knee flexed 90°. **(b)** Reduction as the knee approaches extension.

Loomer Posterolateral Rotatory Instability Test

□ **Procedure:** The patient lies supine and flexes both hips and both knees to 90°. The examiner then grasps the feet and maximally externally rotates both tibias.

□ **Assessment:** The test is considered positive for posterolateral instability if the external rotation of the injured tibia is excessive when compared with the other leg and there is posterior sag of the affected tibial tubercle.

□ **Note:** Other authors describe a modification of the Loomer test that is called the tibial external rotation test or **dial test**. The patient lies supine. The examiner flexes the knee to 30°, and from this positon extends the foot over the side of the examining table. The examiner then externally rotates the tibia on the femur and compares the amount of rotation to that on the good side, while observing the movement of the tibial tuberosity. The test is then repeated with the knee flexed to 90°. If the tibia rotates less at 90° than at 30°, an isolated posterolateral (popliteus corner) injury is probable. If the knee externally rotates more at 90°, one can assume there is injury to both the popliteus corner and posterior cruciate ligament.

Fig. 6.67 Loomer posterolateral rotatory instability test.

Iliotibial Tract

Noble Compression Test

Assesses an iliotibial tract syndrome.

□ **Procedure:** The patient lies supine with the hip flexed at about 50° and the knee at 90°. The examiner presses with the thumbs on the lateral femoral condyle or 1 to 2 cm proximal to it while passively extending the knee.

□ **Assessment:** If pain occurs over the lateral femoral condyle, especially at a knee flexion of 30°, this implies an iliotibial tract syndrome. The character of the pain is usually the same as what the patient feels during athletic activity.

□ **Note:** The iliotibial tract syndrome (iliotibial friction syndrome) is also known as runner's knee. It is an insertion tendinopathy from repeated rubbing and inflammation of the iliotibial tract, especially in long-distance runners.

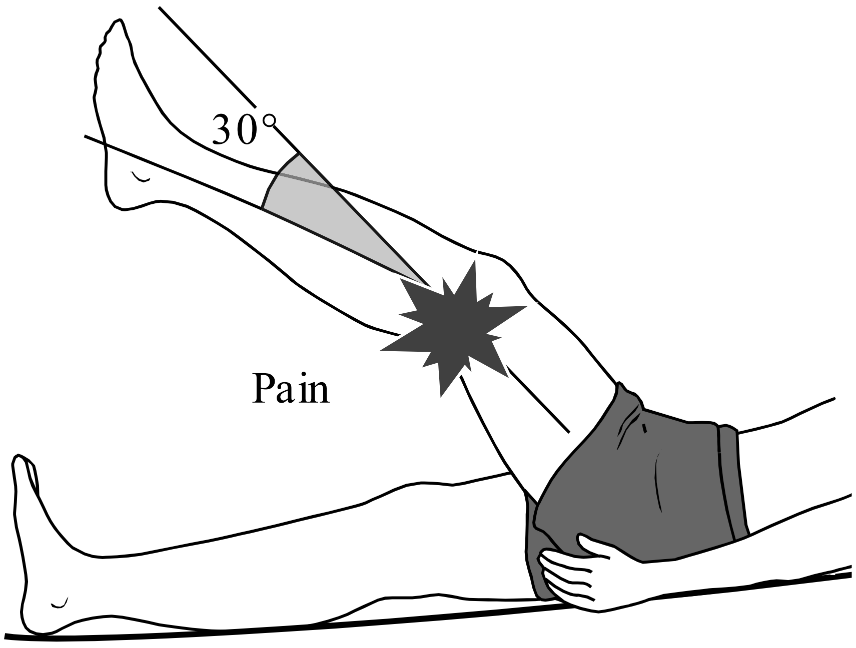


Fig. 6.68 Noble compression test.

7 Foot and Ankle

For humans, the foot and ankle have a unique function: they serve to propel us and also serve as support. For propulsion they act like a flexible lever. For support they form a rigid structure that holds up the entire body.

Disorders of the foot and ankle can lead to changes in gait and thereby affect other joints of the lower extremity. More than 80% of the populace have foot problems. When assessing the lower extremity, including the foot and ankle, it is especially important to assess the neutral position of the foot in both weight-bearing and non–weight-bearing situations. This will help the examiner to differentiate functional from structural deformities.

Almost all patients who come to the practice with foot problems have pain. An exact history is therefore very important in establishing the correct diagnosis. Age, sex, occupation, and leisure activities are factors to be considered in every patient. It is important to inquire about the character of the onset of pain, its location, radiation and nature, and about factors that cause the pain. It is important to examine the feet and adjacent joints, such as the knee, in a side-to-side comparison. One should also consider axial deviations in the legs. Inspection of the shape and soles of the patient's shoes is important—asymmetric wear on the soles may provide an initial indication of the cause of the patient's complaints.

In addition to a palpatory examination with assessment of mobility and tenderness to palpation in the affected region, it is important to observe the foot during weight bearing and walking. Metatarsalgia is a general term for pain in the forefoot. *It is not in and of itself a clinical syndrome, but rather a pain caused not only by regional disorders and biomechanical functional disturbances but often by systemic diseases such as gout, rheumatoid arthritis, and circulatory disorders as well.* Splayfoot is the most common deformity of the foot and the most common cause of metatarsalgia. The collapse of the transverse metatarsal arch as a result of weakness of the muscles and ligaments leads to secondary changes in the foot with the development of claw toe and hammer toe deformities and of hallux valgus. Plantar calluses from the increased stresses on the metatarsal heads in turn lead to additional problems. Other causes of forefoot pain include osteoarthritis (hallux rigidus), neuromas

7

Range of Motion in the Ankle and Foot (Neutral-Zero Method)

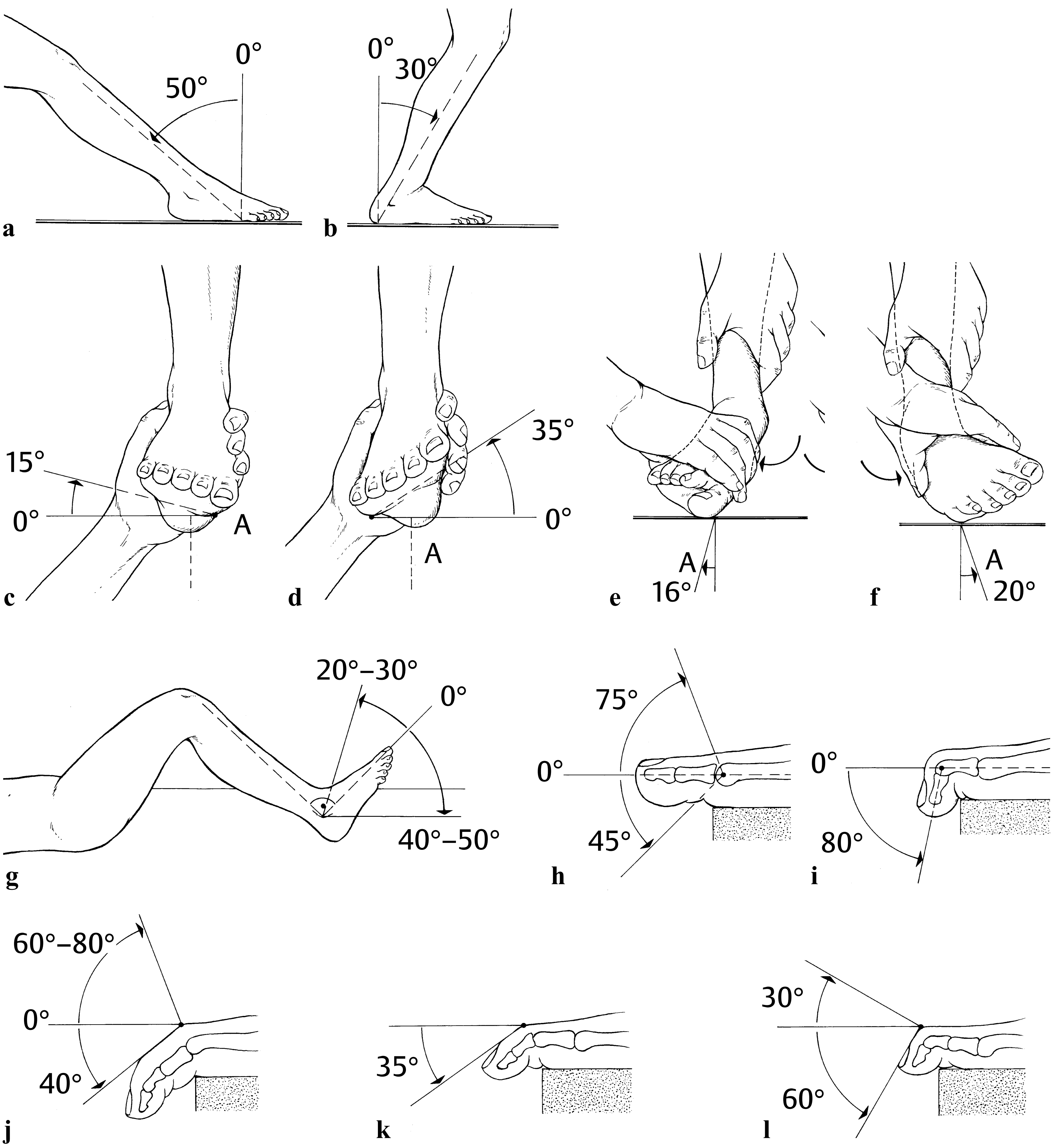
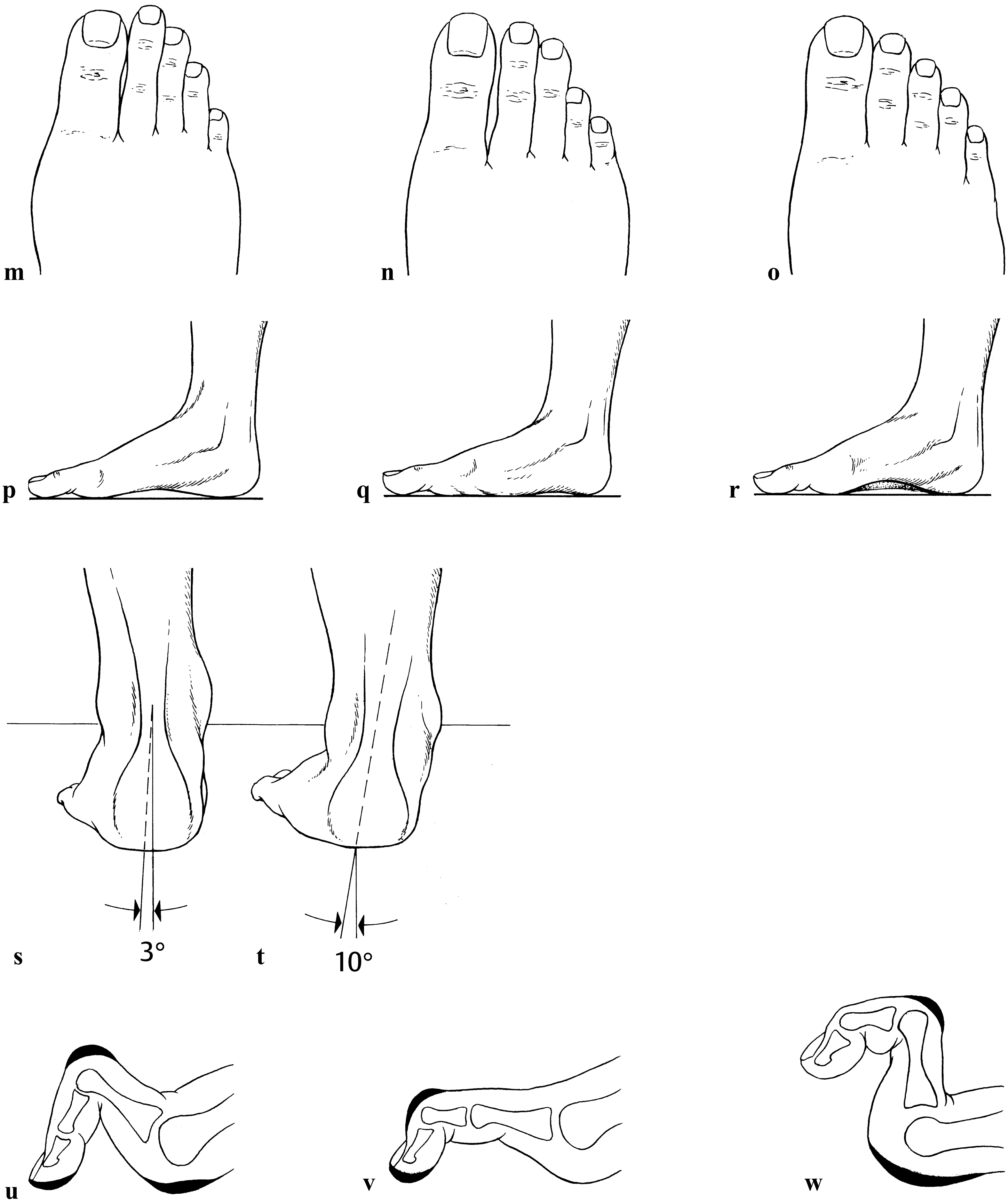


Fig. 7.1 (a,b) Plantar flexion **(a)** and dorsiflexion **(b)** of the foot when standing. **(c,d)** Pronation **(c)** and supination **(d)** of the forefoot. One hand grasps the heel and the other turns the forefoot. Only the angle of the forefoot relative to the hindfoot is measured as pronation and supination. **(e,f)** Eversion **(e)** and inversion **(f)** of the hindfoot. One hand grasps the lower leg and the other grasps the posterior aspect of the forefoot, holding the calcaneus between thumb and forefinger (not shown). The inversion and eversion are evaluated on the calcaneus (axis of the calcaneus, A). Care should be taken to avoid pronation or supination of the foot. **(g)** Plantar flexion and dorsiflexion of the ankle (talocrural joint) with the



foot hanging relaxed. **(h–l)** Motion in the metatarsophalangeal joints: great toe **(h,i)**, other toes **(j–l)**. **(m–o)** The most common variations in forefoot and toe length: Greek **(m)**, square **(n)**, and Egyptian as described by Lelièvre **(o)**. **(p–r)** Assessment of the medial longitudinal arch of the foot: normal arch rising slightly above the floor **(p)**, absent arch or flatfoot **(q)**, abnormally high arch or pes cavus **(r)**. **(s,t)** Assessment of the position of the hindfoot. Normal position is a valgus angle of 0 to 6°. A valgus angle exceeding 6° is pes valgus; any varus angle is pes varus. **(u–w)** The most important toe deformities: hammer toe in the proximal interphalangeal joint **(u)**, hammer toe in the distal interphalangeal joint **(v)**, claw toe as described by Lelièvre **(w)**.

7

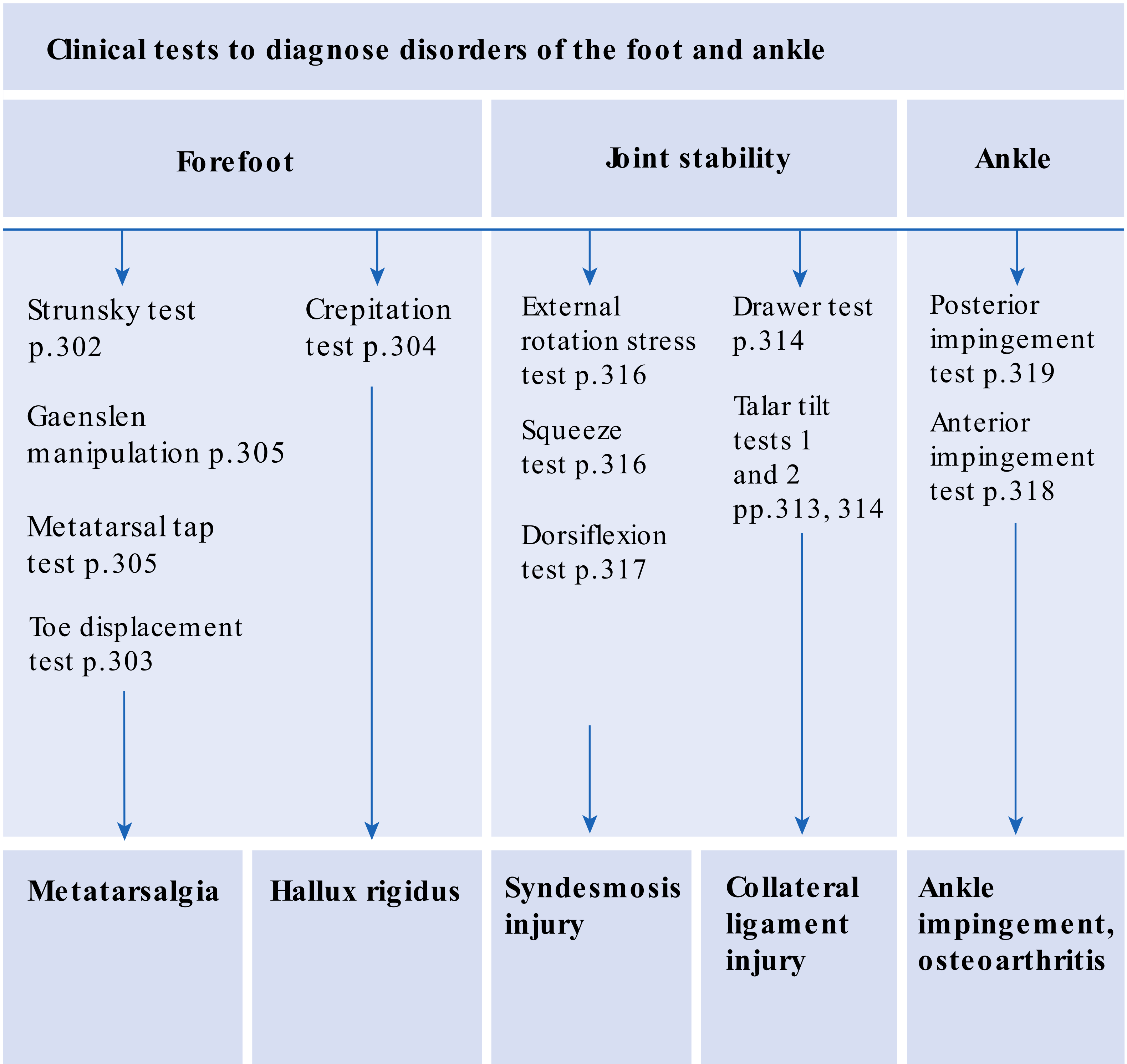
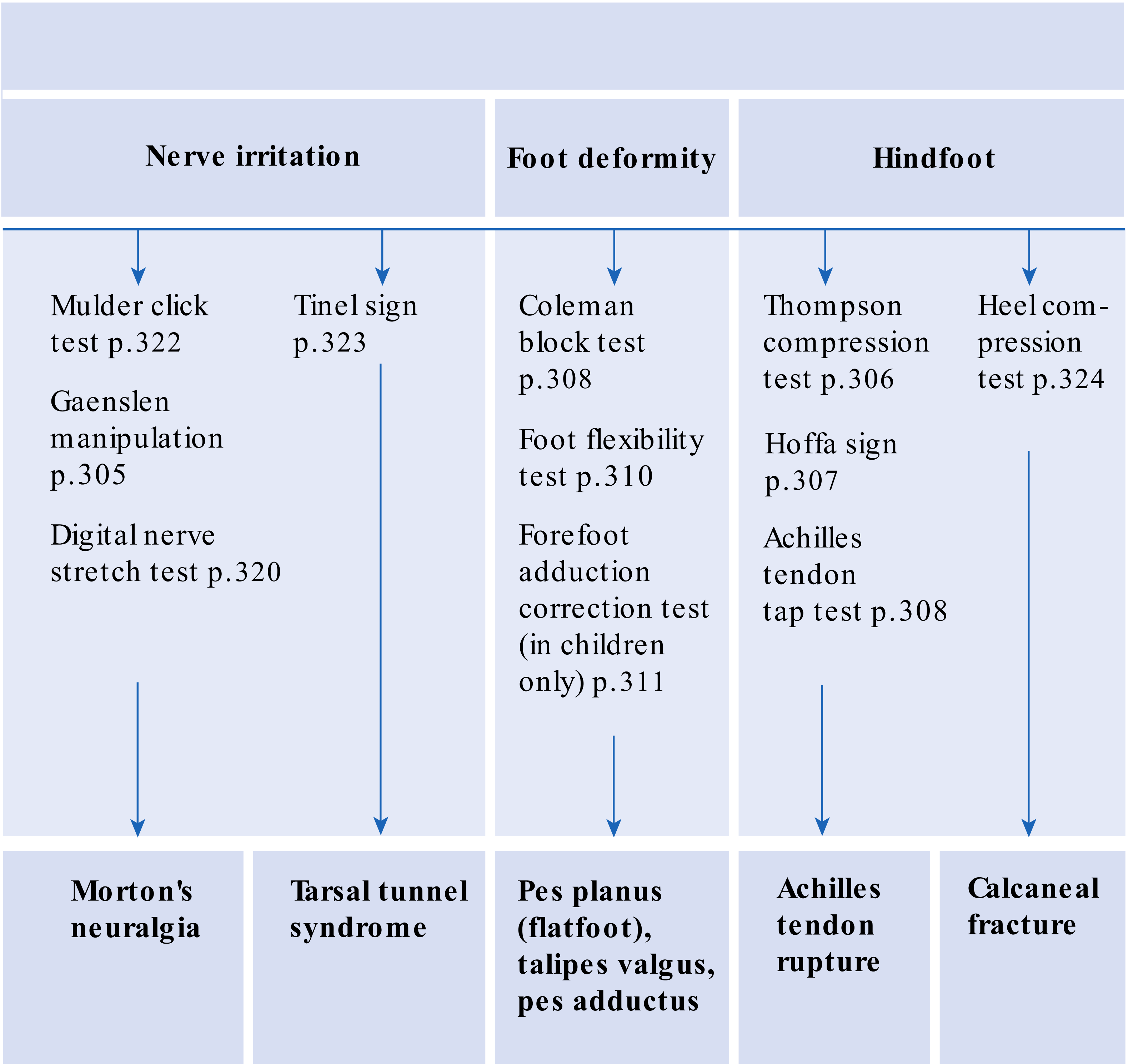


Fig. 7.2 Clinical tests to diagnose disorders of the foot and ankle.


Nerve irritation
Foot deformity
Hindfoot
Mulder click test p.322
Gaenslen manipulation p.305
Digital nerve stretch test p.320
Tinel sign p.323
Coleman block test p.308
Foot flexibility test p.310
Forefoot adduction correction test (in children only) p.311
Thompson compression test p.306
Hoffa sign p.307
Achilles tendon tap test p.308
Heel compression test p.324
Morton's neuralgia
Tarsal tunnel syndrome
Pes planus (flatfoot), talipes valgus, pes adductus
Achilles tendon rupture
Calcaneal fracture

7

(Morton's neuroma), stress fractures, avascular necrosis (Koehler's disease), disorders of the sesamoids, plantar warts, and compression neuropathies (tarsal tunnel syndrome). Certain systemic diseases tend to involve the foot, including diabetes mellitus, peripheral arterial disease, gout, psoriasis, collagen disorders, and rheumatoid arthritis.

Function Tests

Grifka Test

Assesses splayfoot.

▫ **Procedure:** After passively dorsiflexing the toes of one foot, the examiner applies distal and plantar finger pressure to longitudinally compress the metatarsal heads in the metatarsophalangeal joints.

▫ **Assessment:** This compression corresponds to the transfer of compressive forces to the metatarsal heads in the painful toe-off phase of walking. With a splayfoot, this is often painful while plantar compression alone is painless.

Fig. 7.3 Grifka test.

Strunsky Test

Provocation test to assess metatarsalgia.

▫ **Procedure:** The patient is supine with the feet extending over the edge of the examining table. Starting with a thumb medial to each of the patient's great toes, the examiner grasps all the toes in a pincer grip and forcefully plantar flexes the metatarsophalangeal joints of both feet.

□ **Assessment:** Where there is chronic irritation of the metatarsophalangeal joints with metatarsalgia, this test significantly increases symptoms as a result of the increased pressure on the metatarsophalangeal joints. Subsequent palpation of the metatarsophalangeal joints can then identify the painful joint.

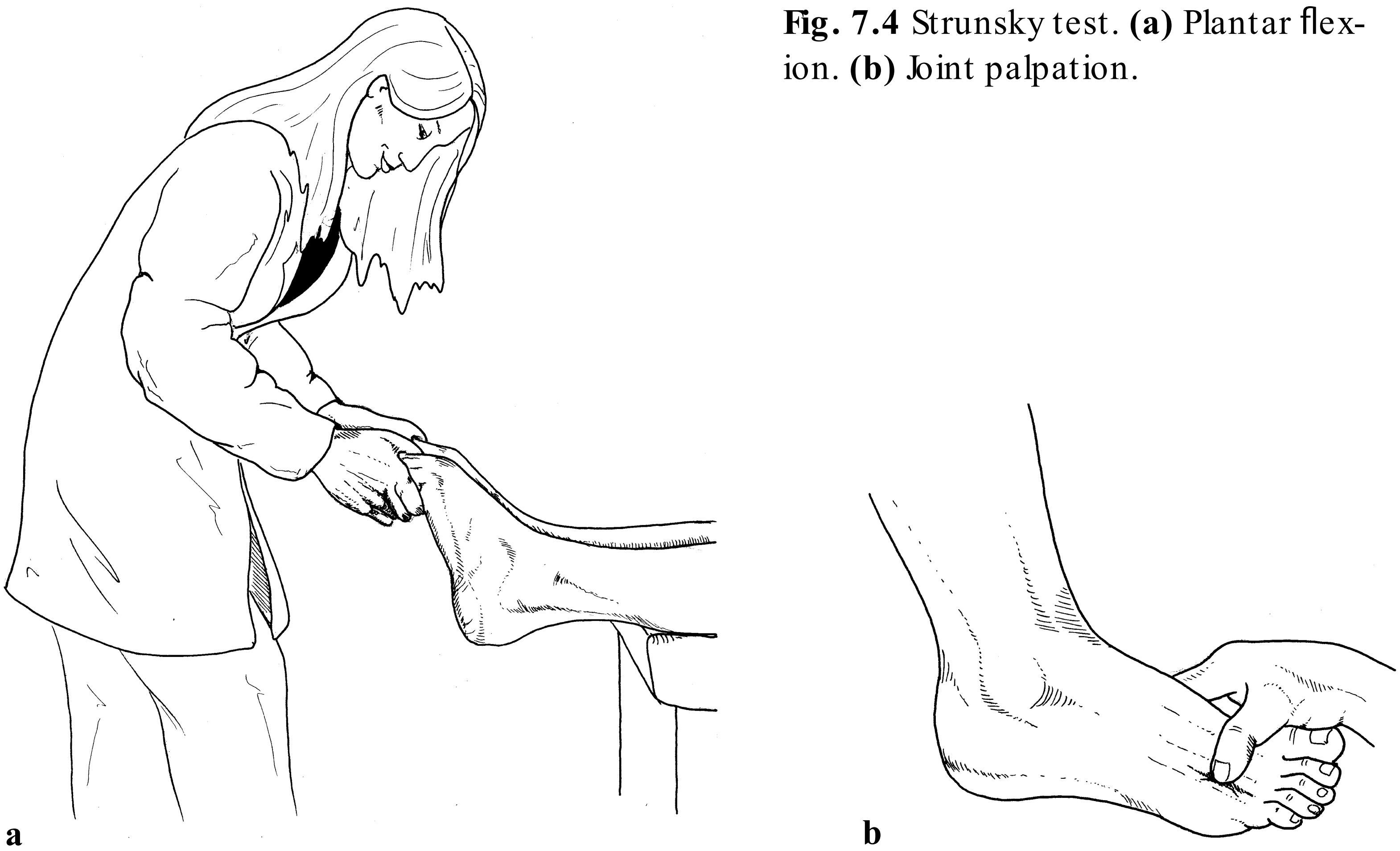


Fig. 7.4 Strunsky test. **(a)** Plantar flexion. **(b)** Joint palpation.

Toe Displacement Test

Tests instability of the metatarsophalangeal joints.

□ **Procedure:** While immobilizing the medial forefoot with one hand, the examiner grasps the distal portion of one proximal phalanx with the other hand and moves it posteriorly and plantarward relative to the metatarsal head.

□ **Assessment:** Motion pain in the metatarsophalangeal joint accompanied by signs of instability suggests an increasing deformity of the toe leading to a functional claw toe deformity during weight bearing. Progression of this instability leads to a permanent claw toe deformity with the metatarsophalangeal joint fixed in dorsiflexion.

In a dislocation of the metatarsophalangeal joint, it will be impossible to reduce the joint in the toe displacement test. The result is metatarsalgia with development of plantar calluses.

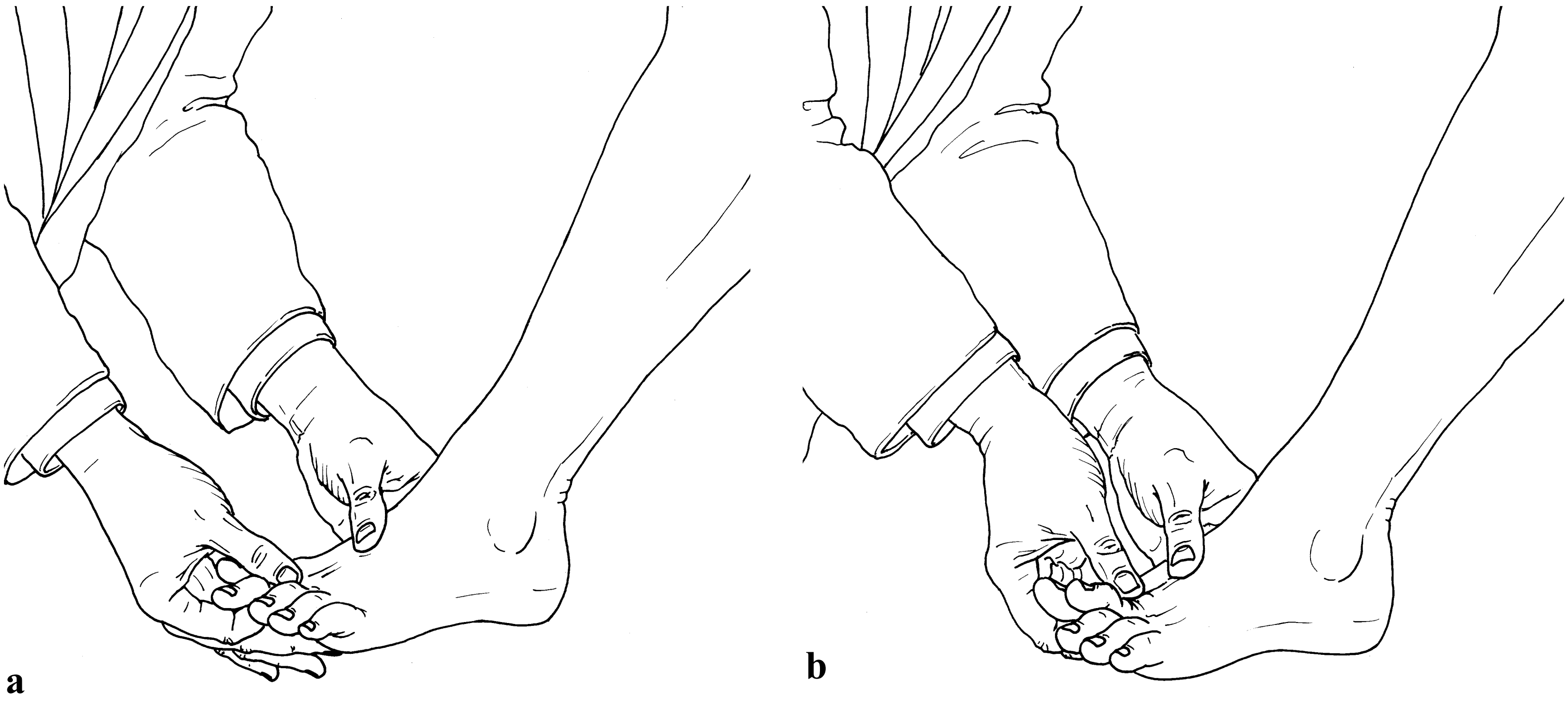


Fig. 7.5 Toe displacement test. (a) Posterior displacement. (b) Plantar displacement.

Crepitation Test

Indicates hallux rigidus.

- **Procedure:** With the patient's foot relaxed and hanging down, the examiner approaches from distally and grasps the proximal phalanx of the great toe, with the thumb on its dorsal aspect and fingers on its plantar aspect. The examiner immobilizes the lateral forefoot with the other hand, placing the thumb on its plantar aspect and the fingers on its dorsal aspect. Then the examiner passively plantar flexes, dorsiflexes, and rotates the metatarsophalangeal joint.
- **Assessment:** In hallux rigidus, joint motion in every direction will be painful and, primarily in dorsiflexion, restricted. This will be accompanied by palpable or audible crepitation as a result of osteoarthritic changes in the joint.

Fig. 7.6 Crepitation test.

Gaenslen Manipulation

Assessment of forefoot pain.

□ **Procedure:** The examiner immobilizes the metatarsal heads in one plane between the fingers of one hand on the plantar aspect of the foot and the thumb on the dorsal aspect. The other hand grasps the toes in a pincer grip, applying medial and lateral compression to the forefoot via the metatarsal heads of the great toe and little toe.

□ **Assessment:** This forefoot "pincer grip" will elicit pain between the metatarsal heads, often with acute episodic pain radiating into the adjacent toes, in the presence of a Morton's neuroma (a painful interdigital neuroma). It will also often cause pain in a significant splayfoot deformity where there is irritation of the joint capsule.

Metatarsal Tap Test

A provocation test for assessment of metatarsalgia.

□ **Procedure:** The patient is supine with the feet extending over the edge of the examining table. The examiner slightly hyperextends the toes with one hand and taps the metatarsal heads or metatarsophalangeal joints with a reflex hammer held in the other hand.

□ **Assessment:** In a patient with metatarsalgia due to chronic irritation of the metatarsophalangeal joints, tapping the ball of the foot will exacerbate the metatarsalgia symptoms. Pain upon tapping that occurs between the metatarsal heads—primarily the third and fourth metatarsals—with acute episodic pain radiating into the adjacent toes suggests a Morton's neuroma (see Mulder Click Test, p. 322).

Fig. 7.7 Gaenslen manipulation

Fig. 7.8 Metatarsal tap test.

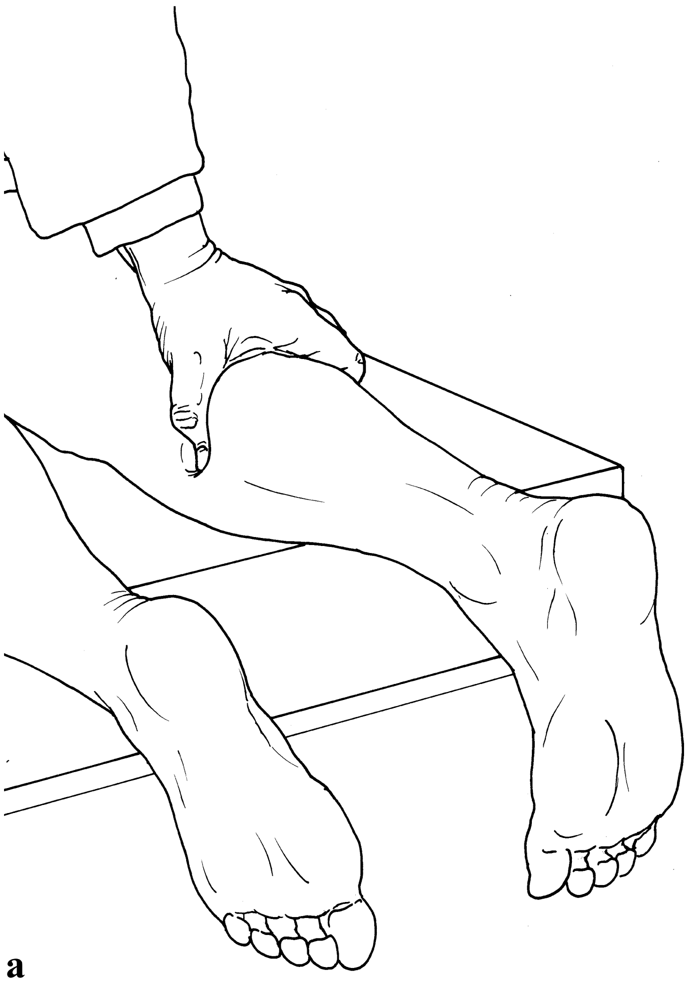
Thompson Compression Test (Calf Compression Test)

Indicates an Achilles tendon tear.

□ **Procedure:** The patient is prone with the feet projecting past the edge of the examining table. The examiner grasps the calf of the affected leg with one hand and forcefully compresses the musculature.

□ **Assessment:** Compressing the calf muscles should normally provoke rapid passive plantar flexion of the foot. Absence of this plantar flexion suggests a torn Achilles tendon. The response to the compression test is not always unambiguous in patients with partial tears and will depend on the degree of disruption. In an Achilles tendon tear, the patient will be unable to stand on tiptoe, especially when standing only on the injured leg, and the Achilles tendon reflex will be absent.

□ **Note:** The test can also be performed with the patient prone and the knee flexed 90°. In this position, the examiner grasps the patient's calf with both hands and forcefully compresses the musculature. Loss of plantar flexion is a sign of an Achilles tendon tear (**Simmonds' test**).



a

b

Fig. 7.9 Thompson compression test. **(a)** With the knee extended. **(b)** With the knee flexed 90°.

Hoffa Sign

Indicates a chronic Achilles tendon tear.

- **Procedure:** The patient is prone with the feet projecting over the edge of the examining table. The examiner passively dorsiflexes both feet.
- **Assessment:** In a chronic Achilles tendon tear, tension in the Achilles tendon will be reduced and the affected foot can be dorsiflexed farther than the contralateral foot. The patient is then requested to stand on tiptoe on each leg. This will be impossible with an Achilles tendon tear in the injured leg.

Fig. 7.10 Hoffa sign.

Achilles Tendon Tap Test

Indicates an Achilles tendon tear.

□ **Procedure:** The patient is prone with the knee flexed 90°. The examiner taps the distal third of the Achilles tendon with a reflex hammer.

□ **Assessment:** Increased pain and loss of plantar flexion (Achilles tendon reflex) are signs of a tear in the Achilles tendon. In the absence of an Achilles tendon reflex, a differential diagnosis should exclude neurologic changes.

Fig. 7.11 Achilles tendon tap test.

Coleman Block Test

Flexibility test for assessment of hindfoot deformities.

□ **Procedure:** The patient is standing. The lateral block test involves placing wooden blocks of varying height beneath the heel and the lateral margin of the foot. The blocks are placed according to the severity and shape of the foot deformity so as to allow the first metatarsal to reach the floor. In the medial block test, the wooden block must be placed beneath the first metatarsal head.

□ **Assessment:** The block test is a good method for determining the flexibility of compensatory hindfoot deformities in the presence of simultaneous fixed forefoot contractures. The lateral block test is used to determine the flexibility of a varus hindfoot deformity in the presence of a simultaneous valgus forefoot contracture. A flexible compensatory varus hindfoot deformity will be corrected by the lateral block. Where a varus forefoot contracture is present, the medial block test will allow evaluation of the flexibility and/or severity of the contracture in the hindfoot deformity.

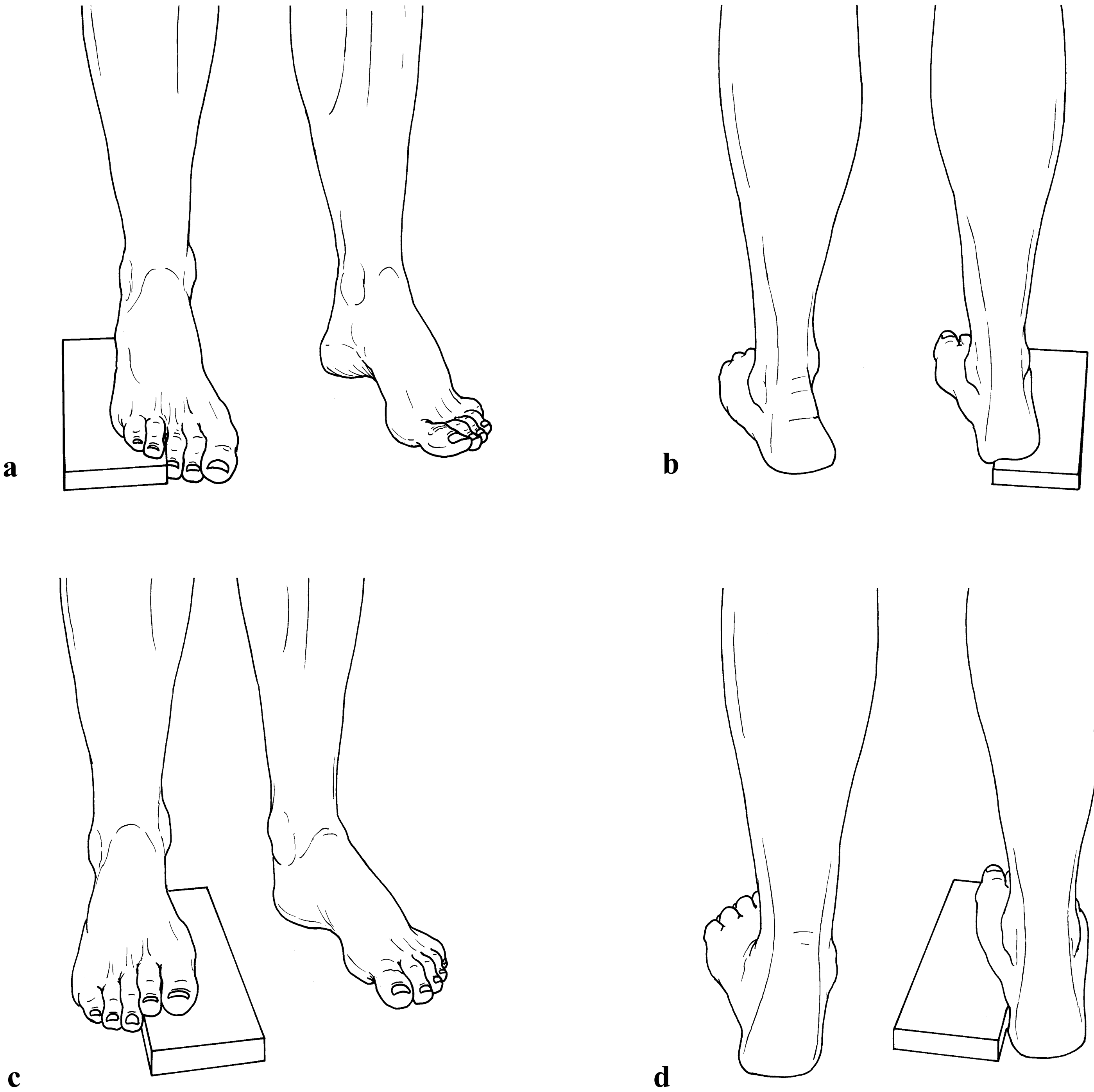


Fig. 7.12 Coleman block test. **(a)** Hindfoot varus and forefoot valgus viewed from the front. **(b)** Hindfoot varus and forefoot valgus viewed from the rear. **(c)** Hindfoot valgus and forefoot varus viewed from the front. **(d)** Hindfoot valgus and forefoot varus viewed from the rear.

Foot Flexibility Test

Assesses rigid or flexible talipes planovalgus deformity.

□ **Procedure:** Talipes planovalgus is a foot deformity in which the medial longitudinal arch of the foot is flattened (flatfoot, talipes planus, or pes planus) and the valgus position of the heel is increased (talipes valgus). The feet are examined from the side and from behind with the patient standing in the normal position and on tiptoe.

□ **Assessment:** Persistent flattening of the medial longitudinal arch and persistent valgus position of the heel when the patient stands on tiptoe indicate a rigid talipes planovalgus deformity. In a flexible talipes planovalgus deformity, the tiptoe stance will bring about a varus shift in the heel to compensate for the valgus deformity, and the medial longitudinal arch will reappear.

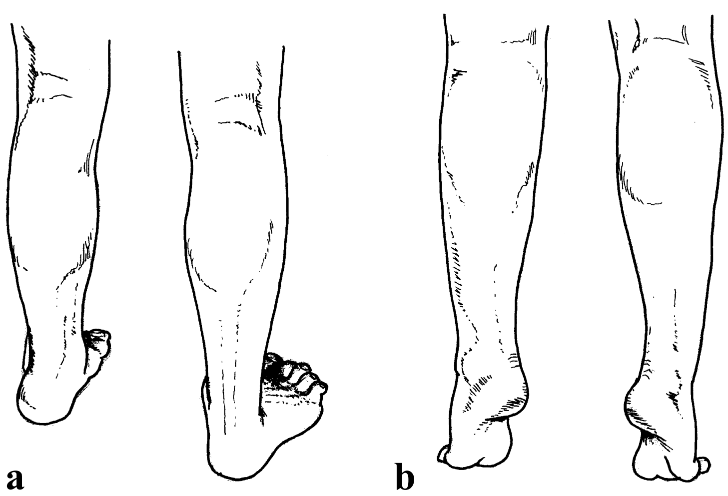
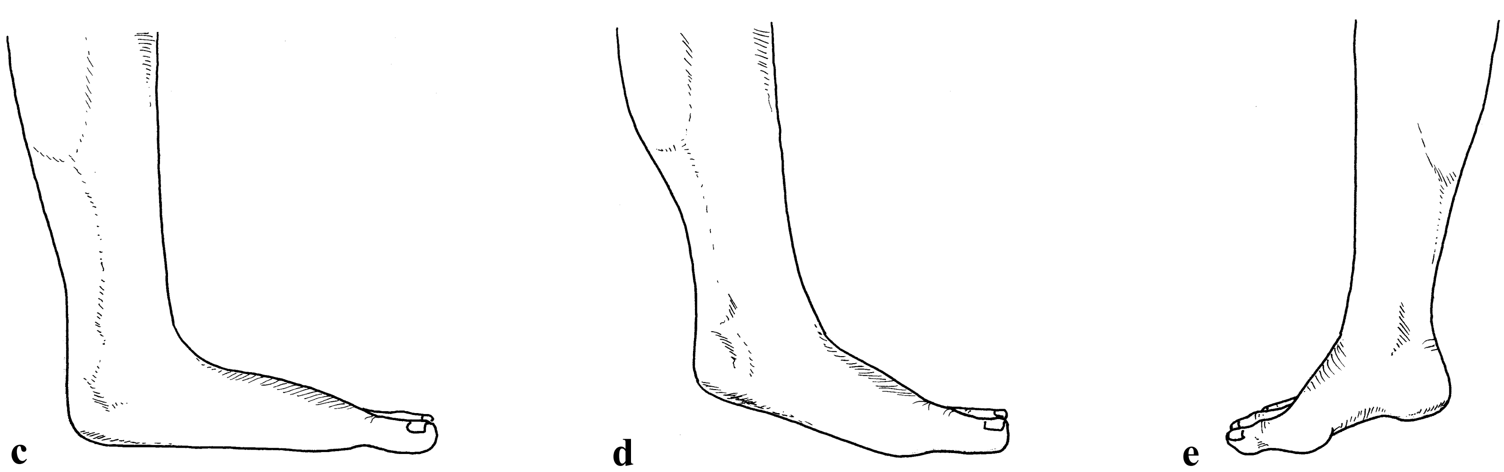


Fig. 7.13 Foot flexibility test. **(a)** Posterior view of talipes valgus. **(b)** Flexible deformity disappears in tiptoe stance. **(c)** Significant flattening of the medial longitudinal arch. **(d)** Contracture persists in tiptoe stance—persistent flatfoot. **(e)** Flexible deformity—arch reappears.

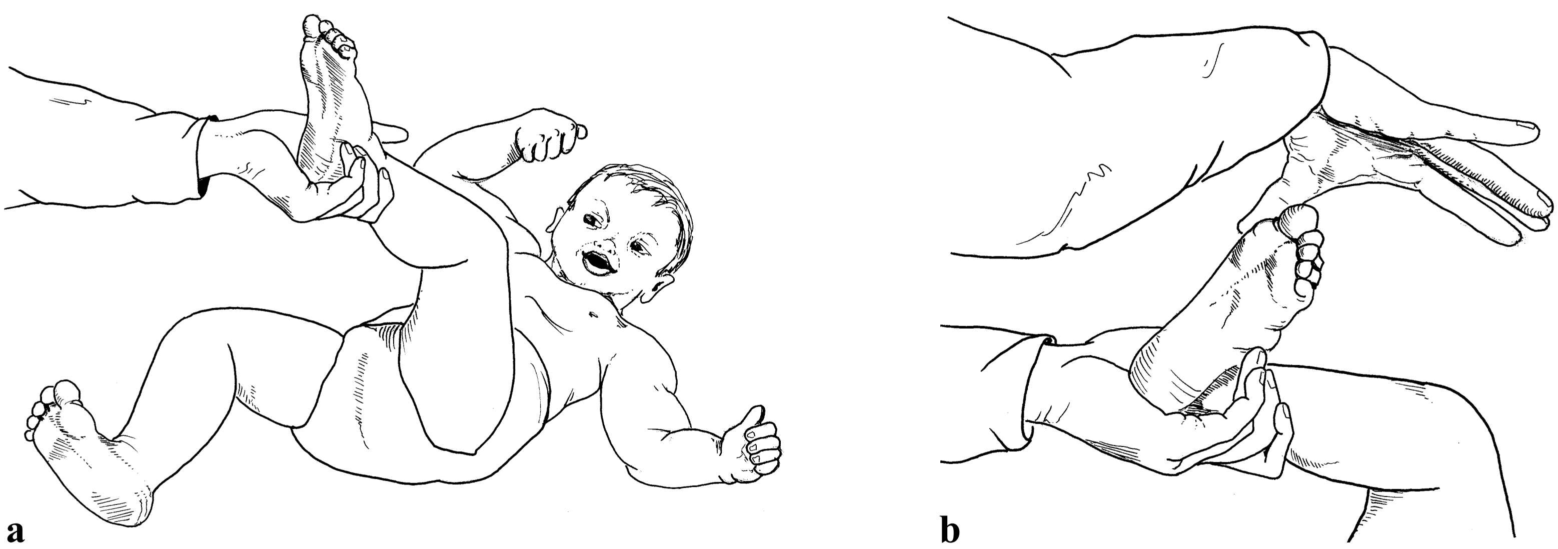


Forefoot Adduction Correction Test

For assessment and differential diagnosis of rigid and flexible pes adductus (talipes varus).

- **Procedure:** The child is supine. The examiner grasps the foot of the affected leg with one hand and attempts to correct the pes adductus deformity by pressing on the medial aspect of the forefoot with thumb of the other hand.
- **Assessment:** Where this maneuver readily moves the forefoot across the midline and eliminates the pes adductus, the deformity is usually flexible and will be spontaneously corrected. A deformity that cannot be passively corrected is a rigid pes adductus.

Congenital pes adductus deformities that resist manual correction will require rigorous timely treatment in corrective plaster casts.



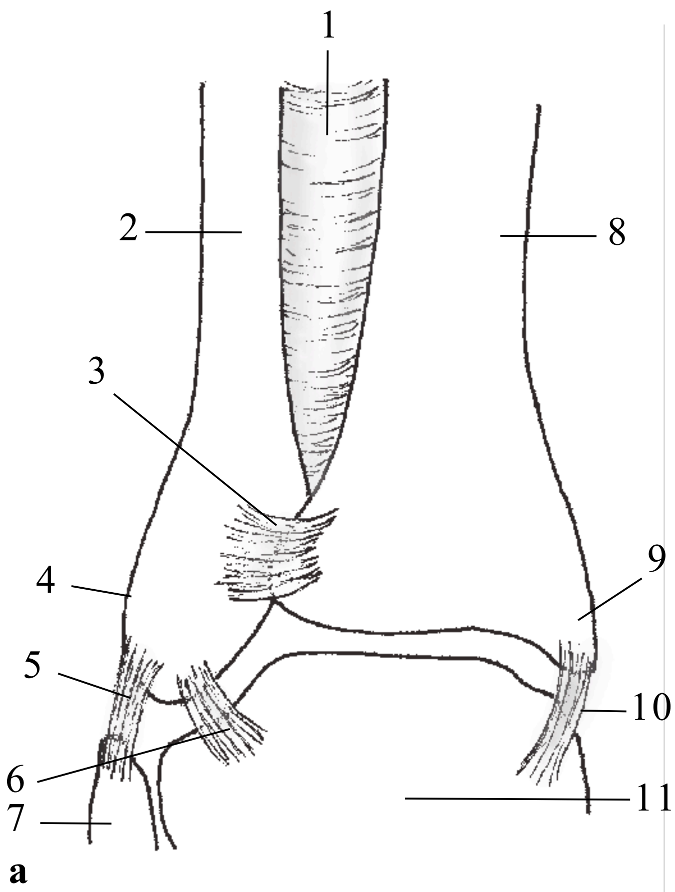
a b

Fig. 7.14 Forefoot adduction correction test. **(a)** Deformity. **(b)** Passively correctable.

Collateral and Syndesmosis Ligaments

The largest ligament of the ankle joint is the deltoid ligament (medial ligament of the ankle), which is a composite ligament consisting of four individual ligaments. On the lateral side, three ligaments stabilize the ankle: the anterior and posterior talofibular ligaments and the calcaneofibular ligament. The mechanism of injury for the deltoid ligament is similar to syndesmotic injuries. The mechanism for a lateral ligament rupture involves supination trauma with a slightly plantar flexed foot. Ten to twenty percent of all ankle injuries involve syndesmotic injury.

The interosseous membrane joins the fibula and tibia as a syndesmosis; the tibiofibular syndesmosis is found at the distal ends of these two bones. This complex is made up in part by the anterior and posterior tibiofibular ligaments, both of which are only minimally flexible, so that no significant shift between the two bones of the lower leg can occur during dorsiflexion. Forced dorsiflexion of the ankle or an impact injury to the proximal extremity or upper body with the foot fixed in external rotation, as occurs frequently in soccer players, can result in syndesmosis injury.



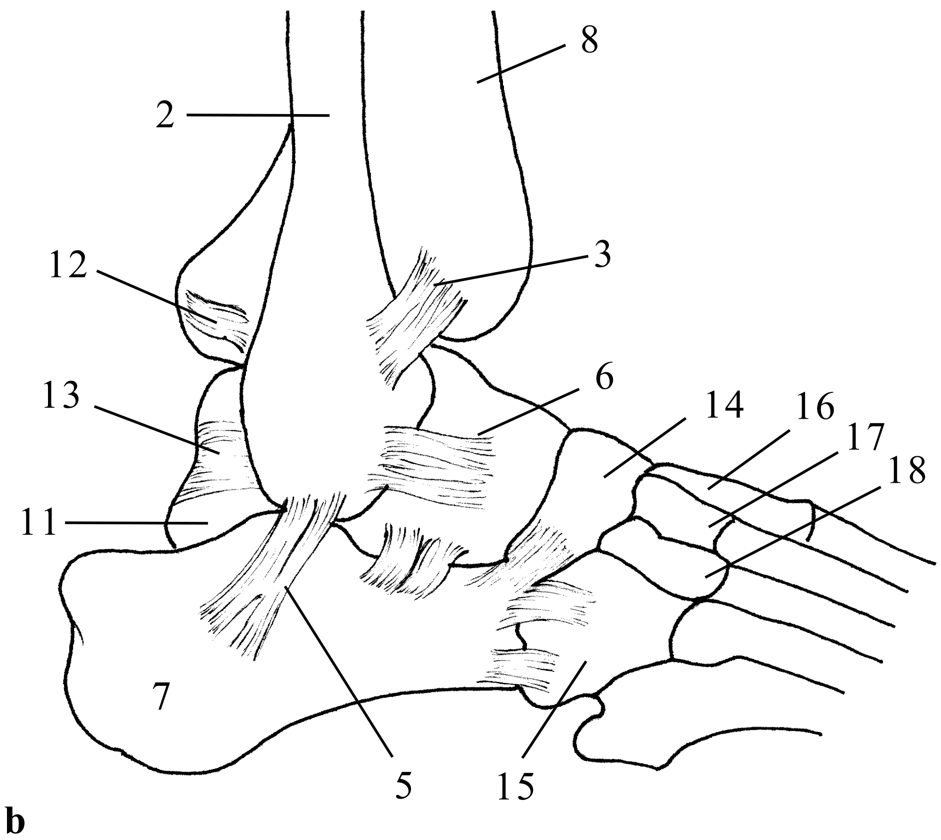


Fig. 7.15 Anatomy of the ankle and posterior foot. **(a)** Anterior view. **(b)** Lateral view.
1 Interosseous membrane.
2 Fibula.
3 Anterior tibiofibular ligament.
4 Lateral malleolus.
5 Calcaneofibular ligament.
6 Anterior talofibular ligament.
7 Calcaneus.
8 Tibia.
9 Medial malleolus.
10 Deltoid ligament.
11 Talus.
12 Posterior tibiofibular ligament.
13 Posterior talofibular ligament.
14 Navicular bone.
15 Cuboid bone.
16 Medial cuneiform bone.
17 Intermediate cuneiform bone.
18 Lateral cuneiform bone.

Talar Tilt Test 1 (Inversion Stress Test or Varus Stress Test)

Evaluation of an injury to the lateral collateral ligament of the ankle (calcaneofibular, posterior talofibular, and anterior talofibular ligaments).

- **Procedure:** The patient lies supine or sits with legs over the edge of the examination table. The examiner holds the calcaneus with one hand in order to bring the foot and ankle into the neutral position. The other hand grasps above the ankle to stabilize the lower leg. The thumb or the fingers palpate the calcaneofibular ligament. From this position and maximal dorsiflexion, the examiner inverts the foot at the ankle joint (hindfoot varus stress).
- **Assessment:** Visibly or palpably obvious strong angulation or a difference of more than 15° between both ankles associated with pain is suggestive of complete tears of both the calcaneofibular and anterior talofibular ligaments on the affected side. Maximum dorsiflexion locks the subtalar joint and improves the sensitivity of this test. The ankle joint and the subtalar joint should be examined individually on both sides, because some individuals (especially children) who are suspected of having joint instability, actually have physiologically lax joints.
- **Note:** One can obtain a better assessment of this test if it is performed together with the anterior drawer test. The combination of an anterior drawer of > 5 mm and gapping of > 10° on the tilt test is suggestive of injury to the anterior talofibular and calcaneofibular ligaments.

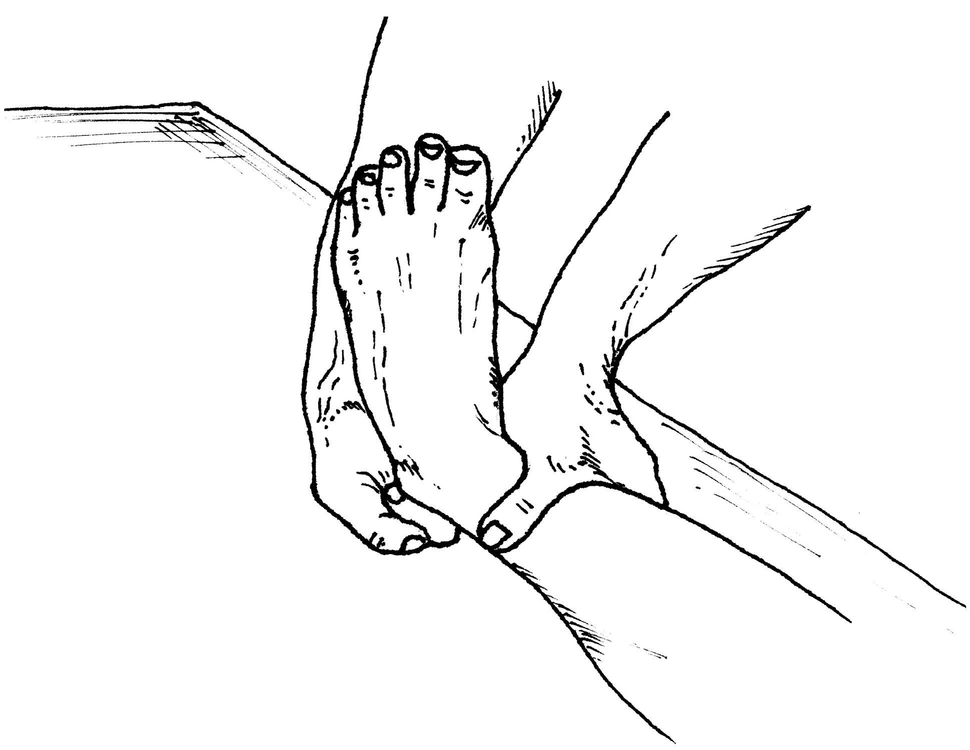


Fig. 7.16 Talar tilt test 1 (inversion stress test or varus stress test).

Talar Tilt Test 2 (Eversion Stress Test or Valgus Stress Test)

Evaluation of an injury to the deltoid ligament, especially the tibiocalcaneal part of the ligament.

□ **Procedure:** The patient lies supine or sits with both legs hanging from the edge of the examination table. The examiner grasps the patient's calcaneus with one hand and keeps the foot in a neutral position. The other hand stabilizes the lower leg above the ankle with the thumb (or fingers) placed along the deltoid ligament to note any gapping between the talus and mortise. From this position, the examiner everts the foot (hindfoot valgus stress).

□ **Assessment:** An injury of the deltoid ligament is likely when the talus tilts or gaps excessively in comparison to the uninjured side and if pain occurs during the test.

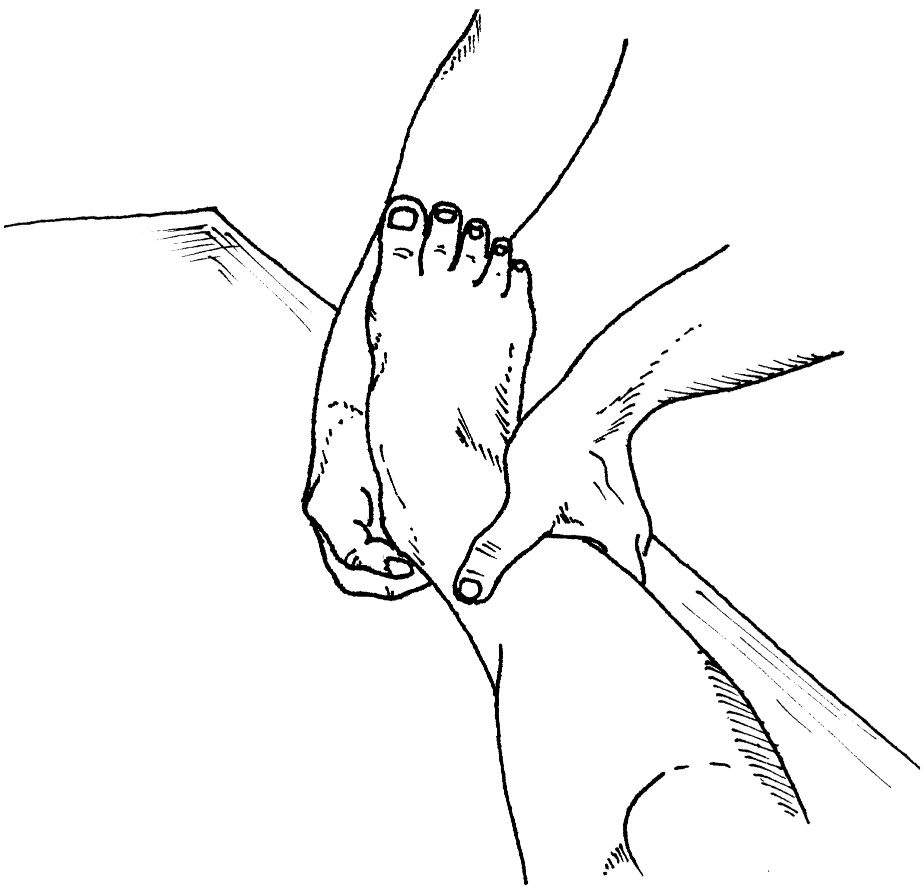


Fig. 7.17 Talar tilt test 2.

Anterior and Posterior Drawer Tests

Evaluation of anterior talofibular ligament injury.

□ **Procedure:** The patient lies supine with the knee slightly flexed to neutralize the pull of the gastrocnemius muscle. With the ankle joint held at 10 to 15° of plantar flexion, the examiner grasps around the heel with one hand and stabilizes the tibia from the anterior side with the other. After asking the patient to relax the muscles, the examiner pulls the heel forward while continuing to stabilize the tibia with the other hand.

□ **Assessment:** In the presence of a rupture of the anterior talofibular ligament, usually combined with injury to the capsule, the talus rotates laterally and anteriorly with reference to the tibia. The center of rotation is the intact lateral ligaments.

The test suggests talofibular instability as a result of rupture of the anterior talofibular ligament.

□ **Note:** The test can also be carried out in the opposite direction. The examiner supports the tibia from posteriorly with one hand while grasping the midfoot with the other. The examiner then moves the foot posteriorly at the ankle joint while continuing to hold the tibia with the other hand. If there is an injury to the posterior talofibular or calcaneofibular ligaments, the talus moves posteriorly and rotates medially.

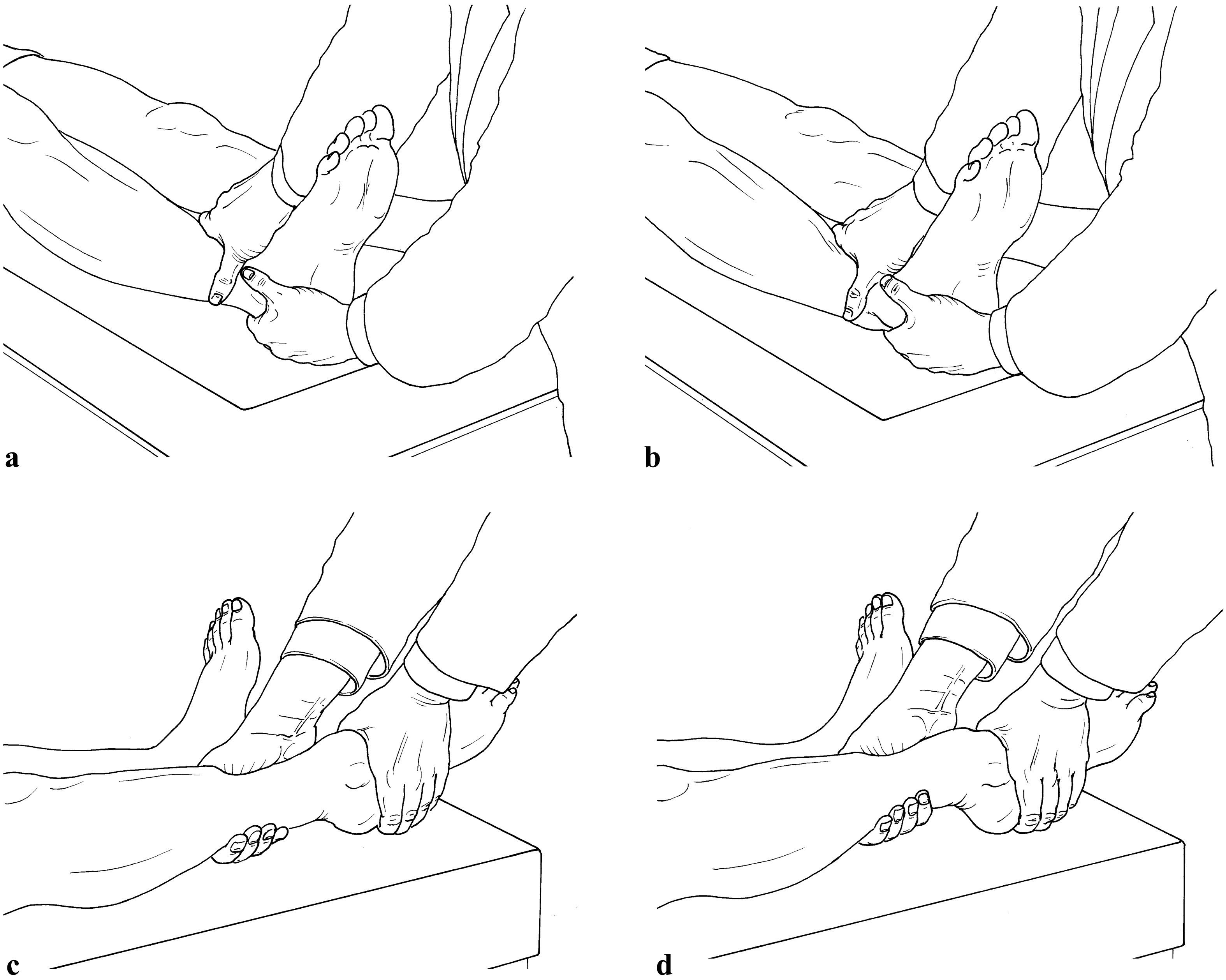


Fig. 7.18 Drawer test. **(a)** Starting position. **(b)** Pulling the foot anteriorly. **(c)** Starting position. **(d)** Moving the foot posteriorly.

External Rotation Stress Test (Kleiger Test)

Evaluation of an injury to the tibiofibular syndesmosis.

□ **Procedure:** The patient sits at the edge of the examination table. The examiner stabilizes the proximal lower leg from the anterior side with one hand. With the other hand over the hindfoot and with the foot in a neutral (0°) position, the examiner applies a forced external rotational load to the foot.

□ **Assessment:** The strong external rotational load imposed on the foot rotates the talus externally, thus causing the fibula and tibia to move apart and thereby putting stress on the distal syndesmosis. Pain experienced during the test in the anterolateral aspect of the ankle joint suggests injury to the syndesmosis. Pain on the medial side of the injured ankle during the external rotation stress test, with the ankle positioned in plantar flexion, may indicate injury to the deltoid ligament.

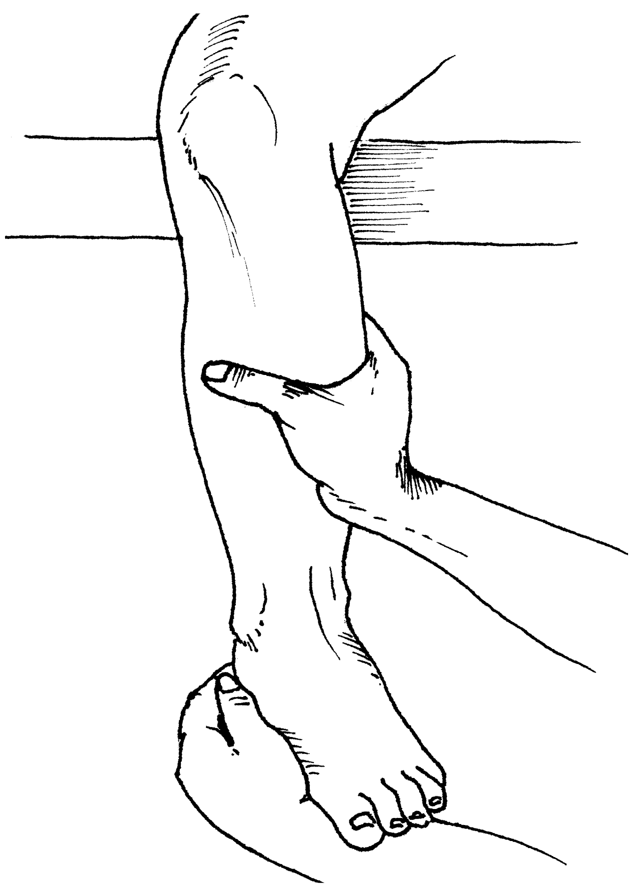


Fig. 7.19 External rotation stress test (Kleiger test).

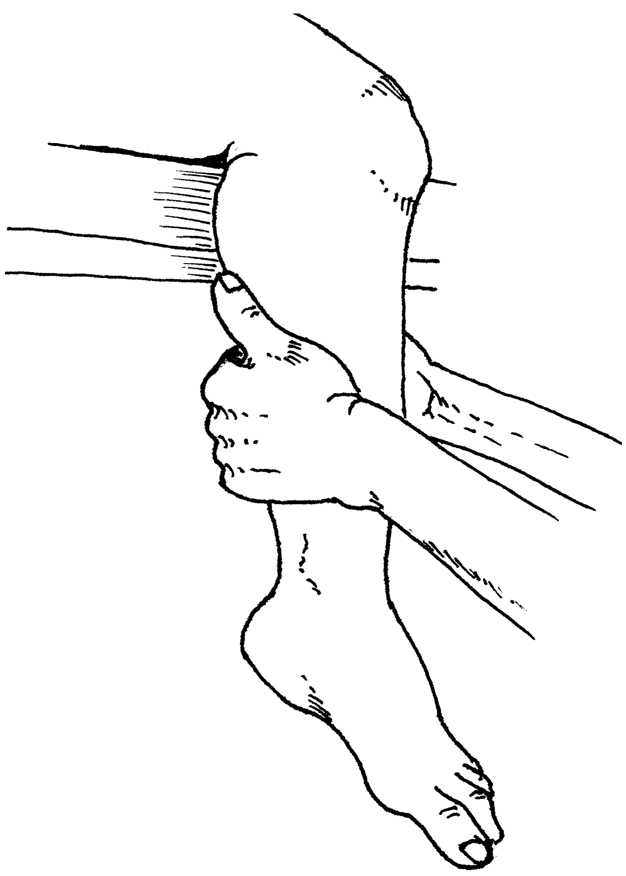


Fig. 7.20 Squeeze test.

Squeeze Test

Evaluation of an injury to the tibiofibular syndesmosis.

□ **Procedure:** The patient sits at the edge of the examination table. The examiner cups both hands around the patient's lower leg and compresses it, attempting to squeeze the tibia and fibula together. The hands must be placed sufficiently far from the distal syndesmosis.

Assessment: The presence of pain over the distal tibiofibular syndesmosis suggests an injury to it.

Dorsiflexion Test

Evaluation of an injury to the tibiofibular syndesmosis.

Procedure: The patient sits at the edge of the examination table with the legs hanging down loosely. While stabilizing the patient's lower leg proximally with one hand, the examiner passively moves the foot into forced dorsiflexion with the other.

Assessment: The test is considered positive if the patient experiences pain in the area around the distal tibiofibular syndesmosis.

Heel Thump Test

Procedure: The patient sits at the edge of the examination table with the lower legs hanging down loosely and both feet plantar flexed at the ankle. The examiner holds the patient's lower leg with one hand and with the other hand delivers an axillary directed gentle thump to the heel with the fist.

Assessment: Pain experienced at the distal tibiofibular syndesmosis suggests the presence of injury. Although the heel thump test has been recommended to help differentiate between a syndesmotic injury and a lateral ankle sprain, it is also used for tibial stress fractures.

Fig. 7.21 Dorsiflexion test.

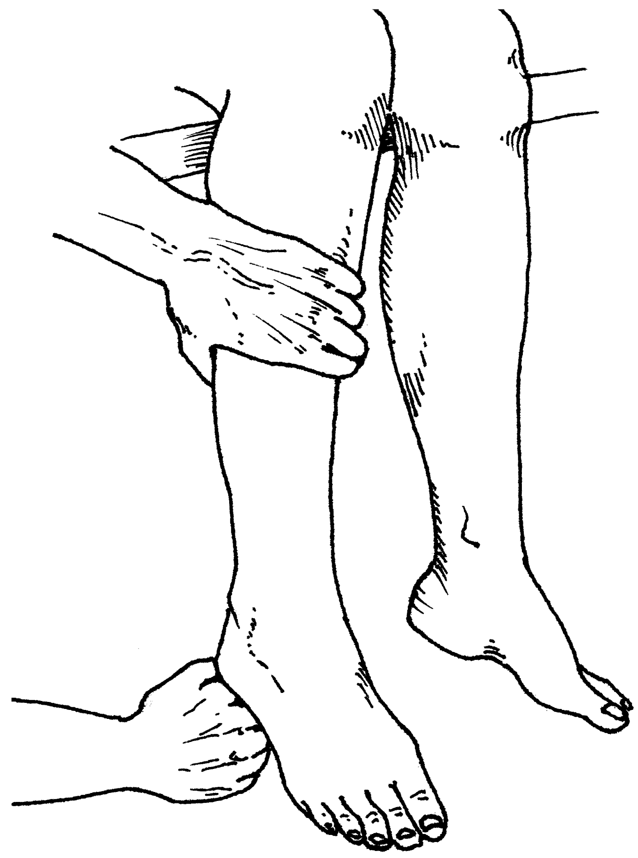


Fig. 7.22 Heel thump test.

7

Ankle Impingement

Painful entrapment of joint structures in the ankle is known as an ankle impingement syndrome. In addition to identifying the involved joint structures, the pathologic cause of the impingement must be differentiated, whether osseous, soft tissue, or neutral.

In addition, an anterior (usually anterolateral) impingement is differentiated from a posterior impingement. The anterior and lateral areas of the ankle are more often affected than the posterior area. It can occur as a result of acute trauma as well as after chronic overuse with repeated microtrauma—especially in soccer players (soccer player's ankle) and in dancers.

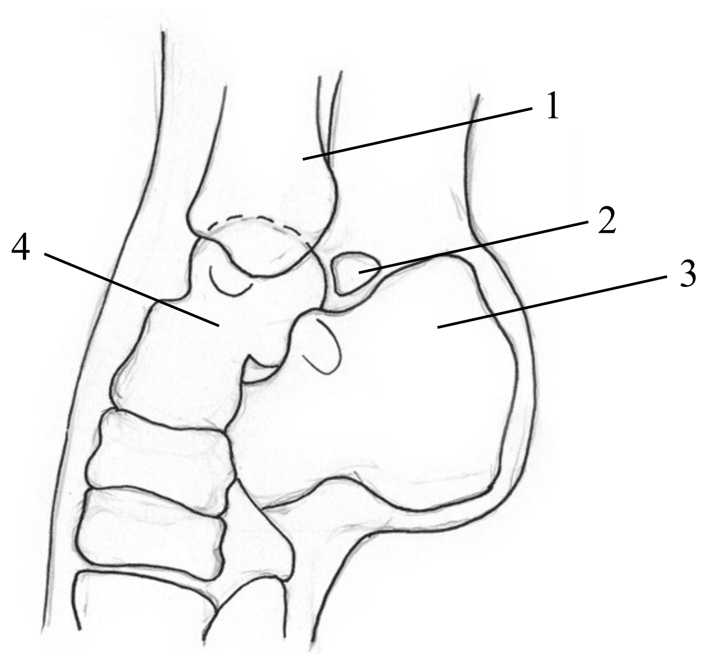


Fig. 7.23 Anatomy of the ankle, posteromedial view.
1 Posterior edge of the tibia.
2 Triangular bone (os trigonum).
3 Calcaneus.
4 Talus.

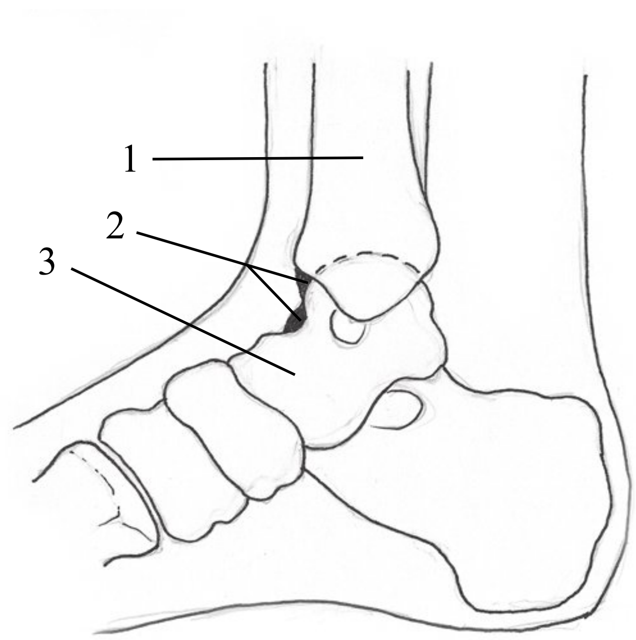


Fig. 7.24 Anatomy of the ankle, anteromedial view.
1 Tibia.
2 Osteophytes of the anterior tibial margin and the talar neck.
3 Talus.

Anterior Ankle Impingement Test: Hyperdorsiflexion Test

□ **Procedure:** The patient sits on the edge of the examination table with the legs hanging down loosely and the knee bent 90°. With one hand, the examiner grasps around and stabilizes the patient's heel, and with the other hand grasps the mid- and forefoot on the plantar side from below and then brings the foot into maximal dorsiflexion. The test should be repeated using slightly internally and externally rotated positions of the foot.

□ **Assessment:** Strong hyperdorsiflexion can provoke pain in the area around the anterior ankle joint. Local pain felt on palpation of the anterior joint line is most commonly located medial to the tendon of the tibialis anterior muscle or lateral to the tendons of the tibialis anterior and extensor digitorum longus muscles.

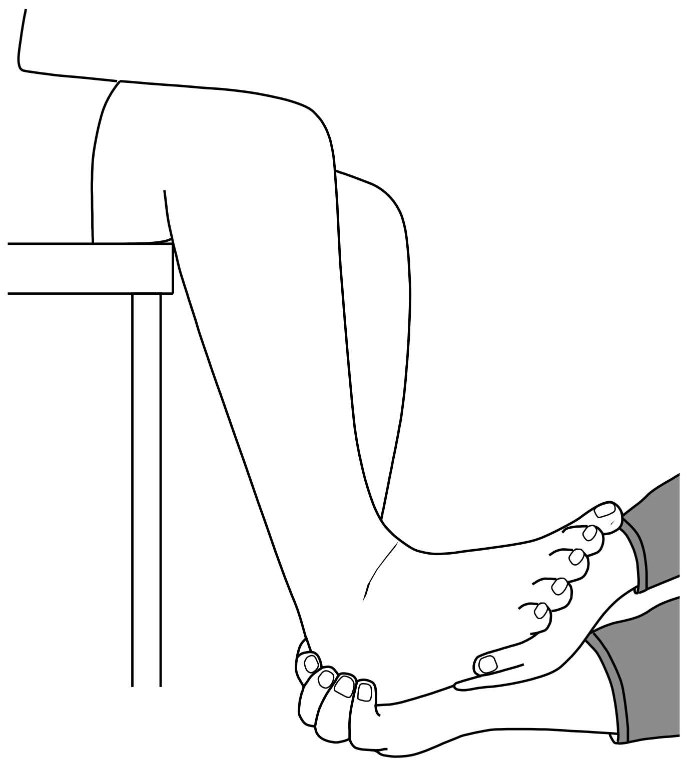


Fig. 7.25 Anterior ankle impingement test.

Posterior Ankle Impingement Test: Hyperplantar Flexion Test

- **Procedure:** The patient sits on the edge of the examination table with the legs hanging down loosely and the knees flexed 90°. With one hand the examiner holds the patient's heel and stabilizes it and with the other grasps the mid- and forefoot over the dorsum of the foot. The examiner then forces the foot into maximum plantar flexion and repeats this action several times. The test should be repeated using slightly externally and internally rotated positions.
- **Assessment:** The test is considered positive if the patient complains of pain at maximal plantar flexion, especially in the posterolateral region of the hindfoot. The cause of the pain is an impingement, which occurs when soft tissue or a bony protuberance become impacted between the tibia and the posterior margin of the calcaneus. The test should be repeated in various degrees of foot rotation.

A positive test combined with pain felt on posterolateral palpation should be followed by a diagnostic anesthetic injection. The infiltration of the joint capsule is carried out under sterile conditions on the posterolateral aspect of the foot between the posterior process of the talus and the posterior edge of the tibia. After the injection, the test is repeated; if the result is now negative (no pain on forced plantar flexion), the diagnosis is confirmed.

□ **Note:** The posterior ankle impingement syndrome due to overuse is most commonly seen in ballet dancers and runners. Jogging, especially downhill, is associated with continually repetitive plantar flexion movements, which can impose repetitive stresses to the posterior aspect of the ankle joint. In dancers, certain dance sequences, such as the "en pointe" or the "demi-pointe" position, are achieved by forceful plantar flexion, which results in soft-tissue and bony changes in the hindfoot; thus posterolateral foot pain in dancers is referred to as "dancer's heel."

The following anatomical and pathologic structures can lead to posterior impingement:

- Displaced os trigonum.
- Hypertrophic posterior talar process.
- Loose bodies located in the posterior part of the ankle joint.
- Osteophytes at the posterior margin of the tibia.
- Posttraumatic scar tissue or calcifications.

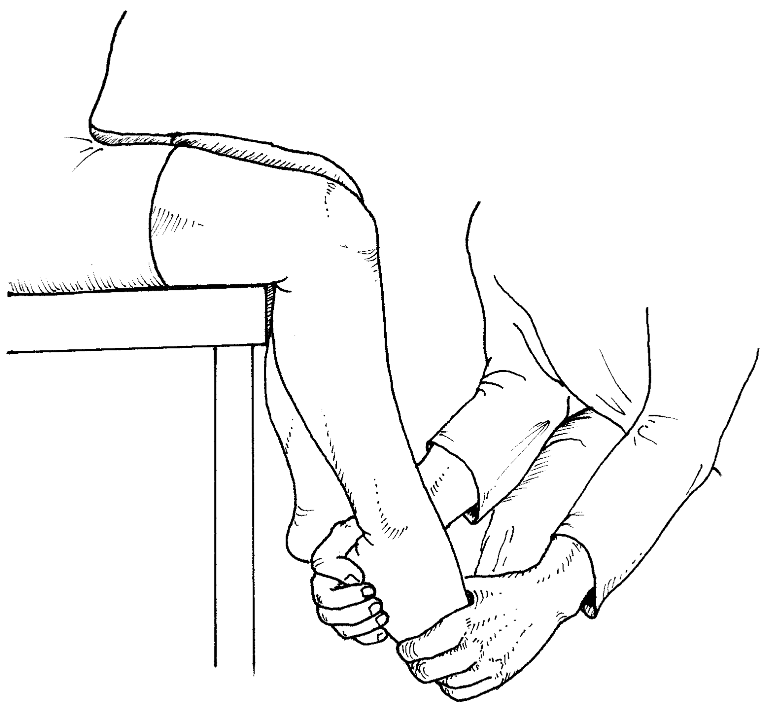


Fig. 7.26 Posterior ankle impingement test.

Nerve Damage

Digital Nerve Stretch Test

Evaluation of Morton's neuralgia.

□ **Procedure:** The patient lies supine. Both ankles are held in full dorsiflexion, while the examiner fully dorsiflexes the lesser toes passively on either side of the suspected web space bilaterally.

□ **Assessment:** The test is positive if the patient complains of burning pain over the plantar surface at the level of the affected metacarpal head with radiation into the respective toe(s). The patient often complains of "electric" pain on standing and when "toeing off" while walking with radiation into the toes, occasionally associated with hypesthesia of the affected toe(s). Even the pressure from narrow shoes can trigger the pain.

□ **Note:** Morton's neuralgia is a pain syndrome caused by an isolated injury to a purely sensory interdigital terminal branch of the tibial nerve. The neuralgia is usually located somewhat distal to the transverse metatarsal ligament in the area around the third and fourth metatarsal heads. Foot deformities such as splayfoot and hallux valgus facilitate Morton's neuralgia. Women are affected in 80%of the cases.

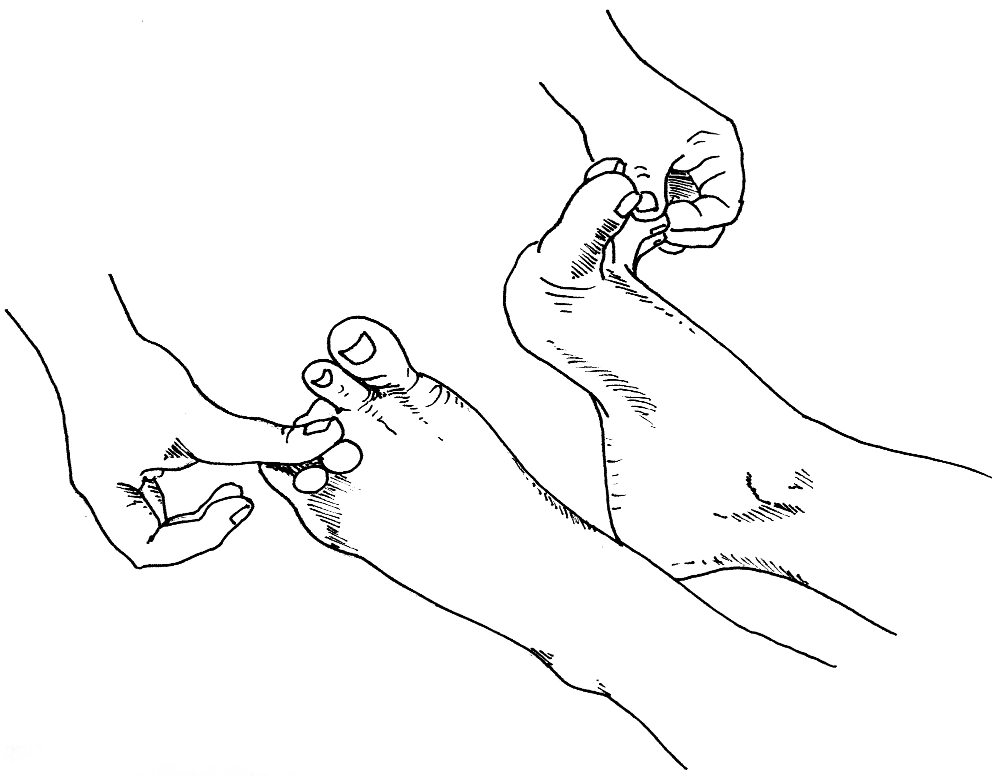


Fig. 7.27 Digital nerve stretch test.

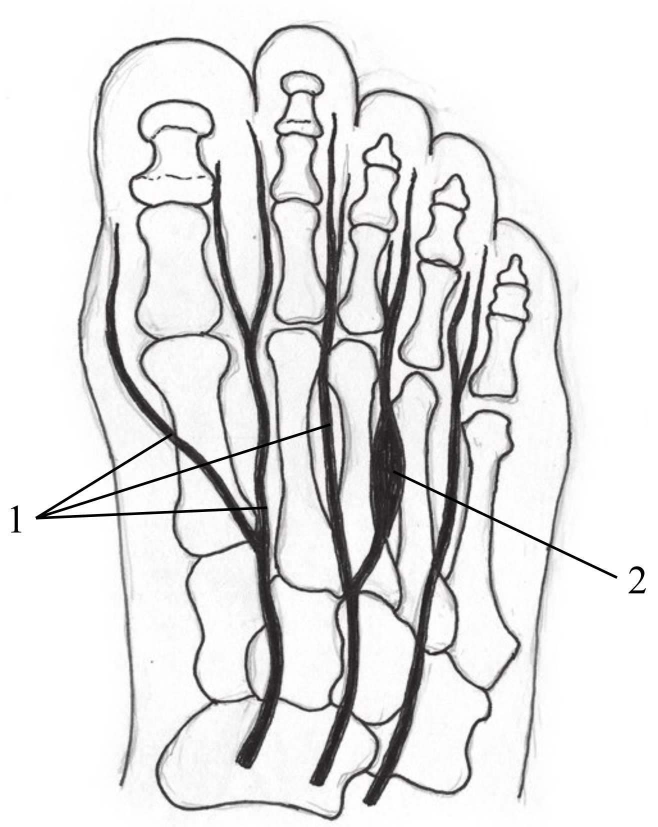


Fig. 7.28 Schematic representation of Morton's neuralgia.

1 Plantar digital nerves (terminal branches of the tibial nerve).
2 Pseudoneuroma of the digital nerve in the third intermetatarsal space.

Mulder Click Test (Morton's Test)

Indicates an interdigital neuroma (Morton's neuroma).

□ **Procedure:** The examiner grasps the patient's forefoot in a pincer grip and compresses it. This pushes the adjacent metatarsal heads against each other.

□ **Assessment:** Where an interdigital neuroma is present, pushing the metatarsal heads against one another will cause pain with occasional paresthesia radiating into the adjacent toes. Small fibromalike hardened areas between the toes will also be palpable and will displace, sometimes with a clicking sound, as the forefoot is compressed. Morton's neuroma is a spindle-shaped bulb that develops in a plantar nerve. Painful interdigital neuromas usually develop in the second or third interdigital fold; neuromas in the first or fourth interdigital fold are rare. Injection of a local anesthetic through the deep transverse metatarsal ligament can confirm the diagnosis by anesthetizing the neuroma. A pain provocation test of the interdigital space from the plantar side is the **Hohmann maneuver**.

Fig. 7.29 Mulder click test.

Tinel Sign

Indicates a tarsal tunnel syndrome.

- **Procedure:** The patient is prone with the knee flexed 90°. The examiner taps the tibial nerve posterior to the medial malleolus with a reflex hammer.
- **Assessment:** Pain and paresthesias in the sole of the foot suggest a tarsal tunnel syndrome. This disorder involves chronic neuropathy at the medial malleolus beneath the flexor retinaculum. The nerve can be palpated posterior to the medial malleolus, which will elicit pain. Advanced neuropathy is associated with sensory deficits in the regions supplied by the plantar nerves and paresthesia and atrophy in the plantar muscles.

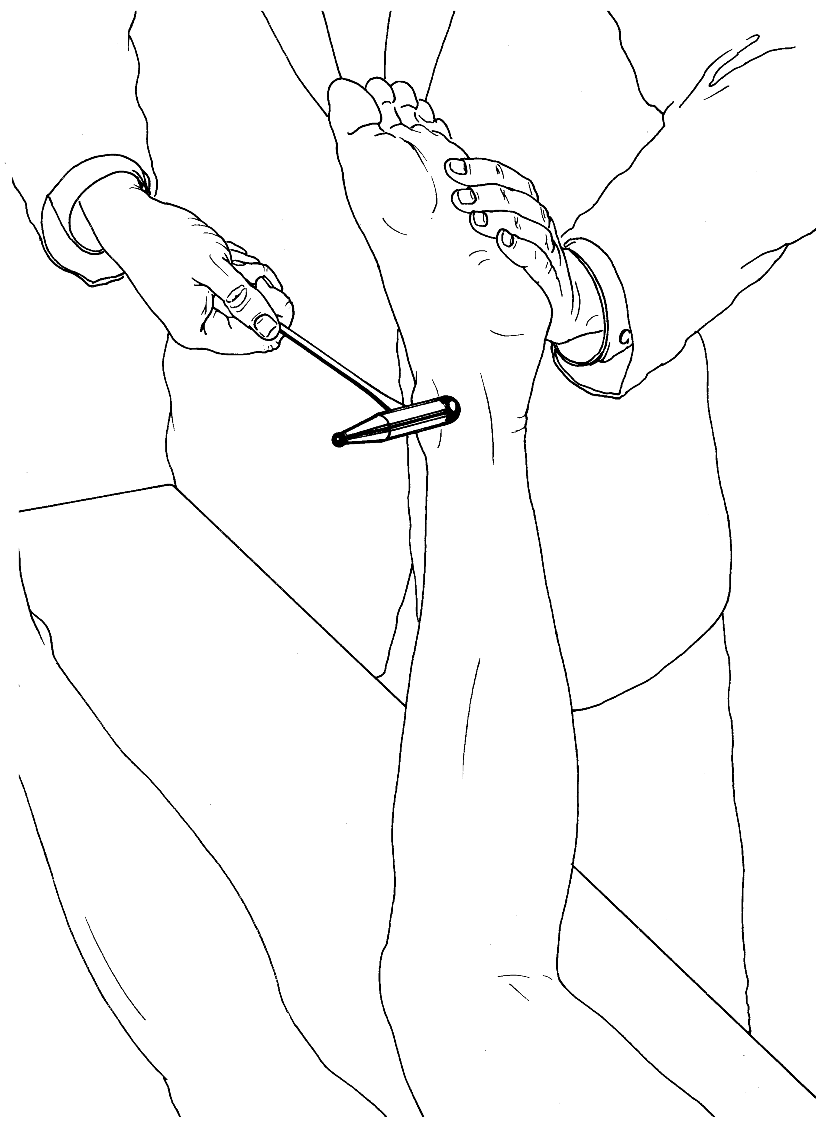


Fig. 7.30 Tinel sign.

Fracture

Heel Compression Test

Assesses a stress fracture of the calcaneus.

□ **Procedure:** The examiner symmetrically compresses the patient's heel between the thenar eminences of both hands.

□ **Assessment:** In a stress fracture of the calcaneus, the patient will feel intense pain in the heel. Stress fractures of the calcaneus primarily occur in patients with significant osteoporosis. Patients with these fractures exhibit a conspicuous antalgic gait, often without any weight bearing on the heel at all. The heel itself may exhibit diffuse swelling and tenderness to palpation. The heel compression test rarely causes serious pain in patients with heel pain from other causes such as retrocalcaneal bursitis.

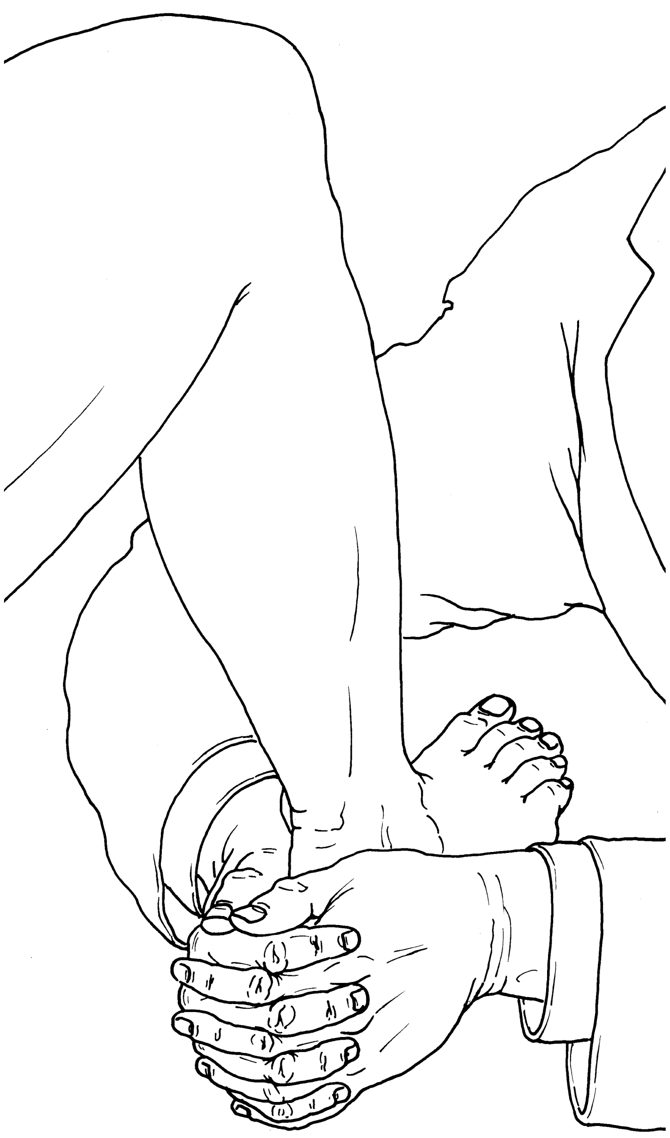


Fig. 7.31 Heel compression test.

8 Posture Deficiency

Erect standing posture is determined not only by the position of the spine (or trunk) but is primarily the result of muscular activity. We differentiate between erect standing posture and relaxed (standing) posture. By erect standing posture we mean a tense attitude of readiness characterized by a balance in the forces within the musculature, whereas relaxed posture is a comfortable stance of rest and recovery. This relaxed posture is usually a habitual posture, characteristic of the individual, and depends largely on the individual's particular spinal and pelvic anatomy.

Postural weakness may be defined as extreme difficulty in achieving and maintaining the erect standing posture. The patient is either unable to shift from a relaxed posture to an erect standing posture or is only able to maintain upright standing temporarily. Chronic postural weakness can lead to deterioration of posture and eventually to a chronic deformity. Postural weakness and deterioration of posture define a continuum, and it is important to promptly identify children and adolescents who are at risk in order to prevent the development of a postural deformity. Posture depends on the quality of the musculature and the existing anatomy. Various functional deviations from the physiologic curvatures have been described. According to Wagenhäuser, they represent deficient variations of normal posture. These include unsteady posture, round back, swayback, flat back, and lateral deformities.

Diagnostically, one must differentiate functional postural weaknesses from deficiencies due to organic spinal disorders such as Scheuermann's disease and spondylolisthesis. A variety of posture tests can be used to assess postural deficiencies.

The Matthias postural competence test allows assessment of the competence of the postural muscles. The Kraus–Weber test allows assessment of the competence of the trunk and pelvic muscles. The strength and endurance of the muscles of the abdomen and back are measured. This test aids in determining the quantitative and qualitative effect of muscular action in neutralizing the effect of the body's weight.

Kraus–Weber Tests

Test the competence of the trunk and pelvic muscles.

Procedure:

A: The patient is supine with the legs and feet extended and the hands clasped behind the head. The patient is then asked to raise his or her extended legs 25 cm and to hold them at this height for 10 seconds. This tests the lower abdominal muscles. It counts for 10 points.

B: The patient is supine with the hands clasped behind the head. The examiner immobilizes the patient's feet. The patient is asked to sit up. This tests the upper abdominal muscles. Sitting up 90° counts for 10 points; sitting up 45° counts for 5 points.

C: The patient is supine with the hands clasped behind the head but with the legs flexed. The examiner immobilizes the patient's feet. The patient is asked to sit up. This tests all of the abdominal muscles with the effect of the psoas neutralized.

8

D: The patient is prone with a cushion beneath the abdomen and the hands clasped behind the head. The examiner immobilizes the patient's hips and feet against the examining table. The patient is asked to raise his or her body off the examining table and to maintain that position for 10 seconds. This tests the upper back muscles. It counts for 10 points.

E: The patient is prone with a cushion beneath the pelvis. The examiner immobilizes the patient's trunk and hips against the examining table. The patient is asked to raise his or her legs off the examining table with the feet extended and to maintain that position for 10 seconds. This tests the lower back muscles. It counts for 10 points.

F: The patient stands barefoot with hands at his or her sides. The patient is then asked to bend over with the knees extended and arms stretched out in front. The examiner measures the distance to the floor.

□ **Assessment:** Normal results for the Kraus–Weber test are indicated by this index:

$$A\frac{10}{10} \qquad B\frac{10}{10} \qquad FTF = 0$$

where A represents the strength of the abdominal muscles and B the strength of the back muscles. The numerators are the values for the upper abdominal muscles and upper back muscles, respectively; the denominators are the values for the lower abdominal muscles and lower back muscles including the psoas, respectively. FTF is the fingertip-to-floor distance.

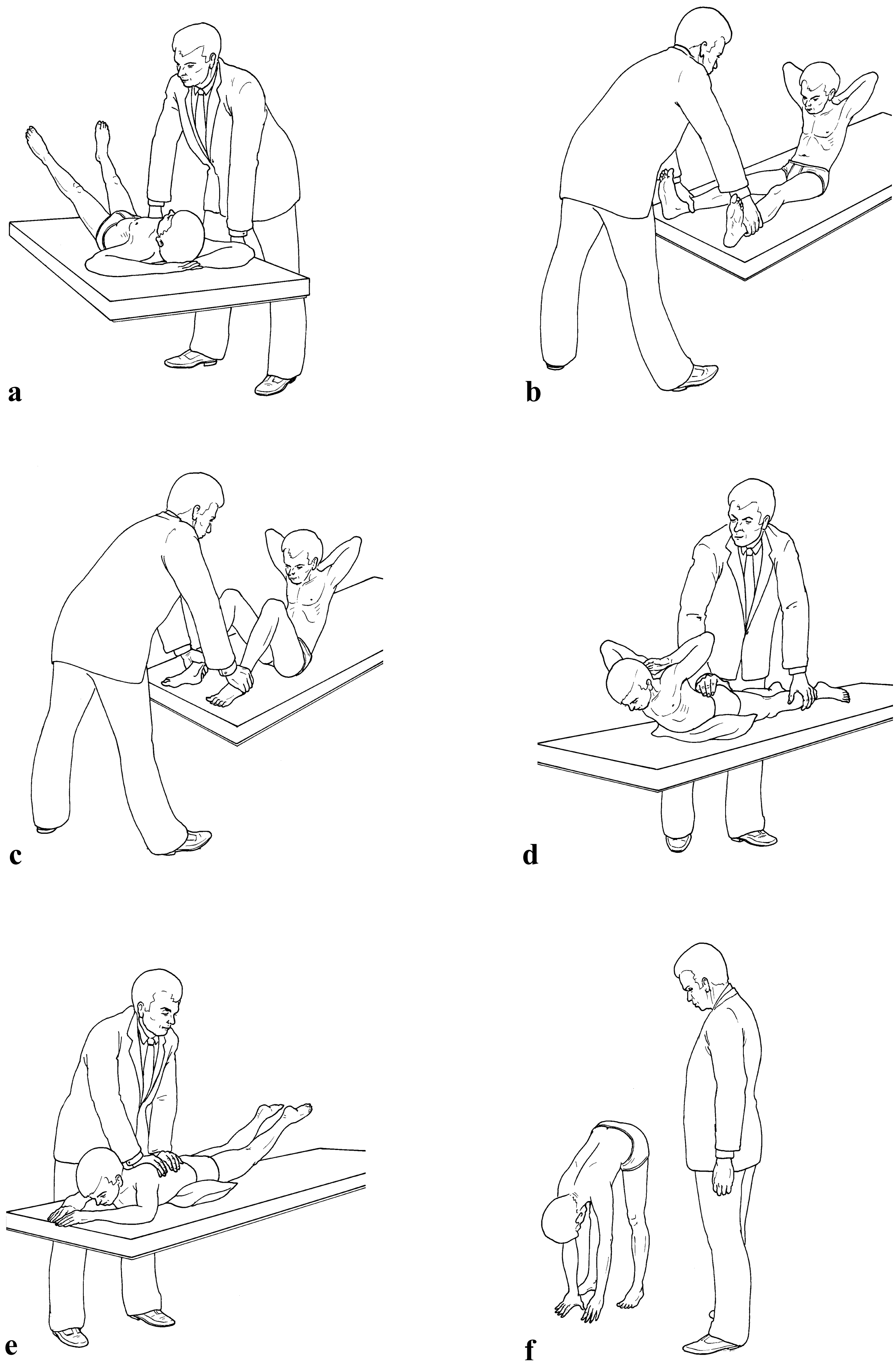


Fig. 8.1 Kraus–Weber tests. **(a)** Test A. **(b)** Test B. **(c)** Test C. **(d)** Test D. **(e)** Test E. **(f)** Test F.

Matthias Postural Competence Tests

Assess the competence of the back and trunk muscles in children and adolescents.

□ **Procedure** The examination is performed with the patient standing. The child is asked to raise both arms straight in front and keep them in that position.

□ **Assessment** Raising the arms shifts the body's center of gravity forward.

The child with normal posture compensates for the shift in the center of gravity by leaning the entire body slightly backward. A child with postural weakness will exhibit increased thoracic kyphosis and lumbar lordosis.

Matthias identifies two degrees of postural weakness.

Patients with full muscular function will usually be able to achieve and maintain full erect posture with minimal backward bending in the arm-raising test. In first-degree postural weakness, the child can actively achieve full erect posture but within 30 seconds slumps into a backward bending posture with increased thoracic kyphosis and lumbar lordosis.

Second-degree postural weakness occurs when the child is unable to actively achieve full erect posture and slumps backward right at the start of the arm-raising test. The child will push the pelvis forward and greatly increase the lumbar lordosis. This is referred to as postural deterioration.

The differential diagnosis must differentiate functional postural deficits from organic spinal disorders. A thorough clinical examination with function tests will allow postural weakness to be distinguished from deformities and idiopathic disorders at an early stage. In particular, examination must exclude scoliosis, kyphosis, and spondylolisthesis, as well as various forms of spinal deformities, such as flat back, round back, and swayback.

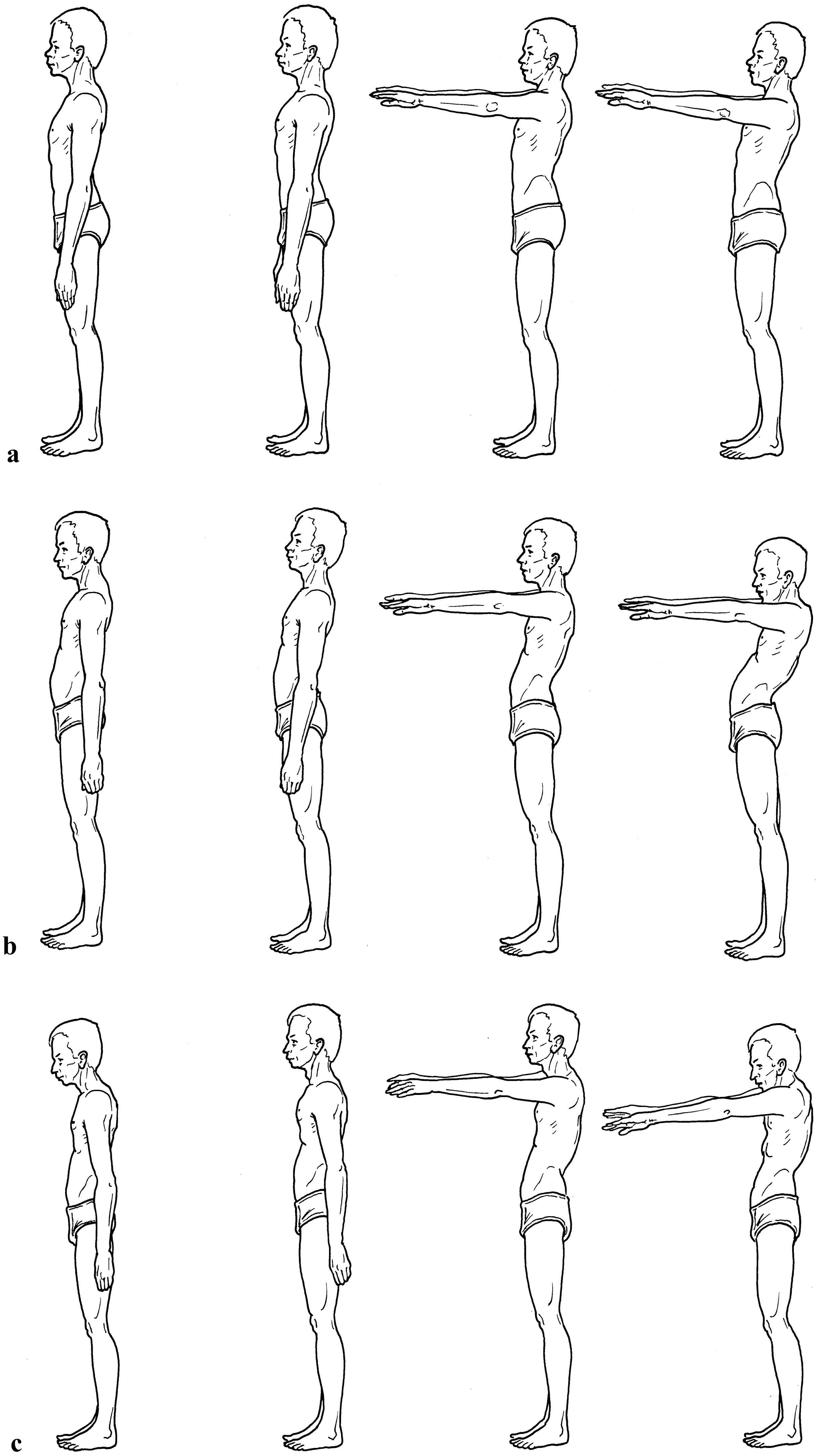


Fig. 8.2 Matthias postural competence tests. **(a)** Normal posture. **(b)** Postural weakness. **(c)** Postural deterioration.

9 Venous Thrombosis

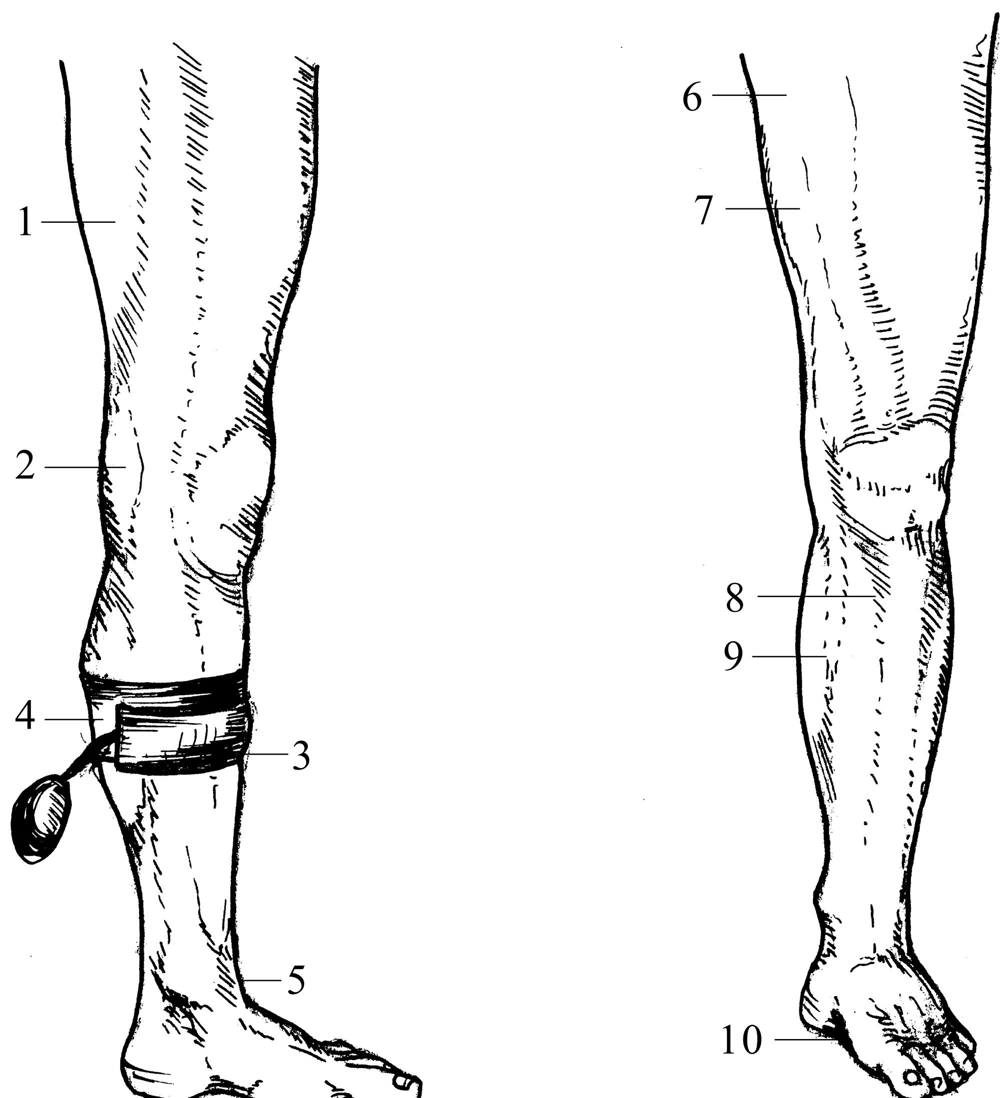
Acute deep venous thrombosis ranks with acute arterial occlusion as one of the most serious and dramatic vascular emergencies. Factors contributing to thrombosis include vessel wall, blood flow, and coagulation characteristics. Thromboses most commonly develop in the lower extremities. They are a feared postoperative complication as they involve the risk of acute massive or recurrent pulmonary embolism. Thrombosis in the deep veins of the leg is less symptomatic yet involves a far greater risk of embolism than thrombosis in the superficial veins. Swelling in the extremity (primarily in the left leg at the vascular spur in the pelvic veins), often associated with spontaneous pain in the groin, and pain radiating into the leg upon coughing or straining, local blue discoloration of the skin, and in some cases elevated temperature and pulse are important signs. A pulmonary infarction will often be the first clinical symptom, but typical early signs of deep venous thrombosis may also occur. These include spots that are painful to palpation, extending from the sole of the foot (Payr) to, in certain cases, the groin (Rielander), and pain upon compression of the calf (Lowenberg) when a blood pressure cuff is applied and pumped up to 100 mm Hg (13.3 kPa). However, these thrombosis signs are nonspecific and should by no means be regarded as conclusive. The unilateral edema that usually occurs develops gradually and begins in the malleolar region. Additional characteristic findings include distended congested peripheral veins in the affected extremity (Pratt "warning" veins), evidence of superficial collateral veins, and an expanding edema, which is dependent on the size and localization of the thrombosis.

In patients with chronic venous disease, a number of test methods are helpful in evaluating the function of the deep veins and perforating veins.

Lowenberg Test

Early sign of venous thrombosis.

- **Procedure:** The examiner applies blood pressure cuffs to each lower leg and pumps them up.
- **Assessment:** Normally, discomfort will occur only beyond 180 mm Hg (24 kPa). Where thrombosis is present, the normal leg will be observed to tolerate compression of the calf musculature with far higher pressure than the affected leg.



a b

Fig. 9.1 (a, b) Early signs of deep venous thrombosis.
1 Tenderness to palpation on the medial aspect of the thigh (sartorius, gracilis).
2 Tenderness to palpation in the knee (muscular insertions and medial joint cavity).
3 Pain on compression of the calf (Lowenberg).
4 Pain in the calf on dorsiflexion of the foot (Homans sign).
5 Tenderness to palpation.
6 Groin pain.
7 Tenderness to palpation along the adductor canal.
8 Pratt warning sign.
9 Meyer pressure points along the greater saphenous vein.
10 Pain in the sole of the foot, Payr sign: pressing or tapping the sole of the foot with the edge of the hand.

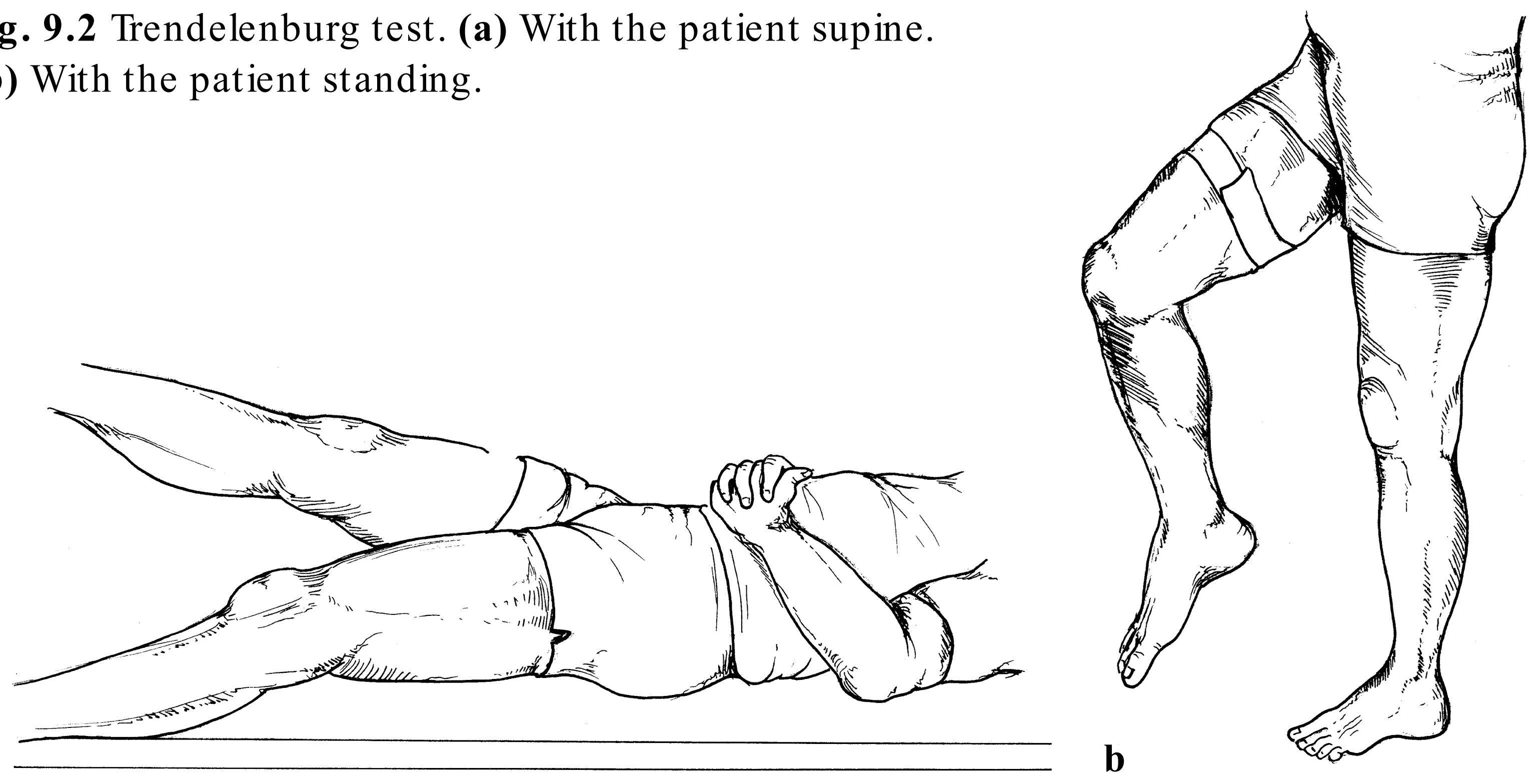
Trendelenburg Test

Assesses varicose veins in the thigh. Tests the function of the lesser saphenous vein and perforating veins.

 Procedure: With the patient supine and the leg raised, the examiner smoothes the distended veins. The examiner then compresses the greater saphenous vein with a tourniquet distal to its junction with the femoral vein at the inguinal ligament and asks the patient to stand up.

 Assessment: If the varices only fill up slowly or not at all within 30 seconds of the patient standing up but then fill rapidly from proximal once the tourniquet is loosened, this indicates valvular insufficiency of the saphenous vein with normal function of perforating veins. Relatively rapid filling from distal can occur as a result of insufficient perforating veins or anastomoses with an insufficient lesser saphenous vein. Rapid filling of the varices from both distal and proximal once the tourniquet is released indicates insufficiency of both the greater saphenous vein and the communication with the deeper venous system.

Fig. 9.2 Trendelenburg test. **(a)** With the patient supine. **(b)** With the patient standing.



Perthes Test

Assesses the function of deep veins and perforating veins.

□ **Procedure:** With the patient standing, the examiner applies a tourniquet to the thigh or lower leg proximal to the filled varices. The patient is then asked to walk around with the tourniquet in place.

□ **Assessment:** Complete emptying of the varices as a result of muscular activity indicates proper function of the perforating veins and intact deep venous drainage. The congestion is attributable to valvular insufficiency in the saphenous vein. Incomplete emptying is observed where there is moderate valvular insufficiency of the communicating veins. Unchanged filling in the varices occurs with significant insufficiency of the perforating veins and impaired blood flow in the deep veins. An increase in filling suggests a severe postthrombotic syndrome with reversed blood flow in the perforating veins.

□ **Note:** The Schwartz test or the percussion method of Schwartz and Hackenbruch is used to assess valvular insufficiency in the region of the greater saphenous vein. With the patient standing, the examiner places one finger on the distended vein being examined and taps on the junction of the greater saphenous and femoral veins with one finger of the other hand. If this tapping is transmitted back to the first finger, the blood flow is continuous, indicating that the valves in the portion of the vein being examined are not intact. The test is not necessarily definitive, but it is a good method for determining whether a superficial venous branch communicates with the greater or lesser saphenous vein.

Fig. 9.3 Perthes test.

Homans Test

Assesses deep venous thrombosis.

□ **Procedure:** The patient is supine. The examiner lifts the affected leg and rapidly dorsiflexes the patient's foot with the knee extended. This maneuver is repeated with the patient's knee flexed while the examiner simultaneously palpates the calf.

□ **Assessment:** Pain occurring upon dorsiflexion of the foot with the knee extended and flexed indicates thrombosis or thrombophlebitis.

Calf pain with the knee extended can also be caused by intervertebral disk disease (radicular symptoms) or muscle contractures.

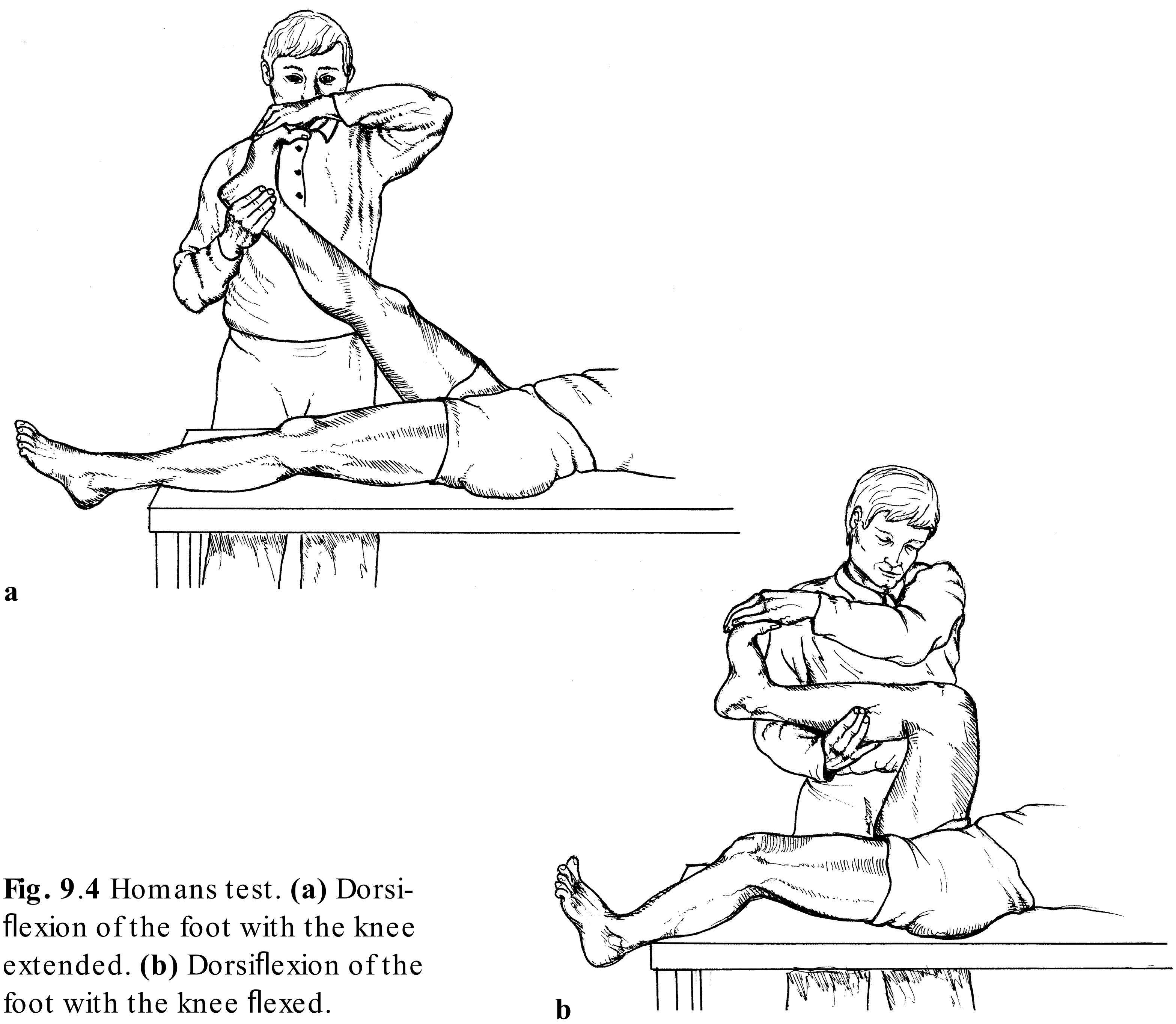


Fig. 9.4 Homans test. **(a)** Dorsiflexion of the foot with the knee extended. **(b)** Dorsiflexion of the foot with the knee flexed.

10 Occlusive Arterial Disease and Neurovascular Compression Syndromes

Occlusive arterial disease is often associated with orthopaedic disorders. Notably, nearly 90% of all cases of obliterative arteriosclerosis involve exclusively the lower extremities. Prior to treating the actual orthopaedic disorder, the physician must take care to exclude or identify any possible arterial ischemic disorders. After obtaining a detailed history, a diagnosis can usually be made on the basis of inspection, palpation, and specific function tests, and usually will not require the use of any diagnostic technology.

Weakened or absent arterial pulse, cool and pale skin (or cyanotic skin), patches of erythema and trophic disturbances are signs of occlusive arterial disease. Ulceration and gangrene are signs of advanced disease. Where typical symptoms of intermittent claudication (calf pain after walking short distances) are present, determining the maximum distance the patient can walk without experiencing these symptoms can help in estimating the severity of the disorder (Fontaine classification of the severity of occlusive arterial disease). The differential diagnosis of intermittent claudication must include spinal claudication from compression of the cauda equina, the cardinal symptom of lumbar spinal stenosis. The intermittent claudication in cauda equina pathology is not a sharply defined clinical syndrome. Radicular symptoms such as paresthesia, pain, sensory deficits, and weakness can occur in one or both legs when the patient stands or walks. These symptoms may improve or disappear when the patient stops moving, as in the vascular form, but more often will do so only on certain body movements.

Note: The walking test allows assessment of peripheral circulatory disruption. The patient is asked to walk up and down a long corridor for up to 3 minutes at about 120 paces per minute. The time of occurrence of symptoms and the site of pain are clinically assessed, as are gait and any pauses. If the patient pauses after only 60 seconds, this suggests severe disruption of vascular supply to the muscles. Symptoms of moderately severe circulatory disruption will manifest themselves after 1 to 3 minutes of walking. Symptoms that occur only after 3 minutes or more of walking indicate only mild circulatory disruption.

Note that exercise tolerance may be limited by cardiac and pulmonary disorders as well as orthopaedic disorders such as osteoarthritis of the hip or degenerative knee disorders.

Allen Test (Fist-Closure Test)

Assesses an arterial ischemic disorder in the upper extremities.

▸ **Procedure:** The patient is seated and raises his or her arm above the horizontal plane. The examiner grasps the patient's wrist and applies finger pressure to block the vascular supply from the radial and ulnar arteries. The patient then makes a fist so as to force the venous blood out of the hand via the posterior veins. After 1 minute, the patient lets the arm hang down and opens the now pale hand. The examiner now releases compression, first from one artery then from the other.

▸ **Assessment:** Rapid, uniform reddening of the hand in the areas supplied by the respective arteries indicates normal arterial supply. If vascular supply to the hand and fingers is compromised, the ischemic changes in the hand will recede only slowly.

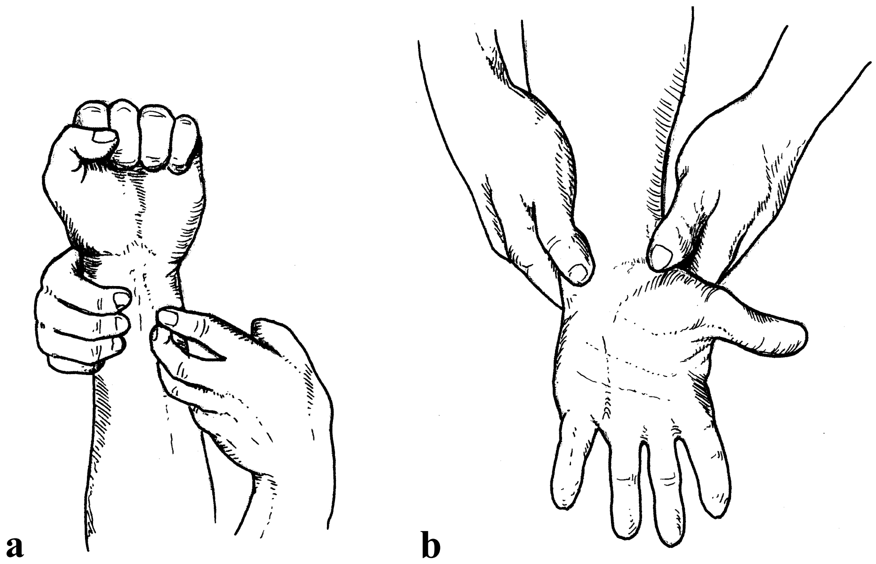


Fig. 10.1 Allen test. **(a)** Palpation of vessels with the arm raised. **(b)** Palpation of vessels with the arm hanging and evaluation of skin perfusion.

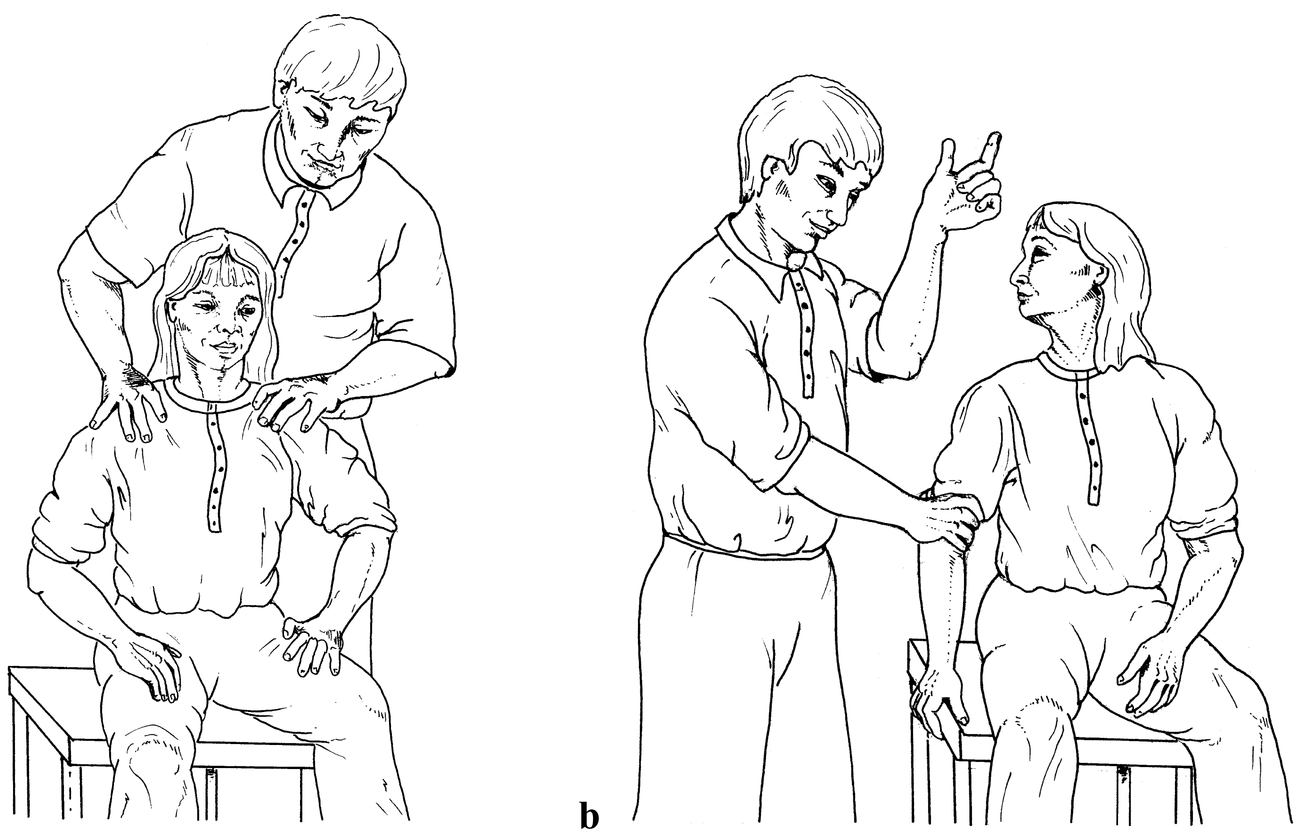
George Vertebral Artery Test (De Klyn Test)

Assessment of vertebral, basilar, or carotid artery stenosis or compression.

▸ **Procedure:** This test requires certain preliminary findings as it is not entirely without risk. Parameters requiring prior assessment include blood pressure, arm pulse, and pulses in the common carotid and subclavian arteries with auscultation to detect any murmurs or bruits. This test should not be performed if any of these prior examinations produces significantly abnormal findings. In the absence of any significant abnormalities, the seated patient is asked to maximally rotate his or her head to one side while extending the neck.

The test can also be performed with the patient supine, in which case the patient's head projects over the edge of the examining table and rests in the examiner's hands. Then with the head hanging down (in the De Klyn position), the head is maximally rotated and the neck extended. The head should remain or be held in maximum rotation and extension for about 20 to 30 seconds. The patient is then requested to count out loud.

□ **Assessment:** Abnormal auscultatory findings in the common carotid artery, vertigo, visual symptoms, nausea, fatigue, or nystagmus occurring during this maximum rotation and extension indicate stenosis of the vertebral artery or common carotid artery. The test is especially important in candidates for treatment (such as traction or manipulative therapy) of cervical spine symptoms associated with vertigo. The vertebral artery provocation test aids in the differential diagnosis because nausea, vertigo, and nystagmus initially increase but then rapidly decrease in intensity when a vertebral blockade is present. In the presence of vertebral artery insufficiency, the intensity of nausea and vertigo symptoms will rapidly increase within a few seconds without abating.



a b

Fig. 10.2 George vertebral artery test. **(a)** Starting position. **(b)** Rotation of the head and extension of the cervical spine.

Ratschow–Boerger Test

Assessment of vascular disease in the pelvis and legs.

□ **Procedure:** The supine patient is asked to raise the legs as high as possible and continuously rotate or plantar flex and dorsiflex the feet.

□ **Assessment:** Patients with normal vascular function will be able to perform this maneuver without any pain and without the soles of the feet becoming pale. Patients with compromised vascular function will experience varying degrees of pain and significant ischemia in the sole of the foot on the affected side. After about 2 minutes, the patient is requested to sit up quickly and let the legs hang over the edge of the examining table. Reactive hyperemia and refilling of the veins will occur within 5 to 7 seconds in patients with normal vascular function. In patients with compromised vascular function, this reaction will be delayed in proportion to the severity of vascular stenosis.

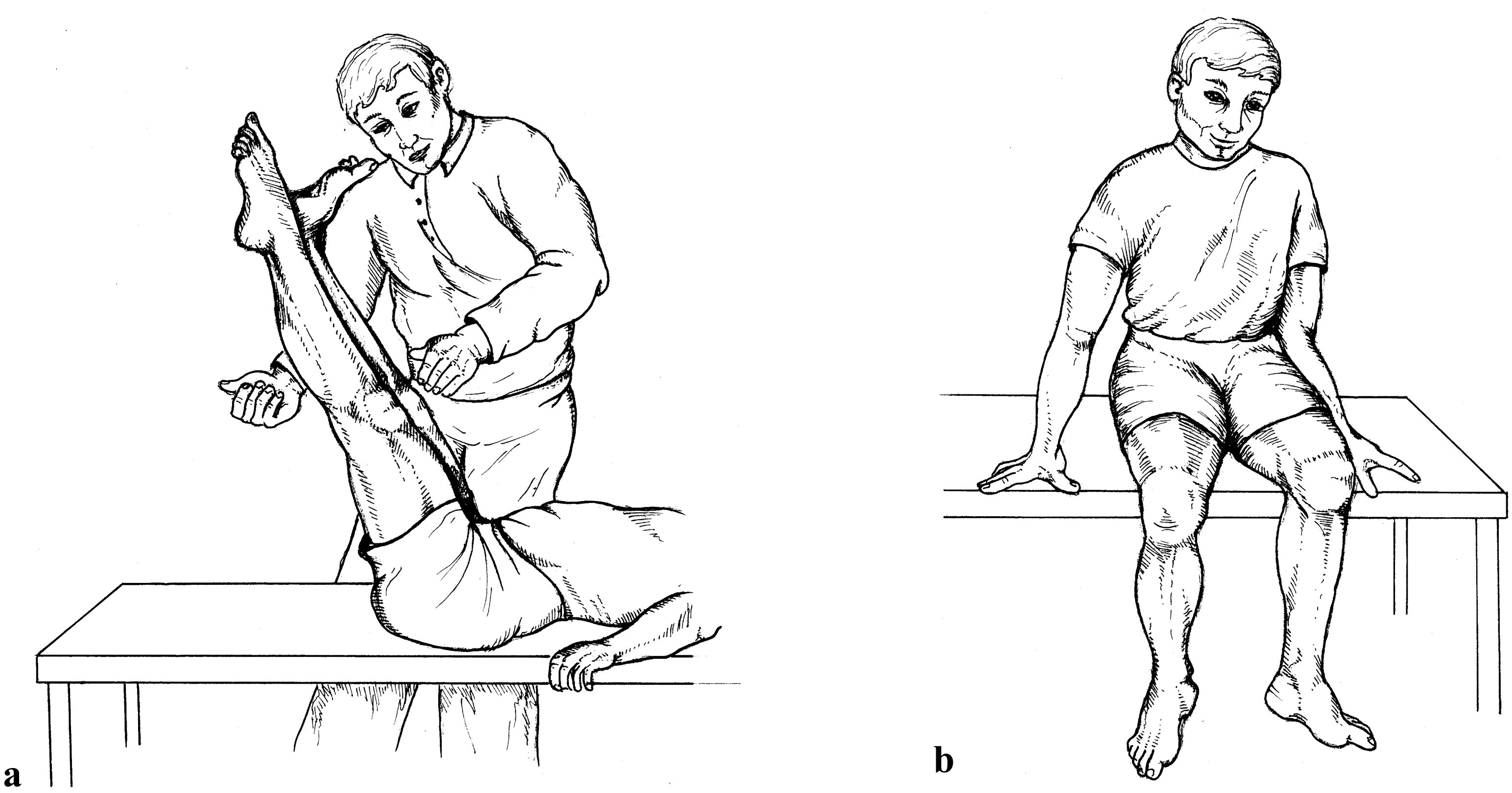


Fig. 10.3 Ratschow–Boerger test. **(a)** Patient supine with the legs raised. **(b)** Patient sitting with the legs hanging down over the edge of the examining table.

Thoracic Outlet Syndrome (TOS)

Thoracic outlet syndrome is a compression syndrome at the base of the neck with compromised neurovascular function. Thoracic outlet syndrome can be a congenital disorder resulting from factors such as a cervical rib, a superiorly displaced first rib, atypical ligaments, and the presence of an atypical scalenus minimus muscle. It may also be acquired as a result of callus formation, osteophytes on the clavicle and first rib, and changes in the scalene muscles such as fibrosis or hypertrophy.

This syndrome may be further differentiated according to the compression site as a cervical rib syndrome, first-rib syndrome, or scalene muscle syndrome.

TOS can coexist with other neurologic and vascular changes such as neurologic deficits and arterial and venous circulatory disorders. For this reason, a diagnosis of thoracic outlet syndrome is usually one of exclusion in which other central and peripheral causes have been eliminated.

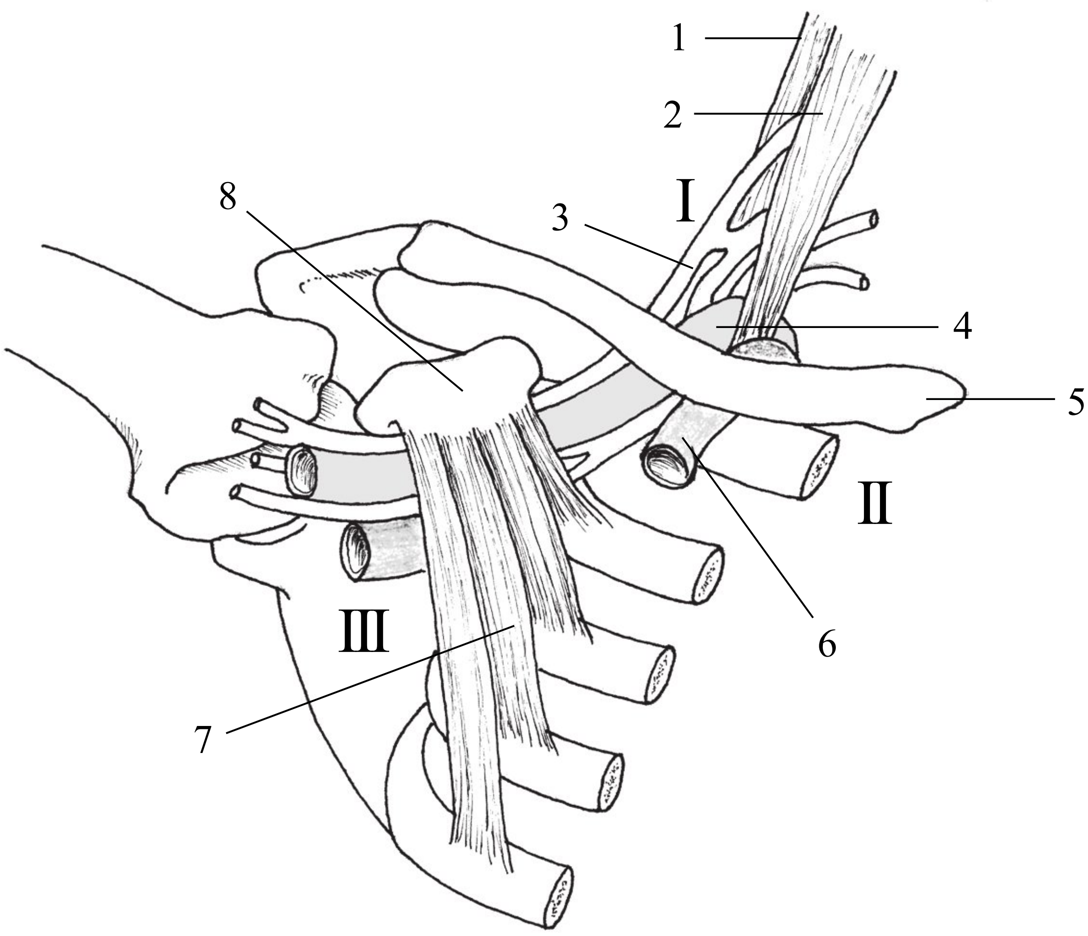


Fig. 10.4 Anatomical sites of compression.
I Scalene hiatus.
II Costoclavicular space.
III Subpectoral space.
1 Scalenus medius muscle.
2 Scalenus anterior muscle.
3 Brachial plexus.
4 Subclavian artery.
5 Clavicle.
6 Subclavian vein.
7 Pectoralis minor muscle.
8 Coracoid process.

Costoclavicular Test (Geisel Manipulation)

Assesses a neurovascular compression syndrome in the costoclavicular region.
 Procedure: The patient sits with the arms hanging relaxed. The examiner palpates the wrists to take the pulse in both radial arteries, noting amplitude and pulse rate. Then the patient abducts and externally rotates both arms and retracts the shoulders (Geisel position). With the patient in this position, the examiner again palpates the wrists and evaluates the pulse in both radial arteries.
 Assessment: Unilateral weakness or absence of the radial pulse, ischemic skin changes, and paresthesia are clear signs of compression of the brachial plexus and the axillary artery and vein in the costoclavicular region between the first rib and clavicle (costobrachial syndrome; droopy shoulder syndrome).

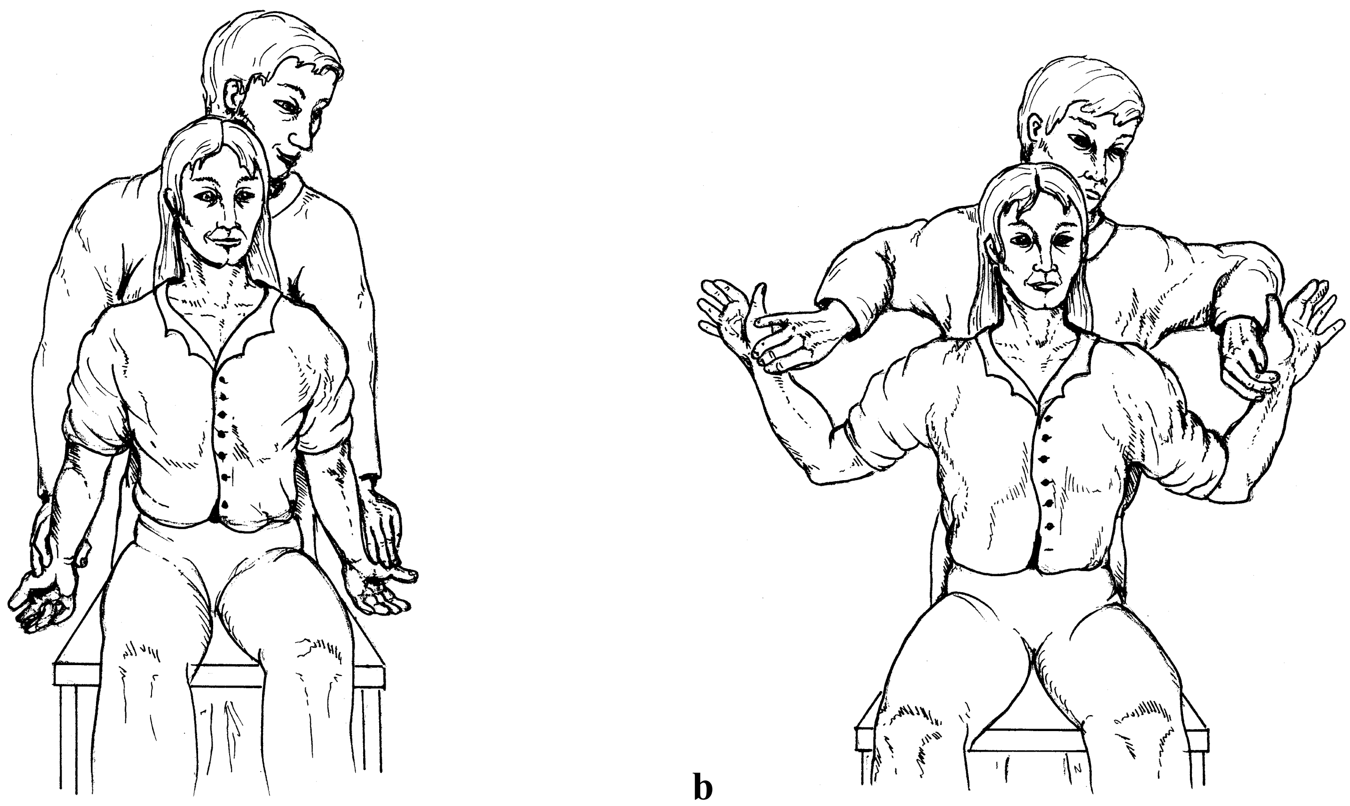


Fig. 10.5 Costoclavicular test. **(a)** Starting position with the examiner palpating the radial pulse. **(b)** Palpation of the radial pulse in abduction, with arms externally rotated and shoulders retracted.

Hyperabduction Test

Indicates a scalene muscle syndrome.

□ **Procedure:** The standing patient abducts both arms past 90° while retracting the shoulders. Then the patient alternately opens and closes both hands for 2 minutes.

□ **Assessment:** Pain in the shoulder and arm, ischemic skin changes, and paresthesia are clear signs of compression of the brachial plexus and/or subclavian artery in the lateral neck, which is primarily attributable to changes in the scalene muscles (fibrosis, hypertrophy, or presence of a scalenus minimus muscle). The scalene hiatus is located in the lateral neck, bordered anteriorly by the by the scalenus anterior muscle, posteriorly by the scalenus medius muscle, and inferiorly by the first rib. The brachial plexus and subclavian artery pass through the hiatus on their course to the axilla (Haven's syndrome).

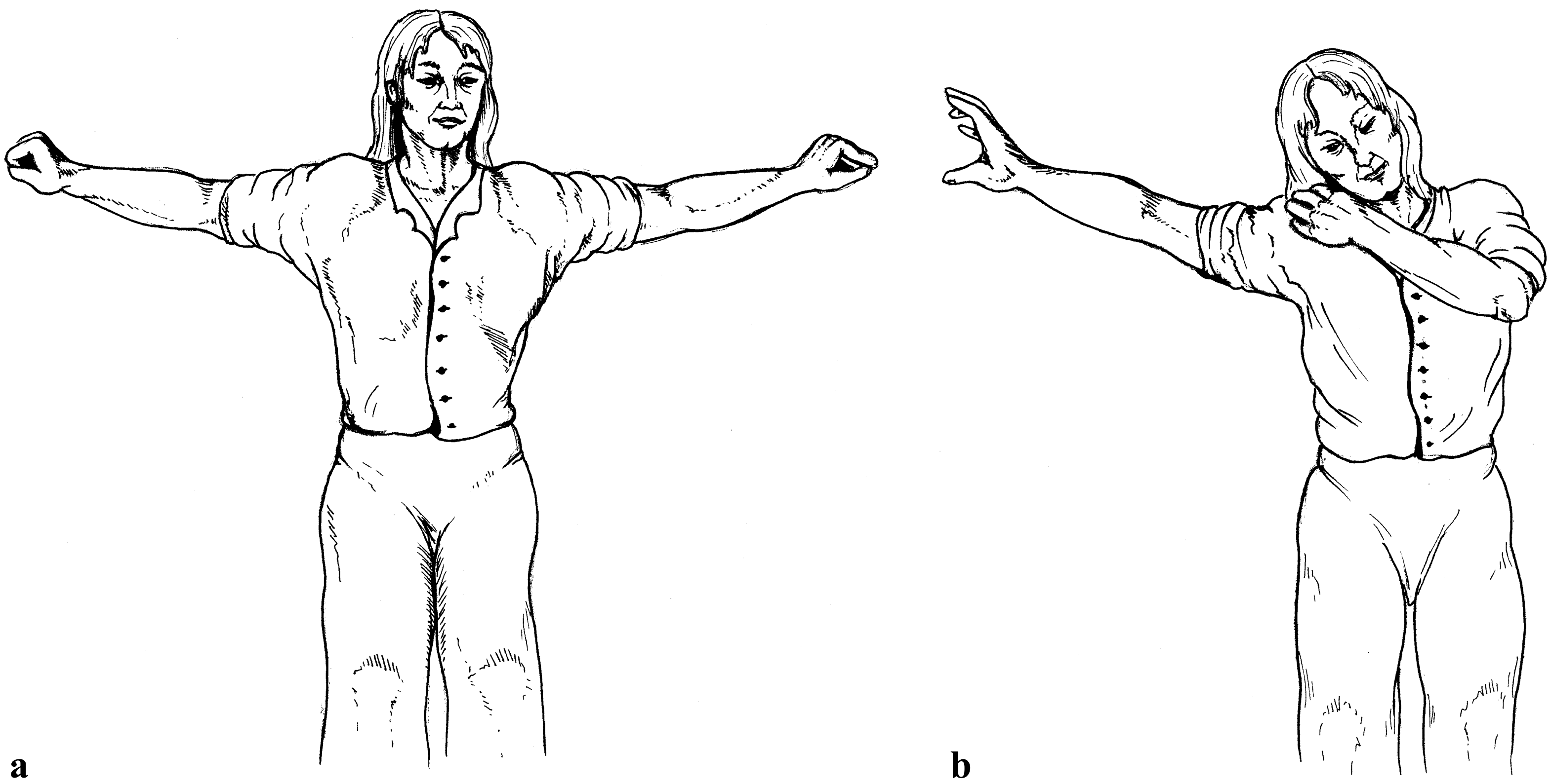


Fig. 10.6 Hyperabduction test. **(a)** Starting position with both arms abducted and shoulders retracted. **(b)** Pain elicited in right shoulder.

Intermittent Claudication Test

Produces the sign of a costoclavicular compression syndrome.

□ **Procedure:** The standing patient abducts and externally rotates both arms. Then the patient is instructed to rapidly flex and extend the fingers of each hand for 1 minute.

□ **Assessment:** If one arm begins to droop after a few cycles of finger motion and ischemic skin changes, paresthesia, and pain in the shoulder and arm occur, this suggests a costoclavicular compression syndrome affecting neurovascular structures.

Causes include osteophytes, rib changes, and anatomical variations in the scalene muscles.

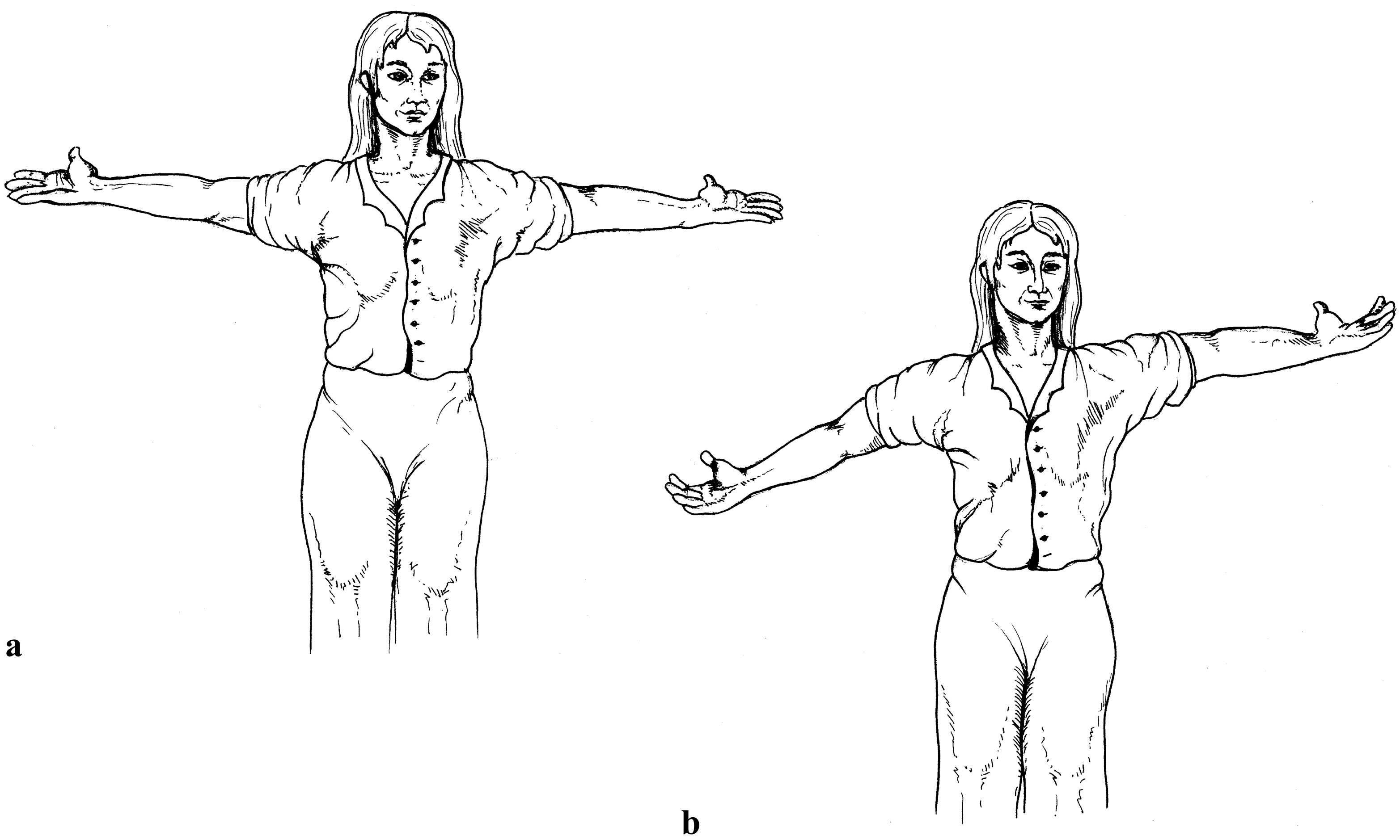
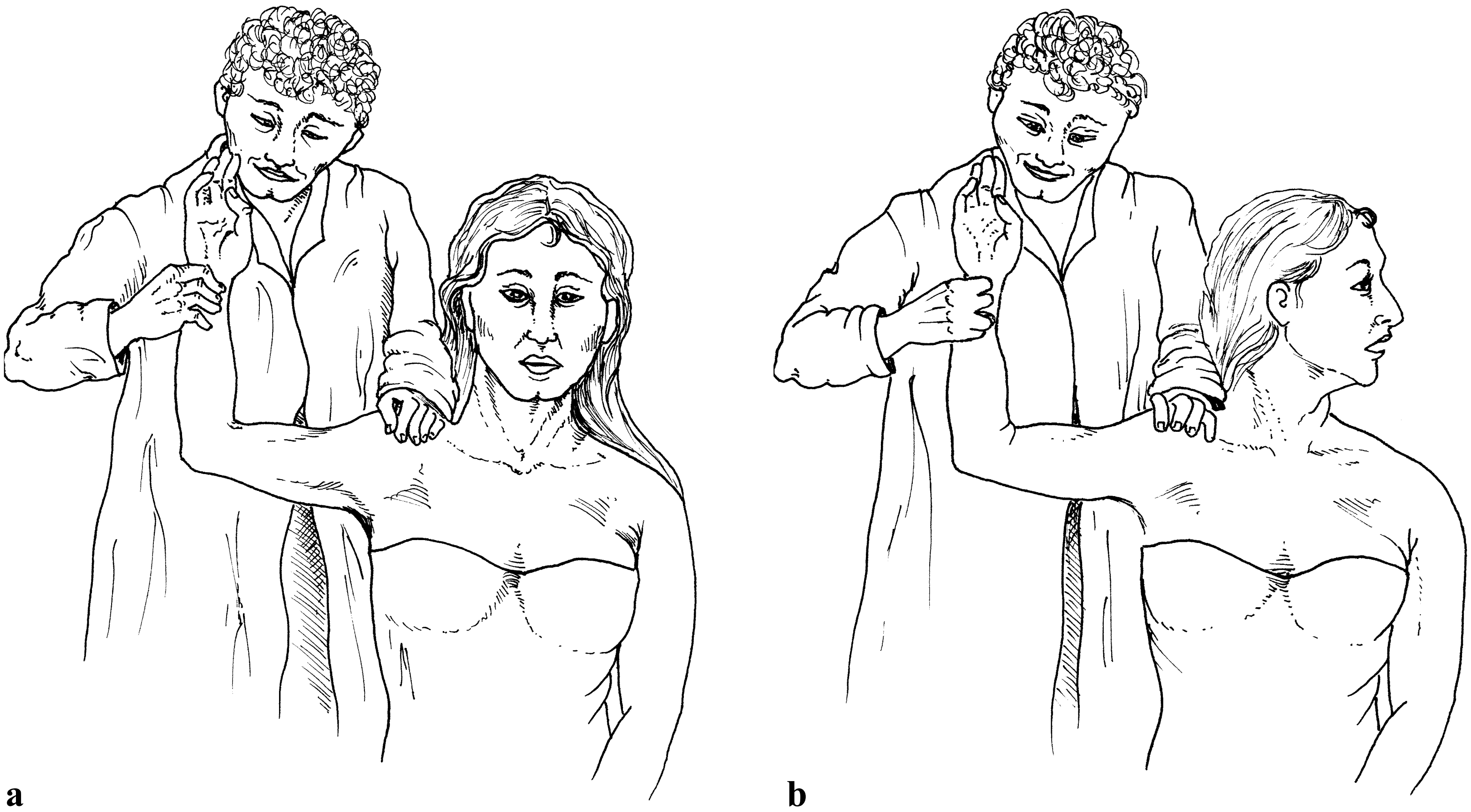


Fig. 10.7 Intermittent claudication test. **(a)** Starting position with both arms abducted and externally rotated. **(b)** Pain on the right side with drooping right arm.

Allen Maneuver

□ **Procedure:** The patient sits or stands. The examiner stands behind the patient and flexes the patient's elbow to 90° while the shoulder is abducted horizontally and placed into external rotation. The patient then rotates the head away from the test side.

□ **Assessment:** The examiner palpates the radial pulse, which disappears when the head is rotated. This disappearance indicates a positive test result for thoracic outlet syndrome, which may be caused by clavicular fractures with excessive callus or residual displacement of fragments, a cervical rib, a bifid clavicle, or abnormalities of the scalenus medius muscle.



a b

Fig. 10.8 Allen maneuver. **(a)** Starting position. **(b)** The patient rotates the head to the opposite side.

Wright Test

With repeated or continuous hyperabduction of the arm, the neurovascular structures in the axilla are stretched under the pectoral minor tendon and the coracoid process, resulting in a neurovascular syndrome.

□ **Procedure:** The patient is sitting or standing with arms hanging down loosely. The examiner palpates the patient's radial pulse. The examiner first tests the affected side by passively abducting the arm to 180° while checking the radial pulse and noting the angle at which the radial pulse weakens or disappears. Then he or she checks the other arm and compares the results.

□ **Assessment:** The test is positive if there is an obvious weakening of the radial pulse and/or neurologic symptoms in the tested arm within a short time. These symptoms include paresthesias and the hand "falling asleep" and possibly even Raynaud's phenomenon, as occurs when sleeping on one's stomach with the arms over the head. The compression occurs in the subpectoral space, between the coracoid process, the pectoralis minor, and the chest wall. The compression can be evaluated at the radial artery, because it arises directly from the axillary artery, which in turn arises from the subclavian artery. Both the brachial plexus and the axillary artery run through the area between the pectoralis minor muscle and the thorax. They usually become compressed between the coracoid process and the pectoralis minor muscle shortly before entering the axilla (see **Fig. 10.4**).

The following should be considered in the differential diagnosis:

- An infiltrative tumor (such as a Pancoast tumor) growing into the plexus.
- A tardive radiation-induced palsy.
- A neuroma within the brachial plexus.

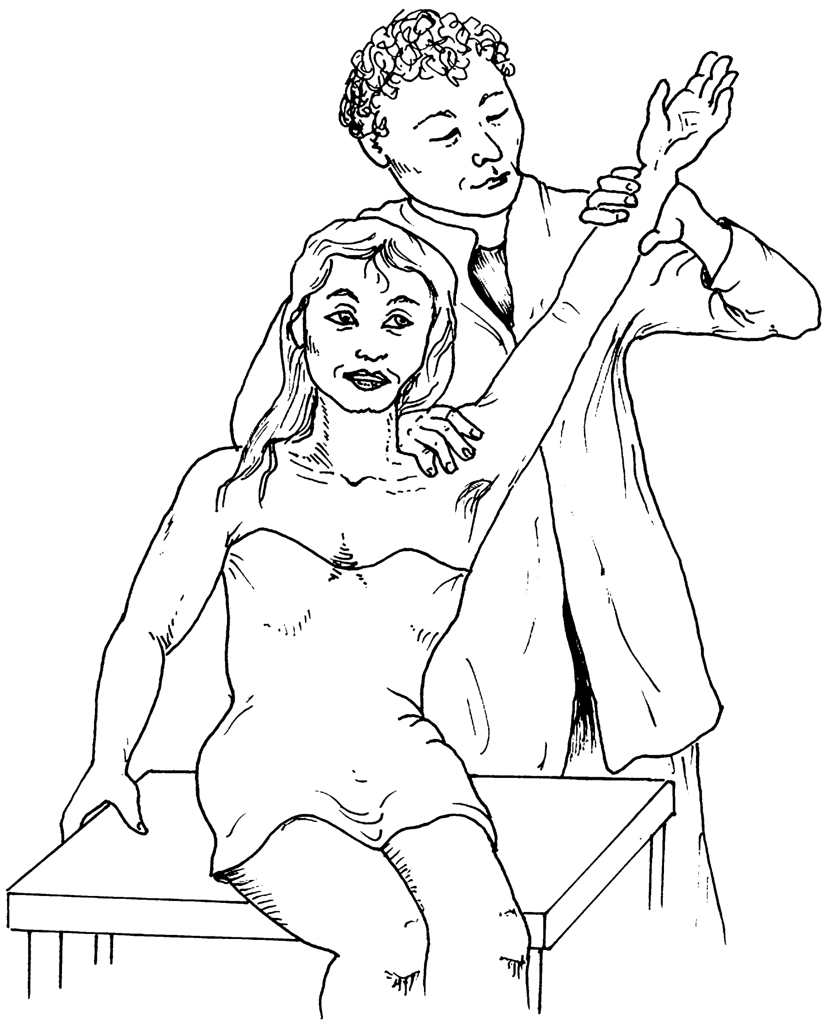


Fig. 10.9 Wright test.

Adson Test

□ **Procedure:** The patient's head is rotated to face the test shoulder. The patient then extends the head while the examiner externally rotates and extends the shoulder. The examiner locates the radial pulse and the patient is instructed to take a deep breath and hold it.

□ **Assessment:** A positive test occurs when the radial pulse disappears while the affected arm is raised laterally with simultaneous turning of the head to the same side.

The test is significant for identifying neurovascular compression of the subclavian artery and brachial plexus of the ipsilateral side, which is commonly caused by hypertrophy of the scalenus anterior muscle, the presence of a rudimentary cervical rib or a significantly widened transverse process of the seventh cervical vertebra with a fibrous band running from the process to the first rib.

A positive Adson test suggests a scalenus anticus syndrome, also called cervical rib syndrome, Adson's syndrome, or Naffziger's syndrome.

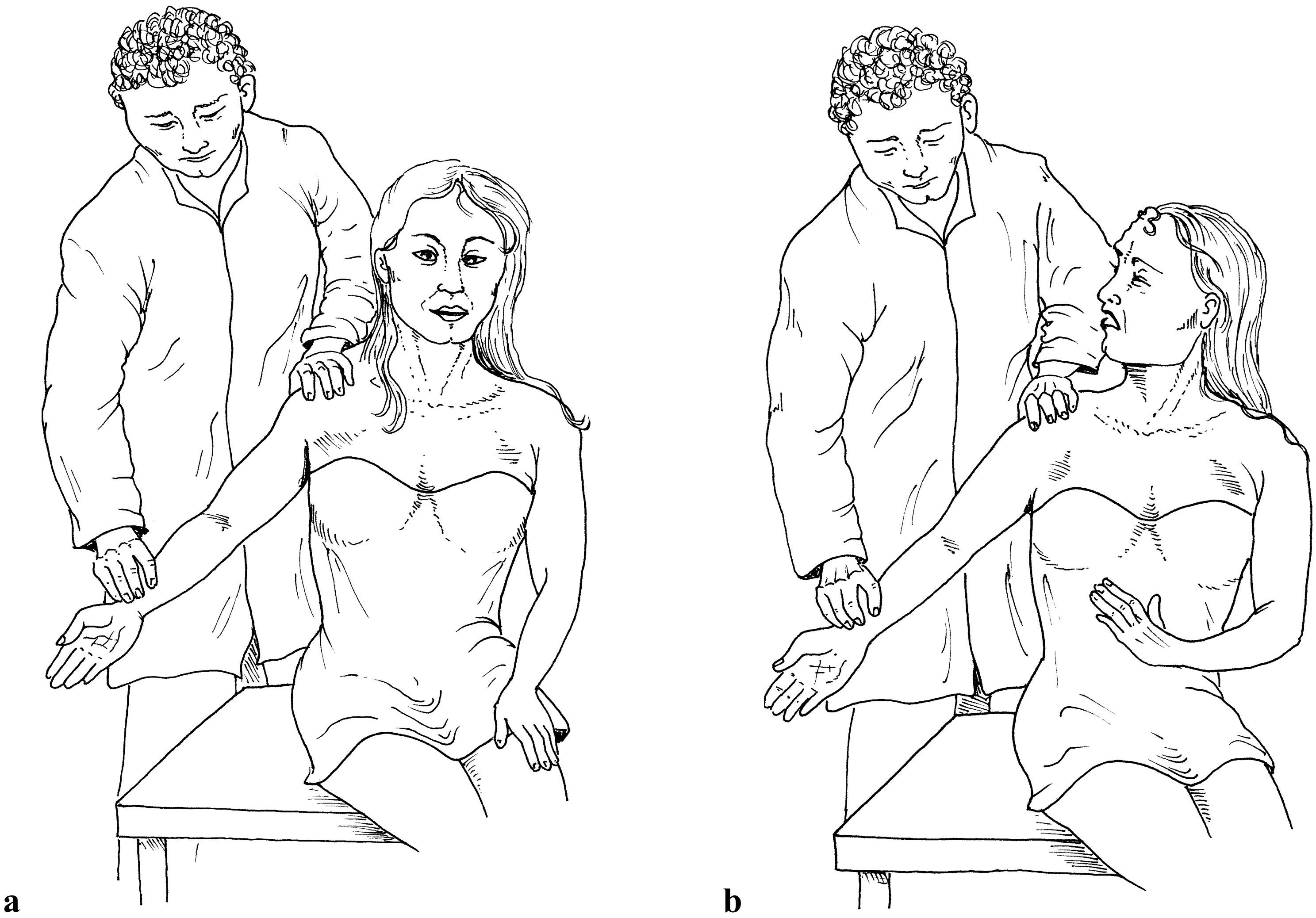


Fig. 10.10 Adson test. **(a)** Starting position. **(b)** The patient rotates the head toward the affected side and extends it.

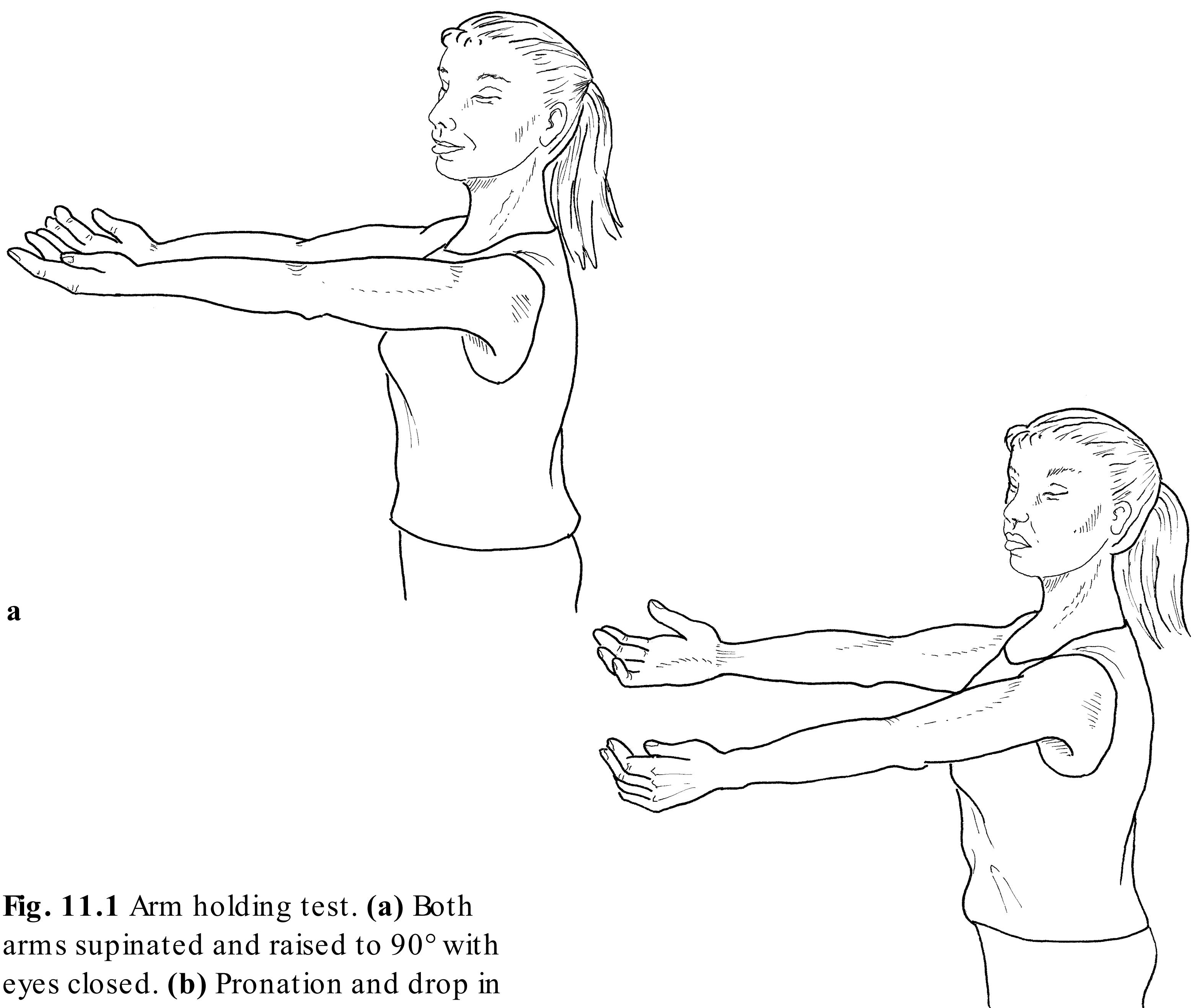
11 Disturbances of the Central Nervous System

Arm Holding Test

Used to grossly exclude hemiparesis.

□ **Procedure:** The patient is asked to supinate both arms and raise them to 90° while keeping his or her eyes closed.

□ **Assessment:** Pronation followed by a drop in one arm suggests latent central hemiparesis caused by a contralateral cerebral lesion. Where the arm first drops and then pronates with the patient's eyes closed, one should consider psychogenic influence.



a

b

Fig. 11.1 Arm holding test. **(a)** Both arms supinated and raised to 90° with eyes closed. **(b)** Pronation and drop in one arm.

Leg Holding Test

Used to grossly exclude central hemiparesis.

□ **Procedure:** From a supine position, the patient is asked to close his or her eyes and flex both hips and both knees to 90°.

□ **Assessment:** The neurologic examination of the lower extremities in a patient capable of standing and walking begins with inspection of stance and gait. The patient is asked to stand and walk on tiptoe and then on his or her heels. This will usually exclude any gross motor deficits. With the patient supine, the strength of the quadriceps is then tested by having the patient extend the knee against the examiner's resistance (L3–L4). Strength in the extensor digitorum and hallucis longus is tested by dorsiflexion of the toes (L5) against resistance, and strength in the triceps surae muscle is tested by plantar flexion of the foot (S1) against resistance. One or both lower legs dropping down during the leg holding test can be a sign of latent central hemiparesis.

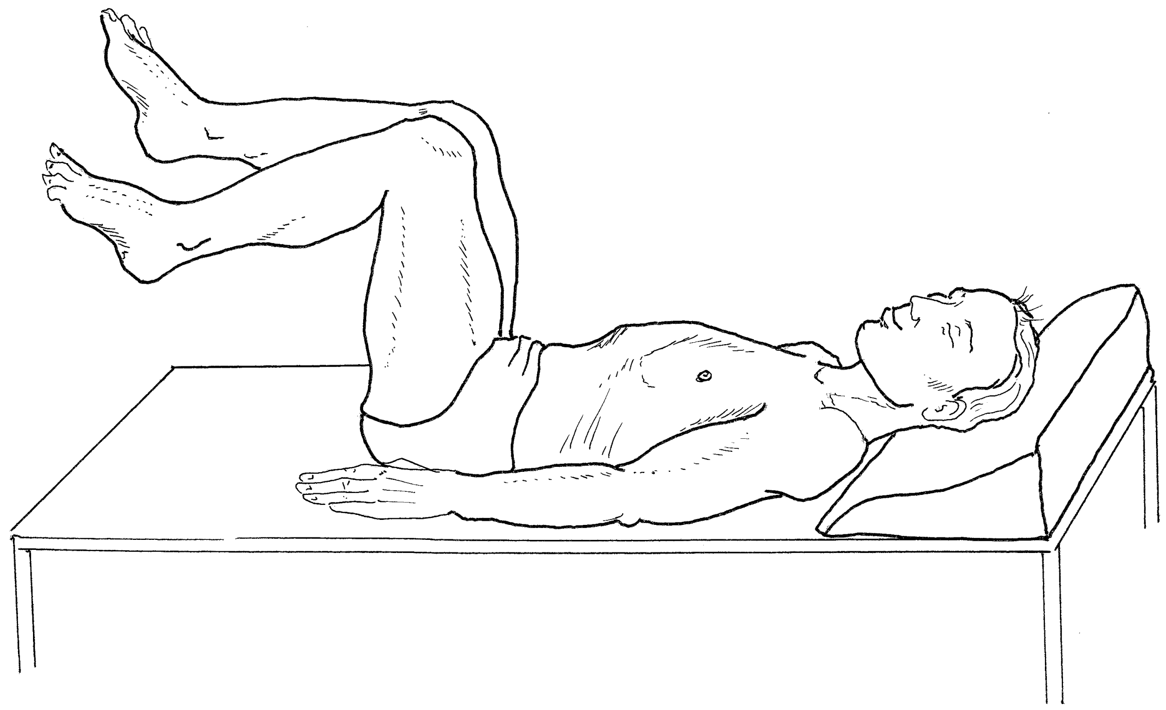


Fig. 11.2 Leg holding test.

References

Primary Texts

Ballmer FT, Lambert M, Hertel R. Napoleon's sign: a test to assess subscapularis function. J Shoulder Elbow Surg 1997;6(2):193

Bunnell P. Opposition of the thumb. J Bone Joint Surg 1938;20:269–284

Finkelstein H. Stenosing tendovaginitis at the radial styloid process. J Bone Joint Surg 1930;12:509–540

Hawkins RJ, Desmond JB. Clinical evaluation of shoulder problems. In: Rockwood CA, Matsen FA, eds. The Shoulder. 2nd ed. Philadelphia: Saunders; 1998:64–97

Hughston JC. The posterior cruciate ligament in knee joint stability. J Bone Joint Surg Am 1969;51:1045–1046

Hughston JC. Extensor mechanism examination. In: Fox JM, Del Pizzo W, eds. The Patellofemoral Joint. New York: McGraw-Hill;1993:63–74

Hughston JC, Andrews JR, Cross MJ, Moschi A. Classification of knee ligament instabilities. Part I: The medial compartement and cruciate ligaments. Part II: The lateral compartement. J Bone Joint Surg Am 1976;58:159–179

Jakob RP, Stäubli HU, Deland JT. Grading the pivot shift. Objective tests with implications for treatment. J Bone Joint Surg Br 1987;69:294–299

Jobe FW, Jobe CM. Painful athletic injuries of the shoulder. Clin Orthop Relat Res 1983;(173):117–124

Jobe FW, Nuber G. Throwing injuries of the elbow. Clin Sports Med 1986;5(4):621–636

Kibler WB. Specificity and sensitivity of the anterior slide test in throwing athletes with superior glenoid labral tears. Arthroscopy 1995;11(3):296–300

Losee RE. Concepts of the pivot shift. Clin Orthop Relat Res 1983; (172):45–51

Ludington NA. Rupture of the long head of the biceps cubiti muscle. Ann Surg 1923;77(3):358–363

Martens MA, Mulier JC. Anterior subluxation of the lateral tibial plateau. A new clinical test and the morbidity of this type of knee instability. Arch Orthop Trauma Surg 1981;98(2):109–111

Mennell J. Joint Manipulation. London: Churchill; 1952

Neer CS II. Involuntary inferior and multidirectional instability of the shoulder: etiology, recognition, and treatment. Instr Course Lect 1985;34:232–238

Noyes FR, Grood E, Torzilli PA. Current concepts review. The definition of terms for motion and position of the knee and injuries of the ligaments. J Bone Joint Surg Am 1989;71:465–472

O'Brien SJ, Pagnani MJ, Fealy S, McGlynn SR, Wilson JB. The active compression test: a new and effective test for diagnosing labral tears and acromioclavicular joint abnormality. Am J Sports Med 1998;26(5):610–613

Ortolani M. Un segno poco noto es sua importanza per la diagnosi precoce de prelussazione congenital dell'anca. Pediatria (Napoli) 1937;46:129–134

Phalen GS. The carpal-tunnel syndrome. Seventeen years' experience in diagnosis and treatment of six hundred fifty-four hands. J Bone Joint Surg Am 1966;48(2):211–228
Scheuermann HW. The classic: kyphosis dorsalis juvenilis. Clin Orthop Relat Res 1977;(128):5–7
Shelbourne KD, Benedict F, McCarroll JR, Rettig AC. Dynamic posterior shift test. An adjuvant in evaluation of posterior tibial subluxation. Am J Sports Med 1989;17(2):275–277
Slocum DB, Larson RL. Rotatory instability of the knee. Its pathogenesis and a clinical test to demonstrate its presence. J Bone Joint Surg Am 1968;50(2):211–225
Walch G, Boulahia A, Calderone S, Robinson AH. The 'dropping' and 'hornblower's' signs in evaluation of rotator-cuff tears. J Bone Joint Surg Br 1998;80(4):624–628
Watson HK, Ashmead D 4th, Makhlouf MV. Examination of the scaphoid. J Hand Surg Am 1988;13(5):657–660
Wilhelm K. Compression syndromes of the ulnar nerve and median nerve in the area of the hand. [Article in German.] Orthopade 1987;16(6):465–471
Yergason RM. Supination sign. J Bone Joint Surg 1931;13:160

Spine

Andersson GBJ, Deyo RA. History and physical examination in patients with herniated lumbar discs. Spine 1996; 21(24, Suppl)10S–18S
Barker S, Kesson M, Ashmore J, Turner G, Conway J, Stevens D. Professional issue. Guidance for pre-manipulative testing of the cervical spine. Man Ther 2000;5(1):37–40
Bland JH. Disorders of the Cervical Spine. Philadelphia: Saunders Co; 1994
Borge JA, Leboeuf-Yde C, Lothe J. Prognostic values of physical examination findings in patients with chronic low back pain treated conservatively: a systematic literature review. J Manipulative Physiol Ther 2001;24(4):292–295
Cameron DM, Bohannon RW, Owen SV. Influence of hip position on measurements of the straight leg raise test. J Orthop Sports Phys Ther 1994;19(3):168–172
Childs JD. One on one: the impact of the Valsalva maneuver during resistance exercise. Strength Condit J 1999;21:54–55
Christodoulides AN. Ipsilateral sciatica on femoral nerve stretch test is pathognomonic of an L4/5 disc protrusion. J Bone Joint Surg Br 1989;71(1):88–89
Devereaux MW. Neck and low back pain. Med Clin North Am 2003;87(3):643–662
Devillé WL, van der Windt DA, Dzaferagić A, Bezemer PD, Bouter LM. The test of Lasègue: systematic review of the accuracy in diagnosing herniated discs. Spine 2000;25(9):1140–1147
Dobbs AC. Evaluation of instabilities of the lumbar spine. Orthop Phys Ther Clin N Am 1999;8:387–400
Dreyfuss P, Michaelsen M, Pauza K, McLarty J, Bogduk N. The value of medical history and physical examination in diagnosing sacroiliac joint pain. Spine 1996;21(22):2594–2602
Dvorák J. Neurophysiologic tests in diagnosis of nerve root compression caused by disc herniation. Spine 1996;21(24, Suppl):39S–44S
Dvorak J, Antinnes JA, Panjabi M, Loustalot D, Bonomo M. Age and gender related normal motion of the cervical spine. Spine 1992;17(10, Suppl):S393–S398
Dyck P. The femoral nerve traction test with lumbar disc protrusions. Surg Neurol 1976;6(3):163–166

Elvey RL. The investigation of arm pain. In: Boyling JD, Palastanga N, eds. Grieve's Modern Manual Therapy: The Vertebral Column. 2nd ed. Edinburgh: Churchill Livingstone; 1994

Evans R, ed. Cervical spine. In: Evans R, ed. Illustrated Orthopaedic Physical Assessment. 2nd ed. St. Louis: CV Mosby; 2001

Fast A, Parikh S, Marin EL. The shoulder abduction relief sign in cervical radiculopathy. Arch Phys Med Rehabil 1989;70(5):402–403

Ginsburg GM, Bassett GS. Back pain in children and adolescents: evaluation and differential diagnosis. J Am Acad Orthop Surg 1997;5(2):67–78

Hall TM, Elvey RL. Nerve trunk pain: physical diagnosis and treatment. Man Ther 1999;4(2):63–73

Hoover CF. A new sign for the detection of malingering and functional paresis of the lower extremities. JAMA 1908;51:746–747

Hourigan CL, Bassett JM. Facet syndrome: clinical signs, symptoms, diagnosis, and treatment. J Manipulative Physiol Ther 1989;12(4):293–297

Johnson EK, Chiarello CM. The slump test: the effects of head and lower extremity position on knee extension. J Orthop Sports Phys Ther 1997;26(6):310–317

Jönsson B, Strömqvist B. The straight leg raising test and the severity of symptoms in lumbar disc herniation. A preoperative evaluation. Spine 1995;20(1):27–30

Kleinrensink GJ, Stoeckart R, Mulder PG, et al. Upper limb tension tests as tools in the diagnosis of nerve and plexus lesions. Anatomical and biomechanical aspects. Clin Biomech (Bristol, Avon) 2000;15(1):9–14

Koehler PJ, Okun MS. Important observations prior to the description of the Hoover sign. Neurology 2004;63(9):1693–1697

Krämer J. Intervertebral Disk Diseases. 3rd ed. Stuttgart: Thieme; 2009

Laslett M, Young SB, Aprill CN, McDonald B. Diagnosing painful sacroiliac joints: a validity study of a McKenzie evaluation and sacroiliac provocation tests. Aust J Physiother 2003;49(2):89–97

Lee DG. Rotational instability of the mid-thoracic spine: assessment and management. Man Ther 1996;1(5):234–241

Lew PC, Briggs CA. Relationship between the cervical component of the slump test and change in hamstring muscle tension. Man Ther 1997;2(2):98–105

Maitland GD. The slump test: examination and treatment. Aust J Physiother 1985;31(6):215–219

Malanga GA, Landes P, Nadler SF. Provocative tests in cervical spine examination: historical basis and scientific analyses. Pain Physician 2003;6(2):199–205

Mitchell J, Keene D, Dyson C, Harvey L, Pruvey C, Phillips R. Is cervical spine rotation, as used in the standard vertebrobasilar insufficiency test, associated with a measureable change in intracranial vertebral artery blood flow? Man Ther 2004;9(4):220–227

Nilsson N, Hartvigsen J, Christensen HW. Normal ranges of passive cervical motion for women and men 20–60 years old. J Manipulative Physiol Ther 1996;19(5):306–309

Perret C, Poiraudeau S, Fermanian J, Colau MM, Benhamou MA, Revel M. Validity, reliability, and responsiveness of the fingertip-to-floor test. Arch Phys Med Rehabil 2001;82(11):1566–1570

Rubinstein SM, Pool JJ, van Tulder MW, Riphagen II, de Vet HC. A systematic review of the diagnostic accuracy of provocative tests of the neck for diagnosing cervical radiculopathy. Eur Spine J 2007;16(3):307–319

Sandmark H, Nisell R. Validity of five common manual neck pain provoking tests. Scand J Rehabil Med 1995;27(3):131–136

Shacklock MO. Positive upper limb tension test in a case of surgically proven neuropathy: analysis and validity. Man Ther 1996;1(3):154–161

Shah KC, Rajshekhar V. Reliability of diagnosis of soft cervical disc prolapse using Spurling's test. Br J Neurosurg 2004;18(5):480–483

Spurling RG, Scoville WB. Lateral rupture of the cervical intervertebral discs: a common cause of shoulder and arm pain. Surg Gynecol Obstet 1944;78:350–358

Sullivan MS, Shoaf LD, Riddle DL. The relationship of lumbar flexion to disability in patients with low back pain. Phys Ther 2000;80(3):240–250

Supik LF, Broom MJ. Sciatic tension signs and lumbar disc herniation. Spine 1994;19(9):1066–1069

Thomas KE, Hasbun R, Jekel J, Quagliarello VJ. The diagnostic accuracy of Kernig's sign, Brudzinski's sign, and nuchal rigidity in adults with suspected meningitis. Clin Infect Dis 2002;35(1):46–52

Tong HC, Haig AJ, Yamakawa K. The Spurling test and cervical radiculopathy. Spine 2002;27(2):156–159

Torg JS, Ramsey-Emrhein JA. Cervical spine and brachial plexus injuries: return-to-play recommendations. Phys Sportsmed 1997;25(7):61–88

Walsh MJ. Evaluation of orthopedic testing of the low back for nonspecific lower back pain. J Manipulative Physiol Ther 1998;21(4):232–236

Wartenberg R. The signs of Brudzinski and of Kernig. J Pediatr 1950;37(4):679–684

White MA, Pape KE. The slump test. Am J Occup Ther 1992;46(3):271–274

Worth DR. Movements of the head and neck. In: Boyling JD, Palastanga N, eds. Grieve's Manual Therapy: The Vertebral Column. 2nd ed. Edinburgh: Churchill Livingstone; 1994

Youdas JW, Garrett TR, Suman VJ, Bogard CL, Hallman HO, Carey JR. Normal range of motion of the cervical spine: an initial goniometric study. Phys Ther 1992;72(11):770–780

Young S, Aprill C. Characteristics of a mechanical assessment for chronic lumbar facet joint pain. J Manual Manip Ther 2000;8:78–84

Young S, Aprill C, Laslett M. Correlation of clinical examination characteristics with three sources of chronic low back pain. Spine J 2003;3(6):460–465

Zaina C, Grant R, Johnson C, Dansie B, Taylor J, Spyropolous P. The effect of cervical rotation on blood flow in the contralateral vertebral artery. Man Ther 2003;8(2):103–109

Zito G, Jull G, Story I. Clinical tests of musculoskeletal dysfunction in the diagnosis of cervicogenic headache. Man Ther 2006;11(2):118–129

Shoulder

Andrews JR, Gillogly P. Physical examination of the shoulder in throwing athletes. In: Zairns B, Andrews J, Carson W, eds. Injuries to the Throwing Arms. Philadelphia: Saunders; 1985:51–65

Bahk M, Keyurapan E, Tasaki A, Sauers EL, McFarland EG. Laxity testing of the shoulder: a review. Am J Sports Med 2007;35(1):131–144

Ballmer FT, Lambert SM, Hertel R. Napoleon's sign: a test to assess subscapularis function. J Shoulder Elbow Surg 1997;6(2):193

Barth JRH, Burkhart SS, De Beer JF. The bear-hug test: a new and sensitive test for diagnosing a subscapularis tear. Arthroscopy 2006;22(10):1076–1084

Bartsch M, Greiner S, Haas NP, Scheibel M. Diagnostic values of clinical tests for subscapularis lesions. Knee Surg Sports Traumatol Arthrosc 2010;18(12):1712–1717

Berg EE, Ciullo JV. A clinical test for superior glenoid labral or 'SLAP' lesions. Clin J Sport Med 1998;8:121–123

Blonna D, Cecchetti S, Tellini A, et al. Contribution of the supraspinatus to the external rotator lag sign: kinematic and electromyographic pattern in an in vivo model. J Shoulder Elbow Surg 2010;19(3):392–398

Caliş M, Akgün K, Birtane M, Karacan I, Caliş H, Tüzün F. Diagnostic values of clinical diagnostic tests in subacromial impingement syndrome. Ann Rheum Dis 2000;59(1):44–47

Castoldi F, Blonna D, Hertel R. External rotation lag sign revisited: accuracy for diagnosis of full thickness supraspinatus tear. J Shoulder Elbow Surg 2009;18(4):529–534

Chao S, Thomas S, Yucha D, Kelly JD IV, Driban J, Swanik K. An electromyographic assessment of the "bear hug": an examination for the evaluation of the subscapularis muscle. Arthroscopy 2008;24(11):1265–1270

Chronopoulos E, Kim TK, Park HB, Ashenbrenner D, McFarland EG. Diagnostic value of physical tests for isolated chronic acromioclavicular lesions. Am J Sports Med 2004;32(3):655–661

Codman EA. The Shoulder: Ruptures of the Supraspinatus Tendon and Other Lesions in or About the Subacromial Bursa. Malabar FL: Krieger; 1934:146–155

De Wilde L, Plasschaert F, Berghs B, Van Hoecke M, Verstraete K, Verdonk R. Quantified measurement of subacromial impingement. J Shoulder Elbow Surg 2003;12(4):346–349

Emery RJ, Mullaji AB. Glenohumeral joint instability in normal adolescents. Incidence and significance. J Bone Joint Surg Br 1991;73(3):406–408

Gagey OJ, Gagey N. The hyperabduction test. J Bone Joint Surg Br 2001;83(1):69–74

Gerber C, Ganz R. Clinical assessment of instability of the shoulder. With special reference to anterior and posterior drawer tests. J Bone Joint Surg Br 1984;66(4):551–556

Gross ML, Distefano MC. Anterior release test. A new test for occult shoulder instability. Clin Orthop Relat Res 1997; (339):105–108

Guanche CA, Jones DC. Clinical testing for tears of the glenoid labrum. Arthroscopy 2003;19(5):517–523

Hawkins RJ, Bokor DJ. Clinical evaluation of shoulder problems. In: Rockwood CA, Matsen FA III, eds. The Shoulder. Vol 1. Philadelphia: Saunders; 1990:149–177

Hawkins RJ, Desmond JB. Clinical evaluation of shoulder problems. In: Rockwood CA, Matsen FA, eds. The Shoulder. 2nd ed. Philadelphia: Saunders; 1998:189

Hawkins RJ, Kennedy JC. Impingement syndrome in athletes. Am J Sports Med 1980;8(3):151–158

Hertel R, Ballmer FT, Lombert SM, Gerber C. Lag signs in the diagnosis of rotator cuff rupture. J Shoulder Elbow Surg 1996;5(4):307–313

Hurschler C, Wülker N, Windhagen H, Hellmers N, Plumhoff P. Evaluation of the lag sign tests for external rotator function of the shoulder. J Shoulder Elbow Surg 2004;13(3):298–304

Ide M, Ide J, Yamaga M, Takagi K. Symptoms and signs of irritation of the brachial plexus in whiplash injuries. J Bone Joint Surg Br 2001;83(2):226–229

Jobe FW, Jobe CM. Painful athletic injuries of the shoulder. Clin Orthop Relat Res 1983; (173):117–124

Kelly JJ. Neurological problems in the athlete's shoulder. In: Pettrone FA, ed. Athletic Injuries of the Shoulder. New York: McGraw-Hill; 1995

Kibler WB. Specificity and sensitivity of the anterior slide test in throwing athletes with superior glenoid labral tears. Arthroscopy 1995;11(3):296–300

Kibler WB. The role of the scapula in athletic shoulder function. Am J Sports Med 1998;26(2):325–337 Review

Kibler WB, McMullen J. Scapular dyskinesis and its relation to shoulder pain. J Am Acad Orthop Surg 2003;11(2):142–151
Kibler WB, Sciascia A. Current concepts: scapular dyskinesis. Br J Sports Med 2010;44(5):300–305
Kim SH, Park JC, Park JS, Oh I. Painful jerk test: a predictor of success in nonoperative treatment of posteroinferior instability of the shoulder. Am J Sports Med 2004;32(8):1849–1855
Kirkley A, Litchfield RB, Jackowski DM, Lo IK. The use of the impingement test as a predictor of outcome following subacromial decompression for rotator cuff tendinosis. Arthroscopy 2002;18(1):8–15
Kirkley A, Nonweiler B, Lo IKY, Woolfrey M. Validation of the apprehension relocation and surprise tests in the diagnosis of anterior shoulder instability. J Bone Joint Surg Br 1997;79B:75
Kölbel R. A modification of the relocation test: arthroscopic findings associated with a positive test. J Shoulder Elbow Surg 2001;10(5):497–498
Koslow PA, Prosser LA, Strony GA, Suchecki SL, Mattingly GE. Specificity of the lateral scapular slide test in asymptomatic competitive athletes. J Orthop Sports Phys Ther 2003;33(6):331–336
Limb D. How I examine the shoulder: a guide from the expert. Curr Orthop 2000;14:435–440
Lo IK, Nonweiler B, Woolfrey M, Litchfield R, Kirkley A. An evaluation of the apprehension, relocation, and surprise tests for anterior shoulder instability. Am J Sports Med 2004;32(2):301–307
Ludington NA. Rupture of the long head of the biceps flexor cubiti muscle. Ann Surg 1923;77(3):358–363
MacDonald PB, Clark P, Sutherland K. An analysis of the diagnostic accuracy of the Hawkins and Neer subacromial impingement signs. J Shoulder Elbow Surg 2000;9(4):299–301
McFarland EG, Kim TK, Savino RM. Clinical assessment of three common tests for superior labral anterior-posterior lesions. Am J Sports Med 2002;30(6):810–815
Meister K, Buckley B, Batts J. The posterior impingement sign: diagnosis of rotator cuff and posterior labral tears secondary to internal impingement in overhand athletes. Am J Orthop 2004;33(8):412–415
Mimori K, Muneta T, Nakagawa T, Shinomiya K. A new pain provocation test for superior labral tears of the shoulder. Am J Sports Med 1999;27(2):137–142
Neer CS II. Anterior acromioplasty for the chronic impingement syndrome in the shoulder: a preliminary report. J Bone Joint Surg Am 1972;54(1):41–50
Neer CS II. Involuntary inferior and multidirectional instability of the shoulder: etiology, recognition, and treatment. Instr Course Lect 1985;34:232–238
O'Brien SJ, Pagnani MJ, Fealy S, McGlynn SR, Wilson JB. The active compression test: a new and effective test for diagnosing labral tears and acromioclavicular joint abnormality. Am J Sports Med 1998;26(5):610–613
Park HB, Yokota A, Gill HS, El Rassi G, McFarland EG. Diagnostic accuracy of clinical tests for the different degrees of subacromial impingement syndrome. J Bone Joint Surg Am 2005;87(7):1446–1455
Plewa MC, Delinger M. The false-positive rate of thoracic outlet syndrome shoulder maneuvers in healthy subjects. Acad Emerg Med 1998;5(4):337–342
Rigsby R, Sitler M, Kelly JD. Subscapularis tendon integrity: an examination of shoulder index tests. J Athl Train 2010;45(4):404–406
Rockwood CA, Matsen FA. The Shoulder. Vol 1. 2nd ed. Philadelphia: Saunders; 1998

Rowe CR, Zarins B. Recurrent transient subluxation of the shoulder. J Bone Joint Surg Am 1981;63(6):863–872
Scheibel M, Magosch P, Pritsch M, Lichtenberg S, Habermeyer P. The belly-off sign: a new clinical diagnostic sign for subscapularis lesions. Arthroscopy 2005;21(10):1229–1235
Stetson WB, Templin K. The crank test, the O'Brien test, and routine magnetic resonance imaging scans in the diagnosis of labral tears. Am J Sports Med 2002;30(6):806–809
Tzannes A, Paxinos A, Callanan M, Murrell GA. An assessment of the interexaminer reliability of tests for shoulder instability. J Shoulder Elbow Surg 2004;13(1):18–23
Valadie AL III, Jobe CM, Pink MM, Ekman EF, Jobe FW. Anatomy of provocative tests for impingement syndrome of the shoulder. J Shoulder Elbow Surg 2000;9(1):36–46
Walch G, Boulahia A, Calderone S, Robinson AHN. The 'dropping' and 'hornblower's' signs in evaluation of rotator-cuff tears. J Bone Joint Surg Br 1998;80(4):624–628
Yergason RM. Supination sign. J Bone Joint Surg. 1931;13:160
Zaslav KR. Internal rotation resistance strength test: a new diagnostic test to differentiate intra-articular pathology from outlet (Neer) impingement syndrome in the shoulder. J Shoulder Elbow Surg 2001;10(1):23–27

Elbow

Alfonso MI, Dzwierzynski W. Hoffman-Tinel sign. The realities. Phys Med Rehabil Clin N Am 1998;9(4):721–736, v
Anderson TE. Anatomy and physical examination of the elbow. In: Nicholas JA, Hershmann EB, eds. The Upper Extremity in Sports Medicine. St. Louis: CV Mosby; 1990
Andrews JR, Wilk KE, Satterwhite YE, Tedder JL. Physical examination of the thrower's elbow. J Orthop Sports Phys Ther 1993;17(6):296–304
Benjamin SJ, Williams DA, Kalbfleisch JH, Gorman PW, Panus PC. Normalized forces and active range of motion in unilateral radial epicondylalgia (tennis elbow). J Orthop Sports Phys Ther 1999;29(11):668–676
Buehler MJ, Thayer DT. The elbow flexion test. A clinical test for the cubital tunnel syndrome. Clin Orthop Relat Res 1988; (233):213–216
Byrd JW. Evaluation of the hip: history and physical examination. N Am J Sports Phys Ther 2007;2(4):231–240
Cohen MS, Hastings H II. Rotatory instability of the elbow. The anatomy and role of the lateral stabilizers. J Bone Joint Surg Am 1997;79(2):225–233
Gruebel-Lee DM. Disorders of the Hip. Philadelphia: J.B. Lippincott; 1983
Hannouche D, Bégué T. Functional anatomy of the lateral collateral ligament complex of the elbow. Surg Radiol Anat 1999;21(3):187–191
Jobe FW, Nuber G. Throwing injuries of the elbow. Clin Sports Med 1986;5(4):621–636
Kalb K, Gruber P, Landsleitner B. Compression syndrome of the radial nerve in the area of the supinator groove. Experiences with 110 patients. [Article in German.] Handchir Mikrochir Plast Chir 1999;31(5):303–310
Landi A, Copeland S. Value of the Tinel sign in brachial plexus lesions. Ann R Coll Surg Engl 1979;61(6):470–471
Leach RE, Miller JK. Lateral and medial epicondylitis of the elbow. Clin Sports Med 1987;6(2):259–272
Lee ML, Rosenwasser MP. Chronic elbow instability. Orthop Clin North Am 1999;30(1):81–89

Leibold MR, Huijbregts PA, Jensen R. Concurrent criterion-related validity of physical examination tests for hip labral lesions: a systematic review. J Manual Manip Ther 2008;16(2):E24–E41

London JT. Kinematics of the elbow. J Bone Joint Surg Am 1981;63(4):529–535

MacDermid JC, Michlovitz SL. Examination of the elbow: linking diagnosis, prognosis, and outcomes as a framework for maximizing therapy interventions. J Hand Ther 2006;19(2):82–97

Martin HD, Kelly BT, Leunig M, et al. The pattern and technique in the clinical evaluation of the adult hip: the common physical examination tests of hip specialists. Arthroscopy 2010;26(2):161–172

Martin RL, Sekiya JK. The interrater reliability of 4 clinical tests used to assess individuals with musculoskeletal hip pain. J Orthop Sports Phys Ther 2008;38(2):71–77

McCall BR, Cain EL Jr. Diagnosis, treatment, and rehabilitation of the thrower's elbow. Curr Sports Med Rep 2005;4(5):249–254

McPherson SA, Meals RA. Cubital tunnel syndrome. Orthop Clin North Am 1992;23(1):111–123

Mehta JA, Bain GI. Posterolateral rotatory instability of the elbow. J Am Acad Orthop Surg 2004;12(6):405–415

Morrey BF. Acute and chronic instability of the elbow. J Am Acad Orthop Surg 1996;4(3):117–128

Novak CB, Lee GW, Mackinnon SE, Lay L. Provocative testing for cubital tunnel syndrome. J Hand Surg Am 1994;19(5):817–820

O'Driscoll SW, Bell DF, Morrey BF. Posterolateral rotatory instability of the elbow. J Bone Joint Surg Am 1991;73(3):440–446

O'Driscoll SW. Classification and evaluation of recurrent instability of the elbow. Clin Orthop Relat Res 2000; (370):34–43

O'Driscoll SW, Lawton RL, Smith AM. The "moving valgus stress test" for medial collateral ligament tears of the elbow. Am J Sports Med 2005;33(2):231–239

Phalen GS. The carpal-tunnel syndrome. Clinical evaluation of 598 hands. Clin Orthop Relat Res 1972;83(83):29–40

Plancher KD, Halbrecht J, Lourie GM. Medial and lateral epicondylitis in the athlete. Clin Sports Med 1996;15(2):283–305

Rayan GM, Jensen C, Duke J. Elbow flexion test in the normal population. J Hand Surg Am 1992;17(1):86–89

Samora JB, Ng VY, Ellis TJ. Femoroacetabular impingement: a common cause of hip pain in young adults. Clin J Sport Med 2011;21(1):51–56

Thériault G, Lachance P. Golf injuries. An overview. Sports Med 1998;26(1):43–57

Yocum LA. The diagnosis and nonoperative treatment of elbow problems in the athlete. Clin Sports Med 1989;8(3):439–451

Wrist, Hand, and Fingers

Bechtol CO. Grip test; the use of a dynamometer with adjustable handle spacings. J Bone Joint Surg Am 1954;36-A(4):820–824, passim

Bednar JM, Osterman AL. Carpal instability: evaluation and treatment. J Am Acad Orthop Surg 1993;1(1):10–17

Bickert B, Sauerbier M, Germann G. Clinical examination of the injured wrist. [Article in German.] Zentralbl Chir 1997;122(11):1010–1015

Bozek M, Gaździk TS. The value of clinical examination in the diagnosis of carpal tunnel syndrome. Ortop Traumatol Rehabil 2001;3(3):357–360

Brüske J, Bednarski M, Grzelec H, Zyluk A. The usefulness of the Phalen test and the Hoffmann-Tinel sign in the diagnosis of carpal tunnel syndrome. Acta Orthop Belg 2002;68(2):141–145

Buch-Jaeger N, Foucher G. Correlation of clinical signs with nerve conduction tests in the diagnosis of carpal tunnel syndrome. J Hand Surg [Br] 1994;19(6):720–724

Bunnell P. Opposition of the thumb. J Bone Joint Surg 1938;20:269–284

Burke FD. Carpal tunnel syndrome: reconciling "demand management" with clinical need. J Hand Surg 2000;25(2):121–127

Campbell DA. How I examine the wrist. Curr Orthop 2001;14:342–346

Finkelstein H. Stenosing tendovaginitis at the radial styloid process. J Bone Joint Surg 1930;12:509

Forman TA, Forman SK, Rose NE. A clinical approach to diagnosing wrist pain. Am Fam Physician 2005;72(9):1753–1758

Gelberman RH, Blasingame JP. The timed Allen test. J Trauma 1981;21(6):477–479

Gelberman RH, Eaton R, Urbaniak JR. Peripheral nerve compression. J Bone Joint Surg Am 1993;75:1854–1878

Gelmers HJ. The significance of Tinel's sign in the diagnosis of carpal tunnel syndrome. Acta Neurochir (Wien) 1979;49(3-4):255–258

Gunnarsson LG, Amilon A, Hellstrand P, Leissner P, Philipson L. The diagnosis of carpal tunnel syndrome. Sensitivity and specificity of some clinical and electrophysiological tests. J Hand Surg [Br] 1997;22(1):34–37

Henderson WR. Clinical assessment of peripheral nerve injuries; Tinel's test. Lancet 1948;2(6534):801–805

Hoffmann P, Buck-Gramcko D, Lubahn JD. The Hoffmann-Tinel sign. 1915. J Hand Surg [Br] 1993;18(6):800–805

Hwang JJ, Goldfarb CA, Gelberman RH, Boyer MI. The effect of dorsal carpal ganglion excision on the scaphoid shift test. J Hand Surg [Br] 1999;24(1):106–108

Johnson RP, Carrera GF. Chronic capitolunate instability. J Bone Joint Surg Am 1986;68(8):1164–1176

Kanaan N, Sawaya RA. Carpal tunnel syndrome: modern diagnostic and management techniques. Br J Gen Pract 2001;51(465):311–314

Kuhlman KA, Hennessey WJ. Sensitivity and specificity of carpal tunnel syndrome signs. Am J Phys Med Rehabil 1997;76(6):451–457

Lane LB. The scaphoid shift test. J Hand Surg Am 1993;18(2):366–368

LaStayo P, Howell J. Clinical provocative tests used in evaluating wrist pain: a descriptive study. J Hand Ther 1995;8(1):10–17

Mathiowetz V, Weber K, Volland G, Kashman N. Reliability and validity of grip and pinch strength evaluations. J Hand Surg Am 1984;9(2):222–226

McConnell EA. Performing Allen's test. Nursing 1997;27(11):26

Mondelli M, Passero S, Giannini F. Provocative tests in different stages of carpal tunnel syndrome. Clin Neurol Neurosurg 2001;103(3):178–183

Murtagh J. De Quervain's tenosynovitis and Finkelstein's test. Aust Fam Physician 1989;18(12):1552

Nagle DJ. Evaluation of chronic wrist pain. J Am Acad Orthop Surg 2000;8(1):45–55

Nichols CM, Cheng C. Update on the evaluation of wrist pain. Mo Med 2006;103(3):293–296

Phalen GS. The carpal-tunnel syndrome. Seventeen years' experience in diagnosis and treatment of six hundred fifty-four hands. J Bone Joint Surg Am 1966;48(2):211–228

Reagan DS, Linscheid RL, Dobyns JH. Lunotriquetral sprains. J Hand Surg Am 1984;9(4):502–514
Ruby LK. Carpal instability. J Bone Joint Surg Am 1995;77:476–487
Ruby LK, An KN, Linscheid RL, Cooney WP III, Chao EY. The effect of scapholunate ligament section on scapholunate motion. J Hand Surg Am 1987;12(5 Pt 1):767–771
Rush J. De Quervain's disease. Curr Orthop 2000;14:380–383
Schuett AM, Gieck J, McCue FC. Evaluation and treatment of injuries to the thumb and fingers. Orthop Phys Ther Clin N Am 1994;3:367–383
Shin AY, Battaglia MJ, Bishop AT. Lunotriquetral instability: diagnosis and treatment. J Am Acad Orthop Surg 2000;8(3):170–179
Skirven T. Clinical examination of the wrist. J Hand Ther 1996;9(2):96–107
Spinner M. Management of nerve compression lesions of the upper extremity. In: Omer GE, Spinner M, eds. Management of Peripheral Nerve Problems. Philadelphia: Saunders; 1980
Szabo RM, Slater RR Jr, Farver TB, Stanton DB, Sharman WK. The value of diagnostic testing in carpal tunnel syndrome. J Hand Surg Am 1999;24(4):704–714
Tetro AM, Evanoff BA, Hollstien SB, Gelberman RH. A new provocative test for carpal tunnel syndrome. Assessment of wrist flexion and nerve compression. J Bone Joint Surg Br 1998;80(3):493–498
Thompson CE, Stroud SD. Allen's test: a tool for diagnosing ulnar artery trauma. Nurse Pract 1984;9(12):13,16–17
Watson HK, Ashmead D 4th, Makhlouf MV. Examination of the scaphoid. J Hand Surg 1988;13(5):657–660
Wolfe SW, Gupta A, Crisco JJ III. Kinematics of the scaphoid shift test. J Hand Surg Am 1997;22(5):801–806

Hip

Asayama I, Naito M, Fujisawa M, Kambe T. Relationship between radiographic measurements of reconstructed hip joint position and the Trendelenburg sign. J Arthroplasty 2002;17(6):747–751
Bartlett MD, Wolf LS, Shurtleff DB, Stahell LT. Hip flexion contractures: a comparison of measurement methods. Arch Phys Med Rehabil 1985;66(9):620–625
Beck M, Kalhor M, Leunig M, Ganz R. Hip morphology influences the pattern of damage to the acetabular cartilage: femoroacetabular impingement as a cause of early osteoarthritis of the hip. J Bone Joint Surg Br 2005;87(7):1012–1018
Brady RJ, Dean JB, Skinner TM, Gross MT. Limb length inequality: clinical implications for assessment and intervention. J Orthop Sports Phys Ther 2003;33(5):221–234
Broadhurst NA, Simmons DN, Bond MJ. Piriformis syndrome: Correlation of muscle morphology with symptoms and signs. Arch Phys Med Rehabil 2004;85(12):2036–2039
Dunn DM. Anteversion of the neck of the femur; a method of measurement. J Bone Joint Surg Br 1952;34-B(2):181–186
Eland DC, Singleton TN, Conaster RR, et al. The "iliacus test": new information for the evaluation of hip extension dysfunction. J Am Osteopath Assoc 2002;102(3):130–142
Fishman LM, Schaefer MP. The piriformis syndrome is underdiagnosed. Muscle Nerve 2003;28(5):646–649
Fitzgerald RH Jr. Acetabular labrum tears. Diagnosis and treatment. Clin Orthop Relat Res 1995; (311):60–68

Gabbe BJ, Bennell KL. Reliability of common lower extremity musculoskeletal screening tests. Phys Ther Sport 2004;5:90–97
Gajdosik RL, Sandler MM, Marr HL. Influence of knee positions and gender on the Ober test for length of the iliotibial band. Clin Biomech (Bristol, Avon) 2003;18(1):77–79
Gautam VK. Anand S. A new test for estimating iliotibial band contracture. Bone Joint Surg [Br] 1998;80(3):474–475
Hanada E, Kirby RL, Mitchell M, Swuste JM. Measuring leg-length discrepancy by the "iliac crest palpation and book correction" method: reliability and validity. Arch Phys Med Rehabil 2001;82(7):938–942
Harvey D. Assessment of the flexibility of elite athletes using the modified Thomas test. Br J Sports Med 1998;32(1):68–70
Klaue K, Durnin CW, Ganz R. The acetabular rim syndrome. A clinical presentation of dysplasia of the hip. J Bone Joint Surg Br 1991;73(3):423–429
Kubiak-Langer M, Tannast M, Murphy SB, Siebenrock KA, Langlotz F. Range of motion in anterior femoroacetabular impingement. Clin Orthop Relat Res 2007;458(458):117–124
Levin U, Nilsson-Wikmar L, Stenström CH, Lundeberg T. Reproducibility of manual pressure force on provocation of the sacroiliac joint. Physiother Res Int 1998;3(1):1–14
Margo K, Drezner J, Motzkin D. Evaluation and management of hip pain: an algorithmic approach. J Fam Pract 2003;52(8):607–617
Marks MC, Alexander J, Sutherland DH, Chambers HG. Clinical utility of the Duncan-Ely test for rectus femoris dysfunction during the swing phase of gait. Dev Med Child Neurol 2003;45(11):763–768
Martin RL, Enseki KR, Draovitch P, Trapuzzano T, Philippon MJ. Acetabular labral tears of the hip: examination and diagnostic challenges. J Orthop Sports Phys Ther 2006;36(7):503–515
Ober FR. The role of the iliotibial band and fascia lata as a factor in the causation of low-back disabilities and sciatica. J Bone Joint Surg Am 1936;18:105–110
Reynolds D, Lucas J, Klaue K. Retroversion of the acetabulum. A cause of hip pain. J Bone Joint Surg Br 1999;81(2):281–288
Ross MD, Nordeen MH, Barido M. Test-retest reliability of Patrick's hip range of motion test in healthy college-aged men. J Strength Cond Res 2003;17(1):156–161
Ruwe PA, Gage JR, Ozonoff MB, DeLuca PA. Clinical determination of femoral anteversion. A comparison with established techniques. J Bone Joint Surg Am 1992;74(6):820–830
Ryder CT, Crane L. Measuring femoral anteversion; the problem and a method. J Bone Joint Surg Am 1953;35-A(2):321–328
Scopp JM, Moorman CT III. The assessment of athletic hip injury. Clin Sports Med 2001;20(4):647–659
Stewart JD. The piriformis syndrome is overdiagnosed. Muscle Nerve 2003;28(5):644–646
Stone M, Ellis D. How I examine the hip. Curr Orthop 2000;14:262–266
Tönnis D, Heinecke A. Acetabular and femoral anteversion: relationship with osteoarthritis of the hip. J Bone Joint Surg Am 1999;81(12):1747–1770
Trendelenburg F. Trendelenburg's test: 1895. Clin Orthop Relat Res 1998; (355):3–7
van der Wurff P, Hagmeijer RH, Meyne W. Clinical tests of the sacroiliac joint. A systematic methodological review. Part 1: Reliability. Man Ther 2000;5(1):30–36
van der Wurff P, Meyne W, Hagmeijer RH. Clinical tests of the sacroiliac joint. Man Ther 2000;5(2):89–96
Vasudevan PN, Vaidyalingam KV, Nair PB. Can Trendelenburg's sign be positive if the hip is normal? J Bone Joint Surg Br 1997;79(3):462–466

Knee

Anderson AF, Lipscomb AB. Clinical diagnosis of meniscal tears. Description of a new manipulative test. Am J Sports Med 1986;14(4):291–293

Anderson AF, Rennirt GW, Standeffer WC Jr. Clinical analysis of the pivot shift tests: description of the pivot drawer test. Am J Knee Surg 2000;13(1):19–23, discussion 23–24

Apley AG. The diagnosis of meniscus injuries; some new clinical methods. J Bone Joint Surg Am 1947;29(1):78–84

Bahk MS, Cosgarea AJ. Physical examination and imaging of the lateral collateral ligament and posterolateral corner of the knee. Sports Med Arthrosc Rev 2006;14(1):12–19

Biedert RM, Warnke K. Correlation between the Q angle and the patella position: a clinical and axial computed tomography evaluation. Arch Orthop Trauma Surg 2001;121(6):346–349

Bollen P. How I examine the knee. Curr Orthop 2000;14:189–192

Caylor D, Fites R, Worrell TW. The relationship between quadriceps angle and anterior knee pain syndrome. J Orthop Sports Phys Ther 1993;17(1):11–16

Cooperman JM, Riddle DL, Rothstein JM. Reliability and validity of judgments of the integrity of the anterior cruciate ligament of the knee using the Lachman's test. Phys Ther 1990;70:225–233

Dimon JH III. Apprehension test for subluxation of the patella. Clin Orthop Relat Res 1974;(103):39

Dupont JY, Bellier G. The jerk-test in external rotation in rupture of the anterior cruciate ligament. Description and significance. [Article in French.] Rev Chir Orthop Repar Appar Mot 1988;74(5):413–423

Eren OT. The accuracy of joint line tenderness by physical examination in the diagnosis of meniscal tears. Arthroscopy 2003;19(8):850–854

Evans PJ, Bell GD, Frank C. Prospective evaluation of the McMurray test. Am J Sports Med 1993;21(4):604–608

Fanelli GCF, Orcutt DR, Edson CJ. The multiple-ligament injured knee: evaluation, treatment, and results. Arthroscopy 2005;21(4):471–486

Fowler PJ, Lubliner JA. The predictive value of five clinical signs in the evaluation of meniscal pathology. Arthroscopy 1989;5(3):184–186

Fredericson M, Yoon K. Physical examination and patellofemoral pain syndrome. Am J Phys Med Rehabil 2006;85(3):234–243

Fulkerson JP. Diagnosis and treatment of patients with patellofemoral pain. Am J Sports Med 2002;30(3):447–456

Galway RD, Beaupre A, Macintosh DL. Pivot shift. J Bone Joint Surg Br 1972;54:763

Gose JC, Schweizer P. Iliotibial band tightness. J Orthop Sports Phys Ther 1989;10(10):399–407

Greene CC, Edwards TB, Wade MR, Carson EW. Reliability of the quadriceps angle measurement. Am J Knee Surg 2001;14(2):97–103

Grood ES, Noyes FR. Diagnosis of knee ligament injuries: Biomechanical precepts. In: Feagin JA, ed. The Crucial Ligaments. Diagnosis and Treatment of Ligamentous Injuries About the Knee. New York: Churchill Livingstone; 1988:245–260

Harrison BK, Abell BE, Gibson TW. The Thessaly test for detection of meniscal tears: validation of a new physical examination technique for primary care medicine. Clin J Sport Med 2009;19(1):9–12

Herrington L, Nester C. Q-angle undervalued? The relationship between Q-angle and medio-lateral position of the patella. Clin Biomech (Bristol, Avon) 2004;19(10):1070–1073

Hughston JC. The posterior cruciate ligament in knee joint stability. J Bone Joint Surg Am 1969;51:1045–1046
Hughston JC. The absent posterior drawer test in some acute posterior cruciate ligament tears of the knee. Am J Sports Med 1988;16(1):39–43
Hughston JC. Extensor mechanism examination. In: Fox JM, Del Pizzo W, eds. The Patellofemoral Joint. New York: McGraw Hill; 1993:63–74
Hughston JC, Andrews JR, Cross MJ, Moschi A. Classification of knee ligament instabilities. Part I: The medial compartment and cruciate ligaments. Part II: The lateral compartment. J Bone Joint Surg Am 1976;58:159–179
Hughston JC, Norwood LA Jr. The posterolateral drawer test and external rotational recurvatum test for posterolateral rotatory instability of the knee. Clin Orthop Relat Res 1980; (147):82–87
Hvid I, Andersen LI. The quadriceps angle and its relation to femoral torsion. Acta Orthop Scand 1982;53(4):577–579
Jakob RP, Hassler H, Stäubli HU. Observations on rotary instability of the lateral compartment of the knee. Acta Orthop Scand Suppl 1981;191:1–32
Jakob RP, Stäubli HU, Deland JT. Grading the pivot shift. Objective tests with implications for treatment. J Bone Joint Surg Br 1987;69:294–299
Jerosch J, Riemer S. How good are clinical investigative procedures for diagnosing meniscus lesions? [Article in German.] Sportverletz Sportschaden 2004;18(2):59–67
Johnson MW. Acute knee effusions: a systematic approach to diagnosis. Am Fam Physician 2000;61(8):2391–2400
Jonsson T, Althoff B, Peterson L, Renström P. Clinical diagnosis of ruptures of the anterior cruciate ligament: a comparative study of the Lachman test and the anterior drawer sign. Am J Sports Med 1982;10(2):100–102
Kannus P, Natri A, Paakkala T, Järvinen M. An outcome study of chronic patellofemoral pain syndrome. Seven-year follow-up of patients in a randomized, controlled trial. J Bone Joint Surg Am 1999;81(3):355–363
Kaplan EB. The iliotibial tract; clinical and morphological significance. J Bone Joint Surg Am 1958;40:817–832
Karachalios T, Hantes M, Zibis AH, Zachos V, Karantanas AH, Malizos KN. Diagnostic accuracy of a new clinical test (the Thessaly test) for early detection of meniscal tears. J Bone Joint Surg Am 2005;87(5):955–962
Kim SJ, Kim HK. Reliability of the anterior drawer test, the pivot shift test, and the Lachman test. Clin Orthop Relat Res 1995; (317):237–242
Konan S, Rayan F, Haddad FS. Do physical diagnostic tests accurately detect meniscal tears? Knee Surg Sports Traumatol Arthrosc 2009;17(7):806–811
König DP, Rütt J, Kumm D, Breidenbach E. Diagnosis of anterior knee instability. Comparison between the Lachman test, the KT-1,000 arthrometer and the ultrasound Lachman test. [Article in German.] Unfallchirurg 1998;101(3):209–213
Kumar AJ, Bickerstaff D. Posterolateral instability of the knee. Curr Orthop 2000;14:337–341
LaPrade RF, Wentorf F. Diagnosis and treatment of posterolateral knee injuries. Clin Orthop Relat Res 2002; (402):110–121
Lin YC, Davey RC, Cochrane T. Tests for physical function of the elderly with knee and hip osteoarthritis. Scand J Med Sci Sports 2001;11(5):280–286
Livingston LA. The accuracy of Q angle values. Clin Biomech (Bristol, Avon) 2002;17(4):322–323, author reply 323–324
Logan M, Williams A, Lavelle J, Gedroyc W, Freeman M. The effect of posterior cruciate ligament deficiency on knee kinematics. Am J Sports Med 2004;32(8):1915–1922

Loomer RL. A test for knee posterolateral rotatory instability. Clin Orthop Relat Res 1991; (264):235–238

Losee RE. Concepts of the pivot shift. Clin Orthop Relat Res 1983; (172):45–51

Malanga GA, Andrus S, Nadler SF, McLean J. Physical examination of the knee: a review of the original test description and scientific validity of common orthopedic tests. Arch Phys Med Rehabil 2003;84(4):592–603

Martens MA, Mulier JC. Anterior subluxation of the lateral tibial plateau. A new clinical test and the morbidity of this type of knee instability. Arch Orthop Trauma Surg 1981;98(2):109–111

Martens M, Libbrecht P, Burssens A. Surgical treatment of the iliotibial band friction syndrome. Am J Sports Med 1989;17(5):651–654

McMurray TP. The semilunar cartilages. Br J Surg 1942;29:407–414

Miller MD, Bergfeld JA, Fowler PJ, Harner CD, Noyes FR. The posterior cruciate ligament injured knee: principles of evaluation and treatment. Instr Course Lect 1999;48:199–207

Noble HB, Hajek MR, Porter M. Diagnosis and treatment of iliotibial band tightness in runners. Phys Sportsmed 1982;10:67–74

Noyes FR, Grood ES, Torzilli PA. Current concepts review. The definition of terms for motion and position of the knee and injuries of the ligaments. J Bone Joint Surg Am 1989;71:465–472

Olerud C, Berg P. The variation of the Q angle with different positions of the foot. Clin Orthop Relat Res 1984; (191):162–165

Orndorff DG, Hart JA, Miller MD. Physical examination of the knee. Curr Sports Med Rep 2005;4(5):243–248

Owens TC. Posteromedial pivot shift of the knee: a new test for rupture of the posterior cruciate ligament. A demonstration in six patients and a study of anatomical specimens. J Bone Joint Surg Am 1994;76(4):532–539

Quarles JD, Hosey RG. Medial and lateral collateral injuries: prognosis and treatment. Prim Care 2004;31(4):957–975, ix

Sandler DA. Homans' sign and medical education. Lancet 1985;2(8464):1130–1131

Scholten RJ, Devillé WL, Opstelten W, Bijl D, van der Plas CG, Bouter LM. The accuracy of physical diagnostic tests for assessing meniscal lesions of the knee: a meta-analysis. J Fam Pract 2001;50(11):938–944

Shelbourne KD, Benedict F, McCarroll JR, Rettig AC. Dynamic posterior shift test. An adjuvant in evaluation of posterior tibial subluxation. Am J Sports Med 1989;17(2):275–277

Shino K, Mitsuoka T, Horibe S, Hamada M, Nakata K, Nakamura N. The gravity sag view: a simple radiographic technique to show posterior laxity of the knee. Arthroscopy 2000;16(6):670–672

Slocum DB, James SL, Larson RL, Singer KM. Clinical test for anterolateral rotary instability of the knee. Clin Orthop Relat Res 1976; (118):63–69

Slocum DB, Larson RL. Rotatory instability of the knee. Its pathogenesis and a clinical test to demonstrate its presence. J Bone Joint Surg Am 1968;50(2):211–225

Soucacos PN, Papadopoulou M, Georgoulis A. The "Noulis" behind the Lachman test. Arthroscopy 1998;14(1):75–76

Stäubli HU, Jakob RP. Posterior instability of the knee near extension. A clinical and stress radiographic analysis of acute injuries of the posterior cruciate ligament. J Bone Joint Surg Br 1990;72(2):225–230

Strobel MJ, Weiler A, Schulz MS, Russe K, Eichhorn HJ. Fixed posterior subluxation in posterior cruciate ligament-deficient knees: diagnosis and treatment of a new clinical sign. Am J Sports Med 2002;30(1):32–38
Tegner Y, Lysholm J. Rating systems in the evaluation of knee ligament injuries. Clin Orthop Relat Res 1985;(198):43–49
Winslow J, Yoder E. Patellofemoral pain in female ballet dancers: correlation with iliotibial band tightness and tibial external rotation. J Orthop Sports Phys Ther 1995;22(1):18–21

Foot and Ankle

Alexander IJ. The Foot. Examination and Diagnosis. London: Churchill Livingstone; 1990
Alonso A, Khoury L, Adams R. Clinical tests for ankle syndesmosis injury: reliability and prediction of return to function. J Orthop Sports Phys Ther 1998;27(4):276–284
Bahr R, Pena F, Shine J, et al. Mechanics of the anterior drawer and talar tilt tests. A cadaveric study of lateral ligament injuries of the ankle. Acta Orthop Scand 1997;68(5):435–441
Bailie DS, Kelikian AS. Tarsal tunnel syndrome: diagnosis, surgical technique, and functional outcome. Foot Ankle Int 1998;19(2):65–72
Beumer A, Swierstra BA, Mulder PGH. Clinical diagnosis of syndesmotic ankle instability: evaluation of stress tests behind the curtains. Acta Orthop Scand 2002;73(6):667–669
Beumer A, van Hemert WL, Swierstra BA, Jasper LE, Belkoff SM. A biomechanical evaluation of clinical stress tests for syndesmotic ankle instability. Foot Ankle Int 2003;24(4):358–363
Bruns W, Maffulli N. Lower limb injuries in children in sports. Clin Sports Med 2000;19(4):637–662
Cloke DJ, Greiss ME. The digital nerve stretch test: A sensitive indicator of Morton's neuroma and neuritis. Foot Ankle Surg 2006;12:201–203
Gaebler C, Kukla C, Breitenseher MJ, et al. Diagnosis of lateral ankle ligament injuries. Comparison between talar tilt, MRI and operative findings in 112 athletes. Acta Orthop Scand 1997;68(3):286–290
Hamilton WG, Geppert MJ, Thompson FM. Pain in the posterior aspect of the ankle in dancers. Differential diagnosis and operative treatment. J Bone Joint Surg Am 1996;78(10):1491–1500
Hedrick MR, McBryde AM. Posterior ankle impingement. Foot Ankle Int 1994;15(1):2–8
Hopkinson WJ, St Pierre P, Ryan JB, Wheeler JH. Syndesmosis sprains of the ankle. Foot Ankle 1990;10(6):325–330
Jahss MH. Disorders of the Foot and Ankle. Philadelphia: Saunders; 1991
Kinoshita M, Okuda R, Morikawa J, Jotoku T, Abe M. The dorsiflexion-eversion test for diagnosis of tarsal tunnel syndrome. J Bone Joint Surg Am 2001;83-A(12):1835–1839
Kleiger B. Anterior tibiotalar impingement syndromes in dancers. Foot Ankle 1982;3(2):69–73
Klenerman L. How I examine the foot. Curr Orthop 2001;15:152–155
Lin CF, Gross ML, Weinhold P. Ankle syndesmosis injuries: anatomy, biomechanics, mechanism of injury, and clinical guidelines for diagnosis and intervention. J Orthop Sports Phys Ther 2006;36(6):372–384
Liu SH, Jason WJ. Lateral ankle sprains and instability problems. Clin Sports Med 1994;13(4):793–809

Liu W, Maitland ME, Nigg BM. The effect of axial load on the in vivo anterior drawer test of the ankle joint complex. Foot Ankle Int 2000;21(5):420–426
Mann RA, Coughlin MJ. Surgery of the Foot and Ankle. 6th ed. Vol 1. St. Louis: Mosby; 1995
Marotta JJ, Micheli LJ. Os trigonum impingement in dancers. Am J Sports Med 1992;20(5):533–536
Neale D, Adams IM. Common Foot Disorders. Edinburgh: Churchill Livingstone; 1989
Ray RG, Christensen JC, Gusman DN. Critical evaluation of anterior drawer measurement methods in the ankle. Clin Orthop Relat Res 1997;(334):215–224
Shookster L, Falke GI, Ducic I, Maloney CT Jr, Dellon AL. Fibromyalgia and Tinel's sign in the foot. J Am Podiatr Med Assoc 2004;94(4):400–403
Sizer PS Jr, Phelps V, Dedrick G, James R, Matthijs O. Diagnosis and management of the painful ankle/foot. Part 2: examination, interpretation, and management. Pain Pract 2003;3(4):343–374
Stamatis ED, Karabalis C. Interdigital neuromas: current state of the art—surgical. Foot Ankle Clin 2004;9(2):287–296
Thompson TC. A test for rupture of the tendo achillis. Acta Orthop Scand 1962;32:461–465
Tohyama H, Yasuda K, Ohkoshi Y, Beynnon BD, Renstrom PA. Anterior drawer test for acute anterior talofibular ligament injuries of the ankle. How much load should be applied during the test? Am J Sports Med 2003;31(2):226–232
Tol JL, van Dijk CN. Etiology of the anterior ankle impingement syndrome: a descriptive anatomical study. Foot Ankle Int 2004;25(6):382–386
Tol JL, Verhagen RA, Krips R, et al. The anterior ankle impingement syndrome: diagnostic value of oblique radiographs. Foot Ankle Int 2004;25(2):63–68
van Dijk CN. Anterior and posterior ankle impingement. Foot Ankle Clin 2006;11(3):663–683
van Dijk CN, Lim LS, Poortman A, Strübbe EH, Marti RK. Degenerative joint disease in female ballet dancers. Am J Sports Med 1995;23(3):295–300
van Dijk CN, Mol BWJ, Lim LS, Marti RK, Bossuyt PMM. Diagnosis of ligament rupture of the ankle joint. Physical examination, arthrography, stress radiography and sonography compared in 160 patients after inversion trauma. Acta Orthop Scand 1996;67(6):566–570
Young CC, Niedfeldt MW, Morris GA, Eerkes KJ. Clinical examination of the foot and ankle. Prim Care 2005;32(1):105–132

Posture Deficiency

Fialka-Moser V, Uher EM, Lack W. Postural disorders in children and adolescents. [Article in German.] Wien Med Wochenschr 1994;144(24):577–592
Widhe T. Spine: posture, mobility and pain. A longitudinal study from childhood to adolescence. Eur Spine J 2001;10(2):118–123

Venous Thrombosis

Allen EV, Brown GE. Raynaud's disease affecting men. Ann Intern Med 1932;5:1384–1386
Cranley JJ, Canos AJ, Sull WJ. The diagnosis of deep venous thrombosis. Fallibility of clinical symptoms and signs. Arch Surg 1976;111(1):34–36

Kim J, Richards S, Kent PJ. Clinical examination of varicose veins—a validation study. Ann R Coll Surg Engl 2000;82(3):171–175
May R. The Lowenberg test for early diagnosis of thrombosis. [Article in German.] Med Klin (Munich) 1955;50(45):1899–1900
McConnell EA. Performing Allen's test. Nursing 1997;27(11):26
Partsch H, Blättler W. Compression and walking versus bed rest in the treatment of proximal deep venous thrombosis with low molecular weight heparin. J Vasc Surg 2000;32(5):861–869
Stone MB, Price DD, Anderson BS. Ultrasonographic investigation of the effect of reverse Trendelenburg on the cross-sectional area of the femoral vein. J Emerg Med 2006;30(2):211–213

Occlusive Arterial Disease and Neurovascular Compression Syndromes

Allen EV. Thromboangiitis obliterans: methods of diagnosis of chronic occlusive arterial lesions distal to the wrist with illustrative cases. Am J Med Sci 1929;178:237–244
Stober R. Thoracic outlet syndrome. [Article in German.] Schweiz Rundschau Med 1989;78(39):1063–1070

Index

B

C

D

I

M

N