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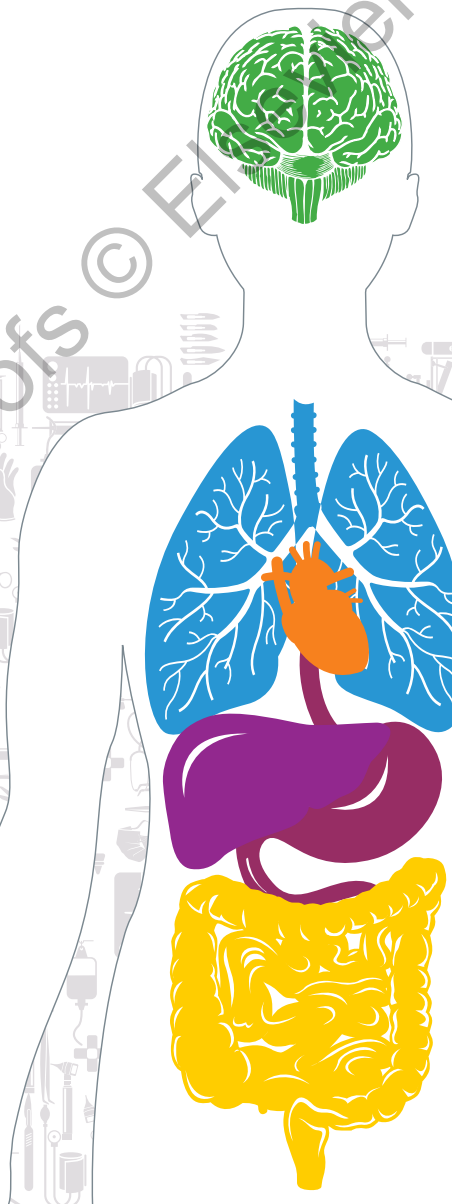
Talley & O'Connor's CLINICAL EXAMINATION 9e

A systematic guide to physical diagnosis

Nicholas J Talley
Simon O'Connor



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9th Edition

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

ISBN: 978-0-7295-4437-5

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National Library of Australia Cataloguing-in-Publication Data

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

Head of Content: Larissa Norrie
Content Project Manager: Shubham Dixit
Edited by Chris Wyard
Proofread by Melissa Faulkner
Permissions Editing and Photo Research: Priyadarshini Gnanasurian
Cover design by Georgette Hall
Typeset and Index by Aptara
Printed in ***

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

Foreword

Medical advances are increasing exponentially, and medical knowledge expands at an alarming rate, but the ability to assess a patient by listening to a medical history and examining the body systems in order to formulate a clinical diagnosis is a timeless skill which all doctors value.

Caring for the sick has been a key part of civilised society for many hundreds of years. It became professionalised in England with the endowment of Royal Charters to ensure that only those with an appropriate mix of skills and knowledge could enter the profession, and so membership of a Royal College had the added benefit of keeping the quacks and charlatans out. The Royal College of Physicians was set up on the 23rd of September 1518, in London, by King Henry VIII together with Thomas Linacre, one of his favoured physicians, to do just that. Since that time, the practice of medicine has developed and expanded into what we see today, more than 500 years later. Medical professional regulation was taken over by the UK General Medical Council in 1858, and a respected worldwide professional reputation has been building since then. It was my privilege to be the President of the Royal College of Physicians from 2014 until its quincentennial year in 2018, and so I understand the responsibility we have as physicians today.

The acquisition of the highest standard of clinical and communication skills coupled with sophisticated clinical reasoning is still at the heart of all clinical practice.

Our knowledge of pathology and disease processes continues to move very fast, but it is based on a foundation of expertise in the assessment of the patient that has been consolidated and enhanced over the years. New challenges appear, but the science underpinning medicine helps us to rise to these challenges. The recent global pandemic caused by coronavirus has demonstrated the strength of the partnership between the extraordinary advances in science and high-quality, good old-fashioned medicine.

Medicine continues to be a wonderful career with a wide variety of specialties, and something for everyone who enters the fold. Developing proficiency in the knowledge and skills required to practise high-quality clinical medicine binds us together as a professional group. Wherever I have travelled in the world, I meet doctors who instantly share a set of core values that have become the international language of medicine. As a profession, this is something to be proud of.

The education of doctors has changed over the years to keep pace with current pedagogy and the increasing knowledge base, but continues to emphasise the importance of basic clinical method. The conditions we see have changed from a predominance of infection and communicable disease to a skew towards long-term conditions and the impact of a modern lifestyle. This all needs to be understood by today's health professionals. We now recognise that a focus on prevention is the key to a healthy future, and all this is now included in medical curricula. Ways of learning now embrace the use of modern technology, and instant access to knowledge via a device, but the fundamental basis of medicine has not changed.

The great medical teacher, and arguably the father of modern medicine, William Osler, is quoted as saying:

The good physician treats the disease; the great physician treats the patient who has the disease.

As doctors we are in a privileged position, and benefit from the trust and confidence of our patients. Maintaining our core skills, and our ability to apply robust clinical reasoning allows us to maintain that position of trust, and to take our responsibilities seriously. We aspire to always treat the patient who has the disease, not just the disease.

Professor Dame Jane Dacre DBE, BSc, MD, FRCP, FHEA
Professor of Medical Education
Consultant Physician and Rheumatologist,
UCL Medical School, London

Contents

Preface

Listen to your patient, he (she) is telling you the diagnosis.

Sir William Osler (1849–1918)

Diagnosticians are great medical detectives who apply rigorous methodology to uncover the truth, solve a puzzle and commence the healing process.

Nick Talley & Simon O'Connor
Clinical Examination, 2017

Clinical medicine is exciting, and clinical skills continue to form the basis of modern clinical practice in order to make an accurate diagnosis. Thirty-five years ago, encouraged by the success of our first textbook on clinical skills *Examination Medicine,* which was aimed at postgraduate physician trainees sitting their barrier specialist internal medicine examinations, we set out to write *Clinical Examination*. The textbook was written over a year while we were senior registrars at the Royal North Shore Hospital, a major teaching hospital of the University of Sydney, Australia. Our goal was simple but ambitious: to write a new type of clinical skills textbook that would speak (and appeal) to medical students in all their clinical years from first patient exposure to final examinations (and beyond), and to teach a modern, systematic and comprehensive method of history taking and examination including an approach to rigorous clinical thinking and diagnosis.

A number of innovations have aided the success of this textbook and have been employed again here. The text is carefully organised and illustrated to maximise comprehension, including chapter summaries, essential lists and useful tables. Full-colour illustrations provide a visual guide for key signs including ethnic variations so important to recognise in clinical practice. We have included amusing comments and anecdotes with the view that optimal learning must be fun. We've added historical footnotes because these can be an *aide-mémoire*, and we believe students should know medicine is ever changing but we all stand on the shoulders of those who came before us. Videos illustrate how to examine in real time.

The book has extensively evolved over multiple editions and includes all specialty areas taught in undergraduate curriculums. We are pleased that generations of students have responded so positively to each edition, and medical schools around the world have adopted the textbook in their medical degree programs. We were very proud to be awarded First Prize in the Medicine category in the 2018 British Medical Association (BMA) Medical Book Awards, and hope this brand new revised edition will be similarly well received.

Every edition including the current one has undergone rigorous peer review by experts, and we have paid careful attention to the comments and suggestions. The book has received numerous reviews in major journals and readers have sent us many suggestions, and again we have taken the opportunity to try and address all reasonable recommendations with every revision. Before embarking on each revision, we also conduct a thorough literature search to identify new key references that inform the text and, where necessary, new references are added (and old ones replaced) for further reading. A strong emphasis on evidence-based medicine continues to permeate this edition. This is because today it takes only about 3 months for existing medical knowledge to double in size, compared with about 50 years mid last century. Despite rapidly accelerating knowledge, an understanding of the diagnostic value of important symptoms and signs is essential to excellent clinical practice. There remain huge research gaps and we hope readers of this book will aspire to fill many of them in the coming years.

The SARS-CoV-2 pandemic has shaken the globe and emphasised the importance of clinical skills and expertise in frontline care. We want to acknowledge all of the expert contributors to this new edition who willingly assisted us despite the pandemic, and the excellent peer reviewers and everyone who has provided us with feedback and encouragement. Do write to the publisher with any suggestions. We also thank all our colleagues and patients who continue to educate and inspire us on a daily basis.

Nicholas J. Talley, AC
Simon O'Connor
Newcastle and Canberra, 2021

Acknowledgements

This book provides an evidence-based account of clinical skills. We are very grateful for the reviews, comments and suggestions from the many outstanding colleagues over the years who have helped us to develop and refine this book. All chapters have again been peer reviewed, a hallmark of our books, and we have taken great care to revise the material based on the detailed reviews obtained. We take responsibility for any errors or omissions.

We would like to especially acknowledge **Professor Ian Symonds**, Dean of Medicine, University of Adelaide, and **Professor Kichu Nair**, Professor of Medicine and Associate Dean Continuing Medical Professional Development, University of Newcastle, for helping produce the videos for the OSCEs.

Dr Tom Wellings, Staff Specialist in Neurology, John Hunter Hospital, provided expert input into the neurology chapters for this edition. **Dr Philip McManis** provided invaluable input into neurology for earlier editions. **Professor Alan Cooper** prepared the original dermatology chapter.

Dr A Manoharan and **Dr J Isbister** provided the original blood film photographs and the accompanying text. **Associate Professor L Schreiber** provided the original section on soft-tissue rheumatology. We have revised and updated all of these sections again.

We thank **Professor Alex Ford** (Leeds Teaching Hospitals Trust, UK) and his team for their systematic review of the evidence supporting (or refuting) key clinical signs that has been retained.

Professor Brian Kelly, Dean of Medicine at the University of Newcastle, provided valuable comments on the psychiatry chapter. **Professor Phillip Boyce** provided the original psychiatry chapter.

Thank you to **Dr Malcolm Thomson** who provided a number of the X-rays and scans for this title. Others have been provided by the Medical Imaging Department at the Canberra Hospital X-ray Library.

We would like to thank **Associate Professor Lindsay Rowe**, Staff Specialist Radiologist at the John Hunter Hospital, for preparing the text and images within the gastrointestinal system section retained from the last edition.

We would like to acknowledge and thank **Glenn McCulloch** for the photographs he supplied for this title. We would also like to acknowledge Coleman **Productions** and the **Hunter Medical Research Institute** who provided with photographic assistance.

The authors thank **Dr Michael Potter, Dr. Yamini Yadav** and **Dr Stephen Brienesse** for their assistance. The authors and publishers also wish to thank **Jasmine Wark**, BBiomedSc, DipLang for her expert editorial support.

Elsevier Australia and the authors extend their appreciation to the following reviewers for their comments and insights on the entire manuscript:

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Clinical methods: an historical perspective

The best physician is the one who is able to differentiate the possible and the impossible.

Herophilus of Alexandria (335–280BC)

Since classical Greek times interrogation of the patient has been considered most important because disease was, and still is, viewed in terms of the discomfort it causes. However, the current emphasis on the use of history taking and physical examination for diagnosis developed only in the 19th century. Although the terms 'symptoms and signs' have been part of the medical vocabulary since the revival of classical medicine, until relatively recently they were used synonymously. During the 19th century, the distinction between *symptoms* (subjective complaints, which the clinician learns from the patient's account of his or her feelings) and *signs* (objective morbid changes detectable by the clinician) evolved. Until the 19th century, diagnosis was empirical and based on the classical Greek belief that all disease had a single cause: an imbalance of the four humours (yellow bile, black bile, blood and phlegm). Indeed the Royal College of Physicians, founded in London in 1518, believed that clinical experience without classical learning was useless, and physicians who were College members were fined if they ascribed to any other view. At the time of Hippocrates (460?–375BC), observation (inspection) and feeling (palpation) had a place in the examination of patients. The ancient Greeks, for example, noticed that patients with jaundice often had an enlarged liver that was firm and irregular. Shaking a patient and listening for a fluid splash was also recognised by the Greeks. Herophilus of Alexandria (335–280BC) described a method of taking the pulse in the 4th century BC. However, it was Galen of Pergamum (AD130–200) who established the pulse as one of the major physical signs, and it continued to have this important role up to the 18th century, with minute variations being recorded. These variations were erroneously considered to indicate changes in the body's harmony. William Harvey's (1578–1657) studies of the human circulation, published in 1628, had little effect on the general understanding of the value of the pulse as a sign. Sanctorius (1561–1636) was the first to time the pulse using a clock, while John Floyer (1649–1734) invented the pulse watch in 1707 and made regular observations of the pulse rate. Abnormalities in heart rate were described in diabetes mellitus in 1776 and in thyrotoxicosis in 1786. Fever was studied by Hippocrates and was originally regarded as an entity rather than a sign of disease. The thermoscope was devised by Sanctorius in 1625. In association with Gabriel Fahrenheit (1686–1736), Hermann Boerhaave (1668–1738) introduced the thermometer as a research instrument and this was produced commercially in the middle of the 18th century. In the 13th century Johannes Actuarius (d. 1283) used a graduated glass to examine the urine. In Harvey's time a specimen of urine was sometimes looked at (inspected) and even tasted, and was considered to reveal secrets about the body. Harvey recorded that sugar diabetes (mellitus) and dropsy (oedema) could be diagnosed in this way. The detection of protein in the urine, which Frederik Dekkers (1644–1720) first described in 1673, was ignored until Richard Bright (1789–1858) demonstrated its importance in renal disease. Although Celsus described and valued measurements such as weighing and measuring a patient in the 1st century AD, these methods became widely used only in the 20th century. A renaissance in clinical methods began with the concept of Battista Morgagni (1682–1771) that disease was not generalised but rather arose in organs, a conclusion published in 1761. Leopold Auenbrugger invented chest tapping (percussion) to detect disease in the same year. Van Swieten, his teacher, in fact

used percussion to detect ascites. The technique was forgotten for nearly half a century until Jean Corvisart (1755–1821) translated Auenbrugger's work in 1808.

The next big step occurred with René Laënnec (1781–1826), a student of Corvisart. He invented the stethoscope in 1816 (at first merely a roll of stiff paper) as an aid to diagnosing heart and lung disease by listening (auscultation). This revolutionised chest examination, partly because it made the chest accessible in patients too modest to allow a direct application of the examiner's ear to the chest wall, as well as allowing accurate clinicopathological correlations. William Stokes (1804–78) published the first treatise in English on the use of the stethoscope in 1825. Josef Skoda's (1805–81) investigations of the value of these clinical methods led to their widespread and enthusiastic adoption after he published his results in 1839. These advances helped lead to a change in the practice of medicine. Bedside teaching was first introduced in the Renaissance by Montanus (1498–1552) in Padua in 1543. In the 17th century, physicians based their opinion on a history provided by an apothecary (assistant) and rarely saw the patients themselves. Thomas Sydenham (1624–89) began to practise more modern bedside medicine, basing his treatment on experience and not theory, but it was not until a century later that the scientific method brought a systematic approach to clinical diagnosis.

This change began in the hospitals of Paris after the French Revolution, with recognition of the work of Morgagni, Corvisart, Laënnec and others. Influenced by the philosophy of the Enlightenment, which suggested that a rational approach to all problems was possible, the Paris Clinical School combined physical examination with autopsy as the basis of clinical medicine. The methods of this school were first applied abroad in Dublin, where Robert Graves (1796–1853) and William Stokes worked. Later, at Guy's Hospital in London, the famous trio of Richard Bright, Thomas Addison (1793–1860) and Thomas Hodgkin (1798–1866) made their important contributions. In 1869 Samuel Wilks (1824–1911) wrote on the nail changes in disease and the signs he described remain important. Carl Wunderlich's (1815–77) work changed the concept of temperature from a disease in itself to a symptom of disease. Spectacular advances in physiology, pathology, pharmacology and the discovery of microbiology in the latter half of the 19th century led to the development of the new 'clinical and laboratory medicine', which is the rapidly advancing medicine of the present day. The modern systematic approach to diagnosis, with which this book deals, is still, however, based on taking the history and examining the patient by looking (inspecting), feeling (palpating), tapping (percussing) and listening (auscultating).

Suggested reading

Bordage G. Where are the history and the physical? *Can Med Assoc J* 1995; 152:1595–1598.

McDonald C. Medical heuristics: the silent adjudicators of clinical practice. *Ann Intern Med* 1996; 124:56–62.

Reiser SJ. The clinical record in medicine. Part I: Learning from cases. *Ann Intern Med* 1991; 114:902–907.

The Hippocratic oath

I swear by Apollo the physician, and Aesculapius, and Hygieia, and Panacea, and all the gods and goddesses that, according to my ability and judgment, I will keep this Oath and this stipulation: To reckon him who taught me this Art equally dear to me as my parents, to share my substance with him and relieve his necessities if required; to look upon his offspring in the same footing as my own brother, and to teach them this Art, if they shall wish to learn it, without fee or stipulation, and that by precept, lecture, and every other mode of instruction, I will impart a knowledge of the Art to my own sons and those of my teachers, and to disciples bound by a stipulation and oath according to the law of medicine, but to none others. I will follow that system of regimen which, according to my ability and judgment, I consider for the benefit of my patients, and abstain from whatever is deleterious and mischievous. I will give no deadly medicine to any if asked, nor suggest any such counsel; and in like manner I will not give a woman a pessary to produce abortion. With purity and with holiness I will pass my life and practise my Art. I will not cut persons laboring under the stone, but will leave this to be done by men who are practitioners of this work. Into whatever houses I enter I will go into them for the benefit of the sick and will abstain from every voluntary act of mischief and corruption; and further from the seduction of females or males, of freemen and slaves. Whatever, in connection with my professional practice, or not in connection with it, I may see or hear in the lives of men which ought not to be spoken of abroad I will not divulge, as reckoning that all such should be kept secret. While I continue to keep this Oath unviolated may it be granted to me to enjoy life and the practice of the Art, respected by all men, in all times! But should I trespass and violate this Oath, may the reverse be my lot!

Hippocrates, born on the Island of Cos (c.460–357 BC) is agreed by everyone to be the father of medicine. He is said to have lived to the age of 109. Many of the statements in this ancient oath remain relevant today, while others, such as euthanasia and abortion, remain controversial. The seduction of slaves, however, is less of a problem.

CHAPTER 34

The neurological examination: the peripheral nervous system

System; any complexure or combination of many things acting together. SAMUEL JOHNSON, A Dictionary of the English Language *(1775)*

LIMBS AND TRUNK

History

The patient may present with symptoms that are purely or predominantly sensory or motor (see Questions box 34.1), or related to disorders of movement such as tremor. Sensory symptoms include pain, numbness and paraesthesias (tingling or pins and needles). It is important to find out whether there is involvement of more than one modality, something the patient may not have noticed. The distribution, time of onset and duration may give clues to the aetiology of the symptoms or at least as to where the sensory examination should be concentrated.

QUESTIONS TO ASK THE PATIENT WITH MUSCLE WEAKNESS

1. Have you felt weakness on both sides of the body? (Suggests spinal cord disease, myopathy or myasthenia gravis)
2. Is the weakness just on one side of the body or face? (Transient ischaemic attack or stroke)
3. Has the weakness affected just an arm or a leg or part of a limb? (Peripheral neuropathy or radiculopathy, stroke or multiple sclerosis)
4. Have you had trouble getting up from a chair, brushing your hair or lifting your head? (Proximal muscle weakness—myasthenia gravis, diabetic amyotrophy [involves lower limbs], polymyositis)
5. Have you had trouble swallowing or difficulty in speaking? (Myasthenia gravis, polymyositis)
6. Have you noticed double vision? (Myasthenia gravis, cranial nerve mononeuritis multiplex)
7. Are you taking any medications? (Steroid-induced proximal myopathy)
8. Have you had problems with your neck or back, or with severe arthritis? (Radiculopathy)
9. Have you had a cancer diagnosed at any stage? (Paraneoplastic, Eaton–Lambert syndrome)
10. Is there any problem like this in your family? (Familial myopathy, Charcot–Marie–Tooth disease)
11. Have you had HIV infection? (Various neurological lesions and drug reactions)
12. Have you ever had multiple sclerosis diagnosed?
13. Are you a diabetic? (Mononeuritis multiplex, amyotrophy)
14. Have you been injured? Where? (Back, pelvis, neck, etc.)
15. Is a compensation claim involved? (Possibility of secondary gain)

QUESTIONS BOX 34.1

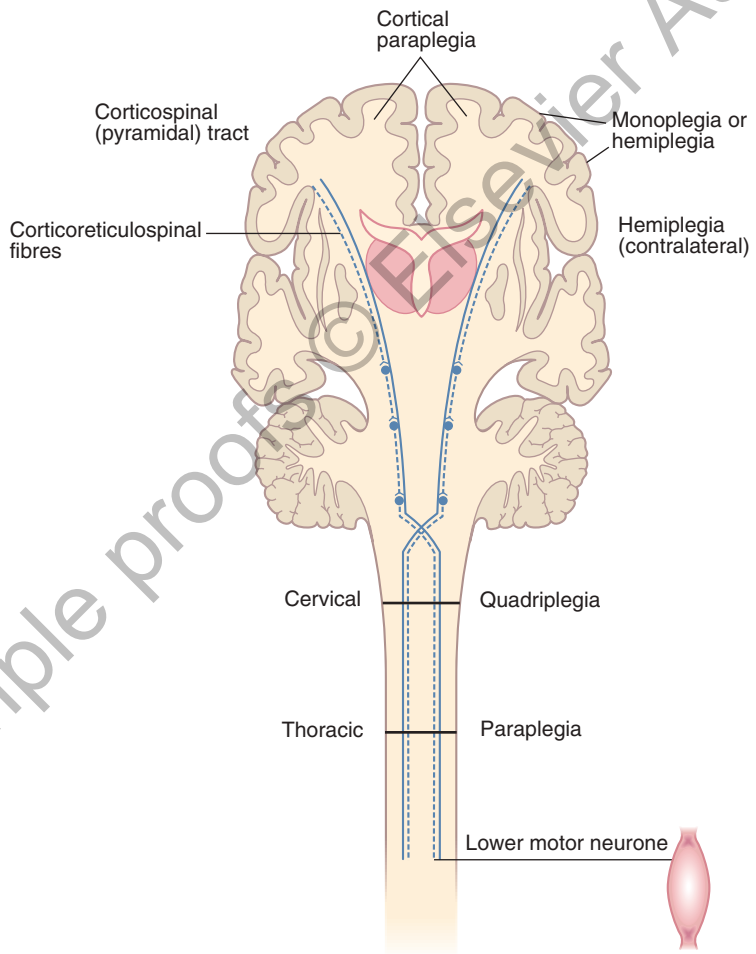
A family history of a similar problem may help provide the diagnosis in conditions such as muscular dystrophy. A previous injury may be responsible, for example, for a peripheral nerve problem but not remembered until asked about specifically.

Ask the patient about medications that can be a cause of neuropathy such as chemotherapeutic agents, metronidazole and amiodarone. Steroids can cause proximal muscle weakness. Alcohol excess is associated with neuropathy and occasionally strict vegans can become vitamin B_{12} deficient. Travel to countries where Lyme disease or leprosy are endemic may be relevant.

Examination anatomy

Muscle weakness has five major causes:

1. Pyramidal or upper motor neurone weakness, which is caused by a lesion in the brain proximal to the 'pyramids' in the brainstem. This is where the nerve fibres decussate or cross to the other side before travelling down the spinal cord (see Fig. 34.1).
2. Lower motor neurone weakness, which is caused by a nerve lesion within the spinal cord or peripheral nerve.



Motor neurone lesions

(Adapted from Lance JG, McLeod JW. *A physiological approach to clinical neurology*, 3rd edn. London: Butterworth, 1981.)

FIGURE 34.1

3. Abnormalities of the neuromuscular junction (myasthenia gravis).
4. Muscle disease.
5. Functional weakness (conversion reaction).

General examination approach

It is most important to have a set order of examination of the limbs for neurological signs so that nothing important is omitted. The following scheme is a standard approach.

1. Motor system
   General inspection
   - Posture
   - Muscle bulk
   - Abnormal movements
   - Fasciculations

   Tone
   Power
   Reflexes
   Coordination
2. Sensory system
   Pain and temperature
   Vibration and proprioception
   ±Light touch

General inspection

Remember to look for *asymmetry*.

1. Stand back and look at the patient for an *abnormal posture*—for example, one due to hemiplegia caused by a stroke. In this case, the upper limb is flexed and there is adduction and pronation of the arm, while the lower limb is extended.
2. Look for *muscle wasting*, which indicates a denervated muscle, a primary muscle disease or disuse atrophy. Compare one side with the other for wasting and try to work out which muscle groups are involved (proximal, distal or generalised, symmetrical or asymmetrical).
3. Inspect for *abnormal movements*, such as tremor of the wrist or arm.
4. Inspect the *skin*—for example, for evidence of neurofibromatosis, cutaneous angiomata in a segmental distribution (associated with syringomyelia) or herpes zoster. Look for scars from old injuries or surgical treatment. Note the presence of a urinary catheter.

Testing for arm drift: 'Shut your eyes and hold your arms out straight. Now turn your palms upwards'

FIGURE 34.2

Upper limbs

Motor system[a]

General

Shake hands with the patient and introduce yourself. A patient who cannot relax his or her hand grip has myotonia (an inability to relax the muscles after voluntary contraction). The most common cause of this is the muscle disease dystrophia myotonica (p 612). Once your hand has been extracted from the patient's, and after pausing briefly for the vitally important general inspection, ask the patient to undress so that the arms and shoulder girdles are completely exposed.

Sit the patient over the edge of the bed if this is possible. Next ask the patient to hold out both hands, palms upwards, with the arms extended and the eyes closed (see Fig. 34.2). Watch the arms for evidence of drifting (movement of one or both arms from the initial

[a] Aretaeus of Cappadocia reasoned, in AD 150, that the nerves cross (decussate) between the brain and the periphery and that injury to the right side of the head causes abnormalities of the left side of the body.

CAUSES (DIFFERENTIAL DIAGNOSIS) OF FASCICULATIONS

Motor neurone disease
Motor root compression
Peripheral neuropathy (e.g. diabetic)
Primary myopathy
Thyrotoxicosis

Note: Myokymia resembles coarse fasciculation of the same muscle group and is particularly common in the orbicularis oculi muscles, where it is usually benign. Focal myokymia, however, often represents brainstem disease (e.g. multiple sclerosis or glioma).
Fibrillation is seen only on the electromyogram.

LIST 34.1

neutral position). There are only three causes of arm drift:

1. *Upper motor neurone (pyramidal) weakness:* the drift of the affected limb(s) here is due to muscle weakness and tends to be in a downward direction. The drifting typically starts distally with the fingers and spreads proximally. There may be slow pronation of the wrist and flexion of the fingers and elbow.
2. *Cerebellar disease:* the drift here is usually upwards. It also includes slow pronation of the wrist and elbow.
3. *Loss of proprioception:* the drift here (pseudoathetosis) is really a searching movement and usually affects only the fingers. It is due to loss of joint position sense and can be in any direction.

Fasciculations

Ask the patient to relax the arms and rest them on his or her lap. Inspect the large muscle groups for *fasciculations* (see List 34.1). These are irregular contractions of small areas of muscle that have no rhythmical pattern. Fasciculation may be coarse or fine and is present at rest, but not during voluntary movement.[b] If present with weakness and wasting, fasciculation indicates degeneration of the lower motor neurone. It is usually benign if not associated with other signs of a motor lesion.

Tone

Tone is tested at both the wrists and the elbows. Rotation of the wrists with supination and pronation of the elbow joints (supporting the patient's elbow with one hand and holding the hand with the other) is performed passively (the examiner does the work), and the patient should be told to relax to allow you to move the joints freely.

When you raise and then drop the patient's arm, it will fall suddenly if tone is reduced. With experience it is possible to decide whether tone is normal or increased (hypertonic, as in an upper motor neurone or extrapyramidal lesion). Hypotonia is a difficult clinical sign to elicit and probably not helpful in the assessment of a lower motor neurone lesion. Most elderly people find it difficult not to try to help you and to relax their muscles completely. This leads to an increase in tone in all directions of movement, which increases with the speed of movement and with encouragement to relax. This is called *gegenhalten*[c] or *paratonia*. When it is severe it may be a result of frontal lobe or diffuse cerebrovascular disease. If the joints are moved unpredictably and at different rates or if the patient is distracted (e.g. by being asked to count backwards from 100) it may be reduced. Young people who are able to relax their muscle completely have little or no tone and hypotonia cannot be diagnosed in these people.

The cogwheel rigidity of Parkinson's disease is another abnormality of tone in the upper limbs. It is best assessed by having the patient move the other arm up and down as you move the hand and forearm, testing tone at the wrist and elbow. It is the result of rigidity and superimposed tremor.

Myotonia as described above is also an abnormality of tone that is worse after active movement. In these patients, tone is usually normal at rest but after sudden movements there may be a great increase in tone and the patient is unable to relax the muscle. Tapping over the body of a myotonic muscle causes a dimple of contraction, which only slowly disappears (*percussion myotonia*). This is best tested by tapping the thenar

[b] If no fasciculation is seen, tapping over the bulk of the brachioradialis and biceps muscles with the finger or with a tendon hammer and watching again has been recommended, but this is controversial. Most neurologists do not do this. The reason is that fasciculations are spontaneous. Any muscle movement from a local stimulus is not spontaneous. Even if they occur, they may have nothing to do with fasciculations.

[c] From the German meaning 'counterpressure' or 'standing your ground'.

eminence or by asking the patient to make a tight fist and then open the hand quickly. The opening of the fist is very slow when the muscles are myotonic.

Power

Muscle strength is assessed by gauging your ability to overcome the patient's full voluntary muscle resistance. Test this joint by joint, one side at a time, holding either side of the joint to isolate the movement in question. To decide whether the power is normal, the patient's age, sex and build should be taken into account. Power is graded based on the maximum observed (no matter how briefly), according to the following modified British Medical Research Council scheme (although this lacks sensitivity at the higher grades because work against gravity may make up only a small component of a muscle's function [e.g. the finger flexors]):

- **0** Complete paralysis (no movement).
- **1** Flicker of contraction possible.
- **2** Movement is possible when gravity is excluded.
- **3** Movement is possible against gravity but not if any further resistance is added.
- **4–** Slight movement against resistance.
- **4** Moderate movement against resistance.
- **4+** Submaximal movement against resistance.
- **5** Normal power.[d]

If power is reduced, decide whether this is symmetrical or asymmetrical, whether it involves only particular muscle groups or whether it is proximal, distal or general. Sometimes painful joint or muscle disease may interfere with the assessment (see Ch 23). Asymmetrical muscle weakness is most often the result of a peripheral nerve, brachial plexus or root lesion or an upper motor neurone lesion. As each movement is tested, the important muscles involved should be observed or palpated.

The testing of muscle power must always be somewhat subjective. It depends on the understanding and cooperation of the patient. Sometimes the patient may not fully cooperate. This can occur subconsciously if the weakness is functional (part of a conversion disorder) or consciously if there is secondary gain (e.g. a family dispute or compensation claim).[e] *The honest palm sign*[1] has been shown to help in determining whether the patient has made a complete effort during motor system examinations. The patient is asked to clench the fists with the fingernails tucked into the palms (as in Figs. 34.3a,b,c) while power is being assessed, e.g. testing for shoulder abduction or biceps power. The examiner urges the patient to try as hard as possible. The presence of nail marks in the palms makes the test positive and suggests maximum effort has been made. The positive LR of the

Testing power—both shoulders (honest palm sign)

FIGURE 34.3

[d] You should not be able to overcome a normal adult patient's power, at least in the legs.

[e] This used to be called malingering but the term has fallen into disfavour.

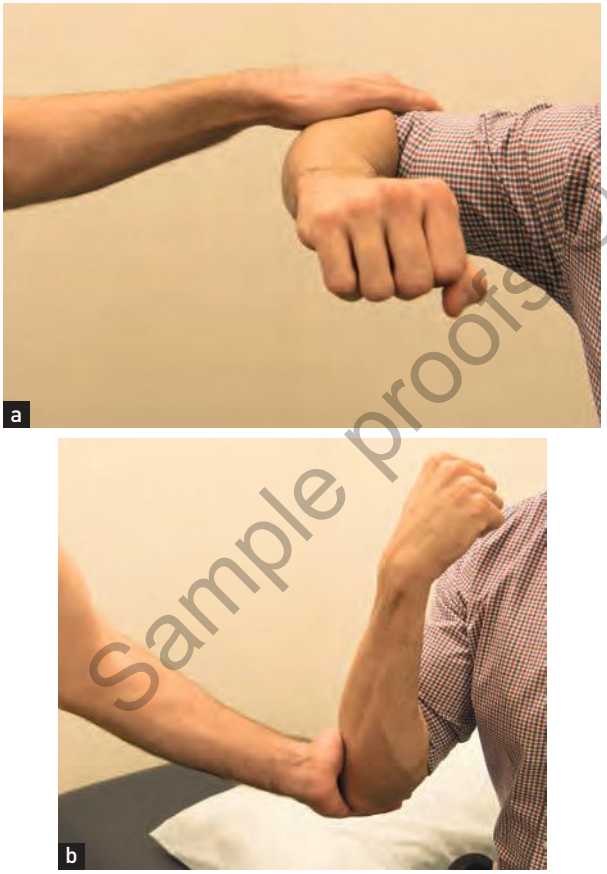
sign has been shown to be 3.4 and the negative LR to be 0.21. The inter-observer reliability was also very good (kappa 0.89). The test does not work if the patient's fingernails have been bitten or cut down to the quick.

Shoulder

- *Abduction*—mostly deltoid and supraspinatus—(C5, C6): the patient should abduct the arm with the elbow flexed and resist your attempt to push it down (see Fig. 34.4a).
- *Adduction*—mostly pectoralis major and latissimus dorsi—(C6–C8): the patient should adduct the arm with the elbow flexed and not allow you to push it to the side (see Fig. 34.4b).

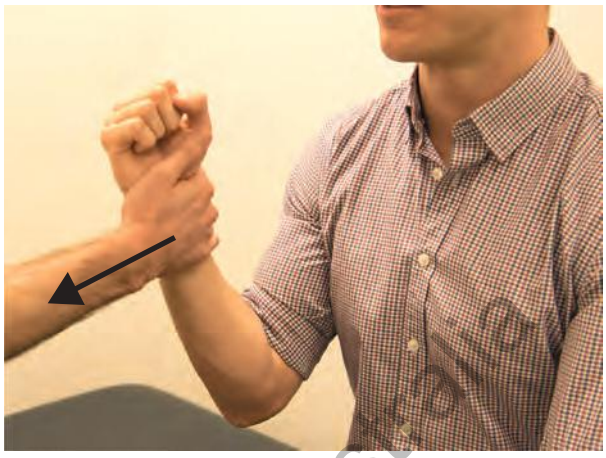
Elbow

- *Flexion*—biceps and brachialis—(C5, C6): the patient should bend the elbow and pull so as not to let you straighten it out (see Fig. 34.5).



Testing power—shoulder (test each arm separately)

FIGURE 34.4



Testing power—elbow flexion: 'Stop me straightening your elbow' (test each arm separately)

FIGURE 34.5

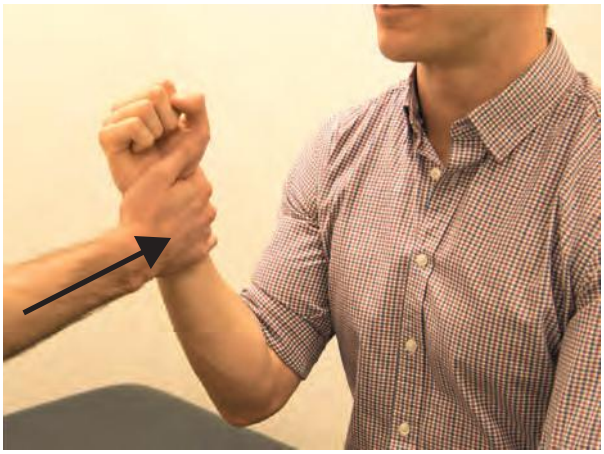
- *Extension*—triceps brachii—(C7, C8): the patient should partly bend the elbow and push so as not to let you bend it further (see Fig. 34.6).

Wrist

- *Flexion*—flexor carpi ulnaris and radialis—(C6, C7): the patient should bend the wrist and not allow you to straighten it. Hold the arm just above the wrist.
- *Extension*—extensor carpi group—(C7, C8): the patient should extend the wrist and not allow you to bend it (see Fig. 34.7).

Fingers

- *Extension*—extensor digitorum communis, extensor indicis and extensor digiti minimi—(C7, C8): the patient should straighten the fingers and not allow you to push them down (push with the side of your hand across the patient's metacarpophalangeal [MCP] joints).
- *Flexion*—flexor digitorum profundus and sublimis—(C7, C8): the patient squeezes two of your fingers (see Fig. 34.8).
- *Abduction*—dorsal interossei—(C8, T1): the patient should spread out the fingers and not allow you to push them together (see Fig. 34.9).
- *Adduction*—volar interossei—(C8, T1): the patient holds the fingers together and tries to prevent you from separating them further.



Testing power—elbow extension: 'Stop me bending your elbow' (test each arm separately)

FIGURE 34.6

Testing power—wrist extension: 'Stop me bending your wrist'

FIGURE 34.7

Alternatively the patient holds a piece of paper between the fingers, and you try to pull it out.

Reflexes

The sudden stretching of a muscle usually evokes brisk contraction of that muscle or muscle group. This reflex is usually mediated via a neural pathway synapsing in the spinal cord. It is subject to regulation via pathways from the brain. As the reflex is a response to stretching of a muscle, it is correctly called a muscle stretch reflex

Testing power—finger flexion: 'Squeeze my fingers hard' (don't offer more than two fingers)

FIGURE 34.8

rather than a tendon reflex.[f] The tendon merely transmits stretch to the muscle.

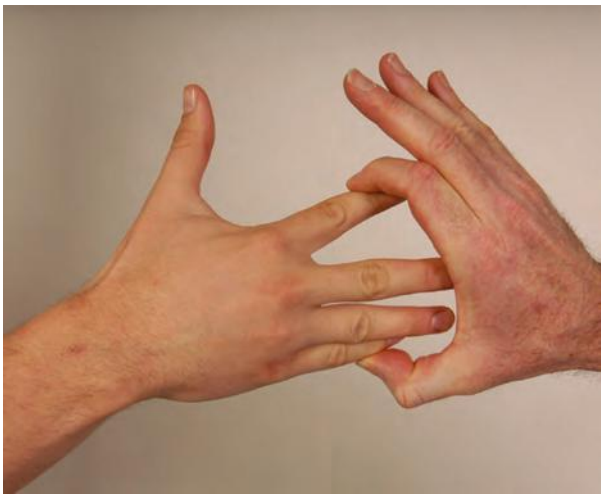
An intact jerk response confirms normal function in the large-diameter, myelinated, sensory fibres, in the spinal cord at the level of the reflex arc (e.g. L4,5 for the ankle jerk) and in the alpha motor neurones innervating the muscle.

Tendon hammers are available in a number of designs. Sir William Gowers[g] used the ulnar side of his hand or part of his stethoscope. In Australia and the United Kingdom, the Queen Square hammer[h] is in common use (see Fig. 34.10). The Taylor hammer is popular in the United States; it is shaped like a tomahawk and has a broad rubber edge for most

[f] Muscle spindles are specialised sensory receptors within the muscle that respond to both stretch and vibration. Action potentials form then move centrally via large-diameter, myelinated, fast-conducting fibres and trigger motor neurones in the spinal cord that cause muscle contraction. The size and speed of this reflex are affected by both autonomic sympathetic tone and descending (upper motor neurone) inhibition. Reflex activation is faster and stronger when sympathetic nervous system tone is high. Patients who are anxious, thyrotoxic or withdrawing from alcohol will have stronger, brisker responses. If descending inhibition from upper motor neurones is reduced as a result of a stroke, myelopathy or by applying a Jendrassik (reinforcement) manoeuvre, there will be stronger, brisker responses.

[g] Sir William Gowers (1845–1915), a professor of clinical medicine at University College Hospital, London, and neurologist to the National Hospital for Nervous Diseases, Queen Square, London. He was also an artist who illustrated his own books and had paintings exhibited at the Royal Academy.

[h] The Queen Square hammer was invented by Miss Wintle, staff nurse at Queen Square. She made hammers from brass wheels covered by a ring pessary and mounted on a bamboo handle; she sold these to medical students and resident medical officers.



Testing power—finger abduction: 'Stop me pushing your fingers together'

FIGURE 34.9

A Queen Square patellar hammer—'triceps jerk'

FIGURE 34.10

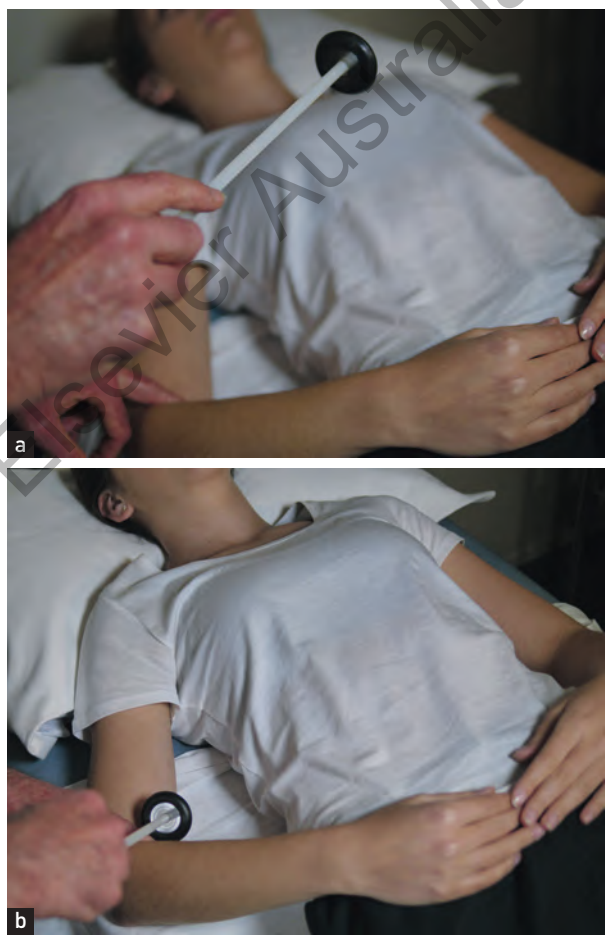
tendons and a more pointed side for the cutaneous reflexes.

Reflexes are graded from absent to greatly increased (see Table 34.1). Remember that patients who are very anxious or are thyrotoxic may have a general increase in the briskness of their reflexes.

Make sure the patient is resting comfortably with the elbows flexed and the hands lying pronated on the lap and not overlapping one another. To test the **biceps jerk** (C5, C6), place one forefinger on the biceps tendon and tap this with the tendon hammer (see Fig. 34.11).

Classification of muscle stretch reflexes	
0	Absent
+	Present but reduced
++	Normal
+++	Increased, possibly normal
++++	Greatly increased, often associated with clonus

TABLE 34.1



The biceps jerk examination

FIGURE 34.11

The hammer should be held near its end and the head allowed to fall with gravity onto the positioned forefinger. You will soon learn not to hit too hard or if the rubber on your tendon hammer is not soft enough. Normally, if the reflex arc is intact, there is a brisk contraction of the biceps muscle with flexion of the forearm at the elbow, followed by prompt relaxation. Practice will help

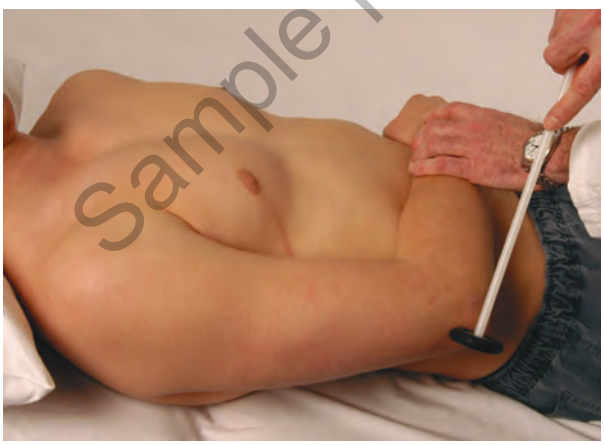
you decide whether the response is within the normal range. When a reflex is greatly exaggerated, it can be elicited away from the usual zone.

If a reflex appears to be absent, always test following a *reinforcement manoeuvre*. For example, ask the patient to clench the teeth tightly just before you let the hammer fall. Various mechanisms have been identified to explain reinforcement, but it works partly as a distraction, especially if the reflex is absent, because an anxious patient has contracted opposing muscle groups. Merely talking to the patient may provide enough distraction for the reflex to be elicited. Sometimes normal reflexes can be elicited only after reinforcement, but they should still be symmetrical.

An increased jerk occurs with an upper motor neurone lesion. The reflex itself may be large and brisk, but another, very specific sign of hyperreflexia is the finding of reflex spread (e.g. finger flexor activation on testing the biceps jerk). A decreased or absent reflex occurs with a breach in any part of the reflex motor arc—the muscle itself (e.g. myopathy), the motor nerve (e.g. neuropathy), the anterior spinal cord root (e.g. spondylosis), the anterior horn cell (e.g. poliomyelitis) or the sensory arc (sensory root or sensory nerve).

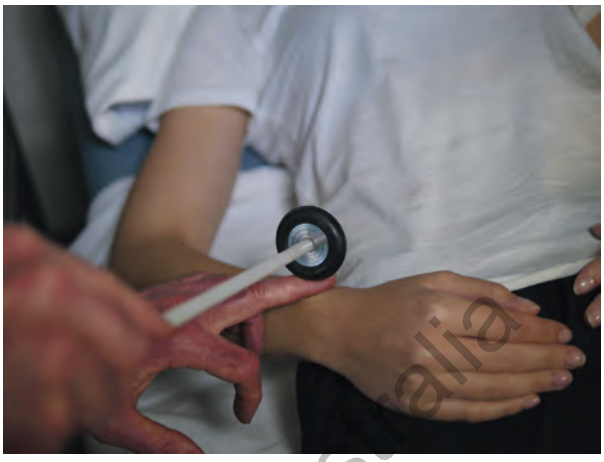
To test the **triceps jerk** (C7, C8), support the elbow with one hand and tap over the triceps tendon (see Fig. 34.12). Normally, triceps contraction results in forearm extension.

To test the **brachioradialis (supinator) jerk** (C5, C6), strike the lower end of the radius just above the wrist (see Fig. 34.13). To avoid hurting the patient by striking the radial nerve directly, place your own first two fingers over this spot and then strike your fingers, as with the biceps jerk. Normally, contraction of the brachioradialis causes flexion of the elbow.



The triceps jerk examination

FIGURE 34.12



The supinator jerk strike zone

FIGURE 34.13

If elbow extension and finger flexion are the only response when the patient's wrist is tapped, the response is said to be inverted, known as the *inverted brachioradialis (supinator) jerk*. The triceps contraction causes elbow extension instead of the usual elbow flexion. This is associated with an absent biceps jerk and an exaggerated triceps jerk. It indicates a spinal cord lesion at the C5 or C6 level due, for example, to compression (e.g. disc prolapse), trauma or syringomyelia. It occurs because a lower motor neurone lesion at C5 or C6 is combined with an upper motor neurone lesion affecting the reflexes below this level.

To test **finger jerks** (C8), the patient rests the hand palm upwards, with the fingers slightly flexed. Place your hand over the patient's and strike the hammer over your fingers (see Fig. 34.14). Normally, slight flexion of all the patient's fingers occurs. Neurologists may test Hoffman's jerks, holding the middle finger and flicking the distal phalanx. Flexion of the index finger and thumb constitutes a positive Hoffman's jerk, a specific sign of hyperreflexia.

Coordination

The cerebellum has multiple connections (afferent and efferent) to sensory pathways, brainstem nuclei, the thalamus and the cerebral cortex. Via these connections

The finger jerk examination

FIGURE 34.14

the cerebellum plays an integral role in coordinating voluntary movement. A standard series of simple tests is used to test coordination. Always demonstrate these movements for the patient's benefit.

Finger–nose test

Ask the patient to touch his or her nose with the index finger and then turn the finger around and touch your outstretched forefinger at nearly full extension of the shoulder and elbow (see Fig. 34.15). The test should be done at first slowly and then briskly, and repeated a number of times with the patient's eyes open and later closed. Slight resistance to the patient's movements by you pushing on his or her forearm during the test may unmask less-severe abnormalities.

Look for the following abnormalities: (1) intention (not *intentional*) tremor, which is tremor increasing as the target is approached (there is no tremor at rest), and (2) past-pointing, where the patient's finger overshoots the target towards the side of cerebellar abnormality. These abnormalities occur with cerebellar disease.

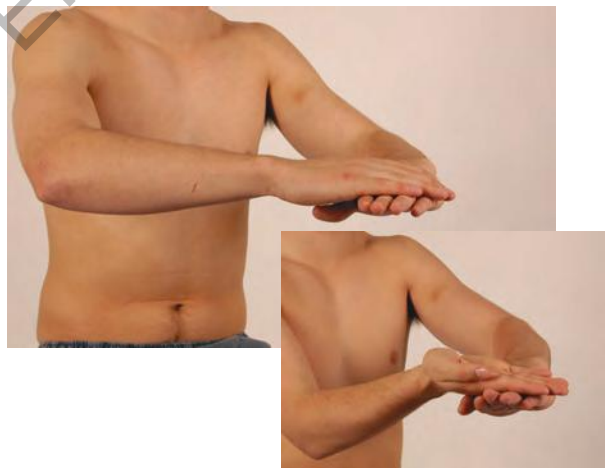
Rapidly alternating movements

Ask the patient to pronate and supinate his or her hand on the dorsum of the other hand as rapidly as possible (see Fig. 34.16). This movement is slow and clumsy in cerebellar disease and is called dysdiadochokinesis.[i]

[i] Actually, dysdiadochokinesis is the inability to perform alternating movements of both wrists with the arms and forefingers extended. *Diadochi* is a Greek word meaning 'succession'. The problem here is with successive movements. The Diadochi were the successors of Alexander the Great. They divided his empire.

Finger–nose test: 'Touch your nose with your forefinger and then reach out and touch my finger'

FIGURE 34.15



Testing for dysdiadochokinesis in the upper limbs: 'Turn your hand over, backwards and forwards on the other one, as quickly and smoothly as you can'

FIGURE 34.16

Rapidly alternating movements may also be affected in extrapyramidal disorders (e.g. Parkinson's disease) and in pyramidal disorders (e.g. internal capsule infarction).

Rebound

Ask the patient to lift the arms rapidly from the sides and then stop. Hypotonia due to cerebellar disease causes delay in stopping the arms. This method of demonstrating rebound is preferable to the more often used one where the patient flexes the arm at the elbow against the examiner's resistance. When the examiner suddenly lets go, violent flexion of the arm may occur and, unless prevented, the patient can strike himself or herself in the face. Therefore, only medical students trained in self-defence should use this method.[j]

Muscle weakness may also cause clumsiness, but motor testing should have revealed any impairment of this sort.

The sensory system

When examining the sensory system, less is more. The more time spent, the more that subjective and irrelevant subtle differences will be noticed, and the more confused you will become. Start distally and work proximally. It is seldom of value to map sensation over every square centimetre of skin.[2]

Spinothalamic pathway (pain and temperature)

Pain and temperature fibres enter the spinal cord and cross, a few segments higher, to the opposite spinothalamic tract (see Fig. 34.17). This tract ascends to the brainstem.

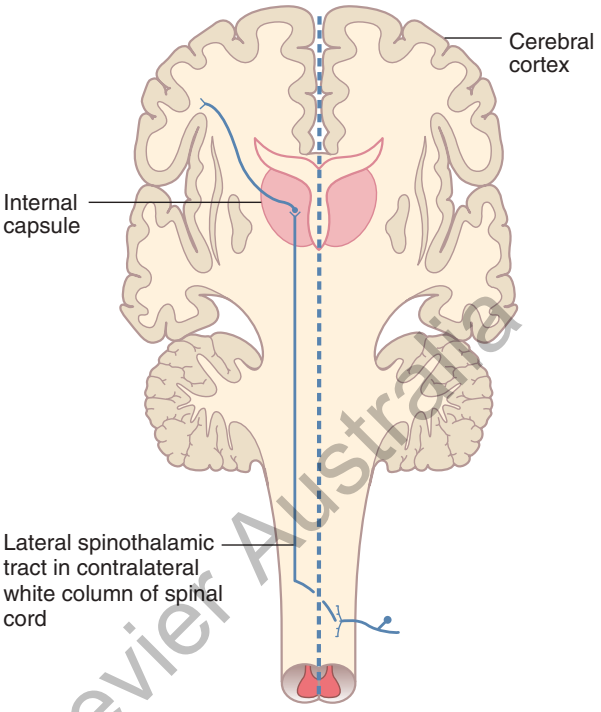
Pain (pinprick) testing

Using a new pin,[3] demonstrate to the patient that this induces a relatively sharp sensation by touching lightly a normal area, such as the anterior chest wall. Then ask the patient to say whether the pinprick feels sharp or dull. Begin distally and work proximally. Test in each dermatome—the area of skin supplied by a vertebral spinal segment (see Fig. 34.18). Also compare right with left in the same dermatome. Map out the extent of any area of dullness. Always do this by going from the area of dullness to the area of normal sensation (hence distal to proximal in most neuropathies).

Temperature testing

Cold sensation can be tested with a metal object, such as a tuning fork or metal end of the tendon hammer. Absence of ability to feel heat is almost always associated with inability to feel cold. Start distally and rapidly move proximally asking whether the temperature changes. Temperature sensation testing is often better tolerated by patients than pain sensation testing and neurologists consider it more helpful—though they may go on to test pain to confirm temperature findings.

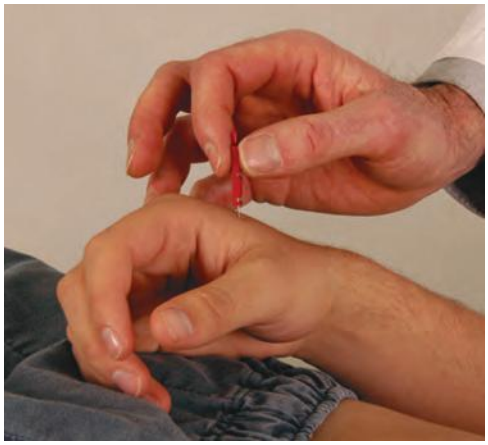
[j] Nick Talley has a black belt in Tae Kwon Do and Tang Soo Do.



Pain and temperature pathways

(Adapted from Snell RS, Westmoreland BF. *Clinical neuroanatomy for medical students*, 4th edition. Lippincott-Raven. 1997.)

FIGURE 34.17



Testing for pinprick (pain) sensation with a disposable neurology pin: 'Does this feel sharp or blunt?'

FIGURE 34.18

Posterior columns (vibration and proprioception[k])

These fibres enter and ascend ipsilaterally in the posterior columns of the spinal cord to the nucleus gracilis and nucleus cuneatus in the medulla, where they decussate (see Fig. 34.19).

Vibration testing

Use a 128 Hz (not a 256 Hz) tuning fork. Ask the patient to close the eyes, and place the vibrating tuning fork on one of the distal interphalangeal joints. Patients who have no experience with testing may let out a surprised exclamation. This confirms normal vibration sensation in that hand. The patient may describe a feeling of vibration, buzzing or may even gesture with their hands. Deaden the tuning fork with your hand; the patient should be able to say exactly when this occurs. Compare one side with the other. If vibration sense is reduced or absent, test over the ulnar head at the wrist, then the elbows (over the olecranon) and then the shoulders to determine the level of abnormality. Although the tuning fork is traditionally placed only over bony prominences, vibration sense is just as good over soft tissues.

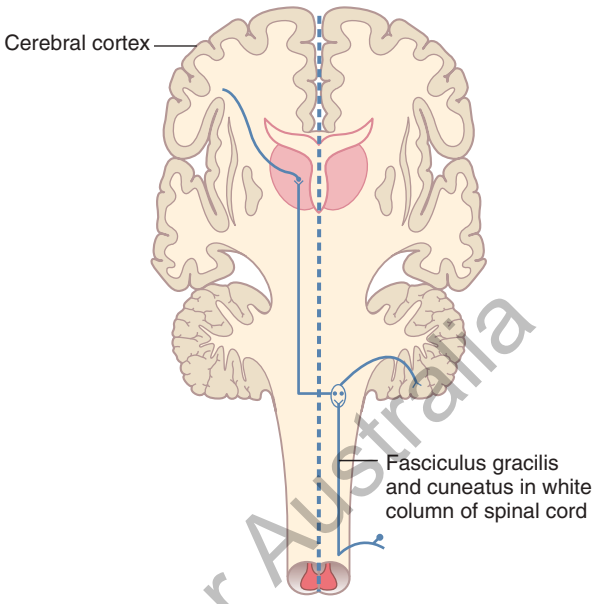
Proprioception testing

Use the distal interphalangeal joint of the patient's little finger. When the patient has his or her eyes open, grasp the distal phalanx from the sides (not the top and bottom) and move it up and down to demonstrate these positions. Start with big movements so the patient gets the idea, and progress to smaller movements. Then ask the patient to close the eyes while these manoeuvres are repeated randomly. Normally, movement through even a few degrees is detectable, and should be reported correctly. If there is an abnormality, proceed to test the wrists and elbows similarly. As a rule, sense of position is lost before sense of movement, and the little finger is affected before the thumb.

Light-touch testing

Some fibres travel in the posterior columns (i.e. ipsilaterally) and the rest cross the middle line to travel in the anterior spinothalamic tract (i.e. contralaterally). For this reason, light touch is of the least discriminating value. It is often omitted, except for bilateral touching with the fingers to assess for inattention or neglect (Ch 33). Irritation of light-touch receptors is probably responsible for paraesthesias—for example, following ischaemia of a limb.

Test light touch by touching the skin with a wisp of cottonwool. Ask the patient to shut the eyes and say 'yes' when the touch is felt. Do not stroke the skin because this moves hair fibres. Test each dermatome,[l] comparing left and right sides.



Vibration and joint position sense pathways

(Adapted from Snell RS, Westmoreland BF. *Clinical neuroanatomy for medical students*, 4th edition. Lippincott-Raven. 1997.)

FIGURE 34.19

Interpretation of sensory abnormalities

Try to fit the distribution of any sensory loss into a dermatome (due to a spinal cord or nerve root lesion), a single peripheral nerve territory, a peripheral neuropathy pattern (glove distribution, p 604) or a hemisensory loss (due to spinal cord or upper brainstem or thalamic lesion).

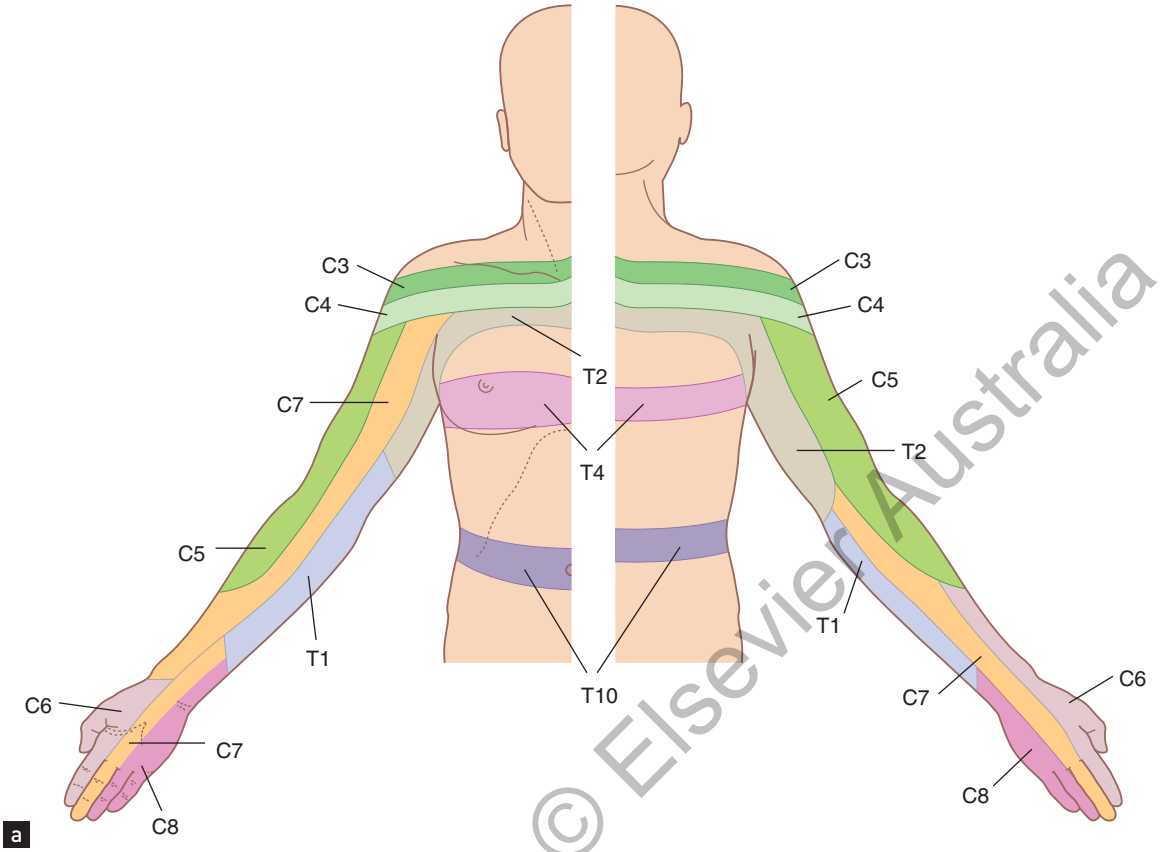
Sensory dermatomes of the upper limb (see Fig. 34.20) can be recognised by memorising the following rough guide:

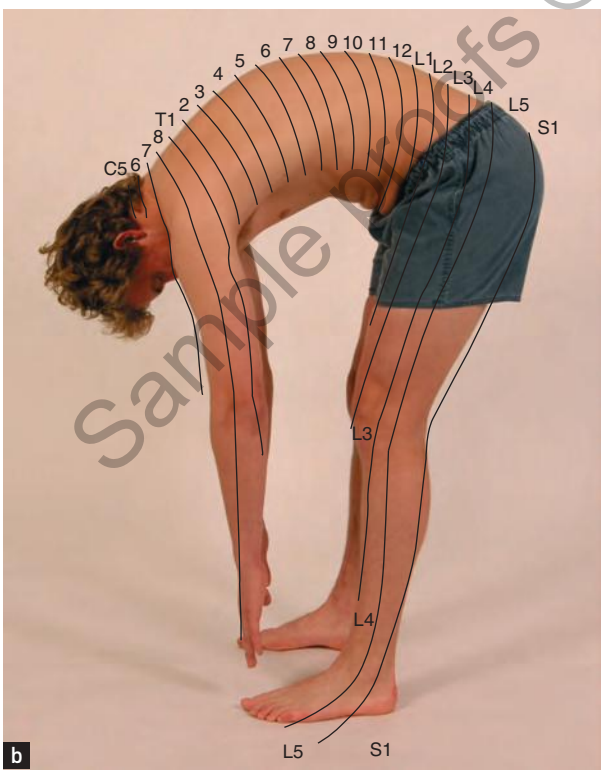
- C5 supplies the shoulder tip and outer part of the upper arm.

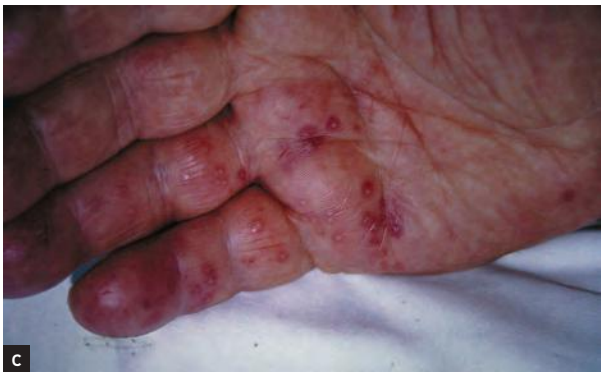
[k] In 1826 Sir Charles Bell recognised that there was a 'sixth sense', which was later called proprioception. Vibration sense had been recognised in the 16th century and tests for it were developed in the 19th century by Rinné and others. Rydel and Seiffer found that vibration sense and proprioception were carried in the posterior columns of the spinal cord.

[l] The human dermatomes (which he called pain spots) were first mapped by Sir Henry Head (1861–1940). He was most famous for his experimental cutting of his own radial nerve. This enabled him to chart the order of return of the sensory modalities.

Dermatomes of the upper limb and trunk.







(a) The dermatomes explained. (b) The distribution of the dermatomes makes more sense if we are thought of as quadrupedal. (c) Herpes zoster of the C8 dermatome showing its distribution

((c) Courtesy of Dr A Watson, Infectious Diseases Department, The Canberra Hospital.)

FIGURE 34.20

- C6 supplies the lateral aspect of the forearm and thumb.
- C7 supplies the middle finger.
- C8 supplies the little finger.
- T1 supplies the medial aspect of the upper arm and the elbow.

Examination of the peripheral nerves of the upper limb

A lesion of a peripheral nerve causes a characteristic motor and sensory loss.[4] Peripheral nerve lesions may have local causes, such as trauma or compression, or may be part of a mononeuritis multiplex, where more than one nerve is affected by systemic disease.

The radial nerve (C5–C8)

This is the *motor nerve* supplying the triceps and brachioradialis and the extensor muscles of the hand. The characteristic deformity that results from radial nerve injury is *wrist drop* (see Fig. 34.21). To demonstrate this, if it is not already obvious, get the patient to flex the elbow, pronate the forearm and extend the wrist and fingers. If a lesion occurs above the upper third of the upper arm, the triceps muscle is also affected. Therefore, test elbow extension, which will be absent if the lesion is high.

Test *sensation* using a pin over the area of the anatomical snuff box. Sensation here is lost with a radial nerve lesion before the bifurcation into posterior interosseous and superficial radial nerves at the elbow (see Fig. 34.22).

The median nerve (C6–T1)

This nerve contains the *motor* supply to all the muscles on the front of the forearm except the flexor carpi ulnaris and the ulnar half of the flexor digitorum profundus. It also supplies the following short muscles of the hand—**LOAF**:

L ateral two lumbricals
O pponens pollicis
A bductor pollicis brevis
F lexor pollicis brevis (in many people).

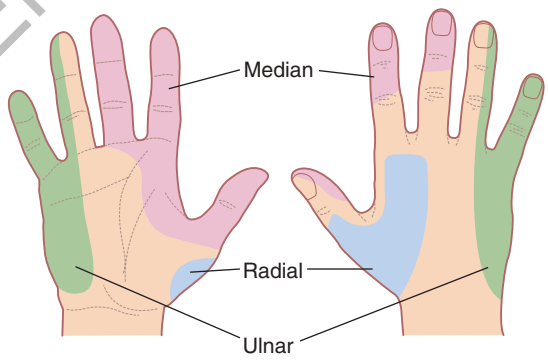
Lesion at the wrist (carpal tunnel)[5,6]

Use the pen-touching test to assess for weakness of the abductor pollicis brevis. Ask the patient to lay the hand flat, palm upwards on the table, and attempt to abduct the thumb vertically to touch your pen held above it (see Fig. 34.23). This may be impossible if there is a median nerve palsy at the wrist or above.

Typical appearance of a left radial nerve palsy with wrist drop

(From Jones Neil F and Machado Gustavo R. Functional hand reconstruction tendon transfers for radial, median, and ulnar nerve injuries: current surgical techniques. *Clin Plastic Surg* 2011; 38 (4):621–642. Copyright ©Elsevier.)

FIGURE 34.21



Average loss of pain sensation (pinprick) with lesions of the major nerves of the upper limbs

FIGURE 34.22

Remember, however, that most patients with the carpal tunnel syndrome have normal power and may indeed have symptoms but no signs at all. Feel at the wrist for a thickened median nerve (e.g. sarcoid, leprosy).

Lesion in the cubital fossa

Ochsner's clasping test[m] (for loss of flexor digitorum sublimis): ask the patient to clasp the hands firmly

[m] Albert Ochsner (1858–1925), an American surgeon of Swiss extraction, who claimed descent from Andreas Vesalius, the great anatomist.

together (see Fig. 34.24(a))—the index finger on the affected side fails to flex with a lesion in the cubital fossa or higher (see Fig. 34.24(b)).

For the *sensory* component of the median nerve, test pinprick sensation over the hand. The constant area of loss includes the palmar aspect of the thumb, the index finger, the middle finger and the lateral half of the ring finger (see Fig. 34.22). The palm is spared in median nerve lesions in the carpal tunnel.

The ulnar nerve (C8–T1)

This nerve contains the *motor* supply to all the small muscles of the hand (except the LOAF muscles), the flexor carpi ulnaris and the ulnar half of the flexor digitorum profundus. Look for wasting of the small muscles of the hand and for partial clawing of the little and ring fingers (a claw-like hand) (see Fig. 34.25). Clawing is hyperextension at the metacarpophalangeal joints and flexion of the interphalangeal joints. Note that clawing is more pronounced with an ulnar nerve lesion at the wrist, as a lesion at or above the elbow also causes loss of the flexor digitorum profundus, and therefore less flexion of the interphalangeal joints. This is the 'ulnar nerve paradox', in that a more distal lesion causes greater deformity. Also, feel at the elbow for a thickened ulna nerve.

Froment's[n] sign

Ask the patient to grasp a piece of paper between the thumb and lateral aspect of the forefinger with each hand. The affected thumb will flex because of loss of the adductor of the thumb.

Causes of a true claw hand are shown in List 34.2, while causes of wasting of the small muscles of the hand are shown in List 34.3; see also Fig. 34.26.

For the *sensory* component of the ulnar nerve, test for pinprick loss over the palmar and dorsal aspects of the little finger and the medial half of the ring finger (see Fig. 34.22).

The brachial plexus

Brachial plexus lesions vary from mild to complete; motor and/or sensory fibres may be involved. Nerve roots form trunks, which divide into cords and then form peripheral nerves (see Tables 34.2 and 34.3). The anatomy is shown in Fig. 34.27.

Patients with brachial plexus lesions may complain of pain or weakness in the shoulders or arms. Pain is often prominent, especially when there has been nerve root avulsion. A neurological cause is more likely if there is dull pain that is difficult to localise, if the pain is not related to limb movement and is worse at night and if there is no associated tenderness. The patient may be unable to get comfortable. An orthopaedic or traumatic cause is more likely if the pain is much worse with movement, or there are

Pen-touching test for loss of abductor pollicis brevis: 'Lift your thumb straight up to touch my pen'

FIGURE 34.23

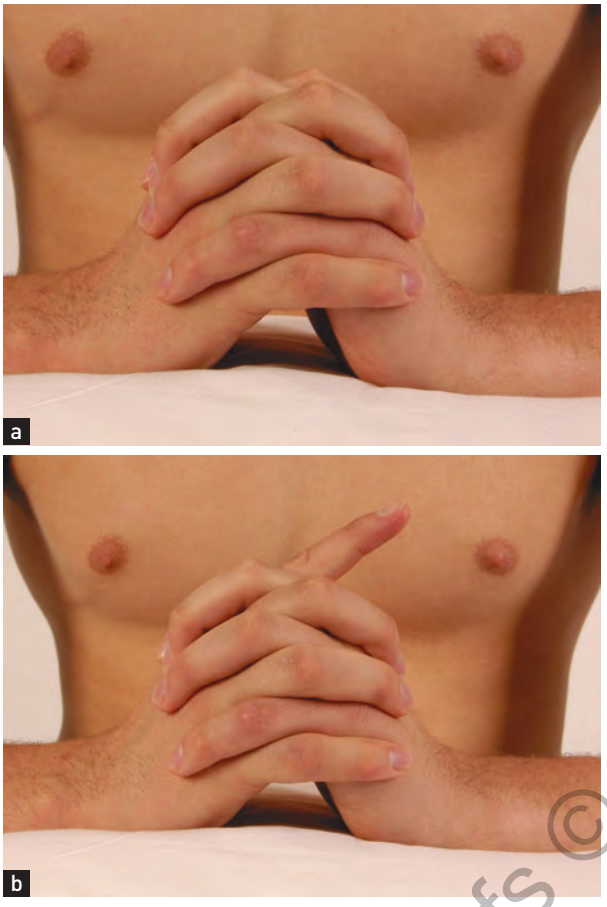
CAUSES (DIFFERENTIAL DIAGNOSIS) OF A TRUE CLAW HAND (ALL FINGERS CLAWED)

- Ulnar and median nerve lesion (ulnar nerve palsy alone causes a claw-like hand)
- Brachial plexus lesion (C8–T1)
- Other neurological disease (e.g. syringomyelia, polio)
- Ischaemic contracture (late and severe)
- Rheumatoid arthritis (advanced, untreated disease)

LIST 34.2

[n] Jules Froment (1878–1946), a professor of medicine in Lyons, France, described the sign in 1915.

Ochsner's clasping test.

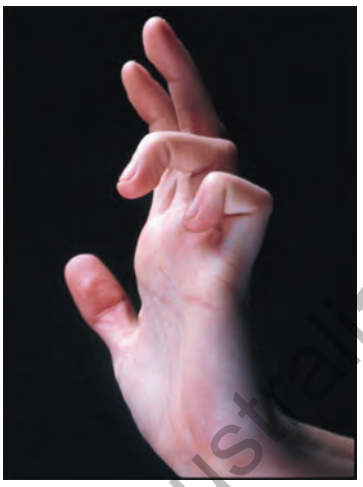


(a) Normal; (b) abnormal due to loss of function of the flexor digitorum (simulated demonstration)

FIGURE 34.24

signs of inflammation, joint deformity or local tenderness. Most plexus lesions are supraclavicular (i.e. proximal), especially when they occur after trauma. When infraclavicular (i.e. distal) lesions occur, they are usually less severe.

Examine the arms and shoulder girdle (see List 34.4). Remember that the dorsal scapular nerve (which supplies the rhomboid muscles) comes from the C5 nerve root proximal to the upper trunk, and so rhomboid function is usually spared in upper trunk lesions. Typical lesions of the brachial plexus are described in List 34.5. The cervical rib syndrome may cause a lower brachial plexus lesion (see List 34.6).



'Claw hand' due to a lesion of the ulnar nerve at the wrist

(From Sinnatamby C. *Last's anatomy: regional and applied*, 12th edn. Edinburgh: Elsevier; 2011, Figure 2.50.)

FIGURE 34.25

Table 34.4 suggests a scheme for distinguishing between plexus and nerve root lesions.

Ask the patient to stand facing away from you with the arms and hands stretched out to touch and push against the wall. Winging of the scapulae is seen typically in fascioscapular–humeral dystrophy (see Fig. 34.28).

Causes of brachial plexus lesions include:

- inflammation and infection, autoimmune disorders (more often upper plexus)
- radiotherapy (more often upper plexus)
- cancer (more often lower plexus)—cancer causes a brachial plexus lesion by local invasion; the lower trunk is usually affected first. These plexus lesions are usually painful and progress rapidly. Weakness and sensory loss are both present
- trauma: direct (motor vehicle crash, surgery including sternotomy, lacerations and gunshots), traction (birth injuries, motor vehicle crashes, sporting injuries such as rugby tackles—more often upper plexus), chronic compression (thoracic outlet, 'backpack palsy', fractures with bone displacement).

CAUSES (DIFFERENTIAL DIAGNOSIS) OF WASTING OF THE SMALL MUSCLES OF THE HAND

Spinal cord lesions

Syringomyelia

Cervical spondylosis with compression of the C8 segment

Tumour

Trauma

Anterior horn cell disease

Motor neurone disease, poliomyelitis

Spinal muscular atrophies (e.g. Kugelberg–Welander* disease)

Root lesion

C8 compression

Lower trunk brachial plexus lesion

Thoracic outlet syndromes

Trauma, radiation, infiltration, inflammation

Peripheral nerve lesions

Median and ulnar nerve lesions

Peripheral motor neuropathy

Myopathy

Dystrophia myotonica—forearms are more affected than the hands

Distal myopathy

Trophic disorders

Arthropathies (disuse)

Ischaemia, including vasculitis

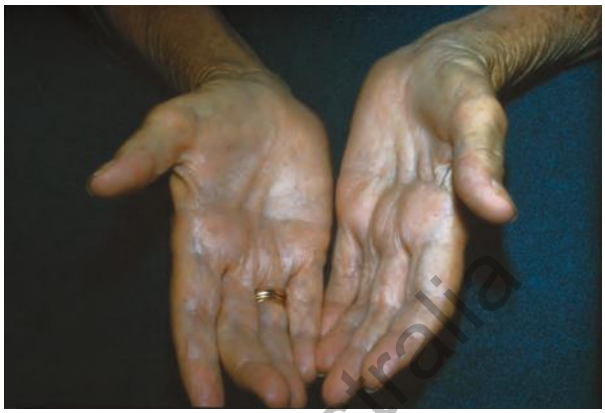
Shoulder hand syndrome

Note: Distinguishing an ulnar nerve lesion from a C8 root / lower trunk brachial plexus lesion depends on remembering that sensory loss with a C8 lesion extends proximal to the wrist, and the thenar muscles are involved with a C8 root or lower trunk brachial plexus lesion. Distinguishing a C8 root from a lower trunk brachial plexus lesion is difficult clinically, but the presence of a Horner syndrome or an axillary mass suggests the brachial plexus is affected.

*Eric Klas Kugelberg (1913–83), a professor of clinical neurophysiology at the Karolinska Institute in Stockholm, and Lisa Welander (1909–2001) described this in 1956. Lisa Welander was Sweden's first female professor of neurology.

LIST 34.3

Motor neurone disease.



Shows wasting of the small muscles of the hand

FIGURE 34.26

Nerve roots and brachial plexus trunks

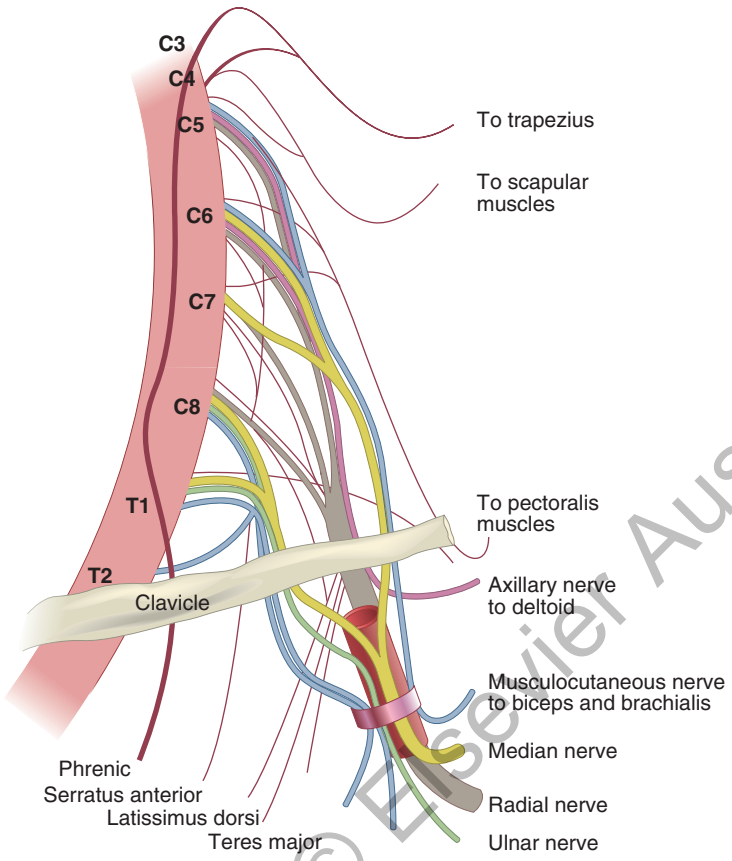
Nerve roots	Trunks	Muscles supplied
C5 and 6	Upper	Shoulder (especially biceps and deltoid)
C7	Middle	Triceps and some forearm muscles
C8 and T1	Lower	Hand and some forearm muscles

TABLE 34.2

Brachial plexus cords, nerves and their supplied muscles

Cords	Nerves formed	Muscles supplied
Lateral	Musculocutaneous, median	Biceps, pronator teres, flexor carpi radialis
Medial	Median and ulnar	Hand muscles
Posterior	Axillary and radial	Deltoid, triceps and forearm extensors

TABLE 34.3



The brachial plexus

(Adapted from Chusid JG. *Correlative neuroanatomy and functional neurology*, 19th edn. Los Altos: Lange Medical, 1985.)

FIGURE 34.27

SHOULDER GIRDLE EXAMINATION

Method

Abnormalities are likely to be due to a muscular dystrophy, single nerve or a root lesion. Inspect each muscle, palpate its bulk and test function as follows:

1. Trapezius (XI, C3, C4): ask the patient to elevate the shoulders against resistance and look for winging of the upper scapula.
2. Serratus anterior (C5–C7): ask the patient to push the hands against the wall and look for winging of the lower scapula.
3. Rhomboids (C4, C5): ask the patient to pull both shoulder blades together with the hands on the hips.
4. Supraspinatus (C5, C6): ask the patient to abduct the arms from the sides against resistance.
5. Infraspinatus (C5, C6): ask the patient to rotate the upper arms externally against resistance with the elbows flexed at the sides.
6. Teres major (C5–C7): ask the patient to rotate the upper arms internally against resistance.
7. Latissimus dorsi (C7, C8): ask the patient to pull the elbows into the sides against resistance.
8. Pectoralis major, clavicular head (C5–C8): ask the patient to lift the upper arms above the horizontal and push them forwards against resistance.
9. Pectoralis major, sternocostal part (C6–T1) and pectoralis minor (C7): ask the patient to adduct the upper arms against resistance.
10. Deltoid (C5, C6) (and axillary nerve): ask the patient to abduct the arms against resistance.

LIST 34.4

BRACHIAL PLEXUS LESIONS

Complete lesion (rare)

1. Lower motor neurone signs affect the whole arm
2. Sensory loss (whole limb)
3. Horner syndrome (an important clue)

Note: This is often painful.

Upper lesion (Erb Duchenne*) (C5, C6)

1. Loss of shoulder movement and elbow flexion—the hand is held in the waiter's tip position
2. Sensory loss over the lateral aspect of the arm and the forearm

Lower lesion (Klumpke†) (C8, T1)

1. True claw hand with paralysis of all the intrinsic muscles
2. Sensory loss along the ulnar side of the hand and the forearm
3. Horner syndrome

*Wilhelm Heinrich Erb (1840–1921), Germany's greatest neurologist.

†Auguste Déjérine-Klumpke (1859–1927), a French neurologist, described this lesion as a student. She was an American, but was educated in Switzerland. As a final-year student she married the great French neurologist Jules Déjérine.

LIST 34.5

CERVICAL RIB SYNDROME

Clinical features

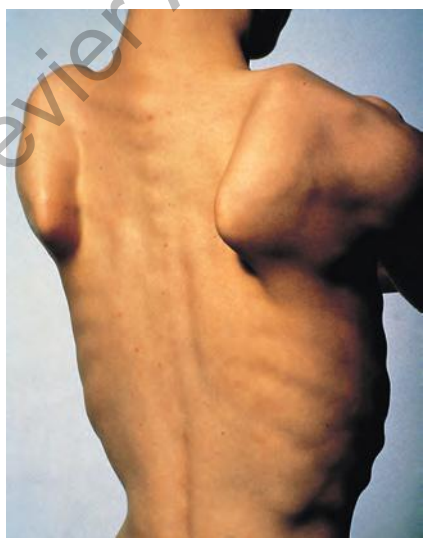
1. Weakness and wasting of the small muscles of the hand (claw hand)
2. C8 and T1 sensory loss
3. Unequal radial pulses and blood pressure
4. Subclavian bruits on arm manoeuvring (may be present in healthy people)
5. Palpable cervical rib in the neck (uncommon)

LIST 34.6

Distinguishing between brachial plexus lesions and nerve root compression

	Plexus	Root
Previous trauma	Some types	Occasionally
Insidious onset	Some types	Usually
Neck pain	No	Yes
Unilateral interscapular pain	No	Yes
Weakness	Often severe	Mild–moderate
Pattern of weakness	Usually shoulder and biceps or hand	Most commonly triceps (C7 lesions, the most commonly affected root)

TABLE 34.4



Winging of the scapulae, often a result of muscular dystrophy

(From Mir MA. *Atlas of clinical diagnosis*, 2nd edn. Edinburgh: Saunders, 2003.)

FIGURE 34.28

Lower limbs

Begin by testing *gait*, if this is possible (see pp 597–8).

Inspect the legs with the patient lying in bed with the legs and thighs entirely exposed (place a towel over the groin). Note whether there is a urinary catheter present, which may indicate that there is spinal cord compression or other spinal cord disease, particularly multiple sclerosis.

The motor system

Fasciculations and muscle wasting

Inspect for fasciculations. Look for muscle wasting. Feel the muscle bulk of the quadriceps and calves. Then run a hand along each shin, feeling for wasting of the anterior tibial muscles.

Tone

Test tone at the knees and ankles. Place one hand under a chosen knee and then abruptly pull the knee upwards, causing flexion. When the patient is relaxed this should occur without resistance. Then, supporting the thigh, flex and extend the knee at increasing velocity, feeling for resistance to muscle stretch (tone). Tone in the legs may also be tested by sitting the patient with legs hanging freely over the edge of the bed. Raise one of the patient's legs to the horizontal and then suddenly let go. The leg will oscillate up to half a dozen times in a healthy person who is completely relaxed. If hypotonia is present, as occurs in cerebellar disease, the oscillations will be wider and more prolonged. If increased tone or spasticity is present, the movements will be irregular and jerky.

Next test for *clonus* of the ankle and knee. This is a sustained rhythmical contraction of the muscles when put under sudden stretch. It is due to hypertonia from an upper motor neurone lesion. It represents an increase in reflex excitability (from increased alpha motor neurone activity).

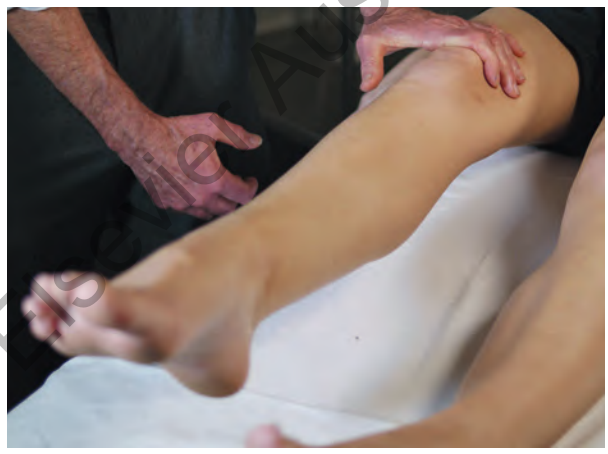
Sharply dorsiflex the foot with the knee bent and the thigh externally rotated. When ankle clonus is present, recurrent ankle plantar flexion movement occurs. This may persist for as long as you sustain dorsiflexion of the ankle. Test for patellar clonus by resting a hand on the lower part of the quadriceps with the knee extended and moving the patella down sharply. Sustained rhythmical contraction of the quadriceps occurs as long as the downward stretch is maintained.

Power

Test power next.

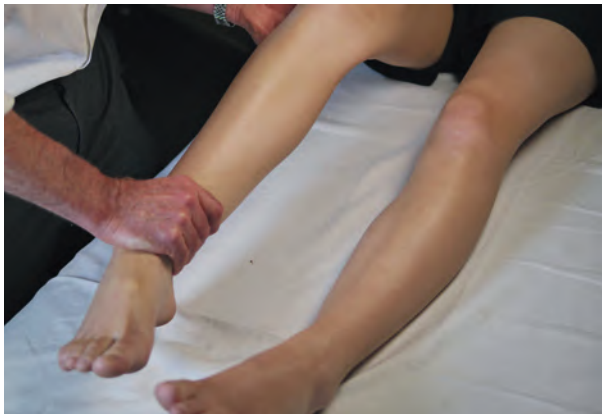
Hip

- *Flexion*—psoas and iliacus muscles—(L2, L3): ask the patient to lift up the straight leg and not let you push it down (having placed your hand above the knee; see Fig. 34.29).
- *Extension*—gluteus maximus—(L5, S1, S2): ask the patient to keep the leg down and not let you pull it up from underneath the calf or ankle (see Fig. 34.30).
- *Abduction*—gluteus medius and minimus, sartorius and tensor fasciae latae—(L4, L5, S1): ask the patient to abduct the leg and not let you push it in (see Fig. 34.31).
- *Adduction*—adductors longus, brevis and magnus—(L2, L3, L4): ask the patient to keep the leg adducted and not let you push it out (see Fig. 34.32).



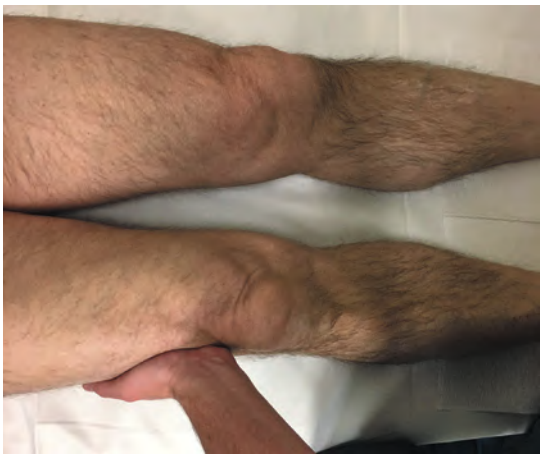
Testing power—hip flexion: 'Lift your leg up and don't let me push it down'

FIGURE 34.29



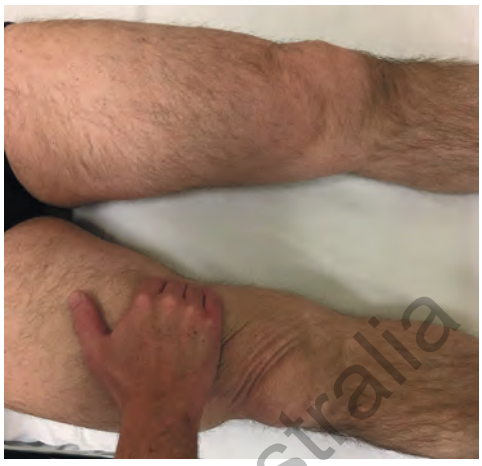
Testing power—hip extension: 'Push your heel down and don't let me pull it up'

FIGURE 34.30



Testing power—hip abduction: 'Don't let me push your hip in'

FIGURE 34.31



Testing power—hip adduction: 'Don't let me push your hip out'; pull hard

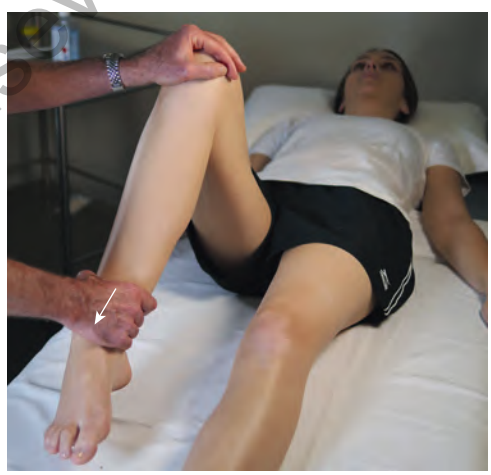
FIGURE 34.32

Knee

- *Flexion*—hamstrings (biceps femoris, semimembranosus, semitendinosus)—(L5, S1): ask the patient to bend the knee and not let you straighten it (see Fig. 34.33). If there is doubt about the real strength of knee flexion, it should be tested with the patient in the prone position. Here possible help from hip flexion is prevented and the muscles can be palpated during contraction.
- *Extension*—quadriceps femoris (this muscle is three times as strong as its antagonists, the hamstrings)—(L3, L4): with the knee slightly bent, ask the patient to straighten the knee and not let you bend it (see Fig. 34.34).

Ankle

- *Plantar flexion*—gastrocnemius, plantaris, soleus—(S1, S2): ask the patient to push the foot down and not let you push it up (see Fig. 34.35).
- *Dorsiflexion*—tibialis anterior, extensor digitorum longus and extensor hallucis longus—(L4, L5): ask the patient to bring the foot up and not let you push it down (see Fig. 34.36). The power of the ankle joint can also be tested by having the patient stand up on the toes (plantar flexion) or on the heels (dorsiflexion); these movements may also be limited if coordination is impaired.

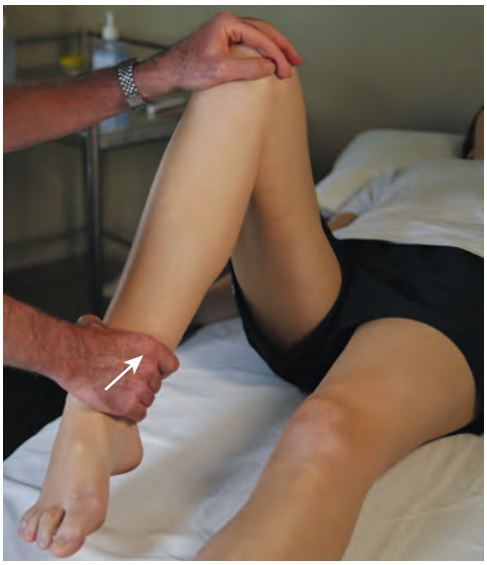


Testing power—knee flexion: 'Bend your knee and don't let me straighten it'; pull hard

FIGURE 34.33

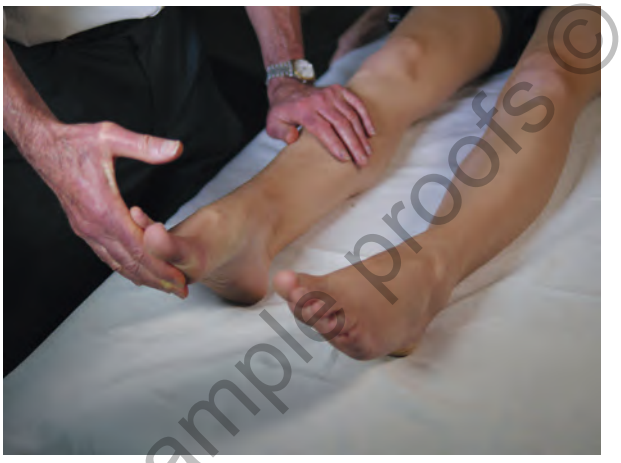
Tarsal joint

- *Eversion*—peroneus longus and brevis, and extensor digitorum longus—(L5, S1): evert the foot for the patient and ask him or her to hold it there (see Fig. 34.37).
- *Inversion*—tibialis posterior, gastrocnemius and hallucis longus—(L5, S1): invert the foot for the patient and ask him or her to hold it there (see Fig. 34.38).



Testing power—knee extension: 'Straighten your knee and don't let me bend it'; push hard

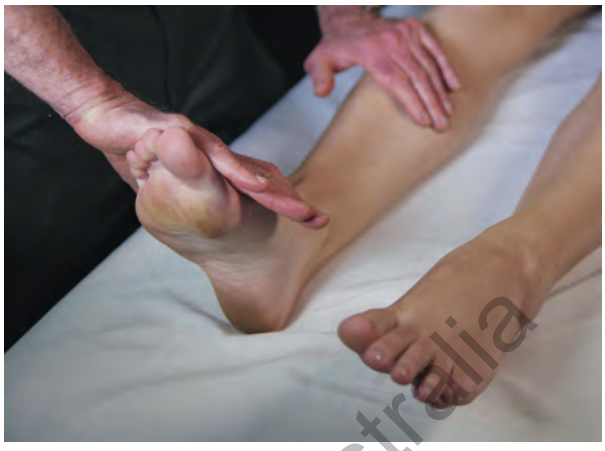
FIGURE 34.34



Testing power—ankle, plantar flexion: 'Don't let me push your foot up'

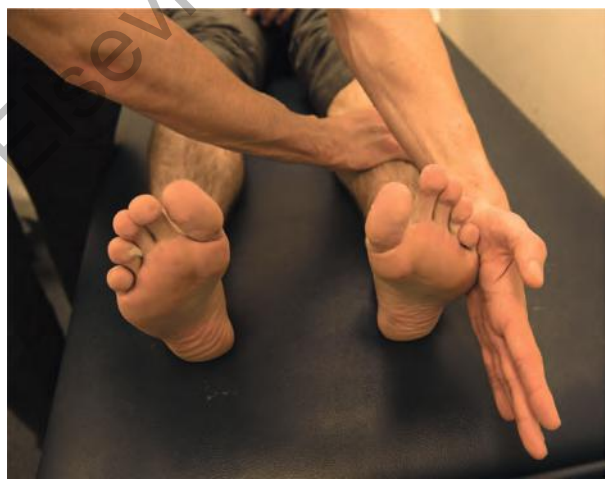
FIGURE 34.35

Non-organic or functional unilateral limb weakness may be detected by Hoover's test. Normally, when a patient attempts to resist movement, the contralateral limb works to support the effort. For example, when a patient attempts to extend the leg against resistance, the other leg pushes down into the bed. If this movement is absent, Hoover's[o] sign is positive.



Testing power—ankle, dorsiflexion: 'Don't let me push your foot down'

FIGURE 34.36



Testing power—ankle (tarsal joint) eversion: 'Stop me turning your foot inwards'

FIGURE 34.37

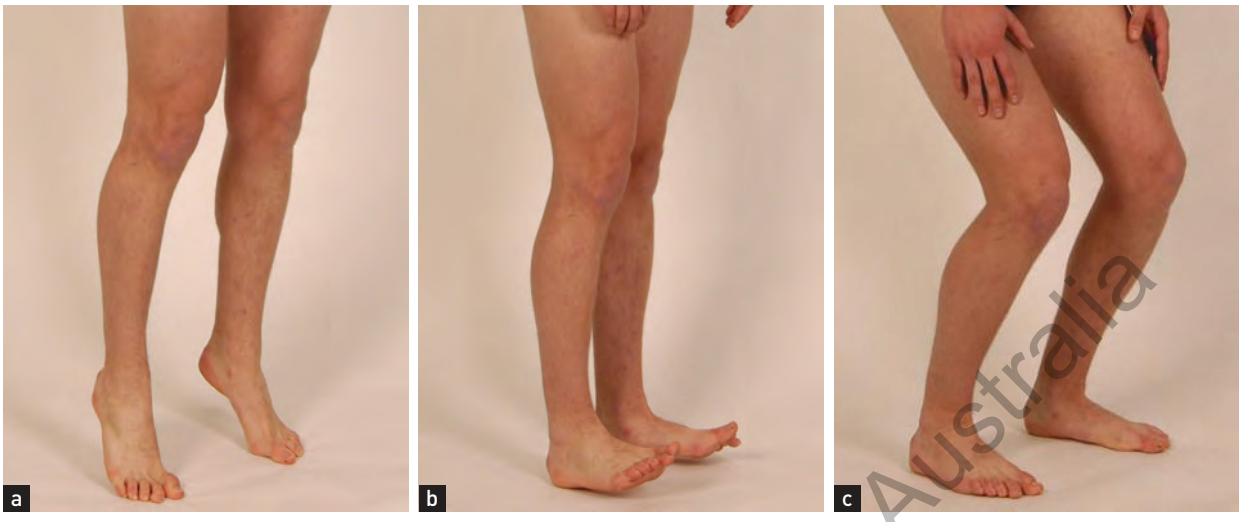
Quick test of lower limb power

The clinician in a hurry can test lower limb power quickly by asking the patient to:

1. stand up on his or her toes (S1) (see Fig. 34.39(a))

[o] Charles Hoover also described an important sign of chronic obstructive pulmonary disease.

Quick test of lower limb power.



(a) 'Stand up on your toes.' (b) 'Now lift up on your heels.' (c) 'Now squat and stand up again.'

FIGURE 34.39

2. stand up on the heels (L4, L5) (see Fig. 34.39(b))
3. squat and stand again (L3, L4) (see Fig. 34.39(c)).

This tests ankle, knee and hip power. Inability to perform any of the tests suggests a need to test more formally.

Testing power—ankle (tarsal joint) inversion: 'Stop me turning your foot outwards'

FIGURE 34.38

Reflexes

Test the following reflexes.

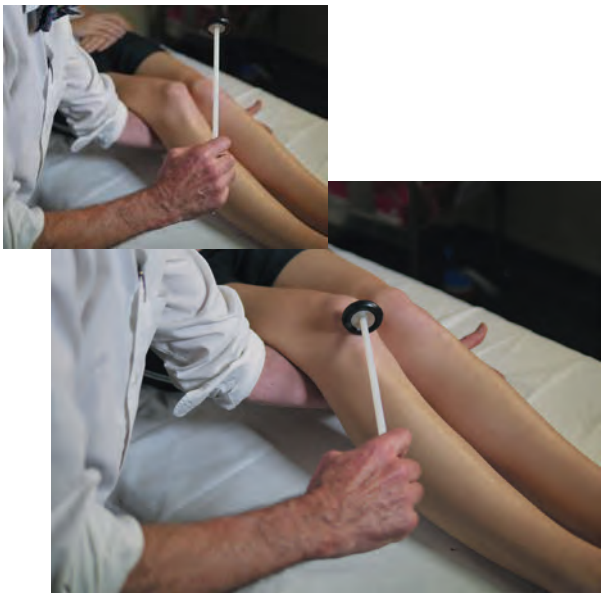
Knee jerk (L3, L4)

Slide one arm under the patient's knees so that they are slightly bent and supported. The tendon hammer is allowed to fall onto the infrapatellar tendon (see Fig. 34.40). Normally, contraction of the quadriceps causes extension of the knee. Compare the two sides. If the knee jerk appears to be absent on one or both sides, it should be tested again following a reinforcement manoeuvre. Ask the patient to interlock the fingers and then pull apart hard at the moment before the hammer strikes the tendon (Jendrassik's manoeuvre;[p] see Fig. 34.41). This manoeuvre has been shown to restore an apparently absent ankle jerk in 70% of elderly people. A reinforcement manoeuvre such as this, or teeth-clenching or grasping an object, should be used if there is difficulty eliciting any of the muscle stretch reflexes.

Ankle jerk (S1, S2)

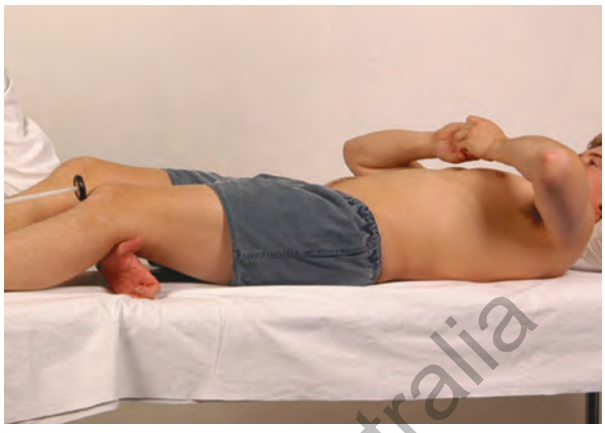
There are a number of patterns of abnormal ankle jerks (see Table 34.5). The examiner's technique is more

[p] Ernst Jendrassik (1858–1921), a Budapest physician.



The knee jerk examination

FIGURE 34.40



The knee jerk with reinforcement: 'Grip your fingers and pull your hands apart'

FIGURE 34.41

Patterns of ankle jerk abnormalities

Pattern	Significance
Asymmetrical loss	Radiculopathy
Symmetrical loss or reduction	• Large fibre sensory neuropathy, e.g. in diabetic neuropathy • Muscle disease, e.g. motor neurone disease • Poor examination technique (rather common)
Symmetrical increase	Increased sympathetic nervous system activity, e.g. anxiety
Asymmetrical increase	Upper motor neurone lesion, e.g. stroke (especially if associated signs such as clonus and increased tone are present)

TABLE 34.5

important for this difficult and interesting reflex than for many of the other tendon reflexes.[q]

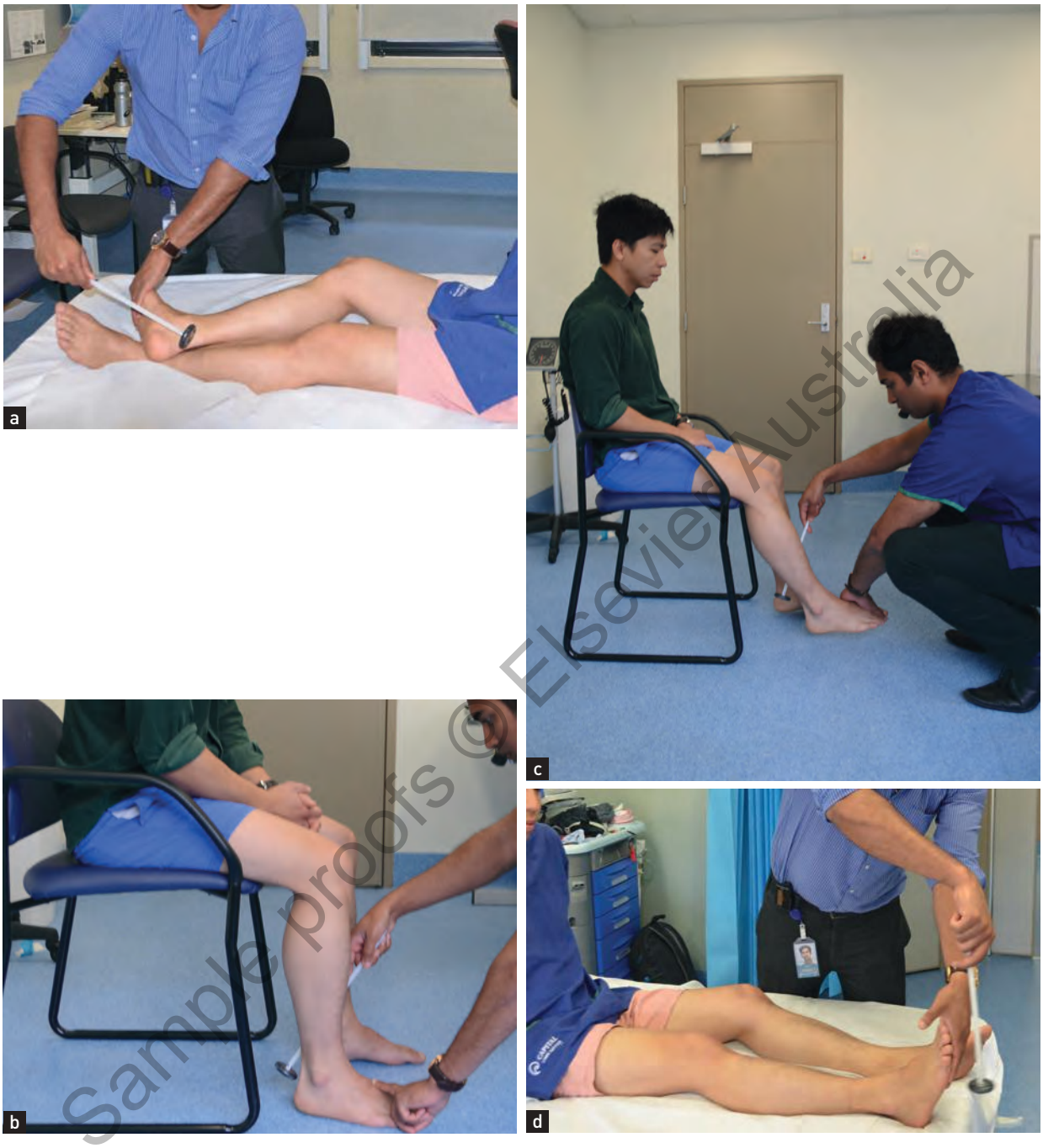
Method 1 (traditional): Have the foot in the mid-position at the ankle with the knee bent, the thigh externally rotated on the bed and the foot held in dorsiflexion by you. Allow the hammer to fall on the Achilles tendon (see Fig. 34.42(a)). The normal response is plantar flexion of the foot with contraction of the gastrocnemius muscle. This method can be convenient as part of the examination of a patient already lying in bed. It is a slightly uncomfortable position sometimes for the patient, especially if external rotation of the hip is painful or limited. If patients have difficulty holding this position, the examiner may try to stabilise the leg by dorsiflexing the foot. This will suppress the reflex.

Method 2 (ideal for hung-up reflex detection): The patient kneels on a chair (see Fig 28.11 on p 475).[7] Do not allow a nervous patient to grip the back of the chair unless the Dendrassik manoeuvre is needed.

Method 3 (clinician not concerned about his or her own dignity): The patient sits in a chair with feet flat on the ground and legs flexed to 90° (Fig. 34.42(b)). This position produces tendon stretch and muscle relaxation.

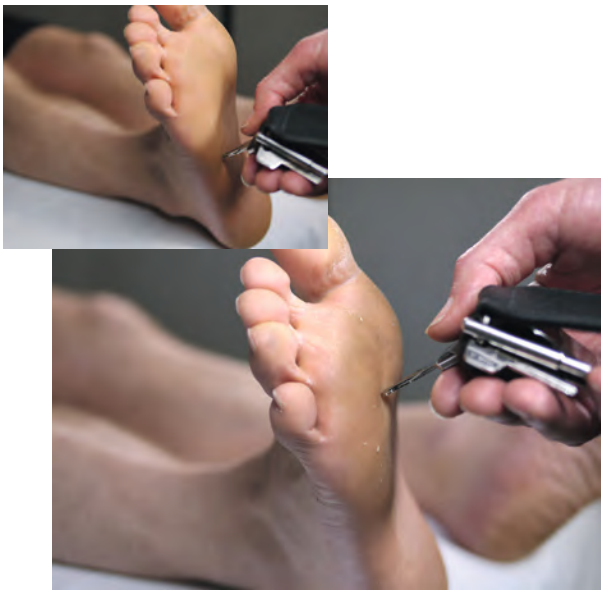
Method 4 (still somewhat undignified position for the clinician): The patient dangles his or her legs over a bed or chair (Fig 34.42(c)). The clinician supports the foot to keep the ankle in the neutral position. This helps prevent the temptation of the patient to dorsiflex the foot, which will prevent the reflex plantar flexion.

[q] When an ankle jerk is elicited, the response can be seen, felt and heard. When the response is present, the tendon produces an undamped oscillation heard as a 'boing', and when it is absent, the damped oscillation is heard as a dull 'thud'.



(a) Method 1 (see also p 475): the examiner dorsiflexes the foot slightly to stretch the tendon. (b) Method 3: patient sitting. (c) Method 4: patient dangles legs. (d) Method 5: the clinician strikes their hand on the sole of the patient's foot

FIGURE 34.42



The plantar reflex examination

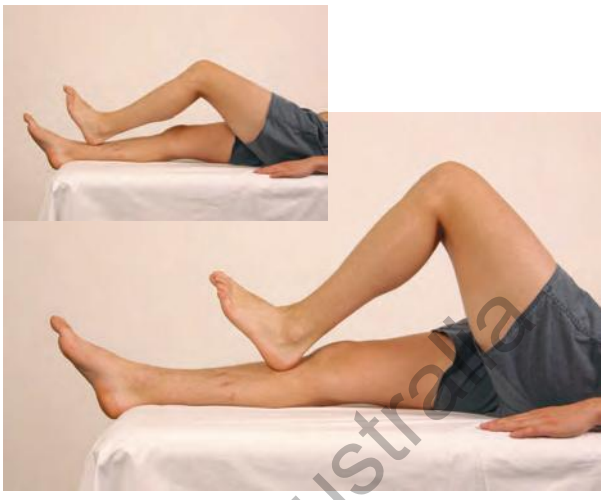
FIGURE 34.43

Method 5 (popular with neurologists): The patient lies in a bed (Fig 34.42(d)). The clinician rests his or her hands on the sole of the foot and is careful not to dorsiflex the ankle.[7] The hammer is struck onto the examiner's hand. This method is thought to lead to more false-negative results, though presumably not when performed by neurologists.

Plantar reflex (L5, S1, S2)

After telling the patient what is going to happen, use a blunt object (such as the key to an expensive car) to stroke up the lateral aspect of the sole and curve inwards before it reaches the toes, moving towards the middle metatarsophalangeal (MTP) joint (see Fig. 34.43). The patient's foot should be in the same position as for testing the ankle jerk. The normal response is flexion of the big toe at the MTP joint in patients over 1 year of age. The extensor (Babinski[r])[8] response is abnormal and is characterised by extension of the big toe at the MTP joint (the upgoing toe) and fanning of the other toes. This indicates an upper motor neurone (pyramidal) lesion, although the test's reliability can be relatively poor. Bilateral upgoing toes may also be found after a generalised seizure, or in a patient in a coma.

[r] Josef Babinski (1857–1932), a Parisian neurologist of Polish extraction, described this sign in 1896. Somatisation disorders vary in their nature according to fashion. In the 19th century, hysterical paralysis was common. Babinski, while strolling through his ward, casually stroked the soles of the feet of two patients in adjoining beds (this would probably lead to his being struck off the medical register today). He noticed that the big toe of the woman thought to have hysterical paralysis moved down and that of the woman whose paralysis was thought genuine and due to a stroke moved up. The sign was probably first described by Ernst Remak in 1893. Babinski was a famous gourmet and assistant to Charcot.



The heel–shin test: 'Run your heel down your shin smoothly and quickly'

FIGURE 34.44

Coordination

Test for cerebellar disease with three manoeuvres.

Heel–shin test

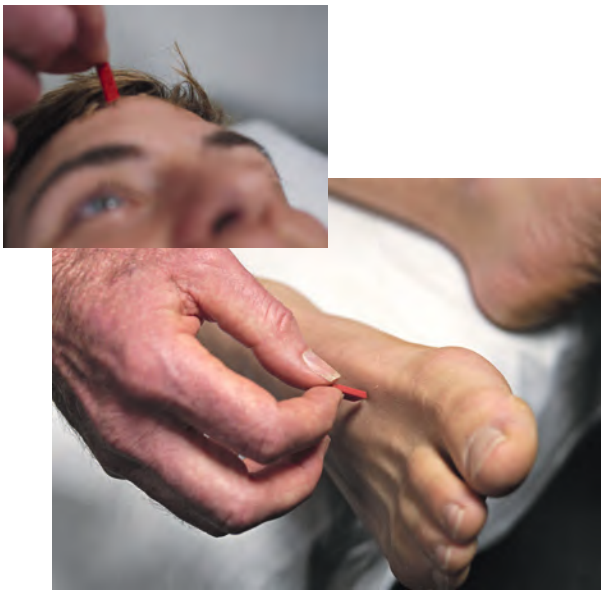
Ask the patient to run the heel of one foot up and down the opposite shin at a moderate pace and as accurately as possible (see Fig. 34.44). In cerebellar disease the heel wobbles all over the place, with oscillations from side to side and overshooting. Closing the eyes makes little difference to this in cerebellar disease, but if there is posterior column loss the movements are made worse when the eyes are shut—that is, there is 'sensory ataxia'.

Toe–finger test

Unfortunately, a toe–nose test is not a practical way of assessing the lower limbs, so a toe–finger test is used. Ask the patient to lift the foot (with the knee bent) and touch your finger with the big toe. Look for intention tremor.

Foot-tapping test

Rapidly alternating movements are tested by getting the patient to tap the sole of the foot quickly on your hand or to tap the heel on the opposite shin. Look for loss of rhythmicity.



Testing pinprick (pain) sensation in the lower limbs (do not draw blood with the pin)

FIGURE 34.45

The sensory system

As for the upper limb, test for pain or cold sensation or both, first in each dermatome, starting distally and comparing the right with the left side (see Fig. 34.45). Map out any abnormality and decide on the pattern of loss.

Then test vibration sense over the great toe or malleoli or both and, if it is absent there, on the knees and if necessary on the anterior superior iliac spines (see Fig. 34.46). Next test proprioception, using the big toes (see Fig. 34.47) and, if necessary, the knees and hips.

Finally, test light touch bilaterally for neglect. Light touch can also be tested using a twirl of cottonwool or a microfibre tester (see Fig. 34.48). This sensation is especially protective against the development of ulcers in the feet.

Dermatomes

Memorise the following rough guide (see Fig. 34.49):

- L2 supplies the upper anterior thigh.
- L3 supplies the area around the front of the knee.
- L4 supplies the medial aspect of the leg.
- L5 supplies the lateral aspect of the leg and the medial side of the dorsum of the foot.
- S1 supplies the heel and most of the sole.
- S2 supplies the posterior aspect of the thigh.
- S3, S4 and S5 supply concentric rings around the anus.

Sensory levels

If there is peripheral sensory loss in the leg, attempt to map out the upper level with a pin, moving up at 5-centimetre intervals initially, from the leg to the abdomen, until the patient reports it to be sharp. This may involve testing over the abdominal or even the chest dermatomes. Establishing a sensory level on the trunk indicates the spinal cord level that is affected. Remember, a level of hyperaesthesia (increased sensitivity) often occurs above the sensory level and it is the upper level of this that should be determined, as it usually indicates the highest affected spinal segment. Also remember that the level of a vertebral body only corresponds to the spinal cord level in the upper cervical cord because the spinal cord is shorter than the spinal canal. The C8 spinal segment lies opposite the C7 vertebra. In the upper thoracic cord there is a difference of about two segments and in the midthoracic cord it is three segments. All the lumbosacral segments are opposite the T11 to L1 vertebrae.

The superficial or cutaneous reflexes

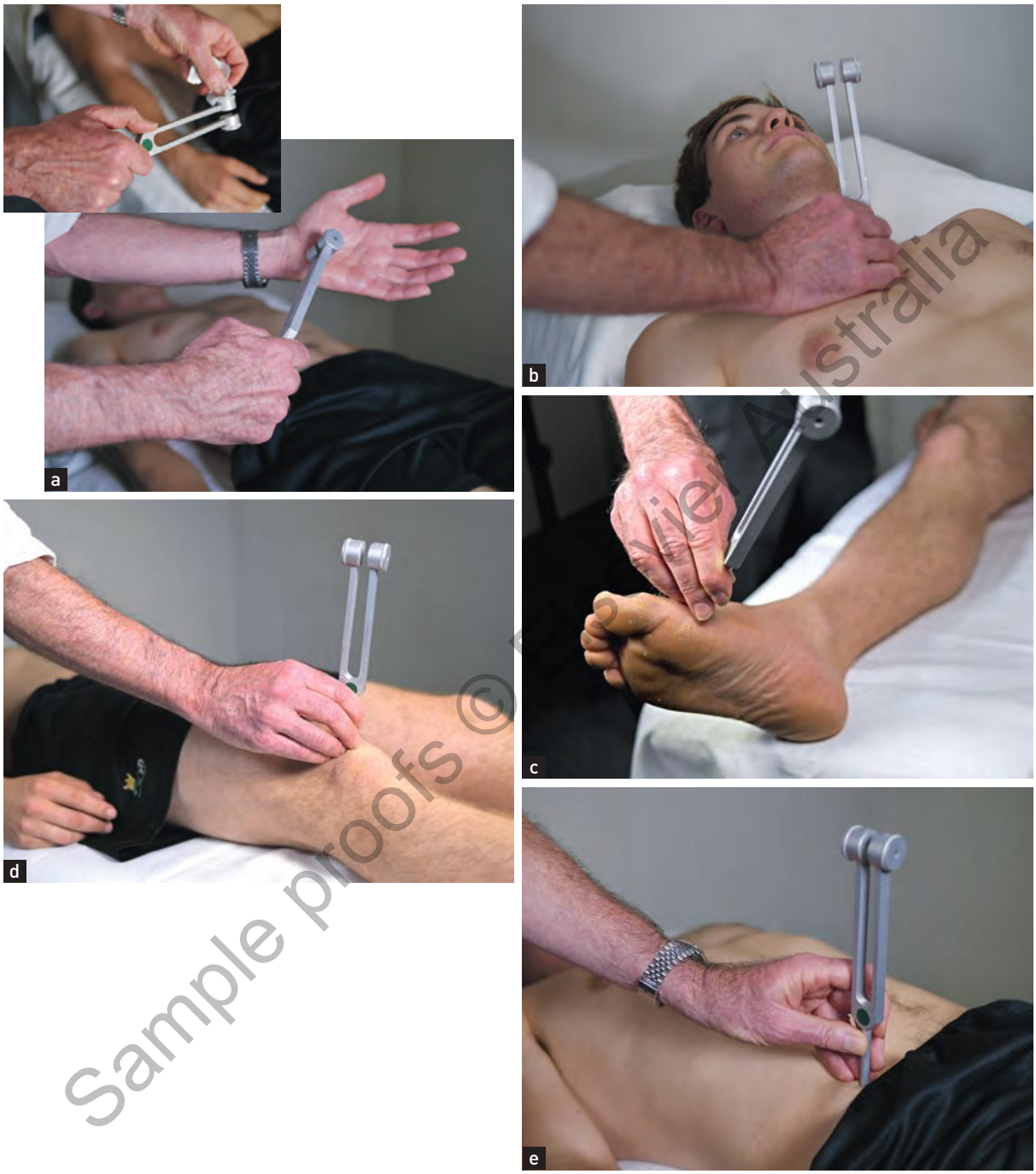
These reflexes occur in response to light touch or scratching of the skin or mucous membranes. The stimulus is more superficial than the tendon (muscle stretch) reflexes. As a rule these reflexes occur more slowly after the stimulus, are less constantly present and fatigue more easily.

Examples include the palmar or grasp reflex, the abdominal reflexes, the cremasteric reflex and the plantar responses.

The abdominal reflexes (epigastric T6–T9, midabdominal T9–T11, lower abdominal T11–L1) (See the OSCE video Abdominal reflexes at Student CONSULT)

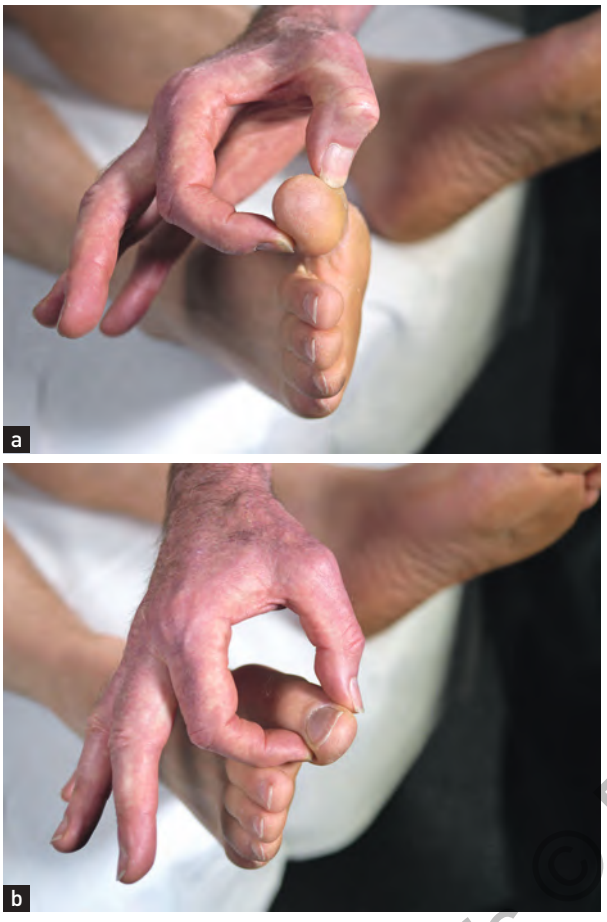
Test these by lightly stroking the abdominal wall diagonally towards the umbilicus in each of the four quadrants of the abdomen (see Fig. 34.50). Reflex contractions of the abdominal wall are *absent* in upper motor neurone lesions above the segmental level and also in patients who have had surgical

Testing vibration sense in the lower limbs.



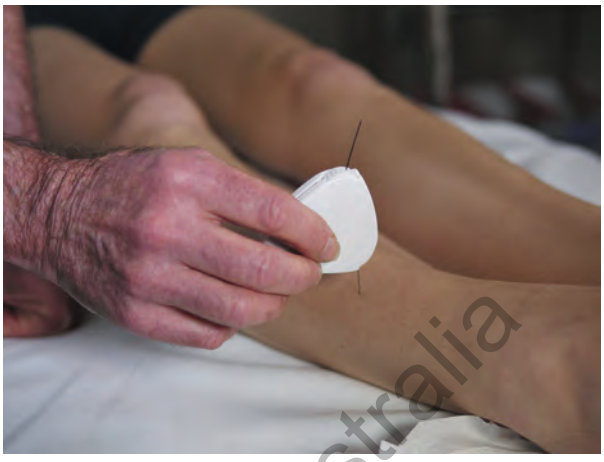
(a) Strike a 128 Hz tuning fork confidently on your thenar eminence. (b) Demonstrate the vibration of the tuning fork on the patient's sternum: 'Can you feel this vibration?' (c) Place the tuning fork on the great toe: 'Can you feel the vibration there? Tell me when it stops.' (d) If vibration sense is absent on the great toe, try testing on the patella. (e) If vibration sense is absent at the knee, try testing on the anterior superior iliac spine

FIGURE 34.46



Position sense: 'Shut your eyes and tell me whether I have moved your toe up or down'

FIGURE 34.47



Testing touch sensation with a monofilament. Cotton wool can be used as an alternative but do not stroke the skin

FIGURE 34.48

operations interrupting the nerves. They disappear in coma and deep sleep, and during anaesthesia. They are usually difficult to elicit in obese patients and can also be absent in some healthy people (20%). Their absence in the presence of increased tendon reflexes is suggestive of corticospinal tract abnormality.

The cremasteric reflexes (L1–L2)

Stroke the inner part of the thigh in a downward direction; normally contraction of the cremasteric muscle pulls up the scrotum and testis on the side stroked. It may be absent in elderly men and in those with a hydrocele or varicocele, or after an episode of orchitis. This is seldom tested, unless there are specific concerns about this region (e.g. from the history or because of a known L1, L2 lesion).

Saddle sensation and anal reflex

Test now for saddle sensation if a cauda equina lesion is suspected (e.g. because of urinary or faecal incontinence). The only sensory loss may be on the buttocks or around the anus (S3–S5) (see List 34.7). In this case also test the anal reflex (S2–S4): normal contraction of the external sphincter in response to pinprick of the perianal skin is abolished in patients with a lesion of the sacral segments of the cauda equina. If, however, the lowest sacral segments are spared but the higher ones are involved, this suggests that there is an intrinsic cord lesion.

Spine

Examine the spine with the patient standing. Look and feel for:

- scars
- scoliosis
- a midline pit or patch of hair (spina bifida)
- tenderness (malignancy or infection).

Ask the patient to lie down and perform the straight-leg-raising test (tests for disc herniation, which causes pain in the sciatic nerve distribution—p 596).

CAUSES OF CAUDA EQUINA SYNDOME

- Spinal tumours and metastatic lesions
- Spinal infection
- Violent lower back injuries (e.g. car accidents, falls)
- Spinal haemorrhage
- Lumbar spinal canal stenosis
- Intervertebral disc disease
- Lumbar spine surgery, or complications of spinal or epidural anaesthetic

LIST 34.7

Examination of the peripheral nerves of the lower limb

Lateral cutaneous nerve of the thigh

Test for sensory loss (see Fig. 34.51). A lesion of this nerve usually occurs because of entrapment between the inguinal ligament and the anterior superior iliac spine. It is more common in people who are overweight and in those who spend much of their time sitting (e.g. truck drivers, public servants). It causes a sensory loss over the lateral aspect of the thigh with no motor loss detectable. If painful, it is called *meralgia paraesthetica*.

Femoral nerve (L2, L3, L4)

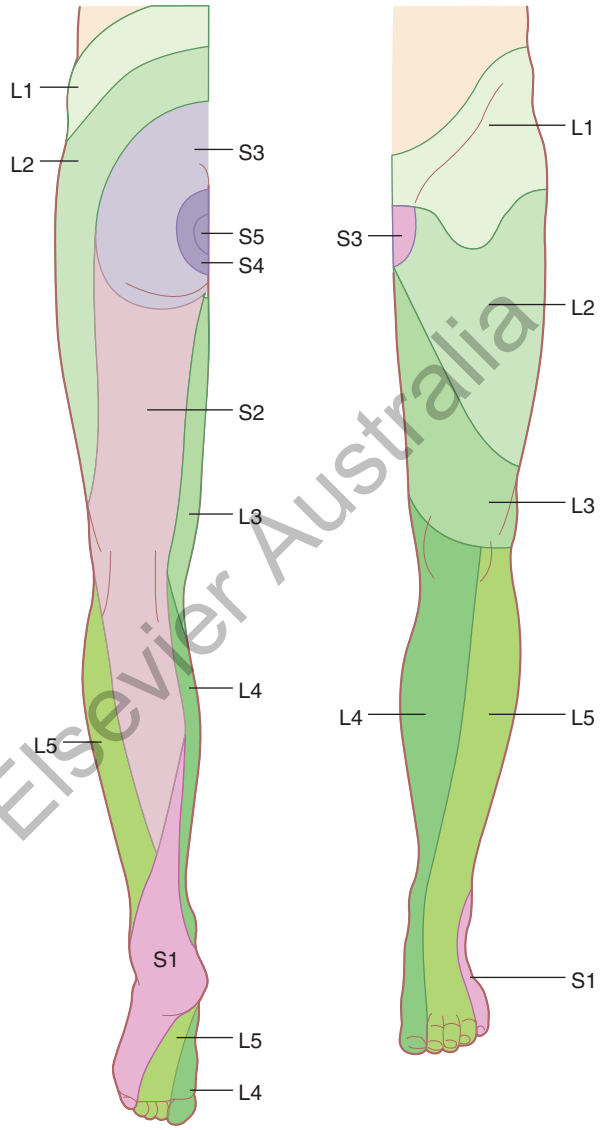
Test for weakness of knee extension (quadriceps paralysis). Hip flexion weakness is only slight, and adductor strength is preserved. The knee jerk is absent. The sensory loss involves the inner aspect of the thigh and leg (see Fig. 34.52).

Sciatic nerve (L4, L5, S1, S2)

This nerve supplies all the muscles below the knee and the hamstrings. Test for loss of power below the knee resulting in a foot drop (plantar-flexed foot) and for weakness of knee flexion. Test the reflexes: with a sciatic nerve lesion the knee jerk is intact but the ankle jerk and plantar response are absent. Test sensation on the posterior thigh, the lateral and posterior calf, and the foot (lost with a proximal nerve lesion; see Fig. 34.53).

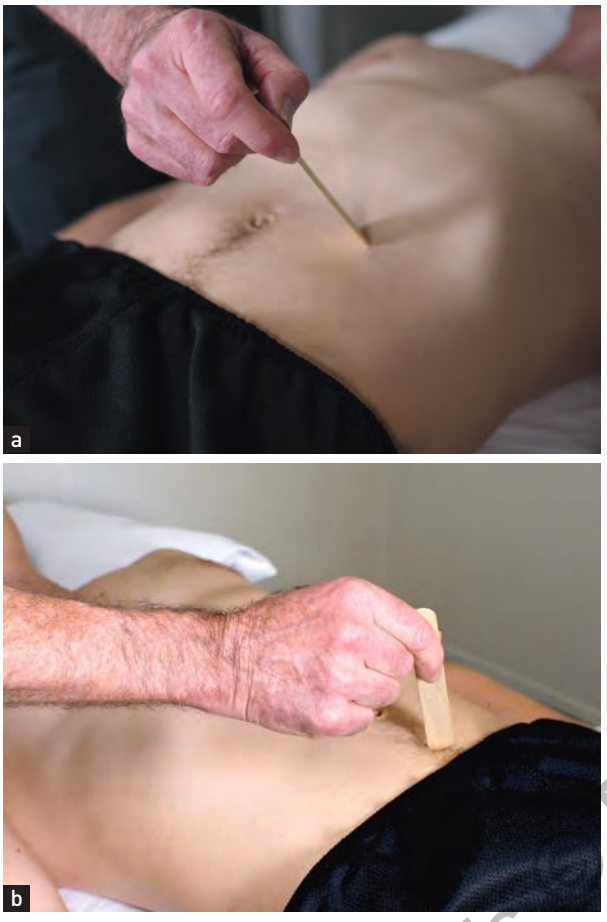
Common peroneal nerve (L4, L5, S1)

This is a major terminal branch of the sciatic nerve. It supplies the anterior and lateral compartment muscles of the leg (see Fig. 34.54). On inspection one may notice a foot drop (see List 34.8 and Fig. 34.55). Test for weakness of dorsiflexion and eversion. Test the reflexes, which will all be intact. Test for sensory loss. There may be only minimal sensory loss over the lateral aspect of the dorsum of the foot. Note that these findings can be confused with an L5 root lesion, but the latter includes weakness of knee flexion and loss of foot inversion as well as sensory loss in the L5 distribution.



Dermatomes of the lower limbs

FIGURE 34.49



Abdominal reflex: stroke towards the umbilicus from each quadrant and watch for abdominal muscle contraction

FIGURE 34.50

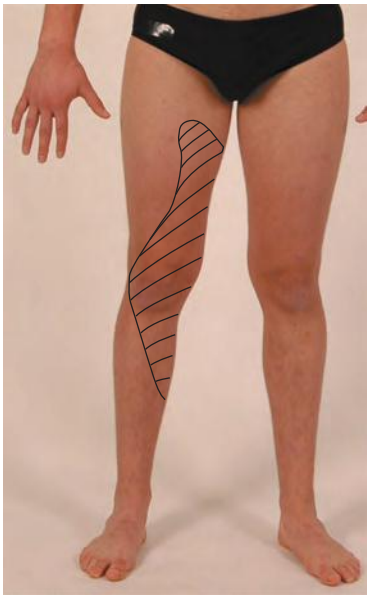
Gait

Make sure the patient's legs are clearly visible. Ask the patient to walk normally for a few metres and turn around quickly and walk back and then walk heel to toe to exclude a midline cerebellar lesion (see Fig. 34.56). Ask the patient to walk on the toes (an S1 lesion will make this difficult or impossible) and then on the heels (an L4 or L5 lesion causing foot drop will make this difficult or impossible).

Test for proximal myopathy by asking the patient to squat and then stand up, or to sit in a low chair and then stand.

Distribution of the lateral cutaneous nerve of the thigh

FIGURE 34.51

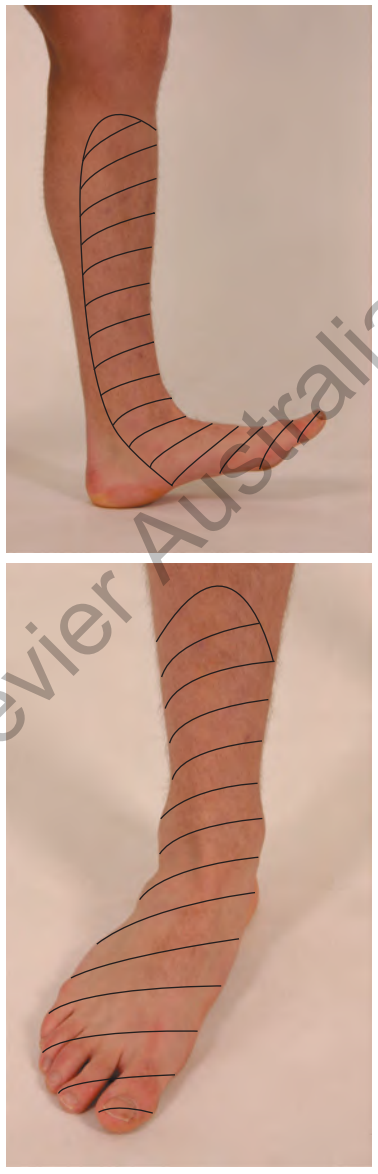


Sensory distribution of the femoral nerve

FIGURE 34.52

Sensory distribution of the sciatic nerve

FIGURE 34.53



Sensory distribution of the common peroneal nerve (compression at the fibular neck)

FIGURE 34.54

To test *station* (Romberg[5] test), ask the patient to stand erect with the feet together and the eyes open (see Fig. 34.57(a)) and then, once the patient is stable, to close the eyes (see Fig. 34.57(b)). Compare the steadiness shown with the eyes open, then closed for up to 1 minute. Even in the absence of neurological disease a person may be slightly unsteady with the eyes closed, but the sign is positive if marked unsteadiness occurs to the point where the patient looks likely to fall. Normal people can maintain this position easily for 60 seconds. The Romberg test is positive when unsteadiness increases with eye closure. This is usually seen with the loss of proprioceptive sensation; unsteadiness is worse when visual information about position is removed.

Marked unsteadiness with the eyes open is seen with severe proprioceptive loss, cerebellar or vestibular dysfunction.

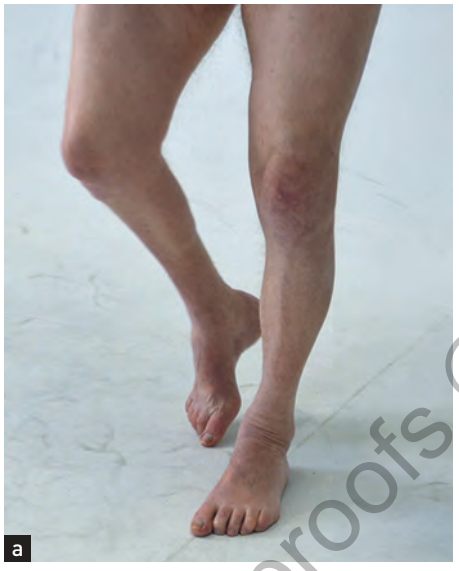
Gait disorders are summarised in List 34.9.

[5] Moritz Heinrich von Romberg (1795–1873), a Berlin professor, wrote the first modern neurology textbook. His original description of the sign was of patients with tabes dorsalis (dorsal column disease caused by syphilis).

CAUSES (DIFFERENTIAL DIAGNOSIS) OF FOOT DROP

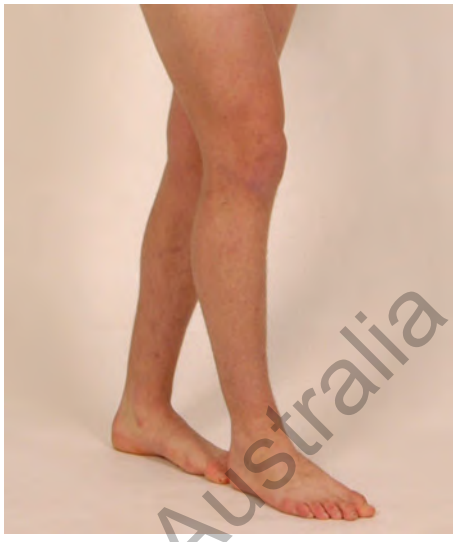
Common peroneal nerve palsy
Sciatic nerve palsy
Lumbosacral plexus lesion
L4, L5 root lesion
Peripheral motor neuropathy
Distal myopathy
Motor neurone disease
Stroke—anterior cerebral artery or lacunar syndrome ('ataxic hemiparesis')

LIST 34.8



(a) Foot drop: the patient lifts the affected leg high in the air to prevent the foot scraping on the ground. (b) Shoe supports to prevent foot drop

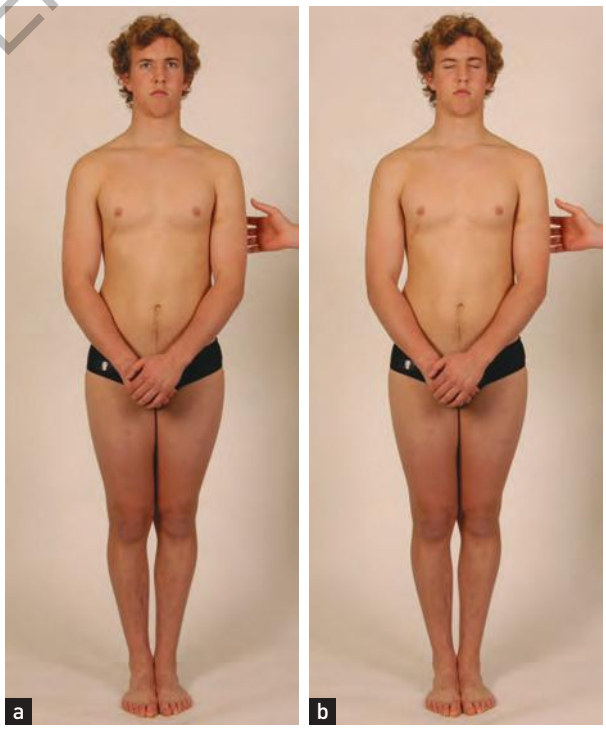
FIGURE 34.55



Cerebellar testing—heel–toe walking

FIGURE 34.56

Romberg test.



(a) 'Stand with your feet together'. (b) 'Now shut your eyes. I won't let you fall'

FIGURE 34.57

GAIT DISORDERS

Hemiplegia: the foot is plantar flexed and the leg is swung in a lateral arc

Spastic paraparesis: scissors gait

Parkinson's disease:

- Hesitation in starting
- Shuffling
- Freezing
- Festination
- Propulsion
- Retropulsion

Cerebellar: a drunken gait that is wide-based or reeling on a narrow base; the patient staggers towards the affected side if there is a unilateral cerebellar hemisphere lesion

Posterior column lesion: clumsy slapping down of the feet on a broad base

Foot drop: high-stepping gait

Proximal myopathy: waddling gait

Prefrontal lobe (apraxic): feet appear glued to the floor when erect, but move more easily when the patient is supine. Arm swing is typically well preserved, helping to distinguish it from a Parkinsonian gait

Conversion disorder (hysteria): characterised by a bizarre, inconsistent gait

LIST 34.9

T&O'C ESSENTIALS

1. *Compare both sides and observe any lack of symmetry in the limb examination.*
2. *Reflexes are best thought of as being present, increased or absent; more specific grades are not really useful.*
3. *There are large normal variations in tone.*
4. *There are even more variations in pinprick sensation and sometimes one can say only that the findings on examination are not useful.*

OSCE REVISION TOPICS – THE PERIPHERAL NERVOUS SYSTEM

Use these topics, which commonly occur in the OSCE, to help with revision.

1. This man has noticed a problem with balance. Please examine his lower limbs from a cerebellar aspect. (pp 592, 597–8)
2. This man has difficulty lifting his right foot when he walks. Please examine him. (pp 585–6, 597–8)
3. Please assess the reflexes in this man's lower limbs. (p 589)
4. Please test the power in this woman's arms. (p 571)
5. This woman has been noticed to have wasting of the small muscles of her left hand. Please examine her. (pp 569, 578–80)

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CHAPTER 40

The gynaecological history and examination

Wendy Carseldine and Ian Symonds

The good physician treats the disease; the great physician treats the patient who has the disease.

SIR WILLIAM OSLER (1849–1919)

Gynaecology is the field of medicine that includes disorders of the female genital tract and reproductive system. It also includes complications of early pregnancy such as miscarriage and ectopic pregnancy.

HISTORY

As with all areas of medicine a precise and comprehensive history is the most important component in making an accurate gynaecological diagnosis. The basic structure of the gynaecological history is similar to that for other systems, but with more emphasis on the patient's menstrual, sexual and past reproductive history. It is important to note the patient's age as this will influence the likely diagnosis for a number of presenting problems. Occupation may be relevant both to the level of understanding that can be assumed and to the effect of different gynaecological problems on the patient's life. Pay attention to LGBTI patients; refer to Chapter 2.

A method for the gynaecological history and examination is given in Text box 40.1.

Presenting symptoms

Ask the patient to describe the nature of her problem. Where there are multiple symptoms these may not be related to the same pathology and it is useful to address each of these in turn with appropriate open and closed questioning. Ascertain the timescale of the problem and, where appropriate, the circumstances surrounding the onset of symptoms and their relationship to the menstrual cycle. It is also important to discover the degree of disability experienced for any given symptom.

More detailed questions will depend on the nature of the presenting complaint. List 40.1 outlines some of the more common presenting symptoms encountered in the outpatient clinic. Further details for some of these are discussed below.

COMMON PRESENTING SYMPTOMS IN GYNAECOLOGY

- Disorders of menstruation
- Heavy menstrual bleeding
- Intermenstrual bleeding
- Oligomenorrhoea / amenorrhoea
- Postmenopausal bleeding
- Premenstrual syndrome
- Disorders of sexual function
- Dyspareunia
- Loss of libido
- Vaginismus
- Infertility
- Pain and / or bleeding in early pregnancy
- Pelvic / lower abdominal pain: acute and chronic
- Perimenopausal symptoms
- Symptoms of uterovaginal prolapse
- Urinary incontinence
- Urinary urgency / frequency
- Voiding difficulties
- Vaginal discharge and genital tract infection
- Vulval pain / pruritus

LIST 40.1

The gynaecological history and examination: a suggested method

1. **History**
- Presenting symptoms
  - Onset and duration of main complaint
  - Associated symptoms, relationship to menstrual cycle
  - Previous treatment and response
  - Specific closed questions
- Previous gynaecological history
  - Previous investigations or treatment
  - Contraceptive history
  - Sexual history
  - Cervical screening test
  - Menstrual history (menarche, last menstrual period, cycle)
- Previous pregnancies
  - How many (gravidity)
  - Outcome (parity)
  - Surgical deliveries
  - Birth weight and health of previous babies
- Past surgical and medical history
  - Previous abdominal surgery
  - Major cardiovascular/respiratory disease
  - Endocrine disease
  - Thromboembolic disease
  - Breast disease
- Drug history and allergies
- Social and family history
  - Home circumstances
  - Support
  - Smoking
  - Family history

2. **Examination**
- General examination
  - General condition, weight, height
  - Pulse, blood pressure
  - Anaemia
  - Goitre
  - Breast examination (if indicated)
  - Secondary sex characteristics, body hair, acne
- Abdominal examination
  - Inspection: distension, scars
  - Palpation: masses, organomegaly, tenderness, peritonism, nodes, hernial orifices
  - Percussion: ascites
- Pelvic examination
  - Explanation, comfort, privacy, chaperone
  - Inspection of external genitalia
  - Speculum examination
  - Bimanual examination
- Rectal examination, if indicated

TEXT BOX 40.1

Menstrual history

A full menstrual history should be taken from all women of reproductive age, starting with the first day of the last menstrual period.

The length of the menstrual cycle is the time between the first day of one period and the first day of the following period. While there is usually an interval of between 26 and 32 days, occasionally the duration of the cycle may be as short as 21 days or as long as 42 days without necessarily indicating any underlying abnormality.

Normal menstruation lasts from 4 to 7 days, and normal blood loss varies between 30 and 80 mL. A change in pattern is often more noticeable and significant than the actual duration and volume of loss. Self-reported assessment of the amount of bleeding is subjective, with up to 50% of women who report excessive bleeding actually having a measured blood loss of less than 80 mL. Excessively prolonged or heavy regular periods are sometimes referred to as **menorrhagia**, but the term **heavy menstrual bleeding (HMB)** is now generally used to describe all excessive menstrual blood loss regardless of the regularity of the cycle (see below).

The cessation of periods for at least 12 months at the end of menstrual life is known as the **menopause**. Bleeding that occurs after this is described as **postmenopausal bleeding**. A history of irregular vaginal bleeding or blood loss that occurs after coitus or between periods should be noted.

Heavy menstrual bleeding

HMB is defined as menstrual loss of more than 80 mL per month or 'excessive menstrual loss leading to

CAUSES OF HEAVY MENSTRUAL BLEEDING

Structural

- Uterine leiomyomata (fibroids)
- Adenomyosis
- Endometrial polyps
- Endometrial hyperplasia
- Endometrial cancer

Non-structural

- Disorders of ovulation (polycystic ovary syndrome, perimenopause, puberty)
- Clotting disorders
- Iatrogenic (intrauterine contraceptive device)
- Dysfunctional bleeding (see below)
- Hypothyroidism

LIST 40.2

interference with the physical, emotional, social and material quality of life in a woman and which occurs alone or in combination with other symptoms.'[1] It affects about 10% of women. In most cases the cause is an imbalance in the mechanisms that regulate clotting at the endometrial level without any obvious structural pathology. However, it may be associated with a number of benign pathologies and, occasionally, with malignancy (see List 40.2).

Diagnosis

In practice, the diagnosis of HMB is made on the basis of symptoms, not measured blood loss. Symptoms of clotting, flooding (soiling of clothing), the use of a large amount of sanitary protection, bleeding lasting more than 7 days and treatment for anaemia are likely to indicate HMB. A change in the pattern of bleeding is more likely to be associated with a structural lesion. Malignancy is rare in women under the age of 40. A history of polycystic ovary syndrome (PCOS), diabetes, hypertension and obesity are associated with an increased risk of endometrial hyperplasia and malignancy.

All women with HMB should have a general examination for signs of anaemia and thyroid disease and a pelvic examination including cervical screening. The finding of a pelvic mass is most likely to be associated with a diagnosis of fibroids but can indicate malignancy.

A full blood count (FBC) should be requested for all patients with HMB. Additional investigation is mainly directed at excluding malignancy and usually includes a pelvic ultrasound scan. Endoscopic examination of the endometrial cavity (hysteroscopy) with endometrial biopsy is indicated when:

- there is a history of irregular or intermenstrual bleeding
- risk factors for endometrial cancer are present
- pelvic examination is abnormal
- there is no response to first-line treatment.

Young women under the age of 25 should have a partial coagulation screen to exclude Von Willebrand's disease.

Bleeding in early pregnancy

Any women of reproductive age presenting with abnormal vaginal bleeding should have the possibility of pregnancy considered. If a pregnancy test is positive, the cause of the bleeding can usually be determined by a combination of vaginal examination and ultrasound (see Table 40.1). The most common cause of bleeding in early pregnancy (before 20 weeks) is miscarriage, or threatened miscarriage, although hydatidiform and ectopic pregnancy may also present with bleeding. Miscarriage is the spontaneous termination of pregnancy prior to 20 weeks' gestation; it affects about 15–20% of pregnancies. It is important to remember that bleeding in pregnancy can result from the same lower genital tract lesions as in non-pregnant women.

Amenorrhoea and oligomenorrhoea

The onset of the first period, the **menarche**, commonly occurs at 12 years of age and can be considered to be abnormally early at 7 years. **Primary amenorrhoea** is the absence of menarche. The age at which the diagnosis is made depends on the presence or absence of other features of puberty. In the absence of other signs of puberty this is 14 years, but where there are other pubertal changes it is 16.

Secondary amenorrhoea is defined as the cessation of menses for 6 months or more in a woman who has previously menstruated. **Oligomenorrhoea** is the occurrence of five or fewer periods over 12 months. In practice, the distinction between the two can be somewhat arbitrary as they share many of the same causes.

Diagnosis of bleeding in early pregnancy

Symptoms and examination	Ultrasound	Diagnosis
Light vaginal bleeding with minimal pain; closed cervix	Viable intrauterine pregnancy	Threatened miscarriage
Heavy vaginal bleeding with cramping lower abdominal pain; open cervix	Products of conception in uterus with or without fetal heartbeat	Inevitable miscarriage
Heavy vaginal bleeding with cramping; open cervix, possibly with tissue	Products of conception in uterus, usually without fetal heartbeat	Incomplete miscarriage
Painless light bleeding or no symptoms; closed cervix	No fetal pole or fetal heartbeat in gestation sac >25 mm diameter	Missed miscarriage
Minimal bleeding after history of heavier bleeding and cramps; closed cervix	Empty uterus	Complete miscarriage (also consider ectopic pregnancy and very early viable pregnancy)
Mild-to-moderate bleeding; uterus large for dates, exaggerated pregnancy symptoms (e.g. hyperemesis)	Snowstorm appearance with or without gestation sac	Molar pregnancy
Light vaginal bleeding with unilateral abdominal pain; closed cervix; cervical excitation	No intrauterine pregnancy when hCG >1500; free fluid outside the uterus and/or an adnexal mass	Ectopic pregnancy

hCG = human chorionic gonadotrophin.

TABLE 40.1

Diagnosis

Amenorrhoea can be physiological or pathological (see List 40.3), with the most common causes being pregnancy, menopause and lactation. The possibility of pregnancy should be excluded in any woman of reproductive age by undertaking a urinary pregnancy test. Ask about recent emotional stress, changes in weight, menopausal symptoms and current medication. Relevant findings on examination are a low or high body mass index, hirsutism (PCOS), galactorrhoea and bitemporal hemianopia (pituitary tumours).

In cases of primary amenorrhoea look for features of secondary sex characteristics development, signs of imperforate hymen (P) and features of Turner syndrome (short stature, wide carrying angle and widely spaced nipples).

The differential diagnosis is established by measurement of follicle-stimulating hormone (FSH), luteinising hormone (LH) and prolactin, and thyroid function tests. A pelvic ultrasound can provide additional evidence of polycystic ovary syndrome (POCS), ovarian tumours and abnormalities of the lower genital tract. In women with primary amenorrhoea, a karyotype should also be obtained.

Dysmenorrhoea

Dysmenorrhoea, or painful menstruation, is the most common of all gynaecological symptoms.

Primary dysmenorrhoea occurs in the absence of any significant pelvic pathology. It usually develops within the first 2 years of the menarche. The pain is typically described as central and cramping. Symptoms can be severely incapacitating, causing major disruption of social activities. The onset of symptoms is usually associated with the onset of menstrual blood loss but may begin on the day preceding menstruation. The pain occurs only in ovulatory cycles and often disappears or improves after the birth of the first child. Dysmenorrhoea may be associated with vomiting and diarrhoea. Pelvic examination reveals no abnormality of the pelvic organs.

Secondary or **acquired dysmenorrhoea** is more likely to be caused by organic pelvic pathology and usually has its onset many years after the menarche. Commonly associated pathologies include endometriosis (see Fig. 40.1), adenomyosis, pelvic infections and intrauterine lesions such as submucous fibroid polyps.

CAUSES OF AMENORRHOEA

Primary

- Constitutional
- Anatomical
  - Imperforate hymen
  - Transverse vaginal septum
  - Müllerian agenesis: Mayer–Rokitansky–Küster–Hauser syndrome
- Hypergonadotrophic hypogonadic
  - Androgen insensitivity (XY)
  - Turner syndrome
  - Gonadal dysgenesis
- Hypogonadotrophic hypogonadic
  - CNS lesions (tumours, infection, trauma)
  - Kallmann's syndrome

Secondary

- Physiological causes
  - Pregnancy
  - Lactation
  - Menopause
- Pathological causes
- Hypothalamic disorders
  - Excessive weight loss or exercise
  - Stress
  - Chronic kidney disease
- Pituitary disorders
  - Prolactin-secreting tumours of the anterior pituitary (micro- or macroadenoma)
  - Postpartum necrosis (Sheehan's syndrome)
  - Antidopaminergic drugs
- Ovarian disorders
  - Premature ovarian failure
  - Autoimmune disease
  - Surgical removal of the ovaries
  - Oestrogen- or testosterone-secreting ovarian neoplasms
- Polycystic ovary syndrome
- Failure of uterine response
  - Surgical removal of the uterus
  - Asherman syndrome
  - Cryptomenorrhoea
  - Cervical stenosis, as a result of surgical trauma or infection

LIST 40.3

Investigations

A careful history is of great importance in this condition. Pelvic examination should be performed and, if this is abnormal, a pelvic ultrasound scan arranged. Laparoscopy is the gold standard investigation for the diagnosis of conditions such as endometriosis. It is performed in cases with primary dysmenorrhoea only if the condition is particularly resistant to therapy.

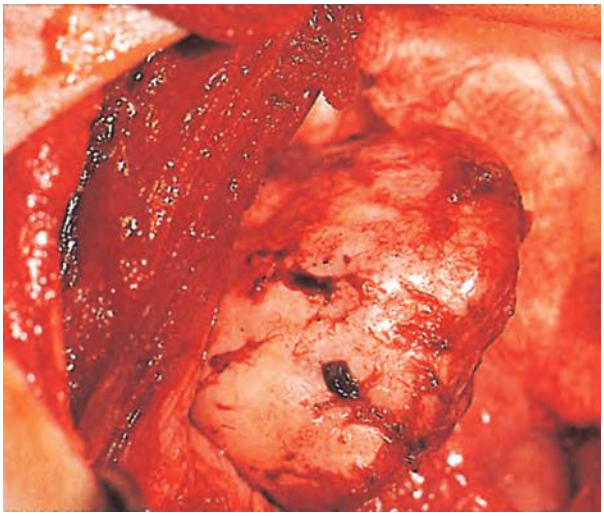
Acute abdominal pain

In women with a negative pregnancy test and acute pelvic pain, gynaecological disorders include pelvic inflammatory disease (PID), functional ovarian cysts, ovarian or peritoneal endometriosis and ovarian torsion. The most common gastrointestinal causes that can present with acute pelvic pain include appendicitis, acute sigmoid diverticulitis and Crohn's disease. In your assessment it is important to exclude those diagnoses that require urgent intervention: PID, ovarian torsion, ectopic pregnancy and appendicitis.

The history should include the onset site and the nature of the pain, the date of the last menstrual period and the presence of associated symptoms. On examination, identify the site of maximal tenderness and the presence of rebound tenderness and guarding. It is vital to always exclude pregnancy, particularly ectopic pregnancy.

Ovarian torsion usually occurs in the presence of an enlarged ovary. Women with torsion present with sudden onset of sharp, unilateral pelvic pain that is often accompanied by nausea and vomiting. The sonographic findings are variable. The ovary is enlarged and can be seen in an abnormal location above or behind the uterus. The absence of blood flow is an important sign, and a lack of venous wave form on Doppler ultrasound has a high positive predictive value.

Endometriotic patches on the surface of the ovary.



(From Symonds EM, Symonds IM. *Essential obstetrics and gynaecology*, 4th edn. Edinburgh: Churchill Livingstone, 2004.)

FIGURE 40.1

However, the presence of arterial and venous flow does not exclude torsion and any cases where it is suspected clinically require laparoscopy to visualise the adnexa (ovaries and fallopian tubes). If the torsion is reversed early in the process, the ovary may be saved.

Approximately 1% of pregnancies are ectopic (occur outside the uterus) but the individual risk depends on the past history (see Table 40.2). Ectopic pregnancy presents classically with sudden onset of unilateral pain and irregular vaginal bleeding after a period of amenorrhoea. The presence of shoulder tip pain (due to diaphragmatic irritation) and peritonism are suggestive of rupture with intraperitoneal bleeding. In practice, it is rare for all three symptoms to be present and chronic presentation with atypical pain and bleeding are more common. Clinical examination findings include cervical excitation (increased pain on movement of the cervix during pelvic examination), adnexal pain and swelling with or without signs of hypovolaemic shock. The diagnosis is usually made by measurement of human chorionic gonadotrophin (hCG) levels and pelvic ultrasound and confirmed at laparoscopy (see Table 40.1).

Risk factors for ectopic pregnancy

	Relative risk
Previous history of PID	4
Previous tubal surgery	4.5
Failed sterilisation	9
Intrauterine contraceptive device in situ	10
Previous ectopic pregnancy	10–15

PID = pelvic inflammatory disease.

TABLE 40.2

Causes of infertility

Cause	Primary infertility (%)	Secondary infertility (%)
Anovulation	32	23
Tubal disease	12	14
Endometriosis	11	10
Sperm quality problems	29	24
Other	14	21
Unknown	29	30

(Adapted from Bhattacharya S, Porter M, Amalraj E et al. The epidemiology of infertility in the North East of Scotland. *Hum Reprod* 2009; 24(12): 3096–3107.)

TABLE 40.3

Infertility

Approximately 80% of normally fertile couples will conceive within a year of unprotected intercourse; when conception does not occur within 12 months the couple can be considered at risk of infertility. The median prevalence of infertility in the developed world at 12 months of unprotected intercourse is 9%, although this varies significantly in relation to age in women. Infertility is called *primary* if the woman has not previously been pregnant and *secondary* if she has had one or more previous pregnancies. Table 40.3 summarises the common causes of infertility (note that it is not uncommon for more than one factor to affect fertility in a given couple). The initial consultation should include both partners. Key questions to ask are given in Questions box 40.1.

INITIAL ASSESSMENT OF THE INFERTILE COUPLE

1. How long have you been trying to fall pregnant?
2. What methods of contraception have you or your partner used previously?
3. Have either you or your partner had a previous pregnancy together or with other partners?
4. If so, did you have any complications associated with the pregnancy?
5. Can you tell me about your periods and whether you have had any bleeding between your periods?
6. How often do you and your partner have sex? Are there any problems with pain during sex or ejaculatory/erection problems for your partner?
7. Have you ever had any sexually transmitted infections or has your partner ever had mumps orchitis (inflammation of the testes)?
8. Do you have any serious chronic illness or have you ever had surgery such as an appendectomy? Has your partner been treated for varicocele or undescended testes?

QUESTIONS BOX 40.1

Diagnosis

Clinical examination findings of significance for women include those features potentially associated with causes of amenorrhoea (see above) and endometriosis. Male examination findings include testicular size, the presence of varicocele and thickening of the epididymis (see Ch 18). Initial investigations are determined by the history; for example, if this is suggestive of previous pelvic infection, an assessment of tubal patency would be a priority. Typically, initial baseline tests include semen fluid analysis, assessment of ovulation (such as a day 21 serum progesterone level) and assessment of tubal patency (such as hysterosalpingogram or laparoscopy).

Sexual history

The amount of detail required will depend on the presenting symptom. In women complaining of disorders of sexual function and suspected sexually transmitted infection (such as vaginal discharge, PID), a full sexual history should be obtained, as described in Questions box 40.2.

Always explain the reason you need to ask questions of such a personal nature. For example, 'From what you have told me I think some of these problems may have a sexual cause. Will it be all right if I ask you some personal questions?'

Previous gynaecological history

A detailed history of any previous gynaecological problems and treatments must be recorded. The amount of detail needed about previous pregnancies will depend on the presenting symptom. In most cases the number of previous pregnancies and their outcome (miscarriage, ectopic or delivery after 20 weeks) is all that is required.

For all women of reproductive age who are sexually active it is essential to ask about contraception. This is important not only to determine the possibility of pregnancy but also because the method of contraception used may itself be relevant to the presenting symptom. For women over the age of 25, ask about the date and result of the last cervical screening test.

Previous medical history

Take particular account of any history of medical disorders of the endocrine, urinary or cardiovascular systems. Make a record of all current medications and any known drug allergies.

Family and social history

A social history is important for all problems but is particularly relevant where the presenting difficulties relate to abortion or sterilisation. Ask about smoking, alcohol and other recreational drug use. A family history of breast or ovarian cancer or both, delayed puberty or premature menopause may be relevant where the patient has the same condition.

QUESTIONS TO ASK A PATIENT COMPLAINING OF SEXUAL DYSFUNCTION OR SUSPECTED SEXUALLY TRANSMITTED INFECTION[2]

1. What is the reason for your attendance today?
2. How long have you had these symptoms?
3. Do you suffer from any of the following?
   - Urethral and vaginal discharge
   - Abnormal vaginal or rectal bleeding
   - Genital and extragenital rashes, lumps or sores
   - Itching and/or discomfort in the perineum, perianal and pubic regions
   - Lower abdominal pain or dyspareunia
   - Difficulties/pain with micturition or defecation or during intercourse
4. When was the last time you had sexual intercourse?
5. Have you had unprotected intercourse?
6. How many sexual contacts have you had in the last 3–5 months? What were the gender(s) of your sexual partners?
7. What type of sexual activity did you practise? (Oral, anal, vaginal, use of toys)
8. Did you take any steps to prevent sexually transmitted infections, such as using condoms? If so, do you use this method consistently?
9. What is your relationship with your sexual contacts (regular, casual, known, unknown)?
10. Have any of your recent sexual contacts had any symptoms of sexually transmitted infection or infections?
11. Have you previously been tested for any sexually transmitted infections? If so, what was the date of the test, and the result?
12. Do you have a current or past history of injecting drug use? Sharing of needles or syringes? Body piercing and/or tattoos? If the last-named, where and when were these done and was sterile equipment used?
13. Have you had sex overseas with anyone other than with the person you were travelling with?
14. Have you ever worked in the sex industry or had sexual contact with a sex worker?
15. Have you been vaccinated for hepatitis A and B and HPV?
16. Are you taking any current medications?
17. Do you have a history of allergies, especially adverse reaction to penicillin?
18. What contraceptives do you use?
19. When was your last menstrual period?
20. When was your last cervical screening test? What was the result? Have you ever had an abnormal result?
21. Do you have any past medical and surgical history (including any overseas medical treatment and transfusions)?
22. What is your current alcohol, tobacco and other drug use?
23. Have you ever had any concerns about your gender identity? Are you happy to tell me about these and how they have affected you?

(Adapted from NSW Sexually Transmissible Infections Programs Unit 2011. www.stipu.nsw.gov.au.)

QUESTIONS BOX 40.2

EXAMINATION

A general examination should always be performed at the first consultation, including assessment of pulse, blood pressure and temperature. Take careful note of any signs of anaemia. The distribution of facial and body hair is often important, as hirsutism may be a presenting symptom of androgen-producing tumours or PCOS. Also record the patient's body weight and height.

The intimate nature of the gynaecological examination makes it especially important to ensure that every effort is made to consider privacy and that the examination is not interrupted (see Text box 40.2). The examination should ideally take place in a separate area to the consultation. Allow the patient to undress in privacy and, if necessary, empty her bladder first. After she has undressed there should be no undue delay to the examination.

Examination of the abdomen

Inspection of the abdomen is undertaken as previously described in Chapters 14 and 18. In a patient presenting with gynaecological symptoms look for the presence of a mass as well as scars, striae and hernias. If there is a mass, try to determine whether it is fixed or mobile, smooth or regular, and whether it arises from the pelvis (you should not be able to palpate the lower edge above the pubic bone). Look for scars in the umbilicus from previous laparoscopies and in the suprapubic region where transverse incisions from caesarean sections and most gynaecological operations are found. Palpate the abdomen to assess for any guarding or rebound tenderness. Check the hernial orifices and feel for any enlarged lymph nodes in the groin. Percuss the abdomen to outline the limits of a tumour, detect the presence of a full bladder or recognise the presence of tympanic loops of bowel.

Pelvic examination

The pelvic examination should be performed *when indicated* as the final part of any complete physical examination.[4] It should not be considered an automatic and inevitable part of every gynaecological consultation. You should consider what information will be gained by the examination, whether this is a screening or diagnostic procedure and whether it is necessary at this time. In a child or in a woman with an intact hymen, speculum and pelvic examinations are not usually performed unless as part of an examination under anaesthesia. Remember that a rough or painful examination rarely produces any useful information and, in certain situations such as tubal ectopic pregnancy, may be dangerous. Throughout the examination remain alert to verbal and non-verbal indications of distress from the patient.

Guidelines for intimate examination[3]

When conducting intimate examinations you should:

- Explain to the patient why an intimate examination is necessary and give the patient an opportunity to ask questions.
- Explain what the examination will involve, in a way the patient can understand, so that the patient has a clear idea of what to expect, including any potential pain or discomfort.
- Obtain the patient's permission before the examination and be prepared to discontinue the examination if the patient asks you to.
- Record that permission has been obtained.
- Keep discussion relevant and avoid unnecessary personal comments.
- Offer a chaperone. If the patient does not want a chaperone, you should record that the offer was made and declined. If a chaperone is present, you should record that fact and make a note of the chaperone's identity. If for justifiable practical reasons you cannot offer a chaperone, you should explain to the patient and, if possible, offer to delay the examination to a later date.
- Give the patient privacy to undress and dress and use drapes to maintain the patient's dignity. Do not assist the patient in removing clothing unless you have clarified with them that your assistance is required.
- Obtain consent prior to anaesthetisation, usually in writing, for the intimate examination of anaesthetised patients. If you are supervising students you should ensure that valid consent has been obtained before they carry out any intimate examination.

(Adapted from General Medical Council (2013). Intimate examinations and chaperones. Available at https://www.gmc-uk.org/ethical-guidance/ethical-guidance-for-doctors/intimate-examinations-and-chaperones/intimate-examinations-and-chaperones.)

TEXT BOX 40.2

RULES FOR CONDUCTING A PELVIC EXAMINATION

When conducting a vaginal examination you should:

- Explain that an intimate examination is needed and why.
- Explain what the examination will involve.
- Obtain the patient's permission.
- Offer a chaperone or invite the patient to bring a relative or friend.
- Allow the patient to undress in privacy.
- Keep discussion relevant and avoid unnecessary personal comments.
- Explain findings and encourage questions and discussion.

LIST 40.4

It is essential to obtain informed consent and for male students and doctors to offer to have a female chaperone (see List 40.4). The patient's privacy must be promised and ensured.

Wear gloves on both hands during vaginal and speculum examinations. Examine the patient either in the supine position or in the left lateral position with the knees drawn up and separated (see Fig. 40.2). The left lateral position is used when the woman cannot assume the lithotomy position or when a view of the anterior vaginal wall is required—for example, when a urinary fistula is suspected (see below). The perineum should be brightly illuminated by a lamp.

Examination of the external genitalia

Parting the lips of the labia minora with the left hand, look at the external urethral meatus and inspect the vulva for any discharge, redness, ulceration or old scars or vaginal prolapse (see Table 40.4).

Bartholin's glands[a]

The Bartholin's glands lie in the posterior vaginal wall at the introitus (entrance) and secrete mucus-like fluid via a short duct into the vagina. They are normally the size of a pea but when the duct becomes blocked a cyst can form. These cysts may present acutely as an oval-shaped lump—in the posterior labia they may sometimes grow to the size of a golf ball or larger. They are usually unilateral and cause discomfort with walking, sitting and sexual intercourse. When the gland is infected, most commonly with skin or genitourinary bacteria (*Staphylococcus*, *Escherichia coli*), an abscess can develop. These abscesses arise more acutely than Bartholin's cysts and are particularly painful.

Speculum examination

Speculum examination should be performed before digital examination to avoid any contamination with lubricant. A bivalve speculum is most commonly used with the patient in the supine position as this enables a clear view of the cervix to be obtained. Where vaginal wall prolapse is suspected, a Sims' speculum should be used as an alternative with the patient in the left lateral position as this affords a better view of the anterior vaginal wall (see Fig. 40.3).

Part the labia minora with the left hand and insert the speculum into the introitus, initially with the widest dimension of the instrument in the anteroposterior orientation and turning gently to the transverse position as the tip passes the introitus, as the vagina is widest in this direction. When the speculum reaches the top of the vagina, gently open the blades to enable the cervix to be visualised. Note the presence of any discharge or bleeding from the cervix and any polyps or areas of ulceration. If the clinical history suggests possible infection (see Table 40.5), take swabs from the vaginal fornices and cervical os and place in a transport medium to look for *Candida*, *Trichomonas* and *Neisseria*; take a separate swab from the endocervix to look for *Chlamydia*.

Appearance of the vaginal wall

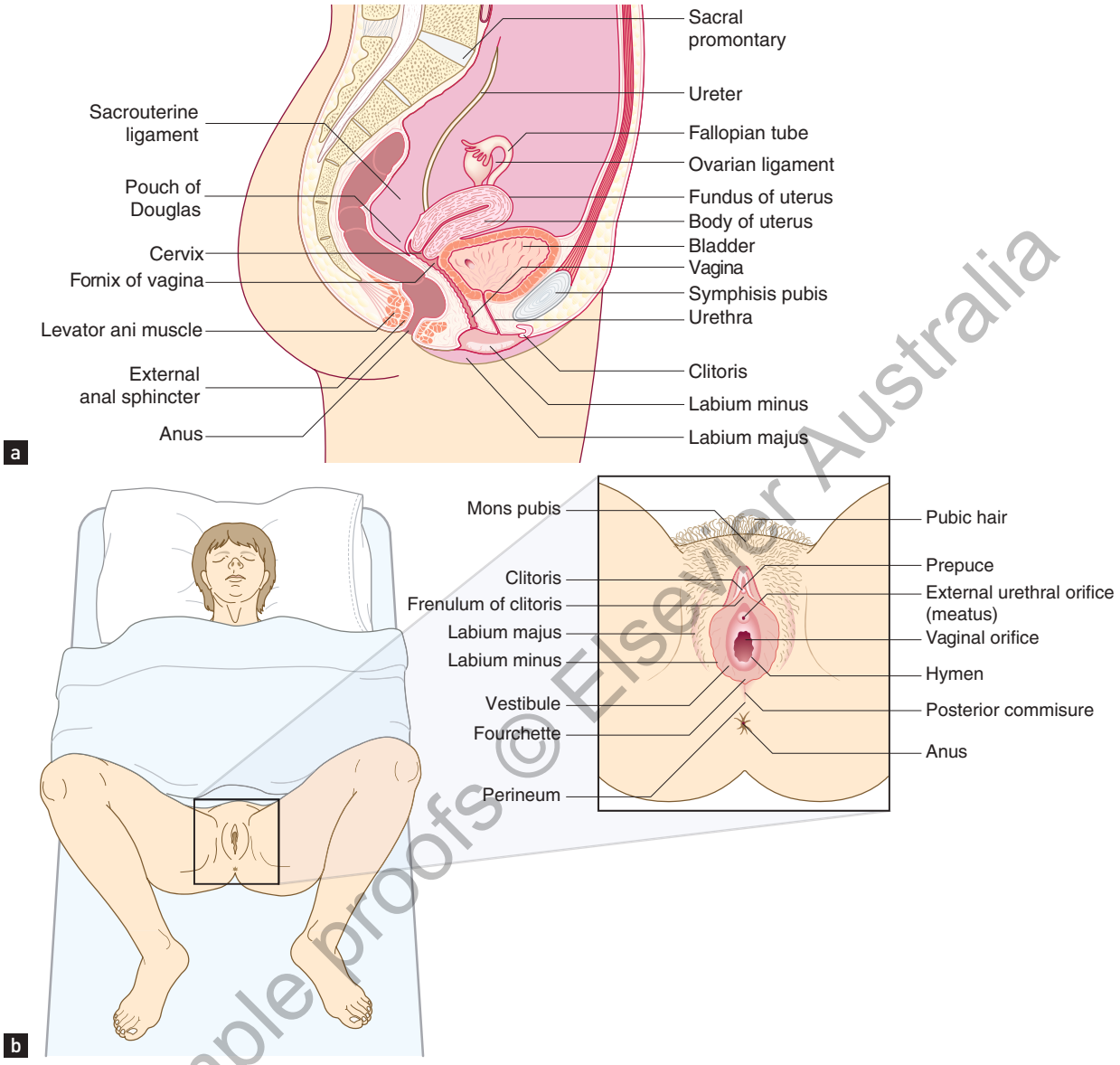
Ask the patient to bear down; a cystocele (descent of the bladder through the anterior vaginal wall) or rectocele (descent of the rectum through the posterior vaginal wall) or uterine prolapse may become apparent. Then ask the patient to cough; this may demonstrate stress incontinence. Note the presence of vaginal atrophy in older women.

Prolapse

Uterovaginal prolapse is the protrusion of the uterus and/or the vaginal walls beyond their normal anatomical confines. Prolapse commonly occurs in women as a result of damage to the supporting

[a] Caspar Bartholin Secundus (1655–1738), a professor of philosophy at Copenhagen at the age of 19, then professor of medicine, anatomy and physics. He described the glands in 1677.

Female reproductive anatomy.



(a) Lateral view, showing the relationship of the genitals to the rectum and bladder.
(b) Position for examination

(From Douglas G, Nicol F, Robertson C. *Macleod's clinical examination*, 12th edn. Edinburgh: Churchill Livingstone, 2009.)

FIGURE 40.2

structures of the vagina and uterus following childbirth, although it may occur in women even after delivery by caesarean section. It is not unusual for minor degrees of prolapse to be asymptomatic and detected only as an incidental finding on vaginal examination. Where symptoms do occur these are commonly a feeling of fullness in the vagina or being able to feel a swelling protruding beyond the vaginal introitus.

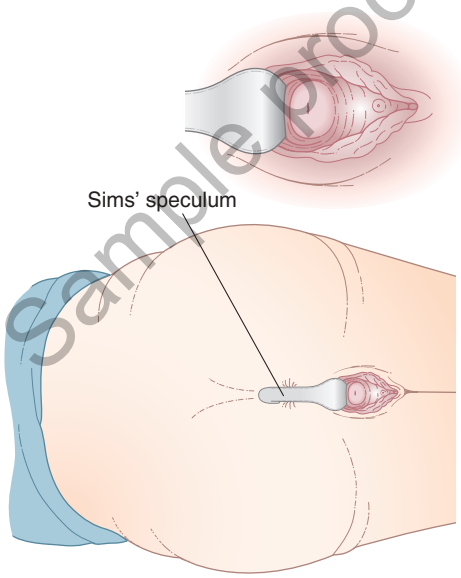
The diagnosis of prolapse is a clinical one based on the appearances at the time of examination (see Fig. 40.6). Swelling of the anterior vaginal wall is described as a **cystocele**. Where swelling of the lower posterior

Vulval skin conditions

Presentation	Condition
Itchy, erythematous rash (endogenous) Atopic/seborrhoeic Allergic or irritant induced (exogenous)	Dermatitis
Chronic irritation, results in thickening and hypertrophy of skin, erythema, excoriations Mucosa not involved	Lichen simplex chronicus
Itch, discomfort, white discharge, dyspareunia, dysuria Hypersensitivity reaction on vulva Requires positive culture to confirm diagnosis	Candidiasis
Whitened parchment-like plaques Classic hourglass appearance involving perianal skin Loss of labial/clitoral architecture, introital narrowing May have tearing or subepithelial haemorrhage/petechiae Mucosa not involved	Lichen sclerosus (see Fig. 40.4)
Lichen simplex chronicus difficult to differentiate from lichen planus Introitus of vagina involved Adhesions and erosions may occur; not responsive to surgical division Oral/gingival involvement possible	Lichen planus
Itchy, scaly red plaques not as well demarcated as elsewhere on the skin; examine hair/scalp and nails also	Psoriasis
Itching Different patterns may be associated with red velvety area or area of lichenification	Vulvar intraepithelial neoplasia
Itch, discharge, bleeding may be painful Associated lymphadenopathy Raised ulcerated lesion	Vulvar cancer (see Fig. 40.5)

TABLE 40.4

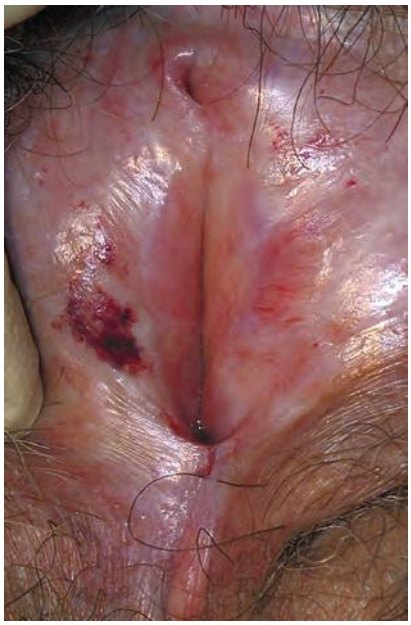
Examination in the lateral semiprone positions with a Sims' speculum.



(From Symonds EM, Symonds IM. *Essential obstetrics and gynaecology*, 4th edn. Churchill Livingstone, 2004.)

FIGURE 40.3

Lichen sclerosus.



(Courtesy of Ruth Murphy, PhD, MBChB, FRCP, Nottingham, UK. In Murphy R. Lichen sclerosus. *Dermatol Clin* 2010; 28(4):707–715.)

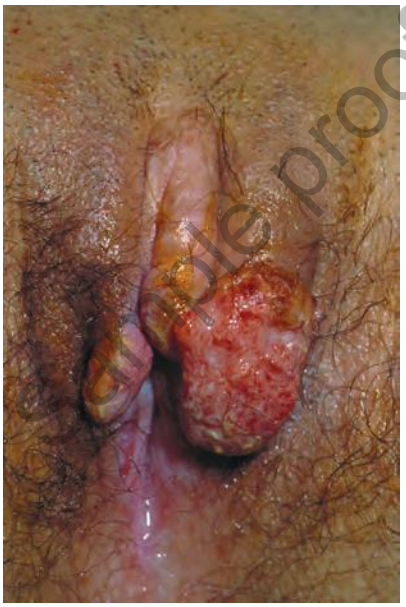
FIGURE 40.4

Common organisms causing lower genital tract infections

Condition	Causative organism	Symptoms	Signs
Bacterial vaginosis	Anaerobic organisms including *Gardnerella* spp	Smelly vaginal discharge Vulval irritation	A typical thin homogeneous vaginal discharge
Chlamydial vulvovaginitis	*Chlamydia trachomatis*	Asymptomatic or vaginal discharge Intermenstrual bleeding	Inflamed cervix Contact bleeding Tenderness on cervical movement
Genital herpes	Herpes simplex virus (HSV) type 2	Vaginal discharge Vulval pain Dysuria Urinary retention	Skin vesicles and multiple shallow skin ulcers Inguinal lymphadenopathy
Genital warts (condyloma acuminata)	Human papillomavirus	Pruritus Vaginal discharge	Papillomatous lesions over vulva, perineum and into vagina
Gonococcal vulvovaginitis	*Neisseria gonorrhoeae*	Vaginal discharge	Mucopurulent vaginal discharge
Trichomoniasis	*Trichomonas vaginalis*	Vaginal bleeding Vaginal soreness Pruritus	Green frothy, watery discharge
Vaginal candidiasis	*Candida albicans*	Increased or changed vaginal discharge associated with soreness and itching in the vulva area	White, curd-like collections attached to the vaginal epithelium

TABLE 40.5

Carcinoma of the vulva.



(From Arjona JE. Pregnancy following radical vulvectomy for carcinoma of the vulva: a case report and literature review. *Eur J Obstet Gynecol Reprod Biol* 2011; 158(1):113–114.)

FIGURE 40.5

wall is present this is described as a **rectocele**, and swelling of the upper posterior vaginal wall or posterior fornix is an **enterocele**. Uterine prolapse is described in terms of movement of the cervix in relation to the level of the vaginal introitus. Complete prolapse of the uterus beyond the introitus is described as **procidentia** (see Fig. 40.7).

Cystic swellings in the vagina and vulva

Congenital cysts arise in the vagina from embryological remnants. The most common varieties are those arising from Gartner's duct (Wolffian duct remnants). They occur in the anterolateral wall of the vagina. They are usually asymptomatic and are found on routine examination.

Vaginal inclusion cysts arise from the inclusion of small particles or islands of vaginal epithelium under the surface. The cysts commonly arise in episiotomy scars and contain thick yellowish fluid.

Solid benign tumours of the vagina are rare but may represent any of the tissues found in the vagina. Thus, polypoid tumours may include fibromyomas, myomas, fibromas, papillomas and adenomyomas.

Appearance of the cervix

'Normal' cervical squamous epithelium has a smooth pink appearance with a circular external os placed centrally. In practice, in women of reproductive age the external os is more irregular and slit-like and there is often a central area extending beyond the edge of the os that is a darker red and more velvety in appearance (see Fig. 40.8). This is sometimes called a cervical ectropion and represents an outgrowth of healthy columnar epithelium beyond the cervical canal. This is a normal variant, although it may be associated with an increase in clear vaginal discharge and, on occasion, contact bleeding. The columnar epithelium appears reddened because, unlike the stratified squamous epithelium of the ectocervix, there is only a single layer of columnar cells between the underlying capillaries and the surface. As the columnar epithelium is exposed to the acid pH of the vagina it tends to revert back to squamous epithelium in a process called squamous metaplasia. This sometimes traps islands of the mucus-secreting columnar epithelium below the surface of the new epithelium, leading to an accumulation of mucus in small retention cysts or Nabothian follicles.

In women with **cervicitis**, the cervix appears reddened and may be ulcerated, and there is a mucopurulent discharge as the endocervix is invariably involved. The diagnosis is established by examination and taking cervical swabs for culture.

Cervical intraepithelial neoplasia is not normally visible without the application of acetic acid or Lugol's iodine. The appearance of a raised or ulcerated lesion with irregular vessels on the surface is suggestive of cervical malignancy. Cervical polyps are common and may arise from the cervical canal or the external surface. Occasionally, an endometrial polyp may prolapse through the cervical canal and appear at the cervical os.

Cervical screening test (previously called a Pap smear)

There are two principle methods of screening for cervical neoplasia. The first is the routine cervical screening test for the detection of high-risk human papilloma virus (HPV) serotypes. The cytological examination of exfoliated cells from the squamous epithelium (previously called a Pap smear) is now performed only if the screening test is HPV positive or the patient is symptomatic. Both methods require collection of a sample from the cervix by speculum examination. The age at which routine screening starts and the frequency of testing vary from country to country. In Australia, testing commences at the age of 25, with the primary test being for the detection of HPV. A Pap smear, if indicated by an HPV-positive result, should be taken at least 6 weeks after pregnancy and not during menstruation.

The procedure is as follows. After inserting the speculum as above, wipe away any discharge from the surface of the cervix and take a 360° sweep with a suitable spatula or brush pressed firmly against the cervix at the junction of the columnar epithelium of the endocervical canal and the squamous epithelium of the ectocervix.

A liquid-based cytology test (LBC) is then performed. The sampling device is transferred into the preservative solution vial by pushing the broom into the bottom of the vial 10 times, forcing the bristles apart. In the laboratory the solution is then passed through a filter, which traps the large squamous cells but allows smaller red cells, debris and bacteria to pass through. The squamous cells are then transferred to a slide.

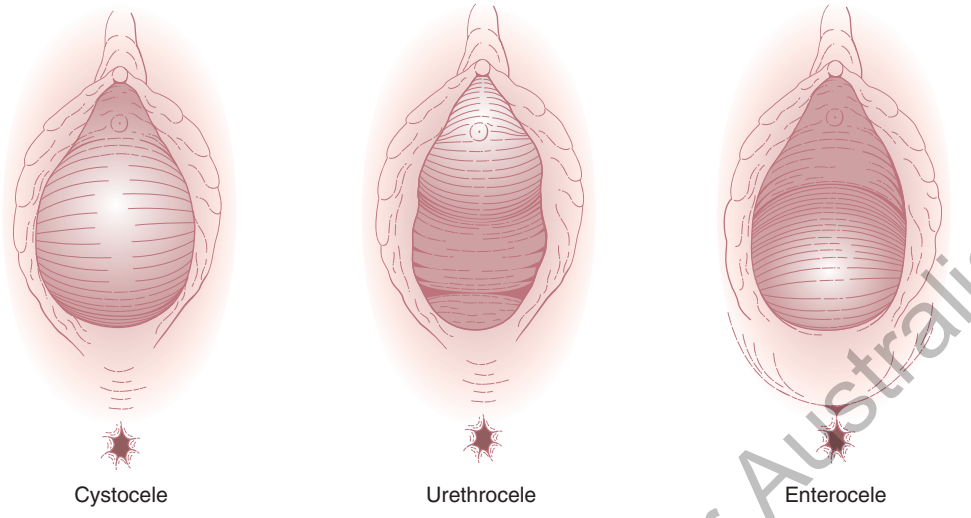
LBC also allows for testing for human papillomavirus and *Chlamydia* infection.

Vaginal swabs

The indications for taking vaginal swabs are symptoms of vaginal discharge (see Table 40.6), irregular bleeding and PID. Swabs may also be taken to screen for sexually transmitted infection in asymptomatic women. Cervicitis is associated with purulent vaginal discharge, sacral backache, lower abdominal pain, dyspareunia and dysuria, although in many cases the symptoms are minimal. The proximity of the cervix to the bladder often results in coexistent trigonitis and urethritis, particularly in the case of gonococcal infections.

A high vaginal swab is taken as part of the speculum examination by dipping the tip of a culture swab moistened in culture medium in the posterior vaginal fornix and then placing the swab immediately back into a suitable culture medium. This procedure is used mainly to identify organisms such as *Candida* or *Trichomonas* and in the assessment of bacterial vaginosis.

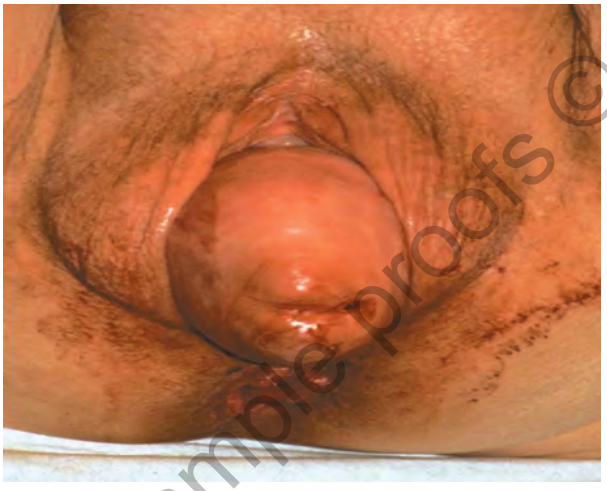
Clinical appearance of vaginal prolapse.



(From Symonds EM, Symonds IM. *Essential obstetrics and gynaecology*, 4th edn. Edinburgh: Churchill Livingstone, 2004.)

FIGURE 40.6

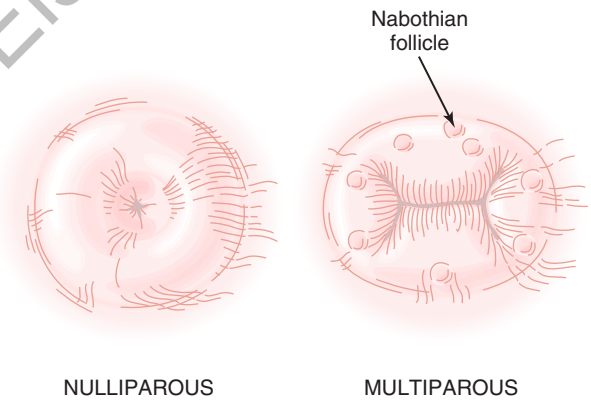
Procidentia (third-degree uterine prolapse).



(From Lentz GM, Lobo RA, Gershenson DM. *Comprehensive gynecology*, 6th edn. Philadelphia: Elsevier Mosby, 2012, pp 453–474.)

FIGURE 40.7

Normal multiparous cervix.



(From Hacker NF, Gambone JC, Hobel CJ. *Hacker and Moore's essentials of obstetrics and gynecology*, 5th edn. Philadelphia: Saunders, 2010.)

FIGURE 40.8

Endocervical swabs are taken by inserting the tip of the swab into the external cervical os and rotating two or three times. Using standard culture medium as for the high vaginal swab, the swab can be tested for *Neisseria gonorrhoeae*. Testing for *Chlamydia* infection can also be done by swabbing the endocervix. However, instead of culturing the organism, the presence of *Chlamydia* DNA is tested for using polymerase chain reaction by placing the swab in a specialised collection fluid in a plastic vial. The same principle can be used to test a first-pass urine sample to diagnose *Chlamydia*, so an endocervical swab is no longer necessary to test for this infection.

Bimanual examination

This is performed by introducing the middle and index fingers of your examining hand into the vaginal introitus and applying pressure towards the rectum. At the same time, place your other hand on the patient's abdomen above the symphysis pubis (see Fig. 40.9). The intravaginal portion of the cervix is tipped by your examining fingers (it can be identified by its consistency, which has a similar texture to the cartilage of the tip of the nose). Note the size, shape, consistency and position of the uterus. The uterus is commonly preaxial or anteverted, but will be postaxial or retroverted in some 10% of women. Provided that the retroverted uterus is mobile, the position is rarely significant. It is important to feel in the pouch of Douglas for the presence of thickening or nodules (a feature of endometriosis), and then to palpate laterally in both fornices for the presence of any ovarian or tubal masses (see Fig. 40.10). An attempt should be made to differentiate between adnexal and uterine masses, although often this is not possible. For example, a pedunculated fibroid may mimic an ovarian tumour, whereas a solid ovarian tumour, if adherent to the uterus, may be impossible to distinguish from a uterine fibroid. The ovaries may be palpable in the normal pelvis if the patient is thin, but the fallopian tubes are palpable only if they are significantly enlarged.

Diagnosis of vaginal discharge

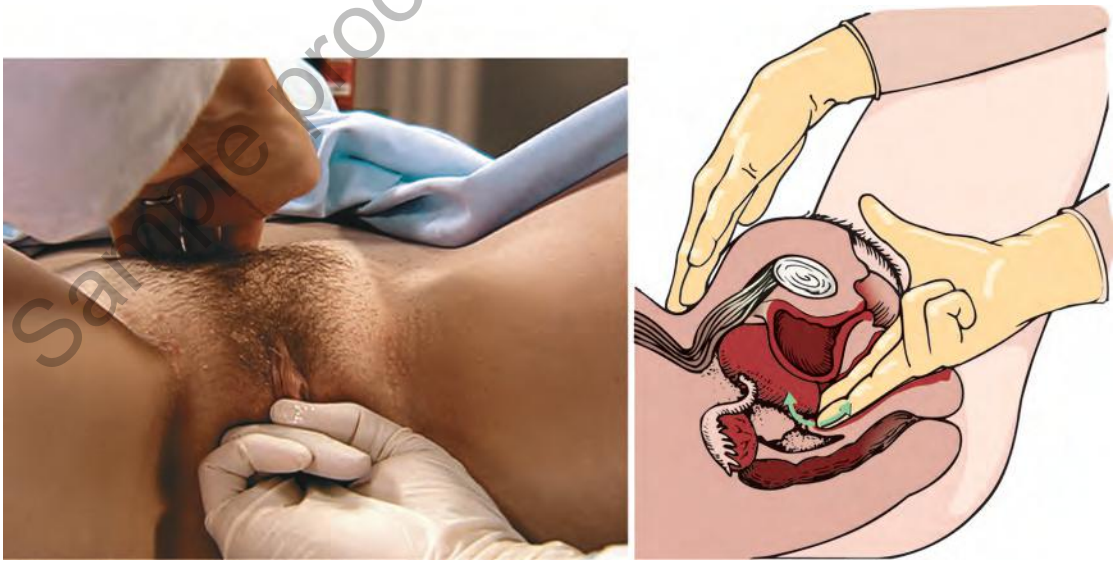
Features of discharge and associated symptoms	Possible causes
Thick, white, non-itchy	Physiological
Bloody	Menstruation, a miscarriage, cancer or a cervical polyp or erosion
Thick, white, cottage cheese discharge, vulval itching, vulval soreness and irritation, pain or discomfort	*Candida albicans*
Yellow-green, itchy, frothy, foul-smelling ('fishy' smell) discharge	*Trichomonas*
Thin, grey or green, discharge with a fishy odour	Bacterial vaginosis
Thick white discharge, dysuria and pelvic pain, friable cervix	Gonorrhoea

TABLE 40.6

Pelvic mass

Table 40.7 lists the common causes of a mass arising from the pelvis in women. The uterus is palpable above the symphysis pubis on abdominal palpation in

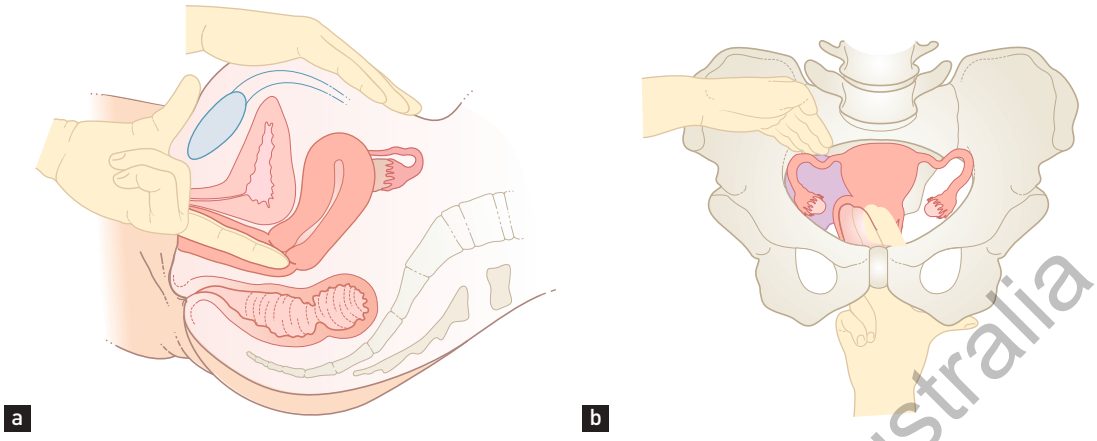
Bimanual examination.



(From Seidel HM, Ball JW, Dains JE et al. *Mosby's guide to physical examination*, 7th edn. St Louis, MO: Mosby, 2011.)

FIGURE 40.9

Bimanual examination of (a) the pelvis and (b) the lateral fornix.



(From Symonds EM, Symonds IM. *Essential obstetrics and gynaecology*, 4th edn. Edinburgh: Churchill Livingstone, 2004.)

FIGURE 40.10

pregnancy after 12 weeks' gestation and typically uterine masses are described in terms of the equivalent uterine enlargement in pregnancy. For example, a uterine mass extending up to the umbilicus would be described as '20 weeks' in size.

Many pelvic masses cause no symptoms, even when large in size. The presence of pain most commonly occurs only when there is bleeding within or from the lesion (for example, in the case of ovarian cysts) or when the lesion becomes infarcted (for example, as a result of torsion; see Fig. 40.11). If large enough, a mass may be associated with pressure symptoms on the adjacent organs, such as urinary frequency or changes in bowel habits.

The first step in evaluating a pelvic mass is an abdominal examination. Masses arising from the pelvis can be distinguished by failure to identify a gap between the mass and the symphysis pubis. The presence of scars from previous surgery or ascites should be noted.

On pelvic examination a pelvic space-occupying lesion may cause downward pressure leading to vaginal prolapse. A speculum examination is essential to exclude a primary cervical lesion such as advanced cervical cancer, although this will normally present earlier with bleeding. The mass can then be assessed by bimanual palpation. This allows a better estimate of the size and other clinical features—in particular, whether the mass is smooth or irregular, fixed or mobile, and whether it feels cystic or solid. Further assessment is normally undertaken using pelvic ultrasound.

Special circumstances

Keep in mind the following:

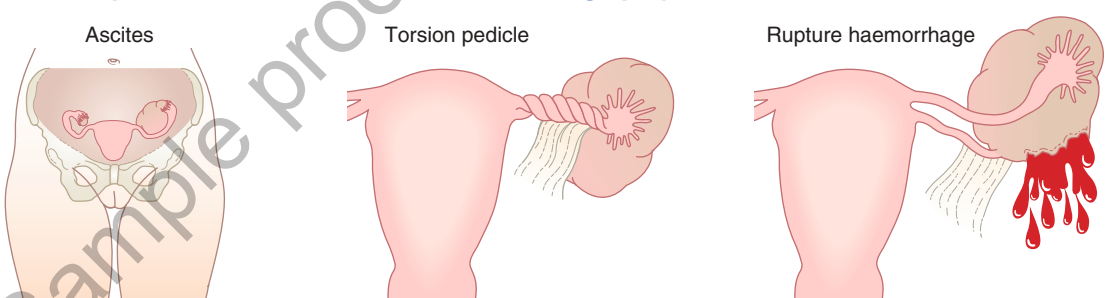
- Except in an emergency situation, a pelvic examination should not be carried out on a non-English-speaking patient without an interpreter. Be aware that examination may be more difficult for women with particular cultural or religious expectations.
- Women who experience difficulty with vaginal examination should be allowed to disclose any underlying sexual difficulties or traumas. However, do not assume that all women who experience difficulty with pelvic examinations have a history of sexual abuse.
- Exceptional gentleness should be displayed in the examination of victims of alleged sexual assault. The woman should be given a choice about the gender of the doctor and be allowed to control the pace of, and her position for, the examination.
- The basic principles of respect, privacy, explanation and consent that apply to the conduct of gynaecological examinations in general apply equally to the conduct of such examinations in women who have temporary or permanent learning disabilities or mental illness.
- When examining an anaesthetised patient, treat the woman with the same degree of sensitivity and respect as if she were awake.

Differential diagnosis of pelvic mass

Origin of mass	Clinical features
Uterine	Central Uterus cannot be felt separately If uterus mobile, moves with uterus
Leiomyomata	Smooth, solid, may be single or multiple giving an irregular outline to the uterus Normally non-tender
Pregnancy	Uniform enlargement, soft and fluid-filled (the presence of fetal heart sounds is pathognomonic!)
Adenomyosis	Smoothly enlarged (rarely more than 12 weeks' size), globular shape and tender to palpation
Cancer of the corpus	Uniform enlargement, usually solid If tender, sarcoma more likely
Cervical cancer	Associated with an irregular mass arising from the cervix and extending into the vagina Often necrotic, with contact bleeding
Ovary / fallopian tubes	More likely to be adnexal than central
Inflammatory (tubo-ovarian abscess)	Tender, cervical excitation, ill-defined Solid or cystic, associated with systemic illness / fever
Ovarian cysts	May be solid (dermoid or fibroma) or cystic (epithelial tumours) Normally unilateral, smooth regular outline, mobile Tenderness suggests bleeding or torsion
Ovarian malignancy	More likely to be cystic fixed and associated with ascites
Endometriosis	May be associated with adnexal mass (usually ovarian endometrioma) More often diffuse change in the pelvis with fixed non-mobile uterus, nodules and thickening in the posterior fornix and tenderness

TABLE 40.7

Common complications of ovarian tumours causing symptoms.



(From Symonds EM, Symonds IM. *Essential obstetrics and gynaecology*, 4th edn. Edinburgh: Churchill Livingstone, 2004.)

FIGURE 40.11

Rectal examination

Rectal examination may be indicated if there are symptoms such as a change in bowel habits or rectal bleeding, which may suggest bowel disease. It is occasionally used as a means of assessing a pelvic mass and in conjunction with a vaginal examination can provide additional information about disease in the rectovaginal septum.

OSCE REVISION TOPICS – THE GYNAECOLOGICAL HISTORY AND EXAMINATION

Use these topics, which commonly occur in the OSCE, to help with revision.

1. This woman has a history of heavy periods. Please take a full history from her and explain what further investigations you would like to perform. (pp 779–80)
2. This couple have been unable to conceive for 2 years. Please take a history and describe what features you would look for on examination that would be relevant to establishing a diagnosis. (pp 783–4, 785, 792–3)
3. This young woman presents with vaginal discharge 3 weeks after having unprotected intercourse with a new partner. Please take a full sexual history from her. (p 818)
4. Demonstrate how you would perform a pelvic examination and take a cervical screening test using this manikin. (pp 821, 825, 827)
5. Explain how you would perform a pelvic examination for a woman with vaginal discharge. (pp 791–2)
6. This woman has a pelvic mass. Please examine her. (pp 793–4)

T&O'C ESSENTIALS

1. *All women complaining of heavy periods should have a pelvic examination, full blood count and Pap smear.*
2. *Worrying features of uterine bleeding requiring further investigation include bleeding after the menopause, bleeding between periods and any abnormality of pelvic examination.*
3. *Pregnancy should be excluded in any women of reproductive age presenting with acute abdominal pain, abnormal bleeding or secondary amenorrhoea.*
4. *Atypical presentations are common in ectopic pregnancy.*
5. *The presence of a darker red area around the external cervical os on speculum examination is usually associated with an extension of the columnar epithelium onto the ectocervix and is a normal finding (ectropion).*
6. *Asymptomatic vaginal prolapse is common in multiparous women and does not require treatment.*
7. *Ovarian tumours can remain asymptomatic unless complicated by torsion, haemorrhage or rupture.*

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3. General Medical Council Standards Committee. Maintaining boundaries: intimate examinations and chaperones, *GMC guidelines for intimate examination ethical practice*. London: GMC, 2013. www.gmc-uk.org/Maintaining_boundaries_Intimate_examinations_and_chaperones.pdf_58835231.pdf.
4. Deneke M, Wheeler L, Wagner G et al. An approach to relearning the pelvic examination. *J Fam Pract* 1982; 14:782–783. This study provides useful hints.