

See discussions, stats, and author profiles for this publication at: <https://www.researchgate.net/publication/334697259>

CLINICAL METHODS: PHYSICAL EXAMINATION AND CLINICAL HISTORY TAKING

Book · January 2016

CITATIONS

0

READS

133,509

1 author:



Abilo Tadesse

University of Gondar

47 PUBLICATIONS 451 CITATIONS

SEE PROFILE

CLINICAL METHODS

(Physical Examination and Clinical History Taking)

for Health Science Students

(Lecture Note)



Prepared by

Abilo Tadesse, MD

Preface

Since text books in “Physical examination and Clinical history taking” for health science students are not available as needed, this lecture note will alleviate the deficiency in ‘knowledge and skill’ transferred from text books of “Clinical Methods”.

The lecture note comprises almost all of the course contents of ‘Clinical Methods’ provided to health science students including Medical, Health Officer, Anesthesia and Physiotherapy students, so then it can be used as a main learning material to these category of students.

I am grateful to internal and external reviewers who offered invaluable comments during write up of the lecture note.

At last but not least, the quality of this lecture note will be kept up to date by continual comments made by users of this lecture note.

Abilo Tadesse, MD

Associate Professor of Medicine

University of Gondar Hospital

February, 2018

Acknowledgment

I would like to acknowledge my students, who were actively involved in the learning-teaching process during “Clinical Bridging Course”, who gave me a good opportunity to improve my skills in clinical history taking and physical examination.

Contents

	Page
Preface	01
Acknowledgment	02
Table of contents	03
Chapter One: Scheme of history taking and physical examination	04
Chapter Two: Vital signs in physical examination	15
Chapter Three: Physical appearance and HEENT	19
Chapter Four: Lymphoglandular system	33
Chapter Five: Respiratory system	45
Chapter Six: Cardiovascular system	68
Chapter Seven: Gastrointestinal system	97
Chapter Eight: Nervous system	125
Chapter Nine: Musculoskeletal system	187
Chapter Ten: Genitourinary system	213
Chapter eleven: Integumentary system	242
Medical case report	250
References	258

CHAPTER ONE

Scheme of history taking and physical examination

Learning Objective

At the end of this lesson, the student should be able to:

1. Elaborate the duties and responsibilities of health professionals towards health care
2. List the classification scheme of clinical history taking
3. Mention the components of history of present illness (HPI)

Physical examination and history taking are the essence of medical practice. The word ‘patient’ is derived from the latin, *patiens*, meaning sufferance or forbearance. The overall purpose of medical practice is to relieve suffering. In order to achieve this purpose, it is important to make a diagnosis, to know how to approach treatment and to design an appropriate scheme of management for each patient.

Two major steps of making the diagnosis

- a. To establish the clinical data by history taking and physical examination
- b. To interpret the clinical data

Questions asked by the doctor can be *direct* or *closed-ended questions* which are specific questions of the doctor’s interest forwarded to the patient, while *indirect* or *open-ended question* are invited questions for the patient to talk about his general complaints. The doctor should use open-ended questions to encourage the patient to give a full and free account of their illness. Encourage the patient to tell their own story without interruption, listen carefully and maintain eye contact. An interview that uses lots of direct questions is often ‘disease-centered’ whereas a ‘patient-centered’ interview will contain enough open-ended questions for the patient to talk through all their problems. The doctor needs to grasp the difference between disease frame work (what is the diagnosis?) and illness frame work (what are the patients’ experiences, ideas, expectations and feelings?).

During any medical consultation, the doctor should be concerned with all aspects of the patients’ health and not just the problem with which patients have presented.

Duties of doctors (British General Medical Council)

- . Make the care of your patient your first concern
- . Treat every patient politely and considerately
- . Respect patients’ dignity and privacy
- . Listen to patients and respect their views
- . Give patients information in a way they can understand
- . Respect the right of patients to be fully involved in all decisions about their care
- . Keep your professional knowledge and skills up-to-date
- . Recognize the limits of your professional competency
- . Be honest and trustworthy

- . Respect and protect confidential information
- . Make sure that your personal beliefs do not prejudice your patients care
- . Avoid abusing your position as a doctor
- . Act quickly to protect patients from risk if you have a good reason to believe that you may not be fit to practice
- . Work with colleagues in the ways that best serve patient's interest
- . Never discriminate unfairly against your patients or colleagues

The Revised Declaration of Geneva: A Modern-Day Physician's Pledge (October, 2017)

As a member of the medical profession

I will solemnly pledge to dictate my life to the service of humanity

The health and well-being of my patient will be my first consideration

I will respect the autonomy and dignity of my patient

I will mainatain the utmost respect for human life

I will not permit considerations of age, disease or disability, creed, ethnic origin, gender, nationality, political affiliation, race, sexual orientation, social standing, or any other factor to intervene between my duty and my patient

I will respect the secrets that are confided in me, even after the patient has died

I will practise my profession with conscience and dignity and in accordance with good medical practice

I will foster the honour and noble traditions of the medical profession

I will give to my teachers, colleagues, and students the respect and gratitude that is their due

I will share my medical knowledge for the benefit of the patient and the advancement of health care

I will attend to my own health, wellbeing, and abilities in order to provide care of the highest standard

I will not use my medical knowledge to violate human rights and civil liberties, even under threat

I make these promises solemnly, freely, and upon my honour

A. Scheme of clinical history taking

1. Identification of the patient

Full name, Age, Sex, Occupation, Address, Religion, name of admitted hospital, Hospital bed number, date of admission

Eg, E.M., 40-year-old male, married, Orthodox Christian, daily laborer from Metema, North Gondar Zone, admitted to University of Gondar hospital, medical ward D, bed number 24, on January 12, 2009 E.C

2. Previous admission

This is a list of previous hospitalization in the order they occurred

. Specify the date, name and location of the hospital, disease led to admission, treatment and outcome of illness

Egs,

1990 E.C, Gondar University Hospital, Gondar, Smear positive pulmonary tuberculosis, anti-tbc drugs, discharged improved

2003 E.C, Black Lion Hospital, Addis Ababa, HIV-associated cerebral toxoplasmosis, treated with ARV drugs and anti-toxoplasmosis drugs and discharged improved

. If the previous admission is related to the current illness, details could be mentioned in appropriate place in the history of present illness

. If details of previous admission required, it could be mentioned under past illness

3. Chief complaint

Chief complaint is symptom that prompts the patient to seek medical attention

It should be written in patient's words and not necessarily in medical terminology

The duration of illness should be clearly written

If there is more than one main complaint, it should be listed in the order of occurrence

Eg: Cough of 3 weeks and hemoptysis of 2 days duration

4. History of present illness (HPI)

This is part of the history where by detailed description of the chief complaint is indicated

Chronological order of HPI

. Date of onset

. Mode of onset, course and duration

. Character and location (especially applicable for pain)

. Exacerbations and remissions

- . Effect of treatment
- . Negative-positive statements
- . Changes in strength, weight and color

Date of onset

Start with ‘the patient was apparently healthy until....’ then write the complaint that led to medical attention

Mode of onset, course and duration

Mode of onset could be abrupt or gradual, and related precipitating cause could be mentioned if present

eg : “.... Sudden onset of back pain while lifting heavy object...”

The course of symptom (s) could be persistent, intermittent or relapsing, and increasing or decreasing in severity

Mention associated symptoms with chief complaint

eg. “...sudden onset of left sided pleuritic chest pain of one day duration associated with shortness of breath, blood streaked sputum and low grade fever...”

Character and location

This is especially applicable for the description for pain

List of clarification for a complaint of pain (SOCRATES)

- . Site/location: somatic pain is well localized while visceral pain is diffuse and ill-defined
- . Onset: speed of onset (sudden, gradual)
- . Character/type: dull/sharp, burning/tingling, boring/stabbing, etc...
- . Radiation: referred pain eg. shoulder pain in diaphragmatic problem
- . Associated symptoms eg. visual aura (zigzag lines) accompanying migraine
- . Timing: episodic or continuous. If episodic, duration and frequency of attacks; if continuous, any change in severity
- . Exacerbating and relieving factors: effects of specific postures or activities
- . Severity: variation in day/month eg. relating to menstrual cycle

eg. ‘... sudden-onset, gradually worsening, throbbing, right sided frontal headache worsened by daily routine activities and relieved by Cafergot/ Triptans ...’ favors migraine

Exacerbations and remissions

Usually mentioned while dealing with mode of onset, course and duration

Effect of treatment

Patients may have taken any medication prior to admission to the hospital.

- . Mention any history of therapy prior to admission to the hospital which might have alleviated, worsened or no effect on their illness.
- . If drugs taken, mention the name, dose and duration of therapy
- . If patients didn't name the drug(s), mention the colour, texture and shape of the drug (s)
- . Mention if there is history of taking local herbal medication

Positive – negative statements

Significant part of HPI where by supportive positive symptoms, risk factors and complications, and other symptoms which rule out other diseases are mentioned

Color, strength and weight

How was the patient admitted to the hospital? Supported by relatives, on a stretcher, or walking by him self

Mention any apparent reduction in strength and weight which provides clues to the general condition of the patient during admission.

5. Past illnesses

List any illnesses experienced in the past unrelated to the current illness including child hood illnesses

A brief mention of each disease with an approximate date, severity, duration, complication and sequelae is essential

6. Functional inquiry

Detailed account of symptoms referable to each system of the body

There is no need to repeat complaints already mentioned in the HPI

Functional inquiry should be recorded as follows:

HEENT (head, eye, ear, nose, throat)

Head: Headache, head injury

Eyes: Blurring of vision, pain in the eyes or orbit, eye itching, lacrimation, photophobia

Ears: Ear ache or pain, deafness, ear discharge, vertigo, tinnitus

Nose: Nose bleeds, discharge

Mouth and throat: Gum bleeding, dental hygiene, sore throat, sore tongue

Lymphoglandar system

Swellings in the neck, axillae and groin, breast lump, nipple discharge, goiter, testicular swelling or pain

Respiratory system

Cough, expectoration (sputum), hemoptysis, chest pain, shortness of breath, wheezing, stridor

Cardiovascular system

Dyspnea (exercise intolerance), palpitation, orthopnea (number of pillows required), paroxysmal nocturnal dyspnea, swelling of legs, chest pain (angina), syncope, intermittent claudication, fatigue

Gastrointestinal system

Loss of appetite, nausea, vomiting, dysphagia, odynophagia, heart burn, abdominal pain, bowel habit change (diarrhea, constipation), jaundice, tarry or clay-colored stool, hematemesis, hematochezia

Genitourinary system

Flank pain, suprapubic pain, urine color change (hematuria), oliguria, polyuria, frequency (in day-to-night ratio), dysuria, urgency, hesitancy, dribbling, incontinence, menstrual history (age at menarche/interval between periods/duration of flow/amount of flow), dysmenorrhea, menorrhagia, metrorrhagia, menometrorrhagia, dyspareunia, amenorrhea, urethral or vaginal discharge, post coital bleeding, menopause, post menopausal bleeding

Integumentary system

Skin, hair and nail: Skin rashes, ulcers, urticaria, or nail changes

Locomotor system

Joint or bone deformities, joint pain or swelling, limping, loss of function of limbs or joints

Central nervous system

Amnesia (cognitive impairment), speech disturbance, seizures, diplopia, dysarthria, vertigo, weakness of extremities, urinary incontinence or retention, fecal incontinence or stool impaction, disturbance in sensation (anesthesia, hyperesthesia), insomnia, nervous breakdown

7. Personal history

Early development: Place of birth, childhood development, health and activities

Education: School history, achievements and failures

Work record: Age begun, type of work, number of jobs (success or failure of each job), job hazards and industrial exposures

Environment: Living condition of the patient

Habits: Alcohol, tobacco smoking, illicit drug use, herbs

Marital status: Health of wife/husband, adjustment (marital harmony), number of children and their health

8. Family history

Father and mother: Age, health, (if dead, mention date, age and cause of death)

Siblings: List with ages, health (if dead, mention date, age, cause of death)

Family disease: Diabetes mellitus, hypertension, migraine, asthma and other allergic diseases, other hereditary diseases

B. Scheme of physical examination

Vital sign

Blood pressure... mmhg (right or left arm, lying or supine)

Pulse rate... per minute (min)

Respiratory rate... per minute (min)

Temperature... $^{\circ}\text{C}$ (Axillary, oral, core)

Weight... Kg

Height... Cm

BMI ... Kg/m²

General appearance

Acuteness of illness (acutely or chronically sick looking), physique, nutritional status, emotional state, color change

HEENT

Head: Shape, size, masses, depression and tenderness of skull; amount, color, texture and distribution of hair; scar and cleanliness of scalp

Ears: Tragus or mastoid tenderness, tophi, cerumen, light reflex, bulging, retraction and perforation of tympanic membrane

Eye: Visual aid (spectacles), periorbital edema, xanthelasma, ptosis, lid lag; conjunctival pallor, injection, hemorrhage and trachomatous changes; scleral colour (icterus), pterygium and granulation

Nose: Deformities, deviation and perforation of septum, polyps and unusual discharges

Mouth and throat: Breath odor; color, fissures and ulceration of lips; bleeding, ulceration, and lidline of gums; tooth caries, extractions, dentures; tongue color, coating, fissure, papillae atrophy; color, ulceration, tumour, monilial patches of buccal mucosa and soft palate; tonsillar inflammation and exudates; post-nasal drip

Lymphoglandular system

Lymph nodes: Site, size, consistency, tenderness, fixation, discrete or matted, regional or generalized enlargement

Thyroid gland: Size, consistency, nodularity, tenderness, bruit

Breasts: Lump, consistency, tenderness, fixation; skin retraction or ulceration, unusual nipple discharge

Respiratory system

Inspection: Cyanosis of lips and nails, clubbing of fingers, rate, depth and character of respiration, symmetry of shape and expansion, use of accessory muscles, retractions

Physical examination and Clinical history taking

Palpation: Tenderness, subcutaneous crepitation, position of trachea, degree of chest expansion (in cm with tape or hand grip), tactile fremitus

Percussion: percussion notes (resonance, hyper-resonance, dull, flat), diaphragmatic excursion

Auscultation: Character of breath sounds (vesicular, broncho-vesicular, bronchial, tracheal), crackles, wheezing, friction rub, vocal resonance

Cardiovascular system

Blood pressure: ... in mmhg, right or left arm, sitting or lying

Arteries: pulse rate, rhythm, volume, character, radio-femoral delay, hardening (cording) of arteries

	Carotid	Brachial	Radial	Femoral	Popliteal	Dorsalis pedis	Posterior tibialis
Right	+++	++	++	+++	+	++	+
Left	+++	++	++	+++	+	++	+

Veins: Internal Jugular vein (JVP) pressure measured from sternal angle at an inclination of 45° and detect ‘a’ and ‘v’ waves, kussmaul sign, hepato-jugular reflex

Precordium (Heart)

Inspection: Presence of precordial bulging, active or quiet precordium, location of apical impulse (interspace, distance from left midclavicular line)

Palpation: Point of maximal impulse and its character, parasternal heave, thrill, shock

Percussion: cardiac outline (not frequently performed)

Auscultation: 1st and 2nd heart sounds, 3rd and 4th heart sounds, other added heart sounds (gallop, ejection click, opening snap, pericardial ‘knock’), murmur, friction rub

Abdominal Examination

Inspection: Abdominal symmetry, shape (round, flat, scaphoid), movement with respiration, flank fullness, everted or inverted umbilicus, dilated vessels, scars, visible peristalsis, presence of hernia at hernia sites

Palpation: tenderness (superficial or deep, site), rebound tenderness, guarding and rigidity, enlarged liver (size in cm below right costal margin along right midclavicular line, consistency, surface, edge, tenderness), enlarged spleen (size in cm along splenic growth line below left costal margin, consistency, surface, edge, tenderness, splenic (medial) notch), abdominal mass (size, consistency, surface, edge, tenderness, fixation, mobility with respiration), and enlarged kidneys (size, consistency, surface, edge and tenderness by bimanual palpation)

Percussion: Total vertical liver span, liver and splenic dullness, shifting dullness, fluid thrill

Auscultation: Bowel sounds, bruit over the liver, friction rub over the liver and the spleen, renal bruit

PR (per digital rectal examination): Fissures, ulcers and hemorrhoid, and fistula in anal area; rectal ulcers or tumor, sphincter tone, enlarged prostate (size, consistency, surface, tenderness, obliteration of medial sulcus)

Genitourinary system

Costovertebral angle and suprapubic tenderness. In male, scrotum (edema, hydrocele, hernia etc...), testes (size, tumor, descent), vas deferens (nodules, tenderness), varicocele, urethral orifice (reddening, discharge, ulcer,

Physical examination and Clinical history taking

phimosis). In females, labia majora and minora (ulcers, nodules, tumors), bartholin's duct, urethral orifice (reddening, discharge), vaginal discharge, cystocele/rectocele, cervix (excitation tenderness, ulcers and erosions), uterus (size, position, consistency), adnexal mass and tenderness

Integumentary system

Skin: Texture, rashes, ulcers, urticaria, pigmentation. Hair: texture, baldness or alopecia. Nails: color, shape (clubbing, spooning, etc...), texture, splinter hemorrhages, capillary refill time

Musculoskeletal system (Locomotor system)

Muscle: muscle tenderness, spasm. Spine: deformity (kyphosis, scoliosis, kyphoscoliosis), gibbus, tenderness on percussion or pressure, limitation of movement. Joints: Swelling, tenderness, heat and redness, crepitus, deformity, limitation of movement on active and passive motions. Bones: deformity, fracture, tenderness, tumors

Nervous system

Mental status

Level of consciousness (GCS), orientation in time, place and person; memory (immediate, recent and remote), speech disturbance (dysphasia), judgement, mood changes

Cranial nerves (CN)

CN I: Olfaction (each nostril)

CN II: Visual acuity, visual fields, color perception

CN III, IV, and VI: Ocular movements; pupillary size and reaction to light (direct and consensual) and accommodation, diplopia, nystagmus

CN V: Sensation over the face, contraction of temporalis and masseter muscles, corneal reflex

CN VIII: Rinne's and Weber's test, Dix-Hallpike maneuver

CN IX and X: Position and symmetry of soft palate and uvula, gag reflex

CN XI: contraction of sternomastoid muscle on turning the head against resistance, contraction of trapezius muscle on shrugging the shoulders against resistance

CN XII: Tongue protrusion, movement from side to side, deviation, atrophy, fasciculation and tremor

Motor system

Position, bulk, fasciculation (spontaneous or provoked), strength (power), tone, reflex (deep and superficial)

Deep tendon reflexes

	Biceps	Triceps	supinators	Patellar	Ankle
Right	+++	++	++	+++	+
Left	+++	++	++	+++	+

Superficial reflexes

	Corneal	Abdomiaal	Cremasteric	Plantar
Right	++	++	++	↓
Left	++	++	++	↓

Sensory system

Superficial: Light touch, pain and temperature. Deep (proprioceptive): Vibration and position sense, Romberg's test

Cortical: Stereognosis, graphesthesia, two-point discrimination, double simultaneous stimulation

Cerebellar: Finger-to-nose test, heel-to-shin test, supination-to-pronation of forearm, rebound phenomenon, tandem walk

Meningeal irritation sign

Nuchal rigidity, Kernig's and Brudzinsky's sign, Jolt accentuation of headache

CHAPTER TWO

Vital signs in physical examination

Learning objective

At the end of this lesson, the student should be able to:

1. Mention components of vital sign
2. List normal value of vital signs
3. Describe patterns of fever
4. Describe the parameters of nutritional assessment

Vital signs

Components of vital sign

- . Temperature
- . Pulse rate
- . Respiratory rate
- . Blood pressure
- . Height
- . Weight
- . BMI (Body mass index)

Temperature

Normal body temperature is maintained, despite environmental variations, because the hypothalamic thermoregulatory center balances heat production in the muscle and liver, and heat dissipation from the skin and lungs

Fever is an elevation of body temperature that exceeds the normal daily variation and occurs in conjunction with an increase in the hypothalamic set point

Fever is mediated by endogenous pyrogen, which acts on hypothalamic thermoregulatory center

Mean oral temperature is 36.2 0c -37.2 0c with low levels at 6 AM and higher levels at 6 PM

Core temperature is 0.5 0c higher than oral temperature

Normal daily temperature variation (diurnal variation) is typically 0.5 0c

Fever: AM $T^0 > 37.2$ 0c or PM $T^0 > 37.7$ 0c

$T^0 > 41.5$ 0c is referred as hyperpyrexia

Causes of fever include infection, trauma, surgery, malignancy, drug reactions and immune disorders.

Patterns of fever

1. Intermittent fever

Paroxysms of high grade fever lasting for only short periods of the day and characterized by high peaks which subside to the normal level or below

eg. Pyaemia, acute pyelonephritis

2. Remittent fever

Raised temperature throughout the whole or almost the whole of the day with difference between maximum and minimum $T^0 > 1.0^\circ\text{C}$

Most fevers are of this type

3. Continuous/ persistent fever

Raised temperature throughout the day with difference of maximum and minimum $T^0 < 1.0^\circ\text{C}$

eg. Enteric fever, military tuberculosis, infective endocarditis

4. Relapsing fever

Periods of continued fever alternating with completely afebrile periods

eg. Brucellosis, Hodgkin's lymphoma, relapsing fever, spirochetal infections

Blood pressure (BP)

Normal value: Systolic BP = 90mmhg to 140 mmhg; Diastolic BP = 60mmhg to 90 mmhg

Hypertension: systolic BP>140mmhg and/or diastolic BP>90mmhg

Hypotension: systolic BP<90mmhg and diastolic BP<60mmhg

The cause of hypertension is unknown in majority of cases (>90-95%), named as primary or idiopathic hypertension; the rest (5-10%) is secondary hypertension, and caused by renal diseases (renal parenchymal disease or renovascular disease), endocrinopathies (primary aldosteronism, acromegaly, and Cushing's syndrome), drugs (corticosteroids), etc...

The common cause of hypotension is body fluid loss including hemorrhage/bleeding, persistent vomiting, diarrhea, and overzealous use of diuretics, etc...

Pulse rate (PR)

Normal value: 60-90 beats per minute (bpm)

The heart rate raises about 18 bpm for every 1.0°C rise in temperature

Relative bradycardia (pulse- T^0 deficit) could be caused by enteric fever, Legionnaire's disease, chlamydial pneumonia and some viral infections

Respiratory rate (RR)

Normal values: 14-16 breaths per min

Tachypnea: RR >20 breaths per minute

Bradypnea: RR <8 breaths per minute

Nutritional assessment

1. Body mass index (BMI)

BMI is weight in kg/ height in squared meter (Kg/M²)

Normal value: BMI of 18.5-24.9 Kg/M²

Table 2.1 Nutritional assessment based on BMI

Undernutrition

. Mild	17.5-18.5 Kg/M ²
. Moderate	16-17.5 Kg/M ²
. Severe	<16 Kg/M ²

Overweight 25-29.9 Kg/M²

Obesity ≥ 30 Kg/M²

. Moderately obese 30-34.9 Kg/M²

. Severely obese 35-39.9 Kg/M²

. Morbid obesity ≥ 40 Kg/M²

2. Mid upper arm circumference (MUAC)

If weight or height can't be measured or obtained, nutritional assessment can be estimated using the MUAC

Mid arm is midpoint of shoulder and elbow

. Measure the circumference of the arm at the midpoint using tape measure

MUAC of 23.5-25 cm may indicate BMI of 18.5-20 Kg/M²

MUAC < 23.5 cm indicates under nutrition

3. Skin fold thickness

Nutritional status can be measured at sites such as the biceps, triceps, infrascapular, and supraclavicular regions using Harpenden calipers

Triceps skin fold (TSF): Mid way between shoulder and elbow is the most commonly used site

It is measured in the vertical plane with arm hanging relaxed by the side of the body

TSF equal to 12.5mm in male, and equal to 16.5mm in female is standard normal

TSF \leq 10mm in male, and \leq 13mm in female indicates undernutrition

4. Waist circumference/ Waist-to-hip ratio

The waist circumference is measured with a flexible tape measure placed on a horizontal plane at the level of the iliac crest

The waist circumference is a strong predictor of the degree of intra-abdominal (visceral) fat

Increased waist circumference can be a marker for increased cardiovascular risk even in persons with normal weight

All adult patients should be screened for overweight and obesity by measuring BMI and waist circumference at periodic health examination

Individuals with $\text{BMI} \geq 25 \text{ kg/m}^2$, waist circumference $> 80 \text{ cm}$ (females) or $> 94 \text{ cm}$ (males), and waist-to-hip ratio of > 0.8 (females) and > 0.9 (males), further evaluation for cardiovascular risk factors (dyslipidemia, diabetes, and hypertension) and comorbidities (coronary heart disease, cerebrovascular disease, and peripheral arterial disease) is required

Waist circumference and waist-to-hip ratio and risk of metabolic complications

Variables Increased risk of metabolic complications

. Waist circumference

Males >94 cm

Females >80 cm

• Waist-to-hip ratio

Males ≥ 0.9

Females >0.8

CHAPTER THREE

General appearance and HEENT

Learning objective

At the end of this lesson, the student should be able to:

1. Mention main symptoms in head, eye, ear, nose and throat problems
2. Interpret abnormal findings in eye, ear, nose and throat examination
3. List common causes of hearing loss and red eye

General appearance

Physical examination requires a cooperative patient and quiet, warm and well lit room equipped with a coach or chair. For a thorough examination, the patient should be asked to undress completely or at least to their under clothes, and to lie or sit on the coach or bed partially covered with a bed sheet or dressing gown.

The general examination begins as soon as the patient enters the consulting room. Greet your patient in a friendly, but professional manner. Facial expression and eye-to-eye contact are indicators of physical and psychological well-being.

At the start of examination on coach or chair, an analysis of the patient's physique, posture, complexion, nutritional status, and emotional status is made, which constitute the individual's appearance.

Individual's appearance should verify the following:

- . Physique
- . Posture
- . Facial expression (complexion)
- . Nutritional status
- . Emotional status

Look for posture and gait of patient while standing and walking. Abnormal posture or gait may indicate locomotor or nervous system disorder.

Complexion may become a remarkably sensitive index of disease; eg. carboxyhemoglobin in carbon monoxide poisoning (pink), jaundice in liver disease (yellowish), uremia in chronic kidney disease (yellow-brownish tinge).

Short stature occurs in achondroplasia, chronic undernutrition or congenital heart disease; while increased height suggests gigantism or marfan's disease.

A look of excitement and anxiety may be features of hyperthyroidism or hypomania; while a look of apathy and poverty of facial expression may suggest hypothyroidism or Parkinsonism.

Clinical history taking and physical examination of HEENT

Head

1. The hair: Notice its quantity, colour, distribution, texture, and loss of hair.

Hair distribution: Is it male or female type of hair distribution?

Recession of hair at forehead margins or temporal recession is in favor of male type hair distribution. Notice for thin, sparse hair in hyperthyroidism, and coarse, brittle hair in hypothyroidism.

2. The scalp: Look for scaliness, lumps, nevi, warts

a. Itchy, scaling of scalp

. Pediculus capitis: Contact scratching with crusting and oozing scalp. Lice resemble grains of wild rice, attached to scalp or loosely adherent to hair. Nits (ova of lice) appear along hair shaft as adherent white granules

b. Scaly, lumpy or inflamed scalp restricted to the scalp

. Fungal infection (Tinea capitis): Patches of thinned and broken scalp hair with crusted with scaly inflamed scalp

. Bacterial infection: Crusted and oozy yellow patches scattered on scalp with unpleasant odor and regional neck lymphadenopathy

. Seborrheic dermatitis: Patchy or diffuse, yellowish greasy itching scale involving scalp

. Lichen simplex chronicus: Recurring, itchy, excoriated papules or patch

3. The skull: Notice for any deformities, depression, masses or tenderness

Eye

History taking in eye disease

Ask

. Have you had any blurring of vision?

. If yes, was it sudden or gradual onset?

Refractive errors most commonly causes gradual blurring of vision

Difficulty with close vision is named as hyperopia (far-sightedness) or presbyopia (aging vision) while difficulty with far vision is named as myopia (near-sightedness)

Sudden loss of vision suggests retinal detachment, vitreous hemorrhage or central retinal artery occlusion

. Is your visual blurring involved the whole visual field or part of it (central, peripheral, one-sided loss)? - refer to cranial nerve II (Nervous system)

Slow central loss is noticed in nuclear cataract and macular degeneration

Peripheral loss is noticed in advanced open-angle glaucoma

One-sided loss (hemianopsia/quadrantanopsia) occurs in optic tract lesion

. Are there speckles (scotomas) in the vision? If yes, do they move around in the visual field or fixed with shifts in gaze?

Moving scotomas are vitrous floaters, while fixed scotomas are retinal or visual pathway lesion

. Is there eye pain, redness and excessive tearing? - refer to causes of 'red eye' below

. Is there diplopia (double vision)? If yes, is it horizontal diplopia (the images are side by side) or vertical diplopia (the images are on top of each other)? Does diplopia persist with one eye closed? Which eye is affected?

Horizontal diplopia is noticed in cranial nerve III or VI lesion

Vertical diplopia is noticed in cranial nerve III or IV lesion

Examination of the eye

Visual acuity (Snellen chart) - refer to cranial nerve II (Nervous system)

The numerator records the distance of the subject from the test chart (usually 6 meter)

The denominator records the line read by the patient

The normal person can read the line designated 6 at 6 meter i.e. 6/6 vision

Visual field by confrontation- refer to cranial nerve II (Nervous system)

Eye lids- Notice for width of palpebral fissure, eye lid edema, and adequacy of eye lid closure

Blepharitis- inflammation of the eyelids along the lid margins, often with crusting or scales

Failure of eyelids to close occurs due to infranuclear facial nerve palsy, and leads to exposure keratitis

Lacrimal apparatus

Examine lacrimal gland by pulling up the outer part of upper eye lid while the patient looks downward and inward

Inspect the lacrimal sac for swelling, look for excessive tearing or dryness of eyes

Excessive tearing is due to increased production (conjunctivitis, keratitis) or impaired drainage of tears (ectropion, nasolacrimal duct obstruction)

Conjunctiva and Sclera

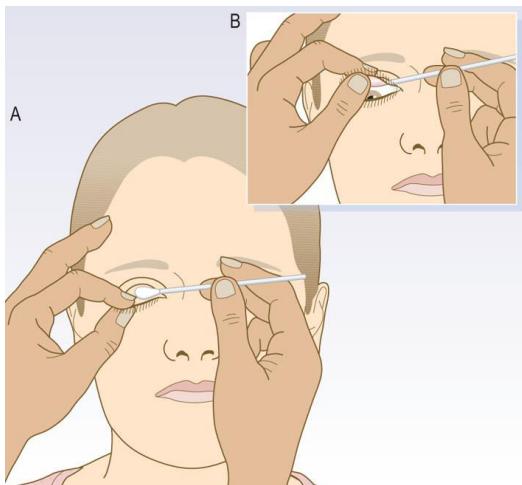
Look for conjunctival pallor, injection, hemorrhage and trachomatous changes, and for scleral jaundice and pterygium

. Ask the patient to look up as you depress both lower eye lids with your thumbs, and inspect the lower conjunctivae

Technique to view upper conjunctiva

. Instruct the patient to look down

- . Grasp the upper eyelashes and pull them gently down and forward
- . Place a stick, tongue depressor, 1cm above lid margin (upper border of tarsal plate)
- . Pull down the stick as you raise the edge of the upper eye lid (everting/taking inside out)
- . Inspect upper conjunctiva for trachomatous papules, other nodules or granuloma



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th ed

Fig 3.1. Technique to view palpebral conjunctiva

Cornea and lens

Inspect the cornea of each eye for opacities with oblique lighting

Iris

Normal iris is flat and forms a relatively open angle with the cornea, and light shining directly from the temporal side casts no shadow

Pupils

Inspect the size, shape and symmetry of pupils (large >5mm, small <3mm, or unequal)

Myosis is constriction of pupils while mydriasis is dilation of pupils

Pupillary inequality < 0.5mm is seen in normal people

- . Perform direct and consensual light reflex, and test for convergence- refer to cranial nerve II (Nervous system)

Extraocular movements

. Assess conjugate movements of the eyes in each direction- refer to cranial nerve III/IV/VI (Nervous system)

Nystagmus is fine rhythmic oscillation of the eyes

A few beats of nystagmus on extreme lateral gaze occurs in normal eye

Sustained nystagmus within the binocular field of gaze is seen in various neurologic problems

Other eye problems

- . Look for lid lag, lid retraction, proptosis, ptosis, xanthelasma, periorbital edema

Lid lag is upper eye lid lags behind the eyeball in a down ward gaze; lid retraction is evident by sclera visible above limbus in wide-eyed staring expression. Lid lag and lig retraction occur in Graves' disease

Proptosis is forward displacement of the eye ball. It is caused by Graves' disease, orbital tumours and carotid cavernous fistula

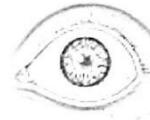
Ptosis is drooping of the upper eyelid, which occurs in oculomotor palsy, myasthenia gravis, myogenic disorders, and Horner's syndrome

Xanthelasma is subcutaneous lipid deposit at periorbital area; and signify presence of lipid disorder (dyslipidemia)

Periorbital edema occurs in nephrotic syndrome, allergic eye diseases, Graves' eye disease, and angio-oedema

Technique of performing lid lag:

- . Position the examiner's finger at patient's primary gaze
- . Instruct the patient to follow the examiner's finger while the examiner moving his finger down ward
- . “The upper eyelid lags behind the eyeball when the patient looks downward” indicates presence of lid lag

	Conjunctivitis	Corneal Injury or Infection	Acute Iritis	Glaucoma	Subconjunctival Hemorrhage
					
Pattern of Redness	Conjunctival injection: diffuse dilatation of conjunctival vessels with redness that tends to be maximal peripherally	Ciliary infection: dilation of deeper vessels that are visible as radiating vessels or a reddish violet flush around the limbus. Ciliary infection is an important sign of these three conditions but may not be apparent. The eye may be diffusely red instead. Other clues of these more serious disorders are pain, decreased vision, unequal pupils, and a less than perfectly clear cornea.			Leakage of blood outside of the vessels, producing a homogeneous, sharply demarcated, red area that fades over days to yellow and then disappears
Pain	Mild discomfort rather than pain	Moderate to severe, superficial	Moderate, aching, deep	Severe, aching, deep	Absent
Vision	Not affected except for temporary mild blurring due to discharge	Usually decreased	Decreased	Decreased	Not affected
Ocular Discharge	Watery, mucoid, or mucopurulent	Watery or purulent	Absent	Absent	Absent
Pupil	Not affected	Not affected unless iritis develops	May be small and, with time, irregular	Dilated, fixed	Not affected
Cornea	Clear	Changes depending on cause	Clear or slightly clouded	Steamy, cloudy	Clear
Significance	Bacterial, viral, and other infections; allergy; irritation	Abrasions, and other injuries; viral and bacterial infections	Associated with many ocular and systemic disorders	Acute increase in intraocular pressure—an emergency	Often none. May result from trauma, bleeding disorders, or a sudden increase in venous pressure, as from cough

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 3.1 Common causes of “Red eye”

Ear

Main symptoms of ear disease

- . Otalgia – earache or pain
- . Otorrhea – ear discharge
- . Hearing loss
- . Tinnitus – Perception of sound in the absence of appropriate auditory stimulus
- . Vertigo – Illusion of movement

Otalgia

Main causes of otalgia

Otological

- . Acute suppurative otitis media
- . Acute otitis externa
- . Barotrauma
- . Herpes zoster (Ramsay-Hunt syndrome- shingles of the facial nerve)
- . Viral myringitis
- . Malignant otitis externa (necrotizing otitis externa due to *Pseudomonas aeruginosa*)

Non-otological

- . Tonsillitis
- . Dental disease
- . Temporo-mandibular joint disease
- . Cervical spine disease
- . Nasopharyngeal cancer

Otorrhea

Main causes of otorrhea

- . Acute/chronic suppurative otitis media
- . Acute/chronic otitis externa
- . Cerebrospinal leak in basal skull fracture

Profuse mucoid ear discharge with pulsation suggests acute otitis media with perforated tympanic membrane

Hearing loss

Conductive hearing loss is due to disease in the external ear canal, tympanic membrane or middle ear

Sensorineural hearing loss is due to pathologies in the cochlea and sensory neural connections

Causes of hearing loss

Conductive hearing loss

- . Cerumen impaction in the external ear canal
- . Acute suppurative otitis media
- . Chronic otitis media- ear drum perforation, ossicular erosion, cholesteatoma
- . Otosclerosis
- . Middle ear effusion
- . Barotrauma to the eardrum or ossicular chain

Sensorineural hearing loss

- . Age associated hearing loss – presbyacusis
- . Noise-induced hearing loss
- . Meniere's disease
- . Drug-induced ototoxicity eg. Aminoglycosides, loop diuretics
- . Infective- meningitis, syphilis, measles, mumps

Hearing loss is of two major types. In *conductive hearing loss*, a disorder of the external or middle ear impairs the conduction of sound to the inner ear. In *sensineural hearing loss*, a disorder of the inner ear, the cochlear nerve, or its central connections impairs the transmission of nerve impulses to the brain. A *mixed hearing loss* has both deficits.

	Conductive Loss	Sensineural Loss
Distortion of Sounds That Impairs the Understanding of Words	Relatively minor	Often present as the upper tones of words are disproportionately lost
Effect of a Noisy Environment	Hearing may seem to improve.	Hearing typically worsens.
Patient's Own Voice	Tends to be soft; the patient's voice is conducted through bone to a normal inner ear and cochlear nerve.	May be loud; the patient has trouble hearing his own voice.
Usual Age of Onset	Most often in childhood and young adulthood, up to age 40	Most often in the middle or later years.
Ear Canal and Drum	An abnormality is usually visible, except in otosclerosis.	The problem is not visible.

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 3.2 Types of hearing loss

Tinnitus

Ringing, rushing or hissing sound in the absence of an appropriate auditory stimulus

Vertigo

Illusion of movement; the patient feels the surrounding environment is moving around, or the patient is moving in the surrounding environment

Causes of vertigo

Sudden onset – Acute labyrinthitis, vestibular neuritis

Vertigo with hearing loss and tinnitus – Meniere's disease

Vertigo (episodic) with position change – benign paroxysmal positional vertigo (BPPV)

Vertigo after trauma – Perilymph fistula

Vertigo with motion – Motion sickness

Drug-induced vertigo – Vestibulotoxic drugs like Gentamicin, Salicylate and Quinine

Vertigo with focal neurologic features (central vertigo) – Brain stem ischemia (TIA), multiple sclerosis, migraine, complex partial seizure

Examination of the ear

- . Inspect the pinna for its size, shape and deformity. Notice for periauricular areas swelling and erosions

Hot, tender postauricular swelling suggests mastoiditis

- . Notice for tragus and mastoid tenderness

Tragus tenderness is tenderness while pulling the tragus, and suggests middle ear infection

Otoscopic examination

- . Hold the otoscope like a pen between thumb and index finger with ulnar border of hand resting against the side of patient's head

- . Gently retract the pinna backwards and upwards to straighten the external meatus into line with the bony canal

- . Light is reflected from intact tympanic membrane at lower end downward and forwards to its periphery

- . Visualize for ear discharge, impacted wax, and membrane perforation



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 3.2 Examination of the ear using an otoscope

Nose

Main symptoms of nasal disease

- . Nasal blockage (commonly due to allergic rhinitis with nasal polyposis)
- . Rhinorrhea (nasal discharge)
- . Epistaxis (nose bleeds)
- . Sneezing

- . Disturbance of smell

Nasal discharge

Purulent discharge- infection in the nose/sinuses

Mucoid discharge- allergic rhinitis

Watery discharge- vasomotor rhinitis and cerebrospinal fluid (CSF) leak

Epistaxis

Unilateral- local cause in the nasal passage

Bilateral- systemic cause (thrombocytopenia, coagulopathy)

Sneezing: Sudden explosive effort to clear passages of irritants

It is common in viral upper respiratory tract infection and allergic rhinitis

Smell disturbance

Anosmia is complete loss of olfaction, and hyposmia is reduced sense of smell

Hyposmia or anosmia occurs in viral upper respiratory tract infection, allergic rhinitis with nasal polyposis blocking nasal passage, and craniofacial trauma causing olfactory nerve damage

Cacosmia is unpleasant smell caused by chronic anaerobic infection of the nasal passages and sinuses (usually unnoticed by the patient)

Important clues in smell disturbance: History of atopy, paranasal infection, maxillo-facial trauma, drug exposure (NSAIDs, anticoagulants), ‘snorting’ cocaine, occupational inhalation of dusts and chemical particulates

Examination of the nose

- . Inspect the nose from the front, side and back in a good light
- . Examine the nasal vestibule and intranasal contents by gently pushing the tip of the nose upwards with a finger, preferably using reflected illumination from a hand mirror
- . Inspect the anterior nasal cavity with nasal speculum or an otoscope
- . Look for nasal blockage, granulation on the nasal septum, nasal polyps, and nasal septum deviation and perforation
- . Notice for the presence of sinus tenderness

Maxillary tenderness is elicited by firmly pressing at the maxillary area (bony cheeks just below zygomatic bone), and frontal tenderness is elicited by pressing just below medial border of eye brow

Presence of sinus tenderness with headache and foul smelling nasal discharge suggests sinusitis

Mouth and throat

. Mouth odor: Notice mouth odor

Haluthosis (bad mouth odor) occurs in patients with poor dental hygiene, suppurative lung disease and peptic ulcer disease

Fetor hepaticus (fruity mouth odor) occurs in patients with hepatic encephalopathy

Uremic fetor (urine mouth odor) occurs in patients with uremia

Acetone breath occurs in patients with diabetic ketoacidosis

. Lip: Notice for lip ulcer, fissures and cracks

. Gingiva: Notice for gum bleeding, lead line and ulcers

. Teeth: Notice for tooth caries, extractions and dentures

. Tongue: Notice for tongue coating, fissures, atrophy of papillae

Whitish tongue coating with erythematic base while scraping with spatula suggests oral candidiasis.

. Buccal mucosa and palate: Notice for ulcers, patches, and masses in the buccal mucosa and palate

. Nasopharynx: Notice for tonsillar exudates, ulcers, masses

. Notice for post-nasal drip

Main symptoms of throat disease

. Throat pain

. Stridor or stertorous (noisy) breathing

. Dysphonia

. Dysphagia

. Neck swelling

Common causes of sore throat

. Viral pharyngitis

. Acute tonsillitis

. Acute follicular tonsillitis

- . Glandular fever (EBV)
- . Diphtheria

Stridor/stertor

Stridor is noisy breathing associated with upper airway obstruction at the laryngeal level

Stertor is noisy breathing with obstruction at the oropharyngeal level and commonly caused by adenotonsillar hypertrophy

Causes of stridor

- . Acute laryngitis
- . Laryngeal trauma
- . Laryngeal carcinoma
- . Epiglottitis

Dysphonia (hoarseness of voice)

Causes of dysphonia

- . Acute and chronic laryngitis
- . Laryngeal papillomatosis
- . Recurrent laryngeal nerve damage (thyroid surgery, lung cancer)
- . Vocal cord paralysis
- . Gastro-esophageal reflux
- . Psychogenic

Dysphagia (difficulty of swallowing)

Causes of dysphagia

- . Neuromuscular- motor neurone disease, myasthenia gravis
- . Intrinsic- acute tonsillitis, esophageal stricture, achalasia, esophageal cancer
- . Extrinsic- goiter
- . Systemic- scleroderma
- . Psychosomatic- globus pharyngeus

Neck lump (swelling)

Causes of neck lump

Midline neck structures

- . Goiter
- . Thyroglossal cyst
- . Submental lymph node swelling
- . Dermoid cyst
- . Laryngeal swelling

Lateral neck structures

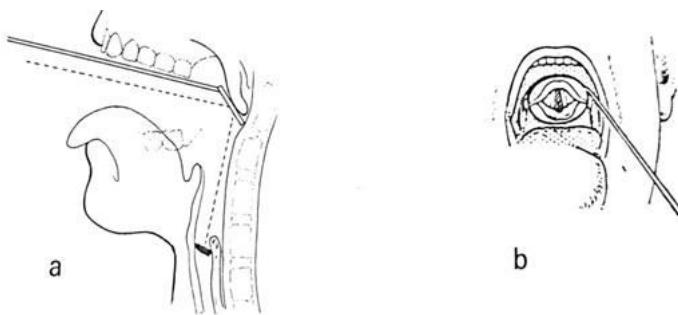
- . Pharyngeal pouch
- . Branchial cyst
- . Cervical lymph node swelling
- . Cystic hygroma
- . Carotid body tumour
- . Carotid artery aneurysm
- . parotid gland swelling

Throat examination

- . Inspect the oral cavity, pharynx and larynx
- . Depress the tongue with tongue depressor to visualize the tonsillar pillars, palatine tonsils, soft palate and uvula

Indirect laryngoscopic examination

- . Have a good light source (head light)
- . Remove if there are artificial dentures
- . Gently hold the protruded tongue with the gloved left hand, and ask the patient to take deep slow breath
- . Widely open mouth and gently introduce the laryngeal mirror
- . Displace the soft palate upwards and backwards with the mirror, and instruct the patient to say ‘ah’ and the larynx elevates towards the examining mirror
- . Examine the vallecula, epiglottis, supraglottis, glottis and vocal folds



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 3.2 Indirect laryngoscope a) Position of mirror in relation to soft palate and larynx b) View of larynx and vocal cord in mirror

CHAPTER FOUR

Lymphoglandular system

Learning objective

At the end of this lesson, the student should be able to:

1. List the peripheral accessible lymphnodes
2. Mention common causes of generalized lymphadenopathy
3. Mention causes of goiter
4. Show how to examine the thyroid gland and breast

Lymph nodes

History taking

Ask for swellings over the neck, axillae or groin

If there are swellings in lymph node areas- Ask for site and duration of swelling, painful or not, presence of B symptoms (fever, sweating and weight loss), history of genital ulcer, cough and expectoration, risk factors and clinical stigma of HIV infection (herpes zoster, oral thrush, chronic diarrhea), exposure to cats and pets, etc...

Examination of lymph nodes

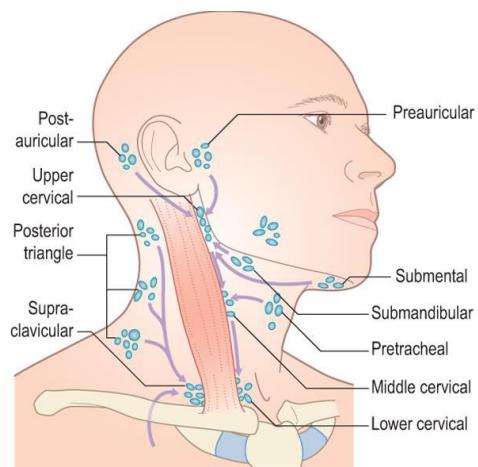
Palpate the peripheral accessible lymphnodes: preauricular, postauricular, occipital, submental, submandibular, anterior cervical, posterior cervical, supraclavicular, axillary, epitrochlear and inguinal lymphnodes

. Using the pad of your fingers, move the skin over the underlying tissues in lymph node areas

Note the size, shape, delimitation (discrete or matted together), mobility, consistency, tenderness, and “*fluctuation sign*” in enlarged lymph nodes (lymphadenopathy)

NB: Small, mobile, discrete, non-tender lymph nodes are frequently found in normal person

Generalized lymphadenopathy: ≥ 2 significant ($>1.5\text{cm}$) extra inguinal lymphadenopathy



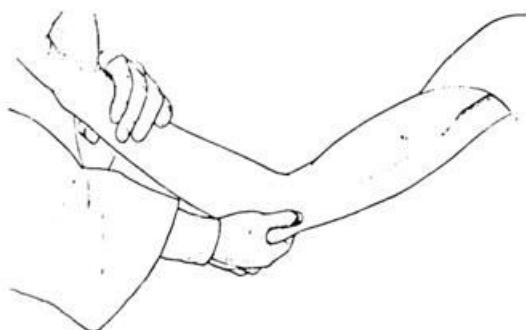
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 4.1 Peripheral lymph nodes in head and neck regions

Technique

Examination of epitrochlear lymph nodes

- . Epitrochlear lymphnodes are located at medial side of arm just proximal to medial epicondyle
- . Support the patient's right arm with your left hand to examine the right epitrochlear lymph node area, and the patient's left arm with your right hand to examine the left epitrochlear lymph node area
- . Palpate the right epitrochlear lymph node area with fingers of right hand, and left epitrochlear lymph node area with fingers of left hand



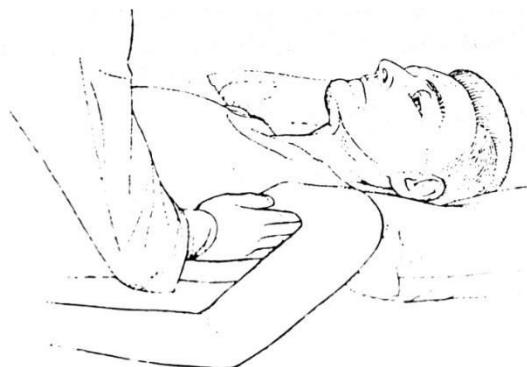
Palpation of the epitrochlear lymph node.

© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 4.2 Epitrochlear lymph node palpation

Examination of axillary lymph nodes

- . Axillary lymph nodes are located at the arm pits.
- . Support the patient's right arm with your right hand to examine the right axilla, and the patient's left arm with your left hand to examine the left axilla
- . Palpate the patient's right axilla with your left hand, and the patient's left axilla with your right hand
- . Cup together the fingers of your hand and reach as higher you can toward the apex of axillae, and milk down against the chest wall



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 4.3 Technique of examining axillae

Common causes of generalized lymphadenopathy

- . Infectious mononucleosis (EBV, CMV)
- . Persistent generalized lymphadenopathy (HIV infection): ≥ 2 extra-inguinal significant ($>1.5\text{cm}$) lymphadenopathy persisting for > 3 months
- . Secondary syphilis (*Treponema pallidum*)
- . Tuberculosis (*Mycobacterium tuberculosis*)
- . Toxoplasmosis (*Toxoplasma gondii*)
- . Hodgkin's lymphoma
- . Non-Hodgkin's lymphoma
- . Acute lymphoblastic leukemia
- . Chronic lymphocytic leukemia

Thyroid gland

The thyroid gland is located in the neck, anterior to the trachea, between the cricoid cartilage and the suprasternal notch. The gland consists of right and left lobes connected by an isthmus. It is highly vascular and soft in consistency. Enlarged thyroid gland is named as goiter.

History taking

Ask about mode of onset and duration of anterior neck swelling? age at neck swelling?

Ask about hoarseness of voice? difficulty in swallowing?

Ask about associated symptoms of hot or cold intolerance, anxiousness or clumsiness, menstrual irregularities, fatigue and weakness, dietary habits, residence in iodine-deficient area, family history of same illness, head and neck irradiation, etc...

Examination of thyroid gland

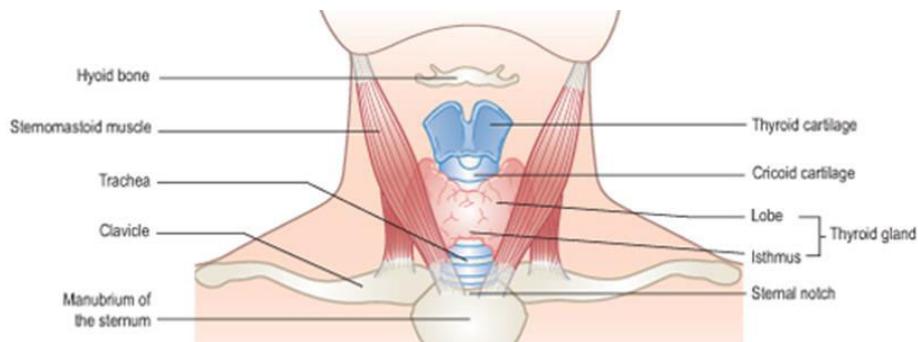
Inspection

- . Ask the patient to sit with neck muscles relaxed, and inspect the neck from the front
- . Look for the thyroid gland while the patient swallows a sip of water, and describe contour and symmetry of the gland

NB: The thyroid gland moves upwards on swallowing since it is enveloped in the pretracheal fascia which is attached to the cricoid cartilage

Palpation

- . Place the patient in sitting position on a chair/coach
- . Palpate enlarged thyroid gland from behind (stand behind the patient)
- . Extend the neck and head a bit backward
- . Put fingers of both hands over the enlarged lobes of the gland
- . Notice if the enlarged lobes move with swallowing, and suggests as goiter if it moves with swallowing
- . Push the left lobe of the gland with fingers of left hand towards the opposite side, and feel for tenderness, consistency, nodularity and surface of right lobe of the gland with your right hand fingers
- . Push the right lobe of the gland with fingers of right hand to the opposite side, and feel for tenderness, consistency, nodularity and surface of left lobe of the gland with your left hand fingers
- . Measure the length, width and depth of the goiter
- . Palpate as well for enlarged cervical and supraclavicular lymph nodes



a) Anatomic structure of the thyroid gland



b) Palpation of the isthmus

c) Palpation of thyroid lobes

© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 4.4 Technique of examining the thyroid gland: Examine the thyroid gland by standing behind the patient

Percussion

Percuss the anterior chest over the sternum for retrosternal extension of the goiter

NB: Dull to percussion over the sternum occurs in retrosternal goiter

Notice for 'Pumberton's sign'

Technique of eliciting 'Pumberton's sign'

- . Ask the patient to lift the arms over the head and wait for one minute
- . Note for development of facial plethora, cyanosis, inspiratory stridor and non-pulsatile elevation of the jugular venous pressure due to compression of the superior vena cava by retrosternal goiter at the thoracic inlet.

Auscultation

- . Put your stethoscope over the upper 1/3 of goiter and listen for bruit

Bruit is present in diffuse goiter due to Graves' disease

NB: Examination of thyroid gland is incomplete without palpating regional lymph nodes

Causes of goiter

a. Euthyroid goiter

- . Thyroid nodule
- . Nodular colloid goiter
- . Thyroid carcinoma

b. Hypothyroid goiter

- . Hashimoto's thyroiditis
- . Subacute thyroiditis (hypothyroid phase)

c. Hyperthyroid goiter

- . Graves' disease
- . Toxic multinodular goiter
- . Toxic adenoma
- . Functioning thyroid carcinoma
- . Subacute thyroiditis (hyperthyroid phase)

NB: Patients with goiter may have euthyroid, hypothyroid or hyperthyroid features

Symptoms of Thyroid Dysfunction		Signs of Thyroid Dysfunction	
<i>Hyperthyroidism</i>	<i>Hypothyroidism</i>	<i>Hyperthyroidism</i>	<i>Hypothyroidism</i>
Nervousness	Fatigue, lethargy	Tachycardia or atrial fibrillation	Bradycardia and, in late stages, hypothermia
Weight loss despite an increased appetite	Modest weight gain with anorexia	Increased systolic and decreased diastolic blood pressures	Decreased systolic and increased diastolic blood pressures
Excessive sweating and heat intolerance	Dry, coarse skin and cold intolerance	Hypodynamic cardiac pulsations with an accentuated S ₁	Intensity of heart sounds sometimes decreased
Palpitations	Swelling of face, hands, and legs	Warm, smooth, moist skin	Dry, coarse, cool skin, sometimes yellowish from carotene, with nonpitting edema and loss of hair
Frequent bowel movements	Constipation	Tremor and proximal muscle weakness	Impaired memory, mixed hearing loss, somnolence, peripheral neuropathy, carpal tunnel syndrome
Muscular weakness of the proximal type and tremor	Weakness, muscle cramps, arthralgias, paresthesias, impaired memory and hearing	With Graves' disease, eye signs such as stare, lid lag, and exophthalmos	Periorbital puffiness

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 4.1 Symptoms and signs of thyroid dysfunction

Thyroid cancer

Most common malignancy of the endocrine system

Presence of hard thyroid nodule fixed to adjacent structures with regional lymphadenopathy suggests thyroid malignancy unless proved otherwise

Risk factors for thyroid cancer in a patient with thyroid nodule

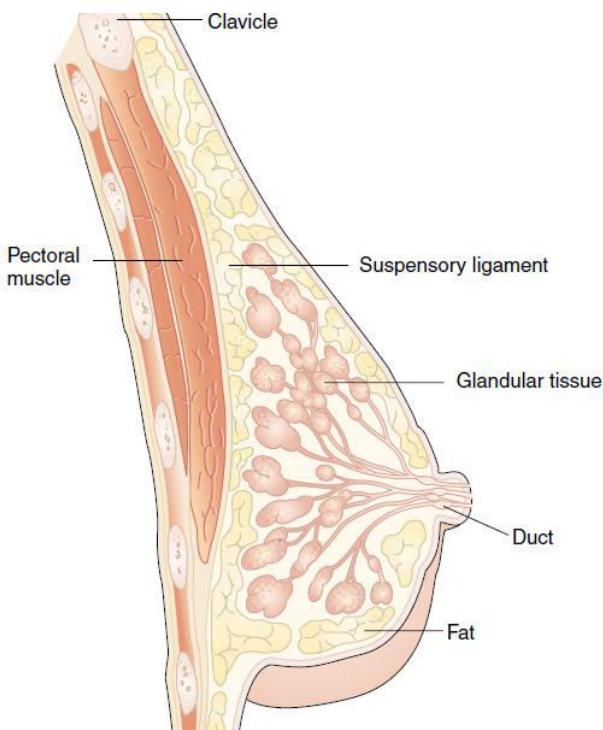
- . History of head and neck irradiation
- . Age <20 yrs or >50 yrs
- . New or rapidly enlarging neck mass
- . Family history of thyroid cancer
- . Vocal cord paralysis (hoarse voice)
- . Nodule fixed to adjacent structure
- . Suspected regional lymph node involvement

Breast

The female breast lies between the 2nd and 6th rib, between the sternal edge and the midaxillary line

Components of breast tissue

- . Glandular tissue- organizes into lobes that open into nipple
- . Fibrous tissue- suspensory ligaments connected to skin and fascia underlying the breast
- . Fat



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 4.5 Components of breast tissue

History taking

Ask presence of breast lump, swelling, skin ulceration and discharges from the nipple

Ask about age at menarche, marital status, parity, age at first child birth, history of lactation and breast-feeding, age at menopause, family history of breast cancer in first degree relatives (mother, sisters), history and duration of use of oral contraceptives and hormone replacement therapy (HRT)

Indicators of risk for breast cancer

- . Female gender
- . Increasing age
- . Family history of breast cancer
- . Early menarche
- . Nulliparity or late age of first child
- . Late menopause
- . Prolonged HRT use
- . Radiation therapy to breast region

Examination of female breast

Tell the patient that you are going to examine the breasts

Use gentleness and a matter-of-fact approach during examination

Inspection

Place the patient in sitting or lying position with arms at her sides

- . Look for size, symmetry and contour of the breasts
- . Look for size and shape of nipples
- . Look for skin ulceration, retraction and discharges

Ask the patient to sit and raise her arms over the head, or rest and press her hands against her hips

- . Look for dimpling or retraction of the breasts, shift in the relative position of the nipples, or a fixed mass distorting the breast

- . Notice for any swelling in the axillae

Palpation

Place the patient in a sitting position with both arms by one's side, leaning forward and then arms above the head. Examination is repeated in a lying down position with arms above the head.

- . Palpate upper outer quadrant followed by the lower outer, lower inner and upper inner quadrant in rotation, and at last the nipple and subareolar region of both breasts with pulps of fingers for any discrete, hard lump. The arm pits and the root of the neck above the clavicle are palpated for any nodular, hard swelling.
- . Note for consistency of breast tissue (soft fat with firmer glandular tissue in normal breast), tenderness, breast dimpling and retraction, breast lump or mass

If you detect breast lump

- . Location by quadrant or clock with cm from the nipple

- . Size in cm
- . Shape (regular, irregular)
- . Mobility in relation to skin and underlying muscle, and chest wall
- . Tenderness
- . Delimitation (well circumscribed or not)
- . Skin changes (dimpling, erythema, peau d' orange)

NB: Hard, irregular, poorly circumscribed breast lump, fixed to underlying tissue or chest wall strongly favors breast cancer

. Palpate each nipple, noting for elasticity

Thickening of the nipple and loss of elasticity suggests underlying breast cancer

- . Compress the areola with your index finger placed in radial positions around the nipple
- . Watch for discharge- describe for color, consistency and quantity of discharge

Blood- ductal papilloma, breast cancer

Yellow serous- fibroadenoma

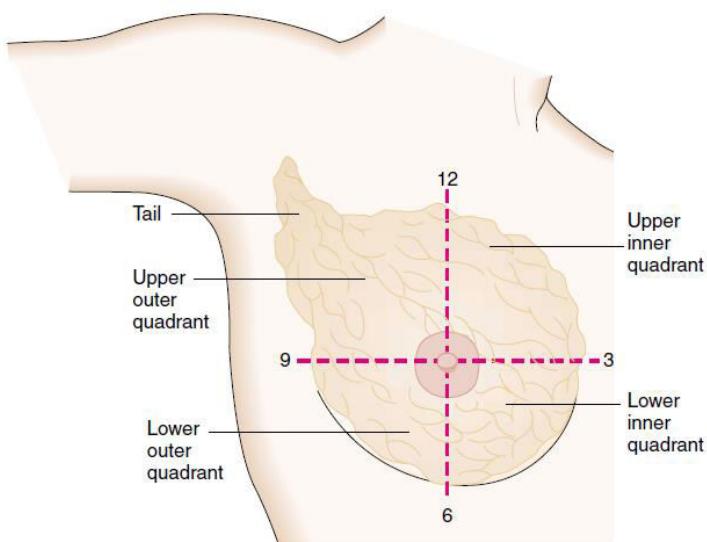
Serous fluid- early pregnancy

Milky- lactation

Green fluid- mammary duct ectasia

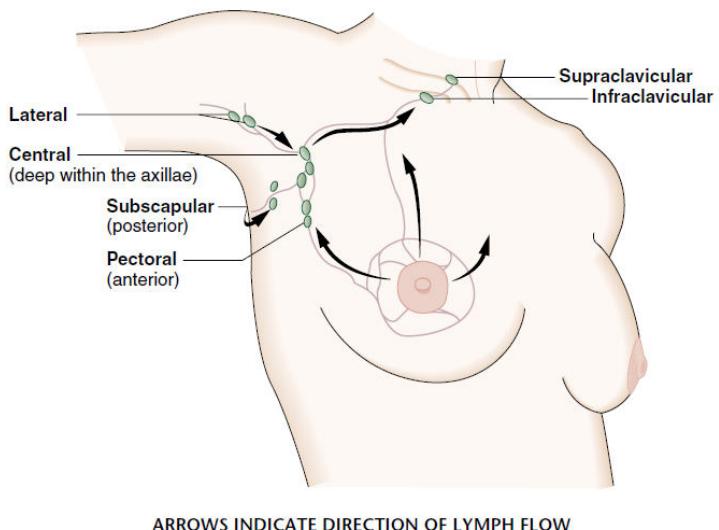
Milky discharge in non-lactating patient suggests nonpuerperal galactorrhea

Non-milky unilateral discharge suggests local breast disease, usually benign but may be malignant



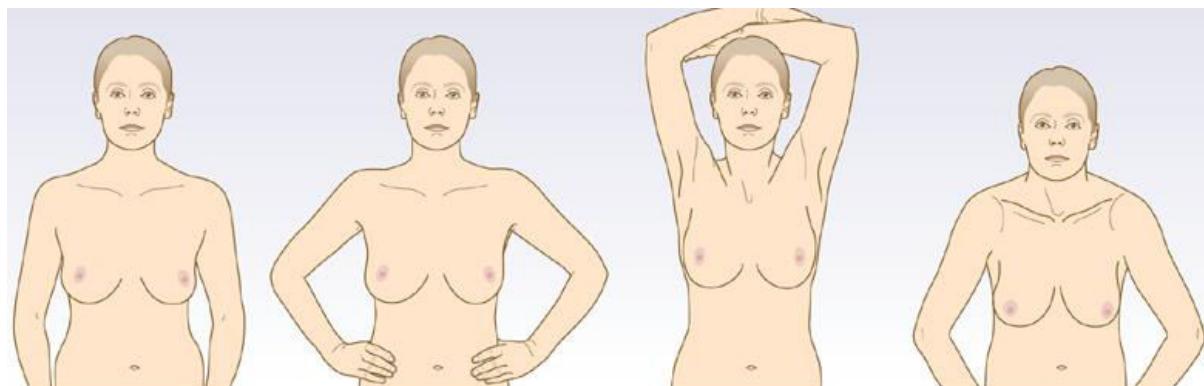
© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 4.6 Quadrants of right breast: Upper outer, lower outer, lower inner, upper inner



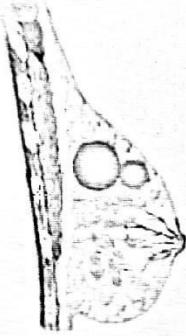
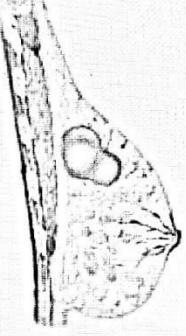
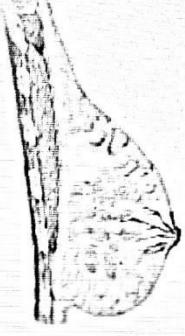
© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 4.7 Direction of lymphatic drainage of the breast tissue



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 4.8 Position in breast examination: Sitting position with both arms by one's side (a), sitting position with both arms pressing the pelvis (b), sitting position with arms raised above the head (c), sitting position with leaning forward (d)

	Fibroadenoma	Cysts	Cancer
			
Usual Age	15–25, usually puberty and young adulthood, but up to age 55	30–50, regress after menopause except with estrogen therapy	30–90, most common over 50 in middle-aged and elderly women
Number	Usually single, may be multiple	Single or multiple	Usually single, although may coexist with other nodules
Shape	Round, disklike, or lobular	Round	Irregular or stellate
Consistency	May be soft, usually firm	Soft to firm, usually elastic	Firm or hard
Delimitation	Well delineated	Well delineated	Not clearly delineated from surrounding tissues
Mobility	Very mobile	Mobile	May be fixed to skin or underlying tissues
Tenderness	Usually nontender	Often tender	Usually nontender
Retraction Signs	Absent	Absent	May be present

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 4.2 Cause of breast lump/mass

CHAPTER FIVE

Respiratory system

Learning objective

At the end of this lesson, the student should be able to:

1. Mention symptoms of respiratory disease
2. Perform techniques of respiratory system examination
3. Interpret clinical findings of various respiratory pathologies and diseases

History taking

Patients with respiratory disease present with one or more of the following symptoms

- . Cough
- . Sputum
- . Dyspnea (shortness of breath)
- . Hemoptysis
- . Wheeze
- . Chest pain
- . Stridor

Cough

Cough is an explosive expiration due to irritation of sensory receptors in the mucosa of the upper airways. It enables the tracheobronchial tree to be cleared of secretions and foreign bodies

Ask:

How long has the cough been present? Acute (< 3wks) or chronic (> 8wks) ?

Is the cough dry or productive (with expectoration of sputum)?

Is the cough worse at any time of the day or night?

Paroxysmal cough disrupting sleep due to nocturnal worsening suggests bronchial asthma

Cough of a worker in cotton industry that lessens on weekends suggest occupational asthma

Is the cough aggravated by any thing? Such as cold air, pollen, house dust (increased reactivity of air ways in bronchial asthma)

What is the quality of cough? Barking cough occurs in epiglottitis; bovine cough indicates vocal cord paralysis; brassy cough occurs in tracheal compression; wheezy muffled cough occurs in chronic bronchitis

Most common causes of cough (duration)

a. Acute cough (< 3 weeks)

- . Common cold
- . Acute bronchitis
- . Pneumonia
- . Pulmonary embolism

b. Chronic cough (>8 weeks)

- . Bronchiectasis
- . Chronic obstructive pulmonary disease
- . Interstitial lung disease
- . Lung cancer
- . Tuberculosis

Hemoptysis

Hemoptysis is an expectoration of blood from the respiratory tract (blood streaked sputum up to coughed up blood)

Massive hemoptysis is defined as expectoration of coughed up blood > 600ml over 24 hrs

Ask: Is there any blood in the sputum? Quantify amount in ml per day, how often and for how long?

Common causes of hemoptysis

- a. Tracheobronchial tree
 - . Bronchiectasis
- b. Pulmonary parenchymal source
 - . Pneumonia
 - . Lung abscess
 - . Tuberculosis
 - . Lung cancer
- c. Pulmonary vascular source
 - . Good pasture's syndrome (pulmonary hemorrhage, glomerulonephritis)
 - . Wegener's granulomatosis
 - . Pulmonary embolism

- . Mitral stenosis
 - . Acute left ventricular failure (pulmonary edema)
- d. Bleeding diatheses (coagulopathy)

Sputum

Ask:

- . What is the color, consistency and amount (in teaspoonful or arabic coffee cup/day)?

Yellow or green sputum is usually purulent

Copious amount of sputum is expectorated in bronchiectasis, lung abscess, and empyema rupturing into bronchial tree

- . Is it position-dependent or not?

Position-dependent expectoration occurs in bronchiectasis

- . Is it foul smelling or not?

Foul smelling expectoration occurs in lung abscess caused by anaerobes

Main types of sputum (color)

- . Serous (watery/pink): pulmonary edema
- . Mucoid (clear, grey): bronchial asthma, chronic bronchitis
- . Purulent (yellow/green): bronchopulmonary infections: bronchiectasis, lung abscess
- . Rusty (red): Pneumococcal pneumonia

Chest pain

Chest pain originates from injured pleura, chest wall and mediastinal structures. The lungs are not a source of pain for their exclusive autonomic innervation.

Pleuritic chest pain is sharp and stabbing worsened by deep breathing and coughing

- . Describe about the site, type, mode of onset, radiation, severity, relieving and aggravating factors of chest pain (characterize using SOCRATES)

Common causes of pleuritic chest pain are pneumonia, pulmonary embolism, pneumothorax and fractured ribs

Chest wall pain occurs in intercostal muscle injury, thoracic herpes zoster, and direct invasion of chest wall by lung cancer, mesothelioma or malignant rib metastasis

Mediastinal pain occurs in irritation or infection of tracheobronchial tree

Dyspnea (shortness of breath)

Dyspnea is uncomfortable awareness of breathing that is inappropriate to the level of exertion

The patient often complains of ‘inability to get enough air into the chest’

. Describe about mode of onset, duration, severity and associated symptoms

Paroxysmal dyspnea with wheeze and cough, worsened with exposure to allergens suggest bronchial asthma

Sudden onset dyspnea with chest pain suggests pneumothorax, pulmonary embolism or rib fracture

Wheeze and stridor

Wheeze is continuous whistling noise during breathing and occurs in bronchial asthma, chronic bronchitis and endobronchial obstruction by foreign bodies

Stridor is rasping or croaking noise loudest on inspiration and indicates narrowing of the larynx, which occurs in laryngitis and croup

Social history

Smoking

. Ask: How many cigarettes per day, for how long?

How many pack-years? Pack-year is defined as number of packs smoked per day multiplied by number of years smoked

Almost all cases of lung cancer and chronic obstructive pulmonary disease (COPD) occur in those who have smoked

Prolonged biomass fuel exposure while indoor cooking is major risk factor for COPD in rural areas of developing world

Alcohol

Drinking alcohol in binge can result in aspiration pneumonia, and alcoholics are more likely to develop klebsiella pneumonia and lung abscess

Family history

Genetic susceptibility occurs in bronchial asthma, bronchiectasis (ciliary dyskinesia syndrome, cystic fibrosis), emphysema (α -1 anti-trypsin deficiency), etc...

Occupational history

- . Occupational asthma
- . Malignant mesothelioma due to chronic asbestos exposure
- . Pneumoconiosis due to chronic exposure to industrial fumes and gases

Problem	Cough and Sputum	Associated Symptoms and Setting
Acute Inflammation		
<i>Laryngitis</i>	Dry cough (without sputum), may become productive of variable amounts of sputum	An acute, fairly minor illness with hoarseness. Often associated with viral nasopharyngitis
<i>Tracheobronchitis</i>	Dry cough, may become productive (as above)	An acute, often viral illness, with burning retrosternal discomfort
<i>Mycoplasma and Viral Pneumonias</i>	Dry hacking cough, often becoming productive of mucoid sputum	An acute febrile illness, often with malaise, headache, and possibly dyspnea
<i>Bacterial Pneumonias</i>	Pneumococcal: sputum mucoid or purulent; may be blood-streaked, diffusely pinkish, or rusty <i>Klebsiella</i> : similar; or sticky, red, and jellylike	An acute illness with chills, high fever, dyspnea, and chest pain. Often is preceded by acute upper respiratory infection. Typically occurs in older alcoholic men
Chronic Inflammation		
<i>Postnasal Drip</i>	Chronic cough; sputum mucoid or mucopurulent	Repeated attempts to clear the throat. Postnasal discharge may be sensed by patient or seen in posterior pharynx. Associated with chronic rhinitis, with or without sinusitis
<i>Chronic Bronchitis</i>	Chronic cough; sputum mucoid to purulent, may be blood-streaked or even bloody	Often longstanding cigarette smoking. Recurrent superimposed infections. Wheezing and dyspnea may develop.
<i>Bronchiectasis</i>	Chronic cough; sputum purulent, often copious and foul-smelling; may be blood-streaked or bloody	Recurrent bronchopulmonary infections common; sinusitis may coexist
<i>Pulmonary Tuberculosis</i>	Cough dry or sputum that is mucoid or purulent; may be blood-streaked or bloody	Early, no symptoms. Later, anorexia, weight loss, fatigue, fever, and night sweats
<i>Lung Abscess</i>	Sputum purulent and foul-smelling; may be bloody	A febrile illness. Often poor dental hygiene and a prior episode of impaired consciousness
<i>Asthma</i>	Cough, with thick mucoid sputum, especially near end of an attack	Episodic wheezing and dyspnea, but cough may occur alone. Often a history of allergy
<i>Gastroesophageal Reflux</i>	Chronic cough, especially at night or early in the morning	Wheezing, especially at night (often mistaken for asthma), early morning hoarseness, and repeated attempts to clear the throat. Often a history of heartburn and regurgitation
Neoplasm		
<i>Cancer of the Lung</i>	Cough dry to productive; sputum may be blood-streaked or bloody	Usually a long history of cigarette smoking. Associated manifestations are numerous.

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 5.1 Common causes of cough and sputum

Examination of the respiratory system

Anatomy

Anteriorly, the apex of each lung rises about 2-4 cm above the inner third of the clavicle, and the lower border of the lung crosses the 6th rib at the mid-clavicular line, the 8th rib at the mid-axillary line and T-10 spinous process posteriorly

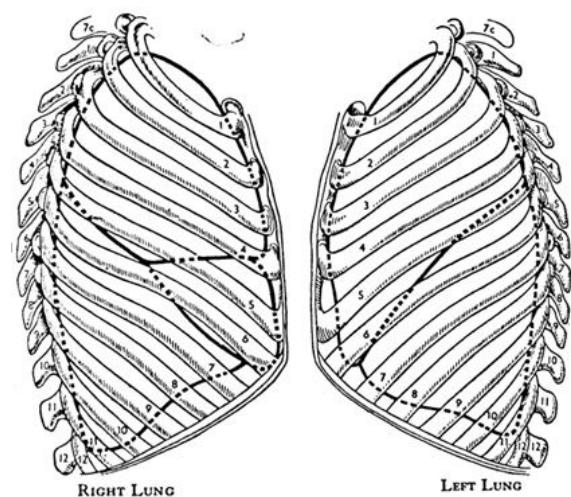
The right lung has 3 lobes; upper, middle and lower lobes. The left lung has 2 lobes, upper and lower lobes

The right lung has 2 inter lobar fissures (major and minor interlobar fissures) while the left lung has one major interlobar fissure

Surface lung markings of the interlobar fissures

A line from the 2nd thoracic spine to the 6th rib along the nipple corresponds to the upper border of the lower lobe (major interlobar fissure) of the right lung. Horizontal line at 4th costal cartilage to meet the line of major interlobar fissure near 5th rib along the midaxillary line marks the boundary between upper and middle lobes (minor inter lobar fissure)

The trachea bifurcates into main bronchi at the level of sternal angle anteriorly and the 4th thoracic spinous process posteriorly



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 5.1 Surface anatomy of the lung: Right lung has three lobes and two fissures (major and minor interlobar fissure), while the left lung has two lobes with single fissure (major interlobar fissure)

Technique of examination

Inspection

- . Listen to the patient's quality of voice while conversation

Clubbing

- . Look for the presence of clubbing

Clubbing is selective bulbous enlargement of the distal segments of fingers and toes

Grading of clubbing

Grade 1: Obliteration of the hyponychial angle (Nail fold angle $\geq 180^{\circ}$)

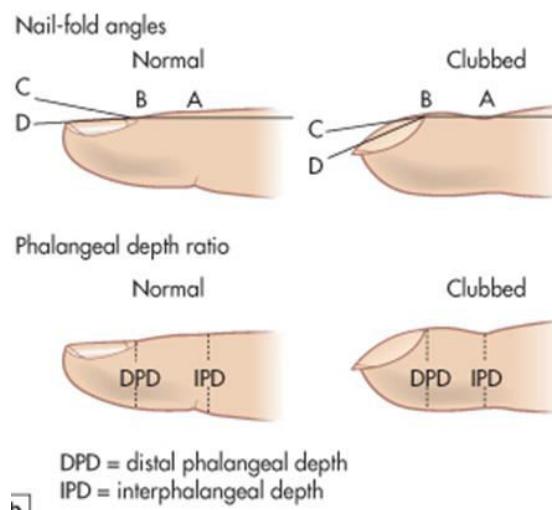
Hyponychial angle (nail fold angle) is 160° in normal individuals

Grade 2: ‘Spongy feel’ at nail fold (nail bed fluctuation)

Grade 3: Drum-stick appearance of distal phalanges of fingers/toes

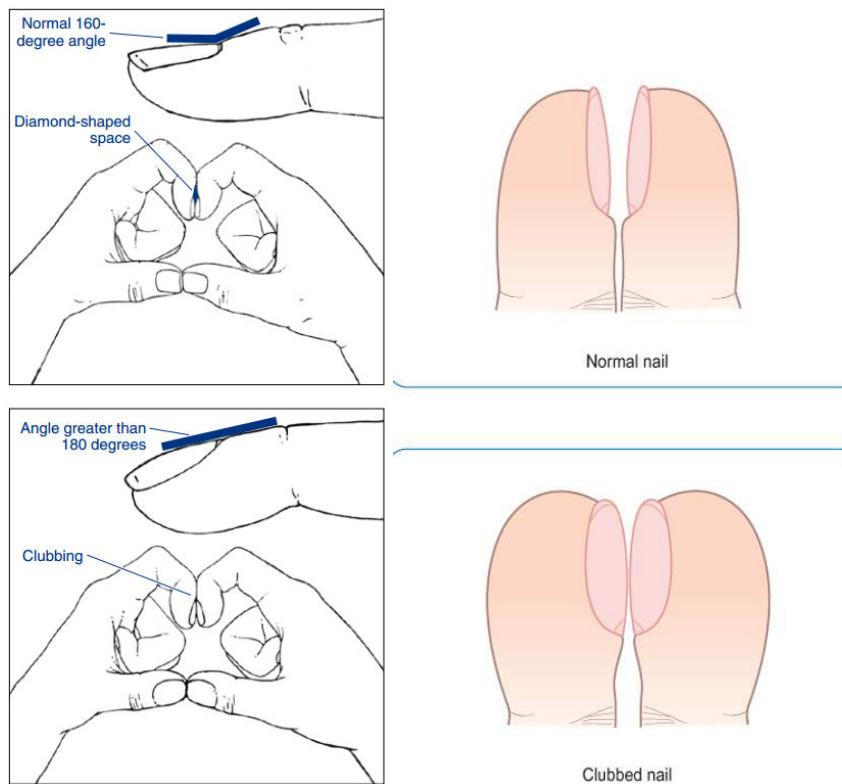
Phalangeal depth ratio >1 (distal phalangeal depth/interphalangeal depth)

Antero-posterior diameter at the distal inter-phalangeal joint (interphalangeal depth) is less than antero-posterior diameter at the nail fold (distal phalangeal depth) in patients with grade 3 clubbing



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 5.2 Nail clubbing: Obliteration of hyponychial angle (Nail fold angle $> 180^{\circ}$) and Phalangeal depth ratio >1 (distal phalangeal depth (DPD) $>$ interphalangeal depth (IPD))



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 5.3 **Schamroth's technique** to identify clubbing: normal nail (top) and Clubbed nail (bottom)

Common causes of clubbing

- . Hereditary (familial)
- . Pulmonary diseases- primary and metastatic lung cancer, bronchiectasis, lung abscess, cystic fibrosis, mesothelioma, tuberculosis
- . Cardiac diseases- cyanotic congenital heart disease, infective endocarditis
- . Gastrointestinal diseases- inflammatory bowel disease, hepatic cirrhosis
- . Idiopathic

Cyanosis

- . Look for the presence of cyanosis at lips and tongue

Cyanosis is bluish discoloration of the skin and mucus membrane resulting from an increased quantity of deoxygenated hemoglobin (reduced oxygen saturation)

Cyanosis becomes evident when the absolute concentration of deoxygenated hemoglobin is $\geq 5\text{gm/dl}$ of capillary blood

Cyanosis is usually obvious when the arterial oxygen saturation (SaO_2) falls below 90% in a person with a normal hemoglobin level

In patients with anemia, cyanosis doesn't occur until even greater levels of arterial desaturation is reached

Central cyanosis: bluish discoloration of lips and tongue due to arterial hypoxemia

Peripheral cyanosis (acrocyanosis): bluish discoloration of the distal parts of extremities due to vasoconstriction

Breathing pattern

- . Look for rate, depth and pattern of breathing

Abnormal breathing pattern

- . Rapid shallow breathing (Tachypnea: Respiratory rate (RR) >20 breaths per minute) occurs due to hypoxia in respiratory diseases eg. Severe pneumonia
- . Slow breathing (Bradypnea: RR <8 breaths per minute) occurs in drug-induced respiratory depression eg. barbiturate poisoning
- . Kussmaul's breathing: Fast, deep and labored breathing usually occurs in metabolic acidosis
- . Cheyne-stokes breathing: Periods of hyperpnea alternating with periods of apnea, due to a delay in the medullary chemoreceptor response to blood gas changes, and occurs in heart failure and cerebral hemispheric damage.
- . Apneustic breathing: Post-inspiratory pause in breathing, occurs in pontine damage
- . Ataxic (Biotic) breathing: Unpredictable, irregularity of respiration, occurs in medullary compression due to central transtentorial herniation
- . Paradoxical respiration: The abdomen sucks inwards with inspiration (it normally pouches outwards due to diaphragmatic descent) due to diaphragmatic paralysis

Sign of respiratory distress

- . Look for signs of respiratory distress

Signs of respiratory distress

- . Tachypnea (RR >20 breaths per minute)
- . Flaring of alae nasa
- . Use of accessory muscles of respiration (sternomastoid, platysma and strap muscles of the neck)
- . Supraclavicular/intercostal/subcostal retraction

The accessory muscles of respiration causes elevation of the shoulders with inspiration and aid respiration by increasing chest expansion

- . Observe the shape of the chest

The normal chest is bilaterally symmetrical and elliptical in cross section

Thoracic ratio (Antero-posterior diameter: transverse diameter ratio) is 0.7-0.9

Chest shape

Types of abnormal chest shape

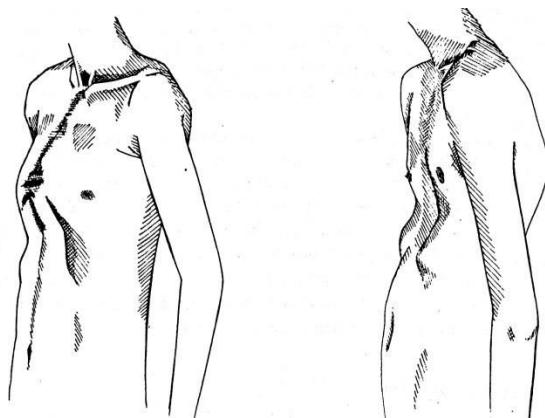
- . Barrel chest: Increased antero-posterior diameter of the chest in comparison to lateral diameter of the chest (Thoracic ratio: Antero-Posterior diameter/Lateral diameter > 0.9), often seen in COPD, chronic asthma, and accompanies normal aging
- . Funnel chest (Pectus excavatum): Depression in the lower end of the sternum
- . Pigeon chest (Pectus carinatum): Anteriorly displaced sternum with depressed costal cartilage
- . Harrison's sulcus: Linear depression of the lower ribs just above the costal margins at the site of attachment of the diaphragm, often seen in rickets and severe childhood asthma
- . Kyphosis: Exaggerated forward curvature of the spine
- . Scoliosis: Lateral curvature of the spine
- . Kyphoscoliosis: Forward and lateral bending of the spine

Kyphoscoliosis is idiopathic (80%) in majority of patients. It may reduce the lung capacity and increase the work of breathing

- . Observe symmetry of the chest during spontaneous breathing
- . Observe presence of chest lag/delay, intercostal and subcostal retraction

Chest lag to affected lung occurs in pneumothorax, hydrothorax, atelectatic and fibrosed lung

- . Look for the presence of paradoxical respiration (diaphragmatic paralysis?)



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 5.4 Chest deformity: Pigeon chest and funnel chest deformity

Palpation

Assess for the presence of chest wall tenderness

Look at the face of the patient while palpating the anterior and posterior chest for chest wall tenderness

Assess for the presence of subcutaneous emphysema (crackling sensation felt on palpating over gas-containing chest wall)

Assess for symmetry of chest wall expansion

NB: Both sides of the chest should expand equally during tidal breathing and maximal inspiration

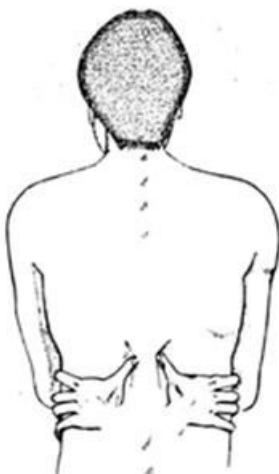
Technique for determining symmetry of chest wall expansion

Anterior chest

- . Place your thumbs along each costal margin, your hands holding the lateral rib cage
- . Slide your thumbs medially to raise skin folds
- . Ask the patient to inhale deeply and watch for divergence of your thumbs as the thorax expands
- . Observe for symmetry and degree of chest wall expansion

Posterior chest

- . Place your thumbs at the level of and parallel to the 10th rib, your hands grasping the lateral ribcage
- . Slide your thumbs medially in order to raise loose skin folds between your thumbs and the spine
- . Ask the patient to inhale deeply
- . Watch divergence of your thumbs during inhalation
- . Observe for symmetry and degree of chest wall expansion



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 5.5 Technique of examination for comparison of chest expansion (Posterior chest)

Technique for determining degree of chest wall expansion

. Measure the total circumference of the chest along the nipple using tape measure during quiet and deep respiration (the difference in chest expansion in deep and quiet breathing is about 4-6 cm)

Reduced chest wall movement on affected side may be due to localized lung fibrosis, lung collapse, pleural effusion or pneumothorax

Symmetrically reduced chest wall expansion occurs in emphysema, interstitial lung disease and diffuse fibrotic lung disease

Trachea position

. Assess position of the trachea

Technique

. Feel for the trachea by putting the index and ring fingers of right hand on each edge of sternal notch, and use the middle finger to assess whether the trachea is central or deviated to one side

A slight deviation of the trachea to the right side may be found in healthy individuals

. Assess for the presence of tracheal tug in air way obstruction

A tracheal tug is demonstrated when the finger resting on the trachea feels it move in inferiorly with each inspiration. It is a sign of hyperinflation of the chest because of tracheobronchial obstruction

Causes of tracheal displacement

Tracheal deviation towards the side of lung lesion

- . Upper lobe fibrosis
- . Pneumonectomy
- . Upper lung collapse

Tracheal deviation away from side of the lung lesion

- . Massive pleural effusion
- . Tension pneumothorax

Mediastinal mass displaces the trachea away from the mass

- . Retrosternal goiter
- . Lymphoma
- . Lung cancer

Tactile fremitus

Assess tactile fremitus (tactile resonance transmitted from the lung)

Technique

- . Put your hand over the chest with the palm touching the chest wall
- . Ask the patient to say ‘ninety-nine’ or ‘arba-arat’ repeatedly while the palm of hand over the chest wall
- . Compare the tactile fremitus in symmetric fashion from the apices to the lung bases in both lung fields anteriorly and posteriorly

Reduced tactile fremitus on affected lung occurs in lung collapse, pneumothorax, hydrothorax and fibrotic lung disease

Increased tactile fremitus on affected lung occurs in lung consolidation due to pneumonia

Percussion

Percussion of the chest sets the chest wall and underlying tissues into motion, producing audible sounds and palpable vibration

It helps to determine whether the underlying tissues are air-filled, fluid-filled or solid-filled

Technique of percussion

- . Put the hyper-extended middle finger of the left hand (the pleximeter finger) on the chest with the distal inter-phallangeal joint firmly on the surface to be percussed
- . Partially flex the right middle finger (the plexor), and strike the pleximeter finger at the distal inter-phallangeal joint with the tip of the plexor finger at 90° with a quick, sharp and relaxed wrist motion.
- . Percuss the anterior, lateral and posterior chest in symmetric fashion from the apices to the lung bases



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 5.6 Technique of percussion: Left middle pleximeter finger and right middle plexor finger

Identify the level of diaphragmatic dullness

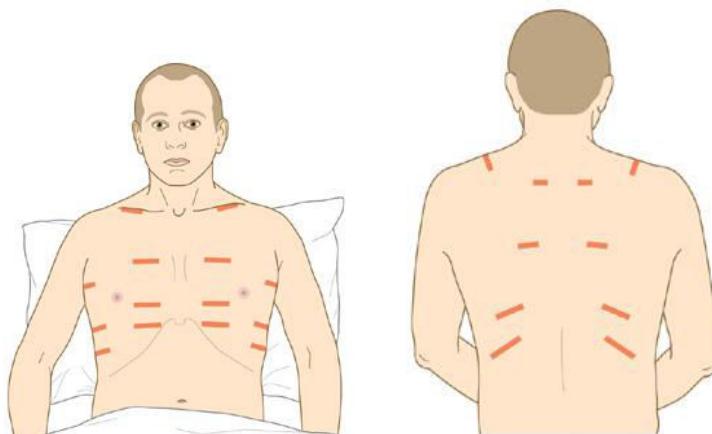
- . Percuss the posterior chest in progressive steps down ward with the pleximeter finger held above and parallel to the expected level of dullness

Diaphragmatic excursion: The distance between the level of dullness on full expiration and full inspiration (normal range of diaphragmatic excursion is 5-6 cm)

Technique of determining diaphragmatic excursion

- . Percuss posterior chest from apices to lung bases
- . Determine the level of dullness at full expiration and then at full inspiration
- . Determine the difference in cm

Reduced diaphragmatic excursion on affected side of lung occurs in hydrothorax, collapsed, atelectatic or fibrosed lung



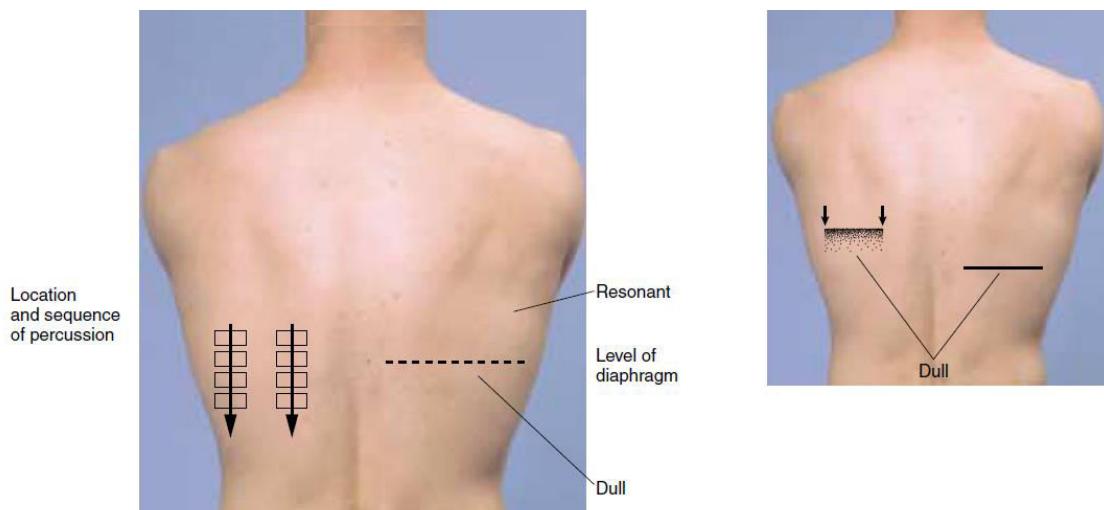
© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 5.7 Sites of percussion on anterior and posterior chest wall

Percussion Notes and Their Characteristics				
	Relative Intensity	Relative Pitch	Relative Duration	Example of Location
Flatness	Soft	High	Short	Thigh
Dullness	Medium	Medium	Medium	Liver
Resonance	Loud	Low	Long	Normal lung
Hyperresonance	Very loud	Lower	Longer	None normally
Tympany	Loud	High*	*	Gastric air bubble or puffed-out cheek

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 5.2 Percussion notes and their characteristics



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 5.8 Determining level of diaphragmatic excursion

Auscultation

Auscultation assesses airflow through the tracheobronchial tree

Breath sounds

Breath sounds probably originate from turbulent airflow in the airways

Normal breath sounds are classified according to their intensity, pitch and duration of their inspiratory and expiratory phases

- . Listen to the breath sounds with the diaphragm of the stethoscope after instructing the patient to breathe deeply through an open mouth
- . Auscultate the anterior, lateral and posterior chest in symmetric fashion from top to bottom
- . Note the intensity, pitch and duration of breath sounds in inspiratory and expiratory phase

NB: Avoid auscultation within 3cm of midline of chest, as these areas transmit sounds directly from trachea or bronchi

<i>Characteristics of Breath Sounds</i>				
	Duration of Sounds	Intensity of Expiratory Sound	Pitch of Expiratory Sound	Locations Where Heard Normally
Vesicular* 	Inspiratory sounds last longer than expiratory ones.	Soft	Relatively low	Over most of both lungs
Broncho-vesicular 	Inspiratory and expiratory sounds are about equal.	Intermediate	Intermediate	Often in the 1st and 2nd interspaces anteriorly and between the scapulae
Bronchial 	Expiratory sounds last longer than inspiratory ones.	Loud	Relatively high	Over the manubrium, if heard at all
Tracheal 	Inspiratory and expiratory sounds are about equal.	Very loud	Relatively high	Over the trachea in the neck

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 5.3 Characteristics of breath sounds

Adventitious (added) sounds

. Listen for adventitious sounds

Crackles

Crackles are produced by sudden changes in gas pressure related to the sudden opening of previously collapsed small airways

Crackles are intermittent and non-musical brief, high-pitched sounds

Wheezes

High pitched sounds with hissing or shrill quality

Wheeze is usually maximal during expiration and is accompanied by prolonged expiration

Rhonchi

Low-pitched sounds with snoring quality

Adventitious Lung Sounds

DISCONTINUOUS SOUNDS (CRACKLES OR RALES) are intermittent, nonmusical, and brief—like dots in time

Fine crackles (· · · ·) are soft, high pitched, and very brief (5–10 msec).

Coarse crackles (■ ■ ■ ■ ■) are somewhat louder, lower in pitch, and not quite so brief (20–30 msec).

CONTINUOUS SOUNDS are > 250 msec, notably longer than crackles—like dashes in time—but do not necessarily persist throughout the respiratory cycle. Unlike crackles, they are musical.

Wheezes (Haaaa) are relatively high pitched (around 400 Hz or higher) and have a hissing or shrill quality.

Rhonchi (Wwww) are relatively low pitched (around 200 Hz or lower) and have a snoring quality.

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 5.4 Additional breath sounds

Pleural friction rub

It occurs in pleural inflammation with creaking or rubbing quality

Transmitted voice sounds

Commonly observed in lung consolidation (eg. pneumonia)

Consolidated lung conducts sounds better than air-containing lung, and vocal resonance is increased and the sounds are louder and clearer

Bronchophony

. Ask the patient to say repeatedly ‘ninety-nine’, and louder, clearer sounds are heard on the chest wall

Aegophony

. Ask the patient to say ‘ee-ee-ee’, E-to-A change with nasal or bleating quality is heard

Whispering pectoriloquy

. Ask the patient to whisper ‘one-two-three’, louder, clearer whispered sounds are heard

Physical findings of common lung problems

Consolidation (lobar pneumonia)

Signs

Chest expansion- reduced on the affected side

Tactile fremitus- increased on the affected side

Percussion- dull to percussion

Breath sounds- bronchial

Additional sounds- medium, late or pan-inspiratory crackles

Vocal resonance- positive (aegophony, bronchophony and whispering pectoriloquy)

Pleural rub may be present

Lung collapse

Signs

Trachea- displaced towards the collapsed lung

Chest expansion- reduced on the affected side with flattening of the chest wall on the same side

Tactile fremitus- reduced on affected side

Percussion- dull over the collapsed lung

Breath sound- reduced +/- bronchial breath sound above the area of collapse

Causes of lung collapse

Intraluminal- mucus (post-operative, asthma, cystic fibrosis)

Mural- lung cancer

Extramural- peribronchial adenopathy

Pleural effusion: Collection of fluid in the pleural space

Pleural collections consisting of blood (hemothorax), chyle (chylothorax) or pus (empyema) have similar findings

Signs

Trachea and apex beat- displaced away from a massive effusion

Chest expansion- reduced on affected side

Tactile fremitus: reduced on affected side

Percussion- stony dullness on affected side

Breath sounds- absent air entry; bronchial breath sound audible above the level of effusion

Causes of pleural effusion

1. Exudative pleural effusion: Fulfill one or more of the following

Pleural protein-to-serum protein ratio > 0.5 , pleural LDH-to-serum LDH ratio > 0.6 , or pleural LDH (lactate dehydrogenase) is above 2/3 of upper normal serum level

- . Pneumonia with parapneumonic effusion
- . Malignancies- lung cancer, metastatic cancer, mesothelioma, lymphoma
- . Tuberculosis
- . Pulmonary infarction
- . Traumatic effusion
- . Connective tissue diseases (rheumatoid arthritis, systemic lupus erythematosus)
- . Acute pancreatitis
- . Drugs (cytotoxins, hydralazine, etc...)

2. Transudative pleural effusion: doesn't fulfill any of the above mentioned parameters

- . Congestive heart failure
- . Hypoalbuminemia from protein-losing enteropathy
- . Nephrotic syndrome
- . Hepatic cirrhosis
- . Hypothyroidism
- . Meig's syndrome (ovarian fibroma causing pleural effusion and ascites)

Pneumothorax: leakage of air from the lung or chest wall punctures into the pleural space

Signs

Chest expansion- reduced on affected side

Trachea is displaced away from affected side

Tactile fremitus- reduced on affected side

Percussion-hyperresonance on affected side

Breath sound- greatly reduced or absent

There may be associated subcutaneous emphysema (crackling sensation over gas-containing tissue)

Causes of pneumothorax

1. Spontaneous pneumothorax
 - . Subpleural bullae rupture (in tall and thin healthy individuals)
 - . Emphysema with rupture of bullae
 - . Pulmonary tuberculosis
 - . Bronchial asthma, lung abscess (rarely)
 - . Iatrogenic (following insertion of central venous catheter)
2. Traumatic
 - . Rib fracture
 - . Penetrating chest wall injury
 - . Iatrogenic (during pleural or pericardial aspiration)

Tension pneumothorax: It occurs when there is a communication between the lung and the pleural space, with a flap of tissue acting as a valve, allowing air to enter the pleural space during inspiration and preventing it from leaving during expiration

It causes displacement of the mediastinum with obstruction and kinking of the thoracic great vessels

Signs

The patient is often cyanotic, tachypneic, and hypotensive

Trachea and apex beat- displaced away from the affected side

Chest expansion- reduced over affected side

Tactile fremitus- reduced over affected side

Percussion- hyperresonant over the affected side

Breath sound- absent over affected side

Causes of tension pneumothorax

- . Penetrating chest wall injury
- . Mechanical ventilation at high pressure
- . Spontaneous

Emphysema

Signs

Barrel-shaped chest with thoracic ratio > 0.9

Pursed lip breathing

Use of accessory muscles of respiration

Positive Hoover's sign: "drawing in" of the lower intercostal muscles with inspiration

Palpation- reduced chest expansion of hyperinflated lung

Percussion- hyperresonant with decreased liver and cardiac dullness

Breath sounds- decreased; early inspiratory crackles

Wheeze is often absent

Pulmonary fibrosis

Signs

General- dyspnea, cyanosis, and clubbing may be present

Palpation- reduced chest expansion and tactile fremitus on affected side

Percussion- reduced on affected lung field

Auscultation- fine (Velcro-like) late inspiratory or pan-inspiratory crackles on affected side

Causes of lung fibrosis

1. Upper lobe: S₂CHA₂RT

. Silicosis

. Sarcoidosis

. Coal-worker's pneumoconiosis

. Histoplasmosis

. Ankylosing spondylitis

. Allergic broncho-pulmonary aspergillosis

. Radiation

. Tuberculosis

2. Lower lobe: RASCO

. Rheumatoid arthritis (RA)

. Asbestosis

- . Scleroderma
- . Cryptogenic fibrosing alveolitis
- . Others: drugs-busulphan, bleomycin, methotrexate, hydralazine, amiodarone

CHAPTER SIX

Cardiovascular system

Learning objective

At the end of this lesson, the student should be able to:

1. Mention the main symptoms of cardiovascular disease
2. Show techniques of precordial examination
3. Describe characters of abnormal arterial pulse
4. characterize murmur of stenotic or regurgitant valvular lesions

History taking

Patients with cardiovascular disease present with one or more of the following symptoms

- . Dyspnea
- . Orthopnea
- . Paroxysmal nocturnal dyspnea
- . Palpitation
- . Angina
- . Syncope
- . Leg swelling
- . Intermittent claudication
- . Fatigue

Dyspnea (shortness of breath)

Dyspnea is uncomfortable awareness of breathing

Dyspnea in cardiac patients occur when ever the work of breathing is excessive due to elevated left atrial and pulmonary capillary pressure causing transudation of fluid into the lung, requiring extra effort to ventilate the stiff lung

Determine level of dyspnea by the type of activity/exertion causing dyspnea

Level of dyspnea

Grade1- Dyspnea at supraordinal activities

eg. Short of breathe while running

Grade2- Dyspnea at ordinary activities

eg. Short of breathe while farming, climbing uphills

Grade3- Dyspnea at subordinary activities

eg. Short of breathe while combing hair, going to the toilet

Grade 4- Dyspnea at rest

Orthopnea

Dyspnea while assuming supine position due to gravitational pooling of blood to the lungs

. Quantify the level of orthopnea by the number of pillows required to alleviate dyspnea (eg. three-pillow orthopnea)

Causes of orthopnea

Common cause: Left ventricular failure (CHF)

Uncommon causes: Massive ascites, massive pleural effusion, bilateral diaphragmatic paralysis, severe pneumonia, pregnancy

Paroxysmal nocturnal dyspnea (PND)

PND is sudden breathlessness at night which wakes the patient from sleep chocking or gasping for air

Patients may sit on the edge of the bed and open windows in an attempt to relieve their distress

Palpitation

Palpitation is unpleasant awareness of the heart beat

Palpitation is caused by a change in the cardiac rhythm or rate, or by an increase in the force of cardiac contraction

Patients commonly describe palpitation as ‘jumping’, ‘pounding’ ‘racing’ ‘skipping’ or ‘fluttering’ heart beats

Ask

. Onset and termination (abrupt or gradual)

. Sustained or paroxysmal (frequency and duration of episodes if paroxysmal)

. Character of rhythm (ask the patient to replicate the rhythm by tapping it out on a table)

. Precipitating factors (eg. exercise, alcohol, drugs etc...)

. Associated symptoms (syncope, light-headedness, dizziness or dyspnea)

NB: Rapid irregular palpitation is typical of atrial fibrillation. Transient skips and flip-flops signify extrasystole (PVC).

Ankle swelling

Ask

- . Are there swelling over the legs in ambulating patient or pre-sacral area in bed-confined patient?
- . Are the rings tight on your fingers?

Syncope

Syncope is a transient loss of consciousness resulting from cerebral anoxia, usually due to reduced cerebral blood flow

Syncope may represent a simple faint, and it is a symptom of cardiac or neurologic disease

Main causes of syncope

- . Postural hypotension (drop in systolic blood pressure >20 mmhg on standing)
- . Arrhythmias (brady- or tachy-arrhythmias)
- . Left ventricular outflow obstruction (severe AS, HOCM)
- . Neurocardiogenic syncope (due to abnormal autonomic reflexes, i.e. painful or emotional stimuli)

Ask

- . What is the circumstance during syncopal attack?

While standing for prolonged periods or standing up suddenly (postural syncope), or while passing urine (micturition syncope), on coughing (tussive syncope) or with sudden emotional stress (vasovagal syncope)

- . Were there warning symptoms or not?

It may occur suddenly without warning or preceded by symptoms of faintness or pre-syncope such as lightheadedness, dizziness, feeling of warmth, diaphoresis, nausea and tunneling of vision

- . How long does the episode last? Is it recurrent or not?

Syncope due to arrhythmia is often sudden onset regardless of the patient's posture

Exertional syncope may occur with obstruction to left ventricular outflow by severe aortic stenosis (AS) or hypertrophic cardiomyopathy (HOCM)

Inquire about use of anti-hypertensive or anti-anginal drugs in postural syncope

Angina

Angina is retrosternal chest pain with squeezing, heaviness, pressure or burning character, radiating to the left shoulder, neck, jaw, teeth and medial border of left arm, which is worsened by exertion and relieved by rest or nitrates.

- . Describe about the location, duration, quality of pain, radiation, aggravating and relieving factors

Types of angina

1. Stable angina

- . Characteristic retrosternal chest discomfort persisting for 2-10 minutes, worsened by exertion and relieved by rest or nitrates

2. Unstable angina

- . Crescendo angina- increasing severity, frequency and duration of retrosternal chest discomfort

- . New onset angina- Worsened angina in the last one month

- . Angina at rest

3. Angina in acute myocardial infarction (AMI)

- . Angina persisting for more than 30 minutes and not relieved by rest or nitroglycerin

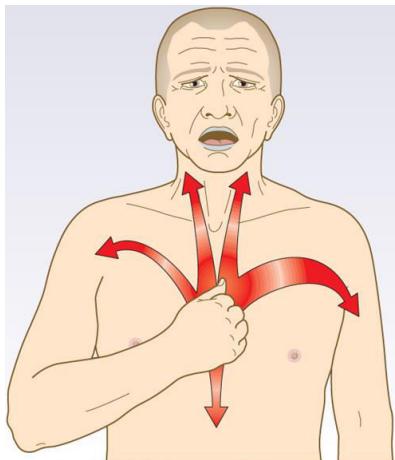
Factor	Angina	Myocardial infarction
Site	Retrosternal; radiates to arm, epigastrium, neck	Retrosternal; radiates to arm, epigastrium, neck
Precipitated	By exercise or emotion	Often spontaneous
Relieved	By rest, nitrates	Not by rest or nitrates
Anxiety	Absent or mild	Severe
Sympathetic activity	None	Increased
Nausea or vomiting	Unusual	Common

© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Table 6.1 Comparison of angina pectoris and myocardial infarction

Differential diagnosis of chest pain mimicking myocardial infarction

1. Pericarditis- Retrosternal or left precordial sharp chest pain radiating to left shoulder and back, which is worsened by deep breathing and change in posture, and relieved by sitting up and leaning forward
2. Esophageal spasm- Retrosternal burning or pressing pain, persisting for 2-30 minutes, precipitated by eating or drinking hot or cold fluids and may be relieved by nitrates.
3. Massive pulmonary embolism- Abrupt onset of pleuritic chest pain, and associated with dyspnea, cyanosis and syncope.
4. Aortic dissection- Abrupt onset of unrelenting, tearing, ripping, knife-like chest pain radiating to the back and between shoulder blades with associated symptoms of autonomic stimulation like pallor, sweating, syncope, bradycardia or hypertension
5. Spontaneous pneumothorax- Sharp pain localized to the chest with severe dyspnea



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 6.1 Site and radiation of angina

Intermittent claudication

Patients with claudication notice pain in one or both calves, thighs, or buttocks when they walk more than a certain distance. The distance walked inducing claudication is named as ‘claudication distance’.

- . Determine the degree of claudication distance

History of claudication suggests peripheral arterial disease (PAD) with poor blood supply to the affected muscles. Other vascular diseases like coronary artery disease are often present.

Fatigue

Common symptom of cardiac disease, and may be associated with reduced cardiac output and poor blood supply to the skeletal muscles

Additional history

- . Ask about history of congenital heart disease: Was she/he not exerting equally with her/his peers during childhood?
- . Ask about previous history of acute rheumatic fever in patients with valvular heart disease (previous history of migratory joint pain and swelling, chorea (abnormal body movement), and recurrent attacks of childhood tonsillitis)
- . Ask about major risk factors for coronary artery disease (CAD)

Hyperlipidemia (family history, on statin therapy or not)

Smoking (duration and amount of smoking in Pack-Years)

Hypertension (duration of hypertension, presence of target organ damage, on anti-hypertensive drugs or not)

Family history of premature CAD (1st degree relatives < 45 years in males or < 55 years in females)

Diabetes mellitus (DM) (duration of DM, presence of chronic complications, on injectable insulin or oral hypoglycemic drugs)

Chronic kidney disease (CKD) (how long? stage of CKD? etiology of CKD)

Examination of cardiovascular system

Blood pressure

Blood pressure is a measure of the force that the circulating blood exerts against the arterial wall

It is usually measured by means of sphygmomanometer cuff

The length and width of inflatable bladder of the cuff should be about 80% and 40% of upper arm circumference respectively (usually 23-35cm in length and 12.5cm in width)

Technique of measuring blood pressure

- . Patients should avoid smoking or ingestion of caffeine 30 minutes before, and rest in quiet, warm room for 5 minutes
- . With the patient seated or lying down, position the arm so that brachial artery is at heart level
- . Wrap the cuff securely over the upper arm of patient, with the centre of the bladder over the brachial artery, and lower border of cuff to be 2.5 cm above the antecubital crease
- . Place the bell of stethoscope lightly over the brachial artery
- . Feel for radial artery with fingers of one hand and inflate the cuff until the radial pulse disappears and read the pressure on the manometer and add 30 mmhg to it
- . Deflate slowly at a rate of 2-3 mmhg per second, and notice at manometer where the sound appear, muffled and disappear

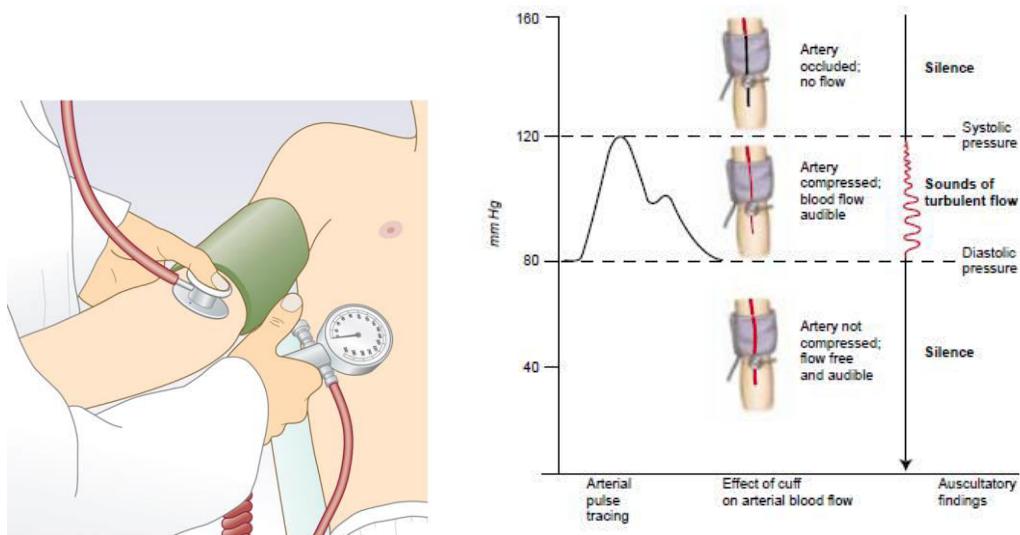
Korotkoff sounds

Phase1- the 1st appearance of the sounds marking systolic pressure

Phase 2 and 3- increasing loud sounds

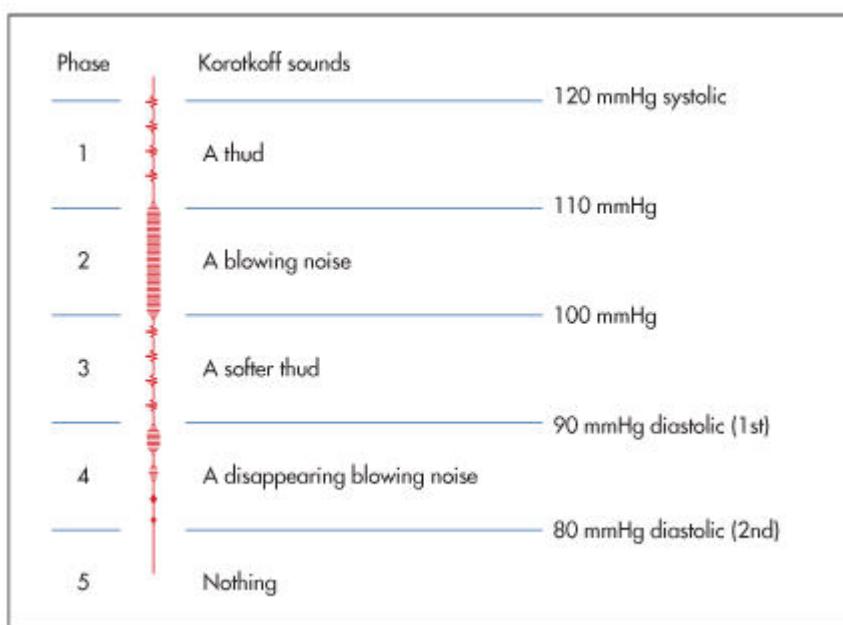
Phase 4- abrupt muffling of the sounds

Phase 5- disappearance of the sounds marking diastolic pressure



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig. 6.2 Measuring blood pressure and Auscultatory findings



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 6.3 Phases of Korotkoff sounds

When Korotkoff sounds remain audible despite complete deflation of the cuff, take phase 4 as diastolic pressure, which usually noticed in aortic regurgitation, pregnancy, etc...

Blood pressure (BP) should be taken in both arms at least once; a difference of ≤ 5 mmhg in both arms is acceptable

BP should be recorded in supine and erect position to rule out postural hypotension

Postural hypotension: Drop in systolic BP >20mmhg or diastolic BP >10mmhg while assuming from supine to erect position

Causes of postural hypotension

- . Hypovolemia (dehydration, acute blood loss)
- . Drugs (eg. vasodilators, anti-depressants, diuretics)
- . Addison's disease
- . Hypopituitarism
- . Autonomic neuropathy (diabetes)
- . Idiopathic

Table 6.2 Classification of hypertension in adults

	SBP (mmhg)	DBP (mmhg)
Normal	<120	<80
Prehypertension	120-139	80-89
Stage 1 hypertension	140-159	90-99
Stage 2 hypertension	≥160	≥100

Hypertension should be diagnosed only when a higher than normal level has been found on ≥ 2 visits after initial screening

Arterial pulse

The arterial pulses should be palpated for evaluation of rate, rhythm, character, volume, radio-femoral delay, and condition of arterial wall

Feel for all peripheral arteries- radial, brachial, carotid, femoral, popliteal, posterior tibial and dorsalis pedis arteries in both upper and lower extremities

1. Rate and rhythm

Radial artery is commonly used to assess heart rate and rhythm

Technique

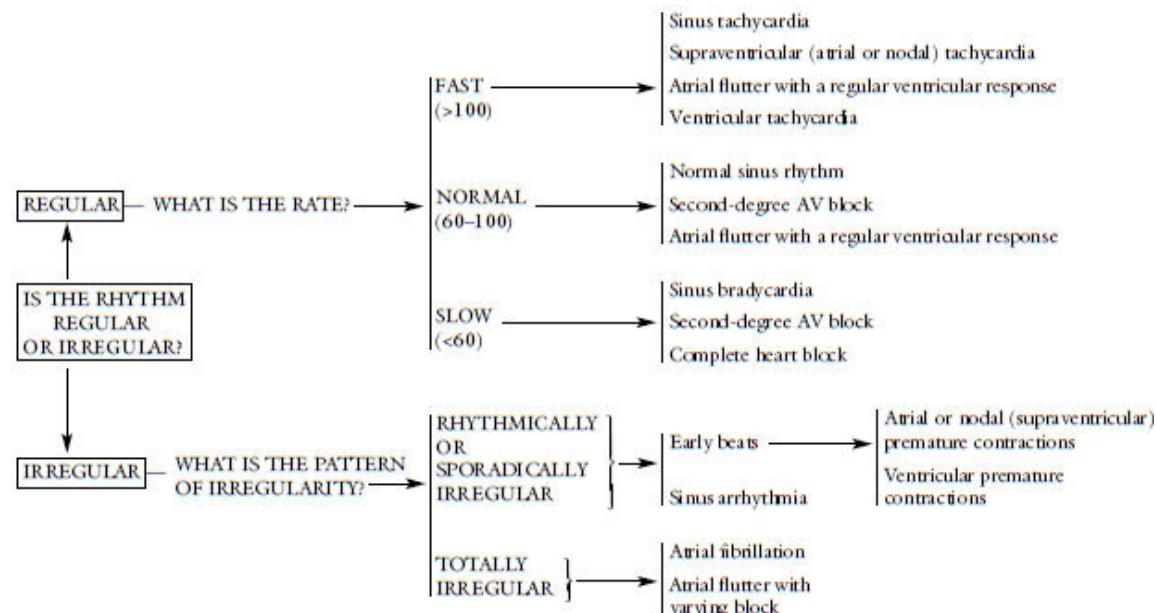
- . Compress radial artery with pads of index and middle fingers and count pulse rate for one minute
- . Determine the rhythm in radial artery; is the rhythm regular, regularly irregular or totally irregular?

Regularly irregular pulse rhythm is often due to ectopic beats, while total irregularity of the pulse is due to atrial fibrillation

NB: Electrocardiography (ECG) is indicated to clearly characterize the arterial pulse rhythm

Determine pulse deficit

Pulse deficit is difference in rate of heart beat and peripheral pulse. It is significant when the difference is >10 beats per minute. It is often observed in atrial fibrillation (due to failure in conducting all central beats to peripheral pulse).



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 6.3 Abnormalities in arterial rate and rhythm

2. Pulse character (amplitude and contour)

Pulse character is best assessed in the carotid artery, except in collapsing pulse where radial artery is preferred

Technique

- . Place your left thumb, or left index and middle fingers on the right carotid artery on the lower third of the neck, roughly at the level of cricoid cartilage

NB: Avoid pressing on the carotid sinus, which lies at the level of thyroid cartilage. Compressing the carotid sinus results in asystole.

Never press both carotid arteries at the same time, which decreases blood flow to the brain and results in syncope

. Characterize pulse amplitude and pulse wave contour

Amplitude of the pulse correlates reasonably with pulse pressure

. Characterize contour of the pulse wave – speed of upstroke, duration of summit and speed of down stroke

Normally, upstroke is smooth and rapid, the summit is smooth and rounded, and down stroke is less abrupt than upstroke

. Notice for variation of amplitude from beat-to-beat and with respiration

Arterial pulse wave forms in altered cardiac hemodynamics

a. Hypokinetic pulse

It occurs in hypovolemia, left heart failure, constrictive pericarditis and mitral stenosis

b. Hyperkinetic pulse

Large, bounding pulse usually associated with increased left ventricular stroke volume, wide pulse pressure and reduced peripheral vascular resistance

It occurs in anemia, patent ductus arteriosus, thyrotoxicosis and pregnancy

c. Anacrotic pulse

Slow-rising pulse with notched wave on upstroke and often seen in aortic stenosis

d. “water-hammer” (collapsing) pulse

Normal rising pulse wave, followed by abrupt decline due to rapid ‘run off’ of blood from arterial tree in diastole, and usually occurs in aortic regurgitation

e. Bisferiense pulse

Rapidly and forcefully rising upstroke (percussion wave) followed by decline in pressure and then followed by smaller and slowly rising pulse wave (tidal wave)

Two systolic peaks in bisferiense pulse, and characteristic of mixed aortic regurgitation and stenosis

f. Pulsus alternans

Alternating high and low systolic amplitude despite regular rhythm, which occurs in severely impaired left ventricular contraction (advanced heart failure)

g. Pulsus paradoxus

Accentuated decrease in arterial pulse amplitude during inspiration, which is observed in pericardial tamponade

3. Pulse volume

Pulse volume provides crude indications of stroke volume, being small in systolic heart failure and large in hyperkinetic heart disease

4. Radio-femoral delay

Press both radial and femoral artery at the same time and notice for pulse delay at femoral artery in comparison to radial artery, and usually observed in coarctation of the aorta

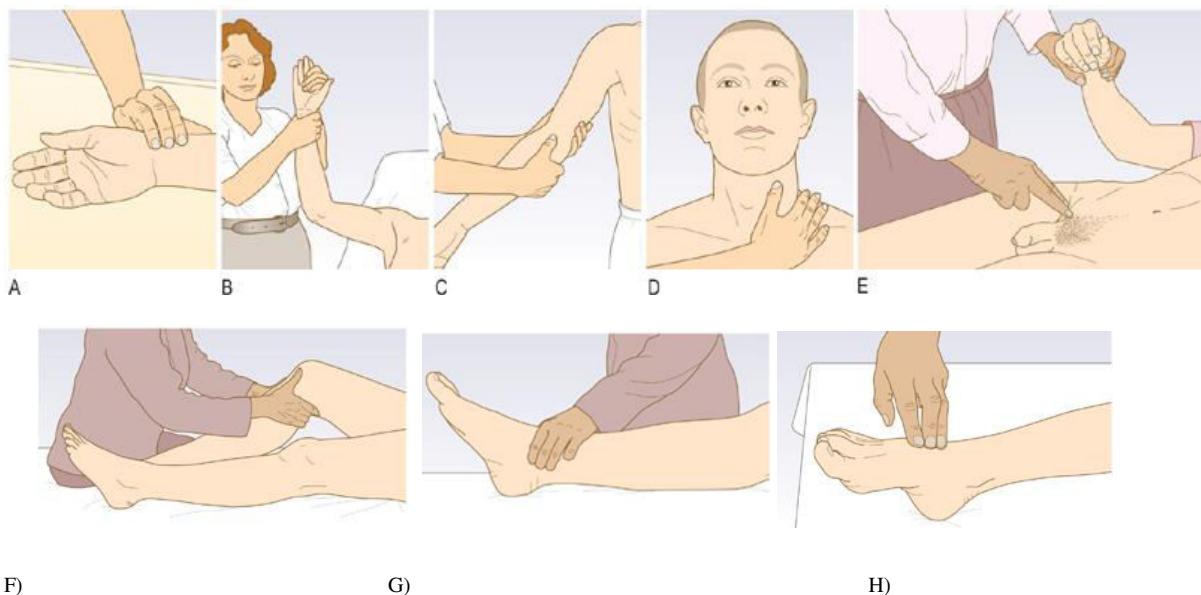
5. Condition of the vessel wall

Thickening (cording) or tortuosity of vessel wall (arteriosclerosis) commonly detected in the arteries of elderly individuals. It doesn't indicate presence of luminal narrowing due to atherosclerosis.

Table 6.4: Surface markings of the arterial pulses

Artery	Surface marking
Radial	At the wrist, lateral to the flexor carpi radialis tendon
Brachial	In the antecubital fossa, medial to the biceps tendon
Carotid	At the angle of the jaw, anterior to the sternocleidomastoid muscle
Femoral	Just below the inguinal ligament, midway between the anterior superior iliac spine and the pubic symphysis (the mid-inguinal point). It is immediately lateral to the femoral vein and medial to the femoral nerve
Popliteal	Lies posteriorly in relation to the knee joint, at the level of the knee crease, deep in the popliteal fossa
Posterior tibial	Located 2 cm below and posterior to the medial malleolus, where it passes beneath the flexor retinaculum between flexor digitorum longus and flexor hallucis longus
Dorsalis pedis	Passes lateral to the tendon of extensor hallucis longus and is best felt at the proximal extent of the groove between the first and second metatarsals. It may be absent or abnormally sited in 10% of normal subjects, sometimes being 'replaced' by a palpable perforating peroneal artery

© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th ed



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 6.4 Techniques of palpating arterial pulses

A) Radial pulse B) Feeling for collapsing radial pulse as in aortic regurgitation C) Brachial pulse medial to biceps tendon D) Examining carotid pulse with thumb E) Femoral pulse midway between the anterior superior iliac spine and the pubic tubercle. Notice for checking radio-femoral delay F) Feeling for popliteal pulse G) Posterior tibial pulse H) Examining dorsalis pedis pulse

Jugular venous pressure (JVP)

The JVP is best assessed from pulsations in the right internal jugular vein, which is directly in line with the superior vena cava and right atrium. The dominant movement of the JVP is inward, coinciding with 'X' descent.

Technique:

. Position the patient

Elevate head of bed at 45° to maximize visibility of the jugular venous pulsation in the lower half of the neck

Turn patient's head slightly away from the side you are inspecting

Use tangential (oblique) lighting and identify the pulsation of internal jugular vein

. Identify the highest point of pulsation of internal jugular vein

. Measure the vertical distance between the highest point of jugular pulsation and sternal angle with a metered ruler and then place tongue blade at an exact right angle to the ruler and read the vertical distance on the ruler

The JVP reflects central venous or right atrial pressure and, indirectly, right ventricular function

The normal upper limit is 4 cm vertically above sternal angle. This is about 8-9 cm above right atrium, corresponding to a JVP of 8-9 cm H₂O (7 mmhg)

JVP > 4 cm vertically above sternal angle is considered as elevated JVP

NB: If the internal jugular vein pulsation is not visible, measure the vertical distance of the point above which the external jugular veins appear to be collapsed from the sternal angle

. Observe the amplitude and timing of the jugular venous pulsation

JVP has 2 or 3 peaks (a, c, v waves) and 2 troughs (x, y waves)

Types of jugular venous waves

‘a’ wave- atrial contraction; ‘c’ wave-bulging of closed tricuspid valve towards the right atrium during right ventricular systole; ‘x’ wave-atrial relaxation; ‘v’ wave-atrial filling, and ‘y’ wave-atrial emptying

‘a’ wave just precedes S₁ and the carotid pulse, the ‘x’ descent seen at the systolic collapse, the ‘v’ wave almost coincides with S₂ and the ‘y’ descent follows early diastole

Positive abdomino-jugular reflex test: An increase in JVP during firm, mid-abdominal compression for 10 seconds followed by a rapid drop in JVP of 4 cm on release of the compression

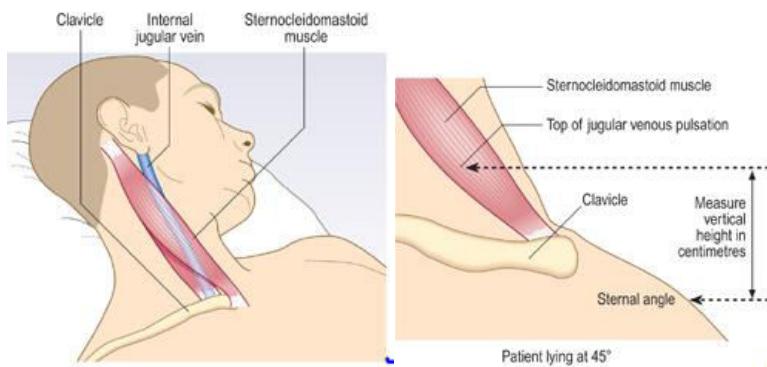
Positive abdomino-jugular (hepato-jugular) reflex is usually observed in heart failure

Differences between jugular and carotid pulsation

Internal Jugular Pulsations	Carotid Pulsations
Rarely palpable	Palpable
Soft, rapid, undulating quality, usually with two elevations and two troughs per heart beat	A more vigorous thrust with a single outward component
Pulsations eliminated by light pressure on the vein(s) just above the sternal end of the clavicle	Pulsations not eliminated by this pressure
Level of the pulsations changes with position, dropping as the patient becomes more upright.	Level of the pulsations unchanged by position
Level of the pulsations usually descends with inspiration.	Level of the pulsations not affected by inspiration

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 6.5 Differences between jugular and carotid pulsation

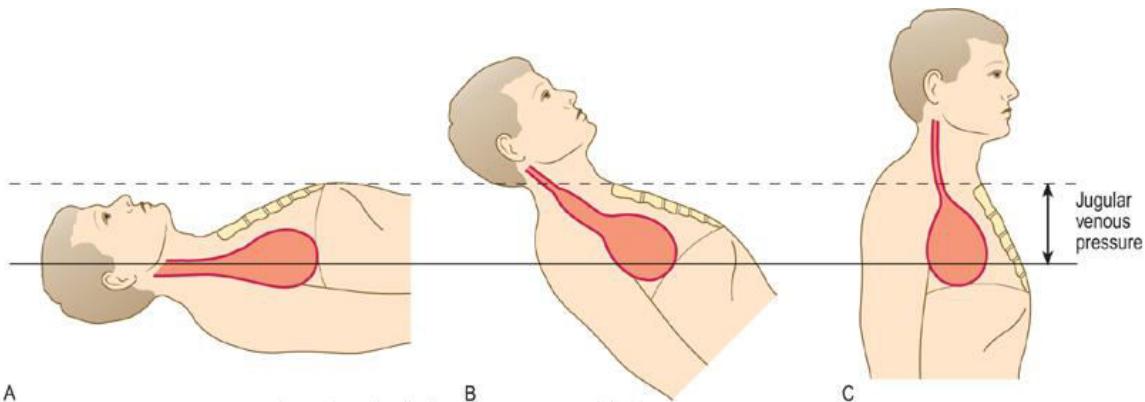


A) Inspecting JVP from the side

B) Measuring the height of JVP

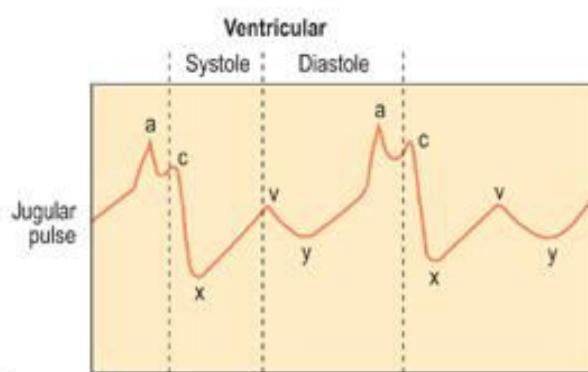
© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 6.5 Technique of estimating height of JVP



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 6.6 Level of JVP in normal subject A) Supine: Jugular vein is distended, pulsation not visible B) Reclining at 45°: Point of transition between distended and collapsed vein can usually be seen to pulsate just above the clavicle C) Upright: Upper part of vein collapsed and transition point obscured by sternum



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 6.7 Wave forms of JVP and their interpretation

‘a’ wave: atrial contraction, ‘C’ wave: atrial bulging of closed tricuspid valve, ‘V’ wave: atrial filling, ‘X’ descent: atrial relaxation, ‘y’ descent: atrial emptying (rapid ventricular filling)

Abnormal wave forms of JVP

Kussmaul's sign: a paradoxical rise of JVP on inspiration. It is seen in pericardial tamponade, severe right ventricular failure and restrictive cardiomyopathy

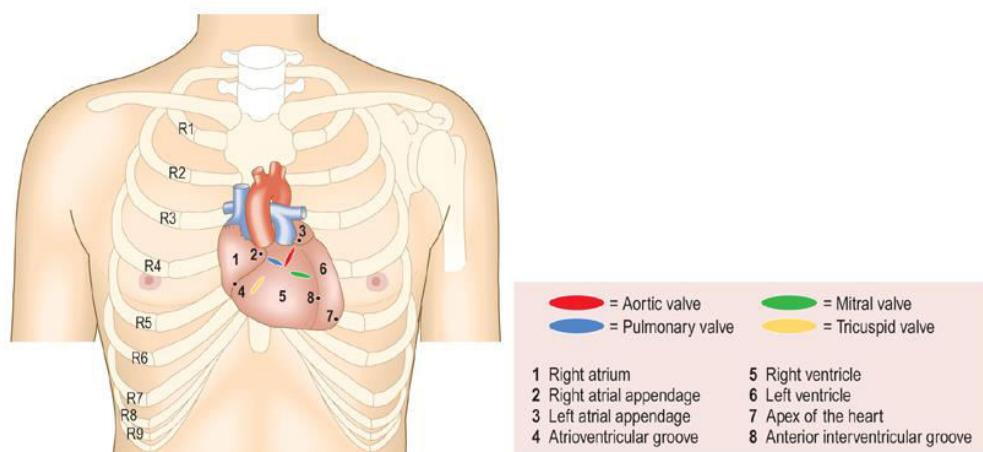
Prominent 'a' wave: seen in any condition with delayed or restricted right ventricular filling, e.g. pulmonary hypertension or tricuspid stenosis

Cannon waves: Irregular cannon waves are seen in complete heart block and are due to atrioventricular dissociation. Regular cannon waves occur during junctional bradycardias and some ventricular and supraventricular tachycardias

'cv' wave: a characteristic fusion of the 'c' and 'v' waves resulting in an increased wave and associated with a pulsatile liver. It is seen in tricuspid regurgitation.

Precordial examination

The precordium is the area on the front of the chest which relates to the surface anatomy of the heart



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th ed

Fig 6.8 Surface anatomy of the heart

Technique of precordial examination

Inspection

Is there precordial bulging?

Presence of precordial bulging indicates chronicity of valvular heart disease

Is the precordium active or not?

Quiet precordium is noticed in chick chest wall, massive pericardial effusion or dilated cardiomyopathy

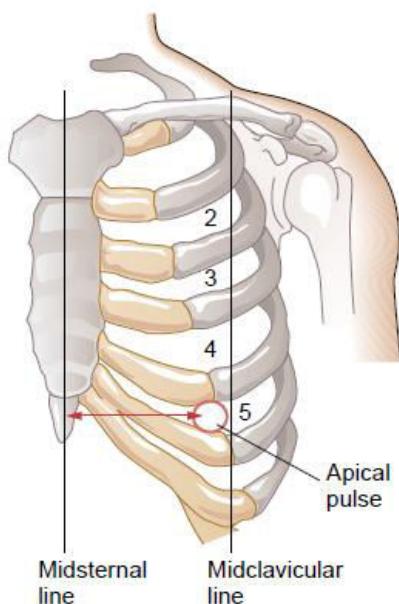
Is the apical impulse visible or not? If visible, determine the location in intercostal space in relation to left mid-clavicular line (point out with a single finger!)

The apex beat is defined as the lowest and most lateral point at which the cardiac impulse can be palpated

Apical impulse is due to the recoil of heart as blood is ejected

The normal apical impulse is located in the 5th left inter costal space at or medial to mid-clavicular line (halfway between the suprasternal notch and acromioclavicular joint)

Displaced apical impulse is encountered in left ventricular enlargement, chest wall deformity (scoliosis), or mediasinal shift



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 6.9 Location of apical impulse in normal precordium

Palpation

- . Localize apical impulse
- . Localize and characterize point of maximal impulse (PMI) (location, diameter, amplitude, duration)

Character of PMI

- . Location: usually located at site of apical impulse under normal circumstance
- . Diameter: 1-2.5 cm in diameter, occupies only 1 interspace
- . Amplitude: brisk and tapping
- . Duration: occupies first 2/3 of systole

Technique

a. Apical impulse

- . Localize the left 2nd intercostal space at lateral to sternal angle
- . Count down the interspaces, and point out the most laterally and down ward located cardiac impulse

b. PMI

Technique

- . Put the tip of index, middle and ring fingers over the site of PMI

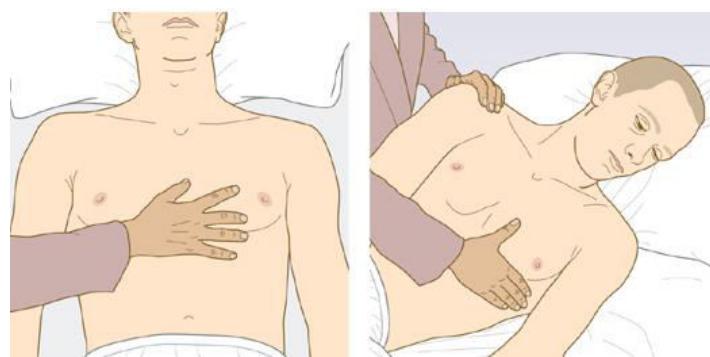
Is it localized or diffuse?

Diffuse PMI occupies >2.5 cm in diameter or occupies more than one interspace

Is it sustained or not?

Sustained PMI occupies more than 2/3 of systole

Sustained and thrusting PMI is noticed in concentric left ventricular hypertrophy as in hypertensive heart disease or aortic valve stenosis, while non-sustained and tapping PMI is observed in mitral valve stenosis



- a) Palpation of cardiac impulse
- b) localizing apex beat (left lateral position if required)

© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 6.10 Localizing apical impulse

Heave/Lifts

Heave is a palpable impulse that lifts examiners hand noticeably and usually noticed in enlarged ventricles

Technique

. Put ulnar border of the hand over left sternal area and look for lift or heave

Left parasternal heave suggests right ventricular hypertrophy

Thrill

Tactile equivalent of a murmur, and feels similar to a vibrating mobile telephone

Technique

Palpate the apex, left sternal border, and the neck with palm of examining hand, and feel for thrill as “purring of a cat”

Timing of the thrill is required (Is it systolic or diastolic?). If the thrill coincides with the carotid pulse, it is systolic thrill.

Shock (palpable heart sound)

Feel for palpable heart sounds at apex and base of heart

Palpable S₂ at pulmonic area occurs in pulmonary hypertension while palpable S₁ at mitral area occurs in mitral stenosis

Percussion

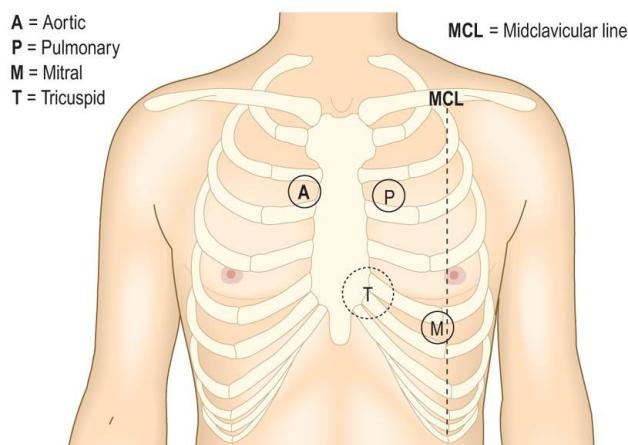
Percussion adds little to inspection and palpation to demarcate cardiac size

Auscultation

The diaphragm and bell of stethoscope permit appreciation of high- and low-pitched auscultatory sounds respectively

Location of heart valves (mitral, tricuspid, pulmonary and aortic valves)

- . Cardiac apex-mitral area
- . Left lower sternal border-Tricuspid area
- . Left upper sternal border-pulmonic area
- . Right upper sternal border-aortic area



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 6.11 Location of heart sounds: mitral, tricuspid, pulmonic and aortic area

Heart sounds: S₁ (Lub) and S₂ (Dub)

First heart sound (S₁) corresponds to atrioventricular valves closure at the onset of systole, and is best heard at the apex of heart

Second heart sound (S₂) corresponds to outlet valves closure following ventricular ejection, and is best heard at base of heart

Use the diaphragm of stethoscope to listen 1st and 2nd heart sounds

NB: Opening of any normal valve is not audible

- . Notice intensity of heart sounds

Increased intensity of S₁ at mitral area occurs in mitral stenosis, tachycardia and pregnancy, while reduced intensity of S₁ occurs in mitral regurgitation and mitral valve prolapse

Increased intensity of S₂ at pulmonic area occurs in pulmonary hypertension

- . Listen for physiologic splitting of S₂ (A₂P₂)

It occurs as left ventricle contracts earlier than right ventricle, and best heard at left sternal border (lub-dub/dub)

Enhanced physiologic splitting of S₂ (A₂...P₂) occurs in right bundle branch block, pulmonary hypertension, etc...

Fixed splitting of S₂ (A₂-P₂) occurs in atrial septal defect (ASD)

Reversed splitting of S₂ (P₂...A₂) occurs in left bundle branch block, aortic stenosis, hypertensive heart disease, hypertrophic cardiomyopathy, etc...

Added heart sounds (S₃ and S₄)

Third (S₃) and fourth (S₄) heart sounds are low-frequency sounds, which occur in ventricular diastole

Use the bell of stethoscope to listen 3rd and 4th heart sounds

S₃ (lub-dub-dum) corresponds to an abrupt deceleration of inflow across the atrioventricular valve

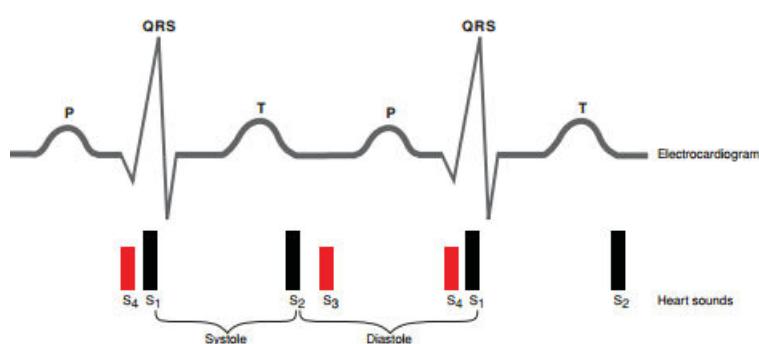
S₄ (da-lub-dub) is noticed in vigorous atrial contraction against increased ventricular end diastolic stiffness

- . Notice for the presence of added heart sound

Physiologic S₃ occurs in young adults <40 yrs of age and pregnancy

Pathologic S₃ often noticed in anemia, thyrotoxicosis, mitral regurgitation, etc...

S₄ is usually pathological and occurs in stiff ventricle due to hypertensive heart disease, aortic stenosis or hypertrophic cardiomyopathy



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 6.12 Heart sounds in relation to electrical activity of the heart

Heart murmurs

Heart murmurs are vibrations set up in the blood stream as a result of turbulent blood flow in the heart and great vessels

Characterizing the heart murmur

- . Location of maximal intensity
- . Timing-systolic, diastolic or continuous
- . Intensity/grading of the murmur
- . Radiation
- . Shape (configuration)
- . Pitch and quality

Location of maximal intensity

Signify origin of a murmur

eg. Murmur of mitral stenosis is located with maximal intensity at mitral area

Timing

Identify whether the murmur is systolic, diastolic or continuous in comparison to carotid pulse

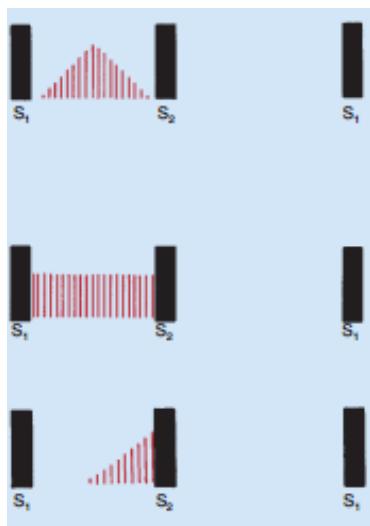
Technique

- . Slightly compress the right carotid artery at the level of cricoid cartilage, and time the murmur whether systolic or diastolic

Systolic murmurs coincide with carotid pulse, while the diastolic murmurs do not

Systolic murmurs

- . Mid-systolic murmur- begin after S₁ and stops before S₂ eg. Murmur of aortic stenosis (AS)
- . Pansystolic murmur- starts with S₁ and stops at S₂ eg. Murmur of mitral regurgitation (MR)
- . Late systolic murmur- starts in mid or late systole and persists up to S₂ eg. Murmur of mitral valve prolapse (MVP)

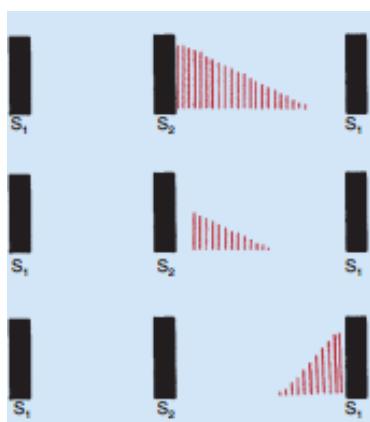


© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 6.13 Systolic murmurs: Midsystolic murmur of AS (upper), pansystolic murmur of MR (middle) and late systolic murmur of mitral valve prolapse (lower)

Diastolic murmur

- . Early diastolic murmur- starts after S₂ and fades into silence before next S₁ eg. Murmur of aortic regurgitation (AR)
- . Mid diastolic murmur- starts after S₂ and fade away eg. Murmur of mitral stenosis (MS)
- . Late diastolic (presystolic) murmur- starts late in diastole and continuous up to S₁ eg. Murmur of mitral or tricuspid stenosis in sinus rhythm

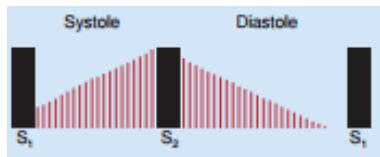


© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 6.14 Diastolic murmurs: Early diastolic murmur of AR (upper), Mid diastolic murmur of MS (middle), and late diastolic or presystolic murmur of MS (lower)

Continuous murmur

Begin in systole, peak at S₂, and continue into all or part of diastole eg. Murmur of PDA (patent ductus arteriosus)



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 6.15 Continuous murmur of PDA

Grading of Intensity or loudness of a murmur

The loudness of a murmur reflects degree of turbulence (volume and velocity of flow) and not the severity of the cardiac lesion

Grade I- Faint murmur, heard only with special efforts

Grade II- Quiet but heard murmur

Grade III- Easily heard murmur

Grade IV- Loud murmur, with a thrill

Grade V- Very loud, heard with a stethoscope partly off the chest

Grade VI- Extremely loud, heard with out a stethoscope

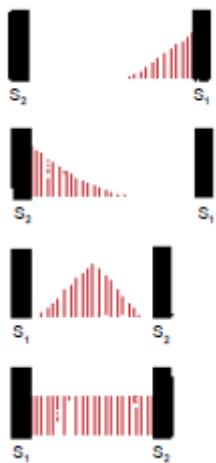
Shape (configuration)

Crescendo- murmur grows louder eg. Pre-systolic murmur of MS

Crescendo-decrescendo- Murmur that grows louder and then fall eg. Mid-systolic murmur of AS

Decrescendo- murmur grows softer and slowly falls eg. Early diastolic murmur of AR

Plateau- murmur has same intensity throughout eg. Pansystolic murmur of MR



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 6.16 Shape (configuration) of the murmurs: Crescendo murmur (presystolic murmur of MS), decrescendo (early diastolic murmur of AR), crescendo-decrescendo (midsystolic murmur of AS), plateau (pansystolic murmur of MR) (from top to bottom)

Radiation

Signify direction of blood flow

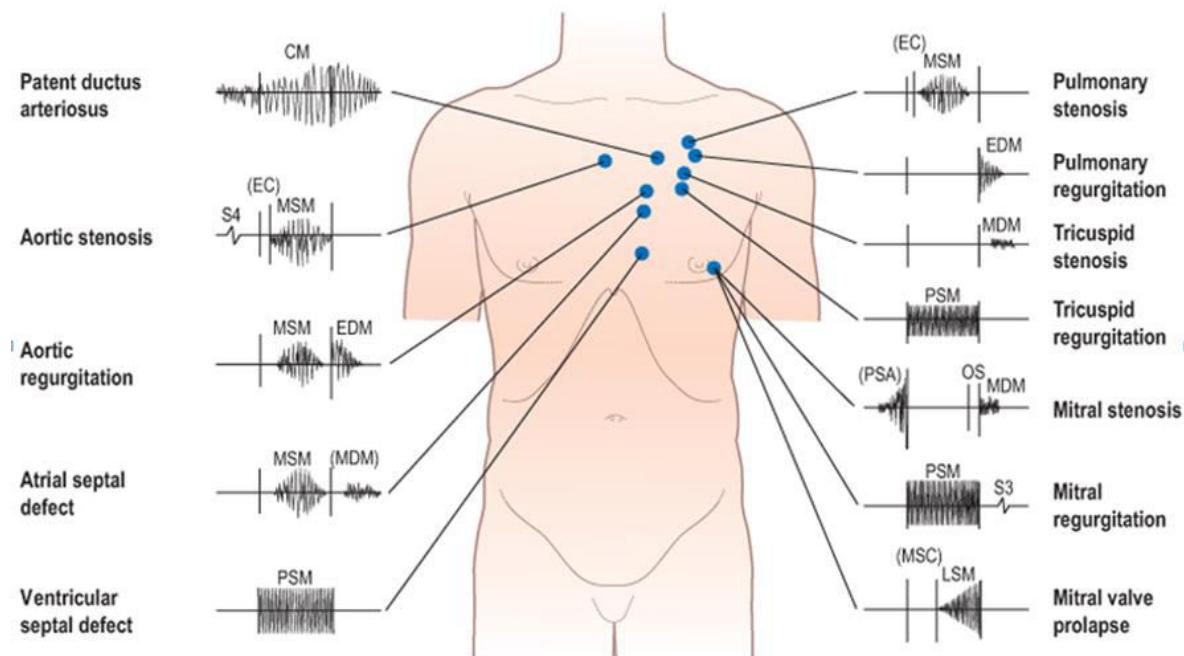
eg. Murmur of MR radiates to left axilla , murmur of AS radiates to neck, murmur of TR radiates to epigastrium, etc...

Pitch

High, medium or low

Quality

Blowing, harsh, rumbling, musical, etc...



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 6.17 Location, timing and configuration of murmurs (CM-continuous murmur, MSM-mid systolic murmur, EDM-early diastolic murmur, PSM-pansystolic murmur, LSM-late systolic murmur, MDM-mid diastolic murmur, PSA-presystolic accentuated murmur)

Special techniques

Valsalva maneuver

Squatting (release phase of Valsalva) and standing (straining phase of Valsalva)

Squatting position increases venous return, left ventricular volume and arterial blood pressure, standing do the opposite.

Table 6.6 Maneuvers to identify systolic murmurs

Maneuver	Cardiovascular Effect	Effect on Systolic Sounds and Murmurs		
		Mitral Valve Prolapse	Hypertrophic Cardiomyopathy	Aortic Stenosis
Standing; Strain Phase of Valsalva	Decreased left ventricular volume from ↓ venous return to heart	↑ prolapse of mitral valve	↑ outflow obstruction	↓ blood volume ejected into aorta
	Decreased vascular tone: ↓ arterial blood pressure; ↓ peripheral vascular resistance	Click moves earlier in systole and murmur lengthens	↑ intensity of murmur	↓ intensity of murmur
Squatting; Release of Valsalva	Increased left ventricular volume from ↑ venous return to heart	↓ prolapse of mitral valve	↓ outflow obstruction	↑ blood volume ejected into aorta
	Increased vascular tone: ↑ arterial blood pressure; ↑ peripheral vascular resistance	Delay of click and murmur shortens	↓ intensity of murmur	↑ intensity of murmur

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Hand grip exercise

Loudness of murmurs of MR and AR increase with hand grip exercise

Respiration

Intensity of left-sided murmurs increase with expiration, and intensity of right-sided murmurs increase with inspiration

Other heart sounds

Pericardial friction rub

High-pitched scratching sound audible at any part of cardiac cycle; heard best at left lower sternal border in maintained expiration and patient leaning forward. It is observed in acute pericarditis.

Pericardial knock

Early diastolic knock sound caused by sudden halt in blood flow into the heart during diastolic filling

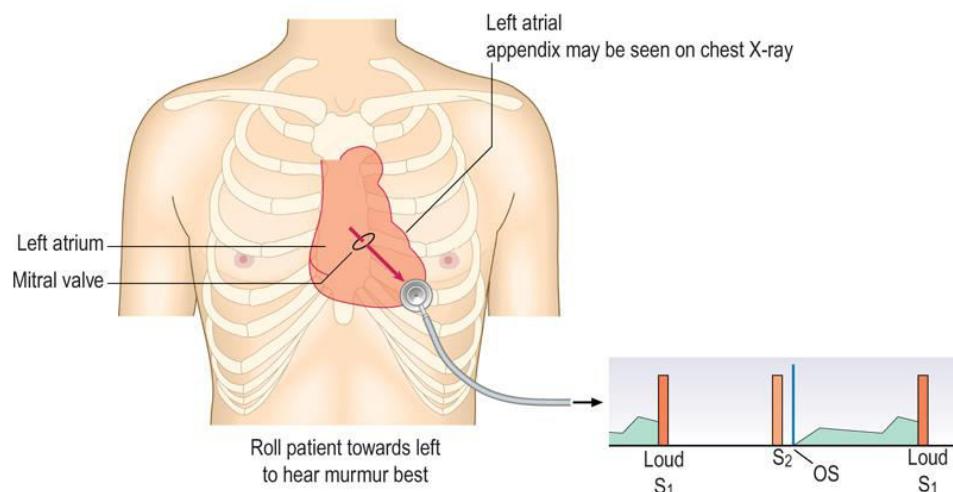
It is observed in constrictive pericarditis

Opening snap

High-pitched snapping sound heard in early diastole, caused by forceful opening of stenosed mitral valve by increased left atrial pressure

Characteristics of common cardiac valvular lesions

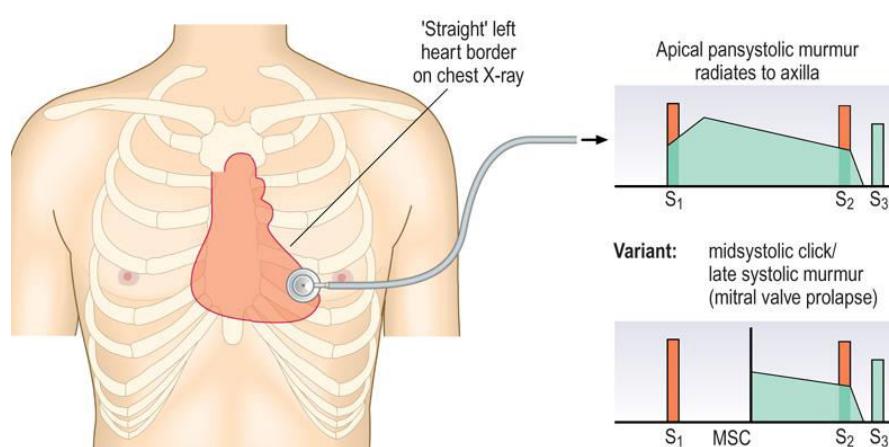
1. Mitral stenosis: Accentuated S₁; opening snap following S₂; low-pitched, rumbling mid-diastolic murmur limited to apex, increased intensity of murmur with exercise and left lateral positioning



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 6.18 Murmur of mitral stenosis

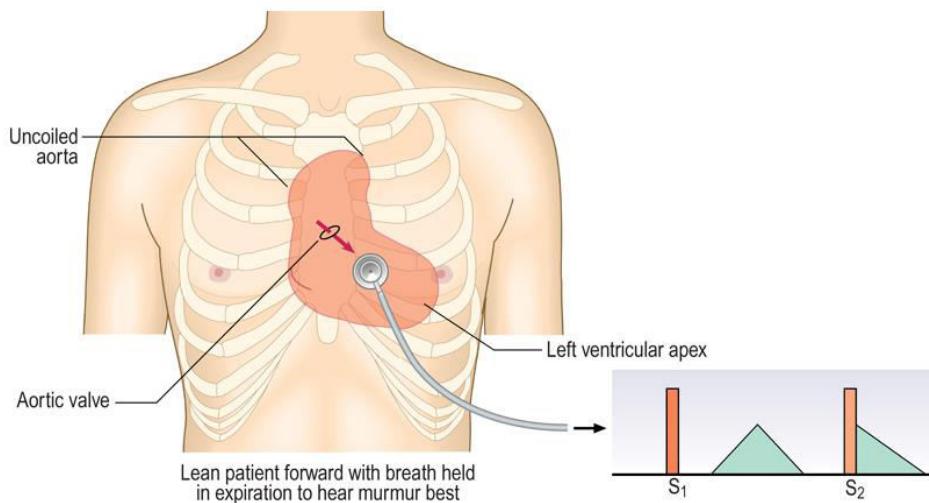
2. Mitral regurgitation: lateral and downward displaced apical impulse; muffled S₁; S₃ sound; medium to high-pitched, blowing pansystolic murmur, radiating to axilla or base of heart



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 6.19 Murmur of mitral regurgitation

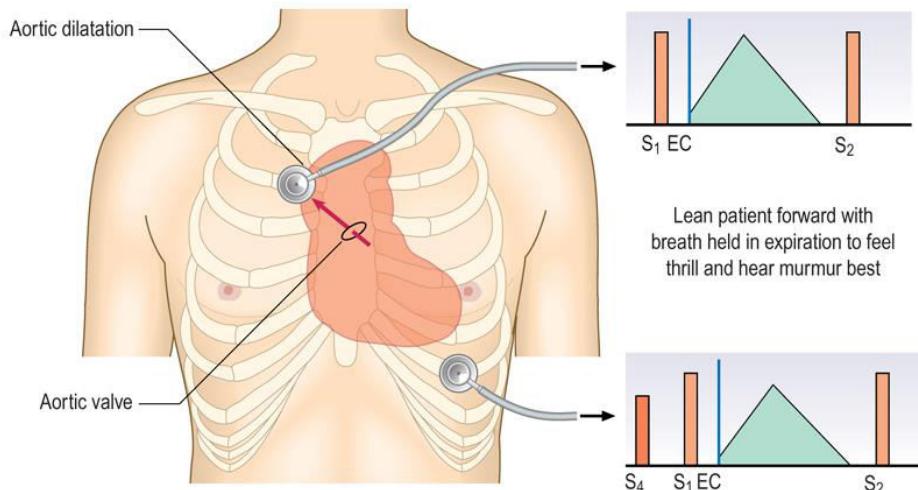
3. Aortic regurgitation: ‘Water-hammer’ pulse with wide pulse pressure, pistol-shot at femoral artery, lateral and down ward displaced apical impulse; high-pitched, blowing early diastolic decrescendo murmur at the 2nd to 4th left interspace (Erb’s point), accentuated by leaning forward and breath held at expiration; Austin-Flint (mid diastolic) murmur at the apex due to back regurgitant flow; and functional mid-systolic flow murmur at the aortic area



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 6.20 Murmur of aortic regurgitation

4. Aortic stenosis: Anacrotic arterial pulse, thrusting and sustained apical impulse; low- pitched, rasping, midsystolic, crescendo-decreasedo murmur at aortic area radiating to the neck (carotid shudder)



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 6.21 Murmur of aortic stenosis

5. Tricuspid regurgitation: Prominent V wave of JVP; low to high-pitched, blowing, pansystolic murmur at the left lower sternal border accentuated by deep inspiration (positive caravello's sign)

Leg edema

Interstitial tissue absorbs upto 5 litres of fluid (up to 10% weight gain) before pitting edema appears

Pedal and pretibial pitting edema occurs in congestive heart failure, varicose veins and DVT (deep vein thrombosis)

Ankle edema of cardiac origin is usually symmetrical and worsens in the evening and improves early in the morning

Technique:

Palpate behind the medial malleolus and the distal shaft of the tibia, and dorsum of foot for pitting edema by gently compressing the area for at least 15 seconds with the thumb

Edema of cardiac origin is symmetrically pitting (the skin is indented and only slowly refills)

Grading of edema

a. Grading by body sites

Grade 1- pedal and pretibial edema

Grade 2- Edema involving leg and thigh

Grade 3- Edema involving abdominal wall and pre-sacrum

Grade 4- Anasarca (generalized edema including serosal fluid accumulation)

b. Grading by time to refill the pitting edema

Grade 1- Refill time for pitting edema takes < 5 seconds

Grade 2- Refill time for pitting edema takes 5-10 seconds

Grade 3- Refill time for pitting edema takes 10-15 seconds

Grade 4- Refill time for pitting edema takes > 15-20 seconds

CHAPTER SEVEN

Gastrointestinal system

Learning objective

At the end of this lesson, the student should be able to:

1. Mention main symptoms in gastrointestinal disease
2. List common causes of chronic diarrhea
3. Show techniques of abdominal examination
4. Perform examination of palpable abdominal swelling

History taking

Patients with gastrointestinal problem present with one or more of the following symptoms

Dysphagia

Odynophagia

Heart burn

Nausea and vomiting

Abdominal pain

Abdominal distension

Diarrhea

Constipation

Hematemesis

Melena

Weight loss

Jaundice

Dysphagia

Dysphagia is difficulty in swallowing

Ask:

- . Does food or drink stick when you swallow (dysphagia)? If Yes, then
 - . How long?
 - . Is dysphagia painful or painless?
 - . At what level does food stick?

- . Is the dysphagia for solids or liquids or both?
- . Is dysphagia intermittent or progressive?

Difficulty in initiating swallowing with fluid regurgitation into the nose, or choking on trying to swallow indicates pharyngeal dysphagia due to neurologic disorders

Food sticking in the lower retrosternal area indicates lower esophageal obstruction

Swallowing difficulty more to liquids than solids suggest esophageal motor disorders like achalasia and diffuse esophageal spasm

Progressive dysphagia early to solids, and then to liquids indicates esophageal stricture or cancer

Intermittent dysphagia within the first few swallowing of food occurs in diffuse esophageal spasm

Causes of dysphagia

1. Mechanical

- a. Extrinsic
 - . Retrosternal goiter
 - . Mediastinal masses
- b. Intrinsic
 - . Esophageal stricture
 - . Esophageal cancer
 - . Esophageal web

2. Neuromuscular motility disorders

- . Achalasia
- . Diffuse esophageal spasm
- . Scleroderma
- . Neurologic diseases: Motor neuron disease (amyotrophic lateral sclerosis), myasthenia gravis

Odynophagia

Odynophagia is pain during swallowing

Causes of odynophagia

- . Infectious esophagitis- herptic and cytomegalovirus ulcers, and esophageal candidiasis in HIV infected patients
- . Esophageal peptic ulceration

- . Chemical esophagitis eg. caustic soda damage
- . Drug allergies eg. sulfonamides, cytotoxic chemotherapeutics, etc...
- . Immunologic dermatological diseases eg. pemphigus vulgaris, dermatitis herpetiformis, etc...

Dyspepsia

Substernal burning pain or indigestion

Clusters of symptoms are used to classify dyspepsia, such as reflux-like dyspepsia (heartburn-predominant dyspepsia), ulcer-like dyspepsia (epigastric pain relieved with food or antacids) and dysmotility-like dyspepsia (nausea, belching, bloating and early satiety)

Nausea and vomiting

Nausea is involuntary effort to vomit

Vomiting is expulsion of gastric contents through the mouth

Describe about mode of onset, timing, content of vomitus, and projectile or not

- . Mode of onset (acute or chronic)

Acute onset occurs in food poisoning, raised intracranial pressure or bowel obstruction

Chronic onset occurs in pregnancy, medications (digoxin, dopamine agonists, chemotherapy), bowel motor diseases (eg. gastroparesis in diabetics) or pyloric stenosis due to scarred peptic ulcer disease

- . Timing of vomiting (related to meal time, early morning or late evening)

Vomiting after one hour of meal is typical of gastric outlet obstruction or gastroparesis

Early morning vomiting before eating occurs in pregnancy or raised intracranial pressure

- . Content of vomitus (bile-stained, blood-stained or faeculent)

Vomiting of bile-stained material rules out obstructive gastropathy

Vomiting of blood suggests upper gastrointestinal bleeding

Vomiting of faeculent material suggests intestinal obstruction

- . Projectile or not

Projectile vomiting of non-bilious old food, which relieves dyspeptic abdominal pain suggests gastric outlet obstruction (pyloric stenosis)

Abdominal pain

Somatic pain from inflamed parietal peritoneum or abdominal wall is well localized, while visceral pain from distended hollow organs, excessive muscle contraction or mesentery traction is poorly localized (autonomic).

Pain from foregut (stomach, pancreas, hepatobiliary structures) localizes to epigastric area, pain solely from midgut (small bowel and proximal large colon) is felt periumbilical and pain from hindgut (large bowel) localizes to suprapubic area.

Characterize abdominal pain- Site, mode of onset, course and duration, character and pattern, areas of radiation, aggravating and relieving factors (SOCRATES)

Common causes of abdominal pain

- . Acute appendicitis
- . Acute cholecystitis
- . Acute pancreatitis
- . Diverticulitis
- . Peptic ulcer disease
- . Intestinal obstruction
- . Renal colic (calculi)
- . Mesenteric arterial occlusion (ischemic bowel disease)

Table 7.1 Common causes of acute abdomen

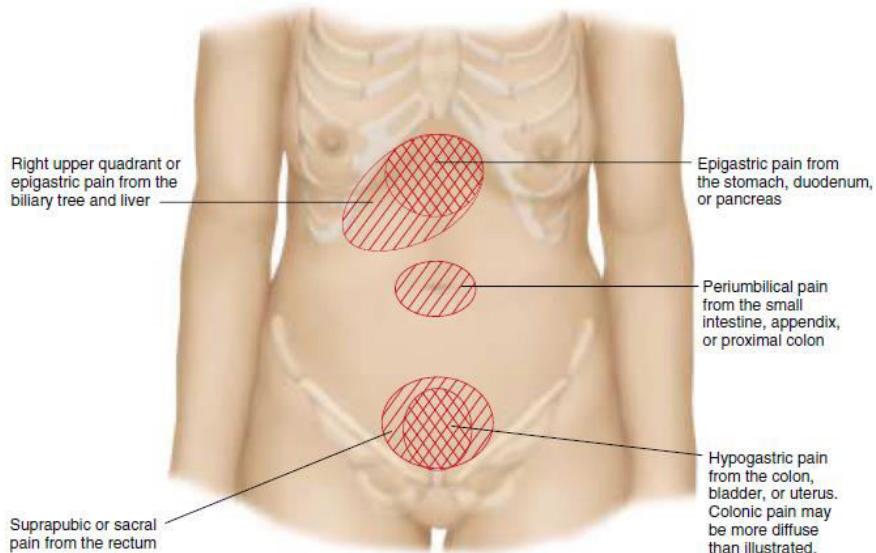
	Peptic ulcer	Biliary colic	Acute pancreatitis	Renal colic
Site	Epigastrium	Epigastrium/right hypochondrium	Epigastrium/left hypochondrium	Loin
Onset	Gradual	Rapidly increasing	Sudden	Rapidly increasing
Character	Gnawing	Constant	Constant	Constant
Radiation	Into back	Below right scapula	Into back	Into genitalia and inner thigh
Timing				
Frequency/periodicity	Remission for weeks/months	Able to enumerate attacks	Able to enumerate attacks	Usually a discrete episode
Special times	Nocturnal and especially when hungry	Unpredictable	After heavy drinking	Following periods of dehydration
Duration	½-2 hours	4-24 hours	>24 hours	4-24 hours
Exacerbating factors	Stress, spicy foods, alcohol, NSAIDs	Unable to eat during bouts	Alcohol Unable to eat during bouts	
Relieving factors	Food, antacids, vomiting		Eased by sitting upright	
Severity	Mild to moderate	Severe	Severe	Severe

© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Table 7.2 Non-gastrointestinal causes of abdominal pain

Disorder	Clinical features
Myocardial infarction	Epigastric pain without tenderness Angor animi (feeling of impending death), hypotension, cardiac arrhythmias
Dissecting aortic aneurysm	Tearing interscapular pain Angor animi, hypotension Asymmetry of femoral pulses
Acute vertebral collapse	Lateralized pain restricting movement Tenderness overlying the involved vertebra
Cord compression	Pain on percussion of thoracic spine Hyperesthesia in dermatomal distribution Spinal cord signs
Pleurisy	Lateralized pain on coughing Chest signs, e.g. pleural rub
Herpes zoster	Hyperesthesia in dermatomal distribution Vesicular eruption
Diabetic ketoacidosis	Cramp-like pain, vomiting, air hunger Tachycardia, ketotic breath
Salpingitis or tubal pregnancy	Suprapubic and iliac fossa pain, localized tenderness, nausea, vomiting, fever
Torsion of the testis/ovary	Lower abdominal pain Nausea, vomiting, localized tenderness

© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 7.1 Abdominal pain and organ of origin

Abdominal distension

Causes of abdominal distension

5 F's: Flatus, faeces, fluid, fetus, fat, functional (bowel obstruction, ascites, pregnancy, obesity, irritable bowel syndrome)

Causes of Ascites (excessive fluid in peritoneal cavity)

- . Hepatic cirrhosis with portal hypertension
- . Intra-abdominal malignancy with peritoneal spread
- . Congestive heart failure
- . Peritonitis eg. tuberculosis, acute pancreatitis
- . Hypoproteinemia eg. nephrotic syndrome, protein-losing enteropathy
- . Hepatic or portal vein occlusion
- . Constrictive pericarditis

Diarrhea

Passage of watery or loose stool > 3 times per day, or passage of large amount of stool >300 gram per day

Describe about mode of onset and duration, frequency of diarrhea/day, character (watery, loose stool, bloody, mucoid), associated symptoms (thirst, oliguria)

Ask:

- . Is the diarrhea acute, persistent or chronic?
- . Is it watery, unformed or semiformed?
- . Is the stool less frequent and voluminous as in small bowel disease?
- . Is the stool of less volume and excessively frequent as in large bowel disease?
- . Is there blood or mucus associated in stool?
- . Is there past medical history of GI surgery, medical diseases?
- . Is there family history of GI disorder? eg. Gluten enteropathy, Crohn's disease, etc...
- . Are there symptoms of systemic disease? eg. anorexia, weight loss, etc...

Classification of diarrhea based on duration

- . Acute diarrhea (<2 weeks)
- . Persistent diarrhea (2-4 weeks)
- . Chronic diarrhea (> 4 weeks)

Acute diarrhea

Almost all cases of acute diarrhea are caused by infectious agents like E.coli, Salmonella spp, Shigella spp, Rotavirus, Norwalk agents, etc...

Traveller's diarrhea: ~40% of tourists who travelled to endemic area develop traveller's diarrhea, most commonly caused by Entrotoxigenic E.coli, Campylobacter spp., Shigella spp. and Salmonella spp.

Chronic diarrhea

It warrants evaluation to exclude serious underlying pathology

Most common causes of chronic diarrhea are non-infectious

Causes of chronic diarrhea

1. Secretory causes (voluminous diarrhea and persists with fasting)
 - . Hormone-producing tumors (colorectal villous adenoma, VIPoma)
 - . Exogenous and endogenous laxatives
2. Osmotic causes (voluminous diarrhea and disappears with fasting)
 - . Osmotic laxatives
 - . Lactase deficiency
3. Steatorrheal causes

Steatorrhea is presence of >7 gm of fat in a 24-hr stool collection

Steatorrheal stool is characterized as fatty, pale colored, smelly voluminous stool that float in a toilet bowel and more difficult to flush away

Sites of malabsorption includes

- . Intra-luminal eg. Pancreatic exocrine deficiency, bacterial overgrowth syndrome, chronic liver disease
 - . Mucosal eg. Celiac disease, tropical sprue, etc...
 - . Post-mucosal eg. Primary and secondary lymphatic obstruction
4. Exudative causes (frequent, small volume of stool and may be associated with blood or mucus)
 - . Non-infectious causes-Inflammatory bowel disease (IBD), diverticulitis, colorectal cancer
 - . Infectious causes- Invasive viral, bacterial and parasitic infections, intestinal tuberculosis

Constipation

Normal frequency of bowel movement ranges from three times daily to once every three days.

Constipation is persistent, difficult, infrequent or seemingly incomplete defecation. Passage of formed stool <3 times per week

Constipation is due to impaired colon motility, mechanical bowel obstruction, impaired rectal sensation with no normal ‘call to stool’ or anorectal dysfunction impairing the process of evacuation (anismus)

Ask:

- . Is it recent onset or chronic (life long)?
- . How often the bowels empty each week?
- . How much time is spent straining at stool?
- . Is there associated abdominal pain, rectal bleeding, or anal pain on defecation?
- . Has there been any change in drug therapy?

Causes of constipation

1. Recent onset

- . Colonic obstruction- colonic cancer, colonic stricture, colonic diverticula
- . Anal sphincter spasm- anal fissure, painful haemorrhoids

2. Chronic onset

- . Inflammatory bowel disease
- . Medications (codeine, antidepressants, aluminium or calcium antacids, iron salts)
- . Endocrinopathies- hypothyroidism, hypercalcemia
- . Neurologic diseases- Parkinsonism, spinal cord injury
- . Psychiatric diseases-depression, eating disorders

Hematemesis

Hematemesis is vomiting of frank blood

It can be fresh and red or dark-brown resembling coffee ground

It usually results from bleeding in the gastrointestinal tract above the ligament of Treiz (duodenojejunal flexure)
Describe mode of onset; duration, frequency and severity of bleeding

Ask:

- . Did the vomitus contain fresh blood or coffee-ground matter?
- . Is there history of alcohol, aspirin/NSAID, or corticosteroid ingestion?
- . Was the hematemesis preceded by retching as in Mallory-Weiss tear?
- . Is there previous history of peptic ulcer disease or liver disease?

Major causes of hematemesis

- . Peptic ulcer disease
- . Esophageal varices
- . Mallory Weiss tear
- . Gastro-duodenal erosions
- . Erosive esophagitis
- . Malignancy- Gastric cancer

Melena

Tarry, foul smelling stool indicates bleeding above ileo-cecal valve

Melena results with as low as 60 ml of bleeding in the dastrointestinal tract

Hematochezia (rectal bleeding)

Passage of bright red or maroon blood from lower gastrointestinal bleeding

It usually occurs to bleeding from the sigmoid colon, rectum or anal canal

Common causes of rectal bleeding includes hemorrhoids, anal fissure, colorectal polyps, colorectal cancer, inflammatory bowel disease, ischemic colitis, diverticular disease or vascular malformation

Weight loss

Quantify weight loss in kg, over how long?

Clinically significant weight loss is defined as $\geq 5\%$ body weight loss over 6-12 months

Usual causes of weight loss

- . Lack of food intake (with associated symptoms of anorexia, vomiting or dysphagia)
- . Malabsorption syndrome
- . Systemic effects due to cancer, tuberculosis, inflammatory bowel disease, etc...

Jaundice

Yellowish discoloration of the skin and mucus membrane resulting from the deposition of bilirubin

The presence of scleral icterus indicates a serum bilirubin level ≥ 2.5 mg/dl (normal bilirubin level < 1.0 mg/dl)

Causes of hyperbilirubinemia

- a. Overproduction of bilirubin
- b. Impaired uptake, conjugation or excretion of bilirubin

An increase in unconjugated bilirubin in serum results from either overproduction, impairment of uptake or conjugation of bilirubin

An increase in conjugated bilirubin is due to decreased excretion into the bile ductules or back leakage of the pigment

Causes of unconjugated (indirect) hyperbilirubinemia

- . Hemolysis- hereditary or acquired hemolytic disorders
- . Inherited disorders- Gilbert's syndrome, Crigler-Najjar syndrome

Causes of conjugated (direct) hyperbilirubinemia

a. Hepatocellular Pattern

- ALT/AST increase out of proportion to alkaline phosphatase
- . Viral hepatitis
 - . Drug-induced hepatitis
 - . Autoimmune hepatitis
 - . Alcoholic hepatitis

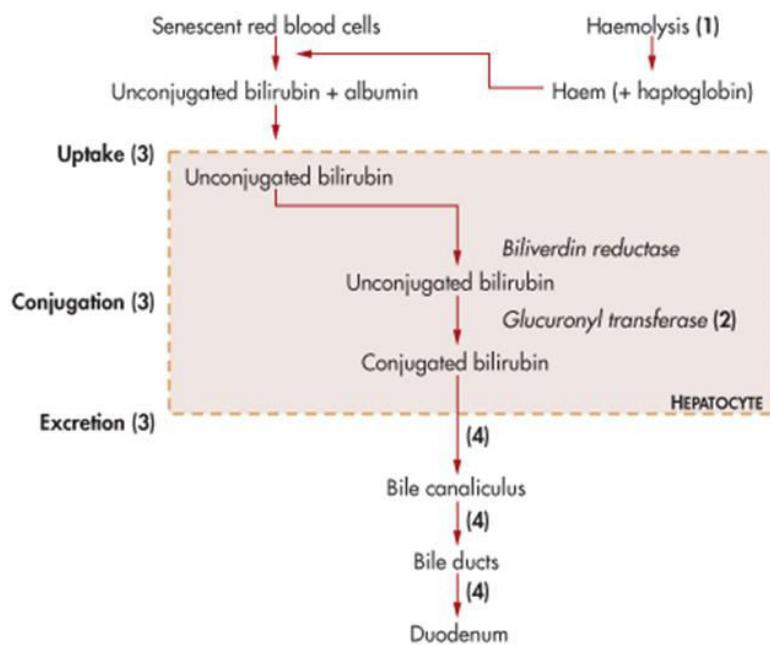
b. Cholestatic pattern

Alkaline phosphatase increase out of proportion to ALT/AST

. Intrahepatic cholestasis eg. Primary biliary cirrhosis, cholestasis of pregnancy, vanishing bile duct syndrome

. Extrahepatic cholestasis: Greenish-yellow discolored sclera, pruritis with excoriation marks and clay-colored stool

Eg. Primary sclerosing cholangitis, choledocholithiasis, cholangiocarcinoma, biliary duct stricture, ampullary stenosis, pancreatic head tumor



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 7.2 Metabolism of bilirubin: Uptake, conjugation and excretion of bilirubin: Increased haemolysis (1) overwhelms the hepatocytes' ability to conjugate bilirubin leading to increased serum levels of unconjugated bilirubin. Low levels of glucuronyl transferase (2) (e.g. Gilbert's disease) cause decreased conjugation. Hepatocellular dysfunction (3) causes decreased uptake, conjugation and excretion with increases of unconjugated bilirubin and conjugated bilirubin. Post-hepatic obstruction (4) from stones or tumor prevents passage of bilirubin through the bile ducts into the bowel, leading to increased serum levels of conjugated bilirubin

Abdominal examination

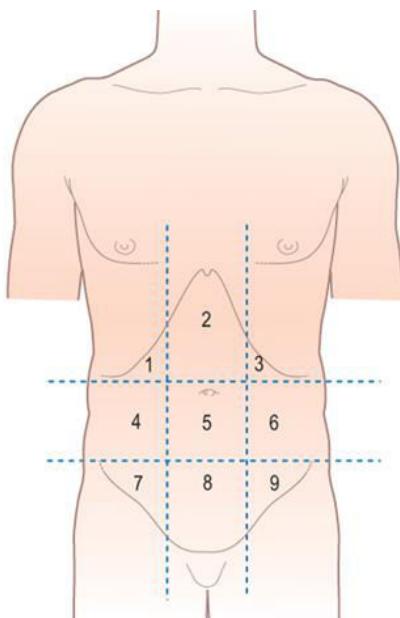
- . Good lighted room and coach is required
- . Make the patient comfortable in the supine position
- . The patient should keep the arms at the sides
- . Exposure of the abdomen from the xiphoid process to the mid thigh; covering the genitalia is required
- . The examiner should have warm hands, warm stethoscope and short finger nails
- . Approach the patient slowly and avoid quick, unexpected movements
- . If the patient is not at ease, distract her/him with conversation
- . Monitor your examination by watching the patient's face for any sign of discomfort

Steps for Enhancing Examination of the Abdomen

- The patient should have an empty bladder.
- Make the patient comfortable in a supine position, with a pillow for the head and perhaps another under the knees. Slide your hand under the low back to see if the patient is relaxed and flat on the table.
- Have the patient keep arms at the sides or folded across the chest. Often patients raise their arms over their heads, but this stretches and tightens the abdominal wall, making palpation difficult.
- Before you begin palpation, ask the patient to point to any areas of pain and examine these areas last.
- Warm your hands and stethoscope, and avoid long fingernails. You may need to rub your hands together or warm them up with hot water; you can also begin palpation through the patient's gown to absorb warmth from the patient's body before exposing the abdomen properly. Anxiety may make the hands cool, a problem that decreases over time.
- Approach slowly and avoid quick unexpected movements. Watch the patient's face closely for any signs of pain or discomfort.
- Distract the patient if necessary with conversation or questions. If the patient is frightened or ticklish, begin palpation with the patient's hand under yours. After a few moments, slip your hand underneath to palpate directly.

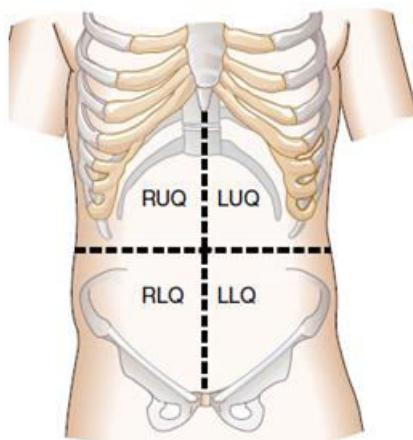
© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 7.3 Steps for enhancing examination of the abdomen



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 7.3 Nine regions of the abdomen: 1 and 3: right and left hypochondrium; 2: epigastrium; 4 and 6: right and left lumbar; 5: umbilical; 7 and 9: right and left iliac; 8: hypogastrium or suprapubic



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 7.4 The four quadrants of the abdomen

Inspection

- . Proper positioning of the patient

Supine position of the patient on a coach with proper exposure of the abdomen

- . Look for symmetry of the abdomen and flank fullness by standing at bed end
- . Look for shape of the abdomen. Is it scaphoid, flat or distended?
- . Look for contour of the umbilicus

Normally the umbilicus is slightly retracted, inverted with horizontal slit

- . Look for abdominal movement with respiration

Normally there is gentle rise in the abdominal wall during inspiration and fall during expiration

Markedly diminished or absent abdominal movement (silent abdomen) signify generalized peritonitis

- . Look for visible peristalsis

Vigorous peristalsis of the abdomen signify pyloric stenosis or bowel obstruction

- . Look for scars and striae

Striae are wrinkled linear marks due to gross stretching of the skin

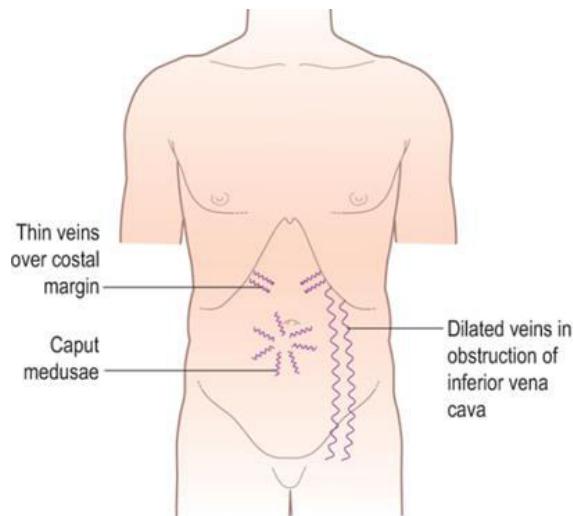
eg. Striae alba or atrophica in ascites, striae gravidarum in pregnancy, purple striae in Cushing's syndrome

- . Look for prominent superficial veins

Thin veins over the costal margin usually has no significance

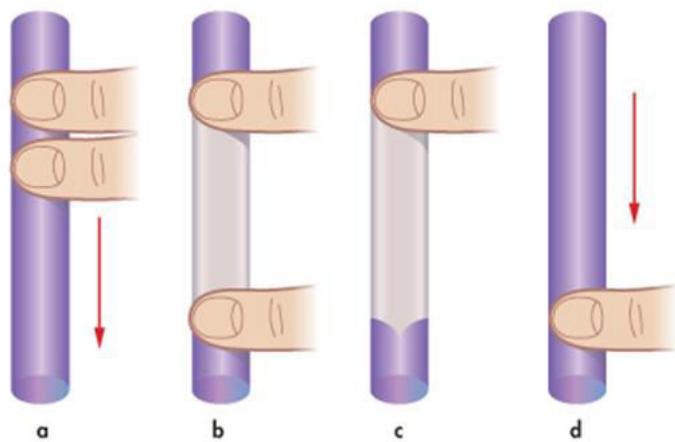
If there are distended veins over the abdomen, determine direction of blood flow (during palpation)

Distended abdominal wall veins which are draining away from the umbilicus suggests presence of portal hypertension



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 7.5 Prominent veins of the abdominal wall: Thin veins over the costal margin occurs in normal individuals; while caput medusae is suggestive of portal hypertension



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 7.6 Determining direction of blood flow in distended abdominal wall veins

- . Look for abdominal wall pigmentation eg. Linea nigra in pregnancy; erythema ab igne (due to application of heat on the abdomen wall for chronic pain)

- . Look for hernial sites

Support and lift patient's head with the shoulder with your left arm and let him cough repeatedly while observing sites of hernia

Sites of hernia: Incisional hernia at surgical scar site, umbilical and periumbilical hernia at or around the umbilicus, lumbar hernia in lumbar region, and inguinal and femoral hernia in groin region

Palpation

Superficial palpation

Ask the patient if there is abdominal area which is tender? If there is tender abdominal area, start palpation far away from the tender site and palpate the tender site at last. If there is no tenderness at any site, start at left lower quadrant, and move quadrant-by-quadrant in clock-wise or anti-clockwise direction. Observe the patient's face for any sign of discomfort during palpation.

- . Mould your relaxed and warm hand to the abdominal wall. Don't hold it rigid and avoid sudden poking with the finger tips
- . Gently palpate the abdomen quadrant-by-quadrant for abdominal tenderness, palpable abdominal masses/organs and abdominal resistance

Deep palpation

Performed to delineate abdominal mass or confirm presence of organomegaly (enlarged liver, spleen or kidney)

- . Check for guarding, rigidity and rebound tenderness

Presence of guarding, rigidity and rebound tenderness indicate generalized peritonitis

Guarding is involuntary reflex contraction of abdominal wall muscles overlying an inflamed viscus

Rigidity is involuntary reflex board-like rigidity of abdominal wall muscle

Rebound tenderness is eliciting pain while the examiner quickly withdraws his palpating hand

Abdominal organ palpation

Spleen

The spleen has to be 2-3 times its normal size to be palpable. Enlargement of the spleen takes place in a superior and posterior direction before it becomes palpable; once palpable, the direction of growth is towards the umbilicus (along the splenic growth line)

- . Start palpating from the right iliac fossa and move diagonally upward towards the left hypochondrium 1 cm at a time between each breath until you reach the left costal margin
- . Ask the patient to breathe in deeply and press in with the fingers of examining right hand beneath the costal margin

If not palpable, turn the patient to half on to the right side (with right leg extended and left leg flexed at hip and knee joint) while supporting and pressing the left costal margin with left hand

Characteristics of enlarged spleen (splenomegaly)

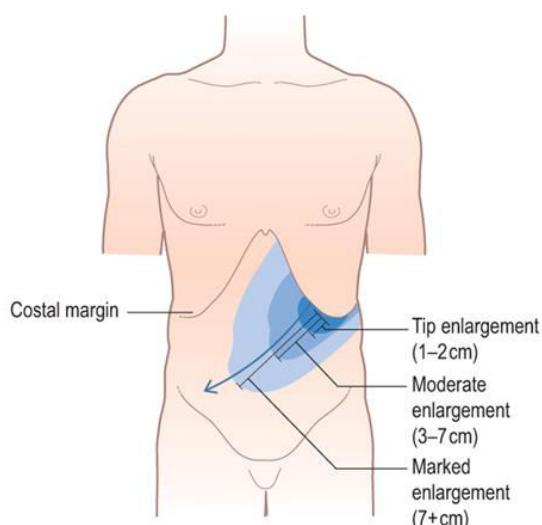
- . Moves with respiration
- . Direction of growth along the splenic growth line (towards the umbilicus)
- . Palpable splenic (medial) notch
- . Unable to go beneath the left costal margin
- . Not bimanually palpable

If there is splenomegaly, characterize size (along splenic growth line), tenderness, consistency (soft, firm, hard), surface (smooth, nodular) and edge (sharp, round)

Causes of massive splenomegaly

(7+ cm along splenic growth line)

- . Hyperreactive malarial splenomegaly
- . Hepatosplenic schistosomiasis
- . Kala-azar (Visceral leishmaniasis)
- . Chronic myeloid leukemia
- . Non-Hodgkin's lymphoma
- . Thalassemia major
- . Gaucher's disease

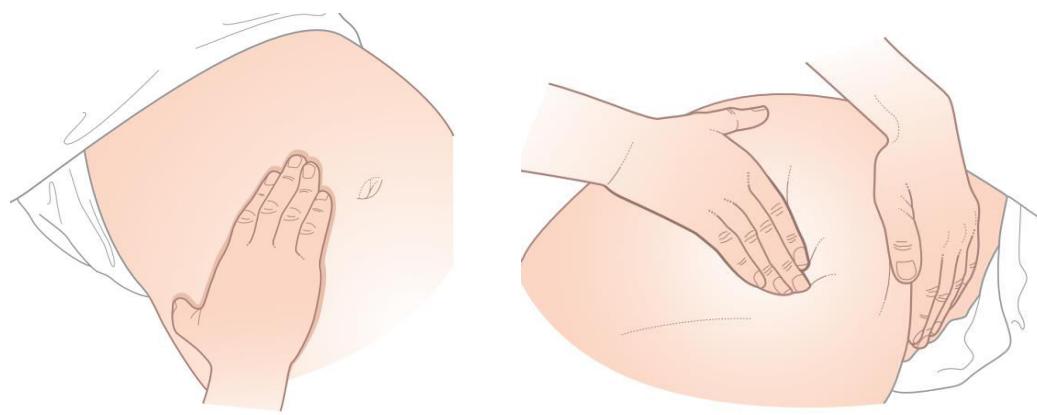


© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 7.7 Direction of splenic growth line

Liver

- . Start palpating from right lower abdomen towards the right hypochondrium with 1 cm at a time between each breath until you reach the costal margin or feel the liver edge. The liver edge may be felt against the radial border of index finger
 - . Ask the patient to breath in deeply when you reach costal margin to feel for liver edge
 - . Repeat the maneuver from lateral to medial regions to trace the liver edge
- The liver edge is often palpable in normal individuals
- . Characterize the enlarged liver - size (below the right costal margin), tenderness, consistency, surface, edge
(Total vertical liver span is determined by palpation and percussion)



A) Palpation of the liver

B) Palpation of the spleen

© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 7.8 Technique of palpation of the liver and spleen

Kidney

Left kidney

- . Place the left hand posteriorly in the left loin and right hand anteriorly in the left lumbar region
- . Ask the patient to take deep breathe in, and press the left hand forwards and the right hand backwards, upwards and inwards

The left kidney is often not palpable

Right kidney

- . Place the right hand horizontally in the right lumbar region anteriorly with left hand placed posteriorly in the right loin

- . Ask the patient to take a deep breath in, and press the left hand forwards and the right hand backwards, upwards and inwards

The lower pole of the right kidney is often palpable in healthy individuals

Enlarged kidney is bimanually palpable



A) Palpation of right kidney



B) Palpation of left kidney

© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 7.9 Technique of palpation of right kidney (A) and left kidney (B)

Gall bladder

The gall bladder is palpated the same as the liver

The normal gall bladder can't be felt

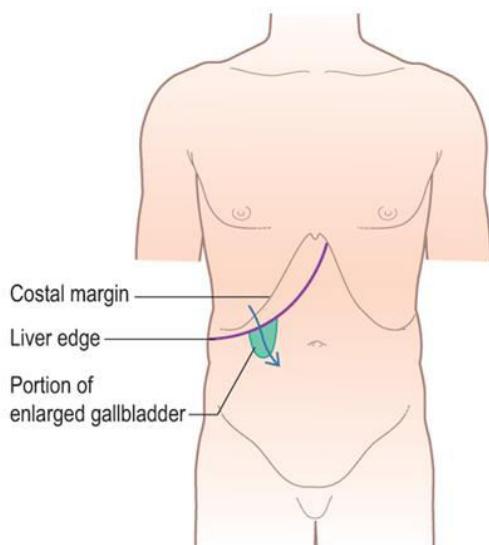
When it is distended, it may be palpated as a firm, smooth or globular swelling with distinct border, just lateral to the edge of rectus muscle near the tip of ninth costal cartilage

The upper border merges with the lower border of the right lobe of the liver or disappears beneath the right costal margin

Murphy's sign indicates acute cholecystitis

- . Ask the patient to breathe in deeply, and palpate for the gall bladder in the normal way
- . The breathing stops with a gasp at the end of inspiration as the inflamed distended gall bladder is felt with palpating hand

Courvoisier's law signify presence of jaundice with palpable gall bladder, which makes common bile duct obstruction by gall stone as an unlikely cause



© Elsevier. Swash & Glynn. Hutchison's Clinical Methods 22e

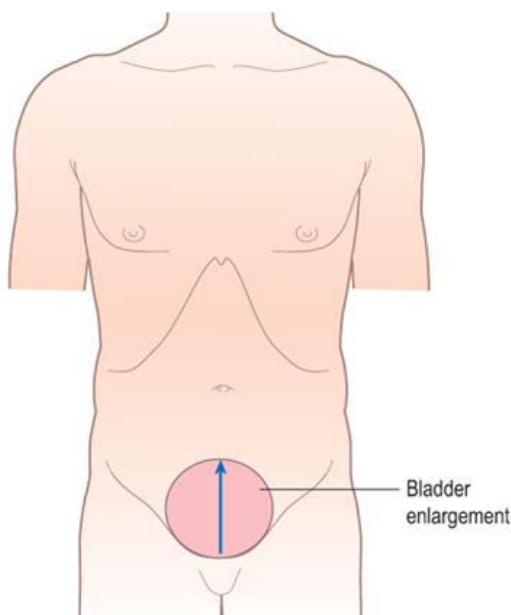
Fig 7.10 Enlarged gall bladder: The fundus and part of body of enlarged gall bladder can be palpated

Urinary bladder

Normally, the urinary bladder is not palpable

Distended urinary bladder due to retention of urine is characterized by symmetric swelling in the supra pubic region below the umbilicus and arising out of the pelvis

Supra pubic swelling is also caused by gravid uterus, uterine myoma and ovarian cyst



© Elsevier. Swash & Glynn. Hutchison's Clinical Methods 22e

Fig 7.11 Physical signs in retention of urine: Smooth, firm and regular swelling arising out of the pelvis which one cannot 'get below', and dull in percussion note

Examination of palpable abdominal swelling

Site/location

Is it abdominal wall swelling or intra-abdominal swelling?

- . Feel the swelling while the patient lifts her/his head with shoulder to tense the abdominal wall

Prominent, protruded swelling indicates abdominal wall mass, while the swelling hides behind in intra-abdominal mass

- . Notice the regions occupied by the swelling

eg. Swelling in the right hypochondrium most probably arises from the liver, right kidney, hepatic flexure of colon, and gall bladder

If the swelling occupies the upper abdomen, is it possible to ‘get above it’?

If one can’t ‘get above it’ hepatic, splenic or gastric origin should be suspected

If the swelling occupies the lower abdomen, is it possible to ‘get below it’?

If one can’t ‘get below it’ the swelling probably arises from the urinary bladder, uterus, ovary or upper rectum

Size and shape

Determine length, width and depth of the abdominal swelling

Characterize the surface, edge and consistency of the abdominal swelling

- . Hard, nodular, irregular in outline mass is more likely to be malignancy
- . Solid, tender, ill-defined mass is more likely to be inflammatory
- . Regular round, smooth, tense swelling is more likely to be cystic

Mobility and attachment

Mobility

Swelling arising from the liver, spleen, gall bladder and stomach moves down ward during inspiration

Swelling arising from the bowel, mesentery and omentum are not usually influenced by respiratory movement

Side-to-side movable lower abdominal swelling favors swelling of uterine origin (gravid uterus, uterine myoma)

Attachment

Fixed swelling usually signify mass of retroperitoneal origin or abdominal tumor with extensive spread to abdominal wall

Is it bimanually palpable /pulsatile?

Does the pulsation come from the swelling or is it transmitted?

Pulsatile and expansile abdominal swelling favors abdominal aortic aneurysm

Percussion

Technique

Put the left pleximeter finger of the examiner on the abdomen in horizontal position and percuss with the right plexor finger from upper to lower abdomen and sideways of the abdomen

The normal abdomen is tympanitic in percussion note

Percuss if there is enlarged organ or any swelling in the abdomen

Liver

Total liver span (TLS) is usually mapped out by percussion

Percussion will detect the upper border of normal liver at about the 5th intercostal space along right mid-clavicular line, and the dullness extends down to the lower border at the right subcostal margin with TLS of 10 +/- 2 cm

Spleen

Dullness of normal spleen extends from the left lower ribs into the left subcostal margin

Percussion for splenic dullness

Techniques

Nixon's method

- . Ask the patient to turn to the right side
- . Begin percussing at the lower level of pulmonary resonance along the left posterior axillary line and proceed diagonally along a perpendicular line towards the lower midanterior costal margin

The upper border of dullness is normally 6-8 cm above the left costal margin

Dullness > 8cm suggests splenomegaly

Castell's method

- . Ask the patient to be on supine position
- . Percuss the left intercostal space at 8th or 9th interspace along left anterior axillary line during full inspiration and expiration

Dullness on full inspiration suggests splenomegaly

Traube's method

- . Borders of Traube's space: Superiorly 6th rib, laterally left mid axillary line, inferiorly left costal margin
- . Ask the patient to be on supine position with the left arm slightly abducted
- . Percuss from medial to lateral margins in normal breathing. It usually produces normal resonant sound at traube's space

Dullness to percussion at Traube's space suggests splenomegaly

Detection of ascites

Shifting dullness

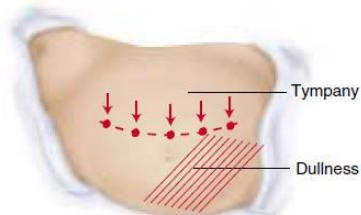
The cardinal sign created by ascites

Technique

- . Position the patient in supine
- . Place your fingers in the longitudinal axis on the midline near the umbilicus
- . Percuss from midline out to the flanks
- . Note any change from tympanitic to dullness
- . Keep your finger on the site of dullness in the flanks and ask the patient to turn onto his opposite side
- . Pause for at least 10 seconds to allow for any peritoneal fluid to gravitate, then percuss again
- . Shifting dullness is present if the area of dullness is now tympanitic

Peritoneal fluid of 1500ml usually elicits shifting dullness

In ascites, dullness shifts to the more dependent side, while tympany shifts to the top.



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 7.12 Shifting dullness in ascites

Fluid thrill

Technique

- . Position the patient in supine
- . Place the palm of your left hand flat against the left side of the abdomen
- . Ask an assistant to place the ulnar border of their hand on the midline of the abdomen
- . Flick a finger of your right hand against the right side of the abdomen
- . Fluid thrill is present if you feel for a ripple (wave) against your left hand



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 7.13 Technique of eliciting fluid thrill

Clinical features of markedly distended abdomen

Gross ascites

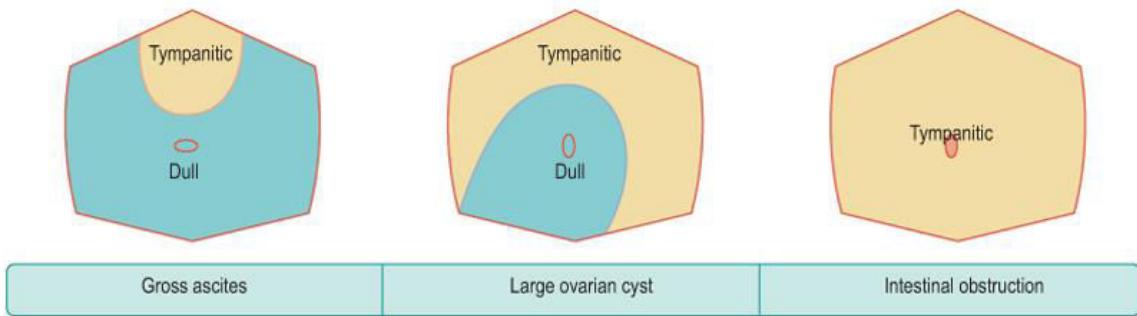
- . Flank fullness and dullness in flanks
- . Umbilicus everted
- . Shifting dullness and fluid thrill present

Large ovarian cyst

- . Resonant in flanks
- . Umbilicus vertical and drawn up
- . Swelling arising out of the pelvis which one can't get below

Intestinal obstruction

- . Tympanitic to percussion
- . Increased and noisy bowel sound



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 7.14 Cause of markedly distended abdomen

Auscultation

Auscultation of the abdomen is for detecting bowel sounds and vascular bruits

Bowel sounds

- . Place the stethoscope just to the right of the umbilicus, and listen to low-to-medium pitched gurgles interposed by high pitched noise or tinkle
- . Note the bowel sounds frequency and character

Normal sounds consist of clicks and gurgles with a frequency of 5-30 per minute

Listen for up to 2 minutes before concluding that the bowel sounds are absent

Increased frequency of bowel sounds occur in diarrhea and mechanical intestinal obstruction

Reduced or absent bowel sounds occur in paralytic ileus and generalized peritonitis

Succession splash

- . Place the patient in supine position
- . Place the diaphragm of the stethoscope over the epigastrium (help may be needed!)
- . Place your hands on the lumbar region of the abdomen, and roll the patient briskly from side to side
- . Splashing sound is heard if the stomach is distended with fluid
- . Positive test is confirmed if there is splashing sound after 4 hours of meal intake

Succession splash is positive in gastric outlet obstruction and paralytic ileus

Vascular bruits and friction rub

Place the stethoscope above and right/left of umbilicus for renal bruits (renal artery stenosis), over the enlarged liver for bruits (hepatocellular cancer), over the abdominal mass (malignancy, aneurysm), and over the enlarged spleen for friction rub (splenic infarction)

Examination of the groin

- . Inspect the groin in supine or standing position

In supine positioned patient, lift the patient's head with the shoulder and ask him to give loud cough, and feel with hand for any impulse at groin area

Inguinal swelling with positive cough impulse suggests inguinal hernia

Patient who presented with swelling (lump) in the groin

- . Ask the patient to stand in front of you
- . Look at the groin lump and note if it extends into the scrotum
- . Feel for an impulse of the lump after the patient gives loud cough
- . If cough impulse is present, place your fingers obliquely over the inguinal canal and ask him to cough again. If an impulse is felt, the lump must be a hernia.

Is the groin lump inguinal or femoral hernia?

- . Determine relationship of hernia sac to the pubic tubercle (bony prominence 2 cm from midline on suprapubic crest)
- . Gently push upwards from beneath the neck of scrotum with the index finger to reach for pubic tubercle
- . In inguinal hernia, hernia sac passes medial to and above the index finger placed on the pubic tubercle
- . In femoral hernia, the hernia sac is lateral to and below the index finger placed on the pubic tubercle

Notice for content of hernial sac

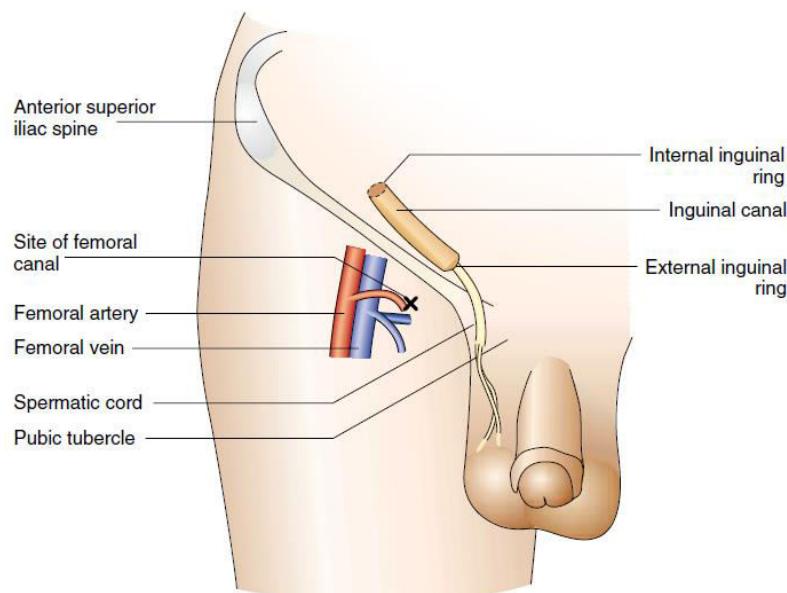
- . If omentum, it feels firm and doughy in consistency
- . If bowel, it tends to gurgle, compressible and soft in consistency

Is the hernia reducible or non-reducible?

- . Place the patient in supine position and ask the patient to reduce the hernia himself (to confirm for you!)

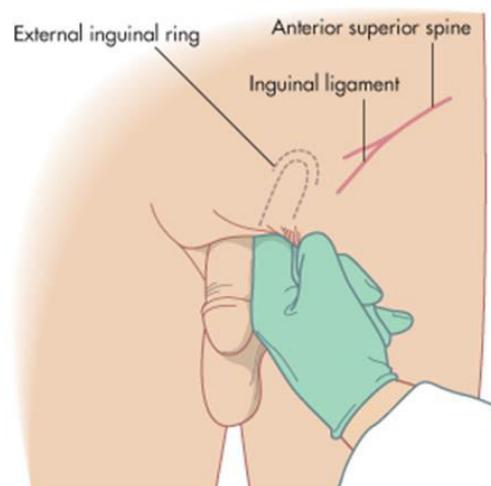
Is it direct or indirect inguinal hernia?

- . Direct hernia tends to bulge straight out through the posterior wall of the inguinal canal
- . The lump with an impulse travelling obliquely down along the inguinal canal is indirect inguinal hernia, and it is controlled by placing one finger over the deep inguinal ring (over the mid inguinal point)



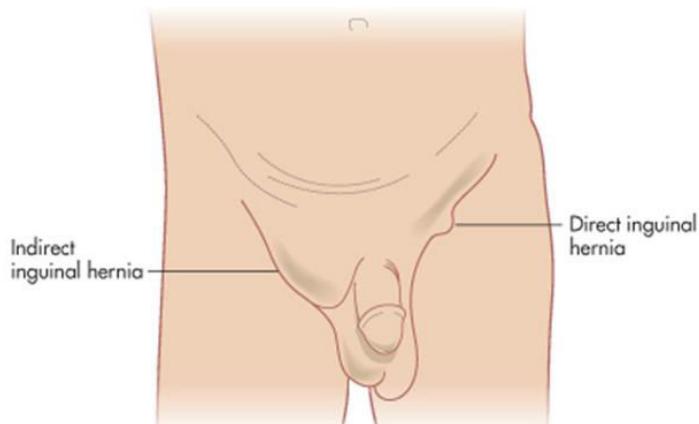
© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 7.15 Sites of inguinal and femoral canal



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 7.16 Technique of examining inguinal hernia



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 7.17 Direct and indirect inguinal hernias

Differential diagnosis of inguinal hernia

- . Femoral hernia
- . Hydrocele (able to 'get above' the swelling)
- . Epididymal cyst
- . Undescended or ectopic testis (empty scrotum on the affected side)
- . Inguinal lymphadenopathy (LAP)

Differential diagnosis of femoral hernia

- . Inguinal hernia
- . Lipoma in femoral triangle
- . Aneurysm of the femoral artery (expansile pulsation positive)
- . Sapheno-varix
- . Psoas abscess
- . Inguinal lymphadenopathy

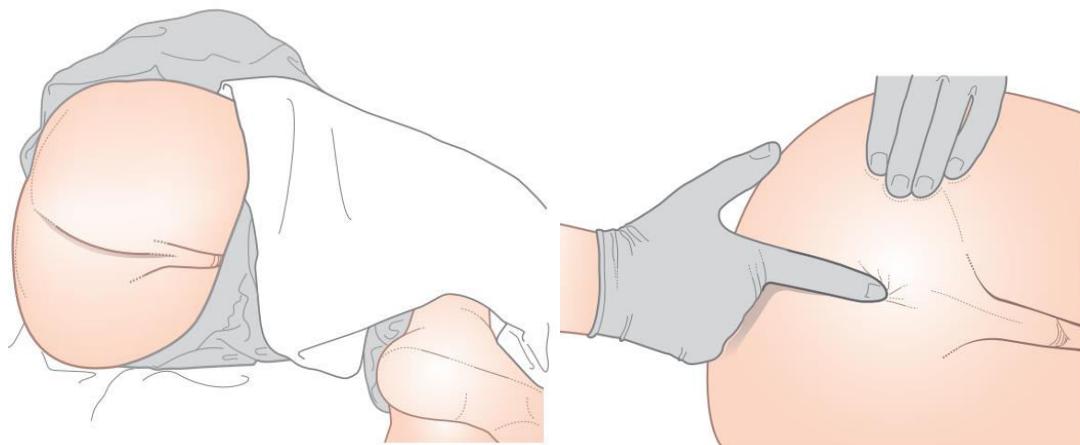
Digital rectal examination

Abdominal examination without per digital rectal examination is incomplete!

Technique

- . Explain what you are going to do and ask for permission to proceed (be gentle during per-rectal examination!)
- . Position the patient to lie on left lateral side with buttocks at the edge of the coach, knees drawn up to the chest

- . Put on gloves and spread the buttocks apart
 - . Inspect for perianal areas for excoriations, inflammation, ulcers and lumps
 - . Lubricate your gloved index finger, and gently insert your index finger tip into the anal canal in a direction pointing toward the umbilicus
- Notice for sphincter tone of the anus, tenderness, induration, irregularities or nodules
- . Insert your finger into the rectum as far as possible
 - . Rotate your finger to palpate the rectal surface for nodules, irregularities or induration
 - . Identify the uterine cervix in women and prostate in men
 - . Examine the prostate gland to identify its lateral lobes and the medial sulcus
 - . Characterize the size, shape, tenderness and consistency of the prostate (normal prostate is rubbery and non tender)
 - . Gently withdraw your finger and wipe the patient's anus
 - . Look for blood staining of fecal matter on the glove



A) Left lateral position for digital rectal examination B) Correct method of inserting index finger

© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 7.18 Technique of digital rectal examination

CHAPTER EIGHT

Nervous system

Learning objective

At the end of this lesson, the student should be able to:

1. Mention main symptoms in nervous system disorders
2. Assess level and content of consciousness
3. Show techniques of cranial nerves and motor system examination
4. Identify clinical features of upper motor neuron lesion from lower motor neuron lesion
5. Identify the location of lesion in a patient presenting with neurologic deficit

History taking

Major symptoms in nervous system

- . Headache
- . Cognitive disturbance
- . Difficulty of speech (dysphasia/aphasia)
- . Loss of consciousness
- . Difficulty in vision, hearing, swallowing
- . Weakness of extremity
- . Abnormal body movement
- . Bladder or bowel dysfunction (urinary retention or incontinence/ fecal impaction or incontinence)
- . Abnormal sensory symptoms

Headache

Causative factors for headache

- . Spasm, inflammation or trauma to cranial or cervical muscles
- . Meningeal irritation and increased intracranial pressure

- . Distension, traction, or dilation of intracranial or extracranial arteries
- . Traction or displacement of large intracranial veins or their dural envelope
- . Activation of brain stem structures (dorsal raphe of the midbrain)

Important clues regarding history of headache

- . Location and type of headache
- . Mode (sudden vs insidious) and course (intermittent, persistent, progressive) of onset
- . Frequency, intensity, duration of headache episodes
- . Precipitating and relieving factors
- . Associated systemic symptoms
- . Presence of aura or prodrome
- . Relation with food, alcohol, drugs (oral contraceptive pills), menses, etc...
- . Response to analgesics
- . Family history of headache
- . Age at onset

Headache features that suggest serious underlying disorder

- . ‘Worst’ headache ever
- . Subacute worsening headache over days to weeks
- . Vomiting which precedes headache
- . Headache, which disturbs sleep or presents immediately upon awakening
- . Headache with fever or unexplained systemic symptoms
- . Headache induced or worsened by bending, coughing or straining
- . Headache with known systemic illness (systemic cancer/HIV infection)
- . Headache with abnormal neurologic deficit
- . Headache onset > 55 yrs of age, or < 5 yrs of age

Serious underlying causes of headache

Subarachnoid hemorrhage: The first ‘worst’ headache ever, neck stiffness and altered mentation

Infective meningitis: Headache with fever, neck stiffness, and altered mentation

Brain tumor: Subacutely worsening head ache with nausea and vomiting, disturbs sleep, worsen during awakening, aggravated by bending, coughing or straining

Temporal arteritis: Elderly individual with pounding, temporal headache and visual changes

Glaucoma: Severe, protracted headache, red eye with eye pain

Cognitive impairment (amnestic state)

. Age at onset (senile or presenile dementia)

Cognitive disturbance under 60 years of age is presenile dementia and usually pathological in nature

. Ask for history of head injury, chronic alcoholism, stroke, brain tumor, CNS infections, etc...

Types of amnesia

1. Retrograde amnesia: Inability to recall experiences that occurred before the onset of amnesia

Relatively recent events are more vulnerable than remote events in retrograde amnesia

2. Anterograde amnesia: Inability to register, store and retrieve new information

Patients with anterograde amnesia have a tendency to fill memory gaps with inaccurate, fabricated, implausible information, named as confabulation

eg. “Patients can’t remember what they ate a few minutes ago or the details of an important event they have experienced few hours ago”

Causes of dementia

a. Most common causes of dementia

. Alzheimer’s disease

. Vascular dementia (multi-cerebral infarction, diffuse white matter disease)

. Alcoholism

. Parkinson's disease

. Drug/medication toxicity

b. Less common causes of dementia

. Vitamin deficiency

Vitamin B₁ (Wernicke's encephalopathy), vitamin B₁₂ (pernicious anemia), and nicotinic acid deficiency (pellagra)

. Endocrine failure- Hypothyroidism, Addison's disease and Cushing's syndrome

. Chronic infections- HIV/AIDS, neurosyphilis

. Head injury- chronic subdural hematoma, dementia pugilistica

. Normal-pressure hydrocephalus (clinical triads of dementia, ataxia and urinary incontinence)

. Neoplastic – primary brain tumors, metastatic brain tumors, paraneoplastic limbic encephalitis

. Toxic disorders- heavy metal intoxication, organic toxins

. Degenerative disorders- Dementia with Lewy bodies, Pick's disease, Huntington's disease, fronto-temporal dementia, etc...

Abnormal body movement

Seizures

Seizure is paroxysmal event due to abnormal, excessive, hypersynchronous discharges from an aggregate of central nervous system neurons

Epilepsy is recurrent seizures due to chronic, underlying process in the brain cortex

Generally, seizure disorder is broadly divided into partial and generalized seizures

Partial seizure: seizure activity is restricted to discrete areas of the cerebral cortex, usually associated with preserved consciousness

Generalized seizure: seizure activity involving diffuse regions of the brain and usually associated with loss of consciousness

. Ask

Was it a seizure or not? (clinical history from an attendant if a seizure activity is associated with loss of consciousness)

If yes, was it associated with loss of consciousness? Impaired or loss of consciousness during ictal-phase of seizure occurs in complex partial and generalized seizure disorder respectively

Was there prodromal phase?, which begins hours to days before the seizure, and manifests with irritability, headache, insomnia, bad temper, depression or manic behavior

. Was there preceding aura?

Presence of preceding aura signals focal onset of the seizure, and presents with extreme fear, strange epigastric sensations, dream-like experiences, unpleasant smells, etc...

. Was there post-ictal syndrome?, which may be brief or last for several hours, and manifests with headache, irritability, confusion, drowsiness, muscle ache/soreness, paralysis (Todd's paralysis), altered speech/aphasia, altered behaviors or emotional outbursts

. Age at onset, frequency and duration of the attack (fit), the time of day that the seizure occurs, altered behaviors during the attack, history of anti-seizure drug use (what drug/s, how long in use, effect of medication)

. Were there underlying endogenous, epileptogenic and precipitating factors?

Endogenous factors

. Family history of epilepsy, genetic susceptibility for epilepsy

Epileptogenic factors

History of head injury, stroke, brain tumor, CNS infections, degenerative CNS diseases, etc...

Precipitating factors

-
- Flashing lights (resulting in reflex epilepsy)
 - Hyperventilation
 - Lower alertness, sleep itself and lack of enough sleep
 - Emotion
 - Physical stress
 - Special smells, sounds| or sensations of touch
 - Alcohol
 - Hormonal changes, e.g., during menses
 - High fever
 - Overhydration
-

Positive likelihood of an initial seizure to be recurrent

- . Initial seizure presenting as status epilepticus
- . Seizure associated with post-ictal Todd's paralysis

- . Family history of epilepsy
- . Seizure associated with abnormal neurologic deficit
- . Seizure associated with abnormal electroencephalography (EEG)

Common causes of seizure in adults

- . Head injury
- . Alcoholism (Binge alcohol intake or alcohol withdrawal)
- . Brain tumor
- . Cerebrovascular disease
- . Metabolic disorders such as uremia, hepatic failure, etc...
- . Degenerative central nervous system (CNS) diseases such as Alzheimer's disease
- . CNS infections such as meningitis, encephalitis
- . Idiopathic

Table 8.1 Classification and Clinical features of seizure disorders

Problem	Clinical Manifestations	Postictal (<i>Postseizure</i>) State
Partial Seizures		
<i>Simple Partial Seizures</i>		
■ With motor symptoms		
Jacksonian	Tonic and then clonic movements that start unilaterally in the hand, foot, or face and spread to other body parts on the same side	Normal consciousness
Other motor	Turning of the head and eyes to one side, or tonic and clonic movements of an arm or leg without the Jacksonian spread	Normal consciousness
■ With sensory symptoms	Numbness, tingling; simple visual, auditory, or olfactory hallucinations such as flashing lights, buzzing, or odors	Normal consciousness
■ With autonomic symptoms	A “funny feeling” in the epigastrium, nausea, pallor, flushing, lightheadedness	Normal consciousness
■ With psychiatric symptoms	Anxiety or fear; feelings of familiarity (<i>déjà vu</i>) or unreality; dreamy states; fear or rage; flashback experiences; more complex hallucinations	Normal consciousness
<i>Complex Partial Seizures</i>	The seizure may or may not start with the autonomic or psychic symptoms that are outlined above. Consciousness is impaired and the person appears confused. Automatisms include automatic motor behaviors such as chewing, smacking the lips, walking about, and unbuttoning clothes; also more complicated and skilled behaviors such as driving a car.	The patient may remember initial autonomic or psychic symptoms (which are then termed an <i>aura</i>), but is amnesic for the rest of the seizure. Temporary confusion and headache may occur.
<i>Partial Seizures That Become Generalized</i>	Partial seizures that become generalized resemble tonic-clonic seizures (see next page). Unfortunately, the patient may not recall the focal onset and observers may overlook it.	As in a tonic-clonic seizure, described on the next page. <i>Two attributes indicate a partial seizure that has become generalized: (1) the recollection of an aura, and (2) a unilateral neurologic deficit during the postictal period.</i>

Problem	Clinical Manifestations	Postictal (Postseizure) State
Generalized Seizures		
<i>Tonic–Clonic Convulsion (grand mal)*</i>	The person loses consciousness suddenly, sometimes with a cry, and the body stiffens into tonic extensor rigidity. Breathing stops and the person becomes cyanotic. A clonic phase of rhythmic muscular contraction follows. Breathing resumes and is often noisy, with excessive salivation. Injury, tongue biting, and urinary incontinence may occur.	Confusion, drowsiness, fatigue, headache, muscular aching, and sometimes the temporary persistence of bilateral neurologic deficits such as hyperactive reflexes and Babinski responses. The person has amnesia for the seizure and recalls no aura.
<i>Absence</i>	A sudden brief lapse of consciousness, with momentary blinking, staring, or movements of the lips and hands but no falling. Two subtypes are recognized. <i>Petit mal absences</i> last less than 10 sec and stop abruptly. <i>Atypical absences</i> may last more than 10 sec.	No aura recalled. In petit mal absences, a prompt return to normal; in atypical absences, some postictal confusion
<i>Atonic Seizure, or Drop Attack</i>	Sudden loss of consciousness with falling but no movements. Injury may occur.	Either a prompt return to normal or a brief period of confusion
<i>Myoclonus</i>	Sudden, brief, rapid jerks, involving the trunk or limbs. Associated with a variety of disorders	Variable
Pseudoseizures		
May mimic seizures but are due to a conversion reaction (a psychological disorder).	The movements may have personally symbolic significance and often do not follow a neuroanatomic pattern. Injury is uncommon.	Variable

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Movement disorders

Common terminologies in movement disorders

Akinesia Motor restlessness; constant semi-purposeful movements of the arms and legs

Asterixis Sudden loss of muscle tone during sustained contraction of an outstretched limb

Athetosis Writhing, slow movements of hands and wrists

Chorea Irregular, jerky and brief rapid extremity movements. Chorea and athetosis often occur combined, and named choreoathetosis

Dyskinesia Purposeless and continuous facial and mouth movements

Dystonia Sustained contractions of agonist and antagonist muscles, which results in bizarre postures (twisting) in flexion or extension

Ballismus Violent flinging movement of extremity

Myoclonus Brief muscle contraction which causes a sudden purposeless jerking of a limb

Myokymia Repeated contraction of a small muscle group involving orbicularis oculi muscles

Tic Repetitive, irresistible, stereotyped movement

Tremor Rhythmic alternating movement

Weakness of extremities

Weakness is reduced strength or power of one or more muscles

Was there weakness of any part of the body (paralysis)? Which body parts were involved? Was it distal or proximal weakness? Date and mode of onset, temporal course, co-morbidities

Proximal weakness is due to muscle disease (myopathy), and manifests with difficulty in standing from sitting position, or difficulty in combing hair

Distal weakness is due to peripheral neuropathy, and manifests with slapping of feet while walking, or difficulty in opening a jar or using hand tools (scissors/screw driver)

Hemiparesis: Weakness of ipsilateral upper and lower extremities

Causes of hemiparesis

- a. Acute hemiparesis (onset in minutes to hours)
 - . Head trauma
 - . Stroke
 - . Bleeding into brain tumor
- b. Subacute hemiparesis (onset in days to weeks)
 - . Subdural hematoma
 - . Brain abscess
 - . Cerebral toxoplasmosis/primary CNS lymphoma (AIDS patients)
- c. Chronic hemiparesis (onset in months)
 - . Tuberculoma
 - . Neurocysticercosis
 - . Brain tumor
 - . Chronic subdural hematoma
 - . Degenerative CNS diseases

Paraparesis: Weakness of both lower extremities

Causes of paraparesis

Usually caused by spinal cord disorders

a. Acute paraparesis (onset in hours)

- . Acute transverse myelopathy, eg. Transverse myelitis
- . Spinal cord injury (spinal shock)
- . Spinal cord infarction

b. Subacute or chronic paraparesis (onset in weeks to months)

- . Disc prolapse or herniation
- . Pott's disease (tuberculous spondylitis)
- . Compressive myelopathy eg. epidural abscess, vertebral osteomyelitis, and secondary tumor deposits
- . Degenerative spondylosis: Disk-osteophyte complex

Monoparesis (brachial or crural): Weakness of one upper (brachial) or lower (crural) extremity

Causes of monoparesis

a. Acute monoparesis (onset in hours to days)

- . Ischemic stroke
- . Poliomyelitis
- . Cerebral vasculitides

b. Subacute or chronic monoparesis (onset in weeks to months)

- . Brachial or lumbosacral plexopathy
- . Cauda-equina syndrome

Abnormal sensory symptoms

What type of abnormal sensory symptoms felt (pins-and-needles or numbness)? Date and mode of onset, temporal course, co-morbidities (eg. diabetes, chronic kidney disease, etc...), exposure to drugs and toxins (herbicides, pesticides and industrial chemical solvents), etc...

Abnormal sensory phenomena

Positive sensory phenomena

- . Paresthesia- Pins and needles sensation
- . Dysesthesia- abnormal sensation whether a stimuli is evident or not

Positive sensory phenomena usually results from ectopic impulses generated at a site or sites of lowered threshold or heightened excitability along a sensory pathway

Negative sensory phenomena

It represents loss of sensory function and is characterized by diminished or absent feeling, often experienced as numbness

In contrast to positive phenomenon, negative phenomenon is accompanied by abnormal findings on sensory examination

At least half of afferent axons of innervating a given site are lost to demonstrate sensory deficit

Sensory signs are always a measure of negative sensory phenomenon

Examination of the nervous system

Level of consciousness

Level of consciousness is assessed by Glasgow Coma Scale (GCS), numerical value to the patient's responses to defined stimuli

Score of $\leq 8/15$ indicates coma, unarousable to any external noxious stimuli

Reduced level of consciousness results from acute cerebral dysfunction from cerebral hypoperfusion, metabolic diseases, CNS infections, and brain stem reticular activating system (RAS) lesions

Assessment variables and scores of GCS

1. Eye opening

Spontaneous --- 4, To speech --- 3, To pain --- 2, None --- 1

2. Best verbal response

Oriented --- 5, Confused --- 4, Inappropriate speech --- 3, incomprehensible sound --- 2, None --- 1

3. Best motor response

Obeys command --- 6, Localizes pain -- 5, Normal withdrawal --4

Abnormal flexion --- 3, Abnormal extension --- 2, None --- 1

Total score out of 15_____

Level of Consciousness (Arousal): Techniques and Patient Response	
Level	Technique
Alertness	Speak to the patient in a normal tone of voice. An alert patient opens the eyes, looks at you, and responds fully and appropriately to stimuli (arousal intact).
Lethargy	Speak to the patient in a loud voice. For example, call the patient's name or ask "How are you?"
Obtundation	Shake the patient gently as if awakening a sleeper.
Stupor	Apply a painful stimulus. For example, pinch a tendon, rub the sternum, or roll a pencil across a nail bed. (No stronger stimuli needed!)
Coma	Apply repeated painful stimuli.
Abnormal Response	
A lethargic patient appears drowsy but opens the eyes and looks at you, responds to questions, and then falls asleep.	
An obtunded patient opens the eyes and looks at you, but responds slowly and is somewhat confused. Alertness and interest in the environment are decreased.	
A stuporous patient arouses from sleep only after painful stimuli. Verbal responses are slow or even absent. The patient lapses into an unresponsive state when the stimulus ceases. There is minimal awareness of self or the environment.	
A comatose patient remains unarousable with eyes closed. There is no evident response to inner need or external stimuli.	

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 8.2 Conventional method of assessing level of consciousness

Content of consciousness (cognitive impairment)

Mini-mental state examination (MMSE) indicates severity of cognitive impairment

It is scored out of 30

MMSE numerical value $\leq 23/30$ indicates presence of impaired cognition (dementia)

Mild dementia 21-23/30

Moderate dementia 18-21/30

Severe dementia <18/30

Assessment variables and scoring system for cognitive impairment (MMSE)

1. Orientation in time and place

Time- Ask day, date, month, season and year (score 1 for each correct answer)

Place- Ask ward, hospital, town, capital city, country (score 1 for each correct answer)

2. Registration

Name three common objects, and tell and rehearse with the patient to remember them (score 1 for each correct registration), remind him that he will be asked to name them after 5 minutes

3. Attention and calculation

Ask him to subtract 7 from 100, or 3 from 30 for a total of 5 times (score 1 for each correct subtraction)

4. Recall

Ask the patient to name the three objects registered in question number 2 (score 1 for each correct recall)

5. Language

a. Show him 2 objects (eg. pen, watch) and assess if named correctly (score 1 for each correct naming)

b. Assess correct repetition of “no ifs, and, buts” (score 1 for correct repetition)

c. Assess if the three-stage command correctly obeyed eg. “Take this piece of paper in your left hand, fold it in half, and place it on the table” (score 1 for each correct answer)

d. Assess response to written command such as “Raise your left hand” (score 1 for correct answer)

e. Ask the patient to write any sentence; and assess if it is meaningful, and has subject and verb (score 1 for a sentence which is meaningful, has subject and verb)

f. Test for patient’s ability to copy complex diagram of 2 intersecting pentagons (score 1 for correct coping)

Total score out of 30 _____

Cognitive impairment

Assess type of cognitive impairment (Is it immediate, recent or remote memory loss?)

Memory

Disturbance in memory is named as amnesia

Major types of amnestic state

Remote memory loss

Ask about autobiographical events from the distance past or national historical events

When did you get married? What is the name of Ethiopian leader in the 1940's E.C?

Recent memory loss

Ask about recent events in the past few minutes or hours

What did you eat at lunch time today?

Immediate memory loss

Tell him to register new information and ask him immediately what he was told

Failed to register new information what he was immediately told suggests immediate memory loss

Speech disturbance

Dysphasia (aphasia): Difficulty in speech language

Major types of aphasia

a. Wernicke's aphasia

- . Impaired comprehension of spoken and written language
- . Fluent speech devoid of meaning (jargon aphasia)
- . Inappropriately uttered phrases of fluent speech (paraphasia)
- . Patient is unaware of his speech deficit, and shows paranoid behavior

b. Broca's aphasia

- . Impaired fluency with poverty of speech
- . Intact comprehension of spoken or written language
- . Patient is aware of the speech deficit and feels depressed

c. Global aphasia

- . Impaired comprehension and poverty of speech (Wernicke's and Broca's aphasia)
- . Generally, the patient is in mute state

Dysarthria: Difficulty in speech articulation

Types of dysarthria

Cerebellar dysarthria: 'scanning' and robotic speech with syllables pronounced individually and slowly

Bulbar dysarthria: difficulty in pronunciation of consonants

Cortical dysarthria: strangled and spastic speech

Disorders of speech fall into three groups: those affecting (1) the voice, (2) the articulation of words, and (3) the production and comprehension of language.

Aphonia refers to a loss of voice that accompanies disease affecting the larynx or its nerve supply. *Dysphonias* refers to less severe impairment in the volume, quality, or pitch of the voice. For example, a person may be hoarse or only able to speak in a whisper. Causes include laryngitis, laryngeal tumors, and a unilateral vocal cord paralysis (Cranial Nerve X).

Dysarthria refers to a defect in the muscular control of the speech apparatus (lips, tongue, palate, or pharynx). Words may be nasal, slurred, or indistinct, but the central symbolic aspect of language remains intact. Causes include motor lesions of the central or peripheral nervous system, parkinsonism, and cerebellar disease.

Aphasia refers to a disorder in producing or understanding language. It is often caused by lesions in the dominant cerebral hemisphere (usually the left).

Compared below are two common types of aphasia: (1) Wernicke's, a fluent (receptive) aphasia, and (2) Broca's, a non-fluent (or expressive) aphasia. There are other less common kinds of aphasia, which may be distinguished from each other by differing responses on the specific tests listed. Neurologic consultation is usually indicated.

	Wernicke's Aphasia	Broca's Aphasia
Qualities of Spontaneous Speech	Fluent; often rapid, voluble, and effortless. Inflection and articulation are good, but sentences lack meaning and words are malformed (paraphasias) or invented (neologisms). Speech may be totally incomprehensible.	Nonfluent; slow, with few words and laborious effort. Inflection and articulation are impaired but words are meaningful, with nouns, transitive verbs, and important adjectives. Small grammatical words are often dropped.
Word Comprehension	Impaired	Fair to good
Repetition	Impaired	Impaired
Naming	Impaired	Impaired, though the patient recognizes objects
Reading Comprehension	Impaired	Fair to good
Writing	Impaired	Impaired
Location of Lesion	Posterior superior temporal lobe	Posterior inferior frontal lobe

While it is important to recognize aphasia early in your encounter with a patient, its full diagnostic meaning does not become clear until you integrate this information with your neurologic examination.

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 8.3 Disorders of speech: Dysphonias, dysarthria and dysphasias

Apraxia

Impairment in executive complex motor tasks, and usually observed in frontal lobe diseases

Judgment and abstract thinking

. Inaccurate judgment and abstract thinking suggest frontal lobe diseases

a. Judgment

Ask a question like "What will you do if you find a closed stamped letter on the street?" and assess judgment

b. Abstractional ability

Ask the patient the meaning of a well known proverb eg. "Birds of the same feather flock together"

Cranial nerves

Location of cranial nerves

Cranial nerve 1 and 2 are central nervous tissue

Cranial nerve 3 and 4 --- Mid brain

Cranial nerves 5-8 --- Pons

Cranial nerves 9-12 --- Medulla

Cranial nerves never cross, except trochlear nerve, and clinical findings are always on the same side of affected cranial nerves

Cranial nerve I: Olfactory nerve

Sensory cells in olfactory epithelium pass through cribiform plate to olfactory bulb, then to olfactory centers in the uncus and parahippocampal gyrus

Technique

- . Evaluate the patency of the nasal passages bilaterally by asking the patient to breathe in through their nose
- . Place non-irritating powder of coffee or solution of alcohol near the patent nostril and ask the patient to smell the object and report what it is, while the patient has closed her /his eyes. Switch nostril and repeat the test.
- . Ask the patient to compare the strength of the smell in each nostril

Anosmia (loss of sense of smell) results from damage to olfactory filaments after head trauma or invasive basal skull tumor

Parosmia (pleasant odor perceived as unpleasant) results from head trauma, sinus infection or drugs side effect

Cranial nerve II: Optic nerve

Fibers of optic nerve from the retina project to optic chiasm, and then to optic tract to end at occipital lobe via optic radiation

1. Examine the Optic Fundi

Technique of using fundoscope

. Darken the room. Switch on the ophthalmoscope light and turn the lens disc until you see the large round beam of white light

. Turn the lens disc to the 0 diopter (a diopter is a unit that measures the power of a lens to converge or diverge light)

- . Keep your finger on the edge of the lens disc so you can turn the disc to focus the lens when you examine the fundus
- . Hold the ophthalmoscope in your right hand to examine the patient's right eye; hold it in your left hand to examine the patient's left eye
- . Hold the ophthalmoscope firmly braced against the medial aspect of your bony orbit, with the handle tilted laterally at about a 20° slant from the vertical
- . Instruct the patient to look slightly up and over your shoulder at a point directly ahead on the wall
- . Place yourself about 50 cm away from the patient and at an angle 15° lateral to the patient's line of vision. Shine the light beam on the pupil and look for the orange glow in the pupil (*red reflex*)
- . Place the thumb of your other hand across the patient's eyebrow. Keeping the light beam focused on the red reflex, move in with the ophthalmoscope on the 15° angle toward the pupil until you are very close to it, almost touching the patient's eyelashes.
- . Observe the optic disc, physiological cup, retinal vessels and fovea. Note the pulsations of the retinal vessels, check for a blurring of the optic disc margin and a change in the optic disc's color.

Fundoscopic findings of papilledema (indicates presence of increased intracranial pressure)

- . Loss of venous pulsations
- . Blurring of the optic disc margin and retinal hemorrhages
- . Loss of physiologic cup and disc hyperemia

2. Test Visual Acuity

Allow the patient to use his glass or contact lens if available

Position the patient 6 meter in front of the Snellen eye chart

Have the patient cover one eye at a time with a card

Ask the patient to read progressively smaller letters until they can go no further

Record the smallest line the patient read successfully (6/6, 6/10, 6/16, etc...)

Repeat with the other eye

If patient can not see the top letter at 1meter, check whether patient can count fingers, see hand movements, or just see light



3. Test for color vision

Color perception, especially red, is affected in optic nerve disease before changes in visual acuity can be detected

Technique

- . Show the patient a red target one eye at a time

- . Ask the patient if there is a difference between the eyes
- . Red appears ‘washed out’ (desaturated) in the affected eye

The Ishihara test assesses both congenital color anomalies (color blindness) and acquired visual disorders

Most inherited color blindness occurs in males (sex-linked recessive inheritance), and it ranges from total color blindness (monochromatopsia) to confusion between colors, typically between red and green

4. Test for Visual Fields by Confrontation

Stand one meter in front of the patient and have him look into your eyes

Hold your hands about half meter away from the patient's ears, and wiggle a finger on one hand

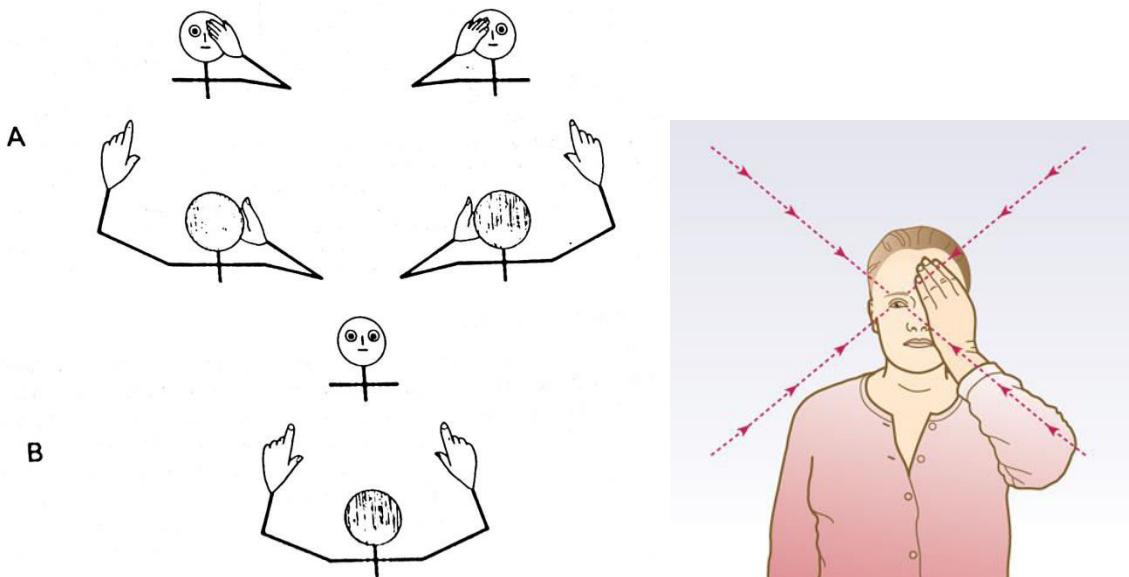
Ask the patient to indicate which side they see the finger move

Repeat two or three times to test both temporal fields

If an abnormality is suspected, test the four quadrants of each eye while asking the patient to cover the opposite eye with a card

Technique

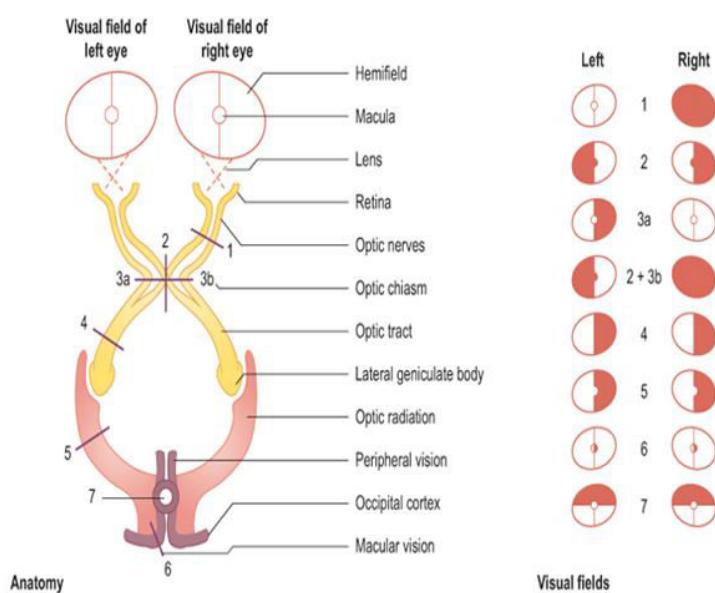
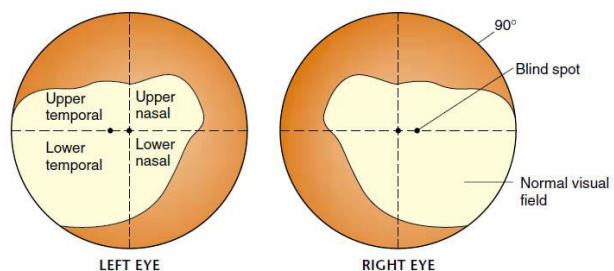
- . Sit or stand in front of the patient at a distance of one meter
- . The patient covers the right eye and the examiner cover the left eye, and the patient fixes her/his gaze on the examiner's opposite eye
- . Keep the wiggling finger of the examiner mid way and out of view for the examiner and the patient
- . Bring the wiggling finger slowly into view from out of view
- . Tell the patient to tell you ‘yes’ when she/he see the examiner’s wiggling finger come in to view
- . Each of the upper temporal, lower temporal, upper nasal and lower nasal quadrants is tested separately
- . Repeat the test in the other eye



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.1 Technique of testing visual fields

Testing of visual fields: A. Testing the temporal field in each eye separately; B. Testing both simultaneously for inattention hemianopsia



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 8.2 Visual field defects in optic pathway abnormalities

A lesion at 1 produces blindness in the right eye with loss of the direct light reflex. A lesion at 2 produces bitemporal hemianopia. Lesions at 3a and 3b produce binasal hemianopia (a rare disorder). A lesion at 4 produces right homonymous hemianopia with macular involvement. A lesion at 5 produces right homonymous hemianopia with sparing of the macular field. A lesion at 6 produces right homonymous central (macular) hemiscotoma. A lesion at 7 produces altitudinal hemianopia (upper half of contralateral visual field is lost)

Visual field abnormalities

Homonymous hemianopia is loss of vision on half of the visual field in both eyes. When the extent of visual loss in the homonymous fields of the two eyes is similar (congruous hemianopia) the lesion is likely to be postgeniculate. Incongruous hemianopia is more likely to be due to a lesion in the optic tract, chiasm or lateral geniculate.

Homonymous quadrantanopia is loss of vision limited to one quadrant of a visual field in both eyes. Contralateral homonymous superior quadrantanopia occurs in temporal lobe lesion, while contralateral homonymous inferior quadrantanopia occurs in parietal lobe lesion.

Bitemporal hemianopia: Loss of vision in the temporal (outer) halves of both fields is due to a lesion of the optic chiasm, often caused by compressive pituitary tumors.

Altitudinal hemianopia: The upper or lower half of the contralateral visual field is lost in altitudinal hemianopia.

5. Test Pupillary Reactions to Light

Dim the room lights as necessary

Ask the patient to look into the distance

Shine a bright light obliquely into each pupil in turn

Look for both the direct (same eye) and consensual (other eye) reactions

Assess pupil size in mm and any asymmetry or irregularity

If abnormal, proceed with the test for accommodation

6. Test Pupillary Reactions to Accommodation

Hold your finger about 10cm from the patient's nose

Ask them to alternate looking into the distance and at your finger

Observe the pupillary response in each eye

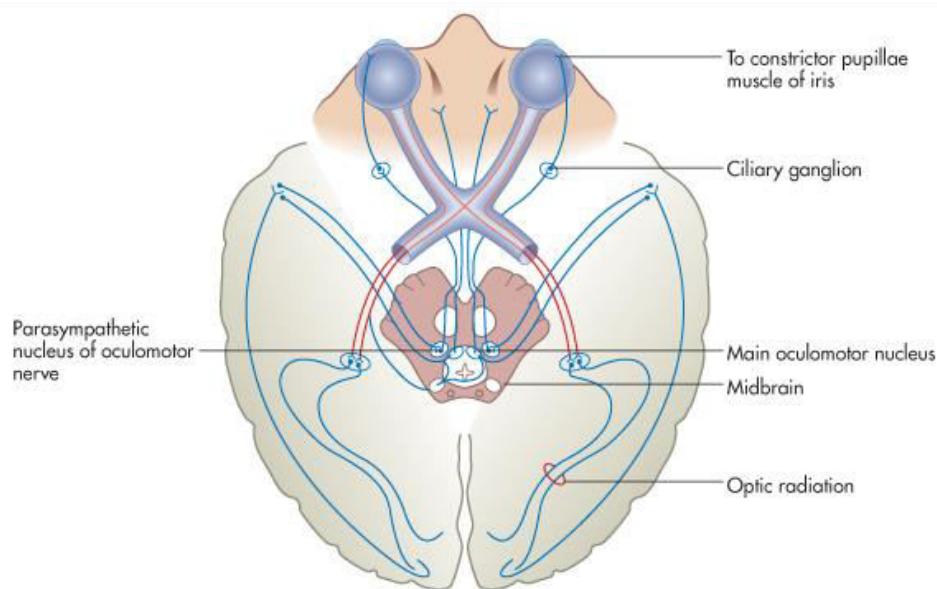
Cranial nerve III – Oculomotor nerve

Function: The 3rd cranial nerve controls eye adduction (medial rectus), abducted elevation of eye (superior rectus), abducted depression of eye (inferior rectus), elevation of eyelid (levator palpebrae superioris), and pupillary size (parasympathetic to the pupil)

The extraocular movements are controlled by the paired oculomotor, trochlear and abducens nerves. They are interconnected by the medial longitudinal fasciculus (MLF). MLF carries fibers from 6th nerve nucleus to contralateral 3rd nerve nucleus to integrate horizontal gaze.

Light reflex loop

Afferent fiber of light reflex arises from optic nerve to relay at Edinger-Westphal nucleus in brain stem, and efferent fiber arises from the nucleus and project into the oculomotor nerve, and then to ciliary ganglion and finally to ciliary muscle and iris (constrictor pupillae muscle)



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 8.3 Light reflex pathway

Light reflex and accommodation abnormalities

Third nerve lesion: Direct and consensual light reflex failure due to disrupted efferent arc of light reflex

Marcus-Gunn pupil: Relative afferent pupillary defect due to partial defect of afferent pathway of light reflex

Light-near dissociation: Constriction to accommodation is better than to light in partial oculomotor nerve lesion

Argyll-Robertson pupil: Bilateral small and irregular pupil, which reacts to accommodation but not to direct and consensual light reflex due to a lesion at pretectum (brain stem)

Adies tonic pupil: Unilateral absent or delayed pupillary constriction to light or to accommodation

Extraocular cranial nerve lesions cause diplopia, worse in the direction of action of the weak extraocular muscle

Technique

Observe for Ptosis

Test extraocular movements

Stand or sit 1-2 meter in front of the patient

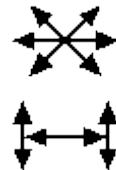
Ask the patient to follow your finger with their eyes without moving their head

Check gaze in the six cardinal directions using a cross or "H" pattern

Pause during upward and lateral gaze to check for nystagmus

Check convergence by moving your finger toward the bridge of the patient's nose

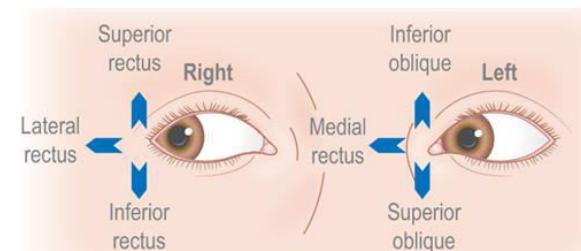
Test pupillary reactions to light (efferent loop of light reflex)



Cranial nerve IV – Trochlear nerve

Function: Trochlear nerve controls eye movement towards the midline ((looking down ward and in), function of superior oblique muscle)

Test Extraocular Movements (Inward and Down Movement)



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 8.4 Muscles involved in extraocular movement

Cranial nerve V – Trigeminal nerve

Function: Trigeminal nerve supplies sensation over the face, muscles of mastication, and mediates jaw jerk and corneal reflex

Technique:

Test Temporal and Masseter Muscle Strength

Ask patient to both open his mouth and clench his teeth

Palpate the temporal and masseter muscles as he does this

NB: Ipsilateral trigeminal nerve lesion results in jaw deviates towards the side of the lesion as the mouth opened

Test the Three Divisions for Pain Sensation

Explain what you intend to do

Use a suitable sharp object to test the forehead, cheeks, and jaw on both sides

Substitute with a blunt object and ask the patient to report "sharp" or "dull."



If you find an abnormality, then

Test the three divisions for temperature sensation with a tuning fork heated or cooled by water

Test the three divisions for sensation to light touch using a wisp of cotton

Jaw jerk: Refer to deep tendon reflex (DTR)

Testing the Corneal Reflex (afferent loop of corneal reflex is mediated by Trigeminal nerve)

Ask the patient to look up and away

From the other side, touch the cornea lightly with a fine wisp of cotton

Look for the normal blink reaction of both eyes

Repeat on the other side

NB: Use of contact lens may decrease this response

Cranial nerve VI – Abducens nerve

Function: Abducens nerve controls eye abduction (lateral rectus muscle)

Test extraocular movements- Lateral movement

Cranial nerve VII – Facial nerve

Function: Facial nerve control muscles of facial expression and taste on anterior 2/3 of tongue

Upper motor neuron (UMN) facial weakness

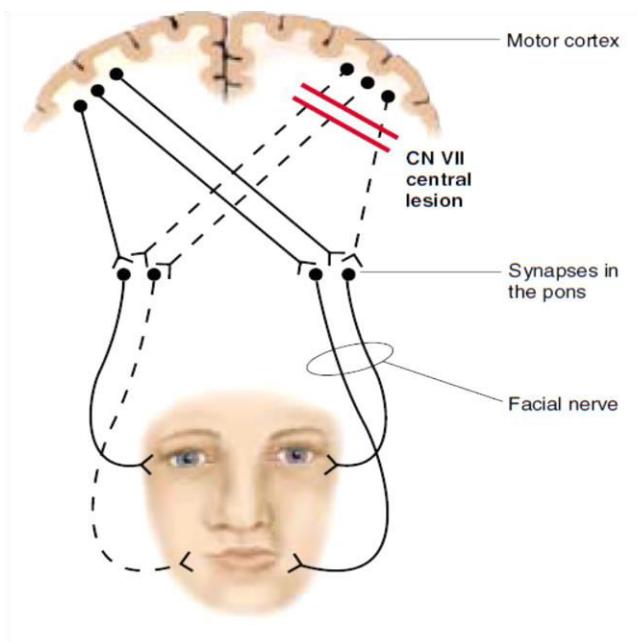
Unilateral UMN lesion causes contralateral weakness of cheek, mouth and platysma but not upper face

Face deviates towards the side of the lesion

Lower motor neuron (LMN) facial weakness

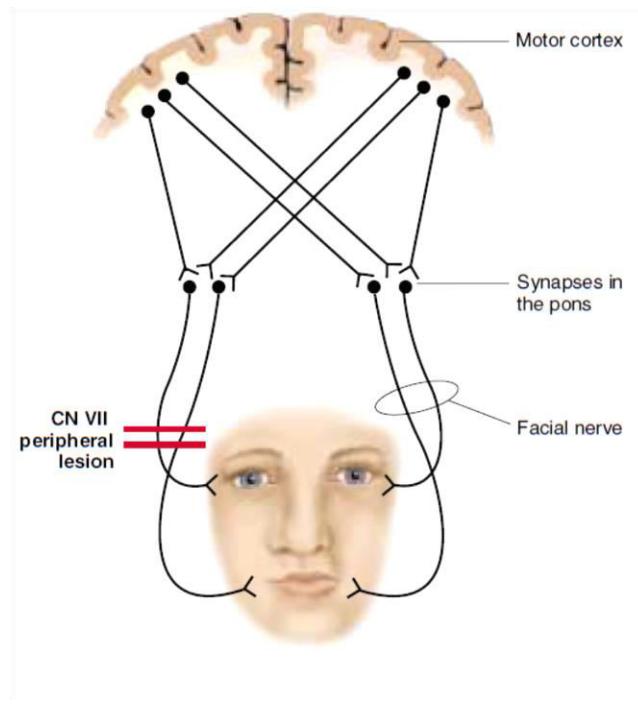
Nuclear and infranuclear lesions cause ipsilateral flaccid paralysis of upper and lower face

Face deviates away from the side of the lesion



© Lippincott. Bates, Bickley & Hoekelman. A Guide to Physical Examination and History Taking 12e

Fig 8.5 Central CN VII lesions: Cortico-pontine tract lesion causes contralateral paralysis of lower face only



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 8.6 Peripheral CN VII lesions: Peripheral facial nerve lesion causes ipsilateral paralysis of upper and lower face

Technique:

Observe for any facial droop or asymmetry

Ask patient to do the following (note any lag, weakness, or asymmetry)

Raise eyebrows, Close both eyes to resistance, Smile, Frown, Show teeth, and Puff out cheeks

Test the Corneal Reflex (efferent loop of corneal reflex is mediated by cranial nerve VII)

Cranial nerve VIII – Vestibulo-cochlear nerve

Vestibulo-cochlear nerve controls hearing (acoustic division) and balance (vestibular division)

Normally, air conduction is better than bone conduction in Rinne test, and does not lateralize in weber's test

Techniques:

Whispered voice test

- . Stand behind the patient
- . Whisper about 60 cm (arm length) from the ear you are testing
- . Mask hearing in the other ear by rubbing the fingers
- . Ask the patient to repeat your words

If a patient can hear a whispered voice at 60cm, his hearing is better than 30 dB i.e. normal

If abnormal, proceed with the Weber and Rinne tests

Test for lateralization (Weber test)

Use a 512 Hz or 1024 Hz tuning fork

Start the fork vibrating by tapping it on your opposite hand

Place the base of the tuning fork firmly on top of the patient's head or in the middle of forehead

Ask the patient where the sound appears to be coming from (normally in the midline)

Lateralizes to normal ear in sensorineural hearing loss

Lateralizes to affected ear in conductive hearing loss



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.7 Weber test

Test for Comparison of air and bone conduction (Rinne test)

Use a 512 Hz or 1024 Hz tuning fork

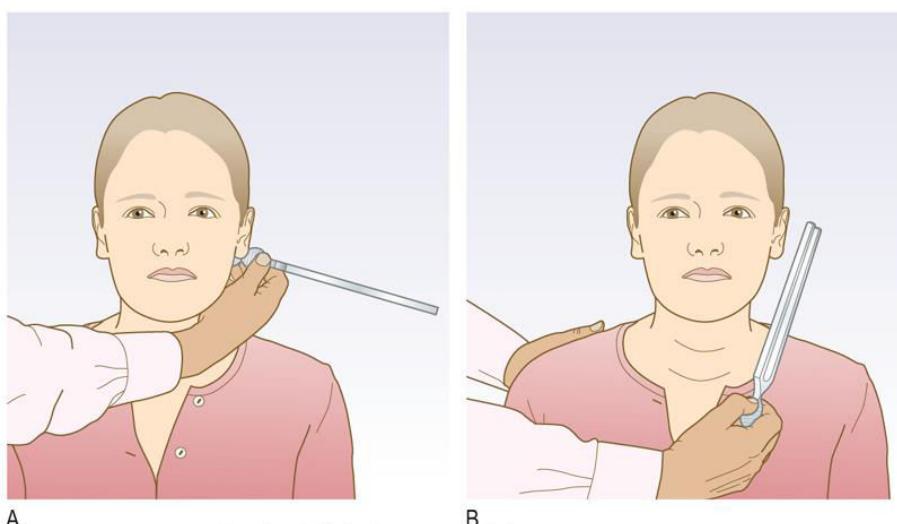
Start the fork vibrating by tapping it on your opposite hand

Place the base of the tuning fork against the mastoid bone behind the ear

When the patient no longer hears the sound, hold the end of the fork near the patient's ear (air conduction is normally greater than bone conduction)

Sensorineural hearing loss: Air conduction > Bone conduction

Conductive hearing loss: Bone conduction > Air conduction



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.8 Rinne test

Dix-Hallpike maneuver

Commonly used in patients with benign positional vertigo (vestibular function)

Dix-Hallpike maneuver assesses the effect of positional change

Technique:

- . Ask the patient to sit upright on the examining couch
- . Turn the patient's head 45° to one side
- . Lie back rapidly with her/his head extended over the end of the bed (keep eyes open)
- . Watch eyes for nystagmus. Repeat the test, turning the head to other side
- . In positional vertigo, torsional nystagmus beating to the lower ear with feeling of vertigo persisting for 5-30 minutes
- . Positional vertigo is thought to be caused by collected calcium debris (otoconia or ear rocks) in the posterior semicircular canal



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.9 Dix-Hallpike's maneuver: Technique for positional nystagmus

Cranial nerves IX and X – Glossopharyngeal and Vagus nerves

Function: Glossopharyngeal and vagus nerves control swallowing

Unilateral vagus nerve lesion causes failure of elevation of the uvula on the affected side. Bilateral vagus nerve lesion causes nasal regurgitation of food or fluid, dysphagia and palatal dysarthria with bovine voice.

Technique:

Listen to the patient's voice, is it hoarse or nasal?

Ask the patient to cough; assess strength of cough

Water swallow test (for conscious patient)

- . Administer 3 teaspoons of water
- . Observe for swallow, cough and voice quality after each teaspoon of water
- . Watch the patient while the patient swallows a glass of water if no problem with above procedure

Ask patient to say "Aaah", and watch the movements of the palate and Uvula. Unilateral damage to the X nerve leads to deviation of the uvula to normal side when the soft palate is elevated saying 'Aaah'.

- . Ask the patient to puff out the cheeks with the lips tightly closed. Air escapes audibly via the nose in palatal weakness

Testing Gag reflex (Unconscious/Uncooperative patient)

- . Stimulate the back of the throat on each side
- . It is normal to gag after each stimulus

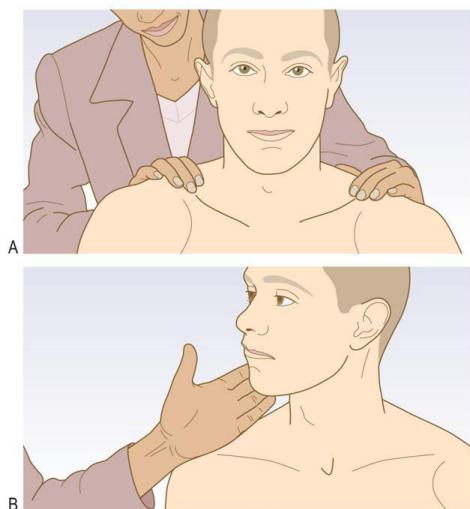
Cranial nerve XI – Accessory nerve

Accessory nerve innervates trapezius and sternomastoid muscle and coordinates head movement at the neck

Technique:

- . Look for atrophy or asymmetry of the trapezius muscles from behind
- . Ask patient to shrug shoulders against resistance
- . Ask patient to turn their head against resistance, and watch and palpate the sternomastoid muscle on the opposite side

Normally, the patient is able to shrug shoulders and contraction of the sternomastoid muscle against resistance



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.10 Testing the trapezius and left sternomastoid muscles

Cranial nerve XII – Hypoglossal nerve

Function: Hypoglossal nerve controls taste in posterior 1/3 of tongue, and tongue movement

Technique:

- . Listen to the articulation of the patient's words
- . Observe the tongue as it lies in the mouth for atrophy and fasciculation
- . Ask patient to protrude tongue, move tongue from side to side
- . Test strength by asking the patient to press the tongue against the inside of each cheek while you press from the outside with your finger

Tongue deviates to the affected side in ipsilateral hypoglossal nerve lesion

Motor system

Weakness is reduction in muscle strength, and occurs in lower and upper motor neuron lesion

Generally, weakness in lower motor neuron lesion causes flaccid paralysis while weakness in upper motor neuron lesion causes spastic paralysis

Technique:

Inspection

- . Look position of the extremities after repositioning by the examiner
- . Compare left to right extremities, and proximal to distal extremities
- . Look for muscle Symmetry
- . Look for presence of muscle bulk atrophy and fasciculation

Pay attention to the hands, shoulders, and thighs

Muscle Tone

Muscle tone is resistance of a muscle to passive stretch

There is normally a small, continuous resistance to passive movement

Technique

- . Ask the patient to relax and ‘go floppy’
- . Passively move each joint both slowly and quickly

Upper limb

- . Hold the patient’s hand as if shaking hands, other hand supporting his elbow

- . Rotate the forearm, and flex and extend the wrist, elbow and shoulder

Lower limb

- . Roll and rotate the leg from side-to-side
- . Flex and extend patient's ankle and knee
- . Observe for decreased (flaccid) or increased (spastic) tone

Types of spasticity

Klasp-knife spasticity: velocity-dependent spasticity with sudden release after reaching a maximum, commonly seen in pyramidal tract lesions

Lead-pipe spasticity: Increased tone present throughout the range of motion is observed in frontal lobe disease

Cogwheel spasticity: Spasticity intertwined by tremor, commonly seen in extrapyramidal (basal ganglia) lesions

Muscle Strength (power)

Muscle strength indicates the capacity of muscle to exert force and expend energy

Test strength by having the patient move against your resistance

Always compare one side to the other

Muscle weakness causes loss of speed or agility of movement and a decrease in the range or amplitude of movement

Table 8.4 Grading of strength on a scale from 0 to 5 "out of five":

Grading Motor Strength	
Grade	Description
0/5	No muscle movement
1/5	Visible flicker muscle movement, but no movement at the joint
2/5	Movement at the joint, but not against gravity
3/5	Movement against gravity, but not against added resistance
4/5	Movement against resistance, but less than normal
5/5	Normal strength

Test the following:

Flexion at the elbow (C5, C6, biceps)

Extension at the elbow (C6, C7, C8, triceps)

Extension at the wrist (C6, C7, C8, radial nerve)

Squeeze two of your fingers as hard as possible ("grip" C7, C8, T1)

Finger abduction (C8, T1, ulnar nerve)

Opposition of the thumb (C8, T1, median nerve)

Flexion at the hip (L2, L3, L4, iliopsoas)

Adduction at the hips (L2, L3, L4, adductors)

Abduction at the hips (L4, L5, S1, gluteus medius and minimus)

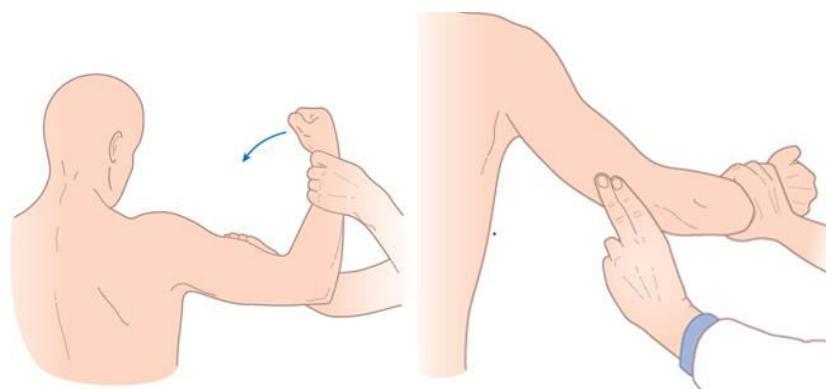
Extension at the hips (S1, gluteus maximus)

Extension at the knee (L2, L3, L4, quadriceps)

Flexion at the knee (L4, L5, S1, S2, hamstrings)

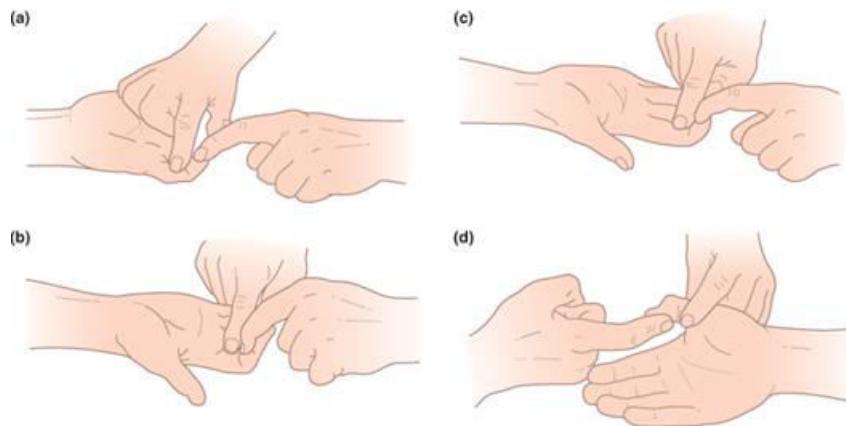
Dorsiflexion at the ankle (L4, L5)

Plantar flexion (S1)



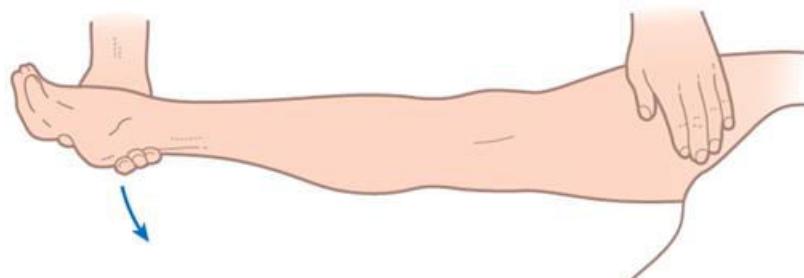
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 8.11 Flexion and extension at the elbow (Biceps and triceps)



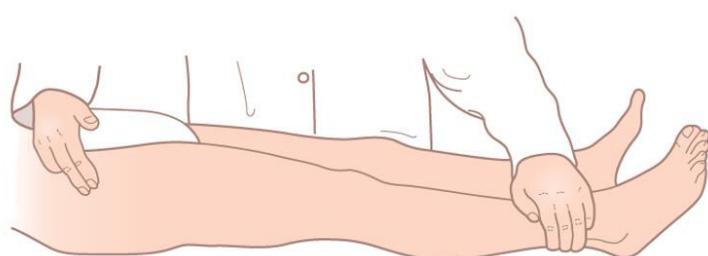
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 8.12 Testing (a) flexor digitorum superficialis; (b) flexor digitorum profundus I and II; (c) flexor digitorum longus; (d) flexor pollicis longus



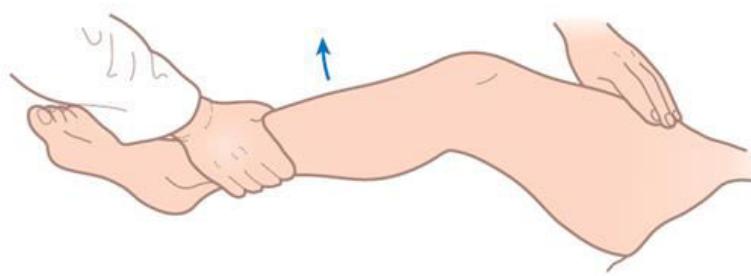
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 8.13 Testing adductors of the hip



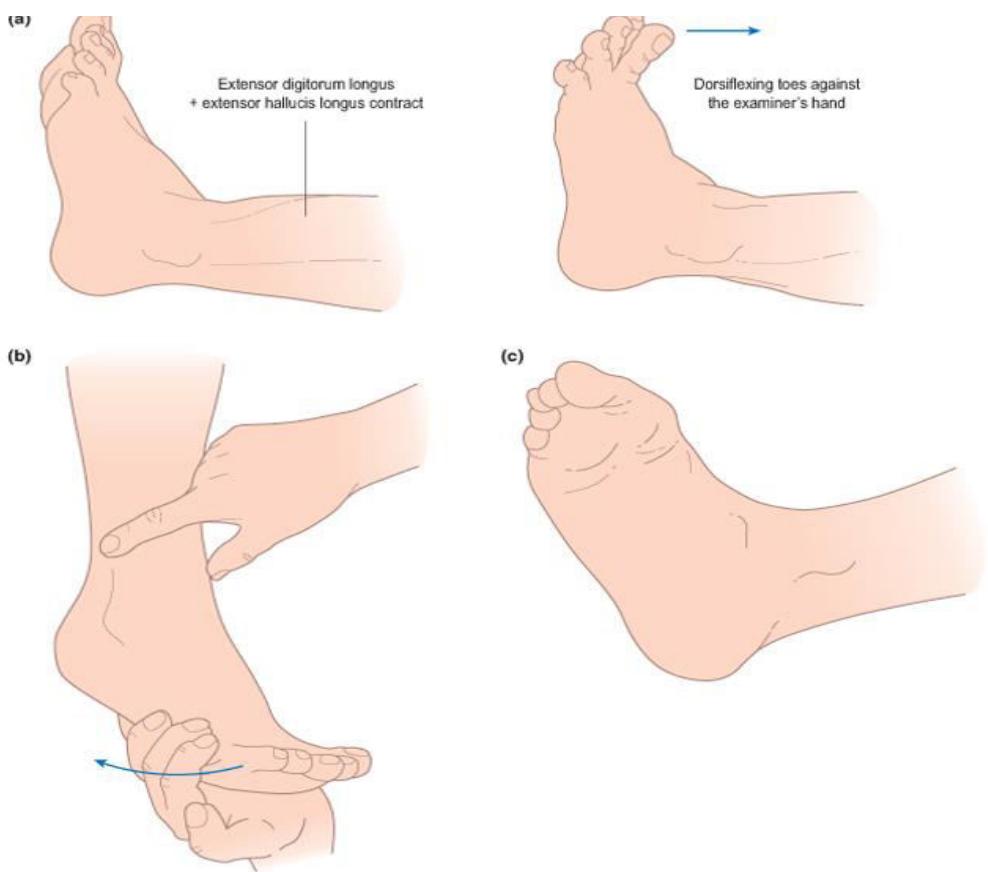
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 8.14 Testing the hip abductors (gluteus medius and minimus)



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 8.15 Testing the quadriceps femoris



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 8.16 Testing (a) Dorsiflexion of foot; (b) Eversion of foot; (c) Plantar flexion of foot

Reflexes

Deep Tendon Reflexes

The patient must be relaxed and positioned properly before starting

Reflex response depends on the force of your stimulus. Use no more force than you need to provoke a definite response

Reflexes can be reinforced by having the patient perform isometric contraction of other muscles (clenched teeth for upper limb reflexes, and hooking up fingers of both hands for lower limb reflexes), and named as Jendrassik's maneuver . Use reinforcement whenever a reflex appears to be absent.

Table 8.5 Grading scale of deep tendon reflex

Deep Tendon Reflex Grading Scale	
Grade	Description
0	Absent (with Jendrasik maneuver)
1+ or +	Hypoactive (less brisk)
2+ or ++	"Normal" (brisk)
3+ or +++	Hyperactive without clonus (very brisk)
4+ or +++++	Hyperactive with clonus

Technique:

Biceps reflex (C5, C6)

The patient's arm should be partially flexed at the elbow with the palm down

Place your thumb firmly on the biceps tendon

Strike your thumb with the reflex hammer

You should feel the response even if you can't see it

Triceps reflex (C6, C7)

Flex the patient's arm at the elbow and hold it close to the chest

Strike the triceps tendon above the elbow with the broad side of the hammer

Observe for contraction of triceps

Brachioradialis/ Supinator reflex (C5, C6)

Have the patient rest the forearm on the abdomen or lap

Strike the radius about 1-2 inches above the wrist

Watch for flexion and supination of the forearm

Finger Jerk

Place your middle and index fingers across the palmar surface of the patient's proximal phalanges

Tap your own fingers with the hammer

Observe for flexion of the patient's fingers

Hoffman's reflex

- . Place your right index finger under the distal interphalangeal joint of the patient's middle finger
- . Using your right thumb, flick the patient's finger downwards
- . Look for any reflex flexion of the patient's thumb

Knee/ Patellar reflex (L2, L3, L4)

Have the patient sit or lie down with the knee flexed

In supine positioned patient with the knee in partial flexion:

Place your left pronated arm below the patient's right knee and left hand over the patient's left knee, and strike the right patellar tendon just below the patella

Place your left supinated arm below the patient's right and left knees, and strike the left patellar tendon just below the patella

Note contraction of the quadriceps and extension of the knee

Ankle (S1, S2)

Dorsiflex the foot at the ankle

Strike the Achilles tendon

Watch for contraction of gastrocnemius muscle and plantar flexion at the ankle

Jaw Jerk (Trigeminal nerve)

- . Partially open the mouth and put your index finger on the jaw
- . Gently strike the index finger on jaw with the hammer
- . Watch for closure of the mouth

Clonus

Test for ankle clonus

If the reflexes seem hyperactive, test for ankle clonus

Support the knee and ankle in a partly flexed position

Quickly dorsiflex and partially evert the foot

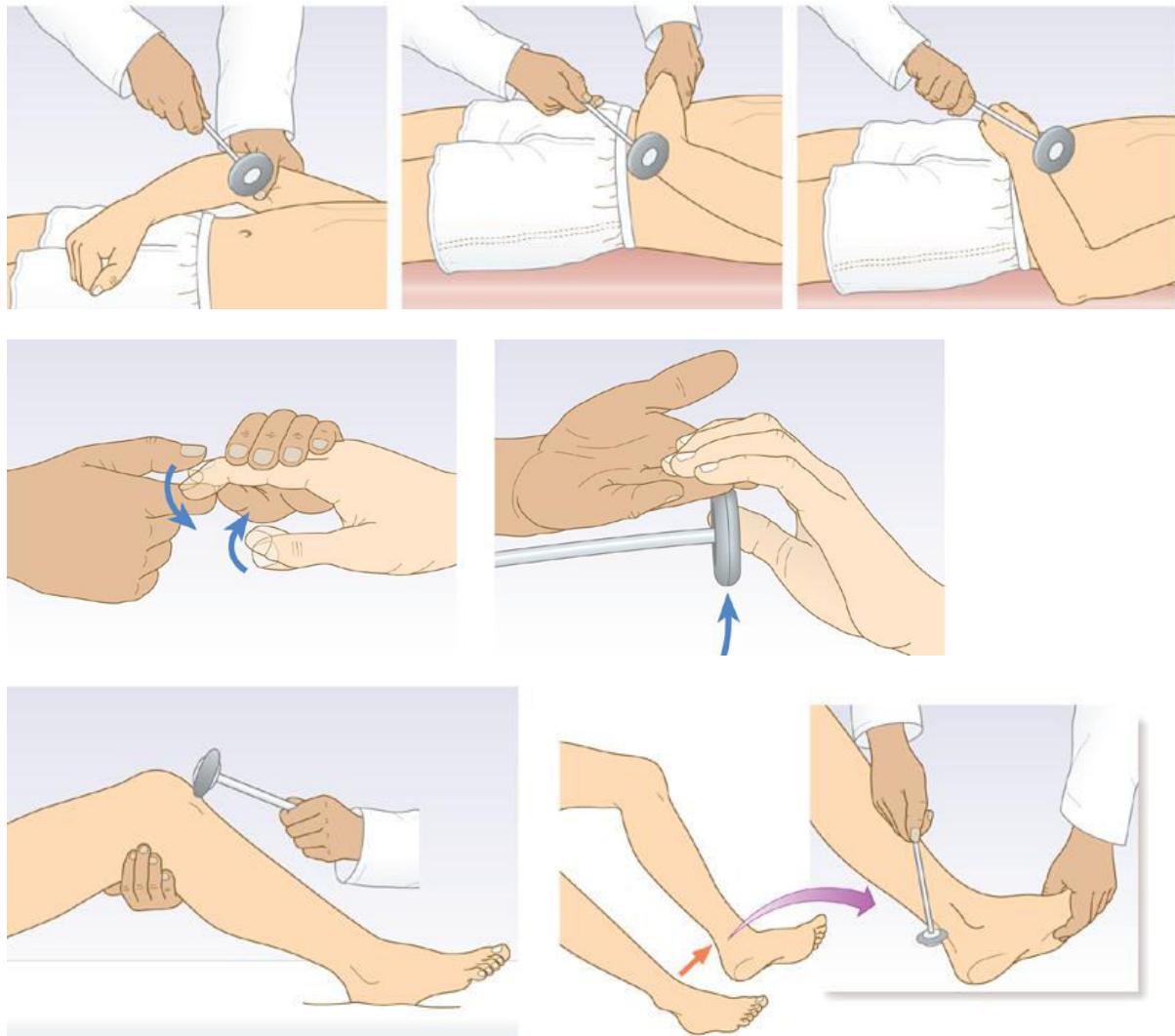
Observe for sustained rhythmic oscillations (≥ 3 rhythmic oscillations)

Test for knee clonus

Partially flex the knee

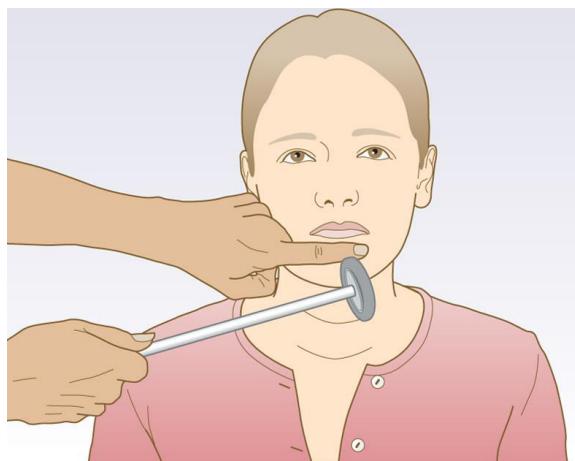
Hold the patella with thumb and forefinger, and quickly push downwards

Observe for sustained rhythmic oscillations



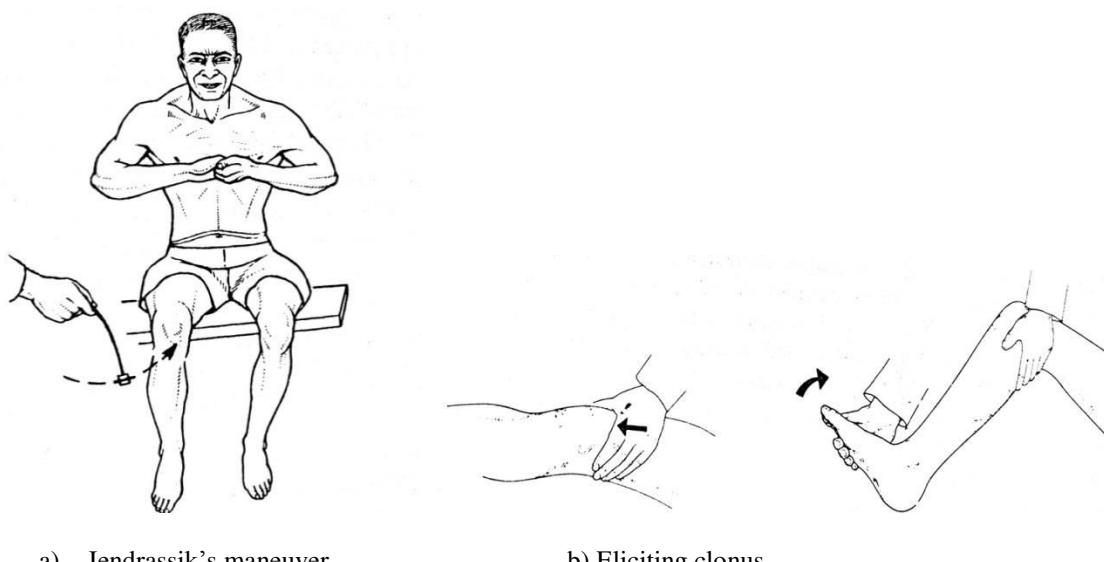
© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.17 Technique of eliciting deep tendon reflexes: Biceps reflex, triceps reflex, brachioradialis (supinator) reflex, finger reflex (look for eliciting Hoffman sign), knee reflex, and ankle reflex



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.18 Technique of eliciting Jaw jerk



a) Jendrassik's maneuver

b) Eliciting clonus

© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.19 Technique of reinforcement (Jendrassik's maneuver) while eliciting knee jerk, and eliciting the knee and ankle clonus

Superficial reflexes

Plantar response (L5/S1)

Stroke the lateral aspect of the sole of each foot with the end of a reflex hammer or key

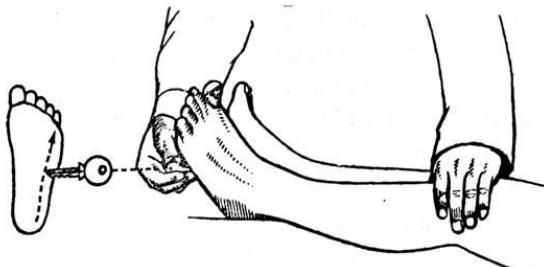
Note movement of the toes, normally flexion (withdrawal)

Extension of the big toe with fanning of the other toes is abnormal. This is referred to as a positive Babinski sign (sign of upper motor neuron lesion)



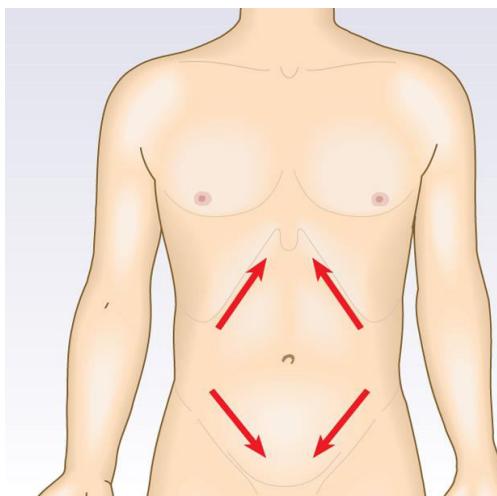
Alternative methods of eliciting Babinski sign: Extension of big toe with fanning of other toes is also obtained by pressing heavily along the medial border of the tibia (Oppenheim's sign), squeezing the calf or achilles tendon (Gordon's sign), stroking the lateral border of foot (Chadok's sign) and pressing the dorsum of big toe (Bing sign)

No response to plantar reflex indicates lower motor neuron lesion



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.20 Technique of eliciting plantar response (Positive Babinski's sign as shown)



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.21 Technique of eliciting abdominal reflex

Abdominal (T8, T9, T10, T11, T12)

Use a blunt object such as a key or tongue blade

Stroke the abdomen lightly on each side in an upward and downward direction above (T8, T9, T10) and below the umbilicus respectively (T10, T11, T12)

Note the contraction of the abdominal muscles and deviation of the umbilicus towards the stimulus

Cremasteric reflex (L1 and 2)

. Stroke the upper medial thigh; look for upward movement of testicle

Anal reflex (S3 and 4)

. Stroke or scratch the skin near the anus, notice for contraction of anal sphincter

Bulbocavernosus reflex (S3 and 4)

. Gently pinch the dorsum of glans penis, Look for contraction of bulbocavernosus muscle

Scapular reflex (C5-T1)

. Stroke the skin in interscapular region; look for contraction of scapular muscles

Corneal reflex (5th and 7th cranial nerves): refer to cranial nerves

Table 8.6 Comparison of lower (LMN) and upper motor neuron (UMN) lesion

Variables	LMN lesion	UMN lesion
Fasciculation	present	absent
Atrophy	present	none, mild
Tone	hypotonia	hypertonia
Reflex	hyporeflexia	hyperreflexia
Plantar reflex	no response	Up-going/Babinski +ve

Arm Drift

Pronator Drift

Ask the patient to stand for 20-30 seconds with both arms straight forward, palms up, and eyes closed

Instruct the patient to keep the arms still for a while

Unable to maintain extension and supination of arm, and "drift into pronation" indicate 'upper motor neuron' lesion

Cerebellar drift

Ask the patient to stand for 20-30 seconds with both arms straight forward and palms up

Instruct the patient to keep the arms still while you tap them briskly downward

Excessive upward rebound movements of arms indicate cerebellar lesion

Parietal drift

Ask the patient to stand for 20-30 seconds with both arms straight forward and palms up

Displace the ulnar border of the supinated hand

Lateral drift of the arm indicates parietal lesion

Coordination and Gait

Technique:

Rapid Alternating Movements

Ask the patient to repeatedly tap the palm of one hand with the palm and back of your opposite hand as quickly and regularly as possible

Ask the patient to repeatedly strike one hand on the thigh, raise the hand, turn it over, and then strike it back as fast as possible

Ask the patient to repeatedly tap the distal thumb with the tip of the index finger as fast as possible

Ask the patient to tap your hand with the ball of each foot as fast as possible

Point-to-Point Movements

Ask the patient to repeatedly touch your index finger and his nose alternately. Move your finger about as the patient performs this task (Finger-to- Nose test)

Ask the patient to move his arm and return to your finger with their eyes closed

Ask the patient to place one heel on the opposite knee and run it down the shin to the big toe (Heel-to-Shin test). Repeat with the patient's eyes closed

Romberg's test

Be prepared to catch the patient if they are unstable

Ask the patient to stand with the feet together and eyes closed for 5-10 seconds without support

The test is said to be positive if the patient becomes unstable (indicating a vestibular or proprioceptive problem)

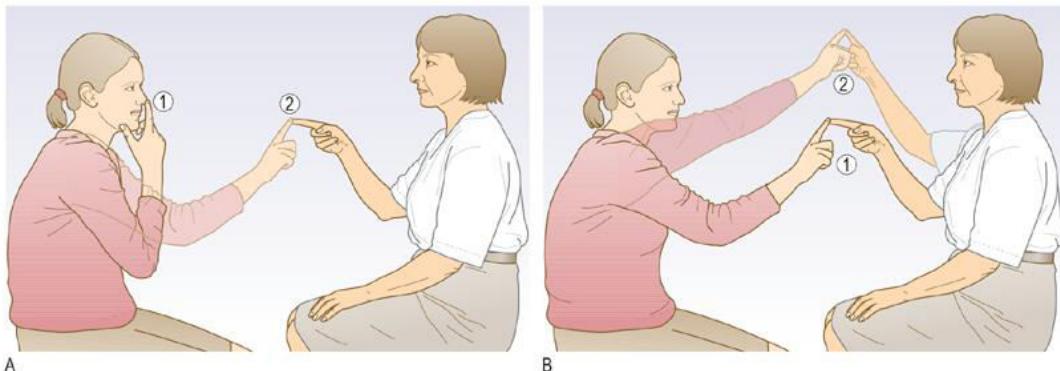
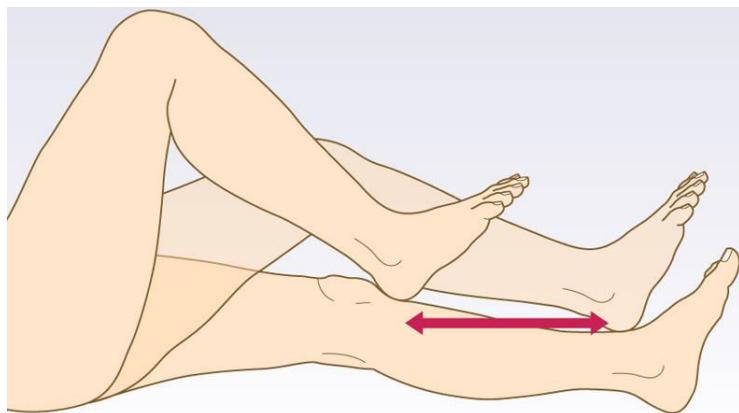


Fig 8.22 Performing Finger-to-Nose test A) Patient touches the tip of her nose then the examiner's finger B) The examiner moves her finger from side-to-side or towards-or-away from the patient as the patient touches examiner's finger



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.23 Performing Heel-to-Shin test

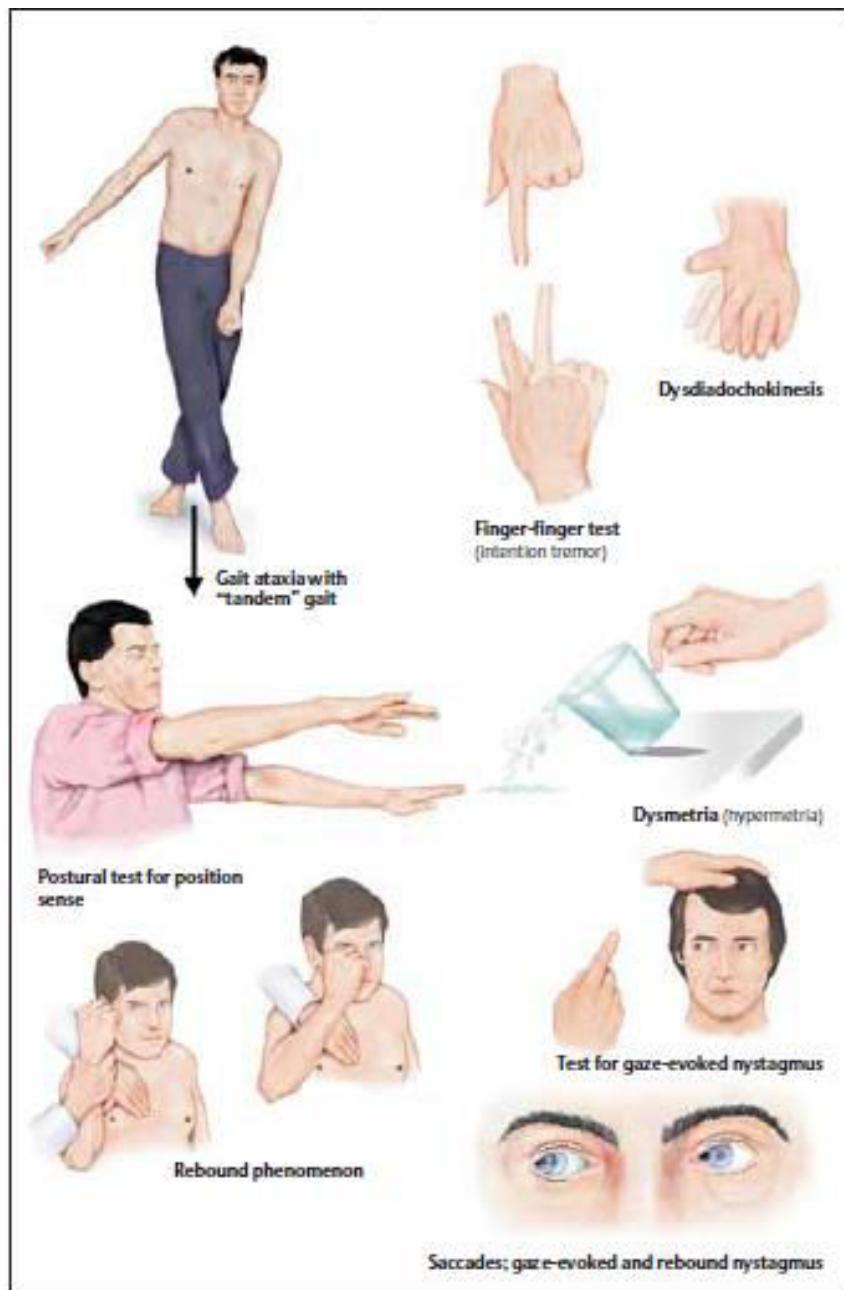
Gait

Difficulty in maintaining an upright posture while standing or walking indicates gait disorder

Technique:

Ask the patient to:

- . Walk across the room, turn and come back
- . Walk heel-to-toe in a straight line (tandem walk)
- . Walk on their toes in a straight line
- . Walk on their heels in a straight line
- . Hop in place on each foot
- . Do shallow knee bend
- . Rise from a sitting position



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 8.24 Cerebellar signs: Difficulty in coordination and balance

Common types of gait

Natural Gait

The patient should be able to walk with a smooth, coordinated gait with normal associated movement of the upper extremities

Hemiplegic Gait

The arm is held in adduction and internal rotation with flexion at the elbow, wrist and fingers; and the leg is in extension at the hip, knee and ankle. Therefore, the patient has to circumduct or swing the leg around to step forward. This type of gait is seen with a pyramidal tract lesion

Cerebellar ataxic Gait

Wide-based with truncal instability and irregular lurching steps which results in lateral veering and falling. This type of gait is seen in cerebellar lesion.

Sensory ataxic Gait

Wide-based, maintained balance during standing with eyes open and rapid loss of balance with fall with loss of visual input (positive Romberg sign). This type of gait is seen in large-fiber peripheral neuropathy.

Sensory system

Positive sensory phenomena

- . Hyperesthesia- pain in response to touch
- . Allodynia- non painful stimuli is perceived as painful
- . Hyperalgesia- severe pain in response to a mildly noxious stimulus
- . Hyperpathia- encompasses all the phenomena described by hyperesthesia, allodynia and hyperalgesia

Negative sensory phenomena

Impaired or absent primary sensory modalities (to touch, pain, and temperature)

- . Hypoesthesia- reduction of cutaneous sensation to a specific type of testing (pressure, light touch, warm or cold stimuli)
- . Anesthesia- complete absence of skin sensation to pin prick
- . Hypalgesia- reduced pain perception (nociception) like pricking quality of a pin

Localization by sensory abnormalities

Nerve and root lesions

Nerve lesion

Polyneuropathy: simultaneous involvement of many nerve trunks

Polyneuropathy is nerve-length dependent, “stocking-glove” type, and generally graded, distal and symmetric in distribution of sensory deficit

Small fiber polyneuropathy- burning, painful dysesthesias with reduced pin prick and thermal sensation, sparing proprioception, motor function and deep tendon reflexes (sensory dissociation)

Large fiber polyneuropathy- position sense deficit, imbalance, absent tendon jerks and variable motor dysfunction with preserved most cutaneous sensation

Root lesion

Deep, aching radicular pain along the course of a related nerve trunk

Spinal cord lesion

Sensory level (all primary sensory modalities lost below the level of cord lesion)

Brain stem lesion

Harlequin pattern of sensory disturbance in lateral medullary lesion: touch and pain sensations are affected in ipsilateral face and contralateral body

Thalamic lesion

Thalamic pain - persistent, unrelenting hemi-painful state

Cortical lesion

Contralateral hemineglect, hemi-inattention, cortical sensory loss (impaired two point discrimination, sensory extinction, astereognosis, agraphesthesia)

Primary sensory tests

General

Explain each test before you do it

Unless otherwise specified, the patient's eyes should be closed during the actual testing

Compare symmetrical areas on the two sides of the body, and also compare distal and proximal areas of the extremities

When you detect an area of sensory loss, map out its boundaries in detail

Vibration

Use a low pitched tuning fork (128Hz)

Test with a non-vibrating tuning fork first to ensure that the patient is responding to the correct stimulus

Place the distal end of the fork over the distal interphalangeal joint of the patient's index fingers and big toes

Ask the patient to tell you if they feel the vibration

If vibration sense is impaired proceed proximally:

Wrists → Elbows → Medial malleoli → Patellas → Anterior superior iliac spines → Spinous processes
→Clavicles

Position Sense

Grasp the patient's big toe at the sides

Show the patient "up" and "down" of big toe

Ask the patient to identify the direction you move the big toe with the patient's eyes closed

If position sense is impaired move proximally to test the ankle joint.

Test the fingers in a similar fashion.

If indicated move proximally to the metacarpophalangeal joints, wrists, and elbows

Pain

Use a suitable sharp object to test "sharp" or "dull" sensation.

Test the following areas:

Shoulders (C4)

Inner and outer aspects of the forearms (T1 and C6)

Thumbs and little fingers (C6 and C8)

Front of both thighs (L2)

Medial and lateral aspect of both calves (L4 and L5)

Little toes (S1)

Temperature

Often omitted if pain sensation is normal

Use a tuning fork heated or cooled by water, and ask the patient to identify "hot" or "cold" respectively

Test the following areas:

Shoulders (C4)

Inner and outer aspects of the forearms (C6 and T1)

Thumbs and little fingers (C6 and C8)

Front of both thighs (L2)

Medial and lateral aspect of both calves (L4 and L5)

Little toes (S1)

Light Touch

Use a fine wisp of cotton or your fingers to touch the skin lightly

Ask the patient to respond when ever a touch is felt

Test the following areas:

Shoulders (C4)

Inner and outer aspects of the forearms (C6 and T1)

Thumbs and little fingers (C6 and C8)

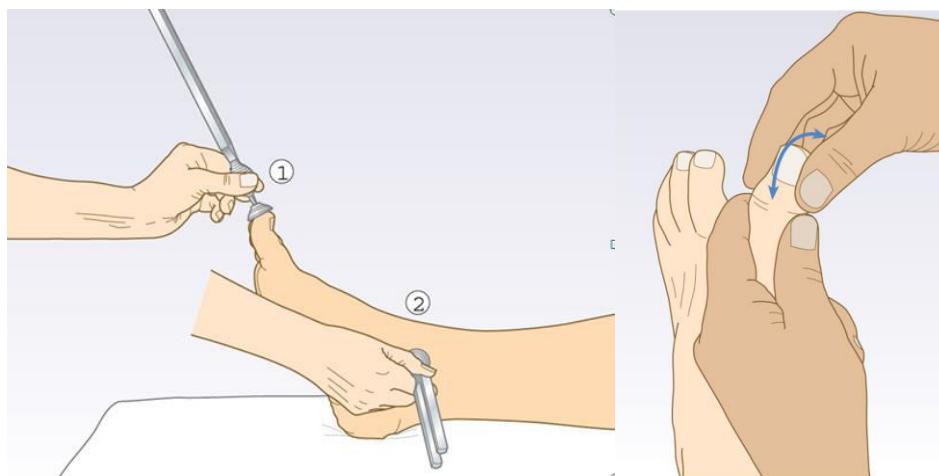
Front of both thighs (L2)

Medial and lateral aspect of both calves (L4 and L5)

Little toes (S1)

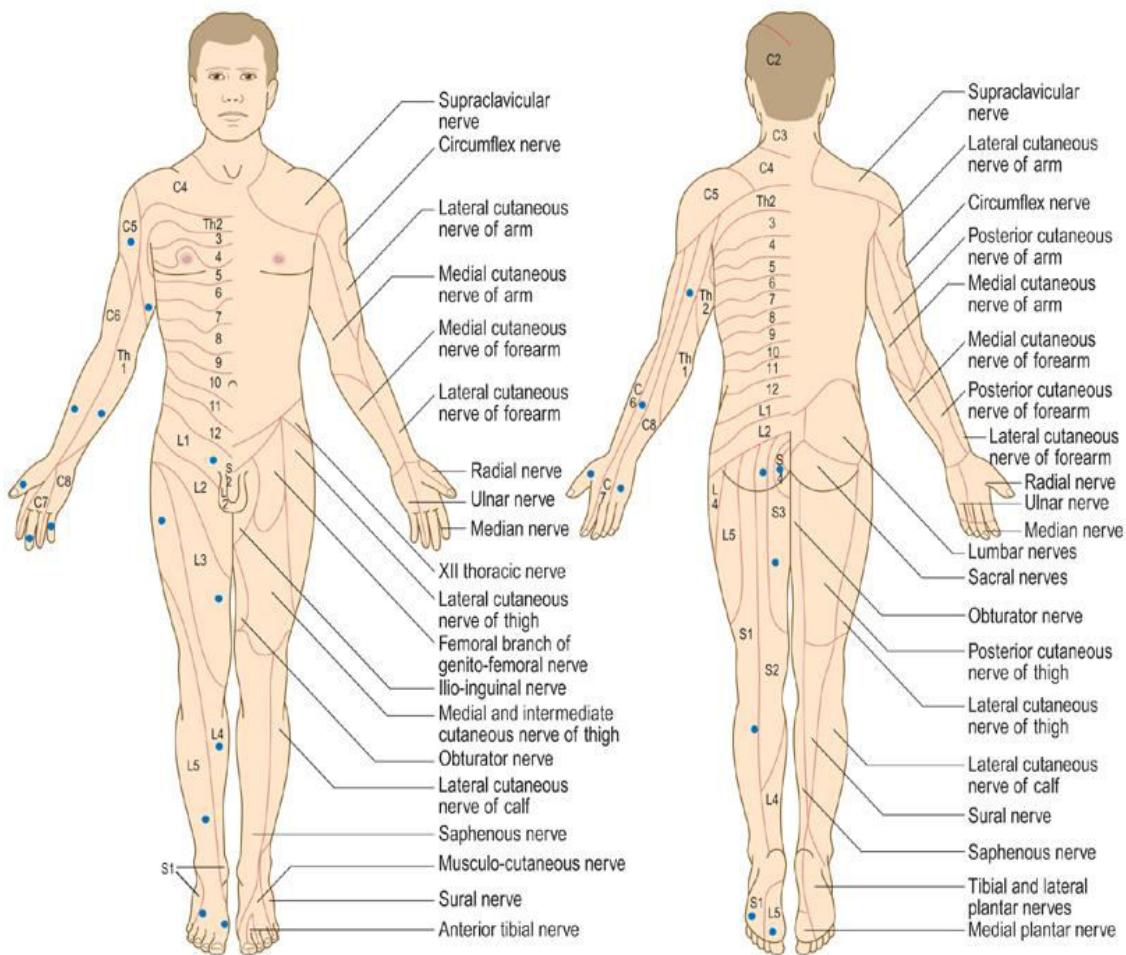
Dermatomal Testing

You need to check primary sensory modalities (touch and pain) following a dermatome.



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.25 Testing vibration sensation on big toe and medial malleolus, and position sense in the big toe



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.26 Segmental and peripheral nerve innervations: anterior and posterior view

Cortical sensory tests

Since these tests are dependent on touch and position sense, they can not be performed when the tests above are clearly abnormal

Cortical sensory loss indicates damage to sensory cortex (parietal lobe) and includes the following:

Astereognosis: failure to identify common objects placed in the hand while the eyes are closed

Agraphesthesia: failure to identify a number written on the palm of hand

Impaired two-point discrimination: failure to recognize two-point separation of 2-4 mm on the lips and finger pads, 8-15 mm on the palms and 3-4 cm on the shins

Extinction or simultanagnosia: Perceiving only one of the stimuli on double stimulation of symmetric sides of the body

Technique:

Graphesthesia

With the blunt end of a pen or pencil, draw a large number in the patient's palm

Ask the patient to identify the number

Stereognosis

Place a familiar object in the patient's hand (coin, paper clip, pencil, etc.)

Ask the patient to tell you what it is

Two Point Discrimination

Use an opened paper clip to touch the patient's finger pads in two places simultaneously

Alternate irregularly with one point touch

Ask the patient to identify "one" or "two."

Find the minimal distance at which the patient can discriminate

Double simultaneous stimulation (DSS)

. Touch both sides of the body with a wisp of cotton at the same time

. Ask the patient whether both stimuli are perceived

NB: Impaired two-point discrimination, astereognosis, agraphesthesia and extinction are noticed in cerebral cortical lesion

Primitive reflexes

Observed in frontal lobe disease

. Snout reflex: Touching the lips causes pouting lip movements

. Sucking reflex: Rubbing the chin causes sucking lip movements

. Palmo-mental reflex: Scratching the palm produces ipsilateral puckering of the chin

. Grasp reflex: The patient tends to grasp objects when placed on the hand, and continually grasp as you try to remove from his hand

. Glabellar reflex: Repeatedly tap between his eye brows with the tip of your index finger. Blink response persists after 3-4 taps

Signs of meningeal irritation

Kernig's sign

Place patient supine with hip flexed at 90°

Attempt to extend the leg at the knee >135°

The test is positive when there is pain in the lower back or resistance to extension

Brudzinski's sign

Place patient in the supine position

Passively flex the head towards the chest

The test is positive when there is flexion of the knees and hips while passively flexing the head

Neck stiffness

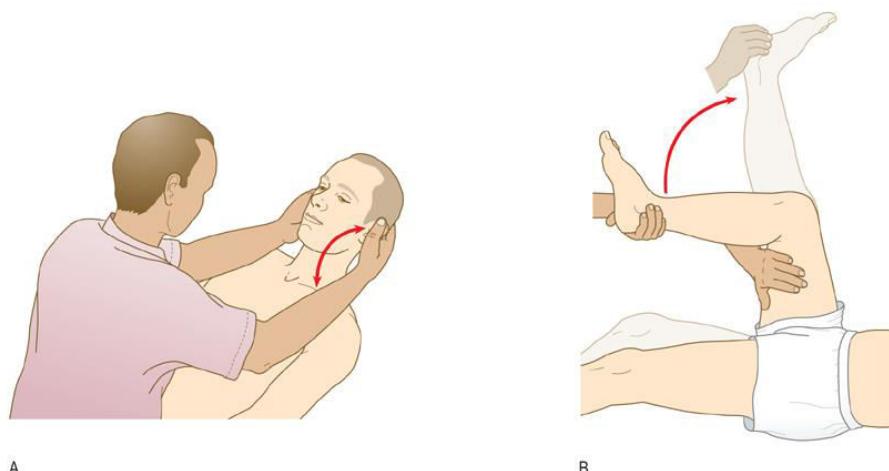
The examiner passively flexes patient's neck towards the chest

The test is positive when there is pain and resistance to neck flexion

Jolt accentuation of headache

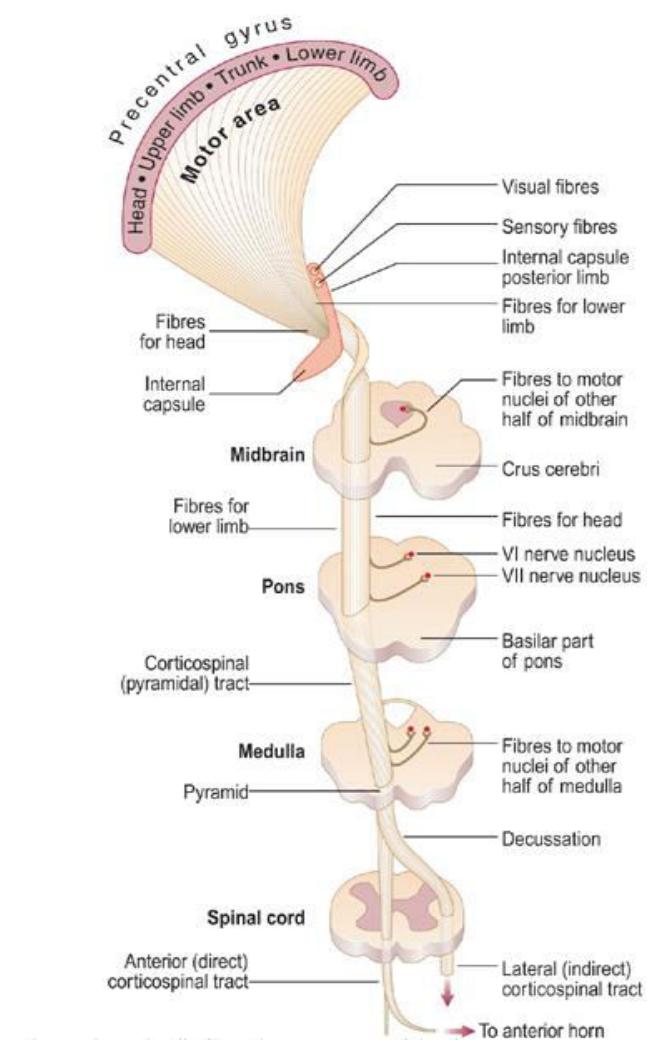
The examiner rotates patient's head horizontally two to three times per second

The test is positive if the patient reports exacerbation of his headache



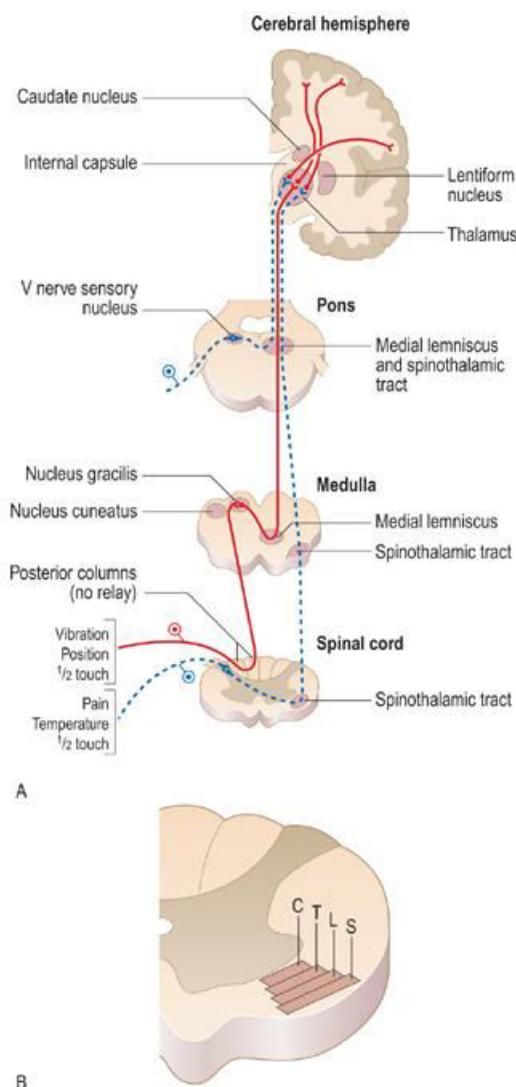
© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th ed

Fig 8.27 Testing meningeal irritation: A) Neck stiffness and B) Kernig's sign



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th ed

Fig 8.28 Motor pathways: Corticospinal tracts



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.29 Sensory pathways: A) Spinothalamic tract and B) Layering of spinothalamic tract in the cervical region

Localization in neurological deficit

Lesions affecting peripheral nerve

Key questions in approach to neuropathy

1. What systems are involved?

– Motor, sensory, autonomic, or combinations

2. What is the distribution of weakness?

– Only distal versus proximal and distal

– Focal/asymmetric versus symmetric

3. What is the nature of the sensory involvement?

– Temperature loss or burning or stabbing pain (e.g., small fiber)

– Vibratory or proprioceptive loss (e.g., large fiber)

4. Is there evidence of upper motor neuron involvement?

– Without sensory loss

– With sensory loss

5. What is the temporal evolution?

– Acute (days to 4 weeks)

– Subacute (4 to 8 weeks)

– Chronic (>8 weeks)

6. Is there evidence for a hereditary neuropathy?

– Family history of neuropathy

– Lack of sensory symptoms despite sensory signs

7. Are there any associated medical conditions?

– Cancer, diabetes mellitus, connective tissue disease or other autoimmune diseases, infection (e.g., HIV, Lyme disease, leprosy)

– Medications

– Preceding events, drugs, toxins

Mononeuropathy

It is caused by trauma, compression or entrapment

All modalities of cutaneous sensibility are lost over the area supplied by affected nerve

The area of light touch sensory loss is greater than the area of pin prick sensory loss in peripheral nerve loss

Hypotonic, atrophic paralysis of muscle groups innervated by affected nerve

Absent/ reduced deep tendon reflex subserved by affected nerve

Thin, dry, scaly skin subserved by affected nerve

Mononeuropathy multiplex (Multifocal mononeuropathy)

Involvement of several widely separated nerves

Sensory and motor disturbances confined to the affected nerves

Polyneuropathy

Simultaneous impairment of many peripheral nerves function resulting in symmetric, usually distal loss of function

Legs are affected before arms

Characteristic features of polyneuropathy

- . Muscle weakness with atrophy (symmetric and distal)
- . Sensory disturbance (Glove-Stocking sensory loss)
- . Autonomic and trophic changes
- . Hypo/areflexia

NB: Early sensory changes and motor weakness at sites distant from dorsal and ventral ganglia respectively (axonal length principle)

Localization of spinal root lesions

Dermatome: Cutaneous area supplied by a single posterior spinal root

Myotome: Muscle groups supplied by a single anterior spinal root

Segmental character of sensory and motor symptoms or signs

Causes: Trauma (Eg. disc prolapse at C6-7, L4-5, L5-S1), Lyme disease, herpes zoster, diabetes, HIV

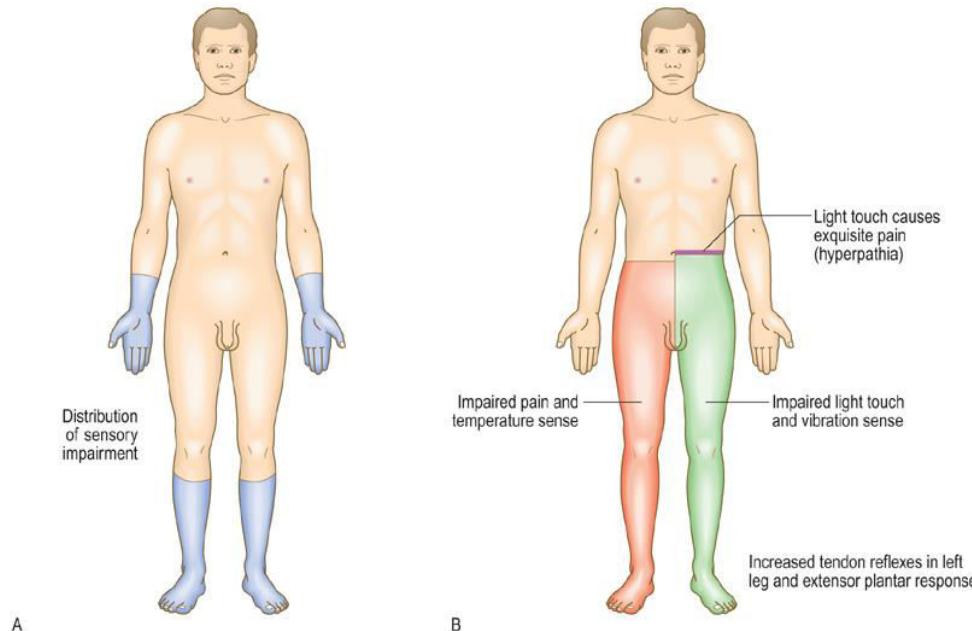
Irritative dorsal root lesion: Radicular pain referred to a specific dermatome

Destructive dorsal root lesion: Hypesthesia or analgesia confined to involved dermatome

Absence of sensory loss does not exclude root lesion / sensory loss evident in multiple root lesions

Weakness, atrophy and fasciculation in myotomal distribution

Hypo/areflexia



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 8.30 A) Polyneuropathy (Glove-stocking type) B) Brown-Sequard syndrome

Localization of lesions affecting the spinal cord

Lesions of the spinal cord

1. Complete spinal cord transaction (Transverse myelopathy)

Sensory and motor dysfunction below the level of the lesion with segmental sign at the level of the lesion

Bowel and bladder dysfunction

Sexual dysfunction (impotence)

Loss of abdominal and cremasteric reflexes

Anhidrosis, trophic changes, impaired temperature control, vasomotor instability below the level of lesion

Extensor and flexor spasms

2. Hemicord syndrome (Brown-Sequard syndrome)

Loss of pain and temperature sensation contra-lateral to the lesion

Ipsilateral loss of proprioceptive function and spastic weakness below the level of the lesion

Segmental LMN and band-like radicular pain at the level of the lesion

3. Central spinal cord syndrome

Dissociative sensory loss: Loss of pain and temperature in bilateral distribution, with preserved vibration and proprioceptive function

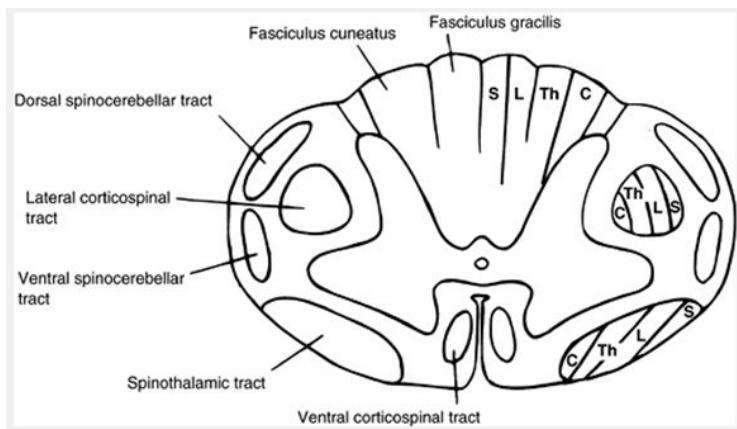
4. Vascular syndromes of spinal cord

a. Anterior spinal artery syndrome

- . Back/ neck pain of sudden onset
- . Rapidly progressive flaccid, areflexic paraplegia
- . Loss of pain and temperature to sensory level
- . Urinary incontinence
- . Preserved vibration/position sensation

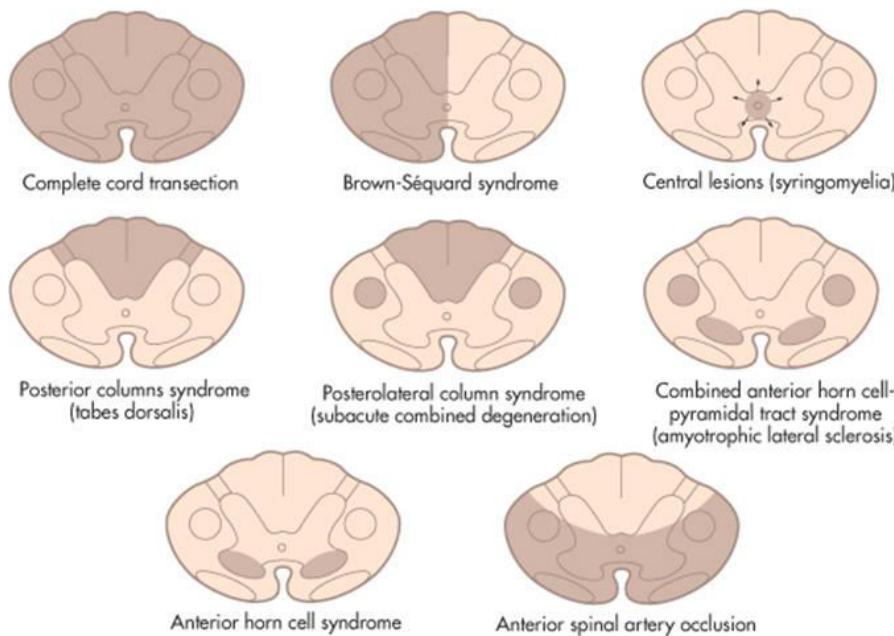
b. Posterior spinal artery syndrome

- . Loss of vibration/position sensation
- . Preserved pain and temperature sensation
- . Loss of myotatic/ cutaneous reflexes below involved segment
- . Absence of motor deficit



© Edwards Brothers. Brazis, Masdeu & Billis. Localization in Clinical Neurology 5e

Fig 8.31 Cross section of the spinal cord (C- cervical, Th-thoracic, L-lumbar, S-sacral)



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 8.32 Spinal cord syndromes

Table 8.8 Clinical features of extramedullary and intramedullary spinal cord lesions

Characteristics	Intramedullary lesion	Extramedullary lesion
Type of pain	Funicular pain	Radicular pain
UMN sign	Yes/late	Yes/early
LMN sign	Prominent/diffuse	Unusual/segmental sign
Sensory loss	Descending	Ascending
Sphincter dysfunction	Early	Late
Trophic changes	Common	Unusual

Localization of lesions affecting the brain stem

Medullary lesions

Medial medullary syndrome: Ipsilateral tongue paralysis/ contra lateral hemiplegia with sparing of face / contra lateral loss of vibration and position sensation

Lateral medullary syndrome: Ipsilateral facial pain and temperature loss/ ipsilateral palatal, pharyngeal and vocal cord paralysis with dysphagia/ ipsilateral horner's syndrome/ ipsilateral cerebellar signs/ contralateral pain and temperature loss excluding face

Pontine lesion

Medial pontine syndrome: Contralateral hemiparesis including face/ ipsilateral ataxia of limbs and gait

Lateral pontine syndrome: Ipsilateral ataxia of limbs/ ipsilateral paralysis of muscle of mastication/ ipsilateral facial sensory loss

Localization of lesion affecting cerebellum

Cerebellar syndromes

Cerebellar hemispheric syndrome: Incoordination of ipsilateral appendicular movements like dysdiadokokinesia, dysmetria, limb ataxia and dysarthria

Cerebellar vermis syndrome: Axial disequilibrium and staggering gait (truncal ataxia/ titubation) / normal arm coordination, no limb ataxia

Localization of lesions affecting the midbrain

Weber syndrome (cerebral peduncle): Contralateral hemiparesis including face with ipsilateral oculomotor nerve palsy

Benedikt's syndrome (tectum and cerebral peduncle): Contralateral hemiparesis including face and intention tremor with ipsilateral oculomotor nerve palsy

Claude's syndrome (tectum and cerebral peduncle): Contralateral hemiataxia with ipsilateral oculomotor nerve palsy

Perinaud's syndrome (superior colliculus): Bilateral vertical gaze palsy and paralysis of convergence

Localization of lesion affecting thalamus and basal ganglia

Thalamic lesion

Amnesia/ apathy and agitation/ attention derangement/ contra lateral sensory loss with dysesthesia

Basal ganglia lesion

Subthalamic nucleus lesion: contralateral hemi-ballismus

Caudate lesion: choreoathetosis

Substantia nigra lesion: rigidity or tremor

Pallidal lesion: bradykinesia

Localization of lesions affecting the cerebral hemisphere

Frontal lobe lesions: Contra lateral hemiparesis, apraxic gait, abulia with perseveration, apathetic, akinetic mutism, inattention, urinary incontinence, Broca's aphasia, positive primitive reflexes

Parietal lobe lesions: astereognosis, agraphesthesia, tactile extinction, impaired two-point discrimination

Occipital lobe lesions: Visual field defect, visual agnosia, visual hallucination, Anton's syndrome (denial of blindness), alexia without agraphia, impaired optokinetic nystagmus

Temporal lobe lesions: Amnesia (impaired storage), Wernicke's aphasia, visual field defect, hallucination (auditory/ complex visual), complex partial seizures

Dominant and non-dominant hemispheric lesions

Dominant hemispheric lesion: Aphasia, apraxia, acalculia, right-left confusion

Non-dominant hemispheric lesion: Anosognosia, spatial disorientation, hemispatial neglect

Acute confusional state and coma

Definition of terminologies

Acute confusional state: State of reduced mental alertness and responsiveness

Coma: state of mental unresponsiveness or unarousal to noxious stimuli

Persistent vegetative state: “Deafferentated state” described as the patient has no detectable awareness but is wakeful and has sleep-wakeful cycle

Locked-in state:” De-efferentated state” described as the patient is wakeful and aware but is unable to communicate verbally and patient communicates with eye blinks and vertical eye movement

Akinetic mutism: awake but immobile and mute

Abulia: mental and physical slowness

Common causes of coma

- a. Damage to reticular activating system (RAS) and its projections
- b. Cerebral hemispheric lesion
- c. Suppression of RAS function by medications, toxins and metabolic disorders

History taking in confusional state and coma

- . Date and mode of onset: is it sudden or gradual? (If onset unknown, when was he seen apparently normal?)
- . Sudden onset of coma is usually due to vascular disease (eg. Stroke, subarachnoid hemorrhage)
- . Rapid progression from hemispheric signs (hemiparesis, hemisensory deficit, or aphasia) to coma within minutes or hours is due to intracerebral hemorrhage
- . Subacute progression leading to coma (days to weeks) is seen with brain tumor, abscess, and subdural hematoma
- . Coma preceded by confusional state or agitated delirium, without localizing signs or symptoms is due to metabolic encephalopathy
- . Ask history of head injury, alcohol abuse, sedative-hypnotic drugs use, exposure to toxins, medical illnesses (hypertension, diabetes, chronic renal failure, and hepatic failure), CNS infections, seizure disorder, etc..., presence of neurologic deficit (hemiparesis, hemisensory deficit, meningeal irritation signs)

Neurologic assessment in acute confusional state and coma

1. Posture

Decorticate posturing occurs in bilateral damage rostral to mid brain

Decerebrate posturing occurs in mid brain and caudal diencephalic motor tract damage

2. Response to noxious stimuli

Abduction or avoidance movement of a limb is usually purposeful, and indicates intact corticospinal system

3. Brain stem ocular reflexes

Oculo-cephalic reflex

Doll's eyes: reflex elevation of eyelids with flexion of the neck

Normally suppressed in awake patient by visual fixation

Presence of oculo-cephalic reflex indicates reduced cortical influence on the brain stem

The eyes move opposite to the side of head rotation in intact brain stem function

Oculo-vestibular reflex: thermal or caloric stimulation of vestibular apparatus

Irrigating the external auditory canal with cold water induces convection currents in the labyrinthines, which causes tonic deviation of both eyes to the side of irrigated ear with correcting nystagmus in the opposite direction (COWS: cold-opposite, warm-same) due to cortical influence

Conjugate deviation of eyes without nystagmus indicates cerebral hemispheric damage

Loss of conjugate ocular movements indicate brain stem damage

4. Pupillary light response

Normal pupil is symmetrically midsized (3-5 mm) and round shape

Unilateral unreactive dilated pupil suggests compression of 3rd cranial nerve by herniation of supratentorial mass

Bilaterally dilated (>7 mm) and unreactive pupils suggests midbrain damage due to supratentorial mass compression

Bilaterally small reactive pupils (1-2.5 mm) is caused by metabolic disorders or thalamic hemorrhage

Very small reactive pupils (<1 mm) occurs in barbiturate poisoning or pontine hemorrhage

5. Ocular movements

Look for resting position of eyes

Conjugate horizontal ocular deviation – opposite to side of pontine lesion and same side of frontal lobe lesion

“The eyes look toward a hemispheric lesion and away from a brainstem lesion”

Down ward and inward turned eyes occur in thalamic and upper midbrain lesions

Ocular bobbing with absent horizontal eye movement occurs in bilateral pontine lesion

Absent reflex eye movement indicates brainstem damage or sedative-hypnotics over dose

6. Respiratory pattern

Cheyne-Stokes respiration- Diffuse hemispheric lesions

Central hyperventilation- Diencephalic lesions

Apneustic respiration- Pontine lesions

Agonal (ataxic) respiration- medullary lesions

Intracranial mass lesions causing acute confusional state or coma

Supratentorial mass causing central transtentorial herniation

Early diencephalic phase: reactive small pupils (<2 mm), intact reflex eye movement, localizes to painful stimuli

Late diencephalic phase: as above with decorticate posturing

Midbrain dysfunction: fixed and midsized pupils, impaired reflex adduction of eyes, decerebrate posturing

Ponto-medullary dysfunction: fixed, midsized pupils, loss of reflex adduction or abduction of eyes, no motor response to painful stimuli

Causes of supratentorial structural lesions

- . Subdural hematoma
- . Putaminal hemorrhage
- . Large cerebral infarction
- . Brain abscess
- . Brain tumor

Causes of infratentorial structural lesions

- . Basilar artery thrombotic stroke
- . Cerebellar hemorrhage
- . Cerebellar abscess or tumor
- . Posterior fossa subdural hematoma

CHAPTER NINE

Musculoskeletal (Locomotor) system

Learning objective

At the end of this lesson, the student should be able to:

1. Mention main symptoms in musculoskeletal problem
2. Apply GALS (Gait/Arm/Leg/Spine) screening tests to detect musculoskeletal disorder
3. Mention common causes of joint disorder
4. perform technique of eliciting stretch test for back pain

History taking in locomotor system

Major symptoms in locomotor system problem include

- . Pain
- . Swelling
- . Morning stiffness
- . Loss of function
- . Deformity
- . Instability
- . Associated systemic symptoms

Joint pain

Describe about location of joint pain (s), mode of onset and course, pattern of spread, effect of exercise and rest, morning stiffness, other symptoms in affected joint, and associated systemic symptoms

Pain

Have you had any pain in joint (s)?

Has the pain involved one joint or its adjacent tissues? Or have several joints been involved?

Involvement of one joint implies a monoarthritis; 2-4 joints, oligoarthritis; and five or more, polyarthritis.

Mode of onset and course

Did the pain develop rapidly over several hours or few days (acute arthritis < 6 weeks) or insidiously over weeks or months (chronic arthritis > 12 weeks)?

Have there been periods of improvement or worsening? How long does the pain last?

Pattern of joint involvement

If more than one joint is involved, determine pattern of spread

Migratory- involving new joint while the initially involved joint improve, eg. arthritis in acute rheumatic fever and gonococcal arthritis

Progressive or additive- progressed to involve new joint while the initially involved joint persisted, eg. rheumatoid arthritis

Assess symmetry of involvement in polyarthritis eg. Symmetrical involvement of joints is observed in rheumatoid arthritis

Effect of rest and exercise

Inflammatory arthritides produce pain at rest and on movement, and often worse in the morning.

Non-inflammatory arthritides tend to become more painful with activity and ease with rest.

Morning stiffness

Stiffness (gelling): Perception of tightness or resistance to movement, and often associated with discomfort (muscle soreness) or pain

Stiffness in inflammatory arthritides often lasts ≥ 30 minutes

Associated symptoms with joint pain

Other symptoms in the involved joint (s) such as swelling, stiffness, limitation of motion, tenderness, warmth or erythema

Problems in tissues around the joint include inflammation of bursae (bursitis), tendons (tendonitis), tendon sheaths (tenosynovitis), or stretching or tearing of ligaments (sprains)

Associated systemic symptoms such as fever, fatigue, anorexia, weight loss, and generalized weakness

High grade fever and chills with monoarthritis suggests infectious cause

Presence of systemic symptoms with chronic polyarthritis suggests connective tissue diseases like rheumatoid arthritis, lupus arthritis, etc...

Other system symptoms giving important clues to the nature of the problem such as butterfly rash over the cheeks (SLE); scaly rash and pitting nails (psoriatic arthritis); dry eyes and mouth (Sjogren's syndrome); penile erosions and scales, and red itchy eyes (Reiter's arthritis)

Deformity

Notice for degree of deformity and functional impairment

Presence of joint deformity with functional impairment indicates advanced disease

Instability

The patient usually say "Giving way" or "Coming out" of the joint, and may be due to true dislocation, or alternatively to muscle weakness or ligamentous problem

Raynaud's phenomenon

Abnormal response of the fingers and toes to cold (white-blue-red response of the fingers after exposure to cold). Patients with Raynaud's disease have Raynaud's phenomenon without an obvious underlying cause (familial, more in females), or occurs in connective tissue diseases (scleroderma) with digital ulcers

Family history

Some diseases with chronic arthritis run in families including rheumatoid arthritis, crystal-induced arthropathy, primary osteoarthritis, seronegative spondyloarthropathy, inflammatory bowel disease, etc...

Musculoskeletal terminologies

Crepitus

A palpable (less commonly audible) vibratory or crackling sensation elicited with joint motion; coarse joint crepitus indicates advanced cartilaginous and degenerative changes (as in osteoarthritis)

Subluxation

Alteration of joint alignment such that articulating surfaces incompletely approximate each other

Dislocation

Abnormal displacement of articulating surfaces such that the surfaces are not in contact

Contracture

Loss of full movement resulting from a fixed resistance caused either by tonic spasm of muscle (reversible) or to fibrosis of periarticular structures (permanent)

Deformity

Abnormal shape or size of a structure; may result from bony hypertrophy, malalignment of articulating structures, or damage to periarticular supportive structures

Enthesitis

Inflammation of the entheses (tendinous or ligamentous insertions on bone)

Examination of the locomotor system

The gait, arms, legs and spine (GALS) screen is a rapid and sensitive screening method for detecting musculoskeletal disorders

Gait

Watch the patient walking and turning back.

Describe the gait. Look for smoothness and symmetry of the gait. Note a limp or use of cane or crutches. The normal gait is divided into the phases of stance (60%) and swing (40%). The stance phase is from foot-strike to toe-off, when the foot is on the ground and load-bearing. The swing phase is from toe-off to foot-strike, when the foot clears the ground. When both feet are on the ground, it is double stance.

Clinically important gaits include

- a. Antalgic gait is a short stance phase on the painful side
- b. Short-leg gait is pelvic obliquity and flexion deformity of the opposite knee
- c. Coxalgic gait is an antalgic gait with a lurch toward the painful hip
- d. Metatarsal gait is avoiding weight bearing on the forefoot

Arms

Ask the patient to put his hands behind his head, with his elbows back'; observe shoulder and elbow function
Have patient bend and straight elbows

Have patient turn palms up (supination) and down (pronation) with arms at sides

Ask patient to extend and spread fingers of both hands

Ask patient to make a fist with thumbs across knuckles

Ask patient to flex, extend, ulnar and radial deviate the wrists

Legs

With the patient in standing position:

Examine the lower limbs for swelling, deformities or limb shortening

With the patient lying on a couch:

Flex each hip and knee with a hand on the knee to feel for crepitus

Flex leg to ninety degree at the hip and knee, and then swing the leg medially for external rotation and laterally for internal rotation (internal rotation restriction is indicative of hip joint disease)

Flex knee upwards and pull firmly against abdomen

Abduct the hips- spread the extended legs apart

Palpate each knee for warmth and swelling, and press on the patella feeling for an effusion

Palpate the anterior surface of ankle joint for swelling and tenderness

Dorsiflex and plantar flex the foot at the ankle

Stabilize the ankle with one hand and invert/evert foot at the subtalar joint

Stabilize heel with one hand and invert/evert the forefoot at transverse tarsal joint

Squeeze gently across the metatarsals for tenderness

Flex toes on metatarsophalangeal joint

Inspect the soles of the feet for calluses and ulcers

Spine

Inspect the standing patient:

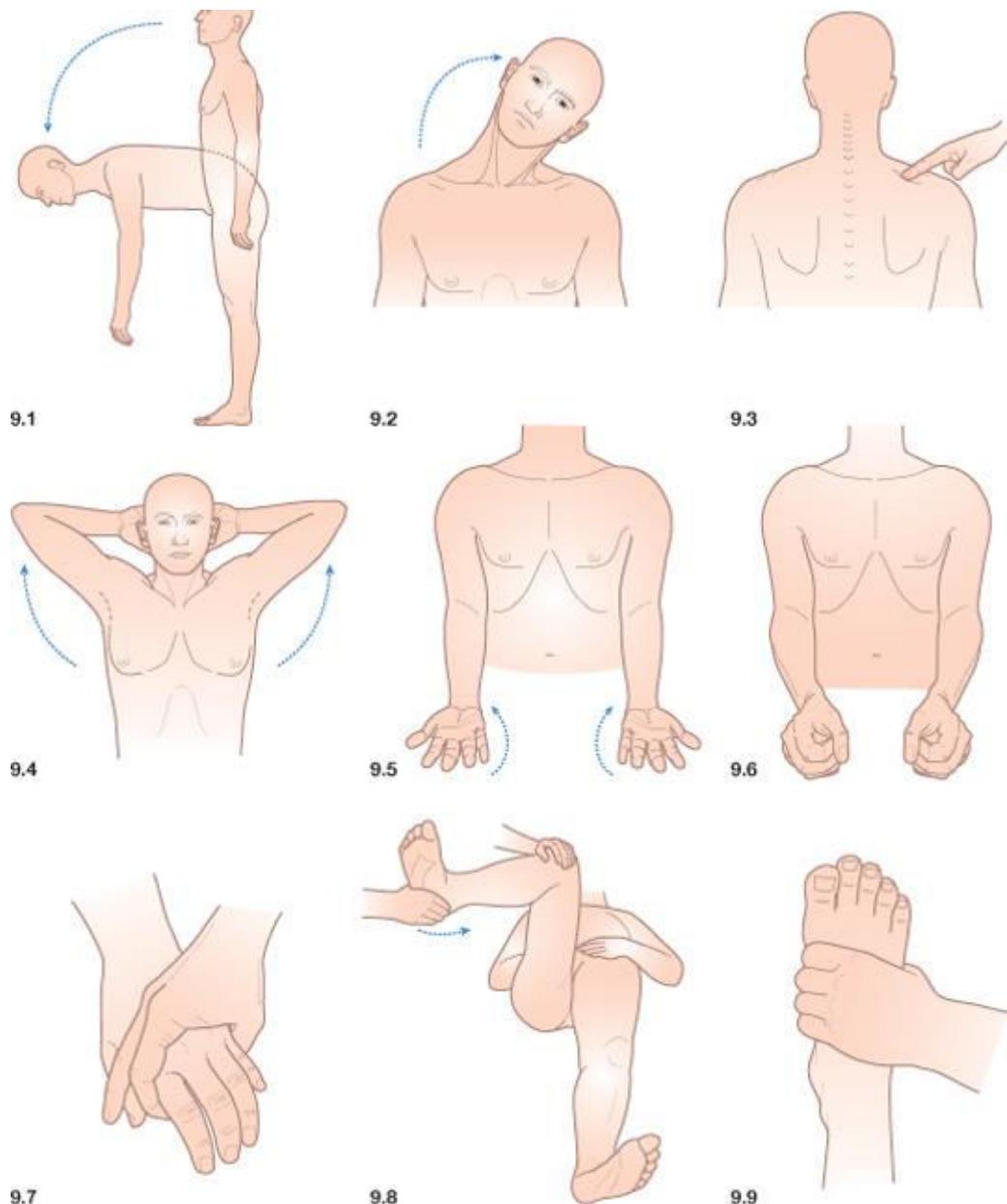
From behind- look for abnormal spinal and paraspinal anatomy and look at the legs

From the side- look for abnormal spinal posture, then ask the patient to bend down and try to touch their toes

From the front- ask the patient to try and put his ear on his left and right shoulder', 'touch chin to chest', 'touch chin to each shoulder', and 'put head back'

Gently press the midpoint of each supraspinatus muscle, spinous process and paravertebral muscles with a thumb to elicit tenderness

In sitting position on a couch: Lateral bending while stabilizing the pelvis, backward bending (extension of spine), and twisting shoulders (rotation of spine)



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 9.1 Screening tests for locomotor system (GALS)

Fig.1: Inspect patient from behind and side, observing for normal spinal curves (cervical and lumbar lordosis, and thoracic spinal kyphosis), and then ask the patient to bend forwards to try and touch his toes

Fig.2: From the front, ask the patient to place his ear on his right and then his left shoulder

Fig.3: Gently press the midpoint of each supraspinatus to elicit tenderness

Fig.4: Ask patient to put his hands behind his head. Observe for pain or restricted movement

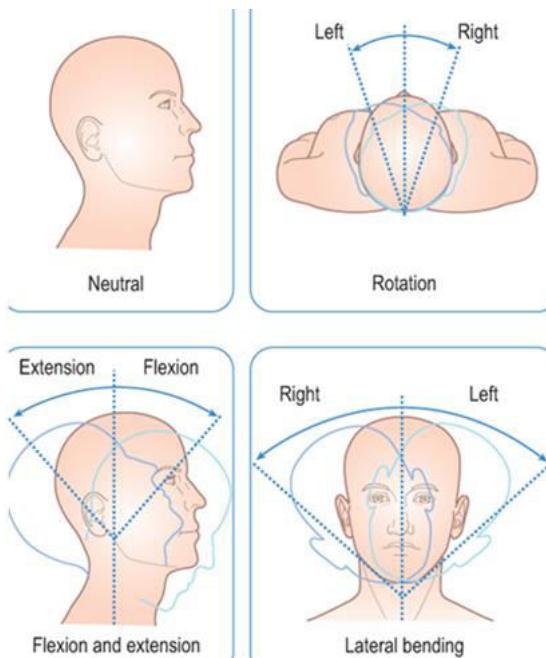
Fig.5: Ask patient to put his hands out palms up and then turn his hands over

Fig.6: Ask patient to make a fist with both hands

Fig.7: Gently squeeze across the metacarpophalangeal (MCP) joints to elicit tenderness

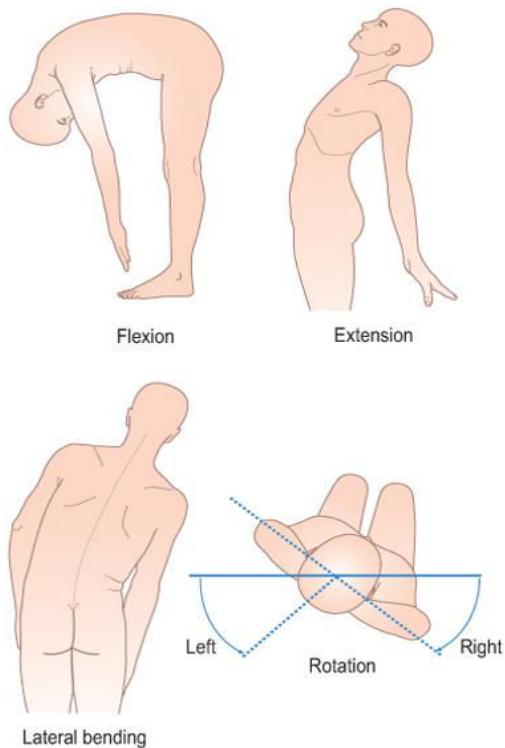
Fig.8: Gently flex the hips and knees, and look for pain and restriction of movement. Look for knee effusion

Fig.9: Squeeze across metatarsophalangeal (MTP) joints and inspect the soles of the feet



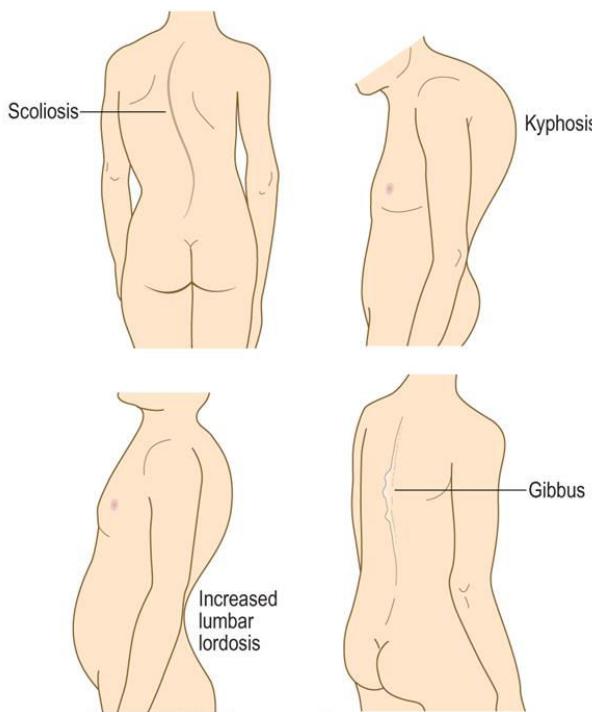
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 9.2 Cervical spine movement: Rotation, flexion and extension, and lateral bending



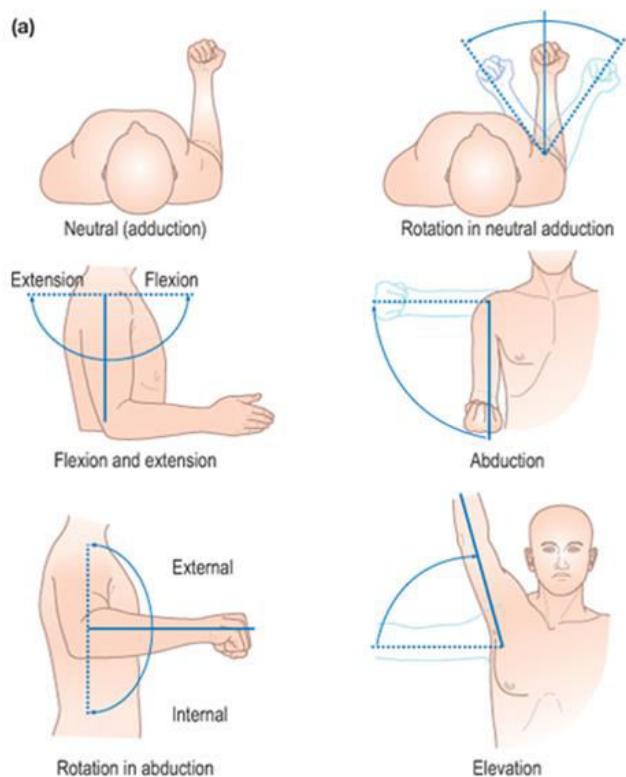
© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.3 Lumbar and dorsal Spine movements: Flexion, extension, lateral bending and rotation



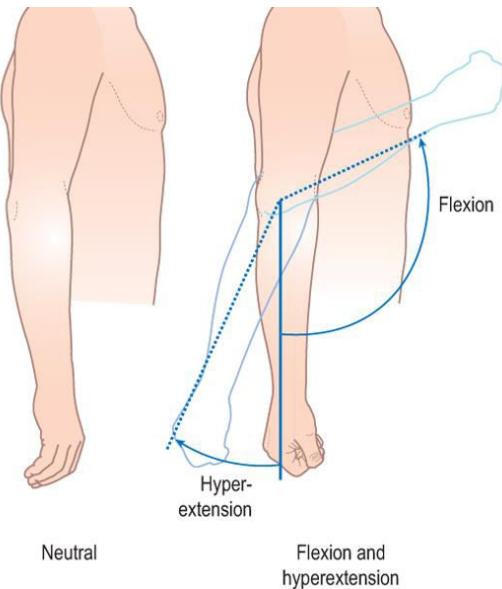
© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.4 Spinal deformities



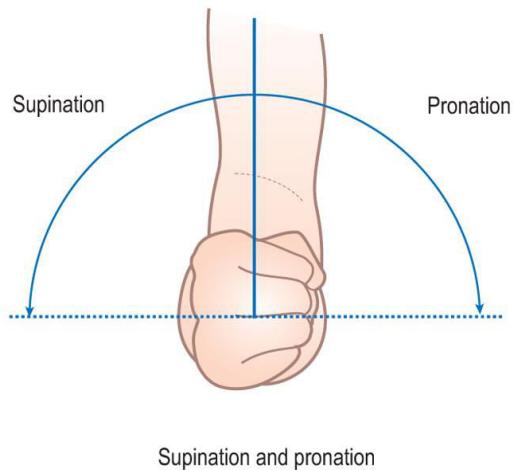
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 9.5 Shoulder movement: Flexion-extension, rotation, adduction-abduction and elevation



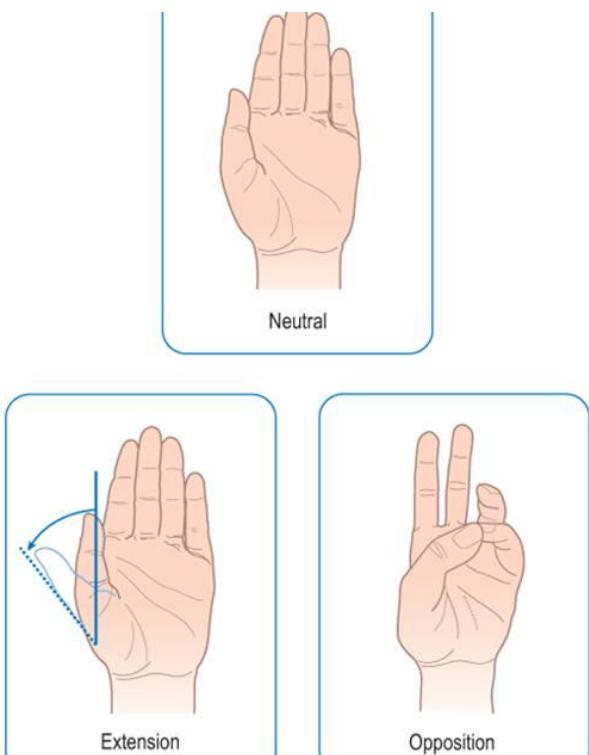
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 9.6 Movement of arm: Arm flexion and hyperextension



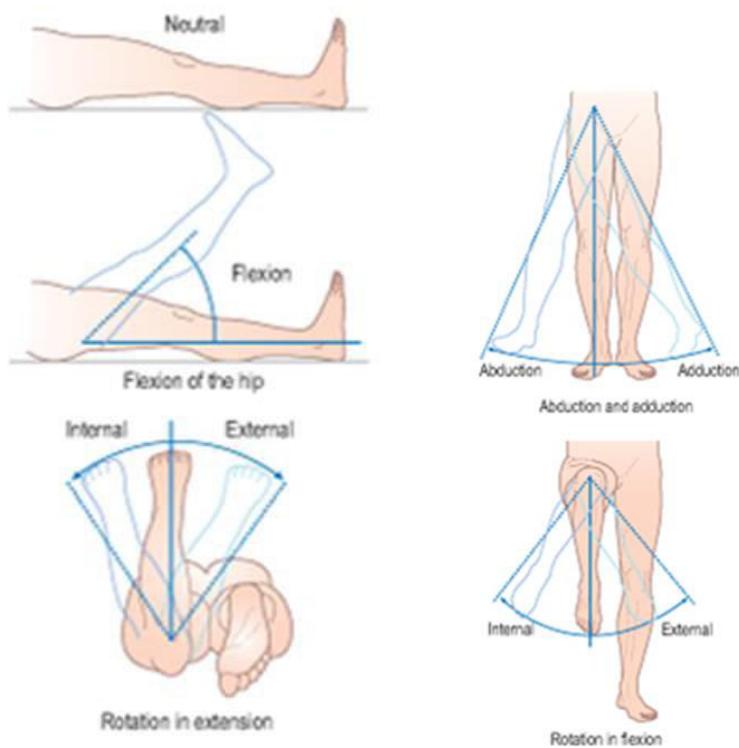
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 9.7 Movement at the wrist: Forearm supination and pronation



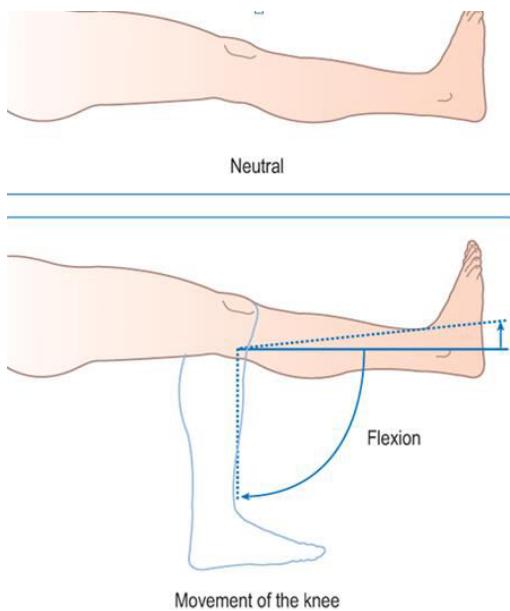
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 9.8 Finger movement: Extension and opposition of thumb



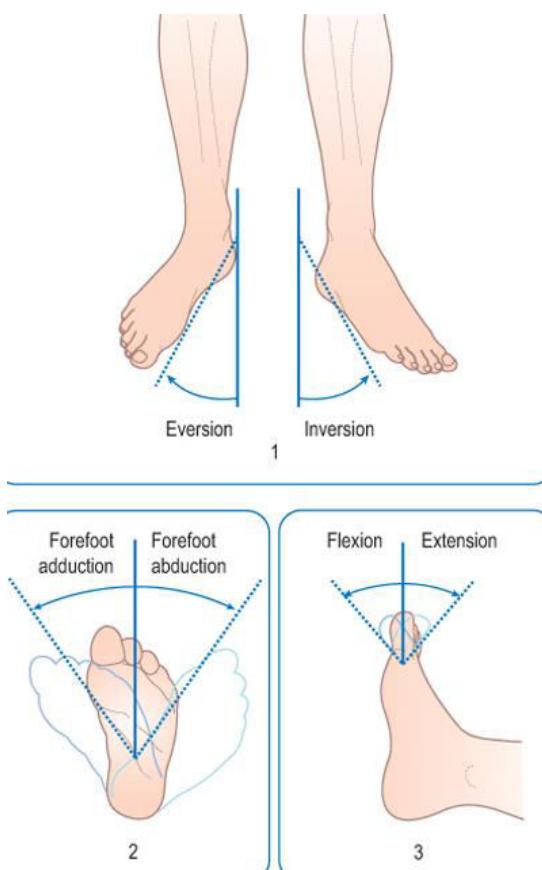
© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 9.9 Movement at the hip joint: Flexion, rotation in extension, abduction -adduction, rotation in flexion



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 9.10 Movement at the knee joint: Flexion and extension



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 9.11 Feet movement: Foot inversion-eversion (1), forefoot adduction-abduction (2), forefoot flexion-extension (3)

Rheumatological (Joint) disease

Combination of joint pain, swelling, stiffness (gelling) and loss of function are the classic features of joint disease

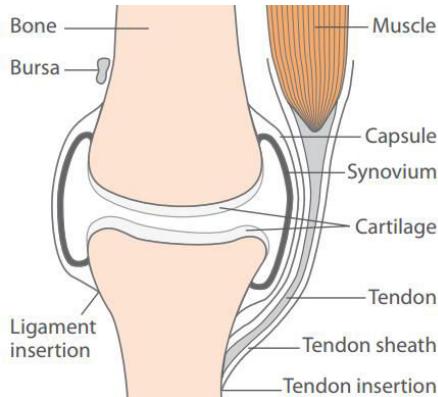
Describe range of movement, distribution of joint involvement, signs of inflammation (warmth, hotness, redness, and swelling), tenderness, crepitus, deformities, and associated clinical conditions

Articular structures include synovium, synovial fluid, articular cartilage, intra-articular ligament, joint capsule, juxtaarticular bone

Non-articular (periarticular) structures include extraarticular ligament, tendon, bursae, muscle, fascia, and overlying skin

Articular disorders are characterized by deep seated or diffuse pain, pain on active or passive movements, swelling (due to synovial proliferation, effusion or bony enlargement), crepitus, instability, locking or deformity

Non-articular disorders tend to have pain on active but not passive movement, range of motion, and point tenderness in regions adjacent to articular structures. Swelling, crepitus, instability or deformity seldom demonstrated.



© Arthritis Research Campaign (Arc). Clinical Assessment of the Musculoskeletal System 2nd e
Fig 9.12 Synovial joint and its periarticular structures

Features of mechanical joint disease

Eg. degenerative joint disease or meniscal tear

- . Brief inactivity stiffness (usually lasting less than 30 minutes), which disappears with activity
- . Pain in the affected joint on activity, usually improving with rest
- . Clicking sensation and locking of a joint. It is due to material within the joint interfering with movement at the articular surfaces
- . Symptoms are confined to affected joint

Features of inflammatory joint disease

Early morning joint stiffness that persists for more than 30 minutes , and precipitated by prolonged rest and improve with activity

Ask about redness (rubor), warmth (calor), tenderness (dolor) and swelling (tumour)

Other systemic symptoms may be present, such as fever, sweating, malaise, weight loss, etc...

Distribution of joint disease

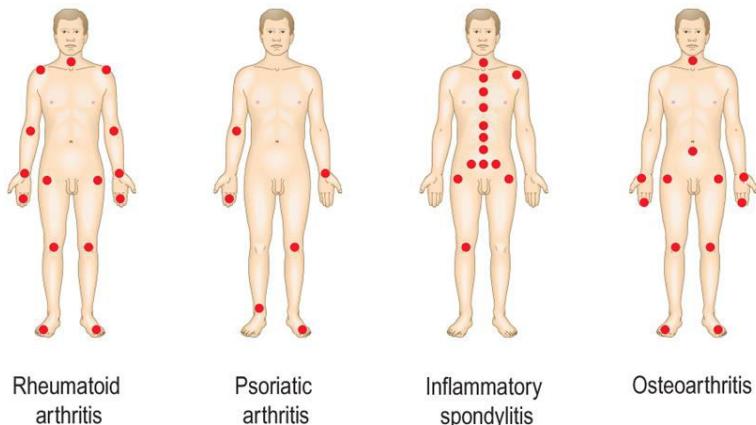
- 1 .Monoarticular (single joint involvement) and oligo- or pauci- articular(2-4 joint involvement) usually occur in septic arthritis, crystal-induced arthritis and seronegative spondyloarthropathies
2. Polyarticular (≥ 5 joint involvement) occurs in immune-mediated arthritis, such as rheumatoid arthritis, SLE and acute rheumatic fever

Causes of monoarthritis

1. Acute monoarthritis: A single hot, red and swollen joint
 - . Septic arthritis: Haematogenous (e.g. Staphylococcal)/Secondary to penetrating injury
 - . Traumatic
 - . Crystal-induced arthropathy (eg. Gout, pseudogout, or hydroxyapatite arthropathy)
 - . Hemarthrosis(e.g. hemophilia)
 - . Seronegative spondyloarthritis (occasionally)
2. Chronic monoarthritis: A single chronic inflamed joint
 - . Chronic infection-e.g. tuberculosis
 - . Seronegative spondyloarthritis

Causes of polyarthritis

- 1 .Acute polyarthritis
 - . Infection-viral, bacterial(gonococcal)
 - . Onset of chronic polyarthritis
- 2 .Chronic polyarthritis
 - . Rheumatoid arthritis
 - . Seronegative spondyloarthritis
 - . Primary osteoarthritis
 - . Crystal-induced arthropathy (occasionally)
 - . Connective tissue disease, e.g. systemic lupus erythematosus
 - . Infection, e.g. Lyme arthritis, spirochaetal infection (rare)



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.13 Patterns of involvement in polyarthritis

(A) Rheumatoid arthritis (symmetrical, small and large joints, upper and lower limbs)

(B) Psoriatic arthritis (asymmetrical, large > small joints, associated periarticular inflammation, giving dactylitis)

(C) Seronegative inflammatory spondylitis (axial involvement, large > small joints, asymmetrical)

(D) Osteoarthritis (symmetrical, small and large joints)

Approach to regional rheumatologic complaints

1. Hand pain

Unilateral hand pain results from trauma, overuse, infections or crystal-induced arthritis

Bilateral hand complaints commonly suggest degenerative (osteoarthritis), systemic or immune inflammatory causes (rheumatoid arthritis)

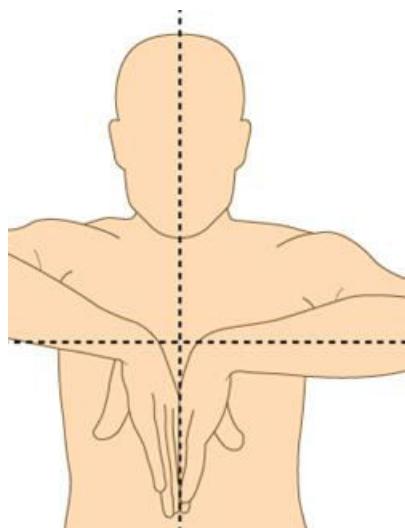
Osteoarthritis causes distal interphalangeal (DIP) and proximal interphalangeal (PIP) joint pain with bony hypertrophy, and pain at base of thumb.

Rheumatoid arthritis (RA) causes DIP, PIP, metacarpophalangeal (MCP), intercarpal and carpometacarpal (CMC) joint pain and deformity.

Psoriatic arthritis causes DIP and PIP joint pain and swelling, nail pitting and onycholysis.

Carpal tunnel syndrome is hand pain along the distribution of median nerve, and occurs in trauma, osteoarthritis and inflammatory arthritis (RA).

Tinel's and Phalen's sign are usually positive in carpal tunnel syndrome: Paresthesia in a median nerve distribution is reproduced by "thumping" the volar aspect of the wrist (Tinel's sign) or pressing the extensor surfaces of both flexed wrists against each other (Phalen's sign)



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.14 Technique of eliciting Phalen's sign (reproducing paresthesia with the above maneuver)

Hand examination

Look

Colour change: Erythema suggests acute inflammation caused by soft tissue infection, septic arthritis or crystal-induced disease (gout and pseudogout)

Swelling: swelling at the MCP or IP joints suggests synovitis

Deformity: Boutonnière (button-hole) deformity is a flexion deformity at the PIP joint with hyperextension at the DIP joint, and 'Swan neck' deformity is hyperextension at the PIP joint with flexion at the DIP joint. Both are hand deformities seen in patients with rheumatoid arthritis.

Dupuytren's contracture affects the palmar fascia, resulting fixed flexion deformity at MCP and PIP joints of the little and ring fingers

Feel

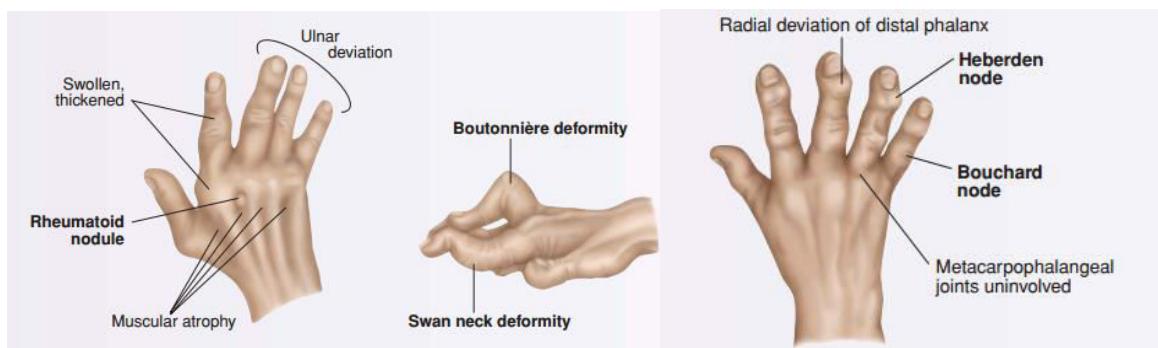
Soft swellings suggest synovitis, while hard swellings suggest bony outgrowths

Heberden's and Bouchard's nodes at DIP and PIP joint outgrowths of fingers suggest osteoarthritis

- . Detect synovitis in the IP joints by gently squeezing with your thumb and index finger above and below the joint to detect sponginess
- . Test the MCP joints by examining for sponginess by squeezing gently across the metacarpal joints
- . Palpate the flexor tendon sheaths in the hand and fingers to detect local swellings or tenderness

Move

Range of motion of hand: Ask the patient to make a fist with each MCP and IP joint flexed to 90°; and ask the patient to squeeze two of your fingers to test grip



a. Rheumatoid arthritis

b. Osteoarthritis

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 9.15 Joint deformities in rheumatoid arthritis (Swan neck and Boutonniere deformities); and bony outgrowths in osteoarthritis (Heberden and Bouchard nodes)

2. Shoulder pain

It results from trauma, fibromyalgia, infections, inflammatory diseases, occupational hazards or cervical disease

It may be referred pain from intrathoracic lesions (pancoast tumor), gall bladder, hepatic, or diaphragmatic diseases

Shoulder pain occurs in subacromial bursitis, bicipital tendonitis, osteoarthritis, rheumatoid arthritis, and rotator cuff tear or tendonitis

Rotator cuff tendonitis is the common cause of shoulder pain. It is characterized by pain on active abduction, night pain, and impingement sign

Shoulder Examination

Look

Examine the whole shoulder girdle from front and back side

Look for deformities in joint dislocation

Look for any swelling, deformity or muscle atrophy

Feel

Palpate the acromioclavicular joint, glenohumeral joint, subacromial space, bicipital groove and the scapula spine

Tenderness in shoulder is due to synovitis, rotator cuff impingement, or bicipital tendonitis

Move

Range of motion of shoulder: Forward flexion (180°), extension (45°), abduction (150°), external rotation (90°), internal rotation (90°), and horizontal adduction (130°)

. Limitation of external rotation is a good sign of true glenohumeral disease as in erosive damage by inflammatory arthritis or adhesive capsulitis (frozen shoulder)

Rotator cuff tendonitis

Ask the patient to abduct the arm from the side of the body. If abduction is painful from 60° to 120° , it suggests rotator cuff tendonitis

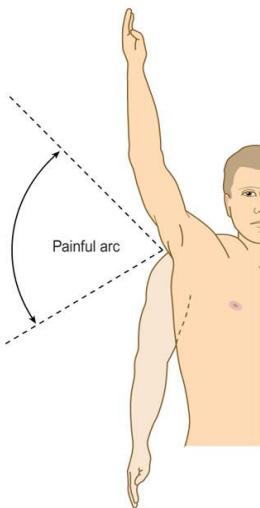
Impingement sign

1. Neer's sign - extreme forward flexion with the forearm pronated

2. Hawkin's sign - 90° forward flexion of the shoulder with the elbow flexed to 90° then internal and external rotation movements of the shoulder

3. Crossover sign - extreme horizontal adduction across the chest

Pain with these maneuvers suggests rotator cuff impingement in the subacromial space



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th ed

Fig 9.16 Painful arc (shoulder pain from 60° to 120° during abduction) indicates rotator cuff tendonitis

3. Knee pain

Knee pain and swelling result from synovial effusions due to inflammatory arthritis (RA, gout, pseudogout or reactive arthritis), hemarthrosis (coagulopathy and torn cruciate ligament), or bony enlargement in degenerative arthritis (osteoarthritis).

Knee locking is block to full extension, which is caused by a loose body or meniscal tear

Knee instability ('giving way') occurs in ruptured or loose ligament from injury or degenerative disease respectively

Knee joint examination

Look

- . Observe the patient walking and standing
- . Look for posture and deformities; Genu varum (bow legs) and genu valgum (knock knee)
- . Look for muscle wasting; measure the thigh girth in both legs 20 cm above the tibial tuberosity
- . Measure leg length discrepancy
- . Notice for enlarged prepatellar bursa (housemaid's knee), effusion of the knee joint, and Baker's cyst (bursa enlargement in the popliteal fossa)

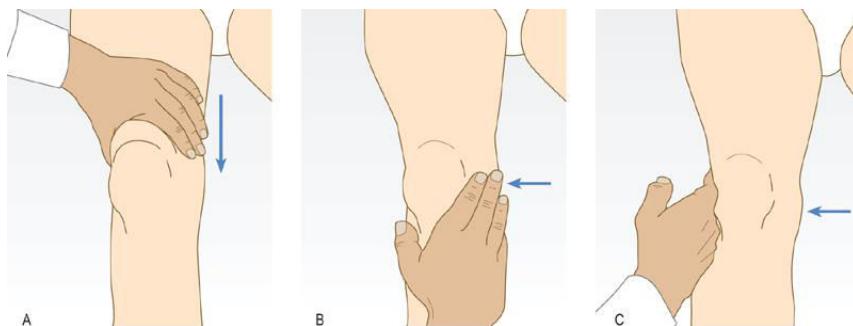
Feel

Knee joint effusions give positive 'bulge sign' and 'balloon sign'

- a. The bulge sign (for minor effusion)

With the knee extended, place the left hand above the knee and apply pressure on the suprapatellar pouch, displacing or "milking" fluid downward. Stroke the medial aspect of the knee and apply pressure to force fluid

into the lateral area. Tap the knee just behind the lateral margin of the patella with the right hand. Bulge on the medial side between the patella and the femur is positive test for knee effusion.



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.17 Testing technique for bulge sign

(A) Empty the suprapatellar pouch **(B)** Stroke the medial side of the joint to displace excess fluid to the lateral side of the joint. **(C)** Stroke the lateral side while watching the medial side closely for a bulge

b. The balloon sign (for large effusion)

With the knee extended, place the thumb and index finger of your right hand on each side of the patella. Compress the suprapatellar pouch against the femur with the left hand. Palpate for fluid ejected or “ballooning” into the spaces next to the patella under your right thumb and index finger. A palpable fluid wave is positive test for knee effusion.



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.18 Testing technique for balloon sign

Move

Range of motion of knee: Ask him to flex the knee up to the chest and then extend the leg back down (normal range 0-140°)

A restriction to full extension occurs with meniscal tears, osteoarthritis or inflammatory arthritis.

Knee meniscal cartilage (medial and lateral) damage

Suspect meniscal cartilage damage when a patient complains of locking, clicking or ‘giving way’ of the joint, usually occurs in trauma or athletic activity

Positive McMurray test indicates meniscal tear

- . Flex the knee at 90°, and then extend the legs while the lower extremity is simultaneously torqued medially or laterally
- . Painful click during inward rotation or outward rotation indicates lateral and medial meniscal tear respectively

Knee collateral ligament damage

- . Hold the ankle between your elbow and side with patient’s knee extended
- . Use both hands to apply a valgus and then varus force to the knee
- . Use your thumbs to feel the joint line and assess the degree to which the joint space opens

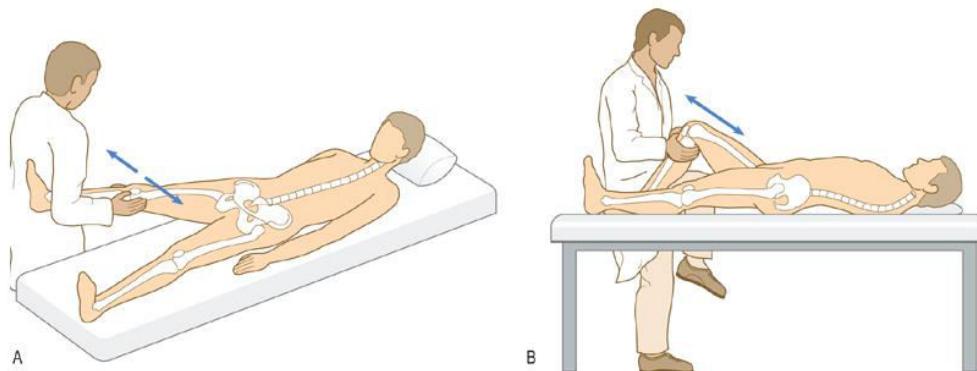
Major opening of the joint indicates collateral and cruciate injury

Knee cruciate ligament damage

It occurs in traumatic injury to knee

Drawer sign: Positive drawar sign indicates cruciate ligament damage

- . While the patient in recumbent position, partially flex the knee and the foot stabilized on the examining surface
- . Manually attempt to displace the tibia anteriorly or posteriorly with respect to the femur
- . Excessive anterior or posterior ‘glide’ or ‘draw’ may indicate respective anterior and posterior cruciate ligament laxity or instability



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.19 Testing A) Collateral and B) Cruciate ligaments of the knee

4. Hip pain

True hip joint pain tends to be located anteriorly over the inguinal ligament and may radiate medially to the groin or along the anteromedial thigh

Anterior hip pain is due to true hip pain, iliopsoas bursitis, enthesitis or meralgia paresthetica

Posterior hip pain is due to sacroiliac pain, buttock pain in sciatica, trochanteric bursitis, ischiogluteal bursitis, or gluteal and trochanteric pain due to fibromyalgia

Hip joint Examination

Look

. Look the two phases of gait

Stance-when the foot is on the ground and bears weight (60% of the walking)

Swing-when the foot moves forward and doesn't bear weight (40% of the walking)

. Observe for pelvic tilt and deformities of hip

Feel

Palpate for tenderness over anterior hip joint, anterior superior or inferior iliac spine, posterior superior iliac spine, sacroiliac joint, ischial tuberosity and gluteus muscles

Move

Range of motion of hip

Internal rotation (30°) - Stabilize knee at 90° flexion with patient seated, move foot away from midline

External rotation (60°) - Stabilize knee at 90° flexion with patient seated, move foot toward midline

Flexion (120°) - with patient supine, grasp bent knee and pull to chest (stop when back flattens)

Extension (15°) - while prone, lift leg off table

Abduction (45°) - with patient supine, hold ankle and pull leg away from midline

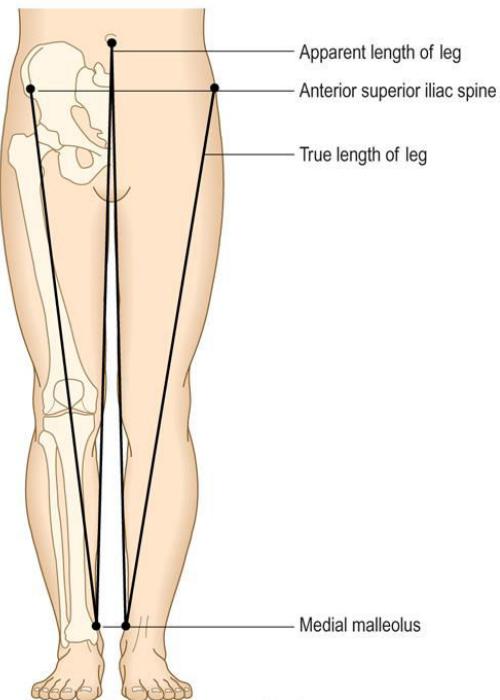
Adduction (30°) - with patient supine, pull leg toward midline (until pelvis tilts)

Special tests for hip joint

1. Measurement of 'true' and 'apparent' shortening of the leg

. Measure the length of the two legs from the anterior superior iliac spine to the medial malleolus. Any difference is termed 'true' shortening'; and results from disease of the hip joint on the shorter side

. Measure the lengths of the two legs from the umbilicus to medial malleolus. 'Apparent' shortening' is due to tilting of the pelvis by abduction deformity of the hip



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.20 Measuring 'apparent' and 'true' length of lower limb

2. Test for flexion deformity (Thomas's test)

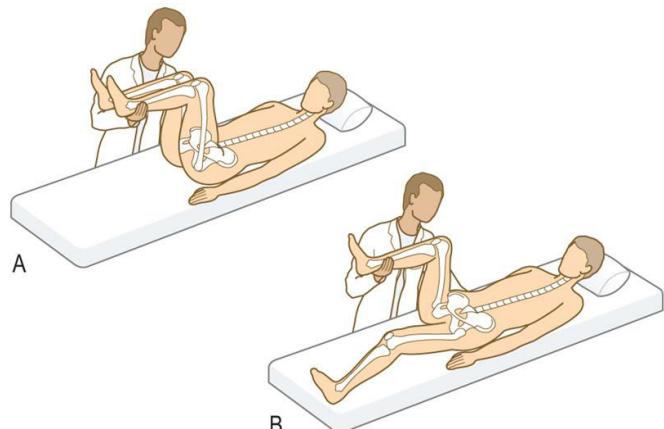
- . Position the patient supine on hard surface
- . Flex both the patient's legs (hips and knees) as far as possible to the point of abolishing the lumbar lordosis
- . Keep the non-test hip maximally flexed, and ask the patient to extend the test hip
- . Incomplete extension of the test hip indicates fixed flexion deformity

3. Trendelenburg's test

- . Observe the patient from behind, and ask the patient to stand on one leg
- . Normally, the pelvis tilts upwards on the side with the leg off the ground
- . When the weight bearing hip is abnormal, the pelvis sags downward due to weakness of the hip abductors on the opposite side

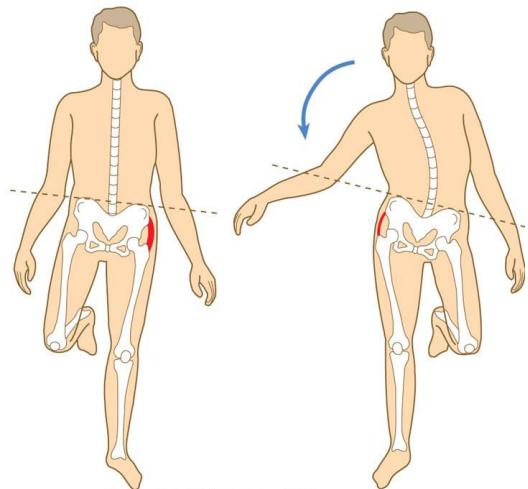
Tests for inflamed hip joint

- . Patrick sign: Hip pain is reproduced by internal or external rotation at the hip with the knee and hip in flexion
- . Hip joint pain is reproduction by tapping the heel of the pt's foot with the examiner's palm while the leg is extended at the hip



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.21 Thomas's test: Testing flexion of hip



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.22 Trendelenburg's test for abnormal hip abductors: Weakness of the right gluteal muscles results in upward pelvic tilt when standing on the right leg

5. Back pain

Types of back pain

- . Local pain- Caused by stretching of pain-sensitive structures that compress or infiltrate sensory nerve endings
- . Pain referred to the back – arises from abdominal or pelvic viscera
- . Pain of spine origin (sclerotomal pain) – located in the back or referred to the buttock or legs
- . Radicular pain – sharp pain radiating from the spine to the legs within the territory of a nerve root
- . Pain associated with muscle spasm- dull spinal pain accompanied by abnormal posture

Common causes of back pain

- . Congenital – spondylosis and spondylolisthesis, tethered spinal cord
- . Fracture
 - . Traumatic- falls, motor vehicle accidents
 - . Non-traumatic- osteoporosis, metastatic neoplasm deposits, prolonged steroid therapy
- . Intervertebral disc prolapse
- . Arthritis- spondyloarthropathies
- . Degenerative – Disk-osteophyte complex, internal disk disruption, spinal stenosis with neurogenic claudication
- . Infection – vertebral osteomyelitis, spinal epidural abscess, septic disk
- . Neoplasm – metastatic solid tumors, multiple myeloma, primary bone tumor
- . Others – referred from visceral disease, postural, psychiatric, chronic pain syndrome

‘Warning features’ of back pain for underlying systemic disease

- . Age < 20 years or >50 years
- . Unexplained fever or weight loss
- . Pain at night or present at rest
- . Back pain lasting > 1 month or not responding to treatment
- . Associated neurologic symptoms or progressive neurologic deficits
- . Presence of immunosuppression or HIV infection
- . History of cancer
- . Long term steroid therapy

Examination of the back

Look

The normal spine has cervical and lumbar lordosis and thoracic kyphosis

Look for hyperkyphosis (lame back) of the thoracic spine or hyperlordosis (sway back) of the lumbar spine

Inspect the lateral curvature of the spine (scoliosis)

Feel

Palpate and percuss the spine: back pain of bony spine origin is often reproduced by palpation or percussion over the spinous process of the affected vertebrae

Move

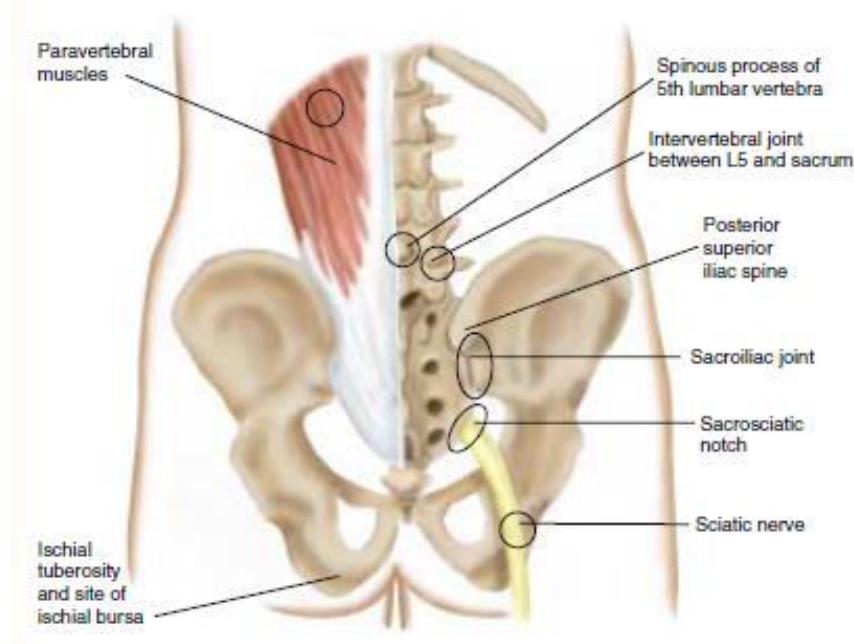
Range of motion of the spine

Forward flexion (normally 80 to 90°) can measure distance of fingertips from floor and is thus more likely to increase disc pain

Extension (20 to 30°) loads the facets and thus is more likely to increase facet pain

Lateral bending (20 to 30° in each direction) stretches muscle and is more likely to aggravate pain from muscle strain

Twisting (30 to 40° in each direction) stretches muscle and increases pain from muscle strain



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 9.23 Anatomic structure of the lower back

Tests for back pain due to disc prolapse

Straight leg-raising (lasegue's) sign

Back pain is reproduced on passive flexion of the extended leg at the hip (straight leg-raising) while the patient lying supine is a feature of prolapsed intervertebral disc, which causes irritation or compression of one of the roots of the sciatic nerve. If in doubt, dorsiflex the foot once the limit of straight leg-raising has been reached, which further stretches the sciatic nerve

Crossed straight leg-raising sign (crossed SLR sign)

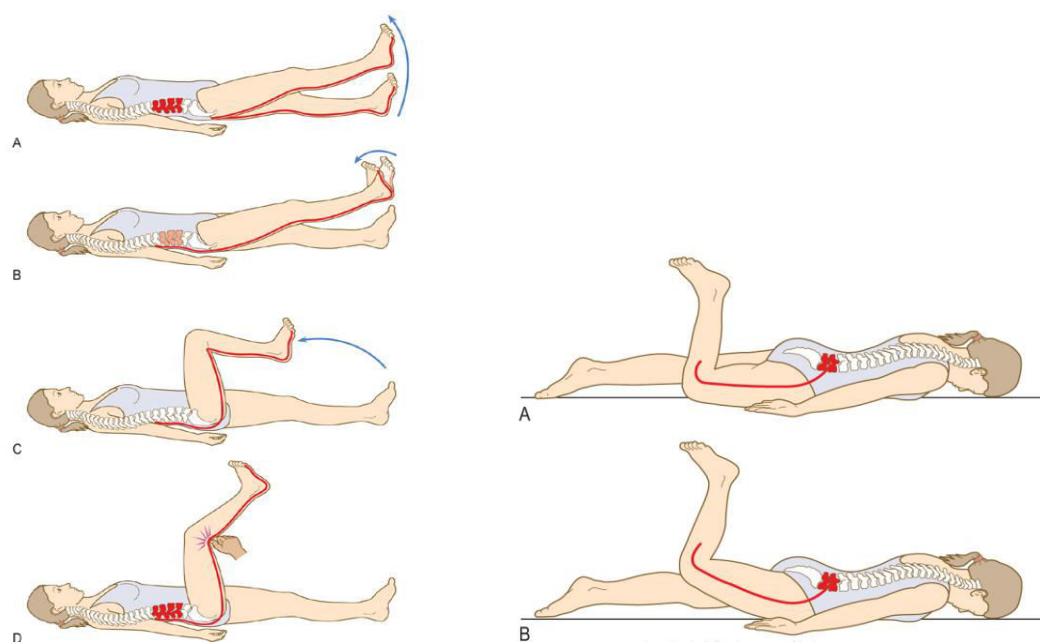
More specific but less sensitive to disc prolapse than straight leg-raising sign

Flexion of one leg at the hip reproduces the pain in the opposite leg or buttock while the patient lying in supine position

The femoral stretch test (reverse SLR test)

It is a useful confirmatory test for prolapsed disc

Back pain is reproduced by flexion at the knee while the patient lying prone



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 9.24 Stretch test

1) Left side (Sciatic nerve)

A) Straight leg raising limited by tension of root over prolapsed disc B) Tension increased by dorsiflexion of foot (Bragard's test) C) Root tension relieved by flexion at the knee D) Pressure over centre of popliteal fossa bears on posterior tibial nerve which is 'bowstringing' across the fossa, causing pain locally and radiation into the back

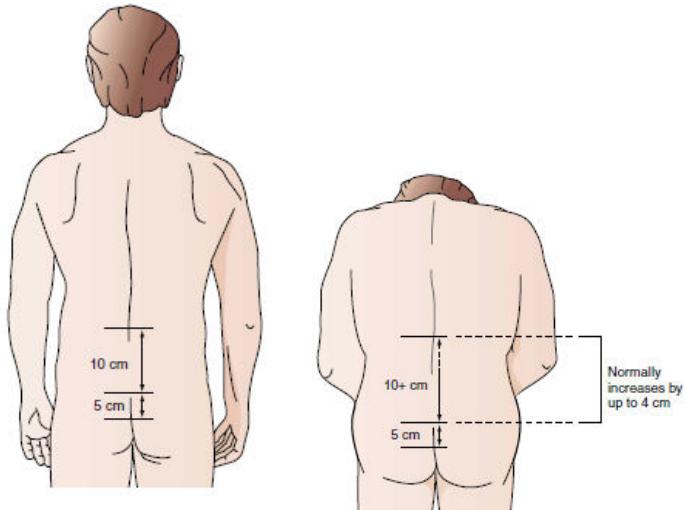
2) Right side (Femoral nerve)

A) Pain may be triggered by knee flexion alone B) Pain may be triggered by knee flexion in combination with hip extension

Special test in back pain

Modified Schober's test

The extent of lumbar flexion can be assessed more accurately by marking a vertical 10 cm line on the skin overlying the lumbar spinous processes and the sacral dimples and measuring the increase in the line length on flexion (normally ≥ 5 cm)



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 9.25 Modified Schober's test: Measuring extent of flexion of the spine

- . Mark the spine at lumbosacral junction, then 10cm above and 5cm below the point.
- . Flattening out of lumbar lordosis and ≥ 5 cm increase between the two upper marks during flexion of the spine is observed in normal individuals. (The distance between the lower two marks should be unchanged)

NB: Persistence of lumbar lordosis with reduced (≤ 4 cm) lumbar flexion length suggests ankylosing spondylitis

Patterns	Possible Causes	Possible Physical Signs
Mechanical Low Back Pain Acute, often recurrent, or possibly chronic aching pain in the lumbosacral area, possibly radiating into the posterior thighs but not below the knees. The pain is often precipitated or aggravated by moving, lifting, or twisting motions and is relieved by rest. Spinal movements are typically limited by pain. This is the back pain common from the teenage years through the 40s.	The exact cause cannot usually be proven. Intervertebral disc disease is probably involved in many cases. Congenital disorders of the spine, such as spondylolisthesis, may be present in a small percentage. In older women or in persons on long-term corticosteroid therapy, consider osteoporosis complicated by a collapsed vertebra.	Local tenderness, muscle spasm, pain on movement of the back, and loss of the normal lumbar lordosis, but no motor or sensory loss or reflex abnormalities. In osteoporosis there may be a thoracic kyphosis, percussion tenderness over a spinous process, or fractures elsewhere such as in the thoracic spine or in a hip.
Radicular Low Back Pain A radicular (nerve root) pain, usually superimposed on low back pain. The sciatic pain is shooting and radiates down one or both legs, usually to below the knee(s) in a dermatomal distribution, often with associated numbness and tingling and possibly local weakness. The pain is usually worsened by spinal movement such as bending and by sneezing, coughing, or straining.	A herniated intervertebral disc with compression or traction of nerve root(s) is the most common cause in persons under age 50. The nerve roots of L5 or S1 are most often affected. Spinal cord tumors or abscesses are much less common causes. Compared to a disc, they tend to affect more nerve roots and to produce more neurologic deficits.	Pain on straight leg raising (see pp. 520), tenderness of the sciatic nerve, loss of sensation in a dermatomal distribution, local muscular weakness and atrophy, and decreased to absent reflex(es), especially affecting the ankle jerks. Dermatomal signs and reflex changes may be absent when only a single root is affected.
Back and Leg Pain From Lumbar Stenosis Pseudoclaudication is a pain in the back or legs that worsens with walking and improves with flexing of the spine, as by sitting or bending forward.	Lumbar stenosis, which is a combination of degenerative disc disease and osteoarthritis that narrows the spinal canal and impinges on the spinal nerves. It is a common cause of pain after age 60.	The posture may become flexed forward. Motor weakness and hyporeflexia in the lower extremities may be present.
Chronic Persistent Low Back Stiffness	Ankylosing spondylitis, a chronic inflammatory polyarthritis, most common in young men Diffuse idiopathic skeletal hyperostosis (DISH), which affects middle-aged and older men	Loss of the normal lumbar lordosis, muscle spasm, and limitation of anterior and lateral flexion Flexion and immobility of the spine

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 9.1 Causes of lower back pain

CHAPTER TEN

Genitourinary system

The urinary tract

Learning objective

At the end of this lesson, the student should be able to:

1. Mention main symptoms in urinary tract problem
2. Show techniques of examining the kidneys
3. List and interpret major urinary syndromes
4. List causes of oliguria

History taking

Major symptoms in urinary tract problem include

- . Flank pain
- . Oliguria or anuria
- . Hematuria
- . Polyuria
- . Urinary incontinence
- . Lower urinary tract symptoms: Dysuria (pain during urination), frequency of micturition, urgency, hesitancy, dribbling, reduced caliber and force of urinary stream, and straining to void

Flank pain

- . Flank pain is visceral pain typically dull, aching, and steady pain due to sudden distension of renal capsule in acute pyelonephritis or acute glomerulonephritis
- . Renal colic is colicky pain in the loin that radiate to the medial thigh or groin, which results from sudden distension of ureter or renal pelvis by renal stone, sloughed papillae or blood clots

Oliguria or anuria

Oliguria is urine volume < 400ml per day

Anuria is urine volume < 100ml per day

Causes of oliguria

Intrinsic renal causes

- . Acute tubular necrosis (ATN) secondary to ischemic or nephrotoxic injury to the kidney
 - Ischemic: Hypovolemia due to GI loss (severe diarrhea or intractable vomiting), blood loss (variceal hemorrhage)
 - Nephrotoxic: Aminoglycosides, radiocontrasts, rhabdomyolysis
- . Acute glomerulonephritis/ vasculitis: Post-infectious glomerulonephritis, anti-neutrophil cytoplasmic antibody (ANCA)-positive glomerulonephritis
- . Acute renal vascular obstruction: Hemolytic uremic syndrome (HUS) /thrombotic thrombocytopenic purpura (TTP), accelerated hypertension, toxemia of pregnancy
- . Acute interstitial nephritis: Allergens (antibiotics, Non-steroidal anti-inflammatory drugs), infections (viral, bacterial, fungal)
- . Intratubular deposition and obstruction: Multiple myeloma, uric acid nephropathy

Pre-renal causes

- . Hypovolemia due to hemorrhage, burns, dehydration, gastrointestinal fluid loss (persistent vomiting or diarrhea)
- . Low cardiac output- congestive heart failure
- . Systemic vasodilatation- sepsis, anaphylaxis, hepatic cirrhosis

Post-renal causes

- . Bilateral ureteric obstruction- calculi, blood clot, sloughed papillae, cancer
- . Bladder neck obstruction- Benign prostatic hyperplasia (BPH), prostatic cancer, neurogenic bladder
- . Urethral obstruction- stricture, phimosis

Hematuria

Hematuria is reddish discoloration of urine due to blood in the urine

It could be gross or microscopic hematuria

Hematuria is defined as presence of $\geq 2-5$ RBCs per high power field (hpf)

Significant hematuria is defined as >3 RBCs/hpf on 3 urinalyses, >100 RBCs/hpf on single urinalysis or gross hematuria

Types of hematuria

- . Initial hematuria- hematuria at the beginning of urination indicates bleeding from urethra
- . Terminal hematuria- hematuria at the end of urination indicates bleeding from the urinary bladder

- . Hematuria throughout urination indicates bleeding from the kidney

Common causes of isolated hematuria

- . Calculi
- . Neoplasms
- . Trauma
- . Renal tuberculosis
- . Prostatitis

The most common causes of isolated glomerular hematuria (hematuria with dysmorphic RBCs, RBC casts and proteinuria >500mg/day)

- . IgA nephropathy
- . Hereditary nephropathy
- . Thin basement membrane disease

Polyuria

Polyuria is urine volume >3 liters in 24-hours

It must be differentiated from urinary frequency, abnormally frequent voiding. Urinary frequency is often associated with relatively small volumes at each passage while polyuria is associated with high volumes of urine with each voiding

Causes of polyuria

- . Neurogenic polydipsia (hypothalamic-pituitary abnormality)
- . Nephrogenic polydipsia (antidiuretic hormone (ADH)-insensitive kidney)
- . Diabetes mellitus
- . Primary polydipsia (psychogenic- excessive water intake)
- . Solute diuresis (diuretics, mannitol infusion, saline infusion)
- . Electrolyte abnormalities- hypercalcemic or hypokalemic nephropathy

Urinary incontinence

Urinary incontinence refers to an involuntary loss of urine

Types of urinary incontinence

- . Urge incontinence- spastic bladder due to strong detrusor muscle contraction

It occurs in diminished cortical control (stroke, Alzheimer's disease), hyperexcitability of sensory pathways (cystitis), or deconditioning of voiding reflex (frequent voiding at low bladder volume)

- . Stress incontinence-weakened urethral sphincter resulting reduced urethral resistance

It results from weakened pelvic floor from repeated child birth, surgery or urethral infection

- . Overflow incontinence- flaccid bladder

It results from weakness of detrusor muscle due to peripheral neuropathy (pudendal nerve damage), or impaired bladder sensation (diabetic neuropathy)

- . Functional incontinence- functional inability to go to the toilet in time due to impaired health or inconvenient environmental conditions

Symptoms of lower urinary tract abnormalities include the following:

- . Frequency of micturition-passing urine more often than usual
- . Urgency-unusually intense and immediate desire to void
- . Hesitancy-difficulty or delay in initiating urine flow
- . Straining to void
- . Reduced caliber and force of the urinary stream
- . Dribbling after micturition
- . Dysuria- pain (burning sensation) on urination, indicates inflammation of the bladder or urethra

Females perceive dysuria as internal urethral discomfort in cystitis and urethritis, or as external burning sensation in vulvovaginitis

Hesitancy, straining, reduced force and caliber of urinary stream and dribbling after micturition occur in bladder outlet obstruction (BOO) due to benign prostatic hyperplasia (BPH) or urethral stricture

If straining increases force and caliber of urinary stream, it favors urethral stricture, but not BPH

Lower urinary tract infection (cystitis or urethritis) frequently presents with suprapubic pain, dysuria, frequency of urination, urgency and hesitancy

Upper urinary tract infection (pyelonephritis) frequently presents with fever, chills, rigors, vomiting, flank pain, hematuria, with or without local urinary tract irritative symptoms

Examinations of the kidneys and urinary bladder- refer to abdominal examination

- . Notice for costo-vertebral angle tenderness (CVA tenderness)

Technique:

- . Locate the costovertebral angle at the back
- . Place the ball of left hand in the costovertebral angle and strike it with the ulnar border of the fisted right hand

Pain with fist percussion at the costovertebral angle suggests pyelonephritis



Fig 10.1 Eliciting costo-vertebral angle (CVA) tenderness to left kidney

© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Major urinary syndromes

1. Acute kidney injury (AKI)

Clues to diagnosis- oliguria, recent decline in glomerular filtration rate (recently raised serum creatinine)

Common findings- hypertension, hematuria, proteinuria, casts, edema

2. Acute nephritic syndrome

Clues to diagnosis- hematuria, red cell cast, proteinuria, azotemia (\uparrow serum Cr)

Common findings- oliguria, hypertension, and edema

3. Chronic kidney disease (CKD)

Clues to diagnosis- azotemia (\uparrow serum Cr) $>$ 3 months, bilateral shrunken kidney, broad casts in urinary sediment

Common findings- anemia, nocturia, hypertension

4. Nephrotic syndrome

Clues to diagnosis- nephrotic-range proteinuria $>$ 3.5gm/1.73m²/day, lipiduria, hypoalbuminemia, hyperlipidemia

Common findings- edema

5. Asymptomatic urinary abnormalities

Clues to diagnosis- hematuria, sub-nephrotic range proteinuria

6. Urinary tract infection

Clues to diagnosis- significant bacteriuria $\geq 10^5$ colonies/ml, pyuria, leukocyte casts

Common findings- fever, flank pain, flank tenderness, +/- lower urinary tract irritative symptoms (suprapubic pain, frequency of urination, urgency, etc...)

7. Renal tubule defects

Clues to diagnosis- polyuria, nocturia, renal calcification, large kidneys, renal transport defects

Common findings- hematuria, ‘tubular’ proteinuria (<1gm/day), enuresis

8. Hypertension

Clues to diagnosis- Blood pressure $\geq 140/90$ mmhg

Common findings- proteinuria, casts, azotemia

9. Nephrolithiasis

Clues to diagnosis- renal colic, history of stone passage, history of stone seen by KUB X-ray

Common findings- hematuria, pyuria

10. Urinary tract obstruction

Clues to diagnosis- oliguria, retention of urine, azotemia, large prostate, large kidneys

Common findings- hematuria, pyuria, enuresis

Female Genitalia

Learning objective

At the end of this lesson, the student should be able to:

1. Mention symptoms in gynecologic problem
2. Describe vaginal discharge and genital ulcer syndromes
3. Perform pelvic examination of gynecologic patient
4. Describe types of abnormal menstrual bleeding

Gynecologic history taking and clinical examination

Gynecologic history taking

Female patients with gynecologic problem present with one or more of the following symptoms

- . Vaginal bleeding (abnormal uterine bleeding)
- . Menstrual irregularities
- . Amenorrhea (1⁰, 2⁰)
- . Vaginal discharge
- . Genital ulcer with/without inguinal swelling
- . Lower abdominal or pelvic pain
- . Pre-menstrual symptoms
- . Pre-menopausal symptoms
- . Dyspareunia
- . Premature menopause

Menstrual history

Age at menarche (age of starting menses), how often do the menstrual periods come? how regular or irregular are menstrual periods? how heavy is the menstrual flow? (number of pads or tampons used daily)

What color is the menstrual flow? Normally, dark red menstrual flow; bright red with clots indicate excessive menstrual flow

Pelvic discomfort or pain before or during menstrual periods (express type of pain, how long does it last)

History of abnormal menstrual bleeding

History of abnormal menstrual bleeding may have organic causes or may be dysfunctional uterine bleeding (DUB)

Menorrhagia- Excessive menstrual flow

Metrorrhagia- Irregular menstrual flow (intermenstrual bleeding)

Metromenorrhagia- Irregular, prolonged and excessive menstrual flow

Oligomenorrhea- Infrequent menstrual periods, occurring at intervals longer than 35 days (frequently occurs during the 1st 2yrs after menarche or before menopause in normal women)

Polymenorrhea-Abnormally frequent menstrual periods

Dysmenorrhea- Crampy or aching lower abdominal or pelvic pain prior to or during menstrual period

Post-coital bleeding- indicates cervical disease such as cervical polyp or cancer

Post-menopausal bleeding- vaginal bleeding more than 1 year after the final menstrual period. Investigate to exclude cancer of genital tract.

History of premenstrual syndrome

Premenstrual syndrome is characterized by the presence of nervousness, irritability, depression and mood swings, abdominal bloating, headache, and noticed tenderness and edema of the breast. These symptoms usually noted during the 4-10 days before menstrual periods

Amenorrhea: Absence of menstrual periods

Primary amenorrhea- failure to initiate menstrual periods by age of 16 years

Secondary amenorrhea- cessation of periods for more than 3 months after menstrual periods have been established

Physiologic causes of secondary amenorrhea includes pregnancy, lactation and menopause

Vaginal discharge with/ without lower abdominal or pelvic pain

. Notice for color, amount, odor and consistency of vaginal discharge

Mucopurulent cervicitis and vaginitis present with abnormal vaginal discharge

All cases of cervicitis are sexually transmitted infections but most cases of vaginitis are reproductive tract infections

Genital ulcer with/without inguinal swelling

Characterize the genital ulcer: Site, single or multiple, painful/tender or not, clean based or dirty ragged ulcer, associated inguinal swelling (painful/tender or not, unilateral or bilateral inguinal lymph node involvement)

Dyspareunia

Discomfort or pain during sexual intercourse

Superficial pelvic pain (vaginismus) may be due to inadequate lubrication, vulvovaginitis or atrophic vaginitis

Deep pelvic pain suggests pelvic diseases like cervicitis, endometritis or adnexitis

Menopause

Cessation of menstrual periods, usually occurs at age of 45-52 yrs

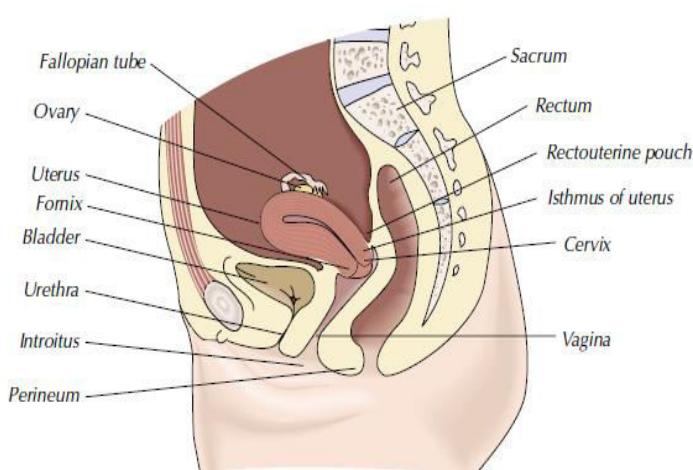
Age at menopause and menopausal symptoms (hot flushes)

Post menopausal bleeding- bleeding that occurs after 6 months of established menopause, usually raises the question of endometrial cancer



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 10.2 Female external genitalia



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 10.3 Female pelvic organs (coronal section)

TIPS FOR THE SUCCESSFUL PELVIC EXAMINATION

<i>The Patient</i>	<i>The Examiner</i>
<ul style="list-style-type: none"> ■ Avoids intercourse, douching, or use of vaginal suppositories for 24 to 48 hours before examination ■ Empties bladder before examination ■ Lies supine, with head and shoulders slightly elevated, arms at sides or folded across chest to reduce tightening of abdominal muscles 	<ul style="list-style-type: none"> ■ Explains each step of the examination in advance ■ Drapes patient from mid-abdomen to knees; depresses drape between knees to provide eye contact with patient ■ Avoids unexpected or sudden movements ■ Warms speculum with tap water ■ Monitors comfort of the examination by watching the patient's face ■ Uses excellent but gentle technique, especially when inserting the speculum (see p. ____).

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Table 10.1 Tips for the successful pelvic examination

Clinical Examination

Pelvic Examination

- . Explain what you are going to do so that she can better understand your gynecologic procedure
- . Explain in advance each step of the examination
- . Ask the patient to empty her bladder before the examination
- . Position the patient and drape her appropriately
- . Wear gloves throughout the examination

Indications for pelvic examination

- . Menstrual abnormalities
- . Unexplained lower abdominal pain
- . Vaginal discharge
- . Contraceptive prescription
- . Bacteriological and cytological studies
- . Rape cases
- . Patients' desire for assessment

Patient positioning

- . The patient in lithotomy position with her arms at her sides or folded across her chest, and assist her to place her legs on the stirrups of examining couch
- . The drape should cover her thighs and knees
- . Ask her to move toward the end of examining couch
- . Her thighs should be flexed, abducted, and externally rotated at the hips

Inspection/Palpation

- . Note for distribution of pubic hair (assessment of sexual maturity in adolescents)
- . Inspect the external genitalia (labia majora and minora, clitoris, urethral meatus, vaginal opening, introitus, Bartholin's gland and Skene's gland) for inflammation, ulceration, discharge, swelling or nodules/warts

Bartholin's cyst

- . Look for Bartholin's cyst in labial swelling
- . Palpate between your index finger (inside) and thumb (outside) for swelling due to Bartholin's cyst, and notice for discharge and tenderness near the posterior end of the introitus

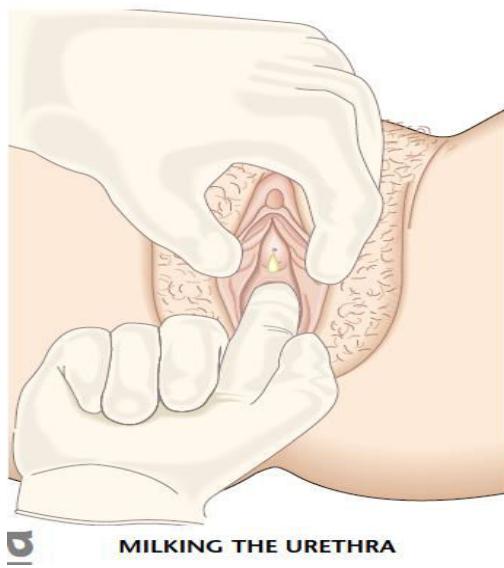


© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 10.4 Technique of palpating the Bartholin's cyst

Paraurethral (Skene's) cyst

- . Insert your index finger into the vagina and milk the urethra from inside to outward, and note for discharge



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 10.5 Milking the urethra (Skene's cyst)

Vaginal wall and Cervix

- . Locate the cervix

Lubricate your index finger with water and insert into the vagina and identify the firm, rounded surface of cervix

It helps to choose the size of speculum and to accurately angle the speculum

- . Assess vaginal walls

Separate the labia with your middle and index finger and ask her to strain down and note for any bulging of vaginal walls (cystocele or rectocele)

- . Insert the speculum

Select speculum of appropriate size and shape, and lubricate with warm water

Hold the speculum with your left hand, and insert and slide the speculum inward along the posterior wall of the vagina

Rotate the speculum in horizontal position, and insert it to its full length

- . Inspect the cervix

Open the speculum and adjust until it cups the cervix

Maintain speculum position by tightening the thumb screw

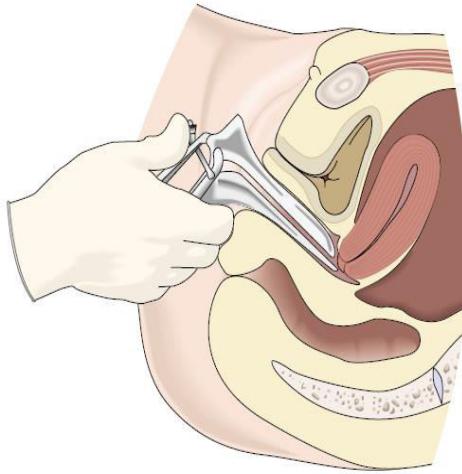
Note the cervical color, position, ulceration, nodules, masses, bleeding or discharge

Obtain specimens for cervical cytology (papanicolaou smear) by endocervical swab, cervical scrape or cervical brush

Release the thumb screw and withdraw the speculum slowly while observing the vagina

Inspect the vaginal mucosa for any inflammation, discharge, ulceration, or masses during speculum withdrawal

Close the speculum as it emerges from the introitus



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 10.6 Technique of inserting the speculum

Bimanual examination

. Perform bimanual examination to appreciate any abnormalities of the cervix, uterus and adnexa (ovary and adjacent structures)

Lubricate and insert index and middle fingers into the vagina with your ring and little finger flexed into your palm and press inward on the perineum with your flexed fingers

Palpate the cervix and note for any nodularity, shape, consistency, regularity, mobility and tenderness

Feel for fornices around the cervix

NB: Presence of cervical motion (excitation) tenderness suggests cervicitis

. Palpate the uterus

Place the left hand on the lower abdomen midway between umbilicus and symphysis pubis

Elevate the cervix and uterus with your pelvic hand, and place and press your abdominal hand in and down to grasp the uterus between your hands

Note for uterine size, shape, consistency, mobility, masses and tenderness

. Palpate the ovary

Place your abdominal hand on the right lower quadrant or left lower quadrant of the abdomen, and your pelvic hand in the lateral fornices

Press your abdominal hand in and down to push the adnexal structures toward your pelvic hand

Identify the ovaries and adjacent adnexal masses

Note for ovary size, shape, consistency, mobility and tenderness

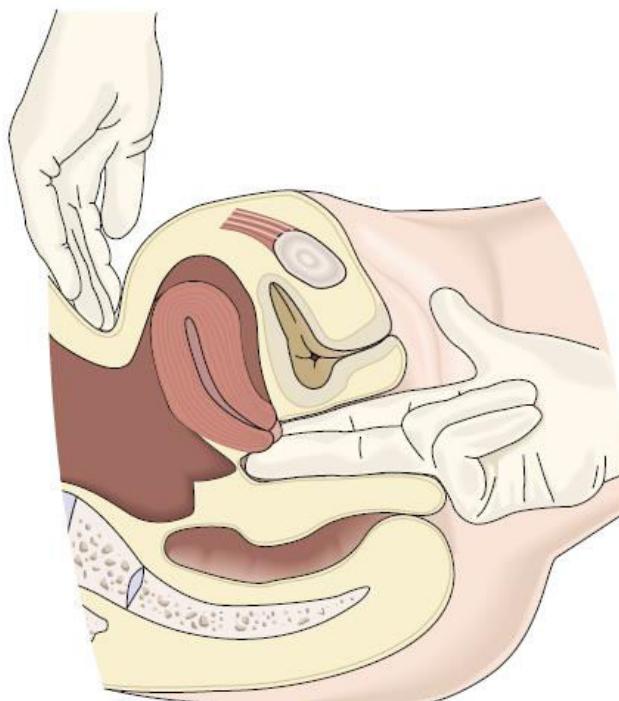
NB: Adnexal tenderness suggests pelvic inflammatory disease (PID)

. Assess the strength of pelvic muscles as you withdraw the pelvic fingers

Ask the patient to squeeze her pelvic muscles around your fingers as hard and as long as possible

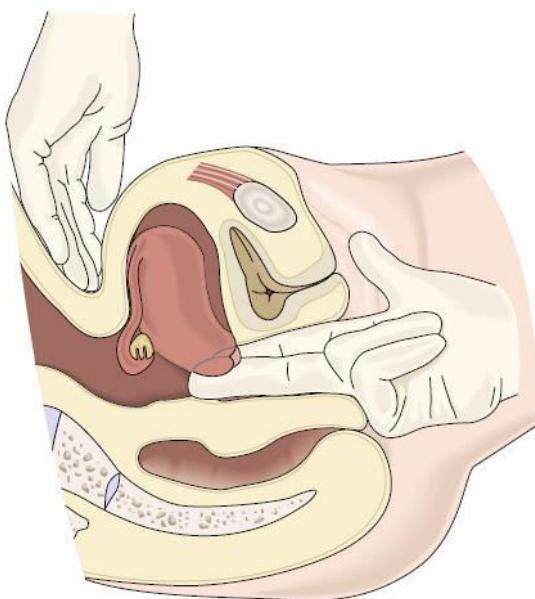
Full strength is noticed by snugly compressing the fingers and moving them upward and inward lasting for ≥ 3 seconds

. Withdraw your pelvic fingers



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 10.7 Technique of bimanual examination of the uterus



© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 10.8 Technique of bimanual examination of the adnexa (Ovary and adjacent structures)

Rectovaginal examination

Reintroduce your index finger into the vagina and your middle finger into the rectum

Repeat the maneuver of bimanual examination

Rectovaginal examination is especially valuable in assessing retroverted uterus

Wipe off the external genitalia and anus after withdrawing the fingers

Vaginal discharge syndrome

Vaginitis and cervicitis

Disease	Etiology
Bacterial vaginosis	<i>Gardnerella vaginalis, Mycoplasma hominis</i>
Vulvo-vaginal candidiasis	<i>Candida albicans</i>
Trichomonal vaginosis	<i>Trichomonas vaginalis</i>
Muco-purulent Cervicitis	<i>Neisseria gonorrhoea, Chlamydia trachomatis</i>

Vaginitis

Majority of cases of vaginitis are not sexually transmitted infections, and caused by endogenous normal flora of the vagina

1. Bacterial vaginitis

Etiology: *Gardnerella vaginalis*

It causes endogenous reproductive tract infection with fishy odor homogenous vaginal discharge

Presence of Clue cells (distorted vaginal epithelial cells coated heavily with gram-negative coccobacilli) in wet mount smear of the vaginal discharge is characteristic of *Gardnerella vaginalis* infection

Predisposing factors: Use of antiseptic/ antibiotic vaginal preparations, or absent/reduced vaginal douching

2. Vulvo-vaginal candidiasis

Etiology: *Candida albicans*

Non-offensive whitish curd-like vaginal discharge with vulval itching and soreness, and erythematous vulva with excoriations from scratching and vulval edema on examination

Predisposing factors: Use of antiseptic/ antibiotic vaginal preparations, or absent/reduced vaginal douching

3. Trichomonas vaginitis

Etiology: *Trichomonas vaginalis*

Sexually transmitted infection which is characterized by offensive profuse foaming greenish-yellow vaginal discharge and vulval itching

Cervicitis

All cases of muco-purulent cervicitis are caused by sexually transmitted bacteria

Etiology Neisseria gonorrhoea, Chlamydia trachomatis

Both bacteria cause cervicitis, and major cause of sexually transmitted infection who present with mucopurulent vaginal discharge with pelvic pain and cervical motion tenderness

Redness, contact bleeding, spotting and endocervical discharge in speculum examination

Positive cervical motion/excitation tenderness on vaginal digital examination

Genital ulcer syndrome

All genital ulcers are sexually transmitted infection

Causative organism	Disease
<i>Treponema pallidum</i>	Syphilis
<i>Haemophilus ducreyi</i>	Chancroid
<i>Chlamydia trachomatis</i>	LGV (lymphogranulomavenerum)
<i>Herpes simplex virus-type 2</i>	Genital herpes

Table 10.3 Causes of genital ulcer

Disease	Genital ulcer	Inguinal Lymphadenopathy
Syphilis	Non-tender, indurated clean based ulcer	Non-tender, non-suppurating rubbery bilateral LAP
Chancroid	Tender, non-indurated shallow ragged ulcer	Suppurative, tender lymphadenitis
LGV	Small, painless papules may be seen	Non-tender, suppurative lymphadenitis with multiple draining sinus tracts
Genital herpes	Tender, multiple vesicles coalescing to form an ulcer	Non-suppurative, tender, bilateral LAP

Pregnant woman

Learning objective

At the end of this lesson, the student should be able to:

1. List main symptoms in obstetric history taking
2. Perform and interpret modified Leopold's maneuver and finding
3. Describe systemic changes during pregnancy

Obstetric history taking and Clinical examination

Obstetric history taking

Current Parity, gravidity

Physiologic amenorrhea (duration), GA (gestational age) by date, EDC (expected date of confinement)

Ante-partal bleeding (placenta previa or abruptio placenta)

Pregnancy-induced hypertension (pre-eclampsia, eclampsia)

Gestational diabetes

Multiple pregnancies

Pregnancy and other medical illnesses, including HIV infection

Obstetric history

Sociodemographic history- age, income, address, woman's attitude toward her pregnancy

Personal and family history of hypertension, diabetes, cardiac disease

Current parity and gravidity

Prior mode of delivery (instrumental delivery, caesarian section)

Prior history of miscarriages, still births, preterm and post term deliveries

History of exposure to teratogenic drugs, and level of stress

History of smoking, use of alcohol or illicit drugs

Duration of amenorrhea (gestational age by date)

Expected weeks of gestation by date if last menstrual period (LMP) is known

. Count in weeks from the first day of the LMP (menstrual age) OR the day of conception (conception age)

Menstrual age is used most frequently to express the weeks of gestation calculated by dates

The 1st day of the LMP is used to calculate the expected date of confinement (EDC)

Physical examination and Clinical history taking

The EDC can be determined by adding 7 days to the 1st day of the LMP, subtracting 3 months and adding 1 year (Naegle's rule)

Early symptoms of current pregnancy: tenderness and increased size of breasts, urinary frequency, nausea and vomiting, easy fatigability, GA by week of quickening (mother's feeling of fetal movement, usually occurs at 20 wk of gestation)

Current history of antepartal bleeding (R/o threatened abortion, antepartal hemorrhage due to placenta previa or abruptio placenta)

Current history of vaginal discharge (note for color, amount, odor and consistency) and vulval itching

Past history of sexually transmitted infections (STIs) such as gonorrhea, syphilis or herpes genitalis

Assess risk factors and clinical stigma of HIV infection (unprotected sexual intercourse, previous or current STIs, chronic diarrhea, and herpes zoster)

Obstetric Examination

Blood pressure (BP)

Record the blood pressure in every visit of the pregnant women

In early and mid pregnancy, BP is normally lower than the baseline BP

High BP prior to 24 weeks of gestation indicates chronic hypertension

High BP after 24 weeks of gestation indicates pregnancy-induced hypertension (preeclampsia)

Weight

Weigh the woman in every antenatal visit

Weight loss may occur in 1st trimester due to excessive vomiting (Emesis gravidarum)

HEENT

Mask of pregnancy (chloasma) is normal in pregnancy

Chloasma is referred as hyper pigmented patches around the cheeks and across the bridge of the nose

Conjunctival pallor: Anemia in pregnancy may occur normally due to hemodilution resulting from disproportionate increase in plasma volume

Nasal congestion and nose bleeds are common during pregnancy

Gingival hypertrophy with bleeding is common during pregnancy

LGS

Mild symmetric thyroid gland enlargement is expected in pregnancy

Breast

Inspect the breasts and nipples

Marked venous pattern and prominent montgomery's glands occurs in pregnancy

Tender and nodular enlarged breast is noticed in pregnancy

Palpate the breast for breast mass/lump

Compress each nipple between your index finger and thumb, and express colostrum from the nipple

Bloody and purulent discharges from the nipple are pathological

Lungs

Shortness of breath with tachypnea occurs late in pregnancy

Heart

Upward and laterally displaced PMI (point of maximal intensity) is noticed in pregnancy

S₃ may occur in pregnancy due to rapid ventricular filling from increased blood volume

Soft, blowing, flow murmur occurs in pregnancy due to increased blood volume

Abdomen

Semi-sitting position with her knees flexed

Inspect for surgical scars, striae, shape and contour of the abdomen

Purplish striae and linea nigra are normal in pregnancy

Scars of Pfennestiel incision at suprapubic area indicates previous caesarean section

Palpate the abdomen for abdominal masses and organs, fundal height, fetal movements and uterine contraction

Measure for fundal height- Measure from the top of symphysis pubis to the top of uterine fundus along the midline of the abdomen using tape measure

Quickening is noticed by the mother at 18-20 weeks of gestation, and felt by the examiner after 24 weeks of gestation

Braxton-Hick's contraction is noticed late in pregnancy

Modified Leopold's maneuver

Adjunct to palpation of the pregnant woman from 28 weeks on wards

Determine lie (longitudinal, transverse, oblique), presentation (cephalic, breech), attitude, descent and estimated weight

1st maneuver (Upper pole)

Stand at the woman's side facing her head

Gently palpate with the finger tips of both examining hands to determine upper pole of the uterine fundus

Most commonly, fetal buttocks are at the upper pole in longitudinal lie

2nd maneuver (sides of maternal abdomen)

Place each hand on each side of the woman's abdomen, and palpate the fetal parts

The hand on the fetal arms and legs feels irregular bumps while the hand on the fetal back feels regular and smooth



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 10.9 Leopold's maneuver to determine fetal lie and location of the back

3rd maneuver (lower pole of fetal parts)

Stand at the woman's side facing her feet

Palpate the fetal part occupying the lower pole above the symphysis pubis with palmar surface of fingers of both hands

Note whether the hands diverge with downward pressure or stay together

The fingers feel a smooth, firm rounded surface on both sides in cephalic presentation

If hands diverge, the presenting part descended into pelvic inlet



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 10.10 Leopold's maneuver to determine presenting part

4th maneuver (confirmation of the presenting part)

Pawlick's grip

Grasp the lower pole of the fetal part with your dominant hand and the upper pole of fetal part with your non-dominant hand

Distinguish between the head and the buttock: fetal head feels smooth, firm and rounded and the buttock feels firm and irregular

Fetal heart beat (FHB)

Auscultate the fetal heart using fetoscope or doptone

Note for FHB location and rhythm

FHB is audible after 18 weeks of gestation

Normal FHB is usually in the 160s during early pregnancy, and then slows to between 120s and 140s near term

Location of FHB

Midline of the lower abdomen from 12-18 weeks of gestation

FHB is best heard at fetal back or chest after 18 weeks of gestation

Rhythm of FHB

Beat-to-beat variation of 10-15 bpm over 1-2 minutes is expected

Lack of beat-to-beat variance of FHB late in pregnancy indicates fetal compromise



© Elsevier. Glynn M, Drake WM. Hutchison's Clinical Methods 23e

Fig 10.11 Listening to fetal heart using 'Pinard stethoscope' (fetoscope)

Genitalia

. Inspect external genitalia for warts, ulcers, abnormal vaginal discharge, and Bartholin's and Skene's gland swellings

Enlargement of labias and clitoris are normal in pregnancy

Look for episiotomy (perineal incision to facilitate delivery of an infant) scars

Palpate Bartholin's and Skene's glands for tenderness

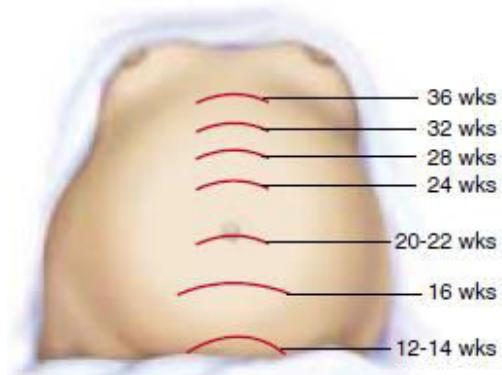
Check for cystocele and rectocele

Speculum examination to visualize vaginal wall, cervix and for any vaginal discharge

Inspect the vaginal wall for color, discharge, rugae and relaxation

Bluish color, deep rugae and milky vaginal discharge (leukorrhea) are normal in pregnancy

Inspect the vagina for color, shape, and healed lacerations



EXPECTED HEIGHT OF THE UTERINE FUNDUS BY MONTH OF PREGNANCY

© Lippincott. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12e

Fig 10.12 Fundal height and GA by week of pregnancy

Bimanual examination

Technique:

- . Insert lubricated index and middle fingers into the introitus, palmar side down with slight pressure downward on the perineum
- . Slide the fingers into the posterior vaginal vault
- . Gently turn the fingers palmar side up while maintaining downward pressure
- . Gently place your fingers in the cervical os, and then sweep around the surface of the cervix

Nulliparous woman has closed cervical os while multiparous woman cervical os admits a finger tip

Inner cervical os (narrow passage between endocervical canal and uterine cavity) is closed in nulliparous and multiparous women

- . Estimate the length of cervix by palpating the lateral surface from the cervical tip to the lateral fornix

Effacement of cervix prior to 32 weeks indicate preterm labor

- . Palpate the uterus for size, shape, consistency and position

Early softening of isthmus (Hegar's sign) is characteristic of pregnancy

Globular uterus by 10-12 weeks of gestation

- . Place pelvic fingers at either side of the cervix, and gently lift the uterus to the abdominal hand and estimate uterine size

- . Palpate the adnexas to rule out tubal pregnancy
- . Palpate the pelvic muscle strength while withdrawing examining fingers

Extremities

Inspect hands and legs for edema: physiologic edema occurs in pregnancy

Significant edema in pregnancy may be due to pregnancy-induced hypertension (preeclampsia-eclampsia) or chronic kidney disease

Male Genitalia

Learning objective

At the end of this lesson, the student should be able to:

1. List common causes of scrotal swelling
2. Mention causes of genital ulcer and urethral discharge
3. Show techniques of examining the testes

History taking in male genitalia

. History of urethral discharge (note for color, amount, odor and consistency)

Common causes of urethral discharge in male are Neisseria gonorrhoea and Chlamydia trachomatis (STIs)

. History of penile sores, ulcers or growths/warts, scrotal swelling or pain

Common causes of penile sores/ulcers are genital herpes, syphilis and chancroid

. Past history of STI such as gonorrhea, syphilis or genital herpes

. History of lack of desire for sex

It could be due to medications, medical illnesses or psychogenic

. History of erectile dysfunction (can't attain and maintain penile erection that is adequate to complete the sexual activity)

Erectile dysfunction may be caused by organic (endocrine, neurologic and vascular causes), medications and psychogenic

. History of premature ejaculation (too soon and out of control)

Lack of orgasm with ejaculation is usually psychogenic

. Risk factors and clinical stigma of HIV infection (history of unprotected sexual intercourse, previous or current STIs, chronic diarrhea, or herpes zoster)

Examination in male genitalia

The penis

. Note appearance and size of penis, presence or absence of prepuce, position of external urethral orifice

. Examine the penile shaft for warts, molluscum, ulcers, scabies and rashes

. Examine the scrotal skin for swelling, erythema and ulceration

Phimosis is narrowing of the preputial orifice which prevents retraction of foreskin

It predisposes to recurrent infections of the glans penis (balanitis), prepuce (prosthis) or both (balanoprosthis)

Hypospadias is external urethral orifice at the ventral surface in the midline, anywhere from the glans to the shaft of penis

Epispadias is external urethral orifice on the dorsal surface of penis

The testis

Palpate both testes and note for size, tenderness, consistency and nodularity of the testis

Technique of examination of the testis

- . Patient in supine position and the examiner stands on the right side of the patient
- . Place both hands on the scrotum with right hand being inferior and palpate the testes
- . Support the posterior testis by middle, ring and little fingers of each hand, leaving the index finger and thumb free to palpate the anterior and lateral part of testis
- . Feel for upper pole of testis between approximated index finger and thumb of left hand
- . Feel for lower pole of testis between approximated index finger and thumb of right hand

Normal testes are equal in size (length 3.5-4 cm) and firm in consistency

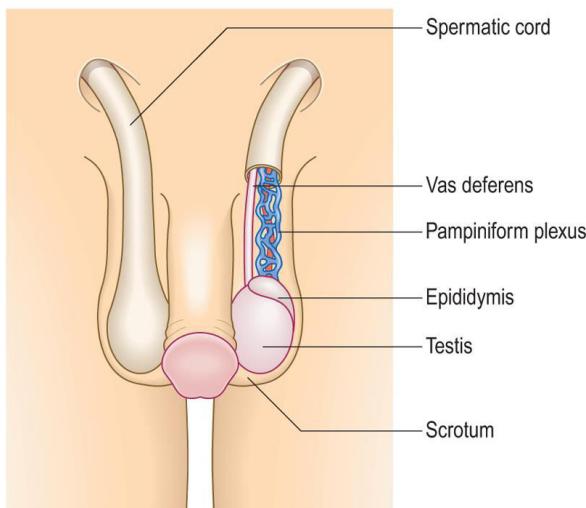
- . Palpate the epididymis on posterior aspect of upper pole of testis
- . Palpate the spermatic cord
 - . Gentle down ward traction on the testis with right hand
 - . Place fingers of right hand behind neck of scrotum and thumb of right hand anteriorly
 - . Press forward with fingers of right hand placed posteriorly and the thumb placed anteriorly
 - . Feel for spermatic cord between the thumb and fingers of right hand
- . Notice for varicocele (dilated tortuous veins), hydrocele of the tunica vaginalis (perform transillumination test to check for hydrocele), spermatic cord cyst

NB: Unilateral hard enlarged testis in an adult is considered to be malignant, unless proved otherwise



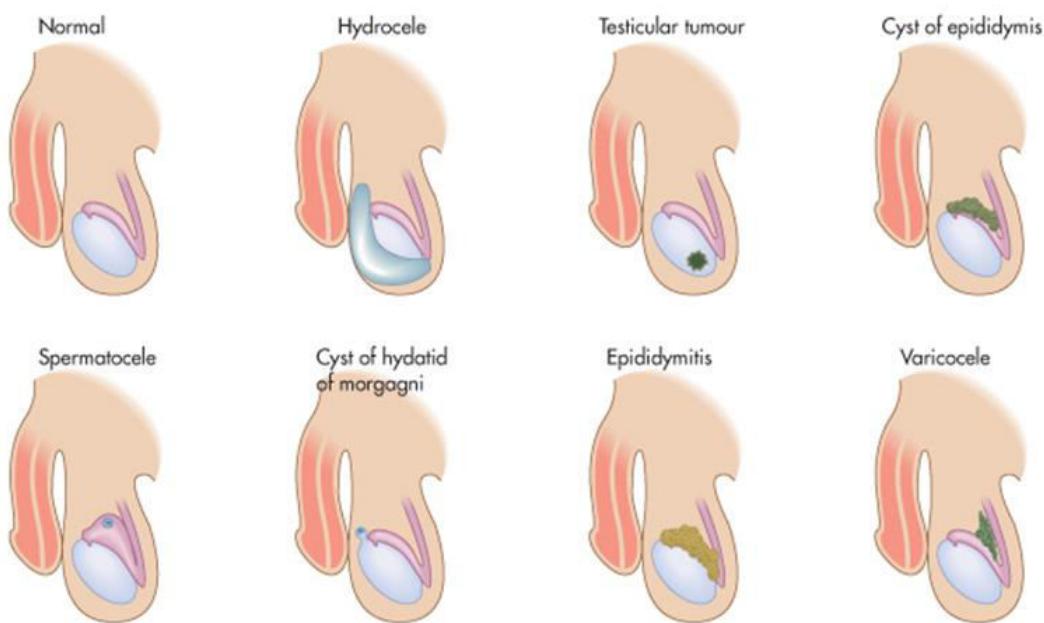
© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th ed

Fig 10.13 Palpation of the testis



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig 10.14 Scrotum and its contents



© Elsevier. Talley & O'Connor. Clinical Examination 5e

Fig 10.15 Cause of scrotal swelling

Chapter Eleven

Integumentary system (Skin, Nail, Hair)

At the end of this lesson, the student should be able to:

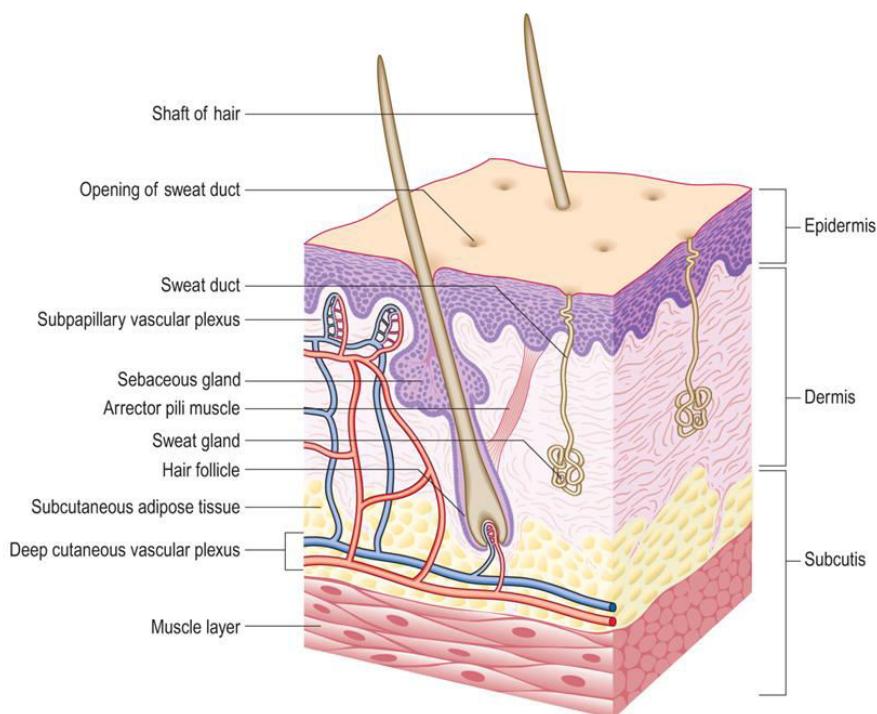
1. Understand how to ask symptoms of skin lesions
2. Identify primary and secondary morphologic skin lesions
3. List clinical syndromes of skin lesions

The skin

The skin is the largest organ of human body. It covers an area of $\sim 2 \text{ m}^2$ and weighs $\sim 4\text{kg}$.

Functions of human skin

- . Protection: Physical, chemical and infection
- . Physiology: Homeostasis of water and electrolytes
- . Thermoregulation
- . Sensation: Specialized nerve endings
- . Immunity: Langerhan's cells and lymphocytes
- . Vitamin D synthesis



© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th e

Fig. 11.1 Structure of the skin

Skin has three layers. These are epidermis, dermis and subcutis.

The epidermis is stratified squamous epithelium. It has four layers (basal, prickle, granular and horny), representing stages of keratin maturation. Hair and nails are specialized epidermal structures.

The dermis is connective tissue containing specialized structures like sebaceous gland, sweat gland and hair follicle.

The subcutis is loose layer of connective tissue and fat.

History taking

Symptoms of skin lesions: Skin eruption (rash), itch (pruritis), growth (lump), disfigurement, etc...

Questions to ask in patients with skin lesions

How long has the lesions been present? (duration)

How did it look when it first appeared, and how is it now different? (morphology)

Where did it first appear? Where is it now? (distribution)

What associated symptoms present? eg. itch, pain, etc...

Was it related to sun exposure, sexual exposure?

Were constitutional symptoms present? eg. fever, malaise, weight loss, etc...

Was systemic disease present? eg. diabetes, connective tissue diseases, inflammatory bowel disease

Are any other family members affected?

Was there recent travel history?

What does the patient think caused the skin lesions? (drugs, personal care products, occupational or recreational exposure)

What treatment has been used?

Physical examination

The skin should be fully exposed, preferably in natural light.

1. Color and pigmentation

Pallor is abnormal whitening of skin and buccal mucosa. Persistent pallor is due to anemia of any cause. Conjunctival and mucosal pallor is a better indicator of anemia than the skin color.

Pigmentation is abnormal coloration with or deposition of pigment.

Dark-brown pigmentation: Abnormal deposition of hemosiderin (eg. hemochromatosis)

Jaundice: Yellow pigmented skin

Depigmentation: Absence of melanin within the skin

- . Albinism-absence of skin pigment
- . Vitiligo-loss of pigment in affected skin

2. Skin lesions and eruptions (rash)

Characterize morphology, distribution and configuration of skin lesions using inspection and palpation technique (put on gloves if the skin is broken).

Observe for size, shape, color and border changes. Palpate the lesions with your finger tips, noting consistency, tenderness, temperature, depth and mobility.

a. Morphology of skin lesions

1. Primary skin lesions: macule, papule, plaque, nodule, papilloma, vesicle, bulla, pustule, wheal, telangiectasia, petechiae, purpura, ecchymosis, erythema, burrow, and comedo.
2. Secondary skin lesions (evolving from primary skin eruption): Scale, crust, excoriation, lichenification, fissure, erosion, ulcer, scar, atrophy, and striae.

Atrophy	Loss of epidermis, dermis or both, thin, translucent and wrinkled skin, visible blood vessels
Bulla	A fluid-filled blister >5 mm in diameter
Burrow	A tunnel in epidermis caused by a parasite, e.g. <i>Acarus</i> in scabies
Callus	Local hyperplasia of horny layer on palm or sole, due to pressure
Comedo	A plug of sebum and keratin wedged in a dilated pilosebaceous orifice on the face
Crust	Dried exudate, e.g. serum, blood or pus, on the skin surface
Cyst	A nodule consisting of an epithelial-lined cavity filled with fluid or semisolid material
Ecchymosis	A macular red or purple haemorrhage, >2 mm in diameter, in skin or mucous membrane
Erosion	A superficial break in the epidermis, not extending into dermis, heals without scarring
Erythema	Redness of the skin due to vascular dilatation
Excoriation	A superficial abrasion, often linear, due to scratching
Fissure	A linear split in epidermis, often just extending into dermis
Freckle	A macular area showing increased pigment formation by melanocytes
Lichenification	Chronic thickening of skin with increased skin markings, from rubbing or scratching
Macule	A localized area of colour or textural change in the skin
Milium	A small white cyst that contains keratin
Nodule	A solid elevation of skin >5 mm in diameter
Papilloma	A nipple-like projection from the surface of the skin
Papule	A solid elevation of skin <5 mm in diameter
Petechia	A haemorrhagic punctate spot 1-2 mm in diameter
Plaque	A palpable elevation of skin >2 cm diameter and <5 mm in height
Purpura	Extravasation of blood resulting in redness of skin or mucous membranes
Pustule	A visible collection of pus in a blister
Scale	Accumulation of easily detached fragments of thickened keratin
Scar	Replacement of normal tissue by fibrous connective tissue at the site of an injury
Stria	Atrophic linear band in skin, white, pink or purple, from connective tissue changes
Telangiectasia	Dilated dermal blood vessels resulting in a visible lesion
Ulcer	A circumscribed area of skin loss extending into the dermis
Vesicle	A clear, fluid-filled blister <5 mm in diameter
Wheal	A transitory, compressible papule or plaque of dermal oedema, red or white, indicating urticaria

© Churchill Livingstone. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th ed

Table 11.1 Morphology of skin eruptions

b. Distribution of skin lesions

Is it symmetrical (generalized) or asymmetrical (localized)? Symmetry often implies an internal causation whereas asymmetry may imply external factors.

Is the skin eruption centripetal or centrifugal? eg. chicken pox and pityriasis rosea are centripetal while erythema multiforme and erythema nodosum are centrifugal.

Is there flexor or extensor bias in body distribution? eg. eczema is flexor while psoriasis is extensor in body distribution

Are only exposed body areas affected?

Are the genitalia involved?

c. Configuration (arrangement) of each skin lesion

- . Nummular/discoid (round or coin-like)
- . Annular (ring-like)
- . Circinate (circular)
- . Arcuate (curved)
- . Gyrate/serpiginous (Wave –like)
- . Linear (in a line)
- . Grouped (clustered)
- . Reticulate (net-like)

Correlation of physical signs and skin diseases

- a. Erythrosquamous eruptions: Red and scaly, asymptomatic or itchy, well demarcated or diffuse bordered skin eruptions
- . Psoriasis (bright pink plaques with silvery scale)
 - . Atrophic eczema (diffuse erythema with fine scales)
 - . Pityriasis rosea (paler pink, scaly, macular lesions)
 - . Nummular eczema (round patches)
 - . Contact dermatitis (irritant or allergic)
 - . Dermatophytosis (ring worm)
 - . Lichen planus (violet colored polygonal papules)
 - . Secondary syphilis (flat, red, hyperkeratotic lesions)

b. Blistering eruptions (blisters and vesicles)

. Traumatic burns and blisters

. Bullous impetigo

. Viral blisters (herpes simplex, varicella)

. Bullous erythema multiforme

. Bullous pemphigoid

. Dermatitis herpetiformis

. Pemphigus

. Porphyria

. Epidermolysis bullosa

. Dermatophyte infections

. Acute contact dermatitis

c. Erythroderma: Inflammatory, erythematous and exfoliative full thickness skin lesions with involved muscle mass loss and edema. It may present as a dermatological emergency.

. Eczema

. Psoriasis

. Drugs eg. phenytoin, allopurinol, etc...

. Pityriasis rubra pilaris

. Lichen planus

. Pemphigus foliaceus

. Dermatophytosis

. Mycosis fungoides (sezary syndrome), leukemia, lymphoma

. Hereditary disorders

d. Pustular and crusted lesions: Primarily infectious process or inflammatory skin lesions

. Acne vulgaris

. Impetigo

. Folliculitis

. Acne rosacea

. Viral lesions

- . Pustular psoriasis
 - . Drug eruptions
 - . Dermatophyte infections
-
- e. Dermal plaques: Localized thickening of skin, caused by chronic inflammatory process or scarring sclerotic process
 - . Granuloma annulare
 - . Necrobiosis lipoidica
 - . Sarcoidosis
 - . Erythema nodosum
 - . Lupus erythematosus
 - . Morphea and scleroderma
 - . Tuberculosis
 - . Leprosy

3. Lump (tumor)

Approach to diagnosis of a lump

- . Determine site, size, shape, consistency and tenderness
- . Note signs of inflammation (redness, swelling, heat and tenderness)
- . Is a lump fluctuant? Perform ‘transillumination test’ if fluctuant
- . Evaluate site of tissue layer attachment of a lump

Lump in the skin- It moves when the skin is moved

Lump in subcutis- The skin moves over the lump

Lump in muscle-Lump mobility limited with muscle contraction

Lump in nerve- Pressing the lump elicits pins and needles in the distribution of the nerve

Lump in bone- The lump is immobile

- . Palpate regional lymph node site if inflammatory or neoplastic lump is suspected

a. Benign skin tumors

- . Warts
- . Molluscum contagiosum
- . Seborrhoeic keratosis
- . Dermatofibroma
- . Neurofibroma
- . Angioma
- . Xanthoma

b. Malignant skin tumors

- . Basal cell carcinoma
- . Squamous cell carcinoma
- . Malignant melanoma
- . Bowen's disease (squamous cell carcinoma confined to epithelial layer of skin-carcinoma in situ)
- . Secondary deposits

The hair

Hair color and texture are genetically determined racial characteristics. Mongols have black and straight hair, Negroid people have black, curly hair, and Caucasians have brown or black silky hair.

Excess hair growth

Hirsutism: Excess male-pattern terminal hair growth in females.

Hypertrichosis: Excess terminal hair growth in a non-androgenic distribution in males and females.

Hair loss

Baldness in men is genetically determined.

Alopecia (scalp hair loss): Localized or diffuse types

Localized hair loss is caused by ring worm infection (tinea capitis), 2⁰syphilis (moth-eaten alopecia), traction alopecia in psychologically disturbed person, autoimmune (alopecia areata).

Diffuse hair loss is caused by hypothyroidism, severe IDA, anti-mitotic chemotherapeutic drugs, autoimmune (alopecia totalis).

The nail

The nail consists of keratinous nail plate over the dorsal surface of the end of each digit.

Nail changes in systemic diseases

Brittle nails: IDA, hypothyroidism, detergent exposure

Clubbing (loss of angle between nail fold and nail plate, or reduction of lozenge-shaped gap during ‘apposition of thumb nails’ (Schamroth’s window test)): Cardiac or respiratory diseases (refer to respiratory system)

Koilonychia (spoon-shaped depression of nail plate): IDA, lichen planus, detergent exposure

Onycholysis (nail separation from nail plate): Psoriasis, thyrotoxicosis, tetracycline (photo-onycholysis), trauma

Onychomycosis (thickening of nail plate): Fungal infection

Pitting nail: Thimble (fine) pitting nail in alopecia areata; coarse pitting nail in eczema

Splinter hemorrhages (longitudinal red streaks in nail plate): Infective endocarditis, collagen vascular disease, psoriasis, trauma

MEDICAL CASE REPORT

Identification

This is E.M., 40-year-old male, married, Orthodox Christian, daily laborer from Metema, North Gondar Zone, admitted to University of Gondar hospital, medical ward D, bed number 24, on January 12, 2009 E.C.

Previous admission

August, 2007 E.C., University of Gondar hospital, Gondar, acute subdural hematoma 2^0 to traumatic head injury, burr hole done to evacuate hematoma, and discharged improved.

Chief complaint

Fever of 1 month duration

History of present illness

The patient was last relatively healthy 1 month back at which time he started to experience a gradual onset of high grade intermittent fever, got worse in the evening and better in the morning, associated with chills, rigors, and drench sweating to the extent it soaked his clothes.

Two weeks after the onset of fever, he began to notice a painless swelling over the left upper quadrant of his abdomen associated with aching abdominal pain, dragging sensation and early satiety. The pain was non-radiating localized to upper abdomen with no specific aggravating or relieving factor.

He visited a local drug store near by, and bought and took Coartum, 4 tabs twice daily for three days thinking it was malaria, but no avail.

1 week before admission, he began to develop blurring of vision, tinnitus, light headedness; and easy fatigability which prohibited him from doing his routine daily activities. He had repeated episodes of bilateral nasal bleeding, which stopped with nasal pack. He had unquantified weight loss to the extent his clothes become loose. He had shortness of breath while engaged in routine activities like loading and unloading objects from lorry.

He lives in kala-azar and malaria endemic area. He had repeated attacks of malaria, last attack being 3 months back.

He had episodes of vomiting of ingested matter, anorexia and nausea, but no bowel habit change.

He gave history of consuming under cooked meat or raw milk, and close contact with domestic animals; but has no contact with animal abortus material.

He had history of unprotected sexual activity, but was not screened for retroviral infection, and had no recurrent oral ulcers, herpes zoster scar, or chronic diarrhea

He had no petechial rash, gum bleeding, hematuria or stool color change.

He had no orthopnea, paroxysmal nocturnal dyspnea or leg swelling.

He had no swelling over the neck, axillae or groin area, and no bone pain.

He had no jaundice, close contact with jaundiced person, blood transfusion, blood letting, ear piercing or tattooing.

He had no joint pain or swelling, malar rash, photosensitivity or oral ulcers.

.He had no cough with expectoration, contact with chronic cougher, or previous treatment for tuberculosis.

He had no history of exposure to herbal or modern medications.

He had no history of exposure to herbicides, pesticides, organic solvents or ionizing radiation.

There was no similar illness in any of family members.

He visited this hospital for better care as treatment in near by health institution didn't help him. Then, he was admitted to the medical ward of the hospital supported by family members with significant weight loss, with ashen gray skin color change noticed by relatives.

Past illness

He had history of measles, mumps, and chicken pox, but no other childhood illness.

Functional enquiry

HEENT

No head trauma

No eye pain, discharge or excessive lacrimation.

No ear pain, discharge or decreased hearing.

No nasal discharge.

Lymphoglandular system: Mentioned in HPI

Respiratory system: Mentioned in HPI

Cardiovascular system: Mentioned in HPI

Gastointestinal system: Mentioned in HPI

Genitourinary system: No flank pain, urgency, frequency of micturition, or suprapubic pain

Musculoskeletal system: Mentioned in HPI

Integumentry system: Mentioned in HPI

Central nervous system: No loss of consciousness, abnormal body movement or weakness of extremities; others mentioned in HPI

Personal history

He was born in a small village 5 km away from Mettema, North Gondar zone, in 1969 E.C. He was breast fed and spent a healthy childhood. He grew up with his family as a shepherd. He attend formal education upto grade 6, and then engaged in labor work. He drinks on casual days but no history of smoking or chewing khat. He is married and has two kids. All are alive and healthy.

Family history

Mother is alive and healthy. Father died at age of 65 years due to unknown cause. He had one brother and two sisters. All are alive and healthy. He is the third child for his family. No family history of diabetes, hypertension or asthma.

Physical Examination

General appearance

Conscious and cooperative; chronically sick looking, looks under-nourished.

Vital sign

BP=110/70 mmhg, right arm, supine position

PR= 80 bpm

RR=20 breathe per minute

T⁰=36.6⁰C, right axilla at 10:00 PM

Anthropometric measurement

Weight-50kg

Height-1.65

MBI=18 kg/m², mildly under-nourished

Mid upper arm circumference (MUAC) =20cm, under-nourished

HEENT

Head: There is clearly visible scalp surgical scar and depressed skull at left temporal area.

Eye: Pale conjunctivae, no icteric sclerae, no eye discharge or periorbital swelling

Ear: Normal contour of pinna, clear external ear canal; no ear discharge, no mastoid or tragus tenderness

Nose: Central nasal septum, no active nasal bleeding or discharge

Mouth and throat: Pale tongue and buccal mucosa; no gum bleeding, ulcers or fissures on lips, or tooth caries

Lymphoglandular: No palpable lymphadenopathy in accessible sites, no anterior neck swelling, or no breast lump

Respiratory system

Inspection: No cyanosis of palm of hands or clubbing of fingers; no cyanosis of lips or tongue; regular and shallow breathing pattern; no subcostal or intercostal retraction; no use of accessory muscle of respiration; no chest wall deformity, chest wall moves symmetrically with respiration

Palpation: Trachea is centrally located; no chest wall tenderness or subcutaneous crepitation; normally comparable tactile fremitus; symmetric chest expansion, and measures 6 cm along mid chest.

Percussion: resonant all over the chest; diaphragmatic chest expansion is 4 cm.

Auscultation: Vesicular breath sound heard all over the chest

Cardiovascular system

Arterial system: All accessible peripheral arteries are palpable; full in volume and regular in rhythm; no radio-femoral delay; no arterial cording.

Venous system: No superficial visible distended veins on the neck. JVP is not raised.

Precordial examination:

Inspection: No precordial bulge; active precordium; apical impulse is visible at 5th intercostal space medial to left midclavicular line.

Palpation: PMI is at apical impulse. PMI is diffuse and tapping. No palpable heart sound; no heave or thrill.

Auscultation: S1 and S2 are well heard. There is grade-3, mid-systolic, high pitched, harsh murmur at pulmonic area which radiates to the neck. No gallop.

Abdominal examination

Inspection: Symmetric abdomen that moves with respiration; no flank fullness; flat umbilicus with transverse slit; no visible peristalsis, no pulsation or distended veins; no scar or pigmentation; hernia sites are free.

Palpation: Superficial and deep palpation

Superficial palpation: No tenderness, guarding or rigidity. There is superficially palpable mass on left side of the abdomen.

Deep palpation: There is a non-tender mass on the right upper quadrant of the abdomen, which is 4 cm below the right costal margin along right midclavicular line; the mass is smooth surface, round edge, firm in consistency, moves with respiration. Not bimanually palpable.

There is non-tender mass on the left upper quadrant of the abdomen which is 10 cm below the left costal margin along the splenic growthline, moves with respiration; difficult to enter fingers through left costal margin; medial notch is palpable. The mass is smooth surface, round edge, firm in consistency. Not bimanually palpable. It is massive splenomegaly.

Percussion: Tympanic all over the abdomen, except at masses which is flat to percussion. No shifting dullness or fluid thrill. Total vertical liver span is 16 cm.

Auscultation: Normoactive bowel sound with a rate 13/min. No friction rub or bruit over the enlarged spleen or liver.

Per digital rectal examination: No rectal mass or ulceration or nodules. Normal anal sphincter tone. No perianal nodules, ulcers or discharges.

Genitourinary system: No costovertebral angle tenderness or suprapubic tenderness

Musculoskeletal system

Look: No deformity, no scar or pigmentation. Symmetrically atrophied muscle bulk on upper and lower extremities

Feel: No tenderness or swelling on extremities

Move: Full active range of movement of extremities

Measure: Comparable circumference and length of extremities

Integumentary system: The skin is dry and warm. Pale palm of hands and sole of feet. No petechial rash. No clubbing or deformity of nails.

Neurologic examination

Mental status examination: Oriented in time, place and person

Glasgow comma scale (GCS): Eye opening; spontaneous =4/4; best verbal response; oriented=5/5; best motor response; obeys command=6/6; Total=15/15

Cranial nerve examination

CN-I: Can identify the odor of lemon in each nostril

CN-II: Has comparable visual field with the examining physician. Normal direct and consensual light reflex.

CN-III, IV and VI: Can move his eye in all cardinal direction.

CN-V: Sensation to light touch and pain is intact over his face. Muscle of mastication contract while clenching his teeth. Intact corneal reflex.

CN-VII: He can smile, frown his forehead, and puff out his cheeks. He can close his eyes against resistance. Intact corneal reflex.

CN-VIII: He can hear finger rub in both ears. Weber's test and Rinne's test are intact.

CN-IXand X: Uvula is centrally located; soft palate rise in the mid line when he says 'Ah'. Intact gag reflex.

CN-XI: He can shrug his shoulder and turn his head against resistance.

CN-XII: He can protrude and move his tongue in both directions.

Motor examination

Inspection: Comparable muscle bulk in both upper and lower extremities. No spontaneous or provoked fasciculation.

Palpation: Normal tone while moving extremities along the joints; he can lift his extremities against full resistance (power=5/5),

Deep tendon reflex is normal

	Biceps	Triceps	Supinator	Knee	Ankle
Right	++	++	+	++	+
Left	++	++	+	++	+

Superficial reflex is normal: Down going plantar response; intact abdominal and corneal reflexes

Sensory examination: Light touch, pain, temperature, vibration and position sensation are intact.

Coordination: Tandem walk, finger-to-nose and heel-to-shin movements are intact.

Meningeal irritation signs: No neck stiffness; Kernig's and Brudzinsky's signs are negative.

Subjective summary

A 40-year-old male patient from Metema , North Gondar Zone, presented with a high grade intermittent fever of 1 month duration associated with chills, rigors and profuse sweating. He also has loss of appetite and weight. He noticed abdominal swelling with dragging sensation. He also has blurring of vision, tinnitus, light headedness, easy fatigability and epistaxis. He lives in Kala-azar endemic area and had repeated attacks of malaria.

Objective summary

He is chronically sick looking patient, conscious and cooperative. Looks under-nourished.

Vital sign: BP=110/70 mmhg, PR=80bpm, RR=20 breaths per minute, T0=36.6 0c, All are within normal limits.

BMI=18 kg/m², mildly under-nourished. MUAC=20 cm, under-nourished.

He has pale conjunctivae, tongue and buccal mucosa, palm of hands and sole of feet. Mid-systolic murmur at pulmonic area in CVS examination. Abdominal examination revealed huge splenomegaly and hepatomegaly.

Diagnosis: Visceral leishmaniasis

Required investigation

Complete blood count (Hgb, Hct, WBC with differential, platelets, ESR)

Blood film

Peripheral morphology

Blood culture

Serologic tests (rK-39, DAT test, ELISA)

Brucella serologic test, Hepatic viral markers (HBsAG, Ant-HBc-antibody, Anti-HCV-antibody)

HIV serologic tests, CD₄ count if positive HIV serology

Tissue aspirate from spleen or liver (FNAC)

Bone marrow aspirate and biopsy: morphology, flow cytometry, cytogenetics

Organ function tests (LFT, RFT), electrolytes

Coagulation profile (PT, aPTT)

Abdominal U/S, CXR

Differential diagnosis (most likely to least likely)

1. Visceral leishmaniasis
2. Chronic myeloid leukemia
3. Non-Hodgkin's lymphoma
4. Hyper-reactive malarial splenomegaly (HMS)

Discussion of differential diagnosis (least likely to most likely)

1. Hyper-reactive malarial splenomegaly (HMS)

HMS is an abnormal immune response to recurrent malarial attack, which is caused by Plasmodia species. It is more common in malaria endemic area, most of tropical regions of the world; commonly seen in older children and young adults. Repeated malarial infections cause uninhibited B-cell production of immunoglobulin and immune complex formation, and stimulate reticuloendothelial tissue hyperplasia characterized by massive splenomegaly. Clinical features include massive splenomegaly +/- perisplenitis, hepatomegaly, hypergammaglobulinemia, anemia +/- pancytopenia, infection of skin, respiratory system or sepsis. Diagnostic criteria for HMS include residence in malaria endemic area ≥ 10 years with recurrent attacks, massive splenomegaly, perisinusoidal lymphocytosis, increased serum titer of IgM and malarial antibodies, pancytopenia and smear negative for malaria. In conclusion, residence in malaria endemic area with repeated malarial attack, and massive splenomegaly favors HMS, but prolonged fever with systemic symptoms is unlikely in HMS, and HMS is a diagnosis of exclusion.

2. Non-Hodgkin's lymphoma (NHL)

NHL represents clonal proliferation of lymphoid cells (B-cell-75%, T-cell-25%) in reticuloendothelial tissue. Cause is unknown in most cases, but etiological factors include pesticide or herbicide exposure, viral and bacterial infections, and inherited or acquired immunodeficiency state. It occurs in middle aged adults. Clinical features include lymphadenopathy +/- organomegaly and systemic symptoms (fever, sweating, and weight loss). Bone pain and symptoms of cytopenia including fever, fatigue and bleeding occurs in advanced disease. In conclusion, systemic symptoms, organomegaly, and clinical features of cytopenia favors advanced extranodal NHL. Tissue biopsy of nodal and extranodal sites is required for morphologic, immunophenotyping and cytogenetic study to settle the diagnosis.

3. Chronic myeloid leukemia (CML)

It is a myeloproliferative disease characterized by clonal proliferation of myeloid cells in bone marrow. No clear etiologic factor is known except ionizing radiation exposure. It is a disease of older adults in westerns data. Ethiopian literatures mention median age of patients with CML is 40ys. Philadelphia chromosome is identified in 90-95% of patients with CML. CML has three phases, named as chronic phase, accelerated phase and blast phase. This patient fits to accelerated/blast phase of CML, due to presence of prominent systemic symptoms, massive splenomegaly and clinical features of cytopenia, like fever, fatigue and bleeding. In conclusion, all clinical symptoms and signs support diagnosis of accelerated/blast phase of CML. All cells of myeloid series in bone marrow aspirate or peripheral smear suggest CML. Cytogenetic study for Ph' chromosome is required to confirm diagnosis of CML.

4. Visceral leishmaniasis (Kalaazar)

Kalaazar means black fever in Hindu. It is an anthroponotic or zoonotic systemic disease caused by an obligate intracellular protozoan, named as Leishmania donovani complex. The vector for transmission of leishmania spp. is named as sandfly (phlebotomus). The disease is prevalent in North West (Mettema, Humera, Wolkait, Llbo/Fogera), North East (Ethio-Djibouti border, Awash valley), and South West (Segen,

Genale, Woito, Konso, Omo, Gambela, Ethio-South Sudan border) regions of Ethiopia. Population migration is main risk factor to acquire the infection. It is transmitted to healthy person by the bite of infected sandfly. Clinical features include fever ≥ 2 weeks, weight loss, splenomegaly, anemia +/- pancytopenia. In conclusion, patient residence in kalazar endemic area, prolonged fever, weight loss and massive splenomegaly, and clinical features of cytopenia including fever, fatigue and bleeding favors kalazar. Positive leishmanial serologic tests (rK-39/ DAT test) or isolation of leishmanial LD-bodies in tissue aspirate from spleen confirm diagnosis of kalazar.

REFERENCES

1. Tsega E. A Guide to Writing Medical Case Reports 1st eds. Addis ababa, Addis Ababa University Press,1985
2. Glynn M, Drake WM. Hutchison's Clinical Methods: An Integrated Approach to Clinical Practice 23rd eds.London,Elsevier Ltd,2012
3. Douglas G, Nicol F, Robertson C. Macleod's Clinical Examination 12th eds.London,Churchil Livingstone Ltd,2009
4. Bickley LS, Szilagyi PG. Bates' Guide to Physical Examination and History Taking 12th eds.Philadelphia,Lippincott Company,2017
5. Manahan FD. Mosby's Expert Physical Exam Hand Book: Rapid Inpatient and Outpatient Assessments 3rd eds.Missouri,Elsevier Ltd,2009
6. Mir MA. Atlas of Clinical Diagnosis 2nd eds.London,Elsevier Science Limited,2003
7. Talley NJ, O'Connor S. Clinical Diagnosis: A Systematic Guide to Physical Diagnosis 5th eds.London, Elsevier Ltd,2007
8. Brazis PW, Masdue JC, Biller J. Localization in clinical neurology 5th eds.Chicago,Edwards Brothers, 2007
9. Campbell WM. DeJong's The Neurologic Examination. 6th eds. Lippincott Williams & Wilkins, 2005
10. Coady D. Clinical Assessment of the Musculoskeletal System: A hand book for medical students 2nd eds. Derbyshire,Arthritis Research Campaign,2005
11. Malanga GA, Mautner K. Musculoskeletal Physical Examination 2nd eds.Philadelphia,ElsevierLtd,2017
12. Kasper DL, Braunwald E, Fauci A *et al.* Harrison's Principles of Internal Medicine 19th eds.New York,Mc Graw-Hill,2016

