

## Outlines:

- Preparing For Physical Examination
- Sequence Of Physical Examination
- Initial Observations

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Introductory: Section 1 CH.3 General Aspect Of Examination.

Note: This summary contains all Macleod's important notes.





## Preparing for physical examination:

## 1- wash your hands before &amp; after the examination.

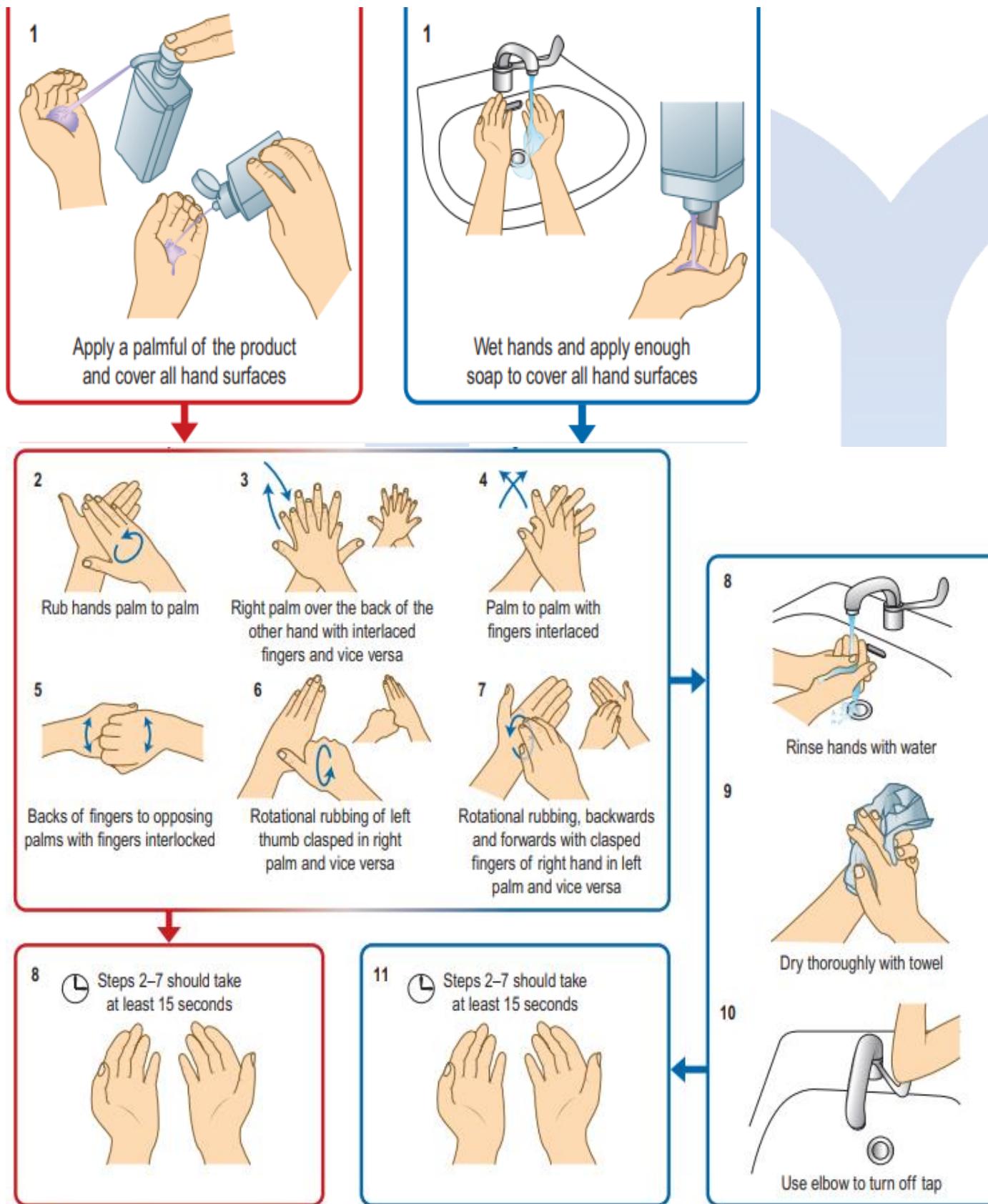


Fig. 3.1 Techniques for hand hygiene. From WHO Guidelines on Hand Hygiene in Health Care First Global Patient Safety Challenge Clean Care is Safer Care; [http://www.who.int/gpsc/clean\\_hands\\_protection/en/](http://www.who.int/gpsc/clean_hands_protection/en/) © World Health Organization 2009. All rights reserved.

## 2- Privacy:

- Privacy is **essential** when you examine a patient.
- **Pulling the curtains around the bed** in a ward obscures vision but not sound.
- **Talk quietly but ensure good communication**, which may be difficult with deaf or elderly patients.

## 3- The room should be warm and with a good light source.

Subtle abnormalities of complexion such as **mild jaundice** are easier to detect in natural light.

## 4- The height of the examination couch or bed should be adjustable, with a step to enable patients to get on to it easily.

An adjustable backrest is essential, **particularly for breathless patients who cannot lie flat**.

## 5- Always Introduce yourself to the patient and Seek permission, shake hands (which may provide diagnostic clues; Box 3.1).

## 3.1 Information gleaned from a handshake

Features	Diagnosis
Cold, sweaty hands	Anxiety
Cold, dry hands	Raynaud's phenomenon
Hot, sweaty hands	Hyperthyroidism
Large, fleshy, sweaty hands	Acromegaly
Dry, coarse skin	Regular water exposure Manual occupation Hypothyroidism
Delayed relaxation of grip	Myotonic dystrophy
Deformed hands/fingers	Trauma Rheumatoid arthritis Dupuytren's contracture



### 6- Expose the areas of the body to be examined:

Cover the rest of the patient with a **blanket or sheet** to ensure that he or she does not become cold.

**Note:** Avoid unnecessary exposure.

### 7- Tactfully ask relatives to leave the room before the physical examination:

Sometimes it is appropriate for a relative to remain if the patient is very apprehensive, if you need a translator or if the patient requests it.

### 8- Parents should always be present when you examine children.

**9- Always offer a chaperone for any intimate** (breast, genital or rectal) **examination** to prevent misunderstandings and to provide support and encouragement for the patient.

- A chaperon is someone of the patient's same gender from medical staff who witnesses your examination, e.g., a nurse.
- Record the chaperone's name.
- If patients decline the offer, respect their wishes and record this in the notes.

### 10- Collect together all the equipment you need before starting the examination.

#### 3.2 Equipment required for a full examination

- Stethoscope
- Pen torch
- Measuring tape
- Ophthalmoscope
- Otoscope
- Sphygmomanometer
- Tendon hammer
- Tuning fork
- Cotton wool
- Disposable Neurotips
- Wooden spatula
- Thermometer
- Magnifying glass
- Accurate weighing scales and a height-measuring device (preferably a calibrated, wall-mounted stadiometer)
- Personal protective equipment (disposable gloves and apron)
- Facilities for obtaining blood samples and urinalysis

### Sequence of physical examination:

- There is a **common sequence** of physical examination that everyone knows but with time you will develop your own sequence.

- **The usual sequence of examination is:**

**1- Inspection:** Observing with eyes.

**2- Palpation with hands.**

**3- Percussion:** we do focus on that in certain areas as in the chest and the abdomen.

**4- Auscultation:** using the stethoscope.

#### Initial observations

- When does the physical examination should be started?

**FIRST IMPRESSIONS:** The physical examination starts as soon as you see the patient.

Assess patients' **general demeanor and external appearance**, and watch how they rise from their chair and walk into the room.

#### Facial expression and speech

1- A patient's **facial expression and how they interact with you** can provide clues to **their physical and psychological wellbeing** (Box 3.3).

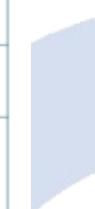
2- **Reluctance to engage in the consultation** may indicate **underlying depression, anxiety, fear, anger or grief**, and it is important to recognize these emotions to ensure that both the physical and the emotional needs of the patient are addressed effectively.

3- **Illness itself may alter demeanor:** **frontal lobe disease** or **bipolar disorders** may lead to **animated disinhibition**, whereas **poverty of expression** may occur in **depression** or **Parkinson's disease**.



### 3.3 Facial expression as a guide to diagnosis

Features	Diagnosis
Poverty of expression	Parkinsonism
Startled expression	Hyperthyroidism
Apathy, with poverty of expression and poor eye contact	Depression
Apathy, with pale and puffy skin	Hypothyroidism



4- Be vigilant for abnormalities in the character of speech, such as:

- **Slurring** due to alcohol.
- **Dysarthria** caused by motor neuron disease.
- **Hoarseness** (which can represent recurrent laryngeal nerve damage).
- **Or abnormality of speech cadence:** Which could be caused by (pressure of speech in hyperthyroidism or slowing of speech in myxedema).



**Face masking (poverty of expression )**  
**Parkinson**

### Startled face (hyperthyroidism)



Fig. 2 A patient before and after successful treatment of primary hypothyroidism.

### Hypothyroidism Face





### Clothing

- Clothing gives clues about **personality, state of mind and social circumstances**.
- Young people wearing dirty clothes may have problems with **alcohol or drug addiction, or be making a personal statement**.
- Unkempt elderly patients with fecal or urinary soiling may be **unable to look after themselves because of physical disease, immobility, dementia or other mental illness**.
- **Anorectic patients** wear baggy clothing to cover weight loss.
- **Consider bloodborne viral infections**, e.g., hepatitis B or C, HIV in patients with tattoos or piercings.
- A Medic Alert bracelet or necklace highlights important **medical conditions and treatments**.



Fig. 3.3 MedicAlert bracelet.



Fig. 3.2 Tattoos can be revealing.



Fig. 3.3 The linear marks of intravenous injection at the right antecubital fossa.

### Gait and posture

- If patients are **ambulant**, watch how they rise from a chair and walk towards you. Are they using a **walking aid**? Is the **gait normal** or is there **evidence of pain, immobility or weakness**?
- Abnormalities of gait can be pathognomonic signs of neurological or musculoskeletal disease: for example, the **hemiplegic gait after stroke**, the **ataxic gait of cerebellar disease** or the **marche à petits pas ('walk of little steps') gait** in a patient with **diffuse cerebrovascular disease or Parkinsonism** (see Fig. 7.17D).

- **Notice any abnormal movements** such as **tremor** (in alcohol withdrawal, for example), **dystonia** (perhaps as a side effect of neuroleptic therapy) or **chorea** (jerky, involuntary movements, characteristic of Huntington's disease).
- **Abnormalities of posture and movement** can also be a clue to the patient's **overall wellbeing**, and may represent **pain, weakness or psychological or emotional disturbance**.

### Hands examination:

- Examination sequence:

#### 1. Inspect the dorsal and then palmar aspects of both hands.

##### Note for:

- **Deformity**.
- **Color**.
- **Nails**.
- **SKIN**.
- **Temperature**.

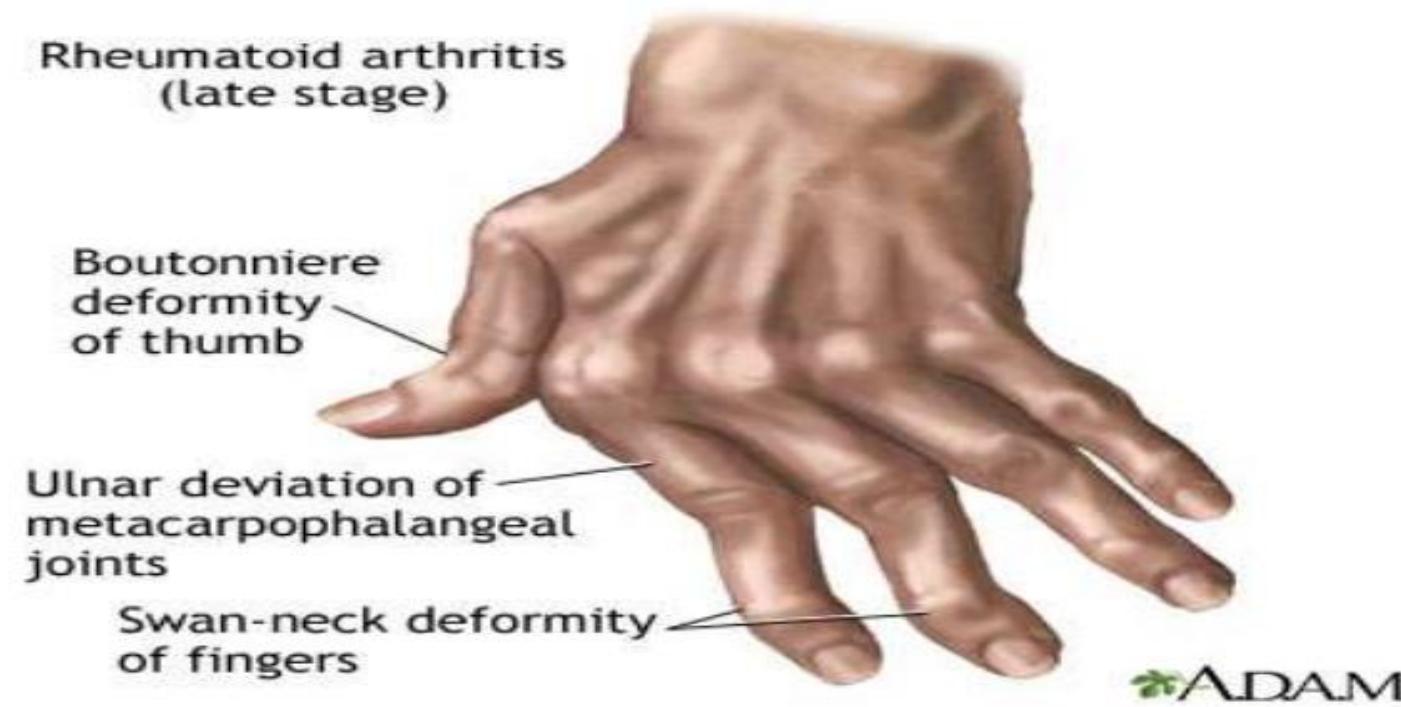
##### Deformity

- **Deformity may be diagnostic**: for example, the **flexed hand and arm of hemiplegia** or **radial nerve palsy, and ulnar deviation** at the metacarpophalangeal joints (MCP) in **longstanding rheumatoid arthritis**.

- **Dupuytren's contracture** is a **thickening of the palmar fascia** causing **fixed flexion deformity and usually affecting the little and ring fingers**.
- **Arachnodactyly** (long thin fingers) are typical of **Marfan's syndrome**.
- **Trauma** is the most common cause of hand deformity.



Fig. 3.13 Dupuytren's contracture.

Arachnodactyly  
in marfan syndrome**Color**

- Look for **cyanosis** in the nail bed and tobacco staining of the fingers.
- Examine the **skin creases** for pigmentation, although pigmentation is normal in many non-Caucasian races.



Fig. 3.8 Tobacco 'tar'-stained finger.



Fig. 3.14 Normal palms. African (left) and European (right).

**Temperature**

In a **cool climate** the temperature of the patient's hand is a good **guide to peripheral perfusion**.

1. In chronic obstructive pulmonary disease (COPD), the hands may be **cyanosed** due to reduced arterial oxygen saturation but **warm** due to vasodilatation from elevated arterial carbon dioxide levels.

2. In heart failure the hands are often **cold and cyanosed** because of vasoconstriction in response to a low cardiac output.

If they are **warm**, heart failure may be due to a high output state, such as **hyperthyroidism**.

**Skin of the hand**

- Skin changes in the hands can indicate **systemic disease**.
- As in the **coarse skin and broad hands of a patient with acromegaly**.
- **Tight, contracted skin** (scleroderma) and **calcium deposits** associated with **systemic sclerosis**.
- The **dorsum of the hand is smooth and hairless** in **children** and in **adult hypogonadism**.
- Manual work may produce **specific callosities** due to **pressure at characteristic sites**.





## Nails

Nail changes occur in a **wide variety of systemic diseases**.

### 3.4 The nails in systemic disease

Nail changes	Description of nail	Differential diagnosis
Beau's lines	Transverse grooves (see Fig. 3.7B)	Sequelle of any severe systemic illness that affects growth of the nail matrix
Clubbing	Loss of angle between nail fold and nail plate (see Fig. 3.8)	Serious cardiac, respiratory or gastrointestinal disease (see Box 3.5)
Leuconychia	White spots, ridges or complete discolouration of nail (see Fig. 3.7C)	Trauma, infection, poisoning, chemotherapy, vitamin deficiency
Lindsay's nails	White/brown 'half-and-half' nails (see Fig. 12.7)	Chronic kidney disease
Koilonychia	Spoon-shaped depression of nail plate (see Fig. 3.7D)	Iron deficiency anaemia, lichen planus, repeated exposure to detergents
Muehrcke's lines	Narrow, white transverse lines (see Fig. 12.6)	Decreased protein synthesis or protein loss
Nail-fold telangiectasia	Dilated capillaries and erythema at nail fold (see Fig. 14.13B)	Connective tissue disorders, including systemic sclerosis, systemic lupus erythematosus, dermatomyositis
Onycholysis	Nail separates from nail bed (see Fig. 3.7A)	Psoriasis, fungal infection, trauma, thyrotoxicosis, tetracyclines (photo-onycholysis)
Onychomycosis	Thickening of nail plate with white, yellow or brown discolouration	Fungal infection
Pitting	Fine or coarse pits in nail (see Fig. 3.7A)	Psoriasis (onycholysis, thickening and ridging may also be present), eczema, alopecia areata, lichen planus
Splinter haemorrhages	Small red streaks that lie longitudinally in nail plate (see Fig. 4.5B)	Trauma, infective endocarditis
Yellow nails	Yellow discolouration and thickening (see Fig. 14.13C)	Yellow nail syndrome



Fig. 3.7 Nail abnormalities in systemic disease. **A** Onycholysis with pitting in psoriasis. **B** Beau's lines seen after acute severe illness. **C** Leuconychia. **D** Koilonychia. (A) From Innes JA. *Davidson's Essentials of Medicine*. 2nd edn. Edinburgh: Churchill Livingstone; 2016.

## Finger clubbing

- **Clubbing:** is **painless soft tissue swelling of the terminal phalanges**. The enlargement **increases convexity** of the nail.
- It may be produced by **growth factors from megakaryocytes and platelets** lodged in nail bed capillaries **stimulating vascular connective tissue**.
- It is an **important sign of major diseases**, although it may be **congenital**.



A



B

Fig. 3.8 Clubbing. **A** Anterior view. **B** Lateral view.

- It usually **takes weeks or months to develop**, and **may disappear if the underlying condition is cured**.
- Clubbing usually **affects the fingers symmetrically**, but **may involve the toes**.
- **Unilateral clubbing** can be caused by **proximal vascular conditions**, e.g., **arteriovenous shunts for dialysis**.
- **Autoimmune hyperthyroidism** may be associated with **thyroid acropachy – clubbing** which is **more pronounced on the radial side** of the hand.





### 3.5 Causes of clubbing

- Congenital or familial (5–10%)
- Acquired

**Thoracic (~70%):**

- Lung cancer
- Chronic suppurative conditions: pulmonary tuberculosis, bronchiectasis, lung abscess, empyema, cystic fibrosis
- Mesothelioma
- Fibroma
- Pulmonary fibrosis

**Cardiovascular:**

- Cyanotic congenital heart disease
- Infective endocarditis
- Arteriovenous shunts and aneurysms

**Gastrointestinal:**

- Cirrhosis
- Inflammatory bowel disease
- Coeliac disease

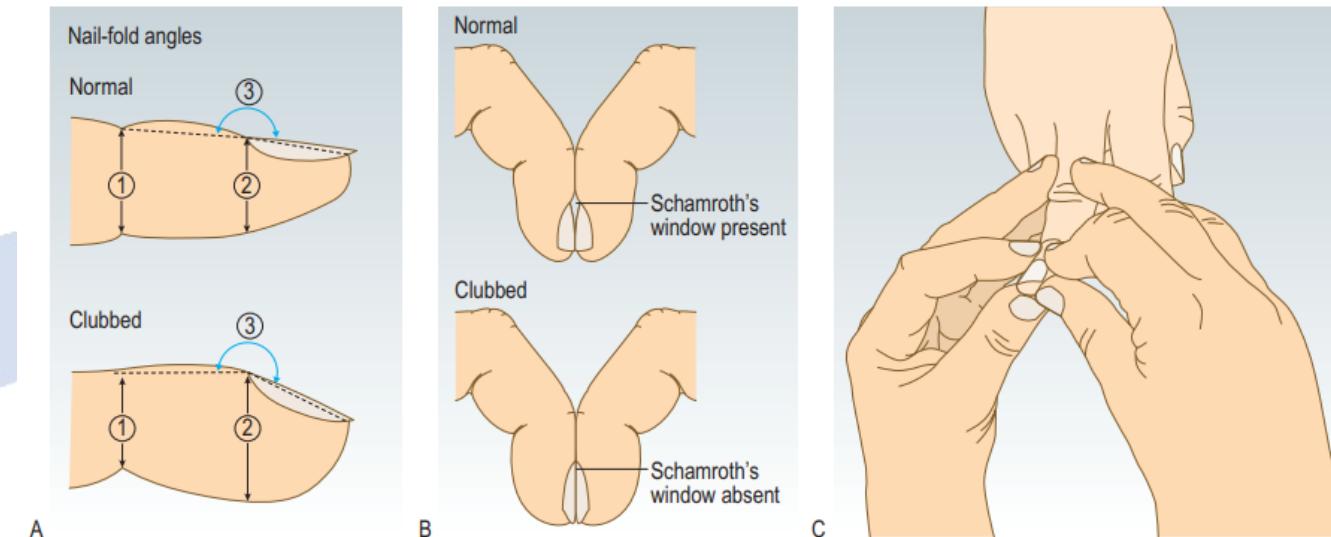
**Others:**

- Thyrotoxicosis (thyroid acropachy)
- Primary hypertrophic osteoarthropathy

### Examination sequence:

#### - Interphalangeal depth ratio: (2/1)

1. Look across the nail bed from the side of each finger. Observe the distal phalanges, nail and nail bed.
2. Measure the anteroposterior distance at the level of the interphalangeal joint. Repeat at the level of the nail bed.
- **Measure the nail bed angle.**
- **Place the nails of corresponding fingers back-to-back** and look for a visible gap between the nail beds – **Schamroth's window sign**.
- **Place your thumbs under the pulp of the distal phalanx** and use your index fingers alternately to see if you can feel movement of the nail on the nail bed. This is **fluctuation**.



**Fig. 3.9** Examining for finger clubbing. **A** Assessing interphalangeal depth at (1) interphalangeal joint and (2) nail bed, and nail-bed angle (3). **B** Schamroth's window sign. **C** Assessing nail-bed fluctuation.

### Abnormal findings:

#### Finger clubbing is present if:

- The interphalangeal depth **ratio is >1** (that is, the digit is thicker at the level of the nail bed than the level of the distal interphalangeal joint).
- The nail bed angle is **>190°**.
- Schamroth's window sign is **absent**.
- **Increased nail bed fluctuation** may be present, but its presence is subjective and less discriminatory than the above features.

### Complexion (Skin color)

- In everyday practice the **skin can provide insights into present and past medical disorders** as well as **information about the patients social or mental status**.
- The skin should be exposed where **appropriate and inspected carefully** for **any abnormalities of pigmentation**.
- Skin color is determined by **pigments** in the **skin-melanin** an **endogenous brown pigment** and **carotene** an **exogenous yellow pigment** (mainly derived from ingestion of carrots and other vegetables)-as well as **by the amount of oxyhemoglobin (red)** and **deoxyhemoglobin (blue)** circulating in the dermis.



## 1. Melanin

### A- (hypopigmentation)

#### Underproduction

Vitiligo (patchy depigmentation)

Autoimmune destruction of melanocytes

Albinism

Genetic deficiency of tyrosinase

Hypopituitarism

Reduced pituitary secretion of melanotrophic peptides, growth hormone and sex steroids

### Vitiligo

- This chronic condition produces **bilateral symmetrical depigmentation**, commonly of the face, neck and extensor aspects of the limbs, resulting in **irregular pale patches of skin**.
- It is associated with autoimmune diseases, e.g., diabetes mellitus, thyroid and adrenal disorders, and pernicious anemia.



Fig. 3.7 Vitiligo.

### B- Hyperpigmentation:

- Can be due to excess of the pituitary hormone adrenocorticotrophic hormone (ACTH). As in **adrenal insufficiency** (or the very rare condition **Nelson syndrome**, in which there is ACTH overproduction following **bilateral adrenalectomy for pituitary Cushing's disease**).
- **Pregnancy and oral contraceptives** may also cause blotchy **hyperpigmentation on the face**, known as **chloasma** and pregnancy may increase **pigmentation of the areolae, axillae, genital skin** and **Linea alba** (producing a dark line in the midline of the lower abdomen called a ("Linea nigra").

#### Overproduction

Adrenal insufficiency (Addison's disease)

Increased pituitary secretion of melanotrophic peptides

Nelson's syndrome (may occur after bilateral adrenalectomy for Cushing's disease)

Increased pituitary secretion of melanotrophic peptides

Cushing's syndrome due to ectopic adrenocorticotrophic hormone secretion by tumours, e.g. small cell lung cancer

Ectopic release of melanotrophic peptides by dysregulated tumour cells

Pregnancy and oral contraceptives

Increased levels of sex hormones

Haemochromatosis

Iron deposition and stimulation of melanocytes



### 2. Iron (Haemochromatosis)

- Increases skin pigmentation due to iron deposition and increased melanin production.
- Iron deposition in the pancreas causes **diabetes mellitus** and the combination with skin pigmentation is called 'bronzed diabetes'.



Fig. 3.9 Haemochromatosis with increased skin pigmentation.



### 3. Hemosiderin

- A hemoglobin breakdown product, is deposited in the skin of the lower legs following **extravasation of blood into subcutaneous tissues from venous insufficiency**.
- **Local deposition of hemosiderin (erythema ab igne or 'granny's tartan')** occurs with heat damage to the skin from **sitting too close to a fire or from applying local heat, such as a hot water bottle, to the site of pain**.



10 Erythema ab igne.

### 4. Easy bruising:

Easy bruising can be a reflection of skin and connective tissue fragility due to **advancing age or glucocorticoid usage** or a more serious **coagulopathy**.

### 5. Carotene:

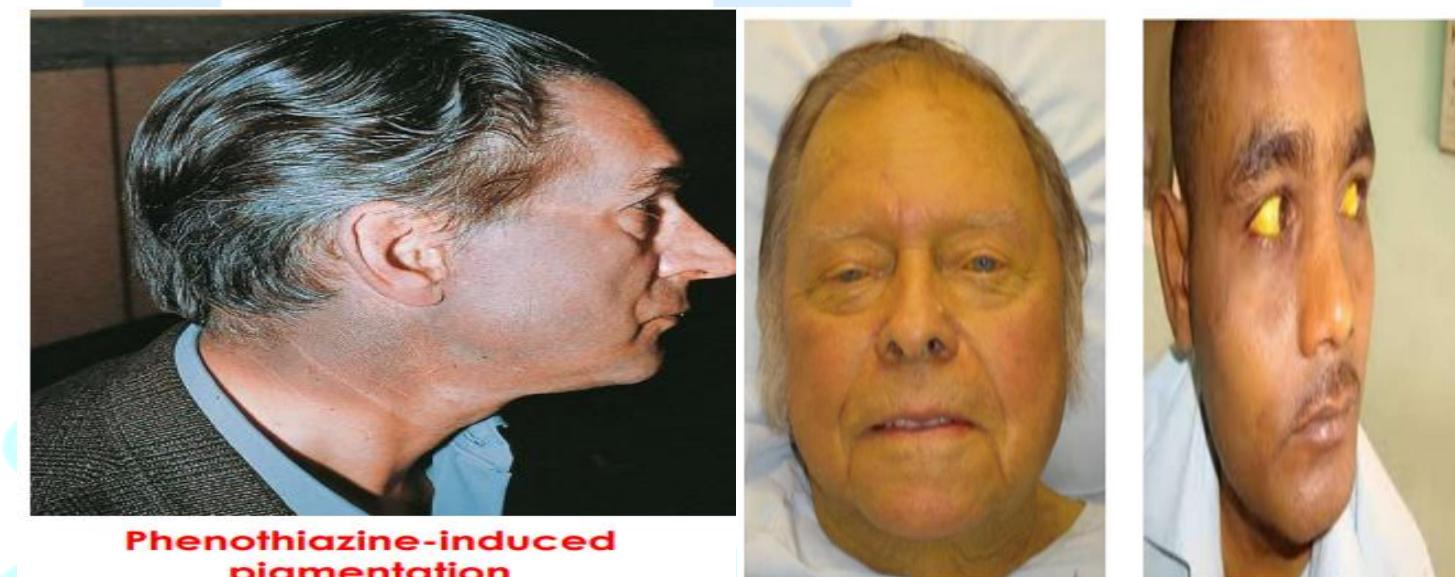
- **Hypercarotenaemia** occurs in people who eat **large amounts of raw carrots and tomatoes**, and in **hypothyroidism**.
- A **yellowish discolouration** is seen on the **face, palms and soles**, but **not the sclerae**, and this distinguishes it from **jaundice**.



Fig. 3.13 Hypercarotenaemia. A control normal hand is shown on the right for comparison.

### 6. Discoloration:

- Skin discolouration can also occur due to **abnormal pigments** such as the **sallow yellow-brownish tinge** in **chronic kidney disease**.
- A **bluish tinge** is produced by **abnormal haemoglobins**, such as **sulphaemoglobin** or **methaemoglobin** (see the section on cyanosis later), or by **drugs such as dapsone**.
- Some **drug metabolites** cause striking **abnormal coloration of the skin** particularly in areas exposed to light, e.g. **mepacrine (yellow)**, **amiodarone (bluish grey)** and **phenothiazines (slate grey)**.



Phenothiazine-induced pigmentation

#### A. Jaundice

- Jaundice is an **abnormal yellow discolouration of the skin, sclera and mucous membranes**.
- It is usually **detectable when serum bilirubin concentration rises**



above 50  $\mu\text{mol/L}$  (3 mg/dL) as a result of parenchymal liver disease, biliary obstruction or hemolysis.

### B. Pallor:

- Pallor can result from **anaemia** in which there is **a reduction in circulating oxyhaemoglobin** in the dermal and subconjunctival capillaries or **from vasoconstriction due to cold exposure or sympathetic activation**.
- The best sites** to assess for the pallor of anaemia **are the conjunctiva** specifically the anterior rim.
- In significant iron deficiency anaemia there may be additional finding of **angular stomatitis, glossitis, koilonychia (spoon-shaped nails)** and **blue sclerae**.



Fig. 3.15 Conjunctival pallor.



Fig. 3.16 Smooth red tongue (glossitis) and angular stomatitis of iron deficiency.

### C. Facial plethora:

- Vasodilatation and flushing may produce a pink complexion even in anemia** and may be due to fever, heat, exercise, food, drugs and other neurological or hormonal disturbances.
- Facial plethora is caused by raised haemoglobin concentration with elevated haematocrit (polycythaemia)**. It may be **primary** or may indicate an **underlying disease (secondary)** resulting in chronic hypoxia or excess erythropoietin production.
- Plethora of the head and neck only may indicate superior vena cava obstruction.**



Fig. 3.17 Flushing due to carcinoid syndrome. [A] Acute carcinoid flush. [B] Chronic telangiectasia.

### 3.6 Conditions associated with facial flushing

#### Physiological

- Fever
- Exercise
- Heat exposure
- Emotional

Drugs (e.g. glyceryl trinitrate, calcium channel blockers, nicotinic acid)

#### Anaphylaxis

#### Endocrine

- Menopause
- Androgen deficiency (in men)
- Carcinoid syndrome
- Medullary thyroid cancer

#### Others

- Serotonin syndrome
- Food/alcohol ingestion
- Neurological (e.g. Frey's syndrome)
- Rosacea
- Mastocytoses



**D. Cyanosis:** Is a **blue discoloration of the skin and mucous membranes** that occurs **when the absolute concentration of deoxygenated hemoglobin is increased.**

#### 1- Central cyanosis:

- Can be seen in the **lips, tongue and buccal or sublingual mucosa**, and can **accompany any disease** (usually cardiac or respiratory) that results in **hypoxia** sufficient to raise the capillary deoxyhemoglobin **concentration above 50 g/L (5 g/dL).**
- Since the detection of cyanosis relies on the presence of an absolute concentration of deoxyhemoglobin, **it may be absent in anemic or hypovolemic patients despite the presence of hypoxia.** Conversely, cyanosis may manifest at relatively **mild levels of hypoxia in polycythemia patients.**

#### 2- Peripheral cyanosis

- This occurs **in the hands, feet or ears, usually when they are cold.**
- When prolonged peripheral capillary flow allows greater oxygen extraction and hence increased levels of deoxyhemoglobin. **As the patient is warmed and the circulation improves, so does the cyanosis.**
- Pathological causes of peripheral cyanosis include low cardiac output states, arterial disease and venous stasis or obstruction.

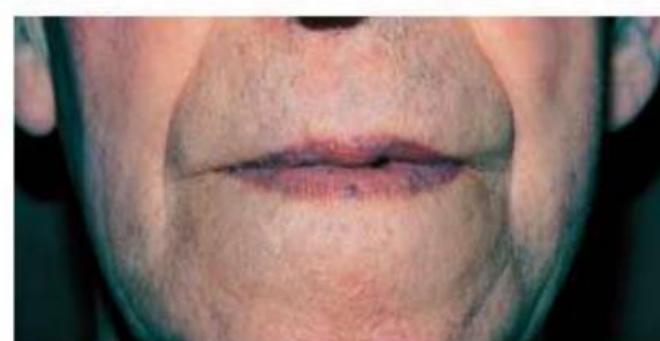


Fig. 3.18 Central cyanosis of the lips.

#### Characteristic skin changes:

- Characteristic skin changes also occur in other conditions such as **scurvy** (Fig.3.19).
- **Neurofibromatosis** (Fig. 3.20).
- **Acanthosis nigricans** (see Fig. 10.15A).



Fig. 3.19 Scurvy. (A) Bleeding gums. (B) Bruising and perifollicular haemorrhages.



Fig. 3.20 Neurofibromatosis.



#### The tongue:

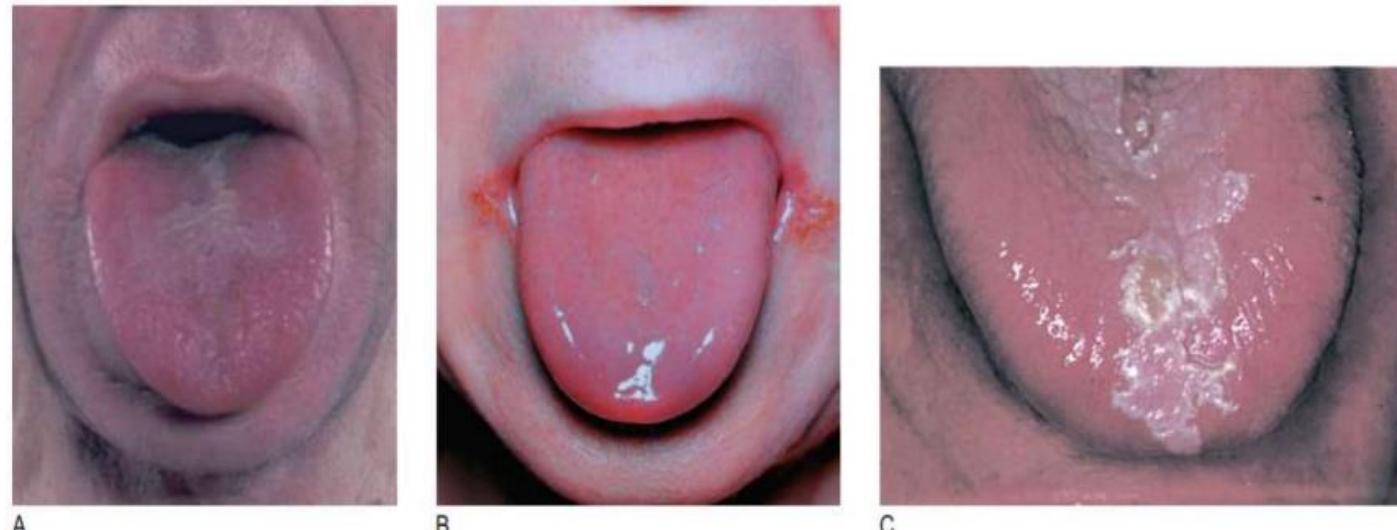
- In addition to revealing central cyanosis, examination may uncover:
  - 1- The **smooth tongue of iron deficiency.**
  - 2- **Enlargement in acromegaly.**
  - 3- **Wasting and fasciculation in motor neuron disease.**
  - 4- **White patches that may be scraped off the tongue are due to the fungal yeast, Candida (oral thrush).**

Common causes include inhaled steroids, immune deficiency, e.g., HIV and terminal illness.



5- **Glossitis** is a smooth reddened tongue due to atrophy of the papillae. It is common in **alcoholics**, in **nutritional deficiencies** of iron, folate and **vitamin B12**, and in 30% of patients with **coeliac disease**.

6- **Leukoplakia** is a thickened white patch that cannot be scraped off the tongue. It may be **premalignant**.



**Fig. 3.19** The tongue as a diagnostic aid. (A) Large tongue (macroglossia) of acromegaly. (B) Smooth red tongue and angular stomatitis of iron deficiency. (C) Leukoplakia.

### Body Odor:

- Odors' can provide clues to a patient's **social or behavioral habits**; the **smell of alcohol, tobacco or cannabis may be readily apparent**. **Stale urine and anaerobic skin infections** also produce distinctive smells.
- **Halitosis (bad breath)** can be due to **poor dental hygiene, gingivitis, stomatitis, atrophic rhinitis, tumors of the nasal passages or suppurative lung conditions** such as **lung abscess or bronchiectasis**.

Other characteristic odors include:

- **Fetor hepaticus**: stale 'mousy' smell of the **volatile amine, dimethylsulphide**, in patients with **liver failure**.
- **Ketones**: a sweet smell (like nail varnish remover) due to **acetone** in **diabetic ketoacidosis or starvation**.

- **Uremic fetor**: **fishy or ammoniacal smell** on the breath in **uremia**.
- **Putrid or fetid smell** of chronic **anaerobic suppuration** due to **bronchiectasis or lung abscess**.
- **Foul smelling belching** in patients with **gastric outlet obstruction**.
- **Strong fecal smell** in patients with **gastrocolic fistula**.

