Oedema

BY

DR.HAZEM SAMY

LECTURER OF INTERNAL MEDICINE

Definition

- tissue swelling due to an increase in interstitial fluid.

Pathophysiology

- The capillary wall separates the interstitial fluid and plasma compartments.
- The distribution of water between the vascular and interstitial spaces is determined by the balance between hydrostatic pressure forcing water out of the capillary, and colloid osmotic (oncotic) pressure, drawing fluid into the vascular space (Starling's forces). Oncotic pressure depends largely on circulating protein concentration, particularly serum albumin.

Pathophysiology

- Oedema can be generalised, localised or postural.
- The cardinal sign of subcutaneous oedema is pitting of superficial tissues. Pitting on pressure may not be demonstrable until body weight has increased by 10–15%.
- Day-to-day alterations in body weight are usually the most reliable index of changes in body water.
- Hypothyroidism is characterised by mucinous infiltration of tissues (myxoedema). In contrast to oedema, myxoedema and chronic lymphoedema do not pit on pressure.

Generalised oedema

Etiology

- There are two principal causes of generalised oedema:
- ✓ fluid overload
- ✓ hypoproteinaemia.

- Distinguish them by assessing the jugular venous pressure
- The jugular venous pressure is usually elevated in fluid overload but not in hypoproteinaemia

Etiology

Low plasma oncotic pressure
Low serum albumin due to: • Increased loss – nephrotic syndrome • Decreased synthesis – chronic liver disease • Malabsorption – protein-losing enteropathy, e.g. Crohn's disease and coeliac disease • Malnutrition – kwashiorkor

Etiology

Increased hydrostatic pressure
High venous pressure/obstruction due to: • Deep vein thrombosis • Venous insufficiency • Pregnancy • Pelvic tumour • Heart failure • Intravascular volume expansion, e.g. excess intravenous fluid, renal failure

Localised oedema

Etiology

- may be caused by venous, lymphatic, inflammatory or allergic disorders.

Etiology

- ***Venous causes***
- Increased venous pressure increases hydrostatic pressure within capillaries, producing oedema in the area drained by that vein.
- Venous causes include **deep vein thrombosis**, **external pressure** from a tumour or pregnancy, or **venous valvular incompetence** from previous thrombosis or surgery. Conditions which **impair the normal muscle pumping action**, e.g. hemiparesis and forced immobility, increase venous pressure by impairing venous return. As a result oedema may occur in immobile, bed-ridden patients, in a paralysed limb, or in a healthy person sitting for long periods, e.g. duringtravel

Etiology

- ***Lymphatic causes:***
- Normally, interstitial fluid returns to the central circulation via the lymphatic system.
- Any cause of impaired lymphatic flow, e.g. intraluminal or extraluminal
- obstruction, may produce localised oedema (lymphoedema) .
- If the condition persists, fibrous tissues proliferate in the interstitial space and the affected area becomes hard and no longer pits on pressure.
-

Etiology

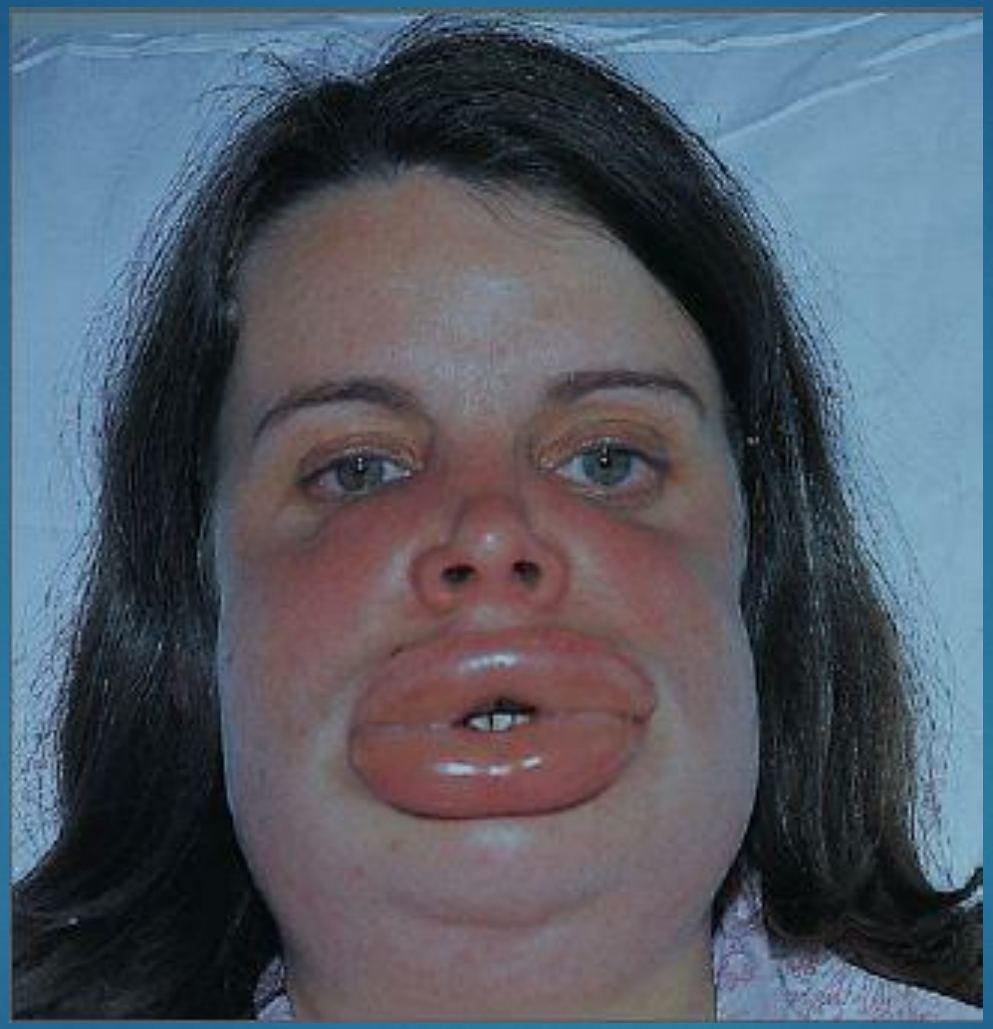
- ***Lymphatic causes(cont.):***
- the commonest cause of leg lymphoedema is congenital hypoplasia of leg lymphatics (Milroy's disease),
- and in the arm after radical mastectomy and/ or irradiation for breast cancer.
- Lymphoedema is common in some tropical countries because of lymphatic obstruction by filarial worms (elephantiasis).

Etiology

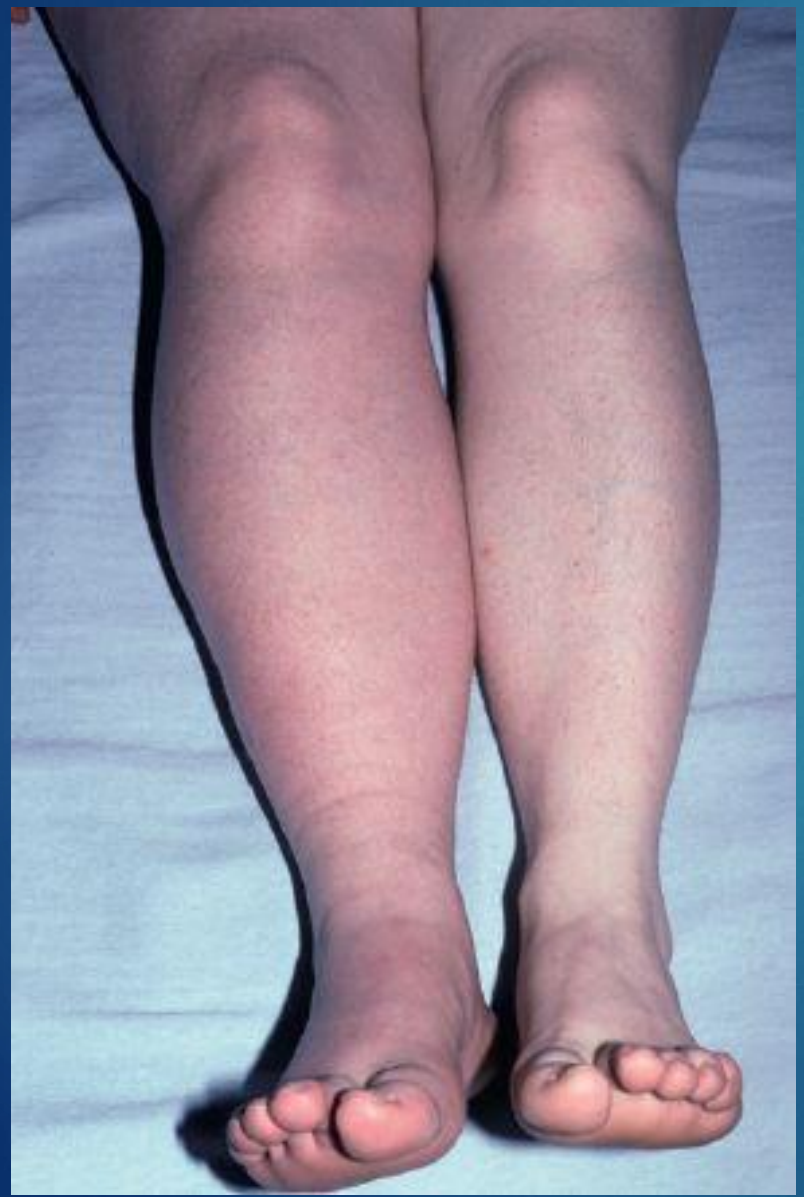
- ***Inflammatory causes:***
- Any cause of tissue inflammation, including infection or injury, liberates mediators, e.g. histamine, bradykinin and cytokines, which cause vasodilatation and increase capillary permeability.
- accompanied by the other features of inflammation (redness, tenderness and warmth) and is therefore painful.

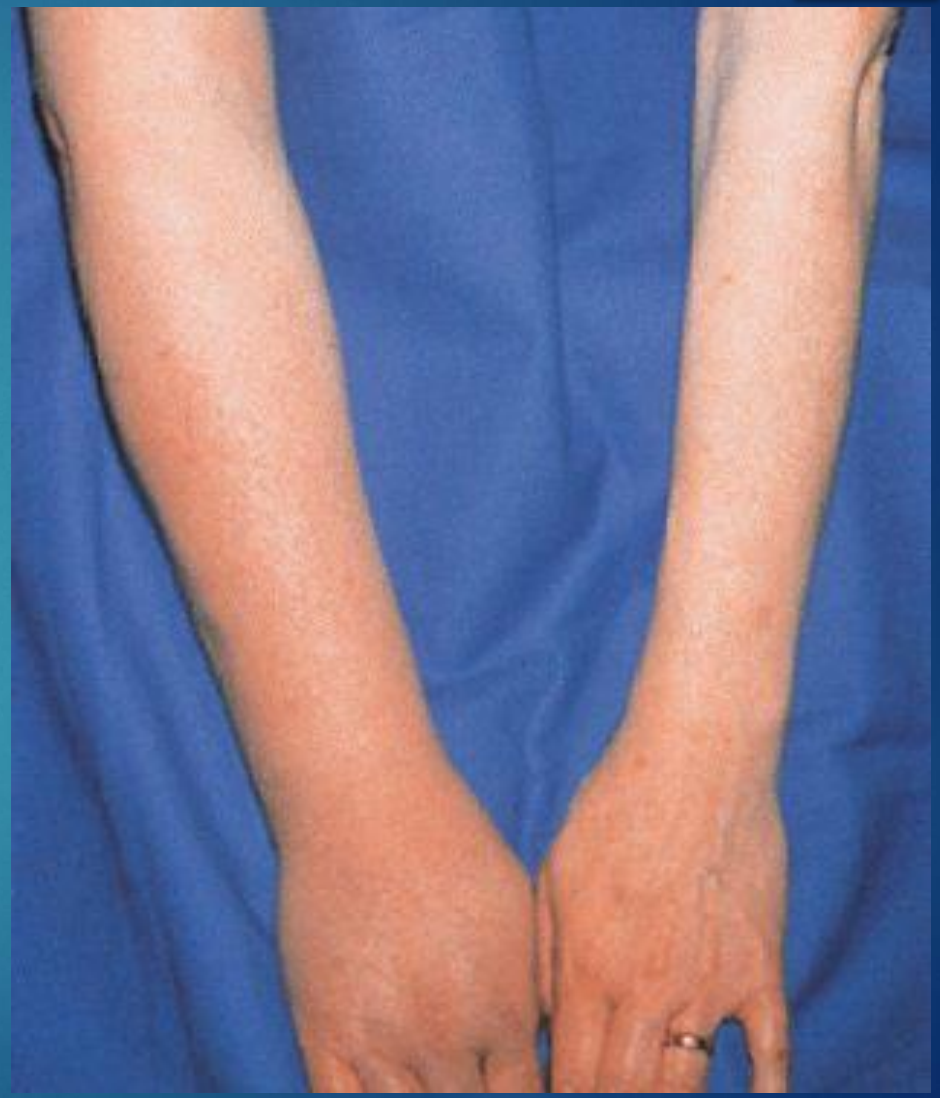
Etiology

- ***Allergic causes:***
- Increased capillary permeability occurs in acute allergic conditions.
- The affected area is usually red and pruritic (itchy) because of local release of histamine and other inflammatory mediators but, in contrast to inflammation, is not painful.
- **Angio-oedema** is a specific form of allergic oedema affecting the face, lips and mouth. Swelling may develop rapidly and may be life-threatening if the upper airway is involved.



Angioneurotic oedema





Postural oedema

- This is due to failure of muscle movement and is commonin the lower limbs of inactive patients.

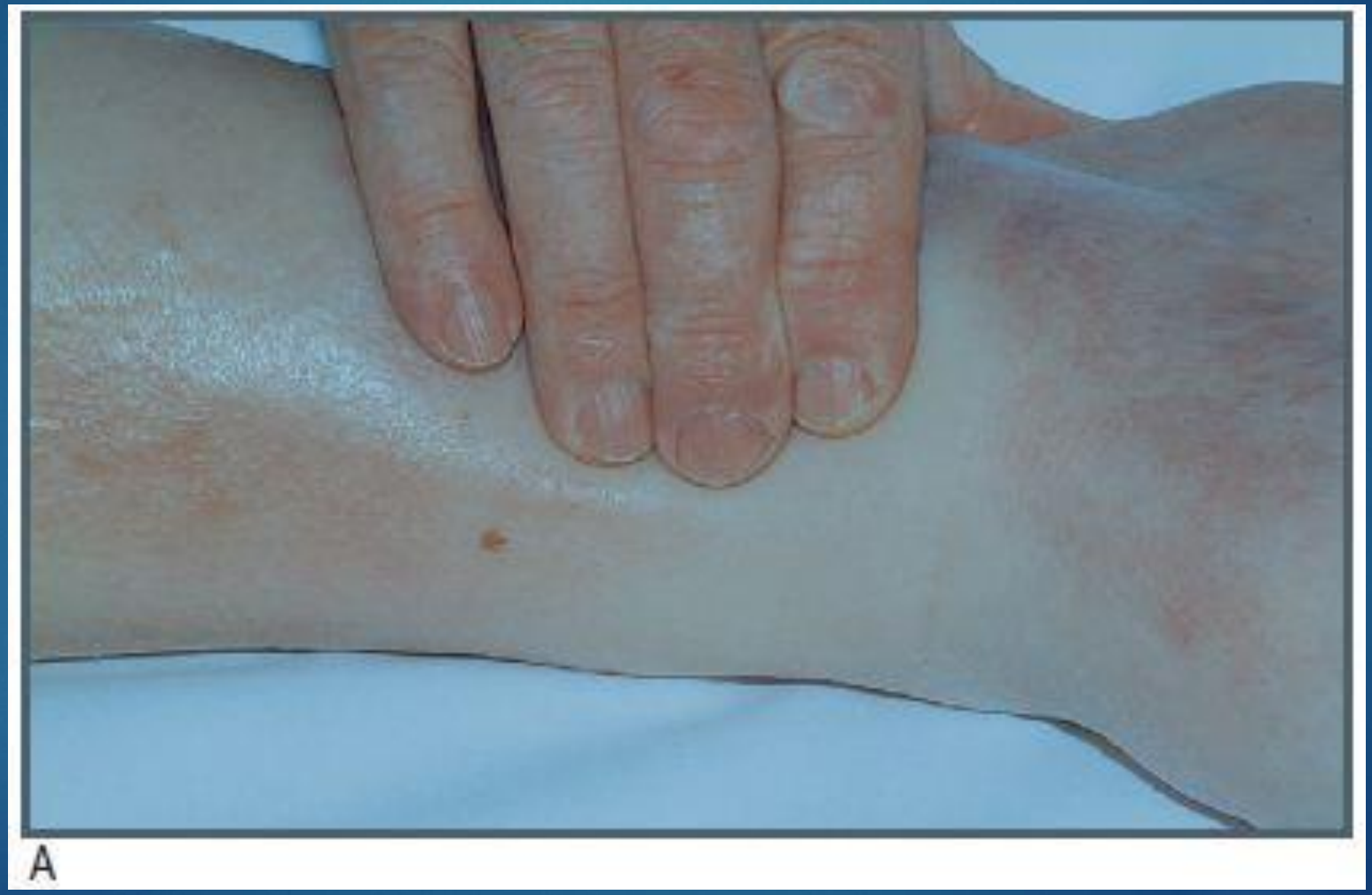
Examination sequence

- Apply **firm pressure** with your fingers or thumb for at least 15 seconds. Pitting may persist for several minutes until it is obliterated by the slow return of the displaced fluid.
- Assess the **state of hydration** by looking for sunken orbits and dry mucous membranes. Gently pinch a fold of skin on the neck or anterior chest wall, hold it for a few seconds and then release. Well-hydrated skin springs back into position immediately, in severe dehydration skin subsides abnormally slowly.

Examination sequence

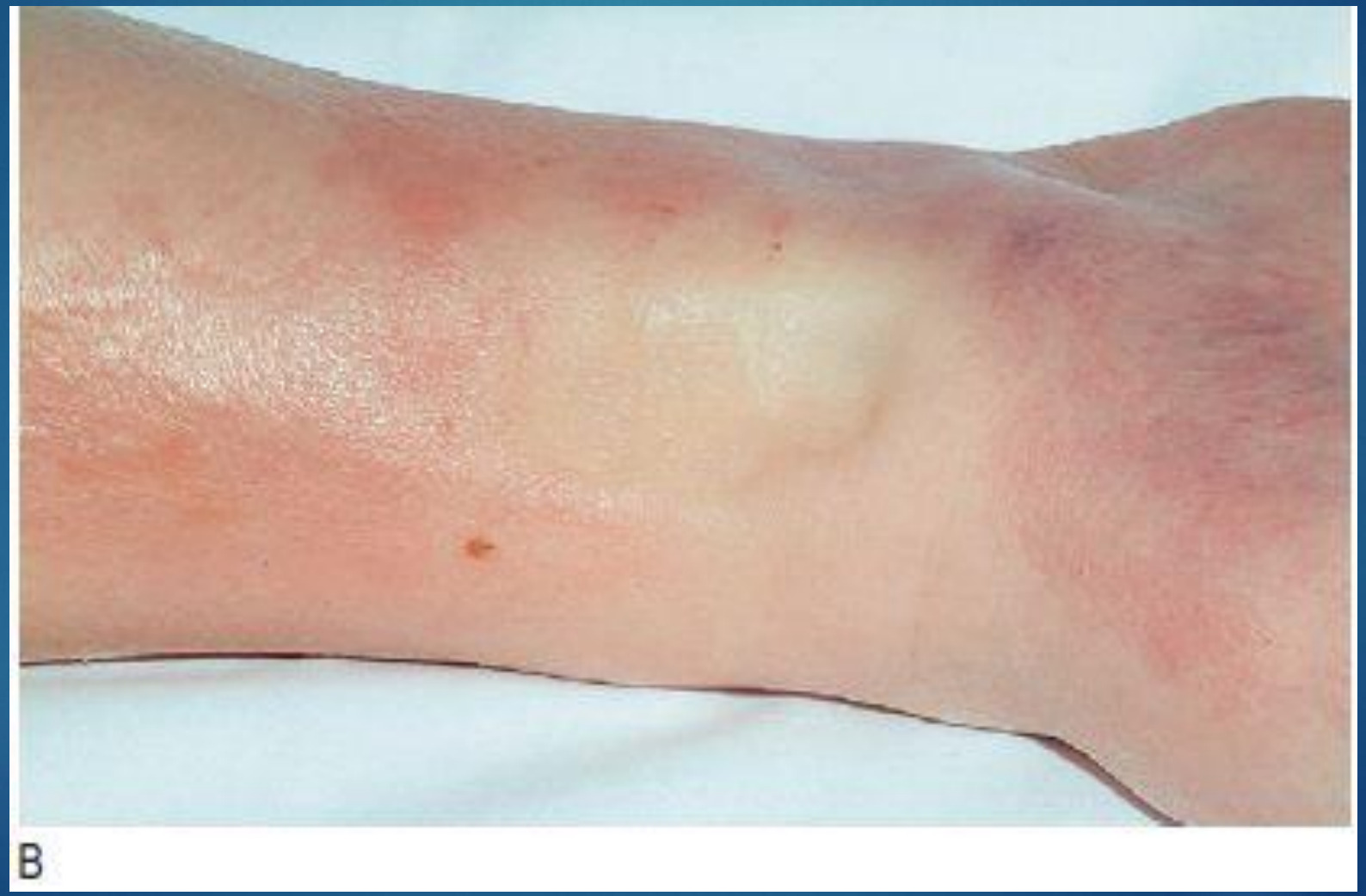
- Record weight and urine output.
- Record the pulse rate and supine/erect blood pressures. Look
- for tachycardia >100 bpm and postural hypotension (a fall >15 mmHg in systolic pressure on standing).
- Check for oedema in the ankles and legs. In bed-bound patients, check for sacral oedema.
- Examine the jugular venous pressure.

Examination sequence

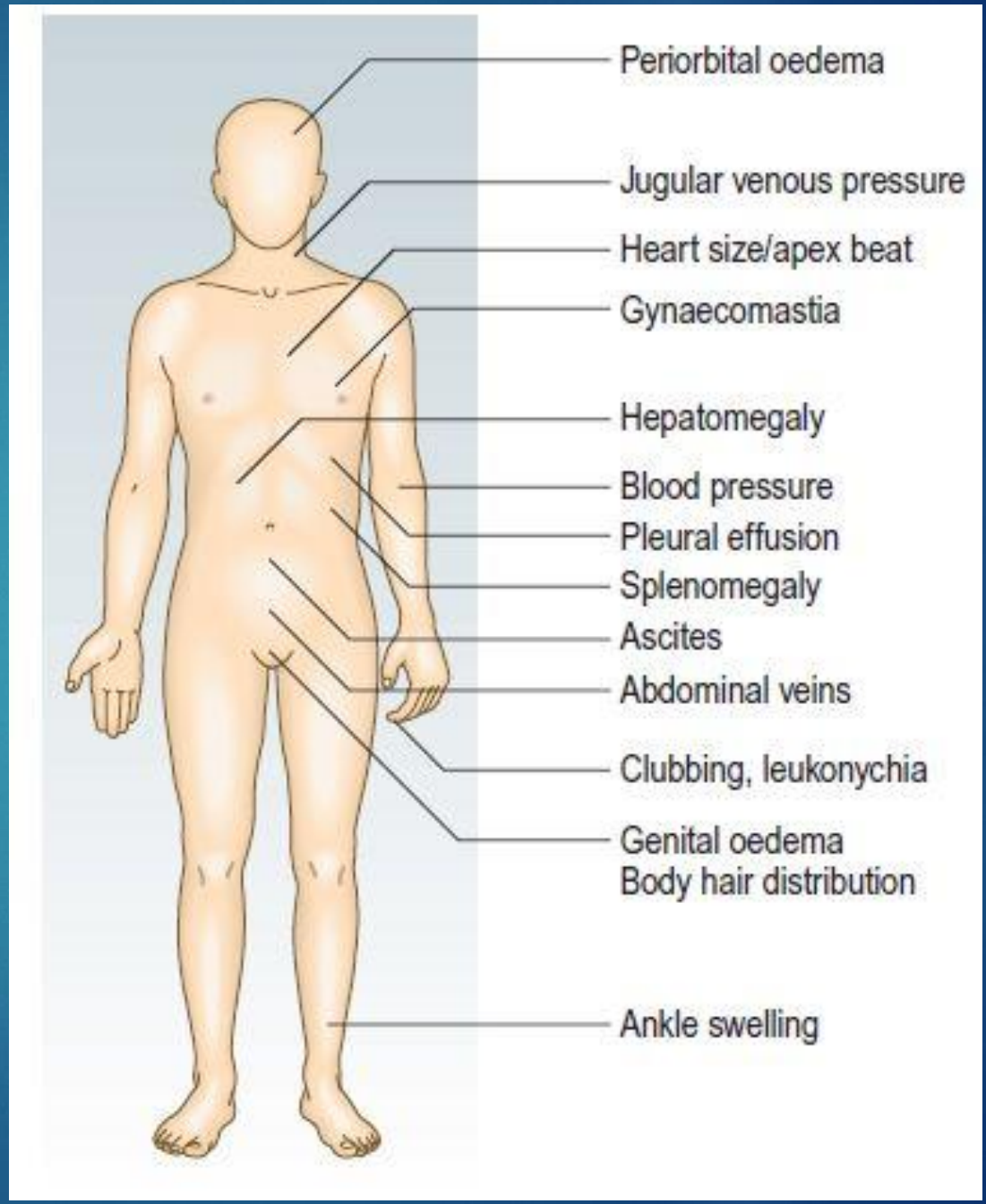


A

Examination sequence



Features to look for in oedema




Thank You