Thromboembolic

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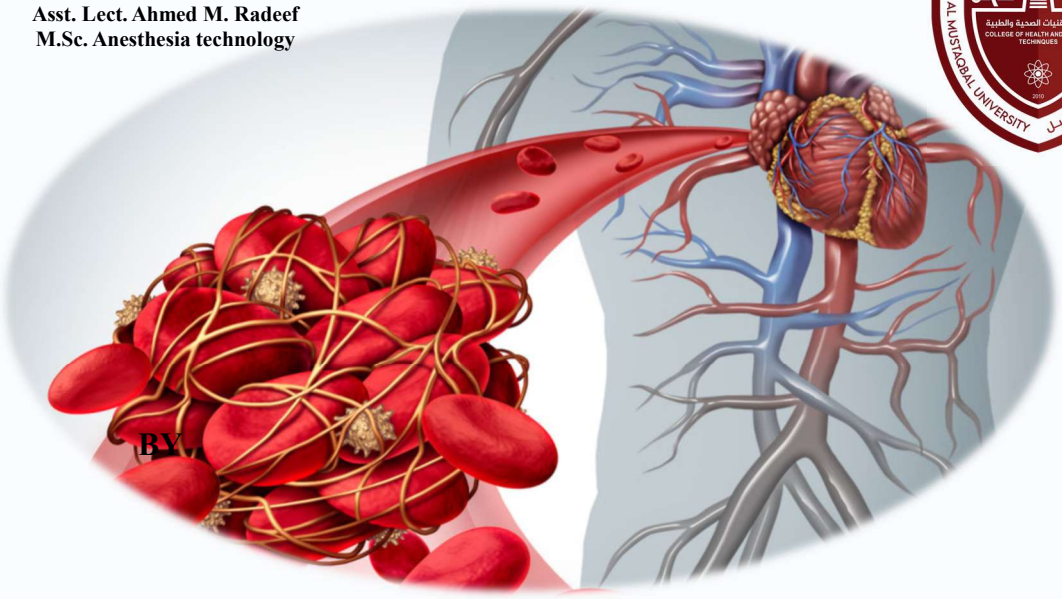


Introduction

Pulmonary embolism (PE) is a commonly considered but relatively uncommonly diagnosed condition in hospitalized patients. It is important to have an adequate understanding of the pathophysiology as well as a rapid and reliable strategy for investigation and management. This is significantly important in intensive care unit (ICU) patients where diagnosis can be difficult and PE may be life threatening. While admission rates for PE are rising, outcomes are improving overall. However, mortality amongst haemodynamically unstable patients remains high.

Early deaths in PE are usually the result of **acute right ventricular (RV) failure and cardiogenic shock**.

Etiology

Deep venous thrombosis (DVT) and PE are components of a single disease termed **venous thromboembolism (VTE).** Embolization of DVT to the pulmonary arteries leads to PE. The incidence of VTE in the population is about 1 in 1000 per year and is more common both with **advancing age and in males**.

Predisposing risk factors for VTE involve one or more components of **Virchow triad**:

I. **Venous stasis**
II. **Vein wall injury**
III. **Hypercoagulability of blood.**

In addition to the main factors such as immobility (from any cause), surgery, trauma, malignancy, pregnancy, thrombophilia and autoimmune disorders have also been associated with increased risk of PE.

Etiology

Factor V Leiden mutation, oral contraceptive use, pregnancy, pueperium, obesity and minor leg injuries pose a higher risk of **DVT as compared to PE**, whereas pulmonary conditions like chronic obstructive pulmonary disease, sickle cell disease and pneumonia raise the risk of PE with little effect on risk of DVT.

Most PE results from DVT in the lower limbs, pelvic veins or inferior vena cava (IVC), although thrombi can develop in the right atrium, right ventricle and upper limbs. Up to **40%** of patients with DVT develop PE, although if the DVT is isolated to below the knee, then clinically obvious PE is rare. Between **2% and 4%** of patients with VTE develop chronic pulmonary hypertension.

Pathophysiology

The effects of PE range from being incidental and clinically irrelevant to causing severe obstruction to the pulmonary circulation and sudden death. Pulmonary arterial obstruction and the subsequent release of vasoactive substances such as serotonin and thromboxane A2 from platelets lead to elevated **pulmonary vascular resistance and acute pulmonary hypertension**.

Acute pulmonary hypertension **increases RV afterload and RV wall tension** which leads to **RV dilatation and dysfunction with coronary ischaemia being a major contributing mechanism**. In massive PE, the combination of **coronary ischaemia**, **RV systolic failure, paradoxical interventricular septal shift and pericardial constraint** leads to **left ventricular (LV) dysfunction and obstructive shock**. In patients with underlying cardiorespiratory disease, a small PE can have profound consequences.

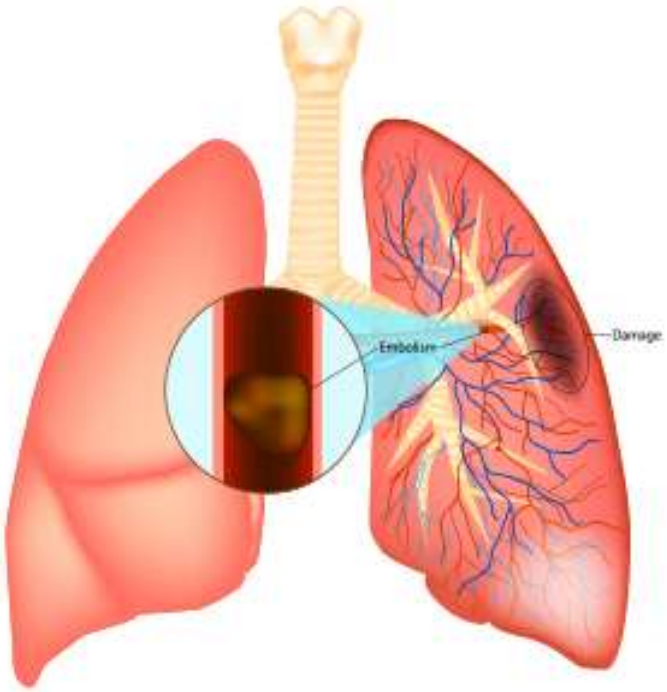
Pathophysiology

Pulmonary arterial obstruction causes a **mismatch** between lung ventilation and perfusion which leads to **hypoxaemia**. The ventilation of lung units that have reduced or no perfusion causes **increased dead-space** ventilation and an increase in the **end-tidal to arterial CO2 gradient**. **Alveolar hyperventilation also occurs leading to hypocapnia**.

Increased right atrial pressure can open a patent foramen ovale, which may result in right-to-left **shunting manifested as either refractory hypoxaemia or paradoxical (arterial) embolisation commonly to the brain leading to cerebral infarction.**

Intensive care unit (ICU) patients have a greater risk of thrombotic. The diagnosis and management of deep vein thrombosis (DVT) and pulmonary embolism (PE) in critically ill patients are challenging and these conditions are associated with high mortality.

According to research, half of the hospitalized patients are at a risk of thromboembolism. The rate of VTE ranges from 10% to 80% percent in patients, who are not being given any prophylaxis



Symptoms

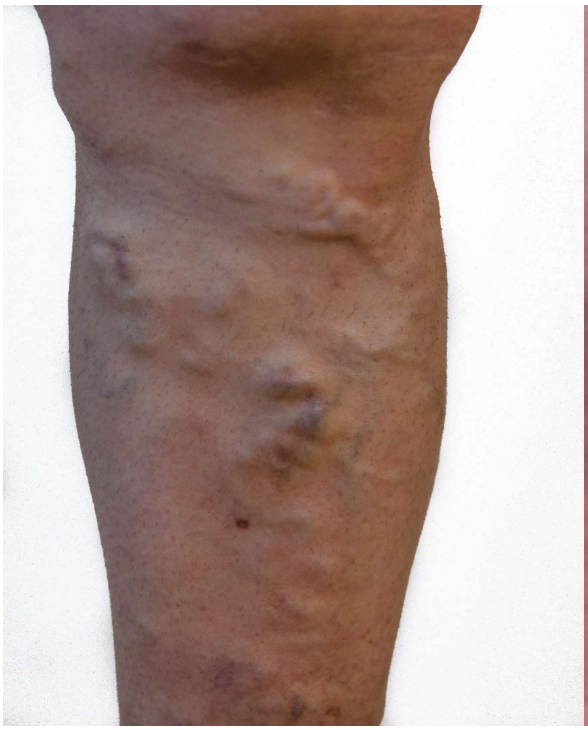
Dyspnoea, pleuritic chest pain, and haemoptysis are the classic symptoms of PE. Most patients will have at least one of these symptoms, with dyspnoea being the most common. The combination of pleuritic chest pain and haemoptysis reflects a late presentation where pulmonary infarction has occurred. If syncope occurs, and there is no other obvious cause, it is likely that this is a massive PE.

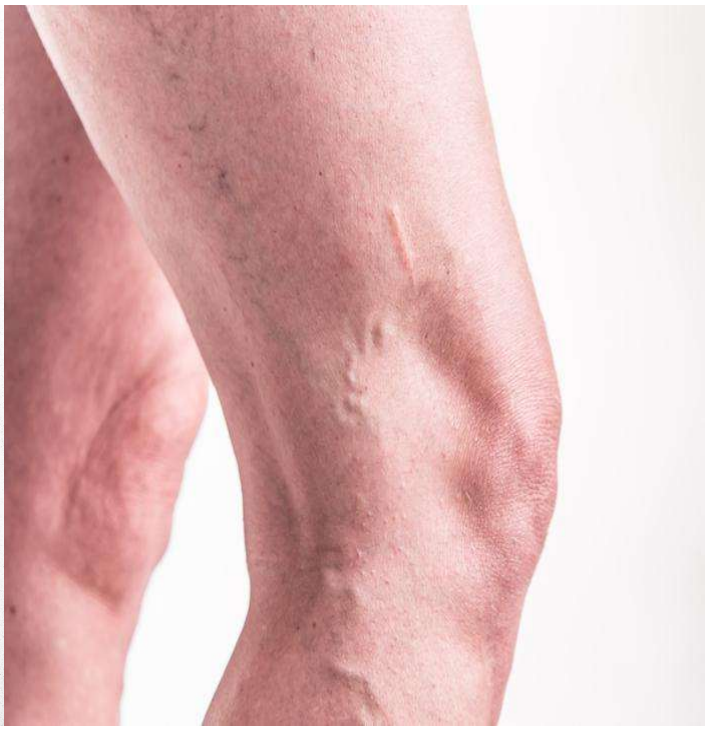
Physical signs

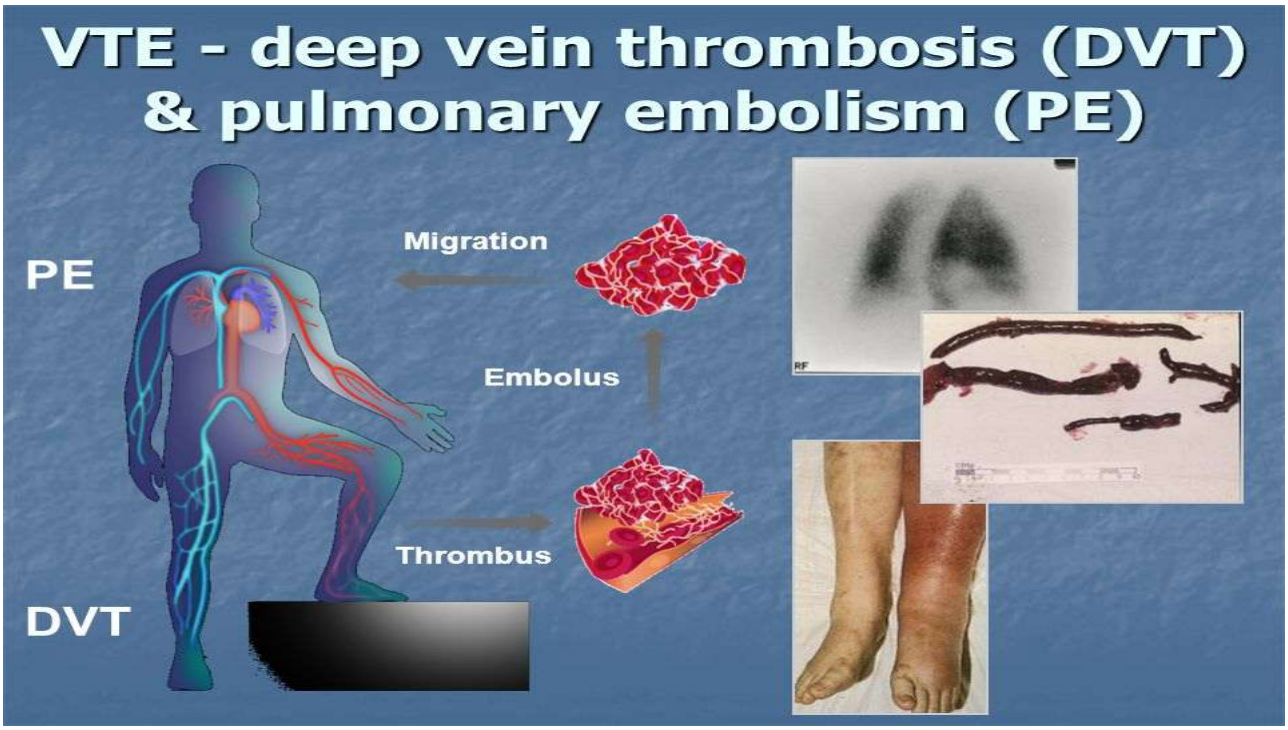
Physical signs can be absent, but the most frequent sign is tachypnoea. Others include tachycardia, fever and **signs of RV dysfunction (raised jugular venous pressure, parasternal heave and loud pulmonary component of the second heart sound)**. In massive PE, signs may include hypotension, pale mottled skin and peripheral or even central cyanosis. It is important to examine for signs of DVT particularly in the legs.

Diagnosis

- Doppler venous ultrasound is the best imaging modality for diagnosis of DVT. Others like contrast venography and magnetic resonance venography are also being use, but they have limitations
- The diagnosis of pulmonary embolism (PE): computed tomography pulmonary angiography (CTPA) is the gold standard for diagnosing PE






VTE - deep vein thrombosis (DVT)
& pulmonary embolism (PE)
PE
Migration
Embolus
Thrombus
DVT

D-dimer

D-dimer is a substance in the blood that rises when a new blood clot forms.

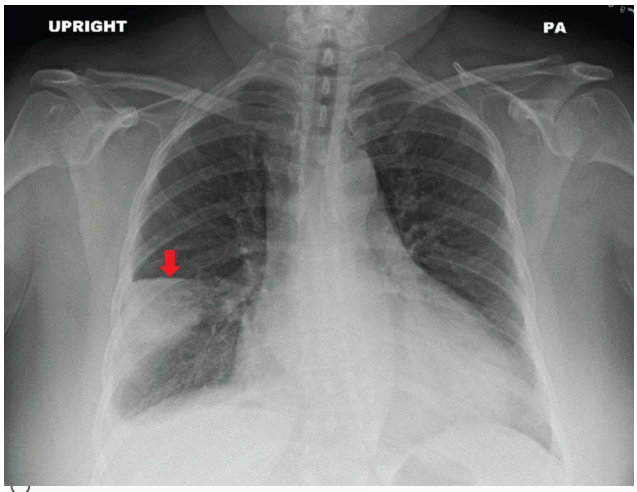
- Because the test is very sensitive, a negative result (normal D-dimer), especially with accurate methods like enzyme-linked immunosorbent assays (ELISA), enzyme-linked immunofluorescence assays (ELFAs) and latex quantitative assays, strongly indicates that there is no deep vein thrombosis (DVT) or pulmonary embolism (PE).
- But if the test is positive, it does not always mean there is a clot, because D-dimer can also be high in many other conditions (infection, inflammation, surgery, pregnancy, etc.).

Arterial Blood Gases (ABG)

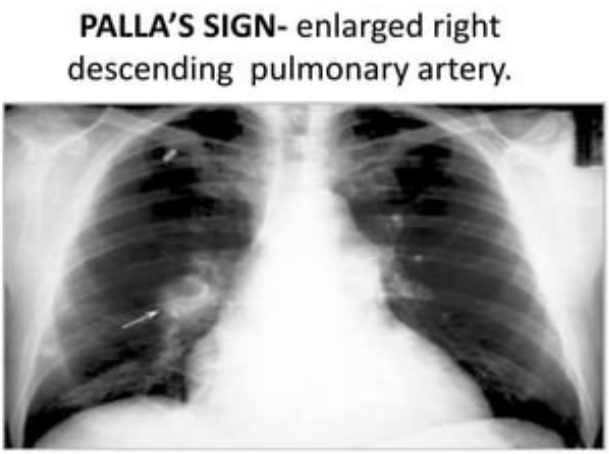
- A **normal arterial blood gas (ABG)** result does **not rule out pulmonary embolism (PE)**.
- In PE, common ABG findings can include:
  - **Low oxygen (hypoxemia)** with a **wide alveolar–arterial (A–a) oxygen gradient**
  - **Low carbon dioxide (hypocapnia)**
  - **High end-tidal CO_2 gradient**
- These changes can raise suspicion of PE, but they are also seen in many other critically ill patients.
- If a **large PE causes shock**, **metabolic acidosis** may appear.
- **Capnography** may become a helpful tool to rule out PE in patients with a **positive D-dimer**, but the best method and cutoff values are still not clear.

CHEST X-RAY

The chest X-ray is often normal or only slightly abnormal with non-specific signs such as cardiac enlargement, pleural effusion, elevated hemidiaphragm, atelectasis and localised infiltrates. More specific findings, including **focal oligaemia**, a peripheral wedge-shaped density above the diaphragm (**Hampton hump**) and an enlarged right descending pulmonary artery (**Palla sign**), are uncommon and may be difficult for non-radiologists to identify. The chest X-ray is also useful in identifying an alternative diagnosis such as pneumothorax, pneumonia, acute pulmonary oedema, rib fracture and pleural effusion.

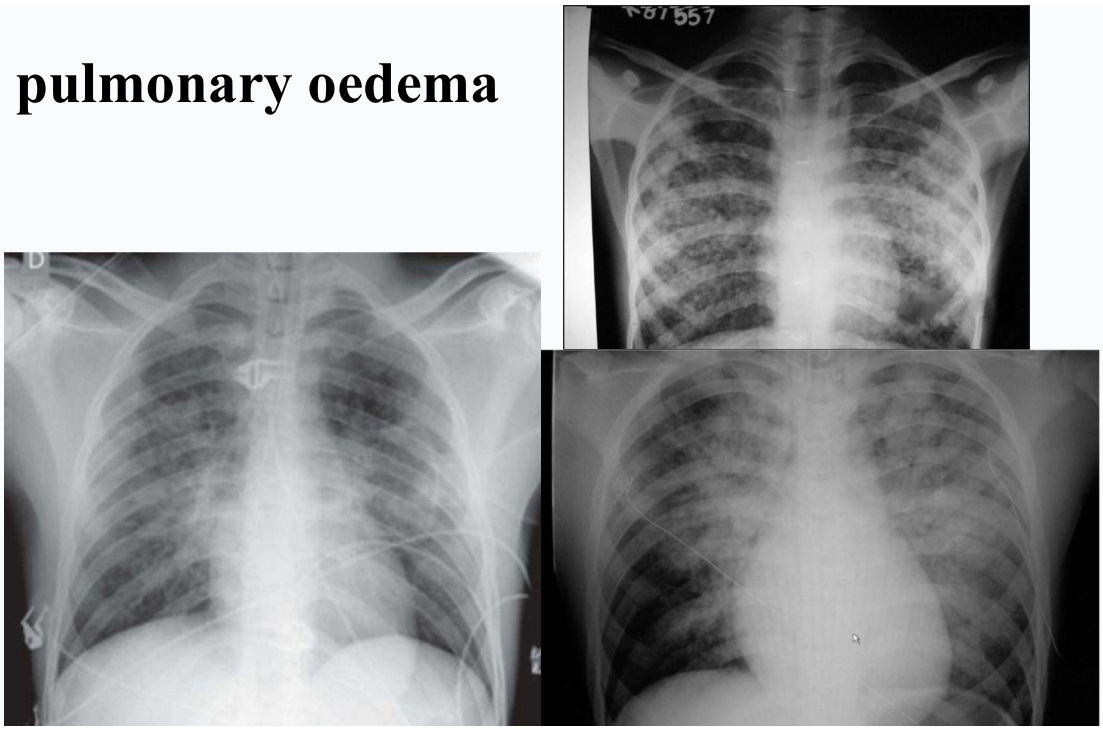


Hampton hump



Palla sign

pulmonary oedema



Management

Management principles

Once pulmonary embolism (PE) is confirmed, patients should receive anticoagulation—either unfractionated heparin, low-molecular-weight heparin (LMWH), or newer oral anticoagulants (NOACs)—to prevent further clots. In more severe PE, treatment focuses on rapid clot removal to relieve right ventricular (RV) dysfunction. Grading PE severity is important for management and prognosis.

Massive pulmonary embolism (haemodynamically unstable)

Patients with PE and hypotension have a high risk of death despite treatment. If cardiopulmonary resuscitation is required the risk is even higher. These patients have the most to benefit from a strategy that includes attempts at urgent embolus destruction (with thrombolytic therapy or embolectomy), concurrent haemodynamic

Submassive pulmonary embolism (haemodynamically unstable with evidence of right ventricular dysfunction)

Patients with PE and evidence of RV dysfunction have higher mortality (around 15%) and recurrence rates than those with normal RV function. They also develop shock and RV thrombi more frequently. These patients require prevention of further embolisation but also warrant strong consideration of embolus destruction using thrombolytic therapy. Thrombolytic therapy in these patients has been shown to reduce haemodynamic decompensation and mortality at the cost of increased risk of intracranial haemorrhage and major extracranial bleeding, especially in elderly patients. Consideration should be given to low-dose thrombolysis.

Mild pulmonary embolism (haemodynamically stable with no right ventricular dysfunction)

Patients with PE who have normal blood pressure, normal RV function (determined by echocardiography or CTPA scan) and non-elevated cardiac biomarkers have a low risk of death or recurrence. The predominant management goal is prevention of further embolisation using anticoagulant therapy, as treatment focused on embolus destruction is unlikely to confer additional benefits.

Summary

In summary, the major principles of management are therefore

- Prevention of further embolisation (for massive, submassive and mild PE)
- Embolus destruction (for massive and submassive PE)
- Concurrent haemodynamic support (for massive PE).

Thromboprophylaxis

Thromboprophylaxis is broadly divided into **primary** and **secondary** forms:

❑**Primary prophylaxis**:

Aimed at preventing the initial occurrence of deep vein thrombosis (DVT).

- **Pharmacological measures**: unfractionated heparin (UFH, Heparin), low-molecular-weight heparin (LMWH, e.g., Enoxaparin/Clexane).
- **Mechanical measures**: graduated compression stockings, intermittent pneumatic compression devices.

❑**Secondary prophylaxis**:

Involves the early detection and prompt management of venous thrombosis to prevent progression or recurrence.

Thromboprophylaxis

The choice of primary prophylaxis depends on several factors, including:

- **Risk of thrombosis**
- **Risk of bleeding**
- **Nature and severity of the underlying illness**

Based on these, patients are classified into **low-, moderate-, or high-risk categories**, and each category has specific recommended prophylactic strategies.

Additionally, the **duration of thromboprophylaxis** varies between patients and is individualized according to their risk classification and clinical condition.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Mobilization and the effects of immobility

Immobility in ICU patients is a **major risk factor for DVT** and contributes to multiple complications.

Key Consequences of Immobility:

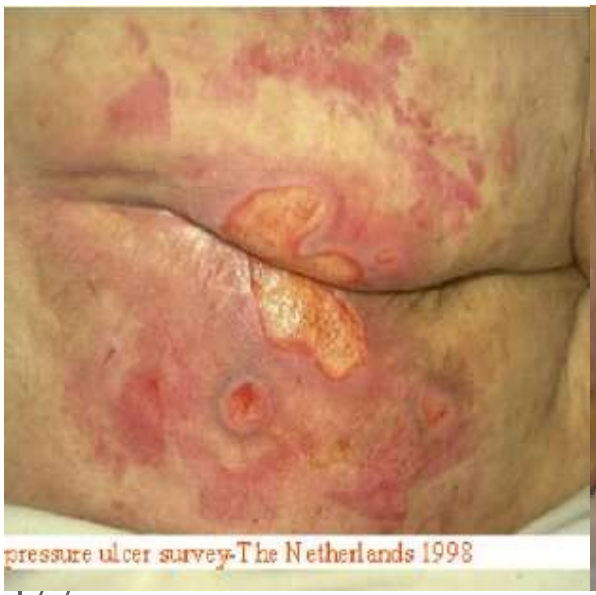
- **ICU-acquired weakness:**
  - Skeletal muscle strength declines rapidly:
    - ❖**1–1.5% loss per day** of strict bed rest
    - ❖**4–5% loss per week** of strict bed rest
  - Neuromuscular weakness develops in **20–25% of ICU patients**

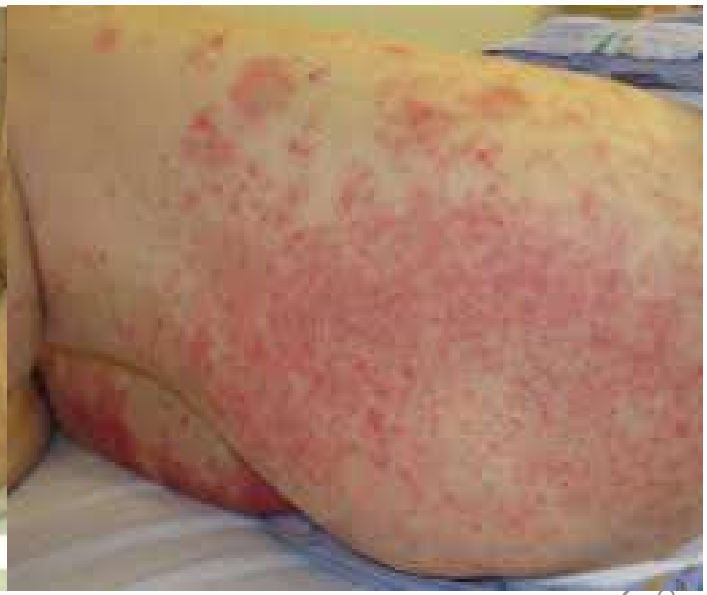
Mobilization and the effects of immobility

- **Prolonged mechanical ventilation:** Weak muscles, especially respiratory muscles, delay weaning.
- **Longer hospital stay:** Due to delayed recovery and complications.
- **Increased mortality:** Higher risk of adverse outcomes.
- **Metabolic effects:** Immobilization reduces insulin-mediated glucose transport, promotes **catabolism and muscle wasting**.
- **Long-term impact:** Functional impairment and weakness can persist **months to years after ICU discharge**.

Sores in ICU

- Pressure ulcers (PrUs) are a common complication in intensive care unit (ICU) patients who are sedated, ventilated, and/or bedridden for long periods.
- In older adults skin becomes weaker and easy breakdown
- Pressure ulcers are formed





Depression and anxiety

- Patients who are critically ill often develop depression and anxiety symptoms during their stay in ICU.
- Delirium is the most prevalent mental disorder among older patients in the ICU associated with…
  - ❑Poor prognosis
  - ❑Increased length of hospital stay

❖Provides psychological support for the…

- ❑ Patient
- ❑ Family members

Benefits of early mobility

- Safe
- Feasible
- Improves patients ICU outcomes
- Discharge patients home earlier
- Decreases neuromuscular weakness
- Decreases bed sores
- Decreases anxiety and depression

Early mobilization in the ICU

Early mobilization refers to a structured, progressive increase in physical activity, starting with passive or active range of motion exercises and advancing to ambulation. It should be initiated within 24–48 hours of ICU admission once hemodynamic and respiratory stability are achieved. Physical activity activates protective pathways, reduces oxidative stress, and promotes the release of anti-inflammatory cytokines.

Early mobilization in the ICU

Risks

- Accidental removal of tubes, lines, or accidental extubation.
- Hemodynamic instability or oxygen desaturation.
- Patient discomfort.
- Challenges due to limited staff or time.

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