BRONCHIECTASIS
LEVEL IV MEDICINE

BY: DR. J. O. MECHA

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INTRODUCTION

- Bronchiectasis and lung abscesses are referred to as suppurative lung diseases

Definition

- Bronchiectasis is a chronic destructive lung disease characterized by:
  - Abnormal and irreversible dilatation of the medium sized bronchi
  - Persistent and variable inflammatory processes producing damage to the bronchial elastic and muscular elements

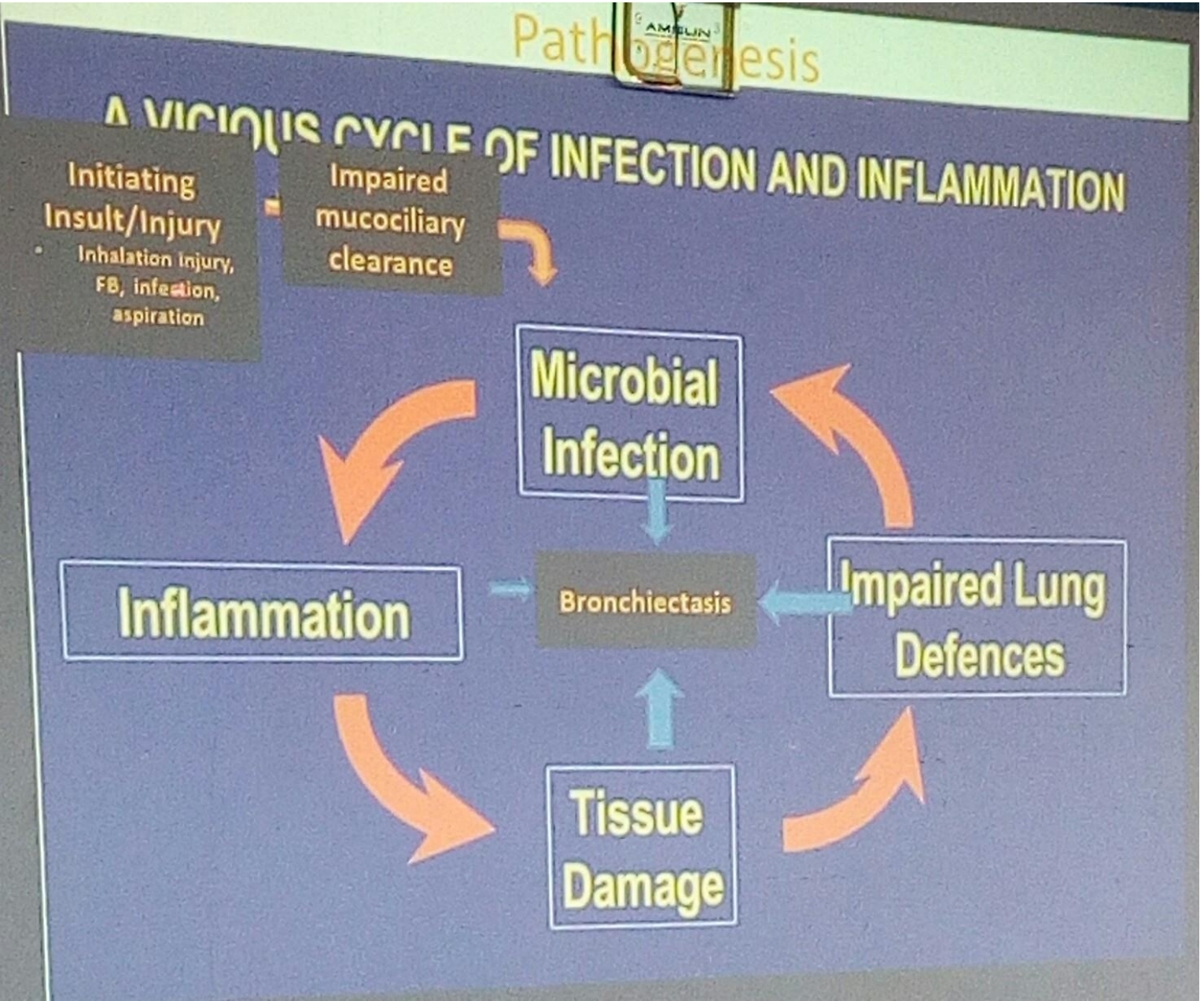
- Clinically, bronchiectasis is characterized by a chronic cough and purulent sputum production.

EPIDEMIOLOGY

- The burden of bronchiectasis has not been well characterized

- Prevalence is influenced by:
  - Access and uptake of childhood vaccination
  - Prevalence of TB
  - Treatment of respiratory tract infections
  - Living conditions
  - Availability of chest CT scan

VICIOUS CYSLE OF INFECTION AND INFLAMMATION



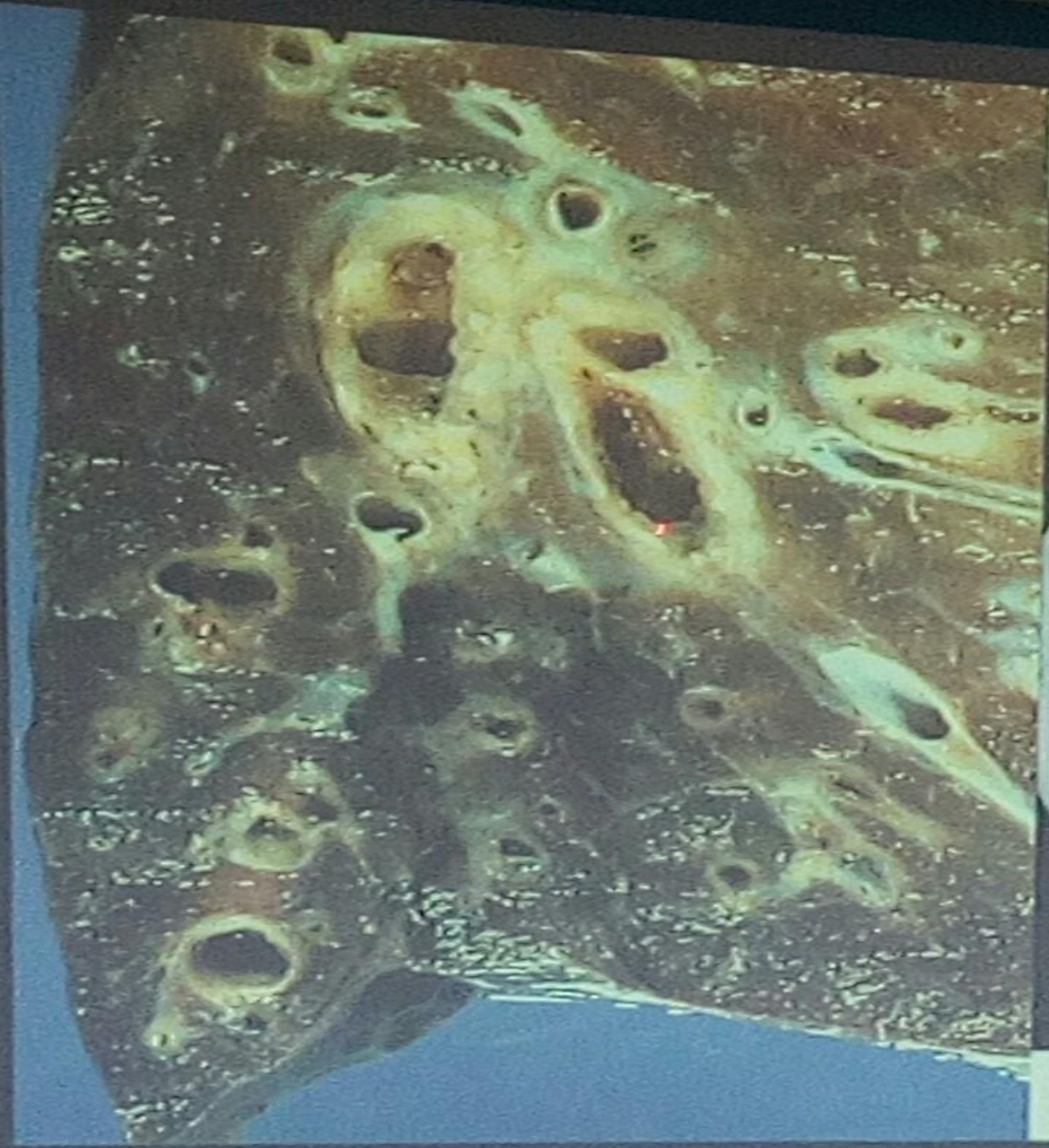
RISKS OF ASPIRATION

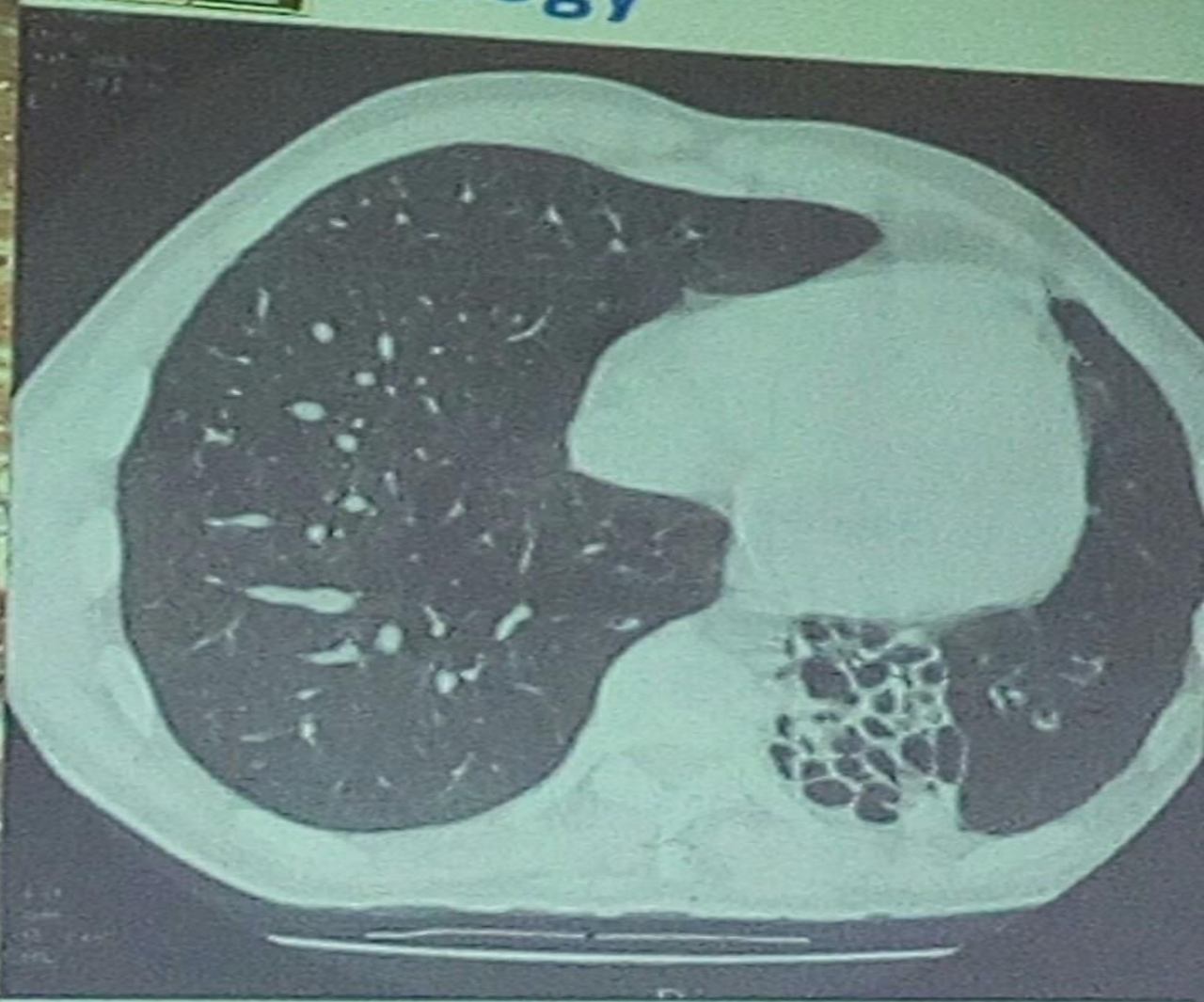
- **During endotracheal intubation**
- **Convulsing**
- **Comatose patients**

PATHOPHYSIOLOGY

- Neutrophil proteases acute infection in a normal or compromised host →
- Eepithelial injury (elastin, muscle and cartilage) + structural protein damage →
- Damaged and dilated airway (+ fibrosis extending to the adjacent lung parenchyma) →
- Mucus retention/chronic, recurrent infection →
- Ongoing inflammation/tissue damage and repair/ chronic inflammation, lymphoid follicles and neutrophil sin the airway wall →
  - Overproduction of viscid mucus
  - Impaired mucociliary clearance
  - Dilatation of airway
  - Mucus stagnation
  - Bacterial colonization

Pathology





ETIOLOGY

- Infection
  - TB. Necrotising pneumonia (*H. influenza, S. aureus,* aspiration), Pertussis, Influenza, Measles
- Systemic disease
  - RA, Ulcerative colitis, SLE, Sjogren's syndrome, ankylosing spondylosis, yellow nail syndrome
- Bronchial obstruction
  - Foreignbody, tumor e.g. carcinoid, mucoid impaction (can can be a complication of asthma), Allergic Bronchopulmonary aspergillosis (ABPA)
- Congenital anatomical lung abnormality
- Inherited disorders
  - Ciliary dysfunction
  - Cystic fibrosis
  - Alpha – 1 AT deficiency
- Undefined (29 – 49 %)
  - Most of these due to congenital or acquired immunodeficiency disorders

EVALUATION

- Take a good history
- Role of investigations
  - Underlying cause (especially those amenable to specific treatment)
  - Prognosis

HISTORY

- Symptoms
  - Chronic cough, purulent sputum production
  - Episodic exacerbations
    - General malaise, joint pains, increasing breathlessness, hemoptysis
  - Age of onset
  - Associated upper airway disease e.g. sinusitis, recurrent otitis media
  - H/O severe respiratory tract infection e.g. PTB
  - Family history (including fertility)
  - GERD
    Asthma features (suggestive of ABPA)
  - Effect on quality of life.

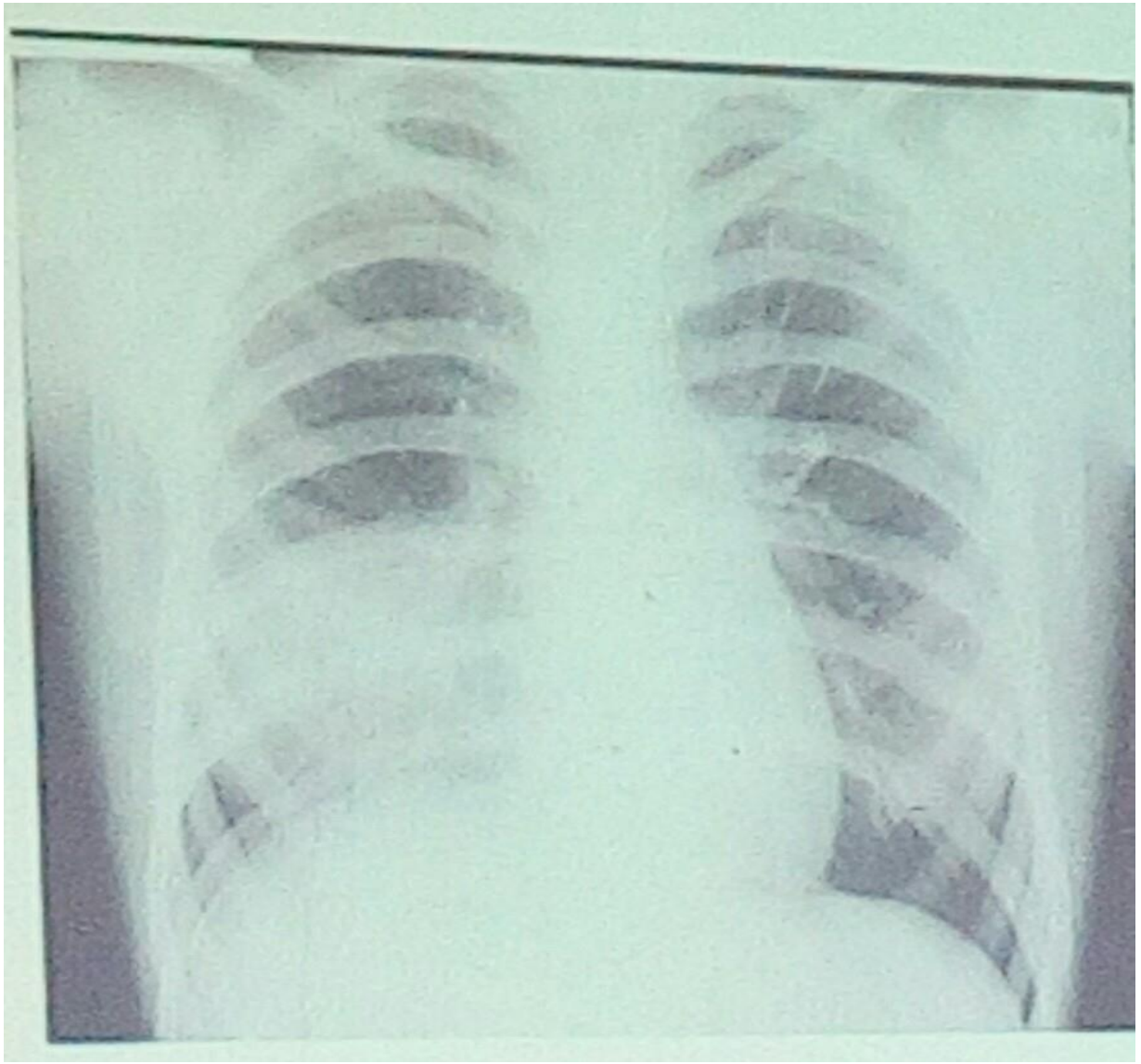
EXAMIANTION

- None – few
- Clubing
- Crackles

RADIOLOGY - CXR

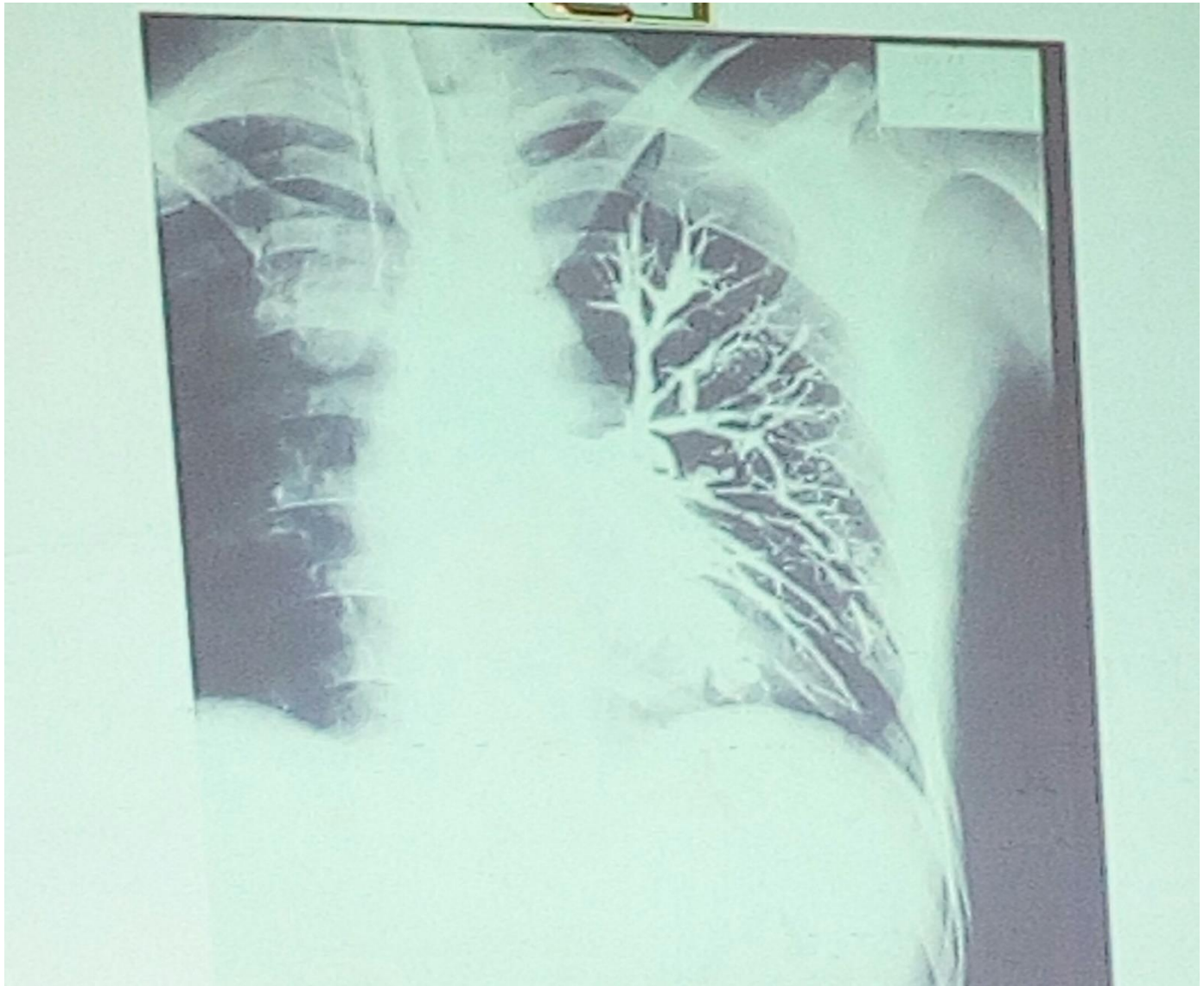
- Bronchiectasis
  - Vessel crowding
  - Loss of vessel markings
  - Tramline/ring shadows
  - Cystic lesions /air fluid levels

- Poor
  - Diagnostic sensitivity
  - Monitoring progression



BRONCHOGRAM → NOT DONE ANYMORE

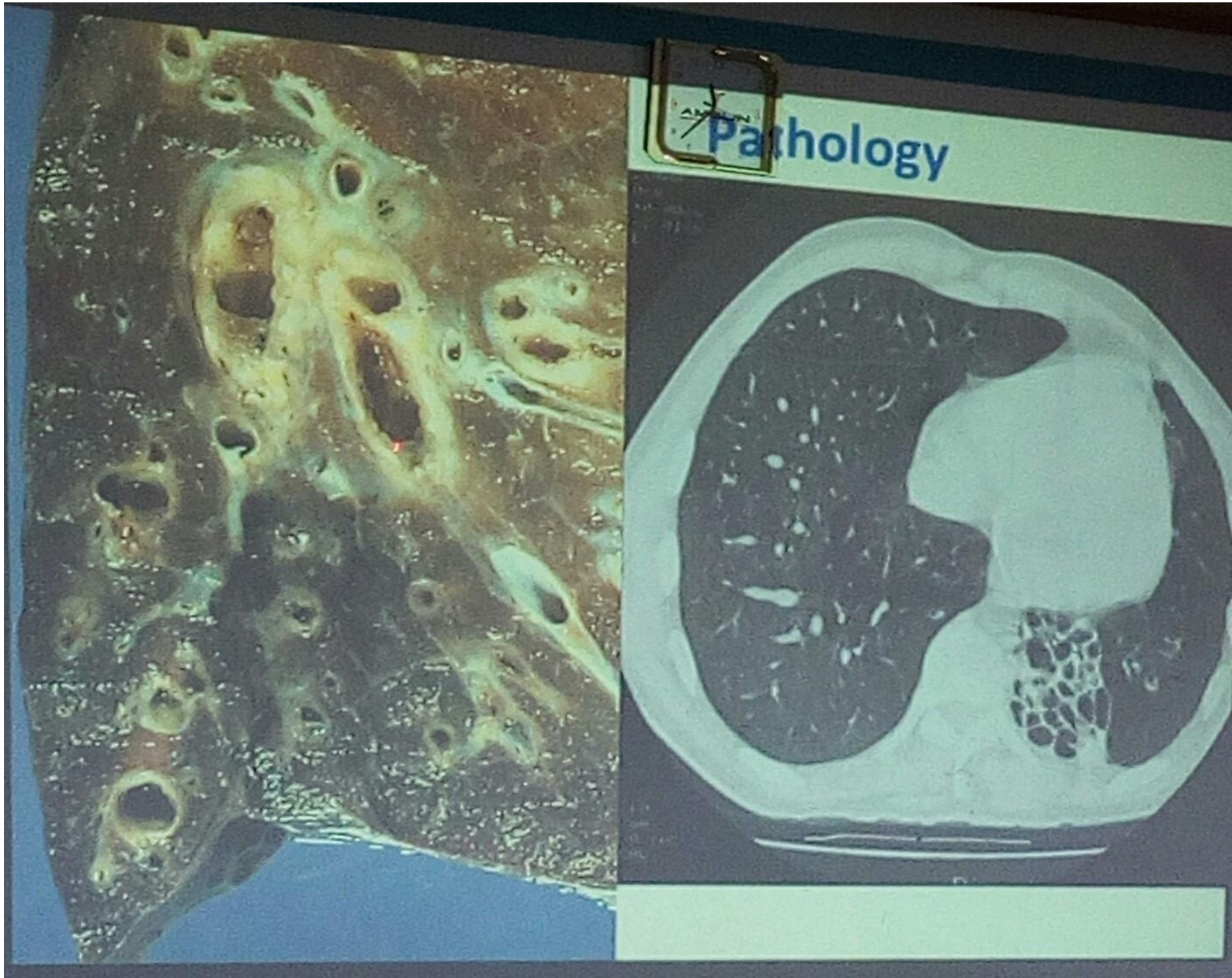
- Contrast is put in airways



HRCT

- Brochial dilatation
- Brochial wall thickening
- Classification (pathology)

- Sensitivity 97% mire than CXR



OTHER INVESTIGATIONS

- TBC
  - Raised eosinophilia → ABPA
- ESR/CRP
  - Raised
- Serum immunoglobulins
  - Immunodeficiency e.g. hematologic malignancies
- Aspergillus IgE (RAST) or IgG (precipitins)
- Skin prick test for Aspergillus
- Sputum microbiology
- Esophageal studies
- Ciliary studies
- CF genotype/sweat test
- Semen analysis
- FOB (Fibre optic Brocnoscopy)
- Lung function tests

BRONCHIECTASIS ECACERBATIONS IN BACERIOLOGY

- Common
  - *H. influenza*
  - *H. parainfluenza*
  - *Pseudomonas aeruginosa*
- Less common
  - *S. pneumonia*
  - *Moraxella catarrhalis*
  - *S. aureus*
  - *Stenotrophomonas maltophila*

MANAGEMENT

- Survival
- Preserve lung function
- Prevent frequent exacerbations
- Reduce symptoms and improve quality of life.

MANAGEMENT PRINCIPLES

- Patient education
- Airway clearance
- Reduced bacterial load
  - Rx of exacerbations
  - Antibiotic prophylaxis
- Anti-inflammatory therapy (inhaled corticosteroids)
- Vaccination
- Airway clearance
  - Postural drainage (percuss while you are at it to produce the sputum which is put in a sputum mug)

PULMONARY VASCULAR DISEASE: PULMONARY EMBOLISM

BY: PROF. ELIJAH N. OGOLA

DATE: 16/9/2016

OUTLINE

- VTE continuum
- Risk factors
- Pathophysiology
- Clinical features
- Investigations
- Diagnosis
- Treatment
  - Immediate
  - Long term
- Prevention

VTE

- DVT and PE are part of a continuum
- PE almost invariably due to DVT
- Same risk factors and similar management

PREAMBLE

- Common problem → 3rd commonest cause of cardiovascular mortality
  - 1. ISCHEMIC HEART DISEASE
  - 2. STROKE
  - 3. VTE
- Increasing incidence because of changing demographics and healthcare

- Associated with specific risk factors therefore amenable to prevention

RISK FACTORS

Strong predisposing factors

- Fracture (hip or leg)
- Hip or knee replacement
- Major general surgery
- Major trauma
- Spinal cord injury

Moderate predisposing factors

- Arthroscopic knee surgery
- Central venous lines
- Chemotherapy
- Chronic heart or respiratory failure
- Hormone replacement therapy
- Malignancy
- Oral contraceptive therapy
- Paralytic stroke
- Pregnancy/post-partum
- Previous VTE
- Thrombophilia (inherited)

CONT.

Weak predisposing factors

- Bed rest > 3 days
- Immobility due to sitting (e.g. prolonged car or air travel)
- Increasing age
- Laparoscopic surgery e.g. cholecystectomy)
- Obesity
- Pregnancy/antepartum
- Varicose veins

PRIMARY THROMBOPHILIAS

- Anti-thrombin deficiency
- Factor V Leiden
- Protein C, S deficiency
- Prothrombin 202120A mutation
- Hyper-homocystinemia
- Dys-fibrinogenemia
- Anti-cardiolipin antibodies
- Excessive PAI
- Plasminogen deficiency
- Thrombomodulin deficiency
- Factor XII excess
- Dysgammaglobulinemia

PATHOPHYSIOLOGY

- Cardiac
  - Obstruction to pulmonary blood flow
  - Increased pulmonary vascular resistance
  - Worsened by vasoconstrictors released
  - RV dysfunction , ischemia
  - Systemic circulation; impaired LV filling – hypotension, shock
- Respiratory → hypoxia
  - Low CO
  - V/Q mismatch
  - R-L shunting
  - Atelectasis → lung compliance
  - Decreased lung compliance (increased stiffness)
  - Infarction

VIRCHOW'S TRIAD

- Hyper-coagulable state
  - Malignancy
  - Pregnancy and post-partum period
  - Estrogen therapy
  - IBD
    Sepsis
  - Thrombophilia
- Circulatory stasis
  - LV dysfunction
  - Immobility or paralysis
  - Venous insufficiency or varicose veins
  - Venous obstruction from tumor, obesity or pregnancy
- Endothelial injury
  - Venous disorders
  - Venous valvular damage
  - Trauma or surgery
  - Indwelling catheters

CLINICAL PRESENTATION

- Non-specific hence high index of suspicion
- Variable dependent on extent of thrombus and underlying cardio-respiratory status
- Symptoms:
  - Dyspnea. Chest pain, cough, hemoptysis, dizziness, syncope
- Signs
  - Tachypnea, tachycardia, hypotension, cyanosis
- Features of DVT

INVESTIGATIONS

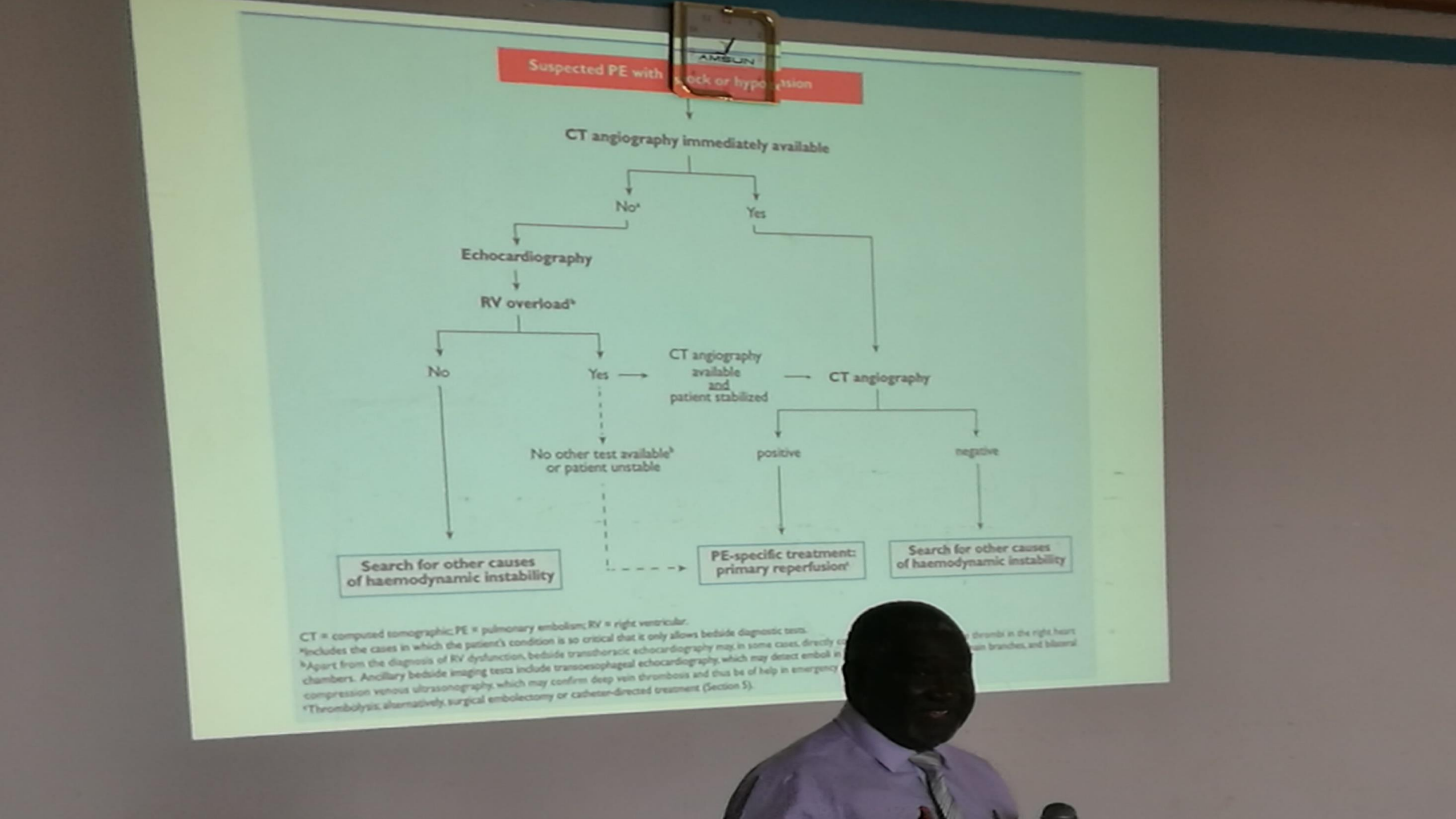
- Aims:
  - Establish diagnosis
  - R/O competing diagnoses
  - Look for risk factors
  - Assess complications
  - Risk stratification → prognosis

INVESTIGATIONS

- **D-dimer**
  - Assess possibility of intravascular coagulation
  - Sensitive but not specific
  - R/O intravascular coagulation
- **CXR**
  - Not diagnostic; helps in R/O competing diagnoses e.g. TB or pneumonia
  - **Find out features of CXR in PTE**
- **ECG**
  - Seeing any evidence of RV function impairment
  - R/O MI
- **Venous U/S**
  - **Gold standard for diagnosis DVT**
- **Cardiac biomarkers** → BNP, Troponin
  - Troponin is a marker of myocardial necrosis
  - BNP is a marker of myocardial stretch
- **Echocardiography**
  - Predominantly in telling the consequences of the PTE to the RV
  - Can provide indirect evidence of acute RV dysfunction
- **Lung scintigraphy (V/Q scan)**
  - Useful for diagnosis
  - Used to be the gold standard but has been overtaken by CT pulmonary angiography
  - Is therefore an alternative to CT
- **CT pulmonary angiogram**
  - **Test of choice for PTE**
  - Contrast is used; injected into a peripheral vein
- **Pulmonary angiography**
  - Involves catheterizing the right heart and injecting dye straight into it
  - Invasive; rarely done
- **Pulmonary Magnetic Resonance Angiography (MRA)**
- **Blood gases**

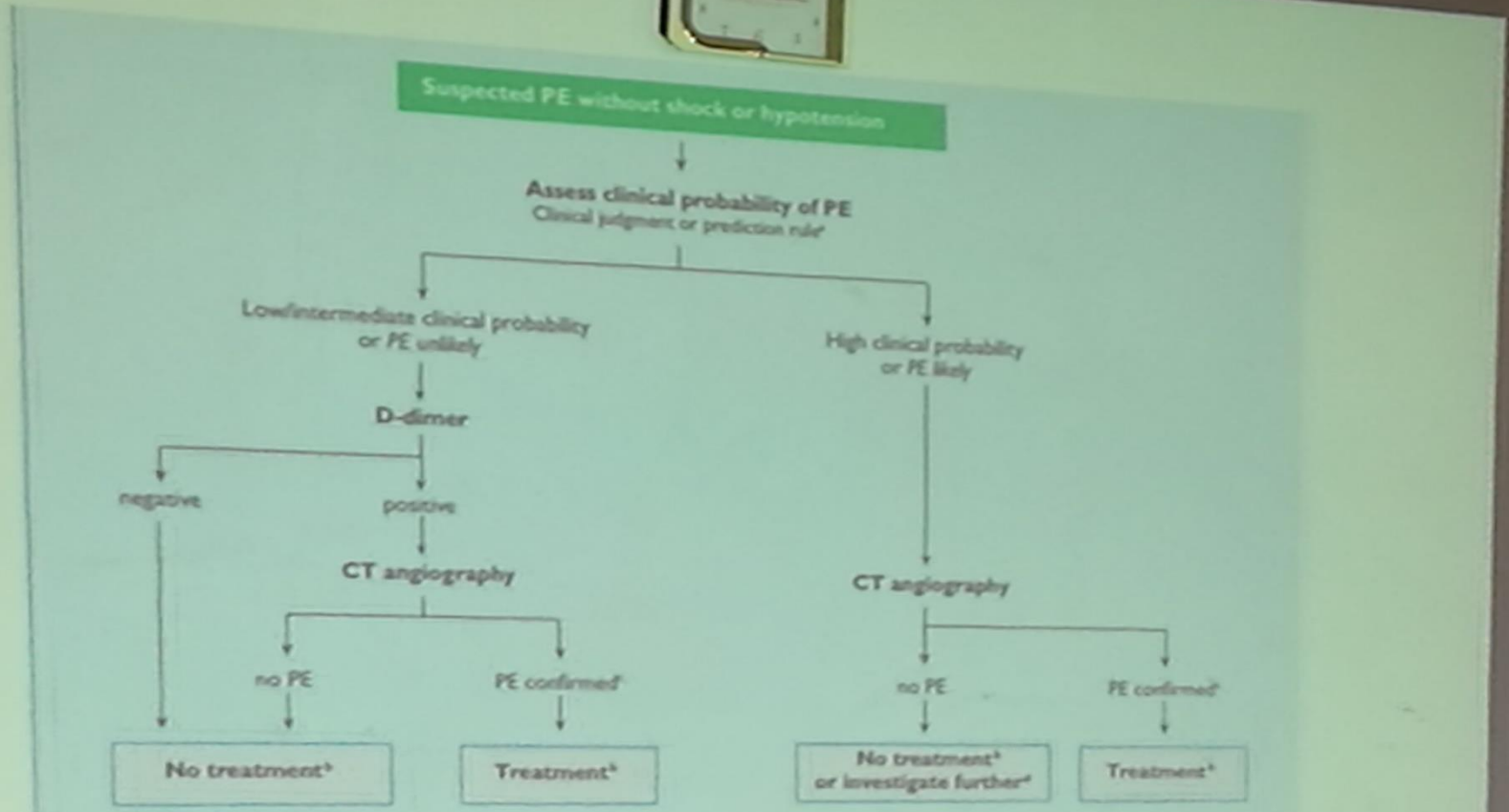
DIAGNOSTIC STRATEGIES

- Is there hypotension or shock
- If not, is the probability of PE high or low, using clinical parameters?


Suspected PE with
ock or hypo ssion
CT angiography immediately available
No[a]
Yes
Echocardiography
RV overload[b]
No
Yes
CT angiography available and patient stabilized
CT angiography
No other test available[b] or patient unstable
positive
negative
Search for other causes of haemodynamic instability
PE-specific treatment: primary reperfusion[c]
Search for other causes of haemodynamic instability
CT = computed tomographic; PE = pulmonary embolism; RV = right ventricular.
[a]Includes the cases in which the patient's condition is so critical that it only allows bedside diagnostic tests.
[b]Apart from the diagnosis of RV dysfunction, bedside transthoracic echocardiography may, in some cases, directly
chambers. Ancillary bedside imaging tests include transoesophageal echocardiography, which may detect emboli in
compression venous ultrasonography, which may confirm deep vein thrombosis and thus be of help in emergency
[c]Thrombolysis; alternatively, surgical embolectomy or catheter-directed treatment (Section 5).
thrombi in the right heart
ain branches, and bilateral

CRITERIA

- If suspected PE with shock or hypotension →
  - CT angiography immediately (if available) →
    - **Yes** → (do CT angiography)
      - Positive → **PE – Specific treatment; primary reperfusion**
      - Negative → search for other causes of hemodynamic instability
    - **No** →
      - Echocardiography → RV overload
        - **No** → search of other causes
        - **Yes**
          - CT angiography available and patient stabilized → (do CT angiography)
          - No other test available or patient unstable → **PE – Specific treatment; primary reperfusion**



CT = computed tomographic, PE = pulmonary embolism.

*Two alternative classification schemes may be used for clinical probability assessment, i.e. a three-level scheme (clinical probability defined as low, intermediate, or high) or a two-level scheme (PE unlikely or PE likely). When using a moderately sensitive assay, D-dimer measurement should be restricted to patients with low clinical probability or a PE-unlikely classification, while highly sensitive assays may also be used in patients with intermediate clinical probability of PE. Note that plasma D-dimer measurement is of limited use in suspected PE occurring in hospitalized patients.

†Treatment refers to anticoagulation treatment for PE.

‡CT angiogram is considered to be diagnostic of PE if it shows PE at the segmental level or more proximal level.

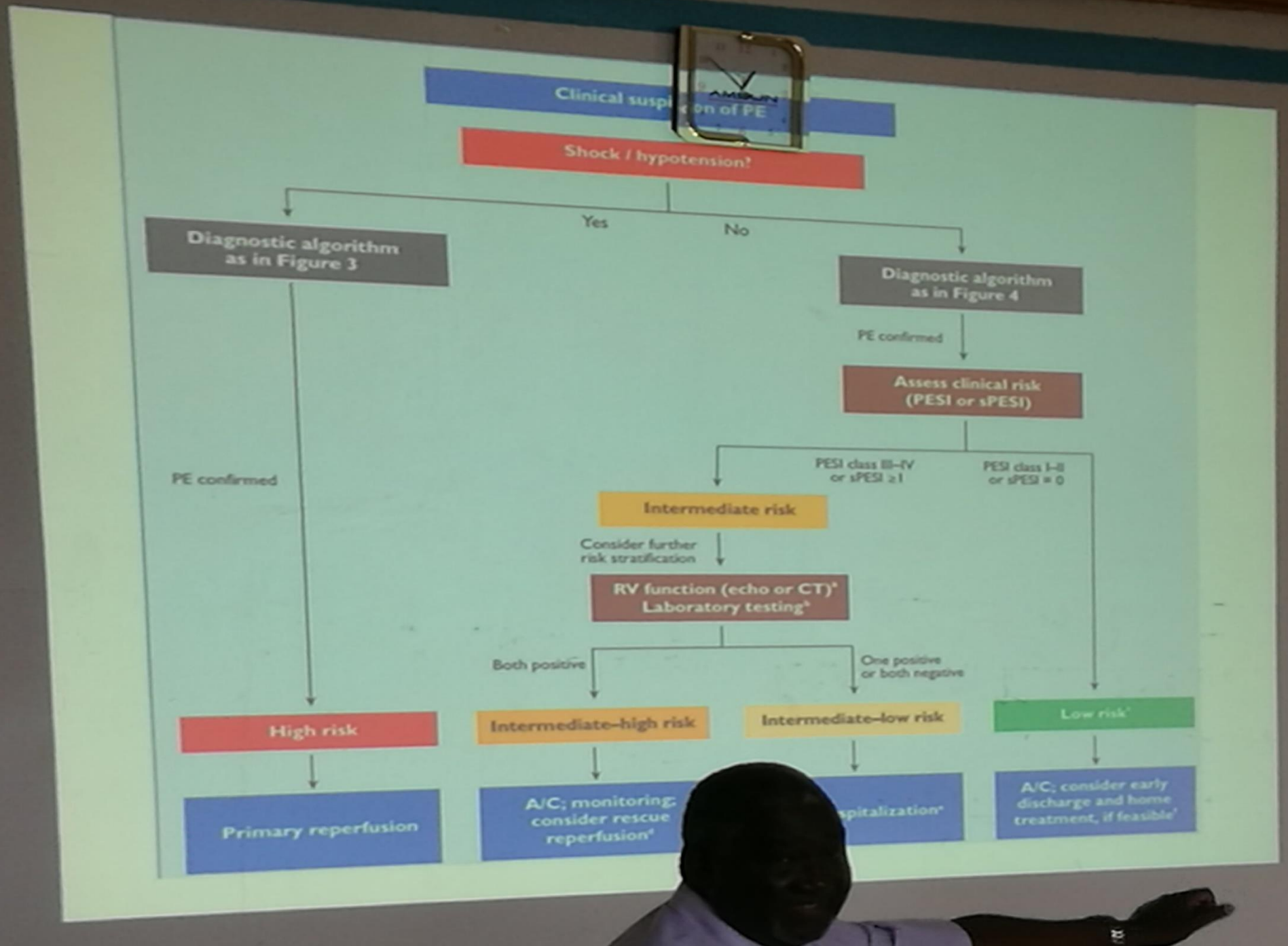
§In case of a negative CT angiogram in patients with high clinical probability, [illegible] be considered before withholding PE-specific treatment.

ALGORITHM

- **Suspected PE without shock →**
  - Assess clinical probability of PE (clinical judgment or prediction rule) →
    - Low intermediate clinical probability or PE unlikely →
      - **D-dimer →**
        - Negative →
          - No treatment
        - Positive →
          - **CT angiography**
            - No PE → no treatment
            - Confirmed → treatment
    - High clinical probability or PE likely →
      - CT angiography
        - No PE
          - No Rx or investigate further
        - PE confirmed
          - Treat

TREATMENT

- Risk stratification of probability of death
  - Hypotension/shock
  - RV dysfunction
  - Elevated BNP/Troponin
- Hypotension/shock → thrombolytic therapy
- None of the above → low risk. Anticoagulation, consider early discharge or home treatment
- No hypotension with one or 2 of the above → intermediate risk. Anticoagulation and observe


Clinical suspicion of PE
Shock / hypotension?
Yes
No
Diagnostic algorithm as in Figure 3
Diagnostic algorithm as in Figure 4
PE confirmed
Assess clinical risk (PESI or sPESI)
PE confirmed
PESI class III–IV or sPESI ≥ 1
PESI class I–II or sPESI = 0
Intermediate risk
Consider further risk stratification
RV function (echo or CT)[3]
Laboratory testing[3]
Both positive
One positive or both negative
High risk
Intermediate–high risk
Intermediate–low risk
Low risk[7]
Primary reperfusion
A/C; monitoring; consider rescue reperfusion[4]
spitalization[6]
A/C; consider early discharge and home treatment, if feasible[7]

TARGETS FOR ANTICOAGULANTS

- Oral
  - VKAs (Warfarin) inhibit hepatic synthesis of functional coagulation factors
  - Rivaroxaban, Apixaban, Edoxaban (FXa Inhibitors)
  - Dabigtaran (FIIa inhibitors)

- Parenteral
  - Fondaparinux
  - LMWH
  - UFH

ANTICOAGULATION STRATEGIES

- Initial parenteral therapy
  - UFH – do frequent aPTT measurements; should be infused; can be used with any kidney function levels
  - LMWH
  - Fondaparinux – indirect FXa inhibitor; given OD

- Followed by OAC
  - VKA (warfarin)
  - FXa inhibitors e.g. rivaroxaban, apixaban, edoxaban
  - FII inhibitor - Dabigatran

CONT.

- Initial parenteral therapy is for 5-10 days
- Simultaneous initiation of OAC
- In case of VKA overlap till INR is in therapeutic range (2-3) for 2 consecutive days
- For rivaroxaban and apixaban → possibility of initiating oral therapy

DURATION OF THERAPY

- 1st provoked (transient risk factors) → 3 months
- 1st unprovoked → at least 3 months. Consider extended Rx depending on bleeding risk
- Recurrence → indefinite
- Same for continuous risk factors e.g. cancer

OTHER TREATMENT MODALITIES

- Acute phase
  - Surgical or catheter embolectomy
  - Direct thrombolysis
- Long term – venous filters
  - High bleeding risk
  - C/I to anticoagulation
  - Recurrence despite adequate anticoagulation

PROPHYLAXIS

- Indicated for moderate to high risk
- Drugs:
  - Parenterals; (N) OAC
- Mechanical
  - Passive exercises
  - Compression stockings
  - Ripple mattresses

TYPED BY EFFIE NAILA

MANY ARE THE AFFLICTIONS OF THE RIGHTEOUS BUT THE LORD DELIVERS HIM FROM THEM ALL