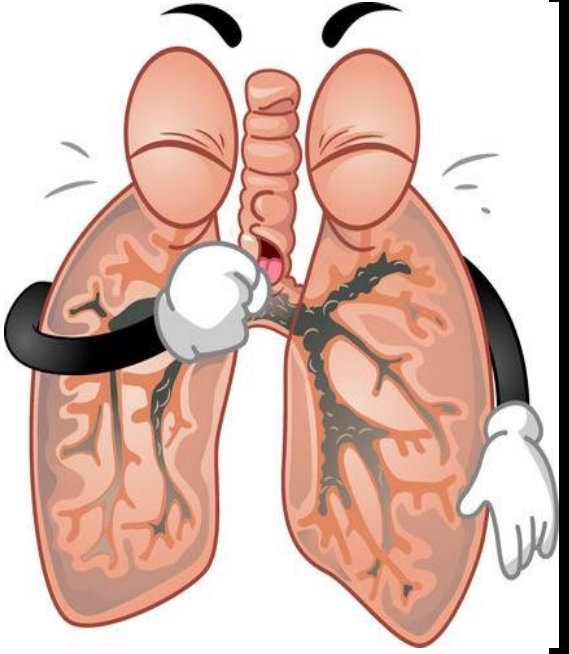

THE RESPIRATORY SYSTEM

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University of Jordan

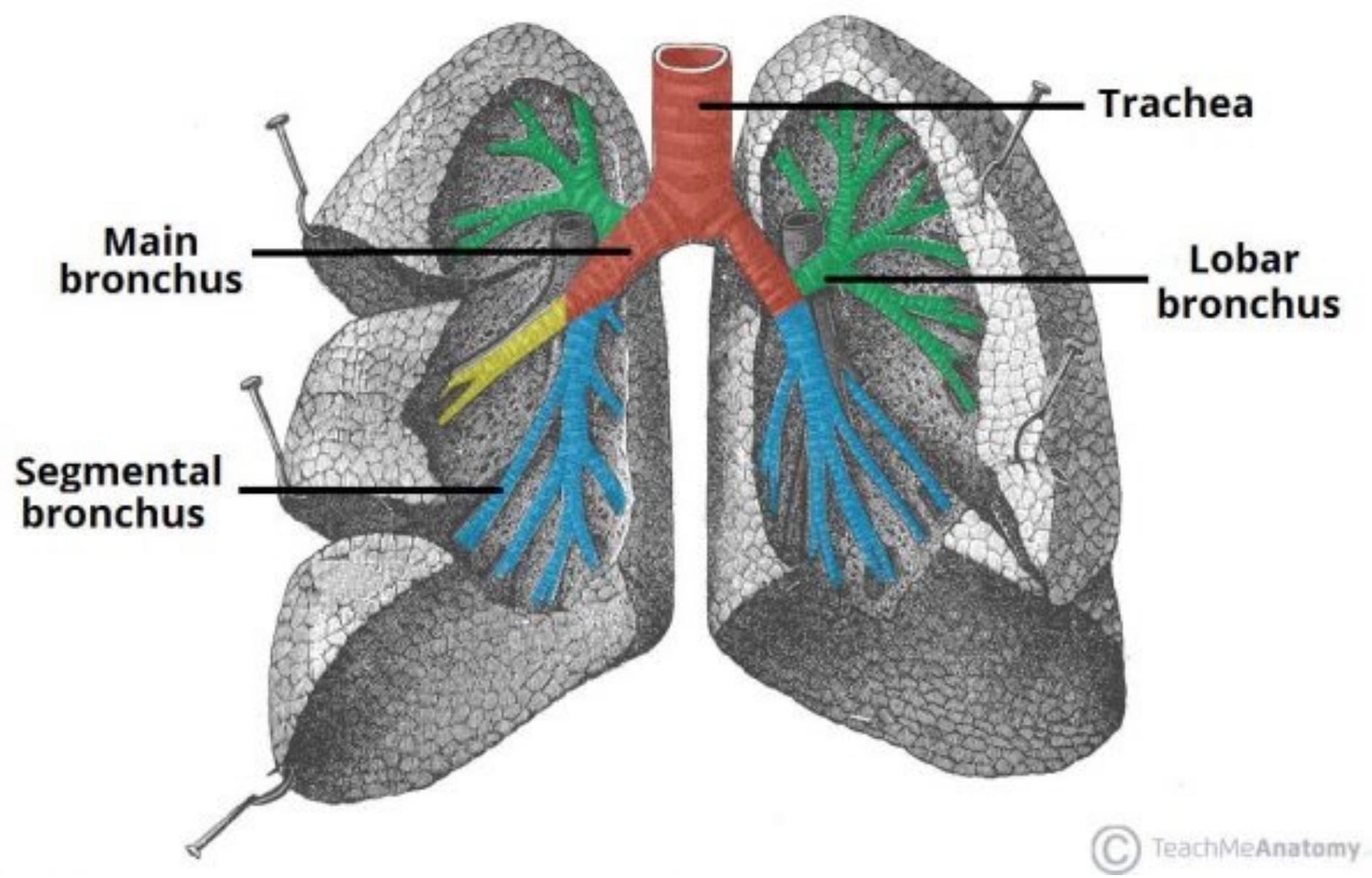
https://www.123rf.com/photo_38644498_stock-illustration-mascot-illustration-of-the-lungs-coughing-violently.html

OBJECTIVES:

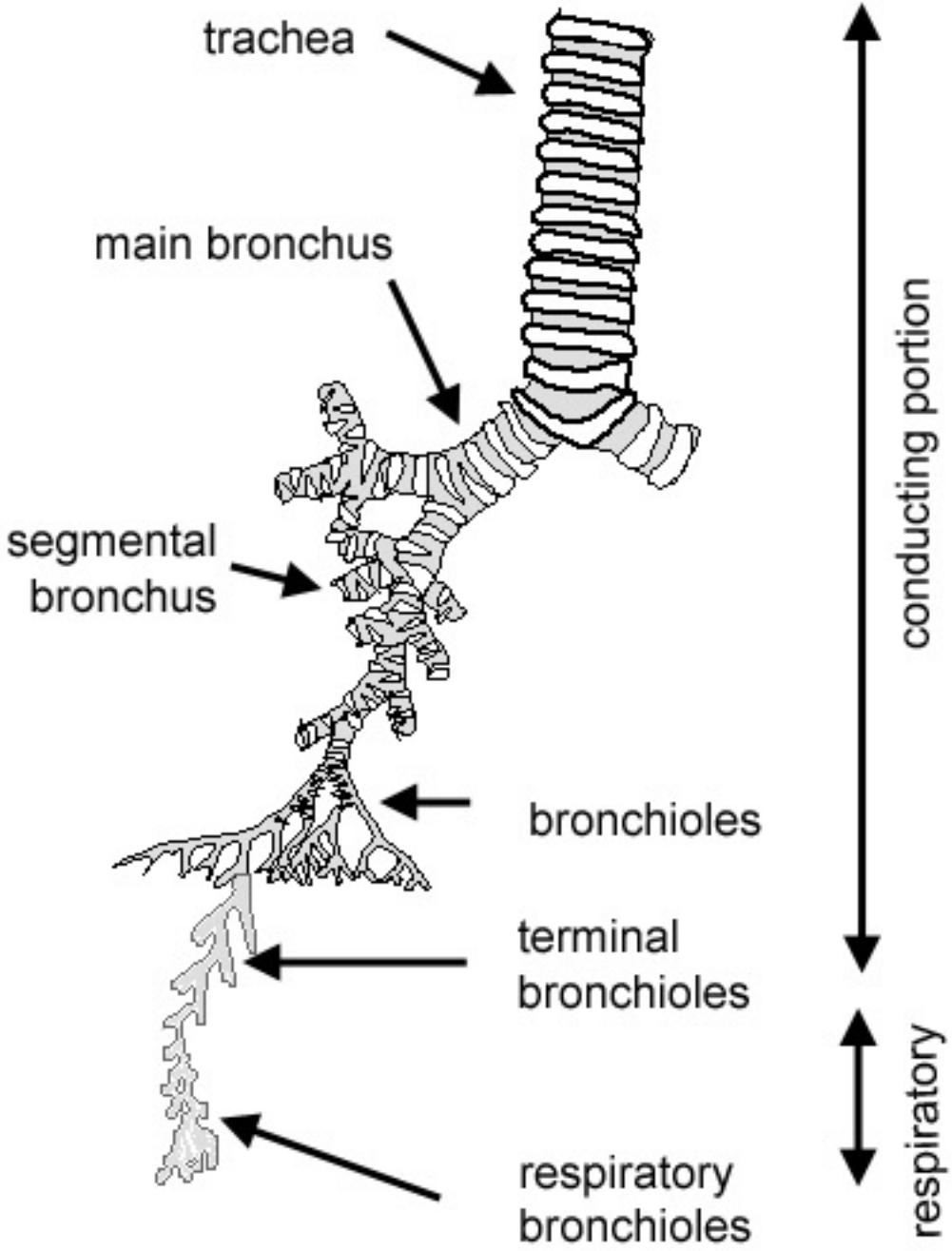
- Function and anatomy
- Atelectasis (Collapse)
- Acute respiratory distress syndrome (ARDS)
- Restrictive vs. Obstructive lung diseases

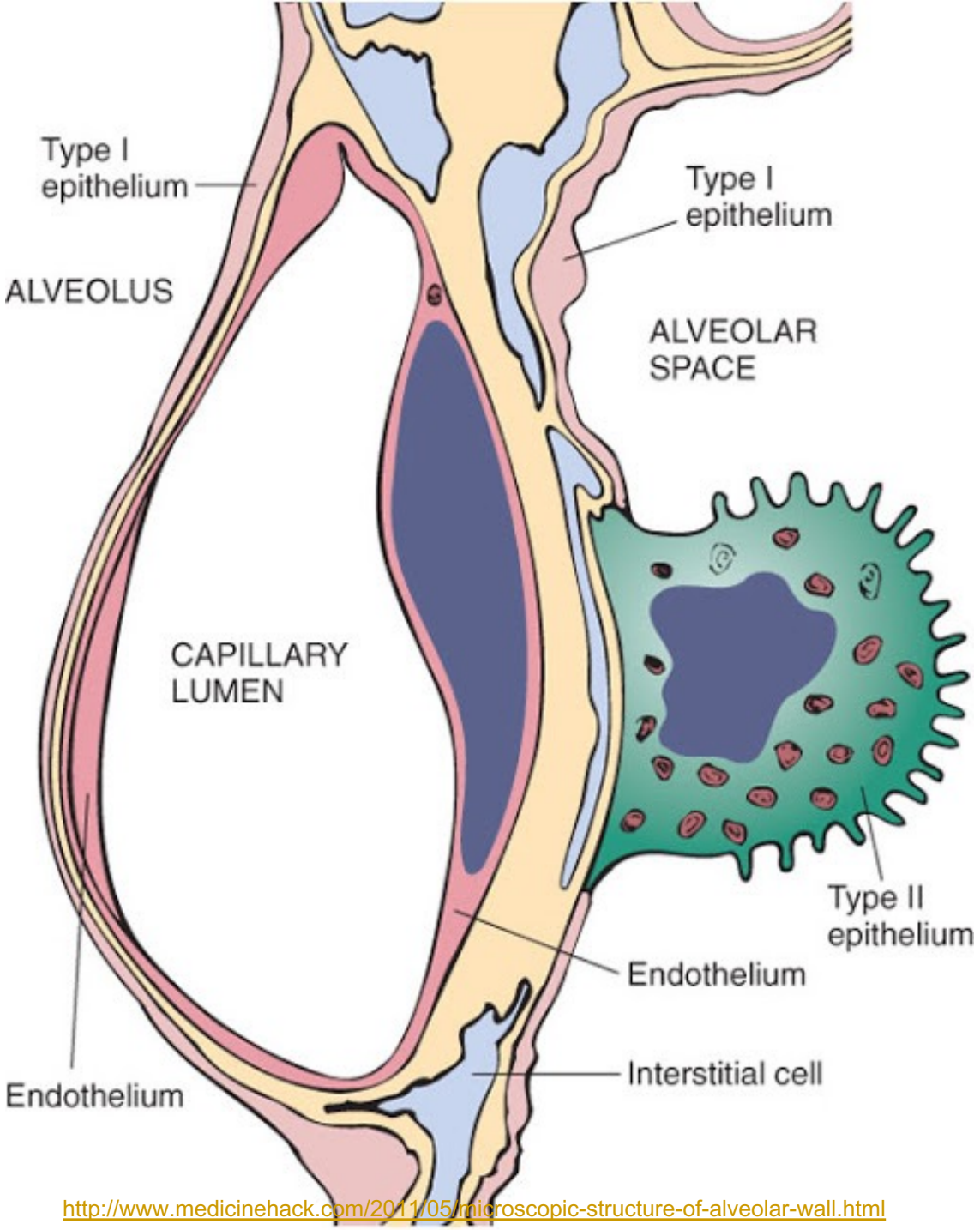
FUNCTION AND ANATOMY:

The major function of the lung is to replenish oxygen and remove carbon dioxide from blood.



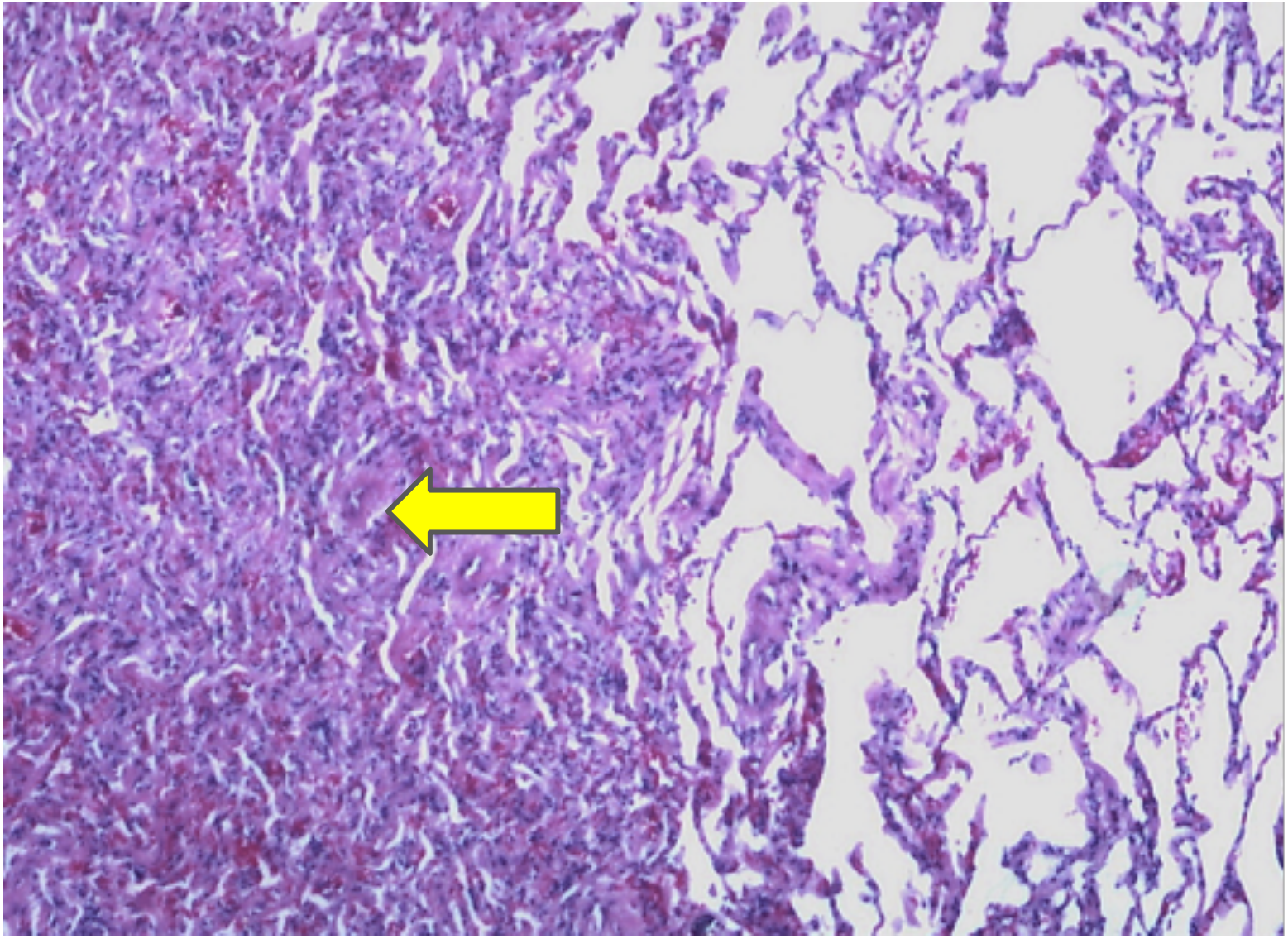
By TeachMeSeries Ltd (2019)https://teachmeanatomy.info/thorax/organs/tracheobronchial-tree/


trachea
main bronchus
segmental bronchus
bronchioles
terminal bronchioles
respiratory bronchioles
conducting portion
respiratory



http://www.medicinehack.com/2011/05/microscopic-structure-of-alveolar-wall.html

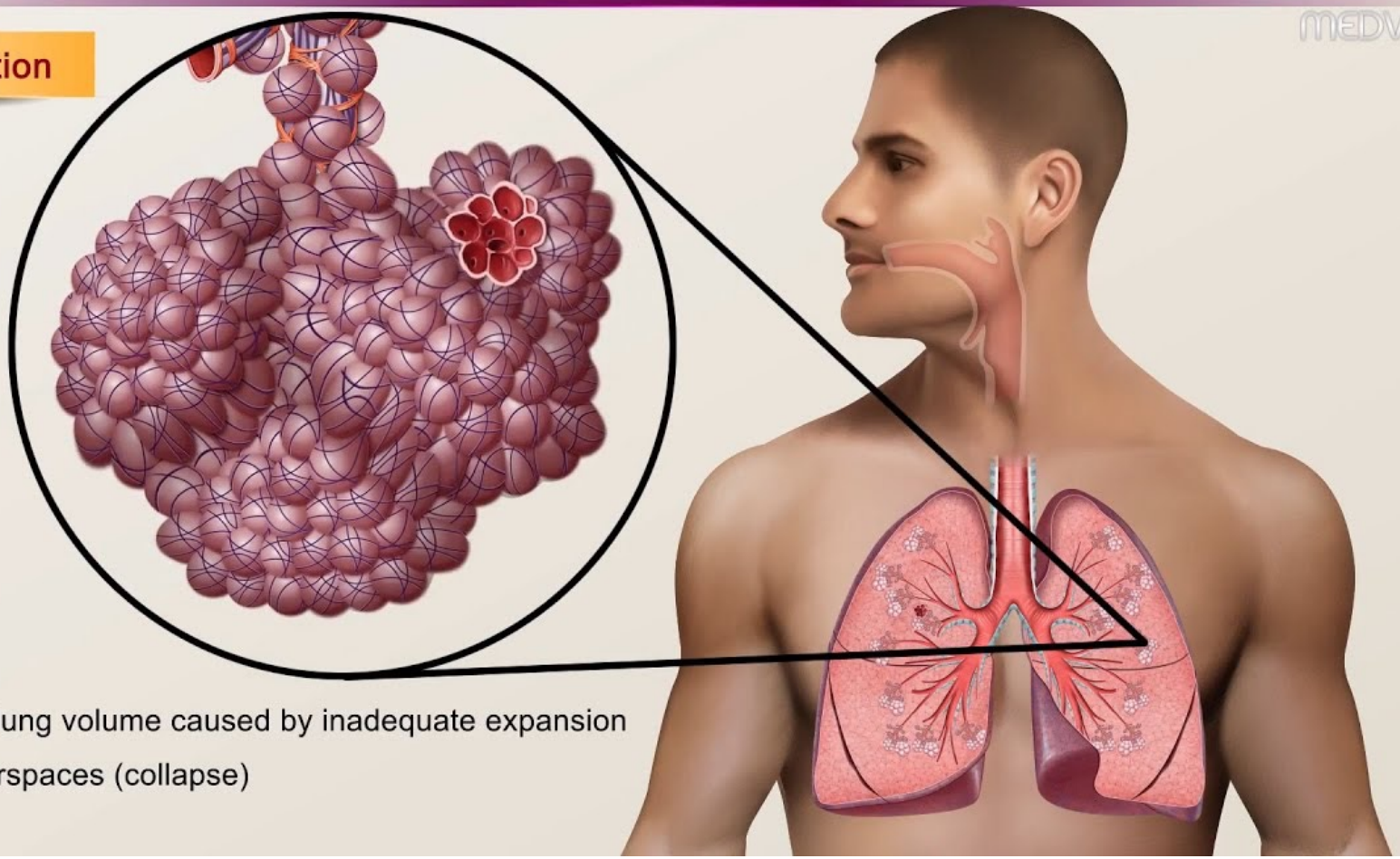
ATELECTASIS (COLLAPSE)



https://teachmesurgery.com/perioperative/cardiorespiratory/atelectasis/

Atelectasis

Definition



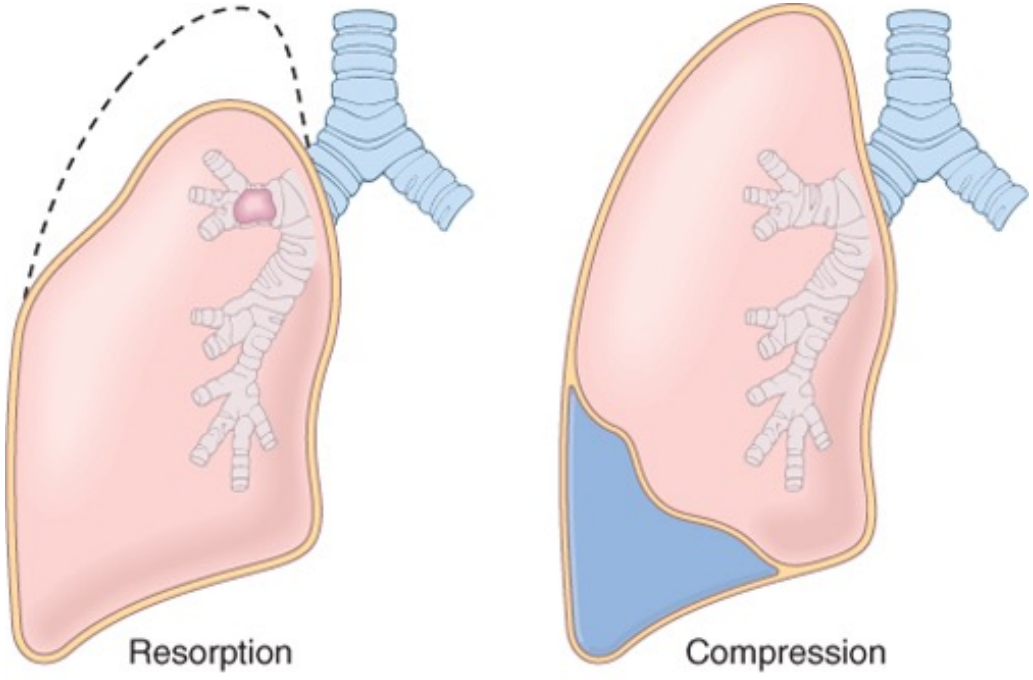
- Loss of lung volume caused by inadequate expansion of the airspaces (collapse)

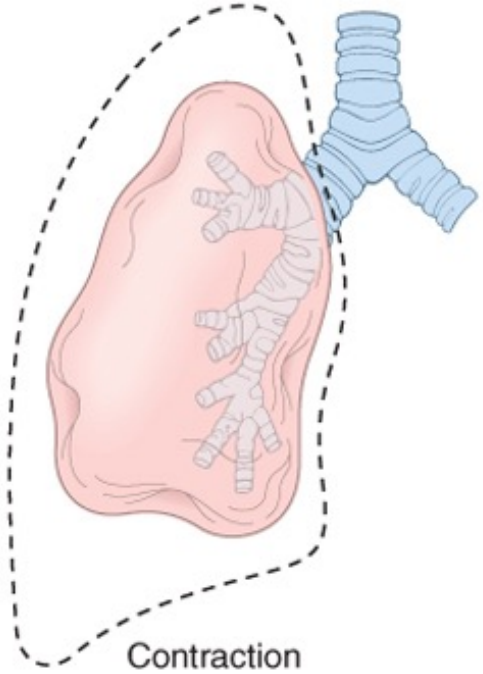
ATELECTASIS (COLLAPSE)

- is loss of lung volume caused by inadequate expansion of air spaces.
- It results in shunting of inadequately oxygenated blood from pulmonary arteries into pulmonary veins→ resulting in ventilation perfusion imbalance and hypoxia.
- The collapsed airway are at risk of infection

THREE TYPES OF ACQUIRED ATELECTASIS:

- Resorption atelectasis
- Compression atelectasis
- Contraction atelectasis (cicatrization atelectasis)



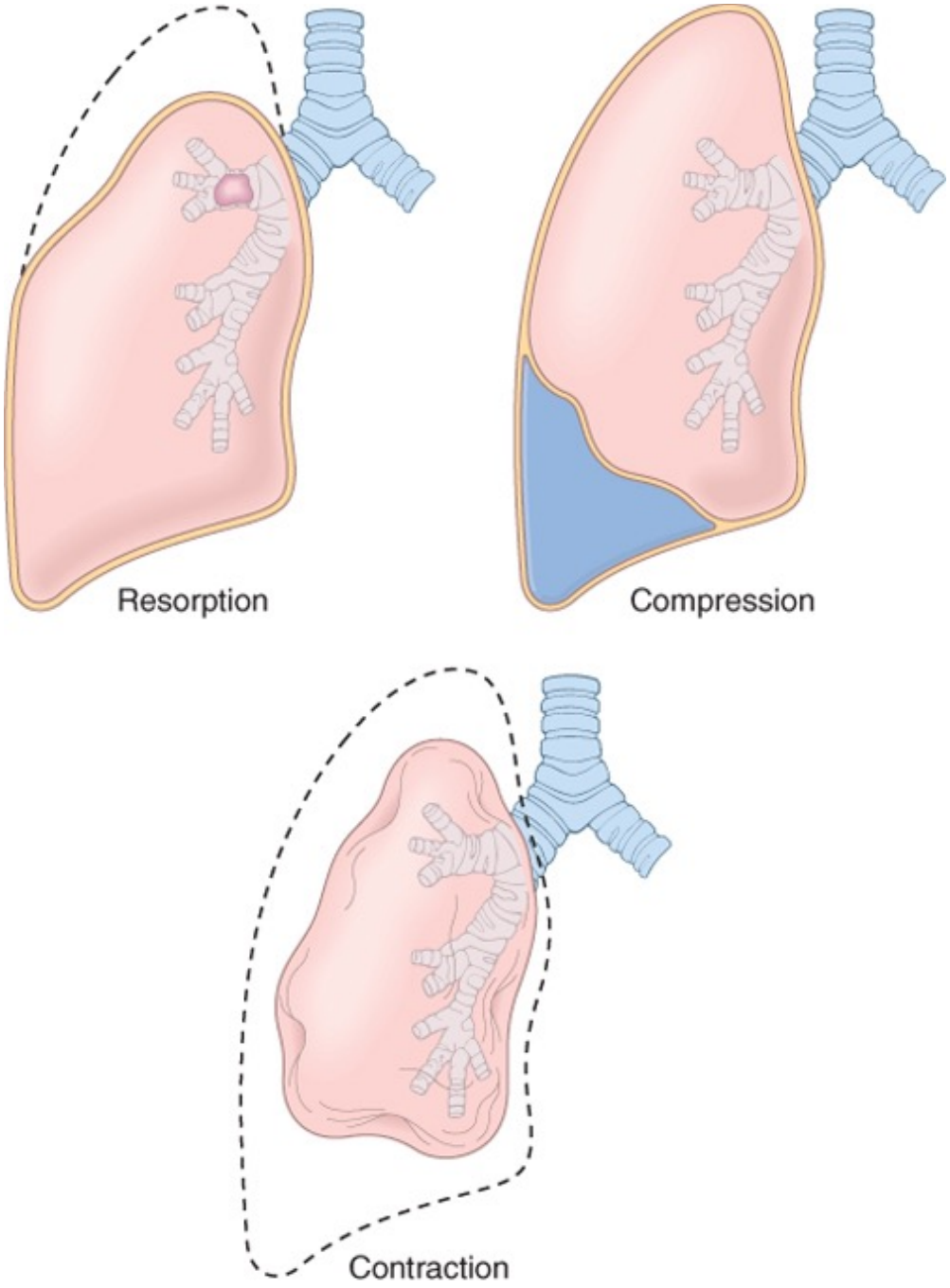


1.RESORPTION ATELECTASIS

- Due to total obstruction of an airway preventing air from reaching distal airways.
- The air already present in the distal airways gradually resorbed resulting in alveolar collapse.
- Alveolar collapse results in diminished lung volume → shifts of the mediastinum toward the atelectatic lung.
- Airway obstruction occurs in bronchi, segmental bronchi or terminal bronchi.

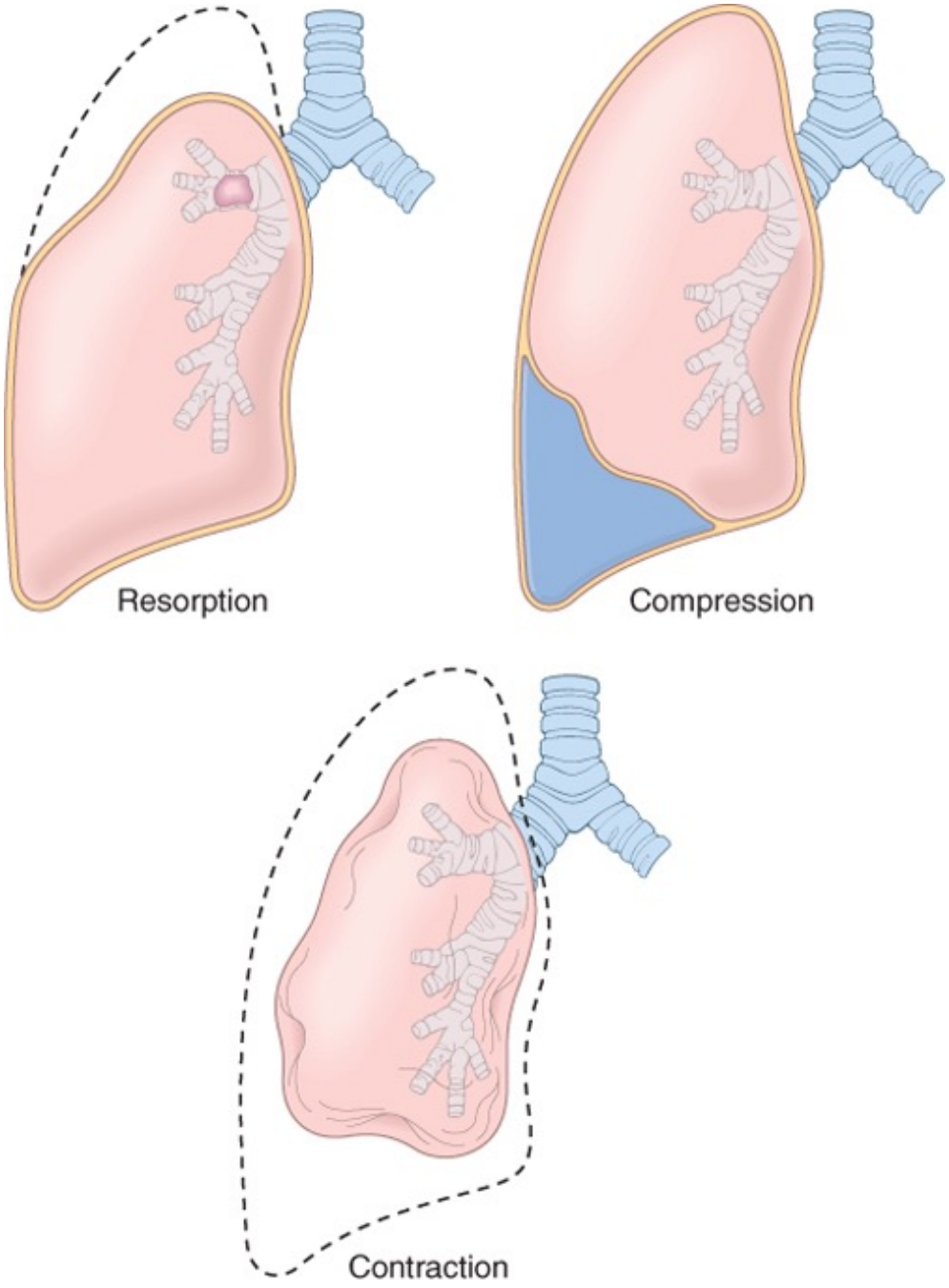
RESORPTION ATELECTASIS, CAUSED BY:

- **The most common cause is <u>Obstruction of a bronchus</u> by:**
  - ✓ Intrabronchial mucous or mucopurelant plugs in post operative patients.
  - ✓ Foreign body aspiration, especially in children
  - ✓ Obstructive lung disease: bronchial asthma, bronchiectasis, chronic bronchitis
  - ✓ Intrabronchial tumors.



2. COMPRESSION ATELECTASIS

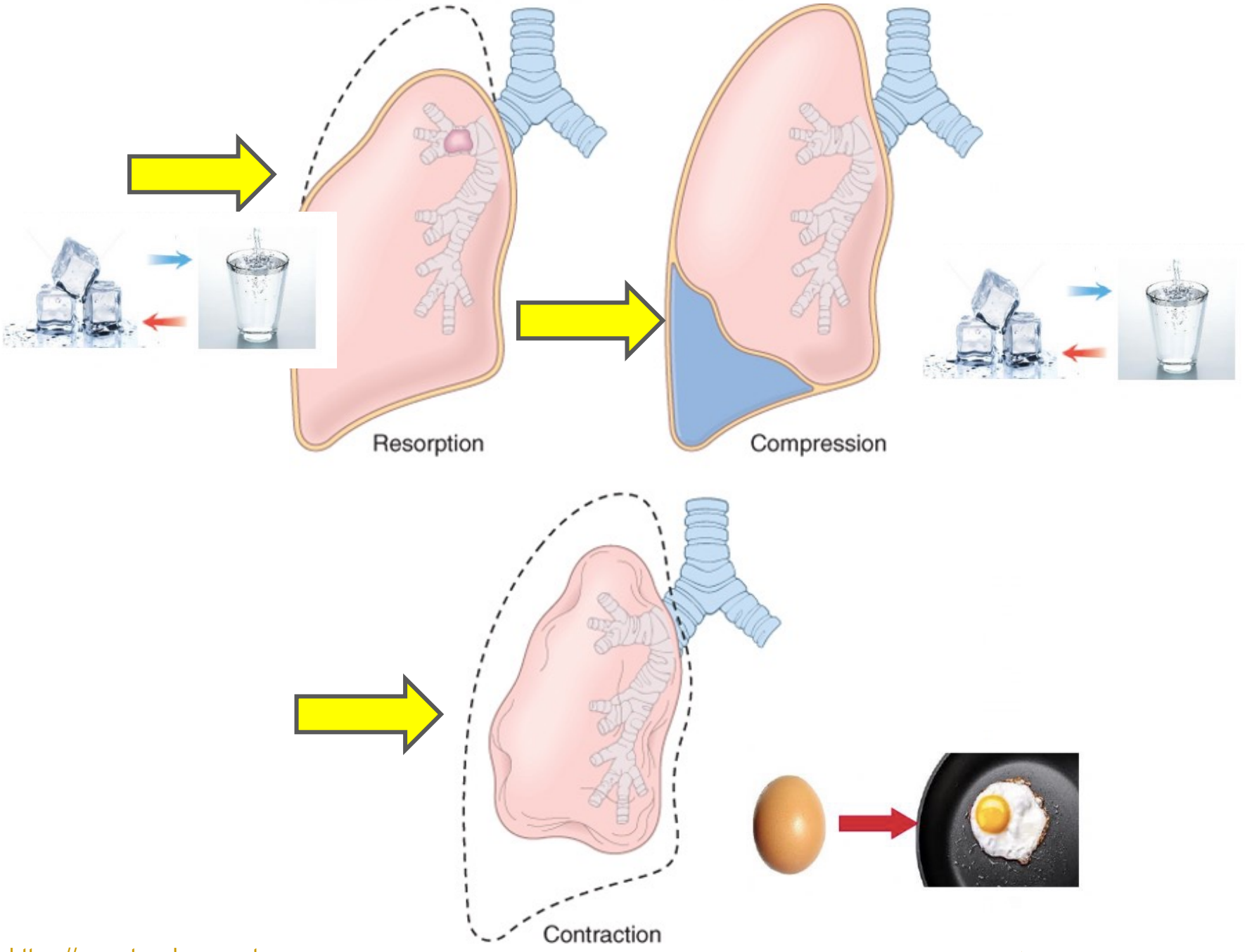
- caused by the accumulation of significant amount of fluid (blood, exudate or transudate), air (pneumothorax) or tumor within pleural cavity, which mechanically collapse adjacent lung (small airways and alveoli)
- mediastinum shifts away from the affected lung.
- E.x:
  a. Pleural effusion: in Congestive Heart Failure
  b. Pneumothorax: air in the pleural cavity due to RTA



3. CONTRACTION ATELECTASIS (CICATRIZATION ATELECTASIS)

- Occurs due to local or generalized fibrosis of the lung or pleura that prevents full expansion of the lung since the alveoli are trapped in fibrosis and scar

Atelectasis (except when caused by contraction) is potentially reversible and should be treated promptly to prevent hypoxemia and superimposed infection of the collapsed lung.



ACUTE LUNG INJURY AND ACUTE RESPIRATORY DISTRESS SYNDROME

- **Acute lung injury (ALI)**: characterized by the abrupt onset of hypoxemia and bilateral pulmonary edema in the absence of cardiac failure

- **Acute respiratory distress syndrome (ARDS)** is a manifestation of severe ALI.

- **ARDS defined as** respiratory failure occurring within 1 week of a known clinical insult with bilateral opacities on chest imaging, NOT fully explained by effusions, atelectasis, cardiac failure, or fluid overload.

ACUTE LUNG INJURY AND ACUTE RESPIRATORY DISTRESS SYNDROME

- Both are associated with inflammation-associated increases in pulmonary vascular permeability, edema, and epithelial cell death.
- The histologic manifestation of these diseases is called **diffuse alveolar damage.**

ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

- **Defined as** respiratory failure occurring within 1 week of a known clinical insult with bilateral opacities on chest imaging, NOT fully explained by effusions, atelectasis, cardiac failure, or fluid overload.
- graded based on the severity of the changes in arterial blood oxygenation into mild, moderate and sever
- Sever ARDS characterized by rapid onset of life-threatening **respiratory insufficiency, Cyanosis , Severe arterial hypoxemia that becomes refractory to oxygen therapy and may progress to multisystem organ failure**

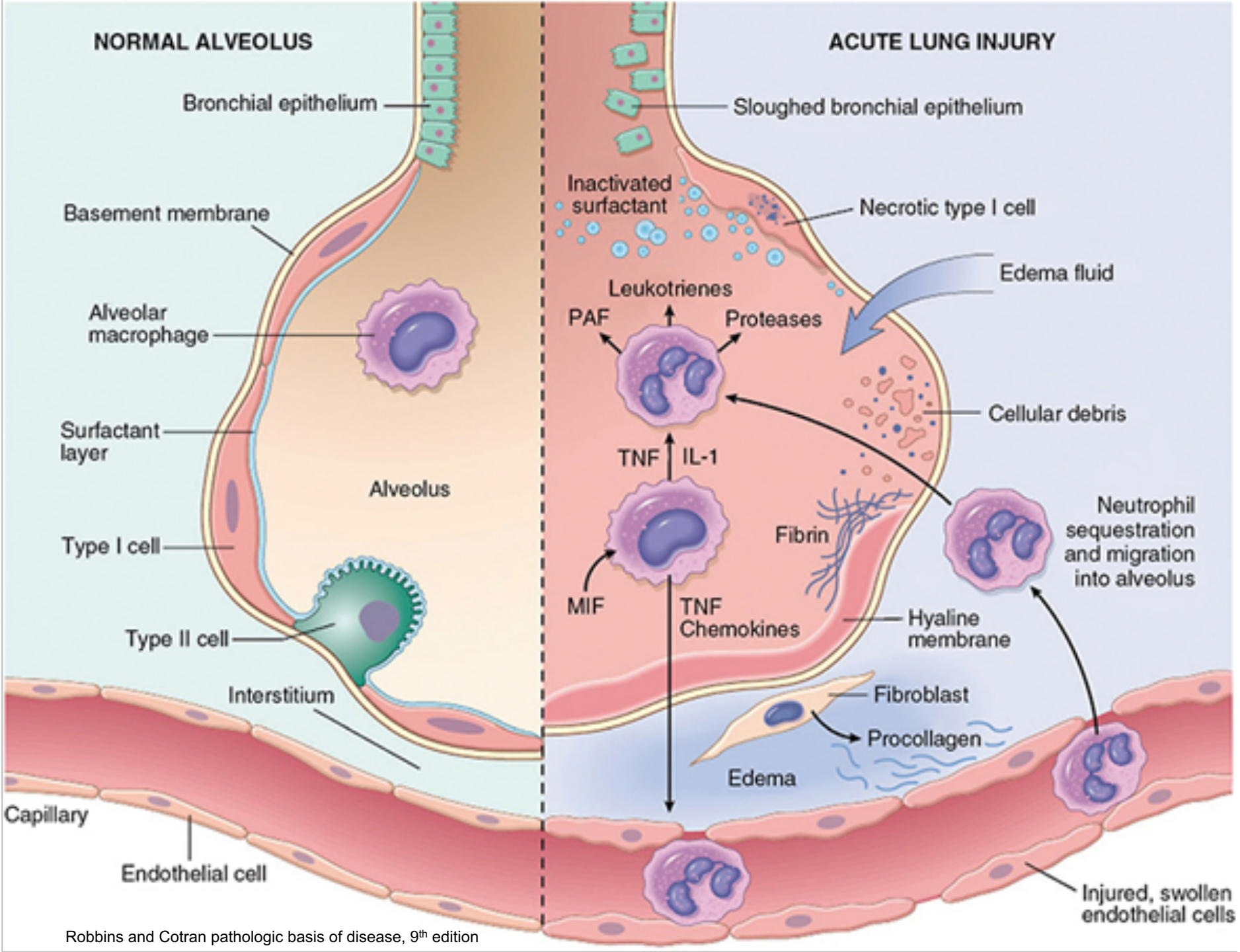
Table 15.2 Conditions Associated With Development of Acute Respiratory Distress Syndrome

Infection
Sepsis[a]
Diffuse pulmonary infections[a]
Viral, *Mycoplasma,* and *Pneumocystis* pneumonia; miliary tuberculosis
Gastric aspiration[a]
Physical/Injury
Mechanical trauma including head injuries[a]
Pulmonary contusions
Near-drowning
Fractures with fat embolism
Burns
Ionizing radiation
Inhaled Irritants
Oxygen toxicity
Smoke
Irritant gases and chemicals
Chemical Injury
Heroin or methadone overdose
Acetylsalicylic acid
Barbiturate overdose
Paraquat
Hematologic Conditions
Transfusion-associated lung injury (TRALI)
Disseminated intravascular coagulation
Pancreatitis
Uremia
Cardiopulmonary Bypass
Hypersensitivity Reactions
Organic solvents
Drugs

[a]More than 50% of cases of acute respiratory distress syndrome are associated with these four conditions.

ARDS should not be confused with respiratory distress syndrome of the newborn; the latter is caused by a deficiency of surfactant caused by prematurity.

PATHOGENESIS



Robbins and Cotran pathologic basis of disease, 9th edition

PATHOGENESIS:

- the integrity of the alveolar-capillary membrane is compromised by endothelial and epithelial injury.

- As early as 30 minutes after an acute insult, there is increased synthesis and release of IL-8, IL-1 and TNF by pulmonary macrophages.

- leading to endothelial activation and sequestration
- activation & chemotaxis of neutrophils in pulmonary capillaries.

PATHOGENESIS/CONT.

- Activated neutrophils release reactive oxygen species & proteases that damage the alveolar epithelium and endothelium causing vascular leakiness and loss of surfactant that render the alveolar unit unable to expand.

- the destructive forces are counteracted by endogenous anti-proteases and anti-oxidants

- **In the end, it is the balance between the destructive and protective factors that determines the degree of tissue injury and clinical severity of the ARDS.**

HISTOLOGY:

- In the acute phase of ARDS :

  - The most characteristic finding is the presence of **hyaline membranes**

  - consists of fibrin-rich edema fluid admixed with remnants of necrotic epithelial cells (similar to respiratory distress syndrome of the newborn)

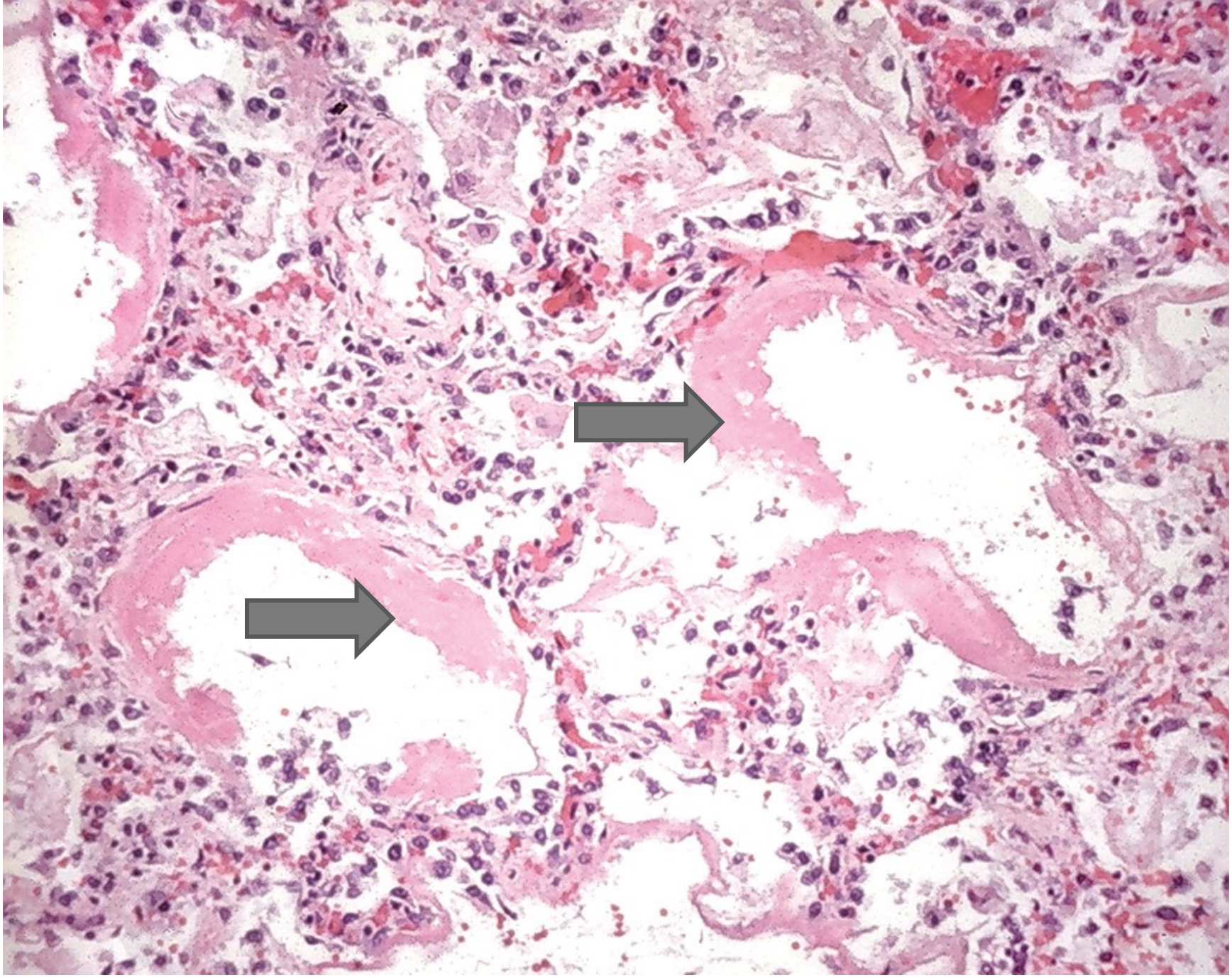


FIGURE 13.3A,ROBBINS BASIC PATHOLOGY, 10TH EDITION

HISTOLOGY:

In the organizing stage:

- **proliferation of type ll pneumocytes**
- **intraalveolar fibrosis** due to organization of the fibrin-rich exudates.
- **Marked thickening of the alveolar septa due to proliferation of interstitial cells and collagen deposition**.

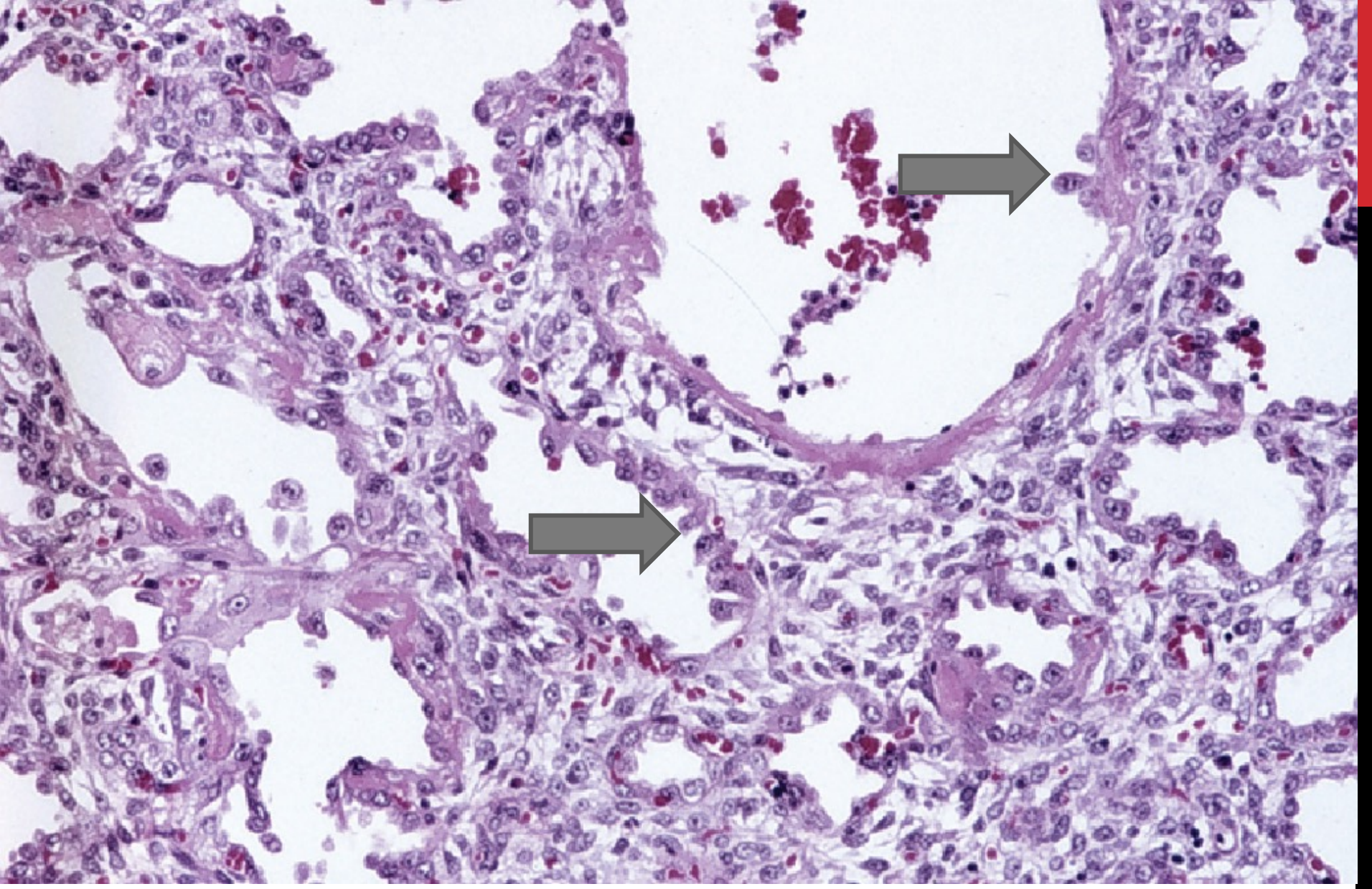


FIGURE 13.3B,ROBBINS BASIC PATHOLOGY, 10TH EDITION

CLINICAL FEATURES

- Patients are hospitalized for one of the predisposing conditions
- Profound *dyspnea and tachypnea* followed by increasing *cyanosis and hypoxemia*, *respiratory failure*, and the appearance of *diffuse bilateral infiltrates* on radiographic examination.
- Hypoxemia may be refractory to oxygen therapy due to ventilation-perfusion mismatch, and respiratory acidosis can develop.
- Early in the course, the lungs become stiff due to loss of functional surfactant, leading to the need for intubation and high ventilatory pressures to maintain adequate gas exchange.

OUTCOME:

- The overall hospital mortality rate is 38.5%.
- The majority of deaths are attributable to sepsis, multiorgan failure, or severe lung injury.
- Most patients who survive the acute insult recover normal respiratory function within 6 to 12 months, but the rest have lung damage resulting in interstitial fibrosis and chronic pulmonary disease.

PREDICTORS OF POOR PROGNOSIS

1. advanced age
2. bacteremia (sepsis)
3. development of multiorgan failure

OBSTRUCTIVE VS. RESTRICTIVE

DIFFUSE PULMONARY DISEASES can be classified into two Categories:

1- **OBSTRUCTIVE AIRWAY DISEASES**: characterized by an increase in resistance to airflow caused by partial or complete obstruction at any level

2- **RESTRICTIVE DISEASES**: characterized by reduced expansion of lung parenchyma and decreased total lung capacity.

Restrictive defects occur in two general conditions:

1. **chest wall disorders in the presence of normal lungs:**
   - severe obesity, diseases of the pleura, and neuromuscular disorders that affect the respiratory muscles

2. **acute or chronic interstitial lung diseases**:
   - The classic **acute** restrictive disease is **ARDS**.
   - **Chronic restrictive diseases** include the **pneumoconioses, interstitial fibrosis of unknown etiology, and sarcoidosis**.

https://baytowncrimestoppers.com/baytowns-most-wanted/unsolved-crimes/

A 58-year-old man with ischemic heart disease undergoes coronary artery bypass graft surgery under general anesthesia. Two days postoperatively, he experiences increasing respiratory difficulty with decreasing arterial oxygen saturation. On physical examination, his heart rate is regular at 78/min, respirations are 25/min, and blood pressure is 135/85 mmHg. The hemoglobin concentration has remained unchanged, at 13.7 g/dL, since surgery. After he coughs up a large amount of mucoid sputum, his condition improves. Which of the following types of atelectasis does he most likely have?

A) Compression

B) Contraction

C) Resorption

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https://www.pennlive.com/news/2019/03/cold-case-findings-generating-news-throughout-pa.html

THANK YOU!