Acute Respiratory Distress Syndrome (ARDS)

Sotiris Papaspyros

Definition

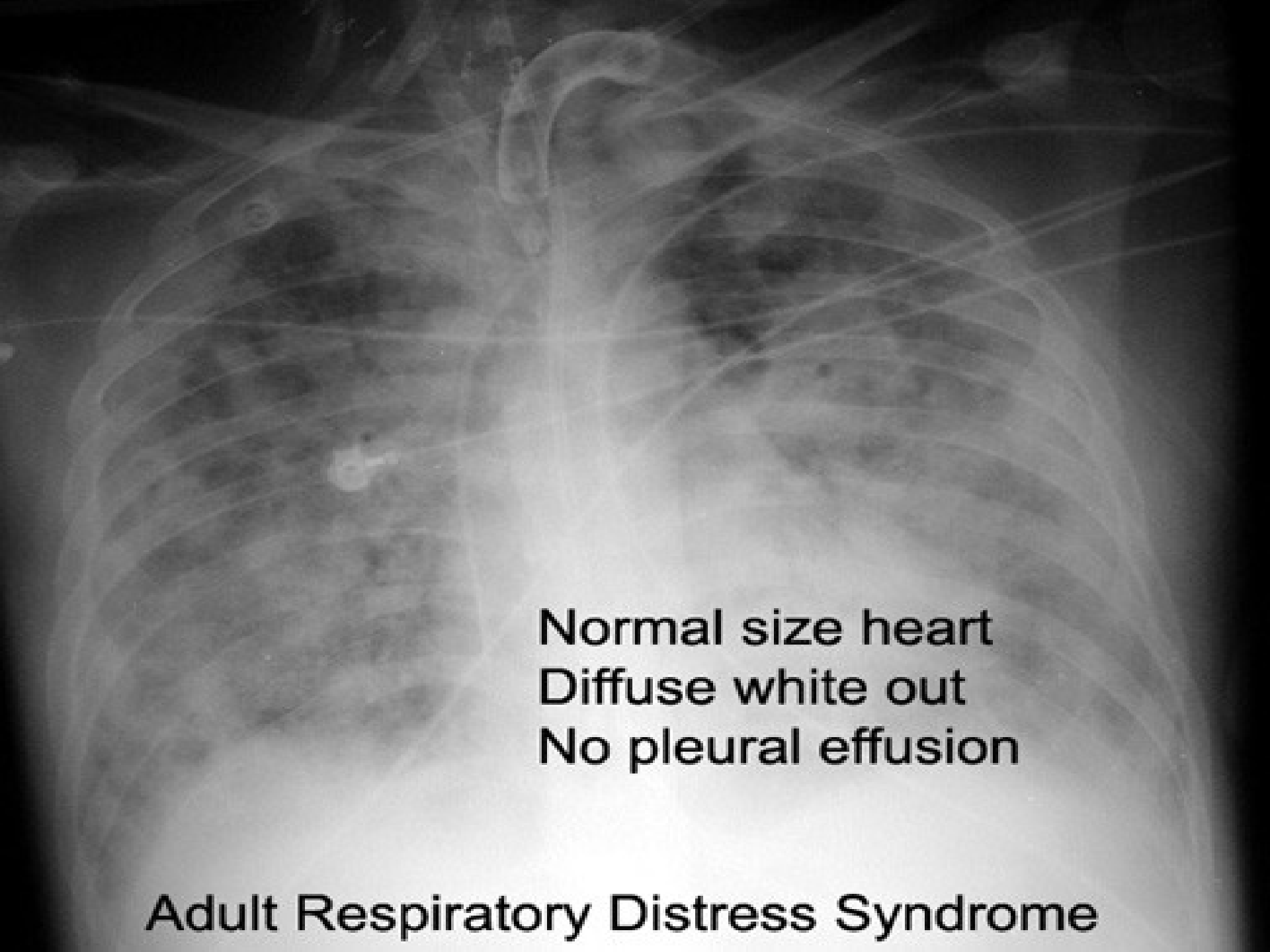
Syndrome of **acute respiratory failure** with

non cardiogenic pulmonary oedema, leading to:

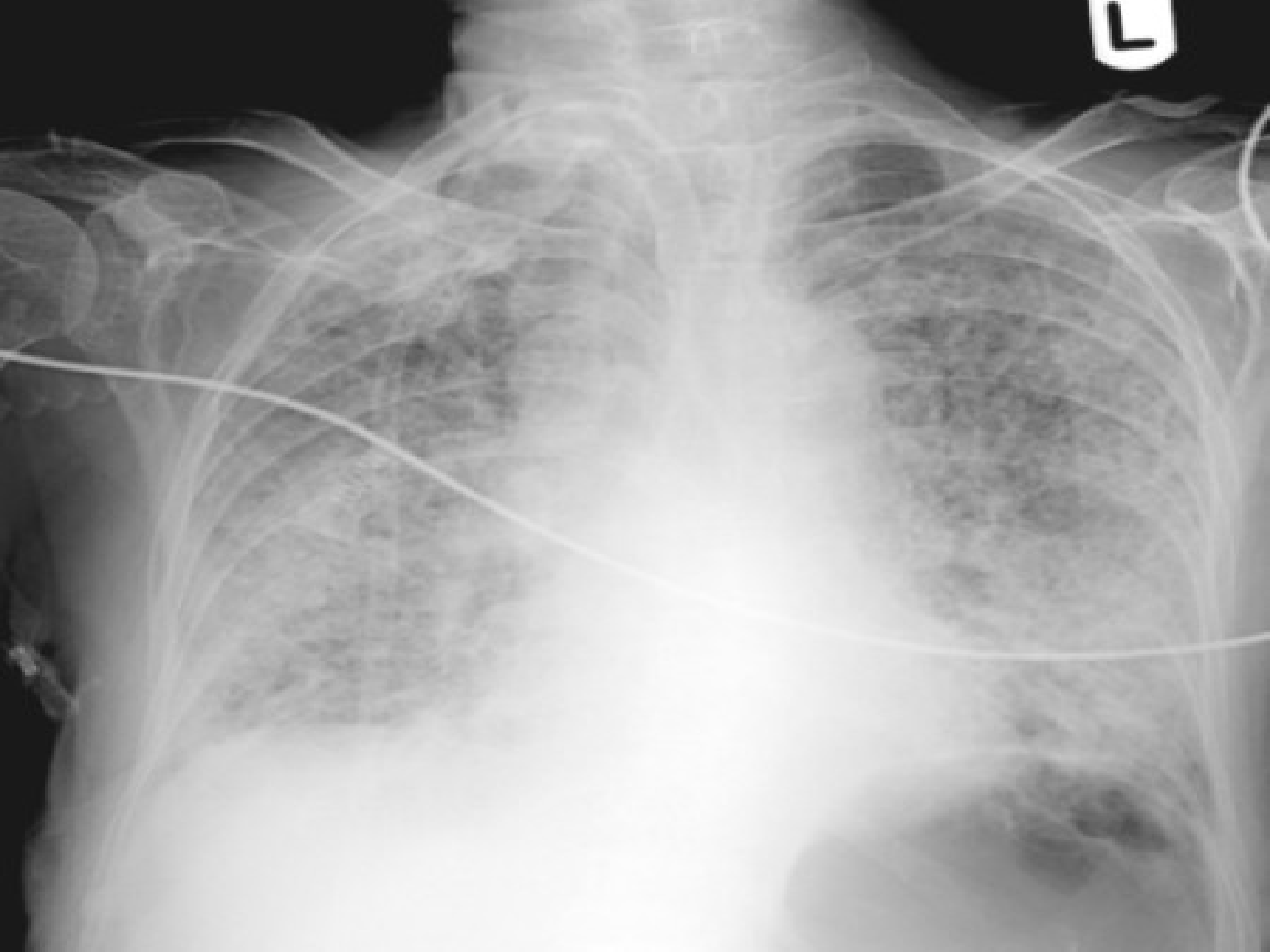
reduced **compliance** and **hypoxaemia**, **refractory to O2 therapy.**

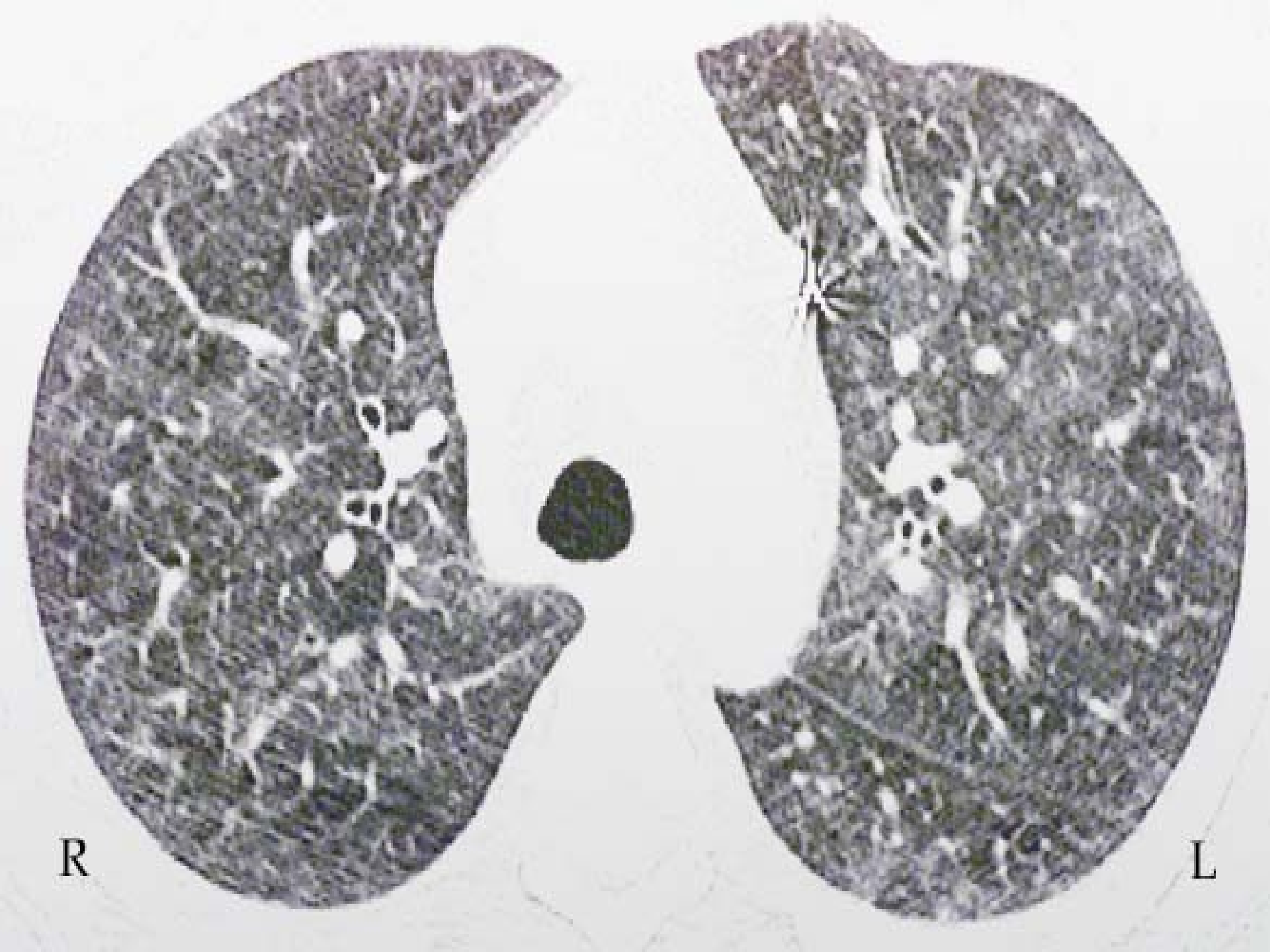
Definition

1. Diffuse bilateral infiltrates on CXR
2. PAWP <18mmHg (non cardiogenic)
3. If PaO2:FiO2 < 300 acute lung injury (ALI) is considered to be present.
4. If PaO2:FiO2 < 200 acute respiratory distress syndrome (ARDS) is considered to be present



Adult Respiratory Distress Syndrome


L


R
L

Aetiology

1. Pulmonary.
   - Pneumonia
   - Aspiration
   - Fat Embolism (long bone fractures)
   - Burns (smoke inhalation)
   - Trauma (lung contusion)

2. SIRS.
   - Trauma (surgery)
   - Pancreatitis
   - Sepsis

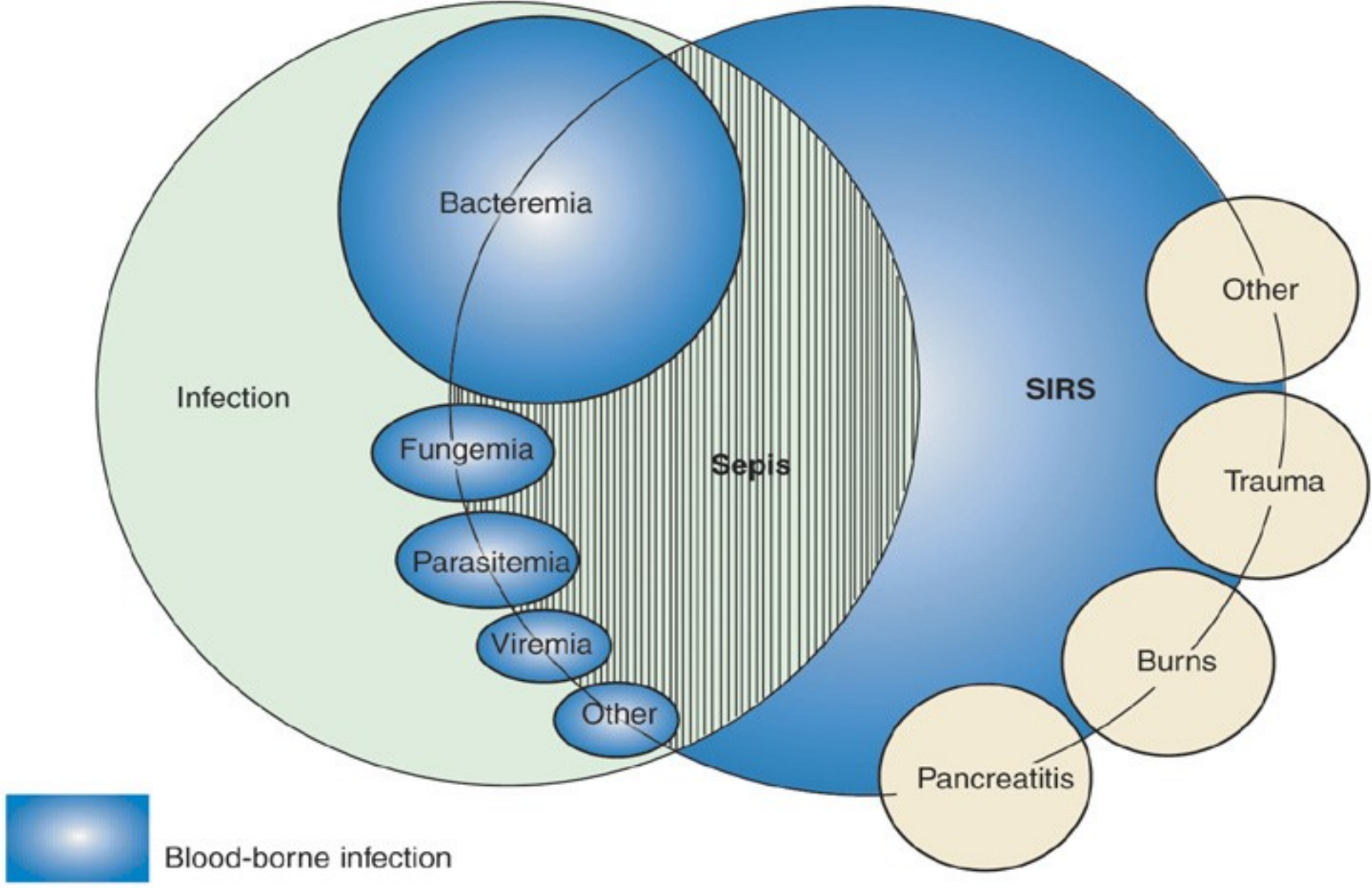


Figure 54-7 The interrelationship between systemic inflammatory response syndrome (SIRS), sepsis, and infection.

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Aetiology

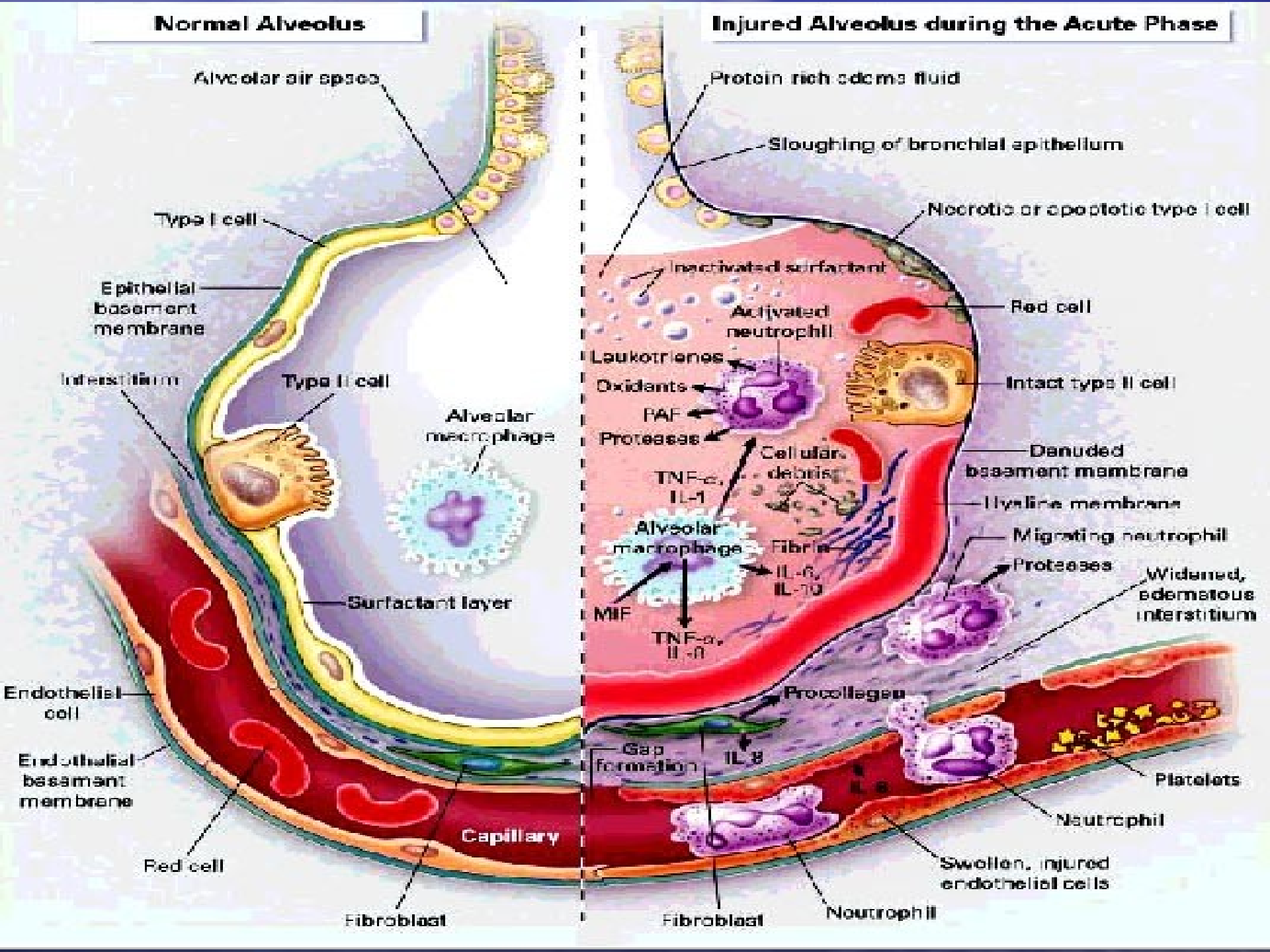
3. CPB,
4. Massive transfusion,
5. Drug overdose,
6. Near drowning.

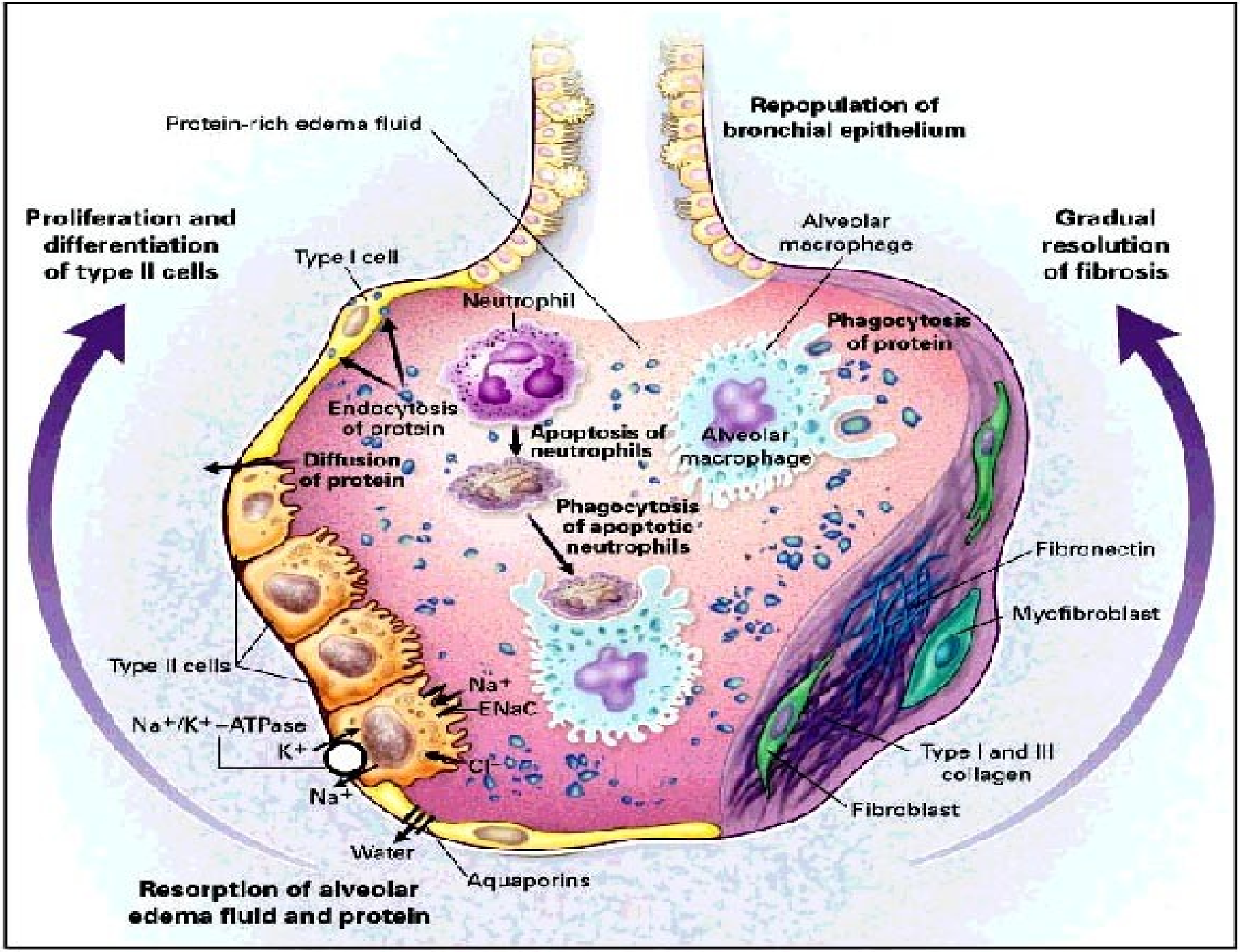
Pathophysiology

1. Initiating stimulus
2. Host response
3. Iatrogenic damage

Pathophysiology

1. **Exudative phase.(day1-7)**
   - Activation of neutrophils, macrophages, complement, coagulation cascades
   - Increase capillary permeability
   - Type 2 pneumocyte injury (surfactant)
2. **Proliferative phase.(day 7-)**
   - Organisation & repair
   - Type 2 pneumocyte proliferation
3. **Interstitial fibrosis (3 weeks-)**

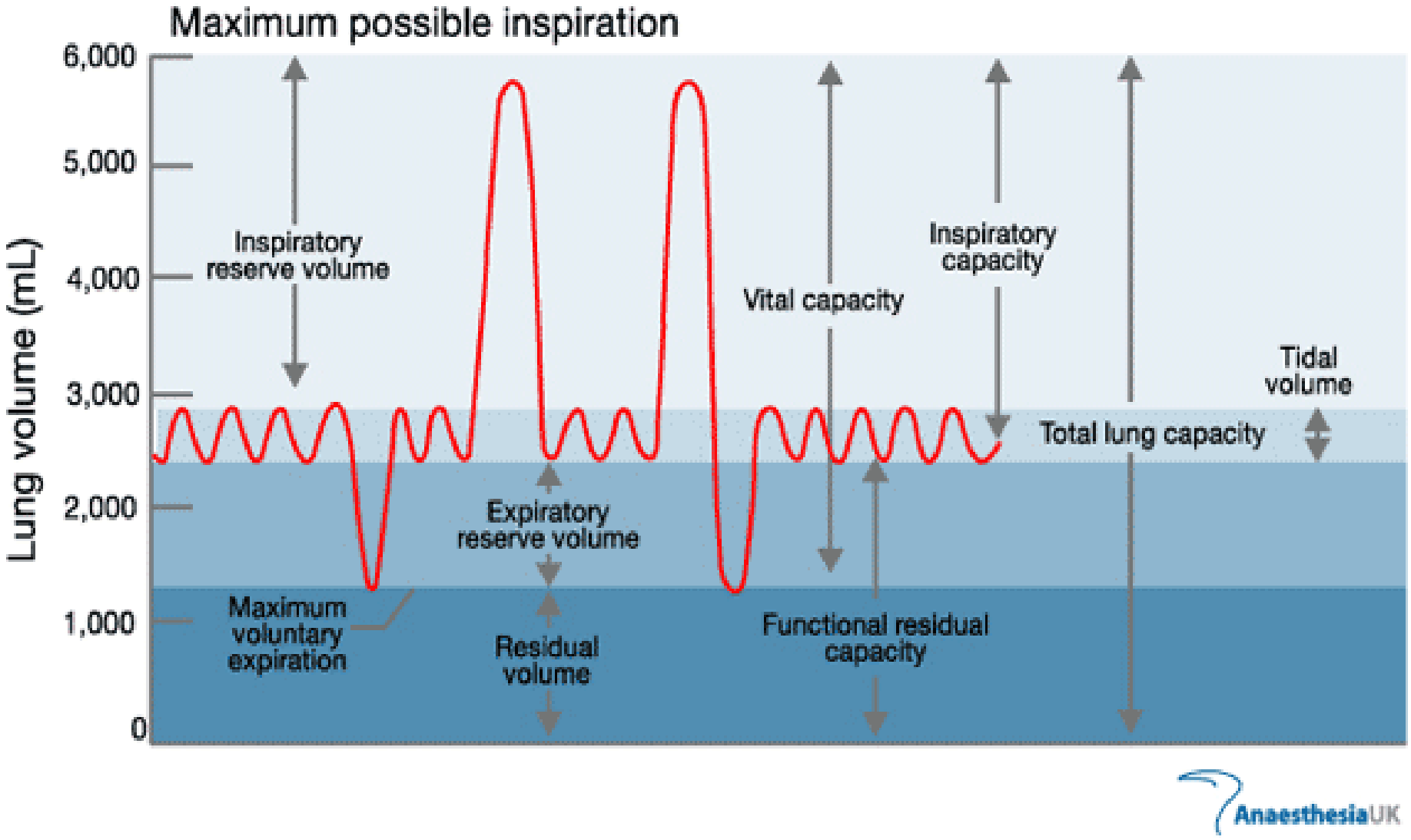

Normal Alveolus
Alveolar air space
Type I cell
Epithelial basement membrane
Interstitium
Type II cell
Alveolar macrophage
Surfactant layer
Endothelial cell
Endothelial basement membrane
Capillary
Red cell
Fibroblast
Injured Alveolus during the Acute Phase
Protein rich edema fluid
Sloughing of bronchial epithelium
Necrotic or apoptotic type I cell
Inactivated surfactant
Activated neutrophil
Red cell
Leukotrienes
Oxidants
PAF
Proteases
Intact type II cell
Cellular debris
TNF-α, IL-1
Denuded basement membrane
Hyaline membrane
Alveolar macrophage
Fibrin
Migrating neutrophil
IL-8, IL-10
Proteases
MIF
TNF-α, IL-8
Widened, edematous interstitium
Procollagen
Gap formation
IL-8
Platelets
Neutrophil
Swollen, injured endothelial cells
Fibroblast
Neutrophil


Protein-rich edema fluid
Repopulation of bronchial epithelium
Proliferation and differentiation of type II cells
Gradual resolution of fibrosis
Type I cell
Alveolar macrophage
Neutrophil
Phagocytosis of protein
Endocytosis of protein
Apoptosis of neutrophils
Alveolar macrophage
Diffusion of protein
Phagocytosis of apoptotic neutrophils
Fibronectin
Myofibroblast
Type II cells
Na+
ENaC
Na+/K+–ATPase
K+
Cl–
Type I and III collagen
Na+
Fibroblast
Water
Aquaporins
Resorption of alveolar edema fluid and protein

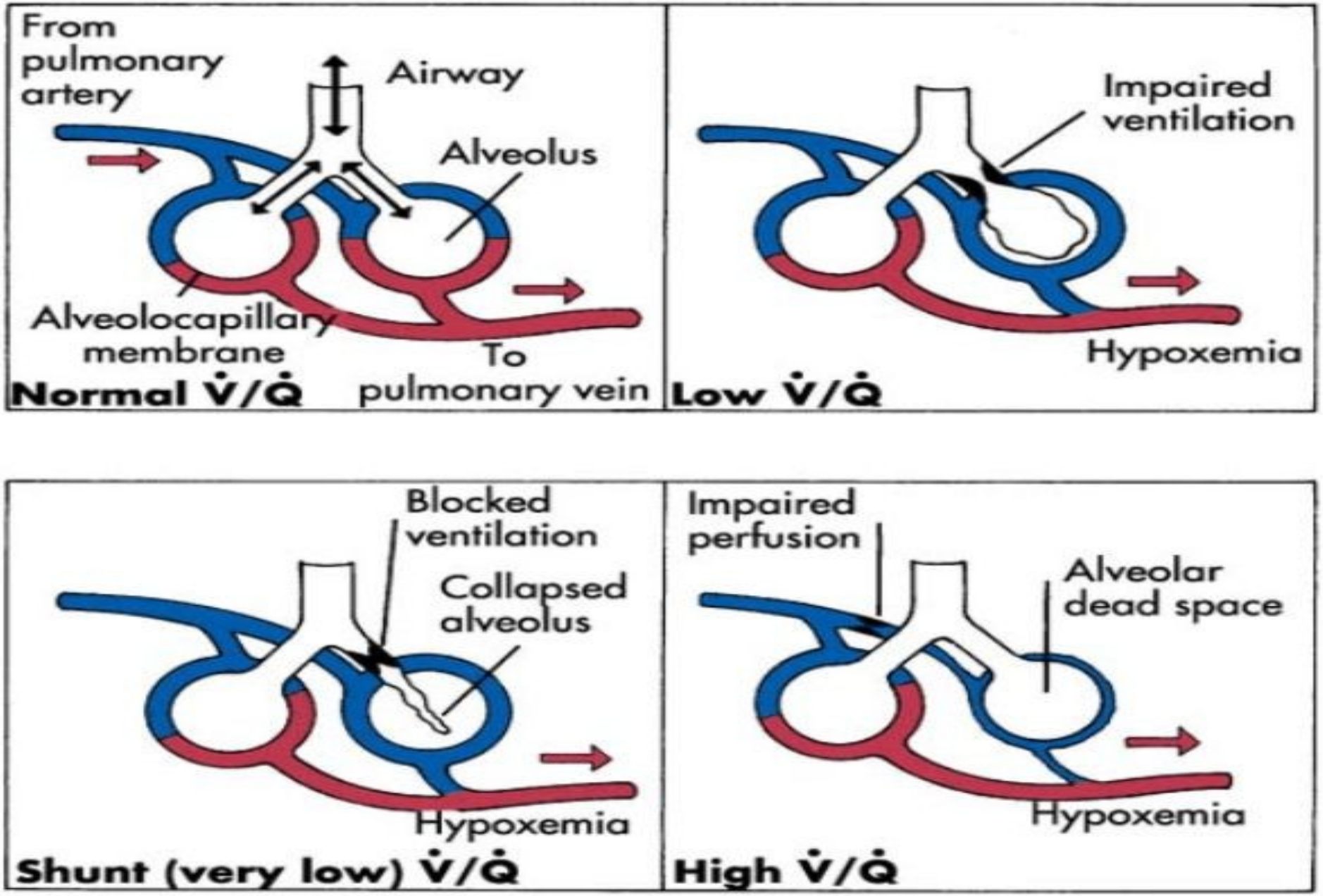
Physiological consequences summary

- ↓FRC
- V/Q Mismatch
- ↓Compliance (↓ surfactant)
- ↑PVR (Hypoxia)→↑RV work
- Absorption atelectasis. (Iatrogenic, high FiO2)

Lung Volumes and Capacities

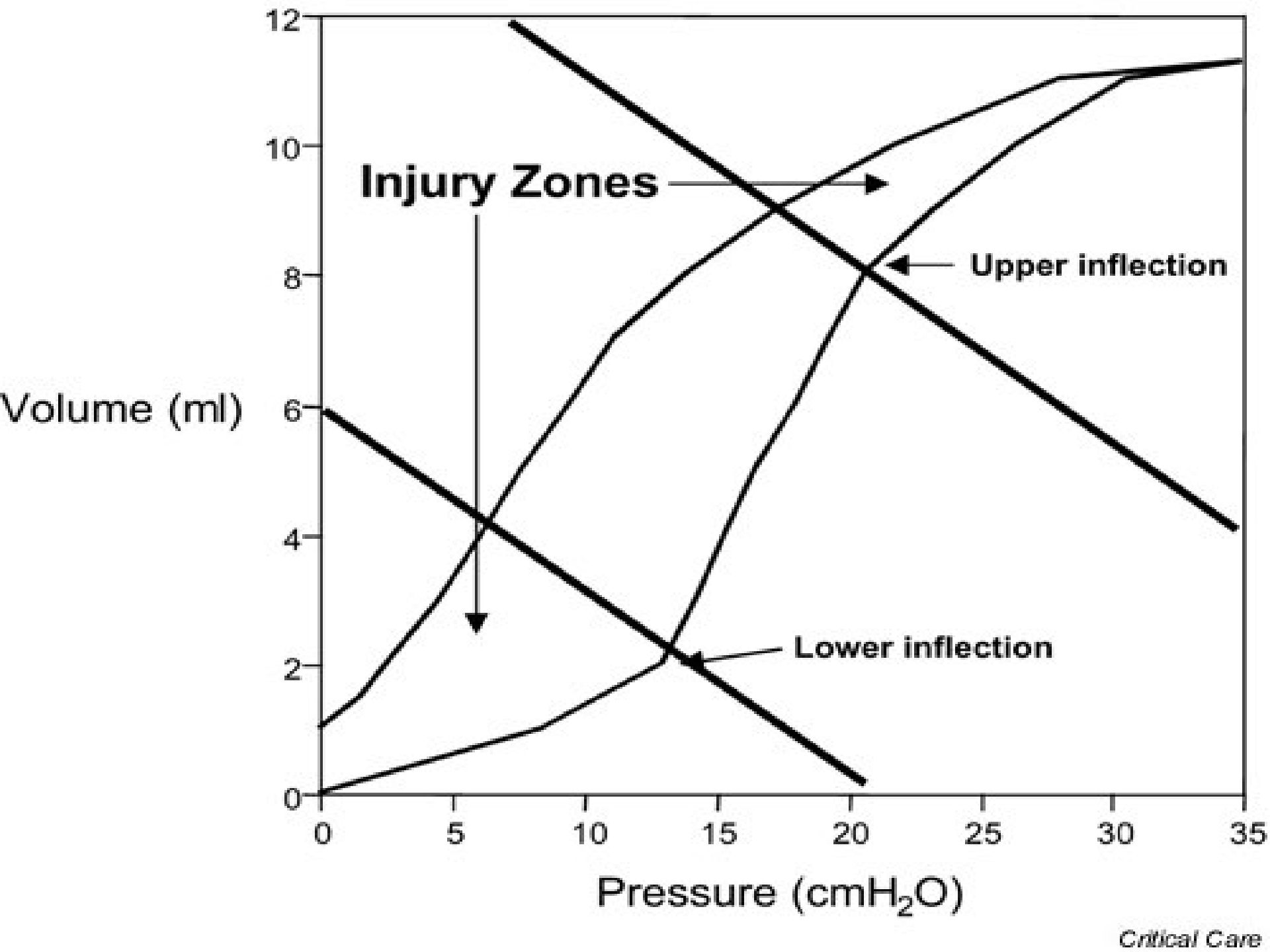


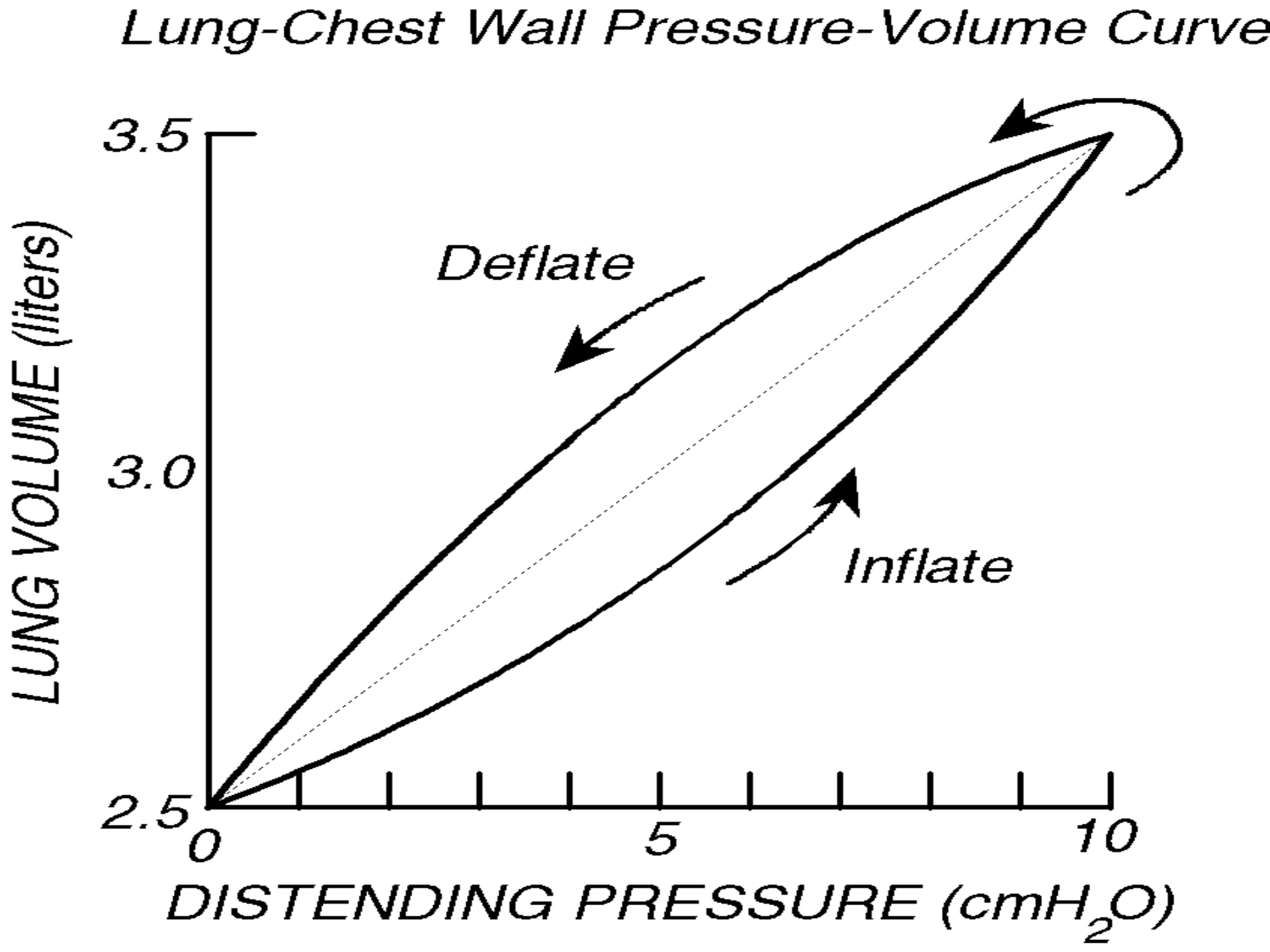
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V/Q MISMATCH

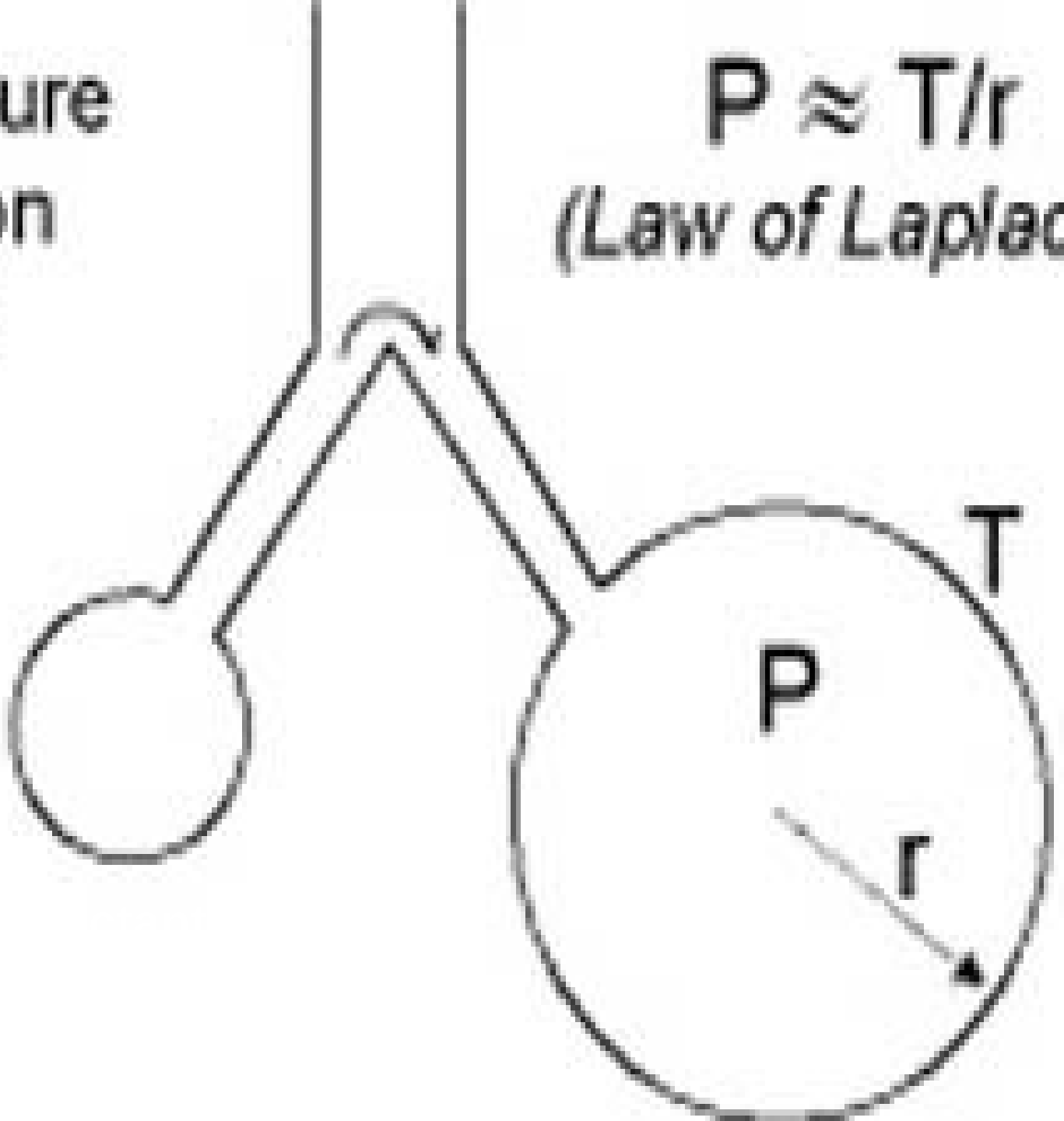

Injury Zones
Upper inflection
Lower inflection
Volume (ml)
Pressure (cmH2O)
0
2
4
6
8
10
12
0
5
10
15
20
25
30
35
Critical Care


Lung-Chest Wall Pressure-Volume Curve
3.5
3.0
2.5
LUNG VOLUME (liters)
Deflate
Inflate
0
5
10
DISTENDING PRESSURE (cmH2O)

P = pressure
T = tension
r = radius

$P \approx T/r$

(Law of Laplace)



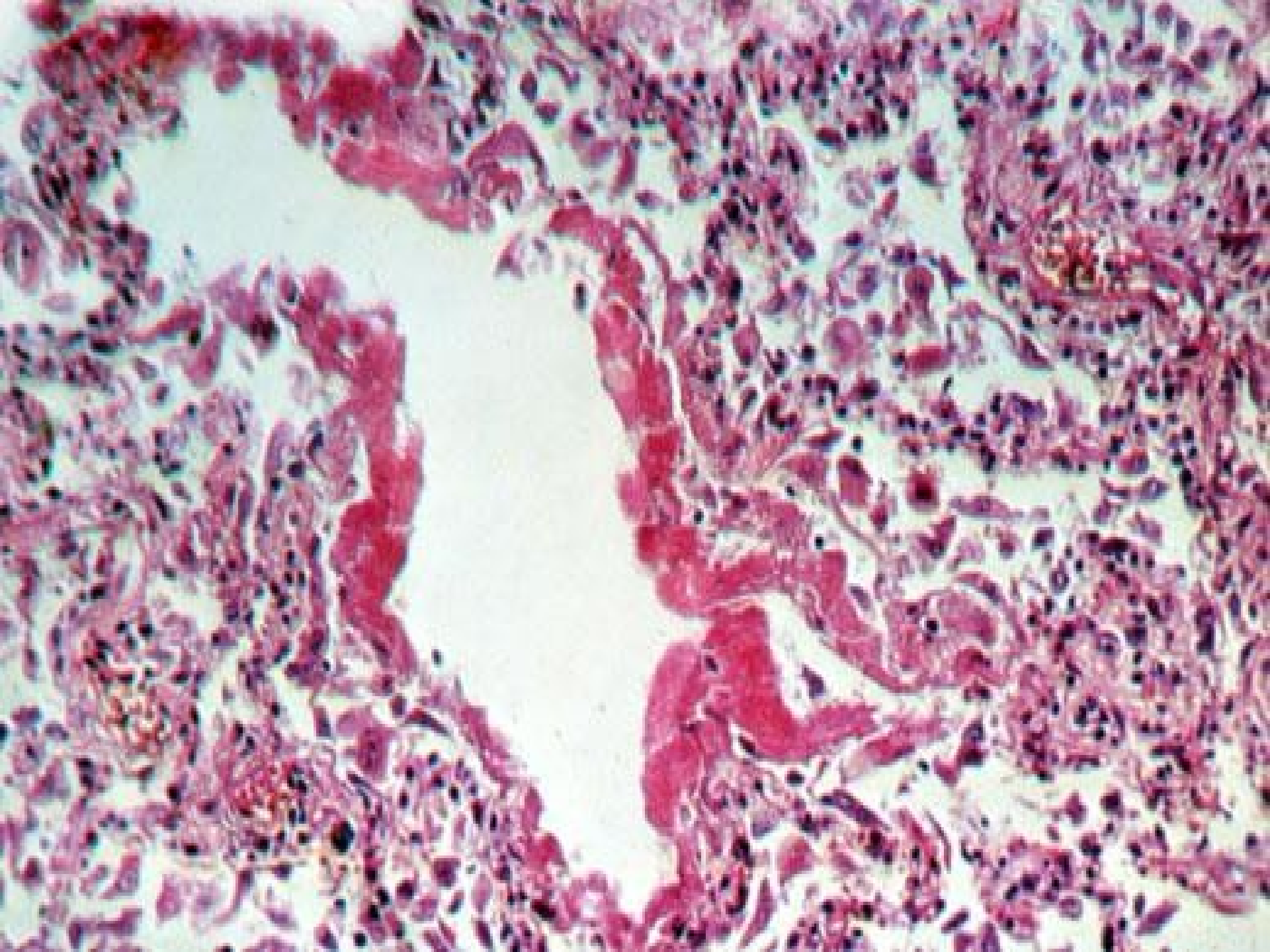
PVR

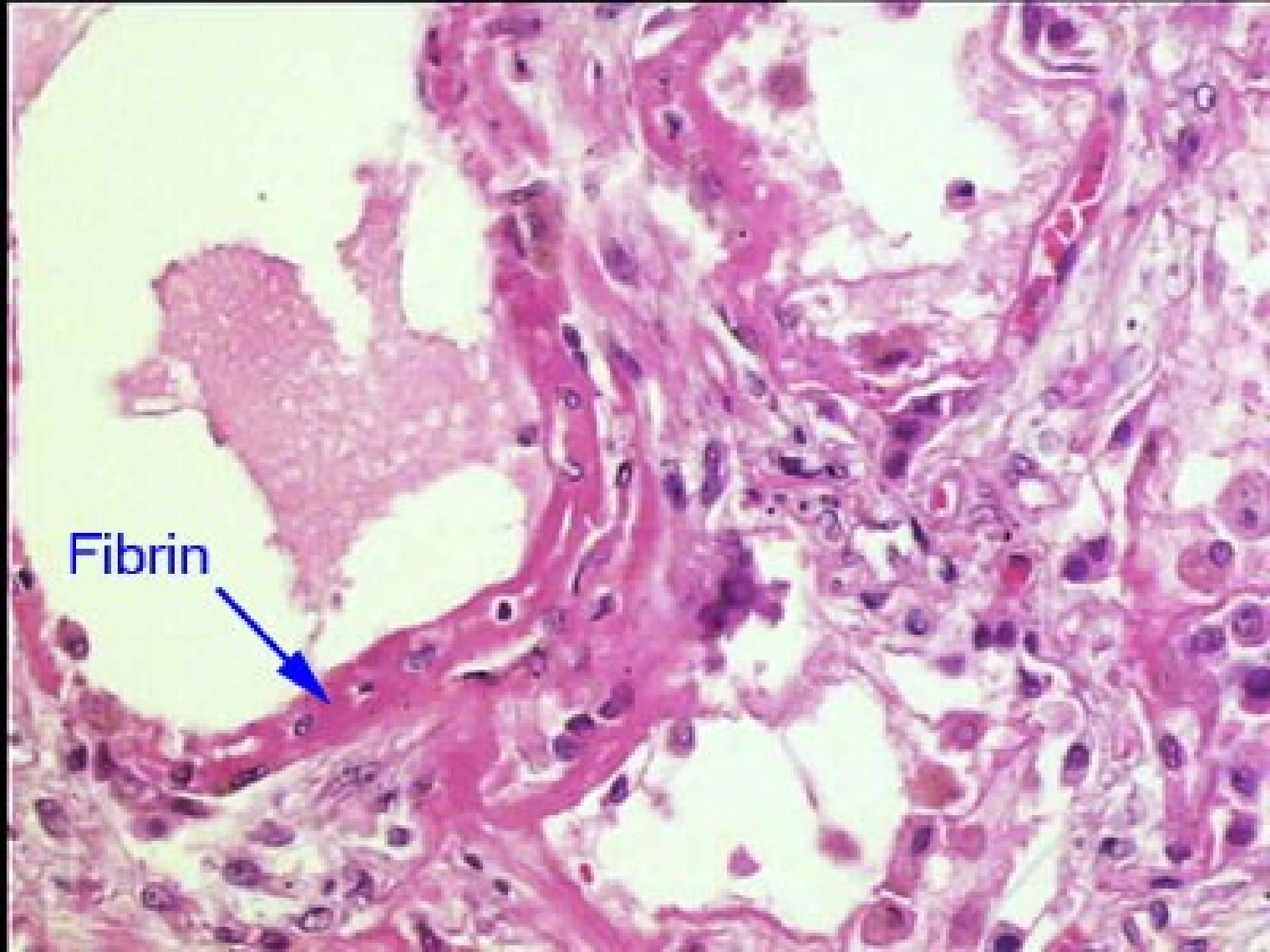
- Hypoxia
- Hypercarbia
- Thrombosis

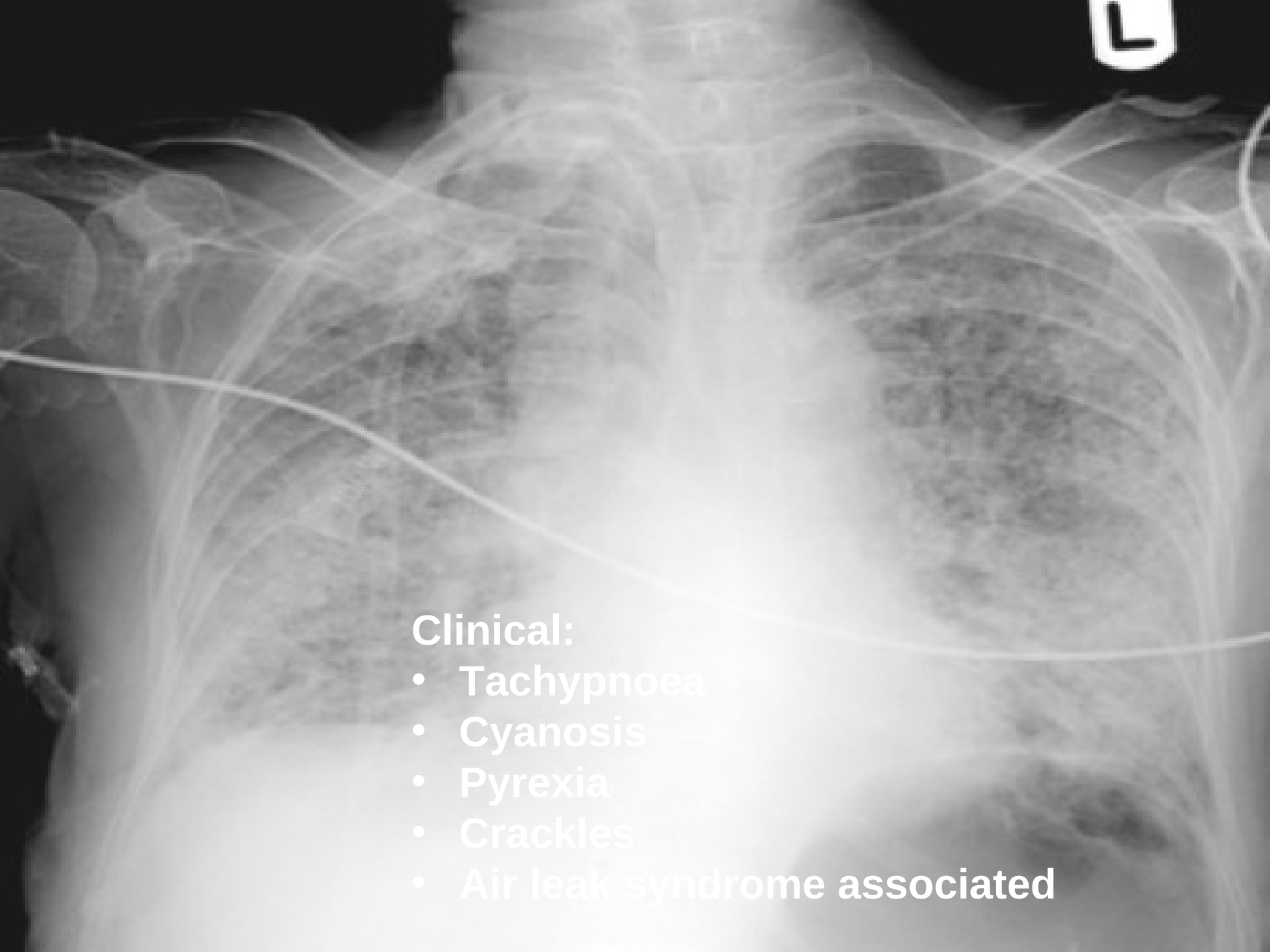
↑PVR
(↓PRELOAD,↓SV)
(CO=HR x SV)

↓CO
(HYPOXIA)

Absorption atelectasis




Fibrin



Clinical:

- **Tachypnoea**
- **Cyanosis**
- **Pyrexia**
- **Crackles**
- **Air leak syndrome associated**

INVESTIGATIONS

- FBC (↑WBC, ↓PLT)
- ABGs (↓PaO2, ↑↓PaCO2,↓pH)
- CXR
  - No Cardiomegaly
  - Rarely Kerley lines
  - Air leak syndromes
- CT:
  - researchers have shown a marked spatial distribution of parenchymal involvement.
  - In patients ventilated supine collapse was more pronounced dorsally. (Role of prone ventilation).

Management strategies

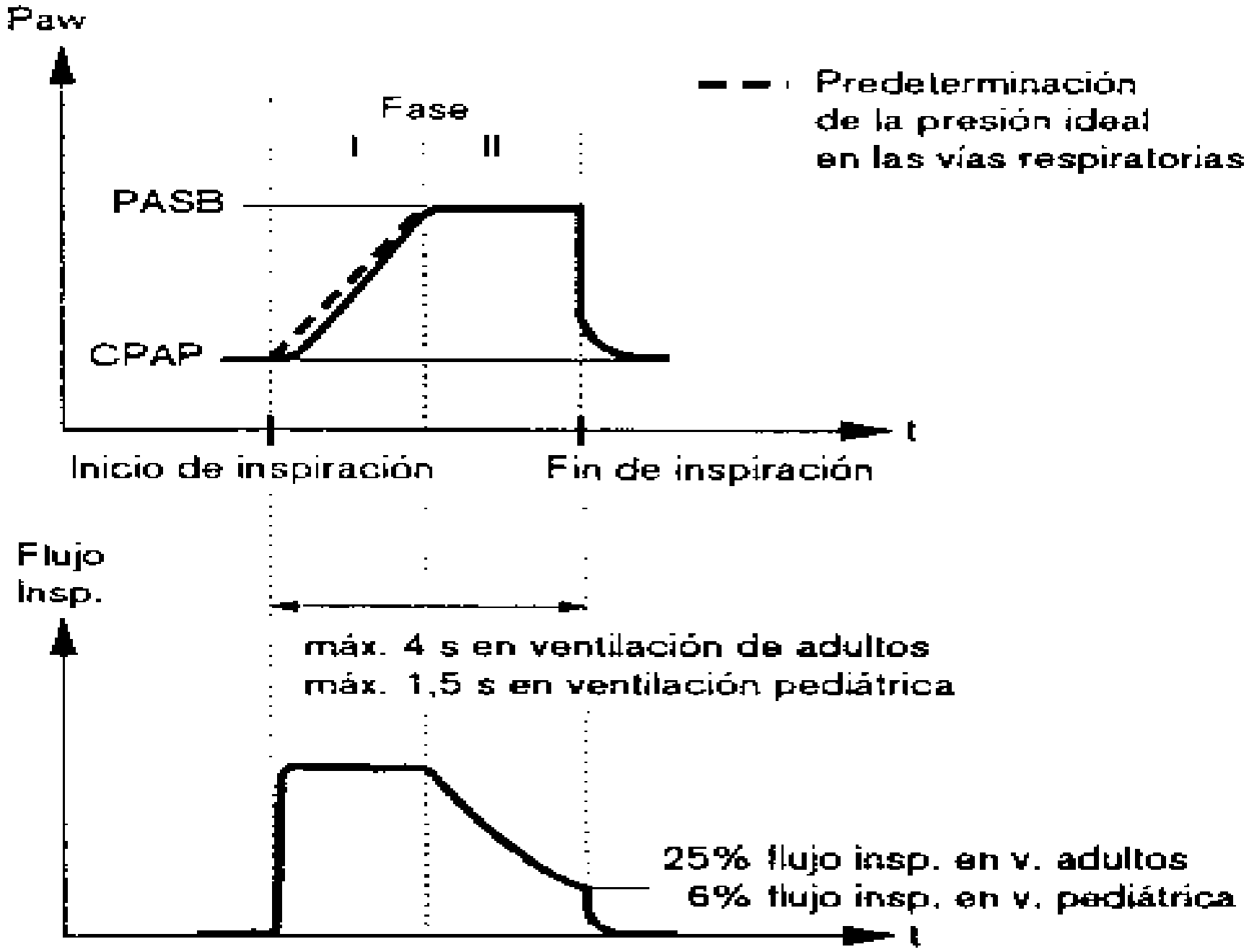
1. Treat initial insult.
2. Ventilation
   - PEEP
   - I:E ratio 1:1
   - Prone ventilation
   - Small TV (permissive hypercapnia)
   - NO
3. Nutritional support
4. Fluid balance (prevent cardiogenic pulmonary oedema)

General

- Acute respiratory distress syndrome is usually treated with mechanical ventilation in the Intensive Care Unit. Ventilation is usually delivered through oro-tracheal intubation, or tracheostomy whenever prolonged ventilation (≥2 weeks) is deemed inevitable.
- Appropriate antibiotic therapy must be administered as soon as microbiological culture results are available. Empirical therapy *may* be appropriate if local microbiological surveillance is efficient. More than 60% ARDS patients experience a (nosocomial) pulmonary infection either before or after the onset of lung injury.
- The origin of infection, when surgically treatable, must be operated on. When sepsis is diagnosed, appropriate local protocols should be enacted.

Mechanical Ventilation

- Conventional therapy aimed at tidal volumes (*V*t) of 12-15 ml/kg. Recent studies have shown that high tidal volumes can overstretch alveoli resulting in volutrauma (secondary lung injury).
- The National Institutes of Health (NIH) multicentre study showed improved mortality when ventilated with a tidal volume of 6 ml/kg compared to the traditional 12 ml/kg. Low tidal volumes (*V*t) may cause hypercapnia and atelectasis.
- Ventilation with low *V*t may improve survival in ARDS, although the real goal seems to be a plateau pressure = 32 mmHg (4.3 kPa).
- To date, further reductions in pressures have not yielded significant benefits, probably because of the associated, more severe respiratory acidosis, caused by permissive hypercapnia.


Paw
Predeterminación
de la presión ideal
en las vías respiratorias
Fase
I
II
PASB
CPAP
t
Inicio de inspiración
Fin de inspiración
Flujo
insp.
máx. 4 s en ventilación de adultos
máx. 1,5 s en ventilación pediátrica
25% flujo insp. en v. adultos
6% flujo insp. en v. pediátrica
t

Steroids

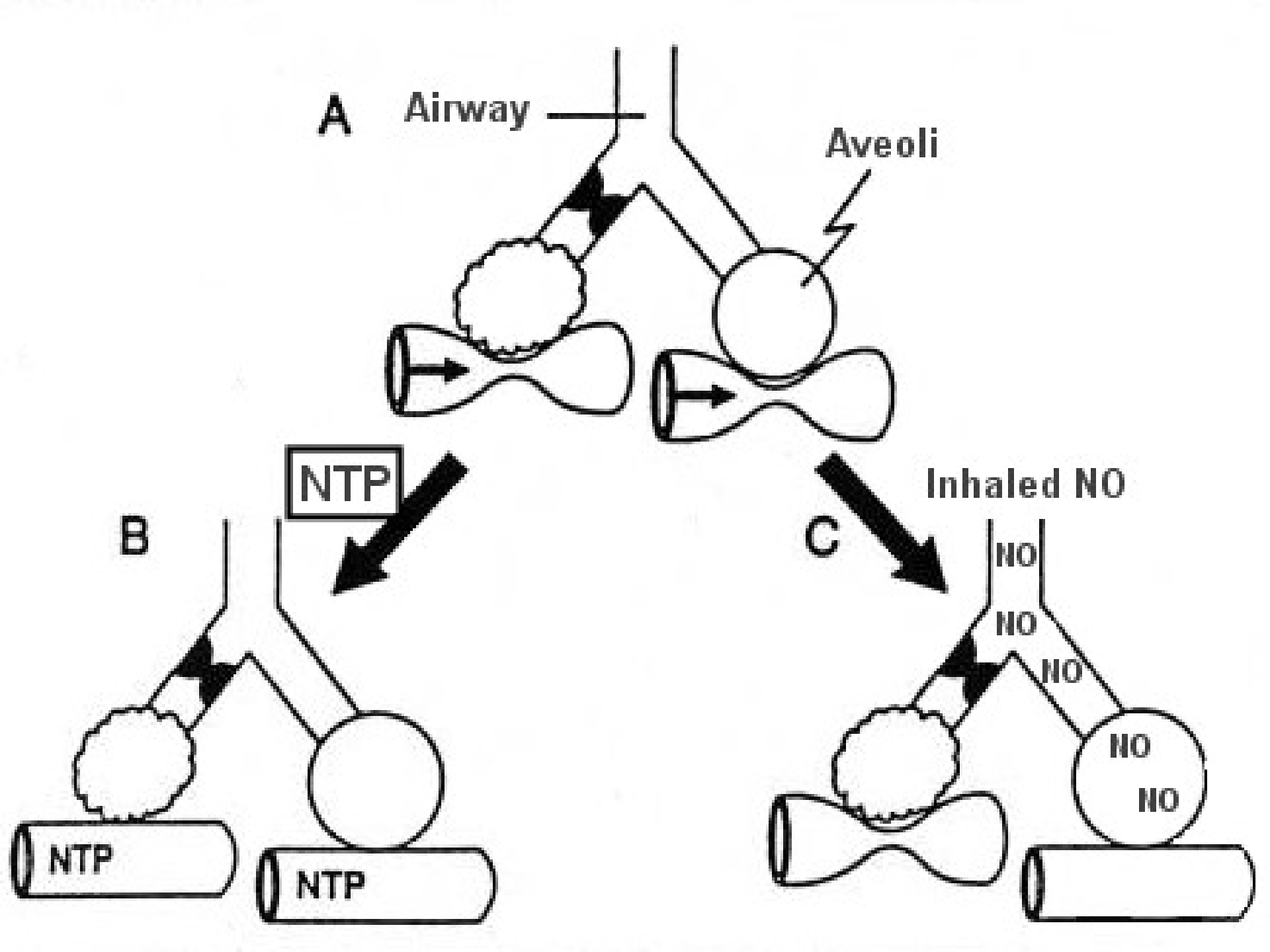
- A number of published trials showed **no benefit** from **high dose** corticosteroids.
- Meduri et al however did find significant improvement using modest doses. This is probably because of a suppression of ongoing inflammation during the fibroproliferative phase of ARDS.

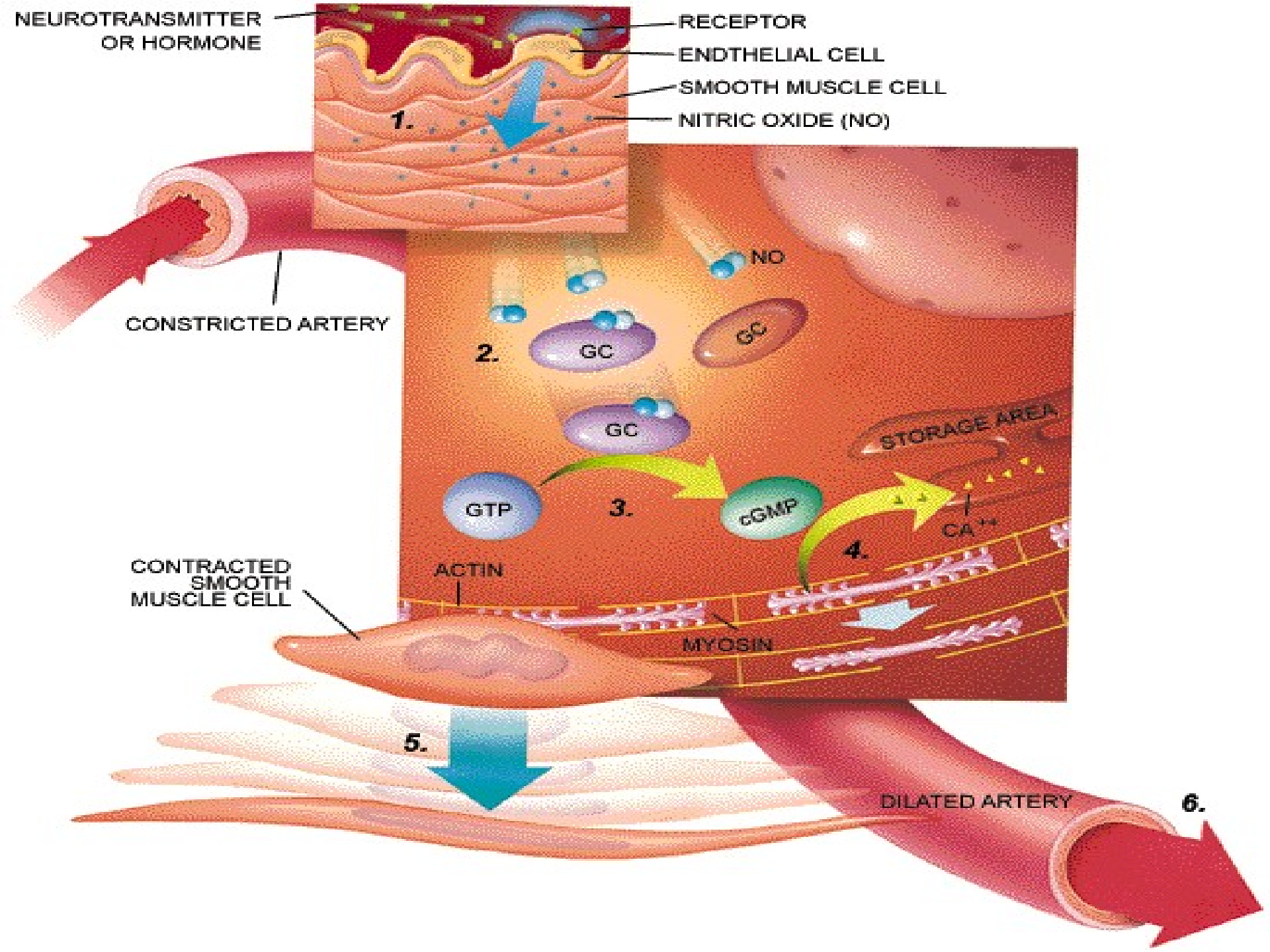
Surfactant

- To date no prospective controlled clinical trial has shown a significant mortality benefit of exogenous surfactant in ARDS

Nitric oxide

- Inhaled nitric oxide (NO) potentially acts as selective pulmonary vasodilator. Rapid binding to hemoglobin prevents systemic effects. It should increase perfusion of better ventilated areas.
- There are **no large studies** demonstrating positive results. Therefore its use must be considered individually.


A
Airway
Aveoli
NTP
B
C
Inhaled NO
NO
NO
NO
NO
NO
NTP
NTP


NEUROTRANSMITTER OR HORMONE
RECEPTOR
ENDTHELIAL CELL
SMOOTH MUSCLE CELL
NITRIC OXIDE (NO)
1.
CONSTRICTED ARTERY
NO
2.
GC
GC
GC
STORAGE AREA
GTP
3.
cGMP
CA++
4.
CONTRACTED SMOOTH MUSCLE CELL
ACTIN
MYOSIN
5.
DILATED ARTERY
6.

Prone position

- Distribution of lung infiltrates in acute respiratory distress syndrome is non-uniform. Repositioning into the prone position (face down) might improve oxygenation by relieving atelectasis and improving perfusion.
- However, although the hypoxemia is overcome there seems to be **no effect on overall survival.**

Fluid management

- Several studies have shown that pulmonary function and outcome are better in patients that **lost weight** or wedge pressure was lowered by diuresis or fluid restriction.

Complications

- Pulmonary: barotrauma (volutrauma), pulmonary embolism (PE), pulmonary fibrosis, ventilator-associated pneumonia (VAP).
- Gastrointestinal: hemorrhage (ulcer), dysmotility, pneumoperitoneum, bacterial translocation.
- Cardiac: arrhytmias, myocardial dysfunction.
- Renal: acute renal failure (ARF), positive fluid balance.
- Mechanical: vascular injury, pneumothorax (by placing pulmonary artery catheter), tracheal injury/stenosis (result of intubation and/or irritation by endotracheal tube.
- Nutritional: malnutrition (katabolic state), electrolyte deficiency.

Epidemiology/Prognosis

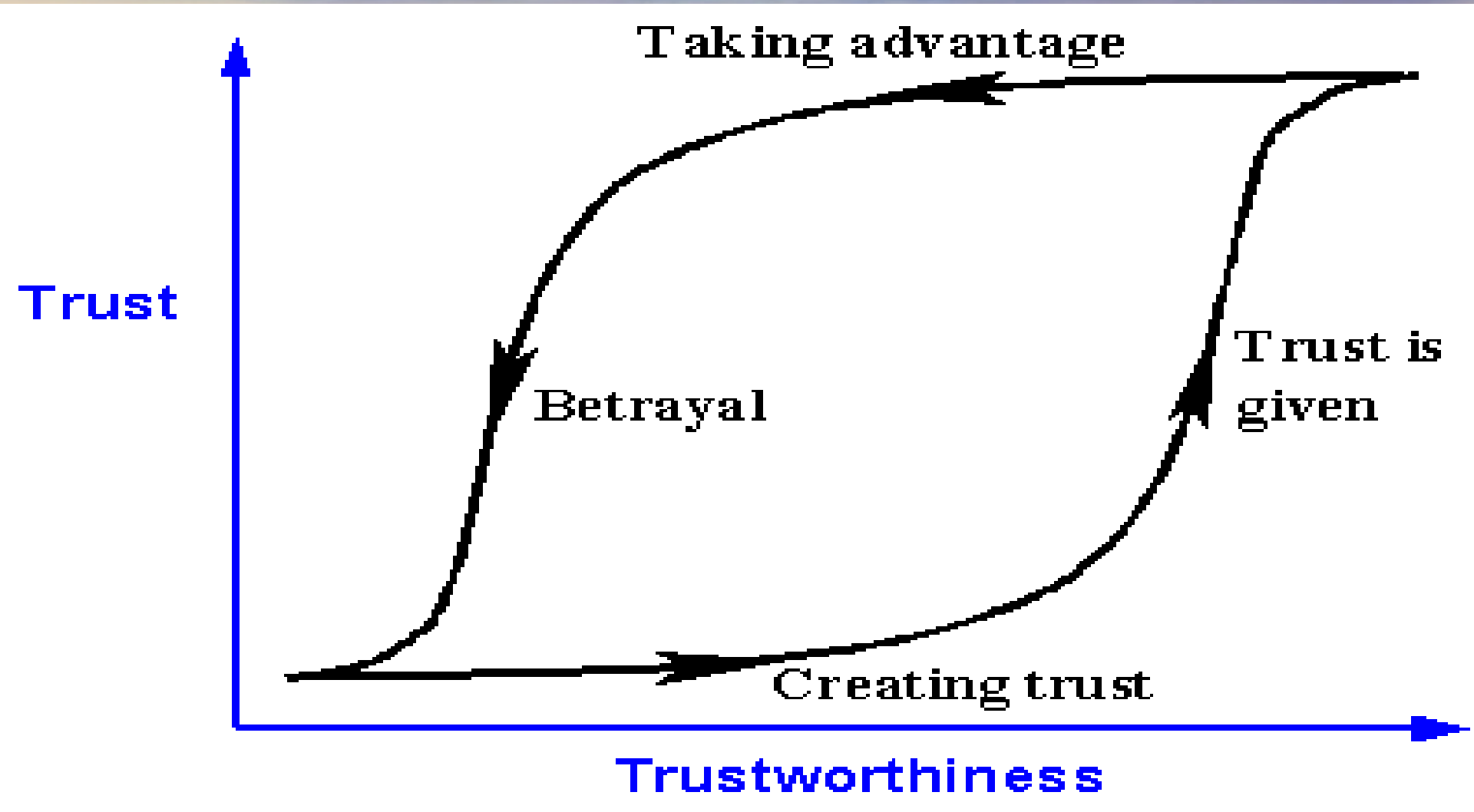
- The annual incidence of ARDS is between 1.5 to 13.5 people per 100,000 in the general population. Its incidence in the intensive care unit (ICU), mechanically ventilatedpopulation is much higher.
- Mortality varies from 30% to 60%. Usually, randomized controlled trials in the literature show lower death rates, both in control and treatment patients. This is thought to be due to stricter enrollment criteria. Observational studies generally report 50%-60% mortality.

Hysteresis

- **Hysteresis** (from Ancient Greek ὑστέρησις) is a property of systems (usually physical systems) that do not instantly follow the forces applied to them, but react slowly, or do not return completely to their original state.
- For instance, if you push on a piece of putty it will assume a new shape, and when you remove your hand it will not return to its original shape, or at least not immediately and not entirely.
- Hysteresis phenomena occur in magnetic and ferromagnetic materials, as well as in the elastic and electromagnetic behavior of materials, in which a lag occurs between the application and the removal of a force or field and its subsequent effect.

The term "hysteresis" is sometimes used in other fields, such as economics or biology. In such cases it describes a memory or lagging effect in which the order of previous events can

influence the order of subsequent events.



Thank you.