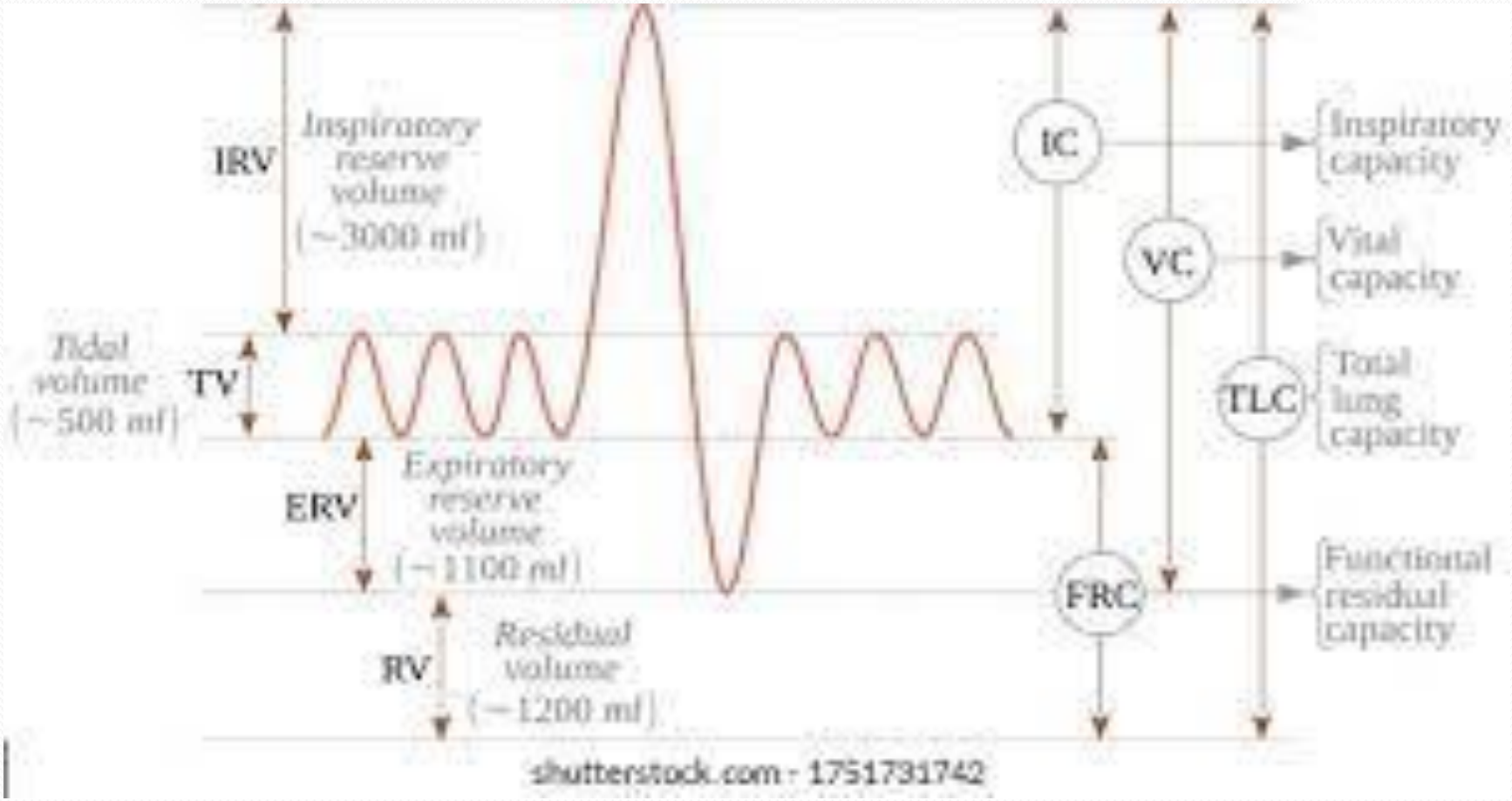
Pulmonary Function Tests

Acute Respiratory Distress Syndrome (ARDS)


IRV
Inspiratory reserve volume (~3000 ml)
Tidal volume (~500 ml)
TV
ERV
Expiratory reserve volume (~1100 ml)
RV
Residual volume (~1200 ml)
IC
Inspiratory capacity
VC
Vital capacity
TLC
Total lung capacity
FRC
Functional residual capacity
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Lung Volumes and Capacities

Generally, the values are lower for females.

The following lung volumes and capacities (a lung capacity is the sum of two or more lung volumes) can be determined

Tidal volume (TV)

The volume of air entering or leaving the lungs during a single breath.

Average value under resting conditions 500 mL.

Inspiratory reserve volume (IRV):

The extra volume of air that can be maximally inspired over and above the typical resting tidal volume.

The IRV is accomplished by maximal contraction of the diaphragm, external intercostal muscles, and accessory inspiratory muscles.

Average value 3000 mL.

Expiratory reserve volume (ERV)

The extra volume of air that can be actively expired by maximally contracting the expiratory muscles beyond that normally passively expired at the end of a typical resting tidal volume.

Average value 1000 mL.

Residual volume (RV)

The minimum volume of air remaining in the lungs even after a maximal expiration.

Average value 1200 mL.

The residual volume cannot be measured directly with a spirometer because this volume of air does not move into and out of the lungs.

It can be determined indirectly, however, through gas dilution techniques involving inspiration of a known quantity of a harmless tracer gas such as helium.

Inspiratory capacity (IC)

The maximum volume of air that can be inspired at the end of a normal quiet expiration (IC =IRV + TV).

Average value 3500 mL.

Functional residual capacity (FRC)

The volume of air in the lungs at the end of a normal passive expiration (FRC =ERV +RV).

Average value 2200 mL.

Vital capacity (VC)

The maximum volume of air that can be moved out during a single breath following a maximal inspiration.

The subject first inspires maximally and then expires maximally (VC =IRV + TV + ERV).

The VC represents the maximum volume change possible within the lungs .

It is rarely used because the maximal muscle contractions involved become exhausting, but it is valuable in determining the functional capacity of the lungs.

Average value 4500 mL.

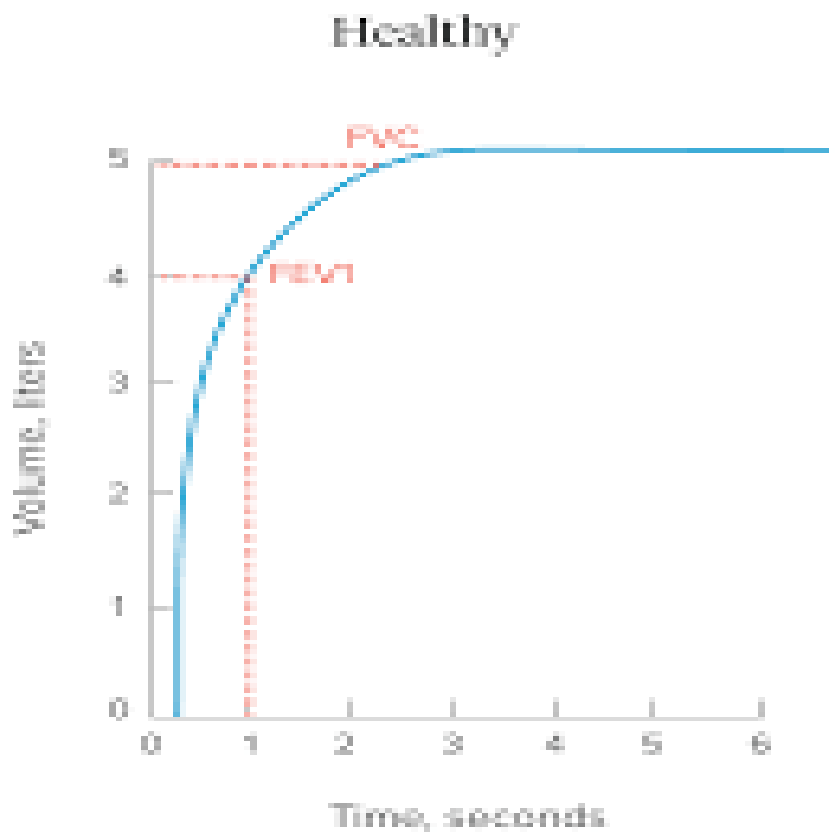
Total lung capacity (TLC)

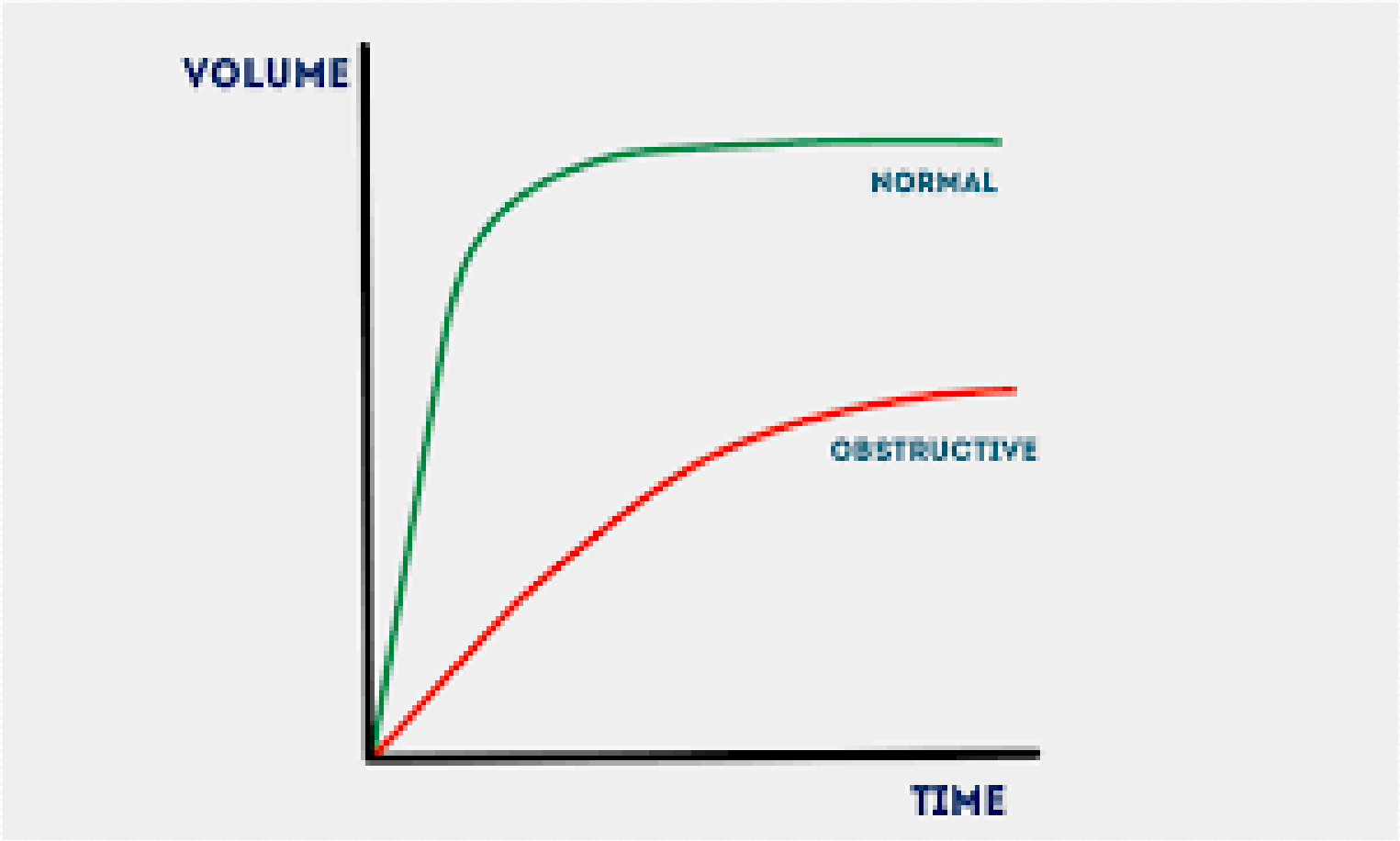
The maximum volume of air that the lungs can hold (TLC =VC +RV).

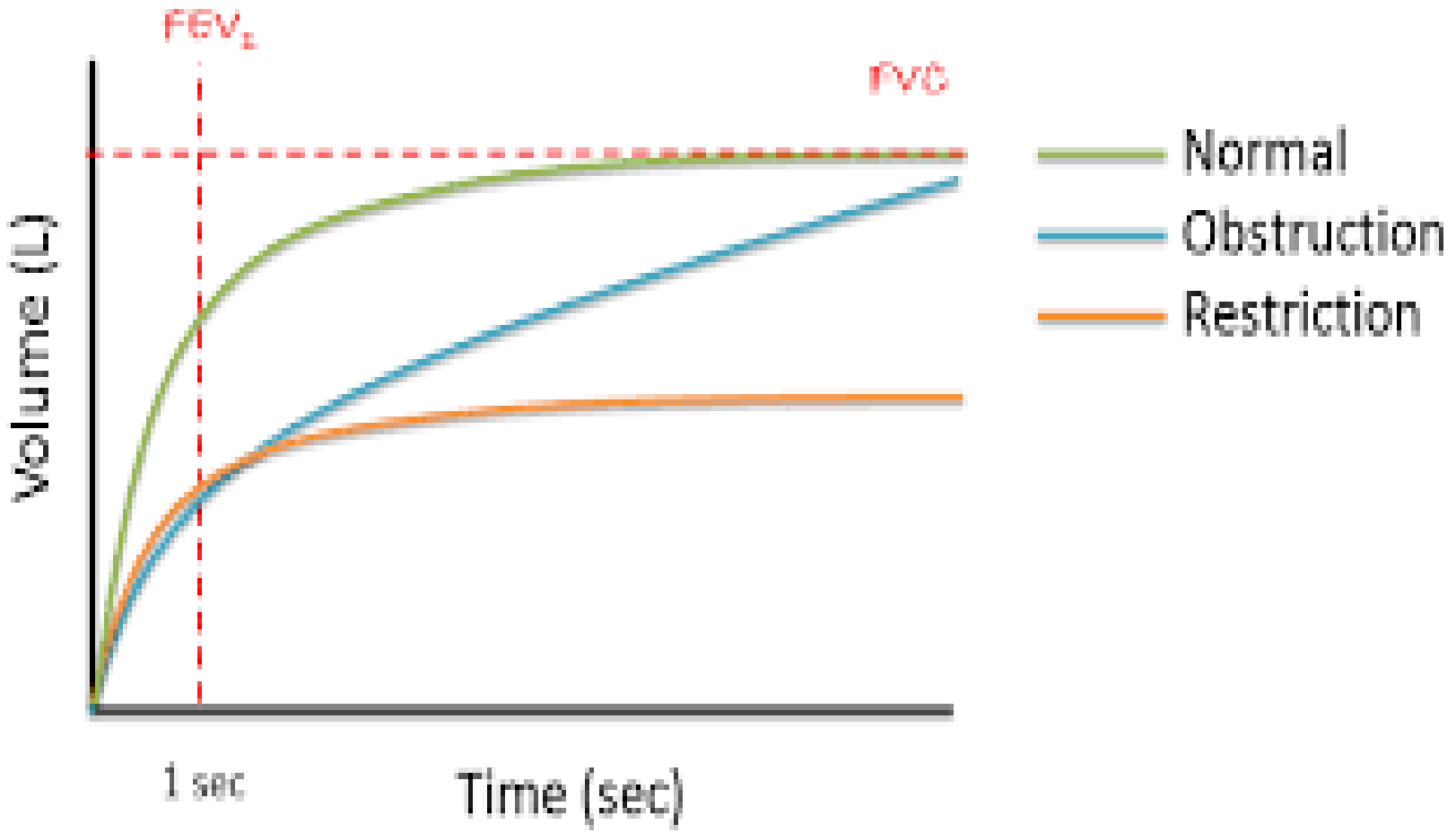
Average value 5700 mL.

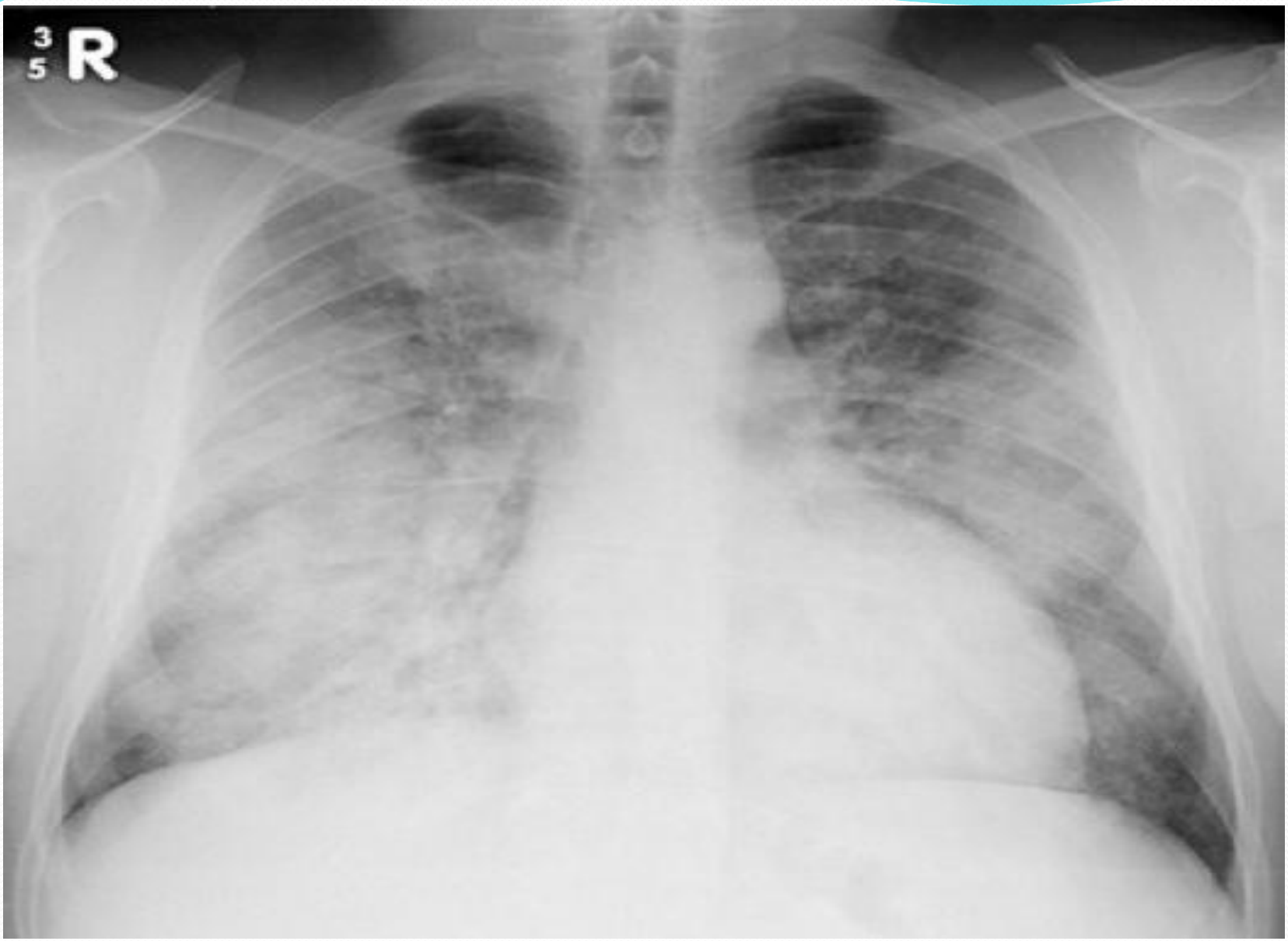
Forced expiratory volume in 1 second (FEV1)

The volume of air that can be expired during the first second of expiration


Healthy
FVC
FEV1
Volume, liters
0
1
2
3
4
5
0
1
2
3
4
5
6
Time, seconds


VOLUME
NORMAL
OBSTRUCTIVE
TIME


FEV1
FVC
Volume (L)
Normal
Obstruction
Restriction
1 sec
Time (sec)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Acute respiratory distress syndrome (ARDS)

- Is a life-threatening condition of seriously ill patients, characterized by poor oxygenation, pulmonary infiltrates, and acuity of onset.

- On a microscopic level, the disorder is associated with capillary endothelial injury and diffuse alveolar damage.

- High mortality
- Few effective therapeutic

ARDS

- An acute disorder that starts within 7 days of the inciting event and is characterized by bilateral lung infiltrates and severe progressive hypoxemia in the absence of any evidence of cardiogenic pulmonary edema.

- Defined by the patient's oxygen in arterial blood (PaO_2) to the fraction of the oxygen in the inspired air (FiO_2). These patients have a PaO_2/FiO_2 ratio of less than 300.

Table 3. The Berlin Definition of Acute Respiratory Distress Syndrome

	Acute Respiratory Distress Syndrome
Timing	Within 1 week of a known clinical insult or new or worsening respiratory symptoms
Chest imaging[a]	Bilateral opacities—not fully explained by effusions, lobar/lung collapse, or nodules
Origin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload Need objective assessment (eg, echocardiography) to exclude hydrostatic edema if no risk factor present
Oxygenation[b]	
Mild	200 mm Hg $<$ Pao_2/Fio_2 $\leq$ 300 mm Hg with PEEP or CPAP $\geq$5 cm H_2O[c]
Moderate	100 mm Hg $<$ Pao_2/Fio_2 $\leq$ 200 mm Hg with PEEP $\geq$5 cm H_2O
Severe	Pao_2/Fio_2 $\leq$ 100 mm Hg with PEEP $\geq$5 cm H_2O

Abbreviations: CPAP, continuous positive airway pressure; Fio_2, fraction of inspired oxygen; Pao_2, partial pressure of arterial oxygen; PEEP, positive end-expiratory pressure.

[a] Chest radiograph or computed tomography scan.

[b] If altitude is higher than 1000 m, the correction factor should be calculated as follows: [Pao_2/Fio_2 × (barometric pressure/760)].

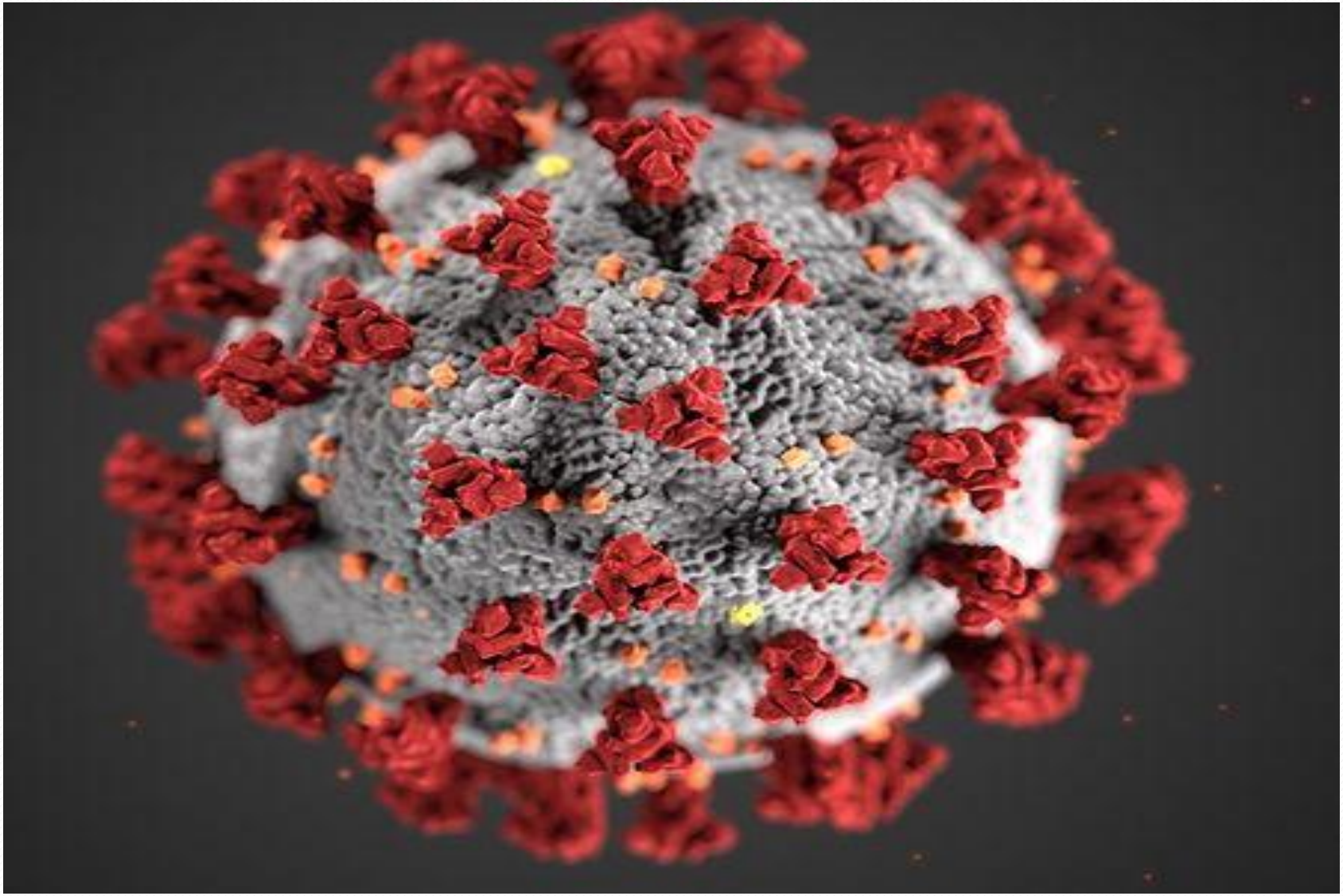
[c] This may be delivered noninvasively in the mild acute respiratory distress syndrome group.

Etiology

ARDS has many risk factors.

- Pulmonary infection eg the occurrence rate of ARDS with COVID-19 infection varies between 17% and 41%
- Pulmonary aspiration
- Extra-pulmonary sources
  - Sepsis
  - Trauma
  - Massive transfusion
  - Drowning
  - Drug overdose
  - Fat embolism
  - Inhalation of toxic fumes, and pancreatitis

(these extra-thoracic illnesses and/or injuries trigger an inflammatory cascade culminating in pulmonary injury)



Risk Factors For ARDS

- Advanced age
- Female gender
- Smoking
- Alcohol use
- Aortic vascular surgery
- Cardiovascular surgery
- Traumatic brain injury

Epidemiology

- Estimates of the incidence of ARDS in the United States range from

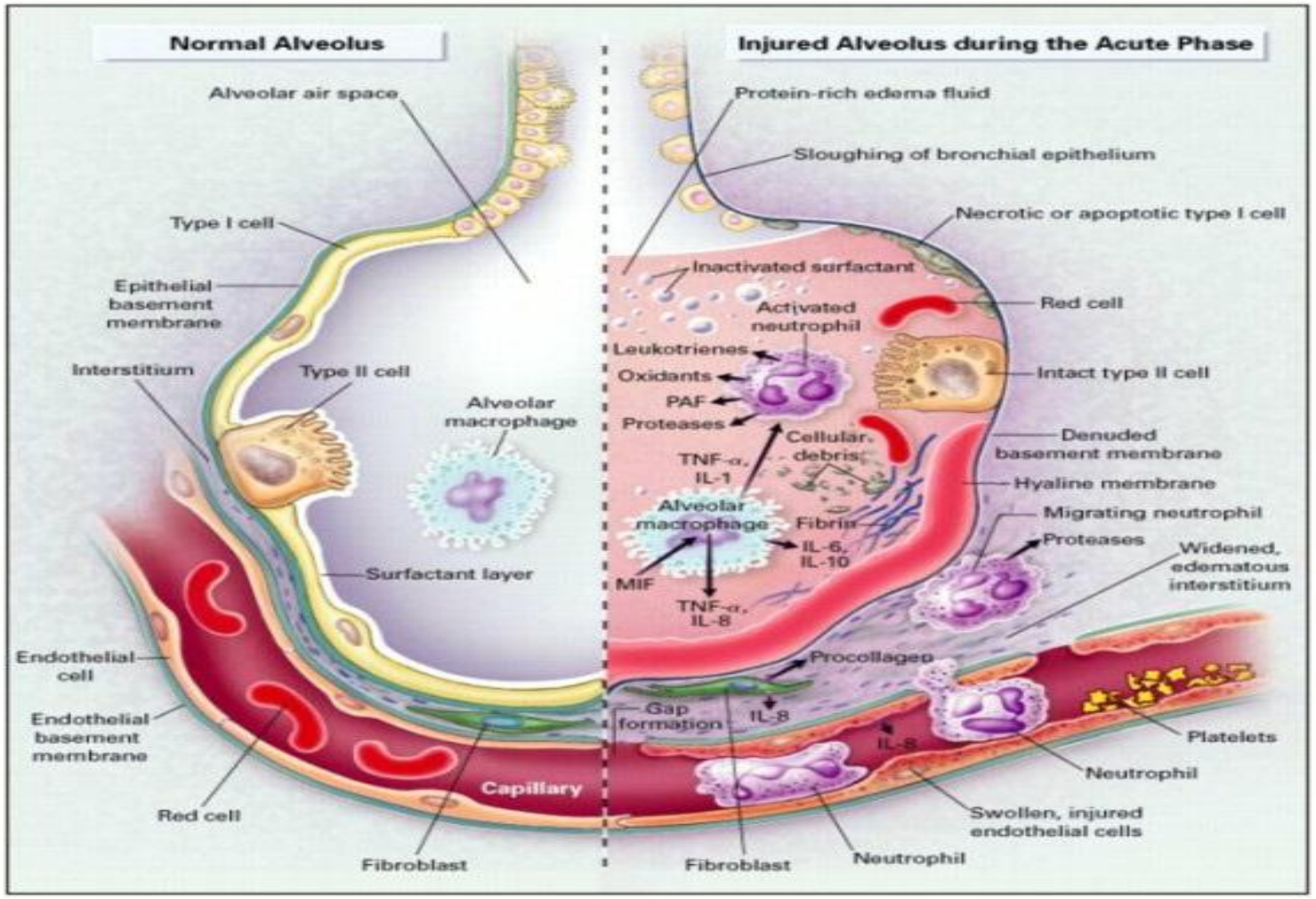
  64.2 to 78.9 cases/100,000 person-years.

- Twenty-five percent of ARDS cases are initially classified as mild (1/3 will progress to severe or moderate) and 75% as moderate or severe.

Epidemiology

- Mortality of ARDS is dependant on the severity of the disease, it is
  - 27% for mild
  - 32% for moderate
  - 45% for severe disease

Pathological Process



- ARDS represents a stereotypic response to various etiologies.
- It progresses through different phases

First phase –

- **Damage of the alveolo-capillary barrier** leading to **pulmonary oedema**.
- The pulmonary epithelial and endothelial cellular damage inflammation, apoptosis, necrosis, and increased alveolar-capillary permeability, which leads to the development of alveolar edema and proteinosis.
- There is **bidirectional leakage of fluids and proteins** into the alveolus and as well as surfactant proteins and alveolar cytokines into the plasma.
- The epithelial barrier becomes disrupted with a **proliferation of type 2 alveolar cells** leading to **surfactant dysfunction**.
- Alveolar edema, in turn, reduces gas exchange, leading to hypoxemia.

- **Proliferative Phase**

characterized by improved lung function and healing

- **Final fibrotic phase**

Signaling the end of the acute disease process.

Surfactant turnover is significantly increased and the fluid that lines the epithelium also highlights fibrosing alveolitis early in the course of lung damage.

- A hallmark of the pattern of injury seen in ARDS is that it is **not uniform**.
- Segments of the lung may be more severely affected, resulting in decreased regional lung compliance, which classically involves the **bases more than the apices.**
- This intrapulmonary differential in pathology results in a variant response to oxygenation strategies. While increased **Positive End-expiratory Pressure (PEEP)** may improve oxygen diffusion in affected alveoli, it may result in deleterious volutrauma and atelectrauma of adjacent unaffected alveoli

Cellular Involvement in ARDS:

- **Neutrophils**

Most abundant in both the epithelial lining and alveolar histological specimens.

Although the chemotaxic nature of neutrophils crossing the epithelium does not cause damage, their pro-inflammatory nature release **reactive oxygen species, cytokines and a number of inflammatory mediators** which contribute to the basement membrane damage.

Cellular Involvement in ARDS:

- **Alveolar Macrophages**

These are the most common cell type and with interstitial macrophages play an important role in defence. In ARDS patients there is a progressive increase in alveolar macrophage number.

- **Epithelium**

Contained within the alveolar epithelial tissue are the highly metabolically **active type 2 alveolar cells**. Damaged epithelium leads to dysfunctional surfactant

Clinical Presentation and Assessment

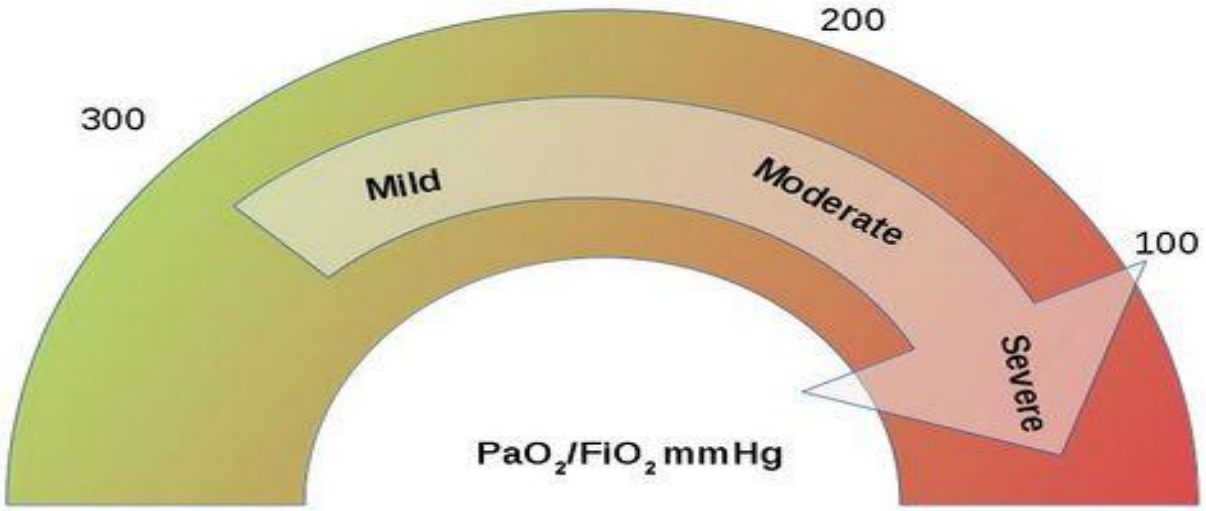
- The syndrome is characterized by :
- The development of dyspnea and hypoxemia, which progressively worsens within hours to days, frequently requiring mechanical ventilation and intensive care unit-level care.
- The history is directed at identifying the underlying cause which has precipitated the disease.

Clinical Presentation and Assessment

When interviewing patients that are able to communicate, often they start to complain of

- Mild dyspnea initially
- Respiratory distress escalates within 12-24 hours
- Becoming severe and
- Requiring mechanical ventilation to prevent hypoxia.

Gradation of severity of Acute Respiratory Distress Syndrome (ARDS)
(Based on the 'Berlin definition')



http://en.wikibooks.org/wiki/Intensive_Care_Medicine

Physical Assessment

- Acute Inflammatory Phase

Lasts 3 – 10 days and results in **Hypoxaemia** and **Multi Organ Failure**. Patients typically present with progressive dyspnoea, tachypnoea, cyanosis, hypoxic confusion and lung crepitations.

- Systemic signs (depending on the severity of illness) eg **central or peripheral cyanosis** as a result of hypoxemia, tachycardia, and altered mental status.

Physical Assessment

- Despite 100% oxygen, patients have low oxygen saturation. Chest auscultation usually reveals rales, especially bibasilar, but are often auscultated throughout the chest

- Healing, Proliferative Phase:

During this phase, lung scarring and pneumothoracies are common

Diagnostic Procedures

According to the National Heart, Lung and Blood Institute a diagnosis will be made via the examination of your

- **Medical History**
- **Physical Exam And**
- **Test Results**

Medical History

- History of heart failure

- Has the patient had any direct or indirect clinical risk factor for ARDS?

Physical Examination

- Added breath sounds on auscultation (e.g crackling)
- Heart auscultation
- Cyanosis

Test Results

- Arterial blood gases
- Chest x-ray
- Blood Tests
- Sputum Culture
- CT Scan

Management / Interventions

Unfortunately, no drug has been proven to be effective in preventing or managing ARDS.

The chief treatment strategy is supportive care and focuses on

1. Reducing shunt fraction,
2. Increasing oxygen delivery
3. Decreasing oxygen consumption
4. Avoiding further injury

- Patients are mechanically ventilated, guarded against fluid overload with diuretics, and given nutritional support until evidence of improvement is observed.

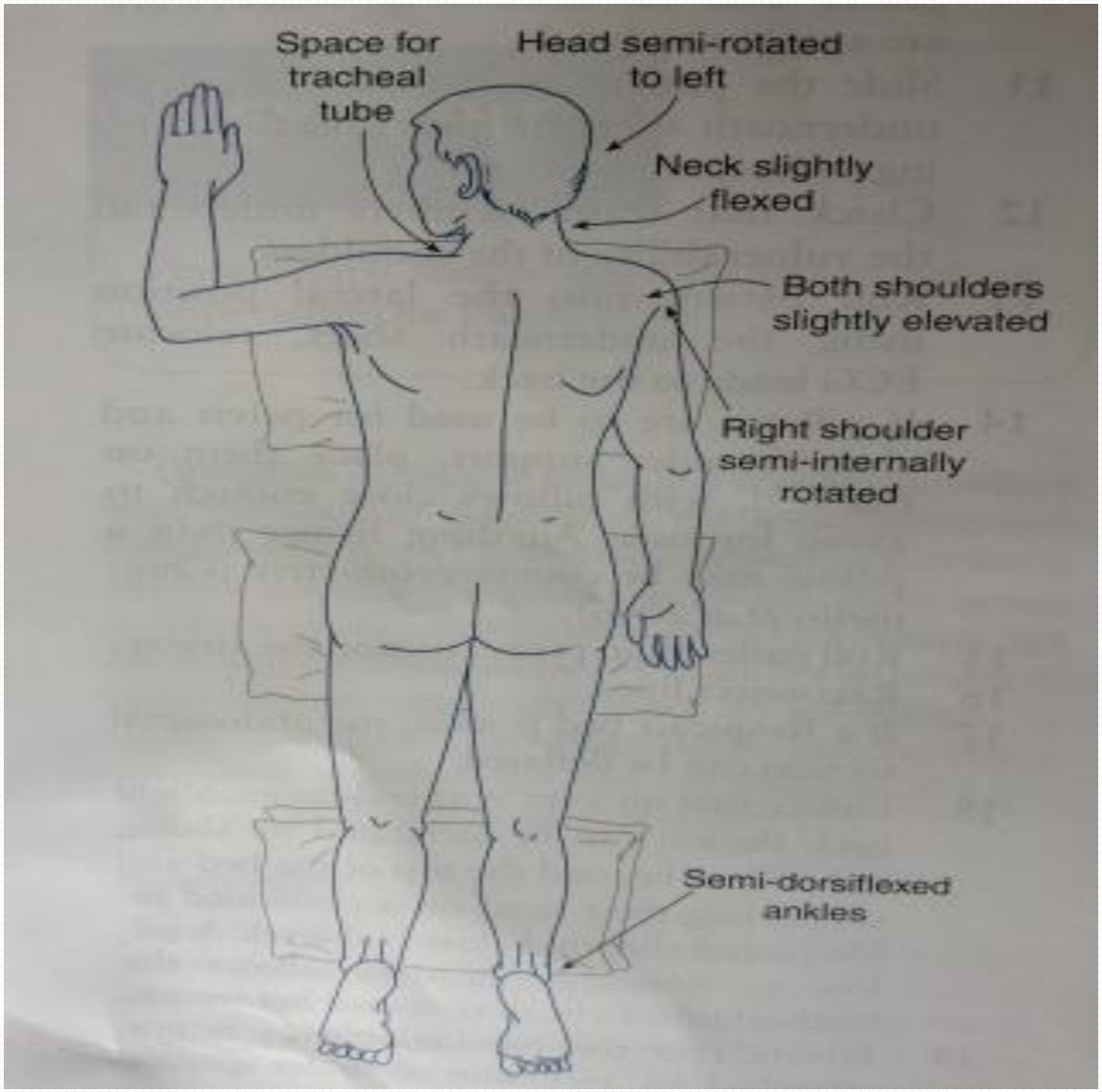
- The mode in which a patient is ventilated has an effect on lung recovery. Evidence suggests that some ventilatory strategies can exacerbate alveolar damage and perpetuate lung injury in the context of ARDS.

- Care is placed in preventing **Volutrauma** (exposure to large tidal volumes), **Barotrauma** (exposure to high plateau pressures), and **Atelectrauma** (exposure to atelectasis)

Possible Interventions for ARDS

Consider the risk vs reward of your intervention, particularly when the lungs are fragile

- Suctioning
- Ventilator Hyperinflation
- Positioning


Space for tracheal tube
Head semi-rotated to left
Neck slightly flexed
Both shoulders slightly elevated
Right shoulder semi-internally rotated
Semi-dorsiflexed ankles

Conclusions

- Even though many risk factors for ARDS are known, there is **No Way Of Preventing ARDS**.
- Careful **management of fluids** in high-risk patients can be helpful.
- Steps should be taken to prevent aspiration by keeping the head of the bed elevated before feeding.
- **Lung Protective Mechanical Ventilation Strategy** in patients without ARDS who are high risk would help prevent ARDS