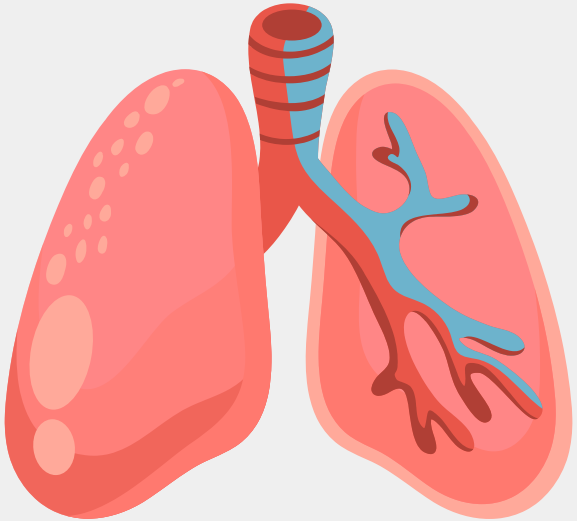
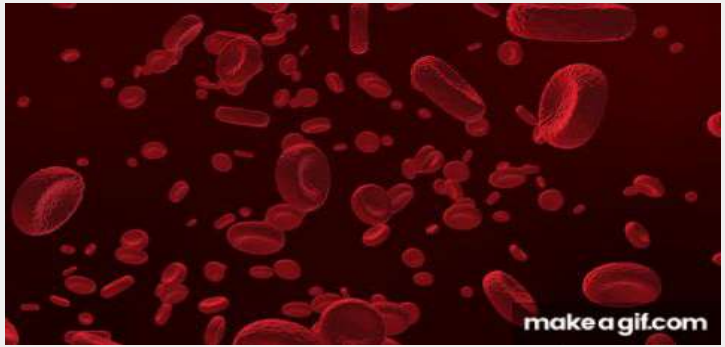
EFFECT OF CPB ON LUNGS

DEEPIKA
Bsc.CPPT



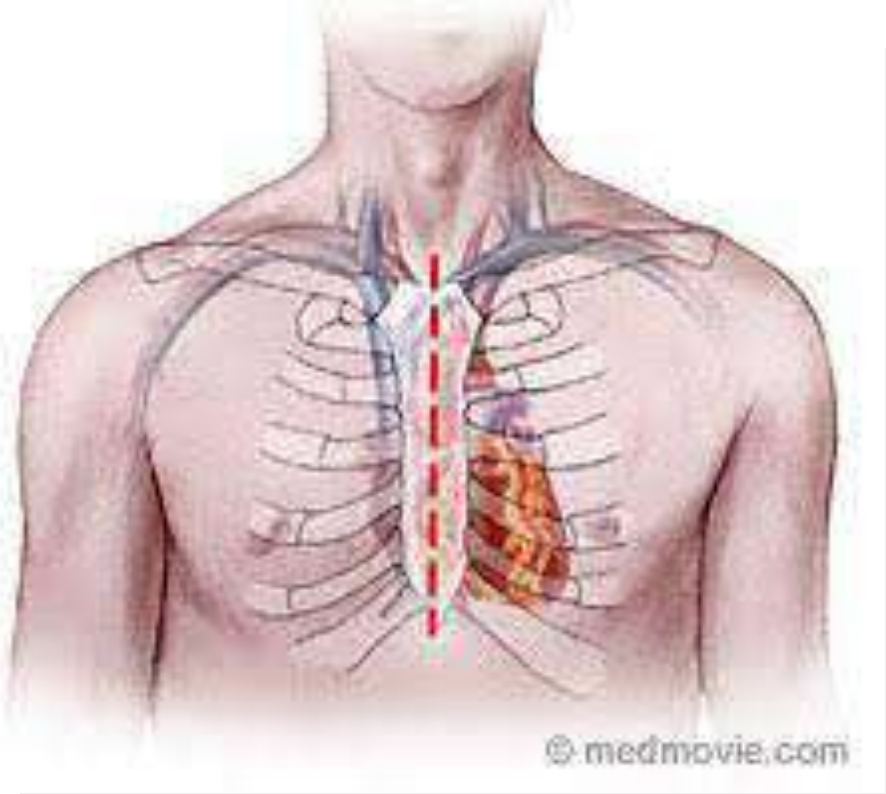
PATHOPHYSIOLOGY OF CPB ON LUNGS



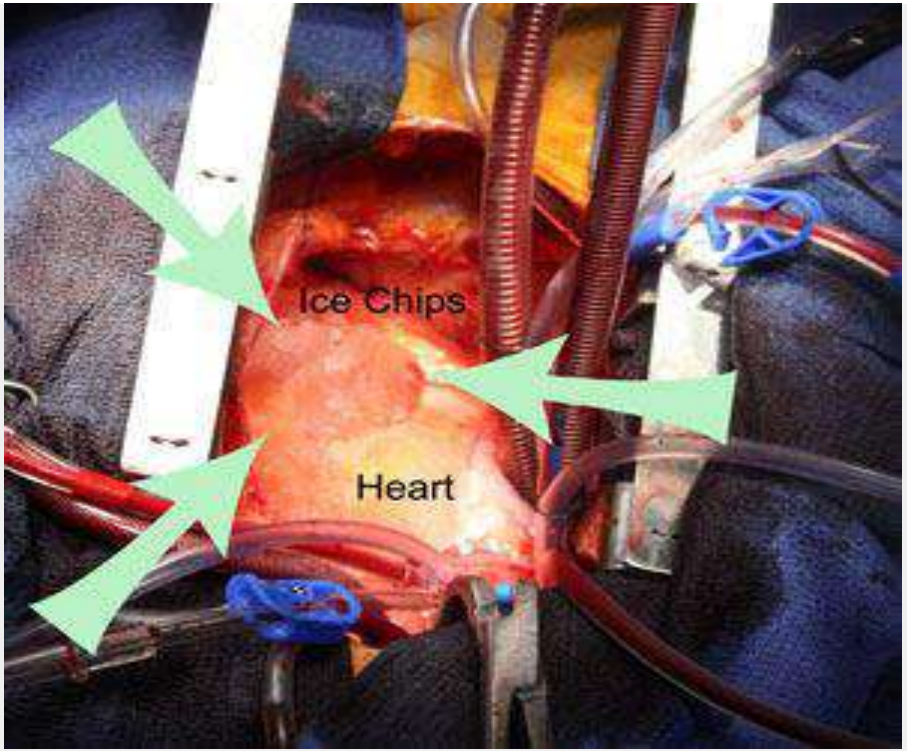
Hemodilution

Hypothermia

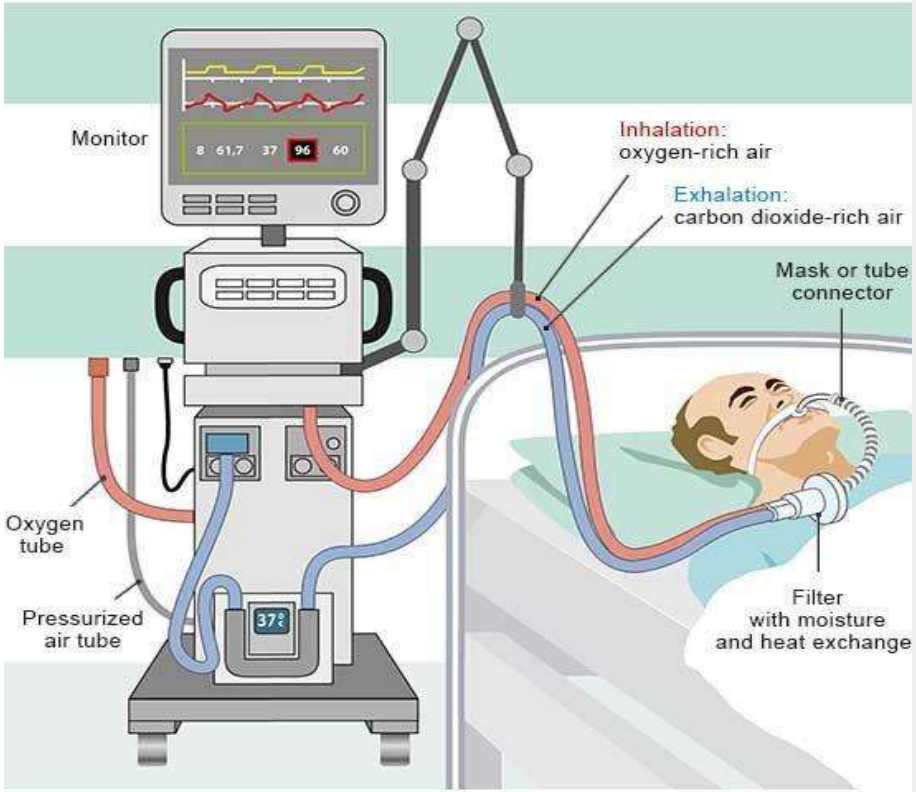
Contact with artificial surfaces



Cardiac surgery either sternotomy or thoracotomy



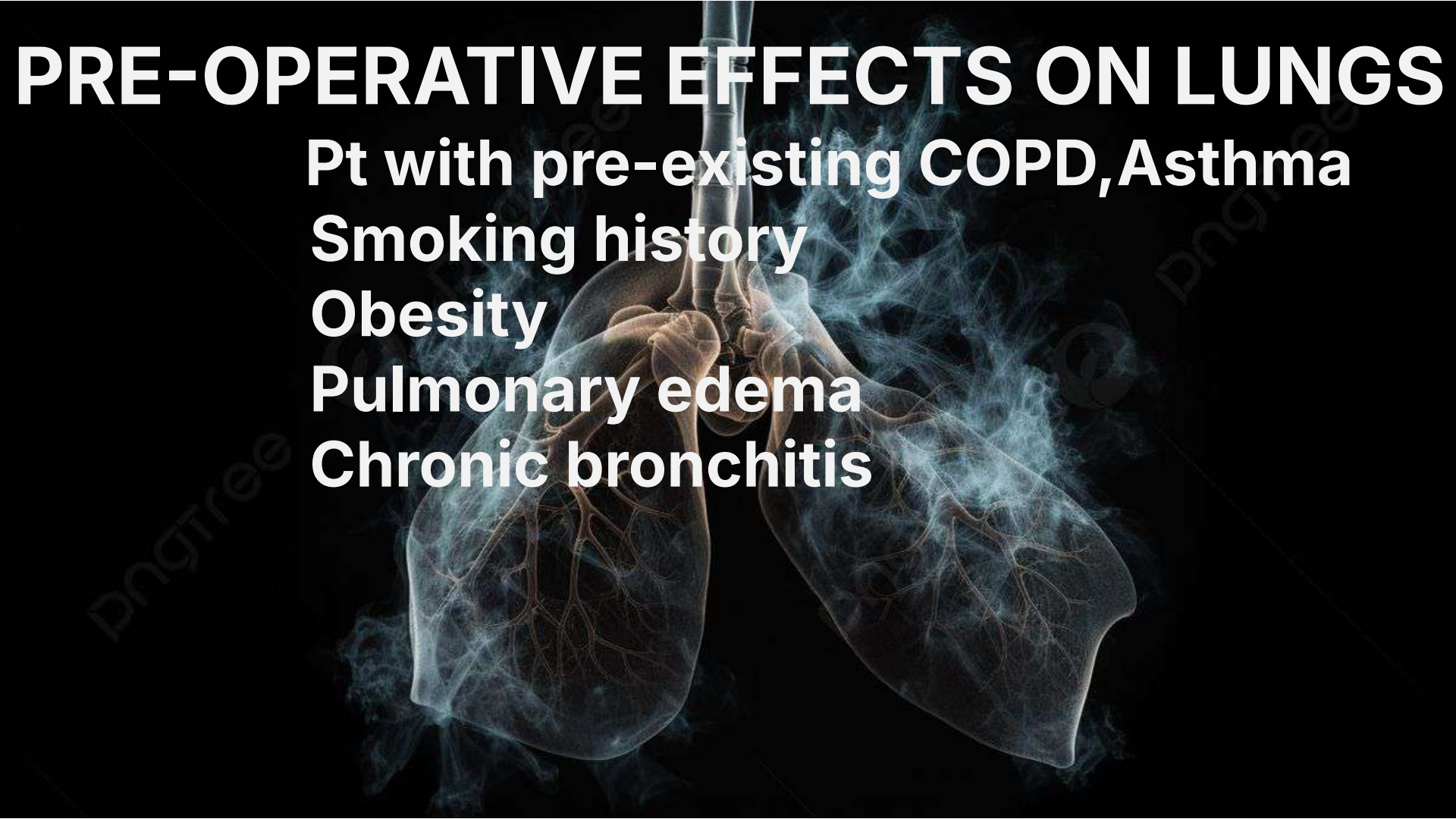
Cold solution applied on pericardium



Pt on ventilatory support

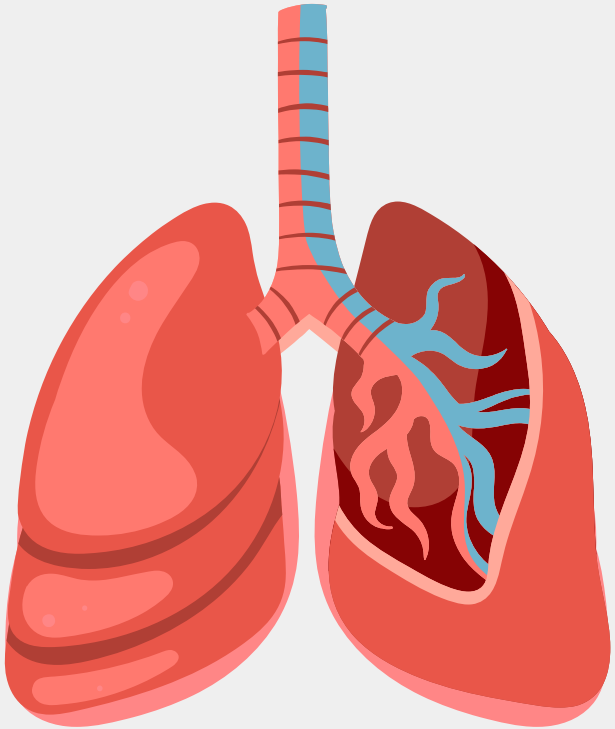
Prolonged pump time

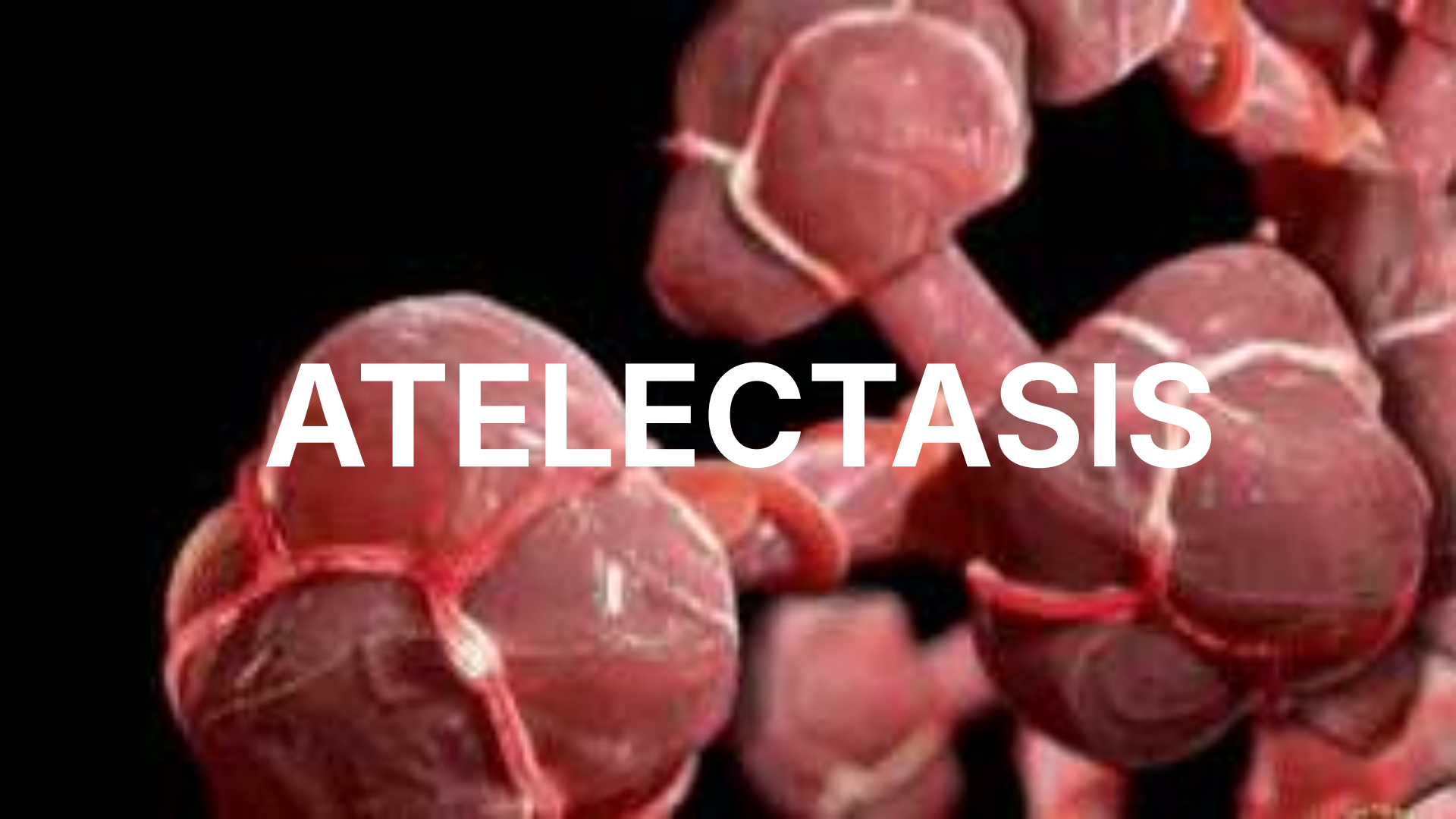
PRE-OPERATIVE EFFECTS ON LUNGS



Pt with pre-existing COPD,Asthma
Smoking history
Obesity
Pulmonary edema
Chronic bronchitis

Respiratory sequelae of CPB

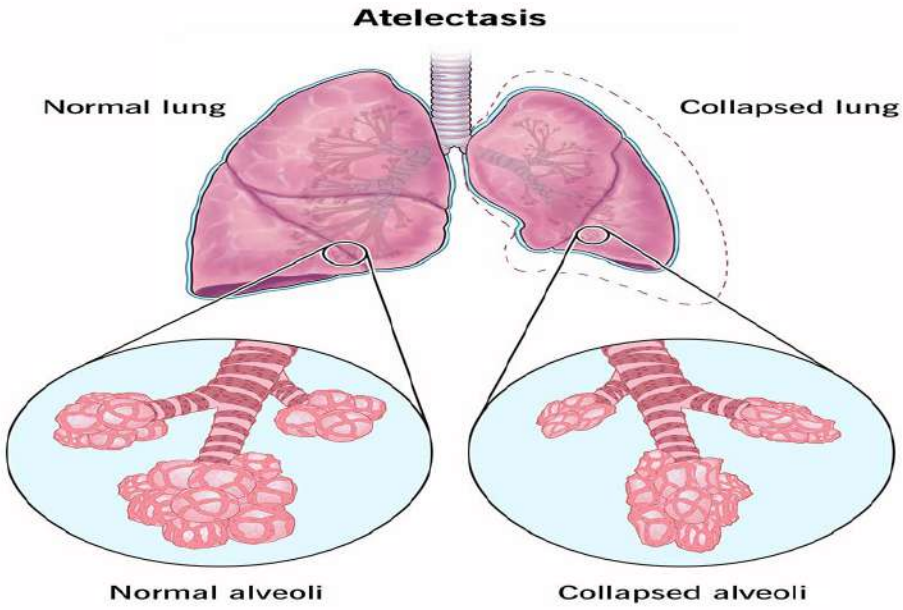


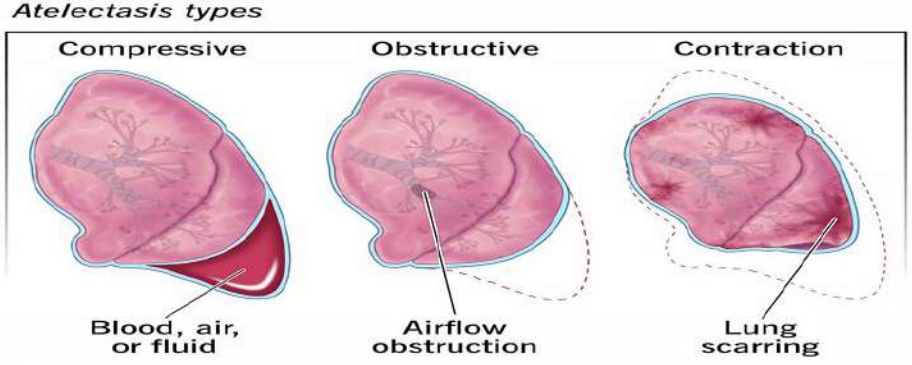

ATELECTASIS

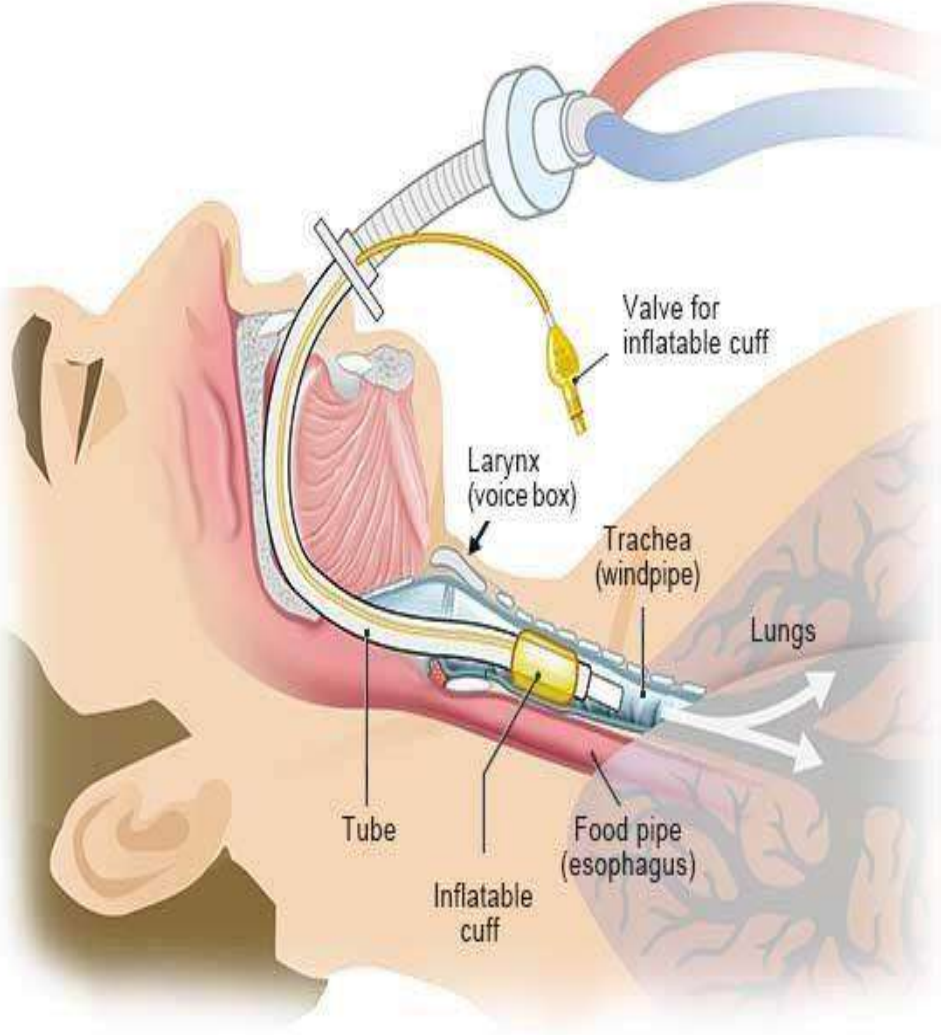
It is **collapse of a lung or part of a lung(lobe).** The most common cause of atelectasis is surgery with anesthesia.

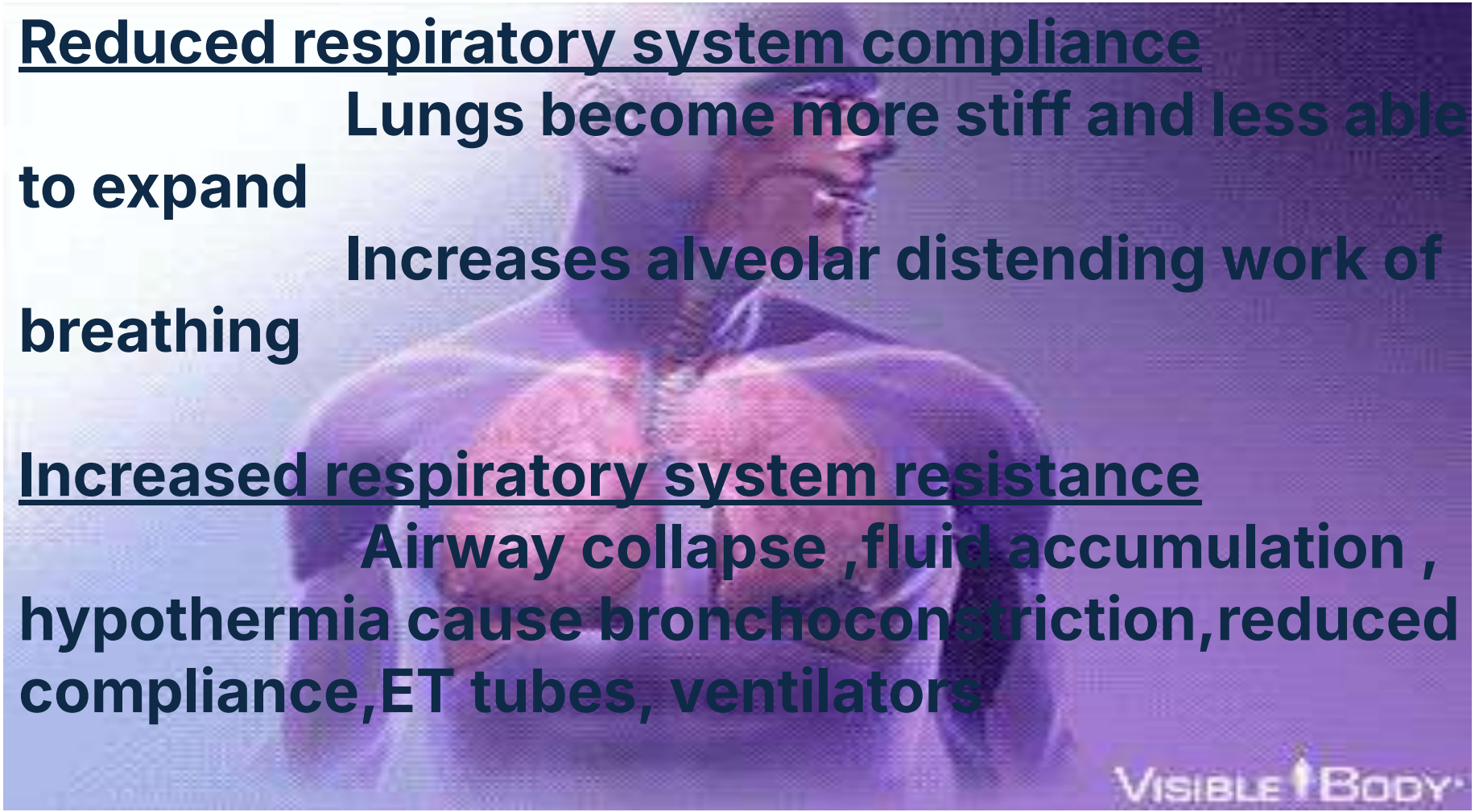
The fall in FRC promotes airway closure because of loss of muscle tone.

Resorptive atelectasis happens when the O_2 and CO_2 in your alveoli move into your bloodstream and no new air moves in. This causes your alveoli to **collapse.**








Reduced respiratory system compliance
Lungs become more stiff and less able to expand
Increases alveolar distending work of breathing
Increased respiratory system resistance
Airway collapse ,fluid accumulation , hypothermia cause bronchoconstriction,reduced compliance,ET tubes, ventilators
VISIBLE BODY

ANESTHESIA

After GA and mechanical ventilation initiates atelectasis within 5mins.Relaxed diaphragm distribute gas flow to non -independent region causing **“ventilation-perfusion mismatch”** promotes hypoventilation and microatelectasis of the dependent lung zones.

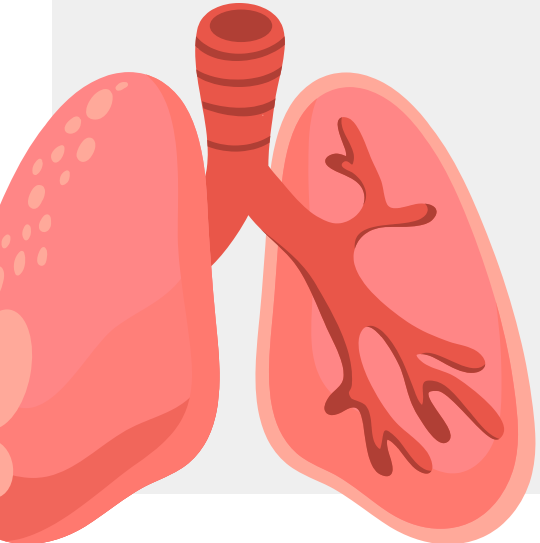
Sufactant depletion

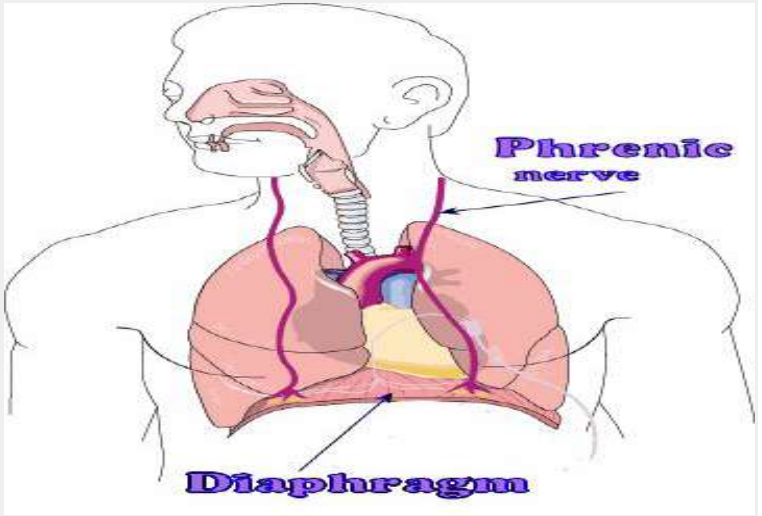
Pulmonary surfactant covers alveolar surface and lowers surface tension to prevent alveolar and small airway collapse.

Exposure to anesthesia reduce surfactant function and increase permeability of alveolar capillary barrier

Atelectasis promotes production proinflammatory cytokines and decrease synthesis of surfactant

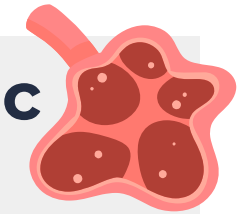
Phrenic nerve damage or dysfunction secondary to trauma or extreme cold (ie,exposure to topical slush) may result in significant postoperative loss of lung volume.
The "frostbitten phrenic nerve”





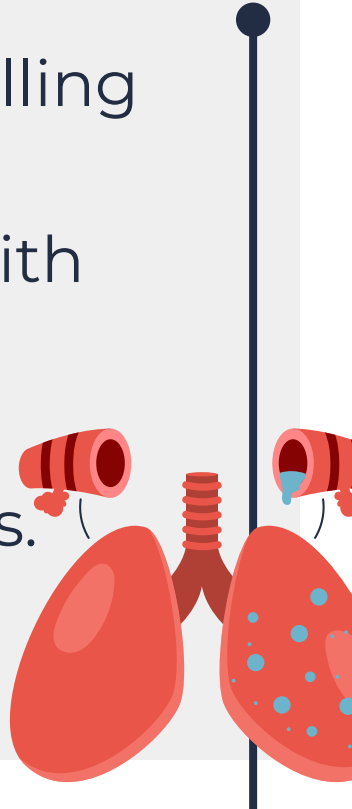
POST OPERATIVE LUNG SYNDROME [PUMP LUNG]

CPB have documented a variety of nonspecific changes including



- patchy atelectasis
- cellular(endothelial and pneumocytes) swelling and vacuolization
- Sequestrationof leukocytes in association with interstitial edema
- damage to cellular components

appear especially with increasing bypass times.



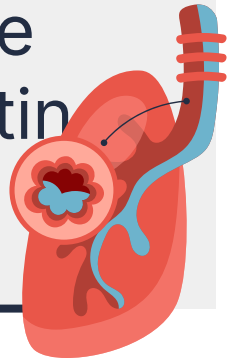
INTRA ALVEOLAR EDEMA

Excessive hemodilution causes accumulation of fluid excessively in alveolar walls and spaces of the lungs.

INTERSTITIAL EDEMA

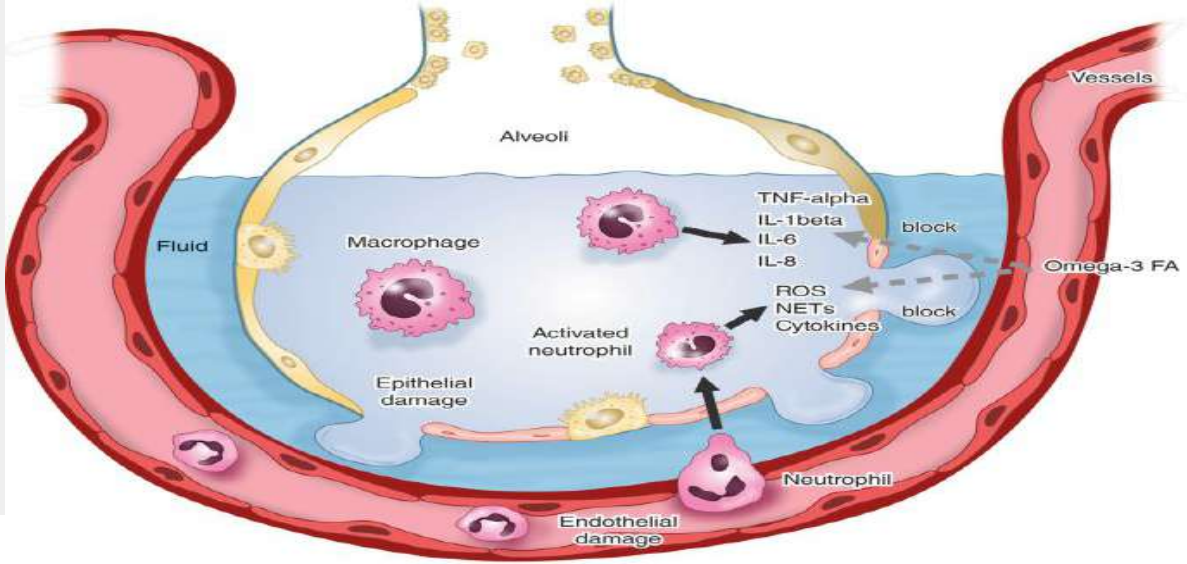
Increased vascular permeability caused by reduction in colloid osmotic pressure.

Overfilling pulmonary vasculature may cause increased hydrostatic pressure resultin in pulmonary edema



ACUTE RESPIRATORY DISTRESS SYNDROME[ARDS]

Sudden and progressive form **acute lung failure** in alveolar capillary membrane becomes damaged and permeable to intravascular fluid resulting in severe dynspnea, hypoxemia,diffuse pulmonary infiltrates.



Possible Mediators of Lung Injury Associated with CPB

Platelet activation and release of histamine and kallikrein/bradykinin occur during CPB may be important mediators of perioperative inflammation

Bacterial endotoxin (or lipopolysaccharide),a potent stimulator of complement and neutrophils,is released into the circulation during bypass.

STRATEGIES

Administration of **methylprednisolone** before bypass can reduce complement activation.

Glucocorticoids inhibit neutrophil activation induced by endotoxin.

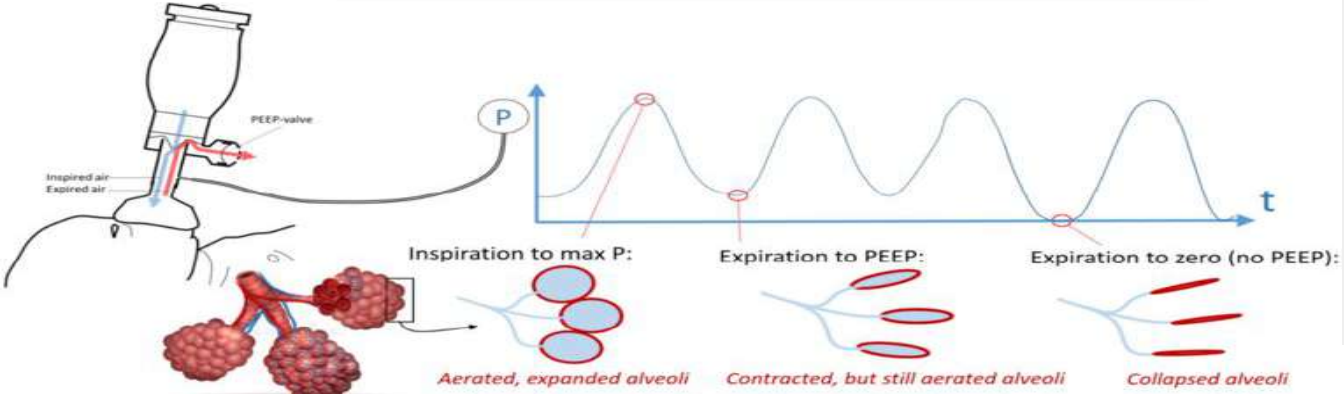
Colloids as pump prime to increase colloid osmotic pressure and reduce edema.

- **Heparin** bonded circuitry used in reducing the degree of complement activation.
- **Hemofiltration/ultrafiltration** incorporated aim of reducing pro-inflammatory mediators.
- **Leukocyte depleting filters** incorporated reducing circulating number of WBC

It has protective effect in reducing severity of lung and myocardial injury post cpb.

- Administration 0f **APROTININ** serine protease inhibitor decreases platelet dysfunction,lung neutrophil accumulation and adhesion, interleukins thereby decrease lung edema and improve compliance.

- volume controlled ventilation with extrinsic maintainence of **PEEP**[positive end expiratory pressure]




THANK YOU
FOR YOUR ATTENTION