2022/07/17
機械通氣重症繼續教育課程(南區)

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Physiologic Effects and Graphic Monitoring during Mechanical Ventilation

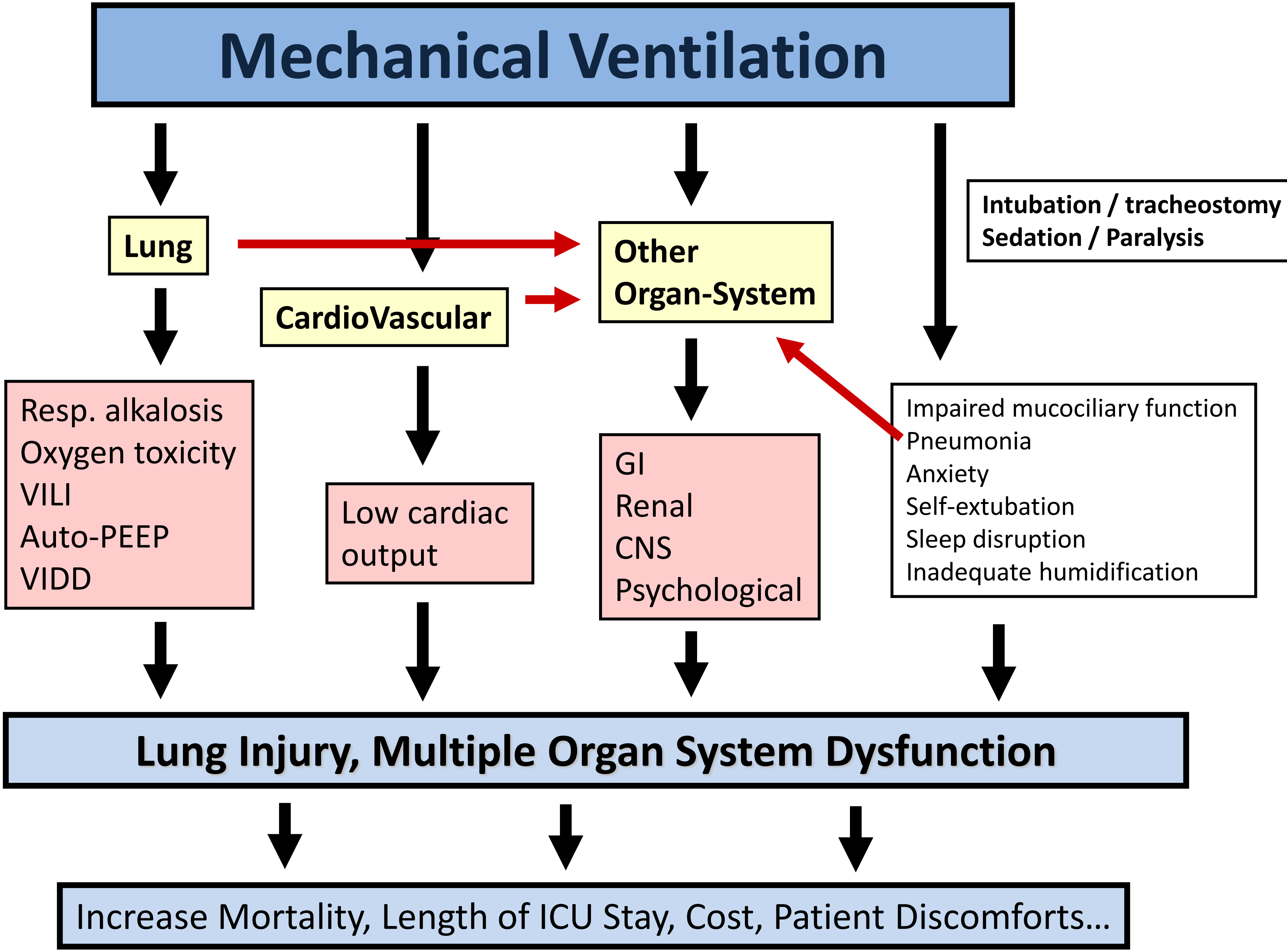


Outline

- Overview
- Pulmonary effects and complications
- Cardiovascular effects and complications
- Systemic effects and complications
- Ventilator Graphic

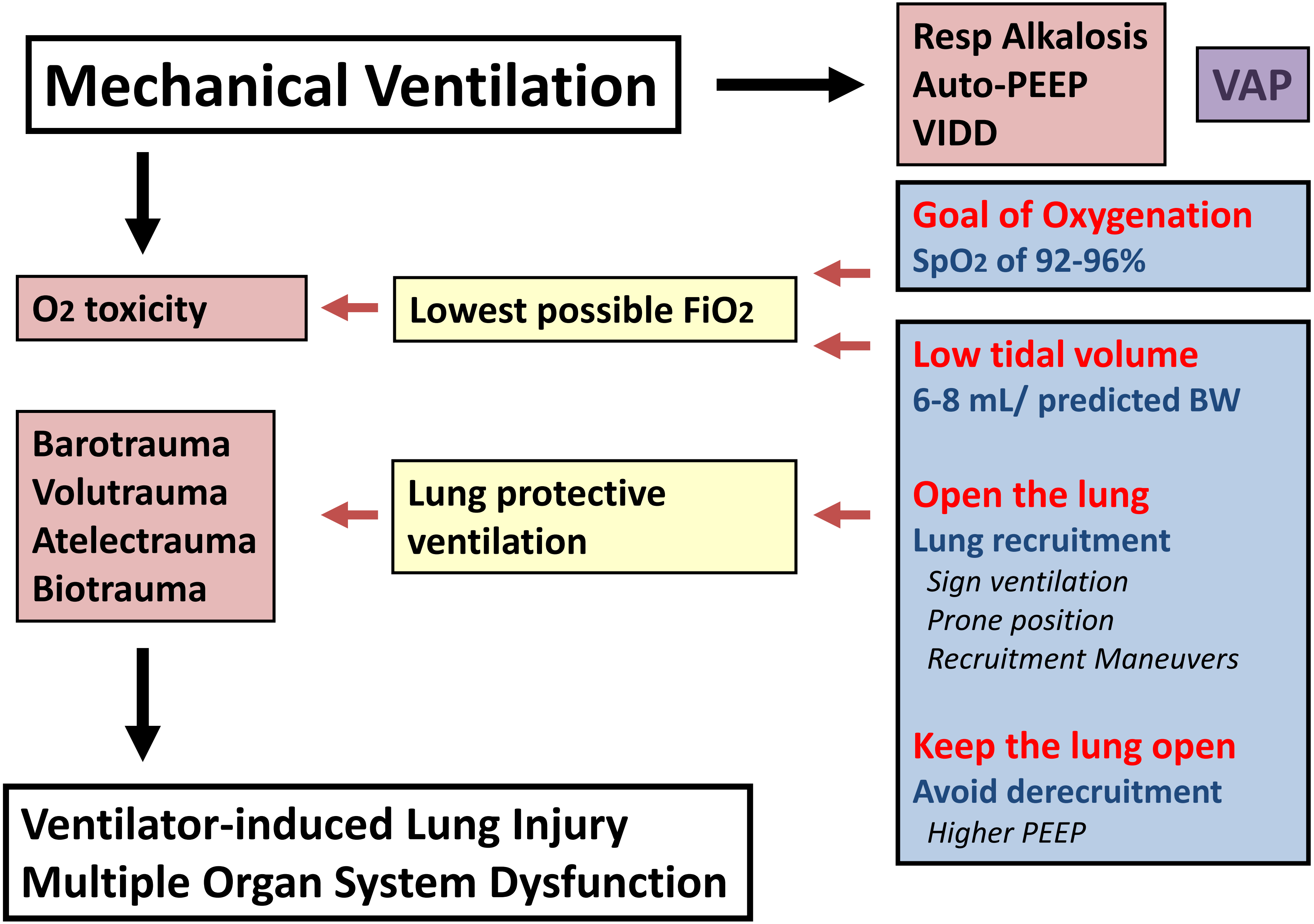
- *Mechanical ventilation* ***provide life saving support to critically*** *ill patients with respiratory failure. However,* ***it can damage the lung and other organ system*** *at the same time.*

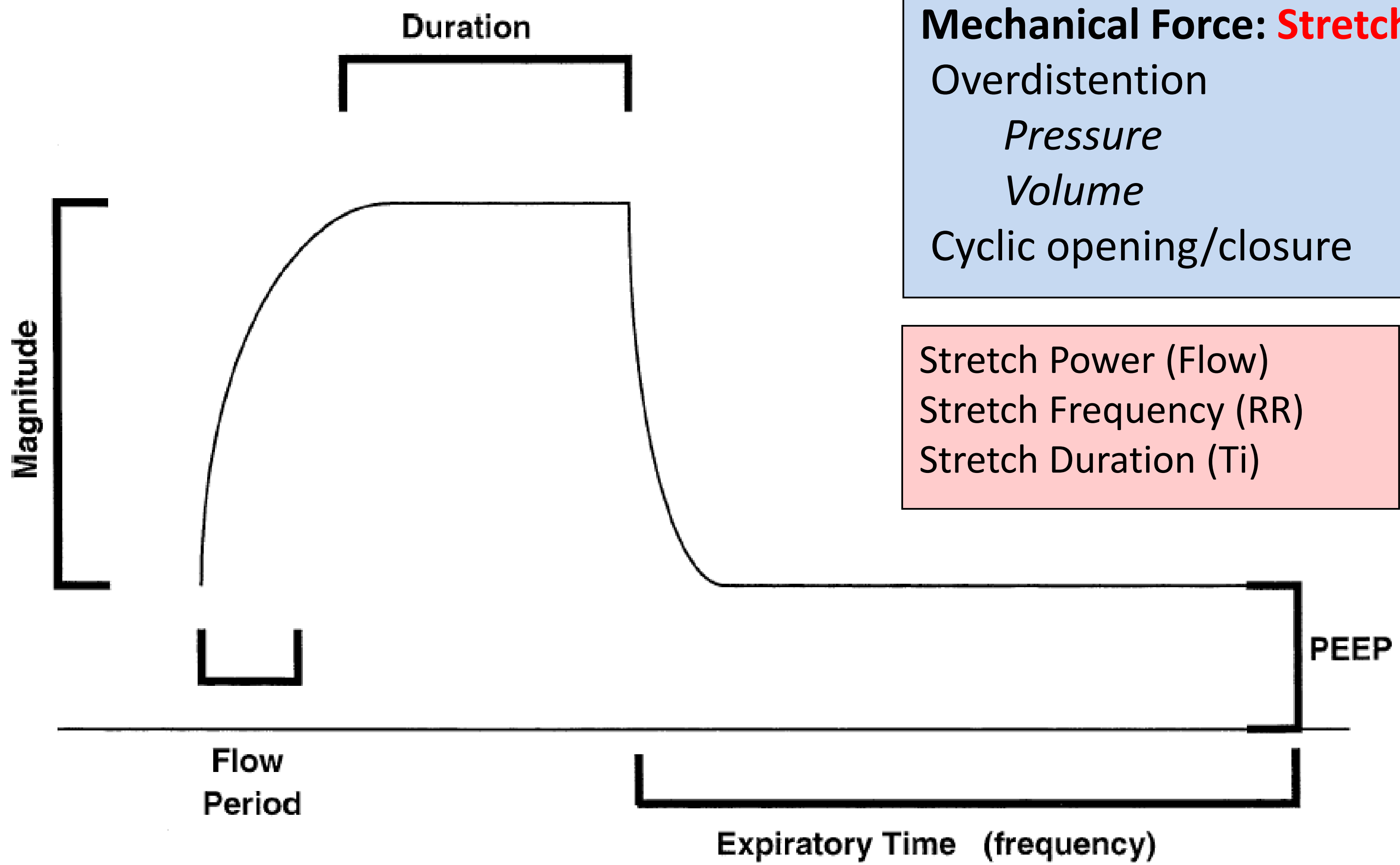
- *The goal of mechanical ventilation is to* ***achieve adequate gas exchange without toxicity or complications****.*


Mechanical Ventilation
Lung
CardioVascular
Other
Organ-System
Intubation / tracheostomy
Sedation / Paralysis
Resp. alkalosis
Oxygen toxicity
VILI
Auto-PEEP
VIDD
Low cardiac
output
GI
Renal
CNS
Psychological
Impaired mucociliary function
Pneumonia
Anxiety
Self-extubation
Sleep disruption
Inadequate humidification
Lung Injury, Multiple Organ System Dysfunction
Increase Mortality, Length of ICU Stay, Cost, Patient Discomforts…

Pulmonary Effects and Complications

- *Respiratory Alkalosis*
- *Oxygen Toxicity*
- *Ventilator-Induced Lung Injury (VILI)*
- *Ventilator-Induced Diaphragmatic Dysfunction (VIDD)*
- *Ventilator-Associated Pneumonia (VAP)*


Mechanical Ventilation
Resp Alkalosis
Auto-PEEP
VIDD
VAP
Goal of Oxygenation
SpO2 of 92-96%
O2 toxicity
Lowest possible FiO2
Low tidal volume
6-8 mL/ predicted BW
Barotrauma
Volutrauma
Atelectrauma
Biotrauma
Lung protective
ventilation
Open the lung
Lung recruitment
Sign ventilation
Prone position
Recruitment Maneuvers
Keep the lung open
Avoid derecruitment
Higher PEEP
Ventilator-induced Lung Injury
Multiple Organ System Dysfunction



Mechanical Force: Stretch

Overdistention

Pressure

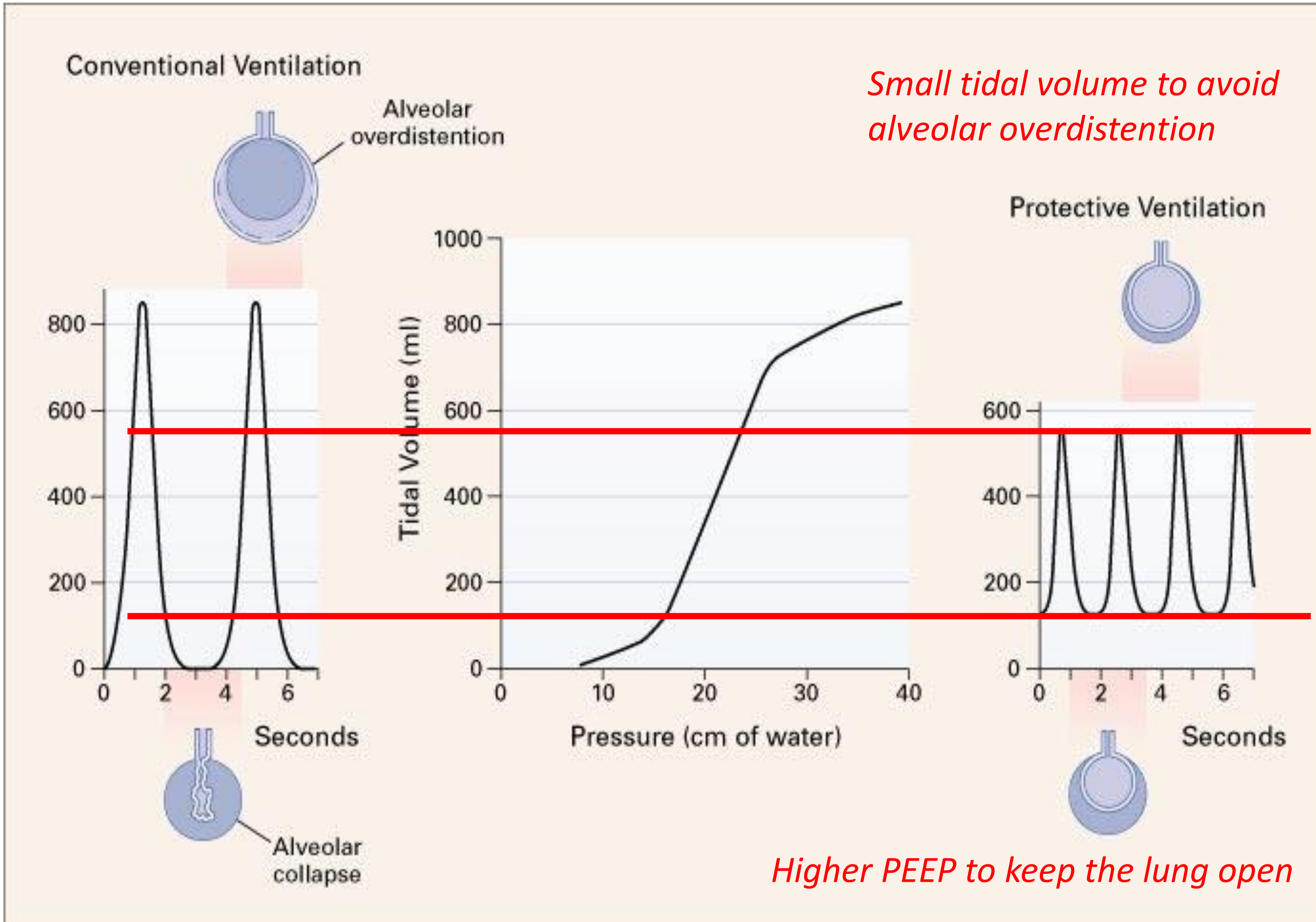
Volume

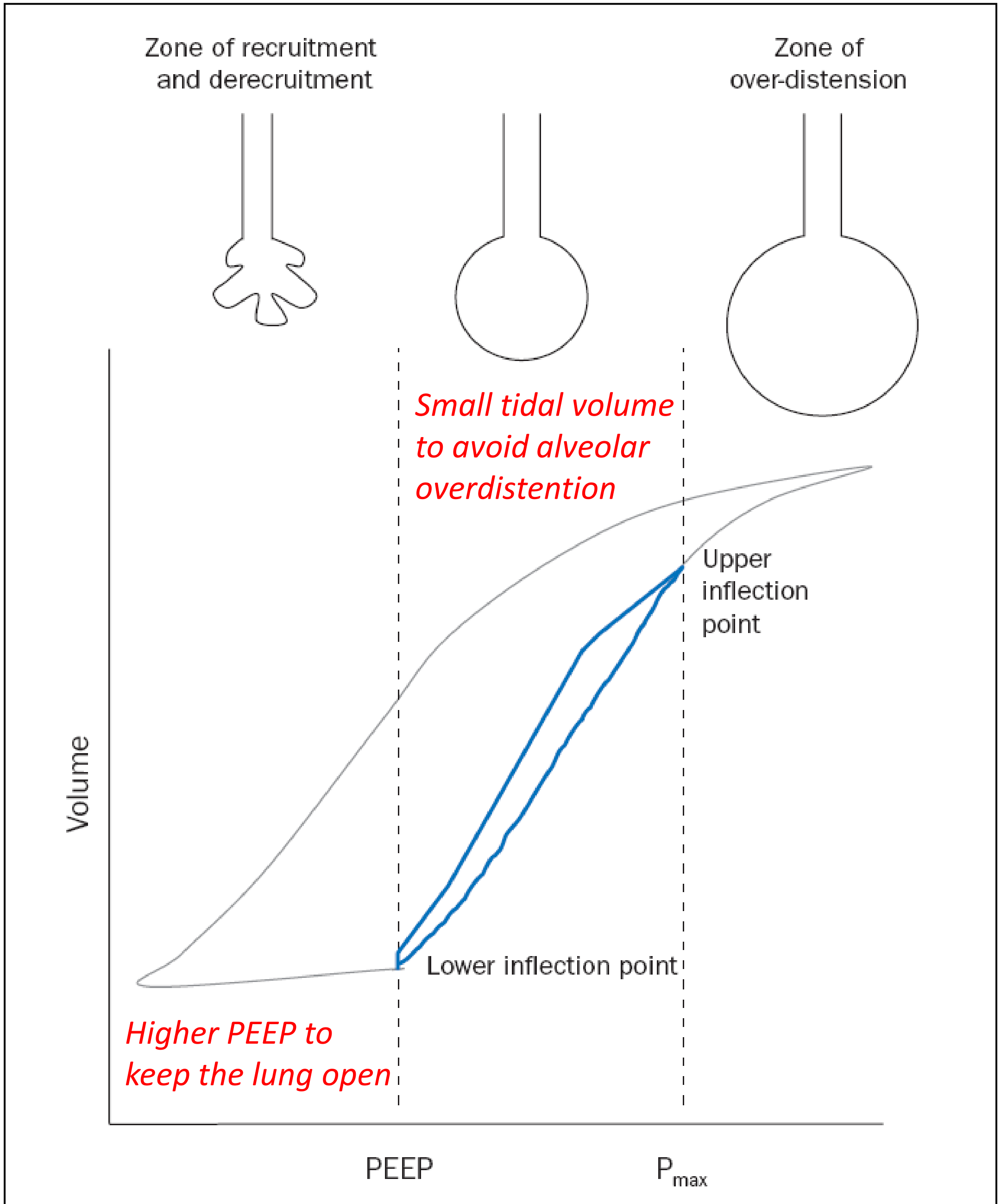
Cyclic opening/closure

Stretch Power (Flow)
Stretch Frequency (RR)
Stretch Duration (Ti)

Fig. 2. Schematic drawing of airway pressure waveform during pressure-controlled ventilation. Several aspects of PPV may contribute to VILI: the pressure magnitude or peak alveolar pressure, the inspiratory flow rate, inspiratory time, expiratory time, respiratory frequency, and PEEP.

Respir Care Clin 2003; 9: 343–362


Conventional Ventilation
Alveolar overdistention
Small tidal volume to avoid alveolar overdistention
Protective Ventilation
1000
800
600
400
200
0
Tidal Volume (ml)
0
2
4
6
Seconds
10
20
30
40
Pressure (cm of water)
Alveolar collapse
Higher PEEP to keep the lung open



Pulmonary pressure-volume relation of a patient with acute lung injury

- The lower inflection point is typically 12–18 cm H_2O and the upper inflection point 26–32 cm H_2O.
- Specific protective ventilation strategies require that PEEP is set just above the lower inflection point and the pressure limit (Pmax) just below the upper inflection point. Hence the lung is ventilated in the safe zone between the zone of recruitment and derecruitment and the zone of overdistension, and both high and low volume injury are avoided.

Lancet 2003; 361: 332–340

Injurious mode of ventilation (high volume or high pressure) will damage the normal lung

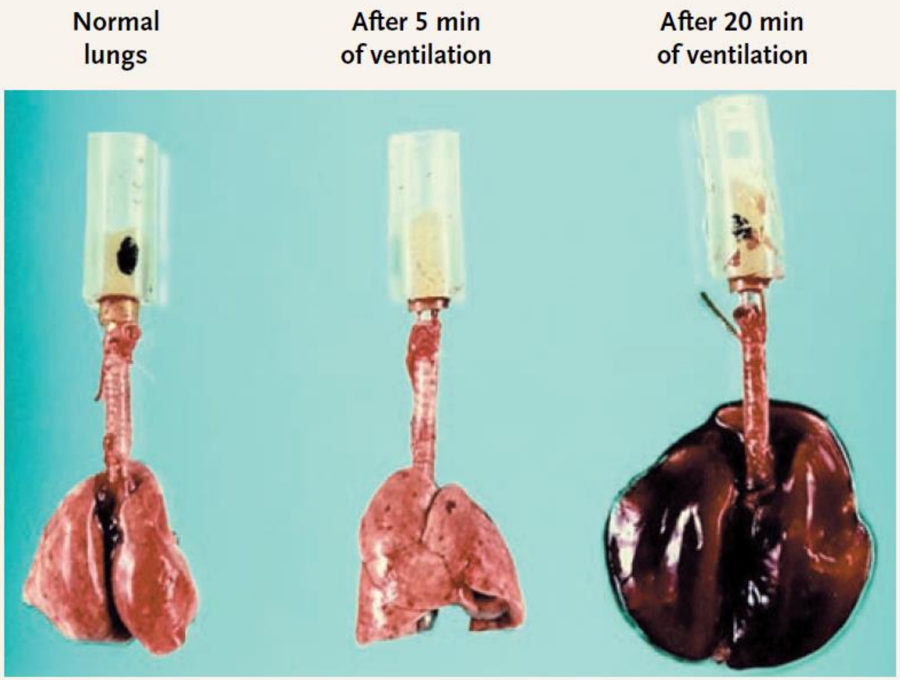
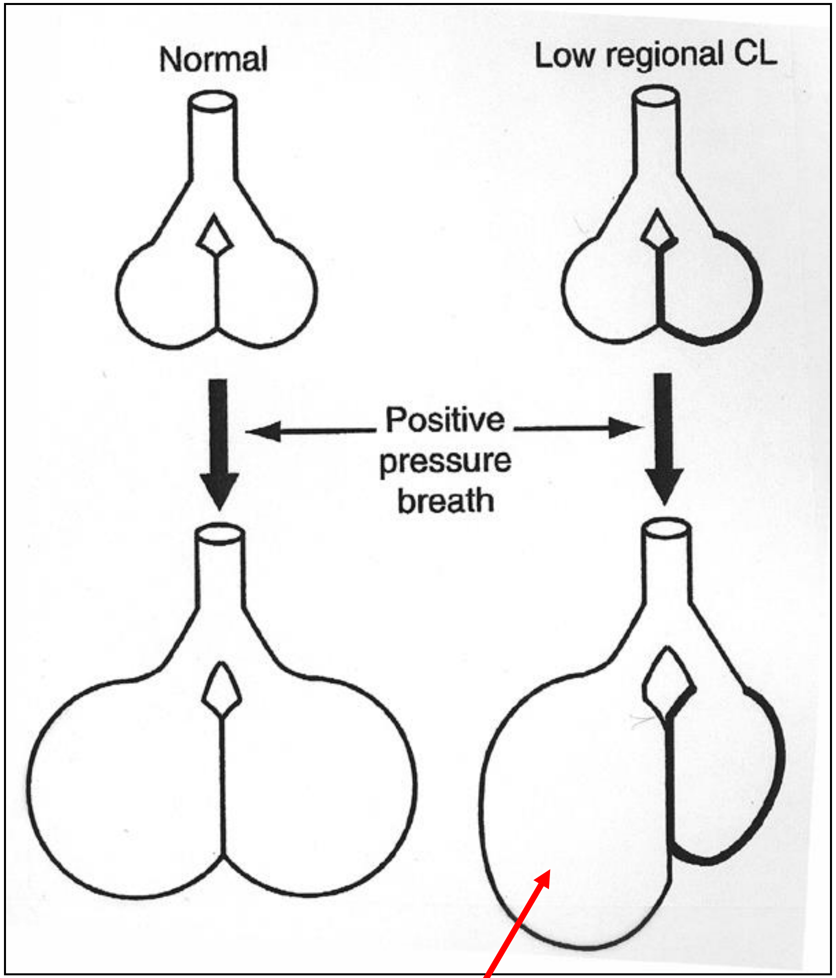


Figure 1. Normal Rat Lungs and Rat Lungs after Receiving High-Pressure Mechanical Ventilation at a Peak Airway Pressure of 45 cm of Water.

After 5 minutes of ventilation, focal zones of atelectasis were evident, in particular at the left lung apex. After 20 minutes of ventilation, the lungs were markedly enlarged and congested; edema fluid filled the tracheal cannula. Adapted from Dreyfuss et al.[8] with the permission of the publisher.



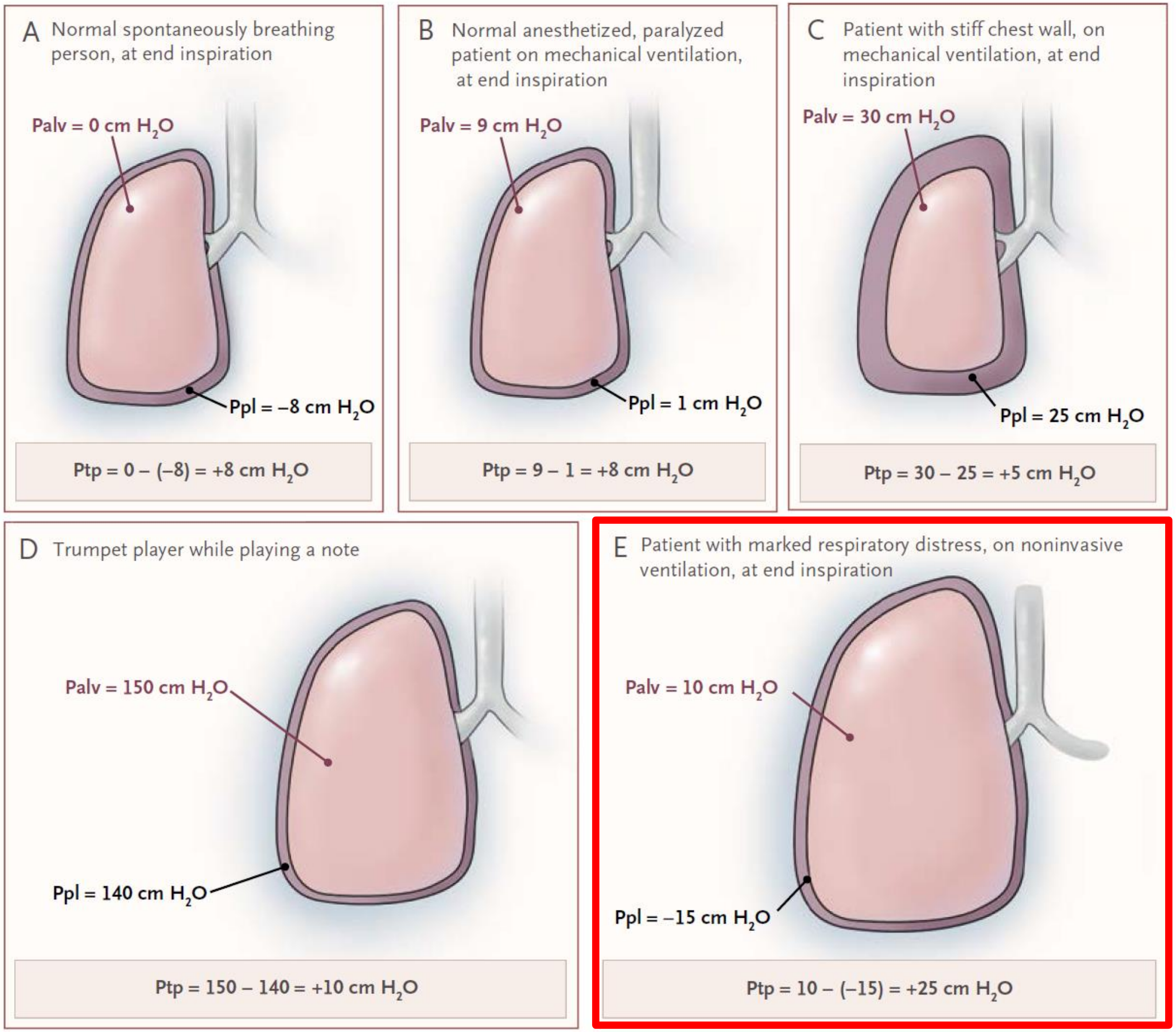
High-tidal-volume ventilation will cause overdistention of normal lung

Barotrauma

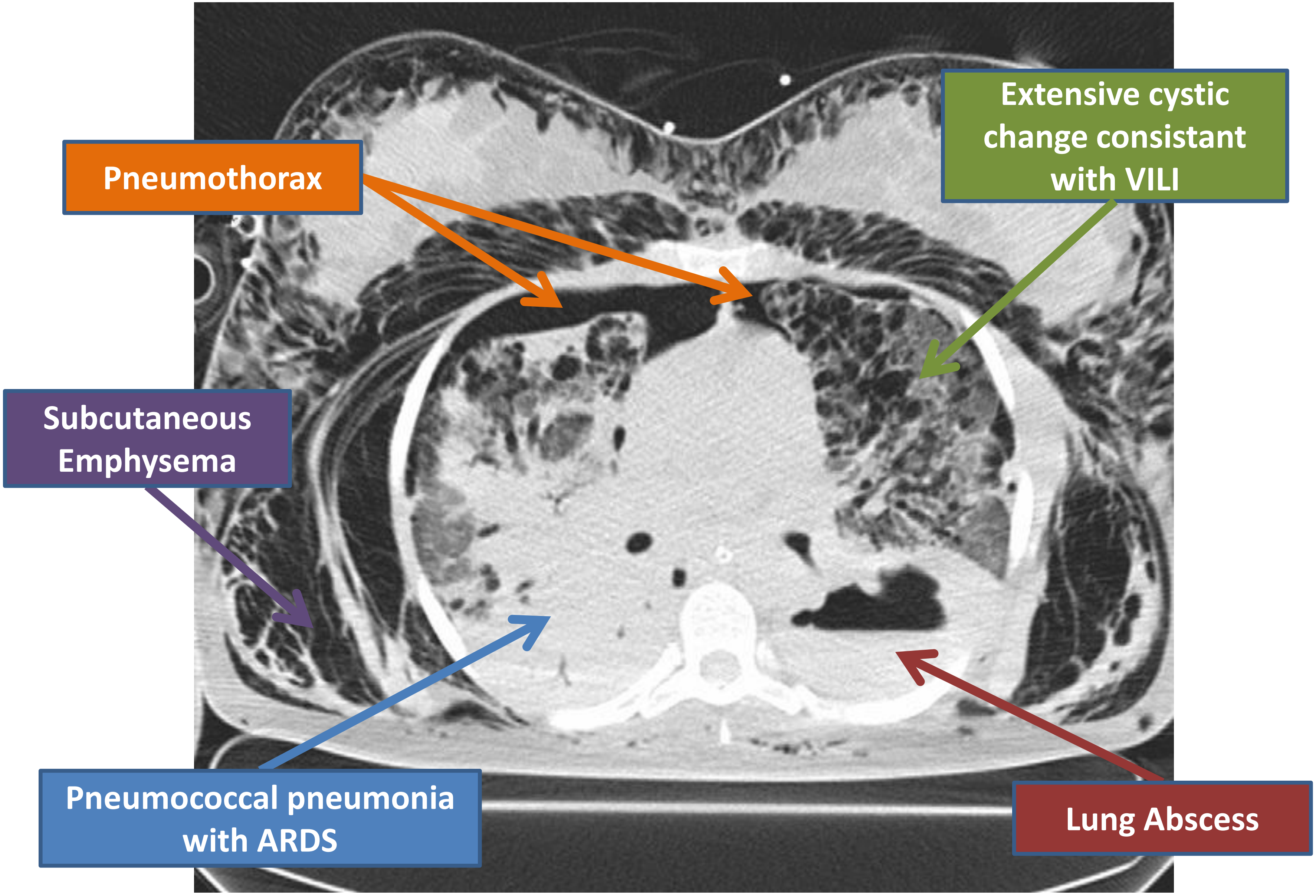
- High pressure induced lung damage
- **Which pressure is the most important?**
  - Peak airway pressure
  - Mean airway pressure
  - PEEP
  - <u>Plateau pressure</u>
  - **<u>Transpulmonary pressure</u>**
    - Alveolar pressure – Pleural pressure
- **What value?**

Lancet 2003; 361: 332–340

Transpulmonary pressure = Alveolar pressure – Pleural pressure



NEJM 2013; 369: 2126-2136



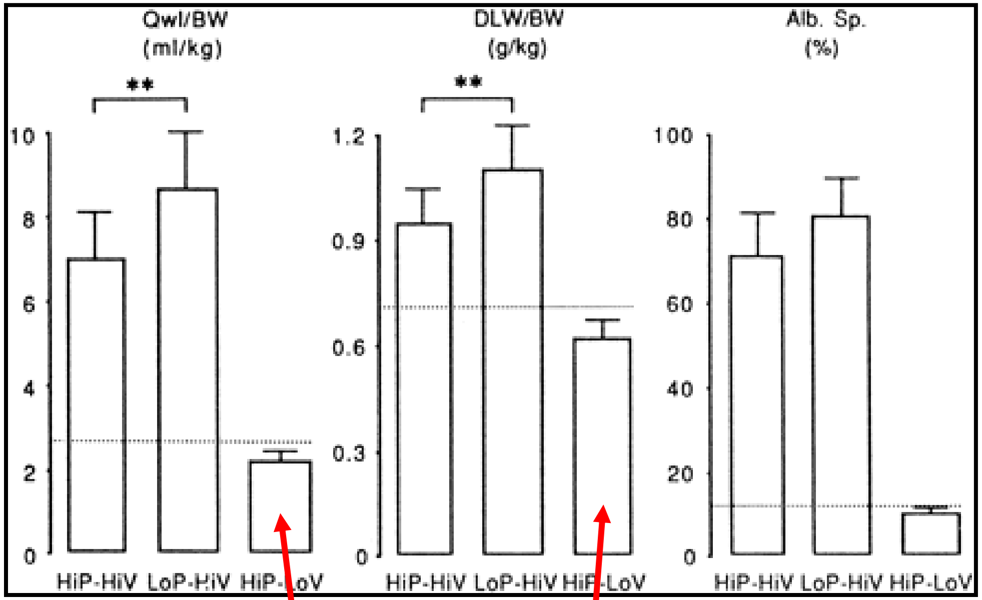
Lancet 2003; 361: 332–340

Strategy

- **Plateau Pressure ≤ 30-35 cmH2O**
  - The normal lung is maximally distended at a transpulmonary pressure between 30-35 cm H2O
  - A plateau pressure above the upper inflection point of pressure volume curve causes alveolar overdistention
- **Peak Airway Pressure ≤ 50 cmH2O**

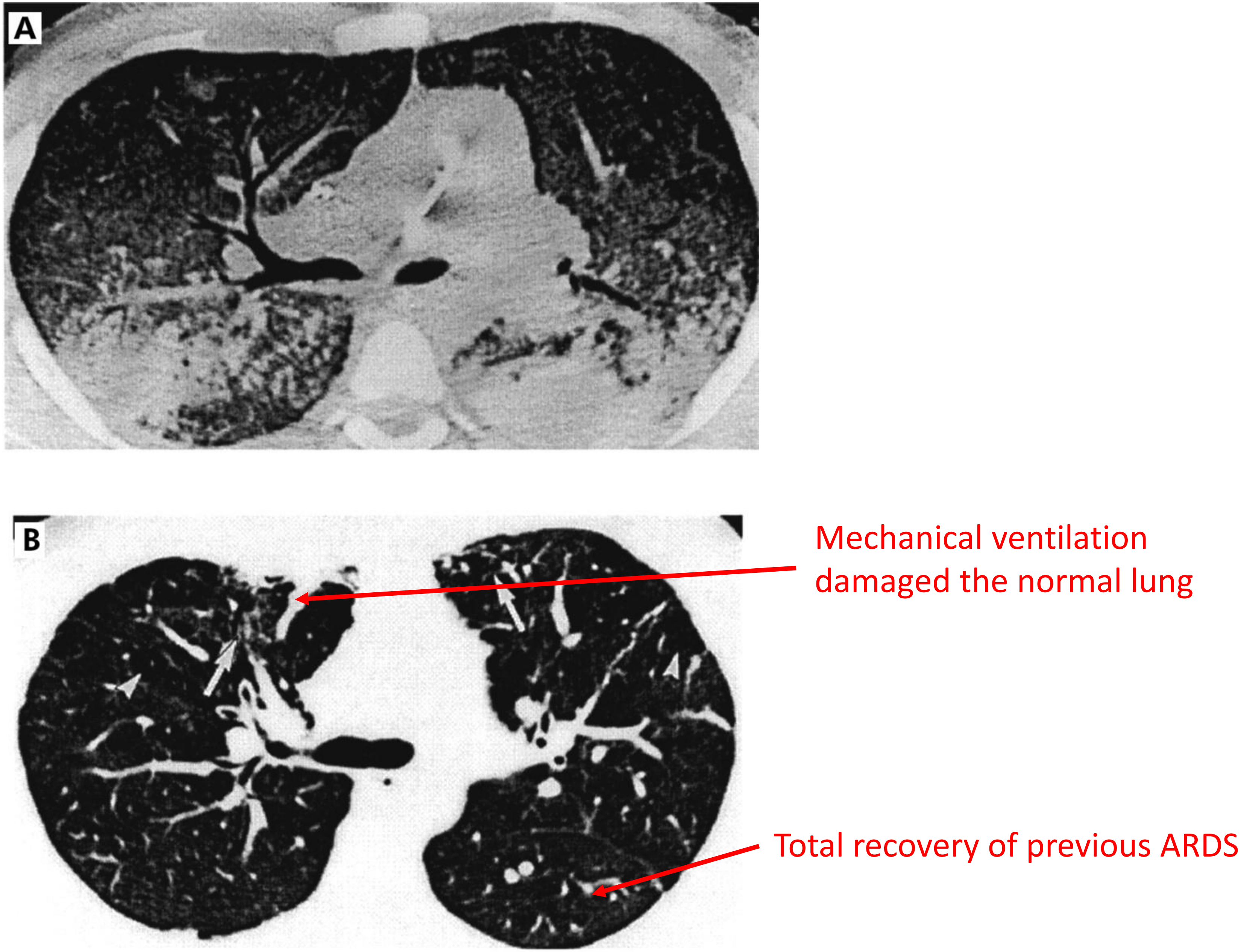
Volutrauma

- Damage caused by over-distension.
- Several animal models showed overdistention lead to increase fluid leak into alveoli (pulmonary edema)
- Mechanism:
  - Stretch injury



Low volume is more important than low pressure

Lancet 2003; 361: 332–40

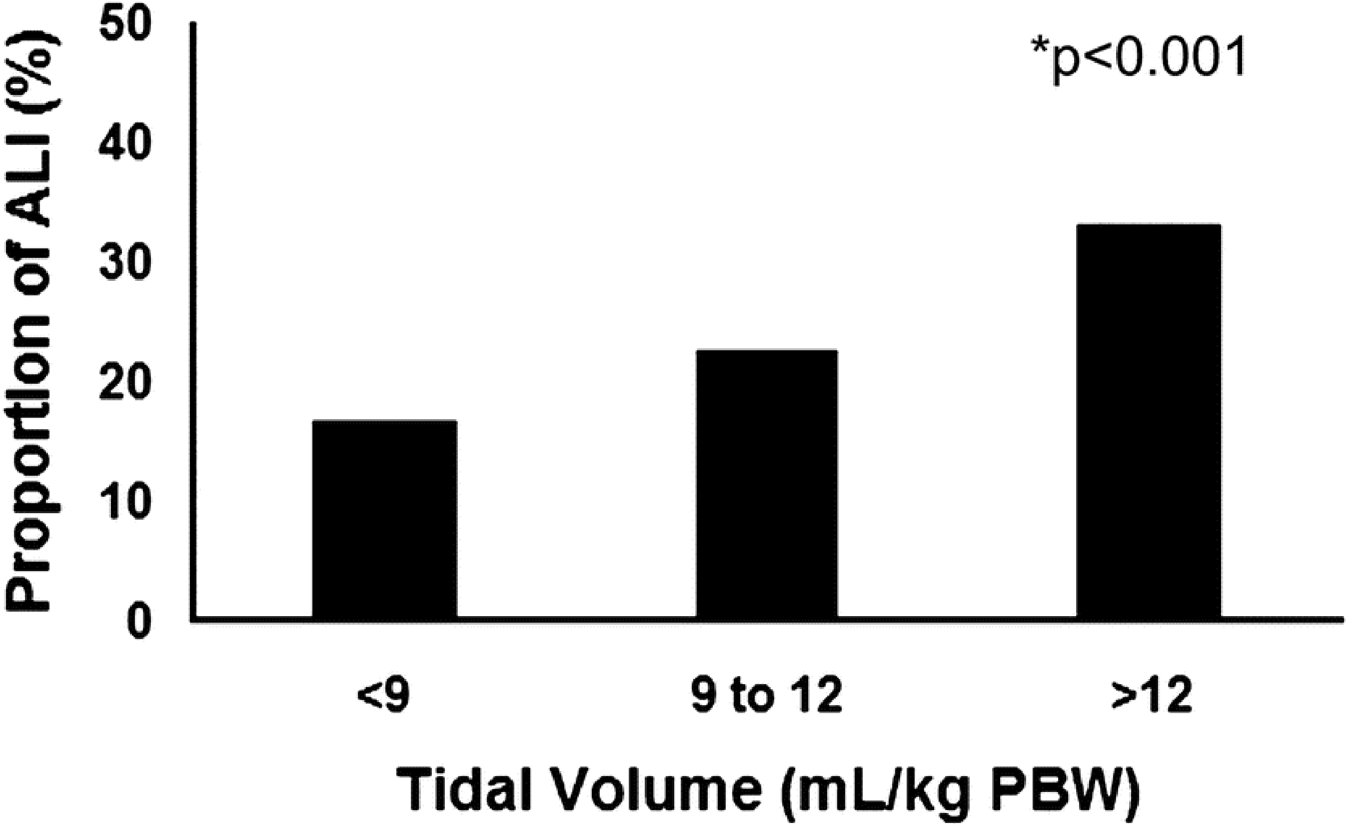

A
B
Mechanical ventilation
damaged the normal lung
Total recovery of previous ARDS

Strategy

- Low volume ventilation:
  - Set tidal volume of 6 mL/kg
    (ARDS Network. NEJM 2000;342:1301-8)
  - Mortality was reduced by 22%

- Is PCV better than VCV ?
  - Clinical trials did not demonstrate the difference

In patients without ARDS, low-tidal-volume ventilation lower the risk of developing acute lung injury



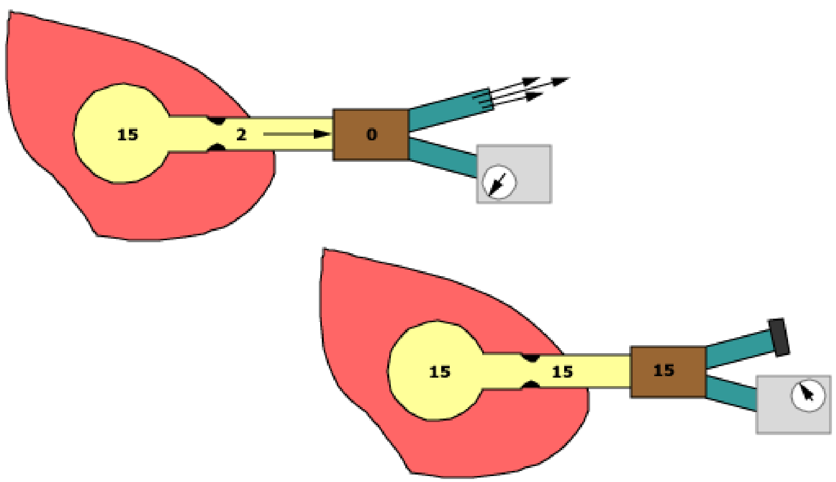
Critical Care Medicine: Volume 32(9) 2004

Auto-PEEP (Intrinsic PEEP)

- **Causes**
  - High minute ventilation
  - Prolonged inspiratory time
  - Time-constant inequality
  - Expiratory flow limitation
- **Consequences**
  - Exacerbates the hemodynamic effects of PPV
  - Increases the risk of barotrauma
  - Increases the dead space
  - Makes it difficult for patients to trigger a ventilator-assisted breath

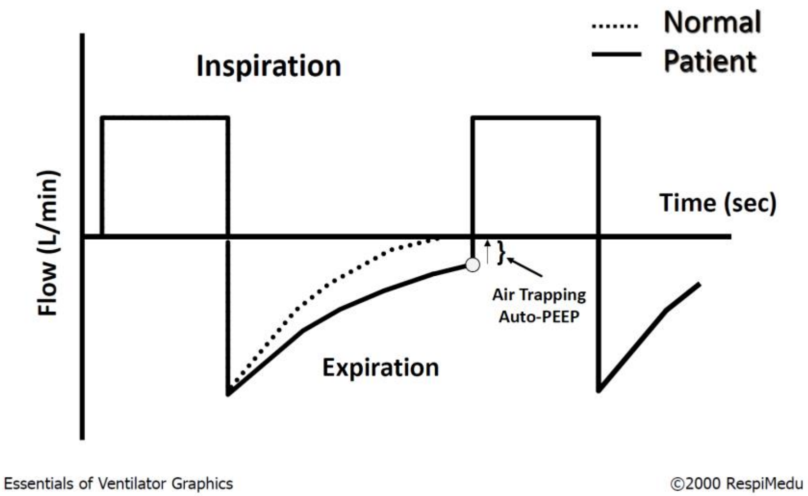
Detection of Auto-PEEP

- Directly measuring the airway pressure at the end of an expiratory breath hold (0.5-1 sec)



O'Quinn, R, Marini, JJ, Am Rev Respir Dis 1983; 128:319

- Ventilator waveforms



- Auscultation or abdominal palpation
  - Continued expiratory airflow from the preceding breath when the next breath is triggered
  - PPV: 95%; NPV 58%

Strategy

- **Adjust ventilator settings**
  - Increase expiratory time
  - Increase inspiratory flow
  - Decrease the tidal volume
  - Decrease the respiratory rate
  - Use applied PEEP to overcome auto-PEEP
- **Reduce ventilatory demand**
  - Low carbohydrate intake
  - Treat anxiety, pain, and fever
- **Reduce the expiratory flow resistance**
  - Suction, bronchodilator, large endotracheal tube…

Atelectrauma

- Lung injury associated with repeated recruitment and collapse, theoretically prevented by using a level of positive end-expiratory pressure greater than the lower inflection point of the pressure volume curve.
- Sometimes called low volume or low end-expiratory volume injury

Lancet 2003; 361: 332–340

Atelectrauma

- ***Repetitive opening / collapse of lung units***

- **Mechanisms:**
  - Recuritment / de-recruitment (shearing force)
  - Reexpansion of atelectatic lung cause increased regional stress
  - Decreased alveolar PaO_2

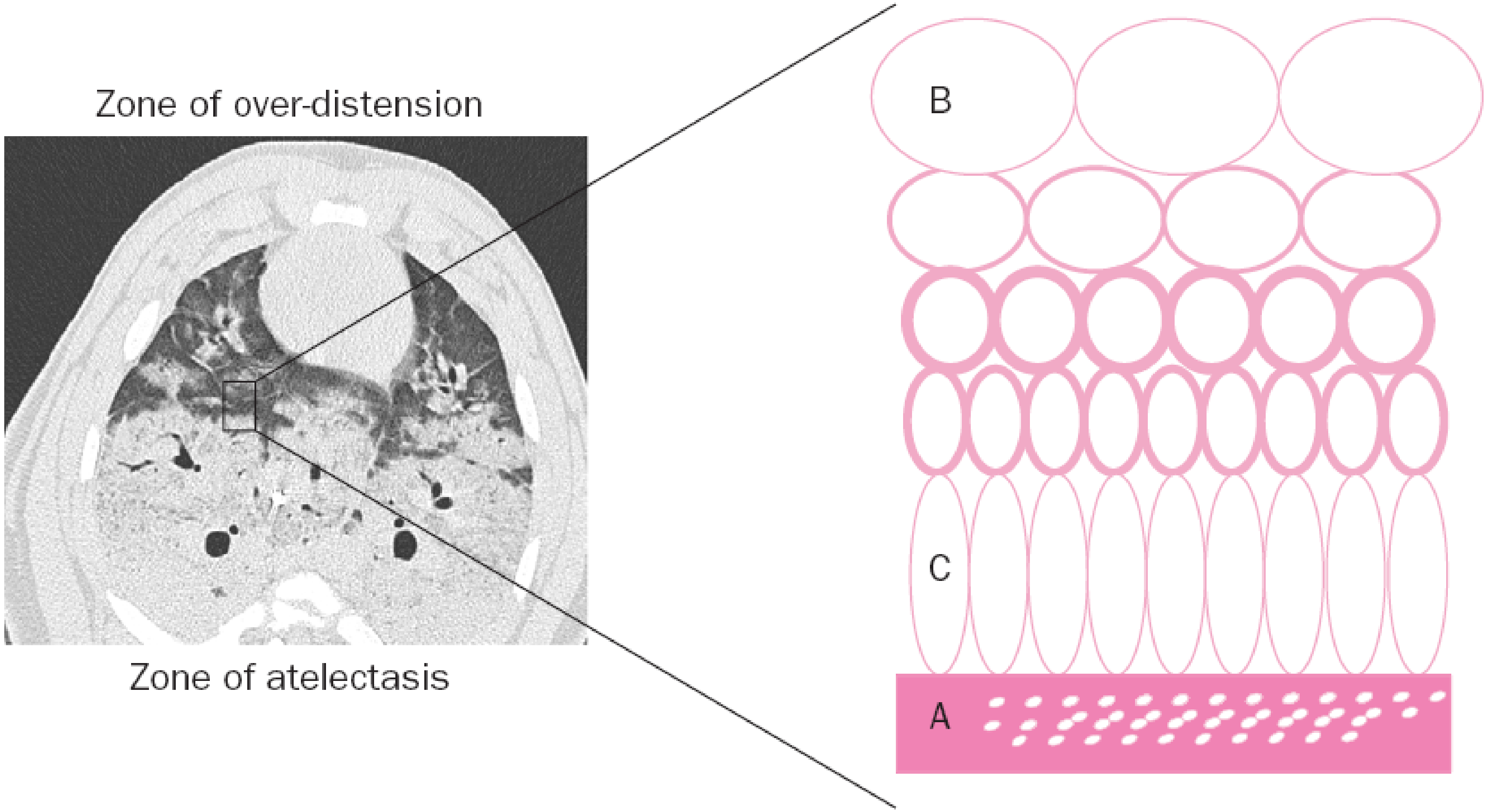


Figure 1: **Atelectotrauma**

The interface between collapsed and consolidated lung (A) and over-distended lung units (B) is heterogeneous and unstable. Depending on ambient conditions this region is prone to cyclic recruitment and derecruitment and localised asymmetrical stretch of lung units (C) immediately apposed to regions of collapsed lung.

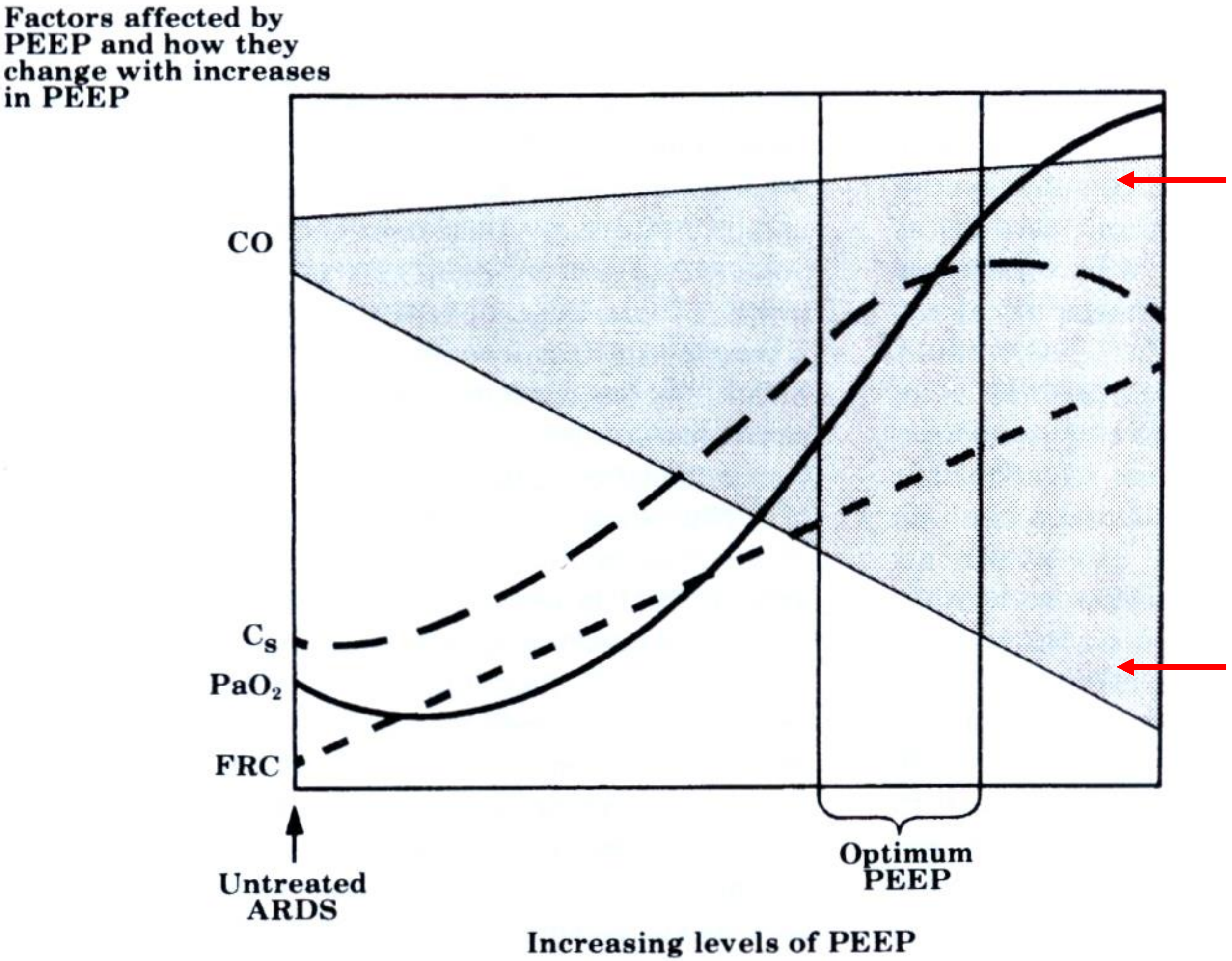
Lancet 2003; 361: 332–340

Strategy

- Use **Recruitment Maneuvers** to open the lung
- Use **higher PEEP** to maintain the lung open (open lung ventilation)

- How to set the best PEEP:
  - 2 cm H_2O above the lower inflection point (LIP) of pressure-volume cure
  - Matches with FiO_2 as protocol of ARDS Network
  - Increasing PEEP while observing Vt, P/F, and BP
  - Apply a larger PEEP, then reducing PEEP while observing PaO_2 (or SaO_2)

Optimal PEEP



High PEEP may increase or decrease the cardiac output

PEEP increase the lung compliance

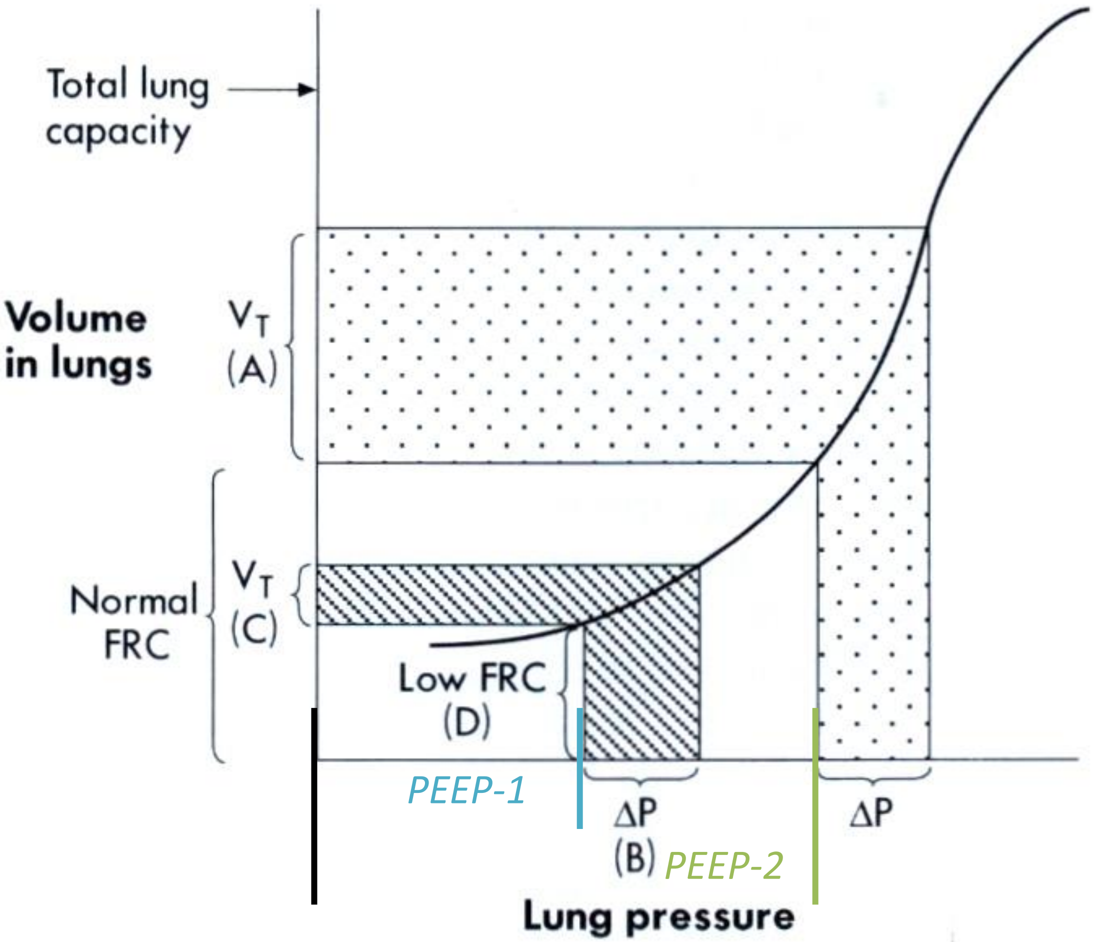


Table 1. **Settings for Positive End-Expiratory Pressure (PEEP), According to the Required Fraction of Inspired Oxygen (F_IO_2).***

F_IO_2	PEEP
0.3	5
0.4	5–8
0.5	8–10
0.6	10
0.7	10–14
0.8	14
0.9	14–18
1.0	18–24

* Settings are from the ARDSNet trial.[19] The required F_IO_2 is the lowest value that maintains arterial oxyhemoglobin saturation above 90%. After the corresponding level of PEEP is selected, arterial oxyhemoglobin saturation and plateau airway pressure should be monitored in the patient.

Malhotra A. NEJM, 2009

Suggested PEEP

FiO_2 = 30-39%：PEEP = 5-8 cm H_2O
FiO_2 = 40-49%：PEEP = 8-12 cm H_2O
FiO_2 = 50-59%：PEEP = 10-14 cm H_2O
FiO_2 = 60-79%：PEEP = 12-16 cm H_2O
FiO_2 = 80-100%：PEEP = 14-18 cm H_2O

KMUH Protocol

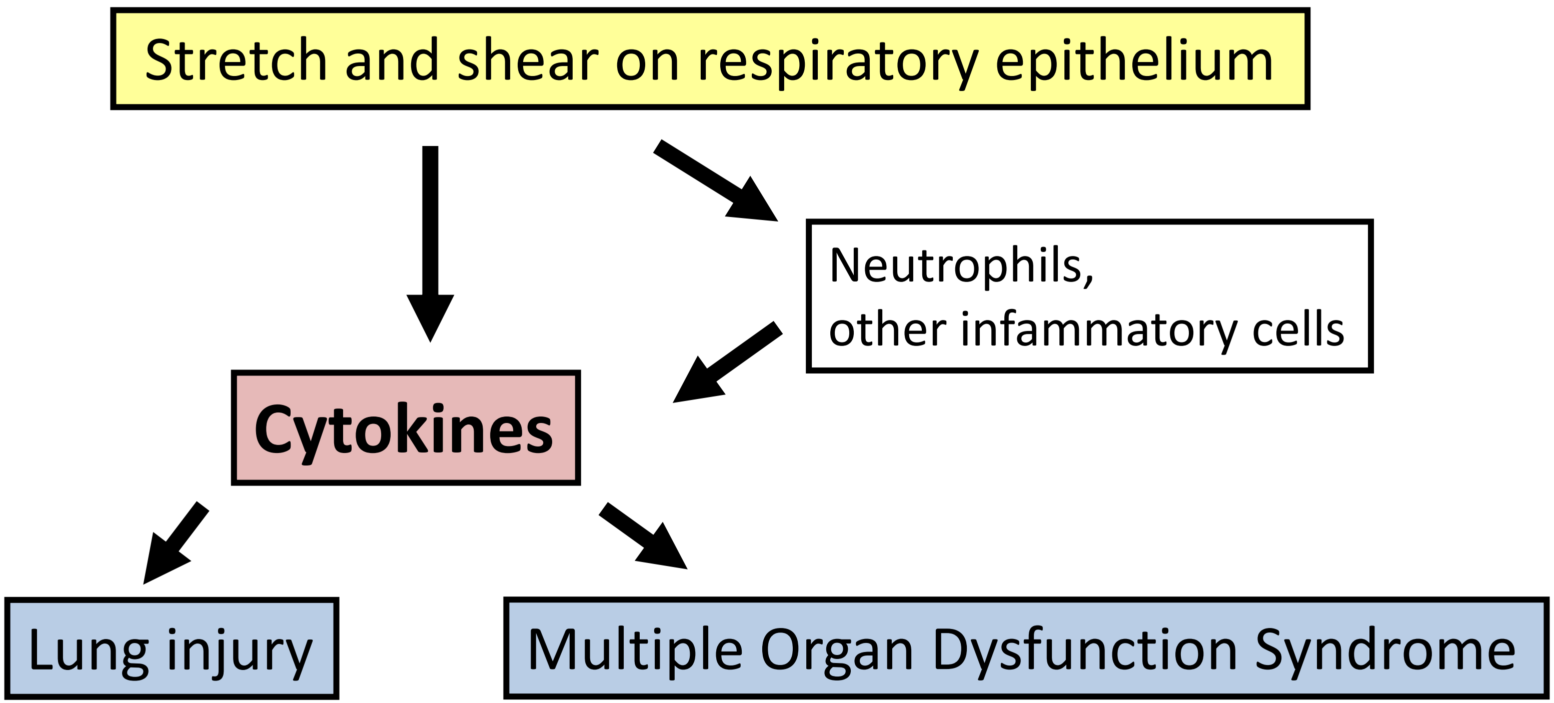
Biotrauma

- Pulmonary and systemic inflammation caused by the release of mediators from lungs subjected to injurious mechanical ventilation

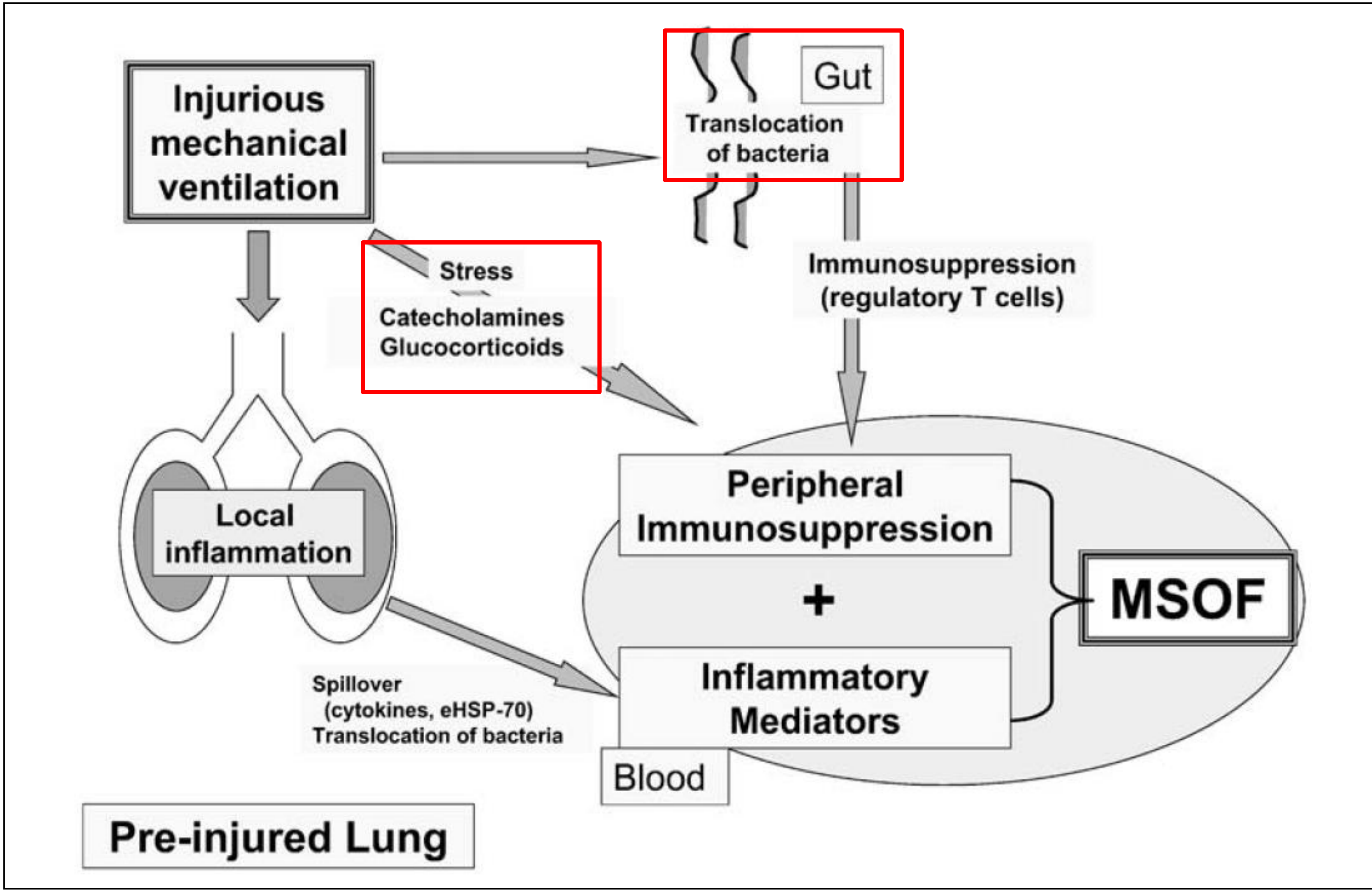
Lancet 2003; 361: 332–340

Biotrauma

- Mechanical factors can lead to injury by inflammatory mediators



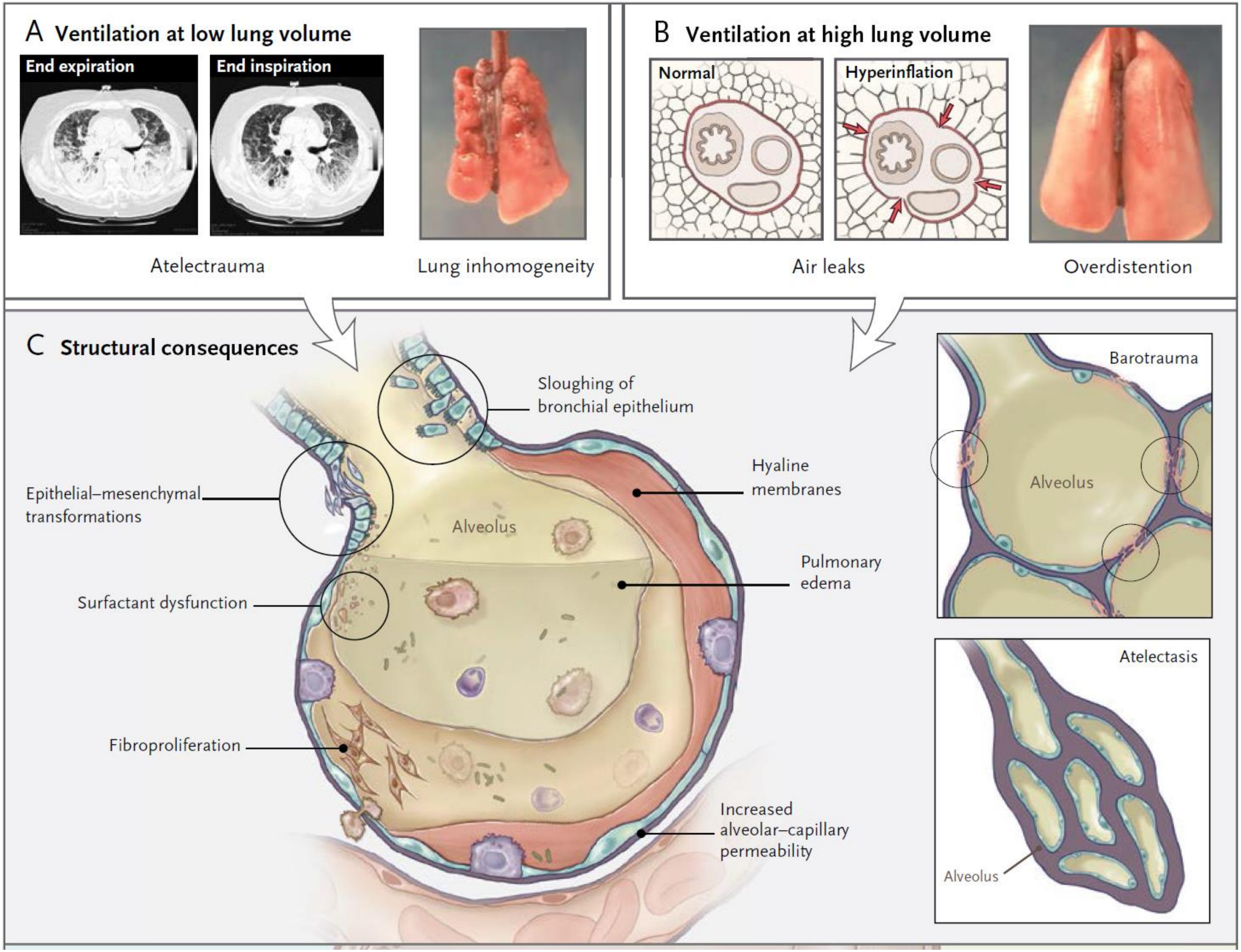
Proposed Mechanisms for Biotrauma



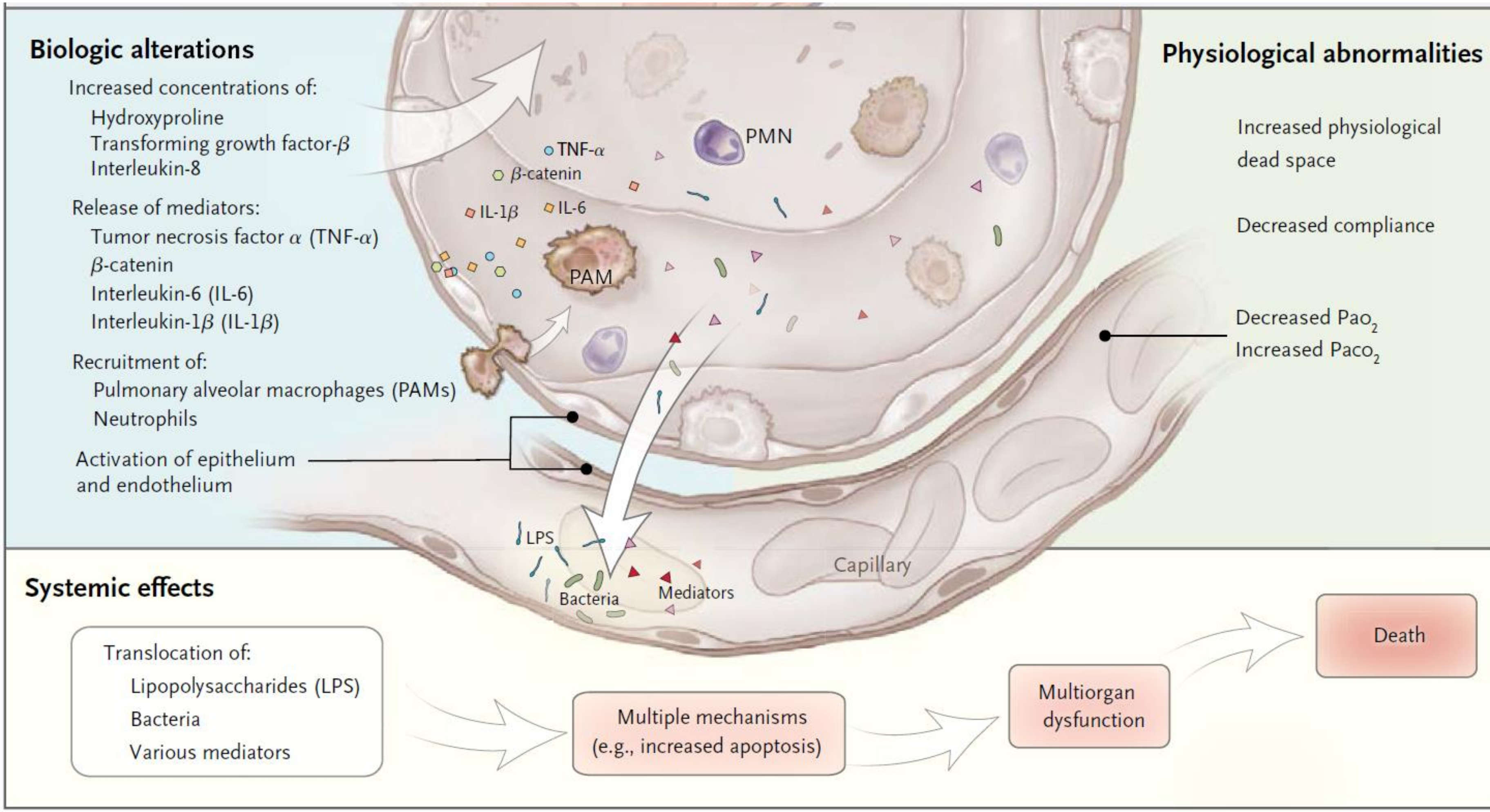
Intensive Care Med 2004; 30:1865–1872

Strategy

- **Lung-protective ventilatory strategy**
  - Low volume
  - Low pressure
  - Low FiO_2
  - Adequate PEEP



NEJM 2013; 369: 2126-2136



NEJM 2013; 369: 2126-2136

VIDD

- **Causes of Decreased Strength in Weaning-Failure Patients**
  - Neuromuscular blockers
  - Neuromuscular disorders (critical illness polyneuropathy)
  - Hyperinflation
  - Shock and ongoing sepsis
  - Malnutrition and electrolyte disturbances
  - **Ventilator-induced diaphragmatic dysfunction (VIDD) that means: disuse atrophy of diaphragm**

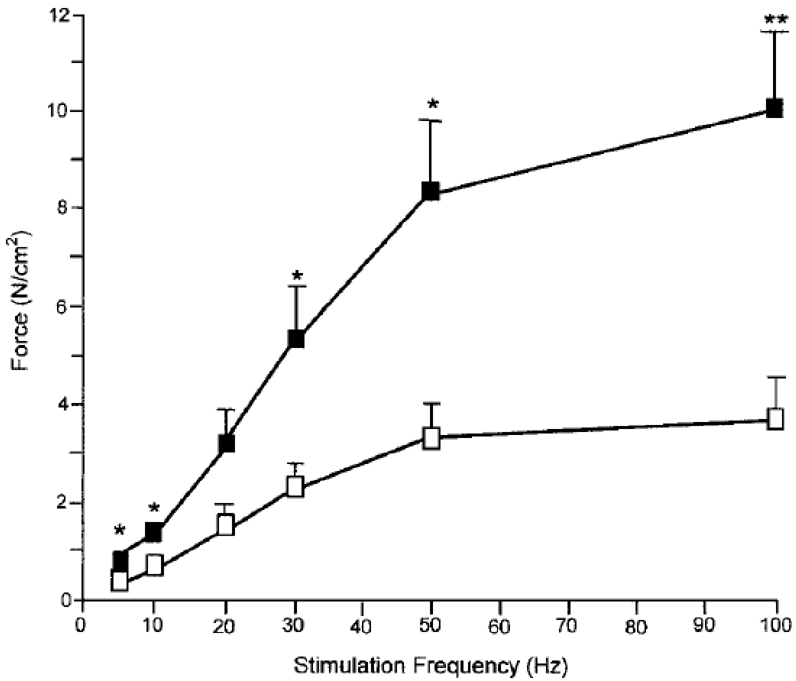


Fig. 1. Diaphragmatic force-versus-frequency curves obtained from mechanically ventilated animals (open boxes) and in control animals (black boxes). Controlled mechanical ventilation generated less force at all frequencies except 20 Hz. (From Reference 16, with permission.)

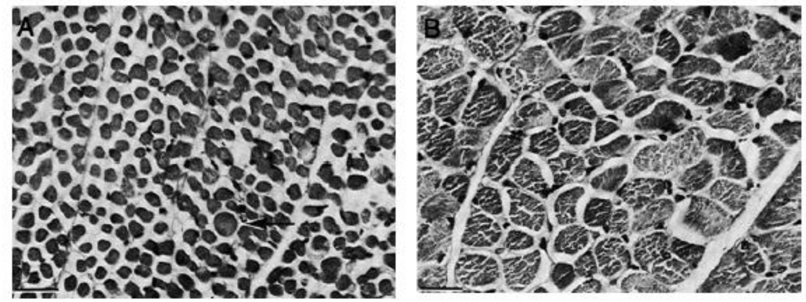


Fig. 3. Diaphragmatic myofibers from an infant ventilated for 47 days (left) and an infant ventilated for 3 days (right) until death. Small myofibers with rounded outlines were seen in the infant who received prolonged mechanical ventilation. (From Reference 25, with permission.)

Petrof BJ et al, Current Opinion in Criitcal Care, 2010

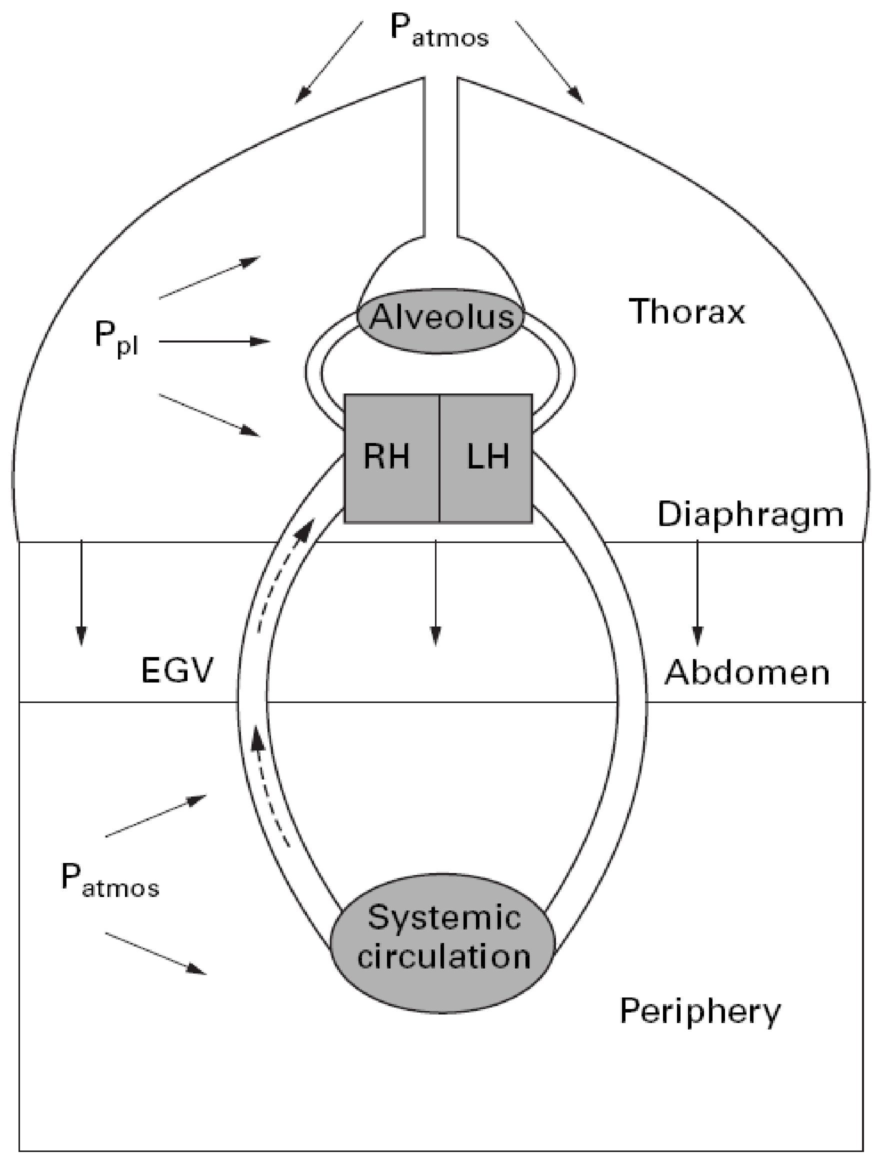
Strategy

- Limit the use of controlled ventilation as possible
- Try to let patients breathe on their own
- Pharmacologic agents in the future?

Cardiovascular Effects and Complications

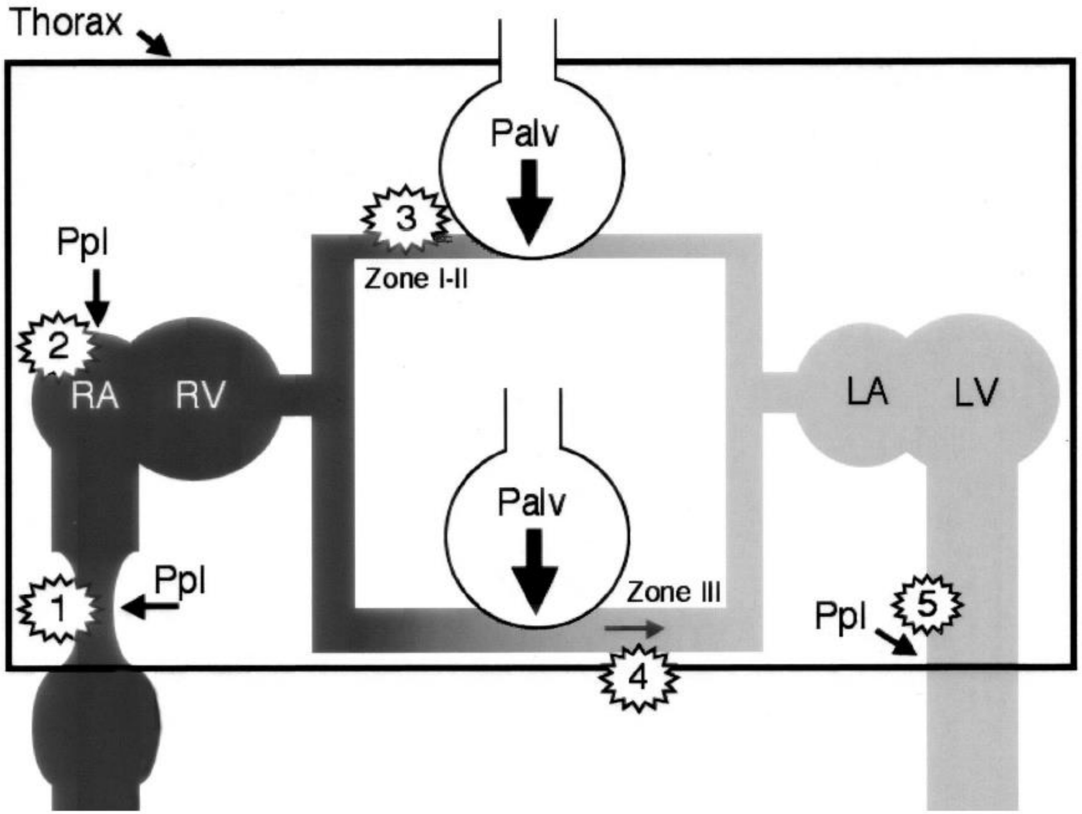
Change in Intrathoracic Pressure
Change in Lung Volume

Figure 1 Model of the circulation, showing factors that influence systemic venous drainage. The right heart (RH) and intrathoracic great veins are subjected to pleural pressure (P_{pl}), which varies throughout the respiratory cycle. Intra-abdominal pressure increases with inspiratory diaphragmatic descent, and normalises to atmospheric (P_{atmos}) with expiration. Peripheral venous pressure is unaffected by respiration and so remains at atmospheric pressure throughout the respiratory cycle. Systemic venous drainage (broken arrow) depends on a driving pressure gradient between extrathoracic great veins (EGV) and the right atrium, and so during spontaneous respiration is maximised during inspiration as the pleural (and right atrial) pressure falls, and the intra-abdominal (and therefore EGV) pressure rises.



Arch Dis Child. 1999;80:475-80.

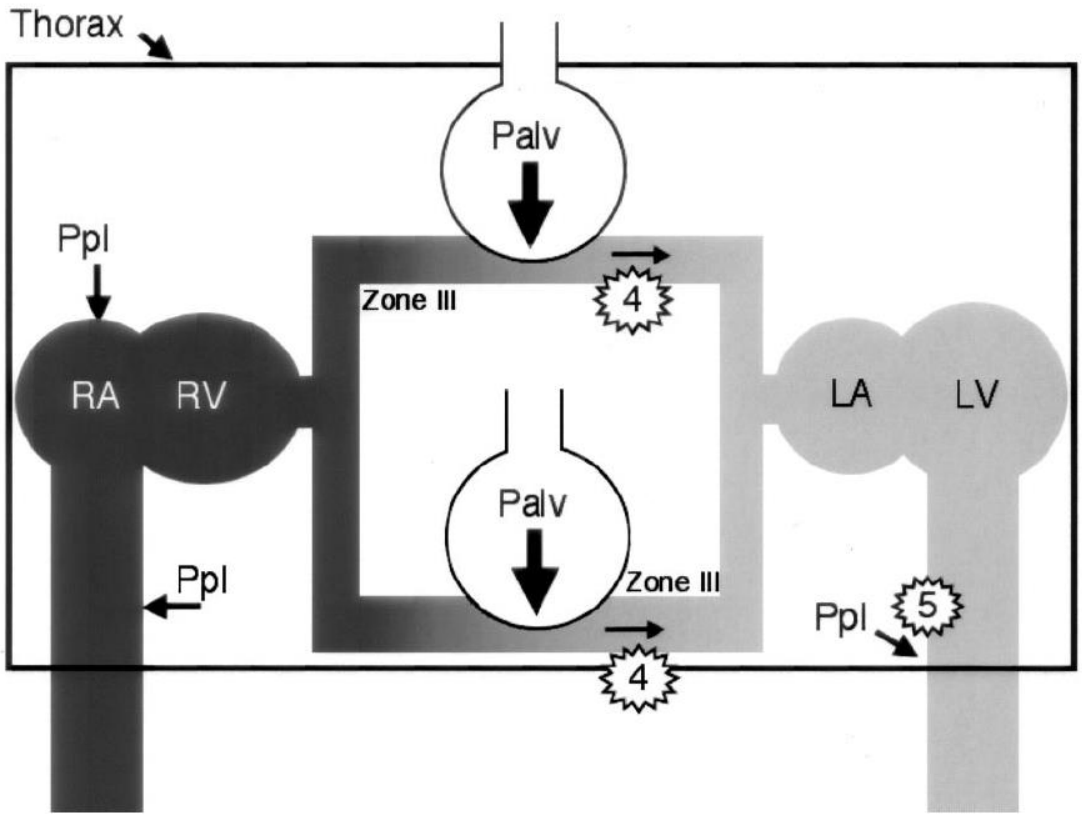
Physiologic effects of MV in hypovolemic conditions



(1,2) RV preload decreases because the increase in pleural pressure induces a compression of the SVC and an increase in intramural RA pressure.
(3) In West zones I (pulmonary arterial pressure < alveolar pressure) and II (pulmonary venous pressure < alveolar pressure), RV afterload increases because pulmonary capillaries are compressed.
(4) In West zones III (alveolar pressure < pulmonary venous pressure), the increase in alveolar pressure squeezes out the blood contained in the capillaries toward the left side of the heart.
(5) The increase in pleural pressure induces a decrease in LV afterload.

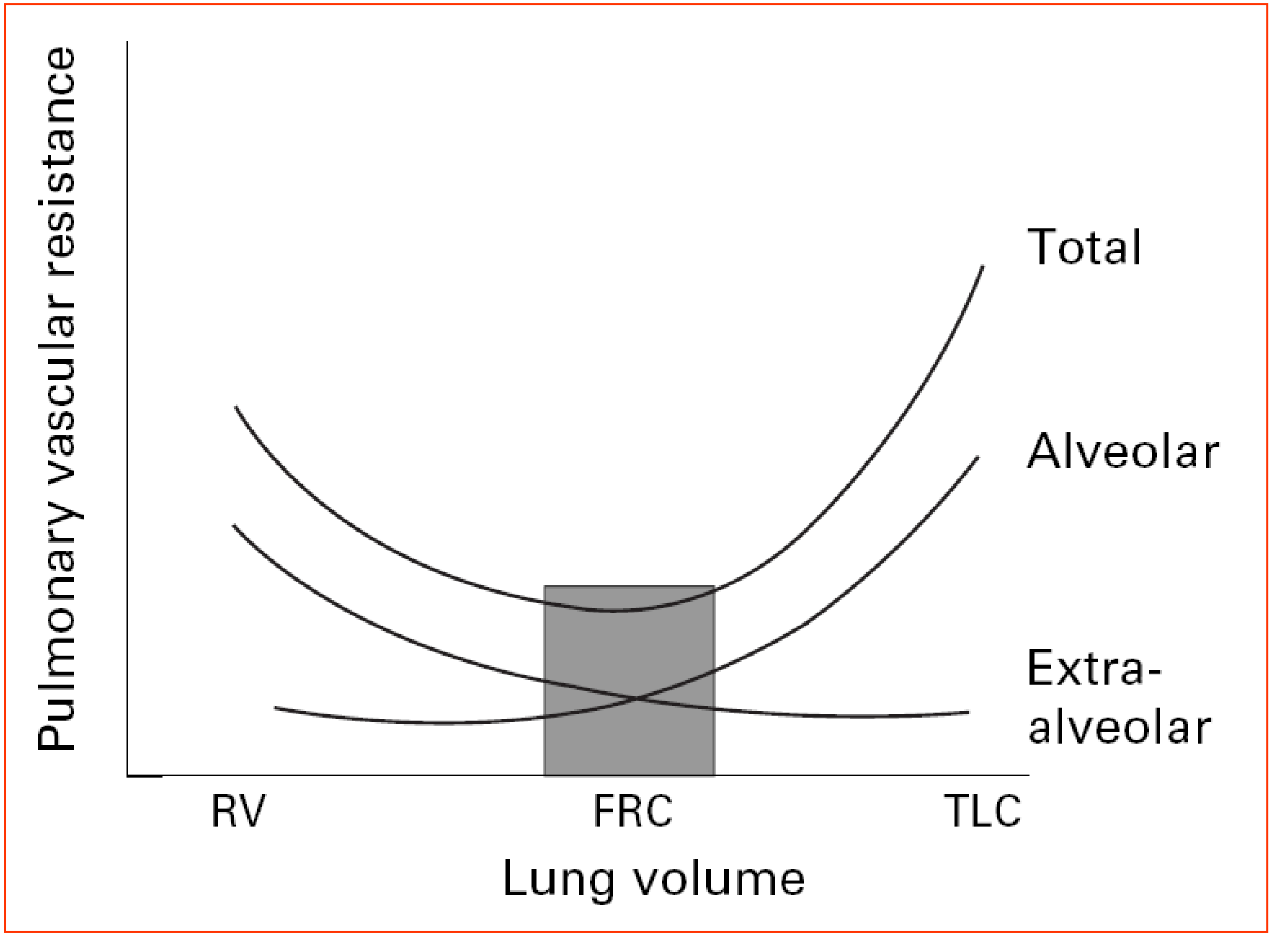
Anesthesiology 2005;103:419-28

Physiologic effects of MV in hypervolemic conditions



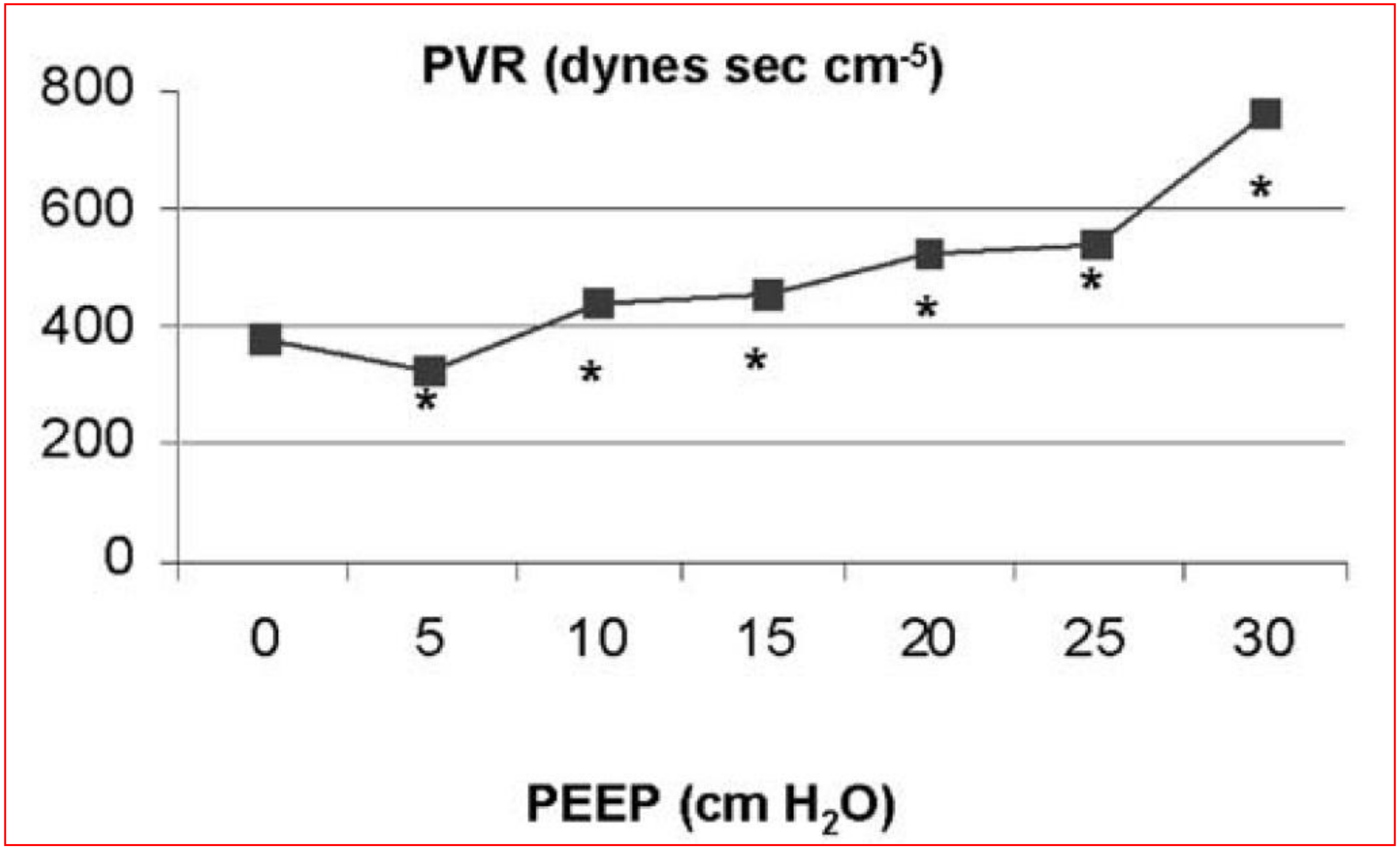
The vena cava and right atrium are poorly compliant and compressible and hence relatively insensitive to changes in pleural pressure.
(4) West zones III (alveolar pressure < pulmonary venous pressure) are predominant in the lungs such that each mechanical breath increases pulmonary venous flow and left ventricular preload.
(5) The increase in pleural pressure induces a decrease in left ventricular afterload.

Anesthesiology 2005;103:419-28



As lung volume increases from residual volume (RV) to total lung capacity (TLC), the alveolar vessels become increasingly compressed by the distending alveoli, and so their resistance increases, whereas the resistance of the extra-alveolar vessels (which become less tortuous as lung volume increases) falls. The combined effect of increasing lung volume on the pulmonary vasculature produces the typical "U shaped" curve as shown, with its nadir, or optimum, at around normal functional residual capacity (FRC).

Arch Dis Child. 1999;80:475-80.



Pulmonary vascular resistance increase with PEEP

Hemodynamic Effects of Positive Pressure Ventilation

↓ ↓

BP= CO X SVR

↑ ↓

HR X SV

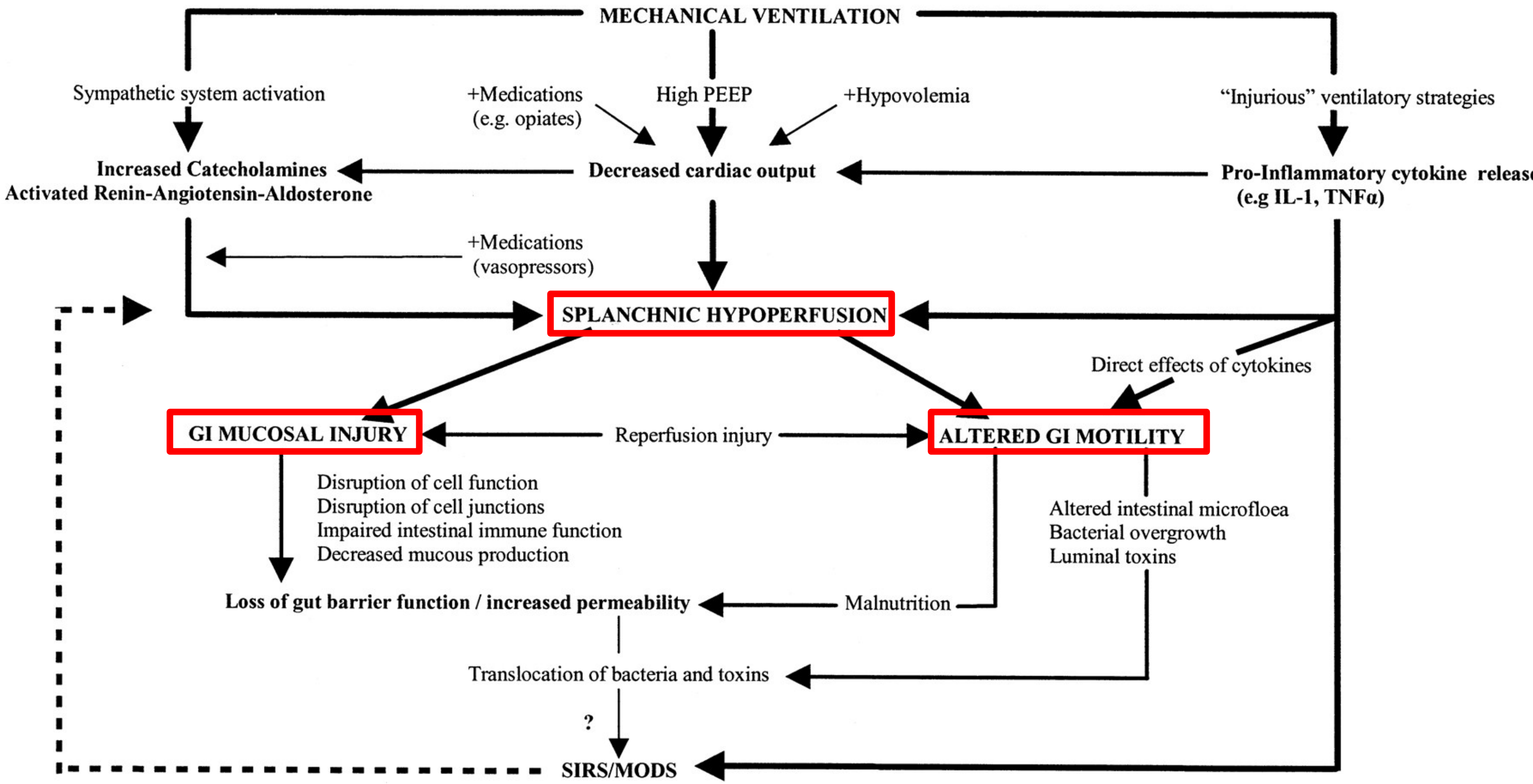
Preload ↓

Contractility

Afterload ↑ **RV** ↓ **LV**

Effects and Complications on Other Organ System

Gastrointestinal
Liver
Kidney
CNS
Immune System



Chest 119;4 April, 2001

Complications	Incidence (%)
Erosive esophagitis	48
SRMD	
Asymptomatic, (endoscopically evident damage)	74–100
Clinically evident bleeding	5–25
Clinically significant bleeding	3–4
Diarrhea	15–51
Decreased bowel sounds	50
High gastric residuals	39
Constipation	15
Ileus	4–10
AAC	0.2–3

Chest 119;4 April, 2001

Common GI Complications of Mechanical Ventilation

- ***Stress Related Mucosal Damage (SRMD)***
  - GI bleeding
- ***Severe Gastric Distention***
  - Possibly from swallowing air that leaks around ET-tube cuffs or when PPV is delivered by mask
- ***Hypomotility***
  - Decreased bowel sound, high gastric residuals, constipation, ileus
- ***Diarrhea***

Prophylactic Treatment of SRMD

- ***Treat the underlying diseases***
- ***Stabilize the hemodynamics***
- ***Enteral feeding***
- ***Medications:***
  may reduce clinically important bleeding rates by 50%
  - Antacids
  - H_2-blockers
  - Proton pump inhibitors
  - Sucralfate

- *Gastric colonization and VAP are the major concerned complications.*
- *The risk could be reduced by methods of VAP prophylaxis.*

Hypomotility

- **Manifestation:**
  - Decrease bowel sounds
  - Abdominal distention
  - High gastric residuals (vomiting → VAP)
  - Constipation

- These patients had longer ICU stays and higher mortality

Treatment and Prophylaxis of Hypomotility

- **Correction of electrolytes:**
  - Hypokalemia, hypomagnesemia
- **Avoid medications that impaired GI motility**
  - Dopamine, Morphine, Diltiazem, Verapamil, Anticholinergic
- **Prokinetic agents**
  - Metoclopramide
  - Erythromycin
  - Cisapride

Causes of Diarrhea in Patients Receiving MV

- **Enteral nutrition**
  - Hyperosmolar formulas
  - High infusion rates (> 50 mL/h)
  - Dietary lipids
- **Infection**
  - *C. difficile infection*
- **Medications**
  - Antacids (Mg-based)
  - H_2-Receptor antagonists (with or without antacids)
  - Antibiotics
- **Hypoalbuminemia**
  - Particularly those with chronic severe hypoalbuminemia (< 2.6 g/dL)
- **Prolonged fasting (> 5 day)**
  - Interfering with bile acid homeostasis due to intestinal mucosal atrophy

Splanchnic Perfusion

- PPV and PEEP increase splanchnic resistance; decrease splanchnic perfusion.
- Bilirubin, GOT, GPT, LDH levels can become elevated.
- Elevated intraabdominal pressure can lead to profound alterations in respiratory mechanics and hemodynamics.

Liver

- ***Liver function impairment*** *(hyperbilirubinemia)*
  - Decreased cardiac output
  - Downward movement of diaphragm against the liver
  - Decrease in portal venous flow
  - Increase in splanchnic resistance
  - → Liver ischemia

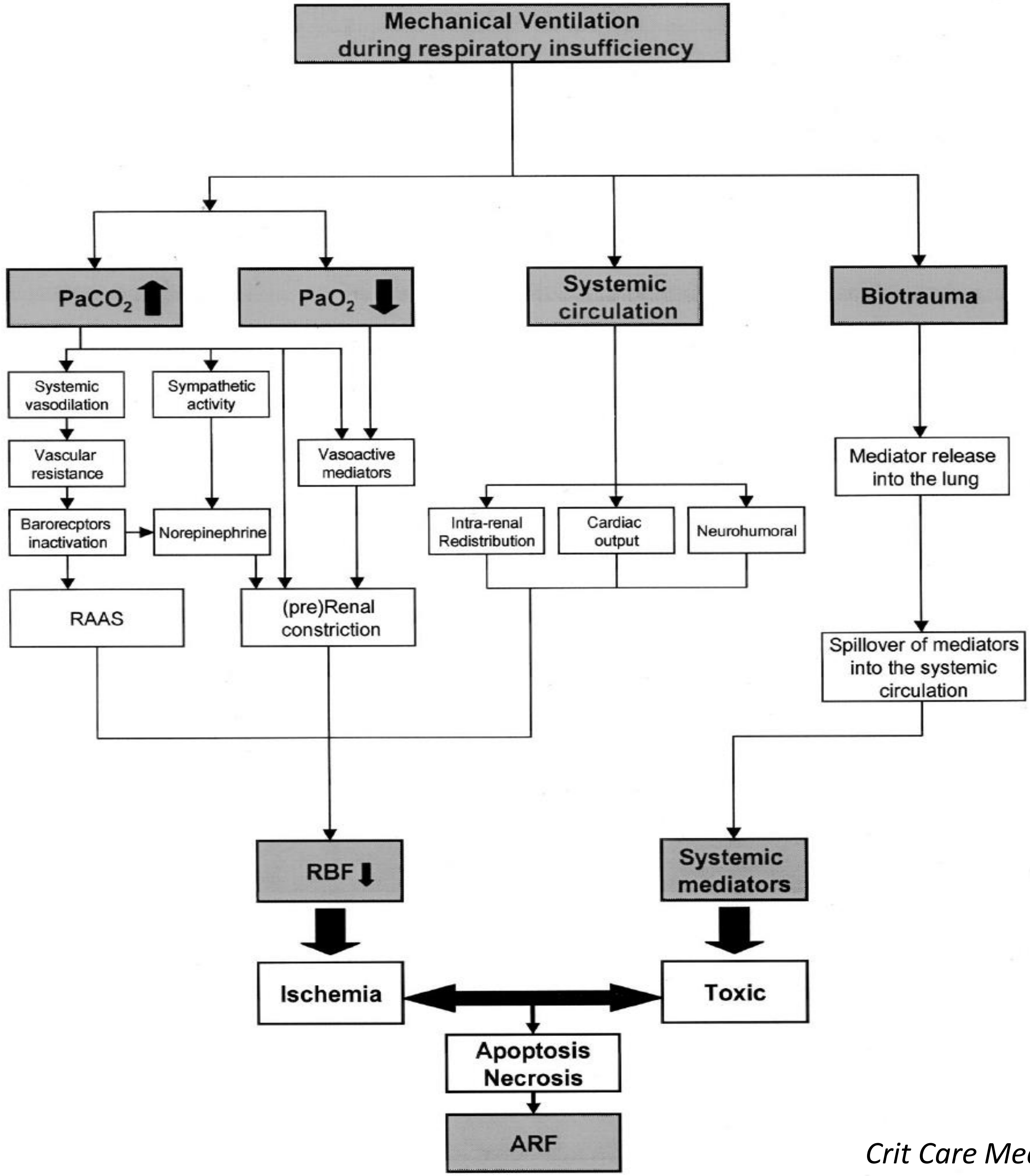
Kidney

- PPV and PEEP
  - → Low cardiac output
  - → Stimulation of the renin-angiotensin system, increased release of antidiuretic hormone, and reduced secretion of atrial natriuretic peptide
  - → **Fluid retention and edema**
- Mechanical ventilation is an independent risk factor for **acute renal failure**

Kidney

- Mechanical ventilation may induce acute tubular necrosis (ATN) leading to acute renal failure (ARF) by three proposed mechanisms:
  - Through effects on **arterial blood gas**
    - $PaO_2 < 40$ mmHg or $PaCO_2 > 65$ mmHg decrease renal function.
  - Through effects on systemic and renal blood flow
  - Through **biotrauma**

Crit Care Med. 2005;33:1408-15



Crit Care Med. 2005;33:1408-15

Strategy

- ***Adequate PaO_2 and $PaCO_2$***
- ***Maintain Cardiac Output***
  - Adequate hydration
  - Optimal PEEP
  - Limited plateau pressure
- ***Avoid Biotrauma***
  - Lung protective strategy

CNS

- **PPV and PEEP**
  - → Elevated intrathoracic pressure
  - → Elevated CVP
  - → Diminished cerebral venous outflow
  - → Increased intracranial pressure

CPP = Cerebral Perfusion Pressure

MAP = Mean Arterial Pressure

ICP = Intracranial Pressure

Mechanical Ventilation

CPP = MAP - ICP

↓ ↓ ↑

**The amount of blood flow to the brain is determined by CPP*

Mechanical Ventilation in Patients with Increased ICP

- Lower $PaCO_2$ to 30-35 mmHg
- Alkalosis from low $PaCO_2$ can constrict cerebral vessels
- This effect only appears to last for 24-36 hours
- No longer widely used.

- *PEEP can increase ICP, but it may be lifesaving and should be used. It's important to monitor the ICP in this patient group.*

Treatment of Increased ICP

Therapy Steps	Levels of Evidence	Therapy	Risk
8	Not reported	Decompressive craniectomy	Infection or delayed hematoma Subdural effusion Hydrocephalus and syndrome of the trephined
7	Level II	Metabolic suppression (barbiturates)	Hypotension and increased number of infections
6	Level III	Hypothermia	Fluid and electrolyte disturbances and infection
5	Level III	Induced hypocapnia	Excessive vasoconstriction and ischemia
4	Level II	Hyperosmolar therapy Mannitol or hypertonic saline	Negative fluid balance Hypernatremia Kidney failure
3	Not reported	Ventricular CSF drainage	Infection
2	Level III	Increased sedation	Hypotension
1	Not reported	Intubation Normocarbic ventilation	Coughing, ventilator asynchrony, ventilator-associated pneumonia

NEJM 2014; 370: 2121-2130

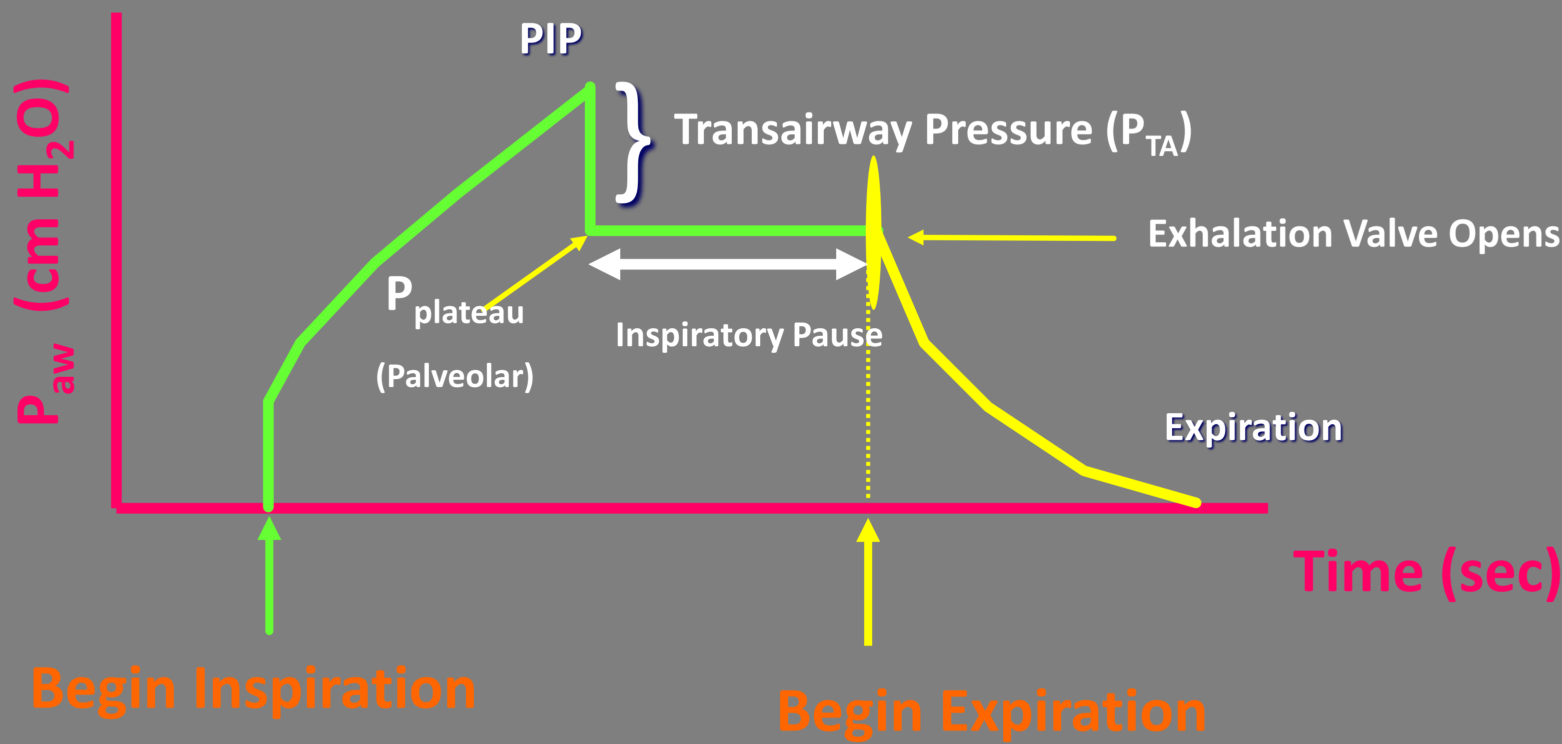
Immune System

- **Biotrauma**
  - higher concentrations of inflammatory mediators in blood and BAL fluid.
- **PPV and PEEP**
  - promote the translocation of intrapulmonary bacteria into the bloodstream.
  - promote the translocation of bacteria from the gut into the bloodstream.

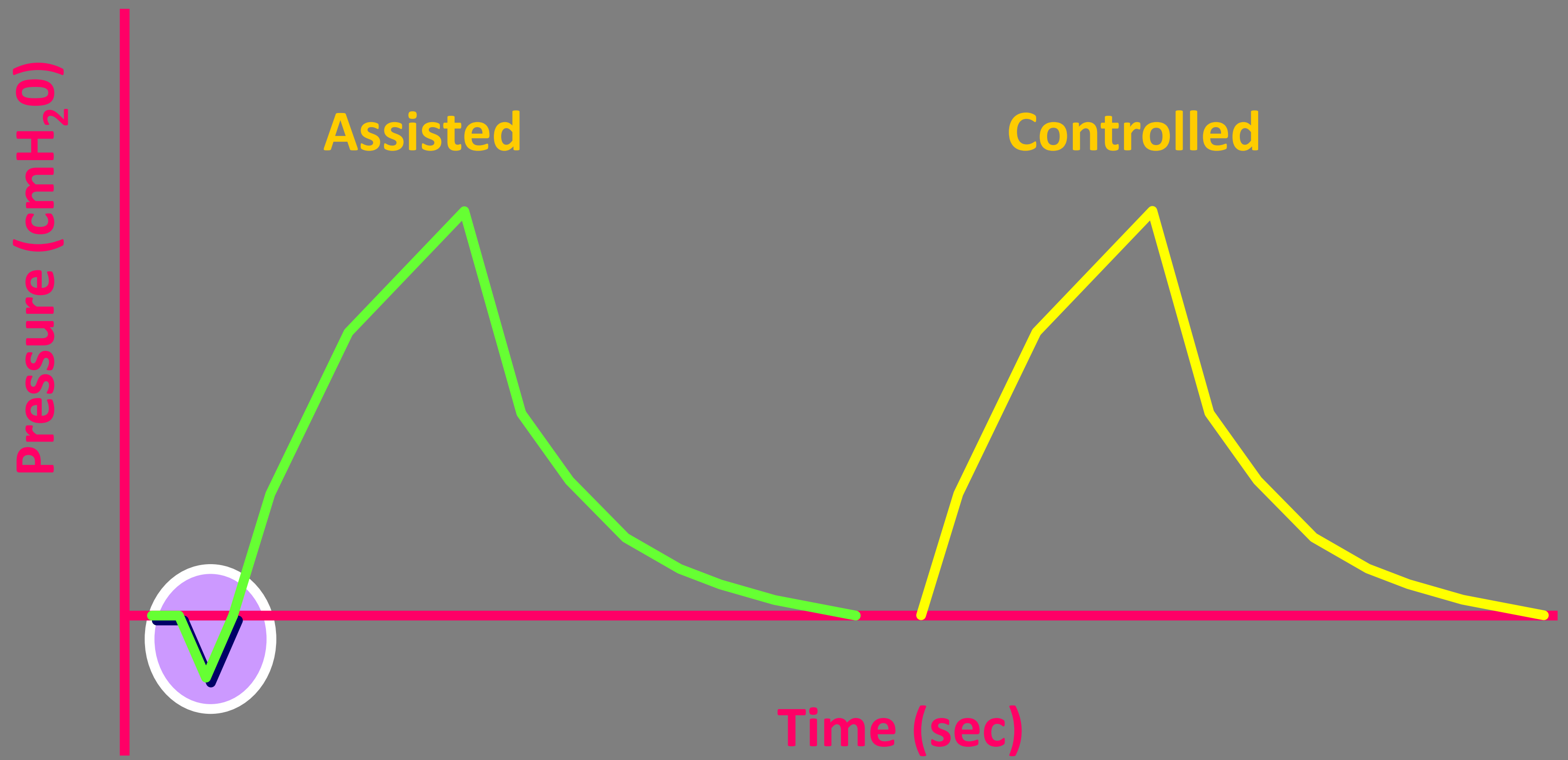
Ventilator Waveforms

- **SCALARS**
  - Flow-time
  - Pressure-time
  - Volume-time
- **LOOPS**
  - Pressure-Volume
  - Flow-Volume

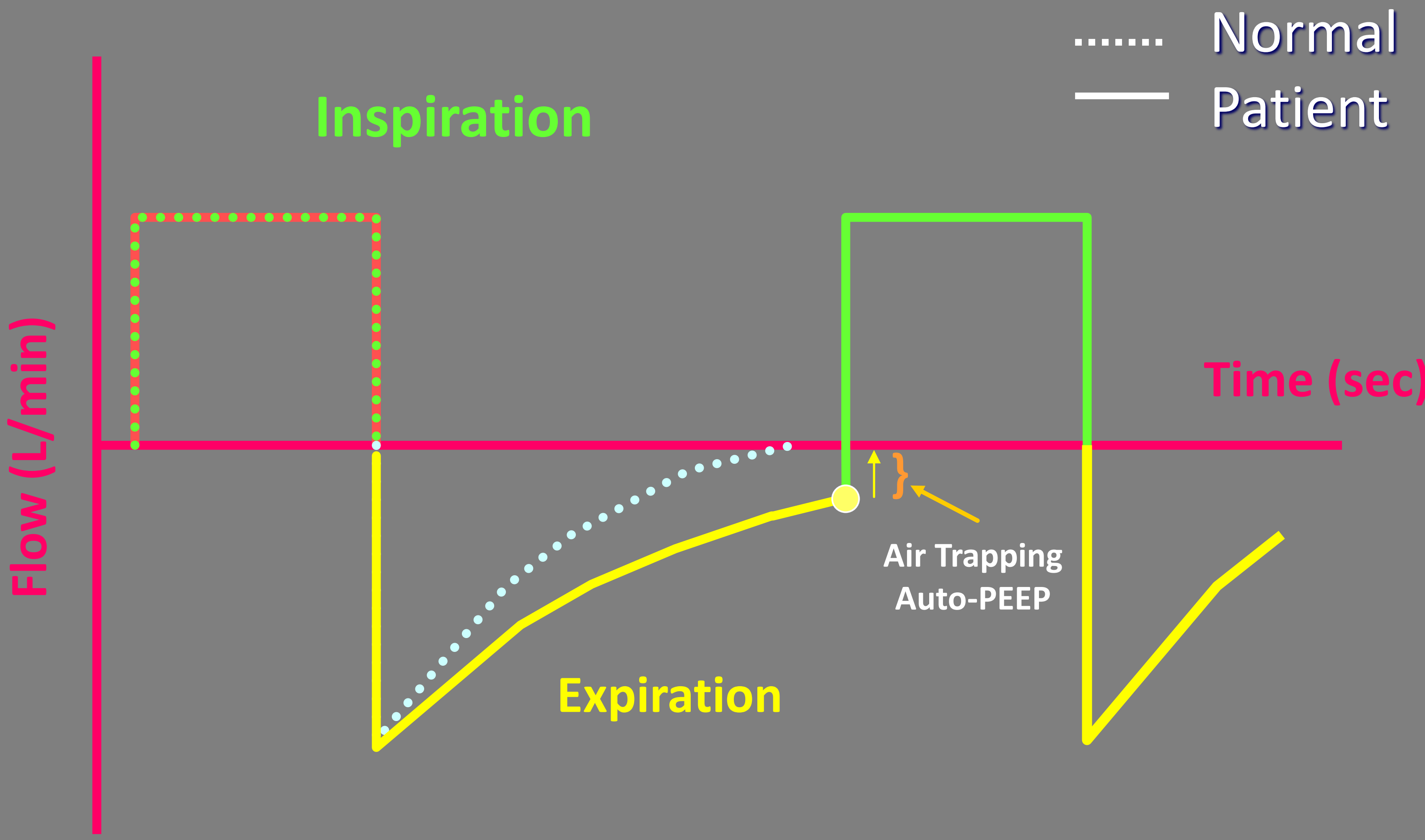
Components of Inflation Pressure



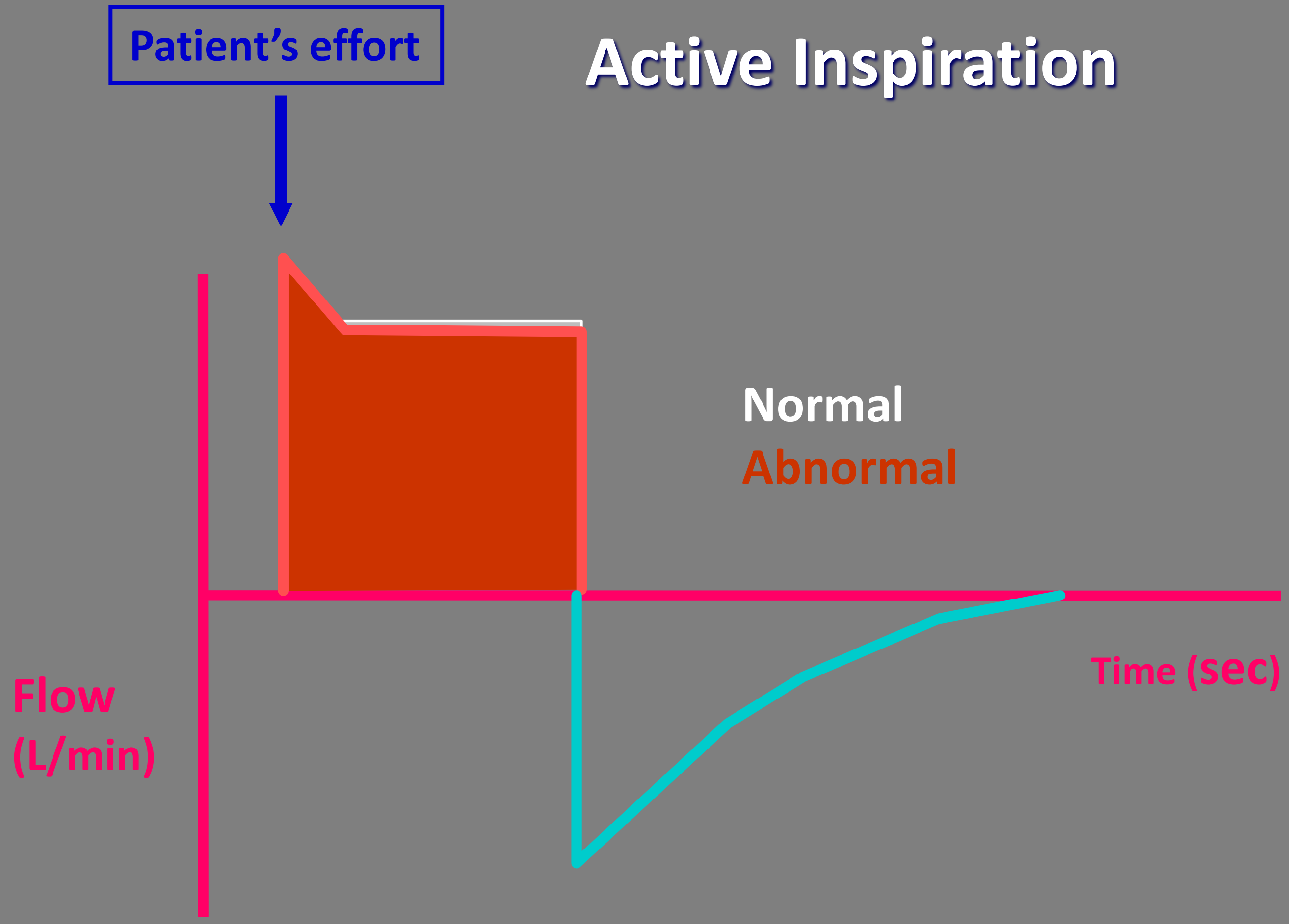
Assisted vs Controlled



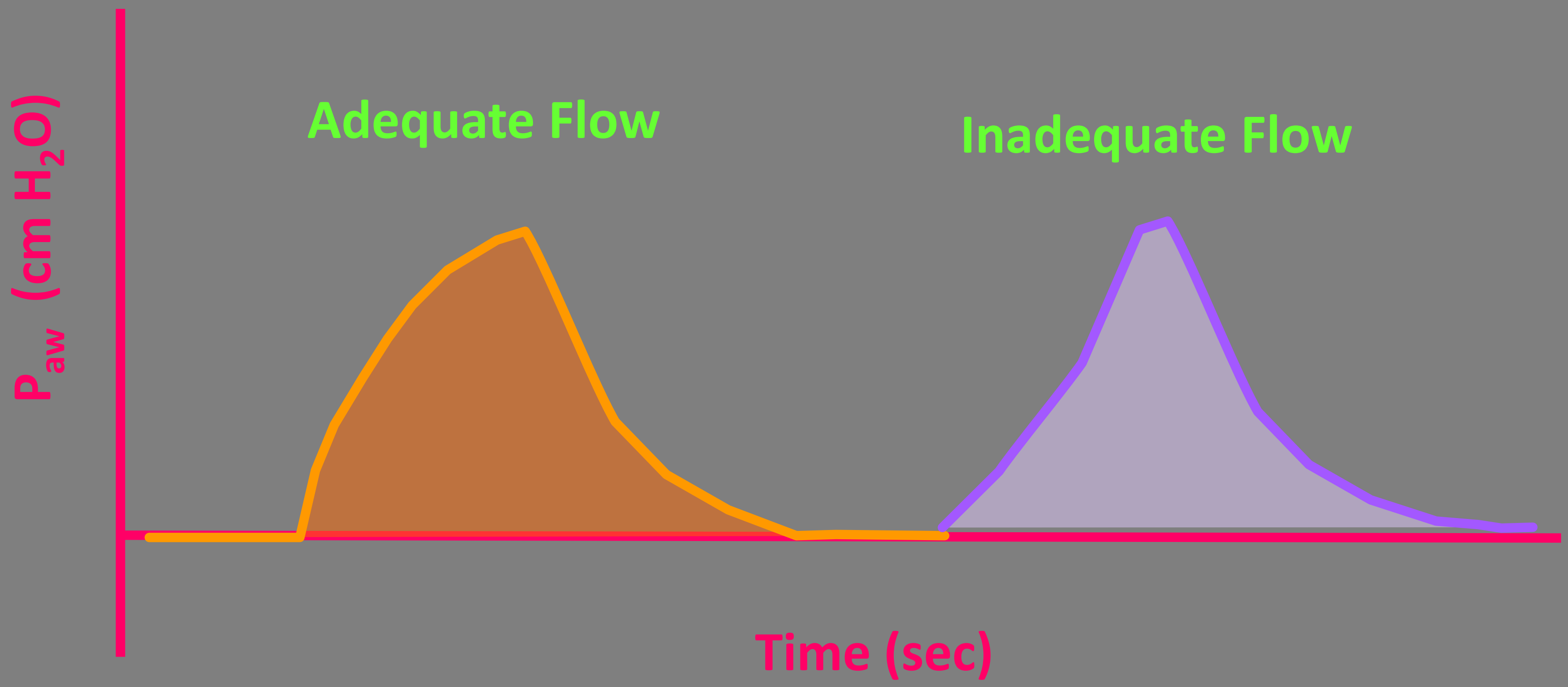
Air Trapping



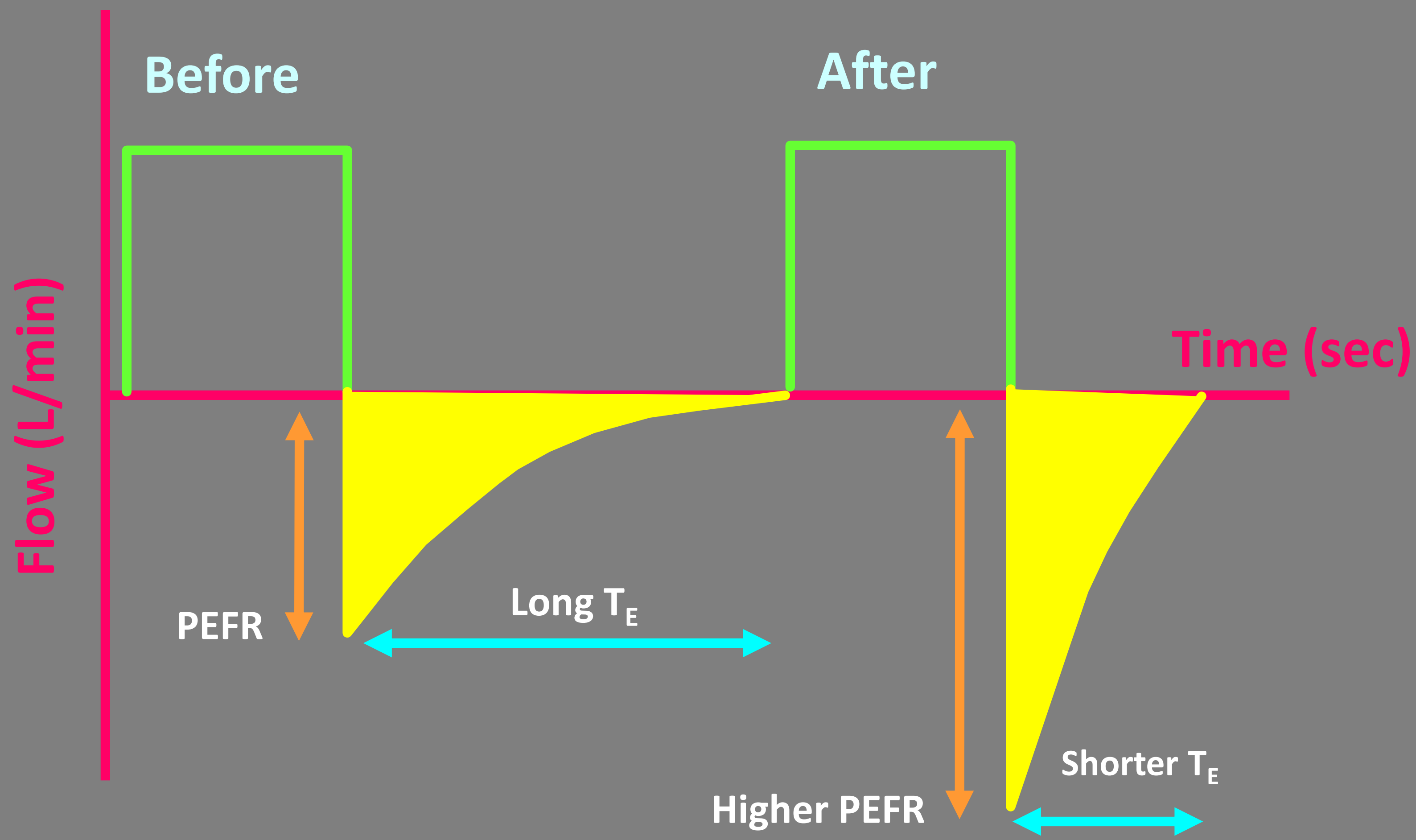
Inadequate Inspiratory Flow



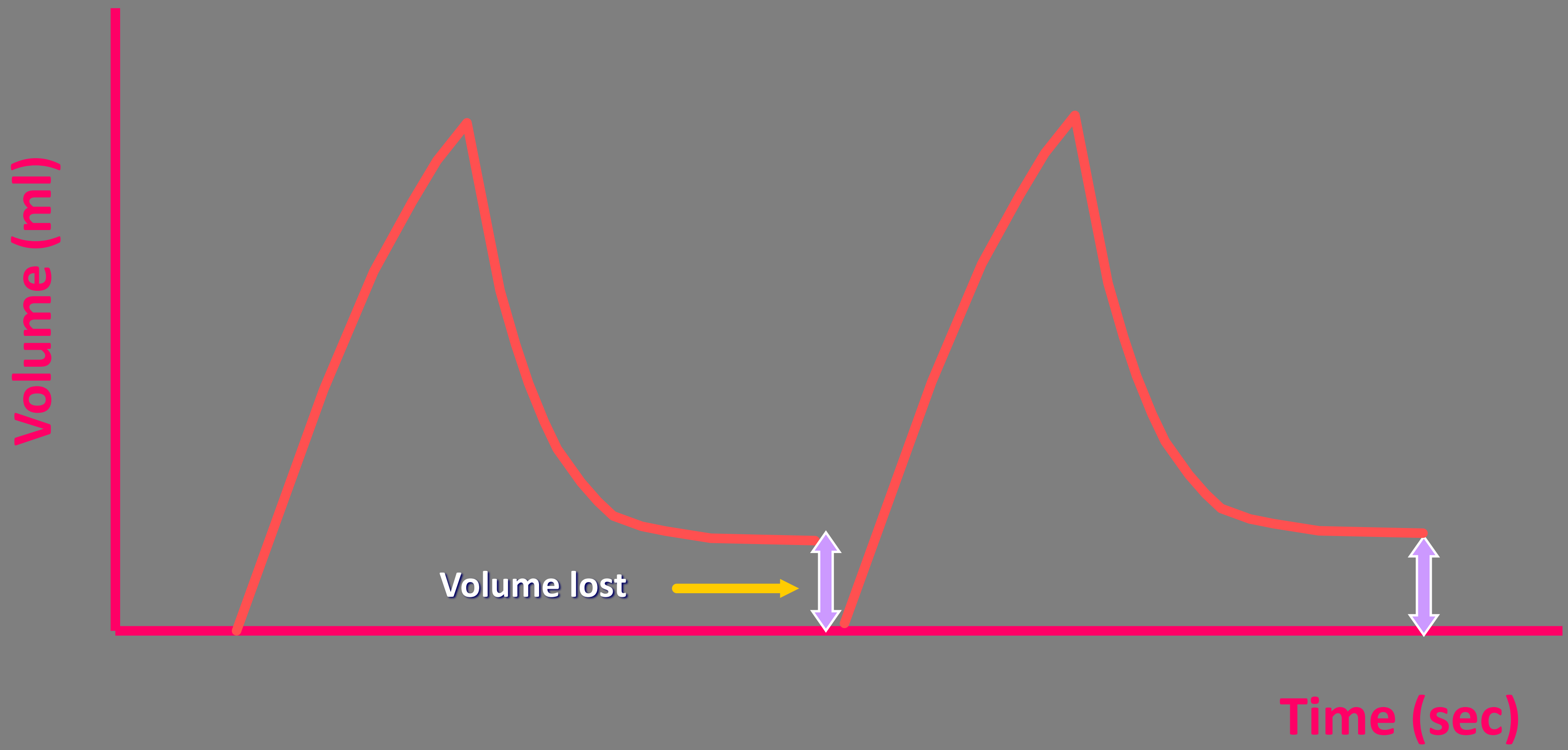
Inadequate Inspiratory Flow



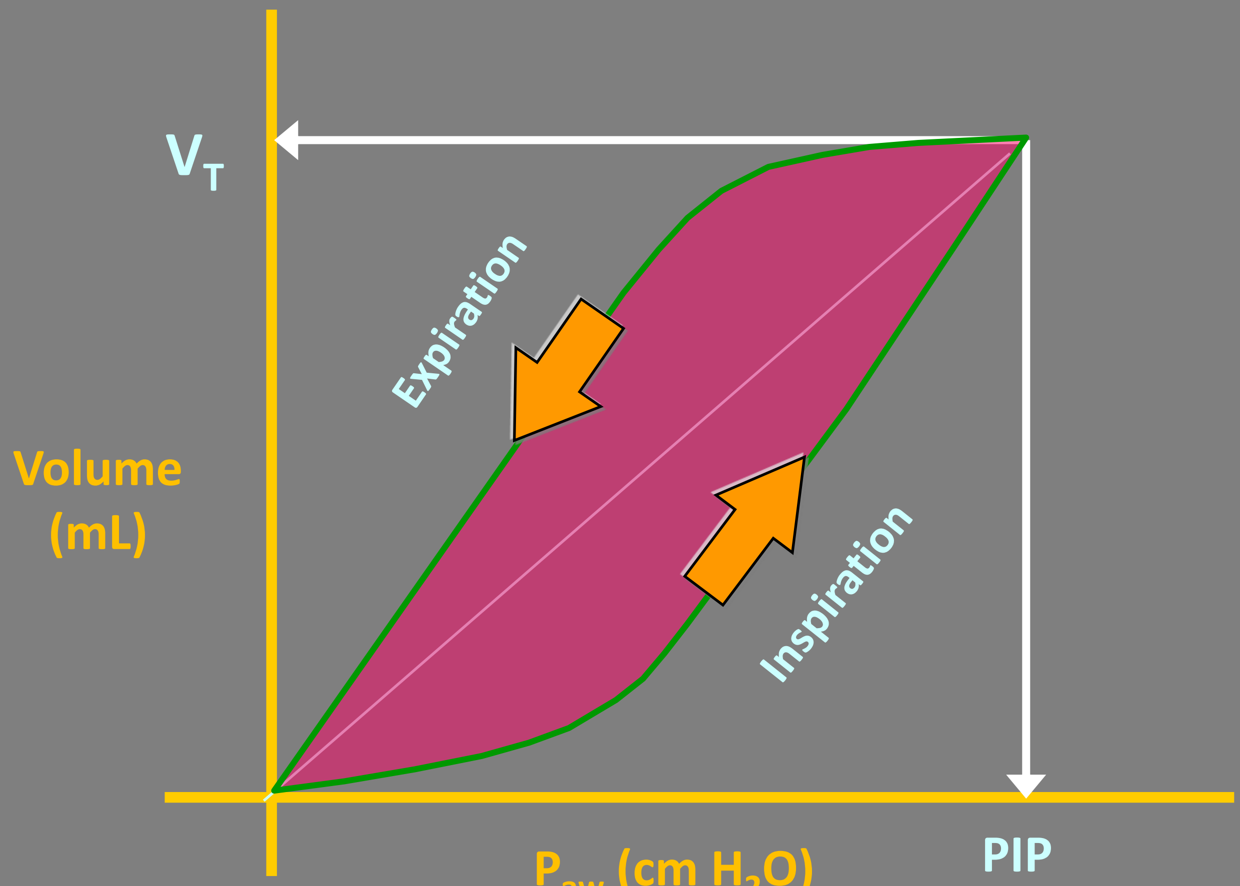
Response to Bronchodilator



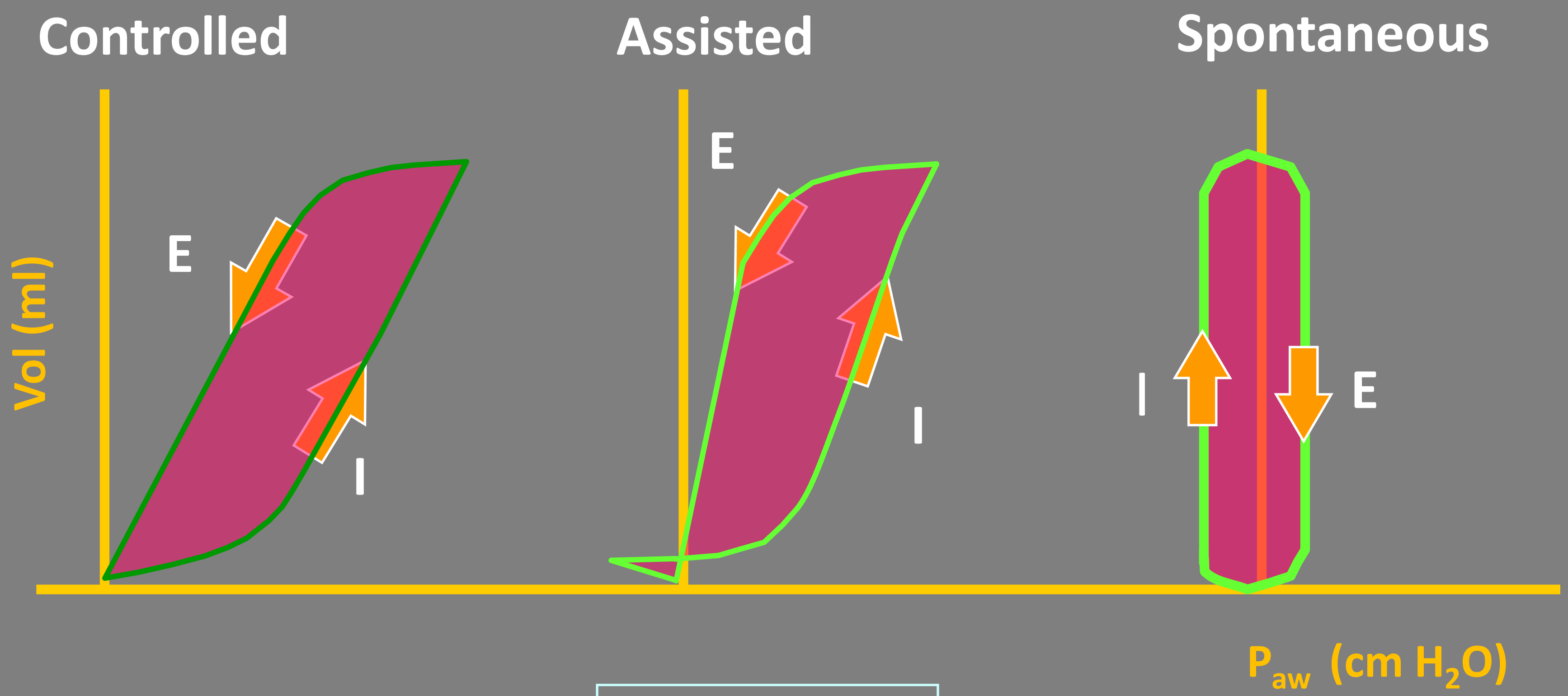
Air Leak/Air Trapping



Components of Pressure-Volume Loop

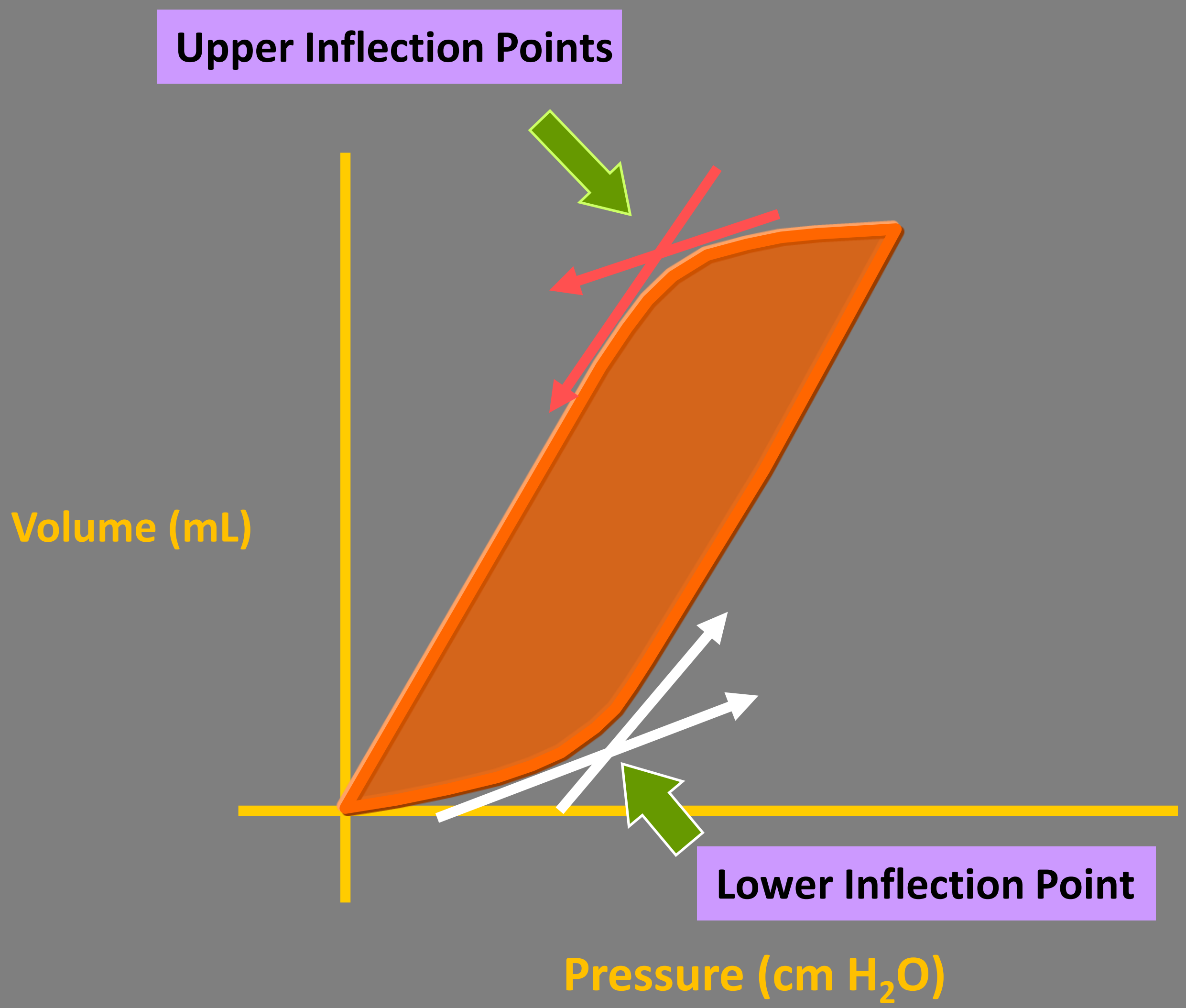


Pressure-Volume Loop
(Type of Breath)

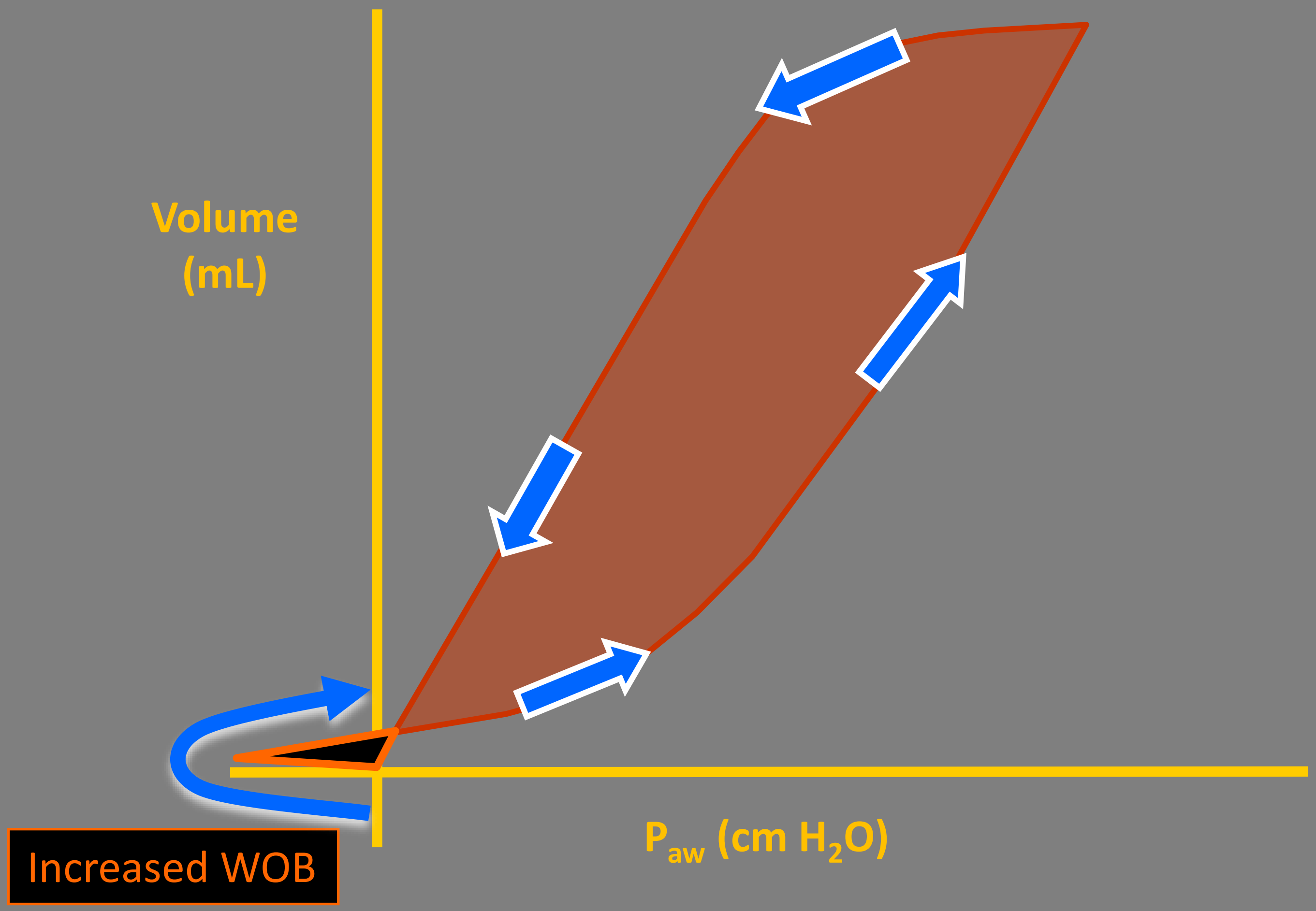


I: Inspiration
E: Expiration

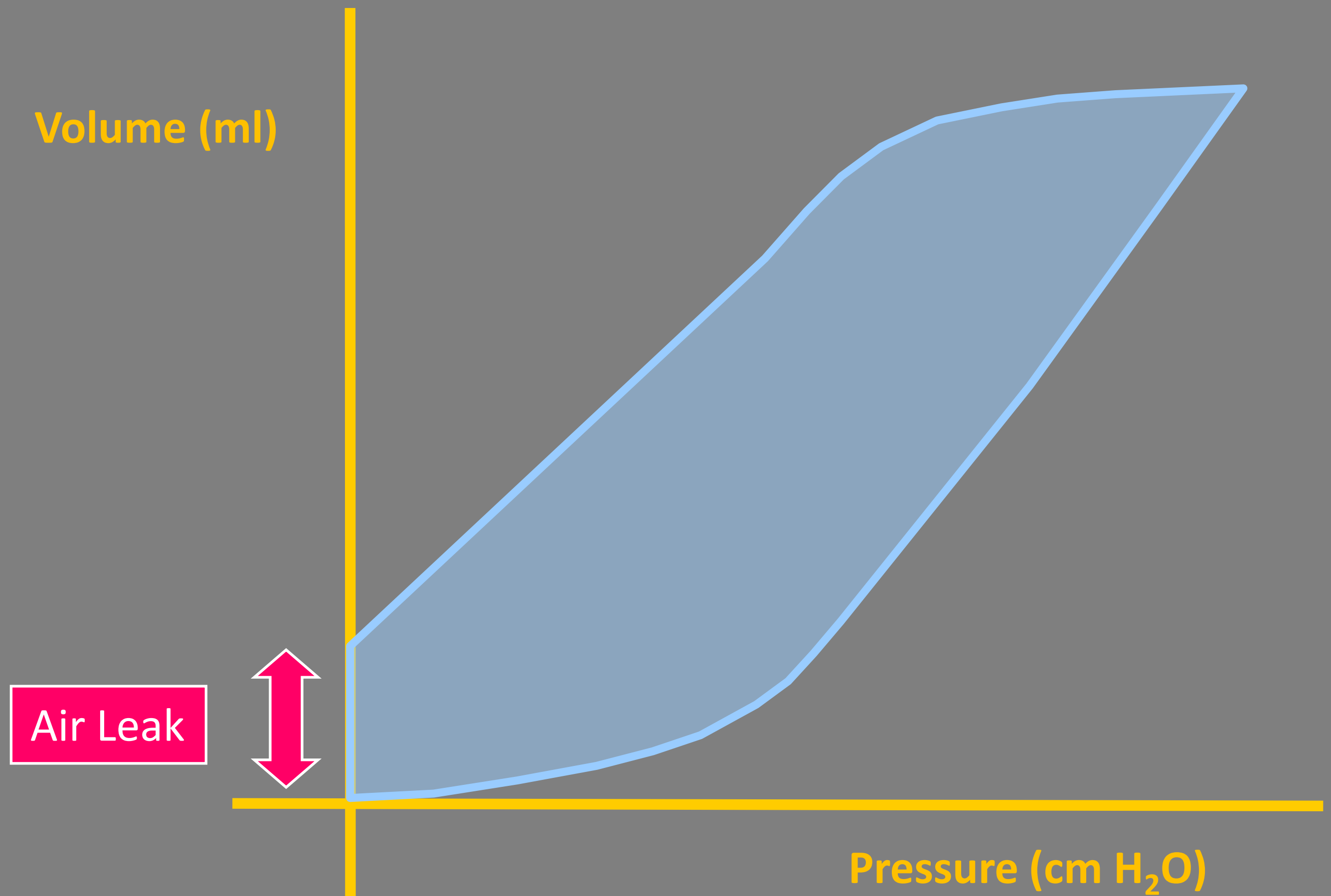
Inflection Points



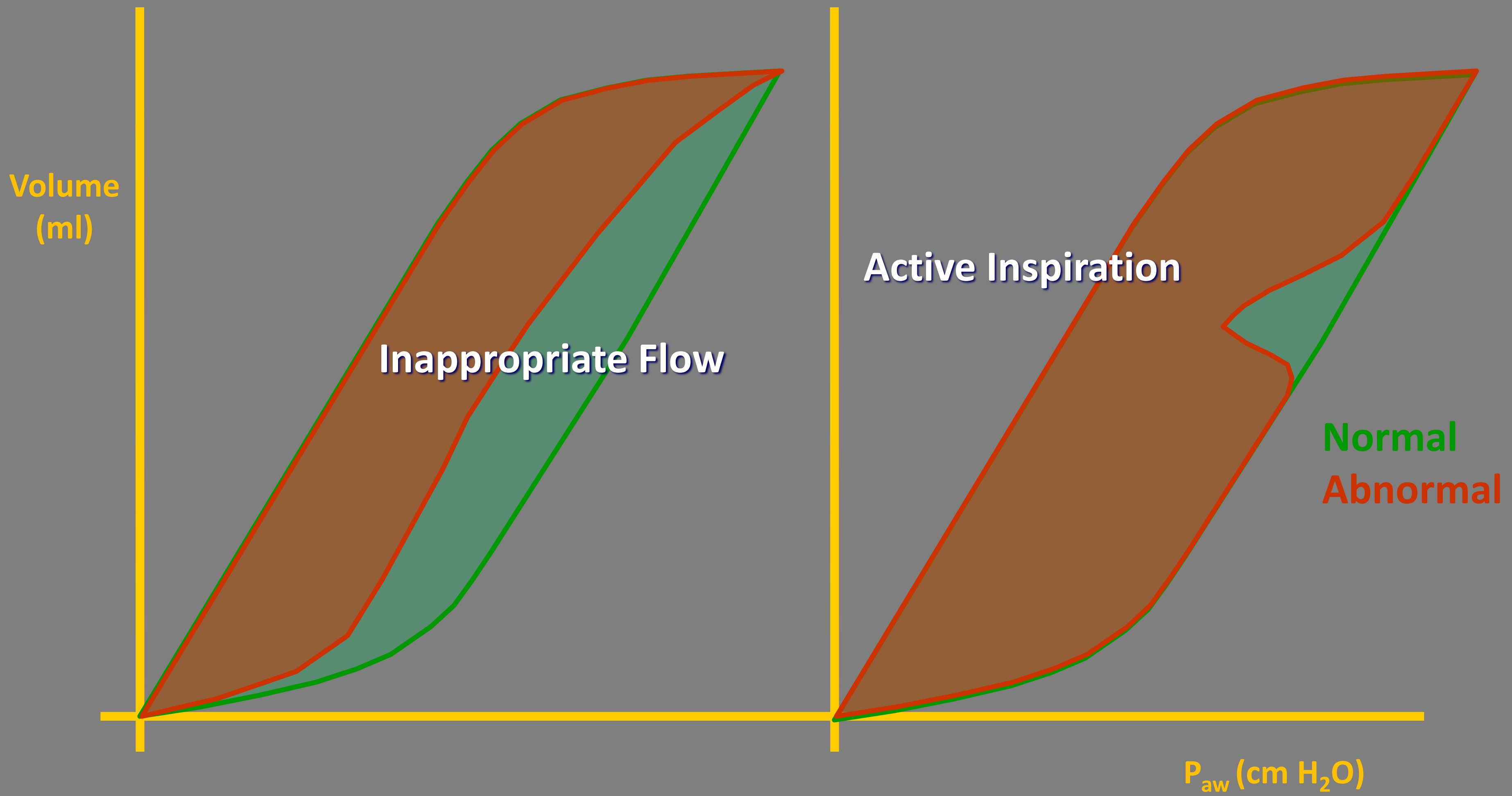
Inadequate Sensitivity



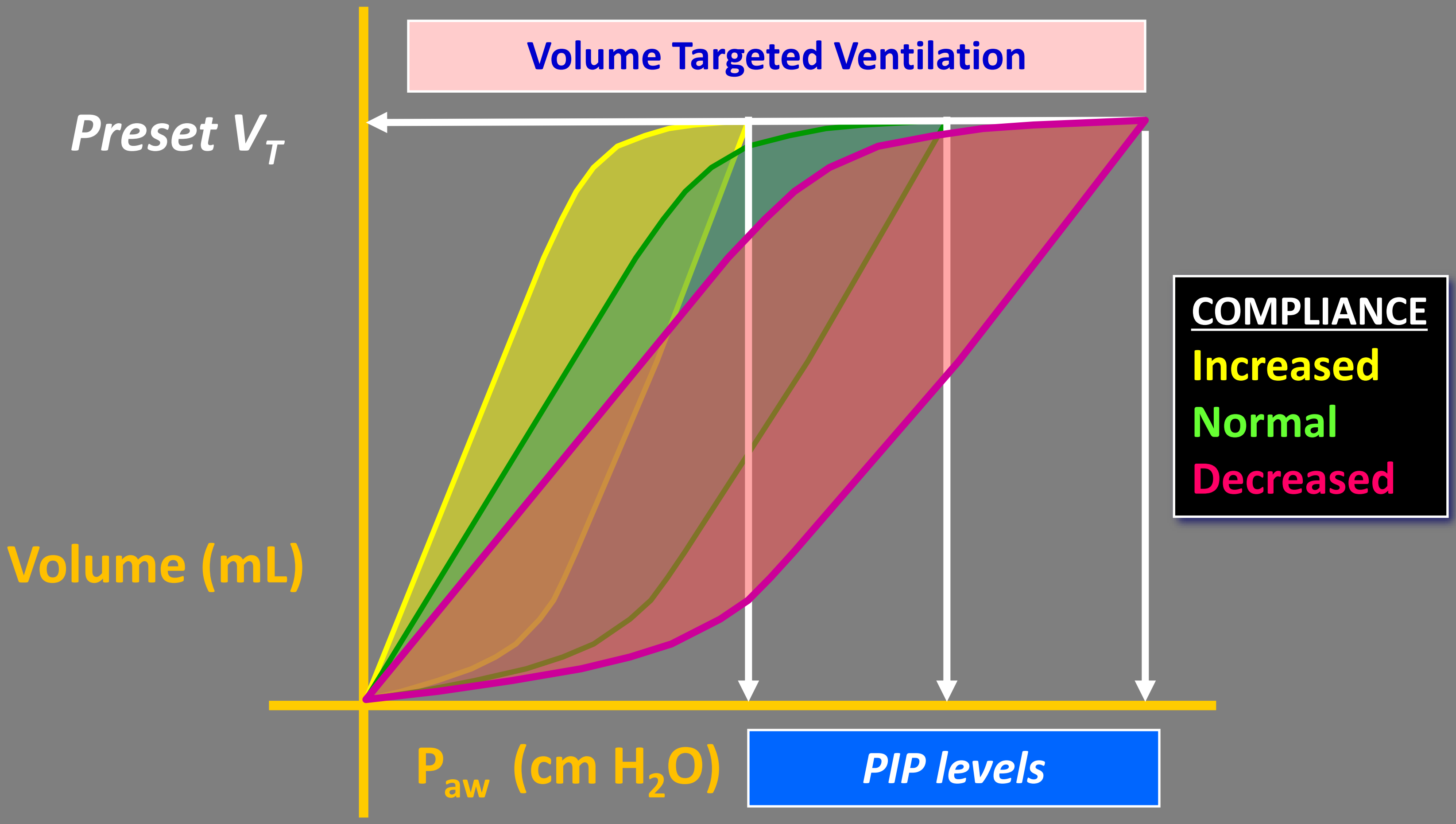
Air Leak/Air Trapping



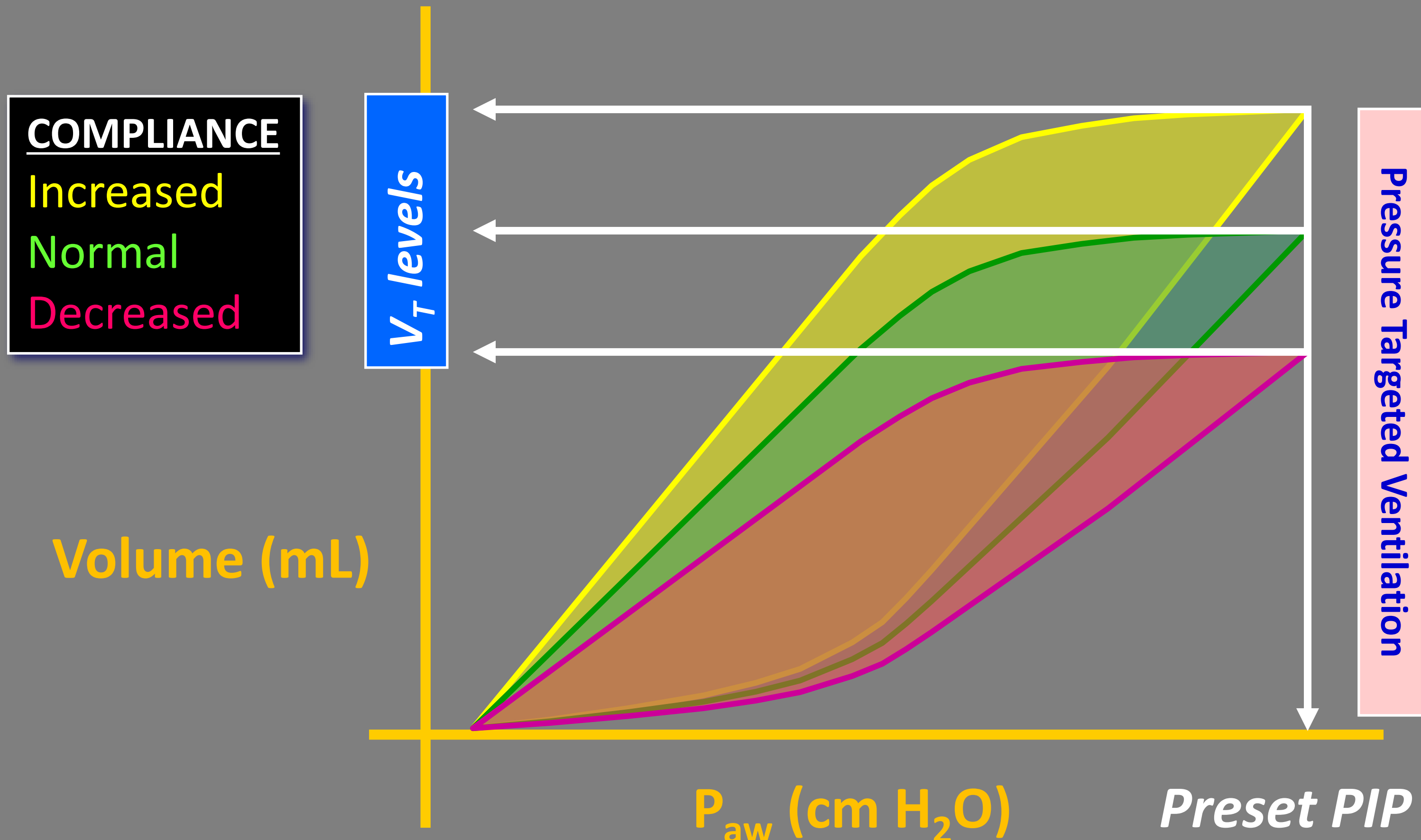
Inadequate Inspiratory Flow



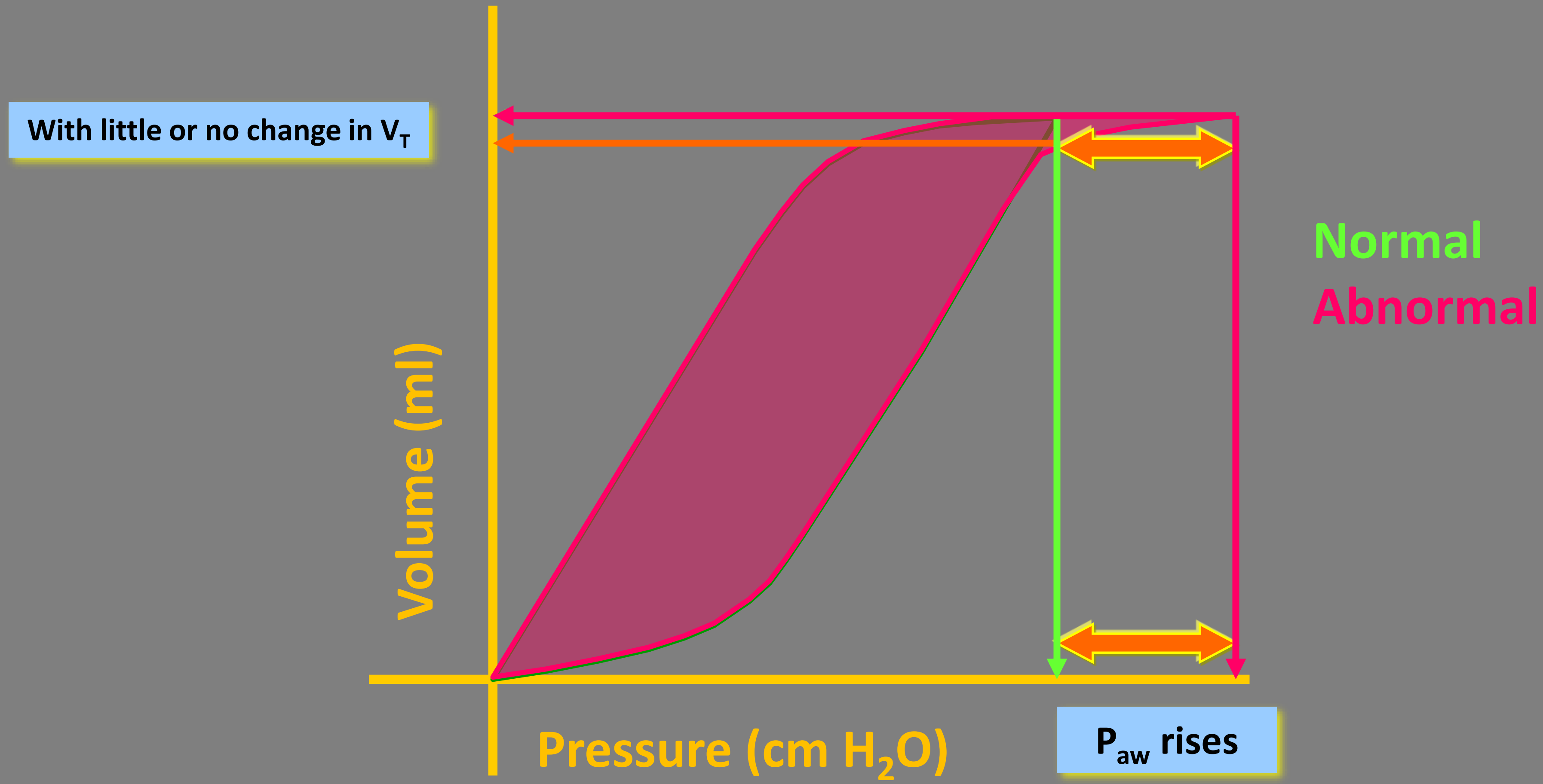
Lung Compliance Changes and the P-V Loop



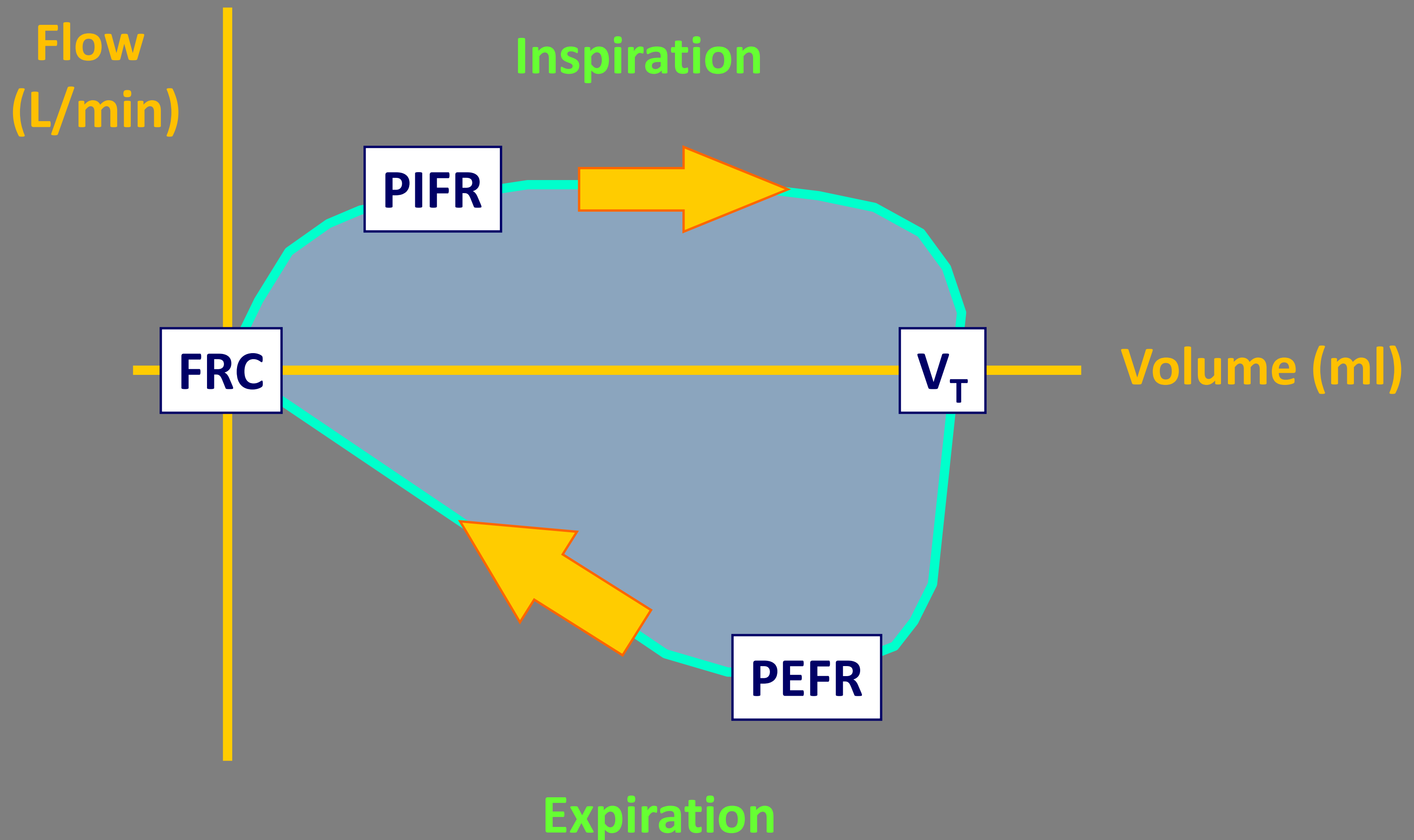
Lung Compliance Changes and the P-V Loop



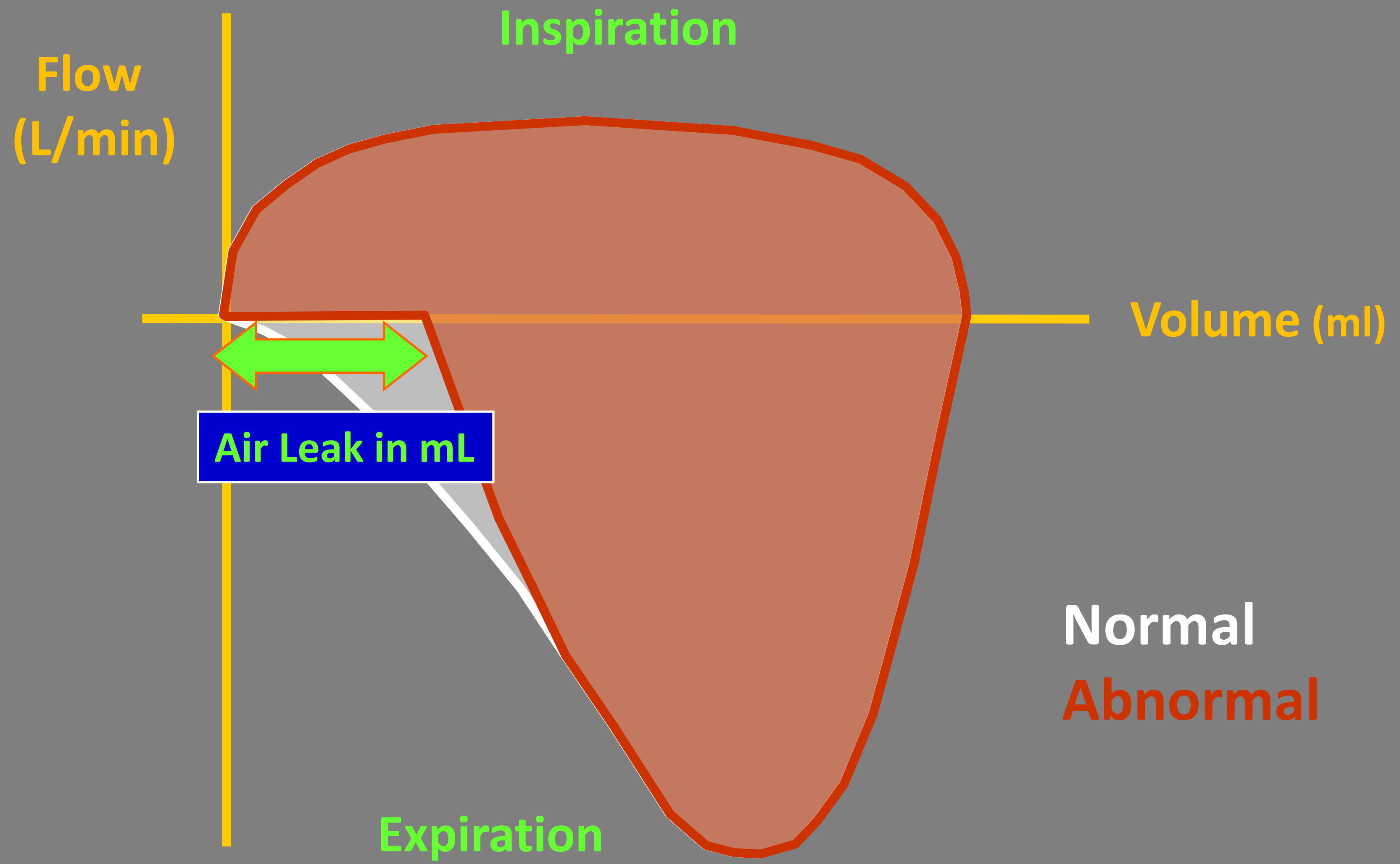
Overdistension



Flow-Volume Loop

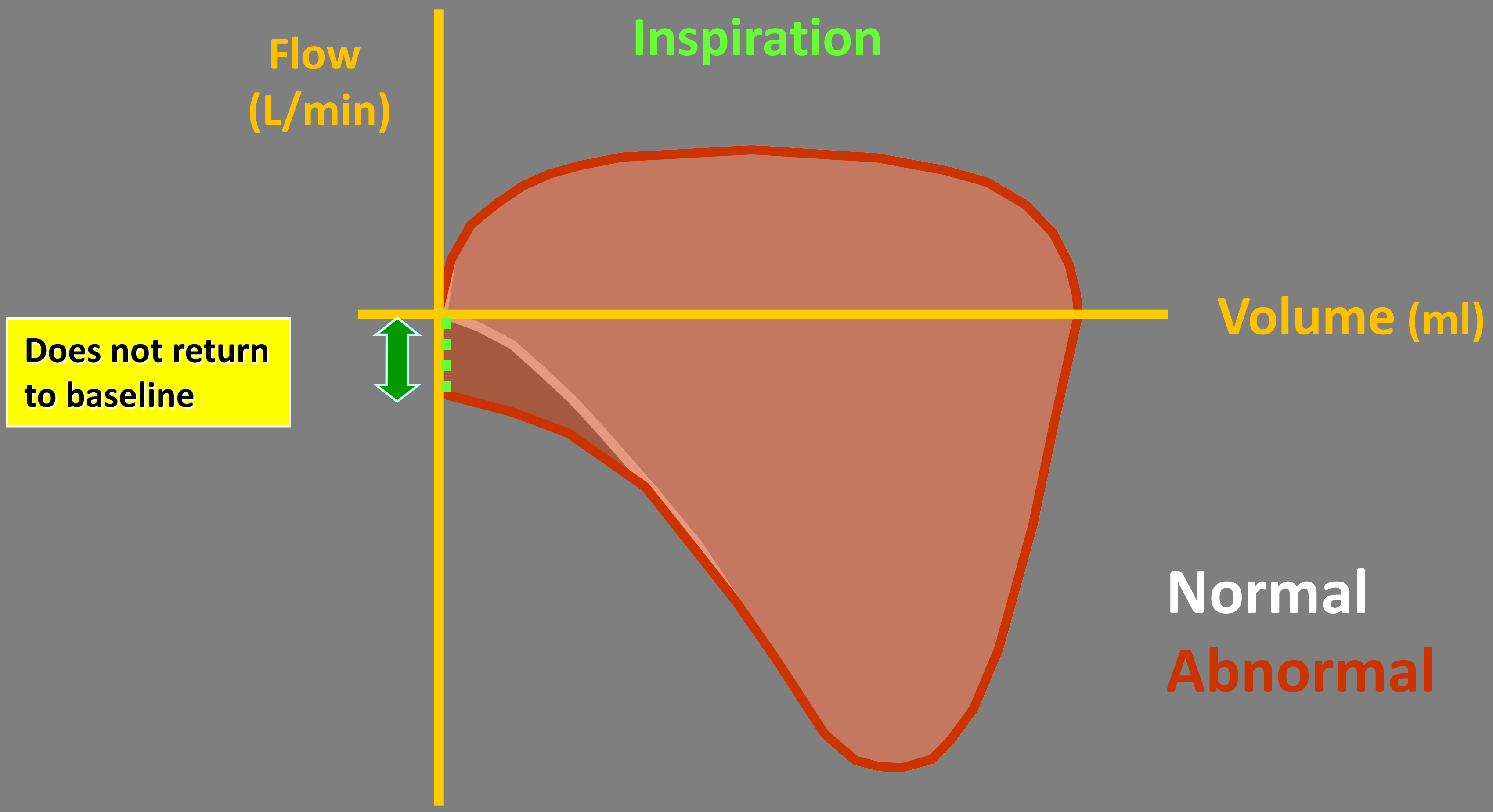


Air Leak

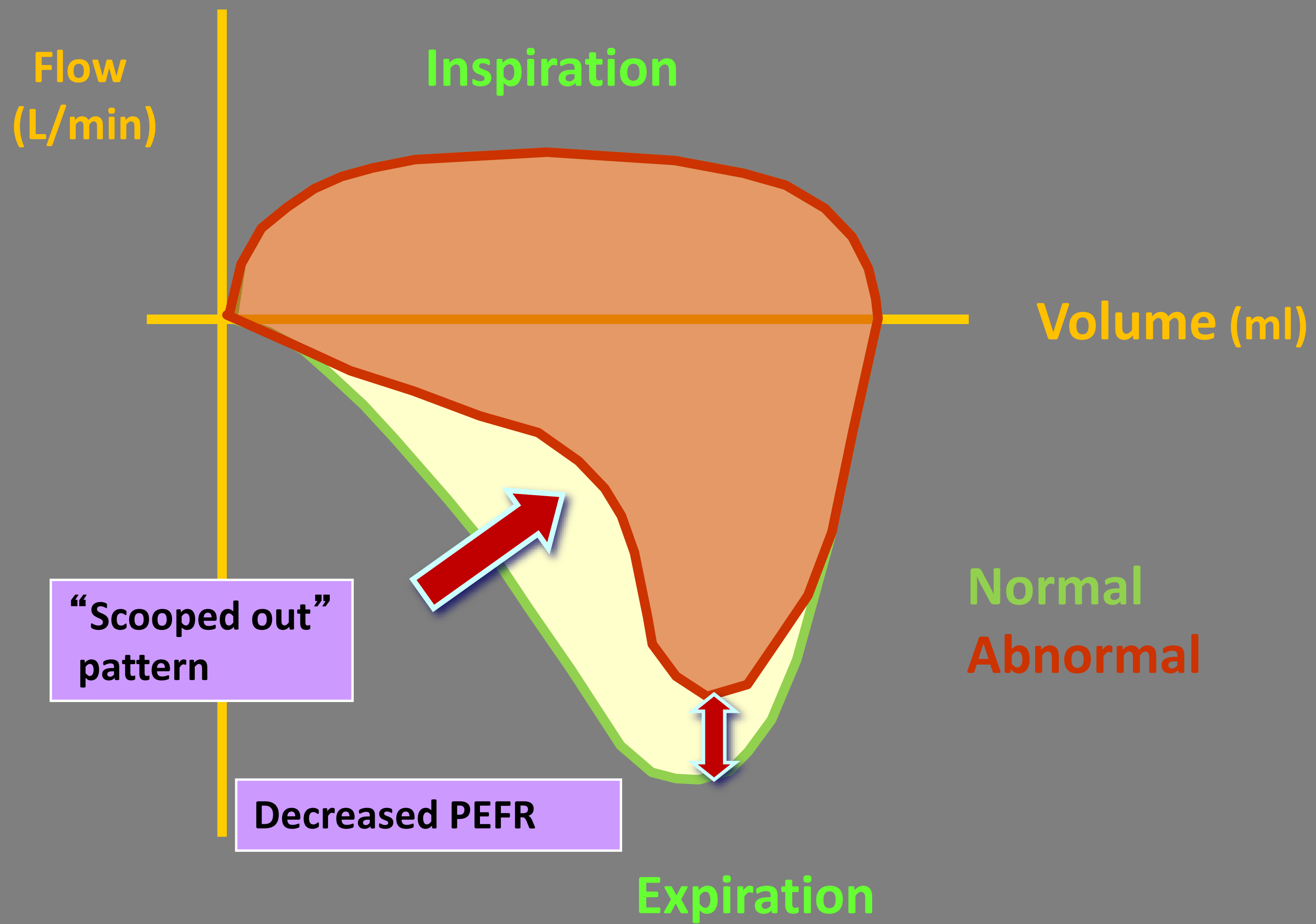


Inspiration
Flow
(L/min)
Volume (ml)
Air Leak in mL
Expiration
Normal
Abnormal

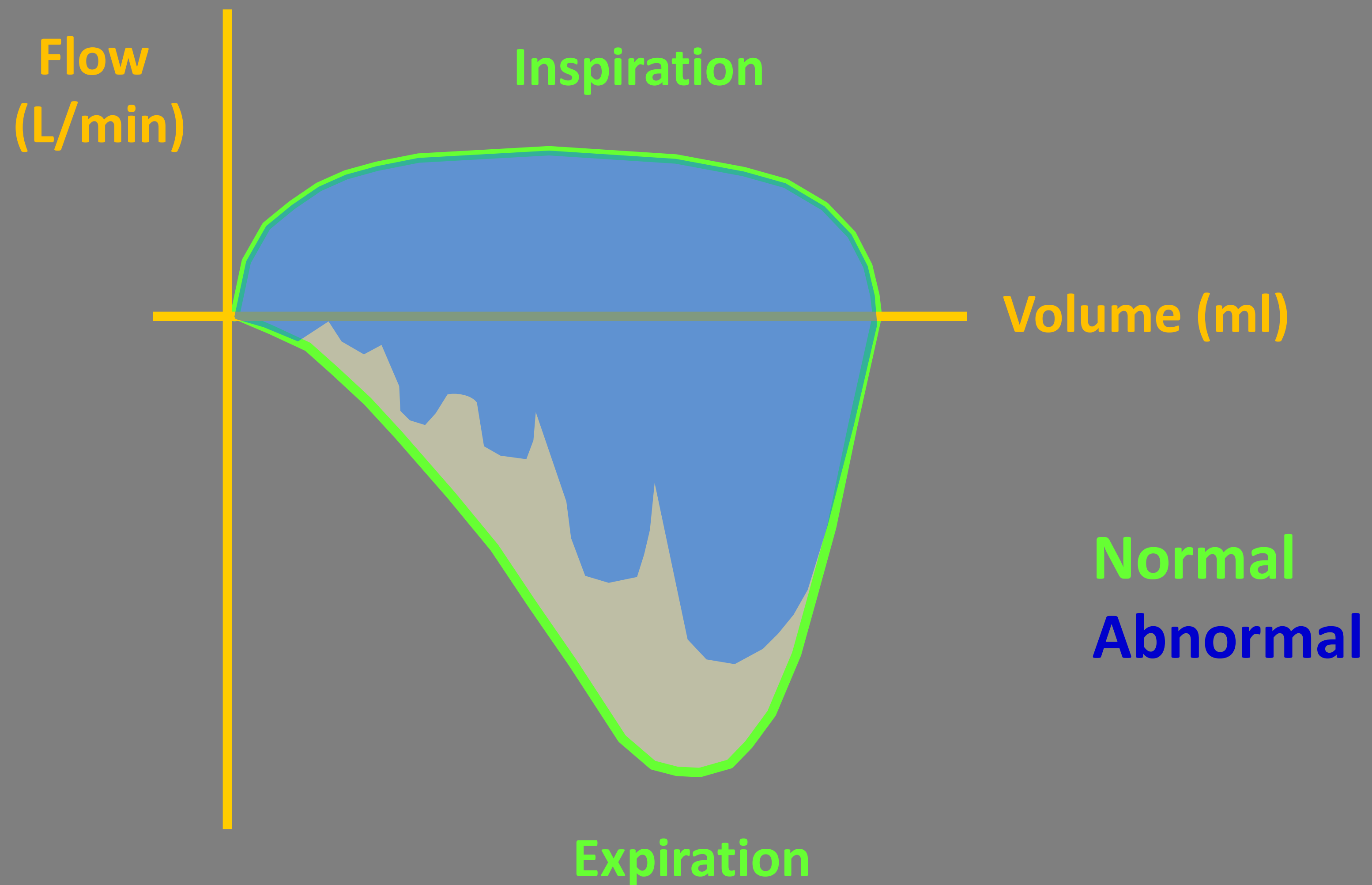
Air Trapping



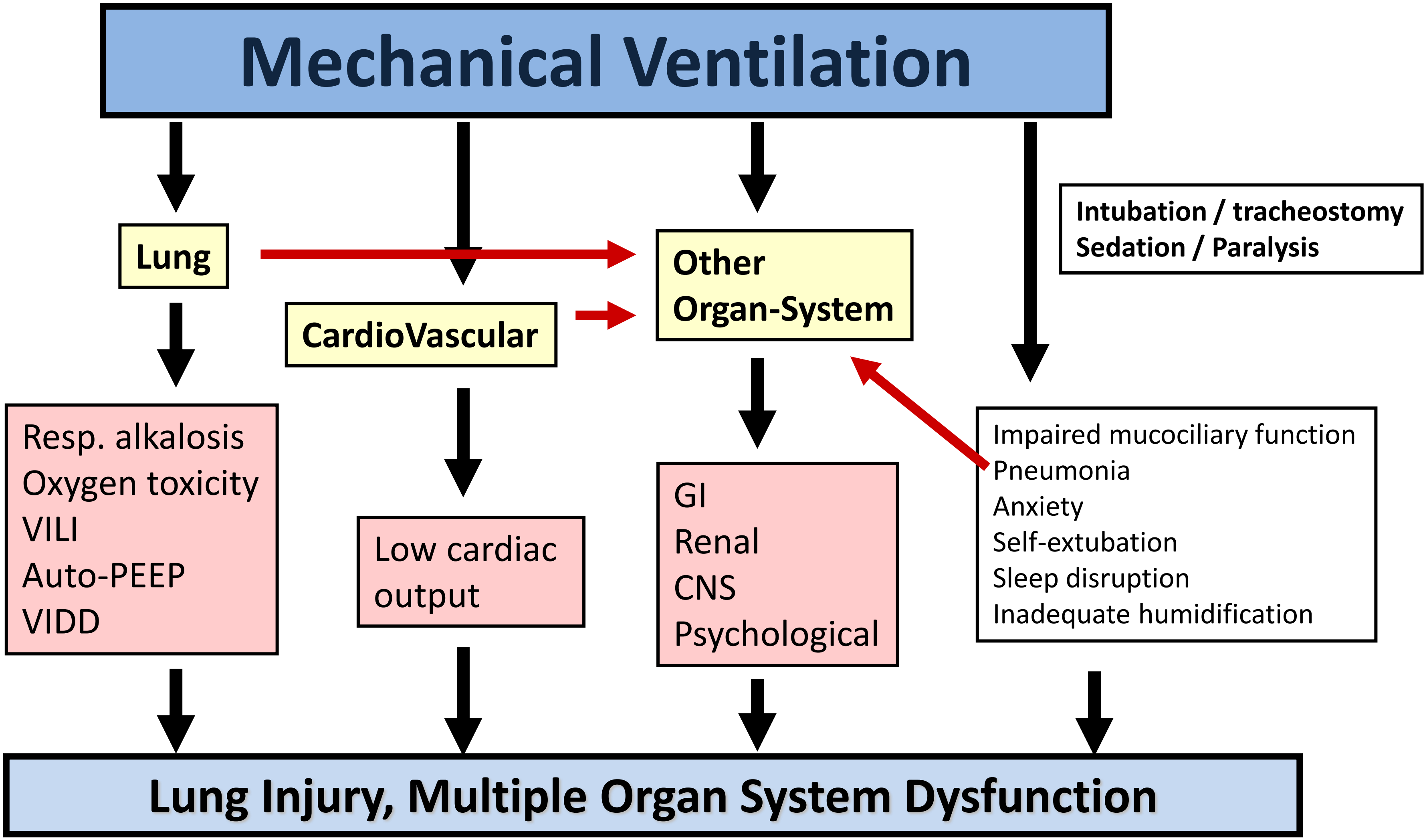
Increased Airway Resistance



Airway Secretions/ Condensate in the Circuit



Summary


Mechanical Ventilation
Lung
CardioVascular
Other
Organ-System
Intubation / tracheostomy
Sedation / Paralysis
Resp. alkalosis
Oxygen toxicity
VILI
Auto-PEEP
VIDD
Low cardiac
output
GI
Renal
CNS
Psychological
Impaired mucociliary function
Pneumonia
Anxiety
Self-extubation
Sleep disruption
Inadequate humidification
Lung Injury, Multiple Organ System Dysfunction