Ventilatory-Induced Lung Injury

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CEEA Tîrgu Mure
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Semantics

- **Ventilatory Induced Lung Injury**
  - *Studied in animal experiments*

researchers were not successful in transferring the measurement of inflammatory mediators during VILI/VALI from bench to bedside

- **Ventilatory Associated Lung Injury**
  - "Bedside" clinical approach

or aggravate ventilator-associated lung injury (VALI) = ventilator-induced lung injury

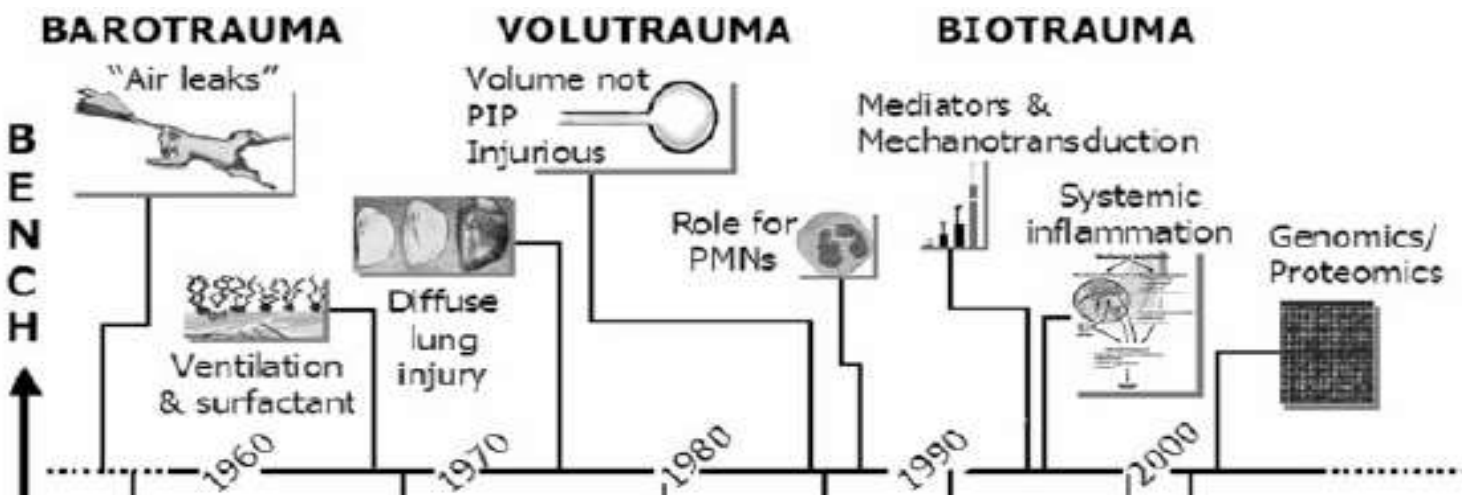
T. Maron-Gutierrez, P. Pelosi and P.R.M. Rocco
Chapter in European Respiratory Monograph · March 2012; DOI: 10.1183/1025448x.10001311

European Society of Anaesthesiology ESA

World Federation of Societies of Anaesthesiologists

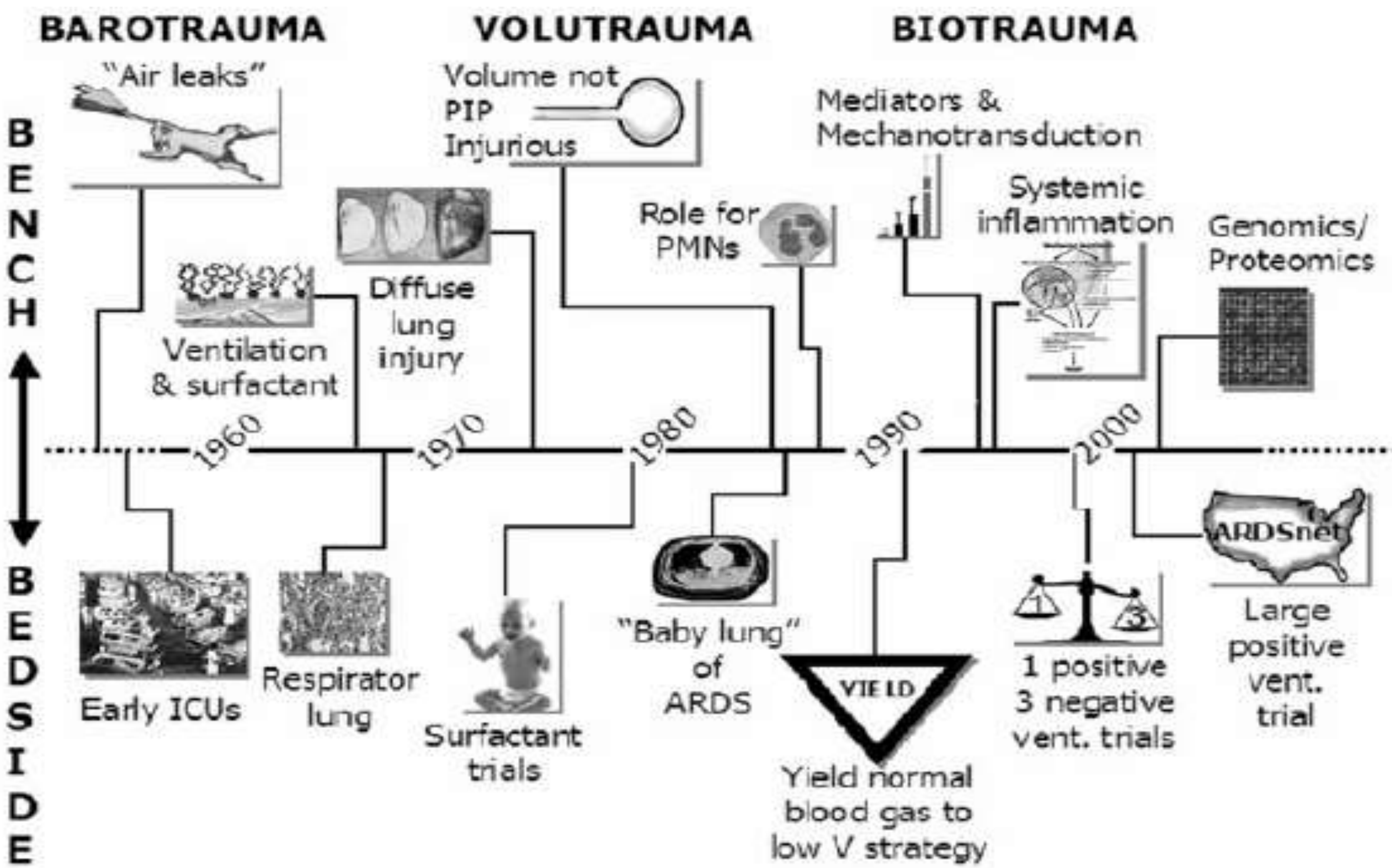
Historical Perspective

Basic Mechanisms and Patophysiology



Historical Perspective

Basic Mechanisms and Patophysiology



Mechanisms of VILI

Concept of ventilatory-induced lung injury (VILI):

- Barotrauma
- Volutrauma
- Atelectotrauma/Stretch injury

1. Physical distruption of cells and tissues

Mechanotrauma

- Biotrauma/Biochemical injury

2. Activation of aberrant cellular pathways

Barotrauma

Seems obvious that inflation of lung will cause damage if air pressures are high enough

High airway pressures during positive pressure ventilation → air leaks

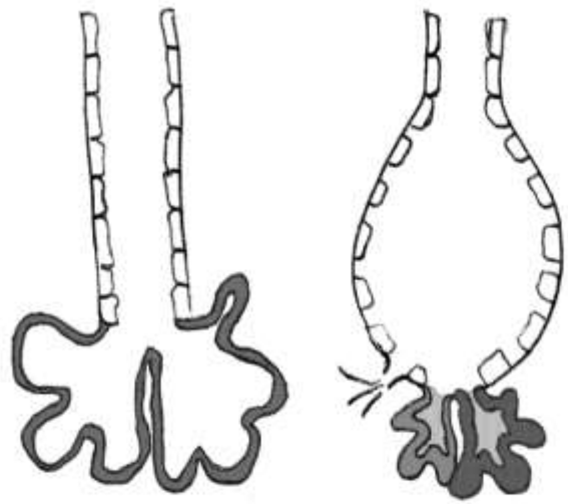
Gross Barotrauma

Most occurs in dependent lung zones (transition zone)

Tearing at Bronchio-Alveolar Junction as lung is recruited and allowed to collapse

Air leaking into interstitial space (PIE)

Air leaking into pleural space

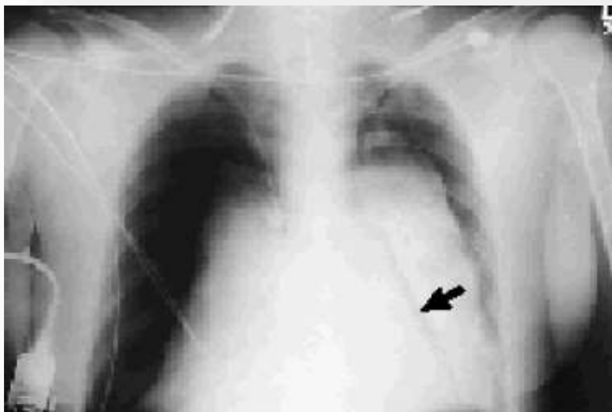


The Manifestations of Gross Barotrauma

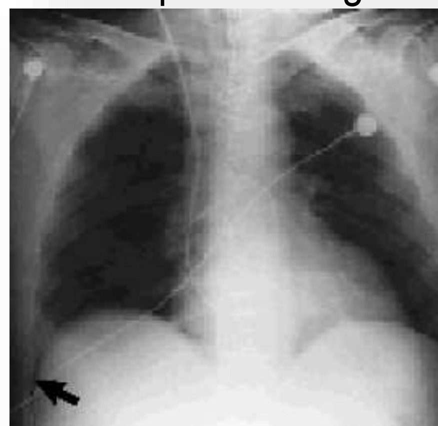
PNEUMOTHORAX	PNEUMOMEDIASTINUM	PNEUMOPERITONEUM	SUBCUNANEOUS EMPHYSEMA
Dyspnea, chest pain	Dyspnea, chest pain	Abdominal pain	Swelling, upper chest, neck, and the face
Tachycardia, tachypnea	Tachycardia, tachypnea	Abdominal distension, tympany	Crepitus
Upright CXR shows air between visceral and parietal pleura in the upper part of the thoracic cavity Supine CXR shows deep costophrenic angle (deep sulcus sign)	Hypotension if tension pneumomediastinum (rare)	Abdominal compartment syndrome with tension pneumoperitoneum (rare)	Radioluncent streaks
CT confirmatory	Mediastinal crunch or “Hamman’s sign”	Upright CXR or left lateral decubitus AXR (5-10min in the position before exposure): Gas shadows seen in the right upper quadrant Rigler’s sign: gas seen on both sides of the bowel wall Football sign: gas seen outlinging the peritoneal cavity inverted-v sign: medial umbilical folds seen outlined by Falciform ligament seen outlined by the air	

Gross Barotrauma

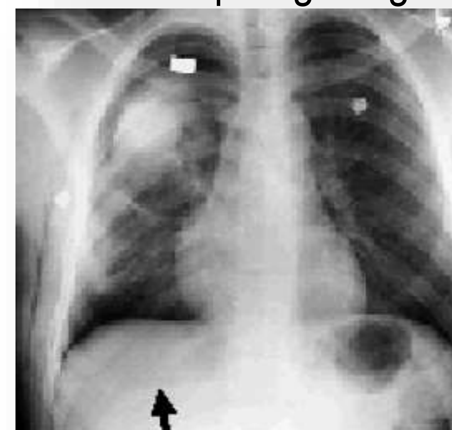
Pneumothoraces & Pneumomediastinum



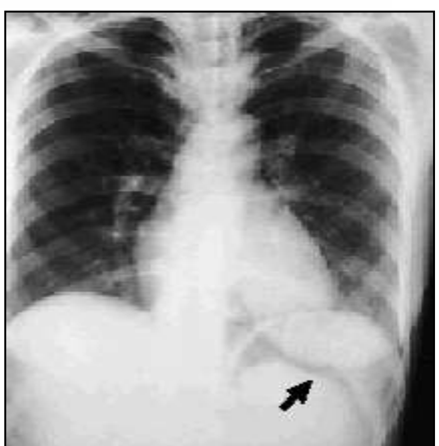
Deep sulcus sign

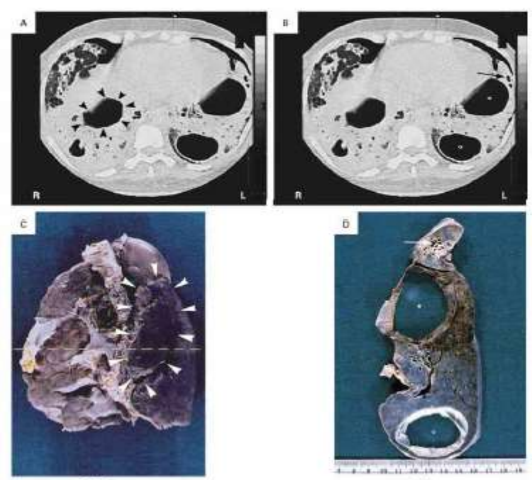


Double diaphragm-sign



Pneumoretroperitoneum





Paramediastinal pneumatocele

Pseudocysts

Possible Actions Directed at Lowering the Plateau Pressure

Reduction in inspiratory pressure

PEEP-lowering measures PEEP

Reduction in the tidal volume

Consideration to increasing sedation (possibly the administration of n-m agents)

Specific treatment of the various conditions:

Pneumothorax

- Closed chest drainage

Pneumomediastinum

- Supportive measures are adequate in most cases since the condition is generally self resolving

Tension pneumomediastinum

- In the rare tension pneumomediastinum, mediastinotomy should be performed and a drain left is situ post-operatively

Pneumoperitoneum

- This is also self limited complication. Supportive treatment frequently suffices

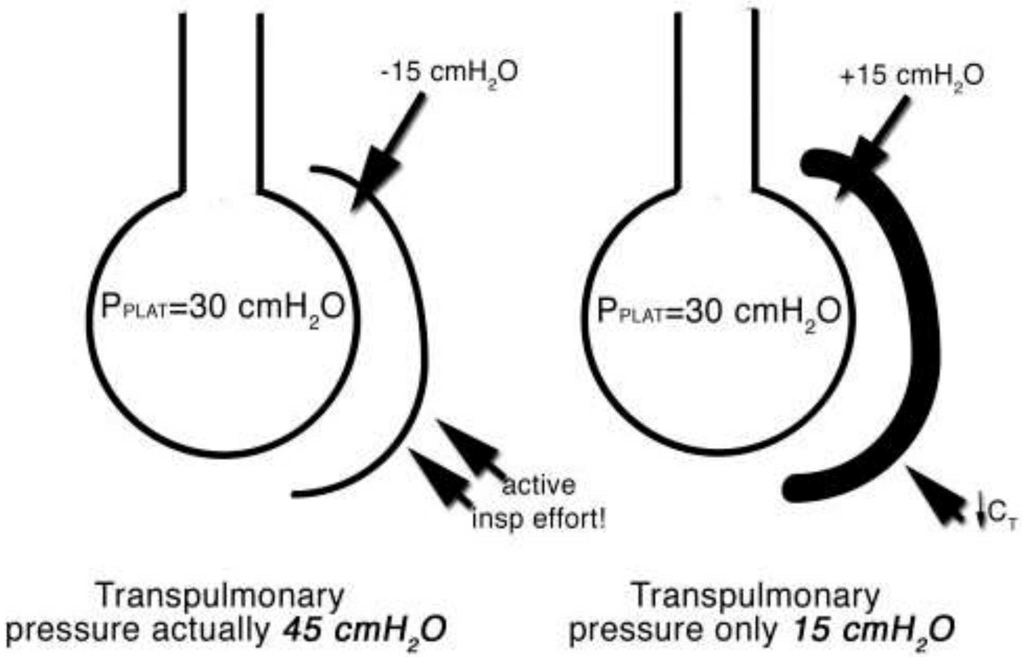
Tension pneumoperotoneum

- In this extremely rare condition, surgical drainage af the air must be carried out to relieve the compartment syndrome

Subcutaneous emphysema

- Again, this condition is self-limited. Although is theoty, a compartment syndrome due to subcutaneous emphysema is possible, it has not yet been reported

Physiological Distruption of Cells and Tissues



High airway pressure alone has little injurious effect on the lung, unless the lung is allowed to expand unchecked

If the lung is allowed to over-distend, it may be damaged by high airway pressure

conceptual distinction between "volutrauma" and "barotrauma."

Dreyfuss D, Soler P, Basset G, Saumon G. Am Rev Respir Dis 1988; 137: 1159-64; J-Da Ricard, D Dreyfuss and G Saumon.Ventilatory-induced lung injury. Eur Rspir J 2003; 22: Suppl 42,2s-9s

Nicholas de Post, Jean-Damien Ricard, Georges Saumon and Didier Dreyfuss. Ventilatory-induced lung injury: historical perspectives and clinical implications; Annals of Intensive Care; 2011,1/28

Volutrauma

Overdistension

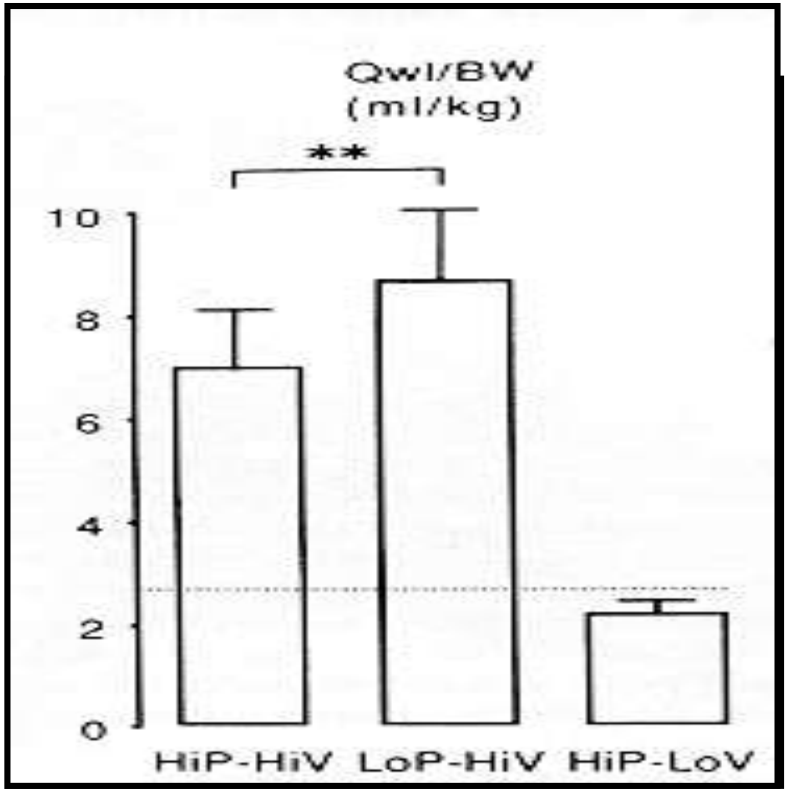
– damage to the lung caused by a mechanical ventilator set for an excessively high tidal volume

Volutrauma

Rodents ventilated with three modes:

1. High Pressure (45 cmH2O), High Volume
2. Low Pressure (negative pressure ventilator), High Volume
3. High Pressure (45 cmH2O), Low Volume

(strapped chest and abdomen)



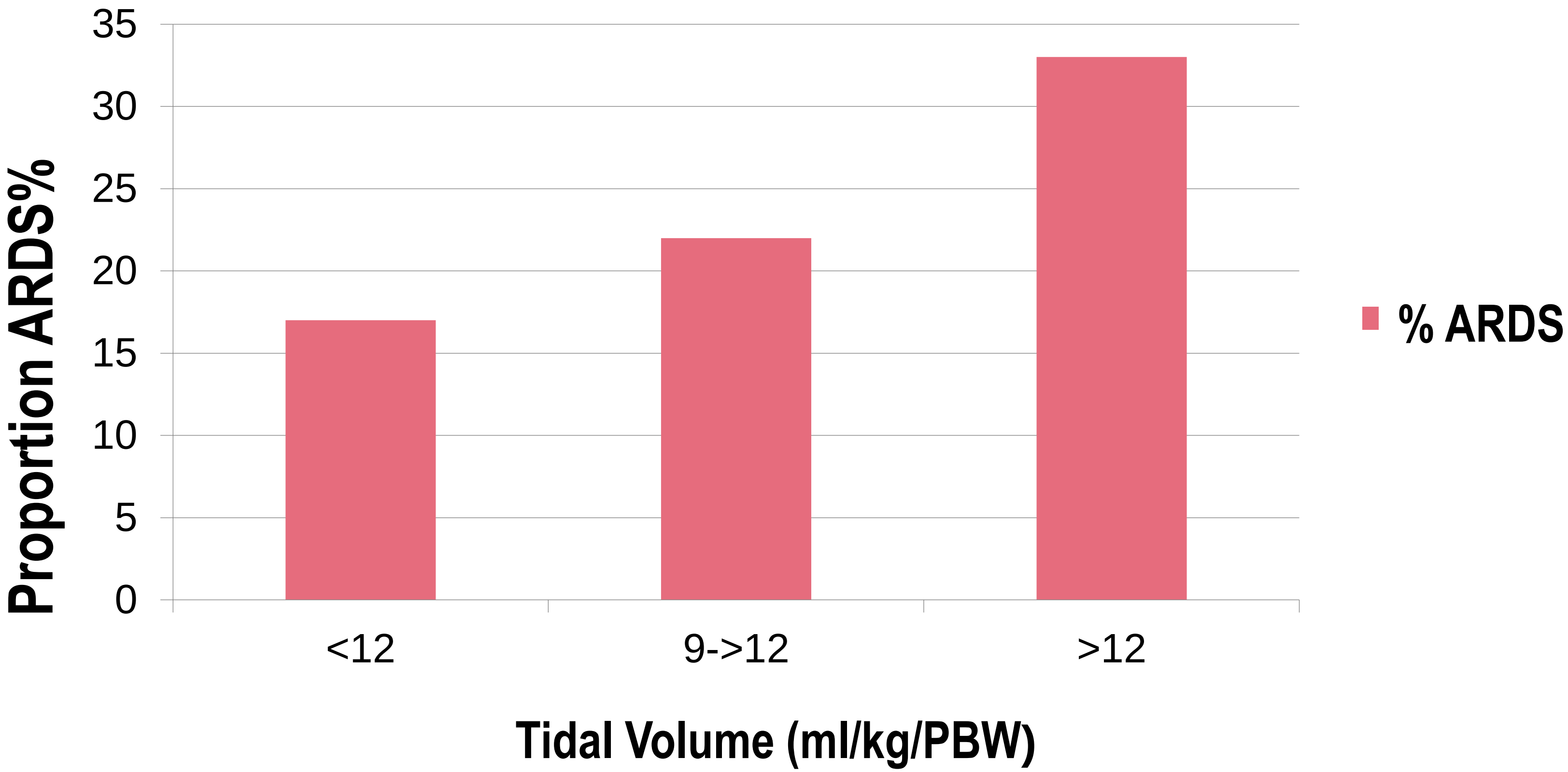
Dreyfuss,D ARRD 1988;137:1159

J-Da Ricard, D Dreyfuss and G Saumon.Ventilatory-induced lung injury. Eur Rspir J 2003; 22: Suppl 42,2s-9s

Nicholas de Post,,Didier Dreyfuss. Ventilatory-induced lung injury: historical perspectives and clinical implications; *Annals of Intensive Care*; 2011,1/28

Proportional of ARDS According to Tidal Volume

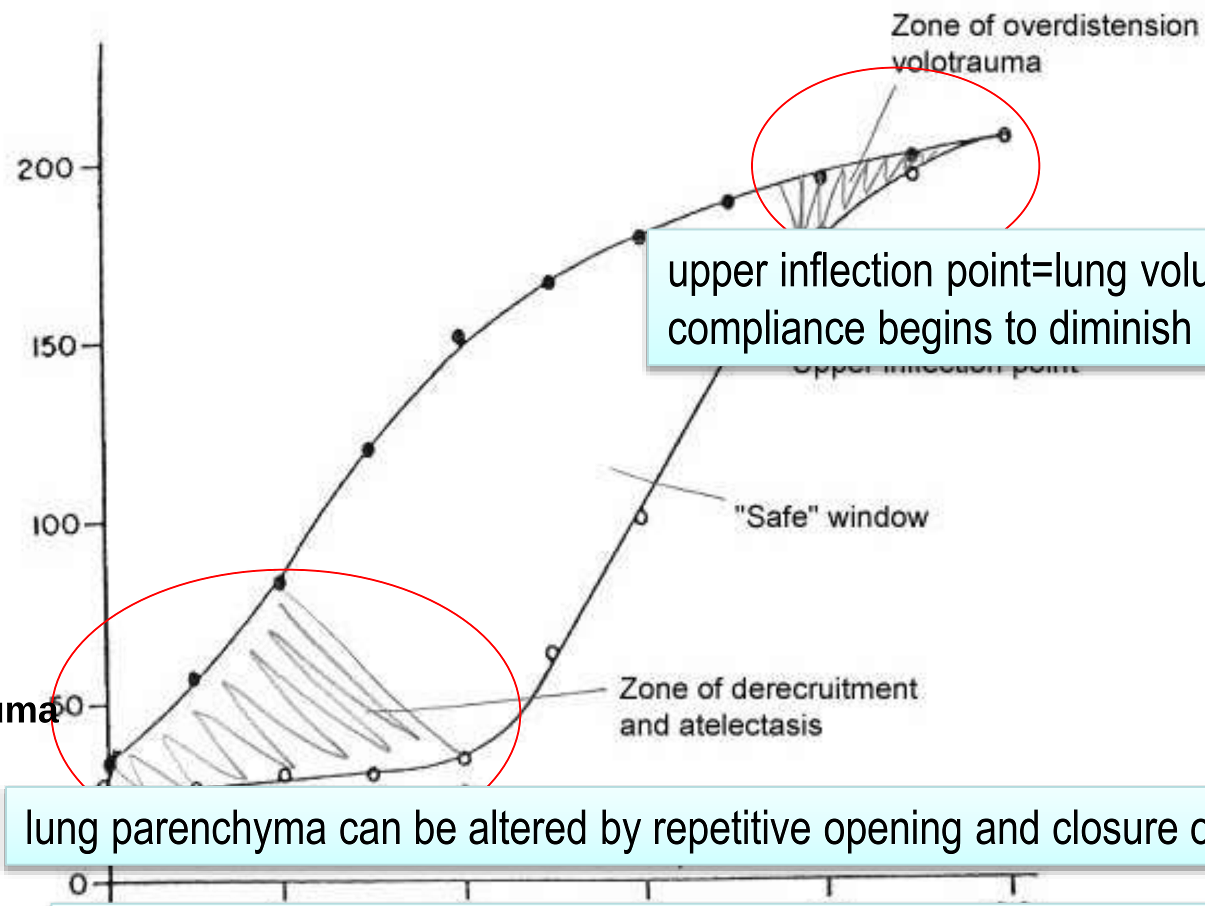
PDW: predicted body weight



The Irish Critical Care Trials Group (2008) Acute respiratory distress in Ireland: a prospective audit of epidemiology and management. Critical Care 12:R30

Atelectrauma/Overdistension

Interest of the pressure-volume curve



upper inflection point=lung volume at wich compliance begins to diminish

Atelectrauma (RACE)

lung parenchyma can be altered by repetitive opening and closure of distal airway units

Lower inflection point (LIP) - minimal pressure required to recruit collapsed alveoli

Atelectrauma

Insufficient PEEP - repeated alveolar collapse and expansion (shear forces)

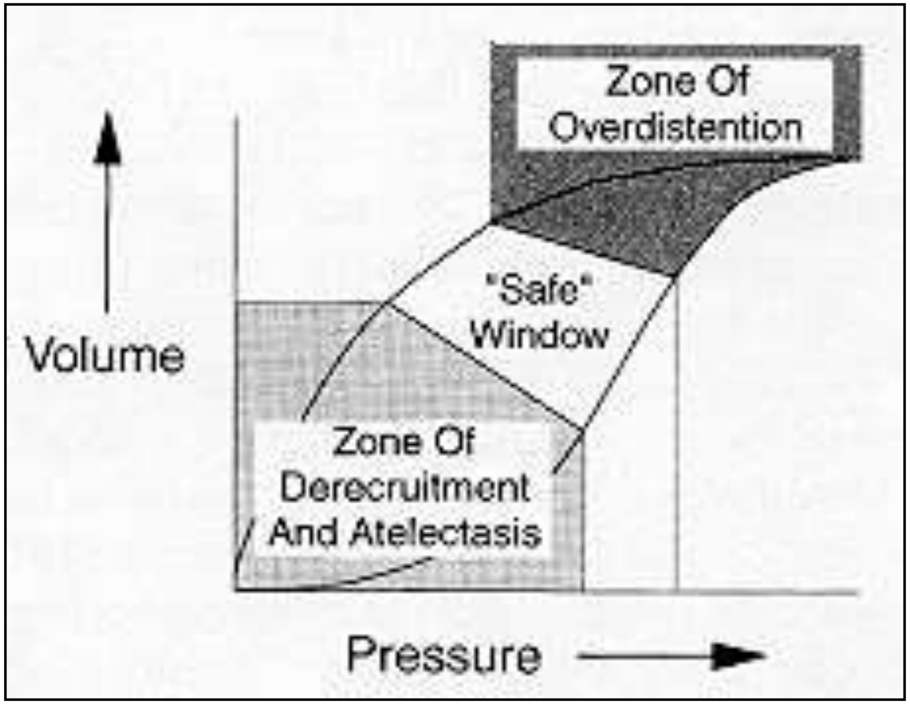
Safe Window

Balance Between Overdistension And Recruitment

There are two injury zones during mechanical ventilation

- Low Lung Volume Ventilation tears adhesive surfaces
- High Lung Volume Ventilation over-distends, resulting in “Volutrauma”

The difficulty is finding the “Sweet Spot”



Froese AB, Crit Care Med 1997; 25:906

Volutrauma - Atelectrauma

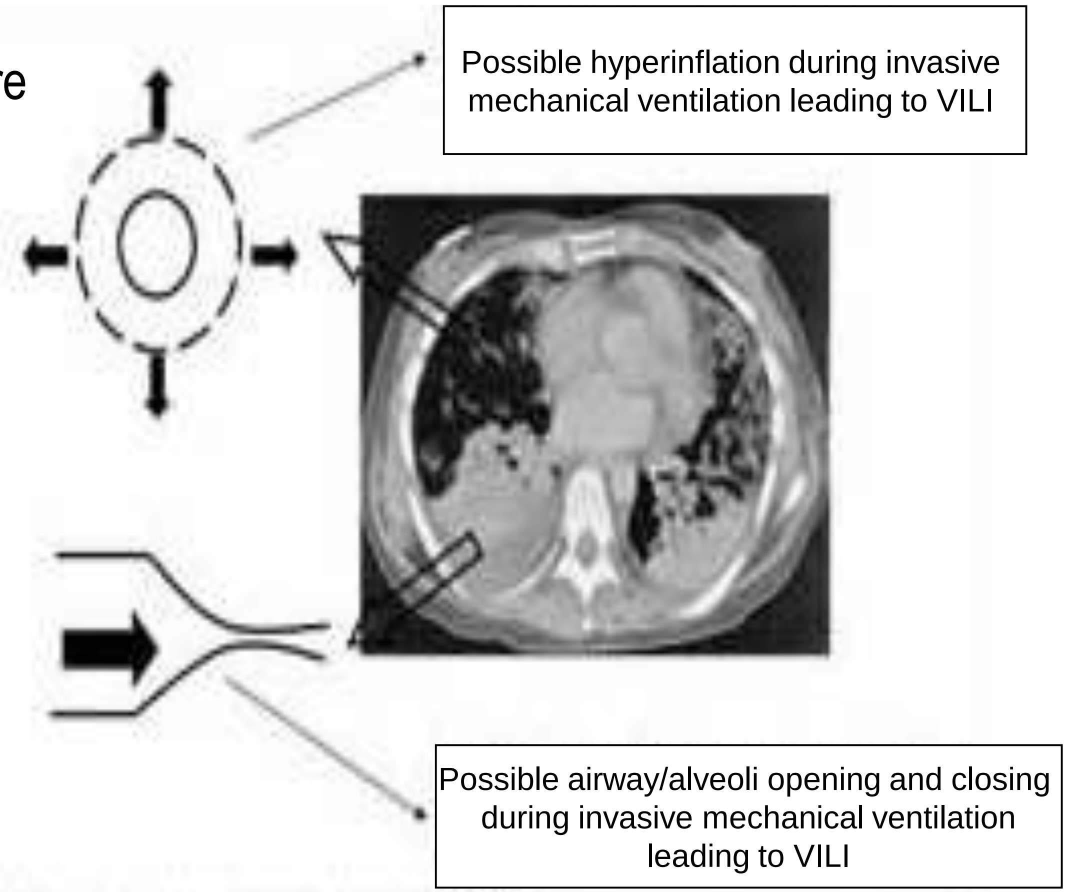
Caused by cycling of the lung (change in surface area), independent of pressure required

Alters Surfactant function

- Promotes Atelectasis

Increases capillary leak of proteinacious material

- Promotes Atelectasis



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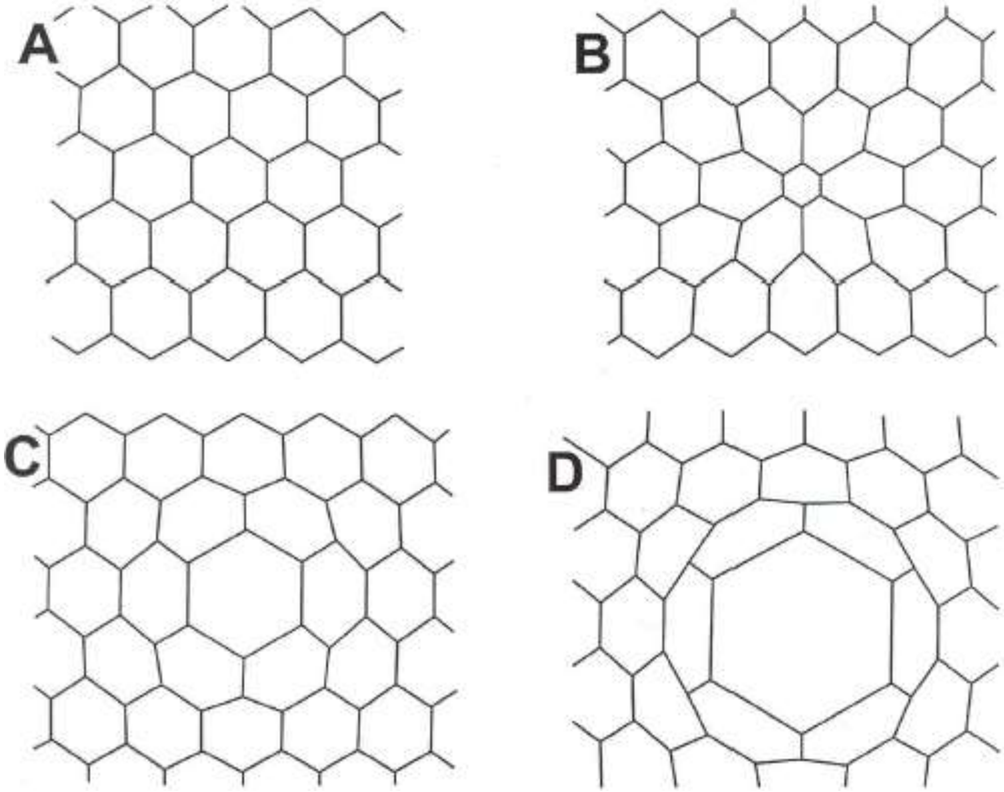
Alveolar Interdependence



Stress

Strain

Stretch

Shear

Mead J et al. J Appl Physiol 1970; 28:596:608

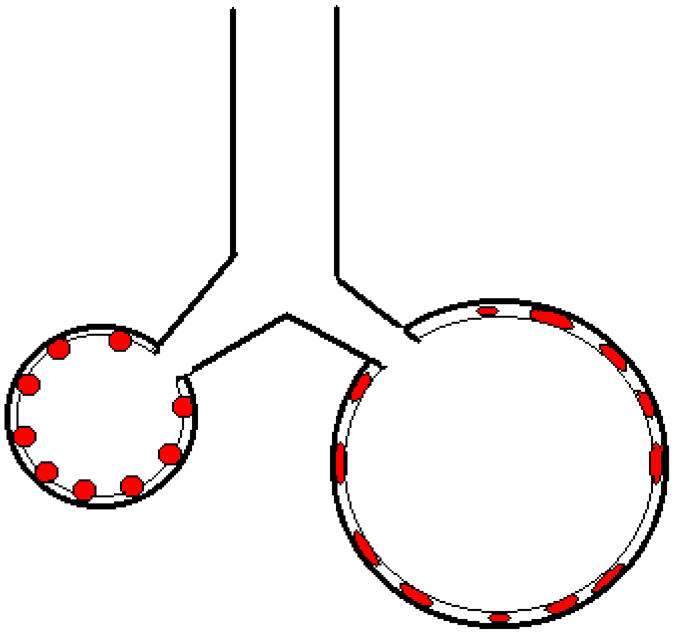
Stretch Injury

Alters capillary transmural pressures

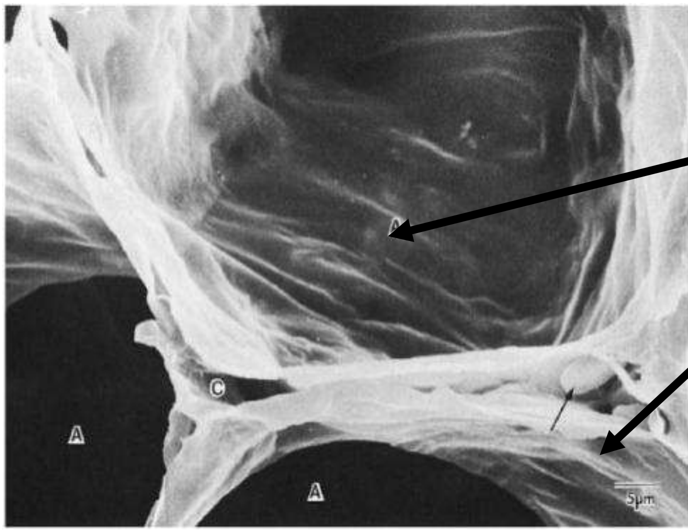
Changes in transmural pressure causes breaks in capillary endo and epithelium

Increases leak of proteinacious material

- Promotes Atelectasis

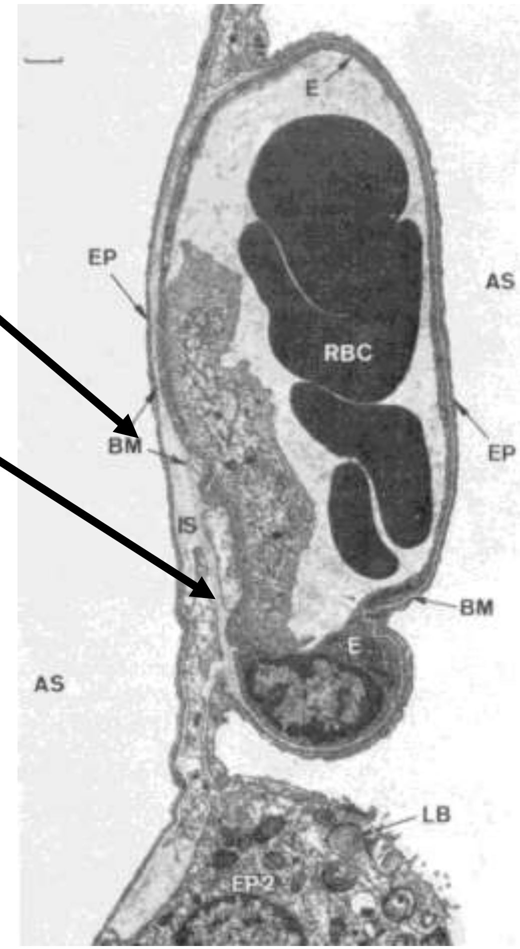


Stretch Injury

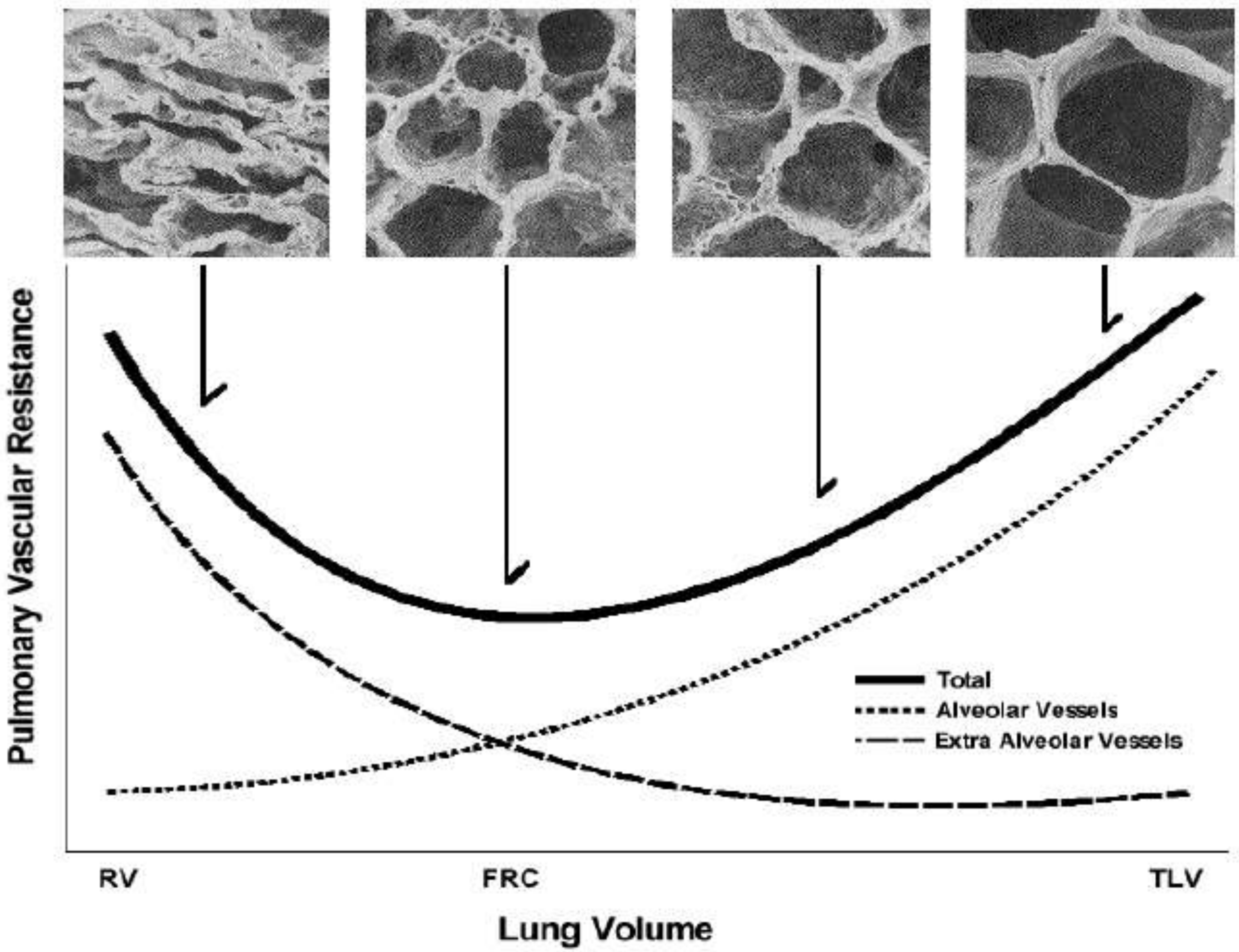


Alveolar space

A-C membrane

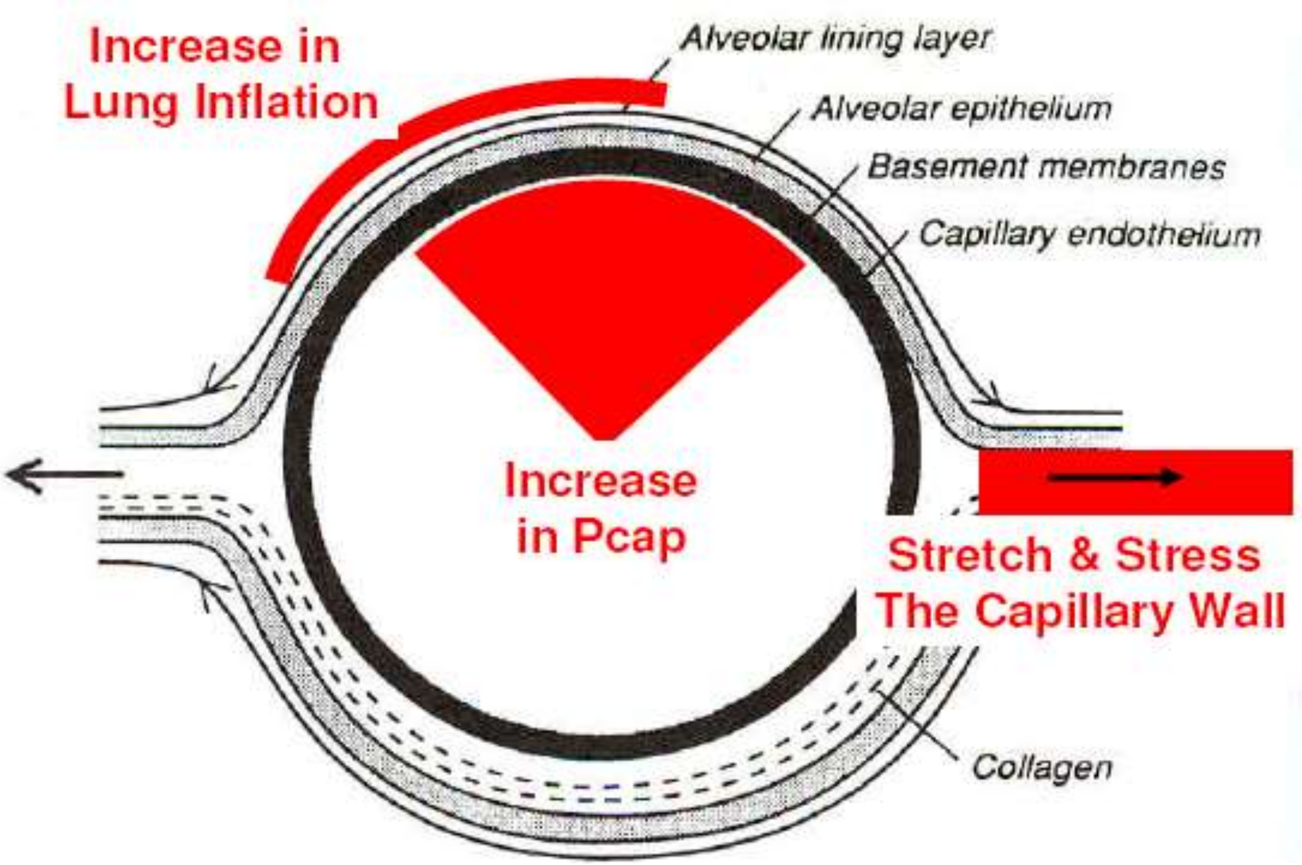


Pulmonary Capillary - Alveolar Wall



Capillary Stress Failure

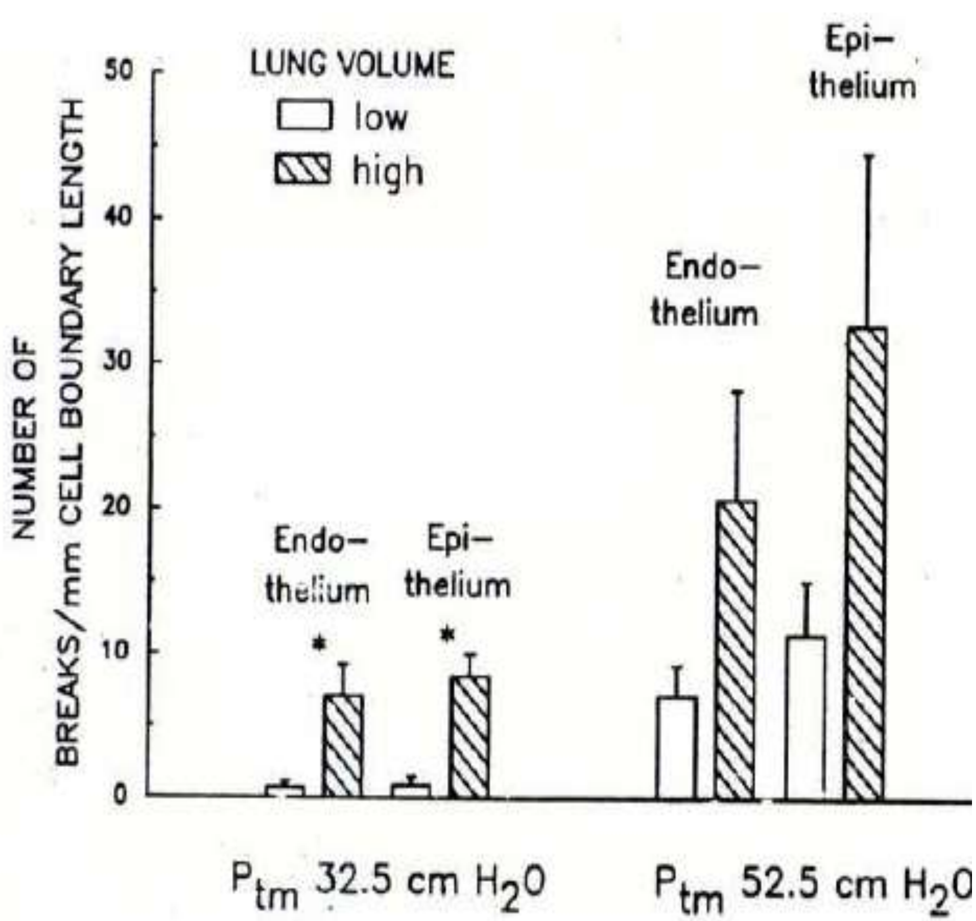
The Three Principal Forces to Which The Vessel Is Exposed



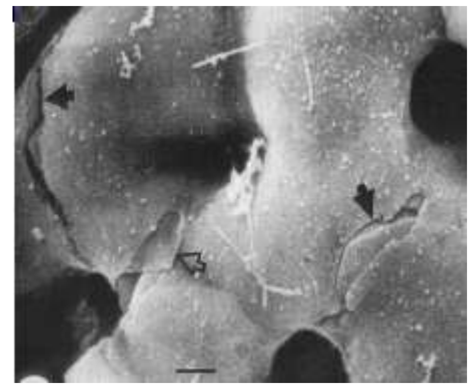
West JB & Mathieu-Costello O. Lancet 1992; 340:762-767

High Capillary Pressure

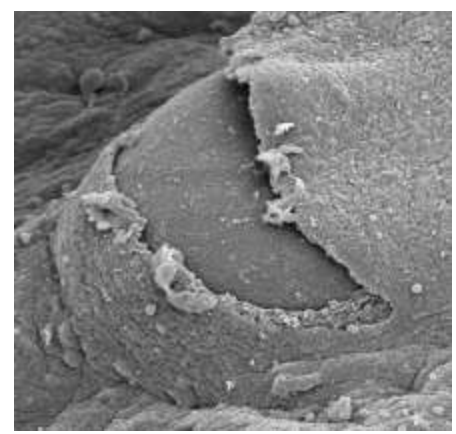
More Injurious at High Lung Volume



Transmission Electron Microscopy



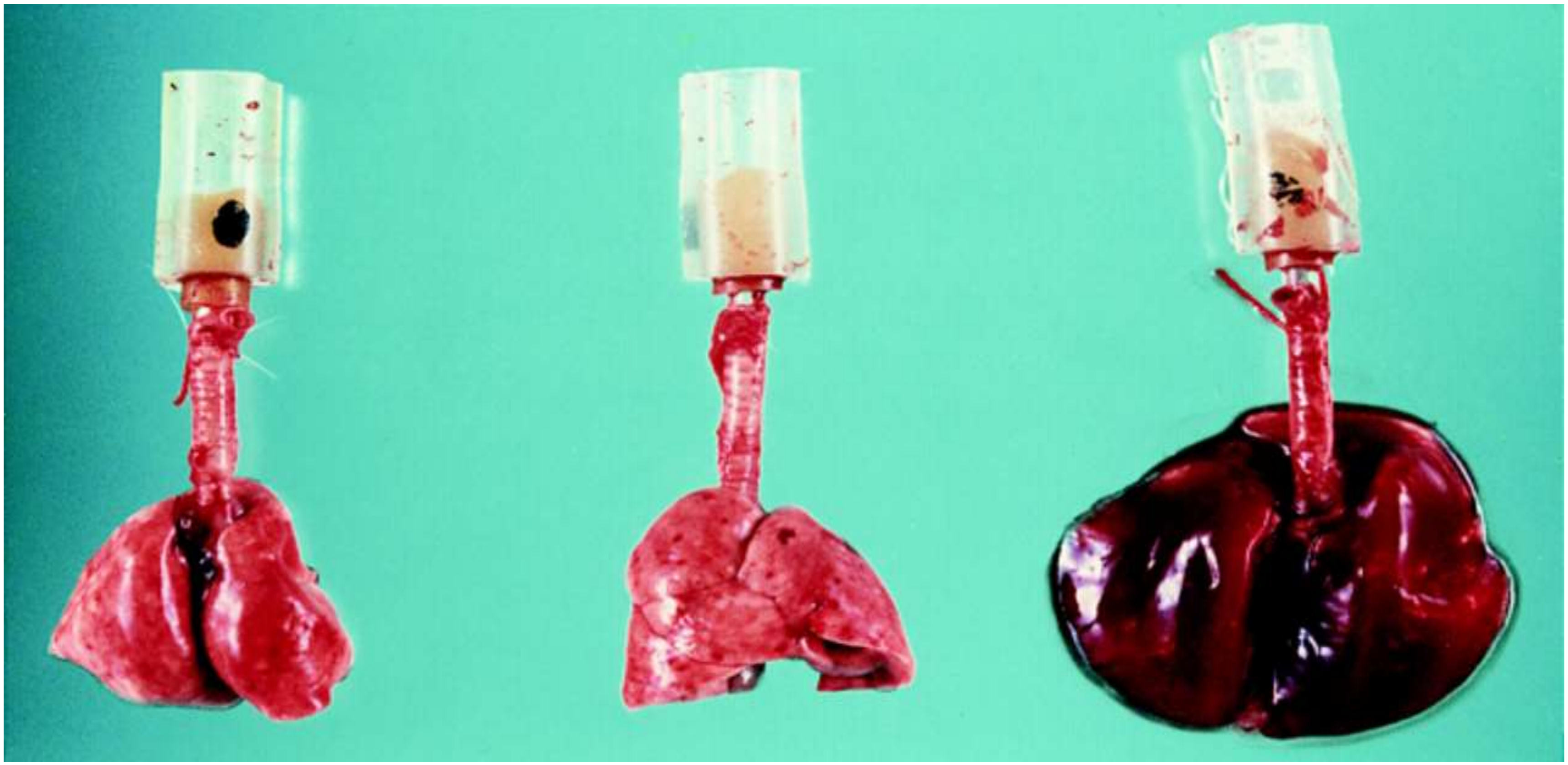
Scanning Electron Microscopy



Fu Z et al. J Appl Physiol 1992; 73:123-133

Effect of 45 cmH_2O PIP

12ml/kgc



Control 5 min 20 min

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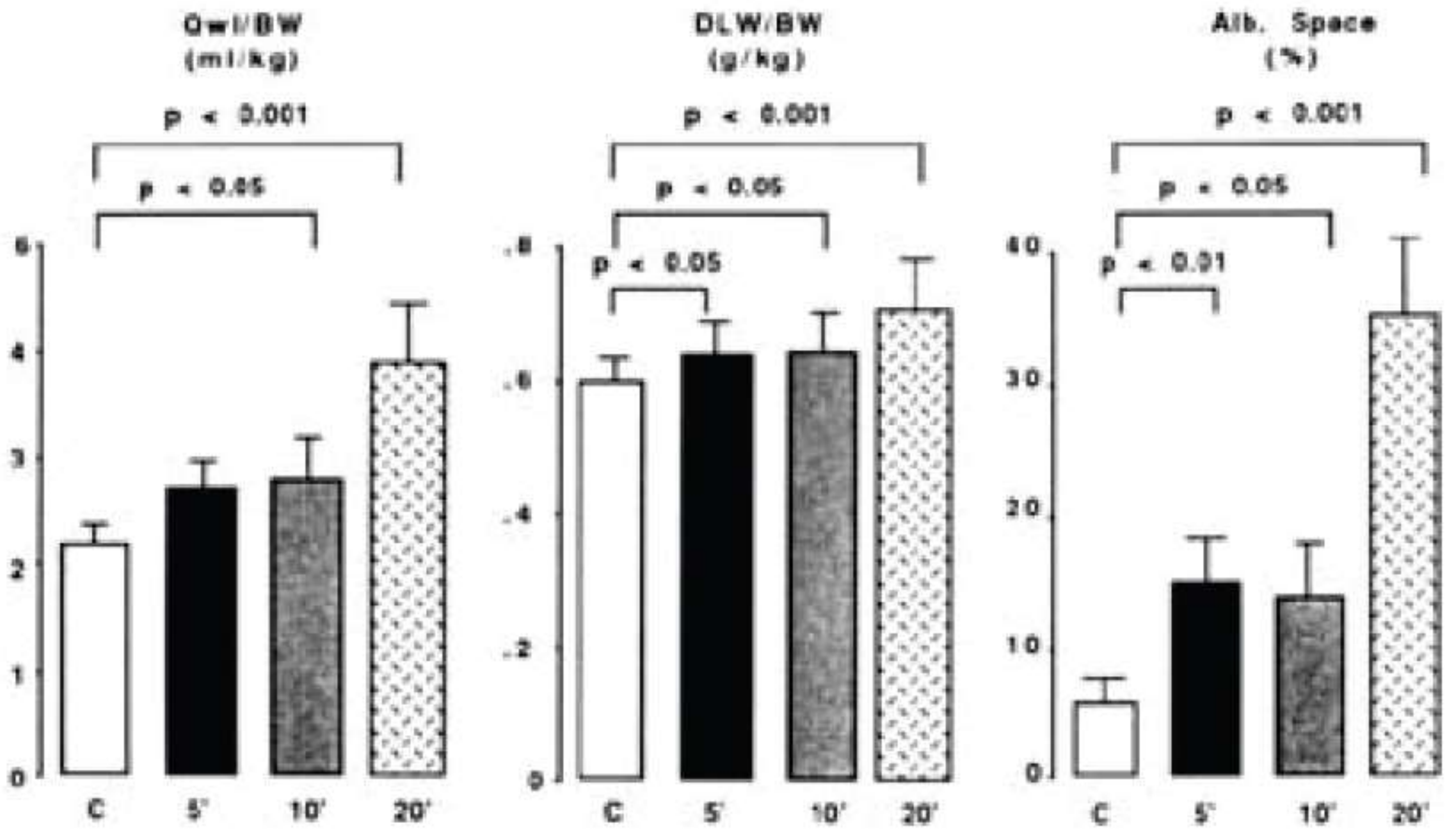
Capillar Permeability

Effects of gradual exposure of normal rats to 45 cmH2O peak airway pressure ventilation on lung water content and pulmonary permeability



Parker JC et al., Increased microvascular permeability in dog lungs due to the high airway presure; Appl Physiol; 57:1809-1816

Liquid Movement in the Pulmonary Capillaries

The Starling Equation

$$Qf = K_f \{(P_C - P_{is}) - \sigma (\Pi_{pl} - \Pi_{is})\}$$

Qf: net flow of fluid

K_f: capillary filtration coefficient

P_c: capillary hydrostatic pressure

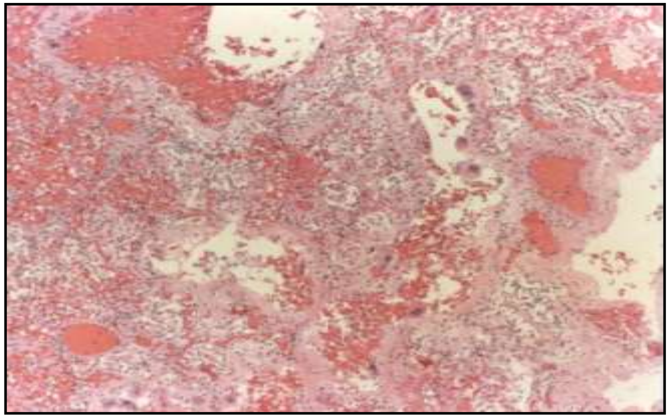
P_{is}: hydrostatic pressure of the interstitial fluid

σ: reflection coefficient

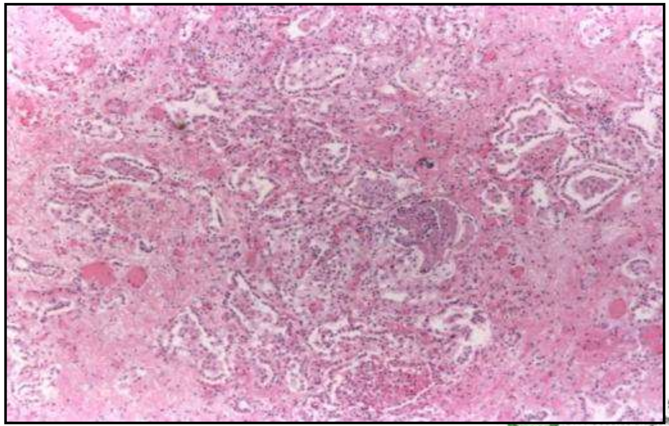
Π_{pl}: oncotic pressure of the plasma

Π_{is}: oncotic pressure of the interstitial fluid

Hemorrhage and Edema



Alveolar Edema and Hyaline Membrane



Mechanisms of VILI

2. Activation of aberrant cellular pathways
   - **Biotrauma**

a process of injury in which biophysical forces can alter the normal physiology of lung cells, increasing the levels of inflammatory mediators and promoting changes in the process of repair/remodelling of lung tissue.

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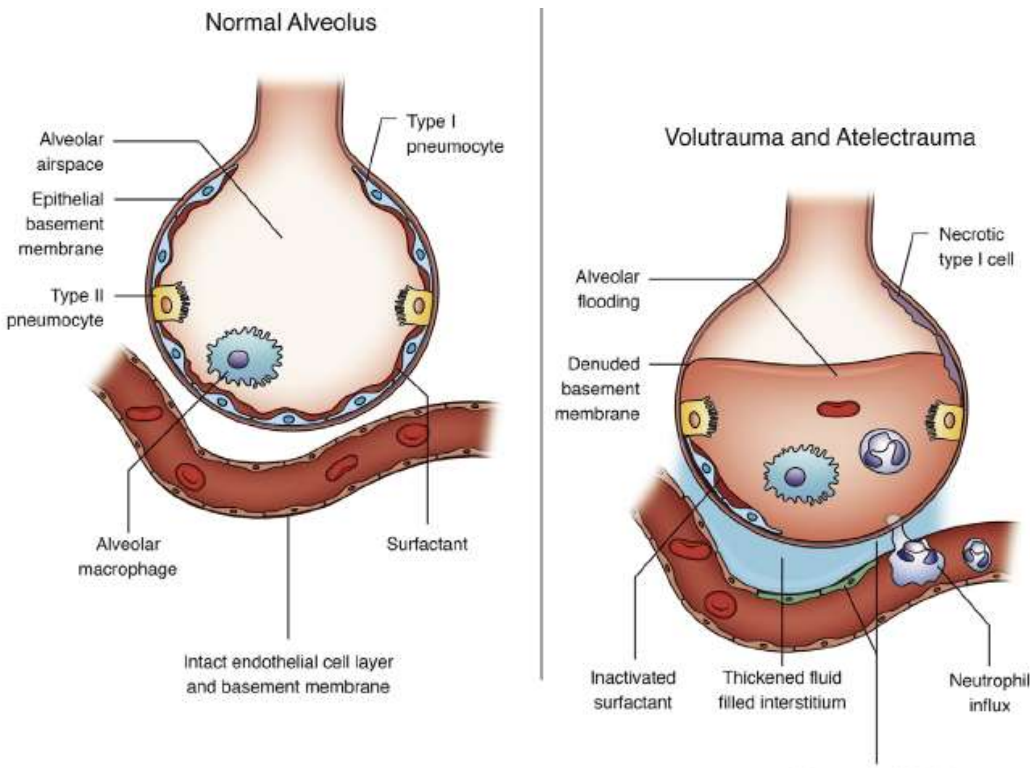
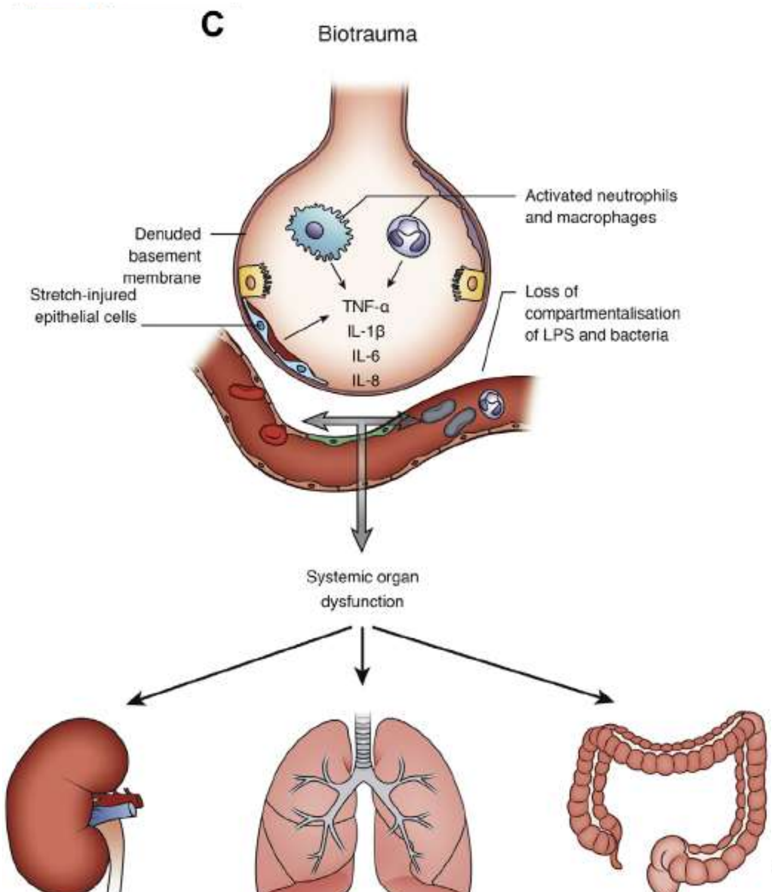
Biotrauma



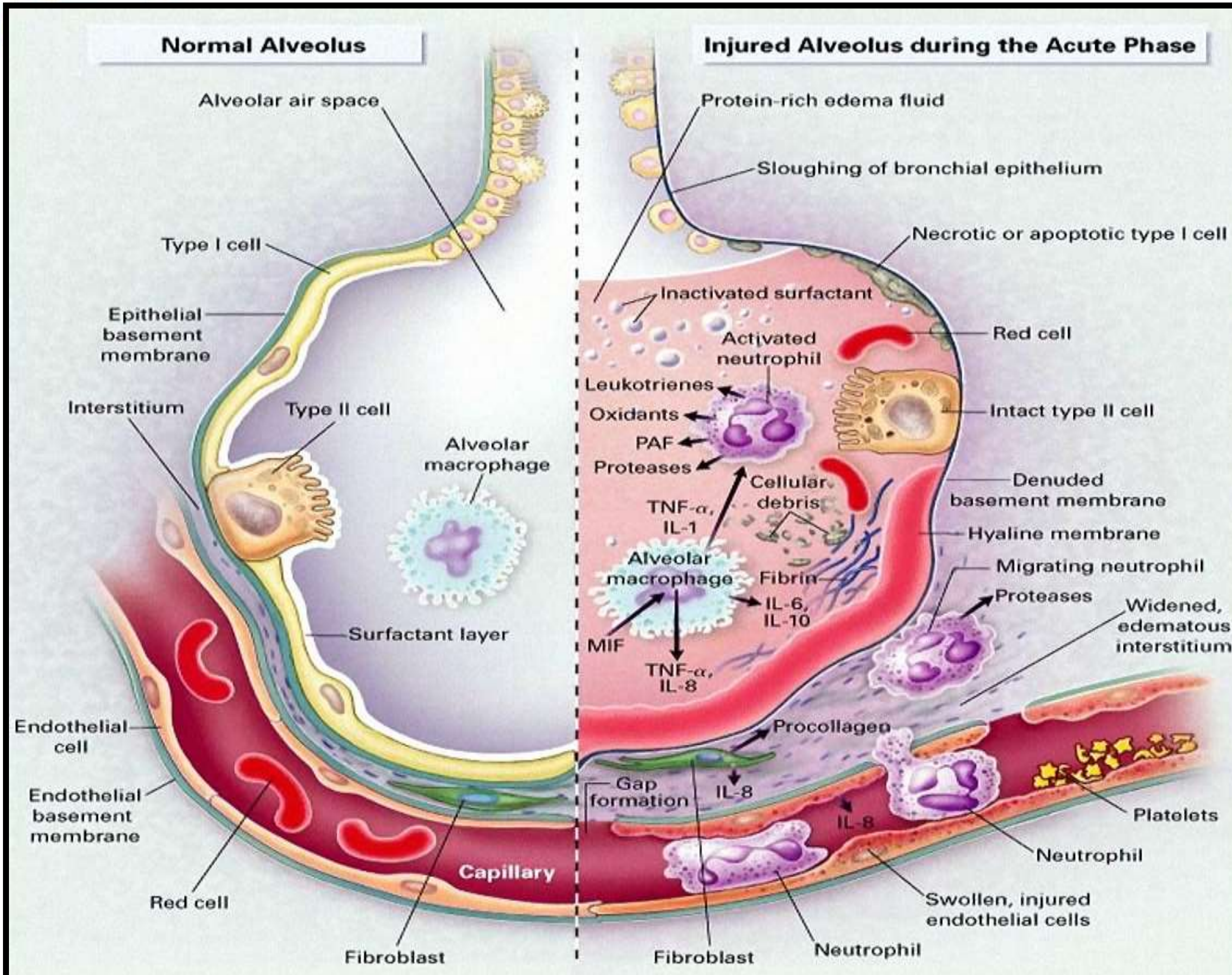


Biotrauma and Ventilator-Induced Lung Injury
Clinical Implications

G.Curley, JG Laffey, AS Slutsky;CHEST 2016; 150:1109-1117

Held HD, Boettcher S, Hamann L, Uhlig S. Am J Respir Crit Care Med. 2001;163:711-716

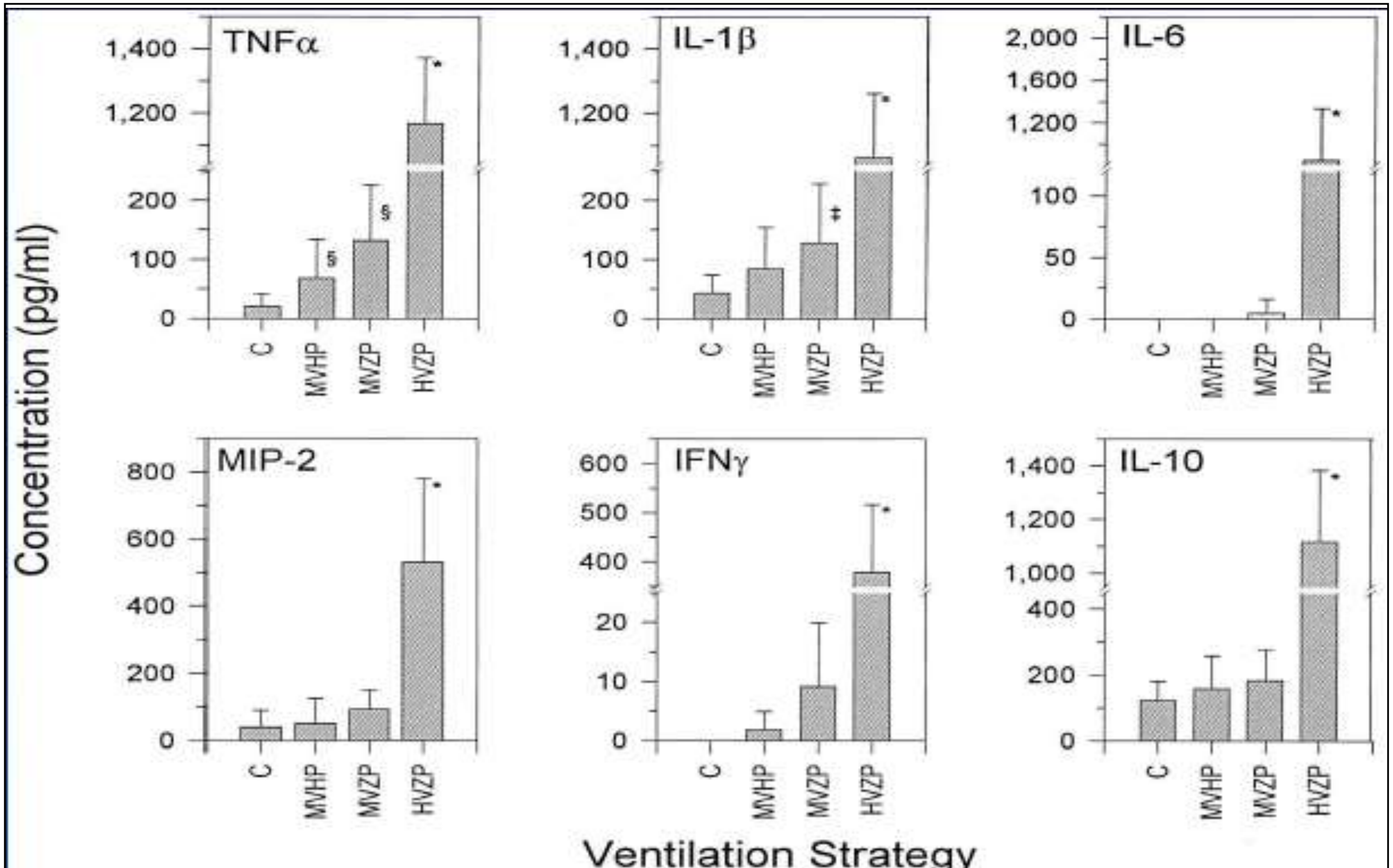
European Society of Anaesthesiology ESA



Ware and Matthay NEJM 342 (18): 1334

Injurious Ventilatory Stategies

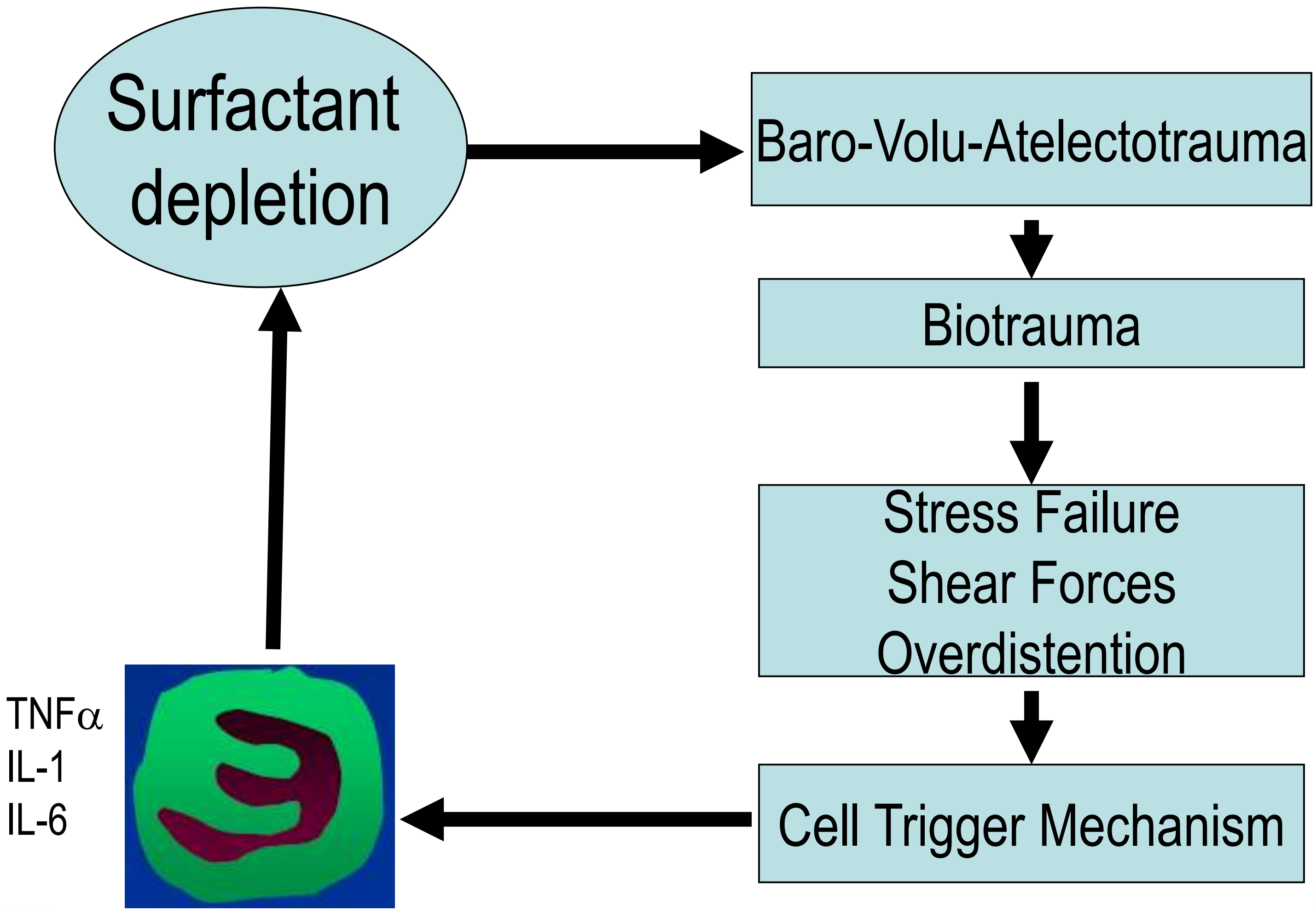
Increase Cytokines in an Isolated Rat Lung Model (Saline Injected Group)



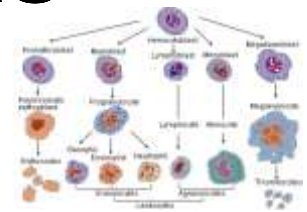
Tremblay L et al. J Clin Invest 1997; 99:944-952

From Barotrauma to Biotrauma

Mechanical Injury Leads to Inflammation



From Barotrauma to Biotrauma



Activation of aberrant cellular pathways

Physical forces

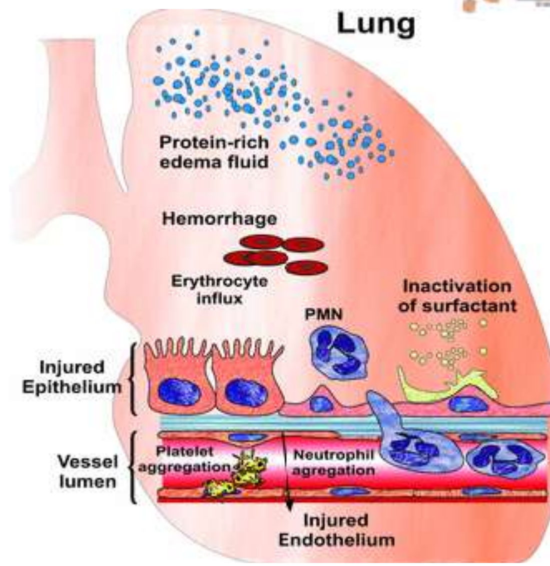
↓

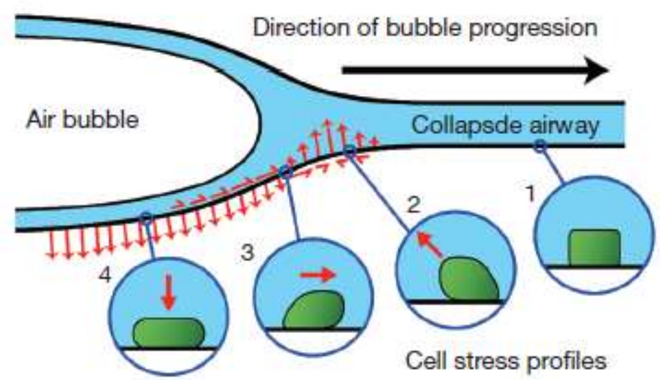
Cellular detection

↓

Biochemical signals

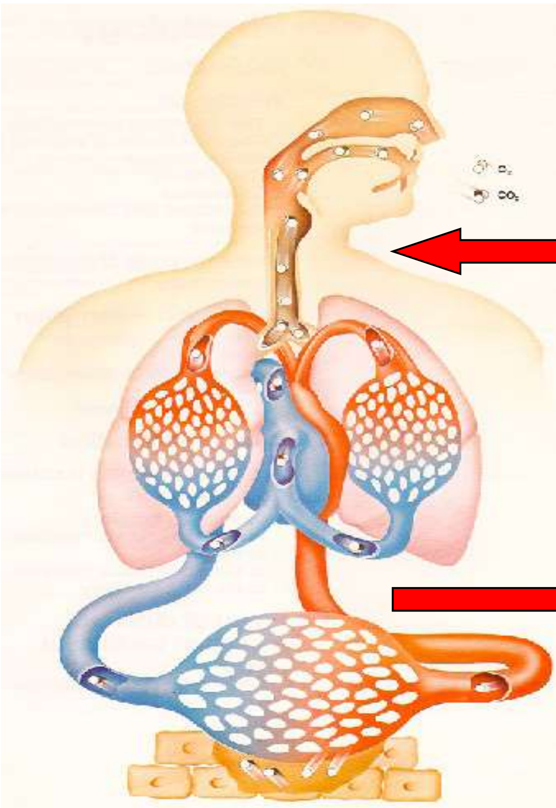
Mechanotransduction

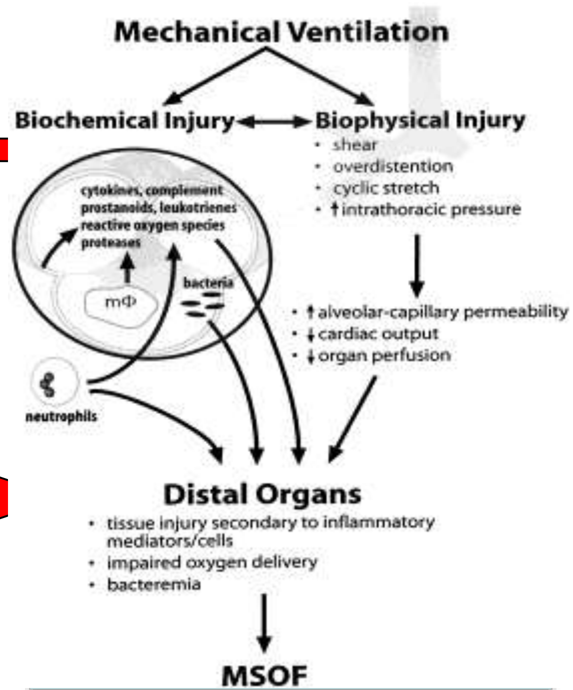


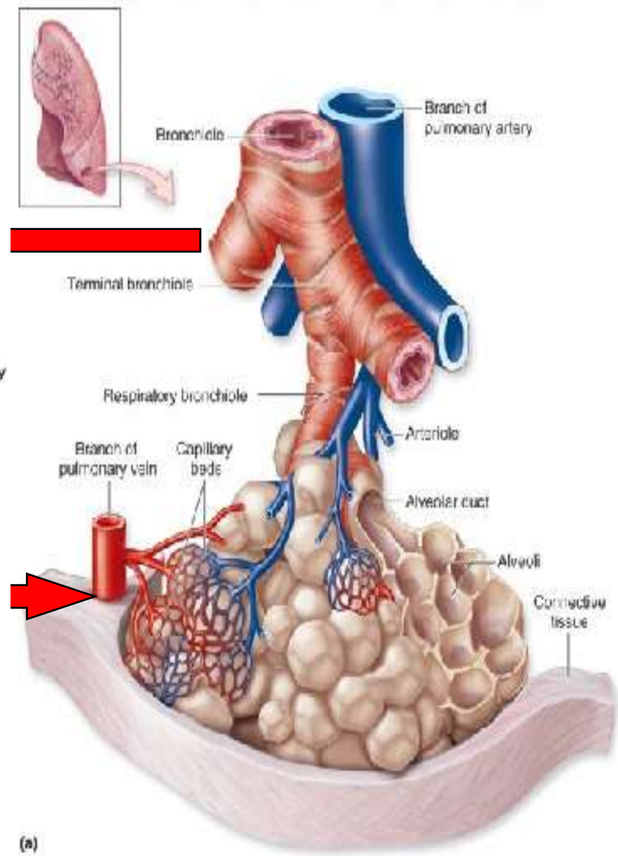


Adapted from Bates JH, Smith BJ. Ventilator-induced lung injury and lung mechanics. Annals of translational medicine. 2018 Oct;6(19).

VILI not limited to lung







Lungs = huge surface,connected to the environment & receiving all the blood volume of the body

Ranieri VM ea. JAMA 1999; 282: 54-61

Two Hit Model of Lung Injury

Normal lung

1st Stage Prevention

Recognition of
"at risk" population

1st hit
Pneumonia, sepsis, shock, trauma, pancreatitis etc.

Initial lung damage

2nd Stage Prevention

Reducing exposure
to 2nd hit factors

2nd hit
Blood transfusion, positive fluid balance, high tidal volum etc.

Lung injury

Springer 2011

Previously Healthy Lungs

- **MV worsens outcomes in patients with previously healthy lungs**
  - during and after prolonged general anesthesia - atelectasis (90%) (spontaneous or mechanically supported) and the type of anesthesia
  - excessive crystalloid use increases capillary hydrostatic pressure and promotes interstitial/alveolar edema, particularly when the lymphatic system is disrupted
  - tissue trauma, ischemia- reperfusion, blood transfusions, and exposure to extracorporeal devices may all combine to result in regional heterogeneity that makes the lung more vulnerable to VILI

*R**othen HU, Sporre B, Engberg G, Wegenius G, Hedenstierna G..** Br J Anaesth. 1998;81(5):681-686. ; **Futier E, Constantin JM, Paugam-Burtz C, et al.. N** Engl J Med. 2013;369(5):428-437. ; **Hemmes SN, Gama de Abreu M, Pelosi P, Schultz MJ. I**. Lancet. 2014;384(9942):495-503.; **Serpa Neto A, Hemmes SN, Barbas CS, et a**l. Lancet Respir Med. 2014;2(12):1007-1015.*

LAS VEGAS

Local **As**sessment of **Ve**ntilatory Management During **G**eneral **A**nesthesia for **S**urgery and effects on Postoperative Pulmonary Complications: a Prospective Observational International Multi–center Cohort Study

Primary Outcome Measures

•Post-operative pulmonary complications, possibly related to ventilation strategy; (PO -day 1, 2,3,4,5 and day 28)

•Effect of Mechanical Ventilation settings during general anesthesia for surgery on the incidence of post-operative pulmonary complications

(new or prolonged invasive or non-invasive mechanical ventilation, need for oxygen therapy, respiratory failure, pneumonia, ARDS, pneumothorax) (1-5 d PO)

LAS VEGAS

Local **As**sessment of **Ve**ntilatory Management During **G**eneral **A**nesthesia for **S**urgery and effects on Postoperative Pulmonary Complications: a Prospective Observational International Multi–center Cohort Study

Secondary Outcome Measures

- Intra-operative complications related to the ventilation strategy
- Mechanical ventilation-settings during general anesthesia for surgery
  - during the surgical procedure, from moment of to tracheal extubation or discharge from operation room
- Variation of applied MV settings within centers
- Variation of applied MV settings between centers on an international basis

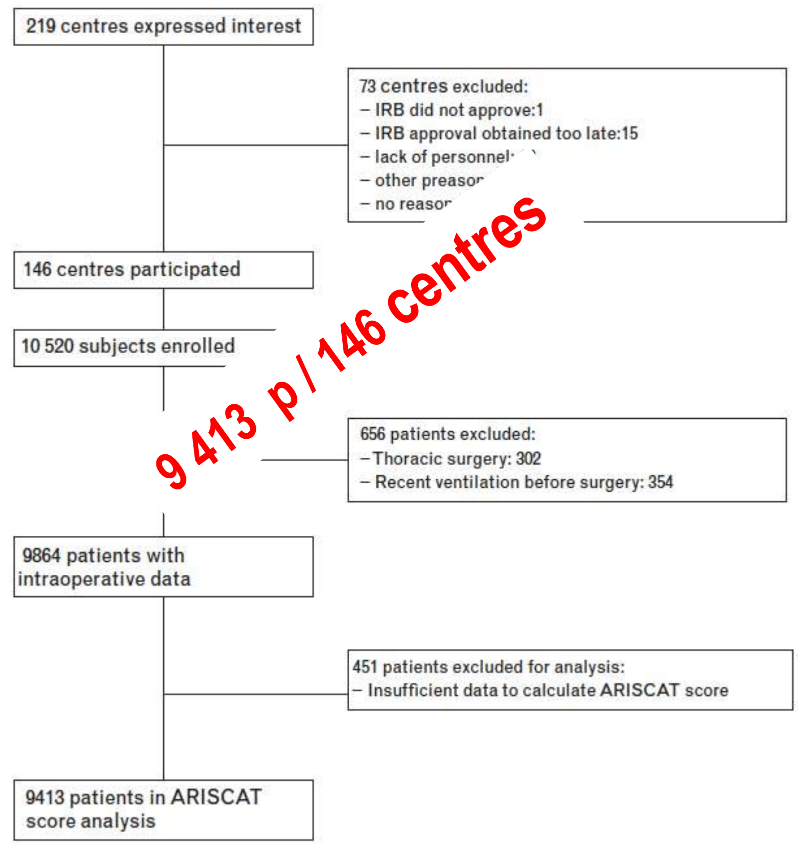
EJA
OPEN
Eur J Anaesthesiol 2017; 34:492–507
ORIGINAL ARTICLE

Epidemiology, practice of ventilation and outcome for patients at increased risk of postoperative pulmonary complications

LAS VEGAS - an observational study in 29 countries

low versus increased risk of PPCs, according to

- (ARISCAT) ; ASA, COPD,;
- **Urgency of surgery**: elective: surgery; medical emergency, urgent: surgery required within<48 h, nonelective surgery performed when the patient's life or well being is in direct jeopardy.
- **Duration of surgery** is the time between skin incision and closure of the incision.
- **Duration of anaesthesia** is the time between start of induction and tracheal extubation or discharge from operation room if MV continued.



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LAS VEGAS - an observational study in 29 countries

PPCs

respiratory infection or failure

bronchospasm

atelectasis

pleural effusion

pneumothorax

aspiration pneumonitis

ɔ PaO2< 8 kPa or SpO2< 90% in room air
2< 90% despite oxygen therapy,
entilation (NIPPV)
echanical ventilation (after discharge from the

efinition of ARDS)
ssive radiographic infiltrate and at least two of
› 100.48 F, leucocytosis or leukopenia (WBC
m l3 and purulent secretions),
ith no vascular bed surrounding the visceral

EJA
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Incidence of patients at increased risk of PPCs

Increased risk of PPCs represented **2670 of 9413** patients ventilated for surgery or **28 cases per 100 surgical procedures** over one week among all types of procedure.

Patients undergoing **transplant surgery or aortic surgery** had the highest incidence of PPCs of all types of surgical procedures

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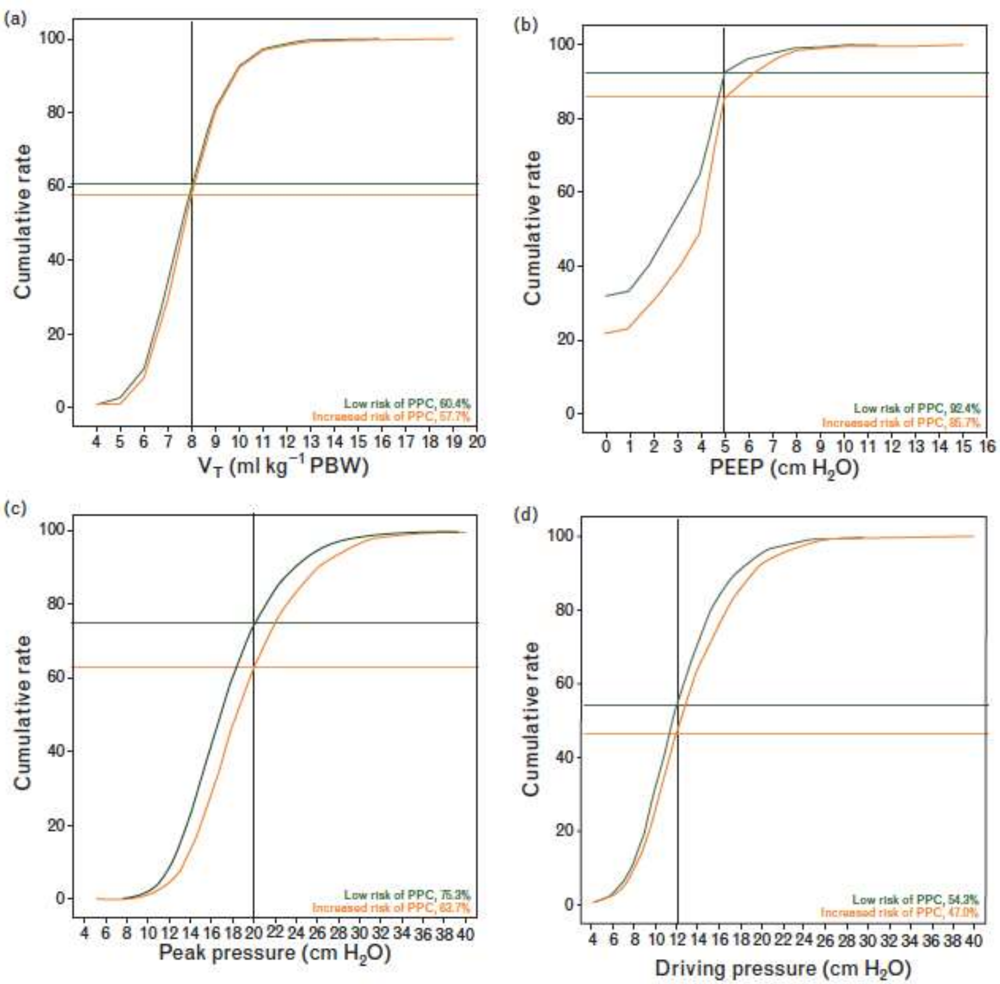

Ventilation parameters in pts at increased vs. pts at low risk of PPCs.

- PBW, predicted body weight
- PEEP, positive end-expiratory pressure
- PPC, postoperative pulmonary complications
- V_T tidal volum

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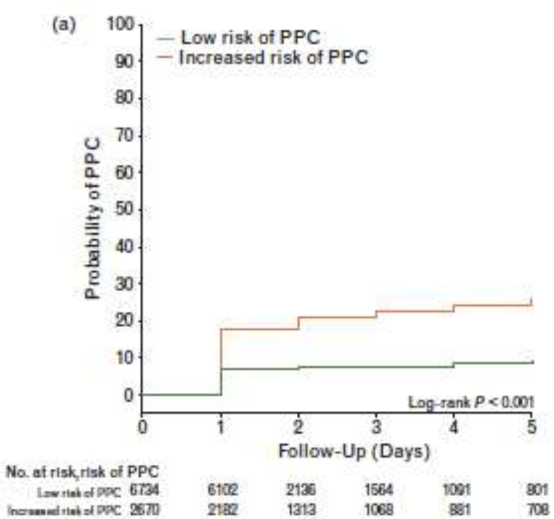
ORIGINAL ARTICLE

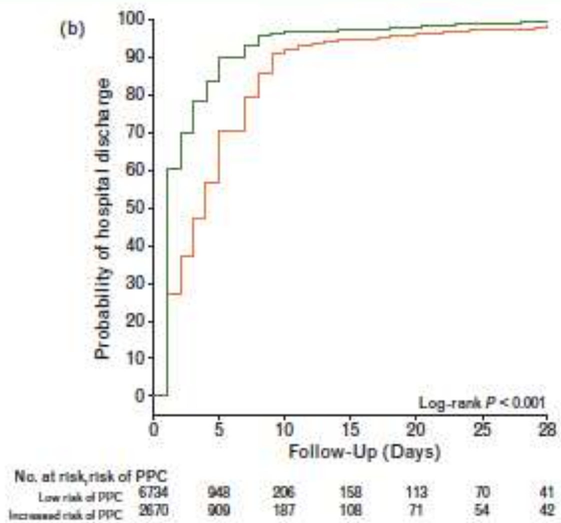
Epidemiology, practice of ventilation and outcome for patients at increased risk of postoperative pulmonary complications

LAS VEGAS - an observational study in 29 countries

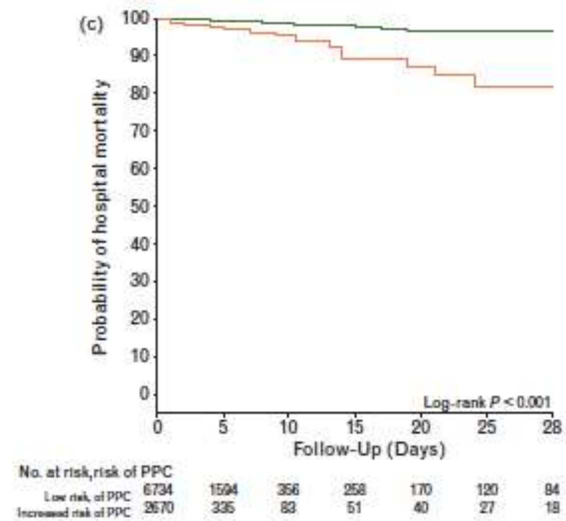
Outcome in patients at increased vs. patients at low risk of PPCs



probability of development of PPCs



probability of hospital discharge



probability of in-hospital mortality

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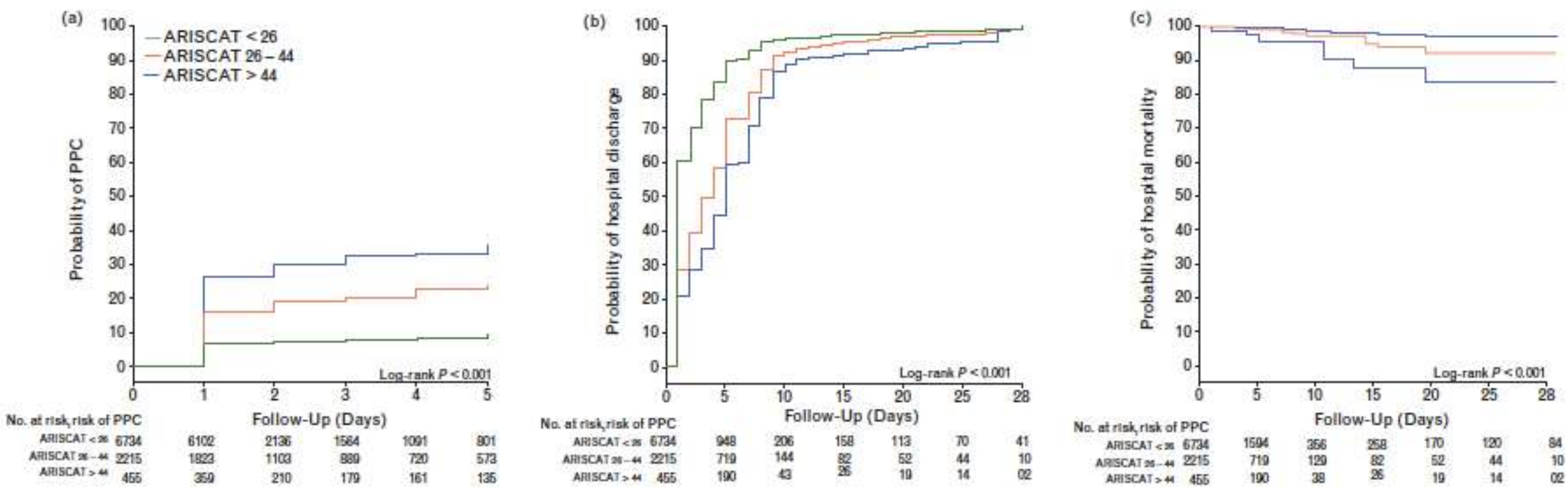

probability of development of PPCs | probability of hospital discharge | probability of in-hospital mortality

ARISCAT - Assess Respiratory Risk in Surgical Patients in Catalonia factors (age, preoperative arterial oxygen saturation in air, acute respiratory infection during the previous month, preoperative anemia, upper abdominal or intrathoracic surgery, surgical duration, and emergency surgery

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Conclusion

- The incidence of patients at risk of **PPCs is high**
- A large proportion of patients receive **high VT and low PEEP levels**, seemingly **independent of the risk of PPCs**
- Patients at increased risk more frequently develop **PPCs, have longer lengths of hospital stay and increased in hospital mortality**
-
- More attention could be given to the use of **lung-protective modes during intraoperative mechanical ventilation in patients at risk of PPCs**.

Other Potential Mechanisms of VILI/VALI

- **Respiratory frequency**
  - cyclic mechanical stress - can lead **to microfractures in lung parenchyma** in the presence of previous lung injury - **reduce the respiratory frequency**

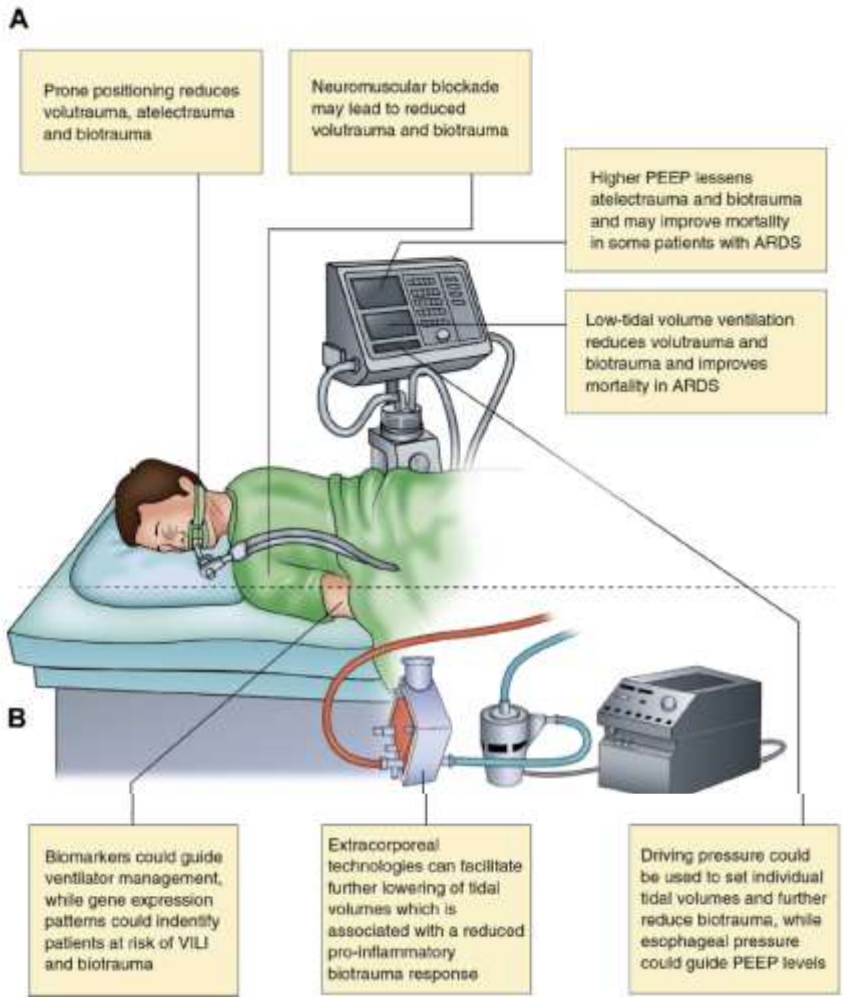
- **Reversed inspiratory time and expiratory time ration (i:e)**
  - increased inspiratory time can exacerbate lung injury, worsening ventilation-perfusion, reducing lung compliance and increasing pulmonary oedema

- **Inspiratory flow**
  - increase tensile stress and transmit the kinetic energy to underlying structures
  - increase shear stress and lead to parenchymal distortion and deformation of the epithelial surface

How to identify VILI/VALI

PRESSURE-VOLUME CURVES	-to identify patients with higher recruitment potential (identification of inflection point and curvilinear or S-shaped pressure-volume curve) -to identify patients with lower recruitment potential (no inflection point and linear pressure-volume curve) -the expiratory pressure-volume curve for evaluating hysteresis in order to identify patients who need PEEP titration -require interrupting mechanical ventilation and could generate data artefacts
COMPUTED TOMOGRAPHY SCAN	-to identify patient with a high or low potential for requirement -can determine the regional response to recruitment -can obtain morpho-functional correlation -distinguish between areas of alveolar collapse and consolidation -limitation: radiation exposure, the need to move patients outside the ICU, lack of information on lung mechanical stress
LUNG ULTRASOUND (at bedside)	-accurate information regarding lung morphology (focal or diffuse aeration loss) -useful for optimizing PEEP -to identify pleural effusion, pneumothorax, alveolar-interstitial syndrome, lung consolidation, pulmonary abscess and lung recruitment/derecruitment
POSITRON EMISSION TOMOGRAPHY	-has improved the understanding of the positive/negative effects of mechanical ventilation interventions and the pathophysiology of ALI/ARDS

Implications for Future Clinical Practice PrecisionVentilation



A. **Protective ventilatory strategy**

- low-tidal volume ventilation
- prone positioning
- PEEP titration

B. **Individualized approach**

- TV and PEEP adjusted using driving pressures or esophageal pressures
- Extracorporeal technologies - facilitate ultralow TVl and reduced biotrauma
- Biomarkers or gene expression patterns could identify patients at high risk of VILI, biotrauma and multiorgan failure prior to intubation and mechanical ventilation.

Individualized Tidal Volumes Using Driving Pressure

- TV adjusted to PBW (a better surrogate than measured weight to adjust for variations in lung size) - predicted body weight [PBW];
  - may be useful in pts with normal lungs, but in pts with ARDS, a variable portion of the lung is not available for ventilation
- normalize tidal volume to the size of the injured lung - performed considerably better than did tidal volume or PEEP
- **"driving pressure" - ratio: TV/C_{RS} = plateau airway pressure – PEEP**
  - suggesting that compliance—an indicator of lung size—is a better surrogate than PBW in TV **C_{RS} = TV / (Ppl - PEEP) = TV / DP**
  - applied experimentally was shown to reduce biotrauma; requires confirmation in prospective randomized trials.

Terragni PP, Rosboch G, Tealdi A, *et al..* ***Am J Respir Crit Care Med****. 2007;175(2):160-166.* ***Amato MB, Meade MO, Slutsky AS, et al****.* ***N Engl J Med.*** *2015;372(8):747-755.* ***Samary CS, Santos RS, Santos CL, et al****.* ***Anesthesiology. 2015****;123(2):423-433.*

Individualized PEEP

Higher PEEP may reduce alveolar stress and improve gas exchange if it recruits lung tissue

Setting the appropriate PEEP is challenging because of the heterogeneity in response (related to the variability in recruitable lung)

Titration of PEEP guided by transpulmonary pressures

- (transpulmonary pressure (Ptp), defined as airway opening pressure minus Pp

$$\mathbf{P_{tp} = P_{alv} - P_p};\qquad Ppt_{end\text{-}exp} = PEEP - Pp_{end\text{-}exp}$$

- (esophageal pressure (Pes) is used as a surrogate for pleural pressure - Pp)

The variable most closely linked with VILI - alveolar distending pressure

- (transpulmonary pressure (Ppl), defined as airway opening pressure minus Pp

***Gattinoni** L, Caironi P, Cressoni M, et al. N Engl J Med. 2006; **Terragni PP**, Filippini C, Slutsky AS, et al.. Anesthesiology. 2013. **Akoumianaki** E, Maggiore SM, Valenza F, et al.. Am J Respir Crit Care Med. 2014. **Talmor D**, Sarge T, Malhotra A, et al.. N Engl J Med. 2008;. **Fish E**, Novack V, Banner-Goodspeed VM, Sarge T, Loring S, Talmor D.. BMJ Open*

New approaches to minimize VILI

1. **Protective ventilatory strategies**:
    a. Low tidal volume
    b. High positive end-expiratory pressure
    c. Recruitment maneuvers
    d. Prone position
    e. High-frequency oscillatory ventilation
    f. Transpulmonary pressure
2. **Super protective ventilatory strategy**
    a. ECMO
    b. ECCO2R

Adapted from Terragni P, Ranieri VM, Brazzi L. Novel approaches to minimize ventilator-induced lung injury. Current opinion in critical care. 2015 Feb 1;21(1):20-5.

Ventilatory Strategies

The NEW ENGLAND JOURNAL of MEDICINE

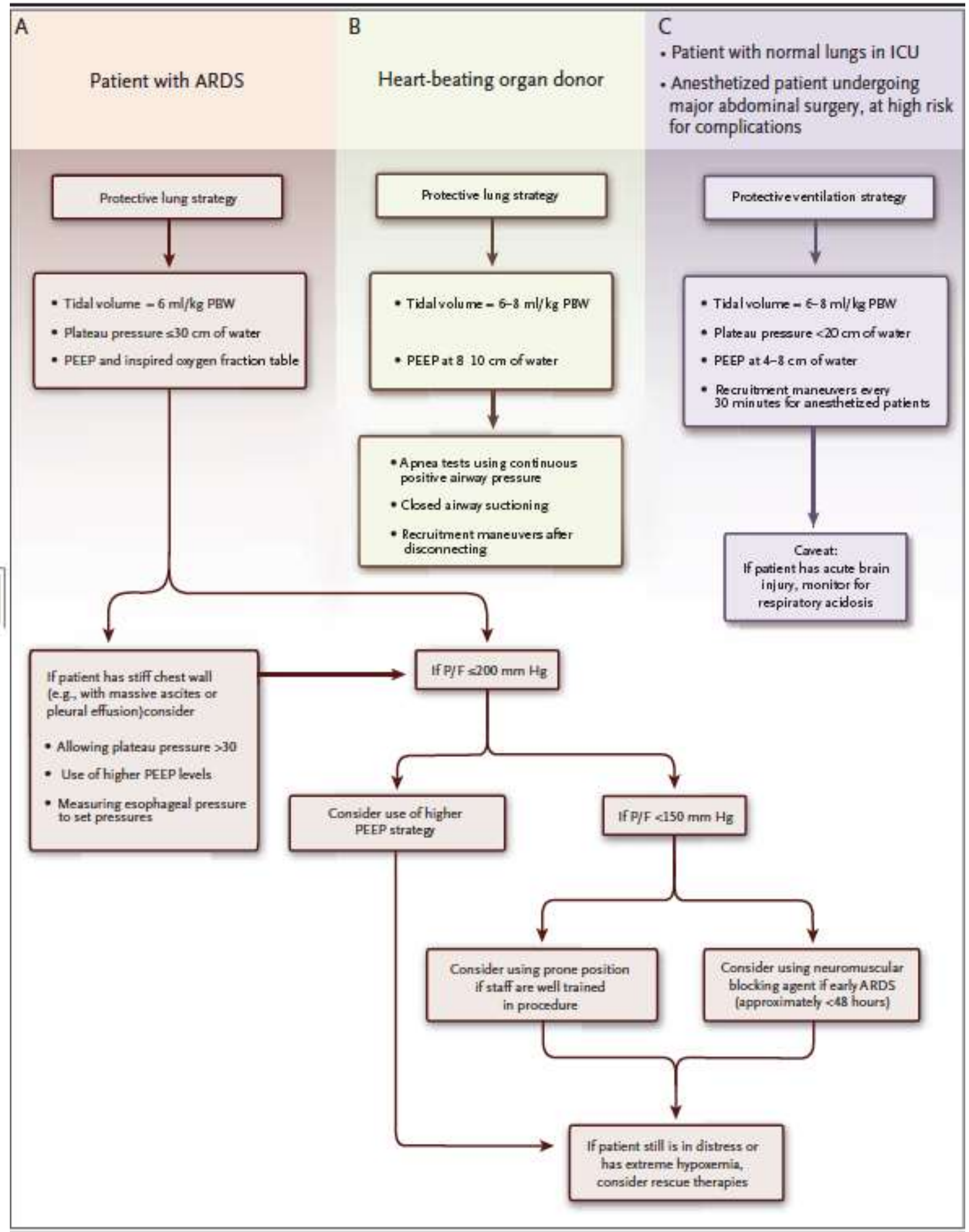
REVIEW ARTICLE

CRITICAL CARE MEDICINE

Simon R. Finfer, M.D., and Jean-Louis Vincent, M.D., Ph.D., *Editors*

Ventilator-Induced Lung Injury

Arthur S. Slutsky, M.D., and V. Marco Ranieri, M.D.



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PATIENT WITH ARDS

If P/F < 200 mmHg

PROTECTIVE LUNG STRATEGY

1. Tidal volume=6 ml/kg PBW
2. Plateau pressure < 30 cm of water
3. PEEP and inspired oxygen fraction table

In case of patients with stiff chest wall (e.g., ascites, pleural effusion) consider:

-plateau pressure >30

-higher PEEP levels

-measuring esophageal pressure to set pressures

Consider use of higher PEEP strategy

- If patient still is in distress or has extreme hypoxemia, consider rescue therapies

If P/F < 150 mmHg

- Consider using prone position if staff are well trained in procedure
- Consider using neuromuscular blocking agent if early ARDS (< 48 hours)

Adapted from Slutsky AS, Ranieri VM. Ventilator-induced lung injury. New England Journal of Medicine. 2013 Nov 28;369(22):2126-36.

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PROTECTIVE LUNG STRATEGY

-Tidal volume = 6-8 ml/kg PBW

-PEEP at 8-10 cm of water

-apnea tests using continuous positive airway pressure

-closed airway suctioning

-recruitment maneuvers after disconnecting

HEART-BEATING ORGAN DONOR

Adapted from Slutsky AS, Ranieri VM. Ventilator-induced lung injury. New England Journal of Medicine. 2013 Nov 28;369(22):2126-36.

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PROTECTIVE VENTILATION STRATEGY

→

-Tidal volume = 6-8 ml/kg PBW

-Plateau pressure < 20 cm of water

-PEEP at 4-8 cm of water

-Recruitment maneuvers every 3-minutes for anesthetized patients

→

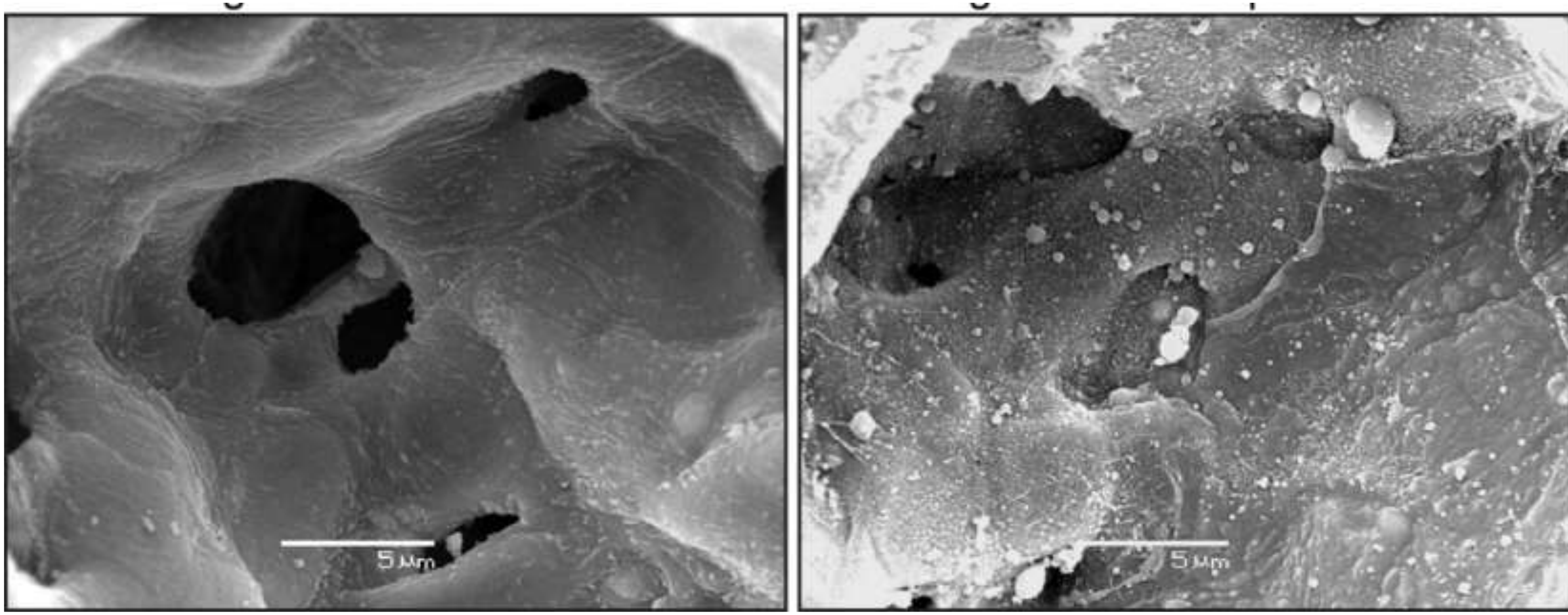
If patient has acute brain injury, monitor for respiratory acidosis

1. **PATIENT WITH NORMAL LUNGS IN ICU**
2. **ANESTHETIZED PATIENT UNDERGOING MAJOR ABDOMINAL SURGERY, AT HIGH RISK FOR COMPLICATIONS**

Adapted from *Slutsky AS, Ranieri VM. Ventilator-induced lung injury. New England Journal of Medicine. 2013 Nov 28;369(22):2126-36.*

Scanning electron microscoph

undamaged alveolar surface vs. fragmented alveolar epithelium



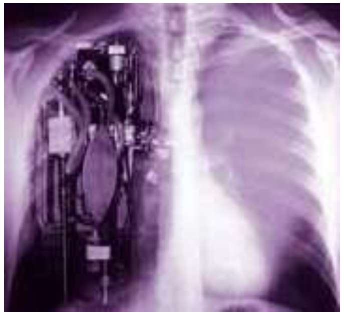
Adapted from *Bates JH, Smith BJ. Ventilator-induced lung injury and lung mechanics. Annals of translational medicine. 2018 Oct;6(19).*

Scanning electron microscoph

undamaged alveolar surface vs. fragmented alveolar epithelium

*Ventilator induced lung injury is a **dysregulated inflammatory response** that occurs as a means of **excessive volume/pressure** (volu- and barotrauma) load in the aerated lung (i.e., the baby lung) along with the cyclic opening and closing of distal airways and/or flooded or collapsed alveoli during tidal ventilation (**atelectrauma**)*

Domenico Luca Grieco, Lu Chen, Laurent Brochard; jul 12,2017



Ventilator-associated lung injury (VALI)

Ventilator-induced lung injury (VILI)

Operator Induced Lung Injury

Decembrie, 2018, Targu Mures

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Happy Holidays!
Sărbători Fericite!