Acute Respiratory Distress Syndrome (ARDS)

and **What You Should Know**

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Conflict of Interest: **None**

I have no perceived conflicts of interest and am not affiliated with any pharmaceutical, equipment, or manufacturing company.

Learning Objectives

As a result of this course, participants will be able to:

1) **Define and describe** acute respiratory distress syndrome in alignment with current Berlin criteria.
2) **Classify** ARDS
3) **Develop evidence-based** mechanical ventilation **strategies** as part of ARDS treatment

Acute Respiratory Distress Syndrome

The Lancet · Satur

ACUTE RESPIRATORY DISTRESS IN ADULTS

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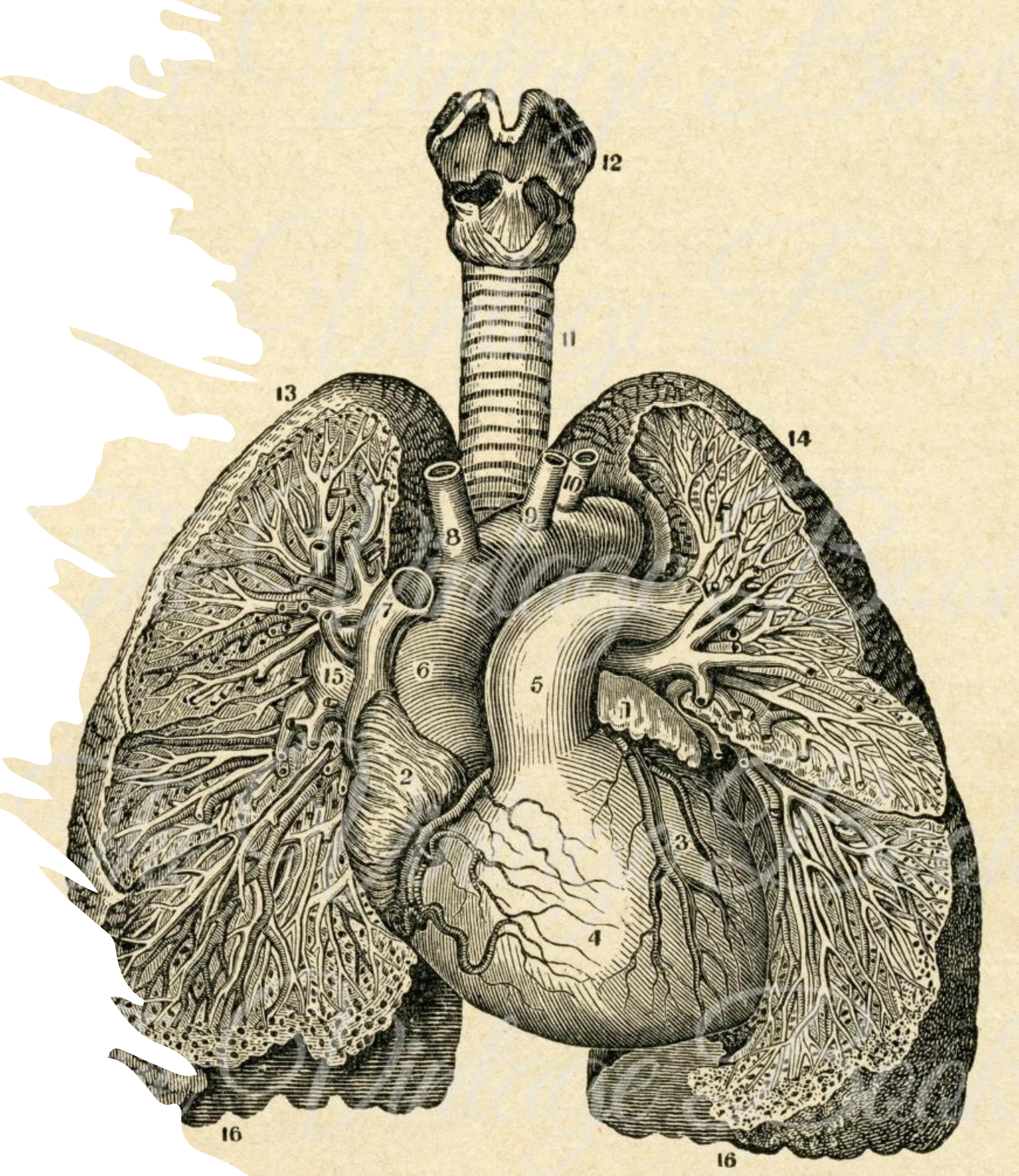
From the Departments of Surgery and Medicine, University of Colorado Medical Center, Denver, Colorado, U.S.A.

Ashbaugh, D., Boyd Bigelow, D., Petty, T., & Levine, B. (1967). ACUTE RESPIRATORY DISTRESS IN ADULTS. *The Lancet*, *290*(7511), 319-323.

Summary The respiratory-distress syndrome in 12 patients was manifested by acute onset of tachypnœa, hypoxæmia, and loss of compliance after a variety of stimuli; the syndrome did not respond to usual and ordinary methods of respiratory therapy. The clinical and pathological features closely resembled those seen in infants with respiratory distress and to conditions in congestive atelectasis and postperfusion lung. The theoretical relationship of this syndrome to alveolar surface active agent is postulated. Positive end-expiratory pressure was most helpful in combating atelectasis and hypoxæmia. Corticosteroids appeared to have value in the treatment of patients with fat-embolism and possibly viral pneumonia.

ARDS Defined

- **1967**
  - First described (*The Lancet*)
- Late 1960’s – early 1990’s
  - various iterations of descriptions and definitions
  - no formal consensus
  - (1988) expanded definition proposed
- **1994**
  - American-European Consensus Conference formal definition
- 2011
  - **Berlin definition** established



SPECIAL COMMUNICATION

ONLINE FIRST

Acute Respiratory Distress Syndrome

The Berlin Definition

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 ≤ 300 mm Hg with PEEP or CPAP ≥5 cm H_2O[c]
Moderate	100 mm Hg < PaO_2/FIO_2 ≤ 200 mm Hg with PEEP ≥5 cm H_2O
Severe	PaO_2/FIO_2 ≤ 100 mm Hg with PEEP ≥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.

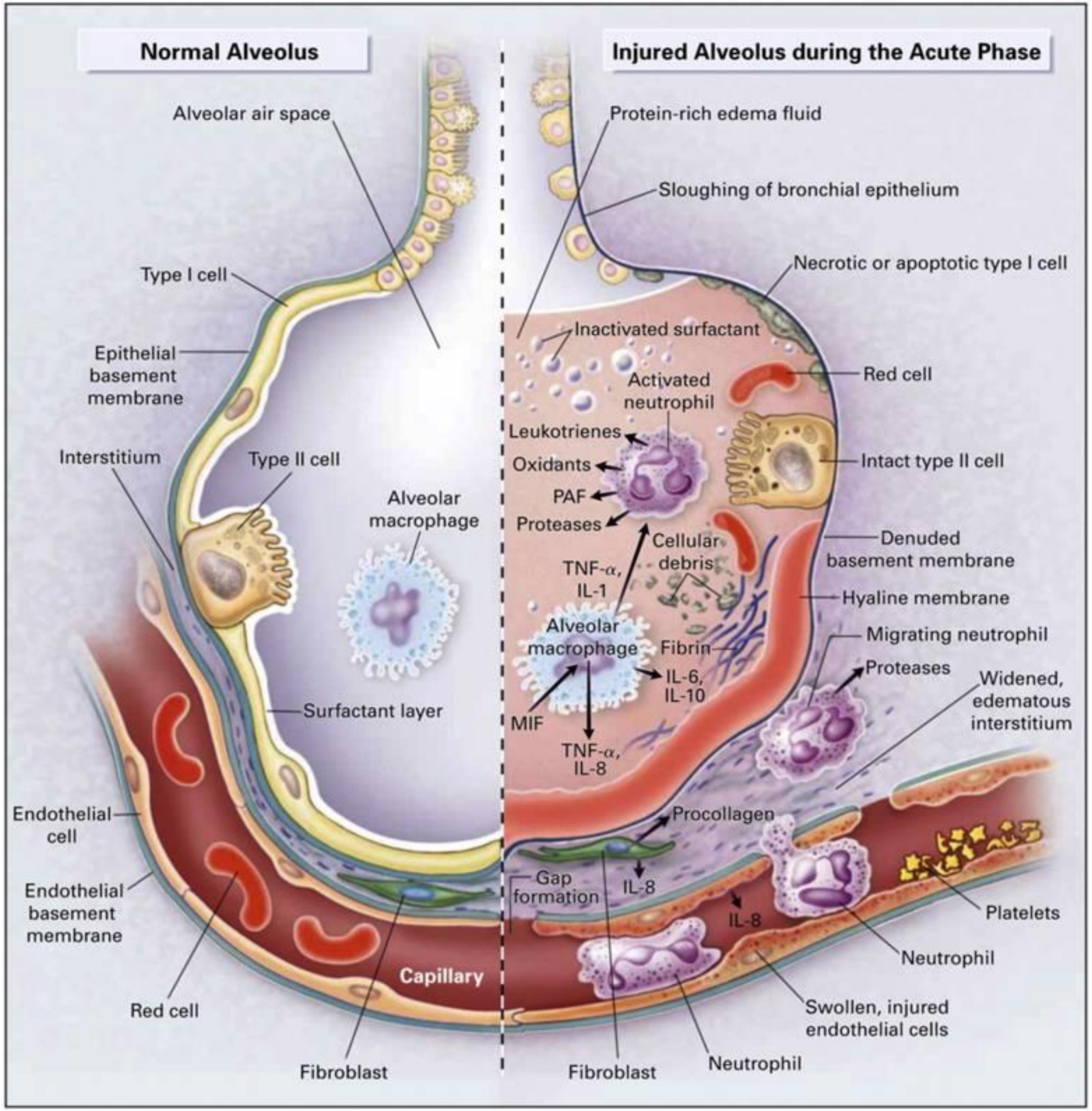
[a] Chest radiograph or computed tomography scan.

[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.

JAMA, June 20, 2012—Vol 307, No. 23

ARDS is a clinical "syndrome" usually **a result of** an insult such as **infection or trauma**



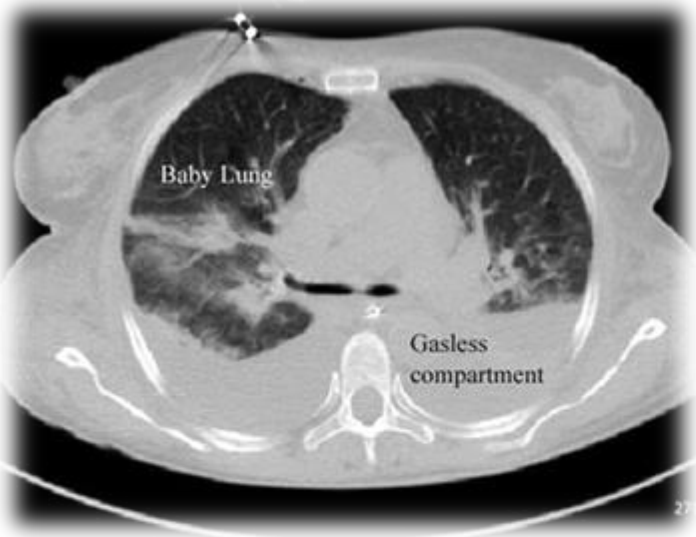
Matthay MA, Zimmerman GA. Am J Respir Cell Mol Biol. 2005;33:319-27

ARDS

Nomenclature

- **A**cute **R**espiratory **D**istress **S**yndrome
  - Acute Lung Injury?
- “Adult” Respiratory Distress Syndrome
- “Wet Lung”; “Shock Lung”
- *Severe* Respiratory Distress Syndrome

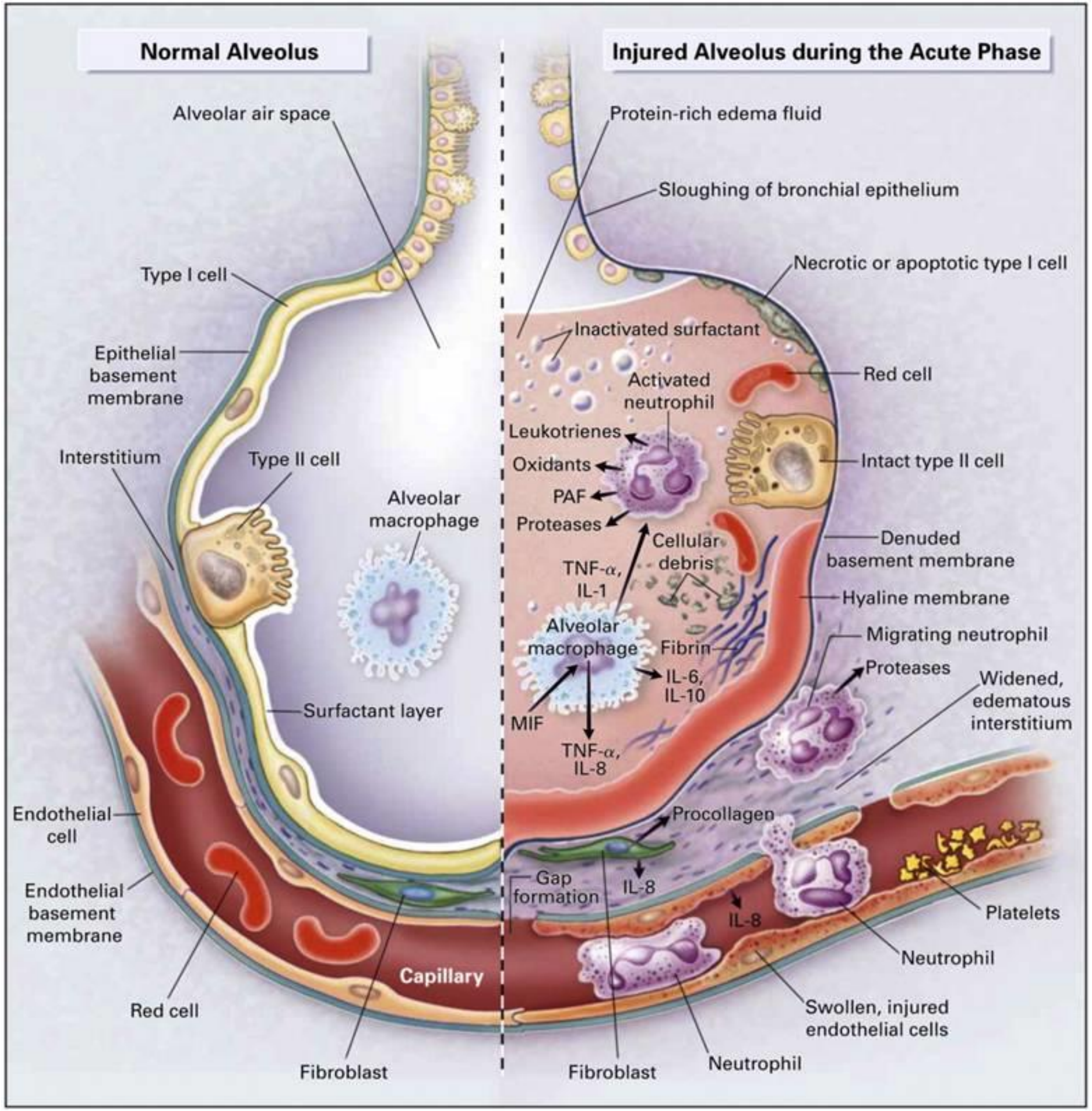
“Baby Lung”



ARDS

Clinical features

- Progressive dyspnea with increasing O2 requirements
- Alveolar infiltrates on chest x-ray & reduced SaO2 (SpO2) within 6-72h of inciting event
- Lab tests are nonspecific



Matthay MA, Zimmerman GA. Am J Respir Cell Mol Biol. 2005;33:319-27

ARDS

CRITICAL CARE PERSPECTIVE

Fifty Years of Research in ARDS

The Epidemiology of Acute Respiratory Distress Syndrome

A 50th Birthday Review

Tài Pham[1,2] and Gordon D. Rubenfeld[1,3]

[1]Interdepartmental Division of Critical Care Medicine, University of Toronto, Toronto, Ontario, Canada; [2]Keenan Research Centre, Li Ka Shing Knowledge Institute, St. Michael's Hospital, Toronto, Ontario, Canada; and [3]Program in Trauma, Emergency, and Critical Care Organization, Sunnybrook Health Sciences Center, Toronto, Ontario, Canada

ATS Journals
Official Publications of the American Thoracic Society

Here's what we know...

Minimizing ventilator-induced lung injury is likely most effective in treating ARDS

AJRCCM, *195*(7), 860-870.

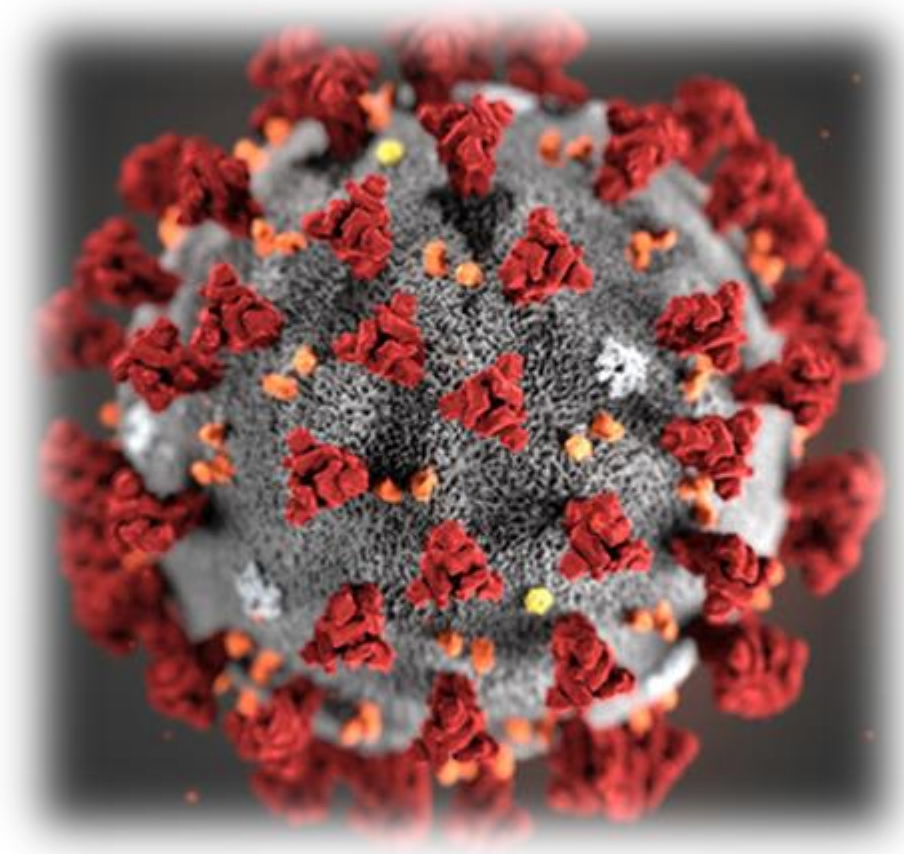
LUNG-SAFE

JAMA | Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

Epidemiology, Patterns of Care, and Mortality for Patients With Acute Respiratory Distress Syndrome in Intensive Care Units in 50 Countries

Giacomo Bellani, MD, PhD; John G. Laffey, MD, MA; Tài Pham, MD; Eddy Fan, MD, PhD; Laurent Brochard, MD, HDR; Andres Esteban, MD, PhD; Luciano Gattinoni, MD, FRCP; Frank van Haren, MD, PhD; Anders Larsson, MD, PhD; Daniel F. McAuley, MD, PhD; Marco Ranieri, MD; Gordon Rubenfeld, MD, MSc; B. Taylor Thompson, MD, PhD; Hermann Wrigge, MD, PhD; Arthur S. Slutsky, MD, MASc; Antonio Pesenti, MD; for the LUNG SAFE Investigators and the ESICM Trials Group

CONCLUSIONS AND RELEVANCE Among ICUs in 50 countries, the period prevalence of ARDS was 10.4% of ICU admissions. This syndrome appeared to be underrecognized and undertreated and associated with a high mortality rate. These findings indicate the potential for improvement in the management of patients with ARDS.



The Epidemiology of Acute Respiratory Distress Syndrome Before and After Coronavirus Disease 2019

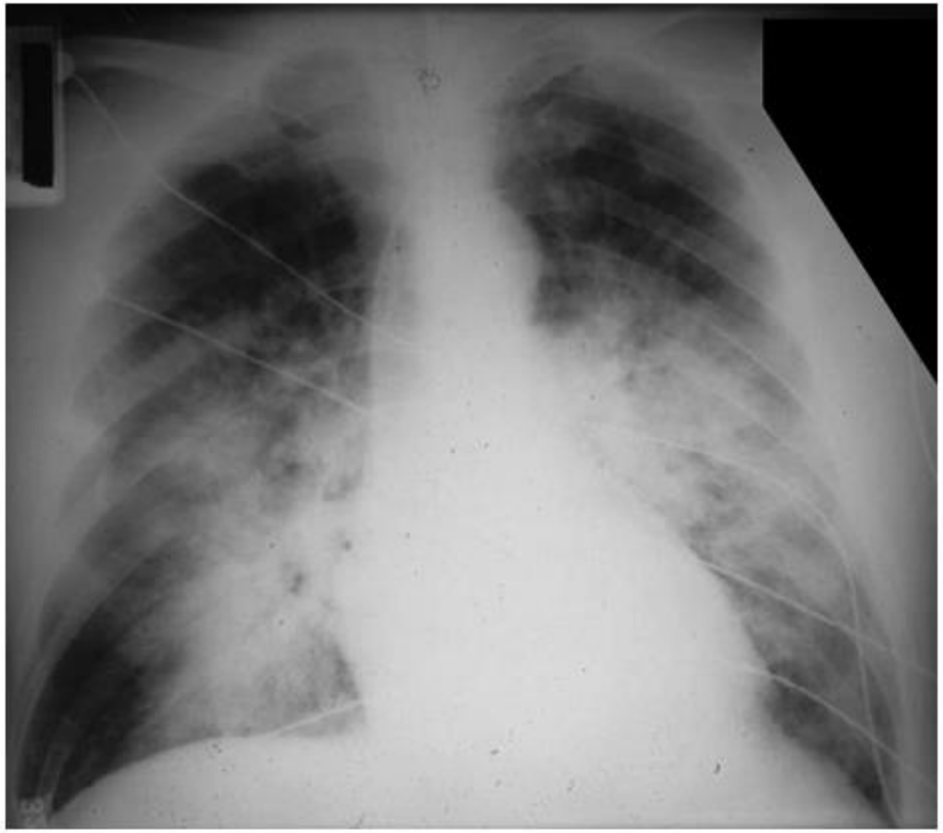
Kathryn W. Hendrickson, MD[a,b], Ithan D. Peltan, MD, MSc[a,c], Samuel M. Brown, MD, MS[a,b,*]

Crit Care Clin. 2021 Oct;37(4):703-716

COVID-19-associated ARDS is a syndrome on the ARDS spectrum and should therefore be treated with the same strategies as classic ARDS while we await results of ongoing trials.

Etiology of acute respiratory distress syndrome*

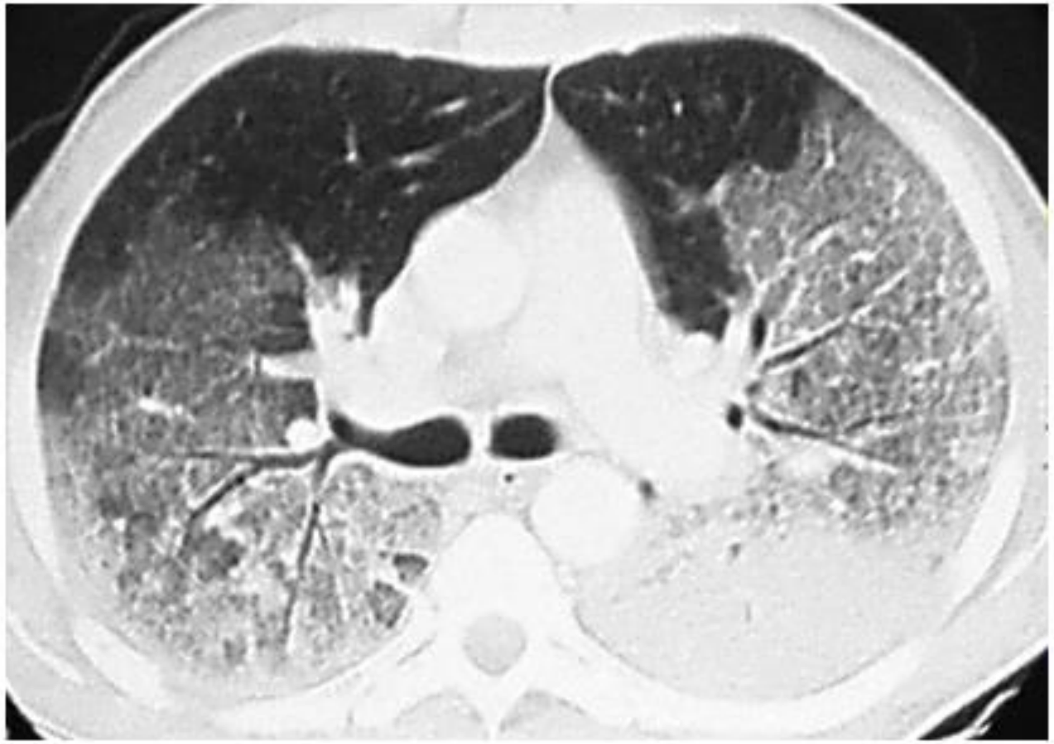
Etiology	Clinical features	Diagnostic tests
Sepsis	Fever, hypotension, leukocytosis, lactic acidosis, infectious source	Appropriate clinical context and positive cultures
Aspiration pneumonitis	Witnessed or risk for aspiration, food, lipid laden macrophages, airway erythema on bronchoscopy	Presumptive diagnosis with negative cultures
Infectious pneumonia (including mycobacterial, viral, fungal, parasitic)	Productive cough, pleuritic pain, fever, leukocytosis, lobar consolidation or bilateral infiltrates in an immunosuppressed patient	Appropriate clinical context and positive respiratory cultures
Severe trauma and/or multiple fractures	History of trauma or fractures within the last week	Diagnosis is apparent
Pulmonary contusion	History of chest trauma (blunt or penetrating), chest pain	Presumptive diagnosis in the correct clinical context, negative cultures
Burns and smoke inhalation	Exposure to fire or smoke, cough, dyspnea, DIC, particulate matter on bronchoscopy, surface burns	Presumptive diagnosis in the correct clinical context, negative cultures
Transfusion related acute lung injury and massive transfusions	History of transfusion, dyspnea during or shortly after transfusion	Diagnosis of exclusion
HSCT¶	History of HSCT	Diagnosis of exclusion
Pancreatitis	Abdominal pain, vomiting, risk actors (eg, gallstones, alcohol, viral infection)	Elevated amylase and lipase, with or without abnormal imaging
Inhalation injures other than smoke (eg, near drowning, gases)	History of inhalation exposure (eg, chlorine gas)	Diagnosis of exclusion
Thoracic surgery (eg, post-cardiopulmonary bypass) or other major surgery	History of surgery, intraoperative ventilation, intraoperative transfusion	Diagnosis of exclusion
Drugs (chemotherapeutic agents, amiodarone, radiation)	New drugs or radiation exposure on history, lymphocytosis on lavage, lavage may have suggestive features of amiodarone toxicity ("foamy macrophages") but is nonspecific	Diagnosis of exclusion, lung biopsy occasionally helpful



Chest radiograph showing diffuse, bilateral, alveolar infiltrates without cardiomegaly in a patient with ARDS.

ARDS: acute respiratory distress syndrome.

Courtesy of Steven E Weinberger, MD.



ARDS due to sepsis after pneumococcal pneumonia.

ARDS: acute respiratory distress syndrome; CT: computed tomography.

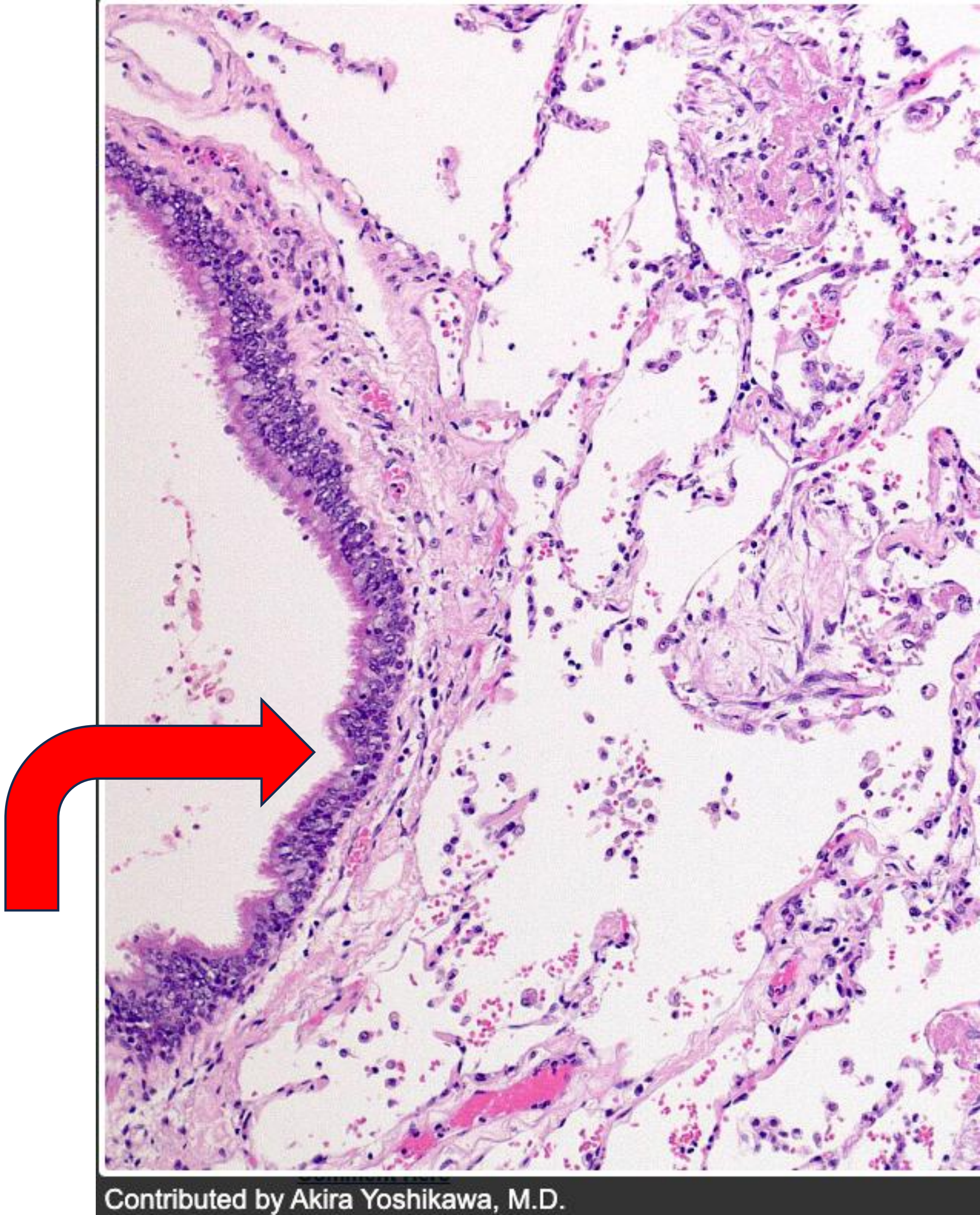
Courtesy of Paul Stark, MD.

Stages of ARDS

Early Exudative Stage (Early)

Fibroproliferative Stage (Mid)

Fibrotic Stage (Later)



RESPIRATORY CARE

Past and Present ARDS Mortality Rates: A Systematic Review

Jan Máca MD, Ondřej Jor MD, Michal Holub MD PhD, Peter Sklienka MD, Filip Burša MD PhD, Michal Burda PhD, Vladimír Janout MD CSc, and Pavel Ševčík MD CSc

Table 5. Absolute Values of the Most Recent Mortality Rates, Since 2010

Mortality Type	Retrospective		Prospective		RCTs		All	
	n	%	*n*	%	*n*	%	*n*	%
In-hospital	345	50	759	47	1,060	41	2,164	45
ICU	547	33	340	46	898	39	1,785	38
28/30-d	1,068	26	606	34	832	31	2,506	30
60-d	751	33	1,294	38	858	23	2,903	32

Retrospective = retrospective observational studies
Prospective = prospective observational studies
RCTs = randomized controlled trials

ARDS-related **mortality** rates remain **high**

Respiratory Care January 2017, 62 (1) 113-122

Ketcham et al. Critical Care (2020) 24:391
https://doi.org/10.1186/s13054-020-03108-w

Critical Care

RESEARCH Open Access

Causes and characteristics of death in patients with acute hypoxemic respiratory failure and acute respiratory distress syndrome: a retrospective cohort study

Scott W. Ketcham[1*], Yub Raj Sedhai[1], H. Catherine Miller[1], Thomas C. Bolig[1], Amy Ludwig[1], Ivan Co[1,2], Dru Claar[1], Jakob I. McSparron[1], Hallie C. Prescott[1,3,4] and Michael W. Sjoding[1,3]

Results: We identified 385 decedents with AHRF, of whom 127 (33%) had ARDS. The most common primary causes of death were sepsis (26%), pulmonary dysfunction (22%), and neurologic dysfunction (19%). Multi-organ failure was present in 70% at time of death, most commonly due to sepsis (50% of all patients), and 70% were on significant respiratory support at the time of death. Only 2% of patients had insupportable oxygenation or ventilation. Eighty-five percent died following withdrawal of life support. Patients with ARDS more often had pulmonary dysfunction as the primary cause of death (28% vs 19%; $p = 0.04$) and were also more likely to die while requiring significant respiratory support (82% vs 64%; $p < 0.01$).

(Continued on next page)

SPECIAL COMMUNICATION

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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.

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

JAMA, June 20, 2012—Vol 307, No. 23

PERSPECTIVE

Redefining ARDS: a paradigm shift

Jesús Villar[1,2,3*], Tamas Szakmany[4,5], Giacomo Grasselli[6,7] and Luigi Camporota[8,9]

$$\text{P/F ratio} = \frac{P_aO_2}{F_IO_2}$$

Table 1 Limitations of the current definition and diagnostic/therapeutic approach of ARDS

Bilateral and diffuse pulmonary edema	Lack of a marker of non-cardiogenic origin of pulmonary edema
	Lack of a (bio)marker of pulmonary vascular permeability
Oxygenation	A single measurement of PaO_2/FiO_2 at ARDS onset or diagnosis has poor performance for definition or predicting severity
	Lack of standardization of respiratory support settings for measuring PaO_2/FiO_2
	Difficult to distinguish ARDS from acute hypoxemic respiratory failure since clinical features and etiologic causes are similar
Lung mechanics	Not required in the current definition
	Missing dead space (VD/VT) measurement in definition and progression
	Hard to conceive a mechanically ventilated ARDS patient receiving PEEP $\leq$ 5 cmH_2O
Systemic inflammation	Definition and categorization do not account for non-pulmonary organ failure, which is present in most patients and a major determinant of outcome
	Too much emphasis on the alveolar side. Little consideration for the pulmonary vascular and endothelial side, presence of pulmonary hypertension or right ventricular function
	Systemic inflammation seen in ARDS based on protein and mRNA biomarkers is not specific for ARDS, especially in septic patients
Categorization and sub-phenotyping	Missing stratification in sub-phenotypes based on VD/VT, endothelial injury, biomarker levels, or modifiable or treatable traits
	It is highly plausible that in a substantial proportion of patients in recent trials, the severity of lung injury was modest
Mechanical ventilation setting	It should be personalized based on etiology, lung physiology, imaging and morphology, and clinical and biological classes or subclasses
	In some ARDS trials, unselected patients could be enrolled missing the opportunity to test whether the experimental MV approach is beneficial due to lack of standardized assessment of severity prior to randomization and to lack of patient sub-phenotyping

ARDS, acute respiratory distress syndrome; mRNA, messenger ribonucleic acid; MV, mechanical ventilation; PEEP, positive end-expiratory pressure; VT/VT, dead space

Villar et al. Critical Care (2023) 27:416

PERSPECTIVE

Redefining ARDS: a paradigm shift

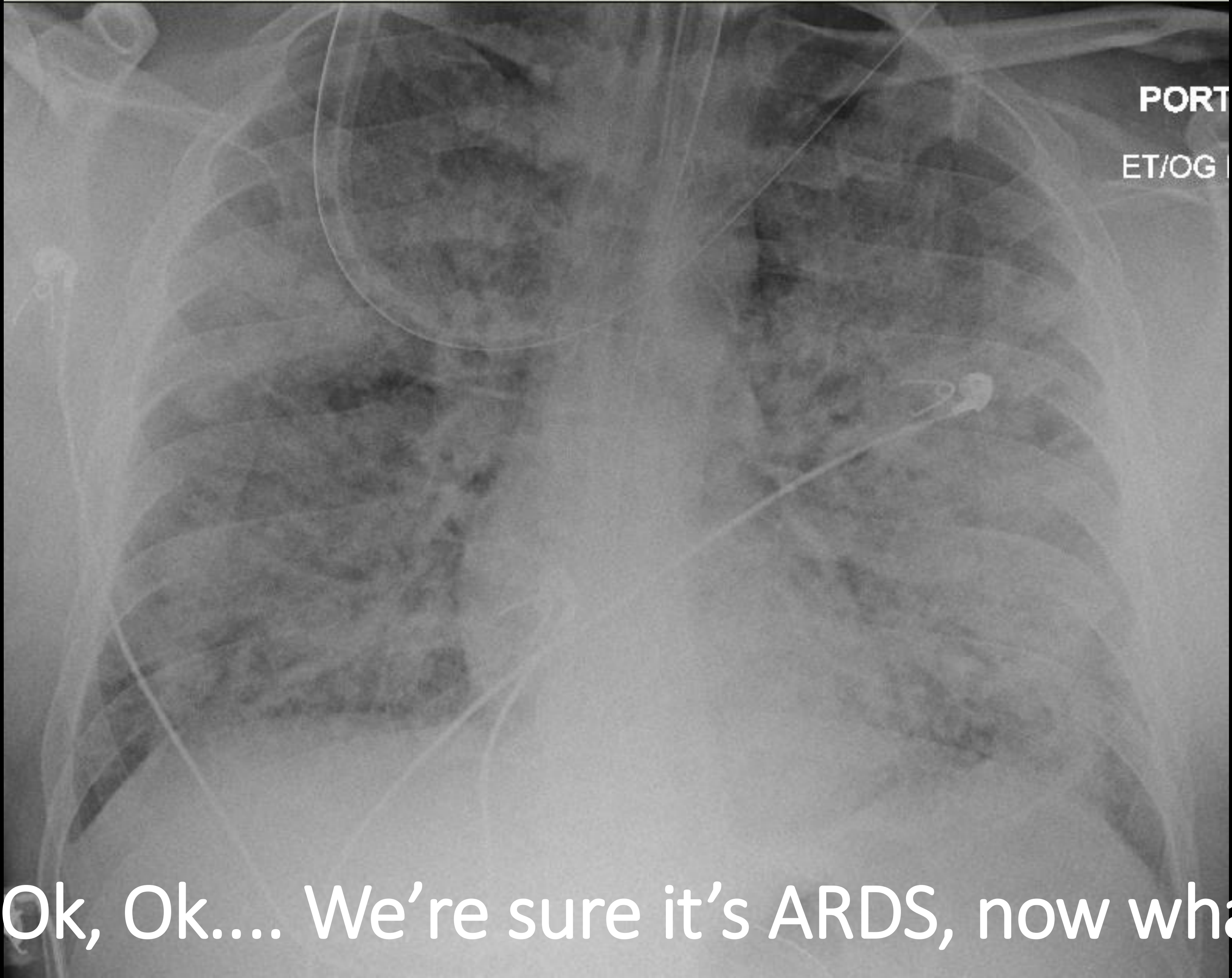
Jesús Villar[1,2,3*], Tamas Szakmany[4,5], Giacomo Grasselli[6,7] and Luigi Camporota[8,9]

Table 2 Potential recommendations for improving the definition of ARDS

New datasets	1. Expiration date for observational studies and trials conducted before year 2010
Actionable criteria	2. Definition should be based on actionable and modifiable criteria, including VD/VT, lung imaging, biomarker levels, etc.
PaO_2/FiO_2	3. It should be assessed under standardized conditions (e.g., measured at predefined FiO_2 and PEEP levels)
	4. Categorization may include the threshold of 150 mmHg (< 150, ≥ 150)
Measures of severity	Two measures of "true" severity of ARDS:
	5. Lung injury per se: "Severe" ARDS should not be based only on PaO_2/FiO_2
	6. Severity of patient illness, including comorbidities and frailty
Enrichment strategies	7. Prediction or prognostic enrichment strategies for inclusion of patients into therapeutic clinical trials. The use of artificial intelligence techniques may help
Pulmonary circulation	8. More precise information about the anatomic/physiologic state of the pulmonary vascular circulation
Stratification, classification, or sub-phenotyping	9. An updated definition requires a new categorization or classification of severity based on gas-exchange, lung imaging, VD/VT, biomarker levels, use of non-invasive mechanical ventilation, degree of vascular permeability
Broadening definition	10. Excessive broadening of criteria required to diagnose ARDS should be avoided
International professional societies	11. Recommendations for management and treatment in the new updated ARDS definition should be implemented by International Professional Societies
Implementation	12. Implementation of a "Surviving ARDS (including patients at risk for) Campaign" with frequent updates

ARDS, acute respiratory distress syndrome; VD/VT, dead space

Villar et al. Critical Care (2023) 27:416



Ok, Ok…. We’re sure it’s ARDS, now what…?

CLINICS IN CHEST MEDICINE

ELSEVIER SAUNDERS

Clin Chest Med 27 (2006) 725–731

Why Do Patients Who Have Acute Lung Injury/Acute Respiratory Distress Syndrome Die from Multiple Organ Dysfunction Syndrome? Implications for Management

Jean-Louis Vincent, MD, PhD*, Massimo Zambon, MD

Department of Intensive Care, Erasme Hospital, Free University of Brussels, Route de Lennik 808, 1070 Brussels, Belgium

Acute respiratory distress syndrome and multiple organ failure

Lorenzo Del Sorbo[a] and Arthur S. Slutsky[b,c]

[a]Dipartimento di Anestesia e Terapia Intensiva, Azienda Ospedaliera San Giovanni Battista, Università di Torino, Italy, [b]Keenan Research Center at the Li Ka Shing Knowledge Institute of St. Michael's Hospital, Interdepartmental Division of Critical Care, Division of Respiratory Medicine, University of Toronto, Toronto, Ontario, Canada and [c]King Saud University, Riyadh, Saudi Arabia

Correspondence to Lorenzo Del Sorbo
Tel: +39 011 633 5502;
e-mail: lorenzo.delsorbo@unito.it

Current Opinion in Critical Care 2011, 17:1–6

Purpose of review

Despite improvements in outcome due to lung protective ventilation strategies using low tidal volumes, the mortality rate from acute respiratory distress syndrome (ARDS) remains unacceptably high, ranging from 34 to 64%. The predominant cause of death in ARDS is not severe hypoxemia, which is one of the defining criteria of ARDS, but multiple organ failure (MOF).

Recent findings

In view of the relationship between ARDS and MOF, two different but complementary pathophysiological perspectives will be developed in this article: ARDS as a consequence of MOF, and ARDS as the cause of MOF. This framework may be useful in guiding the development of novel therapeutic strategies that ultimately improve the

Characteristics of Nonpulmonary Organ Dysfunction at Onset of ARDS Based on the Berlin Definition

Richard H Kallet, Michael S Lipnick, Hanjing Zhuo, Lance P Pangilinan, and Antonio Gomez

Ketcham *et al. Critical Care* (2020) 24:391
https://doi.org/10.1186/s13054-020-03108-w

Critical Care

RESEARCH

Open Access

Causes and characteristics of death in patients with acute hypoxemic respiratory failure and acute respiratory distress syndrome: a retrospective cohort study

Check for updates

Scott W. Ketcham[1*], Yub Raj Sedhai[1], H. Catherine Miller[1], Thomas C. Bolig[1], Amy Ludwig[1], Ivan Co[1,2], Dru Claar[1], Jakob I. McSparron[1], Hallie C. Prescott[1,3,4] and Michael W. Sjoding[1,3]

clinical implications of basic research

Mechanisms of Multiple Nonpulmonary Organ Failure in ARDS*

Paul M. Dorinsky, M.D., F.C.C.P.; and James E. Gadek, M.D., F.C.C.P.

Postgraduate Education Corner

CONTEMPORARY REVIEWS IN CRITICAL CARE MEDICINE

Mechanical Ventilation in ARDS*

A State-of-the-Art Review

Timothy D. Girard, MD; and Gordon R. Bernard, MD, FCCP

- ✓**Low tidal volume strategies (**less than **6 mls/kg predicted body weight**
  - ✓**Maintain plateau below 30 cmH2O**
- ✓**High PEEP**
- ✓**Alveolar recruitment maneuvers**
- ✓**Prone positioning**

Chest. 2007 Mar;131(3):921-929

Volume 148, Issue 2, August 2015, Pages 365-374

CHEST

Original Research **Critical Care**

Mechanical Ventilation and ARDS in the ED: A Multicenter, Observational, Prospective, Cross-sectional Study

Brian M. Fuller MD [a] [b], Nicholas M. Mohr MD [d], Christopher N. Miller MD [e], Andrew R. Deitchman MD [f], Brian J. Levine MD [f], Nicole Castagno MS [g], Elizabeth C. Hassebroek MD [h], Adam Dhedhi BA [i], Nicholas Scott-Wittenborn BA [j], Edward Grace [k], Courtney Lehew MD [a], Marin H. Kollef MD, FCCP [c]

CONCLUSIONS

Lung-protective ventilation is infrequent in patients receiving mechanical ventilation in the ED, regardless of ARDS status. Progression to ARDS is common after admission, occurs early, and worsens outcome. Patient- and treatment-related factors present in the ED are associated with ARDS. Given the limited treatment options for ARDS, and the early onset after admission from the ED, measures to prevent onset and to mitigate severity should be instituted in the ED.

CRITICAL CARE PERSPECTIVE

Fifty Years of Research in ARDS
Spontaneous Breathing during Mechanical Ventilation
Risks, Mechanisms, and Management

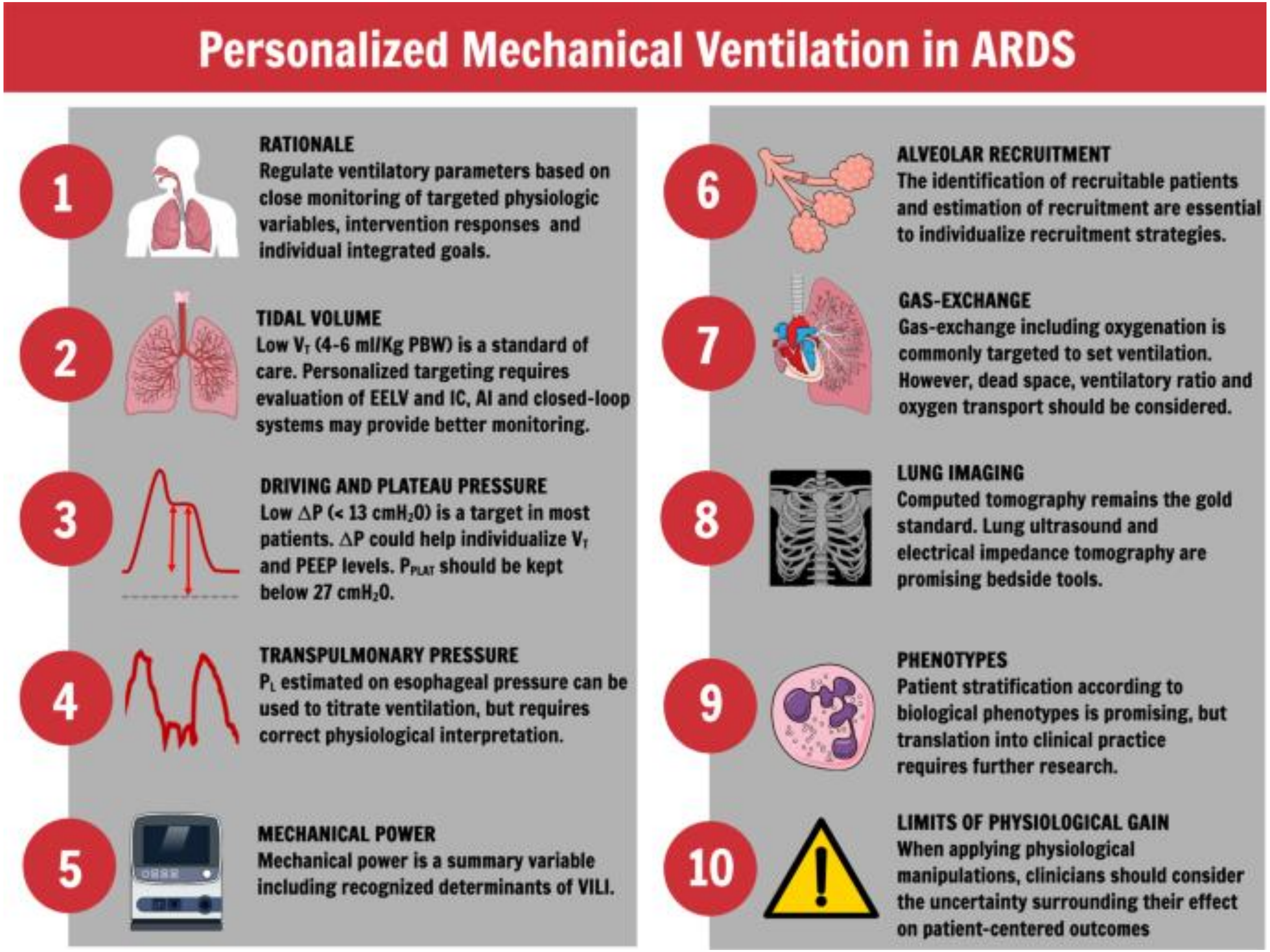
Takeshi Yoshida[1,2,3,4], Yuji Fujino[4], Marcelo B. P. Amato[5], and Brian P. Kavanagh[1,2,3]

[1]Translational Medicine, [2]Department of Critical Care Medicine, and [3]Department of Anesthesia, Hospital for Sick Children, University of Toronto, Toronto, Ontario, Canada; [4]Intensive Care Unit, Osaka University Hospital, Suita, Japan; and [5]Laboratório de Pneumologia LIM-09, Disciplina de Pneumologia, Heart Institute (InCor) Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil

Recommendation:

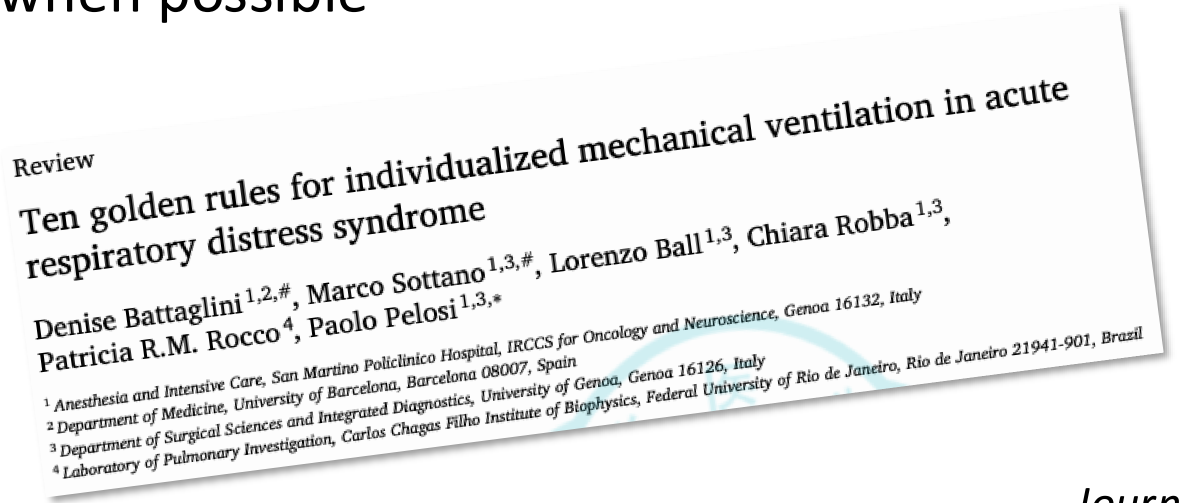
Spontaneous breathing + controlled breathing with (pharmacologically-induced) muscle paralysis

American Journal of Respiratory and Critical Care Medicine (2017), *195*(8), 985-992.



Pelosi et. al. *Critical Care (2021)*, *25*(1), 250

1) **Classify** severity
2) Monitor and **maintain Vt, P_{plat}, and ΔP**
3) Implement/exploit **PEEP**
4) Utilize **recruitment maneuvers**
5) In certain cases, implement **paralyzation**
6) **Decrease MV support** when possible
7) Use **prone positioning**
8) Consider non-traditional **rescue therapies** as available (i.e., artificial lung, iNO)
9) Consider **ECMO**
10) **Wean PPV** when possible


Review

Ten golden rules for individualized mechanical ventilation in acute respiratory distress syndrome

Denise Battaglini[1,2,#], Marco Sottano[1,3,#], Lorenzo Ball[1,3], Chiara Robba[1,3], Patricia R.M. Rocco[4], Paolo Pelosi[1,3,*]

[1] Anesthesia and Intensive Care, San Martino Policlinico Hospital, IRCCS for Oncology and Neuroscience, Genoa 16132, Italy
[2] Department of Medicine, University of Barcelona, Barcelona 08007, Spain
[3] Department of Surgical Sciences and Integrated Diagnostics, University of Genoa, Genoa 16126, Italy
[4] Laboratory of Pulmonary Investigation, Carlos Chagas Filho Institute of Biophysics, Federal University of Rio de Janeiro, Rio de Janeiro 21941-901, Brazil

Journal of Intensive Medicine 1 (2021) 42–51

ARDS-related complications

Barotrauma

Nosocomial infection (VAP/VAE)

Delirium

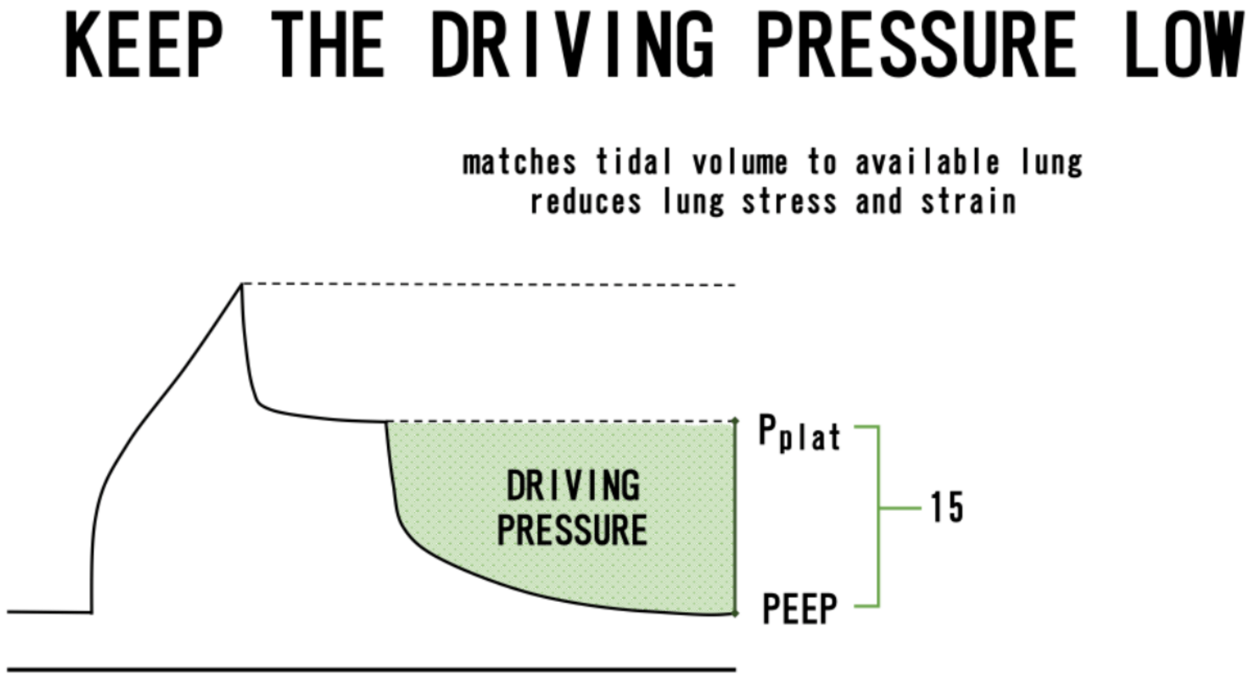
Thrombi

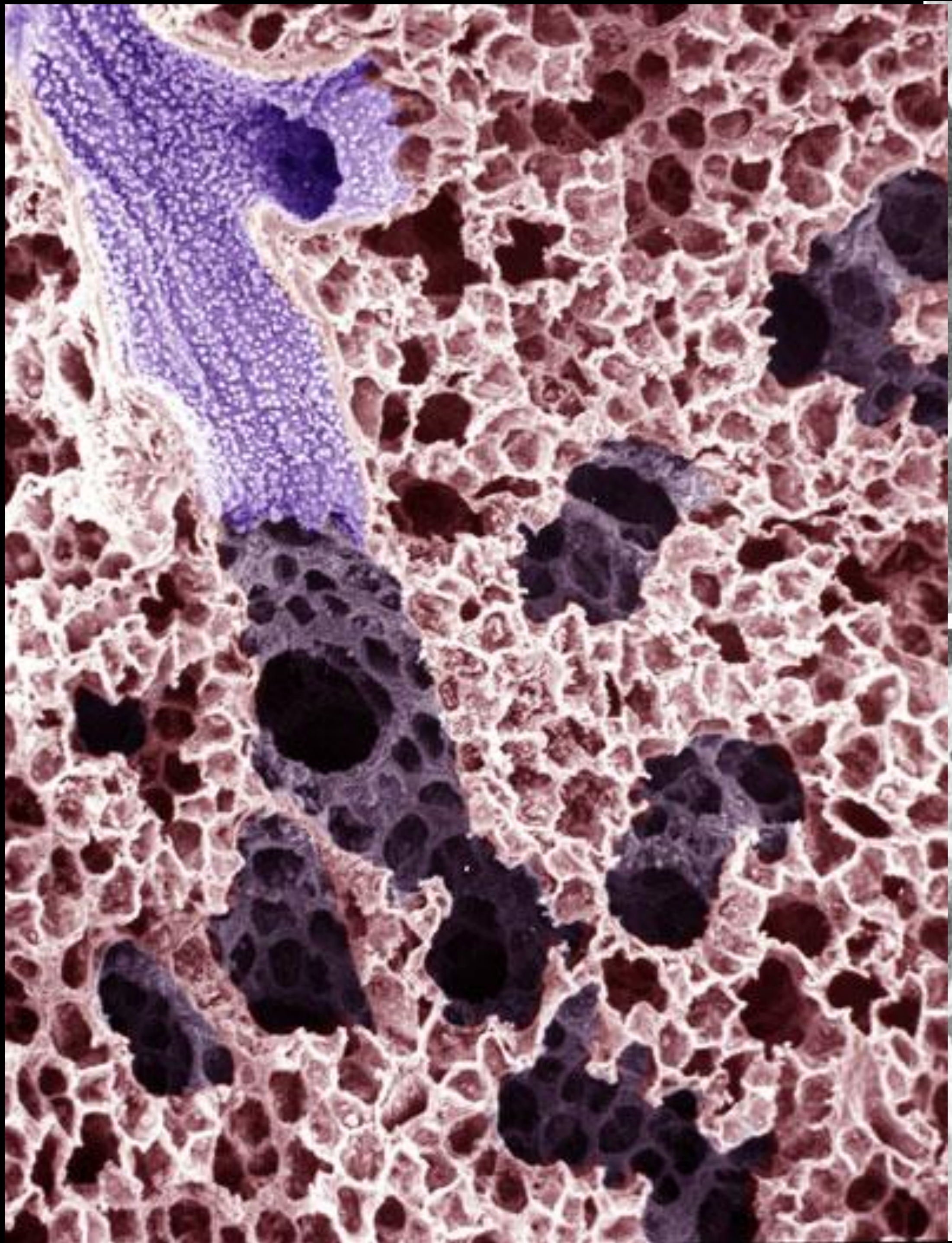
GI bleeding

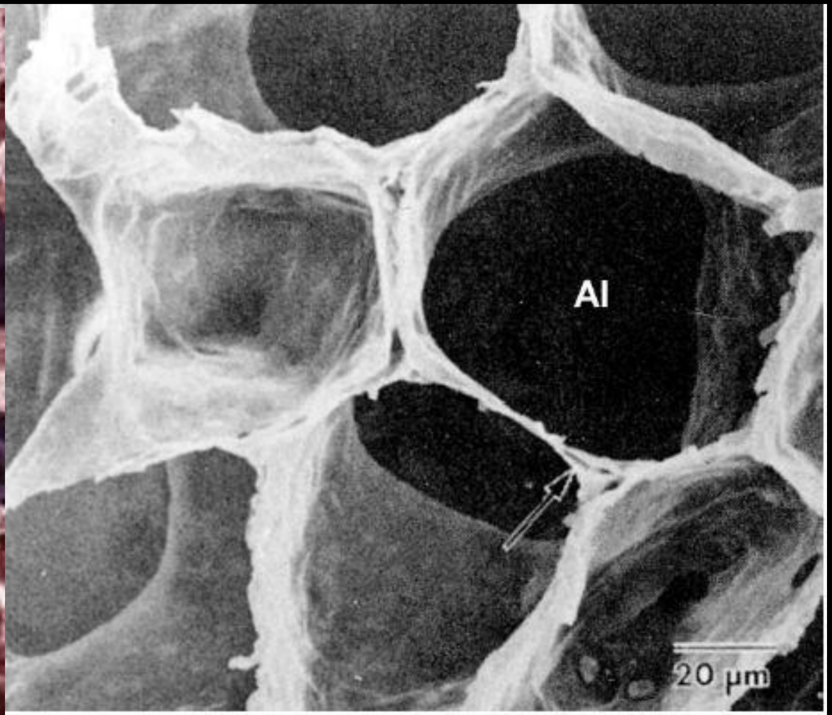
Sleep disturbances

Poor nutrition

Multiorgan failure



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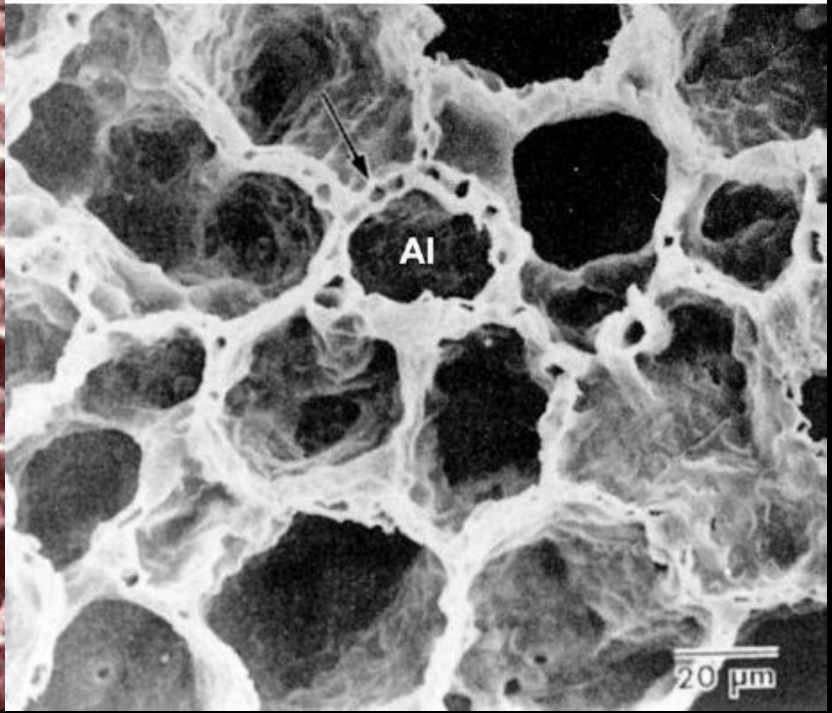
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20 μm

Ventilate This!

Variables of PPV

- ❖ Flow
- ❖ Pressure
- ❖ Volume
- ❖ Time


Flow & Volume

Old Process, New Method

Equation of Motion

$$P_{VENT} + P_{MUSC} = (E \times V) + (R \times V)$$

P_{vent} – pressure generated by ventilator > PEEP
P_{musc} – pressure generated by inspiratory muscles

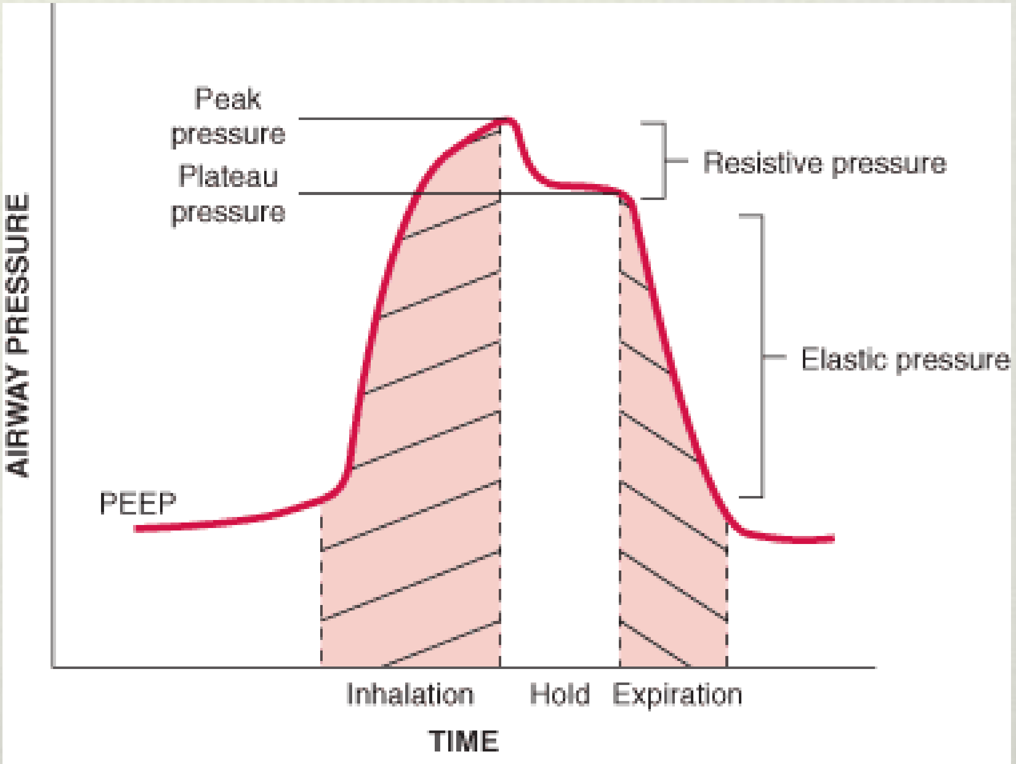
- E – respiratory system elastance
- V – Δ lung volume above FRC

- R – respiratory system resistance
- V - flow

Factors to "Overcome"

- Airways resistance (R_{aw})
- Lung Compliance (C_L)
- Elasticity
- Thoracic Wall

$$V = \Delta P r^4 \pi \ / \ 8 l \eta$$

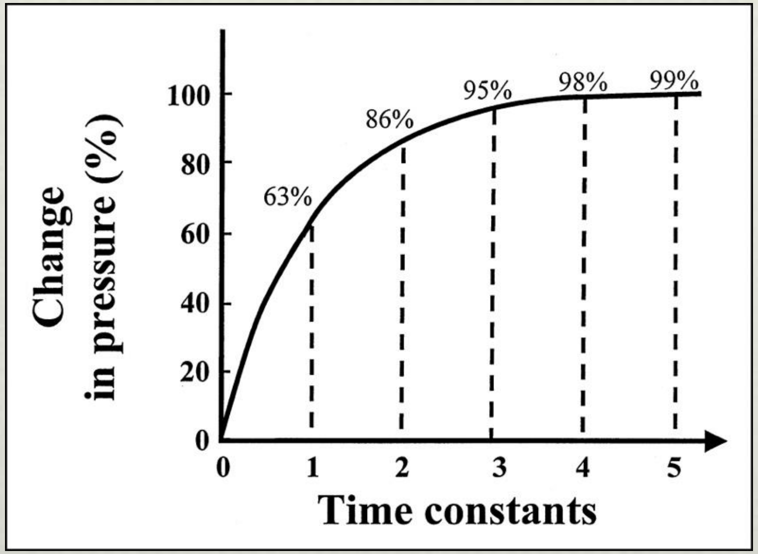


Time Constant (τ)

$1\ TC = C_L \times R_{aw}$

How quickly pressure equilibrates between circuit & alveoli

3-to-5-time constants required for complete inspiration/expiration



What about ventilator mode…

Volume-targeted or Pressure-targeted?

[Original Research **Critical Care**] CHEST

Pressure-Controlled vs Volume-Controlled Ventilation in Acute Respiratory Failure

A Physiology-Based Narrative and Systematic Review

Nuttapol Rittayamai, MD; Christina M. Katsios, MD; François Beloncle, MD; Jan O. Friedrich, MD, PhD; Jordi Mancebo, MD; and Laurent Brochard, MD

CONCLUSIONS: The two modes have different working principles but clinical available data do not suggest any difference in the outcomes. We included all identified trials, enhancing generalizability, and attempted to include only sufficient quality physiologic studies. However, included trials were small and varied considerably in quality. These data should help to open the choice of ventilation of patients with acute respiratory failure. CHEST 2015; 148(2):340-355

2015

Cochrane Library

Trusted evidence.
Informed decisions.
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Cochrane Database of Systematic Reviews

[Intervention Review]

Pressure-controlled versus volume-controlled ventilation for acute respiratory failure due to acute lung injury (ALI) or acute respiratory distress syndrome (ARDS)

Binila Chacko[1], John V Peter[1], Prathap Tharyan[2], George John[1], Lakshmanan Jeyaseelan[3]

Authors' conclusions

Currently available data from RCTs are insufficient to confirm or refute whether pressure-controlled or volume-controlled ventilation offers any advantage for people with acute respiratory failure due to acute lung injury or acute respiratory distress syndrome. More studies including a larger number of people given PCV and VCV may provide reliable evidence on which more firm conclusions can be based.

[Translating Basic Research Into Clinical Practice]

CHEST

Biotrauma and Ventilator-Induced Lung Injury

Clinical Implications

CrossMark

Gerard F. Curley, MB, PhD; John G. Laffey, MD; Haibo Zhang, MD, PhD; and Arthur S. Slutsky, MD

Summary:

Ventilator = Bad

Suggestions for avoiding VILI:

Lung protective strategies (higher PEEP, lower Vt),

Prone positioning, NMBAs, ECMO

2015

Gayat *et al. Critical Care* (2018) 22:8
DOI 10.1186/s13054-017-1922-8

Critical Care

RESEARCH Open Access

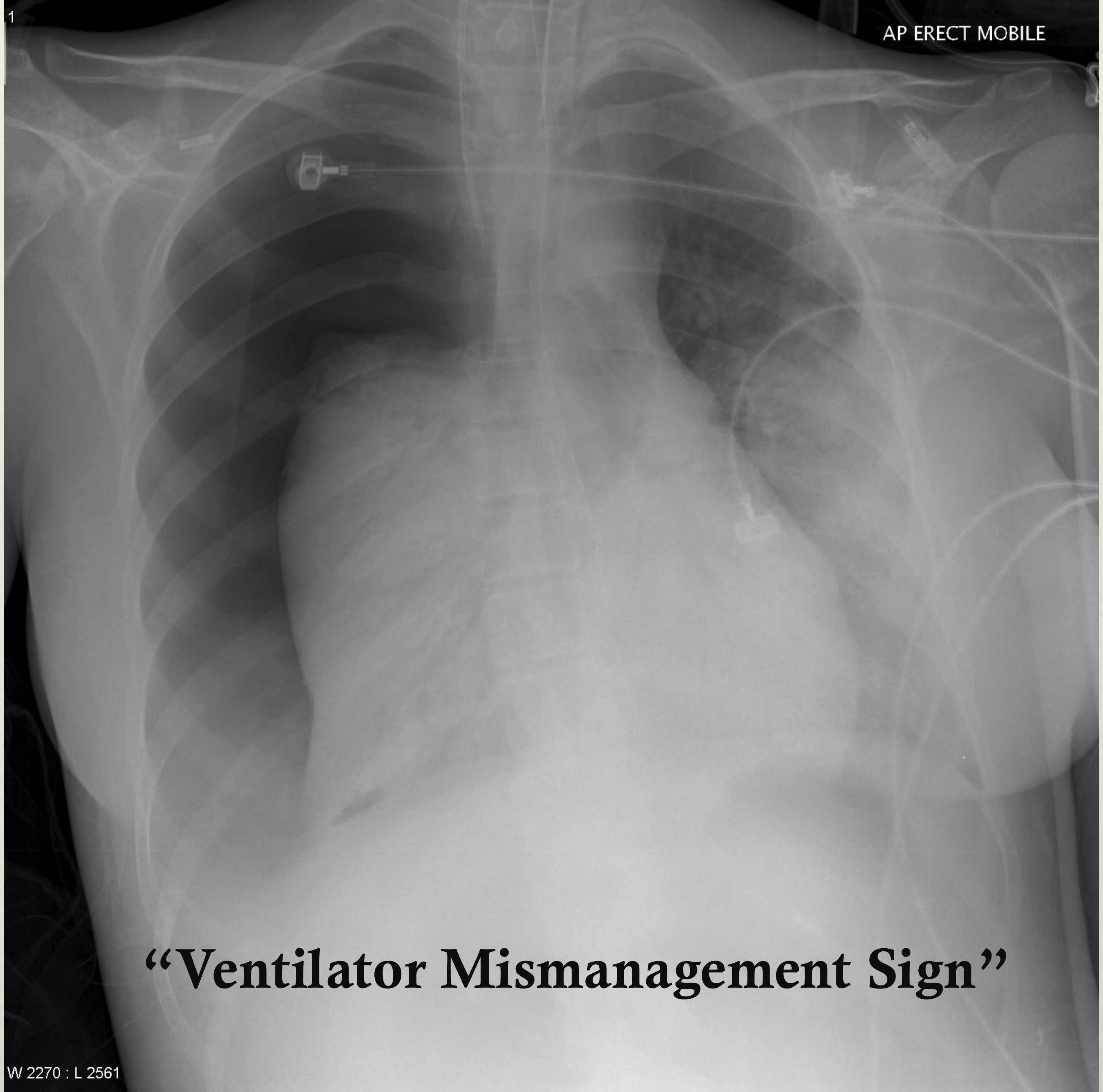
Determinants of long-term outcome in ICU survivors: results from the FROG-ICU study

Etienne Gayat[1,28*], Alain Cariou[2], Nicolas Deye[3], Antoine Vieillard-Baron[4], Samir Jaber[5], Charles Damoisel[1], Qin Lu[6], Xavier Monnet[7], Isabelle Rennuit[8], Elie Azoulay[9], Marc Léone[10], Heikel Oueslati[1], Bertrand Guidet[11], Diane Friedman[12], Antoine Tesnière[13], Romain Sonneville[14], Philippe Montravers[15], Sébastien Pili-Floury[16], Jean-Yves Lefrant[17,18], Jacques Duranteau[19], Pierre-François Laterre[20], Nicolas Brechot[21], Karine Chevreul[22], Morgane Michel[23], Bernard Cholley[24], Matthieu Legrand[1], Jean-Marie Launay[24], Eric Vicaut[25], Mervyn Singer[26], Matthieu Resche-Rigon[27†] and Alexandre Mebazaa[1†]

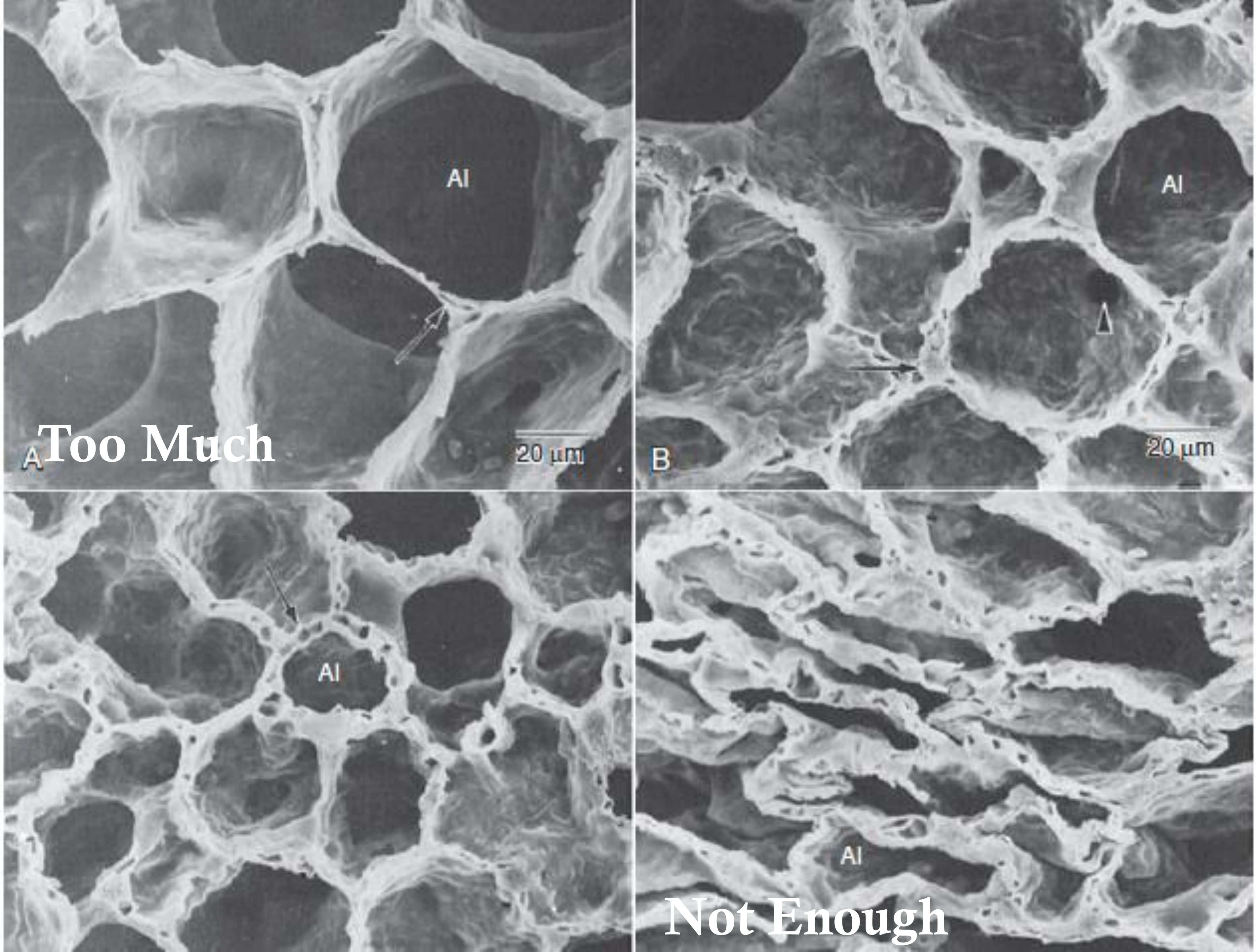
Results: Of 1570 patients discharged alive from the ICU, 333 (21%) died over the following year. Multivariable analysis identified age, comorbidity, red blood cell transfusion, ICU length of stay and abnormalities in common clinical factors at the time of ICU discharge (low systolic blood pressure, temperature, total protein, platelet and white cell count) as independent factors associated with 1-year mortality. Elevated biomarkers of cardiac and vascular failure independently associated with 1-year death when they are added to multivariable model, with an almost 3-fold increase in the risk of death when combined (adjusted odds ratio 2.84 (95% confidence interval 1.73–4.65), $p < 0.001$).

Conclusions: The FROG-ICU study identified, at the time of ICU discharge, potentially actionable clinical and biological factors associated with poor long-term outcome after ICU discharge. Those factors may guide discharge planning and directed interventions.

Trial registration: ClinicalTrials.gov NCT01367093. Registered on 6 June 2011.

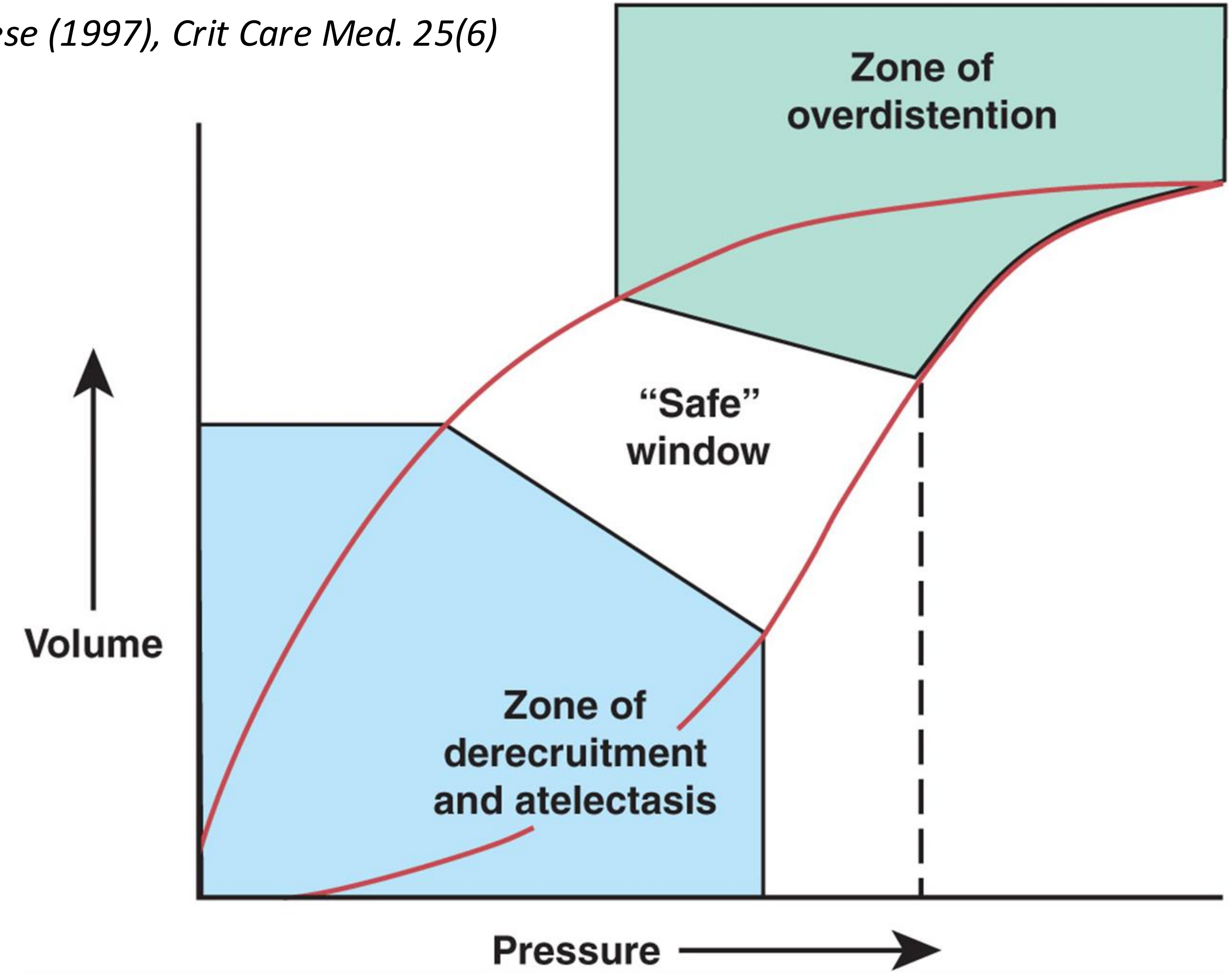


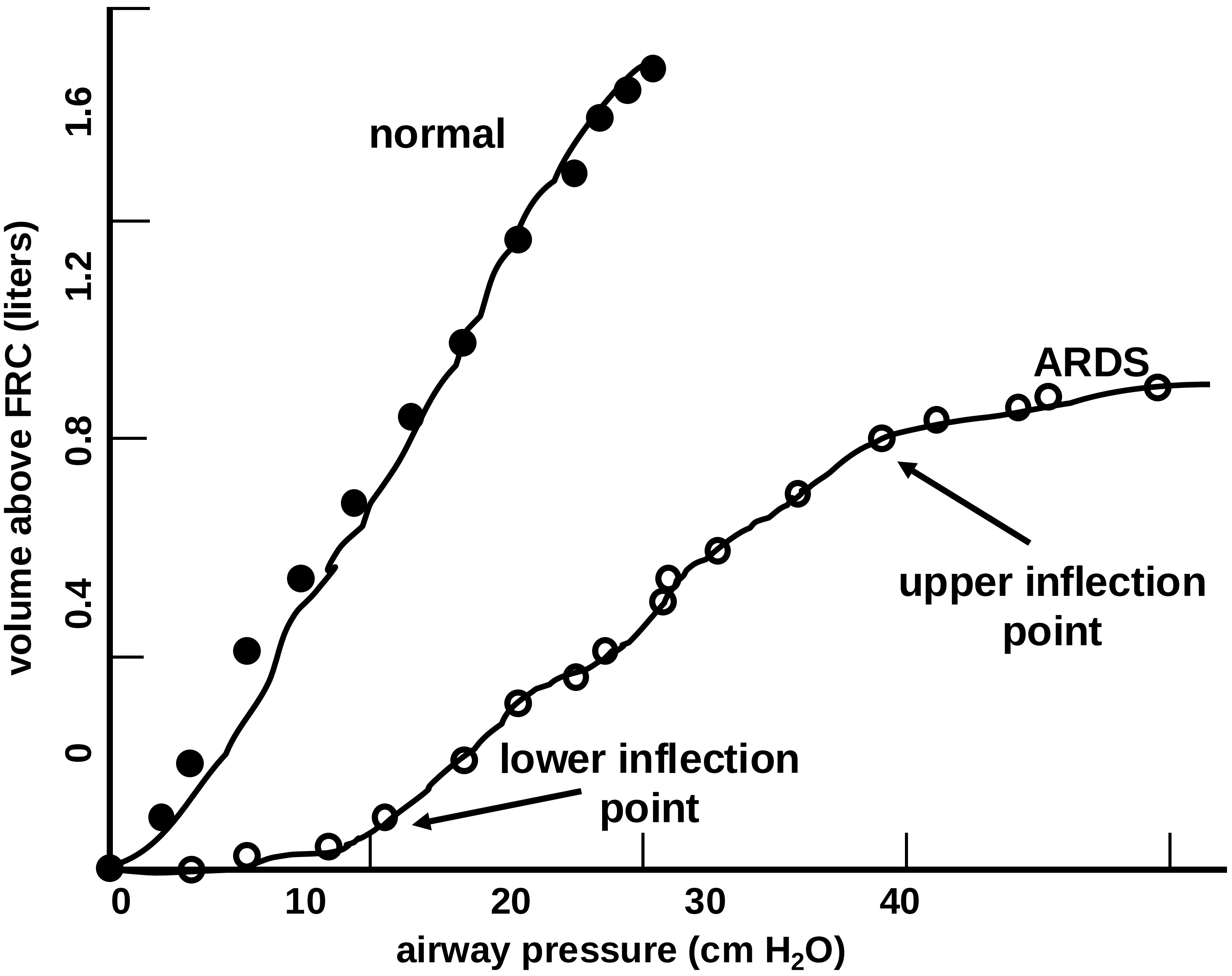
“Ventilator Mismanagement Sign”



Too Much
A
Al
20 µm
B
Al
20 µm
Al
Al
Not Enough

Froese (1997), Crit Care Med. 25(6)





volume above FRC (liters)
1.6
1.2
0.8
0.4
0
normal
ARDS
upper inflection point
lower inflection point
0
10
20
30
40
airway pressure (cm H_2O)

Generally speaking,…

- End-inspiratory **plateau pressure** and **Vt/IBW** ratio are inadequate surrogates to qualify and/or quantify end-inspiratory stress and strain[1]
- Large static strains are less harmful than large dynamic strains[2]
- An optimal "protective breath" describes one of minimal dynamic strain and energy/power load[3]
- Lung protection > diaphragm protection[4]

1.Gattinoni et. al. *Current Opinion in Critical Care* 18 (1), 2012.
2. Protti et. al. *Critical Care Medicine* 41 (4), 2013.
3. Nieman, et. al. *Intensive Care Medicine Experimental* 4 (16), 2016.
4. Goligher et. al. AJRCCM 202 (7). 2020

Summary

- **Lung protective** ventilation is a must
  - Lower tidal volumes
- Application of optimal (and *adequate)* **PEEP** may require higher than traditional settings
- Target **low** plateau **pressure** (and low driving pressure)
- Use **recruitment maneuvers** cautiously
- Consider **prone** positioning
- Seek to maintain **ventilator synchrony** – may require neuromuscular blockade

***Don’t disregard potential **non-traditional intervention** as an effective option: NIV, HFOV, APRV, ECMO, etc.

Journal of clinical medicine (2022), *11*(2), 319

Thanks for Listening!

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