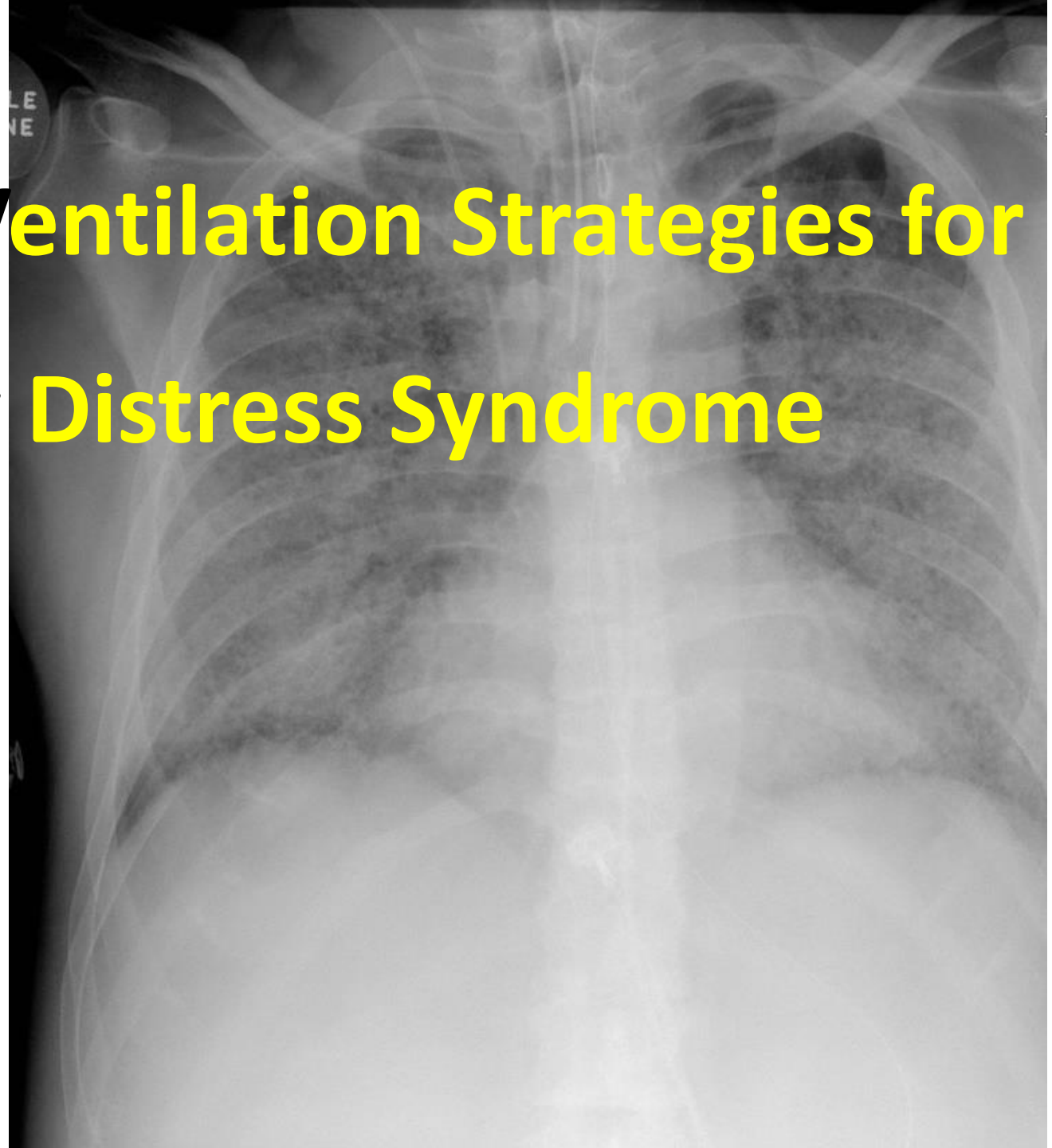


Updated Mechanical Ventilation Strategies for Acute Respiratory Distress Syndrome

臺北榮民總醫院

胸腔部 呼吸治療科

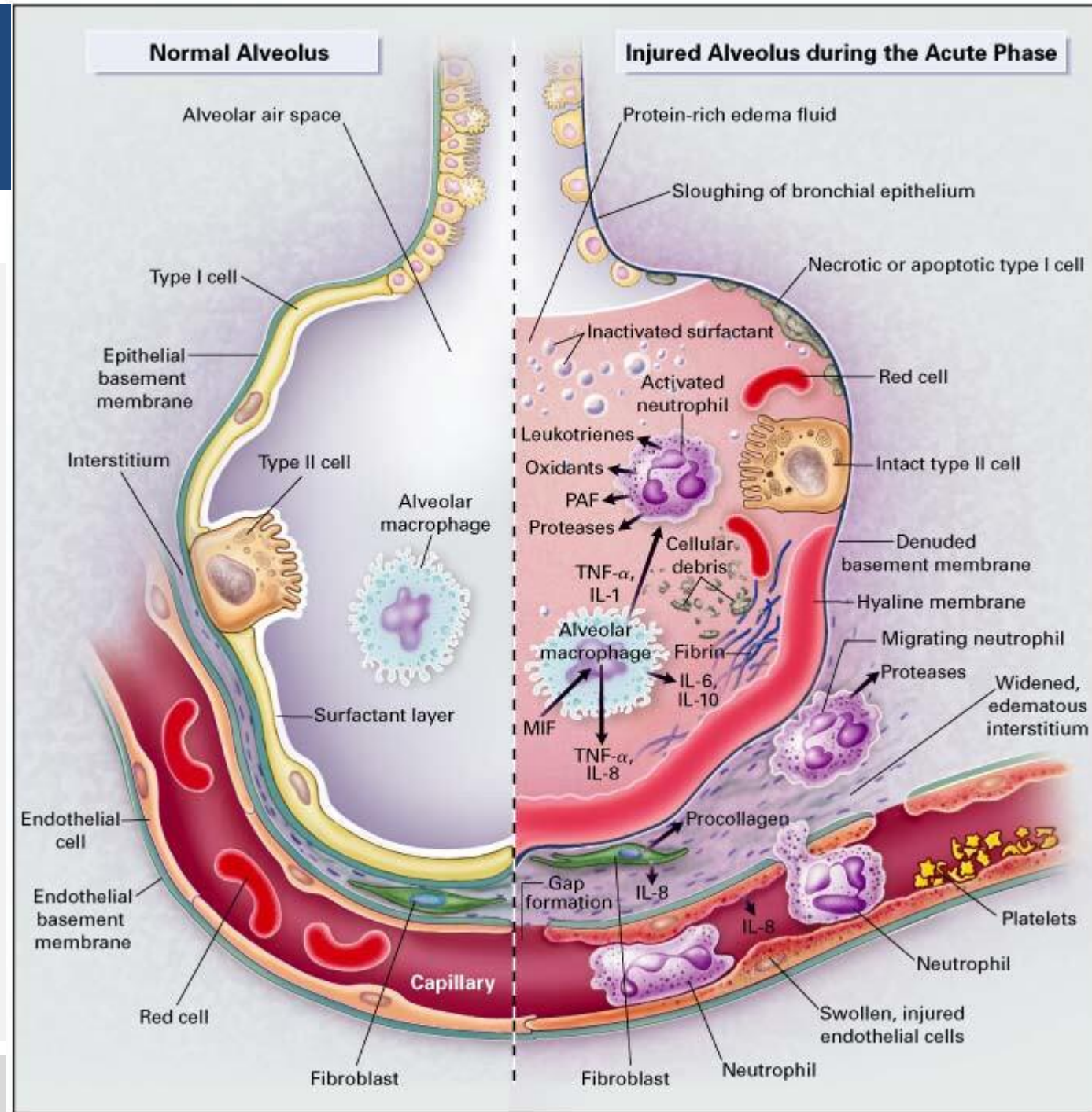
余文光醫師



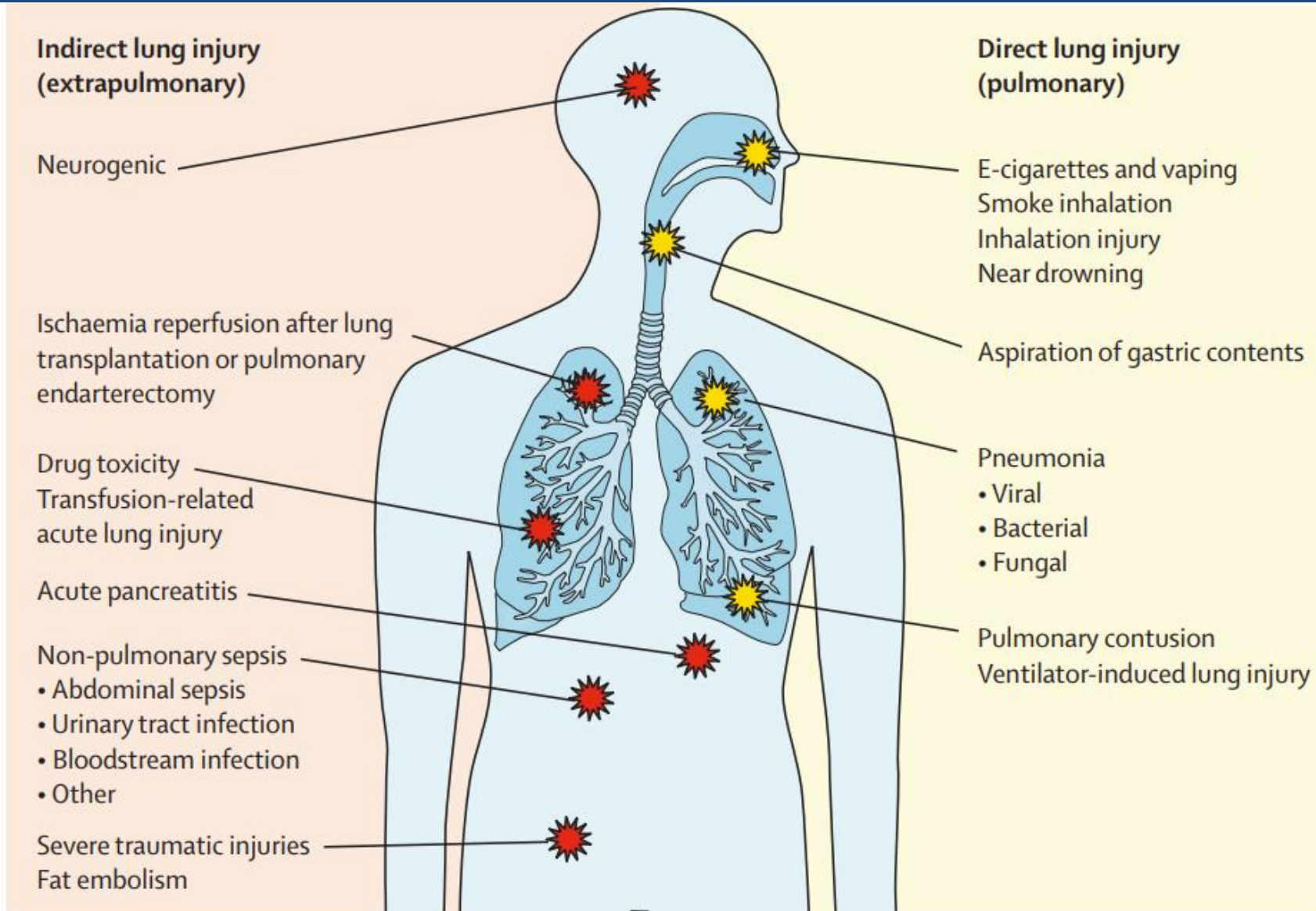
ARDS: Definition

An acute, severe, and diffuse inflammatory injury of the lung parenchyma caused by predisposing risk factors, leading to clinically significant arterial hypoxemia that necessitates oxygen supplementation via invasive, non-invasive mechanical ventilation or high-flow nasal oxygen.

N Engl J Med. 2000;342:1330-1349.



ARDS Causes

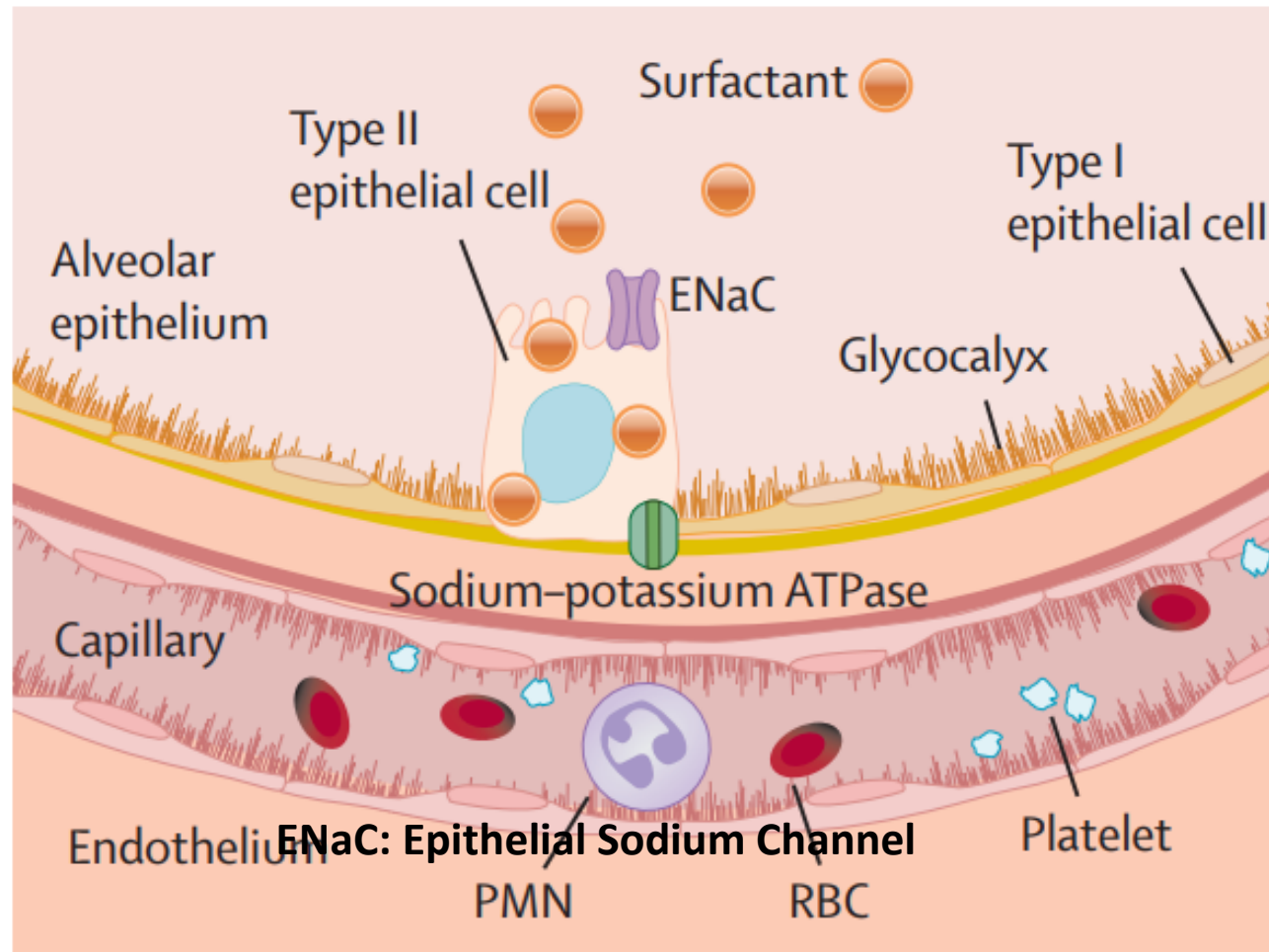


Pathophysiology: Pulmonary Epithelial and Endothelial Injury

Normal alveolar-capillary barrier

Normal epithelium

- Airspace is dry
- Intact epithelial tight junctions
- Intact epithelial glycocalyx
- Normal surfactant production
- Vectorial sodium and chlorine transport



Normal endothelium

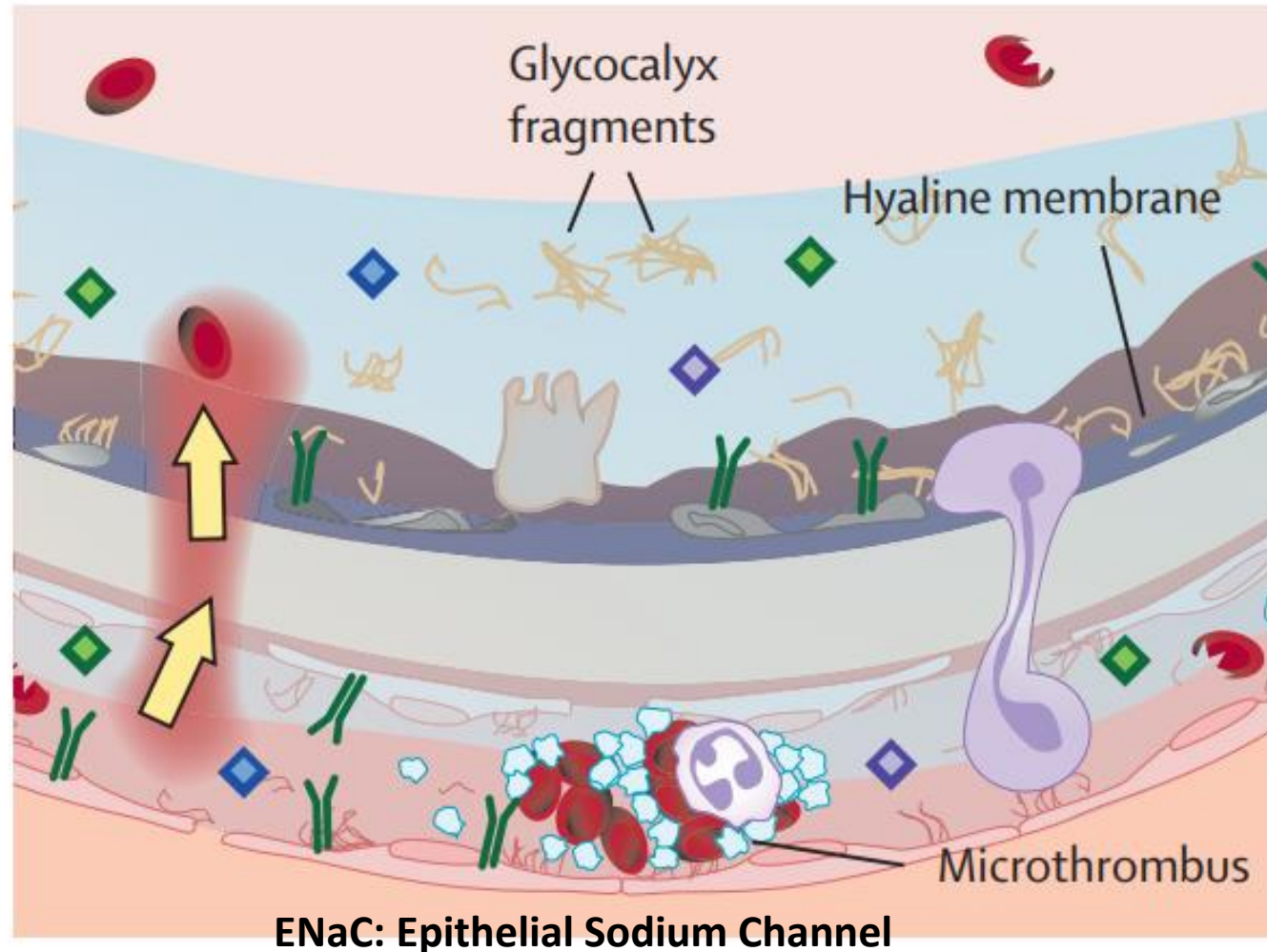
- Intact endothelial tight junctions
- Intact endothelial glycocalyx
- No adhesion molecules displayed
- White blood cells including neutrophils (ie, PMNs), RBCs, and platelets transit the capillary freely

Pathophysiology: Pulmonary Epithelial and Endothelial Injury

Severe injury

Epithelial injury

- Severe oedema formation
- Severe disruption of tight junctions
- Epithelial necrosis
- Hyaline membrane formation
- Absent sodium and chlorine transport
- Glycocalyx shedding
- Increased chemokines and adhesion molecules
- RBCs in airspace



Endothelial injury

- More severe endothelial disruption with transit of fluid out of the capillary
- Loss of endothelial glycocalyx
- RBC injury
- PMN transmigration
- Platelet microthrombi

Mechanisms and Biomarkers of ARDS

Inflammasome activation

IL-18, IL-1beta, and Ferritin

IL-1 inhibitors
NLRP3 inhibitors
Disulfiram

Epithelial injury

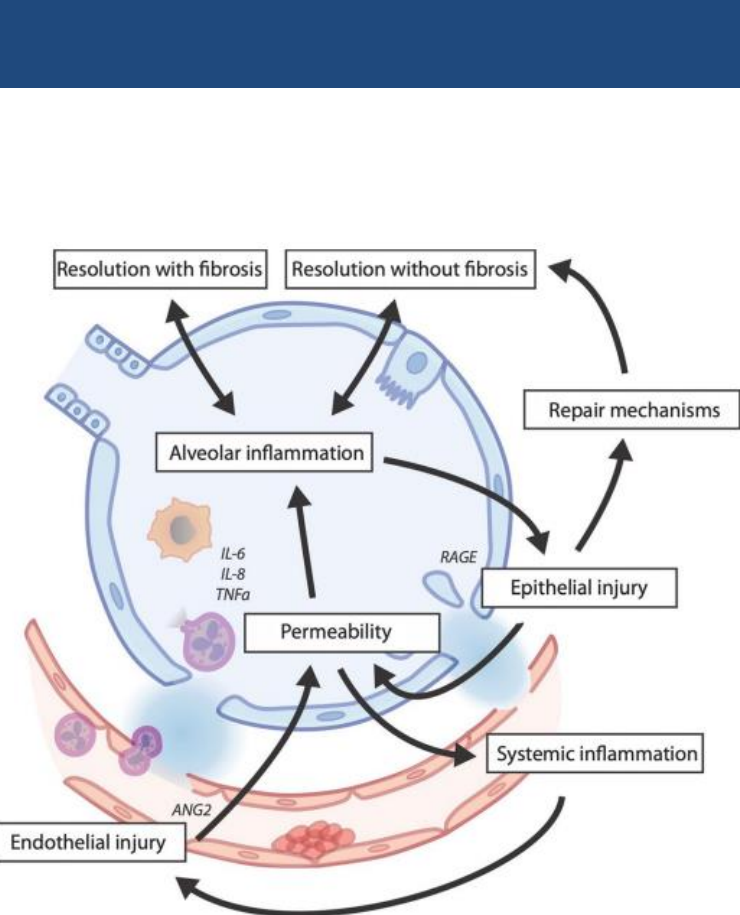
sRAGE and SP-D

Solnatide

Inflammation

IL-6 and sTNR1

Corticosteroids
Mesenchymal stromal cells
Simvastatin
IL-6 inhibitors



Inflammatory pulmonary oedema

Epithelial damage

Platelet activation

Inflammasome activation



Mitochondrial dysfunction

Mitochondrial DNA
Mesenchymal stromal cells

Mitochondrial dysfunction and reactive oxygen species



Cytokine release

Activated neutrophil

Migrating neutrophil

Cellular debris

Neutrophil recruitment

BALF neutrophils
Neutrophil extracellular traps formation

Cathepsin S inhibitor
Ulinastatin

Inflamed endothelial cells

Gap formation

Endothelial injury

Ang-2
Recombinant ACE2
Citrulline

Microthrombi formation

ARDS: Definition

Conceptual model: ARDS is an acute, diffuse, inflammatory lung injury precipitated by a predisposing risk factor, such as pneumonia, nonpulmonary infection, trauma, transfusion, burn, aspiration, or shock. The resulting injury leads to increased pulmonary vascular and epithelial permeability, lung edema, and gravity-dependent atelectasis, all of which contribute to loss of aerated lung tissue. The clinical hallmarks are arterial hypoxemia and diffuse radiographic opacities associated with increased shunting, increased alveolar dead space, and decreased lung compliance. The clinical presentation is influenced by medical management (position, sedation, paralysis, positive end-expiratory airway pressure, and fluid balance). Histological findings vary and may include intraalveolar edema, inflammation, hyaline membrane formation, and alveolar hemorrhage.

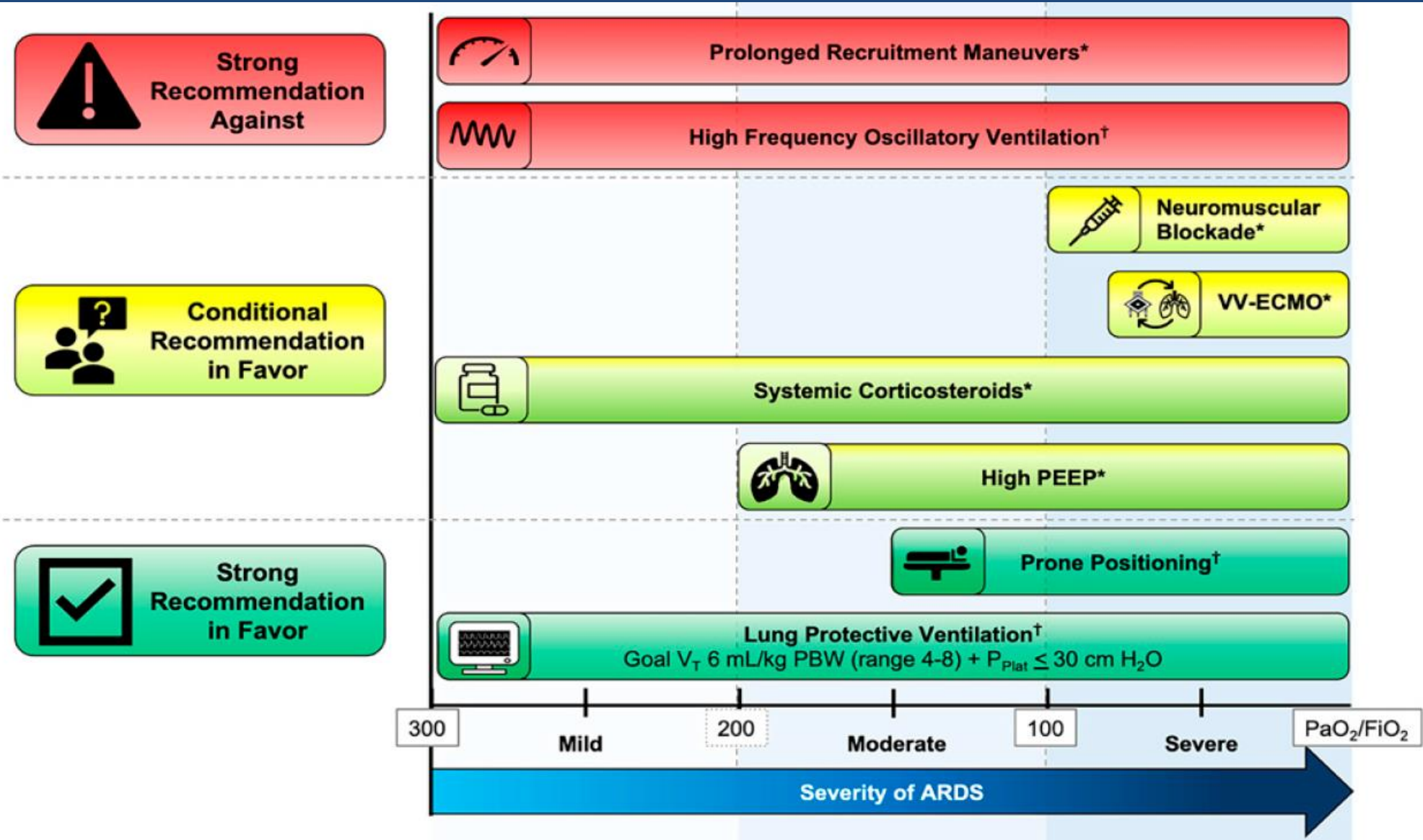
Criteria That Apply to All ARDS Categories

Risk factors and origin of edema	Precipitated by an acute predisposing risk factor, such as pneumonia, nonpulmonary infection, trauma, transfusion, aspiration, or shock. Pulmonary edema is not <i>exclusively or primarily</i> attributable to cardiogenic pulmonary edema/fluid overload, and hypoxemia/gas exchange abnormalities are not primarily attributable to atelectasis. However, ARDS can be diagnosed in the presence of these conditions if a predisposing risk factor for ARDS is also present.
Timing	Acute onset or worsening of <u>hypoxemic respiratory failure within 1 week</u> of the estimated onset of the predisposing risk factor or new or worsening respiratory symptoms.
Chest imaging	<u>Bilateral opacities</u> on chest radiography and computed tomography or bilateral B lines and/or consolidations on ultrasound* not fully explained by effusions, atelectasis, or nodules/masses.

Criteria That Apply to Specific ARDS Categories

	Nonintubated ARDS [†]	Intubated ARDS	Modified Definition for Resource-Limited Settings [‡]
Oxygenation [§]	$\text{PaO}_2:\text{FiO}_2 \leq 300 \text{ mm Hg}$ or $\text{SpO}_2:\text{FiO}_2 \leq 315$ (if $\text{SpO}_2 \leq 97\%$) on HFNO with flow of $\geq 30 \text{ L/min}$ or NIV/CPAP with at least 5 cm H_2O end-expiratory pressure	Mild [¶] : $200 < \text{PaO}_2:\text{FiO}_2 \leq 300 \text{ mm Hg}$ or $235 < \text{SpO}_2:\text{FiO}_2 \leq 315$ (if $\text{SpO}_2 \leq 97\%$) Moderate: $100 < \text{PaO}_2:\text{FiO}_2 \leq 200 \text{ mm Hg}$ or $148 < \text{SpO}_2:\text{FiO}_2 \leq 235$ (if $\text{SpO}_2 \leq 97\%$) Severe: $\text{PaO}_2:\text{FiO}_2 \leq 100 \text{ mm Hg}$ or $\text{SpO}_2:\text{FiO}_2 \leq 148$ (if $\text{SpO}_2 \leq 97\%$)	$\text{SpO}_2:\text{FiO}_2 \leq 315$ (if $\text{SpO}_2 \leq 97\%$) [†] . Neither positive end-expiratory pressure nor a minimum flow rate of oxygen is required for diagnosis in resource-limited settings.

Guideline Recommendations for ARDS Management



Treatment of ARDS

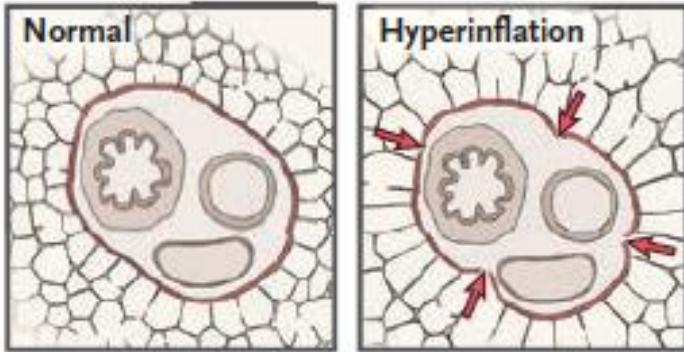
Therapies	Novel Trial Findings
Lung protective ventilation (LPV)	ARMA: Lower mortality with LPV
Open lung ventilation	ALVEOLI: No difference in hospital mortality ExPress: No difference in 28-day mortality LOVS: No difference in 28-day hospital mortality ART: Higher 28-day mortality with open lung ventilation STAMINA: No difference in 90-day in hospital or ICU mortality
High-frequency oscillatory ventilation (HFOV)	OSCILLATE: Higher hospital mortality with HFOV OSCAR: No difference in 30-day mortality
Prone position	PROSEVA: Lower 28-day mortality with prone position ECMOSARS: May be beneficial in patients support by V-V ECMO PRONECMO: No difference in 90-day in hospital or ICU mortality
Neuromuscular blocking agents (NMBA)	ACURASYS: Lower adjusted 90-day mortality with NMBA ROSE: No difference in 90-day mortality
Fluid therapy	FACTT: No difference in mortality; more ventilator free days with conservative fluid strategy
Statins	HARP-2: No difference in 28-day mortality with simvastatin SAILS: No difference in 60-day or hospital mortality with rosuvastatin
Image-guide ventilation	LIVE: Personalisation of mechanical ventilation did not decrease mortality
Extracorporeal membrane oxygenation (ECMO)	CESAR: Lower 6-months mortality with ECMO EOLIA: No difference in 60-day mortality

Treatment of ARDS

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Pathophysiology: Ventilator-Induced Lung Injury

Ventilation at high lung volume

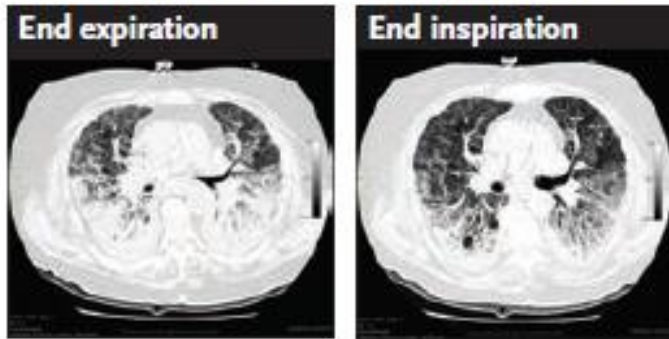


Air leaks

Overdistention



Ventilation at low lung volume



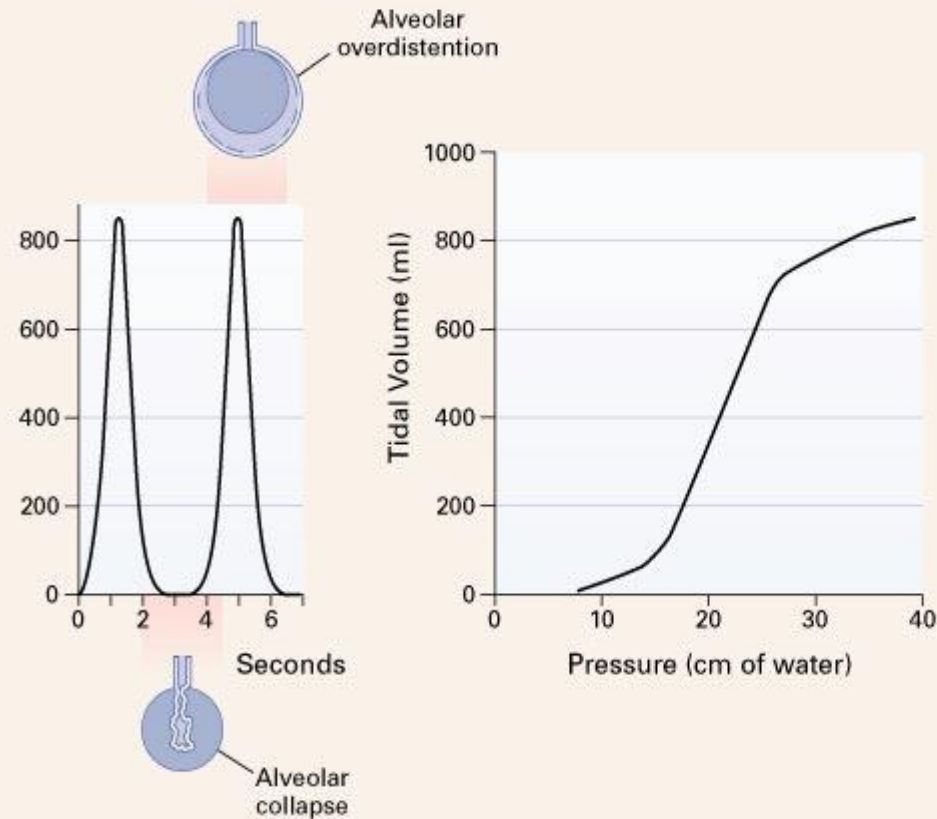
Atelectrauma



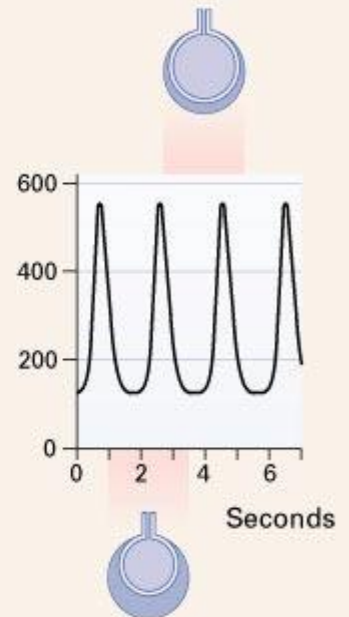
Lung inhomogeneity

Driving Pressure Positive End Expiratory Pressure (PEEP)

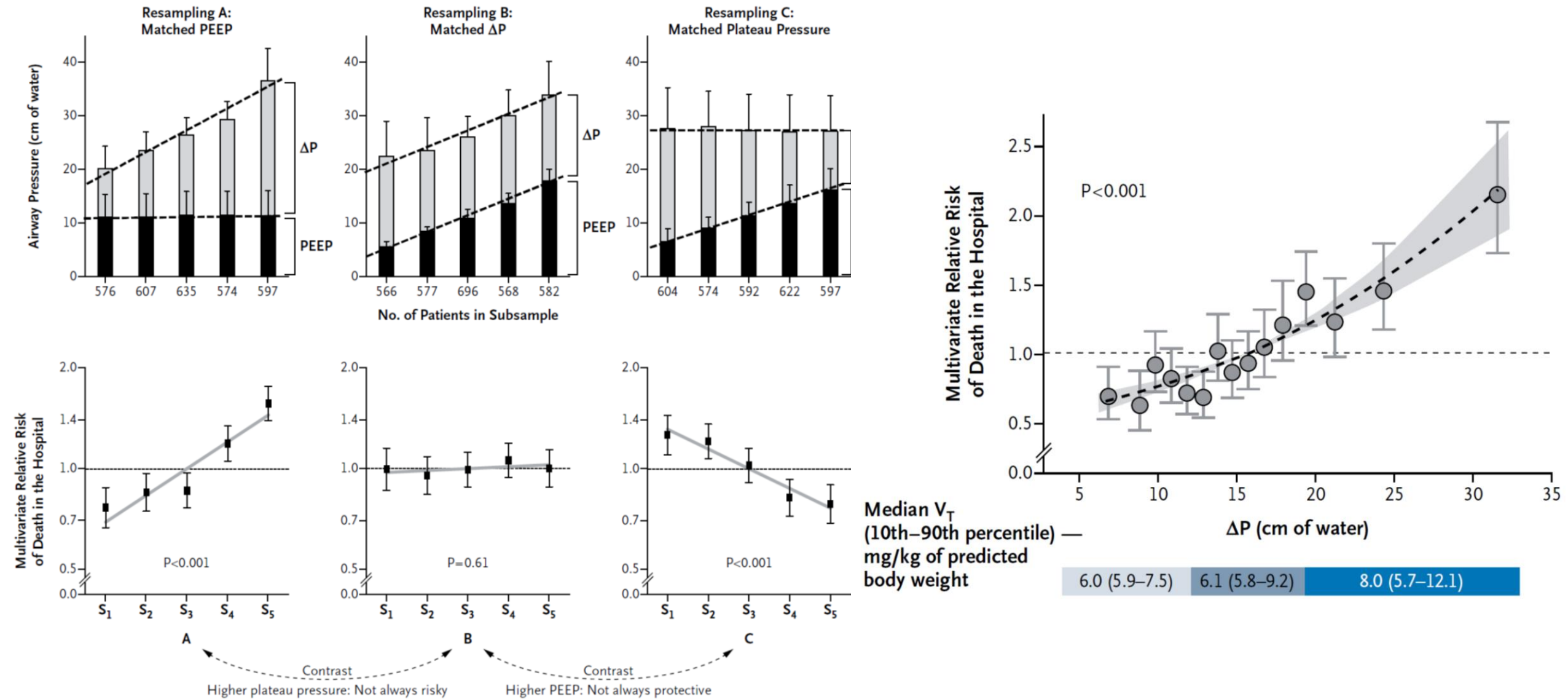
Conventional Ventilation



Protective Ventilation

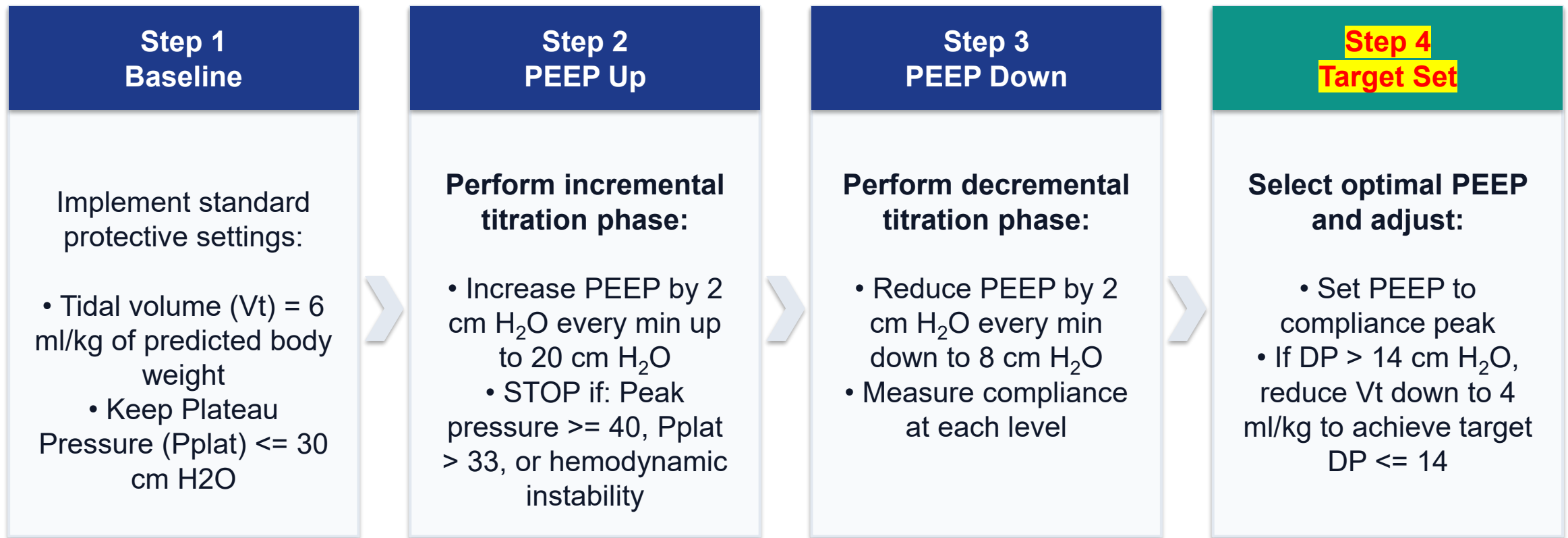


ARDS Management: Driving Pressure



ARDS Management: Driving Pressure

Effect of a Driving Pressure-Limiting Strategy for Patients with Acute Respiratory Distress Syndrome Secondary to Community-Acquired Pneumonia: the **STAMINA** Randomised Clinical Trial



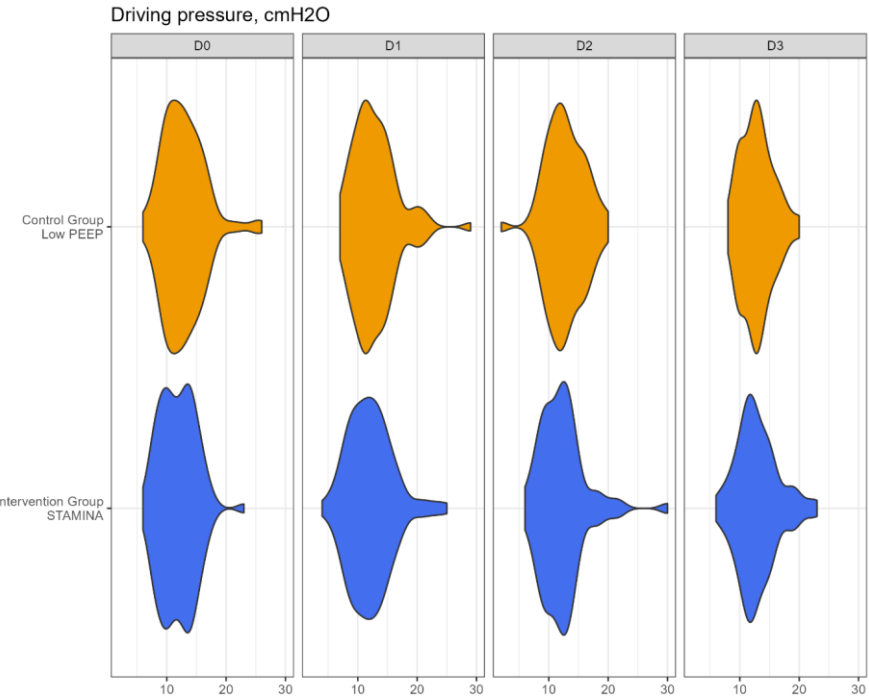
ARDS Management: Driving Pressure

P = 0.28

No statistical difference in primary outcome (proportional OR = 0.72, 95% CI 0.39-1.32).

-0.7 cm H2O

Mean difference in driving pressure between groups up to day 3. Visually narrow and clinically insignificant.



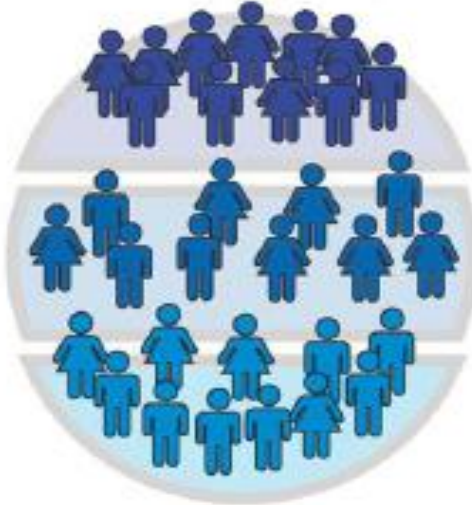
Outcome Metric	STAMINA (n=96)	Control (n=102)	Effect (95% CI) / P-val
Ventilator-Free Days, Mean (SD)	6 (9) days	7 (9) days	MD -1.28 (-3.6 to 1.26)
Ventilator-Free Days, Median	0 (0-15) days	0 (0-17) days	ORp 0.72 (0.39-1.32)
ICU Mortality, n (%)	63/96 (66%)	54/102 (53%)	OR 1.66 (0.87-3.17); P=0.13
Hospital Mortality, n (%)	63/96 (66%)	56/102 (55%)	OR 1.48 (0.77-2.85); P=0.24
Barotrauma (Safety), n (%)	4/96 (4.2%)	3/102 (2.9%)	OR 1.35 (0.28-6.55); P=0.71
ICU Length of Stay, Mean (SD)	17 (16) days	17 (15) days	MD -0.71 (-4.11 to 2.69)

ARDS Heterogeneity

Unselected ARDS



Berlin severity



Pulmonary / non-pulmonary



Focal / non-Focal



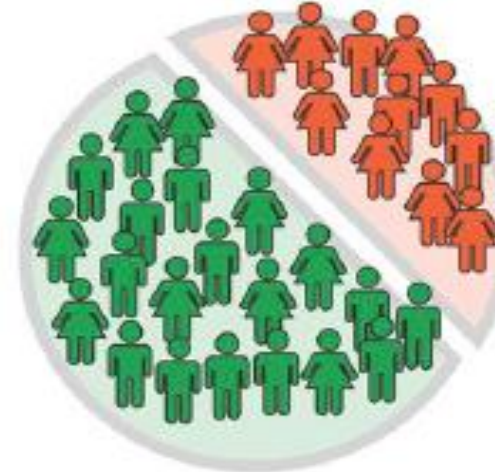
Endothelial dysfunction



Epithelial injury



Systemic host response



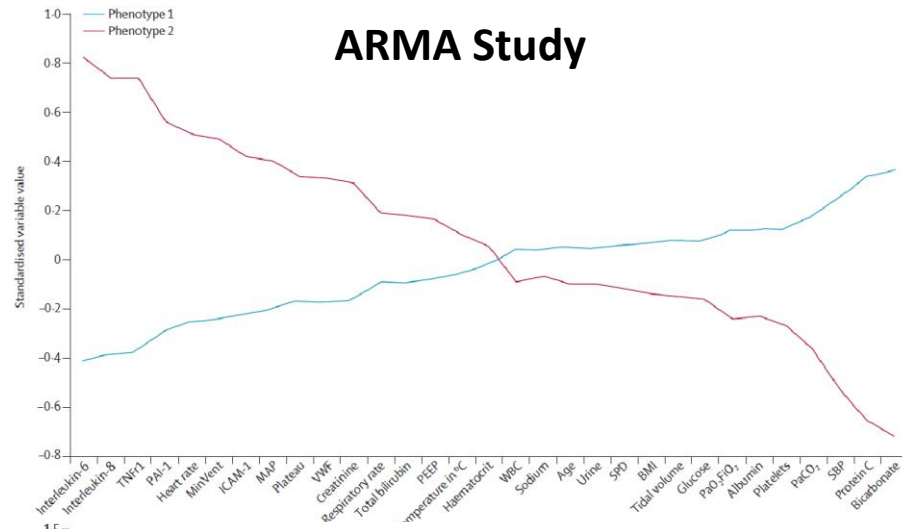
Alveolar host response



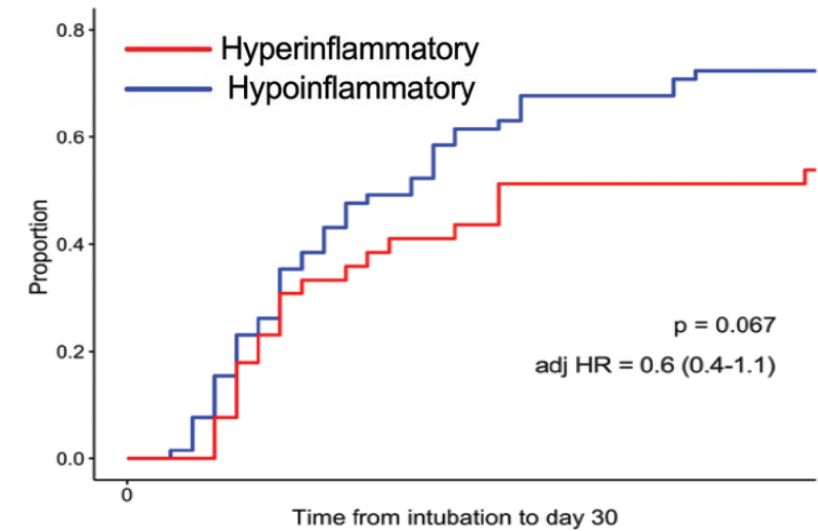
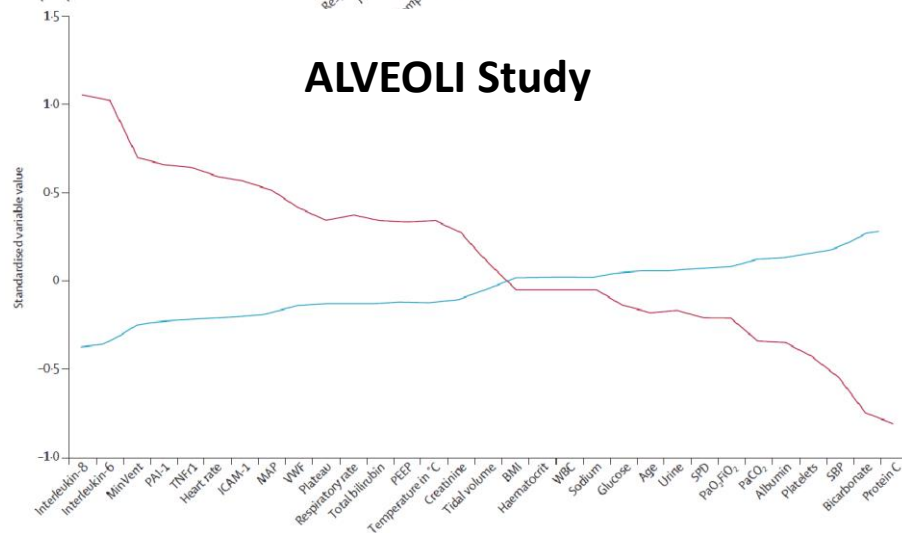
Hypoinflammatory/Hyperinflammatory Phenotypes

Latent Class Analysis of ARDS

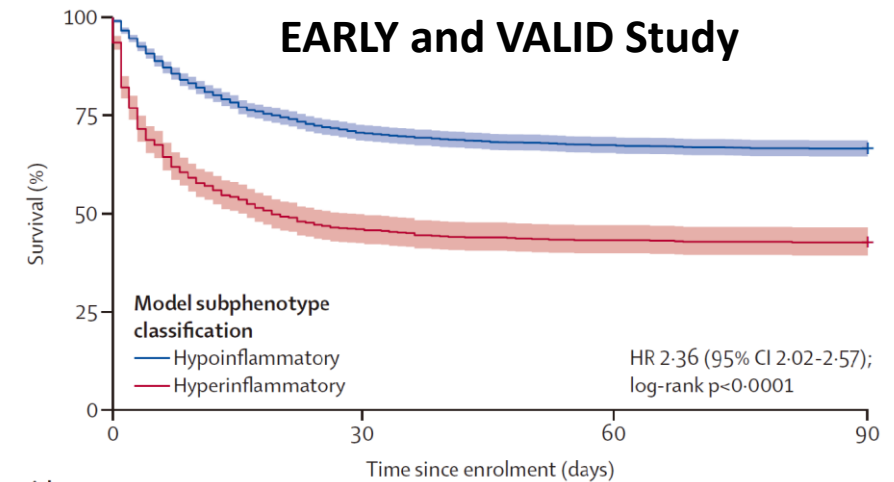
ARMA Study



ALVEOLI Study



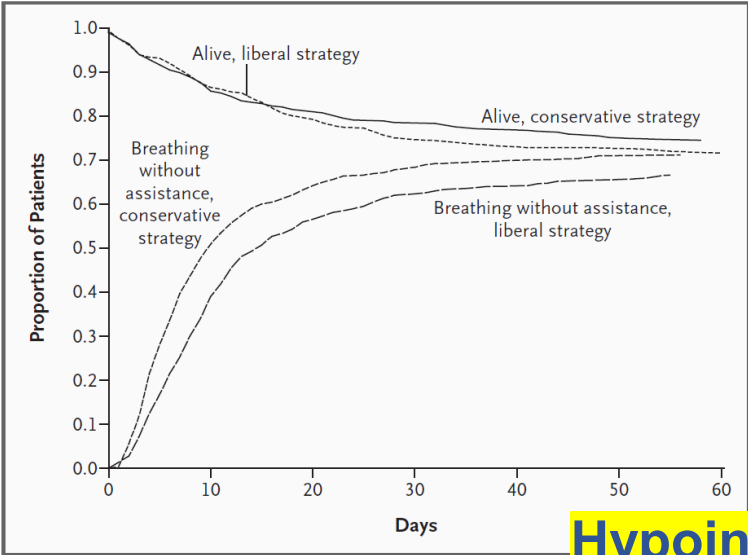
EARLY and VALID Study



Hypoinflammatory/Hyperinflammatory Phenotypes

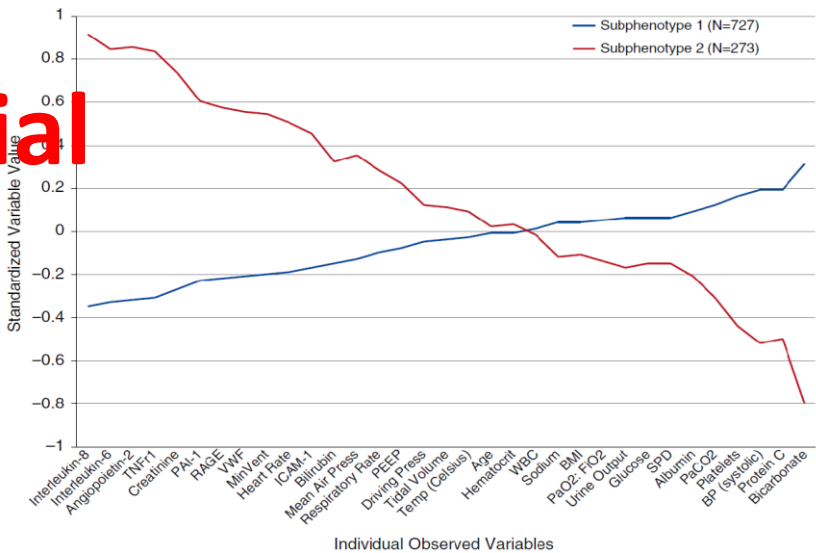
ORIGINAL ARTICLE

Comparison of Two Fluid-Management Strategies in Acute Lung Injury



Hypoinflammatory

Hyperinflammatory



	Subphenotype 1 (n = 727)	Subphenotype 2 (n = 273)	P Value
60-d mortality, %	21	44	<0.0001
90-d mortality, %	22	45	<0.0001
Ventilator-free days, median	19	3	<0.0001

Fluid-management strategy	Subphenotype 1		Subphenotype 2		P Value
	Liberal (n = 355)	Conservative (n = 372)	Liberal (n = 142)	Conservative (n = 131)	
60-d mortality, %	24	17	39	49	0.0093
90-d mortality, %	26	18	40	50	0.0039
Ventilator-free days, median	17	21	5	0	0.35

Hypoinflammatory/Hyperinflammatory Phenotypes

PEEP in ARDS

ARMA and ALVEOLI Study

	Phenotype 1 (n=404)		Phenotype 2 (n=145)		p value*
	Low PEEP (n=202)	High PEEP (n=202)	Low PEEP (n=71)	High PEEP (n=74)	
Mortality at 90 days	33 (16%)	48 (24%)	36 (51%)	31 (42%)	0.049
Ventilator-free days	20 (10-25)	21 (3-24)	2 (0-21)	4.5 (0-20)	0.018
Organ failure free-days	22 (11-26)	22 (9-26)	4 (0-18)	6.5 (0-21)	0.003

Data are n (%) or median (IQR). *p value for interaction between positive end-expiratory pressure (PEEP) assignment and phenotype.

EARLI and VALID Study

ARDS Severity by PaO ₂ /FiO ₂	Mortality in Low PEEP	Mortality in High PEEP	P value
Mild (PaO ₂ /FiO ₂ 200 – 300) n=828	35% (133/384)	34% (59/173)	0.96
Moderate (PaO ₂ /FiO ₂ 100 – < 200) n=1341	39% (172/441)	39% (174/449)	
Severe (PaO ₂ /FiO ₂ ≤ 100) n=644	48% (55/114)	46% (167/366)	

SOFA subgroups based on mean SOFA score			
ARDS Severity by SOFA score	Mortality in Low PEEP	Mortality in High PEEP	P value
Low SOFA (≤ 10) n=1314	30% (139/457)	30% (132/441)	0.51
High SOFA (> 10) n=1063	54% (165/308)	50% (230/464)	

ARDS Heterogeneity and Management

One size doesn't fit all.



ARDS Heterogeneity and Management

One Size Fits One.

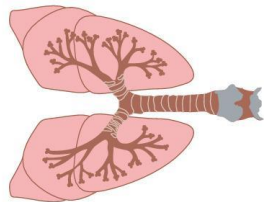


ARDS Treatment: Current and Future Direction

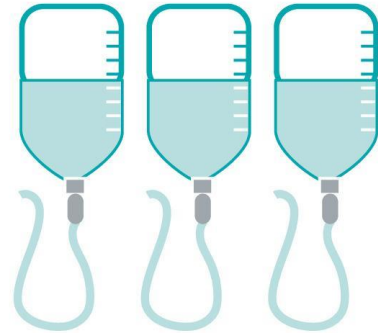
INTERVENTION



Low tidal volume ventilation
Avoiding recruitment maneuvers

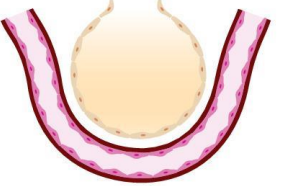


Prone positioning in
moderate-to-severe ARDS

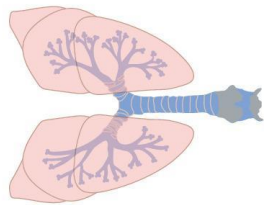


Conservative fluid strategy

OUTCOME



Mortality benefit

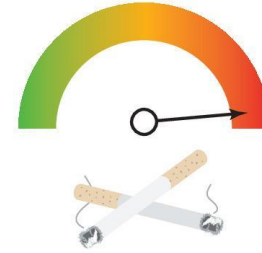


Mortality benefit

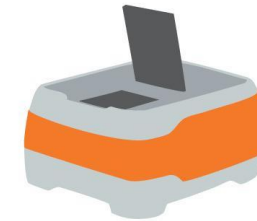


More ventilator-free
days

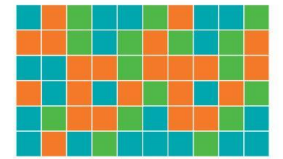
INTERVENTION



Public health strategies to
reduce environmental exposures



Prospective/real time
phenotyping



Metagenomic sequencing

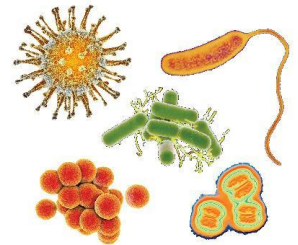
POSSIBLE OUTCOME



Lower background risk
of ARDS



Targeted therapies

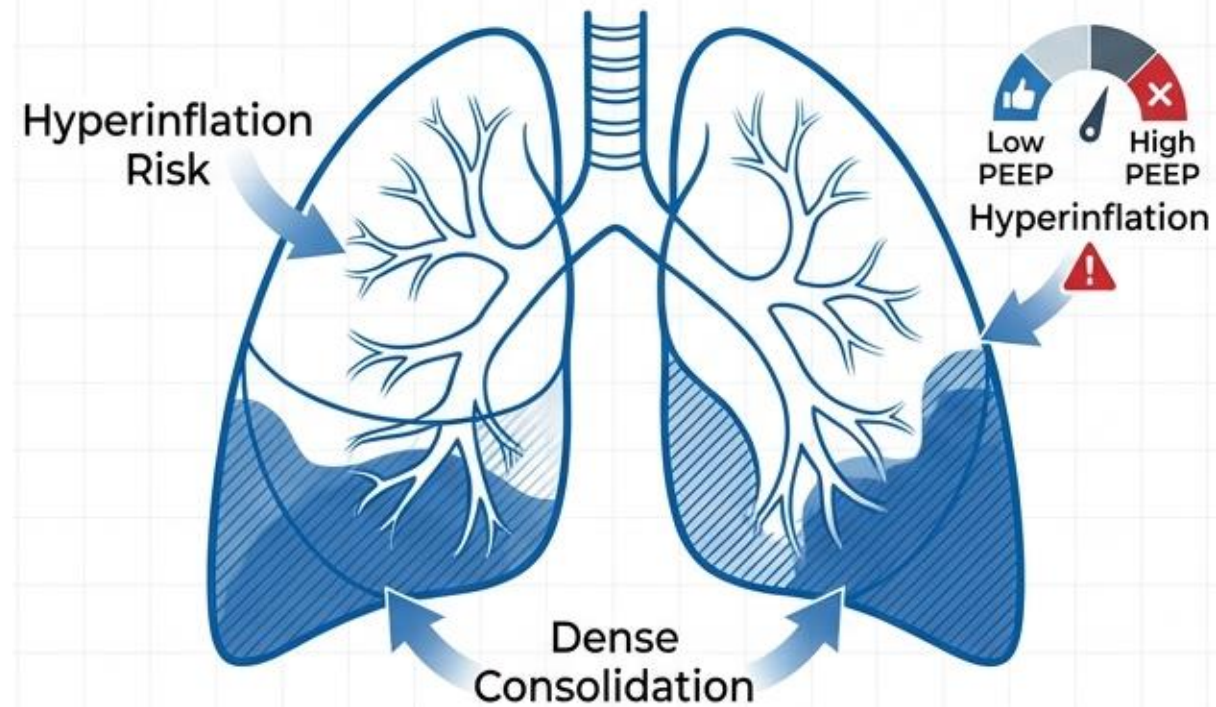


Rapid identification of
treatable pathogens

ARDS Treatment: Focal VS. Non-Focal

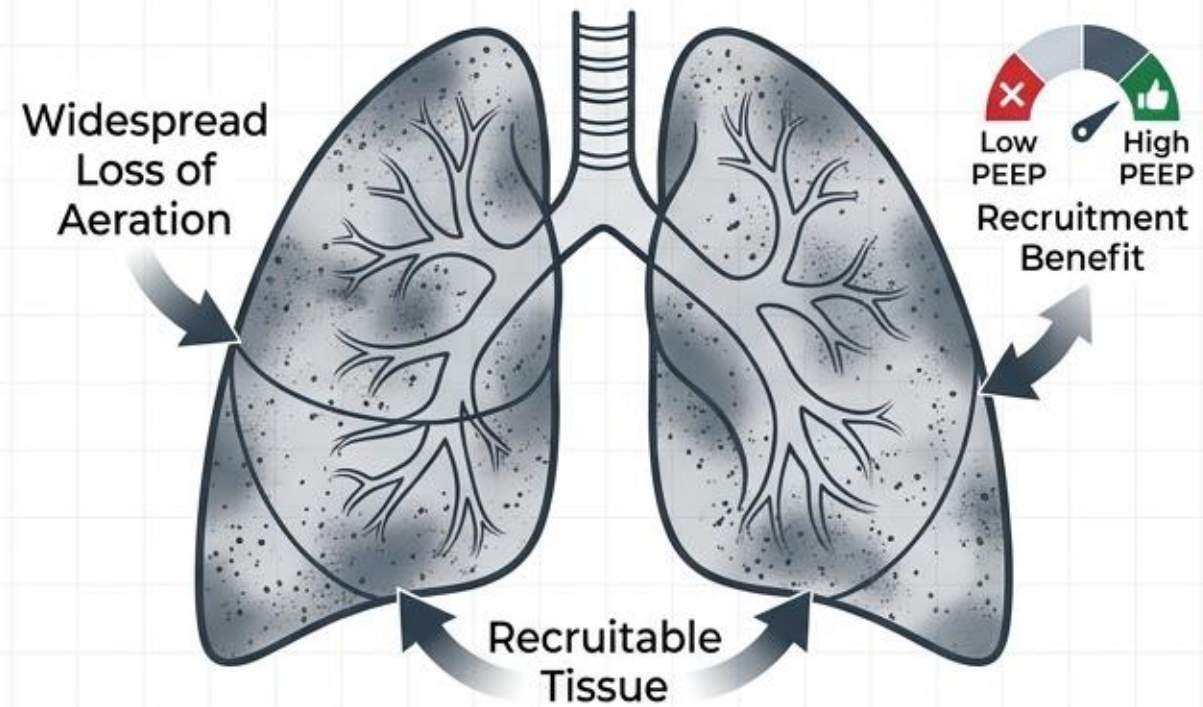
Focal ARDS

Localized consolidations. Higher lung volumes. Poor response to high PEEP (risk of hyperinflation of healthy tissue).

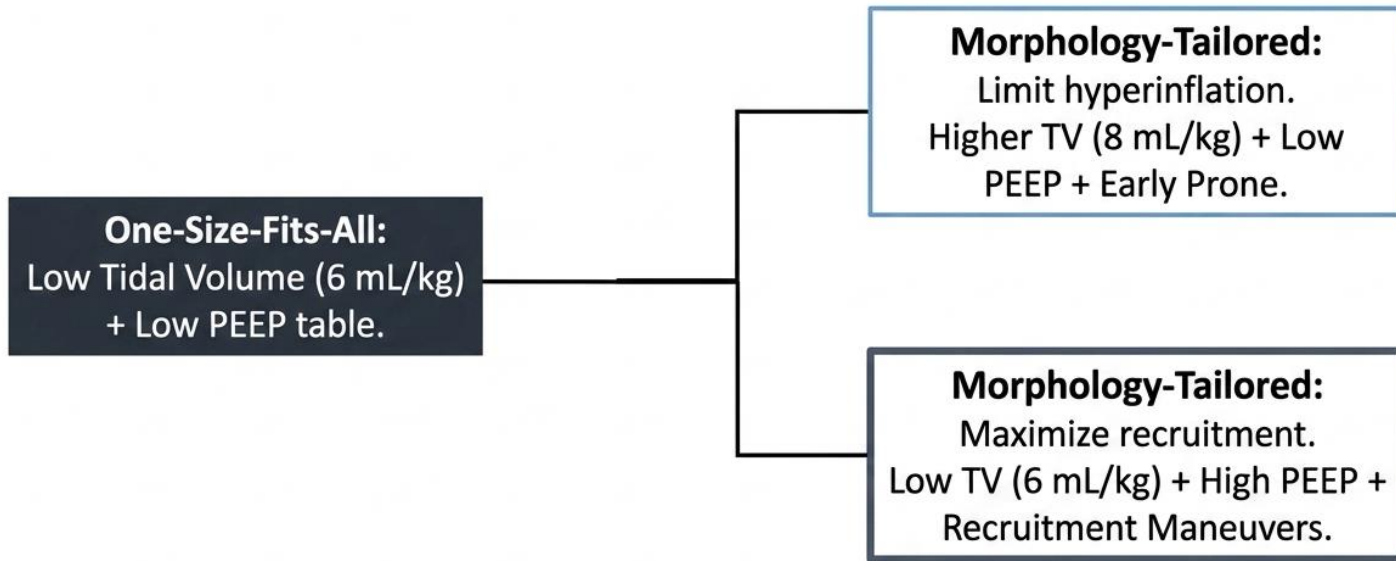


Non-Focal ARDS

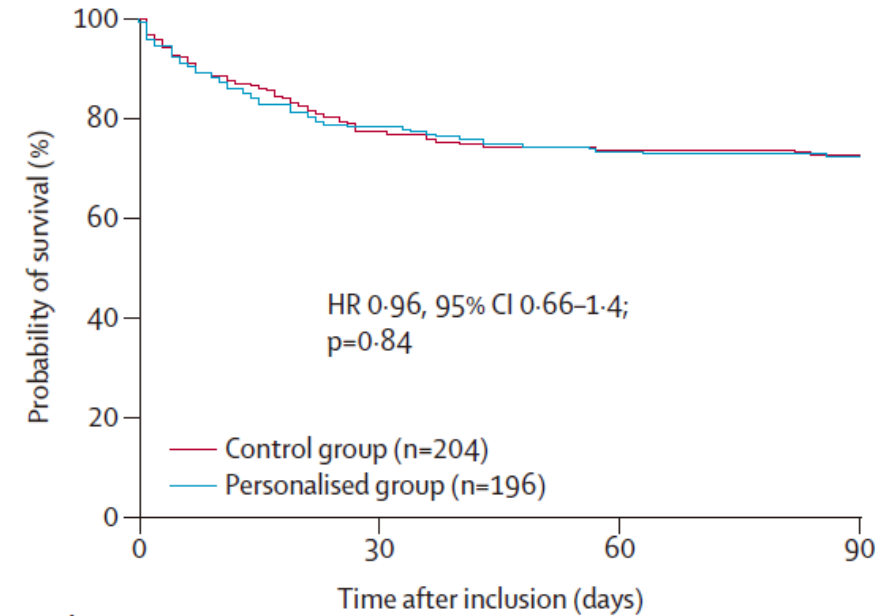
Diffuse, patchy loss of aeration. Lower lung compliance. High amount of recruitable lung tissue (benefits from high PEEP/recruitment).



ARDS Treatment: LIVE Study



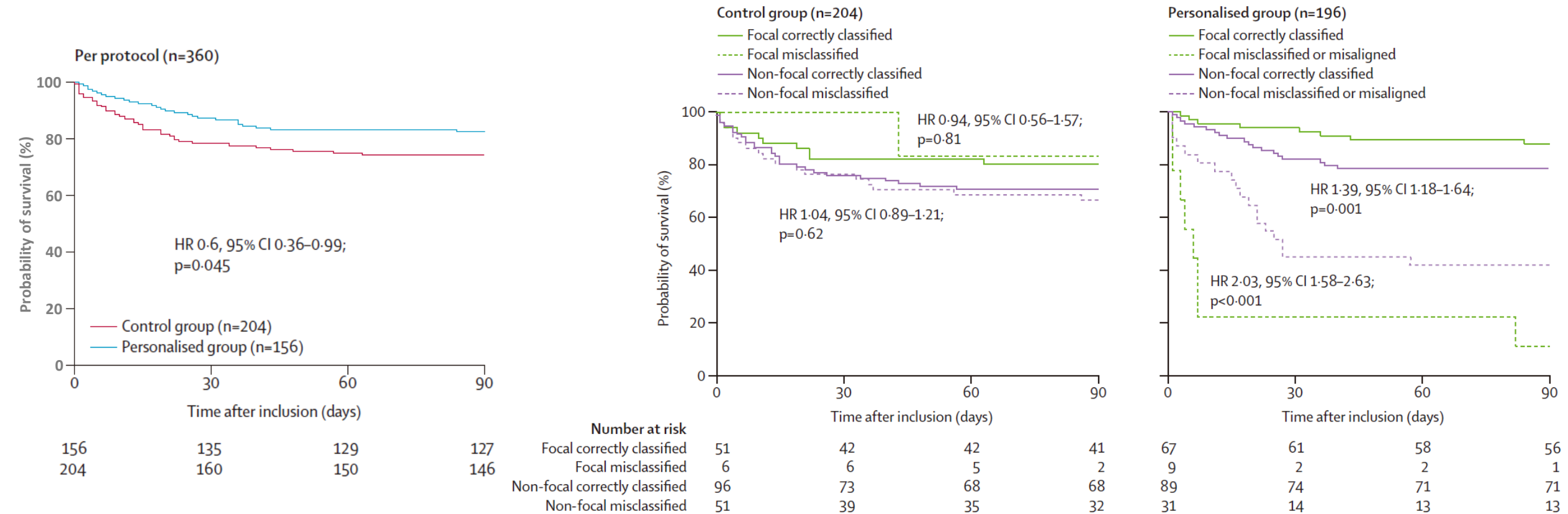
Outcomes



Number at risk				
Personalised group	196	151	144	141
Control group	204	160	150	146

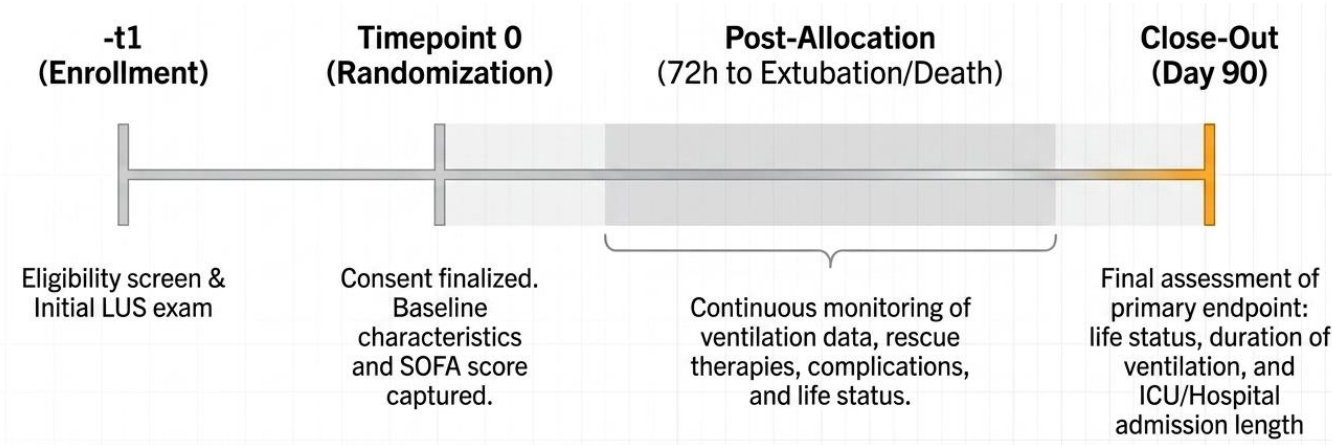
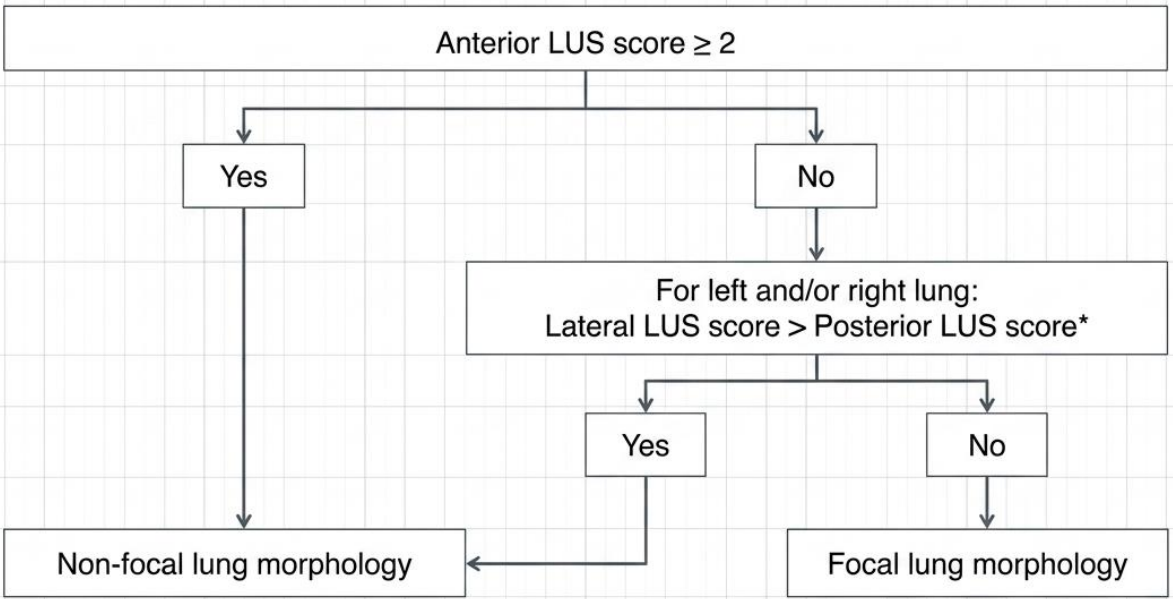
	Control Group (Standard Care)	Personalised Group (Focal)	Personalised Group (Non-Focal)
Tidal Volume	6 mL/kg	8 mL/kg	6 mL/kg
PEEP	via PEEP/FiO2 table	5-9 cm H2O	set to reach Pplat 30 cm H2O
Recruitment Manoeuvre	Rescue	Rescue	Mandatory
Prone Position	Encouraged	Mandatory	Rescue

ARDS Treatment: LIVE Study



ARDS Treatment: The PEGASUS Trial

Ultrasound-Guided Personalized Ventilation for ARDS



Morphology Intervention Matrix		
	Focal ARDS	Non-Focal ARDS
Tidal Volume	6 to 8 mL/kg PBW	4 to 6 mL/kg PBW
PEEP Targets	≤ 9 cmH2O	≥ 15 cmH2O
Recruitment Maneuvers	Only for rescue	Daily
Prone Positioning	PaO2/FiO2 < 200	PaO2/FiO2 < 150

RV Dysfunction in ARDS

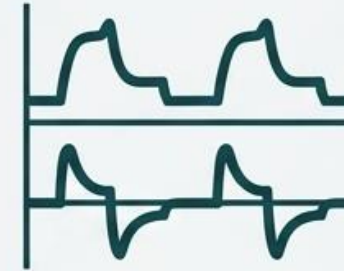
Pulmonary Vascular Effects



- ↑ Pulmonary vascular resistance
- ↓ Pulmonary arterial compliance
- ↑ Pulmonary vascular impedance
- ↑ Capillary derecruitment

Driven by: Alveolar collapse, endothelial injury, microthrombosis, hypoxic vasoconstriction.

Ventilatory/Mechanical Factors



- ↑ Driving pressure
- ↑ Transpulmonary pressure
- ↑ PaCO₂
- PEEP-induced capillary compression

**Acute cor
pulmonale**

**Systemic
venous
congestion**

**Organ
hypoperfusion**

**↓ Oxygen
delivery**

RV Dysfunction in ARDS

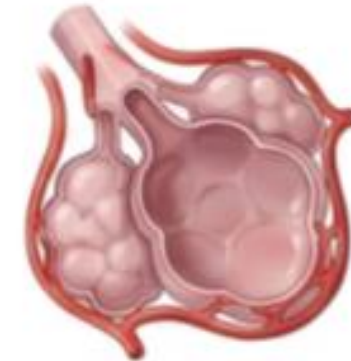
Adequate PEEP (Recruitment Range)



Restoration of FRC
↓ Hypoxic vasoconstriction
↓ PVR
↑ Pulmonary blood flow
↓ RV afterload
Improved RV-PA coupling

Result: Preserved/improved RV function and Cardiac Output.

Excessive PEEP (Overdistension Range)



Alveolar overdistension
Compression of alveolar vessels
↑ PVR
↓ Pulmonary blood flow
↑ RV afterload
RV-PA uncoupling
Dilatation & Septal shift

Result: Impaired RV function and reduced Cardiac Output.

RV Dysfunction in ARDS

Optimize Mechanics

Find the PEEP sweet spot at FRC to minimize PVR. Avoid overdistension and atelectasis.

Unload the RV

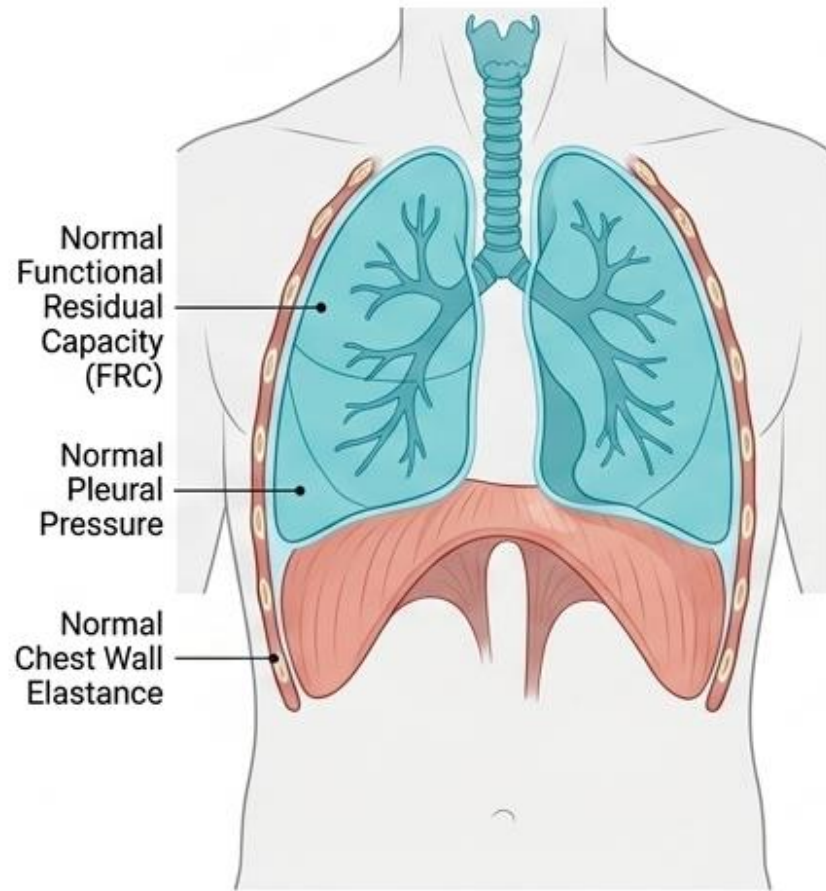
Utilize selective pulmonary vasodilators (iNO, Prostacyclin) and inotropes (Dobutamine) to restore RV-PA coupling.

Maintain Perfusion

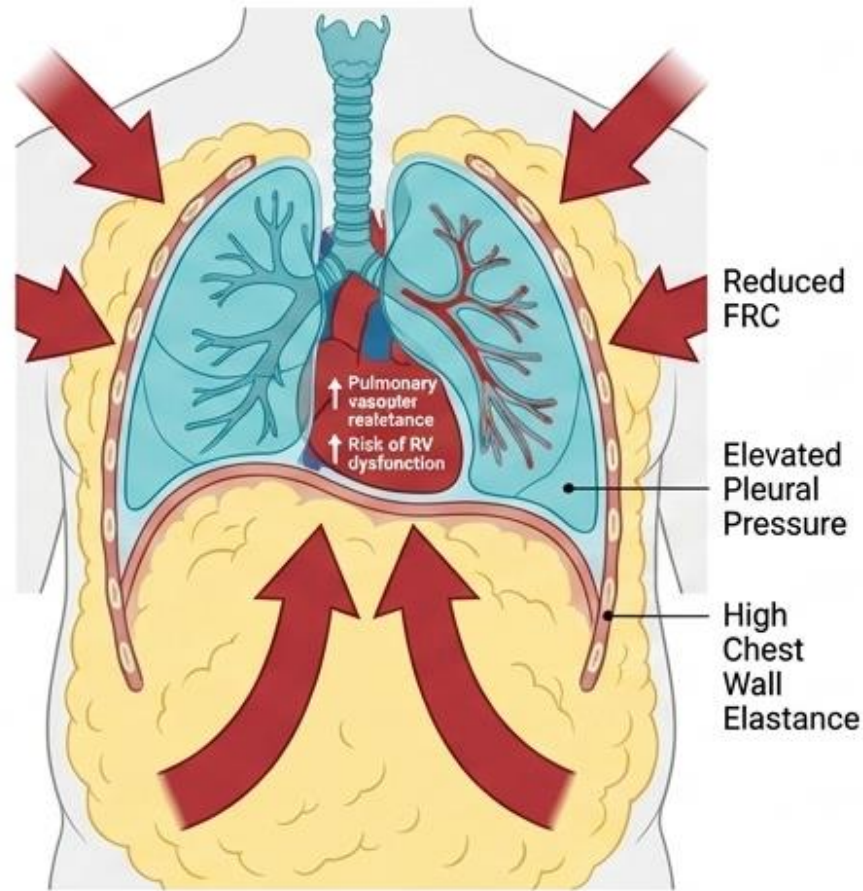
Defend coronary perfusion pressure and systemic hemodynamics with first-line vasopressors (Norepinephrine).

ARDS and Obesity

Normal-Weight Patient



Obese Patient



Four Dimensions of Failure

FRC: Reduced by 5–15% for every 5 kg/m² increase in BMI. Promotes dependent airway closure.

Pleural Pressure: Markedly elevated, collapsing dependent alveoli.

Chest Wall Elastance: High. The chest wall resists expansion, artificially inflating airway pressure readings.

Right Ventricular Afterload: Increased pulmonary vascular resistance due to collapsed vessels and hypoxic vasoconstriction risks RV dysfunction.

ARDS and Obesity



The Physiological Reality

- Standard PEEP (5–10 cmH₂O) is usually insufficient to overcome the heavy pleural pressures of class III obesity.
- Consequence of Under-PEEPing: Recurrent airway closure during tidal breathing, severe atelectasis, and refractory V/Q mismatch.

The Clinical Strategy

- Start with **adequate PEEP** to maintain **positive transpulmonary pressure**.
- Caution with Recruitment Maneuvers: While transient oxygenation improvement is possible, routine aggressive recruitment maneuvers are not supported due to **the high risk** of severe hemodynamic instability (impaired venous return against high intrathoracic pressure).
- Address the atelectasis as the root cause before blindly escalating FiO₂ to avoid absorption atelectasis.

ARDS and Obesity

Lung protection requires 6–8 mL/kg PBW tidal volumes and individualized, higher PEEP titration

Tidal Volume (VT) Selection

PBW Indexing: Tidal volume must always be indexed to Predicted Body Weight, calculated from patient height and sex.

Actual Weight Error: Lung size does not increase with body mass. Using actual weight delivers massive, injurious volumes.

Protective Targets: Maintain low VT of 6-8 mL/kg PBW as the cornerstone of lung protection to avoid overdistension.

PEEP Titration Strategies

Higher PEEP Requirement: Standard PEEP (5–10 cmH₂O) is often insufficient to counteract elevated abdominal/thoracic pressures.

Airway Closure Prevention: Higher PEEP is required to maintain positive transpulmonary pressure and recruit collapsed alveoli.

Individualization: PEEP should be titrated using esophageal pressure, electrical impedance tomography, or driving pressure.

Recruitment Maneuvers

No Routine Use: Aggressive recruitment strategies (such as those in the ART trial) are not supported for routine clinical use.

Refractory Hypoxemia: Selectively consider recruitment maneuvers for severe, refractory hypoxemia.

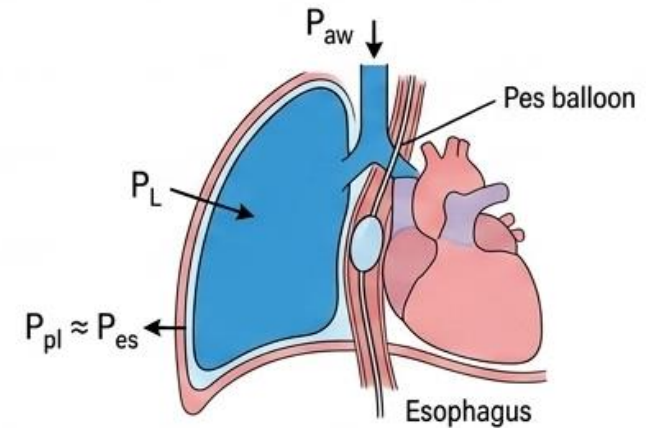
Hemodynamic Risk: Higher intrathoracic pressure can severely impair venous return, leading to circulatory instability.

ARDS and Obesity

Advanced monitoring separates lung from chest wall

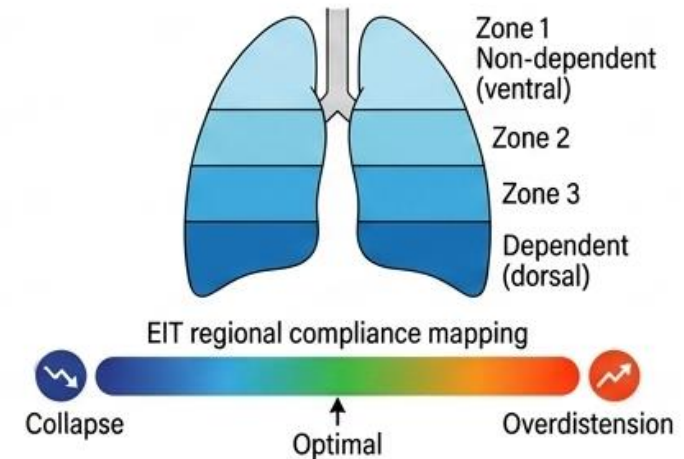
Esophageal Pressure (Pes) Monitoring

- **Function:** Estimates regional pleural pressure.
- **Value:** Allows the clinician to calculate Transpulmonary Pressure (P_L) in real-time, targeting a positive end-expiratory P_L to prevent collapse, and limiting end-inspiratory P_L to prevent over-distension.

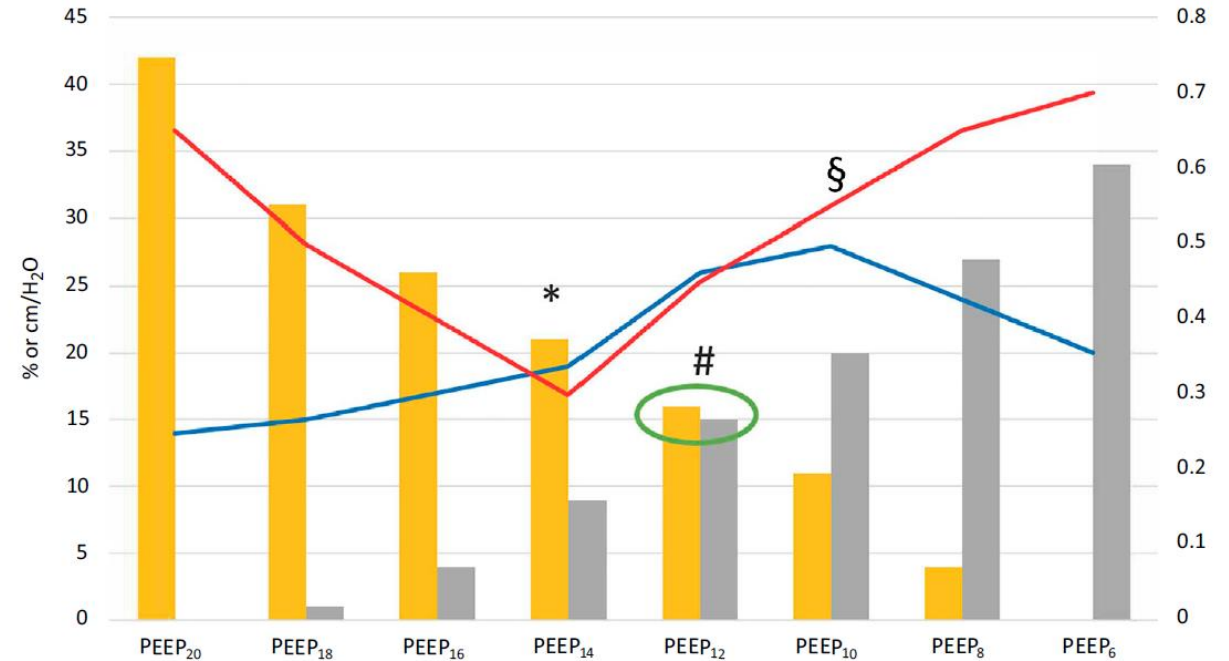
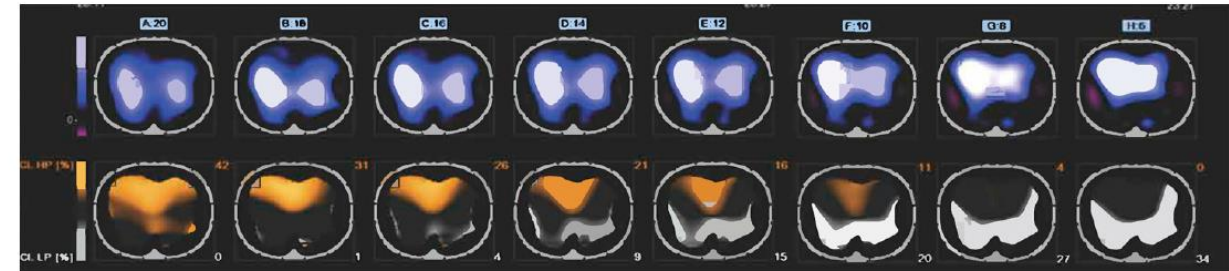
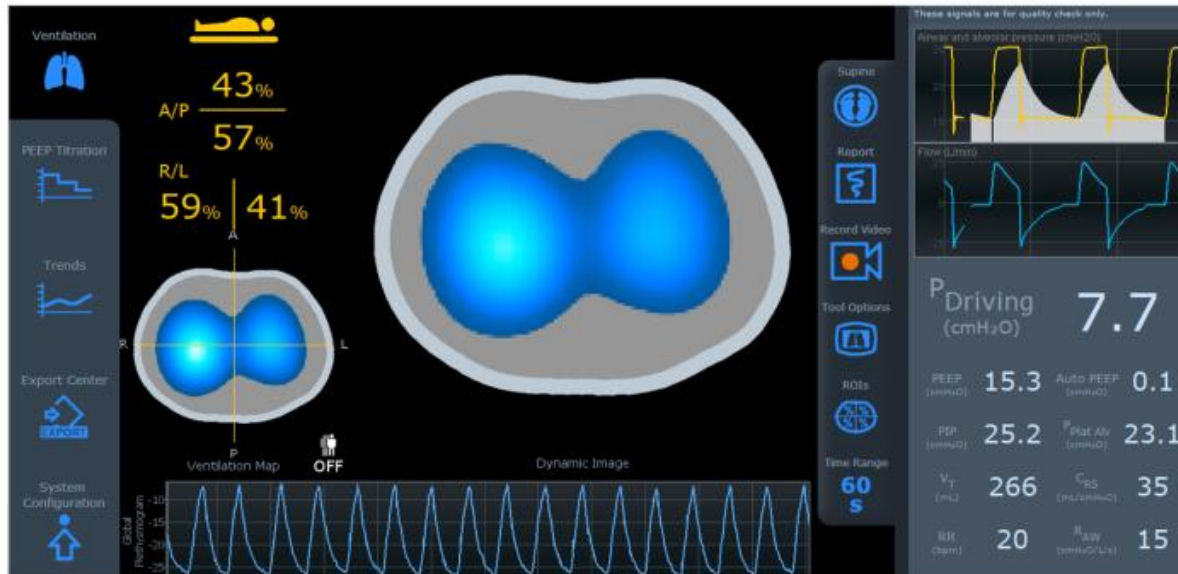
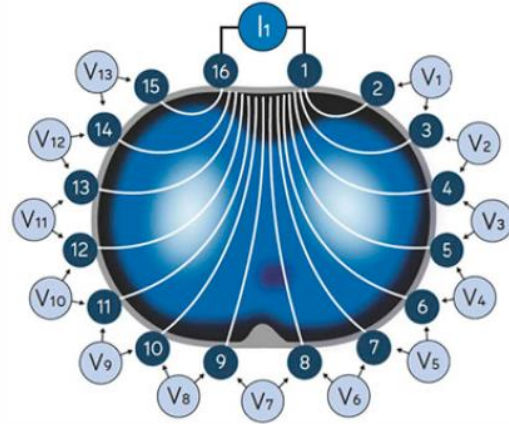
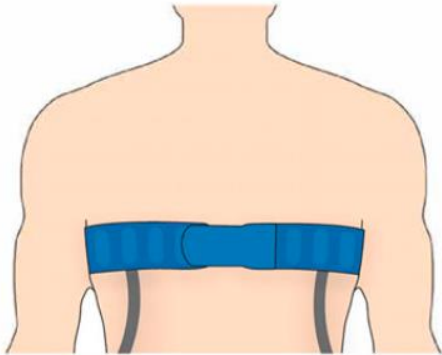


Electrical Impedance Tomography (EIT)

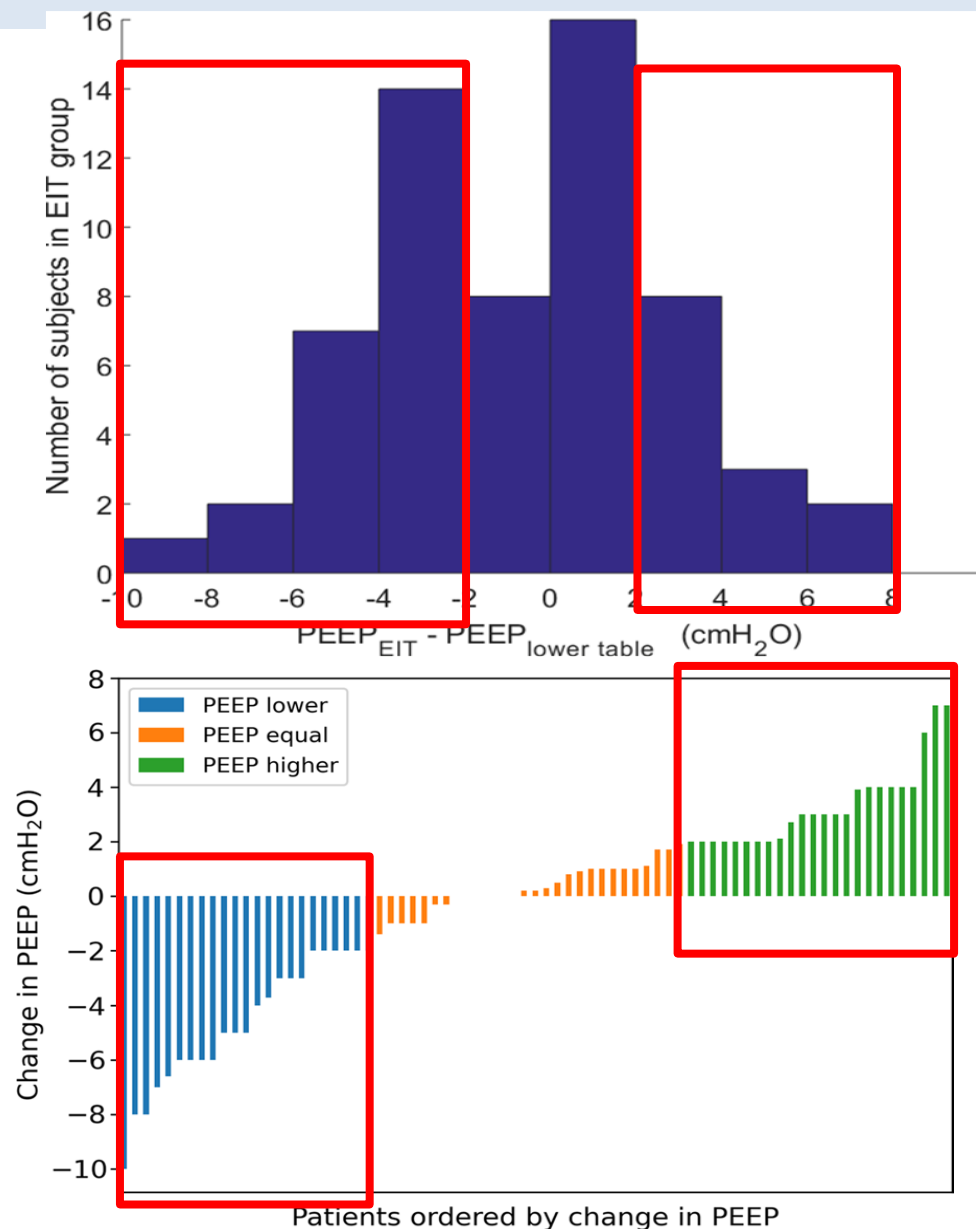
- **Function:** Non-invasive, real-time bedside imaging of regional ventilation.
- **Value:** Identifies hidden regional lung collapse (dorsal) and overdistention (ventral), guiding the exact PEEP required to balance the two.



Electrical Impedance Tomography (EIT)



PEEP Guided by EIT in ARDS



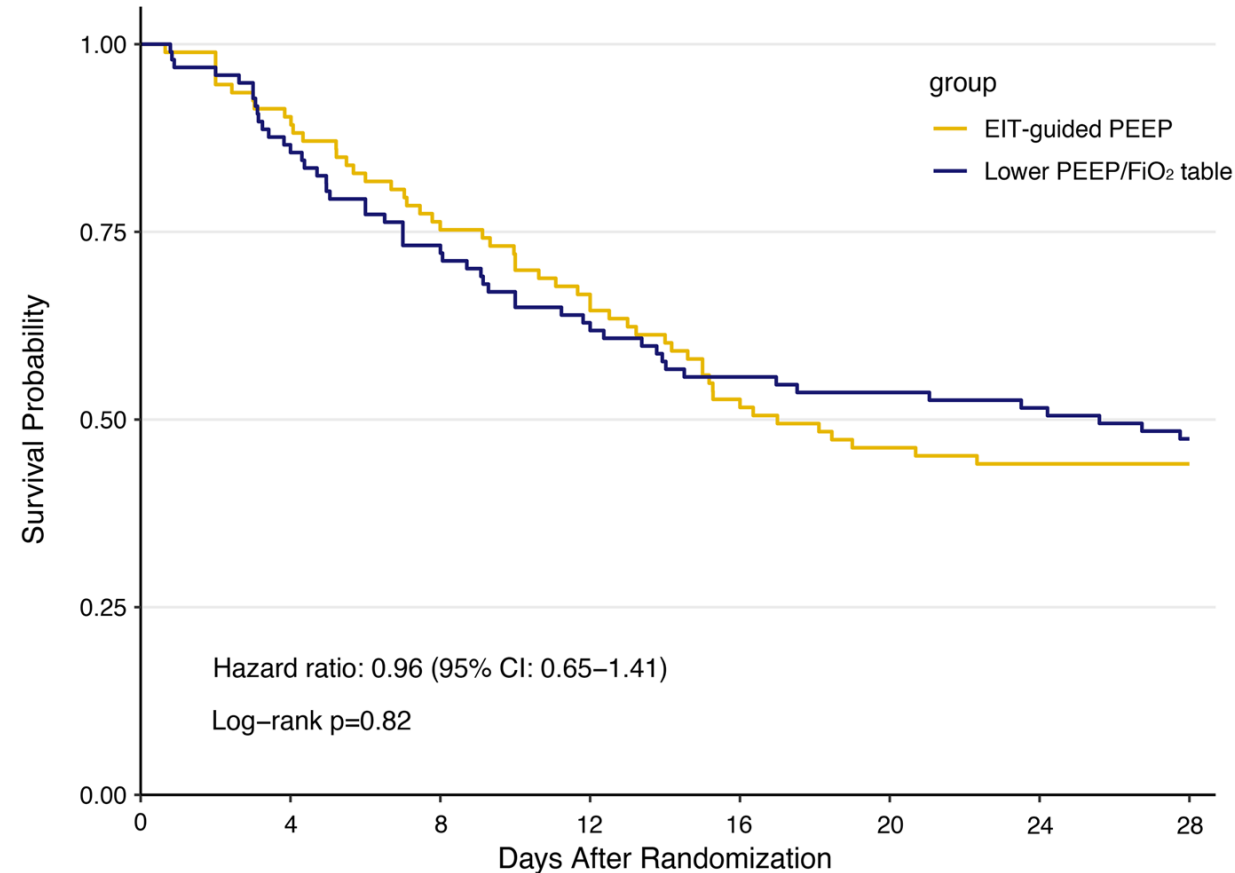
Variables	EIT group N=61	Control group N=56	p value
Clinical outcome			
28-day mortality (<i>n</i> , %)	13 (21%)	15 (27%)	0.634
Ventilator-free days at day 28 (D)	14.0 (0.0, 23.0)	18.5 (0.0, 24.0)	0.764
Length of ICU stay (D)	13.0 (7.0, 25.0)	10.0 (7.0, 14.8)	0.169
ΔD1 SOFA score	0 (− 1, 1)	0.5 (− 1, 2.75)	0.021*
ΔD2 SOFA score	− 1 (− 3.5, 0)	1 (− 2, 2)	<0.0001*
Successful extubation (<i>n</i> , %)	30 (49%)	31 (55%)	0.629
Tracheostomy (<i>n</i> , %)	17 (28%)	11 (20%)	0.409
Adjuvant therapy			
Neuromuscular blocker (<i>n</i> , %)	12 (20%)	5 (9%)	0.166
Prone position (<i>n</i> , %)	30 (49%)	23 (41%)	0.487
Glucocorticoid therapy (<i>n</i> , %)	11 (18%)	6 (11%)	0.390

**p* < 0.05

PEEP Guided by EIT in ARDS

Electrical impedance tomography-guided positive end-expiratory pressure and mortality of patients with the acute respiratory distress syndrome: the **EITVent** randomized clinical trial

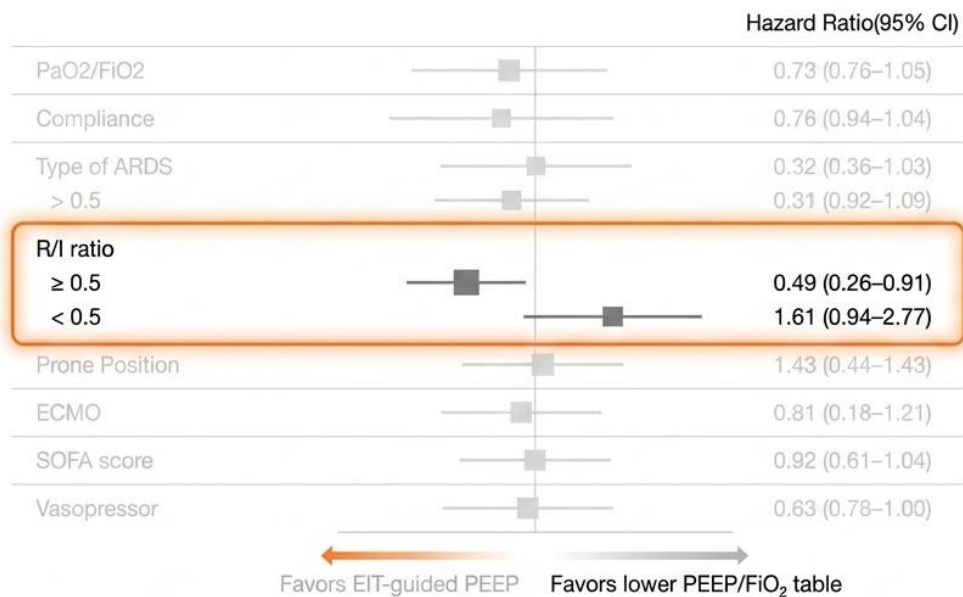
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Number at Risk								
EIT-guided PEEP	93	84	70	62	49	43	41	41
Lower PEEP/FiO ₂ table	97	84	71	61	54	52	50	46

PEEP Guided by EIT in ARDS

Electrical impedance tomography-guided positive end-expiratory pressure and mortality of patients with the acute respiratory distress syndrome: the **EITVent** randomized clinical trial



High Recruiters (R/I ≥ 0.5)

48% Prevalence: ~48% of the trial cohort.

Result: EIT significantly reduced mortality (**35.6% vs 60.0%**).

Hazard Ratio: **0.49**
(p = 0.024).



Low Recruiters (R/I < 0.5)

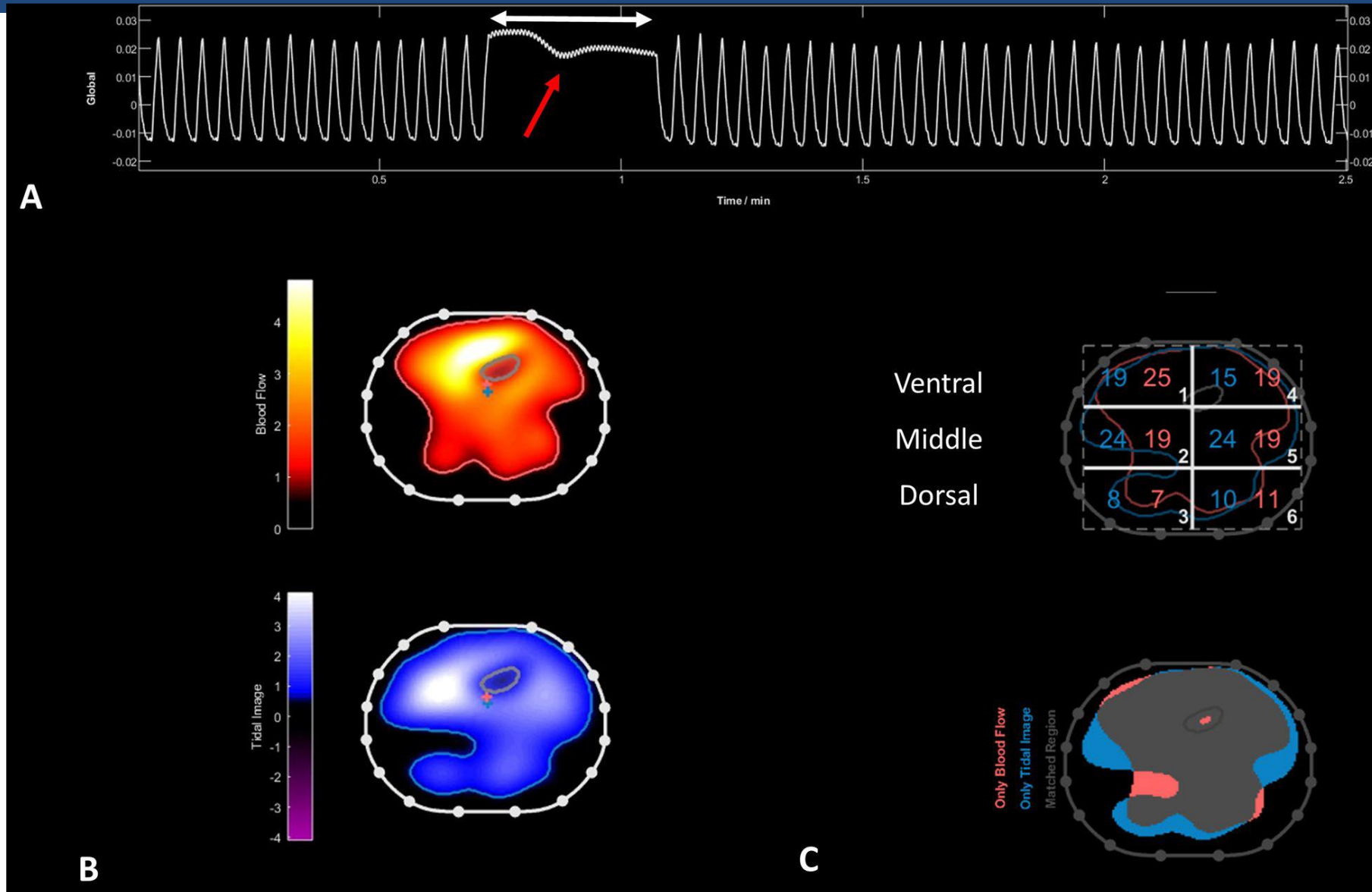
52% Prevalence: ~52% of the trial cohort.

Result: EIT showed a trend toward harm (73.2% vs 51.1%).

Hazard Ratio: 1.61 (Not statistically significant, but clinically concerning).



V/Q measured by EIT in ARDS



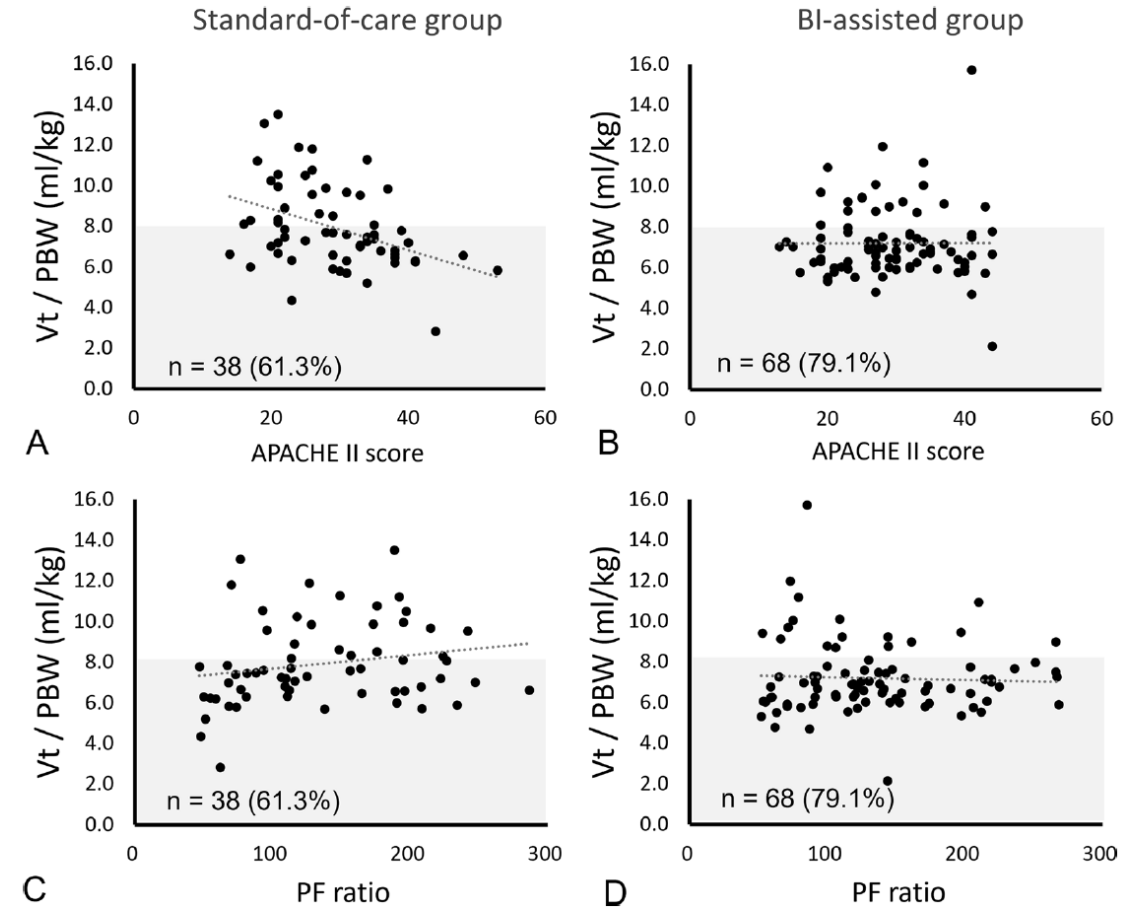
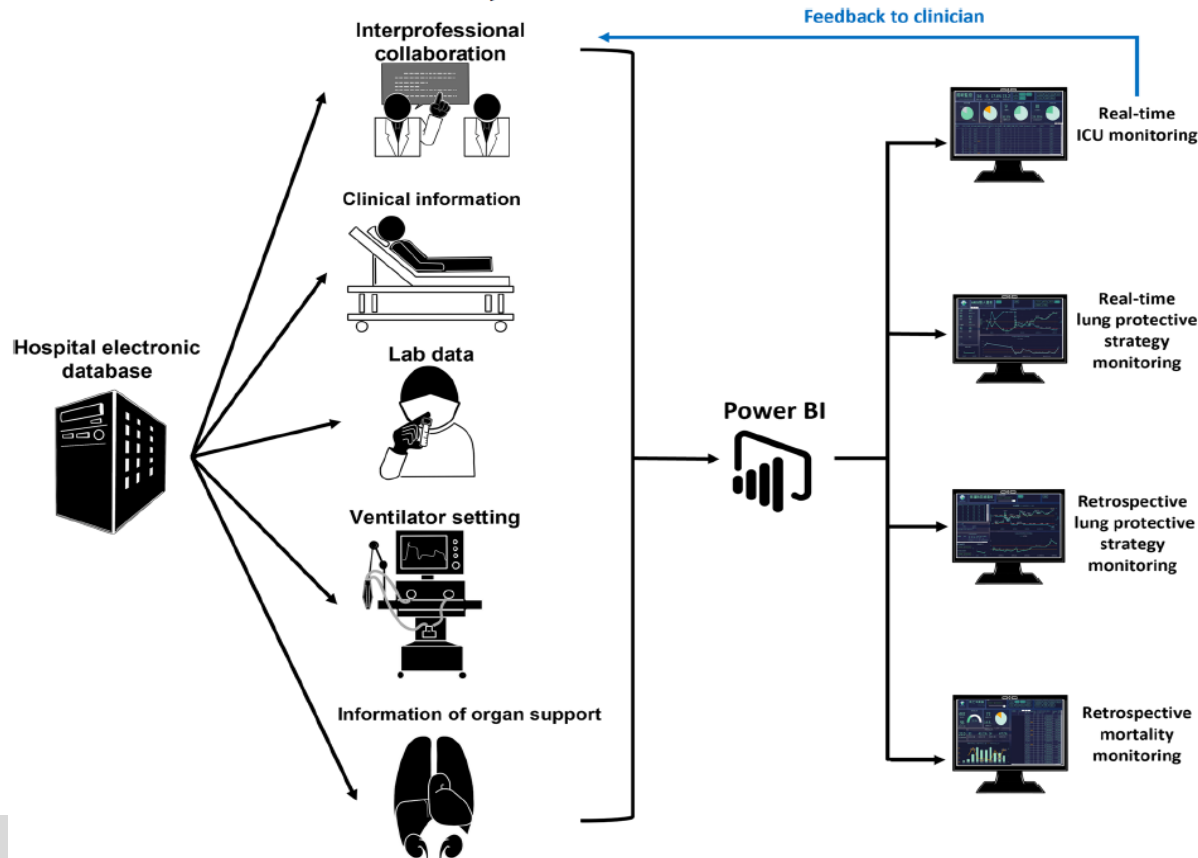
Artificial Intelligence in ARDS

RESEARCH

Open Access



Using real-time visualization system for data-driven decision support to achieve lung protective strategy: a retrospective observational study

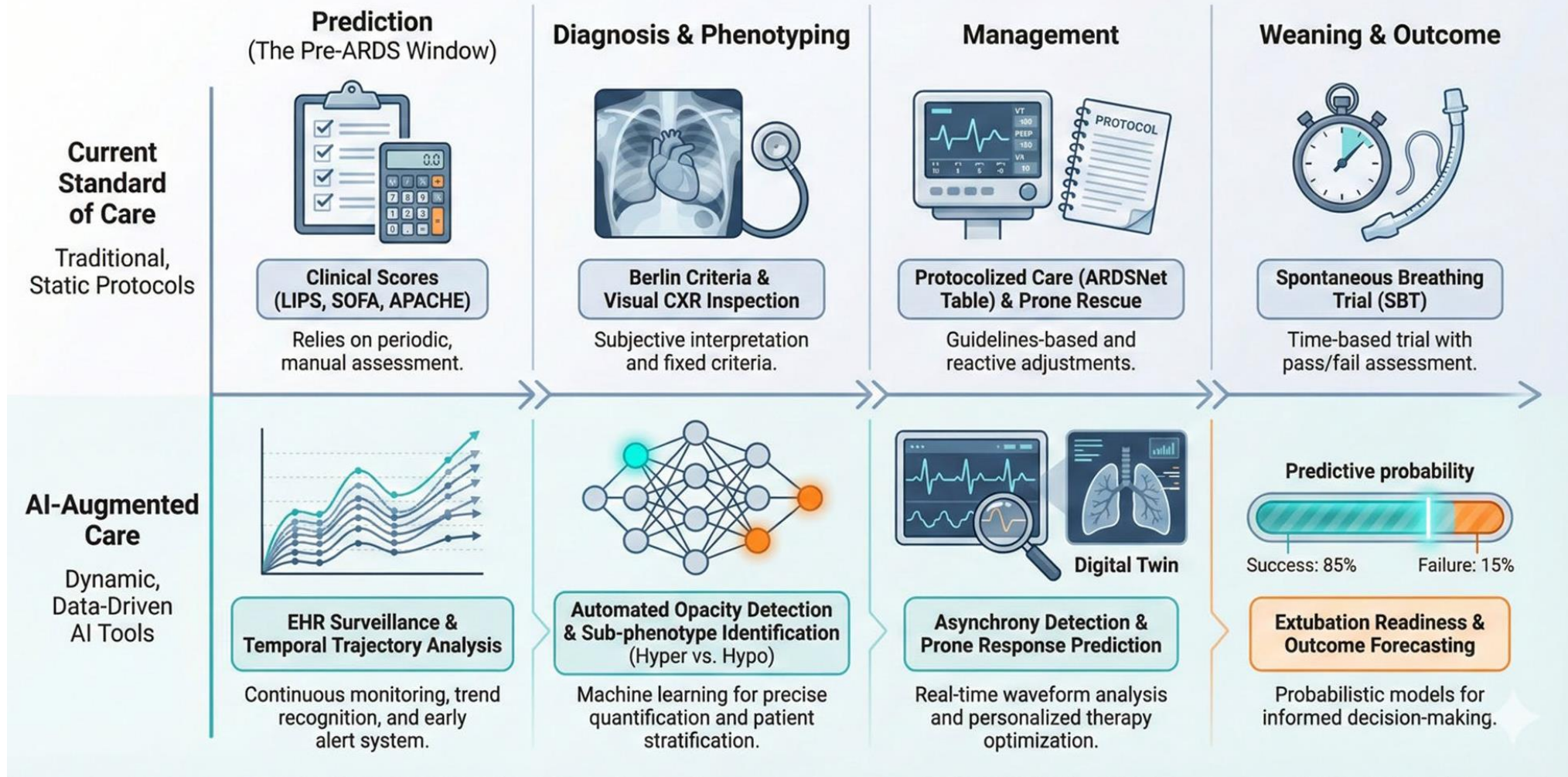


	All (N = 148)	Standard-of-care group (N = 62)	BI-assisted group (N = 86)	p value
Duration of IMV, day	11 (5–26)	13 (6–26.8)	10 (5–26)	0.741
ICU mortality	70 (47.3%)	36 (58.1%)	34 (39.5%)	0.026
Hospital mortality	84 (56.8%)	42 (67.7%)	42 (48.8%)	0.022
ICU LOS, day	12 (6.3–19.8)	13 (8–23.3)	10 (5–15.3)	0.055
Hospital LOS, day	26 (14–49.8)	23 (12.8–39.3)	28 (14.8–50.5)	0.221

IMV invasive mechanical ventilation, ICU intensive care unit, LOS length of stay

Artificial Intelligence in ARDS

The AI-Augmented ARDS Lifecycle



Precision Medicine for ARDS

**Prognostic Enrichment and
Machine Learning**

Predictive Enrichment

Heterogeneity

