Prise en charge du Syndrome de Détresse Respiratoire Aigue (SDRA) en 2016

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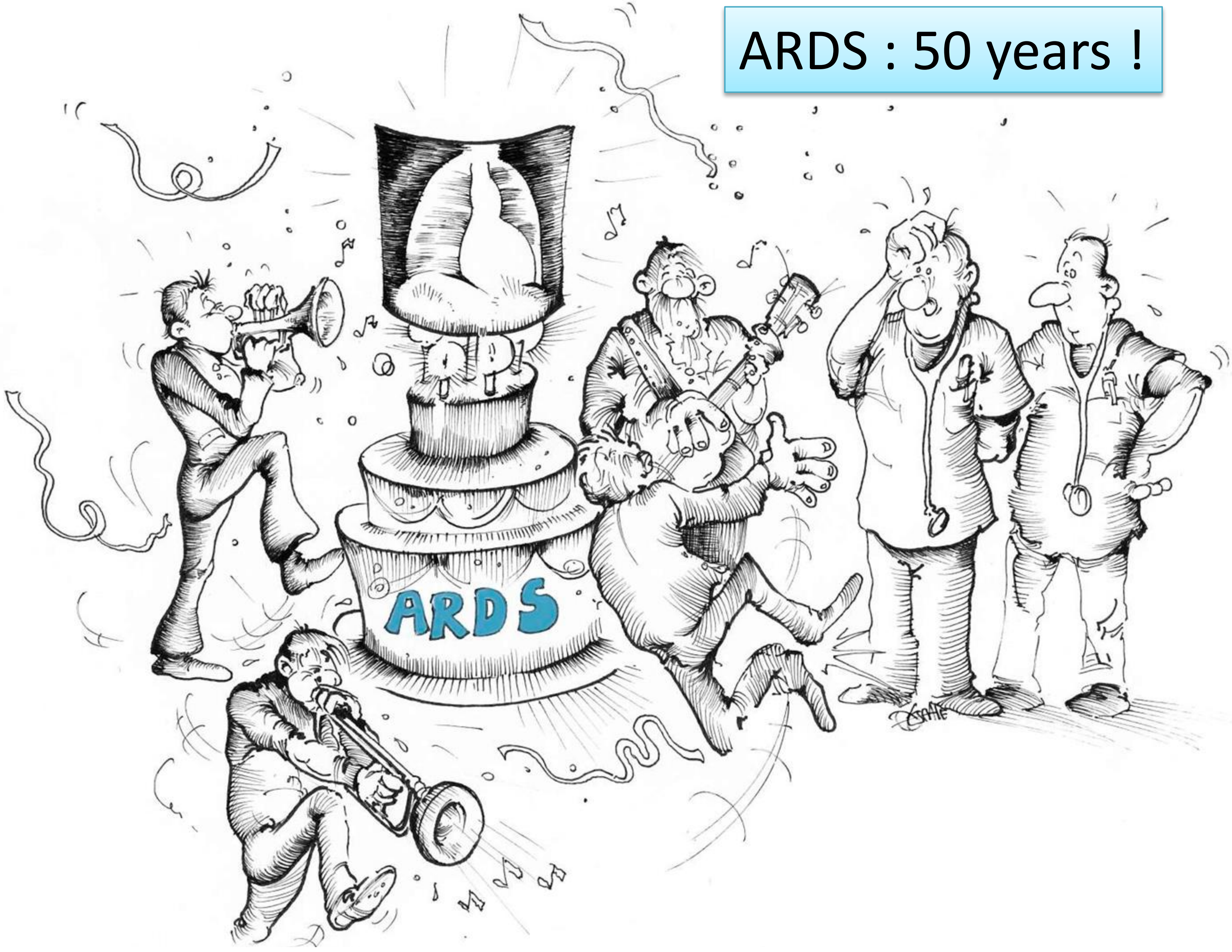
INSERM 1046 Université de Montpellier
34295 MONTPELLIER – France

Mai 2016

ARDS : 50 years !



Qu’est ce qui a changé depuis 50 ans ?

The Lancet · Saturday 12 August 1967

ACUTE RESPIRATORY DISTRESS IN ADULTS

DAVID G. ASHBAUGH
M.D. Ohio State
ASSISTANT PROFESSOR OF SURGERY

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M.D. Colorado
ISTANT IN MEDICINE AND AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE

THOMAS L. PETTY
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BERNARD E. LEVINE
M.D. Michigan
ERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE*

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

Described in 12 patients with:

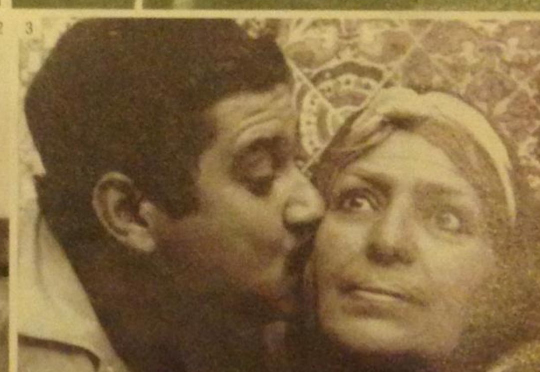
- acute respiratory distress
- cyanosis refractory to oxygen therapy
- decreased lung compliance
- diffuse infiltrates on the chest radiograph

Happy Birthday : 50 years !

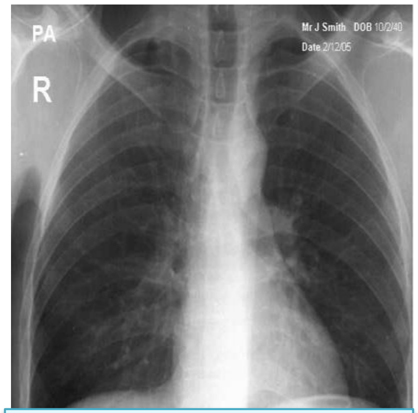
LA TÉLÉVISION TUNISIENNE FÊTE SES 50 ANS !

IL FAUT REMONTER LE TEMPS, 50 ANS PRÉCISÉMENT, POUR ASSISTER À LA NAISSANCE DE LA TÉLÉVISION TUNISIENNE.

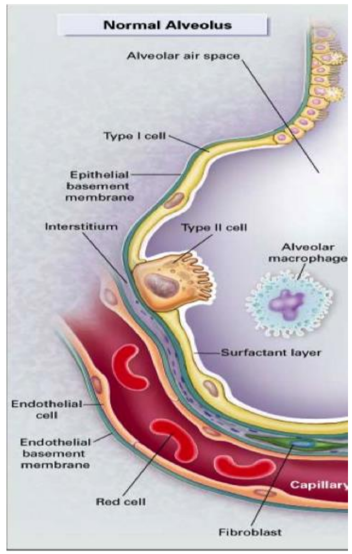
Crédit photos : Archives de la Télévision Nationale Tunisienne

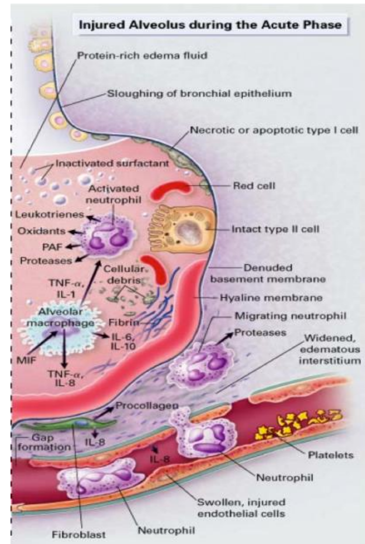


At risk → Insult → Injury → Clinical/Gas exchange/Imaging



« Normal »





ARDS

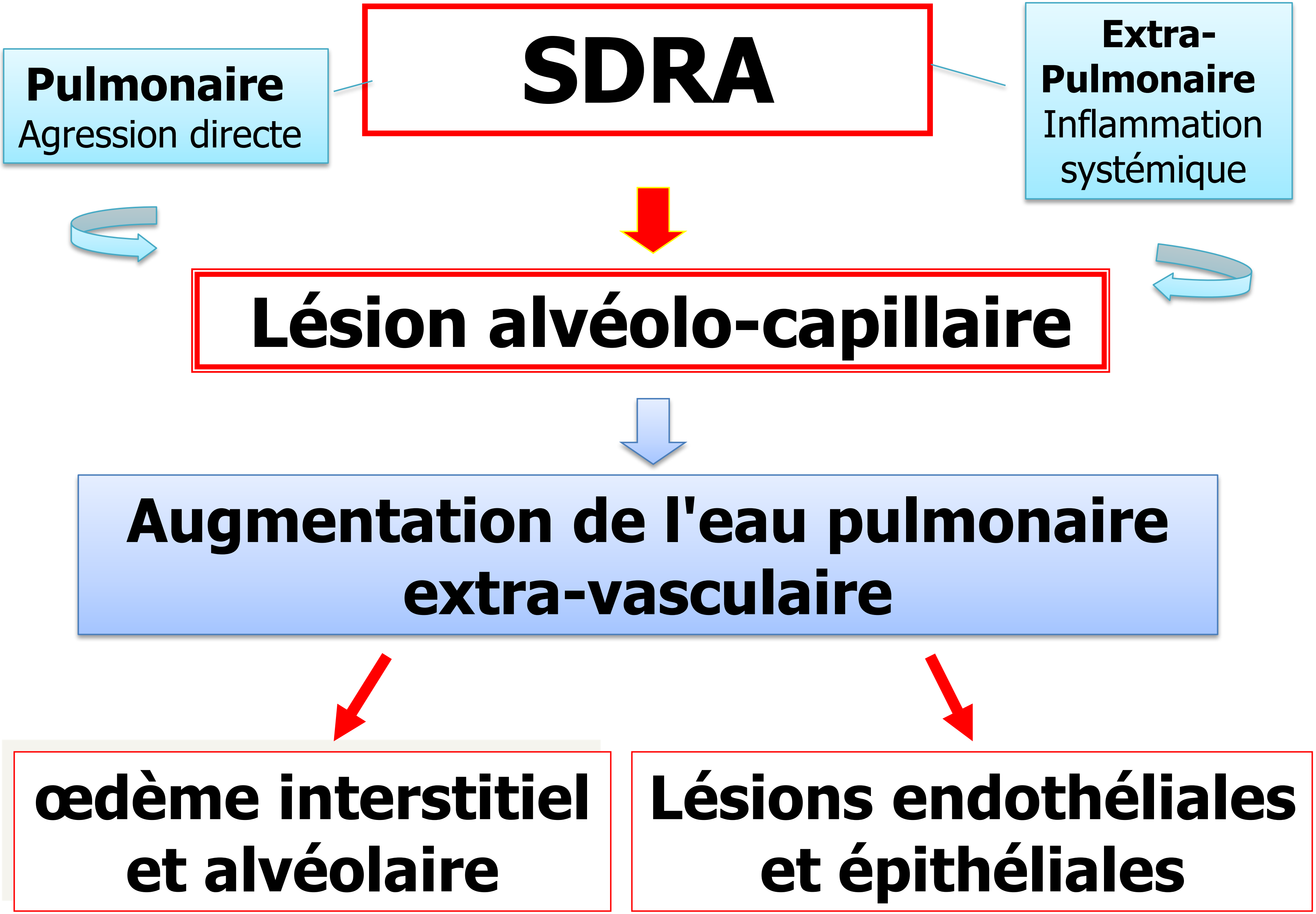
Normal gas exchange

Etiologic cause

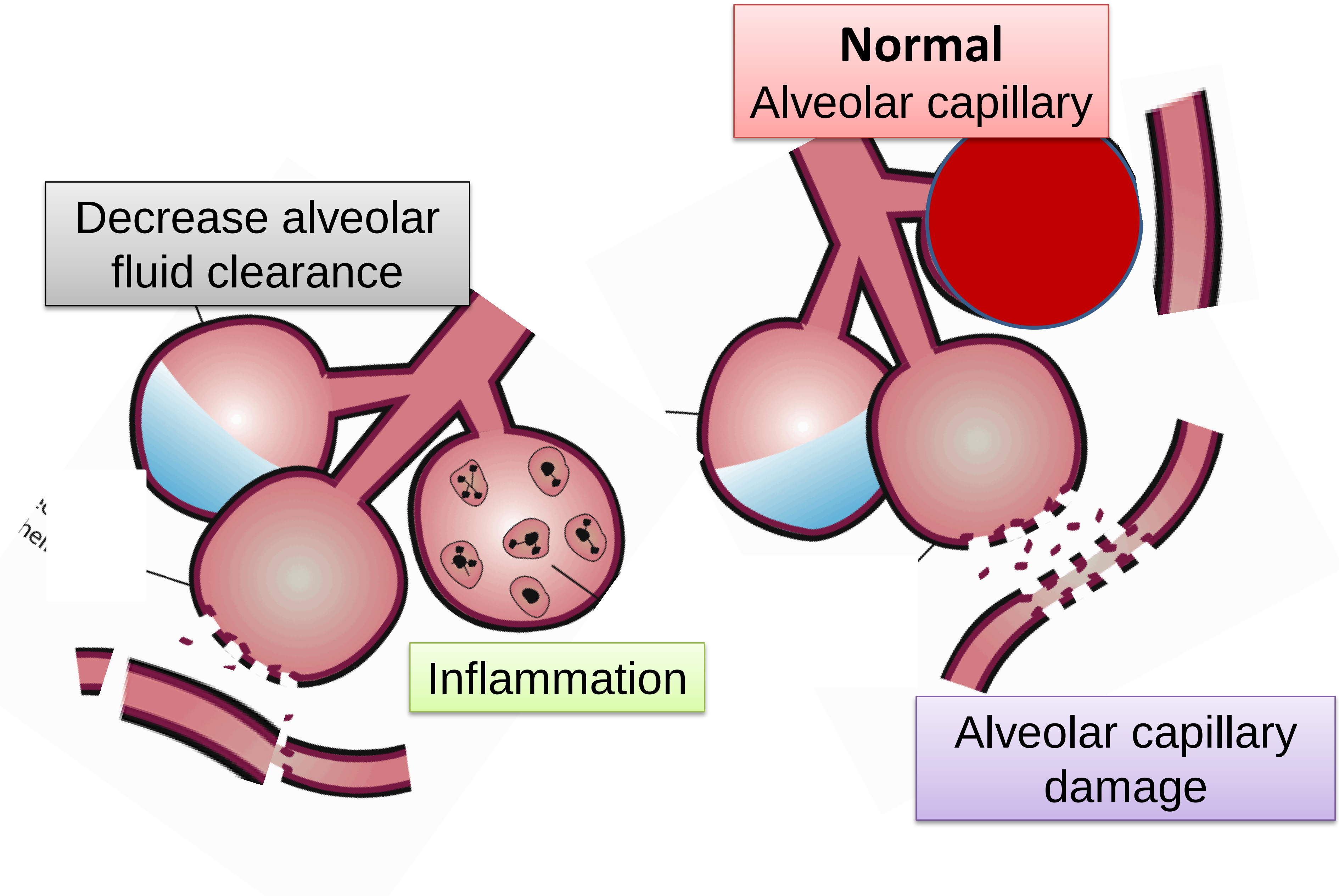
(initial pathology)

→

Alteration gas exchange (hypoxemia)


SDRA
Pulmonaire
Agression directe
Extra-Pulmonaire
Inflammation systémique
Lésion alvéolo-capillaire
Augmentation de l'eau pulmonaire extra-vasculaire
œdème interstitiel et alvéolaire
Lésions endothéliales et épithéliales

Inhomongenity in whole lung failure



The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

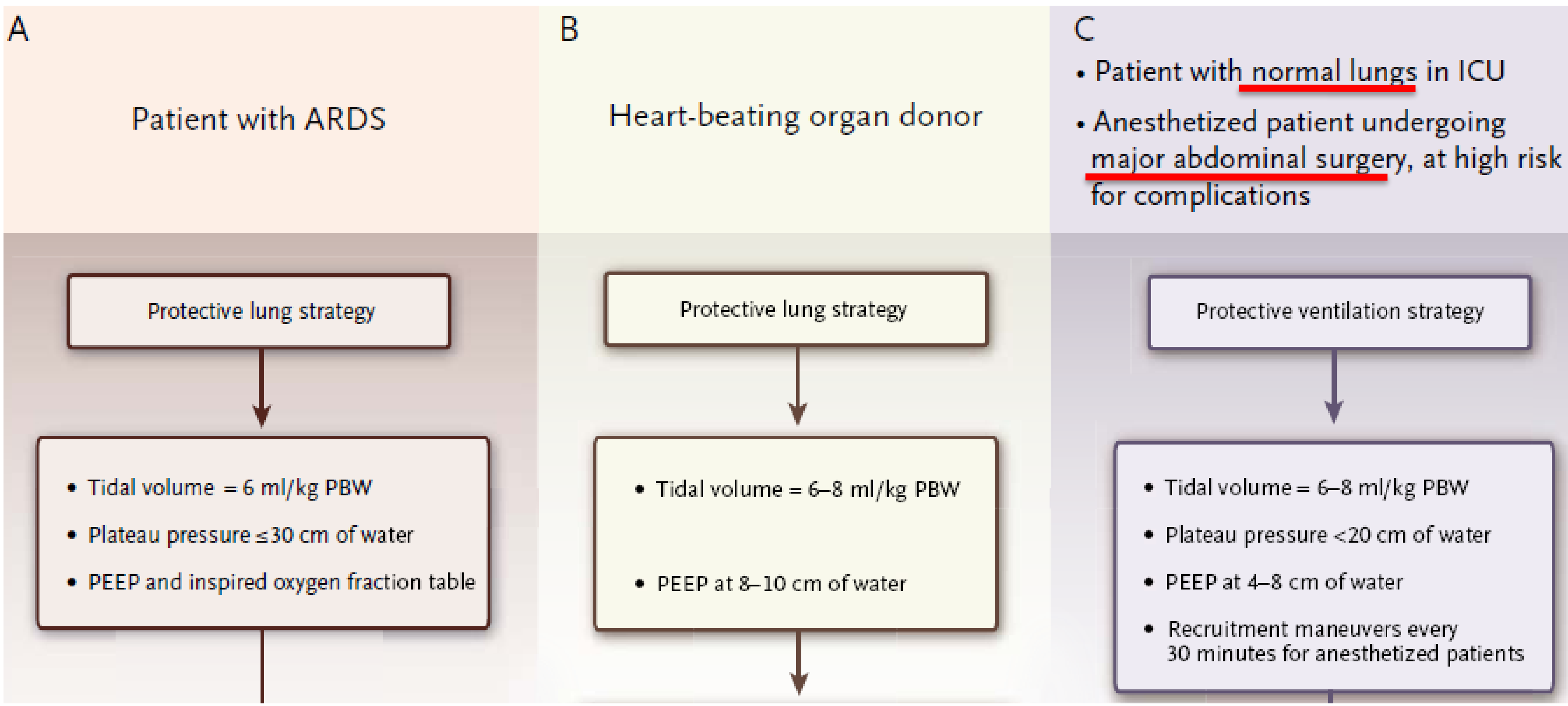
N ENGL J MED 369;22 NEJM.ORG NOVEMBER 28, 2013

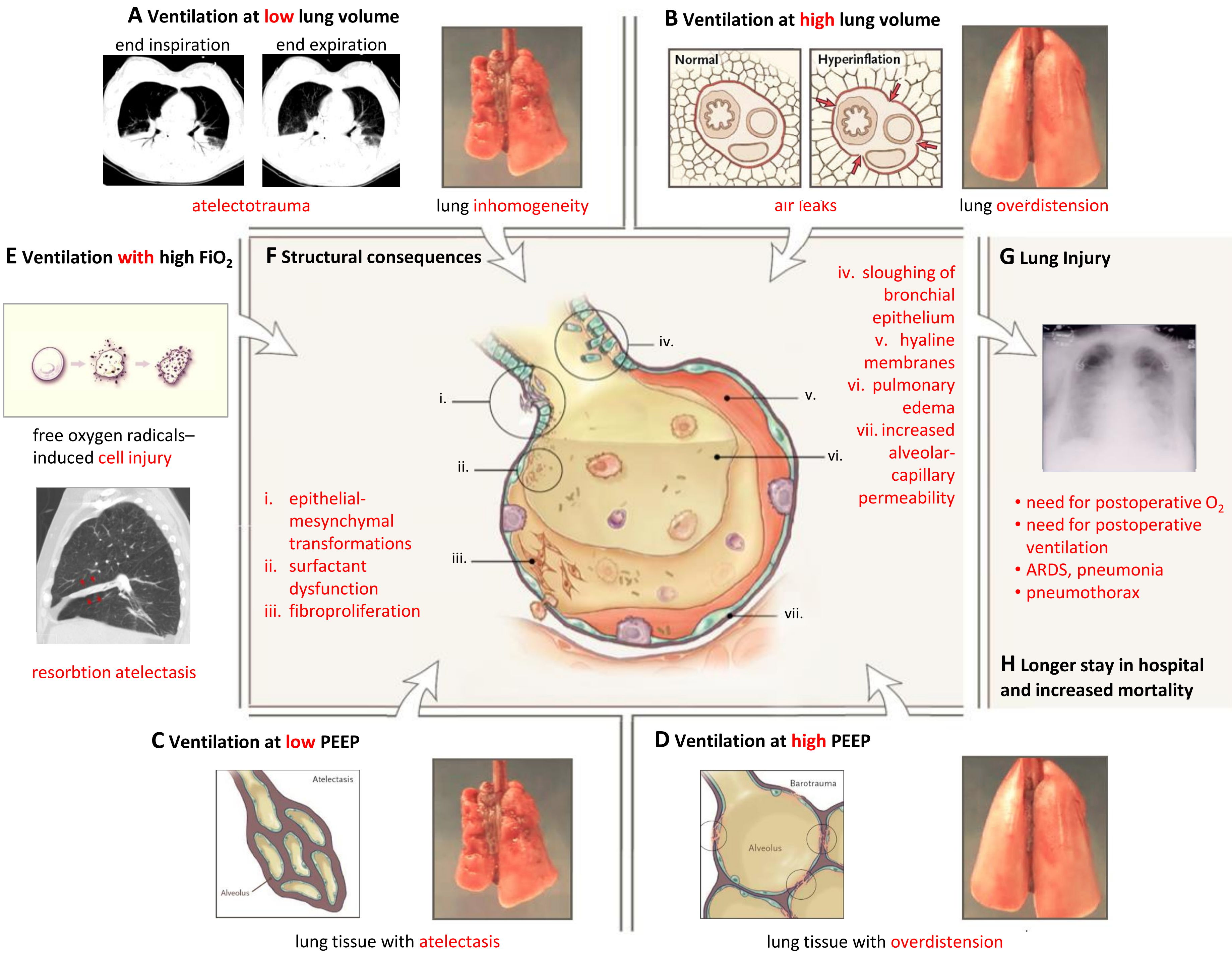
CRITICAL CARE MEDICINE

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.




A Ventilation at low lung volume
end inspiration
end expiration
atelectotrauma
lung inhomogeneity
B Ventilation at high lung volume
Normal
Hyperinflation
air leaks
lung overdistension
E Ventilation with high FiO_2
free oxygen radicals–induced cell injury
resorbtion atelectasis
F Structural consequences
i. epithelial-mesynchymal transformations
ii. surfactant dysfunction
iii. fibroproliferation
iv. sloughing of bronchial epithelium
v. hyaline membranes
vi. pulmonary edema
vii. increased alveolar-capillary permeability
i.
ii.
iii.
iv.
v.
vi.
vii.
G Lung Injury
• need for postoperative O_2
• need for postoperative ventilation
• ARDS, pneumonia
• pneumothorax
H Longer stay in hospital and increased mortality
C Ventilation at low PEEP
Atelectasis
Alveolus
lung tissue with atelectasis
D Ventilation at high PEEP
Barotrauma
Alveolus
lung tissue with overdistension

EDITORIAL

Happy 50th birthday ARDS!

Arthur S. Slutsky[1,2*], Jesús Villar[1,3,4] and Antonio Pesenti[5,6]

Intensive Care Med (2016) 42:637–639

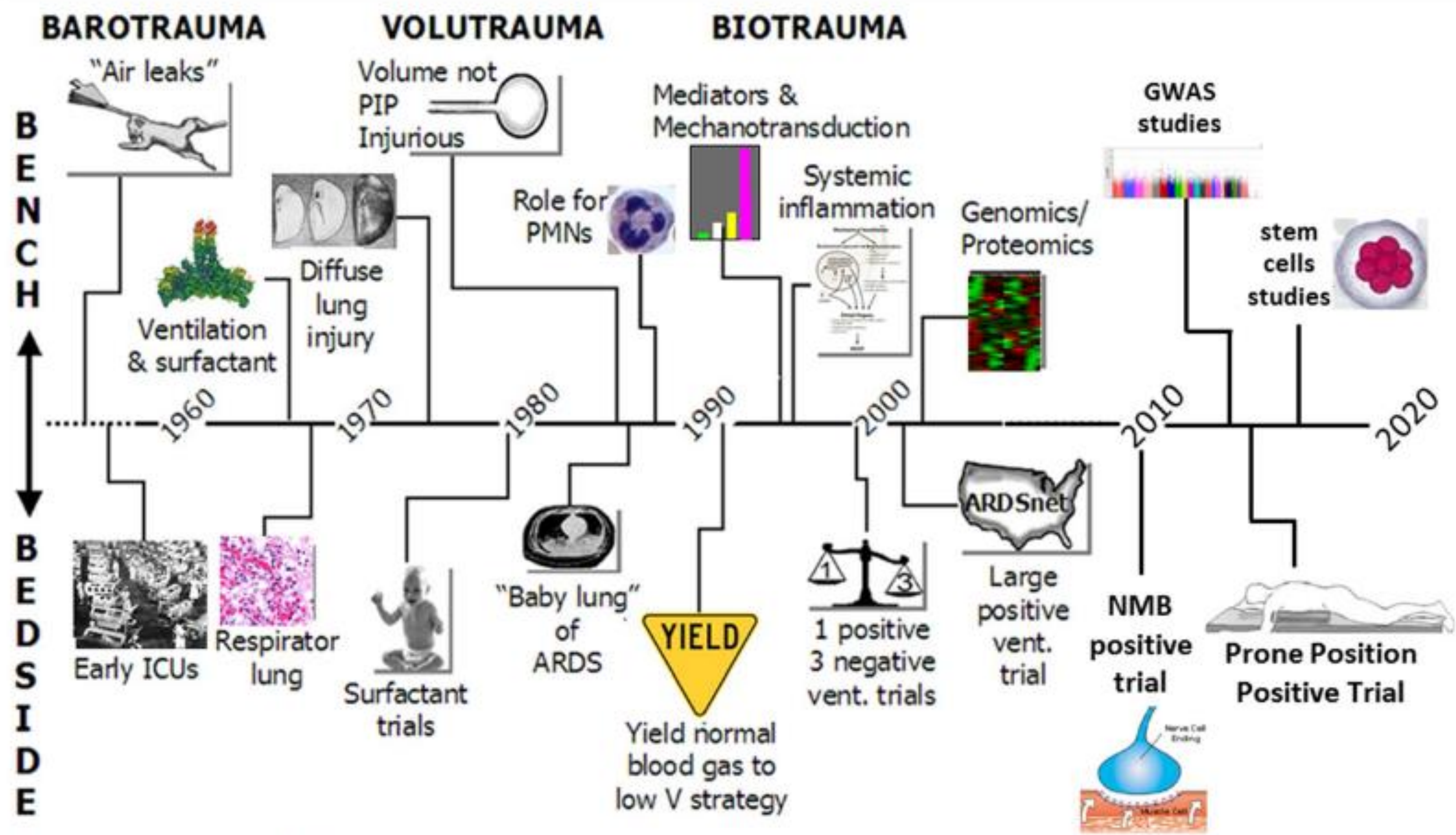


Fig. 1 Major advances related to the acute respiratory distress syndrome (ARDS) and ventilator-induced lung injury (VILI): from the bench to the bedside. *GWAS* genome-wide association studies, *ICU* intensive care unit, *NMB* neuromuscular blocking agents, *PIP* peak inspiratory pressure, *PMN* polymorphonuclear cells, *V* volume, *vent.* ventilation. (Modified from [21])

Among the interventions that have been tested in (**severe**) ARDS, only **3** have proven beneficial to patient survival (RCT) :

1) Low tidal volume (VT = 6 ml/kg PBW)

ARMA study-NEJM 2000

2) Prone Position

PROSEVA study-NEJM 2013

3) Myorelaxants (NMBA)

ACURASYS study-NEJM 2010

Objectifs : SDRA 2016

1. Pourquoi une nouvelle définition du SDRA ?

2. Importance de l'hémodynamique au cours SDRA ?

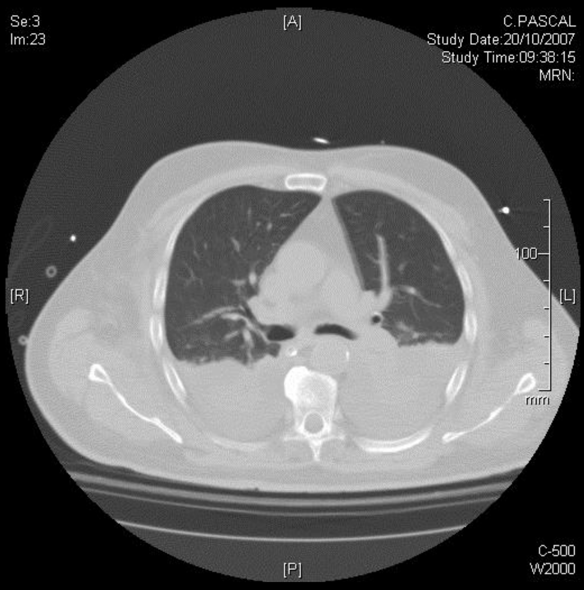
3. Peut on prévenir la survenue du SDRA ?

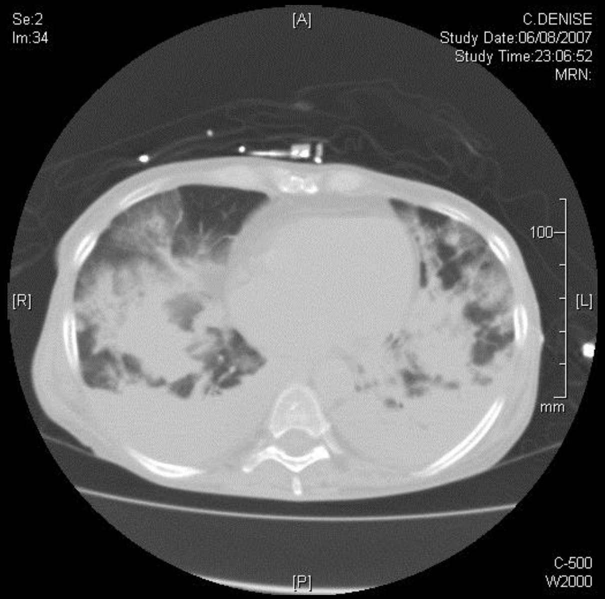
4. Le Décubitus Ventral : faut-il l'appliquer, comment et à qui ?

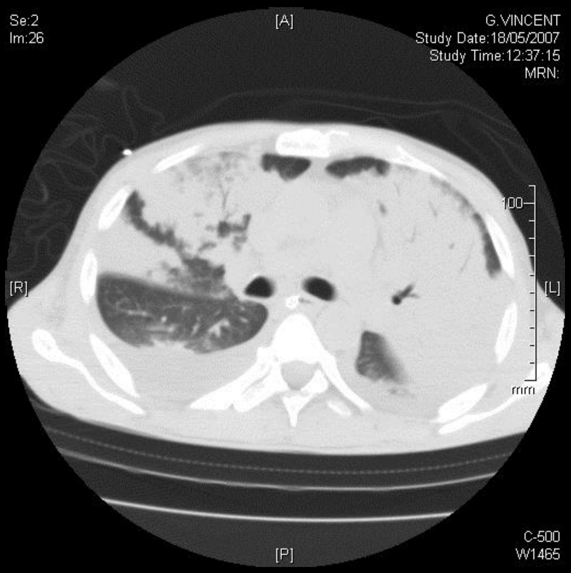
5. Importance de la sédation ? Et/ou des curares ?

6. synthèse

Est-ce bien un SDRA ?







The Conceptual Model Of ARDS

1. ARDS is a type of acute diffuse lung injury associated with recognized risk factors, characterized by inflammation leading to increased pulmonary vascular permeability and loss of aerated lung tissue.

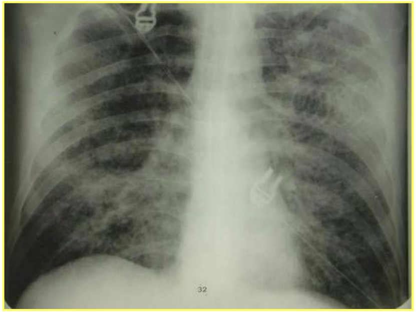
2. The hallmarks of the clinical syndrome are hypoxemia and bilateral radiographic opacities (standard chest x-ray or CT scan),

The Conceptual Model Of ARDS

3. ARDS is associated with recognized risk factors and is characterized by inflammation leading to increased pulmonary vascular permeability and loss of aerated lung tissue.
4. Physiological derangements include increased pulmonary venous admixture, increased physiological dead-space, decreased respiratory system compliance.
5. Morphological hallmarks are lung edema, inflammation, hyaline membrane, and alveolar hemorrhage (i.e., diffuse alveolar damage)

Consensus conference (AJRCCM 1994)

4 criteria :



1. Acute time course
2. Bilateral pulmonary infiltrates
3. Absence of cardiogenic pulmonary edema, hypervolemia or PCWP < 18 mmHg
4. PaO2/FIO2 < 200 mmHg : ARDS

   < 300 mmHg : ALI (Acute Lung Injury) (**with or without PEEP)**

The American-European Consensus Conference on ARDS

Definitions, Mechanisms, Relevant Outcomes, and Clinical Trial Coordination

GORDON R. BERNARD, ANTONIO ARTIGAS, KENNETH L. BRIGHAM, JEAN CARLET, KONRAD FALKE, LEONARD HUDSON, MAURICE LAMY, JEAN ROGER LEGALL, ALAN MORRIS, ROGER SPRAGG, and the Consensus Committee

	Timing	Oxygenation	Chest Radiograph	Pulmonary Artery Wedge pressure
ALI	Acute onset	≤ 300 (regardless of PEEP)	Bilateral infiltrates	≤18 mmHg/no evidence of left atrial hypertension
ARDS	Acute onset	≤ 200 regardless of PEEP	Bilateral infiltrates	≤ 18 mmHg or no evidence of left atrial hypertension

Am J Respir Crit Care Med 1994;149:818–24.

2012 « the Berlin Definition »

JAMA 2012

– Groupe d'experts internationaux
– Critères évalués sur des cohortes de patients

	Léger	**Modéré**	**Sévère**
Timing	Début aigu < 1 semaine d'un contexte clinique compatible, nouveau/aggravation de symptômes cliniques		
Hypoxémie (mmHg)	$200 < PaO_2/FiO_2 \leq 300$	$100 < PaO_2/FiO_2 < 200$	$PaO_2/FiO_2 \leq 100$
Origine de l'œdème	Défaillance respiratoire non expliquée essentiellement par une défaillance cardiaque ou une surcharge volémique		
Anomalies radiologiques	Opacités bilatérales Non expliquées par des épanchements, atélectasies ou nodules		

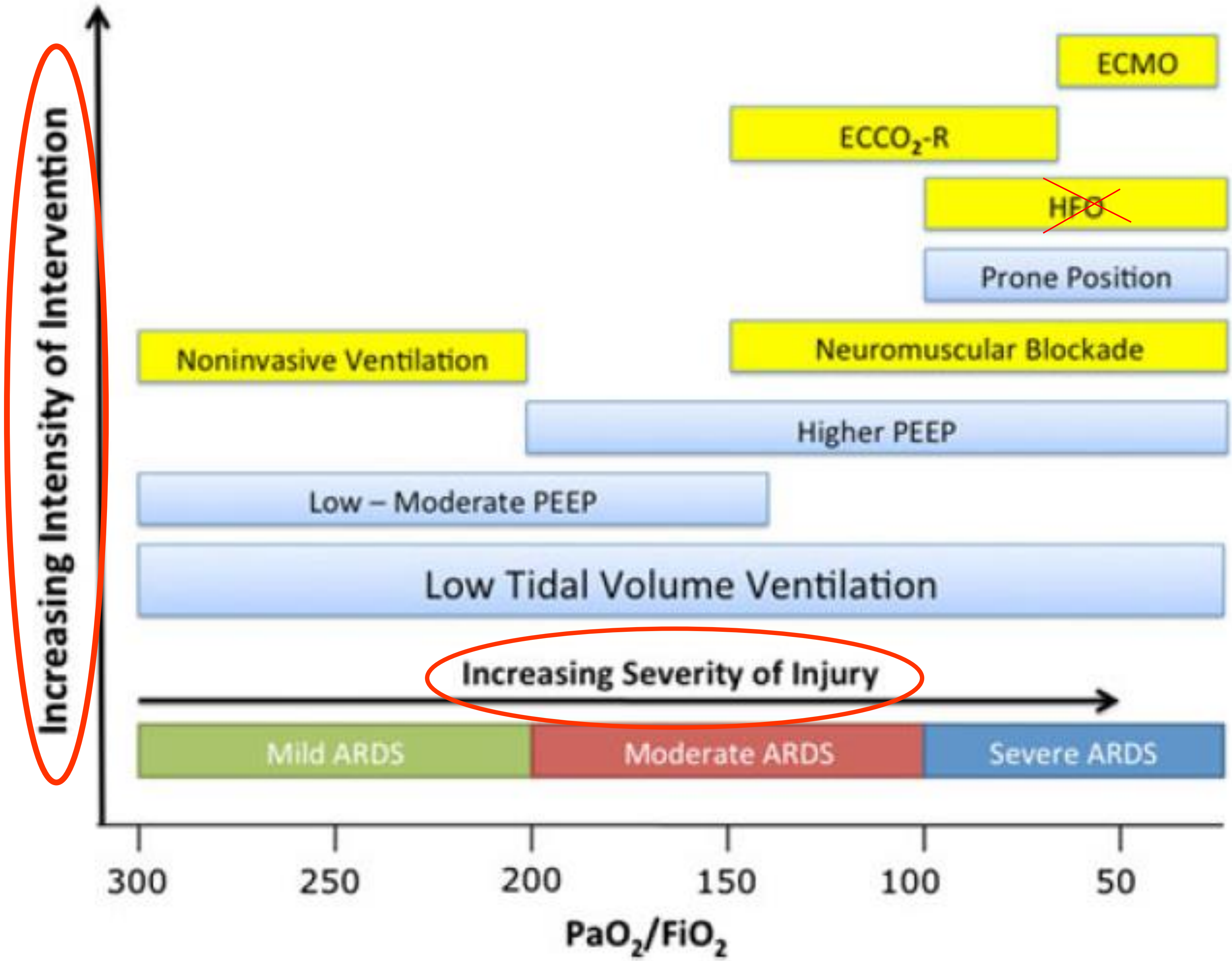
SPECIAL COMMUNICATION

ONLINE FIRST

Acute Respiratory Distress Syndrome

The Berlin Definition

The ARDS Definition Task Force*



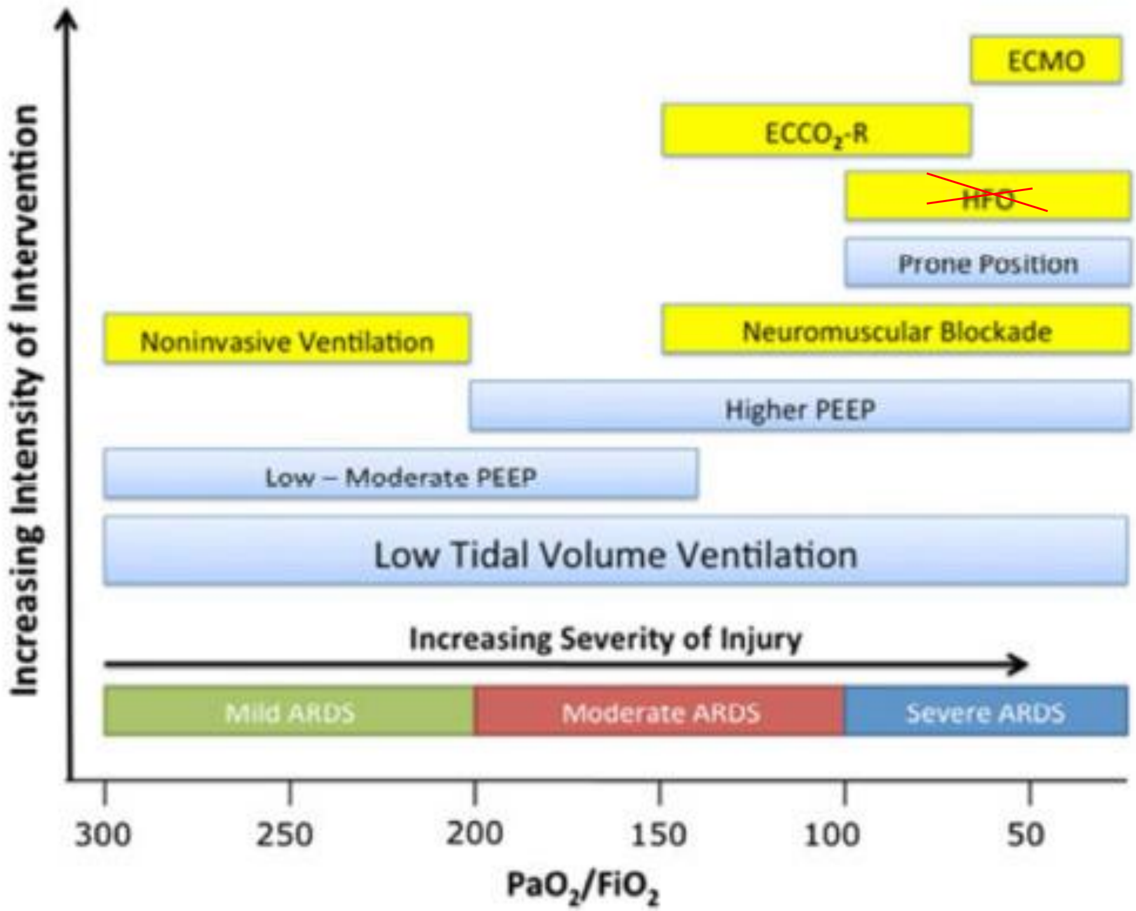
Increasing Intensity of Intervention
ECMO
$ECCO_2$-R
HFO
Prone Position
Noninvasive Ventilation
Neuromuscular Blockade
Higher PEEP
Low – Moderate PEEP
Low Tidal Volume Ventilation
Increasing Severity of Injury
Mild ARDS
Moderate ARDS
Severe ARDS
300
250
200
150
100
50
PaO_2/FiO_2

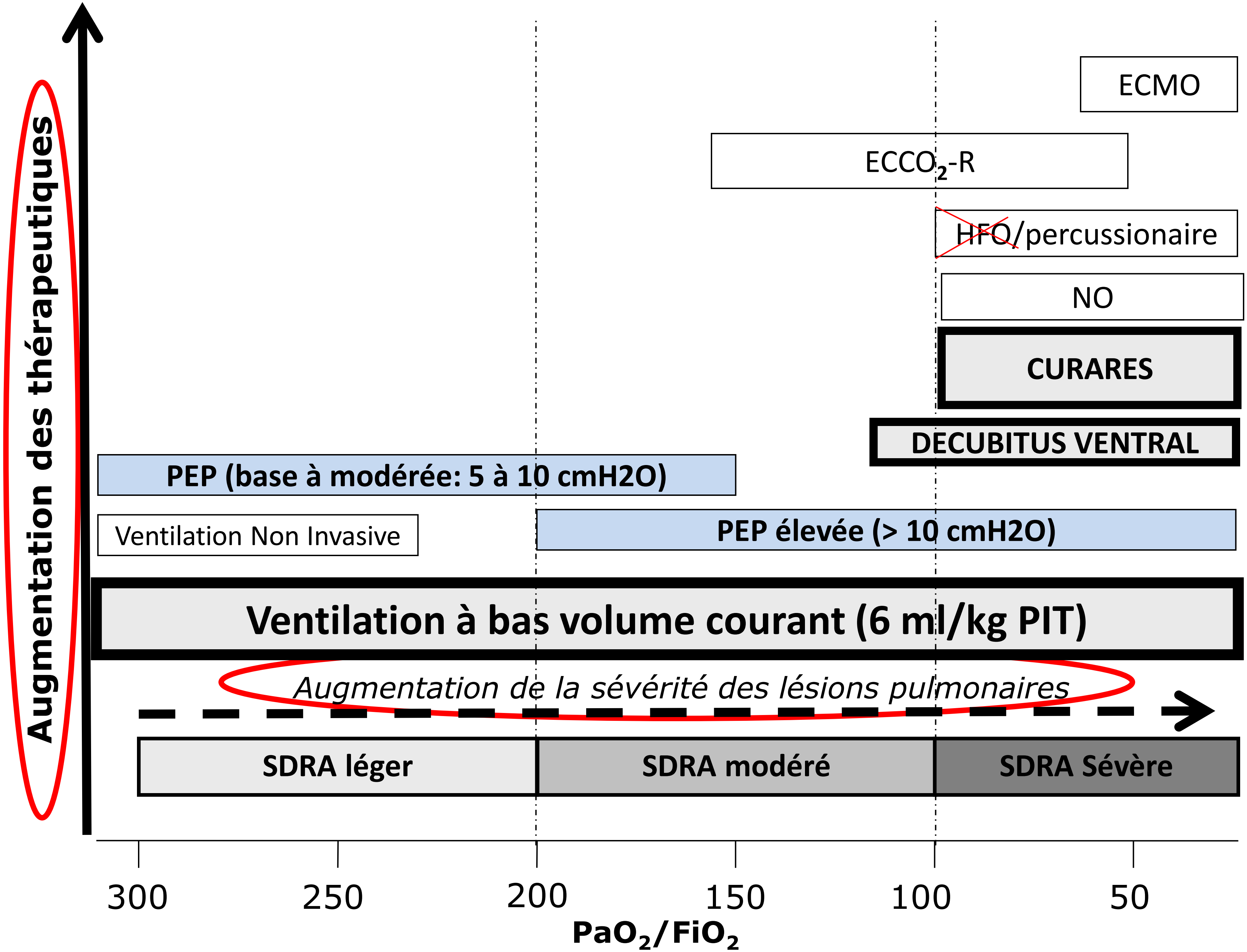
Intensive Care Med (2012) 38:1573–1582
DOI 10.1007/s00134-012-2682-1

SPECIAL ARTICLE

Niall D. Ferguson
Eddy Fan
Luigi Camporota
Massimo Antonelli
Antonio Anzueto
Richard Beale
Laurent Brochard
Roy Brower
Andrés Esteban
Luciano Gattinoni
Andrew Rhodes
Arthur S. Slutsky
Jean-Louis Vincent
Gordon D. Rubenfeld
B. Taylor Thompson
V. Marco Ranieri

The Berlin definition of ARDS: an expanded rationale, justification, and supplementary material




Augmentation des thérapeutiques
ECMO
$ECCO_2$-R
HFO/percussionaire
NO
CURARES
DECUBITUS VENTRAL
PEP (base à modérée: 5 à 10 cmH2O)
Ventilation Non Invasive
PEP élevée (> 10 cmH2O)
Ventilation à bas volume courant (6 ml/kg PIT)
Augmentation de la sévérité des lésions pulmonaires
SDRA léger
SDRA modéré
SDRA Sévère
300
250
200
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100
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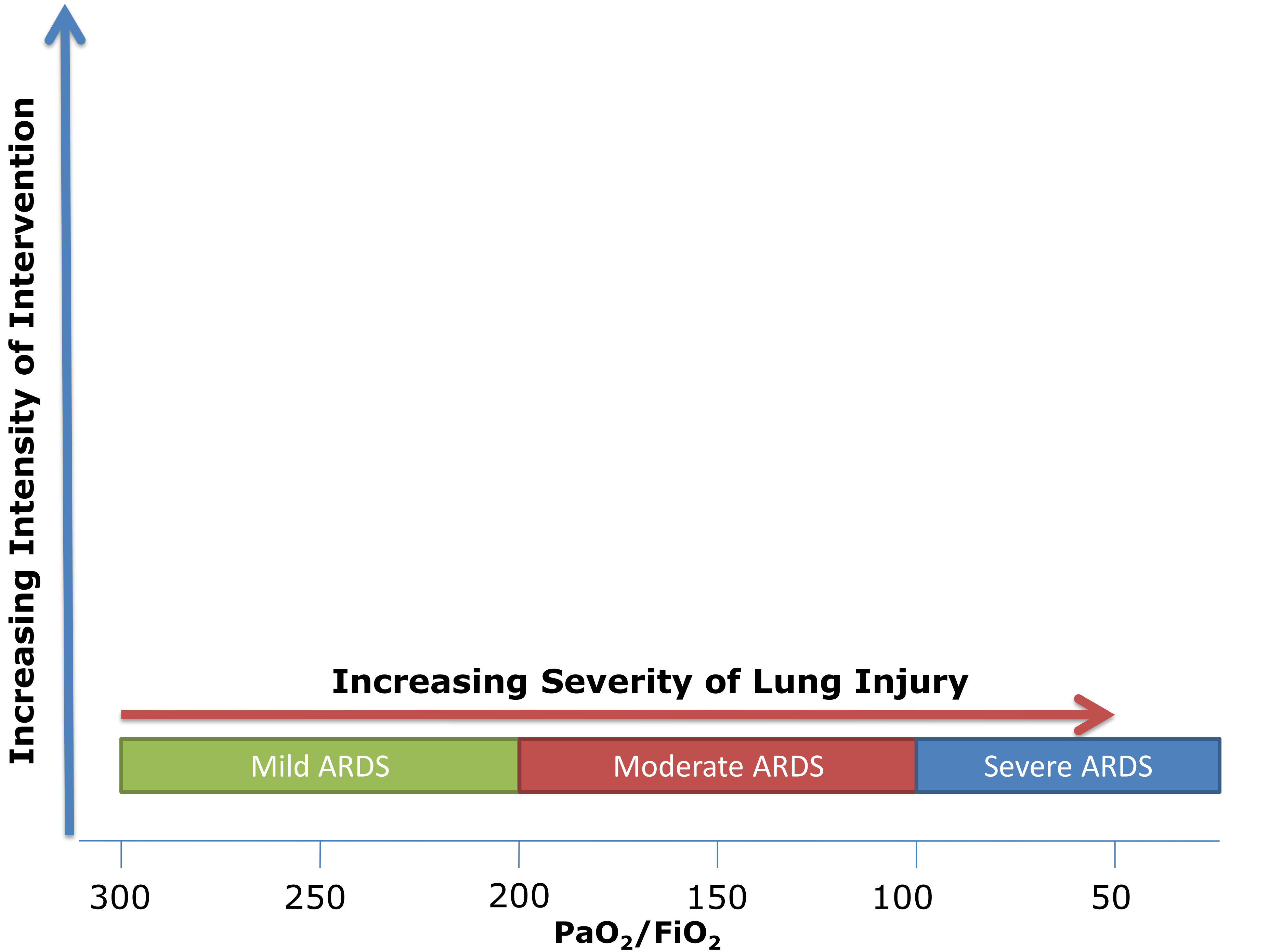
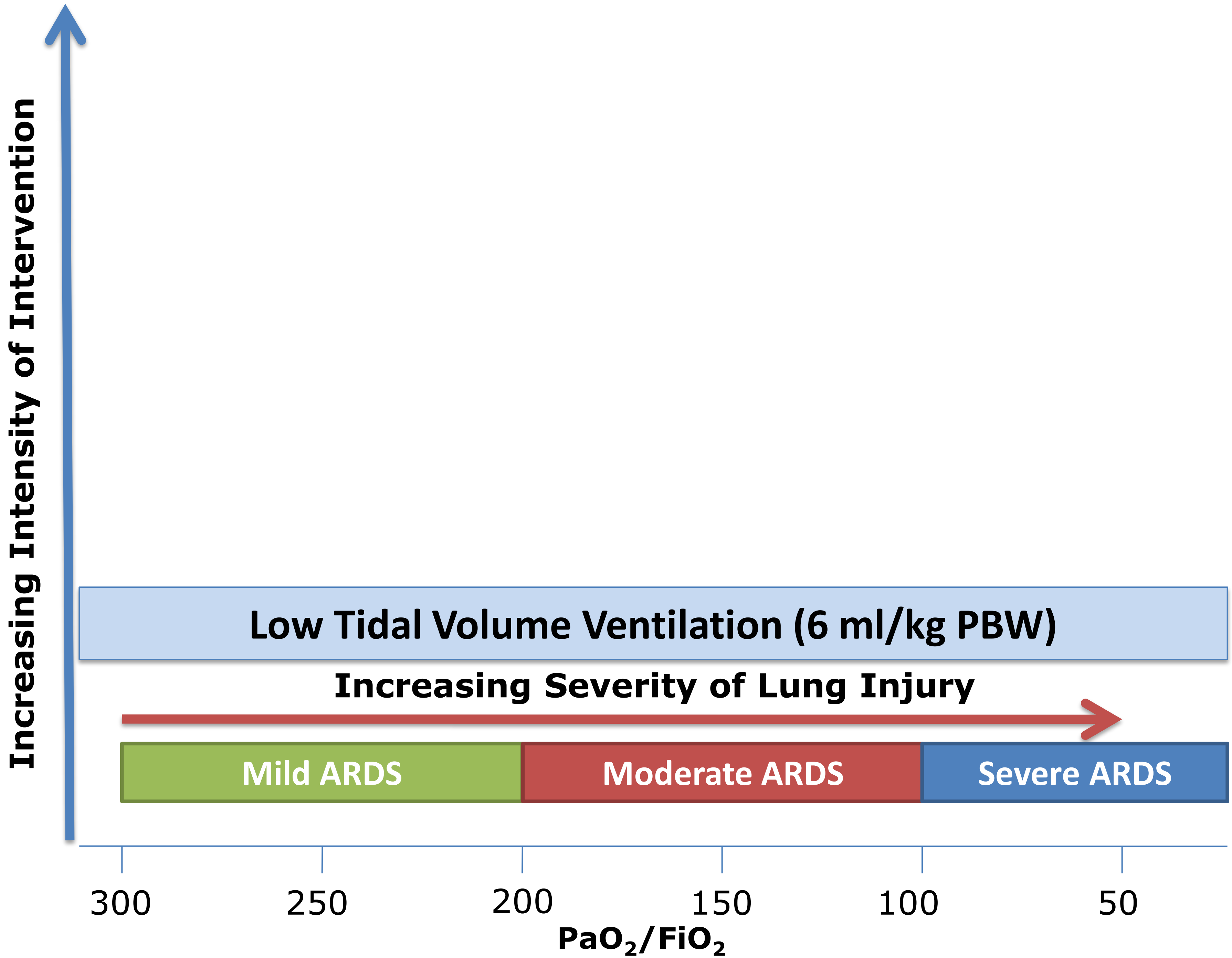
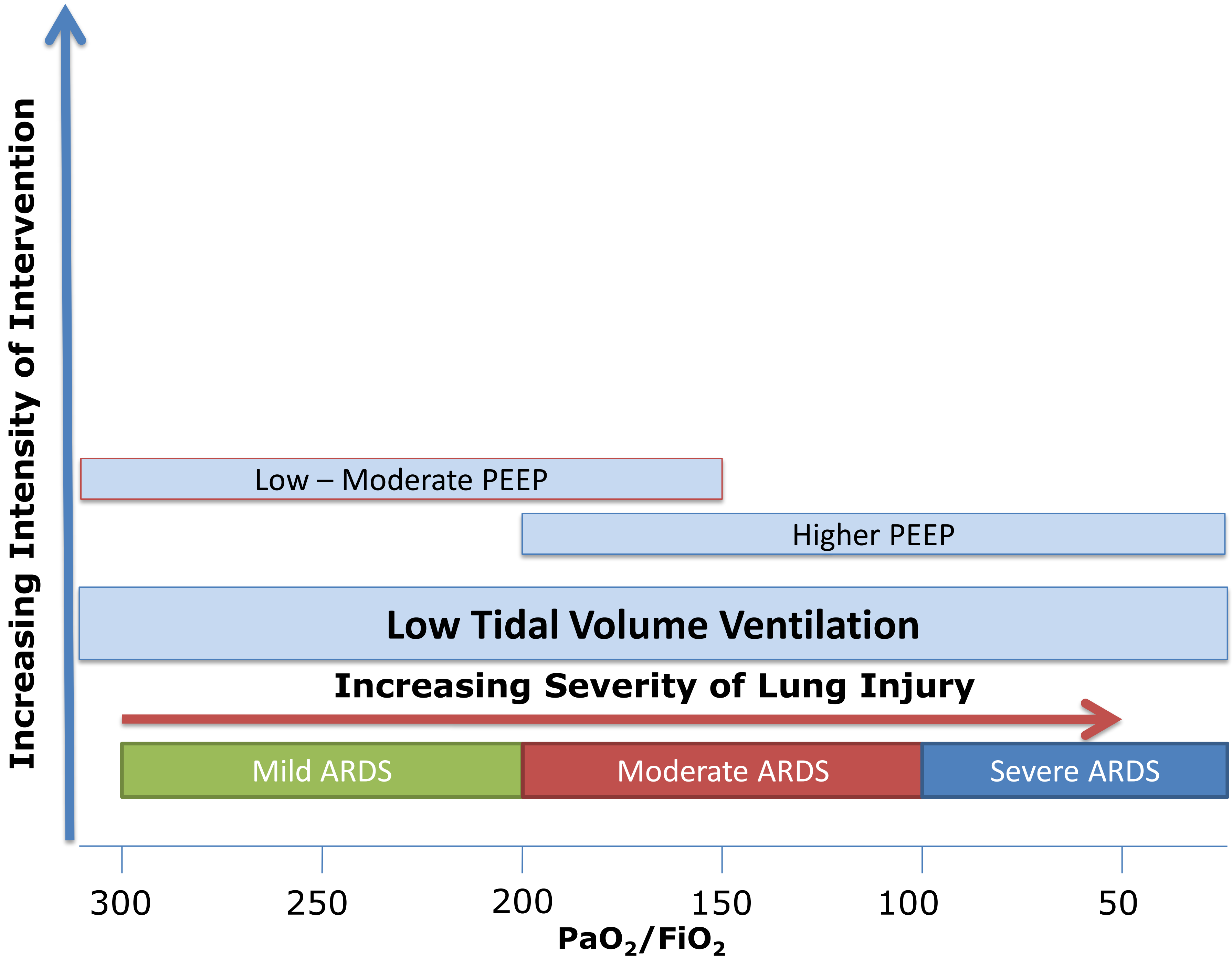
PaO_2/FiO_2


Increasing Intensity of Intervention
Increasing Severity of Lung Injury
Mild ARDS
Moderate ARDS
Severe ARDS
300
250
200
150
100
50
PaO2/FiO2



Increasing Intensity of Intervention
Low Tidal Volume Ventilation (6 ml/kg PBW)
Increasing Severity of Lung Injury
Mild ARDS
Moderate ARDS
Severe ARDS
300
250
200
150
100
50
PaO_2/FiO_2



Increasing Intensity of Intervention
Low – Moderate PEEP
Higher PEEP
Low Tidal Volume Ventilation
Increasing Severity of Lung Injury
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300
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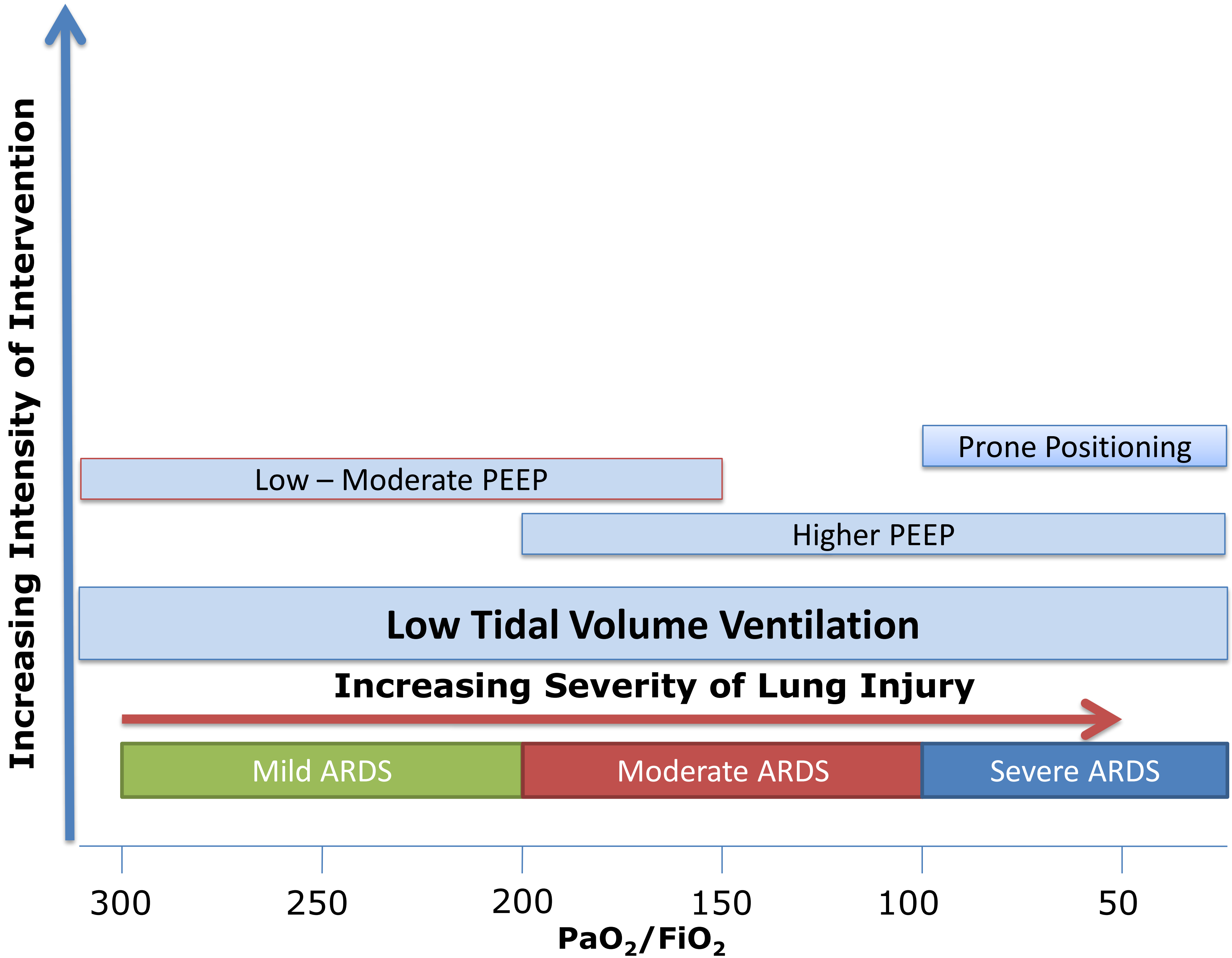
PaO_2/FiO_2


Increasing Intensity of Intervention
Prone Positioning
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Severe ARDS
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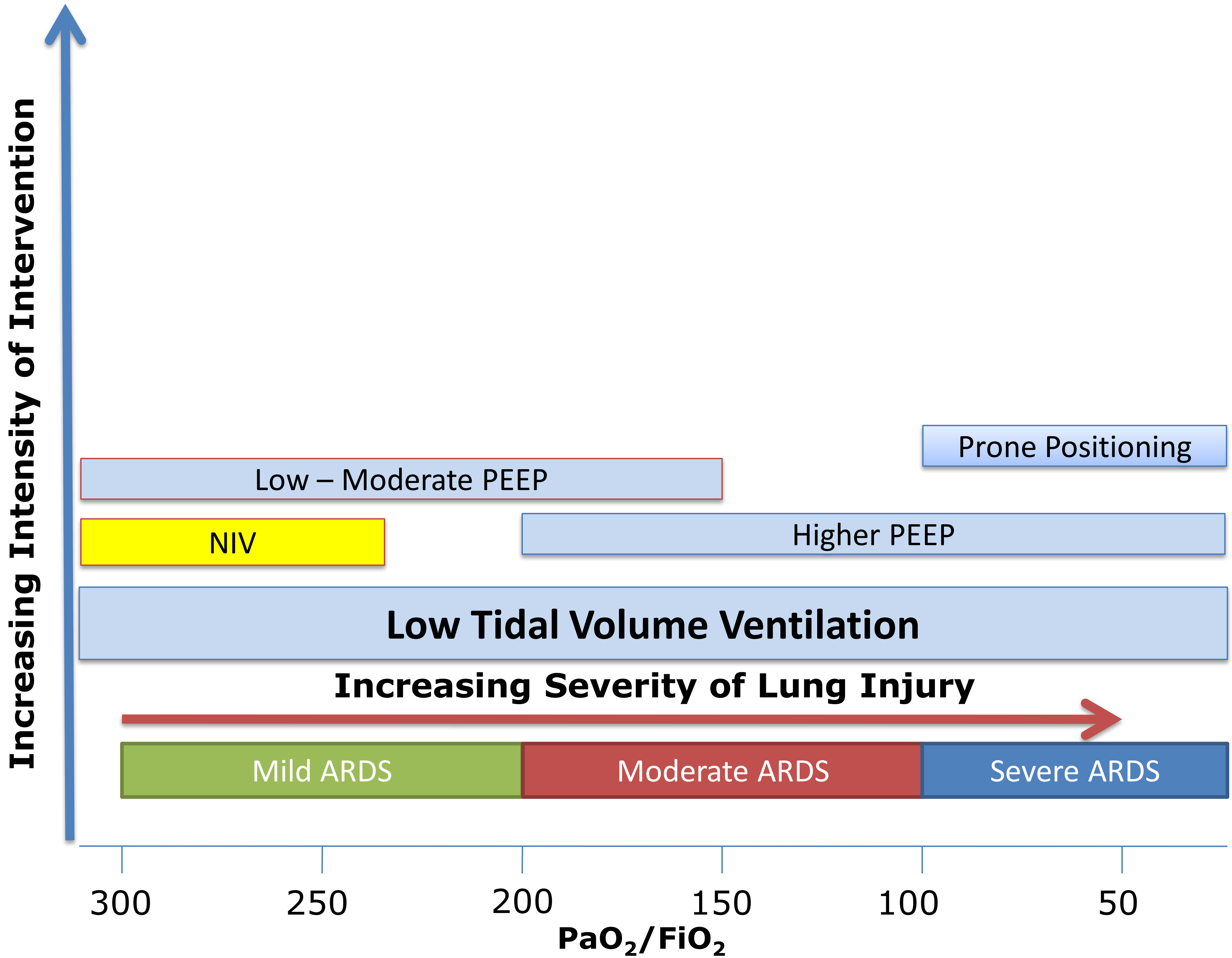
250
200
150
100
50
PaO2/FiO2



Increasing Intensity of Intervention
Prone Positioning
Low – Moderate PEEP
NIV
Higher PEEP
Low Tidal Volume Ventilation
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Severe ARDS
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200
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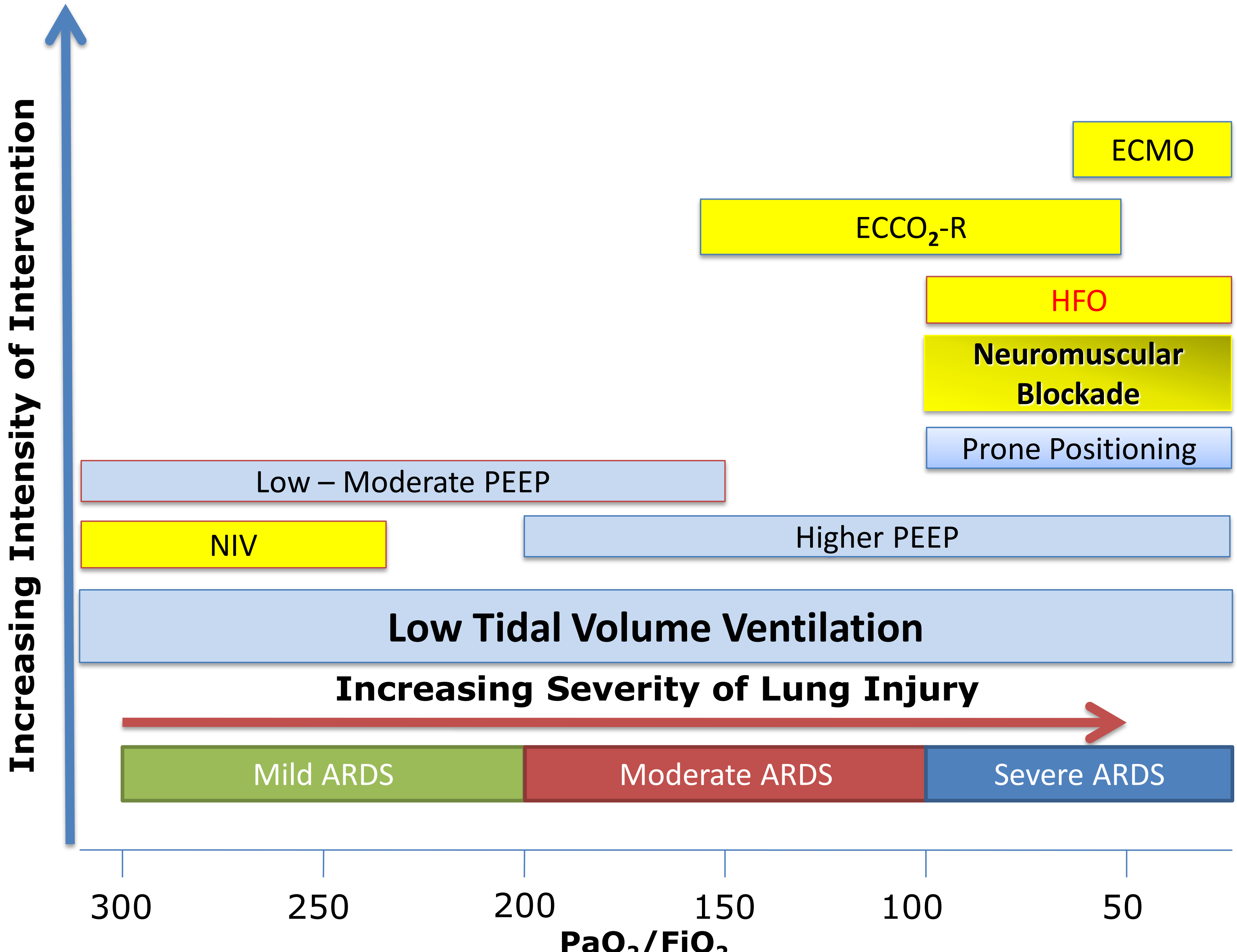
100
50
PaO_2/FiO_2



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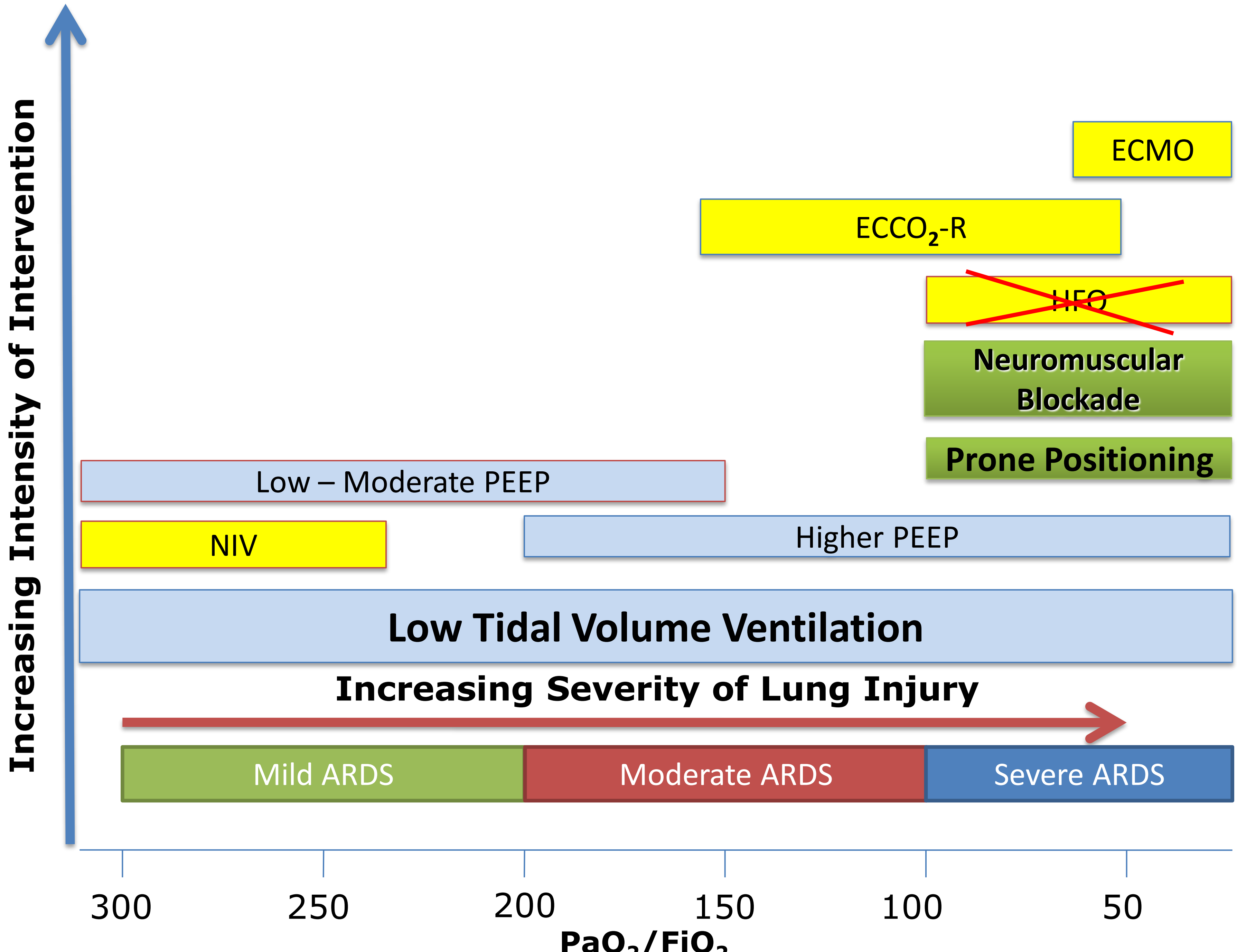
PaO2/FiO2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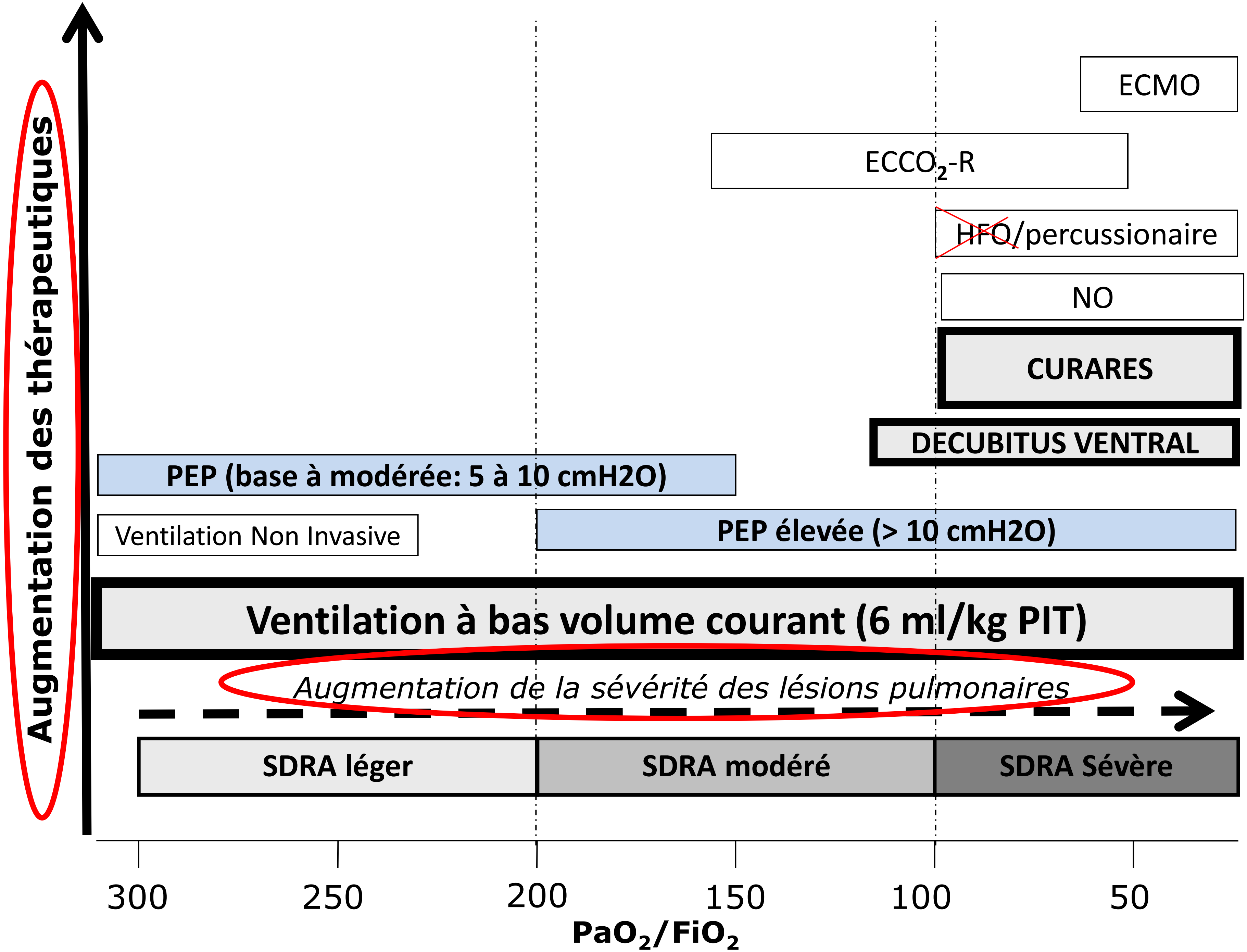
PaO$_2$/FiO$_2$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SDRA léger
SDRA modéré
SDRA Sévère
300
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PaO2/FiO2

Les 10 (5+5) **points clés à savoir pour la ventilation du SDRA**

1. Quel mode ventilatoire ?
2. Quelle Fréquence Respiratoire (FR) ?
3. Quelle Volume Courant (VT)?
4. Quelle Pression Expiratoire Positive (PEP) ?
5. Quelle Fraction Inspiratoire d'oxygène (FiO2) ?

---

6. Quel Temps inspiratoire (insufflation) et rapport I/E ?
7. Quel Temps de pause (Tp) ?
8. Quel débit inspiratoire (insufflation) ?
9. Quelle Manœuvre de recrutement (si besoin) ?
10. Quelles Pression de Plateau et/ou Pression motrice $_{(max)}$?

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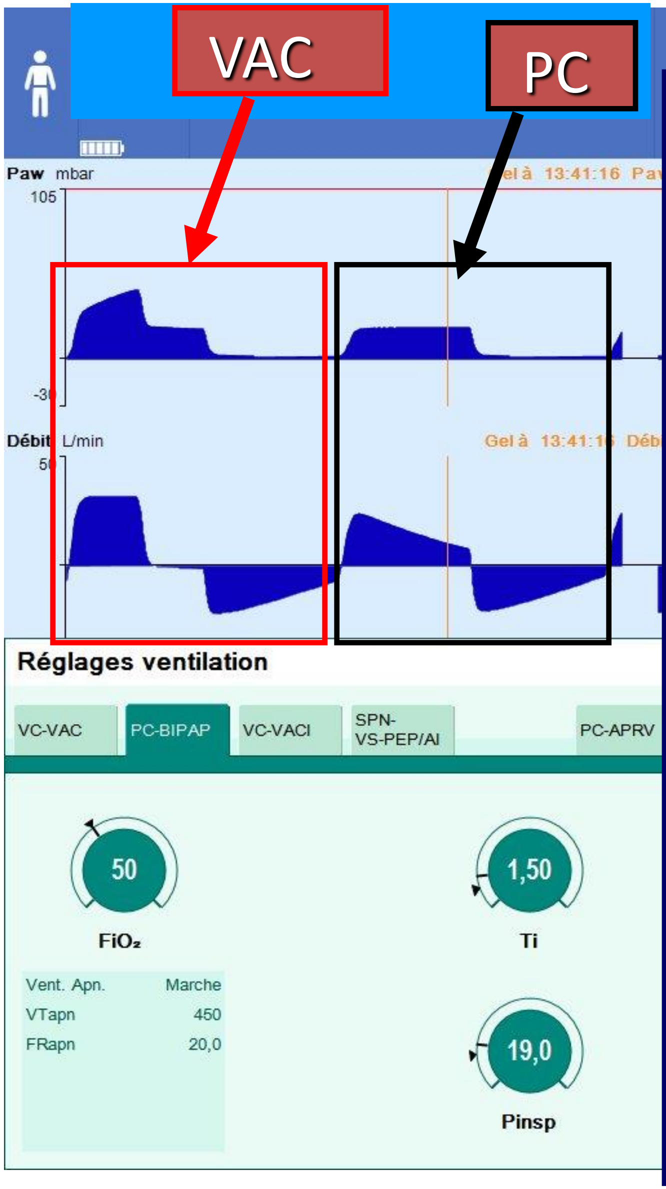
Quel mode ventilatoire au cours du SDRA ?

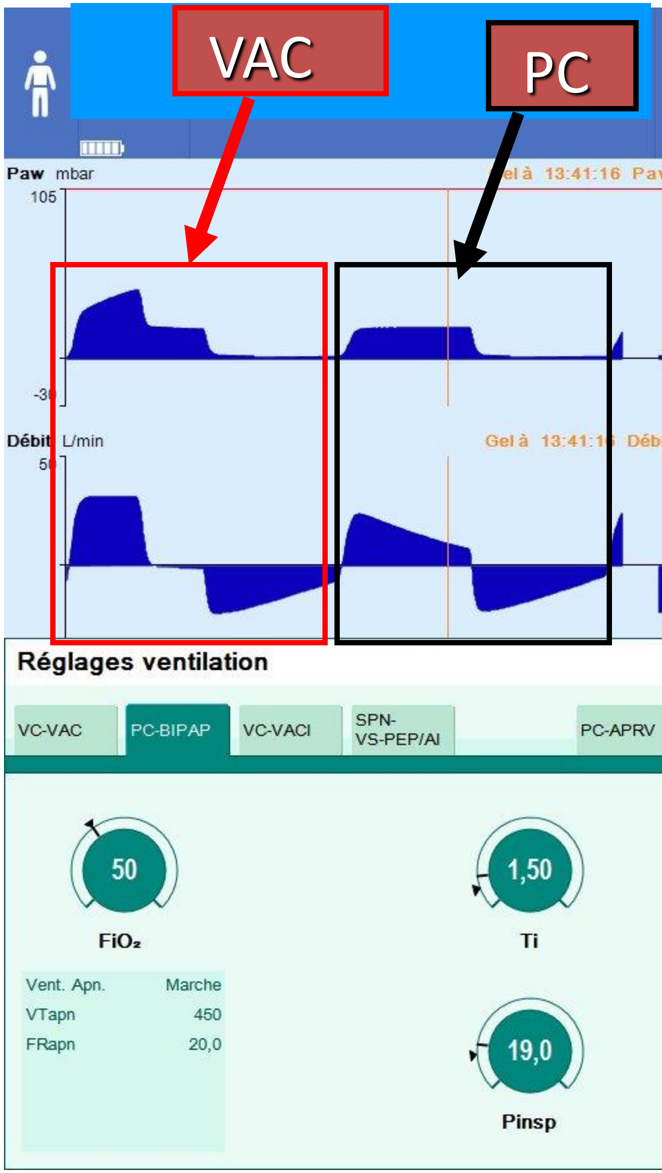
Quelle est la principale différence entre les 2 cycles respiratoires ?



1. Le premier cycle est en volume (débit carré) et le second en pression (débit décélérant)
2. Le capteur de débit inversé
3. L'expiration est différente car ça bloque !
4. Le premier cycle est en pression et le second en volume
5. Aucune

Question N°1

Quelle est la principale différence entre les 2 cycles respiratoires ?



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2. Le capteur de débit inversé
3. L'expiration est différente car ça bloque !
4. Le premier cycle est en pression et le second en volume
5. Aucune

Question-Réponse N°1

Quel mode ventilatoire au cours du SDRA ?

Volume = Pression

(Si niveau d'assistance équivalent)

Surveillance
Pression++

Surveillance
Volume ++

Les 10 (5+5) points clés à savoir pour la ventilation du SDRA

1. Quel mode ventilatoire ?
2. **Quelle Fréquence Respiratoire (FR) ?**
3. Quelle Volume Courant (VT)?
4. Quelle Pression Expiratoire Positive (PEP) ?
5. Quelle Fraction Inspiratoire d'oxygène (FiO2) ?

---

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Quelle Fréquence Respiratoire (FR) au cours du SDRA ?

Optimisation de la FR

objectif: compenser la baisse du VT, pour maintenir une ventilation minute (VE= FR x VT) efficace

Les 10 (5+5) points clés à savoir pour la ventilation du SDRA

1. Quel mode ventilatoire ?
2. Quelle Fréquence Respiratoire (FR) ?
3. **Quelle Volume Courant (VT)?**
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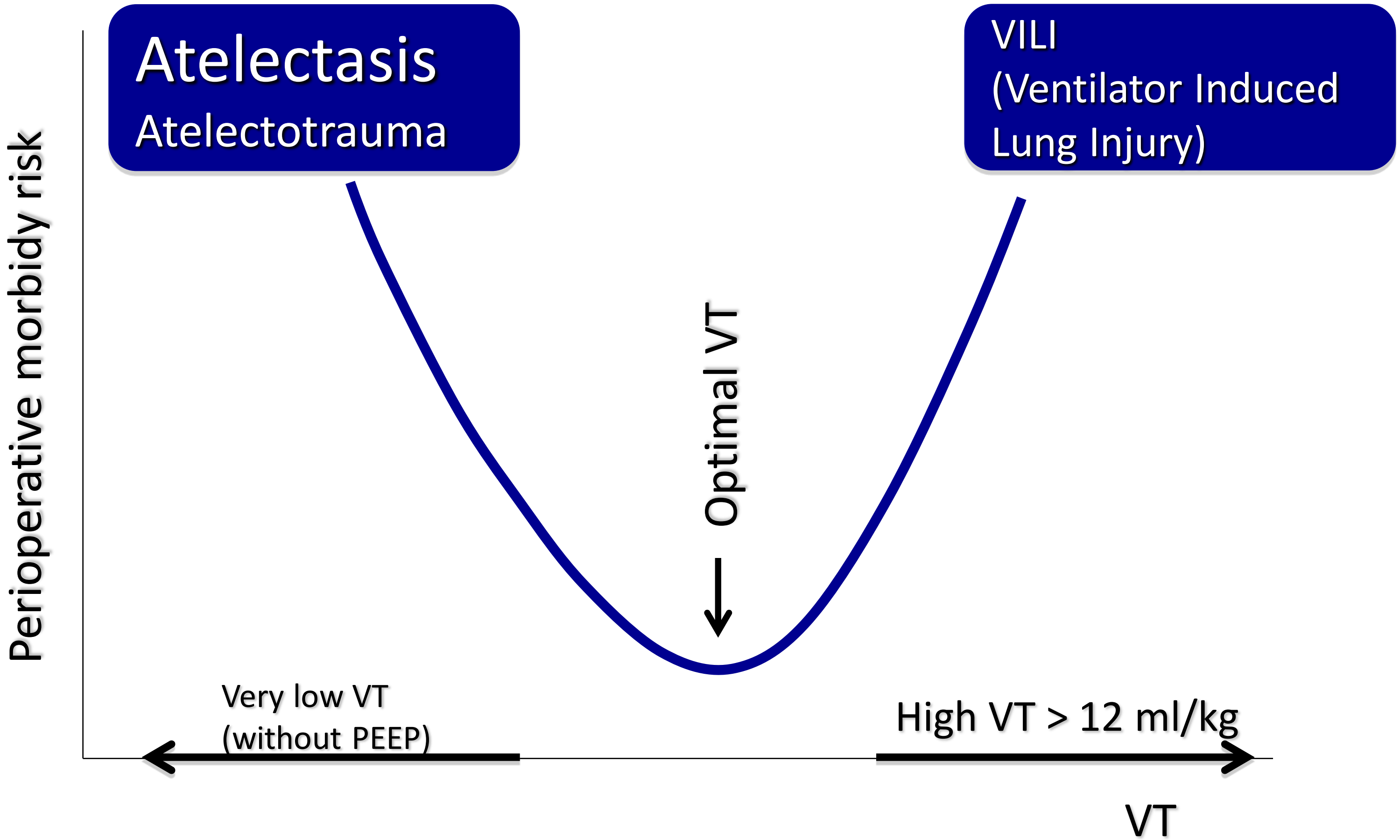
Quel volume courant (VT) au cours du SDRA ?

Tidal volume (VT) : how much is too much ?



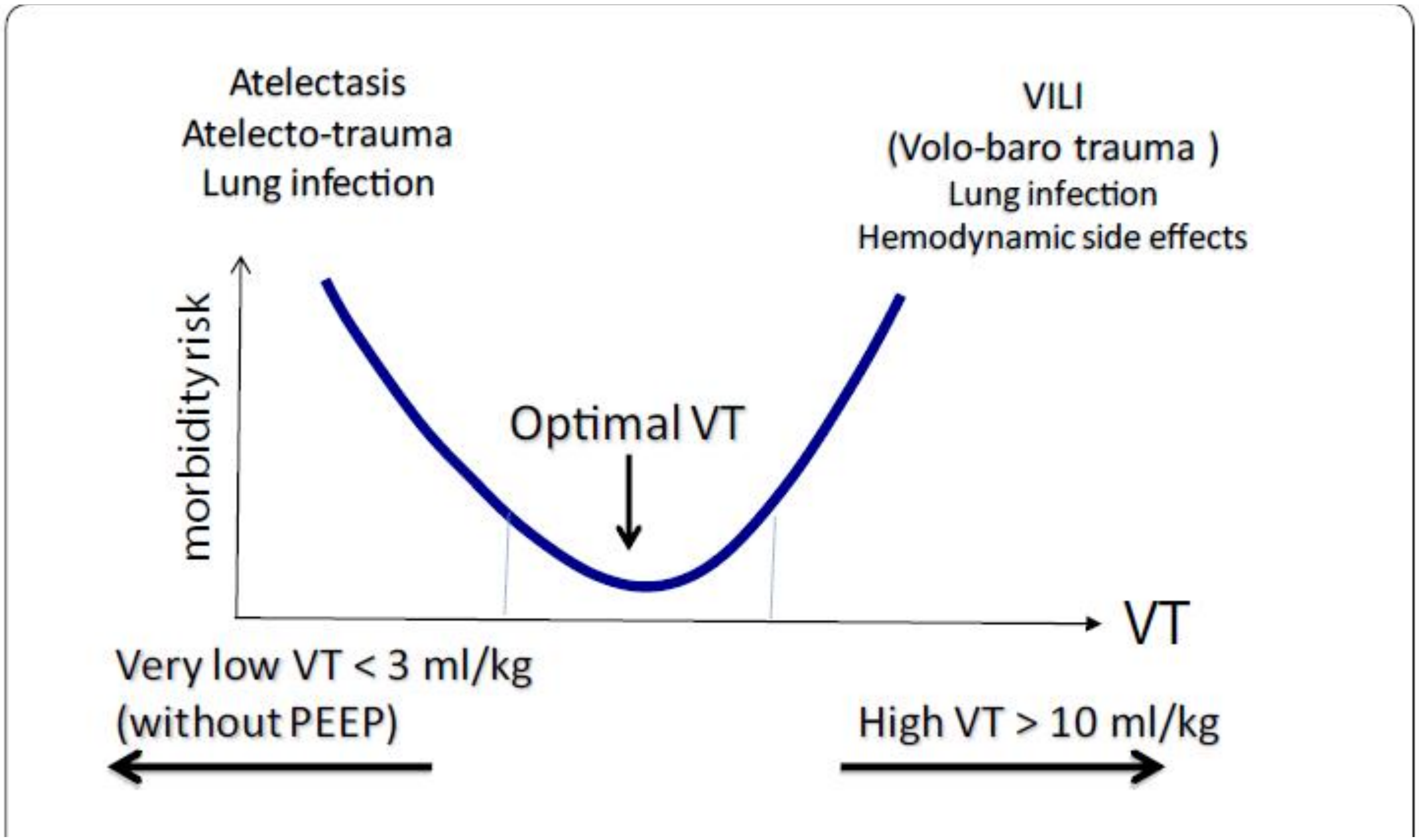
Quel volume courant (VT) au cours du SDRA ?

$4 < VT < 6$ ml/kg P.I.T

What's new in mechanical ventilation in patients without ARDS: lessons from the ARDS literature

Ary Serpa Neto[1,2,3] and Samir Jaber[4*]

4 < VT < 6 ml/kg



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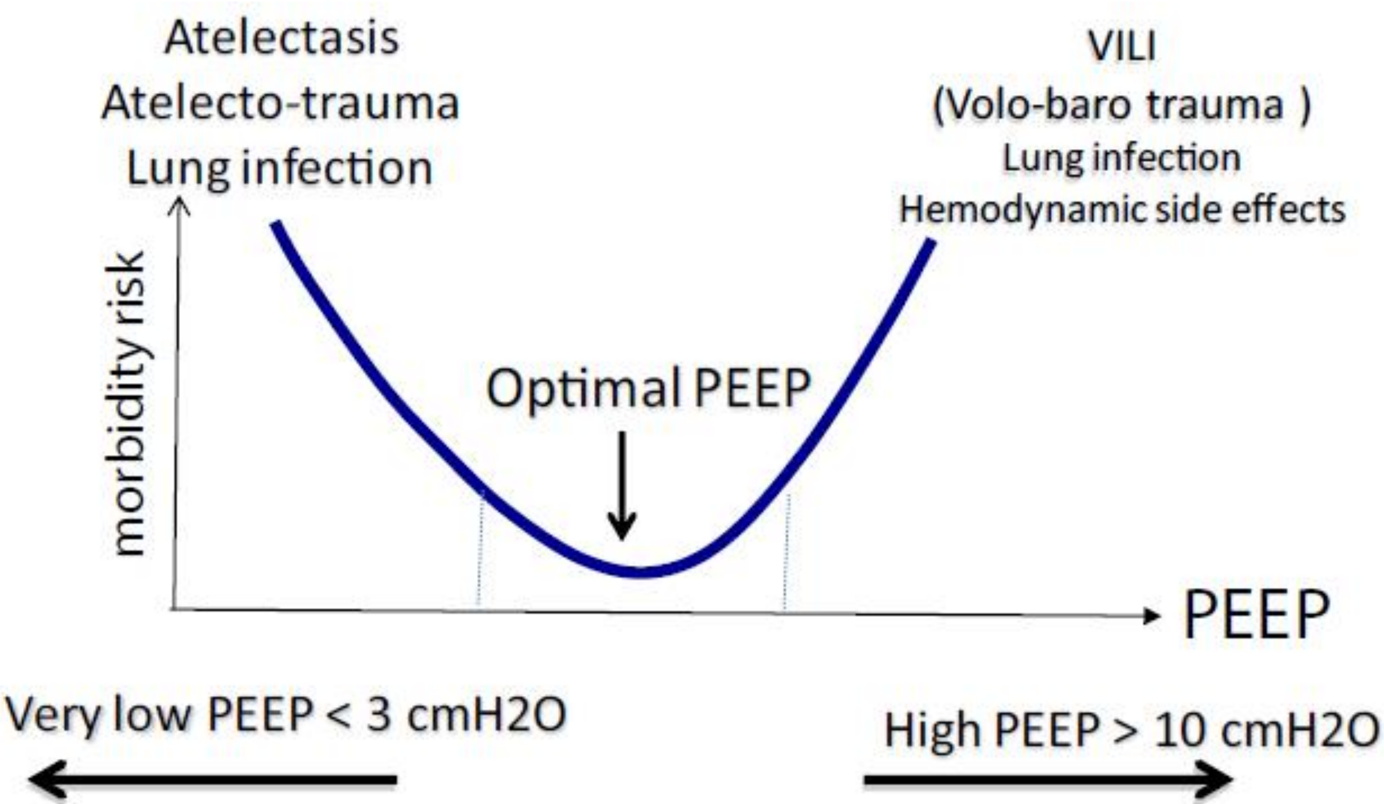
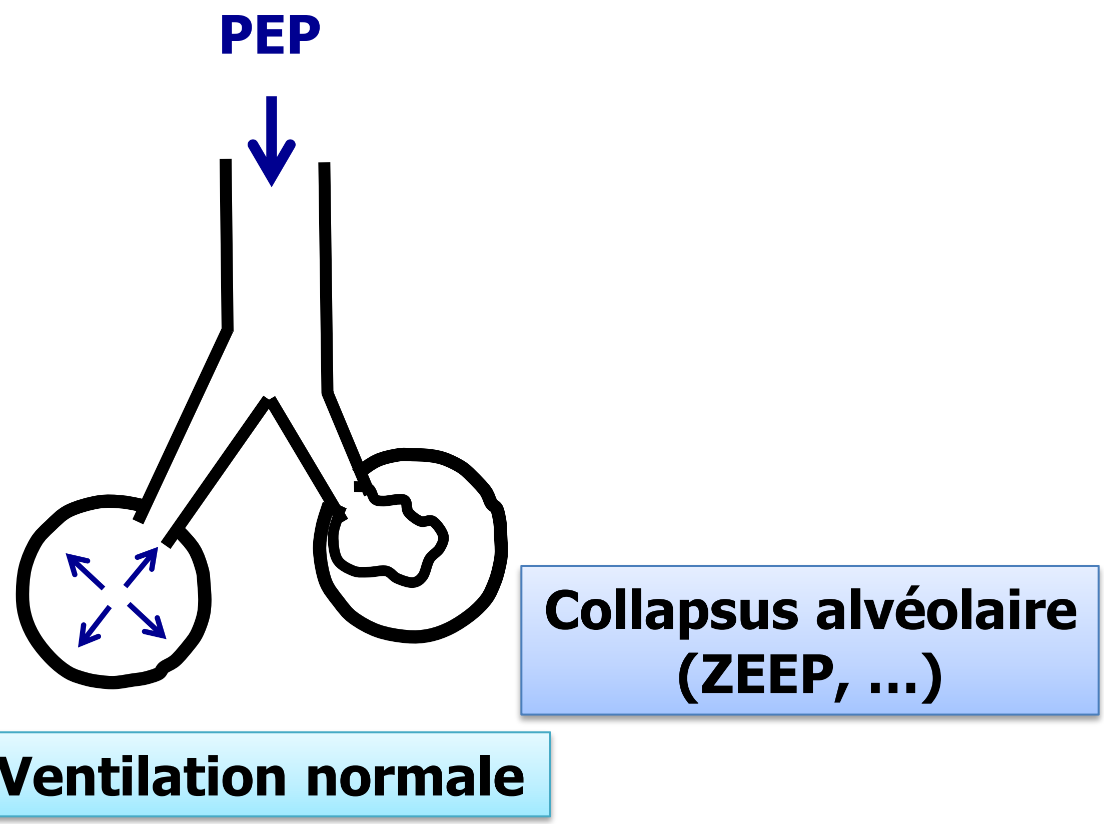
What's new in mechanical ventilation in patients without ARDS: lessons from the ARDS literature

Ary Serpa Neto[1,2,3] and Samir Jaber[4*]

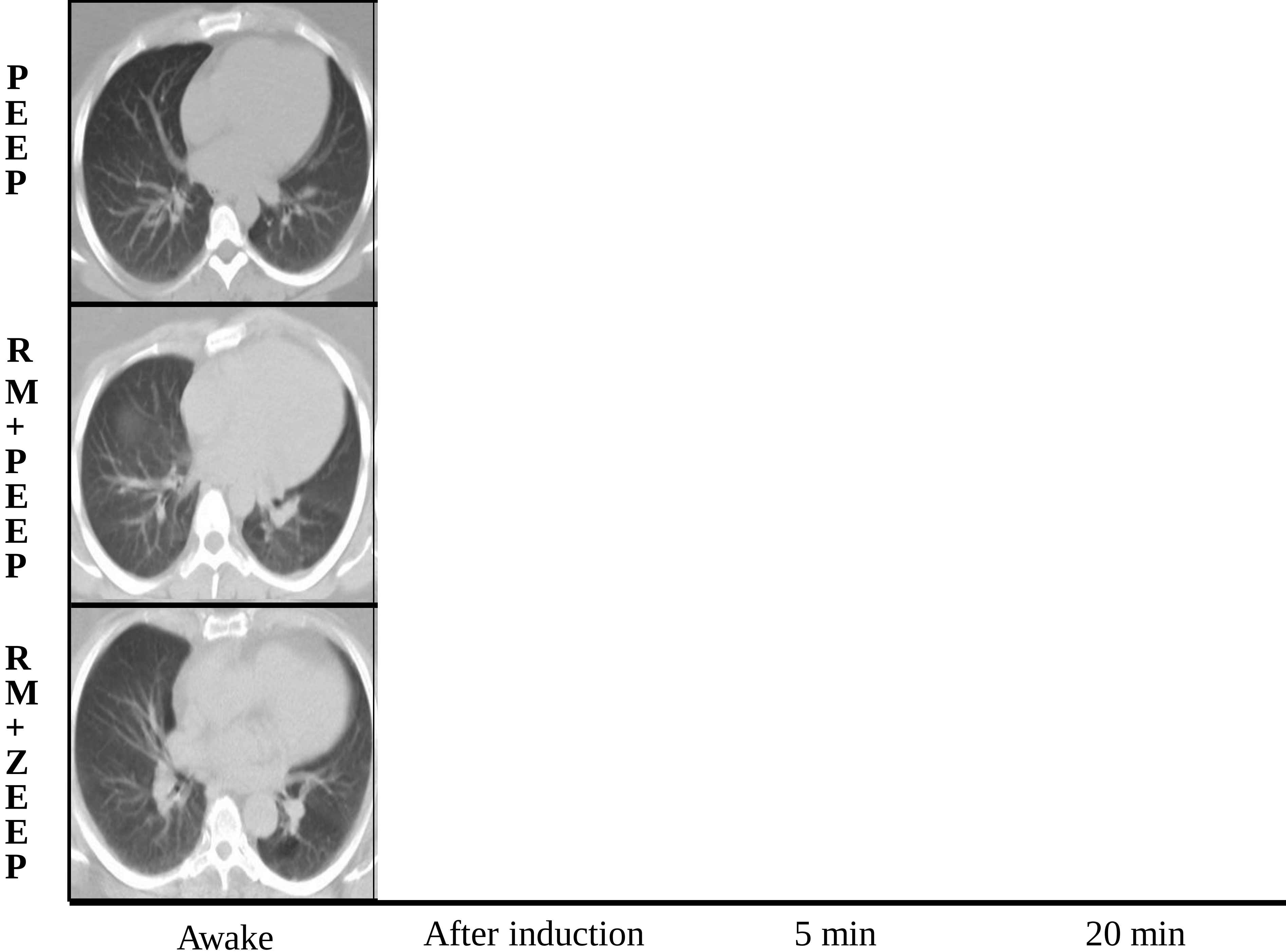
5 < PEEP < 15 cmH2O



Pourquoi la PEEP seule ne suffit pas toujours ?



Prévention de la perte d’aération (dé-recrutement) alvéolaire


PEEP
RM + PEEP
RM + ZEEP
Awake
After induction
5 min
20 min

Les 10 (5+5) points clés à savoir pour la ventilation du SDRA

1. Quel mode ventilatoire ?
2. Quelle Fréquence Respiratoire (FR) ?
3. Quelle Volume Courant (VT)?
4. Quelle Pression Expiratoire Positive (PEP) ?
5. **Quelle Fraction Inspiratoire d'oxygène (FiO2) ?**

---

6. Quel Temps inspiratoire (insufflation) et rapport I/E ?
7. Quel Temps de pause (Tp) ?
8. Quel débit inspiratoire (insufflation) ?
9. Quelle Manœuvre de recrutement (si besoin) ?
10. Quelles Pression de Plateau et/ou Pression motrice $_{(max)}$?

ARDS Network Trial of Higher PEEP

Arterial Oxygenation: SpO_2 = 88 - 95%
PaO_2 = 55 - 80 mm Hg

<u>Lower PEEP/Higher FiO_2</u>

FiO_2	.3	.4	.4	.5	.5	.6	.7	.7	.7	.8	.9	.9	.9	1.0
PEEP	5	5	8	8	10	10	10	12	14	14	14	16	18	18-24

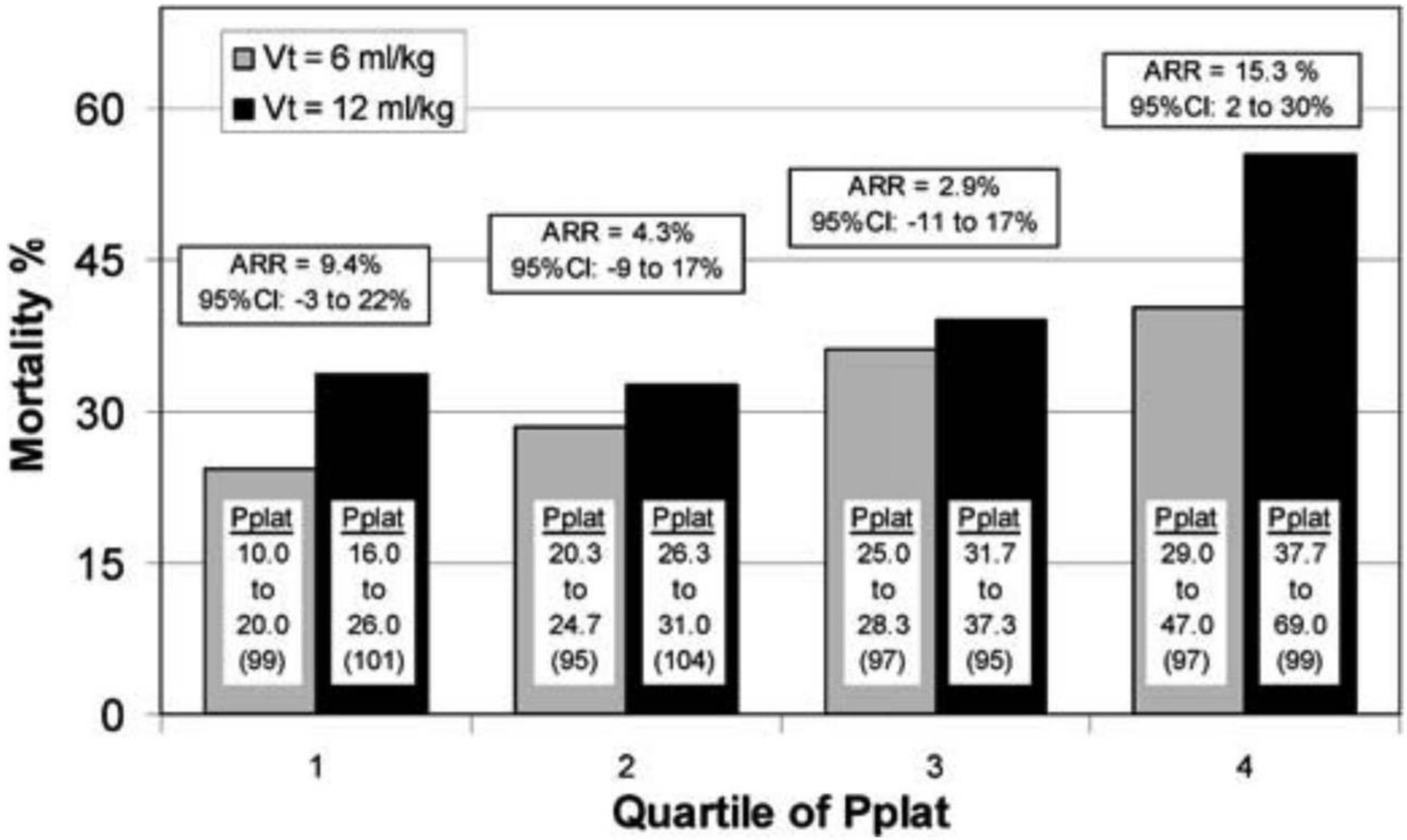
<u>Higher PEEP/Lower FiO_2</u>

FiO_2	.3	.3	.4	.4	.5	.5	.5-.8	.8	.9	1.0
PEEP	12	14	14	16	16	18	20	22	22	22-24

La pression de plateau !

Tidal Volume Reduction in Patients with Acute Lung Injury When Plateau Pressures Are Not High

David N. Hager, Jerry A. Krishnan, Douglas L. Hayden, and Roy G. Brower for the ARDS Clinical Trials Network



Am J Respir Crit Care Med Vol 172. pp 1241–1245, 2005

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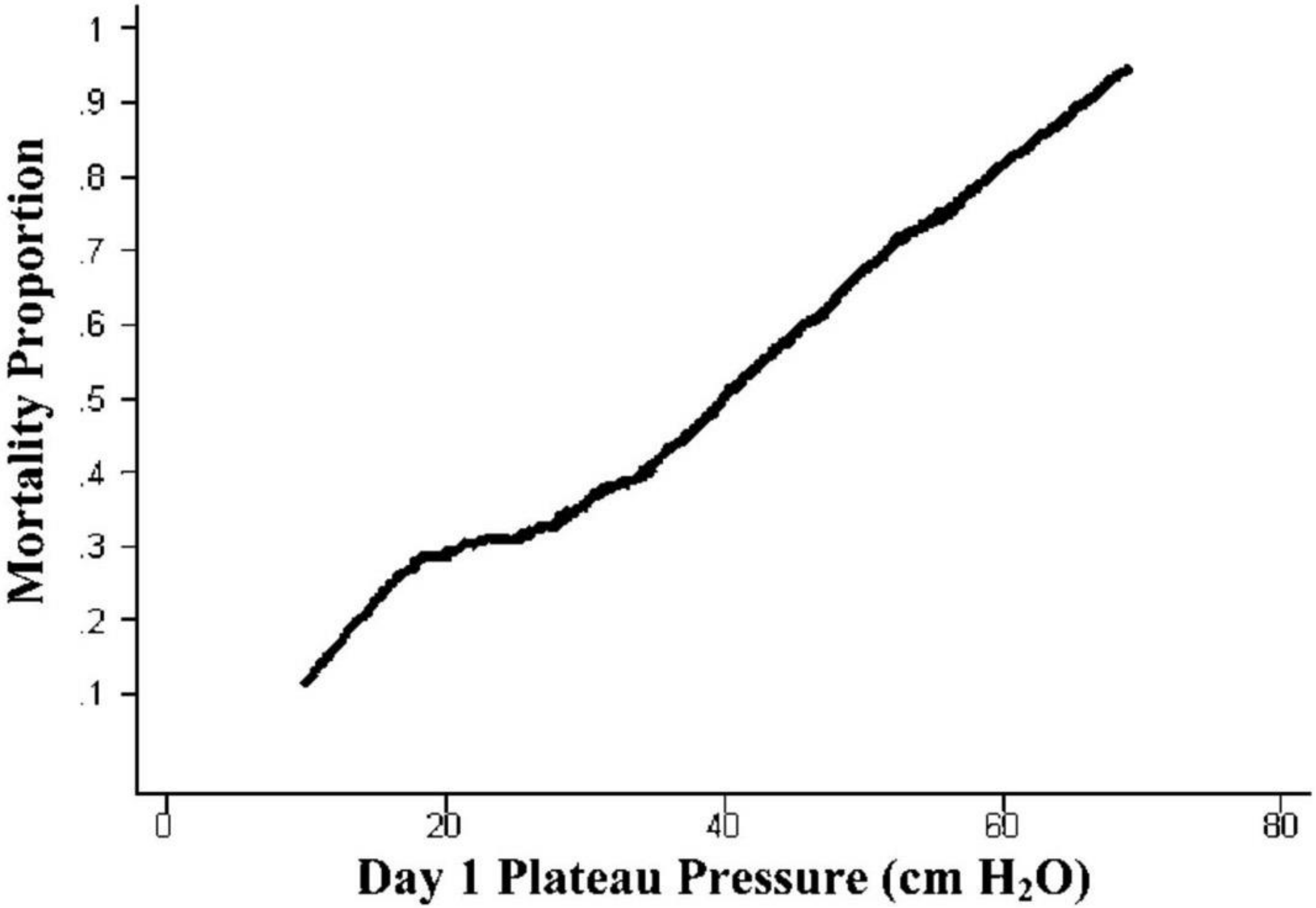


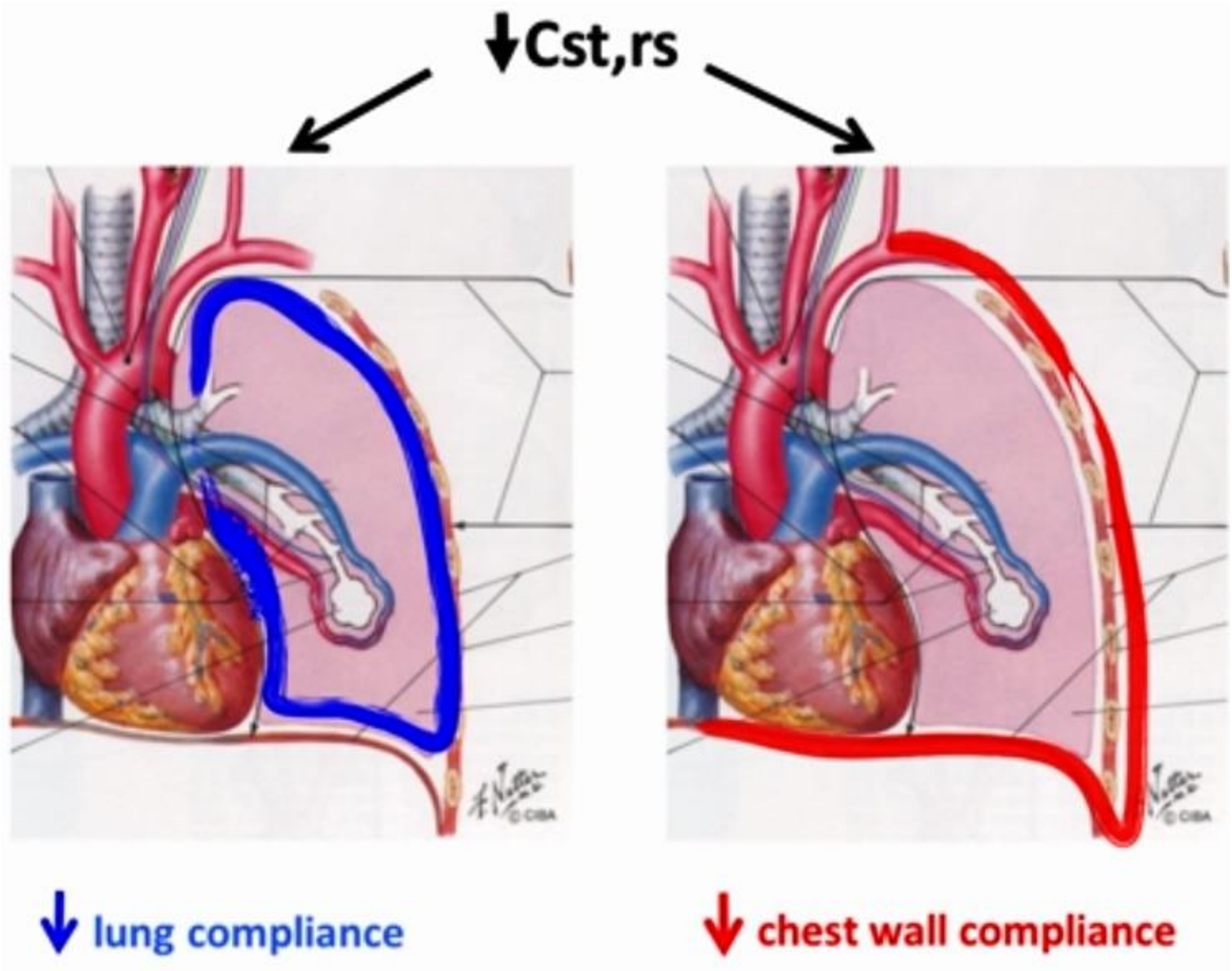
Am J Respir Crit Care Med Vol 172. pp 1241–1245, 2005

Une approche pour titrer la PEEP ?

Utilisation de la pression œsophagienne

La pression transpulmonaire (PTP)

(Paw-Pes)



↓Cst,rs
↓ lung compliance
↓ chest wall compliance

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 NOVEMBER 13, 2008 VOL. 359 NO. 20

Mechanical Ventilation Guided by Esophageal Pressure in Acute Lung Injury

Daniel Talmor, M.D., M.P.H., Todd Sarge, M.D., Atul Malhotra, M.D., Carl R. O'Donnell, Sc.D., M.P.H., Ray Ritz, R.R.T., Alan Lisbon, M.D., Victor Novack, M.D., Ph.D., and Stephen H. Loring, M.D.

Primary endpoint: improvement in oxygenation

V_T 6±2 ml / kg , FR ≤ 35 / mn & 1:3< I:E <1:1

55 mmHg < PaO_2 < 120 mmHg

or 88% < SpO_2 < 98%

Inadequate position of the balloon in 33 % of cases

Esophageal-Pressure–Guided Group ⟷ **PEEP set for P_{Lexp} 0-10 cmH_2O**

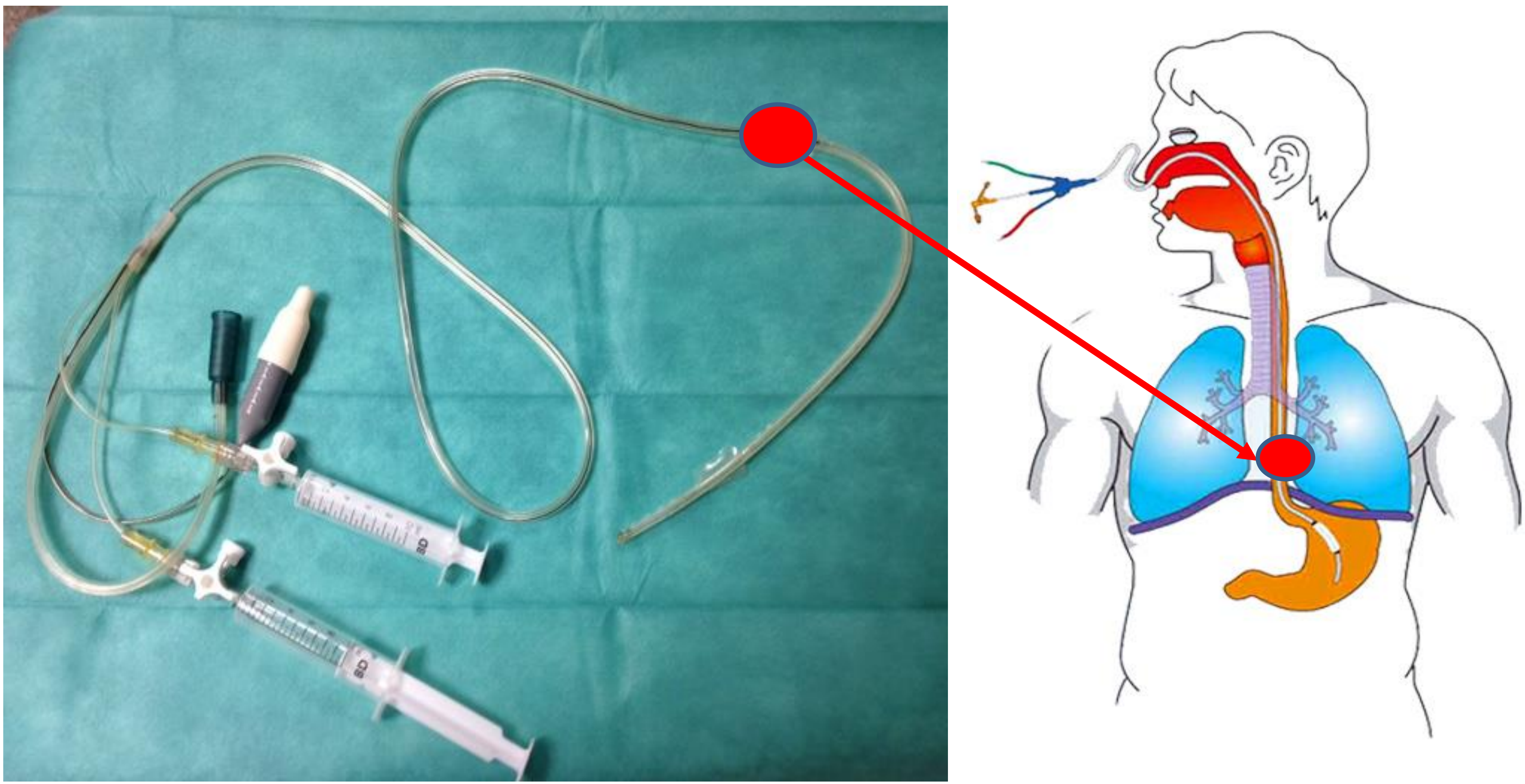
FiO_2	0.4	0.5	0.5	0.6	0.6	0.7	0.7	0.8	0.8	0.9	0.9	1.0
P_{Lexp}	0	0	2	2	4	4	6	6	8	8	10	10

Control Group ⟷ **PEEP set for P_{Plat} < 30 cmH_2O**

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	5	5	8	8	10	10	10	12	14	14	14	16	18	20–24

Monitoring

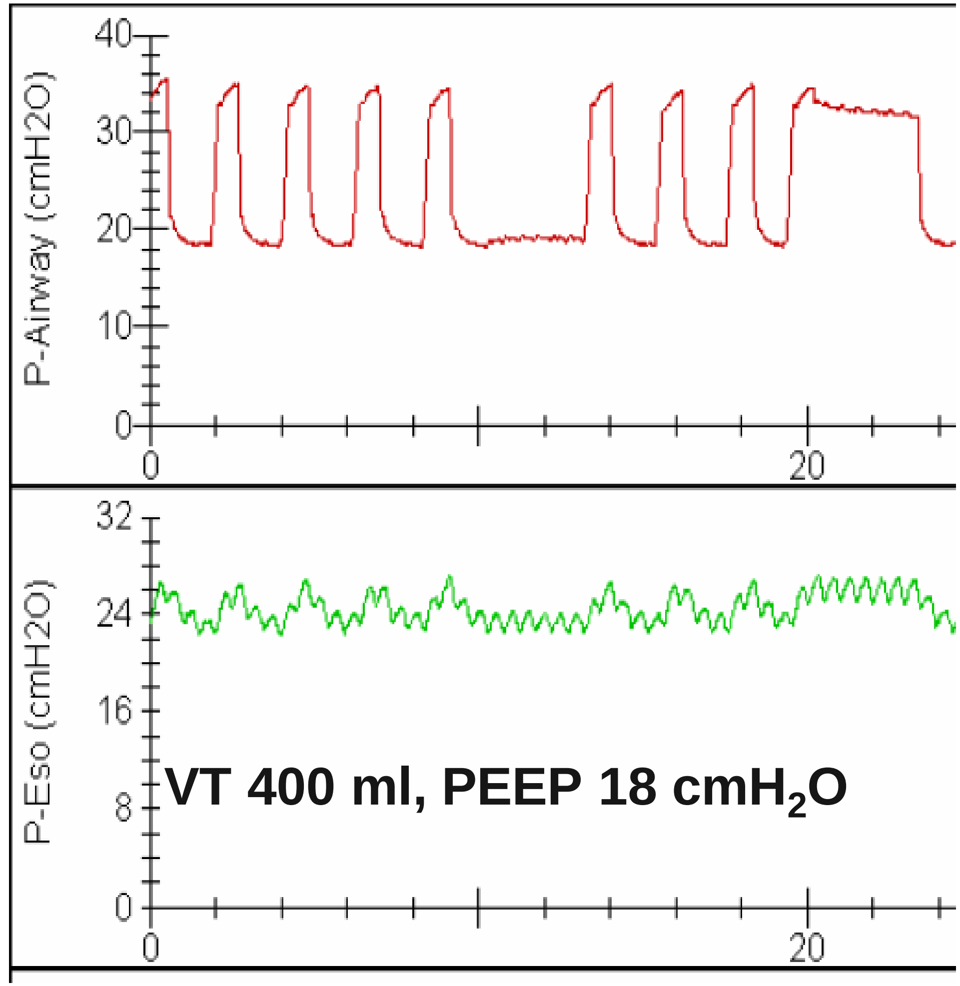
Pression Œsophagienne (Pes) = P pleurale

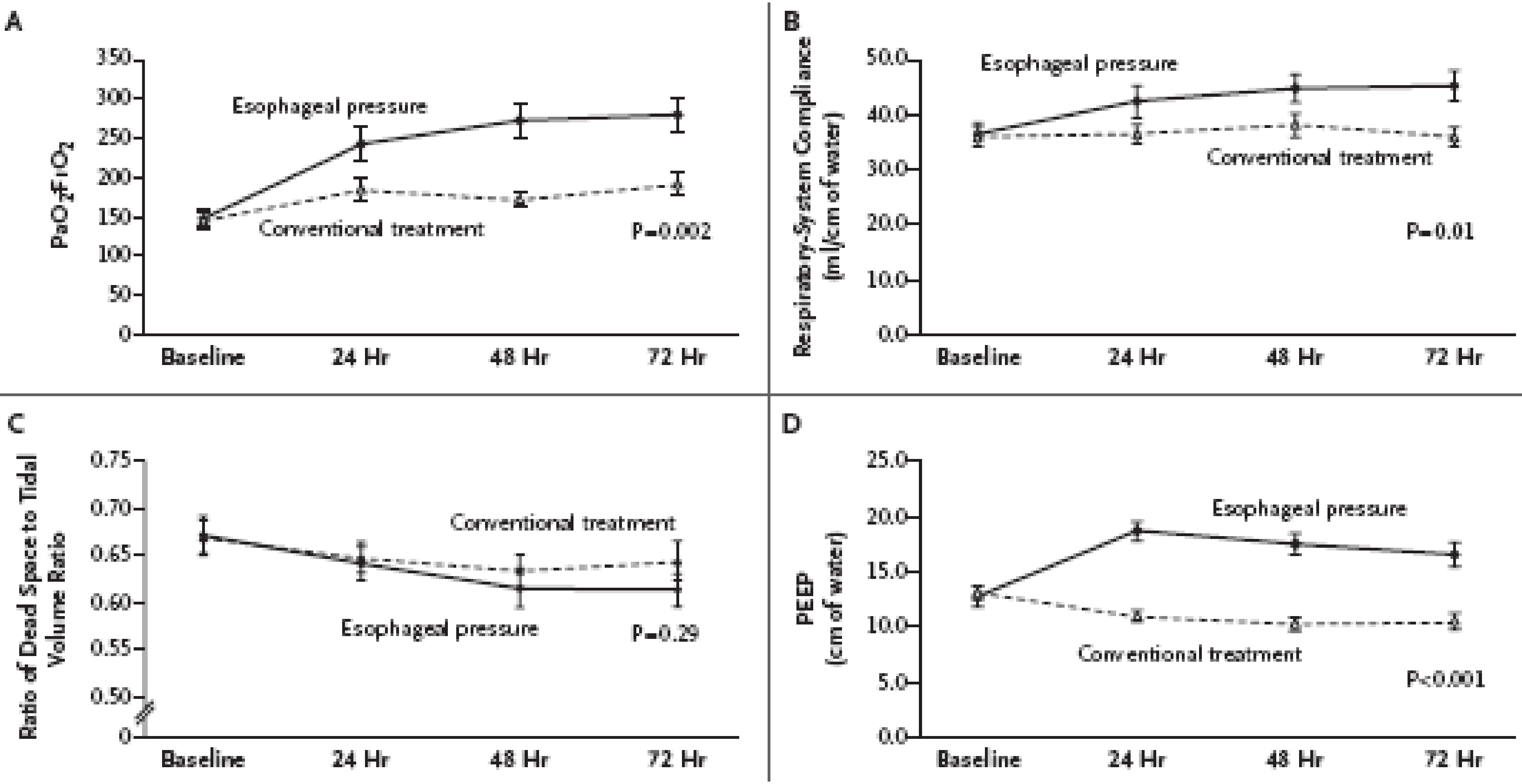


Mechanical Ventilation Guided by Esophageal Pressure in Acute Lung Injury

N ENGL J MED 359;20 WWW.NEJM.ORG NOVEMBER 13, 2008

- Esophageal Catheter
- RM 40 cm H2O for 30 seconds
- PEEP to transpulmonary pressure at end expiration (airway minus oesophageal pressure): 0-10 cmH2O
- Vt: 6 ml/Kg





Outcome	Esophageal-Pressure–Guided (N=30)	Conventional Treatment (N=31)	P Value
28-Day mortality — no. (%)	5 (17)	12 (39)	0.055

N Engl J Med 2008;359:2095-104.

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Table 4. Clinical Outcomes.*

Outcome	Esophageal-Pressure–Guided (N=30)	Conventional Treatment (N=31)	P Value
28-Day mortality — no. (%)	5 (17)	12 (39)	0.055
180-Day mortality — no. (%)	8 (27)	14 (45)	0.13
Length of ICU stay — days			0.16
Median	15.5	13.0	
Interquartile range	10.8–28.5	7.0–22.0	
No. of ICU-free days at 28 days			0.96
Median	5.0	4.0	
Interquartile range	0.0–14.0	0.0–16.0	
No. of ventilator-free days at 28 days			0.50
Median	11.5	7.0	
Interquartile range	0.0–20.3	0.0–17.0	

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Mechanical Ventilation Guided by Esophageal Pressure in Acute Lung Injury

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Table 2. Measurements of Ventilatory Function at Baseline and 72 Hours.*

Measurement	Baseline			72 Hr†		
	Esophageal-Pressure–Guided (N=30)	Conventional Treatment (N=31)	P Value	Esophageal-Pressure–Guided (N=29)	Conventional Treatment (N=29)	P Value
$PaO_2:FiO_2$	147±56	145±57	0.89	280±126	191±71	0.002
Respiratory-system compliance (ml/cm of water)	36±12	36±10	0.94	45±14	35±9	0.005
Ratio of physiological dead space to tidal volume	0.67±0.11	0.67±0.09	0.95	0.61±0.09	0.64±0.10	0.27
PaO_2 (mm Hg)	91±25	107±44	0.09	124±44	101±33	0.03
FiO_2	0.66±0.17	0.77±0.18	0.02	0.49±0.17	0.57±0.18	0.07
PEEP (cm of water)	13±5	13±3	0.73	17±6	10±4	<0.001
Tidal volume (ml)	484±98	491±105	0.80	472±98	418±80	0.03
Tidal volume (ml per kg of predicted body weight)	7.3±1.3	7.9±1.4	0.12	7.1 ±1.3	6.8±1	0.31
Respiratory rate (breaths/min)	26±6	24±6	0.32	26±6	28±5	0.20
Inspiratory time (sec)	0.8±0.1	0.9±0.2	0.19	0.8±0.1	0.8±0.1	0.27
$PEEP_{total}$ (cm of water)	14±5	15±4	0.67	18±5	12±5	<0.001
Peak inspiratory pressure (cm of water)	35±8	35±7	0.85	32±8	28±7	0.007
Mean airway pressure (cm of water)	20±6	20±4	0.88	22±6	16±5	0.001
Plateau pressure (cm of water)	29±7	29±5	0.79	28±7	25±6	0.07
Transpulmonary end-inspiratory pressure (cm of water)	7.9±6.0	8.6±5.4	0.61	7.4±4.4	6.7±4.9	0.58
Transpulmonary end-expiratory pressure (cm of water)	−2.8±5.0	−1.9±4.7	0.49	0.1±2.6	−2.0±4.7	0.06

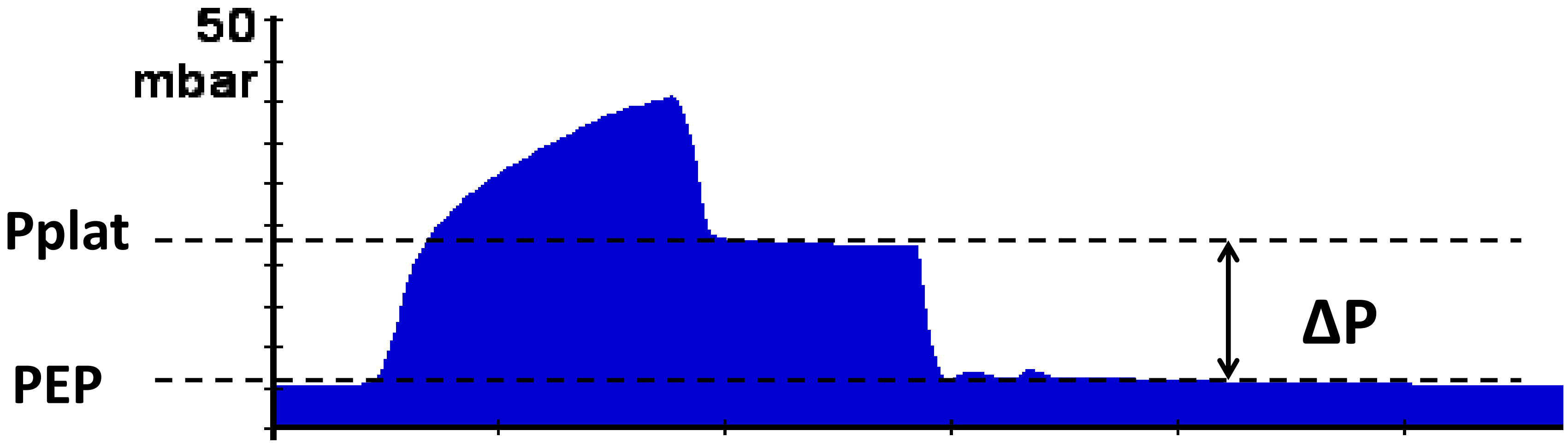
Δ=11 Δ=15

Une autre approche pour titrer la PEEP ?

Driving Pressure : ΔP

(pression motrice)

Driving Pressure : ΔP
(pression motrice)



**ΔP = Pplat-PEP = **

The NEW ENGLAND JOURNAL of MEDICINE

2015

SPECIAL ARTICLE

Driving Pressure and Survival in the Acute Respiratory Distress Syndrome

Marcelo B.P. Amato, M.D., Maureen O. Meade, M.D., Arthur S. Slutsky, M.D., Laurent Brochard, M.D., Eduardo L.V. Costa, M.D., David A. Schoenfeld, Ph.D., Thomas E. Stewart, M.D., Matthias Briel, M.D., Daniel Talmor, M.D., M.P.H., Alain Mercat, M.D., Jean-Christophe M. Richard, M.D., Carlos R.R. Carvalho, M.D., and Roy G. Brower, M.D.

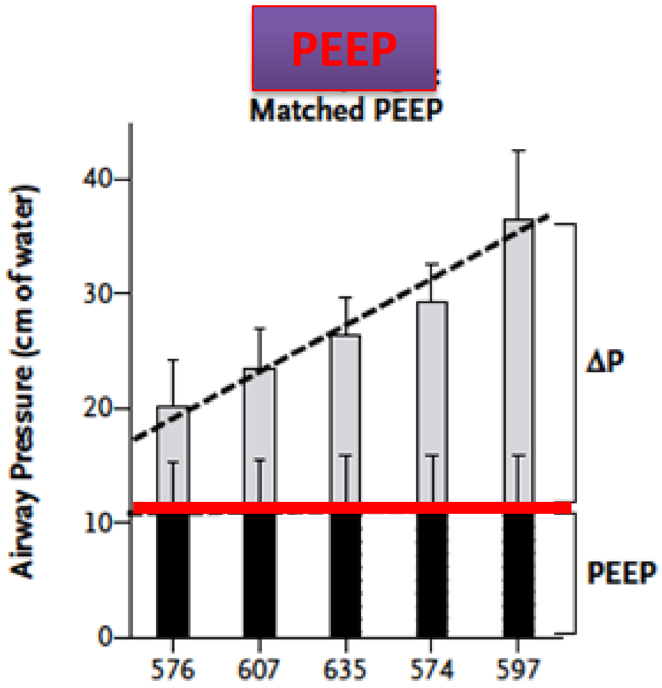
Hypothèse: La « driving pressure » (= pression motrice ΔP = Pplat-PEEP) serait un meilleur indicateur de la taille de poumon fonctionnel restant et donc un meilleur facteur prédictif de survie chez les patients en SDRA que le Vt ou la PEP.

Hypothèse de l'étude :

- ΔP = variable indépendante associée à la survie
- Lien statistique plus fort entre ΔP et survie plutôt qu'entre Vt et survie ou PEP et survie chez les patients en SDRA

Meta-analyse : Données individuelles de patients en SDRA issues de
9 études randomisées (RCT)

Nombre de Sujets n = 3562

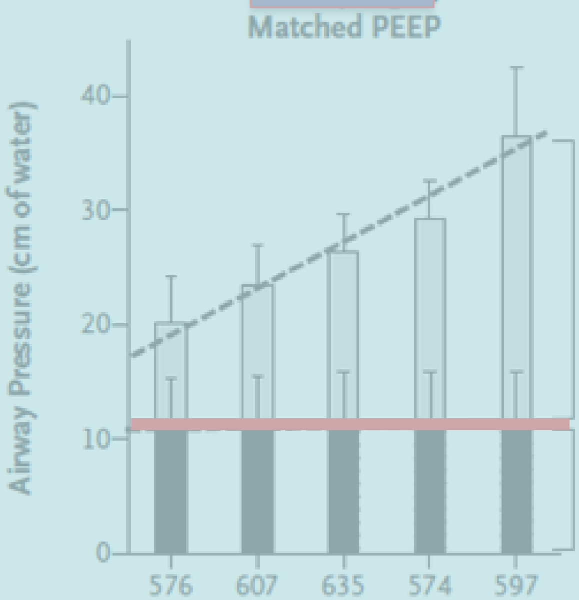


P-plateau 20-24-28-30-35

PEEP 10-10-10-10-10

Pression Motrice (ΔP) 10-14-18-20-25

PEEP

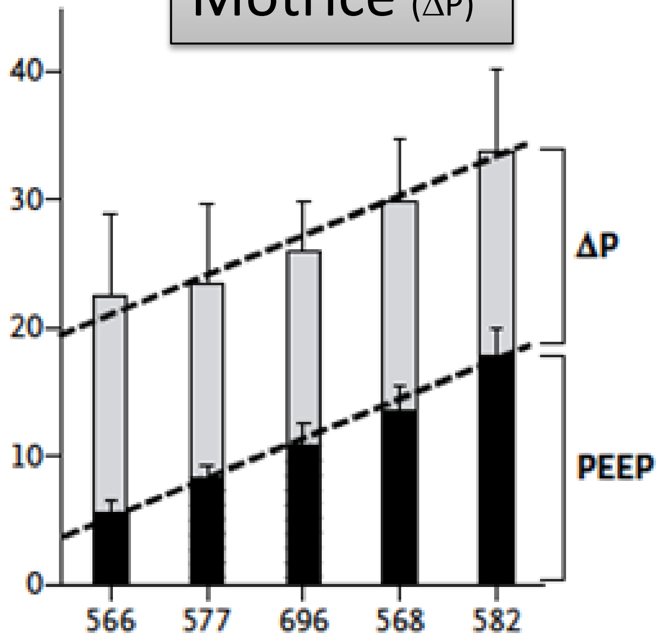


P-plateau 20-24-28-30-35

PEEP 10-10-10-10-10

Pression Motrice (ΔP) 10-14-18-20-25

Pression Motrice (ΔP)

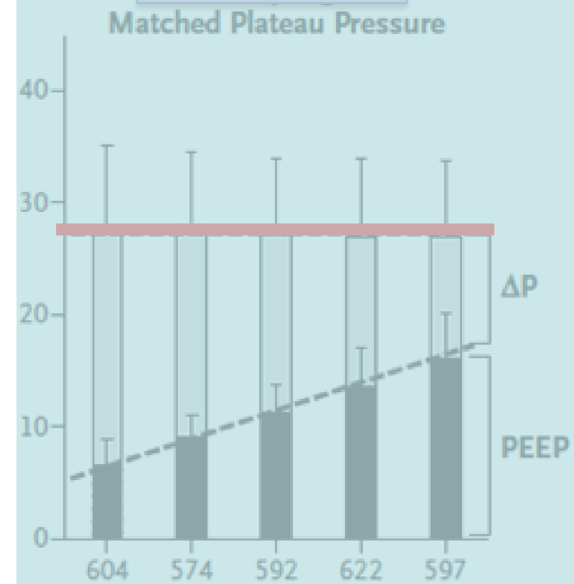


P-plateau 22-24-26-28-31

PEEP 7-9-11-13-16

Pression Motrice (ΔP) 15-15-15-15-15

P-plateau

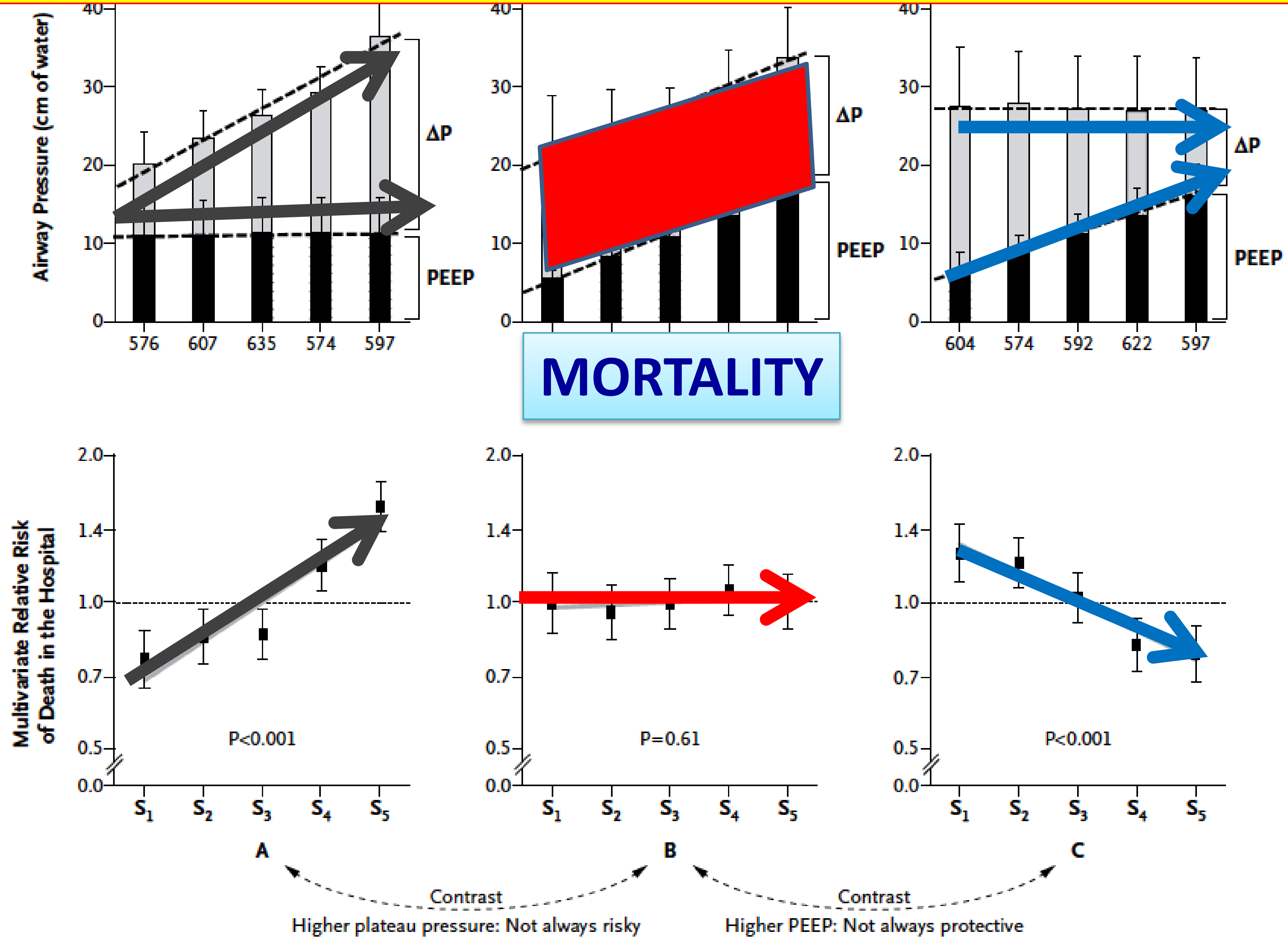


P-plateau 28-28-28-28-28

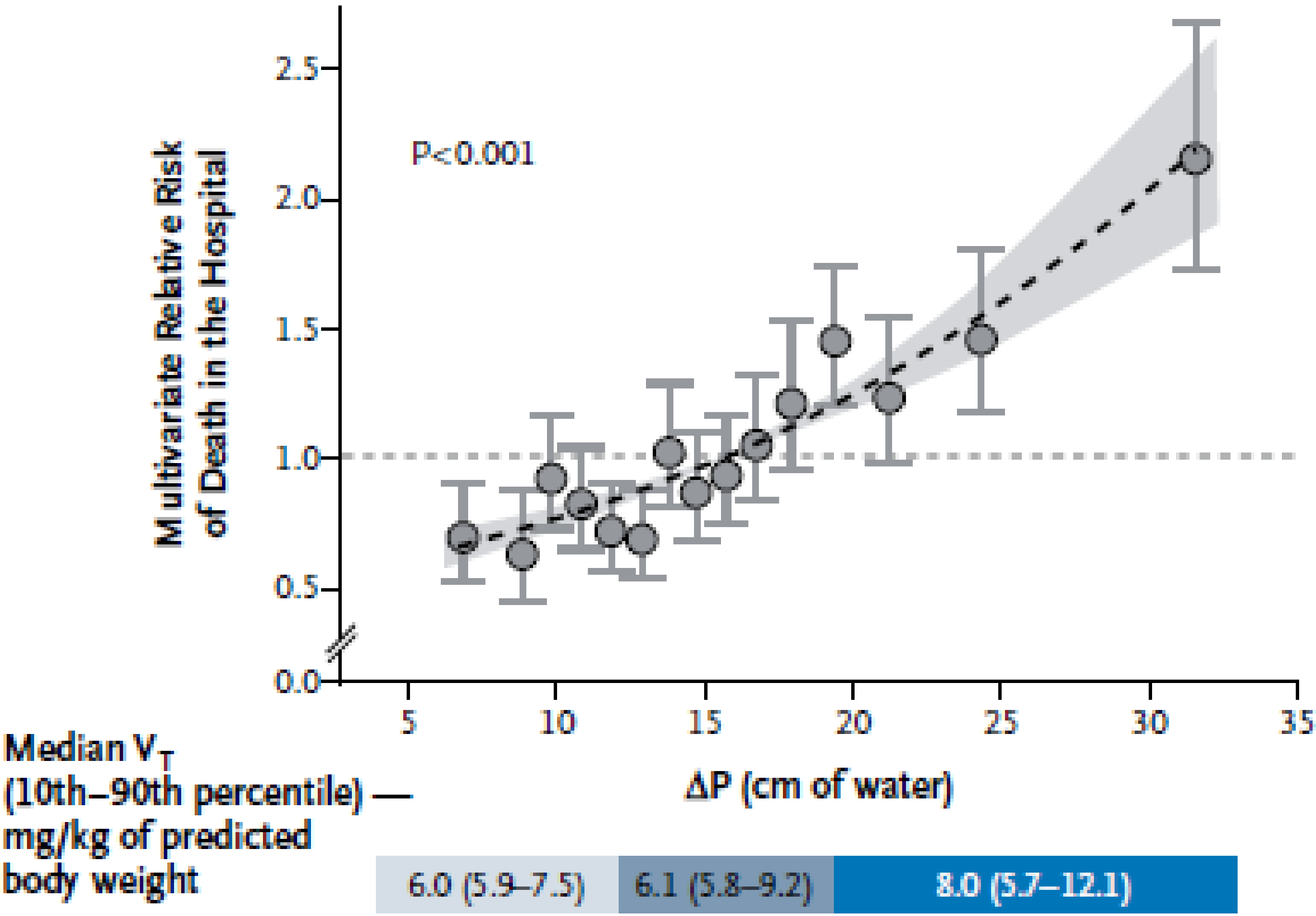
PEEP 6-8-10-12-14

Pression Motrice (ΔP) 22-20-18-16-14

ΔP (et pas la PEEP ou le Vt) **est associée à la mortalité**



Pression motrice (ΔP) associée à la mortalité (RR = 1,4 [1,3-1,5])
Même en cas de ventilation protectrice



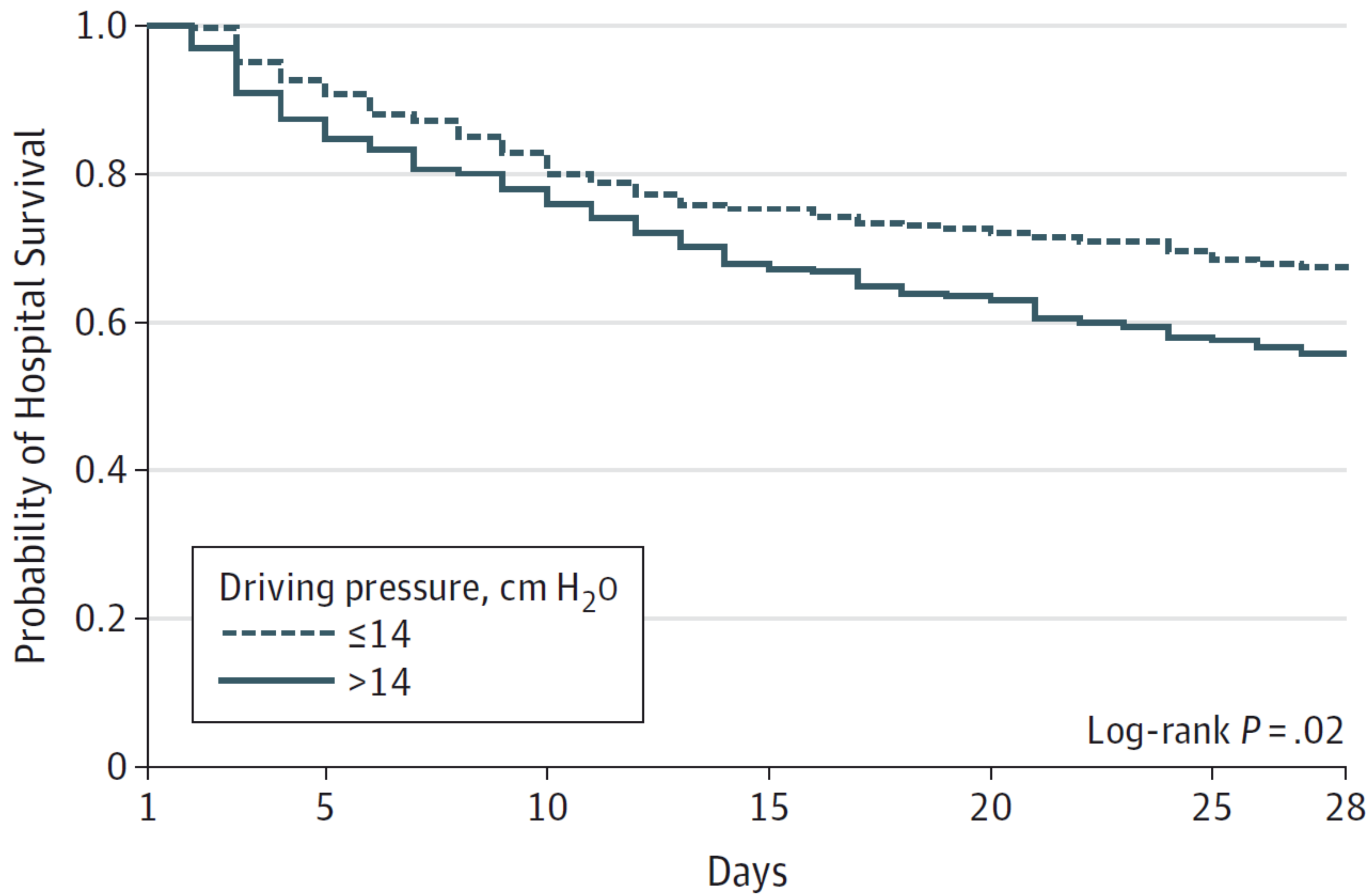
Messages des auteurs

Conclusion : pression motrice (∆P) meilleur paramètre ventilatoire pour prédire la mortalité dans le SDRA

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

JAMA. 2016;315(8):788-800.

C Probability of hospital survival by driving pressure



Incidence of mortality and morbidity related to postoperative lung injury in patients who have undergone abdominal or thoracic surgery: a systematic review and meta-analysis

Articles

Ary Serpa Neto, Sabrine NT Hemmes, Carmen SV Barbas, Martin Beiderlinden, Ana Fernandez-Bustamante, Emmanuel Futier, Markus W Hollmann, Samir Jaber, Alf Kozian, Marc Licker, Wen-Qian Lin, Pierre Moine, Federica Scavonetto, Thomas Schilling, Gabriele Selmo, Paolo Severgnini, Juraj Sprung, Tanja Treschan, Carmen Unzueta, Toby N Weingarten, Esther K Wolthuis, Hermann Wrigge, Marcelo Gama de Abreu, Paolo Pelosi, Marcus J Schultz, for the the PROVE Network investigators

Lancet Respir Med 2014; 2(12):1007-15

	HR for in-hospital mortality (95% CI)	HR for ICU discharge (95% CI)
All patients	9·58 (5·32–17·34)	0·45 (0·33–0·66)
Ventilation		
Conventional	14·22 (5·91–34·26)	0·39 (0·25–0·58)
Protective	6·07 (2·47–14·55)	0·71 (0·42–1·19)

	Total (n=3365)	No postoperative lung injury (n=3150)*	Postoperative lung injury (n=123)*	p value
Tidal volume (mL/kg PBW)	8.2 (1.9)	8.2 (1.8)	9.3 (2.1)	<0.0001
PEEP (cm H2O)	4.4 (3.8)	**4.3 (3.7)**	**2.9 (3.4)**	<0.0001

Protective *versus* Conventional Ventilation for Surgery

A Systematic Review and Individual Patient Data Meta-analysis

Ary Serpa Neto, M.D., M.Sc., Ph.D., Sabrine N. T. Hemmes, M.D., Carmen S. V. Barbas, M.D., Ph.D., Martin Beiderlinden, M.D., Michelle Biehl, M.D., Jan M. Binnekade, Ph.D., Jaume Canet, M.D., Ph.D., Ana Fernandez-Bustamante, M.D., Ph.D., Emmanuel Futier, M.D., Ph.D., Ognjen Gajic, M.D., Ph.D., Göran Hedenstierna, M.D., Ph.D., Markus W. Hollmann, M.D., Ph.D., Samir Jaber, M.D., Ph.D., Alf Kozian, M.D., Ph.D., Marc Licker, M.D., Wen-Qian Lin, M.D., Andrew D. Maslow, M.D., Stavros G. Memtsoudis, M.D., Ph.D., Dinis Reis Miranda, M.D., Pierre Moine, M.D., Thomas Ng, M.D., Domenico Paparella, M.D., Christian Putensen, M.D., Ph.D., Marco Ranieri, M.D., Ph.D., Federica Scavonetto, M.D., Thomas Schilling, M.D., Ph.D., D.E.A.A., Werner Schmid, M.D., Ph.D., Gabriele Selmo, M.D., Paolo Severgnini, M.D., Juraj Sprung, M.D., Ph.D., Sugantha Sundar, M.D., Daniel Talmor, M.D., M.P.H., Tanja Treschan, M.D., Carmen Unzueta, M.D., Ph.D., Toby N. Weingarten, M.D., Esther K. Wolthuis, M.D., Ph.D., Hermann Wrigge, M.D., Ph.D., Marcelo Gama de Abreu, M.D., Ph.D., Paolo Pelosi, M.D., F.E.R.S., Marcus J. Schultz, M.D., Ph.D.

This article has been selected for the Anesthesiology CME Program. Learning objectives and disclosure and ordering information can be found in the CME section at the front of this issue.

Anesthesiology 2015

ABSTRACT

Background: Recent studies show that intraoperative mechanical ventilation using low tidal volumes (V_T) can prevent postoperative pulmonary complications (PPCs). The aim of this individual patient data meta-analysis is to evaluate the individual associations between V_T size and positive end–expiratory pressure (PEEP) level and occurrence of PPC.

Methods: Randomized controlled trials comparing protective ventilation (low V_T with or without high levels of PEEP) and conventional ventilation (high V_T with low PEEP) in patients undergoing general surgery. The primary outcome was development of PPC. Predefined prognostic factors were tested using multivariate logistic regression.

Protective versus Conventional Ventilation for Surgery

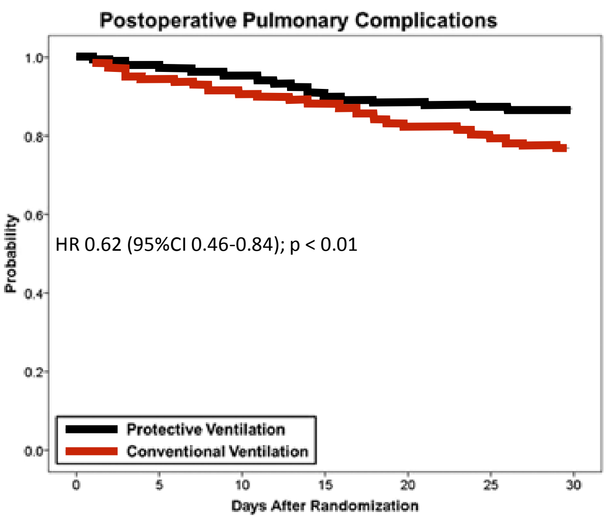
A Systematic Review and Individual Patient Data Meta-analysis

- Data from 15 RCTs (N=2127 patients)
- Surgical procedures: Abdominal, n=5 studies

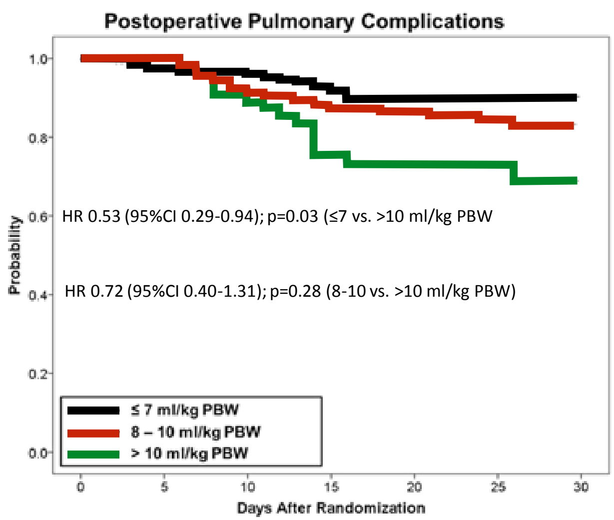
  Cardiothoracic, n=7 studies

  Others, n=3 studies

Protective *vs* Non-protective

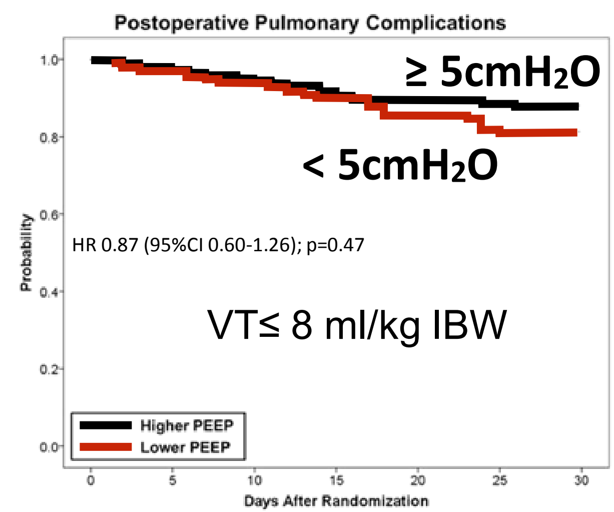


Lower *vs* Higher VT



Lower *vs* Higher PEEP

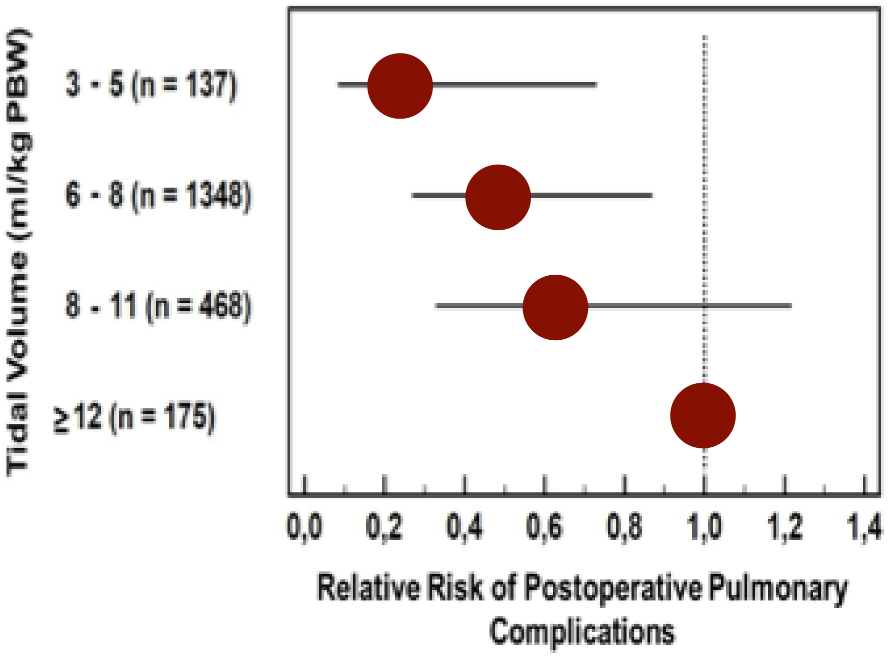
(N=525) (N=977)

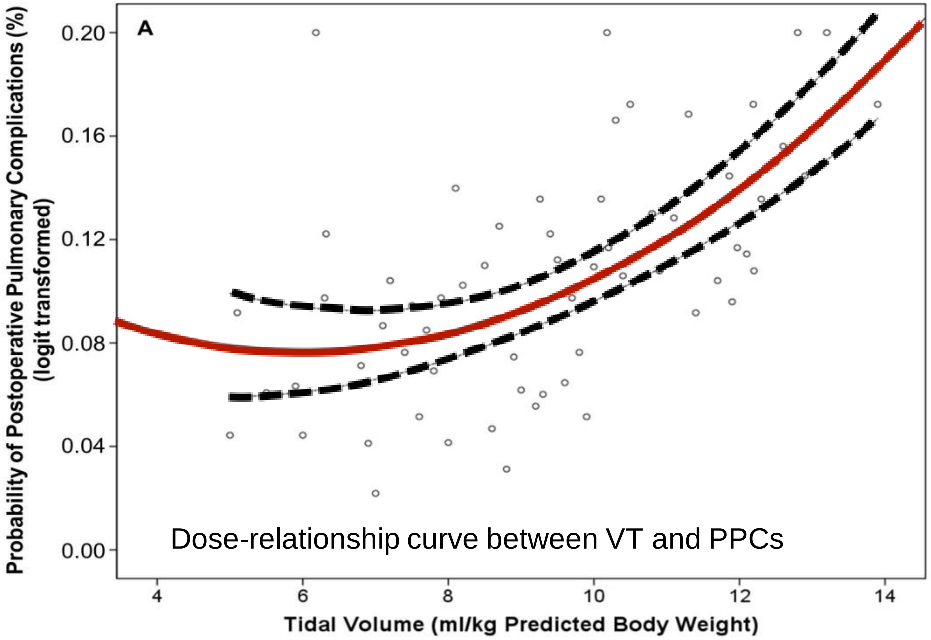


Ary Serpa Neto (for the PROVE Network) et al. Anesthesiology 2015

Dose-Response Relationship Between PPC and VT

IMPROVE trial - N Engl J Med 2013;369:428-37
PROVHILO study - The Lancet 2014 Aug 9;384(9942):495-503





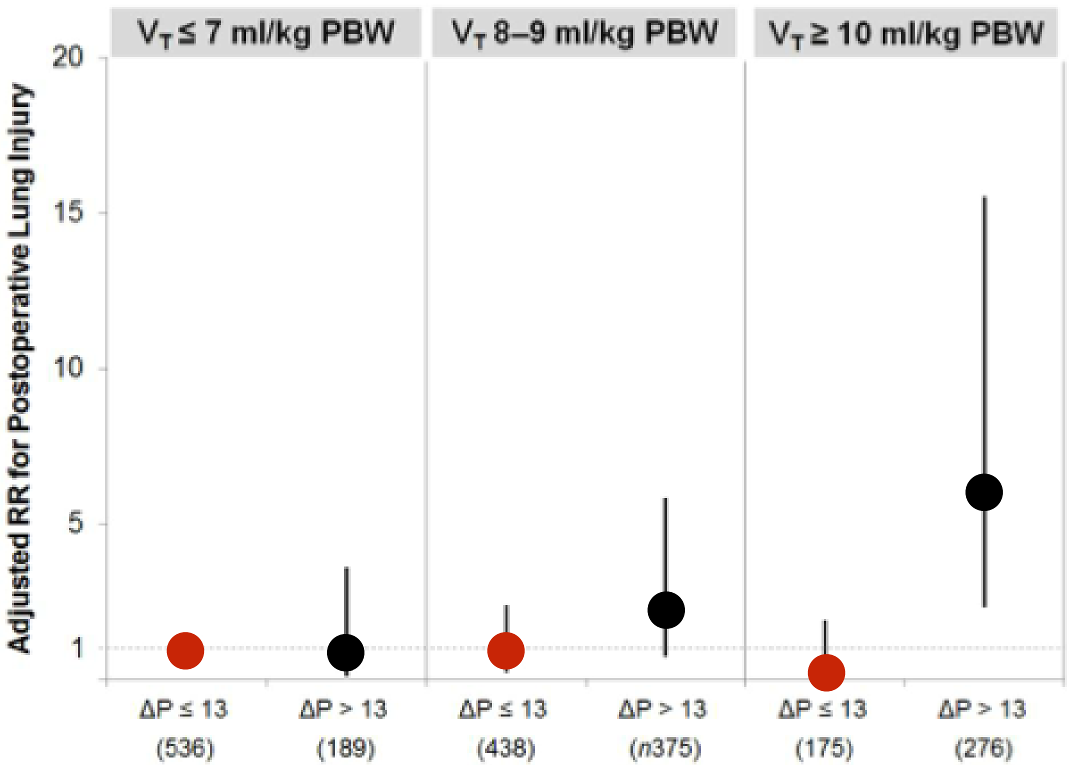
Dose-relationship curve between VT and PPCs

Ary Serpa Neto (for the PROVE Network) et al. Anesthesiology 2015

Dose-Response Relationship Between PPC and Driving Pressure

IMPROVE trial - N Engl J Med 2013;369:428-37
PROVHILO study - The Lancet 2014 Aug 9;384(9942):495-503

Lower VT and Driving Pressure reduce PPCs

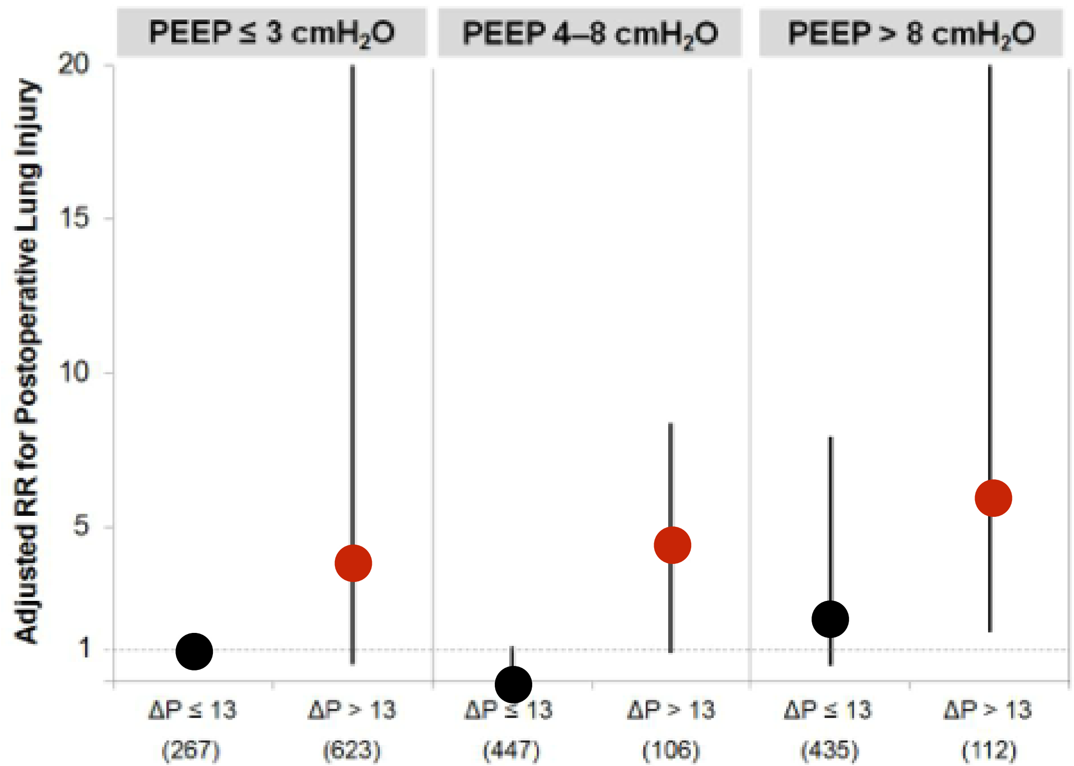


Ary Serpa Neto (for the PROVE Network) et al. Submitted

Dose-Response Relationship Between PPC and Driving Pressure

IMPROVE trial - N Engl J Med 2013;369:428-37
PROVHILO study - The Lancet 2014 Aug 9;384(9942):495-503

Higher Driving Pressure and PEEP increase PPCs



Ary Serpa Neto (for the PROVE Network) et al. Submitted

Dose-Response Relationship Between PPC and Driving Pressure

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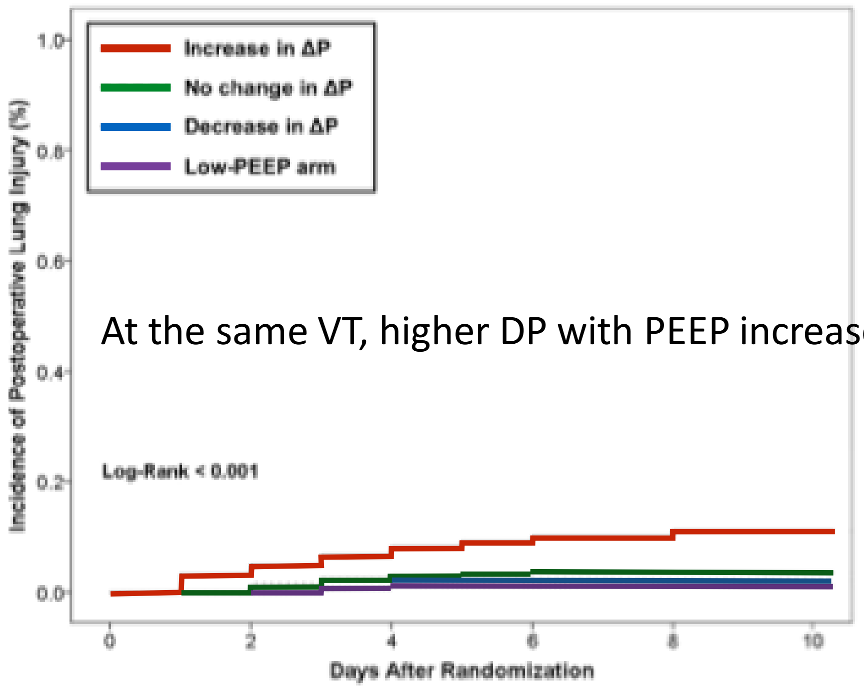

At the same VT, higher DP with PEEP increase PPCs

Number at Risk	0	2	4	6	8	10
mean ΔP-change = – 3.7 cmH_2O	174	171	165	150	128	59
mean ΔP-change = + 0.4 cmH_2O	143	139	129	109	87	39
mean ΔP-change = + 4.3 cmH_2O	124	117	108	91	71	30
Low-PEEP Arm	405	398	372	325	262	115

Ary Serpa Neto (for the PROVE Network) et al Soumis 2015

A retenir

Conclusion – SDRA à retenir

Efficace

1.- Volume bas = 6 ml/kg/PIT
2.- 5 < PEP < 15 cmH2O
3.- Pression plateau < 30 cmH2O
+ Pression motrice (Pplat-PEP)<15 cmH2O
4.- Curares (précoce et durée <48h)
5.- Décubitus Ventral (D.V)

Non-efficace

1.- Béta-2 mimétiques
2.- H.F.O
3.- Surfactant,
4.- Omega 3

Faut voir ?

1.- ECMO / ECCO2R
2.- Réglages aidées par P-esophagienne
3.- Corticoides (précoce et/ou tardive ?)
4.- N.O (sauvetage-rescue)
5.- Manoeuvres de recrutement
6.- Echographie