SINDROME DISTRES RESPIRATORIO PEDIATRICO

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Medico Pediatra Intensivista 2019

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SCORE DE INJURIA PULMONAR

- PEEP
- RX TX
- PAFI
- COMPLIANCE

SDRA SEVERO MAYOR DE 2,5

Murray JF, Matthay MA. Am Rev Respir Dis 1988;138:720-3.

Lung Injury Score

Murray et al, ARRD 1988; 138:720-723

	0	1	2	3	4
C-X-R	No consolidation	In 1 quadrant	In 2 quadrants	In 3 quadrants	In all 4 quadrants
PaO_2/FiO_2	≥300	225-299	175-224	100-174	<100
Compliance	≥80	60-79	40-59	20-39	≤19
PEEP	≤5	6-8	9-11	12-14	≥15

LIS= aggregate sum / number of components

ARDS = LIS > 2.5

CONSENSO AMERICANO-EUROPEO

ALI:

- **Inicio agudo**
- **Infiltrado bilateral en Rxtx**
- **PcP menor de 18 mmHg o ausencia de IVI**
- **PAFI menor de 300 mmHg.**

SDRA:

- **PAFI MENOR DE 200 mmHg.**

BERNARD GR, ARTIGAS A, BRIGHAM KL et all. The American –European consensus Conference on ARDS. Am J Respir Crit Care Med 1994; 149: 818-824.

Nueva definición SDRA BERLIN 2012

	Leve	Moderado	Grave
Tiempo de inicio	Inicio dentro de 1 semana de conocida la injuria clínica o nuevo o deterioro de los síntomas respiratorios		
Imagen torácica[a]	Opacidades bilaterales -no explicable por derrame, atelectasia pulmonar lobar o pulmonar, o nódulos		
Origen del edema	Falla respiratoria no explicable completamente por una insuficiencia cardiaca o la sobrecarga de líquidos. Necesita evaluación objetiva (ej. Ecocardiograma) para excluir edema hidrostático si no hay factor de riesgo presente.		
Hipoxemia PaO_2/FiO_2 ratio	200-300, con PEEP/CPAP ≥5	≤200 - >100, con PEEP ≥5	≤100, con PEEP ≥5

Ranieri VM, Rubenfeld GD, Thompson BT, Caldwell E, Fan E, Camporota L, and Slutsky AS. Acute Respiratory Distress Syndrome The Berlin Definition. JAMA 2012; 307(23): doi:10.1001/jama.2012.5669.

PARDS Pediátrico

Edad	Excluir a los pacientes con enfermedad pulmonar perinatal relacionada.
Tiempo	Dentro de los 7 días de conocida insulto clínica.
Imágenes Radiográficas	Hallazgos de imagen de radiografías de nuevo infiltrado(s), relacionados con la enfermedad parénquima pulmonar aguda.

Pacientes no intubados

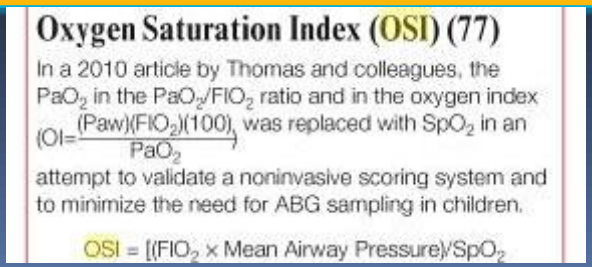
para los pacientes con máscarilla facial con ventilación no invasivo bi-level o CPAP> = 5 cm H2O
- PaO2 disponible, entonces la relación PF <= 300
- PaO2 no disponibles, destetar FiO2 para mantener SpO2 <= 97%, proporción SF <= 264

Para los pacientes con ventilación mecánica invasiva a través de un tubo endotraqueal o traqueostomía

ARDS Leve	ARDS Moderado	ARDS Severo
PaO2 disponibles, OI entre 4 y 8 OSI entre 5 y 7.5 *	PaO2 disponibles, OI entre 8 y 16 OSI entre 7.5 y 12.3 *	PaO2 disponibles, OI > 16 OSI > 12.3 *

* Si la PaO2 no disponible, destetar FiO2 para mantener SpO2 <= 97% para calcular OSI

The Pediatric Acute Lung Injury Consensus Conference Group
2015 • Volume 16 • Number



Oxygen Saturation Index (OSI) (77)

In a 2010 article by Thomas and colleagues, the PaO_2 in the PaO_2/FIO_2 ratio and in the oxygen index $\left(OI=\frac{(Paw)(FIO_2)(100)}{PaO_2}\right)$ was replaced with SpO_2 in an attempt to validate a noninvasive scoring system and to minimize the need for ABG sampling in children.

$OSI = [(FIO_2 \times \text{Mean Airway Pressure})/SpO_2$

The Montreux definition of neonatal ARDS

	Details
Timeframe	Acute onset (ie, within one week) from a known or suspected clinical insult
Exclusion criteria	RDS, TTN, or congenital anomalies as a primary current acute respiratory condition
Lung imaging	Diffuse, bilateral, and irregular opacities or infiltrates, or complete opacification of the lungs, which are not fully explained by local effusions, atelectasis, RDS, TTN, or congenital anomalies
Origin of oedema	Absence of congenital heart disease explaining the oedema (this includes ductus arteriosus with pulmonary overflow if no acute pulmonary haemorrhage exists). Echocardiography is needed to verify the origin of oedema.
Oxygenation deficit expressed as OI*	Mild ARDS: $4 \leq OI < 8$ Moderate ARDS: $8 \leq OI < 16$ Severe ARDS: $OI \geq 16$

ARDS=acute respiratory distress syndrome. RDS=respiratory distress syndrome. TTN=transient tachypnoea of the neonate. OI=oxygenation index. *OI can be calculated by use of arterial or, if arterial values are unavailable, transcutaneous oxygen tension values, with appropriately calibrated transcutaneous devices. In the case of persistent pulmonary hypertension of the neonate and patent ductus arteriosus, preductal PaO_2 values should be used. The definition applies from birth until 44 weeks, post-menstrual age or until 4 weeks, postnatal age (for neonates born after 40 weeks, post-menstrual age). For the syndrome to be defined all criteria must be fulfilled. OI should be calculated with the most accurate measures available. The syndrome can be diagnosed at any gestational age or birthweight, provided that congenital lung anomalies, RDS, and TTN are excluded as primary respiratory disorder. Criteria for the diagnosis of RDS and TTN, exclusion criteria for neonatal ARDS, and suggestions to improve the reliability of transcutaneous blood gas measurements are provided in the Montreux definition of neonatal ARDS section.

***Table 2*: The Montreux definition of neonatal ARDS**

www.thelancet.com/respiratory **Published online July 4, 2017**

The Montreux definition of neonatal ARDS

	ARDS	Paediatric ARDS	Neonatal ARDS				
			Sepsis	Pneumonia*	MAS	Aspiration†	Lung haemorrhage
C_{rs}	Large decrease	Large decrease	Small or large decrease	Within normal range or small decrease‡	Large decrease	Large decrease	Large decrease
R_{rs}§	Within normal range or slightly increased	Within normal range or slightly increased	Within normal range or slightly increased	Within normal range or slightly increased	Usually increased	Increased	Within normal range or increased
Extension of pathological process	Diffuse	Diffuse	Diffuse	Variable	Diffuse	Diffuse	Diffuse
Origin¶	Direct or indirect	Direct or indirect	Indirect	Direct	Direct	Direct	Direct

This list of neonatal respiratory conditions should be considered illustrative, because other disorders could share pathophysiological traits with ARDS. ARDS=acute respiratory distress syndrome. MAS=meconium aspiration syndrome. C_{rs}=respiratory system compliance. R_{rs}=respiratory system resistance. *Pneumonia refers to any type of infectious or biliary pneumonia following intrahepatic cholestasis of pregnancy. †Aspiration refers to lung injuries related to the aspiration of material other than meconium (eg, maternal blood, milk, gastric or bile secretions, and water in the case of underwater birth). ‡Changes in compliance can vary depending on the extension of the pathological process. §R_{rs} is usually within the normal range or slightly increased in ARDS and paediatric ARDS, however underlying obstructive conditions (eg, bronchopulmonary dysplasia) or concomitant airway inflammatory diseases (eg, bronchiolitis) might change these resistances. ¶Origin refers to the triggers occurring directly (eg, neonatal aspiration syndromes and pneumonia) or indirectly in lung tissue (eg, sepsis and necrotising enterocolitis).

Table 1: **Neonatal ARDS respiratory disorders that share similar physiopathology with ARDS and paediatric ARDS**

www.thelancet.com/respiratory **Published online July 4, 2017**

Table 1 Progression of ARDS definitions in paediatrics

Criteria	AECC (9), 1994	Berlin (10), 2012	PALICC (11), 2015	Montreux (12), 2017
Age limit	–	–	–	Birth until 44 weeks post-menstrual age or until 4 weeks postnatal age for neonates born after 40 weeks post-menstrual age (diagnosis may be made at any gestational age or birthweight)
Timing of onset	Acute onset	Within one week from known or suspected clinical insult, OR worsening respiratory symptoms	Within one week from known or suspected clinical insult	Within 1 week from known or suspected clinical insult
Origin of edema	PAWP ≤18 mmHg OR no clinical signs of left atrial hypertension	Respiratory failure not completely explained by fluid overload or heart failure	Respiratory failure not completely explained by fluid overload or heart failure	Excluded if echocardiography reveals congenital heart disease explaining the edema (including PDA)
Exclusion criteria	–	–	Perinatal-related lung disease	Primary congenital lung anomalies, RDS and TTN
Chest radiography	Bilateral infiltrates on frontal CXR	Bilateral infiltrates on CXR or CT, not fully explained by effusions or collapse	New infiltrate(s) consistent with acute pulmonary parenchymal disease	Bilateral, diffuse infiltrates or complete bilateral lung opacification NOT explained by primary congenital lung anomalies, RDS, TTNB, localized effusions or collapse
Severity of hypoxemia				
Mild	ALI: PaO_2/FiO_2 ≤300 mmHg (regardless of PEEP)	200 mmHg < PaO_2/FiO_2 ≤300 mmHg with PEEP ≥5 cmH_2O	4≤ OI <8, OR 5≤ OSI <7.5	4≤ OI <8
Moderate	–	100 mmHg < PaO_2/FiO_2 ≤200 mmHg with PEEP ≥ 5 cmH_2O	8≤ OI <16, OR 7.5≤ OSI <12.3	8≤ OI <16
Severe	ARDS: PaO_2/FiO_2 ≤200 mmHg (regardless of PEEP)	PaO_2/FiO_2 ≤100 mmHg with PEEP ≥5 cmH_2O	OI ≥16, OR OSI ≥12.3	OI ≥16
Non-invasive ventilation	–	200 mmHg < PaO_2/FiO_2 ≤300 mmHg, CPAP ≥5 cmH_2O	PaO_2/FiO_2 ≤300 OR SpO_2/FiO_2 ≤264; full face-mask bi-level ventilation, or CPAP ≥5 cmH_2O	–
Special considerations	–	–	Cyanotic heart disease, chronic lung disease, left ventricular dysfunction must meet criteria above and oxygenation defect must not be explained by underlying illness	–

Table 1 *(continued)*

Criteria	AECC (9), 1994	Berlin (10), 2012	PALICC (11), 2015	Montreux (12), 2017
Advantages	Provided consensus to the various definitions used in the past	Defined the term acute	Focus on children with ARDS	Completes the definition of ARDS for all age groups
		Distinct classification of the three ARDS subtypes and elimination of the ALI category	Offered alternative to PaO_2/FiO_2 given the potential difficulty in obtaining invasive arterial access	Improves cross-disciplinary awareness of neonatal ARDS and its treatment, guides research and prognostication
		Minimal PEEP level stated	The use of OI to account for settings used in positive pressure ventilation	Takes into account the possible effects of fetal hemoglobin concentration on oxygen saturation
		Clearer definition of chest radiograph's findings	Distinguished PARDS from hypoxemic respiratory failure secondary to perinatal-related disease	
		PAWP criteria removed	Removal of requirement of bilateral infiltrates on chest radiograph due to the lack of ability to characterize risk for poor outcome, and the difficulty in distinguishing between bilateral and unilateral involvement	
			Allow for definition to be used in patients supported on NIV	
			Specific criteria exist for those with chronic lung disease or cyanotic heart disease. These two conditions are often encountered in clinical practice	

Table 1 *(continued)*

Criteria	AECC (9), 1994	Berlin (10), 2012	PALICC (11), 2015	Montreux (12), 2017
Limitations	Definition for acute onset not provided	Focused mainly on adult ARDS	Neonates with ARDS excluded although the definition is independent of age of patients	Criteria for use of NIV has not been addressed
	PaO_2/FiO_2 does not take into account degree of ventilatory support required	Potential difficulty in obtaining invasive arterial measurement in children and underestimating ARDS prevalence in children		
	Inter-observer variation in interpreting PAWP and lack of consistency in clinical assessment of left atrial hypertension	Degree of support of positive pressure ventilation affects the PaO_2/FiO_2 ratio		
	Chest radiograph criteria not clearly defined	Differences in causes, comorbidities, pathophysiology and outcome between children and adults were not considered		
	Focused mainly on adult ARDS			
	No criteria for use of NIV			

J Emerg Crit Care Med 2018;2:24

		Onset	Chest Radiograph	Etiology	Failure of Oxygenation
1967 Ashbaugh	12 patients Lancet	Acute			
1994 American/European Consensus Statement	A=Adult	Acute	Bilateral infiltrates	Not secondary to heart failure: PA Pressure < 18 or no LA HTN	**Acute Lung Injury:** PaO2:FiO2 Ratio < 300 **ARDS :**PaO2:FiO2 Ratio < 200
2012 Berlin Criteria		Within 1 week of inciting event	Bilateral infiltrates not explained by effusions or atelectasis	NOT cardiac failure or fluid overload by some objective measure (eg, echocardiogram)	**Mild ARDS** PF ratio 201-300 on CPAP/PEEP ≥ 5 **Moderate ARDS** PF 101-200 on PEEP ≥ 5 **Severe ARDS** PF ≤ 100 on PEEP ≥ 5
2015 PALICC-PARDS	Pediatric Specific	Within 1 week of known inciting event	**Unilateral** or bilateral infiltrates not explained by effusions or atelectasis	NOT cardiac failure or fluid overload by some objective measure (eg, echocardiogram)	Not intubated :CPAP ≥ 5 with PF ≤ 300 or SF ≤ 264 **Mild ARDS** OI 4 to 8; OSI 5 to 7.5 **Moderate ARDS** OI 8 to 16; OSI 7.5 to 12.3 **Severe ARDS** OI ≥ 16; OSI ≥ 12.3

ORIGINAL RESEARCH ARTICLE

Front. Pediatr., 09 April 2018 | http://sci-hub.tw/https://doi.org/10.3389/fped.2018.00093

Comparison of Prevalence and Outcomes of Pediatric Acute Respiratory Distress Syndrome Using Pediatric Acute Lung Injury Consensus Conference Criteria and Berlin Definition

Samriti Gupta, Jhuma Sankar*, Rakesh Lodha and Sushil K. Kabra

Department of Pediatrics, All India Institute of Medical Sciences, New Delhi, India

Objectives: Our objective was to compare the prevalence and outcomes of pediatric acute respiratory distress syndrome using the Pediatric Acute Lung Injury Consensus Conference (PALICC) criteria and Berlin definitions.

Conclusión: En comparación con la definición de Berlín, los criterios PALICC identificaron más pacientes con SDRA. La proporción con SDRA grave y complicaciones fue mayor en el grupo "Berlín con o sin PALICC" en comparación con el grupo "PALICC solamente". No hubo diferencias en los resultados clínicos entre los grupos.

•Department of Pediatrics, All India Institute of Medical Sciences, New Delhi, India

Paediatric acute respiratory distress syndrome incidence and epidemiology (PARDIE): an international, observational study

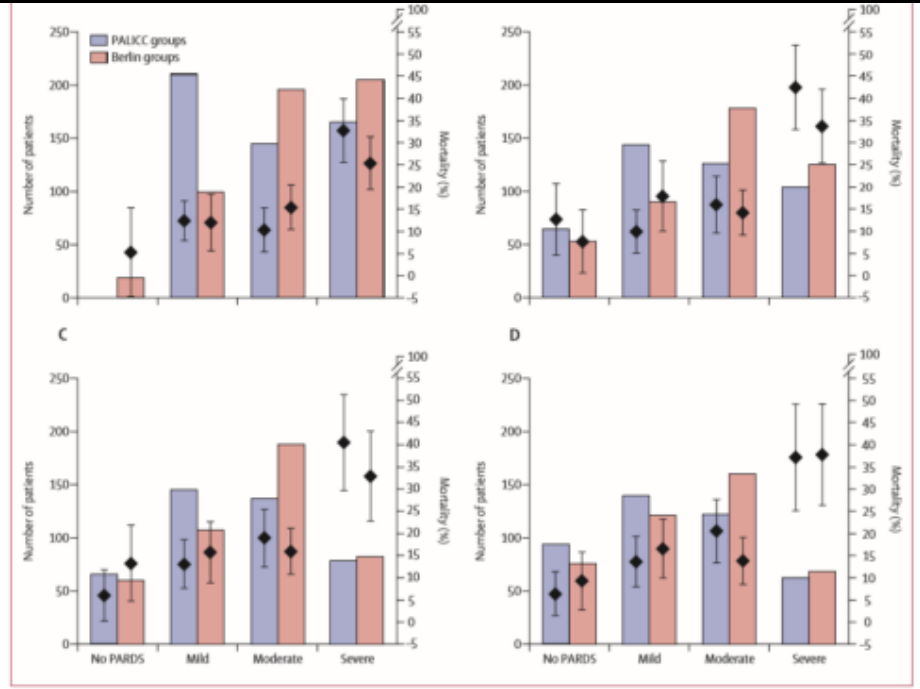


Figure 3: Severity of PARDS based on PALICC and Berlin stratification for patients initially on invasive mechanical ventilation (excluding at-home ventilation) at diagnosis (A), 6 h after diagnosis (B), 12 h after diagnosis (C), and 24 h after diagnosis (D)
PARDS severity by hypoxaemia metrics at distinct timepoints after PARDS diagnosis. PICU mortality for each group shown by datapoint with 95% CIs. ICU=intensive care unit. PALICC=Pediatric Acute Lung Injury Consensus Conference. PARDS=paediatric acute respiratory distress syndrome.

PALICC identificó a más niños con PARDS que Berlín, y los grupos de gravedad de PALICC PARDS mejoraron la estratificación del riesgo de mortalidad, especialmente cuando se aplicaron 6 h después del diagnóstico de PARDS.

www.thelancet.com/respiratory Published online October 22, 2018 http://dx.doi.org/10.1016/S2213-2600(18)30344-8

Causes of acute respiratory distress syndrome

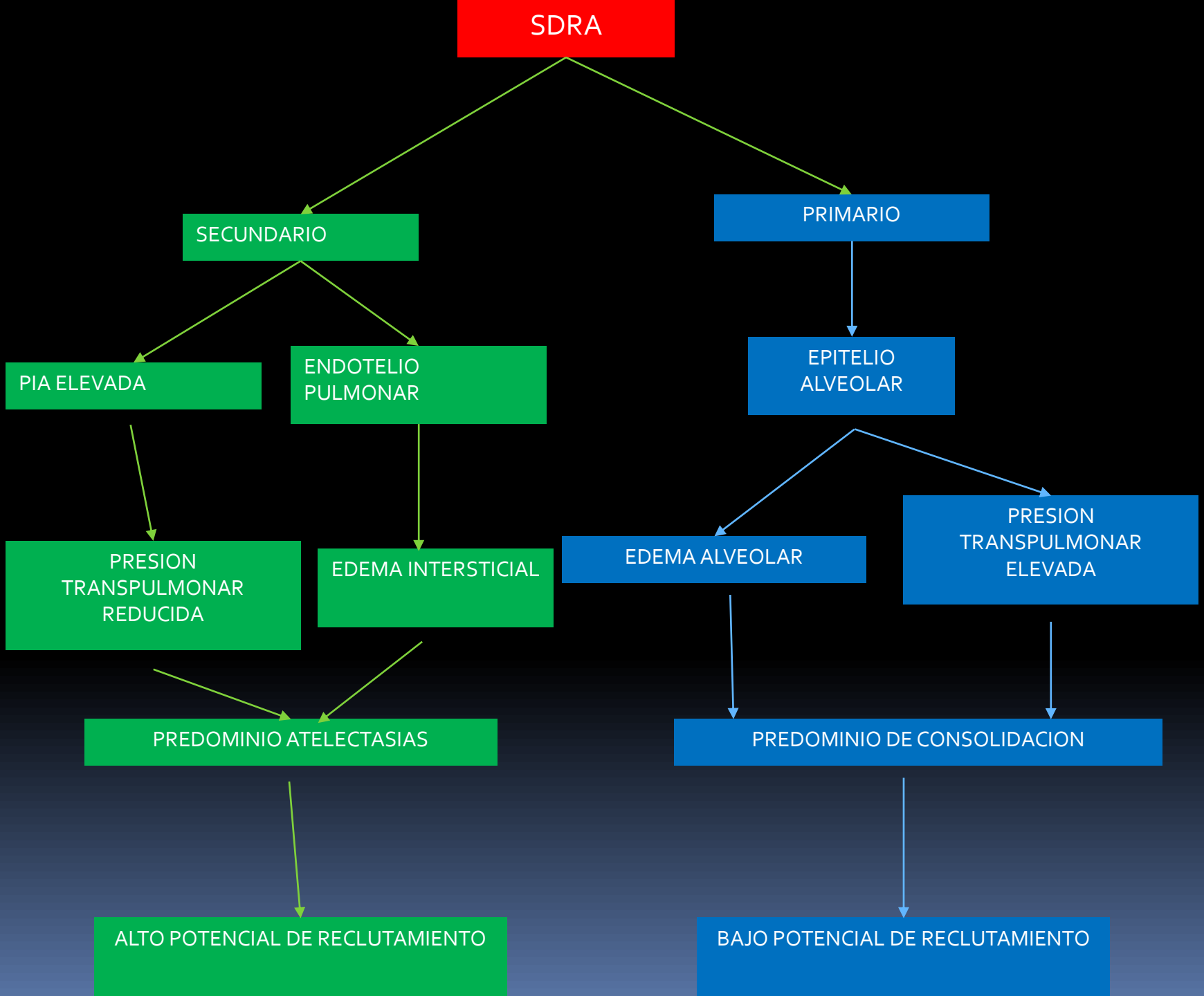
Direct Lung Injury (Alveolar-Epithelial)

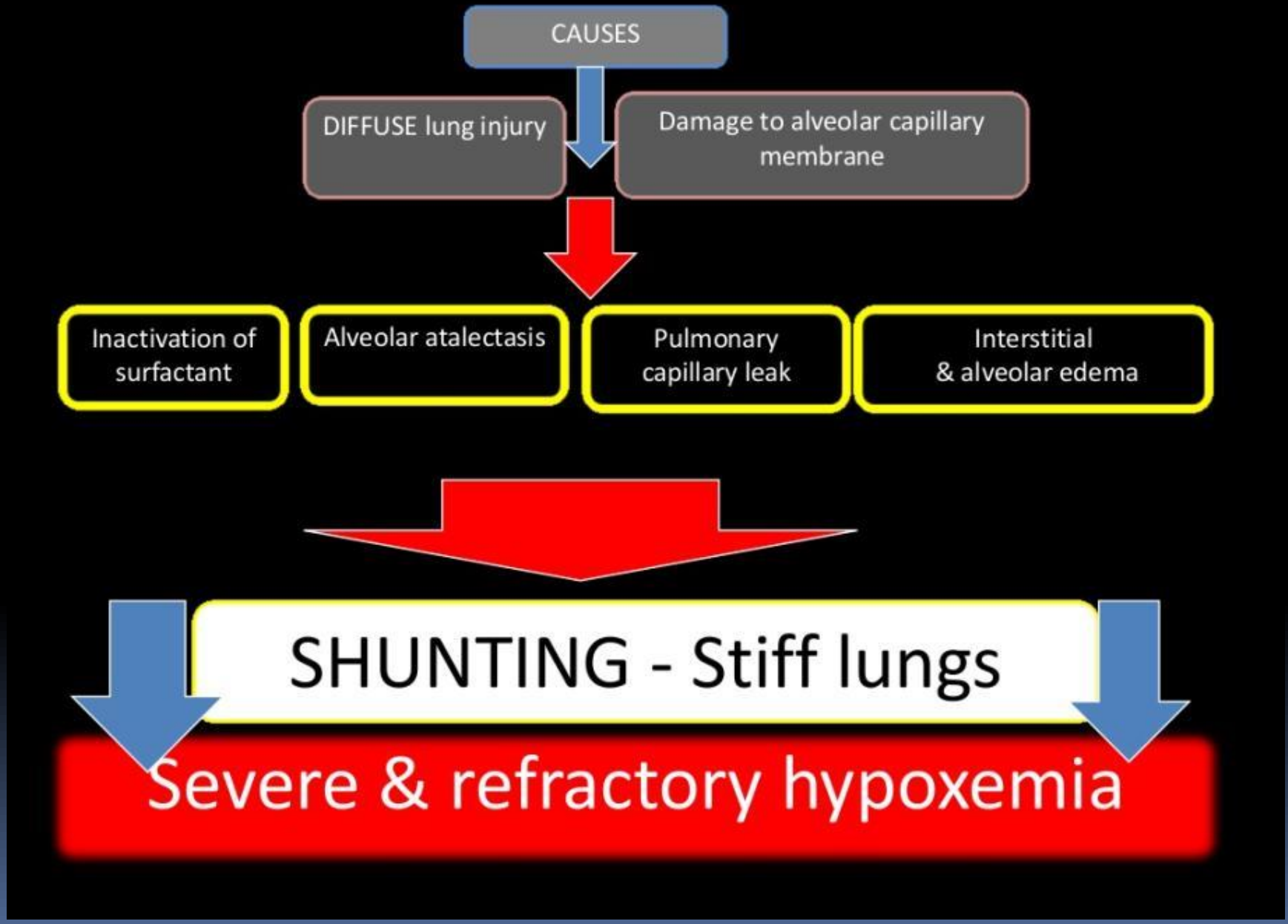
Pneumonia
Aspiration
Inhalation injury
Drowning
Pulmonary contusion

Indirect Lung Injury (Alveolar-Capillary)

Sepsis
Systemic inflammatory response syndrome
Major trauma
Pancreatitis Severe burns Massive transfusion or TRALI
Shock
Cardiopulmonary bypass
Head injury Drug overdose

Pediatr Clin N Am 64 (2017) 1017–1037

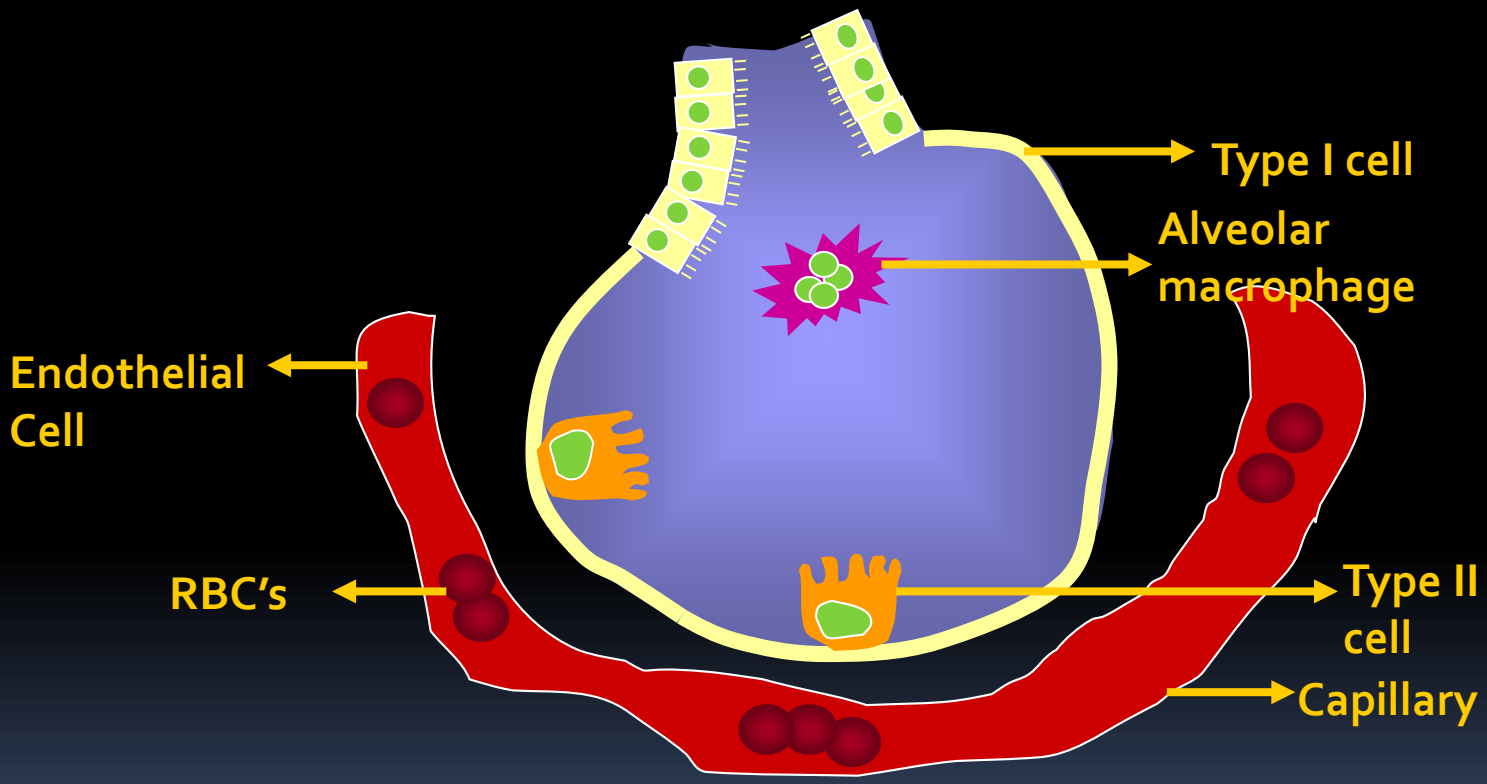

SDRA
SECUNDARIO
PRIMARIO
PIA ELEVADA
ENDOTELIO PULMONAR
EPITELIO ALVEOLAR
PRESION TRANSPULMONAR REDUCIDA
EDEMA INTERSTICIAL
EDEMA ALVEOLAR
PRESION TRANSPULMONAR ELEVADA
PREDOMINIO ATELECTASIAS
PREDOMINIO DE CONSOLIDACION
ALTO POTENCIAL DE RECLUTAMIENTO
BAJO POTENCIAL DE RECLUTAMIENTO

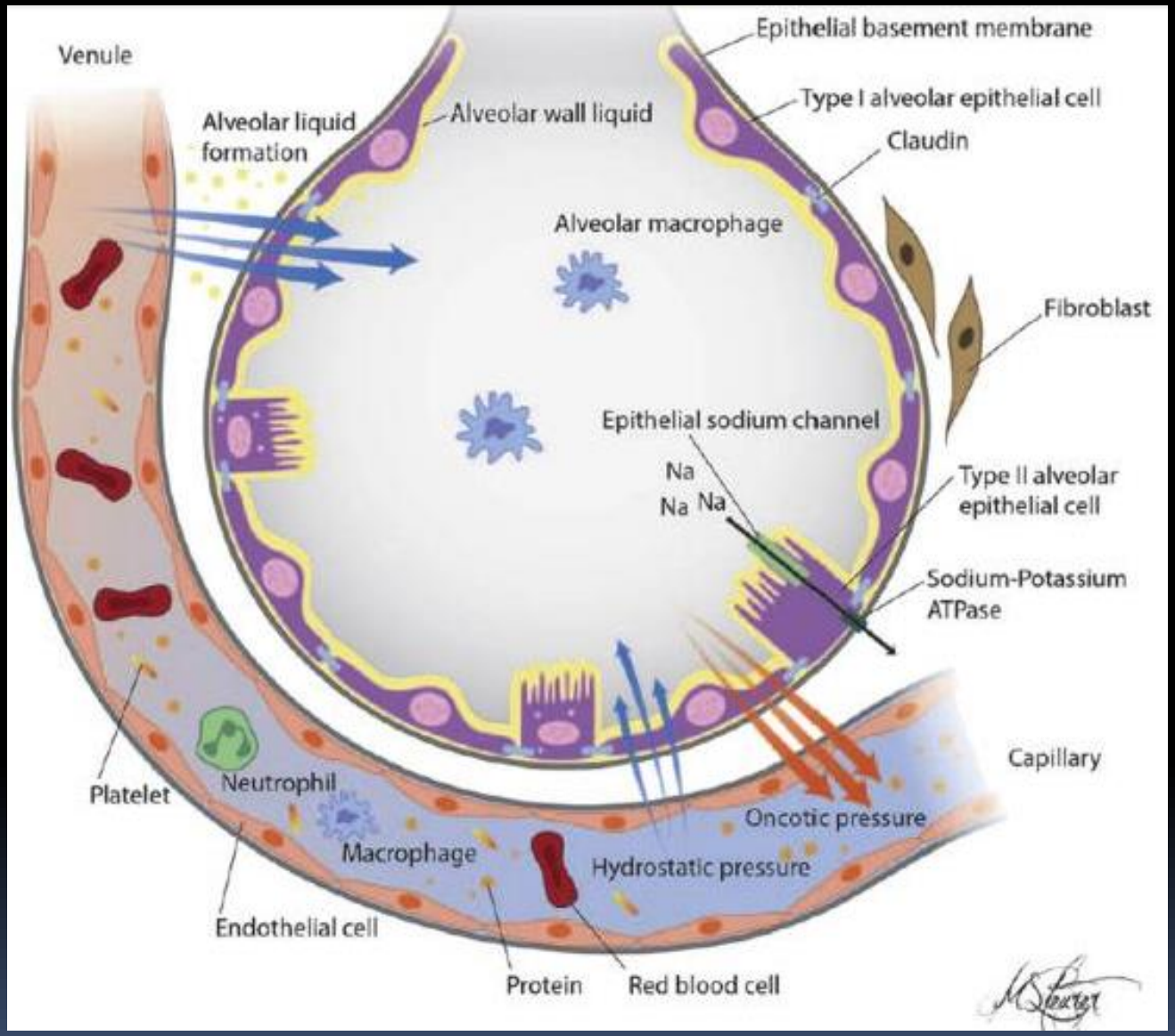

CAUSES
DIFFUSE lung injury
Damage to alveolar capillary membrane
Inactivation of surfactant
Alveolar atalectasis
Pulmonary capillary leak
Interstitial & alveolar edema
SHUNTING - Stiff lungs
Severe & refractory hypoxemia

PATHOGENESIS

Presencia de mediadores inflamatorios
Daños al endotelio microvascular
Daños al epitelio alveolar
Aumento de permeabilidad alveolar con la acumulación de líquido de edema alveolar.

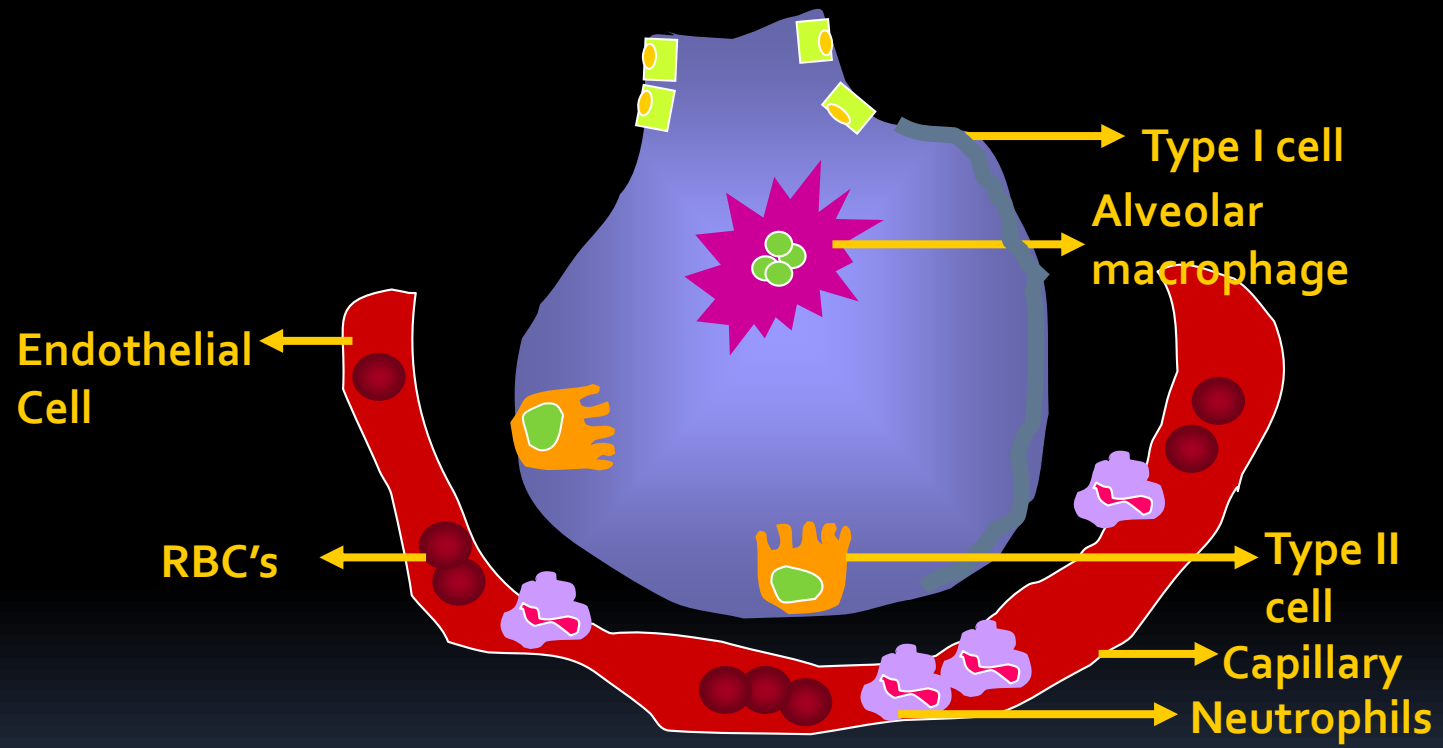
NORMAL ALVEOLUS

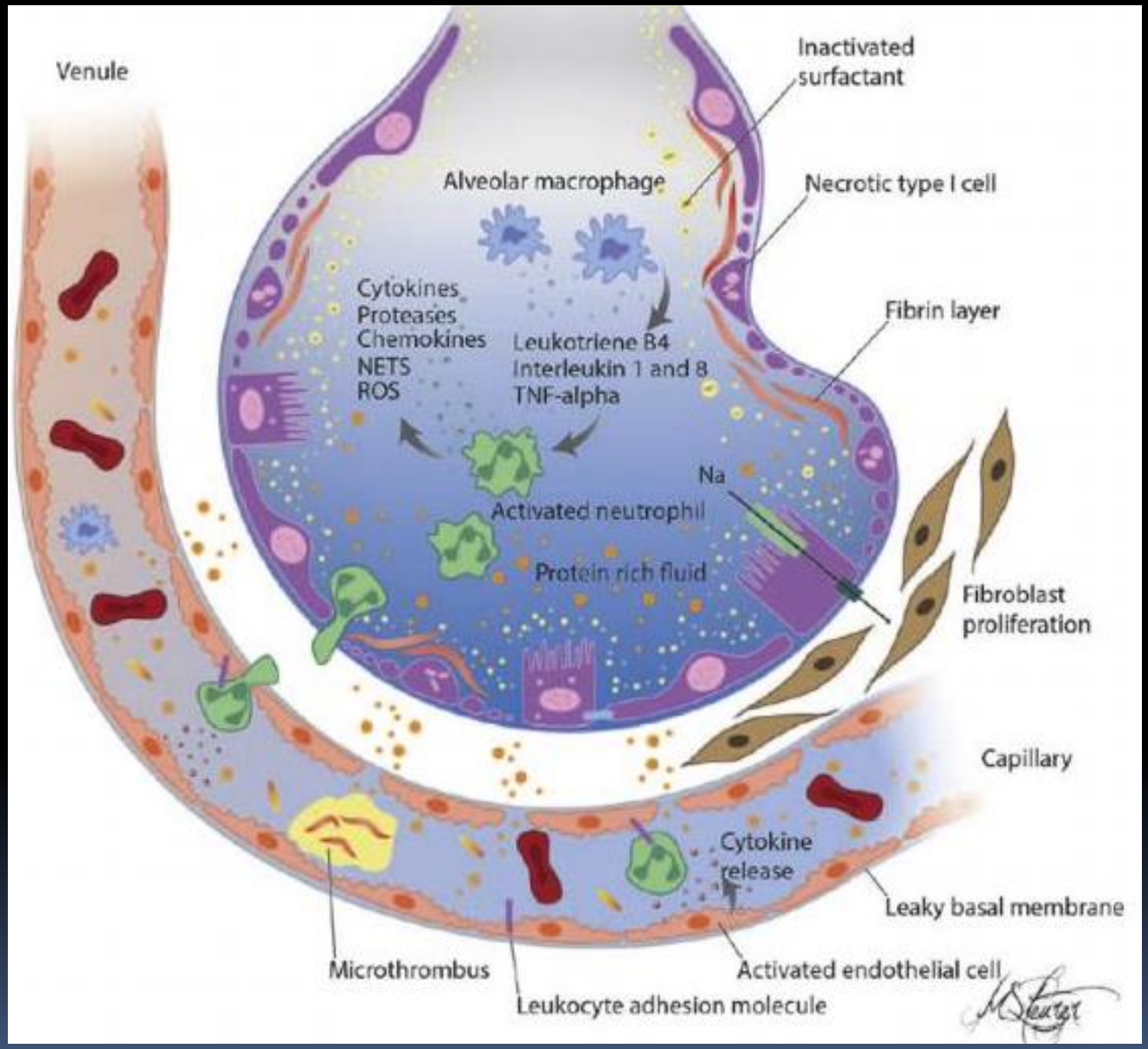




Pediatr Clin N Am 64 (2017) 1017–1037

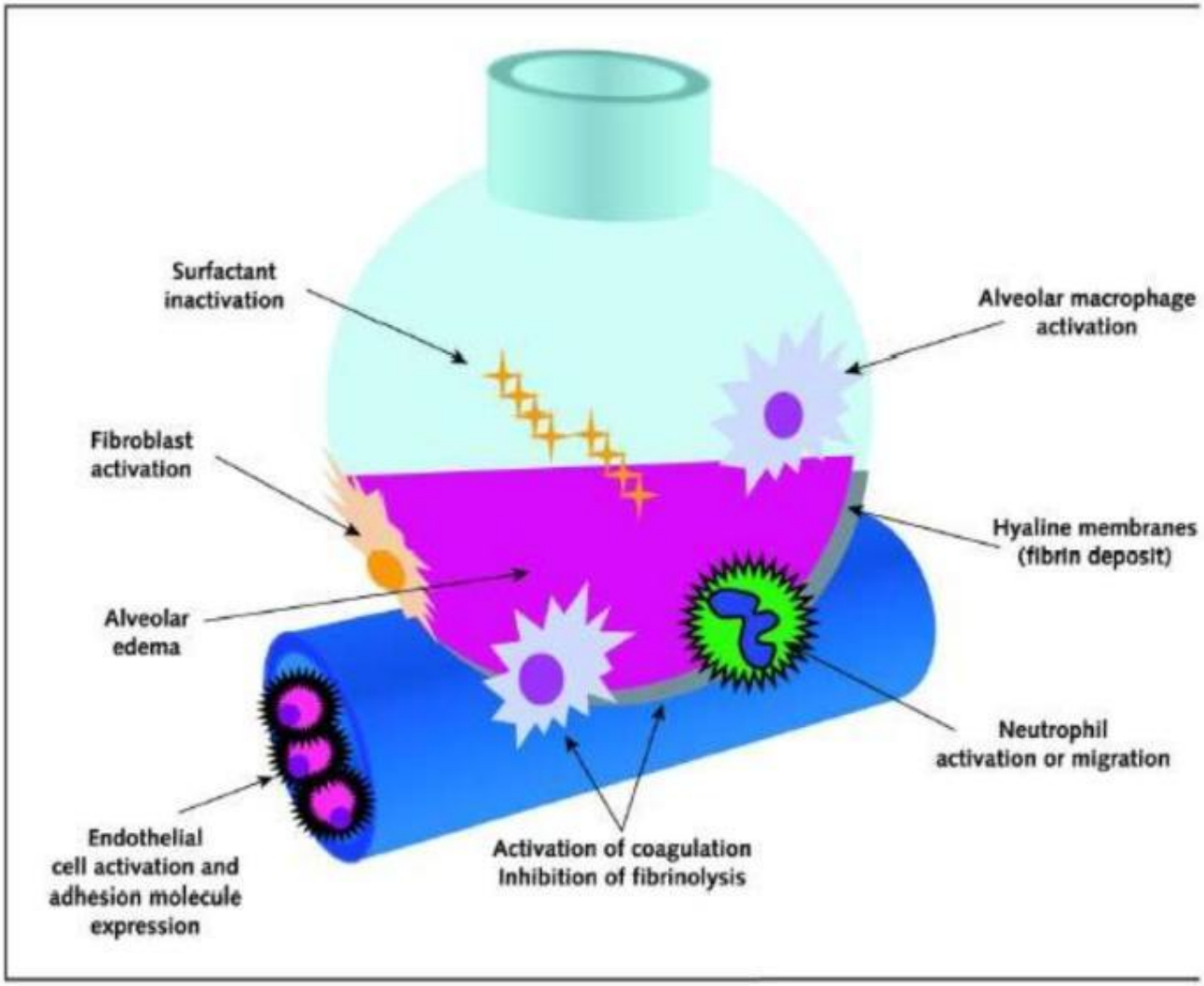
ACUTE PHASE OF ARDS

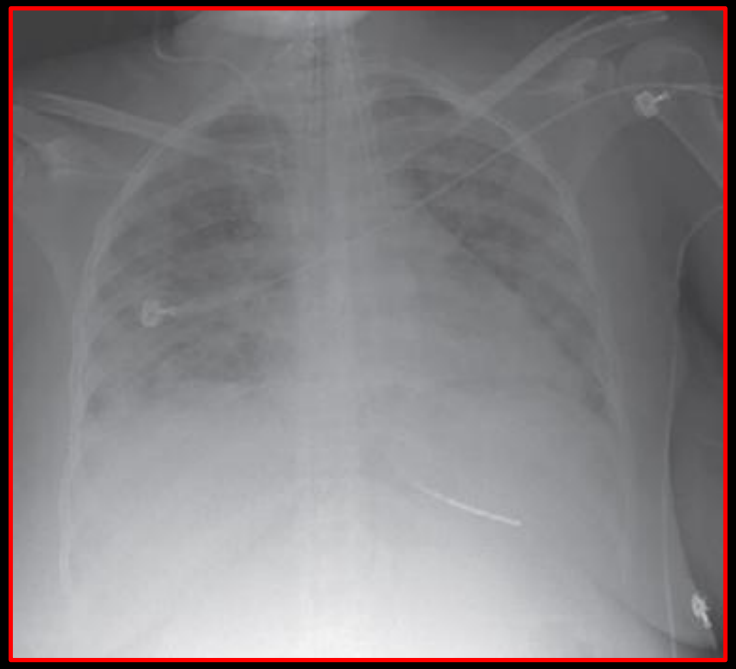


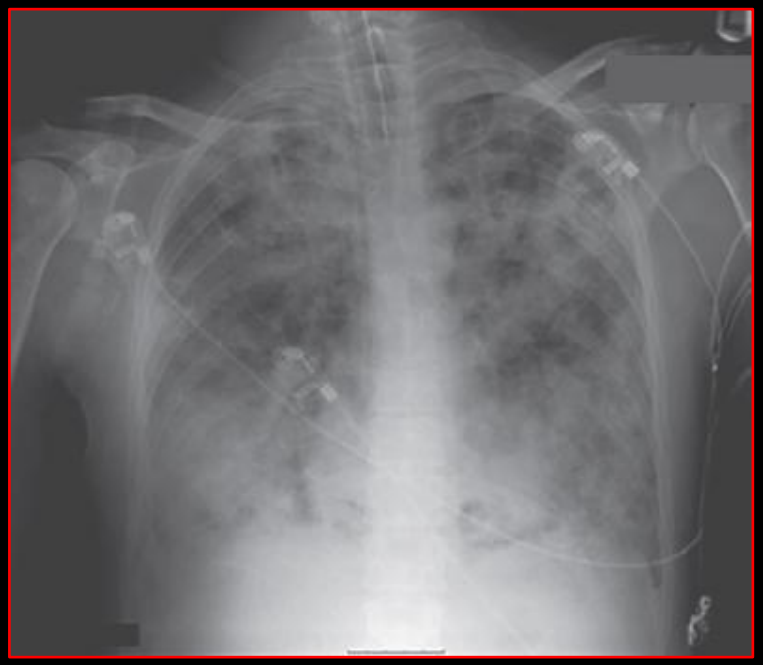


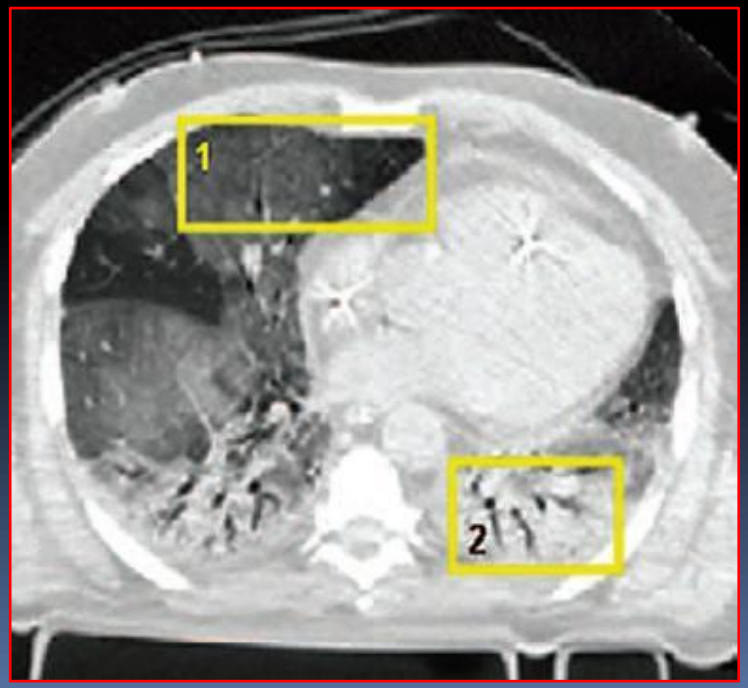
Pediatr Clin N Am 64 (2017) 1017–1037

Cellular and molecular events that interfere with gas exchange in the acute respiratory distress syndrome.



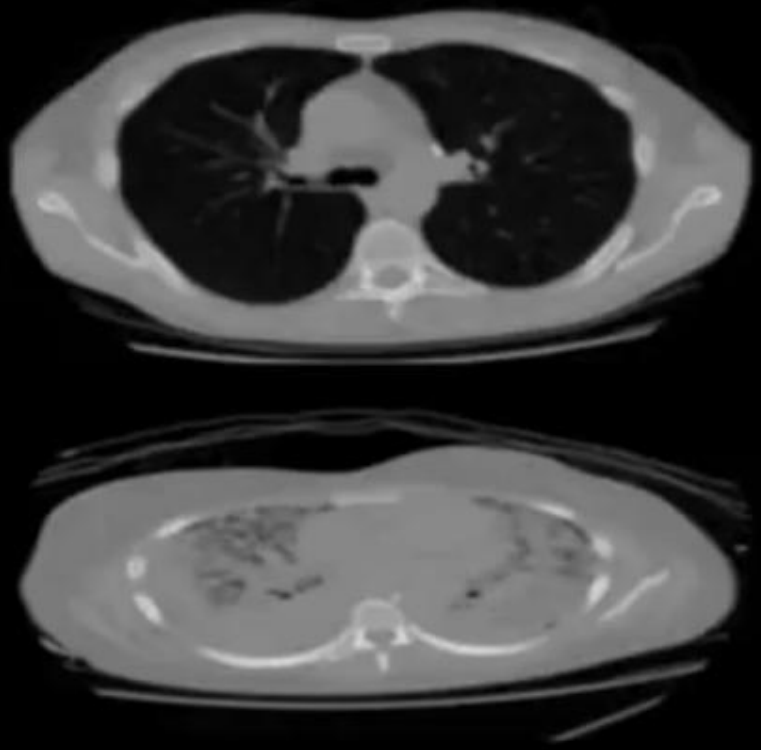


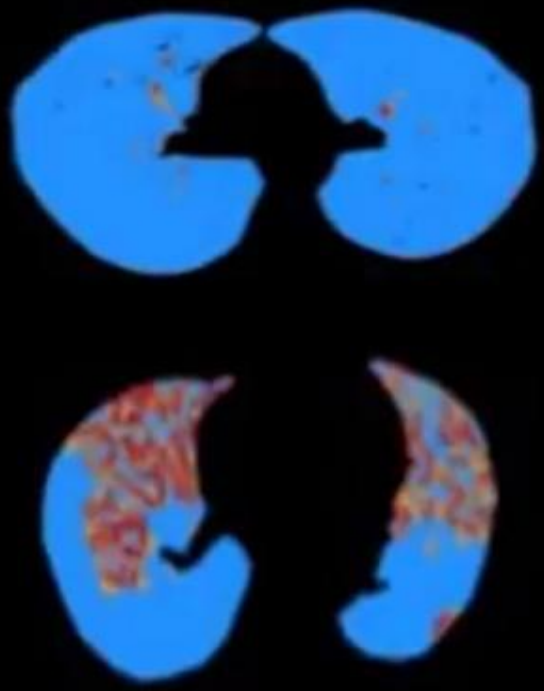




N engl j med 377;6 nejm.org August 10, 2017

Pulmón in homogéneo





Am J Respir Crit Care Med. 2014 Jan 15;189(2):149-58.

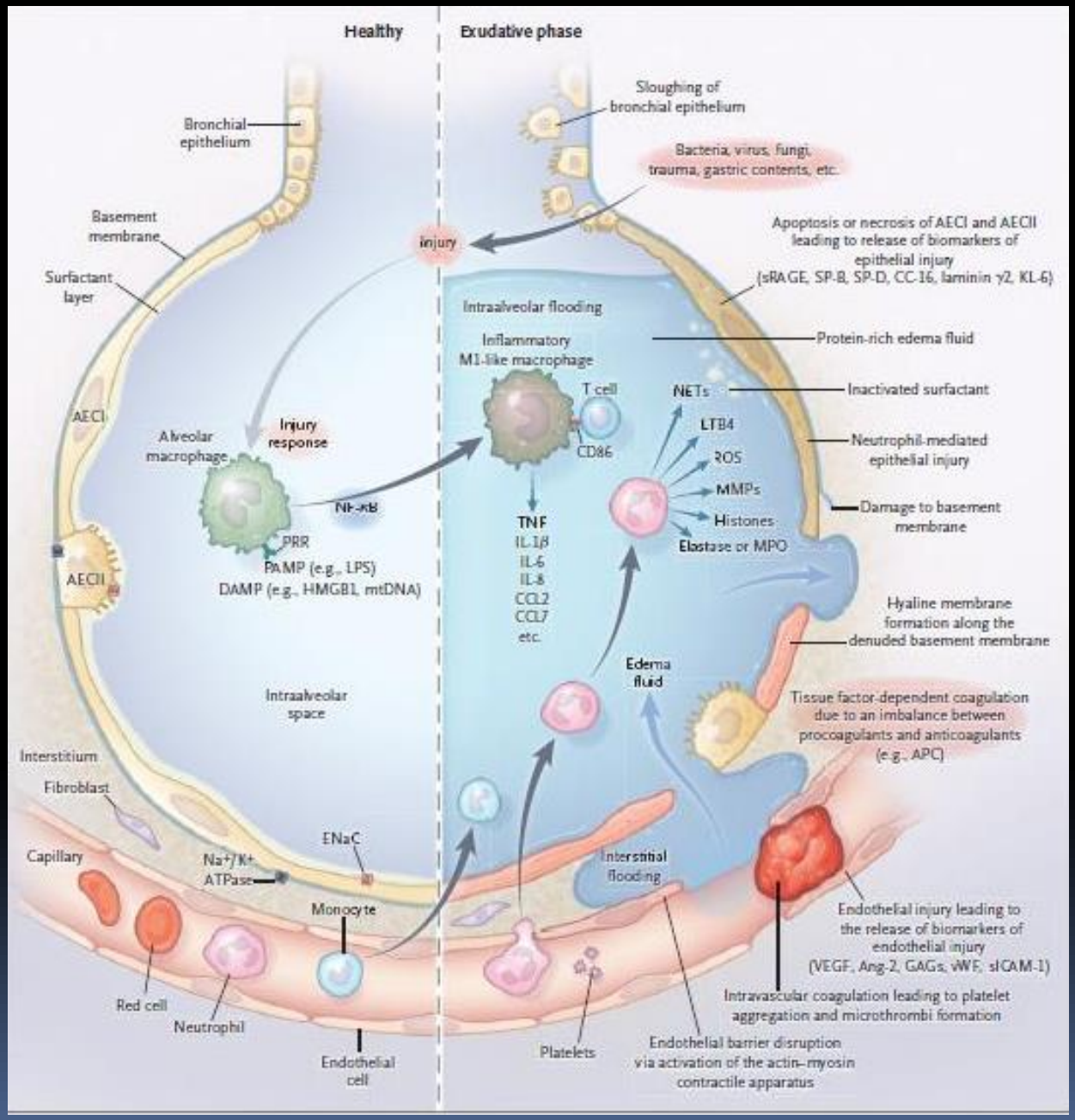
PATOGENIA

3 Etapas:

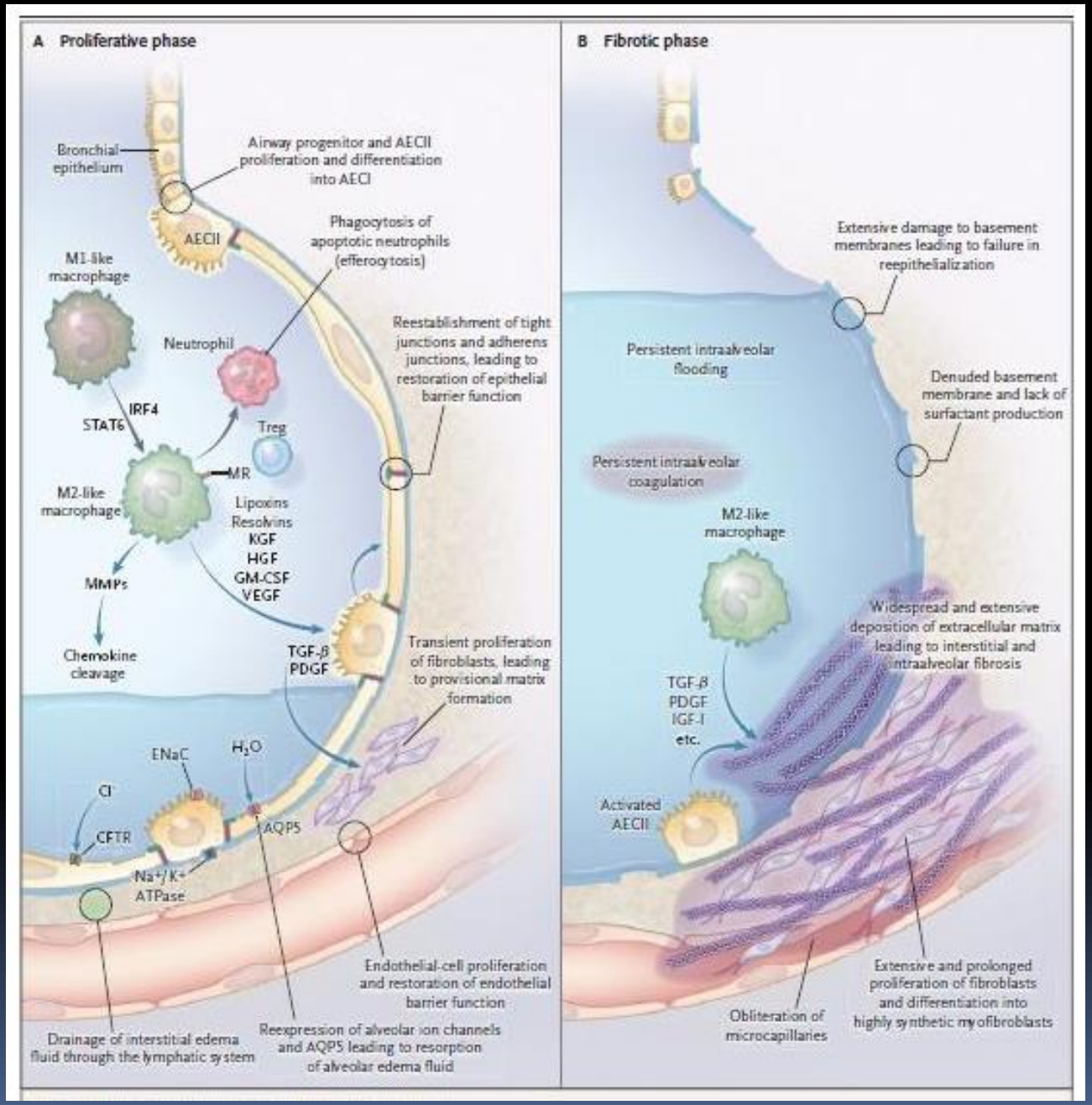
1) EXUDATIVA : * Edema pulmonar lesional
* Surfactante
* Membranas hialinas
* Respuesta inflamatoria

2) FIBROPROLIFERATIVA

3) FIBROSIS



N Engl j med 377;6 nejm.org August 10, 2017



N Engl j med 377;6 nejm.org August 10, 2017

FISIOPATOLOGIA (1)

INTERCAMBIO DE GASES

- **IRA tipo I**
- **Dif. Aa aumentada**
- **Refractaria a altas concentraciones de Oxígeno**

→ **SHUNT**

FISIOPATOLOGIA (2)

MECANICA PULMONAR

- Disminución de la compliance
- Disminución de la CRF
- Aumento de las resistencias VA
- Aumento del trabajo respiratorio

FISIOPATOLOGIA (3)

HEMODINAMIA

- **Aumento de RVP**
- **Fase temprana: * Vasoconsticción**
  *** Microtrombosis**
- **Fase tardía: * Destrucción vascular**

TRATAMIENTO

OBJETIVOS DE LA VENTILACION MECANICA EN EL SDRA

- **MEJORAR OXIGENACION**
- **EVITAR LPAVM**

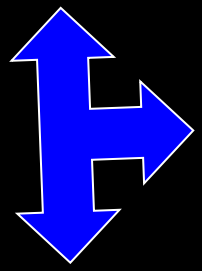
TRATAMIENTO

MEJORAR OXIGENACION

- **PEEP**
- **MANIOBRA DE RECLUTAMIENTO**
- **DECUBITO PRONO**

TRATAMIENTO

EVITAR LPAVM



PEEP OPTIMA

EVITAR DESRECLUTAMIENTO

VC BAJOS

Se puede desarrollar una hipoxemia refractaria grave en un subconjunto de pacientes con SDRA grave.

El SDRA grave a menudo se asocia con hipoxemia refractaria, y es obligatorio la identificación y el tratamiento tempranos de la hipoxemia.

Intensive Care Med (2016) 42:699–711
DOI 10.1007/s00134-016-4325-4

TRATAMIENTO

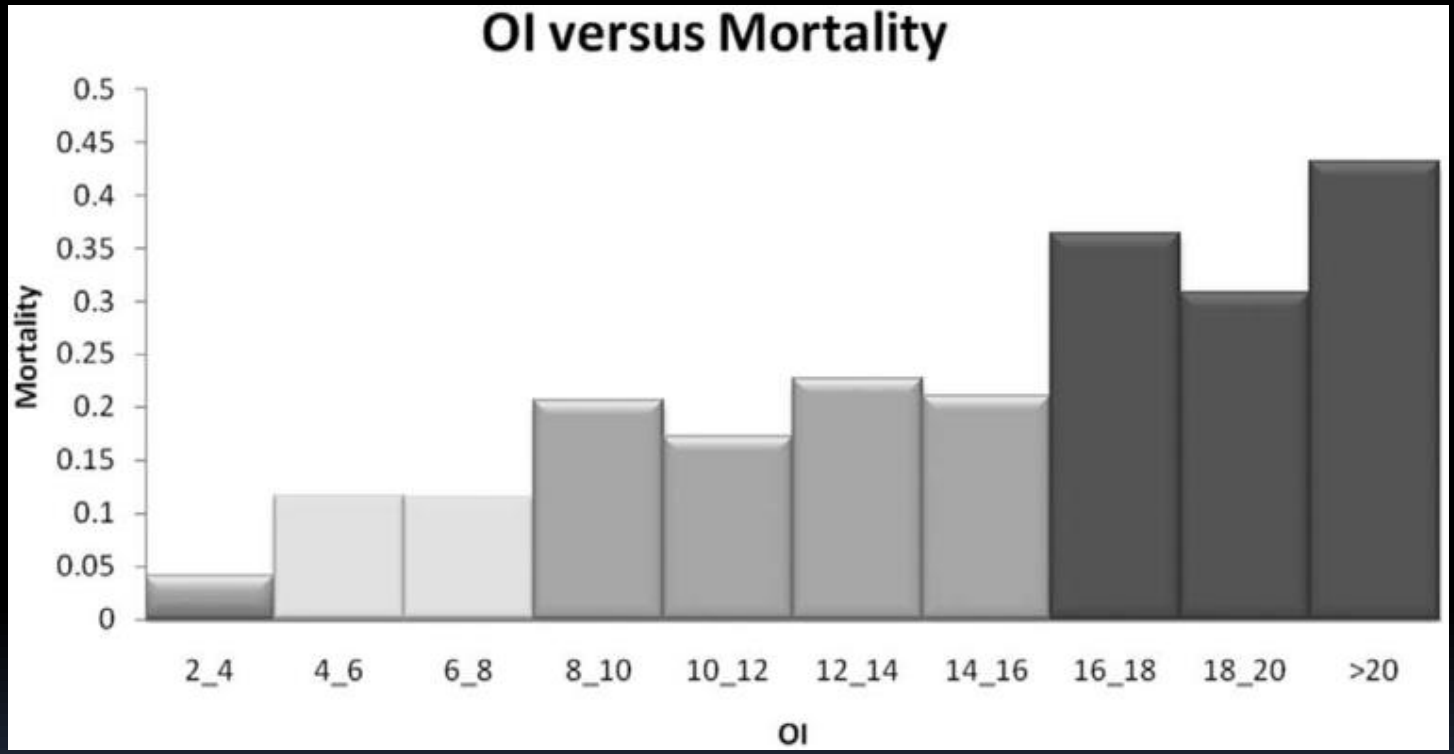
Table 2
Clinician recognition and application of adjunctive therapies in patients with acute respiratory distress syndrome

	Total MV + ARDS (n = 2377)	Severe ARDS (n = 729)
Clinician Recognition	1525 (64.2%)	437 (78.5%)
Recruitment Maneuver	496 (20.9%)	238 (32.7%)
HFOV	28 (1.2%)	11 (1.5%)
Neuromuscular Blockade	516 (21.7%)	274 (37.8%)
Prone Positioning	187 (7.9%)	119 (16.3%)
Inhaled Vasodilators	182 (7.7%)	95 (13.0%)
ECMO	76 (3.2%)	48 (6.6%)
Any of the Above	946 (39.8%)	445 (61.0%)

Abbreviation: MV, mechanical ventilation.

Adapted from Bellani G, Laffey JG, Pham T, et al. 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.

Pediatric Acute Respiratory Distress Syndrome: Definition, Incidence, and Epidemiology: Proceedings From the Pediatric Acute Lung Injury Consensus Conference



Distribution of initial oxygenation index (OI) and mortality from Children's Hospital of Los Angeles dataset (Khemani et al [6]) (*n* = 397). Mortality increases as OI increases, but there appear to be clear groups where mortality steps up (OI, < 4, 4–8, 8–16, and > 16).

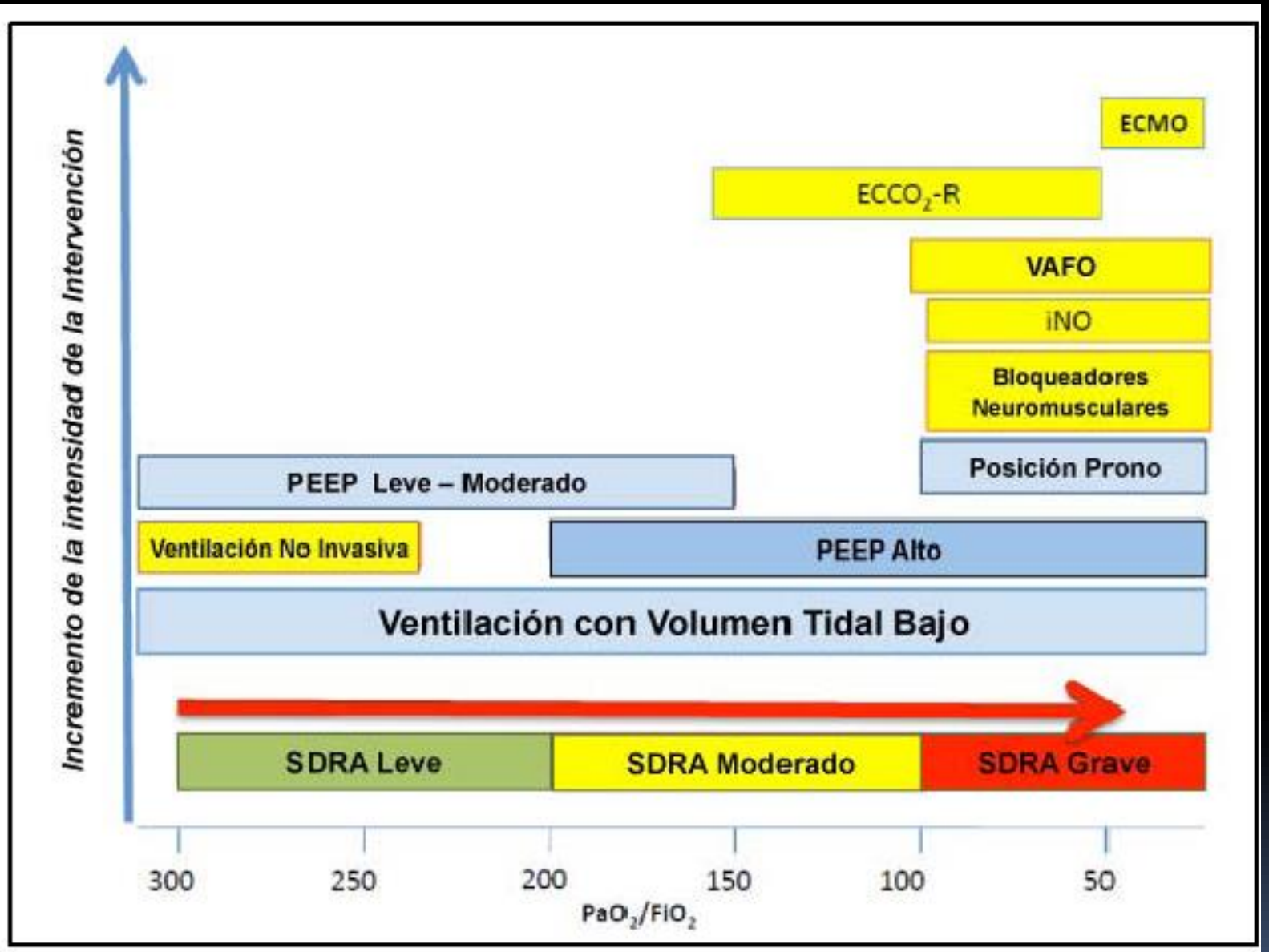
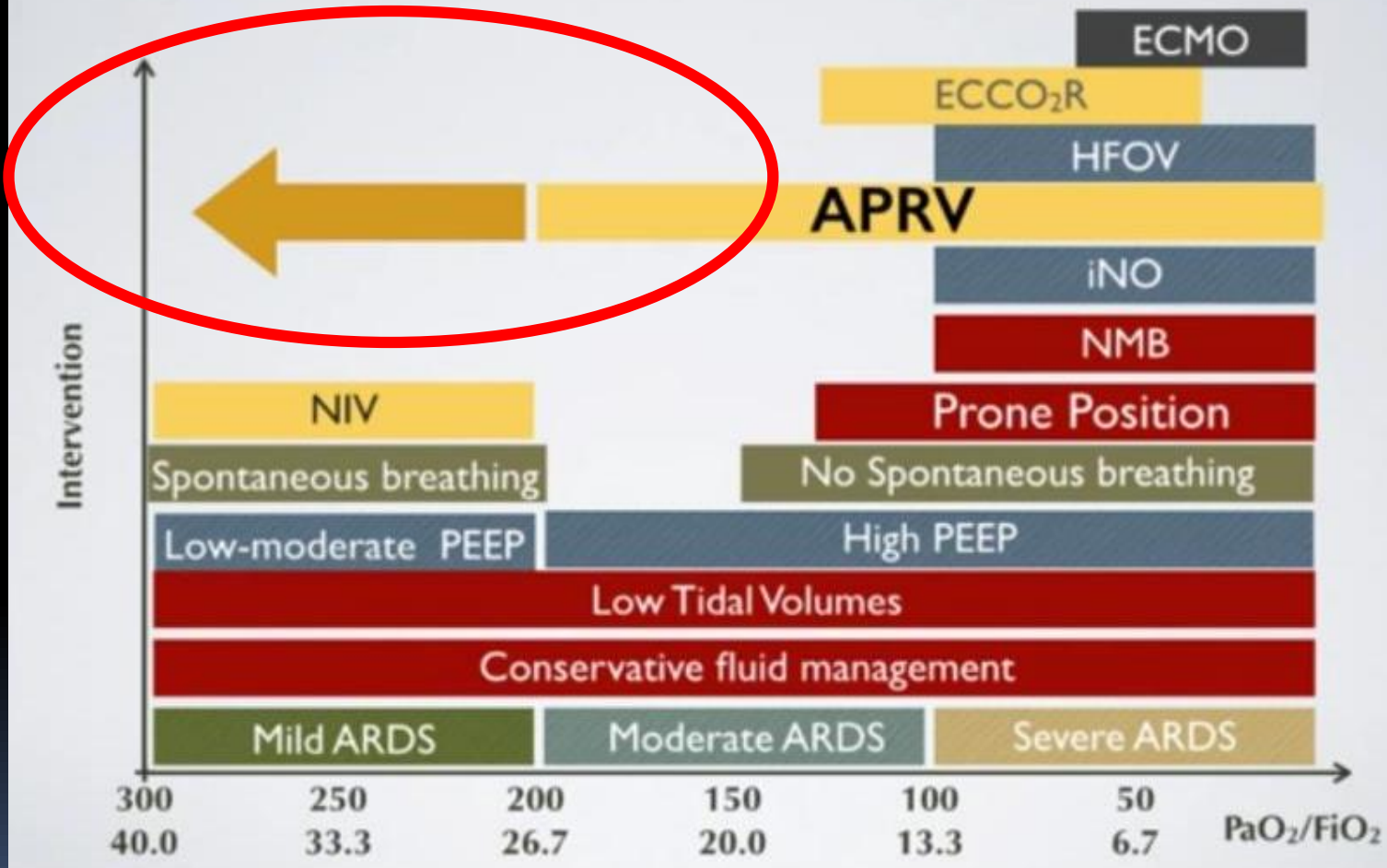


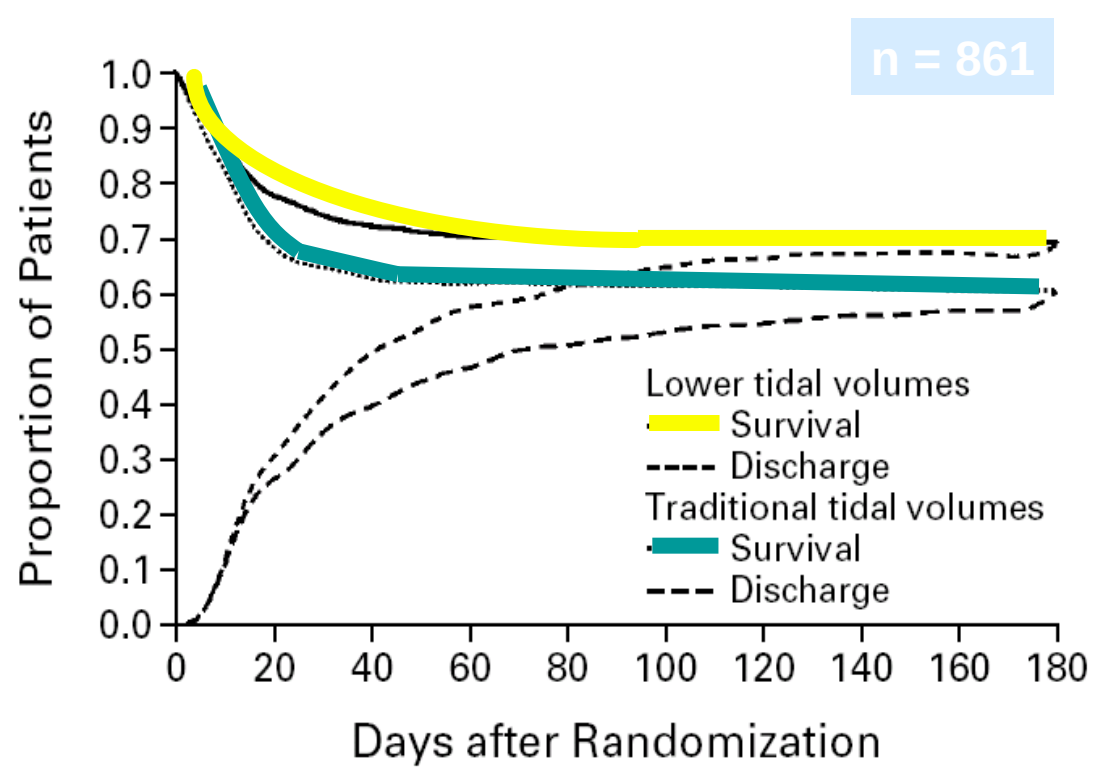
Figura 2. Estrategias de tratamiento según la gravedad del síndrome de distrés respiratorio agudo (Ref. 10).

THERAPEUTIC OPTIONS IN ARDS



Small Vt ventilation

ARDS network trial (6 vs.12 ml/kg)



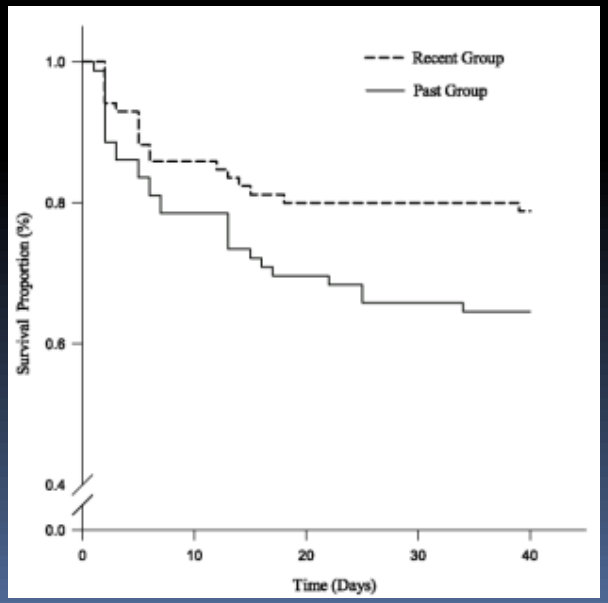
Mortality: 31 vs. 38 ($p < 0.007$)

PIP: 32 vs. 39 cmH_2O
Pplat: 25 vs. 33 cmH_2O

NEJM 2000;342:1301-1308

Have changes in ventilation practice improved outcomes ?

	Past (1988–1992) (n = 79)	Recent (2000–2004) (n = 85)	p^a Value
V_T, mL·kg^{-1}	10.2 ± 1.7 (211)	8.1 ± 1.4 (233)	<.001
PIP, cm H_2O	31.5 ± 7.3 (223)	27.8 ± 4.2 (233)	<.001
PEEP, cm H_2O	6.1 ± 2.7 (223)	7.1 ± 2.4 (232)	.007
$Paco_2$, mm Hg	37.0 ± 5.0 (225)	47.2 ± 11.8 (231)	<.001
Pao_2, mm Hg	84.4 ± 14.4 (225)	78.9 ± 14.9 (245)	.017
OI	14.7 ± 5.0 (223)	17.7 ± 5.3 (232)	<.001
Pao_2/Fio_2	153.0 ± 59.9 (225)	139.2 ± 53.1 (239)	.12
VI	28.4 ± 13.6 (225)	28.6 ± 15.6 (235)	.94
PC, %	52 (225)	55 (245)	.99
VC, %	47 (225)	37 (245)	.02
HFOV, %	1 (225)	8 (245)	<.001



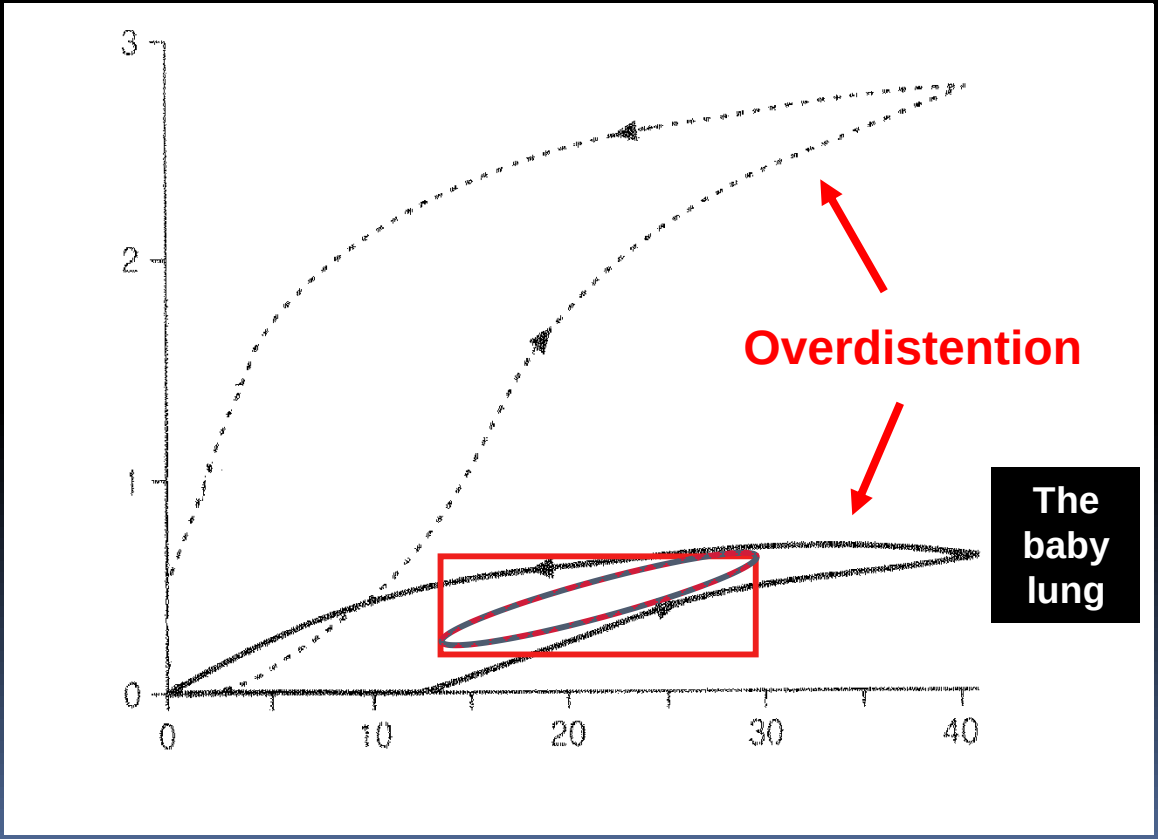
Albuali WH Pediatr Crit Care Med 2007; 8:324 –330

The baby lung

The ARDS lung is small, with a normal portion having the dimension of the lung of a 5- to 6- year old child

(200 – 300 g of lung tissue as compared to 700 g)

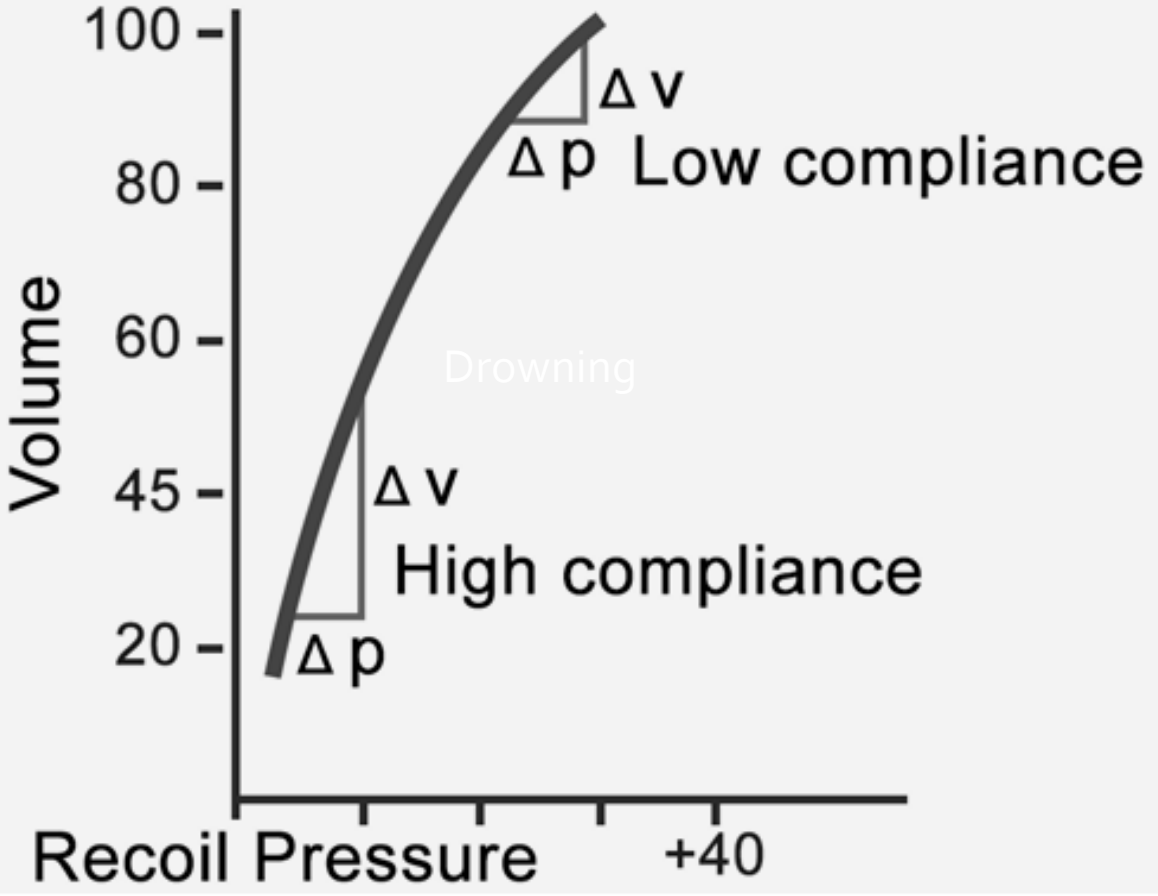
Gattinoni L, Pesenti A Intensive Care Crit Dig 1987; 6:1-4



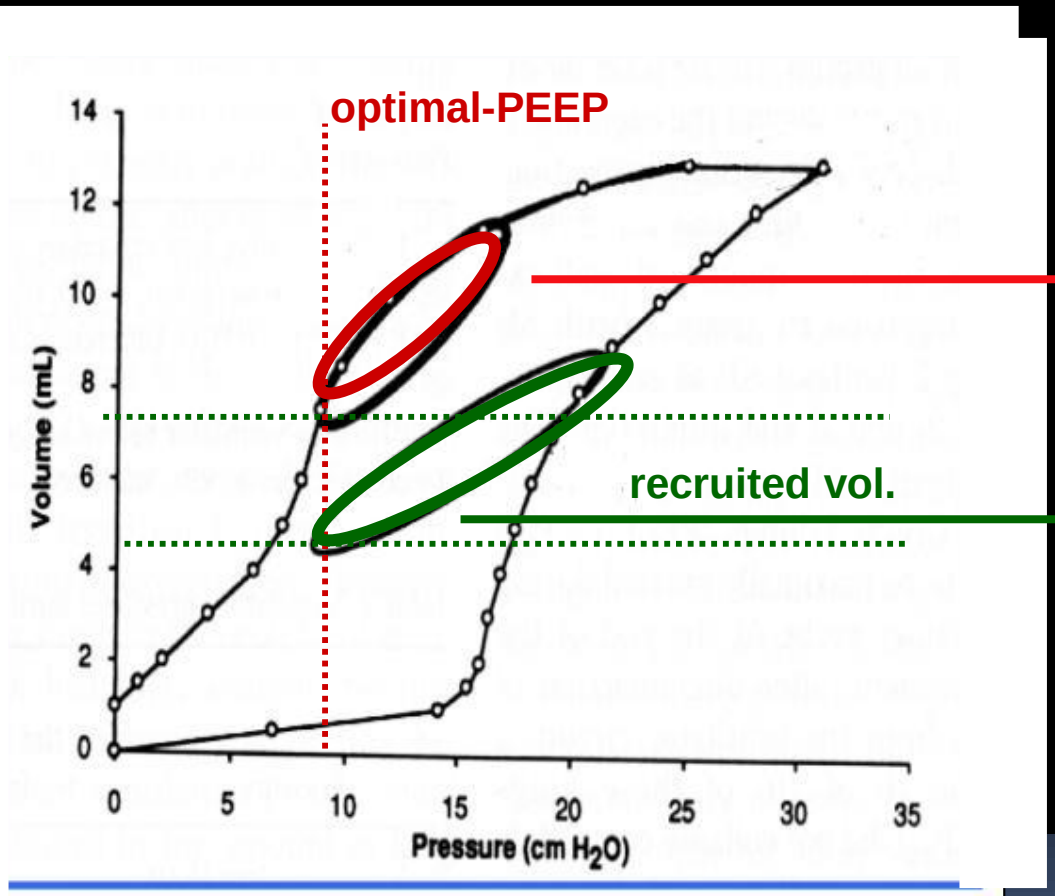
~~Vt / kg ratio~~

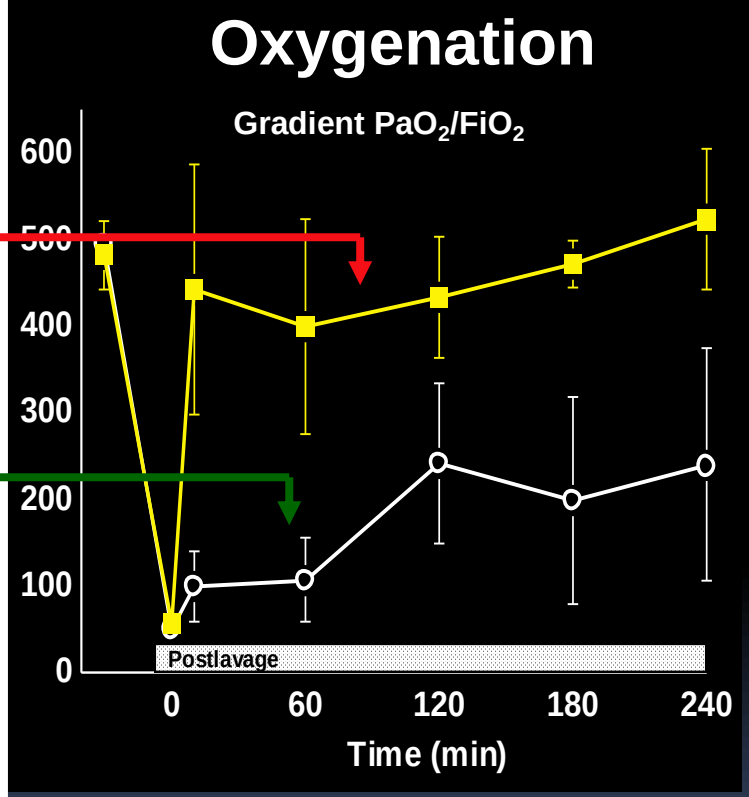
Vt / "baby lung" ratio

LUNG COMPLIANCE CURVE



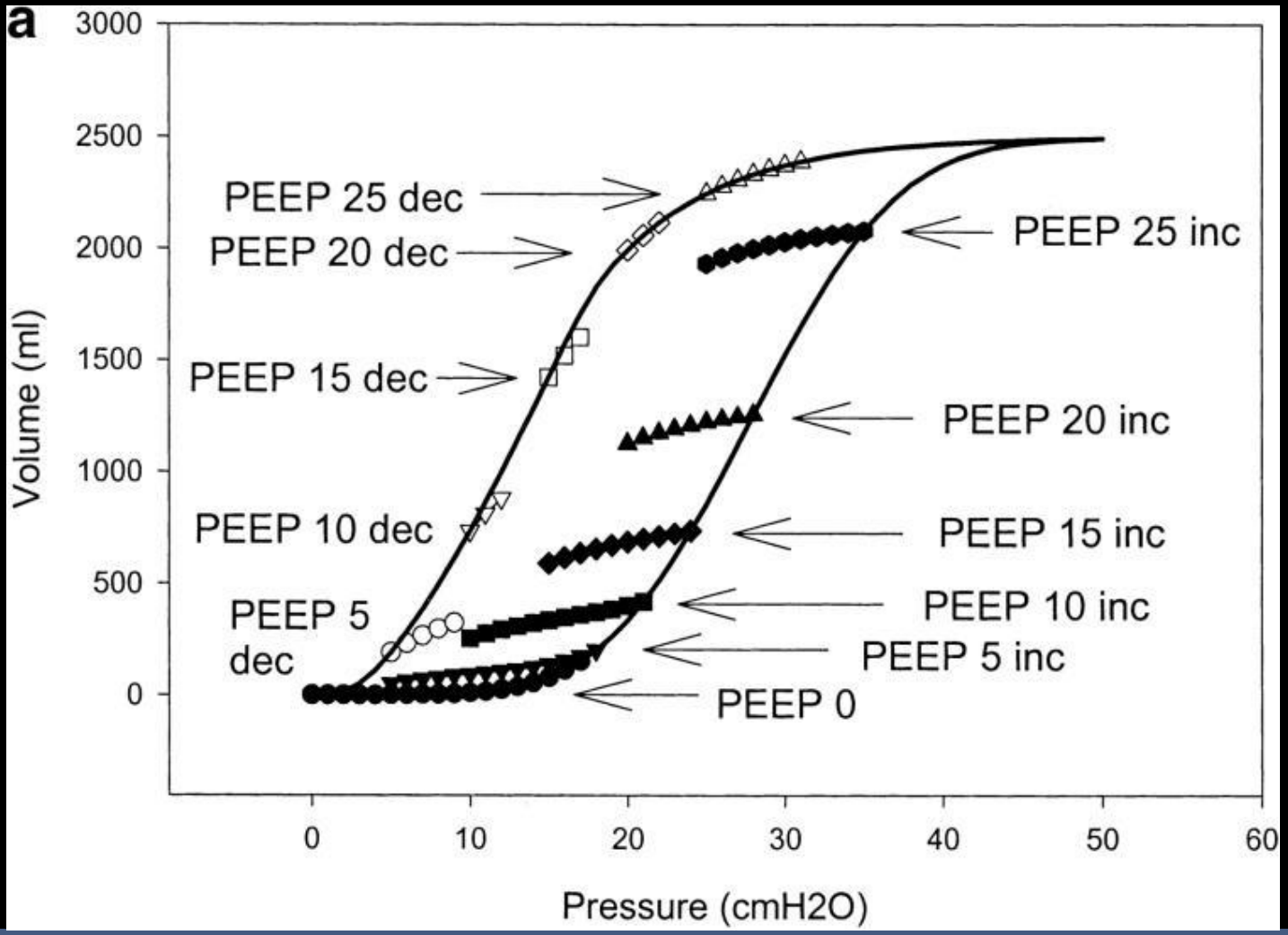
Oxygenation response in two groups; with and without recruitment (identical PEEP)





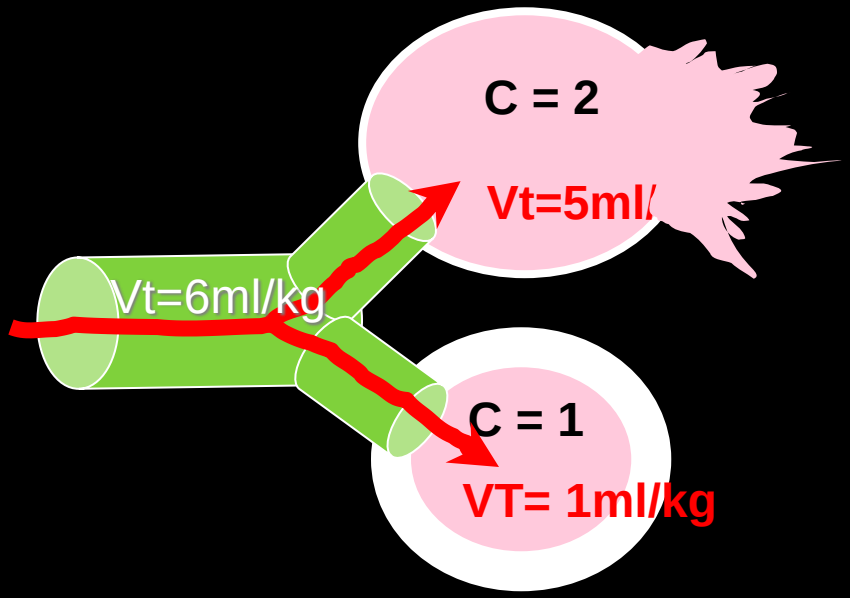
Rimensberger PC Crit Care Med 1999; 27:1946-52

Optimal = “Maximum dynamic compliance and best oxygenation at the least pressure required”



Hickling KG et al. AJRCCM 2001; 163:69-78

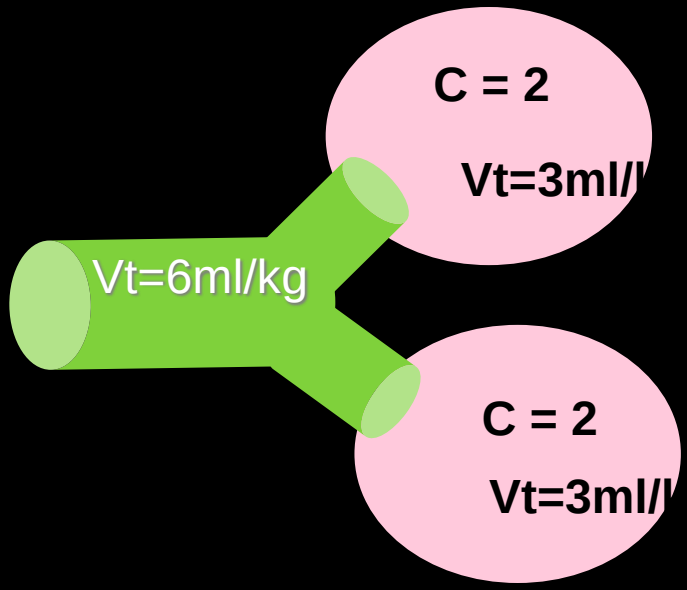
ARDS / RDS lung
(Heterogeneous)



→ various time constants

→ heterogeneous
Vt distribution

Normal lung / recruited lung
at optimal lung volumes

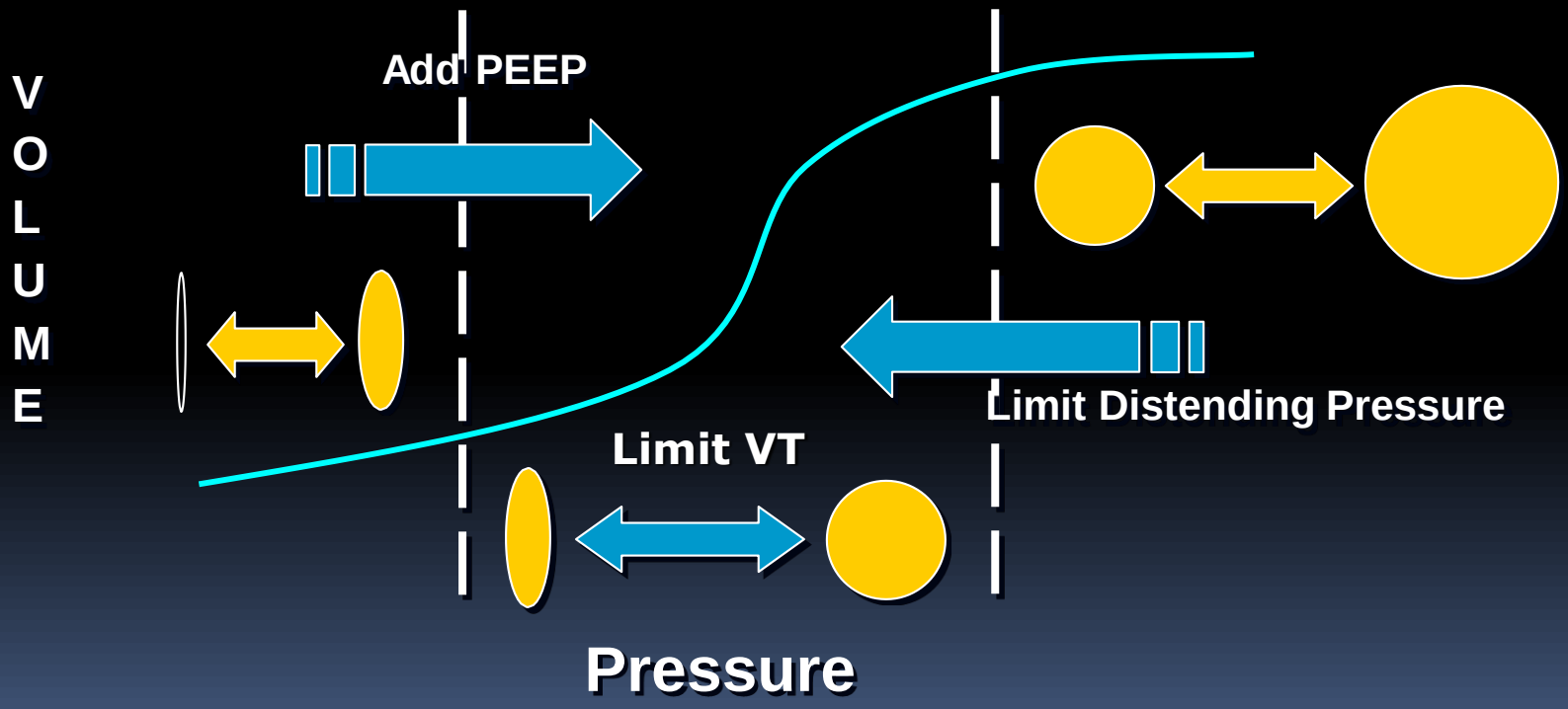


→ similar time constants

→ homogeneous
Vt distribution

Preventing Overdistention and Collapse Injury

"Lung Protective" Ventilation



LUNG PROTECTIVE STRATEGY

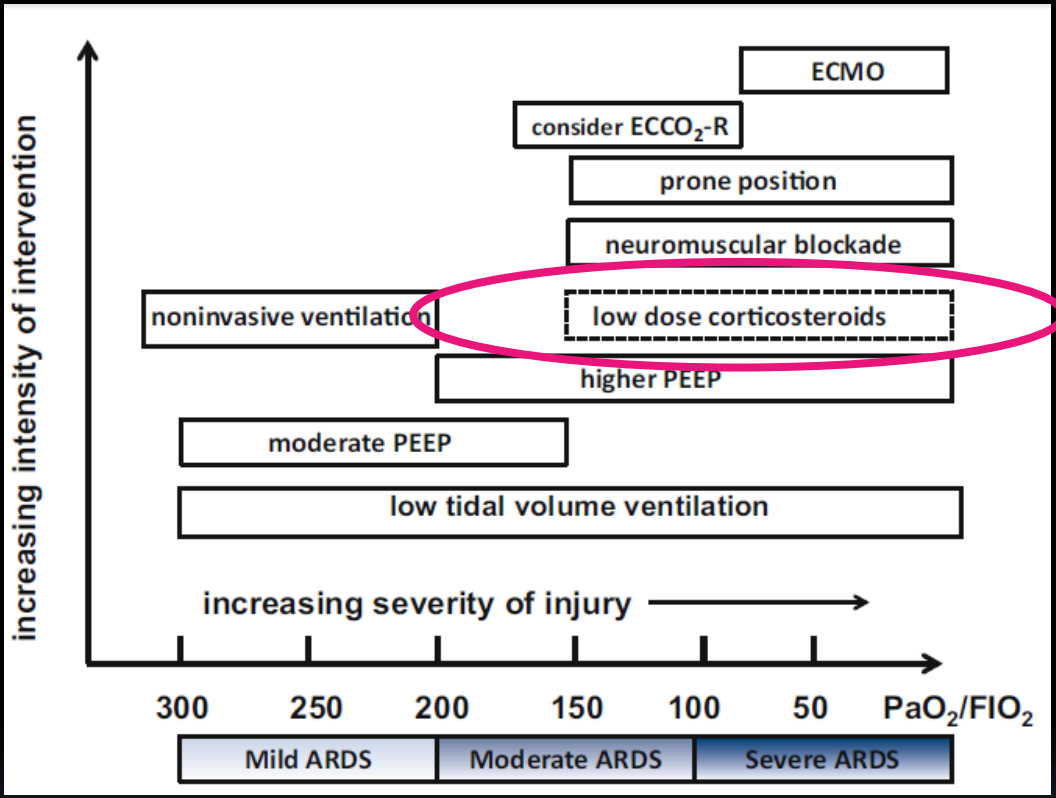
1. Minimise atelectrauma *(under-recruitment injury)*
- open up alveoli with recruitment manoeuvre
- keep alveoli open (prevent de-recruitment) by applying optimal PEEP

2. Minimise volutrauma *(over-distension injury)*
- keep plateau pressure < 30cm H_2O
- use low tidal volume ventilation 6ml/kg IBW

3. Minimise O_2 toxicity
- maintain FiO_2 below 0.6

4. Accept physiologic target outside normal range
- permissive hypercapnia
- permissive hypoxemia

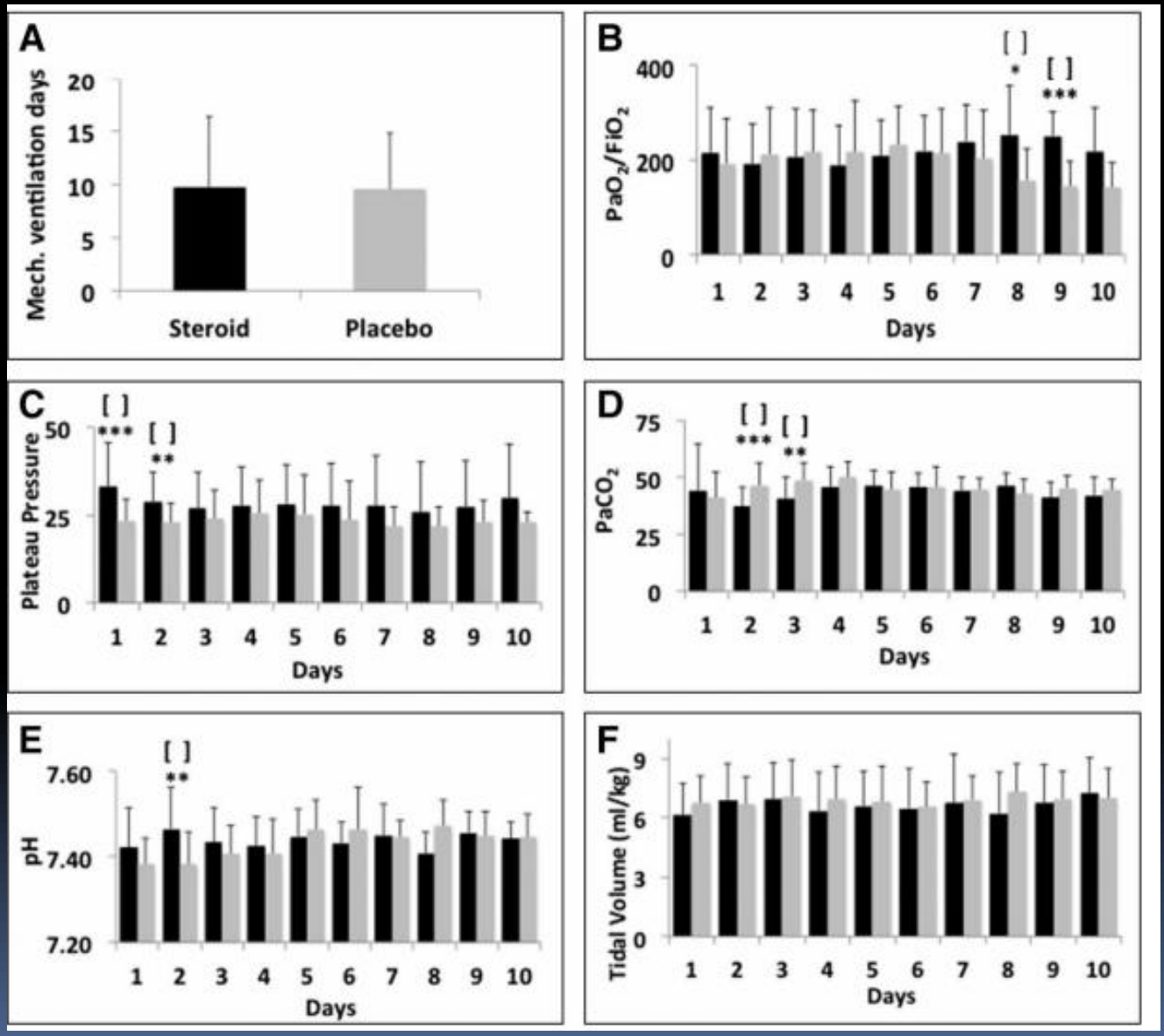
Steroids are part of rescue therapy in ARDS patients with refractory hypoxemia: yes



Such a management should include lung protective ventilation, adequate PEEP, prone positioning, early and low-dose glucocorticoids (methylprednisolone initially 1 mg/kg/day, then dose tapering), and a short course of a short-acting neuromuscular blocker. Steroids are part of rescue therapy in ARDS patients with refractory hypoxemia: yes!

Springer-Verlag Berlin Heidelberg and ESICM 2016

Double-Blind, Placebo-Controlled Pilot Randomized Trial of Methylprednisolone Infusion in Pediatric Acute Respiratory Distress Syndrome



Double-Blind, Placebo-Controlled Pilot Randomized Trial of Methylprednisolone Infusion in Pediatric Acute Respiratory Distress Syndrome

TABLE 3. Proposed Power Analyses Based on Duration of Mechanical Ventilation as the Primary Outcome

DMV in Placebo Group	DMV in Steroid Group	Percent Reduction in DMV (%)	α-Error	Power (1 – β Error) (%)	No. of Subjects (*N*1)	No. of Subjects (*N*2)
12.5±9.6	10.6±8.5	15	0.01	80.0	547	547
12.5±9.6	9.0±6.9	28	0.01	80.0	137	137
12.5±9.6	8.0±8.5	36	0.01	80.3	98	98

DMV = duration of mechanical ventilation.

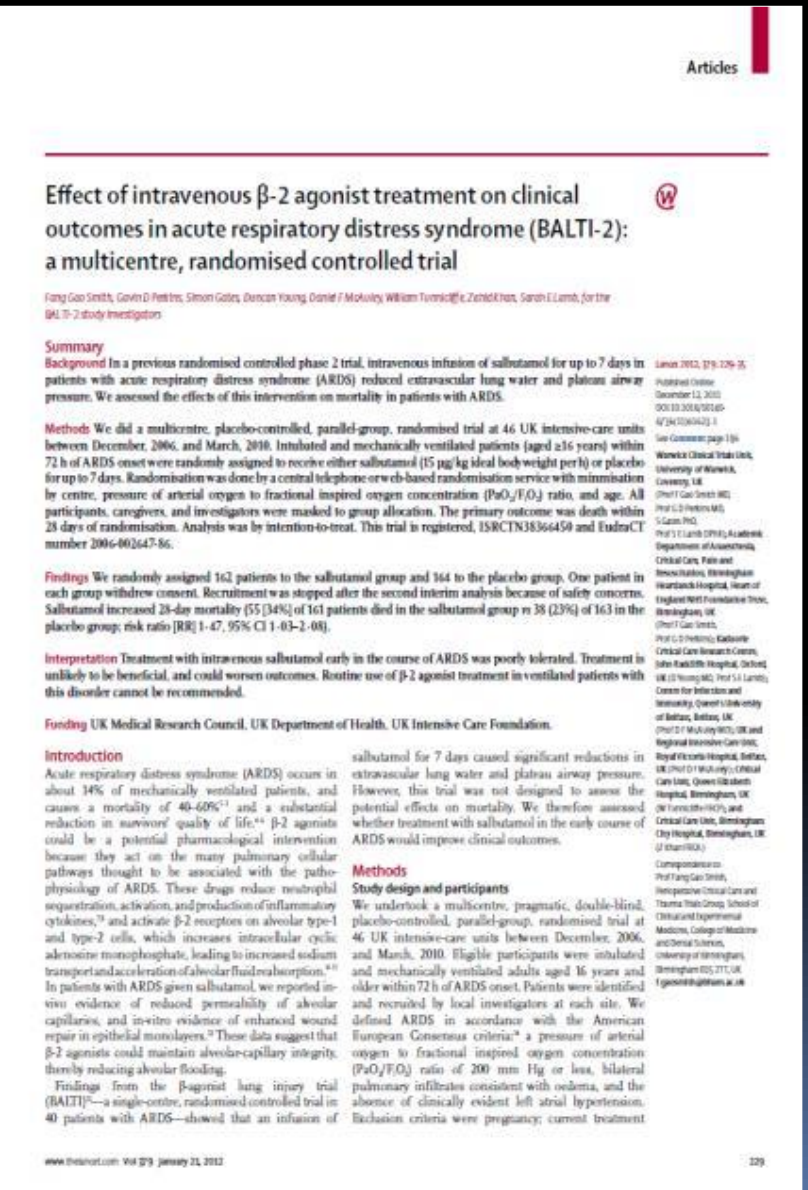
All values = mean ± SD. Power calculations were based on an α-error of $p < 0.01$, a threshold of 80% power (1 – β error), and a two-sided *z* test with adjustment for one interim analysis after 50% enrollment of patients (using an αt^2 α-spending function).

Demuestra la viabilidad de administrar una terapia con glucocorticoides de dosis baja y medir resultados clínicamente relevantes en el síndrome de dificultad respiratoria aguda pediátrica.

Los cambios en la oxigenación y/o ventilación son consistentes con la fisiopatología del síndrome de dificultad respiratoria aguda temprana y los resultados de ensayos clínicos similares en adultos.

March 2015 • Volume 16 • Number 3

BALTI - 2012



Articles

Effect of intravenous β-2 agonist treatment on clinical outcomes in acute respiratory distress syndrome (BALTI-2): a multicentre, randomised controlled trial

Fang Gao Smith, Gavin D Perkins, Simon Gates, Duncan Young, Daniel F McAuley, William Tunnicliffe, Zaheed Khan, Sarah E Lamb, for the BALTI-2 study investigators

Summary

Background In a previous randomised controlled phase 2 trial, intravenous infusion of salbutamol for up to 7 days in patients with acute respiratory distress syndrome (ARDS) reduced extravascular lung water and plateau airway pressure. We assessed the effects of this intervention on mortality in patients with ARDS.

Methods We did a multicentre, placebo-controlled, parallel-group, randomised trial at 46 UK intensive-care units between December, 2006, and March, 2010. Intubated and mechanically ventilated patients (aged ≥16 years) within 72 h of ARDS onset were randomly assigned to receive either salbutamol (15 μg/kg ideal bodyweight per h) or placebo for up to 7 days. Randomisation was done by a central telephone or web-based randomisation service with minimisation by centre, pressure of arterial oxygen to fractional inspired oxygen concentration (PaO_2/F_iO_2) ratio, and age. All participants, caregivers, and investigators were masked to group allocation. The primary outcome was death within 28 days of randomisation. Analysis was by intention-to-treat. This trial is registered, ISRCTN38366450 and EudraCT number 2006-002647-86.

Findings We randomly assigned 162 patients to the salbutamol group and 164 to the placebo group. One patient in each group withdrew consent. Recruitment was stopped after the second interim analysis because of safety concerns. Salbutamol increased 28-day mortality (55 [34%] of 161 patients died in the salbutamol group *vs* 38 (23%) of 163 in the placebo group; risk ratio [RR] 1·47, 95% CI 1·03–2·08).

Interpretation Treatment with intravenous salbutamol early in the course of ARDS was poorly tolerated. Treatment is unlikely to be beneficial, and could worsen outcomes. Routine use of β-2 agonist treatment in ventilated patients with this disorder cannot be recommended.

Funding UK Medical Research Council, UK Department of Health, UK Intensive Care Foundation.

Lancet 2012; 379: 229–35

Introduction

Acute respiratory distress syndrome (ARDS) occurs in about 14% of mechanically ventilated patients, and causes a mortality of 40–60%[1–3] and a substantial reduction in survivors' quality of life.[4,5] β-2 agonists could be a potential pharmacological intervention because they act on the many pulmonary cellular pathways thought to be associated with the pathophysiology of ARDS. These drugs reduce neutrophil sequestration, activation, and production of inflammatory cytokines,[6] and activate β-2 receptors on alveolar type-1 and type-2 cells, which increases intracellular cyclic adenosine monophosphate, leading to increased sodium transport and acceleration of alveolar fluid reabsorption.[6,7] In patients with ARDS given salbutamol, we reported in-vivo evidence of reduced permeability of alveolar capillaries, and in-vitro evidence of enhanced wound repair in epithelial monolayers.[8] These data suggest that β-2 agonists could maintain alveolar-capillary integrity, thereby reducing alveolar flooding.

Findings from the β-agonist lung injury trial (BALTI)[9]—a single-centre, randomised controlled trial in 40 patients with ARDS—showed that an infusion of salbutamol for 7 days caused significant reductions in extravascular lung water and plateau airway pressure. However, this trial was not designed to assess the potential effects on mortality. We therefore assessed whether treatment with salbutamol in the early course of ARDS would improve clinical outcomes.

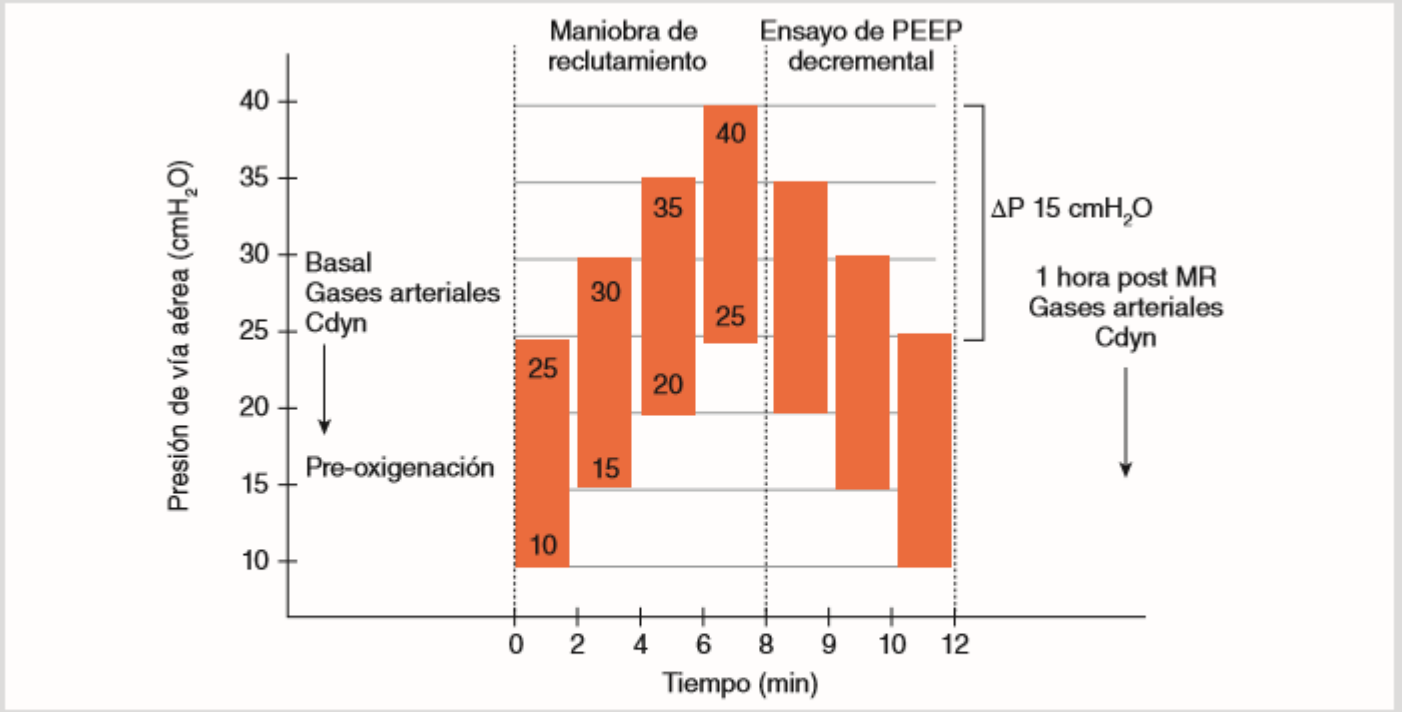
Methods

Study design and participants

We undertook a multicentre, pragmatic, double-blind, placebo-controlled, parallel-group, randomised trial at 46 UK intensive-care units between December, 2006, and March, 2010. Eligible participants were intubated and mechanically ventilated adults aged 16 years and older within 72 h of ARDS onset. Patients were identified and recruited by local investigators at each site. We defined ARDS in accordance with the American European Consensus criteria:[10] a pressure of arterial oxygen to fractional inspired oxygen concentration (PaO_2/F_iO_2) ratio of 200 mm Hg or less, bilateral pulmonary infiltrates consistent with oedema, and the absence of clinically evident left atrial hypertension. Exclusion criteria were pregnancy; current treatment

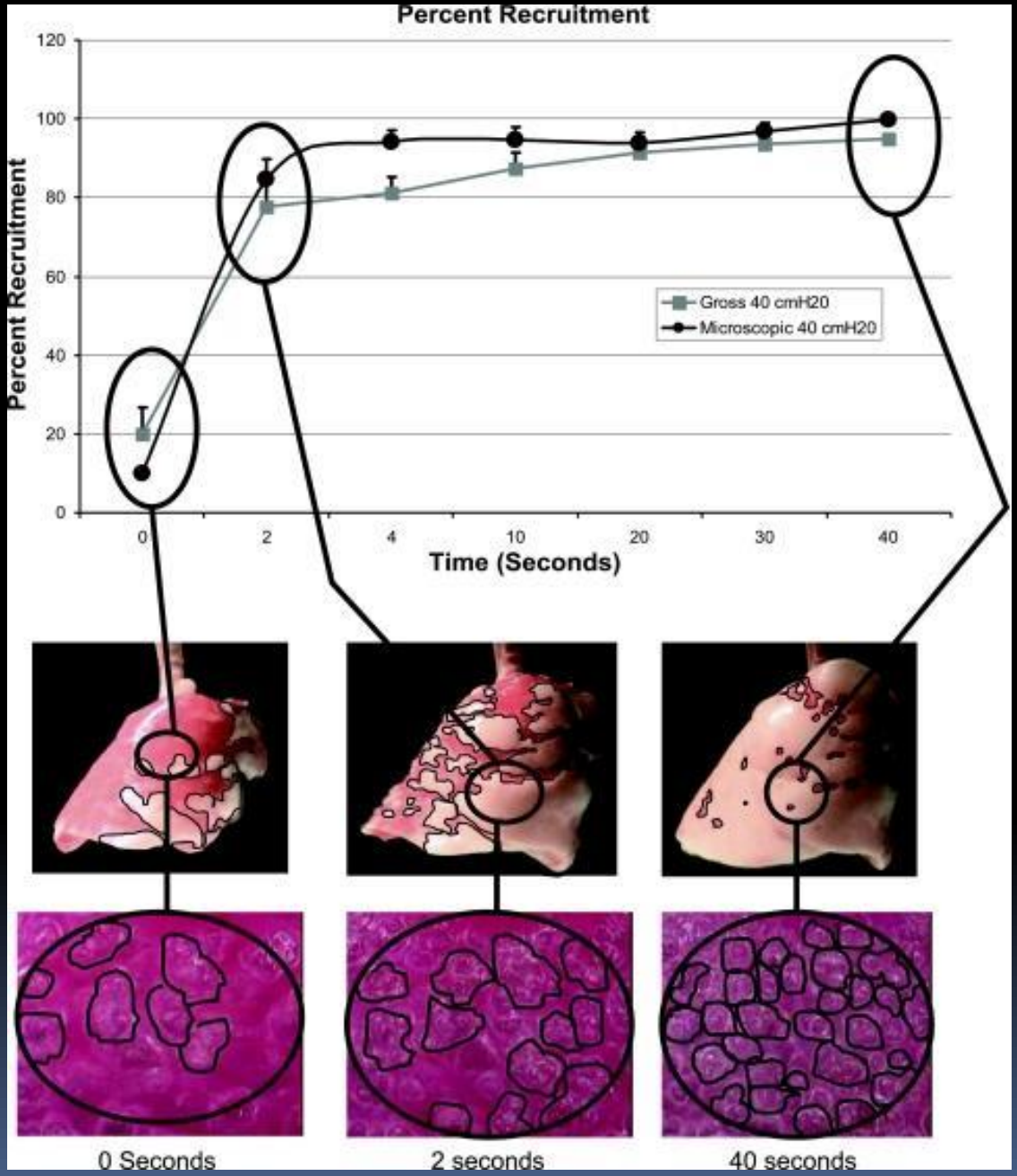
- 162 patients; 46 UK ICU's
- ARDS & MV
  - salbutamol 15mcg/kg/hr or placebo
  - Treatment for up to 7 d
- Mortalidad mayor en los que recibieron salbutamol 34% vs 23% a 28d

TITULACION DE LA PRESION POSITIVA AL FINAL DE LA ESPIRACION



. *Protocolo de reclutamiento alveolar y ensayo decreciente de la PEEP. Esta estrategia es realizada bajo modalidad presión-control. Se inicia con PEEP de 10 cmH_2O manteniendo una presión de distensión fija en 15 cmH_2O. La maniobra de reclutamiento se realiza de forma secuencial incrementando la PEEP 5 cmH_2O cada 2 min hasta alcanzar una PEEP de 25 cmH_2O. La titulación de la PEEP se basa en gasometría y mecánica pulmonar* (modificado de Cruces, et al.[33]). *MR: maniobra de reclutamiento.*

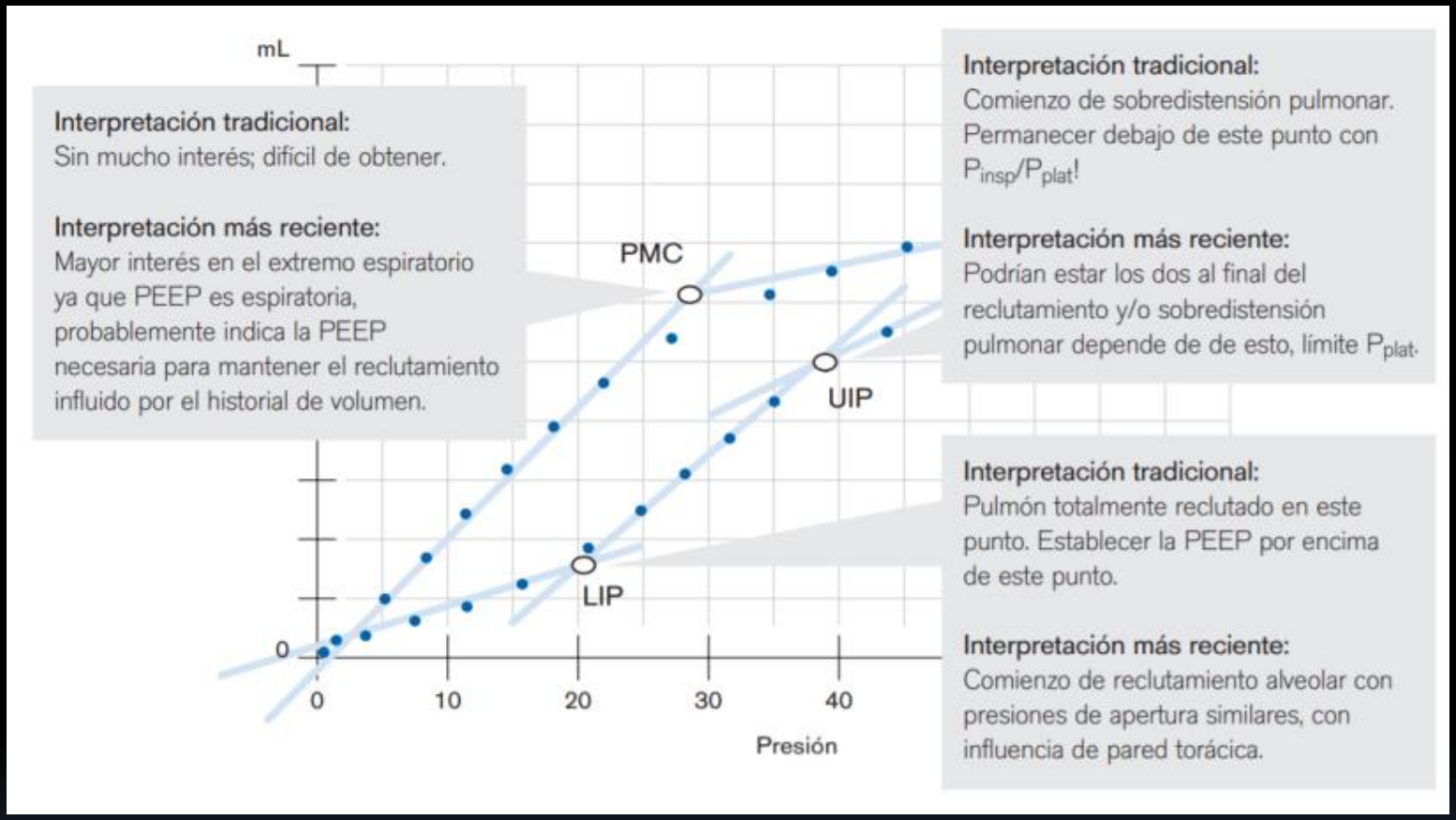
Gaceta Médica de México. 2015;151



J Appl Physiol (1985). 2009 Mar; 106(3): 757–765

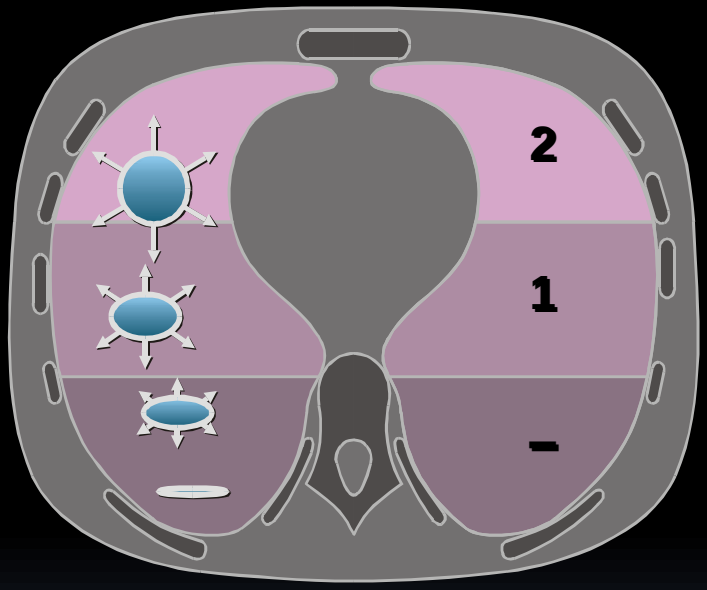
- Aumento del PEEP de 5 cm H2O c/10 resp. Hasta alcanzar 20 cmH2O y aumento de vt hasta 18 ml/kg o Pr Pico de 40 cm H2O.
- Presiones de 35 a 40 cm H2O por 20 seg. Insuflando vt de hasta 12 ml/kg.
- Aumento gradual de la PIP y PEEP hasta un nivel de 40 cm H2O respectivamente durante 10 resp.
- Posicion prono.
- PEEP ideal (2-3 cm H20 por encima del PII o PMC) y Pausa Insp. Continua de hasta 0.5 seg c/ciclo.

Puntos característicos en un bucle PV y su interpretación

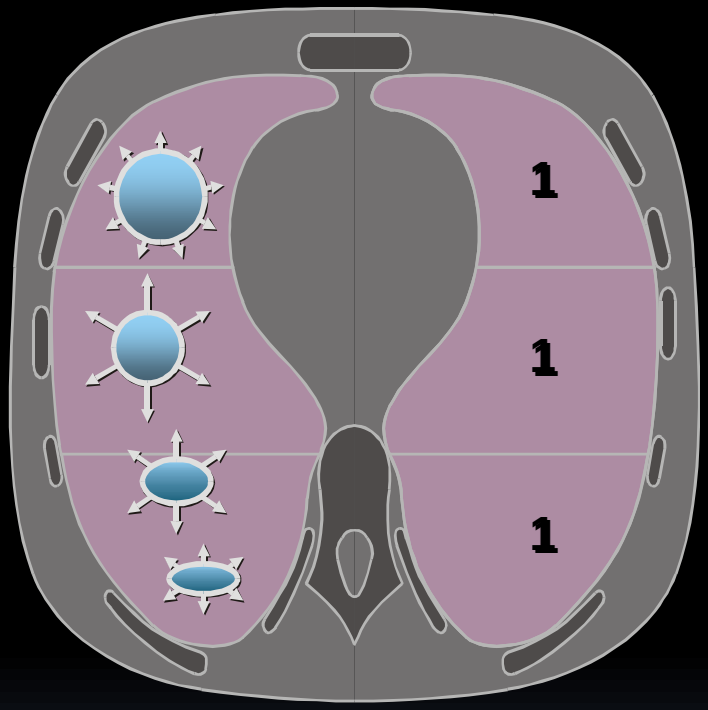


Más recientemente, el interés se ha dirigido hacia el punto de máxima curvatura (PMC) en el extremo espiratorio del bucle de volumen de presión. Puesto que la PEEP se usa para evitar la pérdida de volumen espiratorio final durante la exhalación, tendría sentido identificar una pérdida de volumen si el pulmón se desinfla lentamente.

Sobreinflación prevalente = efecto de espacio muerto

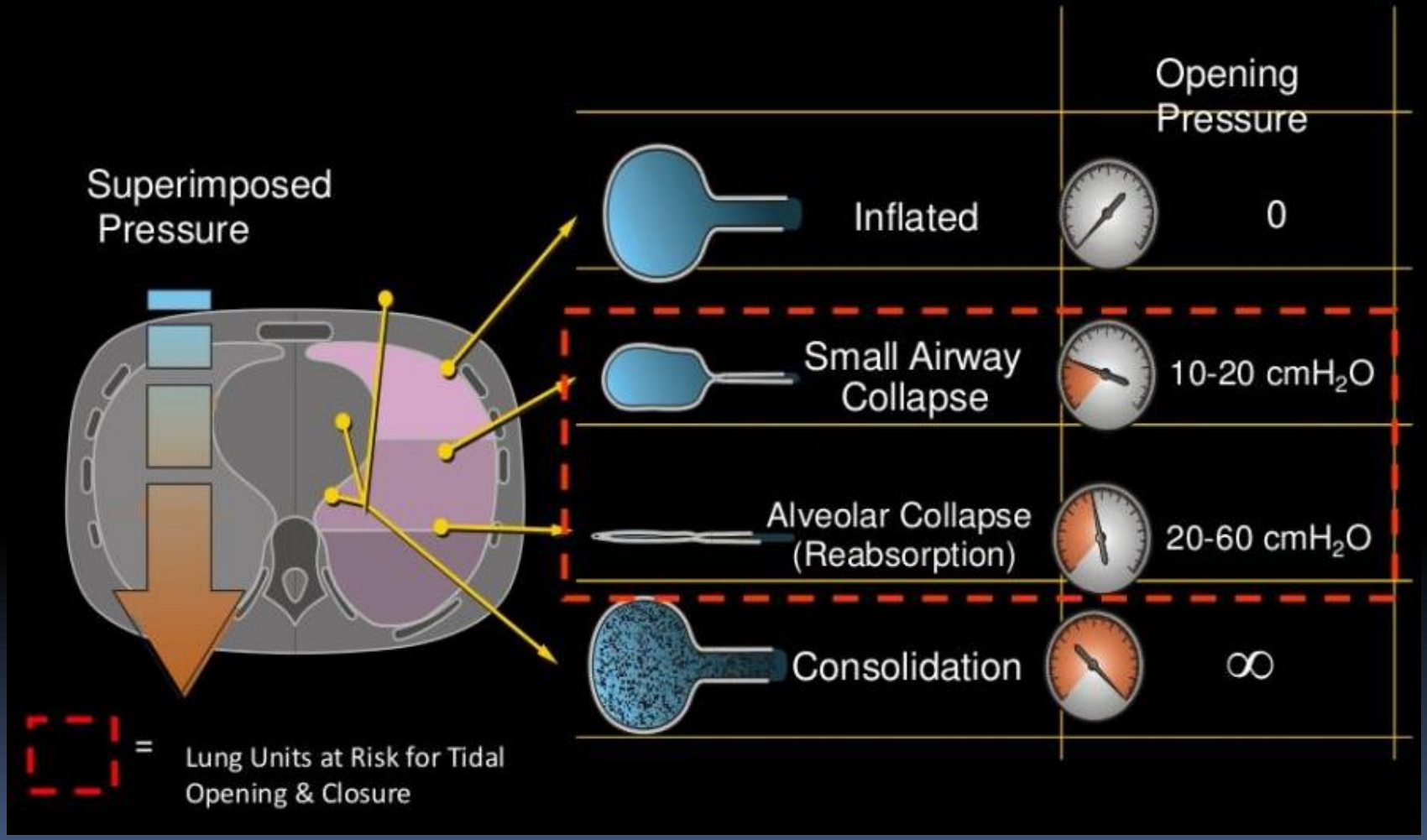


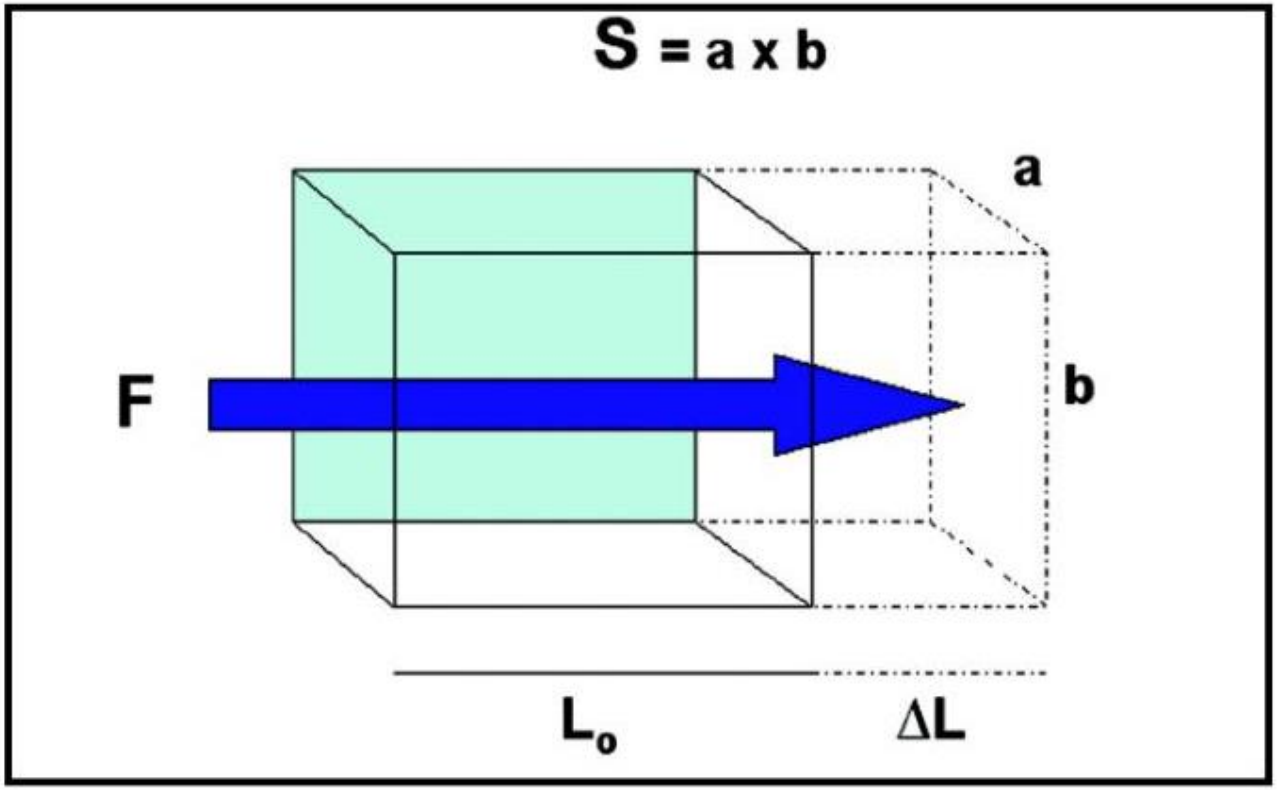
PEEP 5



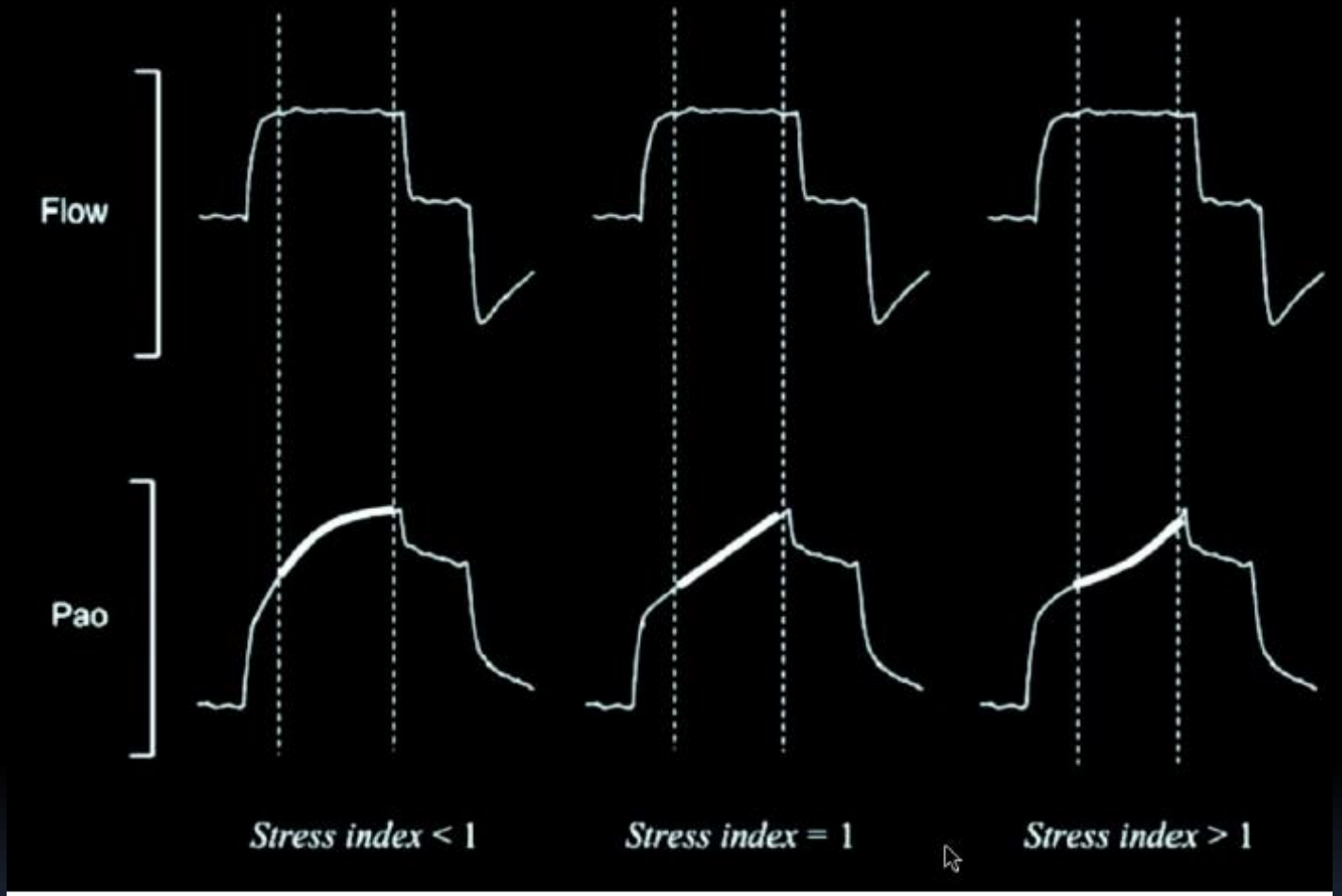
PEEP 20

Spectrum of Regional Opening Pressures (Supine Position)



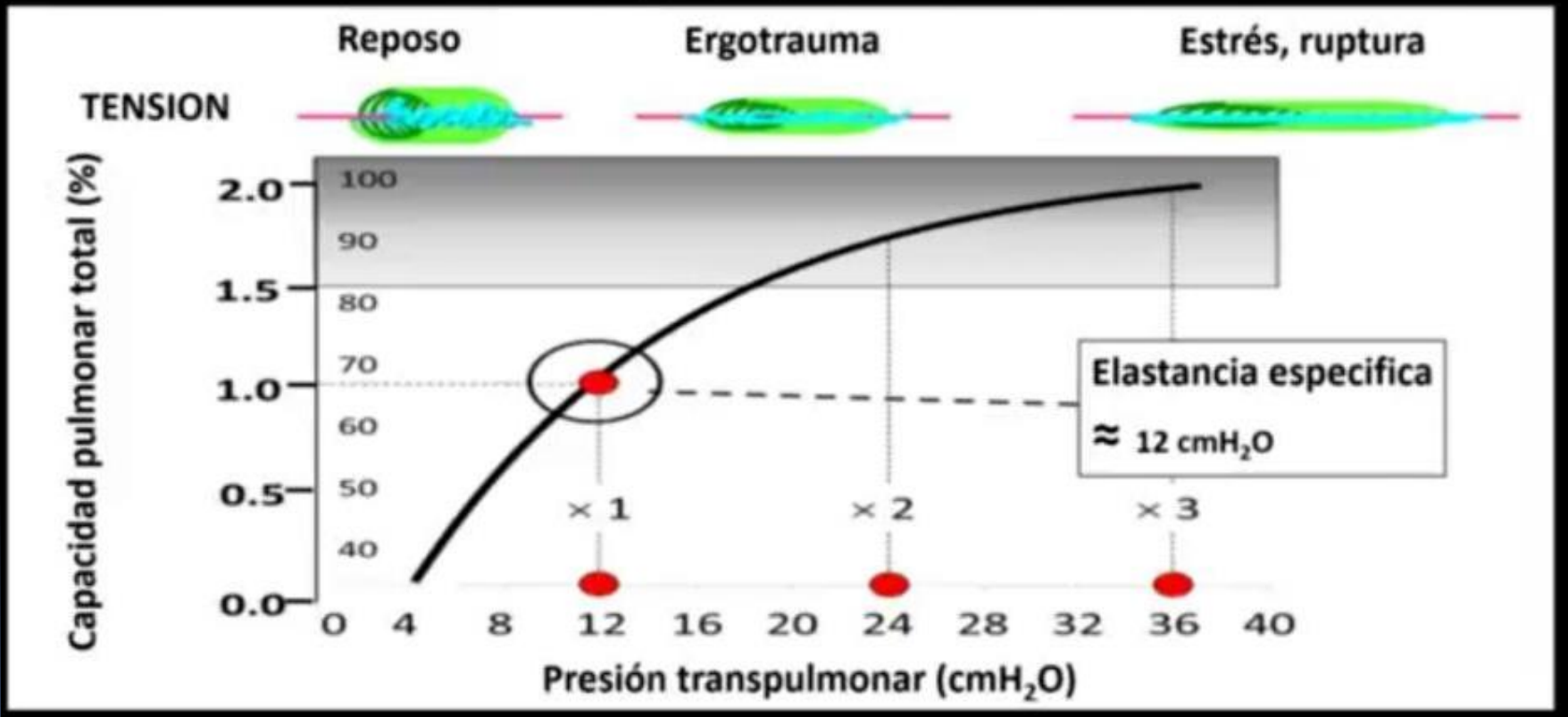


Firuga 2. La figura representa esquemáticamente un alveolo y según Gattinoni *stress* corresponde a la presión de distensión o tensión aplicada sobre una superficie (_ = F/S) y *strain* a la deformación (_ = $\Delta L/L_o$) generada por esta fuerza. F =tensión, S =superficie, Lo =posición de reposo, ΔL =elongación de las fibras desde su posición de reposo.



Firuga 4. Morfología de la curva presión/tiempo y su relación con el *stress index*. En la figura un *stress index* <1 implica reclutamiento y puede aumentar el PEEP y un *stress index* >1 evidencia sobredistensión de alvéolos y debe disminuir el PEEP.

Energía aplicada



Critical Care 2017;21(Suppl 3):312

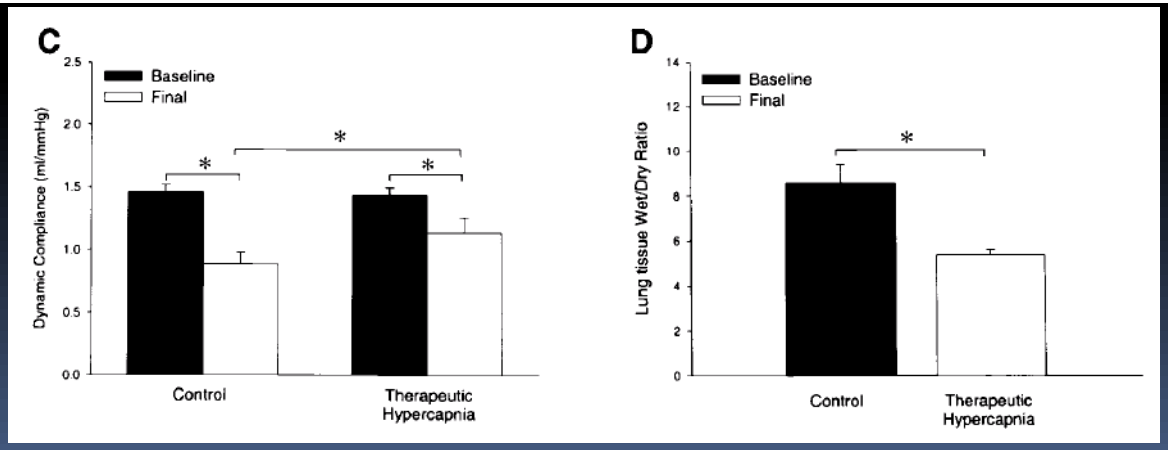
Hipercapnia permisiva:
¿Un objetivo o una consecuencia indeseable?

¿Protección por estrés pulmonar reducido o hipercapnia terapéutica?

Therapeutic Hypercapnia Reduces Pulmonary and Systemic Injury following *In Vivo* Lung Reperfusion

JOHN G. LAFFEY, MOTOSHI TANAKA, DOREEN ENGELBERTS, XIAOPING LUO, SHIZENG YUAN, A. KEITH TANSWELL, MARTIN POST, THOMAS LINDSAY, and BRIAN P. KAVANAGH

The Lung Biology Programme and the Departments of Critical Care Medicine and Paediatrics, The Research Institute, The Hospital for Sick Children; and The University of Toronto, Toronto, Ontario, Canada



Laffey JG Am J Respir Crit Care Med 2000; 162: 2287–2294

HYPERCAPNIA in pediatric ARDS

La hipoventilación con hipercapnia moderada parece segura.

Sin embargo, ciertas categorías de pacientes están en riesgo, incluidos aquellos con traumatismo craneoencefálico, presión intratorácica alta, inestabilidad hemodinámica, irritabilidad miocárdica y disfunción.

- → **La seguridad de una $PaCO_2$ muy alta no está probada.**
- → **Aún no está claro qué tan bajo puede considerarse seguro el valor del pH arterial.**
- → **El efecto beneficioso de la hipercapnia permisiva sobre el resultado del paciente sigue siendo controvertido.**

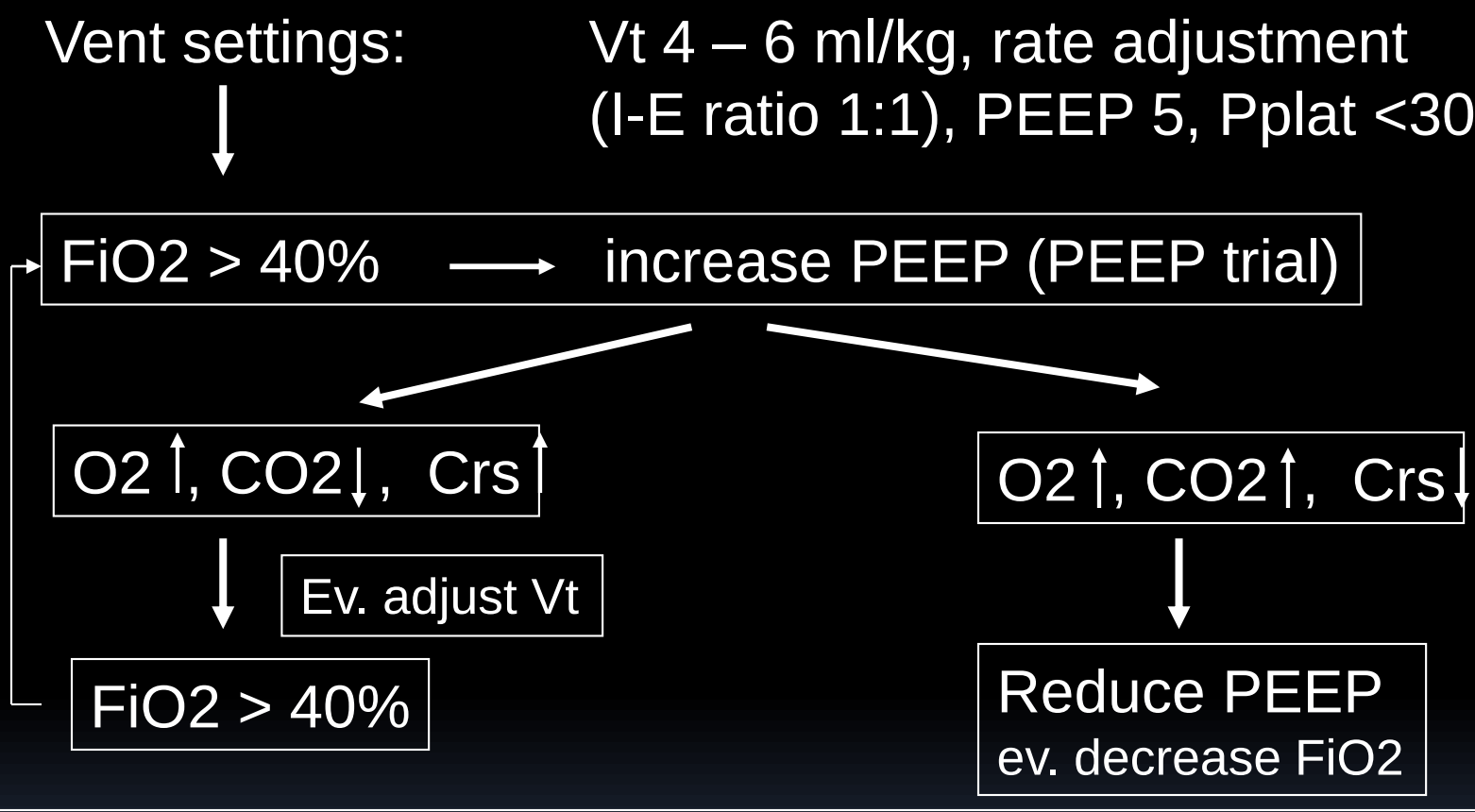
Varios estudios en la literatura han identificado la hiperventilación y la hipocapnia como factores de riesgo para resultados adversos en el SDRA. Una posible explicación podría ser la vasoconstricción y la curva de disociación del oxígeno de la hemoglobina a la izquierda.

Tanto la hiperventilación como la hipocapnia pueden ser causas independientes de displasia broncopulmonar en lactantes.

La hipocapnia reduce el cumplimiento pulmonar, afecta la producción de surfactante funcional y aumenta la permeabilidad microvascular en la mucosa traqueal.

Trimble et al, en 1971 demostraron que la hipocapnia aumentaba el trabajo respiratorio, la falta de ventilación / perfusión severa, aumentaba la resistencia de las vías respiratorias, aumentaba el gradiente de A-aO2 y disminuía la PaO2 en pacientes con SDRA.

Enfoque guiado por algoritmo (oxigenación / ventilación)

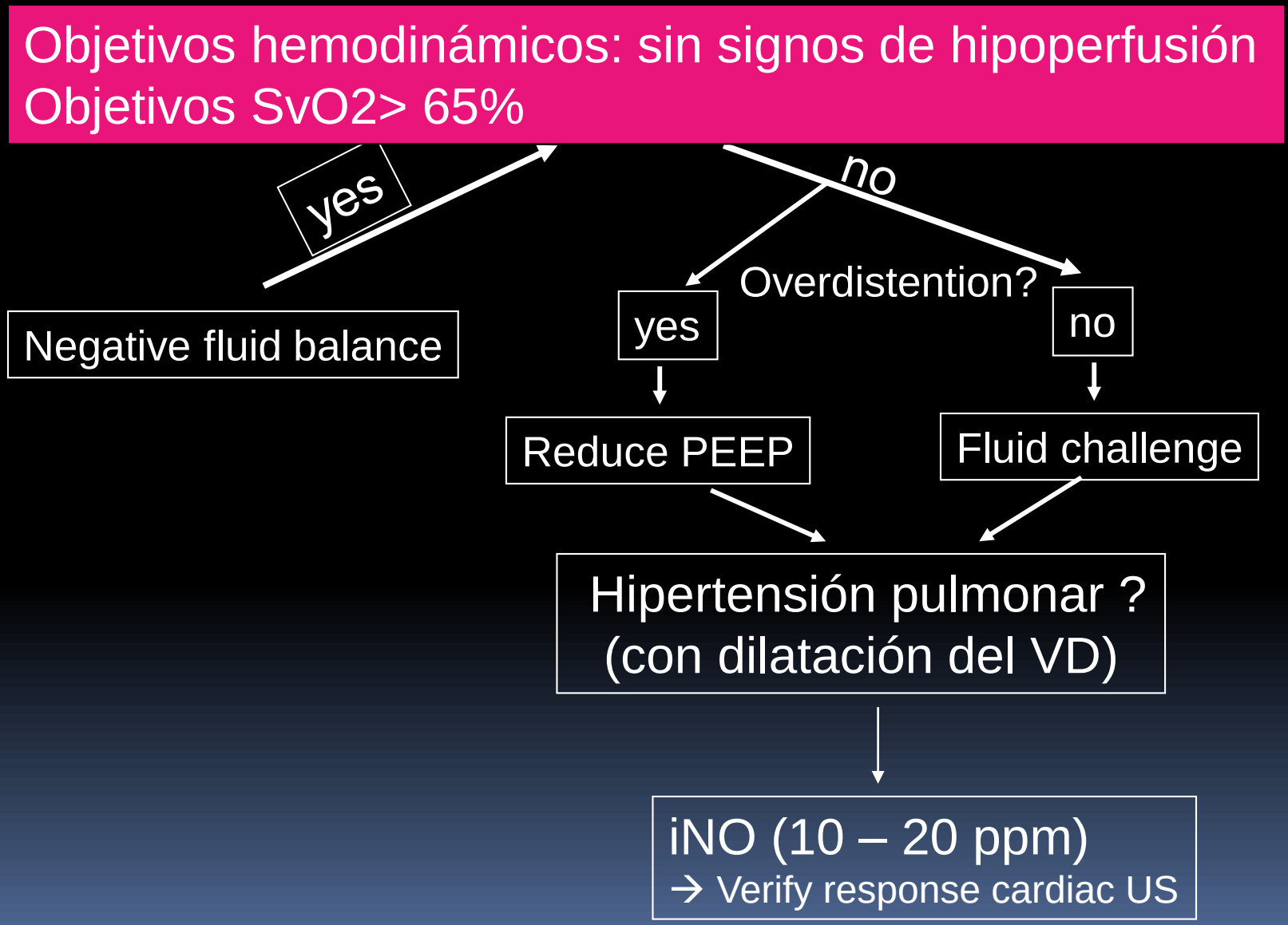


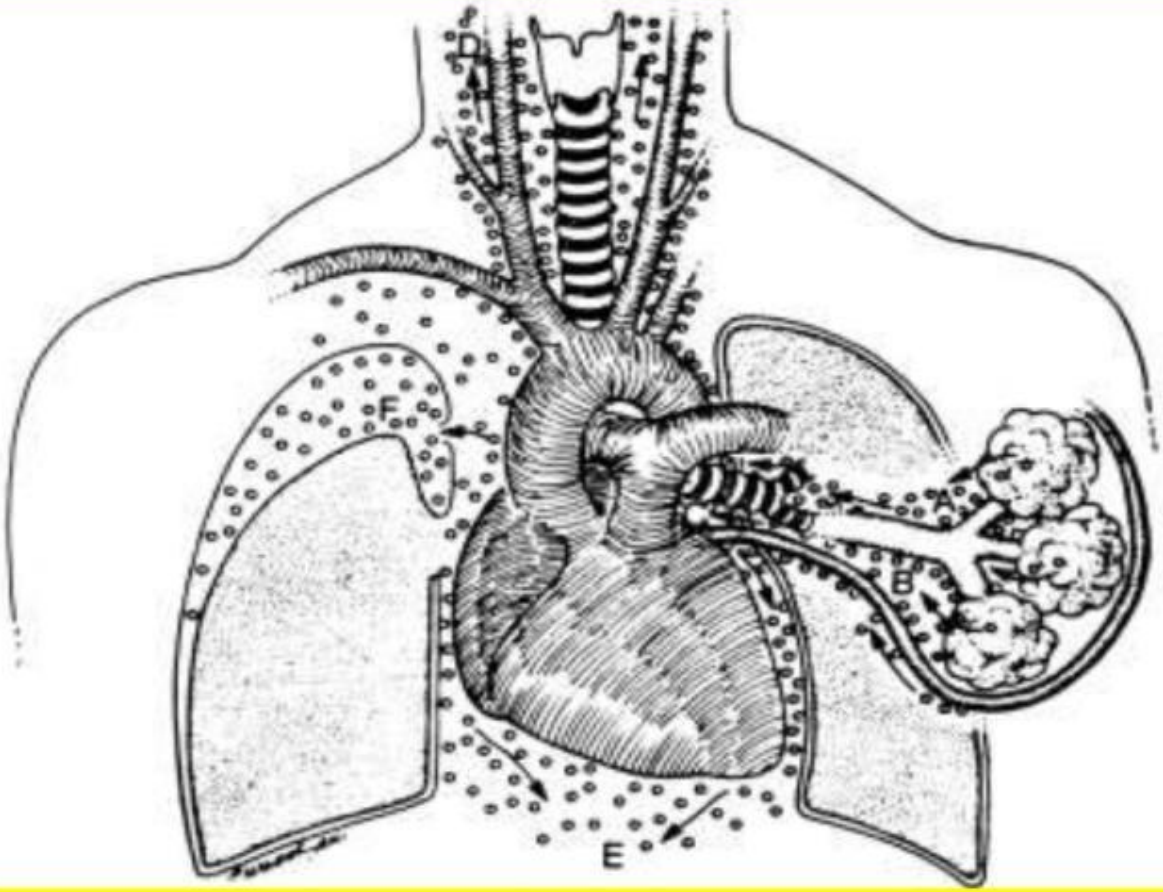
If FiO2 > 60%

(1) try prone position: response if P/F increase > + 40% and / or compliance increase > 25%

(2) try iNO 8 to 12 ppm: stop if no response after 10 minutes response if P/F > + 15%

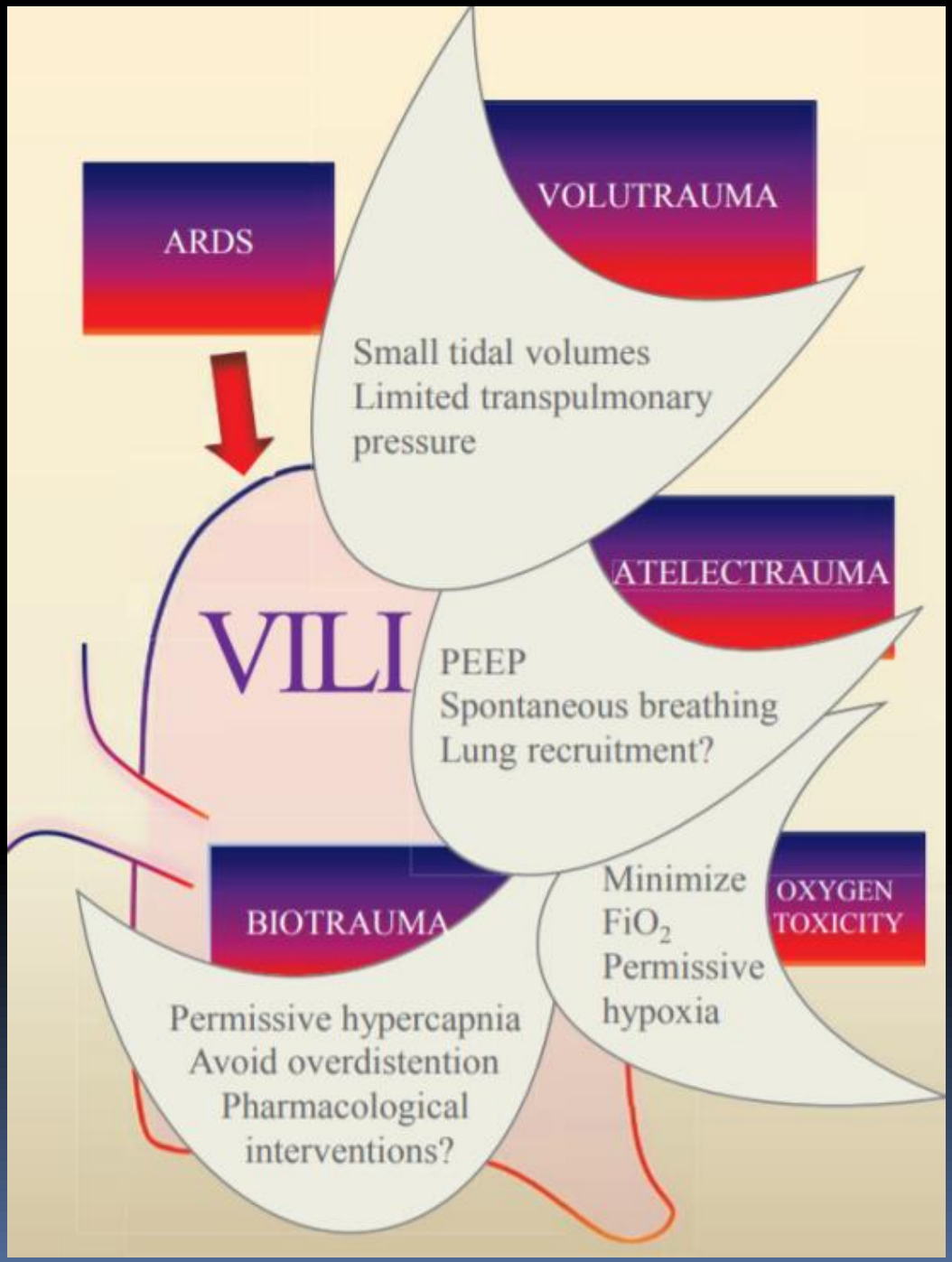
Enfoque guiado por algoritmo (hemodinámica)





Gas Extravasation Barotrauma

Subcutaneous emphyse

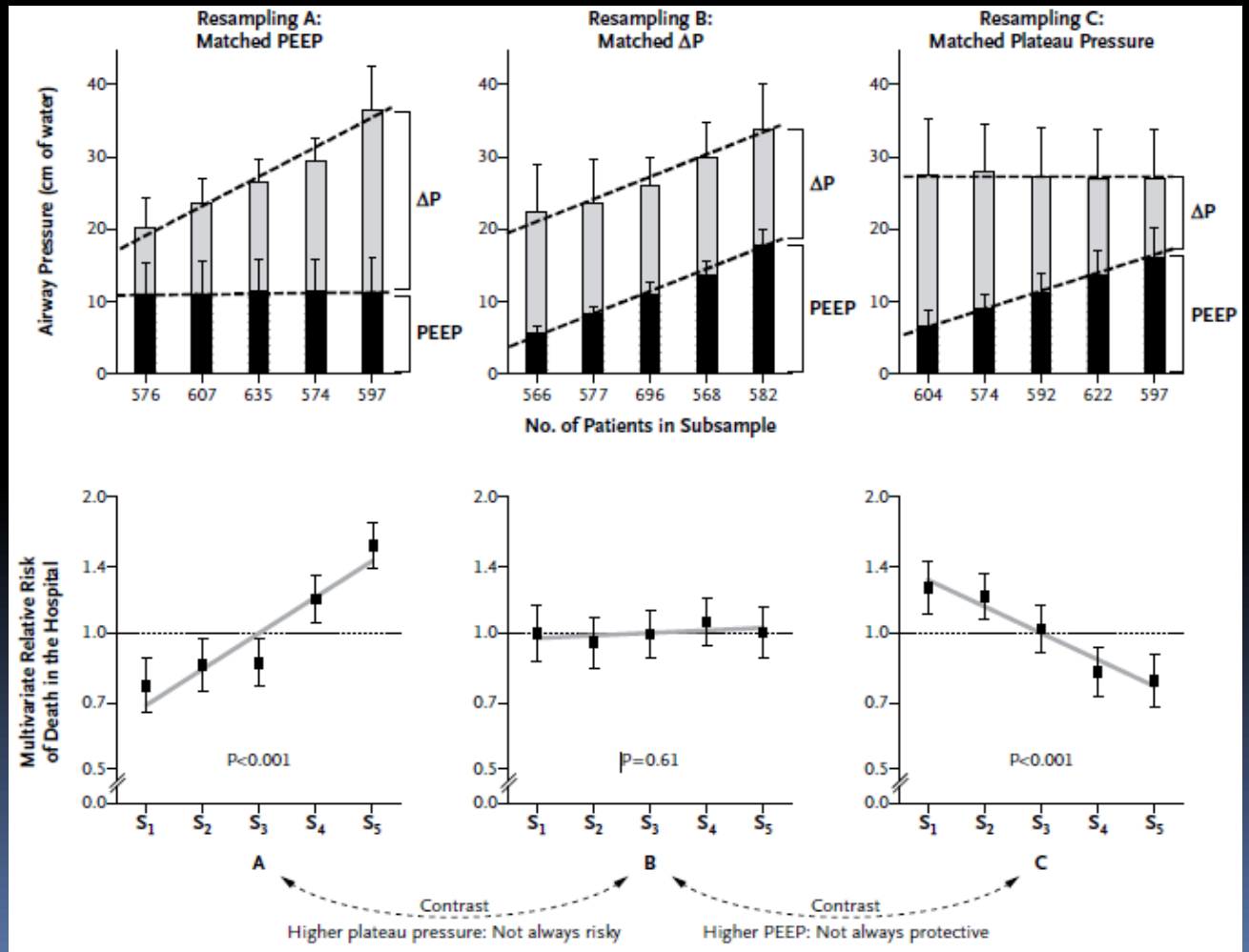

ARDS
VOLUTRAUMA
Small tidal volumes
Limited transpulmonary pressure
VILI
ATELECTRAUMA
PEEP
Spontaneous breathing
Lung recruitment?
BIOTRAUMA
Permissive hypercapnia
Avoid overdistention
Pharmacological interventions?
OXYGEN TOXICITY
Minimize FiO_2
Permissive hypoxia

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.

DRIVING PRESSURE

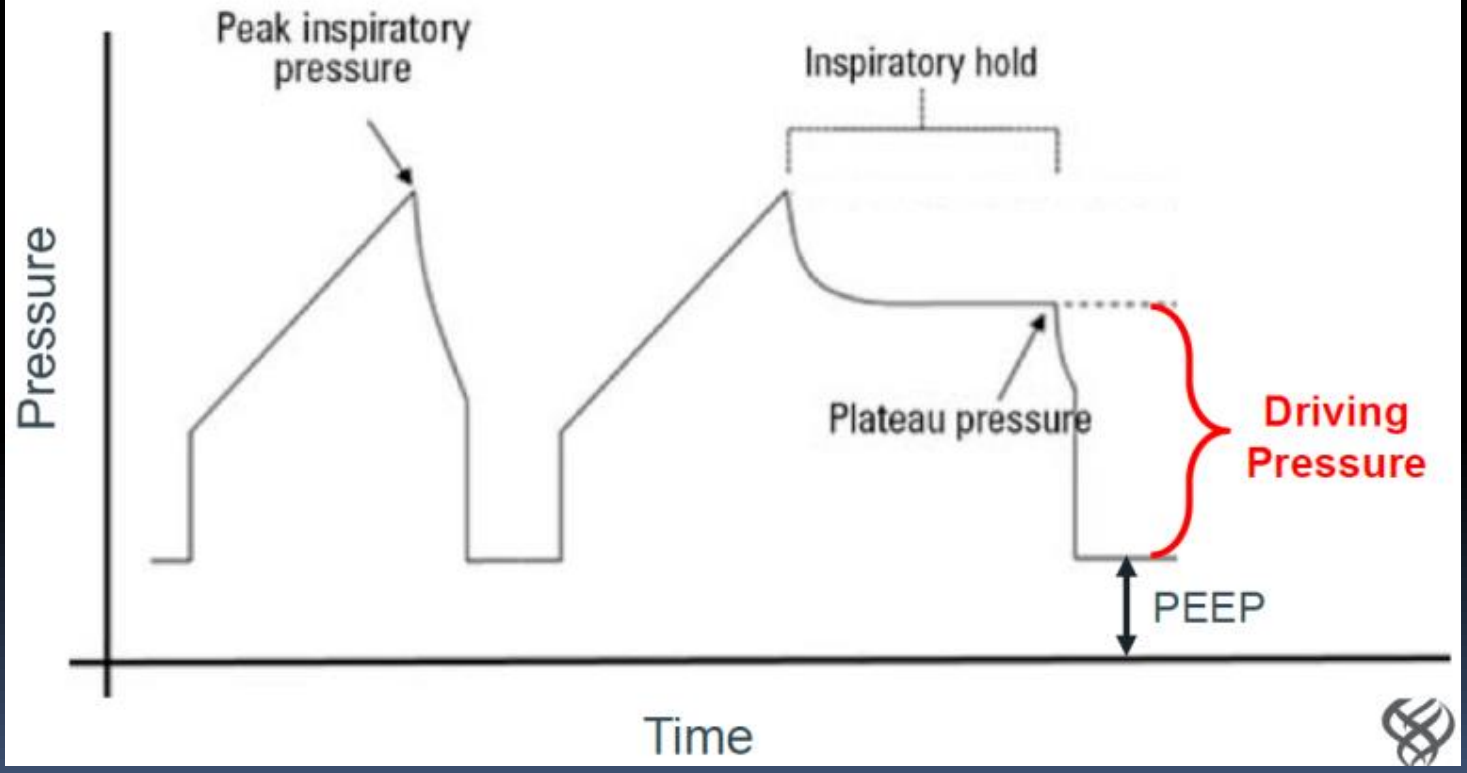


Se encontró que ΔP fue la variable de ventilación que mejor estratificó el riesgo.

Las disminuciones en ΔP debido a cambios en los ajustes del ventilador se asociaron fuertemente con el aumento de la supervivencia.

N engl j med 372;8
February 19, 2015

Driving Pressure (□P)



Driving pressure: a marker of severity, a safety limit, or a goal for mechanical ventilation?

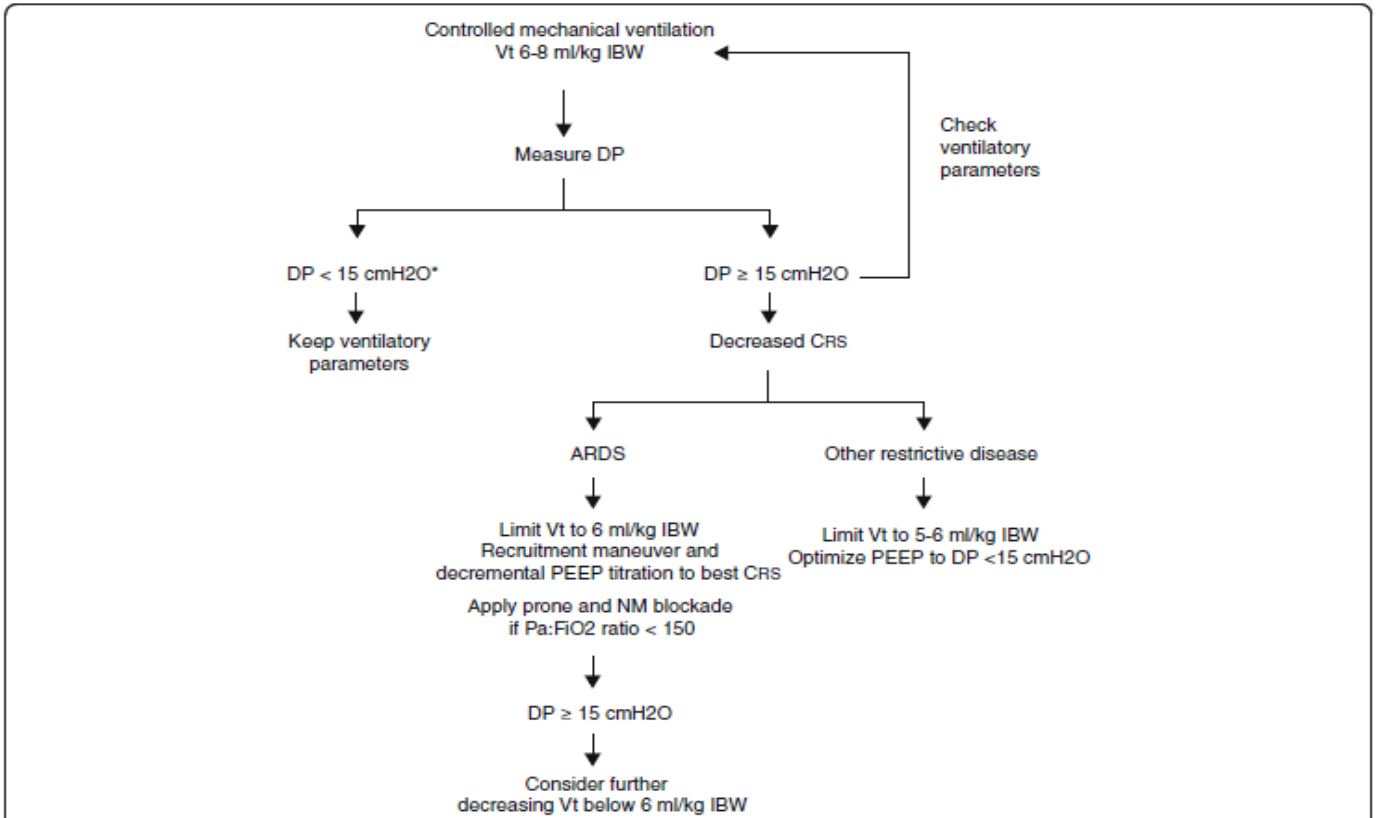


Fig. 3 Suggested flowchart for adjusting ventilatory parameters according to driving pressure in patients requiring invasive mechanical ventilation. *The limit of 15 cm H_2O is only speculative as no safe limit for driving pressure has been identified (see text). *Abbreviations*: *Vt* tidal volume, *IBW* ideal body weight, *DP* airway driving pressure, C_{RS} static compliance of the respiratory system, *NM* neuromuscular, *PaO2:FiO2 ratio* ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen

Bugedo et al. Critical Care (2017) 21:199

Intensive Care Med (2016) 42:1865–1876
DOI 10.1007/s00134-016-4571-5

SEVEN-DAY PROFILE PUBLICATION

Potentially modifiable factors contributing to outcome from acute respiratory distress syndrome: the LUNG SAFE study

John G. Laffey[1,2*], Giacomo Bellani[3,4], Tài Pham[5,6,7], Eddy Fan[8,9], Fabiana Madotto[10], Ednan K. Bajwa[11], Laurent Brochard[12,13], Kevin Clarkson[14], Andres Esteban[15], Luciano Gattinoni[16], Frank van Haren[17], Leo M. Heunks[18], Kiyoyasu Kurahashi[19], Jon Henrik Laake[20], Anders Larsson[21], Daniel F. McAuley[22], Lia McNamee[22], Nicolas Nin[15], Haibo Qiu[23], Marco Ranieri[24], Gordon D. Rubenfeld[25], B. Taylor Thompson[11], Hermann Wrigge[26], Arthur S. Slutsky[12,13,27], Antonio Pesenti[28,29] and The LUNG SAFE Investigators and the ESICM Trials Group

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- ✓ Estudio observacional prospectivo
- ✓ Una cohorte de 459 pacientes.
- ✓ Multicentrico 50 países.
- ✓ Pacientes con SDRA

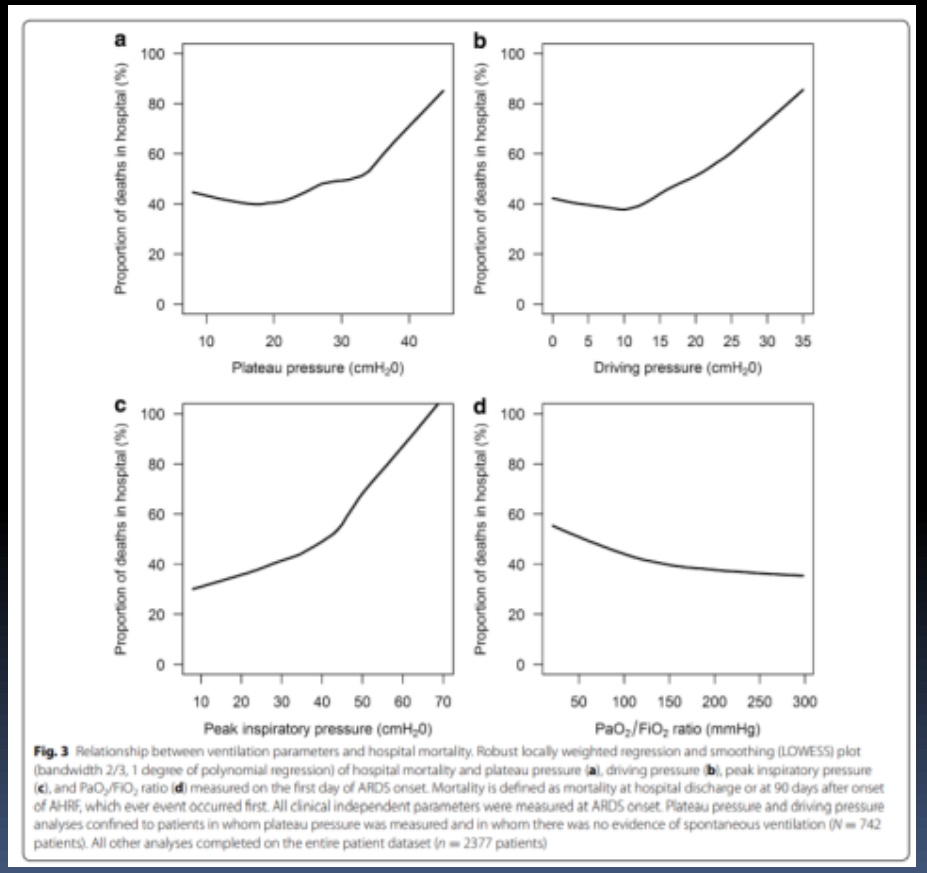

Fig. 3 Relationship between ventilation parameters and hospital mortality. Robust locally weighted regression and smoothing (LOWESS) plot (bandwidth 2/3, 1 degree of polynomial regression) of hospital mortality and plateau pressure (**a**), driving pressure (**b**), peak inspiratory pressure (**c**), and PaO_2/FiO_2 ratio (**d**) measured on the first day of ARDS onset. Mortality is defined as mortality at hospital discharge or at 90 days after onset of AHRF, which ever event occurred first. All clinical independent parameters were measured at ARDS onset. Plateau pressure and driving pressure analyses confined to patients in whom plateau pressure was measured and in whom there was no evidence of spontaneous ventilation ($N = 742$ patients). All other analyses completed on the entire patient dataset ($n = 2377$ patients)

Intensive Care Med (2016) 42:1567–1575
DOI 10.1007/s00134-016-4505-2

ORIGINAL

Ventilator-related causes of lung injury: the mechanical power

L. Gattinoni[1*], T. Tonetti[1], M. Cressoni[2], P. Cadringher[3], P. Herrmann[1], O. Moerer[1], A. Protti[3], M. Gotti[2], C. Chiurazzi[2], E. Carlesso[2], D. Chiumello[4] and M. Quintel[1]

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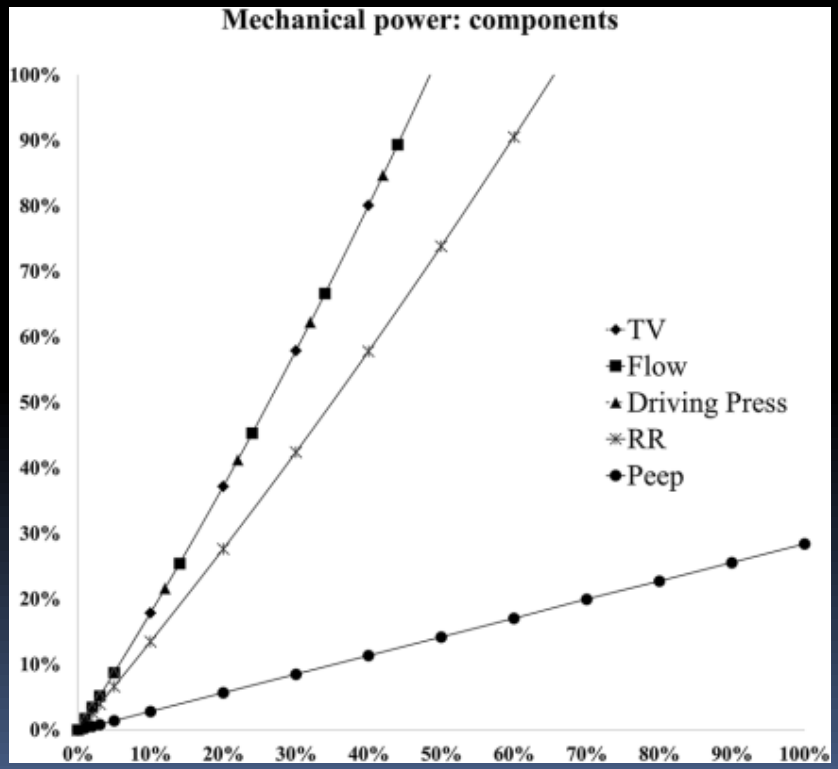


$$\text{Power}_{rs} = \text{RR} \cdot \left\{ \Delta V^2 \cdot \left[\frac{1}{2} \cdot \text{EL}_{rs} + \text{RR} \cdot \frac{(1 + I:E)}{60 \cdot I:E} \cdot R_{aw} \right] + \Delta V \cdot \text{PEEP} \right\}$$

Distender pulmon — Mover el gas — Mantenerlo abierto

$$PM = 0.098 \times FR \times VC \times \left\{ \text{Presión pico} - \frac{(\text{Meseta} - \text{PEEP})}{2} \right\}$$

Aumento exponencial de mortalidad por aumento en PM por arriba de 17 Joule / min

The percent increase of mechanical power as a function of TV/ΔPaw/inspiratory flow (diamonds, squares and triangles), RR (stars), PEEP (circles). The variations of mechanical power with TV, ΔPaw and inspiratory flow are exactly the same, lying on the same line (for clarity, we show the mechanical power variations at different percent variations of TV, ΔPaw and inspiratory flow, otherwise all points would have been overlapped).

While the mechanical power increases by 37 % with a 20 % increase of TV/ΔPaw/inspiratory flow, it increases by 27 % and by 5.7 % with a 20 % increase of respiratory rate and PEEP, respectively.

All the computations were done by changing one variable at a time, while keeping all the others constant.

DECUBITO PRONO

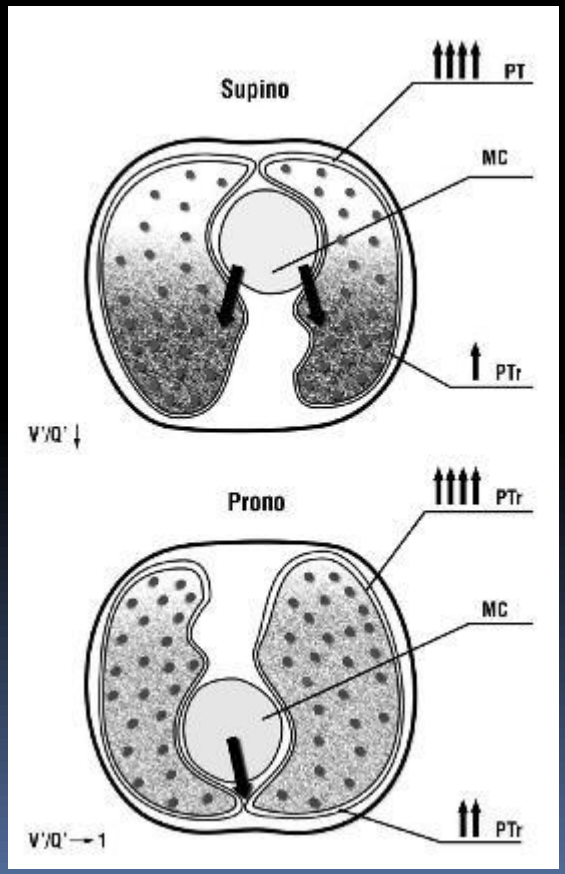
Mejoría en la oxigenación.

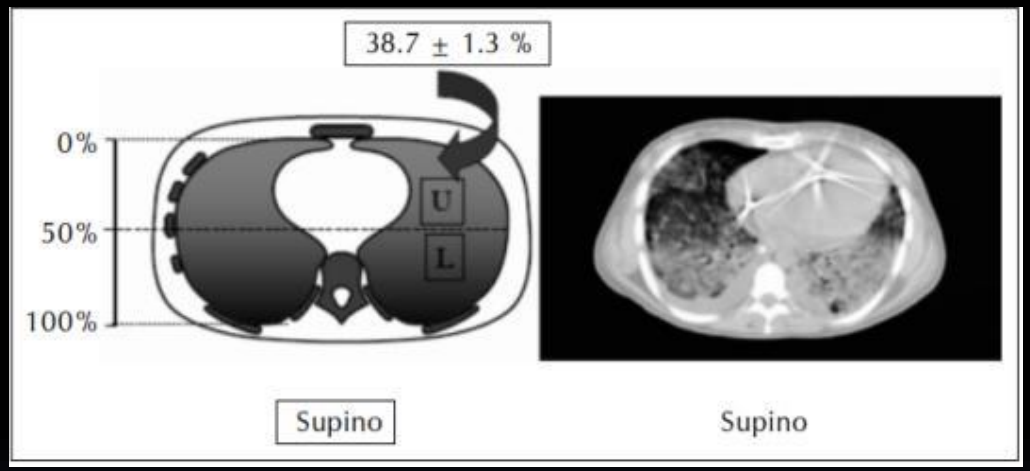
Redistribución de la relación ventilación perfusión.

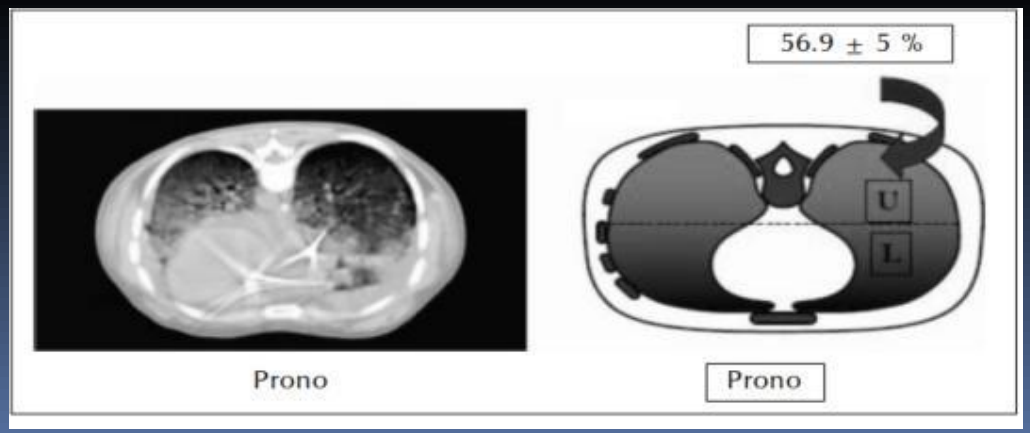
Menor peso de las estructuras mediastínicas.

Cambios en la movilidad regional diafragmática.

Mejor movilización de secreciones.



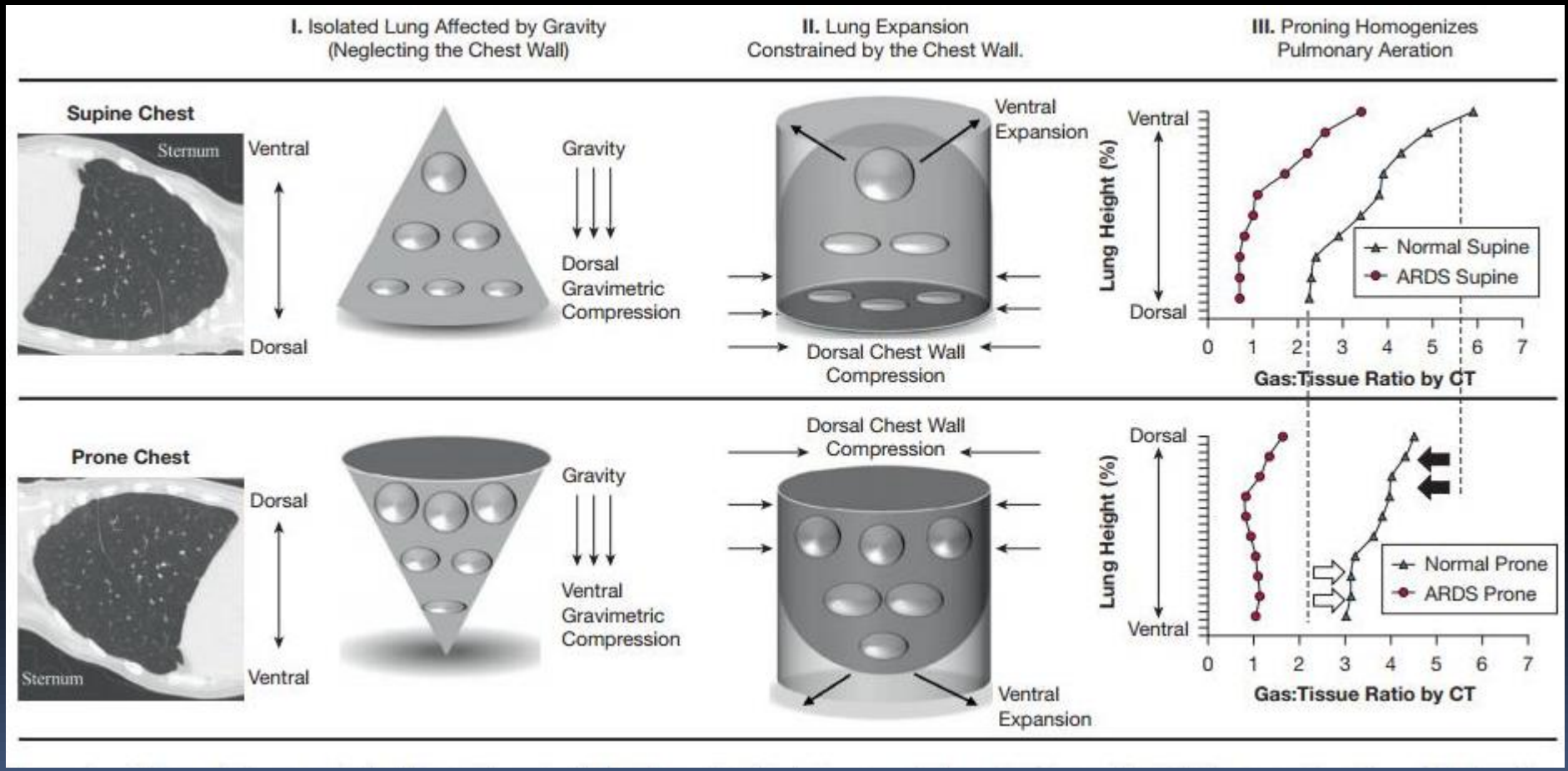




CHEST

Treatment of ARDS With Prone Positioning

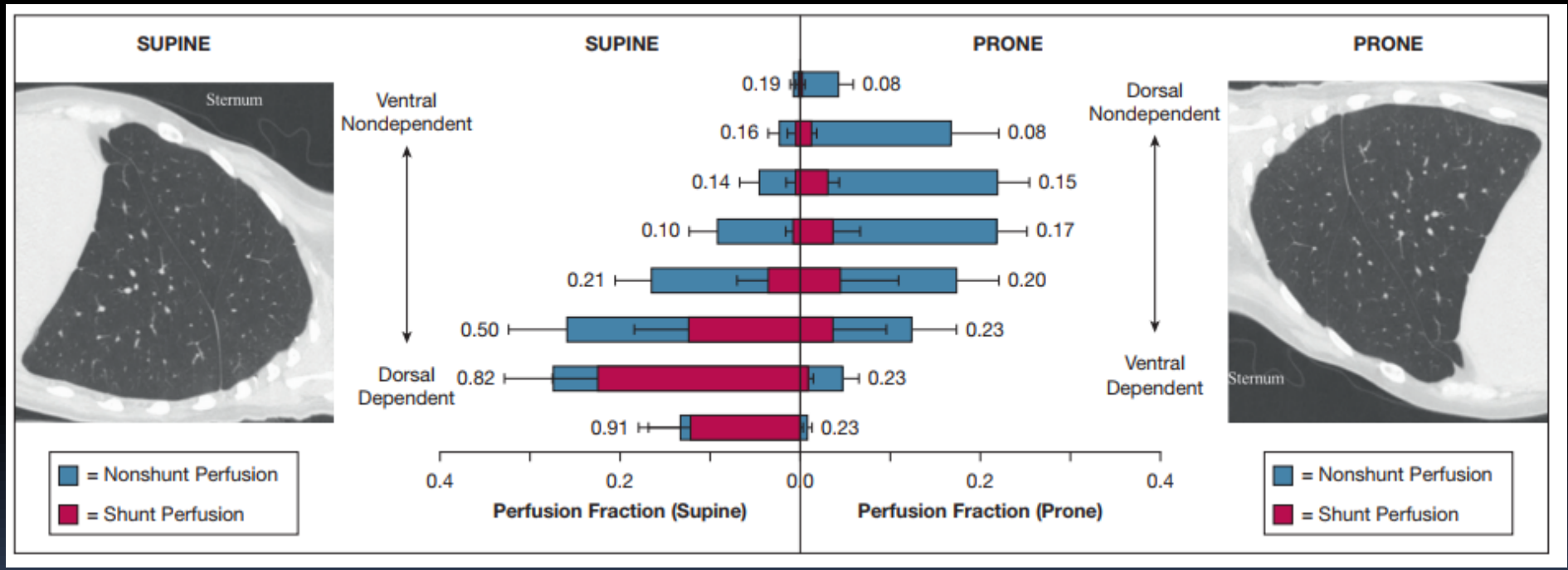
Eric L. Scholten, MD; Jeremy R. Beitler, MD, MPH; G. Kim Prisk, PhD, DSc; and Atul Malhotra, MD



CHEST

Treatment of ARDS With Prone Positioning

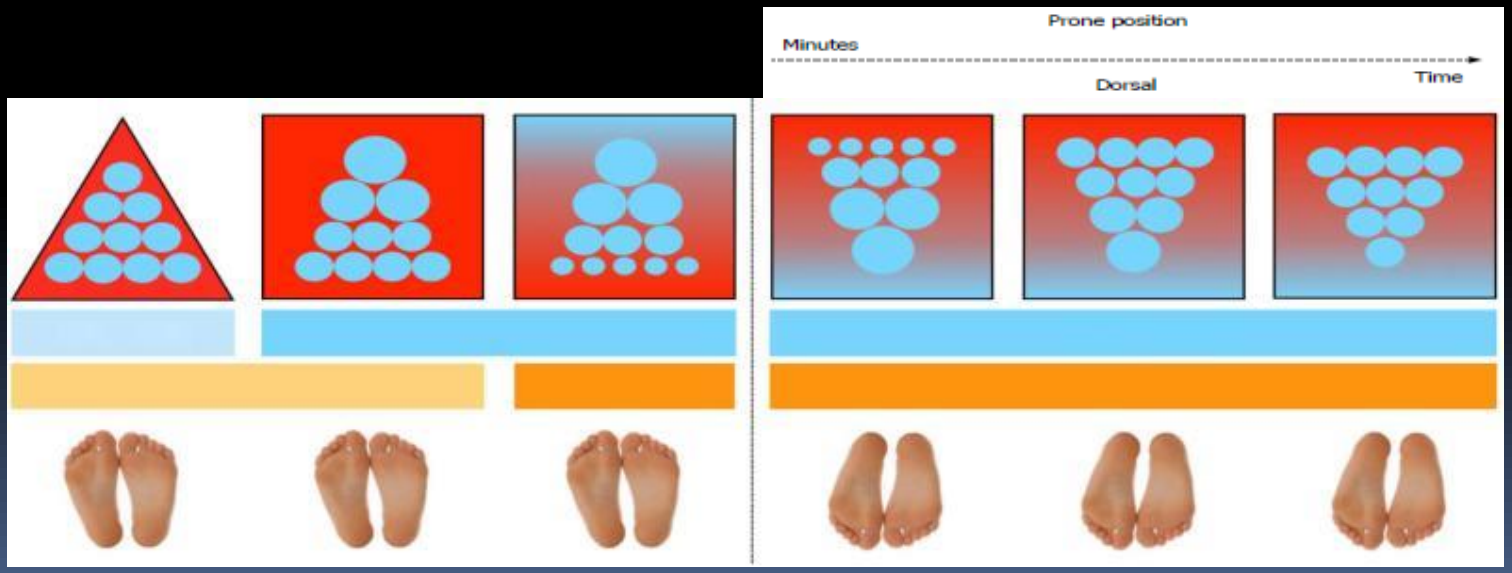
Eric L. Scholten, MD; Jeremy R. Beitler, MD, MPH; G. Kim Prisk, PhD, DSc; and Atul Malhotra, MD



Koulouras V et al . Pathophysiology of prone position in ARDS

Supine position

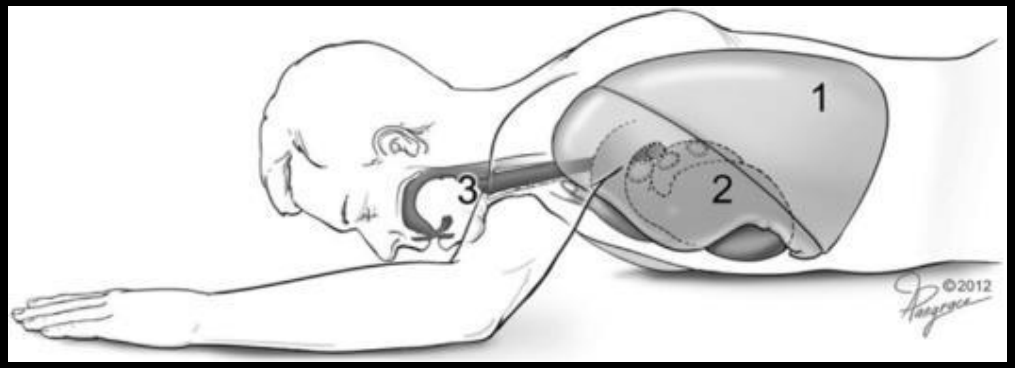
Prone position



Prone positioning allows the heart to lay on the sternum and the compressive force of the heart on dorsal lung regions to be eliminated

Koulouras V et al .
Pathophysiology of prone position in ARDS

In their meta-analysis, Beitler *et al* stratified analysis by high (≥ 12 h/d) or low (< 12 h/d) proning dose and demonstrated a significant reduction in mortality with high doses (RR = 0.71; 95%CI: 0.56-0.90; *P* = 0.004) but not low doses (RR = 1.05; 95%CI: 0.92-1.19; *P* = 0.472).



Difficulties with Oxygenation

Perfusion and Ventilation mismatch

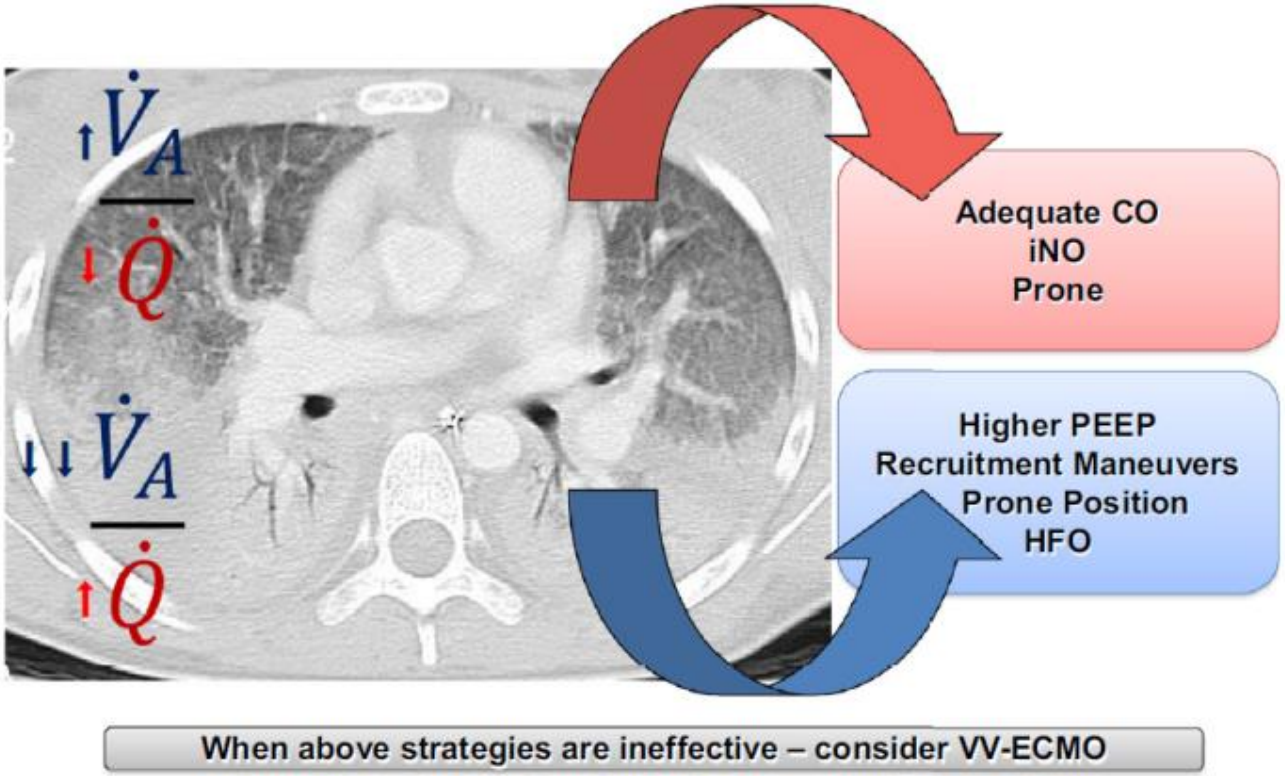


Fig. 1 Mechanisms of action of adjunct therapies during severe ARDS on ventilation/perfusion $\left(\frac{\dot{V}_A}{\dot{Q}}\right)$ distribution. Due to massive loss of aeration and increase in lung tissue as shown on the lung CT slice, hypoxemia results from large amounts of shunt and $\frac{\dot{V}_A}{\dot{Q}}$ mismatch, which may be due to low alveolar ventilation $(\dot{V}_A)$ as compared to lung perfusion $(\dot{Q})$ or to more lung perfusion relative to $\dot{V}_A$. This latter occurs when hypoxic pulmonary vasoconstriction is impaired making the lung perfusion predominant in non-aerated lung regions. To improve $\frac{\dot{V}_A}{\dot{Q}}$ matching and hence oxygenation, interventions may act on both sides of the equation. By increasing perfusion in well ventilated regions, inhaled nitric oxide (iNO) and intravenous fluid challenges redistribute lung perfusion and cardiac output (CO) to well-aerated areas and better match $\frac{\dot{V}_A}{\dot{Q}}$. PEEP can increase aeration in non- or poorly ventilated areas thereby improving $\frac{\dot{V}_A}{\dot{Q}}$. CO may decrease with PEEP, which also contributes to $\frac{\dot{V}_A}{\dot{Q}}$ improvement. High frequency oscillation

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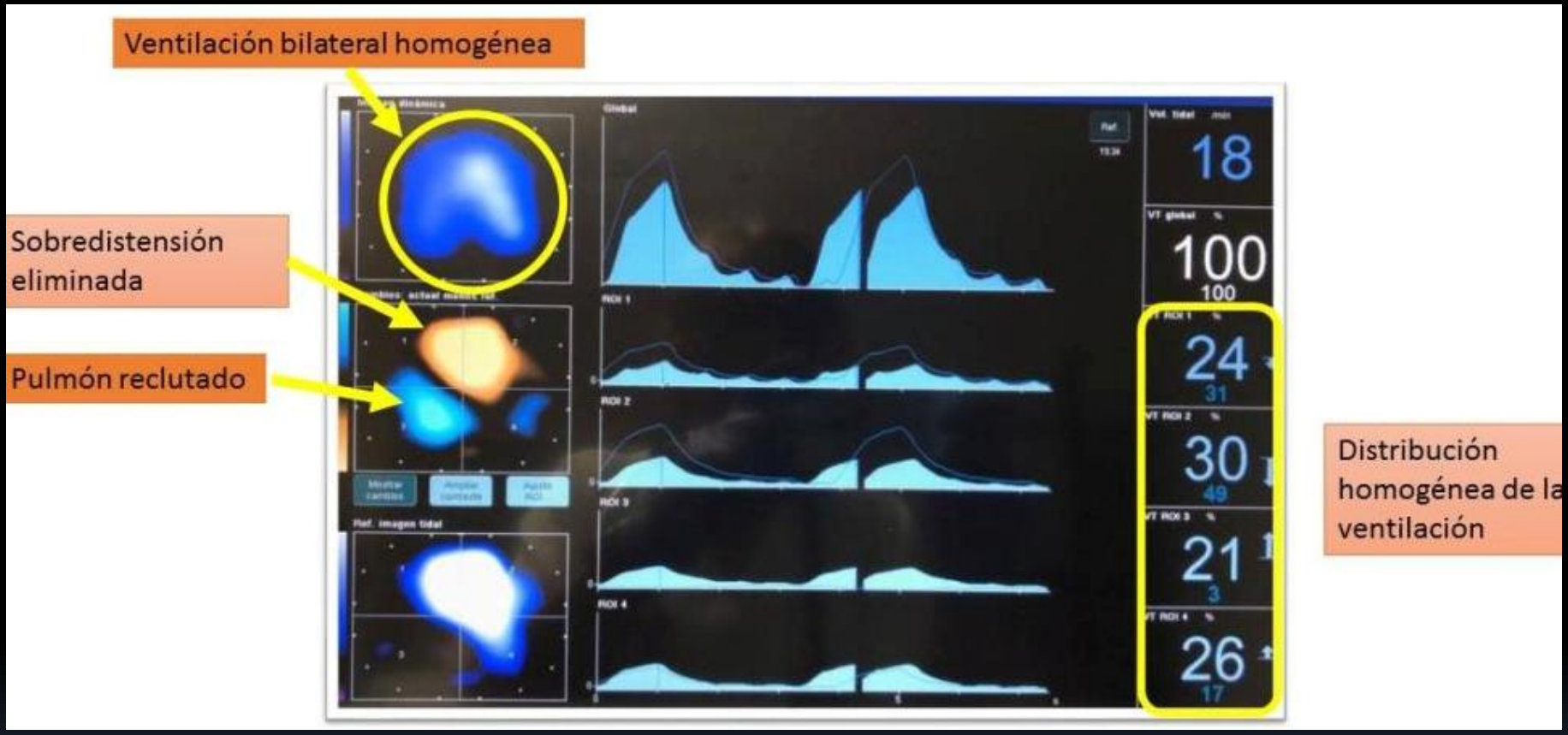
RESPUESTA A PRONO.



Estos cambios se acompañan de aumento de la PaO2/FiO2, disminución del espacio muerto y nos permite bajar la presiones de distensión. AJUSTES INDIVIDUALES PARA CONSEGUIR HACER HOMOGÉNEA LA VENTILACIÓN.

The Proning Severe ARDS Patients (PROSEVA) study investigators

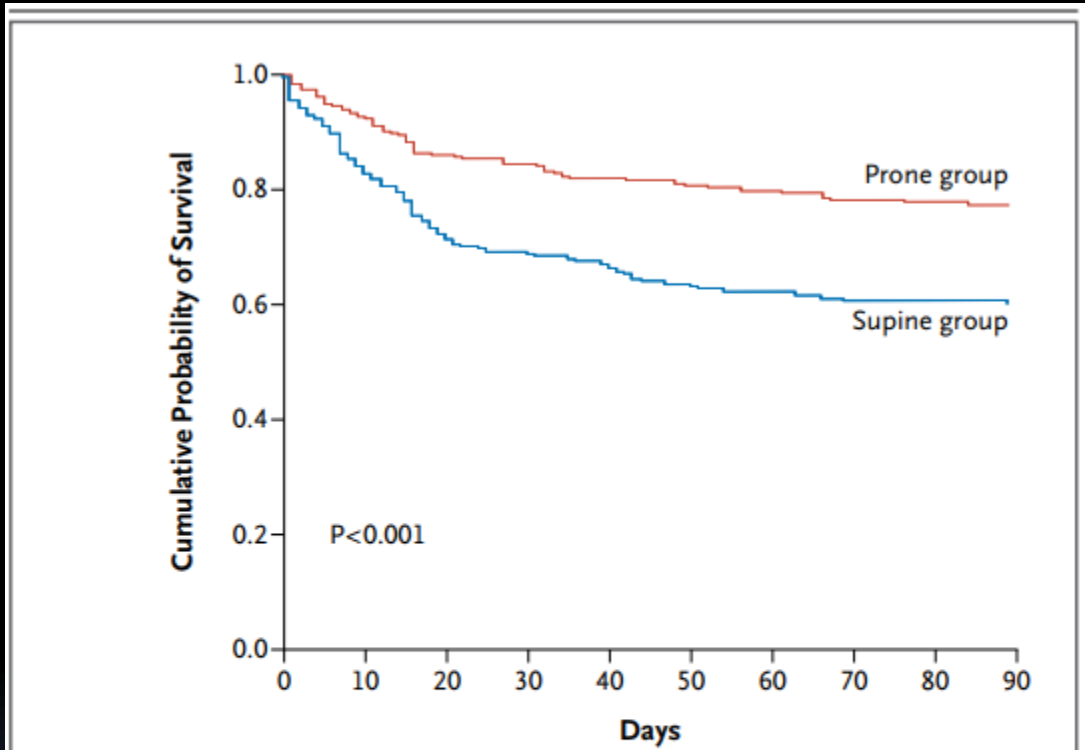


Figure 2. Kaplan–Meier Plot of the Probability of Survival from Randomization to Day 90.

N Engl J Med 2013; 368:2159-2168June 6, 2013 DOI: 10.1056/NEJMoa1214103

PROSEVA

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 JUNE 6, 2013 VOL. 368 NO. 23

Prone Positioning in Severe Acute Respiratory Distress Syndrome

Claude Guérin, M.D., Ph.D., Jean Reignier, M.D., Ph.D., Jean-Christophe Richard, M.D., Ph.D., Pascal Beuret, M.D., Arnaud Gacouin, M.D., Thierry Boulain, M.D., Emmanuelle Mercier, M.D., Michel Badet, M.D., Alain Mercat, M.D., Ph.D., Olivier Baudin, M.D., Marc Clavel, M.D., Delphine Chatellier, M.D., Samir Jaber, M.D., Ph.D., Sylvène Rosselli, M.D., Jordi Mancebo, M.D., Ph.D., Michel Sirodot, M.D., Gilles Hilbert, M.D., Ph.D., Christian Bengler, M.D., Jack Richecoeur, M.D., Marc Gainnier, M.D., Ph.D., Frédérique Bayle, M.D., Gael Bourdin, M.D., Véronique Leray, M.D., Raphaele Girard, M.D., Loredana Baboi, Ph.D., and Louis Ayzac, M.D., for the PROSEVA Study Group*

ABSTRACT

BACKGROUND

Previous trials involving patients with the acute respiratory distress syndrome (ARDS) have failed to show a beneficial effect of prone positioning during mechanical ventilatory support on outcomes. We evaluated the effect of early application of prone positioning on outcomes in patients with severe ARDS.

METHODS

In this multicenter, prospective, randomized, controlled trial, we randomly assigned 466 patients with severe ARDS to undergo prone-positioning sessions of at least 16 hours or to be left in the supine position. Severe ARDS was defined as a ratio of the partial pressure of arterial oxygen to the fraction of inspired oxygen (F_{IO_2}) of less than 150 mm Hg, with an F_{IO_2} of at least 0.6, a positive end-expiratory pressure of at least 5 cm of water, and a tidal volume close to 6 ml per kilogram of predicted body weight. The primary outcome was the proportion of patients who died from any cause within 28 days after inclusion.

RESULTS

A total of 237 patients were assigned to the prone group, and 229 patients were assigned to the supine group. The 28-day mortality was 16.0% in the prone group and 32.8% in the supine group (P<0.001). The hazard ratio for death with prone positioning was 0.39 (95% confidence interval [CI], 0.25 to 0.61). Unadjusted 90-day mortality was 23.6% in the prone group versus 41.0% in the supine group (P<0.001), with a hazard ratio of 0.44 (95% CI, 0.29 to 0.67). The incidence of complications did not differ significantly between the groups, except for the incidence of cardiac arrests, which was higher in the supine group.

CONCLUSIONS

In patients with severe ARDS, early application of prolonged prone-positioning sessions significantly decreased 28-day and 90-day mortality. (Funded by the Programme Hospitalier de Recherche Clinique National 2006 and 2010 of the French Ministry of Health; PROSEVA ClinicalTrials.gov number, NCT00527813.)

The New England Journal of Medicine
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- 466 patients with severe ARDS
- Prone position vs supine position
- Prone position was associated with
  - Mejora de la mortalidad
  - 28 day: 16% vs 33%
  - 90 day: 24% vs 41%
  - Menos paros cardíacos
  - No hay diferencias en las complicaciones

(OSCAR) study group

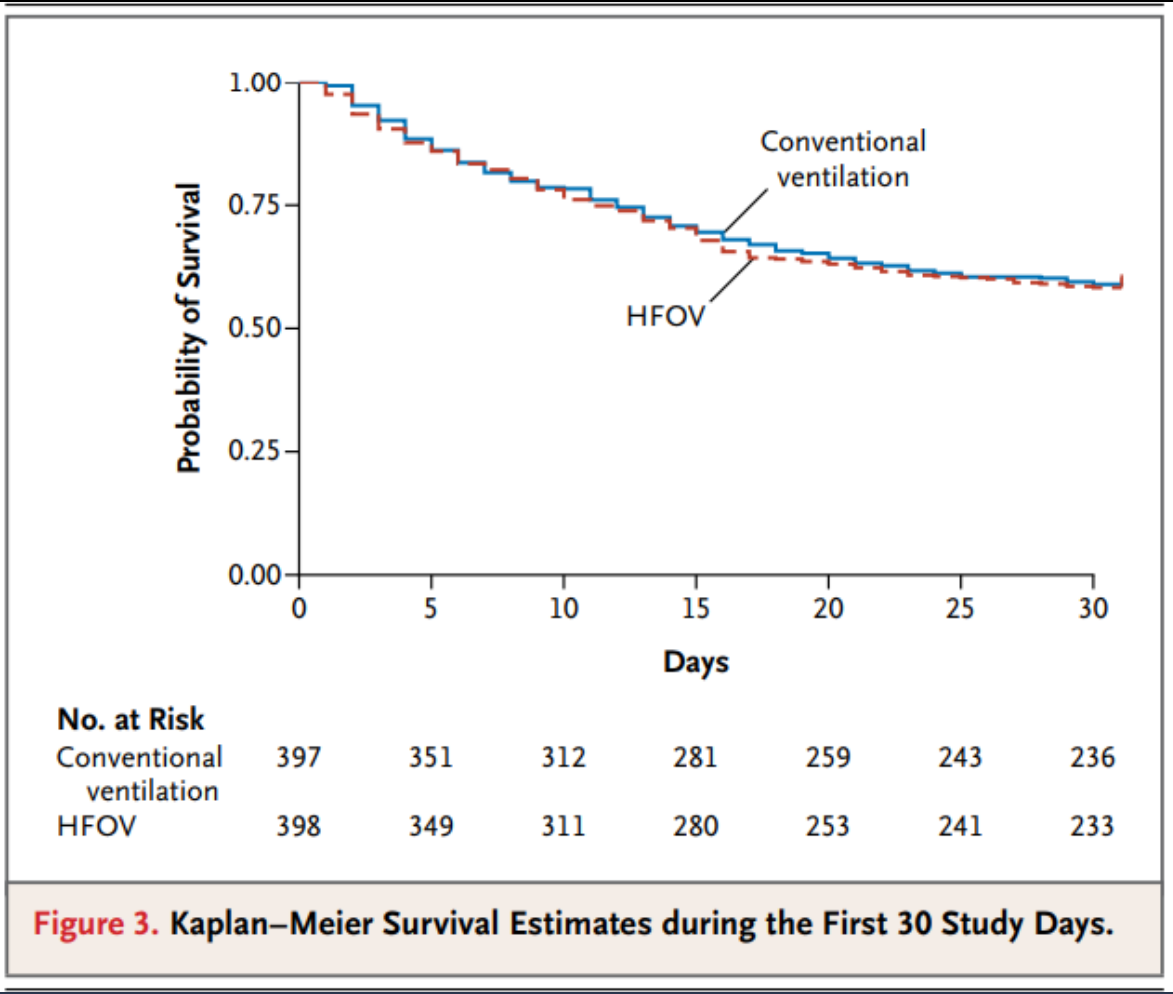

Figure 3. Kaplan–Meier Survival Estimates during the First 30 Study Days.

El uso de VAFO no tuvo un efecto significativo sobre la mortalidad a los 30 días en pacientes ventilación mecánica para SDRA.

N Engl J Med 2013;368:806-13. DOI: 10.1056/NEJMoa1215716

OSCILLATE

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 FEBRUARY 28, 2013 VOL. 368 NO. 9

High-Frequency Oscillation in Early Acute Respiratory Distress Syndrome

Niall D. Ferguson, M.D., Deborah J. Cook, M.D., Gordon H. Guyatt, M.D., Sangeeta Mehta, M.D., Lori Hand, R.R.T., Peggy Austin, C.C.R.A., Qi Zhou, Ph.D., Andrea Matte, R.R.T., Stephen D. Walter, Ph.D., Francois Lamontagne, M.D., John T. Granton, M.D., Yaseen M. Arabi, M.D., Alejandro C. Arroliga, M.D., Thomas E. Stewart, M.D., Arthur S. Slutsky, M.D., and Maureen O. Meade, M.D., for the OSCILLATE Trial Investigators and the Canadian Critical Care Trials Group*

ABSTRACT

BACKGROUND

Previous trials suggesting that high-frequency oscillatory ventilation (HFOV) reduced mortality among adults with the acute respiratory distress syndrome (ARDS) were limited by the use of outdated comparator ventilation strategies and small sample sizes.

METHODS

In a multicenter, randomized, controlled trial conducted at 39 intensive care units in five countries, we randomly assigned adults with new-onset, moderate-to-severe ARDS to HFOV targeting lung recruitment or to a control ventilation strategy targeting lung recruitment with the use of low tidal volumes and high positive end-expiratory pressure. The primary outcome was the rate of in-hospital death from any cause.

RESULTS

On the recommendation of the data monitoring committee, we stopped the trial after 548 of a planned 1,200 patients had undergone randomization. The two study groups were well matched at baseline. The HFOV group underwent HFOV for a median of 3 days (interquartile range, 2 to 8); in addition, 34 of 273 patients (12%) in the control group received HFOV for refractory hypoxemia. In-hospital mortality was 47% in the HFOV group, as compared with 35% in the control group (relative risk of death with HFOV, 1.33; 95% confidence interval, 1.09 to 1.64; P=0.005). This finding was independent of baseline abnormalities in oxygenation or respiratory compliance. Patients in the HFOV group received higher doses of midazolam than did patients in the control group (199 mg per day (interquartile range, 100 to 382) vs. 141 mg per day (interquartile range, 68 to 248), P<0.001), and more patients in the HFOV group than in the control group received neuromuscular blockers (83% vs. 68%, P<0.001). In addition, more patients in the HFOV group received vasoactive drugs (91% vs. 84%, P=0.01) and received them for a longer period than did patients in the control group (5 days vs. 3 days, P=0.01).

CONCLUSIONS

In adults with moderate-to-severe ARDS, early application of HFOV, as compared with a ventilation strategy of low tidal volume and high positive end-expiratory pressure, does not reduce, and may increase, in-hospital mortality. (Funded by the Canadian Institutes of Health Research; Current Controlled Trials numbers, ISRCTN42992782 and ISRCTN87124254, and ClinicalTrials.gov numbers, NCT00474656 and NCT01506401.)

From the Interdepartmental Division of Critical Care Medicine (N.D.F., S.M., J.T.G., T.E.S., A.S.S.), Departments of Medicine and Physiology (N.D.F.), the Department of Medicine, Division of Respiratory (S.M., J.T.G., T.E.S.), and the Departments of Medicine, Surgery, and Biomedical Engineering (A.S.S.), University of Toronto, University Health Network and Mount Sinai Hospital (N.D.F., J.T.G.), Mount Sinai Hospital (S.M., T.E.S.), St. Michael's Hospital (A.S.S.), and the Critical Care Program, University Health Network (A.M.), Toronto; the Interdepartmental Division of Critical Care, Hamilton Health Sciences (D.J.C., M.O.M.), the Departments of Medicine and Clinical Epidemiology and Biostatistics (D.J.C., G.H.G., S.D.W., M.O.M.), and the CLARITY Research Centre (D.J.C., G.H.G., S.D.W., M.O.M., L.H., P.A., Q.Z.), McMaster University, Hamilton, ON, and the Centre de Recherche Clinique Etienne Le Bel, Université de Sherbrooke, Sherbrooke, QC (F.L.) — all in Canada; the Intensive Care Department, King Saud Ibn Abdulaziz University for Health Sciences, Riyadh, Saudi Arabia (Y.M.A.); and the Department of Medicine, Scott and White Healthcare and Texas A&M Health Science Center College of Medicine, Temple (A.C.A.). Address requests to Dr. Meade at 1280 Main St. W., Hamilton, ON L8N 3Z5, Canada, or at meade@mcmaster.ca.

*A complete list of the investigators in the Oscillation for Acute Respiratory Distress Syndrome Treated Early (OSCILLATE) trial is provided in the Supplementary Appendix, available at NEJM.org.

This article was published on January 22, 2013, at NEJM.org.

N Engl J Med 2013;368:795-805.
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- 548 patients with moderate - severe ARDS
- HFOV vs low Vt/High PEEP CV (MV < 3d)
- Trial stopped early as harm with HFOV
- HFOV
  - Hospital mortality 47% vs 35%
  - More sedation
  - More NMBA's
  - More vasopressors

From: **Comparison of High-Frequency Oscillatory Ventilation and Conventional Mechanical Ventilation in Pediatric Respiratory Failure**

JAMA Pediatr. 2014;168(3):243-249. doi:10.1001/jamapediatrics.2013.4463

Table. Summaries for Matched Study Population

Variable	HFOV vs CMV			Early HFOV vs CMV		
	HFOV (n = 882)	CMV (n = 882)	*P* Value[a]	Early HFOV (n = 471)	CMV (n = 471)	*P* Value[a]
Baseline characteristics						
Age at ICU admission, mean (SD), mo	63.5 (66.8)	64.6 (68.2)	.73	68.9 (68.8)	71.2 (70.2)	.63
Weight, mean (SD), kg	22.6 (23.4)	22.7 (22.9)	.97	24.5 (24)	24.3 (23.5)	.93
Male, No. (%)	448 (50.8)	432 (48.9)	.47	234 (49.7)	252 (53.5)	.27
Developmental delay, No. (%)	95 (10.7)	110 (12.5)	.28	50 (10.6)	45 (9.5)	.66
Genetic syndrome, No. (%)	83 (9.4)	79 (9.0)	.80	44 (9.3)	45 (9.5)	.99
Center volume, mean (SD), cases per center	210.6 (154.5)	206.7 (142.9)	.59	207.9 (154.9)	208.0 (142.2)	.99
Clinical data at admission, mean (SD)						
PIM-2 score	−3.2 (1.9)	−3.2 (1.8)	.37	−3.0 (1.8)	−3.0 (1.8)	.64
PRISM-3 score	11.1 (8.7)	11.2 (8.6)	.60	13.1 (9.3)	12.9 (9.2)	.48
Admission from inpatient unit, No. (%)	325 (39.2)	338 (40.5)	.96	170 (38.5)	179 (40.2)	.62
Systolic BP, mean (SD), mm Hg						
High	124.9 (22.7)	125.3 (21.5)	.77	129.1 (24.2)	129.9 (24.9)	.70
Low	78.7 (18.6)	78.7 (18.4)	.64	77.3 (19.3)	78.3 (19.7)	.48
Heart rate, mean (SD), /min						
High	166.4 (26.5)	166.4 (28.5)	.88	166.2 (26.0)	166.1 (28.9)	.96
Low	119.3 (27.9)	119.1 (29.0)	.87	117.1 (29.4)	117.7 (29.3)	.67
Other clinical data, No. (%)						
CPR or defibrillation	130 (15.5)	131 (15.4)	.89	81 (18.2)	82 (18.2)	.93
ECMO	60 (6.8)	52 (5.9)	.42	31 (6.6)	36 (7.6)	.58
Arterial catheter	787 (89.2)	797 (90.3)	.25	418 (88.7)	430 (91.3)	.09
Central venous access	852 (96.6)	857 (97.2)	.53	451 (95.7)	447 (94.9)	.61
Hemodialysis catheter	116 (13.1)	113 (12.8)	.88	65 (13.8)	70 (14.9)	.69
Outcomes						
Mortality, No. (%)	152 (17.3)	74 (8.4)	<.001	85 (18.1)	39 (8.3)	<.001
Duration of mechanical ventilation, mean (SD), d	20.3 (19.7)	14.6 (15.4)	<.001[b]	15.9 (15.9)	14.6 (16.4)	<.001[b]
ICU length of stay, mean (SD), d	24.9 (22.3)	19.1 (18.5)	<.001[b]	19.5 (16.9)	19.3 (18.0)	.03[b]

Abbreviations: CMV, conventional mechanical ventilation; CPR, cardiopulmonary resuscitation; ECMO, extracorporeal membrane oxygenation; HFOV, high-frequency oscillatory ventilation; ICU, intensive care unit; PIM-2, Pediatric Index of Mortality; PRISM-3, Pediatric Risk of Mortality.

[a] *P* values were obtained using the paired 2-sample *t* test for continuous variables and the McNemar test for categorical variables.

[b] *P* values were obtained using the frailty model for the paired time-to-event data.

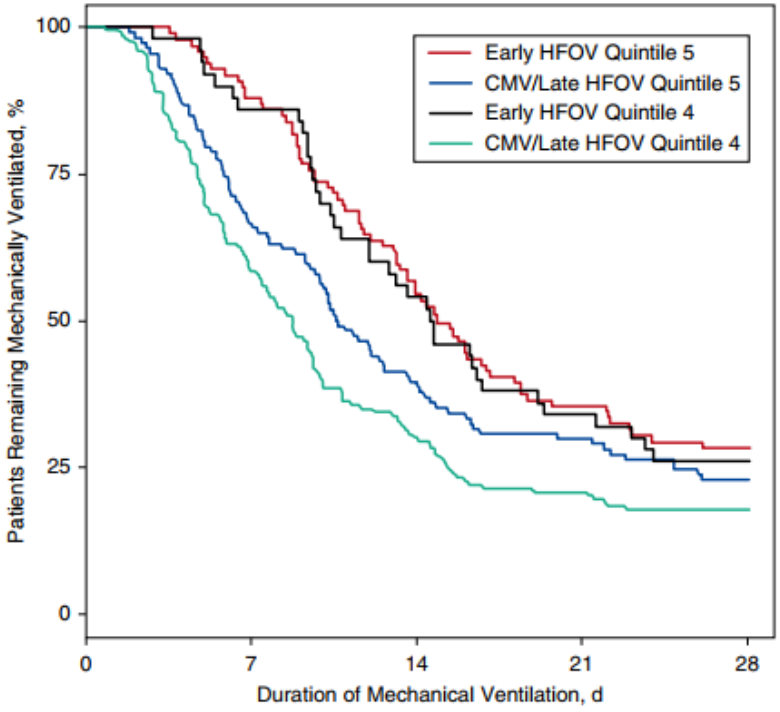
Más mortalidad, Más días de VM, Más días de estancia UCI

ORIGINAL ARTICLE

Early High-Frequency Oscillatory Ventilation in Pediatric Acute Respiratory Failure

A Propensity Score Analysis

Scot T. Bateman[1], Santiago Borasino[2], Lisa A. Asaro[3], Ira M. Cheifetz[4], Shelley Diane[5], David Wypij[3,6,7], and Martha A. Q. Curley[8,9,10]; for the *RESTORE* Study Investigators



No. at risk:

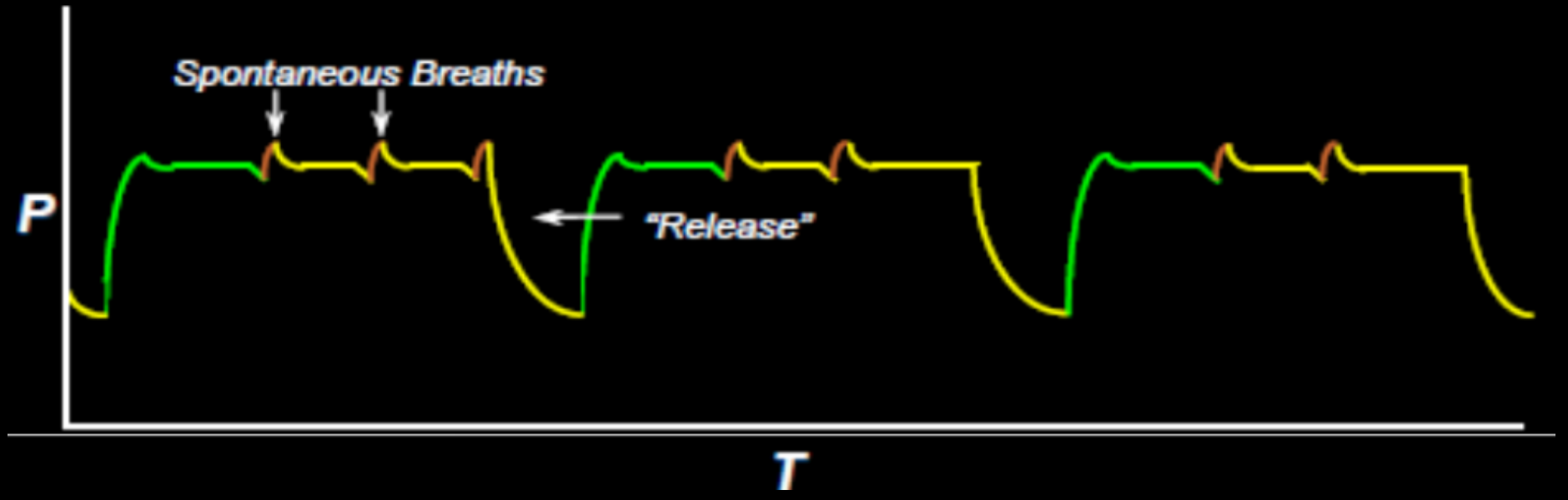
Early HFOV Q5	99	87	54	35	28
CMV/Late HFOV Q5	114	76	45	34	26
Early HFOV Q4	50	43	27	17	13
CMV/Late HFOV Q4	163	95	49	34	29

Figure 1. Duration of mechanical ventilation to Day 28 by model 1 propensity score quintile and group. CMV = conventional mechanical ventilation; HFOV = high-frequency oscillatory ventilation; Q = quintile.

Nuestro análisis de la base de datos RESTORE, las variables de los ajustes del ventilador demuestran impacto negativo estadísticamente significativo temprano en niños con insuficiencia a PARDS severo. El VAFO puede tener un papel en el PARDS en el momento de lesión pulmonar agresiva seguida recuperación pulmonar y más rápida conversión a CMV. La aplicación de VAFO para PARDS puede no ser completamente hasta que tengamos una ensayo controlado aleatorio.

Am J Respir Crit Care Med. 2016 Mar 1;193(5):495-503. doi: 10.1164/rccm.201507-1381OC.

Hablando de APRV: ¿Qué es y qué no es?



"Proporciona periodos largos de insuflación intercalados con breves periodos de deflación pulmonar"

Programando APRV:

Configuración Pediatría

Phigh similar a la presión plateau deseada (típicamente 20-30 cm H2O)

Nota: Phigh 30 cm H2O puede ser necesario en pacientes con distensibilidad torácica/abdominal reducida o obesidad mórbida.

Phigh 25 cm H2O, se recomienda el uso de un circuito de ventilador no conforme para minimizar la compresión del volumen del circuito.

Plow: 0 cm H2O

Thigh: 3-5 segundos

Tlow: 0.2-0.8

Configuración Neonatos

Phigh similar a la presión plateau deseada (típicamente 10-25 cm H2O)

Nota: puede ser necesario un Phigh 25 cm H2O en pacientes con distensibilidad torácica/abdominal disminuida. Con un Phigh: 25 cm H2O, se recomienda el uso de un circuito de ventilador no conforme para minimizar la compresión del volumen del circuito.

Plow: 0 cm H2O

Thigh: 2-3 segundos

Tlow: 0.2-0.4

Airway pressure release ventilation (APRV) clinical guide
Crit Care Med 2005 Vol. 33, No. 3

Mode
Volume Control
Admit patient
Status
10-29 11 33
Set Ventilation Mode
Bi-Vent
Bi-Vent RR 10 b/min
Bi-Vent I:E 6.9:1
Basic
Insp. times
Trigger
Supported breath
P high
30
0
cmH2O
50
T high
5.5
0.2
s
10.0
Trigg. Flow
5
-20
0
PS above P high
0
0
cmH2O
120
PEEP
0
0
cmH2O
50
T PEEP
0.8
0.2
s
10.0
Insp. cycle off
30
1
%
40
PS above PEEP
0
0
cmH2O
120
O2 conc.
100
21
%
100
T insp. rise
0.15
0.00
s
0.40
PEEPtot
11:33
0
C static
11:33
29
E
11:33
35
C dyn
(ml/cmH2O)
17
Ri
11:32
18
Re
11:33
27
WOB v
(J/l)
2.93
Tc
11:33
0.80
Show Previous Mode
time: 11:30
Cancel
Accept
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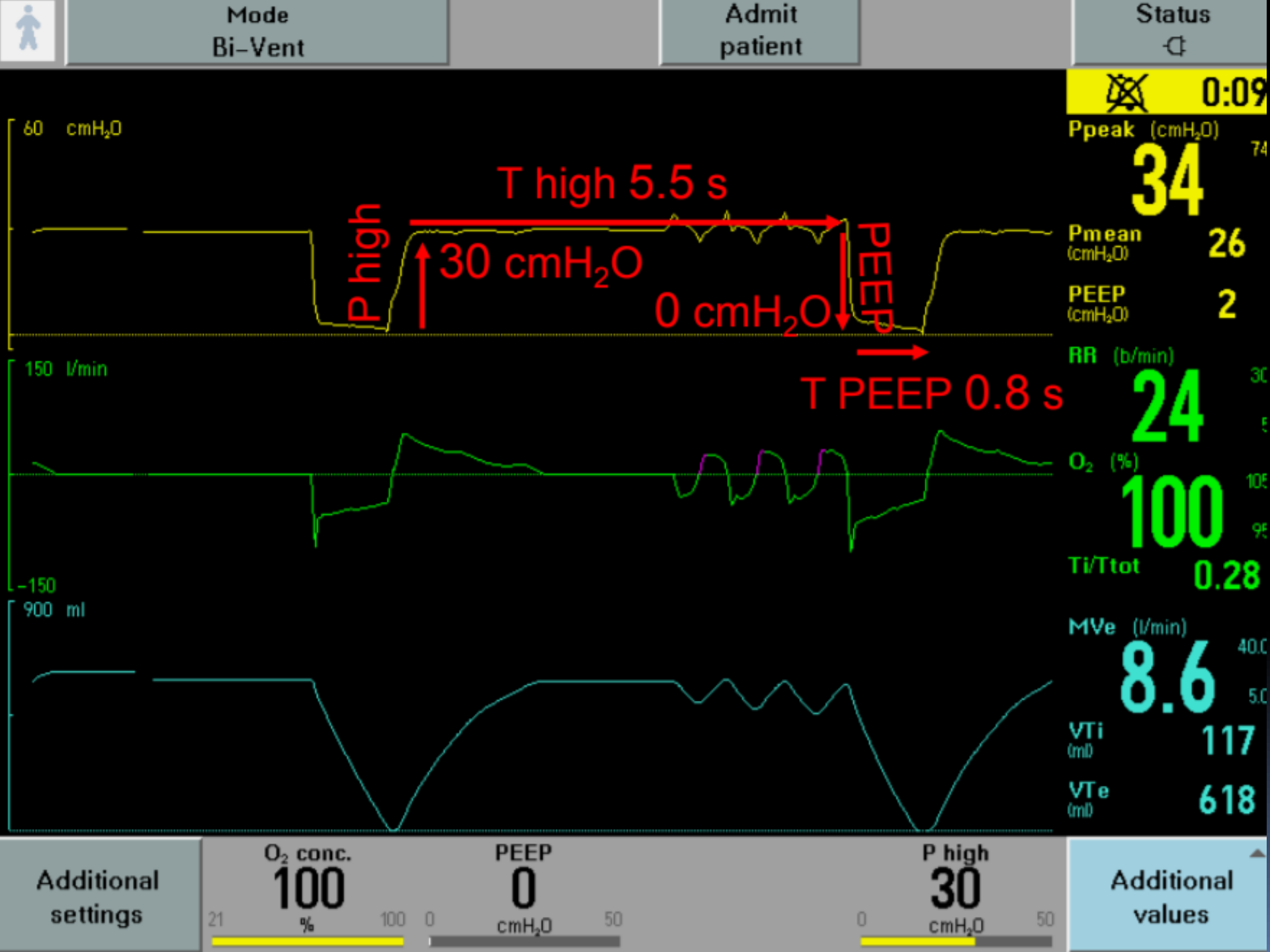

Mode
Bi-Vent
Admit patient
Status
0:09
60 cmH2O
T high 5.5 s
P high
30 cmH2O
0 cmH2O
PEEP
T PEEP 0.8 s
150 l/min
-150
900 ml
Ppeak (cmH2O)
34
74
Pmean (cmH2O)
26
PEEP (cmH2O)
2
RR (b/min)
24
30
O2 (%)
100
105
95
Ti/Ttot
0.28
MVe (l/min)
8.6
40.0
5.0
VTi (ml)
117
VTe (ml)
618
Additional settings
O2 conc.
100
21
%
100
PEEP
0
0
cmH2O
50
P high
30
0
cmH2O
50
Additional values

Transición de ventilación volumen-control a APRV

Presión meseta se mantuvo constante: 30kg

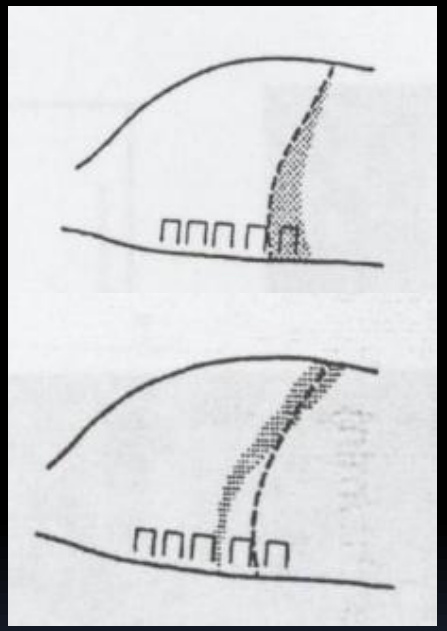
	CMV-VC	APRV
Presión Pico	32 cm H2O	26 cm H2O
Presión Meseta	26 cm H2O	26 cm H2O
Paw media	11 cm H2O	22 cm H2O
PEEP	5 cm H2O	5 cm H2O
FR	20/min	20/min
Vt liberado	240 cc	320 cc
Ventilacion minuto	4.8 L/min	6.4 L/min
PaCO2(mmhg)	35	31

APRV

Efectos a nivel pulmonar

Putensen estudio a 30 pacientes con múltiples trauma y Distrés a APRV vs SIMV más PS.

- Incremento en la compliansa pulmonar.
- Mejoría en la oxigenación.
- Reducción del shunt.
- Menos días de asistencia respiratoria mecánica.
- Menor estadía en UCI.
- Menos uso de sedación y vasopresores.



Reduce la atrofia de los músculos respiratorios asociados a la ventilación mecánica y el uso de agentes paralizantes.

Al preservar la ventilación espontánea el movimiento normal del diafragma previene el desarrollo de atelectasias.

Putensen, AJRCCM, 2001;164: 43-49.

CORRESPONDENCE

Airway pressure release ventilation in acute respiratory distress syndrome: children are not miniature adults

Vijai Williams[1], Suresh Kumar Angurana[1], Muralidharan Jayashree[1*], Yongfang Zhou[2] and Yan Kang[2]

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Beneficios fisiológicos notables de APRV han sido reportado en experimentos animales recientes. Sin embargo, los datos de pacientes con SDRA aún se limitan a pequeños ensayos clínicos con una amplia variación en la configuración de APRV, y las conclusiones de estos estudios son controvertidas.

APRV altamente sofisticado, impulsado por la fisiología, perfil mecánico dinámico con ajustes precisos.

EVALUATING THE EFFECTS OF AIRWAY PRESSURE RELEASE VENTILATION, A NOVEL MODE OF VENTILATION, IN CHILDREN WITH ACUTE RESPIRATORY FAILURE

S Kambhampati, S Kache, J Williamson, T Roberts, S Messing, J Arimura. *Stanford University, Stanford, CA, USA*

Esta revisión retrospectiva de casos estudia la seguridad y utilidad de APRV en niños con SDRA entre abril de 2010 y noviembre de 2011

UCI pediátrica de Stanford

Abstract 996 Table 1 Respiratory Parameters

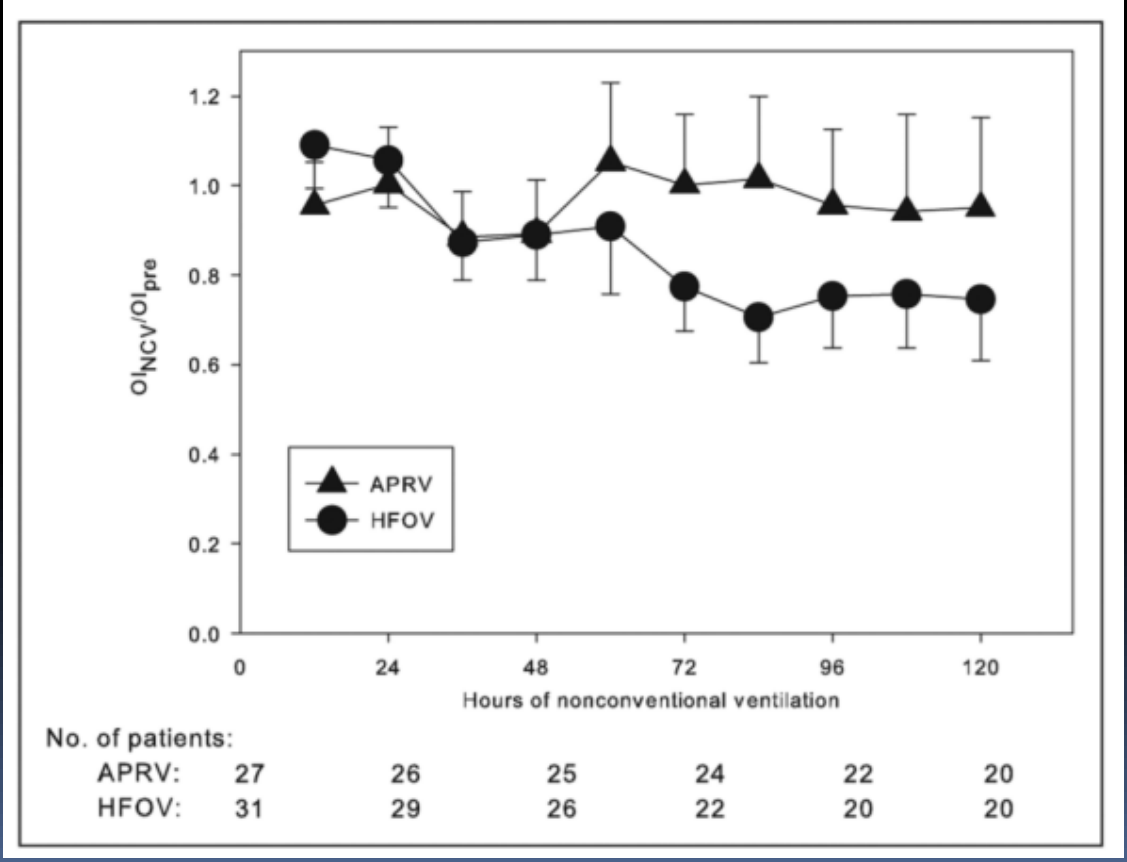
	-24 hours (baseline)	-2 hours (prior to APRV)	+2 hours (after APRV)	+24 hours (after APRV)	Mean Difference (95% CI)
PaO2/FiO2	127.19584	111.99251	156.91240	198.98324	28.4980173 (3.444559 to 58.5142879)
Oxygenation Index	19.31633	22.85918	22.34526	18.16486	3.56910055 (–3.7849048 to 9.62734808)

P / F y OI mejoraron significativamente después de la conversión a APRV. Todas las medidas secundarias evaluadas se mantuvieron estables (datos no mostrados).

doi:10.1136/archdischild-2012-302724.0996

Improved Oxygenation 24 Hours After Transition to Airway Pressure Release Ventilation or High-Frequency Oscillatory Ventilation Accurately Discriminates Survival in Immunocompromised Pediatric Patients With Acute Respiratory Distress Syndrome*

Nadir Yehya, MD1, Alexis A. Topjian, MD, MSCE1, Neal J. Thomas, MD, MSc2, and Stuart H. Friess, MD3



Pediatric patients with an ICC failing conventional ventilation and transitioning to either APRV or HFOV have a high mortality rate. Improved oxygenation at 24 hours expressed as PF24/PF pre or OI24/OIpre reliably discriminates survivors from nonsurvivors. A prospective trial between these two modes of NCV for pediatric ARDS in an ICC population is warranted.

Pediatr Crit Care Med. Author manuscript; available in PMC 2014 July 29.

Zhongguo Dang Dai Er Ke Za Zhi. 2017 Apr;19(4):430-435.

[A Meta analysis of the efficacy of high-frequency oscillatory ventilation versus conventional mechanical ventilation for treating pediatric acute respiratory distress syndrome].

[Article in Chinese]

Qiao JY[1], Li YZ, Wang HY, Zhang SD.

Cochrane Library, PubMed, Medline, CNKI, and Wanfang Data

6 studies involving 246 patients were included

CONCLUSIONES:

Aunque la tasa de mortalidad no es reducida por la VAFO en niños con SDRA, este tratamiento puede resultar en una mejora significativa en la oxigenación en comparación con CMV. Se requerirán otros ensayos clínicos aleatorios de gran muestra, multicéntricos, para establecer una conclusión definitiva.

PALICC: APRV in children

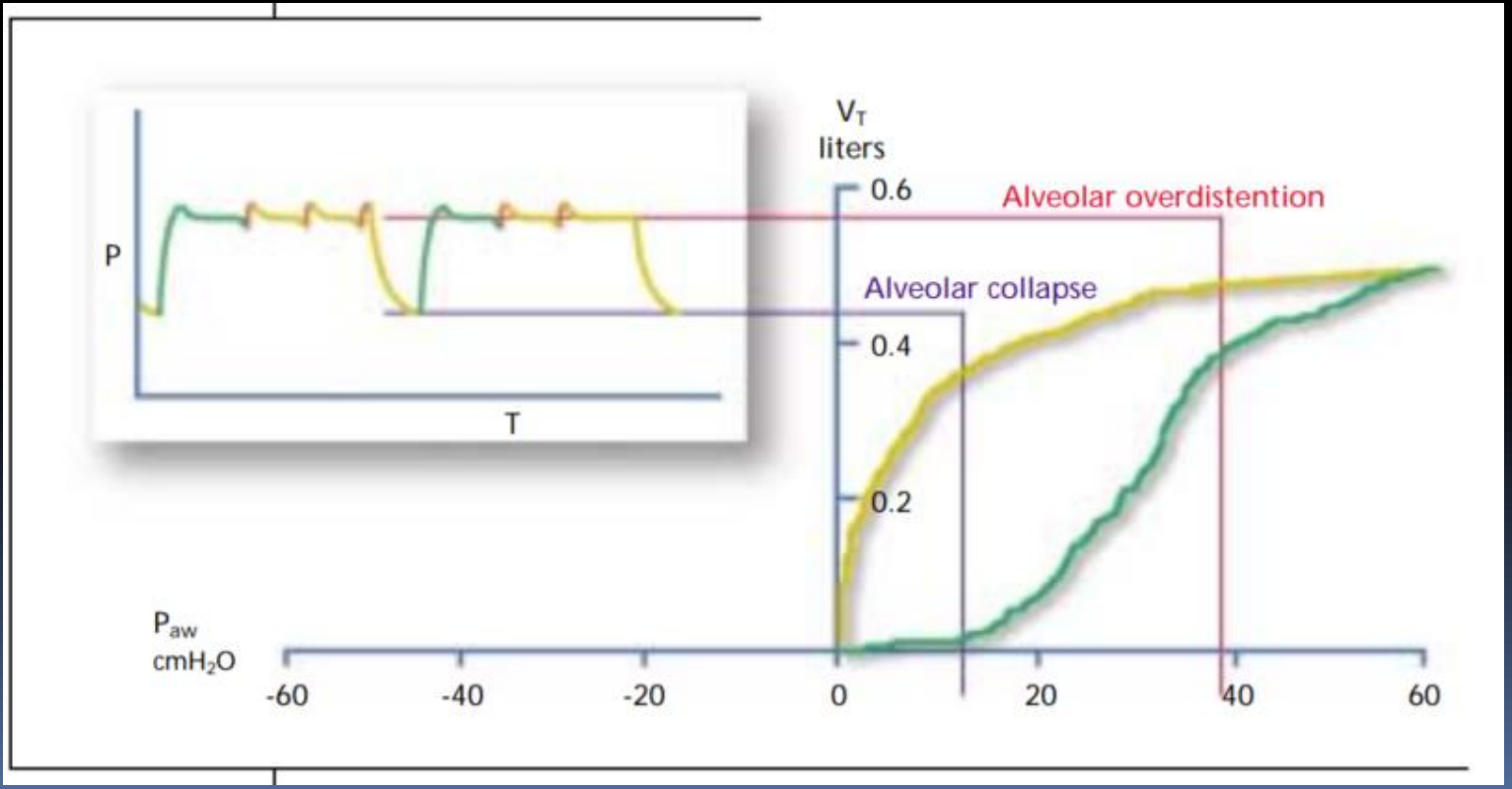
No hay datos para mostrar superioridad sobre cualquier otro enfoque ventilatorio

PALICC concluyó que no hay datos adecuados para respaldar su uso.

La redacción es importante porque PALICC no dice que no puede o no debe usarlos; las declaraciones dicen que su uso de rutina no puede recomendarse.

APRV in children

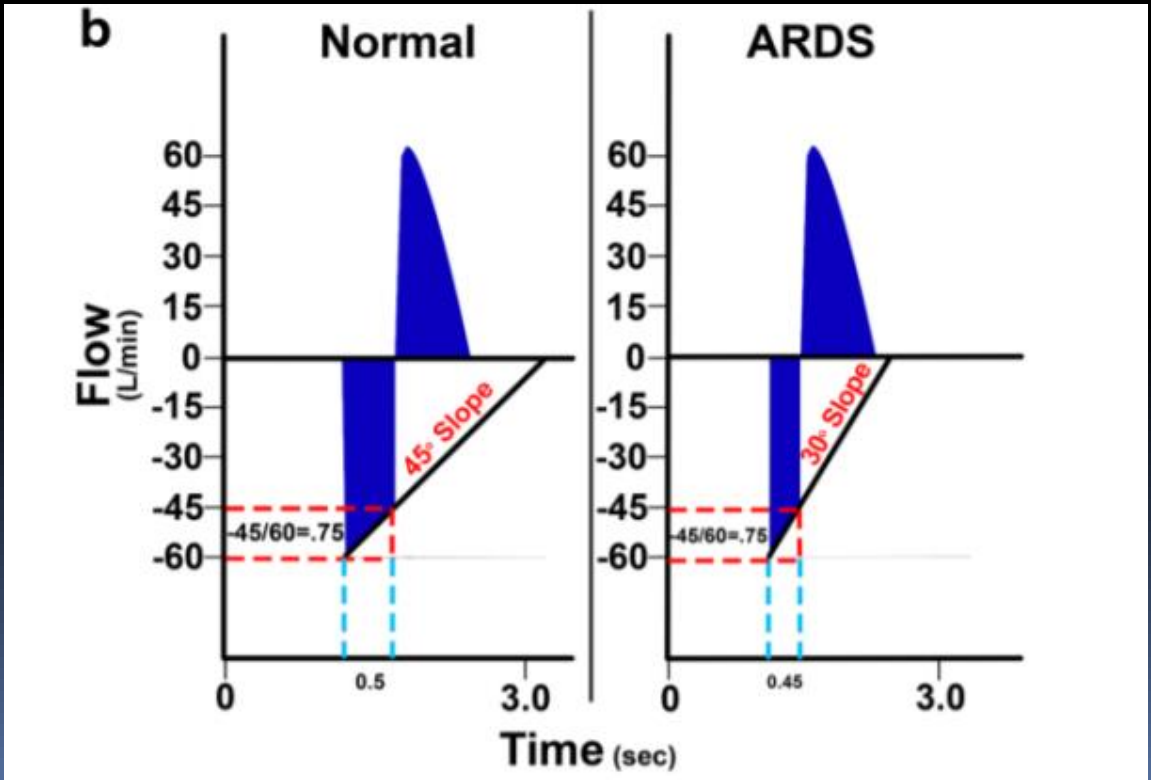
Increase mean airway pressures
Preventing alveolar collapse
Reduce the risk of overdistention
Reduce intrapulmonary presures
Increased cardiac output

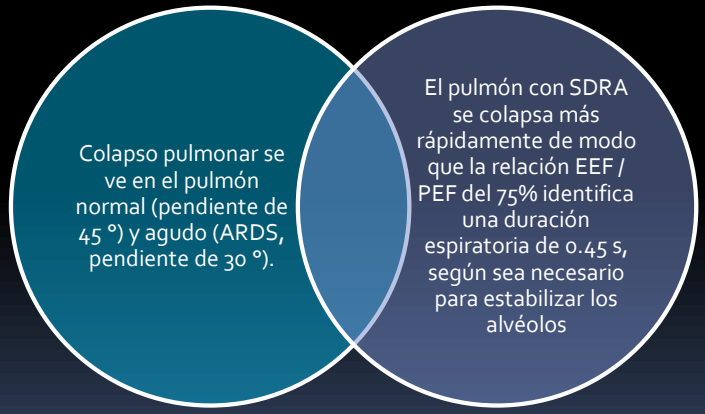


The 30-year evolution of airway pressure release ventilation (APRV)

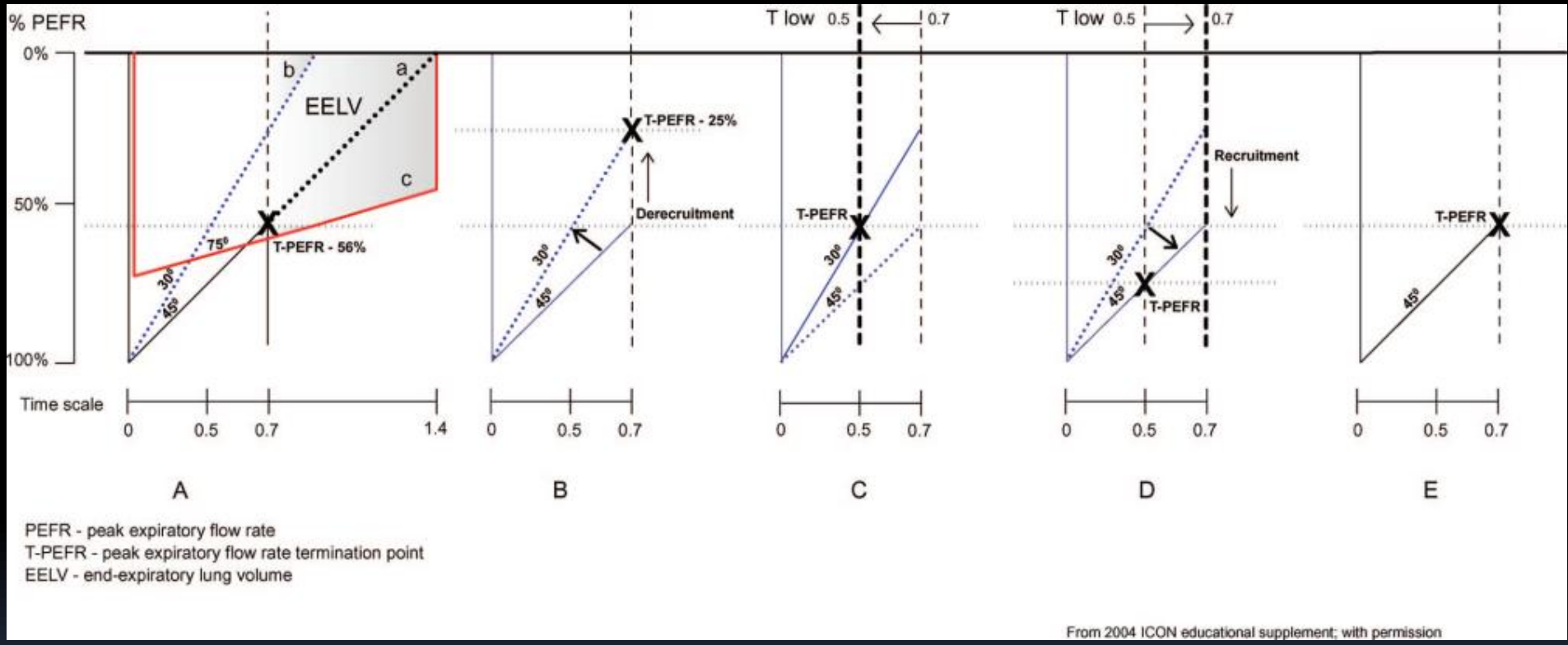
Sumeet V. Jain1 , Michaela Kollisch-Singule1 , Benjamin Sadowitz1 , Luke Dombert1 , Josh Satalin1*, Penny Andrews2 , Louis A. Gatto1,3, Gary F. Nieman1 and Nader M. Habashi2

Estudios APRV divididos en dos básicas categorías:
(1) APRV de fijación fija (F-APRV) en el que se establece la fase de liberación y se deja constante;
(2) APRV personalizado (P-APRV) en el cual se establece sobre los cambios en la mecánica pulmonar usando la pendiente de la curva de flujo espiratorio





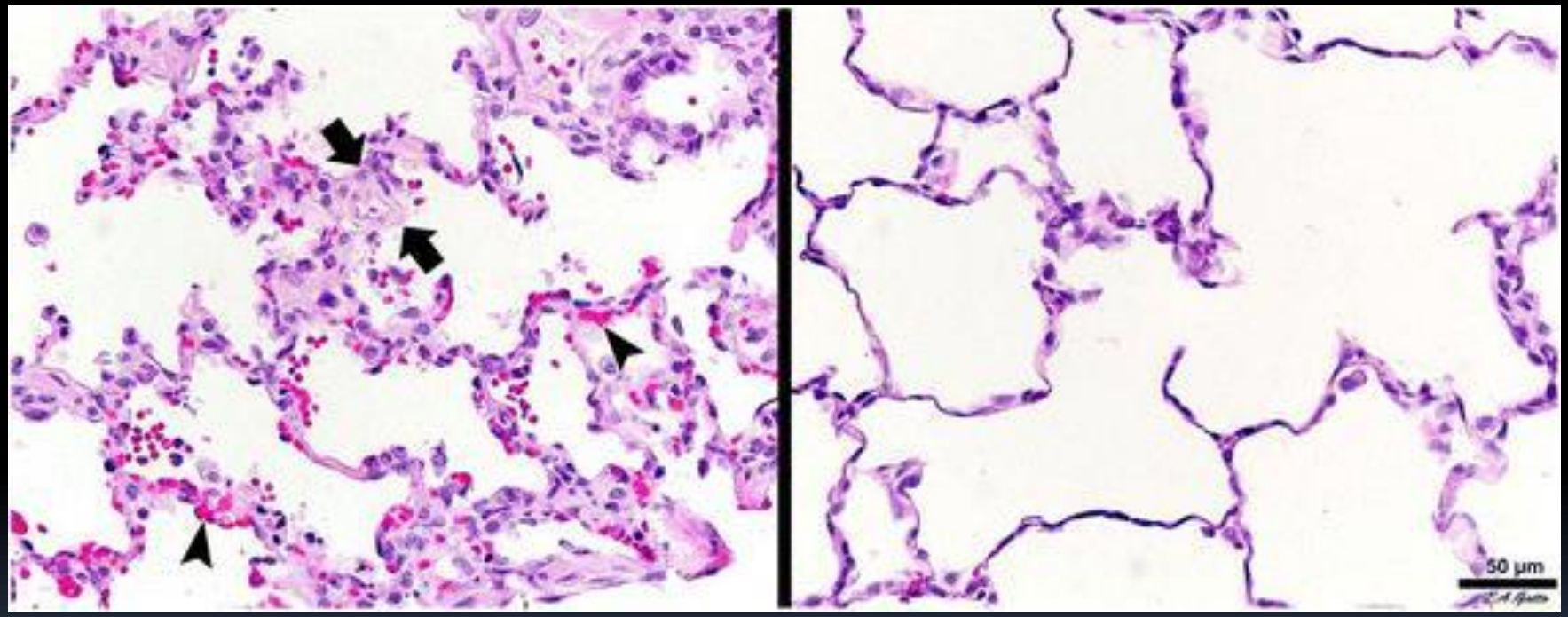
Jain et al. Intensive Care Medicine Experimental (2016) 4:11 DOI 10.1186/s40635-016-0085-2



PEFR - peak expiratory flow rate
T-PEFR - peak expiratory flow rate termination point
EELV - end-expiratory lung volume

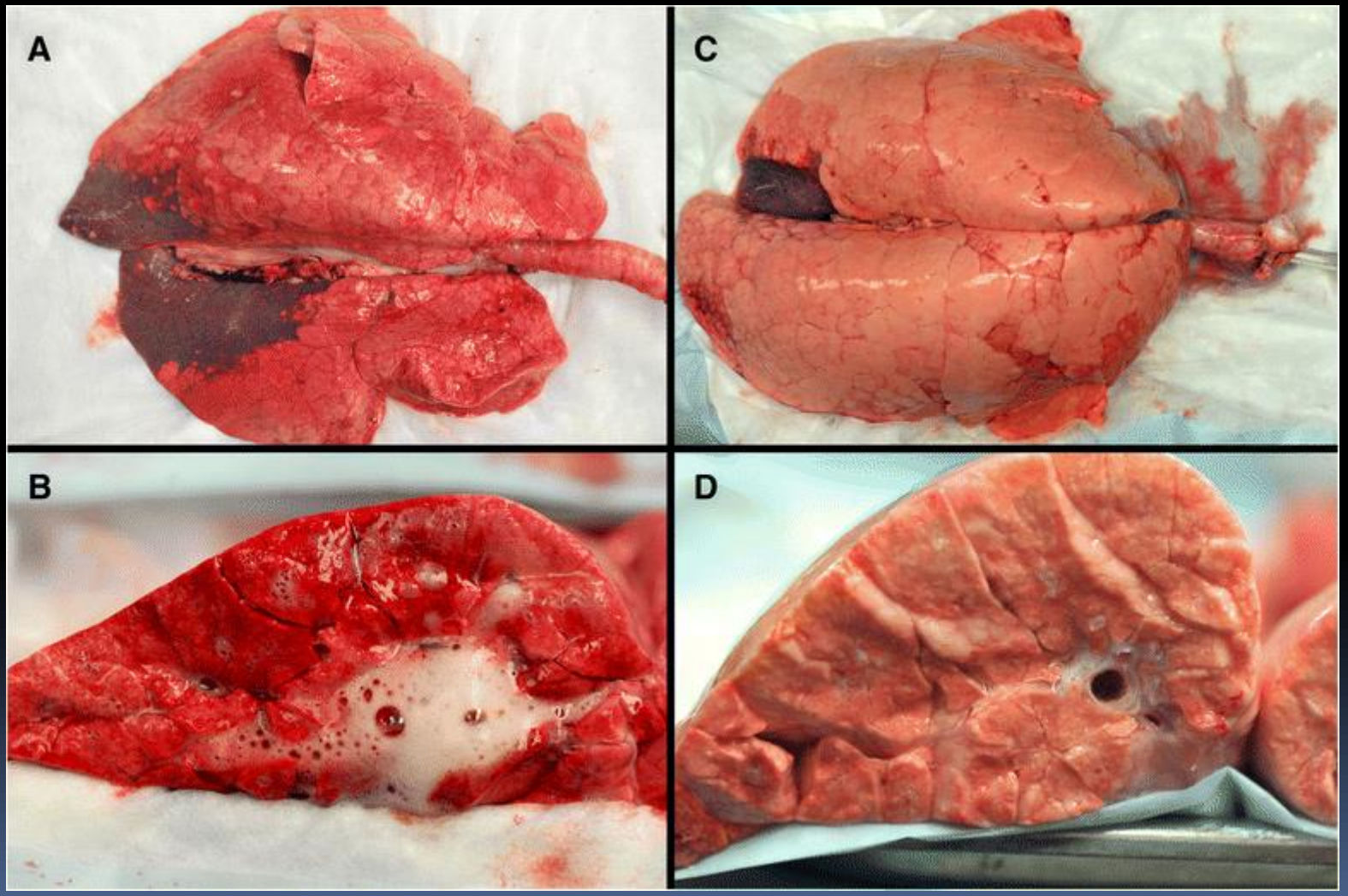
From 2004 ICON educational supplement; with permission

Low tidal volume (LVt; *left*) demonstrated increased alveolar wall thickness (*between arrows*) and vessel congestion (*arrowheads*) as compared with Airway Pressure Release Ventilation (APRV; *right*)



Intensive Care Medicine Experimental 2015 **3**:35

Low tidal volume ventilation lungs (**a**) inflated heterogeneously with prominent dependent and basilar atelectasis and the cut surface (**b**) revealing airway edema. Airway Pressure Release Ventilation gross lungs (**c**) and cut surface (**d**) were pink, light and inflated homogeneously without airway edema



Intensive Care Medicine Experimental 2015 **3**:35

APRV in children

- ✓ El tiempo High es una estrategia usada para mejorar la oxigenación.
- ✓ La válvula de exhalacion activa permite la respiracion espontanea en cualquier parte del ciclo. El paciente control el tiempo de su respiracion espontanea, idenpendientemente de Phigh o Plow.
- ✓ Vt. Liberado es dependiente de Distensibilidad, Resistencia y tiempo de Plow.
- ✓ Al igual que en modos convencionales, la Pmeseta y Vt altos deben de ser evitados.
- ✓ Controversia en Vt bajo cuando la Pmeseta es controlada.

APRV in children

- ✓ Disminucion en la presion intratoraxica en respiracion espontanea.
- ✓ No se han descrito efectos cardiovasculares. En 24 pac con SIRPA se encontro increment en VDFVD y en IC, con mejoria en oxigenación.
- ✓ Se ha documentado incremento en el flujo sanguíneo renal y filtración glomerular en forma significativa.
- ✓ También se ha observado incremento en el flujo sanguineo de mucosa del estomago, intestine Delgado y grueso.

APRV in children

- ✓ Por ser un modo volume variable, existe el riesgo de dar excesivo volume corriente.
- ✓ Al igual que en otros modos ventilatorios, se puede generar presión transpulmonar excesiva, causando sobredistensión y lesion pulmonar.

Airway Pressure Release Ventilation in Pediatric Acute Respiratory Distress Syndrome: A Randomized Controlled Trial

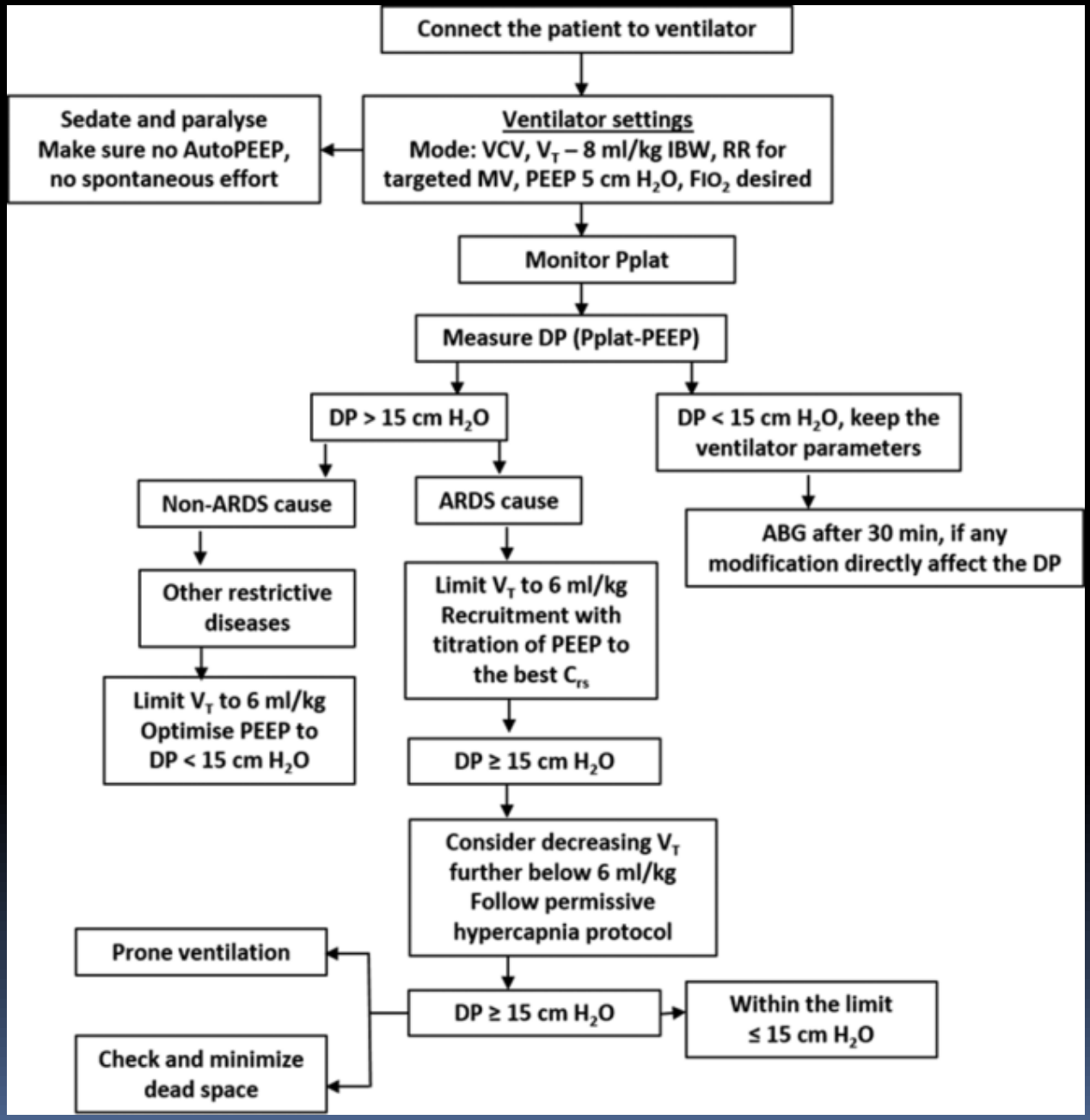
Aunque las series de casos describen los beneficios de la liberación de presión de las vías respiratorias ventilación (APRV), este modo de ventilación no ha sido evaluado contra la ventilación convencional con volumen corriente bajo en niños con insuficiencia respiratoria aguda.

Objetivos: Comparar el efecto de APRV y VM convencional en numero de días en niños con SDRA.

Los días sin ventilación fueron estadísticamente similar en ambos grupos. La mortalidad por cualquier causa a los 28 días fue del 53.8% en APRV en comparación con 26.9% entre los controles.

APRV, como estrategia de ventilación primaria en niños con SDRA, fue asociado con una tendencia hacia una mayor mortalidad en comparación con el convencional. Se deben tener en cuenta las limitaciones al interpretar estos resultados.

Am J Respir Crit Care Med. 2018 Apr 11


Connect the patient to ventilator
Ventilator settings
Mode: VCV, V_T – 8 ml/kg IBW, RR for targeted MV, PEEP 5 cm H_2O, FIO_2 desired
Sedate and paralyse
Make sure no AutoPEEP, no spontaneous effort
Monitor Pplat
Measure DP (Pplat-PEEP)
DP > 15 cm H_2O
DP < 15 cm H_2O, keep the ventilator parameters
ABG after 30 min, if any modification directly affect the DP
Non-ARDS cause
ARDS cause
Other restrictive diseases
Limit V_T to 6 ml/kg Optimise PEEP to DP < 15 cm H_2O
Limit V_T to 6 ml/kg Recruitment with titration of PEEP to the best C_{rs}
DP ≥ 15 cm H_2O
Consider decreasing V_T further below 6 ml/kg Follow permissive hypercapnia protocol
Prone ventilation
DP ≥ 15 cm H_2O
Within the limit ≤ 15 cm H_2O
Check and minimize dead space

Recommendations for mechanical ventilation of critically ill children from the Paediatric Mechanical Ventilation Consensus Conference (PEMVECC)

Invasive ventilation	
Mode	No recommendation
High-frequency oscillatory ventilation	Consider when conventional ventilation fails May be used in cardiac patients
High-frequency jet/percussive ventilation	No recommendation Do not use high-frequency jet ventilation in obstructive airway disease
Liquid ventilation	Do not use
Extra-corporeal life support	Consider in reversible disease if conventional ventilation and/or HFOV fails
Triggering	Target patient-ventilator synchrony
Inspiratory time/I:E ratio	Set inspiratory time by respiratory system mechanics and underlying disease (use time constant and observe flow-time scalar). Use higher rates in restrictive disease
Maintaining spontaneous breathing	No recommendation
Plateau pressure	Keep ≤28 or ≤29–32 cmH_2O with increased chest wall elastance, ≤30 cmH_2O in obstructive airway disease
Delta pressure	Keep ≤10 cmH_2O for healthy lungs, unknown for any disease condition
Tidal volume	Keep ≤10 mL/kg ideal bodyweight, maybe lower in lung hypoplasia syndromes
PEEP	5–8 cmH_2O, higher PEEP necessary dictated by underlying disease severity (also in cardiac patients) Use PEEP titration, consider lung recruitment (also in cardiac patients) Add PEEP in obstructive airway disease when there is air-trapping and to facilitate triggering Use PEEP to stent upper airways in case of malacia

Intensive Care Med DOI 10.1007/s00134-017-4920-z

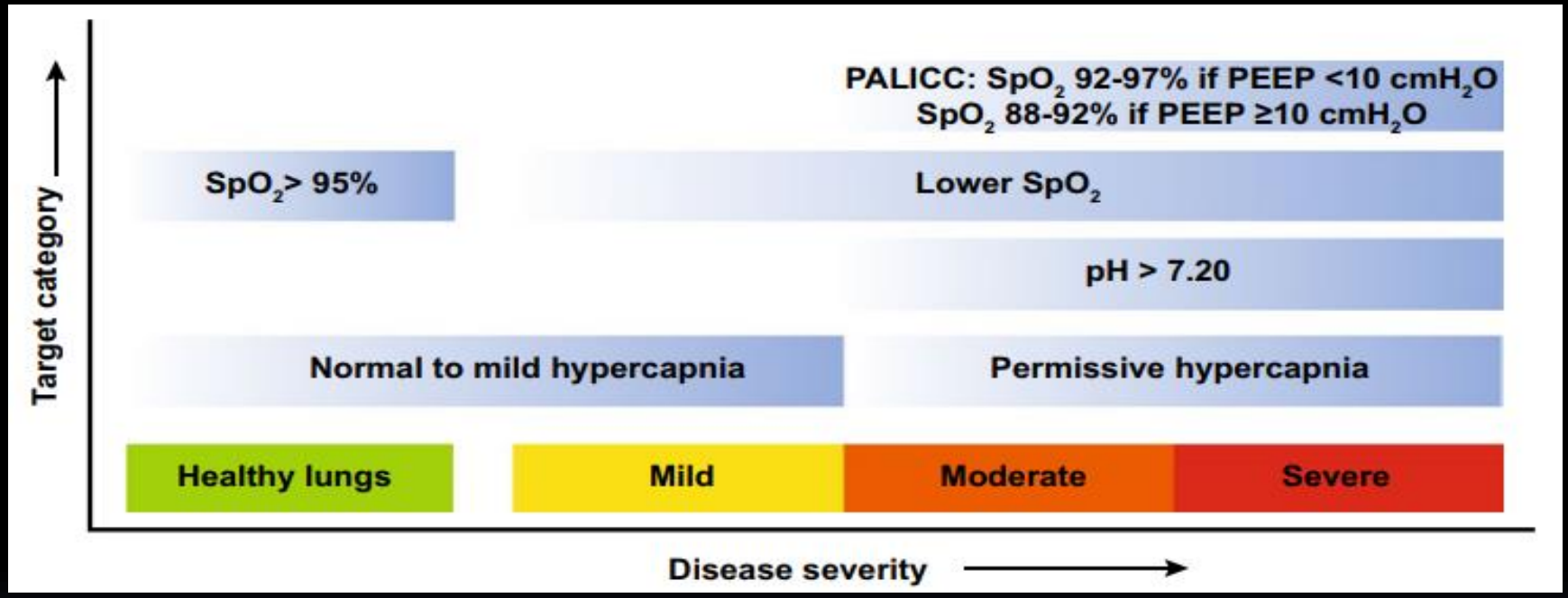
Recommendations for mechanical ventilation of critically ill children from the Paediatric Mechanical Ventilation Consensus Conference (PEMVECC)

La rutina instilación de solución salina isotónica antes de la succión endotraqueal se recomienda (acuerdo fuerte).

No existe una base científica para aspiración endotraqueal de rutina o el acercamiento a la succión (abierto vs. cerrado), aunque la aspiración abierta puede dar lugar a desreclutamiento y la instilación de solución salina isotónica antes a la succión.

Intensive Care Med DOI 10.1007/s00134-017-4920-z

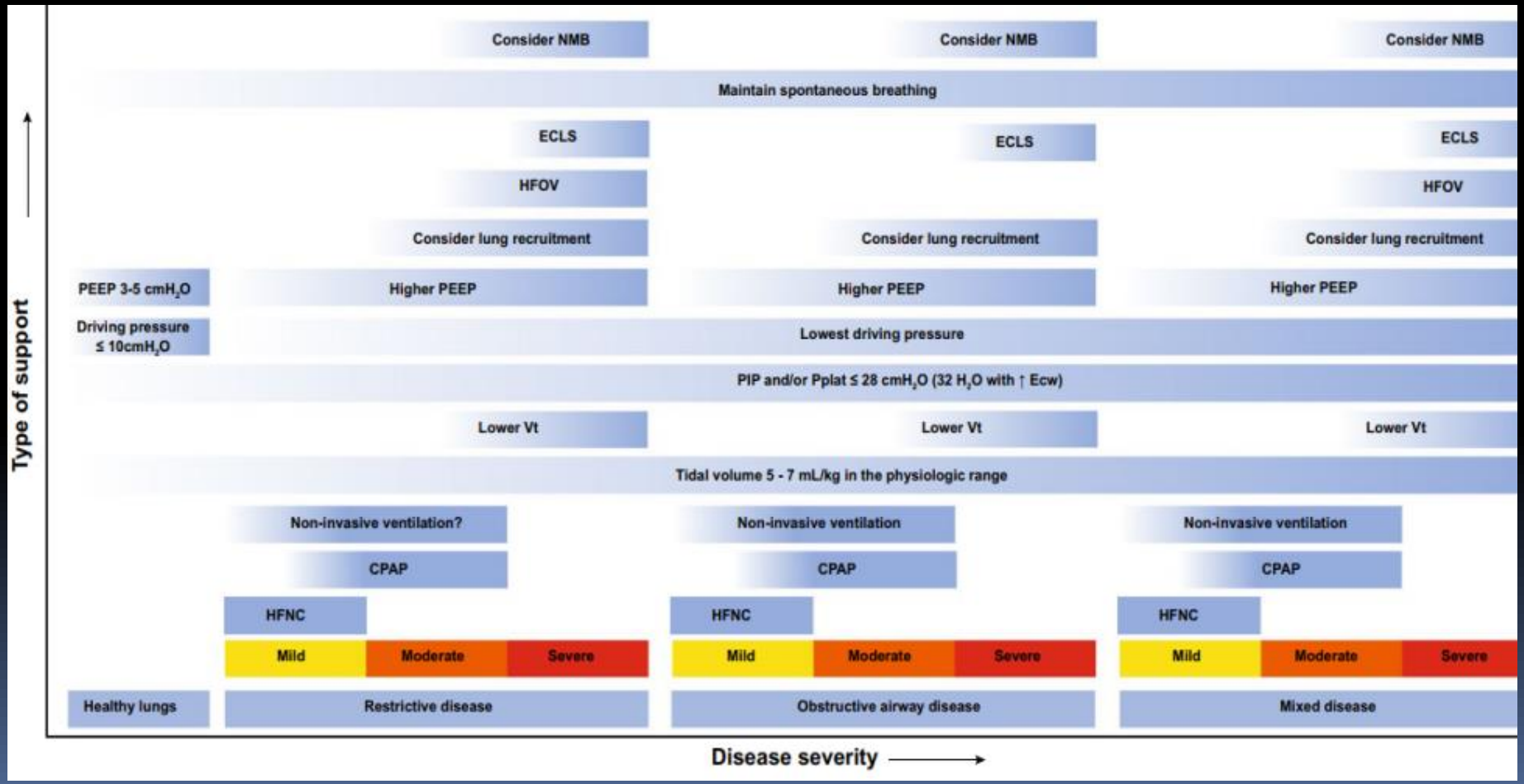
Recommendations for mechanical ventilation of critically ill children from the Paediatric Mechanical Ventilation Consensus Conference (PEMVECC)



Targets	
Oxygenation	$SpO_2 \geq 95\%$ when breathing room air for healthy lungs No threshold for any disease condition or cardiac patients, but keep $SpO_2 \leq 97\%$ For PARDS: SpO_2 92–97% when PEEP < 10cmH_2O and 88–92% when PEEP ≥10 cmH_2O
Ventilation	PCO_2 35–45 mmHg for healthy lungs Higher PCO_2 accepted for acute (non-)pulmonary patients unless specific diseases dictate otherwise Target pH >7.20 Target normal pH for patients with pulmonary hypertension

Intensive Care Med DOI 10.1007/s00134-017-4920-z

Recommendations for mechanical ventilation of critically ill children from the Paediatric Mechanical Ventilation Consensus Conference (PEMVECC)



Functional disability 5 years after ARDS

- 109 survivors from ’98 - ’01
- Interview, PFT’s, 6 min walk test, resting & exercise oximetry, chest imaging, QOL survey
- PFT’s normalish
- BUT 6 min walk test 76% predicted, physical/psychological problems

The NEW ENGLAND JOURNAL of MEDICINE

APRIL 7, 2011

Functional Disability 5 Years after Acute Respiratory Distress Syndrome

Margaret S. Herridge, M.D., M.P.H., Catherine M. Tansey, M.Sc., Andrea Matté, B.Sc., George Tomlinson, Ph.D., Natalia Diaz-Granados, M.Sc., Andrew Cooper, M.D., Cameron B. Guest, M.D., C. David Mazer, M.D., Sangeeta Mehta, M.D., Thomas E. Stewart, M.D., Paul Kudlow, B.Sc., Deborah Cook, M.D., Arthur S. Slutsky, M.D., and Angela M. Cheung, M.D., Ph.D., for the Canadian Critical Care Trials Group

ABSTRACT

BACKGROUND

There have been few detailed, in-person interviews and examinations to obtain follow-up data on 5-year outcomes among survivors of the acute respiratory distress syndrome (ARDS).

METHODS

We evaluated 109 survivors of ARDS at 3, 6, and 12 months and at 2, 3, 4, and 5 years after discharge from the intensive care unit. At each visit, patients were interviewed and examined, underwent pulmonary-function tests, the 6-minute walk test, resting and exercise oximetry, chest imaging, and a quality-of-life evaluation, and reported their use of health care services.

RESULTS

At 5 years, the median 6-minute walk distance was 486 m (76% of predicted distance) and the Physical Component Score on the Medical Outcomes Study 36-Item Short-Form Health Survey was 41 (mean norm score matched for age and sex, 50). With respect to this score, younger patients had a greater rate of recovery than older patients, but neither group returned to normal predicted levels of physical function at 5 years. Pulmonary function was normal to near-normal. A constellation of other physical and psychological problems developed or persisted in patients and family caregivers for up to 5 years. Patients with more coexisting illnesses incurred greater 5-year costs.

CONCLUSIONS

Exercise limitation, physical and psychological sequelae, decreased physical quality of life, and increased costs and use of health care services are important legacies of severe lung injury.

from the Department of Medicine, University Health Network (M.S.H., C.M.T., A.M., G.T., P.K., A.M.C.); the Interdepartmental Division of Critical Care (M.S.H., A.C., C.B.G., C.D.M., S.M., T.E.S., A.S.S.); the Department of Public Health Sciences (N.D.-G., A.M.C.); the Departments of Critical Care Medicine and Anesthesia, Sunnybrook Health Sciences Centre (A.C., C.B.G.); the Departments of Anesthesia (C.D.M.) and Medicine and Critical Care Medicine (A.S.S.) and the Keenan Research Center at the Li Ka Shing Knowledge Institute (C.D.M., A.S.S.), St. Michael’s Hospital, the Dalla Lana School of Public Health (G.T., N.D.-G., A.M.C.) and the Departments of Medicine (S.M., T.E.S.) and Anesthesia (T.E.S.), Mount Sinai Hospital — all at the University of Toronto, Toronto; and the Departments of Medicine and Clinical Epidemiology and Biostatistics, McMaster University, Hamilton, ON, Canada (D.C.). Address reprint requests to Dr. Herridge at Toronto General Hospital, NCSB 13C-1180, 585 University Ave., Toronto, ON M5G 2N2, Canada, or at margaret.herridge@uhn.on.ca.

N Engl J Med 2011;364:1293-304.

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The NEW ENGLAND JOURNAL of MEDICINE
ESTABLISHED IN 1812 SEPTEMBER 16, 2010 VOL. 363 NO. 12

Neuromuscular Blockers in Early Acute Respiratory Distress Syndrome

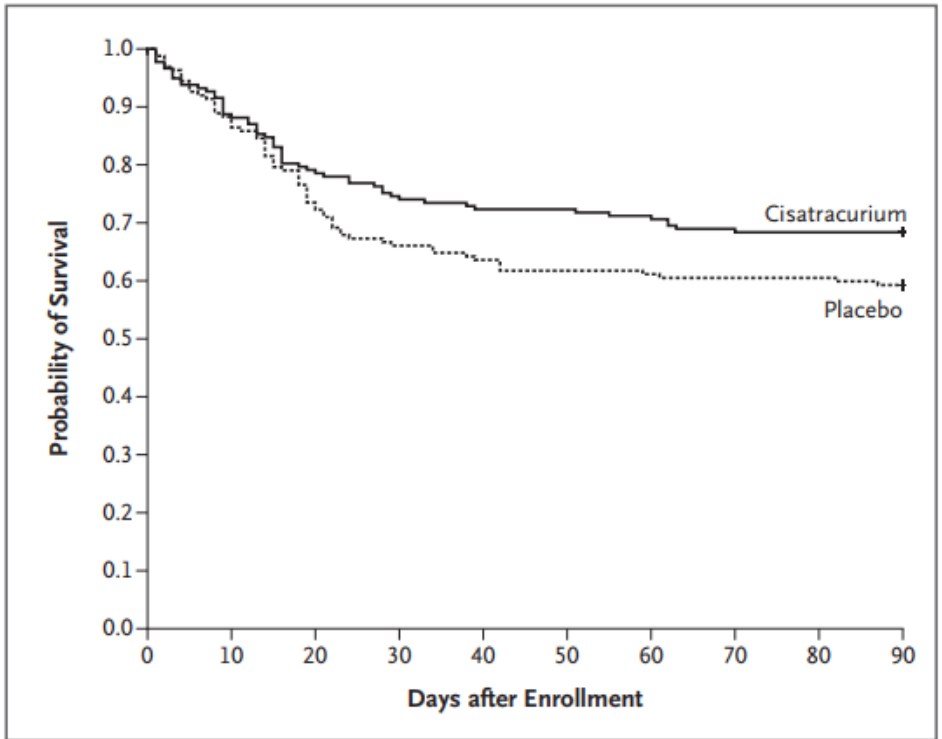


Figure 2. Probability of Survival through Day 90, According to Study Group.

En conclusión, este ensayo multicéntrico prueba que la administración de un medicamento neuromuscular agente de bloqueo temprano en el transcurso de ARDS administrado con ventilación de bajo volumen de corriente puede mejorar los resultados.

PALICC recommends the use of NMB at the minimum effective dose in patients who are unable to achieve effective mechanical ventilation with sedation alone.

Initial Trophic vs Full Enteral Feeding in Patients With Acute Lung Injury The EDEN Randomized Trial

Table 2. Clinical Outcomes

Outcome	Trophic Feeding (n = 508)	Full Feeding (n = 492)	*P* Value
Ventilator-free days, No. (95% CI)	14.9 (13.9-15.8)	15.0 (14.1-15.9)	.89
Failure-free days, No. (95% CI)			
Cardiovascular	19.1 (18.2-20.0)	18.9 (18.1-19.8)	.75
Renal	20.0 (19.0-20/9)	19.4 (18.4-20.5)	.43
Hepatic	22.0 (21.2-22.9)	22.6 (21.8-23.5)	.37
Coagulation	22.3 (21.4-23.1)	23.1 (22.3-23.9)	.16
ICU-free days, No. (95% CI)	14.4 (13.5-15.3)	14.7 (13.8-15.6)	.67
60-d mortality, No. (%) [95% CI]	118 (23.2) [19.6-26.9]	109 (22.2) [18.5-25.8]	.77
Development of infections, No. (%) [95% CI]			
VAP	37 (7.3) [5.0-9.5]	33 (6.7) [4.5-8.9]	.72
Clostridium difficile colitis	15 (3.0) [1.5-4.4]	13 (2.6) [1.2-4.1]	.77
Bacteremia, No. (%)	59 (11.6) [8.8-14.4]	46 (9.3) [6.8-11.9]	.24

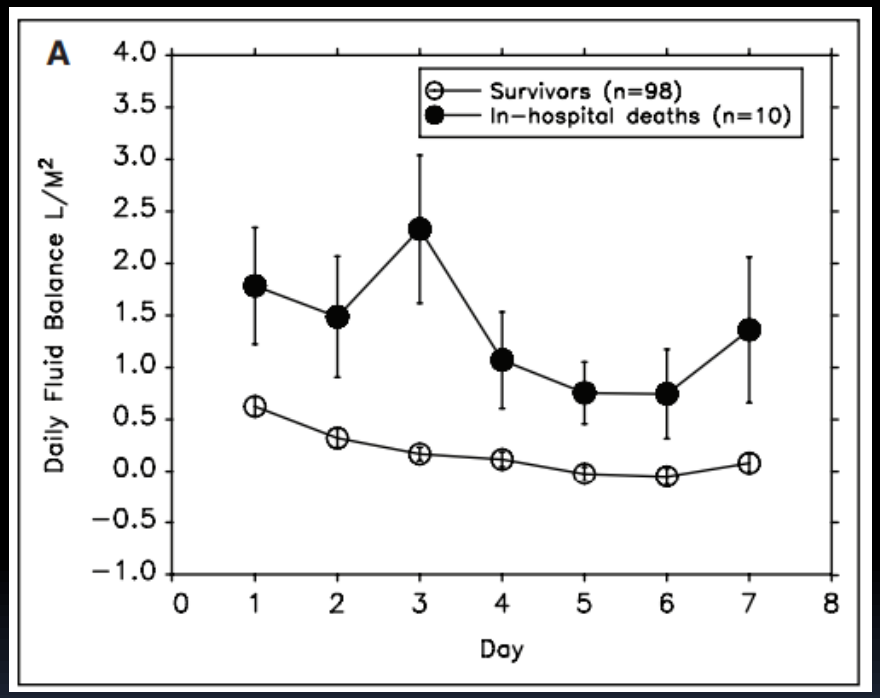
Conclusión En pacientes con lesión pulmonar aguda, en comparación con la alimentación enteral total, una estrategia inical de alimentación enteral trófica de hasta 6 días no mejoró los días sin ventilador, la mortalidad a 6o días o complicaciones infecciosas, pero se asoció con menos intolerancia gastrointestinal.

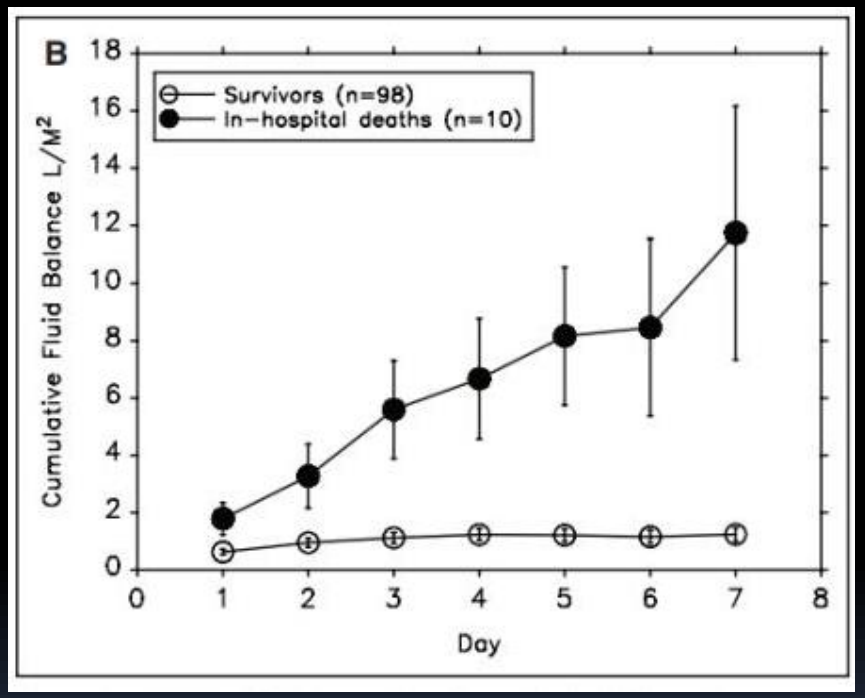
JAMA, February 22/29, 2012—Vol 307, No. 8

PALICC destaca la importancia de la nutrición, preferentemente enteral, en el mantenimiento del crecimiento, el cumplimiento de las necesidades metabólicas y la facilitación de la recuperación

The Relationship of Fluid Administration to Outcome in the Pediatric Calfactant in Acute Respiratory Distress Syndrome Trial*

Douglas F. Willson, MD[1]; Neal J. Thomas, MD, MSc[2]; Robert Tamburro, MD[2]; Edward Truemper, MD[3]; Jonathon Truwit, MD, MBA[4]; Mark Conaway, PhD[5]; Christine Traul, MD[1,6]; Edmund E. Egan, MD[7,8]; in collaboration with Pediatric Acute Lung and Sepsis Investigators Network



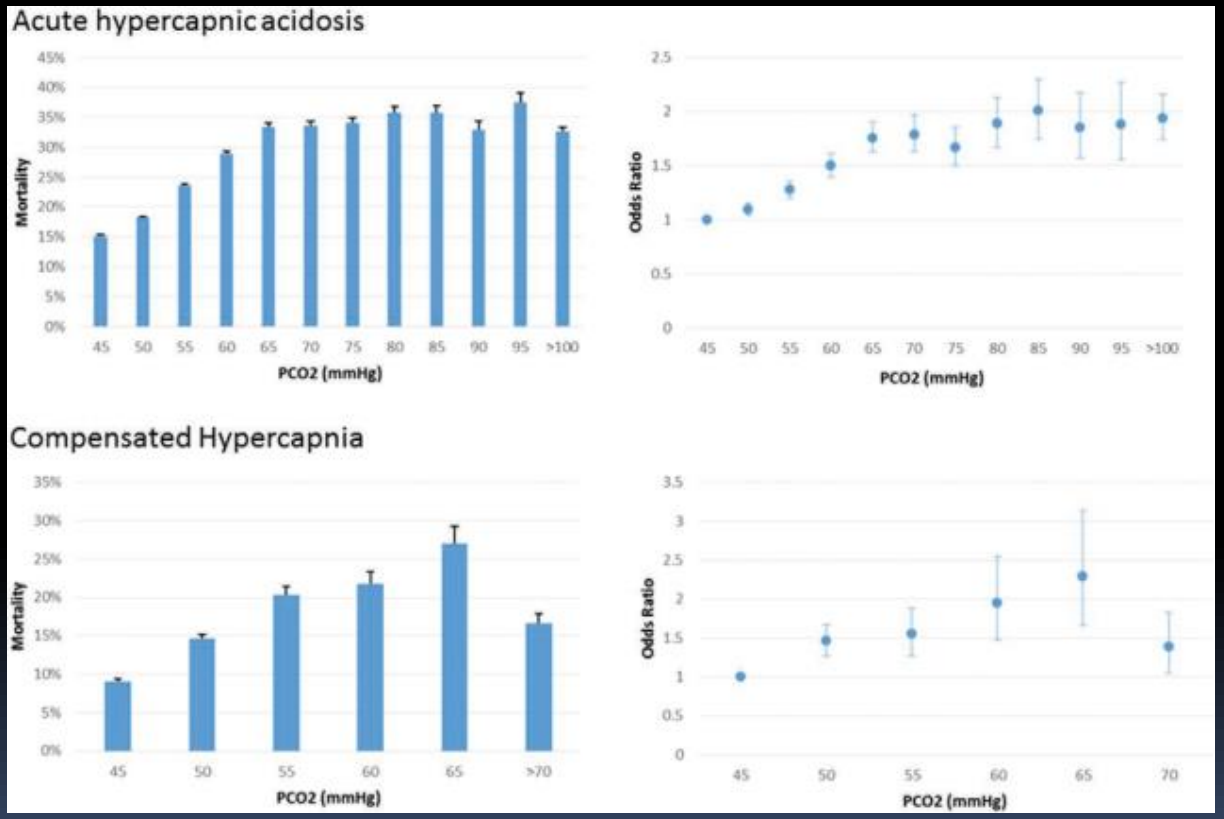


La acumulación de fluidos se asoció con el empeoramiento de la oxigenación, mayor días en ventilador y un aumento de la mortalidad. Un enfoque más conservador de la administración de líquidos puede mejorar los resultados en niños con lesión pulmonar aguda / síndrome de dificultad respiratoria aguda

(Pediatr Crit Care Med 2013; 14:666–672)

Effects of Hypercapnia and Hypercapnic Acidosis on Hospital Mortality in Mechanically Ventilated Patients

Ravindranath Tiruvoipati, FCICM[1–3]; David Pilcher, FCICM[3]; Hergen Buscher, FCICM[4,5]; John Botha, FCICM[1,2]; Michael Bailey, PhD[3]



CONCLUSIONES

La acidosis hipercápnica y la hipercapnia compensada fueron asociado con una mayor mortalidad hospitalaria, duración prolongada de la UCI, y la duración de la estadía en el hospital. El aumento de la mortalidad se observó incluso después de ajustar por la gravedad de la enfermedad, tipo de la UCI, y el año en que los pacientes fueron tratados. Un progresivo aumento en la mortalidad se observó con el aumento de Pco2 hasta 65 mm Hg después de lo cual la mortalidad se estabilizó.

Dado que diseño retrospectivo, no aleatorizado y controlado de nuestro estudio, estos resultados sugieren una asociación pero no causalidad. Futuro

estudios controlados pueden aclarar aún más los efectos de acidosis hipercápnica e hipercapnia compensada.

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The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MAY 24, 2018 VOL. 378 NO. 21

Extracorporeal Membrane Oxygenation for Severe Acute Respiratory Distress Syndrome

A. Combes, D. Hajage, G. Capellier, A. Demoule, S. Lavoué, C. Guervilly, D. Da Silva, L. Zafrani, P. Tirot, B. Veber, E. Maury, B. Levy, Y. Cohen, C. Richard, P. Kalfon, L. Bouadma, H. Mehdaoui, G. Beduneau, G. Lebreton, L. Brochard, N.D. Ferguson, E. Fan, A.S. Slutsky, D. Brodie, and A. Mercat, for the EOLIA Trial Group, REVA, and ECMONet*

EOLIA

La eficacia de la oxigenación por membrana extracorpórea venovenosa (OMEC) en pacientes con síndrome de dificultad respiratoria aguda grave (SDRA) sigue siendo controvertido.

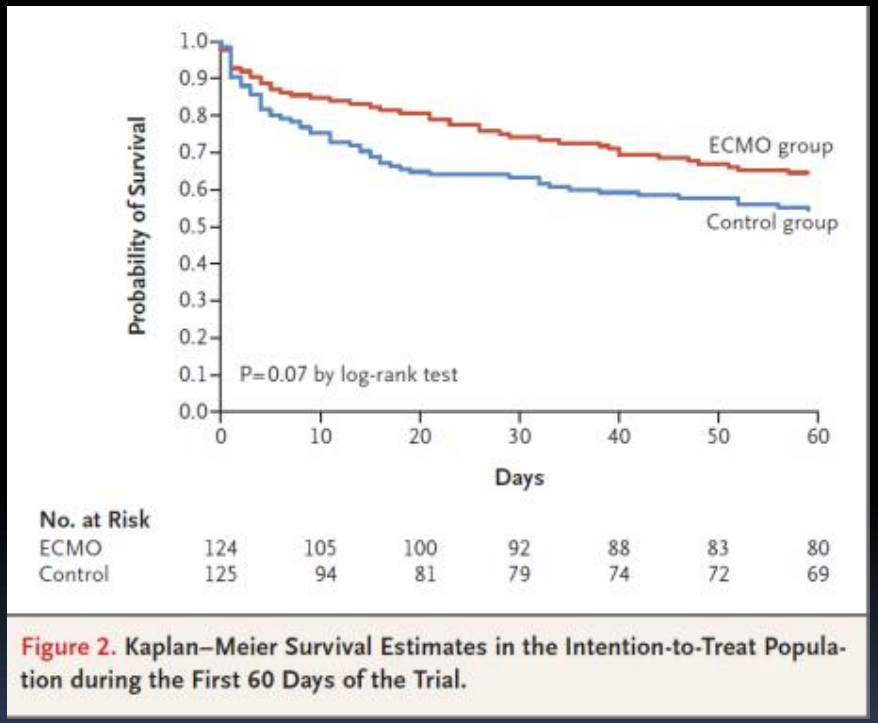


Figure 2. Kaplan–Meier Survival Estimates in the Intention-to-Treat Population during the First 60 Days of the Trial.

Entre los pacientes con SDRA muy grave, la mortalidad a los 60 días no fue significativa más bajo con ECMO que con una estrategia de ventilación mecánica convencional que incluía ECMO como terapia de rescate.

Quality of Life and Lung Function in Survivors of ECMO for Acute Respiratory Distress Syndrome (ARDS)

Prospective, observational, single-center study of 84 adults with ARDS

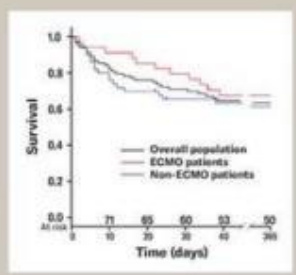
One-year follow-up of 34 extracorporeal membrane oxygenation (ECMO) and 50 non-ECMO patients:

- Pulmonary function tests
- Computed tomography of the chest
- Health-related quality-of-life questionnaires

ECMO patients had more severe respiratory failure at admission.

Patient Characteristic (Median [IQR])	ECMO (n = 34)	Non-ECMO (n = 50)	*P* Value
PaO_2/FIO_2, mmHg	72 [50-103]	114 [87-133]	0.001
Compliance, ml/cm H_2O	27 [19-37]	31 [26-37]	0.043

At 1 yr, survival was similar (22/33 ECMO vs. 28/47 non-ECMO, 66% vs. 59%; $P = 0.52$) and pulmonary function tests and CTs were almost normal in both groups.



Kaplan–Meier estimates of the unadjusted cumulative probability of survival.

In ARDS patients, ECMO and non–ECMO patients had similar survival and almost full recovery of lung function at 1 yr, despite ECMO patients having more severe respiratory failure at admission. Non–ECMO patients reported greater impairment of health-related quality of life.

Schulman, *et al.* ANESTHESIOLOGY. April 2019.

ANESTHESIOLOGY

Trusted Evidence: Discovery to Practice

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Usar ECMO da igual que No Usar ECMO en cuanto a supervivencia y función pulmonar a un año, este estudio refiere que el ECMO “podría” mejorar la calidad de vida en los pacientes que lo usaron pero No tiene significancia estadística, lo que Sí puede mejorar la Calidad de Vida es la Rehabilitación temprana

P-PREP

Pediatric Pulmonary Rescue with Extracorporeal Membrane Oxygenation Score

*** Tool developed and validated using the Extracorporeal Life Support Organization Registry data from 2001-2013**

For more information, see: Development and Validation of a Score to Predict Mortality in Children Undergoing Extracorporeal Membrane Oxygenation for Respiratory Failure: Pediatric Pulmonary Rescue With Extracorporeal Membrane Oxygenation Prediction Score

ECMO Mode

- OFF Veno-venous
- OFF Venoarterial

Mechanical ventilation > 14 days: No

PaO2/FiO2

- OFF > 300 mmHg
- OFF 201-300 mmHg
- OFF 101-200 mmHg
- OFF < = 100 mmHg

pH

- OFF >7.34
- OFF 7.11-7.34
- OFF <7.11

Primary pulmonary diagnosis

- OFF Asthma
- OFF Aspiration
- OFF Respiratory syncytial virus
- OFF Sepsis-induced ARDS[1]
- OFF Pertussis
- OFF Other

Comorbid conditions

- OFF Pre-ECMO cardiac arrest
- OFF Cancer
- OFF Acute renal failure
- OFF Acute liver necrosis

[1] Primary diagnosis is sepsis with no additional pulmonary infections

Your Score	Risk of in-hospital mortality at ECMO initiation for pediatric respiratory failure
Reset	

*Shaded band represents the 95% CI of the development data set

DISASSOCIATING LUNG MECHANICS AND OXYGENATION IN PEDIATRIC ACUTE RESPIRATORY DISTRESS SYNDROME

Nadir Yehya, MD[1] and **Neal J. Thomas, MD, MSc**[2]

Mejoría en la oxigenación, pero no en la mecánica respiratoria, se asoció con una menor mortalidad en SDRA pediátrico. Los ensayos futuros de ventilación en niños deberían centrarse en la respuesta a la oxigenación (mayor PaO2 / FIO2) en lugar de una PIP o ΔP más bajas, ya que la oxigenación se asoció de manera más consistente con el resultado y, por lo tanto, es una estrategia más apropiada. La relevancia de VILI para los resultados pediátricos de SDRA requiere un estudio más a fondo, ya que las conclusiones de SDRA en adultos extrapolan mal a los niños.

Crit Care Med. 2017 July ; 45(7): 1232–1239

GUIDELINES ON THE MANAGEMENT OF ACUTE RESPIRATORY DISTRESS SYNDROME

Topic	GRADE Recommendation	Conditions
Tidal Volume	Strongly in favour	Tidal volume ≤ 6 ml/Kg ideal body weight; Plateau pressure < 30cmH_2O
Prone Positioning	Strongly in favour	Proning for ≥ 12 hours per day Patients with moderate/severe ARDS (P:F ratio ≤ 20kPa)
High frequency oscillation (HFOV)	Strongly against	
Conservative Fluid Management	Weakly in favour	
Higher Peek End-Expiratory Pressure (PEEP)	Weakly in favour	Patients with moderate or severe ARDS (PF ratio ≤ 27kPa)
Neuromuscular Blocking Agents (NMBA)	Weakly in favour	Evidence for cisatracurium besylate Continuous 48-hour infusion Patients with moderate/severe ARDS (≤ 20kPa)
Extra-Corporeal Membrane Oxygenation (ECMO)	Weakly in favour	With lung-protective mechanical ventilation Patients with severe ARDS, lung injury score ≥3 or pH <7.20 due to uncompensated hypercapnoea
Inhaled Vasodilators	Weakly against	Evidence for inhaled nitric oxide
Corticosteroids	Research recommendation	
Extra-Corporeal Carbon Dioxide Removal (ECCO2R)	Research recommendation	

The Faculty of Intensive Care Medicine

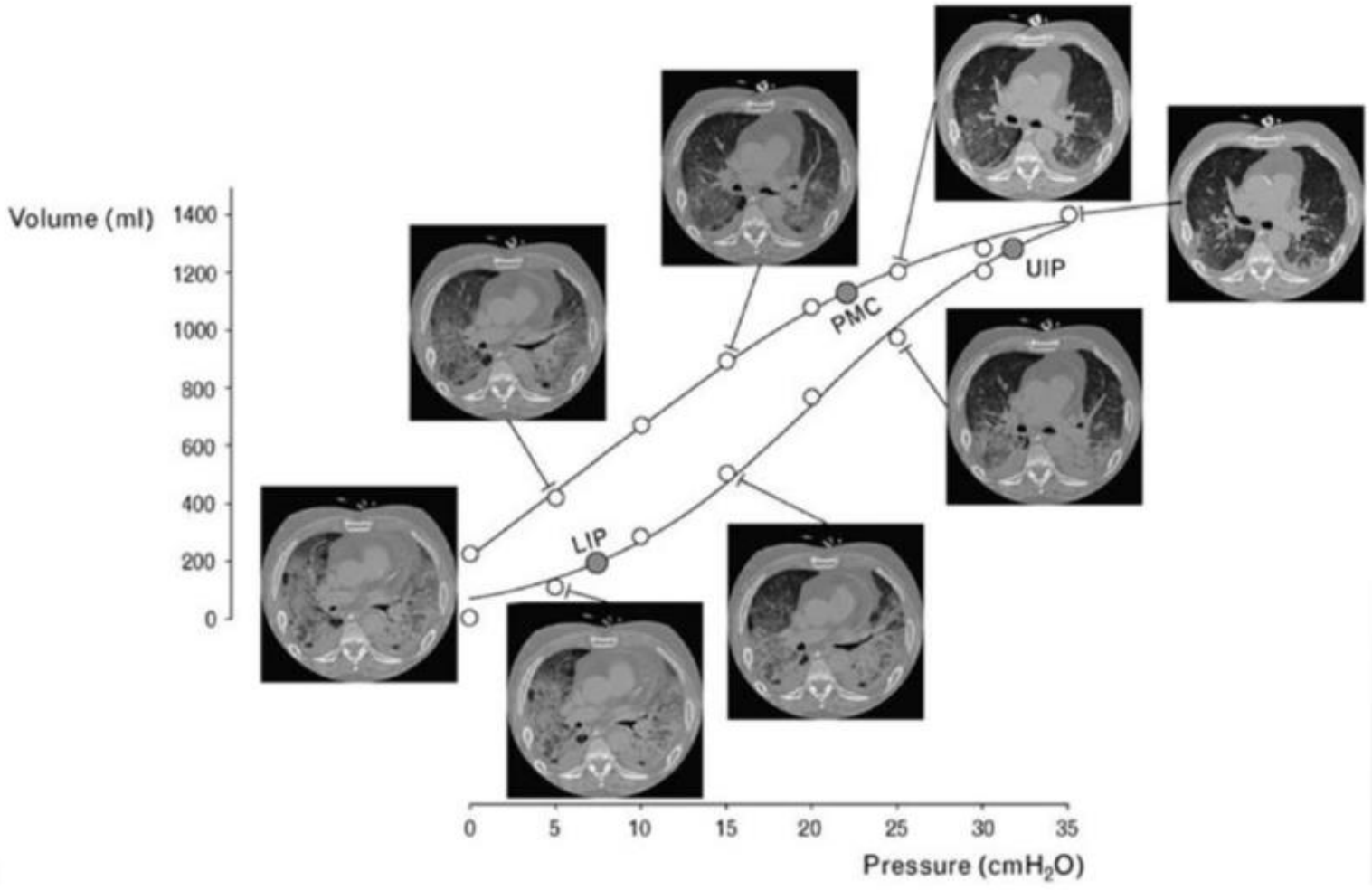
intensive care society
care when it matters

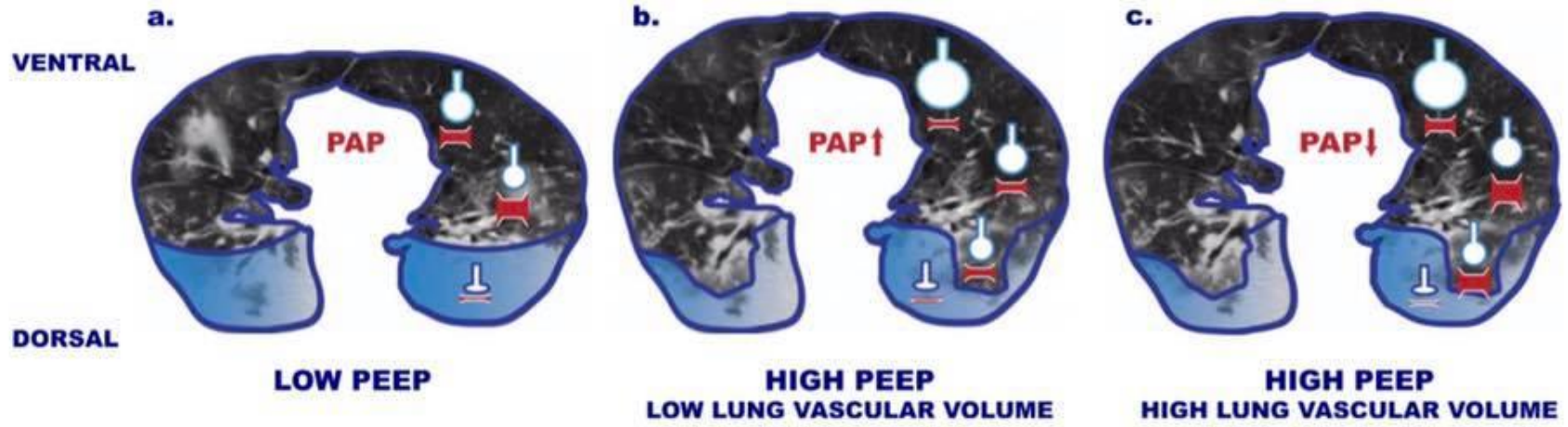
Julio 2018

REGLAS DE ORO ARDS:

PEEP “IDEAL” NO EXISTE

- PEEP is a way to regain FRC



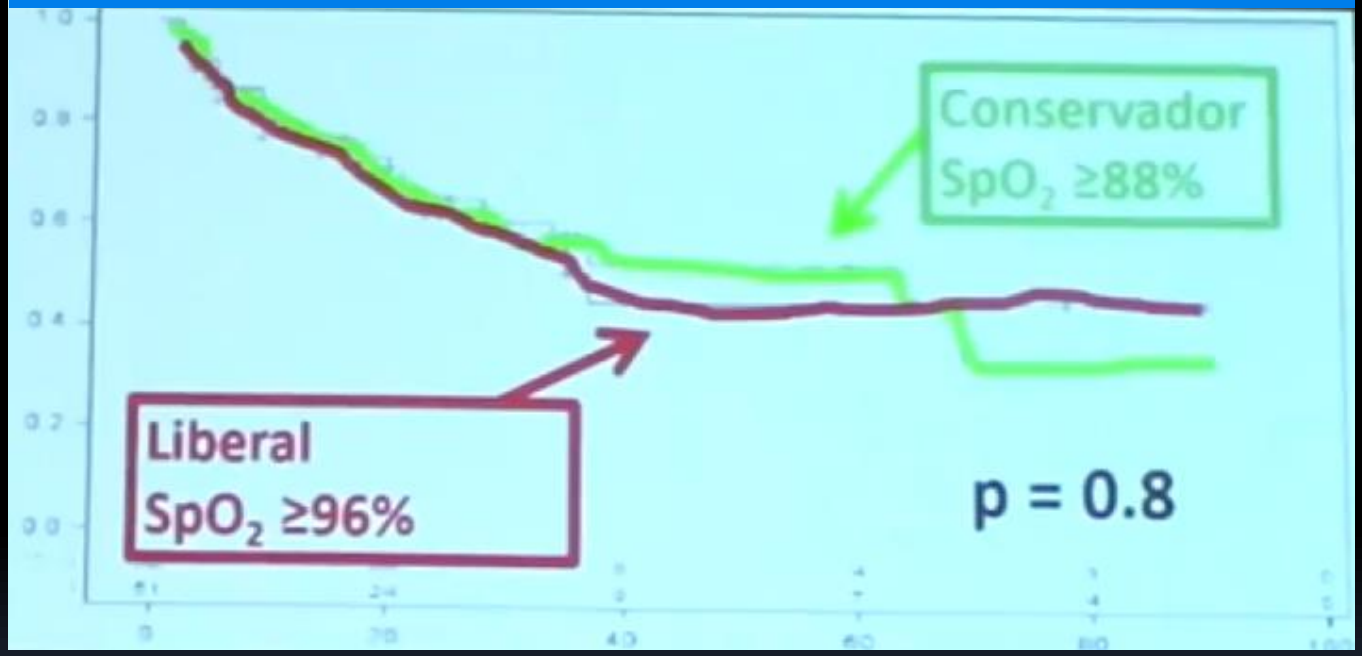


Izq. PEEP bajo, colapso alveolar y atelectasias, capilar ocluido dentro de la ocupación alveolar, capilares abiertos en el resto del pulmón. Buena perfusion, baja ventilación, vasoconstricción hipoxemica, poco o nulo daño al VD.
Medio. PEEP alto con poco volumen vascular induce Sobredistensión alveolar apical, Colapso Capilar (vascular y linfático), incrementa la presión arterial pulmonar y comienza el daño al VD por aumento de la resistencia vascular pulmonar.
Der. PEEP alto con alto volumen vascular. Induce sobredistension alveolar sin colapso capilar, la presión arterial pulmonar baja por disminución de la vasoconstricción hipoxemica, el colapso alveolar basal disminuye y mejora la ventilación/perfusión.

Cruz FF, et al. Expert Rev Respir Med. 2018.

REGLAS DE ORO ARDS:

LA META NO ES LA MEJOR OXIGENACION



American Journal of Respiratory & Critical Care Medicine 2015

Metrics of Arterial Hyperoxia and Associated Outcomes in Critical Care*

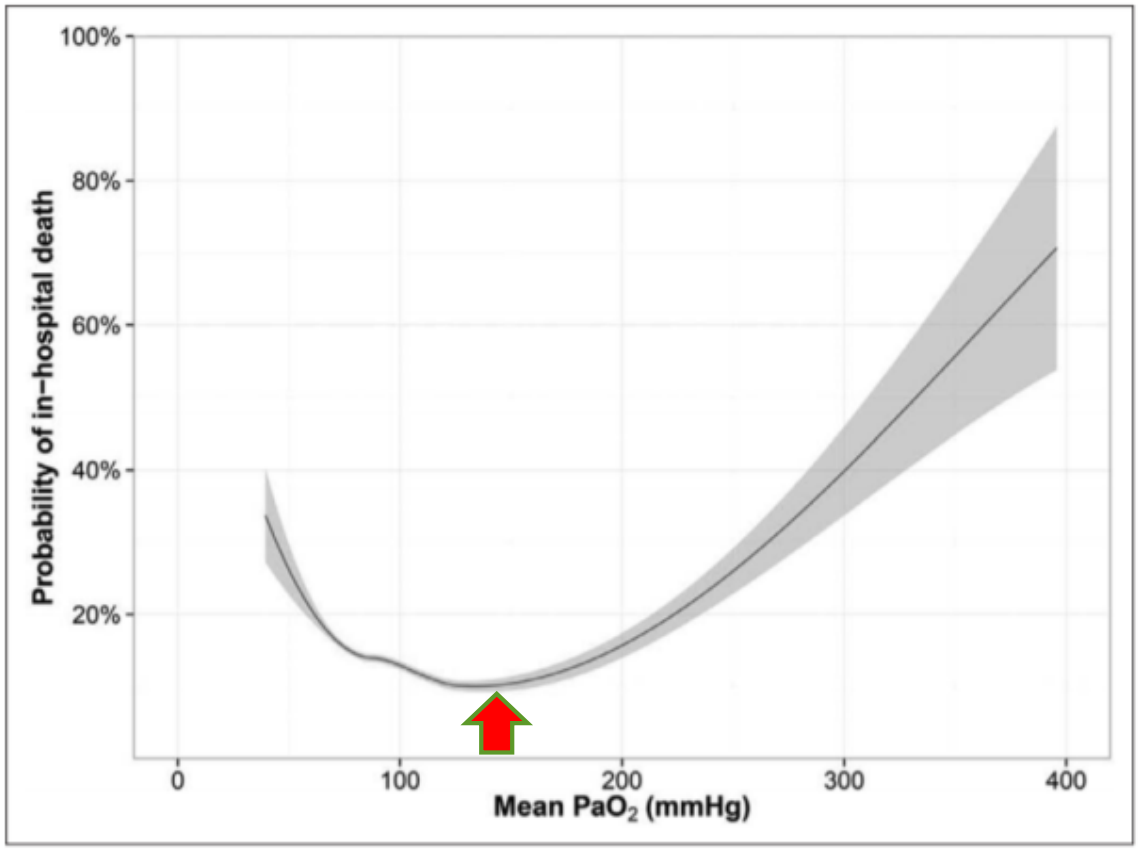


Figure 1. Adjusted probability of in-hospital death by mean Pao_2. Loess smoothing curve predicted from logistic regression model adjusted for age, Acute Physiology and Chronic Health Evaluation IV score, and ICU length of stay (LOS). ***Solid line*** represents oxygenation by mean Pao_2 over the total ICU LOS. ***Gray zones*** represent 95% CIs.

Crit Care Med 2017; 45:187–195

REGLAS DE ORO ARDS:

Volúmenes corrientes bajos: 6ml/Kg "Peso Ideal

Por cada mililitro que se ventila por arriba de 6ml/Kg
La mortalidad se incrementa exponencialmente.

American Journal of Respiratory & Critical Care Medicine 2015;191:171-185

Variability in Pediatric Ideal Body Weight Calculation: Implications for Lung-Protective Mechanical Ventilation Strategies in Pediatric Acute Respiratory Distress Syndrome*

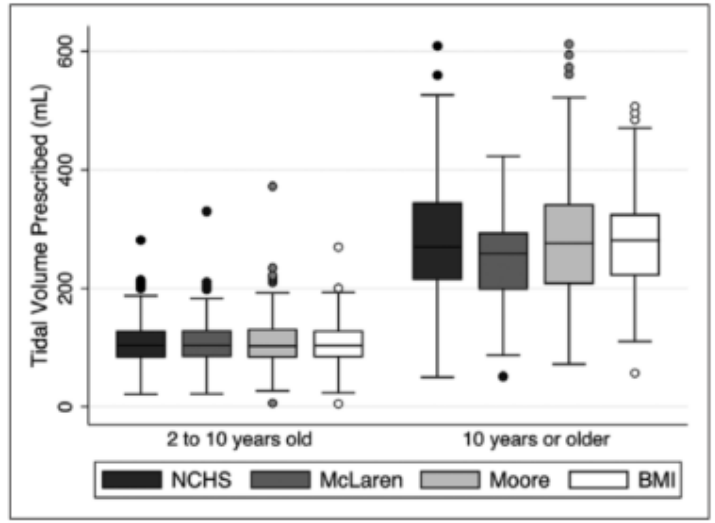


Figure 4. Tidal volumes calculated by the four ideal body weight (IBW) methods for children 2–10 yr old and 10 yr old or older when a 6 mL/kg IBW tidal volume is prescribed. $p < 0.001$ for mean difference between McLaren and National Center for Health Statistics (NCHS) and McLaren and body mass index (BMI) methods in the 10 yr old or older group.

Hasta que se logre un consenso más amplio, recomendamos el uso del método de McLaren para calcular el IBW en niños con PARDS tanto para la evaluación del estado nutricional como para la prescripción de una estrategia de ventilación mecánica protectora pulmonar con volumen corriente bajo.

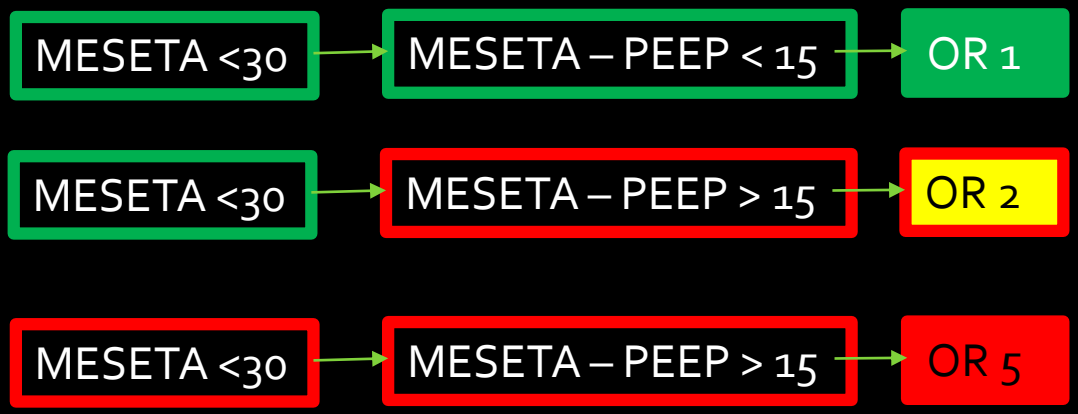
REGLAS DE ORO:

Presión meseta < 30 cmH20

Critical Care Medicine. 2015;42:2278-2289

Meseta – PEEP <15 cm H20

N Engl J Med 2015;372:747-55



Critical Care Medicine 2017: 45:843-850

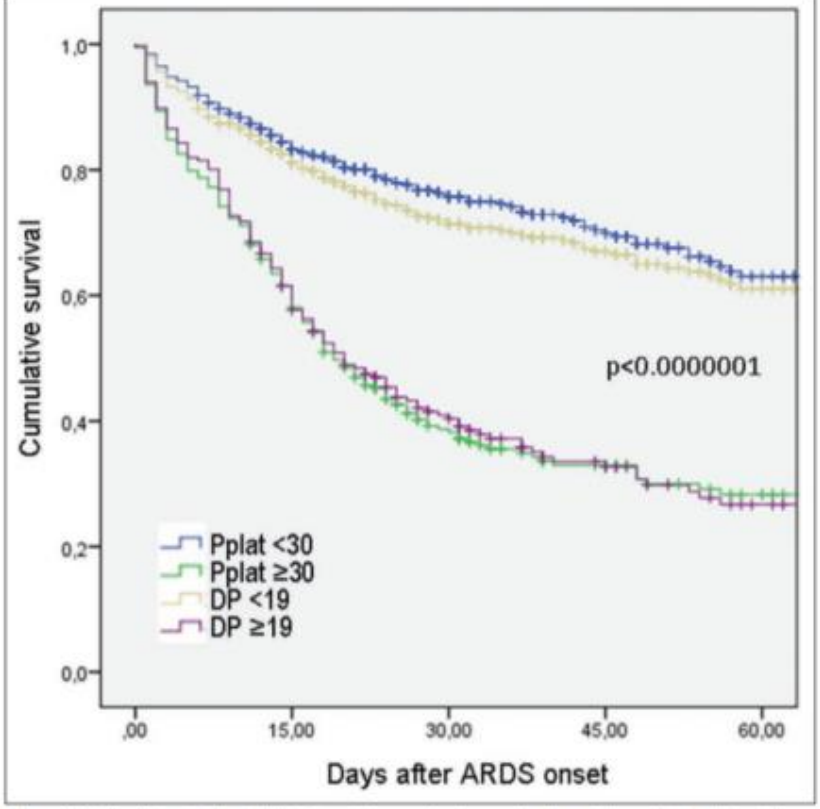


Figure 4. Kaplan-Meier 60-d probability of survival curves for the combined population of 778 patients with acute respiratory distress syndrome (ARDS). Patients are stratified in four subgroups according to individual values of plateau pressure (Pplat) and driving pressure (DP) at 24 hr after ARDS diagnosis (Pplat < 30 cm H_2O, Pplat ≥ 30 cm H_2O, DP < 19 cm H_2O, and DP ≥ 19 cm H_2O). Most deaths occurred within the first 20 d of inclusion into the study.

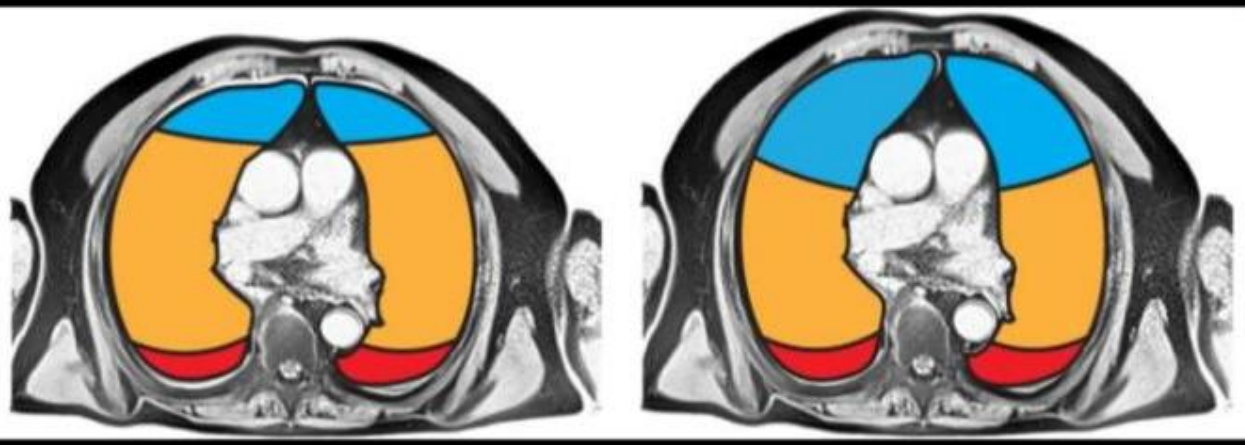
Critical Care Medicine

Close the lungs and keep them rested !

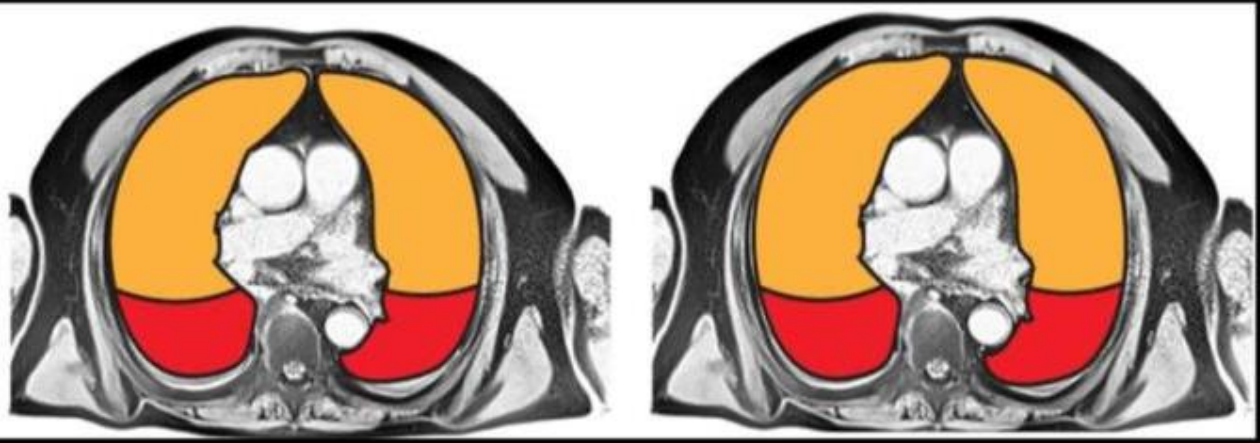
Pelosi P, Rocco PRM, de Abreu MG Crit Care. 2018 Mar 20;22(1):72

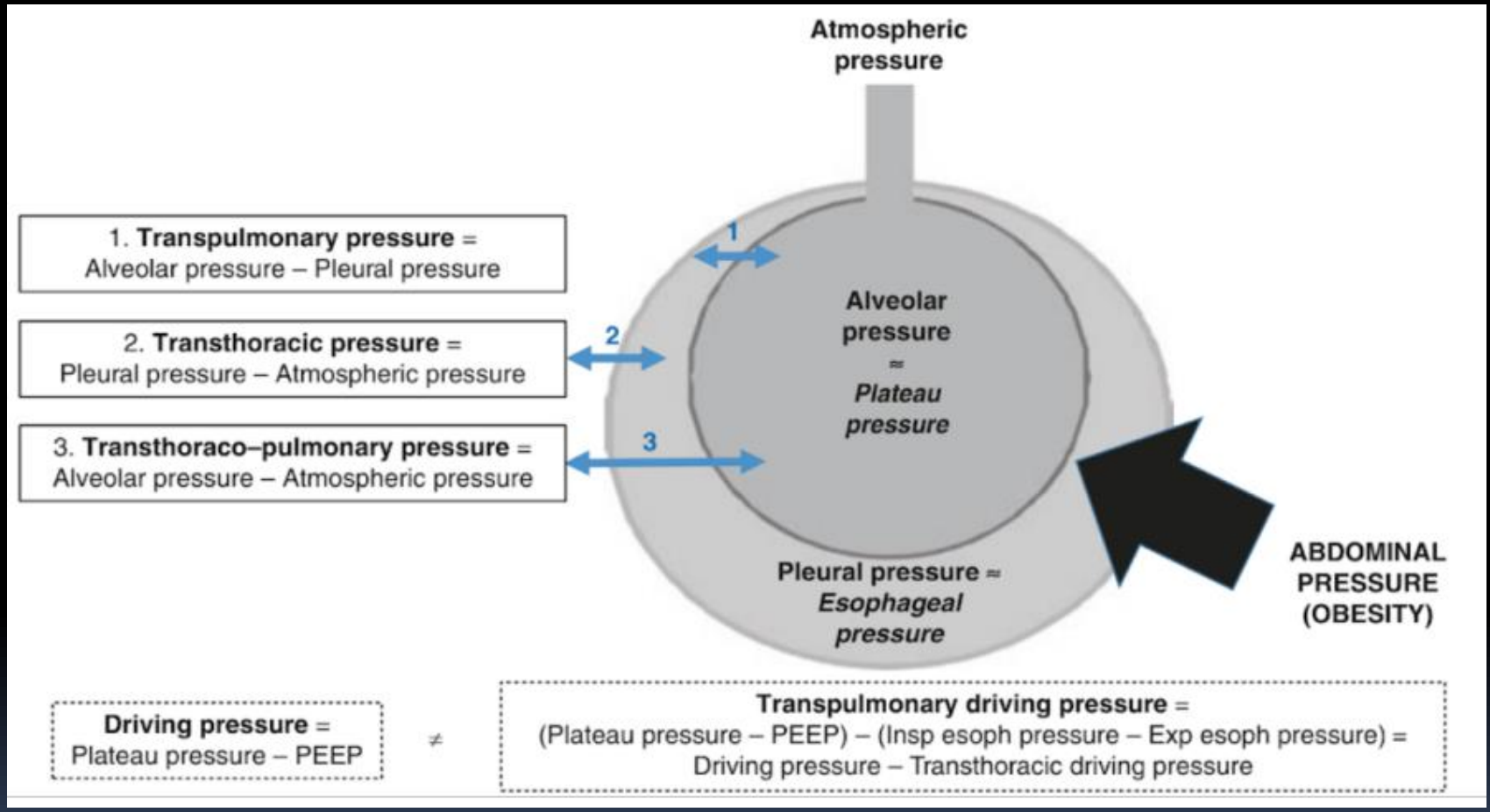
Expiration Inspiration

LOW V_T
HIGH P_{Plat}
HIGH PEEP
LOW-MODERATE DP



LOW V_T
LOW P_{Plat}
LOW PEEP
LOW DP




1. **Transpulmonary pressure** = Alveolar pressure – Pleural pressure
2. **Transthoracic pressure** = Pleural pressure – Atmospheric pressure
3. **Transthoraco–pulmonary pressure** = Alveolar pressure – Atmospheric pressure
Atmospheric pressure
1
2
3
Alveolar pressure ≈ *Plateau pressure*
Pleural pressure ≈ *Esophageal pressure*
ABDOMINAL PRESSURE (OBESITY)
Driving pressure = Plateau pressure – PEEP
≠
Transpulmonary driving pressure = (Plateau pressure – PEEP) – (Insp esoph pressure – Exp esoph pressure) = Driving pressure – Transthoracic driving pressure

Marcelo Amato

>7.5 ml/Kg

MESETA – PEEP < 15 cmH20
Meseta < 27 cm H20

346 pacientes
Validado 2360

N Engl J Med 2015;372:747-55

Jesús Villar

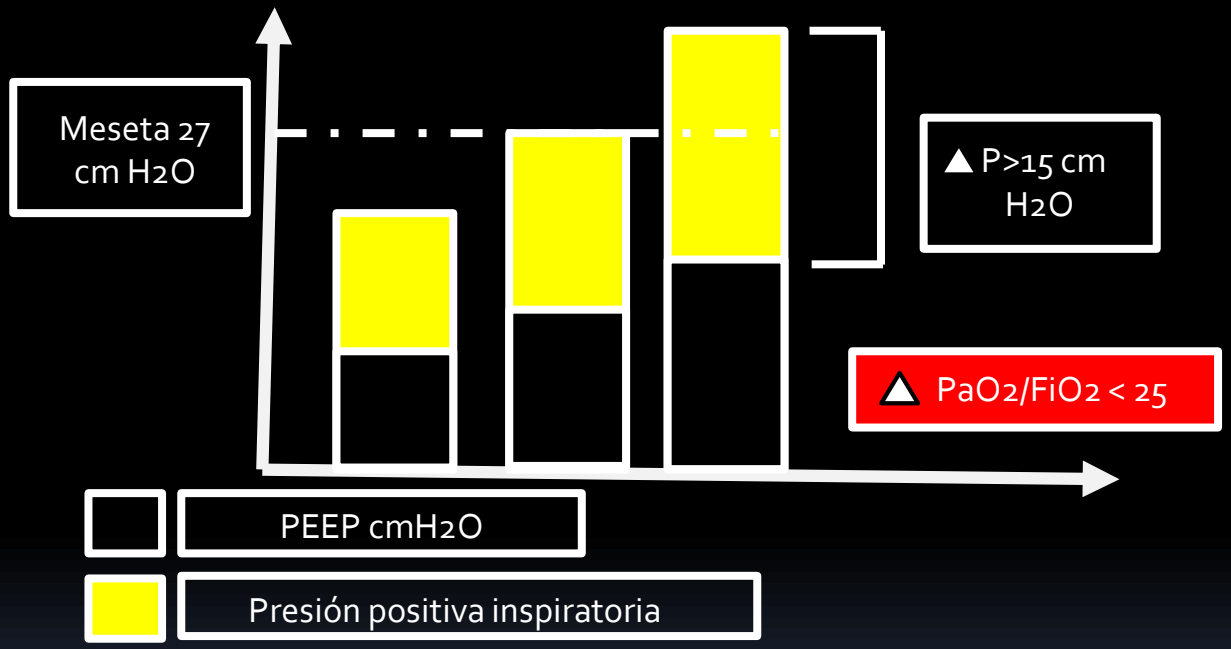
<7.5 ml/Kg

MESETA – PEEP < 18 cmH20
Meseta < 30 cm H20

478 pacientes
Validado 300

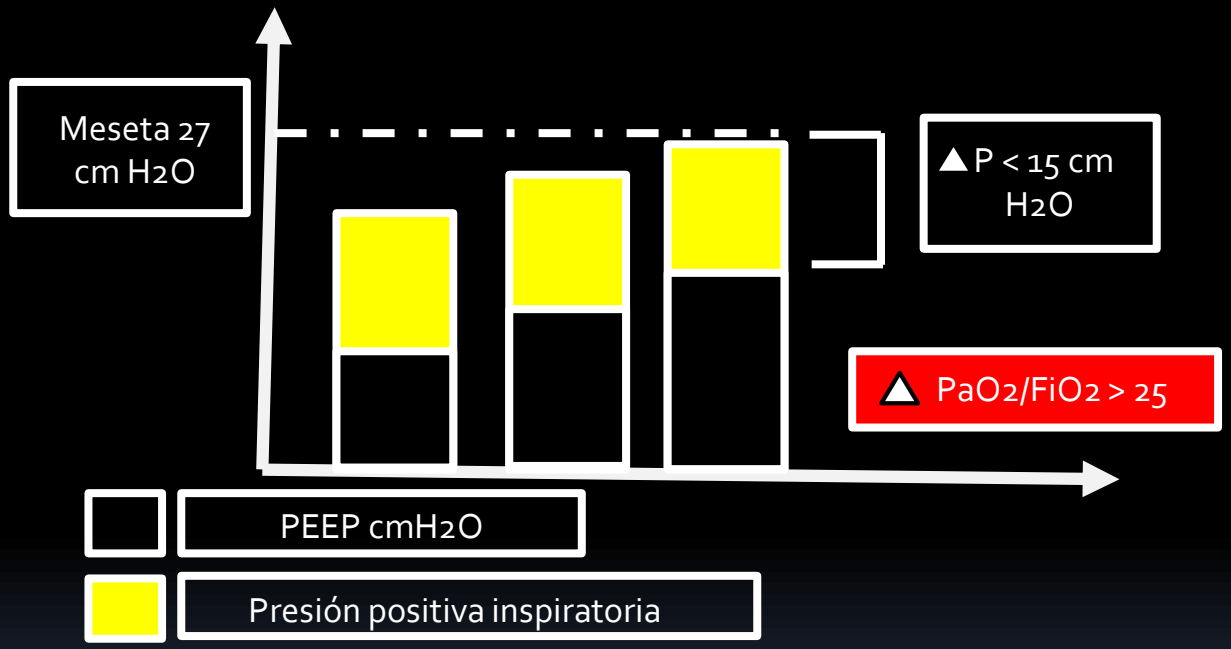
Crit Care Med 2017; 45:843-850

Grupo de mayor mortalidad



N Engl J Med 2015;372:747-55

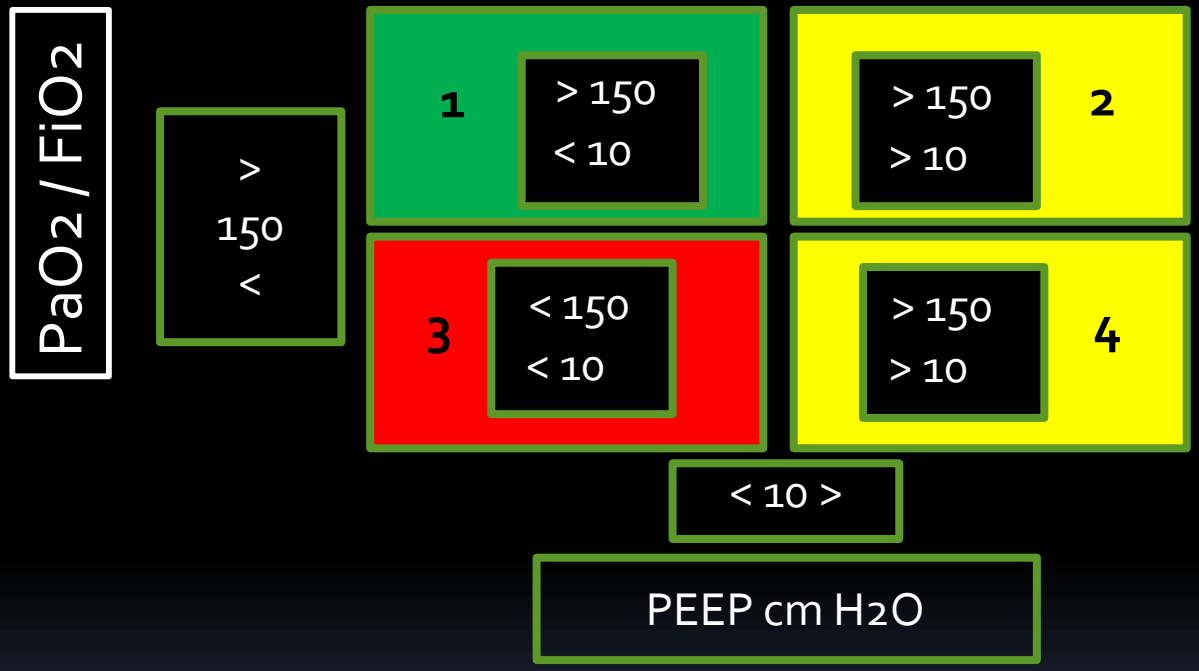
Unico grupo que se beneficia de aumento de PEEP



Am J Respir Crit Care Med 2015;15;192:1416-9

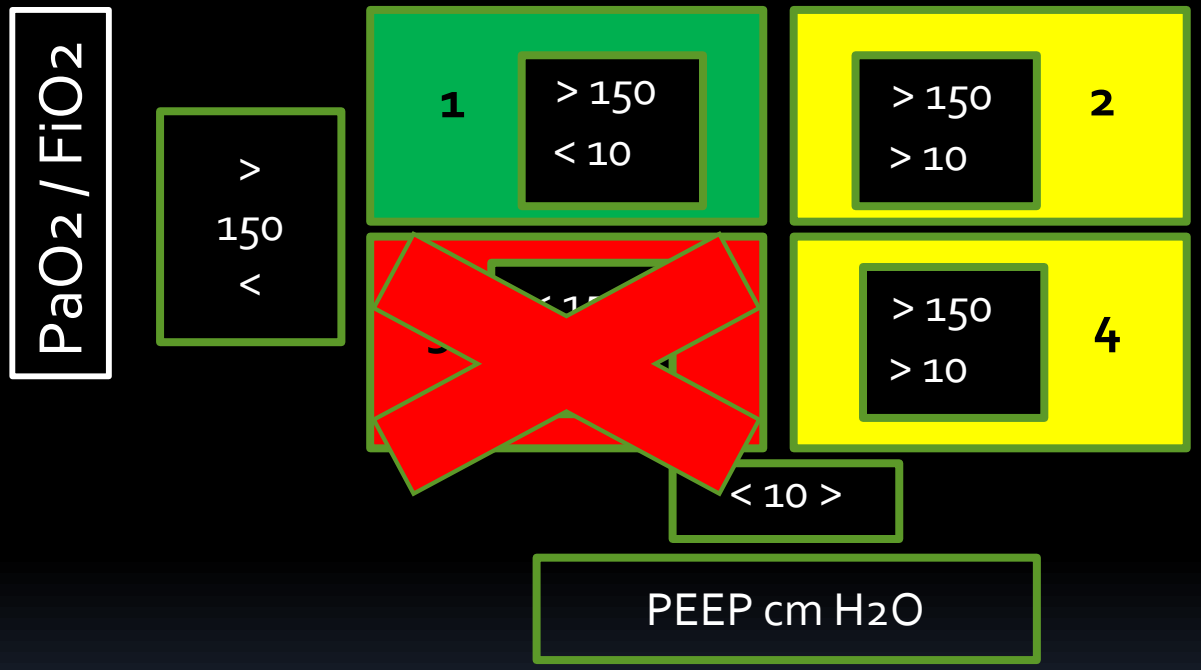
N Engl J Med 2015;372:747-55

A clinical classification of the acute respiratory distress syndrome for predicting outcome and guiding medical therapy*



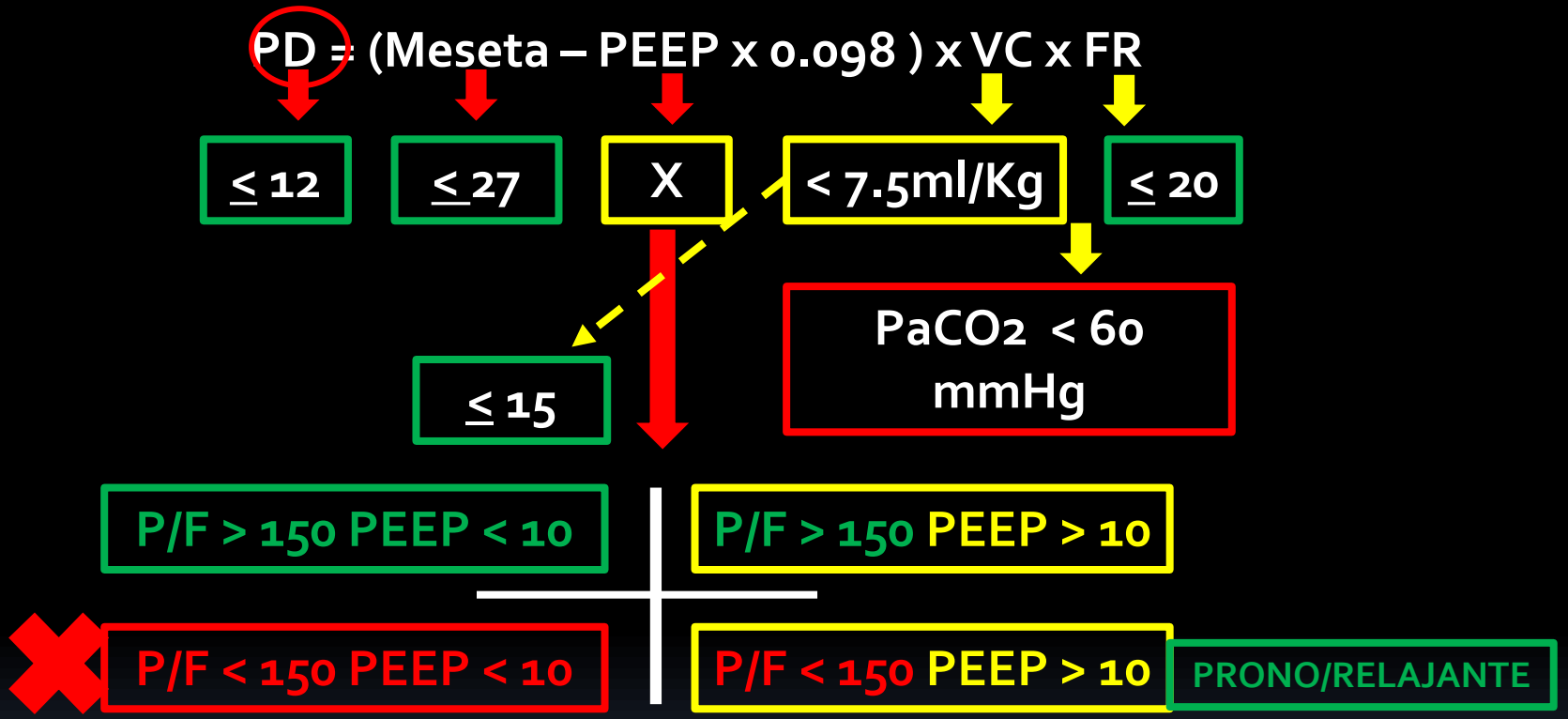
Crit Care Med 2015; 43:346-35

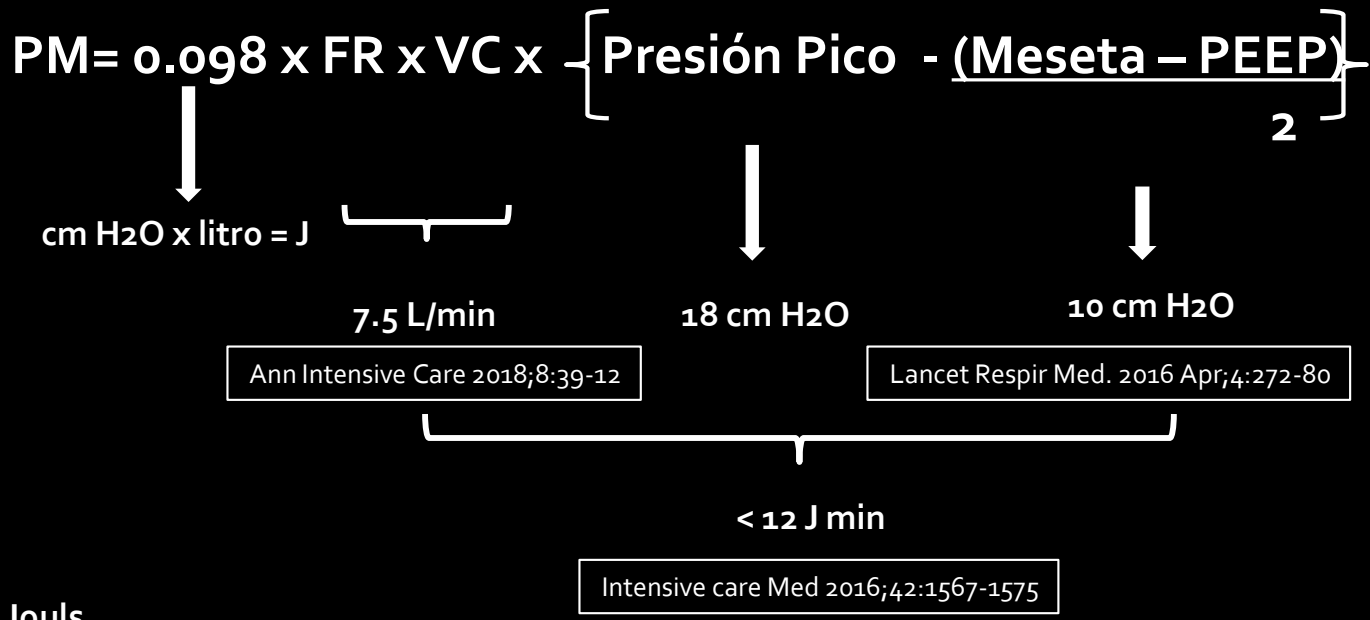
A clinical classification of the acute respiratory distress syndrome for predicting outcome and guiding medical therapy*



El grupo 3: PO2 /FiO2 < 150 con PEEP < 10 cm H2O no debe existir

Crit Care Med 2015; 43:346-35


PD = (Meseta – PEEP x 0.098) x VC x FR
≤ 12
≤ 27
X
< 7.5ml/Kg
≤ 20
≤ 15
PaCO2 < 60 mmHg
P/F > 150 PEEP < 10
P/F > 150 PEEP > 10
P/F < 150 PEEP < 10
P/F < 150 PEEP > 10
PRONO/RELAJANTE



J = Jouls
PM = Poder mecánico
FR = frecuencia respiratoria
VC = volumen corriente

$$PM = 0.098 \times FR \times VC \times \left[\text{Presión Pico} - \frac{(\text{Meseta} - \text{PEEP})}{2} \right]$$

ARDS 2002	ARDS 2016	
PODER 31 J/min MORTALIDAD 53 %	PODER 23 J/min MORTALIDAD 35%	PODER 21 J/min MORTALIDAD 29%

JAMA 2002;287:345-355

JAMA 2016;3158:788-800

Intensive care Med 2018;44:76-78

$$PM = 0.098 \times FR \times VC \times \left\{ \text{Presión Pico} - \frac{(\text{Meseta} - \text{PEEP})}{2} \right\}$$

Aumento exponencial de mortalidad por aumento en PM por arriba de 12 Joules / min

>5 Joules / min

12	17	22	27
basal	20%	29%	35%

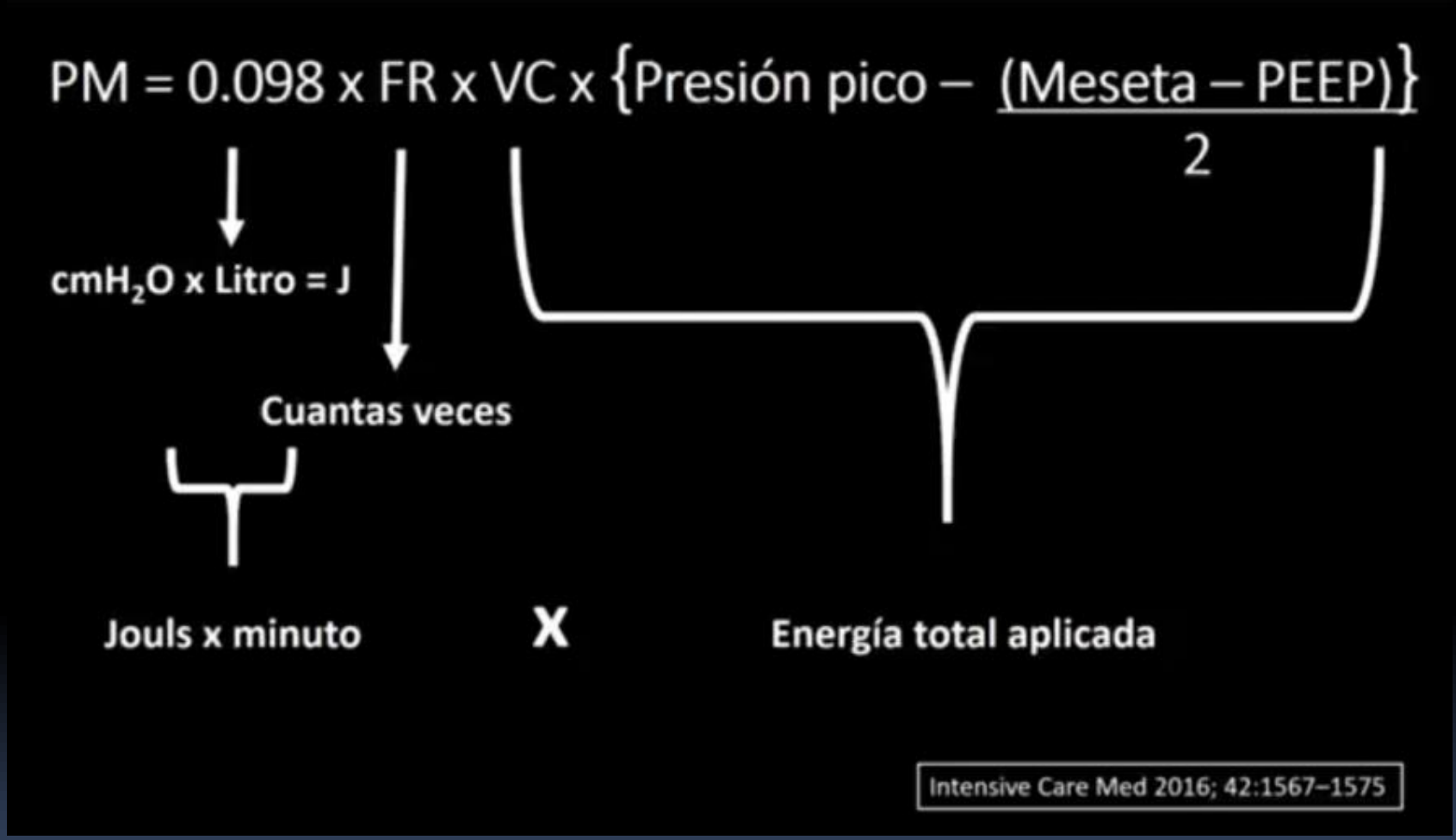
JAMA 2002;287:345-355

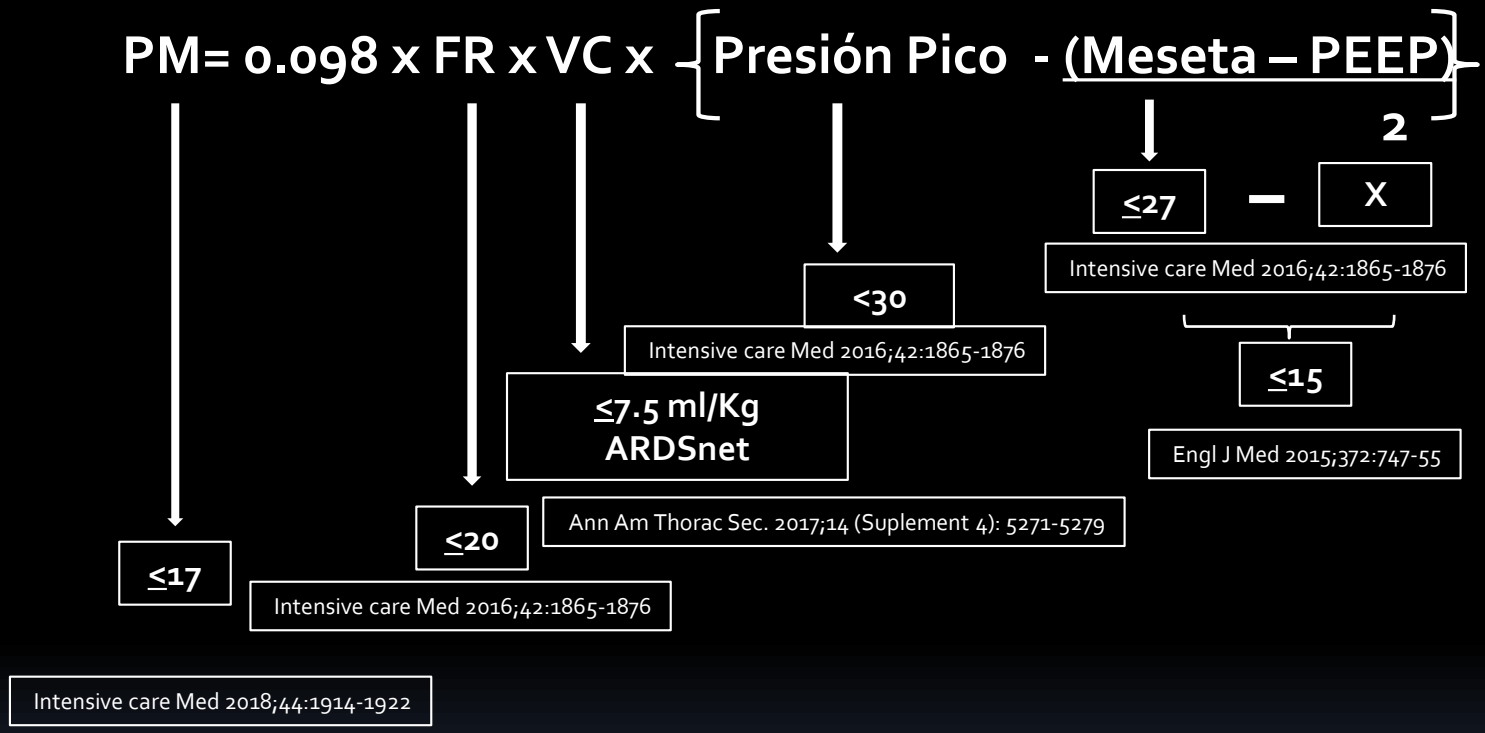
JAMA 2016;3158:788-800

J = Joules
PM = Poder mecánico
FR = frecuencia respiratoria
VC = volumen corriente

P = 0.001

Intensive care Med 2018;44:1914-1922


PM = 0.098 x FR x VC x {Presión pico – (Meseta – PEEP)/2}
cmH2O x Litro = J
Cuantas veces
Jouls x minuto
X
Energía total aplicada
Intensive Care Med 2016; 42:1567–1575



J = Jouls
PM = Poder mecánico
FR = frecuencia respiratoria
VC = volumen corriente

$$PM = 0.098 \times FR \times VC \times \left\{ \text{Presión pico} - \frac{(\text{Meseta} - \text{PEEP})}{2} \right\}$$

ARDS Berlín 1	ARDS Berlín 2	ARDS Berlín 3
21 J/min	23 J/min	29 J/min

J = Jouls
PM = Poder mecánico
FR = frecuencia respiratoria
VC = volumen corriente

Intensive Care Med 2018;44:76–78

$$PM = 0.098 \times FR \times VC \times \left\{ \text{Presión pico} - \frac{(\text{Meseta} - \text{PEEP})}{2} \right\}$$

¿LA CLASIFICACION CORRECTA DE LA LESION PULMONAR?

MODERADA	GRAVE	ARDS
P/F>150	P/F<150	P/F<150
< 21 J/min	< 21 J/min	≥ 24 J/min

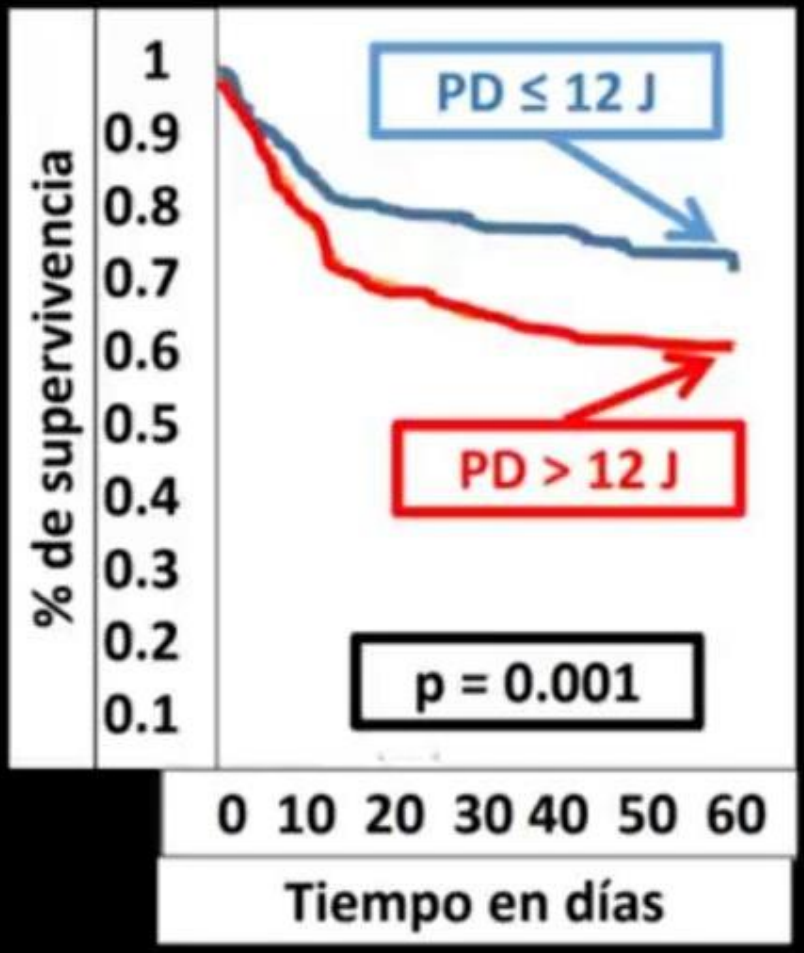
Am J Resp & Crit Care Med 2018 On line first

$$PM = 0.098 \times FR \times VC \times \left\{\text{Presión pico} - \frac{(\text{Meseta} - \text{PEEP})}{2}\right\}$$

SIN DESARROLLO DE LESION PULMONAR
P/F>300
12 J/min

CON DESARROLLO DE LESION PULMONAR
P/F>300
>17 J/min

Shock 2018 On line first



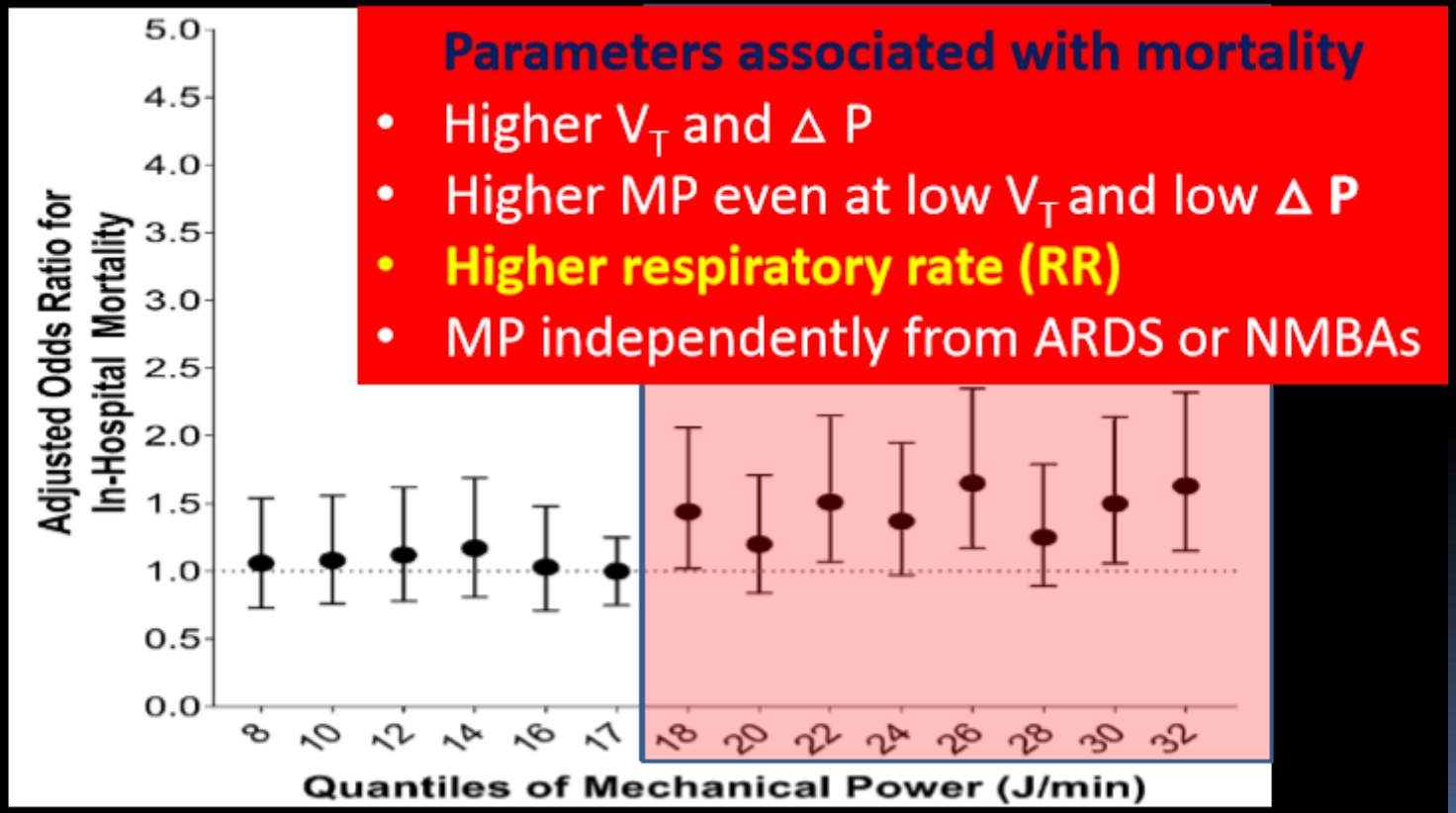
PD = 0.098 x FR x VC x (Meseta – PEEP)

Critical Care 2016;20:384

Mechanical power is associated with mortality in critically ill patients

Serpa-Neto A et al Intensive Care Med. 2018 Nov;44(11):1914-1922

Increased risk of death with MP > 17 J/min



Pelosi P for the PROVE Network (www.provenet.eu)

EFICIENCIA VENTILATORIA

$$EV = \frac{PaCO_2 \times \text{Volumen minuto actual}}{\text{Peso ARDSnet} \times 100 \times 37.52a}$$

>2

Am J Resp Crit Care 2019

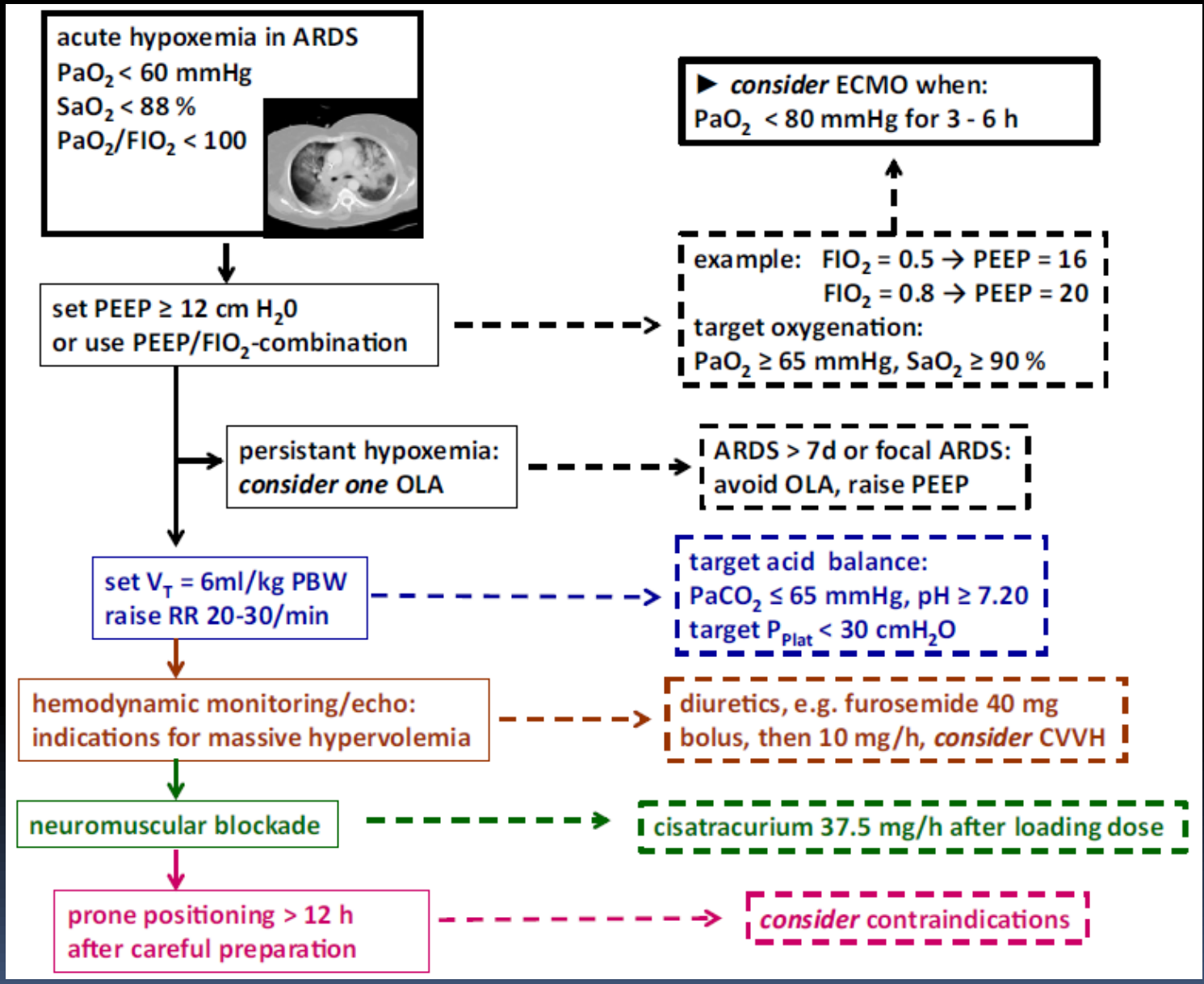
Hemodinamia y PEEP

Delta $PvCO_2 - PaCO_2 \leq 6$

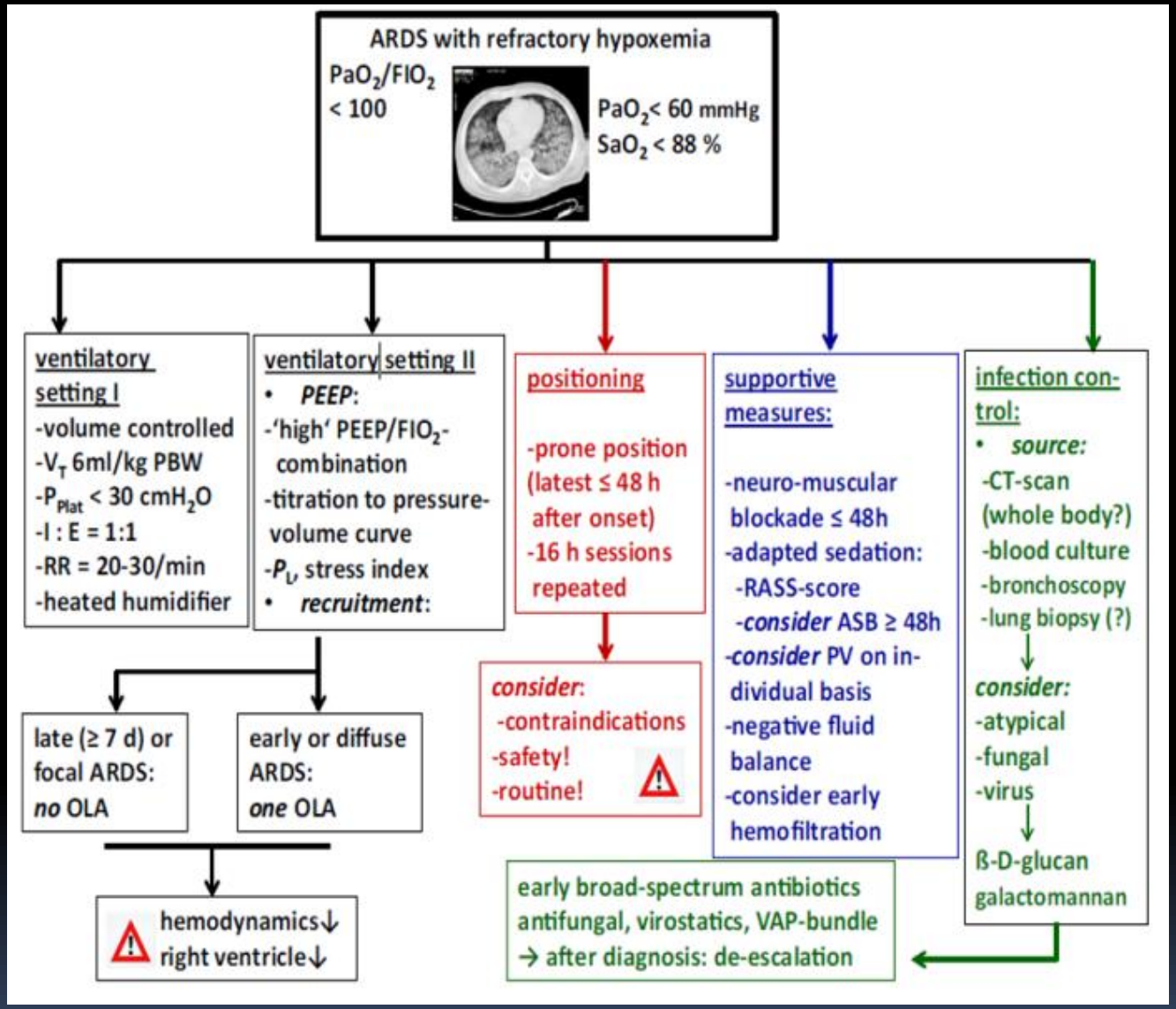
J Appl Physiol 2000, 89:1317-1321

PVC > 8 mmHg <12

Crit Care Med 2004;32:250-255



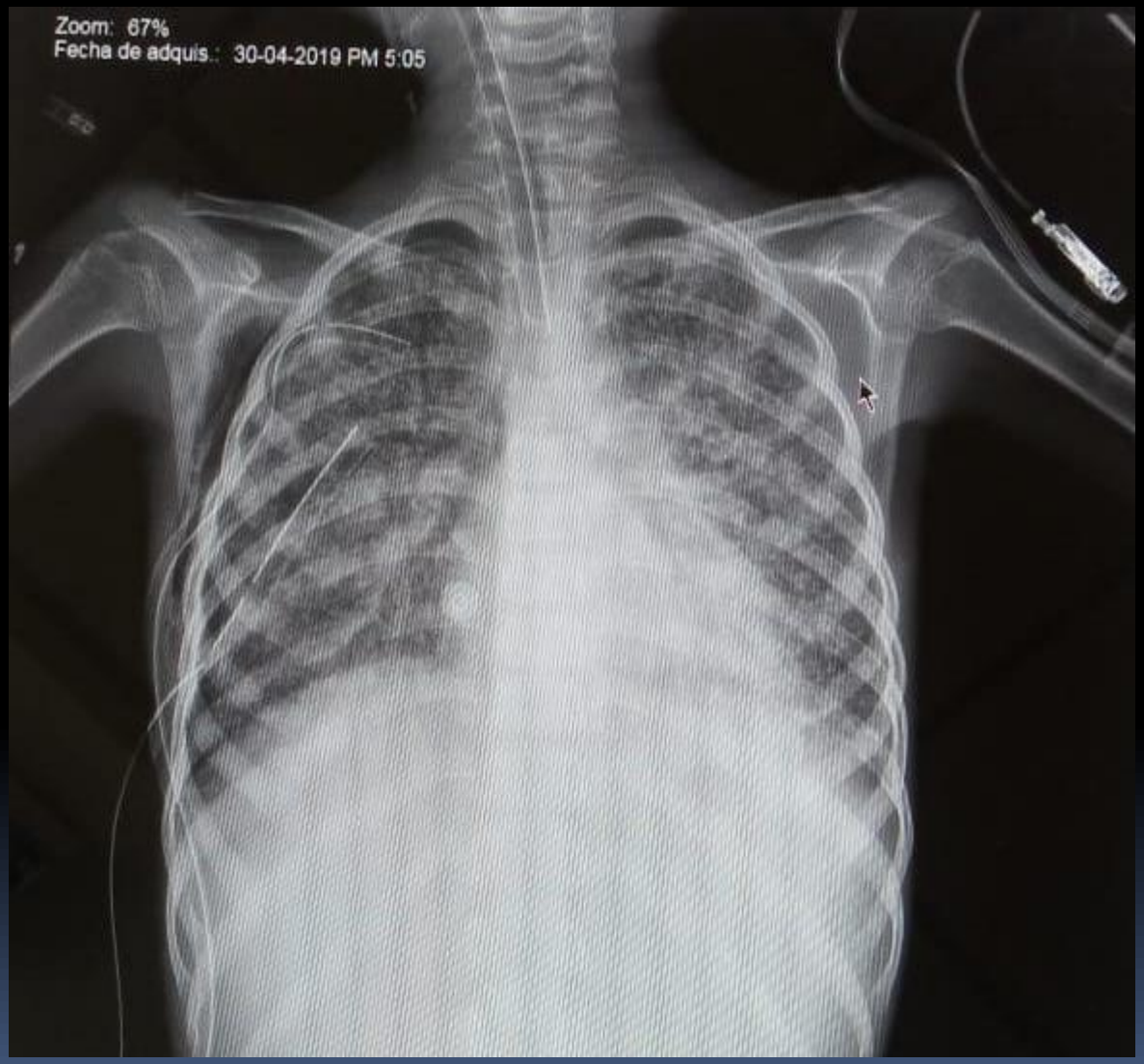
Intensive Care Med (2016) 42:699–711 DOI 10.1007/s00134-016-4325-4



Intensive Care Med (2016) 42:699–711 DOI 10.1007/s00134-016-4325-4

10 PUNTOS EN ARDS

1. 6ml Kg peso ideal
2. Presión Meseta < 30 cm H2O
3. Meseta – PEEP ≤ 15 cm H2O
4. No permita exista el grupo III
5. Poder ventilatorio < 17 J/min
6. Eficiencia ventilatoria < 2
7. Vigila el ventrículo derecho
8. ¿?
9. ¿?
10. ¿?



FO2(i): 80 %
Temp. del paciente: 36,5 °C

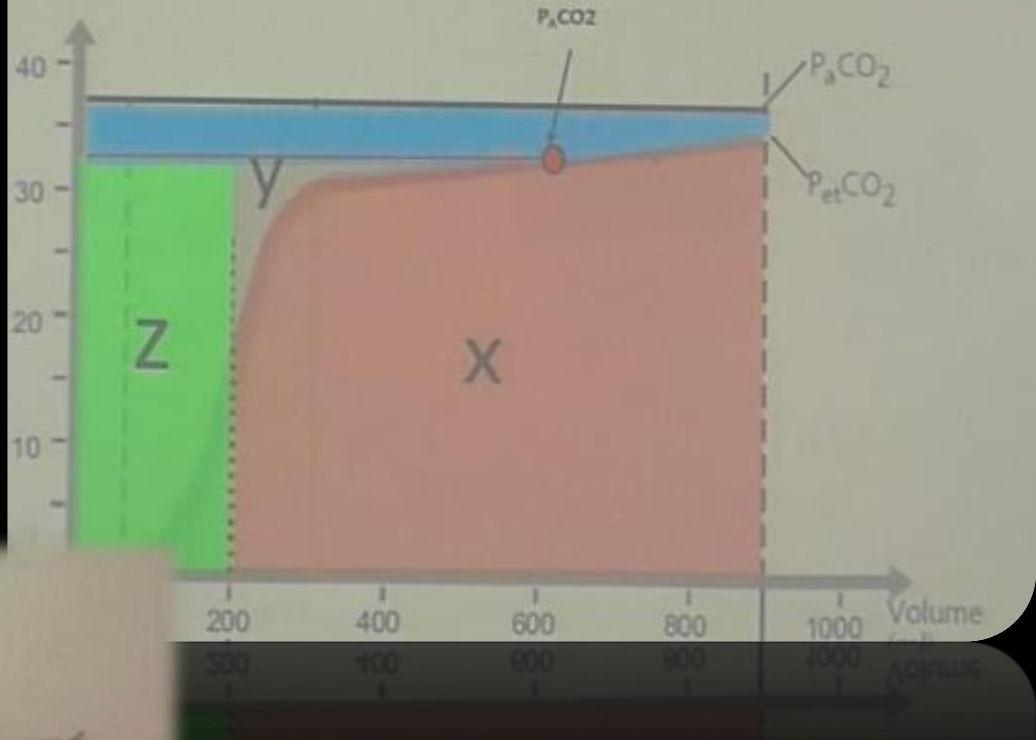
VALORES MEDIDO

Gases en Sangre (37°C)		
pH	↓	7,16
pCO_2	↑	72,4 mmHg
pO_2		92 mmHg
Temp. corregida (36.5 °C)		
pH(T)		7,16
$pCO_2(T)$		70.6 mmHg
$pO_2(T)$		90 mmHg
Hematocrito		
Hct		40 %
Electrólitos/Metabolitos		
cNa^+	↓	132 mmol/L
cK^+		4,67 mmol/L
cCa^{2+}	↓	0,52 mmol/L
cCl	↓	90 mmol/L
cLac		I/A mmol/L

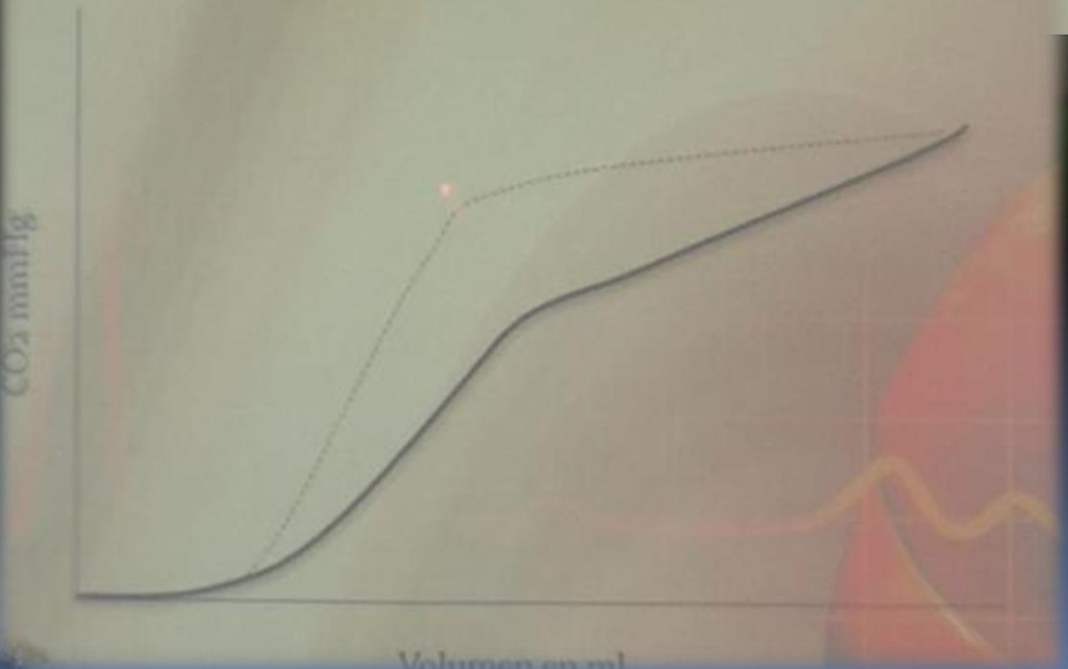
VALORES DERIVAD

sO_2	94,0 %
ctHb	13,1 g/dL
$cHCO_3$ (P)	24,6 mmol/L
$cHCO_3$ (P,st)	19,9 mmol/L
cBase(B)	-5,5 mmol/L
cBase(Ecf)	-3,1 mmol/L
$ctCO_2(B)$	23,4 mmol/L
$ctCO_2(P)$	26,8 mmol/L
$cCa^{2+}(7.40)$	N/D mmol/L
Anion Gap(K^+)	22,0 mmol/L
$pO_2(A)$	N/D mmHg
$pO_2(A\text{-}a)$	N/D mmHg
$pO_2(a/A)$	N/D %
ctO_2	17,2 Vol%
RI	N/D %

$$VD/VT = \frac{P_aCO_2 - P_ECO_2}{P_aCO_2}$$



SDRA



Factores que modifican el Vdalv Esp. Muerto Alveolar

Alto PEEP/ alto Vt.
Enfisema Pulmonar
Hipovolemia
Hipoperfusión
Hipertensión arterial
Embolismo Pulmonar

Factores que modifican el Vdaw (de las vías áreas)

Talla del paciente
Presión Pico Inspiratorio
Extensión del Cuello
Disminuye con Pausa Inspiratoria

Factores que modifican el VD mecánico

Humidificadores pasivos
Nebulizadores
Sondas de succión cerrada
Evitar aditamientos después de la Y

GRACIAS.....