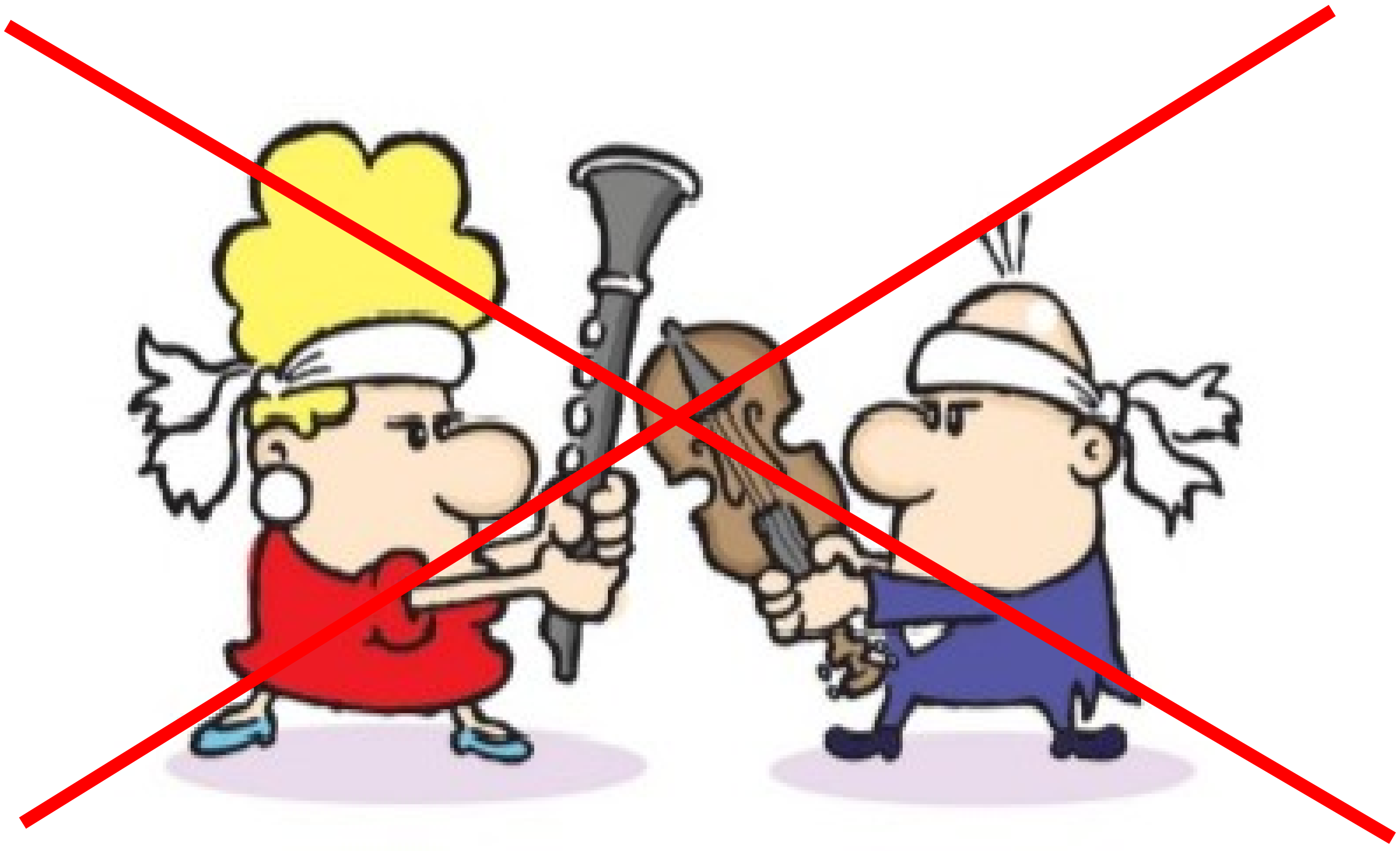
Driving pressure
není
můj cíl

Jan Máca

AKUTNĚ.CZ®

2021

NO Conflict of Interest



ΔP není cíl

dostupná evidence - nejasnosti

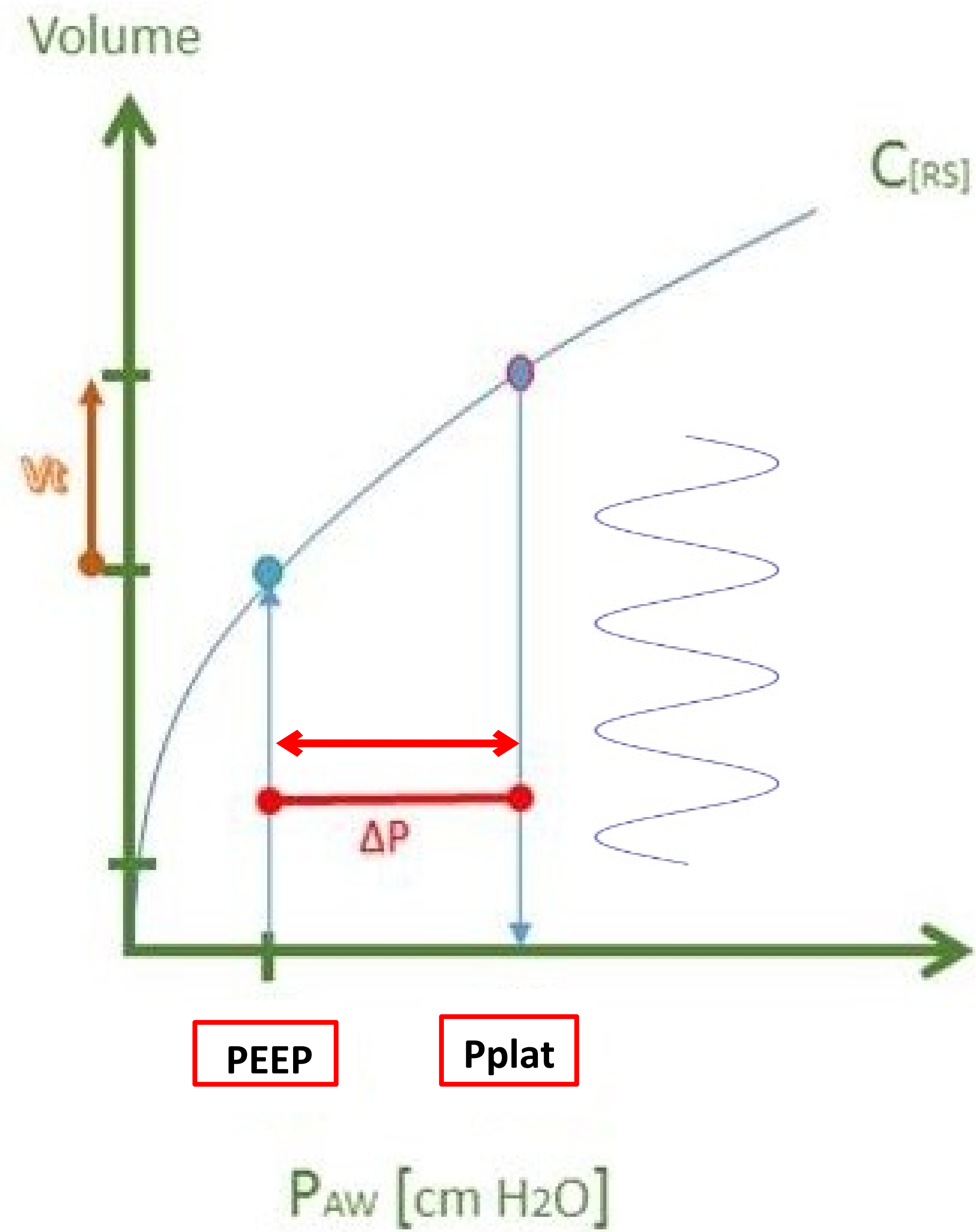
driving pressure vs. mechanical power

problémy užití v klinické praxi

závěr

$$\Delta P = Vt/Crs$$

$$\Delta P = Pplat - PEEP$$



ΔP není cíl

dostupná evidence - nejasnosti

Chicken or Egg?

driving pressure

ΔP není cíl

The NEW ENGLAND JOURNAL of MEDICINE

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.

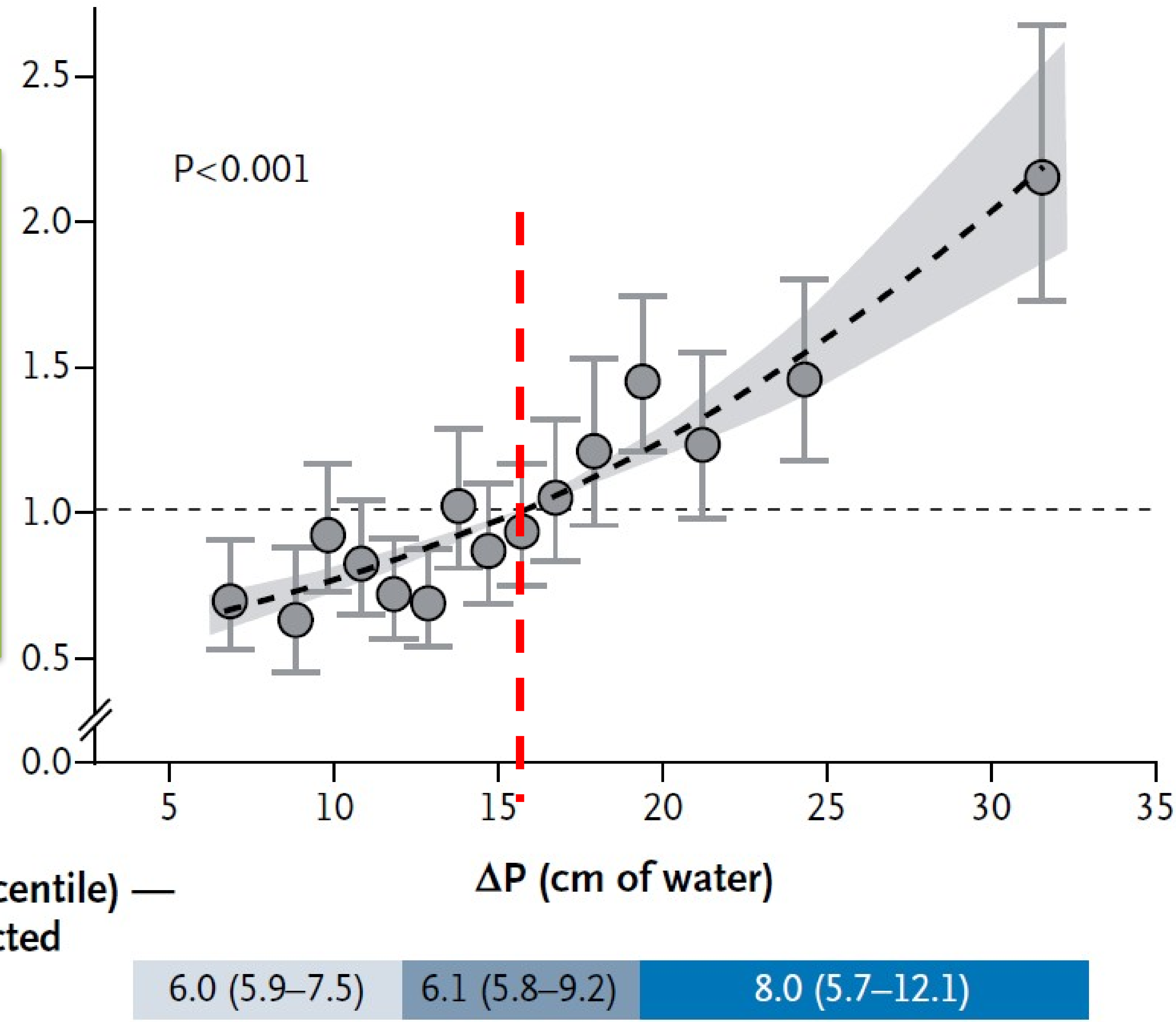
FEBRUARY 19, 2015

n = 3562, 9 studií

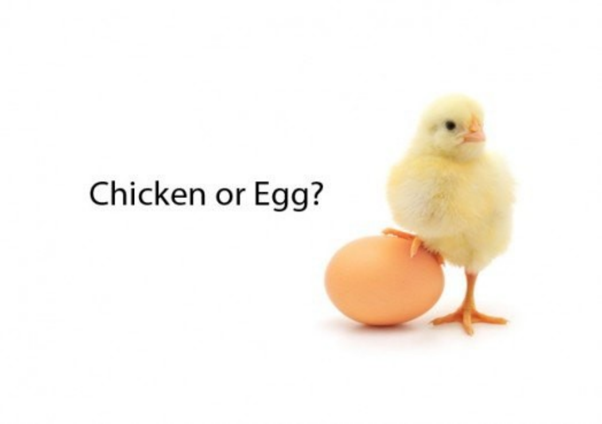
post hoc analysis

no inspiratory effort

Mutlivariantní analýza **relativního rizika** **smrti** v nemocnici



Median V_T (10th–90th percentile) — mg/kg of predicted body weight



ΔP jako klinický end-point

vs.

ΔP jako marker závažnosti

ΔP není cíl

Effect of driving pressure on mortality in ARDS patients during lung protective mechanical ventilation in two randomized controlled trials

Claude Guérin[1,2,3*], Laurent Papazian[4,5,6], Jean Reignier[7], Louis Ayzac[8], Anderson Loundou[5], Jean-Marie Forel[9]
and on behalf of the investigators of the Acurasys and Proseva trials

Critical Care (2016)

n = 787

2 LPV RCT

24 hodin after randomization

post hoc analysis

přežití 90 dní

Guerin, 2016

Table 1 Characteristics at the time of inclusion or day 1 between survivors and non-survivors at day 90

Variables	All ($n = 787$)	Survivors ($n = 533$)	Nonsurvivors ($n = 254$)	*P*
Male gender	542 (68.9)	366 (68.7)	176 (69.3)	0.923
Age, years	59 ± 16	56 ± 15	66 ± 14	<0.001
SAPS II on ICU admission	45 ± 15	45 ± 16	51 ± 15	<0.001
SOFA score on day 1	7 ± 4	7 ± 4	9 ± 4	<0.001
Continuous NMBA as allocation group	173 (22.0)	117 (22.0)	56 (22.0)	0.976
Prone position as allocation group	233 (34.4)	181 (34.0)	52 (20.5)	<0.001
Arterial pH on day 1	7.35 ± 0.09	7.36 ± 0.08	7.32 ± 0.10	<0.001
$PaCO_2$ on day 1, mmHg	47 ± 11	46 ± 11	47 ± 11	0.076
PaO_2/FIO_2 ratio on day 1	159 ± 74	163 ± 76	152 ± 68	0.056
Lactate on day 1, mmol/L	2.0 ± 1.9	1.8 ± 1.6	2.4 ± 2.2	<0.001
Respiratory rate on day 1,/minute	27 ± 6	26 ± 5	27 ± 6	0.010
Tidal volume on day 1, ml	397 ± 76	398 ± 76	395 ± 78	0.413
Tidal volume on day 1, ml/PBW kg	6.3 ± 0.8	6.2 ± 0.8	6.3 ± 0.8	0.691
PEEP on day 1, cm H2O	10 ± 3	10 ± 3	10 ± 3	0.210
Plateau pressure on day 1, cm H_2O	23 ± 4	23 ± 4	24 ± 4	<0.001
Tidal compliance on day 1, ml/cm H_2O	33 ± 12	34 ± 12	31 ± 12	0.016
Driving pressure on day 1, cm H_2O	13 ± 4	13 ± 4	14 ± 4	0.002
Mechanical power on day 1, J/min	13.4 ± 5.0	13.0 ± 4.8	14.3 ± 5.4	<0.001

Crs

Pplat

RR

prone

lactate

age

SOFA, SAPS II

ΔP jako jediný cíl

vs.

ΔP jako jeden z několika cílů

neovlivnitelné a **ovlivnitelné** parametry

věk, pohlaví…

pronace, RR, Pplat, flow

ΔP není cíl

VILI ↔ ΔP

hemostáza ↔ apTT, Q

mechanical power

ΔP není jediný cíl

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Guerin, 2016

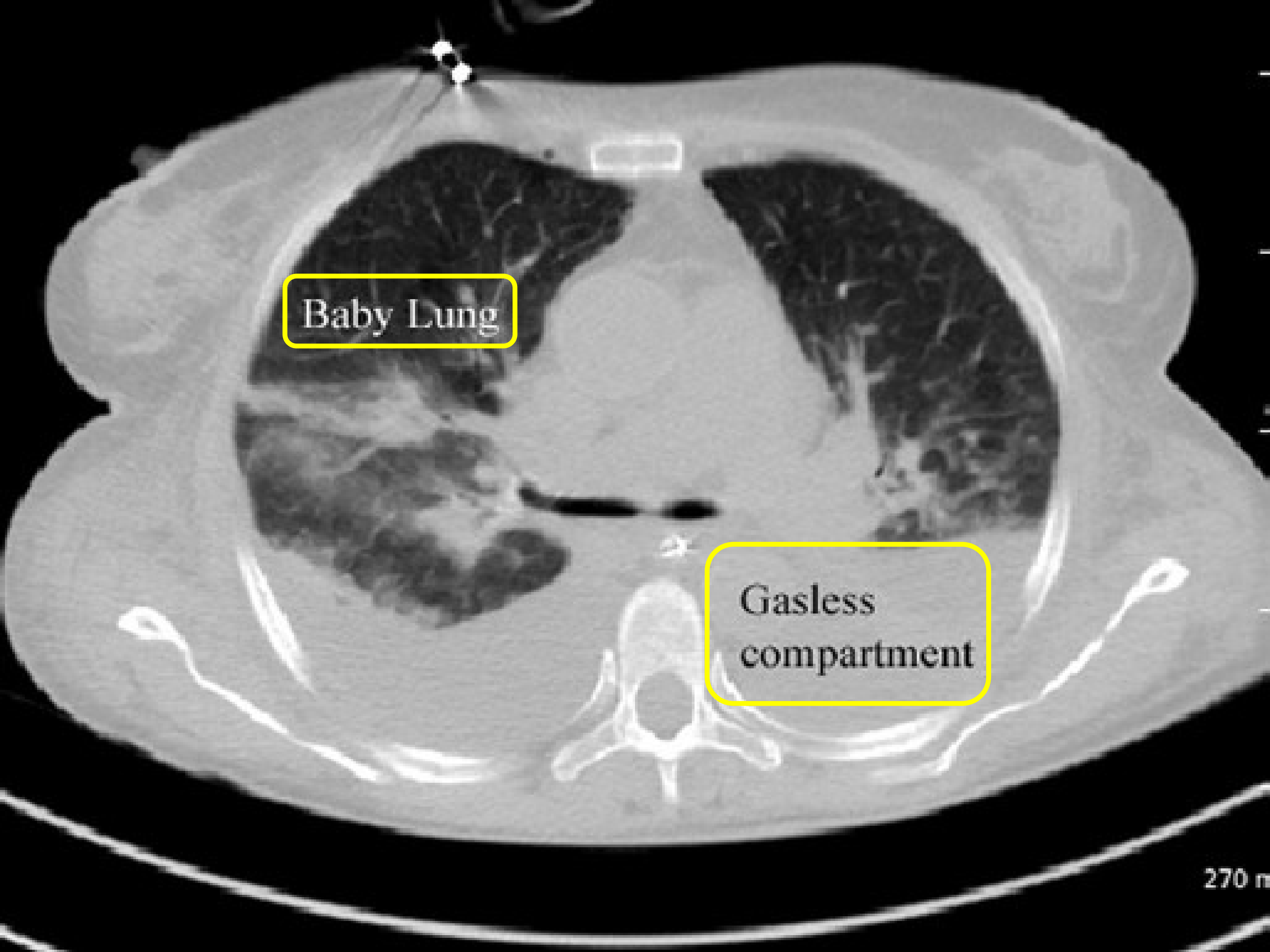
"Often, as new knowledge progresses, old knowledge is abandoned or forgotten."

Luciano Gattinoni

Gattinoni 2005
the baby lung concept

Amato 2015
driving pressure

Protti/Gattinoni/Cressoni/Marini
2016
mechanical power/driving energy


Baby Lung
Gasless compartment
270

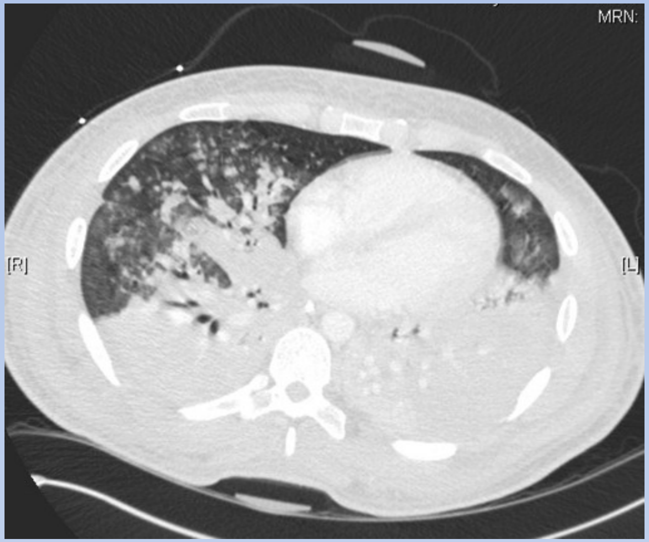
The "baby lung" became an adult

Intensive Care Med (2016) 42:663–673
DOI 10.1007/s00134-015-4200-8

Luciano Gattinoni
John J. Marini
Antonio Pesenti
Michael Quintel
Jordi Mancebo
Laurent Brochard

the aerated ARDS baby lung is

- **not stiff but small**
- with **near normal** intrinsic **mechanical characteristics**



The "baby lung" became an adult

Gattinoni 2016 ICM

strain

ratio between tidal volume and the baseline gas volume of the baby lung

Vt/FRC

limit 1.5

strain rate = strain/Ti

Crs : FRC (EELV, baby lung) ≈ **1 : 1**

ΔP = Vt/Crs

ΔP odpovídá velikosti strain

airway ΔP a predikce rozvoje VILI

ΔP není jediným ukazatelem VILI

- junctional forces
- vascular flow and pressures
- RACE (repetitive alveolar collapse and expansion)
- frequency (V_E)
- flow

MP

ΔP může přecenit riziko u

- stiff chest wall (obezita, ↑ IAP)
- neměřený autoPEEP

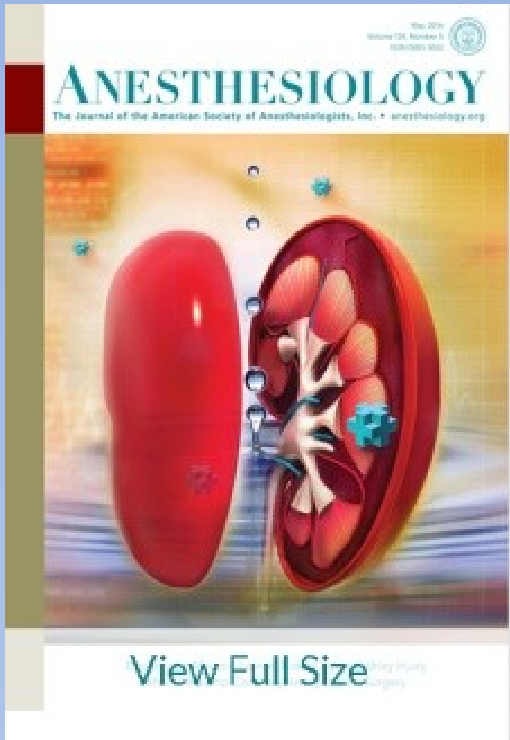
„falešně“ ↑ **ΔP**

ΔP může podcenit riziko u

- ↑ Raw
- spontánní ventilace

„falešně“ ↓ **ΔP**

driving pressure vs. mechanical power

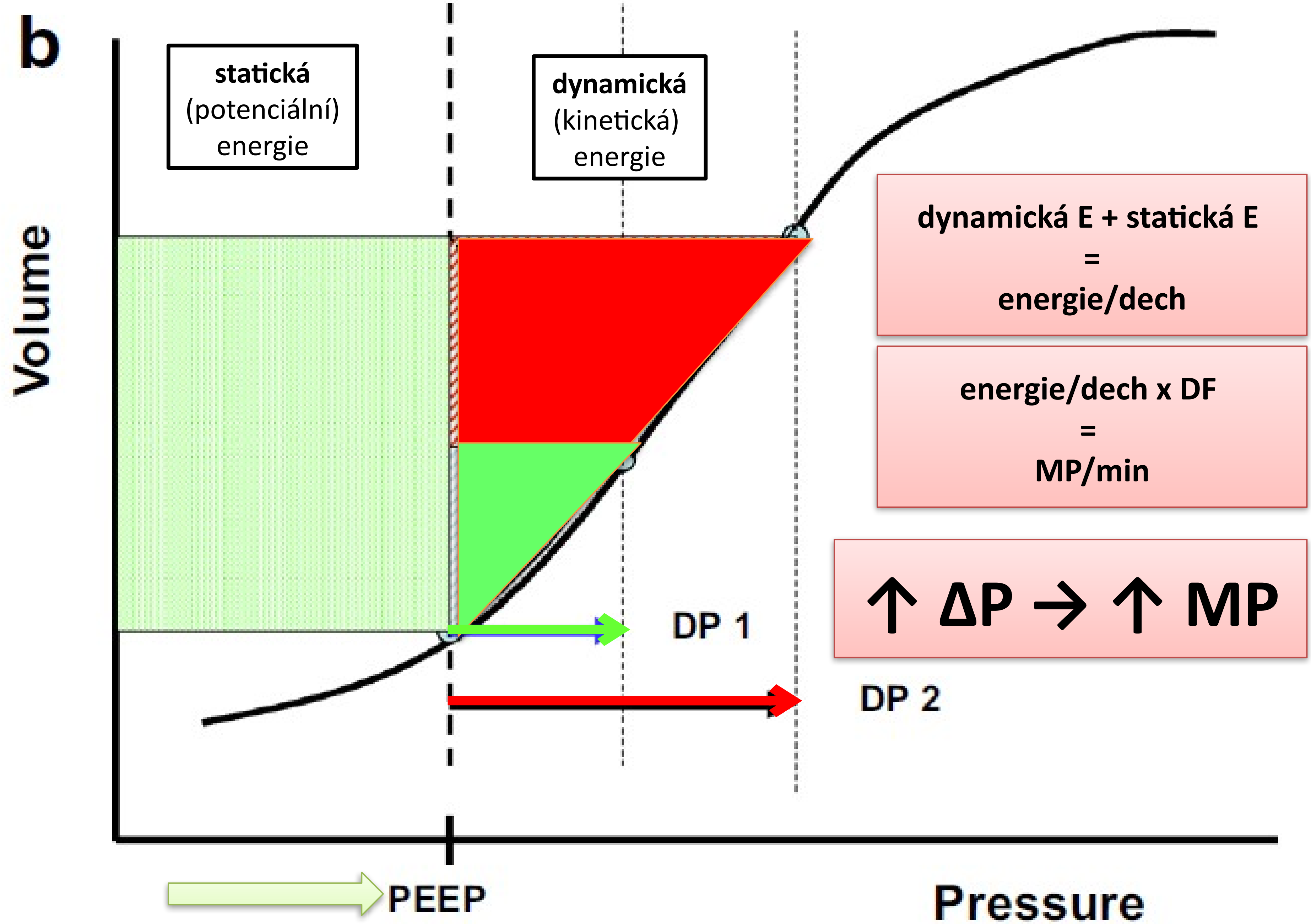


Critical Care Medicine | May 2016

Mechanical Power and Development of Ventilator-induced Lung Injury

Massimo Cressoni, M.D.; Miriam Gotti, M.D.; Chiara Chiurazzi, M.D.; Dario Massari, M.D.; Ilaria Algieri, M.D.; Martina Amini, M.D.; Antonio Cammaroto, M.D.; Matteo Brioni, M.D.; Claudia Montaruli, M.D.; Klodiana Nikolla, M.D.; Mariateresa Guanziroli, M.D.; Daniele Dondossola, M.D.; Stefano Gatti, M.D.; Vincenza Valerio, Ph.D.; Giordano Luca Vergani, M.D.; Paola Pugni, M.D.; Paolo Cadringher, M.Sc.; Nicoletta Gagliano, Ph.D.; Luciano Gattinoni, M.D., F.R.C.P.

- **subjekty (prasata)**
- **MP větší než 12 J/min rozvinuly VILI**
- **velikost MP korelovala s hmotností plic, plicní elastancí a poklesem indexu PaO_2/FiO_2**
- pod stanovenou hranicí se rozvinuly pouze **izolované** plicní denzity.


b
statická
(potenciální)
energie
dynamická
(kinetická)
energie
Volume
dynamická E + statická E
=
energie/dech
energie/dech x DF
=
MP/min
↑ ΔP → ↑ MP
DP 1
DP 2
PEEP
Pressure

ΔP reprezentuje dynamickou složku MP

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]

ICM 8/2016

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

MP increases:

- **Vt, ΔP, and flow (exponentially, exponent = 2)**
- **RR (exponent = 1.4)**
- **PEEP (linearly)**

driving power

relevatní část energie mechanického dechu je produktem ΔP a V_E

EDITORIAL

CrossMark

Dynamic predictors of VILI risk: beyond the driving pressure

John J. Marini[1*] and Samir Jaber[2]

ICM 2016

driving power = $\Delta P \times V_T \times RR/10C$

If damage resulted from **excess power**, it would not depend exclusively on the **maximal pressures** achieved during the individual **tidal cycle (Vt)** but rather on the entirety of the **inspiratory pressure excursion (DP)** and on the **frequency (RR)** of its

role hrudní stěny, stress raisers (inhomogenities), airway resistace, strain rate/flow, spontánní ventilace, PEEP

note: whether raising PEEP proves protective or deleterious has been thought to depend on its ability to recruit new units and whether DP or plateau pressure remains unchanged during the increase

ΔP není cíl

Effect of driving pressure on mortality in ARDS patients during lung protective mechanical ventilation in two randomized controlled trials

CrossMark

Claude Guérin[1,2,3*], Laurent Papazian[4,5,6], Jean Reignier[7], Louis Ayzac[8], Anderson Loundou[5], Jean-Marie Forel[9]
and on behalf of the investigators of the Acurasys and Proseva trials

Critical Care (2016)

n = 787

2 LPV RCT

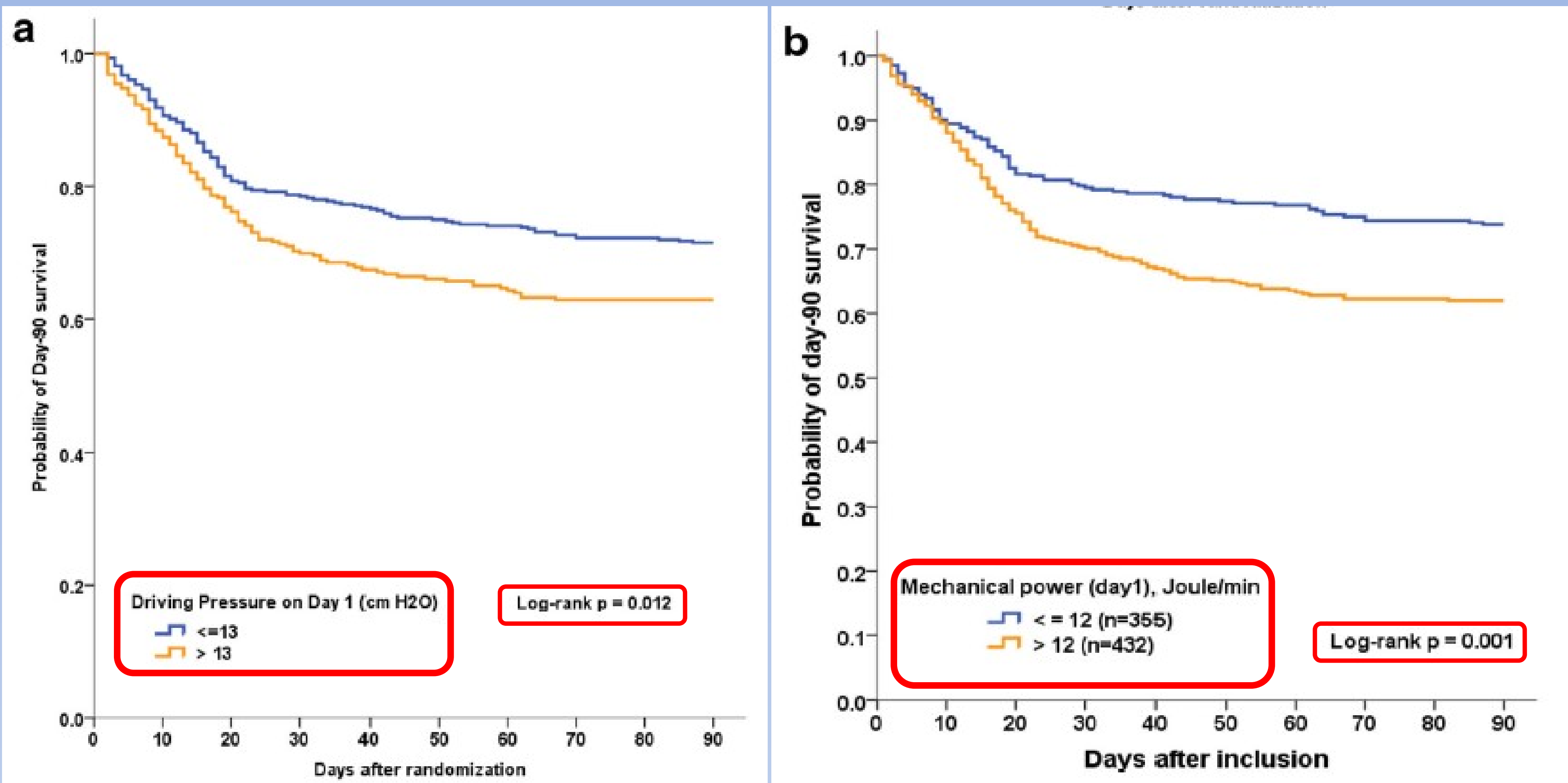
24 hodin after randomization

post hoc analysis

přežití 90 dní

ΔP

MP



90 day survival

problémy užití v klinické praxi

nejasná cílová skupina

nejasná hranice

nejasná cílová skupina

nejasná cílová skupina

heterogenita ARF/ARDS

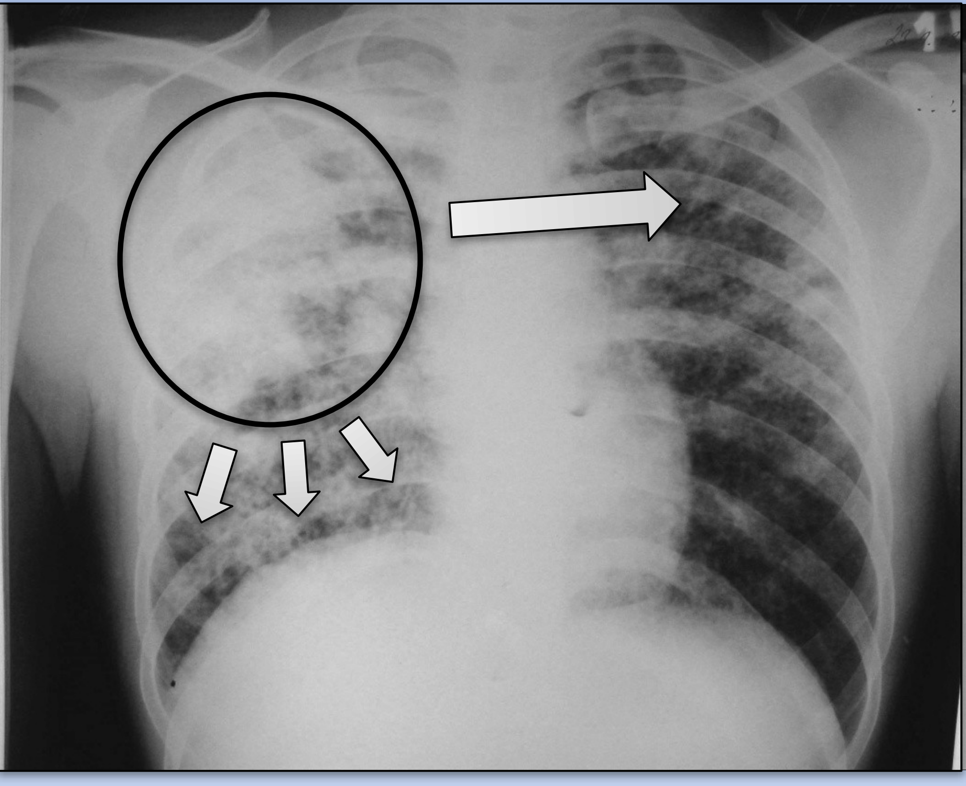
- etiologie
- převažující patomechanizmus
- míra poškození plicního parenchymu
- nehomogenity parenchymu plic, stress raisers
- dynamika a reverzibilita poškození

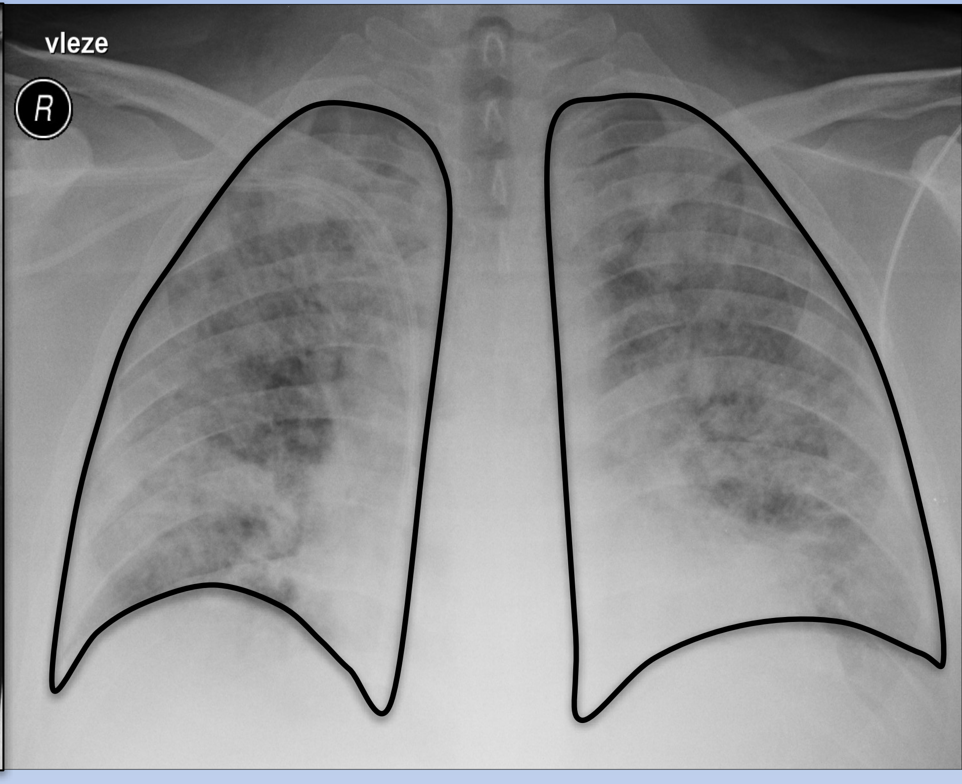
nejasná cílová skupina

ARDS

PRIMARY
(pulmonary, direct)

SECONDARY
(extrapulmonary, indirect)





nejasná cílová skupina

Intensive Care Med (2004) 30:1111–1116
DOI 10.1007/s00134-004-2163-2

ORIGINAL

Niall D. Ferguson
Robert M. Kacmarek
Jean-Daniel Chiche
Jeffrey M. Singh
David C. Hallett
Sangeeta Mehta
Thomas E. Stewart

Screening of ARDS patients using standardized ventilator settings: influence on enrollment in a clinical trial

Standardizované nastavení mechanické ventilace před zařazením do HFOV studie

- Vt 7-8ml/kg PBW
- PEEP 10 cm H_2O nebo více dle oxygenace, k SpO_2 nad 88%
- FiO_2 1.0
- pacienti $PaO_2/FiO_2 < 200$ mm Hg

aplikace 30min

nejasná cílová skupina

Intensive Care Med (2004) 30:1111–1116
DOI 10.1007/s00134-004-2163-2

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Screening of ARDS patients using standardized ventilator settings: influence on enrollment in a clinical trial

41.5% pacientů
PaO_2/FiO_2 < 200 mm Hg

mortalita 52.9%

58.5% pacientů
PaO_2/FiO_2 > 200 mm Hg

mortalita 12.5%

nejasná cílová skupina

Intensive Care Med (2004) 30:1111–1116
DOI 10.1007/s00134-004-2163-2

ORIGINAL

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Sangeeta Mehta
Thomas E. Stewart

Screening of ARDS patients using standardized ventilator settings: influence on enrollment in a clinical trial

mortalita 52.9%

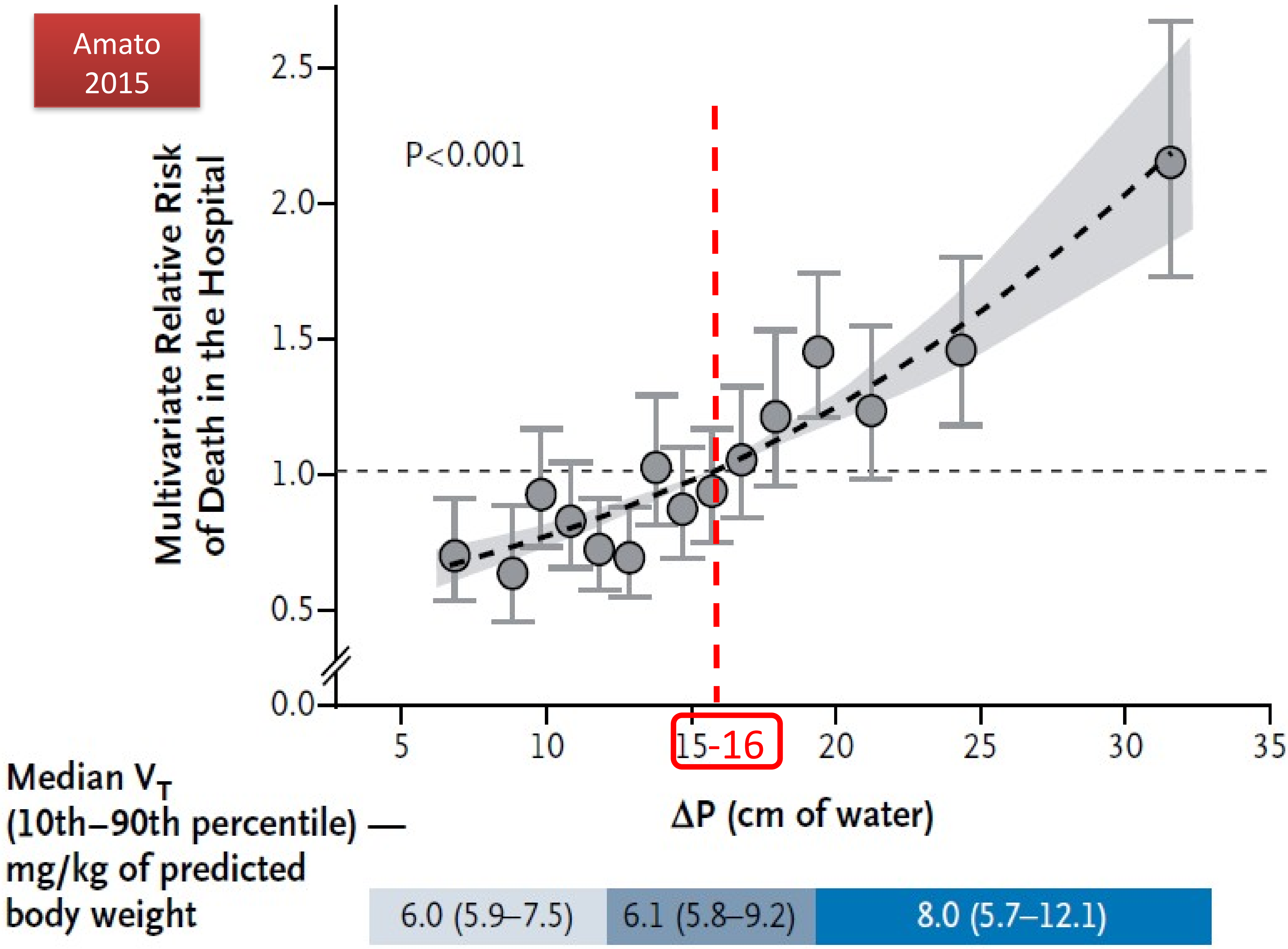
- pneumonie
- šok
- inhalační poškození
- sepse (35%)

mortalita 12.5%

- aspirace
- pankreatitis
- mnohočetné transfůze
- sepse (33%)

„trazientní“ ARDS? etiologie?

nejasná hranice

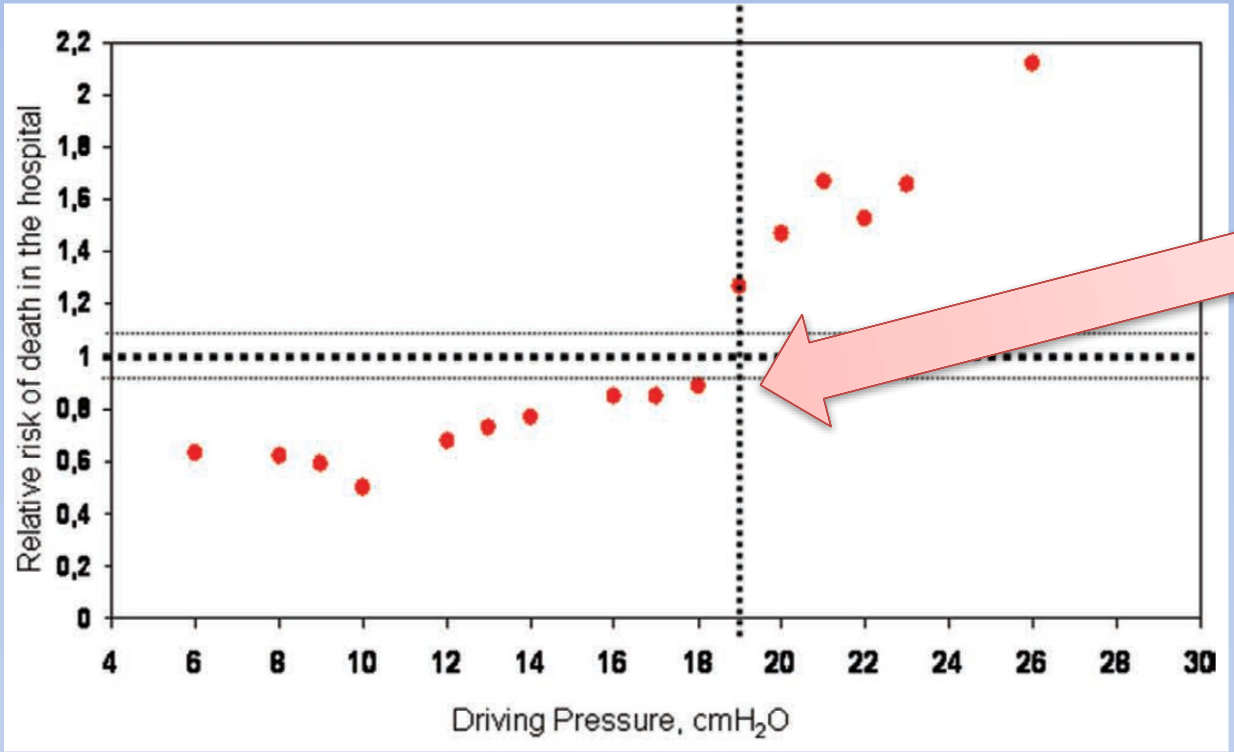

Amato
2015
P<0.001
Multivariate Relative Risk
of Death in the Hospital
2.5
2.0
1.5
1.0
0.5
0.0
5
10
15-16
20
25
30
35
ΔP (cm of water)
Median V_T
(10th–90th percentile) —
mg/kg of predicted
body weight
6.0 (5.9–7.5)
6.1 (5.8–9.2)
8.0 (5.7–12.1)

nejasná hranice

A Quantile Analysis of Plateau and Driving Pressures: Effects on Mortality in Patients With Acute Respiratory Distress Syndrome Receiving Lung-Protective Ventilation

Jesús Villar, MD, PhD, FCCM[1,2]; Carmen Martín-Rodríguez, MD[3]; Ana M. Domínguez-Berrot, MD[4]; Lorena Fernández, MD[5]; Carlos Ferrando, MD, PhD[6]; Juan A. Soler, MD, PhD[7]; Ana M. Díaz-Lamas, MD[8]; Elena González-Higueras, MD, PhD[9]; Leonor Nogales, MD[10]; Alfonso Ambrós, MD, PhD[3]; Demetrio Carriedo, MD, PhD[4]; Mónica Hernández, MD[11]; Domingo Martínez, MD[12]; Jesús Blanco, MD, PhD[5]; Javier Belda, MD, PhD[6]; Dácil Parrilla, MD[13]; Fernando Suárez-Sipmann, MD, PhD[1,14]; Concepción Tarancón, MD[15]; Juan M. Mora-Ordoñez, MD[16]; Lluís Blanch, MD, PhD[1,17]; Lina Pérez-Méndez, MD, PhD[1,18]; Rosa L. Fernández, MSc[1,2]; Robert M. Kacmarek, PhD, RTT[19,20]; for the Spanish Initiative for Epidemiology, Stratification and Therapies of ARDS (SIESTA) Network

Critical Care Medicine 2017



ΔP limit 19 cmH_2O

- **Guerin 13 cm H_20**
- **Amato 15-16 cm H_2O**
- **Villar 19 cmH_2O**

Intraoperative protective mechanical ventilation and risk of postoperative respiratory complications: hospital based registry study

Karim Ladha, research fellow, Marcos F Vidal Melo, associate professor, [...], and Matthias Eikermann, associate professor of anesthesia and critical care medicine, clinical director

BMJ 2015

retrospektivní studie

intraoperační

$\Delta P > 13 cm\ H_2O$

↑ PPC

(2x)

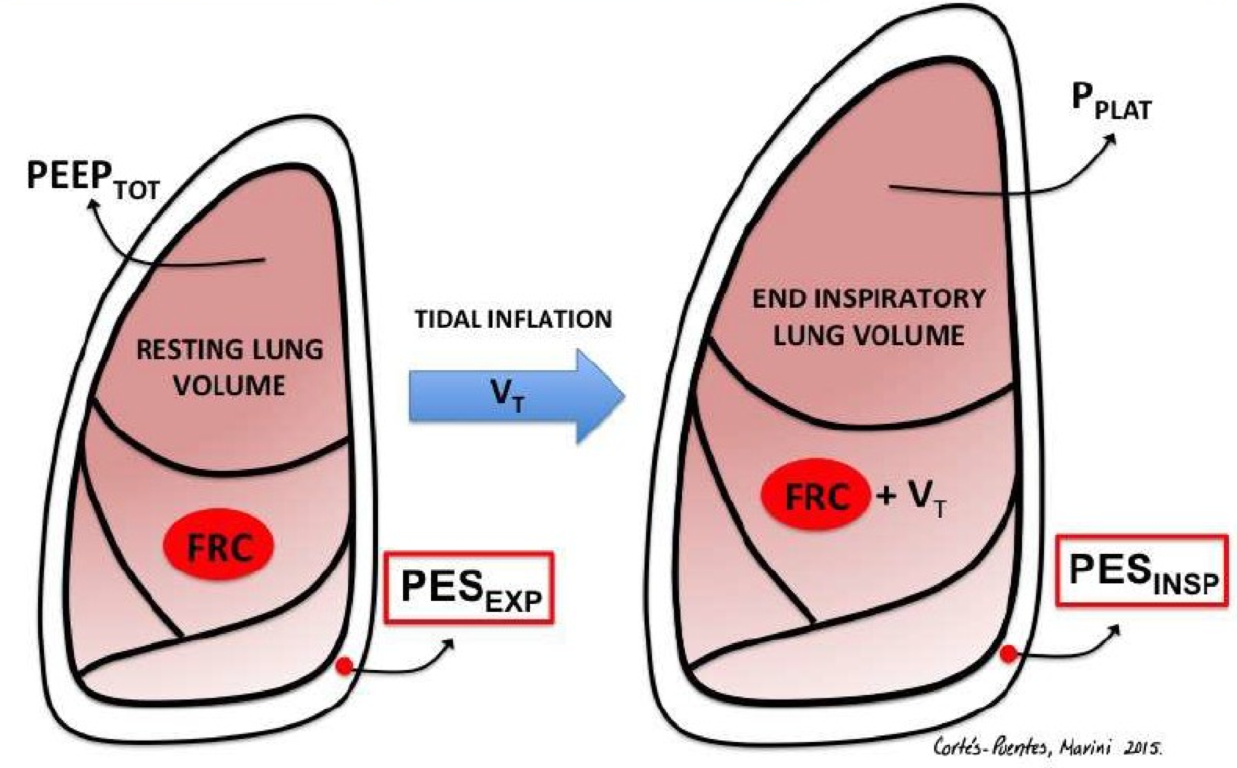
transpulmonální ΔP

Trans-Pulmonary Driving Pressure

$DP_{AW} = V_T / C_{RS}$
$= P_{PLAT} - PEEP$
$= P_{PLAT} - PEEP_{TOT}$

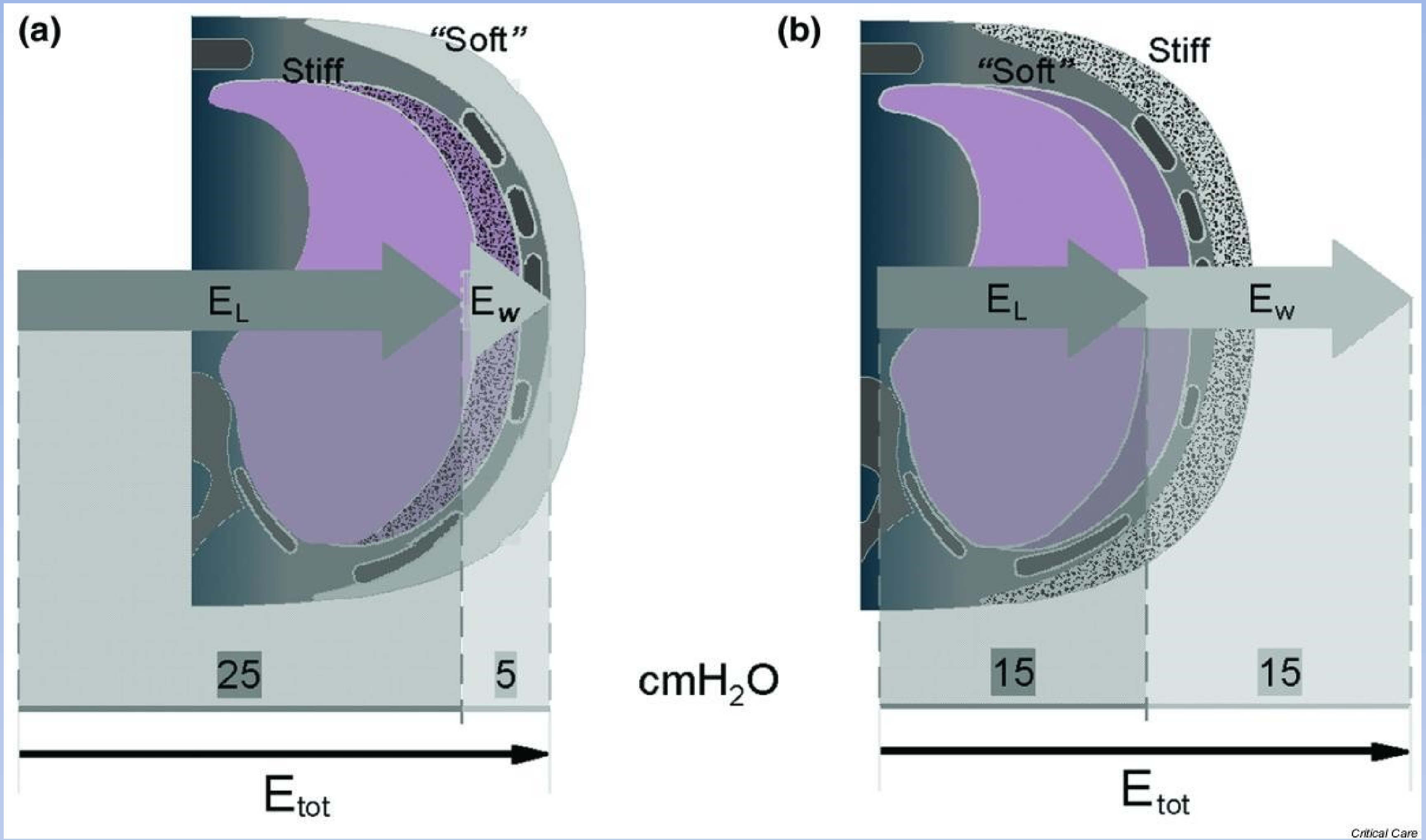
$P_{TP} = P_{AW} - P_{ES}$
$\text{INSP } P_{TP} = P_{PLAT} - PES_{INSP}$
$\text{EXP } P_{TP} = PEEP_{TOT} - PES_{EXP}$

$DP_{TP} = V_T / C_L$
$= \text{INSP } P_{TP} - \text{EXP } P_{TP}$



Insp TP
-
Exp TP

vlastnosti hrudní stěny zkreslují reálný distenční tlak působící na plicní parenchym



(a)
"Soft"
Stiff
E_L
E_w
25
5
cmH$_2$O
E_{tot}
(b)
Stiff
"Soft"
E_L
E_w
15
15
E_{tot}
Critical Care

$$E_{tot} = E_L + E_W$$

Intensive Care Med (2014) 40:1670–1678
DOI 10.1007/s00134-014-3415-4

ORIGINAL

Davide Chiumello
Massimo Cressoni
Andrea Colombo
Giovanni Babini
Matteo Brioni
Francesco Crimella
Stefan Lundin
Ola Stenqvist
Luciano Gattinoni

The assessment of transpulmonary pressure in mechanically ventilated ARDS patients

ARDS Chest Wall is Often ***NOT*** Normal

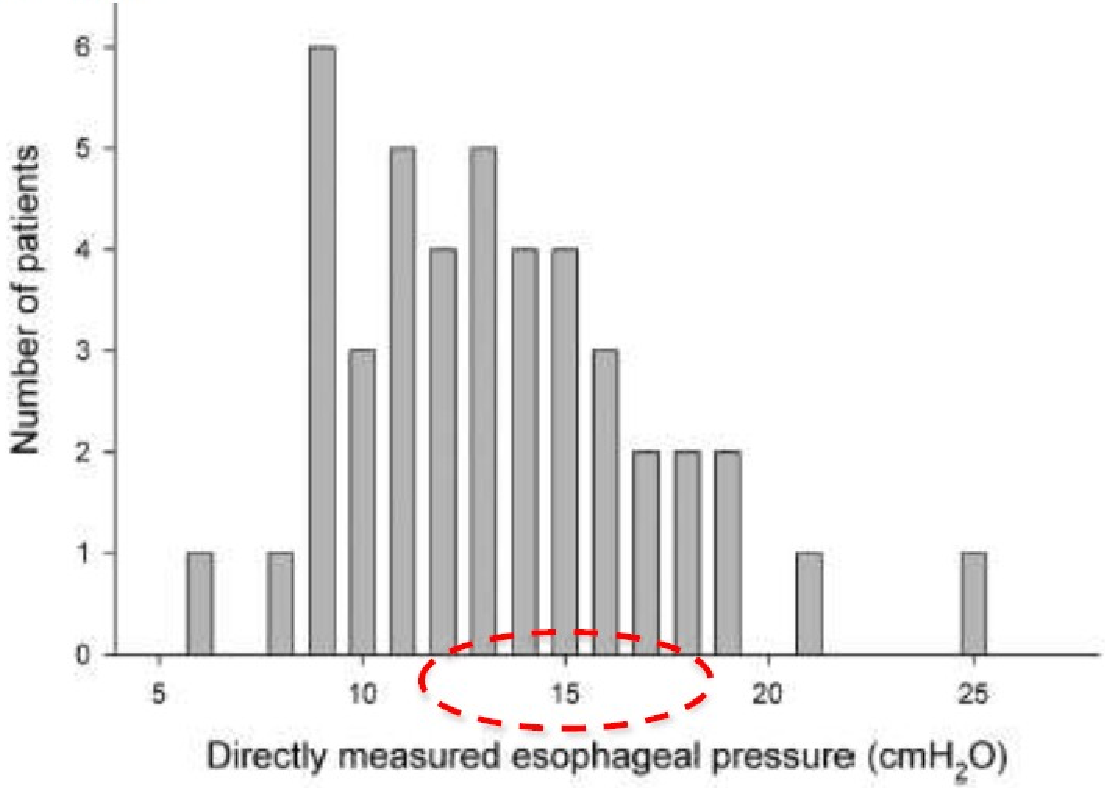
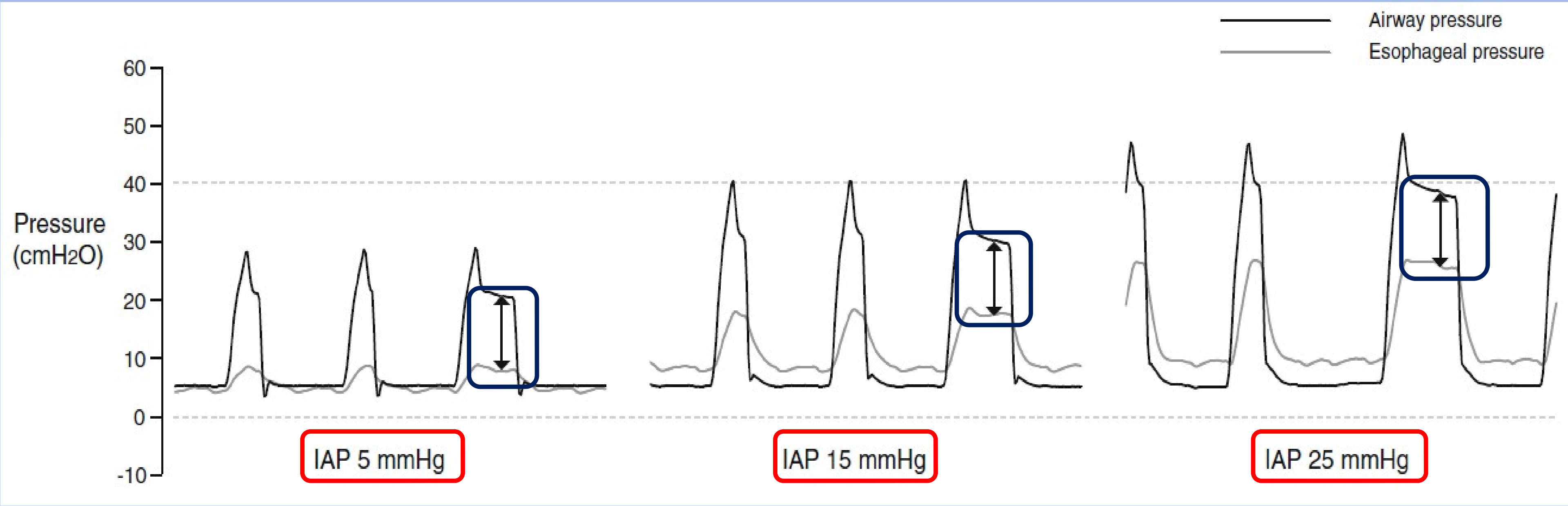


Fig. 5 Distribution (histogram) of directly measured esophageal pressure at 5 cmH_2O of PEEP

nejasná hranice

IAP a ezofageální tlak



různé ΔP

stejné TP ΔP

nejasná hranice

ORIGINAL

Mortality and pulmonary mechanics in relation to respiratory system and transpulmonary driving pressures in ARDS

Elias Baedorf Kassis[1*], Stephen H. Loring[2] and Daniel Talmor[2]

Intensive Care Med (2016)

Conclusions: The results suggest that utilizing PEEP titration to target positive transpulmonary pressure via esophageal manometry causes both improved elastance and driving pressures. Treatment strategies leading to decreased respiratory system and transpulmonary driving pressure at 24 h may be associated with improved 28 day mortality. Studies to clarify the role of respiratory system and transpulmonary driving pressures as a prognosticator and bedside ventilator target are warranted.

TP ΔP

limit < 10 cmH_2O

nejasná hranice

role hrudní stěny

TP ΔP

lepší posouzení role kvality hrudní stěny

přesnější (lepší) než airway driving pressure?

- obezita
- extrapulmonální ARF/ARDS
- IAP

další faktory

role aktivity pacienta

- řízená vs. asistovaná vs. **spontánní ventilace** na UPV
- **míra svalové relaxace**
- **míra analgosedace**
- přítomnost **pacient-ventilatorové asynchronie**
- **„dávka" ΔP** (časový faktor/expozice)

role ECMO

The NEW ENGLAND JOURNAL of MEDICINE

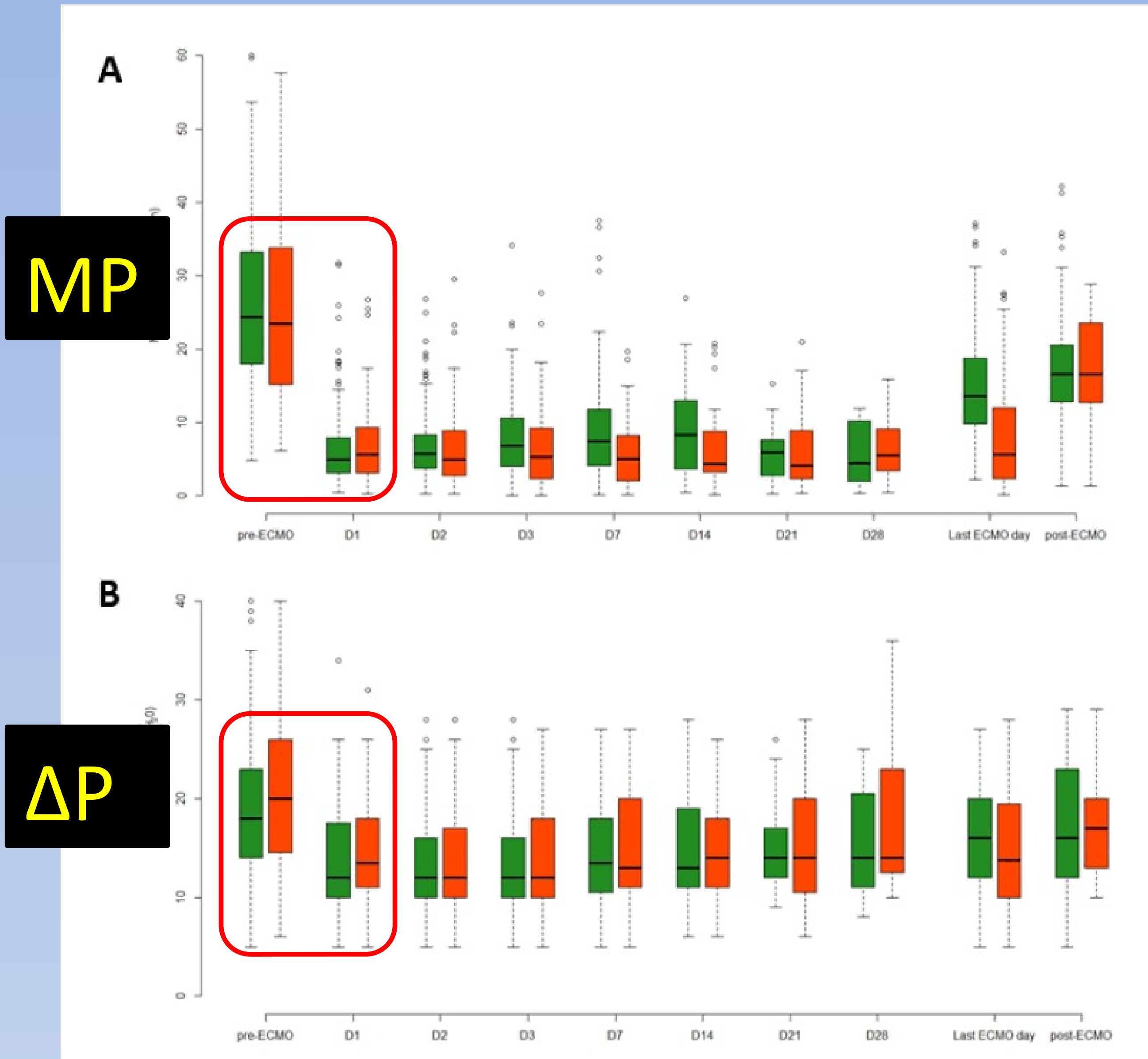
ESTABLISHED IN 1812 MAY 24, 2018 VOL. 378 NO. 21

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, 2019

EOLIA 2019



významné snížení ΔP (MP) při uLPV

bez zlepšení mortality

závěr I

airway ΔP

nemůže být **jediný** cíl nastavení LPV

role **Vt, Pplat, RR, Crs, flow**

komplexní role **MP**

role homogenity plic, doby expozice

role **TP ΔP** a hrudní stěny (hrudní koš, bránice) závěr II

airway ΔP

nikdo **zatím** neprokázal prospektivně benefit ΔP jako klinického cíle

není jasný **limit**

není jasná role ΔP u **různé etiologie ARF/ARDS**

není jasná role ΔP u **spontánní ventilace,** vliv **analgosedace**

nutná je perzonalizace nastavení UPV

(dynamika změn, etiologie, mechanizmus poškození,…)

future clinical evidence needed

děkuji za pozornost