AMARM
ASSOCIATION DES MEDECINS ANESTHÉSISTES RÉANIMATEURS DE MARRAKECH

La 15éme édition
Des Journées d'Anesthésiologie de Marrakech

SMAR
Société Marocaine d'Anesthésie et de Réanimation

Actualités dans le SDRA chez l'adulte

A. Hachimi
Service de Réanimation Médicale
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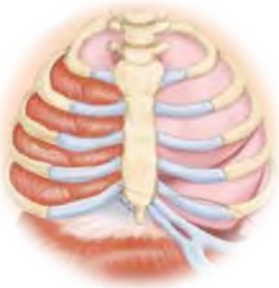

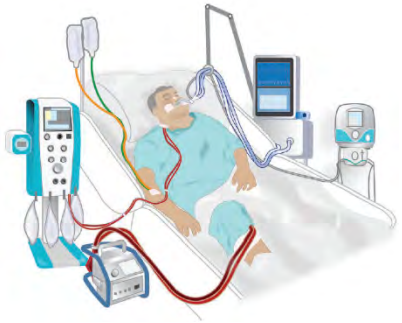

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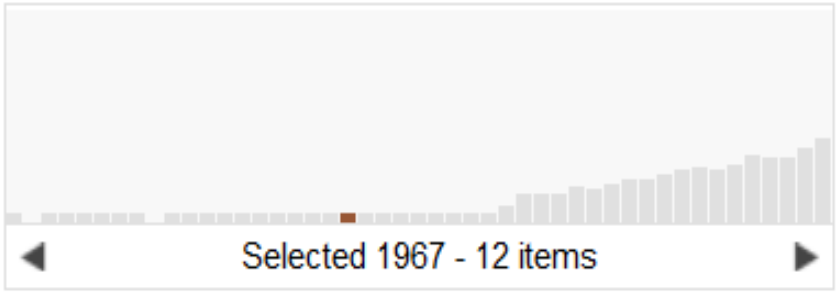
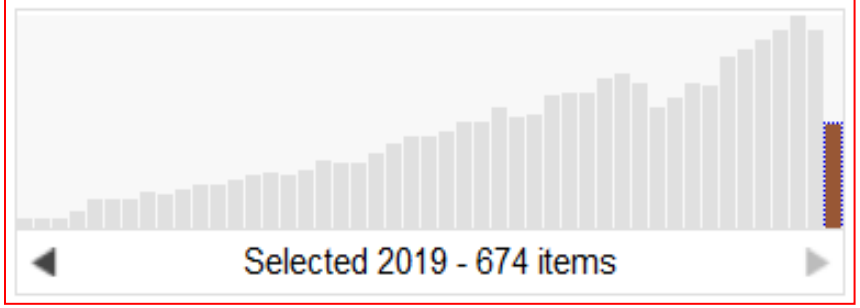


Results by year



THE LANCET

ORIGINAL ARTICLES | VOLUME 290, ISSUE 7511, P319-323, AUGUST 12, 1967

ACUTE RESPIRATORY DISTRESS IN ADULTS

DavidG. Ashbaugh, M.D. Ohio State • D. Boyd Bigelow, M.D. Colorado • ThomasL. Petty, M.D. Colorado • BernardE. Levine, M.D. Michigan • Show footnotes

Published: August 12, 1967 • DOI: https://doi.org/10.1016/S0140-6736(67)90168-7

Acute Respiratory Distress Syndrome

The Berlin Definition

Table 3. The Berlin Definition of Acute Respiratory Distress Syndrome

	Acute Respiratory Distress Syndrome
Timing	Within 1 week of a known clinical insult or new or worsening respiratory symptoms
Chest imaging[a]	Bilateral opacities—not fully explained by effusions, lobar/lung collapse, or nodules
Origin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload Need objective assessment (eg, echocardiography) to exclude hydrostatic edema if no risk factor present
Oxygenation[b]	
Mild	200 mm Hg $<$ Pa_{O_2}/F_{IO_2} $\leq$ 300 mm Hg with PEEP or CPAP $\geq$5 cm H_2O[c]
Moderate	100 mm Hg $<$ Pa_{O_2}/F_{IO_2} $\leq$ 200 mm Hg with PEEP $\geq$5 cm H_2O
Severe	Pa_{O_2}/F_{IO_2} $\leq$ 100 mm Hg with PEEP $\geq$5 cm H_2O

JAMA. 2012;307(23):2526-2533

Hospital Incidence and Outcomes of the Acute Respiratory Distress Syndrome Using the Kigali Modification of the Berlin Definition

Elisabeth D. Riviello[1,2], Willy Kiviri[3], Theogene Twagirumugabe[3], Ariel Mueller[4], Valerie M. Banner-Goodspeed[4], Laurent Officer[4], Victor Novack[5], Marguerite Mutumwinka[6], Daniel S. Talmor[4], and Robert A. Fowler[7]

- Timing and origin: as in the Berlin definition
- Imaging: bilateral opacities on chest radiography or ultrasonography scan not fully explained by effusion, collapse or nodules
- Oxygenation: SpO_2/FiO_2 <315; no PEEP requirement

Am J Respir Crit Care Med Vol 193, Iss 1, pp 52–59, Jan 1, 2016

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Saudi J Anaesth. 2018 Jul-Sep; 12(3): 457–461.
doi: 10.4103/sja.SJA_73_18: 10.4103/sja.SJA_73_18
PMCID: PMC6044168
PMID: 30100847

Lung ultrasound: Predictor of acute respiratory distress syndrome in intensive care unit patients

Ying Zhou,[1,2] Qianqian Fan,[1,3] Omer Cavus,[1] and Xuezheng Zhang[4]

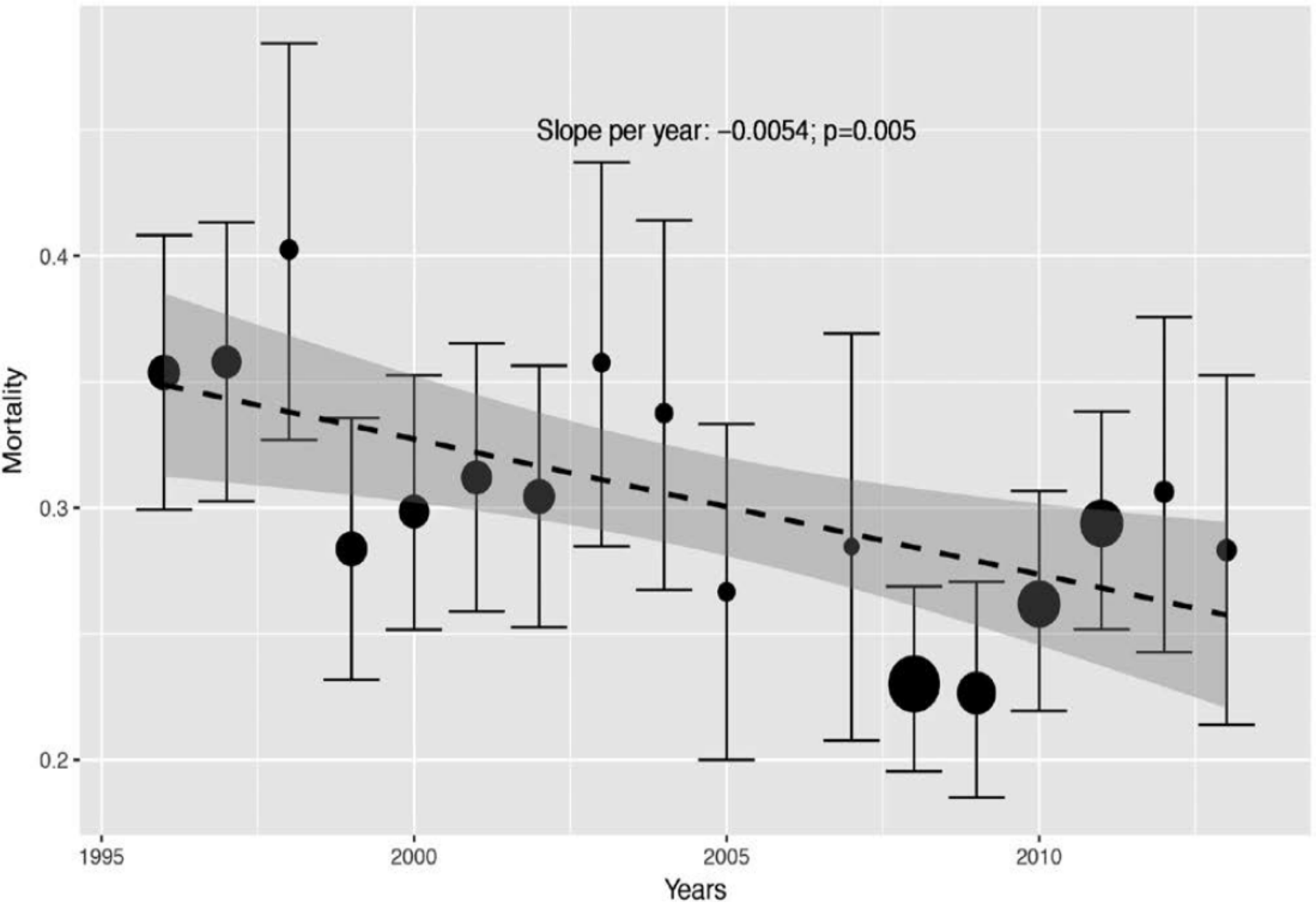
Conclusions

The limitations of LUS are not essential when considering its diagnosis and prognosis capabilities for patients with ARDS. It is an easily available, user-friendly, and cost-effective medical technique that involves no ionizing radiation. It is complementary to bedside chest X-ray and reduces the need to use a CT scan. LUS is a helpful tool used to diagnose, treat, and predict the prognosis of ARDS patients.

Year (timespan of study sample)	Location of study sample	Severity of ARDS	Incidence estimate	Mortality estimate (%)
Incidence per 100,000 person-years				
1990–1994	North Beaches Peninsula, Australia[42]	Moderate, severe	7.3–9.3	59[a]
1997	Sweden, Denmark, and Iceland[35]	All	17.9	42.2[b]
1999	Southern, Western, and Tasmania, Australia[36]	All	34	32[c]
1999–2000	King Co., Washington, United States[27]	All	78.9	38.5[a]
2001	Granada, Spain[37]	Moderate, severe	23	66[a]
2005 (3 days)	Netherlands[43]	All	29.3	NR
2006–2007	Victoria, Brazil[38]	All	10.1	49.2[a]
2007	Finland[25]	All	10.6	47[b]
2008	Olmsted Co., Minnesota, United States[24]	All	38.3	45[a]
2008–2009	Spain[29]	Moderate, severe	7.2	47.8[a]
1988–2010	Iceland[28]	Moderate, severe	3.6–9.6	12–78[a]
Incidence among admissions to intensive care unit				
1999 (2 winter months)	10 European countries[23]	All	7.1%	54.7[a]
2002	24 European countries[41]	All	12.5%	45.5[a]
2006 (10 summer/fall wk)	Ireland[46]	All	19%	32.3[d]
2014 (4 winter wk)	50 countries worldwide[20]	All	10.4%	40[a]

Abbreviations: ARDS, acute respiratory distress syndrome; ICU, intensive care unit; NR, not reported.
[a]In-hospital mortality.
[b]90-day mortality.
[c]28-day mortality.
[d]ICU mortality.

Semin Respir Crit Care Med 2019;40:3–11.



Crit Care Med 2019; 47:315–323

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

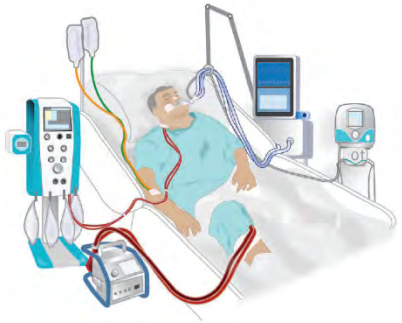
eTable 4. Ventilation of invasively ventilated patients with recognized versus unrecognized ARDS

Extent of ARDS Recognition	All (n=2377)	Mild (n=714; 30.0%)	Moderate (n=1106; 46.5%)	Severe (n=557; 23.4%)	P value [1]
ARDS Recognition at any time No. (%) (95% CI)	1525 (64.2%) (62.2-66.1)	366 (51.3%) (47.5-55.0)	722 (65.3%) (62.4-68.1)	437 (78.5%) (74.8-81.8)	<0.001

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

Acute Respiratory Distress Syndrome Phenotypes

John P. Reilly, MD, MSCE[1] Carolyn S. Calfee, MD, MAS[2] Jason D. Christie, MD, MSCE[1]



Semin Respir Crit Care Med 2019;40:19–30.

Phenotype	Description	Differences	Potential therapies	References
Hypoxia severity phenotypes	Berlin categories: Mild: 200 < PaO_2/FiO_2 < 300 Mod: 100 < PaO_2/FiO_2 < 200 Severe: PaO_2/FiO_2 < 100	• Severity of hypoxia • DAD more likely pathology in severe	• Prone positioning (PaO_2/FiO_2 < 150) • Cisatracurium (PaO_2/FiO_2 < 150)	4,7,41
ARDS by precipitating risk factor	Precipitating factors including: sepsis, trauma, pneumonia, aspiration, transfusion, pancreatitis	• Differences in ARDS risk, severity, and mortality		53,56–60
Direct versus indirect lung injury	Direct: pneumonia, pulmonary contusion, aspiration Indirect: nonpulmonary sepsis, nonthoracic trauma, transfusions	• Epithelial vs. endothelial injury • Differences in mortality	• Epithelial vs. endothelial targeted therapies • Indirect more likely to respond to PEEP	61,64–74
Timing of onset phenotypes	Early onset developing <48 h from admission versus late onset >48 h from admission	• Different clinical characteristics • Elevated RAGE and Ang-2 in early onset		75–78
Radiographic phenotypes	Nonfocal/diffuse vs. focal/lobar on chest imaging	• Differences in morality, lung compliance, indirect lung injury, and plasma RAGE level	• Diffuse more likely to respond to PEEP	81–86
Genetic defined endotypes	Endotypes of ARDS defined by genetic variability that alters ARDS risk, outcome, or response to treatment	• Distinct ARDS risk, outcome, or response to treatment	• Therapies targeting biology implicated by genetic variants	88,89
Biomarker defined endotypes	Endotypes of ARDS defined by biomarker measurements	• Distinct ARDS risk, outcome, or response to treatment	• Therapies targeting biology implicated by biomarker elevation	109–119,124 130,131
Hyperinflammatory versus uninflamed	Endotypes of ARDS determined from unbiased latent class analysis and cluster analysis	• Hyperinflammatory characterized by elevated plasma inflammatory biomarkers, and higher mortality	• Phenotypes responded differently to PEEP and fluid strategy • Survival benefit observed in response to simvastatin in hyperinflammatory phenotype	142–144,147 148,151,152

The NEW ENGLAND JOURNAL of MEDICINE
N Engl J Med 2006;354:2564-75.

ORIGINAL ARTICLE

Comparison of Two Fluid-Management Strategies in Acute Lung Injury

Table 3. Main Outcome Variables.*

Outcome	Conservative Strategy	Liberal Strategy	P Value
Death at 60 days (%)	25.5	28.4	0.30
Ventilator-free days from day 1 to day 28†	14.6±0.5	12.1±0.5	<0.001
ICU-free days†			
Days 1 to 7	0.9±0.1	0.6±0.1	<0.001
Days 1 to 28	13.4±0.4	11.2±0.4	<0.001

Acute Respiratory Distress Syndrome Subphenotypes Respond Differently to Randomized Fluid Management Strategy

Katie R. Famous[1], Kevin Delucchi[2], Lorraine B. Ware[3,4], Kirsten N. Kangelaris[5], Kathleen D. Liu[6,7], B. Taylor Thompson[8], and Carolyn S. Calfee[1,7]; for the ARDS Network

Sous phénotype 1	Sous phénotype 2
• SDRA d'origine pulmonaire	• SDRA d'origine extra-pulmonaire
• Biomarqueurs inflammatoire • Acidose • Etat de choc • Vasopresseurs • Durée de VM	• Biomarqueurs inflammatoire + • Acidose + • Etat de choc + • Vasopresseurs + • Durée de VM +
• Mortalité J90 : 22 %	• Mortalité J90 : 45 % (p < 0,0001)

Fluid-management strategy	Subphenotype 1		Subphenotype 2		*P* Value
	Conservative (*n* = 349)	Liberal (*n* = 367)	Conservative (*n* = 142)	Liberal (*n* = 131)	
60-d mortality, %	24	17	39	49	0.0093
90-d mortality, %	26	18	40	50	0.0039
Ventilator-free days, median	17	21	5	0	0.35

Am J Respir Crit Care Med Vol 195, Iss 3, pp 331–338, Feb 1, 2017

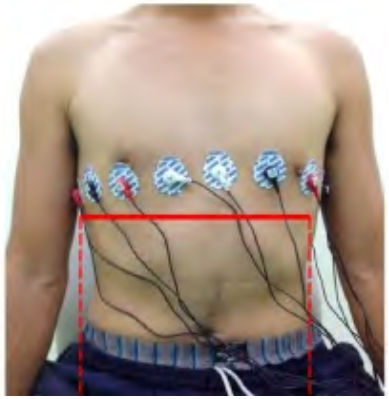
Bachmann *et al. Critical Care* (2018) 22:263
https://doi.org/10.1186/s13054-018-2195-6

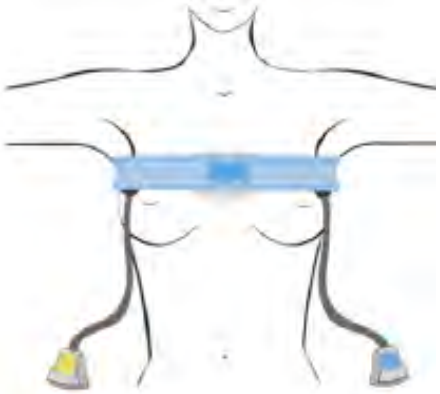
Critical Care

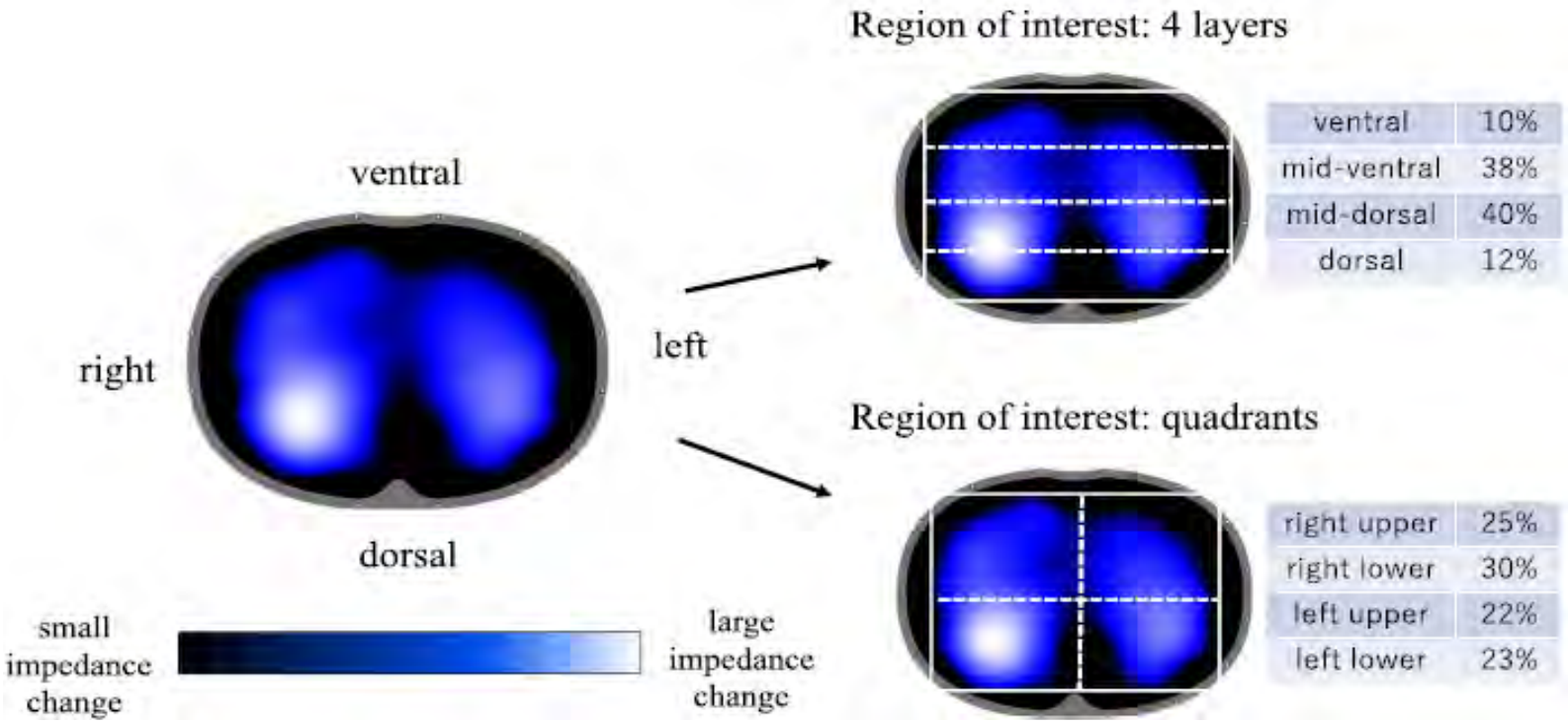
REVIEW Open Access

Electrical impedance tomography in acute respiratory distress syndrome

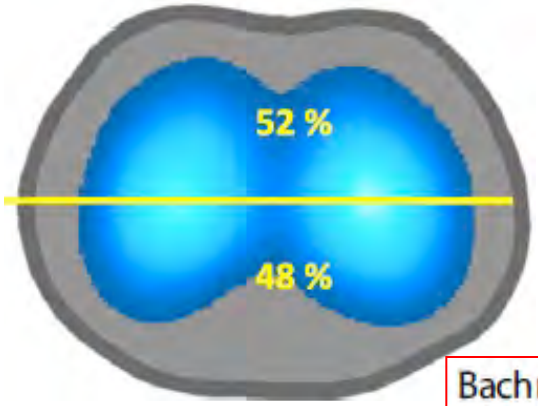
- Technique non invasive au lit du malade
- Pas rayon x
- 8-32 électrodes en circonférentiel
- Monitorage continu en temps réel
- Distribution de la ventilation pulmonaire
- ➔ Optimisation des paramètres ventilatoires





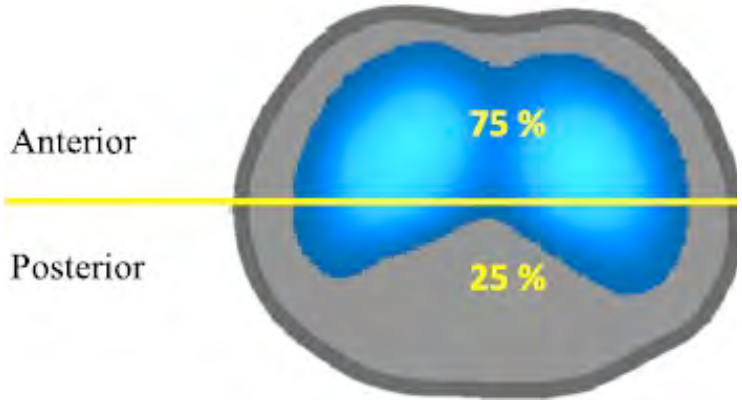


Shono and Kotani *Journal of Intensive Care* (2019) 7:4
https://doi.org/10.1186/s40560-019-0358-4

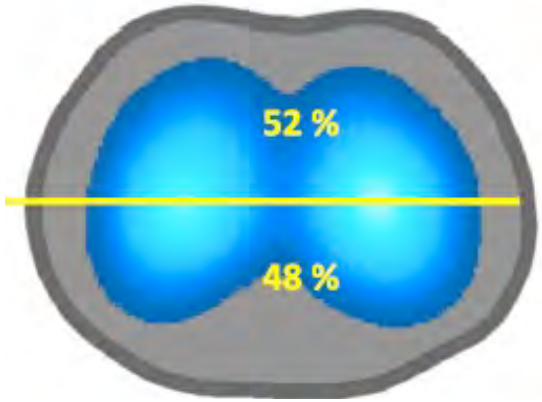


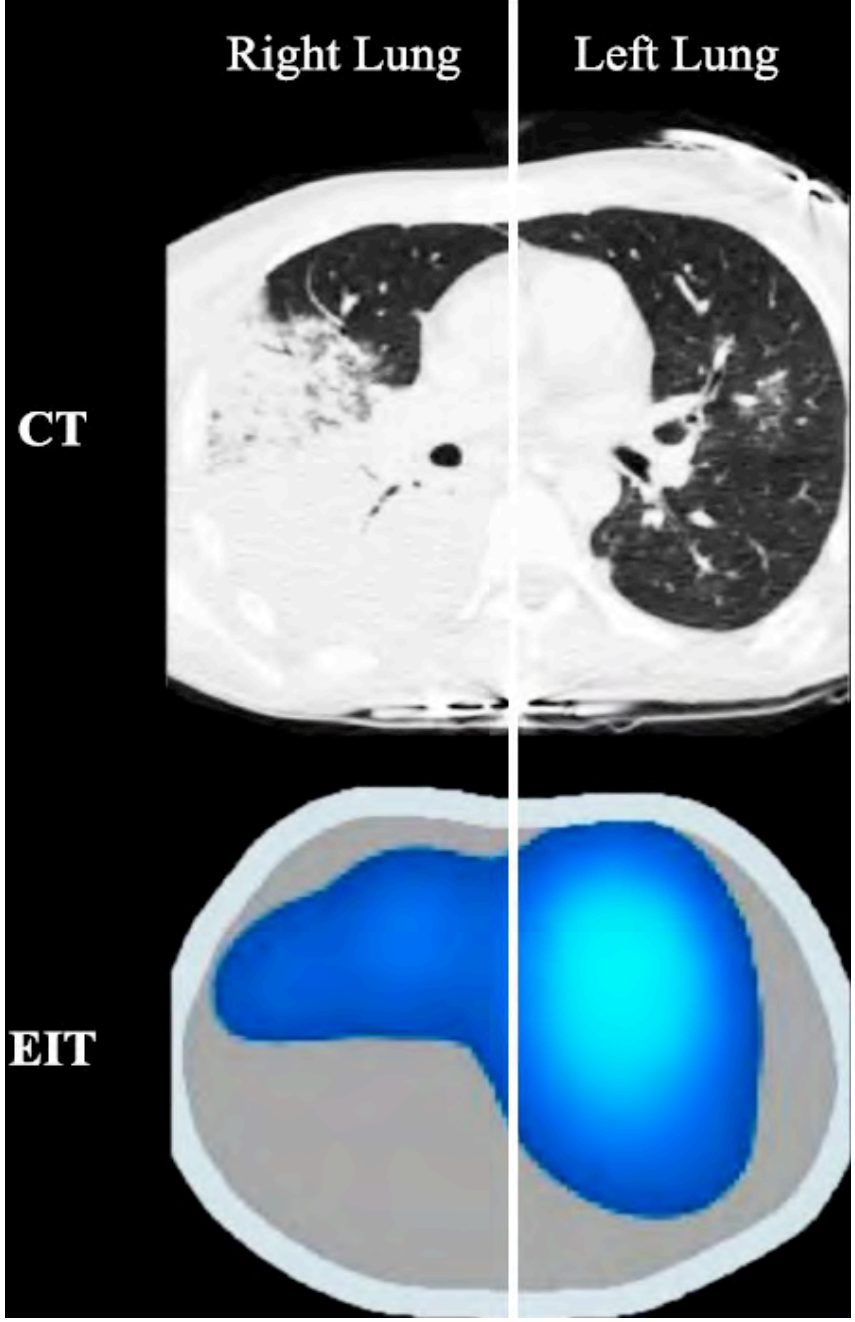
Bachmann *et al. Critical Care* (2018) 22:263
https://doi.org/10.1186/s13054-018-2195-6

PEEP = 5 cmH_2O



PEEP = 15 cmH_2O





Bachmann *et al. Critical Care* (2018) 22:263
https://doi.org/10.1186/s13054-018-2195-6

Recommandations Formalisées d'Experts

Prise en charge du Syndrome de Détresse Respiratoire Aigüe (SDRA) de l'adulte à la phase initiale

RFE sous l'égide de la SRLF

Société de Réanimation de Langue Française

Janvier 2019

Réglage du volume courant

R2.1.1 – Il faut utiliser un faible volume courant autour de 6 ml/kg de poids prédit par la taille (PPT) comme première approche pour les patients ayant des SDRA reconnus, en l'absence d'acidose métabolique sévère, y compris avec SDRA léger, dans le but de diminuer la mortalité.

GRADE 1+, ACCORD FORT

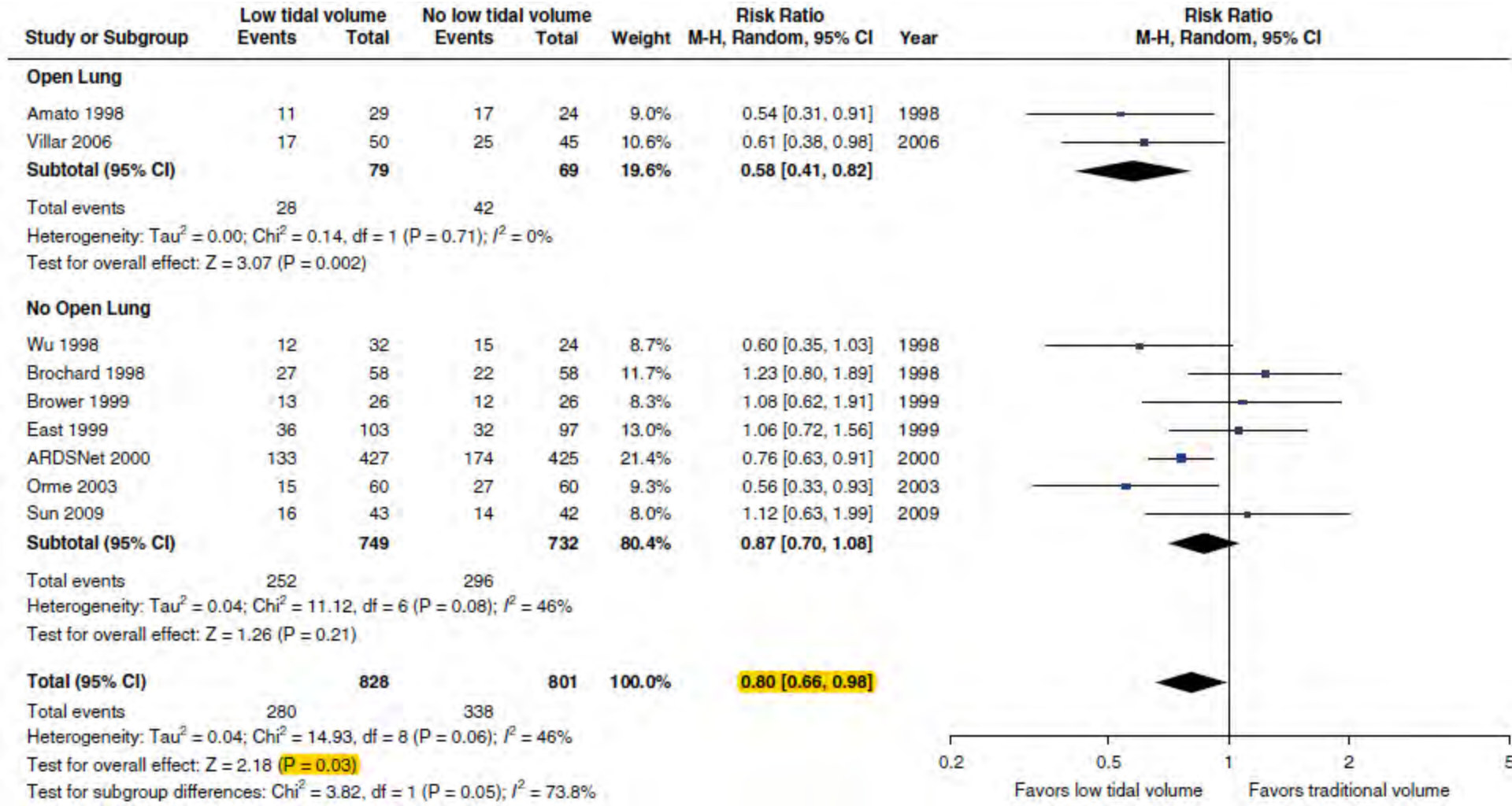
R2.1.2 – Les experts suggèrent une approche similaire pour tous les patients soumis à une ventilation mécanique invasive et sous sédation en réanimation étant donné le taux élevé de non-reconnaissance du SDRA et l'importance d'une protection pulmonaire appliquée rapidement.

AVIS D'EXPERTS

Low Tidal Volume versus Non–Volume-Limited Strategies for Patients with Acute Respiratory Distress Syndrome

A Systematic Review and Meta-Analysis

Ann Am Thorac Soc Vol 14, Supplement 4, pp S271–S279, Oct 2017



Ann Am Thorac Soc Vol 14, Supplement 4, pp S271–S279, Oct 2017

Pression de plateau

R2.2.1 – Une fois le Vt réglé autour de 6 ml/kg de poids prédit par la taille, il faut monitorer de façon continue la pression de plateau et faire en sorte qu'elle ne dépasse pas 30 cmH2O afin de réduire la mortalité.

GRADE 1+, ACCORD FORT

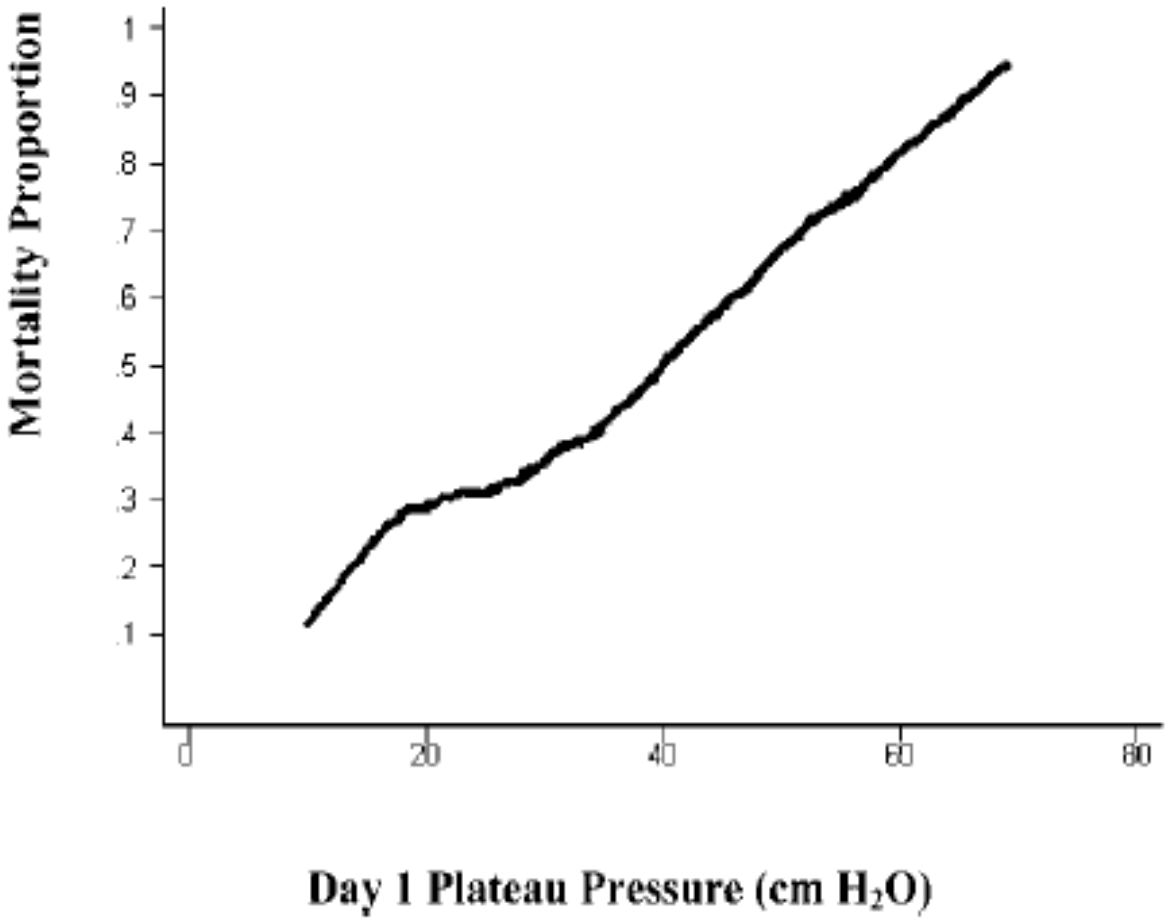
R2.2.2 - Les experts suggèrent de ne pas augmenter le Vt lorsque la pression de plateau est très inférieure à 30 cmH2O en dehors d'une hypercapnie importante persistant malgré la réduction de l'espace-mort instrumental et l'augmentation de la fréquence respiratoire.

AVIS D'EXPERTS

Critical Care Perspective

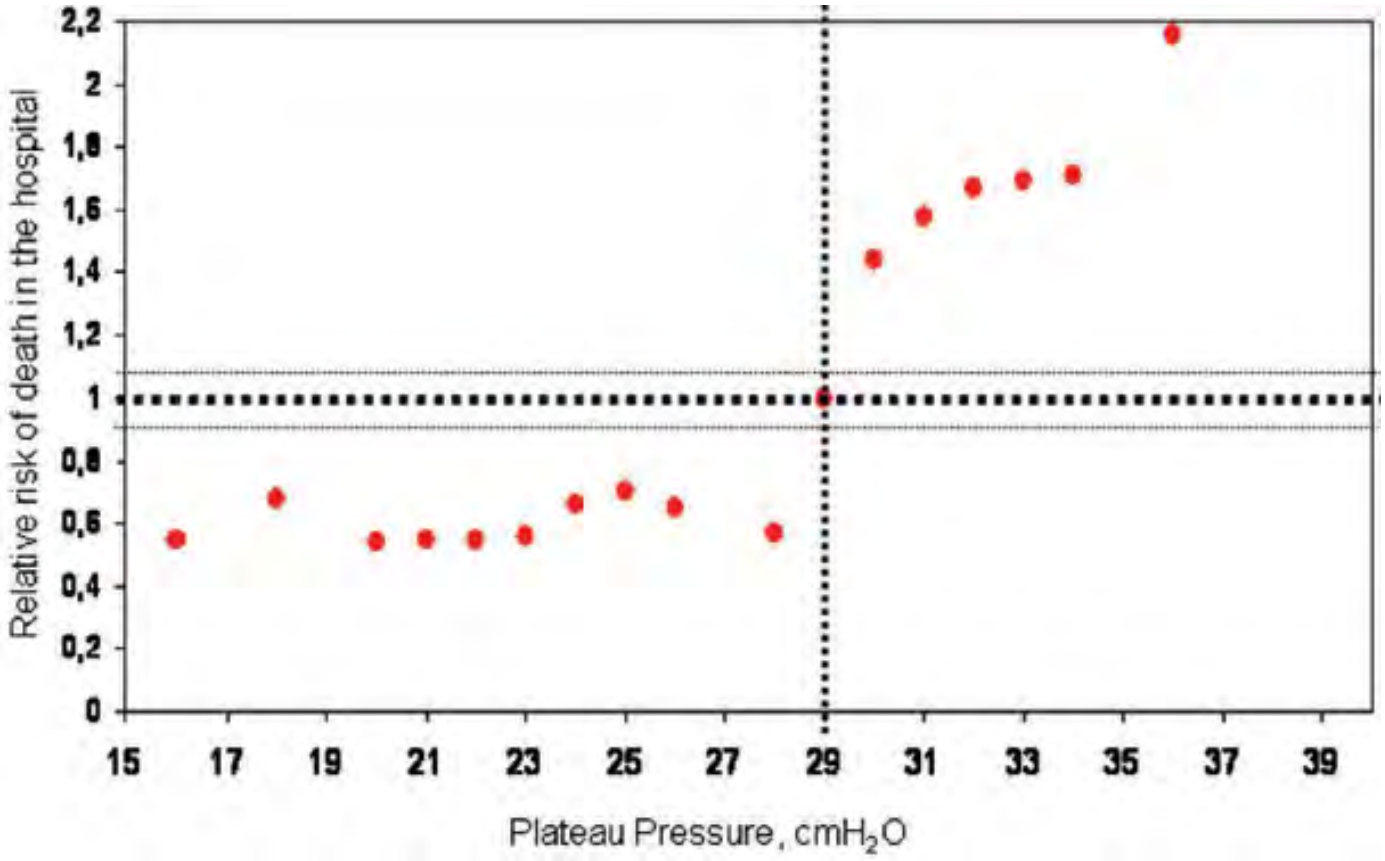
Tidal Volume Reduction in Patients with Acute Lung Injury When Plateau Pressures Are Not High

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

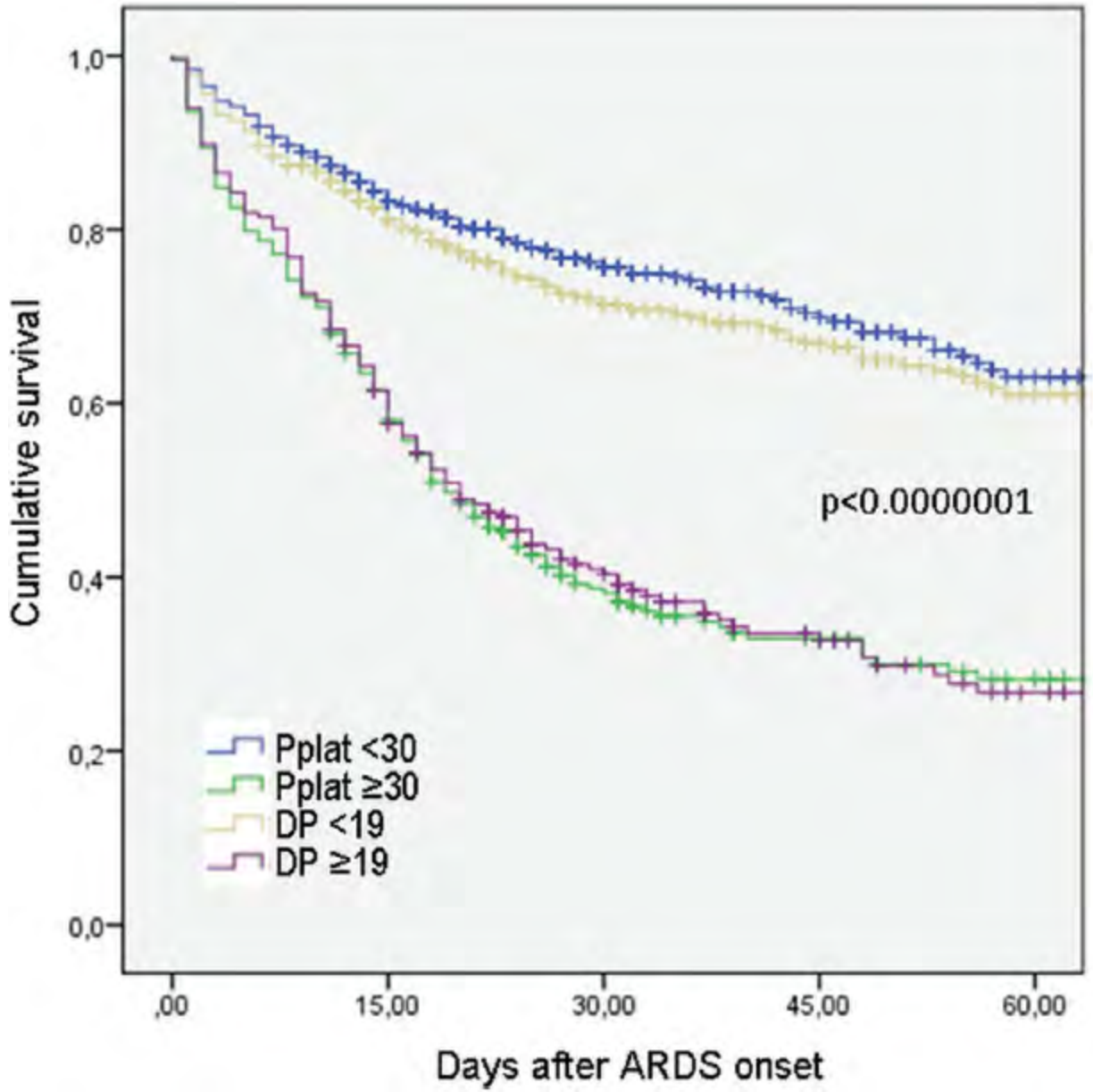


Am J Respir Crit Care Med Vol 172. pp 1241–1245, 2005

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



Crit Care Med. 2017 May;45(5):843-850.



Crit Care Med. 2017 May;45(5):843-850.

Pression expiratoire positive

R3.1.1 – La PEP est un élément indispensable à la prise en charge du SDRA. Les experts suggèrent d'utiliser une PEP supérieure à 5 cmH_2O chez tous les patients présentant un SDRA.

AVIS D'EXPERTS

R3.1.2 – Il faut probablement utiliser des niveaux élevés de PEP chez les patients atteints de SDRA modéré ou sévère mais pas chez les patients atteints de SDRA léger.

GRADE 2+, ACCORD FORT

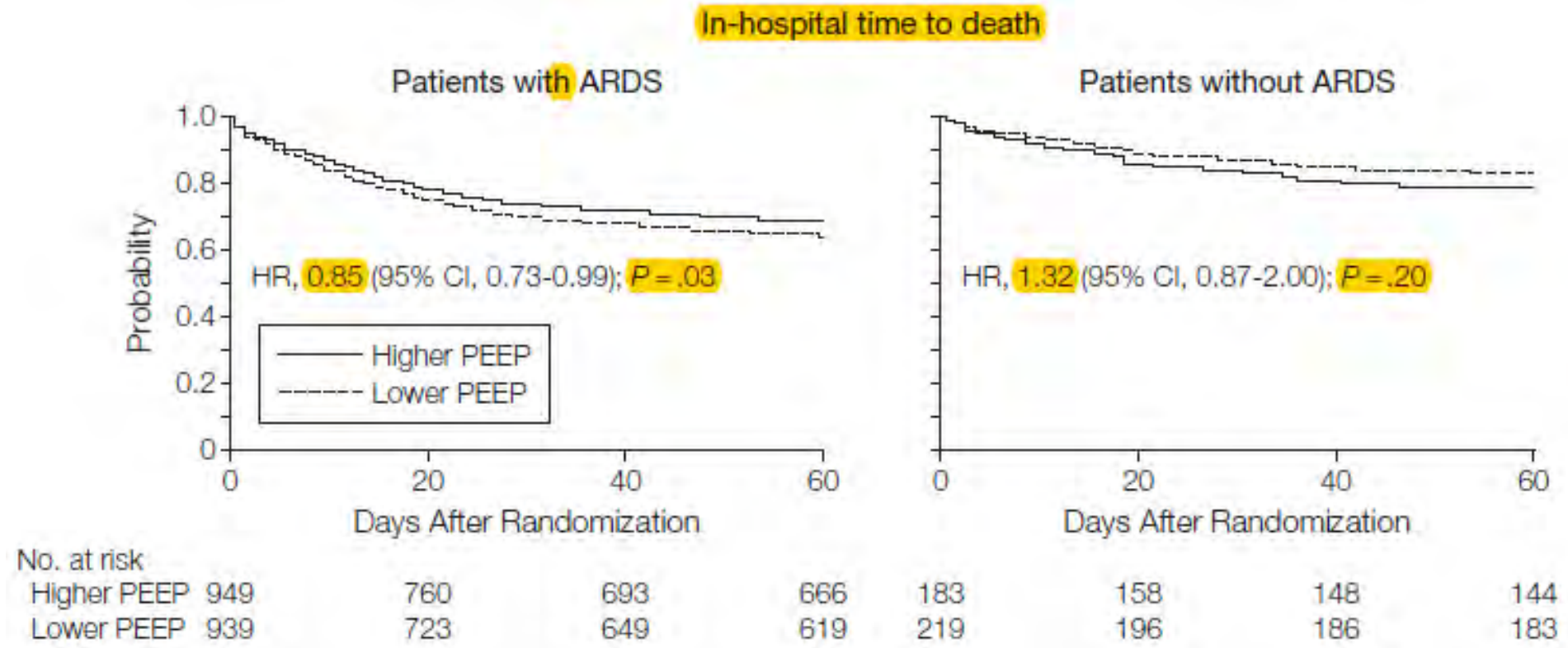
R3.1.3 –Les experts suggèrent de réserver les niveaux élevés de PEP aux patients chez qui ils induisent une amélioration de l'oxygénation sans dégradation marquée de la compliance du système respiratoire et de l'état hémodynamique. Le réglage de la PEP doit être individualisé.

AVIS D'EXPERTS

Higher vs Lower Positive End-Expiratory Pressure in Patients With Acute Lung Injury and Acute Respiratory Distress Syndrome

Systematic Review and Meta-analysis

JAMA. 2010;303(9):865-873



JAMA. 2010;303(9):865-873

Pression motrice

R2.3 – Aucune donnée ne permet d'émettre de recommandation sur un réglage du respirateur fondé uniquement sur la limitation de la pression motrice. Cette limitation peut être envisagée en complément de la limitation de la pression de plateau dans certains cas particuliers.

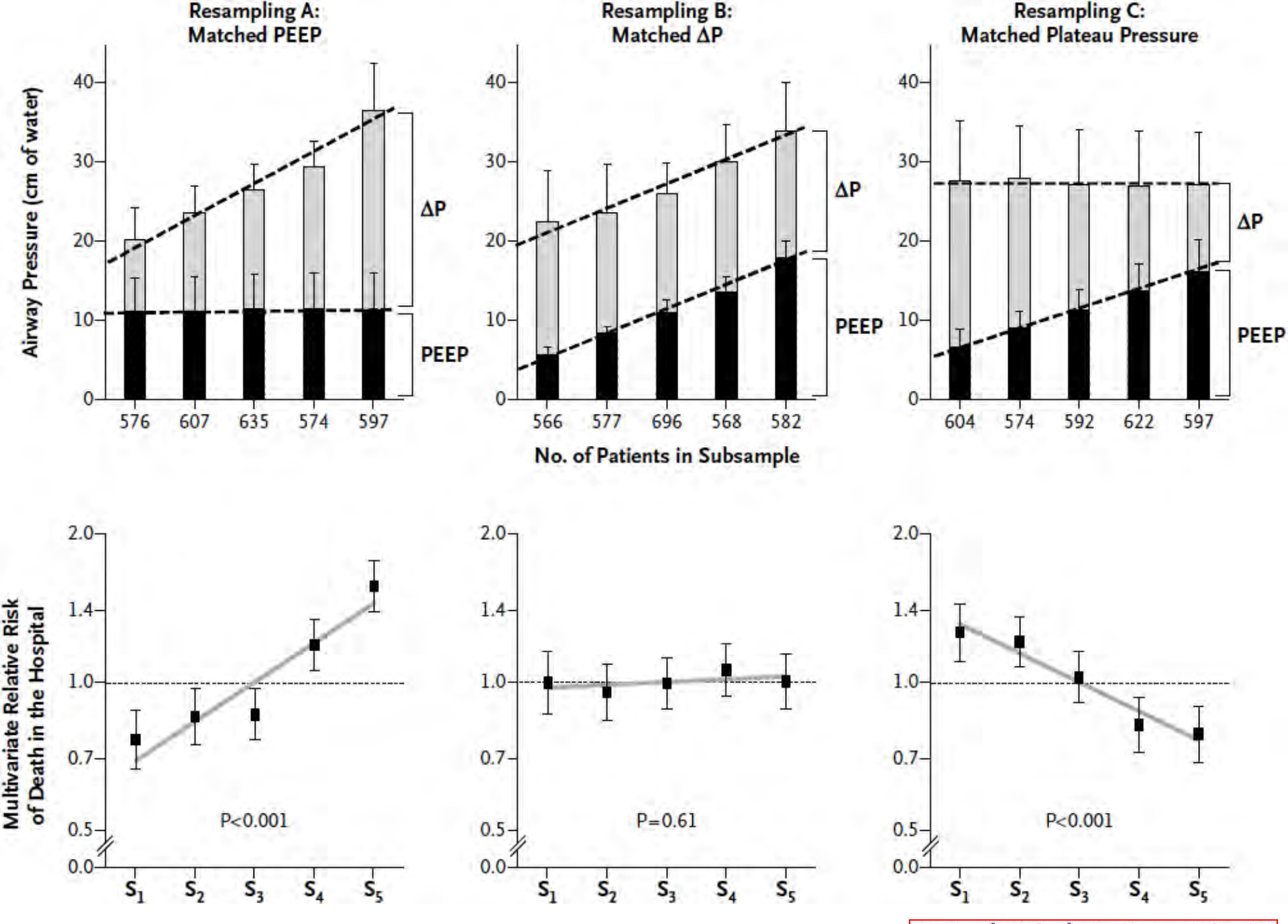
PAS DE RECOMMANDATION

SPECIAL ARTICLE

Driving Pressure and Survival in the Acute Respiratory Distress Syndrome

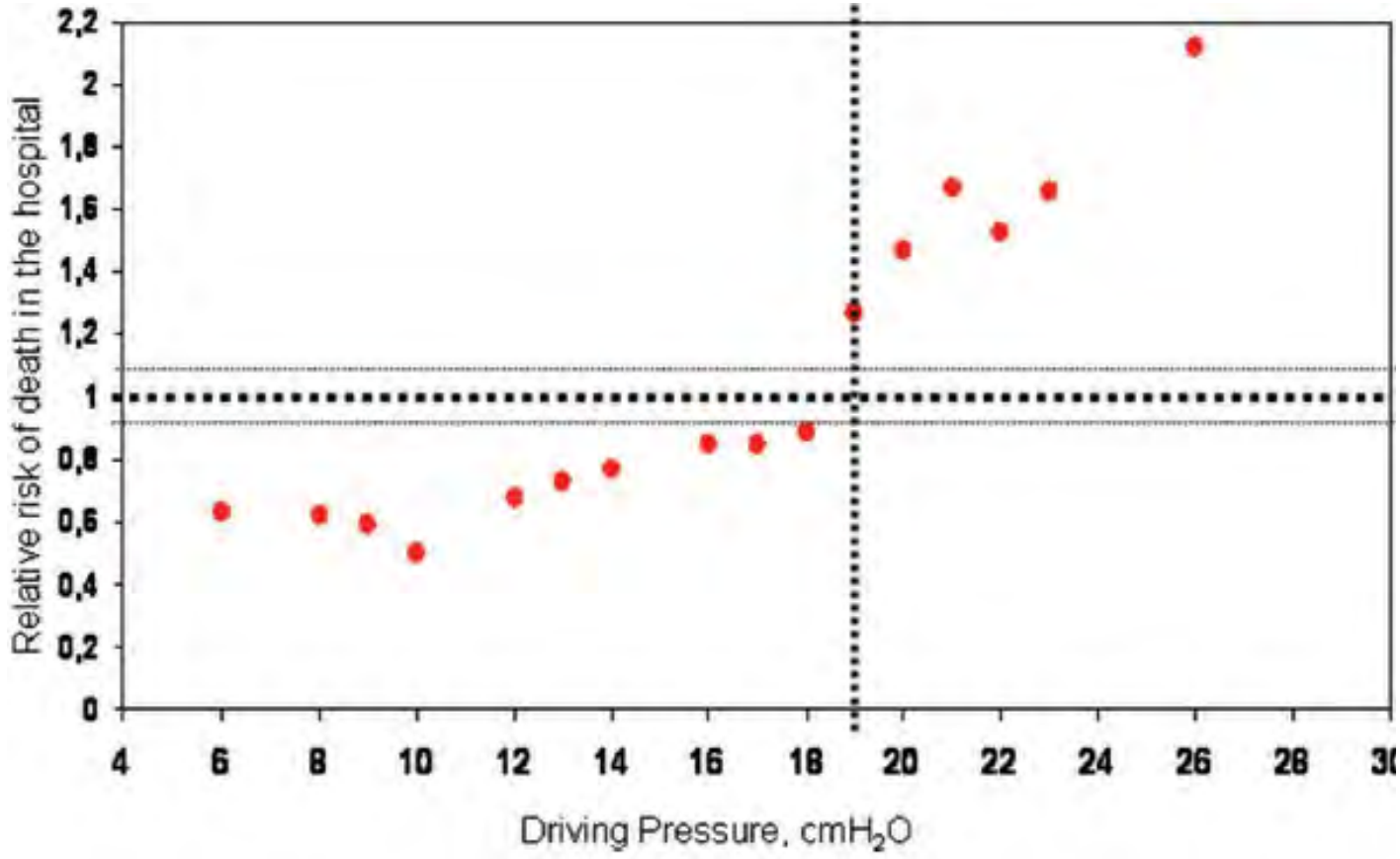
Marcelo B.P. Amato, M.D., Maureen O. Meade, M.D., Arthur S. Slutsky, M.D., Laurent Brochard, M.D., Eduardo L.V. Costa, M.D., David A. Schoenfeld, Ph.D., Thomas E. Stewart, M.D., Matthias Briel, M.D., Daniel Talmor, M.D., M.P.H., Alain Mercat, M.D., Jean-Christophe M. Richard, M.D., Carlos R.R. Carvalho,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

N Engl J Med 2015;372:747-55.

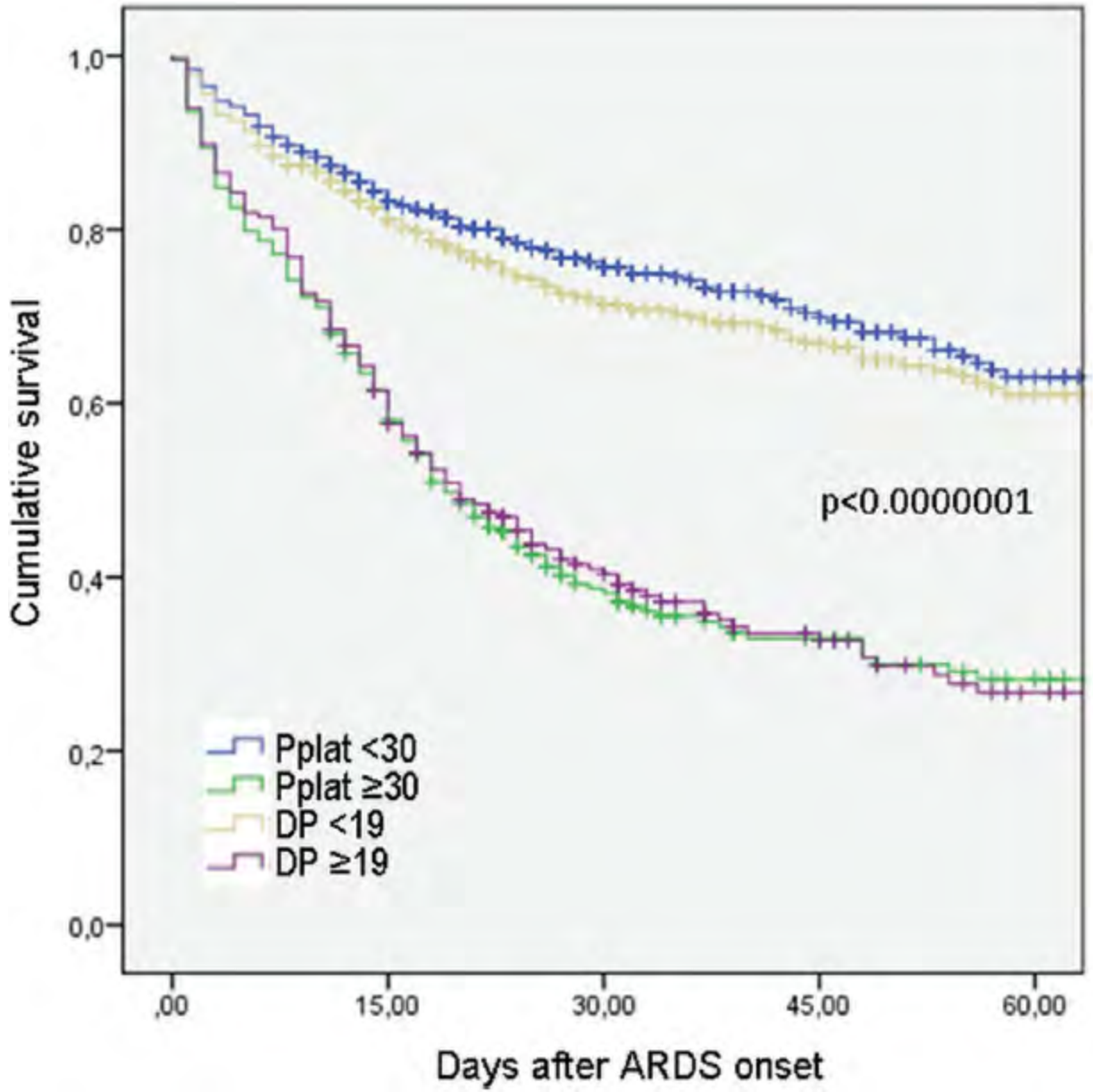


N Engl J Med 2015;372:747-55.

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

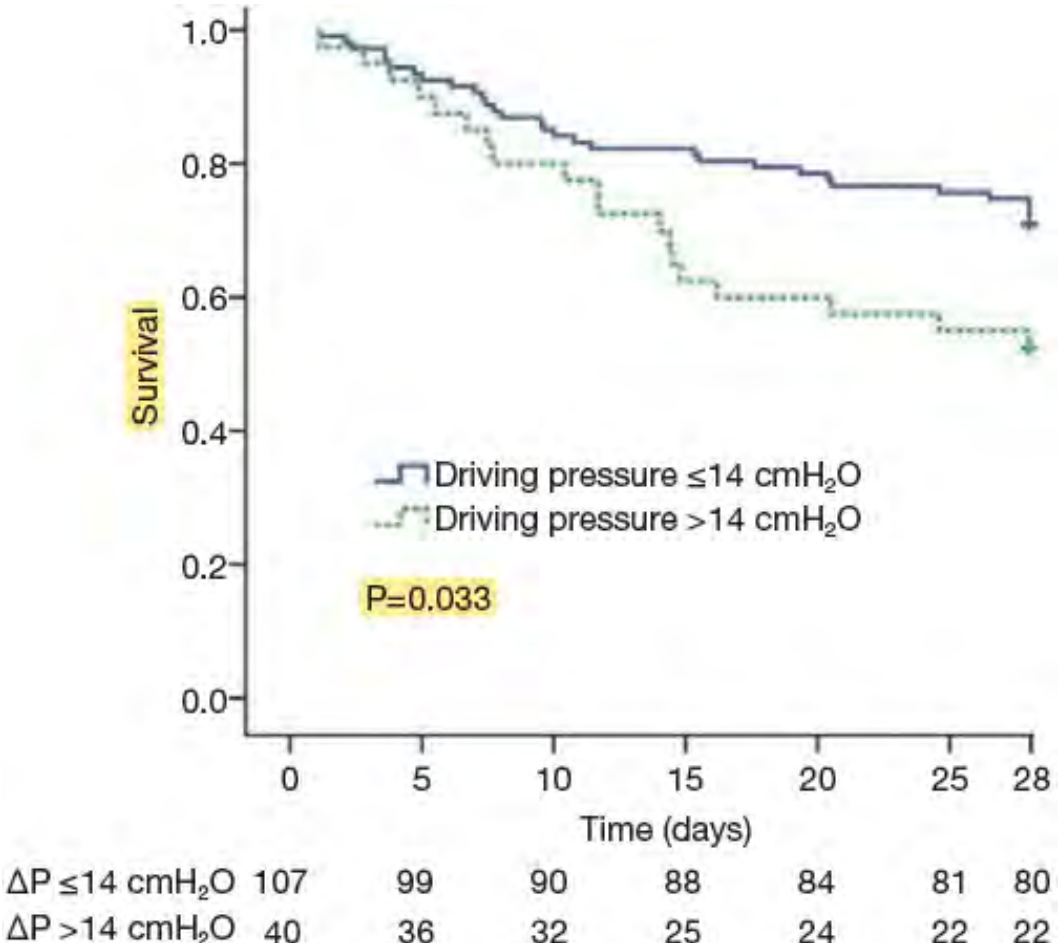


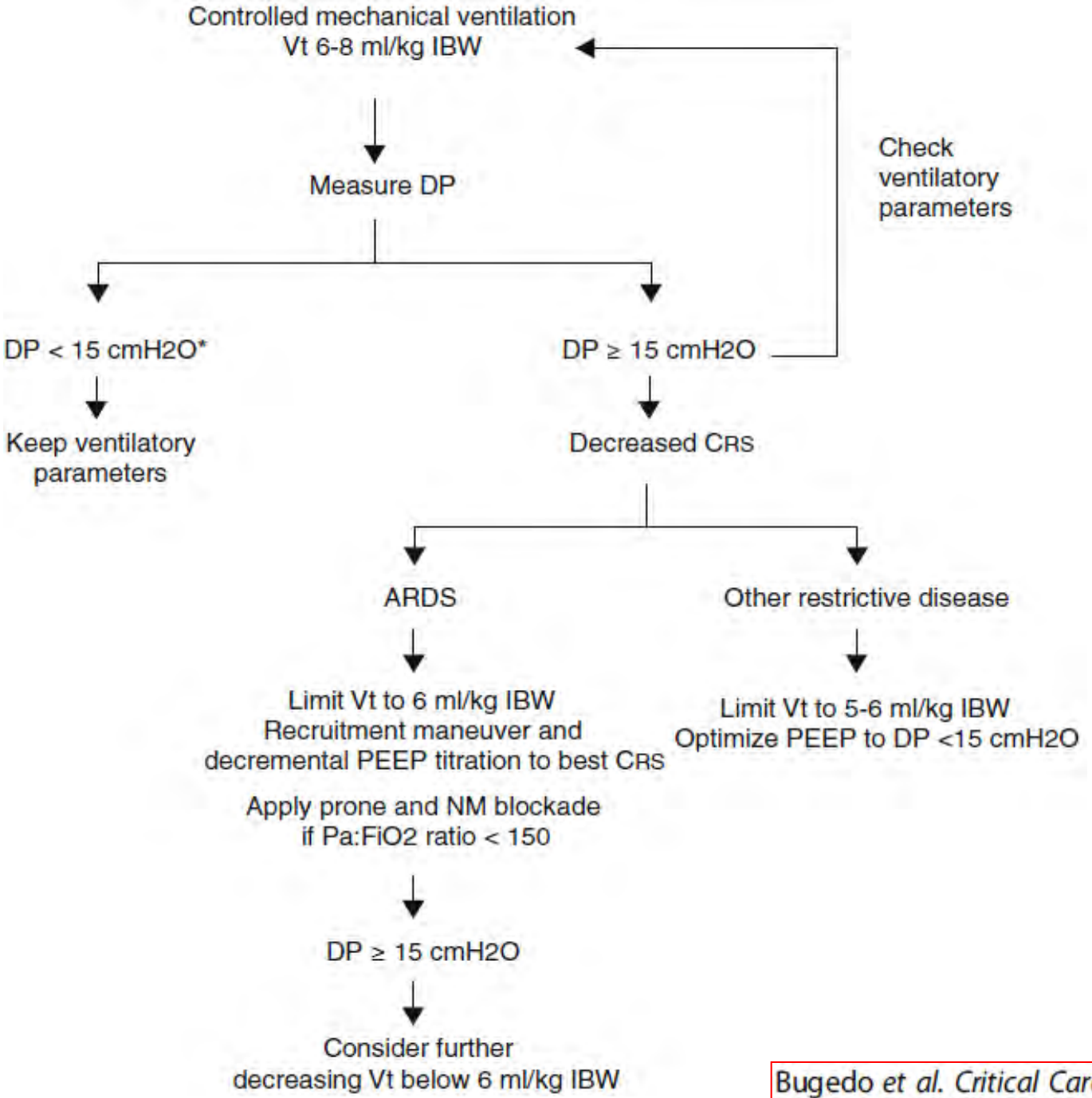
Crit Care Med. 2017 May;45(5):843-850.



Crit Care Med. 2017 May;45(5):843-850.

Practice of diagnosis and management of acute respiratory distress syndrome in mainland China: a cross-sectional study





Bugedo *et al. Critical Care* (2017) 21:199
DOI 10.1186/s13054-017-1779-x

Ventilation par oscillations à haute fréquence

R3.2. – Il ne faut pas utiliser la ventilation par oscillations à haute fréquence (HFOV) comme mode de ventilation chez des patients en SDRA.

GRADE 1-, ACCORD FORT

Cochrane Library

Cochrane Database of Systematic Reviews

High-frequency oscillatory ventilation versus conventional ventilation for acute respiratory distress syndrome (Review)

Cochrane Database of Systematic Reviews 2016, Issue 4. Art. No.: CD004085.
DOI: 10.1002/14651858.CD004085.pub4.

Analysis 1.1. Comparison 1 Mortality, Outcome 1 Hospital or 30-day mortality.

Review: High-frequency oscillatory ventilation versus conventional ventilation for acute respiratory distress syndrome

Comparison: 1 Mortality

Outcome: 1 Hospital or 30-day mortality

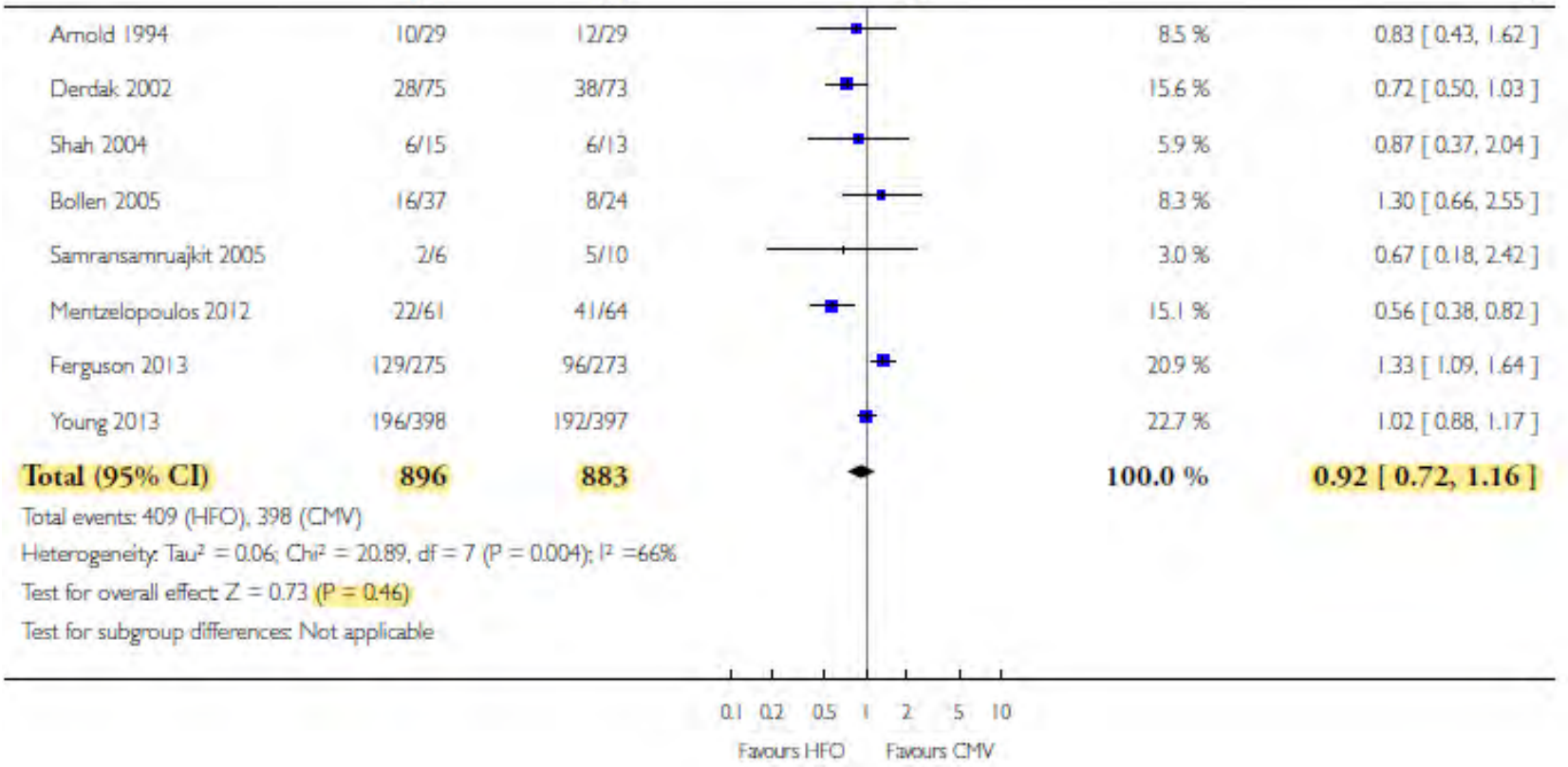
Study or subgroup	HFO n/N	CMV n/N	Risk Ratio M-H,Random,95% CI	Weight	Risk Ratio M-H,Random,95% CI
Arnold 1994	10/29	12/29		8.5 %	0.83 [0.43, 1.62]
Derdak 2002	28/75	38/73		15.6 %	0.72 [0.50, 1.03]
Shah 2004	6/15	6/13		5.9 %	0.87 [0.37, 2.04]
Bollen 2005	16/37	8/24		8.3 %	1.30 [0.66, 2.55]
Samransamruajkit 2005	2/6	5/10		3.0 %	0.67 [0.18, 2.42]
Mentzelopoulos 2012	22/61	41/64		15.1 %	0.56 [0.38, 0.82]
Ferguson 2013	129/275	96/273		20.9 %	1.33 [1.09, 1.64]
Young 2013	196/398	192/397		22.7 %	1.02 [0.88, 1.17]
Total (95% CI)	**896**	**883**		**100.0 %**	**0.92 [0.72, 1.16]**

Total events: 409 (HFO), 398 (CMV)

Heterogeneity: $Tau^2 = 0.06$; $Chi^2 = 20.89$, df = 7 (P = 0.004); I^2 =66%

Test for overall effect: Z = 0.73 (P = 0.46)

Test for subgroup differences: Not applicable



Cochrane Database of Systematic Reviews 2016, Issue 4. Art. No.: CD004085.
DOI: 10.1002/14651858.CD004085.pub4.

Manœuvres de recrutement systématiques

R3.3 – Il ne faut probablement pas faire de manœuvres de recrutement systématiques dans le SDRA.

GRADE 2-, ACCORD FORT

RESEARCH Open Access

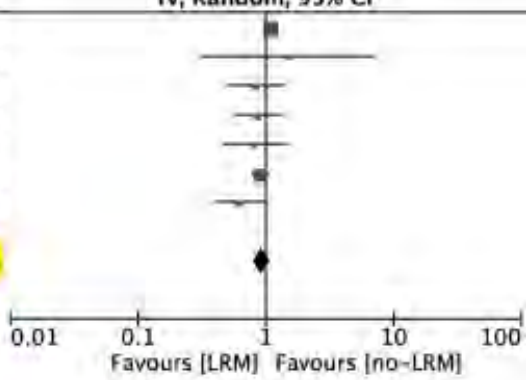
Recruitment maneuver does not provide any mortality benefit over lung protective strategy ventilation in adult patients with acute respiratory distress syndrome: a meta-analysis and systematic review of the randomized controlled trials

Bhattacharjee *et al. Journal of Intensive Care* (2018) 6:35
https://doi.org/10.1186/s40560-018-0305-9

Mortalité après sortie

Study or Subgroup	LRM Events	LRM Total	no-LRM Events	no-LRM Total	Weight	Risk Ratio IV, Random, 95% CI
ART 2017	327	501	305	509	37.4%	1.09 [0.99, 1.20]
Hodgson 2011	3	10	2	10	0.9%	1.50 [0.32, 7.14]
Huh 2009	14	30	15	27	7.4%	0.84 [0.50, 1.40]
Kacmarek 2016	28	99	33	101	10.0%	0.87 [0.57, 1.32]
Liu 2011	14	50	17	50	5.8%	0.82 [0.46, 1.48]
Meade 2008	173	475	205	508	29.6%	0.90 [0.77, 1.06]
Xi 2010	18	55	29	55	8.9%	0.62 [0.39, 0.98]
Total (95% CI)		**1220**		**1260**	**100.0%**	**0.93 [0.80, 1.08]**
Total events	577		606			

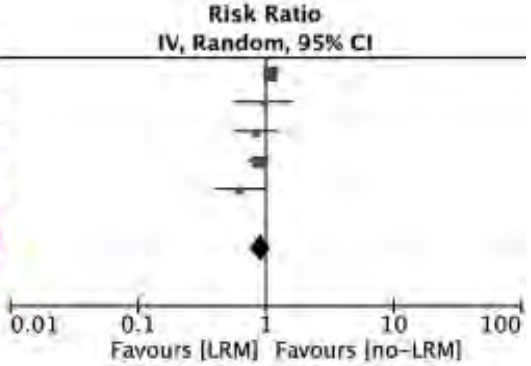
Heterogeneity: $Tau^2 = 0.01$; $Chi^2 = 10.46$, df = 6 (P = 0.11); $I^2 = 43\%$
Test for overall effect: Z = 0.98 (P = 0.33)



Mortalité en réanimation

Study or Subgroup	LRM Events	LRM Total	no-LRM Events	no-LRM Total	Weight	Risk Ratio IV, Random, 95% CI
ART 2017	303	500	284	509	36.3%	1.09 [0.98, 1.21]
Huh 2009	14	30	13	27	8.8%	0.97 [0.56, 1.68]
Kacmarek 2016	29	99	35	101	13.6%	0.85 [0.56, 1.27]
Meade 2008	145	475	178	508	29.5%	0.87 [0.73, 1.04]
Xi 2010	18	55	29	55	11.7%	0.62 [0.39, 0.98]
Total (95% CI)		**1159**		**1200**	**100.0%**	**0.91 [0.76, 1.10]**
Total events	509		539			

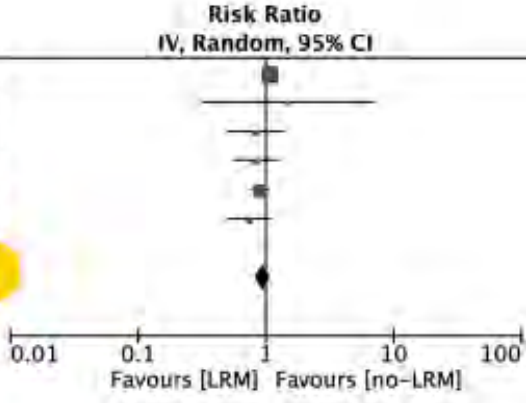
Heterogeneity: $Tau^2 = 0.02$; $Chi^2 = 9.55$, df = 4 (P = 0.05); $I^2 = 58\%$
Test for overall effect: Z = 0.98 (P = 0.33)



Mortalité hospitalière

Study or Subgroup	LRM Events	LRM Total	no-LRM Events	no-LRM Total	Weight	Risk Ratio IV, Random, 95% CI
ART 2017	319	500	301	508	43.9%	1.08 [0.98, 1.19]
Hodgson 2011	3	10	2	10	0.7%	1.50 [0.32, 7.14]
Huh 2009	14	30	15	27	5.9%	0.84 [0.50, 1.40]
Kacmarek 2016	29	99	35	101	8.8%	0.85 [0.56, 1.27]
Meade 2008	173	475	205	508	31.3%	0.90 [0.77, 1.06]
Xi 2010	23	55	31	55	9.4%	0.74 [0.50, 1.09]
Total (95% CI)		**1169**		**1209**	**100.0%**	**0.95 [0.83, 1.08]**
Total events	561		589			

Heterogeneity: $Tau^2 = 0.01$; $Chi^2 = 7.42$, df = 5 (P = 0.19); $I^2 = 33\%$
Test for overall effect: Z = 0.75 (P = 0.45)



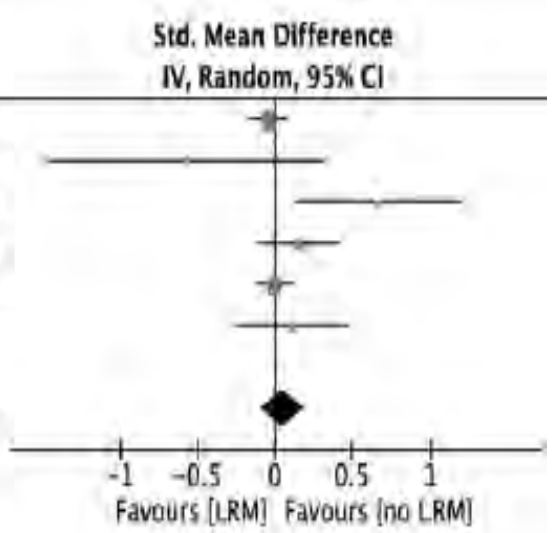
Bhattacharjee et al. Journal of Intensive Care (2018) 6:35
https://doi.org/10.1186/s40560-018-0305-9

Durée d'hospitalisation en réanimation

Study or Subgroup	LRM Mean	LRM SD	LRM Total	no-LRM Mean	no-LRM SD	no-LRM Total	Weight	Std. Mean Difference IV, Random, 95% CI
ART 2017	18.2	22.4	501	19.2	25.4	509	32.6%	-0.04 [-0.17, 0.08]
Hodgson 2011	9.9	9.2	10	16	11.2	10	2.3%	-0.57 [-1.47, 0.33]
Huh 2009	25.1	5.6	30	21.4	5.3	27	5.9%	0.67 [0.13, 1.20]
Kacmarek 2016	18	13.3	99	16	12.6	101	16.1%	0.15 [-0.12, 0.43]
Meade 2008	13	11.1	475	13	10.4	508	32.4%	0.00 [-0.13, 0.13]
Xi 2010	22.5	22.2	55	19.8	24.8	55	10.6%	0.11 [-0.26, 0.49]
Total (95% CI)			**1170**			**1210**	**100.0%**	**0.05 [-0.09, 0.19]**

Heterogeneity: $Tau^2 = 0.01$; $Chi^2 = 9.44$, df = 5 (P = 0.09); $I^2 = 47\%$

Test for overall effect: Z = 0.70 (P = 0.49)

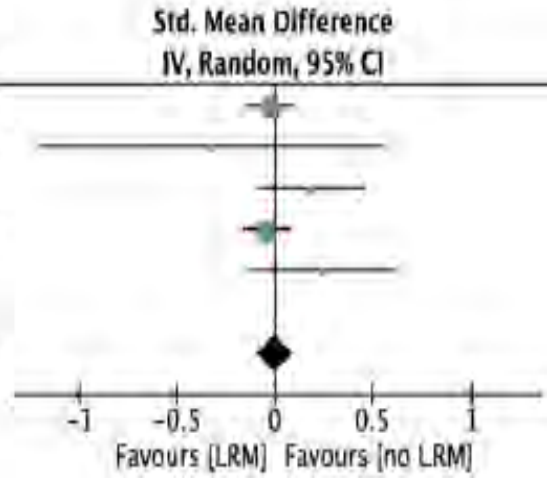


Durée d'hospitalisation globale

Study or Subgroup	LRM Mean	LRM SD	LRM Total	no-LRM Mean	no-LRM SD	no-LRM Total	Weight	Std. Mean Difference IV, Random, 95% CI
ART 2017	25.5	32.3	501	26.2	31.7	509	41.8%	-0.02 [-0.15, 0.10]
Hodgson 2011	17.9	20.8	10	24.7	19.3	10	1.1%	-0.32 [-1.21, 0.56]
Kacmarek 2016	27	22.2	99	23	20	101	10.3%	0.19 [-0.09, 0.47]
Meade 2008	28	22.9	475	29	25.9	508	40.9%	-0.04 [-0.17, 0.08]
Xi 2010	43.2	45.6	55	33.2	34	55	5.8%	0.25 [-0.13, 0.62]
Total (95% CI)			**1140**			**1183**	**100.0%**	**0.00 [-0.09, 0.10]**

Heterogeneity: $Tau^2 = 0.00$; $Chi^2 = 4.48$, df = 4 (P = 0.34); $I^2 = 11\%$

Test for overall effect: Z = 0.10 (P = 0.92)



Bhattacharjee et al. Journal of Intensive Care (2018) 6:35
https://doi.org/10.1186/s40560-018-0305-9

Curarisation précoce et de courte durée

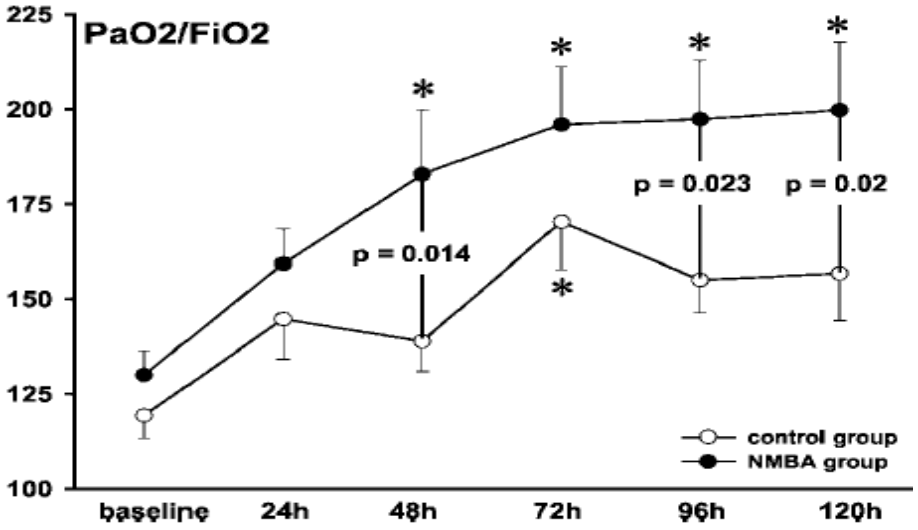
R4.1 – Il faut utiliser un curare en cas de SDRA avec rapport PaO2/FiO2 < 150 mm Hg afin de réduire la mortalité.

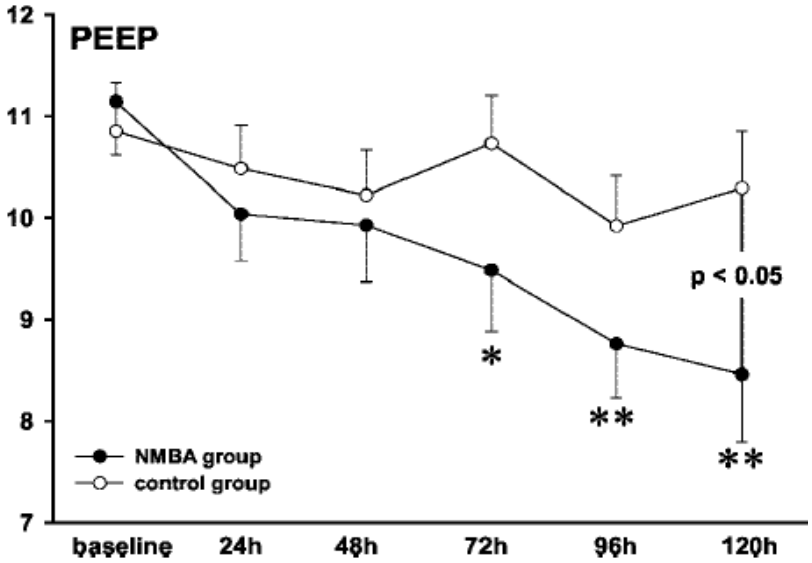
Le curare doit être administré de manière précoce (dans les 48h après le début du SDRA), en perfusion continue, pour une durée maximale de 48 h avec réévaluation au moins quotidienne.

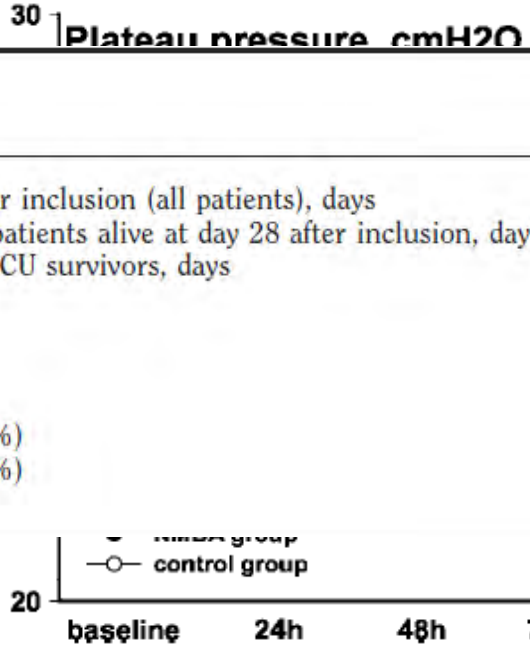
GRADE 1+, ACCORD FORT

Bénéfices potentiels	Références
• Diminution réaction inflammatoire • Diminution consommation O_2 • Diminution débit cardiaque • Augmentation recrutement alvéolaire • Augmentation PvO_2 et PaO_2	• N Engl J Med 2010;363:1176-80 • Intensive CareMed 2015;41:2201-3 • Curr Opin Crit Care 2012;18:495-502
• Amélioration des pressions transpulmonaires • Homogénéisation de l'inflation régionale • Augmentation recrutement alvéolaire • Diminution activité musculaire respiratoire	Intensive Care Med 2017;43:408-18
• Diminution DC ➔ Diminution œdème alv • Diminution asynchronisme ➔ ↓ surdistension ➔ ↓ atelectraumatisme, baro et volotraumatisme	Crit Care Med 1990;18:103-13

Effect of neuromuscular blocking agents on gas exchange in patients presenting with acute respiratory distress syndrome*



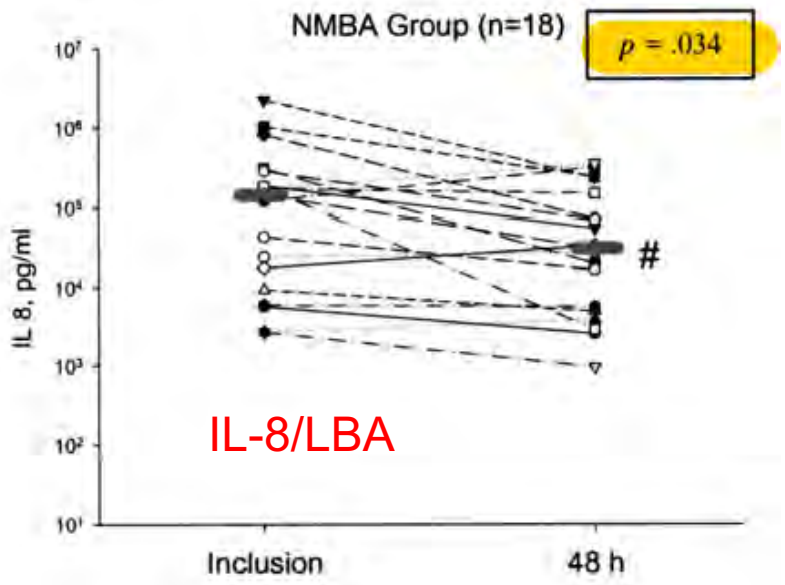




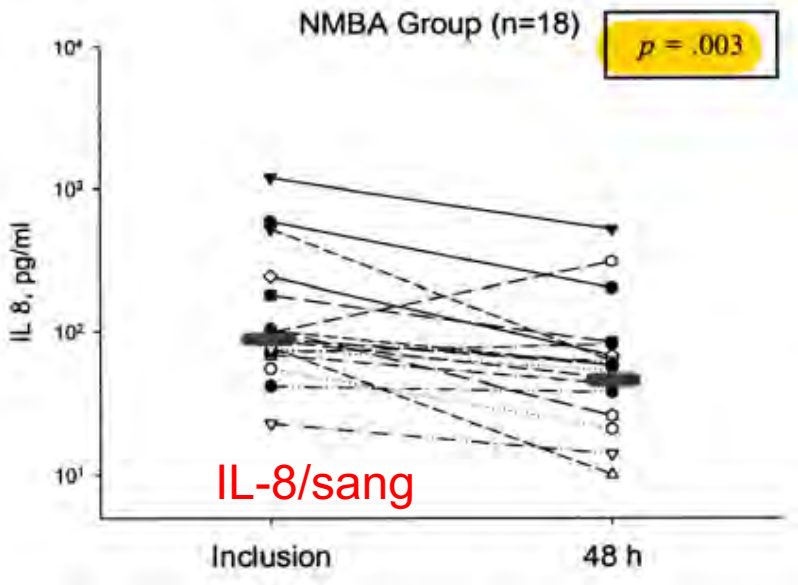
	NMBA Group (n = 28)	Control Group (n = 28)	*p* Value
Duration of mechanical ventilation after inclusion (all patients), days	20.9 ± 15.0	21.2 ± 17.4	.94
Duration of mechanical ventilation in patients alive at day 28 after inclusion, days	27.4 ± 14.2	34.1 ± 20.3	.30
Duration of mechanical ventilation in ICU survivors, days	24.5 ± 13.1	26.4 ± 13.9	.76
VFD at day 28, days	3.7 ± 7.2	1.7 ± 5.3	.24
Median (25th–75th percentiles)	0 (0–5)	0 (0–0)	.24
VFD at day 60, days	19.0 ± 20.3	9.8 ± 16.9	.071
Median (25th–75th percentiles)	14 (0–37)	0 (0–18)	.11
Mortality at day 28 after inclusion, n (%)	10 (35.7)	17 (60.7)	.061
Mortality at day 60 after inclusion, n (%)	13 (46.4)	18 (64.3)	.18
ICU mortality, n (%)	13 (46.4)	20 (71.4)	.057

...it Care Med 2004; 32:113–119

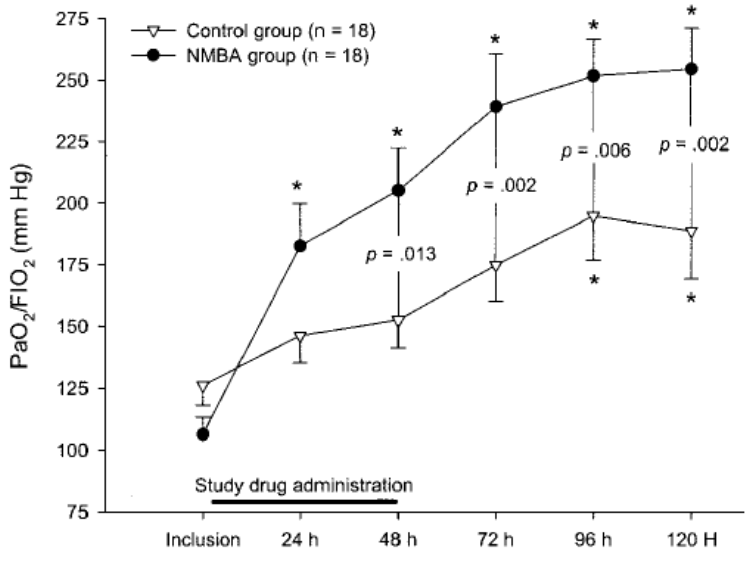
Neuromuscular blocking agents decrease inflammatory response in patients presenting with acute respiratory distress syndrome*



IL-8/LBA



IL-8/sang



Crit Care Med 2006; 34:2749–2757

Neuromuscular Blockers in Early Acute Respiratory Distress Syndrome

N Engl J Med 2010;363:1107-16.

Table 3. Secondary Outcomes, According to Study Group.*

Outcome	Cisatracurium (N=177)	Placebo (N=162)	Relative Risk with Cisatracurium (95% CI)	P Value
Death — no. (% [95% CI])				
At 28 days	42 (23.7 [18.1–30.5])	54 (33.3 [26.5–40.9])	0.71 (0.51–1.00)	0.05
In the ICU	52 (29.4 [23.2–36.5])	63 (38.9 [31.7–46.6])	0.76 (0.56–1.02)	0.06
In the hospital	57 (32.2 [25.8–39.4])	67 (41.4 [34.1–49.1])	0.78 (0.59–1.03)	0.08
No. of ventilator-free days†				
From day 1 to day 28	10.6±9.7	8.5±9.4		0.04
From day 1 to day 90	53.1±35.8	44.6±37.5		0.03
No. of days without organ failure, from day 1 to day 28				
No cardiovascular failure	18.3±9.4	16.6±10.4		0.12
No coagulation abnormalities	22.6±8.9	20.5±9.9		0.05
No hepatic failure	21.3±9.6	19.1±10.6		0.05
No renal failure	20.5±10.1	18.1±11.6		0.05
None of the four	15.8±9.9	12.2±11.1		0.01
No. of days outside the ICU				
From day 1 to day 28	6.9±8.2	5.7±7.8		0.16
From day 1 to day 90	47.7±33.5	39.5±35.6		0.03
Hospital survivors admitted to other health care facilities from day 1 to day 90 — % (95% CI)	22.3 (15.8–30.5)	18.8 (12.2–27.8)		0.52
Barotrauma — no. (% [95% CI])‡	9 (5.1 [2.7–9.4])	19 (11.7 [7.6–17.6])	0.43 (0.20–0.93)	0.03
Pneumothorax — no. (% [95% CI])	7 (4.0 [2.0–8.0])	19 (11.7 [7.6–17.6])	0.34 (0.15–0.78)	0.01
MRC score — median (IQR)§				
At day 28	55 (46–60)	55 (39–60)	1.07 (0.80–1.45)	0.49
At ICU discharge	55 (43–60)	55 (44–60)	0.92 (0.71–1.19)	0.94
Patients without ICU-acquired paresis¶				
By day 28 — no./total no. (% [95% CI])	68/96 (70.8 [61.1–79.0])	52/77 (67.5 [56.5–77.0])		0.64
By ICU discharge — no./total no. (% [95% CI])	72/112 (64.3 [55.1–72.6])	61/89 (68.5 [58.3–77.3])		0.51

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MAY 23, 2019 VOL. 380 NO. 21

Early Neuromuscular Blockade in the Acute Respiratory Distress Syndrome

Mortalité hospitalière: 42,5 % vs 42,8%; p=0,9

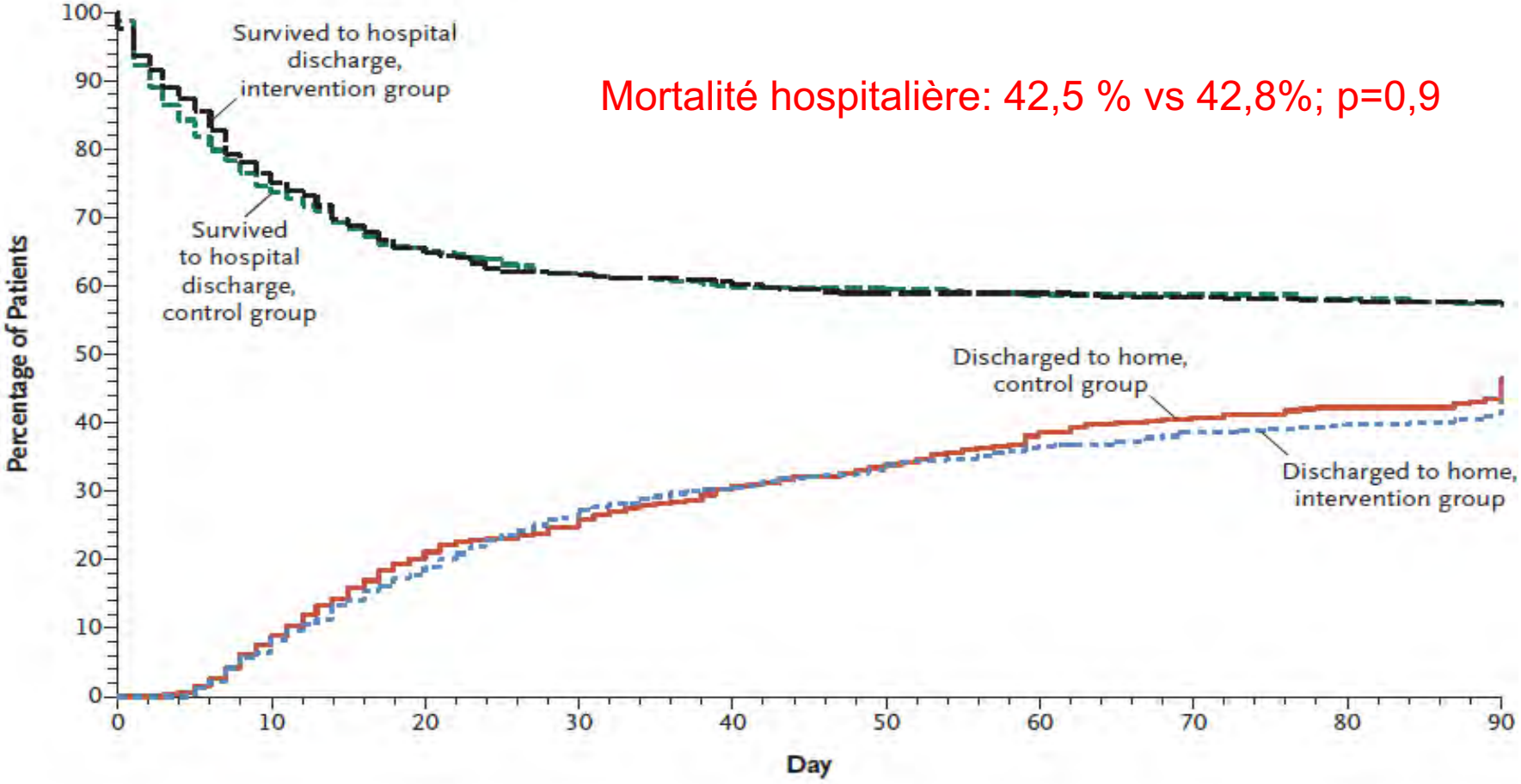


Figure 3. Patients Who Survived to Hospital Discharge and Were Discharged Home during the First 90 Days after Randomization.

Ventilation spontanée précoce

R4.2.1 – Il n'est pas possible d'émettre de recommandation sur une stratégie de ventilation spontanée (VS) systématique à la phase aigüe du SDRA.

PAS DE RECOMMANDATION

R4.2.2 - Après la phase aiguë du SDRA, les experts suggèrent qu'un mode ventilatoire en pression autorisant la ventilation spontanée puisse être utilisé en s'assurant que le volume courant généré avoisine 6 ml/kg PPT sans dépasser 8 ml/kg PPT.

AVIS D'EXPERTS

Intensive Care Med (2017) 43:1648–1659
DOI 10.1007/s00134-017-4912-z

CrossMark

ORIGINAL

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Early application of airway pressure release ventilation may reduce the duration of mechanical ventilation in acute respiratory distress syndrome

Variable	Baseline			Day 3 after enrollment [c,d]		
	APRV	LTV	*P* value	APRV	LTV	*P* value
No. of patients	71	67		62	56	
Respiratory variables						
Ventilator setting (tidal volume in mL)	437.8 ± 40.6	429.6 ± 47.5	0.277	_	423.8 ± 51.8	
Ventilator setting (tidal volume in mL/kg of predicted body weight)	7.2 ± 0.7	7.1 ± 0.7	0.534	_	7.0 ± 1	
Ventilator monitoring (tidal volume in mL)	466.6 ± 54.9	461.2 ± 59.7	0.578	476.9 ± 111.3	461.8 ± 64.1	0.364
Ventilator monitoring (tidal volume in mL/kg of predicted body weight)	7.6 ± 1.1	7.7 ± 1.3	0.619	7.8 ± 1.9	7.7 ± 1.1	0.575
Ventilator setting frequency (cycles/min)	15.1 ± 4.3	15.1 ± 3.8	0.977	12.7 ± 1.8	14.9 ± 4.8	0.002
P_{high}	_	_		24.1 ± 3.6	_	
PEEP (cmH_2O)	11.4 ± 3.0	10.4 ± 2.6	0.063	6.9 ± 1.8	10.4 ± 2.8	<0.001
FIO_2	0.66 ± 0.19	0.62 ± 0.19	0.198	0.43 ± 0.09	0.53 ± 0.19	0.001
Respiratory rate (cycles/min)	21.5 ± 6.6	19.5 ± 4.6	0.039	19.0 ± 6.0	20.3 ± 5.1	0.225
Peak inspiratory pressure (cmH_2O)	31.7 ± 4.5	30.4 ± 4.0	0.061	26.2 ± 3.6	28.5 ± 4.8	0.005
Mean airway pressure (cmH_2O)	18.3 ± 3.9	17.4 ± 3.5	0.140	21.8 ± 3.5	16.0 ± 3.3	<0.001
Plateau pressure (cmH_2O)	26.5 ± 4.0	25.3 ± 3.6	0.081	19.3 ± 3.9	23.3 ± 4.6	<0.001
Driving pressure (cmH_2O)[a]	15.2 ± 3.6	14.8 ± 3.4	0.550	12.6 ± 3.5	12.8 ± 4.1	0.822
Respiratory system compliance (mL/cmH_2O)	30.1 ± 7.6	32.6 ± 7.7	0.058	43.7 ± 11.3	34.1 ± 8.9	<0.001
Total minute ventilation (L/min)[b]	8.37 ± 2.36	8.42 ± 1.98	0.905	6.86 ± 2.06	8.22 ± 2.30	0.001
Spontaneous minute ventilation (L/min)	-	-		1.78 ± 1.37	-	
pH	7.37 ± 0.09	7.38 ± 0.10	0.427	7.42 ± 0.05	7.42 ± 0.07	0.648
$PaCO_2$ (mmHg)	40.1 ± 7.4	41.7 ± 10.5	0.307	40.8 ± 7.3	42.3 ± 8.6	0.291
PaO_2 (mmHg)	72.5 ± 13.1	76.8 ± 20.5	0.149	116.2 ± 28.5	84.8 ± 20.1	<0.001
PaO_2:FiO_2	121.7 ± 46.8	138.3 ± 56.1	0.060	280.3 ± 83.9	180.5 ± 68.6	<0.001
Hemodynamic variables						
Heart rate (beats/min)	105.4 ± 22.5	110.2 ± 24.6	0.238	92.7 ± 16.6	103.6 ± 19.3	0.001
Systolic blood pressure (mmHg)	122.2 ± 17.9	116.2 ± 22.5	0.088	126.6 ± 18.0	125.0 ± 20.3	0.646
Diastolic blood pressure (mmHg)	72.8 ± 13.2	68.6 ± 12.1	0.053	76.1 ± 14.5	69.3 ± 13.3	0.009
Mean arterial pressure (mmHg)	87.4 ± 14.7	84.2 ± 13.4	0.194	92.8 ± 14.9	87.1 ± 13.6	0.032

Main outcome variables	APRV ($n = 71$) [b]	LTV ($n = 67$) [b]	*P* value
No. of days of ventilation	8 [5–14]	15 [7–22]	0.001
No. of ventilator-free days at 28 days	19 [8–22]	2 [0–15]	<0.001
Successful extubation	47 (66.2%)	26 (38.8%)	0.001
Tracheostomy	9 (12.7%)	20 (29.9%)	0.013
Length of ICU stay (days)	15 [8–21]	20 [10–32]	0.015
Pneumothorax between day 1 and day 28[a]	3 (4.2%)	7 (10.4%)	0.199
Death during the ICU stay	14 (19.7%)	23 (34.3%)	0.053
Length of hospital stay (days)	21 [14–30]	27 [18–41]	0.055
Death during the hospital stay	17 (23.9%)	25 (37.3%)	0.088

Une autre étude: essai clinique (BiRDS) est terminé après l'inclusion de 700 malades et les résultats sont en attente (www.clinicaltrials.gov NCT01862016)

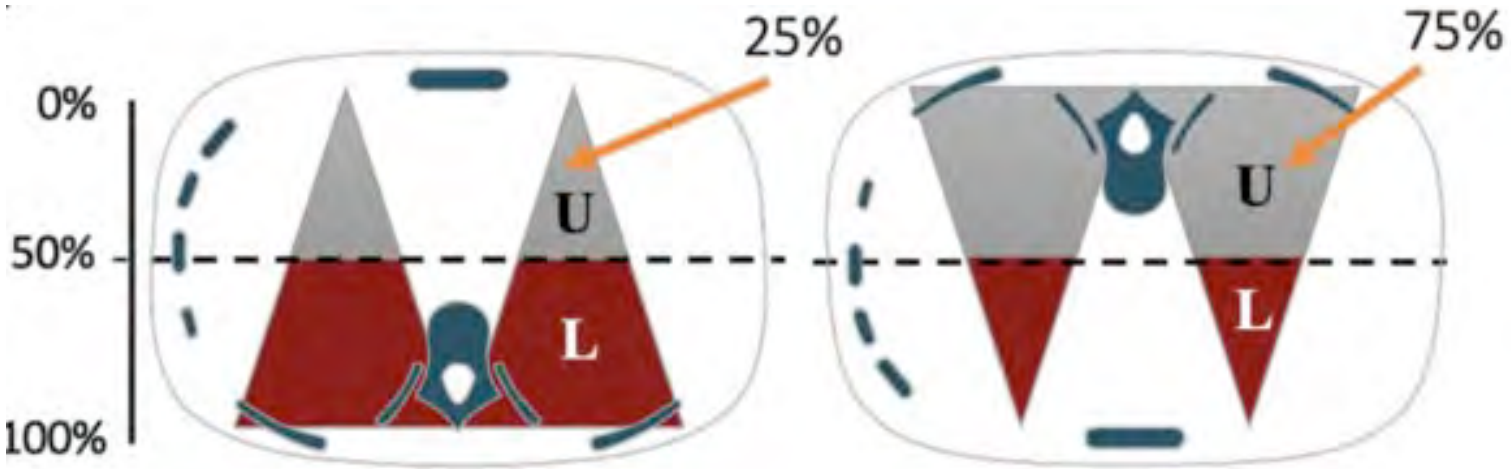
Champ 5 : Décubitus ventral

R5.1 – Il faut utiliser le décubitus ventral en cas de SDRA avec rapport PaO2/FIO2 < 150 mmHg pour diminuer la mortalité. Dans ce cas, il faut réaliser des séances prolongées d'au moins 16 heures consécutives.

GRADE 1+, ACCORD FORT

Prone Positioning in Acute Respiratory Distress Syndrome

Luciano Gattinoni, MD, FRCP[1] Mattia Busana, MD[1] Lorenzo Giosa, MD[1] Matteo Maria Macrì, MD[1]
Michael Quintel, MD[1]



Semin Respir Crit Care Med 2019;40:94–100.

Clinical Trials in the Prone Positioning					
Year	2001	2004	2006	2009	2013
	Gattinoni et al.	Guérin et al.	Mancebo et al.	Taccone et al.	Guérin et al.
Study period	1996–1999	1998–2002	1998–2002	2004–2008	2008–2011
Patients	304	802	142	344	466
Average PaO_2/FiO_2 at enrollment	127	152	105	113	100
PEEP at enrollment	10	8	7	10	10
SAPS II	40	46	41	41	46
Duration of prone position	7 h × 5 d	9 h × 4 d	17 h × 10 d	18 h × 8 d	17 h × 4 d
Protective ventilation	No	No	VT < 10 mL/kg	VT < 10 mL/kg	6 mL/kg
Follow-up	6 mo	90 d	Hospital discharge	6 mo	90 d
Mortality (%)					
Supine	58.3	42.2	60	52.9	41
Prone	62.2	43.3	50	47.6	23.6
p-Value	0.5	0.74	0.22	0.33	0.001

Arrêtée pour problème de recrutement

Arrêtée pour problème de recrutement

Semin Respir Crit Care Med 2019;40:94–100.

Oxygénation extra-corporelle (ECMO) veino-veineuse

R6.1 – Il faut probablement considérer la mise en place d'une ECMO veino-veineuse en cas de SDRA sévère avec PaO_2/FiO_2 <80 mmHg et/ou lorsque la ventilation mécanique devient dangereuse du fait de l'augmentation de la pression de plateau et malgré l'optimisation de la prise en charge du SDRA incluant niveaux élevés de PEP, curarisation et décubitus ventral. La décision de mise en place de l'ECMO doit être évaluée précocement par le contact avec un centre expert.

GRADE 2+, ACCORD FORT

➔Etude CESAR: Lancet 2009;374(9698):1351-1363

- Le critère principal décès et/ou incapacité fonctionnelle à 6 mois était significativement moins fréquent dans le groupe ECMO
- Mais :
  - nombre important de malades contrôles n'ayant pas bénéficié d'une ventilation protectrice
  - 25% des patients randomisés pour ECMO ne l'ont finalement pas reçu

➔Etude EOLIA: N Engl JMed 2018;378(21):1965-1975

- Mortalité J60 : diminution de 11 % (35% vs 46%; p=0,09)
- 28% des patients du groupe contrôle ont dû recevoir une ECMO de sauvetage

Venovenous extracorporeal membrane oxygenation for acute respiratory distress syndrome: a systematic review and meta-analysis

Lancet Respir Med 2019; 7: 163–72

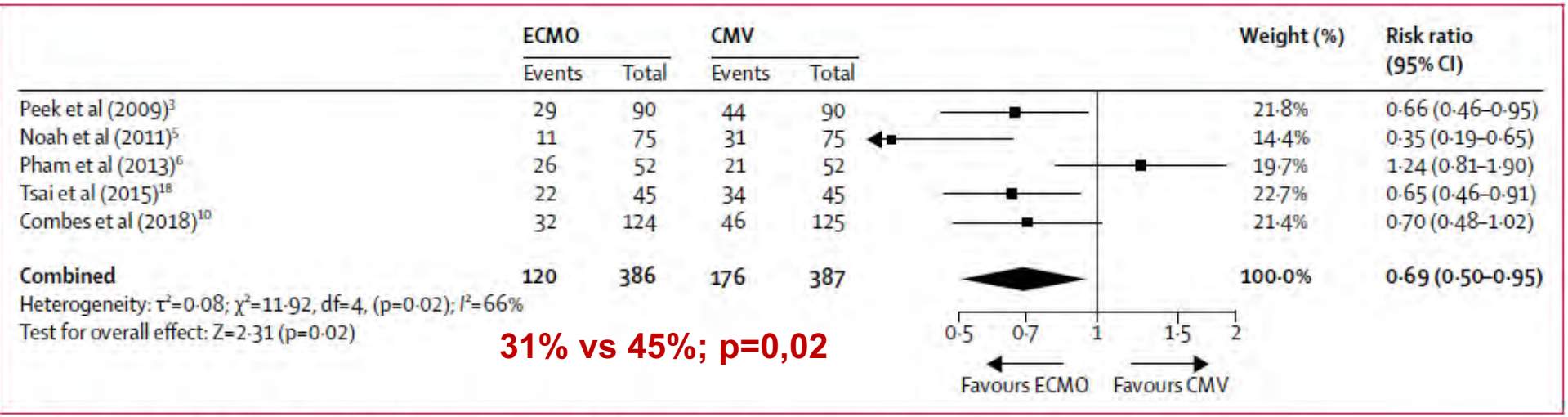


Figure 5: Forest plot of 30-day mortality across all studies of ECMO *vs* CMV in adults with severe acute respiratory distress syndrome

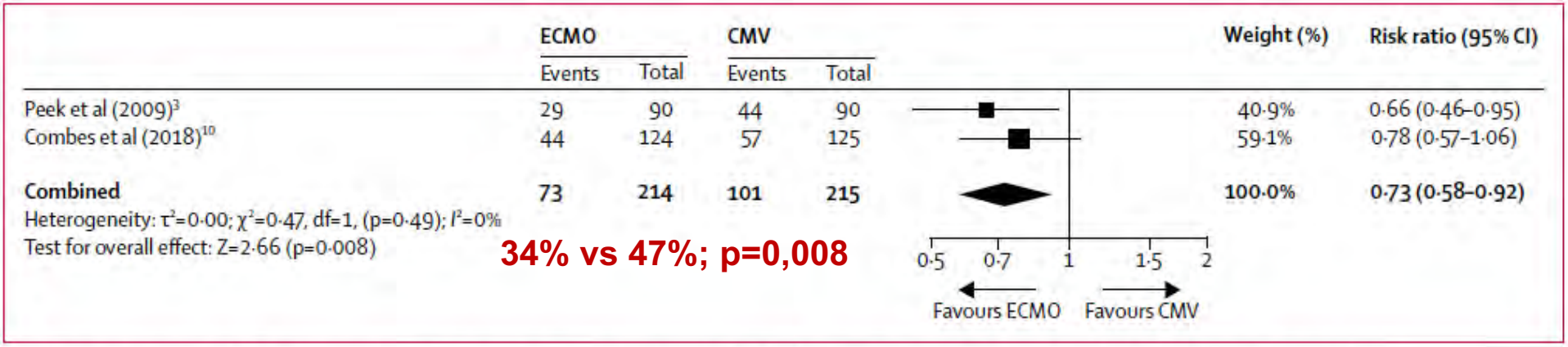


Figure 2: Forest plot of 60-day mortality in randomised controlled trials of ECMO *vs* CMV in adults with severe acute respiratory distress syndrome

Epuration extra-corporelle à faible débit de CO2

R6.2 – Au vu des données disponibles il n'est pas possible d'émettre de recommandation sur l'utilisation des techniques d'épuration extra-corporelle du CO2 à faible débit (ECCO2-R) au cours du SDRA.

PAS DE RECOMMANDATION

Champ 7 : Monoxyde d'azote inhalé

R7.1 – Les experts suggèrent que le NO inhalé puisse être utilisé en cas de SDRA avec hypoxémie profonde malgré l'implémentation d'une stratégie de ventilation protectrice et mise en décubitus ventral et avant d'envisager le recours à l'ECMO veino-veineuse.

AVIS D'EXPERTS

Inhaled nitric oxide for acute respiratory distress syndrome (ARDS) in children and adults (Review)

Cochrane Database of Systematic Reviews 2016, Issue 6. Art. No.: CD002787.

Mortalité globale

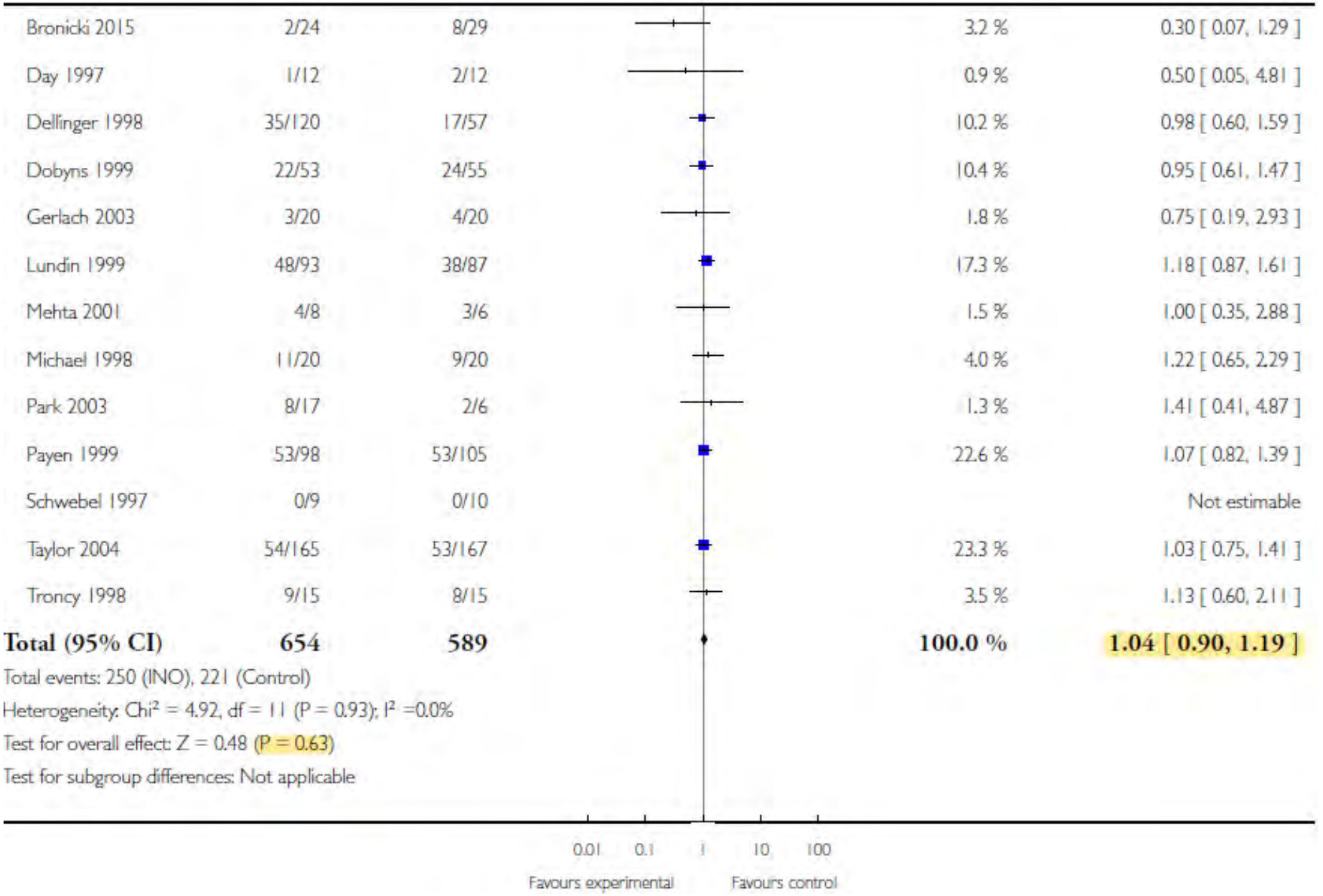
Study or subgroup	INO n/N	Control n/N	Risk Ratio M-H,Fixed,95% CI	Weight	Risk Ratio M-H,Fixed,95% CI
Bronicki 2015	2/24	8/29		3.2 %	0.30 [0.07, 1.29]
Day 1997	1/12	2/12		0.9 %	0.50 [0.05, 4.81]
Dellinger 1998	35/120	17/57		10.2 %	0.98 [0.60, 1.59]
Dobyns 1999	22/53	24/55		10.4 %	0.95 [0.61, 1.47]
Gerlach 2003	3/20	4/20		1.8 %	0.75 [0.19, 2.93]
Lundin 1999	48/93	38/87		17.3 %	1.18 [0.87, 1.61]
Mehta 2001	4/8	3/6		1.5 %	1.00 [0.35, 2.88]
Michael 1998	11/20	9/20		4.0 %	1.22 [0.65, 2.29]
Park 2003	8/17	2/6		1.3 %	1.41 [0.41, 4.87]
Payen 1999	53/98	53/105		22.6 %	1.07 [0.82, 1.39]
Schwebel 1997	0/9	0/10			Not estimable
Taylor 2004	54/165	53/167		23.3 %	1.03 [0.75, 1.41]
Troncy 1998	9/15	8/15		3.5 %	1.13 [0.60, 2.11]
Total (95% CI)	**654**	**589**		**100.0 %**	**1.04 [0.90, 1.19]**

Total events: 250 (INO), 221 (Control)

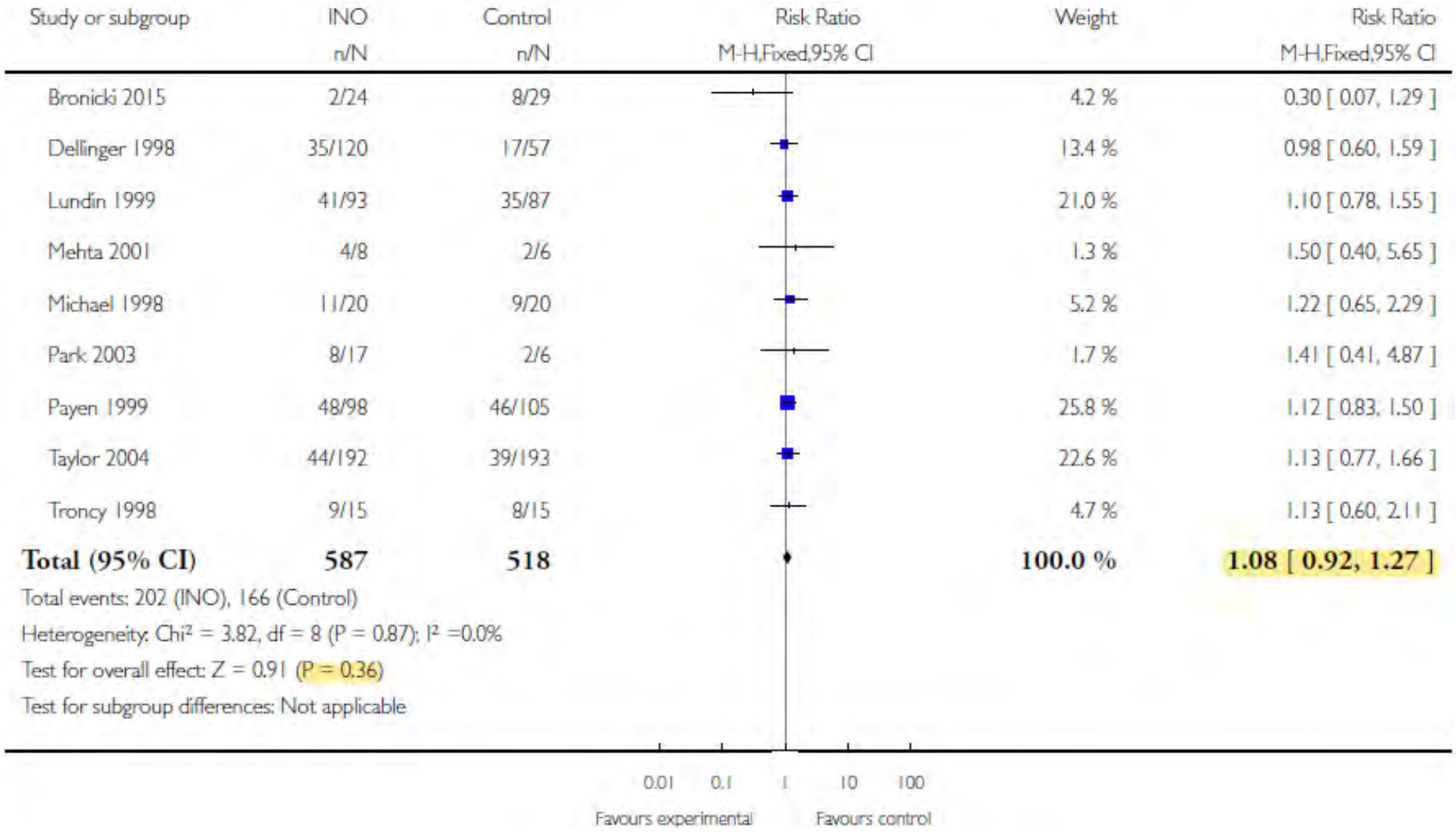
Heterogeneity: $Chi^2 = 4.92$, $df = 11$ ($P = 0.93$); $I^2 = 0.0\%$

Test for overall effect: $Z = 0.48$ ($P = 0.63$)

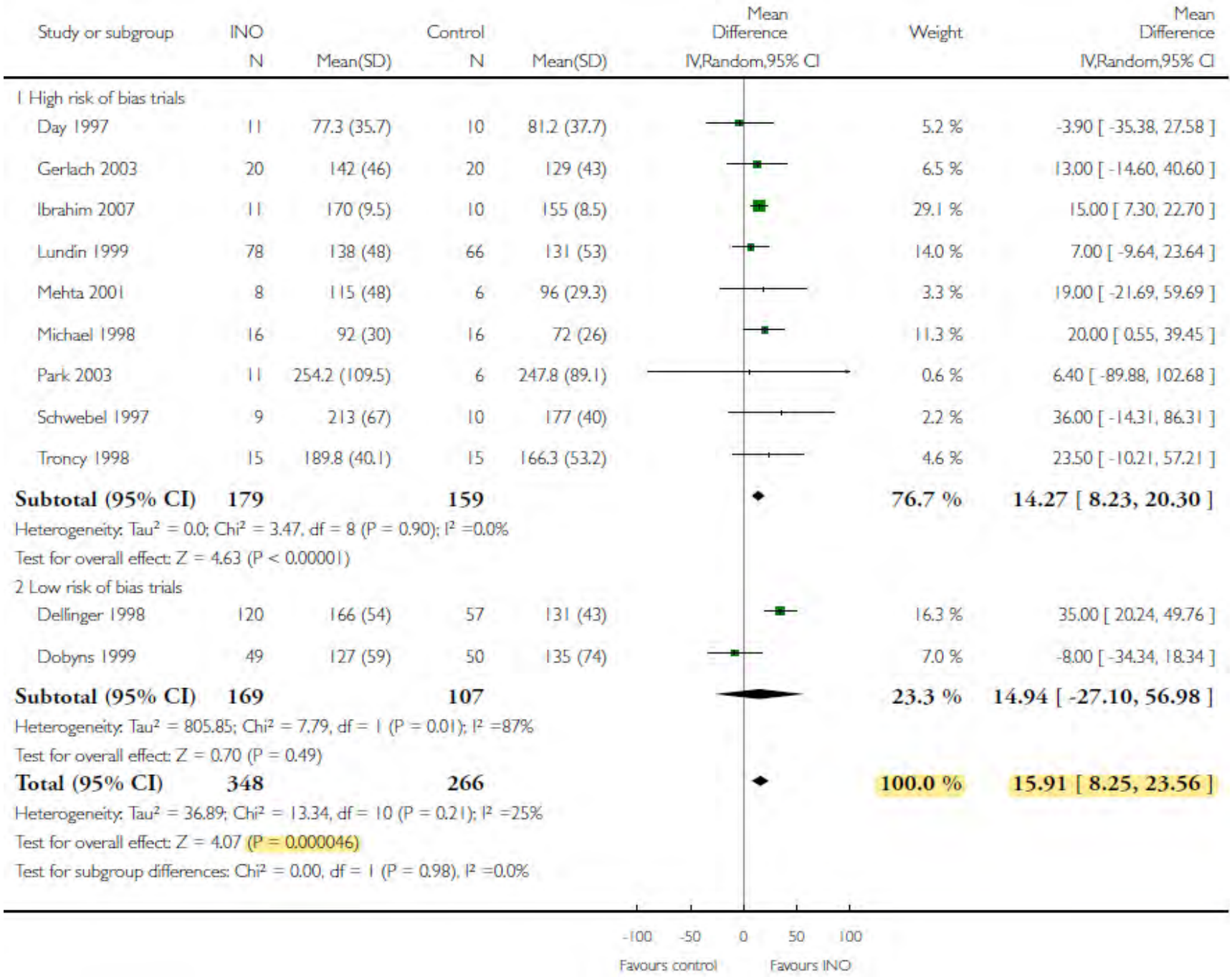
Test for subgroup differences: Not applicable



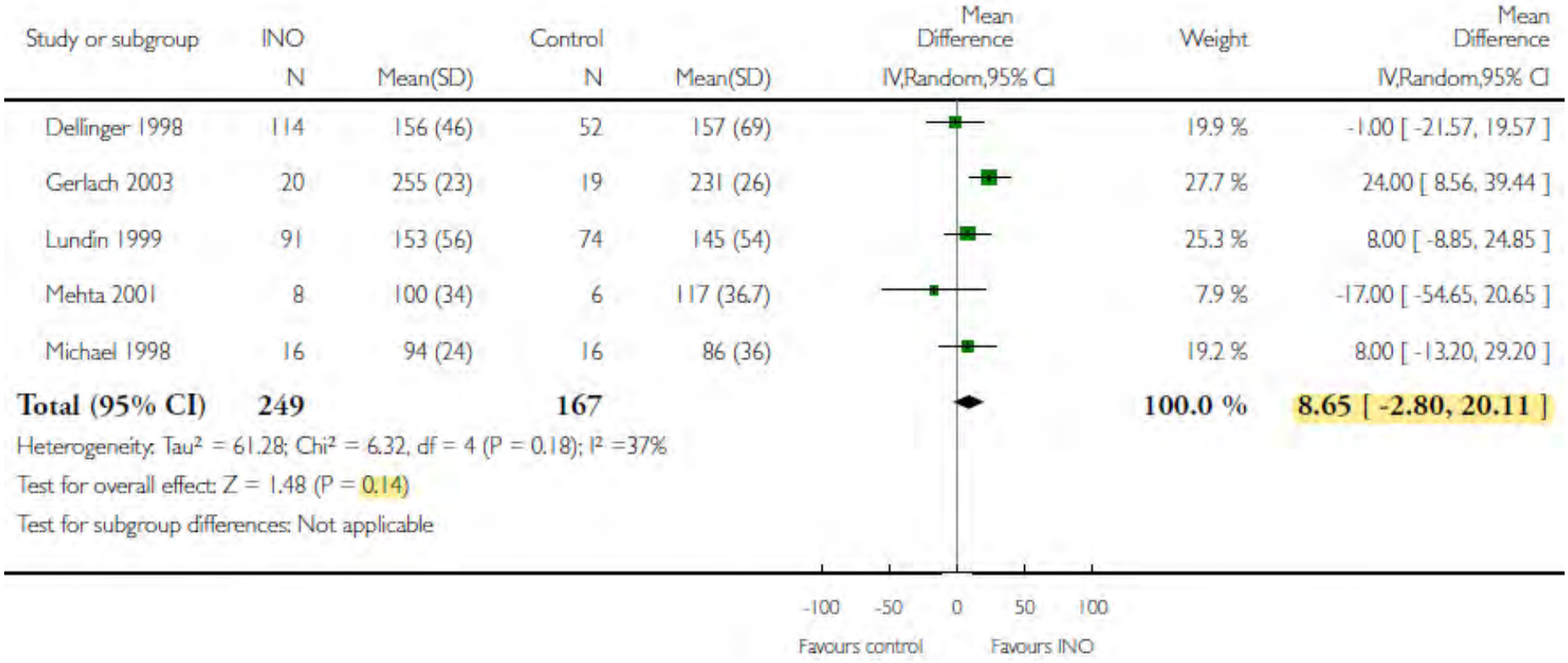
Mortalité J28-J30



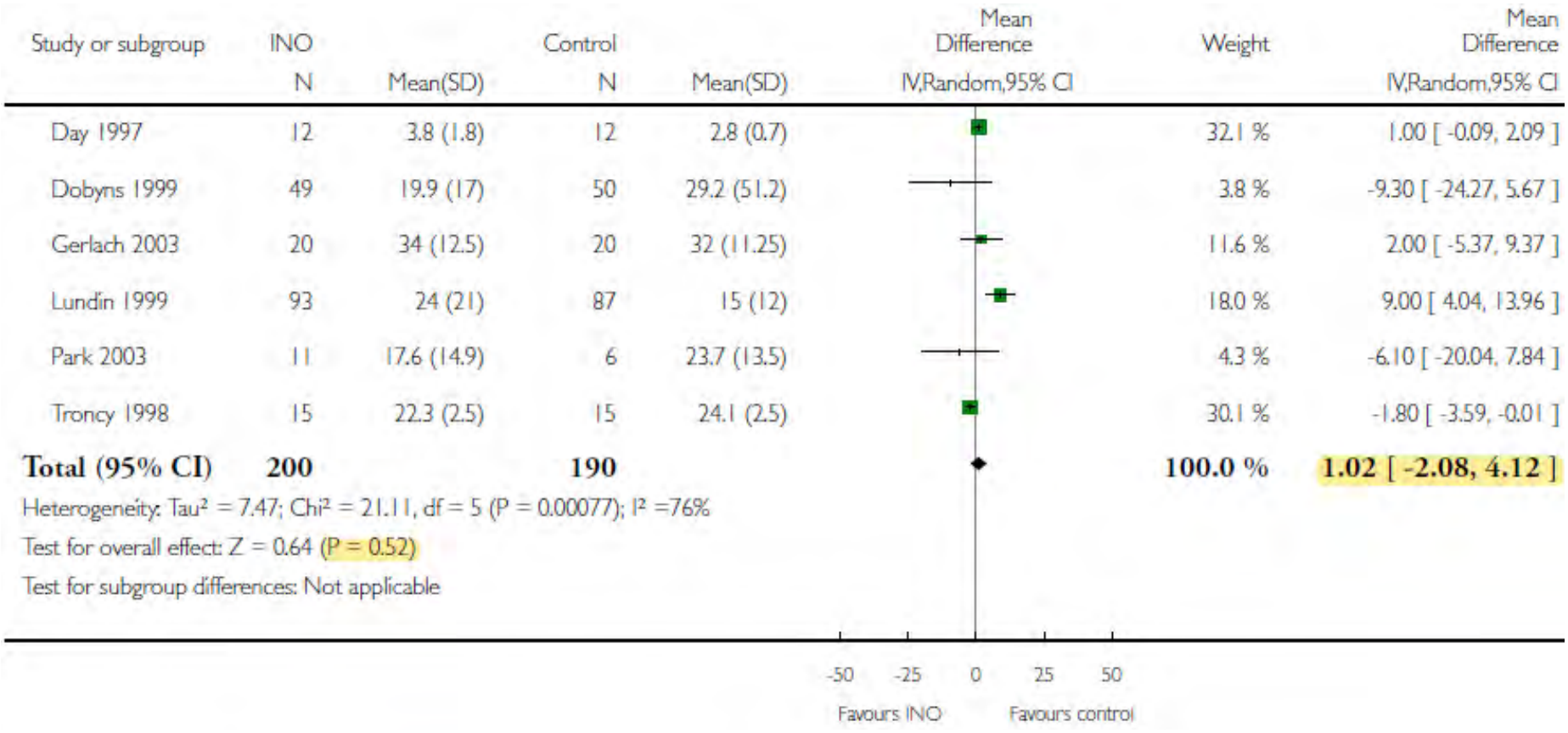
PaO_2/FiO_2 à H24



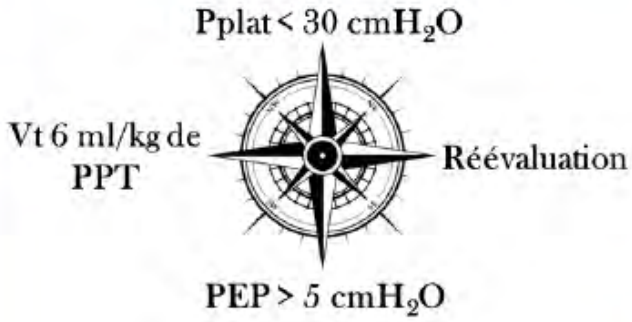
PaO2/FiO2 à H48



Durée de VM


Study or subgroup
INO
N
Mean(SD)
Control
N
Mean(SD)
Mean Difference
IV,Random,95% CI
Weight
Mean Difference
IV,Random,95% CI
Day 1997 12 3.8 (1.8) 12 2.8 (0.7) 32.1 % 1.00 [-0.09, 2.09]
Dobyns 1999 49 19.9 (17) 50 29.2 (51.2) 3.8 % -9.30 [-24.27, 5.67]
Gerlach 2003 20 34 (12.5) 20 32 (11.25) 11.6 % 2.00 [-5.37, 9.37]
Lundin 1999 93 24 (21) 87 15 (12) 18.0 % 9.00 [4.04, 13.96]
Park 2003 11 17.6 (14.9) 6 23.7 (13.5) 4.3 % -6.10 [-20.04, 7.84]
Troncy 1998 15 22.3 (2.5) 15 24.1 (2.5) 30.1 % -1.80 [-3.59, -0.01]
Total (95% CI) 200 190 100.0 % 1.02 [-2.08, 4.12]
Heterogeneity: Tau² = 7.47; Chi² = 21.11, df = 5 (P = 0.00077); I² =76%
Test for overall effect: Z = 0.64 (P = 0.52)
Test for subgroup differences: Not applicable
-50
-25
0
25
50
Favours INO
Favours control

Prise en charge initiale du SDRA en 2019



Toute initiation de ventilation invasive avec sédation en réanimation

SDRA confirmé

Vt autour de 6 ml/kg de PPT à la phase initiale en l'absence d'acidose métabolique profonde

Recherche systématique des critères diagnostiques de SDRA

P/F < 200 — Vt autour de 6 ml/kg de PPT
Pplateau < 30 cmH2O
PEP > 5 cmH2O
Surveiller hypercapnie

P/F < 150 — PEP élevée si améliore oxygénation

P/F < 80 — Curarisation
Décubitus ventral

Discuter ECMO VV

Sévérité du SDRA

Réévaluation des réglages et de la stratégie de prise en charge au moins toutes les 24h

Poids prédit par la taille
- Tableau

ECMO veino-veineuse
- ❑ Si hypoxémie réfractaire ou ventilation protectrice non applicable
- ❑ A discuter avec un centre expert

Modalités de la curarisation : IVSE
- ❑ Précocement, dans les 48h du diagnostic

Modalités du décubitus ventral (DV) : VIDEO
- ❑ séance ≥ 16 heures, plusieurs séances

SDRA modéré ou sévère → Test PEP élevée (> 12 cmH2O)
Utilisation PEP élevée si :
- ❑ Amélioration de l'oxygénation
- ❑ Sans dégradation significative de la compliance du système respiratoire et de l'hémodynamique
- ❑ Maintien Pplateau < 30 cmH2O, monitorage continu

Critères du SDRA
- ❑ PaO2/FiO2 ≤ 300 mmHg
- ❑ PEP ≥ 5 cmH2O
- ❑ Opacités bilatérales sur l'imagerie thoracique
- ❑ Non expliquées par défaillance ventriculaire gauche
- ❑ Évolution depuis moins de 7 jours

Traitement possible
- Monoxyde d'azote inhalé (iNO), si hypoxémie persistante en DV avant discussion de l'ECMO VV
- Ventilation spontanée après la phase aiguë avec Vt généré autour de 6 ml/kg sans dépasser 8 ml/kg

Pas de recommandation possible
- ECCO2R
- Pression motrice
- Ventilation spontanée à la phase aiguë

Probablement ne pas faire
- Manœuvres de recrutement systématiques

Ne pas faire
- HFOV

Corticosteroids in the prevention and treatment of acute respiratory distress syndrome (ARDS) in adults: meta-analysis

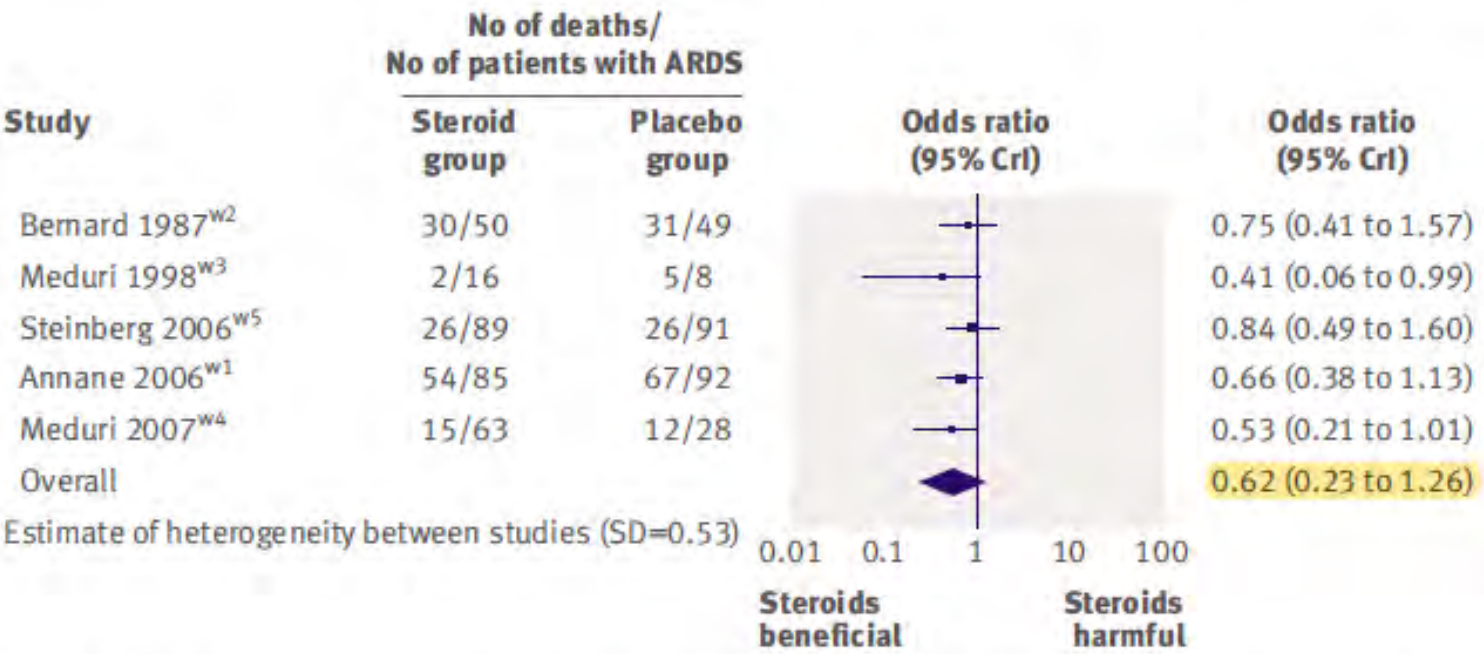
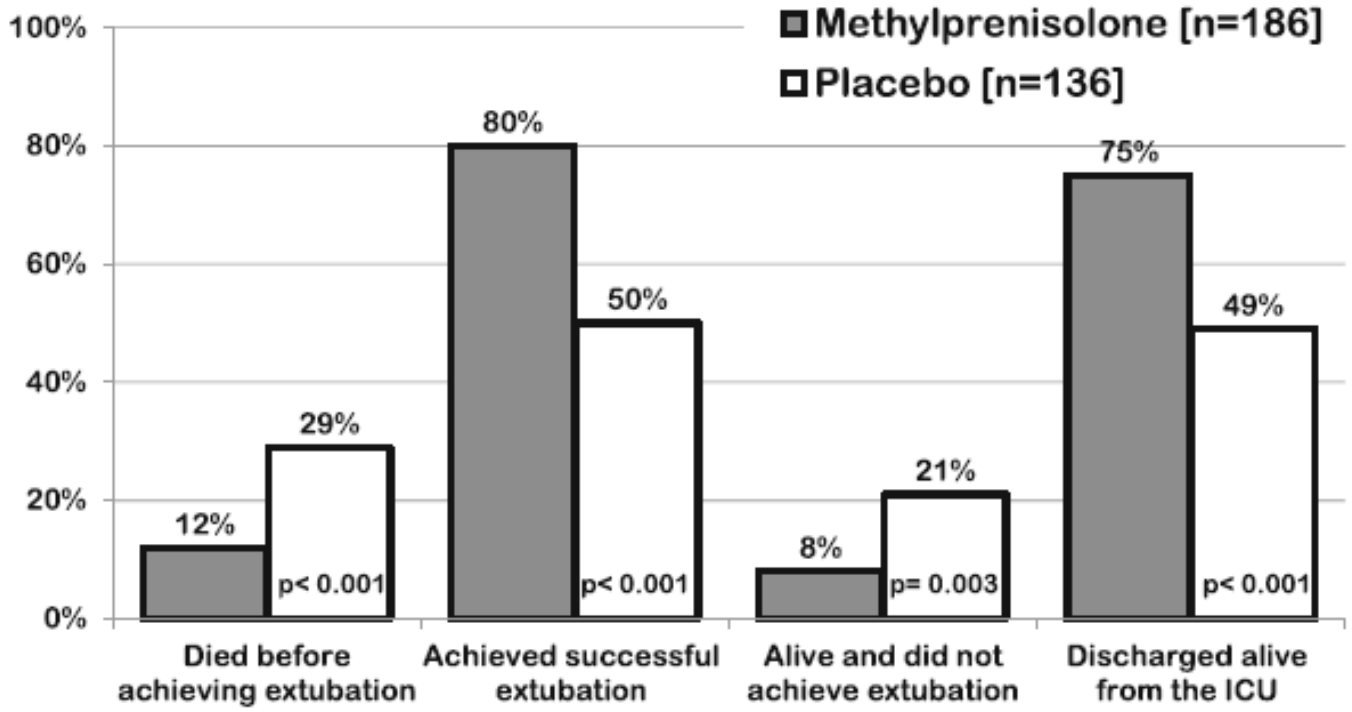


Fig 4 | Effect of therapeutic steroids on mortality in patients with acute respiratory distress syndrome

BMJ. 2008 May 3; 336(7651): 1006–1009.

Prolonged glucocorticoid treatment is associated with improved ARDS outcomes: analysis of individual patients' data from four randomized trials and trial-level meta-analysis of the updated literature

Intensive Care Med
DOI 10.1007/s00134-015-4095-4



Published online: 27 October 2015

Table 1 Methylprednisolone treatment of early moderate-to-severe ARDS and late unresolving ARDS

Early moderate-to-severe ARDS (PaO_2:$FiO_2 \leq 200$ on PEEP 5 cmH_20)

Time	Intravenous administration form	Dosage
Loading	Bolus over 30 min	1 mg/kg
Days 1 to 14*,†,‡	Infusion at 10 cc/hour	1 mg/kg/day
Days 15 to 21*,‡	Infusion at 10 cc/hour	0.5 mg/kg/day
Days 22 to 25*,‡	Infusion at 10 cc/hour	0.25 mg/kg/day
Days 26 to 28*,‡	Infusion at 10 cc/hour	0.125 mg/kg/day

Unresolving ARDS = less than (a) one-point reduction in lung injury score or (b) or 100 improvement of in PaO_2:FiO_2

- By day 7 of ARDS in patients not receiving methylprednisolone for early ARDS
- By days 5–7 of ARDS in patients receiving methylprednisolone (above) for early ARDS

Time	Intravenous administration form	Dosage
Loading	Bolus over 30 min	2 mg/kg
Days 1 to 14*,†,‡	Infusion at 10 cc/hour	2 mg/kg/day
Days 15 to 21*,‡	Infusion at 10 cc/hour	1 mg/kg/day
Days 22 to 25*,‡	Infusion at 10 cc/hour	0.5 mg/kg/day
Days 26 to 28*,‡	Infusion at 10 cc/hour	0.25 mg/kg/day
Days 29 to 28*,‡	Bolus over 30 min	0.125 mg/kg/day

Meduri *et al. Journal of Intensive Care* (2018) 6:53
https://doi.org/10.1186/s40560-018-0321-9

Review

Emerging drugs for treating the acute respiratory distress syndrome

Timothy D. Shaw, Daniel F. McAuley & Cecilia M. O'Kane

Pages 29-41 | Received 01 Dec 2018, Accepted 04 Mar 2019, Accepted author version posted online: 06 Mar 2019, Published online: 18 Mar 2019

Download citation https://doi.org/10.1080/14728214.2019.1591369 Check for updates

Table 2: Characteristics of pharmacological therapies for ARDS in current clinical trials

Intervention	Company	Mechanism of action	Reference	Trial Design	Status
Dexamethasone	Non-proprietary	Glucocorticoid receptor agonist	NCT01731795	Phase 3	Recruiting
Interferon-β	Faron Pharmaceuticals Ltd	Interferon beta 1 agonist	NCT02622724	Phase 3	Completed Provisional results reported
Vitamin D	Non-proprietary	Activation of Vitamin D Receptor	NCT03096314	Phase 3	Active, not recruiting
Aspirin	Non-proprietary	Non-selective inhibitor of cyclooxygenase	NCT02326350	Phase 2	Stopped
Bone-marrow derived multi-potent adult progenitor cells (MAPCs)	Athersys	Paracrine and cell-contact dependent immunomodulation	NCT02611609	Phase 2	Recruitment completed
Umbilical cord derived mesenchymal stromal cells (MSCs)	Orbsen Therapeutics	Paracrine and cell-contact dependent immunomodulation	NCT03042143	Phase 1/2	Recruiting
Vitamin C	Non-proprietary	Inhibition of NK-KB expression Expression of alveolar epithelial sodium channel	NCT02106975	Phase 2	Completed: results awaited
Solnatide (AP-301)	Apeptico Forschung und Entwicklung GmbH	Activation of alveolar epithelial sodium channel	NCT01627613	Phase 2	Completed
Nebulised liquid heparin	Non-proprietary	Activation of anti-thrombin III	ACTRN12612000418875	Phase 2	Ongoing
ALT-836	Altor	Inhibition of	NCT00879606	Phase	Complete
(Anti-TF antibody)	Bioscience Corporation	tissue factor		2	d Results awaited
Dilmapimod (SB-681323)	GlaxoSmithKline	p38 Mitogen-Activated Protein Kinase (MAPK) Inhibitor	NCT00996840	Phase 2	Completed Results reported
Insulin	Non-proprietary	Inhibition of NF-KB expression Modulation of endothelial nitric oxide production	NCT00605696	Phase 2	Completed Full results awaited
Ulinastatin	Takeda	Inhibition of polymorphonuclear granulocyte elastase	NCT02895191	Phase 2	Recruiting
GM-CSF	Savara Inc	Maturation of alveolar epithelial cells and macrophages	NCT02595060	Phase 2	Recruiting
IC14 (anti-CD14 antibody)	Implicit Bioscience	Inhibition of CD14 pattern recognition receptor	NCT03017547	Phase 2	Not yet recruiting
Treprostinil Sodium (prostaglandin)	United Therapeutics	Stimulation of pulmonary vasodilation	NCT02370095	Phase 2	Recruiting

CONCISE CLINICAL REVIEW

FIFTY YEARS OF RESEARCH IN ARDS

Is Acute Respiratory Distress Syndrome a Preventable Disease?

Hemang Yadav[1], B. Taylor Thompson[2], and Ognjen Gajic[1]

[1]Division of Pulmonary and Critical Care Medicine, Mayo Clinic, Rochester, Minnesota; and [2]Division of Pulmonary and Critical Care Medicine, Massachusetts General Hospital, Boston, Massachusetts

ORCID ID: 0000-0002-1889-2524 (H.Y.).

Am J Respir Crit Care Med Vol 195, Iss 6, pp 725–736, Mar 15, 2017

Early Identification of Patients at Risk of Acute Lung Injury

Am J Respir Crit Care Med Vol 183. pp 462–470, 2011

Evaluation of Lung Injury Prediction Score in a Multicenter Cohort Study

Predisposing Conditions	
Shock	2
Aspiration	2
Sepsis	1
Pneumonia	1.5
High-risk surgery*	
Orthopedic spine	1
Acute abdomen	2
Cardiac	2.5
Aortic vascular	3.5
High-risk trauma	
Traumatic brain injury	2
Smoke inhalation	2
Near drowning	2
Lung contusion	1.5
Multiple fractures	1.5
Risk modifiers	
Alcohol abuse	1
Obesity (BMI $>$ 30)	1
Hypoalbuminemia	1
Chemotherapy	1
$F_{IO_2} > 0.35$ ($>$ 4 L/min)	2
Tachypnea (RR $>$ 30)	1.5
$Sp_{O_2} < 95\%$	1
Acidosis (pH $<$ 7.35)	1.5
Diabetes mellitus†	−1

(*1*) Patient with history of alcohol abuse with septic shock from pneumonia requiring $F_{IO_2} > 0.35$ in the emergency room: Sepsis + shock + pneumonia + alcohol abuse + $F_{IO_2} > 0.35$
$1 + 2 + 1.5 + 1 + 2 = 7.5$

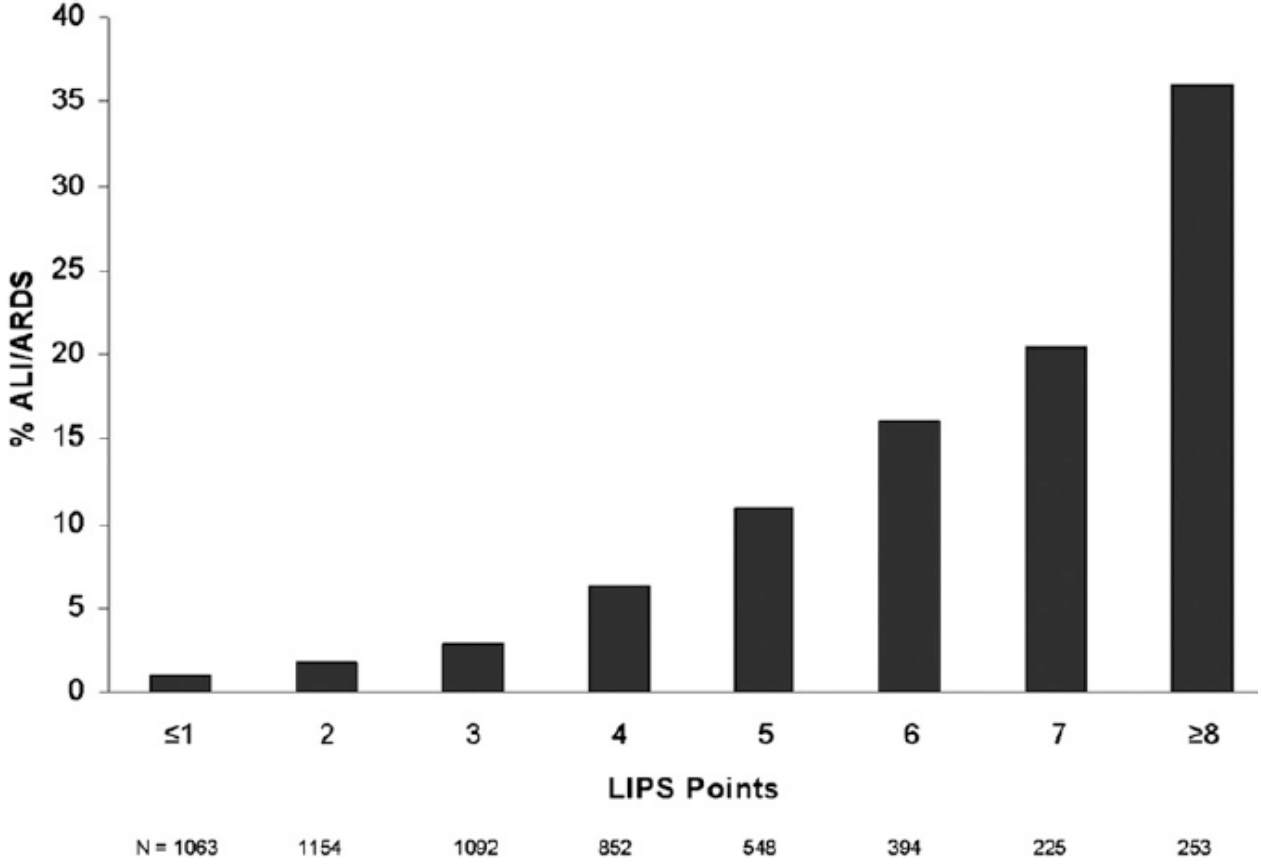
(*2*) Motor vehicle accident with traumatic brain injury, lung contusion, and shock requiring $F_{IO_2} > 0.35$
Traumatic brain injury + lung contusion + shock+ $F_{IO_2} > 0.35$
$2 + 1.5 + 2 + 2 = 7.5$

(*3*) Patient with history of diabetes mellitus and urosepsis with shock
Sepsis + shock + diabetes
$1 + 2 - 1 = 2$

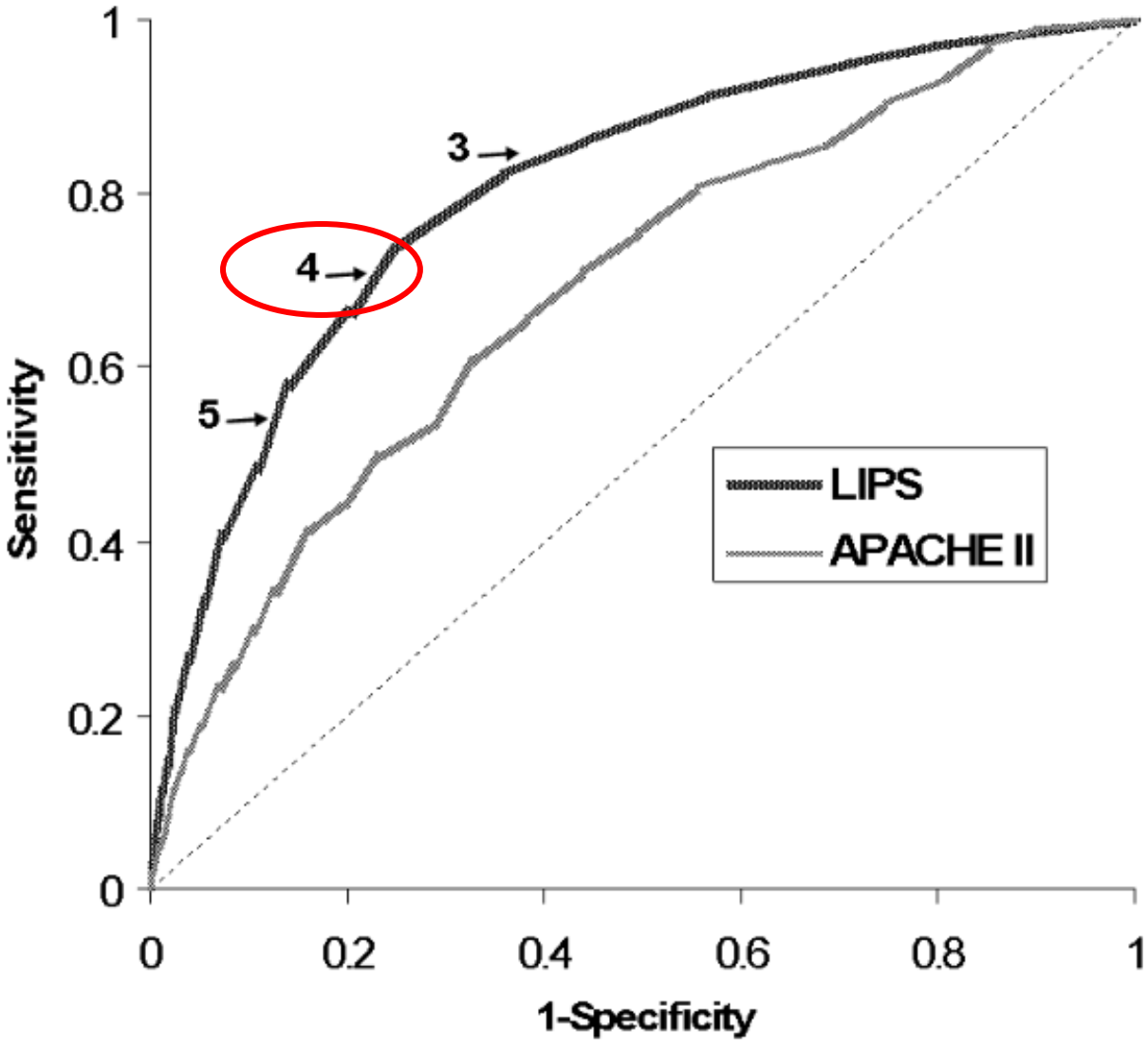
Definition of abbreviations: BMI = body mass index; RR = respiratory rate; Sp_{O_2} = oxygen saturation by pulse oximetry.

* Add 1.5 points if emergency surgery.

† Only if sepsis.



Am J Respir Crit Care Med Vol 183. pp 462–470, 2011



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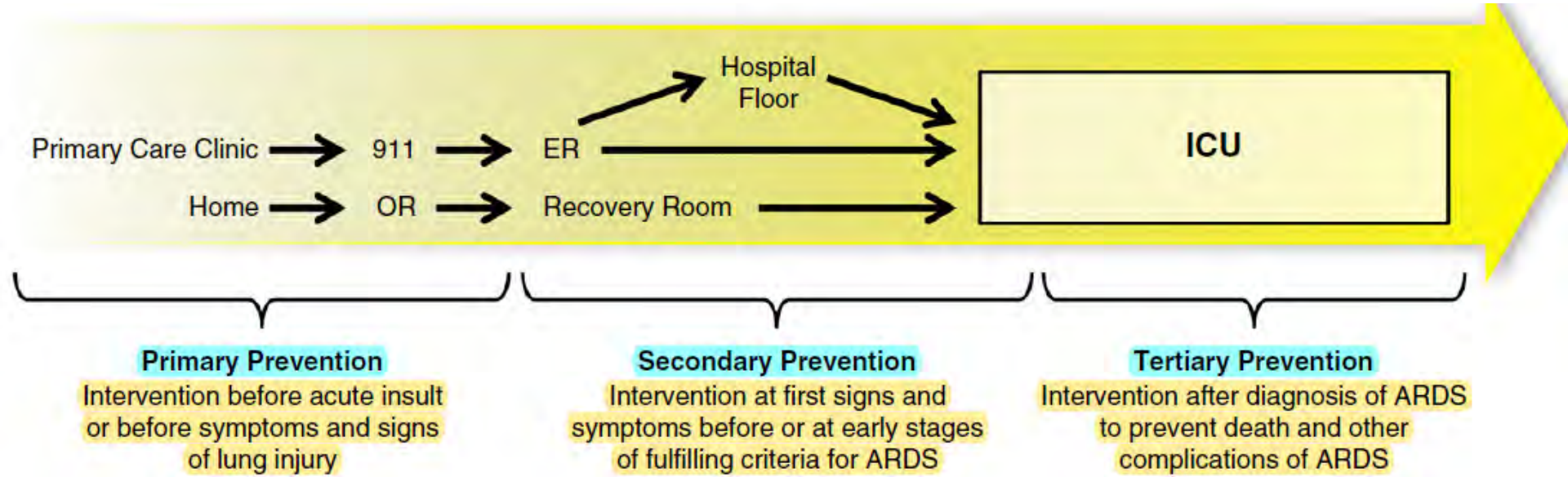

Am J Respir Crit Care Med Vol 195, Iss 6, pp 725–736, Mar 15, 2017

Table 1. Checklist for Lung Injury Prevention

CLIP Element	Best Practice
Adequate empiric antimicrobial treatment and source control	According to suspected site of infection, health care exposure, and immune suppression
Lung-protective mechanical ventilation	V_T < 6–8 ml/kg predicted body weight Plateau pressure < 25 cm H_2O PEEP ⩾ 5 cm H_2O, Minimize F_{IO_2} (target Sp_{O_2} = 88–92% after early shock
Aspiration precautions	Rapid sequence intubation supervised by experienced providers Elevated head of the bed Oral care with chlorhexidine Gastric acid neutralization Early reassessment of noninvasive ventilation to prevent delayed intubation
Limiting fluid overload	Modified ARDS Network FACTT protocol after early shock
Restrictive transfusion	Hemoglobin target > 7 g/dl Male-predominant plasma donor pool
Assess readiness for extubation	Limit continuous sedation and perform spontaneous breathing trial as soon as feasible

Definition of abbreviations: ARDS = acute respiratory distress syndrome; CLIP = Checklist for Lung Injury Prevention; FACTT = Fluid and Catheters Treatment Trial; PEEP = positive end-expiratory pressure; Sp_{O_2} = oxygen

Am J Respir Crit Care Med Vol 195, Iss 6, pp 725–736, Mar 15, 2017

NIH NHLBI ARDS Clinical Network
Mechanical Ventilation Protocol Summary

INCLUSION CRITERIA: Acute onset of

1. $PaO_2/FiO_2 \leq 300$ (corrected for altitude)
2. Bilateral (patchy, diffuse, or homogeneous) infiltrates consistent with pulmonary edema
3. No clinical evidence of left atrial hypertension

PART I: VENTILATOR SETUP AND ADJUSTMENT

1. Calculate predicted body weight (PBW)
   Males = 50 + 2.3 [height (inches) - 60]
   Females = 45.5 + 2.3 [height (inches) -60]
2. Select any ventilator mode
3. Set ventilator settings to achieve initial V_T = 8 ml/kg PBW
4. Reduce V_T by 1 ml/kg at intervals ≤ 2 hours until V_T = 6ml/kg PBW.
5. Set initial rate to approximate baseline minute ventilation (not > 35 bpm).
6. Adjust V_T and RR to achieve pH and plateau pressure goals below.

OXYGENATION GOAL: PaO_2 55-80 mmHg or SpO_2 88-95%

Use a minimum PEEP of 5 cm H_2O. Consider use of incremental FiO_2/PEEP combinations such as shown below (not required) to achieve goal.

Lower PEEP/higher FiO2

FiO_2	0.3	0.4	0.4	0.5	0.5	0.6	0.7	0.7
PEEP	5	5	8	8	10	10	10	12

FiO_2	0.7	0.8	0.9	0.9	0.9	1.0
PEEP	14	14	14	16	18	18-24

Higher PEEP/lower FiO2

FiO_2	0.3	0.3	0.3	0.3	0.3	0.4	0.4	0.5
PEEP	5	8	10	12	14	14	16	16

FiO_2	0.5	0.5-0.8	0.8	0.9	1.0	1.0
PEEP	18	20	22	22	22	24

PLATEAU PRESSURE GOAL: ≤ 30 cm H_2O

Check Pplat (0.5 second inspiratory pause), at least q 4h and after each change in PEEP or V_T.

If Pplat > 30 cm H_2O: decrease V_T by 1ml/kg steps (minimum = 4 ml/kg).

If Pplat < 25 cm H_2O and V_T< 6 ml/kg, increase V_T by 1 ml/kg until Pplat > 25 cm H_2O or V_T = 6 ml/kg.

If Pplat < 30 and breath stacking or dys-synchrony occurs: may increase V_T in 1ml/kg increments to 7 or 8 ml/kg if Pplat remains ≤ 30 cm H_2O.

pH GOAL: 7.30-7.45

Acidosis Management: (pH < 7.30)

If pH 7.15-7.30: Increase RR until pH > 7.30 or $PaCO_2$ < 25 (Maximum set RR = 35).

.

If pH < 7.15: Increase RR to 35.

If pH remains < 7.15, V_T may be increased in 1 ml/kg steps until pH > 7.15 (Pplat target of 30 may be exceeded).

May give $NaHCO_3$

Alkalosis Management: (pH > 7.45) Decrease vent rate if possible.

I: E RATIO GOAL: Recommend that duration of inspiration be ≤ duration of expiration.

PART II: WEANING

A. Conduct a SPONTANEOUS BREATHING TRIAL daily when:

1. $FiO_2 \leq 0.40$ and PEEP ≤ 8 OR $FiO_2 \leq 0.50$ and PEEP ≤ 5.
2. PEEP and FiO_2 ≤ values of previous day.
3. Patient has acceptable spontaneous breathing efforts. (May decrease vent rate by 50% for 5 minutes to detect effort.)
4. Systolic BP ≥ 90 mmHg without vasopressor support.
5. No neuromuscular blocking agents or blockade.

B. SPONTANEOUS BREATHING TRIAL (SBT):

If all above criteria are met and subject has been in the study for at least 12 hours, initiate a trial of UP TO 120 minutes of spontaneous breathing with FiO2 ≤ 0.5 and PEEP ≤ 5:

1. Place on T-piece, trach collar, or CPAP ≤ 5 cm H_2O with PS ≤ 5
2. Assess for tolerance as below for up to two hours.
   a. $SpO_2 \geq 90$: and/or $PaO_2 \geq 60$ mmHg
   b. Spontaneous $V_T \geq 4$ ml/kg PBW
   c. RR ≤ 35/min
   d. pH ≥ 7.3
   e. No respiratory distress (distress= 2 or more)
      - HR > 120% of baseline
      - Marked accessory muscle use
      - Abdominal paradox
      - Diaphoresis
      - Marked dyspnea
3. If tolerated for at least 30 minutes, consider extubation.
4. If not tolerated resume pre-weaning settings.

Definition of UNASSISTED BREATHING
(Different from the spontaneous breathing criteria as PS is not allowed)

1. Extubated with face mask, nasal prong oxygen, or room air, OR
2. T-tube breathing, OR
3. Tracheostomy mask breathing, OR
4. CPAP less than or equal to 5 cm H_20 **without pressure support or IMV assistance.**

Angiotensin-converting enzymes in acute respiratory distress syndrome

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	P	OR	CI 95%
AGE, years	0.016	1.042	1.008-1.078
LACTATE, mmol/L	0.041	1.22	1.008-1.47
SOFA SCORE	0.008	1.17	1.04-1.33
ACE on T1, ng/mL	0.016	1.007	1.001-1.013

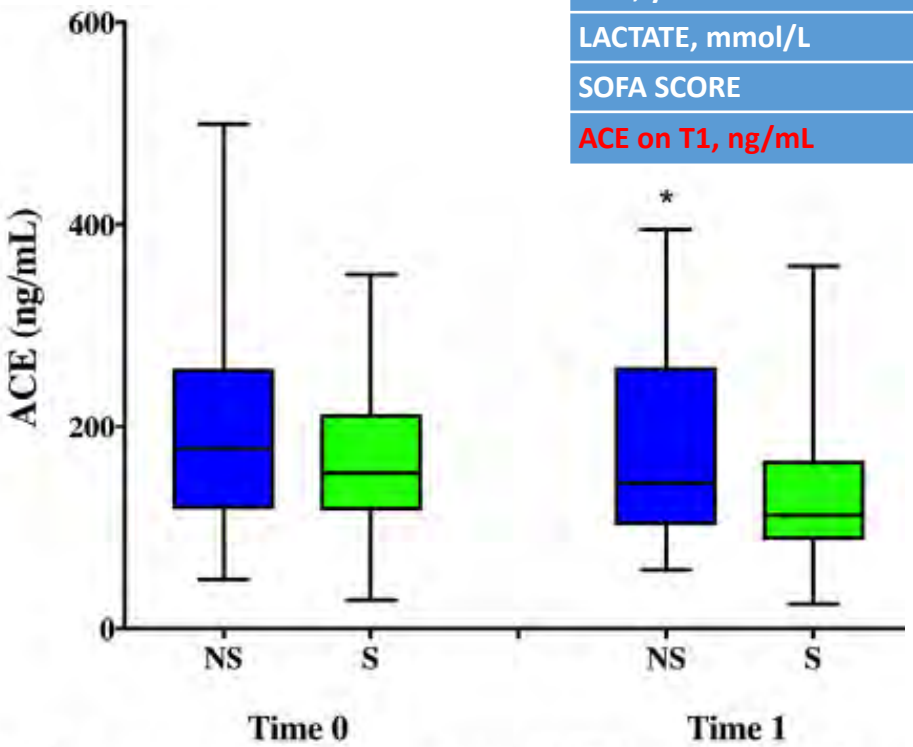

Fig. 1 Angiotensin-converting enzyme (ACE) levels at T0 and T1 according to ICU mortality (*NS* non-survivors; *S* survivors). *$p = 0.03$

Conclusion

- Considérer le SDRA comme complication évitable
- Identifier les patients à risque
- Mécanismes et phénotypes du SDRA
- Piliers du traitement :
  - ✓Ventilation protectrice
  - ✓Sédation ± curarisation
  - ✓Traitement de la cause