Traitement non ventilatoire du SDRA

Dr Islem OUANES
Service de Réanimation Polyvalente
CHU Fattouma Bourguiba Monastir

Cours du collège de Réanimation Médicale
Sfax le 08/03/2013

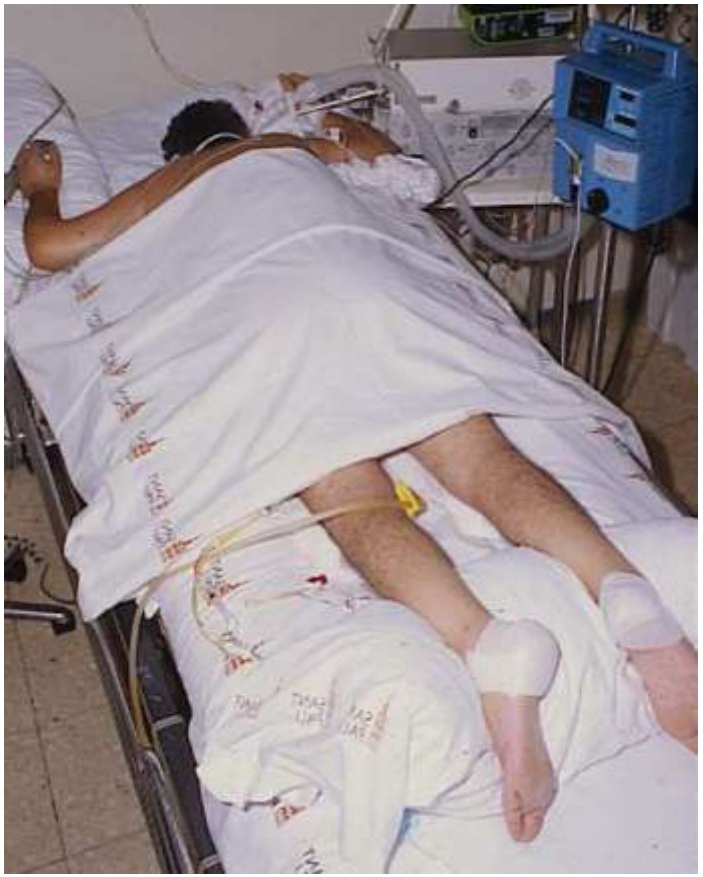
Introduction

- La prise en charge du SDRA est surtout ventilatoire et étiologique +++.
- De nombreux essais (molécules ou procédures) ont été conduits avec des résultats négatifs en terme de mortalité.
- Mais la mortalité est-elle le seul objectif acceptable?
- Récemment: quelques études positives sur la mortalité avec des traitements non ventilatoires du SDRA.

Plan

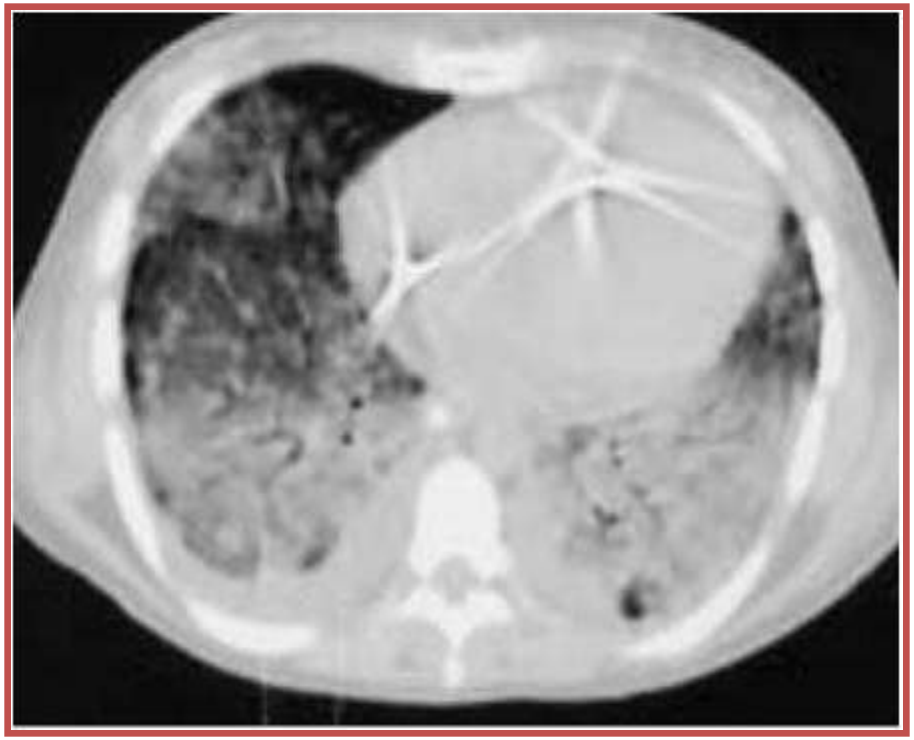
- **Procédures**
- Décubitus ventral (DV)
- Gestion du bilan hydrique
- ECMO
- **Traitement pharmacologique**
- Curares
- Anti-inflammatoires (corticostéroïdes…)
- Vasodilatateurs pulmonaires (NO…)
- Almitrine
- Agonistes β2-Adrénergiques
- Autres

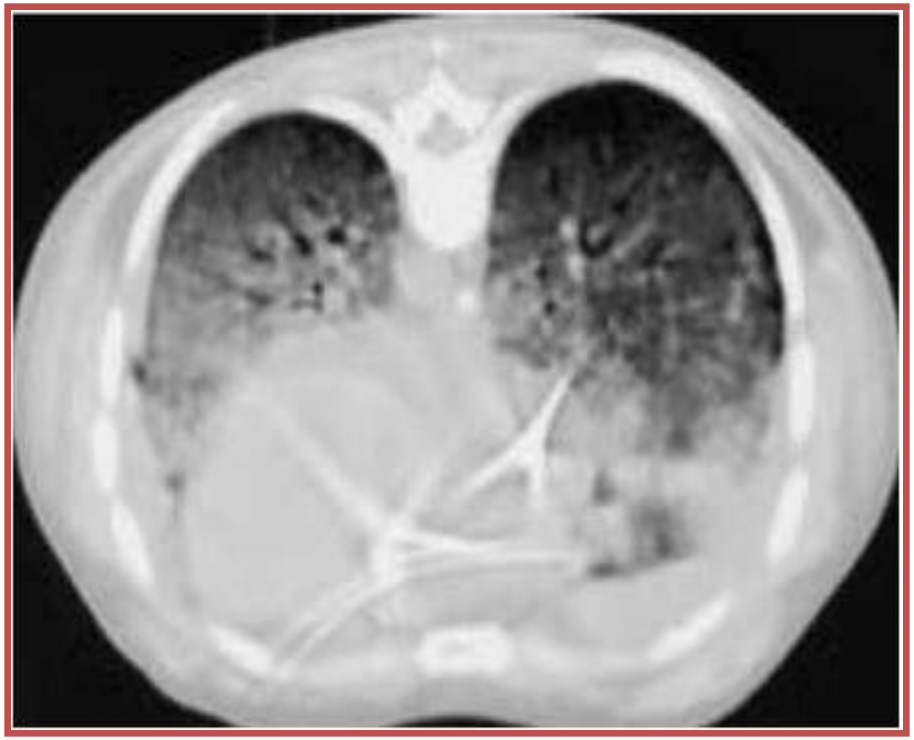
Décubitus Ventral (DV)



DV: rationnel physiopathologique

Meilleure distribution de la ventilation vers les régions dorsales



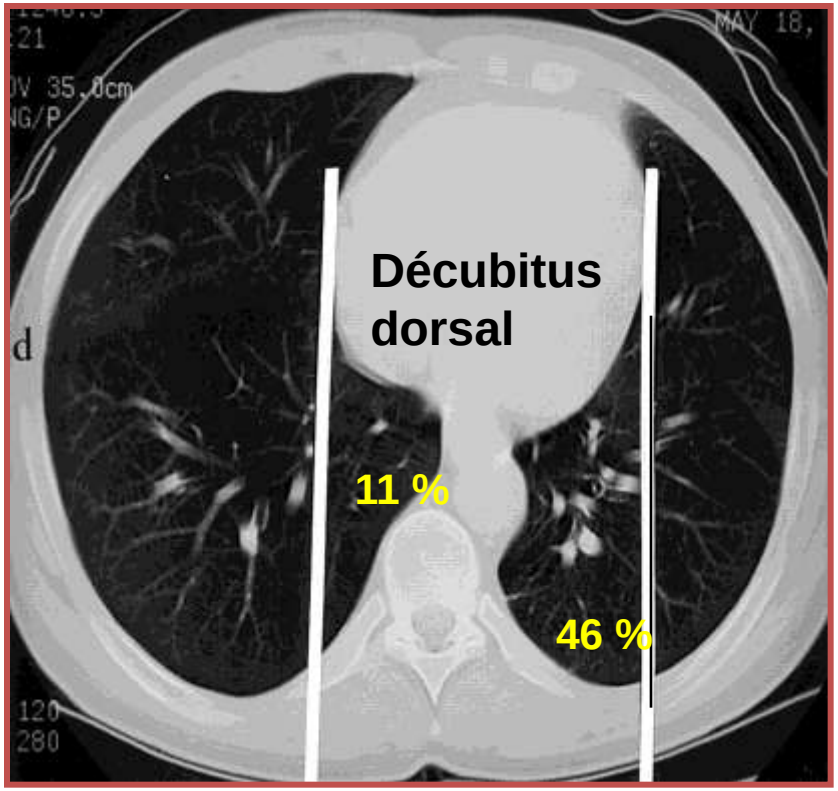


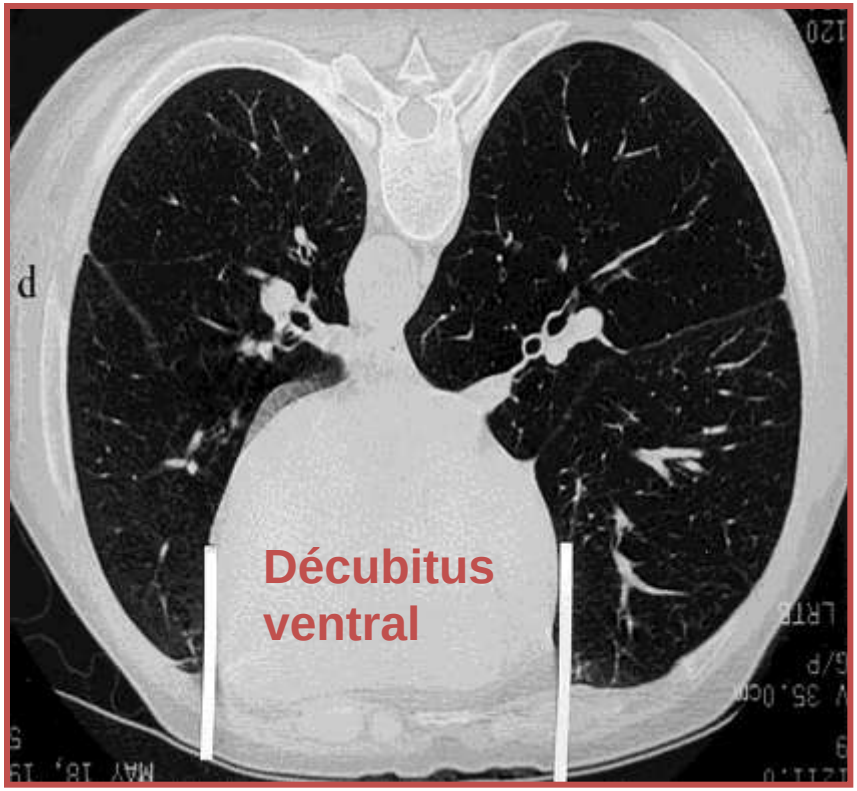
Pelosi, Eur Resp J 2002

DV: rationnel physiopathologique

Le DV élimine la compression exercée par le cœur sur les lobes inférieurs

% du lobe inférieur situé sous le coeur





Albert & Hubmayr, AJRCCM , 161: 1660-1665, 2000

DV: rationnel physiopathologique

- Moins de surdistension des espaces aériens (réduction des VILI)
- Facilite la course diaphragmatique
- Augmentation de la CRF
- Amélioration de la mécanique pulmonaire.
- Favorise le drainage des sécrétions (effet préventif sur les PAVM)

Etudes négatives: sévérité variable, patients hétérogènes (BPCO) et durées variables.

Trial	Number and type of Patients	PaO2/FiO2 (mmHg)	Prone (n)	Supine (n)	Prone duration (hours)	Cross over allowed
Gattinoni 2001	304 ARDS/ALI	127	152	152	7±1.8	Yes
Guerin 2004	791 ARF	152	413	378	8 (7.7;9.8)	Yes
Curley 2005	101 ALI	99.5	51	50	18±4	No
Voggenreiter 2005	40 ARDS/ALI	221	21	19	11±5	No
Mancebo 2006	136 severe ARDS	146	76	60	17	Yes
Total/Mean	1372	149±45	713	659	17	Yes

Méta-analyses DV

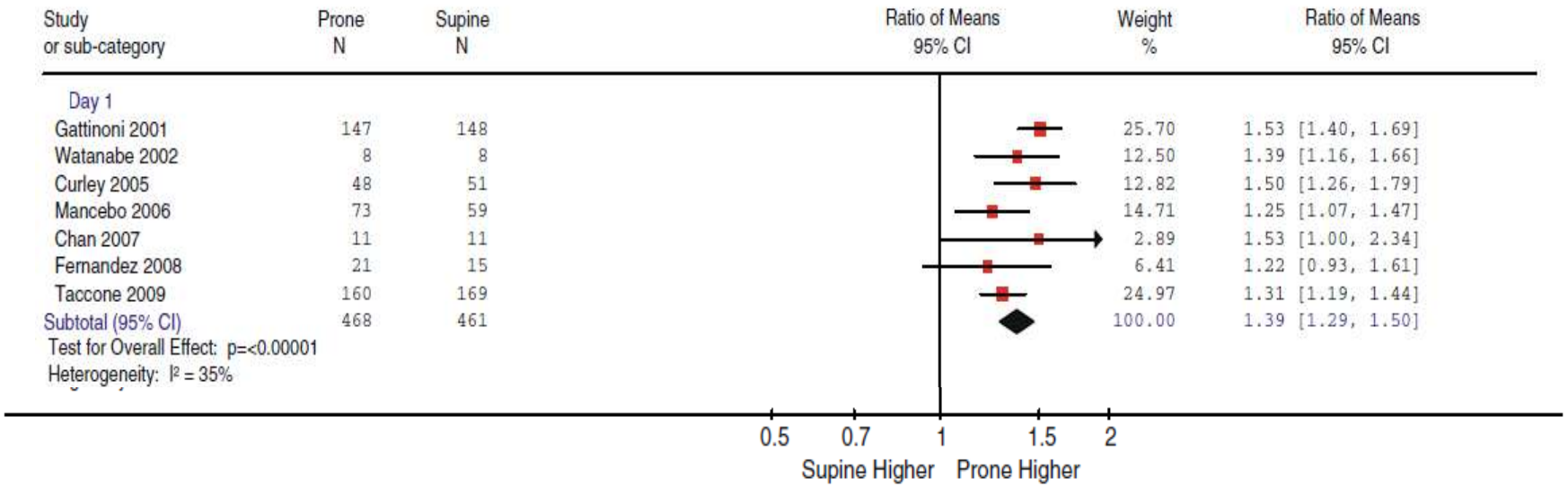
Intensive Care Med (2010) 36:585–599
DOI 10.1007/s00134-009-1748-1

REVIEW

Sachin Sud
Jan O. Friedrich
Paolo Taccone
Federico Polli
Neill K. J. Adhikari
Roberto Latini
Antonio Pesenti
Claude Guérin
Jordi Mancebo
Martha A. Q. Curley
Rafael Fernandez
Ming-Cheng Chan
Pascal Beuret
Gregor Voggenreiter
Maneesh Sud
Gianni Tognoni
Luciano Gattinoni

Prone ventilation reduces mortality in patients with acute respiratory failure and severe hypoxemia: systematic review and meta-analysis

Effets du DV sur l'oxygénation (PaO2)

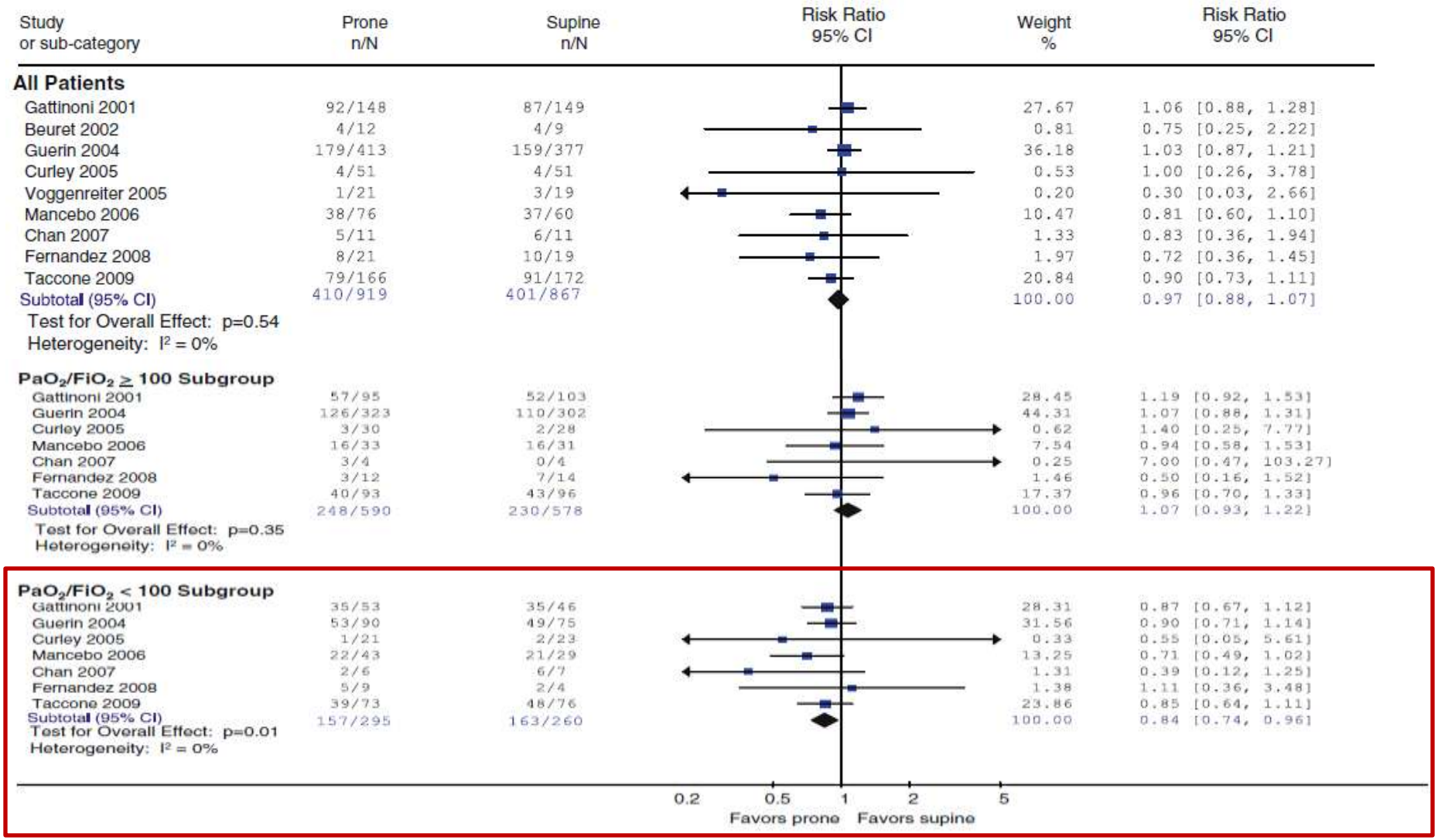


DV: effets indésirables

Table 2 Adverse events

Adverse events	Trials (patients, events)	Treatment effect	
		Risk ratio [95% CI]	*p*-Value
Ventilator-associated pneumonia	7 (1,066, 242)	0.81 [0.67, 1.00]	0.05
Pressure ulcers	6 (1,279, 620)	1.29 [1.16, 1.44]	<0.00001
Endotracheal tube obstruction	7 (1,351, 184)	1.58 [1.24, 2.01]	<0.001
Unplanned extubation or endotracheal tube dislodgement[a]	10 (1,813, 155)	1.07 [0.69, 1.65]	0.77
Unplanned removal of central or arterial lines	8 (886, 59)	1.49 [0.42, 5.27]	0.54
Thoracostomy tube dislodgement	8 (886, 17)	3.14 [1.02, 9.69]	0.05
Pneumothorax	7 (1,167, 67)	0.75 [0.47, 1.20]	0.23
Cardiac arrests	7 (1,031, 164)	0.96 [0.73, 1.26]	0.77

Effets du DV sur la mortalité



Abroug et al. Critical Care 2011, 15:R6
http://ccforum.com/content/15/1/R6

CRITICAL CARE

RESEARCH Open Access

An updated study-level meta-analysis of randomised controlled trials on proning in ARDS and acute lung injury

Fekri Abroug[1*], Lamia Ouanes-Besbes[1], Fahmi Dachraoui[1], Islem Ouanes[1], Laurent Brochard[2,3,4]

Group by Patient Type	Study name	Odds ratio	Lower limit	Upper limit	p-Value
ALI/ARDS	Gattinoni_2001	1,111	0,709	1,742	0,646
ALI/ARDS	Guerin_2004	1,045	0,775	1,410	0,772
ALI/ARDS	Voggenreiter_2005	0,267	0,025	2,815	0,272
All studies with ALI/ARDS		1,049	0,819	1,344	0,706
ARDS	Mancebo_2006	0,548	0,276	1,087	0,085
ARDS	Chan_2007	0,593	0,078	4,498	0,613
ARDS	Fernandez_2008	0,554	0,157	1,952	0,358
ARDS	Taccone_2009	0,810	0,530	1,238	0,330
All studies with ARDS		0,708	0,503	0,997	0,048
Overall		0,916	0,750	1,120	0,392

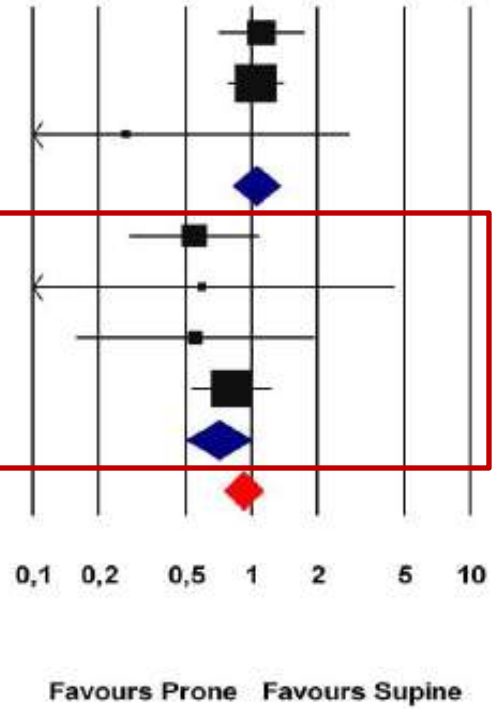


Figure 3 Effects of prone ventilation on intensive care unit mortality. Point estimates (by random-effects model) are reported separately for the groups of studies that included both acute lung injury (ALI) and acute respiratory distress syndrome patients (ARDS), those that included only ARDS patients, and the pooled overall effects of all meta-analysis-included patients. CI, confidence interval.

Etude PROSEVA (C Guérin et al)

Essai randomisé multicentrique (27 centres) Janvier 2008-Juillet 2011.

Inclusion

- >18 ans, intubés≥36 heures

- SDRA selon consensus AECC

-Sévérité: PaO2/FiO2<150 mmHg, PEP ≥5 mmHg, FiO2 ≥50%

Protocole

-≥ 16 h consécutives DV (débuté dans l'heure suivant la randomisation)

-Vt=6 ml/kg poids prédit, table PEP/FiO2 (NEJM 2000), Pplat≤30 cmH2O

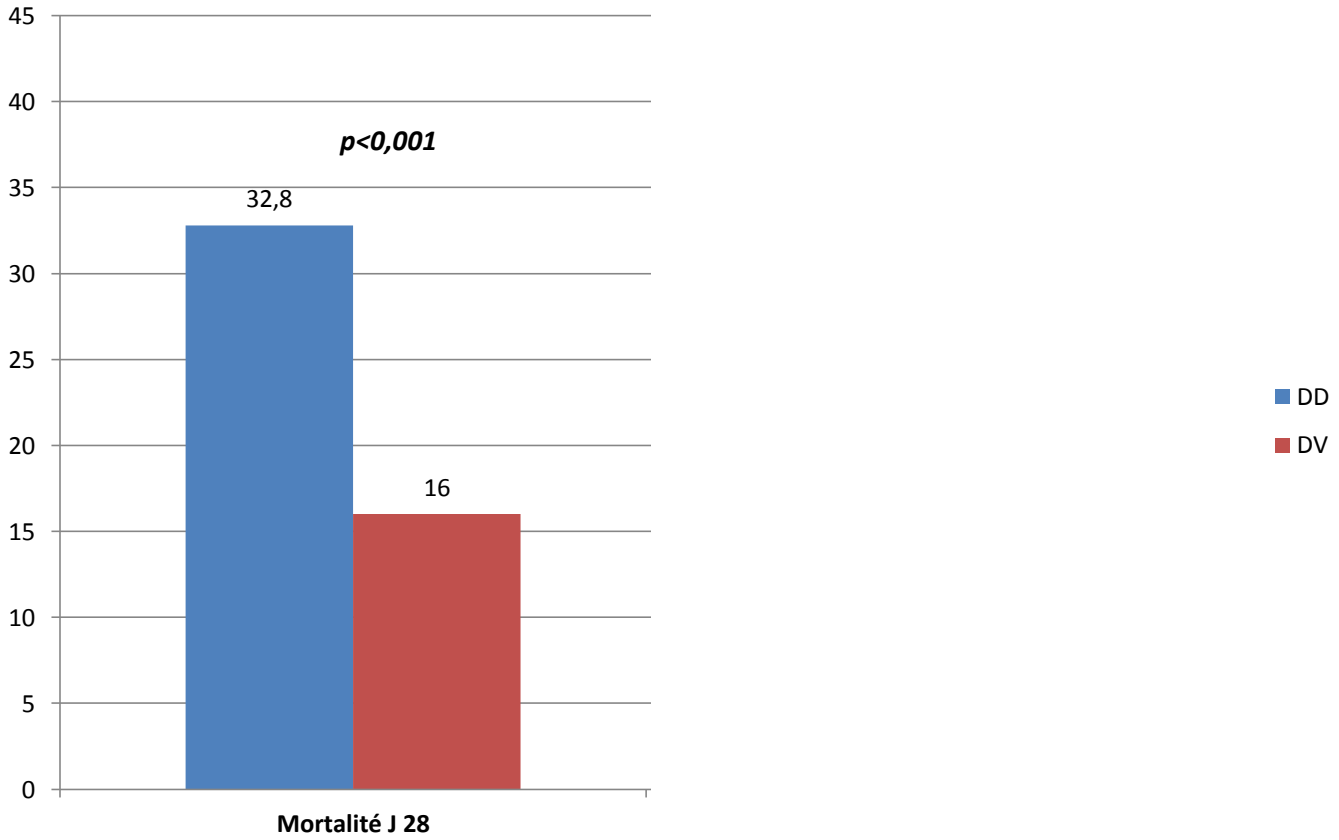
Critère de jugement principal: mortalité J28

Critères secondaires: Mortalité J90, l'incidence des PAVM

Etude PROSEVA (C Guérin et al)

	DD n=229	DV n=237
Age	60±16	58±16
IGS II	47±17	45±15
Sepsis	85,2	82,2
SOFA	10,4±3,4	9,6±3,2 *
Immunodépression	16,6	13,5
P/F	100±20	100±30
Délai intubation-randomisation (heures)	31,4±25,6	33,0±23,9

Etude PROSEVA (C Guérin et al)



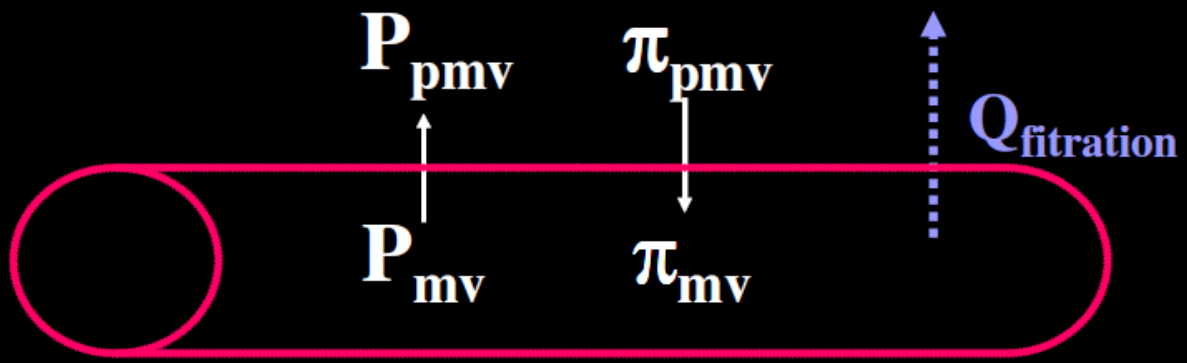
Après ajustement sur le SOFA, le RR de décès avec le DV est de:

- 0,42 [0,26-0,66] à J28
- 0,48 [0,32-0,72] à J90.

Gestion du bilan hydrique

Gestion du bilan hydrique

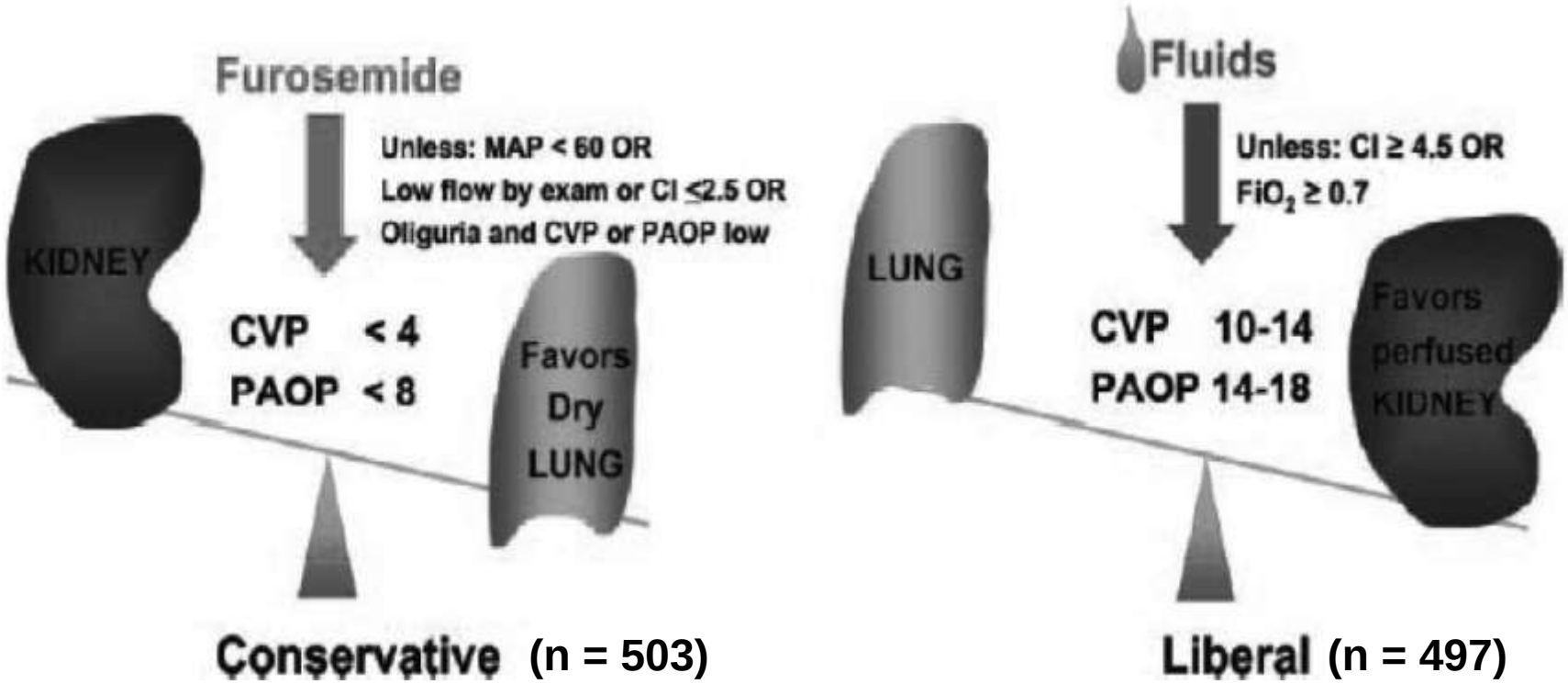
Filtration transendothéliale: la loi de Starling



$$Qf = Kf\,[(P_{mv}-P_{pmv}) - \sigma(\pi_{mv}-\pi_{pmv})]$$

P. hydrostatique — P. oncotique

Gestion du bilan hydrique



Bilan hydrique 7 jours:	**-136±491ml**	**VS**	**6992±502ml**

FACCT study N Engl J Med 2006; 354:2564–2575

Gestion du bilan hydrique

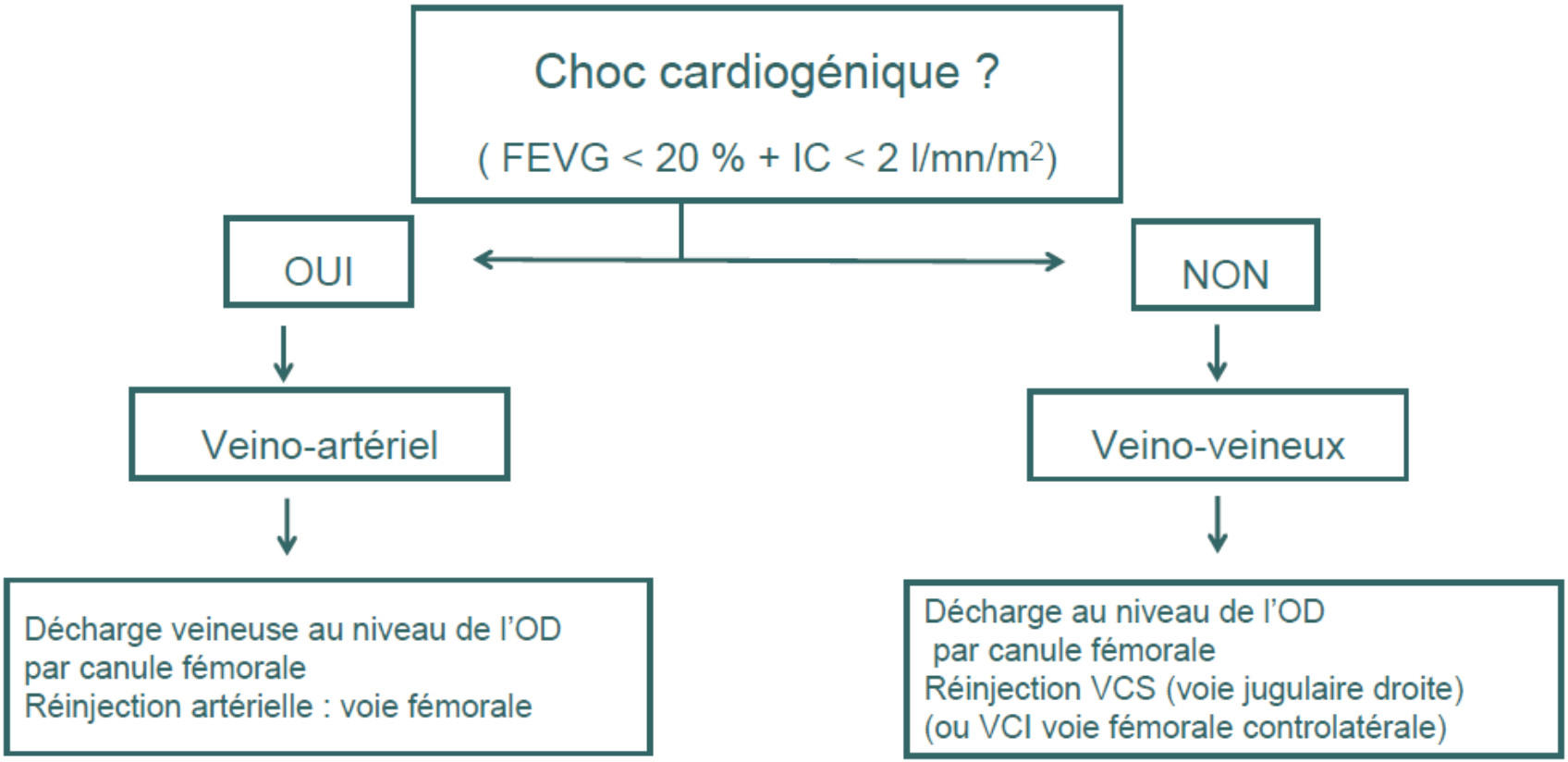
Table 3. Main Outcome Variables.*

Outcome	Conservative Strategy (n = 503)	Liberal Strategy (n= 497)	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

FACCT study *N Engl J Med 2006; 354:2564–2575*

Extracorporeal Membrane Oxygenation (ECMO)

ECMO : Quelle technique ?



(ECMO) Les études « historiques »

ECMO machine

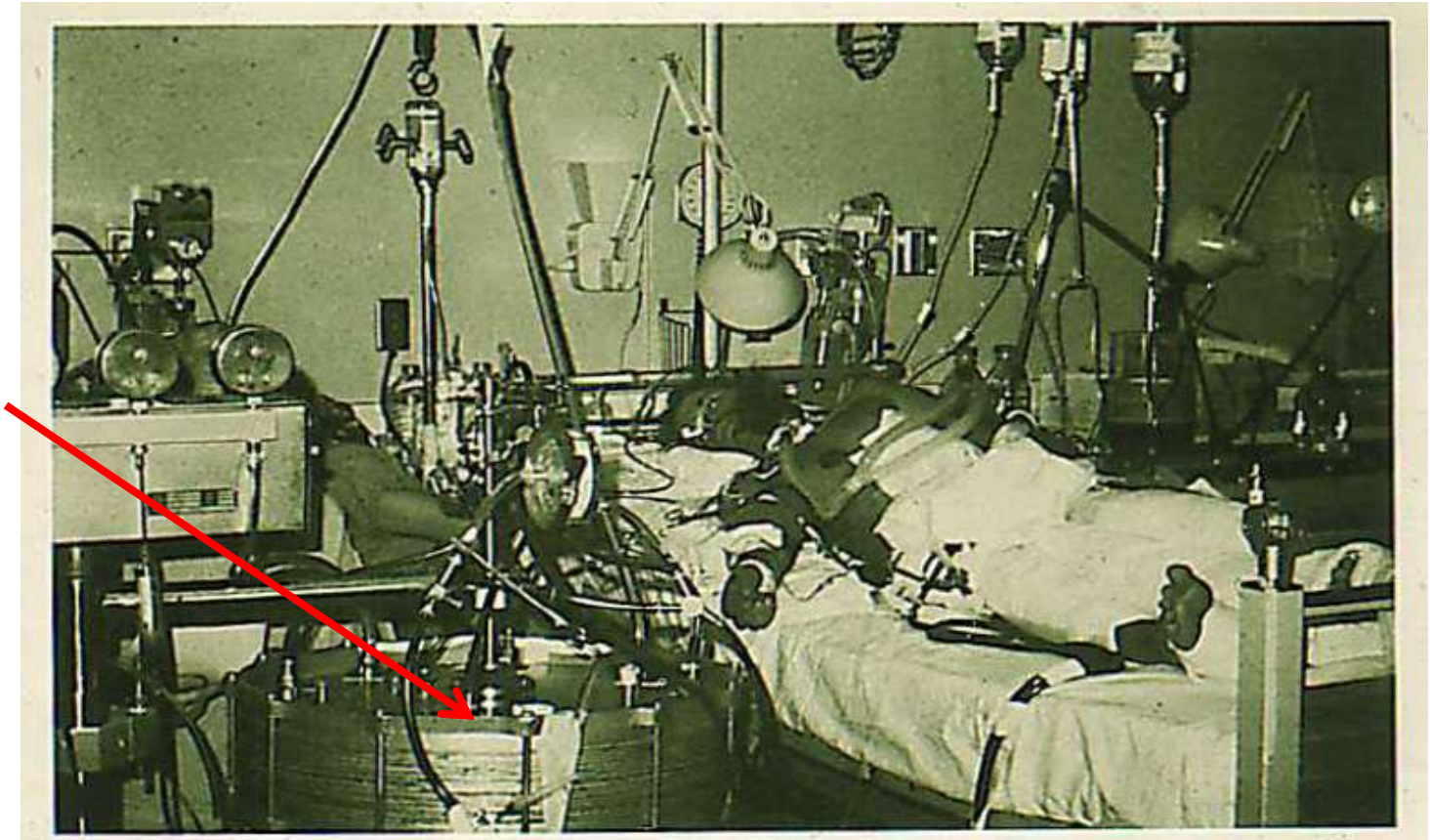


FIGURE 3.4 The first successful extracorporeal life support patient, treated by J. Donald Hill using the Bramson oxygenator (foreground), Santa Barbara, 1971.

ECMO

Extracorporeal Membrane Oxygenation in Severe Acute Respiratory Failure

A Randomized Prospective Study

Zapol, W *JAMA* 242:2193-2196, 1979

- Nine medical centers
- Prospective randomized trial to evaluate ECMO for ARDS
- 90 adult patients selected
  - Mechanical ventilation + venoarterial ECMO (42 patients)
  - Conventional mechanical ventilation (48 patients)
- Four patients in each group survived
- Patients died of:
  - Progressive reduction of transpulmonary gas exchange
  - Decreased compliance due to diffuse pulmonary inflammation, necrosis, and fibrosis
- « ECMO can support respiratory gas exchange but did not increase the probability of survival for severe ARDS

ECMO usage pratique

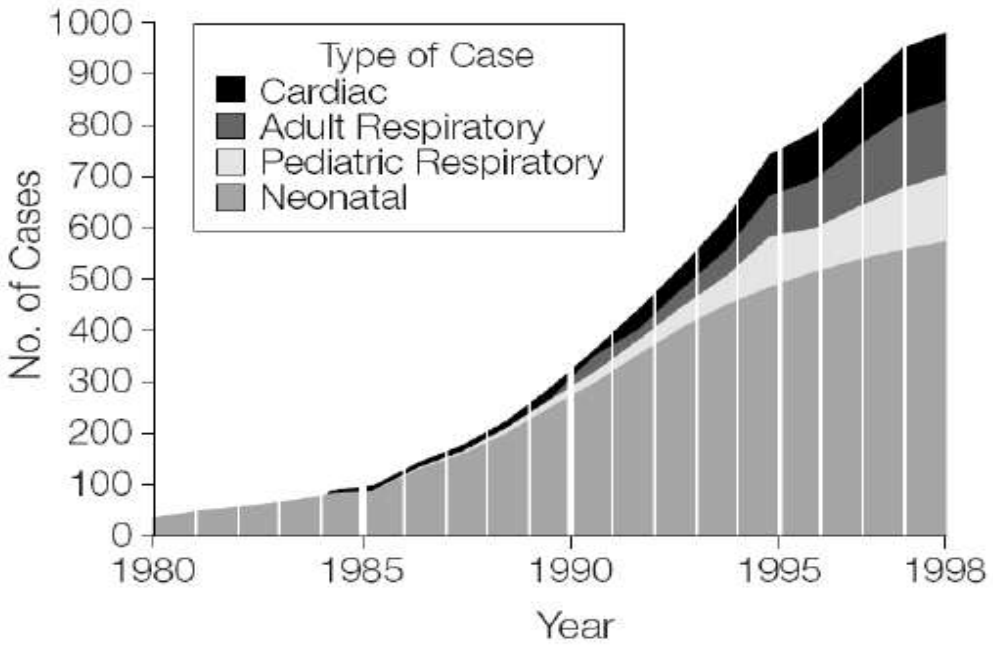
Extracorporeal Life Support

The University of Michigan Experience

Robert H. Bartlett, MD
Dietrich W. Roloff, MD
Joseph R. Custer, MD
John G. Younger, MD
Ronald B. Hirschl, MD

JAMA. 2000;283:904-908

Figure. University of Michigan Extracorporeal Life Support Cases



ECMO: étude CESAR

Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial

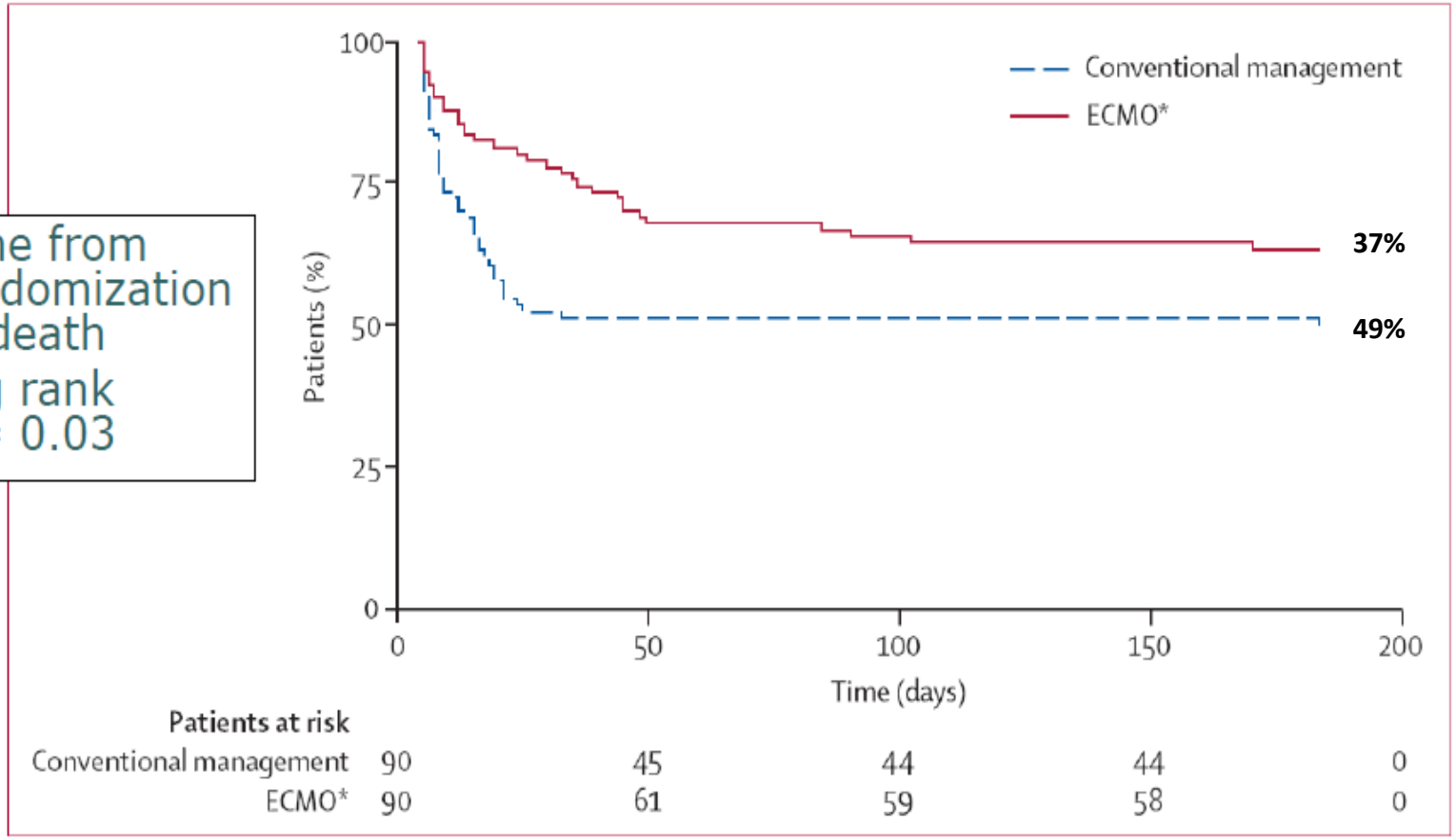
Giles J Peek, Miranda Mugford, Ravindranath Tiruvoipati, Andrew Wilson, Elizabeth Allen, Mariamma M Thalanany, Clare L Hibbert, Ann Truesdale, Felicity Clemens, Nicola Cooper, Richard K Firmin, Diana Elbourne, for the CESAR trial collaboration

www.thelancet.com **Published online September 16, 2009**

ECMO réalisée dans des centres « experts » (n=90) versus VM « conventionnelle » (n=90).

ECMO: étude CESAR

- Time from randomization to death
- Log rank p = 0.03



ECMO: étude CESAR

	ECMO group (n=90)*†	Conventional management group (n=90)	p value
Treatment by other management			
Missing all data	2 (2%)	0	NA
High-frequency oscillation or jet ventilation	6 (7%)	13 (14%)	0·21
Nitric oxide	9 (10%)	6 (7%)	0·60
Prone position	32 (4%)	38 (42%)	0·58
Steroids	76 (84%)	58 (64%)	0·001
MARS	15 (17%)	0	<0·0001
Continuous venovenous haemofiltration	72 (80%)	76 (84%)	0·61
Treatment by low-volume low-pressure ventilation strategy at any time	84 (93%)	63 (70%)	<0·0001
Time under strategy (days)	23·9 (20·4)	15·0 (21·1)	<0·0001

Pas de « standardisation » de la VM dans le groupe contrôle

ECMO et grippe A H1N1

CARING FOR THE
CRITICALLY ILL PATIENT

JAMA. 2011;306(15):1659-1668

Mortalité: 23.7%

ONLINE FIRST

Referral to an
Membrane Ox
and Mortality
With Severe 2

CARING FOR THE
CRITICALLY ILL PATIENT

JAMA-EXPRESS

JAMA. 2009;302(17):1888-1895

Mortalité: 21%

Extracorporeal Membrane Oxygenation for 2009 Influenza A(H1N1) Acute Respiratory Distress Syndrome

The Australia and New Zealand Extracorporeal Membrane Oxygenation (ANZ ECMO) Influenza Investigators*

Context The novel influenza A(H1N1) pandemic affected Australia and New Zealand during the 2009 southern hemisphere winter. It caused an epidemic of critical illness and some patients developed severe acute respiratory distress syndrome (ARDS) and were treated with extracorporeal membrane oxygenation (ECMO).

Curares

Curares

ACURASYS

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 SEPTEMBER 16, 2010 VOL. 363 NO. 12

Neuromuscular Blockers in Early Acute Respiratory Distress Syndrome

Laurent Papazian, M.D., Ph.D., Jean-Marie Forel, M.D., Arnaud Gacouin, M.D., Christine Penot-Ragon, Pharm.D., Gilles Perrin, M.D., Anderson Loundou, Ph.D., Samir Jaber, M.D., Ph.D., Jean-Michel Arnal, M.D., Didier Perez, M.D., Jean-Marie Seghboyan, M.D., Jean-Michel Constantin, M.D., Ph.D., Pierre Courant, M.D., Jean-Yves Lefrant, M.D., Ph.D., Claude Guérin, M.D., Ph.D., Gwenaël Prat, M.D., Sophie Morange, M.D., and Antoine Roch, M.D., Ph.D., for the ACURASYS Study Investigators*

- PaO2/FiO2<150

- Cisatracrium (Nimbex ®)37,5 mg/h/48 premières heures

Table 2. Baseline Characteristics of the Patients, According to Study Group.*

Characteristic†	Cisatracurium (N=177)	Placebo N=162)	P Value
Age — yr	58±16	58±15	0.70
Tidal volume — ml/kg of predicted body weight	6.55±1.12	6.48±0.92	0.52
Minute ventilation — liters/min	10.0±2.5	10.1±2.2	0.83
PEEP applied — cm of water	9.2±3.2	9.2±3.5	0.87
Plateau pressure — cm of water	25.0±5.1	24.4±4.7	0.32
Respiratory-system compliance — ml/cm of water	31.5±11.6	31.9±10.7	0.71
F_IO_2	0.79±0.19	0.77±0.20	0.33
PaO_2:F_IO_2‡	106±36	115±41	0.03
pH	7.31±0.10	7.32±0.10	0.11
PaO_2 — mm Hg	80±24	85±28	0.09
$PaCO_2$ — mm Hg	47±11	47±11	0.62
Prone position or inhaled nitric oxide or almitrine mesylate — no. (%)	33 (18.6)	23 (14.2)	0.31
SAPS II§	50±16	47±14	0.15
Nonfatal condition according to McCabe–Jackson score — no. (%)¶	133 (75.1)	125 (77.2)	0.66
Main reason for ICU admission — no. (%)			
Medical	129 (72.9)	113 (69.8)	0.52
Surgical, emergency	27 (15.3)	31 (19.1)	0.34
Surgical, scheduled	21 (11.9)	18 (11.1)	0.83
Corticosteroids for septic shock — no. (%)	70 (39.5)	73 (45.1)	0.30
Direct lung injury — no. (%)	142 (80.2)	123 (75.9)	0.34

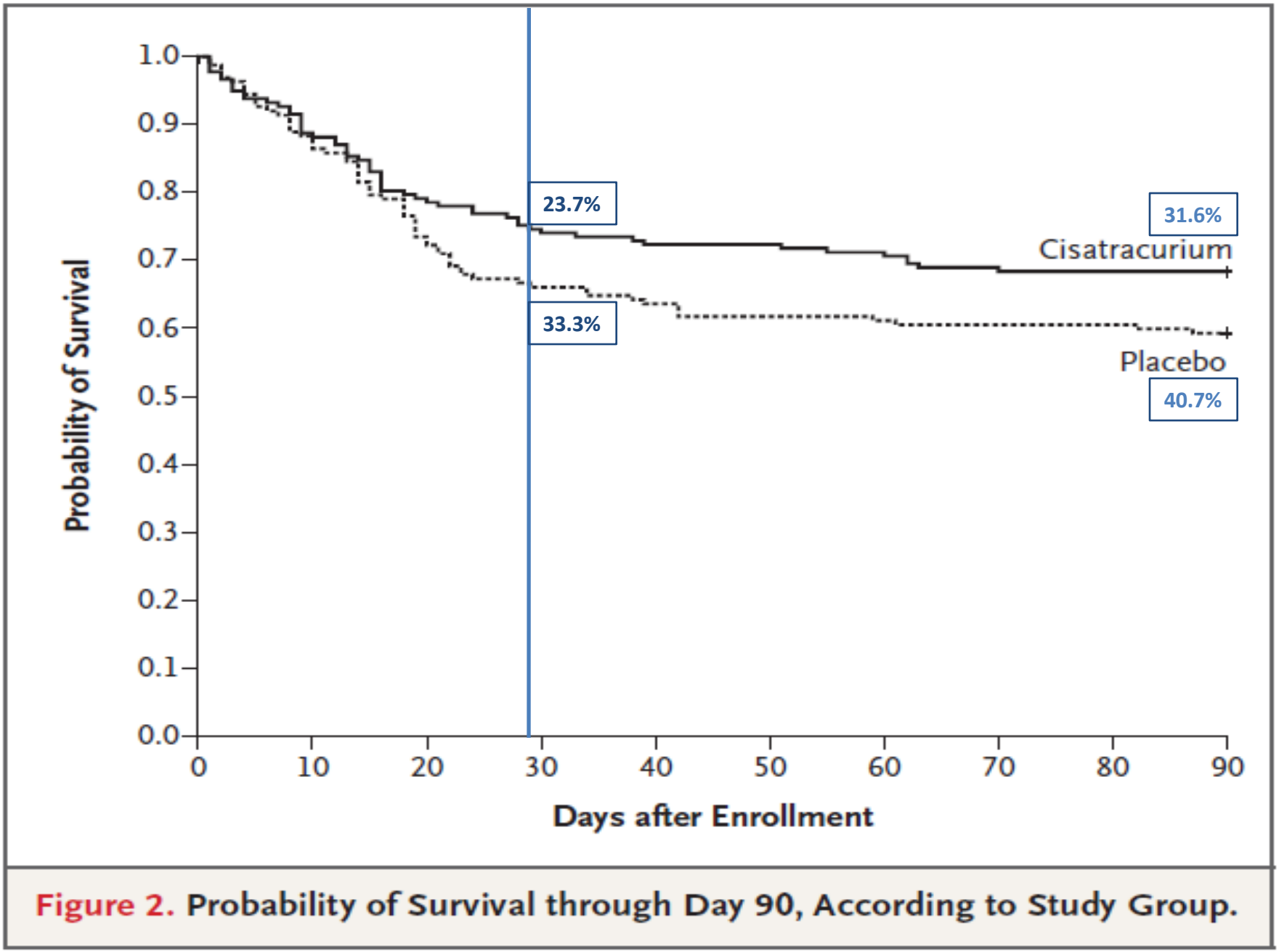


Figure 2. Probability of Survival through Day 90, According to Study Group.

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

Corticostéroïdes

Corticoïdes- rationnel:

Efficacité supposée des corticoïdes par :

- Diminution de l'activité collagénase des macrophages

  (dépôt de fibres de collagène type 1 et 3).

- Diminution de la fibroprolifération.

- Fabrication des pneumocytes de type 2.

Loletta Lancet 2003 ;361:1615-17

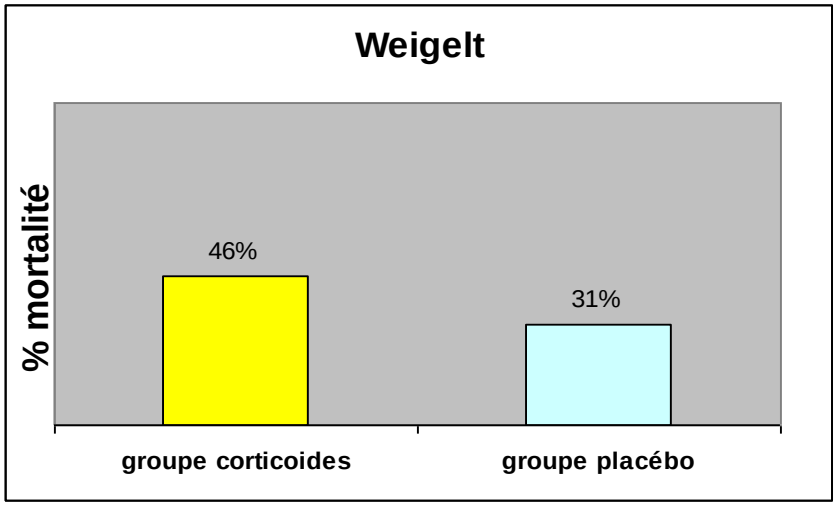
Corticoïdes

- Efficacité prouvée dans certaines étiologies pouvant être responsable de SDRA

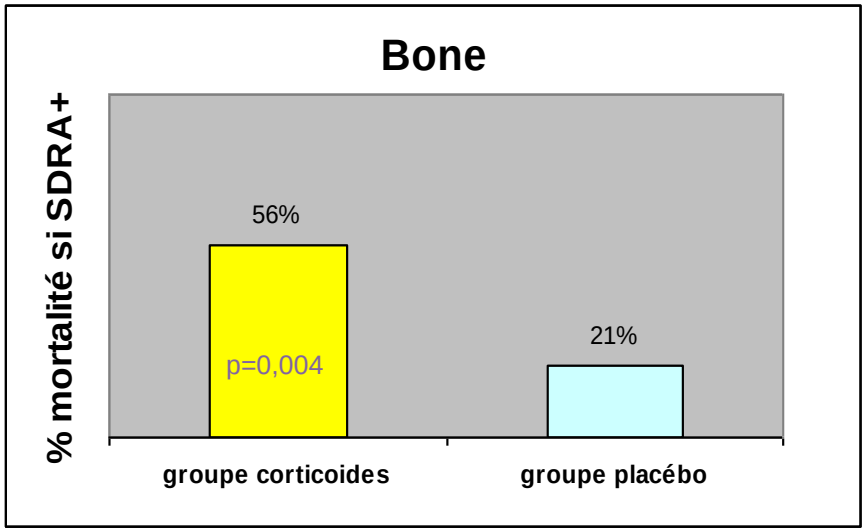
  - Pneumonie à pneumocystis carinii — *Masur et al N Engl J Med 1990, 323: 1500-1504*
  - Pneumonie à éosinophile — *Allen Am J Respir Crit Care Med 150:1423-1438*
  - Embolie graisseuse — *Schonfeld Ann Intern Med 1983, 99 (4) :438-443*)
  - Bronchiolite oblitérante — *Jantz Am J Respir Crit Care Med 160:1079-1100*

- SDRA secondaire aux autres étiologies?

A la phase précoce ou en prévention du SDRA

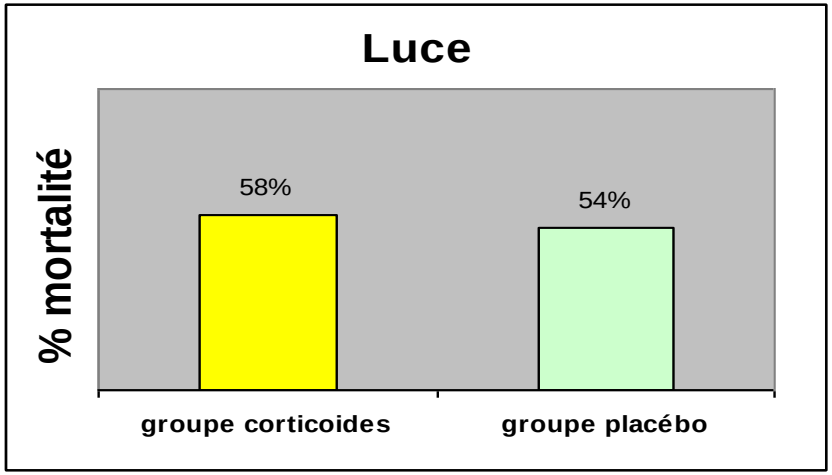


Weigelt et al ***Arch Surg 1985;120:536-540***



Bone et al ***Chest 1987,92:1032-1036***

Pas d'amélioration de la survie si corticoïdes utilisés à la phase précoce de l'agression voir même: Augmentation de la mortalité!



Luce et al ***Am Rev Resp Dis 1988;138:62-68***

Protocoles: Doses élevés ; faibles doses?

Ferguson ND: ACP Journal Club. Review: Low-dose corticosteroids improve outcomes in acute lung injury and the acute respiratory distress syndrome. Ann Intern Med 2009; 151:JC3–JC12

Meduri JAMA 1998

	Corticoïdes(n=16)	Placebo (n=8)
Pa02/Fi02	262*	148
MODS	0,7*	1,8
LIS	1,7 *	3
Amélioration du LIS d'1 point (n)	16*	2
Durée de ventilation (j)	11,5*	23
Extubation(n)	7*	0
Mortalité en réanimation	0%*	62%
Mortalité intrahospitalière	12%*	62%

The NEW ENGLAND JOURNAL of MEDICINE

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Efficacy and Safety of Corticosteroids for Persistent Acute Respiratory Distress Syndrome

The National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network*

180 Patients

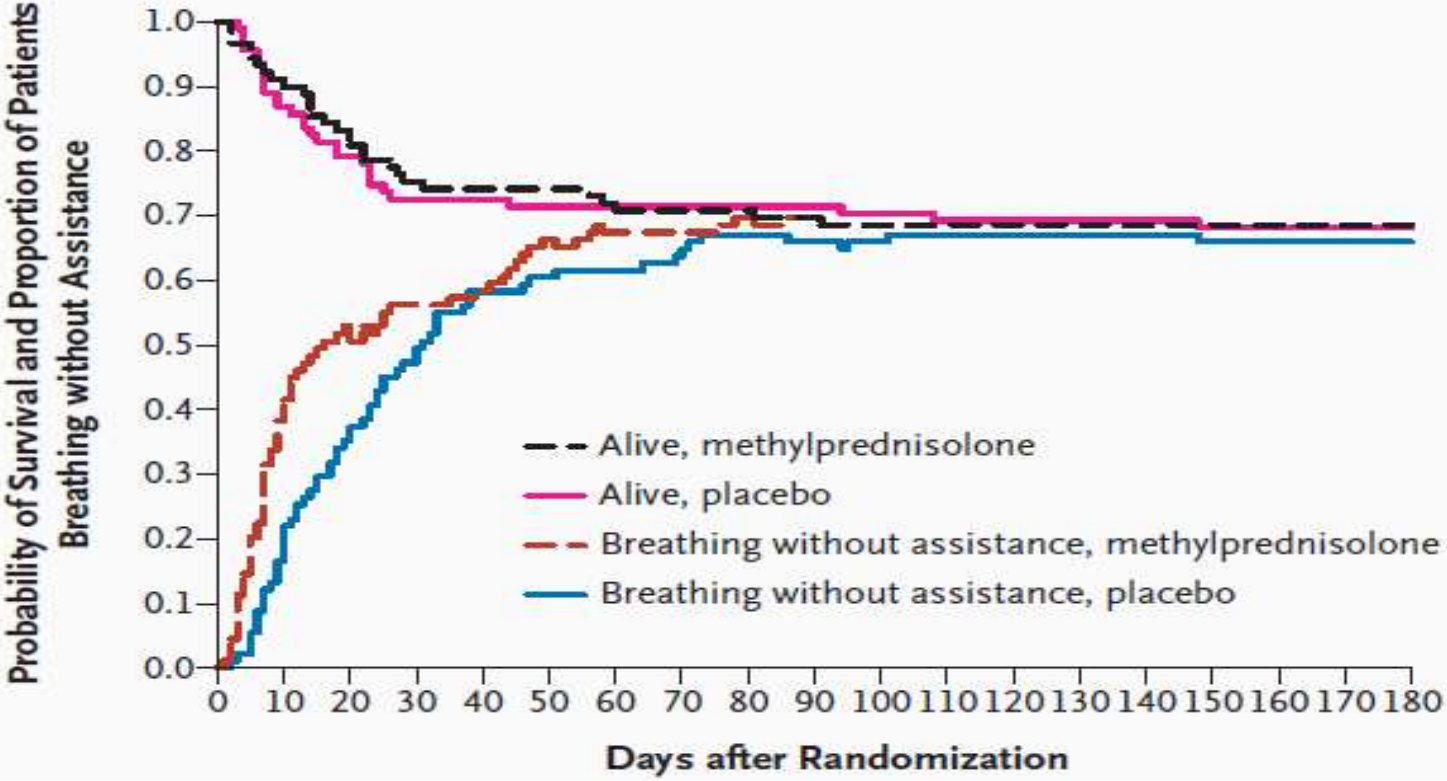


Figure 2. Probability of Survival and the Proportion of Patients with Persistent ARDS Who Became Able to Breathe without Assistance during the First 180 Days after Randomization.

At 180 days, 29 patients in the placebo group had died, 58 had been discharged home, and 4 had not been discharged home; the respective values in the methylprednisolone group were 28, 57, and 4. The status was known for all 180 patients at 180 days.

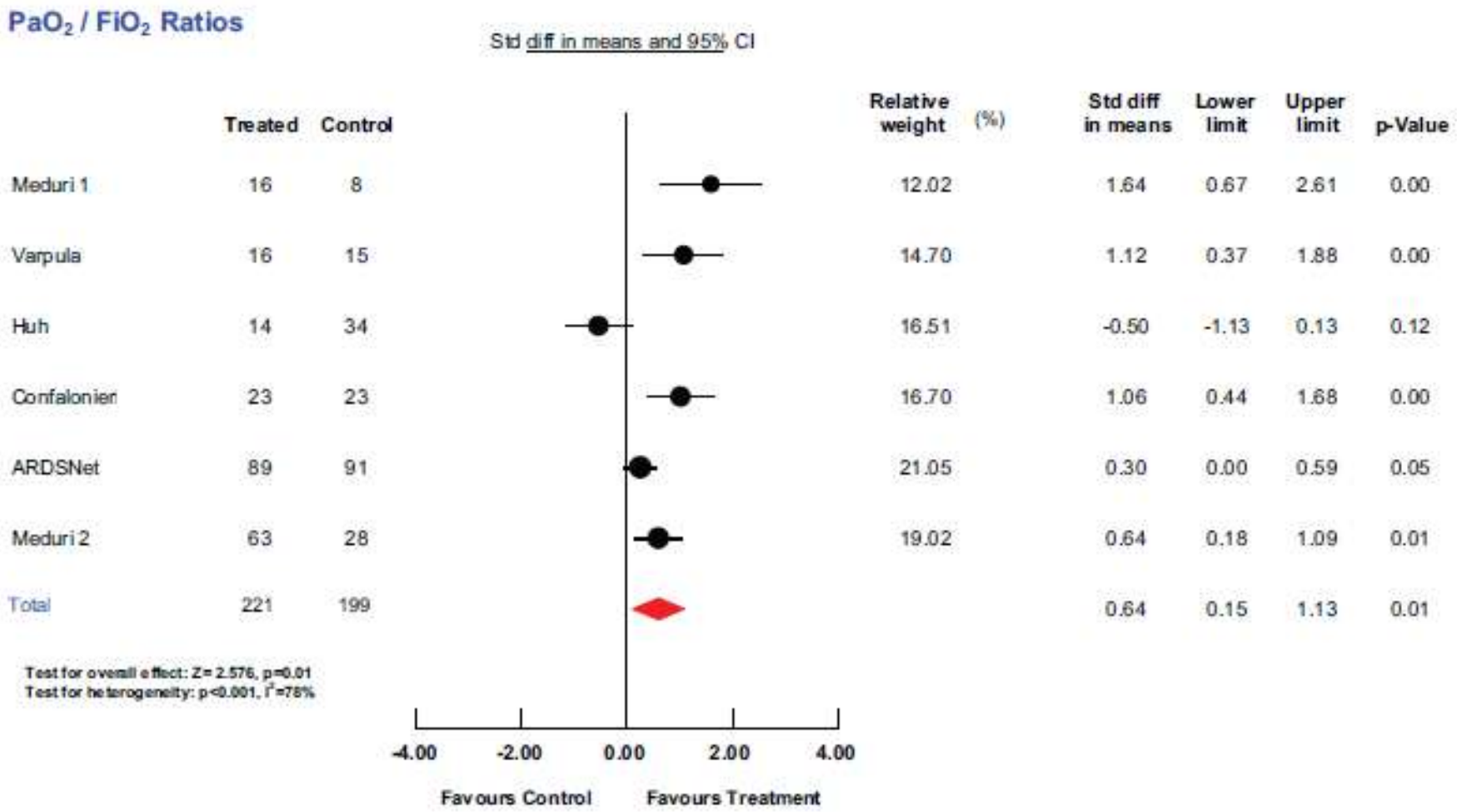
Continuing Medical Education Article

Use of corticosteroids in acute lung injury and acute respiratory distress syndrome: A systematic review and meta-analysis*

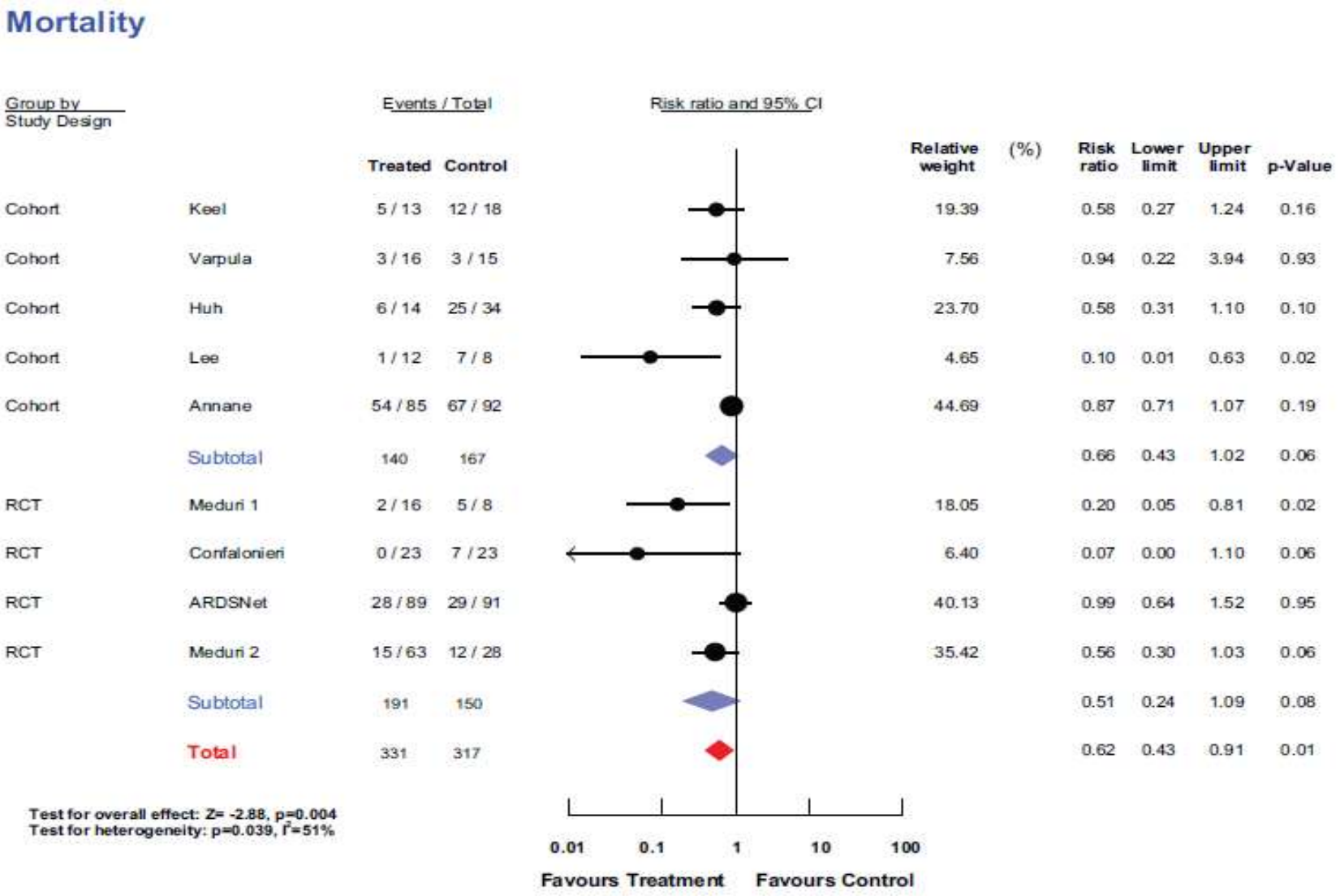
Benjamin M. P. Tang, PhD; Jonathan C. Craig, PhD; Guy D. Eslick, PhD; Ian Seppelt, MBBS; Anthony S. McLean, MBBS

Crit Care Med 2009 Vol. 37, No. 5

Effets sur l'oxygénation



Effets sur la mortalité

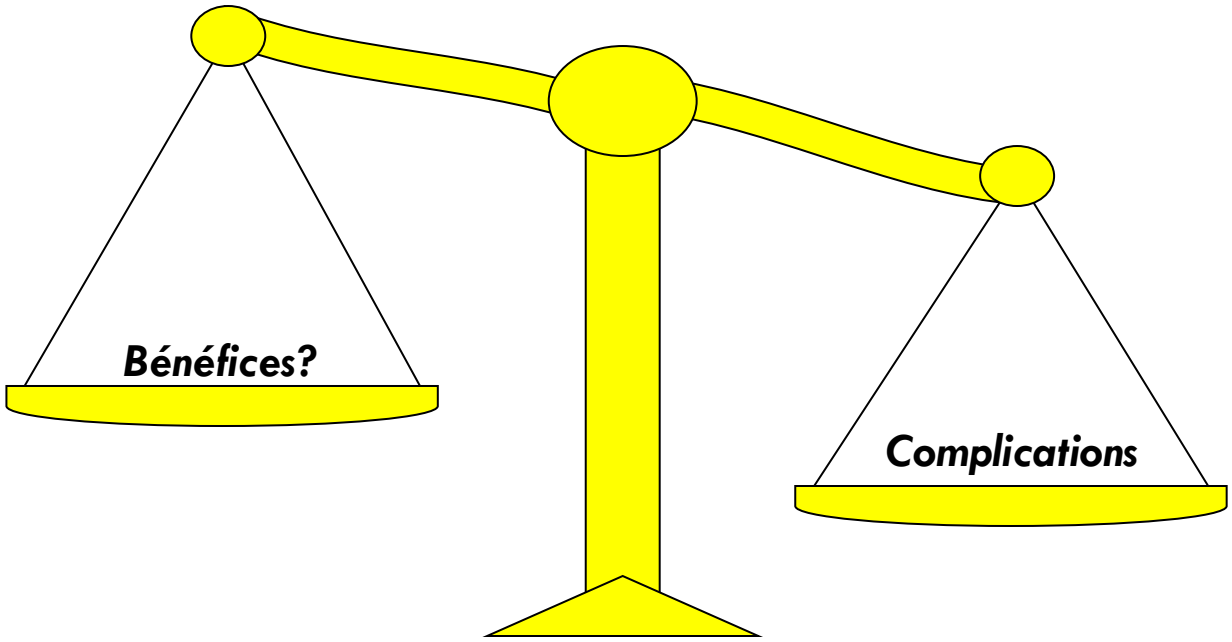


Corticoïdes et études récentes A H1N1 2009

Pays [Réf]	N	VMI	DV	NO	ECMO	Cortico	DVM j (IQR)	DRéa j (IQR)	DCD
ANZ [1]	722	64,6%	ND	11,6%	11,6%	18,4%	8 (4-16)	7,4 (3-16)	14,3%
Espagne [5]	32	75%	33%	ND	0	34,1%	10 (1-21)	ND	25%
Mexique [6]	58	83%	17,4%	ND	0	69%	15 (8-25)	13,5 (6-24)	41,4%
Canada [3]	168	76,2%	3%	14%	7%	50,6%	12 (6-20)	12 (5-20)	17,3%
USA [4]	67	65%	ND	ND	ND	52%	8 (4-16)	7,4 (3-16)	28,3%
ANZ [2]	252	79,7%	ND	ND	27%	ND	8 (4-14)	12 (7-18)	ND 15,4%*
ECMO [2]	*68*	*100%*	*20%*	*32%*	*100%*	*ND*	*25 (13-34)*	*27 (16-37)*	*20,5%*

Corticoïdes mais?

- Neuro-myopathie de réanimation
- Intolérance aux hydrates de carbones
- Risque infectieux

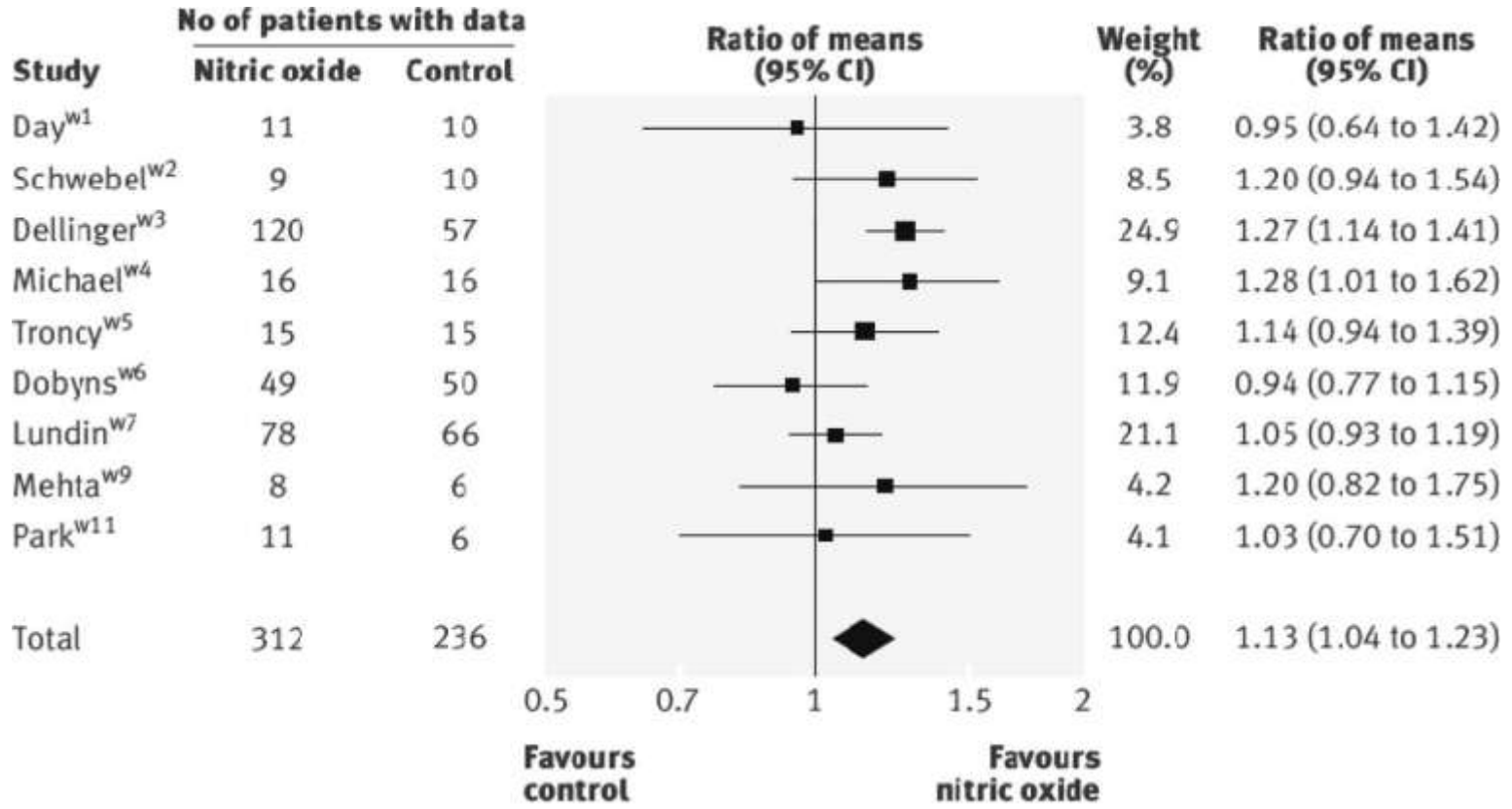


Vasodilatateurs artériels pulmonaires

Monoxyde d'azote (NO)

- Le NO: un vasodilatateur artériel pulmonaire sélectif,
- Vasodilatation artériolaire pulmonaire des zones ventilées dans lesquelles il diffuse (amélioration rapport ventilation–perfusion).
- Effets sont dose-dépendants, effet maximal entre 2 et 10 ppm chez les patients non-septiques et 10 à 20 chez les septiques.

Monoxyde d'azote (NO)



- **NO: Amélioration de l'oxygénation;**
- Il n'a jamais été mis en évidence un effet sur **la mortalité**.
- Le NO peut être utilisé lorsque l'hypoxémie est menaçante (pas en routine).

Adhikari NKJ et al: Effect of nitric oxide on oxygenation and mortality in acute lung injury: Systematic review and meta-analysis. BMJ 2007; 334:779.

Autres vasodilatateurs pulmonaires

- Prostacycline (PGI2)
- Alprostadil

Almitrine

Almitrine

- Un analeptique respiratoire qui majore la vasoconstriction pulmonaire hypoxique.
- Les posologies usuelles sont de 2 à 4 mcg/kg/min.
- Sur un plan physiopathologique, son association avec le NO est séduisante.
- EI: insuffisance hépatique aigue et neuropathie.
- L'HTAP sévère et/ou une défaillance ventriculaire droite sont des CI.
- Associée au NO ou en monothérapie, les études sur le devenir sont négatives,
- Son utilisation sera réservée aux hypoxémies réfractaires.

Autres traitements

Autres traitements Adjuvants

- Antioxydants
- Pentoxifylline
- Dépelestat (antiélastases)
- Kétoconazole
- Anticoagulants
- **Agonistes β2-Adrénergiques**

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, Zahid Khan, Sarah E Lamb, for the BALTI-2 study investigators

	Salbutamol (n=162)	Placebo (n=164)
Age (years)	55·8 (17·2)	54·2 (17·5)
Male sex	102 (63%)	110 (67%)
Height (cm)	168·8 (10·8)	169·0 (12·2)
APACHE II score	19·5 (6·2)	18·9 (6·7)
APACHE II predicted mortality	0·43 (0·20)	0·42 (0·21)
Tidal volume (mL/kg ideal bodyweight)	8·0 (1·7)	8·3 (1·9)
PaO_2/FiO_2 ratio (mm Hg)	103·5 (36·75)	103·5 (36·75)
100–200	82 (51%)	81 (49%)
51–99	74 (46%)	78 (48%)
≤50	6 (4%)	4 (2%)
Missing data	0	1
Cause of ARDS		
Direct lung injury	103 (64%)	105 (64%)
Smoke or toxin inhalation	1	2
Gastric content aspiration	6	9
Near drowning	1	0
Thoracic trauma	5	9
Pneumonia	86	79
Drug related	2	1
Other	2	5
Missing data	1	0
Indirect lung injury	58 (36%)	59 (36%)
Sepsis	39	47
Cardiopulmonary bypass	1	1
Pancreatitis	6	4
Non-thoracic trauma	2	6
Transfusion related	6	1
Other	4	0

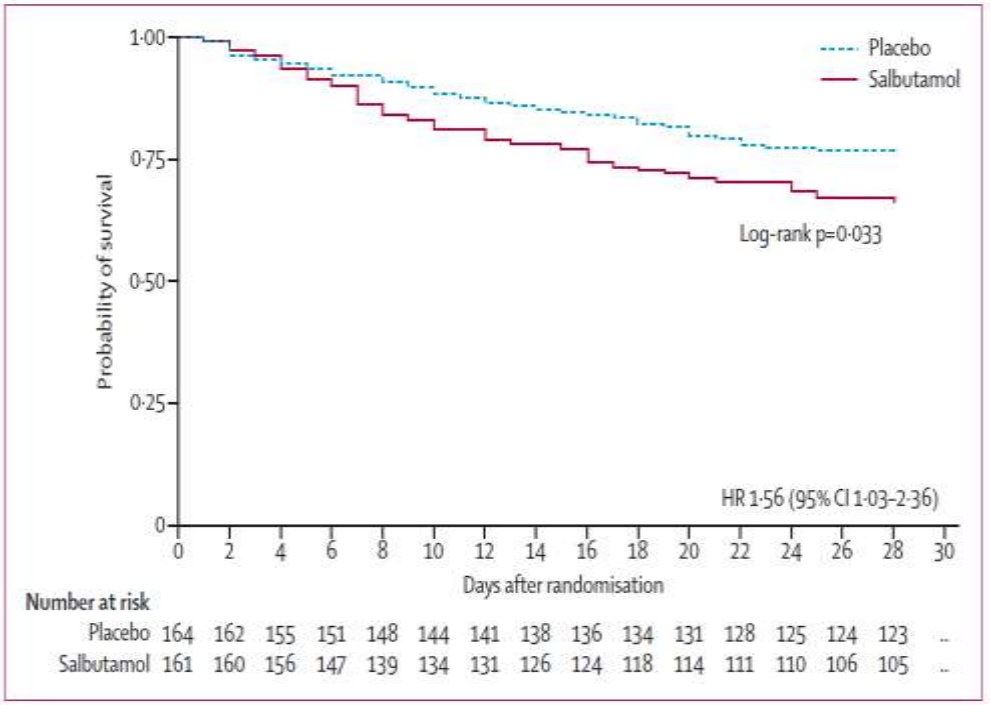
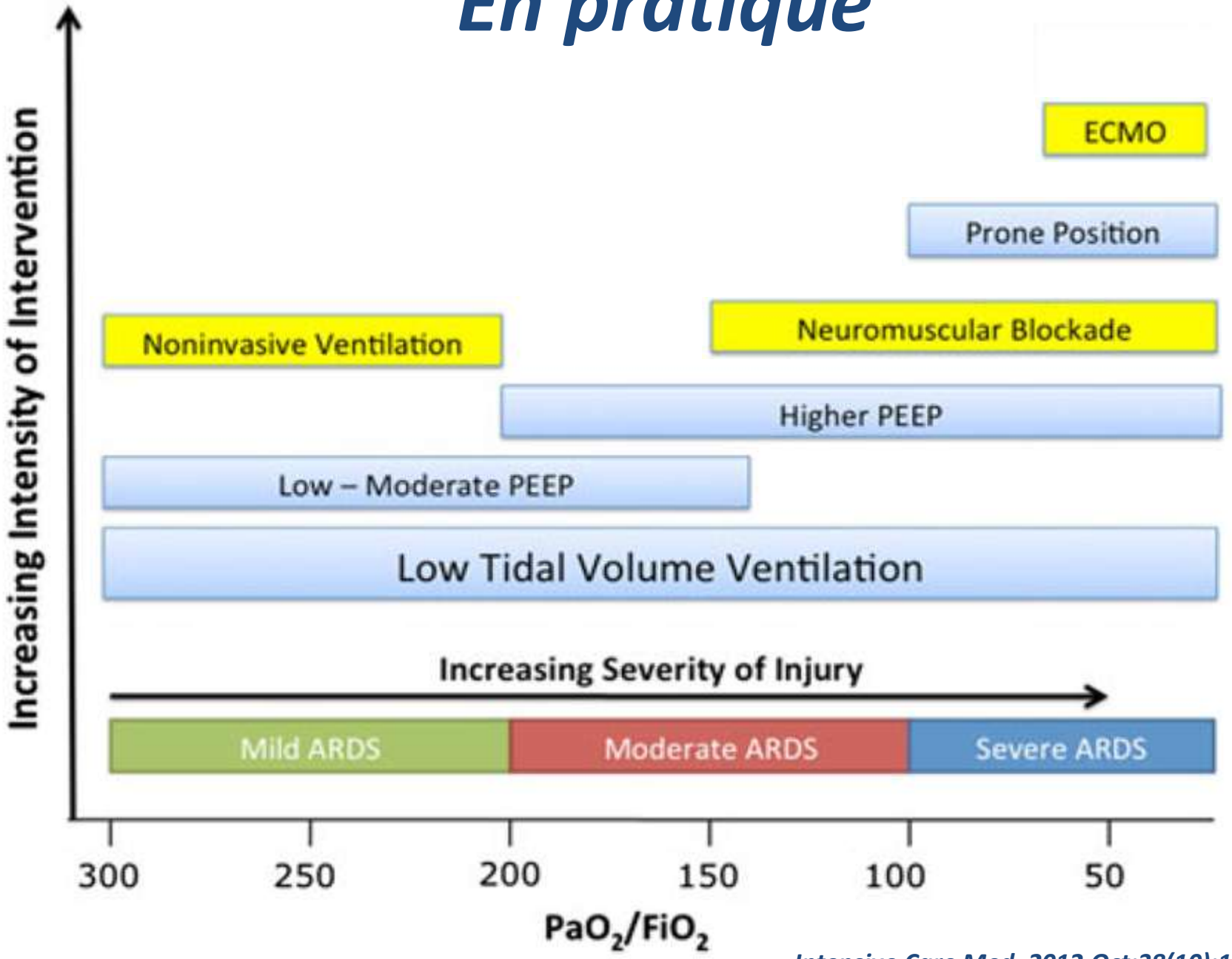


Figure 3: **Kaplan-Meier curves for mortality**
Rate of death in the two study groups up to 28 days after randomisation.

Prise en charge globale

- ***Traitement étiologique +++***
- Prévention des PAVM
- Thromboprophylaxie
- Prévention des complications de réanimation

En pratique



Intensive Care Med. 2012 Oct;38(10):1573-82

Points à retenir

- La prise en charge du SDRA en **dehors de la ventilation** a connue récemment des progrès **considérables.**
- Les **curares**, le **DV** et l'**ECMO** réduisent très probablement la mortalité.
- Les **corticoïdes** améliorent l'oxygénation mais l'effet sur la mortalité?
- Les autres moyens: « **rescue therapy** »: indications ponctuelles
- La molécule qui révolutionnera le SDRA ne semble pas encore avoir été découverte.

Merci