



ROYAUME DU MAROC
UNIVERSITE CADI AYYAD
FACULTE DE MEDECINE ET DE PHARMACIE
MARRAKECH

ACTUALITÉS DANS LE SDRA EN PÉDIATRIE

Pr S.YOUNOUS
chef de service d'anesthésie réanimation pédiatrique
CHU Mohamed VI Marrakech



Cas clinique

-Enfant de 2 ans , admise le 21/02/2019 aux urgences pour détresse respiratoire fébrile évoluant progressivement depuis 4 jours

-ATCD: 0

-Poids a 14 Kg

Examen a l'admission en Réanimation pédiatrique (H0) :

-GCS 14/15

-Polypnée a 80 cycles/min , SLR (BAN, tirage sous costal et sus sternal ,
balancement thoraco abdominal, entonnoir xiphoïdien), cyanose péribuccales ,
SpO2 a 72 % sous MHC, auscultation râles ronflants bilatéraux

-Tachycarde a 160 bat/min , TA:60/20mmHg TRC > 3s , marbrures généralisés,
froideur des extrémités

-Fébrile a 39°C , GC: 1,28g/l

CAT :

-Position $\frac{1}{2}$ assise + VNI via casque d'helmet FIO₂ (60%) debit 60l/min

-2 Remplissages a raison de 20 cc/kg de SS0,9% → échec → NAD
0,5µg/kg/min → amélioration hémodynamique

-**GDS**: pH 7,38 ,PaO₂ : 30 mmHg, PCO₂: 35 mmHg , HCO₃⁻: 21,2 , BE: -4
SaO₂ a 57% sous VNI

→ Intubation + sedation + curarisation sous VAC : vt : **85 ml (6ml/kg)** , FR: 30 cycles/min , **PEEP a 10 cmH₂O** (augmentée progressivement) , **Pplateau a 30 cmH₂O**

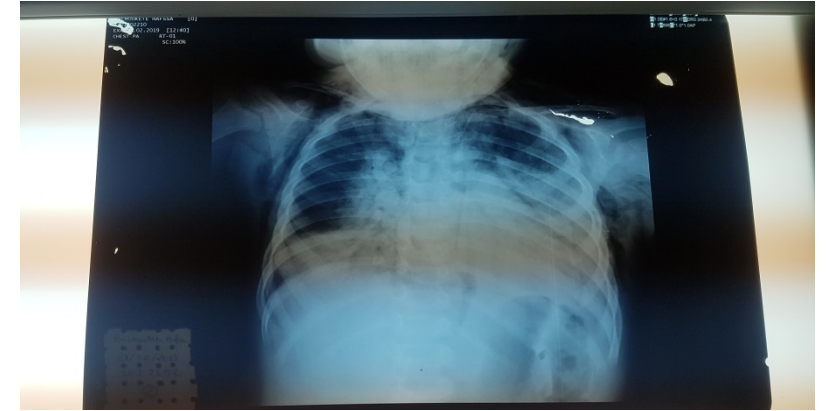
Examens para cliniques :

-GB: 15 250 , CRP : 312 , Pq: 380 000

-Rx thorax : pneumopathie extensive

-PCR respiratoire : + au virus de la Grippe A (H1N1 2009)

→ C3G / Josamycine / Tamiflu



En réanimation (H12) :

-Episodes de désaturations, nécessitant des manœuvres de recrutements manuelles de plus en plus fréquents

-GDS : **pH: 7,04 , PCO₂ : 92 , PO₂: 72** , BE: -6, HCO₃⁻: 24 , SaO₂ 84% , sous FiO₂ 100% , P plateau 30 cmH₂O

Questions

1-Quel est votre diagnostic?

2-Quelle est la sévérité de cette maladie?

Pediatric Acute Respiratory Distress Syndrome: Consensus Recommendations From the Pediatric Acute Lung Injury Consensus Conference*

The Pediatric Acute Lung Injury Consensus Conference Group

Pediatric Acute Lung Injury Consensus Conference Group

GDS : pH: 7,04 ,
PCO₂ : 92 , PO₂:
72 , BE: -6, HCO₃-
: 24 , SaO₂ 84% ,
sous FiO₂ 100%

IO à 41

Age	Exclude patients with peri-natal related lung disease			
Timing	Within 7 days of known clinical insult			
Origin of Edema	Respiratory failure not fully explained by cardiac failure or fluid overload			
Chest Imaging	Chest imaging findings of new infiltrate(s) consistent with acute pulmonary parenchymal disease			
Oxygenation	Non Invasive mechanical ventilation	Invasive mechanical ventilation		
	PARDS (No severity stratification)	Mild	Moderate	Severe
	Full face-mask bi-level ventilation or CPAP ≥5 cm H ₂ O ² PF ratio ≤ 300 SF ratio ≤ 264 ¹	4 ≤ OI < 8 5 ≤ OSI < 7.5 ¹	8 ≤ OI < 16 7.5 ≤ OSI < 12.3 ¹	OI ≥ 16 OSI ≥ 12.3 ¹
Special Populations				
Cyanotic Heart Disease	Standard Criteria above for age, timing, origin of edema and chest imaging with an acute deterioration in oxygenation not explained by underlying cardiac disease. ³			
Chronic Lung Disease	Standard Criteria above for age, timing, and origin of edema with chest imaging consistent with new infiltrate and acute deterioration in oxygenation from baseline which meet oxygenation criteria above. ³			
Left Ventricular dysfunction	Standard Criteria for age, timing and origin of edema with chest imaging changes consistent with new infiltrate and acute deterioration in oxygenation which meet criteria above not explained by left ventricular dysfunction.			

Figure 2. Pediatric acute respiratory distress syndrome definition. OI = oxygenation index, OSI = oxygen saturation index. ¹Use Pao₂-based metric when available. If Pao₂ not available, wean FiO₂ to maintain Spo₂ ≤ 97% to calculate OSI or oxygen saturation/FiO₂ ratio. ²For nonintubated patients treated with supplemental oxygen or nasal modes of noninvasive ventilation, see Figure 3 for at-risk criteria. ³Acute respiratory distress syndrome severity groups stratified by OI or OSI should not be applied to children with chronic lung disease who normally receive invasive mechanical ventilation or children with cyanotic congenital heart disease. $OI = (FiO_2 \times \text{mean airway pressure} \times 100) / Pao_2$ $OSI = (FiO_2 \times \text{mean airway pressure} \times 100) / Spo_2$

Age	Exclude patients with peri-natal related lung disease		
Timing	Within 7 days of known clinical insult		
Origin of Edema	Respiratory failure not fully explained by cardiac failure or fluid overload		
Chest Imaging	Chest imaging findings of new infiltrate(s) consistent with acute pulmonary parenchymal disease		
Oxygenation	Non Invasive mechanical ventilation		Invasive mechanical Ventilation
	Nasal mask CPAP or BiPAP	Oxygen via mask, nasal cannula or High Flow	Oxygen supplementation to maintain $SpO_2 \geq 88\%$ but $OI < 4$ or $OSI < 5^1$
	$FiO_2 \geq 40\%$ to attain SpO_2 88-97%	SpO_2 88-97% with oxygen supplementation at minimum flow ² : < 1 year: 2 L/min 1 – 5 years: 4 L/min 5 – 10 years: 6 L/min >10 years: 8 L/min	

Figure 2. At risk of pediatric acute respiratory distress syndrome (PARDS) definition. ¹If Pao_2 is not available, wean FiO_2 to maintain $SpO_2 \leq 97\%$ to calculate oxygen saturation index (OSI). ²Given lack of available data, for patients on an oxygen blender, flow for at-risk calculation = $FiO_2 \times$ flow rate (L/min) (e.g., 6 L/min flow at 0.35 $FiO_2 = 2.1$ L/min). BiPAP = bilevel positive airway pressure, CPAP = continuous positive airway pressure, OI = oxygenation index.

TABLE 7. Predicted FiO_2 in Patients Supported With Nasal Modes of Respiratory Support

Age	Minute Ventilation	Nasal Respiratory Flow	Predicted FiO_2 (%)
< 1 yr old	240 mL/kg/min (10-kg infant will breathe 2.4 L/min at rest)	2 L/min 100% O_2	40
1–5 yr old	180 mL/kg/min (25-kg child will breathe 4.5 L/min)	4 L/min 100% O_2	40
5–10 yr old	120 mL/kg/min (45-kg child will breathe 5.4 L/min)	6 L/min 100% O_2	40
> 10 yr old	Adult minute ventilation = 6 L/min	6–8 L/min 100% O_2	40

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



Robinder G Khemani, Lincoln Smith, Yolanda M Lopez-Fernandez, Jeni Kwok, Rica Morzov, Margaret J Klein, Nadir Yehya, Douglas Willson, Martin C J Kneyber, Jon Lillie, Analia Fernandez, Christopher J L Newth, Philippe Jouvret, Neal J Thomas, on behalf of the Pediatric Acute Respiratory Distress Syndrome Incidence and Epidemiology (PARDIE) Investigators* and the Pediatric Acute Lung Injury and Sepsis Investigators (PALISI) Network

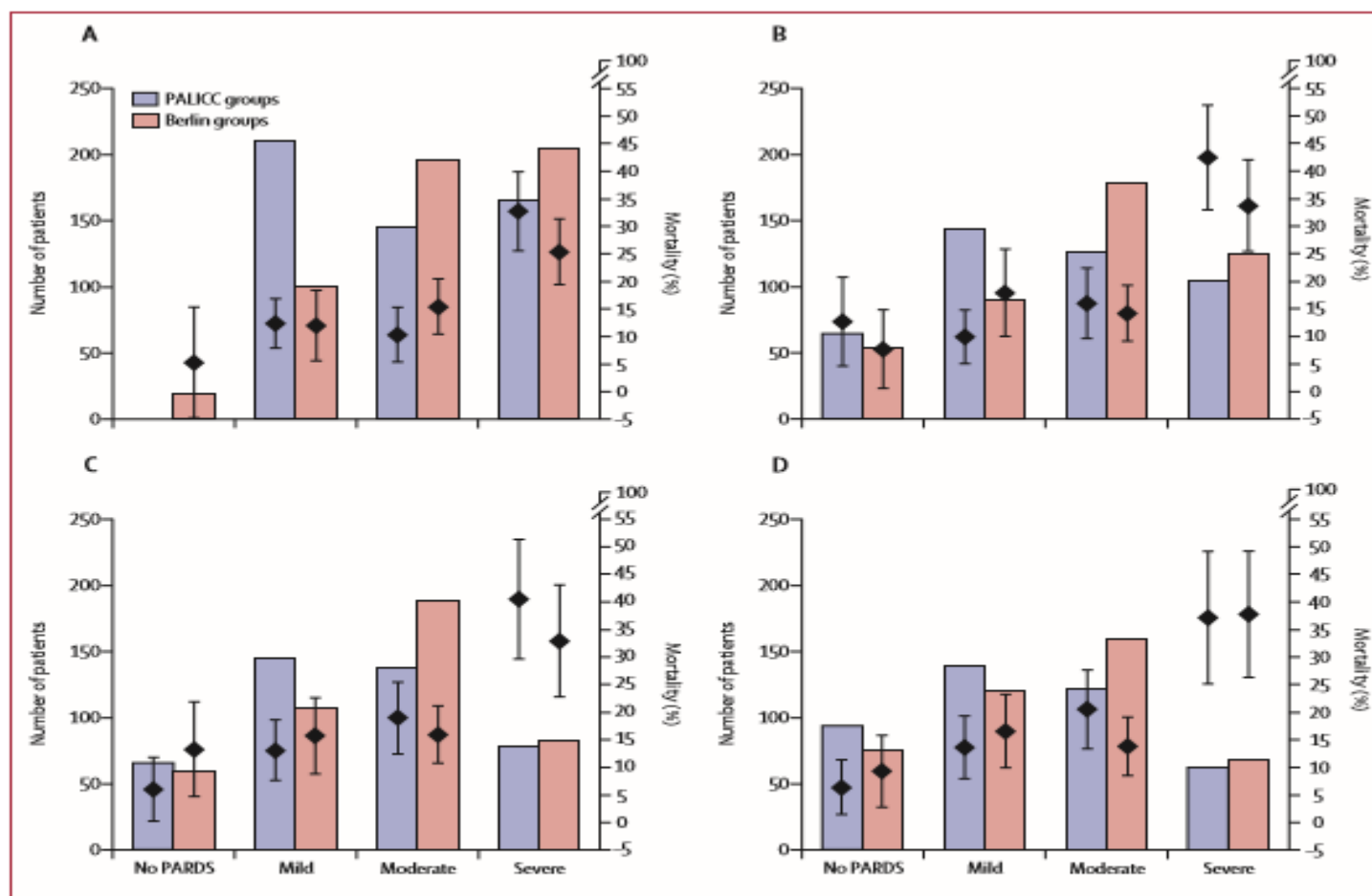


Figure 3: Severity of PARDS based on PALICC and Berlin stratification for patients initially on invasive mechanical ventilation (excluding at-home ventilation) at diagnosis (A), 6 h after diagnosis (B), 12 h after diagnosis (C), and 24 h after diagnosis (D). PARDS severity by hypoxaemia metrics at distinct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

TABLE 5. Distrubution of Patients and Mortality Based on Initial and Worst Oxygenation Index in the First 3 Days of Mechancial Ventilation

Timing	Oxygenation Index				Total
	> 16	8–16	4–8	< 4	
Baseline (first) (%)					
<i>n</i>	137 (26.8)	135 (26.4)	183 (35.8)	56 (11.0)	511
Mortality	60 (43.8)	31 (23)	22 (12.0)	2 (3.6)	115 (22.5)
Worst value 3 days (%)					
<i>n</i>	180 (35.2)	140 (27.4)	156 (30.5)	35 (6.8)	511
Mortality	73 (40.6)	29 (20.7)	12 (7.7)	1 (2.9)	115 (22.5)

Data from both Children's Hospital of Los Angeles (6) and Australia New Zealand Intensive Care Society (5) studies combined.

Interpretation The PALICC definition identified more children as having PARDS than the Berlin definition, and PALICC PARDS severity groupings improved the stratification of mortality risk, particularly when applied 6 h after PARDS diagnosis. The PALICC PARDS framework should be considered for use in future epidemiological and therapeutic research among children with PARDS.

Facteurs de risques

Abbreviated list of conditions associated with acute respiratory distress syndrome (ARDS)



Sepsis
Aspiration
Infectious pneumonia
Severe trauma
Surface burns
Multiple blood transfusions
Leukoagglutin reactions
Pancreatitis
Drug overdose
Near drowning
Smoke inhalation
Cardiopulmonary bypass
Pulmonary contusion
Multiple fractures
Following upper airway obstruction
Following bone marrow transplantation
Drug reaction
Venous air embolism
Amniotic fluid embolism
Neurogenic pulmonary edema
Acute eosinophilic pneumonia*
Bronchiolitis obliterans organizing pneumonia (BOOP)*
Miliary tuberculosis*

* Specific treatment required.

Questions

Pour améliorer l'oxygénation:

- Que pensez vous du remplissage?
- Est-ce que la VNI était justifiée?
- Comment vous gérez la ventilation invasive?

Message fort:

Une administration liquidienne
excessive est clairement délétère en
cas de choc septique et de SDRA

From: Nonventilatory Treatments for Acute Lung Injury and ARDS*

Chest. 2007;131(3):913-920. doi:10.1378/chest.06-1743

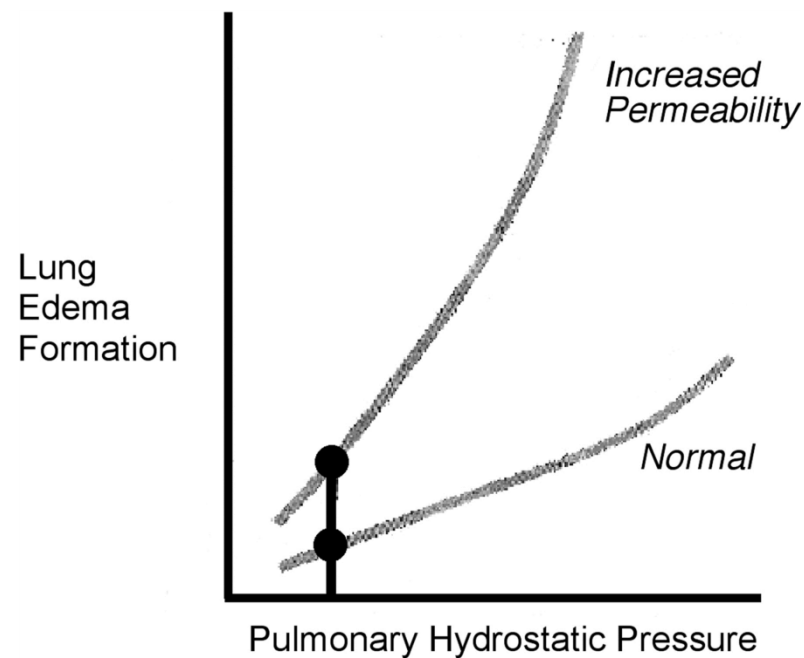


Figure Legend:

Relationship between pulmonary hydrostatic pressure and lung edema formation under normal conditions and increased permeability. Even under normal conditions, an increase in pulmonary hydrostatic pressure results in increased lung edema formation. This relationship, however, is dramatically accentuated under conditions of increased lung permeability. Adapted with permission from Staub.⁴

Sepsis in European intensive care units: Results of the SOAP study*

Crit Care Med 2006 Vol. 34, No. 2

Jean-Louis Vincent, MD, PhD, FCCM; Yasser Sakr, MB, BCh, MSc; Charles L. Sprung, MD; V. Marco Ranieri, MD; Konrad Reinhart, MD, PhD; Herwig Gerlach, MD, PhD; Rui Moreno, MD, PhD; Jean Carlet, MD, PhD; Jean-Roger Le Gall, MD; Didier Payen, MD; on behalf of the Sepsis Occurrence in Acutely Ill Patients Investigators

Table 7. Multivariate, forward stepwise logistic regression analysis in sepsis patients (n = 1177), with intensive care unit mortality as the dependent factor

	OR (95% CI)	p Value
SAPS II score ^a (per point increase)	1.0 (1.0–1.1)	<.001
Cumulative fluid balance ^b (per liter increase)	1.1 (1.0–1.1)	.001
Age (per year increase)	1.0 (1.0–1.0)	.001
Initial SOFA score (per point increase)	1.1 (1.0–1.1)	.002
Blood stream infection	1.7 (1.2–2.4)	.004
Cirrhosis	2.4 (1.3–4.5)	.008
<i>Pseudomonas</i> infection	1.6 (1.1–2.4)	.017
Medical admission	1.4 (1.0–1.8)	.049
Female gender	1.4 (1.0–1.8)	.044

OR, odds ratio; CI, confidence interval; SAPA, Simplified Acute Physiology Score; SOFA, Sequential Organ Failure Assessment.

^aAt admission; ^bwithin the first 72 hrs of onset of sepsis.

Extravascular Lung Water is an Independent Prognostic Factor in Patients with Acute Respiratory Distress Syndrome*

Mathieu Jozwiak, MD; Serena Silva, MD; Romain Persichini, MD; Nadia Anguel, MD; David Osman, MD; Christian Richard, MD; Jean-Louis Teboul, MD, PhD; Xavier Monnet, MD, PhD (Crit Care Med 2013;41:472-480)

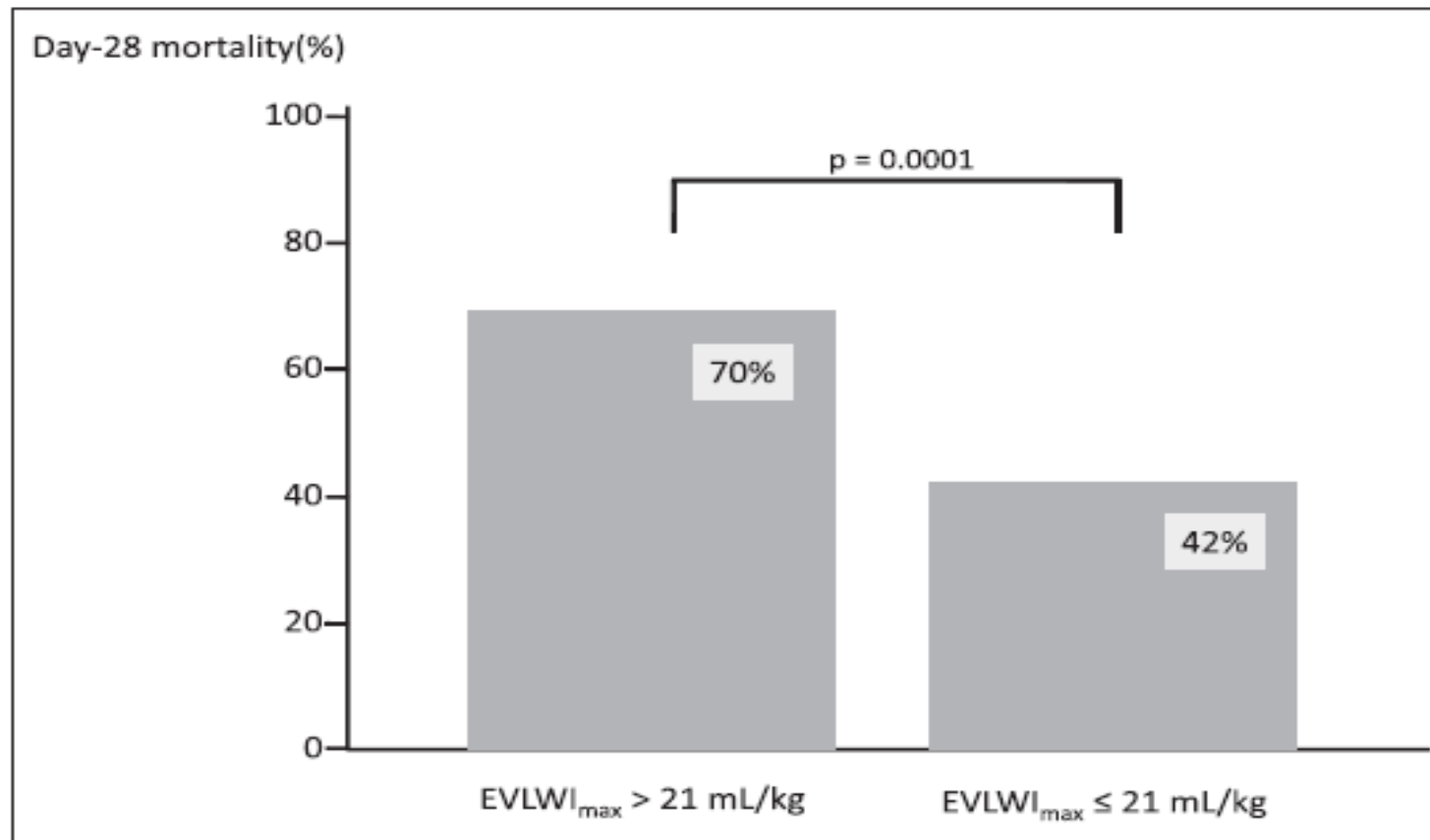


Figure 1. Day-28 mortality rate (in %) in patients with a maximum value of extravascular lung water indexed (EVLWI_{max}) to predicted body weight superior or inferior to 21 mL/kg.

Limiter l'administration liquidienne
lors du SDRA

THE EFFECTS OF FLUID OVERLOAD IN MECHANICALLY VENTILATED CHILDREN WITH PEDIATRIC ACUTE RESPIRATORY DISTRESS SYNDROME

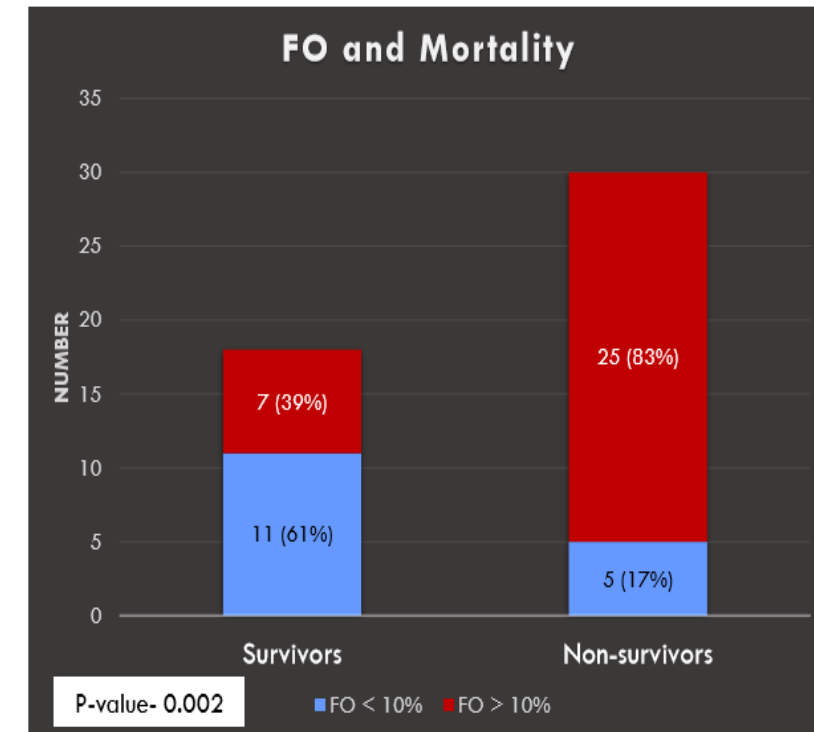
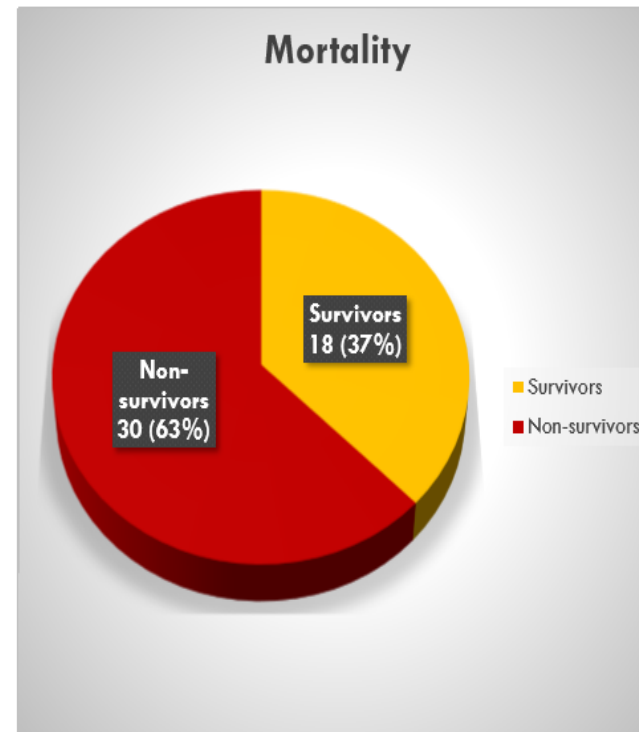
Muralidharan Jayashree, Rajalakshmi Iyer, Arun Bansal, Sahul Bharti

Advanced Pediatrics Centre, PGIMER, Chandigarh

ESPNIC 2019

Calculation of fluid overload:
(Total Intake – Total output)/ weight

FO% > 10 was considered fluid overload



A prospective, randomized, controlled trial of noninvasive ventilation in pediatric acute respiratory failure.

[Yañez LJ¹](#), [Yunge M](#), [Emilfork M](#), [Lapadula M](#), [Alcántara A](#), [Fernández C](#), [Lozano J](#), [Contreras M](#), [Conto L](#), [Arevalo C](#), [Gayan A](#), [Hernández F](#), [Pedraza M](#), [Feddersen M](#), [Bejares M](#), [Morales M](#), [Mallea F](#), [Glasinovic M](#), [Cavada G](#).

Heart rate and respiratory rate improved significantly with NIV after 1 hr of treatment compared with admission ($p = 0.0009$ and $p = 0.004$, respectively).

The trend continued over time after 6 hrs.

With NIV, **Po₂/Fio₂ improved** significantly from the first hour.

The endotracheal intubation was significantly lower (28%) in the NIV group than in the control group (60%; $p = 0.045$).

It could be debated that noninvasive ventilation **should only be considered in patients with less severe disease and not used in patients with moderate to severe lung disease.**

Patients who will respond to therapy will likely show **improvement in respiratory distress and oxygenation within the first 30–60 minutes**

Lung-Protective Strategies: prevent (VILI)

1-Tidal Volume Delivery: Volutrauma

2-PEEP Titration: Atelectrauma

3-Plateau Pressure and Drive Pressure (ΔP): Barotrauma

4- High-Frequency Oscillatory Ventilation

5-Autres

Ventilation protectrice en pédiatrie

Have changes in ventilation practice improved outcome in children with acute lung injury?*

Waleed H. Albuali, MD; Ram N. Singh, MD, FRCPC; Douglas D. Fraser, MD, PhD, FRCPC; Jamie A. Seabrook, MA; Brian P. Kavanagh, MD, FRCPC; Christopher S. Parshuram, MD, FRACP; Alik Komecki, MD

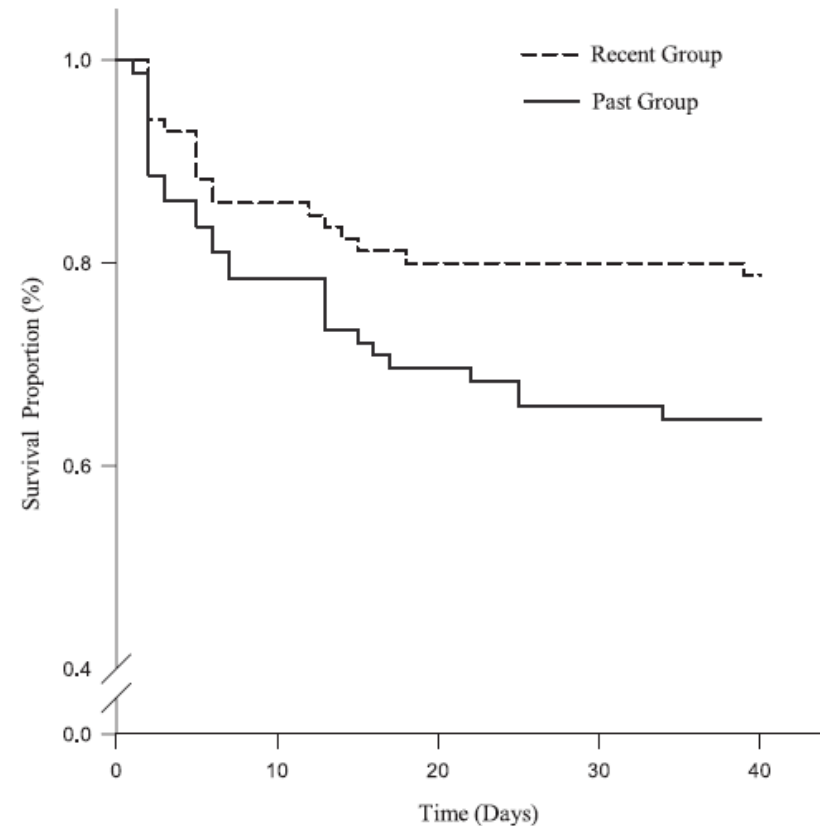
Pediatr Crit Care Med 2007 Vol. 8, No. 4

Table 5. Multivariable analysis for clinical variables^a associated with mortality and ventilation-free days

Variable	Odds Ratio	Mortality		Ventilation-Free Days	
		95% CI	p Value	95% CI	p Value
PRISM III	1.35	1.21, 1.50	<.001	-1.20, -0.66	<.001
Group (recent vs. past group)	0.86	-0.82, 0.99	.23	-6.21, 0.06	.06
Tidal volume ^b	1.59	1.20, 2.10	<.001	-1.99, -0.43	.003
PEEP ^b	1.08	0.83, 1.41	.36	-0.84, 0.46	.36
PIP ^b	0.95	0.88, 1.02	.15	-0.06, 0.50	.13
PaO ₂ /FiO ₂ <200	3.58	0.45, 28.34	.23	-5.38, 1.44	.25
Immunodeficiency	3.58	1.09, 11.76	.04	-7.41, -0.32	.03
Male gender	0.44	0.17, 1.15	.09	0.44, 5.29	.02
Sepsis	1.75	0.49, 6.23	.39	-4.99, 1.25	.24
Pneumonia	1.64	0.50, 5.38	.42	-4.42, 1.60	.36
Prone position	3.14	0.67, 14.67	.15	-9.58, -0.17	.04
HFOV	0.65	0.13, 3.36	.61	-3.30, 5.49	.62

PRISM, Pediatric Risk of Mortality; PEEP, positive end-expiratory pressure; PIP, peak inspiratory pressure; HFOV, high-frequency oscillatory ventilation.

^aOnly for associated variables ($p \leq .1$) from univariate analysis; ^bmean for the first 3 days of ventilation.



Ventilatory Support in Children With Pediatric Acute Respiratory Distress Syndrome: Proceedings From the Pediatric Acute Lung Injury Consensus Conference

Peter C. Rimensberger, MD¹; Ira M. Cheifetz, MD, FCCM²; for the Pediatric Acute Lung Injury Consensus Conference Group

1-Tidal Volume Delivery: Volutrauma

3.2.2 We recommend to use patient-specific tidal volumes according to disease severity. Tidal volumes should be **3–6 mL/ kg PBW for patients with poor respiratory system compliance** and closer to the physiologic range (5–8 mL/kg ideal body weight) for patients with better preserved respiratory system compliance.

2-PEEP Titration: Atelectrauma

3.3.1 We recommend moderately elevated levels of PEEP (**10–15 cm H₂O**) titrated to the observed oxygenation and hemodynamic response in patients with severe PARDS.

3.3.2 We recommend that PEEP levels **greater than 15 cm H₂O** may be needed **for severe PARDS** although attention should be paid to limiting the plateau pressure as previously described.

3.3.3 We recommend that markers of oxygen delivery, respiratory system compliance, and hemodynamics should be **closely monitored as PEEP is increased.**

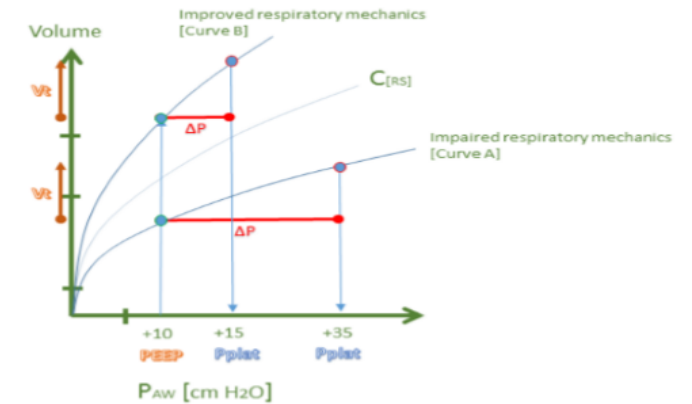
3-Plateau Pressure and Drive Pressure (ΔP): Barotrauma

3.2.3 In the absence of transpulmonary pressure measurements, we recommend an **inspiratory plateau pressure limit of 28 cm H₂O**, allowing for slightly higher plateau pressures (**29–32 cm H₂O**) for patients with increased chest wall elastance (i.e., reduced chest wall compliance).

$$\Delta P \text{ (drive pressure)} = PIP - PEEP ???$$

Airway Driving Pressure and Outcome in Children with Acute Hypoxemic Respiratory Failure

Dr ANIL SACHDEV MD, FICCM
DIRECTOR
PEDIATRIC EMERGENCY, CRITICAL CARE and PULMONOLOG
SIR GANGA RAM HOSPITAL
NEW DELHI
INDIA



Analysis of outcome parameters with ARDS

Outcome parameter	Low ΔP group (n=29)	High ΔP group (n=36)	p value
Duration of ventilation [Median (IQR)]	5 days (4-7)	9 days (6-12)	<0.001
Ventilator free days at day 28 [Median (IQR)]	22 days (19-23)	16 days (0-21)	<0.001
Length of ICU stay (days) [Median (IQR)]	7 days (6-9)	14 days (7-15)	<0.001
Length of hospital stay (days) [Median (IQR)]	12 days (7-15)	19 days (13-25)	<0.001
In hospital mortality (%)	18 %	25 %	0.33

Driving Pressure	< 15	10 (24)	37 (62)	< 0.001	3.3	1.2 - 8.8
(cm H ₂ O)	≥ 15	31 (76)	23 (38)			

To be continued...

1973



Barotrauma

1988



Volutrauma

1997



Atelectrauma
(Biotrauma)

Rheology &
Material Engineering

2016



Ergotrauma

4- High-Frequency Oscillatory Ventilation

3.4.1 We recommend that HFOV **should be considered** as an alternative ventilatory mode in **hypoxic** respiratory failure in patients in whom **plateau airway pressures exceed 28 cm H₂O** in the absence of clinical evidence of reduced chest wall compliance. Such an approach should be considered for those patients with moderate-to-severe PARDS.

High-frequency ventilation versus conventional ventilation for treatment of acute lung injury and acute respiratory distress syndrome (Review)

Wunsch H, Mapstone J

There is no evidence that high-frequency oscillatory ventilation increases mortality for patients with ALI or ARDS when compared with conventional ventilation.

There is not enough evidence to conclude whether high-frequency oscillatory ventilation reduces mortality and improves quality of life for survivors of ALI or ARDS.

5-Autres

Endotracheal Tubes : Cuffed ETTs are recommended

Oxygenation

- Mild PARDS with PEEP less than 10 cm H₂O, **Spo2** at **92–97%**.
- PARDS with PEEP greater than or equal to 10 cm H₂O. Spo2 levels:**88–92%**
- Spo2 is less than 92%, monitoring of **central venous saturation**

Ventilation: permissive hypercapnia

-pH 7.15–7.30

-Exceptions: intracranial hypertension, severe pulmonary,....

-Bicarbonate supplementation is not routinely recommended

En réanimation (H24) :

-Réponse médiocre au DV sur une durée de 12h

-Sédation , curares , NO (non disponible)

- Vt : 100 ml , FR: 36 , I/E : 1/1 , FiO2 100% , Pplat a 32 cmH2O
- pH: 7,17 , PO2: 47 , PCO2: 49, HCO3- : 18 , SaO2: 71 % , Lactate : 0,74
- Hémodynamique stabilisé par: NAD 2 µg/kg/min

Adjunctive Therapies

Recruitment Maneuvers

Using slow incremental and decremental PEEP

Not using sustained insufflation maneuvers

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

Afshari A, Brok J, Møller AM, Wetterslev J



14 ESSAIS RANDOMISES

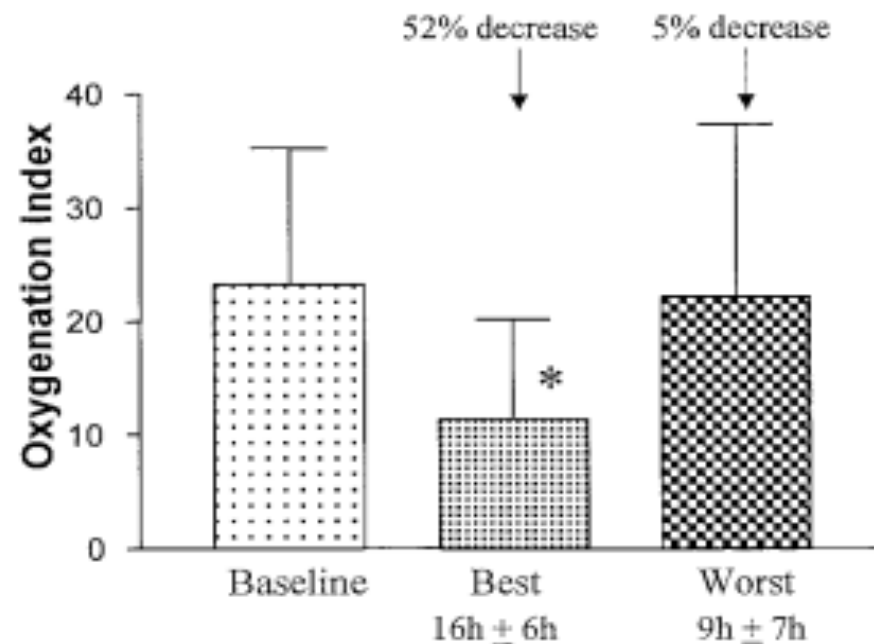
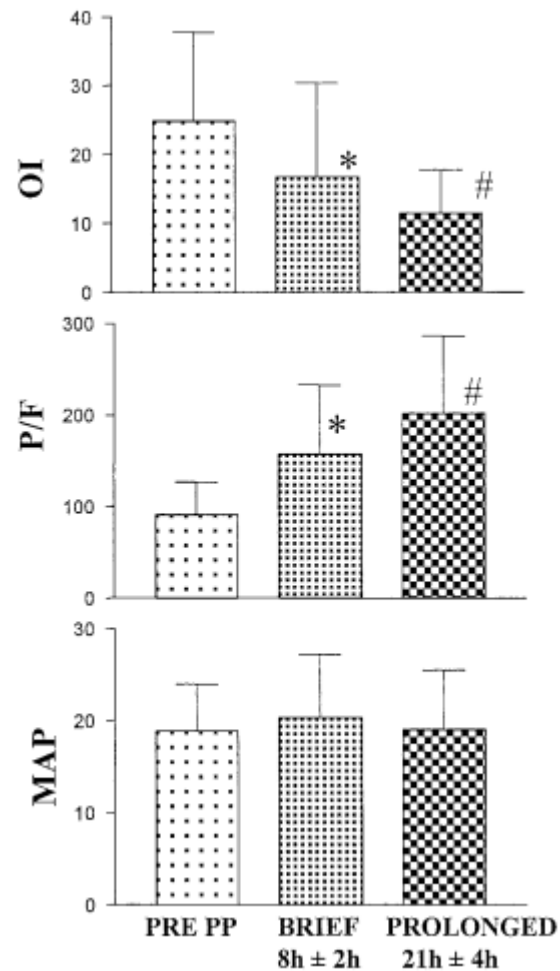
1340 PATIENTS

AUTHORS' CONCLUSIONS:

INO *cannot be recommended* for patients with AHRE. INO results in a transient improvement in oxygenation but does not reduce mortality and may be harmful

Prone Positioning of Pediatric Patients With ARDS Results in Improvement in Oxygenation if Maintained > 12 h Daily*

*Monica S. Relvas, MD; Peter C. Silver, MD, FCCP; and
Mayer Sagy, MD, FCCP*



Effect of Prone Positioning on Clinical Outcomes in Children with Acute Lung Injury: A Randomized Controlled Trial

Martha A.Q. Curley, R.N., Ph.D., Patricia L. Hibberd, M.D., Ph.D., Lori D. Fineman, R.N., M.S., David Wypij, Ph.D., Mei-Chiung Shih, Ph.D., John E. Thompson, R.R.T., Mary Jo C. Grant, R.N., Ph.D., Frederick E. Barr, M.D., M.S., Natalie Z. Cvijanovich, M.D., Lauren Sorce, R.N., M.S., Peter M. Lockett, M.D., Michael A. Matthay, M.D., and John H. Arnold, M.D.

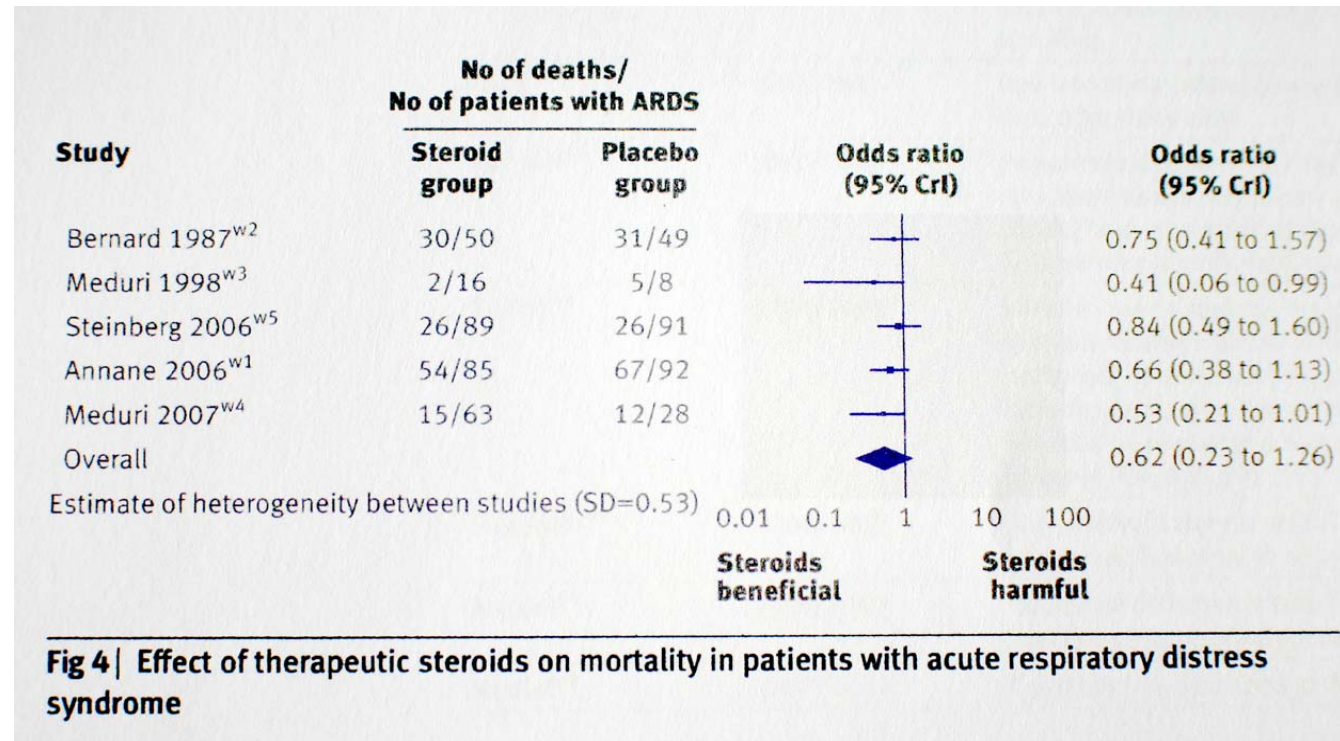
Conclusions: Prone positioning does not significantly improve ventilator-free days or other clinical outcomes in pediatric patients with acute lung injury.

Surfactant

Aucun bénéfice clair n'a été démontré, quel que soit le type de surfactant ou le mode d'administration
(aérosols ou injection intra-trachéale)

SDRA et Corticothérapie ?

Aucun effet à la phase précoce (*Peter, BMJ, 2008;336:1006*)



Pas d'effet à phase tardive (*ARDS Net, NEJM, 2006;354:1671*)

Aucun effet sur ARDS lié à H5N1 chez l'animal (*Xu T, Eur Respir J, 2009*)

Quelle est votre conduite a tenir a ce stade ?

En réanimation (H24) :

-Réponse médiocre au DV sur une durée de 12h

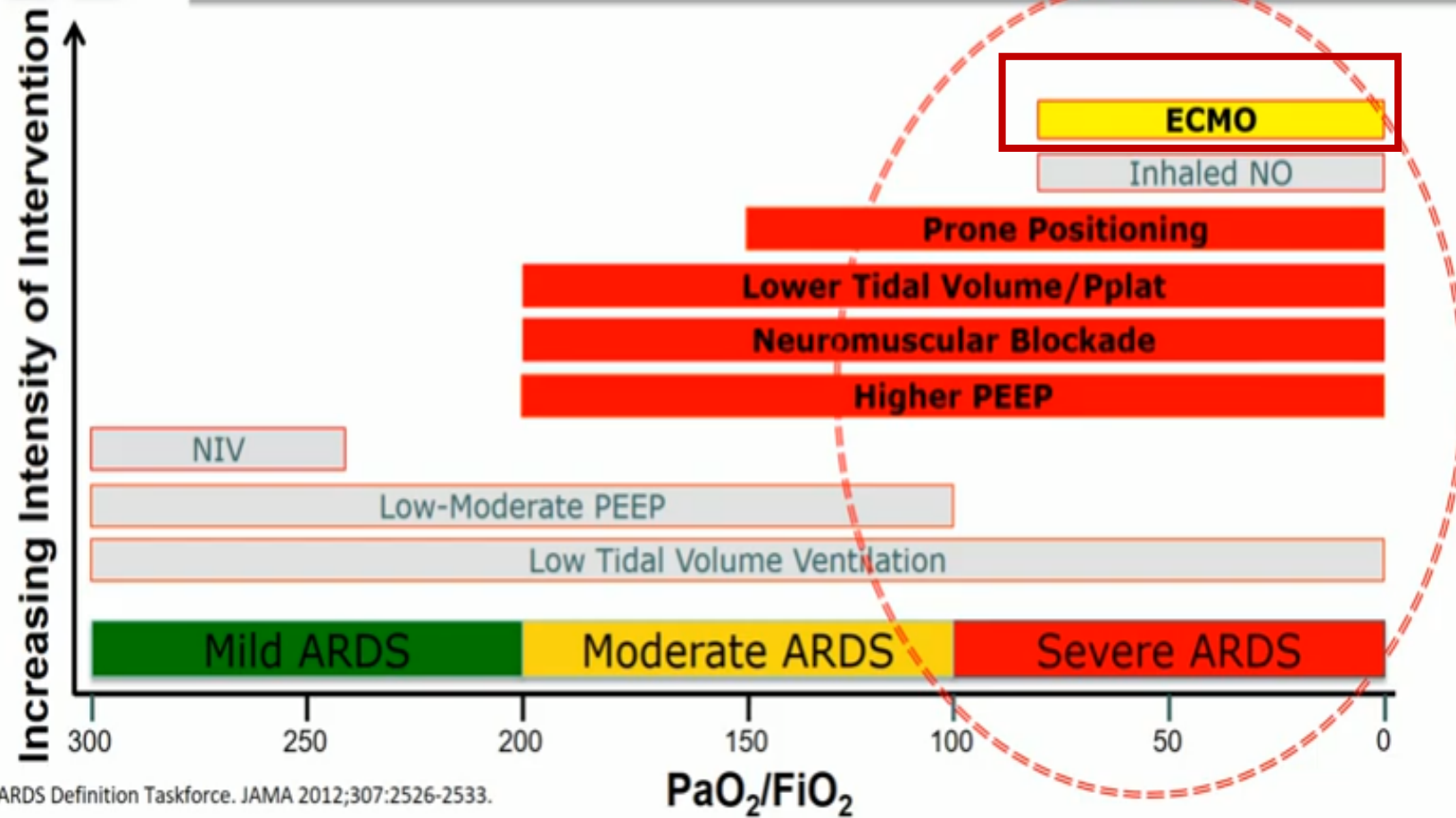
-Sédation , curares , NO (non disponible)

- Vt : 100 ml , FR: 36 , I/E : 1/1 , FiO2 100% , Pplat a 32 cmH2O
- pH: 7,17 , PO2: 47 , PCO2: 49, HCO3- : 18 , SaO2: 71 % , Lactate : 0,74
- Hémodynamique stabilisé par: NAD 2 µg/kg/min



Acute Respiratory Distress Syndrome

The Berlin Definition



The ARDS Definition Taskforce. JAMA 2012;307:2526-2533.

Beaucoup de terminologie....

Terme	définition
ECLS Extracorporeal Life Support	Tout dispositif extracorporel ayant pour vocation de maintenir, l'oxygénation, l'épuration du CO ₂ et une hémodynamique satisfaisante.
ECMO Extracorporeal Membrane Oxygenation	ASSISTANCE RESPIRATOIRE
V-V ECLS Veino-Veineuse Extracorporeal Life Support	Support respiratoire stricte. Hémodynamique assurée par le patient ASSISTANCE RESPIRATOIRE
V-A ECLS Veino-Arterial Extracorporeal Life Support	Support hémodynamique et respiratoire. ASSISTANCE CIRCULATOIRE
ECCOR Extracorporeal CO ₂ Removal	Épuration du CO ₂ uniquement. Faible oxygénation. Pas de support hémodynamique
AREC Assistance Respiratoire Extra-corporelle	ASSISTANCE RESPIRATOIRE

Any 1 of the following criteria qualifies a patient for ECMO:

Respiratory Criteria:

Oxygenation Index (OI) = $\text{MAP} \times \text{FiO}_2 \times 100 / \text{PaO}_2$:

All Infants

>60 for 30 min.
>40 for 60 min
>35 for 6 hours
>30 for 24 hours
>25 for 72 hours

Infants with Diaphragmatic Hernia:

>35 for 30 min.
>30 for 2 hours
>25 for 4 hours
OR need for $\text{MAP} > 15$, HFO AMP or Jet PIP > 40, or
conventional PIP > 30

Barotrauma:

Ventilator settings exceeding: PIP > 35, MAP > 20, HFOV AMplitude > 40, or Jet PIP > 45.
Hypercarbia with pH < 7.10 on: PIP = 35, Jet PIP = 45, or HFO AMplitude = 40 for 4 hours.
Severe air leak unresponsive to other therapies.

Acute Deterioration without rapid solution:

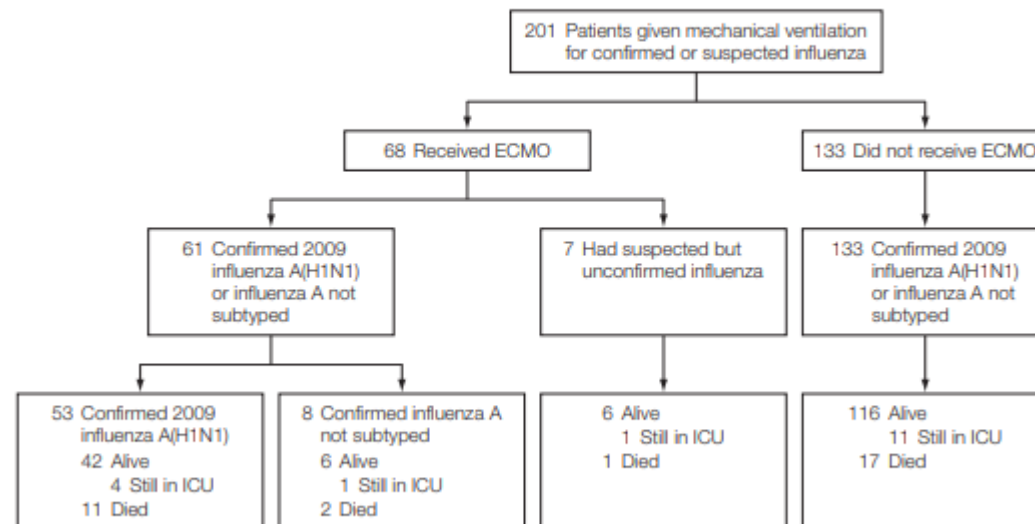
$\text{PaO}_2 < 30$ or preductal $\text{SaO}_2 < 70\%$

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

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

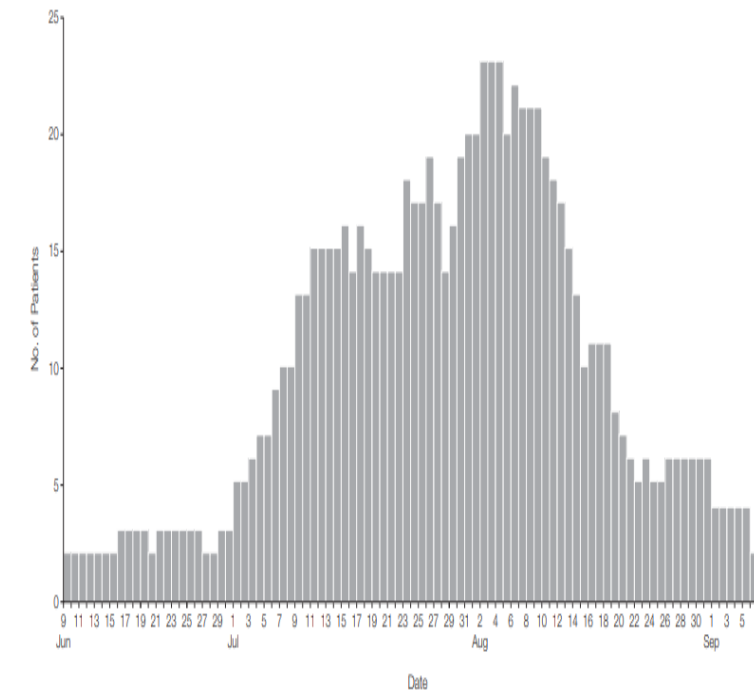
Context The novel influenza A(H1N1) pandemic affected Australia and New Zealand during the 2009 southern hemisphere winter. It caused an epidemic of critical illness and some patients developed severe acute respiratory distress syndrome (ARDS) and were treated with extracorporeal membrane oxygenation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

Figure 1. Flow Diagram of Patients Receiving Mechanical Ventilation for Suspected 2009 Influenza A(H1N1) Infection at ECMO Centers



ECMO indicates extracorporeal membrane oxygenation; ICU, intensive care unit.

Figure 2. Histogram of Number of Concurrent Patients Receiving ECMO Across Australia and New Zealand in 2009

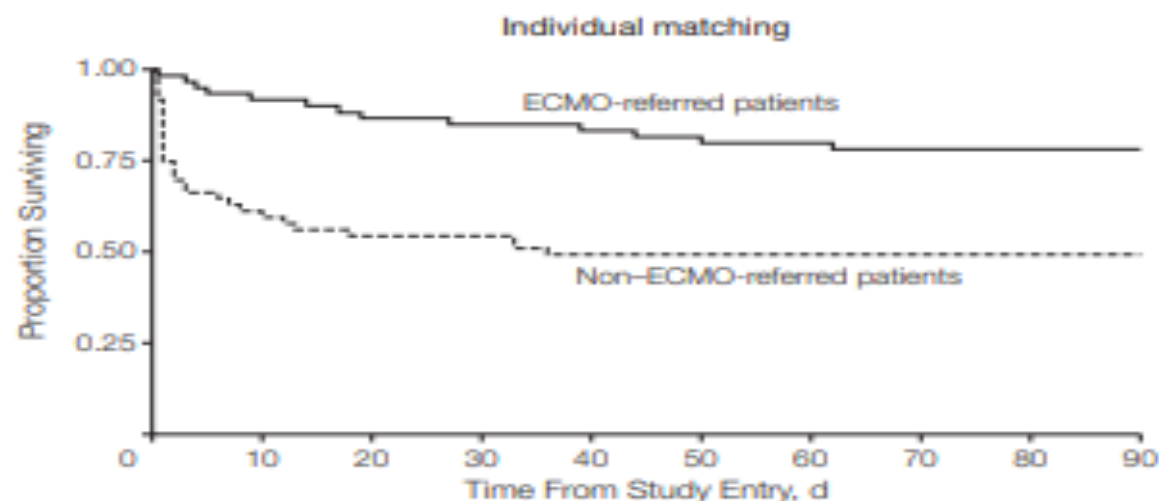


ECMO indicates extracorporeal membrane oxygenation.

ONLINE FIRST

Referral to an Extracorporeal Membrane Oxygenation Center and Mortality Among Patients With Severe 2009 Influenza A(H1N1)

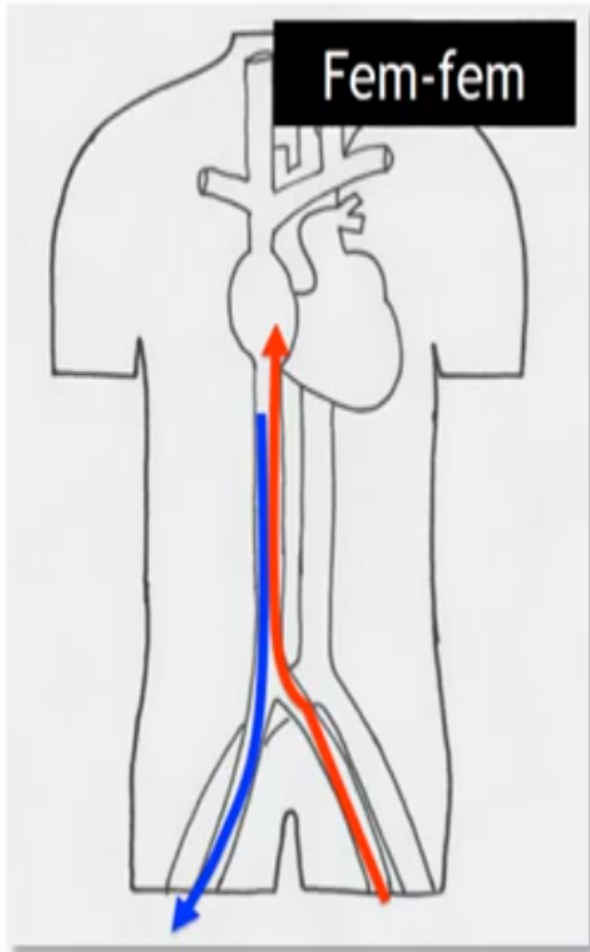
Figure 2. Survival Curves for ECMO-Referred Patients vs Matched Non-ECMO-Referred Patients



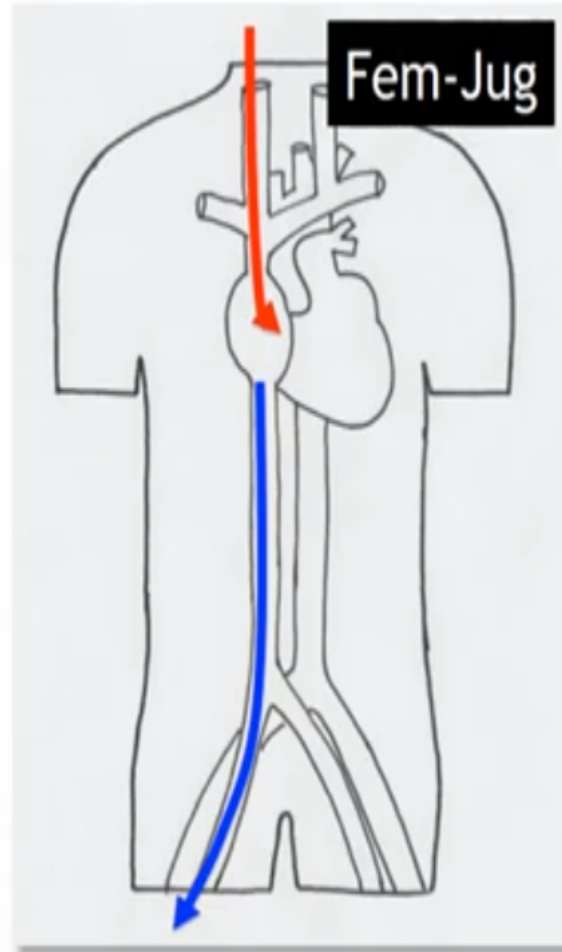
ECMO VV ou VA ?

ECMO VV

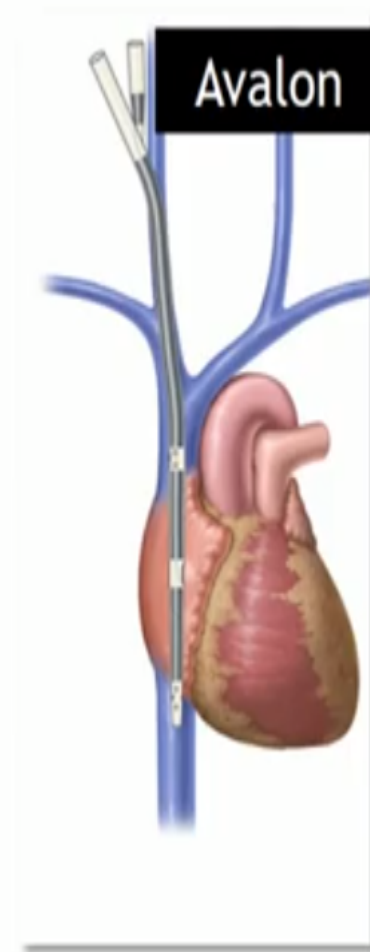
- Oxygénation
- E.puration du Co2
- Eviter le syndrome d'arlequin: Tête bleu-Jambes rouges
- Diminution des pressions intra thoracique en réduisant les paramètres ventilatoires → soulager le CPA
- Mettre au repos les poumons malades



Bas débit
Risque de recirculation



Contrôle ETO
déplacement



GDS en **DV** avant la canulation :

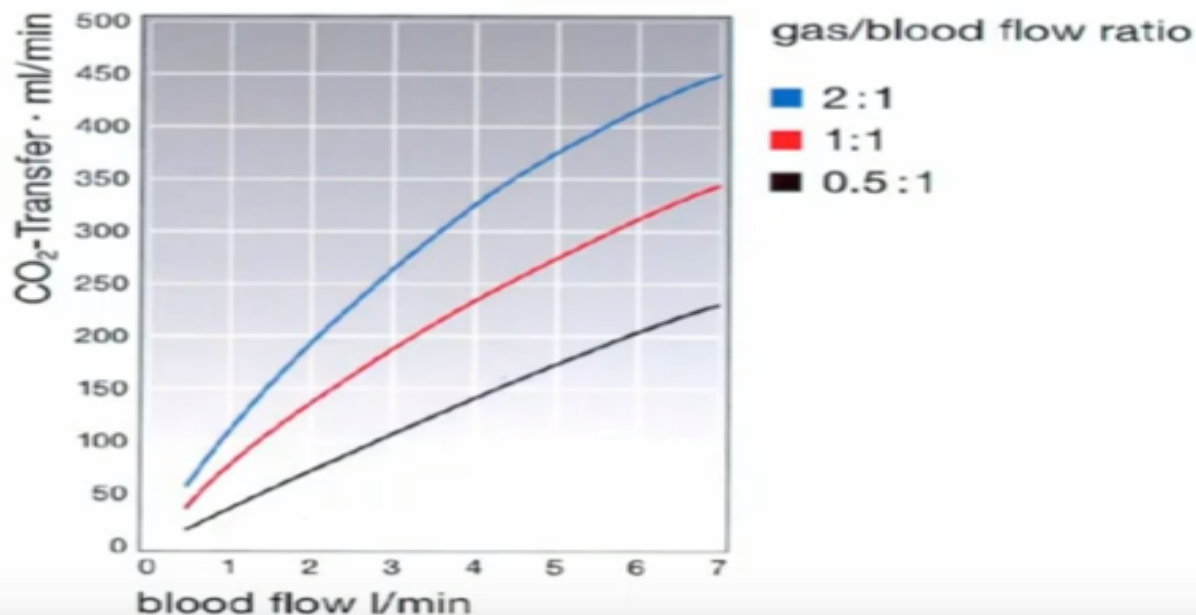
pH: 7,04 , PCO2: 92 , PO2: 72, HCO3- : 24,9, SaO2: 84% sous FiO_2 a 100%

Canulation veino-veineuse jugulo – fémorale Canule jugulaire 17
canule fémorale 15



**Comment assurer la correction de la
PaCo₂?**

Balayage de l'ECMO : par principe de diffusion



ORIGINAL

Brain injury during venovenous extracorporeal membrane oxygenation

Charles-Edouard Luyt^{1,2*}, Nicolas Bréchet^{1,2}, Pierre Demondion³, Tamara Jovanovic¹, Guillaume Hékimian^{1,2}, Guillaume Lebreton³, Ania Nieszkowska^{1,2}, Matthieu Schmidt^{1,2}, Jean-Louis Trouillet^{1,2}, Pascal Leprince³, Jean Chastre^{1,2} and Alain Combes^{1,2}



La dégression doit se faire **progressivement** afin de réduire le risque de séquelles neurologique
La dégression rapide de PaCO₂ conduit à une **vasoconstriction** importante

Comment assurer l'oxygénation (PaO₂)

La ventilation mécanique (ultra protectrice)

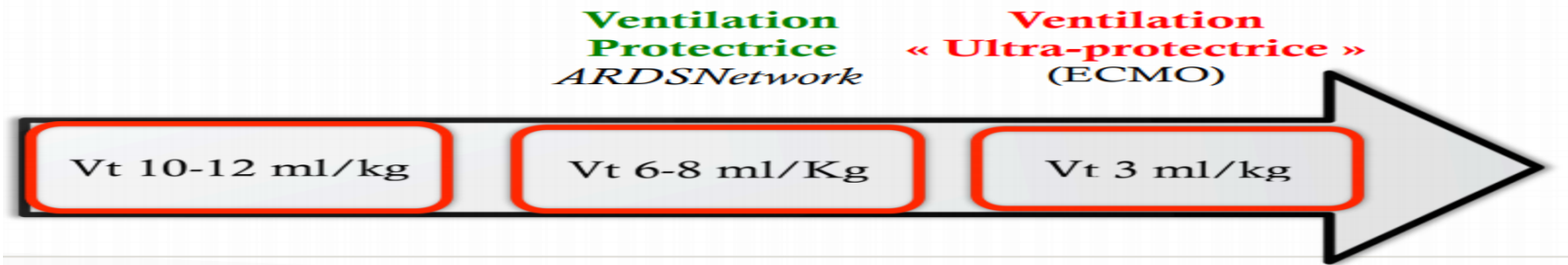
Débit d'ECMO : Taille des canules

Attention au syndrome de recirculation ($SvO_2 = SaO_2$)

Taux d'hémoglobine

Ventilation mécanique sous ECMO

- Evolution de la ventilation mécanique :



Les experts du réseau de recherche en ventilation artificielle (REVA). 2009

- Mode assisté contrôlé en volume
- PEEP ≥ 10 cmH₂O
- Volume courant réduit pour obtenir une pression de plateau ≤ 20 à 25 cmH₂O
- Fréquence respiratoire de 6 à 20 cycles/min
- FiO₂ entre 30 et 50 %

Extracorporeal Membrane Oxygenation for Severe Acute Respiratory Distress Syndrome (EOLIA)

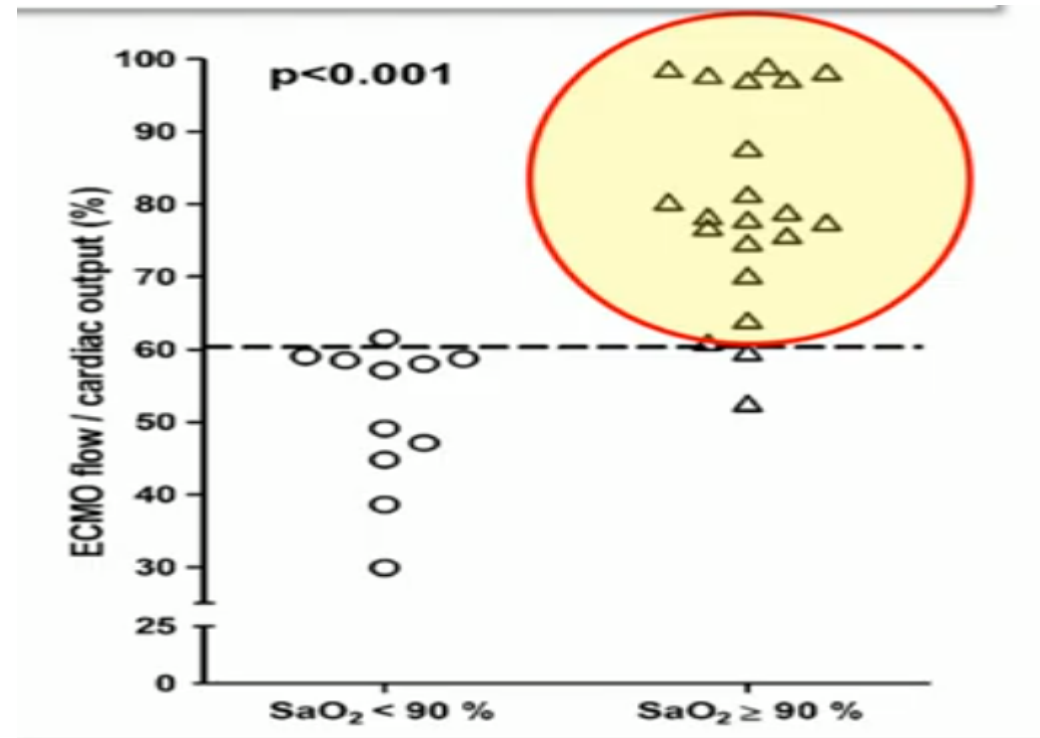
- Etude randomisée, multicentrique, prospective
- Paramètres de VM sous ECMO standardisés
 - VAC,
 - FiO₂ 30-60%,
 - PEEP ≥ 10 cmH₂O,
 - Vt pour P_{plat} < 25 cmH₂O,
 - FR 10 à 30/min
 - (ou APRV)

Matthieu Schmidt
Guillaume Tachon
Christine Devilliers
Grégoire Muller
Guillaume Hekimian
Nicolas Bréchet
Sybille Merceron
Charles Edouard Luyt
Jean-Louis Trouillet
Jean Chastre
Pascal Leprince
Alain Combes

Blood oxygenation and decarboxylation determinants during venovenous ECMO for respiratory failure in adults

Intensive Care Med (2013)

60 % du débit cardiaque est suffisante pour permettre une bonne oxygénation sous ECMO



Syndrome de recirculation

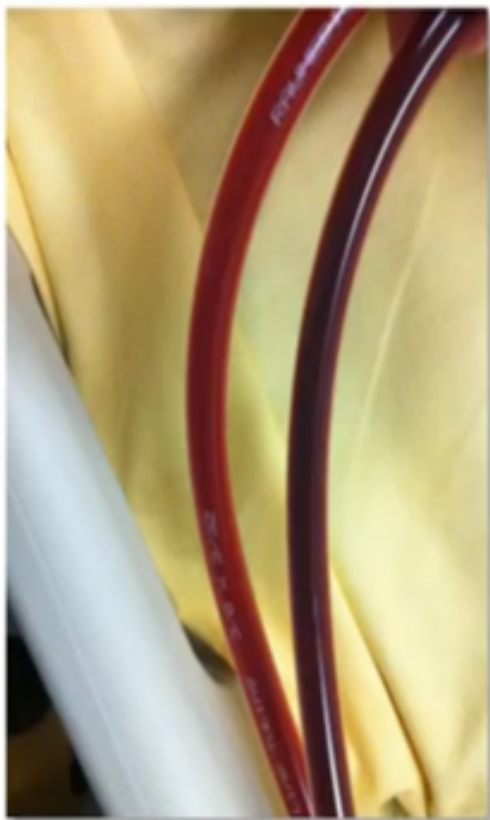
Prélèvement d'un sang Oxygéné par l'ECMO

$SvO_2 \approx SaO_2 \rightarrow$ hypoxémie

Dg :

- couleur du sang dans les 2 canules
- Rx thorax / ETO
 - Les 2 canules qui s'embrassent

Traitement : retrait d'une des 2 canules



Normal



Recirculation+++



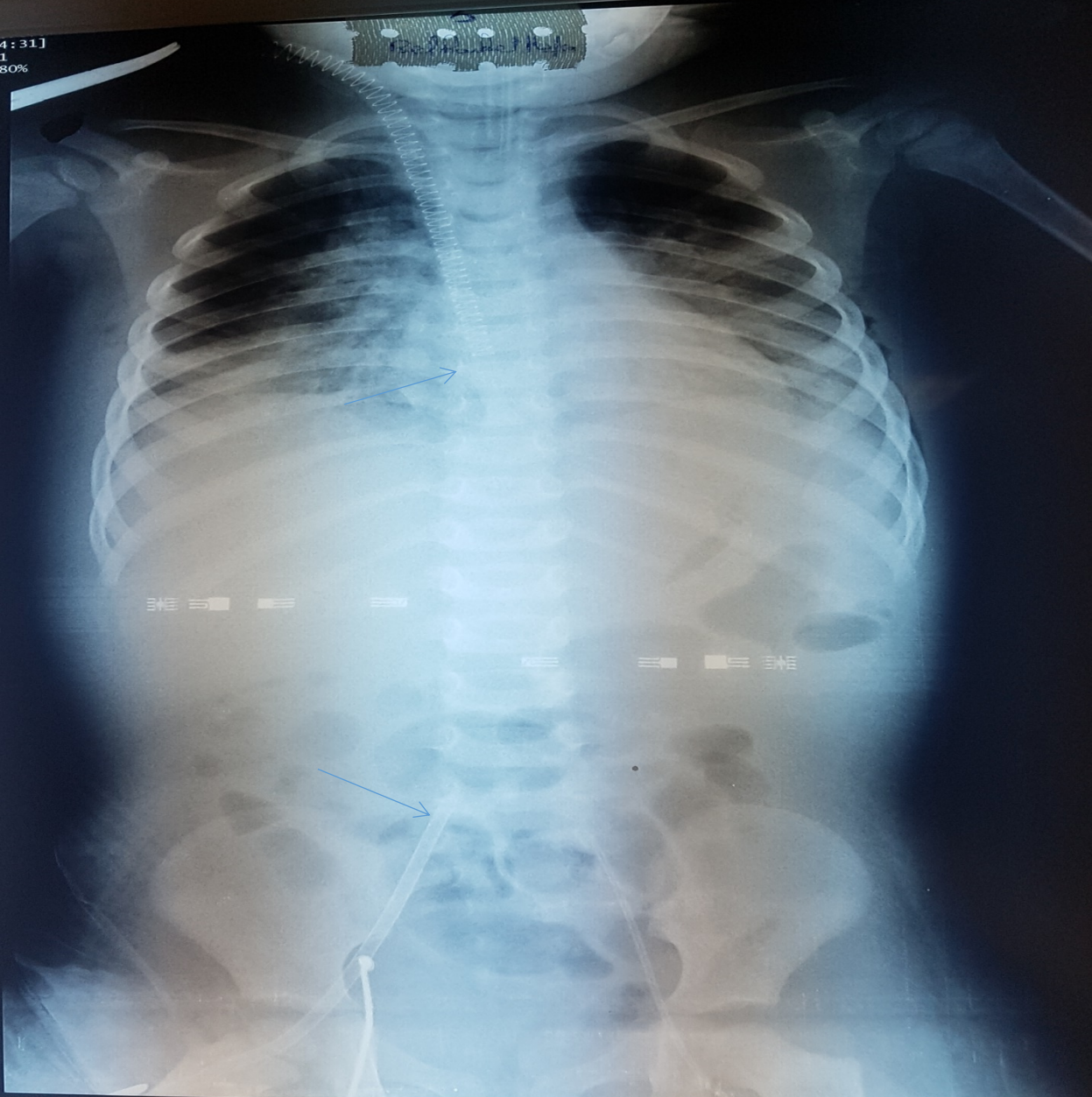
Q_{ECMO} 7.2L/min
 SaO_2 :88%
 SvO_2 :82%



Q_{ECMO} 7,2L/min
 SaO_2 :100%
 SvO_2 :66%

019 [14:31]
RT-01
SC:80%

1.65540 1.0*1.0AP



-A J3 d'hospitalisation sous ECMO:

- Débit 0,8l/min , FiO2 ECMO : 60%, Balayage 0,8l/min

-Enfant IVC , ventilation ultra protectrice

- Vt: 50 ml FR: 10 , PEEP: 10 , I/E: $\frac{1}{2}$, Pplat: 25 mbar, FiO2 dégressé a 40 %

-GDS:

- pH: 7,34 , PaO2: 81 , PCo2: 46,9 , HCO3: 25,4, SaO2: 96%

-Sevré de la NAD et dobutamine

-Sous HNF 50UI/kg/h , avec saignement modéré par les sites de canulation

-Monitoring du NIRS cérébral et somatique correcte

-J5 d'ECMO:

-Enfant intubé ventilé , sedation allégée

-GDS: ph:7,38 , PO₂: 143 , PCO₂: 34 , SaO₂ 100% , HCO₃⁻: 29

- → dégression progressive du balayage d'un rapport 1/1 à 1/0,5 puis arrêt sous contrôle gazométrique
- → réglage des paramètre ventilatoire V_t a 90 ml , PEEP a 8 , FR: 20 cycle/min , FIO₂: à 40 % avec une P_{plat}: 18

-Décision de sevrage de l'ECMO avec décanulation sous ventilation mécanique

-Décanulation à J8 avec succès

-Extubation a J11 avec succès et mise en CPAP sous casque d'Helmet + dimar

-Patiente déclarée sortante a J20 de son hospitalisation



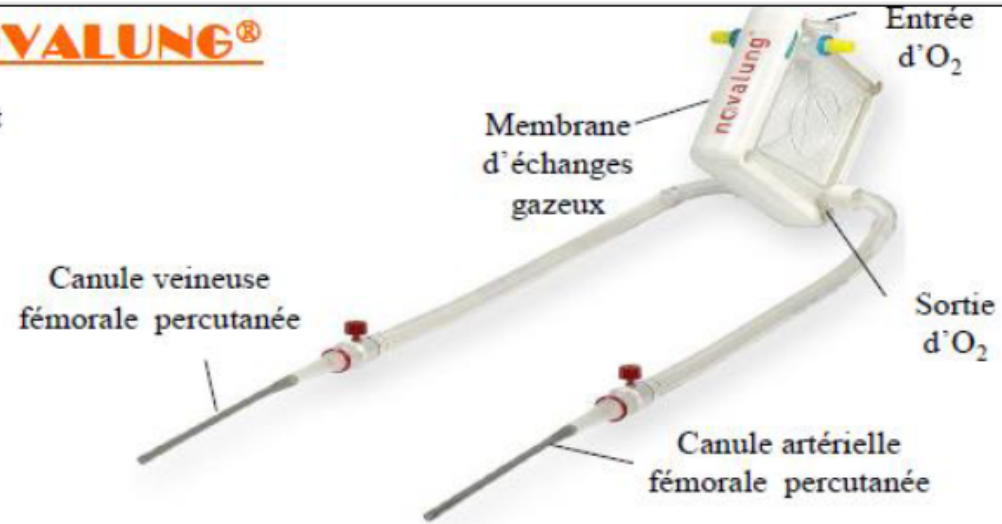
Avenir de la CEC en pédiatrie : miniaturisation ?

Système ILA Membran NOVALUNG®



Set de cathéterisme Novaport
Vascular Access®

Moniteur de débit sanguin
NovaFlow®



Comparaison NOVALUNG® / ECMO

NOVALUNG

- Flux sanguin
 - faible 0,7 à 1,5L/min
 - généré par le cœur du patient
- Équilibre acido-basique
 - Épuration du CO₂
 - Normalisation du pH
- Anticoagulation légère

ECMO

- Flux sanguin
 - élevé 3 à 4L/min
 - généré par une pompe mécanique
- Équilibre acido-basique
 - Oxygénation
- Anticoagulation forte

Conclusion

- PALICC: définition pour les pays à haut et faible revenus
- La stratégie: **Lung protective mechanical ventilator**, à réduit la mortalité
- ECMO V-V: Poser l'indication à temps
- Beaucoup d'opportunités de recherches dans le PARDS: Ergotrauma; biomarqueurs;.....