



PRISE EN CHARGE HEMODYNAMIQUE DU SDRA

LA FONCTION VENTRICULAIRE DROITE

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LE RATIONNEL

LE SDRA EST UNE MALADIE
DES ALEOLES MAIS AUSSI DE
LA CIRCULATION PULMONAIRE

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for the ALIVE Study Group

Epidemiology and outcome of acute lung injury in European intensive care units

Results from the ALIVE study

Intensive Care Med 2003

- 401 SDRA
 - 53% avec une insuffisance circulatoire associée
 - 26% dans un contexte de sepsis sévère ou de choc septique

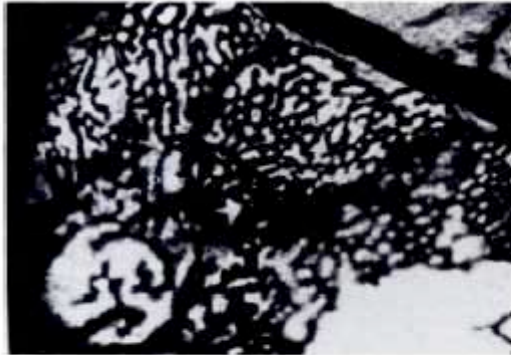


FIGURE 3. Normal human capillary network at 470 \times (original magnification). Note the high frequency of capillary anastomoses after vascular filling at 50 mm Hg silicone infusion pressure in this normal human lung.

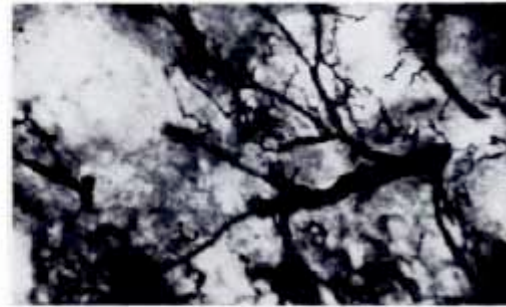
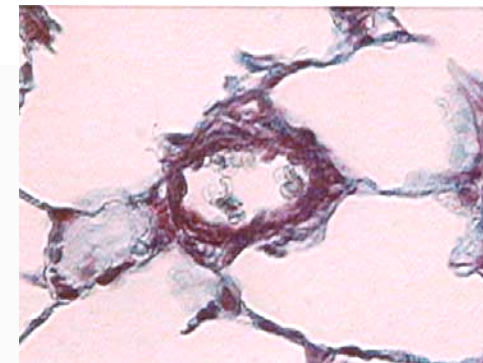
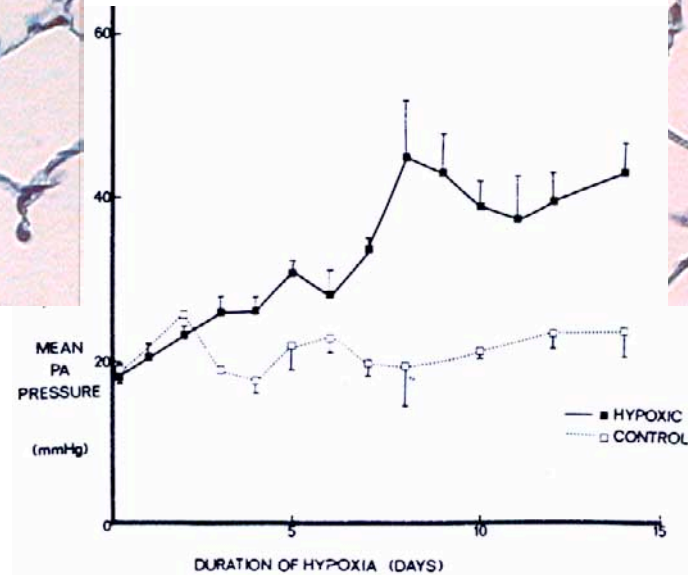
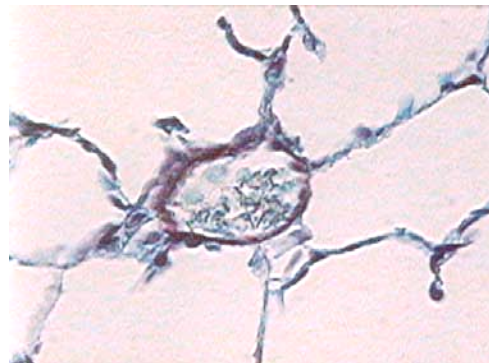


FIGURE 5. Close-up view (original magnification $\times 190$) of alveolar capillary network of Figure 4, shows absence of capillary anastomoses and rare arteriovenous communication. This correlated with markedly elevated hemodynamic pulmonary vascular resistance, 7-10 mm Hg min liter.

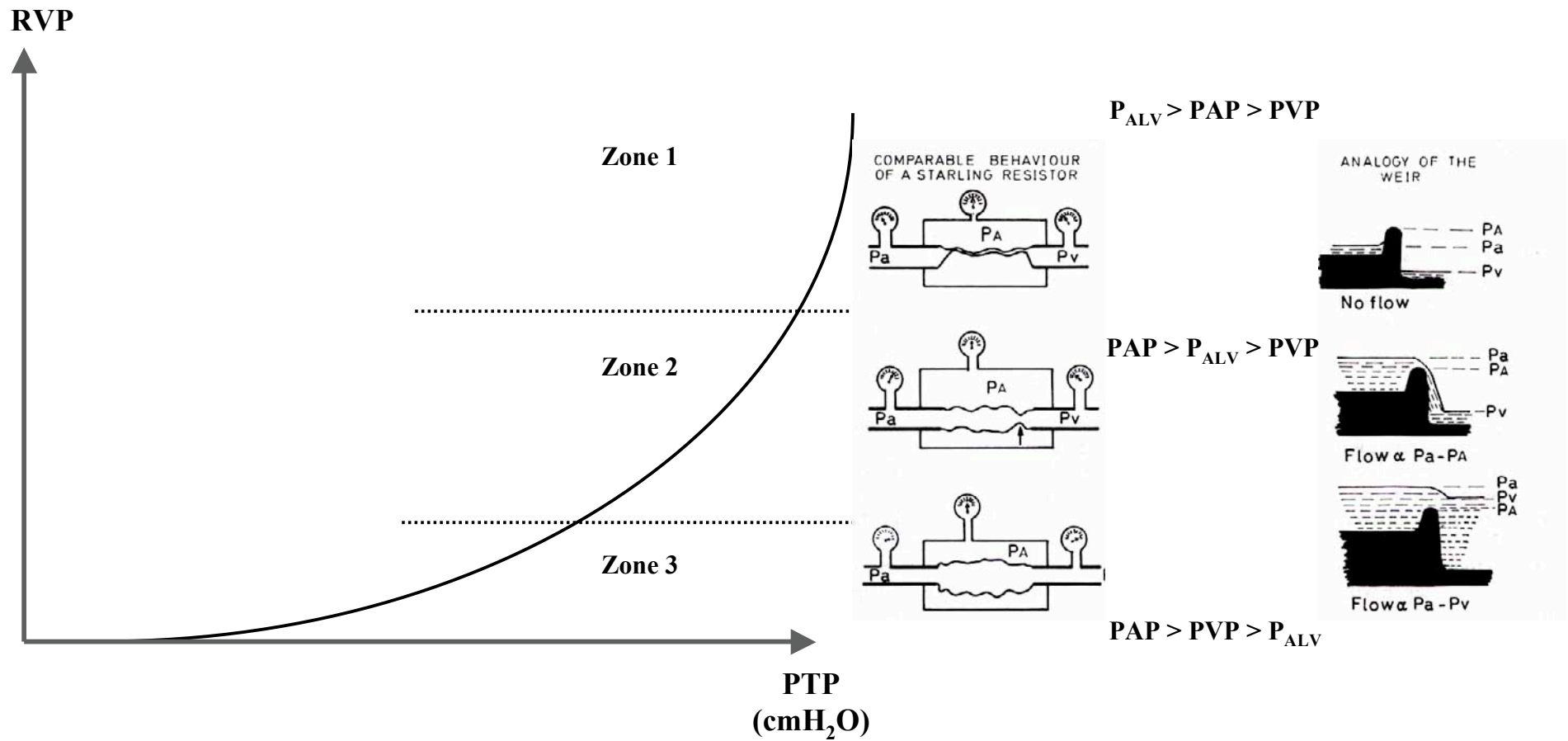




WHITTENBERGER
JAP 1960



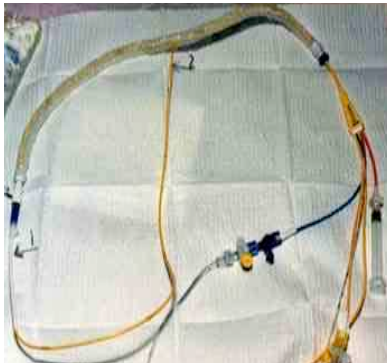
WEST
JAP 1964



INVASIVE

**“MINIMALLY”
INVASIVE**

**NON
INVASIVE**



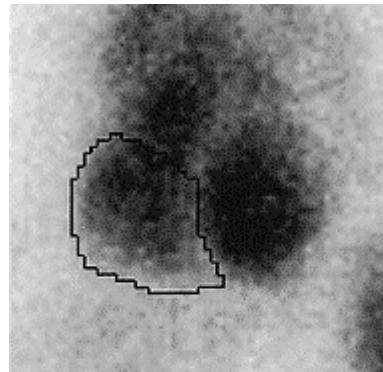
PAC

**Fast response
thermistor**

- RV volumes
- RV EF
- CO

Pressures

- RAP/PAOP gradient
- PAPd - PAOP



Radionuclide ventriculography

- RV volumes
- RV EF

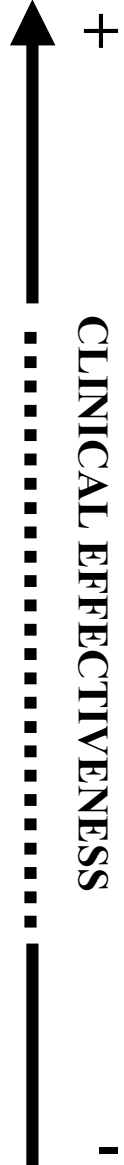


TEE



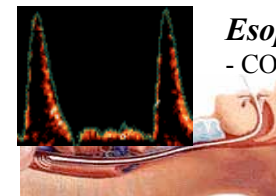
TTE

- RV size (RV/LV EDA)
- Septal motion
- Acceleration time (RV outflow)
- RV SI



CLINICAL EFFECTIVENESS

TPT
- CO



Esophageal Doppler
- CO

I

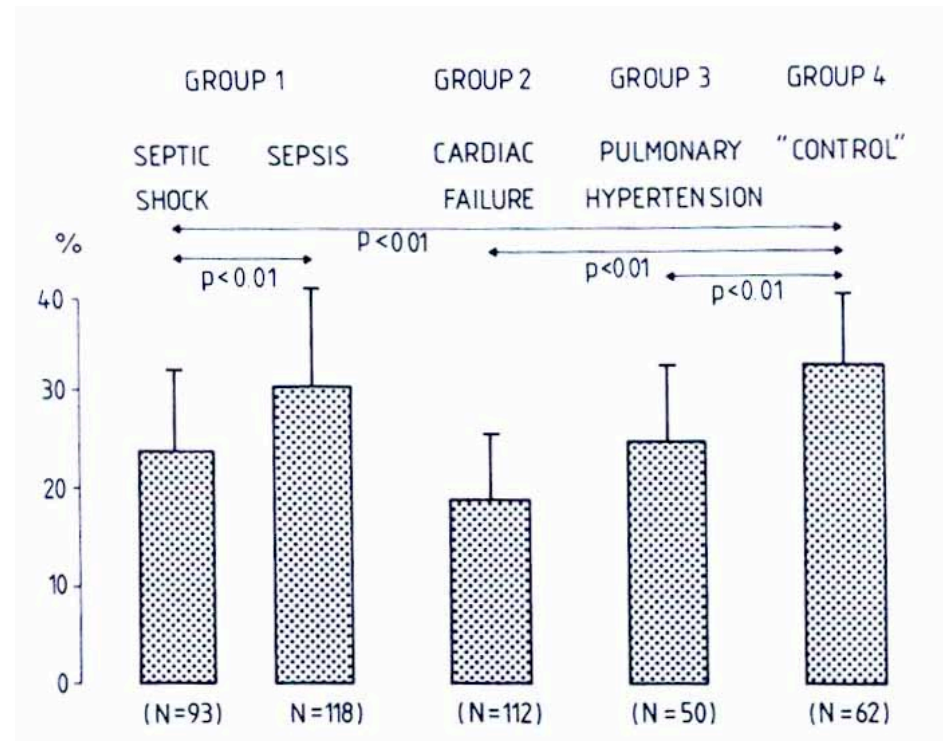
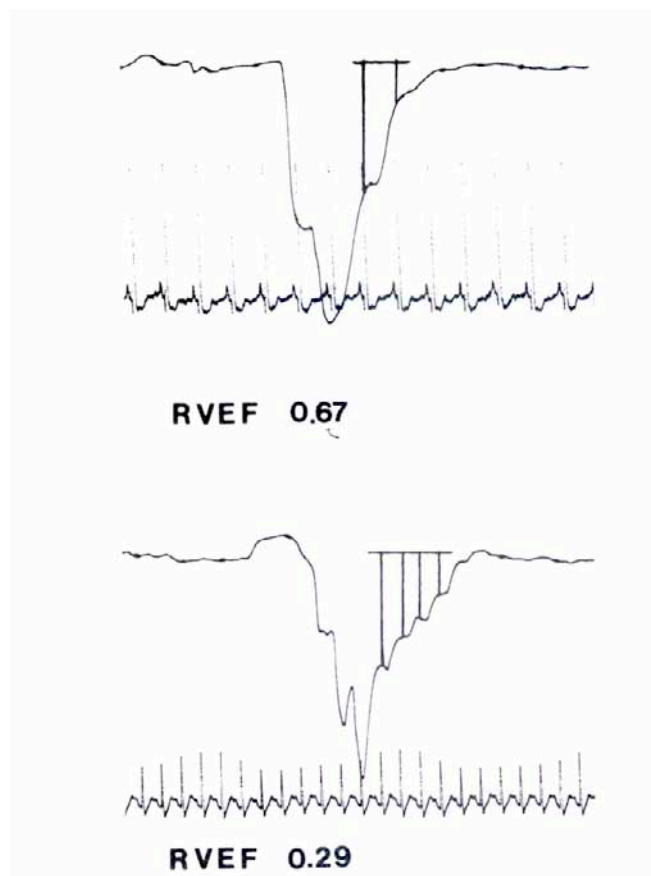
CAP

- FEVD
- PAP, RVP
- PAPd/PCWP gradient
- PVC/PAPO

Right ventricular dysfunction in septic shock: assessment by measurements of right ventricular ejection fraction using the thermodilution technique

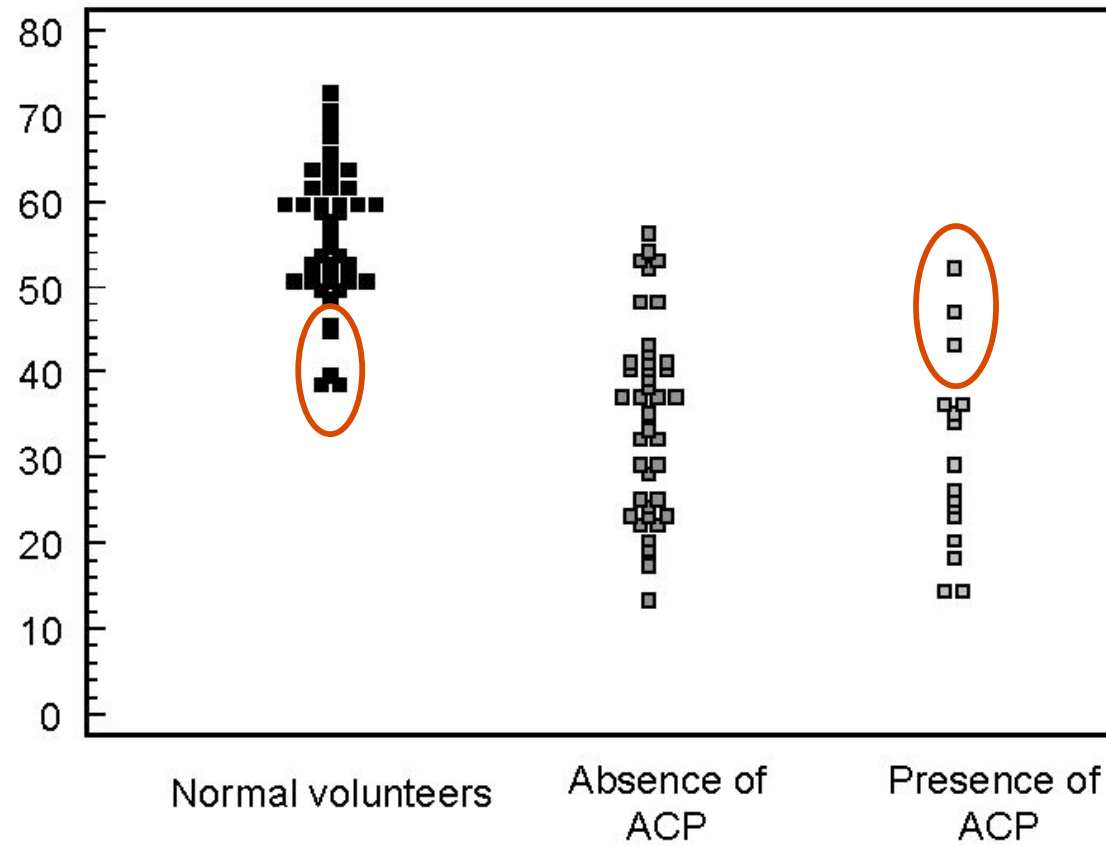
J. L. VINCENT, C. REUSE, N. FRANK, B. CONTEMPRÉ and R. J. KAHN
Department of Intensive Care, Erasme University Hospital, Free University of Brussels, Belgium

Acta Anaesthesiol Scand 1989



RVEF (%)

RVFAC (%)



Vascular Obstruction Causes Pulmonary Hypertension in Severe Acute Respiratory Failure

Warren M. Zapol, Koichi Kobayashi, Michael T. Snider, Reginald Greene and Myron B. Laver

Chest 1977;71;306-307

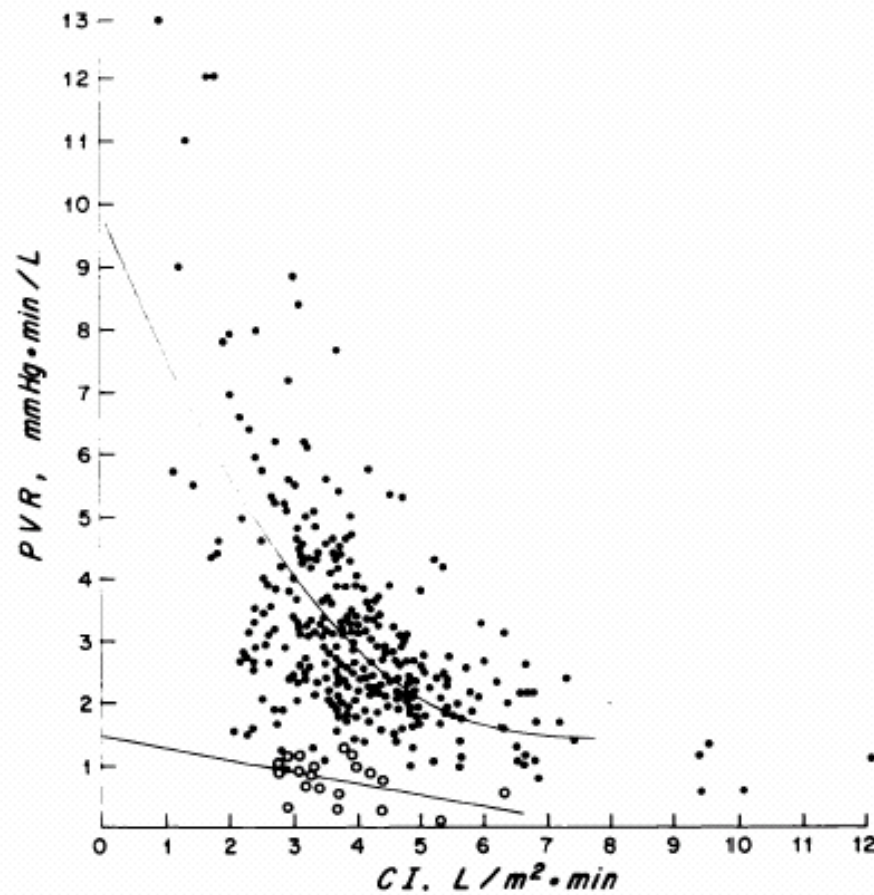


FIGURE 1. Statistical correlation of 290 measurements of PVR and CI on 16 patients with ARF. Normal data from Barratt-Boyes et al.⁽¹⁾ The groups are significantly different ($P < 0.001$).

Significance of the pulmonary artery diastolic-pulmonary wedge pressure gradient in sepsis

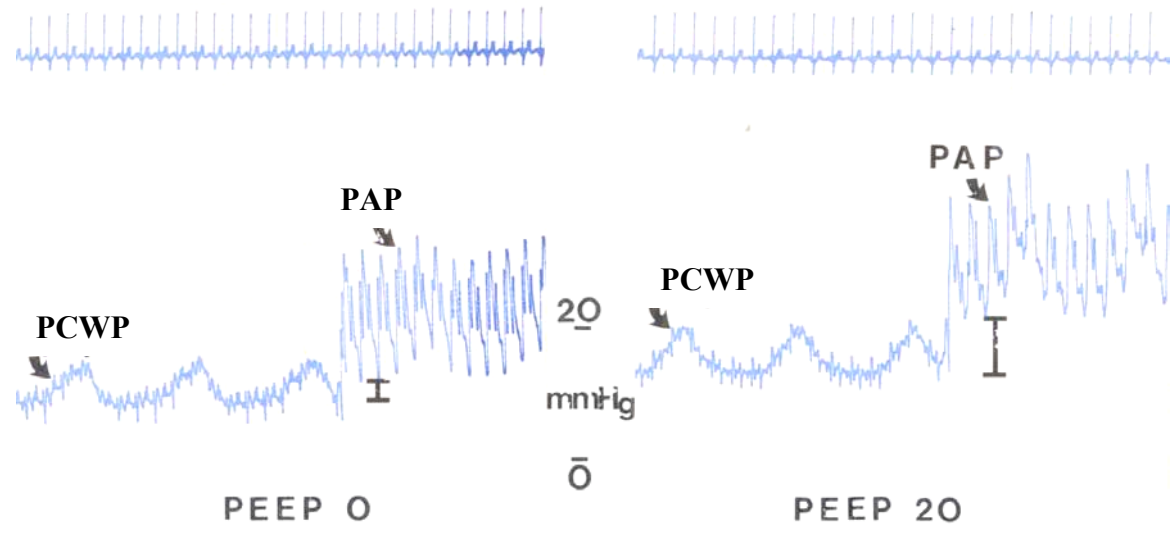
A. M. MARLAND, MD; FREDERICK L. GLAUSER, MD

Crit Care Med 1982

TABLE 2. Causes of widened PAD-PWP gradient

-
1. Pulmonary vasoconstriction and muscular hyperplasia secondary to longstanding hypoxemia as occurs in chronic bronchitis, sleep apnea syndromes, chronic upper airway obstruction, kyphoscoliosis, etc.
 2. Far-advanced fibrotic lung disease, usually with the vital capacity <60% of predicated.
 3. Pulmonary emboli
 4. Primary pulmonary hypertension
 5. Veno-occlusive disease
 6. Longstanding left to right shunts
 7. Longstanding mitral stenosis
 8. Effects of PEEP when the right heart catheter is under zone I conditions
 9. Burns
 10. Sepsis
-

SDRA



David Osman
 Xavier Monnet
 Vincent Castelain
 Nadia Anguel
 Josiane Warszawski
 Jean-Louis Teboul
 Christian Richard
 for the French Pulmonary
 Artery Catheter Study Group

Incidence and prognostic value of right ventricular failure in acute respiratory distress syndrome

Intensive Care Med 2008

Early Predictive Factors of Survival in the Acute Respiratory Distress Syndrome

A Multivariate Analysis

MEHRAN MONCHI, FLORENCE BELLENFANT, ALAIN CARIOU, LUC-MARIE JOLY, DOMINIQUE THEBERT, IVAN LAURENT, JEAN-FRANÇOIS DHAINAUT, and FABRICE BRUNET

The Medical Intensive Care Unit of Cochin-Port-Royal University Hospital, Paris, France

AJRCCM 1998

145 ARDS

Model B			
	Adjusted HR	95%CI	P
MPAP	1.14	1.06–1.23	<0.01
CVP > PAOP	4.65	1.52–14.28	<0.01
SVI	0.96	0.91–1.00	0.05
Age	1.02	0.99–1.06	0.09
Murray score	4.22	0.9–25.2	0.06
PaO ₂ /FiO ₂	0.97	0.95–0.99	<0.01
pHa	0.001	0.00–0.20	<0.01
Cst	0.94	0.90–0.99	<0.05
MAP	0.95	0.91–0.98	<0.01
SvO ₂	0.92	0.87–0.97	<0.01

177 ARDS

Variable	p Value	Odds Ratio	95% Confidence Interval of OR
SAPS-II, per point	< 0.001	1.1	1.06–1.15
McCabe Score, per point	< 0.001	4.0	1.9–8.4
Cirrhosis	0.012	27.0	2–341
Duration of prior mechanical ventilation, per day	0.020	1.1	1.01–1.15
Direct lung injury	< 0.05	2.6	1.1–6.9
Oxygenation index, per point	0.007	1.05	1.02–1.09
Pra/Ppao relation			
Pra ≤ Ppao	Reference	1.0	
Pra > Ppao, right ventricular dysfunction	0.009	5.1	1.5–17.1
No right heart catheterization	0.16	2.5	0.7–8.6

Definition of abbreviations: OR = odds ratio; Ppao = pulmonary occlusive pressure; Pra = right atrial pressure; SAPS = simplified acute physiology score on the first day of ARDS.

II

ECHOCARDIOGRAPHIE

- Taille du VD
- Mouvement du SIV



Surcharge diastolique



Surcharge systolique

CONSEQUENCES DU CPA

SUR HEMODYNAMIQUE

Acute cor pulmonale in acute respiratory distress syndrome
submitted to protective ventilation: Incidence, clinical
implications, and prognosis

CCM 2001

	Group 1 (n = 56)	Group 2 (n = 19)
HR, beats/min	96 ± 19	112 ± 16 ^a
SAP, mm Hg	114 ± 23	123 ± 25
CVP, mm Hg	12 ± 3	16 ± 3 ^a
SI, cm ³ /m ²	32 ± 9	25 ± 9 ^a
CI, L/min/m ²	3.1 ± 0.9	2.7 ± 0.9
LVEDV, cm ³ /m ²	60 ± 16	50 ± 15 ^a
LVESV, cm ³ /m ²	22 ± 10	24 ± 10
LVEF, %	53 ± 11	51 ± 16
E/A ratio, %	1.3 ± 0.4	0.8 ± 0.2 ^a

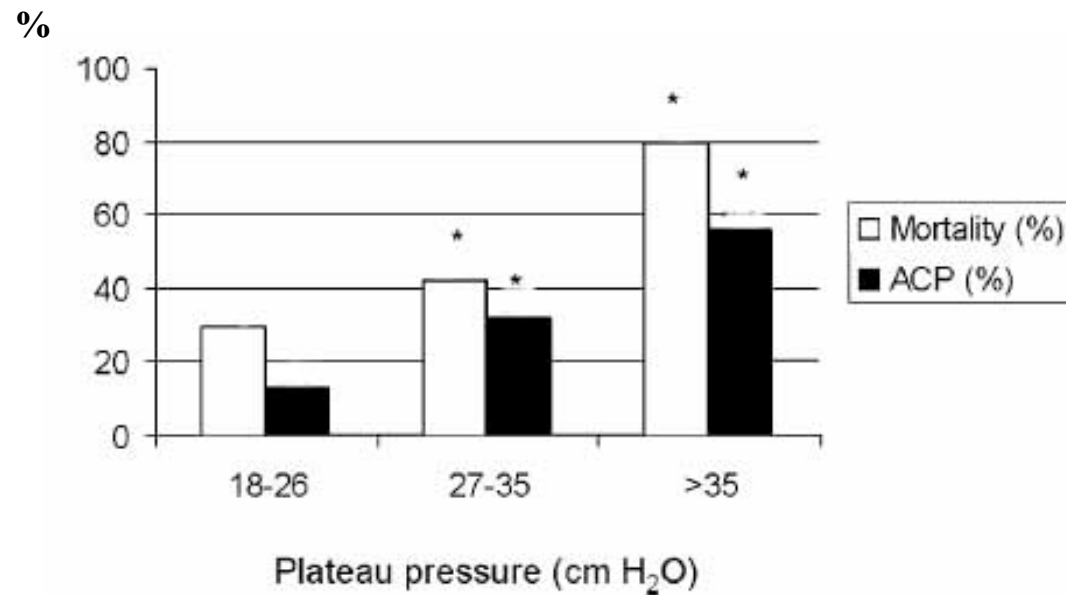
Is there a safe plateau pressure in ARDS? The right heart only knows

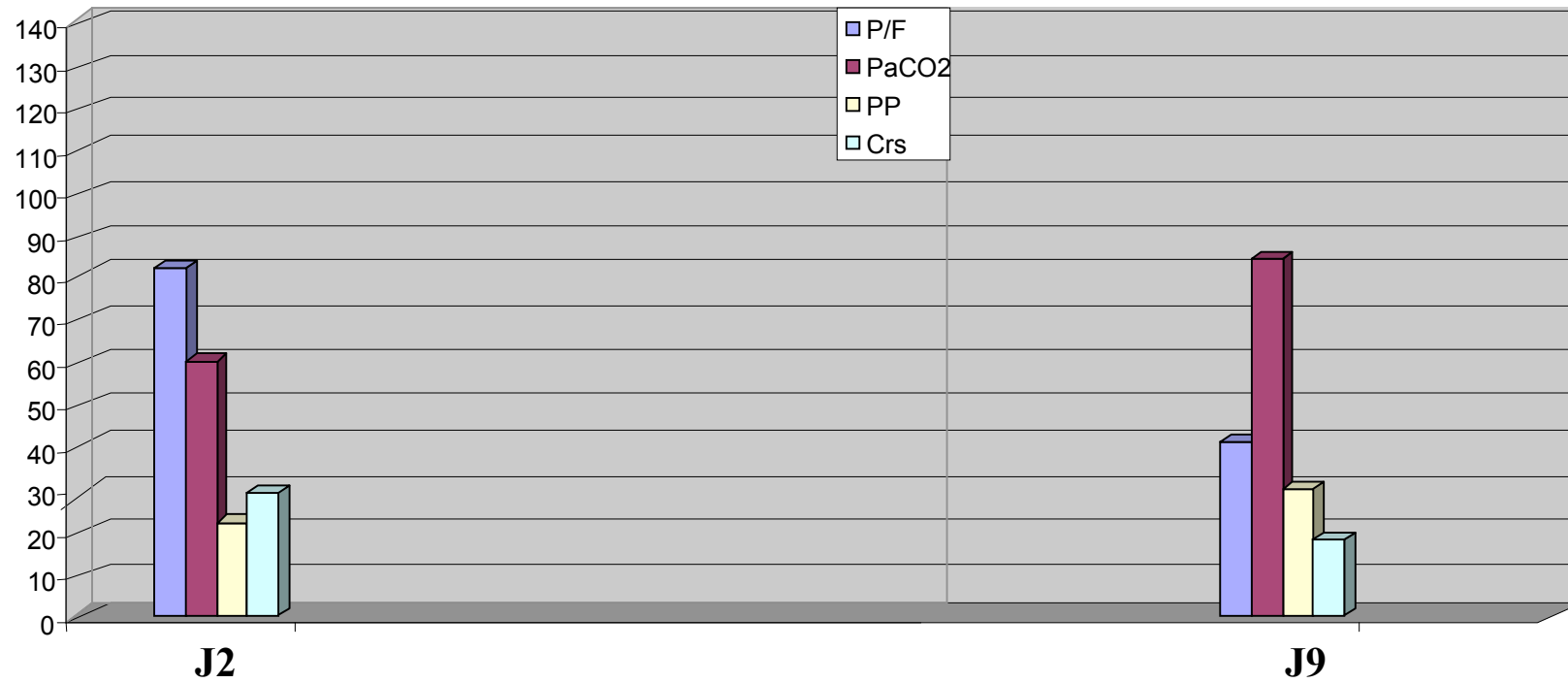
ICM 2007

1980-2006 352 SDRA avec ECHO

1980-1992
156 SDRA
Pas de limitation PP

1993-2006
196 SDRA
Low stretch strategy





NA 0



NA 1.5 $\mu\text{g}/\text{kg}/\text{min}$

EN PRATIQUE CLINIQUE

ADAPTER LES REGLAGES DU
VENTILATEUR A LA FONCTION
VD

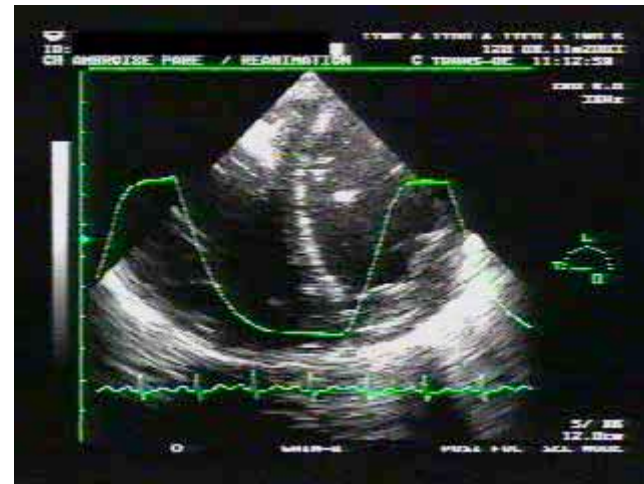
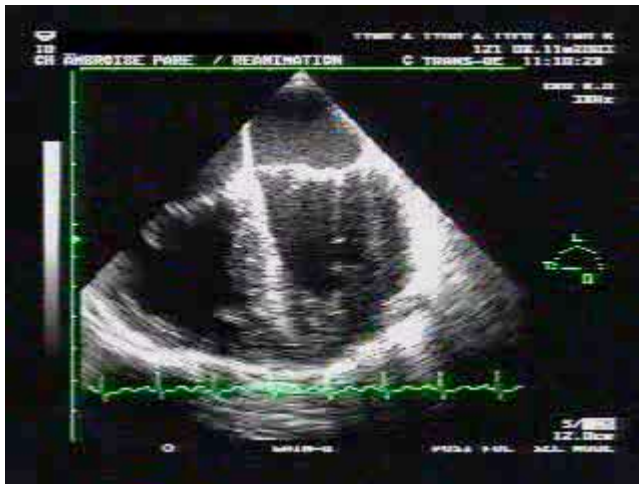
- Patient 41 ans
 - Insuffisance respiratoire aiguë
 - Pneumopathie bilatérale extensive
 - SaO₂ 85% sous 15L/min O₂

- Hémodynamique stable

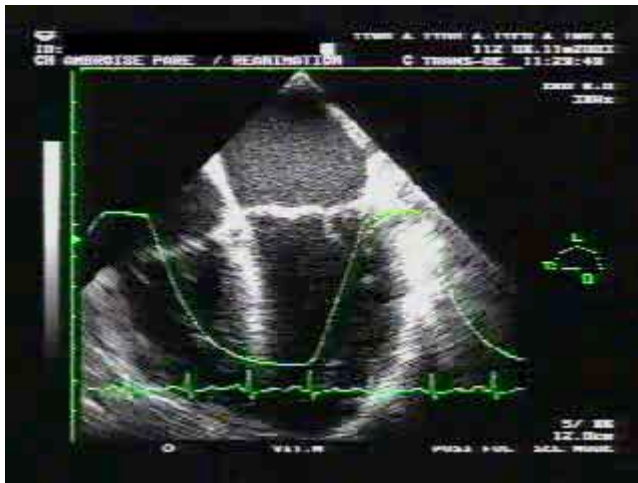
- ETT
 - VG hyperkinétique
 - VD normal

- Épuisement
 - Sédation
 - IOT, VC

- Gaz du sang
 - FiO₂ 0,6, PEEP 5 cmH₂O, VT 400 ml (5,7 ml/kg), FR 20/min
 - PaO₂ 60 mmHg
 - PaCO₂ 67 mmHg
- Pression plateau 33 cmH₂O
- PAS 90 mmHg, Fc 128/min



- Réduction du VT: 350 ml (5 ml/kg)
 - Pression de plateau 26 cmH₂O
 - PAS 123 mmHg
 - Fc 112/min
- Humidificateur chauffant, FR 25/min
 - PaO₂ 60 mmHg
 - PaCO₂ 63 mmHg





Impact of Acute Permissive Hypercapnia and Augmented Positive End-Expiratory Pressure at Constant Plateau Pressure on Right Ventricle Function in Severe Acute Respiratory Distress Syndrome



	MODE A	MODE B
PEEP (cmH ₂ O)	6 [5-7]	12 [11-12]*
TV (ml)	548 [468-605]	336 [260-360]*
PP (cmH ₂ O)	24 [22-27]	24 [22-27]
RR (/min)	15 [15-20]	26 [25-30]*
P/F	88 [60-110]	103 [74-138]*
PaCO ₂ (mmHg)	52 [43-68]	71 [60-94]



	MODE A	MODE B
HR	107 [80-114]	112 [93-118]*
BD (mmol/l)	0 [-5.4 to 1]	-3.7 [-6.45 to -0.3]*
EDA RV/LV	0.6 [0.6-0.8]	0.9 [0.6-1.1]*
LVEIs	1.10 [1.02-1.25]	1.19 [1.07-1.54]**
RVSI (ml/m ²)	23 [21-36]	18 [11-21]*

PEEP 7 PP 27



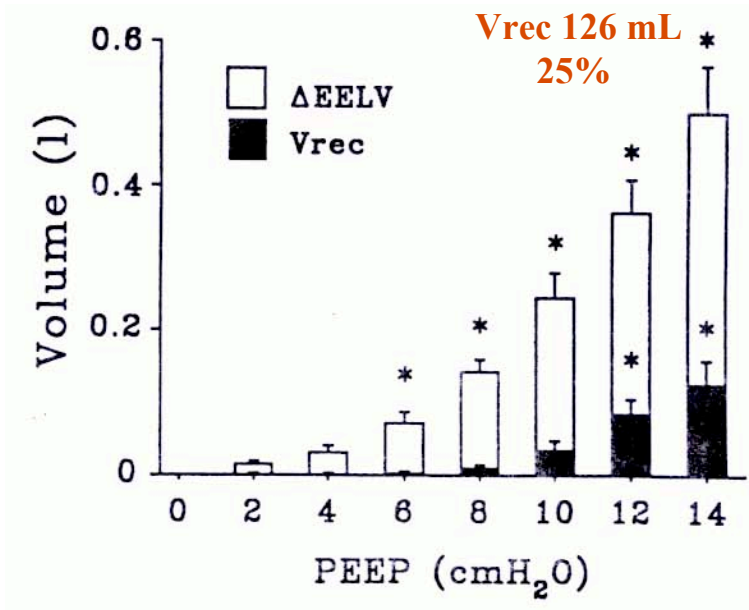
IS_{VD} 23 ml/m²
PAS 135 mmHg
FC 100/mn

PEEP 14 PP 27

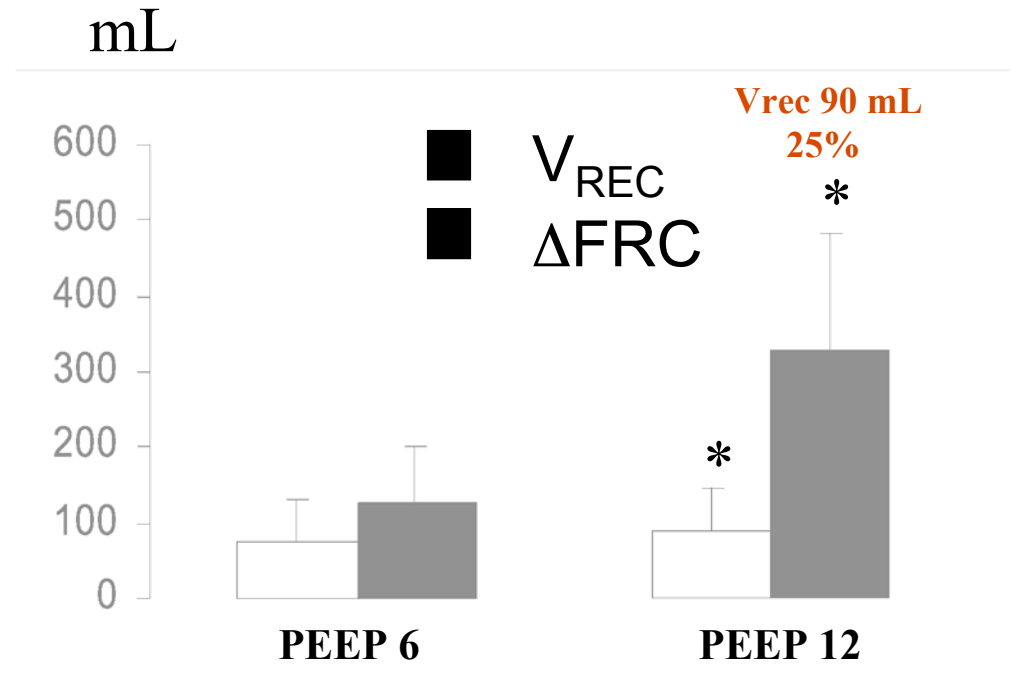


IS_{VD} 12 ml/m²
PAS 115 mmHg
FC 121/mn

EFFET DE LA PEEP?

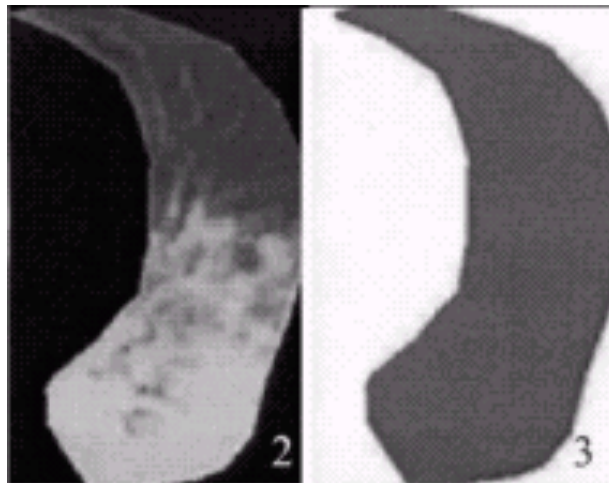


Valta JCC 1993

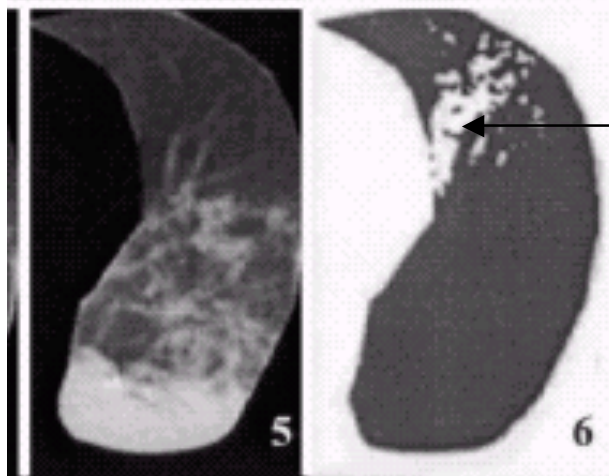


Vieillard-Baron ICM 2003

ZEEP



PEEP 15



Overdistension
Anterior area

Rouby CCM 2004

EFFET DE L'HYPERCAPNIE?

Acute cor pulmonale in acute respiratory distress syndrome submitted to protective ventilation: Incidence, clinical implications, and prognosis

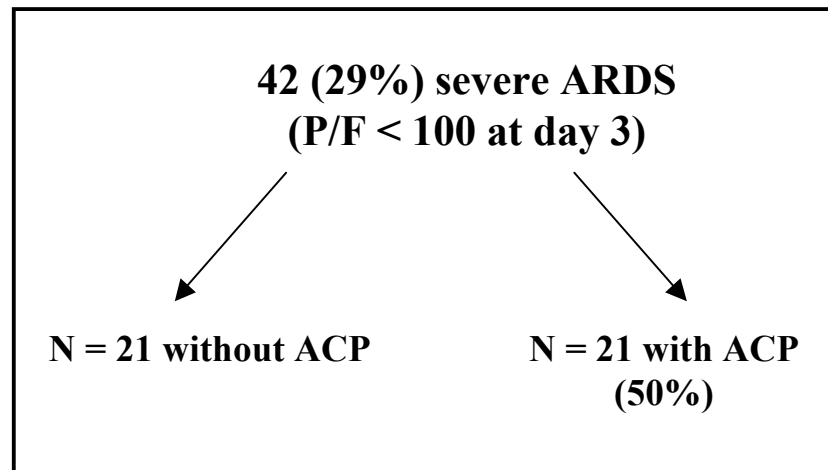
CCM 2001

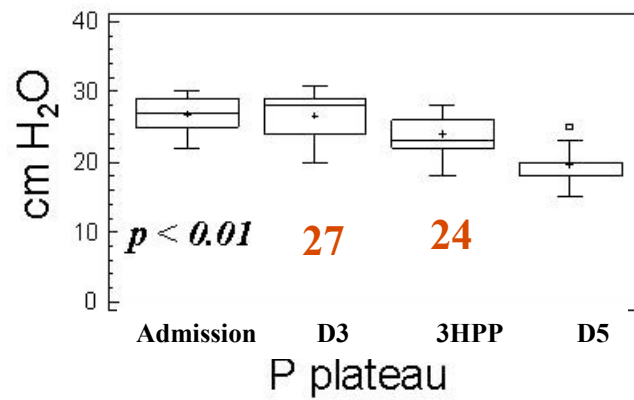
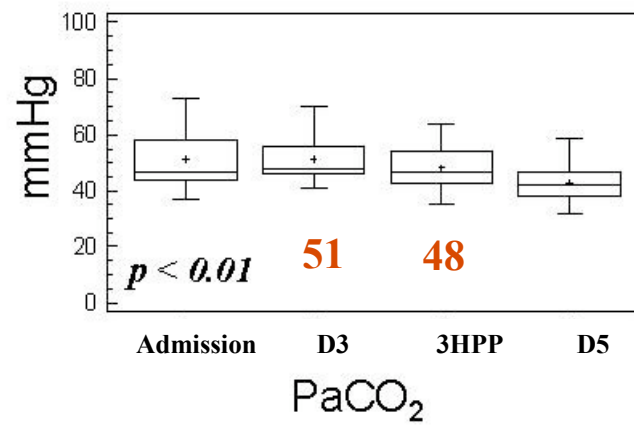
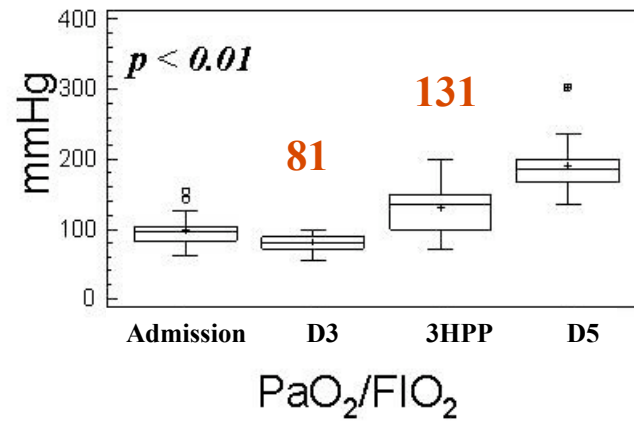
	Group 1	Group 2	Univariate <i>p</i>	Multivariate <i>p</i>	OR (CI)
Age, yrs	56 ± 16	52 ± 17	NS		
SAPS II	55 ± 17	49 ± 16	NS		
LODS	10.1 ± 4	9.9 ± 3.4	NS		
Pao ₂ /Fio ₂ , mm Hg	115 ± 32	87 ± 24	.001	NS	
Paco ₂ , mm Hg	47 ± 9	64 ± 12	.000003	.0001	1.15 (1.05-1.25)
CT, mL/cm H ₂ O	37 ± 11	31 ± 12	NS		
TV, mL/kg	8 ± 1	8 ± 1	NS		
Plateau, cm H ₂ O	23 ± 5	27 ± 4	.004	NS	
PEEP, cm H ₂ O	6 ± 3	9 ± 4	.0003	NS	
Fluid balance, mL	2300 ± 2400	3200 ± 2000	NS		

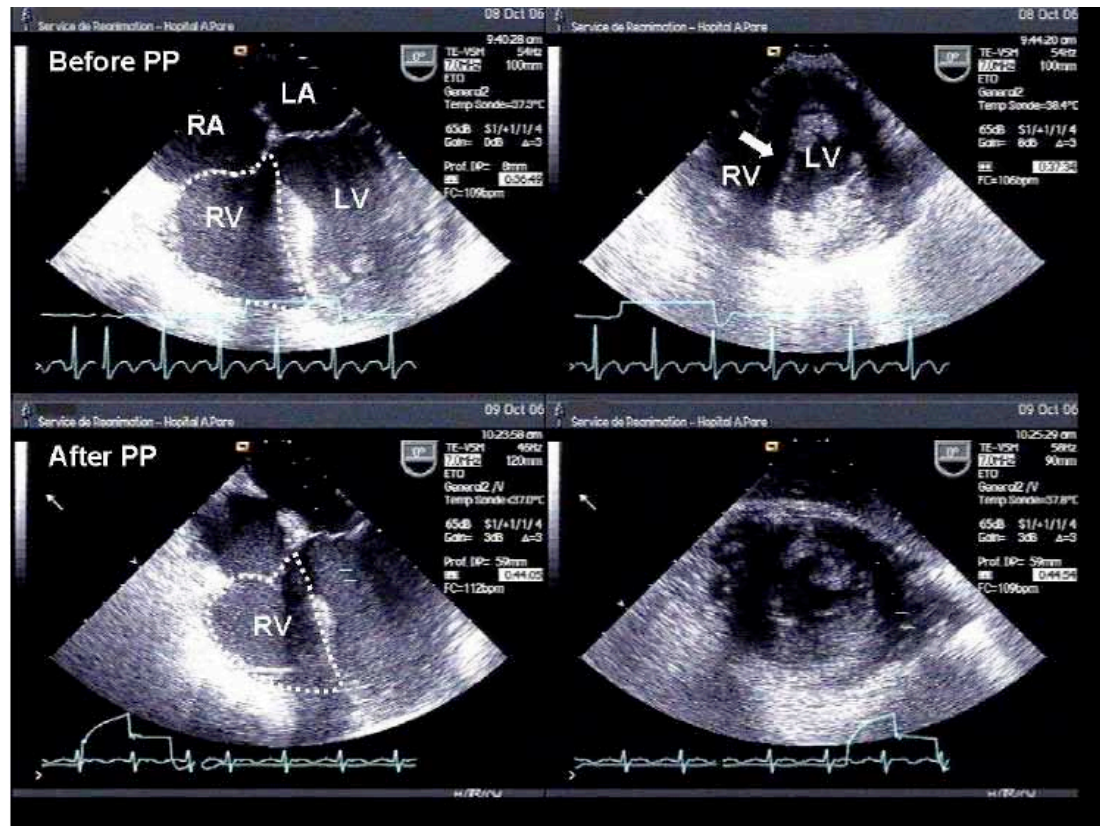
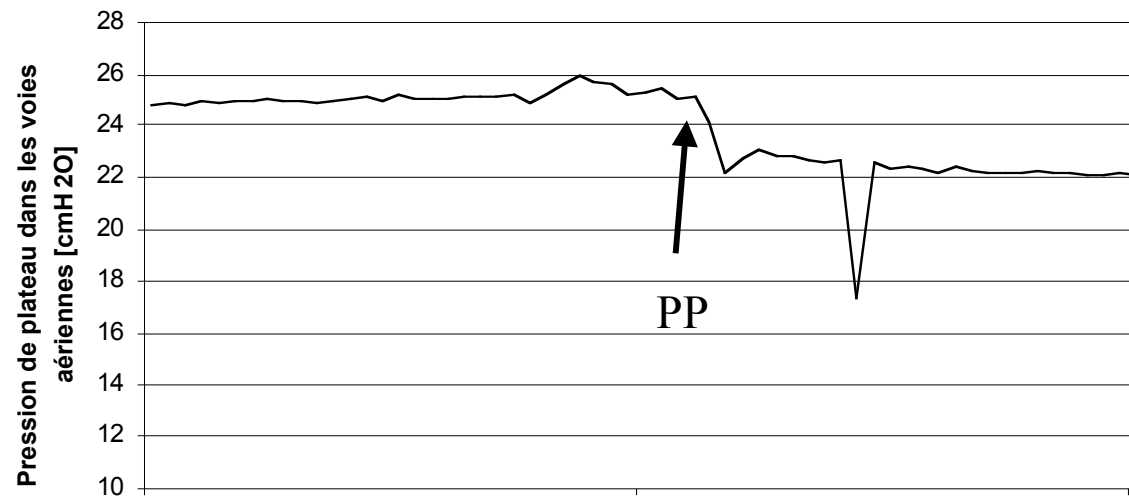
Prone Positioning Unloads the Right Ventricle in Severe ARDS*

Antoine Vieillard-Baron, MD; Cyril Charron, MD; Vincent Caille, MD; Guillaume Belliard, MD; Bernard Page, MD; and François Jardin, MD

Chest 2007







CONCLUSION

- Le SDRA est responsable d'une atteinte de la circulation pulmonaire.
- La conjonction de cette atteinte avec la ventilation en pression positive peut être responsable d'une insuffisance circulatoire liée à une défaillance du VD.
- L'échocardiographie est le meilleur outil pour monitorer cette défaillance.
- La présence d'un CPA oblige à modifier les réglages du respirateur:
 - $PP < 28 \text{ cmH}_2\text{O}$
 - Limiter la PEEP et la PaCO_2
 - Décubitus ventral