Comment régler la Ventilation Mécanique en 2011

Laurent Brochard

HUG
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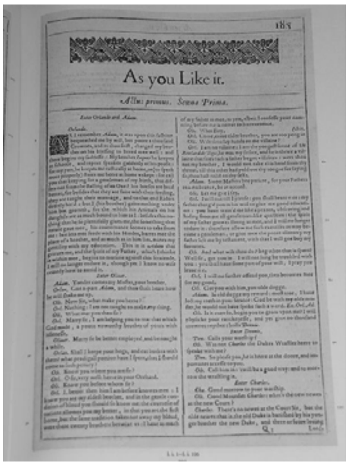
ReVA

2011



As you Like it.

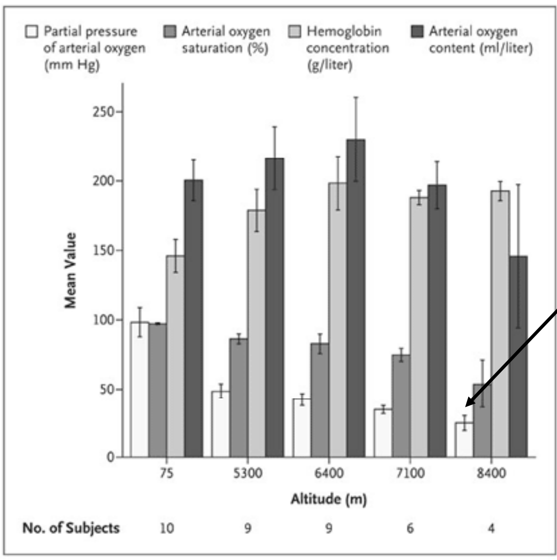
Actus primus. Scœna Prima.

Too much of a good thing?

Objectifs de la ventilation mécanique

- Optimiser l'oxygénation est toujours bon?
- La ventilation mécanique ne peut qu'améliorer le pronostic?
- Permettre la ventilation spontanée est toujours utile?
- Quel degré de liberté pour le patient?

Arterial Blood Gases and Oxygen Content in Climbers on Mount Everest



PaO2 = 24.6 mm Hg
SaO2= 54 %
PaCO2 = 13.3 mm Hg

Grocott NEJM 2009

Association Between Arterial Hyperoxia Following Resuscitation From Cardiac Arrest and In-Hospital Mortality

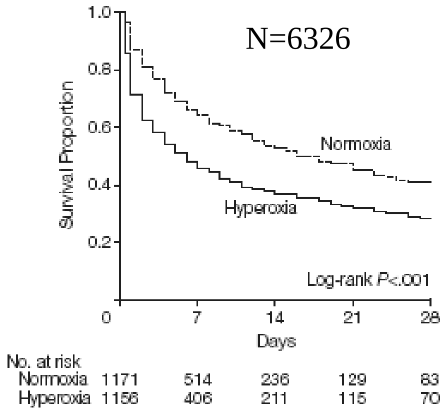
J. Hope Kilgannon, MD
Alan E. Jones, MD
Nathan I. Shapiro, MD, MPH
Mark G. Angelos, MD
Barry Milcarek, PhD
Krystal Hunter, MBA
Joseph E. Parrillo, MD
Stephen Trzeciak, MD, MPH
for the Emergency Medicine Shock Research Network (EMShockNet) Investigators

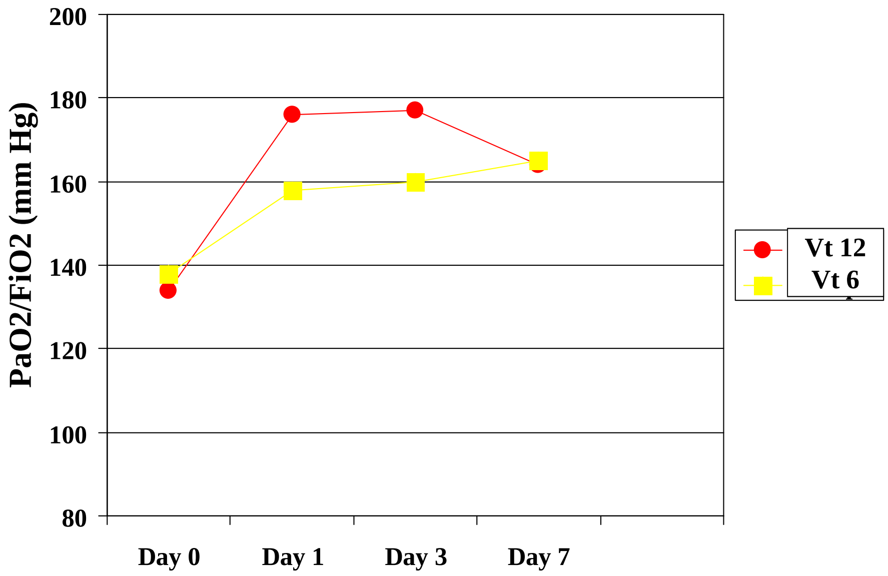
Context Laboratory investigations suggest that exposure to hyperoxia after resuscitation from cardiac arrest may worsen anoxic brain injury; however, clinical data are lacking.

Object
increase

Design
critical c
and 200
diac arre
terial blc
divided
values o
hypoxia
<300);

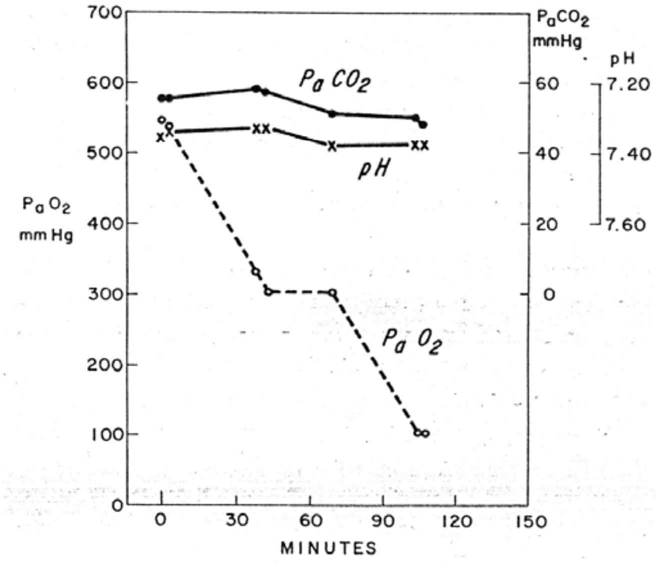
Main C





PaO2/FiO2 ratio in ARDS: comparison of two ventilatory modes

Why did we use large tidal volumes?



Bendixen 1963

Occult Positive End-Expiratory Pressure in Mechanically Ventilated Patients with Airflow Obstruction[1-2]

The Auto-PEEP Effect

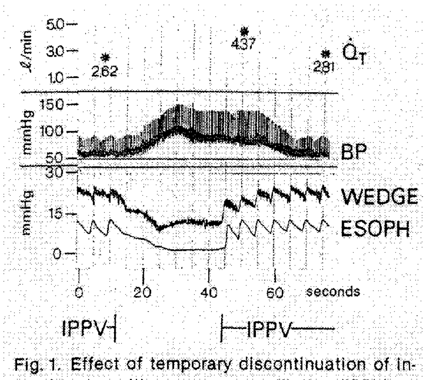


Fig. 1. Effect of temporary discontinuation of intermittent positive pressure ventilation (IPPV) on cardiac output ($\dot{Q}T$), systemic blood pressure (BP), wedge and esophageal (ESOPH) pressures in a patient with severe airflow obstruction (Patient 1).

Pepe & Marini . ARRD 1982

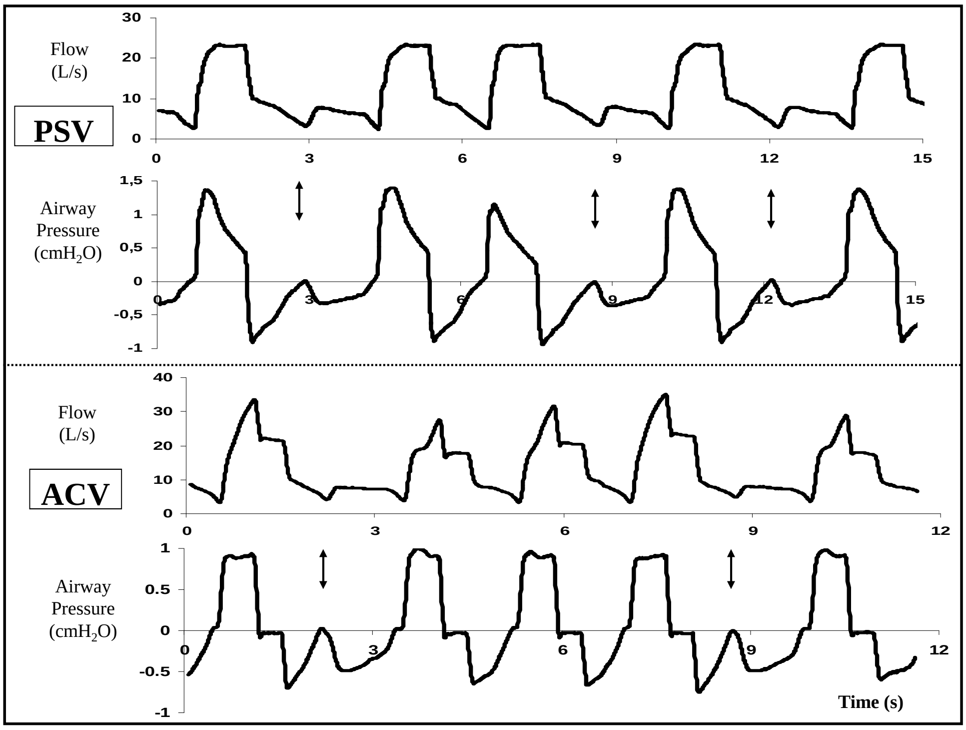
Mechanical Controlled Hypoventilation in Status Asthmaticus[1,2]

TABLE 2

PROGNOSIS IN PATIENTS REQUIRING MECHANICAL VENTILATION IN STATUS ASTHMATICUS

Study	Year	Episodes (*n*)	Deaths	Mortality (%)
Riding and Ambiavagar (8)	1967	26	4	15
Iisalo and associates (4)	1969	29	4	14
Lissac and Labrousse (5)	1971	19	4	21
Sheehy and associates (10)	1972	22	2	9
Scoggin and associates (9)	1977	21	8	38
Cornil and associates (25)	1977	58	6	10
Westerman and associates (12)	1979	42	4	9, 5
Webb and associates (11)	1979	20	7	35
Picado and associates (7)	1983	26	6	23
Present study	1983	34	0	0

Darioli & Perret . ARRD 1984

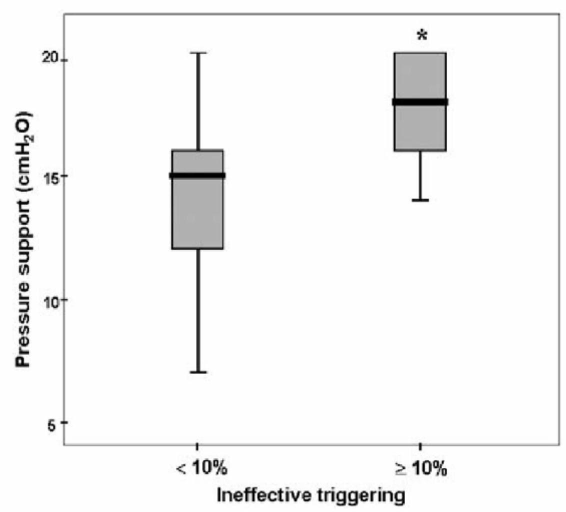


Intensive Care Med (2006) 32:1515–1522
DOI 10.1007/s00134-006-0301-8

ORIGINAL

Arnaud W. Thille
Pablo Rodriguez
Belen Cabello
François Lellouche
Laurent Brochard

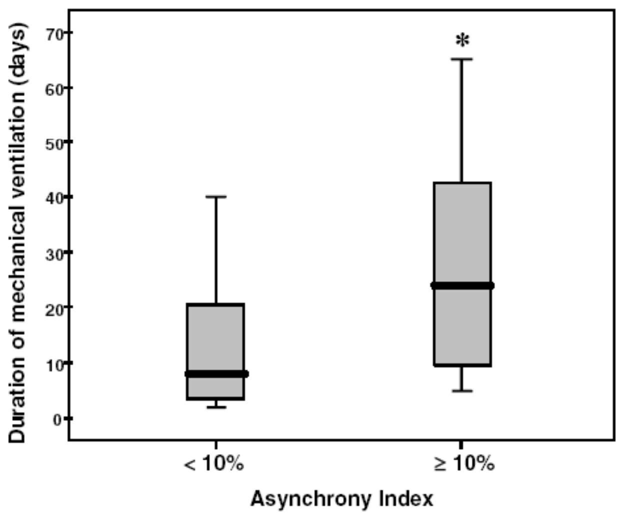
Patient-ventilator asynchrony during assisted mechanical ventilation



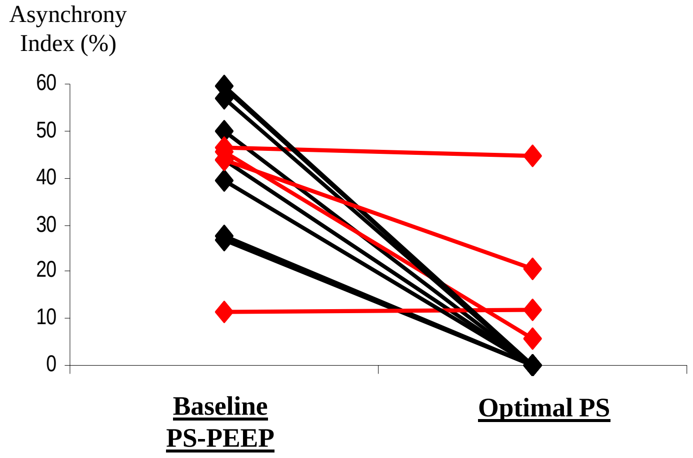
COPD

- Higher PS level
- Larger tidal volume
- Alkalosis

Thille et al., Intensive Care Med 2006; 32: 1515-1522



Thille A. ICM 2006



Thille et al., Intensive Care Med 2008

Reducing tidal volume to reduce patient-ventilator asynchrony

	Baseline PS	Optimal PS
PS (cmH_2O)	20.0 [19.5-20.0]	13.0 [12.0-14.0]
RR ventilator	16.1 [12.4-17.2]	22.4 [22.0-31.3] *
RR patient	26.5 [23.1-31.9]	29.4 [24.6-34.5]
Ti Ventilator (s)	1.3 [1.0-1.8]	0.8 [0.8-1.0] *
PTP (cmH_2O.s/min)	61 [58-81]	82 [61-106]
VT (ml)	571 [487-638]	349 [336-368] *
VT (ml/kg, IBW)	10.2 [7.2-11.5]	5.9 [4.9-6.7] *

Thille et al., Intensive Care Med 2008

Intensive Care Med (1990) 16:372–377

Intensive Care Medicine
© Springer-Verlag 1990

Originals

Low mortality associated with low volume pressure limited ventilation with permissive hypercapnia in severe adult respiratory distress syndrome

K. G. Hickling, S. J. Henderson and R. Jackson

Departments of Intensive Care and Radiology, Christchurch Hospital, Christchurch, New Zealand

Received: 28 September 1989; accepted: 16 February 1990

Abstract. Many animal studies have shown that high peak inspiratory pressures (PIP) during mechanical ventilation can induce acute lung injury with hyaline membranes. Since 1984 we have limited PIP in patients with ARDS by reducing tidal volume, allowing spontaneous breathing with SIMV and disregarding hypercapnia. Since 1987 50 patients with severe ARDS with a "lung injury score" ⩾2.5 and a mean PaO_2/FiO_2 ratio of 94 were managed in this manner. The mean maximum $PaCO_2$ was 62 mmHg, the highest being 129 mmHg. The hospital mortality was significantly lower than that predicted by Apache II (16% vs. 39.6%, $\chi^2 = 11.64$, $p < 0.001$). Only one death was due to respiratory failure, caused by pneumocystis pneumonia. 10 patients had a "ventilator score" >80, which has previously predicted 100% mortality from respiratory failure. Only 2 died, neither from respiratory failure. There was no significant difference in lung injury score, ventilator score, PaO_2/FiO_2 or maximum $PaCO_2$ between survivors and non-survivors. We suggest that this ventilatory management may substantially reduce mortality in ARDS, particularly from respiratory failure.

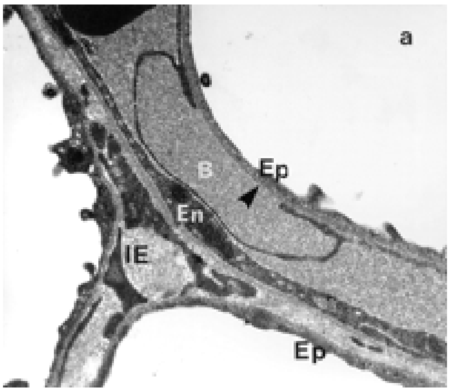
Key words: ARDS – Mortality – Mechanical ventilation – Hypercapnia – Fluid Therapy

ventilation or apnoeic oxygenation with extracorporea CO_2 removal, which avoid high PIP [1, 2, 9–11]. It ap pears that positive end-expiratory pressure (PEEP) ma also reduce the extent of such lung injury [7, 8].

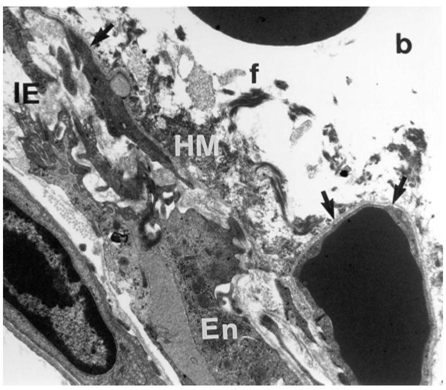
Because of this and related evidence [12, 13], sinc 1984 we have gradually adopted the policy of limitin PIP in patients with severe ARDS. Since using this ap proach to management we have been aware of only death from progressive respiratory failure in patients wit ARDS, and we wondered whether this ventilatory man agement may have resulted in a reduction in mortalit from respiratory failure. This study was conducted t evaluate the total mortality and that due to respirator failure in patients managed in this manner.

Patient management

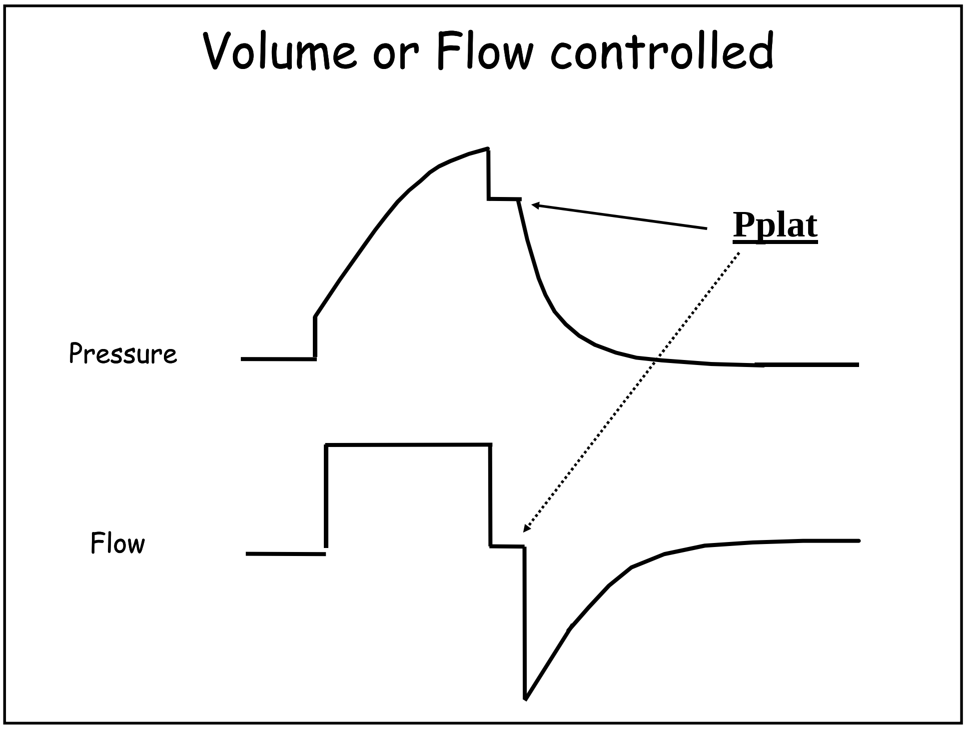
During the period of this study, patients with ARDS had the PIP limi ed to <30 cm H_2O when this was easily achieved, and always <40 cm H_2O, by reducing Vt as necessary, sometimes to volumes low as 350 ml (5 ml/kg) in adults with very low lung compliance. Th was done in conjunction with spontaneous breathing using synchroni ed intermittent mandatory ventilation (SIMV), and frequently result in a rapid spontaneous respiratory rate and hypercapnia. In patien with less severe lung injury however, spontaneous hyperventilation fr quently resulted in a $PaCO_2$ within or even below the normal ran even when PIP was limited. We did not increase the Vt or minute vent

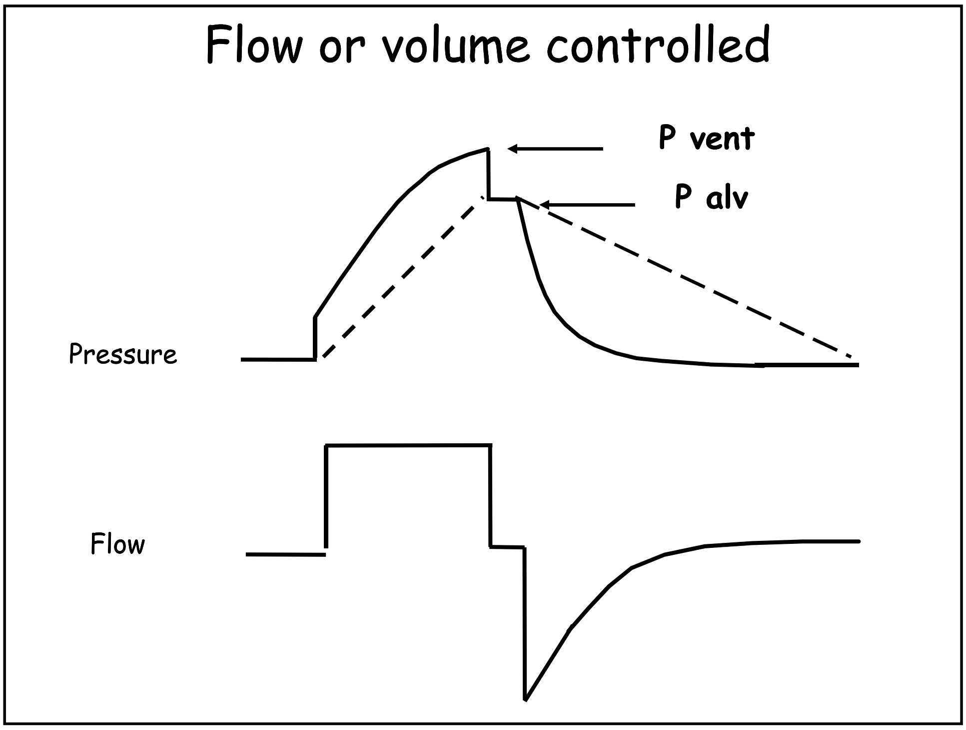


Webb, HH, Tierney DF (1974) Experimental pulmonary edema due to intermittent positive pressure ventilation with high inflation pressures. Protection by positive end-expiratory pressure. Am Rev Respir Dis 110:556–565



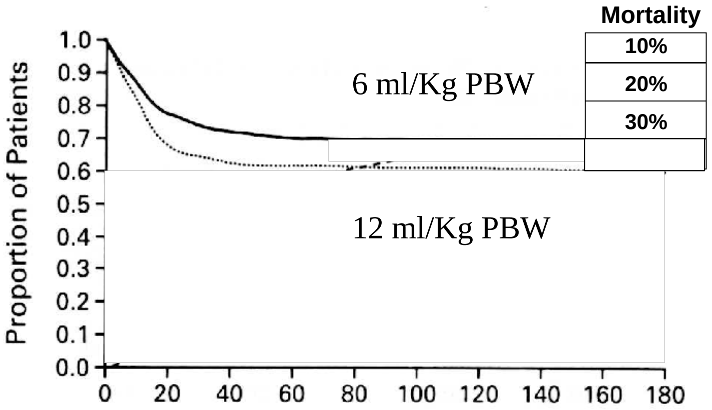
Dreyfuss D & Saumon G. AJRCCM 1998; 157: 294-323


Volume or Flow controlled
Pplat
Pressure
Flow


Flow or volume controlled
P vent
P alv
Pressure
Flow

The Acute Respiratory Distress Syndrome Network.

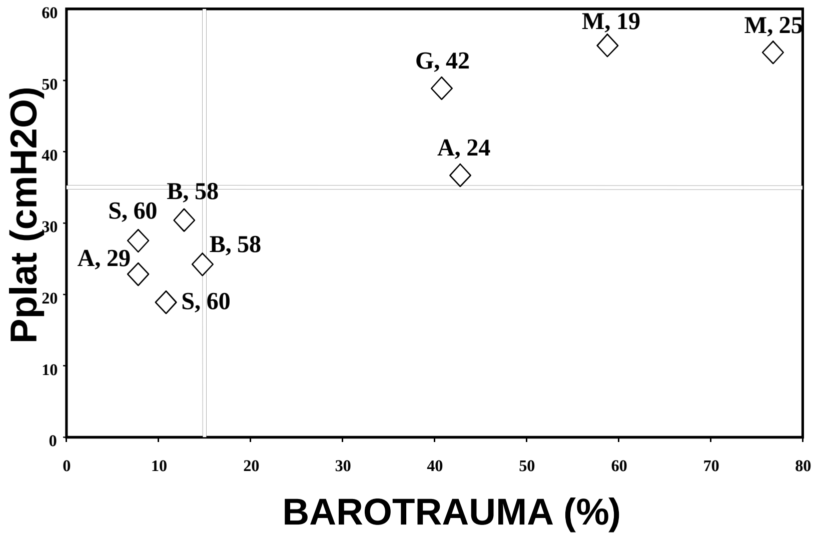
N Engl J Med 2000; 342:1301-1308



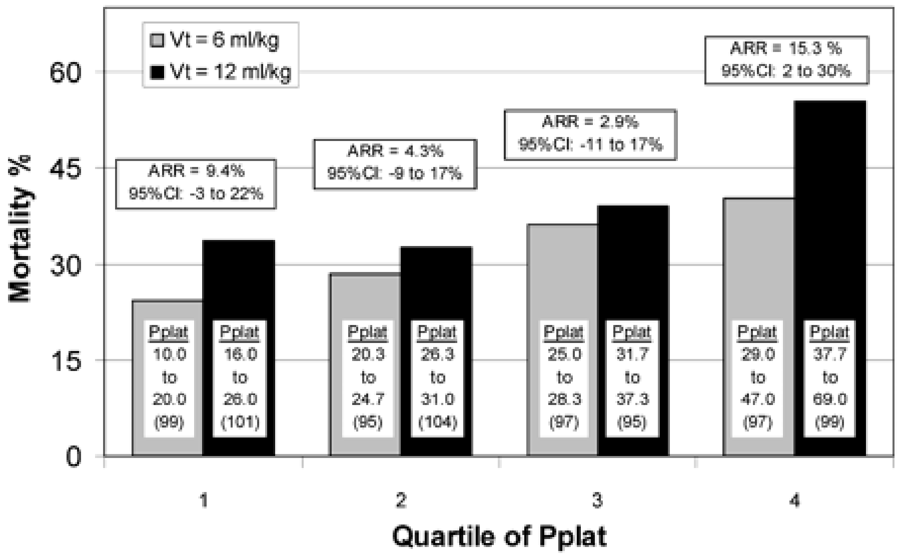
Ventilation Mécanique & Mortalité

Effets de la VM dans le SDRA? Pourquoi réduire le VT est-il bénéfique?

- Bénéfice de la VM: améliorer les échanges gazeux, mettre au repos les muscles respiratoires
- Risques: VILI, effets hémodynamiques
- VT coupable? Distension? Ouverture/fermetures répétées de territoires? Autre effet lié à la taille du VT?



Boussarssar Intensive Care Med 2001



Hager AJRCCM 2005

The Lancet · Saturday 12 August 1967

ACUTE RESPIRATORY DISTRESS IN ADULTS

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Summary The respiratory-distress syndrome in 12 patients was manifested by acute onset of tachypnœa, hypoxæmia, and loss of compliance after a variety of stimuli; the syndrome did not respond to usual and ordinary methods of respiratory therapy. The clinical and pathological features closely resembled those seen in infants with respiratory distress and to conditions in congestive atelectasis and postperfusion lung. The theoretical relationship of this syndrome to alveolar surface active agent is postulated. Positive end-expiratory pressure was most helpful in combating atelectasis and hypoxæmia. Corticosteroids appeared to have value in the treatment of patients with fat-embolism and possibly viral pneumonia.

of lung compliance, and diffuse alveolar infiltration seen on chest X-ray.

No patient had a previous history of respiratory failure. 1 patient gave a history of mild asthma since childhood but had no disability or recent attacks. Another patient had a chronic cough that was attributed to cigarette smoking. The remaining 10 patients did not have any previous pulmonary disease.

Severe trauma preceded respiratory distress in 7 patients (table I). Viral infection in 4 patients and acute pancreatitis in 1 patient were precipitating factors in the remainder. Respiratory distress occurred as early as one hour and as late as ninety-six hours after the precipitating illness or injury. Shock of varying degree and duration was present in 5 patients and excessive fluid administration occurred in 7 patients. 4 patients developed acidosis with pH less than 7·3 before the onset of respiratory distress.

Methods

All patients were admitted to intensive-care units of the surgical or medical services. Blood-gas studies were performed on arterial blood drawn by percutaneous puncture of either brachial or femoral artery. In most instances, blood was drawn only during a steady state. P_aO_2 measurements were determined with a Clark electrode and oxygen saturation was measured on

TABLE I—ACUTE RESPIRATORY DISTRESS

Case	Age (yr.)	Sex	Illness	Onset of acute respiratory distress (hr. after illness)	Possible contributory factors		
					Hypotension	Acidosis	Fluid overload
1	29	M	Multiple trauma; lung contusion	8	++	++	+++ 7500 ml.
2	19	F	Multiple trauma; lung laceration and contusion	1	+++	++	+++ 3000 ml.
3	19	F	Multiple trauma	72	+	[illegible]	[illegible]

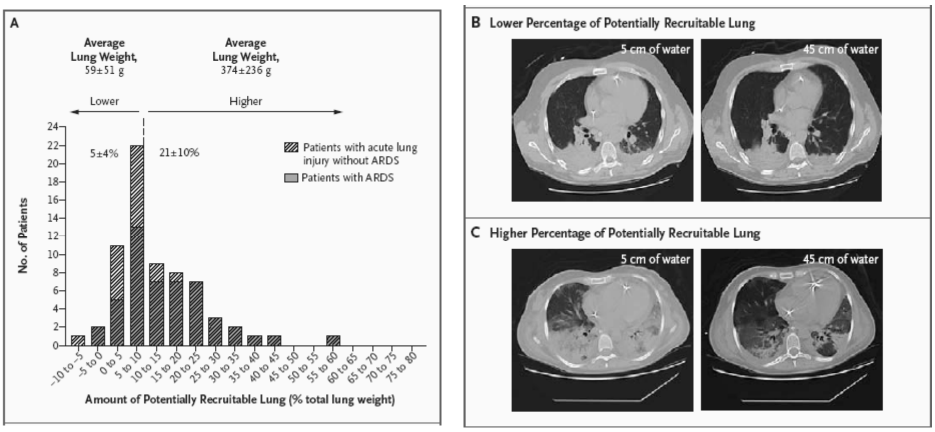
Pourquoi recruter le poumon?

- 1) Améliorer les échanges gazeux
- 2) Protéger le poumon?

- Différencier les recruteurs et les non recruteurs pourrait être important pour éviter une distension inutile (strain)

Lung Recruitment in Patients
with the Acute Respiratory Distress Syndrome

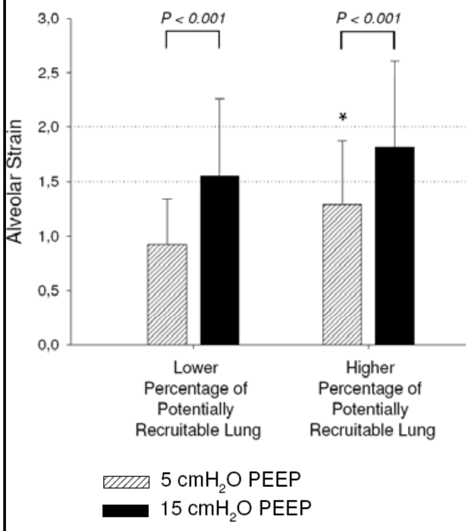
- 68 patients with ALI / SDRA
- Whole lung CT at 5, 15 and 45 cmH_2O



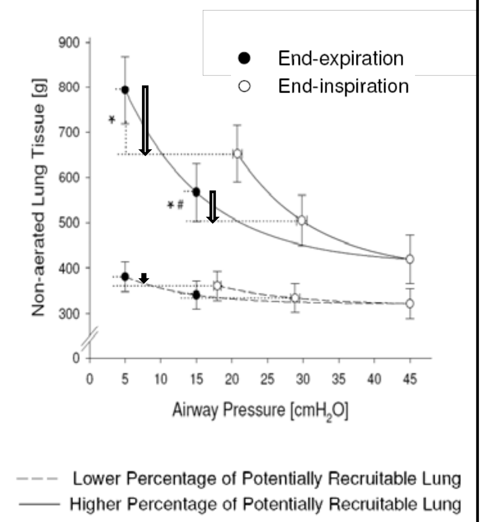
Gattinoni et al. NEJM 2006

Lung Opening and Closing during Ventilation of SDRA

Strain



Recruitment



Caironi et al. AJRCCM 2009

CARING FOR THE CRITICALLY ILL PATIENT

JAMA®

Online article and related content current as of March 7, 2010.

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

Systematic Review and Meta-analysis

Matthias Briel, MD, MSc
Maureen Meade, MD, MSc
Alain Mercat, MD
Roy G. Brower, MD
Daniel Talmor, MD, MPH
Stephen D. Walter, PhD
Arthur S. Slutsky, MD
Eleanor Pullenayegum, PhD
Qi Zhou, PhD
Deborah Cook, MD, MSc
Laurent Brochard, MD
Jean-Christophe M. Richard, MD
Francois Lamontagne, MD
Neera Bhatnagar, MLIS
Thomas E. Stewart, MD
Gordon Guyatt, MD, MSc

Context Trials comparing higher vs lower levels of positive end-expiratory pressure (PEEP) in adults with acute lung injury or acute respiratory distress syndrome (ARDS) have been underpowered to detect small but potentially important effects on mortality or to explore subgroup differences.

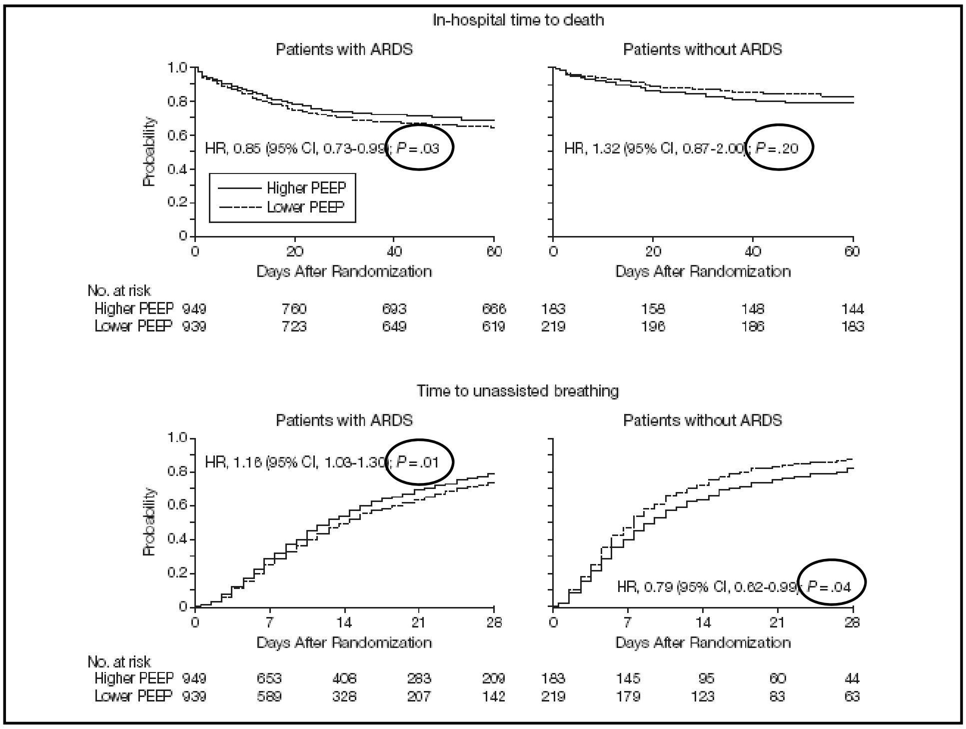
Objectives To evaluate the association of higher vs lower PEEP with patient-important outcomes in adults with acute lung injury or ARDS who are receiving ventilation with low tidal volumes and to investigate whether these associations differ across prespecified subgroups.

Data Sources Search of MEDLINE, EMBASE, and Cochrane Central Register of Controlled Trials (1996-January 2010) plus a hand search of conference proceedings (2004-January 2010).

Study Selection Two reviewers independently screened articles to identify studies randomly assigning adults with acute lung injury or ARDS to treatment with higher vs lower PEEP (with low tidal volume ventilation) and also reporting mortality.

Data Extraction Data from 2299 individual patients in 3 trials were analyzed using uniform outcome definitions. Prespecified effect modifiers were tested using multivariable hierarchical regression, adjusting for important prognostic factors and clustering effects.

Results There were 374 hospital deaths in 1136 patients (32.9%) assigned to treatment with higher PEEP and 409 hospital deaths in 1163 patients (35.2%)



The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 NOVEMBER 13, 2008 VOL. 359 NO. 20

Mechanical Ventilation Guided by Esophageal Pressure in Acute Lung Injury

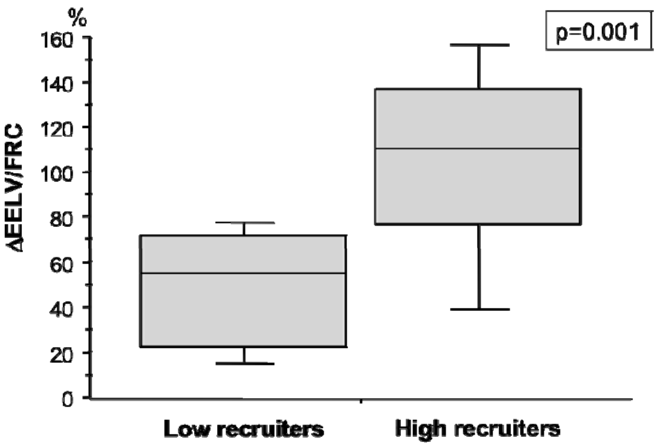
Daniel Talmor, M.D., M.P.H., Todd Sarge, M.D., Atul Malhotra, M.D., Carl R. O'Donnell, Sc.D., M.P.H., Ray Ritz, R.R.T., Alan Lisbon, M.D., Victor Novack, M.D., Ph.D., and Stephen H. Loring, M.D.

- Pleural pressure measurements, despite technical limitations, could enable to find a PEEP value that maintain oxygenation while preventing lung injury due to repeated alveolar collapse or overdistention
- Transpulmonary pressure (the difference between the airway pressure and the esophageal pressure) kept between 0 and 10 cm H2O at end expiration.

Intensive Care Med (2011) 37:1595–1604
DOI 10.1007/s00134-011-2333-y
ORIGINAL

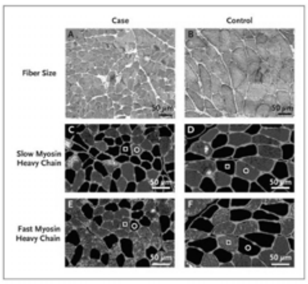
J. Dellamonica
N. Lerolle
C. Sargentini
G. Beduneau
F. Di Marco
A. Mercat
J. C. M. Richard
J. L. Diehl
J. Mancebo
J. J. Rouby
Q. Lu
G. Bernardin
L. Brochard

PEEP-induced changes in lung volume in acute respiratory distress syndrome. Two methods to estimate alveolar recruitment

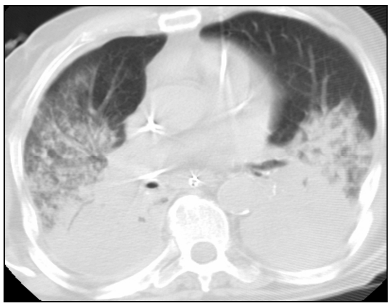


Why is spontaneous breathing desirable?

- Preserve Respiratory Muscle Function
- Improve VA/Q and Regional Ventilation

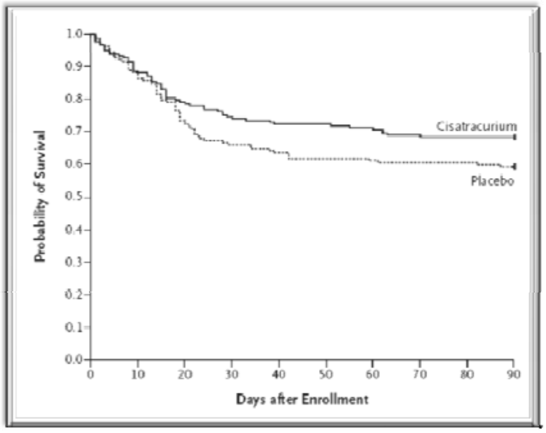


Levine S et al. N Engl J Med 2008;358:1327-1335



Neuromuscular Blockers in Early Acute Respiratory Distress Syndrome

The NEW ENGLAND JOURNAL *of* MEDICINE



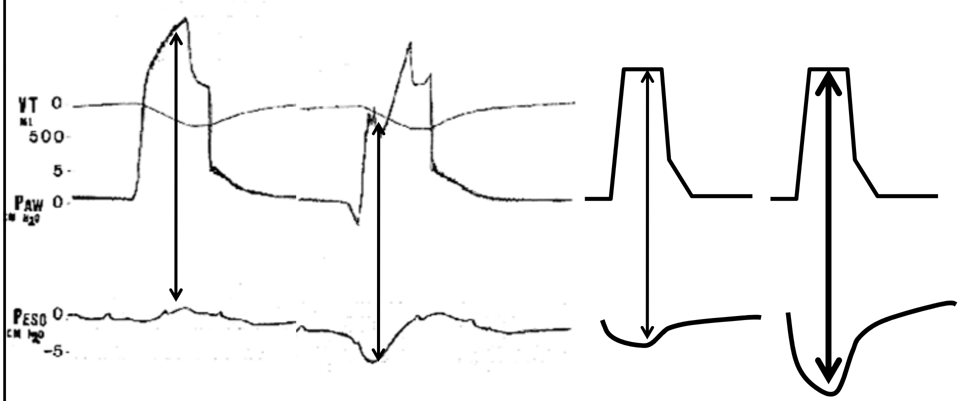
PNO 4% vs 12%

Papazian L. NEJM 2010

Volume vs. Pressure-targeted modes: what's the difference?

Volume-control:
TransPulmonary Pressure is controlled

Pressure-control:
TransPulmonary Pressure is NOT controlled



Intensive Care Med
DOI 10.1007/s00134-009-1632-z

EDITORIAL

Laurent J. Brochard

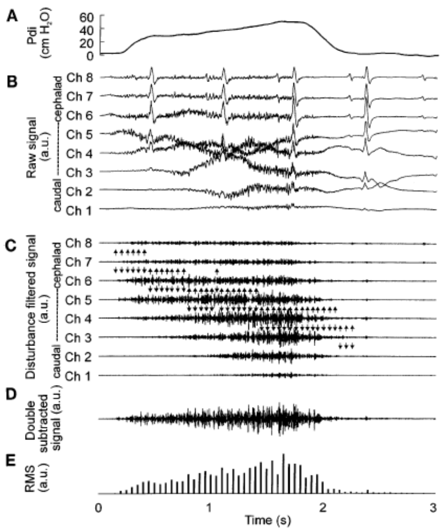
Tidal volume during acute lung injury: let the patient choose?

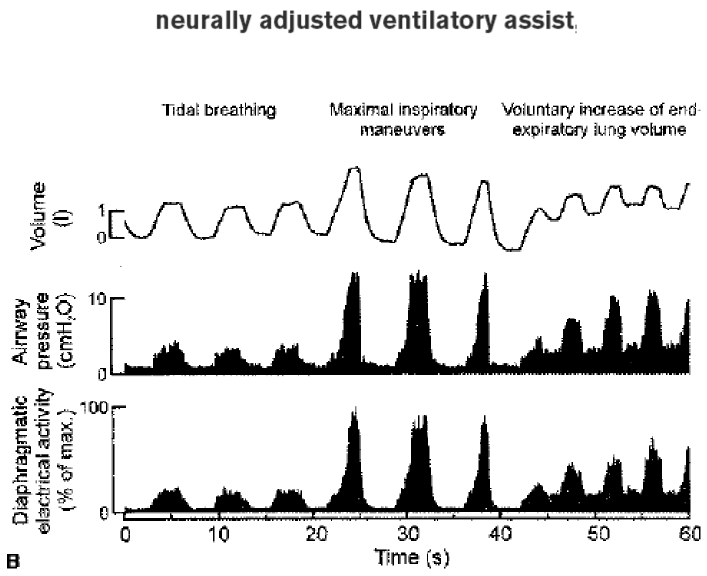
- PAV : Ventilation assistée proportionnelle
- NAVA : Ventilation neuro-assistée

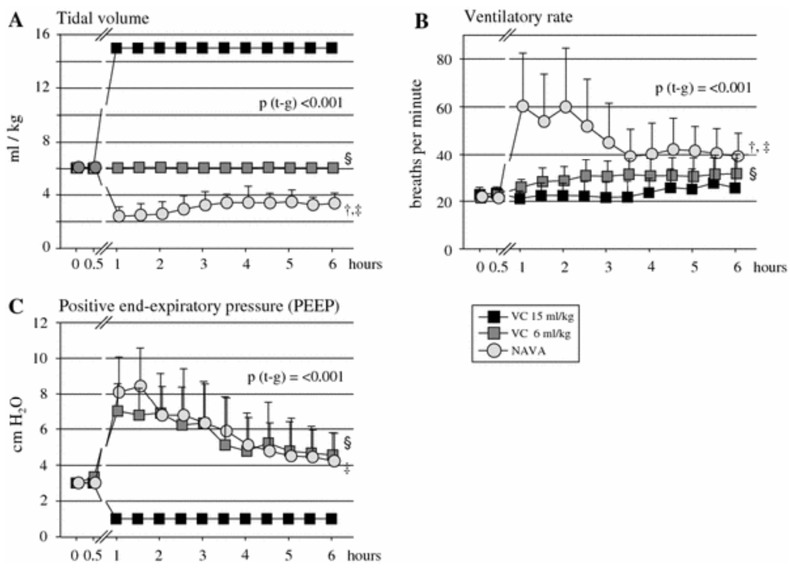
Voluntary activation of the human diaphragm in health and disease

CHRISTER SINDERBY,[1,2] JENNIFER BECK,[3,4] JADRANKA SPAHIJA,[4,5] JAN WEINBERG,[6] AND ALEX GRASSINO[4]

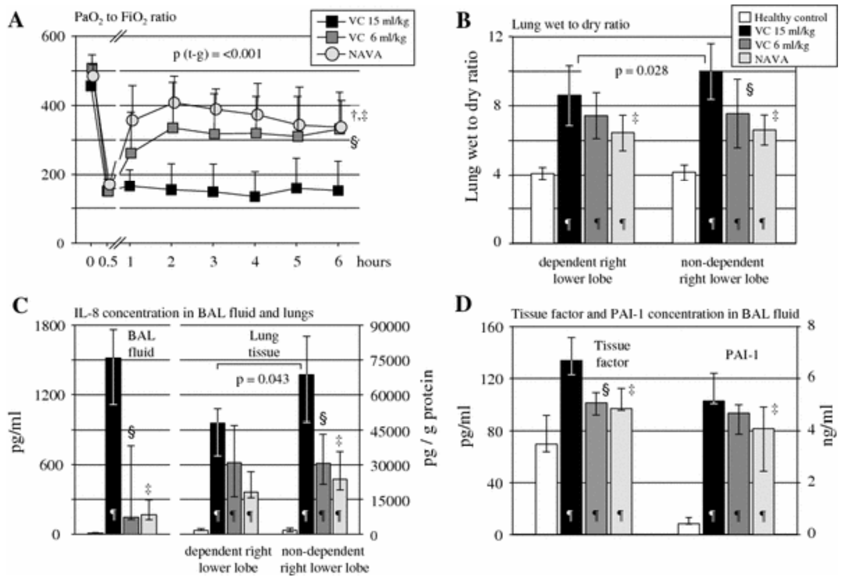
***J. Appl.Physiol.* 1998, 85: 2146–2158,**







Brander L. ICM 2009



Brander L. ICM 2009

Intensive Care Med (1988) 15:8–14

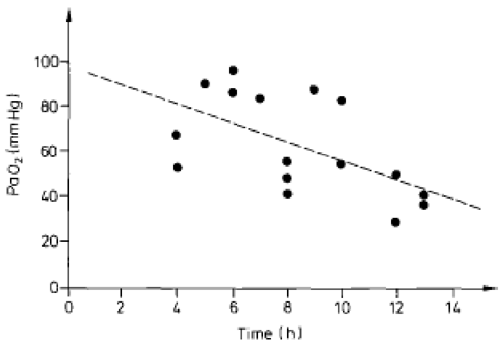
Intensive Care Medicine

Original articles

Acute respiratory failure following pharmacologically induced hyperventilation: an experimental animal study

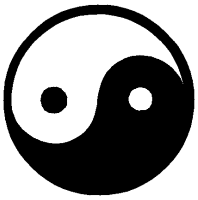
D. Mascheroni *, T. Kolobow, R. Fumagalli *, M. P. Moretti **, V. Chen and D. Buckhold

National Institutes of Health, National Heart, Lung and Blood Institute, Laboratory of Technical Development, Bethesda, Maryland, USA

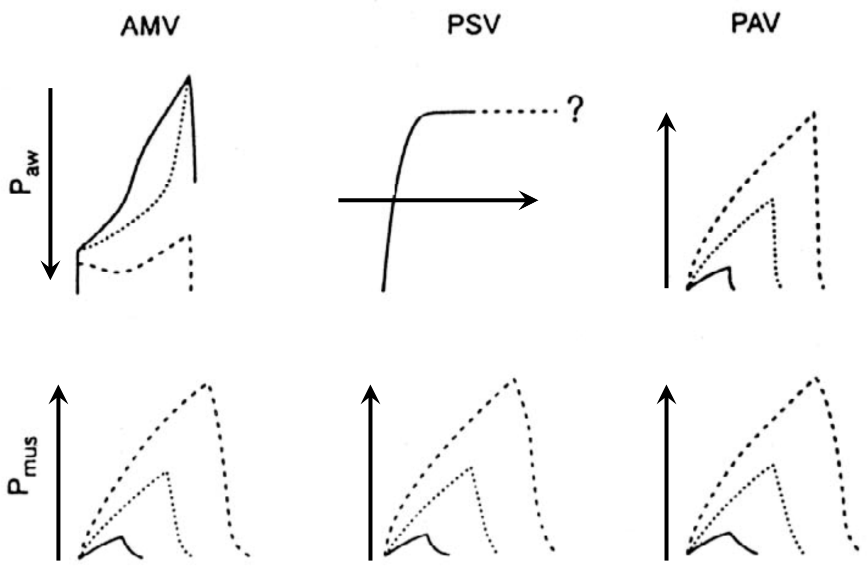


Objectifs de la ventilation mécanique

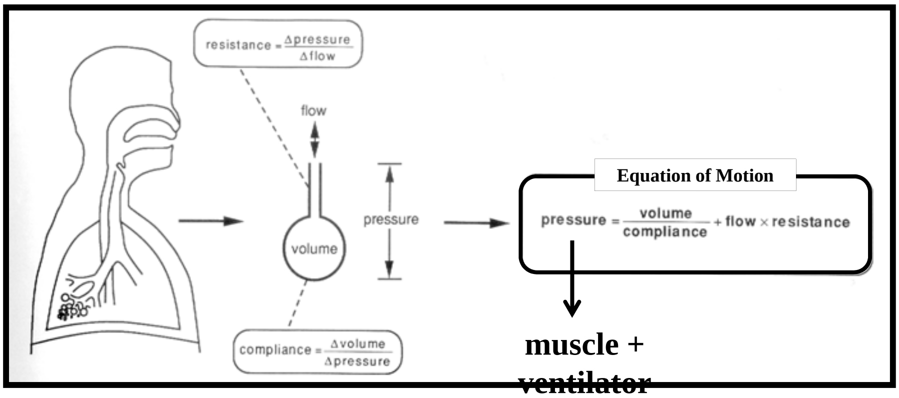
- Schizophrène?
- Non, zen…



Merci



RESPIRATORY SYSTEM MODEL



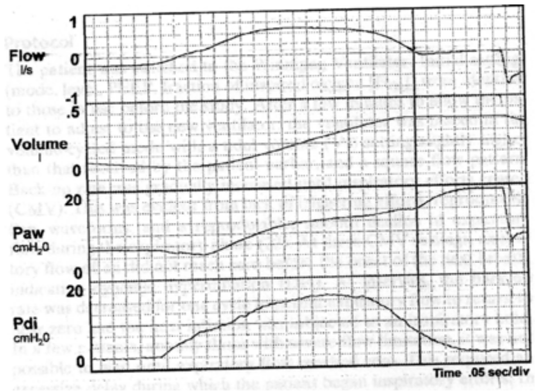
Compliance and Resistance = CONSTANTS

Pressure, Volume, Flow = VARIABLES

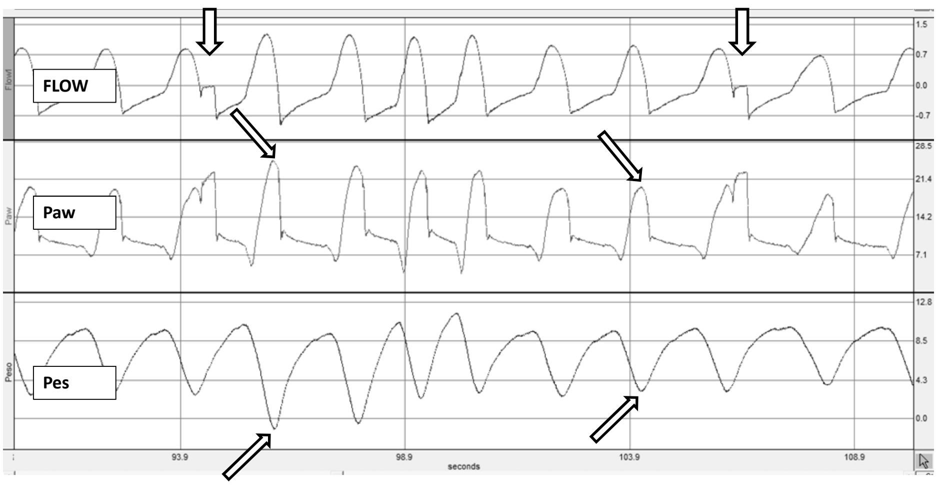
Estimation of Elastance

300 ms end inspiratory pause manoeuvre at a random intervals of 4 to 10 breaths : **Pplat$_{PAV}$**

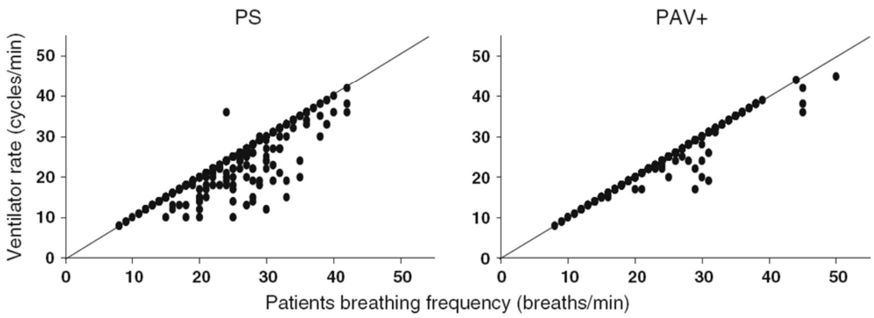
E_{PAV} = ($Pplat_{PAV}$ - $PEEP_{tot}$) / Vt



Younes M, *et al*. AJRCCM 2001;164:50-60



GAIN = 40%



Xirouchaki, Intensive Care Med 2008