Les muscles respiratoires dans le sevrage : Intérêt des nouveaux modes ventilatoires

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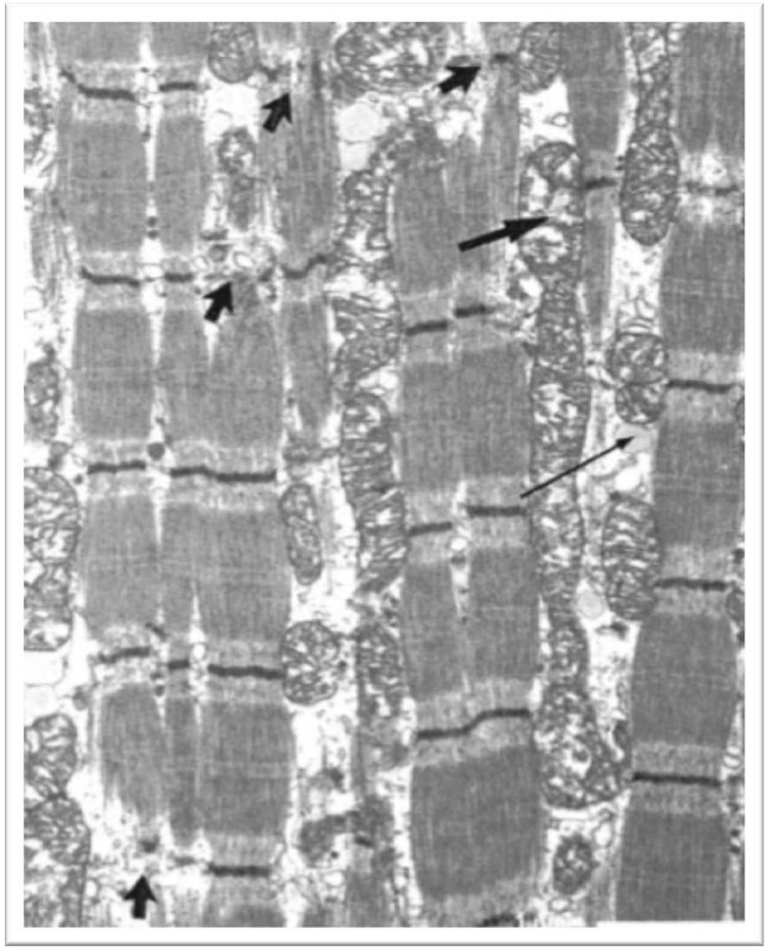
UCL
BRUXELLES

Atteintes neuromusculaires sévères

Des muscles squelettiques

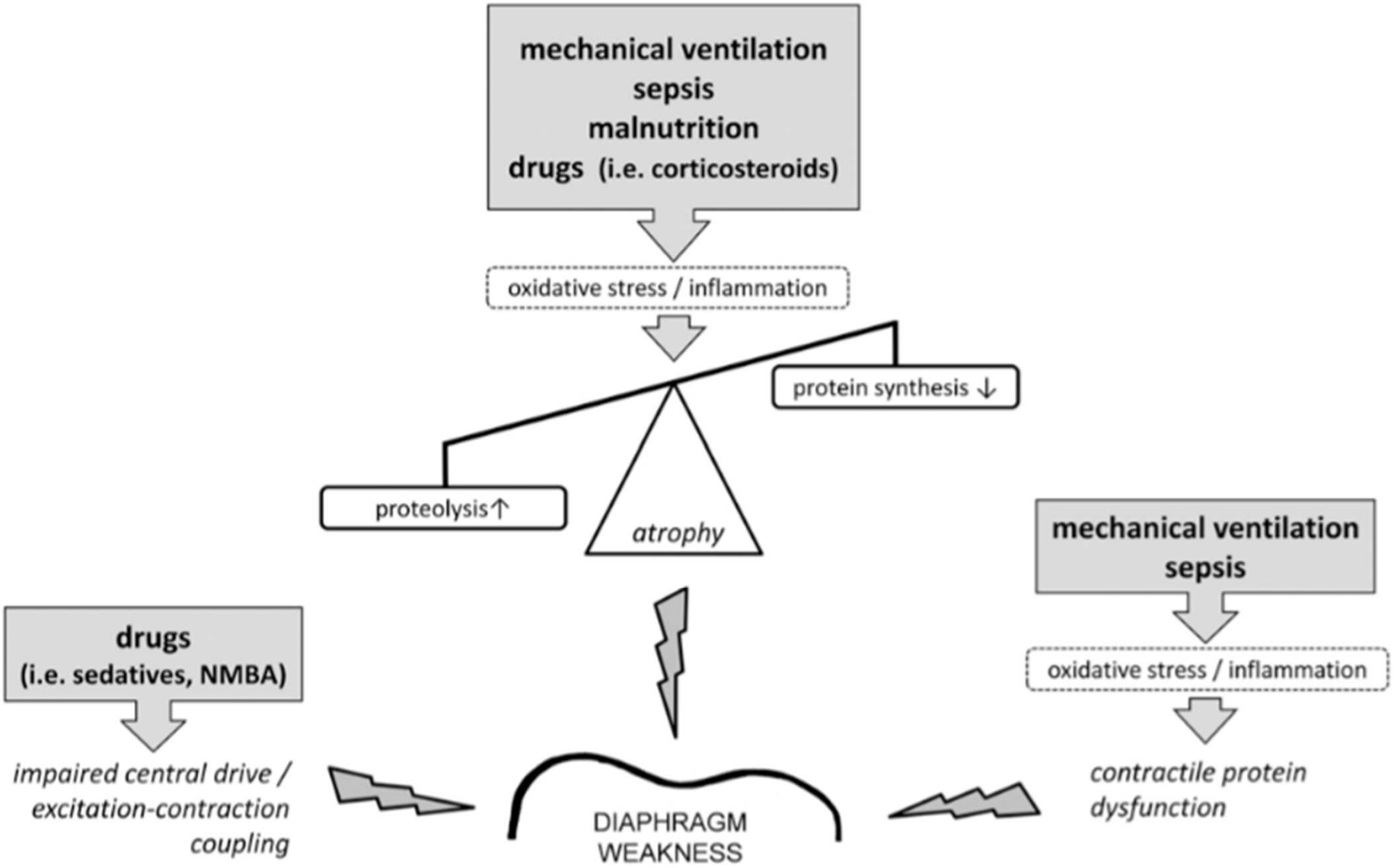
Des muscles respiratoires

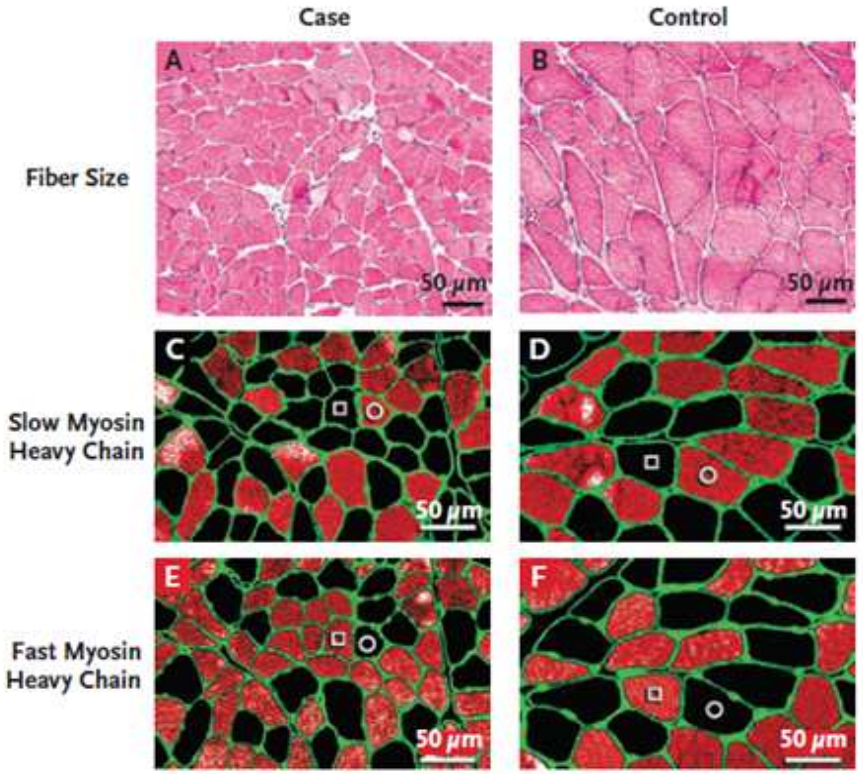
FIGURE 1. Electron micrograph of skeletal muscle biopsy from patient 1 contrasting normal myofibrils *(above)* to severe myopathic fibrils *(below)* (original magnification × 13,750).

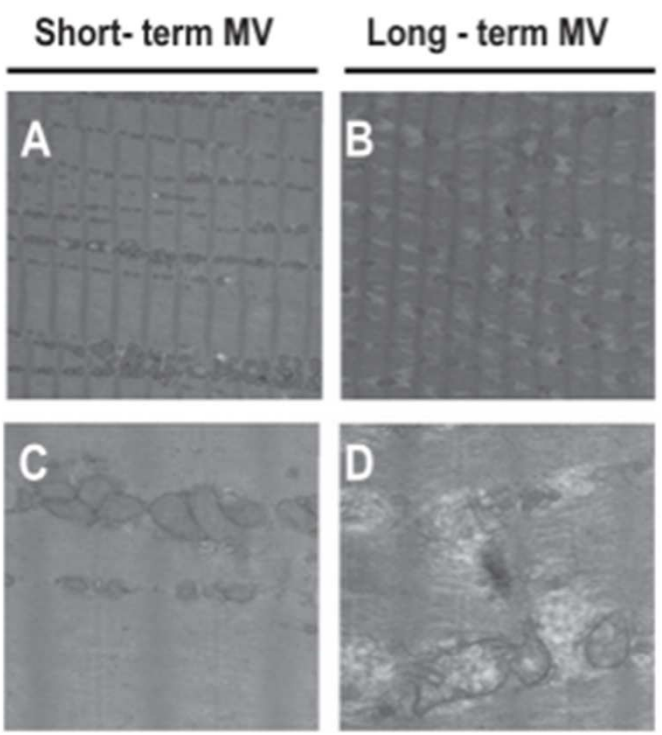


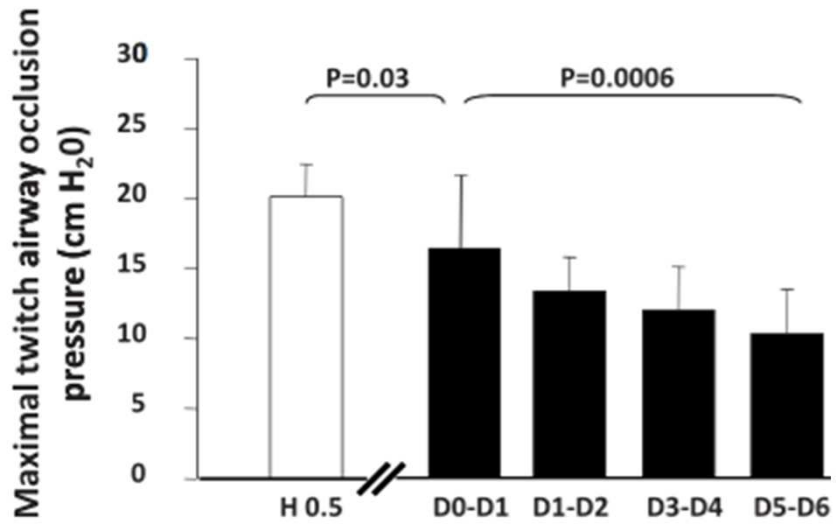
Griffin, D., et al. (1992).CHEST Journal **102**(2): 510-514.

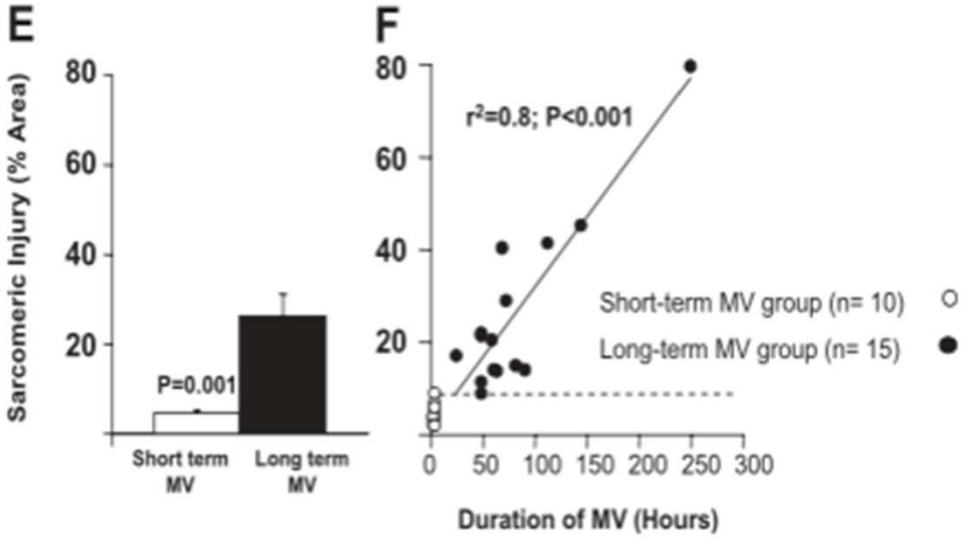
Laghi and Tobin (2003) Am J Respir Crit Care Med ; 168:10-48


mechanical ventilation
sepsis
malnutrition
drugs (i.e. corticosteroids)
oxidative stress / inflammation
protein synthesis ↓
proteolysis↑
atrophy
drugs
(i.e. sedatives, NMBA)
impaired central drive / excitation-contraction coupling
DIAPHRAGM WEAKNESS
mechanical ventilation
sepsis
oxidative stress / inflammation
contractile protein dysfunction









Levine, S., et al. (2008). NEJM **358**(13): 1327-1335.
Jaber et al. Am J Respir Crit Care Med 183:364–371

Sédation ?

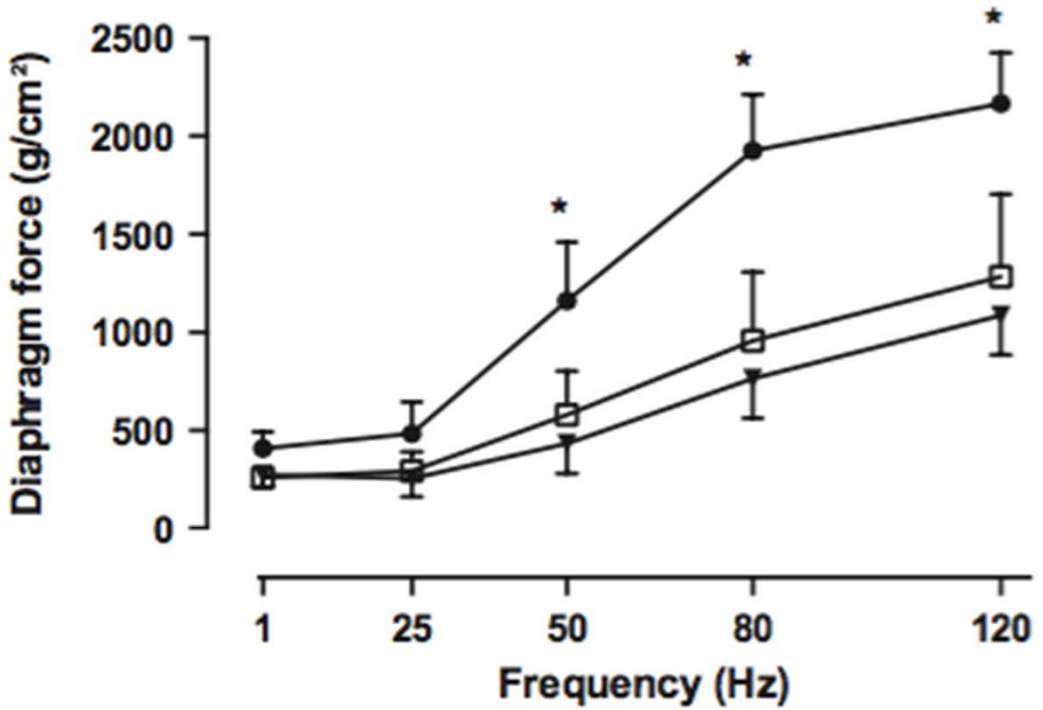
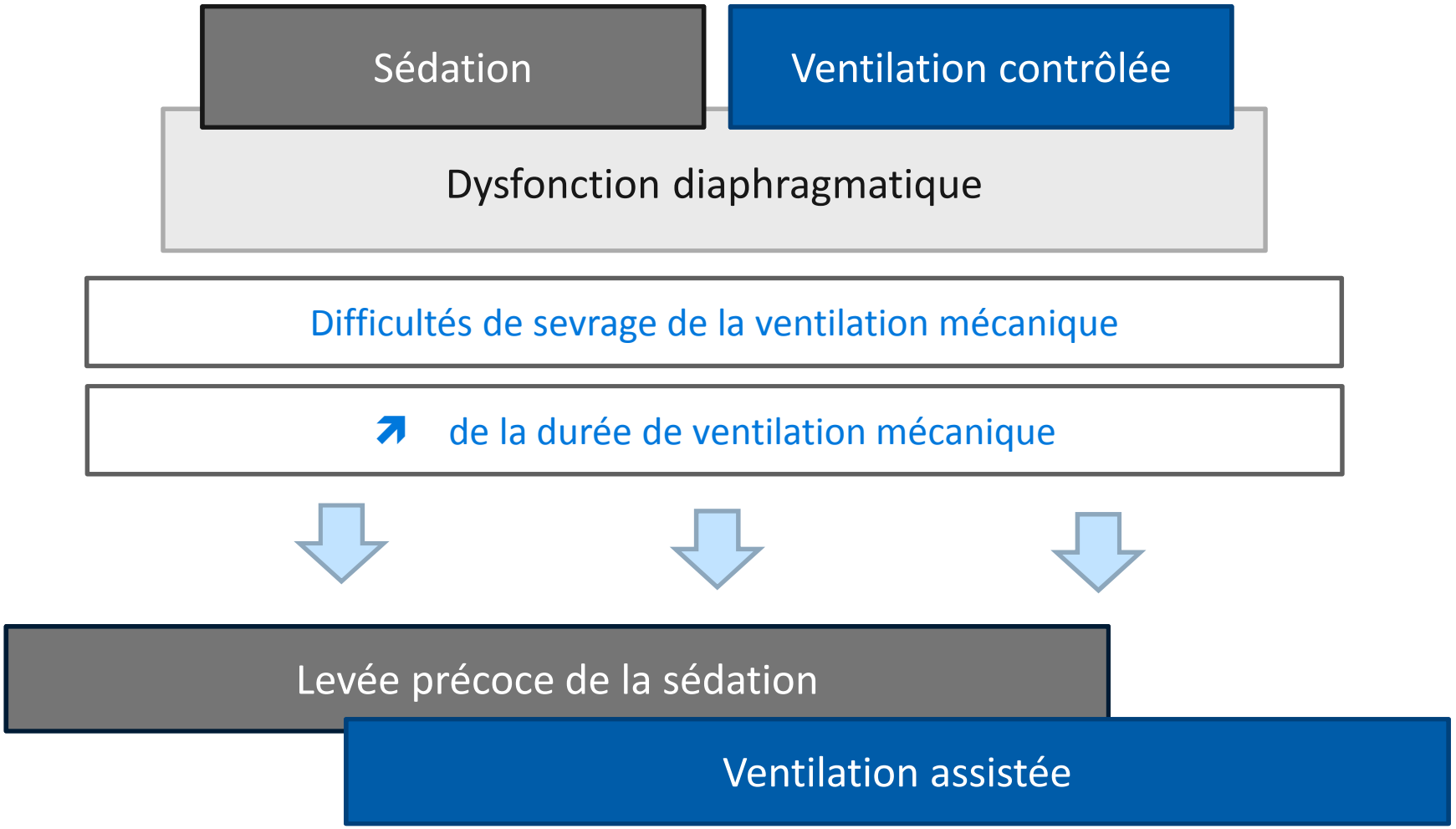
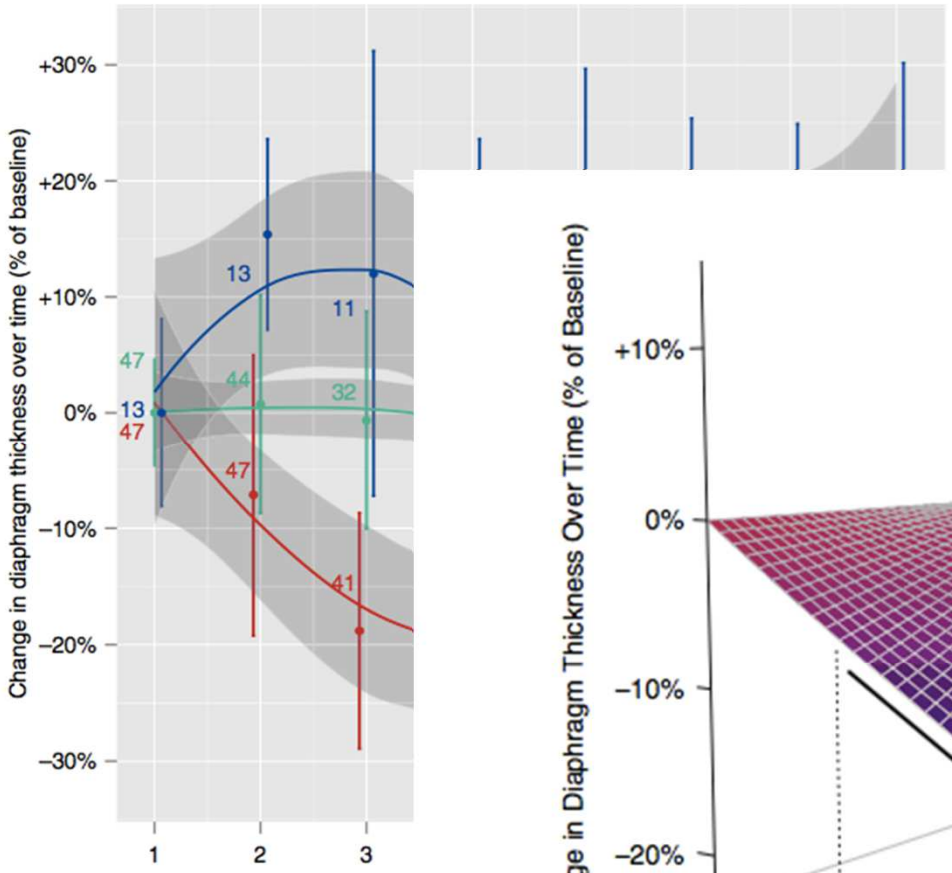
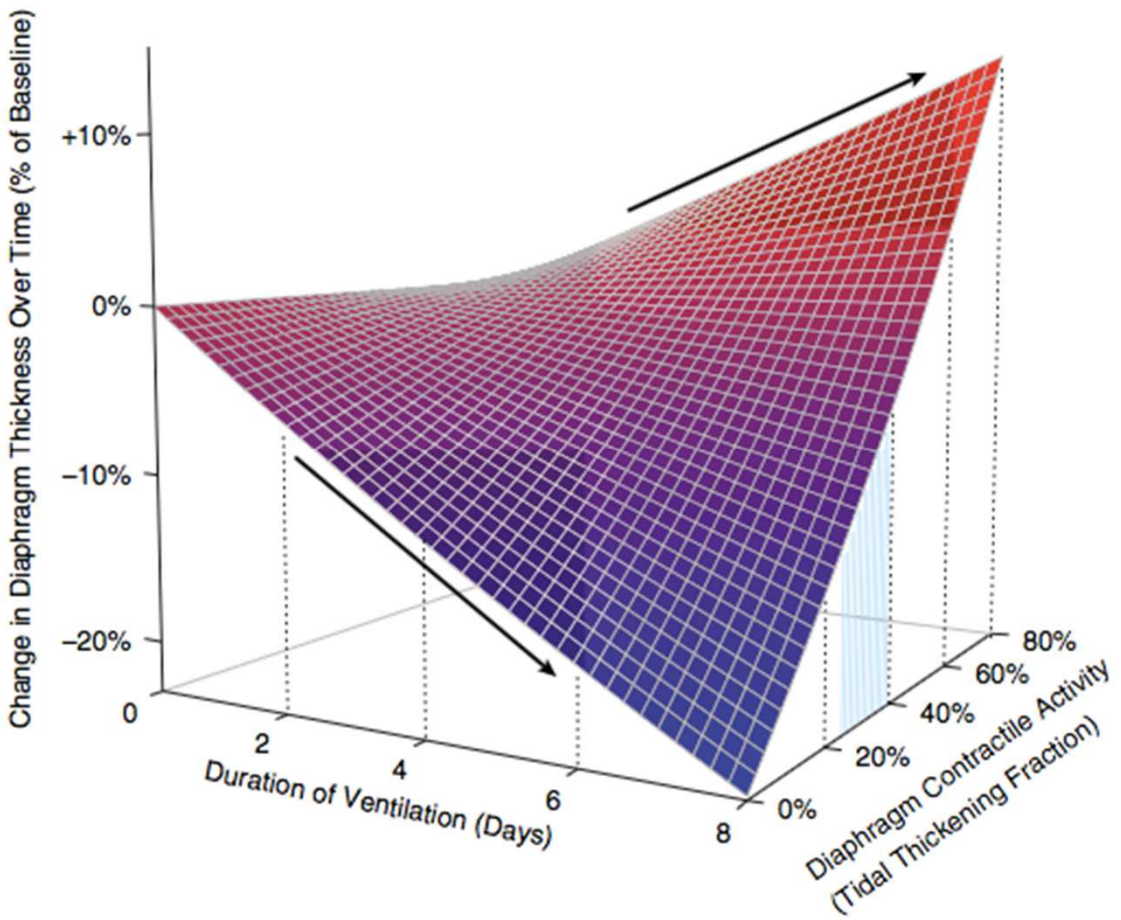


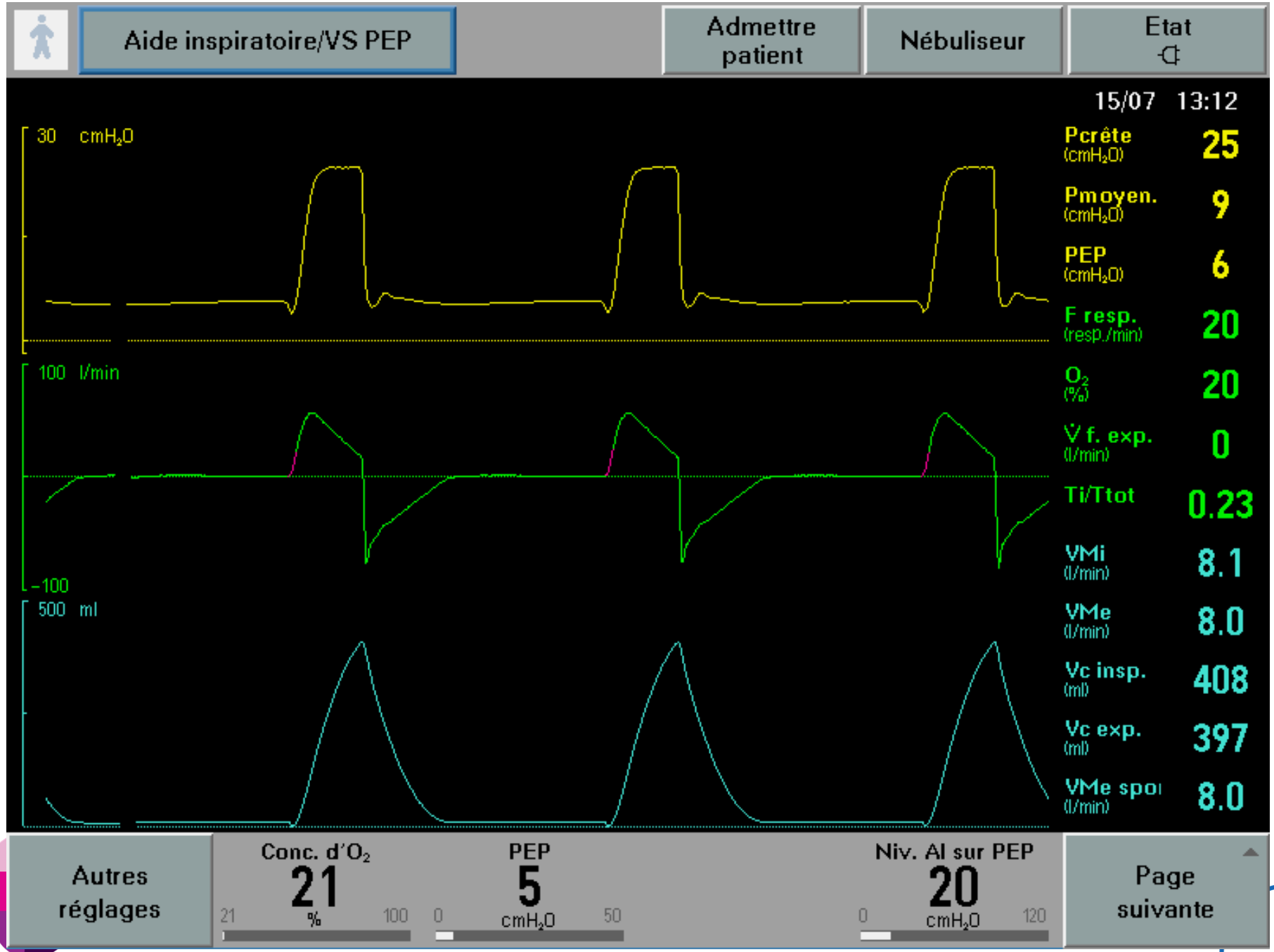
Fig. 1. Diaphragm force frequency curve in the controlled mechanical ventilation and spontaneous breathing sedated with propofol compared with control. Propofol under spontaneous breathing resulted in diaphragmatic dysfunction. *Open squares*: spontaneous breathing (n = 8); *triangles*: controlled mechanical ventilation (n = 7); *filled circles*: control (n = 8); **P* < 0.05 *versus* control.

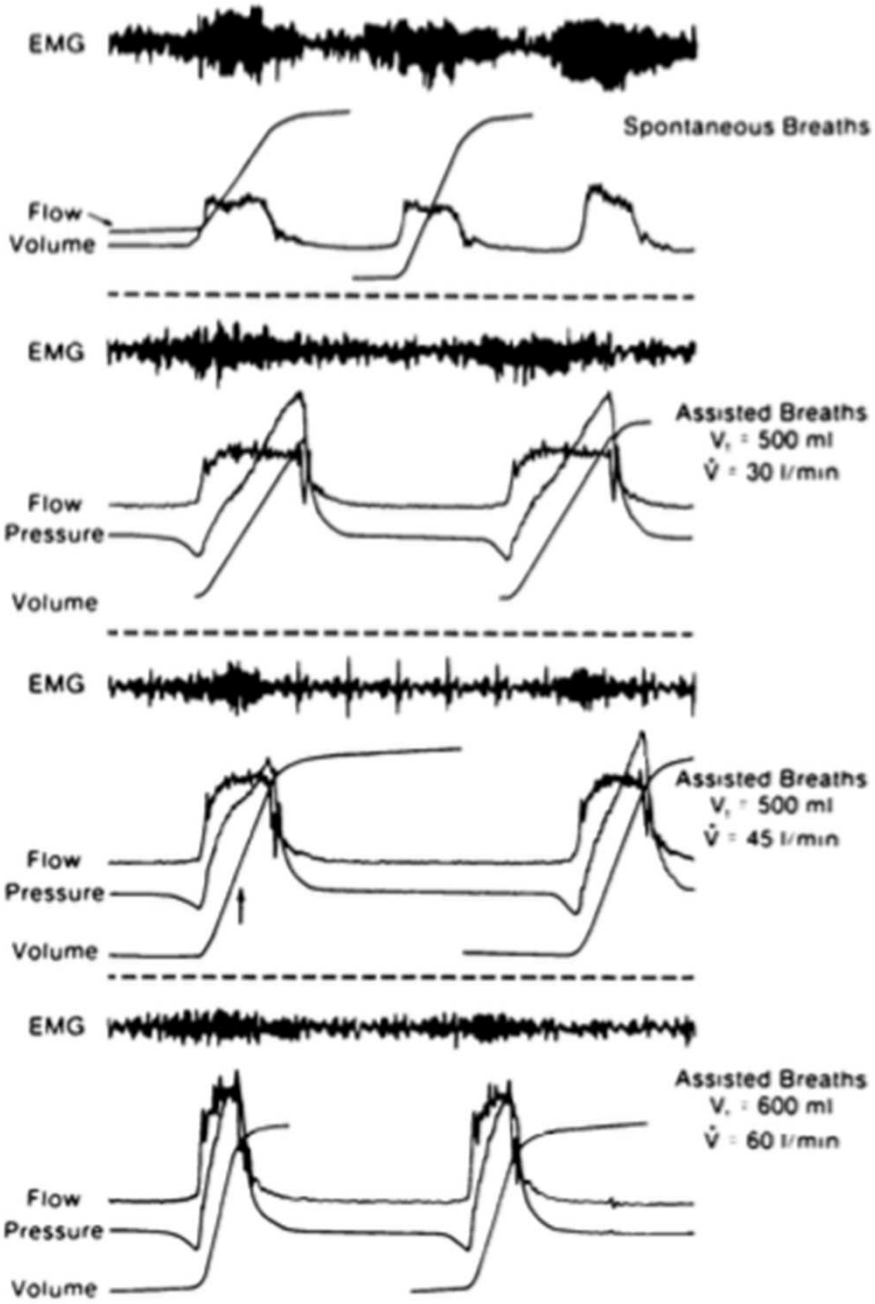

Sédation
Ventilation contrôlée
Dysfonction diaphragmatique
Difficultés de sevrage de la ventilation mécanique
↗ de la durée de ventilation mécanique
Levée précoce de la sédation
Ventilation assistée



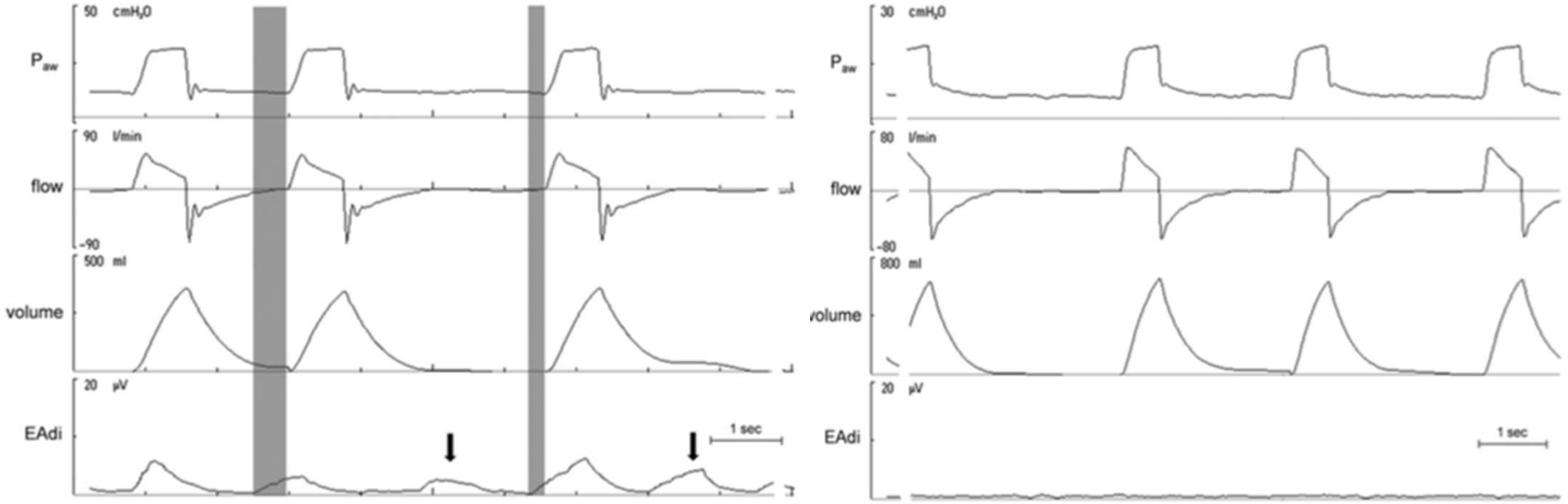


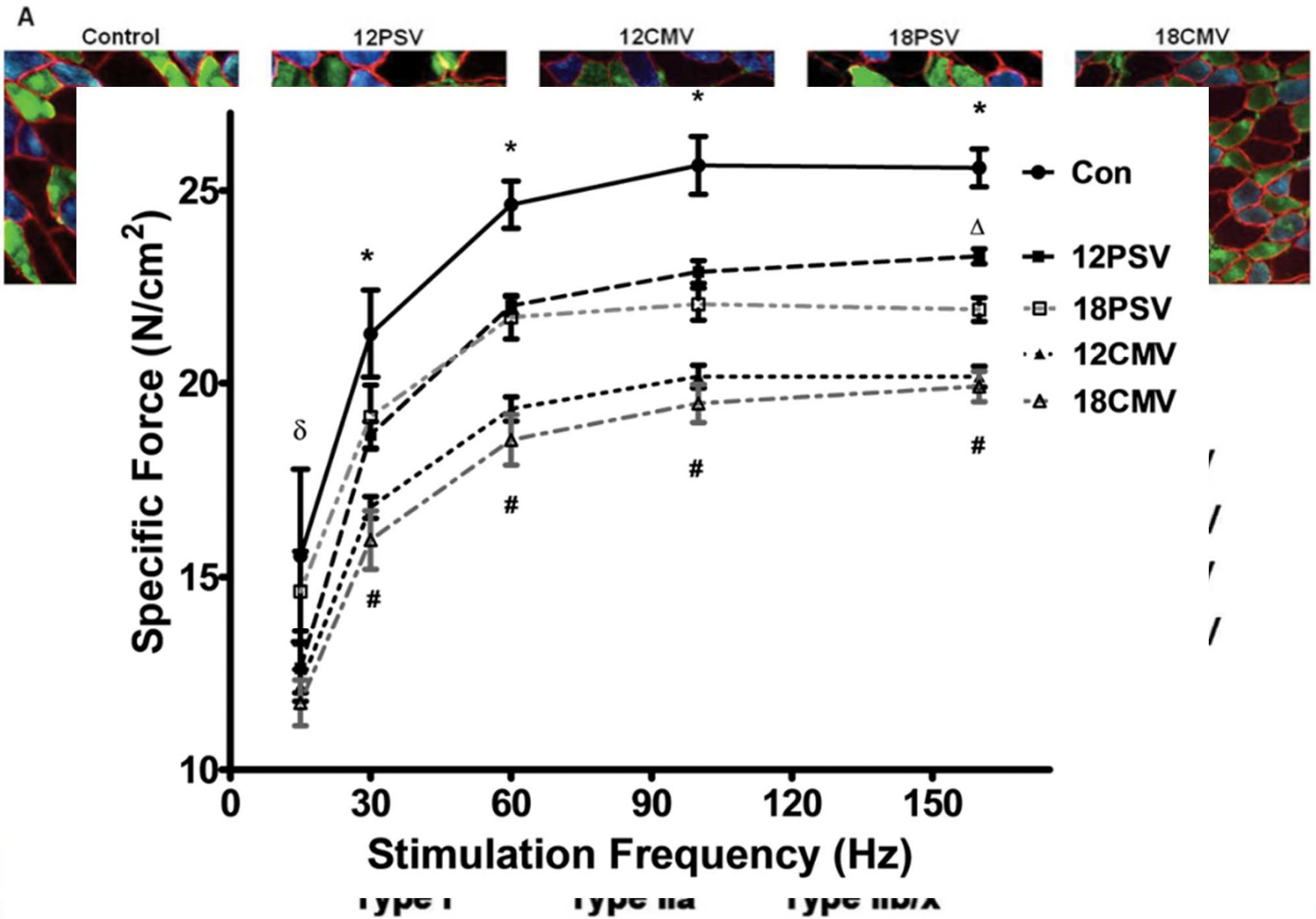
Goligher et al. 2015 AJRCCM. 192:1080-1088


Aide inspiratoire/VS PEP
Admettre patient
Nébuliseur
Etat
15/07 13:12
30 cmH2O
100 l/min
-100
500 ml
Pcrête (cmH2O) 25
Pmoyen. (cmH2O) 9
PEP (cmH2O) 6
F resp. (resp./min) 20
O2 (%) 20
V f. exp. (l/min) 0
Ti/Ttot 0.23
VMi (l/min) 8.1
VMe (l/min) 8.0
Vc insp. (ml) 408
Vc exp. (ml) 397
VMe spol (l/min) 8.0
Autres réglages
Conc. d'O2 21 % 21 100
PEP 5 cmH2O 0 50
Niv. AI sur PEP 20 cmH2O 0 120
Page suivante



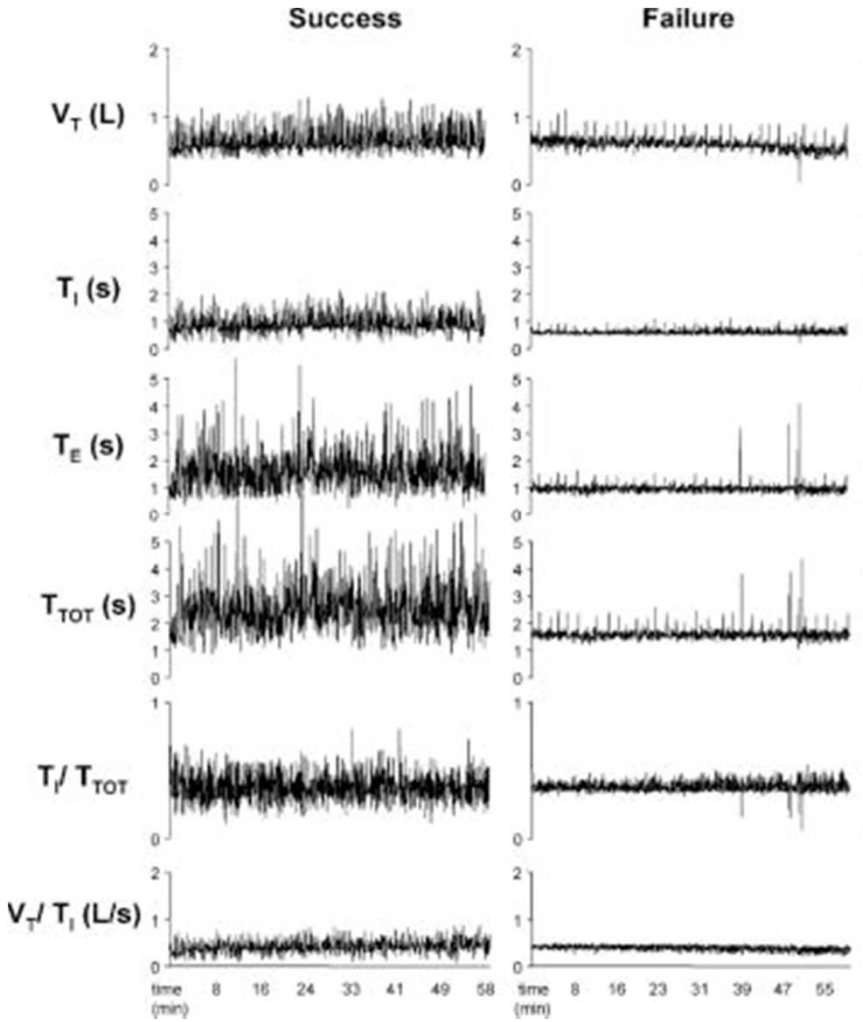
Flick et al. **1989**
Chest 96:130-135


50 cmH2O
Paw
90 l/min
flow
-90
500 ml
volume
20 µV
EAdi
1 sec
30 cmH2O
Paw
80 l/min
flow
-80
800 ml
volume
20 µV
EAdi
1 sec

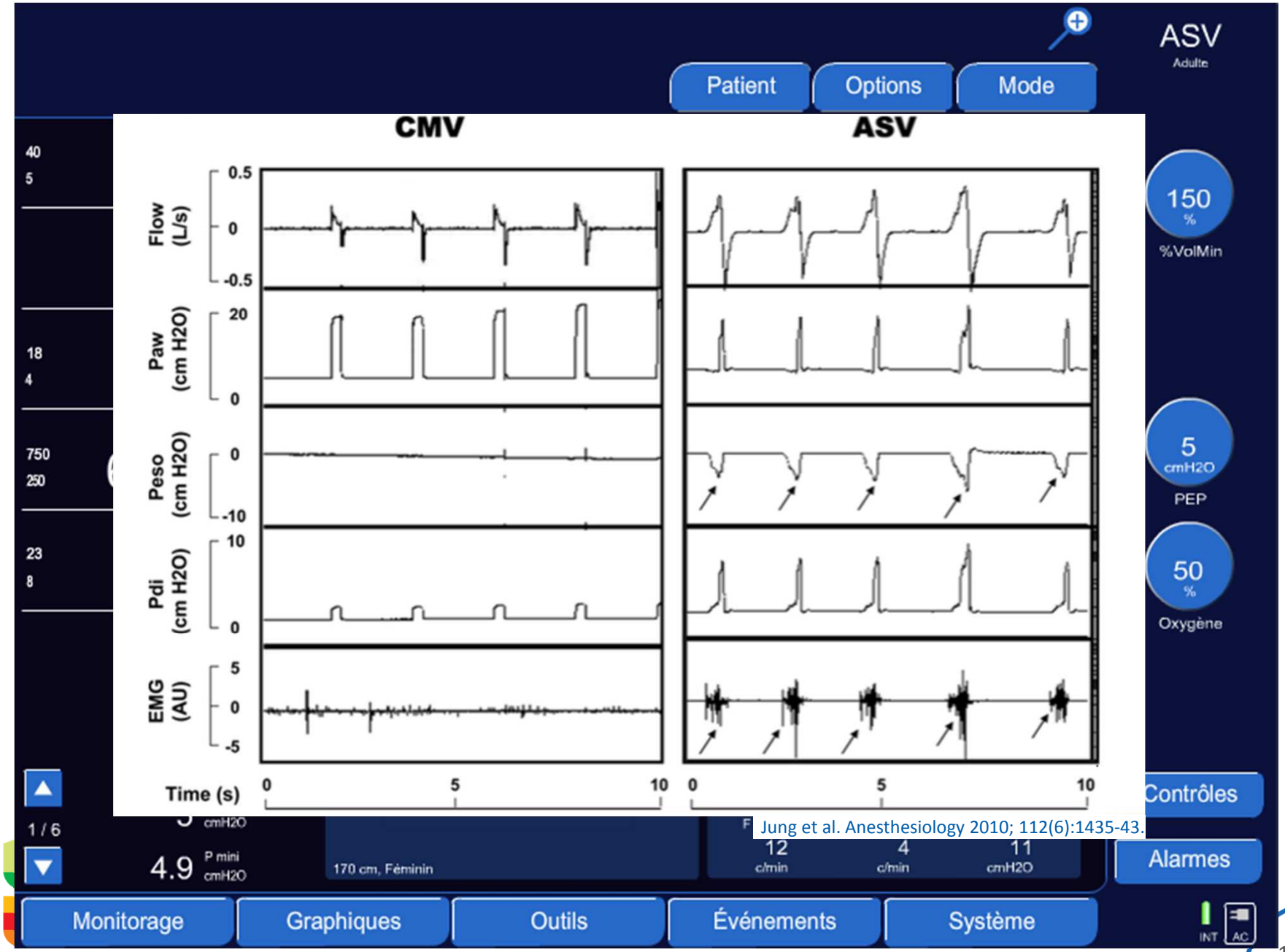

A
Control
12PSV
12CMV
18PSV
18CMV
Specific Force (N/cm²)
25
20
15
10
0
30
60
90
120
150
Stimulation Frequency (Hz)
Con
12PSV
18PSV
12CMV
18CMV
δ
*
Δ
#

Reduced breathing variability as a predictor of unsuccessful patient separation from mechanical ventilation*

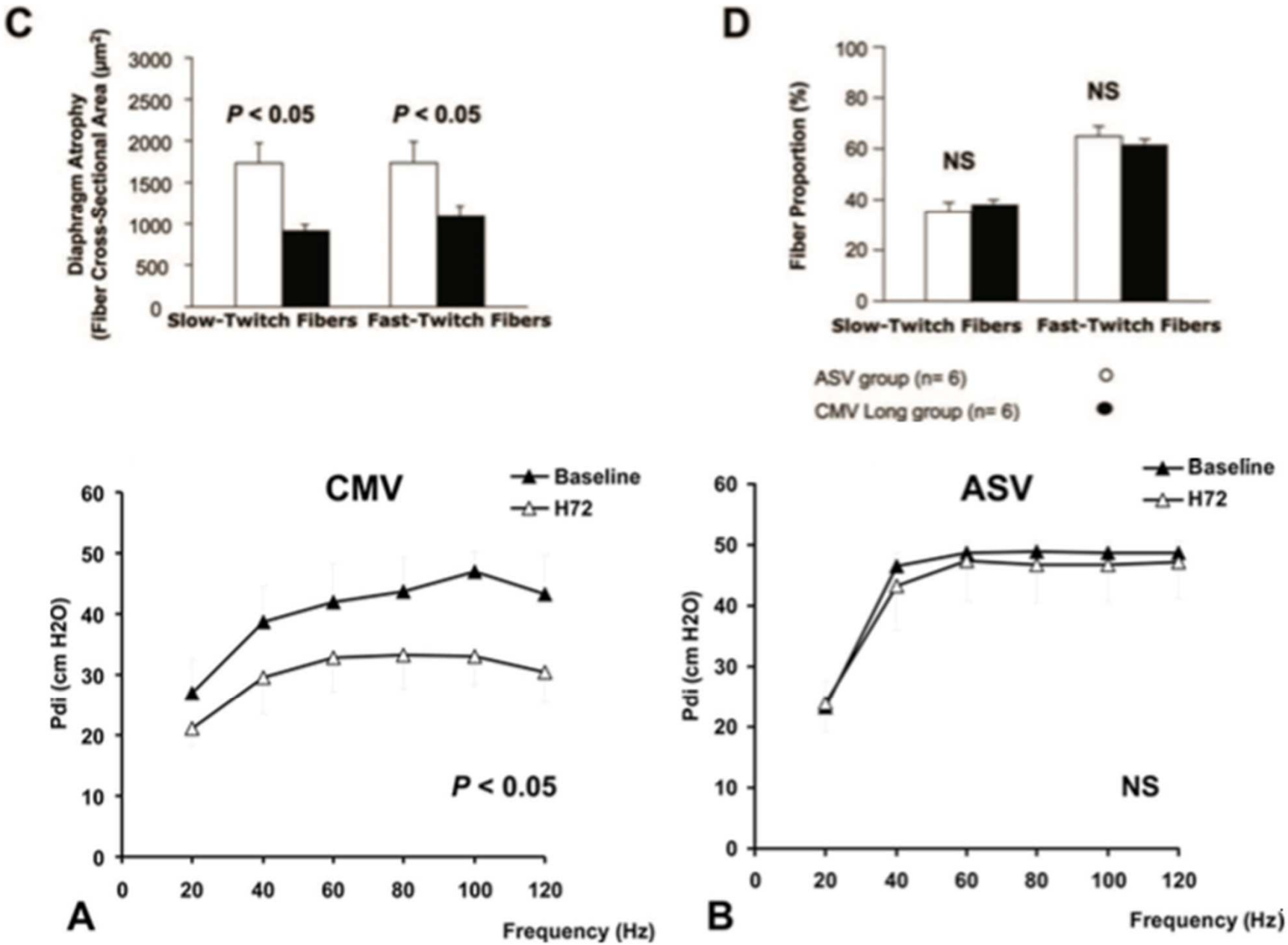
Marc Wysocki, MD; Christophe Cracco, MD; Antonio Teixeira, MD, MSci Biostat; Alain Mercat, MD; Jean-Luc Diehl, MD; Yannick Lefort, MD; Jean-Philippe Derenne, MD; Thomas Similowski, MD, PhD



Wysocki M. et al Crit Care Med 2006.



Jung et al. Anesthesiology 2010; 112(6):1435-43.



Jung et al. Anesthesiology 2010; 112(6):1435-43.

P PEAK 22 cmH2O
f TOT 28 1/min
PEEP 4.1 cmH2O
I:E 1:1.3
f TOT 28 1/min
V TE 504 mL
V E TOT 14.1 L/min
O2 35 %
C PAV 37 mL/cmH2O
R PAV 8.4 cmH2O/L/s
PEEP I 0.9 cmH2O
P CIRC
cmH2O
P LUNG
cmH2O
Menu
WOB
J/L
E
R
WOB PT
WOB TOT
V T
mL
Adult
SPONT
PAV+
80kg
5.63mL/kg
Manual Insp
V T 450 mL
% Supp 70 %
V SENS 3.0 L/min
O2 35 %
E SENS 3 L/min
PEEP 5.0 cmH2O
Manual Event
O2 100%
03:20:00pm

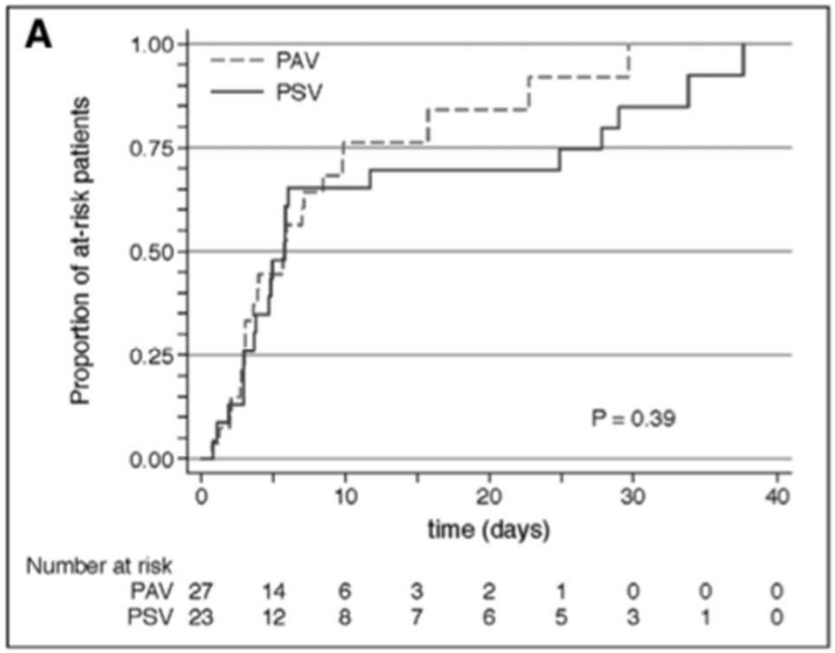
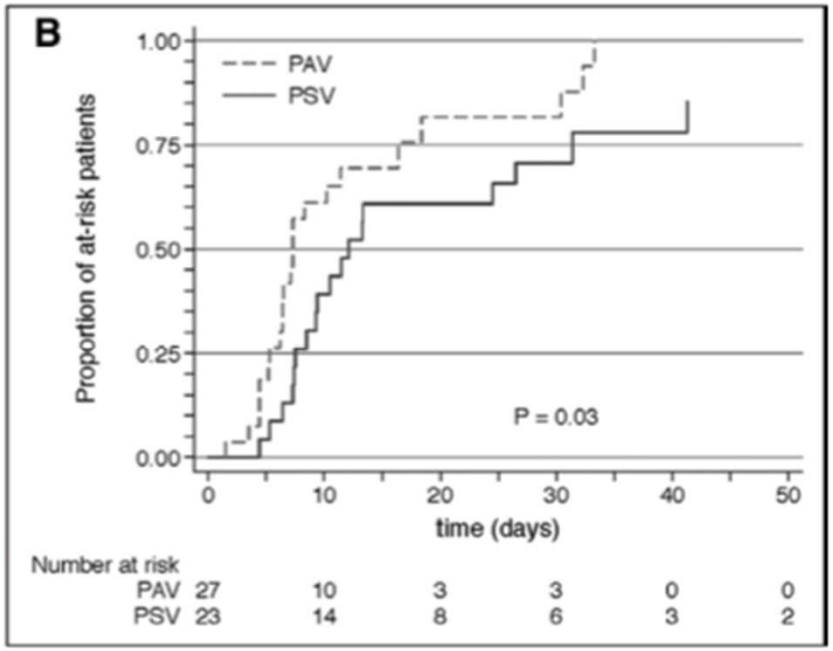


Figure 3. A, Time to successful extubation for the two treatment groups (Kap



plan-Meier curves). **B**, Time to live ICU discharge for the two treatment

CONCLUSIONS

In conclusion, this pilot study demonstrates that protocols using stepwise reductions in PAV or PSV to maintain patients at the lowest level of support without fatigue, coupled with daily assessment for SBTs, may be used safely and effectively in the population at risk for difficult or prolonged weaning from early in their recovery until ventilation discontinuation. Although patients may be supported with these protocols throughout their recovery period, approximately 75% of patients required intermittent use of A/C mode for respiratory distress not alleviated at maximal support, and used A/C mode intermittently on average 6 hours/d. As a physiologic study, this trial included a sufficiently large sample size to show that PAV+ may facilitate weaning and aid in recovery of the respiratory muscles, by imparting a greater share of the respiratory workload to the diaphragm, without inducing clinical signs of respiratory distress, and by reducing patient-ventilator asynchrony, even at high levels of support. Whether or not these attributes of PAV will translate into improved clinical outcomes merits further study, but we now have sufficient clinical evidence and physiologic rationale to proceed to a rigorous, multicenter RCT designed to answer these specific questions. This report not only demonstrates that such a trial is feasible but also informs study design by providing valuable insight into the utility, safety, and constraints of PAV+ and PSV protocols applied throughout the trajectory of recovery and weaning.

Bosma et al. Crit Care Med. 2016 Jun;44(6):1098-108

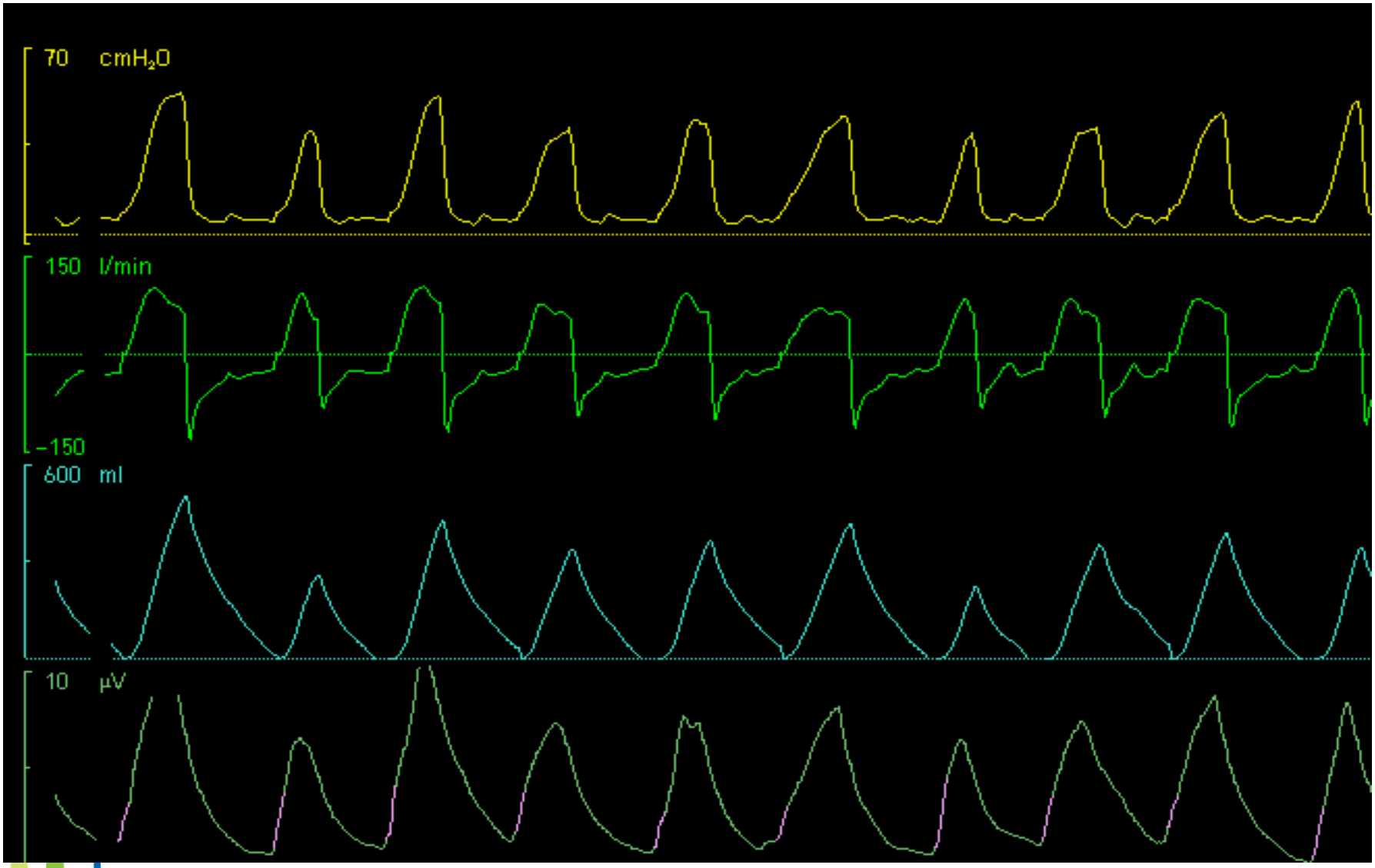
Crossover study of proportional assist versus assist control ventilation

Table 1 Comparison of airway pressures, physiological outcomes and blood gas exchange by ventilator mode

	PAV	ACV	p Value
Mean airway pressure (cm H_2O)	8.4 (7.1–9.0)	9.1 (7.1–11.0)	0.028
Peak inflation pressure (cm H_2O)	17 (13–20)	18 (14–20)	0.036
PTPdi (cm H_2O.s/min)	**217 (60–556)**	**309 (55–544)**	**0.005**
Pimax (cm H_2O)	21.9 (11.2–37.3)	23.6 (13.6–38.6)	0.182
Pdimax (cm H_2O)	**44.3 (21.3–66.4)**	**37.9 (19.5–45.2)**	**0.002**
TAA (degrees)	1.2 (0.4–3.5)	1.9 (1.2–3.3)	0.050
Respiratory rate (bpm)	54 (40–66)	57 (46–74)	0.025
Expiratory tidal volume (mL/kg)	6.8 (3.7–7.4)	7.2 (2.7–7.8)	0.182
SaO_2 (%)	96 (93–98)	93 (92–98)	0.021
FiO_2	0.40 (0.21–0.50)	0.50 (0.30–0.60)	0.005
PaO_2 (kpa)	7.2 (4.4–13.2)	6.1 (3.9–11.7)	0.158
Oxygenation index	5.6 (5.0–10.7)	10.1 (7–16.1)	0.002
$PaCO_2$ (kpa)	7.6 (5.7–10.0)	7.6 (5.7–12.1)	0.814
Desaturation episodes (n)	1 (0–3)	1 (0–5)	0.200

The results are expressed as the median (range).
ACV, assist control ventilation; PAV, proportional assist ventilation; TAA, thoracoabdominal asynchrony.

Bhat P, et al. Arch Dis Child Fetal Neonatal Ed 2014;0:F1–F4.


70 cmH2O
150 l/min
-150
600 ml
10 µV

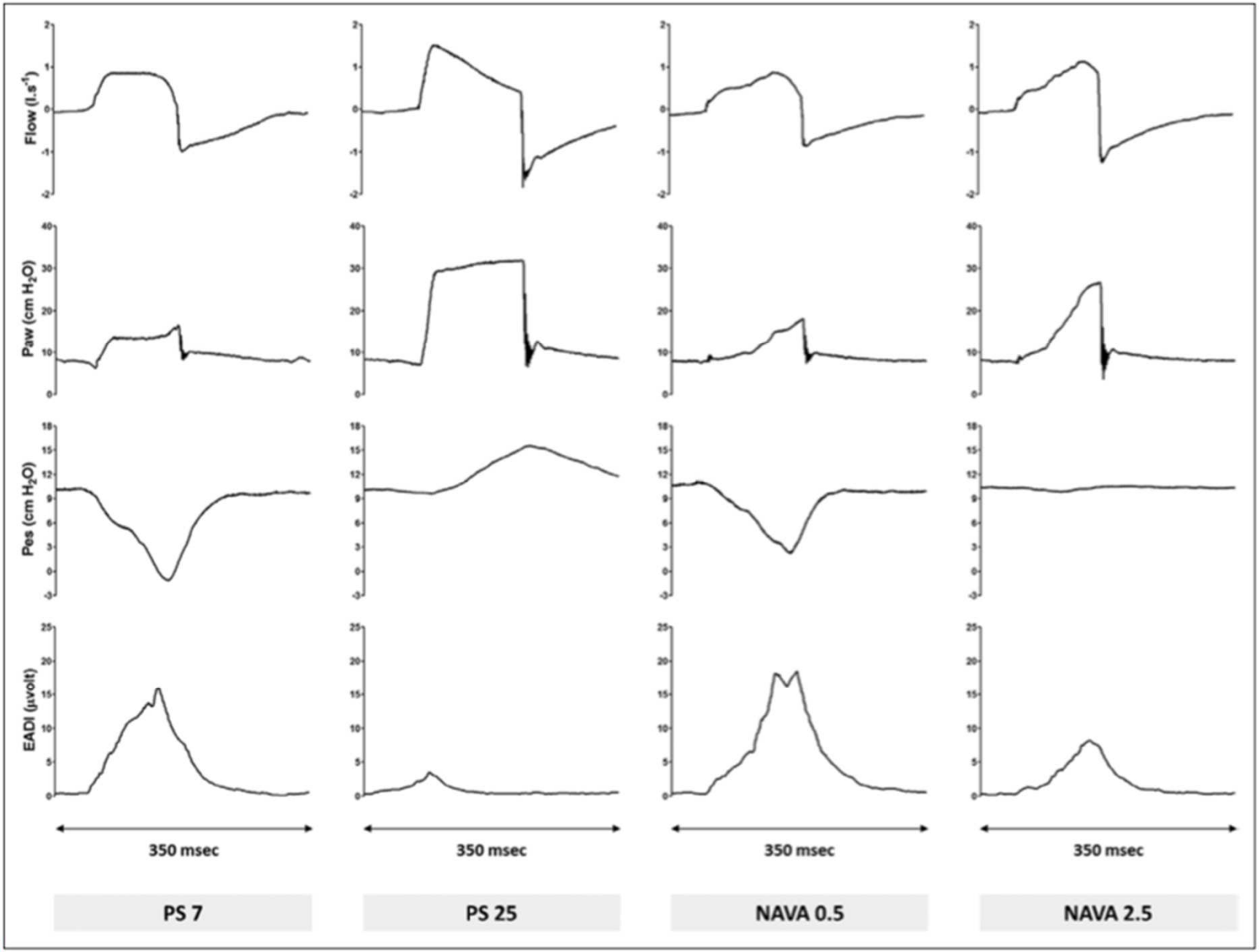


Figure 1. Representative recordings of flow, airway pressure (Paw), esophageal pressure (Pes), and electrical activity of the diaphragm (EAdi) over one breath at pressure support level 7 cm H_2O (PS 7) and 25 cm H_2O (PS 25) and neurally adjusted ventilatory assist level 0.5 cm H_2O/µvolt (NAVA 0.5) and 2.5 cm H_2O/µvolt (NAVA 2.5). At high levels of assistance, note that contrary to NAVA most of the insufflation occurred passively during PS ventilation.

Carteaux et al. *Crit Care Med* 2016; 44:503–511

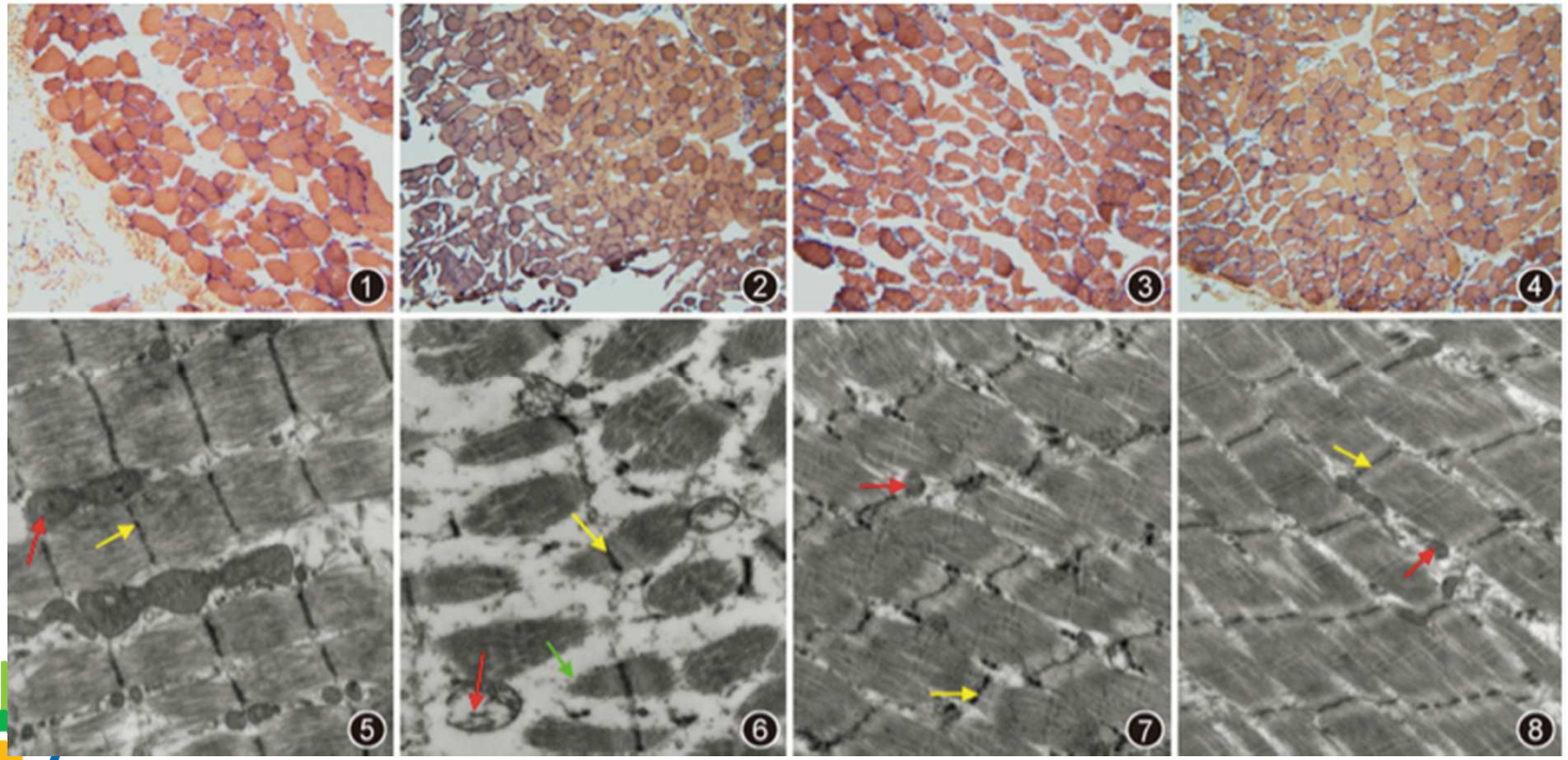
·论著·

Zhonghua Jie He He Hu Xi Za Zhi. 2011 Apr;34(4):288-93.

[Effects of neurally adjusted ventilatory assist on prevention of ventilator-induced diaphragmatic dysfunction in acute respiratory distress syndrome rabbits].

[Article in Chinese]

Huang DY[1], Liu J, Wu XY, Liu HG, Wu AP, Jiang DW, Yang Y, Qiu HB.



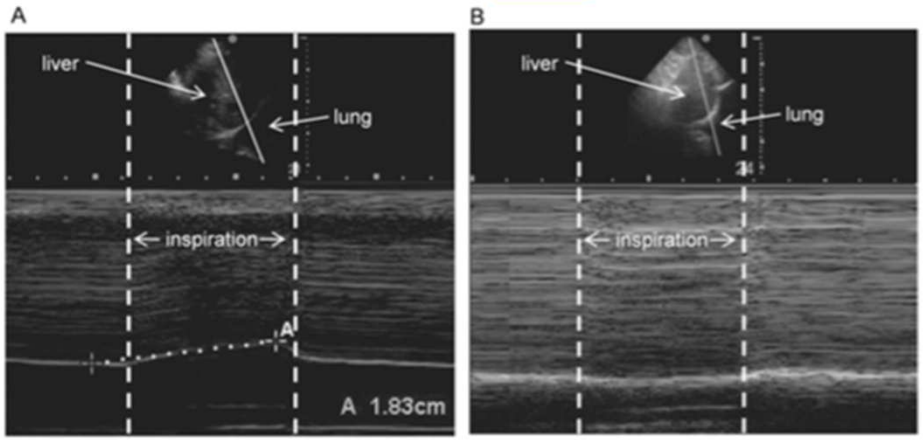


Figure 3. Ultrasonographic M-mode images from the right hemidiaphragm. (*A*) Normal diaphragm motion: the diaphragm moves 1.8 cm cephalad during inspiration. (*B*) Caudal movement of the diaphragm in a patient with diaphragm paralysis.

Ventilation mécanique altère le diaphragme ➔
Ventilation contrôlée et ventilation assistée !

Respecter le travail du diaphragme.

Plus amples investigations quant à l'impact des modes non-conventionnels.

Limiter les autres causes de dysfonctions diaphragmatiques.

Merci de votre attention !