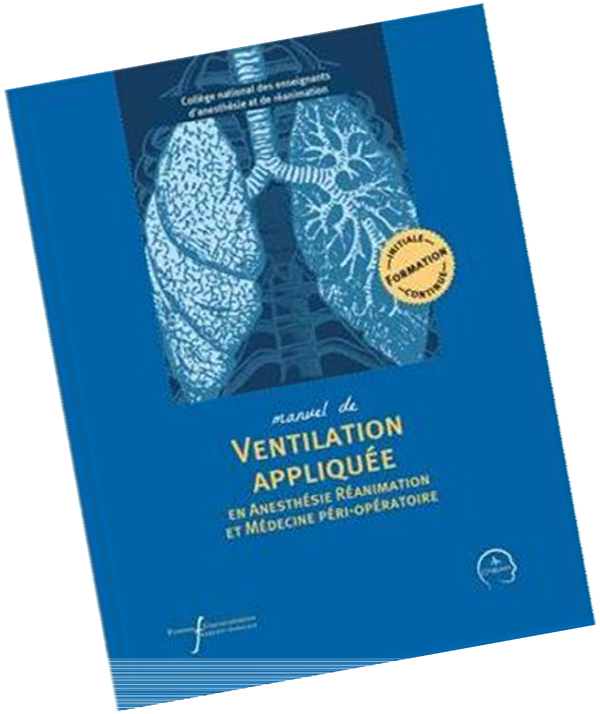
DU Kiné et IDE 2024

Physiopathologie respiratoire
Ventilation artificielle

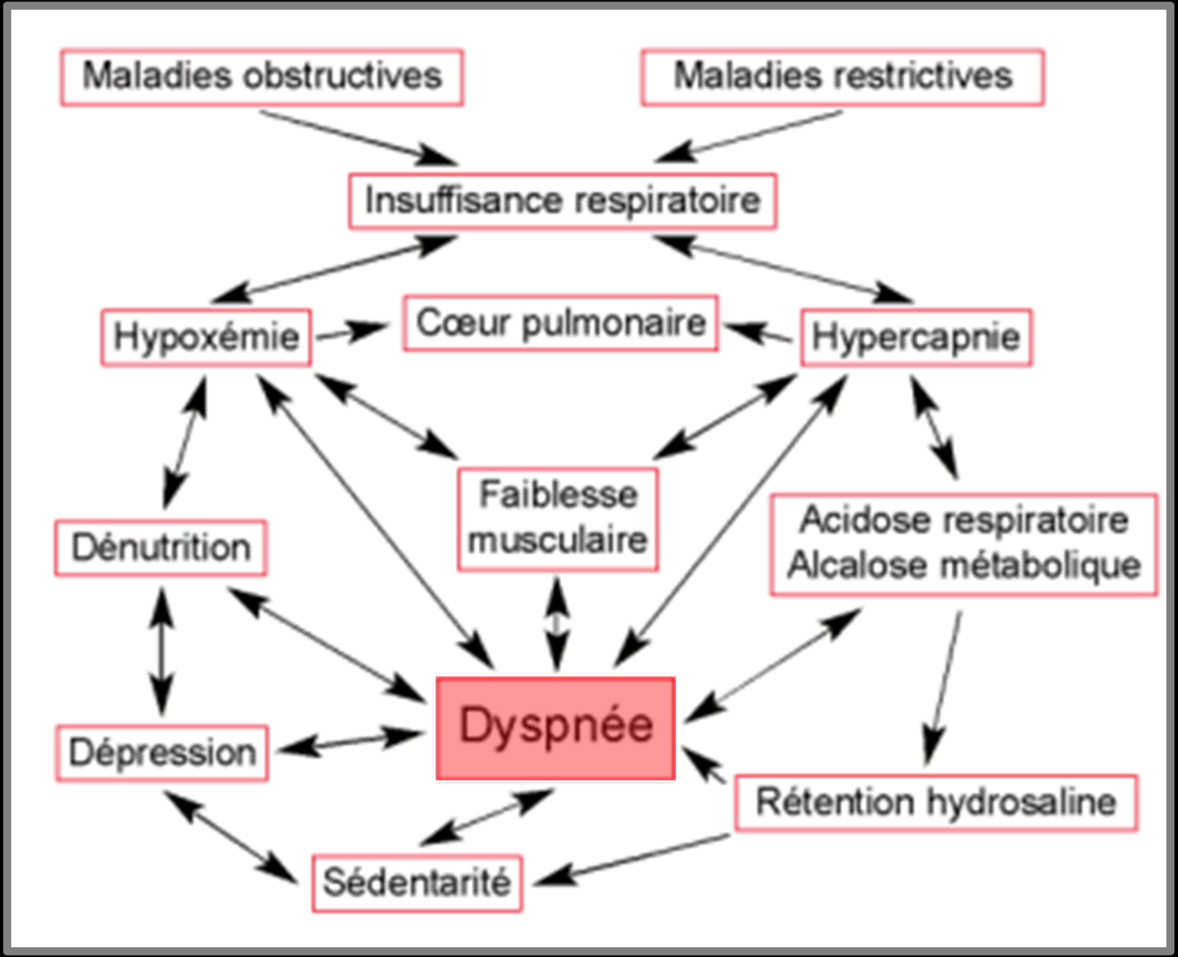
Pr Hadrien Rozé

Référentiels

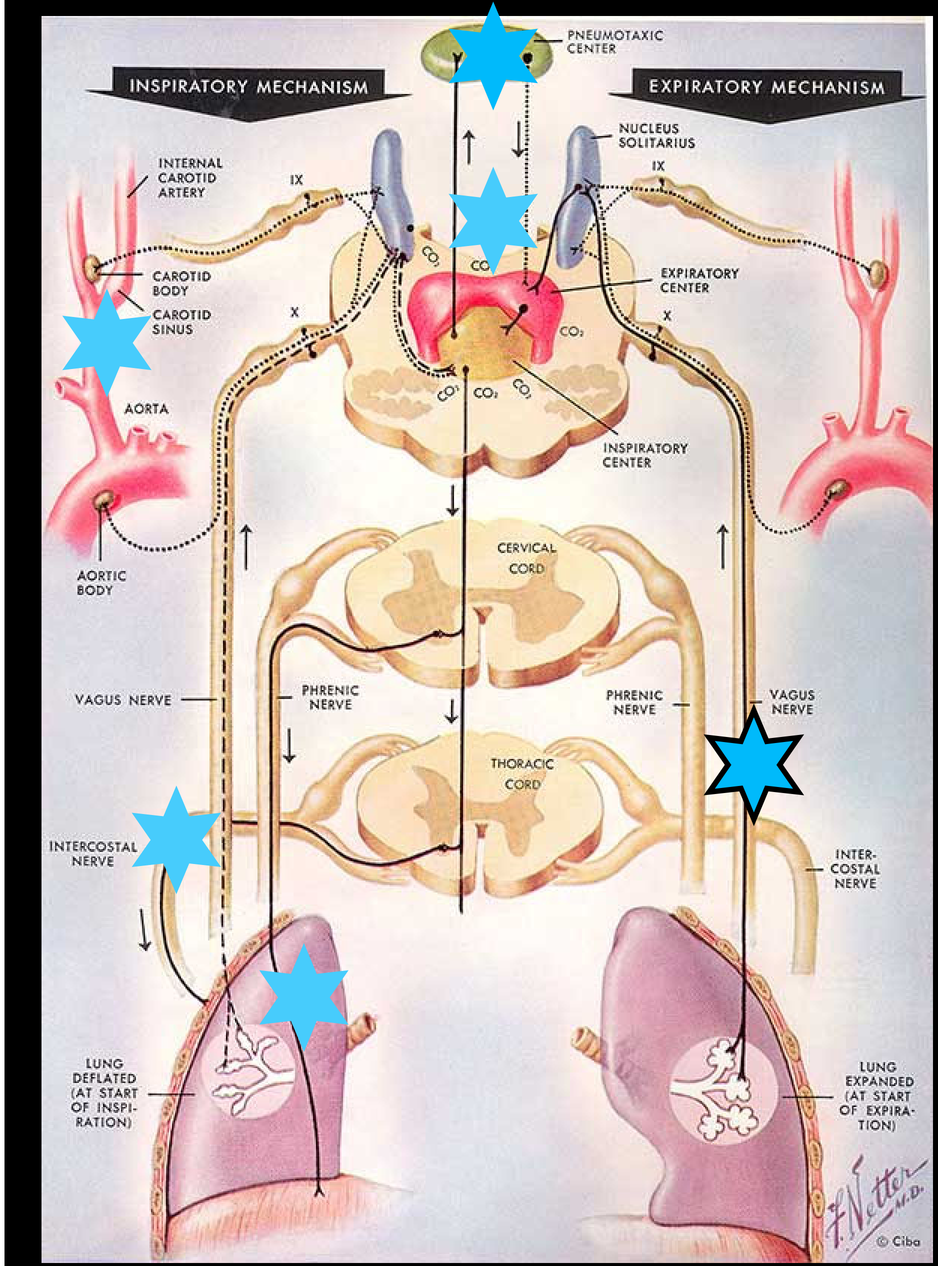

Collège national des enseignants d'anesthésie et de réanimation
Initiale Formation Continue
manuel de
Ventilation appliquée
en Anesthésie Réanimation et Médecine péri-opératoire

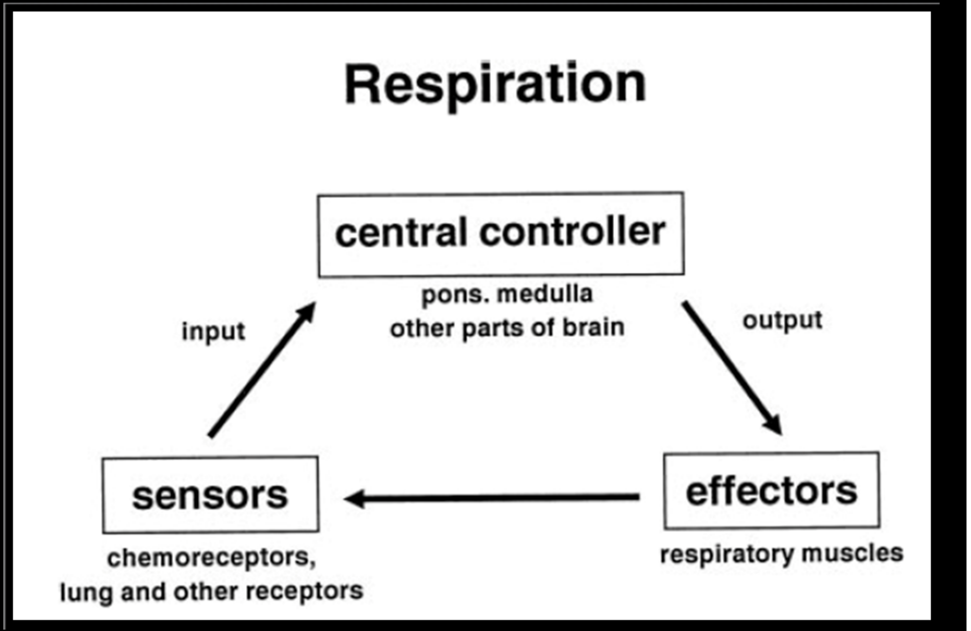
iVentilate

Physiopathologie respiratoire


Maladies obstructives
Maladies restrictives
Insuffisance respiratoire
Hypoxémie
Cœur pulmonaire
Hypercapnie
Dénutrition
Faiblesse musculaire
Acidose respiratoire
Alcalose métabolique
Dépression
Dyspnée
Rétention hydrosaline
Sédentarité

Innervation
Contrôle ventilatoire


INSPIRATORY MECHANISM
EXPIRATORY MECHANISM
PNEUMOTAXIC CENTER
NUCLEUS SOLITARIUS
INTERNAL CAROTID ARTERY
IX
IX
CAROTID BODY
CAROTID SINUS
X
X
CO₂
CO₂
CO₂
CO₂
CO₂
CO₂
EXPIRATORY CENTER
INSPIRATORY CENTER
AORTA
AORTIC BODY
CERVICAL CORD
VAGUS NERVE
PHRENIC NERVE
PHRENIC NERVE
VAGUS NERVE
THORACIC CORD
INTERCOSTAL NERVE
INTER-COSTAL NERVE
LUNG DEFLATED (AT START OF INSPI-RATION)
LUNG EXPANDED (AT START OF EXPIRA-TION)
F. Netter M.D.
© Ciba



Respiration

central controller

pons. medulla
other parts of brain

input

output

sensors

chemoreceptors,
lung and other receptors

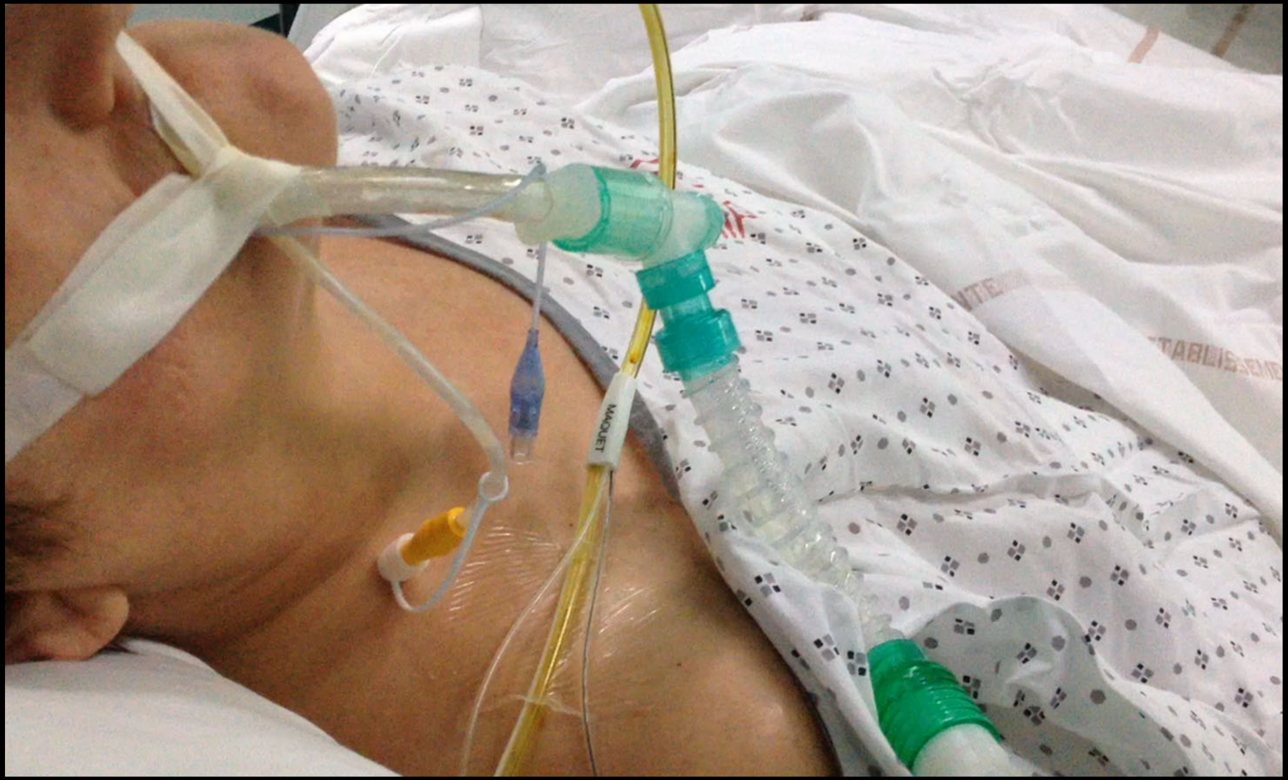
effectors

respiratory muscles

CHEST 2000; 117:205–225

Acidose respiratoire post opératoire

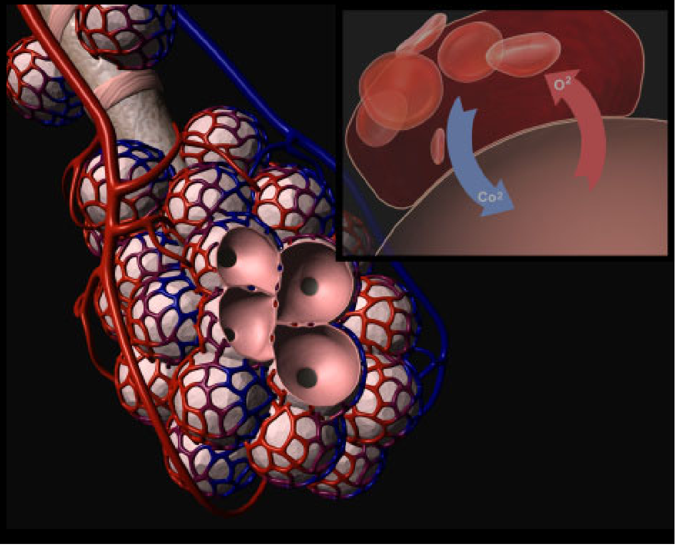
Muscles accessoires



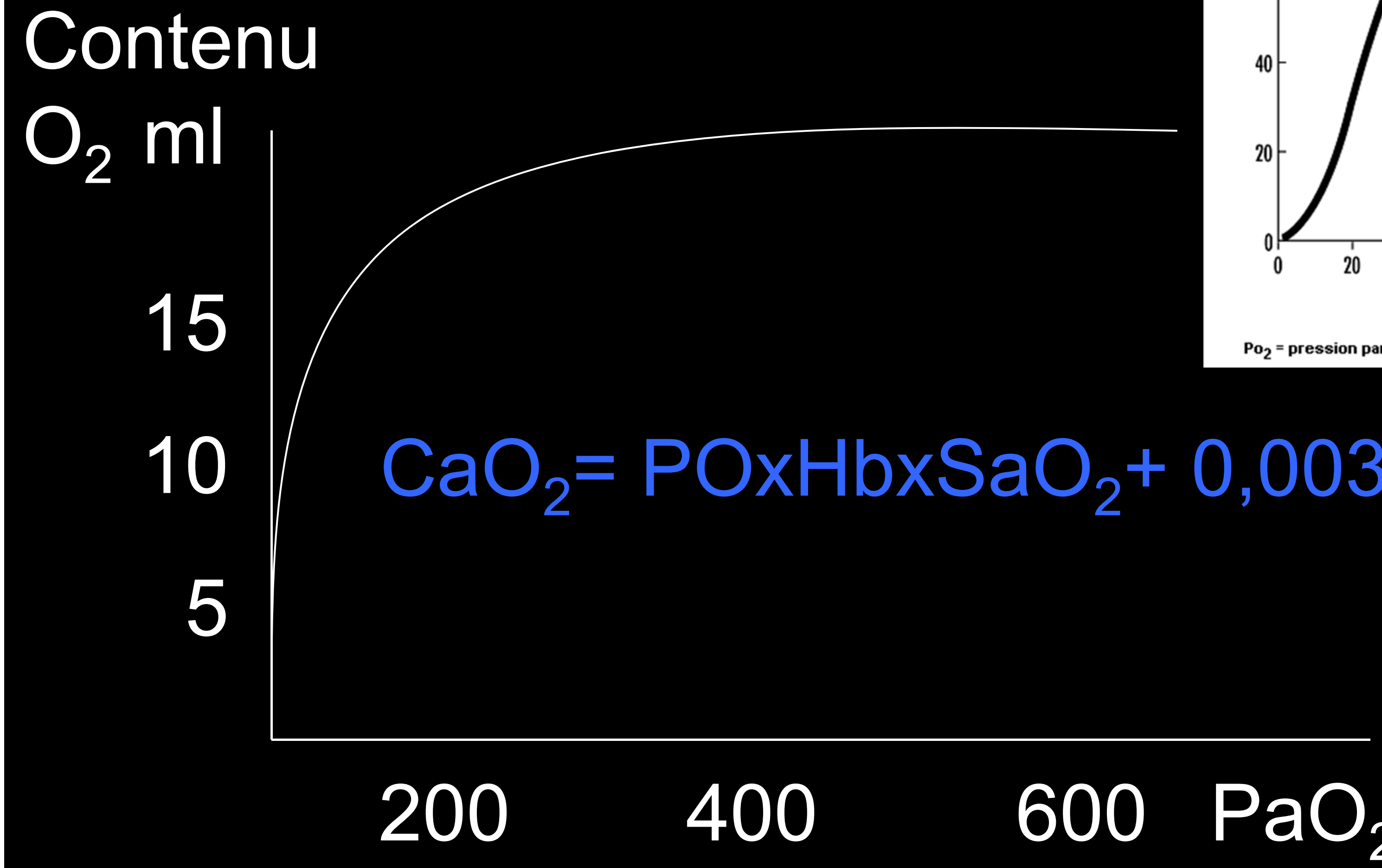
Définition

- Incapacité du poumon à assurer une oxygénation satisfaisante et/ou de prévenir une rétention de CO_2 :
  - Hypoxie < 60 mmHg
  - Hypercapnie > 50 mmHg

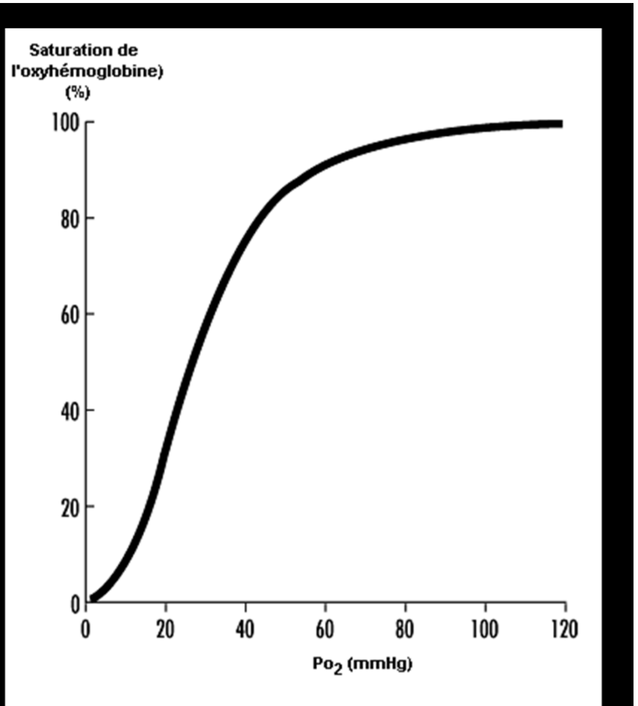
Ces valeurs limites dépendent de l ' histoire clinique



Maintenir CaO2



CaO_2= POxHbxSaO_2+ 0,003 PaO_2

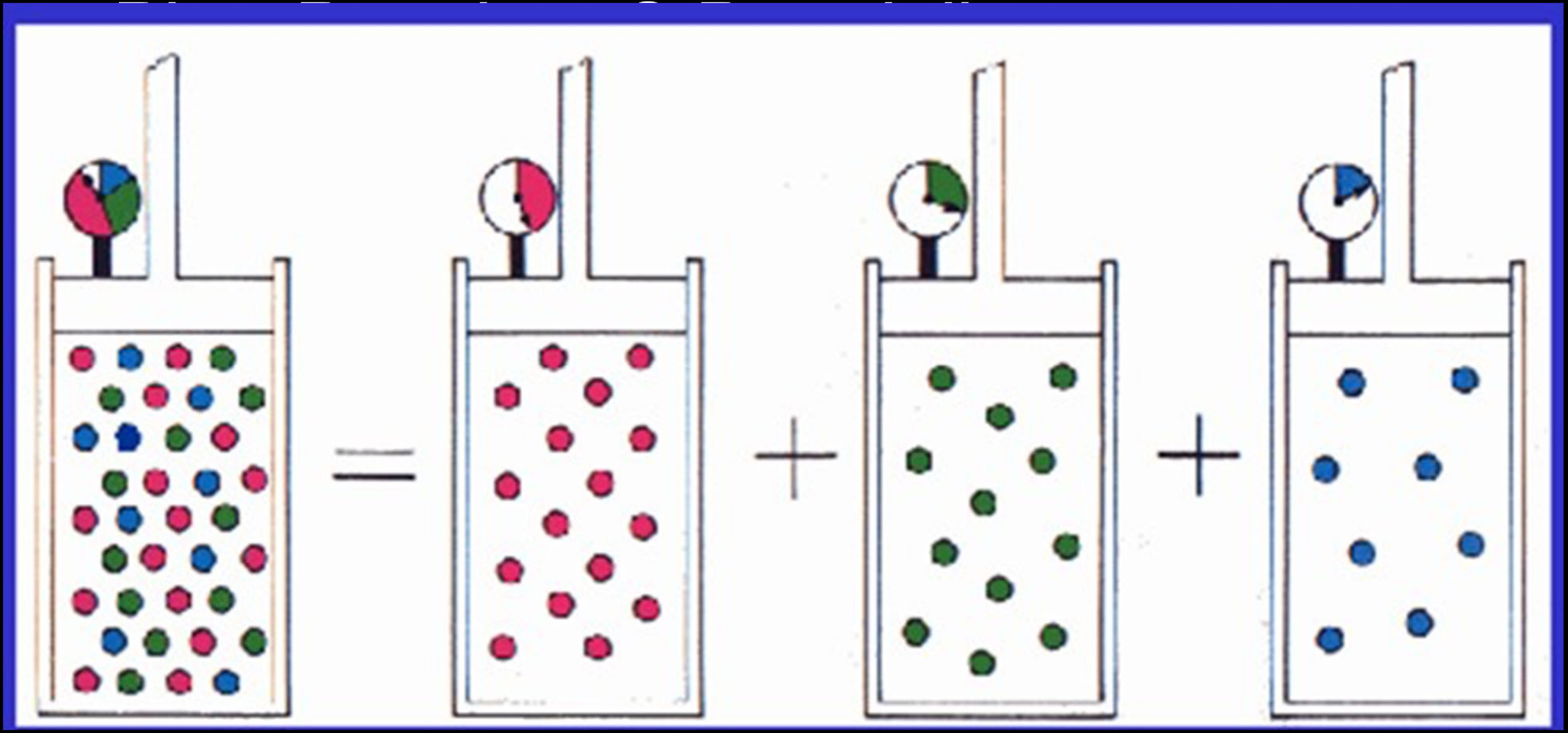


Po_2 = pression partielle d'oxygène dans le sang artériel.

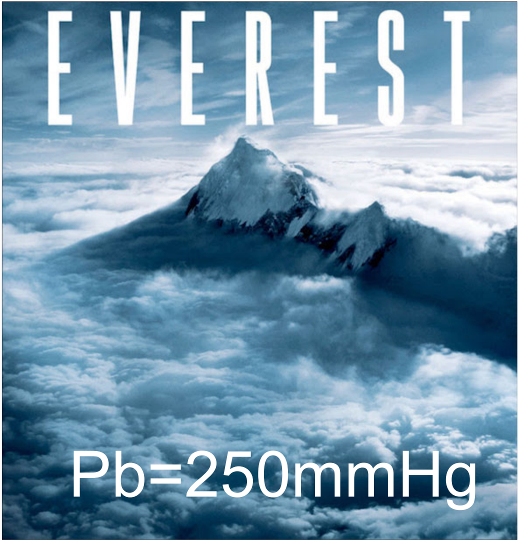
4 Causes de l’hypoxémie

- L’hypoventilation
- L’anomalie de diffusion
- Le shunt
- L’inégalité des rapports ventilation/perfusion

Loi des gaz



Hypoventilation

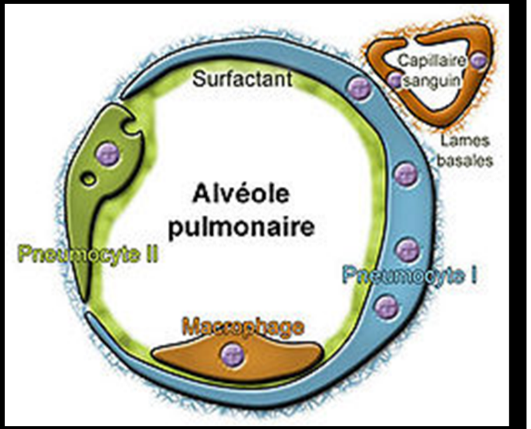

EVEREST
Pb=250mmHg

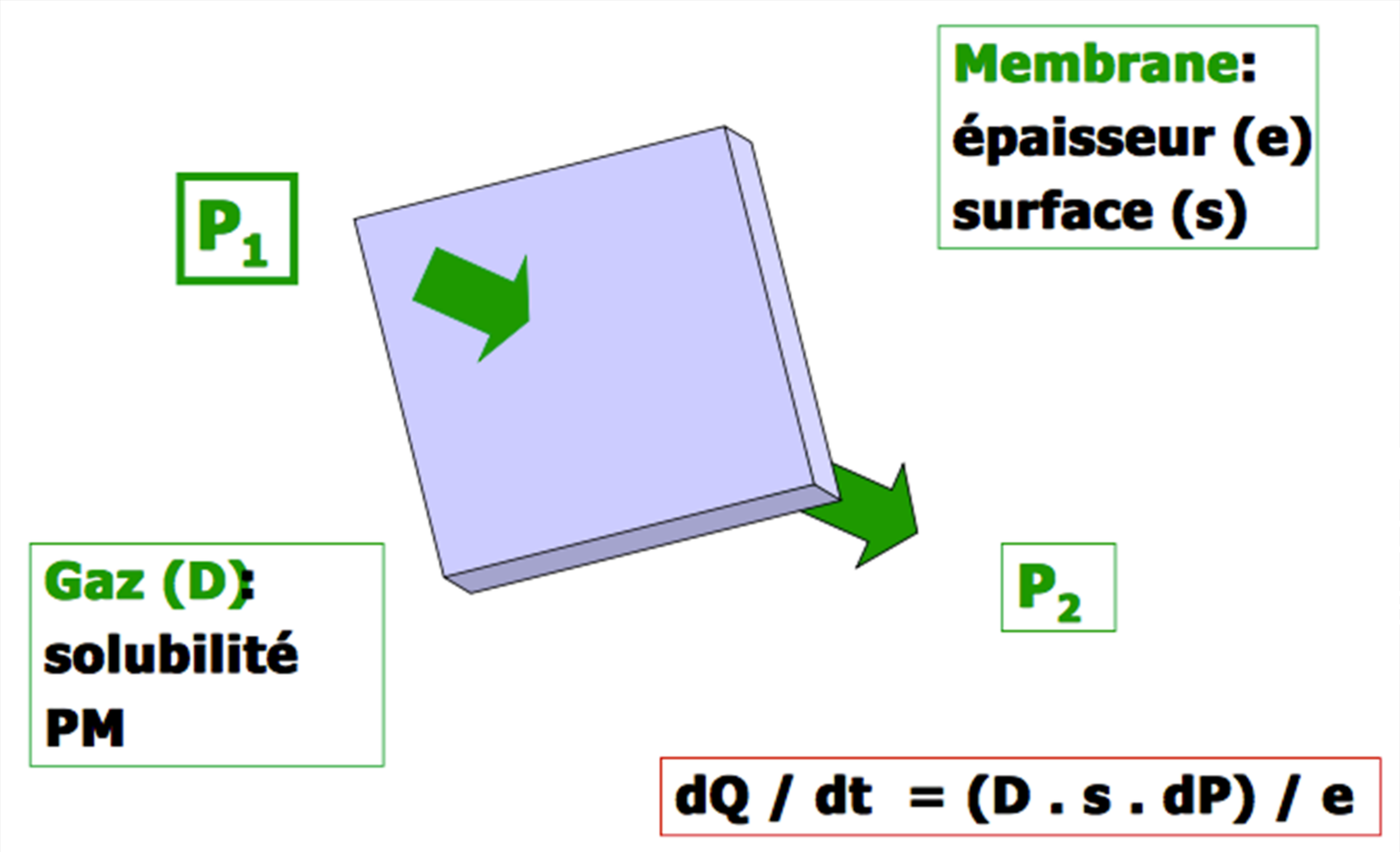
- Hypercapnie:
  - PaCO2 =K x VCO_2 / V_A
- Hypoxémie:
  - $P_AO_2 = PiO_2 - P_ACO_2 / R$
  - $PiO_2 = FiO_2 \times (Pb - PH_2O)$
  - $P_AO_2 = FiO_2 \times (Pb - PH_2O) - P_ACO_2 / R$

Etiologies

- Dépression des centres respiratoires : médicaments
- Atteinte du bulbe rachidien : encéphalite, TC, hémorragie, tumeur…
- Atteinte de la corne antérieure : poliomyélite
- Atteinte des nerfs des muscles respiratoires : Guillain Barré, diphtérie
- Atteinte des muscles respiratoires : dystrophie musculaire progressive
- Lésions de la cage thoracique : volet costal
- Obstruction des voies aériennes supérieures : tumeurs

Anomalies de diffusion


Capillaire sanguin
Surfactant
Lames basales
Alvéole pulmonaire
Pneumocyte II
Pneumocyte I
Macrophage



Membrane:
épaisseur (e)
surface (s)

P_1

P_2

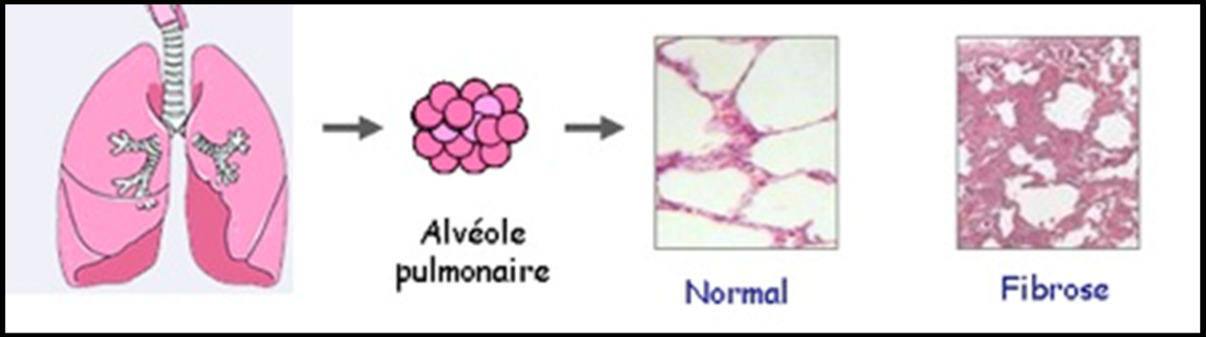
Gaz (D):
solubilité
PM

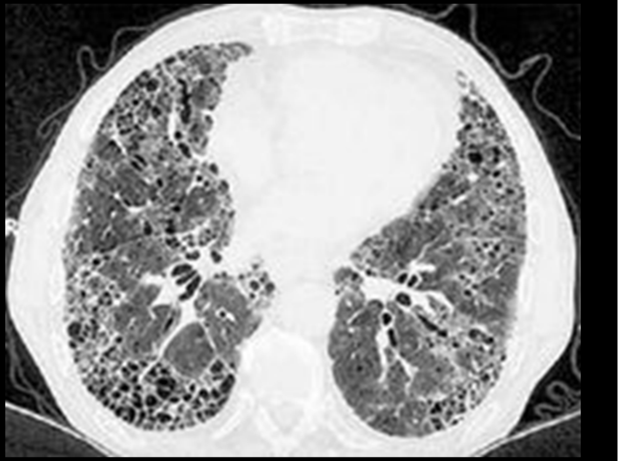
$$dQ / dt = (D . s . dP) / e$$

ilibre au 1/3.
coeur droit
entation

Etiologie

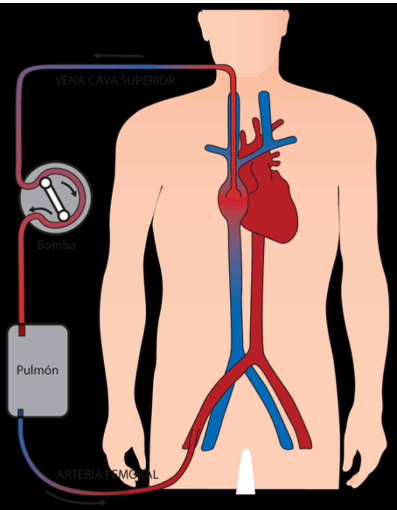
- Asbestose, sarcoidose, granulomatoses, fibrose interstitielle pulmonaire, sclerodermie , lupus ...


Alvéole pulmonaire
Normal
Fibrose



Le shunt

TURN
ONE WAY
ONE WAY
STOP
POSTED
NO HUNTING FISHING TRESPASSING
ONE WAY
KEEP LEFT
DETOUR
thinkstock by Getty Images


VENA CAVA SUPERIOR
Bomba
Pulmón
ARTERIA FEMORAL

- Du sang veineux atteint le système artériel :

  Calcul : $Q_S/Q_T=(Cc' - Ca)/(Cc' - Cv)$
  - Cc' calculé sous O_2 pur ($Sc'O_2=100\%$)
  - Ca GDS et Cv KT dans l'AP

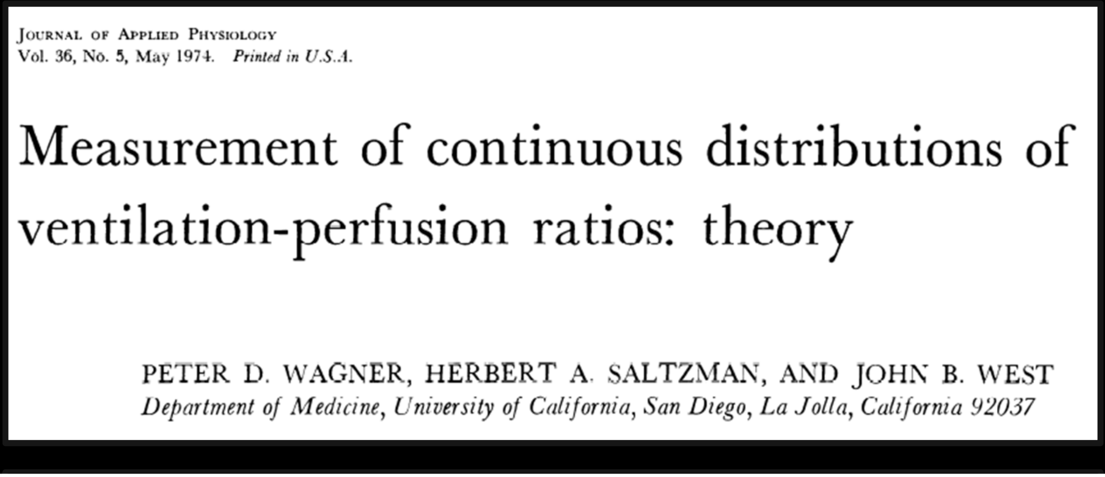
  normale < 10%

$PaCO_2$ rarement élevée car hyperventilation

Etiologies

- shunt vrai
  - Fistules artério-veineuses Syndrome hépatopulmonaire des Cirrhoses
  - Canal artériel, CIV, CIA et HTAP
- effet shunt

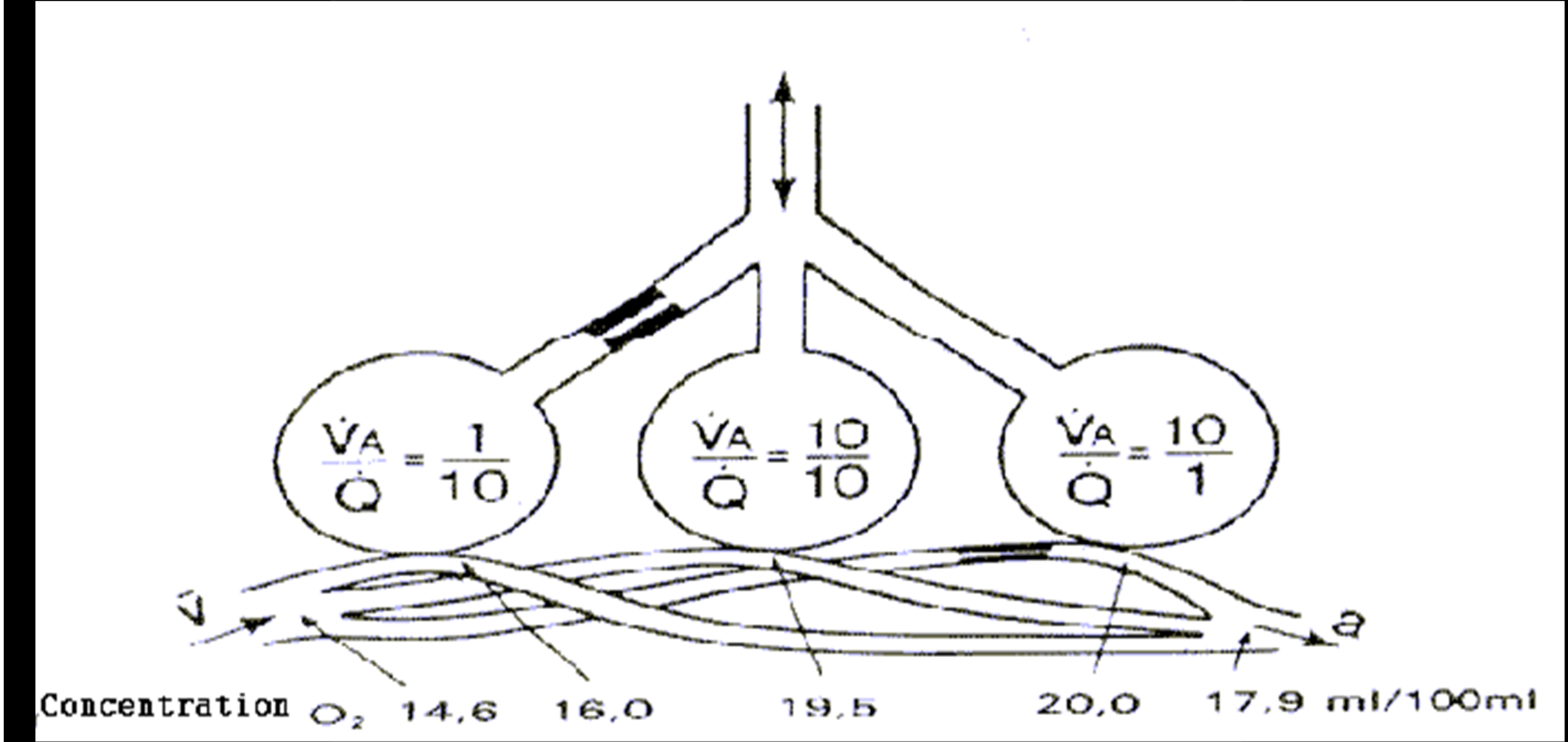
  = $V_A/Q \rightarrow 0$ correspond à une inégalité des rapports ventilation / perfusion
  - Atélectasies de l ’ anesthésie générale, PFLA,SDRA etc ...



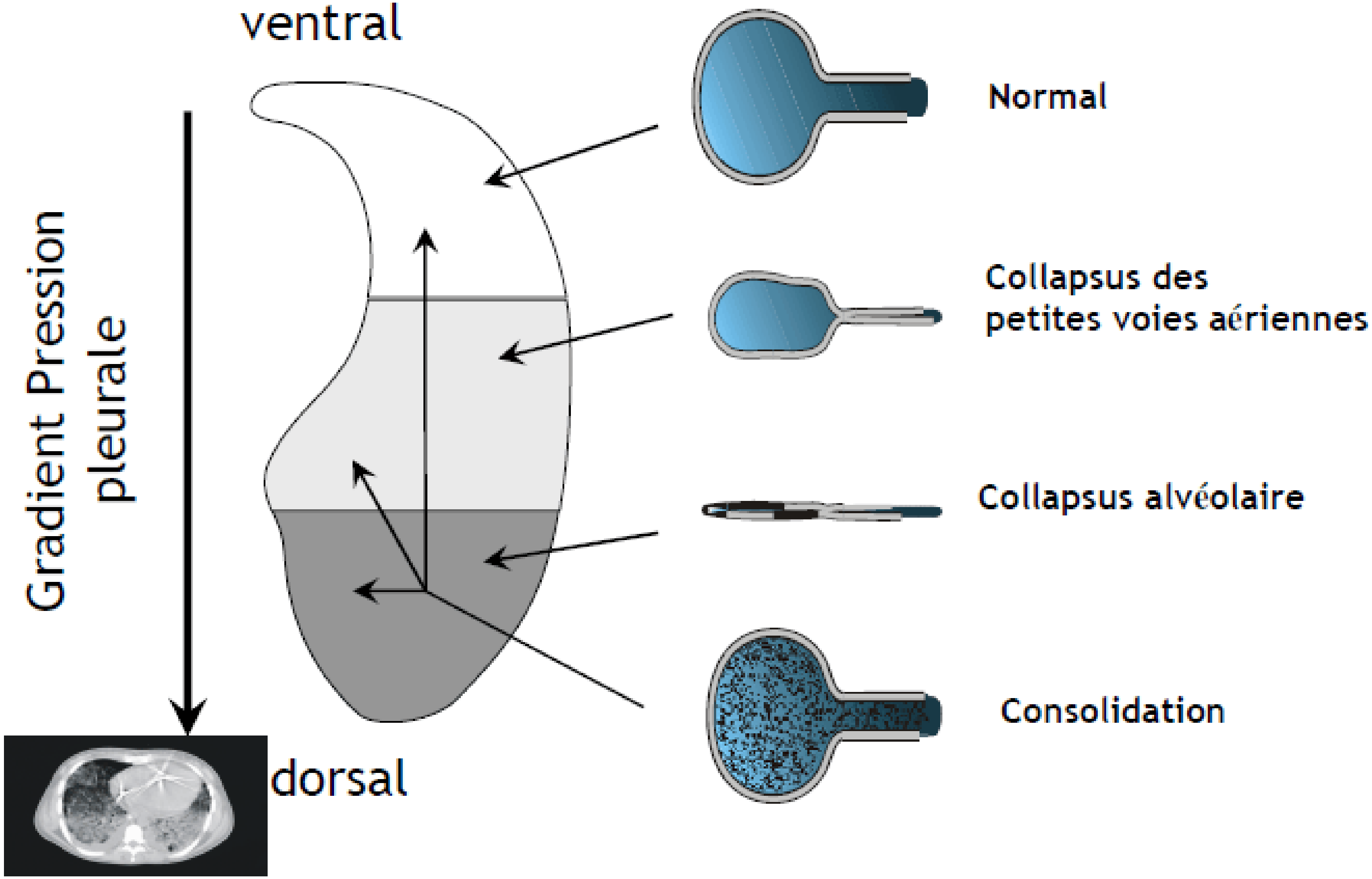
JOURNAL OF APPLIED PHYSIOLOGY
Vol. 36, No. 5, May 1974. *Printed in U.S.A.*

Measurement of continuous distributions of ventilation-perfusion ratios: theory

PETER D. WAGNER, HERBERT A. SALTZMAN, AND JOHN B. WEST
Department of Medicine, University of California, San Diego, La Jolla, California 92037


$\dot{V}_A / \dot{Q} = 1/10$
$\dot{V}_A / \dot{Q} = 10/10$
$\dot{V}_A / \dot{Q} = 10/1$
$\bar{v}$
a
Concentration O_2 14,6 16,0 19,5 20,0 17,9 ml/100ml

Inégalités des V_A/Q



ventral

Gradient Pression
pleurale

dorsal

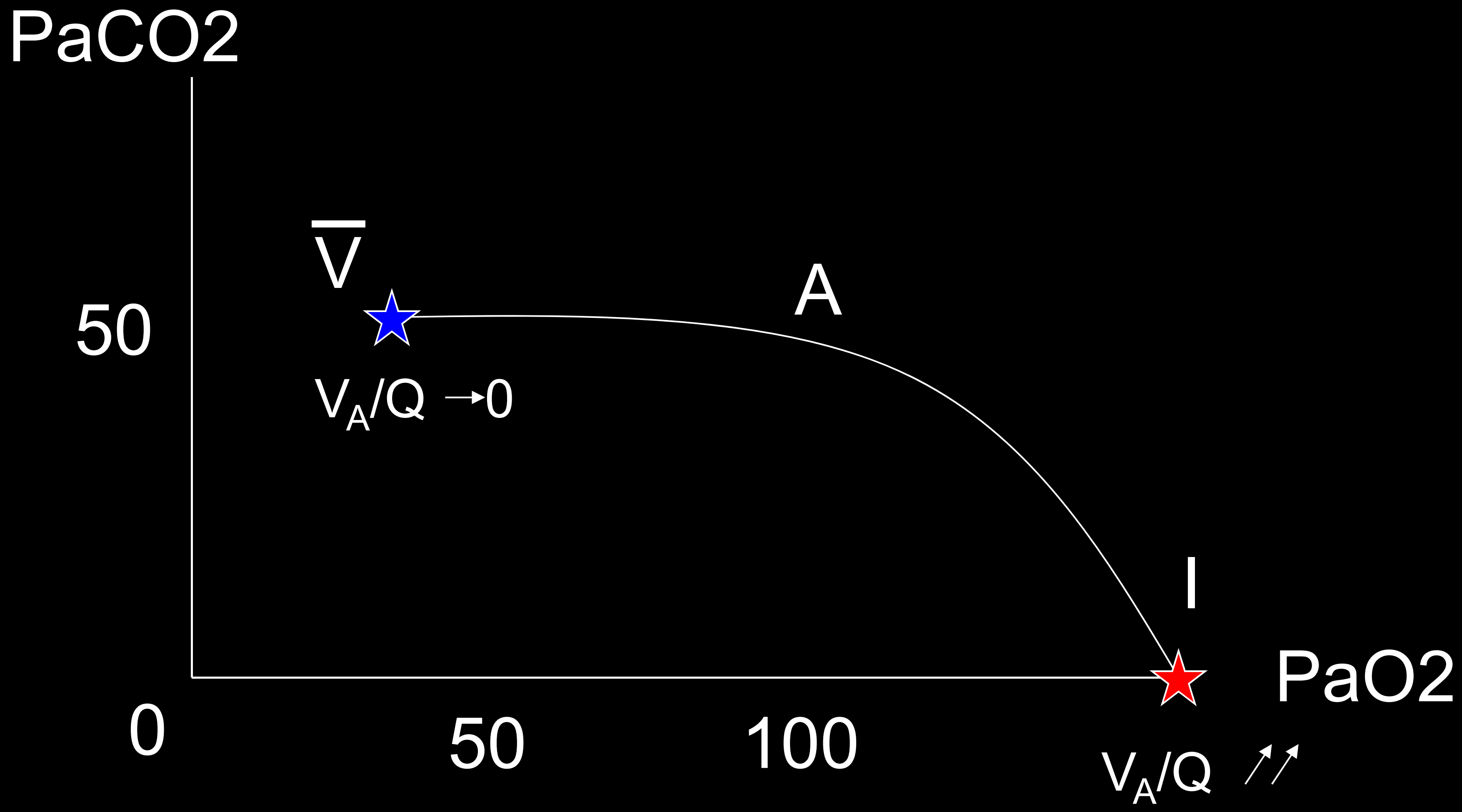
Normal

Collapsus des
petites voies aériennes

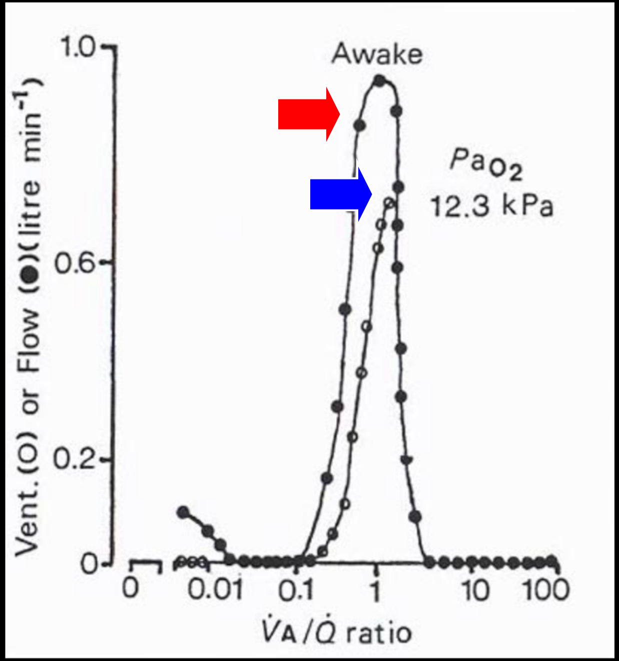
Collapsus alvéolaire

Consolidation

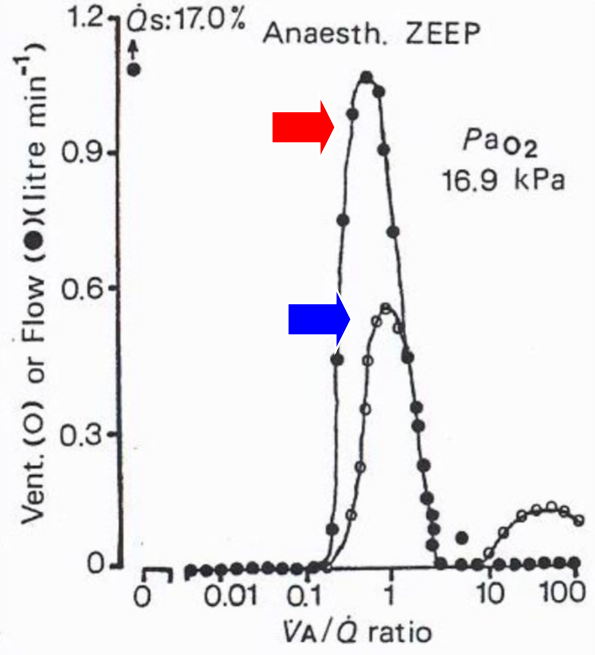
Inégalités des V_A/Q



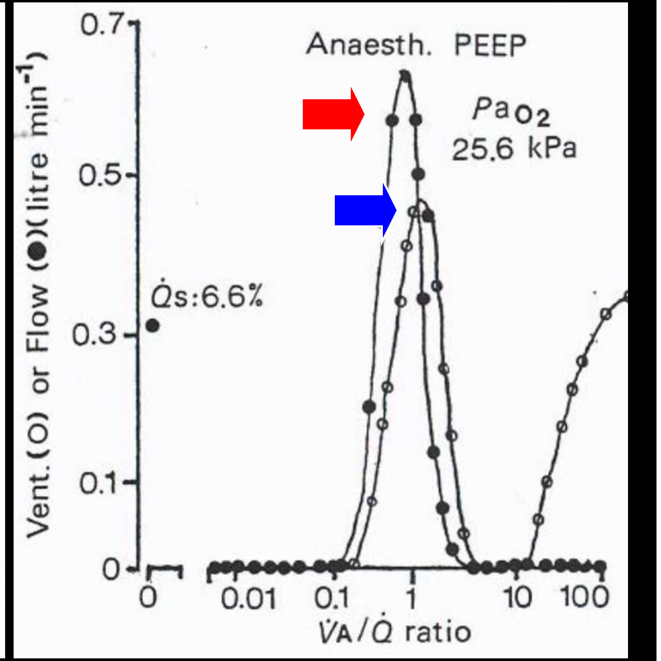
MESURE DES V_A/Q



Patient avant induction



Patient après induction



Patient avec une PEP

➡ Q

➡ V_A

Wagner PD, Saltzman HA, West JB.

Measurement of continuous distributions of ventilation-perfusion ratios: theory.

J Appl Physiol. 1974 May;36(5):588-99.

ADAPTATION

- $V_A/Q \searrow \Rightarrow PaO_2 \searrow \; PaCO_2 \nearrow$
- Stimulation des chémorécepteurs périphériques et centraux : Hyperventilation, VPH, agents vaso actifs, système nerveux autonome…
- $PaO_2 \nearrow \; PaCO_2 \searrow$

Etiologies

- Pneumopathie infectieuse
- Embolie pulmonaire,embolie graisseuse, chest syndrome, TRALI
- OAP
- SDRA pulmonaires et extrapulmonaires
- Insuffisance respiratoire chronique décompensée, surinfection, bronchospasme: aggravation des inégalité des V_A/Q augmentation du W respiratoire.
- Atélectasies, inhalation, pneumo-hémothorax, contusion pulmonaire

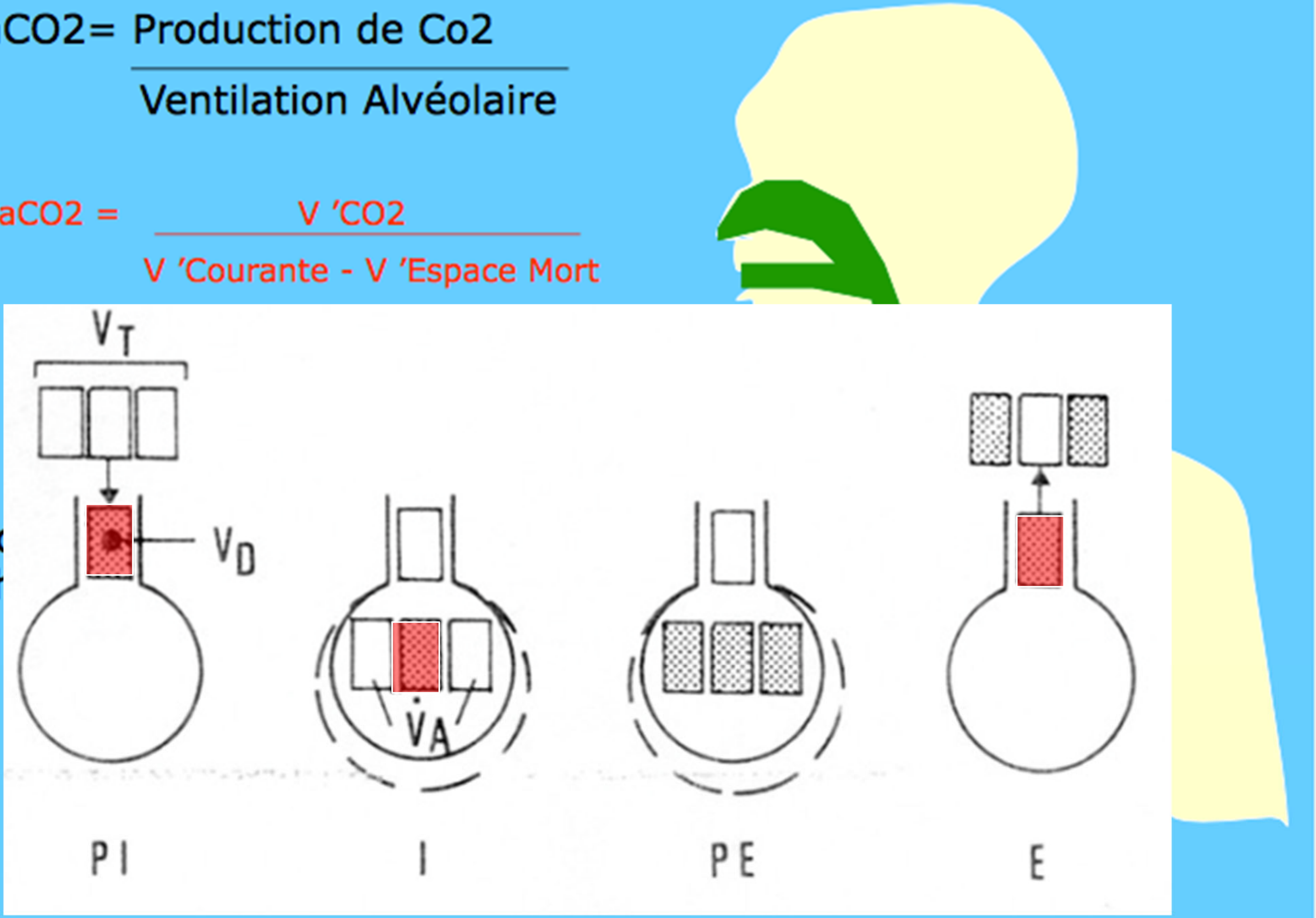
V 'Alvéolaire et PaCO$_2$

$$PaCO2 = \frac{\text{Production de Co2}}{\text{Ventilation Alvéolaire}}$$

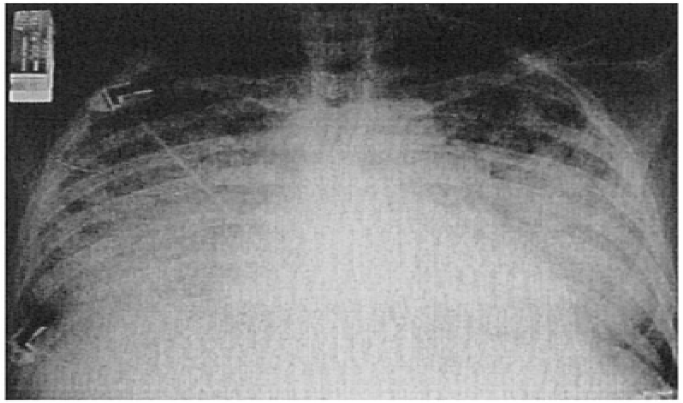
$$PaCO2 = \frac{V\ 'CO2}{V\ 'Courante - V\ 'Espace\ Mort}$$

Vo

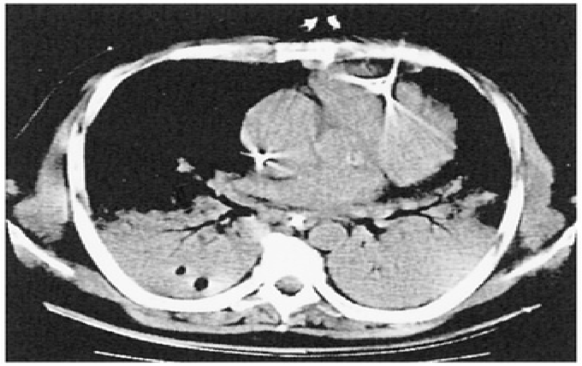
Ph


V_T
V_D
$\dot{V}_A$
PI
I
PE
E

Améliorer la ventilation alvéolaire

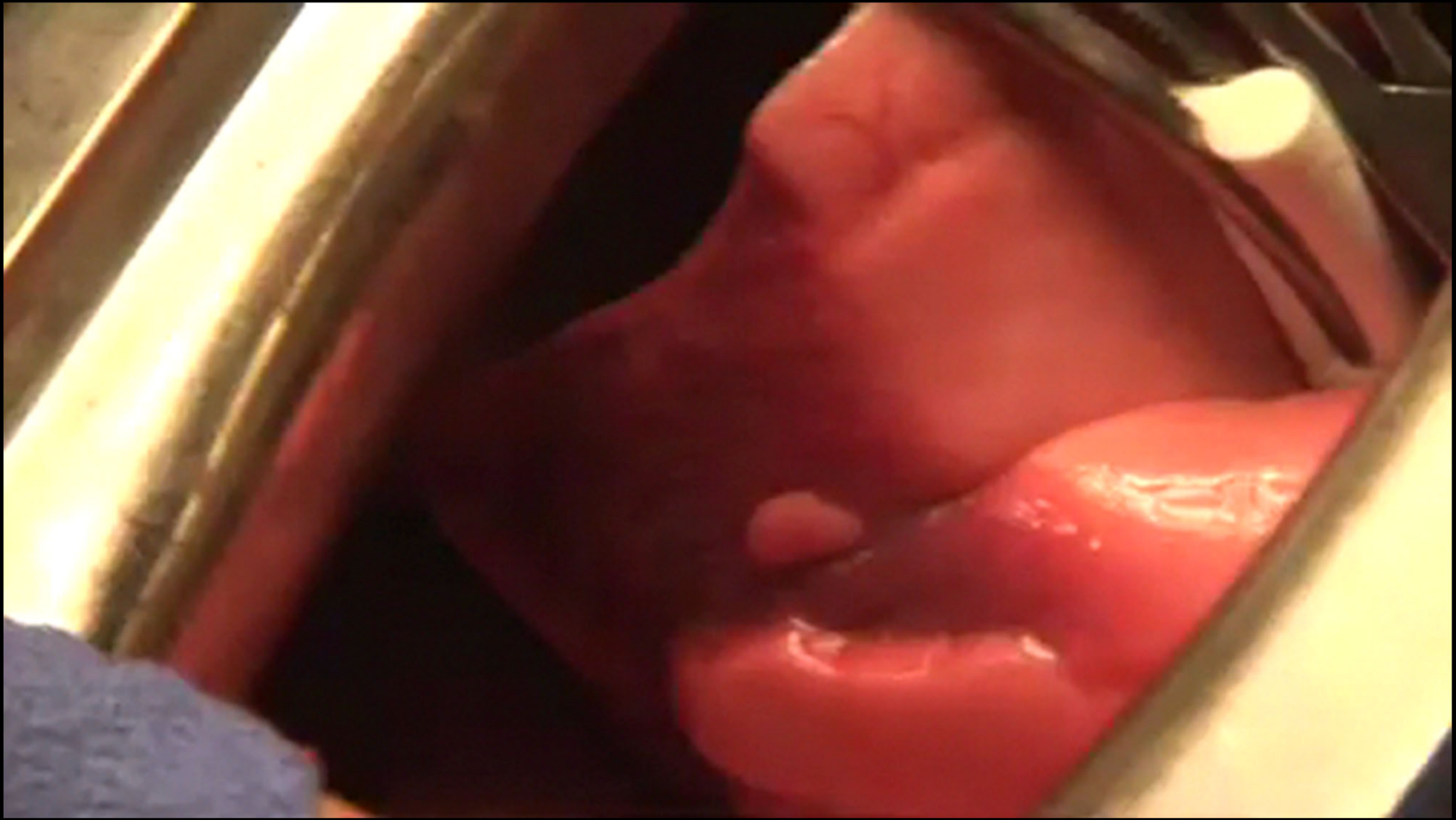


FiO_2 = 100%, TV = 4 mL/kg,
PEEP = 0 cm H_2O

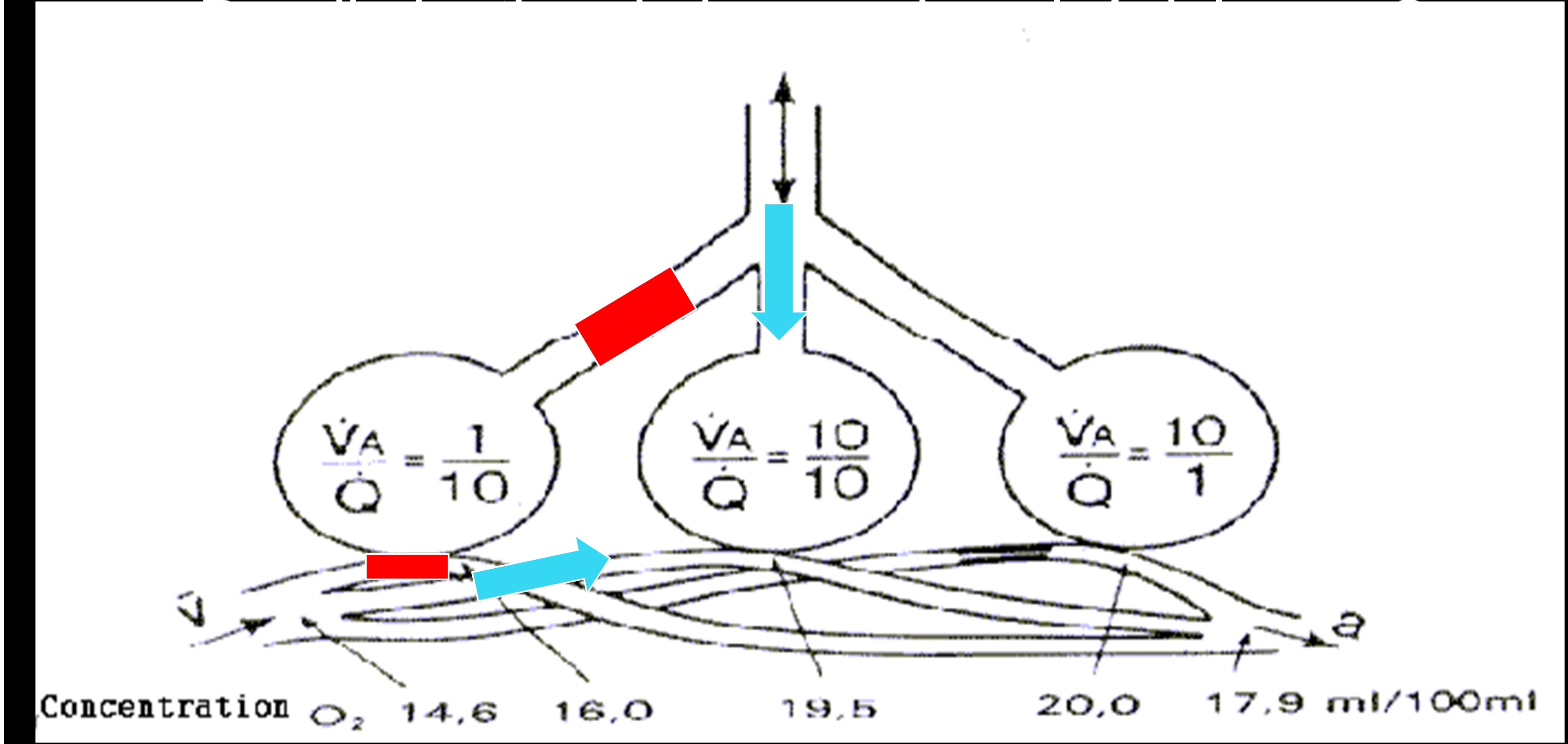


Nonaerated tissue = 55.6%
PaO_2/FiO_2 = 100

Trouble de la ventilation



Stimulation de la VPH

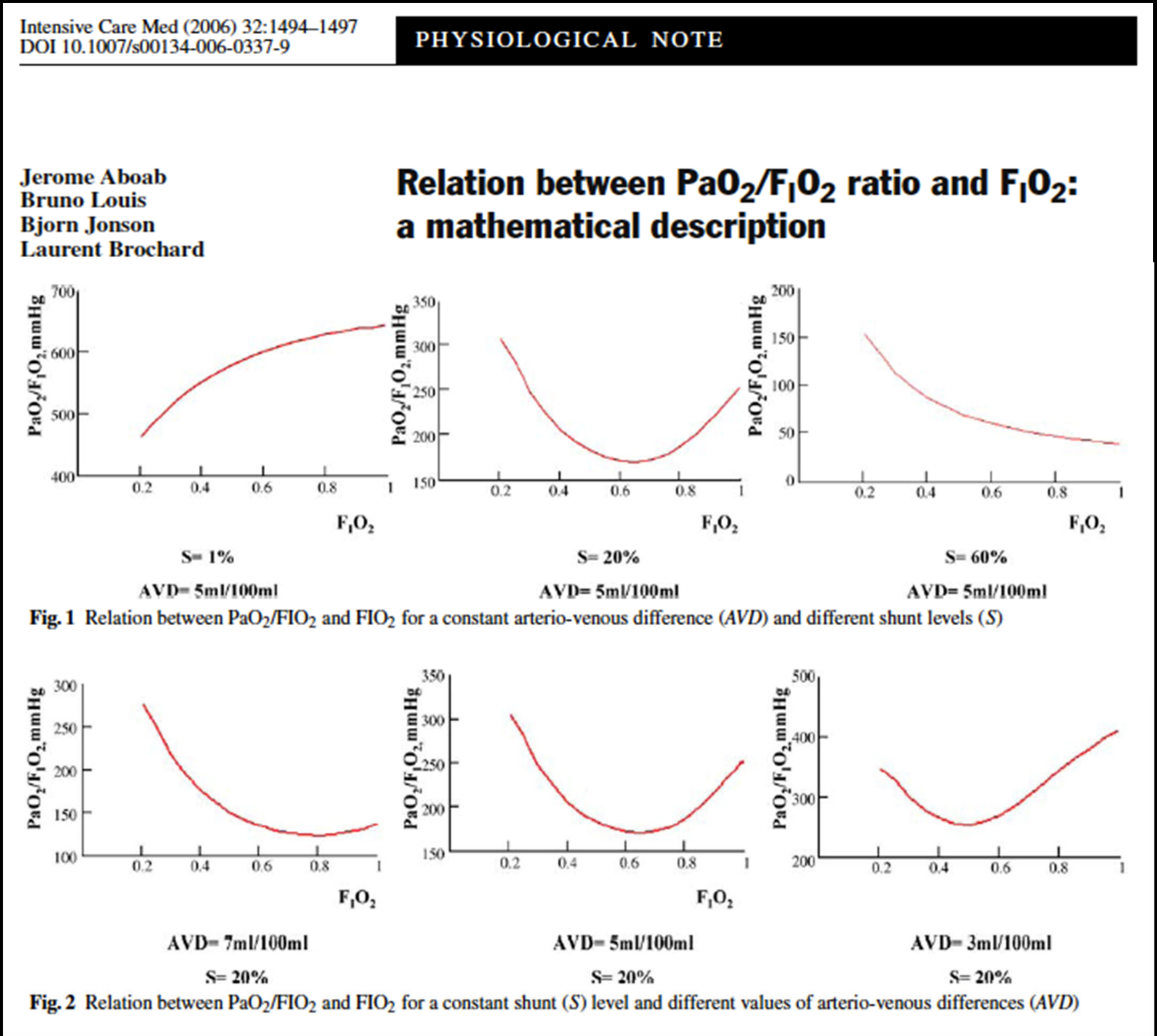

$\frac{\dot{V}_A}{\dot{Q}} = \frac{1}{10}$
$\frac{\dot{V}_A}{\dot{Q}} = \frac{10}{10}$
$\frac{\dot{V}_A}{\dot{Q}} = \frac{10}{1}$
$\bar{v}$
a
Concentration O_2 14,6 16,0 19,5 20,0 17,9 ml/100ml

Débit cardiaque et V_a/q

Il y peut y avoir en pratique clinique une amélioration de la PaO_2 sans recrutement alvéolaire :

-La diminution du débit cardiaque entraine une diminution de l'effet shunt (ex verticaliser un patient en ARDS)
redistribution du sang des zones de hauts vers bas Va/Q
(effet du NO inhalé) .On observe alors une augmentation de l'espace mort et donc de la $PaCO_2$.

Par contre à ventilation alvéolaire constante une diminution de la $PaCO_2$ correspond plus spécifiquement à un recrutement alvéolaire.



Intensive Care Med (2006) 32:1494–1497
DOI 10.1007/s00134-006-0337-9

PHYSIOLOGICAL NOTE

Jerome Aboab
Bruno Louis
Bjorn Jonson
Laurent Brochard

Relation between PaO_2/F_IO_2 ratio and F_IO_2: a mathematical description

S= 1%
AVD= 5ml/100ml

S= 20%
AVD= 5ml/100ml

S= 60%
AVD= 5ml/100ml

Fig. 1 Relation between PaO_2/FIO_2 and FIO_2 for a constant arterio-venous difference (*AVD*) and different shunt levels (*S*)

AVD= 7ml/100ml
S= 20%

AVD= 5ml/100ml
S= 20%

AVD= 3ml/100ml
S= 20%

Fig. 2 Relation between PaO_2/FIO_2 and FIO_2 for a constant shunt (*S*) level and different values of arterio-venous differences (*AVD*)

Capacités pulmonaires

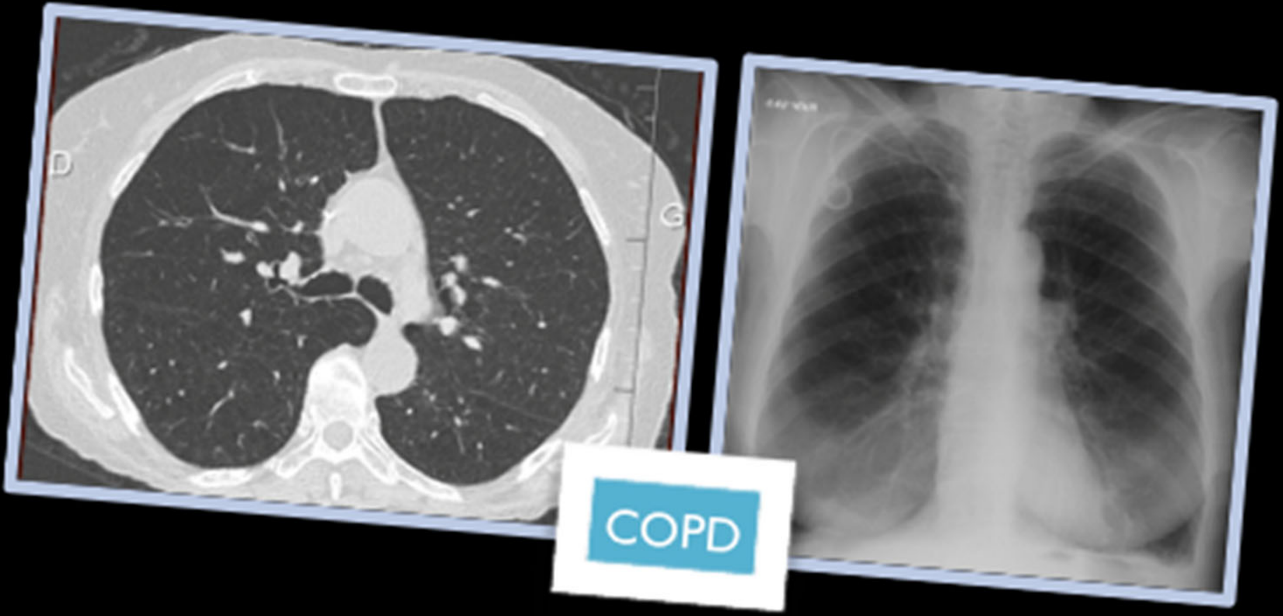
- Capacité pulmonaire totale =

  capacité vitale + volume résiduel

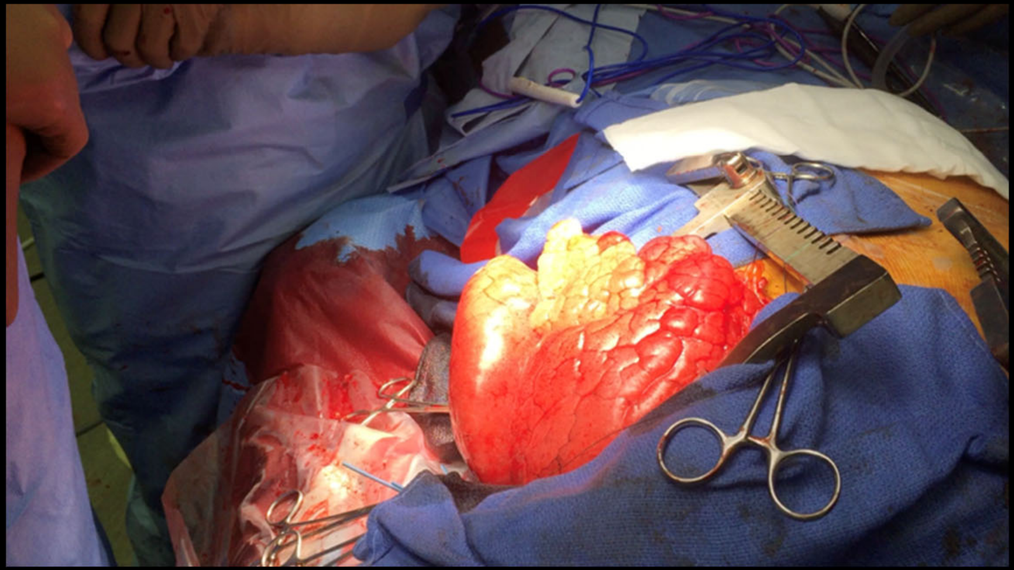
  =3,6 à 4,8 L + 1,2 à 2,4 L
- Capacité vitale = VRE + VC + VRI

  =1 à 1,2 L + 0,5 L + 2,1 à 3,1L
- CRF = VR + VRE

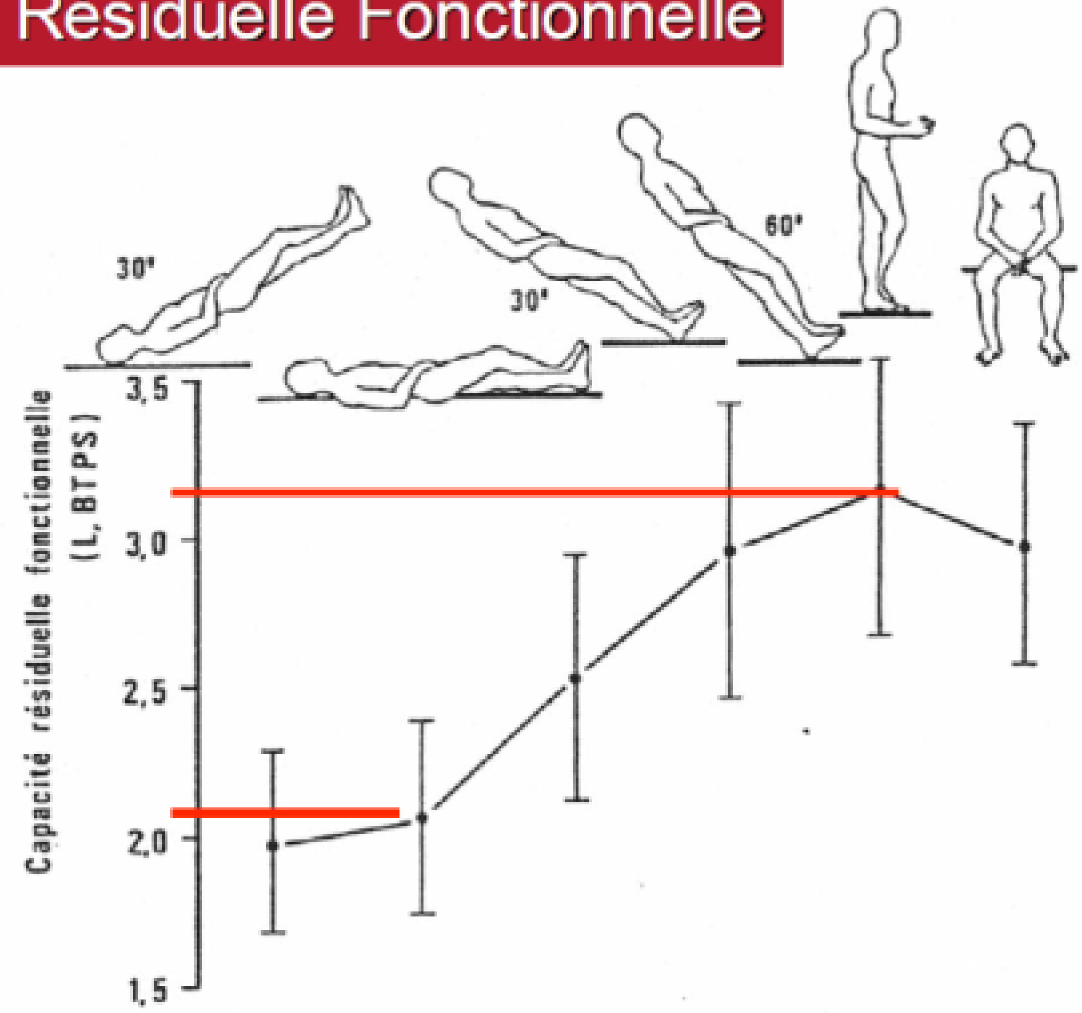
  = 1,2 à 2,4 L + 1 à 1,2 L



COPD



Effets de la position sur la Capacité Résiduelle Fonctionnelle



GDS

- Ph
- PaO2
- PaCO2
- HCO-
- SaO2
- Hypoxémie, Hyper Hypocapnie, Acidose Alcalose, métabolique, respiratoire

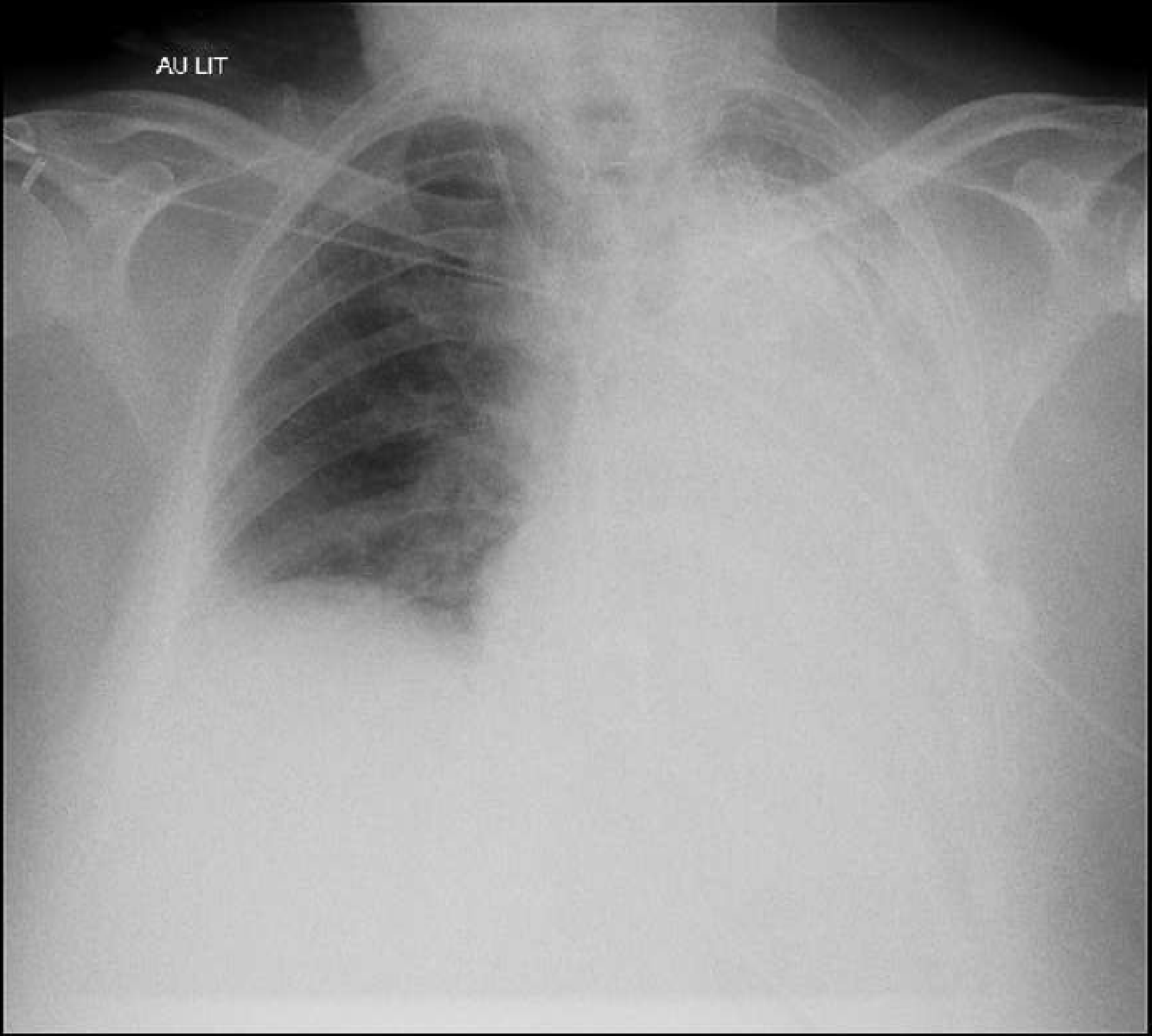
Cas clinique N° 1

- Mr X. Toxicomane
- Admis au SAU pour coma par overdose.
- FR : 10
- GDS en AA:
  - PO2 : 50 mmHg
  - PCO2 : 60 mmHg
  - pH : 7,25
  - Bicar : 22 mmol/l
  - BE : - 2

Analyse GDS

- Hypoventilation alvéolaire
  - Hypoxie
  - Hypercapnie
- Acidose mixte
  - Hypercapnie
  - Souffrance tissulaire (faible)
- Cause :
  - Bradypnée
  - PNP ?

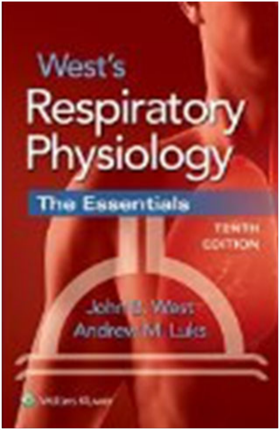
- Injection de Narcan.
- FR : 32
- GDS en AA:
  - PO2 : 60 mmHg
  - PCO2: 25 mmHg
  - pH : 7, 46
  - Bicar : 22 mmol/l
- RT


AU LIT

Analyse des GDS

- Hypoxie
- Hypocapnie par hyperventilation
- Hétérogénéité des rapports ventilation perfusion
- RT: PNP poumon G

RÉFÉRENCES

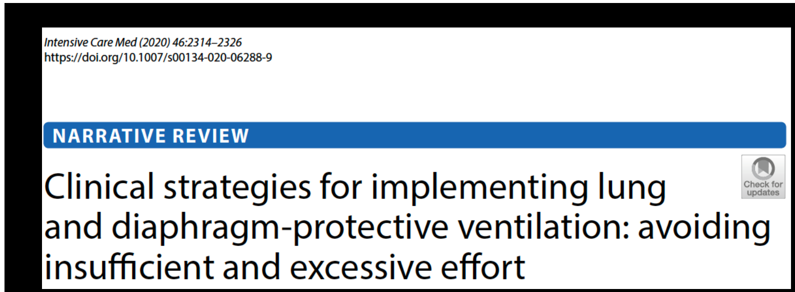

West's
Respiratory
Physiology
The Essentials
TENTH EDITION
John B. West
Andrew M. Luks

- West JB. Pulmonary Pathophysiologie - the essential. *1995 édition Pradel*.

You Tube https://www.youtube.com/watch?v=qLroD-Qcg4o

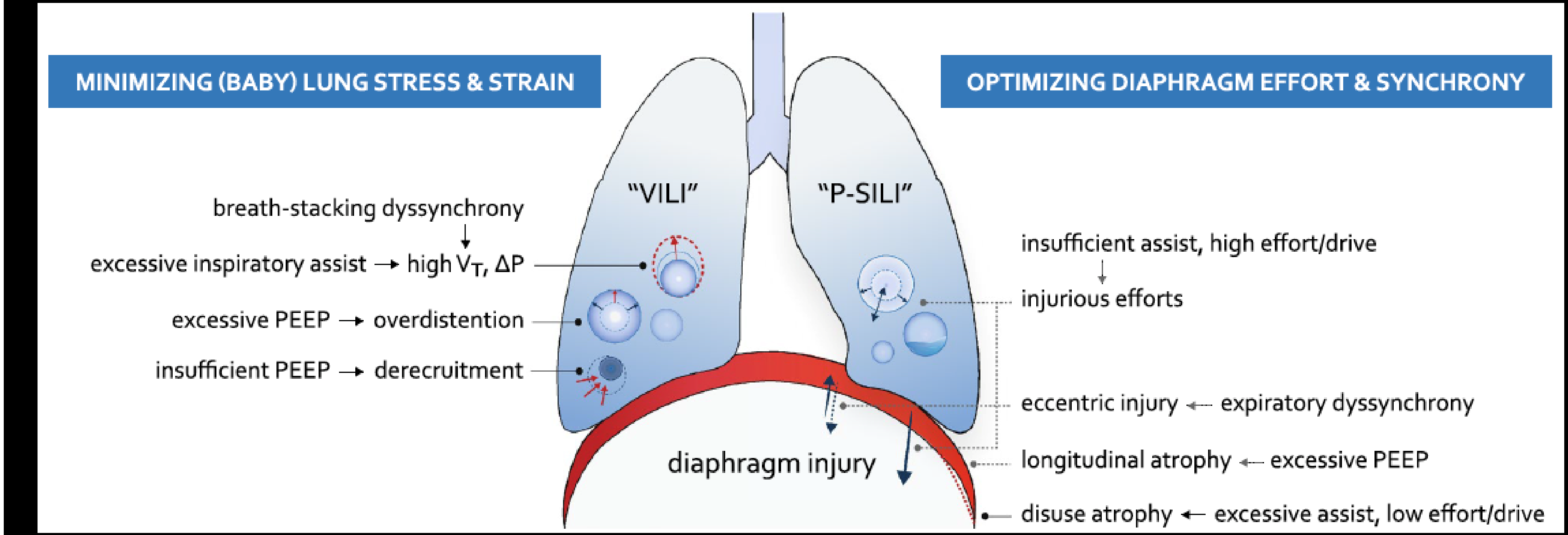
UC San Diego
Health Sciences

Les lésions induites par la ventilation


Intensive Care Med (2020) 46:2314–2326
https://doi.org/10.1007/s00134-020-06288-9

NARRATIVE REVIEW

Clinical strategies for implementing lung and diaphragm-protective ventilation: avoiding insufficient and excessive effort


MINIMIZING (BABY) LUNG STRESS & STRAIN

OPTIMIZING DIAPHRAGM EFFORT & SYNCHRONY

breath-stacking dyssynchrony
↓
excessive inspiratory assist → high V_T, ΔP

excessive PEEP → overdistention

insufficient PEEP → derecruitment

insufficient assist, high effort/drive
↓
injurious efforts

eccentric injury ← expiratory dyssynchrony

longitudinal atrophy ← excessive PEEP

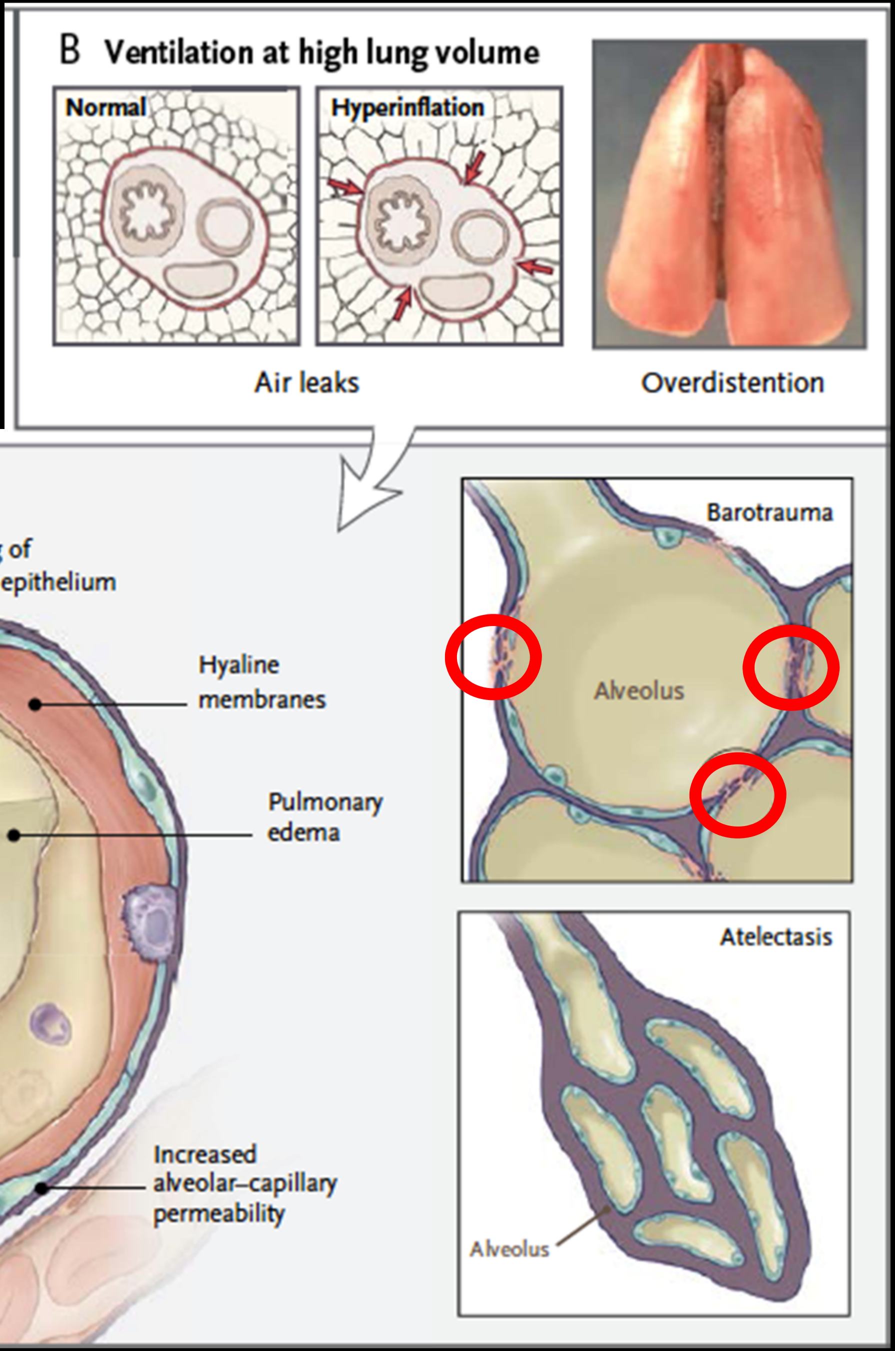
disuse atrophy ← excessive assist, low effort/drive

Lésions

Ruptures

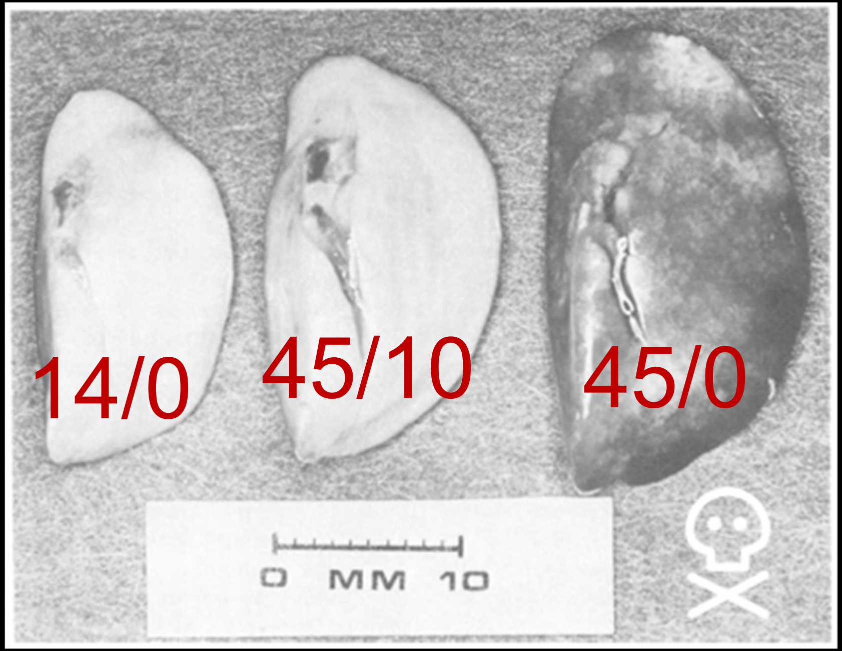
ViLi

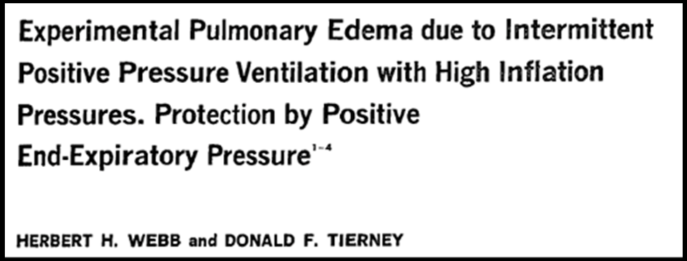
Barotraumatismes


B Ventilation at high lung volume
Normal
Hyperinflation
Air leaks
Overdistention
C Structural consequences
Sloughing of bronchial epithelium
Epithelial–mesenchymal transformations
Hyaline membranes
Alveolus
Pulmonary edema
Surfactant dysfunction
Fibroproliferation
Increased alveolar–capillary permeability
Barotrauma
Alveolus
Atelectasis
Alveolus

Lésions induites par la ventilation

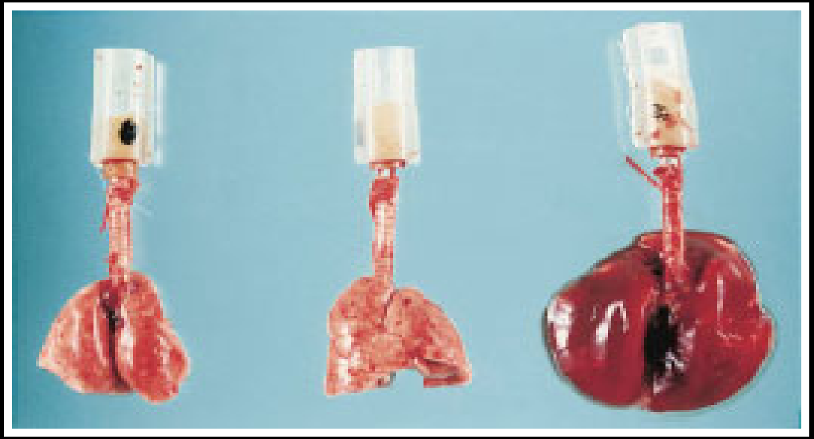
Pplat/PEP




Experimental Pulmonary Edema due to Intermittent Positive Pressure Ventilation with High Inflation Pressures. Protection by Positive End-Expiratory Pressure[1-4]

HERBERT H. WEBB and DONALD F. TIERNEY

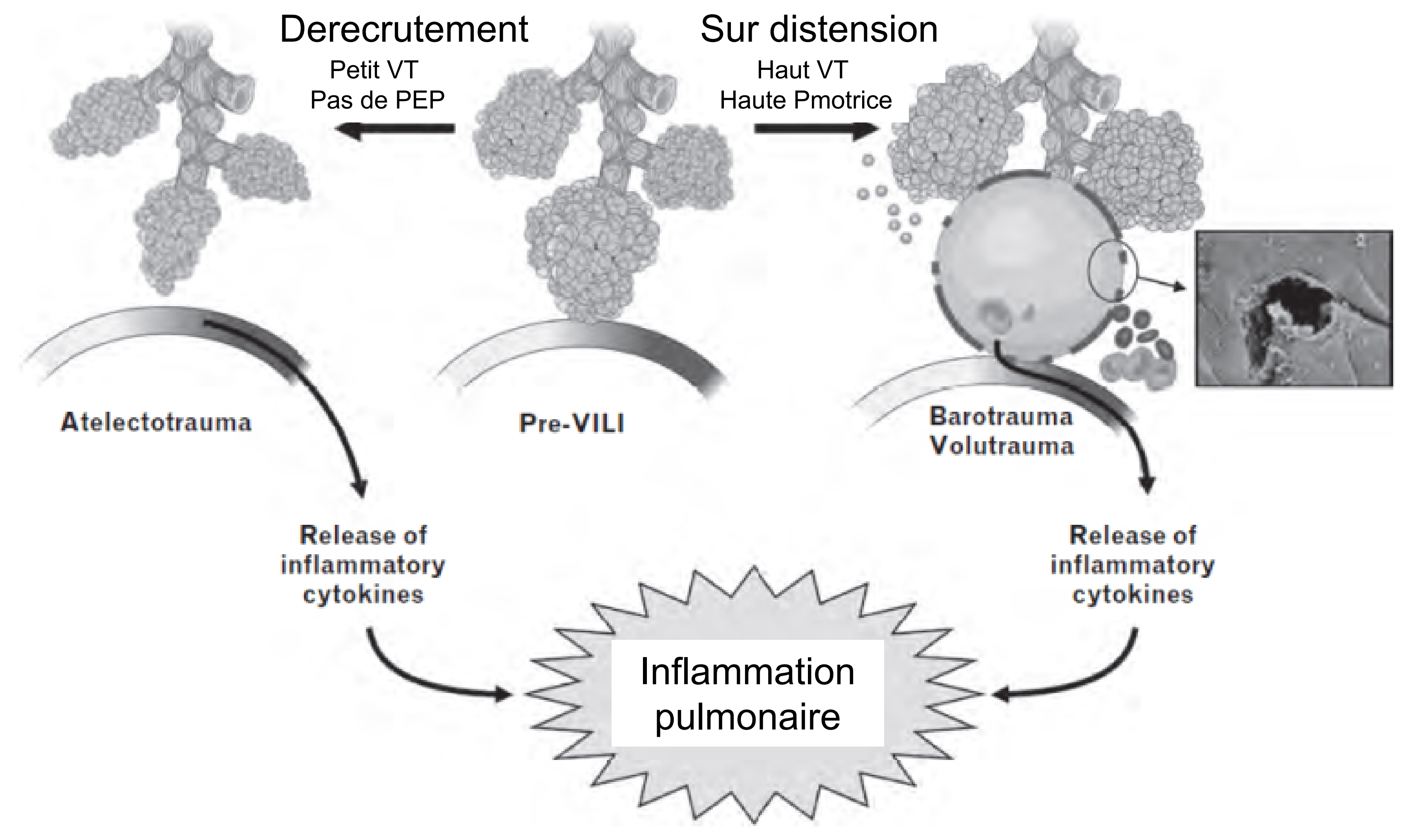
AMERICAN REVIEW OF RESPIRATORY DISEASE, VOLUME 110, 1974



Dreyfuss D, Saumon G

Ventilator-induced lung injury: lessons from experimental studies.

Am J Respir Crit Care Med. 1998 ;157:294-323. Review.



Derecrutement

Petit VT
Pas de PEP

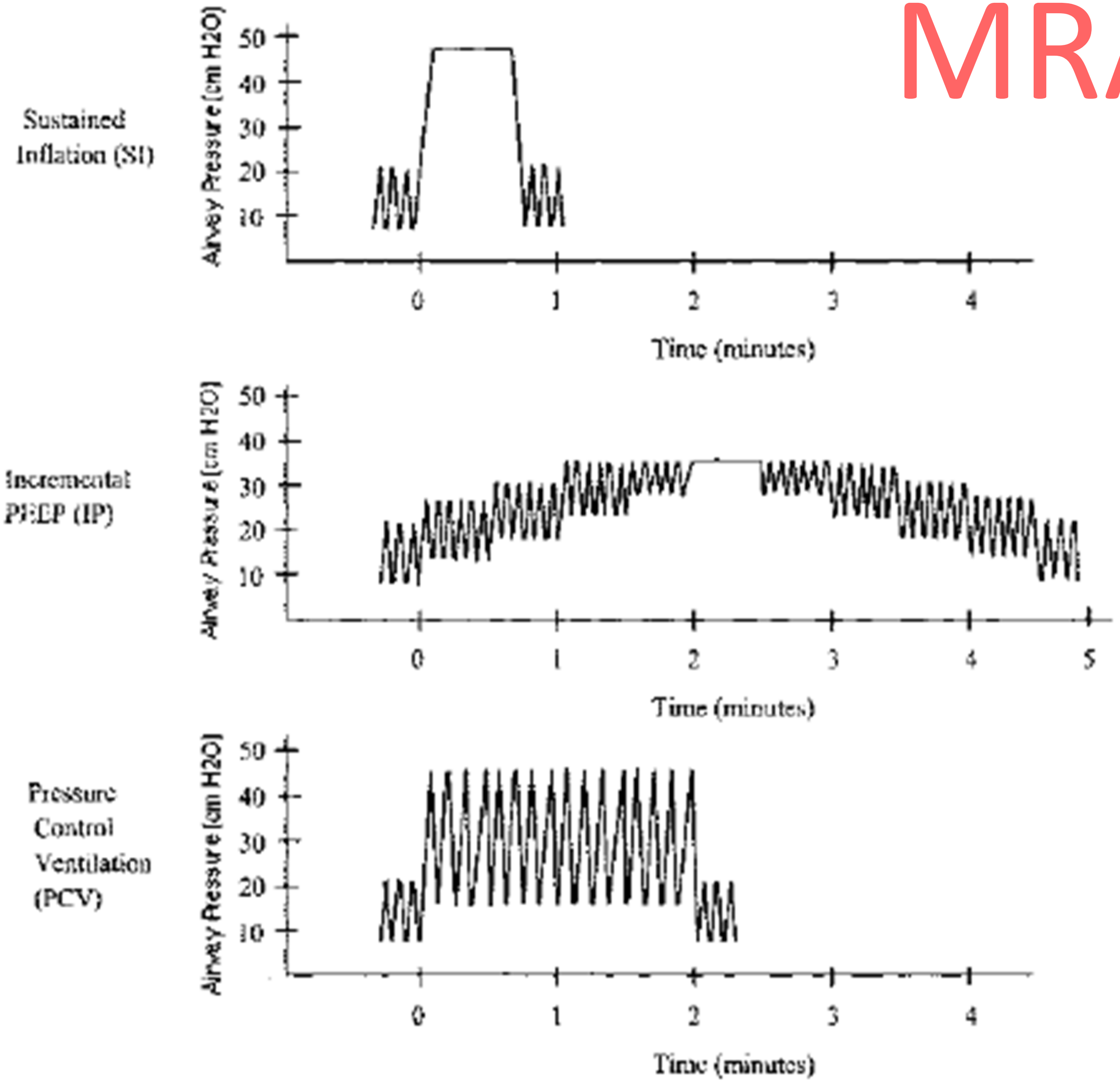
Sur distension

Haut VT
Haute Pmotrice

Atelectotrauma
Pre-VILI
Barotrauma
Volutrauma
Release of inflammatory cytokines
Release of inflammatory cytokines

Inflammation pulmonaire

MRA

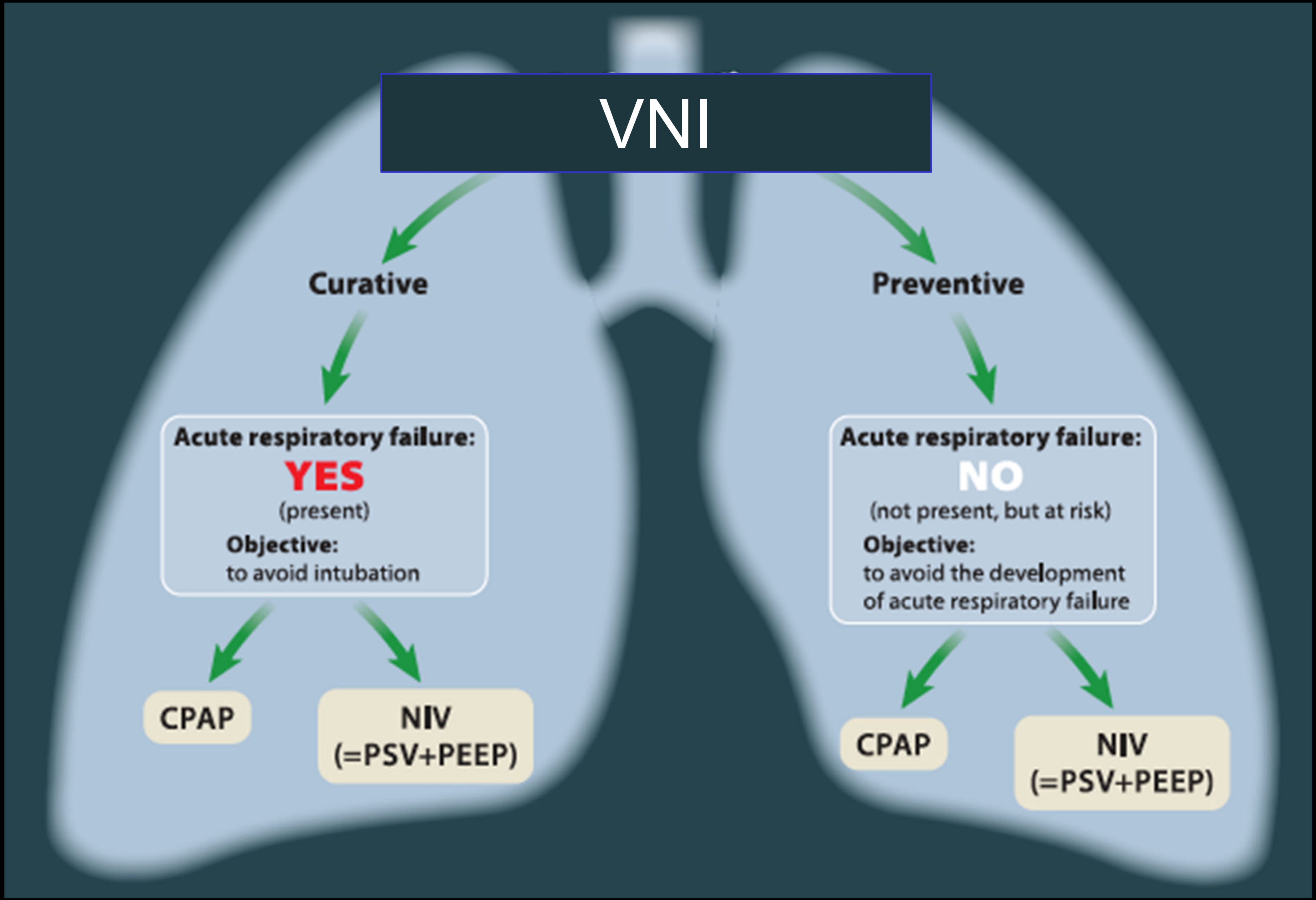

Sustained Inflation (SI)
Incremental PEEP (IP)
Pressure Control Ventilation (PCV)
Airway Pressure (cm H2O)
50
40
30
20
10
0
1
2
3
4
5
Time (minutes)

Temps 40 secondes

Bénéfices/Risques

- Bénéfices:
  - Augmenter le volume pulmonaire aéré
  - Améliorer l’oxygénation
- Risques:
  - Surdistension
  - Barotraumatisme
  - Dégradation hémodynamique

VNI



Curative

Acute respiratory failure:
YES
(present)
Objective:
to avoid intubation

CPAP

NIV
(=PSV+PEEP)

Preventive

Acute respiratory failure:
NO
(not present, but at risk)
Objective:
to avoid the development of acute respiratory failure

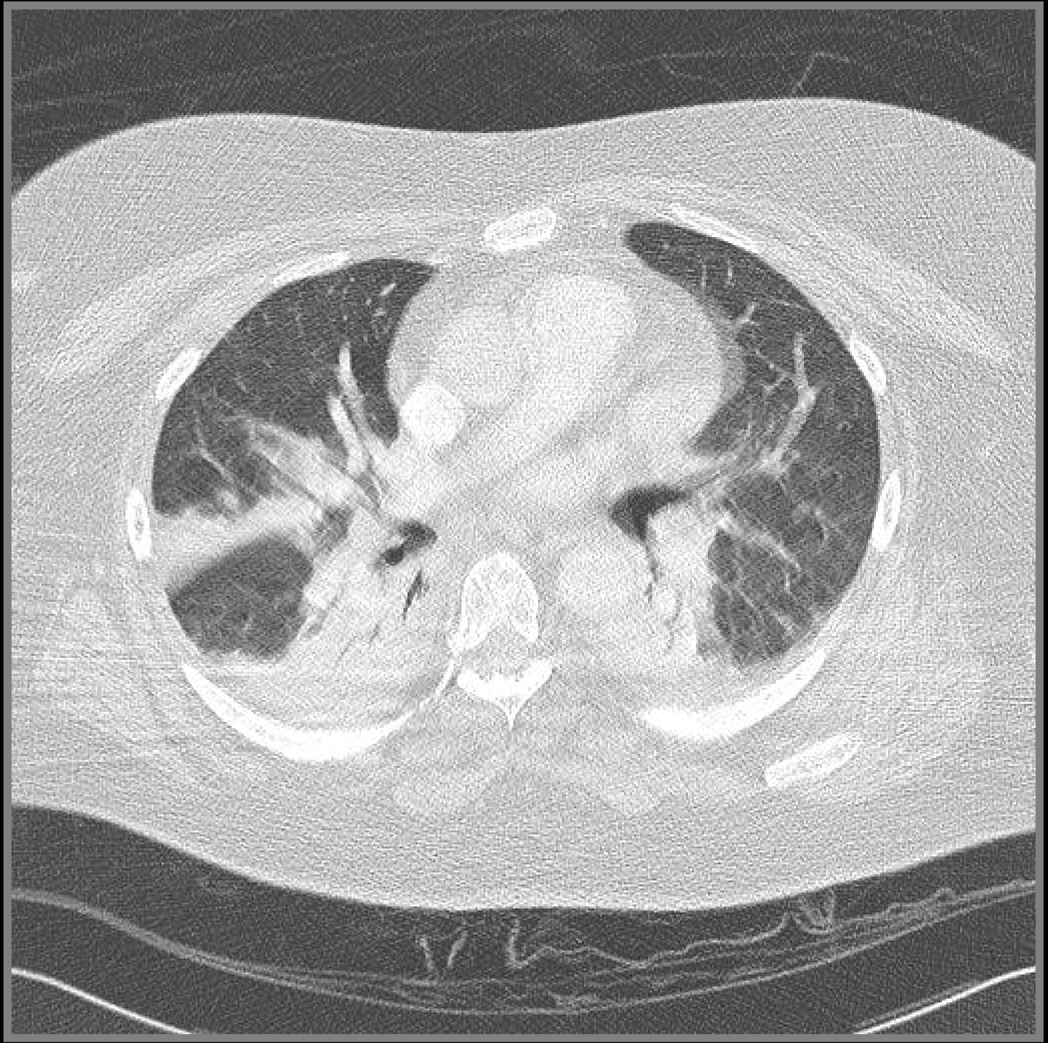
CPAP

NIV
(=PSV+PEEP)

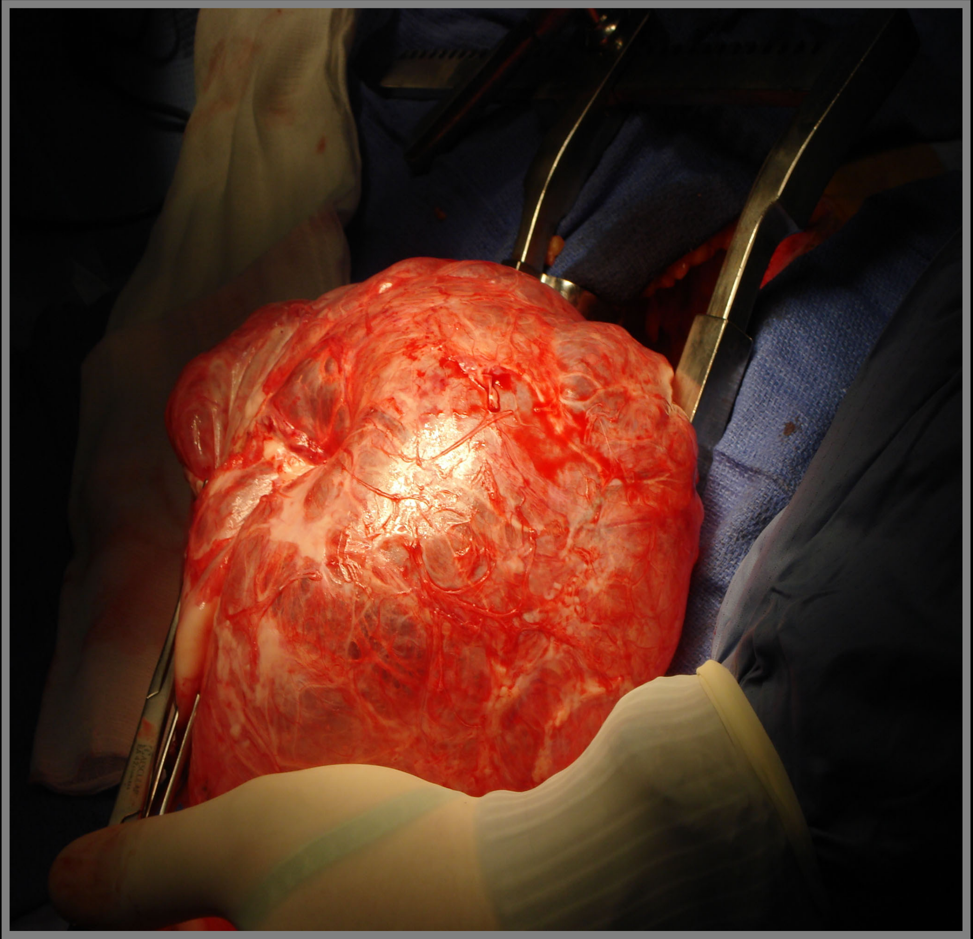
Anesthesiology 2010; 112:453-61

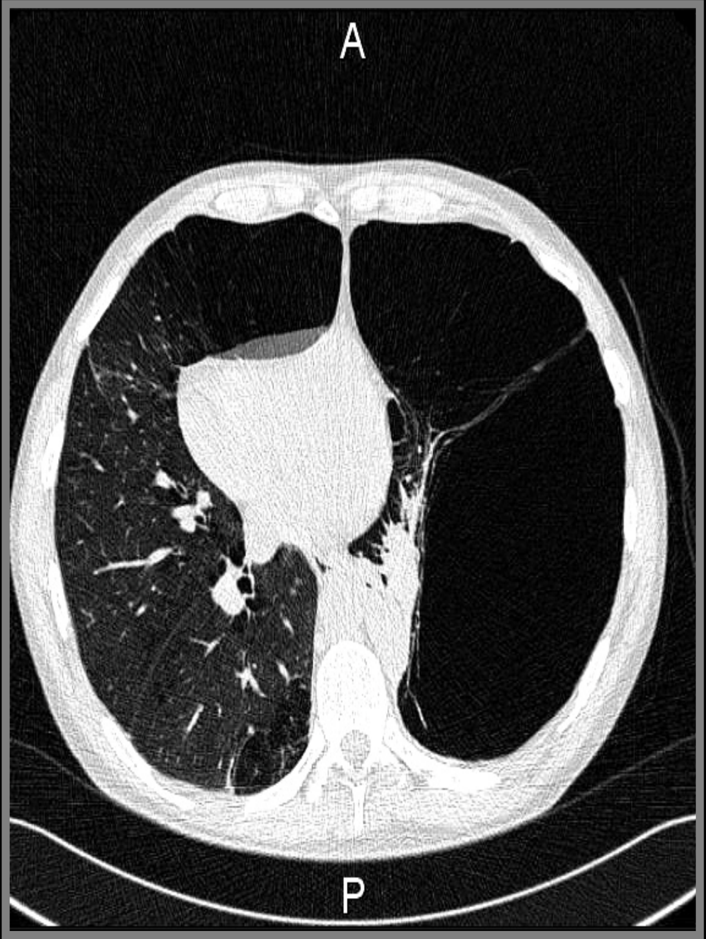
Atelectasies de l’obésité

J2 Post Op
Anneau
Gastrique
coelio

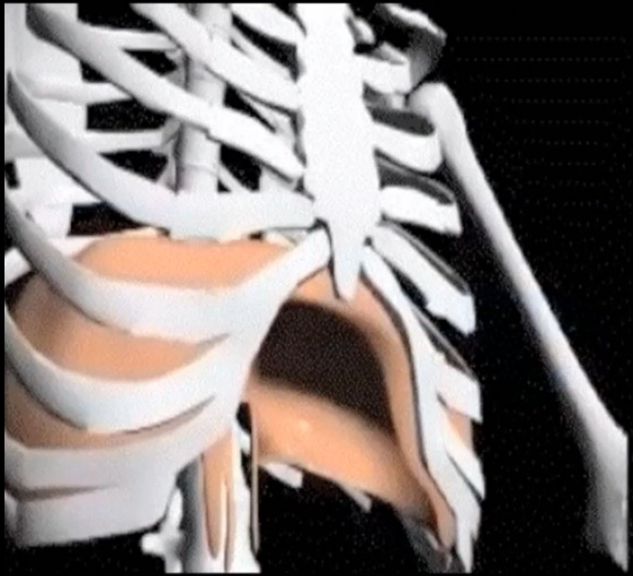


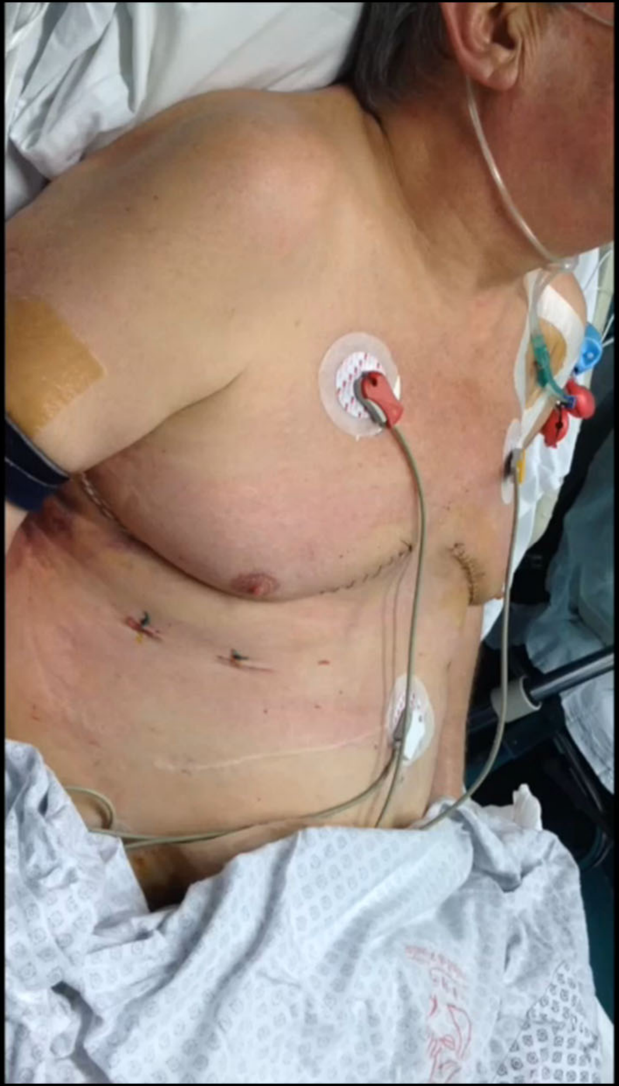
Chirurgie l’emphysème





Dysfonction Diaphragmatique



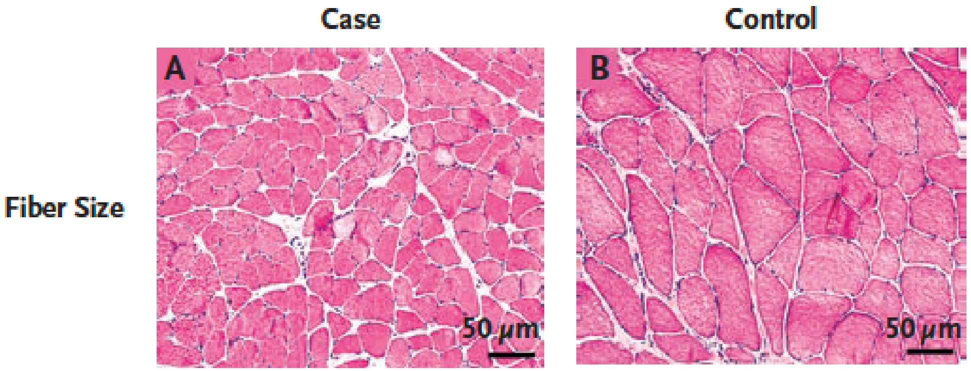


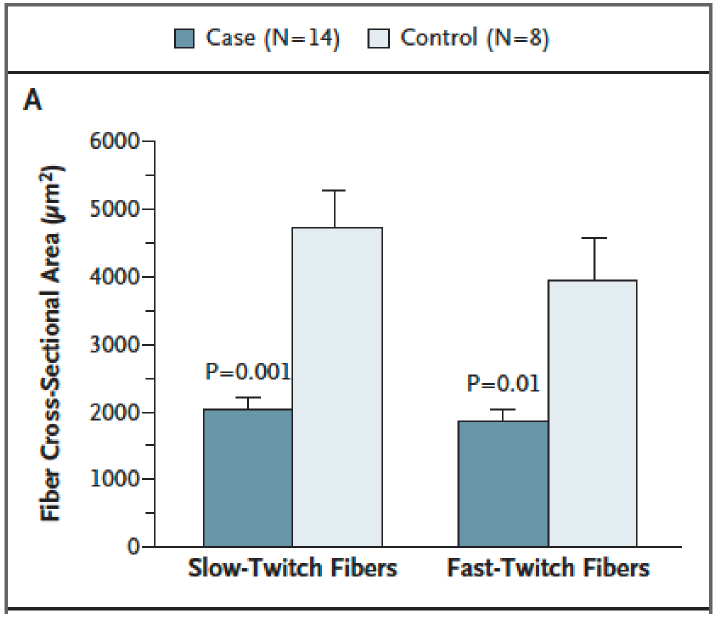
The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MARCH 27, 2008 VOL. 358 NO. 13

Rapid Disuse Atrophy of Diaphragm Fibers in Mechanically Ventilated Humans

Sanford Levine, M.D., Taitan Nguyen, B.S.E., Nyali Taylor, M.D., M.P.H., Michael E. Friscia, M.D., Murat T. Budak, M.D., Ph.D., Pamela Rothenberg, B.A., Jianliang Zhu, M.D., Rajeev Sachdeva, M.D., Seema Sonnad, Ph.D., Larry R. Kaiser, M.D., Neal A. Rubinstein, M.D., Ph.D., Scott K. Powers, Ph.D., Ed.D., and Joseph B. Shrager, M.D.

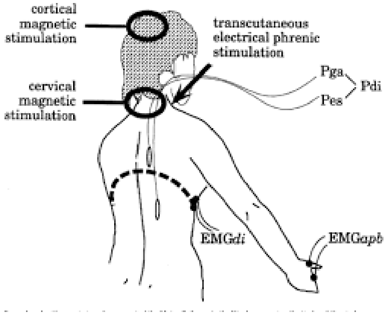


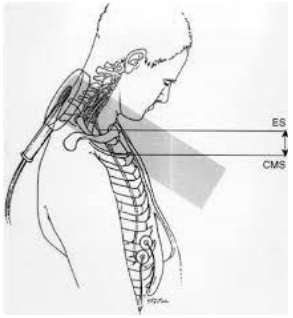


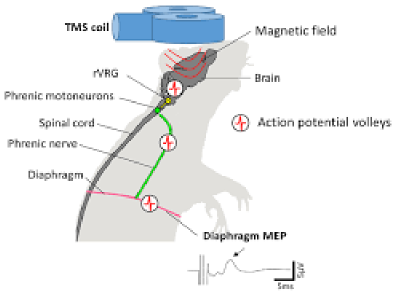
Diaphragm Dysfunction on Admission to the Intensive Care Unit

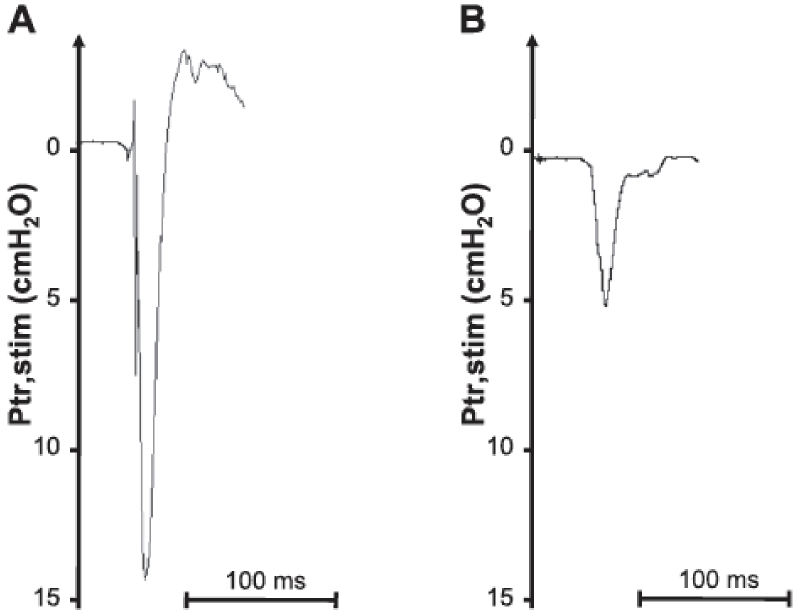
Prevalence, Risk Factors, and Prognostic Impact—A Prospective Study

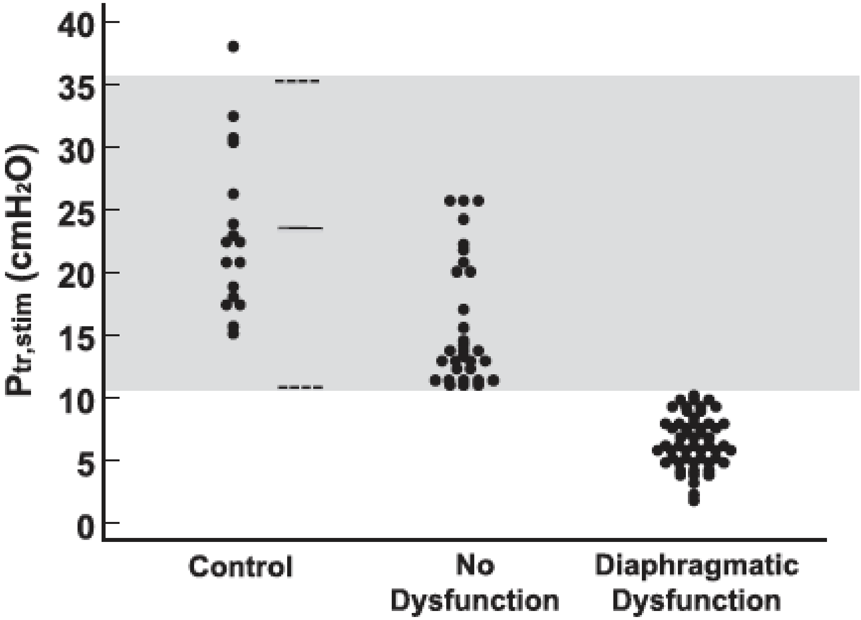
Alexandre Demoule[1,2,3], Boris Jung[4,5], Hélène Prodanovic[2], Nicolas Molinari[6], Gerald Chanques[4,5], Catherine Coirault[3], Stefan Matecki[5,7], Alexandre Duguet[1,2], Thomas Similowski[1,2*], and Samir Jaber[4,5*]


cortical magnetic stimulation
transcutaneous electrical phrenic stimulation
Pga
Pdi
Pes
cervical magnetic stimulation
EMGdi
EMGapb



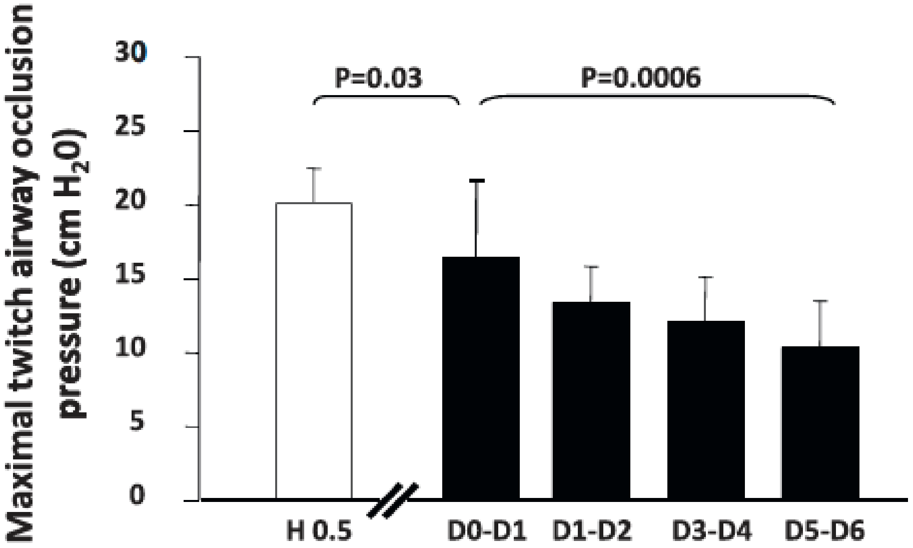

TMS coil
Magnetic field
rVRG
Brain
Phrenic motoneurons
Action potential volleys
Spinal cord
Phrenic nerve
Diaphragm
Diaphragm MEP

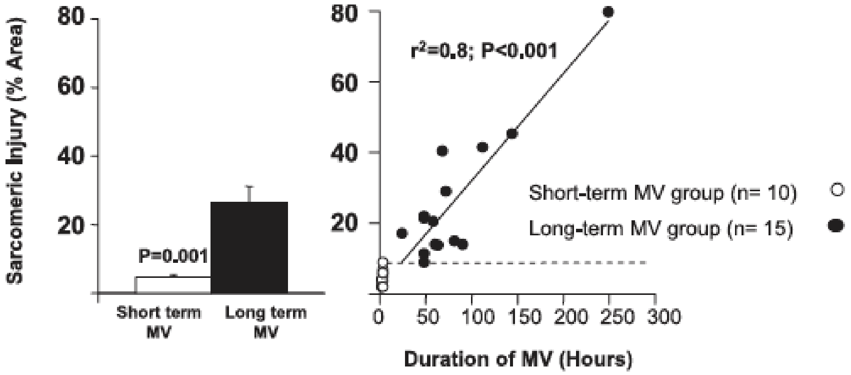




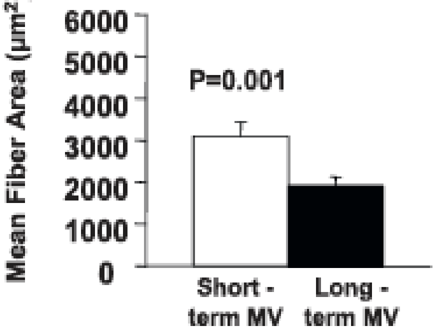
Rapidly Progressive Diaphragmatic Weakness and Injury during Mechanical Ventilation in Humans

Samir Jaber[1,2,6], Basil J. Petrof[3], Boris Jung[1,2], Gérald Chanques[1,2], Jean-Philippe Berthet[4], Christophe Rabuel[5], Hassan Bouyabrine[6], Patricia Courouble[1,2], Christelle Koechlin-Ramonatxo[7], Mustapha Sebbane[1,2], Thomas Similowski[8], Valérie Scheuermann[9], Alexandre Mebazaa[5], Xavier Capdevila[1,2], Dominique Mornet[2], Jacques Mercier[2,10], Alain Lacampagne[9], Alexandre Philips[2], and Stefan Matecki[2,10]

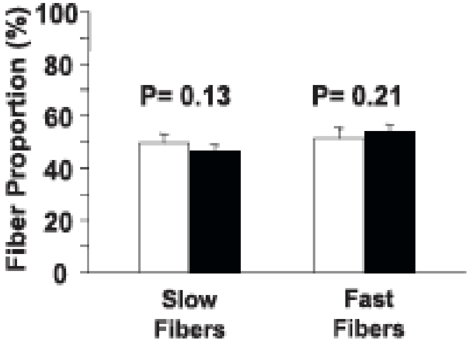




G

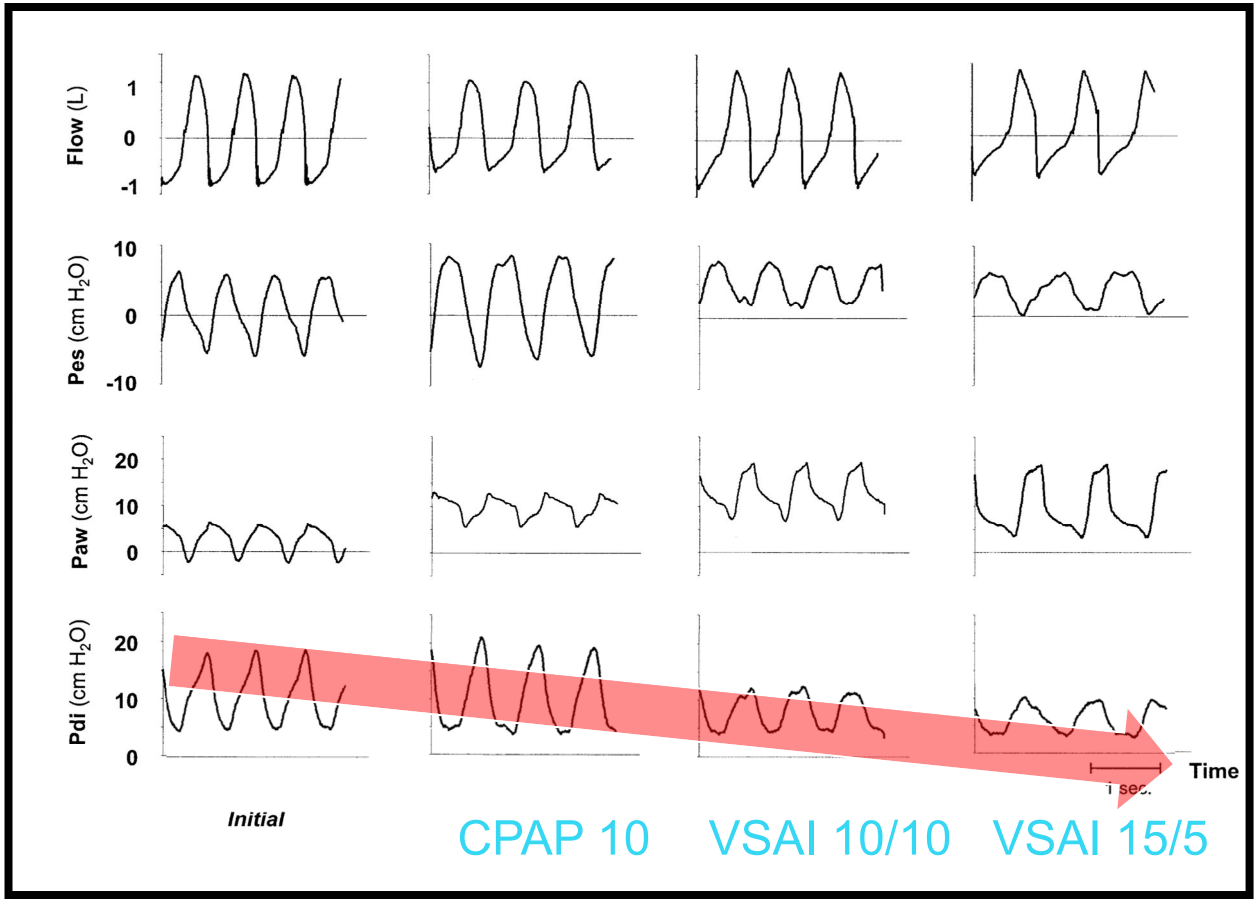


H



Am J Respir Crit Care Med Vol 183. pp 364–371, 2011

CPAP vs VSAI: (PEP ou PEP+Aide)



Pression

Effort

AJRCCM (2005) 172:1112

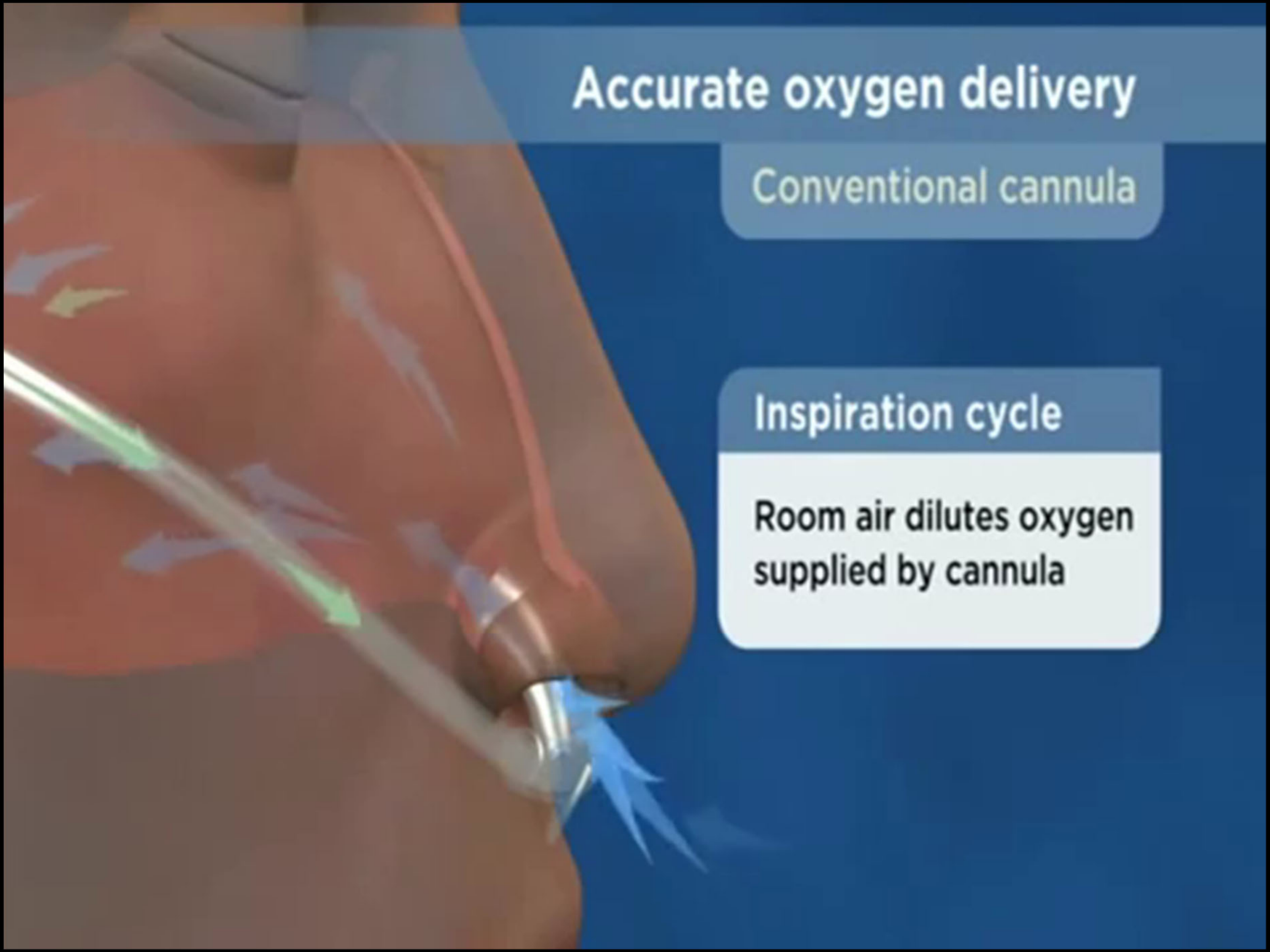
	CON Group (n = 22)	NPPV Group (n = 22)	NPPV+RM Group (n = 22)	Overall Group Effect
P_{max}, cm H_2O	35 [32–39]	32 [29–36]	29 [28–34]*	$P = 0.03$
[redacted]	[redacted]	[redacted]	[redacted]	$P < 0.001$
E,rs, cm H_2O ml^{-1}	34 [27–39]	20 [17–25]*	13 [11–20]*†	$P < 0.001$
VTe, ml	485 [430–546]	458 [420–550]	475 [425–550]	$P = 0.94$
RR, c min^{-1}	20 [18–20]	20 [20–20]	20 [20–21]	$P = 0.15$

Noninvasive Ventilation and Alveolar Recruitment Maneuver Improve Respiratory Function during and after Intubation of Morbidly Obese Patients

A Randomized Controlled Study

Emmanuel Futier, M.D.,* Jean-Michel Constantin, M.D., Ph.D.,† Paolo Pelosi, M.D., Ph.D.,‡
Gerald Chanques, M.D., Ph.D.,§ Alexandre Massone, M.D.,‖ Antoine Petit, M.D.,#
Fabrice Kwiatkowski, Ph.D.,** Jean-Etienne Bazin, M.D., Ph.D.,†† Samir Jaber, M.D., Ph.D.‡‡

Anesthesiology 2011; 114:1354–63



Accurate oxygen delivery

Conventional cannula

Inspiration cycle

Room air dilutes oxygen supplied by cannula

Oxygénation à haut débit

J Appl Physiol 132: 1580–1590, 2022.
First published May 5, 2022; doi:10.1152/japplphysiol.00416.2021

american physiological society
JOURNAL OF APPLIED PHYSIOLOGY.

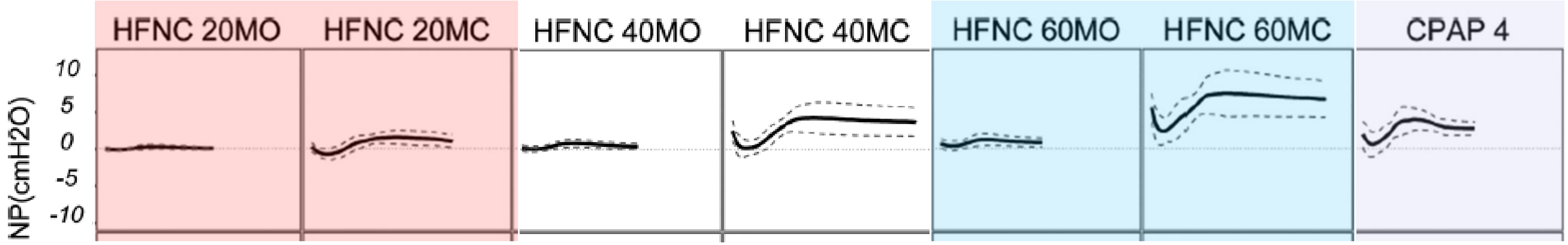
RESEARCH ARTICLE

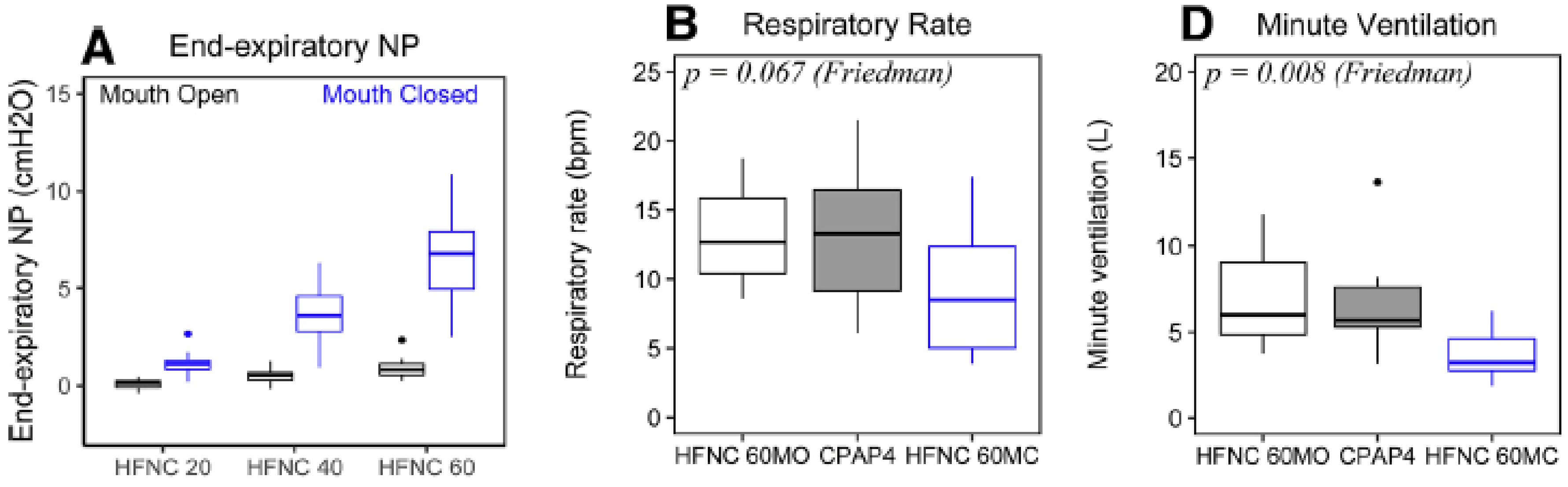
High-flow nasal cannula compared with continuous positive airway pressure: a bench and physiological study

Fernando Vieira,[1,2] Frank Silva Bezerra,[1,2,4] Remi Coudroy,[1,2,6,7] Annia Schreiber,[1,2] Irene Telias,[1,2,3] Sebastián Dubo,[1,8] Giulia Cavalot,[1,2,5] Sergio Martins Pereira,[1,2,9,10] Thomas Piraino,[1,2] and Laurent Jean Brochard[1,2]

Healthy volunteer study

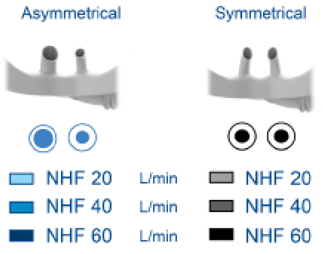
HFNC
vs.
CPAP

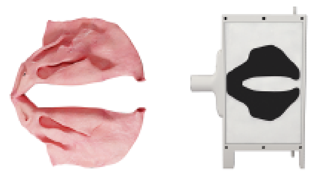


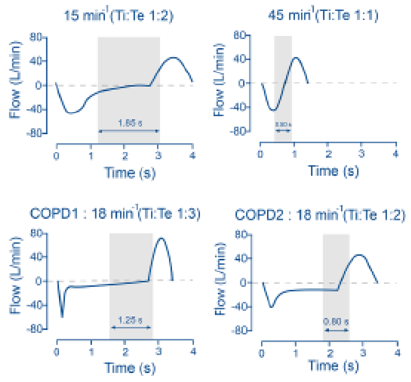


Asymmetrical nasal high flow ventilation improves clearance of CO_2 from the anatomical dead space and increases positive airway pressure

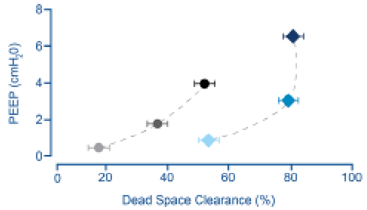
Methods

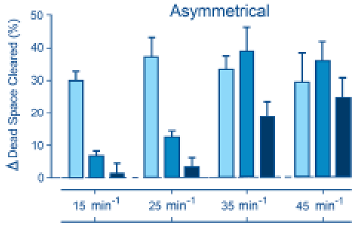

Asymmetrical
Symmetrical
NHF 20 L/min
NHF 40 L/min
NHF 60 L/min
NHF 20
NHF 40
NHF 60

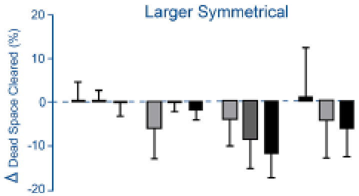


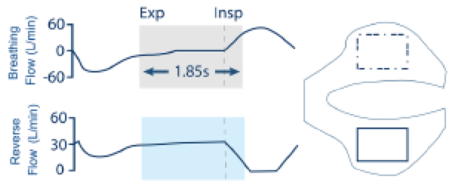


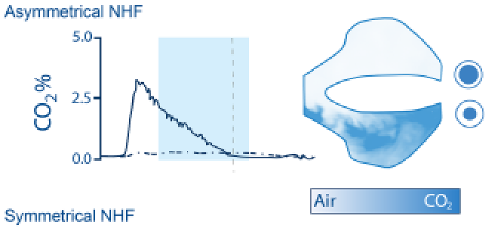
Results

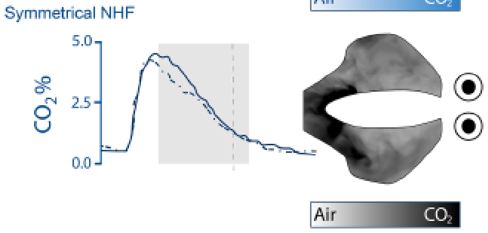










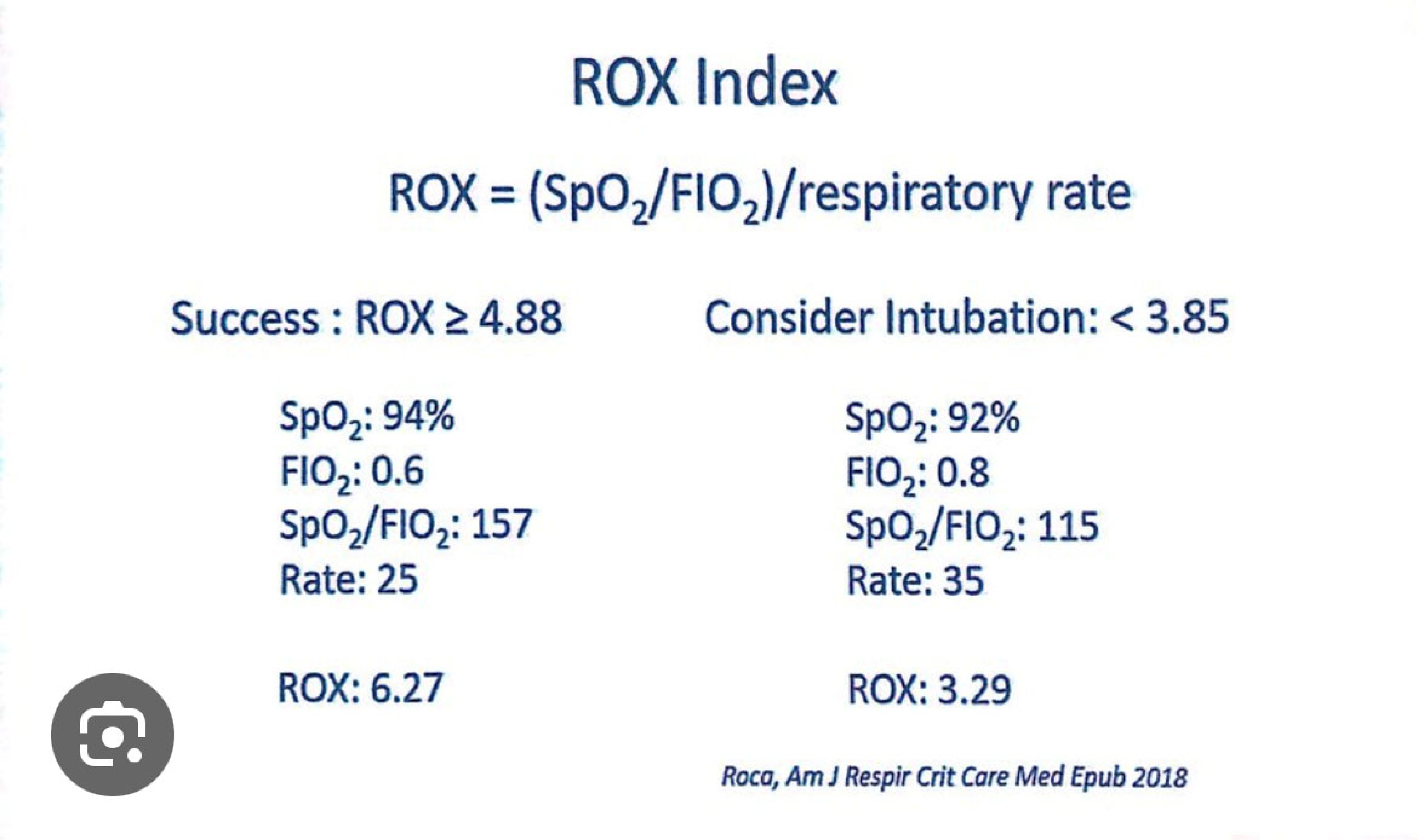


Conclusion

Asymmetrical NHF increases dead space clearance by reverse flow through the choanae and accelerates purging of expired gas via the less occluded nare. Larger asymmetrical interface may increase airway pressure and reduce risk of a complete occlusion.

JOURNAL OF APPLIED PHYSIOLOGY. © 2023

american physiological society



ROX Index

ROX = (SpO_2/FIO_2)/respiratory rate

Success : ROX ≥ 4.88	Consider Intubation: < 3.85
SpO_2: 94%	SpO_2: 92%
FIO_2: 0.6	FIO_2: 0.8
SpO_2/FIO_2: 157	SpO_2/FIO_2: 115
Rate: 25	Rate: 35
ROX: 6.27	ROX: 3.29

Roca, Am J Respir Crit Care Med Epub 2018

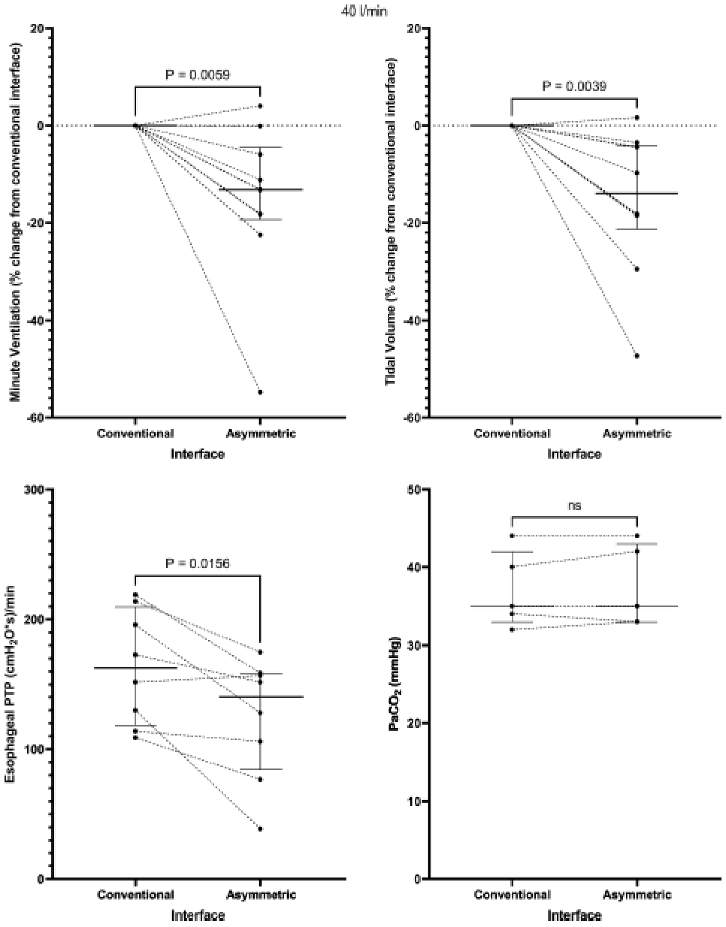
Slobod *et al. Critical Care* (2023) 27:145
https://doi.org/10.1186/s13054-023-04441-6

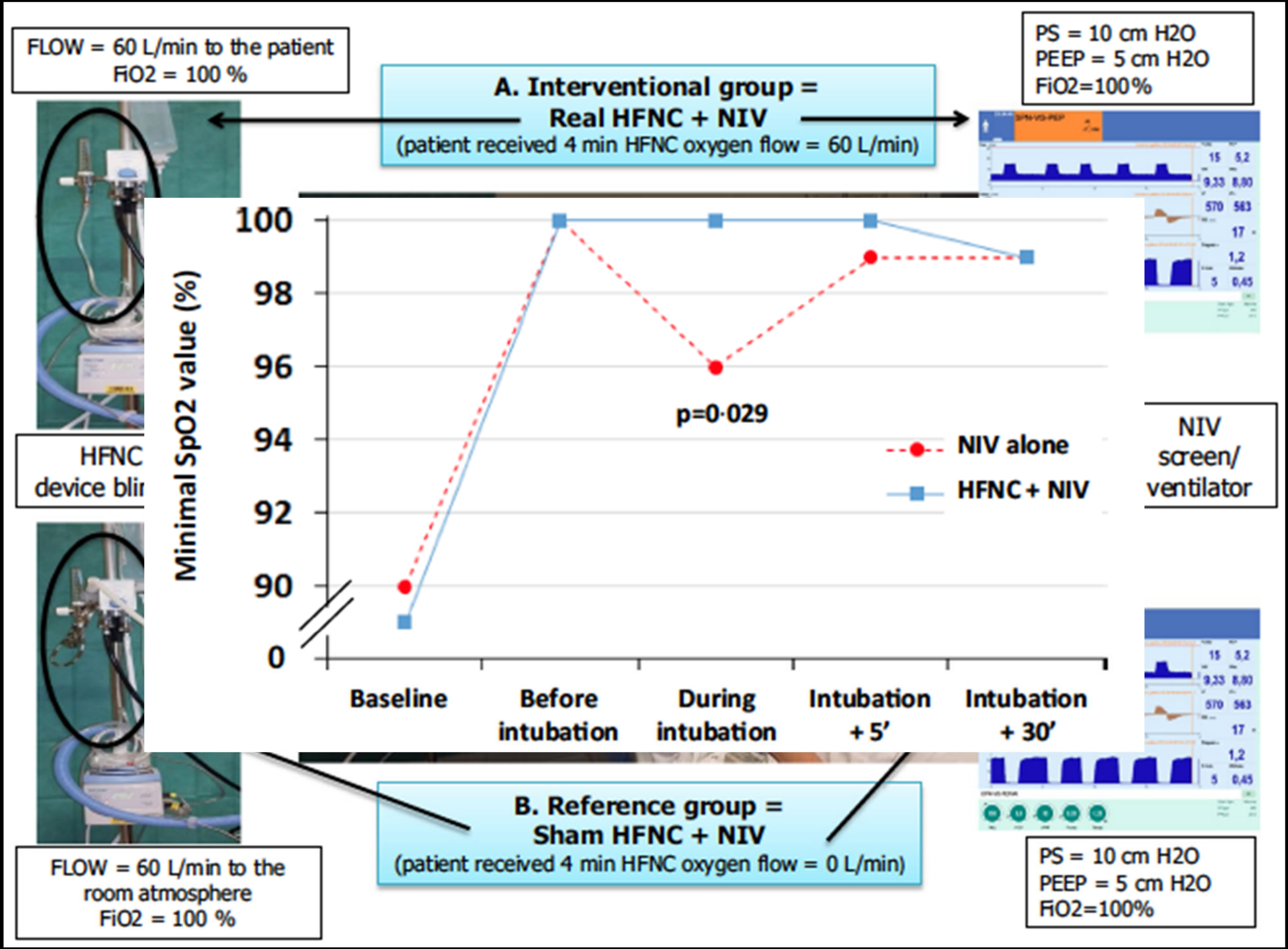
Critical Care

RESEARCH **Open Access**

Effects of an asymmetrical high flow nasal cannula interface in hypoxemic patients

Douglas Slobod[1,2], Elena Spinelli[1], Stefania Crotti[1], Alfredo Lissoni[1], Alessandro Galazzi[3], Giacomo Grasselli[1,4] and Tommaso Mauri[1,4*]





FLOW = 60 L/min to the patient
FiO2 = 100 %

A. Interventional group =
Real HFNC + NIV
(patient received 4 min HFNC oxygen flow = 60 L/min)

PS = 10 cm H2O
PEEP = 5 cm H2O
FiO2=100%

HFNC
device blir

NIV
screen/
ventilator

FLOW = 60 L/min to the
room atmosphere
FiO2 = 100 %

B. Reference group =
Sham HFNC + NIV
(patient received 4 min HFNC oxygen flow = 0 L/min)

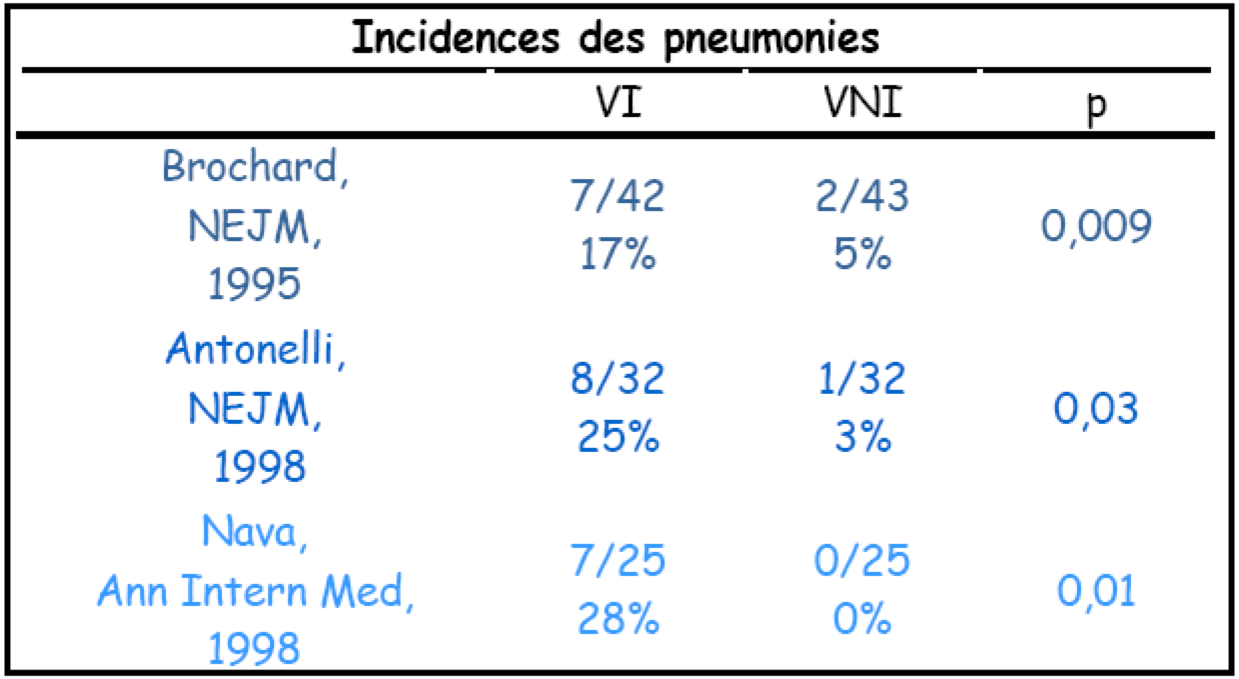
PS = 10 cm H2O
PEEP = 5 cm H2O
FiO2=100%

Intensive Care Med (2016) 42:1877–1887

Eviter l' intubation et ses complications (PAVM)

- VNI curative du BPCO:

diminuer le W et l'acidose hypercapnique

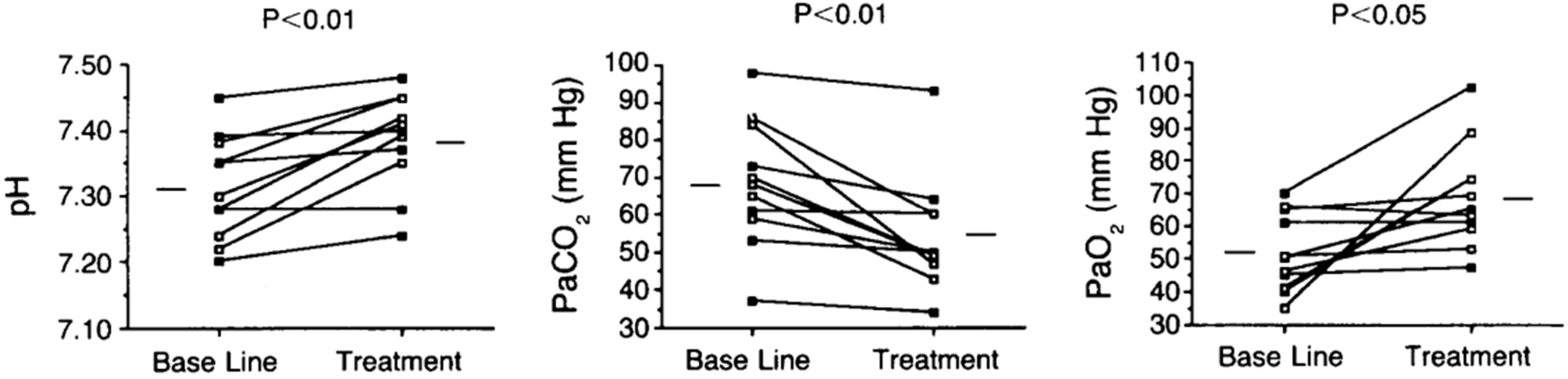


Incidences des pneumonies

	VI	VNI	p
Brochard, NEJM, 1995	7/42 17%	2/43 5%	0,009
Antonelli, NEJM, 1998	8/32 25%	1/32 3%	0,03
Nava, Ann Intern Med, 1998	7/25 28%	0/25 0%	0,01

The NEW ENGLAND JOURNAL of MEDICINE

Reversal of acute exacerbation of chronic obstructive lung disease by inspiratory assistance with a face mask



Patient No.	Age (yr)/Sex	FEV_1	FEV_1/FVC	Respiratory Rate	PaO_2	$PaCO_2$	pH
9	73/M	0.45	24	35	41	68	7.35
10	53/M	0.62	31	30	40	66	7.37
11	62/M	0.75	34	22	53	59	7.38

Brochard et al. **NEJM 1990**;323:1523-30

The NEW ENGLAND
JOURNAL of MEDICINE

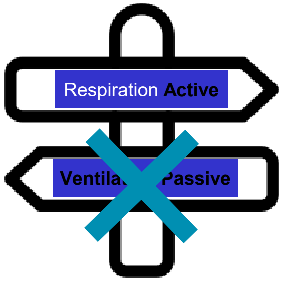
Noninvasive ventilation for acute exacerbation of chronic obstructive pulmonary disease.

85 patients BPCO VNI : masque facial, Aide inspiratoire

La **VNI** permet de **réduire :**

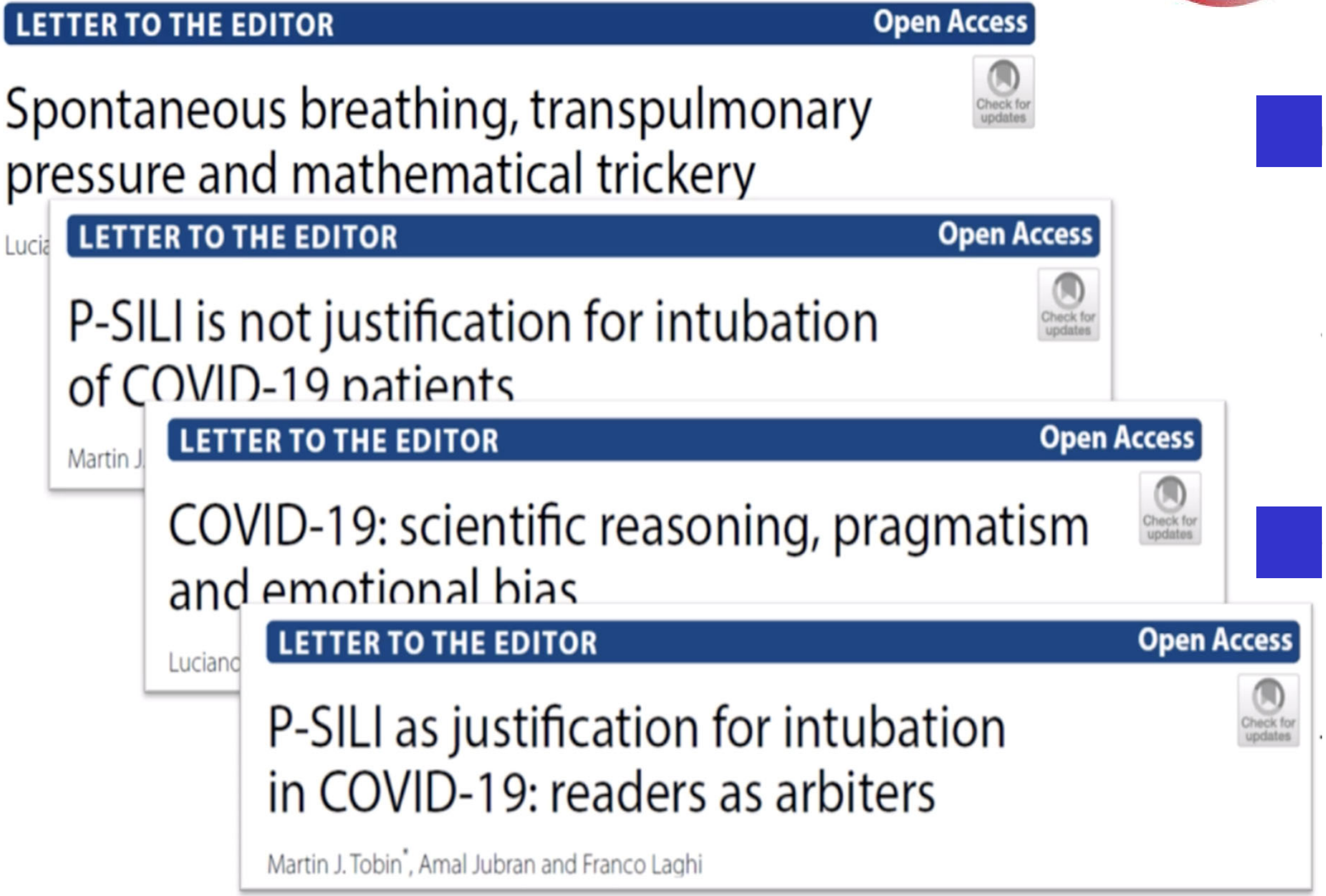
- l’ **intubation** de **74** à **26** %
- la **durée de séjour** hospitalier
- les **complications** en réanimation de 48 à 16 %
- la **mortalité** de **29** à **9** %

Brochard L, et al. **NEJM 1995**;333:817-822


Respiration Active
Ventila
Passive

Ventilation spontanée

Washington Post / YouTube



LETTER TO THE EDITOR Open Access

Spontaneous breathing, transpulmonary pressure and mathematical trickery

Lucia

LETTER TO THE EDITOR Open Access

P-SILI is not justification for intubation of COVID-19 patients

Martin J.

LETTER TO THE EDITOR Open Access

COVID-19: scientific reasoning, pragmatism and emotional bias

Luciano

LETTER TO THE EDITOR Open Access

P-SILI as justification for intubation in COVID-19: readers as arbiters

Martin J. Tobin*, Amal Jubran and Franco Laghi

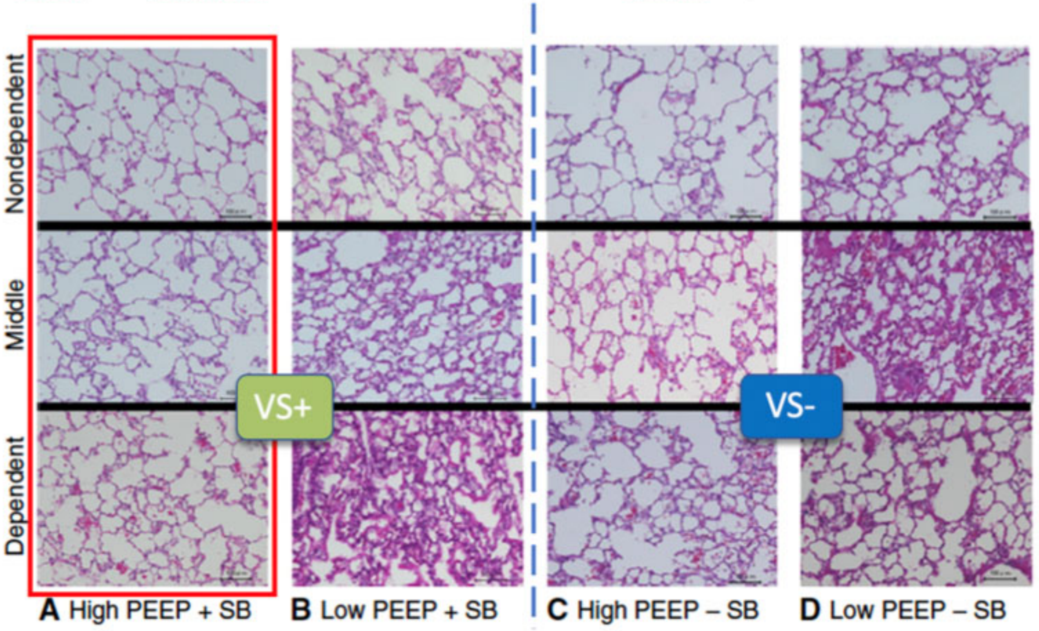
High Positive End-Expiratory Pressure Renders Spontaneous Effort Noninjurious

Caio C. A. Morais[1]*, Yukiko Koyama[2]*, Takeshi Yoshida[2,3]*, Glauco M. Plens[1], Susimeire Gomes[1],
Cristhiano A. S. Lima[1], Ozires P. S. Ramos[1], Sérgio M. Pereira[1], Naomasa Kawaguchi[4], Hirofumi Yamamoto[4],
Akinori Uchiyama[2], João B. Borges[5], Marcos F. Vidal Melo[6], Mauro R. Tucci[1], Marcelo B. P. Amato[1],
Brian P. Kavanagh[3], Eduardo L. V. Costa[1], and Yuji Fujino[2]

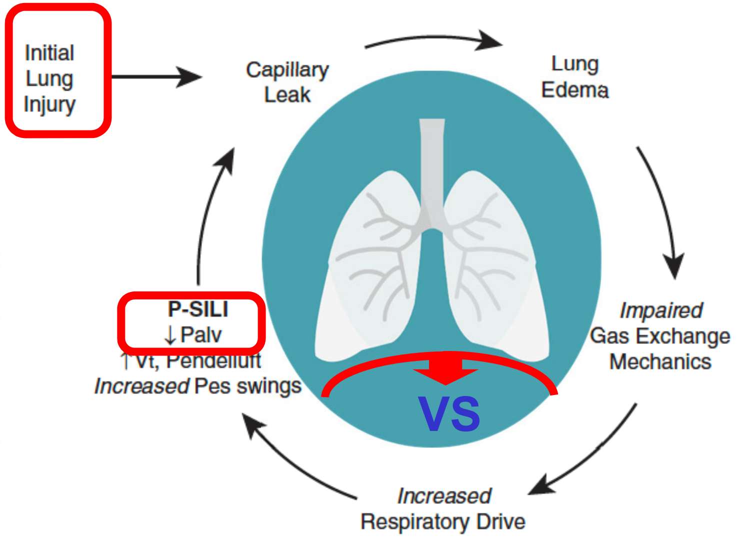
Am J Respir Crit Care Med Vol 197, Iss 10, pp 1285–1296, May 15, 2018

Palv = PEEP + VT/Crs – Pmus

Palv plus negative si moins de PEEP et/ou si Pmus augmente


Nondependent
Middle
Dependent
VS+
VS-
A High PEEP + SB
B Low PEEP + SB
C High PEEP – SB
D Low PEEP – SB

Plus homogène


Initial
Lung
Injury
Capillary
Leak
Lung
Edema
Impaired
Gas Exchange
Mechanics
Increased
Respiratory Drive
P-SILI
↓Palv
↑Vt, Pendelluft
Increased Pes swings
VS

Grace à la PEEP, la VNI et la CPAP évitent une pression alvéolaire trop négative (PSiLi)

JAMA | Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

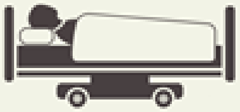
Effect of Helmet Noninvasive Ventilation vs High-Flow Nasal Oxygen on Days Free of Respiratory Support in Patients With COVID-19 and Moderate to Severe Hypoxemic Respiratory Failure

The HENIVOT Randomized Clinical Trial

Domenico Luca Grieco, MD; Luca S. Menga, MD; Melania Cesarano, MD; Tommaso Rosà, MD; Savino Spadaro, MD, PhD; Maria Maddalena Bitondo, MD;
Jonathan Montomoli, MD, PhD; Giulia Falò, MD; Tommaso Tonetti, MD; Salvatore L. Cutuli, MD; Gabriele Pintaudi, MD; Eloisa S. Tanzarella, MD;
Edoardo Piervincenzi, MD; Filippo Bongiovanni, MD; Antonio M. Dell'Anna, MD; Luca Delle Cese, MD; Cecilia Berardi, MD; Simone Carelli, MD;
Maria Grazia Bocci, MD; Luca Montini, MD; Giuseppe Bello, MD; Daniele Natalini, MD; Gennaro De Pascale, MD; Matteo Velardo, PhD;
Carlo Alberto Volta, MD; V. Marco Ranieri, MD; Giorgio Conti, MD; Salvatore Maurizio Maggiore, MD, PhD; Massimo Antonelli, MD;
for the COVID-ICU Gemelli Study Group

POPULATION

88 Men
21 Women

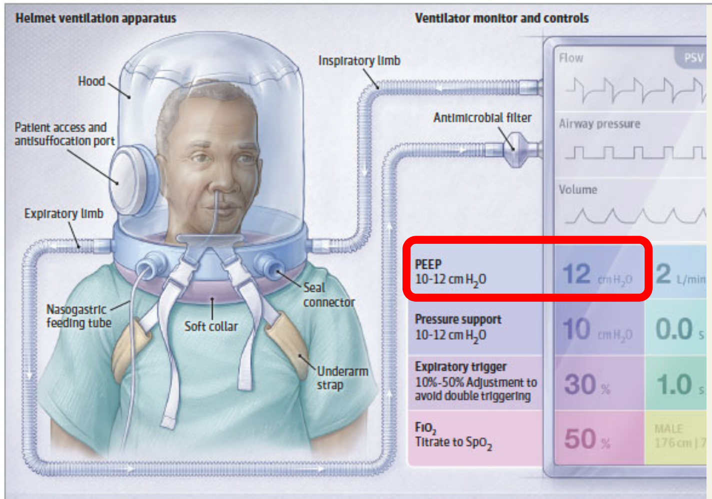


Adults in the intensive care unit with COVID-19 and moderate to severe hypoxemic respiratory failure

Median age: 65 years

LOCATIONS

4
ICUs in Italy


Helmet ventilation apparatus
Ventilator monitor and controls
Hood
Inspiratory limb
Patient access and antisuffocation port
Antimicrobial filter
Expiratory limb
Nasogastric feeding tube
Soft collar
Seal connector
Underarm strap
Flow
PSV
Airway pressure
Volume
PEEP
10-12 cm H_2O
12 cm H_2O
2 L/min
Pressure support
10-12 cm H_2O
10 cm H_2O
0.0 s
Expiratory trigger
10%-50% Adjustment to avoid double triggering
30 %
1.0 s
FIO_2
Titrate to SpO_2
50 %
MALE
176 cm | 7

Patient monitoring protocol

Every 1-3 hrs
- PaO_2/FIO_2 ratio
- Respiratory rate
- Signs of respiratory muscle fatigue
- $PaCO_2$
- Dyspnea

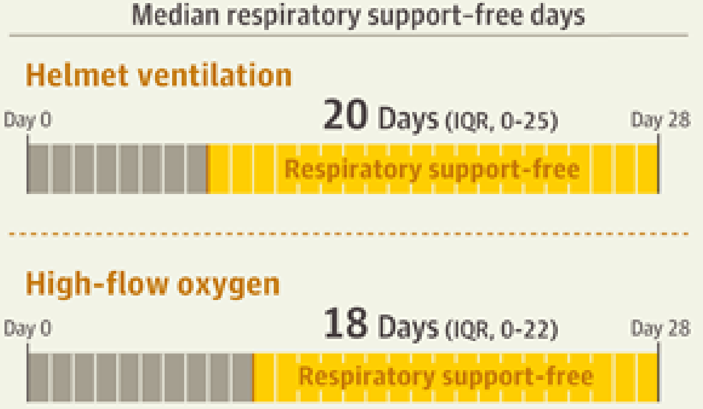
Continuous
- SpO_2
- Blood pressure
- Heart rate

When to consider intubation

- Worsening or unbearable dyspnea
- Lack of improvement in oxygenation
- Hemodynamic instability
- Persisten
- Persisten
- Neurolog

© AMA

FINDINGS



Mean between-group difference was not statistically significant:

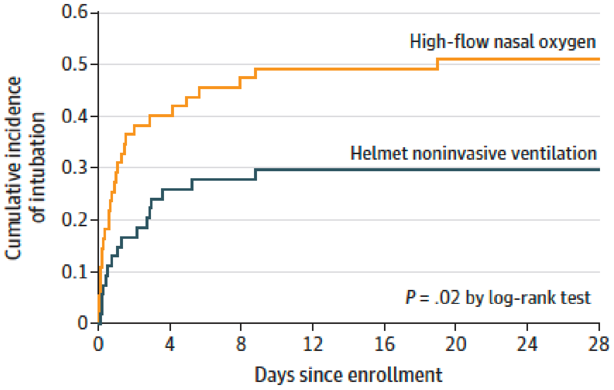
2 days (95% CI, −2 to 6)

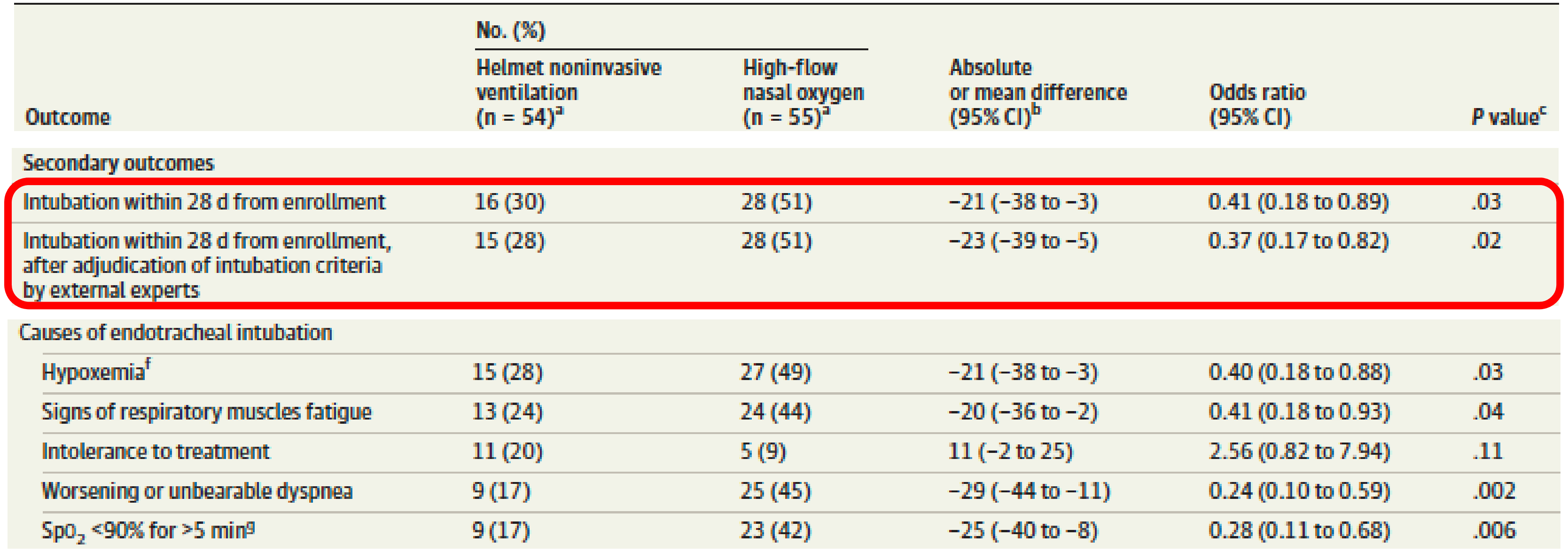
JAMA | Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

Effect of Helmet Noninvasive Ventilation vs High-Flow Nasal Oxygen on Days Free of Respiratory Support in Patients With COVID-19 and Moderate to Severe Hypoxemic Respiratory Failure

The HENIVOT Randomized Clinical Trial

Domenico Luca Grieco, MD; Luca S. Menga, MD; Melania Cesarano, MD; Tommaso Rosà, MD; Savino Spadaro, MD, PhD; Maria Maddalena Bitoni
Jonathan Montomoli, MD, PhD; Giulia Falò, MD; Tommaso Tonetti, MD; Salvatore L. Cutuli, MD; Gabriele Pintaudi, MD; Eloisa S. Tanzarella, MD;
Edoardo Piervincenzi, MD; Filippo Bongiovanni, MD; Antonio M. Dell'Anna, MD; Luca Delle Cese, MD; Cecilia Berardi, MD; Simone Carelli, MD;
Maria Grazia Bocci, MD; Luca Montini, MD; Giuseppe Bello, MD; Daniele Natalini, MD; Gennaro De Pascale, MD; Matteo Velardo, PhD;
Carlo Alberto Volta, MD; V. Marco Ranieri, MD; Giorgio Conti, MD; Salvatore Maurizio Maggiore, MD, PhD; Massimo Antonelli, MD;
for the COVID-ICU Gemelli Study Group





Outcome	No. (%) Helmet noninvasive ventilation (n = 54)[a]	No. (%) High-flow nasal oxygen (n = 55)[a]	Absolute or mean difference (95% CI)[b]	Odds ratio (95% CI)	P value[c]
Secondary outcomes					
Intubation within 28 d from enrollment	16 (30)	28 (51)	−21 (−38 to −3)	0.41 (0.18 to 0.89)	.03
Intubation within 28 d from enrollment, after adjudication of intubation criteria by external experts	15 (28)	28 (51)	−23 (−39 to −5)	0.37 (0.17 to 0.82)	.02
Causes of endotracheal intubation					
Hypoxemia[f]	15 (28)	27 (49)	−21 (−38 to −3)	0.40 (0.18 to 0.88)	.03
Signs of respiratory muscles fatigue	13 (24)	24 (44)	−20 (−36 to −2)	0.41 (0.18 to 0.93)	.04
Intolerance to treatment	11 (20)	5 (9)	11 (−2 to 25)	2.56 (0.82 to 7.94)	.11
Worsening or unbearable dyspnea	9 (17)	25 (45)	−29 (−44 to −11)	0.24 (0.10 to 0.59)	.002
Sp_{O_2} <90% for >5 min[g]	9 (17)	23 (42)	−25 (−40 to −8)	0.28 (0.11 to 0.68)	.006

Plus de PEEP avec la casque
moins d'hypoxémies et moins d'intubations

Effect of Noninvasive Ventilation Delivered by Helmet vs Face Mask on the Rate of Endotracheal Intubation in Patients With Acute Respiratory Distress Syndrome

A Randomized Clinical Trial

Bhakti K. Patel, MD; Krysta S. Wolfe, MD; Anne S. Pohlman, MSN; Jesse B. Hall, MD; John P. Kress, MD

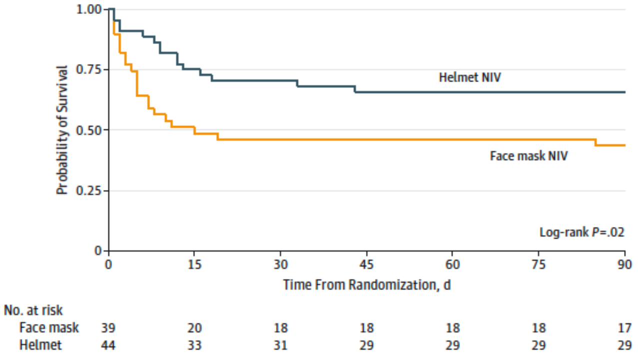


Table 2. Primary and Secondary Outcomes and Adverse Events

	Face Mask (n = 39)	Helmet (n = 44)	Absolute Difference (95% CI)	*P* Value
Primary outcome, No. (%)				
Endotracheal intubation	24 (61.5)	8 (18.2)	−43.3 (−62.4 to −24.3)	<.001
Reason for intubation				
Respiratory failure	20 (83.3)	3 (37.5)	−45.3 (−82.5 to −9.1)	.01
Circulatory failure	3 (12.5)	0 (0)	−12.5 (−25.7 to 0.7)	.55
Neurologic failure	1 (4.2)	5 (62.5)	58.3 (24.8 to 92.8)	.001
Secondary outcomes, median (IQR), d				
Ventilator-free days	12.5 (0.49-28)	28 (13.7-28)	8.4 (13.4 to 3.4)	<.001
ICU length of stay	7.8 (3.9-13.8)	4.7 (2.5-8.7)	−2.76 (−6.07 to 0.54)	.04
Hospital length of stay	15.2 (7.8-19.7)	10.1 (6.5-15.9)	−2.92 (−8.47 to 2.63)	.16
Mortality, No. (%)				
Hospital	19 (48.7)	12 (27.3)	−21.4 (−41.9 to −1.0)	.04
90 d[a]	22 (56.4)	15 (34.1)	−22.3 (−43.3 to −1.4)	.02
Adverse events				
Mask deflation	0 (0)	2 (4.5)		
Skin ulceration	3 (7.6)	3 (6.8)		

	Noninvasive Ventilation, Median (IQR)		
	Face Mask (n = 39)	Helmet (n = 44)	*P* Value
Respiratory support with NIV[a]			
Duration of NIV, h	26.4 (7.0-60.0)	19.8 (8.4-45.6)	.68
PEEP, cm H_2O	5.1 (5.0-8.0)	8 (5.0-10.0)	.006
Pressure support, cm H_2O	11.2 (10.0-14.5)	8 (5.6-10.0)	<.001
FIO_2, %	60 (50.0-68.6)	50 (40.0-60.0)	.02
SpO_2, %	95.3 (92.3-96.7)	96.2 (94.8-98.4)	.13
Respiratory rate, breaths/min			
Baseline	28.3 (22.1-34.4)[b]	27.7 (21.5-34.6)[b]	
After randomization	29.1 (22.1-37.6)	24.5 (20.4-30.5)	

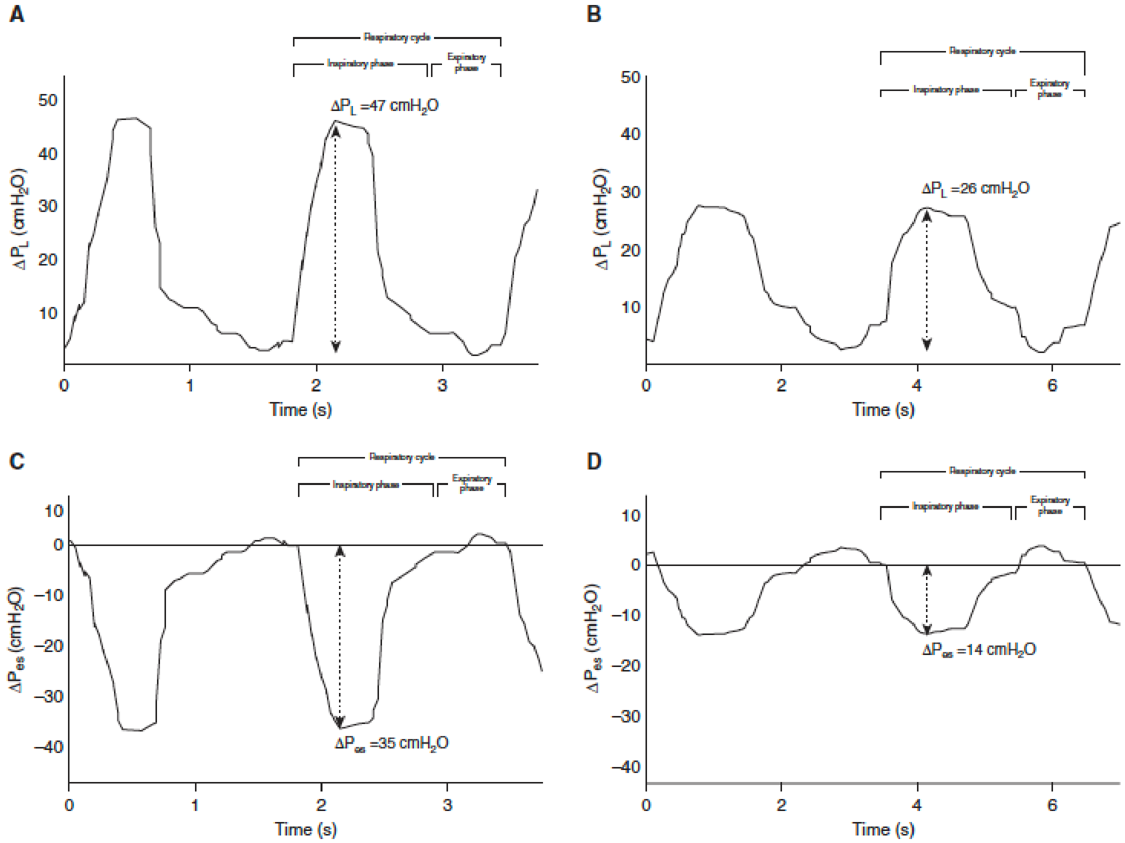
Plus de PEEP avec la casque évitent une pression alvéolaire trop négative (PSiLi)

JAMA. 2016;315(22):2435-2441.

Early Inspiratory Effort Assessment by Esophageal Manometry Predicts Noninvasive Ventilation Outcome in *De Novo* Respiratory Failure

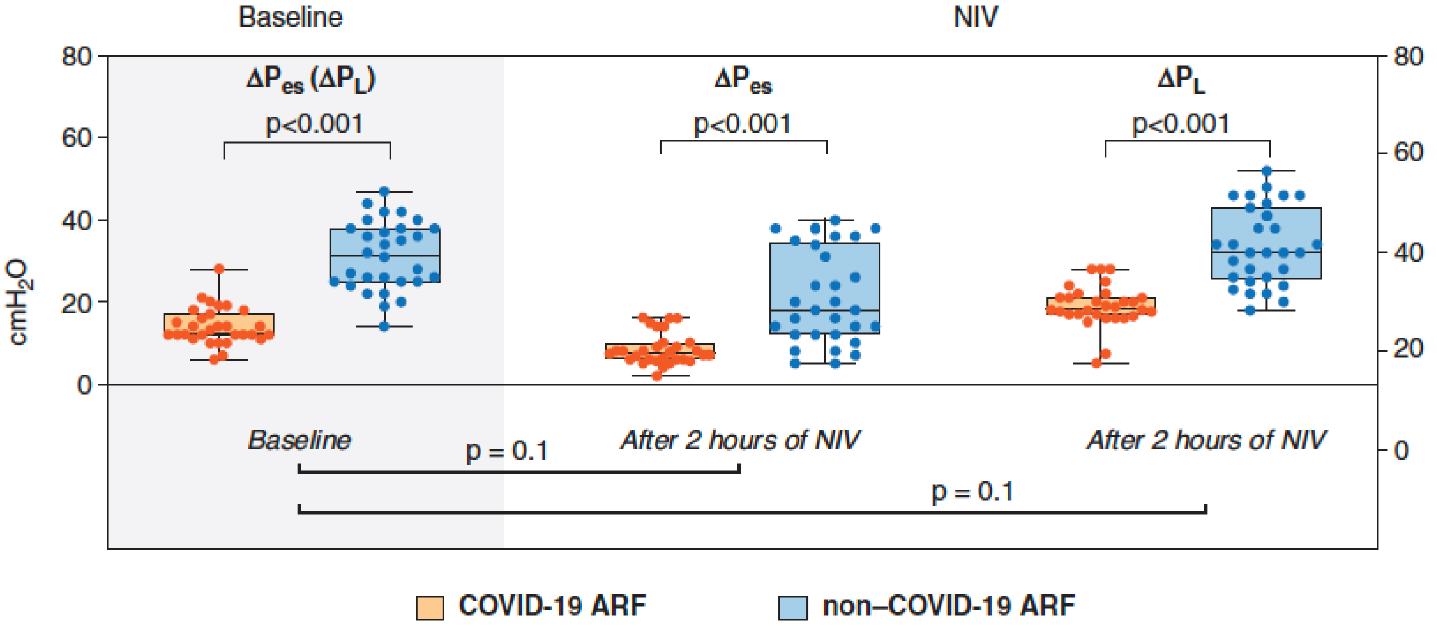
A Pilot Study

Roberto Tonelli[1,2], Riccardo Fantini[1], Luca Tabbì[1], Ivana Castaniere[1,2], Lara Pisani[3], Maria Rosaria Pellegrino[1],
Giovanni Della Casa[4], Roberto D'Amico[5], Massimo Girardis[6], Stefano Nava[3], Enrico M. Clini[1], and
Alessandro Marchioni[1]



Am J Respir Crit Care Med Vol 202, Iss 4, pp 558–567, Aug 15, 2020

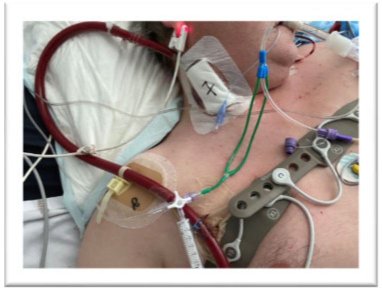
Inspiratory Effort and Lung Mechanics in Spontaneously Breathing Patients with Acute Respiratory Failure due to COVID-19: A Matched Control Study



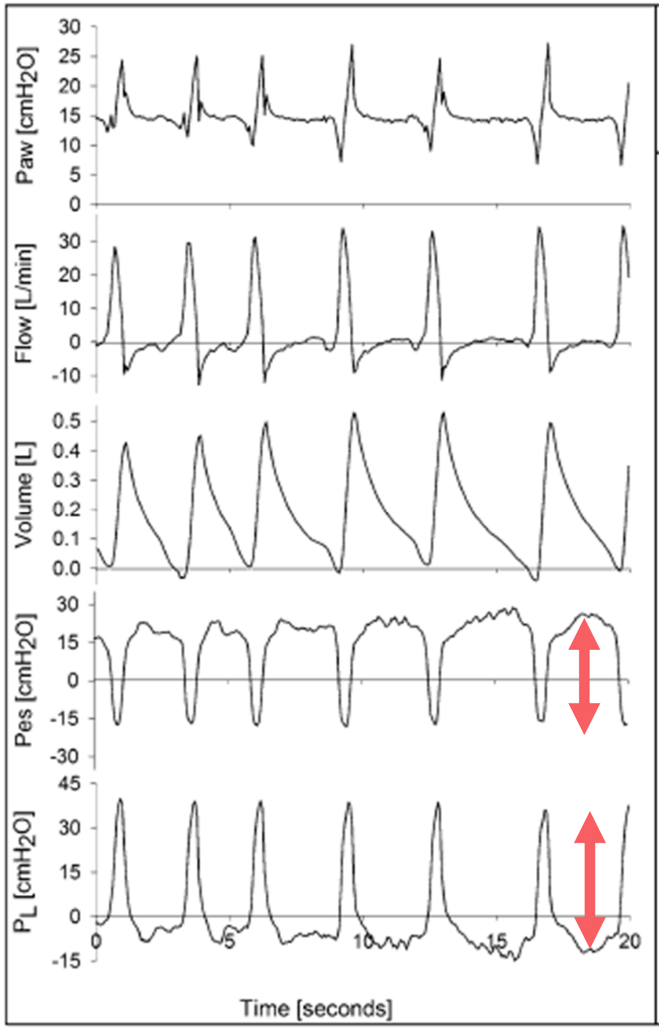
American Journal of Respiratory and Critical Care Medicine Volume 204 Number 6 | September 15 2021

Extremely high transpulmonary pressure in a spontaneously breathing patient with early severe ARDS on ECMO

Tommaso Mauri[1], Thomas Langer[2], Alberto Zanella[1], Giacomo Grasselli[1] and Antonio Pesenti[1,2*]



ICM
INTENSIVE CARE MEDICINE



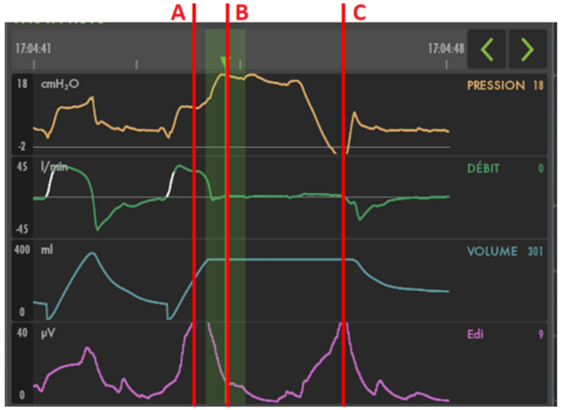
	Volume controlled	Pressure support
PEEP (cmH_2O)	15	15
Vt (ml/kg PBW)	4	6.5
RR (bpm)	10	21
Ppeak (cmH_2O)	37	24
ECMO GF (l/min)	6.5	11.0
VCO_2-NL (ml/min)	n.a.	74
VCO_2-ML (ml/min)	n.a.	253
VCO_2-ML/VCO_2-TOT (%)	n.a.	77
ΔPes (cmH_2O)	+2	-35
P_L (cmH_2O)	12	38
pH	7.42	7.47
PaO_2 (mmHg)	63	63
$PaCO_2$ (mmHg)	45	37

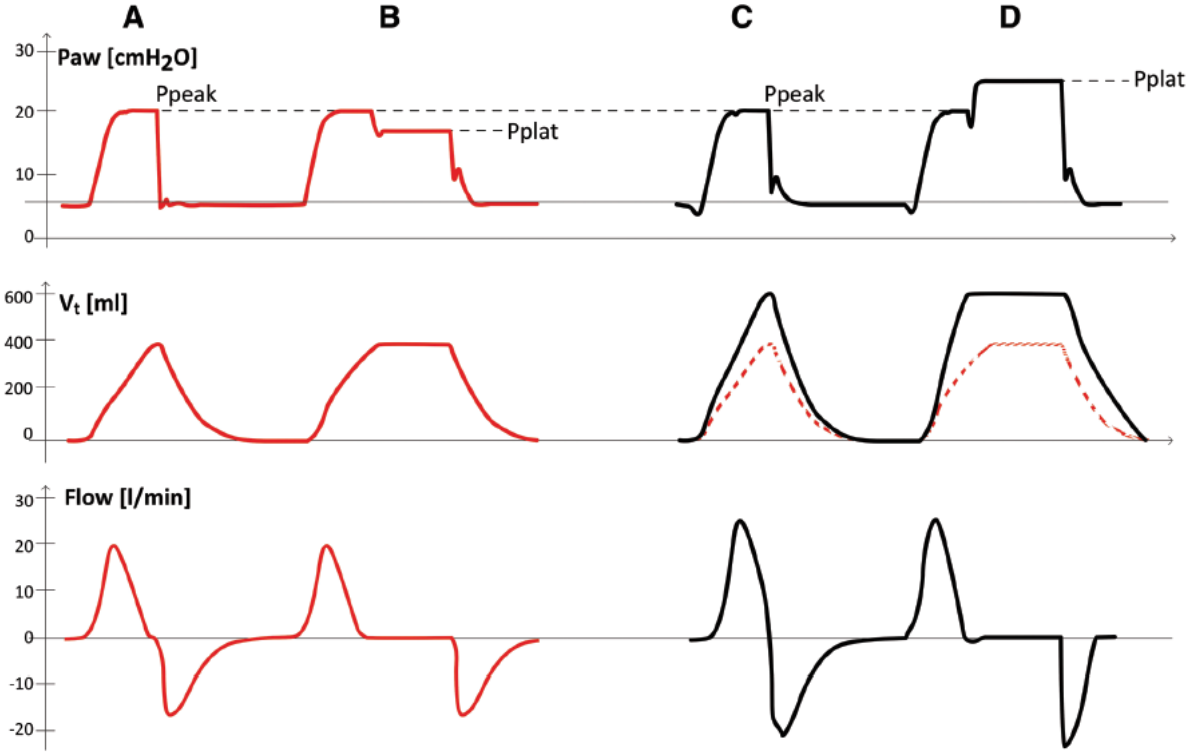
Aide à 9 cmH_2O

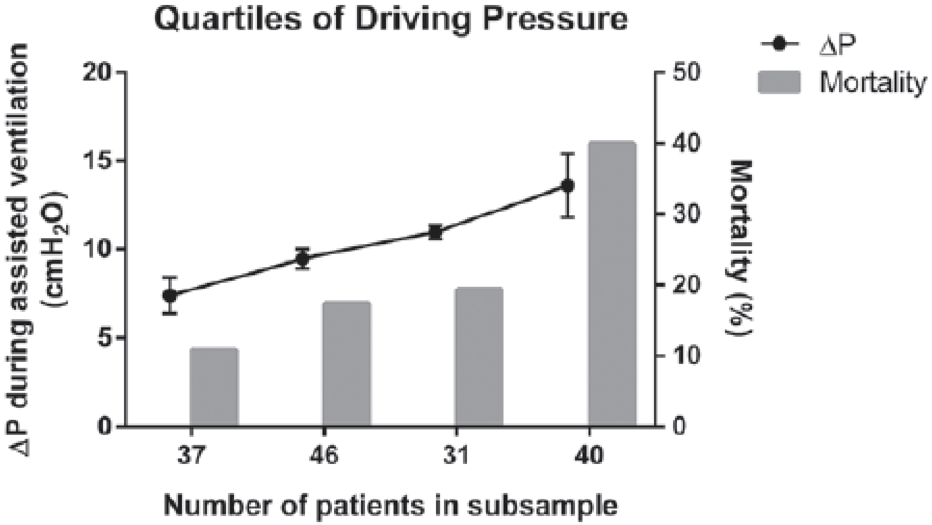
Driving Pressure Is Associated with Outcome during Assisted Ventilation in Acute Respiratory Distress Syndrome

Giacomo Bellani, M.D., Ph.D., Alice Grassi, M.D.,
Simone Sosio, M.D., Stefano Gatti, M.D.,
Brian P. Kavanagh, M.B., Antonio Pesenti, M.D.,
Giuseppe Foti, M.D.

Anesthesiology 2019; 131:594–604


A
B
C
17:04:41
17:04:48
18
cmH₂O
-2
PRESSION 18
45
l/min
-45
DÉBIT 0
400
ml
0
VOLUME 301
40
μV
0
Edi 9





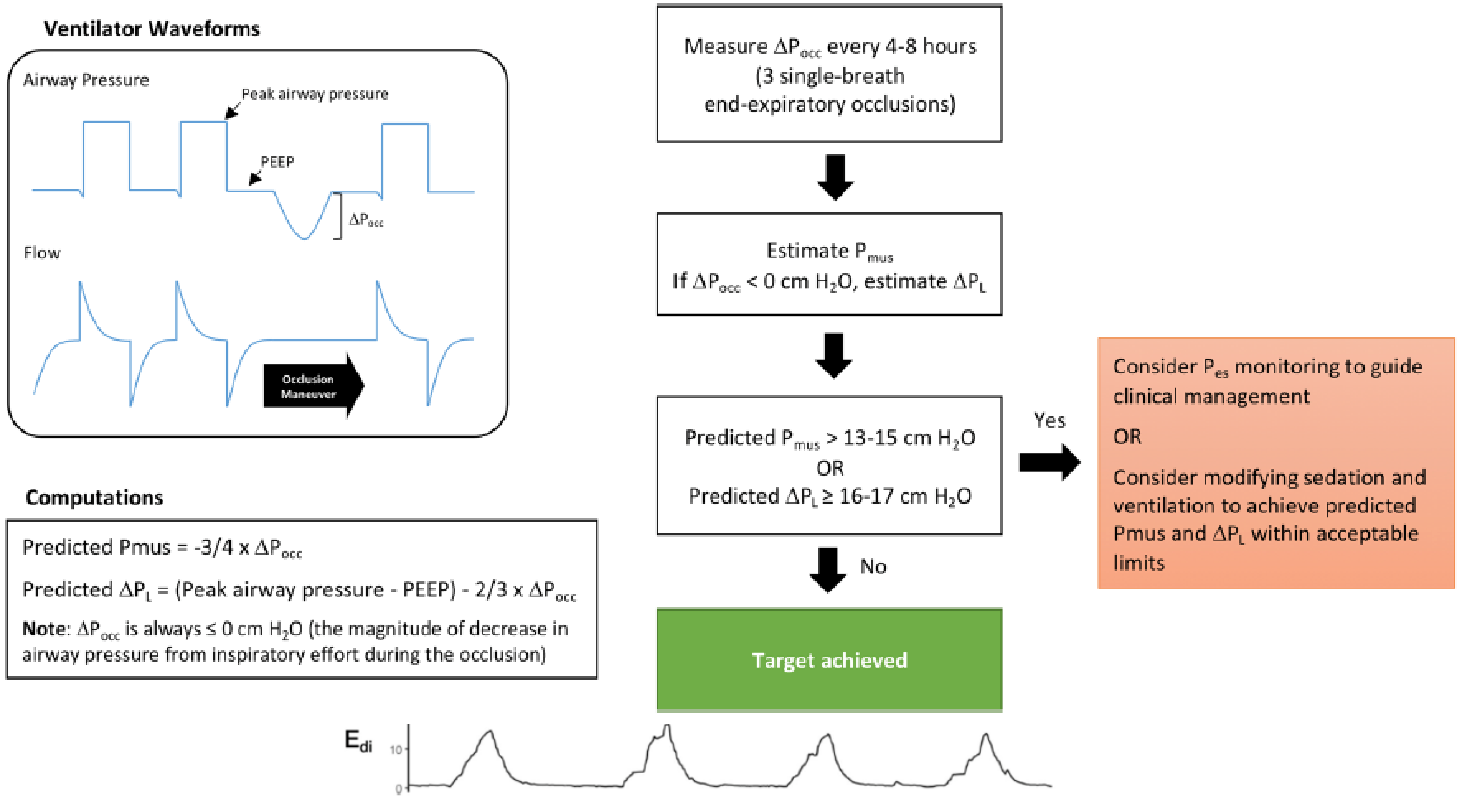
Bertoni *et al. Critical Care* (2019) 23:346
https://doi.org/10.1186/s13054-019-2617-0

Critical Care

RESEARCH **Open Access**

A novel non-invasive method to detect excessively high respiratory effort and dynamic transpulmonary driving pressure during mechanical ventilation

Michele Bertoni[1,2], Irene Telias[3,4], Martin Urner[3,5], Michael Long[6], Lorenzo Del Sorbo[3,5], Eddy Fan[3,5,7], Christer Sinderby[3,4], Jennifer Beck[3,4], Ling Liu[8], Haibo Qiu[8], Jenna Wong[5], Arthur S. Slutsky[3,4], Niall D. Ferguson[3,5,7,9,10], Laurent J. Brochard[3,4] and Ewan C. Goligher[3,5,10,11*]



Measure ΔP_{occ} every 4-8 hours (3 single-breath end-expiratory occlusions)

Estimate P_{mus}
If $\Delta P_{occ} < 0$ cm H_2O, estimate ΔP_L

Predicted $P_{mus} > 13$-15 cm H_2O
OR
Predicted $\Delta P_L \geq 16$-17 cm H_2O

Consider P_{es} monitoring to guide clinical management

OR

Consider modifying sedation and ventilation to achieve predicted Pmus and ΔP_L within acceptable limits

Target achieved

Computations

Predicted Pmus = $-3/4 \times \Delta P_{occ}$

Predicted ΔP_L = (Peak airway pressure - PEEP) - $2/3 \times \Delta P_{occ}$

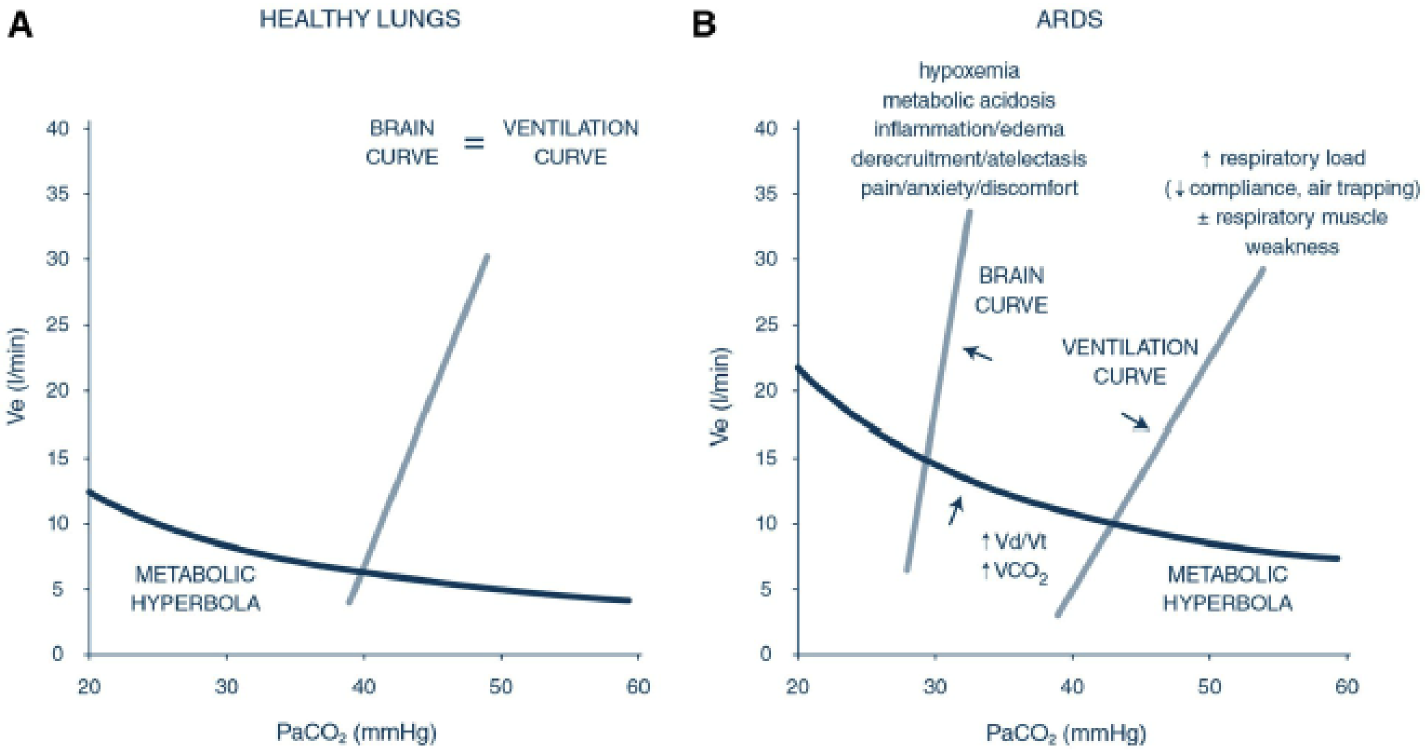
Note: ΔP_{occ} is always ≤ 0 cm H_2O (the magnitude of decrease in airway pressure from inspiratory effort during the occlusion)

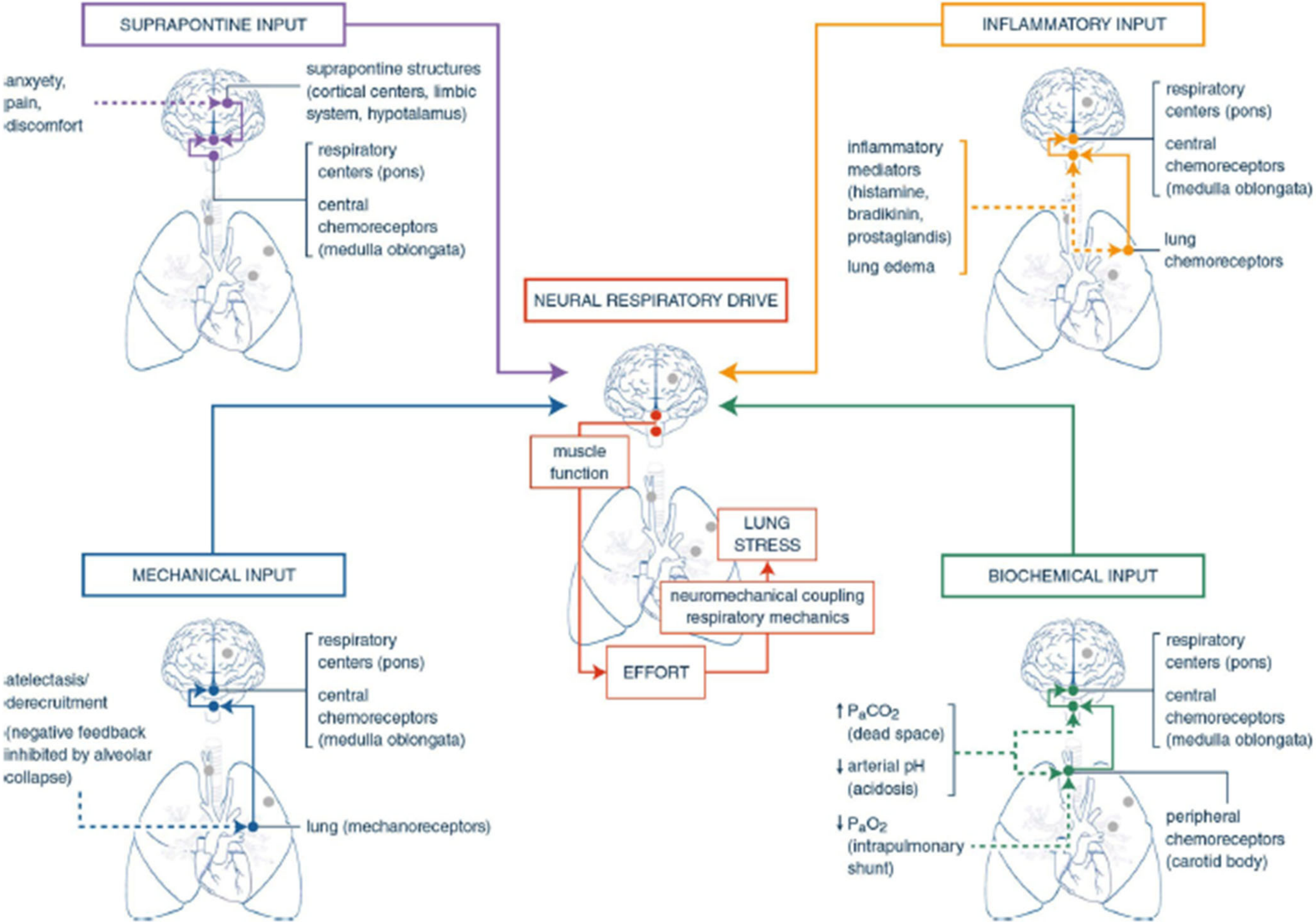
Intensive Care Med (2020) 46:606–618
https://doi.org/10.1007/s00134-020-05942-6

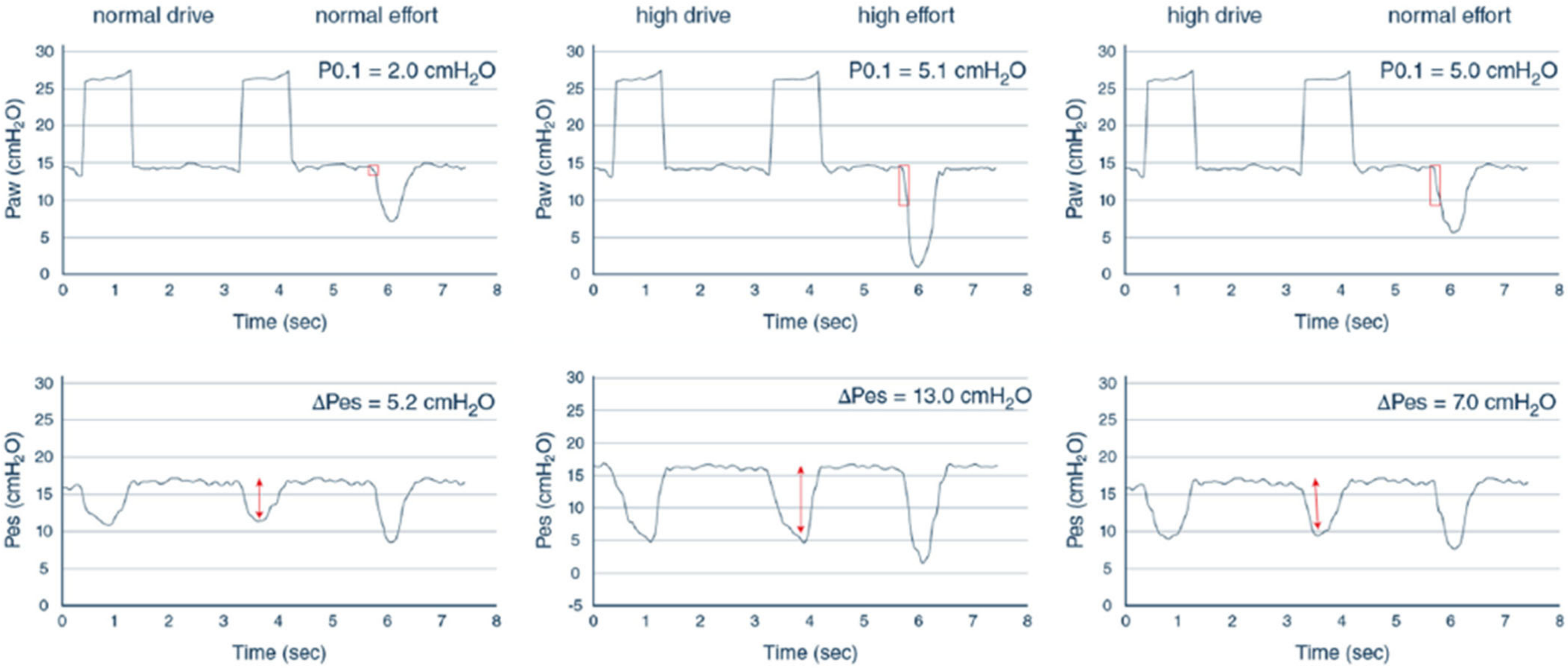
NARRATIVE REVIEW

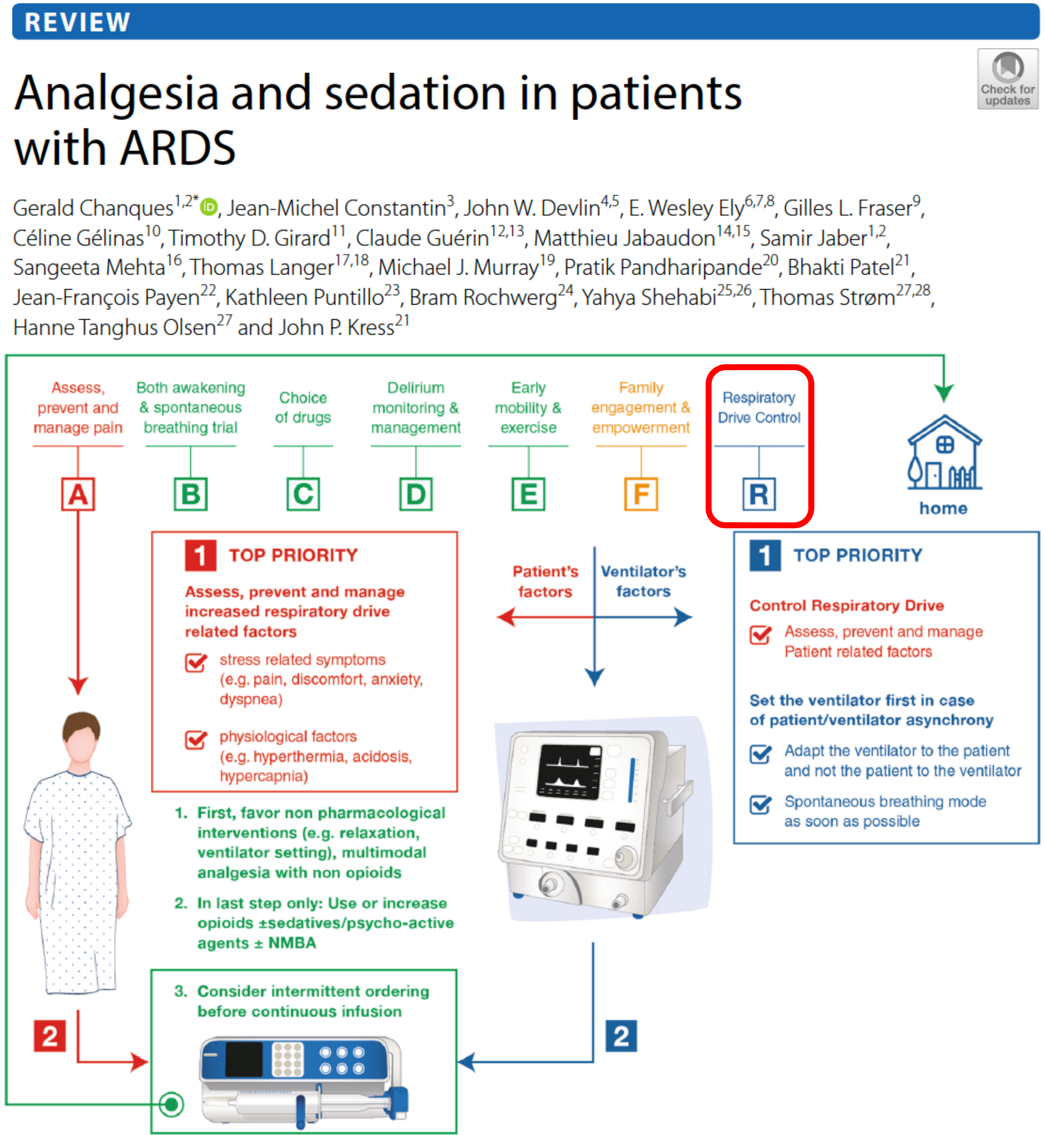
Respiratory drive in the acute respiratory distress syndrome: pathophysiology, monitoring, and therapeutic interventions

Elena Spinelli[1], Tommaso Mauri[1,2*], Jeremy R. Beitler[3], Antonio Pesenti[1,2] and Daniel Brodie[3]




SUPRAPONTINE INPUT
anxyety,
pain,
discomfort
suprapontine structures (cortical centers, limbic system, hypotalamus)
respiratory centers (pons)
central chemoreceptors (medulla oblongata)
INFLAMMATORY INPUT
respiratory centers (pons)
central chemoreceptors (medulla oblongata)
inflammatory mediators (histamine, bradikinin, prostaglandis)
lung edema
lung chemoreceptors
NEURAL RESPIRATORY DRIVE
muscle function
LUNG STRESS
neuromechanical coupling respiratory mechanics
EFFORT
MECHANICAL INPUT
respiratory centers (pons)
central chemoreceptors (medulla oblongata)
atelectasis/
derecruitment
(negative feedback inhibited by alveolar collapse)
lung (mechanoreceptors)
BIOCHEMICAL INPUT
respiratory centers (pons)
central chemoreceptors (medulla oblongata)
↑ P_aCO_2 (dead space)
↓ arterial pH (acidosis)
↓ P_aO_2 (intrapulmonary shunt)
peripheral chemoreceptors (carotid body)


normal drive
normal effort
high drive
high effort
high drive
normal effort
P0.1 = 2.0 cmH2O
P0.1 = 5.1 cmH2O
P0.1 = 5.0 cmH2O
Paw (cmH2O)
ΔPes = 5.2 cmH2O
ΔPes = 13.0 cmH2O
ΔPes = 7.0 cmH2O
Pes (cmH2O)
Time (sec)
0
1
2
3
4
5
6
7
8
0
5
10
15
20
25
30
-5



REVIEW

Analgesia and sedation in patients with ARDS

Gerald Chanques[1,2*], Jean-Michel Constantin[3], John W. Devlin[4,5], E. Wesley Ely[6,7,8], Gilles L. Fraser[9], Céline Gélinas[10], Timothy D. Girard[11], Claude Guérin[12,13], Matthieu Jabaudon[14,15], Samir Jaber[1,2], Sangeeta Mehta[16], Thomas Langer[17,18], Michael J. Murray[19], Pratik Pandharipande[20], Bhakti Patel[21], Jean-François Payen[22], Kathleen Puntillo[23], Bram Rochwerg[24], Yahya Shehabi[25,26], Thomas Strøm[27,28], Hanne Tanghus Olsen[27] and John P. Kress[21]

Check for updates
Assess, prevent and manage pain
Both awakening & spontaneous breathing trial
Choice of drugs
Delirium monitoring & management
Early mobility & exercise
Family engagement & empowerment
Respiratory Drive Control
A
B
C
D
E
F
R
home
Patient's factors
Ventilator's factors
2
2

1 TOP PRIORITY

Assess, prevent and manage increased respiratory drive related factors

☑ stress related symptoms (e.g. pain, discomfort, anxiety, dyspnea)

☑ physiological factors (e.g. hyperthermia, acidosis, hypercapnia)

1. First, favor non pharmacological interventions (e.g. relaxation, ventilator setting), multimodal analgesia with non opioids
2. In last step only: Use or increase opioids ±sedatives/psycho-active agents ± NMBA
3. Consider intermittent ordering before continuous infusion

1 TOP PRIORITY

Control Respiratory Drive

☑ Assess, prevent and manage Patient related factors

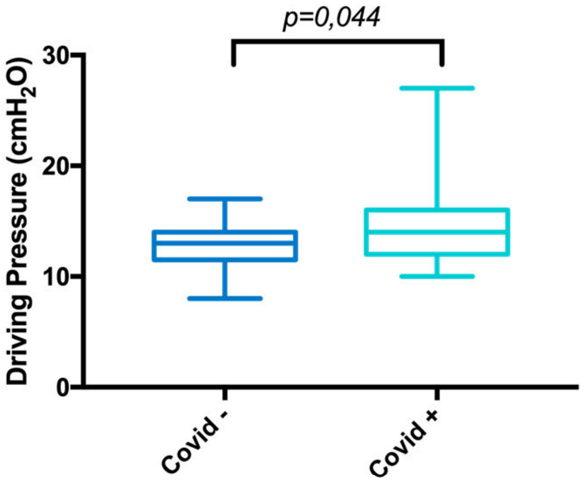
Set the ventilator first in case of patient/ventilator asynchrony

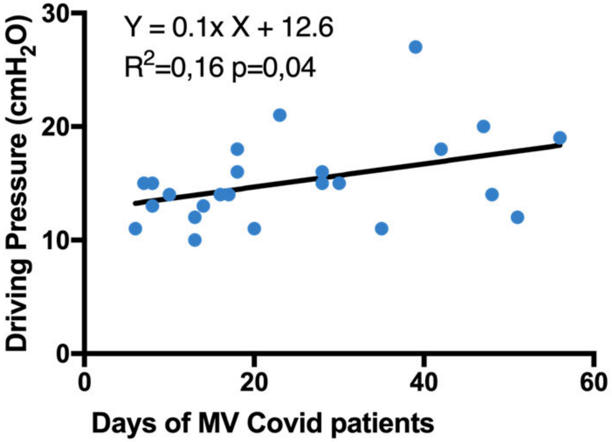
☑ Adapt the ventilator to the patient and not the patient to the ventilator

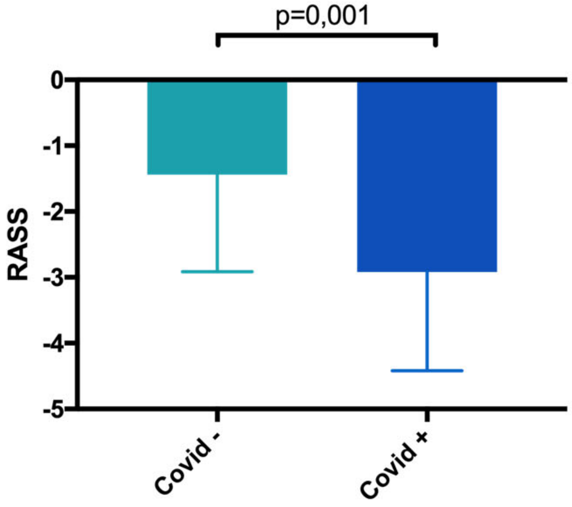
☑ Spontaneous breathing mode as soon as possible

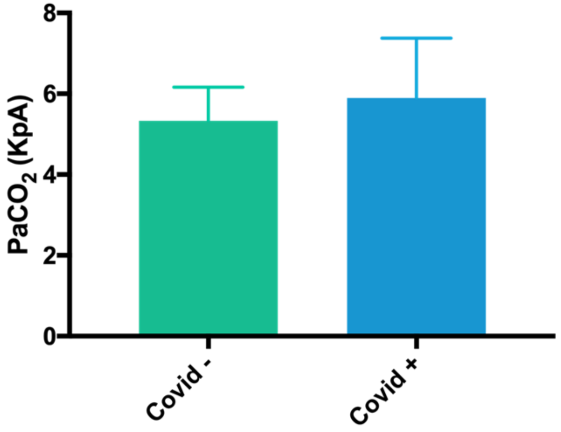
Chanques et al. Intensive Care Med. 2020;46:2342-2356.

Driving Pressure en VSAI

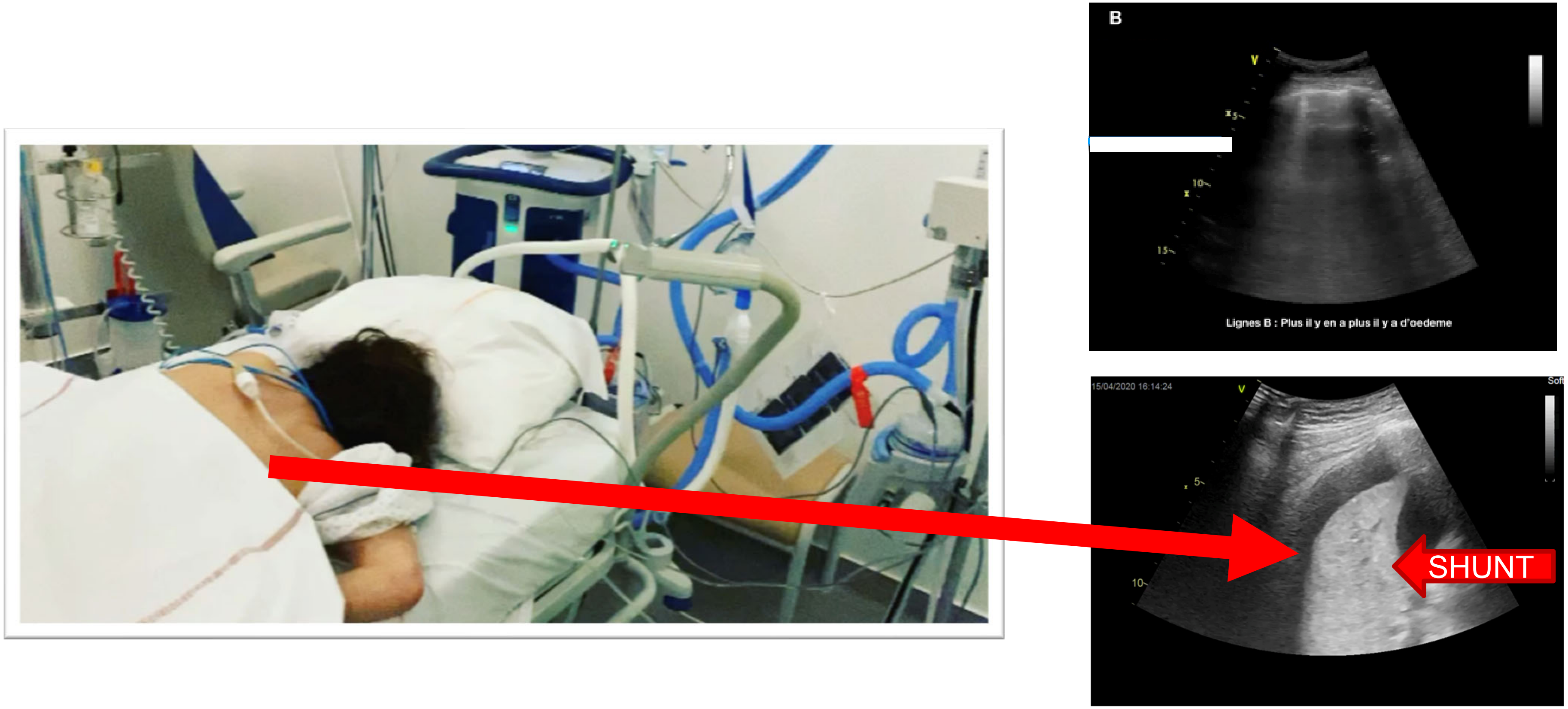


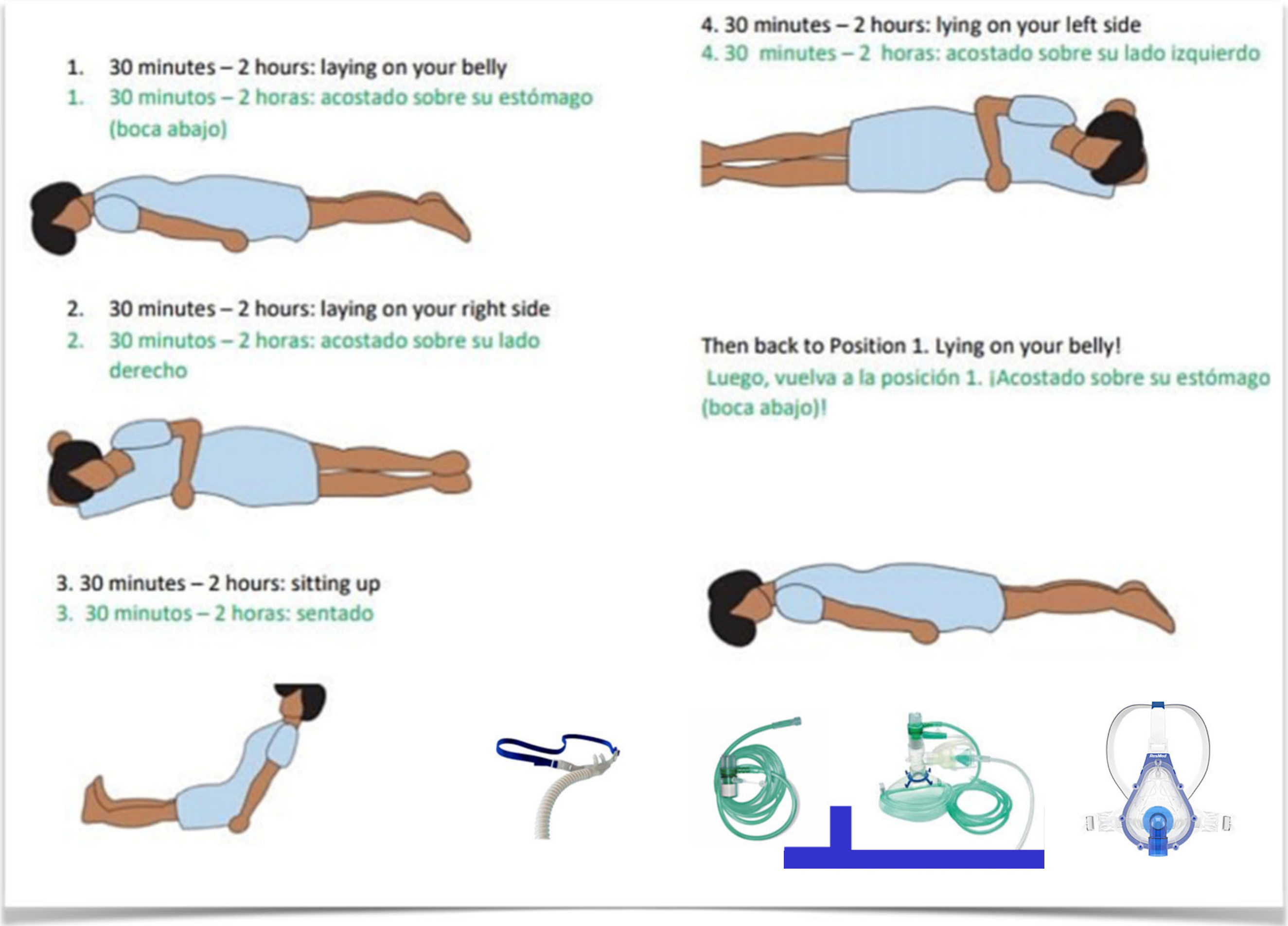






DV en VS sous HNF ou VNI


B
V
5
10
15
Lignes B : Plus il y en a plus il y a d'oedeme
15/04/2020 16:14:24
Soft
V
5
10
SHUNT



1. 30 minutes – 2 hours: laying on your belly
1. 30 minutos – 2 horas: acostado sobre su estómago (boca abajo)

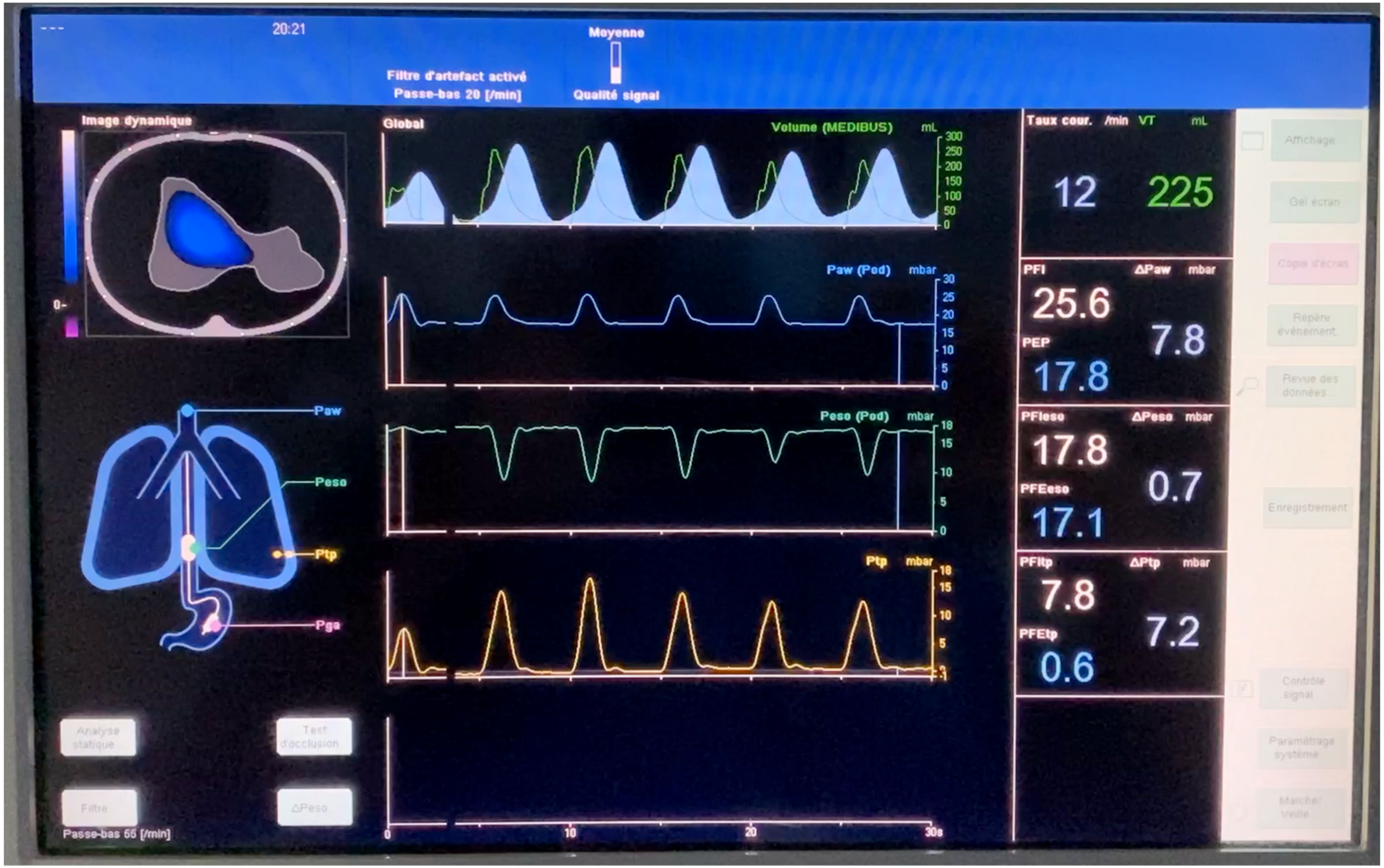
2. 30 minutes – 2 hours: laying on your right side
2. 30 minutos – 2 horas: acostado sobre su lado derecho

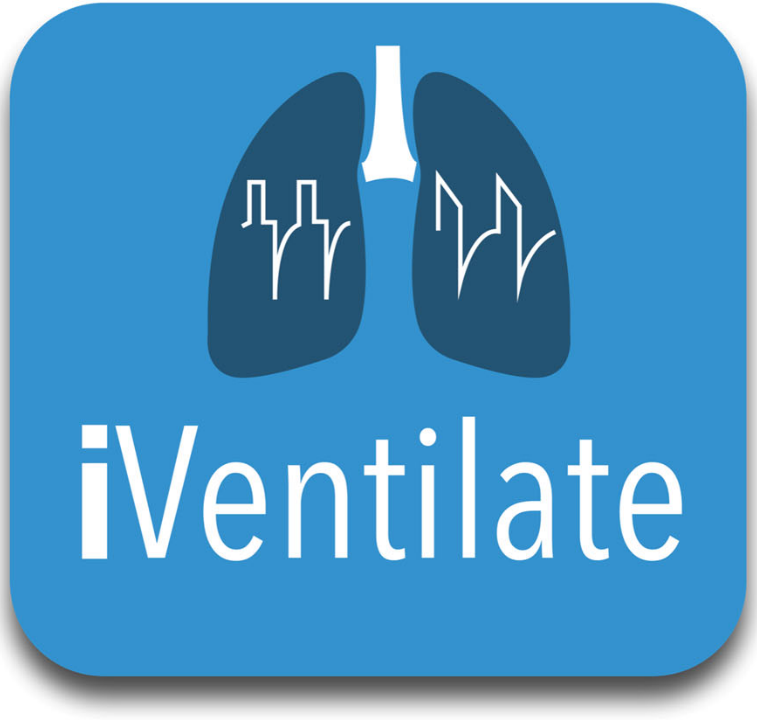
3. 30 minutes – 2 hours: sitting up
3. 30 minutos – 2 horas: sentado

4. 30 minutes – 2 hours: lying on your left side
4. 30 minutos – 2 horas: acostado sobre su lado izquierdo

Then back to Position 1. Lying on your belly!

Luego, vuelva a la posición 1. ¡Acostado sobre su estómago (boca abajo)!

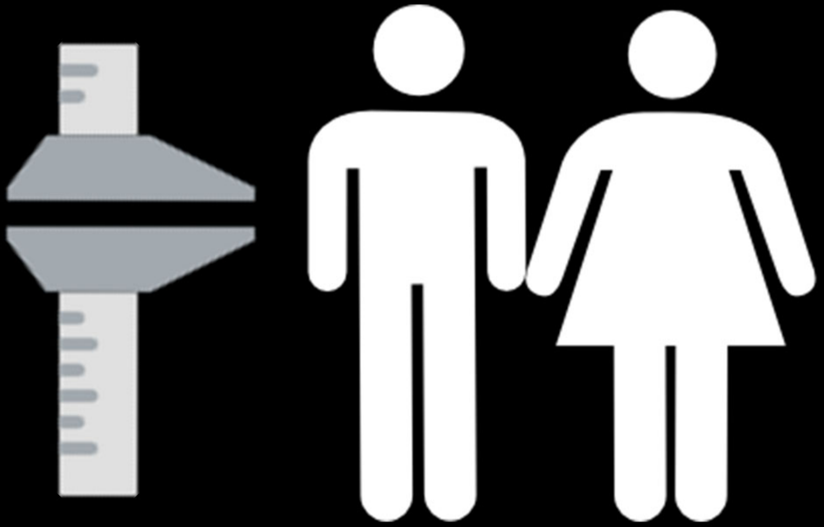

20:21
Moyenne
Filtre d'artefact activé
Passe-bas 20 [/min]
Qualité signal
Image dynamique
0-
Global
Volume (MEDIBUS)
mL
300
250
200
150
100
50
0
Paw (Pod)
mbar
30
25
20
15
10
5
0
Peso (Pod)
mbar
18
15
10
5
0
Ptp
mbar
18
15
10
5
Paw
Peso
Ptp
Pga
Analyse statique
Test d'occlusion
Filtre
ΔPeso
Passe-bas 55 [/min]
0
10
20
30s
Taux cour. /min
VT mL
12
225
PFI
ΔPaw mbar
25.6
PEP
17.8
7.8
PFIeso
ΔPeso mbar
17.8
PFEeso
17.1
0.7
PFItp
ΔPtp mbar
7.8
PFEtp
0.6
7.2
Affichage
Gel écran
Copie d'écran
Repère événement
Revue des données
Enregistrement
Contrôle signal
Paramétrage système
Marche/Veille


iVentilate

Equation du mouvement du poumon :

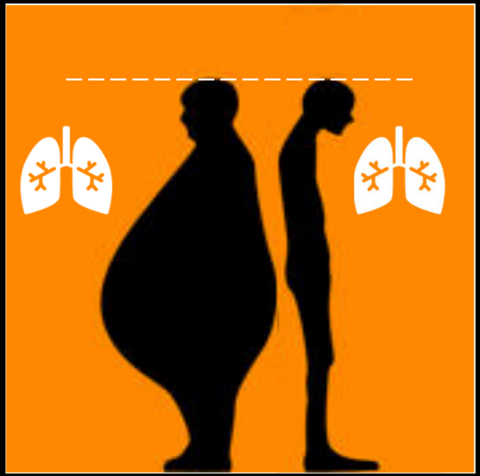
Le volume courant

- V_T entre 6 et 8 ml.kg-1 Poids Prédit



Taille et sexe

2 morphotypes avec les mêmes poumons



2 hommes de même taille

Ventilation avec le mode VSAI

SimVA

Ventilation Artificielle Virtuelle