

Mise en place d'une ventilation non invasive à domicile

Cas cliniques

Borel JC

Medrinal C

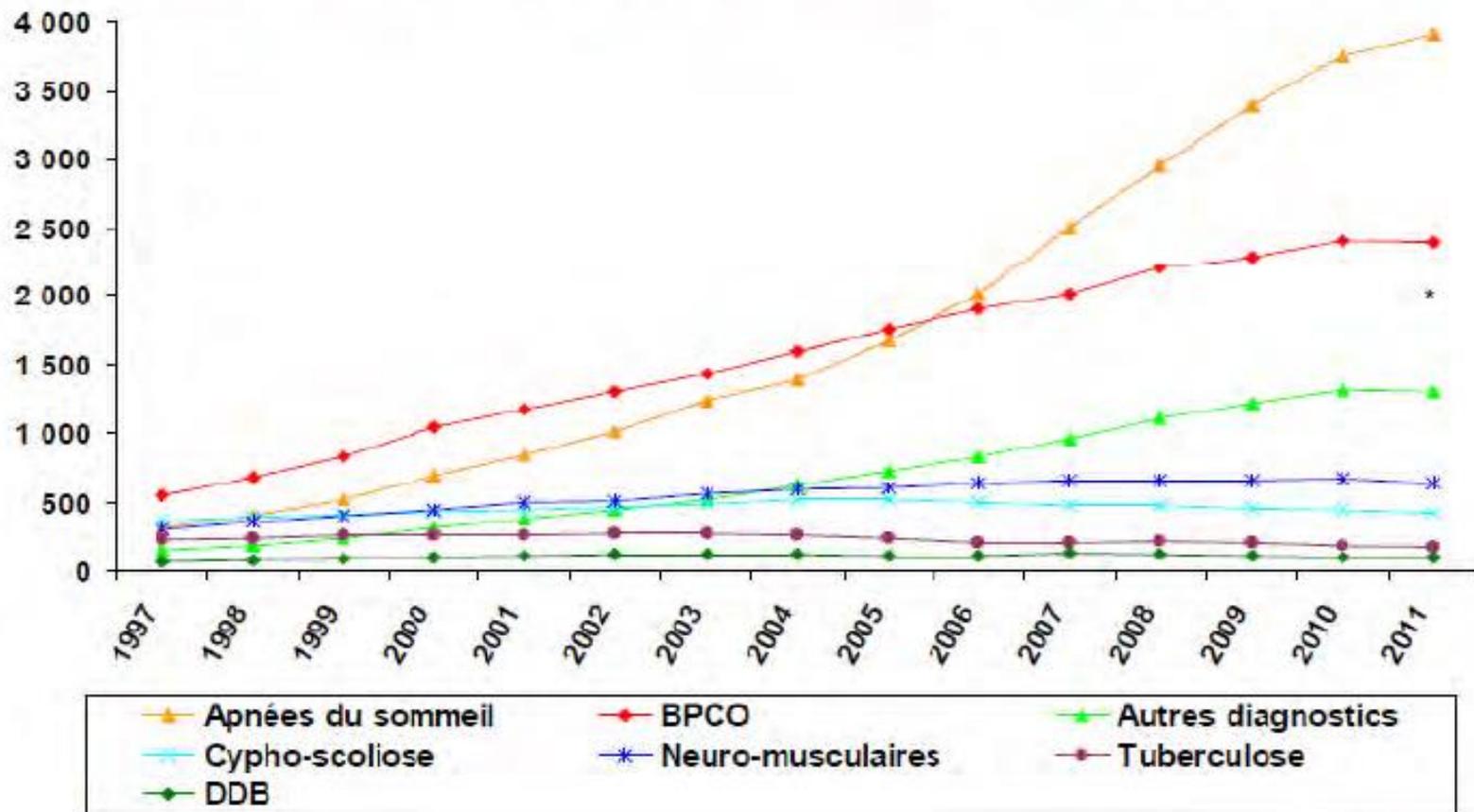
CPLF 2015, Lille

- Aucun conflit d'intérêt

Introduction

- Toute les structures de soins peut en théorie être sollicitée pour la mise en place d'une Ventilation non invasive.
- La tendance actuelle à l'augmentation du nombre de prescriptions conduit vers l'installation au domicile des patients.
- Augmentation de 12% par an du nombre de patients traités par VNI.

Ventilation par masque \pm oxygène : évolution des étiologies au 31 décembre



Où installer une VNI?

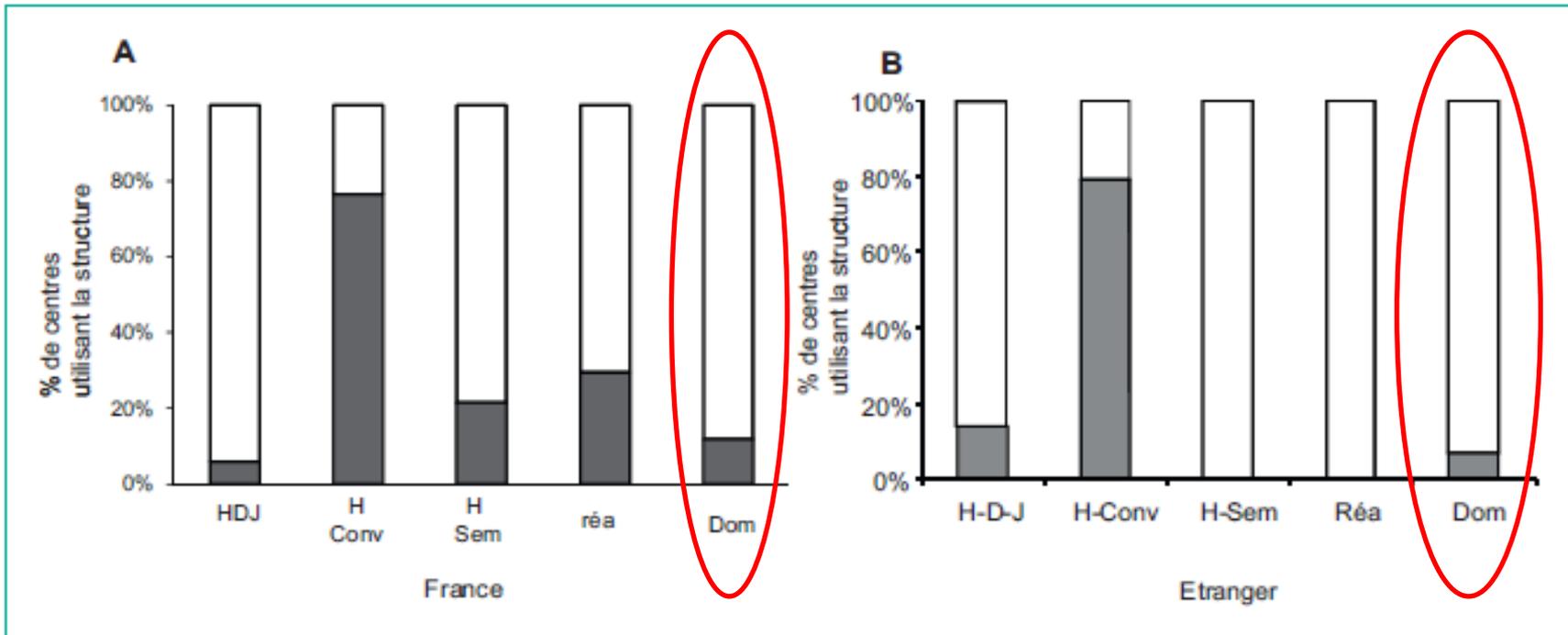
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Groupe CasaVNI





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respiratoryMEDICINE 

Non-invasive home mechanical ventilation: Effectiveness and efficiency of an outpatient initiation protocol compared with the standard in-hospital model

**Manel Luján, Amalia Moreno, Carmen Veigas, Concepción Montón,
Xavier Pomares, Christian Domingo***

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Departament de Medicina, Universitat Autònoma de Barcelona (Bellaterra), Spain*

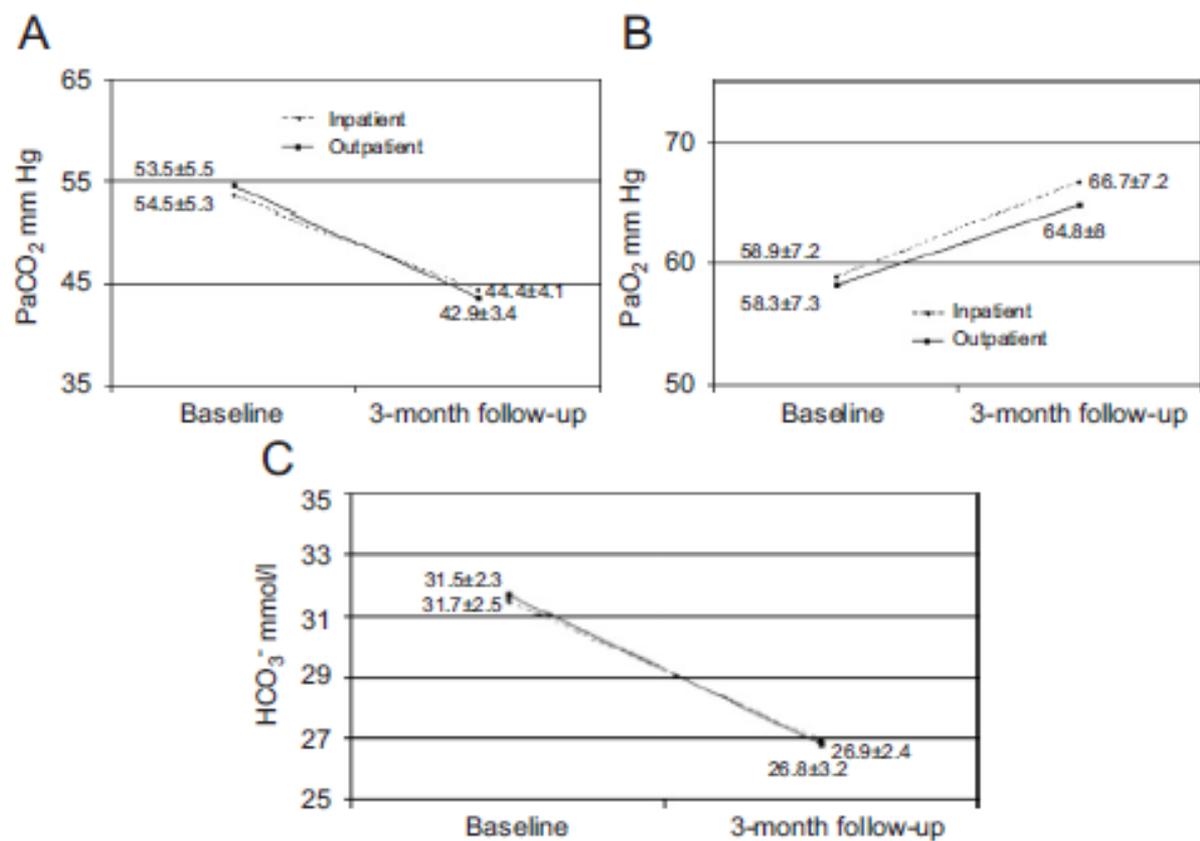


Figure 2 (A) PaCO₂ values at baseline and after three months' follow-up. (B) PaO₂ values at baseline and after three months' follow-up. (C) Plasma bicarbonate values at baseline and after three months' follow-up. The comparison reveals significant differences at the end of the follow-up period in both groups, though no differences were associated with the choice of protocol.



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Initiation of home mechanical ventilation at home: A randomised controlled trial of efficacy, feasibility and costs



A. Hazenberg^{a,b,*}, H.A.M. Kerstjens^{a,b}, S.C.L. Prins^c,
K.M. Vermeulen^d, P.J. Wijkstra^{a,b}

Table 2 Changes in daytime arterial blood gasses and lung function pre home mechanical ventilation to 6 months after the start.

	Home group (<i>n</i> = 30)			Hospital group (<i>n</i> = 31)		Between groups	
	Baseline	Follow-up	<i>P</i> -Value ^a	Baseline	Follow-up	<i>P</i> -Value ^a	<i>P</i> -value ^b
Ph	7.40 ± 0.3	7.40 ± 0.3	0.261	7.40 ± 0.3	7.40 ± 0.3	0.913	0.423
PaCO ₂ (kPa)	6.6 ± 0.9	5.7 ± 0.8	0.000	6.6 ± 1.1	5.9 ± 0.8	0.000	0.631
PaO ₂ (kPa)	10.0 ± 1.7	11.3 ± 2.2	0.002	9.5 ± 1.3	10.3 ± 1.7	0.015	0.579
SaO ₂ %	95 ± 2.9	96 ± 1.9	0.020	94 ± 2.7	95 ± 3.6	0.348	0.598
HCO ₃ (mmol/l)	30.2 ± 3.8	26.6 ± 3.1	0.000	30.4 ± 3.8	26.9 ± 2.5	0.000	0.996
aBE	4.9 ± 2.6	2.6 ± 1.7	0.000	4.8 ± 2.8	2.2 ± 1.8	0.000	0.283
FVC (% pred)	51.6 ± 22.8	53.4 ± 21.8	0.528	52.4 ± 18.5	49.8 ± 19.0	0.301	0.428

Data are presented as *n* or mean ± SD.

kPa: kilopascal. Ph: acidity level. PaCO₂: partial pressure of arterial carbon dioxide. PaO₂: partial pressure of arterial oxygen. SaO₂: arterial oxygen saturation. HCO₃: bicarbonate. VC: vital capacity. Follow-up: six months after the initiation of home mechanical ventilation.

Bold: *p* < 0.05 significant change.

^a *P*-value refers to paired *t* test analysis from starting ventilatory support to six months follow-up within each group.

^b *P*-value for difference in change Δ from baseline between groups.



HAUTE AUTORITÉ DE SANTÉ

RÉVISION DE CATÉGORIES HOMOGÈNES DE DISPOSITIFS MÉDICAUX

Ventilation mécanique à domicile

Dispositifs médicaux et prestations associées pour traitement de l'insuffisance respiratoire

Date de validation par la CNEDiMTS : 20 novembre 2012

Les caractéristiques des trois niveaux de prestations sont décrites ci-après.

	Prestation de niveau 1	Prestation de niveau 2	Prestation de niveau 3
Typologie de patients	Hypercapnie diurne ou nocturne corrigée par une ventilation nocturne exclusive	Persistance d'une hypercapnie diurne malgré une ventilation nocturne bien conduite	Dépendance du patient au ventilateur > 16 h/j
Durée de ventilation prescrite	Au cours du sommeil	Au cours du sommeil et ≥ 1 h dans la journée	> 16 h/j
Autonomie respiratoire estimée	16 h/j	8 à 16 h/j	< 8 h/j
Type de ventilateur	Ventilateur sans batterie interne	Ventilateur avec batterie interne non support de vie	Ventilateur support de vie
2^{ème} ventilateur	Non	Non	Oui, avec circuit en place
Mise à disposition des interfaces	3/an	6/an	8/an
Circuit de secours	Non obligatoire	Obligatoire	Obligatoire



HAUTE AUTORITÉ DE SANTÉ



- la mise en place d'une ventilation à domicile ne doit concerner que des patients adultes, non trachéotomisés ;
- le patient doit être en état clinique « stable » et éligible à la prestation de ventilation mécanique de niveau 1 exclusivement ;
- le rôle des différents intervenants (notamment prestataire) doit être défini et en conformité avec le champ de compétences de chacun. Conformément aux compétences décrites dans le Code de la santé publique, le groupe de travail considère que la présence d'un médecin ou d'un kinésithérapeute est indispensable lors de la mise sous ventilation du patient à domicile ;
- les outils de surveillance à mémoire doivent pouvoir être mis à disposition à domicile (relevé des données du ventilateur, mesure de SpO₂ et de PtcCO₂, etc.) et les résultats transmis si besoin à un médecin capable d'adapter la prescription en conséquence ;

Installation de VNI



- De quel matériel ai-je besoin?

1-Ventilateurs

Mode?

Aide inspiratoire?

Pression expiratoire Positive?

Fréquence de sécurité?

2-Circuits

Valve ou Fuites?

3- Interfaces

Narinaire, Nasal, Naso-buccal, Facial, Casque?



Quels patients?

Maladie neuromusculaires et thoraco-restrictives



S
O
H



Allez on continue

On respire à fond!

KOFF!
KOFF!

Aah... désolé...

BPCO

SOH

- Auchincloss 1955: Clinical and physiological aspects of a case of obesity, polycythemia and alveolar hypoventilation.
- « Obesity Hypoventilation Syndrome » formulé en 1974 par Rochester et Enson.
- $BMI \geq 30 \text{ Kg/m}^2$.
- Hypoventilation diurne ($\text{PaCO}_2 > 45 \text{ mmHg}$).
- Seulement 10% n'ont pas de SAOS.
- Entre 11 000 et 22 000 nouveaux cas par an.

OBESEITY HYPOVENTILATION SYNDROME: A STATE-OF-THE-ART REVIEW

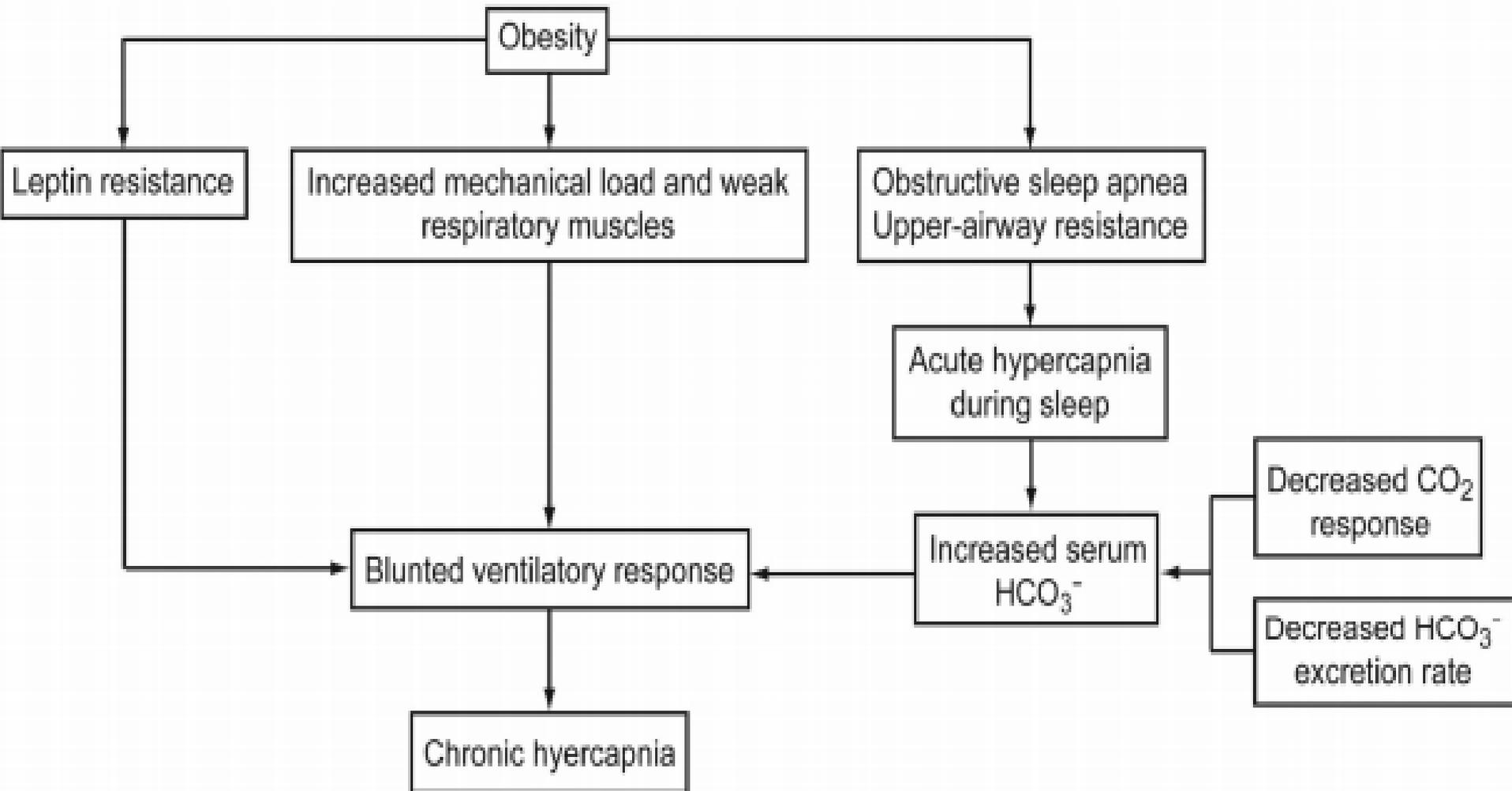
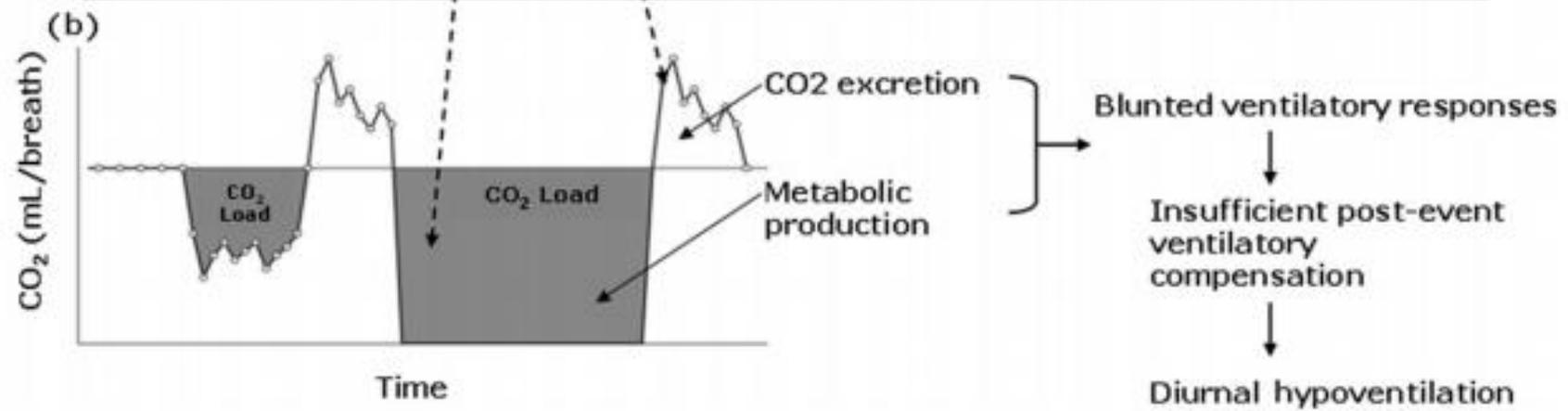
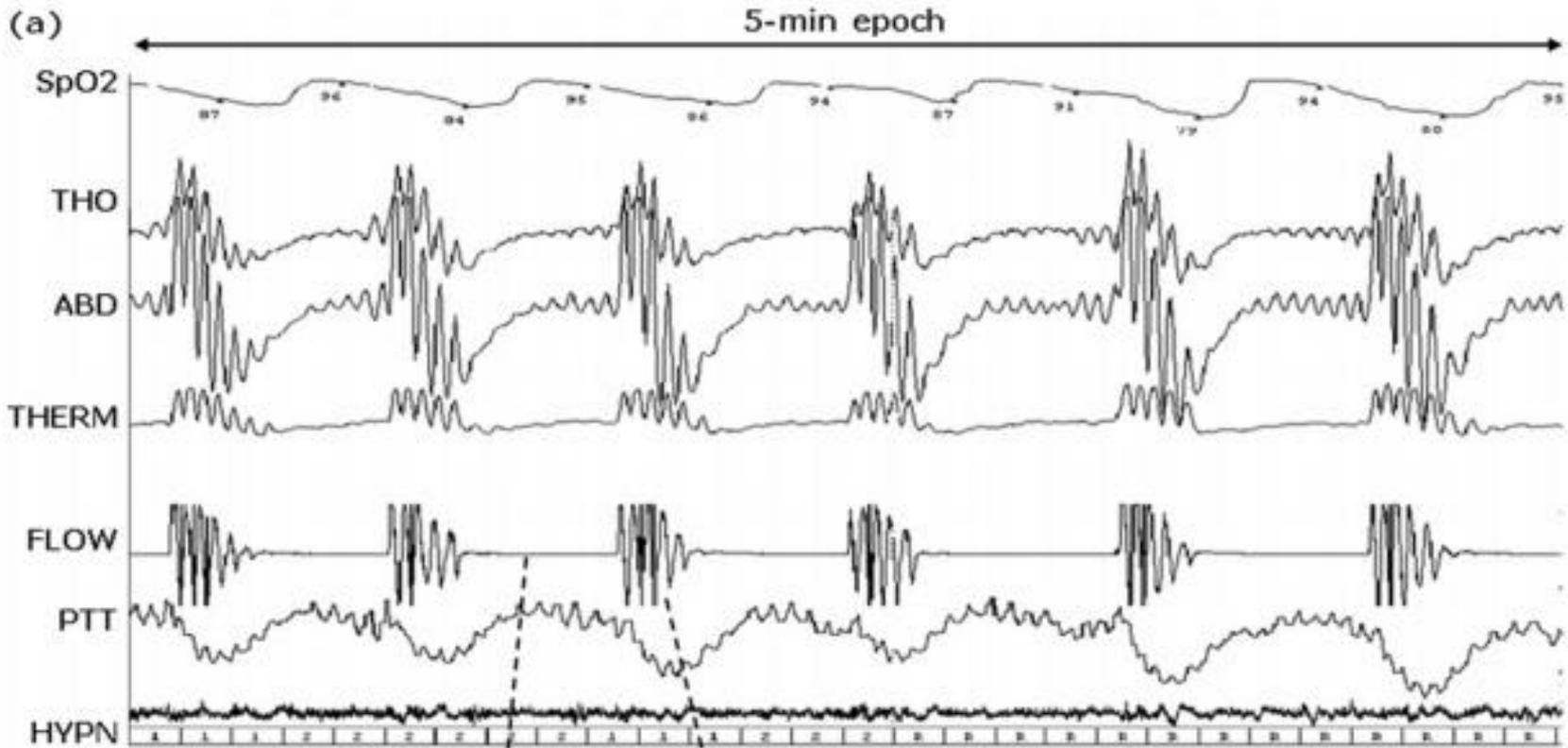


Fig. 8. Mechanisms by which obesity can lead to chronic daytime hypercapnia.



OBEesity HYPOVENTILATION SYNDROME: A STATE-OF-THE-ART REVIEW

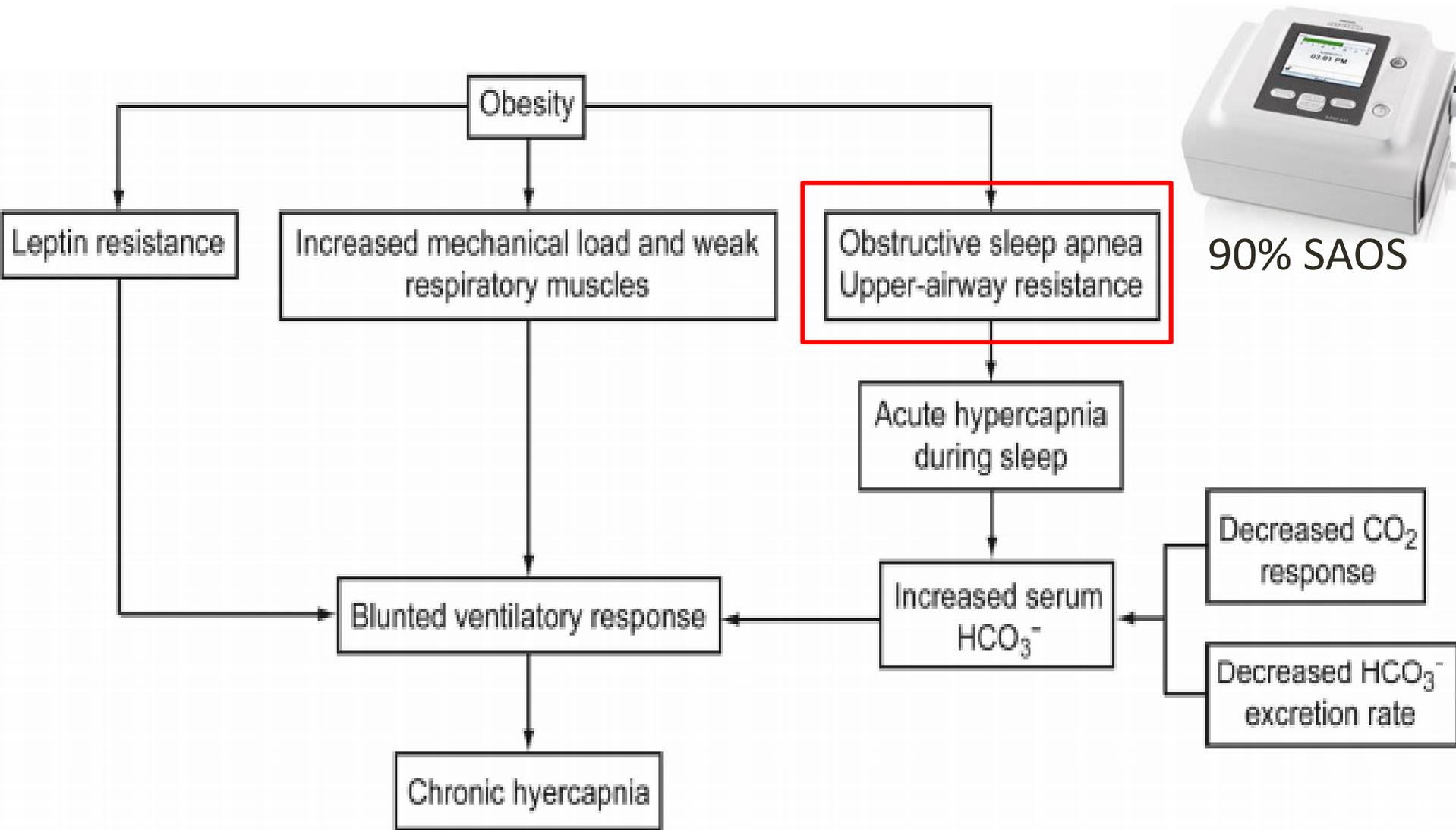
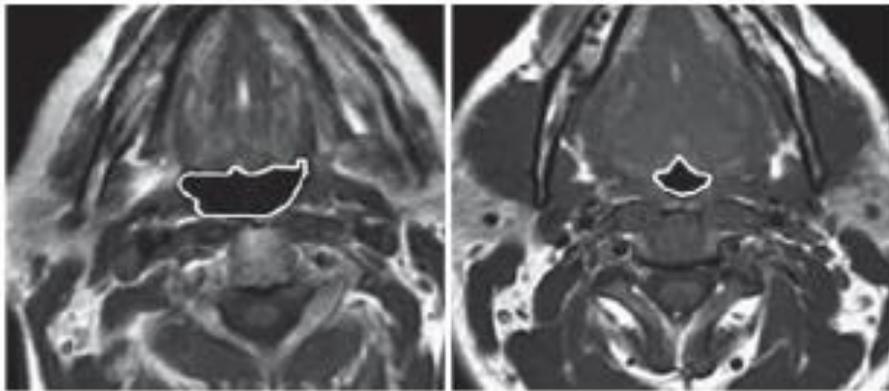
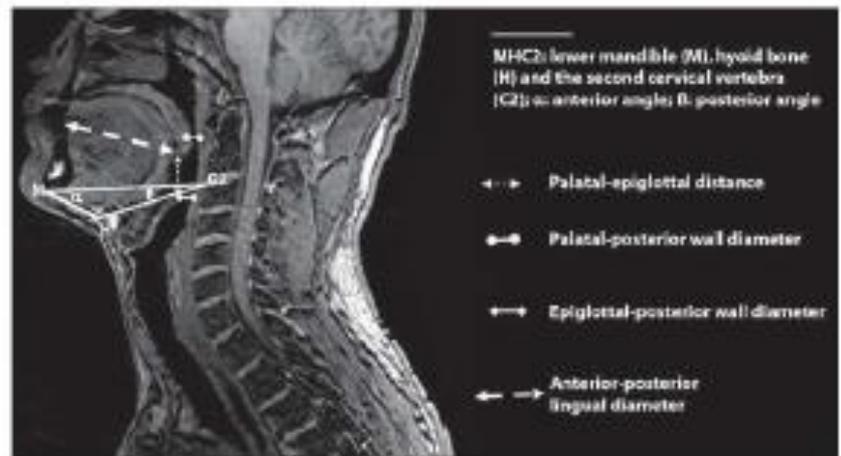
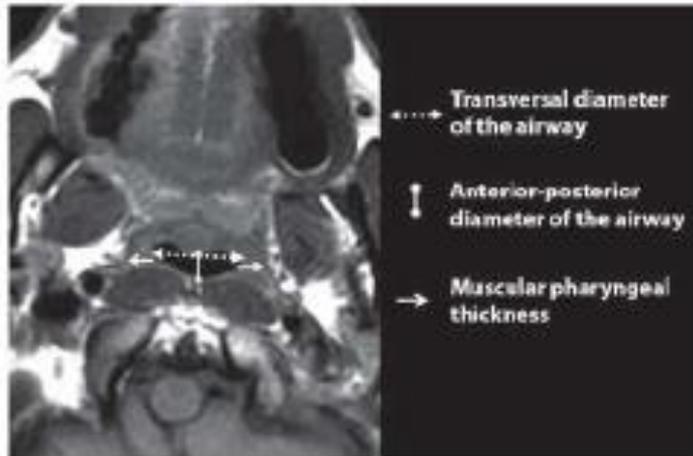
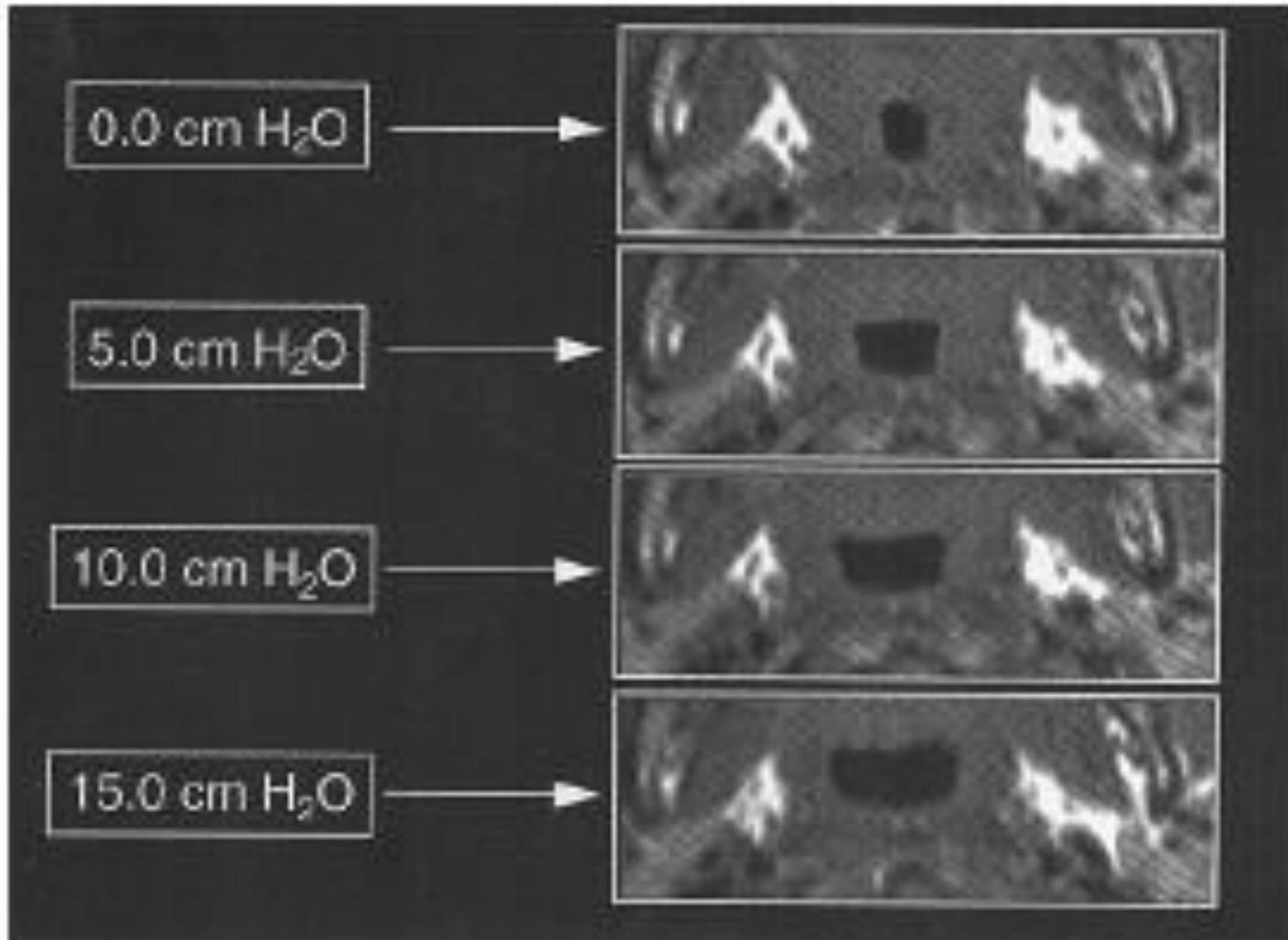


Fig. 8. Mechanisms by which obesity can lead to chronic daytime hypercapnia.



Effet de la Pression expiratoire Positive

Positive

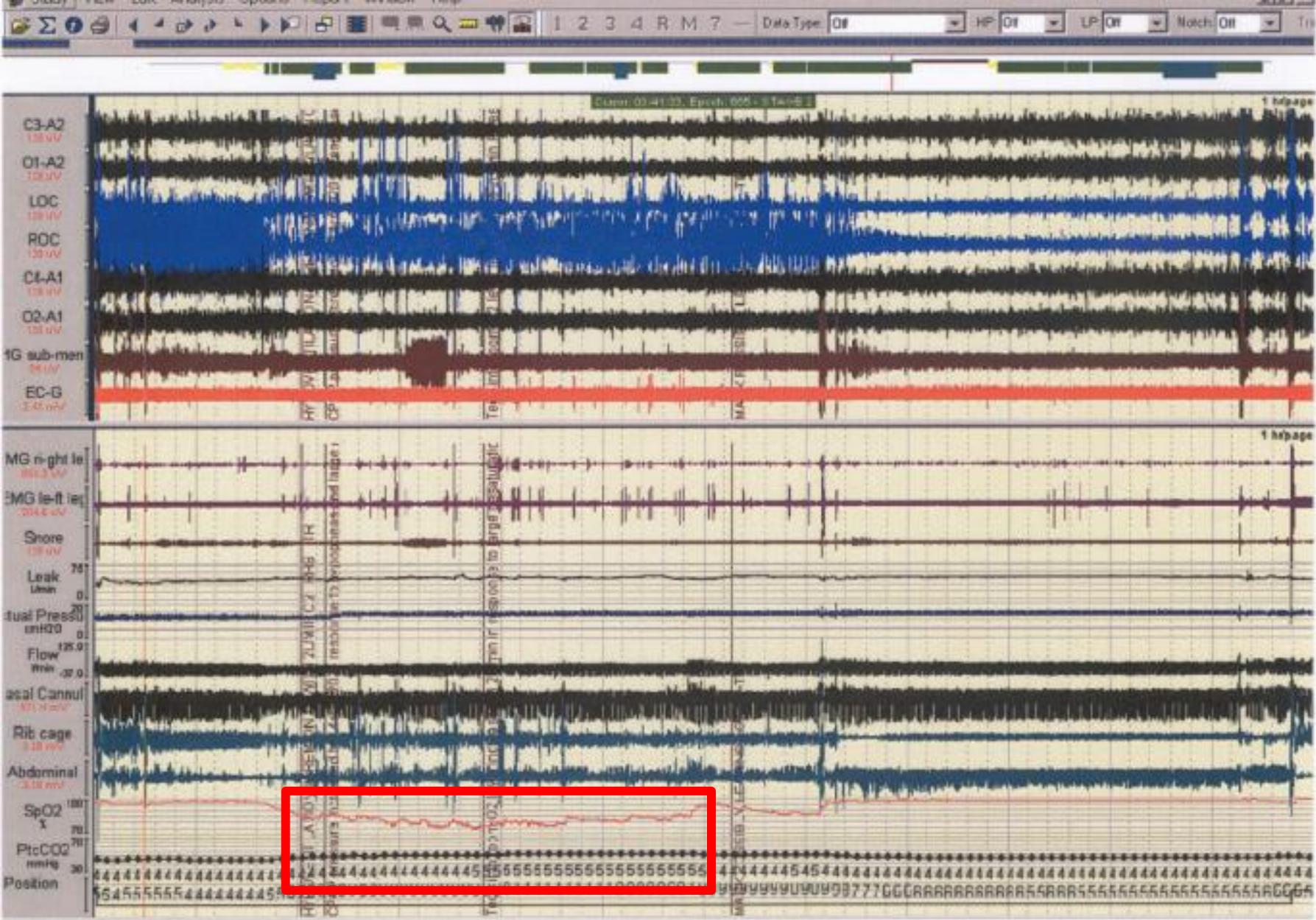


Cas clinique

Crummy, Thorax, 2008

- Homme de 22ans, 228 Kg, 158 cm, PaCO₂ diurne: 67mmHg





Mauvais contrôle de l'hypoventilation (hypoxie+hypercapnie) par CPAP seule pendant la phase REM



Millimètres de mercure

Le patient présente une hypoventilation réfractaire sous CPAP malgré une correction du SAOS. Quels paramètres pourraient améliorer son oxygénation?

A: Une augmentation de la P_{ep}

B: Passer à un mode à deux niveaux de pression

C: Augmenter l'oxygène

D: La réponse D

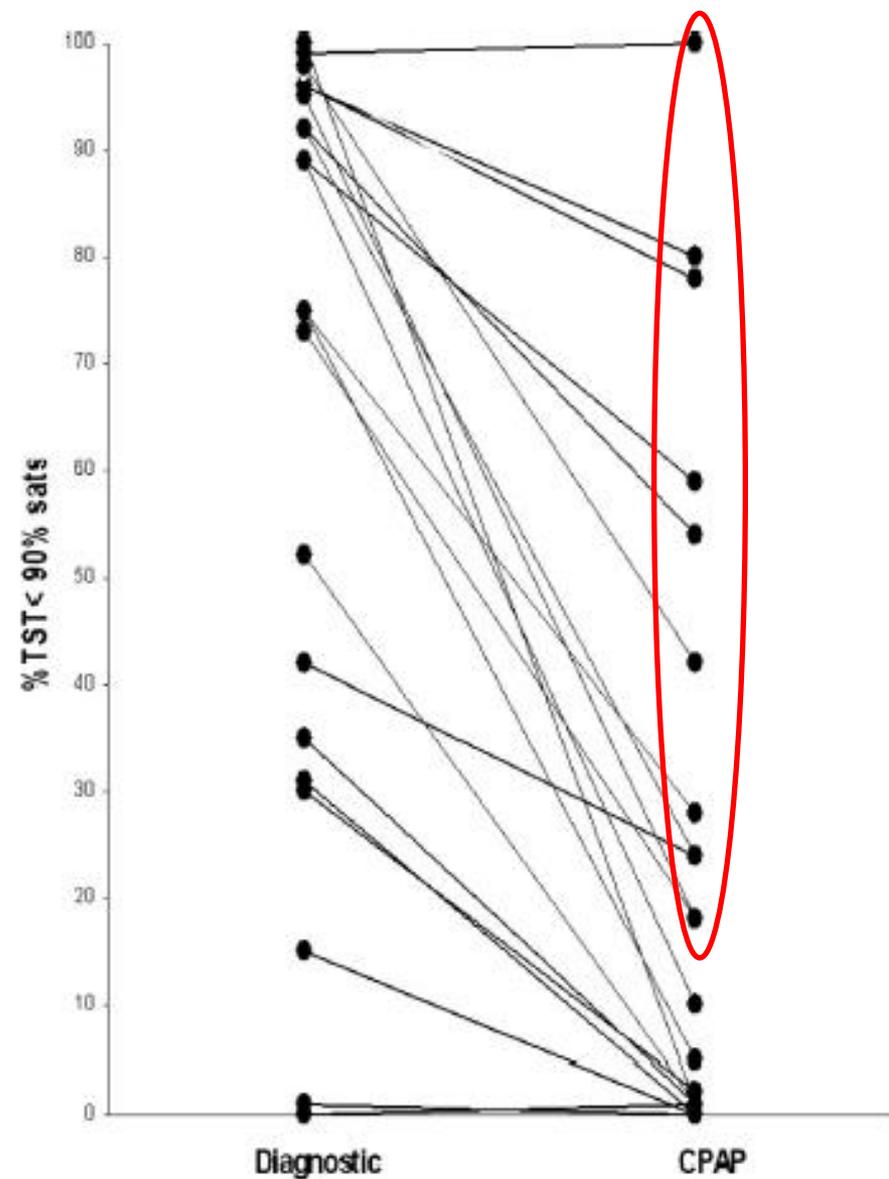
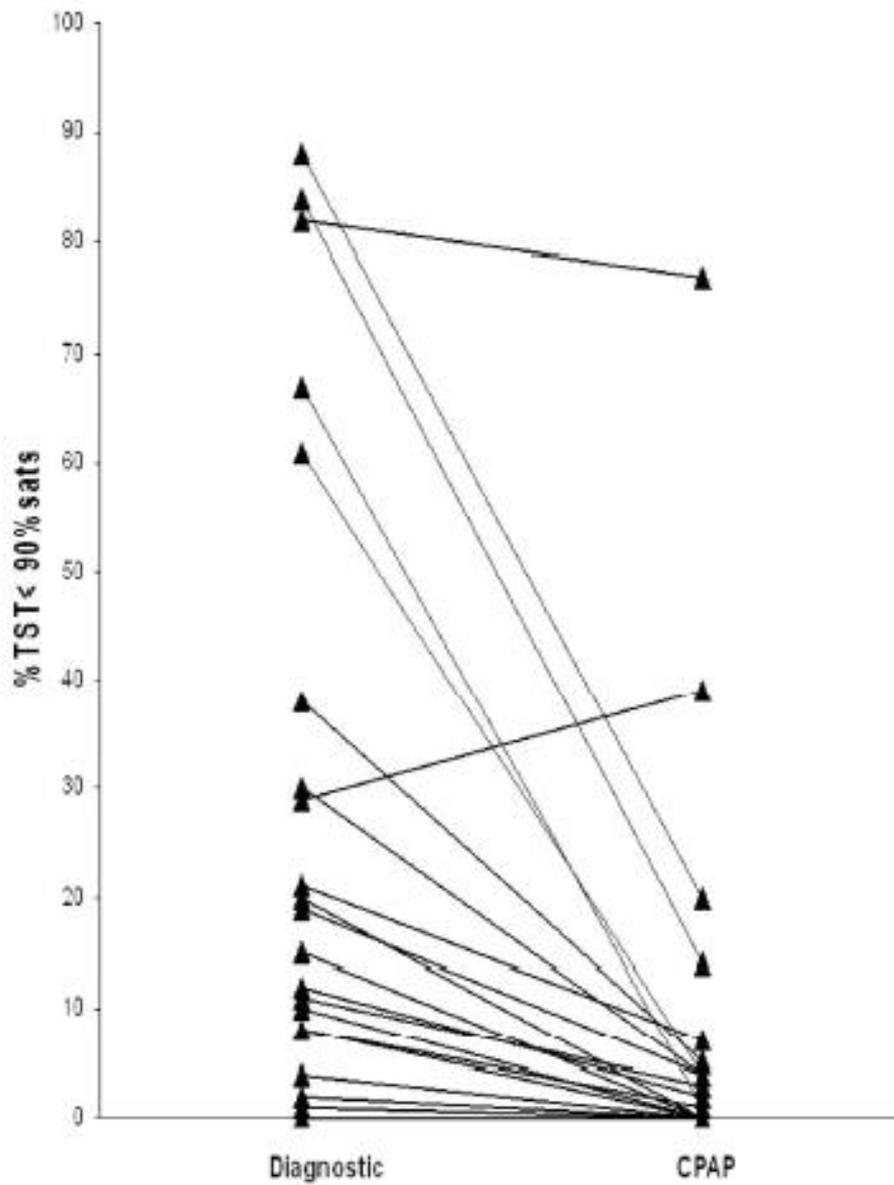


Table 4—Comparison of OHS Subjects Who Spend > 20% of TST With SpO₂ < 90% (n = 10) With Those Who Spend ≤ 20% of TST With SpO₂ < 90% (n = 13) During CPAP*

Parameters	>20% of TST With SpO ₂ < 90%	≤ 20% of TST With SpO ₂ < 90%	p Value
BMI	61.6 (1.7)	56.5 (1.2)	0.02
Percentage of TST with SpO ₂ < 90%	87 (5)	46 (10)	0.004
Pao ₂ , mm Hg	59.4 (2.1)	62.3 (1.5)	NS
Paco ₂ , mm Hg	55.5 (3.0)	53.3 (1.4)	NS
FEV ₁ , L	2.07 (0.23)	2.17 (0.23)	NS
FVC, L	2.55 (0.32)	2.79 (0.30)	NS
Percentage of REM (CPAP)	26.6 (5.0)	22.1 (2.3)	NS
REM AHI, /h	15.3 (2.9)	5.3 (0.8)	0.009
Total AHI, /h	25.1 (12.5)	9.8 (5.0)	NS
Percentage of TST with SpO ₂ < 90% (CPAP)	56.5 (8.4)	4.4 (1.8)	0.0001

*Data were normally distributed and presented as mean (SE). See Table 1 for expansion of abbreviation.



CHEST

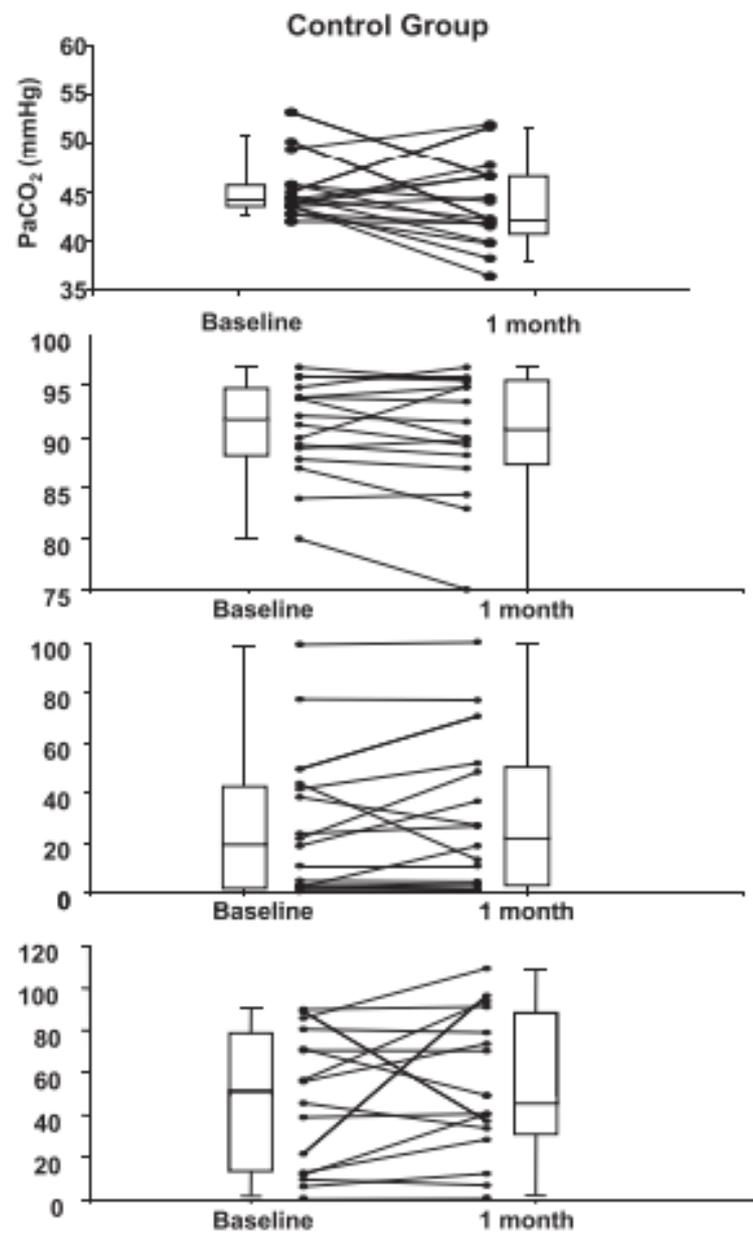
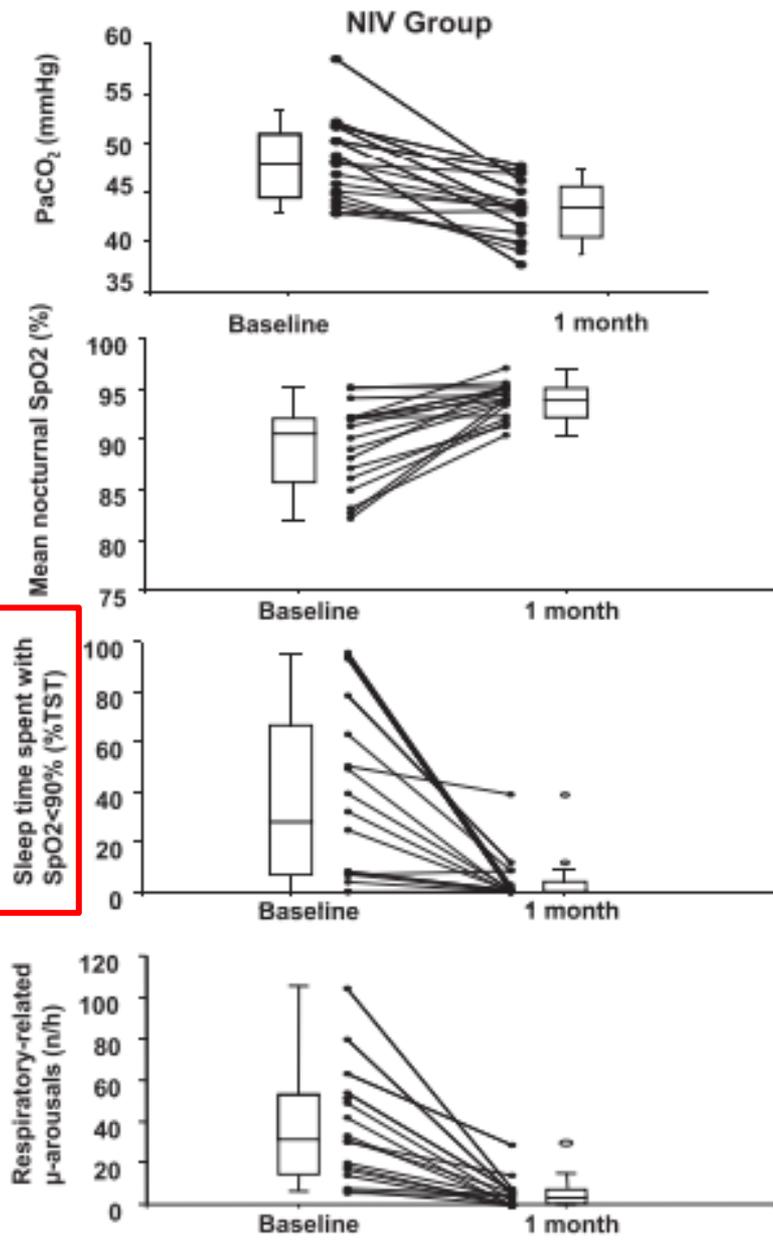
Original Research

SLEEP DISORDERS

Noninvasive Ventilation in Mild Obesity Hypoventilation Syndrome

A Randomized Controlled Trial

*Jean-Christian Borel, PhD; Renaud Tamisier, MD, PhD; Jesus Gonzalez-Bermejo, MD, PhD;
Jean-Philippe Baguet, MD, PhD; Denis Monneret, PharmD; Nathalie Arnol, MSc;
Pascale Roux-Lombard, MD; Bernard Wuyam, MD, PhD; Patrick Levy, MD, PhD;
and Jean-Louis Pépin, MD, PhD*





Millimètres de mercure

Quels réglages en plus de la Pep pour une ventilation à deux niveaux de pression?

A: Favoriser une A_i élevée

B: Un mode à volume cible

C: Une aide inspiratoire et une fréquence de sécurité

D: La réponse D

Berger ¹³ 2001	group 2: 12 patients BPAP Rx	Piper ²² 2007	CPAP mean pressure was 14 cm H ₂ O
Retrospective Case Series	IPAP 18 cm H ₂ O range (12 to 25) cm H ₂ O	RCT	BPAP S mode mean
Level IV	EPAP 8 range (3 to 14)	Level I CPAP vs BPAP	IPAP = 16 cm H ₂ O EPAP = 10 cm H ₂ O
Budweiser ¹⁸ 2007	BPAP ST	Redolfi ²⁰ 2006	BPAP Synchrony device
Survival of OHS on NPPV	IPAP 22.5 ± 3.6 mbar EPAP 5.8 ± 3.1 mbar backup rate 19.2/min	Leptin in OHS	mean pressures
Case Series		prospective case controlled	IPAP = 12 EPAP = 4
De Lucas- Ramos ¹⁹ 2004	end IPAP = 19 ± 2 EPAP of 5 cm H ₂ O	Level IV	
Case Series		Guo ²¹ 2007	BPAP ST mode used
Level IV		Level IV	mean IPAP = 18.5 ± 4.6 mean EPAP = 6.2 ± 1.4 mean backup rate = 13.7 ± 2.2 bpm

NIV Settings and Compliance to NIV

Mean inspiratory and expiratory therapeutic pressures were 18 ± 3 cm H₂O and 11 ± 2 cm H₂O, respectively (mean pressure support, 8 ± 2 cm H₂O); mean back-up rate breathing frequency was set at 13 ± 2 cycles/min. None of the patients received additional oxygen therapy. At 1 month, the mean use of nocturnal NIV was 5.6 ± 2.2 h/night. Four of the 18 patients in the NIV group used the device < 4 h per night.

Table 1—Characteristics of Patients Included and Usual Ventilator Settings

Characteristic	Mean	SD	% Predicted	SD
Age, y	55.7	9.2
BMI, kg/m ²	48.5	5.1
TLC, L	5.0	1.5	82	18
FVC, L	3.2	1.1	86	22
FEV ₁ , L	2.6	0.8	87	23
FEV ₁ /FVC	0.82	0.05	100	1
Arterial blood gases				
pH	7.44	0.03
PaCO ₂ , mm Hg	41.3	5.6
PaO ₂ , mm Hg	72.7	10.9
HCO ₃ ⁻ , mmol/L	28.1	2.6
Baseline ventilator settings				
Duration of NPPV, mo	42.7	46.8
Compliance, h/min	7:16	1:44
BURR, ^a cycles/min	14	1.5
IPAP, cm H ₂ O	20.5	3.7
EPAP, cm H ₂ O	9.2	1.8
Rise time, ms	155	65
T _{imin} , s	0.7	0.06
T _{imax} , s	1.3	0.1

Et la fréquence respiratoire?



CHEST

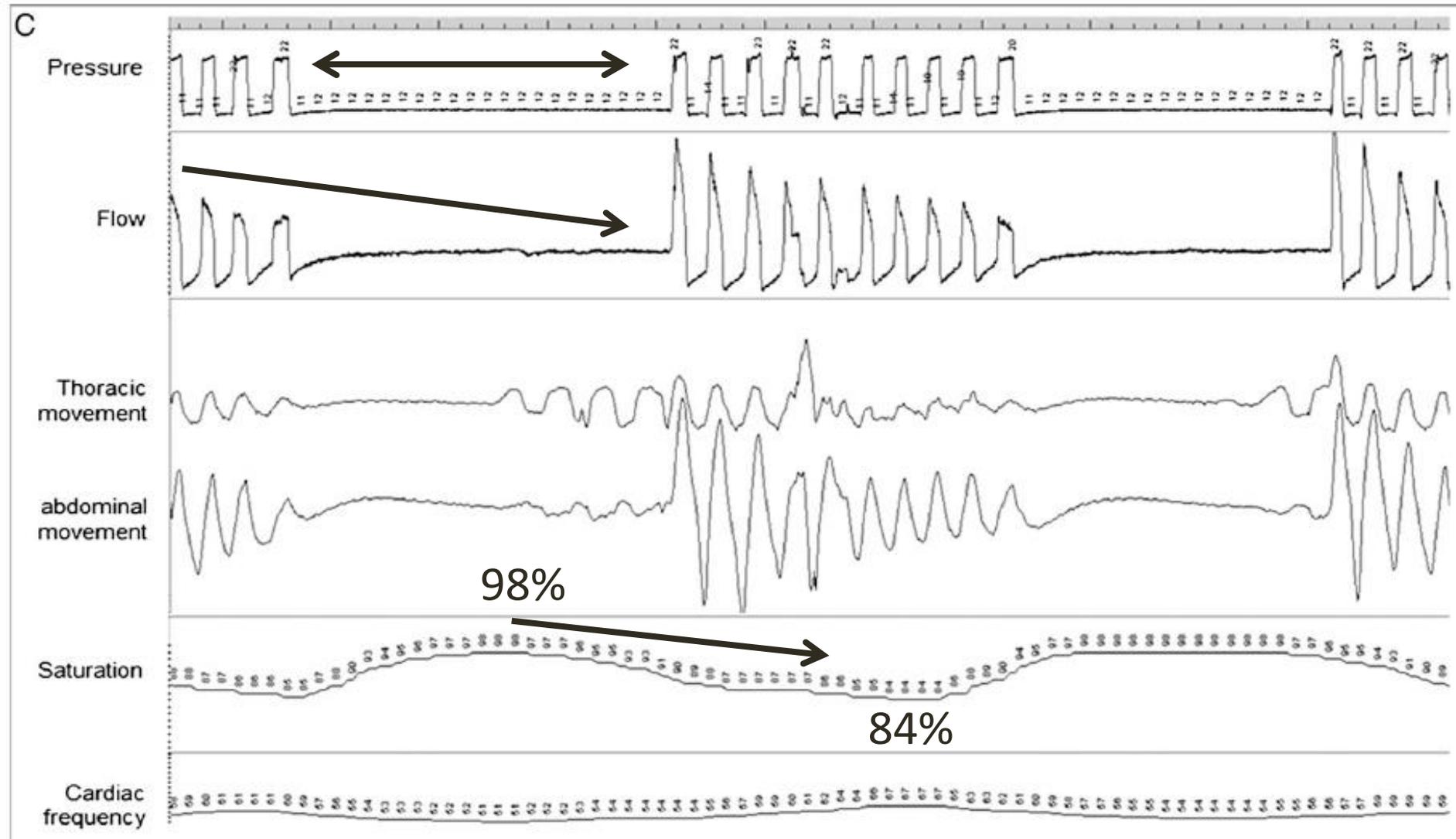
Original Research

SLEEP DISORDERS

Impact of Different Backup Respiratory Rates on the Efficacy of Noninvasive Positive Pressure Ventilation in Obesity Hypoventilation Syndrome

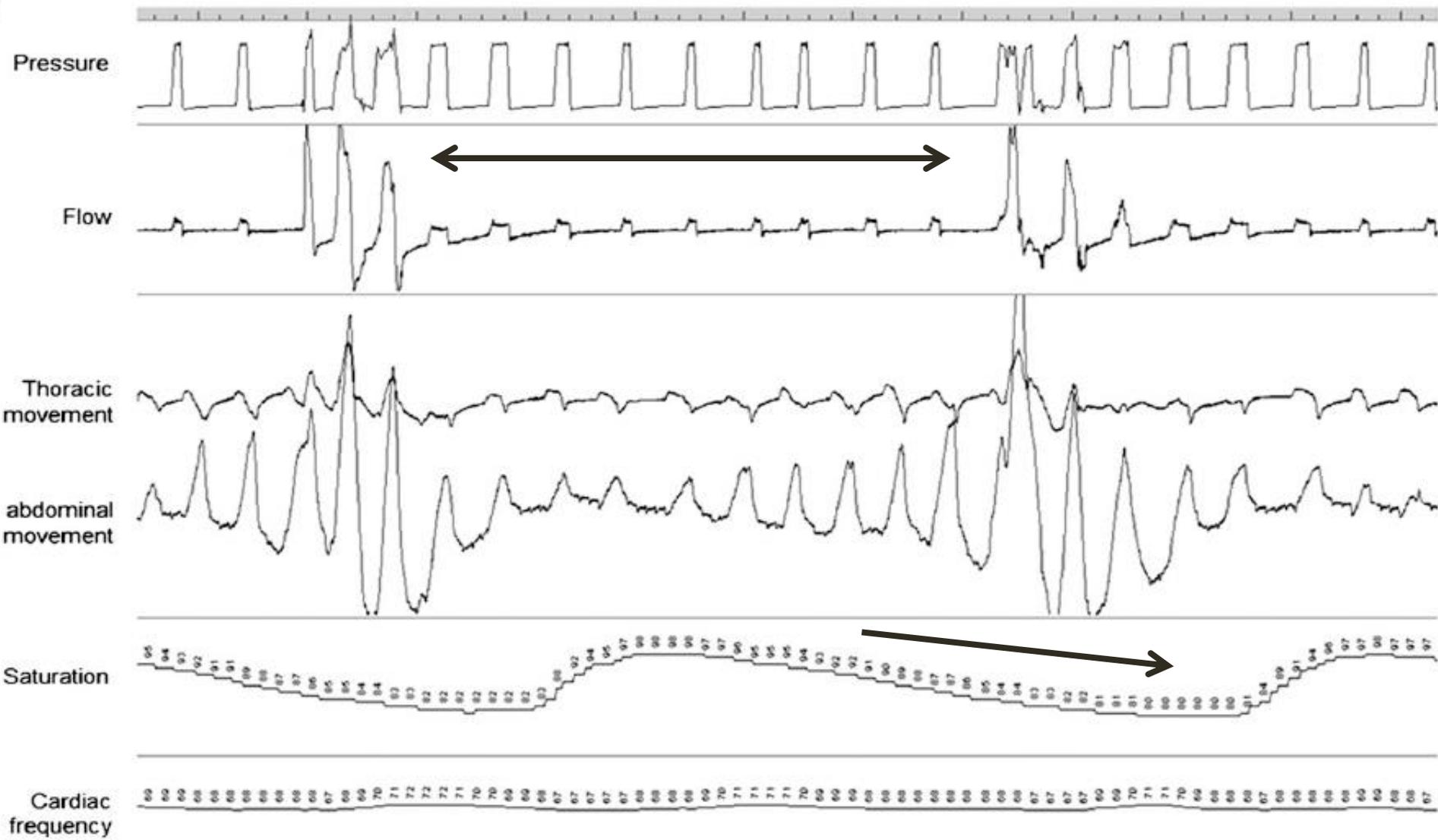
A Randomized Trial

Olivier Contal, PhD, PT; Dan Adler, MD; Jean-Christian Borel, PhD; Fabrice Espa, RPSGT; Stephen Perrig, MD; Daniel Rodenstein, MD; Jean-Louis Pépin, MD; and Jean-Paul Janssens, MD

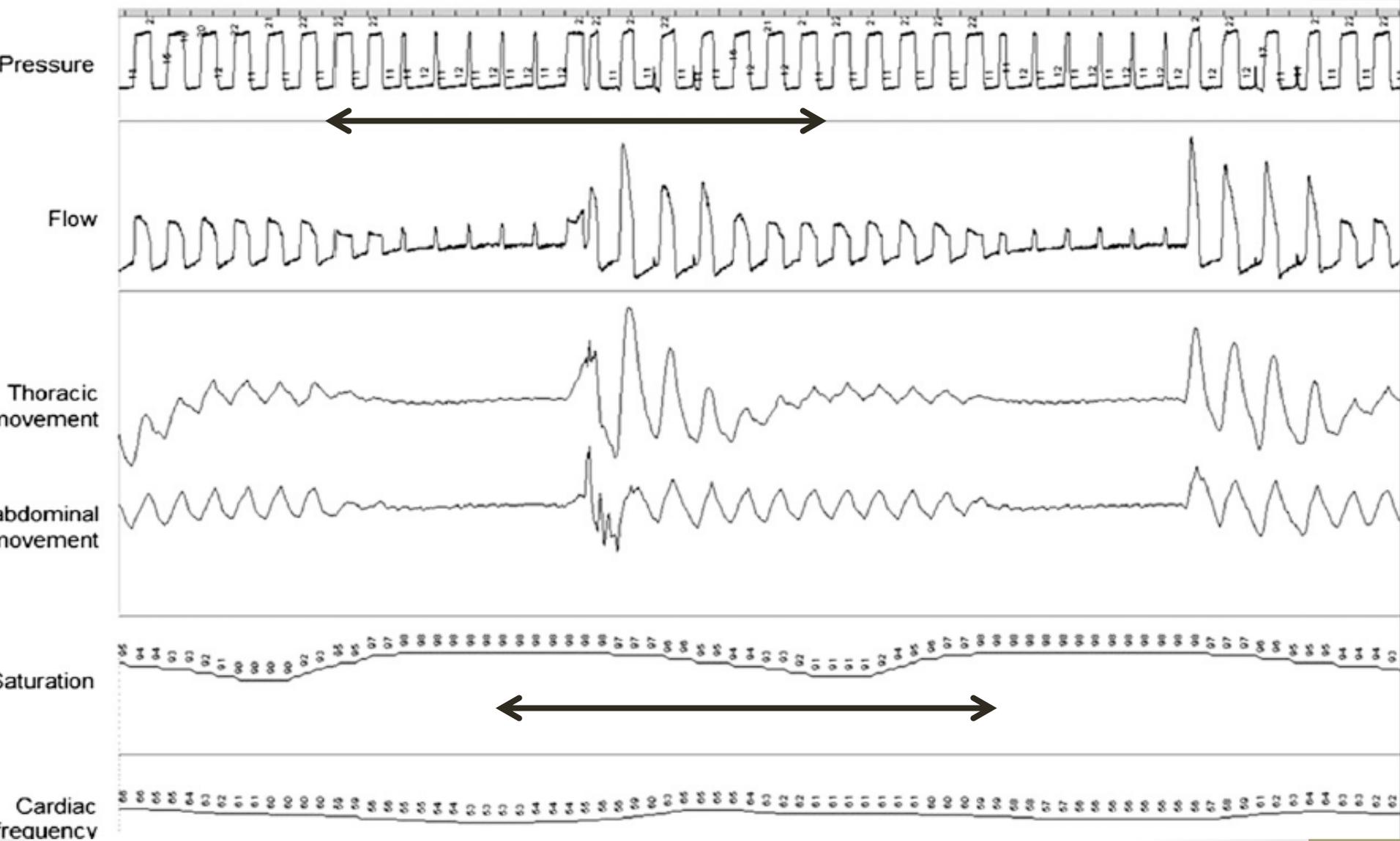


Fréquence de sécurité = 0

3

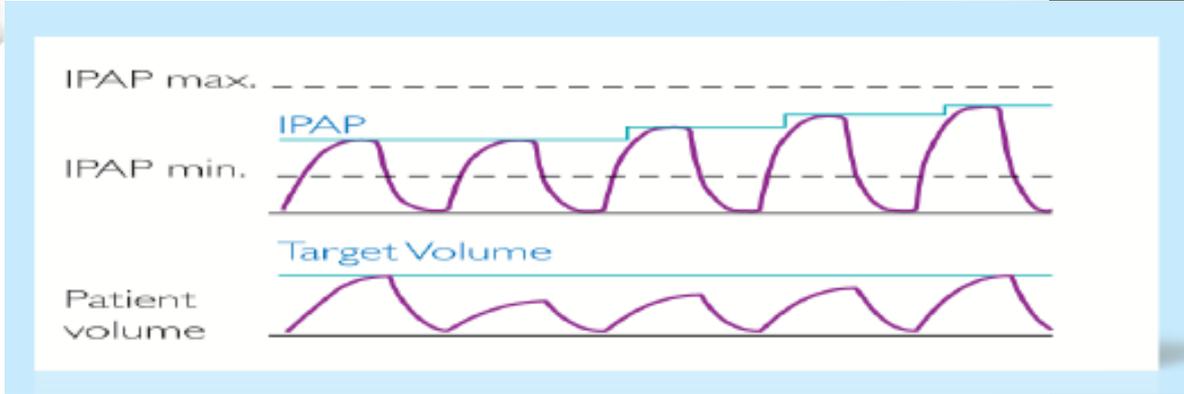


Fréquence de sécurité = 11

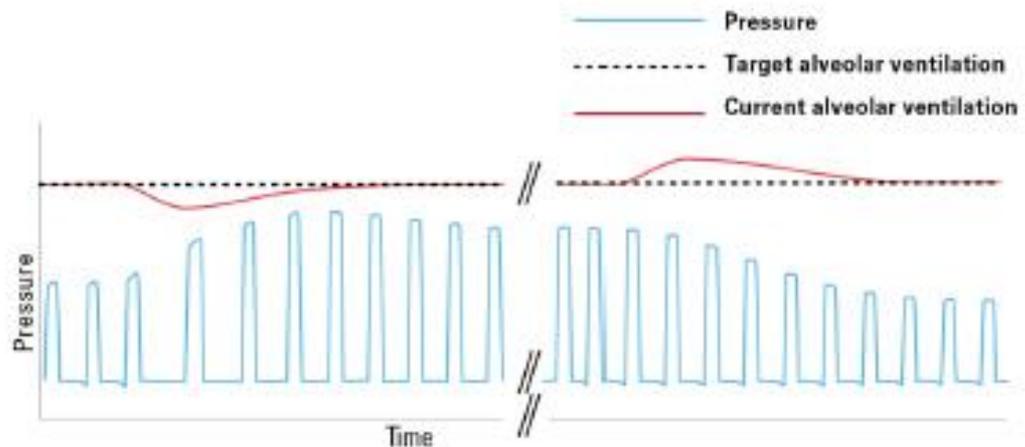


Fréquence de sécurité = 21

Mode à volume cibles



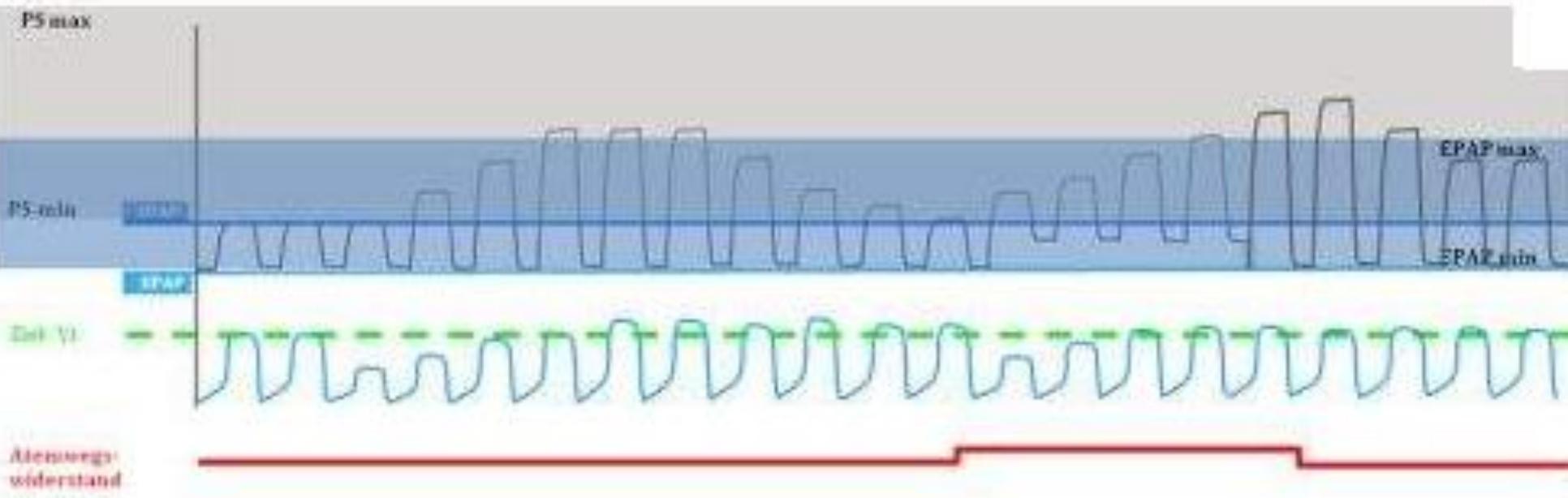
Average Volume Assured Pressure Support Ventilation

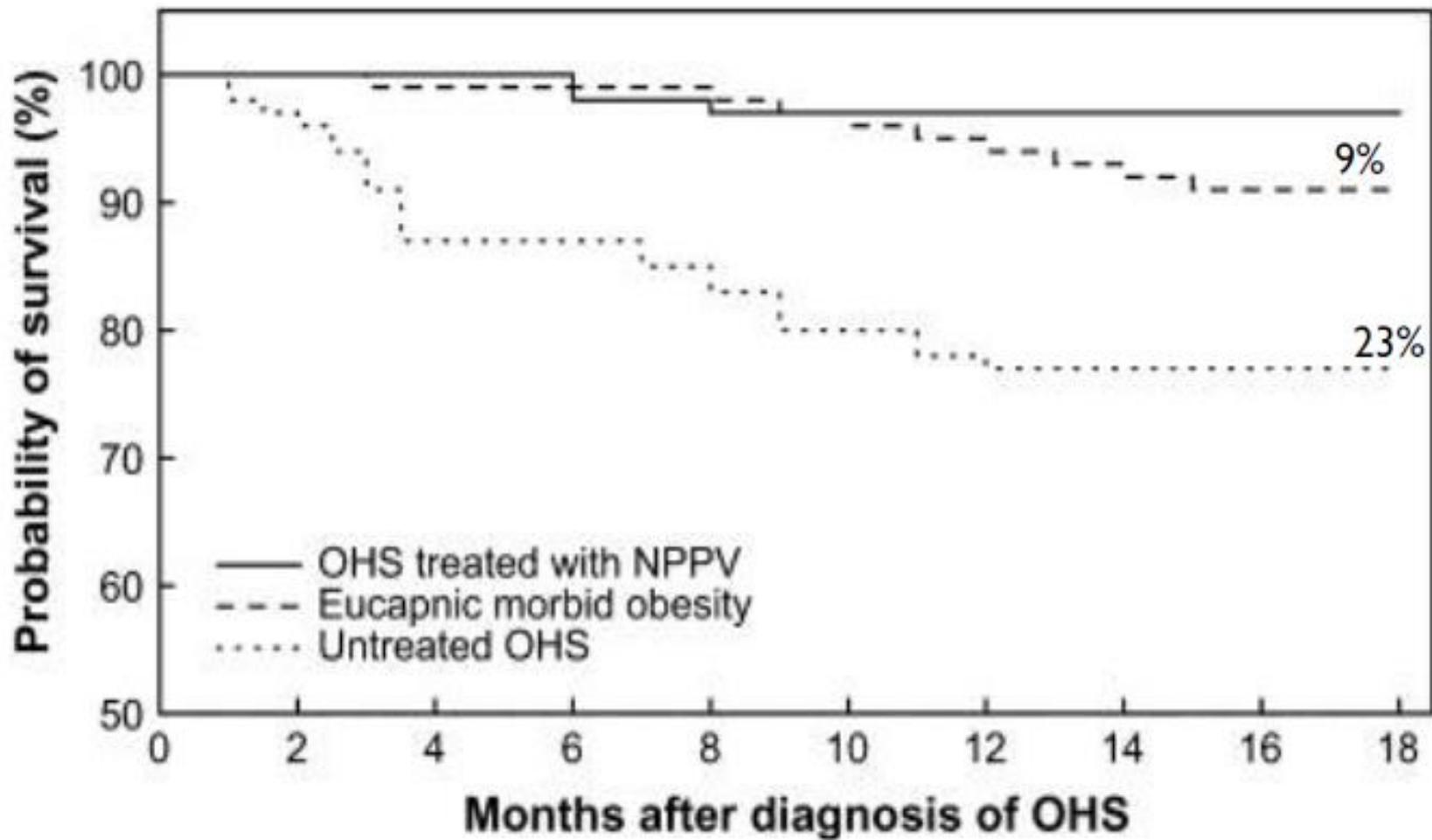


Intelligent Volume Assured Pressure Support

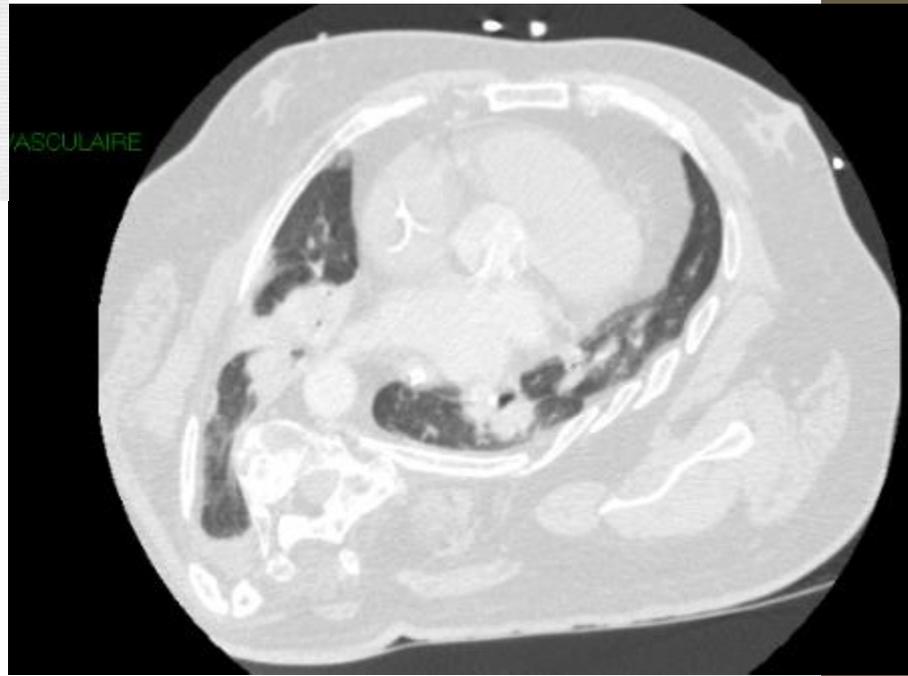
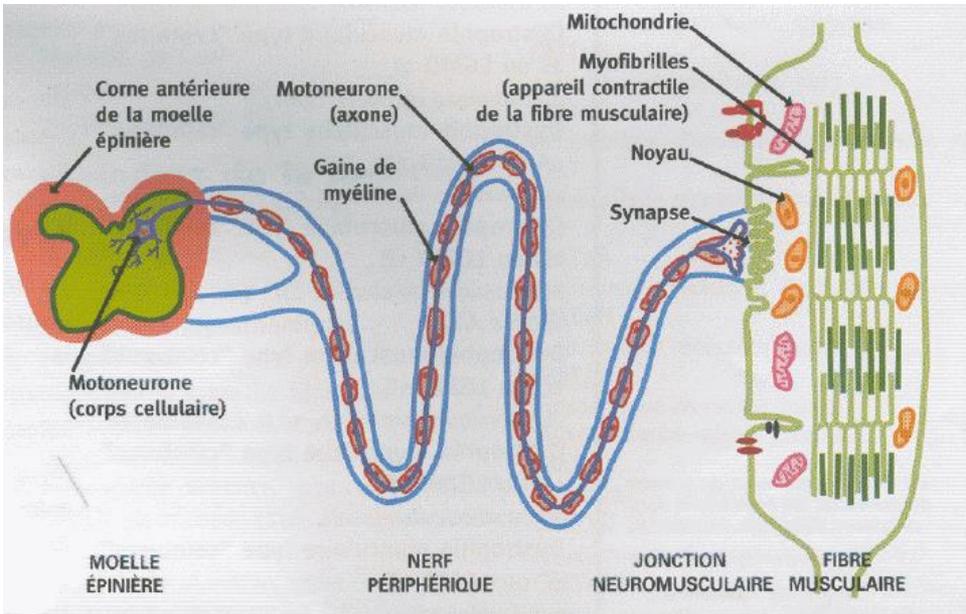


Mode AVAPS-AE?

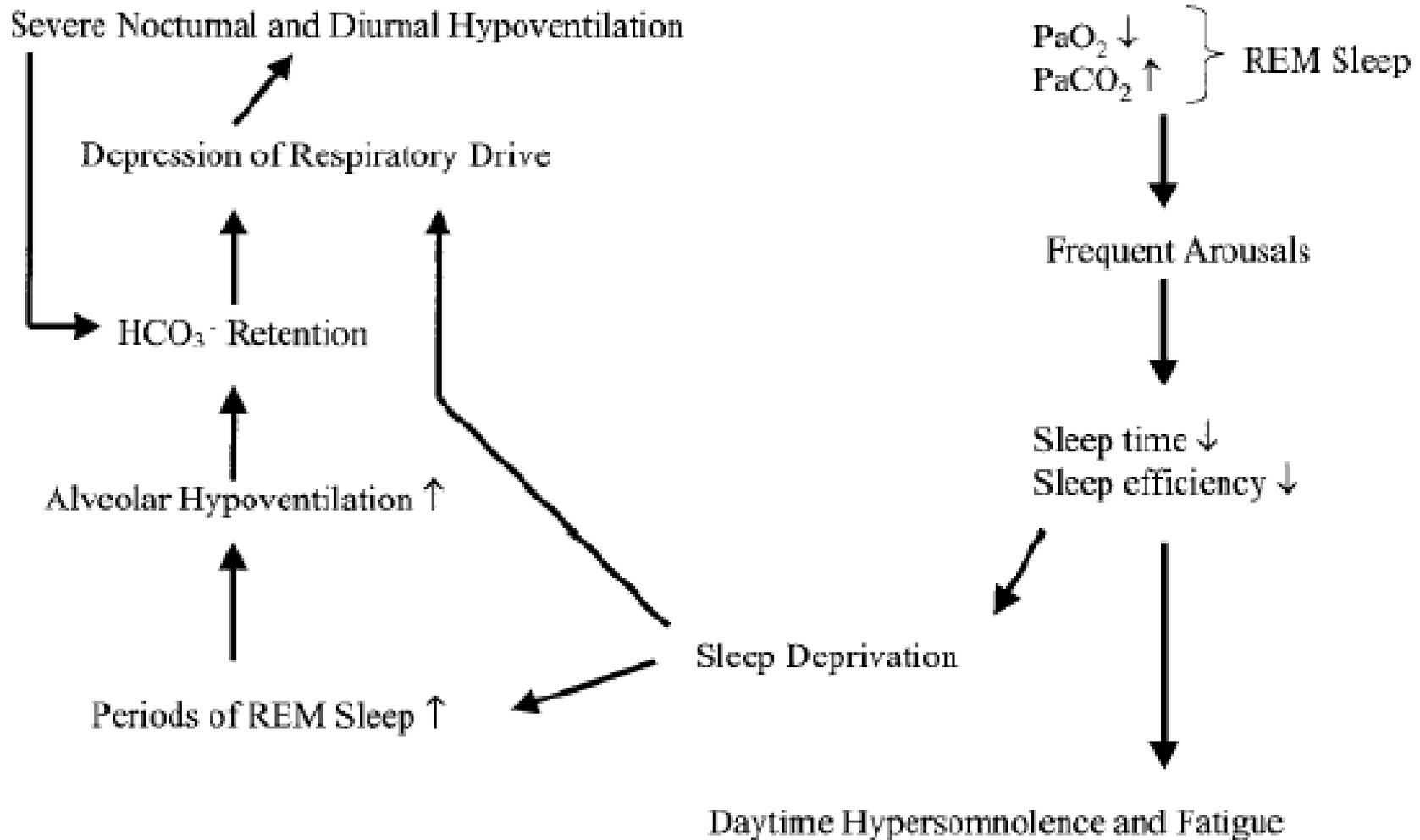




Maladie Neuromusculaire et thoraco-restrictive



NEUROMUSCULAR WEAKNESS



RLD

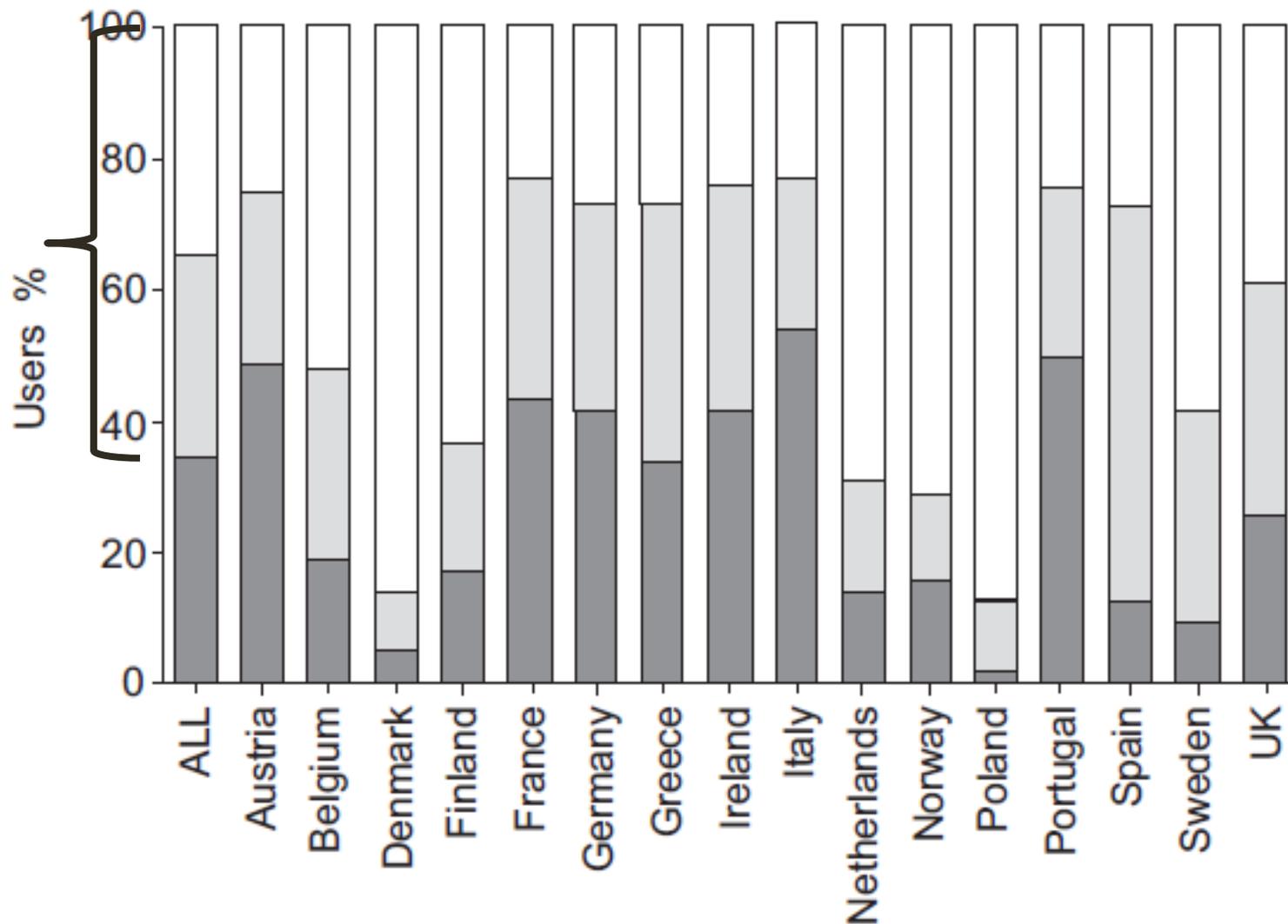


FIGURE 4. Percentage of users in each disease category by country (see *Methods* section for an explanation of disease categories). ■: lung/airways; ▒: thoracic cage; □: neuromuscular.



Millimètres de mercure

Quels sont les effets de la VNI dans les pathologies Neuromusculaires et thoraco-restrictives?

A: Améliore l'espérance de vie

B: Détérioré la qualité du sommeil

C: Améliore la capacité vitale et la force des muscles respiratoires

D: La réponse D

Kyphoscoliosis

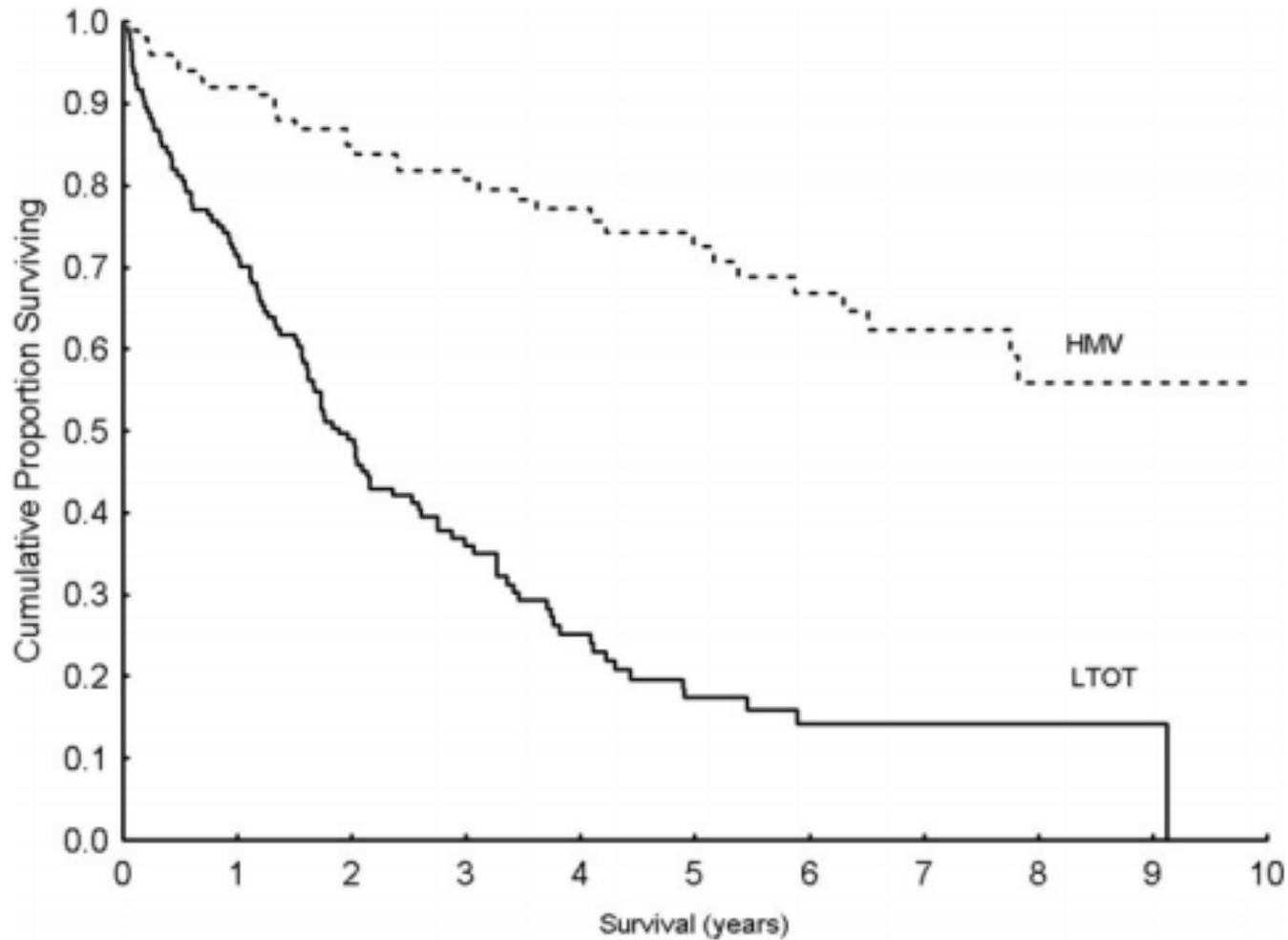


FIGURE 2. Cumulative proportion of surviving patients receiving HMV or LTOT alone.

Et les autres pathologies?

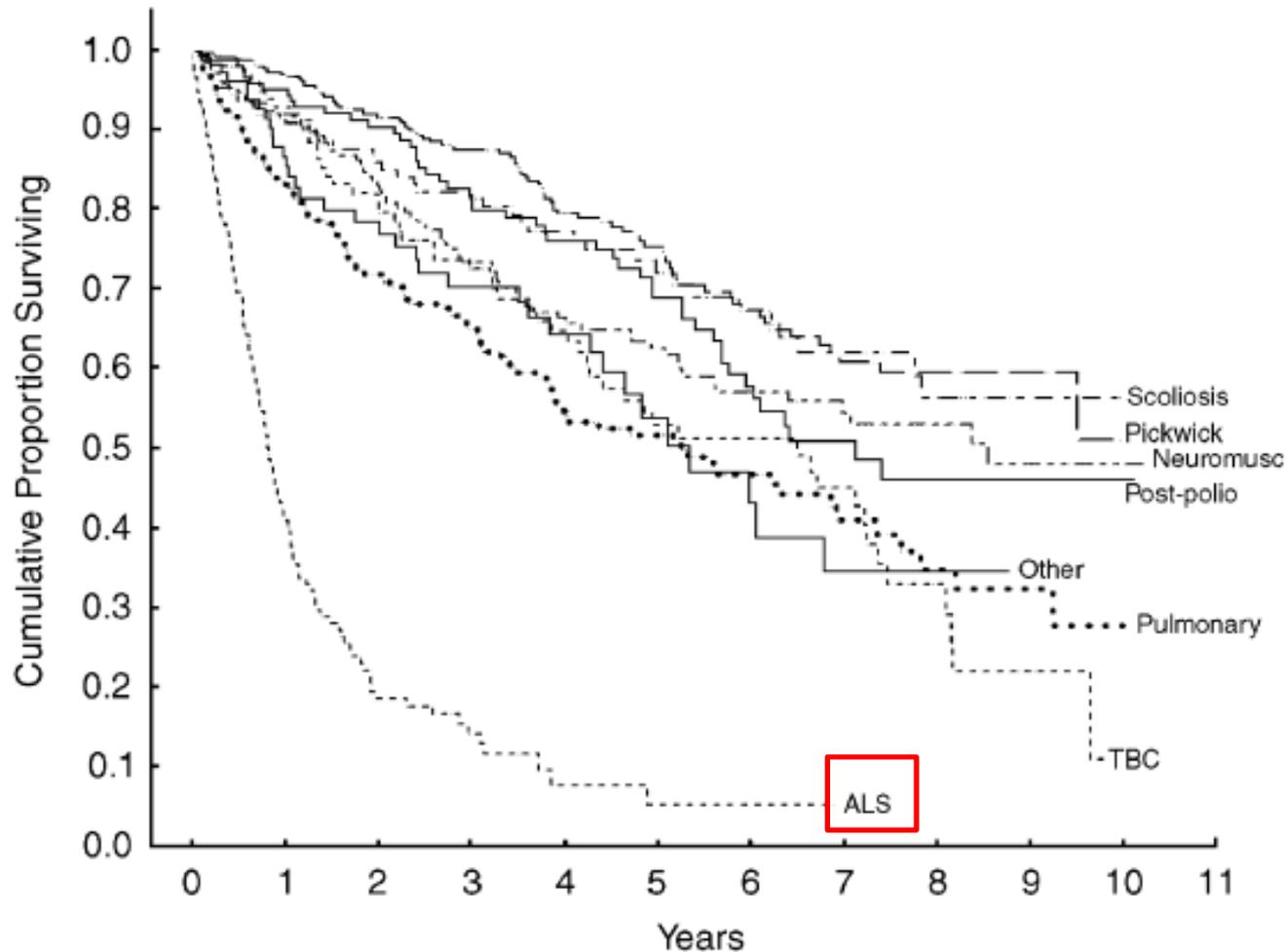
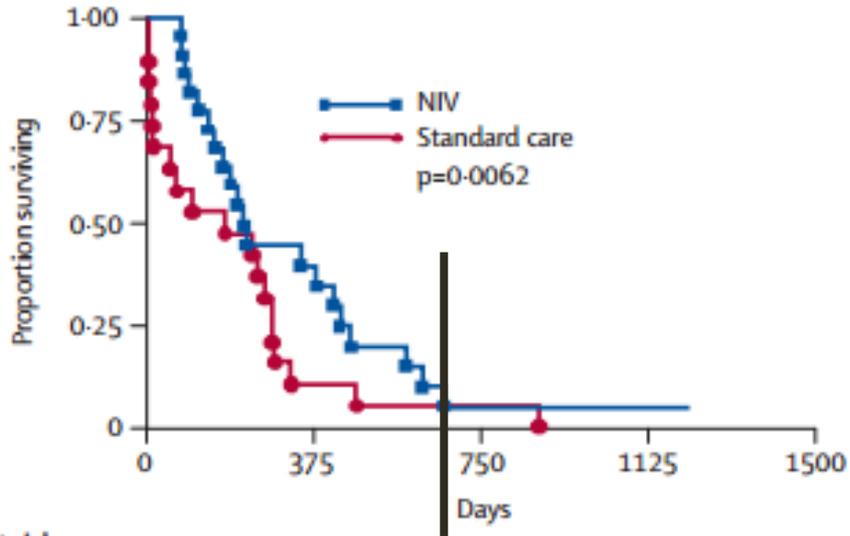


Figure 1 Probability to survive in 1526 adult patients after starting HMV.

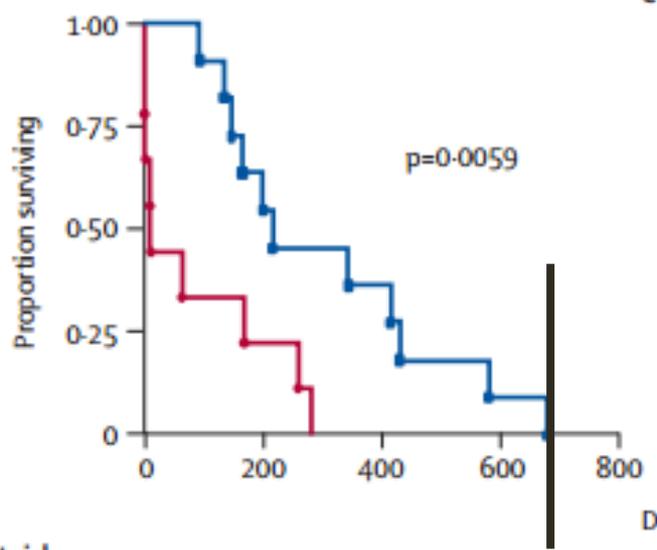
A



Numbers at risk

NIV	22
Standard care	19

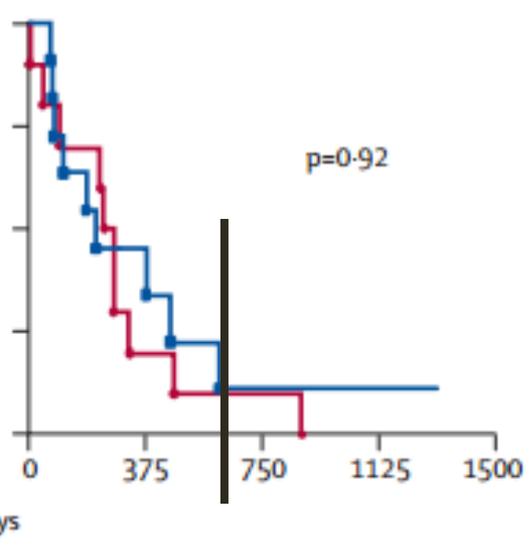
B



Numbers at risk

NIV	11
Standard care	9

C



Numbers at risk

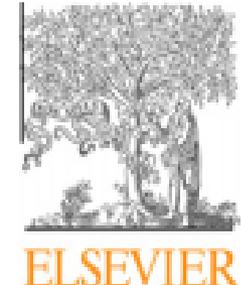
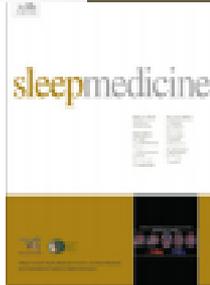
NIV	11
Standard care	10

Qualité du sommeil?

Contents lists available at [ScienceDirect](#)

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep



Original Article

Sleep in ventilatory failure in restrictive thoracic disorders. Effects of treatment with non invasive ventilation

Olivier Contal^a, Jean-Paul Janssens^a, Myriam Dury^b, Pierre Delguste^b, Geneviève Aubert^b, Daniel Rodenstein^{b,*}

^aDivision of Pulmonary Diseases, Geneva University Hospitals, 1211 Geneva 14, Switzerland

^bService de pneumologie et Centre de médecine du sommeil, Cliniques Universitaires Saint Luc, Université Catholique de Louvain, 1200 Brussels, Belgium

Table 4

Results of initial PSG and PSG under NIV.

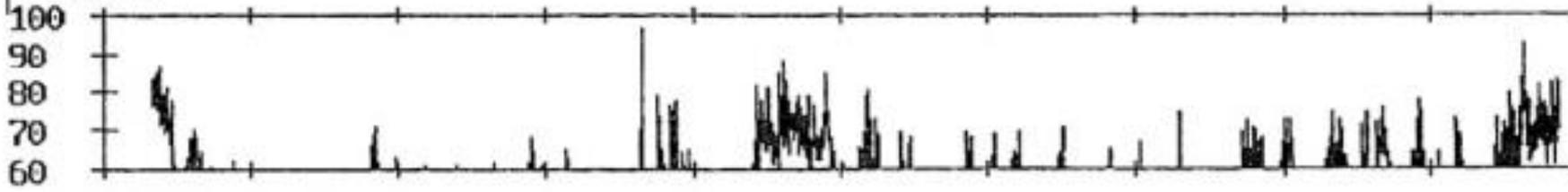
	N	Mean before NIV	Mean with NIV	Difference between before and after NIV	95% CI	p value
Total sleep time (min)*	54	360	361	1	-31 ; 32	0.98
Sleep efficiency (%)	55	64	71	7	2 ; 12	0.004
Sleep latency (min)	55	43	28	-15	-30 ; 1	0.07
REM (% of TST)	60	12	18	6	2 ; 9	0.002
Sleep stage 1 (% of TST)	60	26	14	-12	-18 ; -5	0.001
Sleep stage 2 (% of TST)	60	40	41	1	-5 ; 7	0.83
SWS (% of TST)	55	22	30	8	-0.02 ; 15	0.051
Movement arousal index	57	25	14	-11	-18 ; -4	0.003
Mean SpO ₂ %	59	85	93	8	4 ; 10	<0.001
Lowest SpO ₂ %	56	63	77	14	10 ; 18	<0.001
Oxygen desaturation index	43	37	13	-24	-33 ; -15	<0.001

TST: total sleep time* (6 split-night studies were excluded for this parameter); REM: rapid-eye movement; SWS, slow wave sleep; SpO₂: oxyhemoglobin saturation; 95% CI: 95% confidence interval.

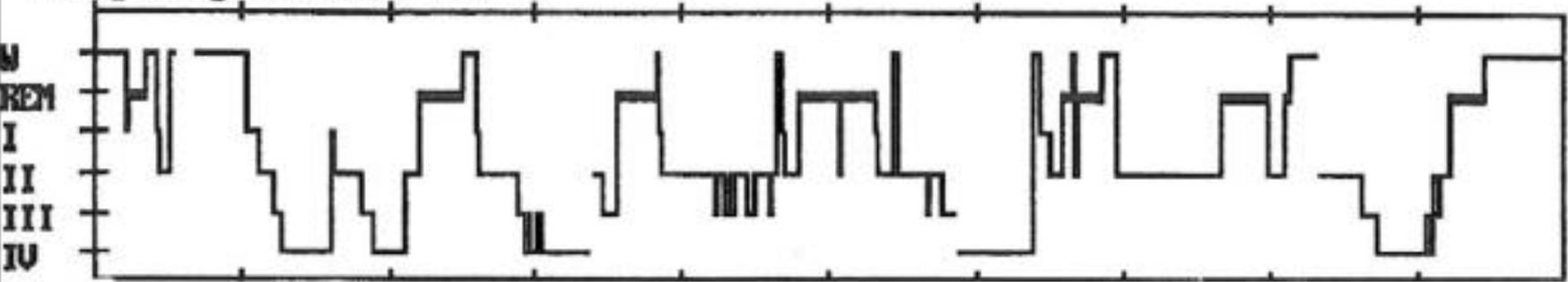
Sleep Stage (MA/hS: 14)



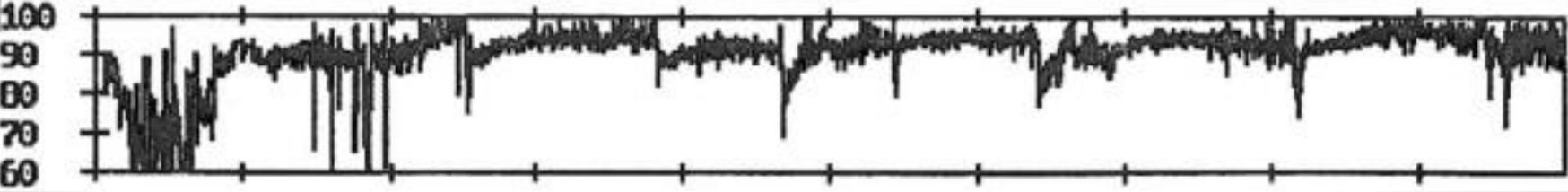
SaO2 %

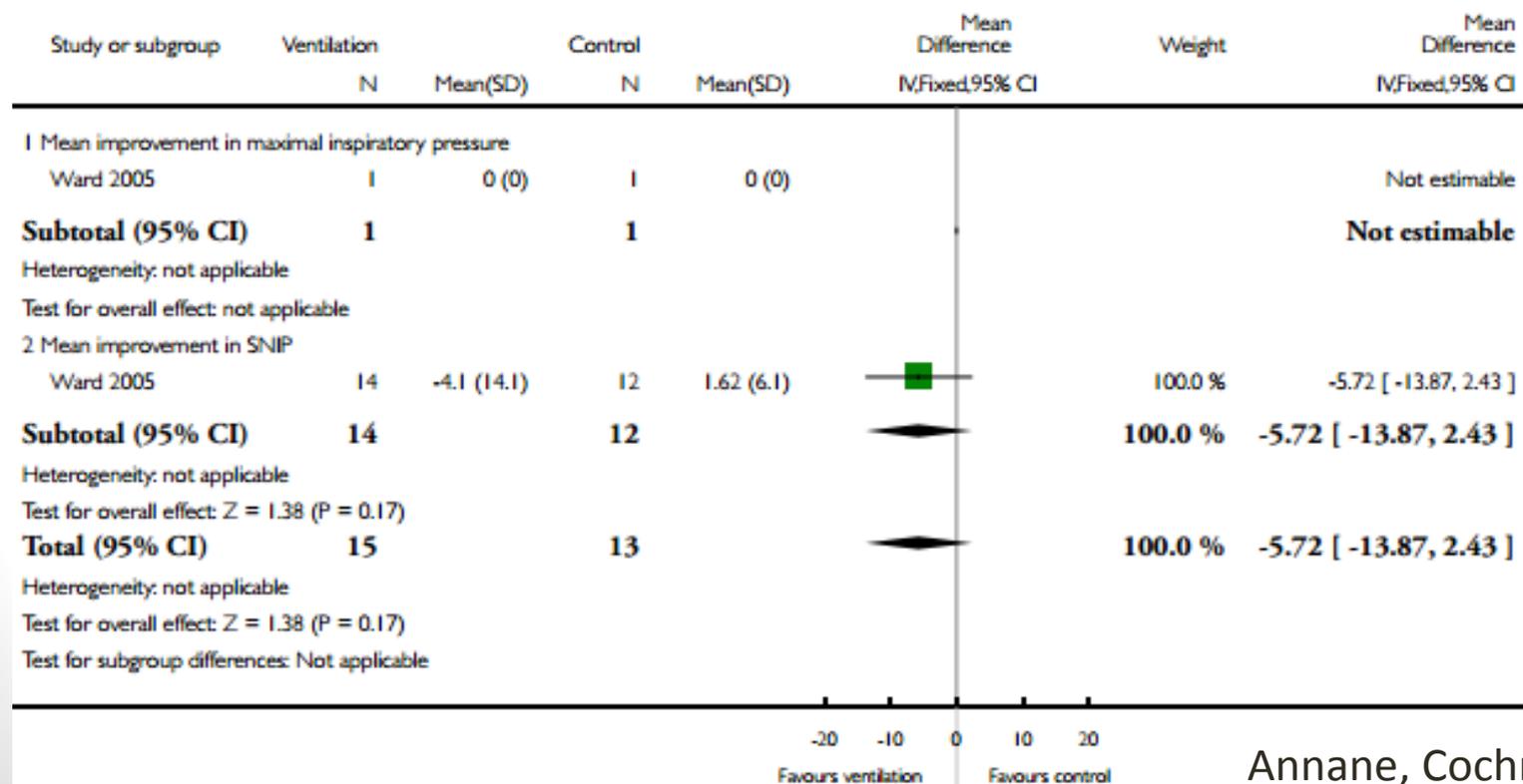
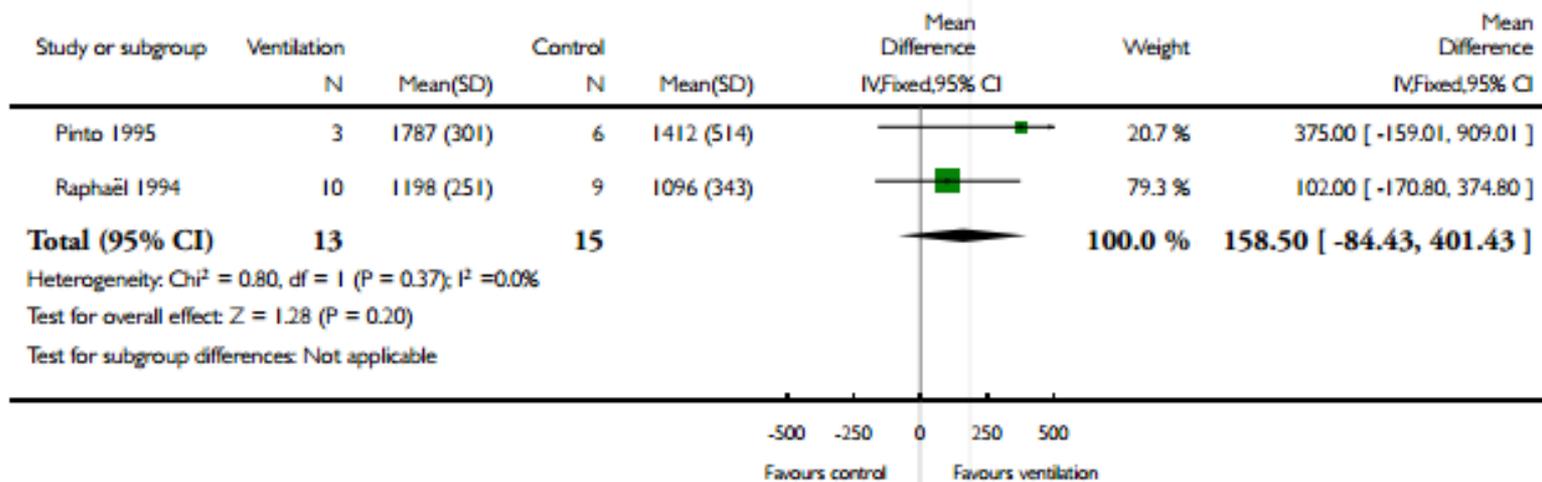


Sleep Stage (MA/hS: 2)



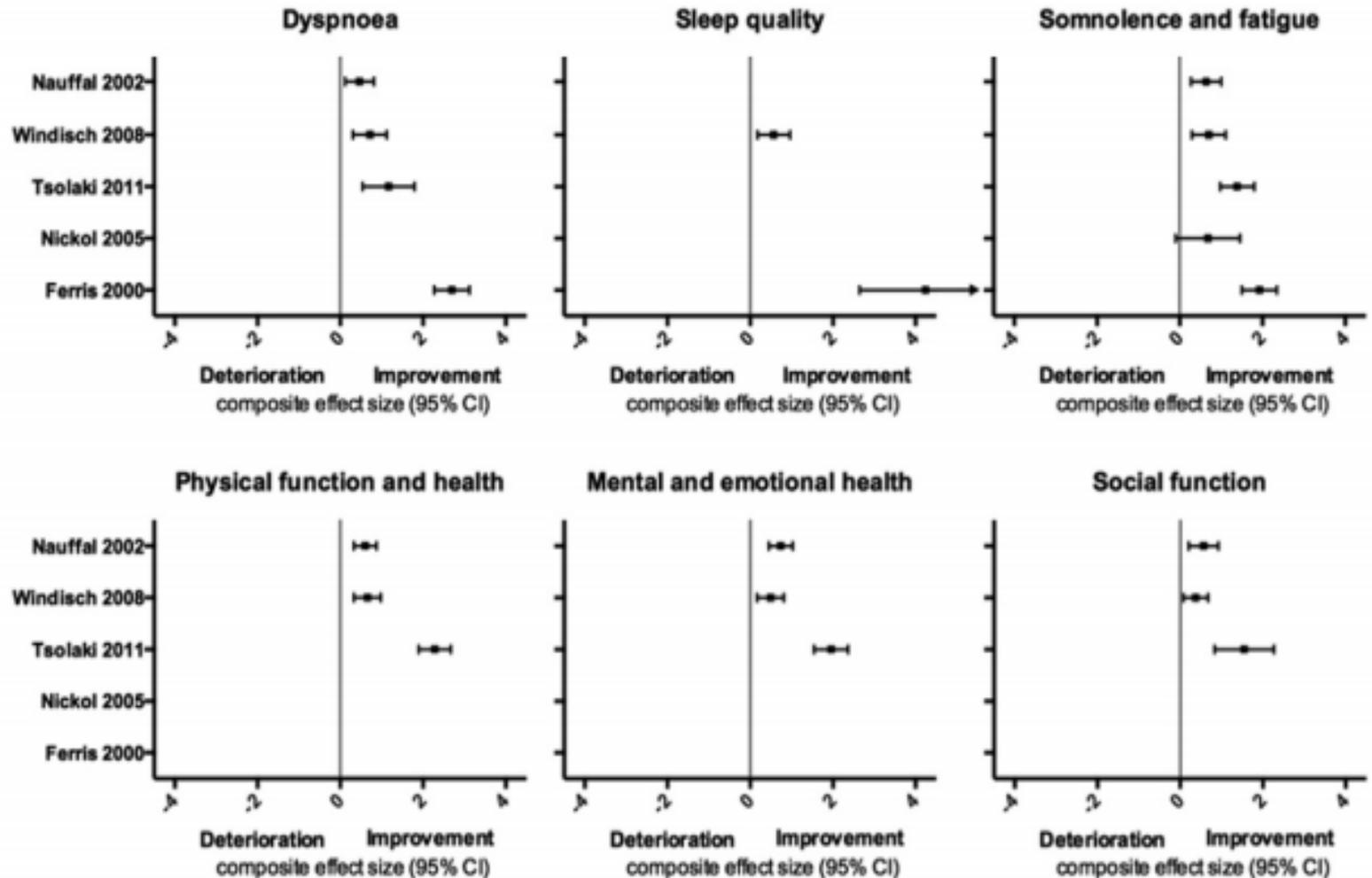
SaO2 %





Autres paramètres?

(b) - Restrictive thoracic disease.



BPCO

- Première description de l'intérêt de la VNI dans la décompensation du BPCO en 1995.
- Aujourd'hui, la VNI est acceptée à l'unanimité dans la décompensation respiratoire hypercapnique.

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NONINVASIVE VENTILATION FOR ACUTE EXACERBATIONS OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

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GIORGIO CONTI, M.D., ALAIN RAUSS, M.D., GÉRALD SIMONNEAU, M.D., SALVADOR BENITO, M.D.,
ALESSANDRO GASPARETTO, M.D., FRANÇOIS LEMAIRE, M.D., DANIEL ISABEY, PH.D., AND ALAIN HARF, M.D.



Millimètres de
mercure

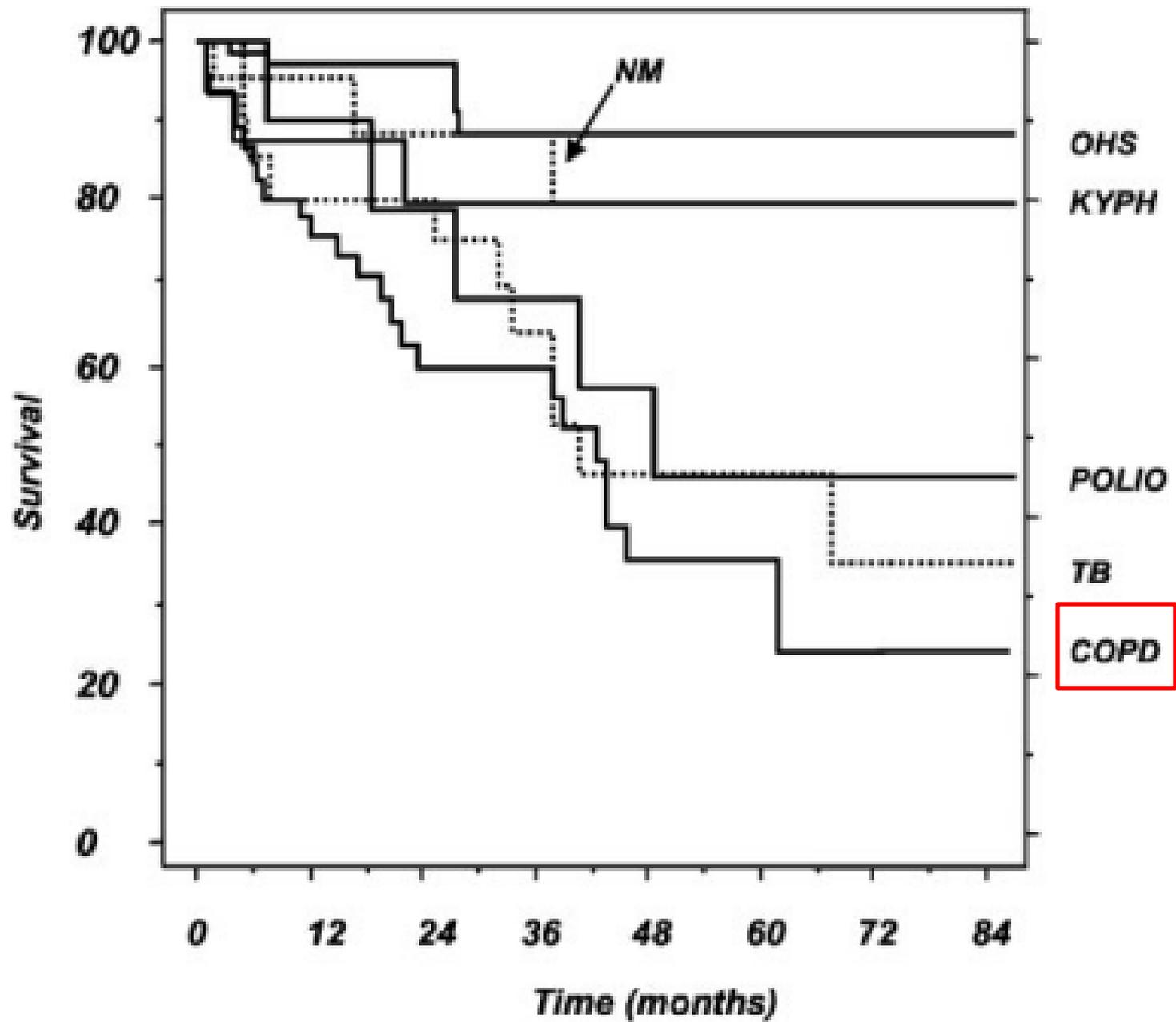
Quels sont les effets de la VNI au long
court chez les BPCO?

A: Diminue la mortalité

B: Diminution de la PaCO₂

C: Améliore la qualité de vie

D: La réponse D



VNI Vs O2?

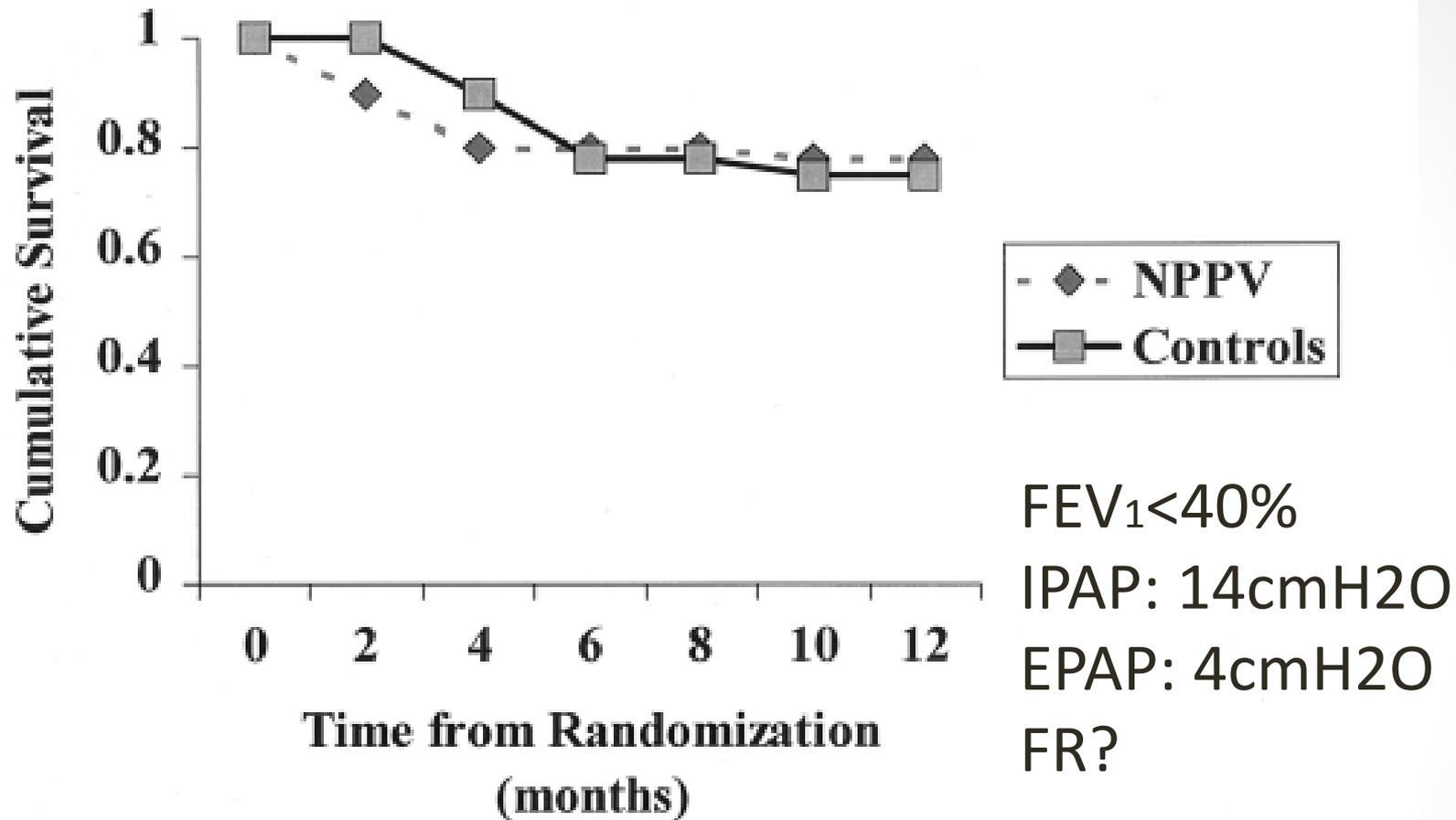
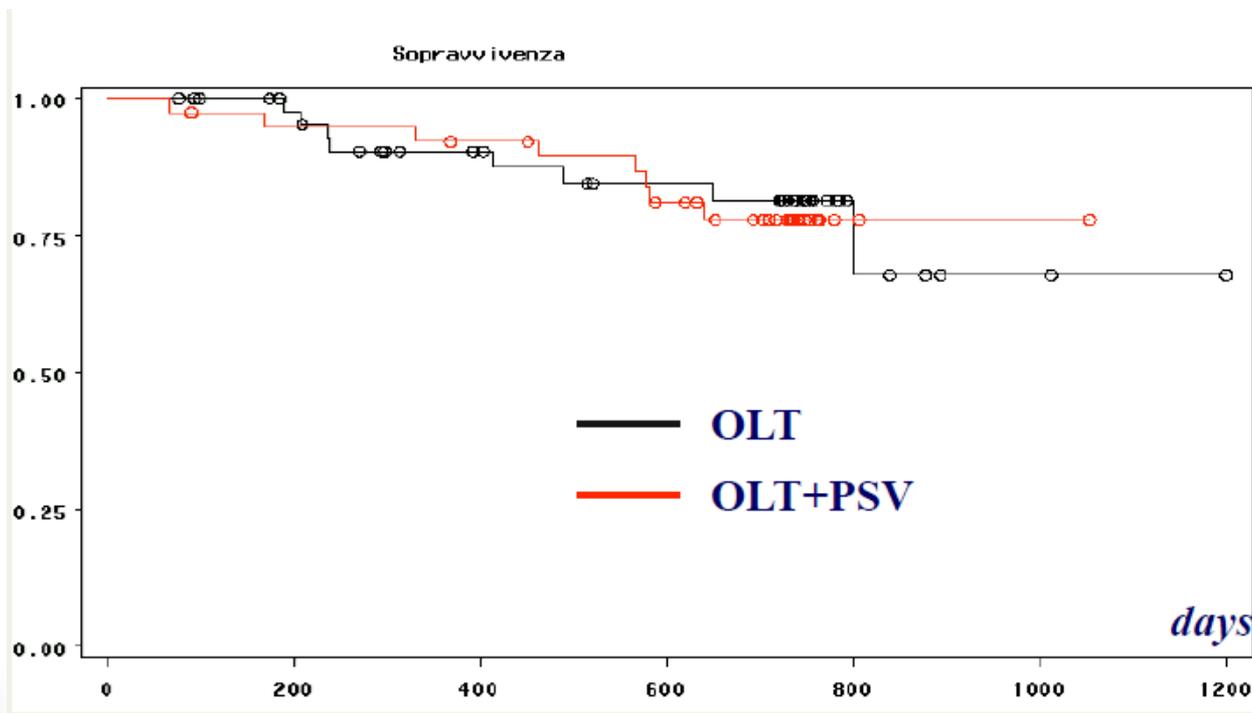
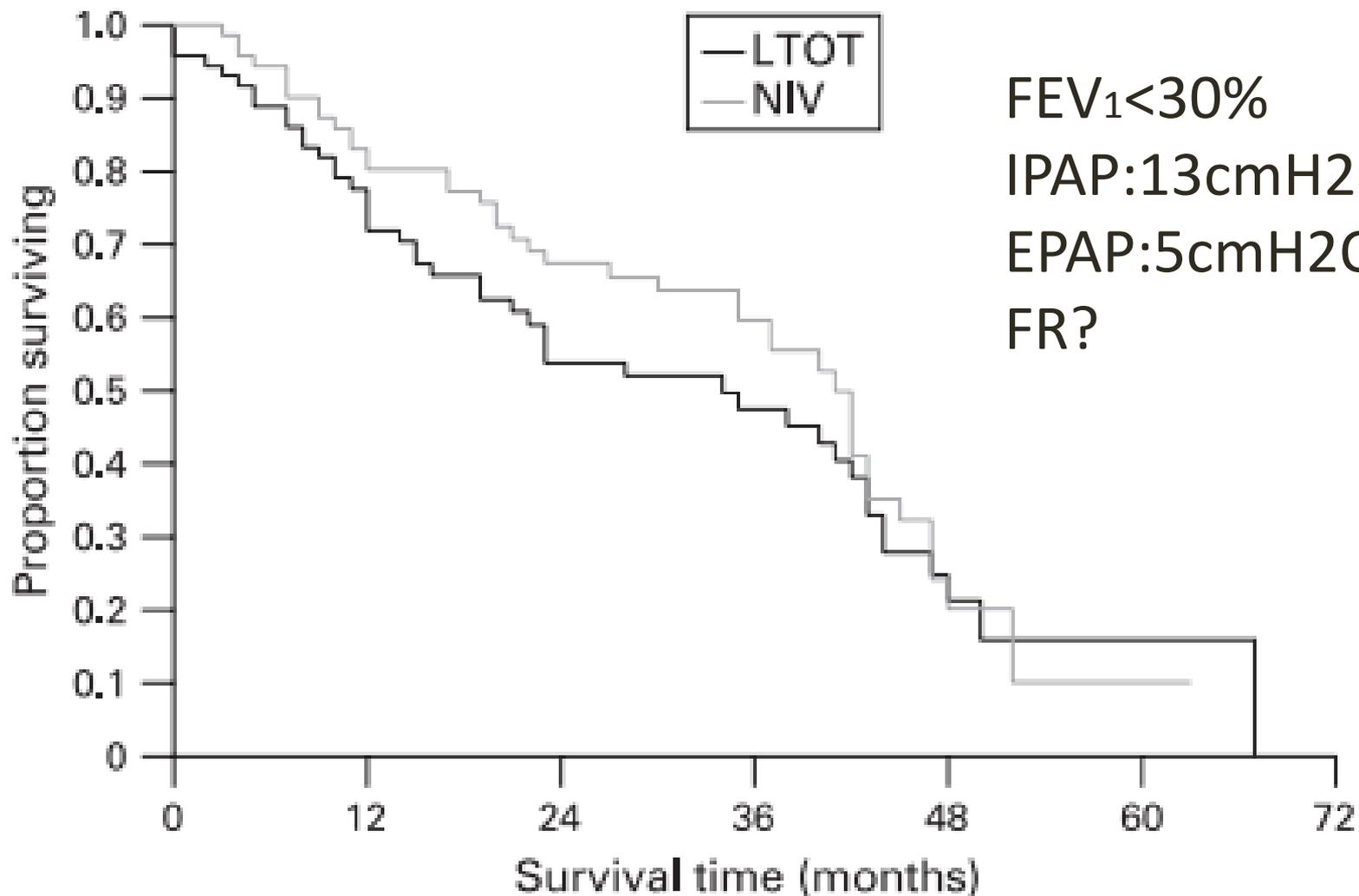


FIGURE 2. One-year survival was similar in patients with severe COPD after oxygen treatment alone and oxygen plus NPPV.

Mortality rate was similar in the two groups (18 and 17% in NPPV and LTOT, respectively). Deaths were due to acute respiratory failure (three and five patients in LTOT and NPPV groups, respectively), cardiac failure (one patient in both LTOT and NPPV groups) and other acute diseases (four and two patients in LTOT and NPPV, respectively).



FEV₁<30%
IPAP: 14cmH₂O
EPAP: 2cmH₂O
FR?



Nocturnal non-invasive nasal ventilation in stable hypercapnic COPD: a randomised controlled trial

Pas de diminution de la PaCO₂!

Table 3 Follow-up Paco₂ and FEV_{1.0} results

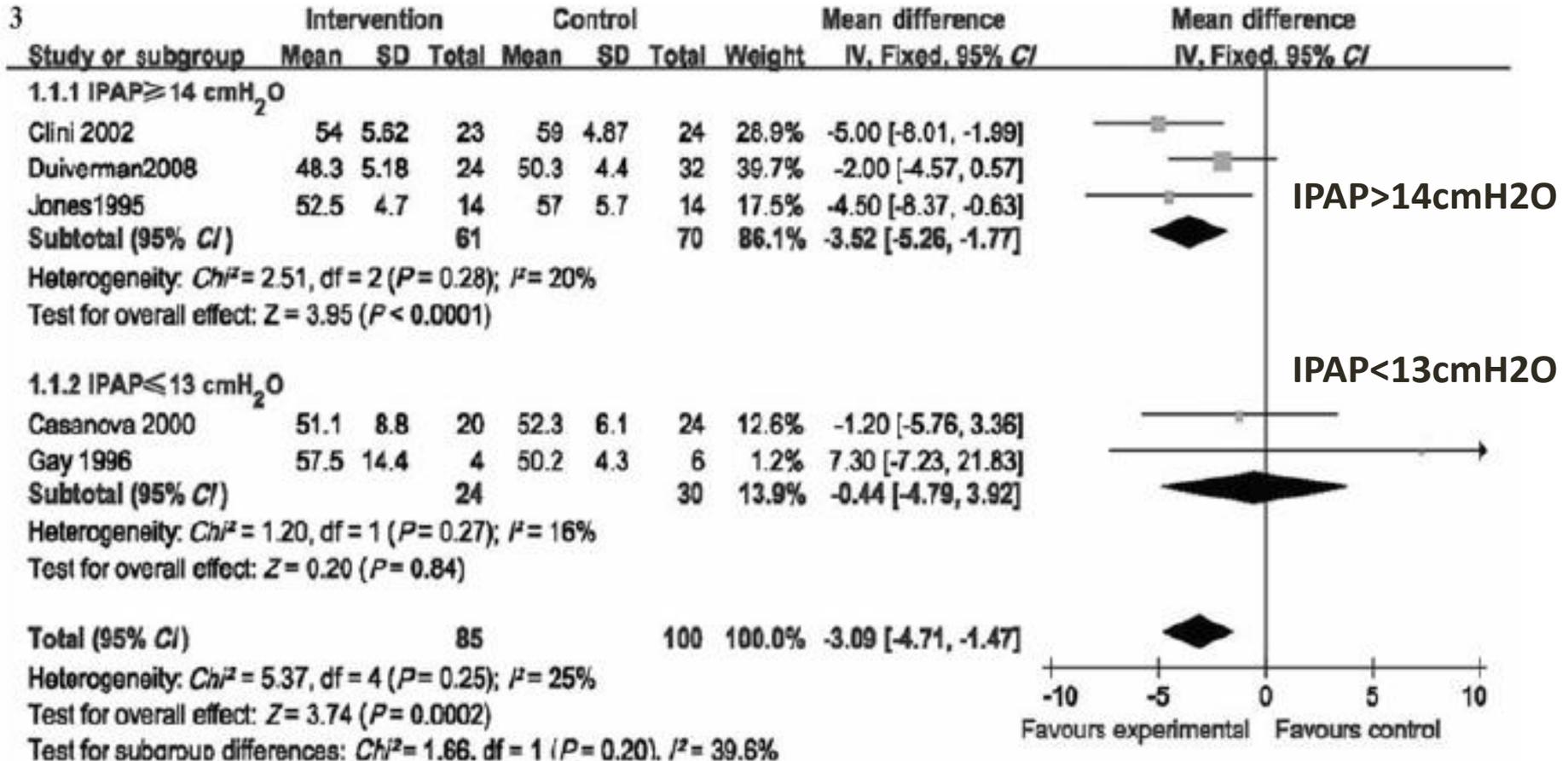
		Baseline	6 months	12 months
Paco ₂ (mm Hg)	LTOT	54.2 (52.0 to 56.4) n = 45	55.1 (52.1 to 58.1) n = 43	52.2 (49.5 to 54.9) n = 29
	NIV+LTOT	54.1 (51.7 to 56.5) n = 54	52.9 (50.5 to 55.3) n = 44	53.2 (50.6 to 55.8) n = 43
FEV _{1.0} (% pred)	LTOT	23.9 (21.9 to 25.9) n = 51	23.4 (21.4 to 25.4) n = 47	26.3 (24.1 to 28.5) n = 38
	NIV+LTOT	25.3 (22.3 to 28.3) n = 58	24.9 (21.7 to 28.1) n = 56	24.1 (21.1 to 27.1) n = 47

Diminution de la qualité de vie!

patients treated

with NIV had poorer general and mental health and reported less vigour and more confusion and bewilderment.

Quels réglages?





Millimètres de
mercure

Quels seraient les réglages à faire pour
un patient BPCO?

A: IPAP élevée malgré le risque
d'effort inefficace

B: Pep élevée pour l'obstruction
expiratoire

C: Pressions élevées malgré le
risques de fuites

D: La réponse D

IPAP élevée?

Table I. Ventilator settings for 69 patients receiving pressure-limited NPPV

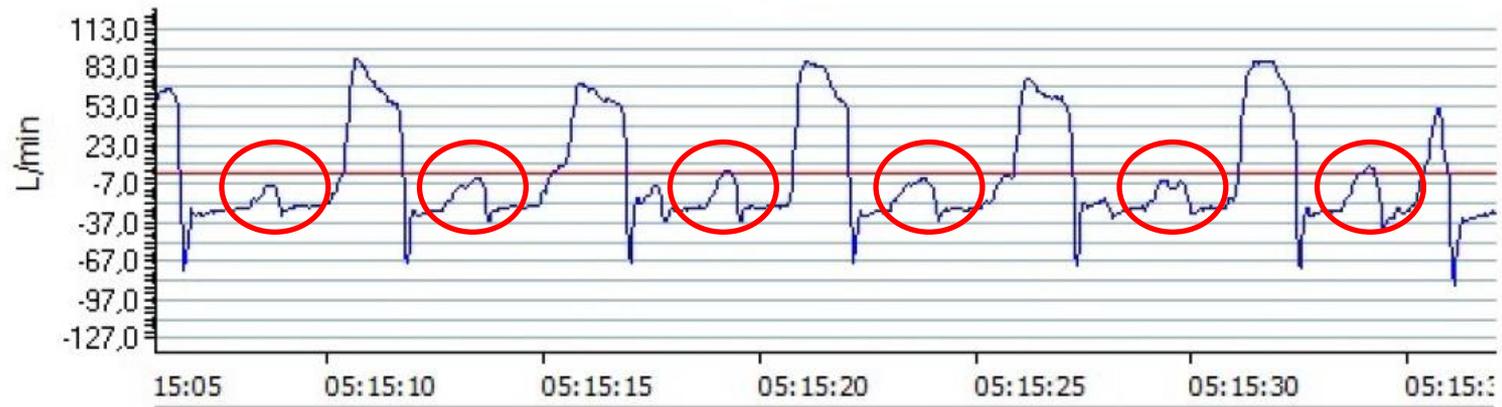
	Mean \pm SD	Min	Max
IPAP (cmH ₂ O)	28.0 \pm 5.4	17	42
EPAP (cmH ₂ O)	4.6 \pm 1.3	2	9
b _f (/min)	21.0 \pm 2.8	10	26
Supplemental oxygen (l/min)	1.6 \pm 1.5	0	6

IPAP = inspiratory positive airway pressure, EPAP = expiratory airway pressure, b_f = breathing frequency; SD = standard deviation.

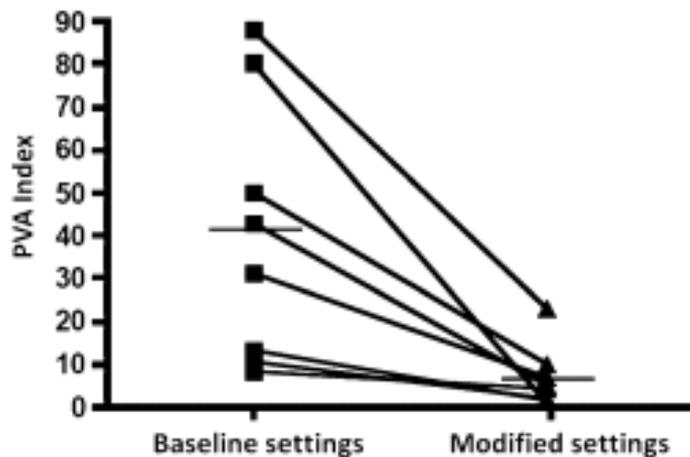
↘ de la PaCO₂ à 2 mois

IPAP élevée? Oui mais...

Débit



lundi, 2 avril 2012



P= 0.006

a) Morning Dyspnea



High-intensity versus low-intensity non-invasive ventilation in patients with stable hypercapnic COPD: a randomised crossover trial

Michael Dreher,¹ Jan H Storre,¹ Claudia Schmoor,² Wolfram Windisch¹

NPPV

	Period	LI-NPPV	HI-NPPV
IPAP (mbar)		14.6 ± 0.8	28.6 ± 1.9
EPAP (mbar)		4.0 ± 0	4.5 ± 0.7
Bf (/min)		8.0 ± 0	17.5 ± 2.1

2 patients ont refusé de passer dans le groupe low-intensity!

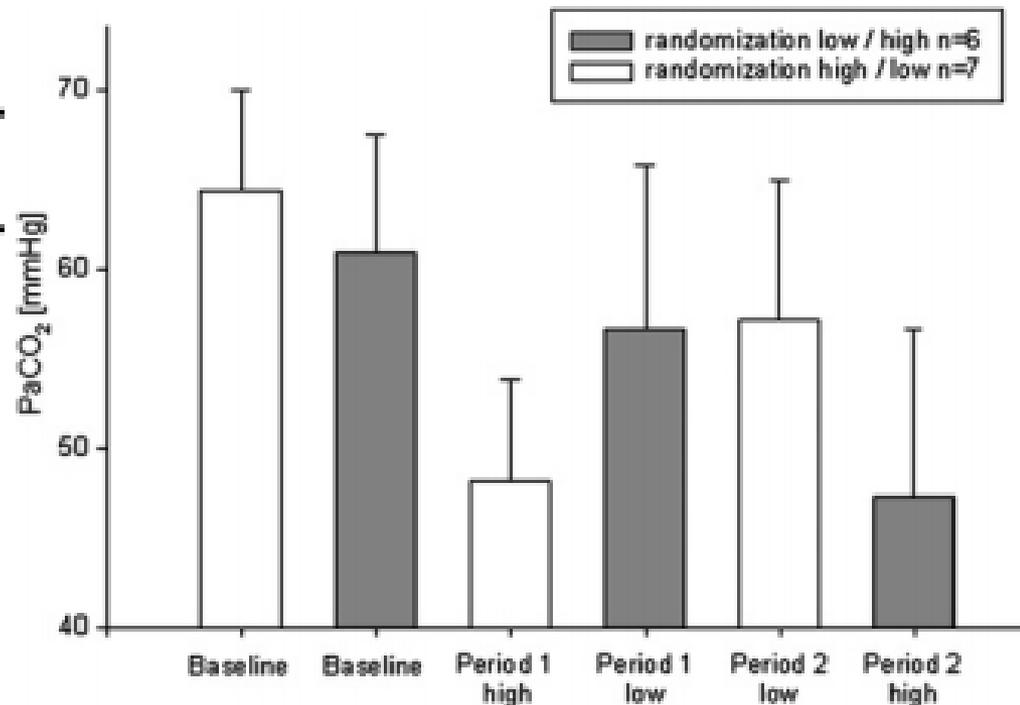
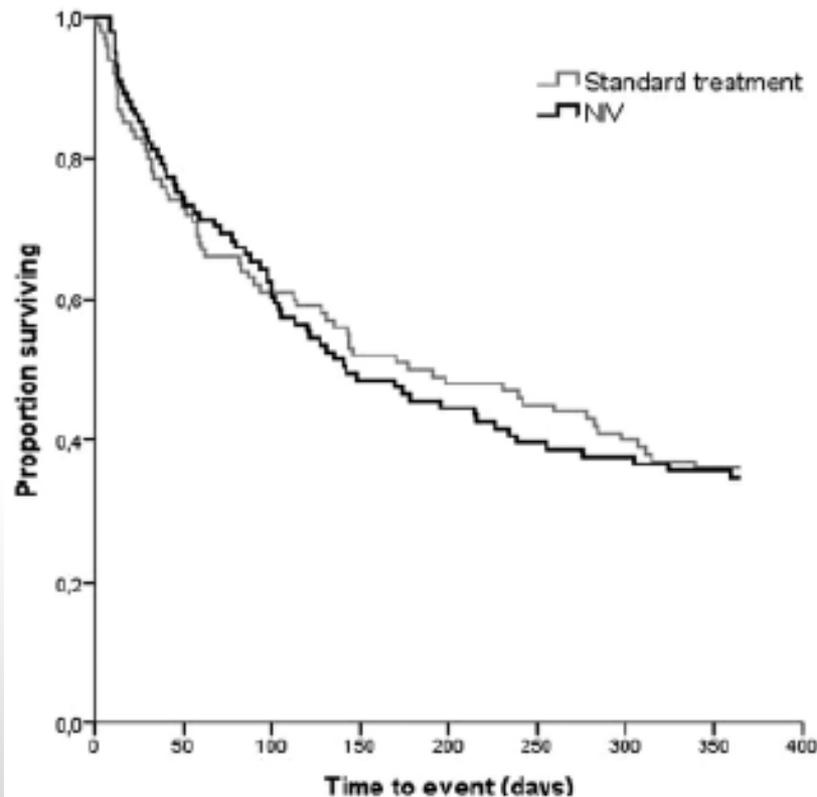


Figure 2 Nocturnal mean ± SD arterial carbon dioxide tension (Paco₂) at baseline and at follow-up visits.

Nocturnal non-invasive ventilation in COPD patients with prolonged hypercapnia after ventilatory support for acute respiratory failure: a randomised, controlled, parallel-group study

F M Struik,^{1,2} R T M Sprooten,³ H A M Kerstjens,^{1,2} G Bladder,¹ M Zijnen,⁴ J Asin,⁵
N A M Cobben,³ J M Vonk,^{2,6} P J Wijkstra^{1,2}



de la PaCO₂; p=0,03

Tendance à
l'amélioration de HRQL;
p=0,054

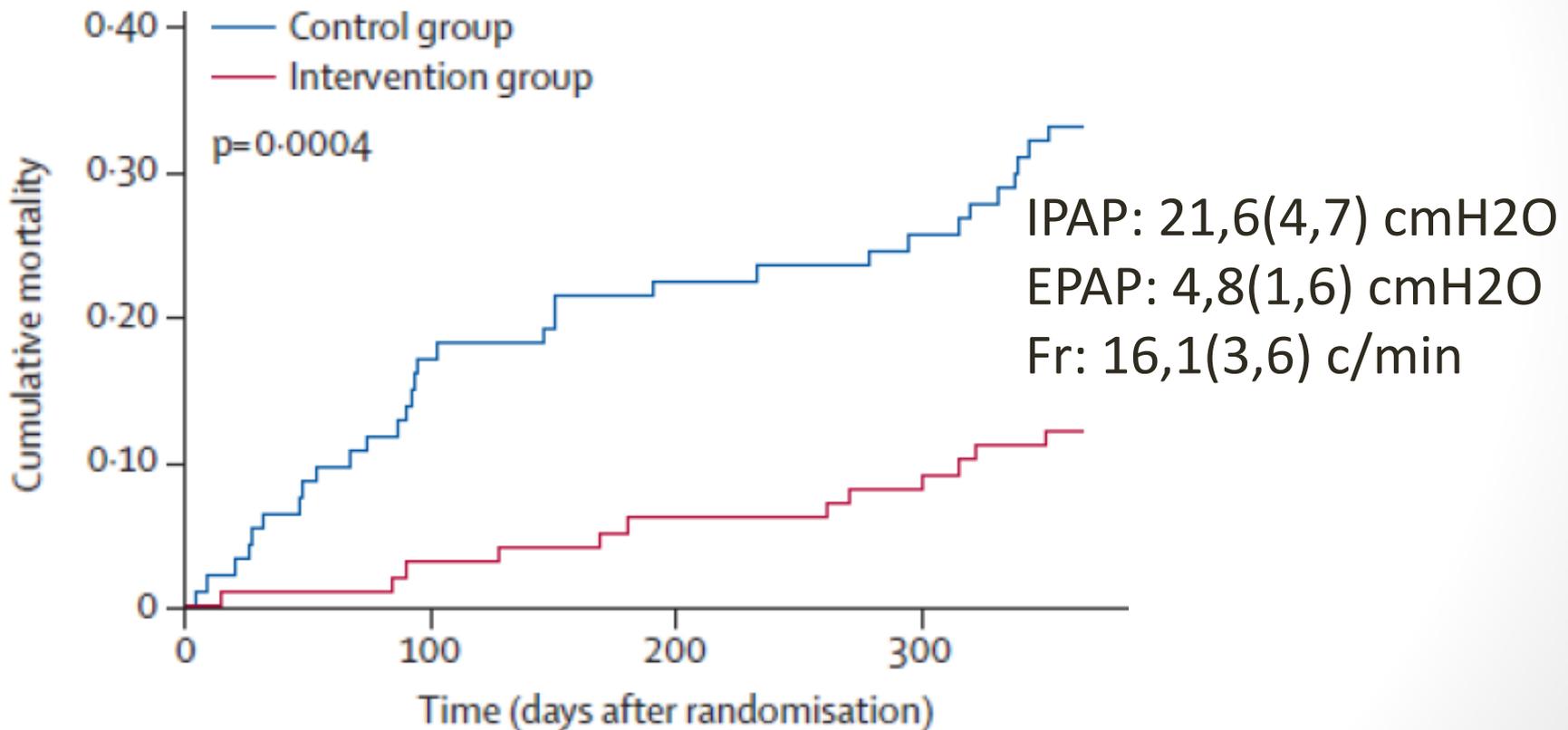
IPAP: 19,2(3,4)cmH₂O

EPAP: 4,8(1)cmH₂O

Fr: 15(3)c/min

Non-invasive positive pressure ventilation for the treatment of severe stable chronic obstructive pulmonary disease: a prospective, multicentre, randomised, controlled clinical trial

Thomas Köhnlein, Wolfram Windisch, Dieter Köhler, Anna Drabik, Jens Geiseler, Sylvia Hartl, Ortnud Karg, Gerhard Laier-Groeneveld, Stefano Nava, Bernd Schönhofer, Bernd Schucher, Karl Wegscheider, Carl P Criée, Tobias Welte

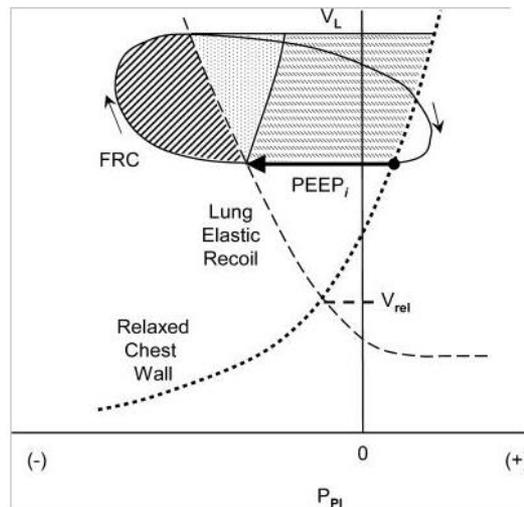


IPAP élevée? OUI! et l'EPAP...?

Patients	ZEEP	
	RF	INT
ΔP , cmH ₂ O	8.8 ± 2.3	7.22 ± 2.0
R_{int} , cmH ₂ O/L/s	9.1 ± 2.1	10.9 ± 3.7
E_{st} , cmH ₂ O/L	22 ± 8	22 ± 6.2
PEEP _{i,stat} , cmH ₂ O	9.2 ± 2.4	7.1 ± 1.7
PEEP _{i,dyn} , cmH ₂ O	2.5 ± 0.7	2.4 ± 0.9
PEEP _{i,dyn} /PEEP _{i,stat}	0.27 ± 0.09	0.34 ± 0.07 ^s
$\Delta EELV$ (L)	0.45 ± 0.09	0.35 ± 0.10
f_H beats/min	89 ± 25	91 ± 22
MAP, mmHg	80 ± 16	85 ± 17

Fig. 3.

Antonaglia, Respir Physiol Neurobiol, 2012



PEEPi at rest = 4 cmH2O

Loring, J Appl Physiol, 2009

Initier une VNI en résumé

SOH:

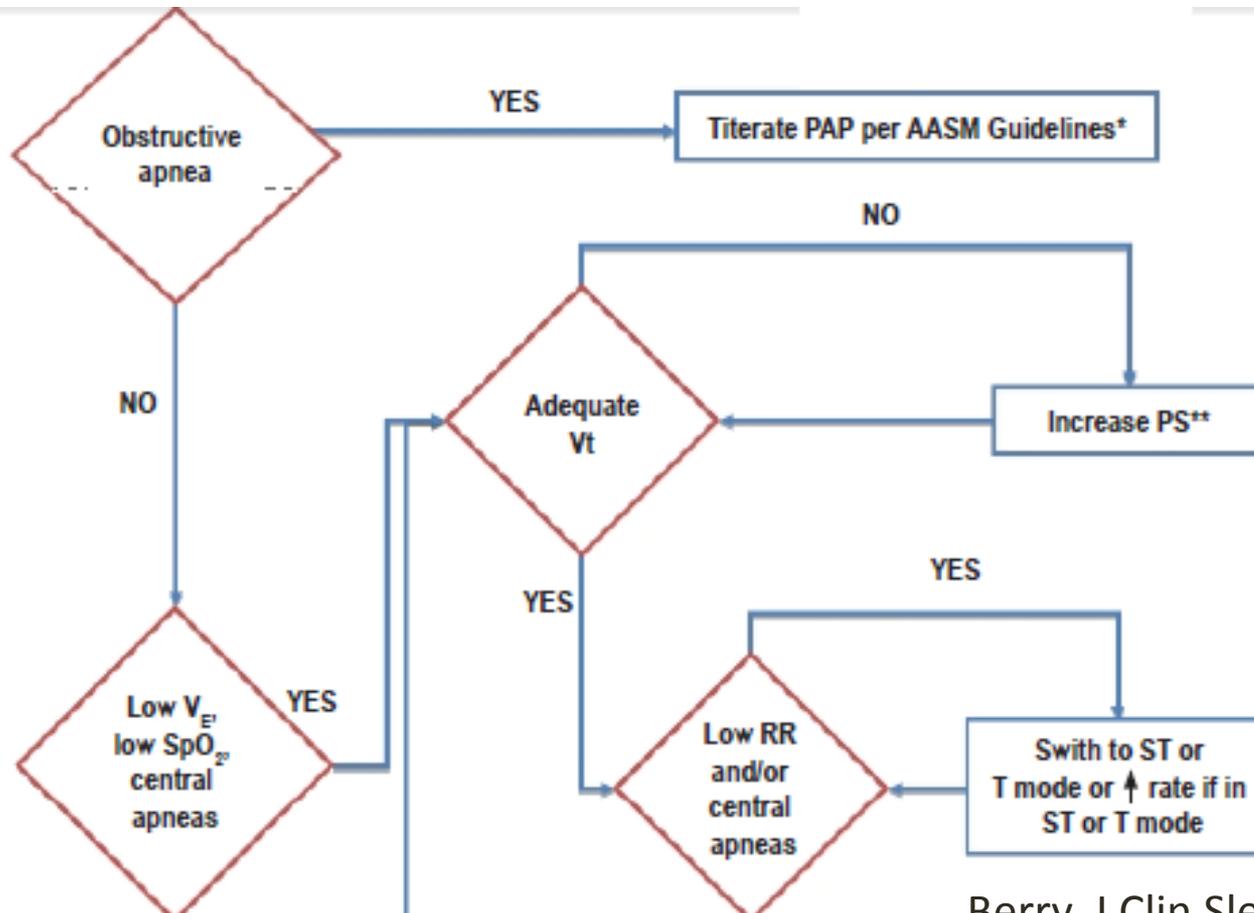
IPAP: 15-16 cmH₂O
EPAP: 7-8cmH₂O

Réglages minimum:

IPAP: 8cmH₂O
EPAP: 4cmH₂O

BPCO:

↑ IPAP selon la tolérance



Les interfaces à domicile



OU



?

Le choix des patients?

- **n= 29 états stables naïfs de VNI**
 - 20 OHS, 4 pariéto-restrictifs, 3 neuro- μ , 2 BPCO
- **2 x 2h de VNI diurne (2 j distincts)**
 - MN
 - MNB
- **Mode ST**
- **Prescription: préférence patient**
 - sauf 3 sujets: NB imposé car meilleure correction PCO₂)
- **Evaluation à 3 mois**
 - GDS/SpO₂/observance

Table 1. Results of the Adaptation Process to Nasal vs Oronasal Mask

	Nasal Mask (n = 29)	Oronasal Mask (n = 29)	P
P _a CO ₂ after NIV (2 h day session), mm Hg	47.3 ± 6.3	50.2 ± 5.1	.87
pH after NIV (2 h day session)	7.40 ± 0.30	7.40 ± 0.28	.81
HCO ₃ ⁻ after NIV (2 h day session), mmol/L	29.3 ± 3.2	32.2 ± 1.8	.23
T90 after NIV (night session), %	39.4 ± 20.3	36.6 ± 29.5	.67
IPAP, cm H ₂ O	13.7 ± 2.7	15.4 ± 2.4	.02
EPAP, cm H ₂ O	6.3 ± 1.1	6.2 ± 1.2	.89
Respiratory rate, breaths/min	15.5 ± 3.1	14.5 ± 2.3	.71
Comfort score, analog scale	6.9 ± 2.3	5.3 ± 2.3	.01

38% préférence MNB

62% préférence MN

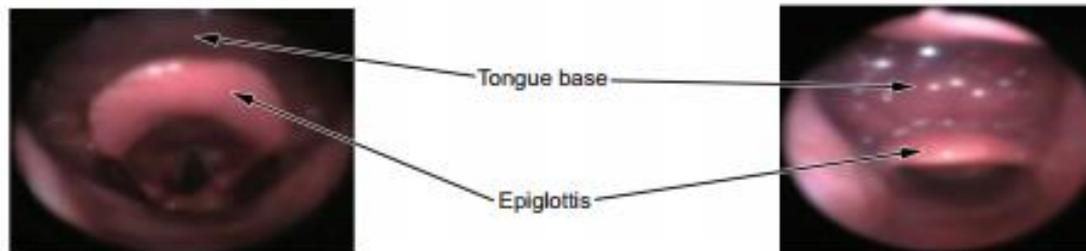
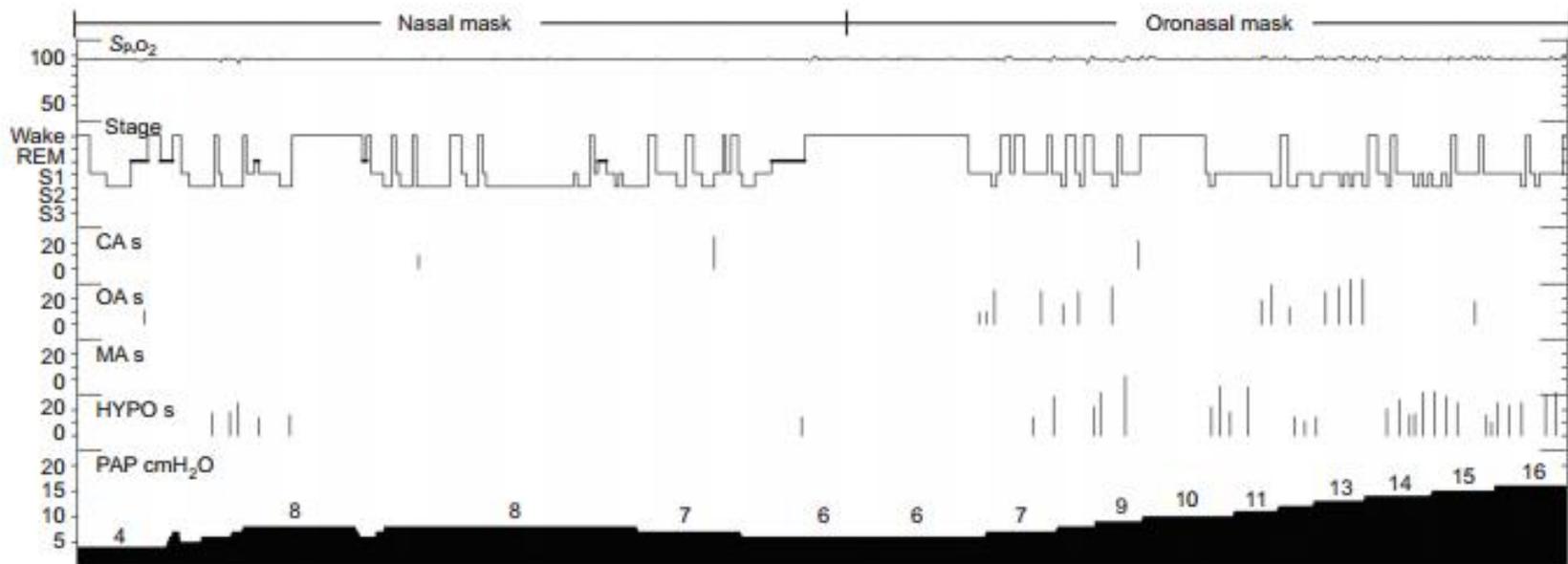


FIGURE 1. Polysomnography summary of a natural sleep continuous positive airway pressure (CPAP) titration study (top) and sleep endoscopy images (bottom) using both nasal and oronasal masks. During the first part of the natural sleep CPAP titration, a nasal CPAP of 7 cmH_2O adequately controlled obstructive events. During the second part, an oronasal mask was used. Observe the persistence of obstructive respiratory events even at pressures up to 16 cmH_2O . The endoscopic image taken with nasal CPAP of 7 cmH_2O shows a wide open oropharynx. In contrast, the image taken with oronasal CPAP of 16 cmH_2O shows the tongue base posteriorly displaced, pushing the epiglottis and significantly narrowing the airway. SpO_2 : arterial oxygen saturation measured by pulse oximetry; CA: central apnoea; OA: obstructive apnoea; MA: mixed apnoea; HYPO: hypopnoea; PAP: pulmonary artery pressure; REM: rapid eye movement. Respiratory event time is shown in seconds.

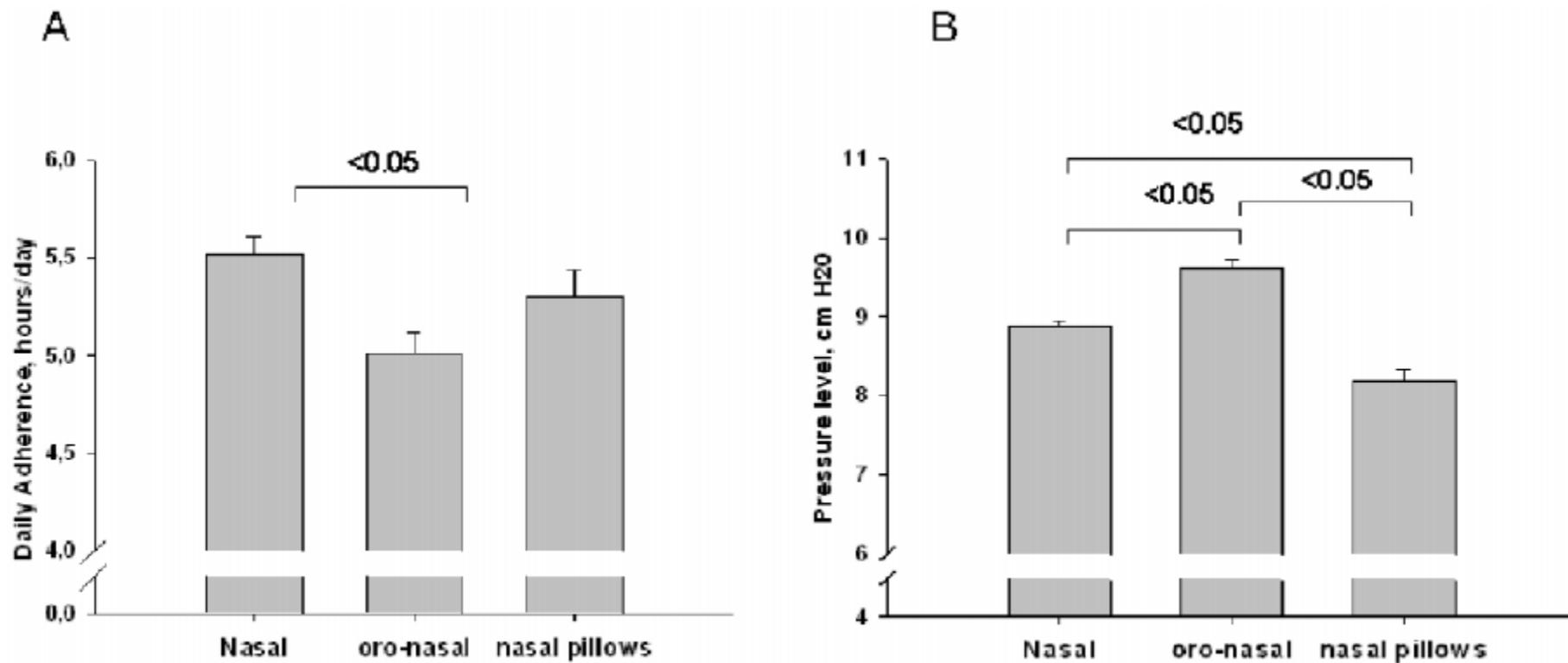


Figure 2. Adherence (a) and positive airway pressure level (b) according to the type of interface.
 doi:10.1371/journal.pone.0064382.g002

Attention si changement nasal pour oronasal!

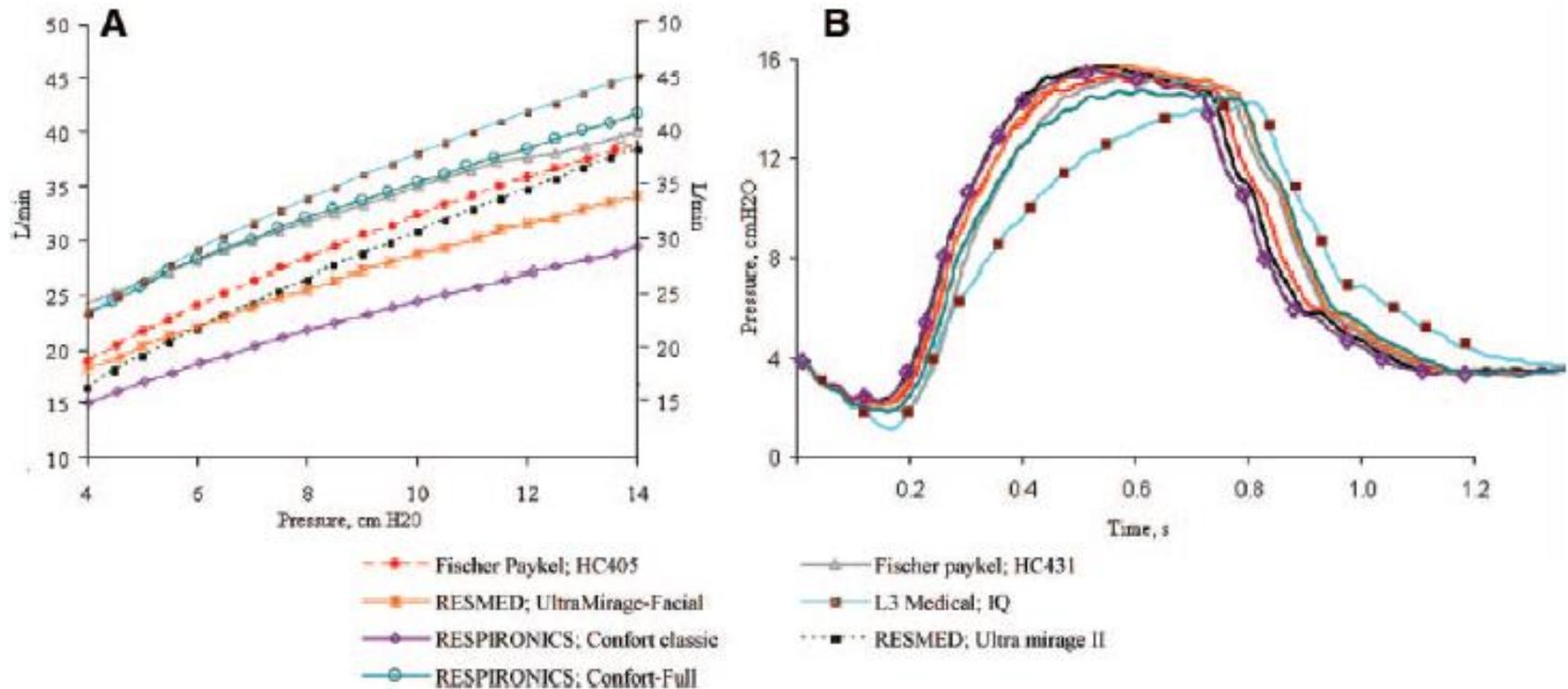
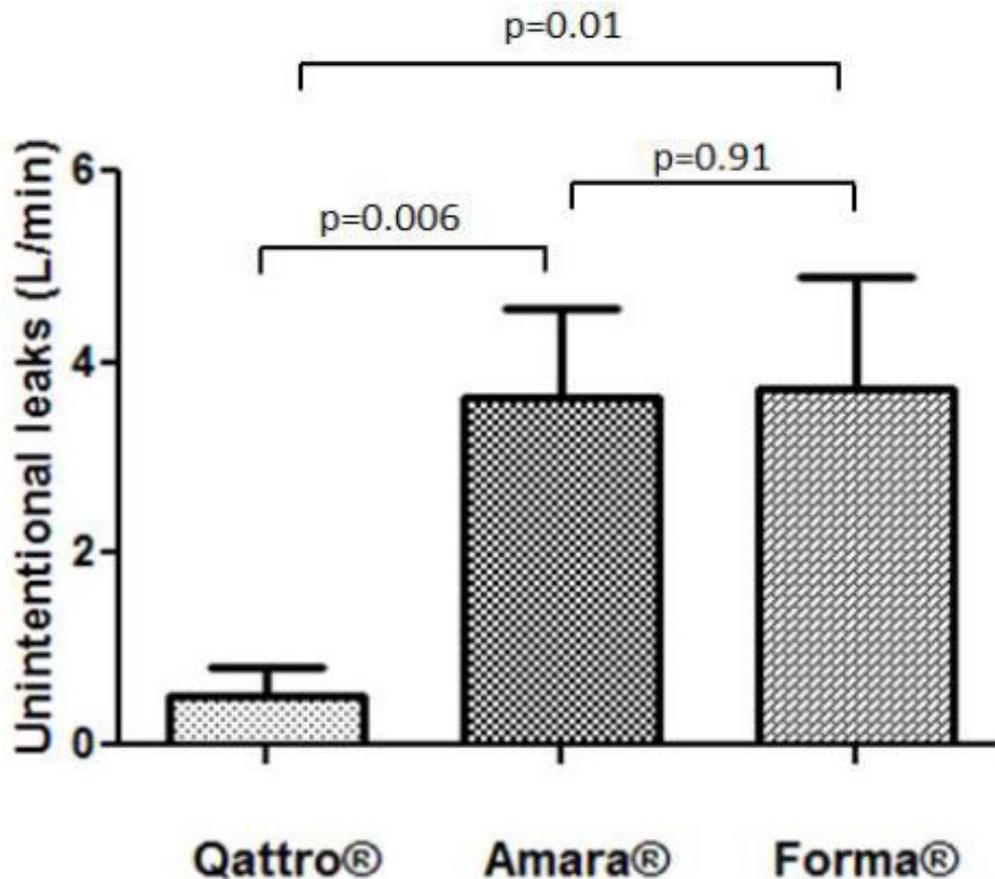


FIGURE 1. *Left, A:* Intentional leaks expressed as function of the pressure in seven different masks. *Right, B:* Pressure-time plot of VPAP III ST with the seven masks for the normal lung condition.

Même pour des masques de dernière génération!



Pas de différence sur la PtCO₂ avec une EPAP à 4cmH₂O!

Pas de lien entre les fuites non intentionnelles et le confort du masque!

Conclusion

- L'installation de la ventilation non invasive au domicile est l'objectif de demain et le kinésithérapeute sera confronté aux premiers appareillages des patients.
- SOH, Neuro-musculaires, BPCO en état stables sont les principales indications de la VNI à domicile.
- Privilégier une interface nasale pour débiter.
- Les études nous guident sur les réglages optimaux.
- En pratique quotidienne, les réglages sont à adapter selon les patients et leur tolérance.

Merci de votre
attention

