Indications de la VNI

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Polyclinique Saint Laurent
Rennes

Plan

1) Physiopathologie : pourquoi va t-on avoir besoin de ventilation mécanique ?

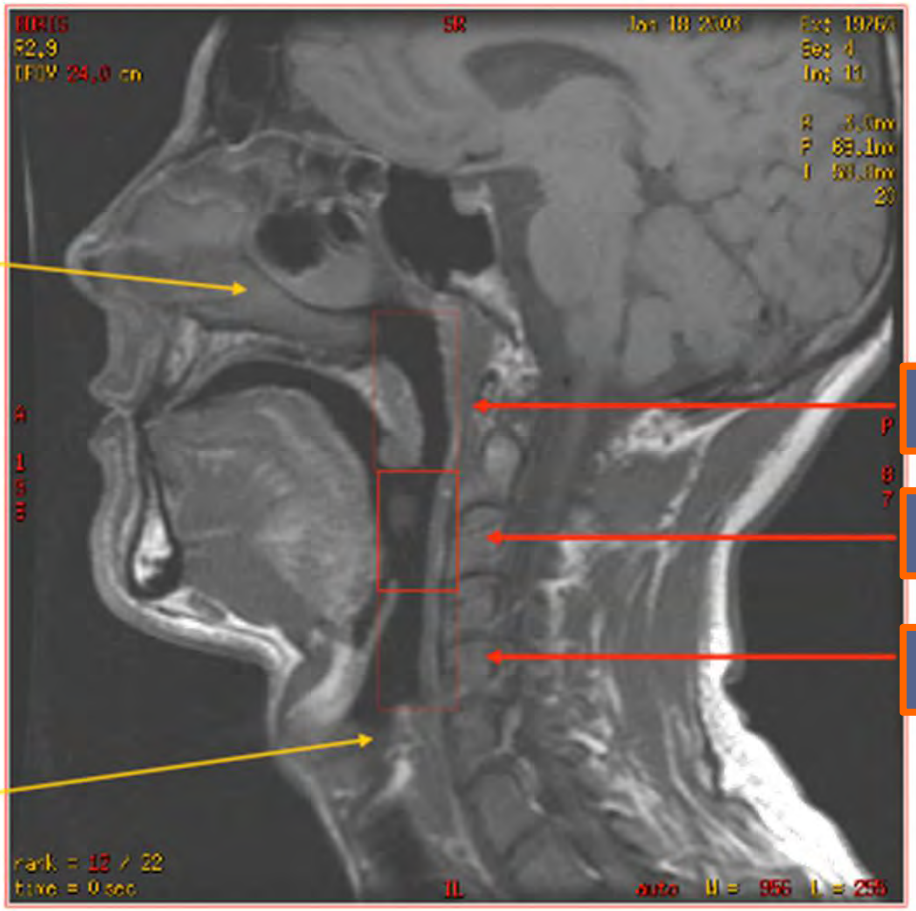
Principes de base

- $Pmus + Pméca = Ers \times \dot{V} + Rrs \times V$

  Elastance — Resistance

- Ventilation minute = (freq x Vt)-(freq X Espace mort)

Augmentation des résistances des VAS



Film augmentation des résistances

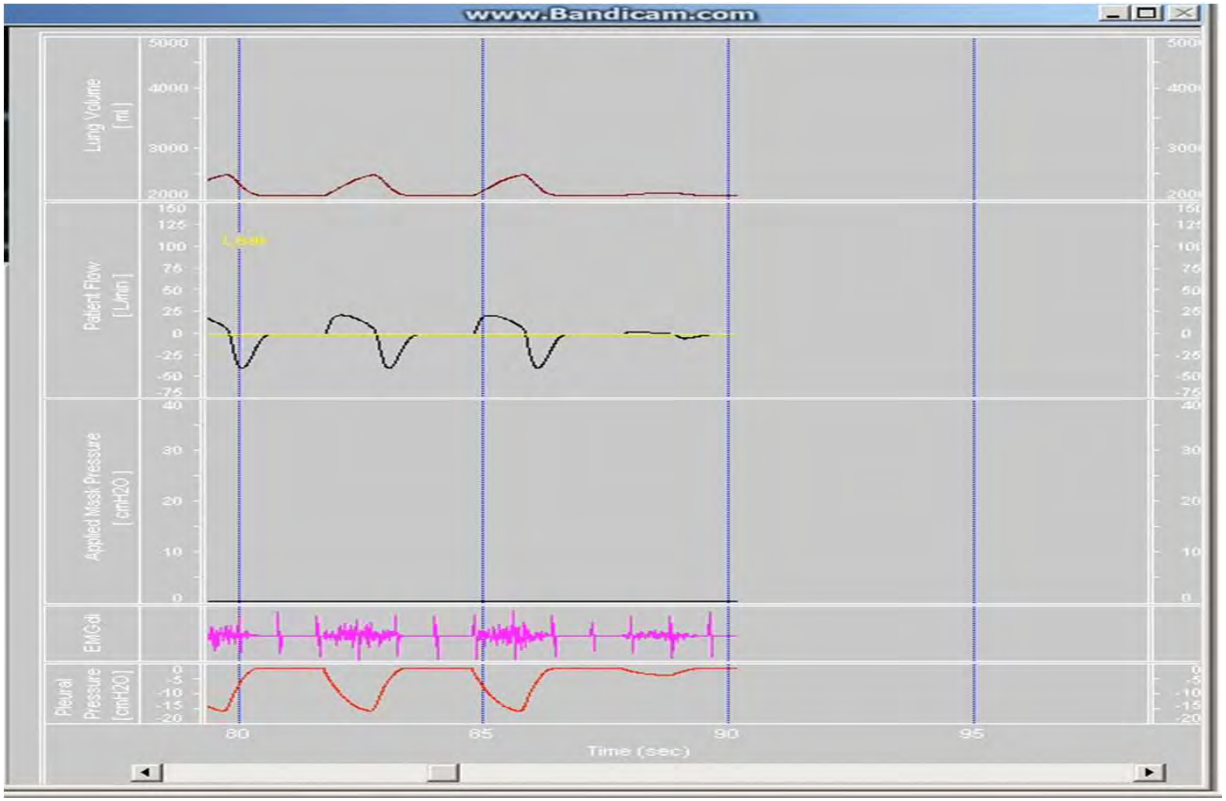
Volume pulmonaire

Flux patient

Pression masque

EMG diaphragmatique

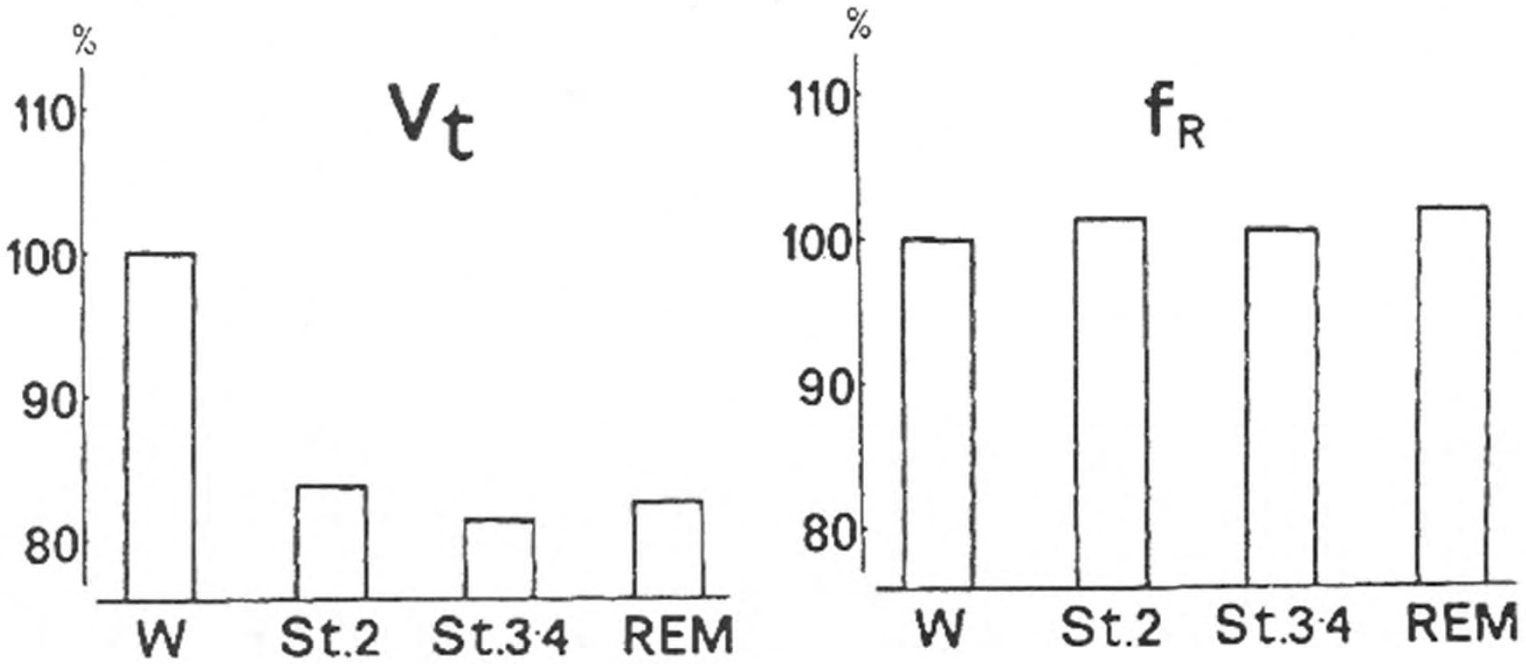
Pression pleurale



Ventilation au cours du sommeil

1. Diminution de la sensibilité des chémorécepteurs :
   modification de la réponse à l'hypoxémie et à l'hypercapnie.
2. Modification du comportement des muscles respiratoires.

Ventilation au cours du sommeil



Kryger,Roth and Dement,
Principles and practice of sleep Medecine

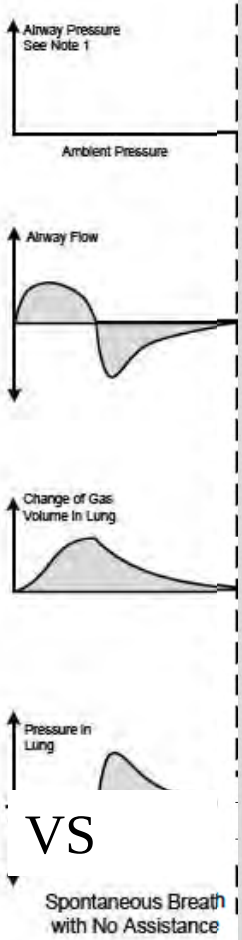
Physiologie de la mécanique ventilatoire

Pression bouche

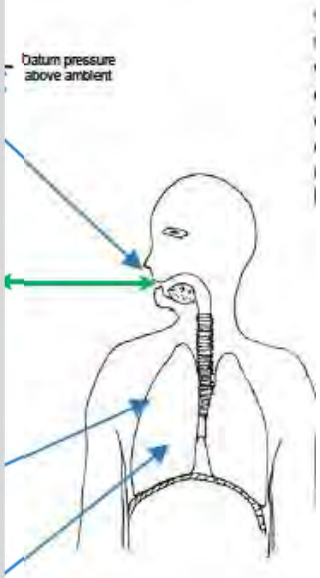
Débit

Volume

Pression Pulmonaire



RESPIRATION EN VENTILATION SPONTANEE NORMALE



Film : sommeil BPCO

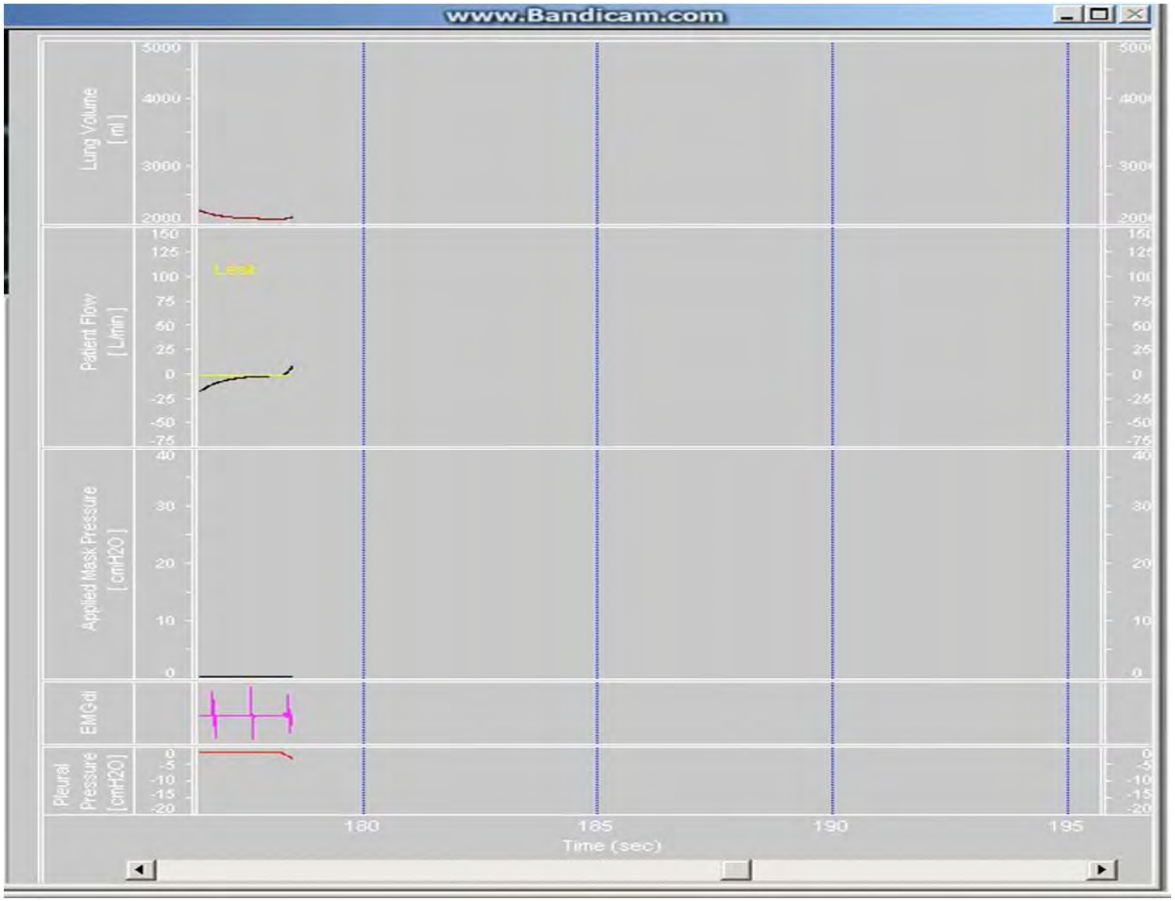
Volume pulmonaire

Flux patient

Pression masque

EMG diaphragmatique

Pression pleurale



Film : sommeil restrictif

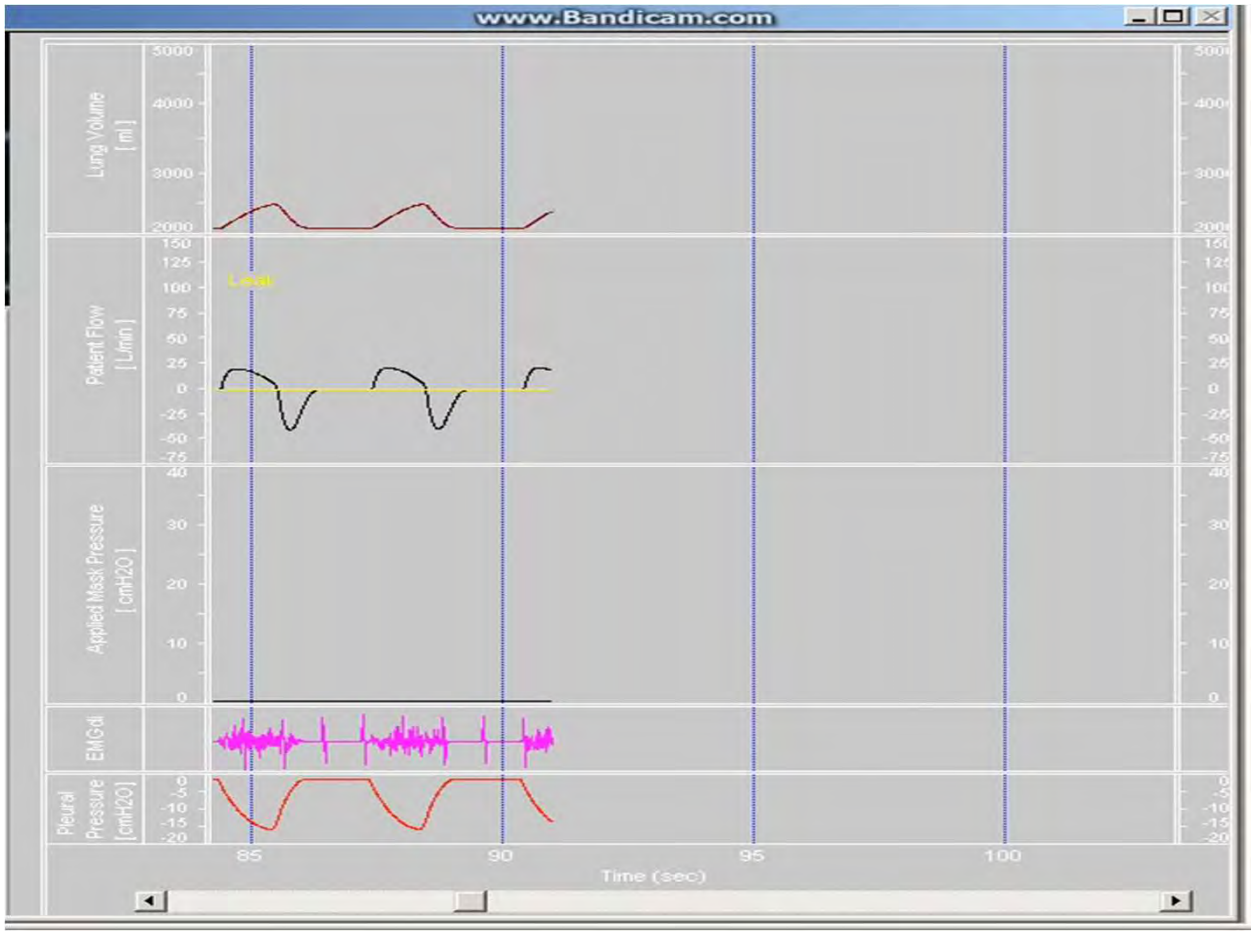
Volume pulmonaire

Flux patient

Pression masque

EMG diaphragmatique

Pression pleurale



Plan

1) Physiopathologie : pourquoi va t-on avoir besoin de ventilation mécanique ?

2) Indication de la VNI : l'hypoventilation

Indications de la VNI

- La VNI est indiquée en cas

  d'hypoventilation alvéolaire

- 3 catégories :
  - Ceux qui ne peuvent pas
  - Ceux qui ne veulent pas
  - Ceux qui ne veulent et ne peuvent pas

Film : sommeil puis VNI

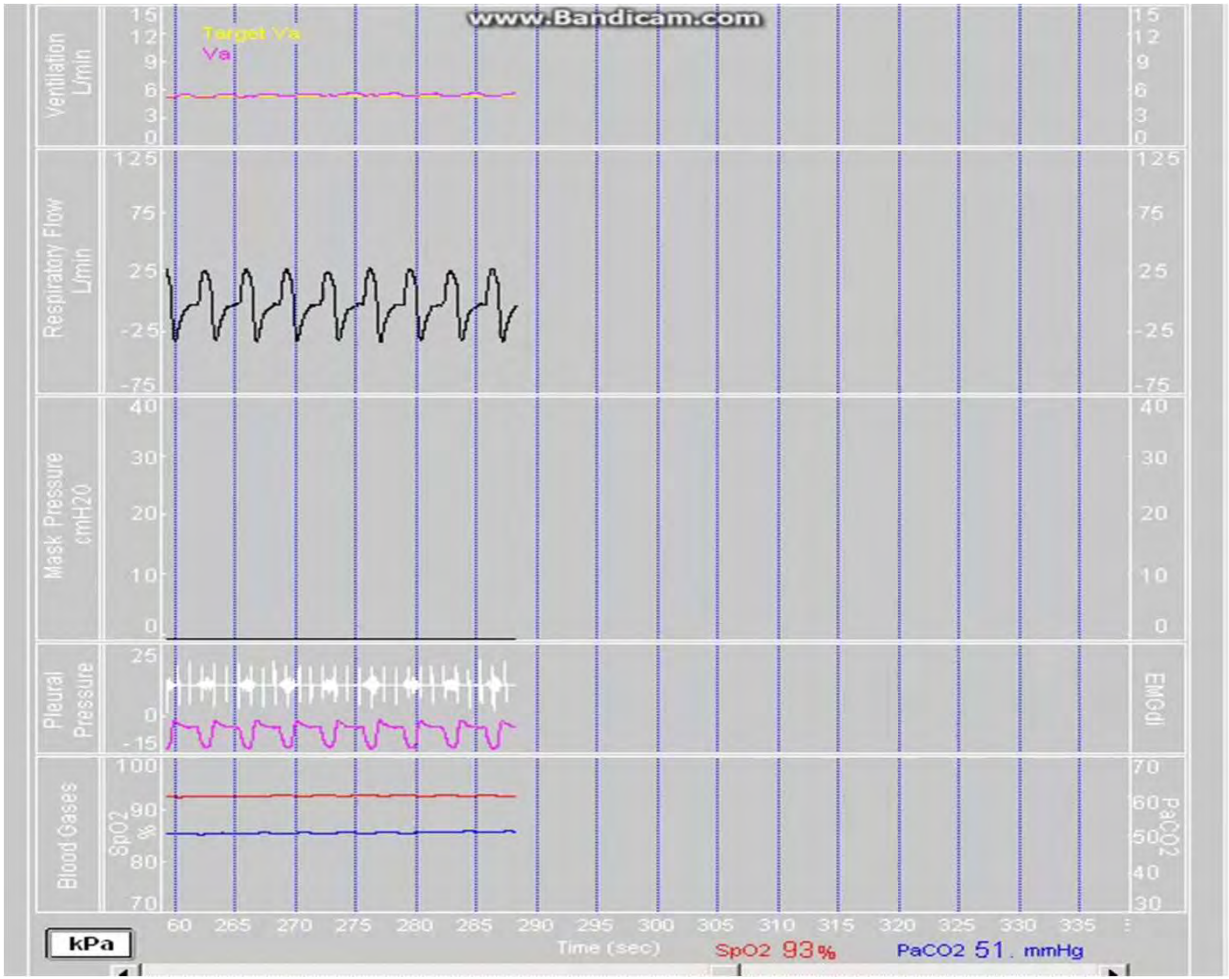
Ventilation minute

Flux patient

Pression masque

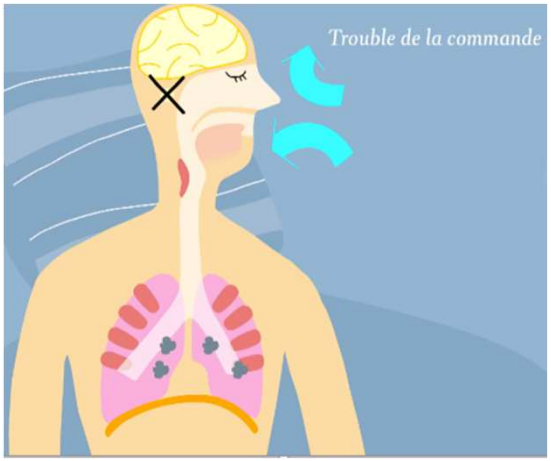
Pression pleurale

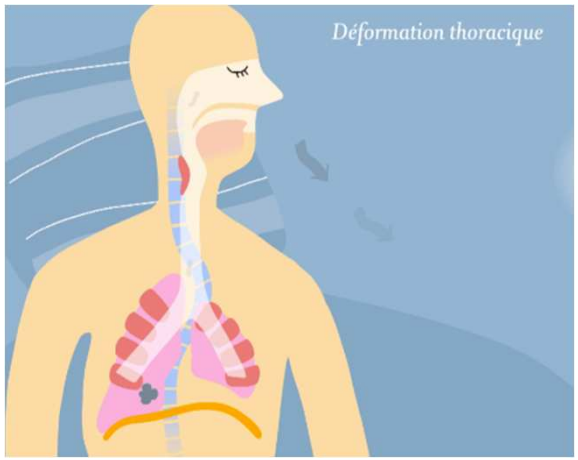
SaO_2
$PaCO_2$



Indications de la VNI

- **« ne peuvent pas.. »**
  - Pathologie neuro-musculaire (SLA- traumatismes médullaires)
  - Paralysie diaphragmatique
  - Myopathies, Myasthénie
  - Cyphoscoliose, séquelles de tuberculose…



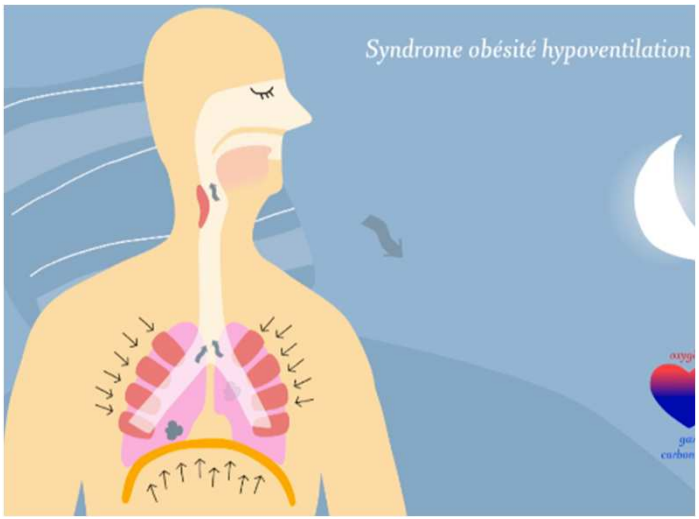


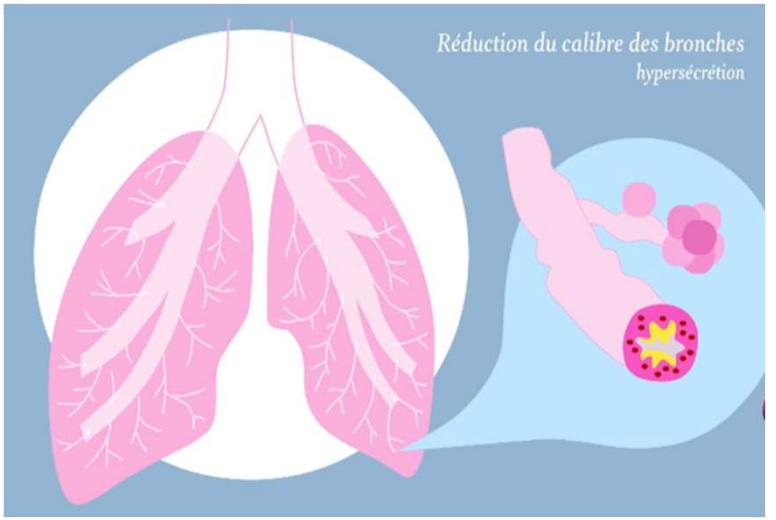
Indications de la VNI

- **« ne veulent pas.. »**
  - Hypoventilation alvéolaire primaire – syndrome d'Ondine
  - AVC, poliomyélite bulbaire
  - Malformation d'Arnold – Chiari

Indications de la VNI

- **« ne peuvent et ne veulent pas… »**
  - BPCO
  - Obésité hypo-ventilation
  - ….





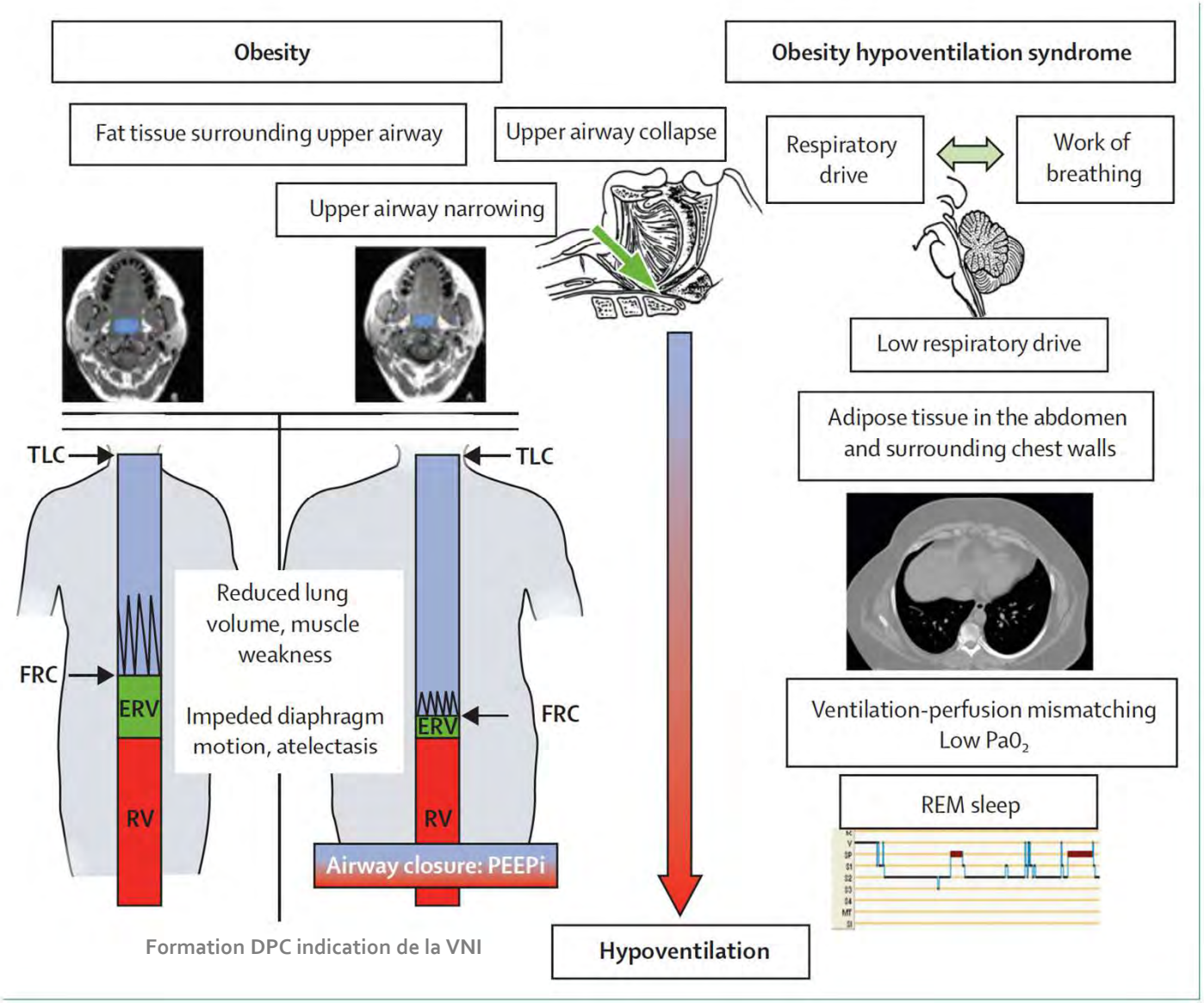
Le syndrome obésité hypoventilation alvéolaire

- Hypoventilation alvéolaire chronique $PaCO_2$ >45 mmHg
- Obésité IMC>30 Kg/m2
- Absence d'affection respiratoire associée
- Indépendamment de l'association ou non à un SAOS (90%).
- +/- ou $[HCO_3^-]$ > 27 mmol/L ou excès de base + 3 mmol/L

Hart N, Mandal S, Manuel A, Mokhlesi B, Pépin JL, Piper A, Stradling J. Thorax 2013/08/28.

SOH physiopathologie

Lancet Respir Med 2016; 4: 407–18



Formation DPC indication de la VNI

Obésité et retentissement respiratoire : Mécanismes

RÔLE DU SAS : Obstructions de la voie aérienne supérieure

ALTÉRATION VENTILATION PERFUSION

Dégradation des rapports ventilation/perfusion aux bases

Augmentation des résistances des petites bronches

HYPOVENTILATION ALVÉOLAIRE

Diminution de la compliance de la cage thoracique

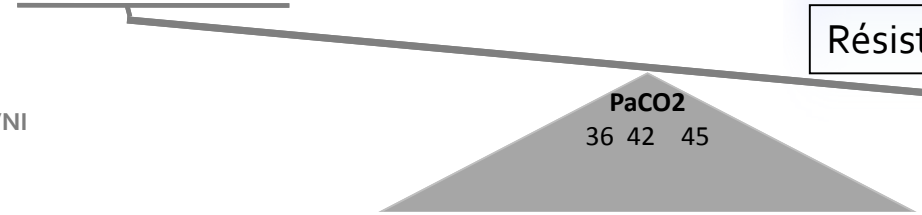
Diminution de l'endurance du diaphragme

Diminution de la réponse à l'hypercapnie et/ou à l'hypoxémie

Résistance à la leptine

Augmentation de la ventilation minute

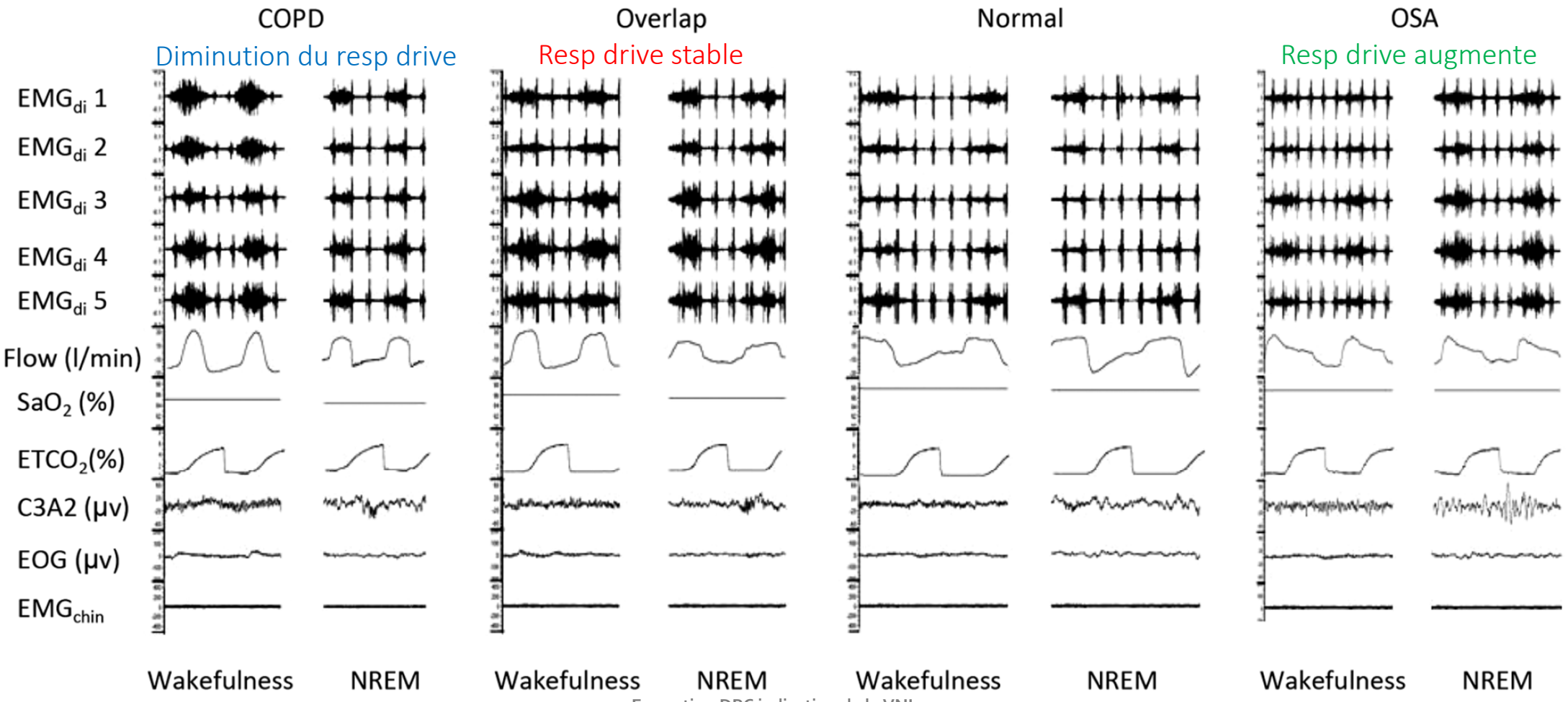
Augmentation de la force du diaphragme



BPCO et sommeil

- Mécanismes de la désaturation nocturne dans la BPCO
  - ✓ Hypoventilation
    - Diminution de la ventilation par diminution du Vt
  - ✓ Majoration des inégalités ventilation/perfusion
  - ✓ La sévérité de la désaturation dépend du degré d'hypoxémie diurne
- Diminution de la commande centrale et diminution des réponses ventilatoires à l 'hypoxie et à l'hypercapnie
- Abolition au cours du sommeil paradoxal de l'activité des muscles intercostaux
- Élévation de la résistance des voies aériennes supérieures
  - ➔ Causes :
    - Accumulation des sécrétions bronchiques pendant le sommeil
    - Fermeture des voies aériennes des territoires inférieurs (diminution de la CRF au cours du sommeil)

Overlap : cas particulier



He B-T, et al. Thorax 2017;72:256–262. doi:10.1136/thoraxjnl-2016-208467

Indications VNI

Changing Patterns in Long-term Noninvasive Ventilation*A 7-Year Prospective Study in the Geneva Lake Area(CHEST 2003; 123:67–79)

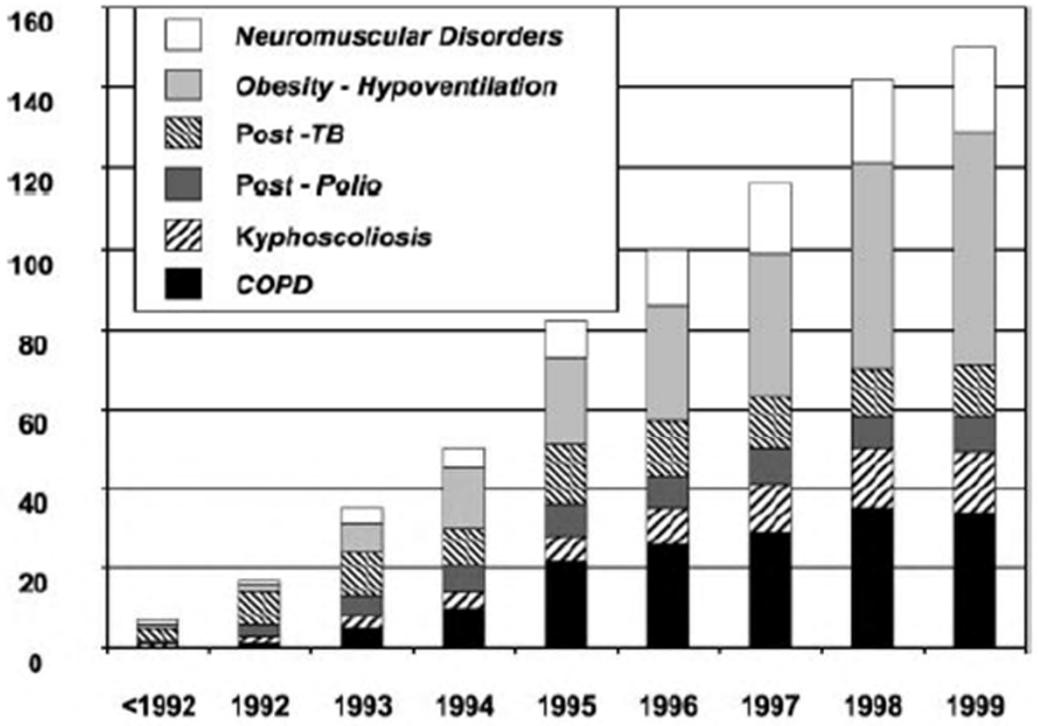


FIGURE 2. Yearly count of the cumulative population of patients treated by NPPV during the study period (1992 to 2000), by diagnostic category.

Indications de la VNI

- Hypercapnie ($PaCO_2$) supérieure à 45 mmHg
- Mesure de la pression transcutanée en dioxyde de carbone ($PtcCO_2$) nocturne moyenne supérieure à 50 mmHg
- Pour la BPCO : $PaCO_2$ >55 mmHg et décompensations fréquentes ou a la suite d'une décompensation aigue.

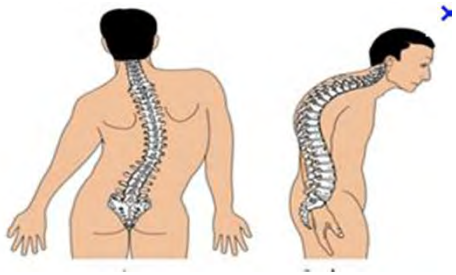
Plan

1) **Physiopathologie** : pourquoi va t-on avoir besoin de ventilation mécanique ?

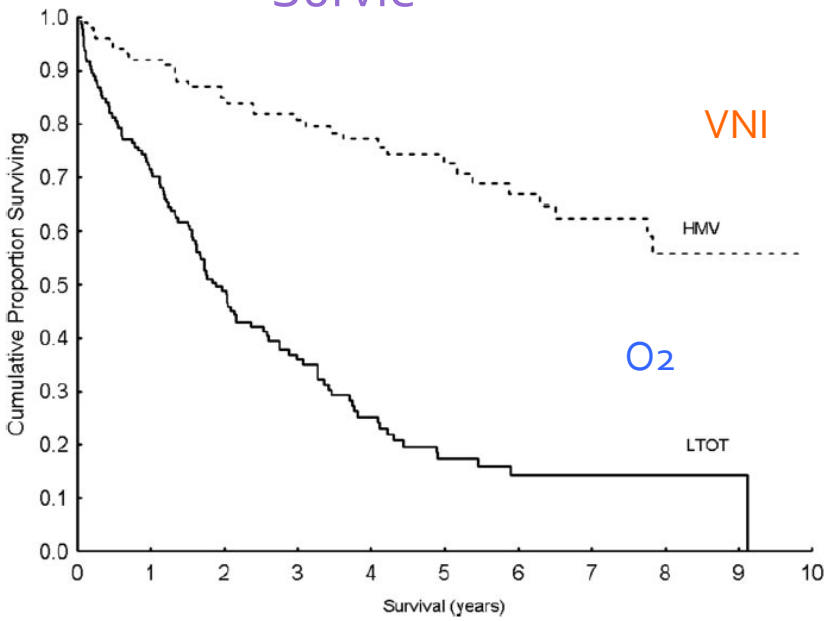
2) **Indication de la VNI** : l'hypoventilation

3) La VNI est elle très efficace ?

VNI et cyphoscoliose



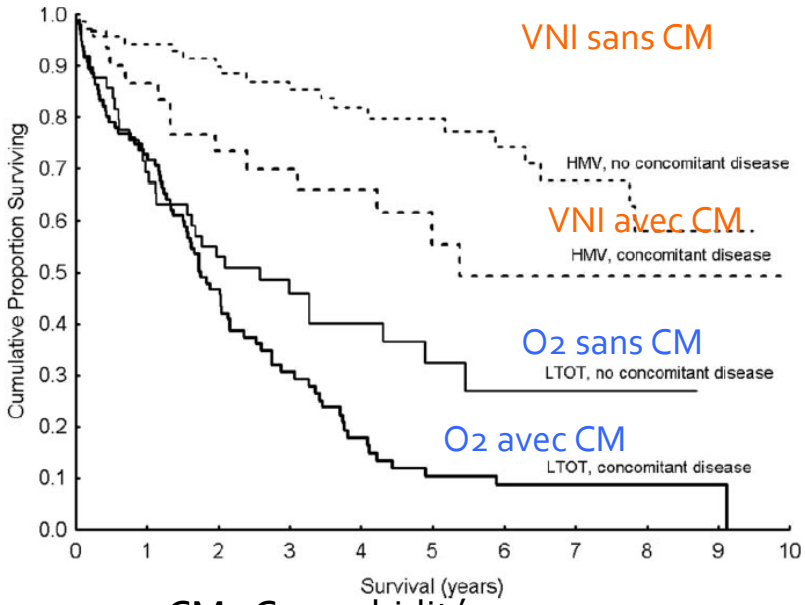
Survie



VNI

O2

Survie



VNI sans CM

VNI avec CM

O2 sans CM

O2 avec CM

CM : Comorbidités

Gustafson T. CHEST 2006;130:1828-33

Oxygénothérapie long terme versus VNI dans les séquelles mutilantes de Tuberculose

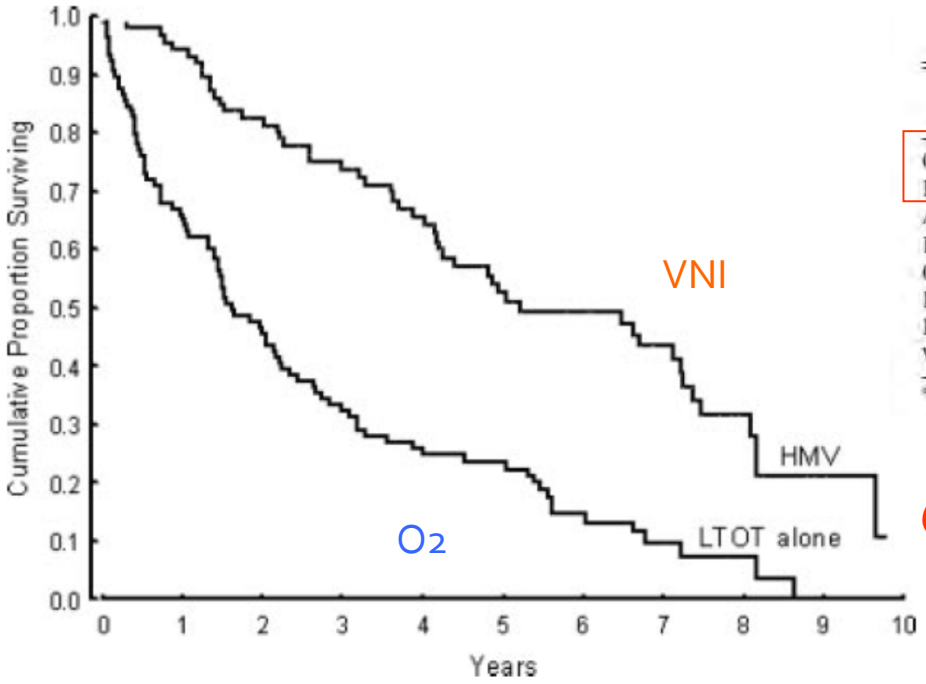


Table 2—*Multivariate Analysis of Relative Risk of Death*

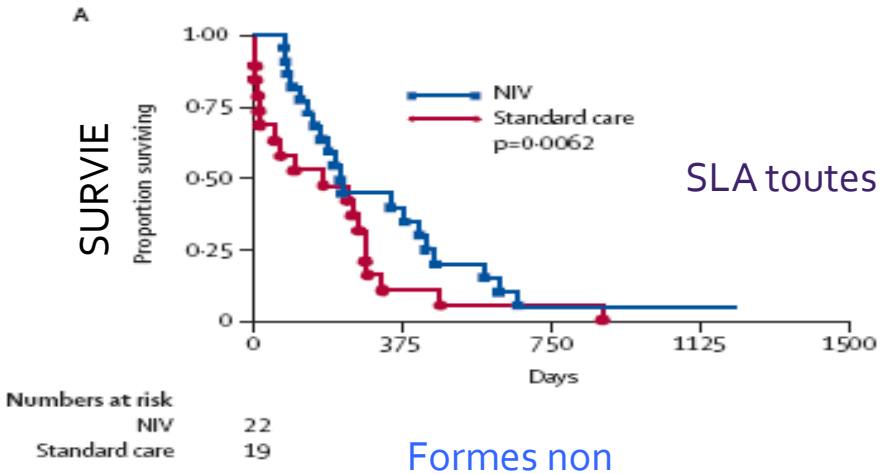
Variables	Hazard Ratio	95% CI	p Value
Oxygen therapy alone	1		
Home mechanical ventilation	0.35	0.17–0.70	0.0028
Age > 70 yr	2.67	1.48–4.83	0.0012
Female gender	0.76	0.50–1.16	0.20
Concomitant disease	0.85	0.45–1.61	0.62
Pao_2, mm Hg*	1.00	0.98–1.03	0.75
$Paco_2$, mm Hg*	0.98	0.95–1.01	0.19
Vital capacity, % predicted*	0.99	0.98–1.01	0.36

*Continuous variables.

(18% concomitant O2)

Jager L. CHEST 2008;133:156-60

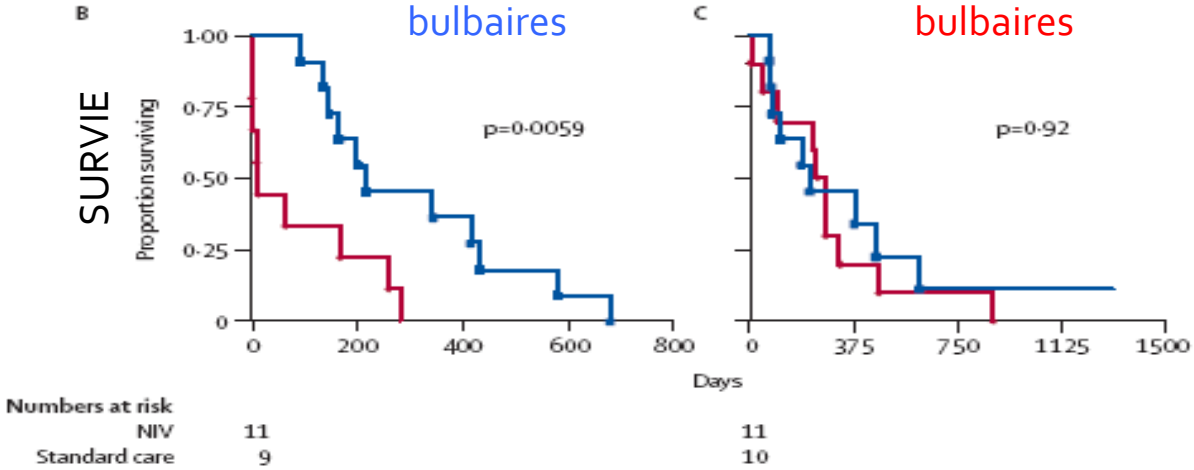
SLA et VNI : survie



SLA toutes formes

Formes non bulbaires

Formes bulbaires



Bourke Lancet Neurol. 2006 Feb;5(2):140-7.

Mortalité SOH/SAOS

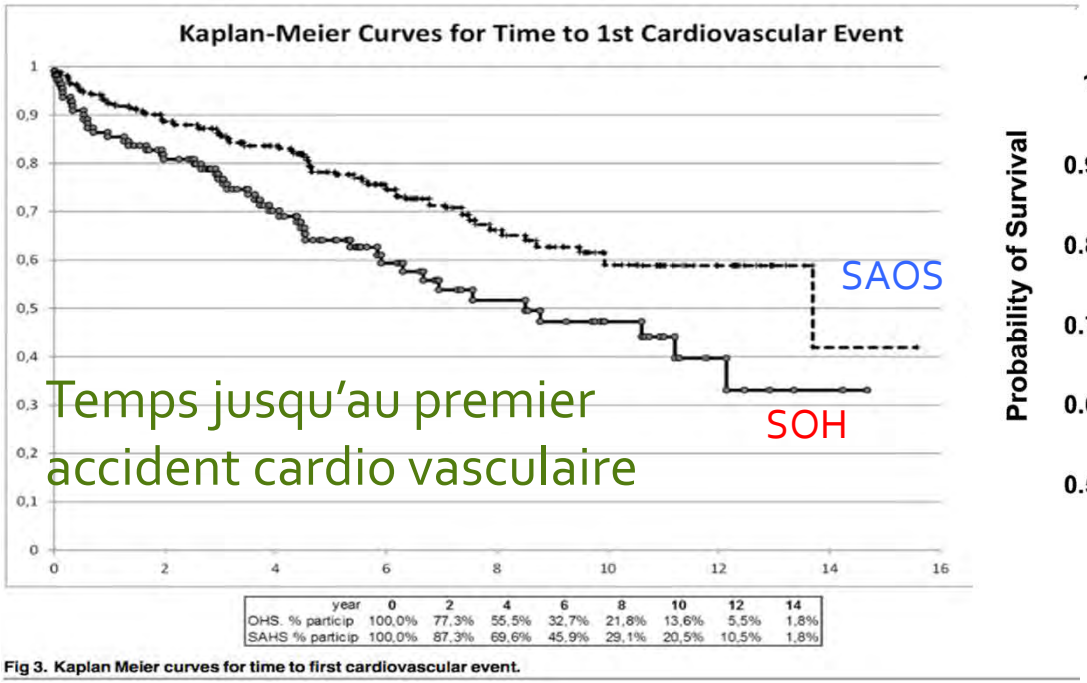
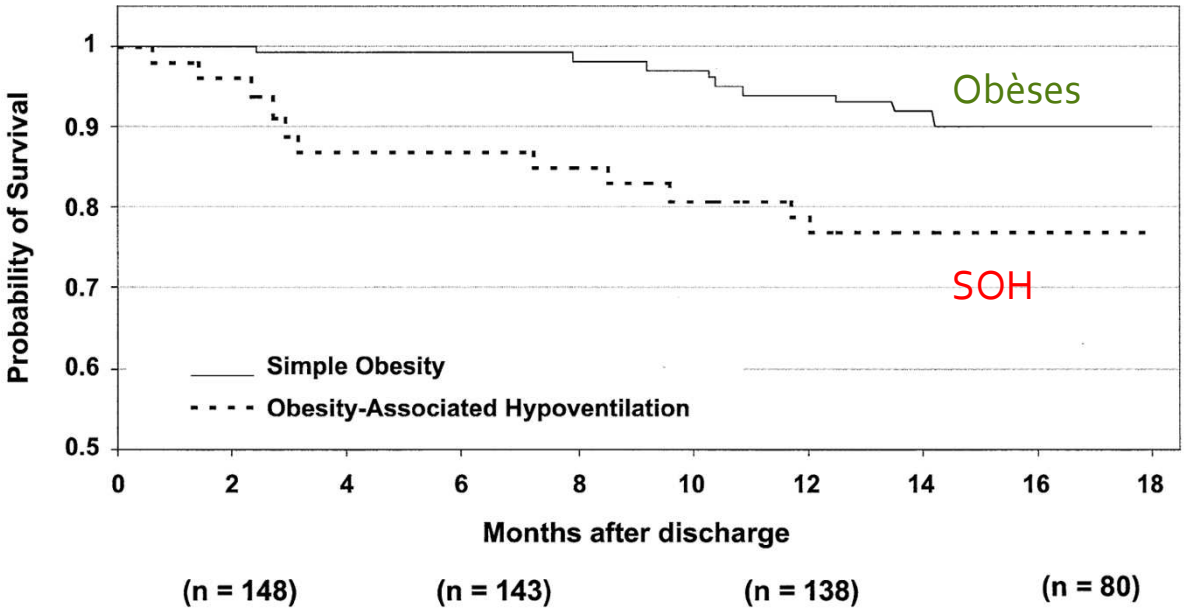


Fig 3. Kaplan Meier curves for time to first cardiovascular event.

plos one. 2015 feb 11;10(2) 2015.obesity-hypoventilation syndrome: increased risk of death over sleep apnea syndrome.castro-añón o1

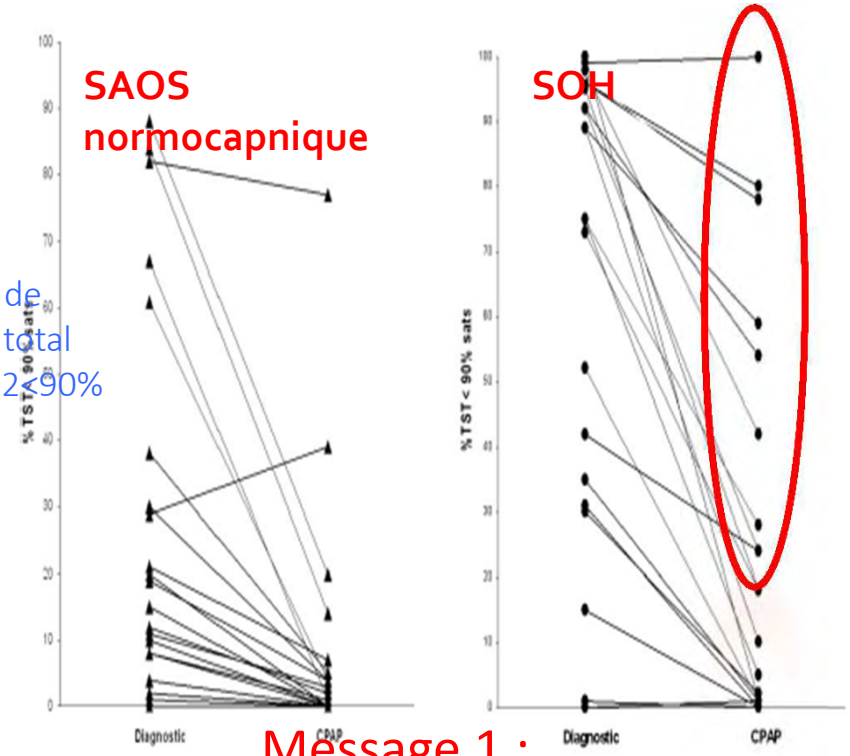


Obesity-associated hypoventilation in hospitalized patients: prevalence, effects, and outcome. Nowbar S Am J Med. 2004 Jan 1;116(1):1-7.

CPAP SOH

SAOS normocapnique

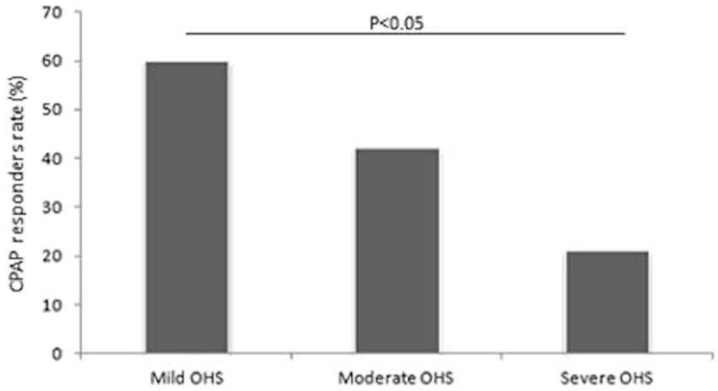
SOH



% temps de sommeil total avec SaO2<90%

	Mild OHS[a] (*n* = 46)	Moderate OHS[b] (*n* = 24)	Severe OHS[c] (*n* = 14)
AHI, events/h	65.4 ± 17.8	73.8 ± 16.5	70.7 ± 17.6
TST90, %	37.8 ± 21.2	55.5 ± 19.4*	86.3 ± 24.5**[§]
Mean SaO_2, %	89.3 ± 3.3	86.6 ± 3.9*	84.1 ± 2.8[§]
SaO_2 nadir, %	68.5 ± 11.7	63.4 ± 12.5	59.2 ± 10.7[#]

Répondeurs à la CPAP la 1ere nuit



Multidiscip Respir Med. 2017 May 18;12:14. Using PaCO2 values to grade obesity-hypoventilation syndrome severity: a retrospective study. Damiani MF

Message 1 :

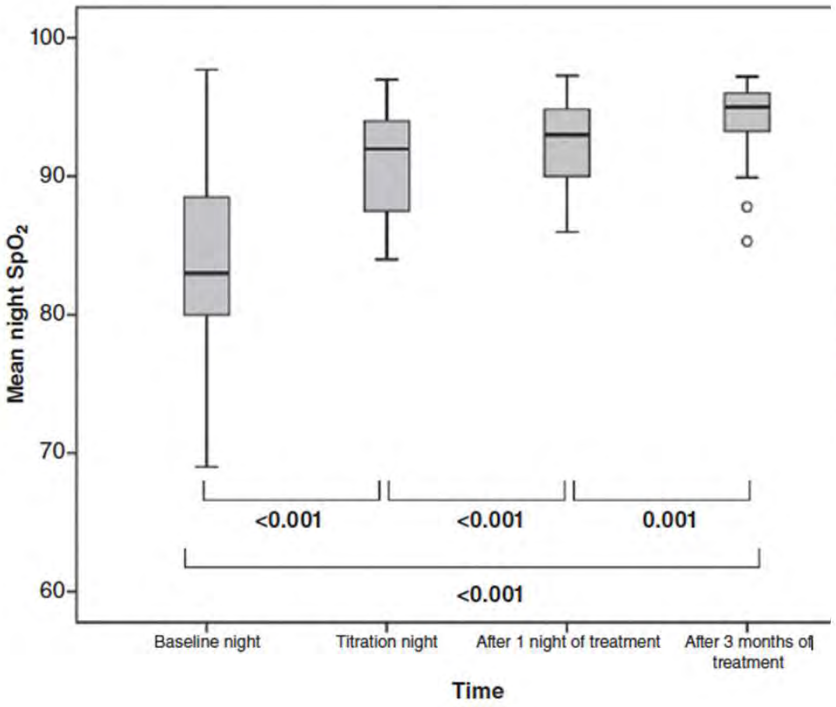
Certains SOH sont non répondeurs à la CPAP

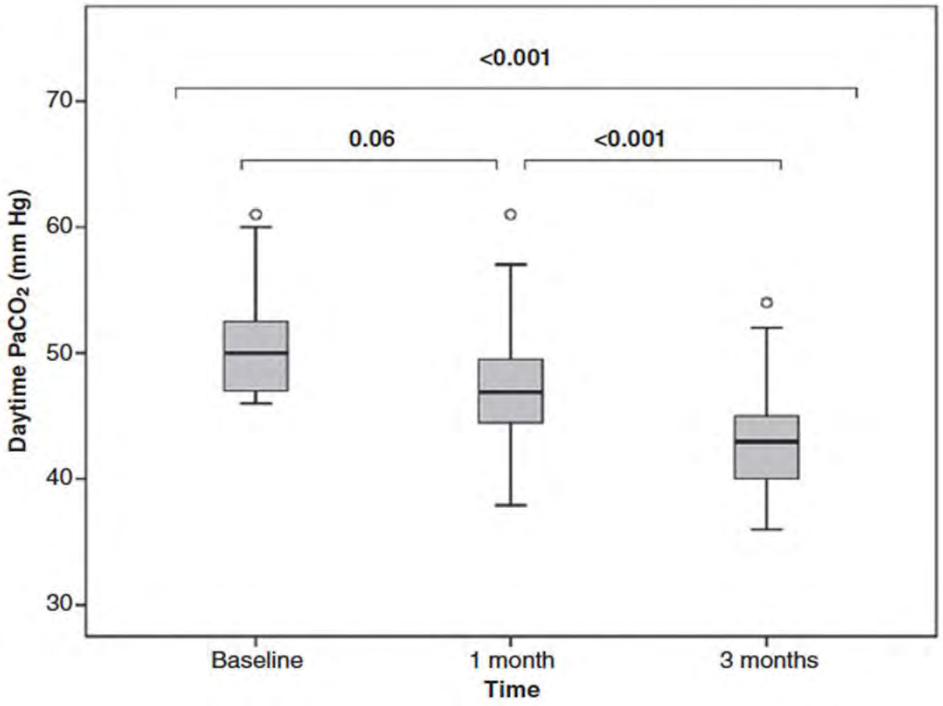
Obesity Hypoventilation Syndrome*Hypoxemia During Continuous Positive Airway Pressure Dev Banerjee, (CHEST 2007; 131:1678–1684)

SOH : effets CPAP

27 pts BMI 42

7 Echecs





SpO2

N=27

PaCO2

Echec si hypoxémie nocturne le 1er jour ou hypercapnie ++ à un mois

IAH médian 74,7

Continuous positive airway pressure in clinically stable patients with mild-to-moderate obesity hypoventilation syndrome and obstructive sleep apnoea NEUS SALORD,Respirology (2013) 18, 1135–1142

Obésité et retentissement respiratoire : Mécanismes

PPC +

RÔLE DU SAS : Obstructions de la voie aérienne supérieure

ALTÉRATION VENTILATION PERFUSION

PPC +

Dégradation des rapports ventilation/perfusion aux bases

PPC +

Augmentation des résistances des petites bronches

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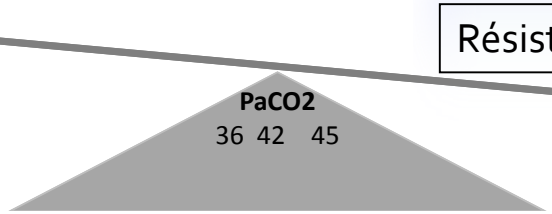
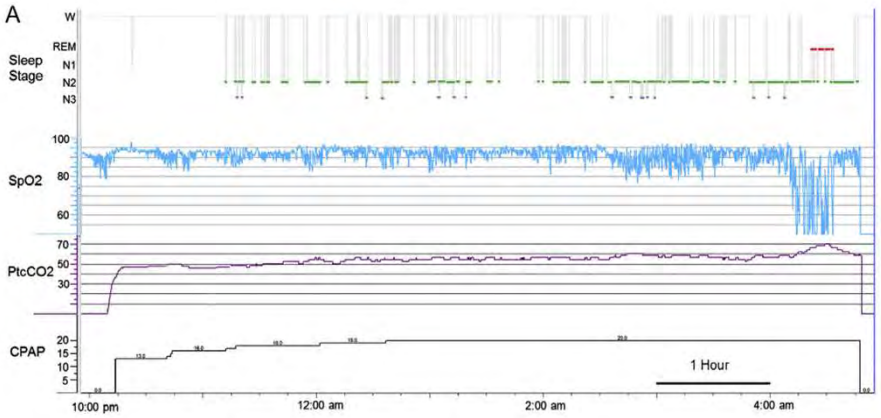
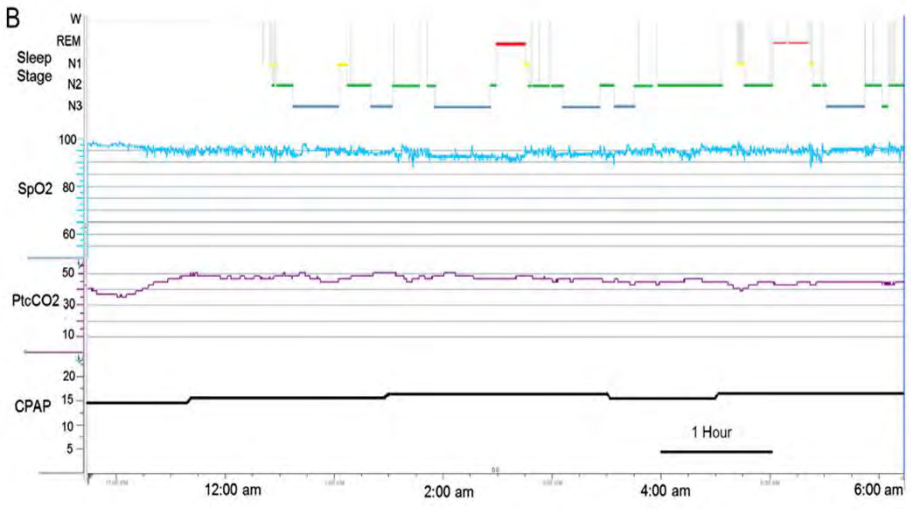


CPAP SOH patience…



(BMI, 46.8 kg/m2; PaCO2, 49 mm Hg Poids 126 kgs

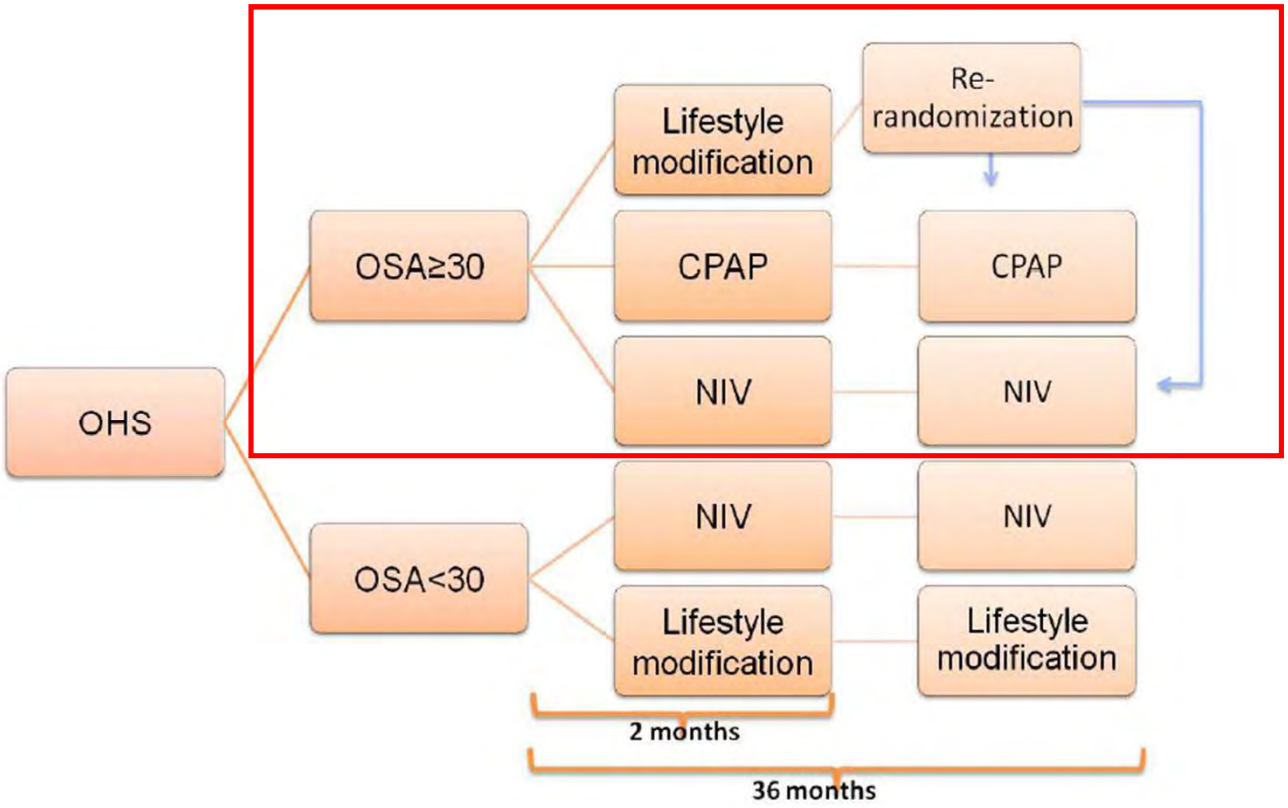
3 mois plus tard



Amanda Piper CHEST 2016; 149(3):856-868

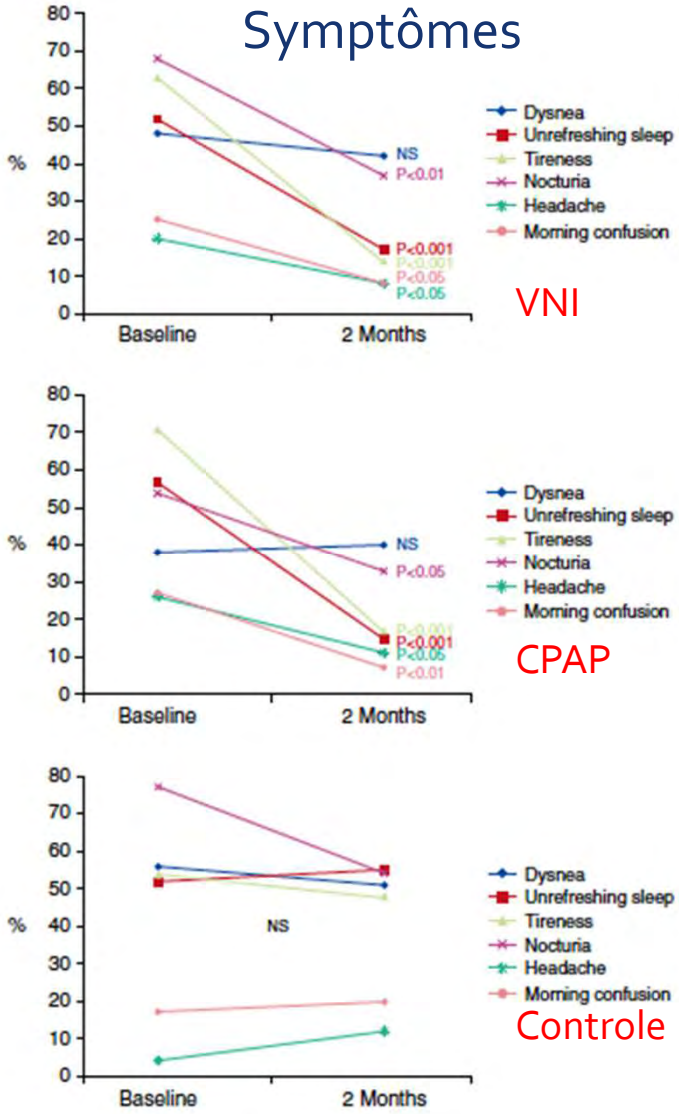
PaCO2 40 mmHg Poids 113 Kgs

CPAP ou VNI

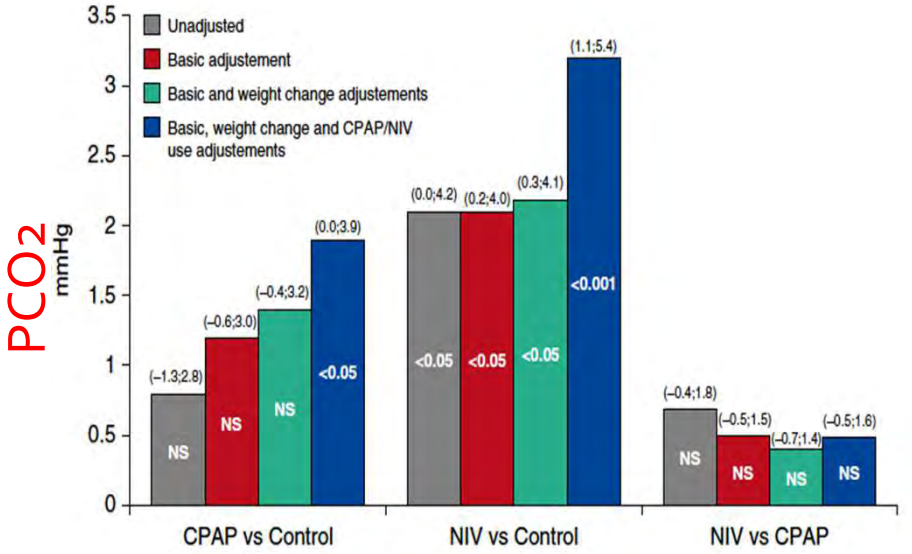


Efficacy of Different Treatment Alternatives for Obesity Hypoventilation Syndrome Pickwick Study Juan F. Masa1,2, American Journal of Respiratory and Critical Care Medicine Volume 192 Number 1 | July 1 2015

Symptômes



CPAP ou VNI



	Baseline [*Mean (SD)*]			Intragroup Differences [*Mean (SD)*]		
	NIV	CPAP	Control	NIV	CPAP	Control
Pa_{CO_2}, mm Hg	51 (4.3)	50 (4.5)	51 (4.2)	−5.5 (7)*	−3.7 (6.6)*	−3.2 (6)*
Bicarbonate, mmol/L	30 (3.4)	30 (4)	30 (3.2)	−2.1 (3.2)*	−1.9 (3.7)*	0.7 (3.1)
pH	7.405 (0.032)	7.403 (0.041)	7.393 (0.036)	0.006 (0.036)	0.007 (0.032)§	0.020 (0.032)*

Efficacy of Different Treatment Alternatives for Obesity Hypoventilation Syndrome Pickwick Study Juan F. Masa1,2, American Journal of Respiratory and Critical Care Medicine Volume 192 Number 1 | July 1 2015

Obésité et retentissement respiratoire : Mécanismes

PPC + RÔLE DU SAS : Obstructions de la voie aérienne supérieure

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PPC + Dégradation des rapports ventilation/perfusion aux bases

PPC + Augmentation des résistances des petites bronches

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VNI + Diminution de la réponse à l'hypercapnie et/ou à l'hypoxémie

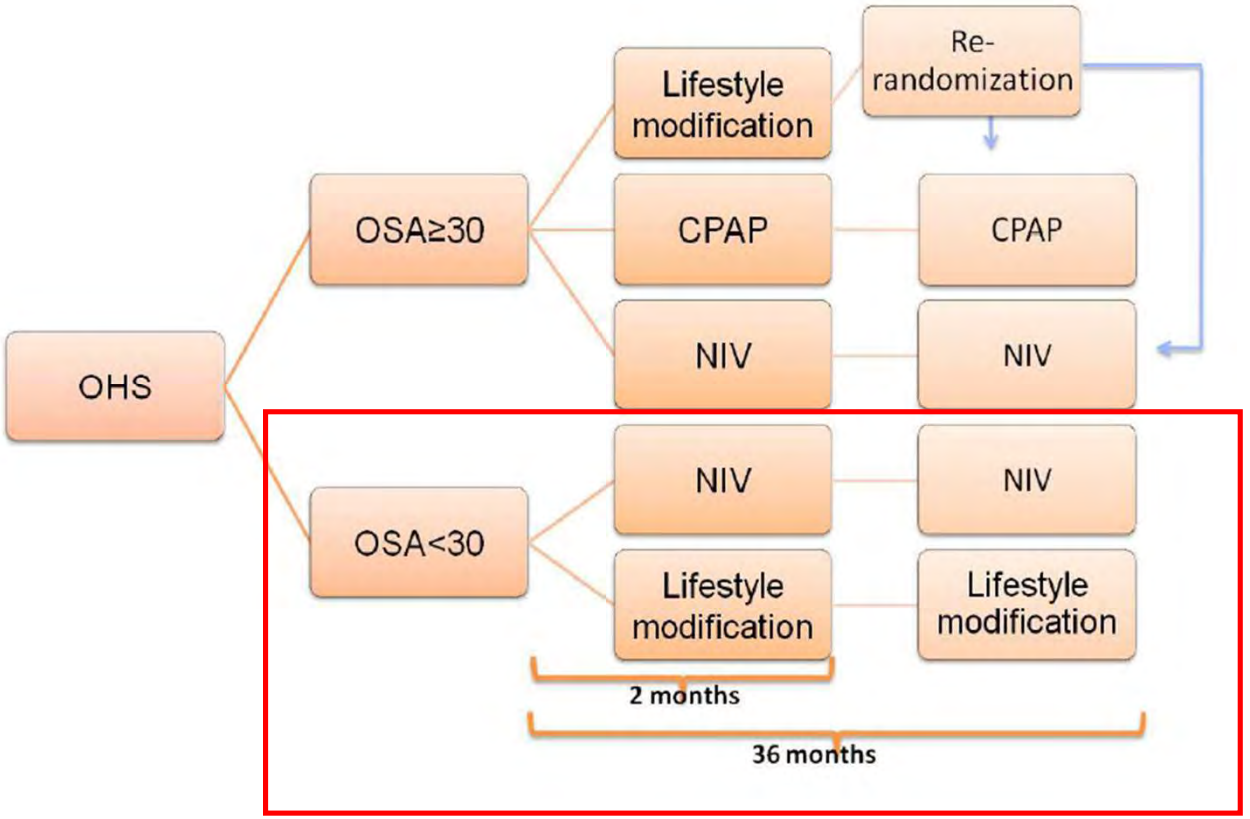
Résistance à la leptine

Augmentation de la ventilation minute

Augmentation de la force du diaphragme

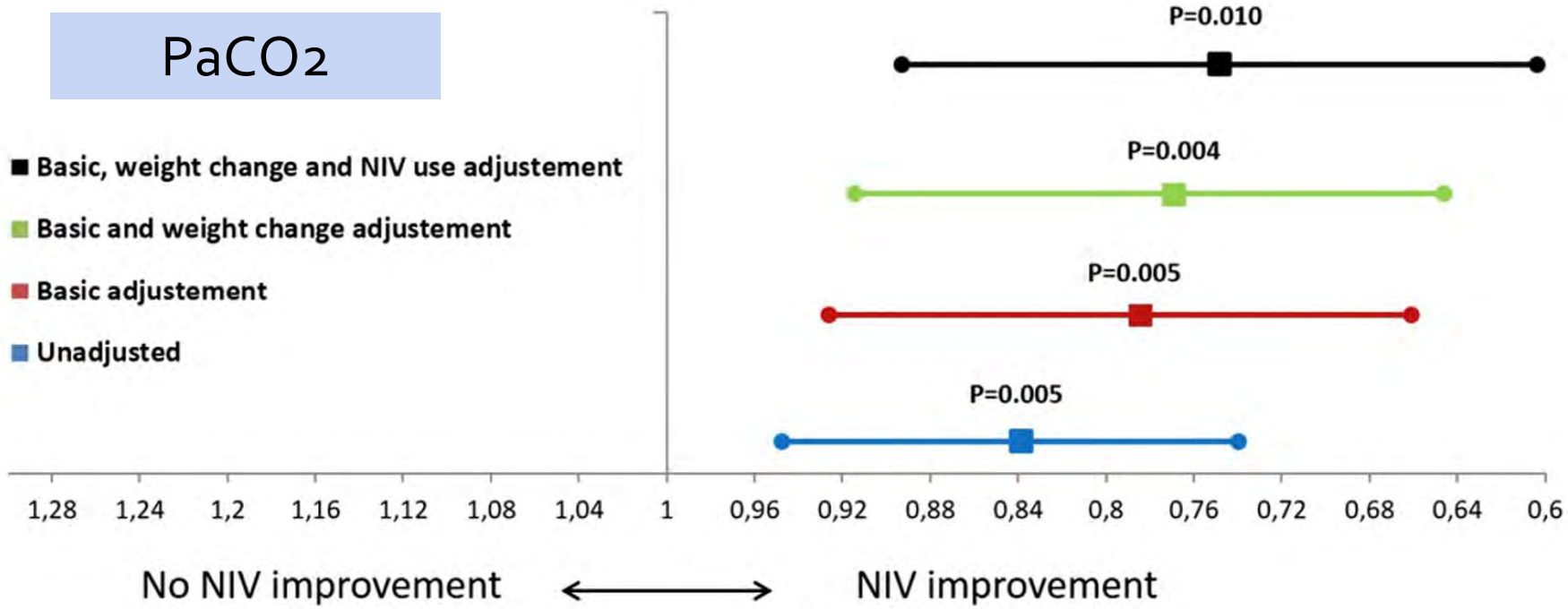
PaCO2
36 42 45

CPAP ou VNI



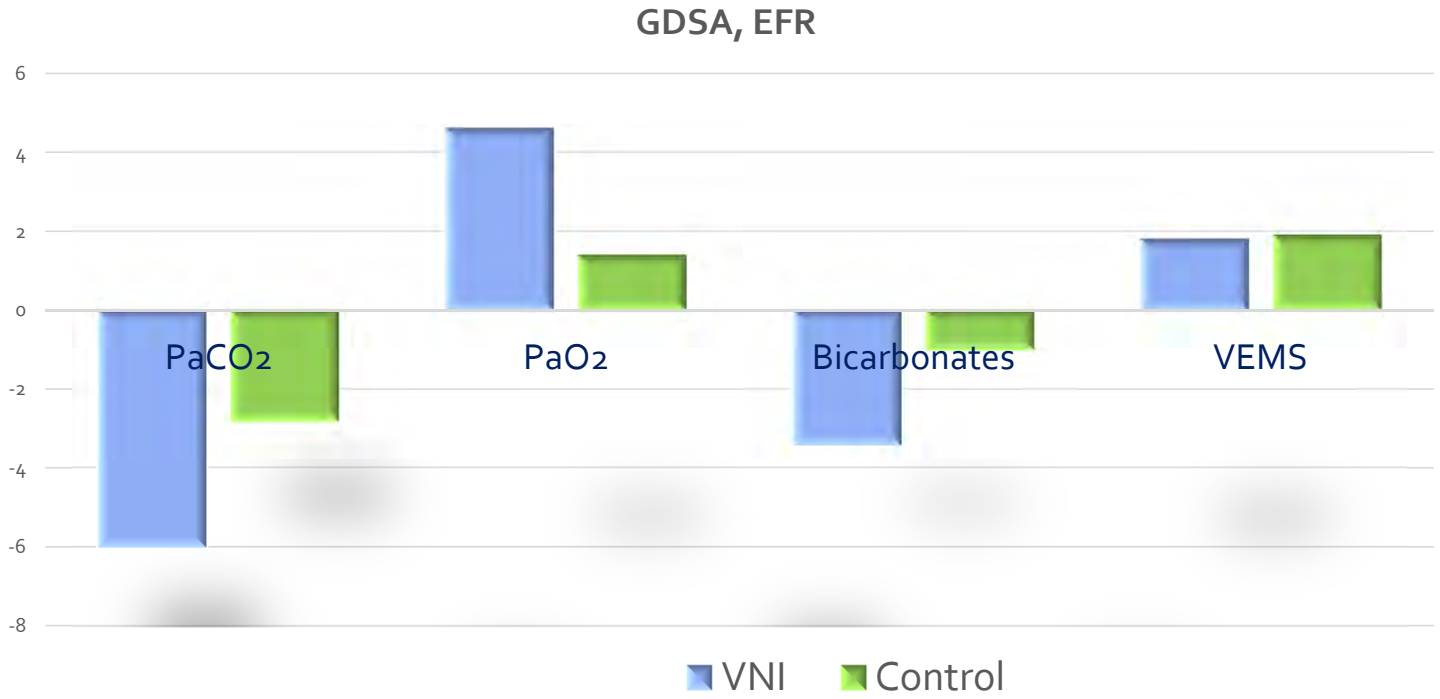
Efficacy of Different Treatment Alternatives for Obesity Hypoventilation Syndrome Pickwick Study Juan F. Masa1,2, American Journal of Respiratory and Critical Care Medicine Volume 192 Number 1 | July 1 2015

VNI ou mesures hygièno diététiques pour SOH sans SAOS



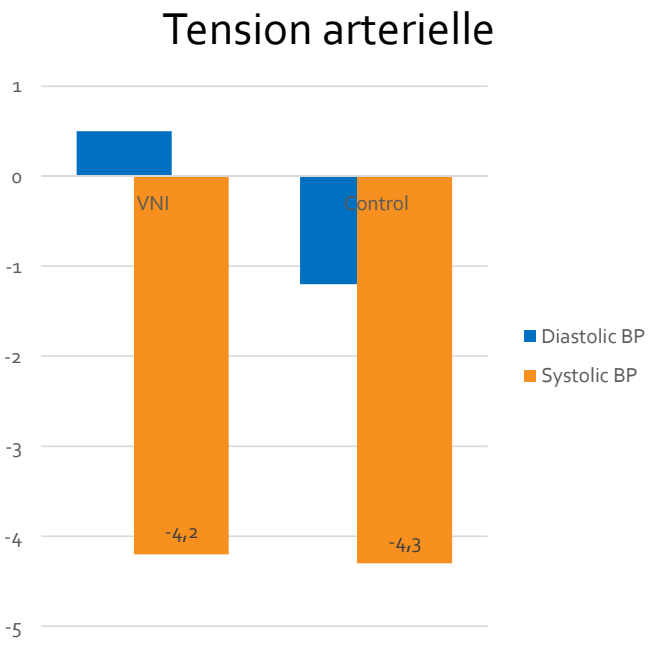
Thorax. 2016 Oct;71(10):899-906. Non-invasive ventilation in obesity hypoventilation syndrome without severe obstructive sleep apnoea. Masa JF

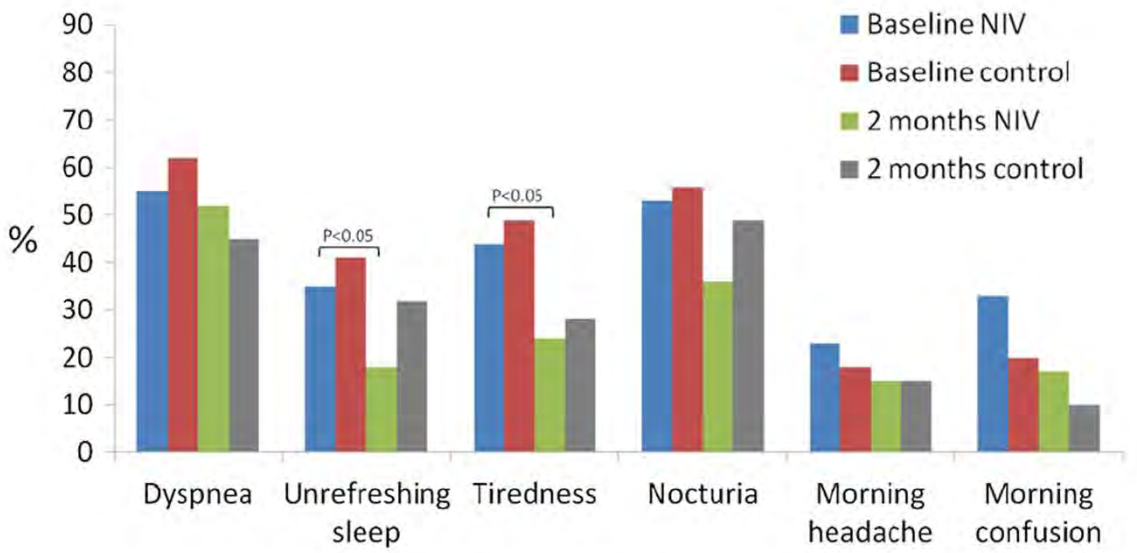
VNI ou mesures hygièno dietetiques pour SOH sans SAOS



Thorax. 2016 Oct;71(10):899-906. Non-invasive ventilation in obesity hypoventilation syndrome without severe obstructive sleep apnoea. Masa JF

VNI ou mesures hygièno diététiques pour SOH sans SAOS

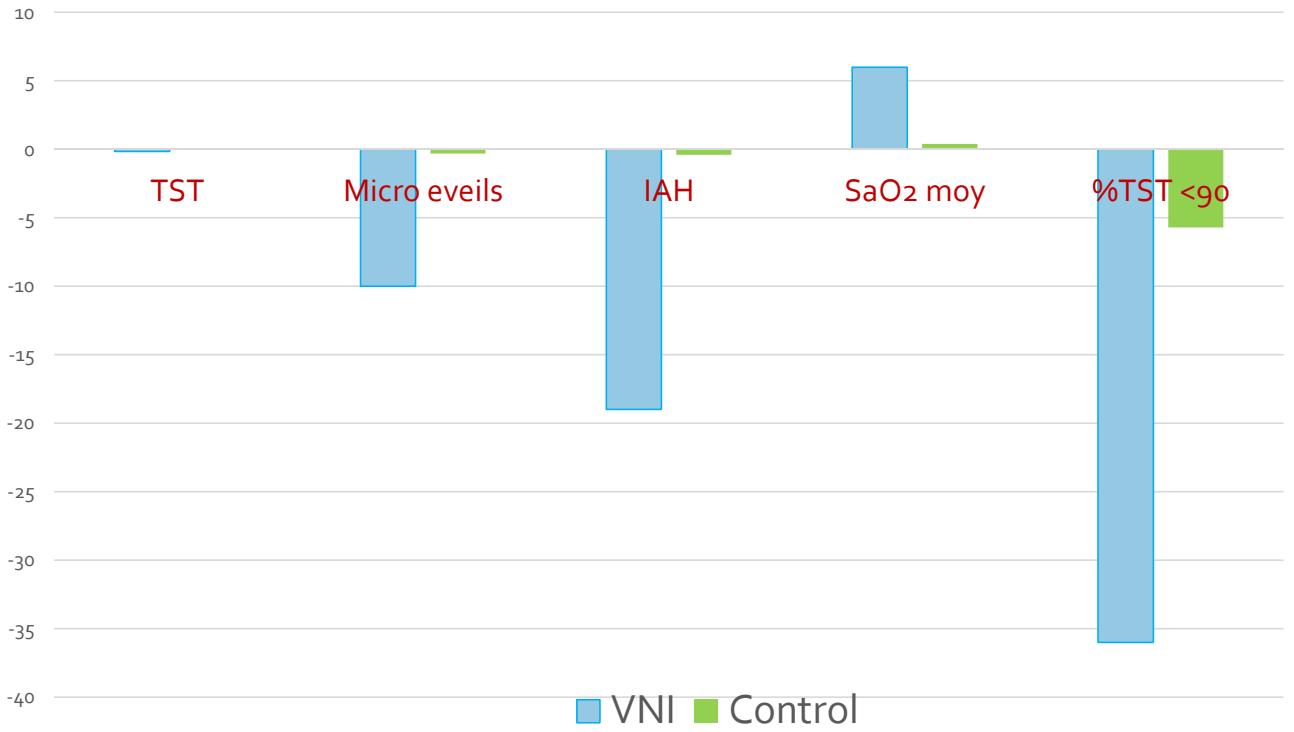




Thorax. 2016 Oct;71(10):899-906. Non-invasive ventilation in obesity hypoventilation syndrome without severe obstructive sleep apnoea. Masa JF

VNI ou mesures hygièno diététiques pour SOH sans SAOS

Critères PSG



Thorax. 2016 Oct;71(10):899-906. Non-invasive ventilation in obesity hypoventilation syndrome without severe obstructive sleep apnoea. Masa JF

VNI ou mesures hygièno diététiques pour SOH sans SAOS

Table 5 Therapy settings, compliance and use of hospital resources

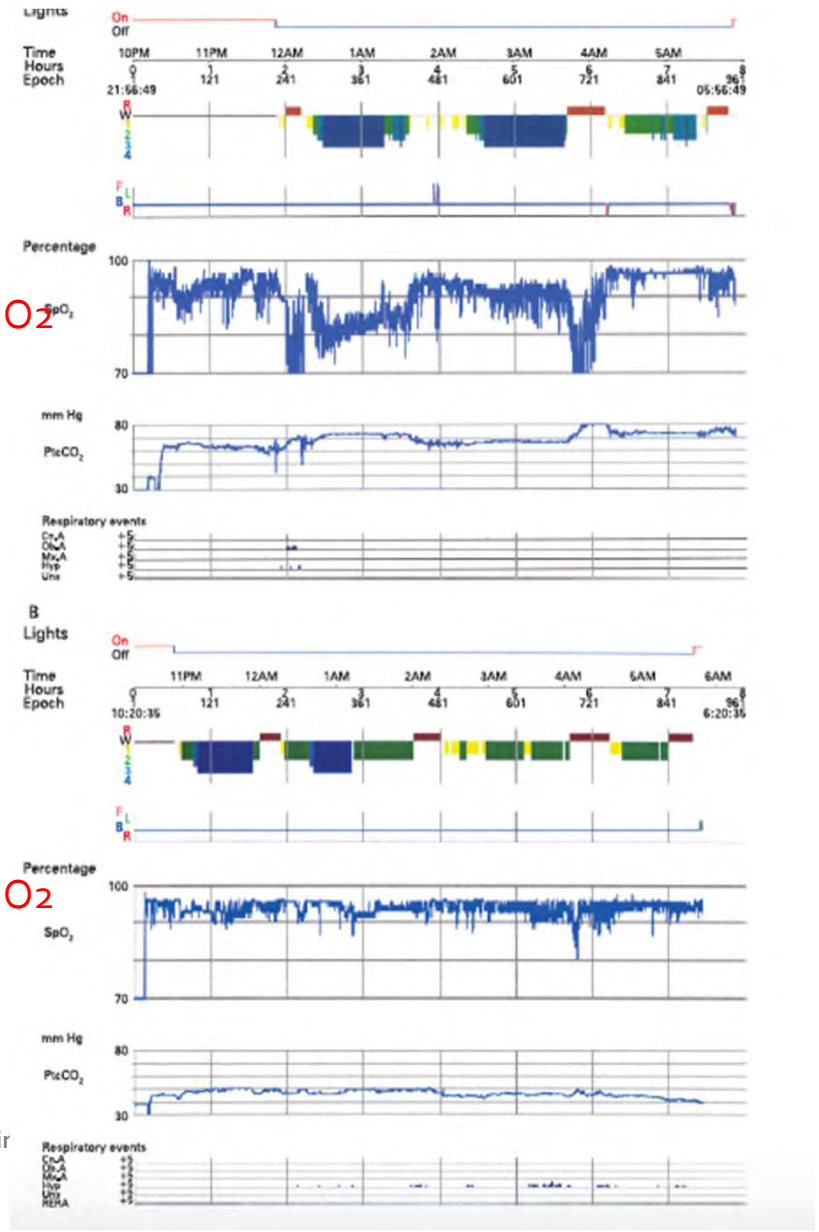
	Baseline	
	NIV	**Control**
Oxygen therapy, %	25	35
Oxygen flow, L/min, mean (SD)	1.8 (09)	1.4 (0.4)
Pressures, cm H_2O, mean (SD)		
IPAP	18.2 (3.4)	–
EPAP	7.1 (1.8)	–
Respiratory rate, mean (SD)	15 (3)	
Mask, %		
Nasal	18	–
Full-face	82	–
Compliance, hours/day, mean (SD)	6 (2.7)	–
Emergency room visit, mean (SD)	**0.05 (0.22)**	**0.23 (0.52)**
Hospital admission rate, mean (SD)	0	0.05 (0.21)
Hospital days, mean (SD)	0	0.65 (3)

Thorax. 2016 Oct;71(10):899-906. Non-invasive ventilation in obesity hypoventilation syndrome without severe obstructive sleep apnoea. Masa JF

Effets de la réduction pondérale

22 ans 156 cm 228 Kgs

Perte de 110 Kgs en 18 mois par régime seul



SaO2

SaO2

Crummy thorax 2008

VNI : amélioration du contrôle neurologique respiratoire

Table 4 Respiratory muscle activity on and off NIV at baseline and during the follow up period

Parameters	Day 0	Day 1	6 weeks	3 months	P value
EMGpara (μV) off NIV	5.5±1.3	5.0±1.1	5.5±1.6	5.1±1.8	0.699
EMGpara (μV) on NIV	–	2.7±0.7	3.8±1.1	3.4±0.9	0.324
EMGpara (%max) off NIV	21.7±8.5	16.2±4.6	18.7±7.9	17.5±7.5	0.080
EMGpara (%max) on NIV	–	8.94±4.1	12.9±5.7	12.0±5.9	0.481
NRDI (a.u.) off NIV	484.2±214.8$^{*,\#,\%}$	316.5±106.2*	369.1±173.2$^{\#}$	351.2±167.1$^{\%}$	0.004
NRDI (a.u.) on NIV	–	166.8±99.4	219.6±105.3	221.2± 116.4	0.626

Values are presented as mean ± SD or median (IQR) unless otherwise stated. Day 0, Day pre non-invasive titration; Day 1, Day post non-invasive titration; 3 months: follow up in 3 months; $^{*,\#,\%}$, significantly different. EMGpara, parasternal intercoastal electromyography; NRDI, neural respiratory drive index; NIV, non-invasive ventilation; SD, standard deviation; IQR, interquartile range.

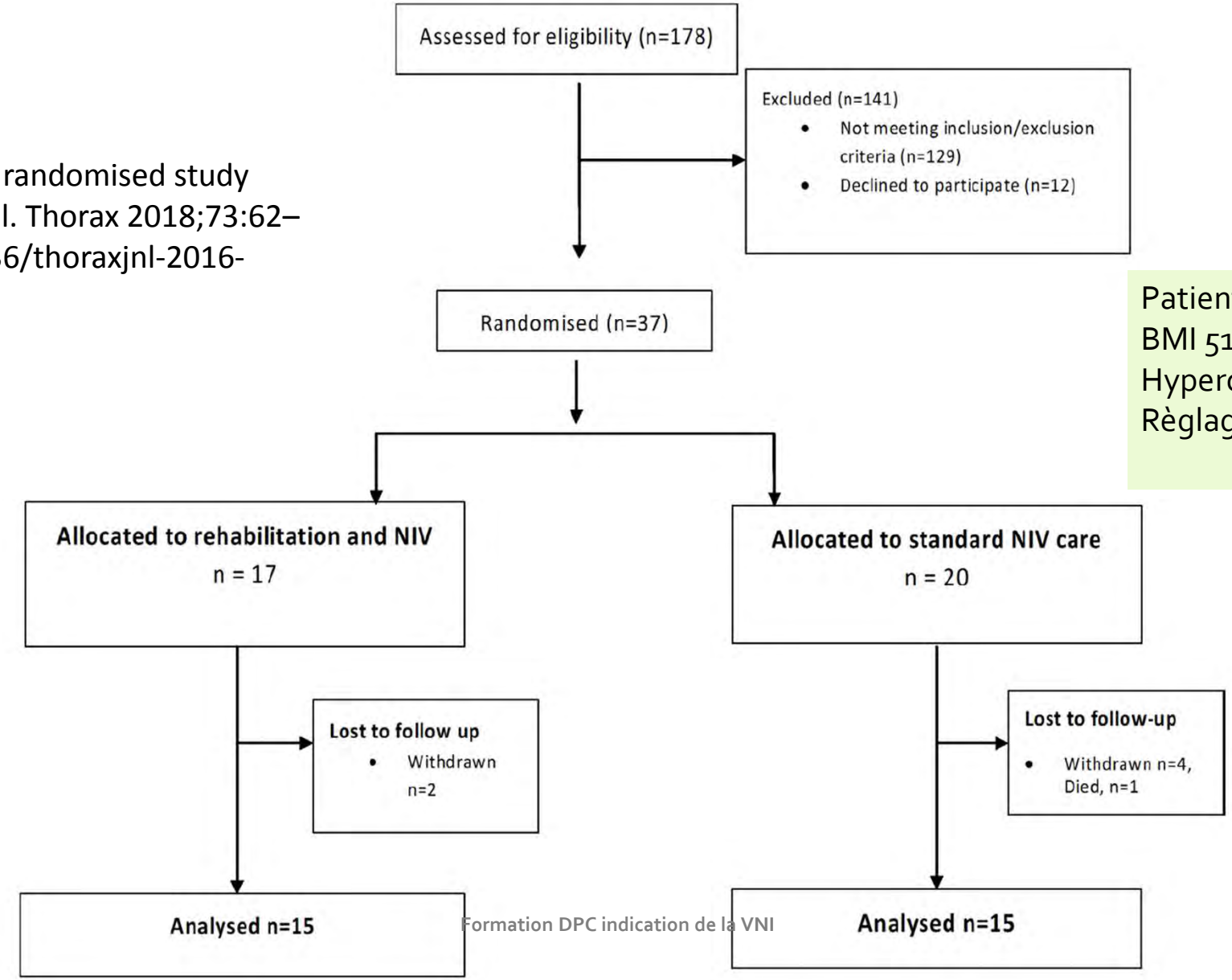
CPAP ou VNI : effets cardio vasculaires

	Baseline, mean (SD)			Intragroup differences, mean (95% CI)			P value of intergroup differences	
	NIV n=71	CPAP n=80	Control n=70	NIV	CPAP	Control	Unadjusted	Adjusted
Systolic PAP, mm Hg	41 (9.2)	38 (12)	41 (10)	−3.4 (−5.3 to −1.5)***	0.02 (−1.7 to 1.7)	−0.44 (−2.7 to 1.9)	0.021† 0.008‡	0.040† 0.033‡
LVEF, %	66 (7.6)	62 (11)	63 (7.9)	−1.9 (−4.1 to 0.23)	1.6 (−0.6 to 3.8)	0.49 (−1.2 to 2.1)	0.027‡	NS
Septum, mm	13 (2.1)	12 (2.4)	13 (2.6)	−0.52 (−0.96 to −0.08)*	0.29 (−0.08 to 0.67)	0.15 (−0.16 to 0.46	0.003† 0.017‡	0.033† 0.031‡
LVPW, mm	12 (2)	11 (2)	12 (2.5)	−0.38 (−0.76 to 0)*	0.29 (−0.35 to 0.41)	0.20 (−0.20 to 0.60	0.042†	0.011†
LV mass, g	225 (52)	211 (63)	237 (73)	−13 (−24 to −2.1)*	5.5 (−5.1 to 16)	8 (−3.1 to 19)	0.016† 0.008‡	0.006† NS
LV mass index, g/m^2	108 (27)	98 (29)	102 (37)	−5.7 (−11 to −4.4)**	2.9 (−2.1 to 8)	3.5 (−1.5 to 8.6)	0.017† 0.014‡	0.013† NS

La VNI est plus efficace que CPAP et control pour améliorer l'HTAP, l'HVG.

Corral J, et al. Thorax 2018;73:361–368.

NERO : a pilot randomised study
Mandal S, et al. Thorax 2018;73:62–69. doi:10.1136/thoraxjnl-2016-209826



Patients SOH
BMI 51
Hypercapnique
Règlages VNI modernes

Table 2 Change anthropometric measures from baseline to 3 months: exploratory secondary analyses post intervention

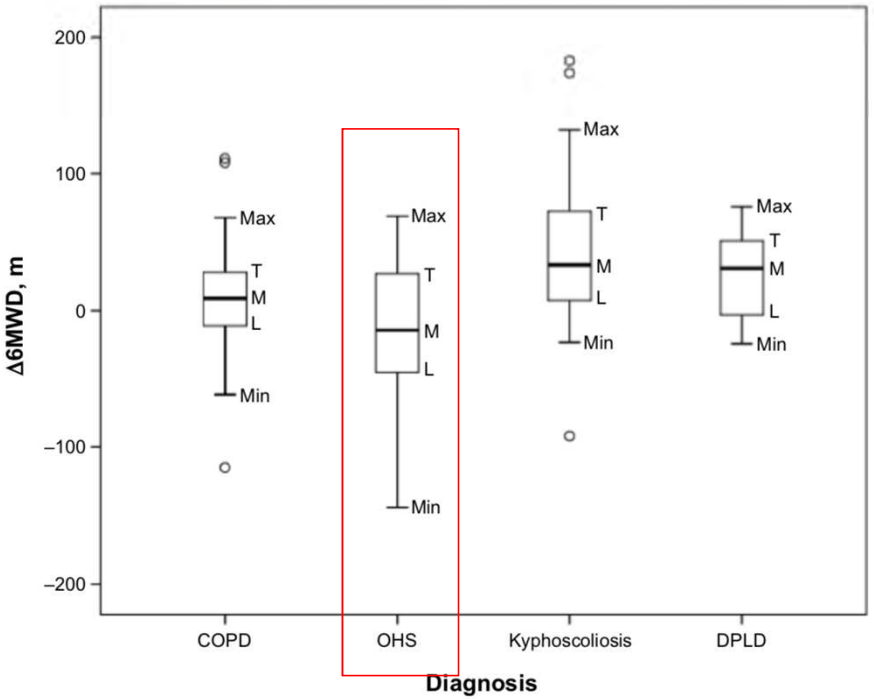
	Control		Intervention				
	Baseline n=20	Δ 3 months n=15	Baseline n=17	Δ 3 months n=15	Mean difference	95% CI	p Value
Weight (kg)	141.2±30.7	−3.0±6.2	139.3±28.8	−9.6±6.7*	−11.8†	−22.1 to −1.45	0.03†
			*p=0003				
BMI (kg/m^2)	50.8±7.5	−1.0±2.6	51.4±8.2	−3.5±2.5*	−2.5†	−4.44 to −0.57	0.01†
			*p=0.0003				
NC (cm)	44.0 (42 to 48)	−1.0 (−2.7 to 1.0)	48.0 (38.0 to 49.4)	−1.5 (−4.8 to 1.9)	−0.52	−2.4 to 1.37	0.58
CC (cm)	138 (129 to 144)	−3.0* (−7.1 to 0)	133.0 (130.2 to 138.7)	−3.0 (−7.2 to 1.5)	0.37	−4.38 to 5.11	0.16
	*p=0.034						
WC (cm)	135.5 (129.5 to 142.0)	−4.0 (−7.0 to 2.6)	132.0 (127.0 to 141.6)	−7.0* (−12.8 to −4)	−6.1†	−11.27 to −1.05	0.02†

Table 3 Changes in exercise capacity from baseline to 3 months: exploratory secondary analyses post intervention

	Control		Intervention				
	Baseline n=18	Δ 3 months n=15	Baseline n=15	Δ 3 months n=14	Mean difference	95% CI	p Value
DASI (AU)	13.1 (5.2 to 28.7)	2.8 (0 to 4.5)	15.5 (10.3 to 31.2)	8* (−2.8 to 14.5)	5.62	−0.63 to 11.86	0.08
DASI (mL/min) estimated peak oxygen uptake	15.1 (11.8 to 22.0)	1.2 (0 to 1.9)	16.2 (14.0 to 23.0)	3.4* (−1.18 to 6.2)	2.41	−0.19 to 5.0	0.07

Etude négative à un an

SOH et activité physique



Comparison of exercise capacity in COPD and other etiologies of chronic respiratory failure requiring non-invasive mechanical ventilation at home: retrospective analysis of 1-year follow-up. **Salturk** C.Int J Chron Obstruct Pulmon Dis. **2015** Nov 26;10:2559-69

	COPD, n=37			OHS, n=34		
	Baseline	Δ in 1 year		Baseline	Δ in 1 year	
	Mean ± SD	Mean	95% CI	Mean ± SD	Mean	95% CI
6MWD,[a] m	332±99	10.43	−4.3 to 25.2	315±74	−13.9	−31.9 to 3.96
Ideal 6MWD, m	550±100	−6.04	−22.02 to 9.92	418±77	−8.84	−13.26 to −4.41
Predicted,[b] %	61.3±15.9	1.09	−1.55 to 3.73	75.7±14.5	−2.39	−6.48 to 1.69
LLN, m	398±15.9	−0.83	−20.36 to 18.7	272±71	−10.31	−16.14 to −4.47

La majorité des patients SOH présentent d'autres incapacités qui limitent l'activité physique

SOH activité physique

53 patients obèses traités par CPAP
IMC entre 35 et 45

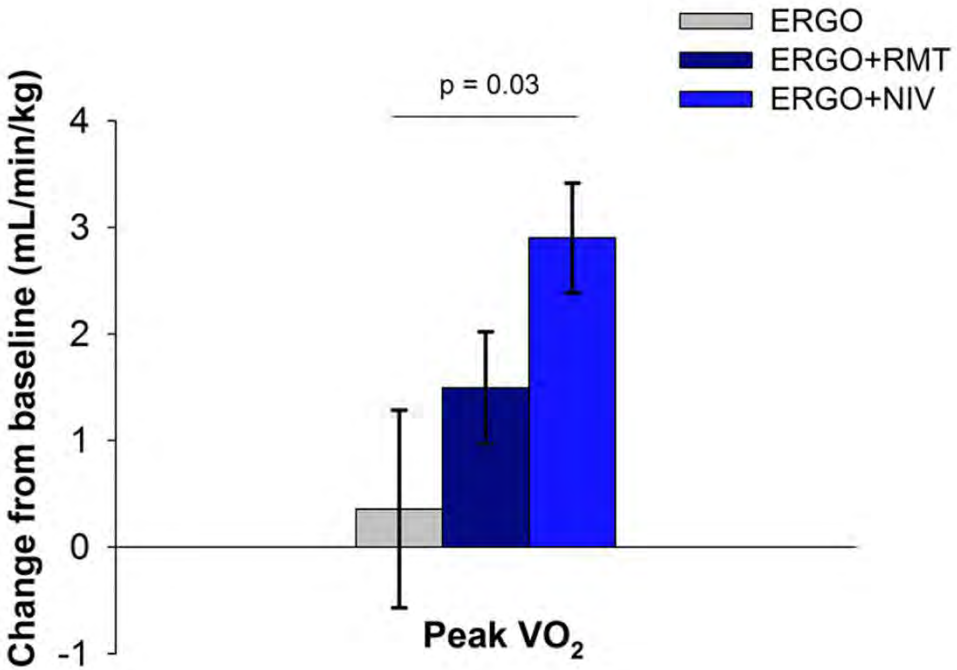
Pas de différence significative en ITT
sur le test de marche 6 minutes entre

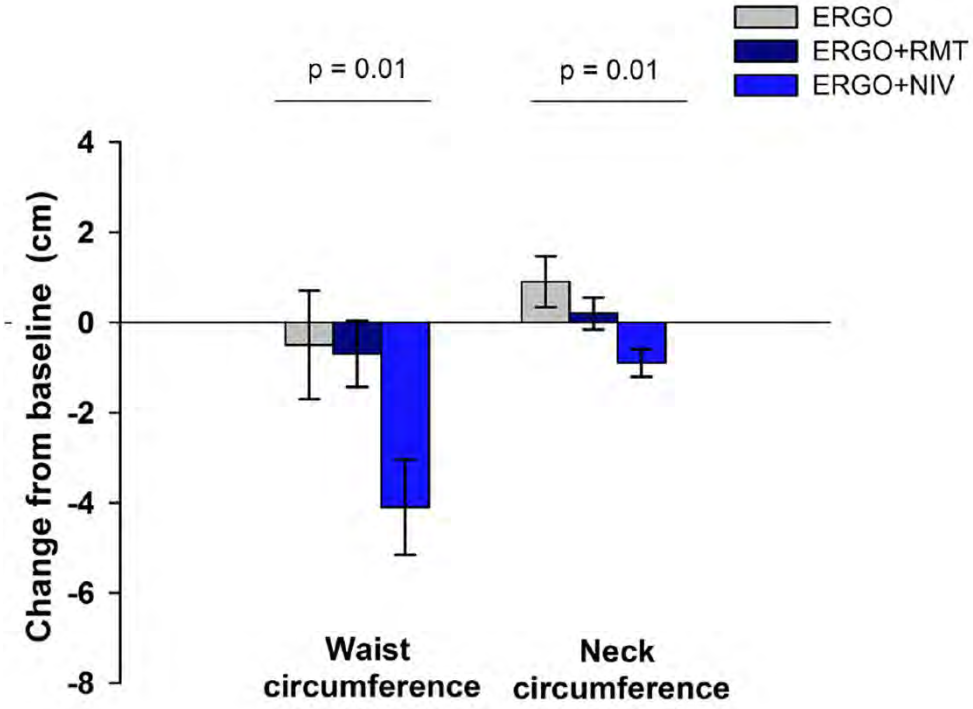
- Ergocycle
- Ergocycle + reentrainement muscles respiratoires
- Ergocycle + VNI

Ventilatory support or respiratory muscle training as adjuncts to exercise in obese CPAP-treated patients with obstructive sleep apnoea: a randomised controlled trial.
Vivodtzev I Thorax. 2018 Feb 20. pii: thoraxjnl-2017-211152.

SOH activité physique

53 patients obèses traités par CPAP
IMC entre 35 et 45





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SOH activité physique

53 patients obèses traités par CPAP
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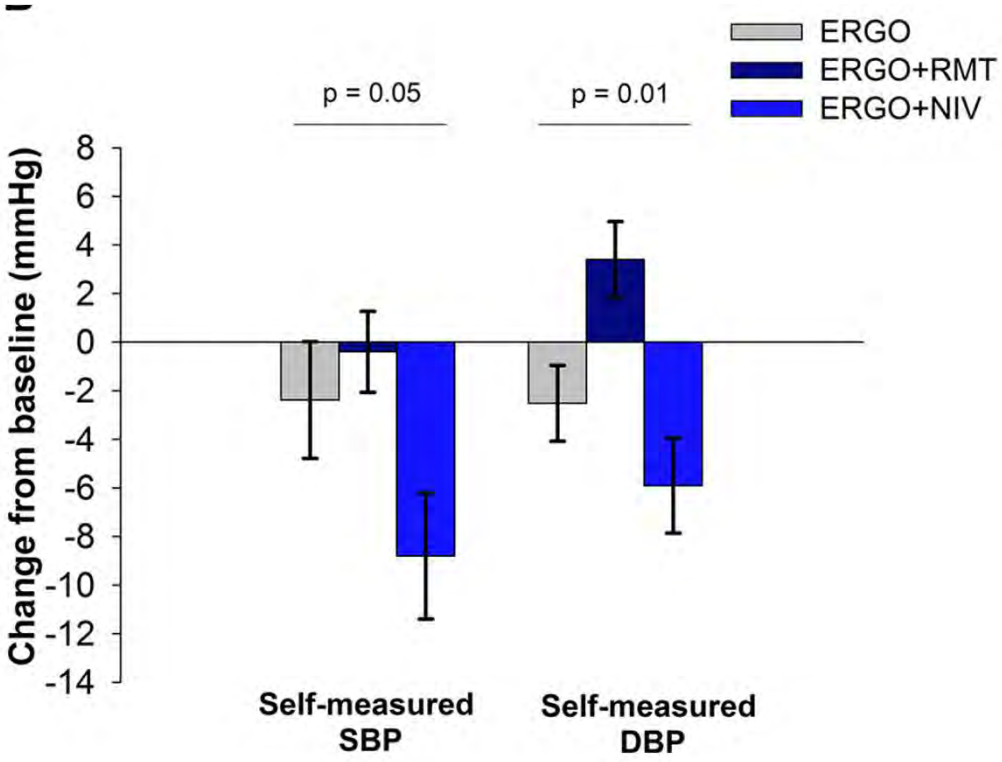


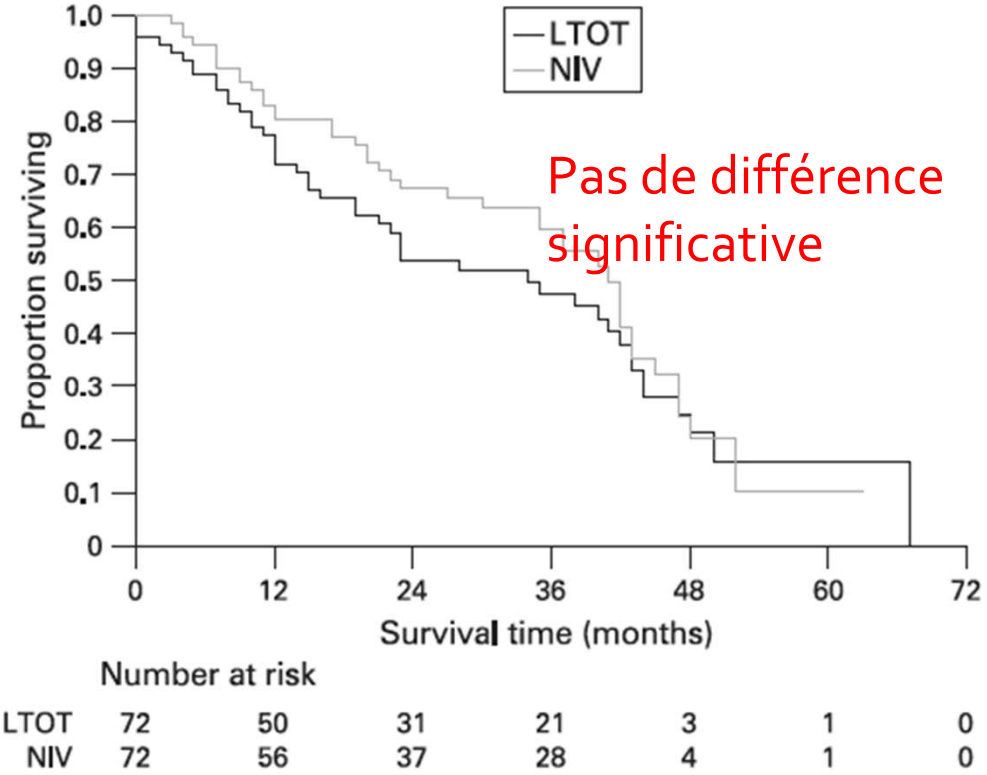
Effet sur la TA

Ventilatory support or respiratory muscle training as adjuncts to exercise in obese CPAP-treated patients with obstructive sleep apnoea: a randomised controlled trial.
Vivodtzev I Thorax. 2018 Feb 20. pii: thoraxjnl-2017-211152.

VNI/BPCO effets bénéfiques

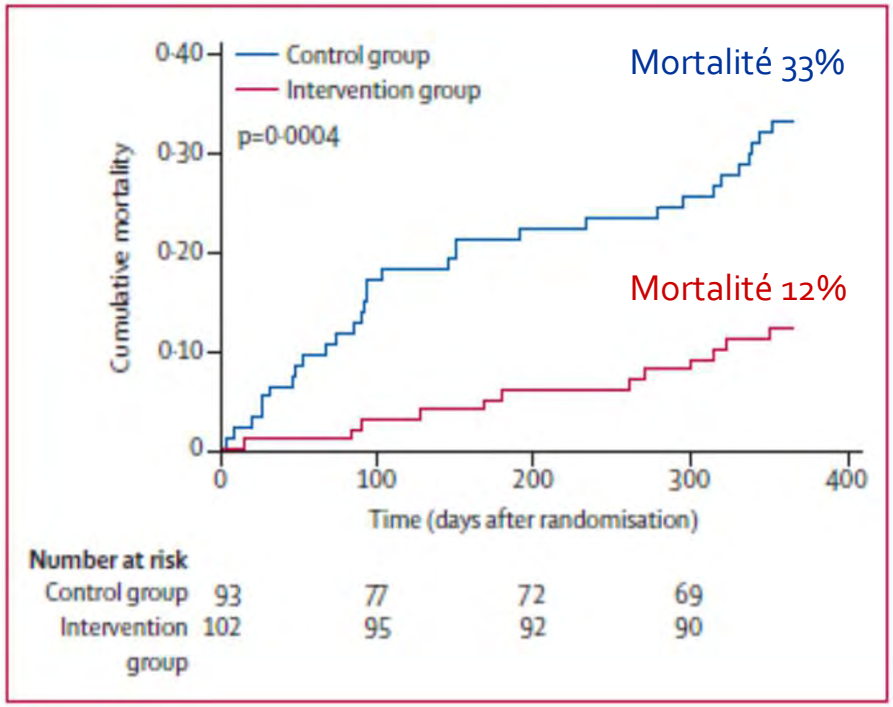
Survie

Nocturnal non-invasive nasal ventilation in stable hypercapnic COPD: a randomised controlled trial
R D McEvoy, Thorax 2009;64:561–566.



Pas de différence significative

VNI / BPCO :2014 tout change

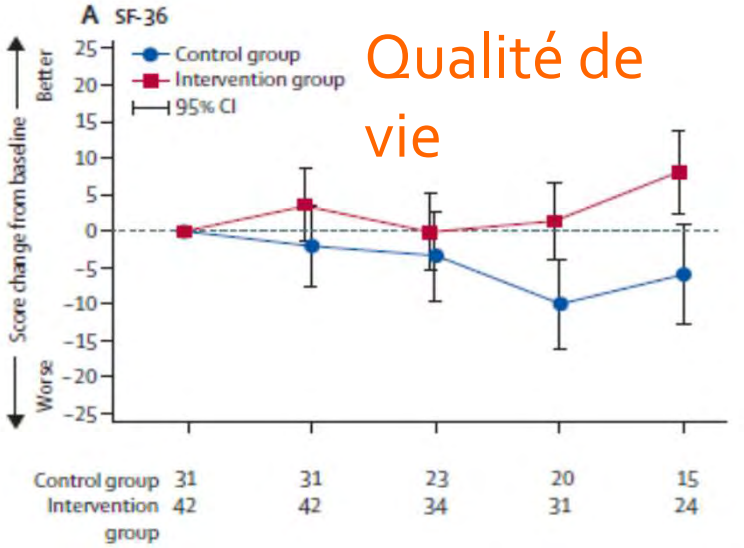


	3 months	6 months	9 months	12 months
Overall	0·8 (3·5)	2·1 (5·7)	0·9 (4·0)	2·6 (8·6)
Non-invasive positive pressure ventilation group	0·2 (1·1)	1·4 (4·7)	1·3 (4·9)	2·2 (10·2)
Control group	1·5 (4·9)	3·0 (6·9)	0·4 (1·9)	3·1 (5·4)

Values are mean (SD).

Hospitalisations

Table 2: **Emergency hospital admissions per patient by follow-up period and treatment group**



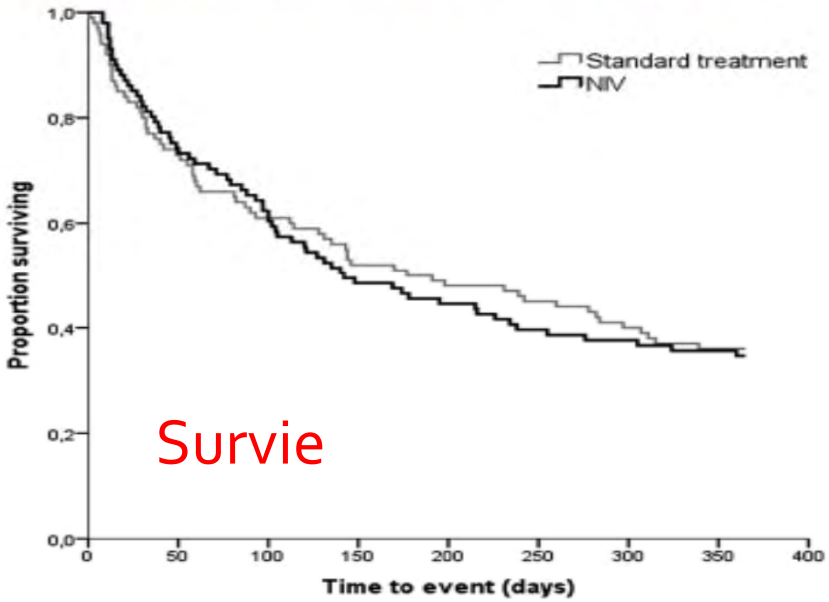
Thomas Köhnlein, Lancet Respir Med 2014 July 25, 2014

BPCO au décours d'une IRA hypercapnique

Etude randomisée multicentrique

BPCO admis en réa pour IRA et restant hypercapnique 48h après sevrage de la VNI

TT standard vs TT standard + VNDP; CJP = délai avant réadmission pour IRA ou DC



Survie

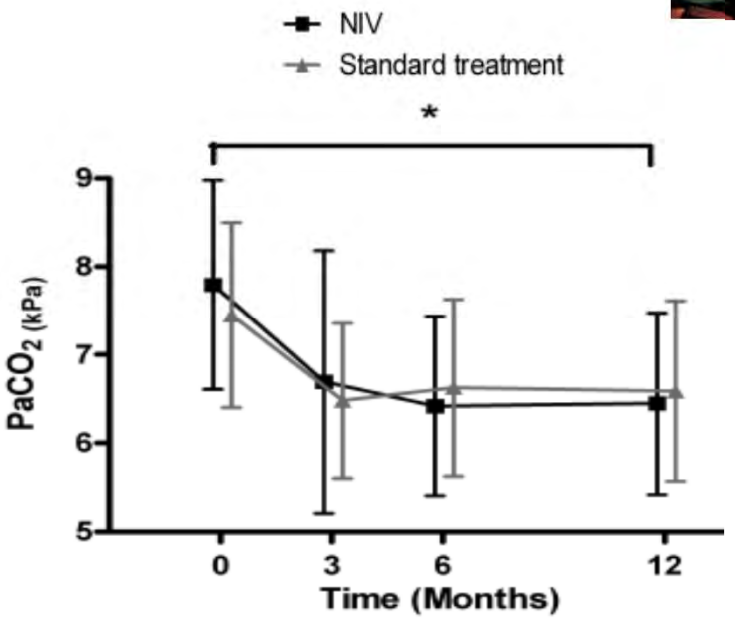


Table 4 Changes in health-related quality-of-life (HRQL) measurements

	ITT baseline	n	Completers baseline	n	Completers 12 months	n	Change over 1 year	n
SRI-Total								
NIV	48.14±14.97	100	47.9±15.1	50	55.0±15.4	50	7.0 (3.4 to 10.7)*	50
Controls	51.33±15.87	90	53.6±16.9	51	55.8±16.3	51	2.2 (−1.2 to 5.6)	51

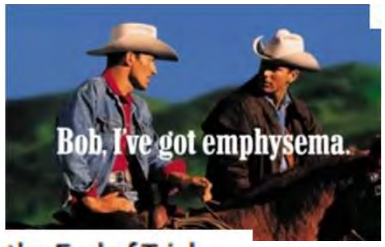
Qualité de vie

Struik, Thorax 2014

P=0.054

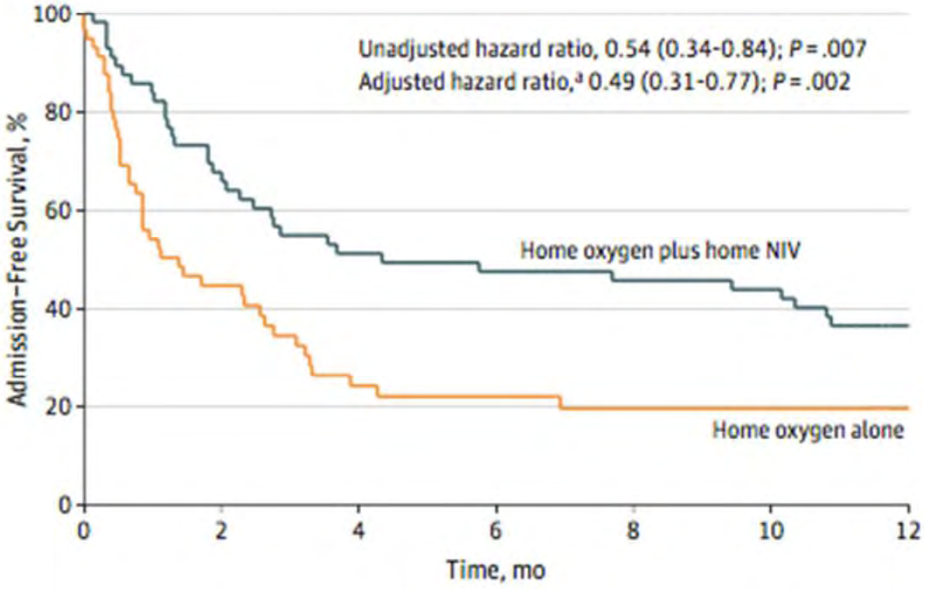
BPCO et VNI après exacerbation

(entre 2 et 4 semaines)



Chez BPCO hypercapnie persistante après exacerbation, la VNI ajoutée à l'o2 **allonge le temps avant la rehospitalisation et diminue le risque de décès**.

Figure 2. Kaplan-Meier Survival Plot of Time to Readmission or Death From Randomization to the End of Trial Follow-up at 1 Year



No. at risk							
Home oxygen plus home NIV	57	37	28	26	25	24	16
Home oxygen alone	59	23	11	10	8	8	6

BPCO sévères
VEMS 23%
PaO2 48 mmHg AA
PaCO2 59 mmHg

Effect of Home Noninvasive Ventilation With Oxygen Therapy vs Oxygen Therapy Alone on Hospital Readmission or Death After an Acute COPD Exacerbation A Randomized Clinical Trial Patrick B. Murphy, **JAMA** Published online May 21, 2017

VNI/BPCO

- Pas d'initiation de la VNI juste après une exacerbation hypercapnique. Attendre deux à six semaines après
- Si la capnie est >55 mm de mercure ou necessité d'O2, la VNI doit être envisagée.
- L'efficacité de la VNI doit être confirmée par une mesure transcutanée nocturne de la pression partielle en CO2, et les réglages sont ajustés pour obtenir une reduction d'au moins 20% de la capnie.
- Si cela n'est pas obtenu alors réaliser une PG sous VNI.
- Si non tolérée => arret VNI

Sommeil BPCO

Overlap syndrome

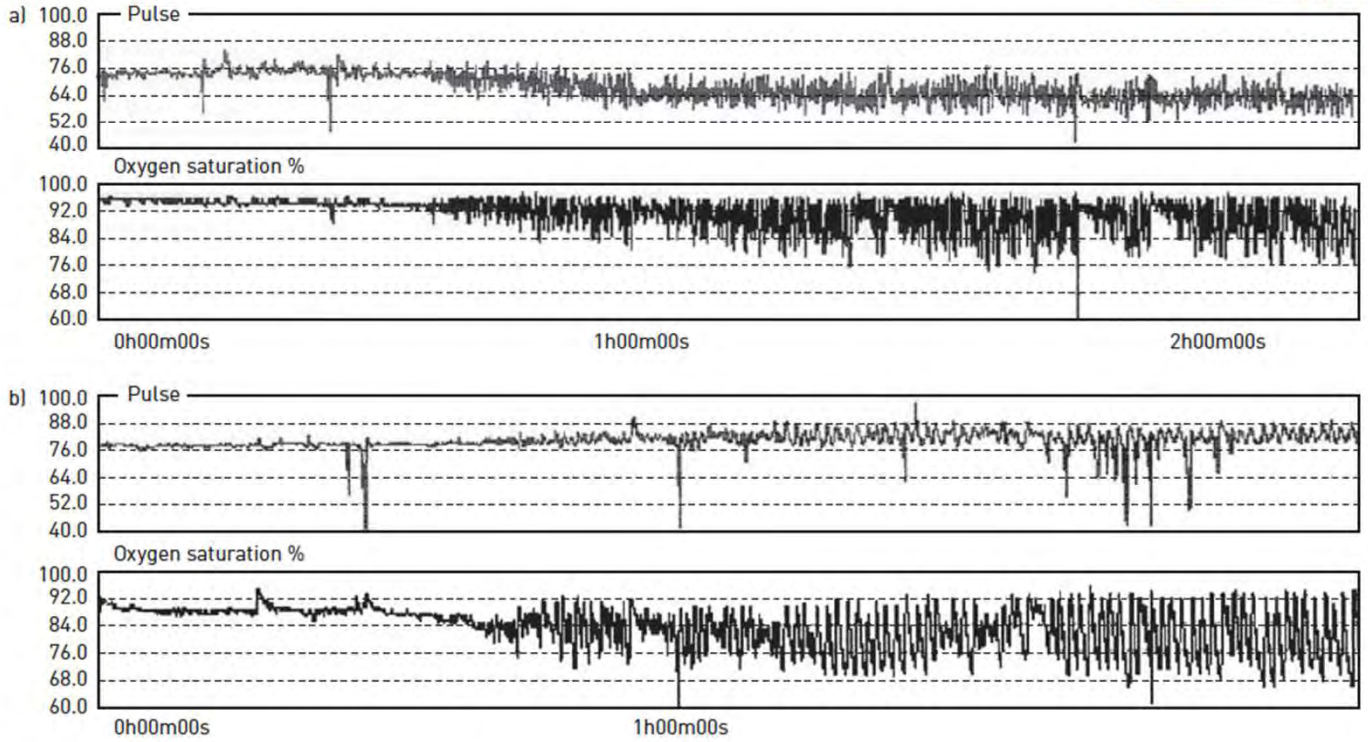
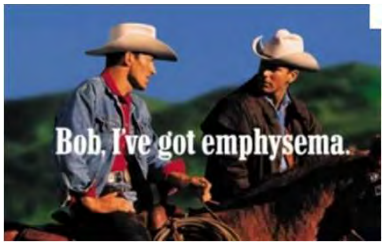


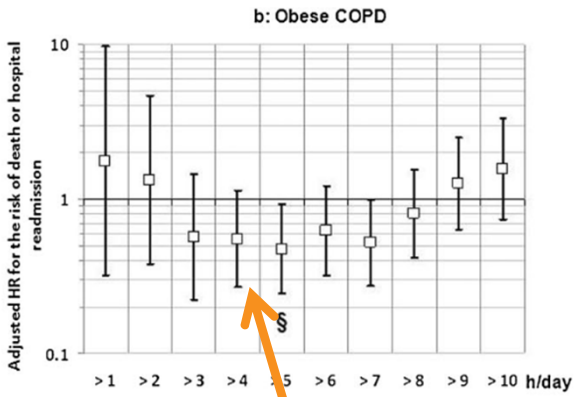
FIGURE 3 Arterial oxygen saturation (SaO_2) patterns during sleep in obstructive sleep apnoea (OSA) alone and the overlap syndrome. SaO_2 patterns in a patient with a) OSA alone and b) overlap syndrome demonstrating the persisting pattern of desaturation in the overlap patient whereas the OSA patient returns to normal SaO_2 between apnoea events.

Sleep disorders in COPD: the forgotten dimension Walter T. McNicholas1,Eur Respir Rev 2013; 22: 365–375

VNI BPCO obese/non obese

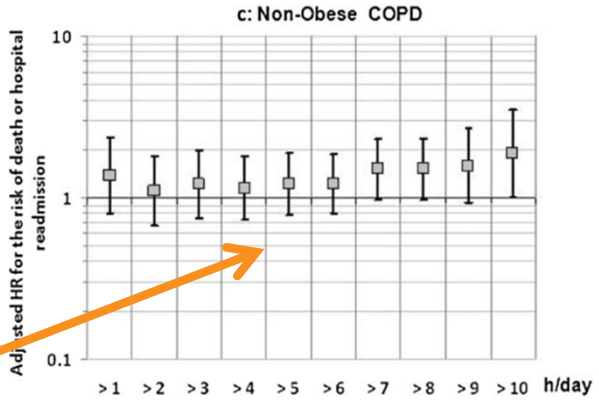


BPCO Obèse



Risque de décès ou d'hospitalisation

BPCO non Obèse



Le risque d'hospitalisation est supérieur dans le groupe VNI non obèse / groupe VNI obèse

JC Borel *Respirology* (2014) **19,** 857–865

Message

BPCO obèse : meilleur pronostic

choix du type de masque, circuit

- AI 10
- EPAP 6
- Fr 15
- Trigger inspi permettant un faible effort inspiratoire pour declencher
- Trigger expi avec un important effort pour declencher l'expiration
- Pente 100 ms
- Ti 1,4 sec