

## Déclaration de liens d'intérêts

J'ai actuellement, ou j'ai eu au cours des trois dernières années, une affiliation ou des intérêts financiers ou intérêts de tout ordre avec les sociétés commerciales suivantes **en lien avec la santé**.

Pas de Conflit d'Intérêt

# Ventilation Non Invasive Etat de Veille, Sommeil, Quelle Influence ? !...

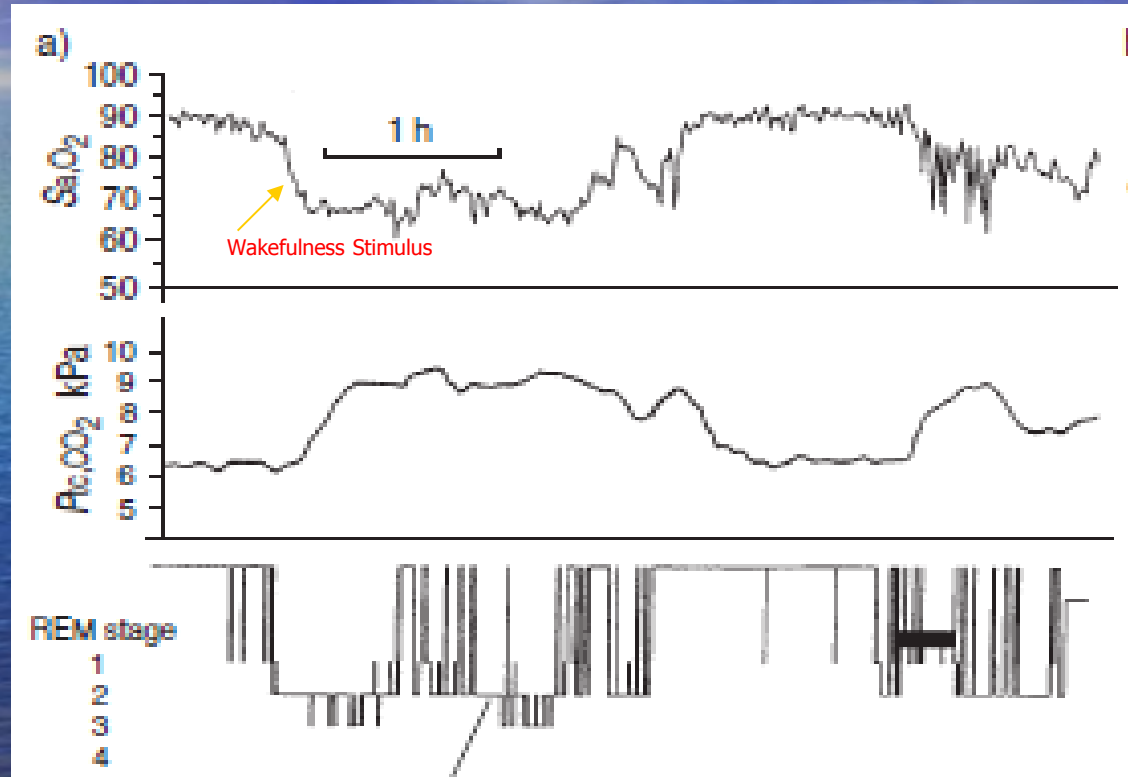


B.Langevin CH Alès

CPLF Janvier 2017

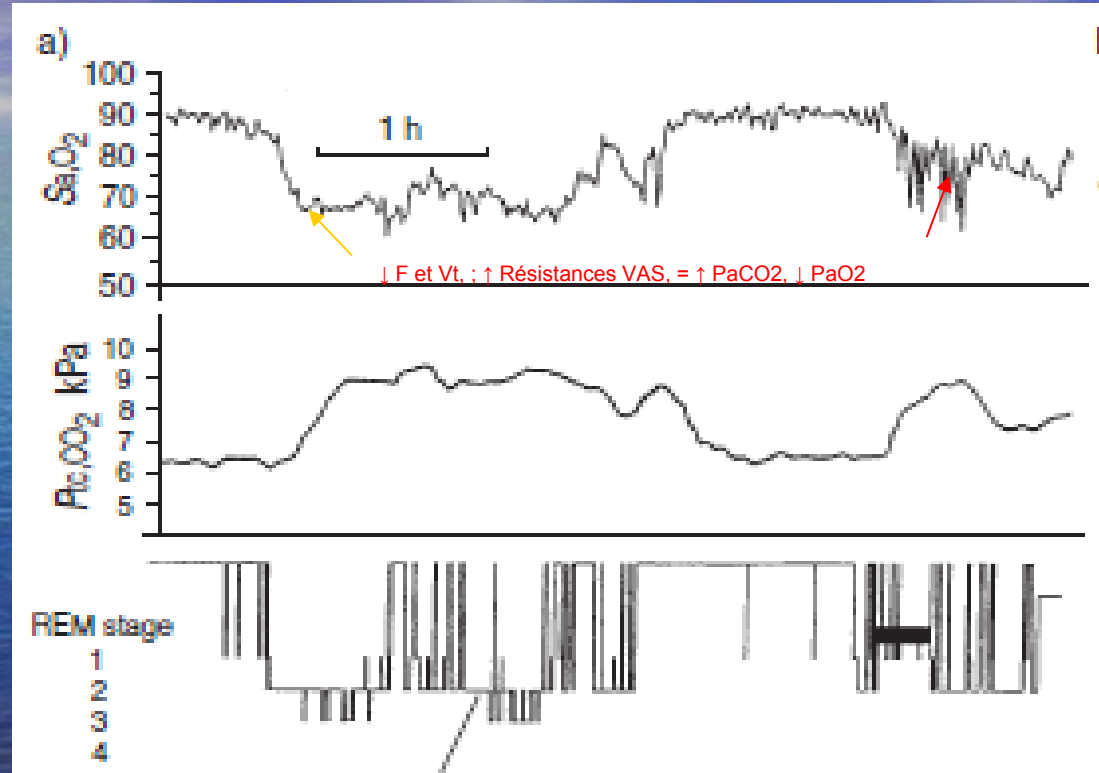
# Sommeil et Ventilation

## Mécanismes de l'HypoVentilation Nocturne



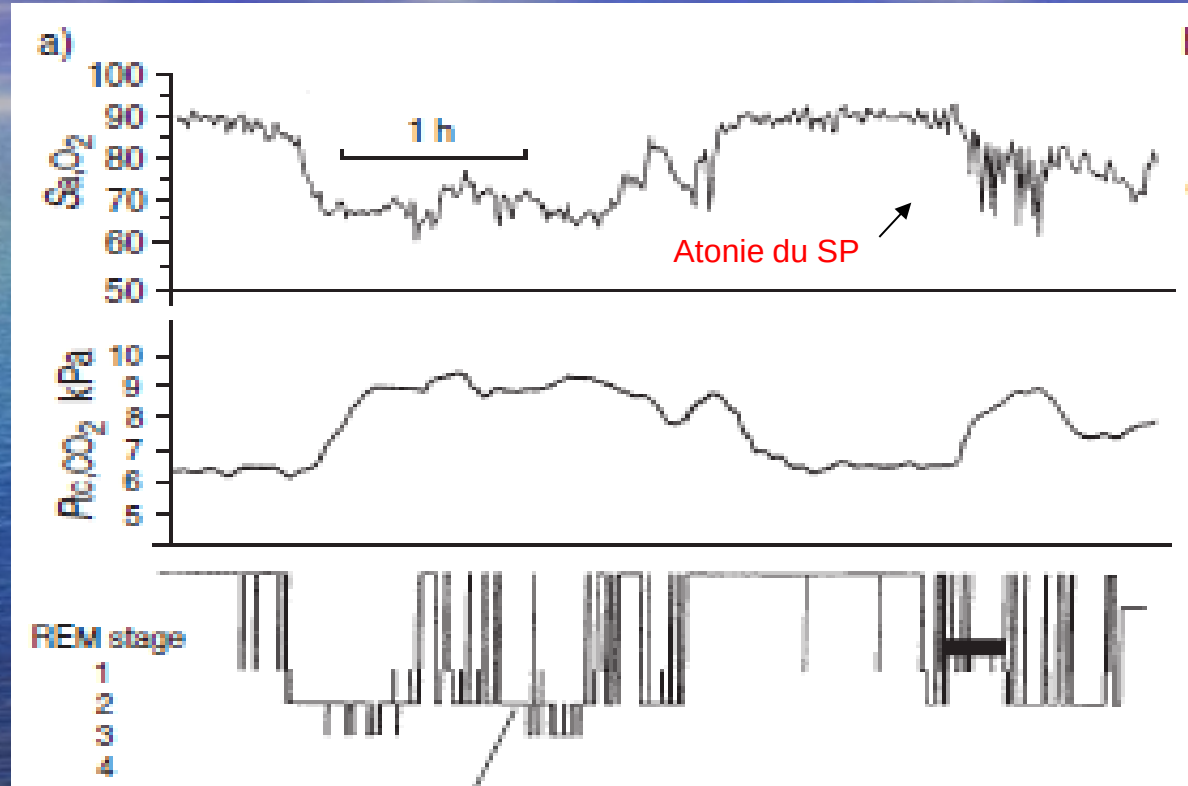
# Sommeil et Ventilation

## Mécanismes de l'Hypoventilation nocturne



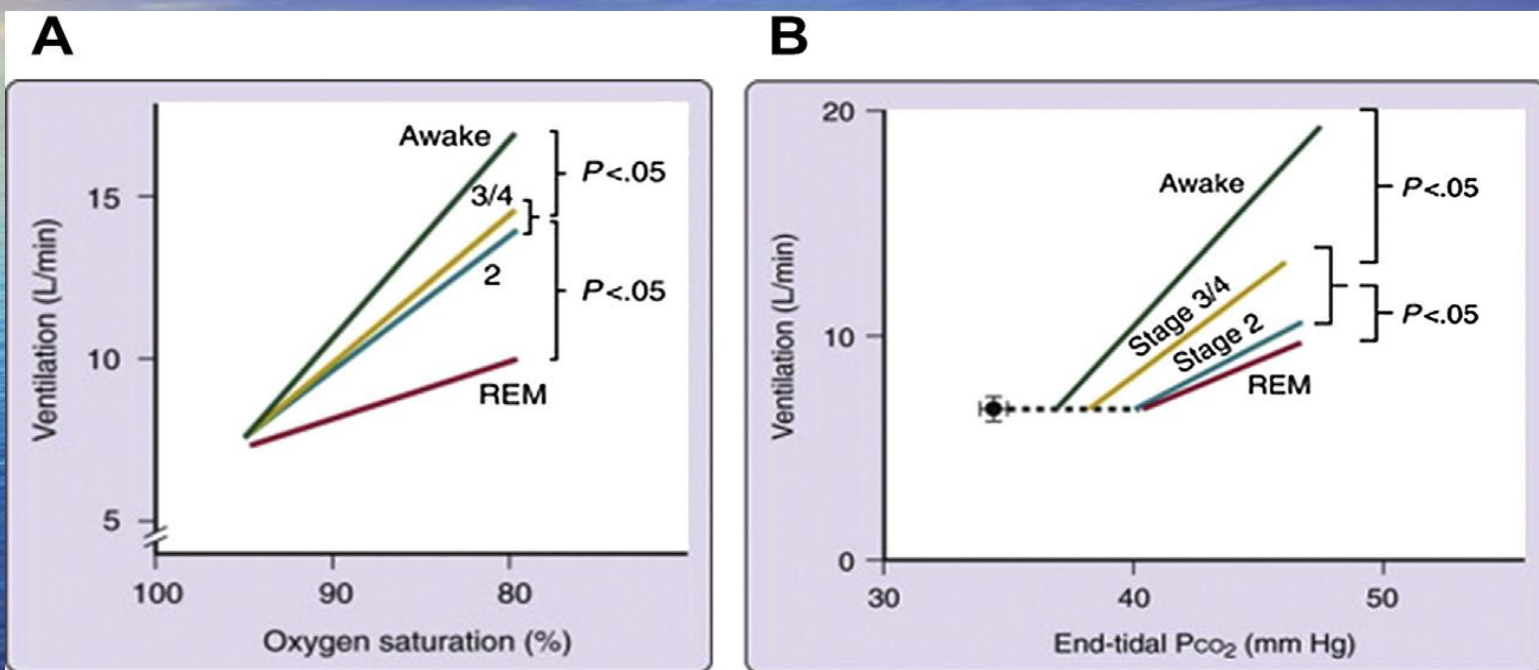
# Sommeil et Ventilation

Mécanismes de l'Hypoventilation nocturne



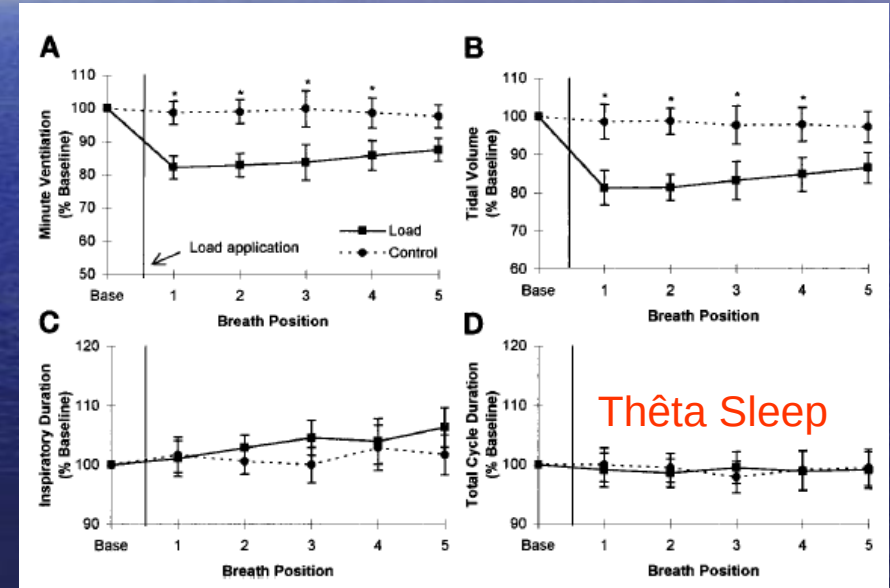
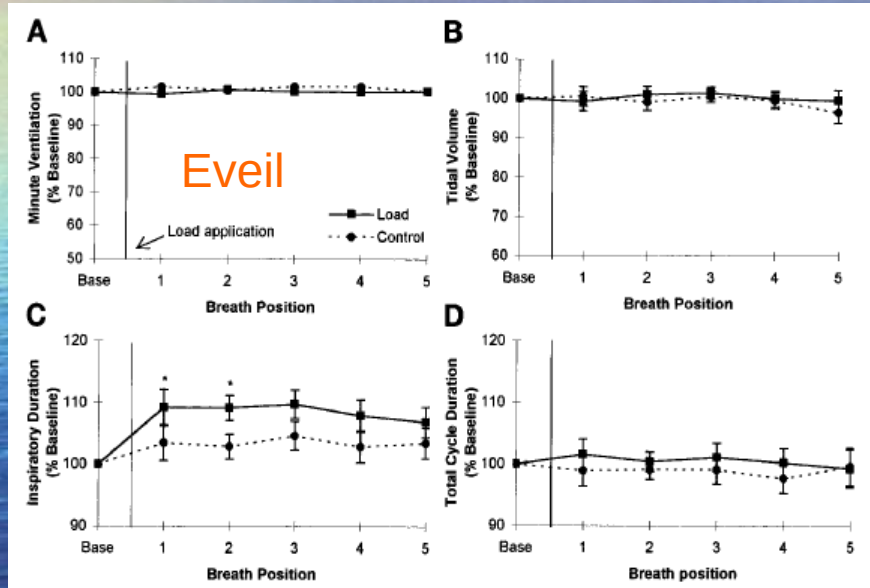
# Sommeil et Contrôle de la Ventilation

## Mécanismes de l'Hypoventilation Nocturne



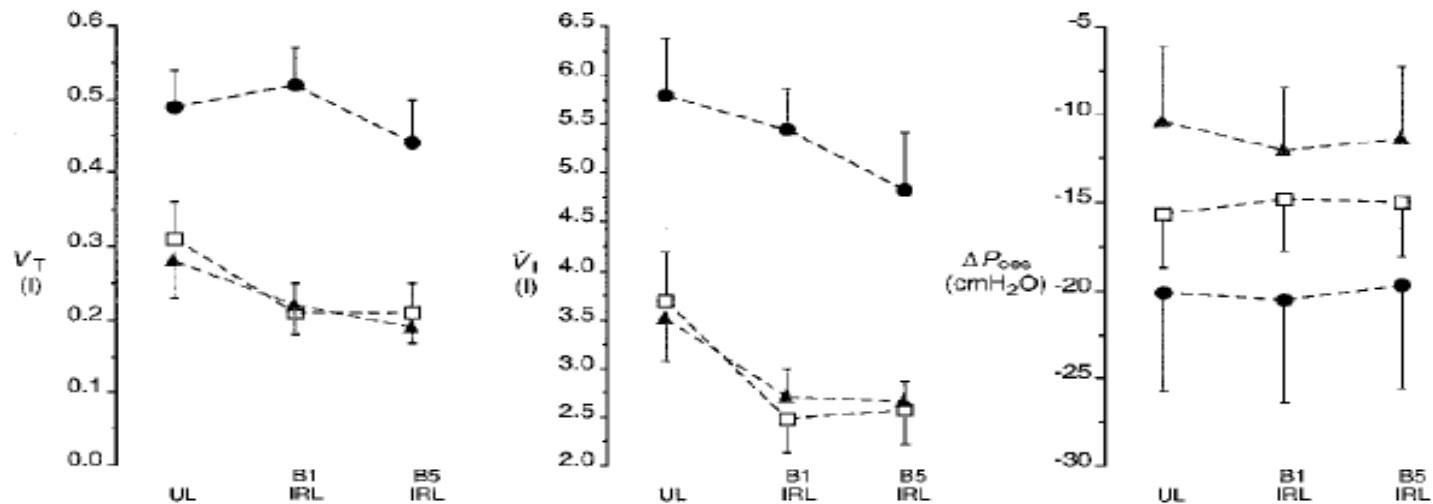
# Sommeil et Contrôle de la Ventilation

## Mécanismes de l'Hypoventilation Nocturne « Load Compensation »



# Sommeil et Contrôle de la Ventilation

## Mécanismes de l'Hypoventilation Nocturne « Load Compensation »



**Figure 1. State-related changes in respiratory load compensation**

Group mean ( $\pm$  s.e.m.) results for unloaded breathing (UL, mean of 5 breaths) and for the first (B1 IRL) and the fifth (B5 IRL) breaths following application of a 12 cmH<sub>2</sub>O inspiratory resistive load during wakefulness (●), NREM sleep (□) and REM sleep (▲).

*Journal of Physiology* (2000), 526.1, pp.195–202

The respiratory response to inspiratory resistive loading during rapid eye movement sleep in humans

Mary J. Morrell\*, Helen A. K. Browne and Lewis Adams

CPLF Janvier 2017

# Les Systèmes de Contrôle

## Facteurs d'Instabilité de la Commande Ventilatoire

- La Menace Représentée par les Altérations des Réponses Adaptatives fait du Sommeil une « Décompensation » Respiratoire.
- Le Facteur Musculaire du SP Réalise une « Myopathie Aiguë »
- Selon Phillipson il faut Sortir de ce système pour s'adapter

# Sommeil et Contrôle de la Ventilation

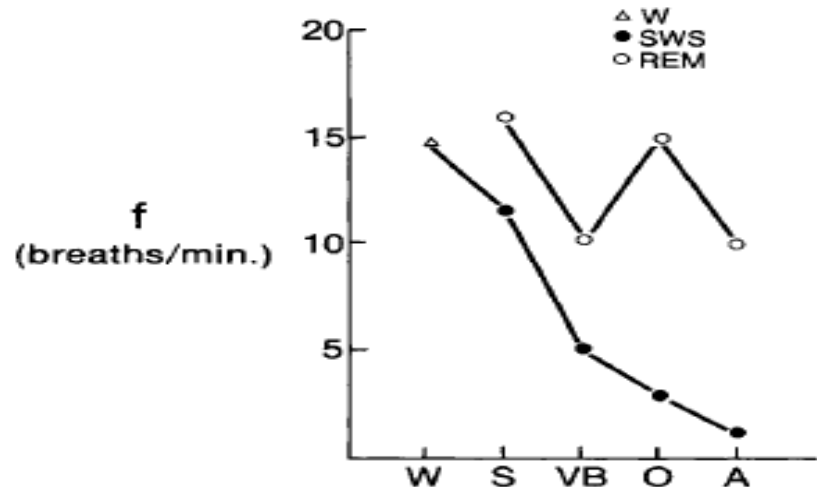


Fig. 3. Cumulative effect on respiratory frequency ( $f$ ) of decreases in afferent respiratory stimuli during slow-wave sleep (●) and rapid-eye-movement sleep (○). W = awake; S = asleep; VB = asleep plus vagal blockade; O = asleep plus vagal blockade plus hyperoxia; A = asleep plus vagal blockade plus hyperoxia plus metabolic alkalosis [modified from Sullivan and co-workers (61); reprinted by permission from the *Journal of Applied Physiology*, 1978, 45, 11.

## State of the Art

### Control of Breathing during Sleep<sup>1,2</sup>

ELIOT A. PHILLIPSON

AMERICAN REVIEW OF RESPIRATORY DISEASE, VOLUME 118, 1978

# Les Systèmes de Contrôle

ELIOT A. PHILLIPSON

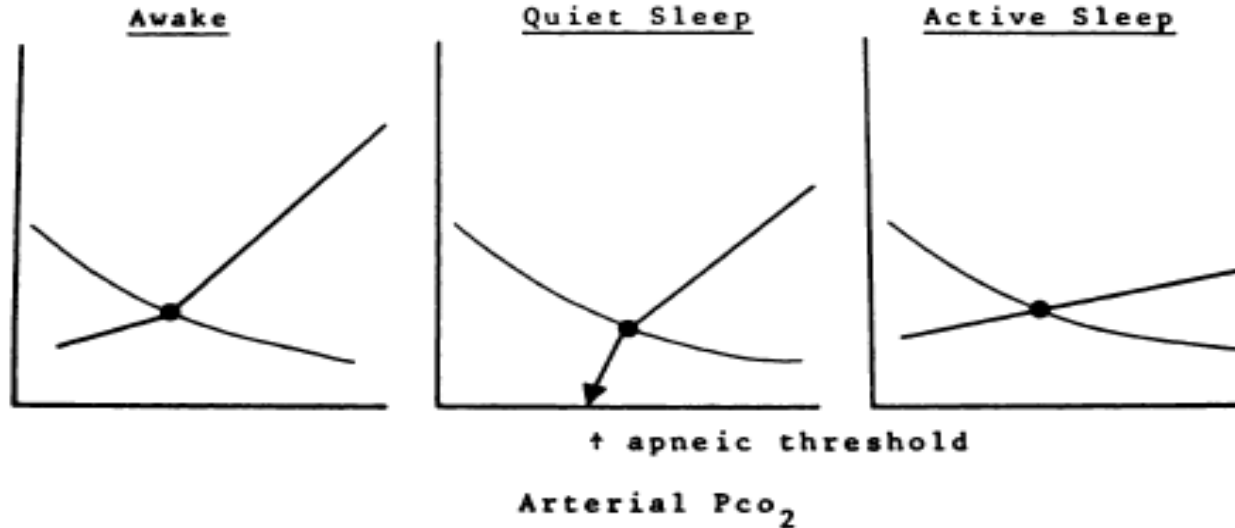
TABLE 1

## SUMMARY OF RESPIRATORY CONTROL MECHANISMS DURING SLEEP

|                                 | Awake              |                        | NonREM Sleep       |             | REM Sleep           |                               |
|---------------------------------|--------------------|------------------------|--------------------|-------------|---------------------|-------------------------------|
|                                 | Inactive           | Active                 | Stages 1, 2        | Stages 3, 4 | Tonic               | Phasic                        |
| Dominant influence on breathing | State<br>Metabolic | Behavioral             | Metabolic<br>State | Metabolic   | State<br>Metabolic  | Nonmetabolic<br>(Behavioral?) |
| Pattern of breathing            | Regular            | Irregular              | Periodic           | Regular     | Regular             | Irregular                     |
| Response to metabolic stimuli   | Present            | Decreased<br>or absent | Present            | Present     | Probably<br>present | Decreased<br>or absent        |

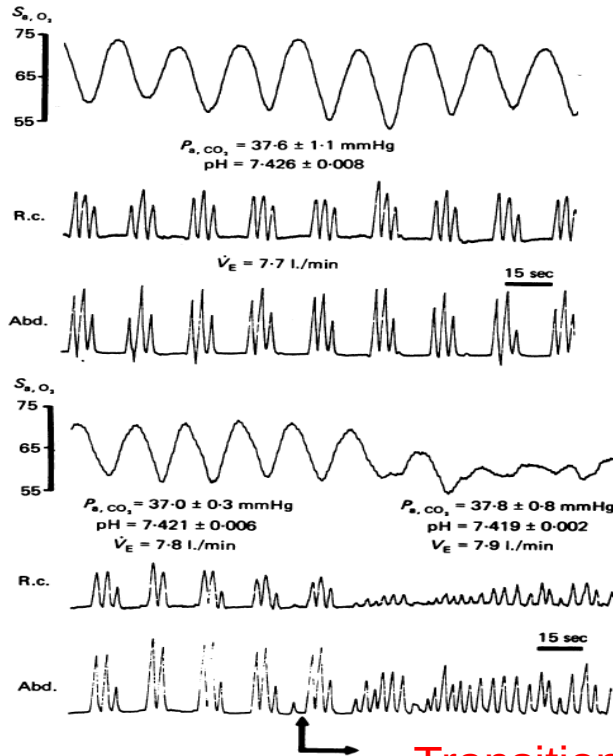
# Les Systèmes de Contrôle

## Ventilatory Responses to $\text{CO}_2$ : Normoxia



# Sommeil et Contrôle de la Ventilation

## PERIODIC BREATHING DURING HYPOXIC SLEEP



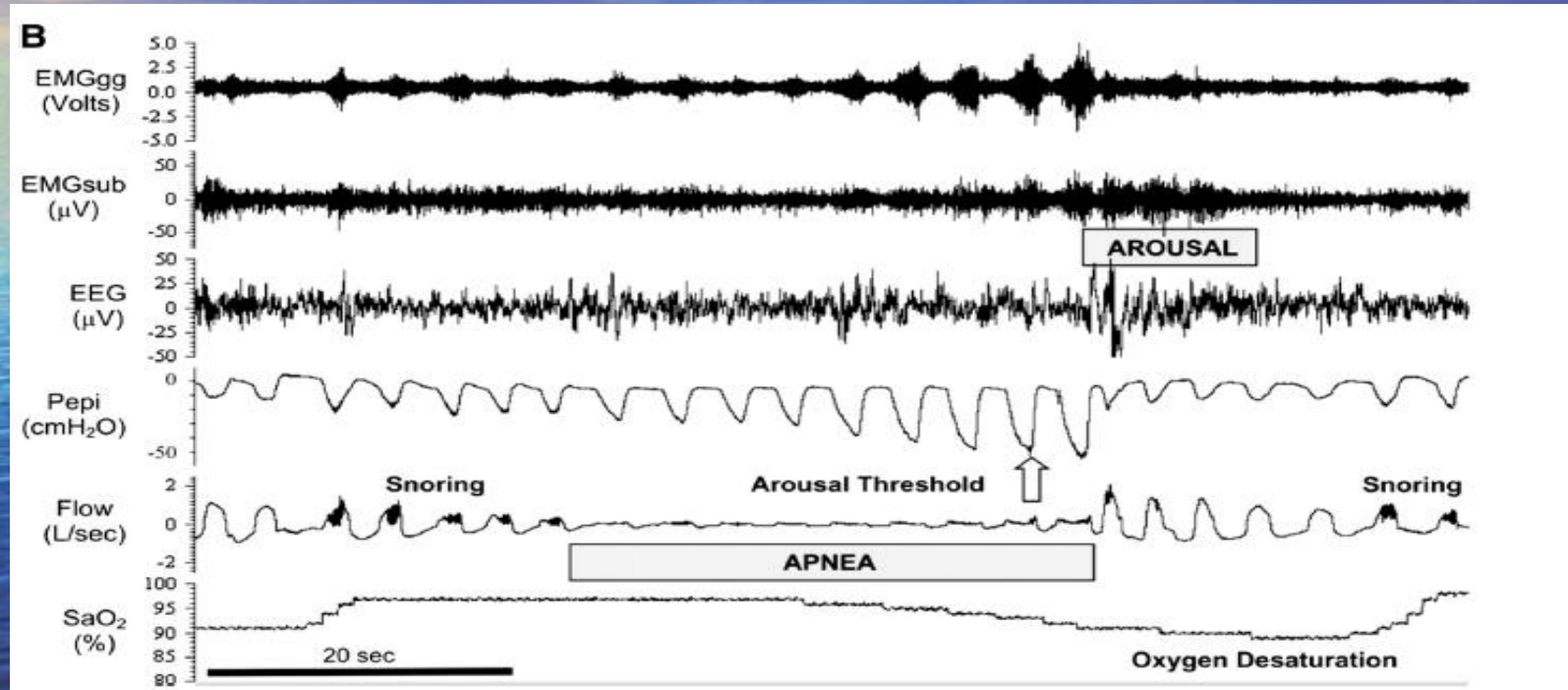
*J. Physiol.* (1983), **343**, pp. 507-524  
With 6 text-figures  
Printed in Great Britain

507

## MECHANISMS OF HYPOXIA-INDUCED PERIODIC BREATHING DURING SLEEP IN HUMANS

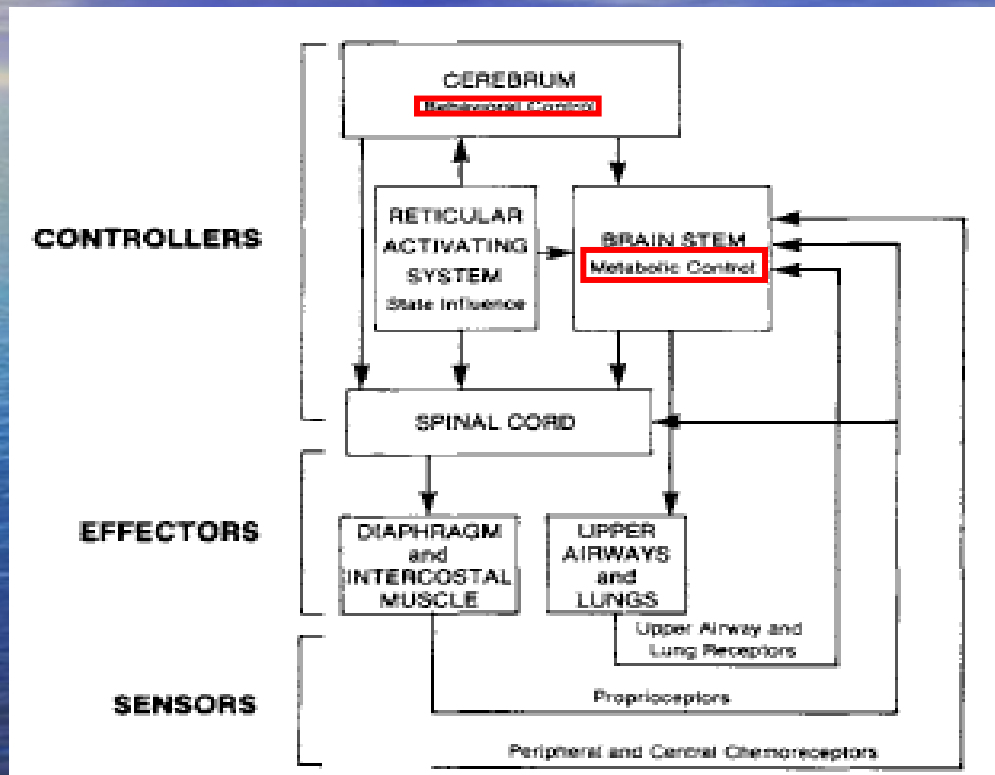
By ANNE BERSSENBRUGGE\*, JEROME DEMPSEY\*, CONRAD IBER†, JAMES SKATRUD† and PAMELA WILSON†

# Le Micro Eveil: Adaptation Ultime



Eckert DJ and Malhotra A. 2008. Pathophysiology of adult obstructive sleep apnea. *Proc Am Thorac Soc* 5: 144–153. Official Journal of the American Thoracic Society.

# Les Systèmes de Contrôle



State of the Art

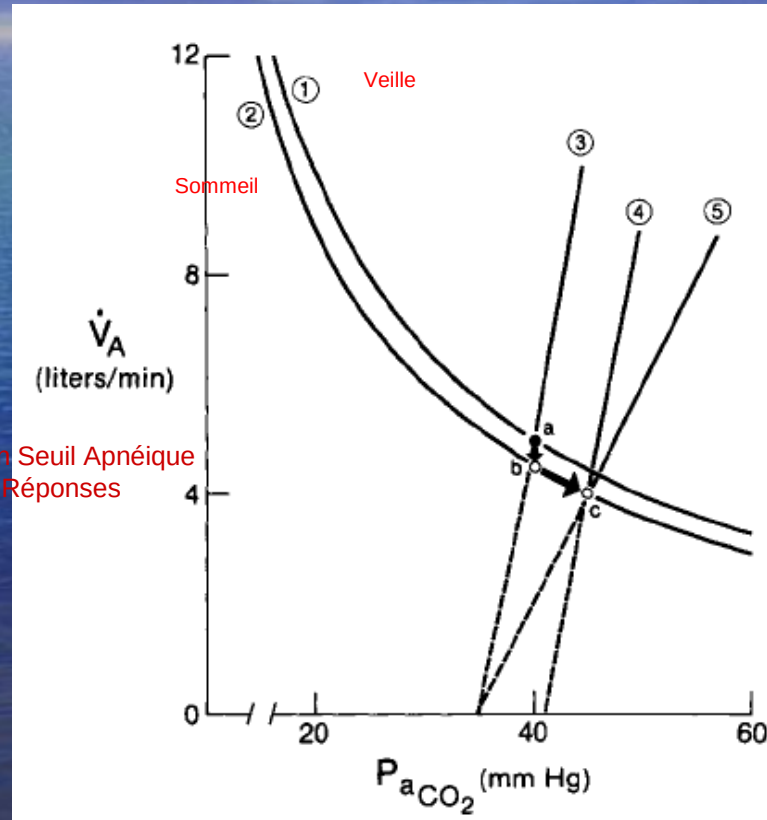
Control of Breathing during Sleep<sup>1, 2</sup>

ELIOT A. PHILLIPSON

AMERICAN REVIEW OF RESPIRATORY DISEASE, VOLUME 118, 1978

# Les Systèmes de Contrôle

## Contrôle Métabolique et Ventilation Périodique



- 3: Réponses CO<sub>2</sub> Veille
- 4: Réponses CO<sub>2</sub> Sommeil /modification Seuil Apnéique
- 5: Réponses CO<sub>2</sub> Sommeil/ Diminution Réponses

State of the Art

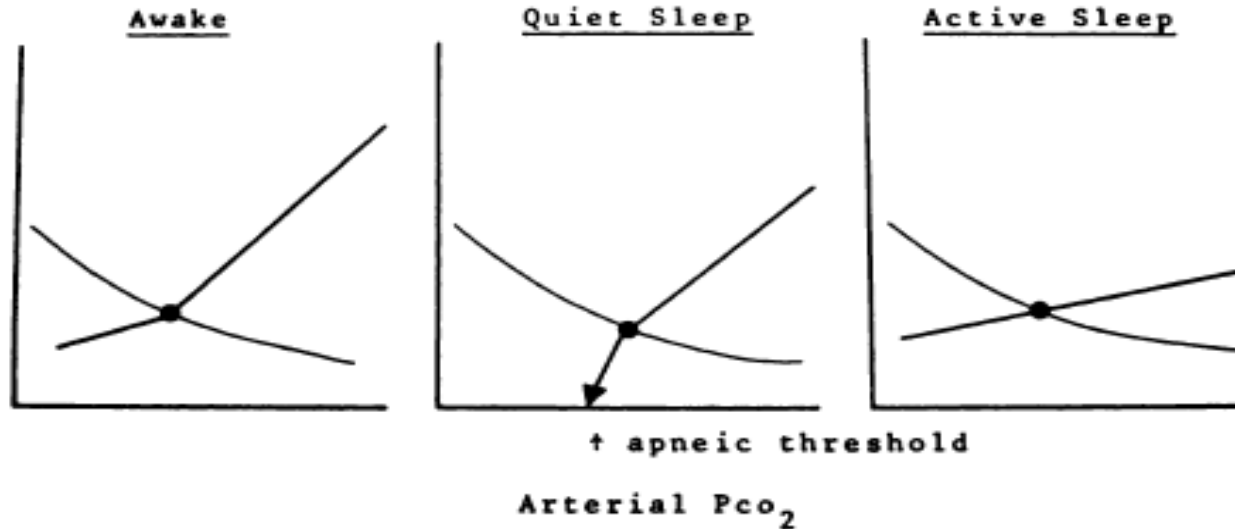
Control of Breathing during Sleep<sup>1, 2</sup>

ELIOT A. PHILLIPSON

AMERICAN REVIEW OF RESPIRATORY DISEASE, VOLUME 118, 1978

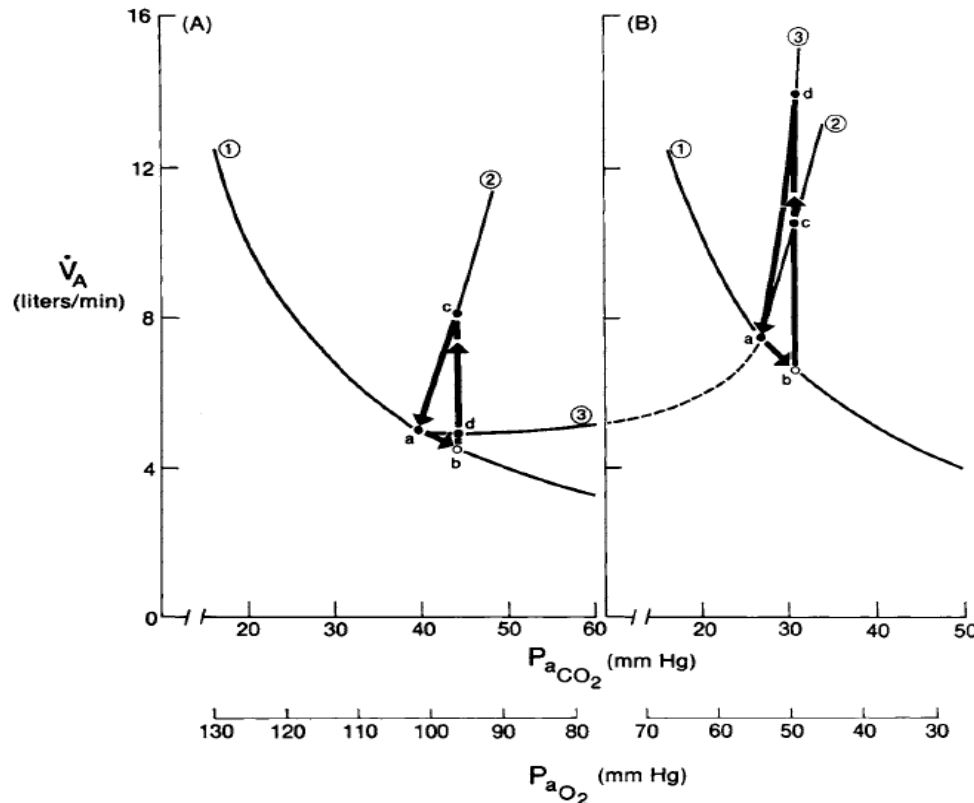
# Les Systèmes de Contrôle

## Ventilatory Responses to $\text{CO}_2$ : Normoxia



# Les Systèmes de Contrôle

## Contrôle Métabolique et Ventilation Périodique



**State of the Art**

**Control of Breathing during Sleep<sup>1, 2</sup>**

**ELIOT A. PHILLIPSON**

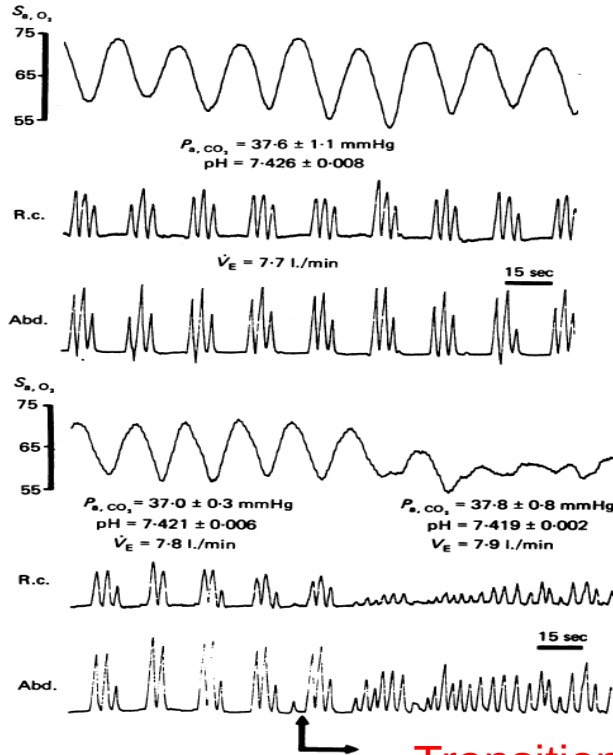
AMERICAN REVIEW OF RESPIRATORY DISEASE, VOLUME 118, 1978

En Condition de Normoxie : boucle abdca

En Condition d'Hypoxie : boucle abcdca

# Le Sommeil: Facteur d'Instabilité de la Ventilation

## PERIODIC BREATHING DURING HYPOXIC SLEEP



*J. Physiol.* (1983), **343**, pp. 507–524  
With 6 text-figures  
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## MECHANISMS OF HYPOXIA-INDUCED PERIODIC BREATHING DURING SLEEP IN HUMANS

By ANNE BERSSENBRUGGE\*, JEROME DEMPSEY\*, CONRAD IBER†, JAMES SKATRUD† AND PAMELA WILSON†

Transition to REM

CPLF Janvier 2017

# Le Sommeil: Facteur d'Instabilité de la Ventilation

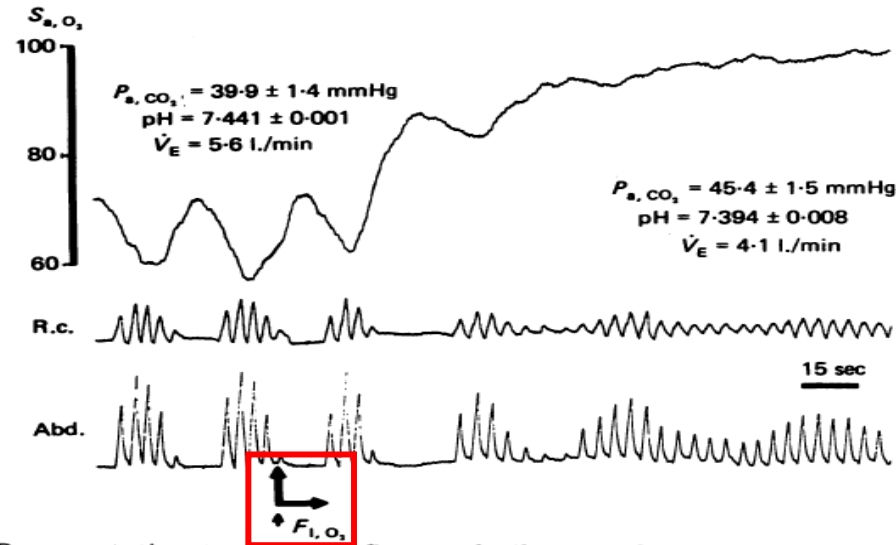
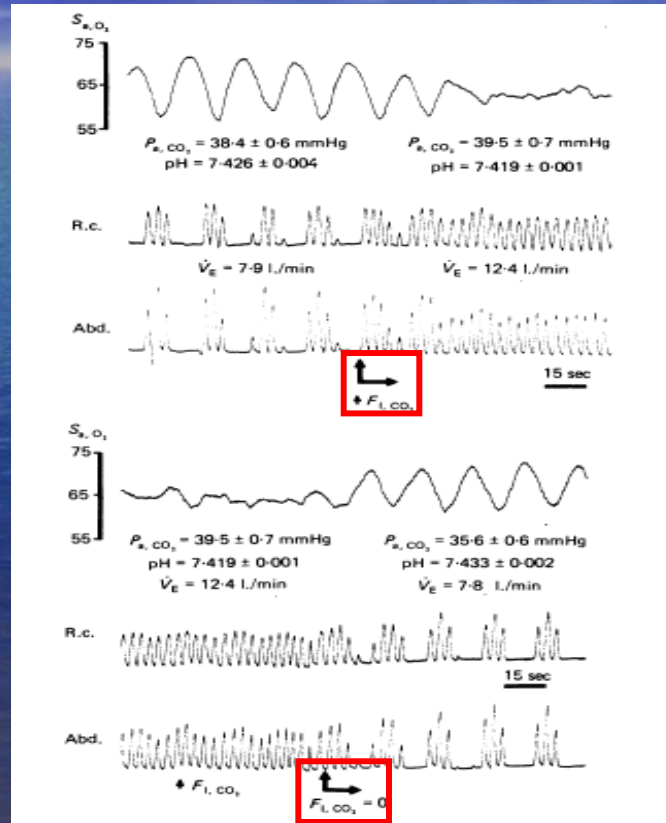
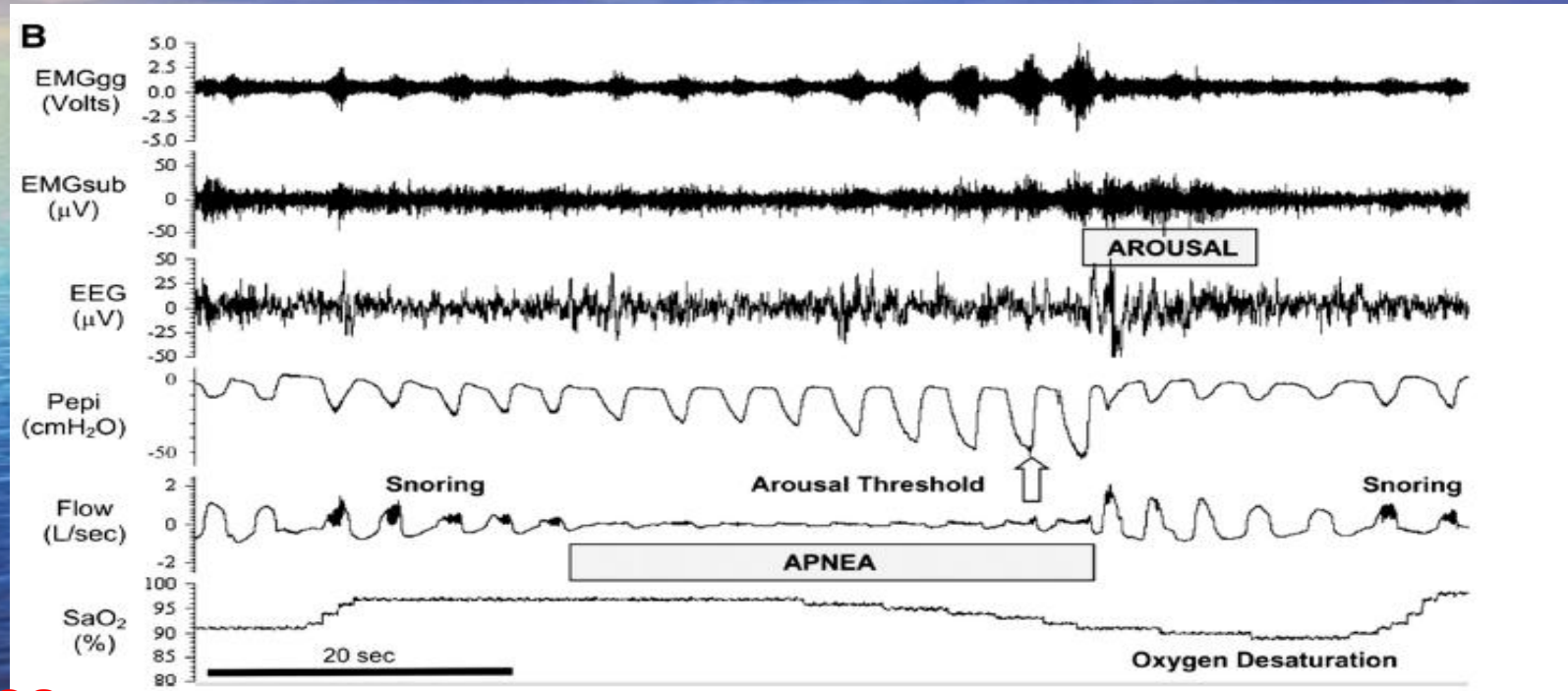


Fig. 3. Representative tracings of  $S_{a,O_2}$  and rib cage (R.c.) and abdominal (Abd.) movements during acute restoration of normoxia in hypoxic non-r.e.m. sleep.  $F_{I,O_2}$  was increased at arrow.

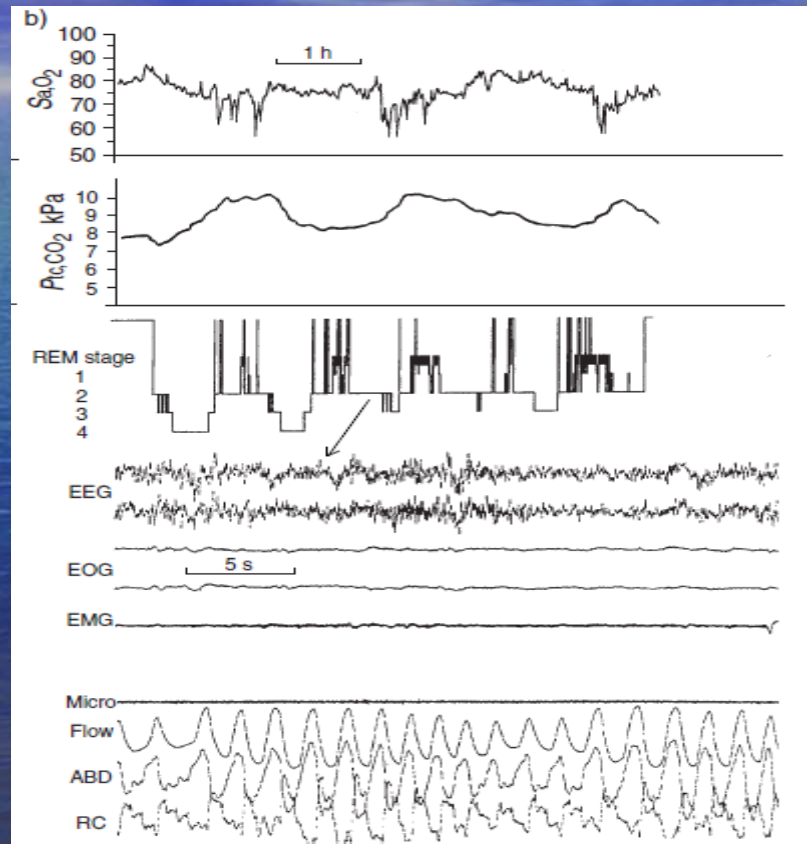
# Le Sommeil: Facteur d'Instabilité de la Ventilation



# Le Micro Eveil: Adaptation Ultime



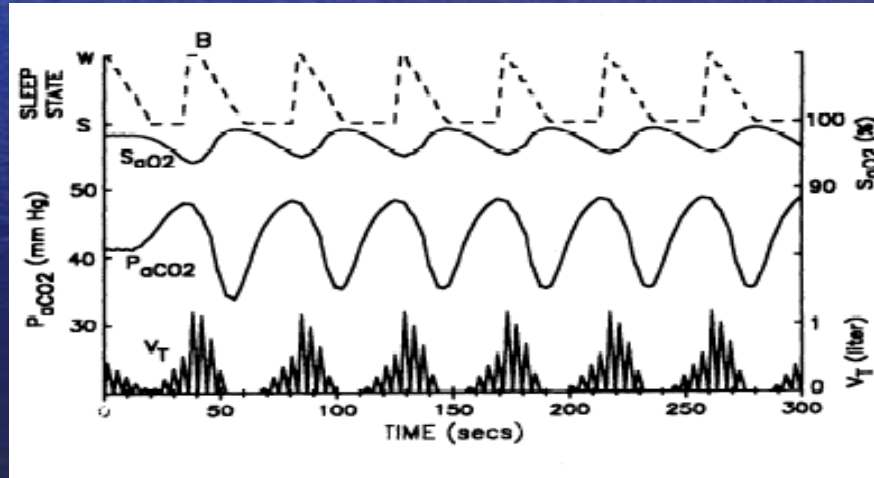
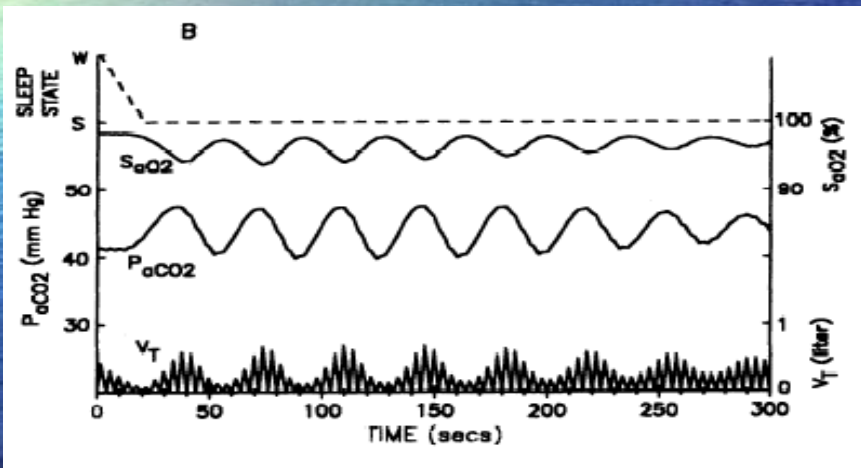
# Le Micro Eveil: Adaptation Ultime



IRC

# Le Micro Eveil: Facteur d'Instabilité du Sommeil et donc de la Ventilation

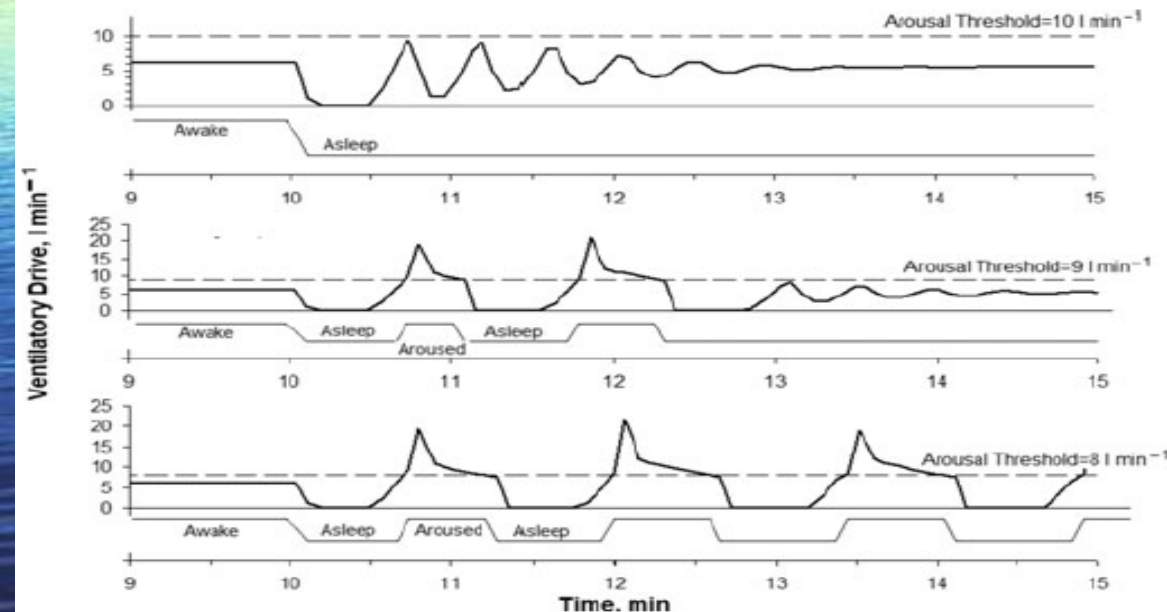
KHOO, MICHAEL C. K., ALLAN GOTTSCHALK, AND ALLAN I. PACK. *Sleep-induced periodic breathing and apnea: a theoretical study.* J. Appl. Physiol. 70(5): 2014-2024, 1991.



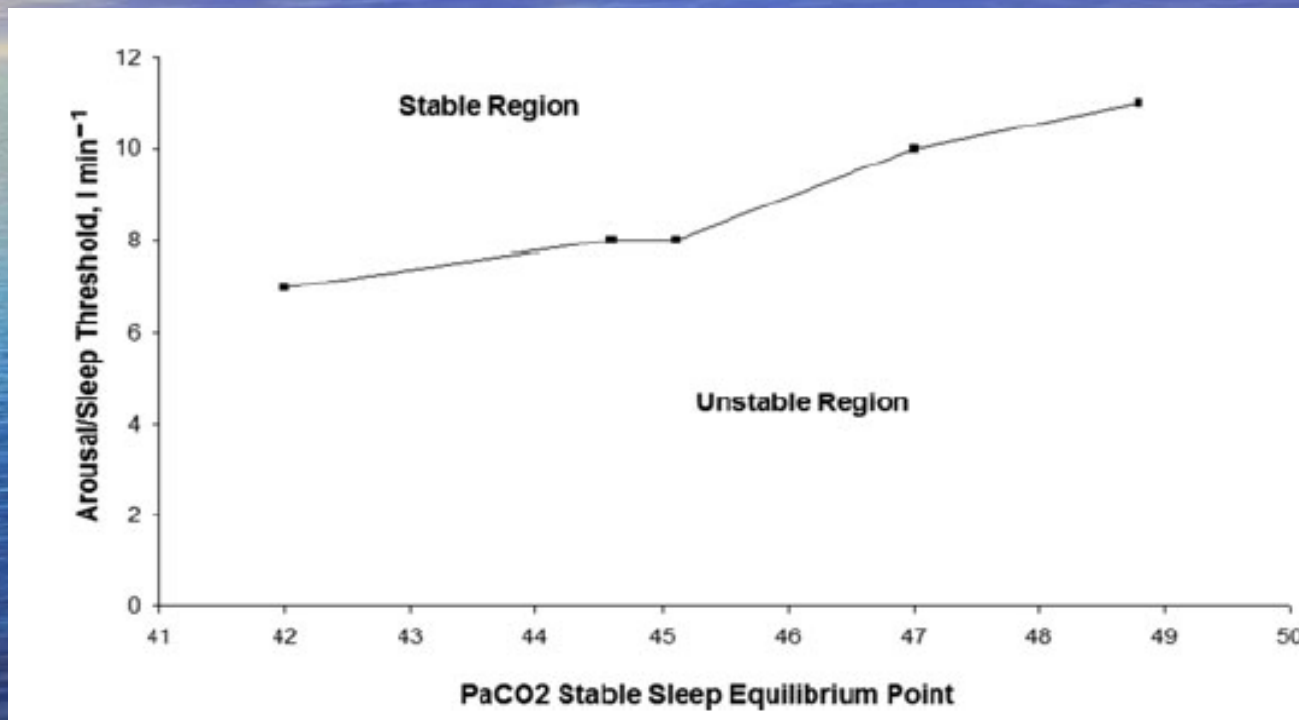
# Le Micro Eveil: Facteur d'Instabilité du Sommeil et donc de la Ventilation

**Influence of arousal threshold and depth of sleep on respiratory stability in man: analysis using a mathematical model**

G. S. Longobardo, C. J. Evangelisti and N. S. Cherniack



# Le Micro Eveil: Facteur d'Instabilité de la Ventilation en Fonction de la Ventilation

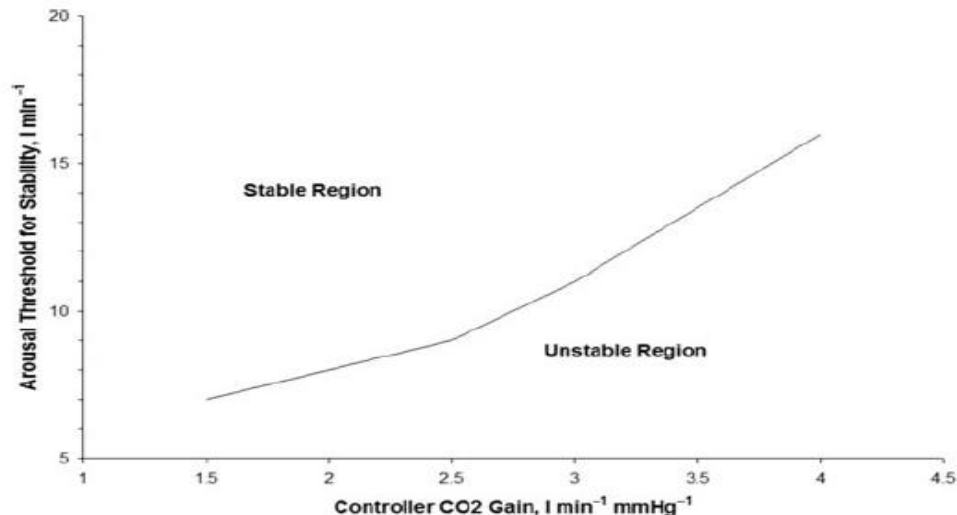


Influence of arousal threshold and depth of sleep on respiratory stability in man: analysis using a mathematical model

G. S. Longobardo, C. J. Evangelisti and N. S. Cherniack

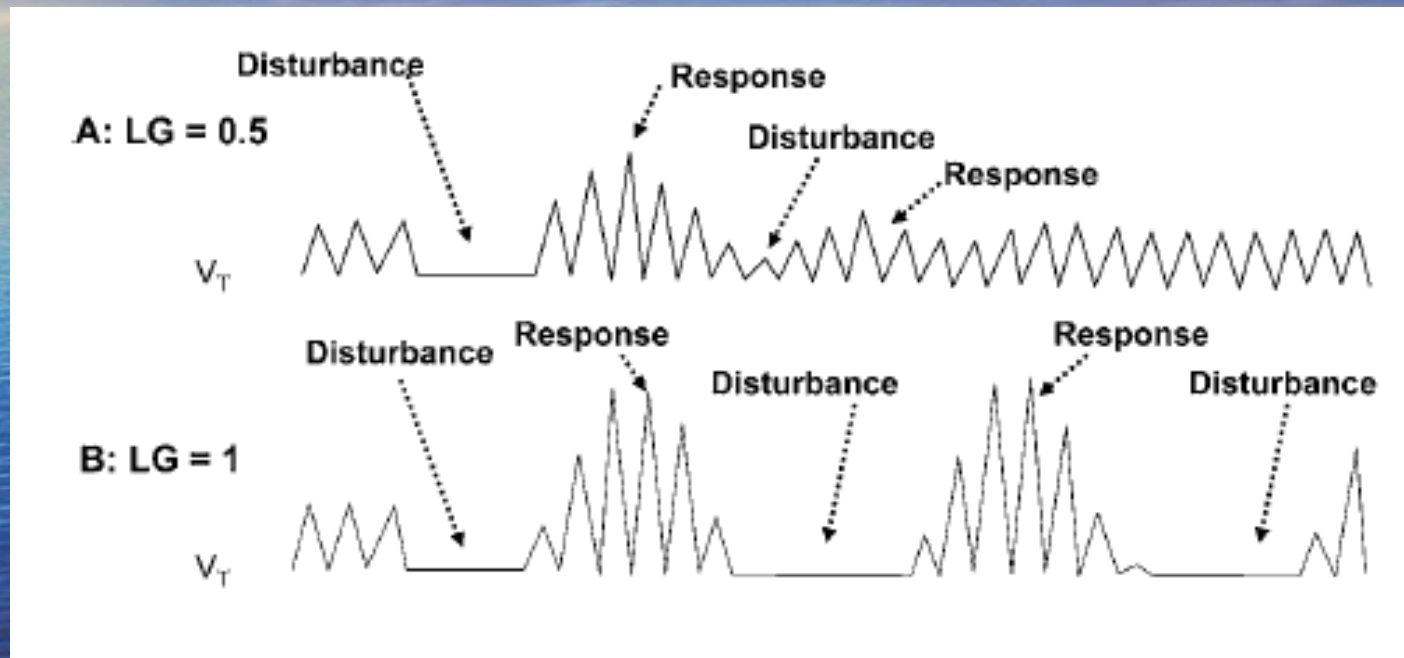
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# Le Micro Eveil: Facteur d'Instabilité de la Ventilation en Fonction de la Ventilation



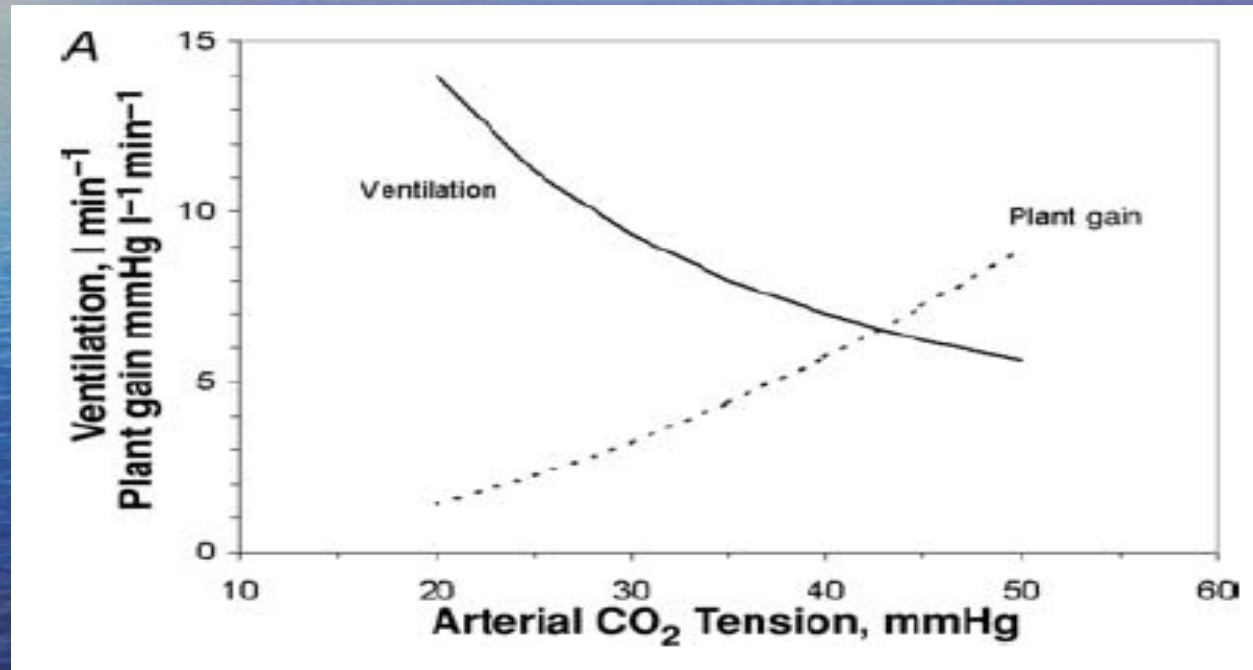
**Figure 2. The effect of arousal threshold and controller gain on stability for rigid upper airway muscles**  
Higher arousal thresholds are required for stability with higher controller gain because the ventilatory response at higher gains is greater. Lower arousal thresholds are thus more easily reached and can perpetuate cycling. In the stable region, there were some arousals before sleep equilibrium was achieved, the maximal number being 2. For controller gain of  $1 \text{ l min}^{-1} \text{ mmHg}^{-1}$ , an arousal threshold of  $5.8 \text{ l min}^{-1}$  results in sleep equilibrium. However, an arousal threshold of  $5.75 \text{ l min}^{-1}$  results in wakefulness because after the initial arousal, ventilatory drive never drops back to the sleep threshold. Sleep equilibrium conditions in these controller gain studies are  $P_{\text{aCO}_2}$  of  $44.6 \text{ mmHg}$  and  $P_{\text{aO}_2}$  of  $95.6 \text{ mmHg}$ , and ventilation of  $5.6 \text{ l min}^{-1}$ .

# Le Gain de la Réponse Ventilatoire: Facteur d'Instabilité de la Ventilation

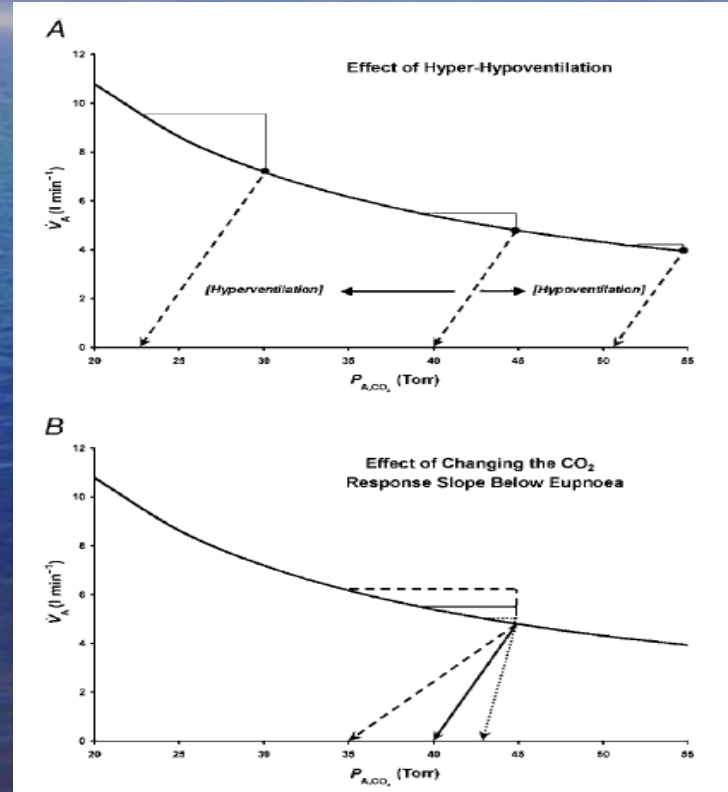


## Pathogenesis of Obstructive and Central Sleep Apnea

# Le Gain de la Réponse Ventilatoire: Facteur d'Instabilité de la Ventilation



# Le Gain de la Réponse Ventilatoire: Facteur d'Instabilité de la Ventilation



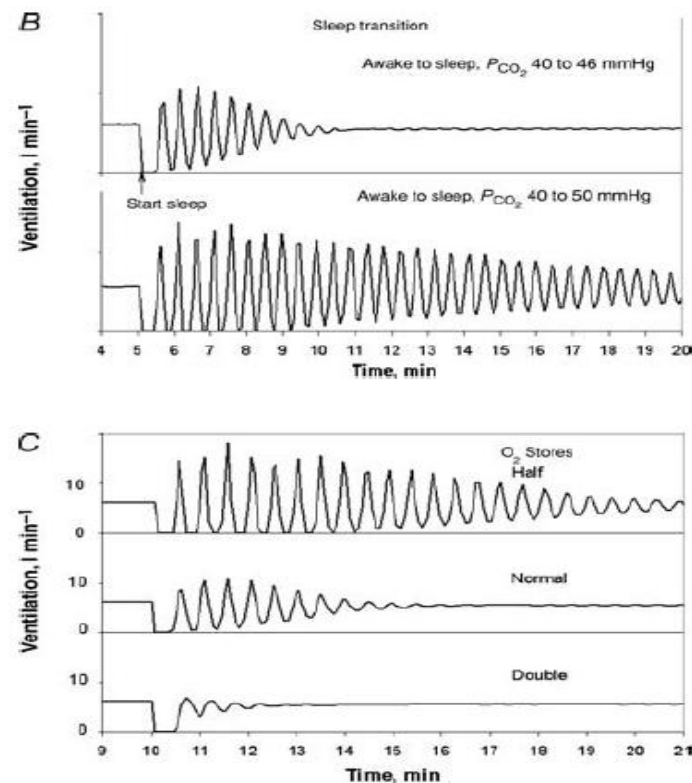
Plant Gain ou gain du système contrôlé

Contrôleur Gain, ou Gain de la réponse

# Le Gain de la Réponse Ventilatoire: Facteur d'Instabilité de la Ventilation

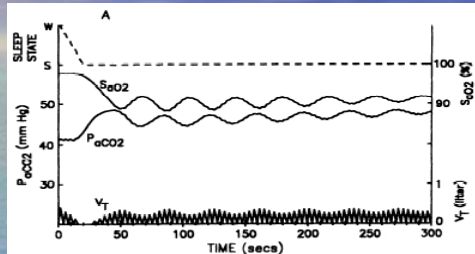
**Mathematical models of periodic breathing and their usefulness in understanding cardiovascular and respiratory disorders**

Neil S. Cherniack and Guy S. Longobardo

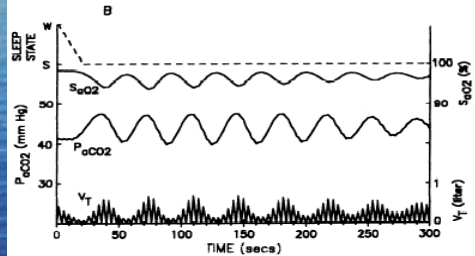


# Le Gain de la Réponse Ventilatoire: Facteur d'Instabilité de la Ventilation

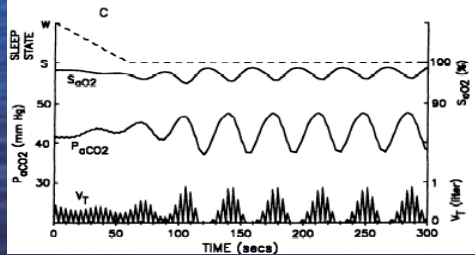
Low Gain



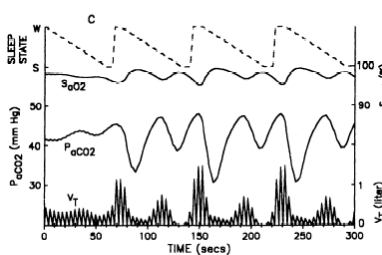
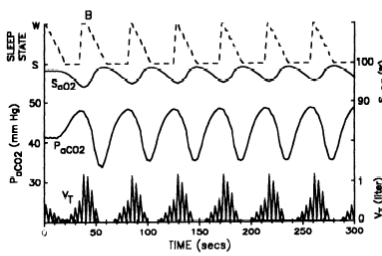
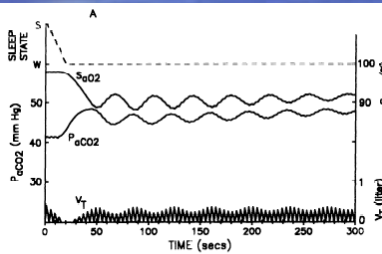
Moderate Gain I



High Gain



KHOO, MICHAEL C. K., ALLAN GOTTSCHALK, AND ALLAN I. PACK. *Sleep-induced periodic breathing and apnea: a theoretical study.* J. Appl. Physiol. 70(5): 2014-2024, 1991.



Moderate Gain +  
Instabilité du Sommeil

High Gain +  
Instabilité du Sommeil

# Le Gain de la Réponse Ventilatoire: Facteur d'Instabilité de la Ventilation

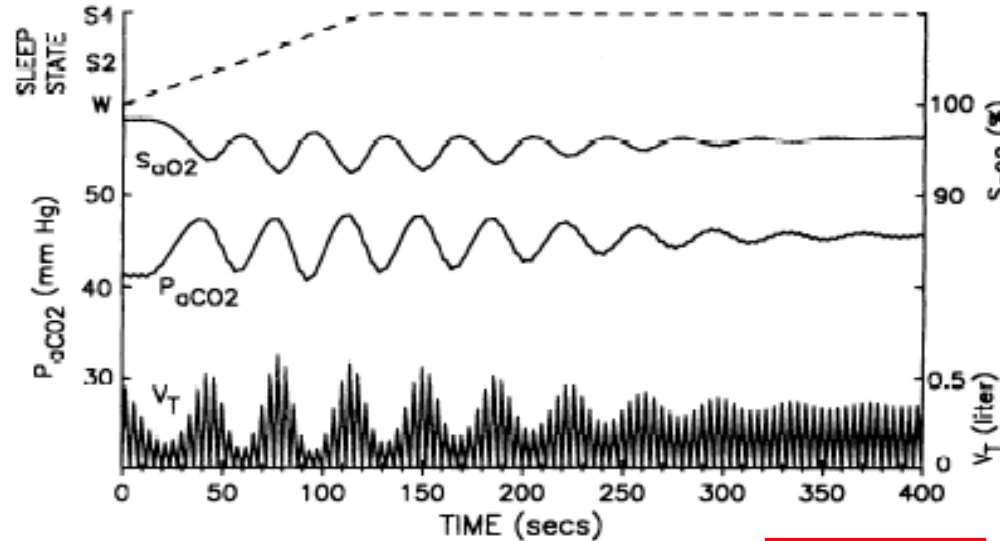
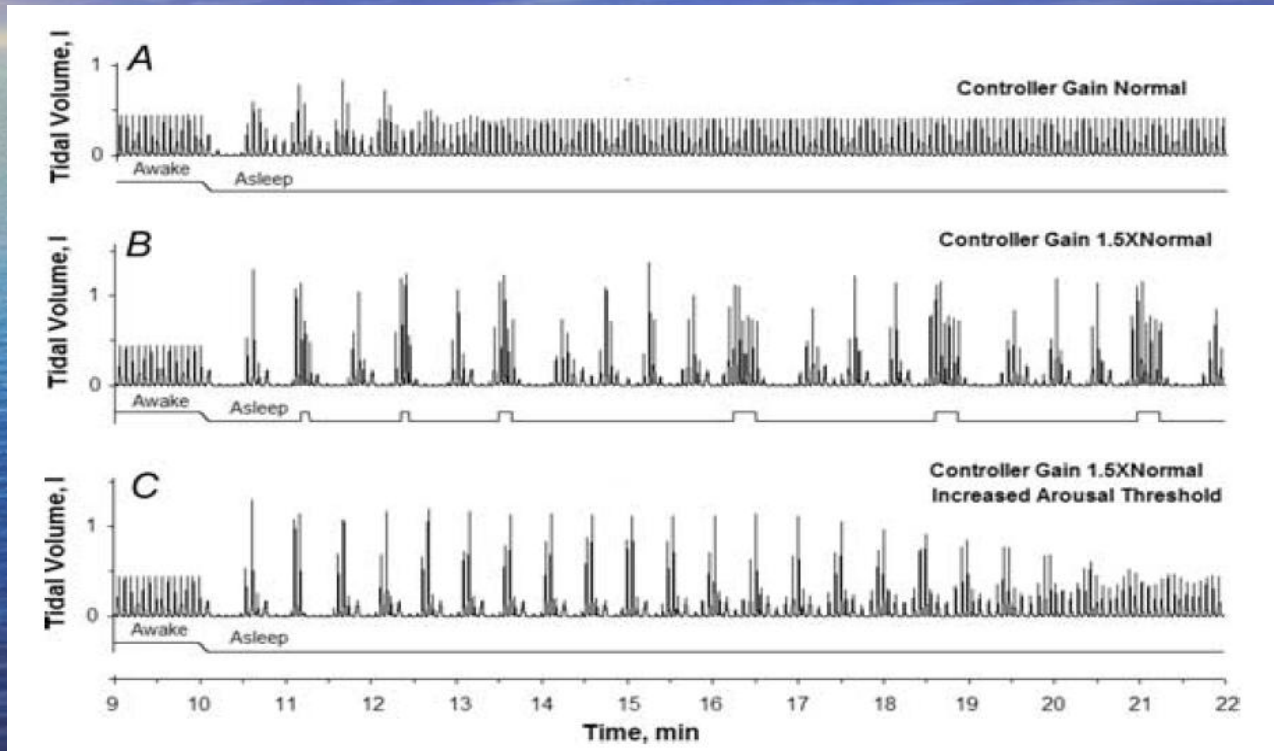


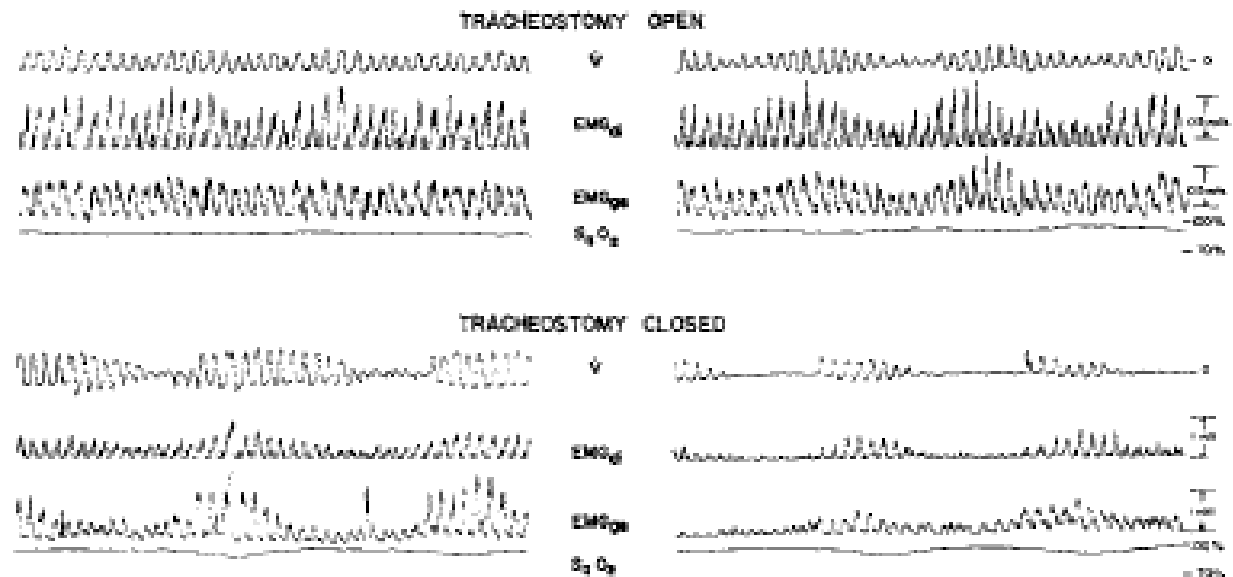
FIG. 10. Effect of transition from wakefulness to **stage 4 sleep** on model 1. Magnitude of wakefulness stimulus is chosen ( $S_0 = 30$  l/min) such that a sustained oscillation would result if progression of sleep state is terminated once stage 1 sleep has been attained. Progression to deeper sleep stages stabilizes model.

Seuil d'Eveil Bas  
Gain des Réponses Ventilatoires Bas  
« Pression de Sommeil » Elevée

# Le Gain de la Réponse Ventilatoire: Facteur d'Instabilité de la Ventilation



# Et Voilà Les Voies aériennes Supérieures (VAS) !

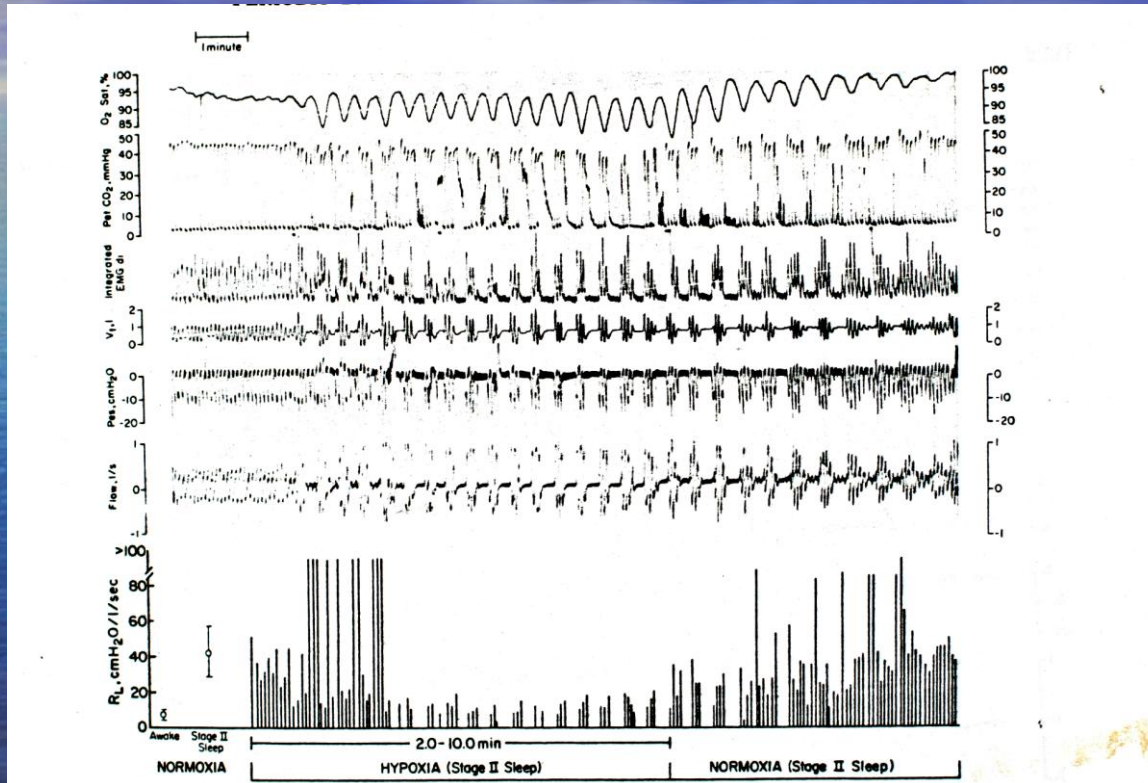


**Periodic Breathing and the Pathogenesis of Occlusive Sleep Apneas<sup>1-3</sup>**

ERGÜN ÖNAL and MELVIN LOPATA

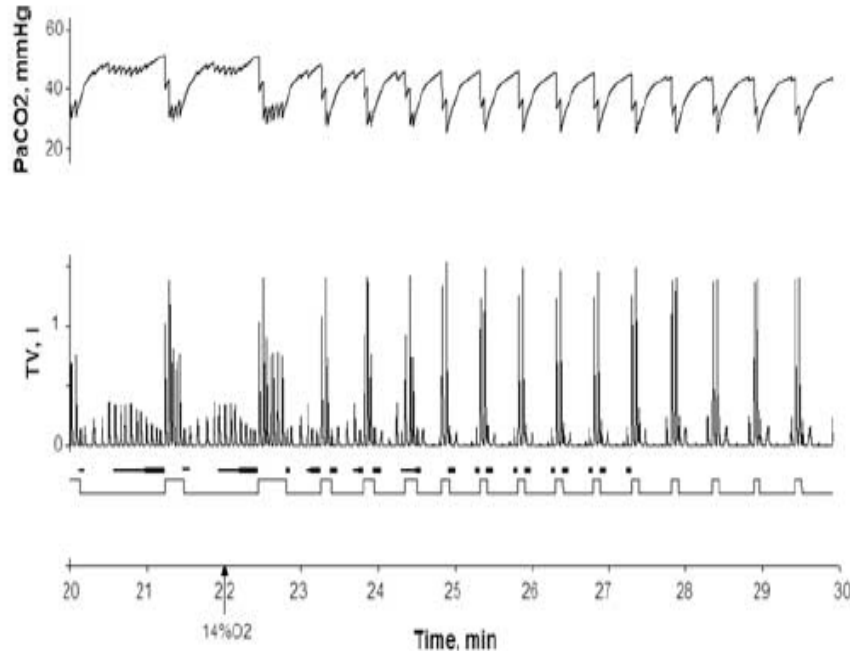
CPLF Janvier 2017

# Les Voies aériennes Supérieures (VAS)



Effect of hypoxia-induced periodic breathing on upper airway obstruction during sleep  
 G. Warner, J. B. Skatrud, J. A. Dempsey Journal of Applied Physiology Published 1 June 1987  
 Vol. 60, 6, 2201-2211

# Les Voies aériennes Supérieures (VAS)



**Figure 9. Inhalation of 14% O<sub>2</sub> (at the 22 min mark) after sleep onset converts obstructive apnoeas to central apnoeas**  
In the simulation, Elastance =  $0.175 + (0.304 \times \text{Ventilatory drive}) \text{ mmHg cm}^{-3}$ , and controller gain is normal. TV, tidal volume.

# Les Voies aériennes Supérieures

Guilleminault C, Quera-Salvá MA, Nino-Murcia G, Partinen M: Central sleep apnea and partial obstruction of the upper airway. *Ann Neurol* 21:465–469, 1987

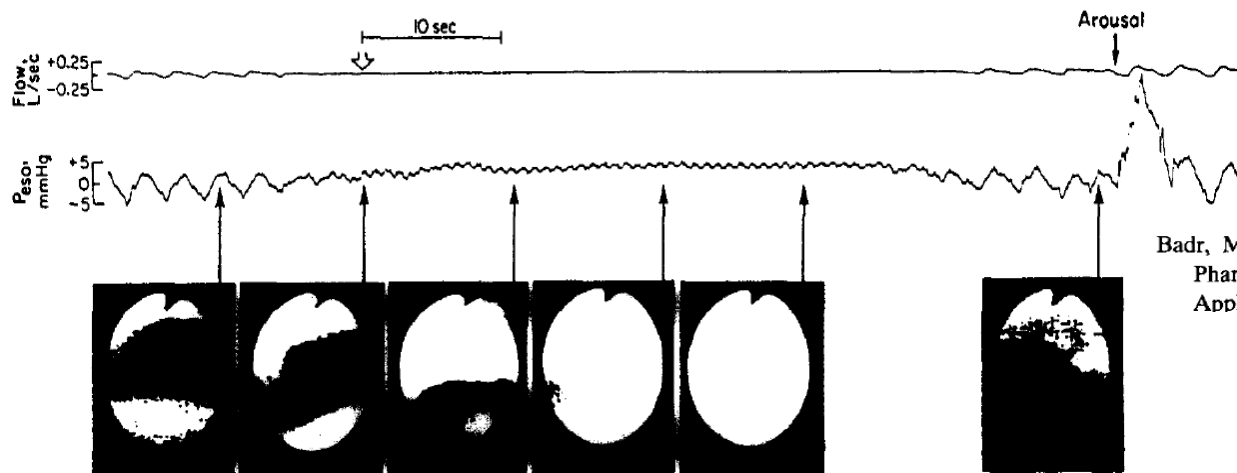


Fig. 5. Pharyngeal occlusion during central sleep apnea in a patient with central sleep apnea syndrome. Pes/esophageal pressure. Note the progressive reduction of the pharyngeal lumen during central apnea, culminating in complete occlusion with no evidence of intrathoracic pressure (4th image). Upper airway opening was associated with arousal. (From Badr et al., 1995, with permission.)

Effect of ventilatory drive on upper airway patency in humans during NREM sleep

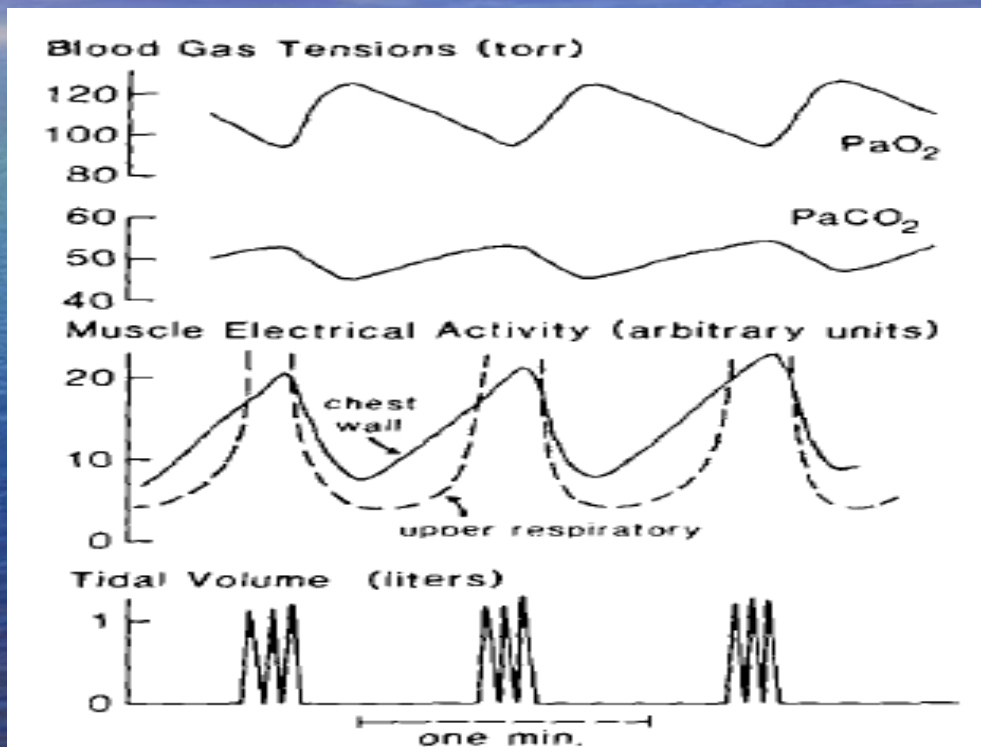
M. Safwan Badr \*

*Respiration Physiology* 103 (1996) 1–10

Badr, M.S., F. Toiber, J.B. Skatrud and J.A. Dempsey (1995). Pharyngeal narrowing/occlusion during central sleep apnea. *J. Appl. Physiol.* 78: 1806–1815.

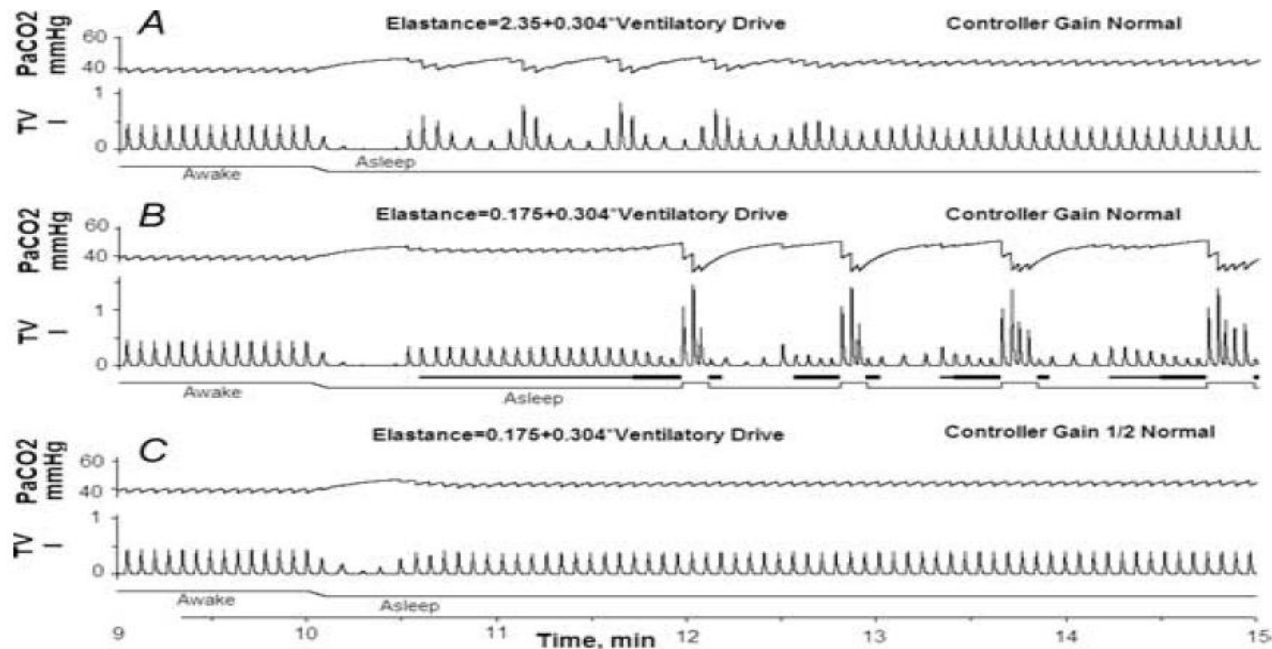
# Les Voies aériennes Supérieures (VAS)

## Modélisations des Boucles d'Instabilité



# Les Voies aériennes Supérieures (VAS)

## Modélisations des Boucles d'Instabilité

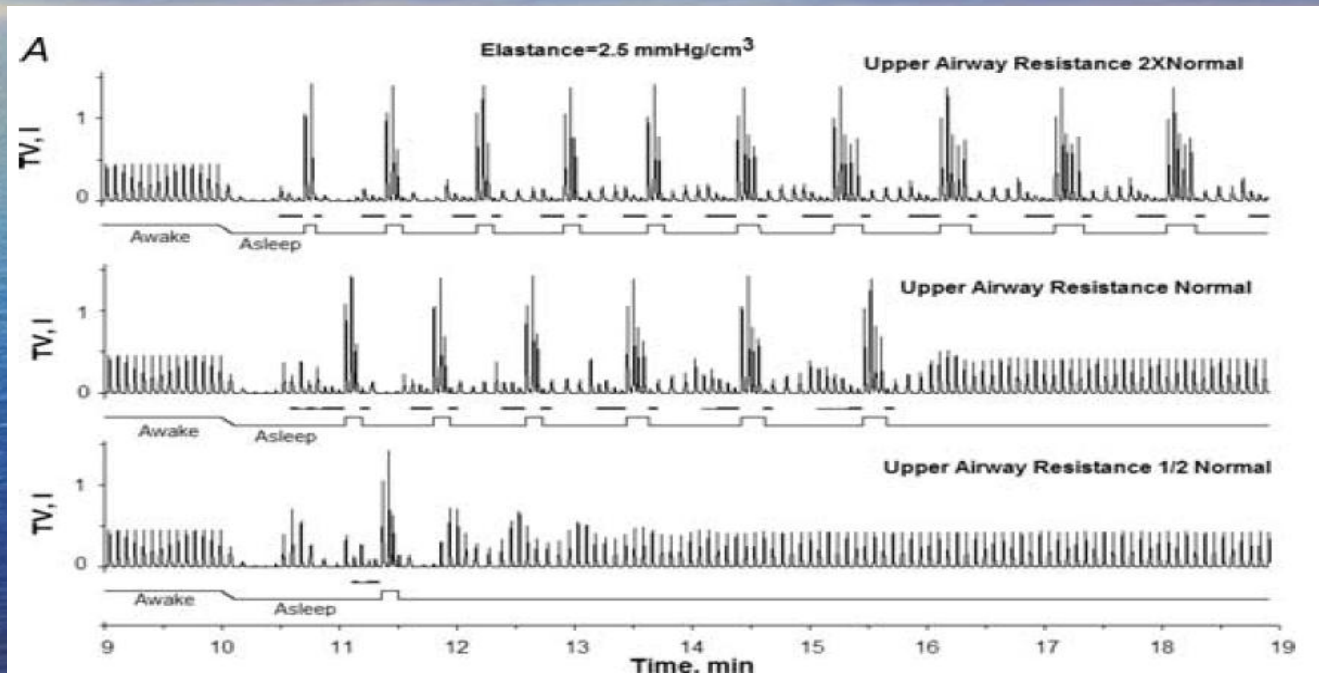


Analysis of the interplay between neurochemical control of respiration and upper airway mechanics producing upper airway obstruction during sleep in humans

G. S. Longobardo, C. J. Evangelisti and N. S. Cherniack *Exp Physiol* 93.2 pp 271–287

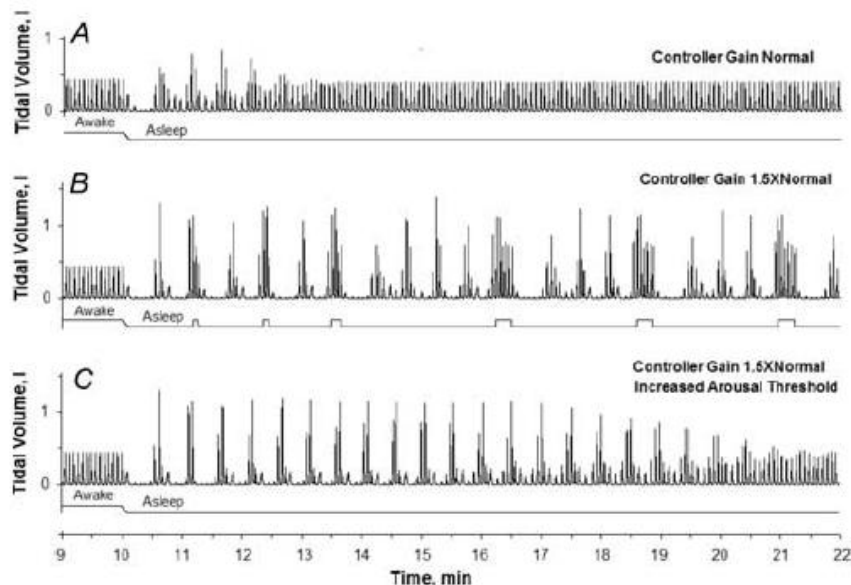
# Les Voies aériennes Supérieures (VAS)

## Modélisations des Boucles d'Instabilité



# Les Voies aériennes Supérieures (VAS)

## Modélisations des Boucles d'Instabilité

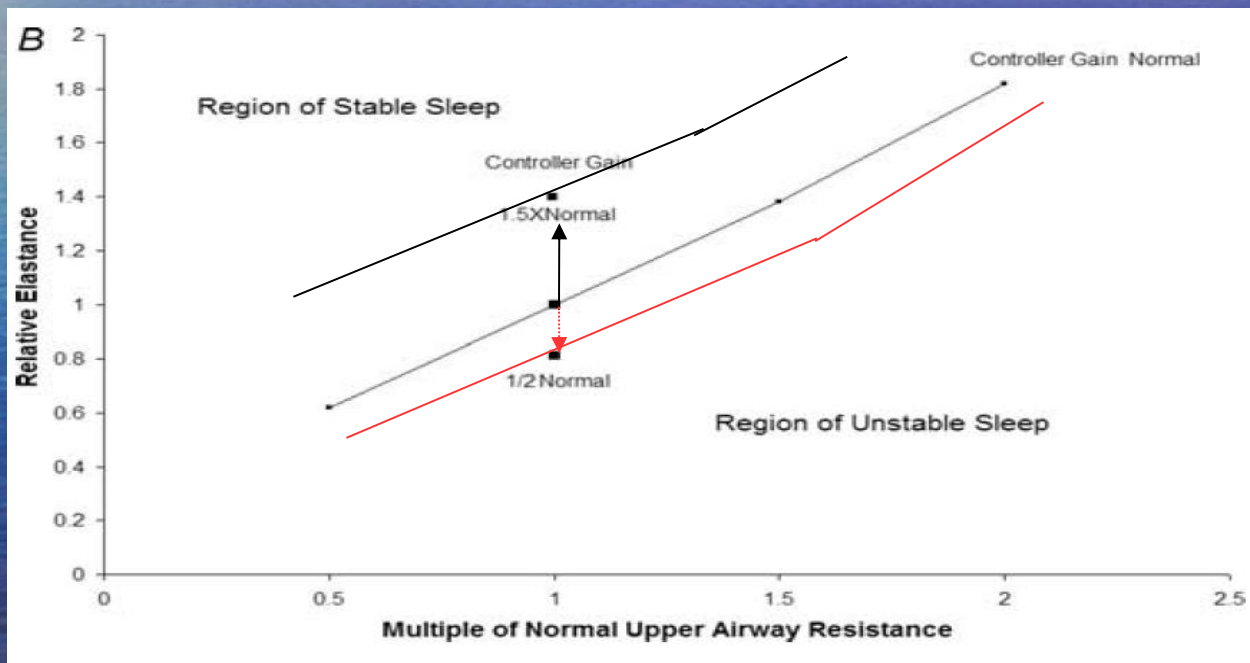


**Figure 5. Response of a rigid pharynx to sleep**

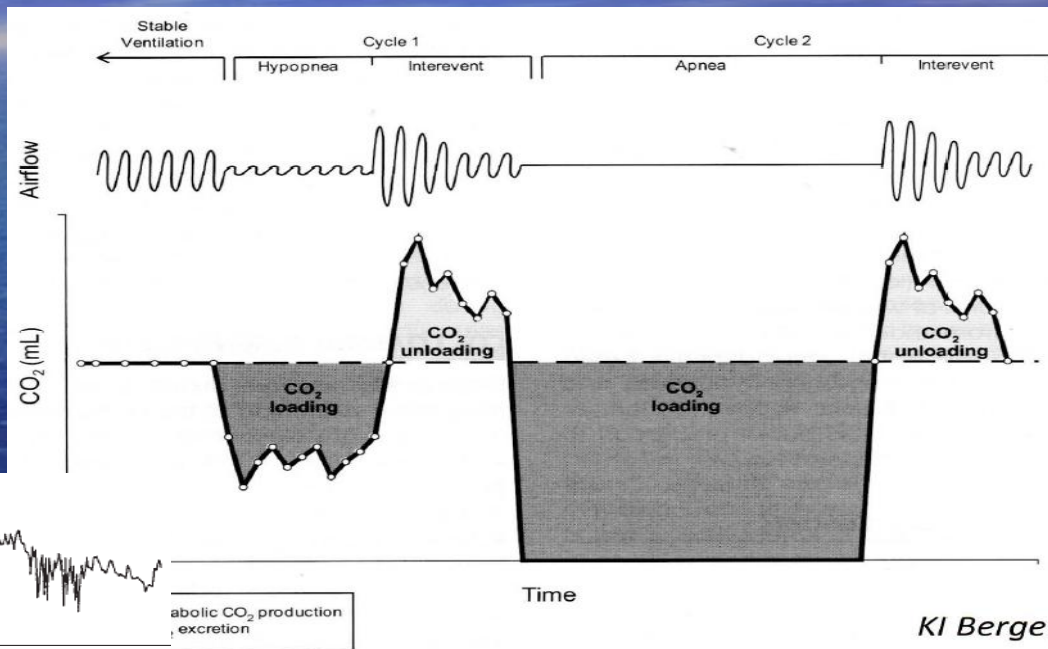
For normal controller gain, in *A*, there is one central apnoea followed by several non-apnoeic respiratory cycles after sleep onset. The arousal threshold and sleep thresholds during sleep are 15 and 14 l min<sup>-1</sup> of ventilatory drive, respectively. *B*, when controller gain is 1½ times normal, with the same arousal thresholds, the result is cycles of central apnoeas, each followed by arousal and hyperpnoea, which are present long after sleep onset. *C*, when the arousal threshold is raised so that arousal does not occur, apnoeas disappear much sooner.

# Les Voies aériennes Supérieures (VAS)

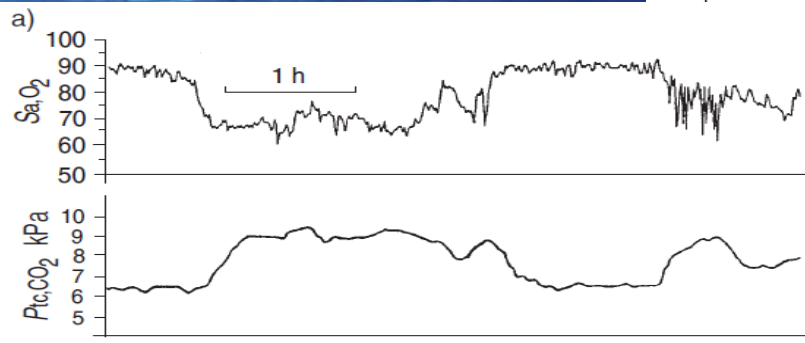
## Modélisations des Boucles d'Instabilité



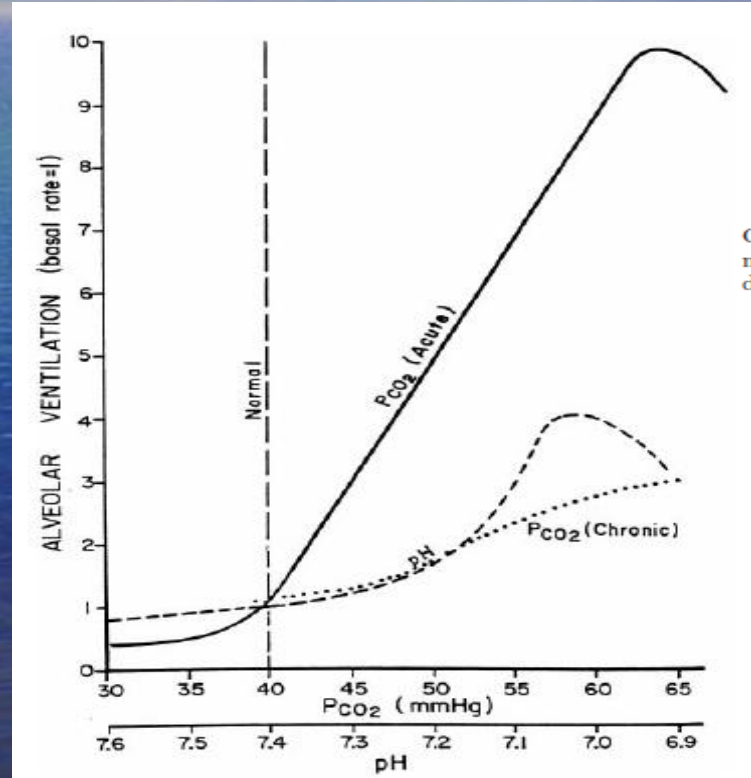
# Hypoventilation Nocturne: le lit de l'Hypoventilation Diurne ?



KI Berger, JAP, 2002



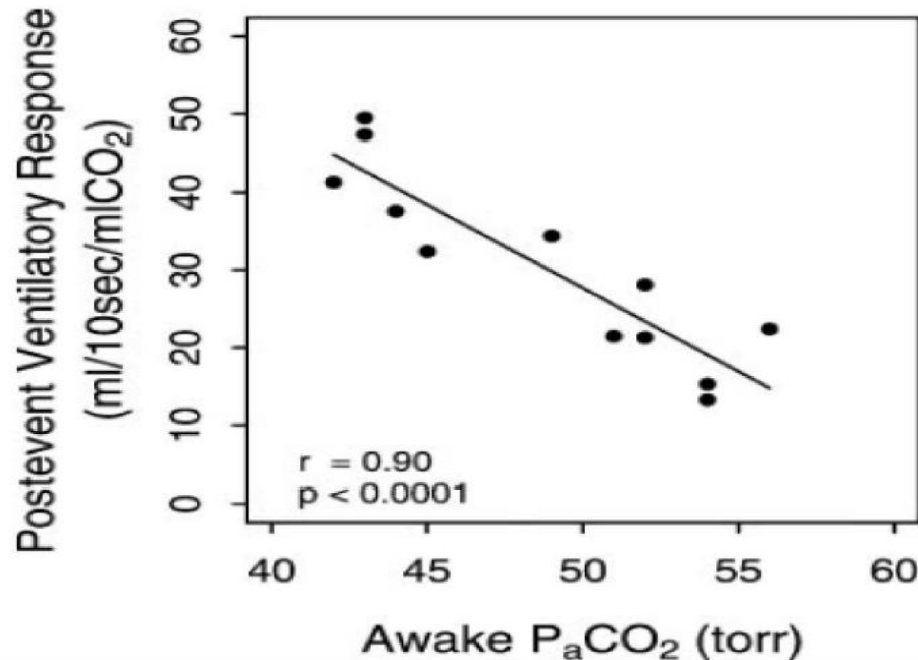
# Hypoventilation Nocturne: le lit de l'Hypoventilation Diurne ?



Guyton AC. Regulation of respiration, and respiratory abnormalities. In: Human physiology and mechanisms of disease. Philadelphia, PA: WB Saunders, 1982; 318-328

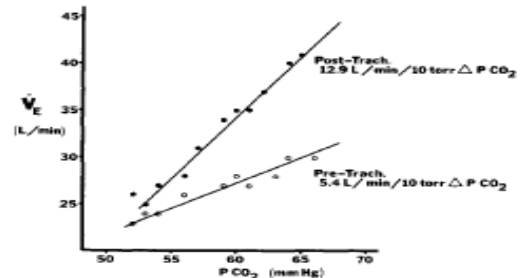
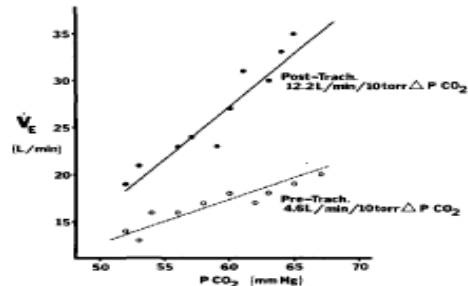
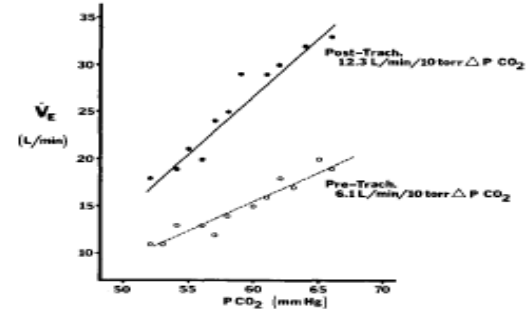
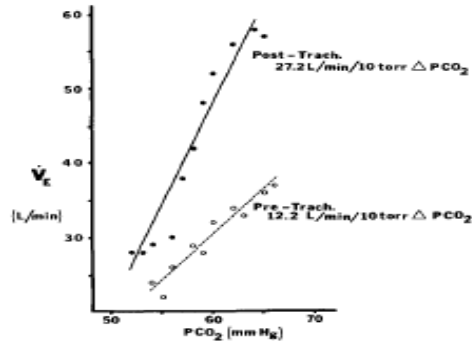
# Hypoventilation Nocturne: le lit de l'Hypoventilation Diurne ?

Centres Respiratoires



# Hypoventilation Nocturne: le lit de l'Hypoventilation Diurne ?

## Centres Respiratoires

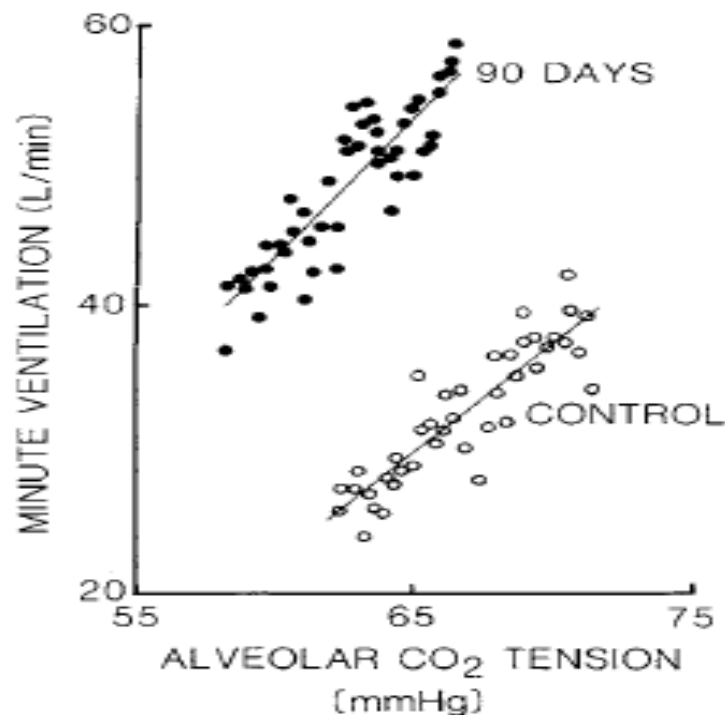


Progressive Improvement of Apnea Index and Ventilatory Response to  $CO_2$  after Tracheostomy in Obstructive Sleep Apnea Syndrome<sup>1-3</sup>

CHRISTIAN GUILLEMINAULT and JOSEPH CUMMISKEY

# Hypoventilation Nocturne: le lit de l'Hypoventilation Diurne ?

## Centres Respiratoires

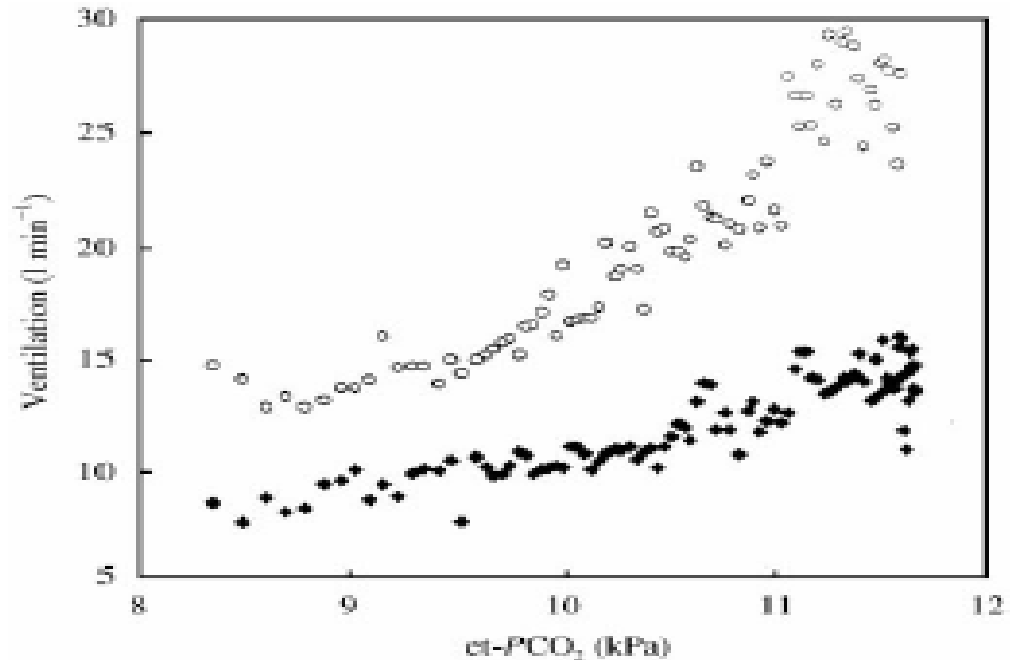


Time Course of Change in Ventilatory Response to CO<sub>2</sub> with Long-Term CPAP Therapy for Obstructive Sleep Apnea<sup>1,2</sup>

M. BERTHON-JONES<sup>1</sup> and C. E. SULLIVAN

# Hypoventilation Nocturne: le lit de l'Hypoventilation Diurne ?

## Centres Respiratoires

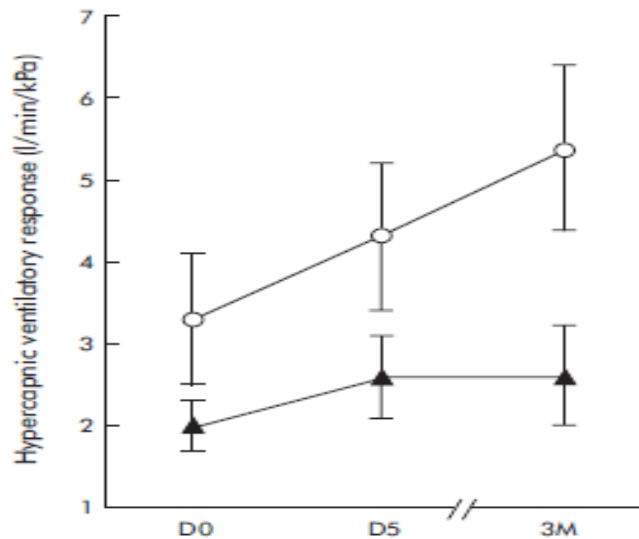


Ventilatory response to CO<sub>2</sub> re-breathing before and after Nocturnal Nasal Intermittent Positive Pressure Ventilation in patients with chronic alveolar hypoventilation *Respir. Med.* (2000) 94, 1154–1160

C. DELLBORG, J. OLOFSON, C.-H. HAMNÉGÅRD, B.-E. SKOOGH AND B. BÅKE

CPLF Janvier 2017

# Hypoventilation Nocturne: le lit de l'Hypoventilation Diurne ?



**Figure 2** Effect of NIV on hypercapnic ventilatory response (HCVR) in patients with neuromuscular weakness (open circles) and kyphoscoliosis (closed triangles). Mean (SE) HCVR at day 0 (D0), day 5 (D5), and 3 months (3M) is shown. For the group as a whole there was a significant rise in HCVR ( $p=0.04$ ).

Mechanisms of improvement of respiratory failure in patients with restrictive thoracic disease treated with non-invasive ventilation

A H Nickol, N Hart, N S Hopkinson, J Moxham, A Simonds, M I Polkey

*Thorax* 2005;60:754-760. doi: 10.1136/thx.2004.039388



# VNI Mécanismes d'Action

## Les muscles ? Les Centres ?

**Table 2—Strength and Endurance at Baseline, Three Months, and 14 Months After Nocturnal Ventilation\***

|          | MIP <sub>RV</sub> ,<br>– cm H <sub>2</sub> O | MIP <sub>FRC</sub> ,<br>– cm H <sub>2</sub> O | MEP,<br>cm H <sub>2</sub> O | MVV,<br>L/min | $\bar{P}_m$ ,<br>%MIP <sub>RV</sub> | TLIM,<br>min |
|----------|----------------------------------------------|-----------------------------------------------|-----------------------------|---------------|-------------------------------------|--------------|
| Baseline | 42 ± 10                                      | 30 ± 7                                        | 81 ± 28                     | 29 ± 22       | 44 ± 13                             | 7.1 ± 3.4    |
| 3 mo     | 51 ± 17                                      | 32 ± 7                                        | 92 ± 18                     | 34 ± 20       | 38 ± 10                             | 14.8 ± 7.6†  |
| 14 mo    | 47 ± 14                                      | 28 ± 10                                       | 84 ± 42                     | 39 ± 32       | 49 ± 5                              | 17.2 ± 6.3   |

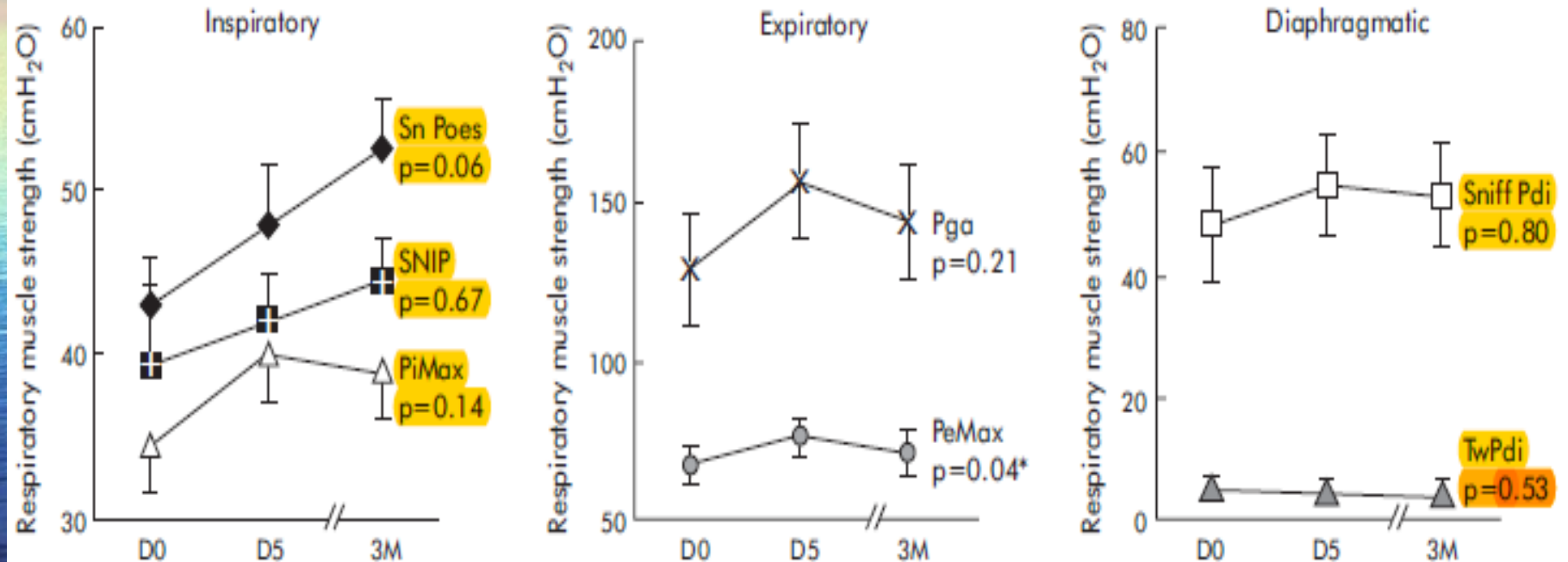
### Influence of Noninvasive Positive Pressure Ventilation on Inspiratory Muscles\*

Roger S. Goldstein, M.D., F.C.C.P.; Jim A. De Rosie, B.Sc.;  
Monica A. Avendano, M.D., F.C.C.P.; and Tom E. Dolmage, M.Sc.

Amélioration d'un Index d' « Endurance »  
des Muscles Respiratoires

# VNI Mécanismes d'Action

## Les muscles ? Les Centres ?



Mechanisms of improvement of respiratory failure in patients with restrictive thoracic disease treated with non-invasive ventilation

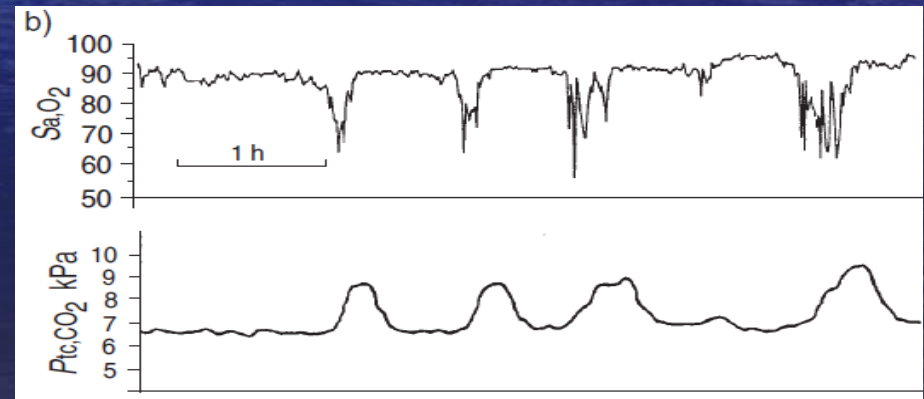
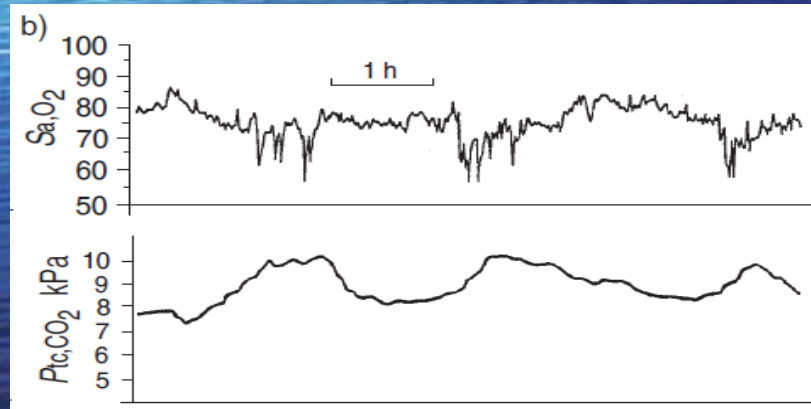
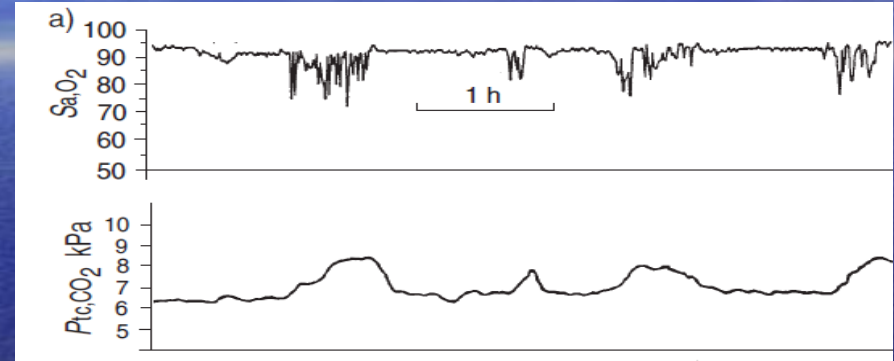
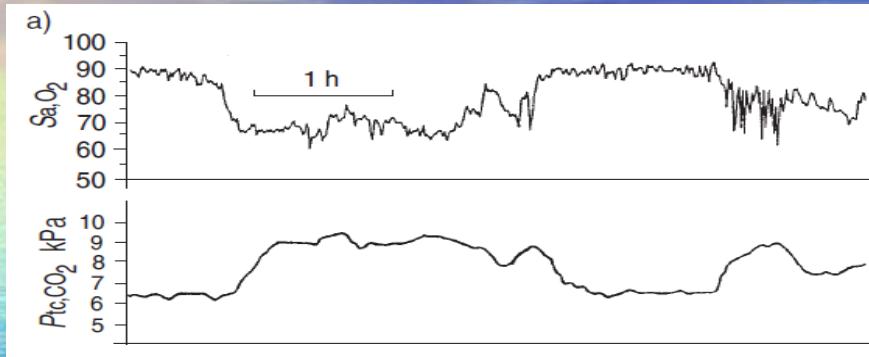
A H Nickol, N Hart, N S Hopkinson, J Moxham, A Simonds, M I Polkey

Thorax 2005;60:754-760. doi: 10.1136/thx.2004.039386



# VNI Mécanismes d'Action

## Leçons du Sevrage de VNI



# VNI et Respiration Périodique

## Sleep Fragmentation in Kyphoscoliotic Individuals With Alveolar Hypoventilation Treated by NIPPV\*

John R. Bach, MD, FCCP;† Dominique Robert, MD;  
Patrick Leger, MD; and Bruno Langevin, MD *CHEST* 1995; 107:1552-58.

## Reduced survival in patients with ALS with upper airway obstructive events on non-invasive ventilation

Marjolaine Georges,<sup>1,2</sup> Valérie Attali,<sup>2,3</sup> Jean Louis Golmard,<sup>4</sup>  
Capucine Morélot-Panzini,<sup>1,2</sup> Lise Crevier-Buchman,<sup>5,6</sup> Jean-Marc Collet,<sup>3</sup>  
Anne Tintignac,<sup>1</sup> Elise Morawiec,<sup>1</sup> Valéry Trosini-Desert,<sup>1</sup> François Salachas,<sup>7</sup>  
Thomas Similowski,<sup>1,2</sup> Jesus Gonzalez-Bermejo<sup>1,2</sup>

Georges M, et al. *J Neurol Neurosurg Psychiatry* 2016;0:1–6. doi:10.1136/jnnp-2015-312606

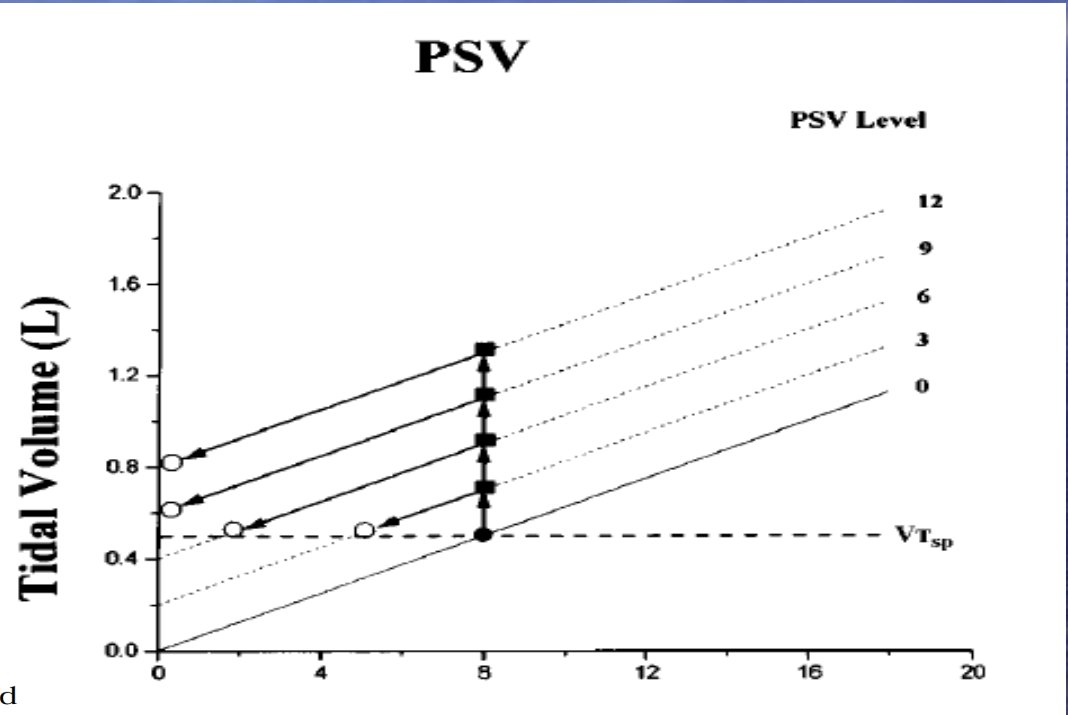
## Susceptibility to periodic breathing with assisted ventilation during sleep in normal subjects

SONIA MEZA, MANUEL MENDEZ, MICHELE OSTROWSKI, AND MAGDY YOUNES  
*Respiratory Medicine, University of Manitoba, Winnipeg, Manitoba, Canada R3A 1R8*

*J. Appl. Physiol.* 85(5): 1929–1940, 1998.

# Interaction Ventilateur/Compartment Ventilé

Equivalent d'une augmentation du  
Gain du Contrôleur

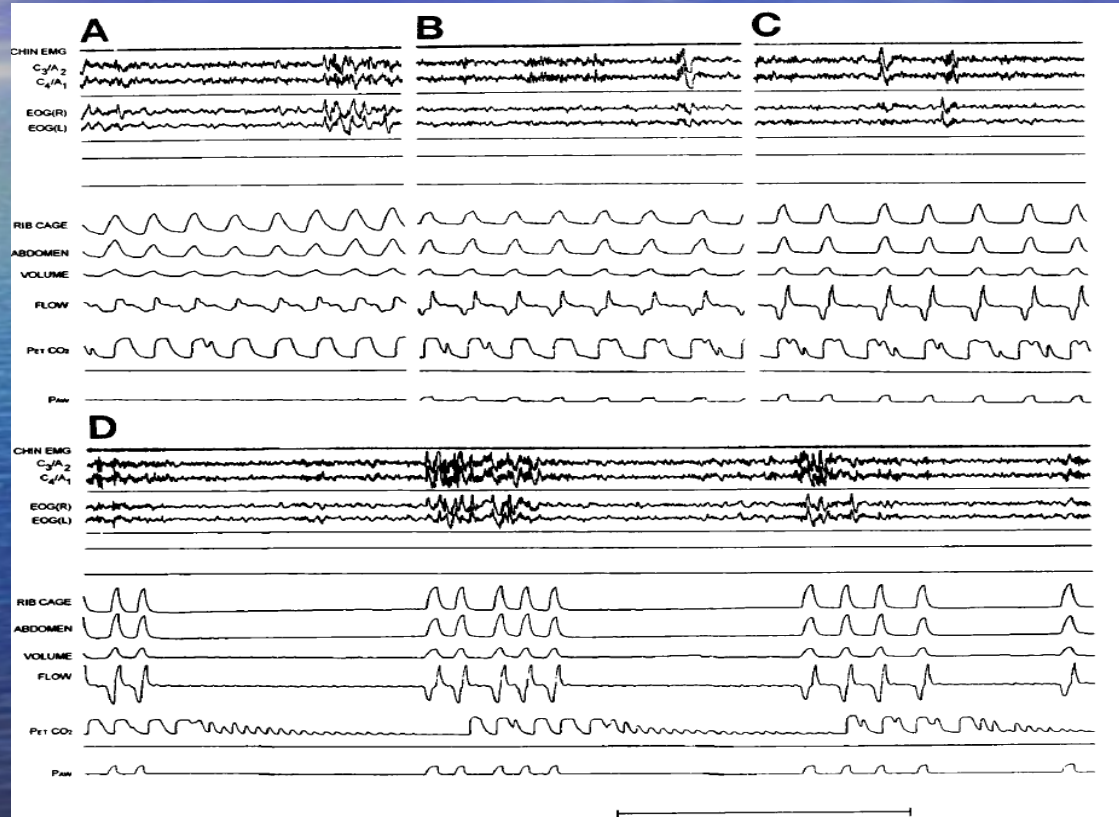


Susceptibility to periodic breathing with assisted ventilation during sleep in normal subjects

# Interaction Ventilateur/Compartment Ventilé

Susceptibility to periodic breathing with assisted ventilation during sleep in normal subjects

SONIA MEZA, MANUEL MENDEZ, MICHELE OSTROWSKI, AND MAGDY YOUNES  
Respiratory Medicine, University of Manitoba, Winnipeg, Manitoba, Canada R3A 1R8



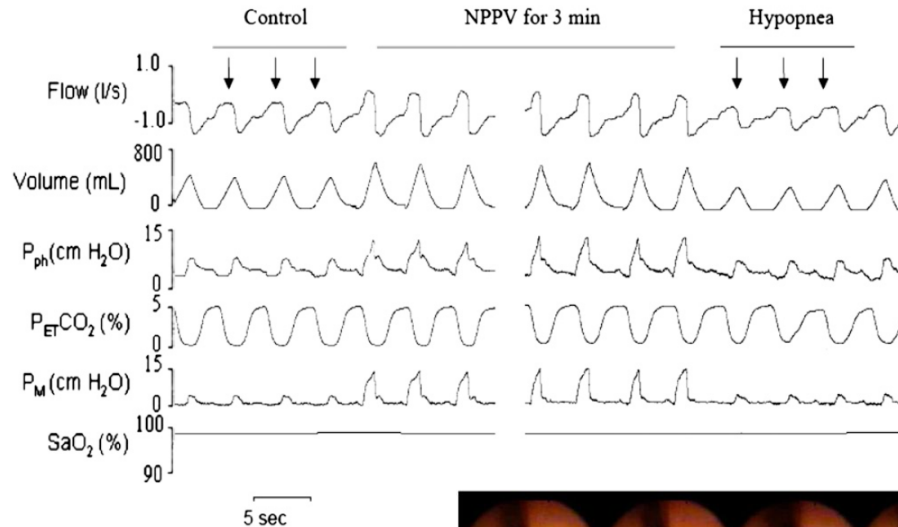
AI : A=0

B=3

C=6

D=8

# Les Voies aériennes Supérieures



## Expiratory Pharyngeal Narrowing during Central Hypocapnic Hypopnea

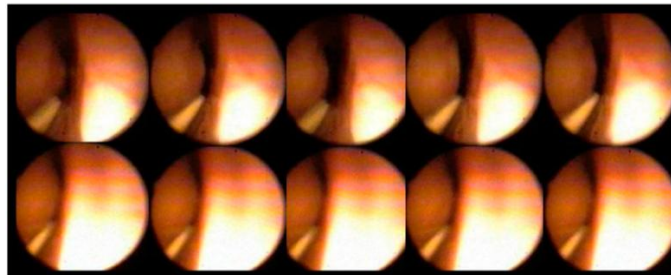
Abdul Ghani Sankri-Tarbichi<sup>1</sup>, James A. Rowley<sup>1</sup>, and M. Safwan Badr<sup>1</sup>

Am J Respir Crit Care Med Vol 179. pp 313-319, 2009

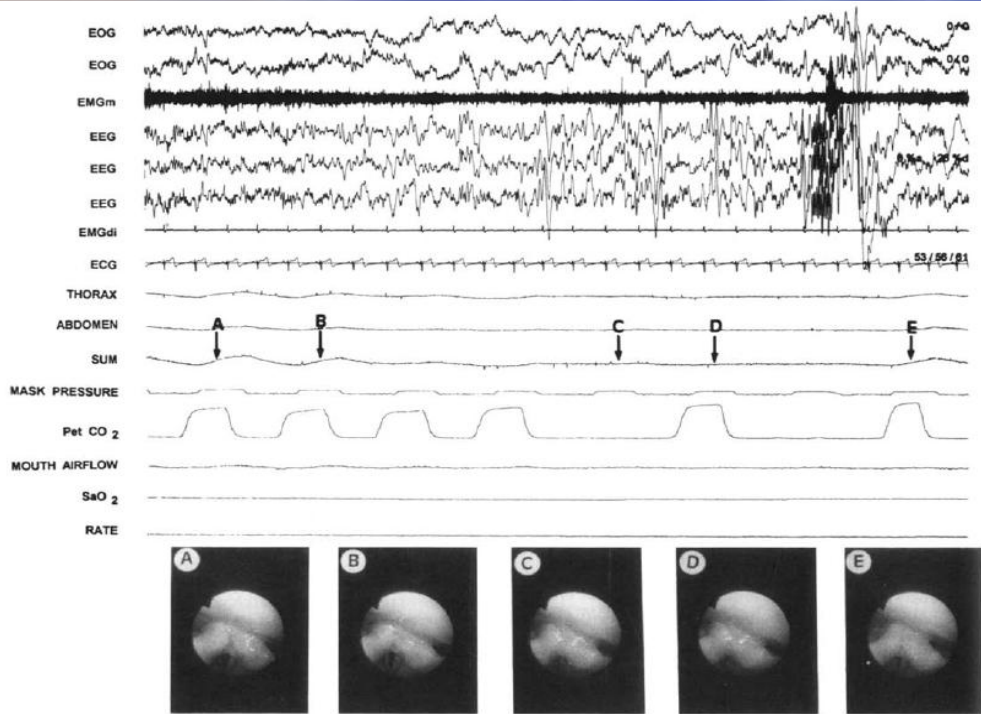
**Figure 1.** Polygraph record of a trial from analysis 1 that illustrates breaths before and during noninvasive positive pressure ventilation (NPPV) followed by central hypopnea. Three breaths preceding NPPV (control, arrows) were compared with the breaths after termination of NPPV (hypocapnic hypopnea, arrows).  $P_{ETCO_2}$  = end tidal CO<sub>2</sub>;  $P_{ph}$  = pharyngeal pressure;  $P_M$  = mask pressure;  $SaO_2$  = arterial oxygen saturation.

C

H



# Les Voies aériennes Supérieures !



**Figure 4.** Top panel. Polygraph recording of electrooculogram (EOG), submental chin electromyogram (EMGm), electroencephalogram (EEG), raw diaphragmatic electromyogram (EMGdi), electrocardiogram (ECG), thorax, abdomen, and SUM signals from the RespiTrace, mask pressure, end-tidal CO<sub>2</sub> pressure (PETCO<sub>2</sub>), mouth airflow, oxygen saturation (SaO<sub>2</sub>) and pulse rate (RATE) recorded in Subject 4 mechanically ventilated with an inspiratory pressure of 15 cm H<sub>2</sub>O during Stage 2 NREM sleep. Bottom panel. The glottis (A to E) was photographed during five mechanical insufflations, which are indicated by arrows on the polygraph recording. The inspiratory vocal cords angles were as follows: A: 27.5 degrees, B: 25 degrees, C: 5 degrees, D: 0 degrees, E: 27.5 degrees. Note the complete glottic closure and absence of Vt corresponding to an apnea in Cycle D without changes in sleep/wake status. This record was performed with a digital system and corresponds to 30 s.

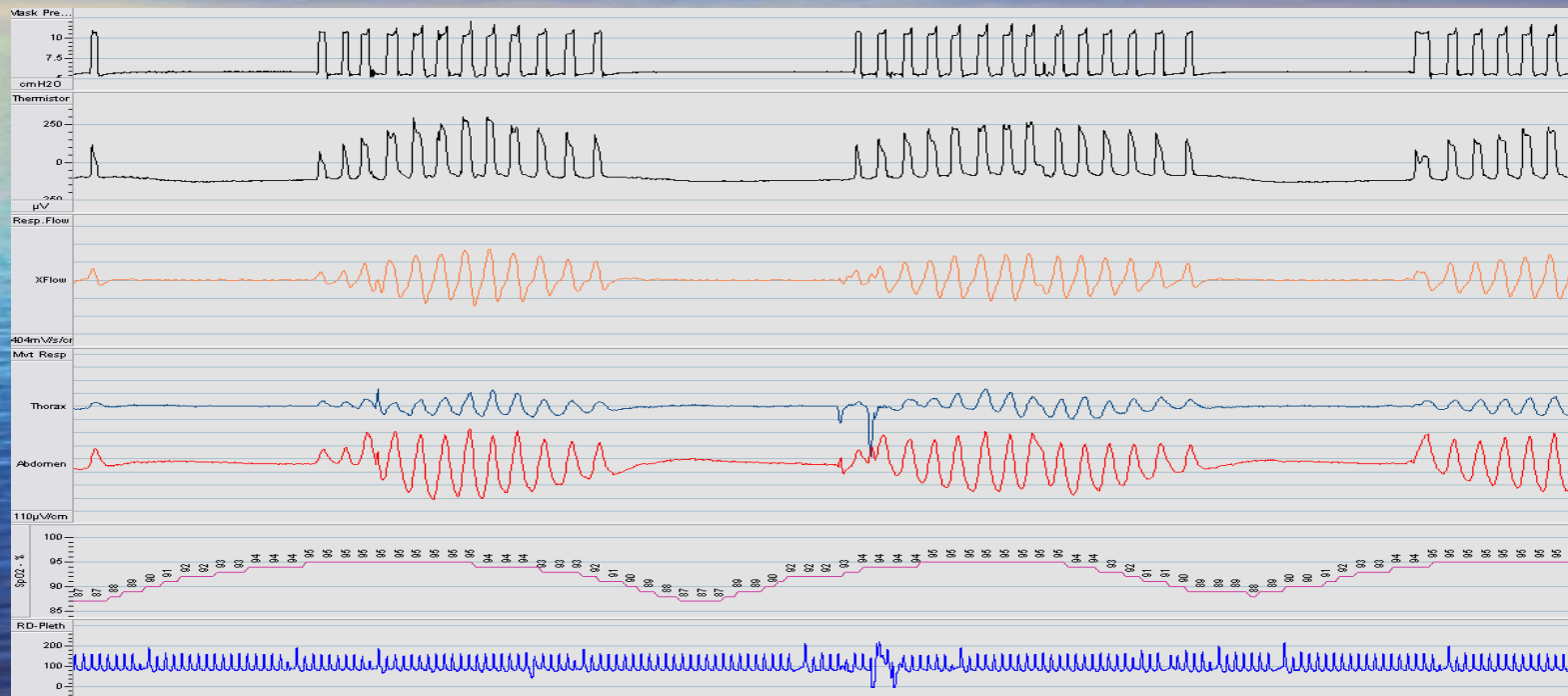
## Nasal Two-Level Positive-pressure Ventilation in Normal Subjects

Effects on the Glottis and Ventilation

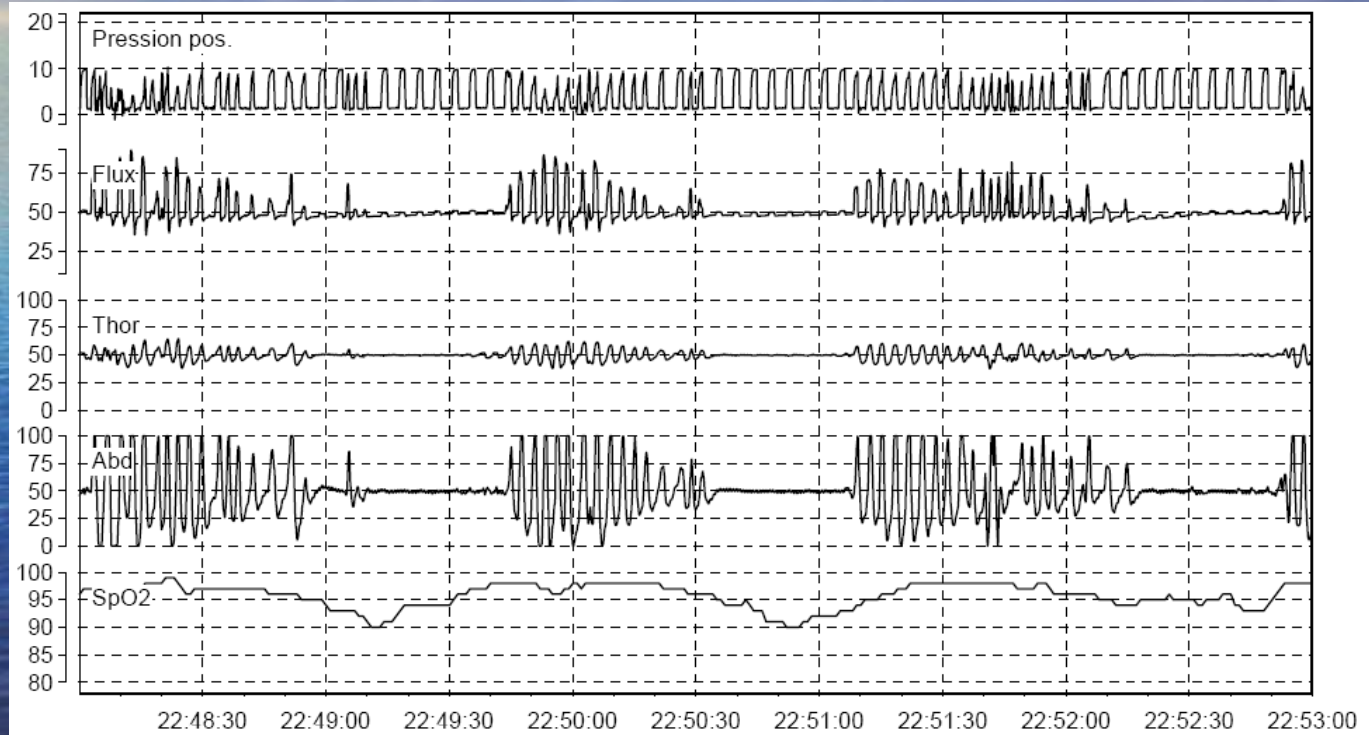
VERONICA FRANCO PARREIRA, VINCENT JOUNIEAUX, GENEVIEVE AUBERT, MYRIAM DURAN, PIERRE EMILE DELGUSTE, and DANIEL OSCAR RODENSTEIN

AM J RESPIR CRIT CARE MED 1996;153:1616-23.

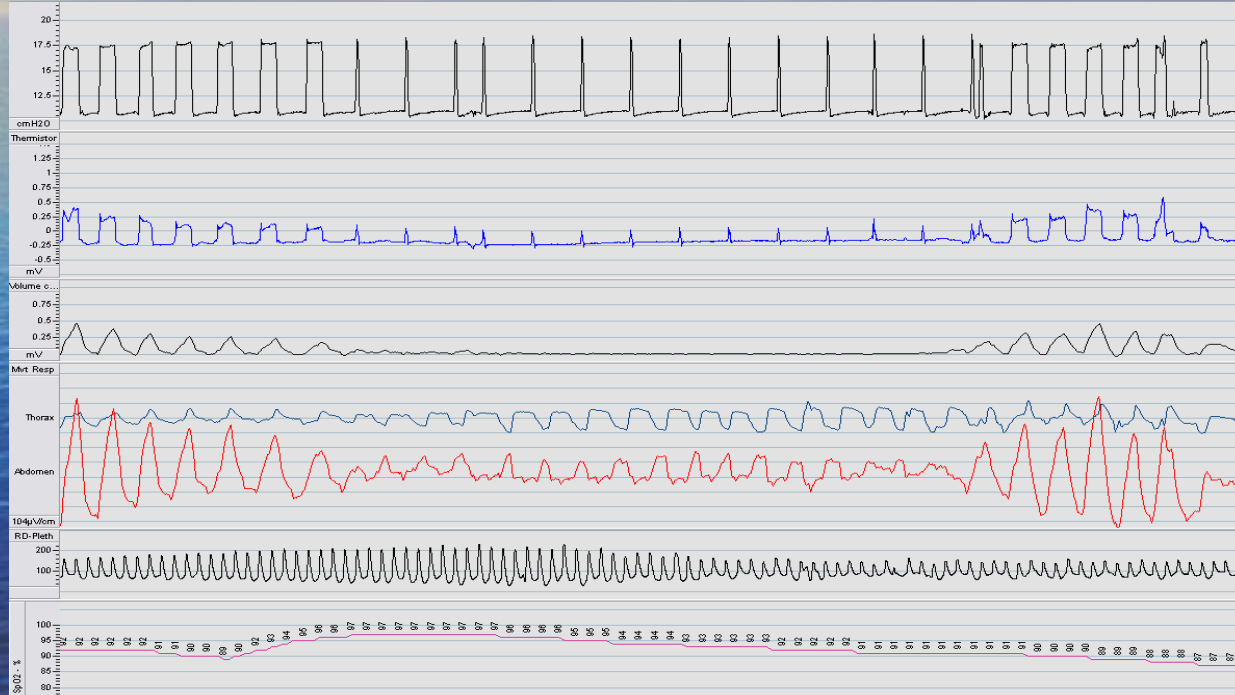
# Interaction Ventilateur/Compartment Ventilé



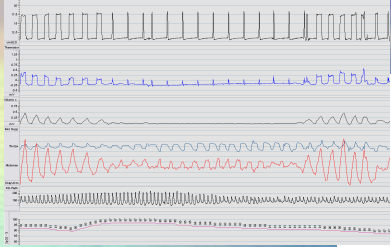
# Interaction Résistances VAS et Ventilation Alvéolaire



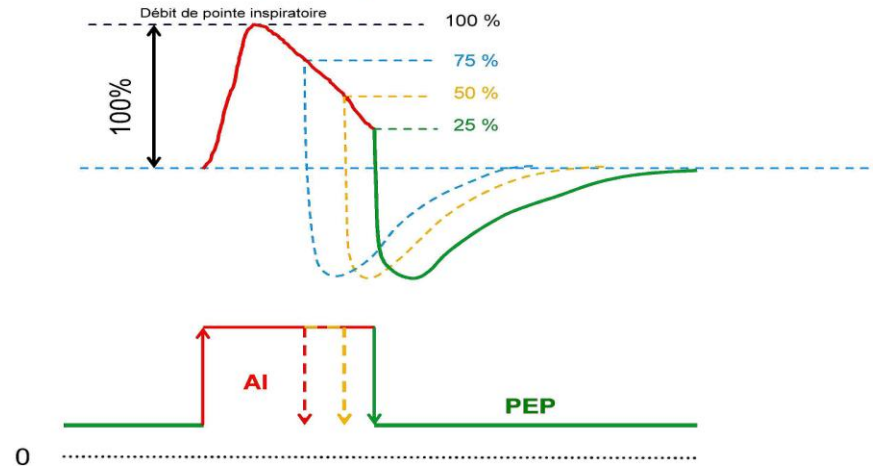
# Interaction Résistances VAS et Ventilation Alvéolaire



# Interaction Résistances VAS et Ventilation Alvéolaire. Cyclage

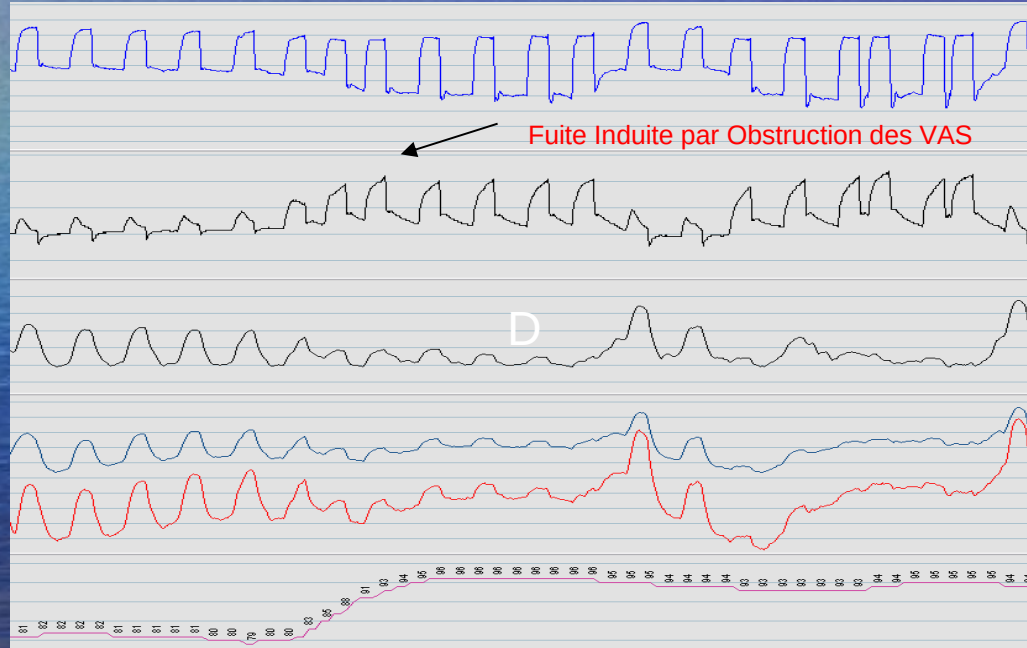


## Les ventilateurs conventionnels Méthode du % du débit de pointe inspiratoire



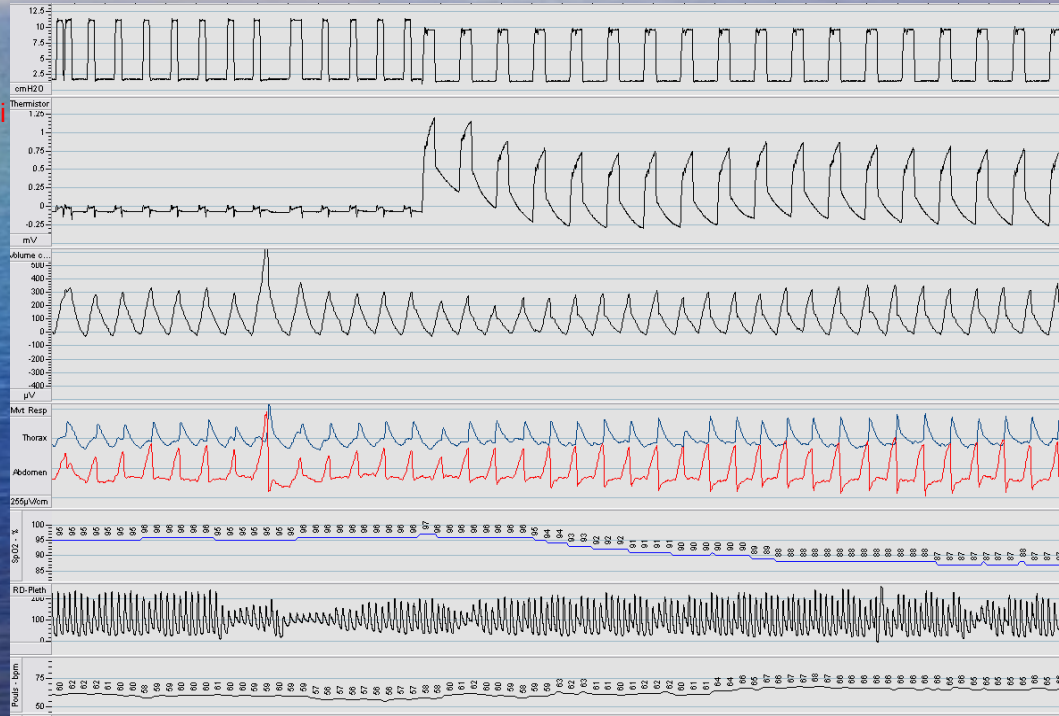
# Interaction Résistances VAS et Ventilation Alvéolaire

Le Cyclage dépend  
Aussi de Ti Min et des  
Réglages du Mode T  
Selon Ventilateur

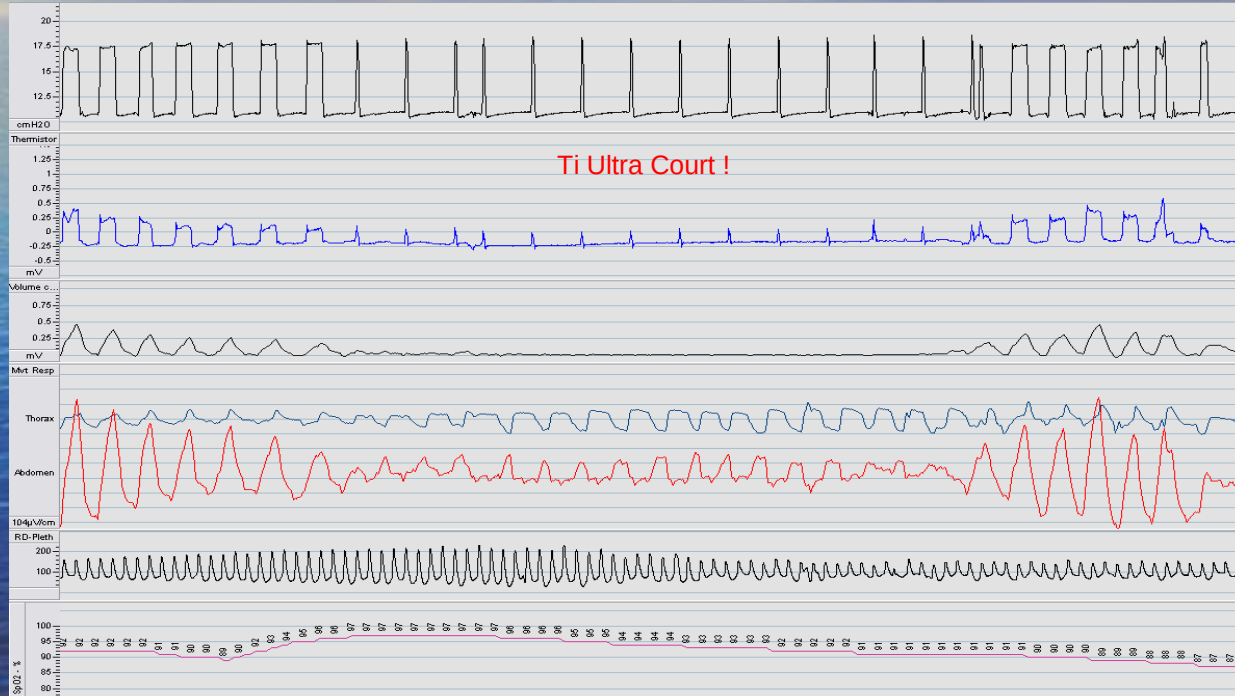


# Interaction Résistances VAS et Ventilation Alvéolaire

Noter le Raccourcissement du T<sub>i</sub>  
Par cyclage précoce



# Interaction Résistances VAS et Ventilation Alvéolaire

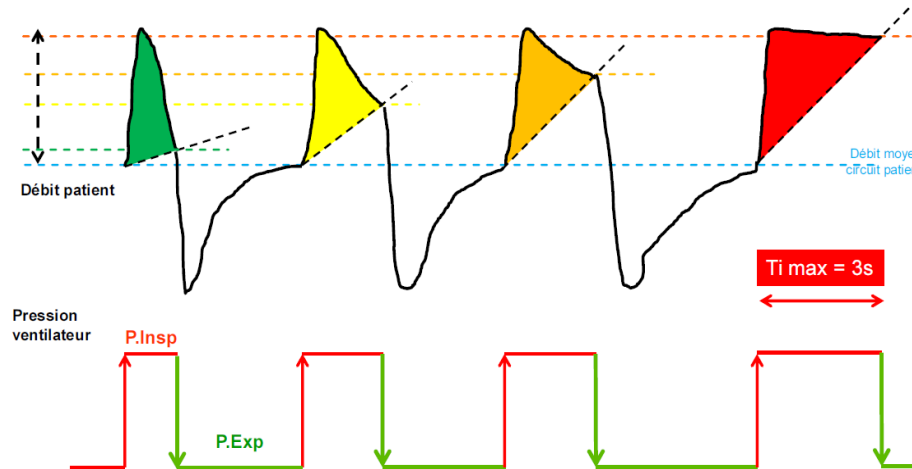


# Interaction Résistances VAS et Ventilation Alvéolaire

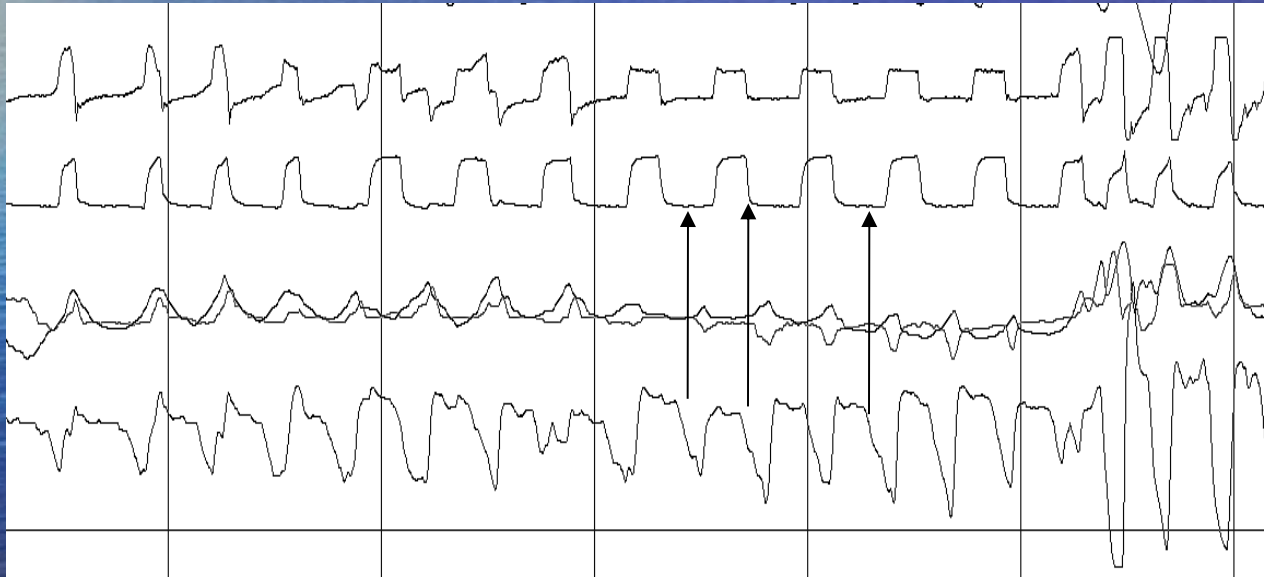
## Comment Régler le Cyclage ?

Les ventilateurs de technologie BiPAP®

Algorithme Auto-Trak: Seuil Expiratoire Spontané (SES)

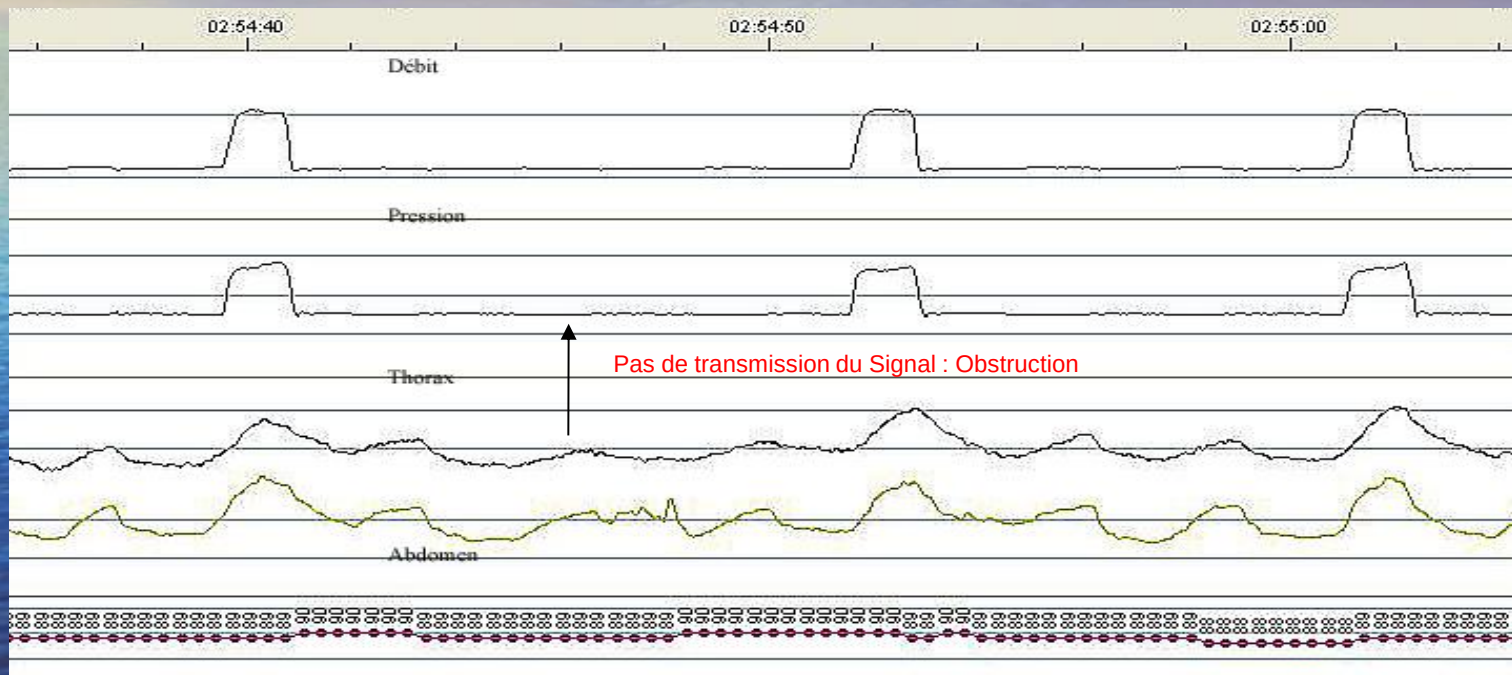


# Interaction Résistances VAS et Trigger



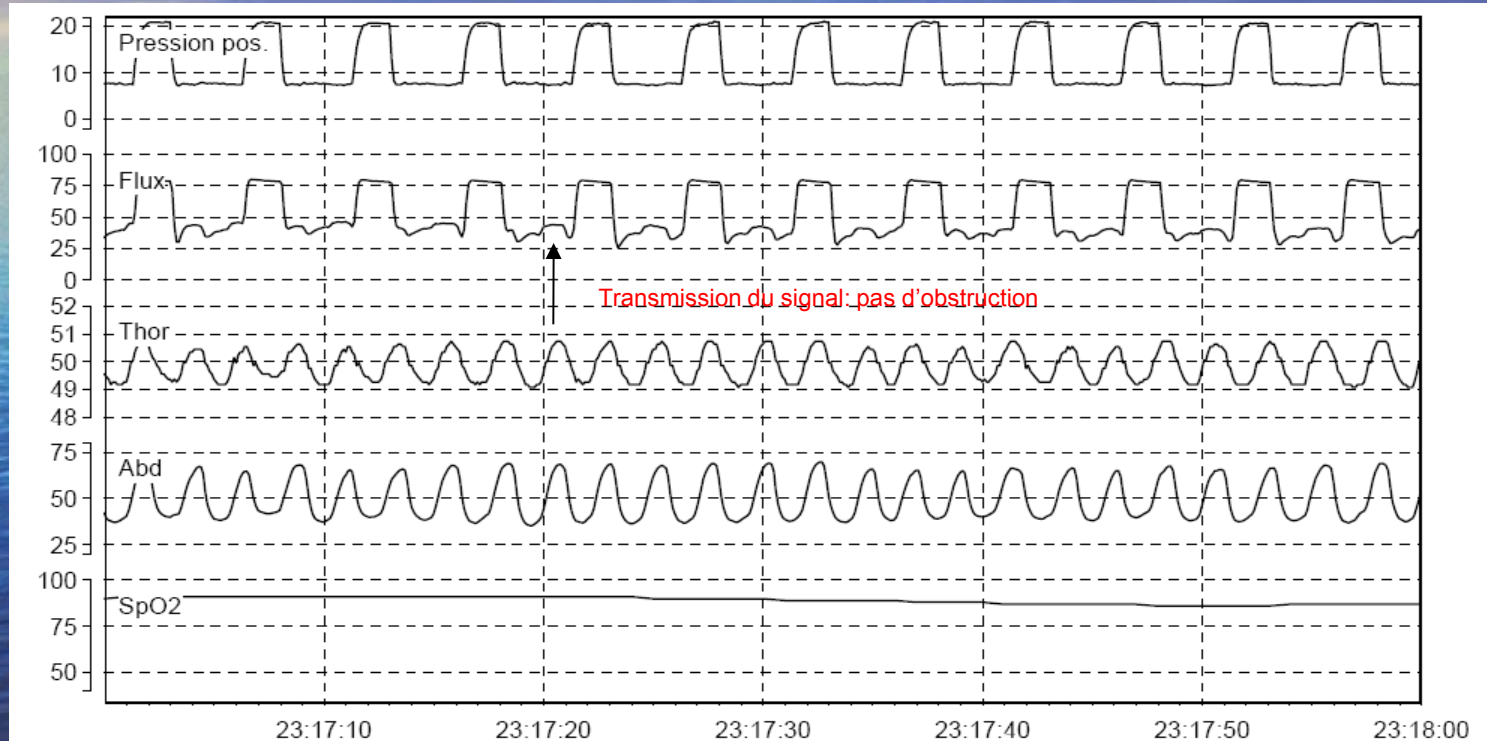
# VNI: Stabiliser les VAS

## Déclenchement du Ventilateur (Trigger)

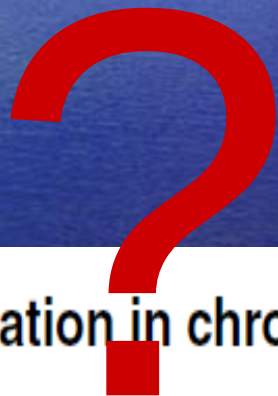


# VNI: Stabiliser les VAS

## Déclenchement du Ventilateur (Trigger)



# CONCLUSIONS



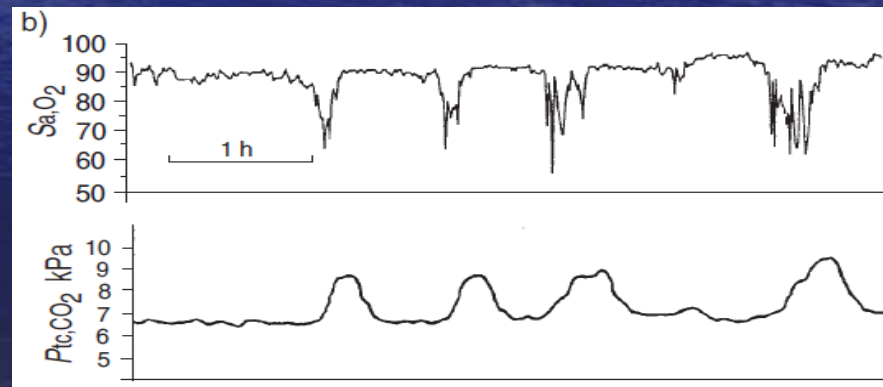
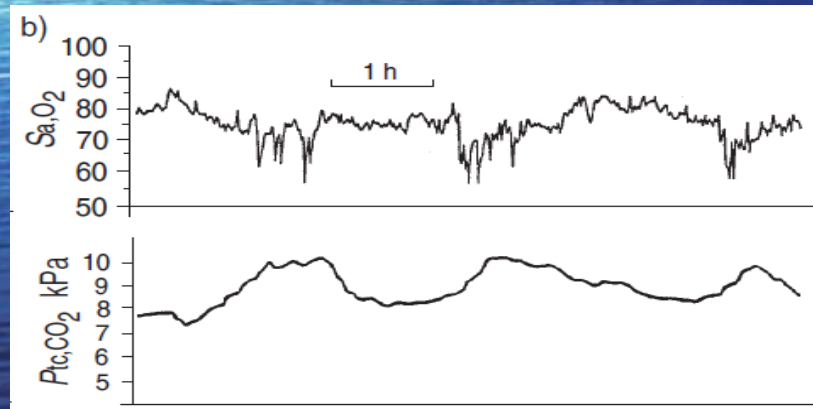
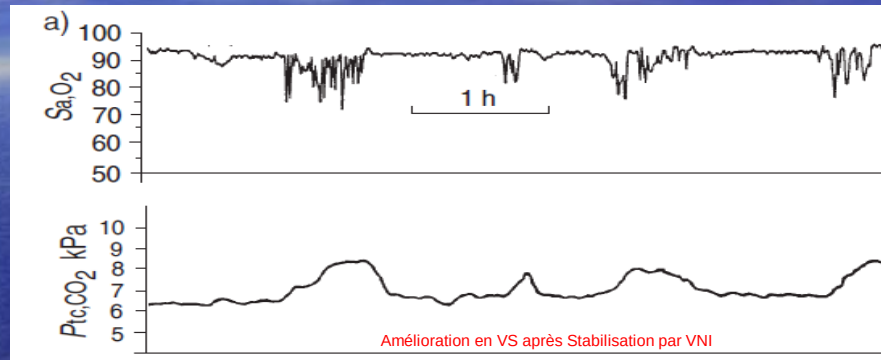
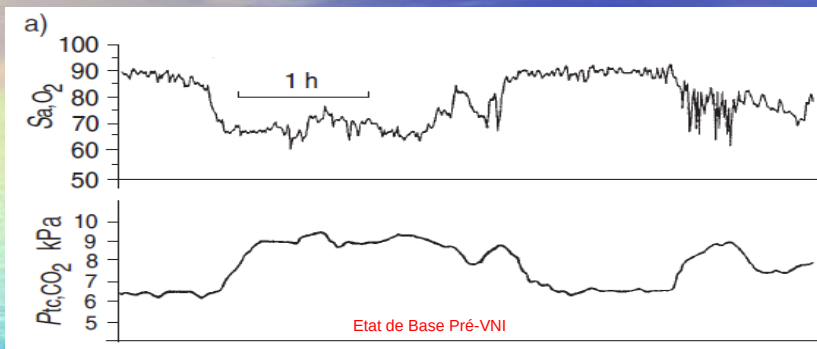
## **Daytime mechanical ventilation in chronic respiratory insufficiency**

*Eur Respir J 1997; 10: 2840–2846.*

B. Schönhofer, M. Geibel, M. Sonneborn, P. Haidl, D. Köhler

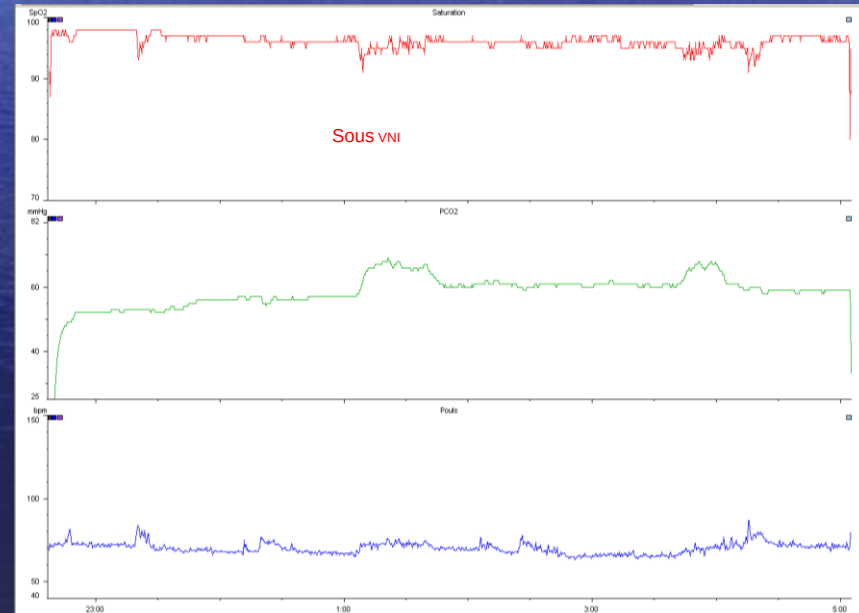
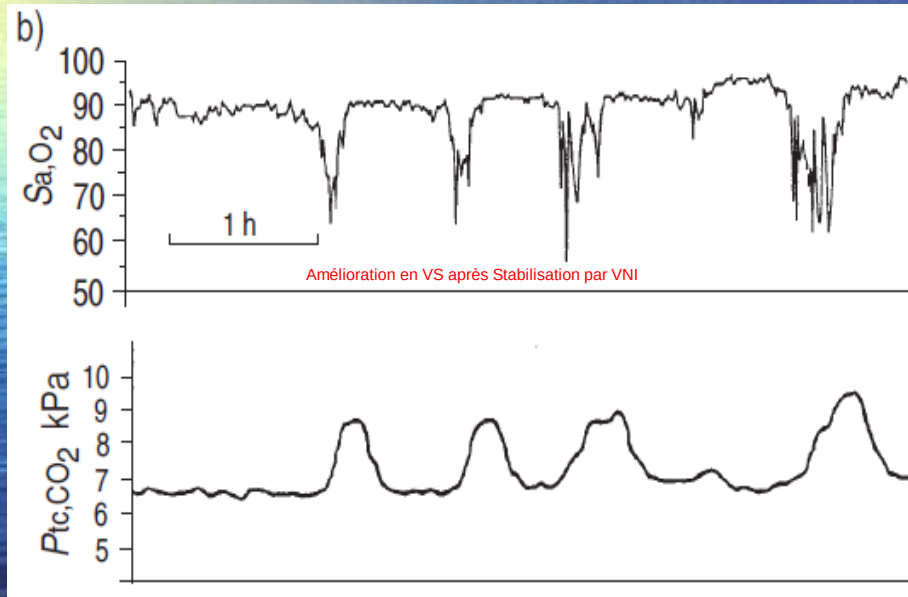
# CONCLUSIONS

## a) Ventilation Nocturne

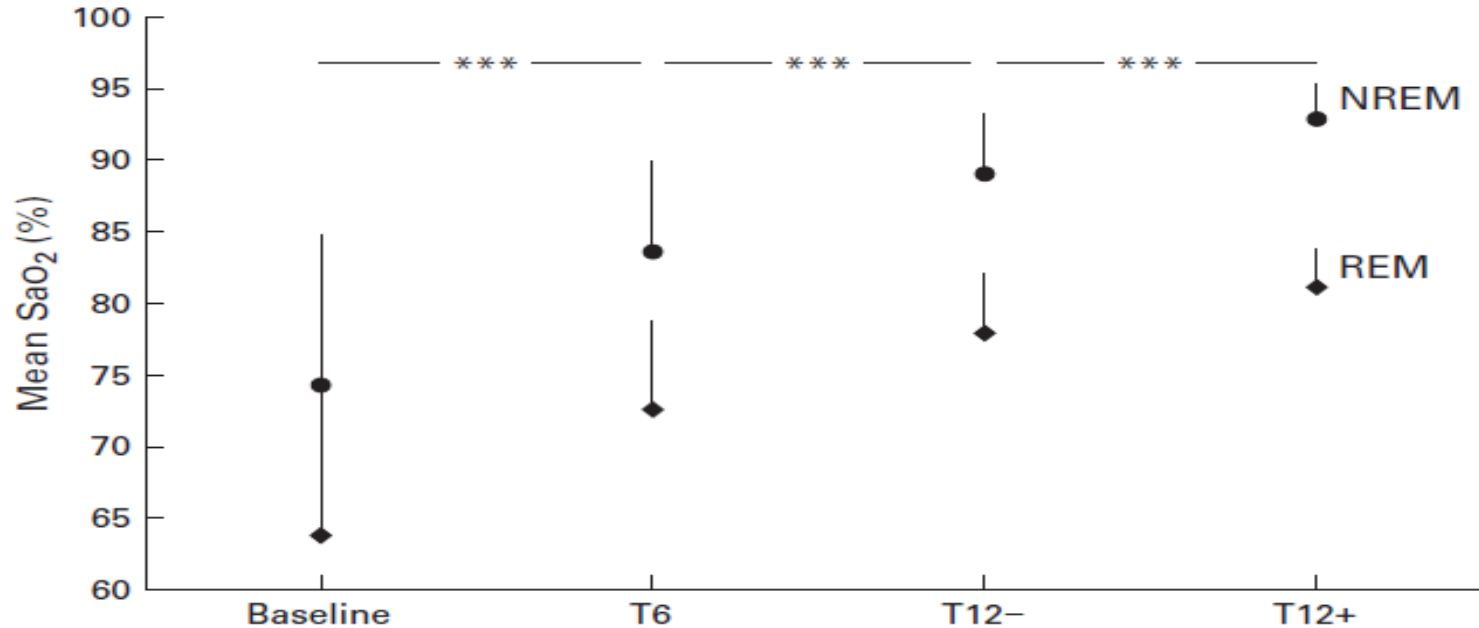


## b) Ventilation Diurne

# CONCLUSIONS



# Conclusions



Effect of non-invasive mechanical ventilation on sleep and nocturnal ventilation in patients with chronic respiratory failure

Bernd Schönhofer, Dieter Köhler

*Thorax* 2000;55:308–313

Hors VNI

Sous VNI

CPLF Janvier 2017