XXXIII Diada Pneumològica

17 i 18 d'abril de 2015

L'Hospitalet - Barcelona

SOCAP

Hospital Universitari de Bellvitge

Oxigenoteràpia en el pacient amb IRA/IRC hospitalitzat "L'oxígen com a medicació"

Efectes beneficiosos i deleteris de l'oxigenoteràpia

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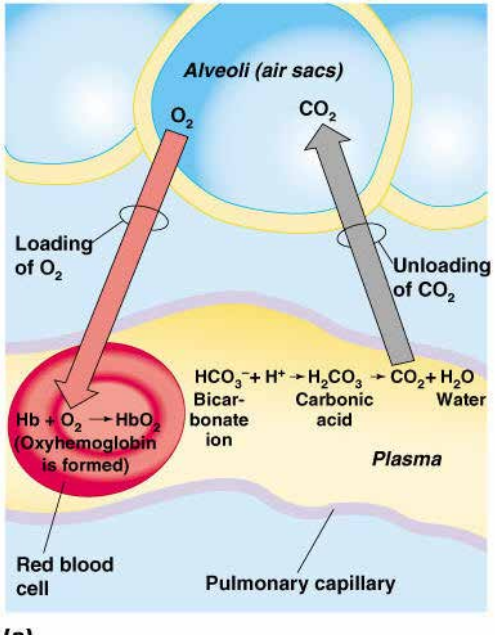
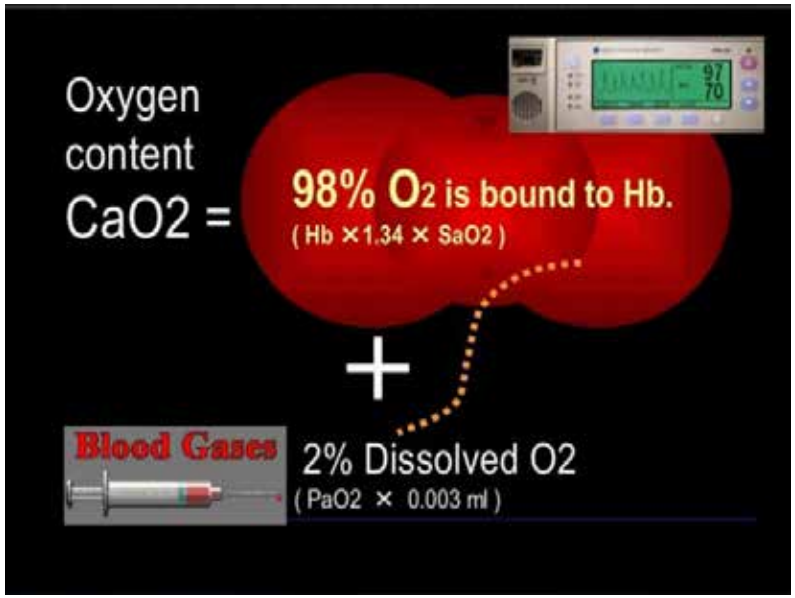
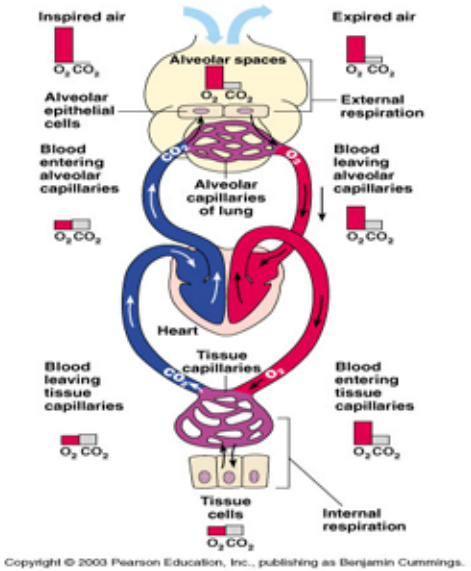
Guió

- Efectes beneficiosos de la oxigenoteràpia. El concepte de transport d'O2 en el pacient agut
- Efectes adversos de la oxigenoteràpia
  - Associats a FiO2 inadequades
    - Efectes sobre l'intercanvi pulmonar de gasos.
    - Efectes de la hiperoxigenació.
  - Associats a fluxe de gas no condicionat.
- Conclusions

Breu recordatori de la fisiologia de l'O2.

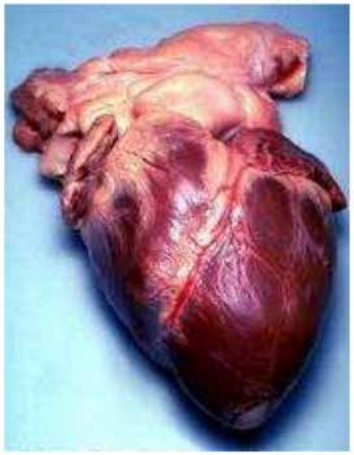




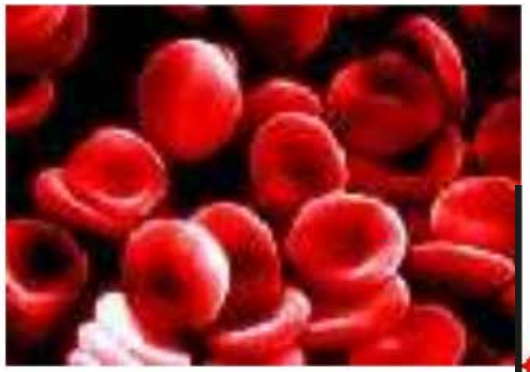


Transport d'O2 (DO2) = CO x (1.34 x Hb x Sat + 0.0031 x PaO2)

Consum d'O2 "normal"= 250 ml/ min.

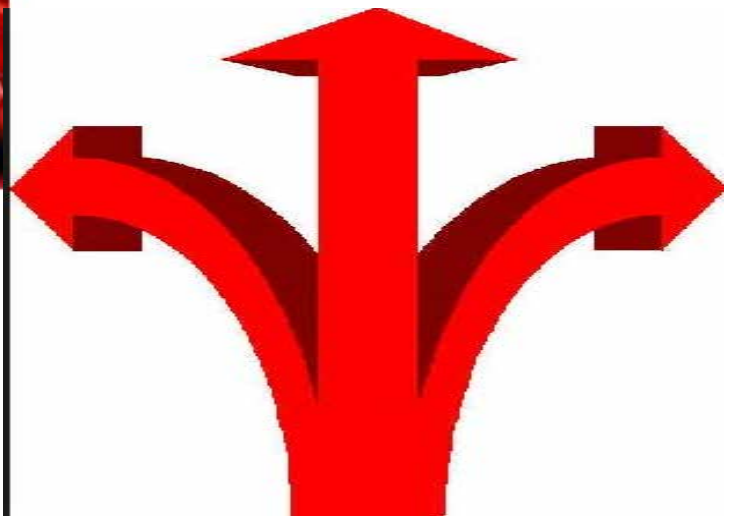


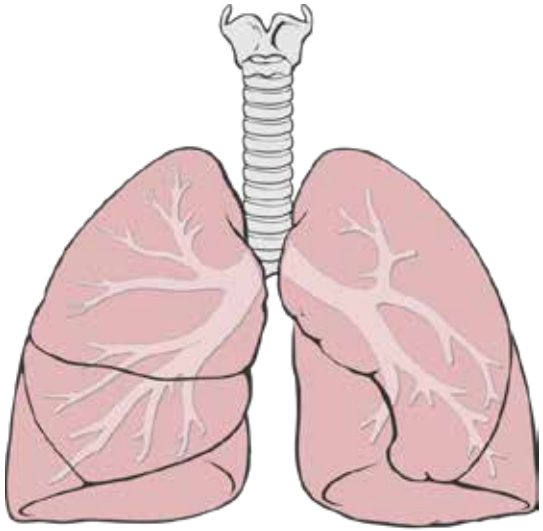
Insuficiència cardiaca
CO < 5 l/min.



Anèmia

Hb < 10 g/dL





Alteracions d’intercanvi
o bomba ventilatòria

Transport d’O2 (DO2) = CO x (1.34 x Hb x Sat + 0.0031 x PaO2)

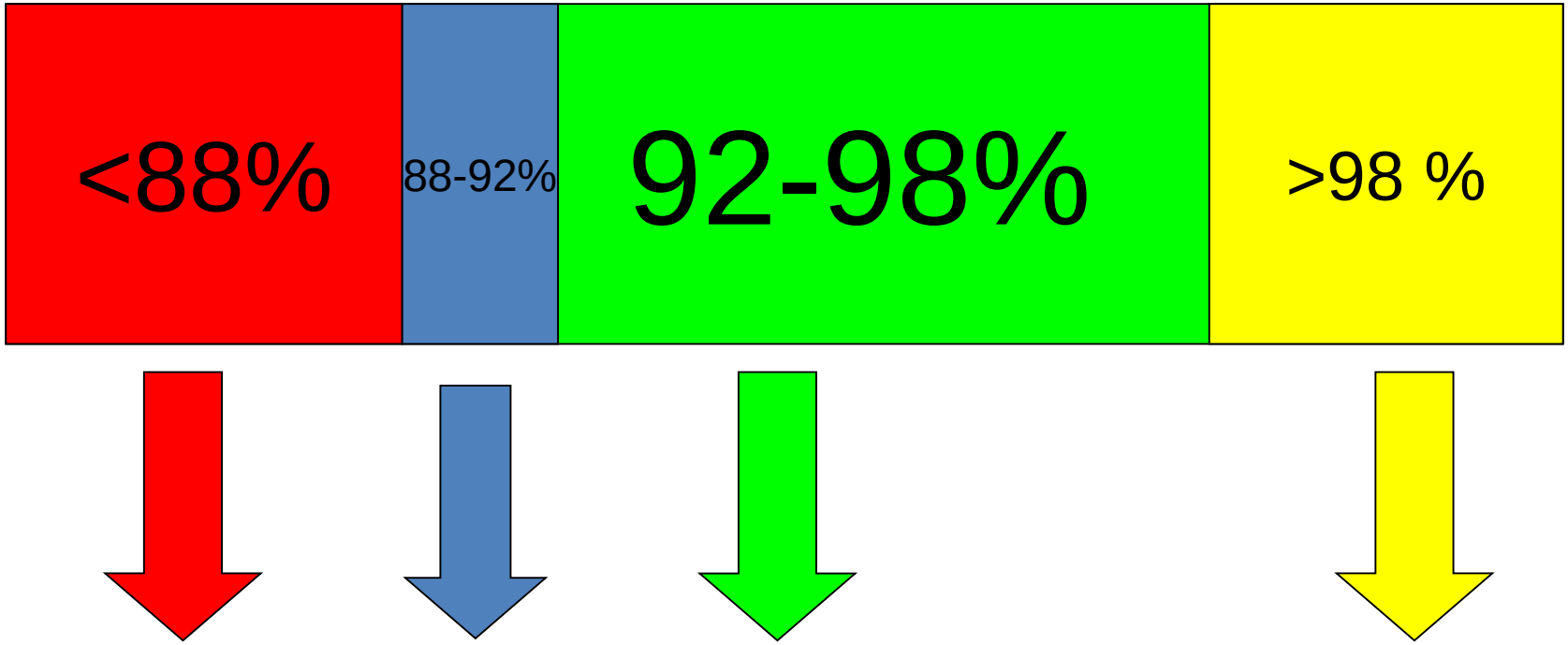
Efectes de la hipòxia.

	Hypoxia	
	Effects	**Risks**
Respiratory system	Increased ventilation Pulmonary vasoconstriction	Pulmonary hypertension
Cardiovascular system	Coronary vasodilation Decreased systemic vascular resistance (transient) Increased cardiac output Tachycardia	Myocardial ischaemia/ infarction Ischaemia/infarction of other critically perfused organs Hypotension Arrhythmias
Metabolic system	Increased 2,3-DPG Increased CO_2 carriage (Haldane effect)	Lactic acidosis
Neurological system	Increased cerebral blood flow due to vasodilation	Confusion Delirium Coma
Renal system	Renin-angiotensin axis activation Increased erythropoietin production	Acute tubular necrosis

- Factors concomitants
  - Severitat de la hipòxia
  - Edat
  - Patologia de base
  - Velocitat d'instauració

B R O'Driscoll *Thorax* 2008;**63**(Suppl VI):vi1–vi68.

Definir un objectiu (target) de SaO2



Transport O2

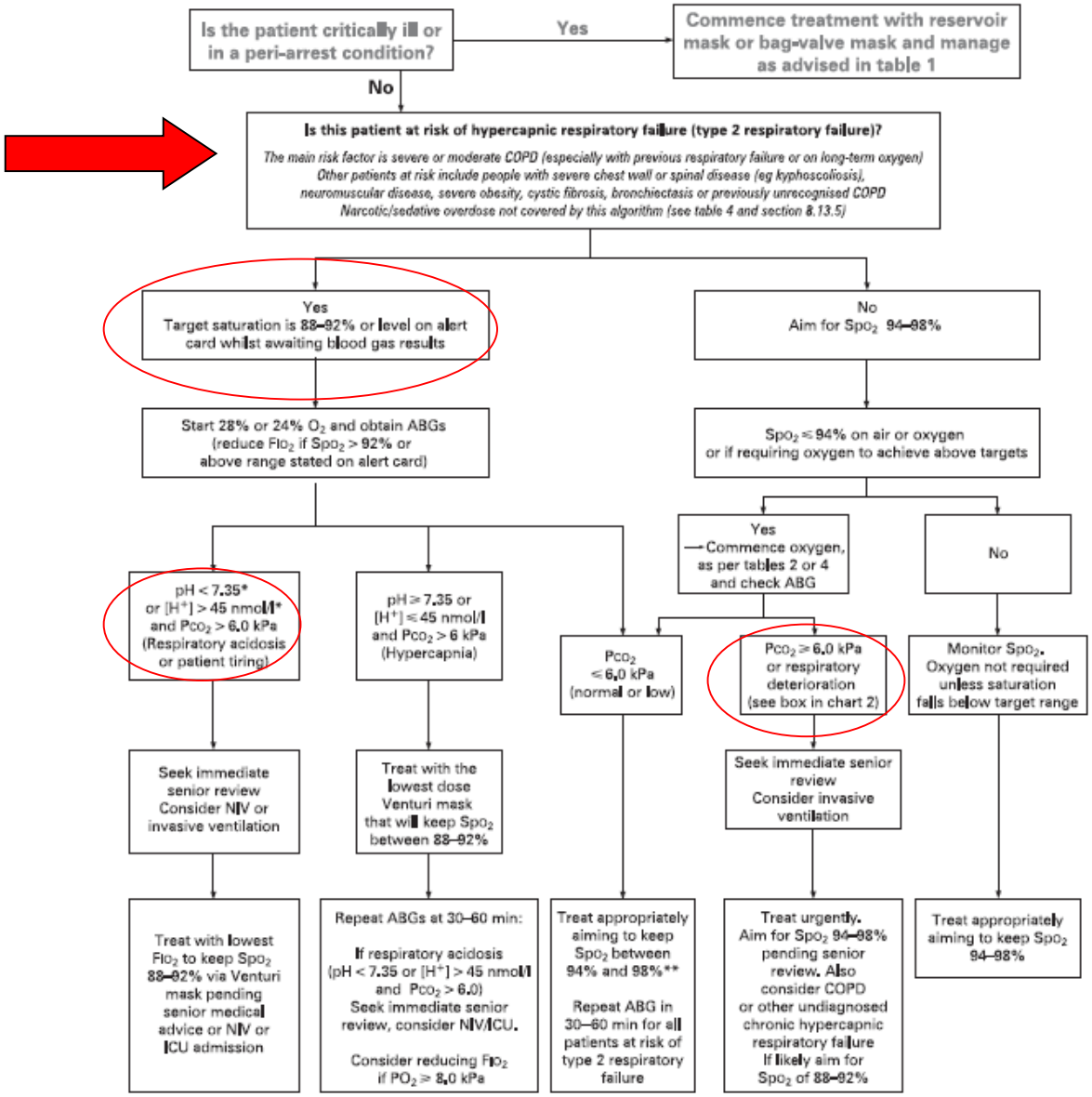
Situacions especials

Transport normal en situació aguda

Poden aparèixer efectes indesitjables

- Util en:
  - Intoxicació CO
  - Pneumotòrax sense drenatge

Algoritme de decisió d'oxigenoteràpia en situació aguda.

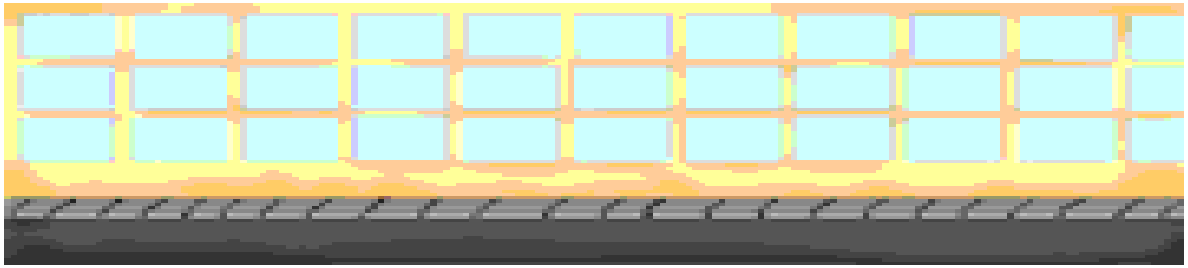


B R O'Driscoll *Thorax* 2008;**63**(Suppl VI):vi1–vi68.

Efectes adversos de l'oxigenoteràpia

Efectes sobre l'intercanvi pulmonar de gasos.

La hipercàpnia en resposta a l'administració d'O2 prehospitalària.



"Acidosi de l'ambulància": (20 %)

Plant PK, *Thorax*. 2000;55:550–554.

Probabilitat d'efectes adversos: OR=9.17 (95% CI =4.08–20.6) per hiperoxèmia
OR=2.16 (95% CI =1.11–4.20) per hipoxèmia

Cameron L. Postgrad Med J. 2012;88(1046):684–689.

An audit of hypoxaemia, hyperoxaemia, hypercapnia and acidosis in blood gas specimens (n=3524)

TABLE 1 Summary of blood gas results grouped according to oxygen saturation levels

Oxygen saturation range	Samples	Hypercapnia[#]	Uncompensated respiratory acidosis[¶]	Compensated respiratory acidosis[+]	Uncompensated metabolic acidosis[§]
>98%	1458 (41.3)	305 (21)	83 (6)	161 (11)	111 (8)
94–98%	1291 (36.6)	294 (23)	65 (5)	167 (13)	90 (7)
92.1–93.9%	237 (6.7)	85 (36)	14 (6)	58 (24)	15 (6)
88–92%	288 (8.2)	119 (41)	35 (12)	61 (21)	17 (6)
80–87.9%	154 (4.3)	76 (49)	18 (12)	41 (27)	6 (4)
70–79.9%	53 (1.5)	40 (75)	12 (23)	21 (40)	1 (2)
60–69.9%	22 (0.6)	14 (64)	6 (27)	4 (18)	4 (18)
<60%	21 (0.6)	15 (71)	6 (29)	5 (24)	2 (10)
Total	3524 (100)	948 (26.9)	239 (6.8)	518 (14.7)	246 (7.0)

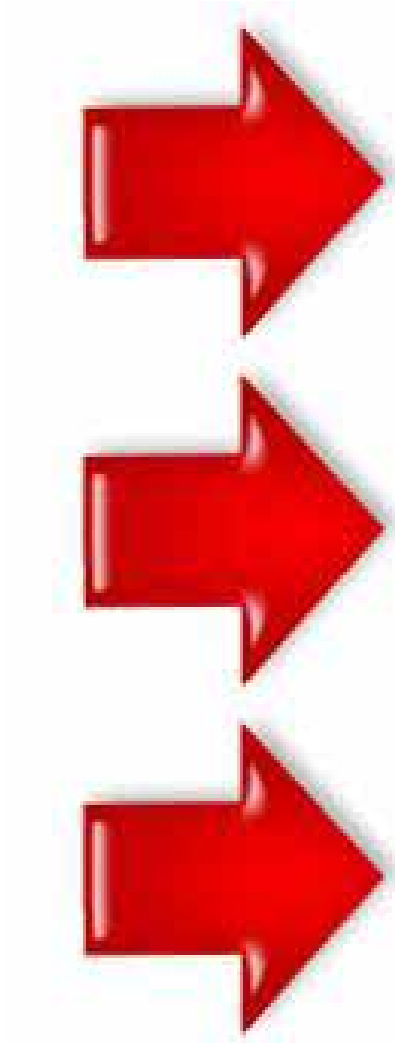
- La hipoxèmia severa va ser poc freqüent
- Hiperoxèmia en 41 % de pacients
- Hipercàpnia en 27 %

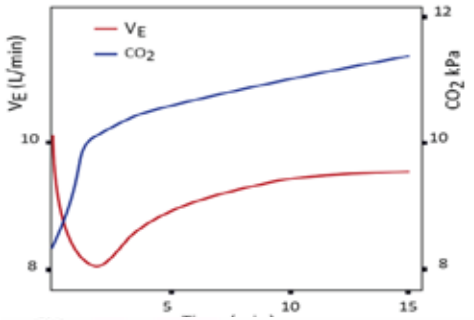
O'Driscoll Eur Respir J. 2012; 39: 219-21 .

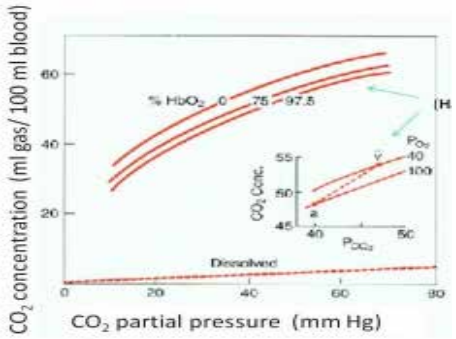
Causes hipercàpnia secundària.

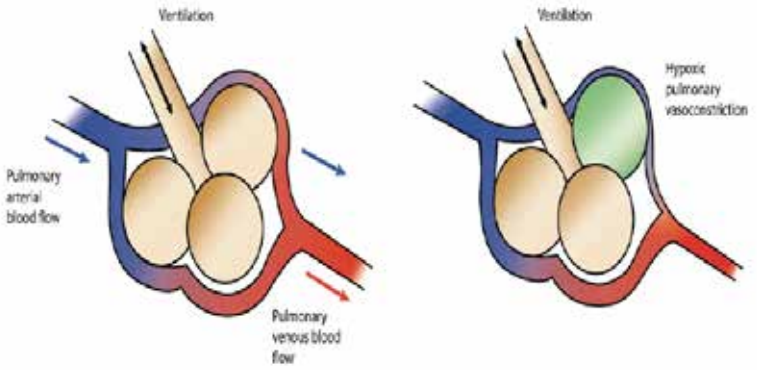
PaCO2

- Disminució impuls ventilatori
- Efecte Haldane
- Alteracions V/Q









- Patients with exacerbations of COPD are at risk of hypercapnic (type 2) respiratory failure with respiratory acidosis. **[Evidence level IIa]**
- The risk of respiratory acidosis in patients with hypercapnic respiratory failure is increased if the arterial oxygen tension is above 10.0 kPa due to previous excessive oxygen use. **[Evidence level IIa]**
- These patients with chronic lung disease are usually "acclimatised" to living with an oxygen saturation which may be in the high 80s or low 90s and there is not likely to be any benefit from increasing the saturation above these levels during acute illness. **[Evidence level III]**

Pacients en risc d'hipercàpnia secundària a la oxigenoteràpia

Oxygen alert card

Name: _____

I am at risk of type II respiratory failure with a raised CO_2 level.

Please use my % Venturi mask to achieve an

oxygen saturation of _____% to ____% during exacerbations

Use compressed air to drive nebulisers (with nasal oxygen at 2 l/min).
If compressed air not available, limit oxygen-driven nebulisers to 6 minutes.

B R O'Driscoll *Thorax* 2008;**63**(Suppl VI):vi1–vi68.

Predicting Nocturnal Hypoventilation in Hypercapnic Chronic Obstructive Pulmonary Disease Patients Undergoing Long-Term Oxygen Therapy

Julia Tarrega[a] Antonio Anton[b] Rosa Guell[b] Mercedes Mayos[b, e, f]
Daniel Samolski[b] Sergi Marti[c] Eva Farrero[d] Enric Prats[d] Joaquin Sanchis[b]

Table 1. Clinical characteristics and pulmonary function variables in both subgroups of patients (NHV and non-NHV)

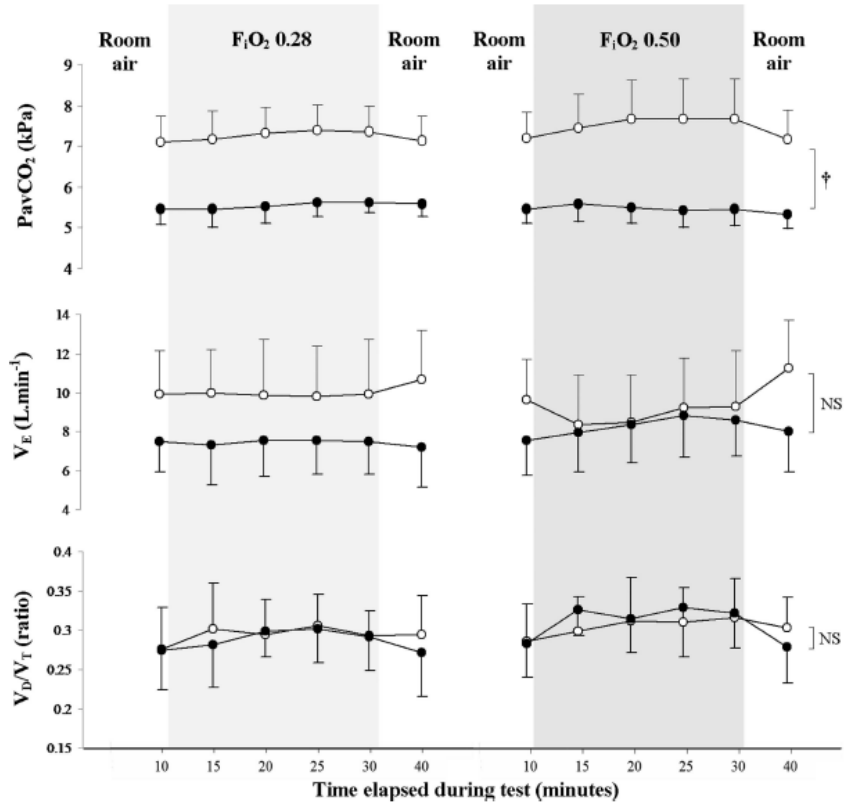
	Total	NHV (n = 17)	Non-NHV (n = 63)	p
Age, years	68 ± 7	67 ± 7	68 ± 7	0.463
BMI	27.5 ± 4.5	30 ± 4	27 ± 4	0.004
FVC, % ref.	49 ± 12	50 ± 15	49 ± 11	0.638
FEV_1, % ref.	23 ± 7	25 ± 7	23 ± 7	0.169
FEV_1/FVC, %	34 ± 9	37 ± 6	34 ± 9	0.205
TLC, % ref.	111 ± 23	109 ± 21	112 ± 23	0.774
RV, % ref.	209 ± 64	197 ± 62	211 ± 65	0.494
DLCO, %	46 ± 20	57 ± 22	44 ± 19	0.027
KCO, %	70 ± 30	89 ± 19	66 ± 30	0.028
PI_{max}, % ref.	77.5 ± 30	79 ± 51	77 ± 29	0.942
PE_{max}, % ref.	77 ± 34	73 ± 49	78 ± 33	0.856
MVV, % ref.	25 ± 7	28 ± 6	24 ± 7	0.165
CoHb, %	1.84 ± 1.34	2.5 ± 2	1.7 ± 1	0.149

21 % pacients presentaven NHV
Factors associats:
- **BMI més alt.**
- **Menor PaO2 amb O2**
- Increment Hb
- Millor DLCO.

Respiration 2011;82:4–9

Moderate concentrations of supplemental oxygen worsen hypercapnia in obesity hypoventilation syndrome: a randomised crossover study

Carly Ann Hollier,[1,2] Alison Rosemary Harmer,[2] Lyndal Jane Maxwell,[3] Collette Menadue,[1] Grant Neville Willson,[4] Gunnar Unger,[5] Daniel Flunt,[1] Deborah Ann Black,[2] Amanda Jane Piper[1,5]



What is the bottom line?

- In OHS, breathing moderate concentration supplemental oxygen for 20 min worsened hypercapnia and induced acidaemia due to hypoventilation and a worsening of V_D/V_T.

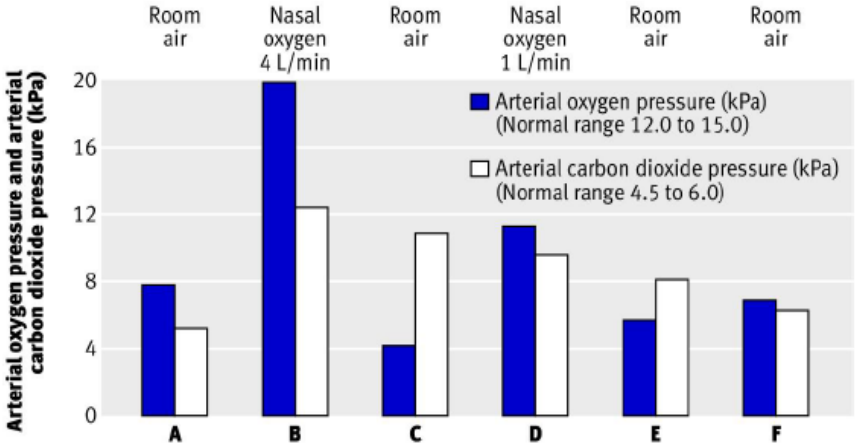
Hollier CA, *et al*. *Thorax* 2014;**69**:346–353.

Altres patologies que poden cursar amb hipoxèmia i risc d'hipercàpnia

- In the initial management of musculoskeletal and neurological disorders with acute respiratory failure, aim at an oxygen saturation of 88–92%. Many such patients will be suitable for non-invasive ventilation. **[Grade D]**

- In the initial management of the obesity-hypoventilation syndrome with acute exacerbation, aim at an oxygen saturation of 88–92%. **[Grade D]**
- Non-invasive ventilation should be considered for all of the above groups of patients if the pH is <7.35 or $[H^+]$ >45 nmol/l. **[Grade C]**

La “hipoxèmia de rebot”

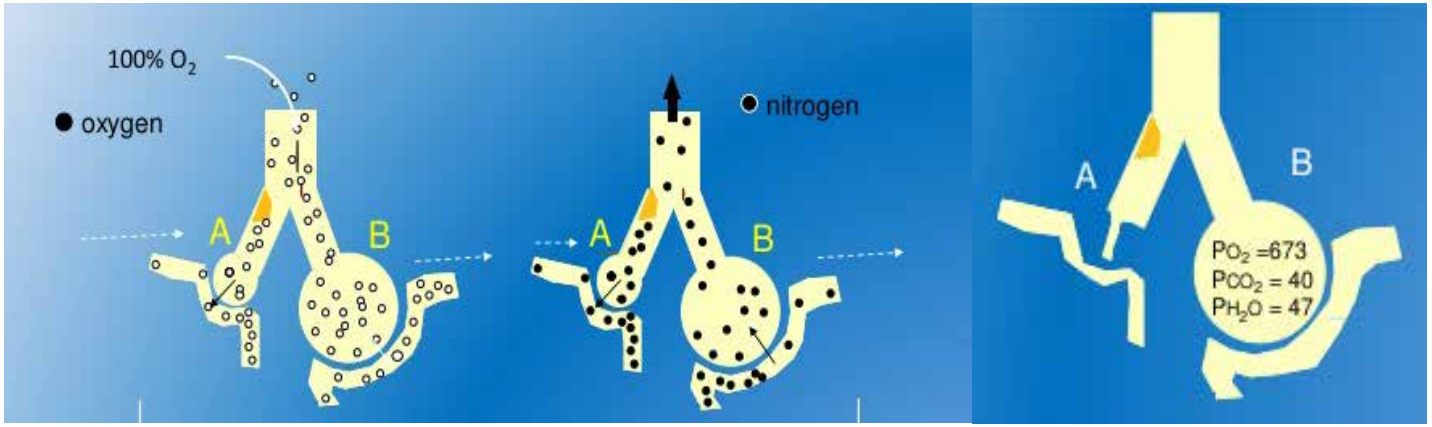


Time	A: 15 months before admission	B: Emergency callout	C: 30 minutes after B	D: 14 hours after C	E: 2 hours after D	F: 2 weeks after E
pH	7.40	7.22	7.28	7.31	7.36	7.41
Bicarbonate	24.3	36.9	37.0	35.0	33.2	29.5
Aterial oxygen saturation	90.6%	99.2%	59.6%	96.4%	79.2%	86.5%

- Sudden cessation of supplementary oxygen therapy can cause rebound hypoxaemia with a rapid fall in oxygen tension to below the tension that was present prior to the commencement of supplementary oxygen therapy. **[Evidence level III]**

Kane BMJ. 2011 Mar 31;342:d1557

Atelectàssies per resorció o desnitrogenació



N2=78 %
O2=21%

The New England
Journal of Medicine

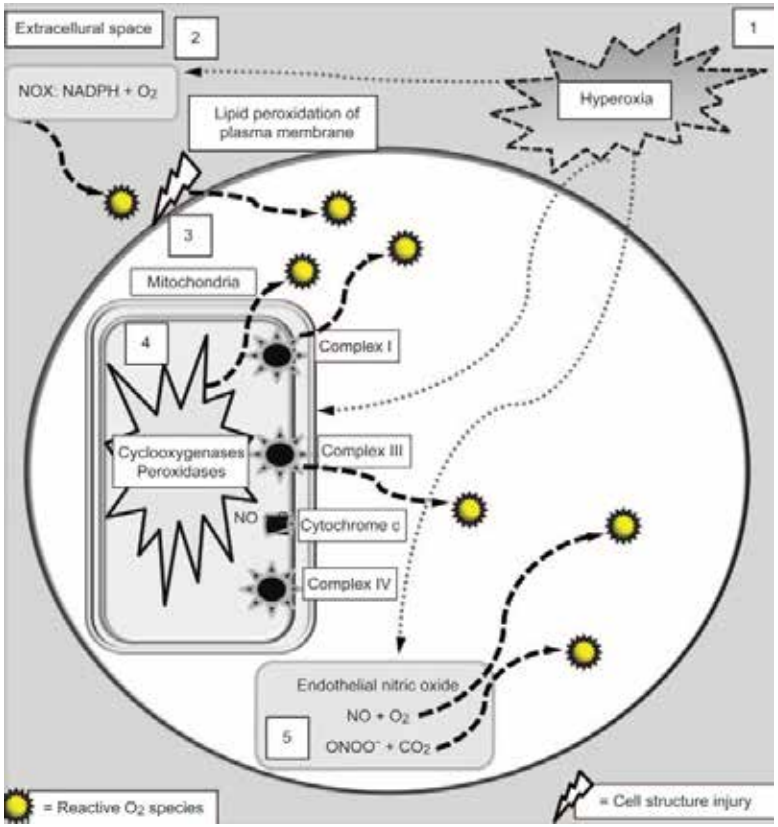
Volume 269 NOVEMBER 7, 1963 Number 19

IMPAIRED OXYGENATION IN SURGICAL PATIENTS DURING GENERAL ANESTHESIA WITH CONTROLLED VENTILATION*

A Concept of Atelectasis

H. H. Bendixen, M.D.,† J. Hedley-Whyte, M.B., B.Chir.,‡ and M. B. Laver, M.D.§

Efectes metabòlics de la hiperòxia: Toxicitat directa per O2.

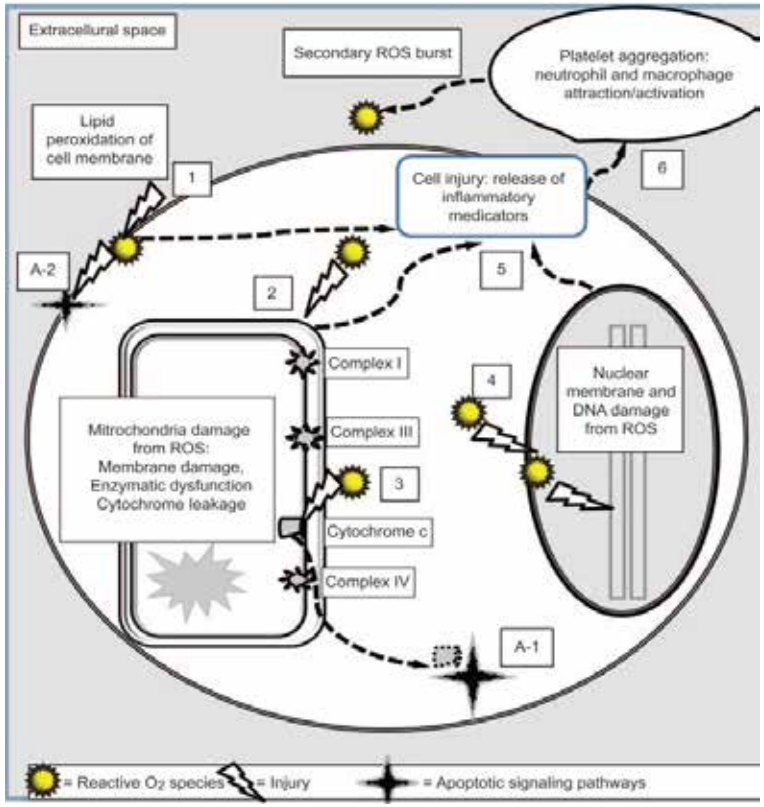


- Anió superòxid (O2–)
- Peròxid d'hidrògen (H2O2)
- Radicals hidroxil (OH–)
- Anió peroxinitril (ONOO–).

Producció de Reactive Oxygen Species (ROS)

Kallet et al. Respir Care 2013;58(1):123–140

Toxicitat directa per O2.



Conseqüències de la producció de ROS

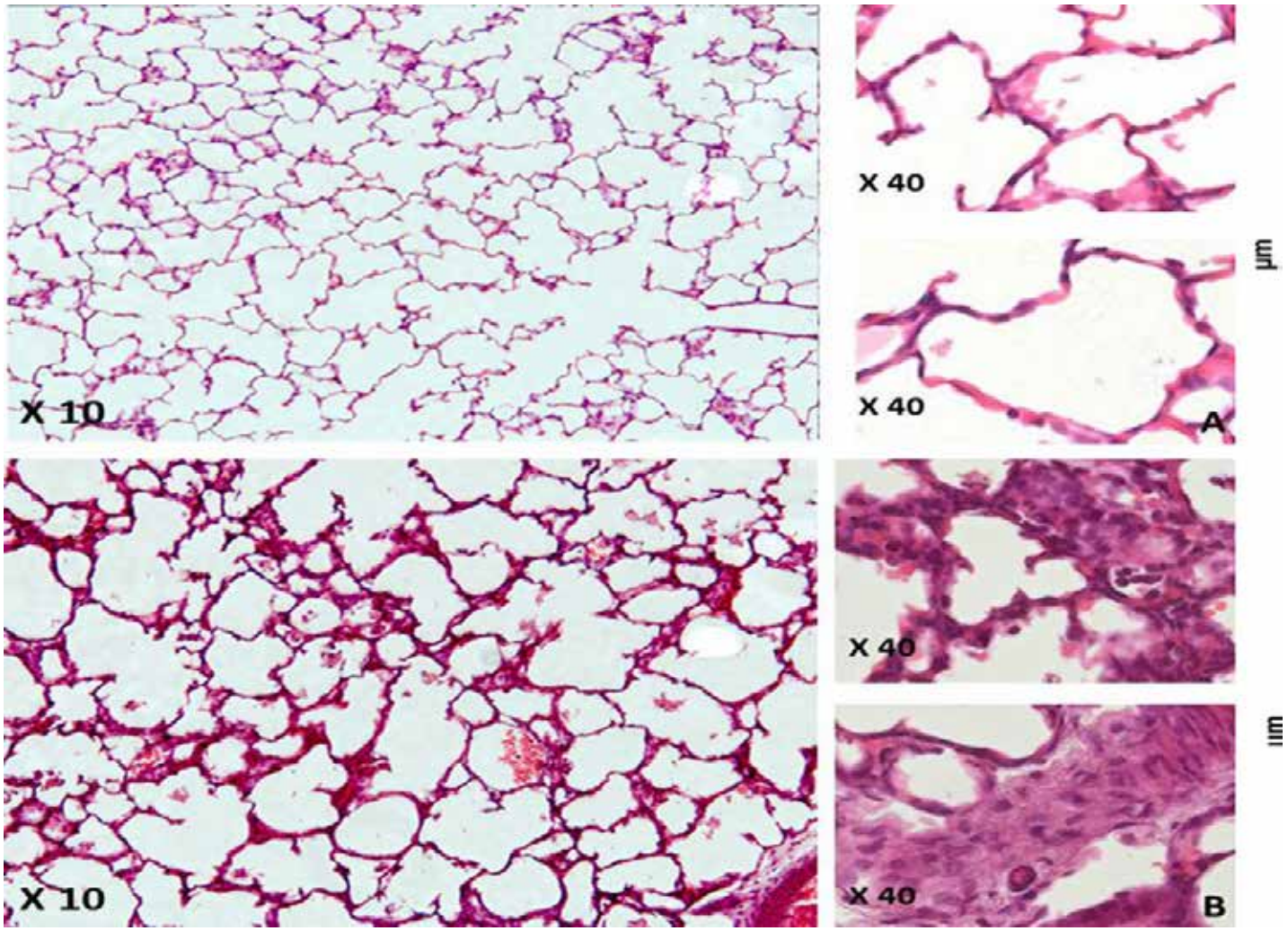
Ruptura de membranes celulars

Disfunció enzimàtica

Lesió mitocondrial I del DNA

Mort celular

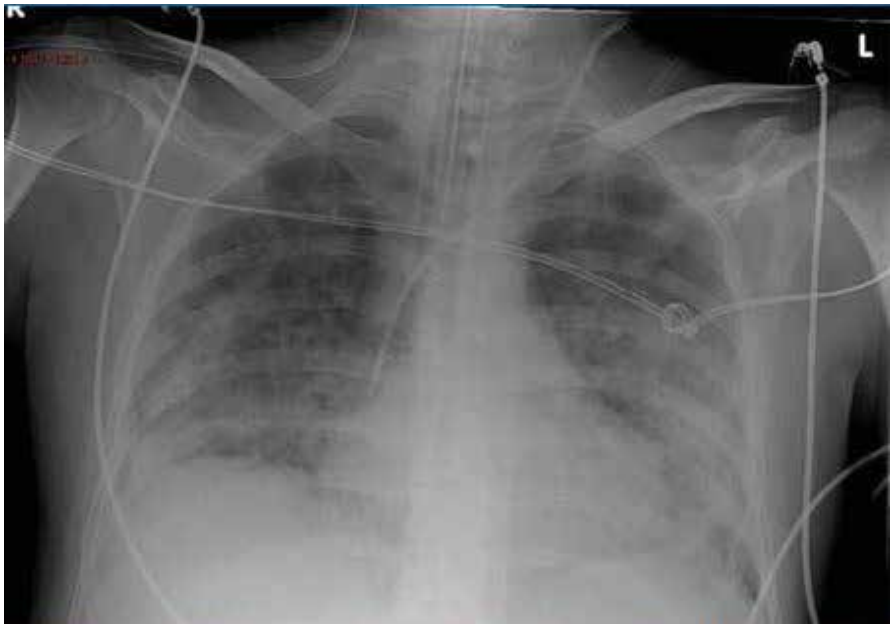
Hematoxylin and eosin (H&E) staining and morphometry [linear intercept (Lm) − mean wall transection length (Lmw)] of the right lung.



Jute Richter et al. Am J Physiol Lung Cell Mol Physiol 2014;306:L277-L283

AMERICAN JOURNAL OF PHYSIOLOGY
Lung Cellular and Molecular Physiology

Lesió pulmonar aguda per oxigen



- Factors coadjuvants
  - Ventilació mecànica
  - Infeccions
  - Traumatisme toràcic
  - Predisposició genètica
  - Fàrmacs

Oxygen Therapy in Critical Care: A Double Edged Sword

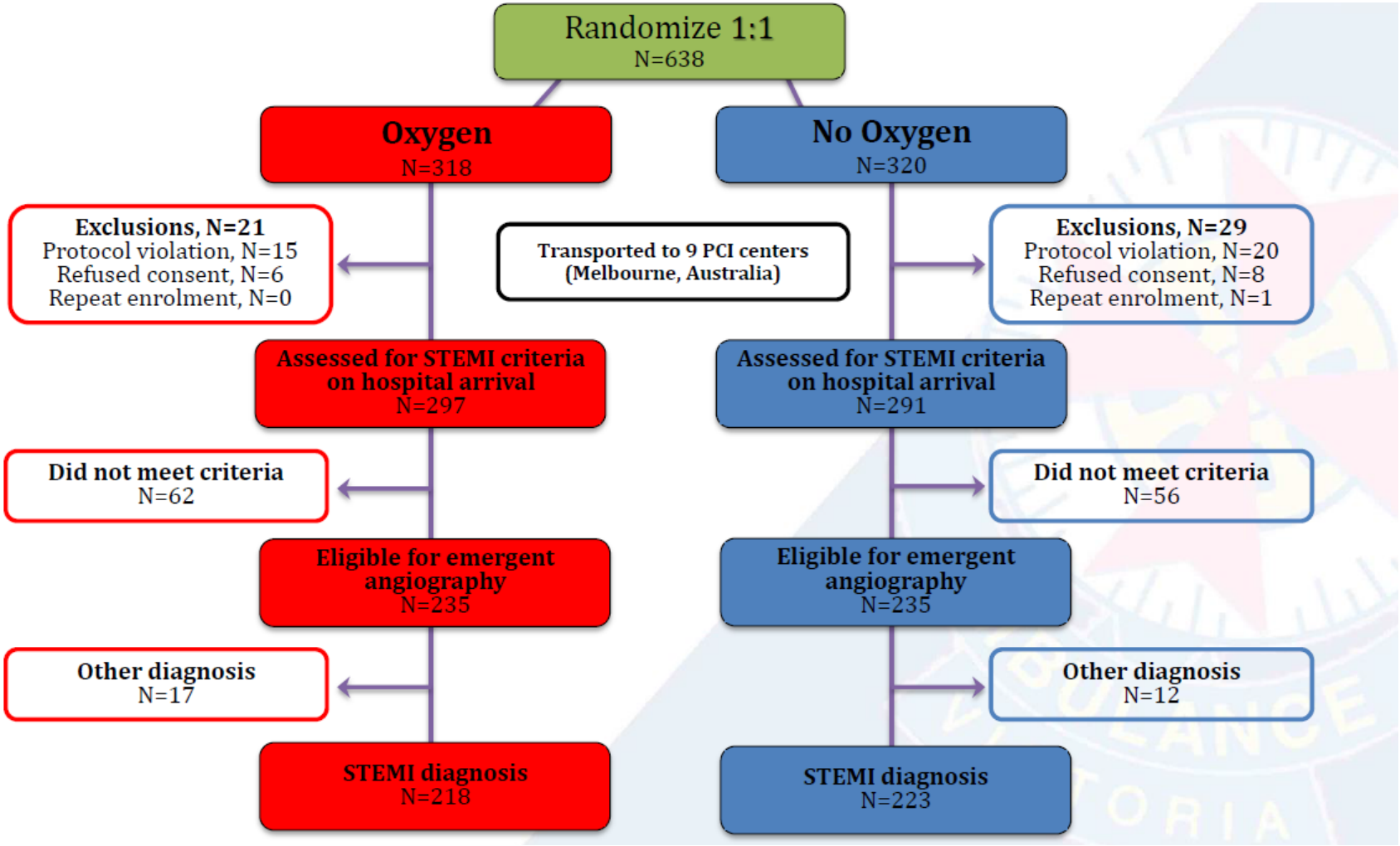
Isidro Prieto del Portillo, Susana Temprano Vázquez, Jesús Barea Mendoza, Rubén Viejo Moreno

Health, 2014, 6, 2035-2046

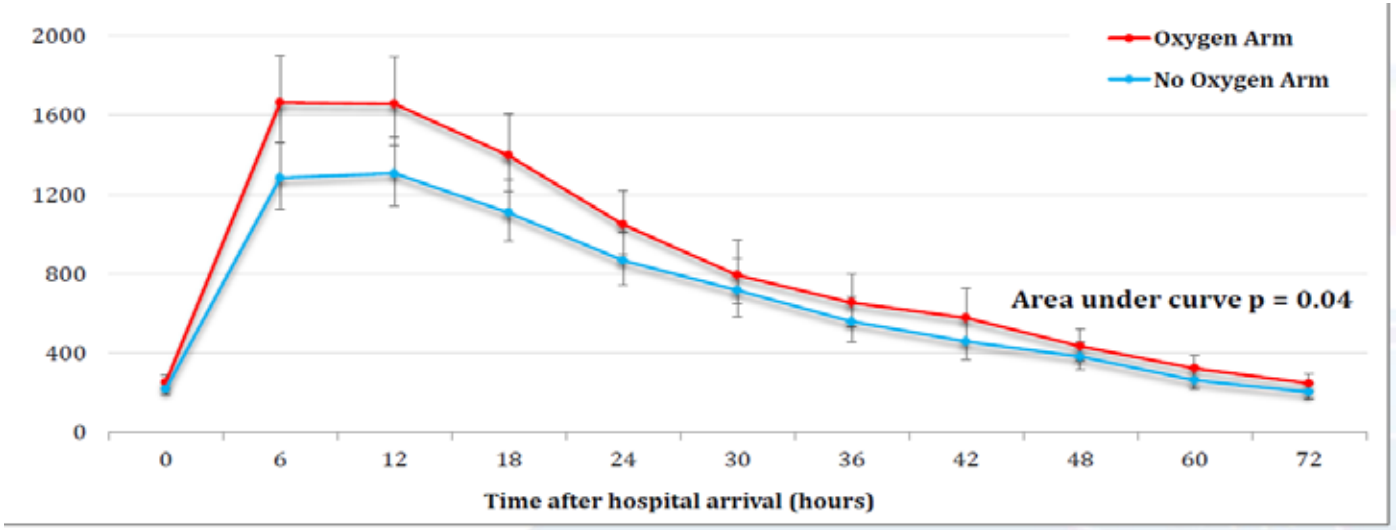
Altres efectes adversos de la hiperòxia

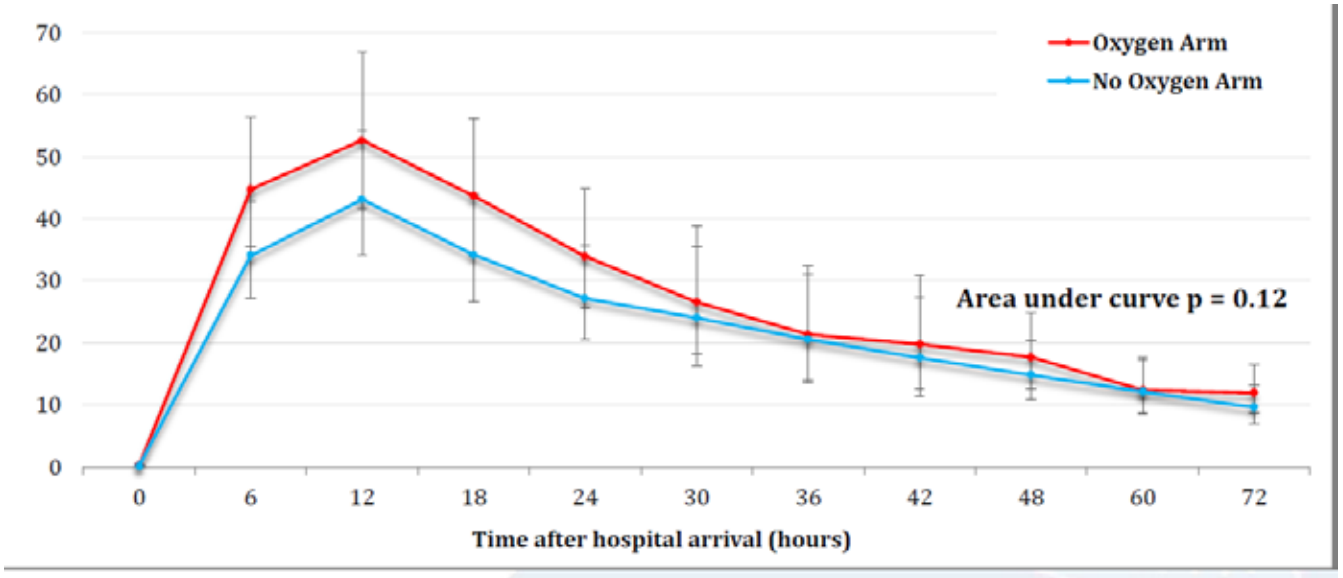
	Hyperoxia	
	Effects	**Risks**
Respiratory system	Decreased ventilation (minimal)	Worsened ventilation/ perfusion matching Absorption atelectasis
Cardiovascular system		Myocardial ischaemia (in context of decreased haematocrit) Reduced cardiac output Reduced coronary blood flow Increased blood pressure Increased peripheral resistance
Metabolic system	Decreased 2,3-DPG Decreased CO_2 carriage (Haldane effect)	Increased reactive oxygen species
Neurological system	Decreased cerebral blood flow	
Renal system		Reduced renal blood flow

Estudi AVOID. Oxigen suplementari a IAM sense hipoxèmia



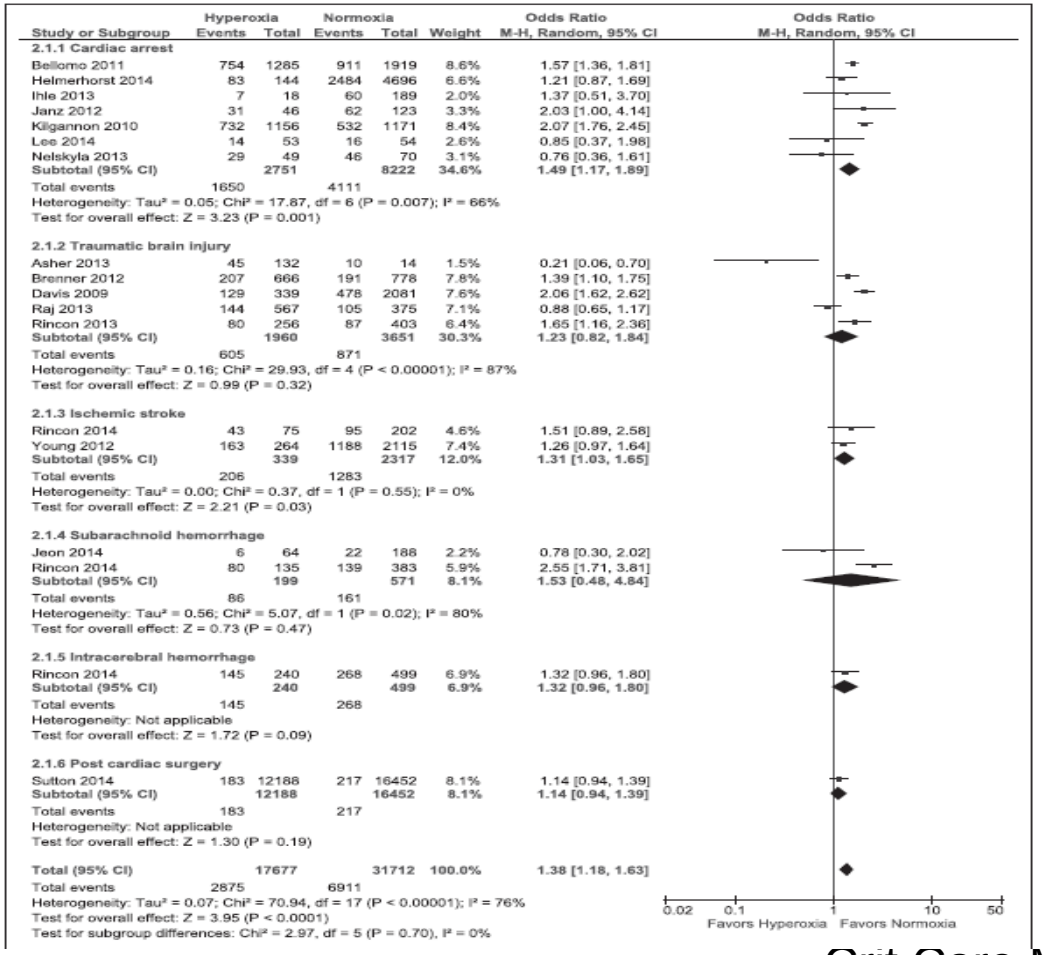
Evolució CK i mida de l'infart





Association Between Arterial Hyperoxia and Outcome in Subsets of Critical Illness: A Systematic Review, Metaanalysis, and Meta-Regression of Cohort Studies

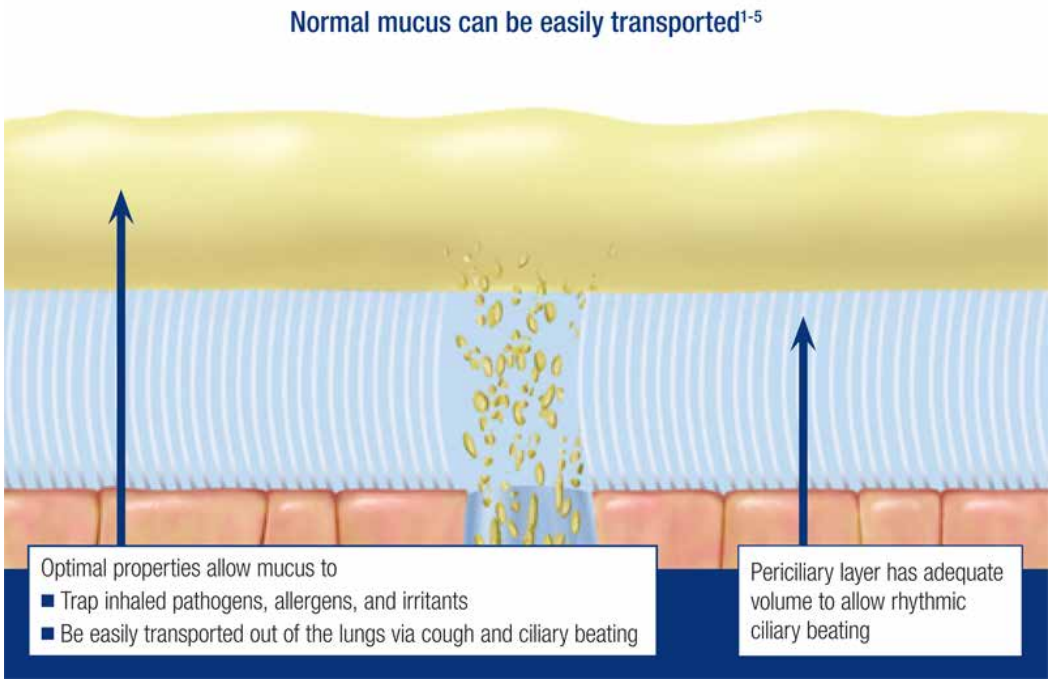
Hendrik J. F. Helmerhorst, MD[1,2]; Marie-José Roos-Blom, MSc[3]; David J. van Westerloo, MD, PhD[1]; Evert de Jonge, MD, PhD[1]



Crit Care Med 2015 (April, in press)

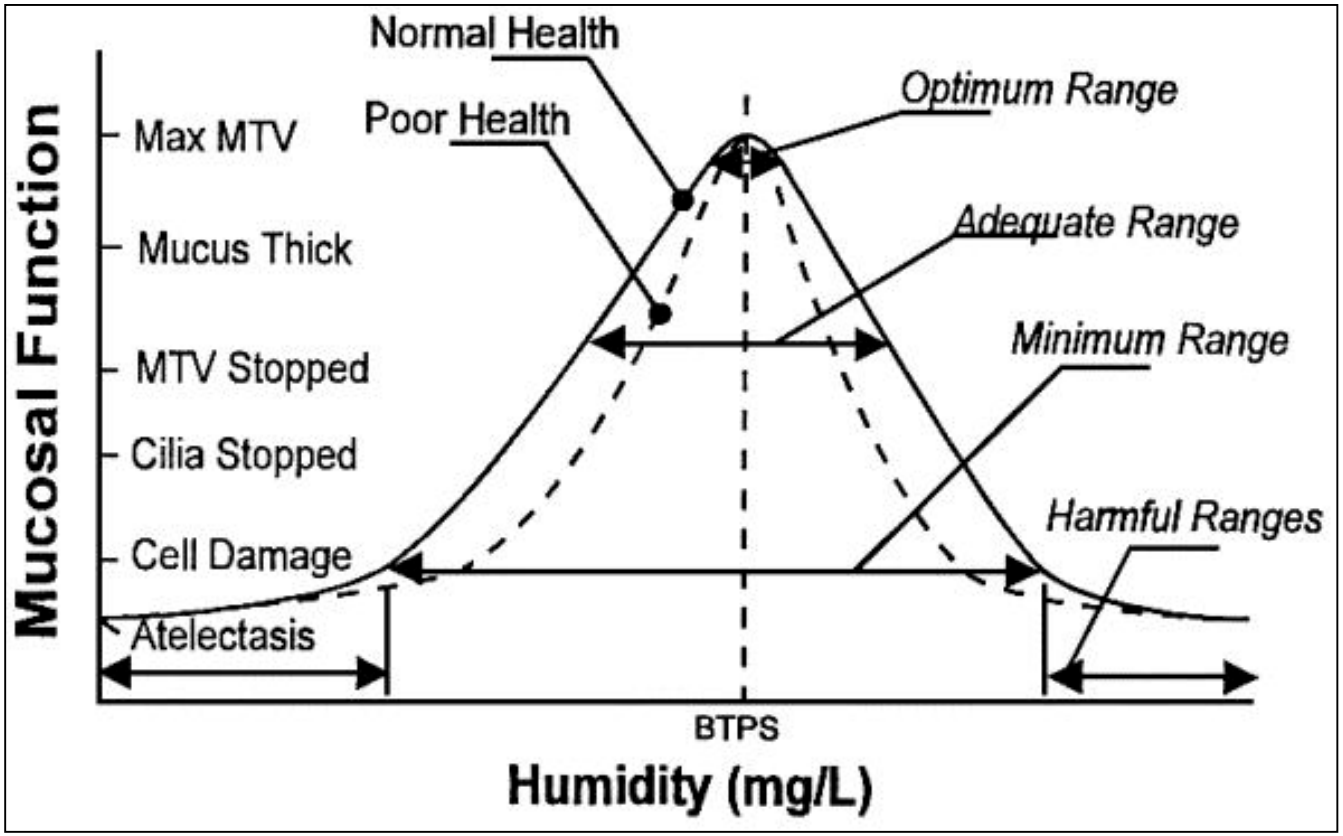
Associats a fluxe de gas no condicionat.

Condicions òptimes de la via aèria.



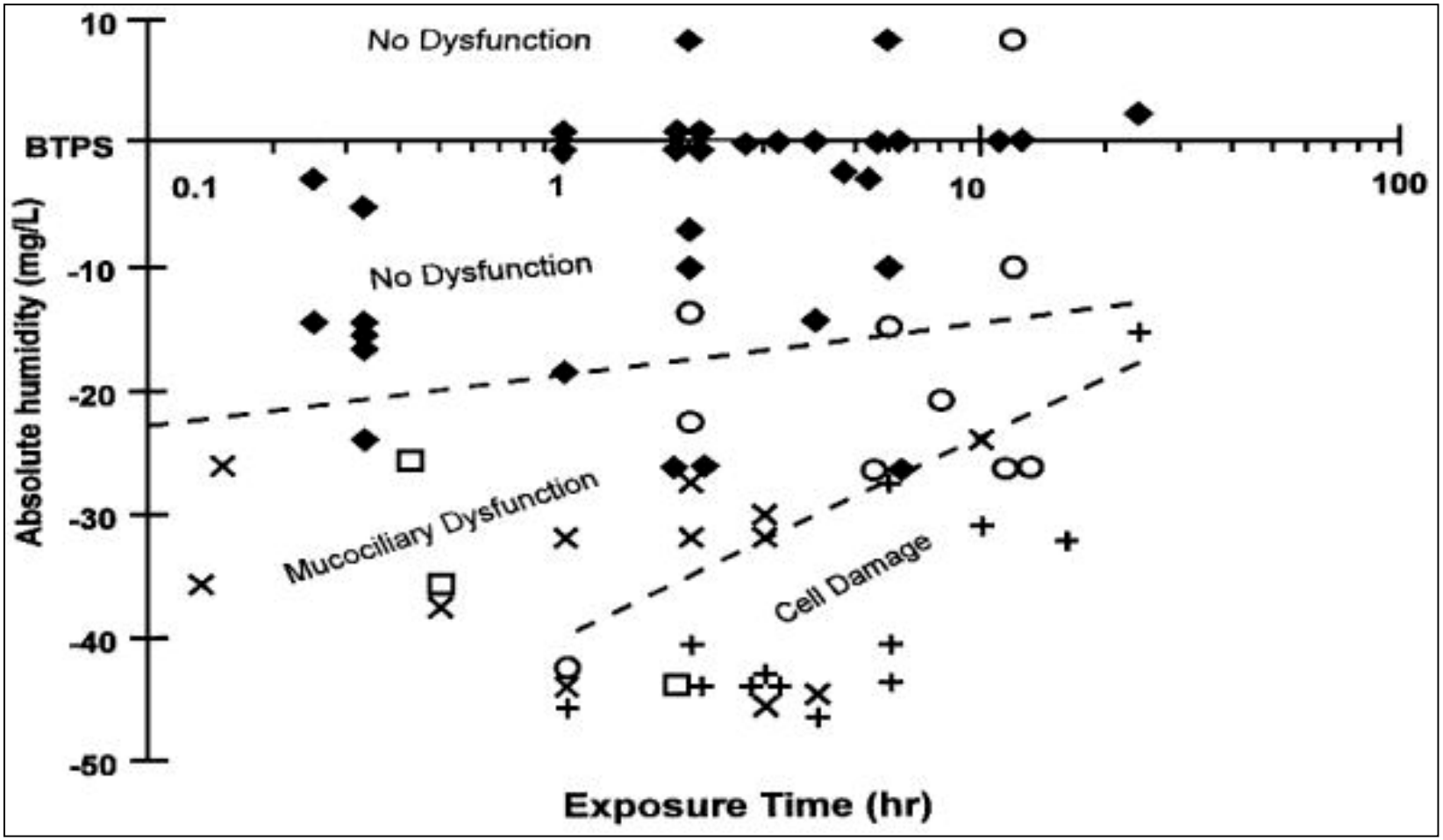
- 37 °C
- 44 mg/L HA
- 100% HR

Relationship between the humidity and temperature of inspired gas and the function of the airway mucosa



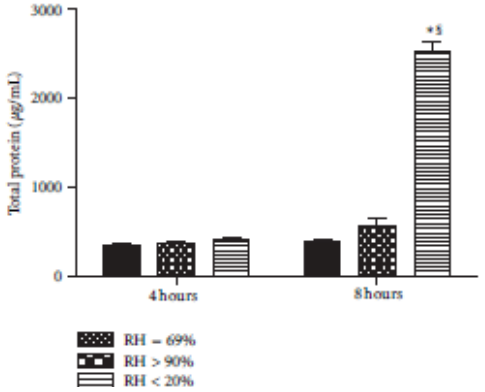
Williams et al. Crit Care Med 1996; 24:1920-1929

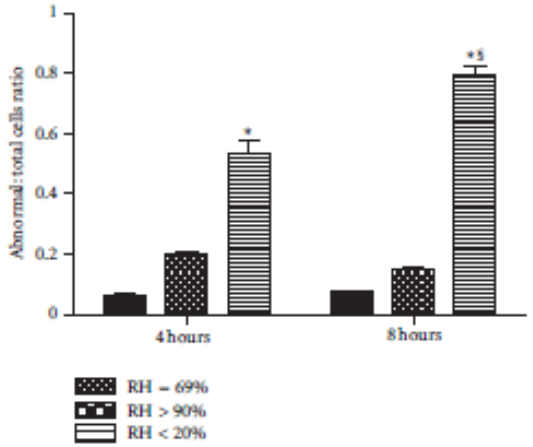
Relationship between the humidity and temperature of inspired gas and the function of the airway mucosa

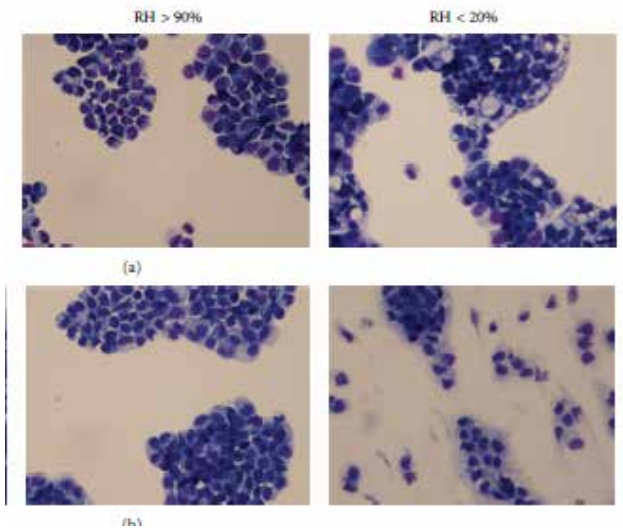


Williams et al. Crit Care Med 1996; 24:1920-1929

The Effects of Gas Humidification with High-Flow Nasal Cannula on Cultured Human Airway Epithelial Cells



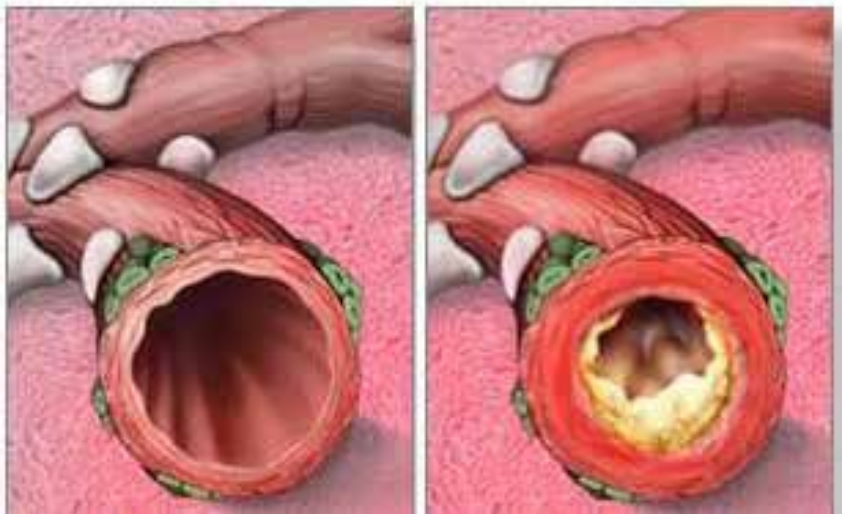




Chidekel Pulm Med 2012

Quantitats de gas no condicionat en màscara d'efecte Venturi.

FiO2	L/min
0.26	24
0.31	30
0.35	36
0.5	44



L'oxígen com a medicació. Conclusions

- Té unes propietats terapèutiques
  - Fixar un "objectiu de SpO2"
- Té uns efectes secundaris
  - Associats a fisiologia de O2/CO2
  - De tipus metabòlic (hiperòxia)
  - Per "l'excipient" (gas)
  - Es requereix ajust en determinades condicions
  - Pot requerir una retirada gradual