Insufficienza Respiratoria Acuta: gestione clinica e terapeutica in ambito internistico

Paolo Groff

Pronto Soccorso

Azienda Ospedaliera di Perugia

Insufficienza Respiratoria Acuta: gestione clinica e terapeutica in ambito internistico

- Limiti del discorso
- Caso clinico 1
- Caso clinico 2
- Caso clinico 3
- Prognosi
- Monitoraggio

Limiti del discorso

- Insufficienza respiratoria
- Alterazione di una o entrambe le funzioni respiratorie di **OSSIGENAZIONE** e di **RIMOZIONE di CO_2** dal sangue venoso misto

Convenzionalmente definita da:

-$PaO_2 < 60$ mmHg in a.a.

-$PaCO_2 > 45$ mmHg in a.a.

-entrambe

Insufficienza Respiratoria

Tipo I
Tpo II
Tipo III

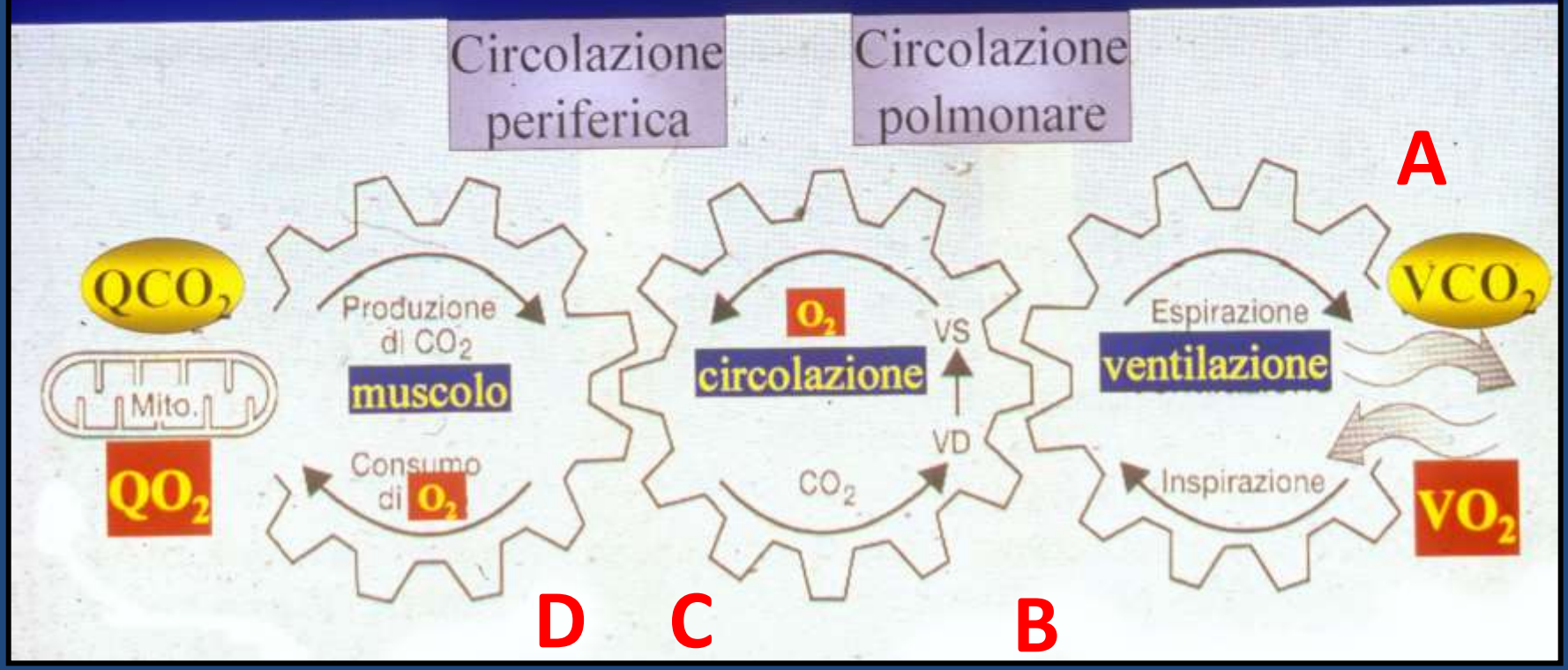
Acuta
Cronica
Acuta su Cronica

Cardiogenica
Acuta su
Cronica
De Novo

Back to the bases

La respirazione può essere, molto semplicemente, definita
come la funzione assicurante gli scambi gassosi tra
l'ambiente esterno e le cellule

Ventilazione
Circolazione
Respirazione tissutale



Fase tissulo-cellulare | **Fase circolatoria** | **Fase alveolo-capillare** | **Fase ventilatoria**

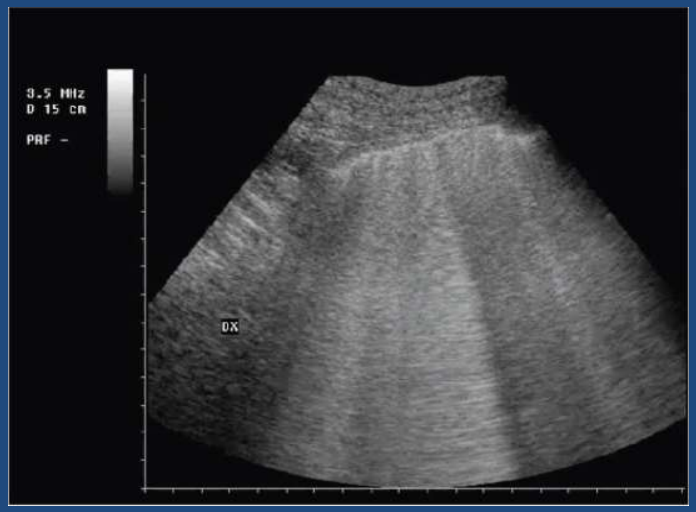
Caso clinico 1

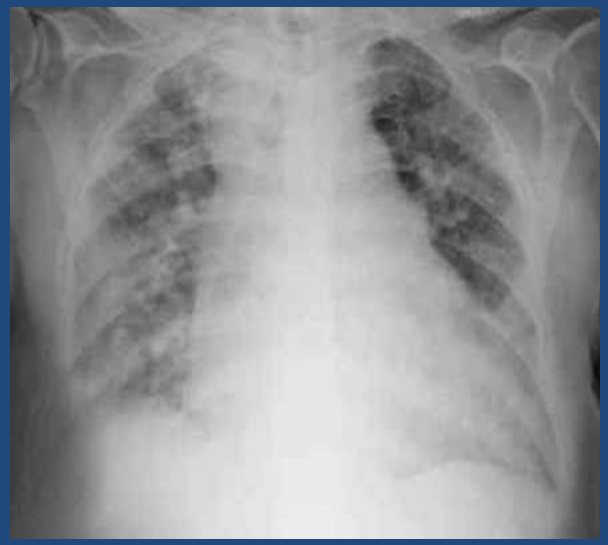
- Uomo, 75 anni
- Ipertensione arteriosa
- SCA (pregresso IMA inferiore), BBD

- Dispnea ad insorgenza notturna.

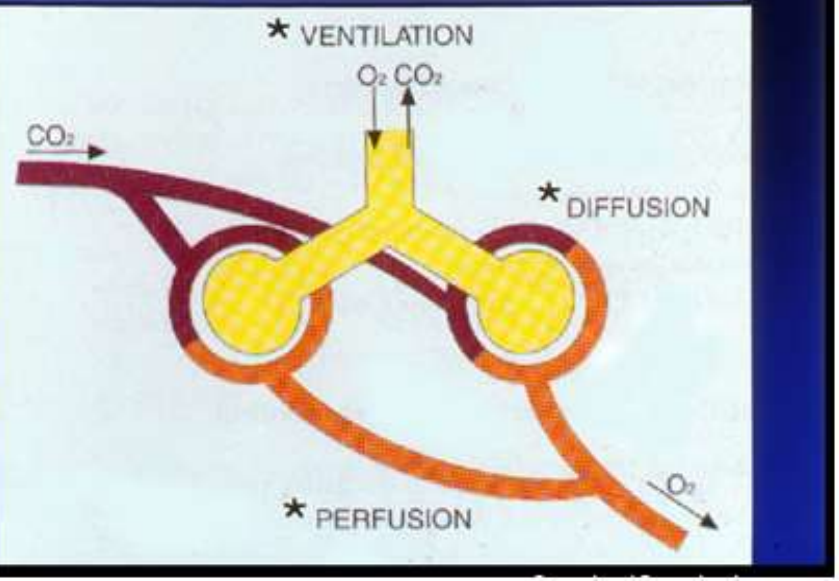
Caso Clinico 1

- In PS:
  - PA:175/110, FC: 110/min, FR: 34/min, SpO_2:80% AA→85% con VM 50%, T: 37°; Kelly 3
  - EGA: pH=7,25, PaO_2=52, $PaCO_2$=65, HCO_3^-= 16,2

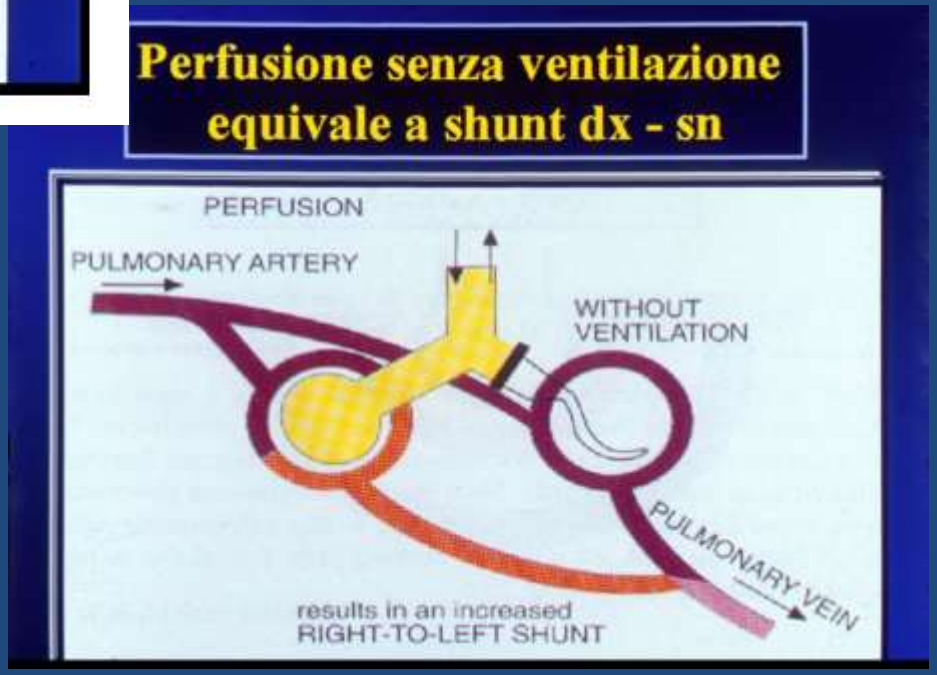




Le tre componenti degli scambi gassosi

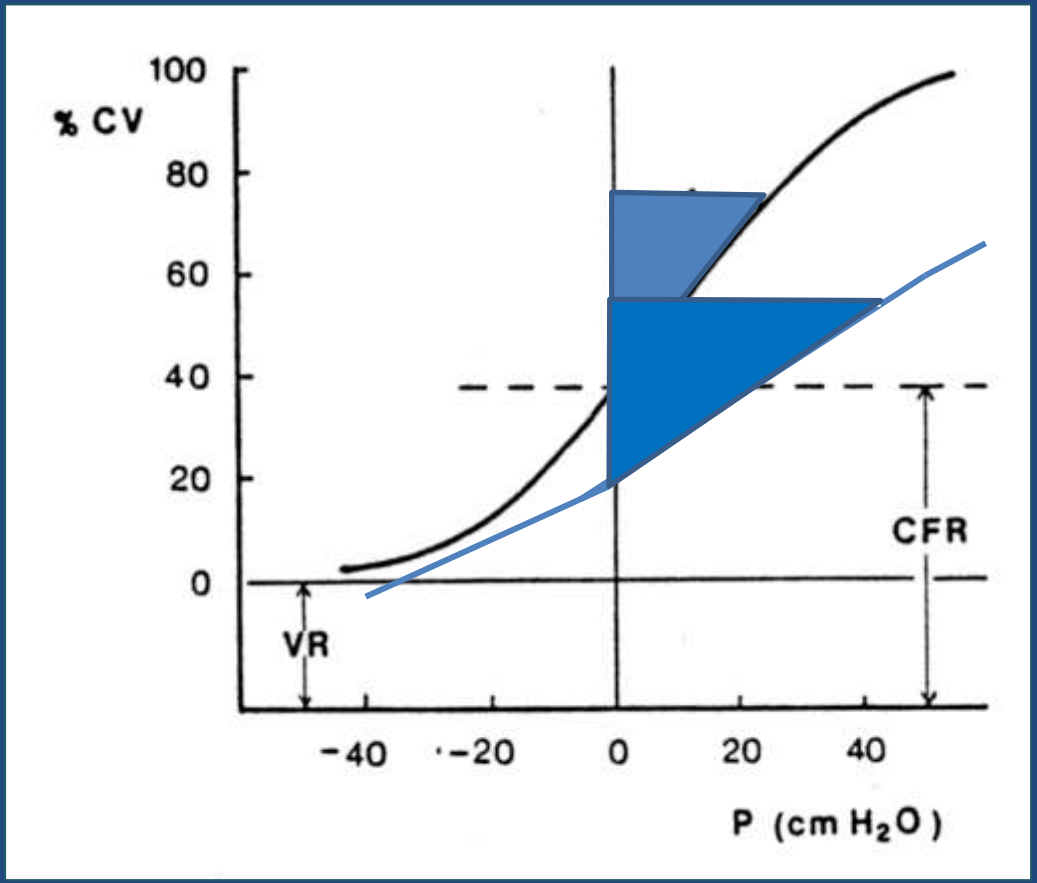


Perfusione senza ventilazione equivale a shunt dx - sn



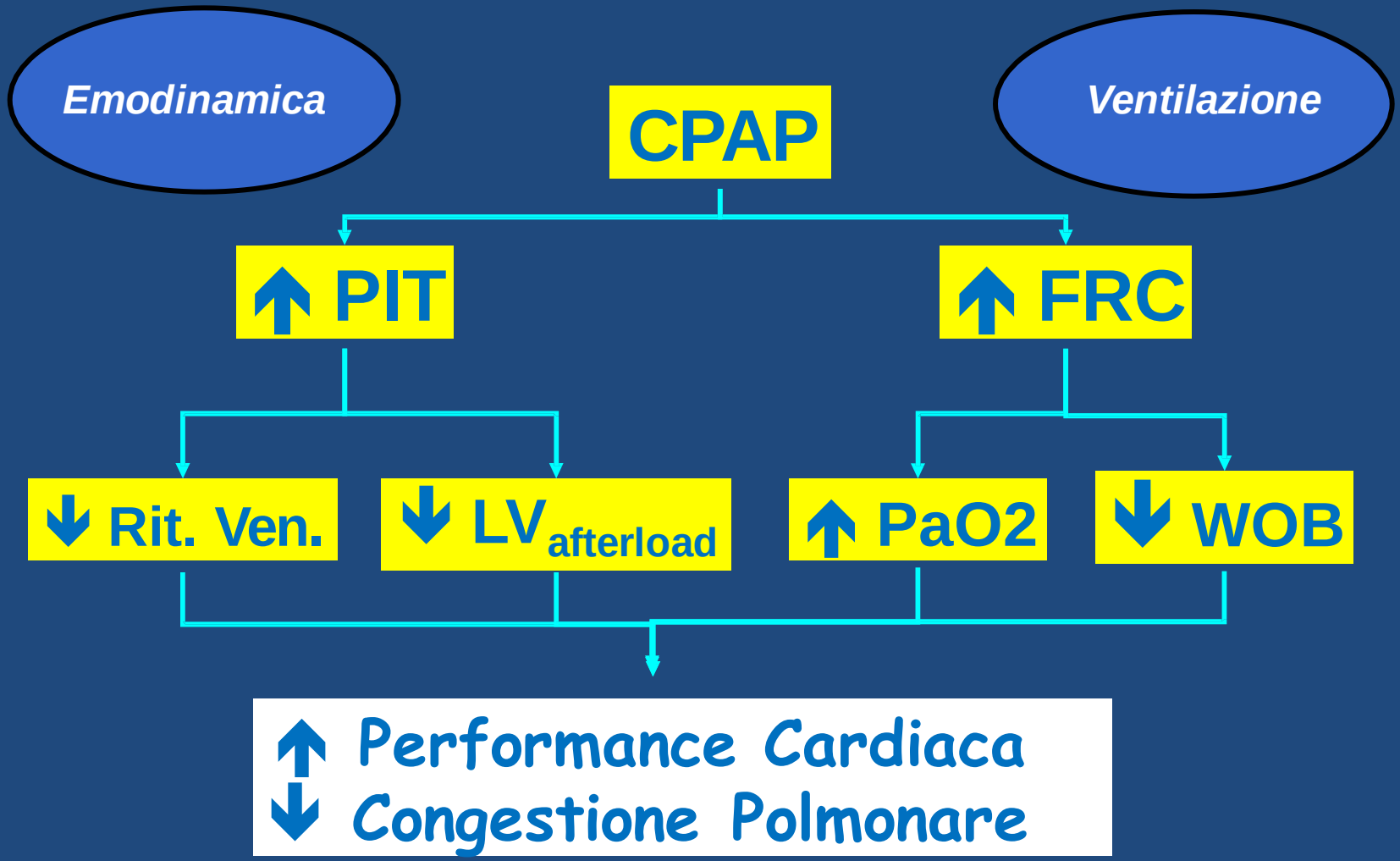
Lavoro Respiratorio, normale

Lavoro Respiratorio, EPAc



WOB = F X S = P X V

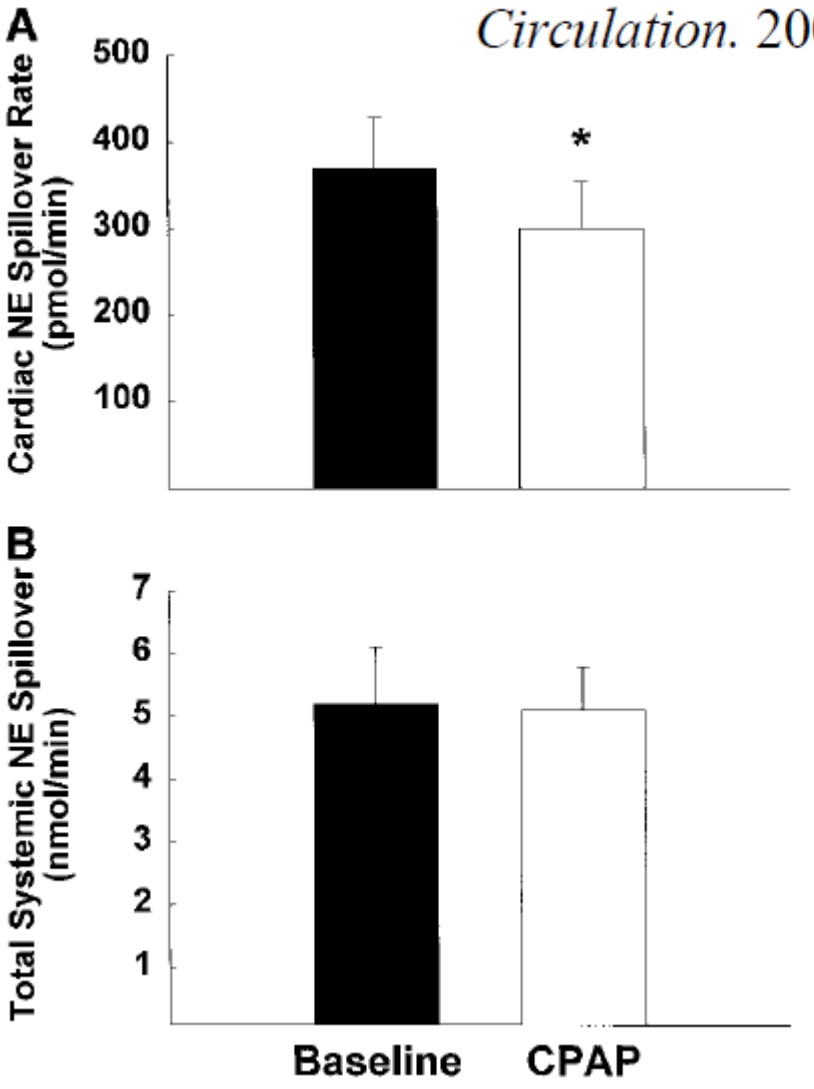
RAZIONALE DELLA CPAP NELL'EPAc



Acute Effects of Continuous Positive Airway Pressure on Cardiac Sympathetic Tone in Congestive Heart Failure

David M. Kaye, MBBS, PhD; Darren Mansfield, MBBS; Ann Aggarwal, MBBS;
Matthew T. Naughton, MDBS; Murray D. Esler, MBBS, PhD

Circulation. 2001;103:2336-2338



Continuous positive airway pressure decreases myocardial oxygen consumption in heart failure

David M. KAYE*†, Darren MANSFIELD‡ and Matthew T. NAUGHTON‡
*Wynn Department of Metabolic Cardiology, Baker Heart Research Institute, St Kilda Rd Central, Melbourne, VIC 8008, Australia, †Department of Cardiovascular Medicine, Alfred Hospital, Commercial Rd, Prahran, VIC 3181, Australia, and ‡Department of Respiratory Medicine Alfred Hospital, Commercial Rd, Prahran, VIC 3181, Australia

Clinical Science (2004) **106**, 599–603

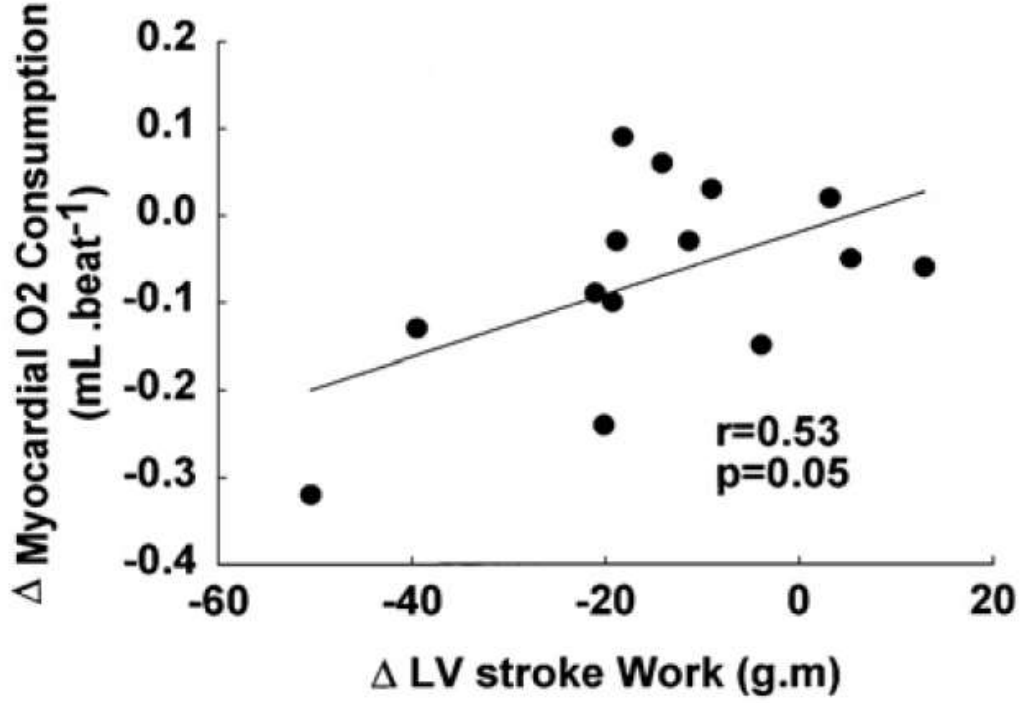


Figure 1 Relationship between the CPAP-mediated change in LV stroke work and myocardial $\dot{V}O_2$

Noninvasive Ventilation in Patients With Acute Cardiogenic Pulmonary Edema

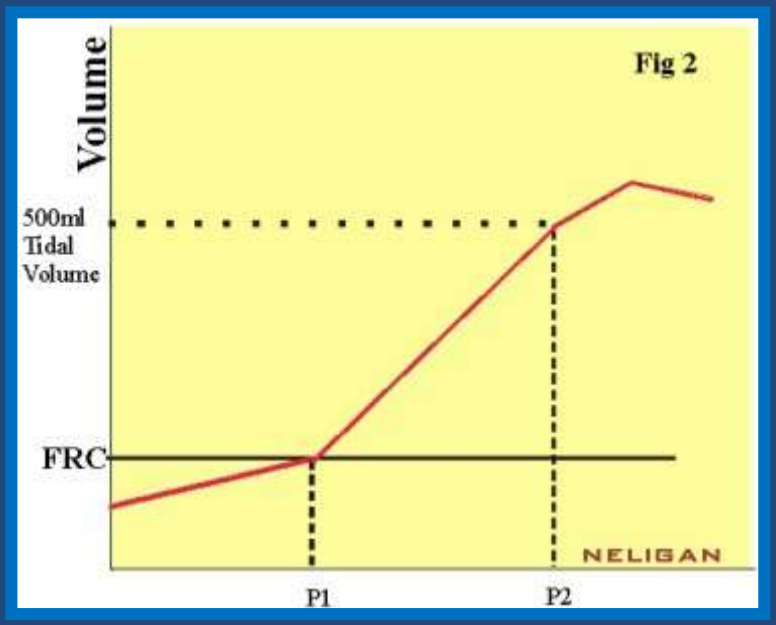
Sangeeta Mehta MD FRCPC, Abdul Hakeem Al-Hashim MD FRCPC, and Sean P Keenan MD FRCPC MSc

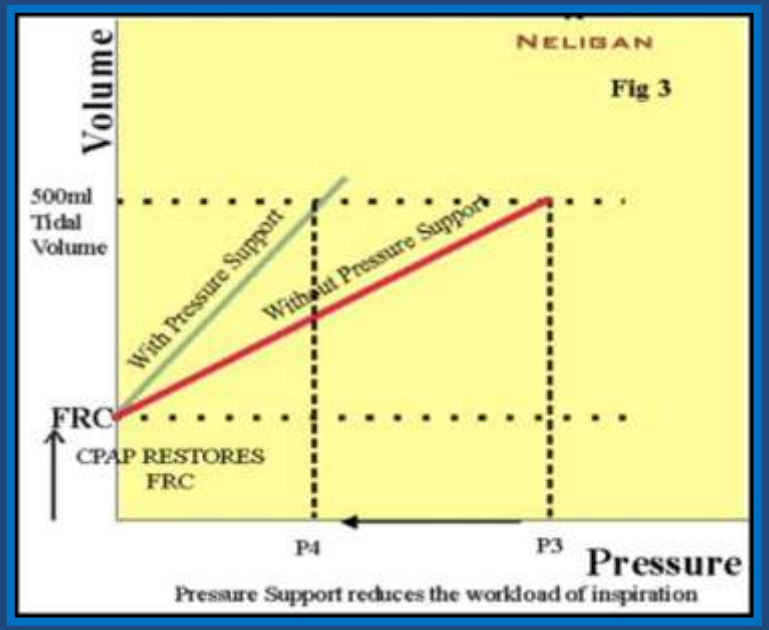
Table 1. Potential Mechanisms of Action of CPAP and NIV in Patients With Acute Cardiogenic Pulmonary Edema

CPAP
Increased functional residual capacity
Reduced atelectasis
Reduced right-to-left intrapulmonary shunt
Reduced work of breathing from improved pulmonary compliance
Increased cardiac output from reduced pre-load and after-load
Reduced mitral regurgitation
NIV
Same benefits as CPAP
Unloads the respiratory muscles

CPAP = continuous positive airway pressure
NIV = noninvasive ventilation

RESPIRATORY CARE • FEBRUARY 2009 VOL 54 NO 2





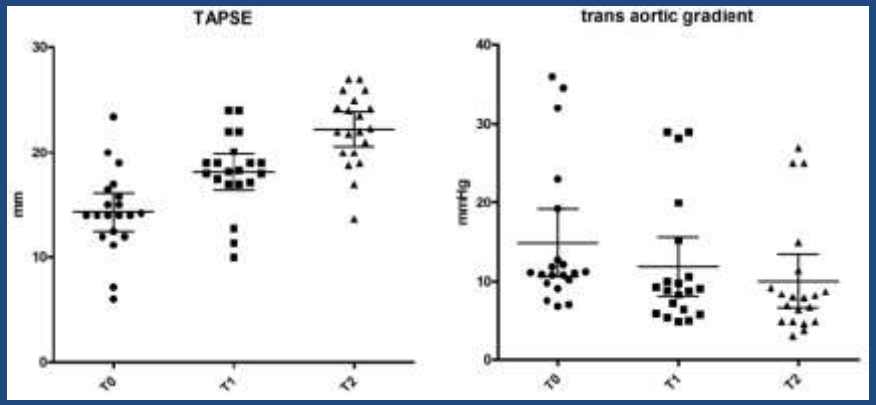
Compared to CPAP, NIV has been variably found to more rapidly improve clinical and physiologic variables, but NIV does not reduce intubation rate or mortality. Systematic reviews have not found NIV superior to CPAP in avoiding intubation or lowering mortality (see Figs. 1 and 2) or myocardial infarction rate.[24-26]

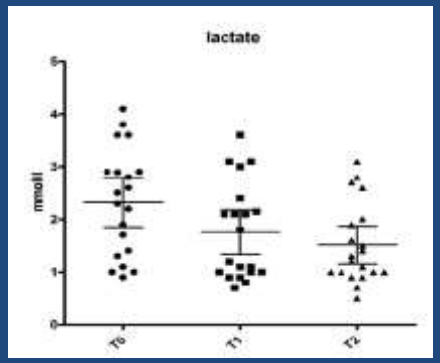
Hyperacute Hemodynamic Effects of BiPAP Noninvasive Ventilation in Patients With Acute Heart Failure and Left Ventricular Systolic Dysfunction in Emergency Department

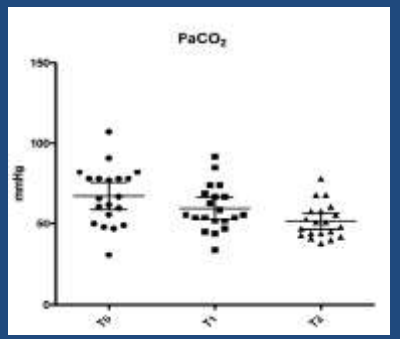
Cinzia Moret Iurilli, MD[1], Natale Daniele Brunetti, MD, PhD, FESC[2], Paola Rita Di Corato, MD[1], Giuseppe Salvemini, MD[1], Matteo Di Biase, MD[2], Marco Matteo Ciccone, MD[3], and Vito Procacci, MD[1]

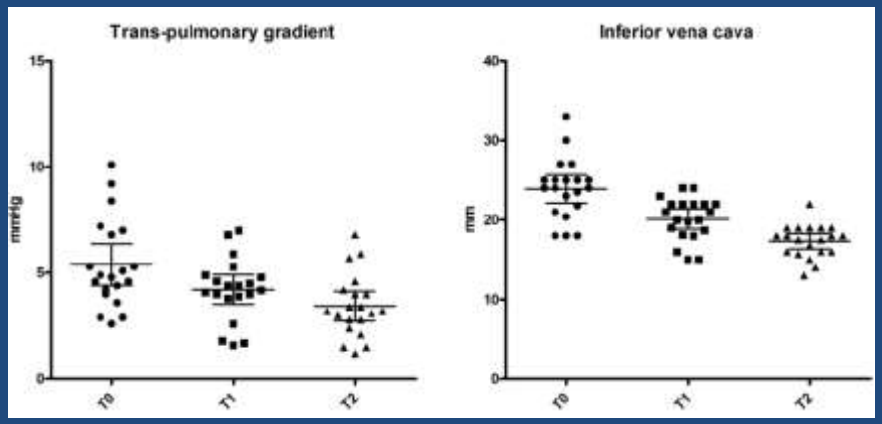
Journal of Intensive Care Medicine
1-6

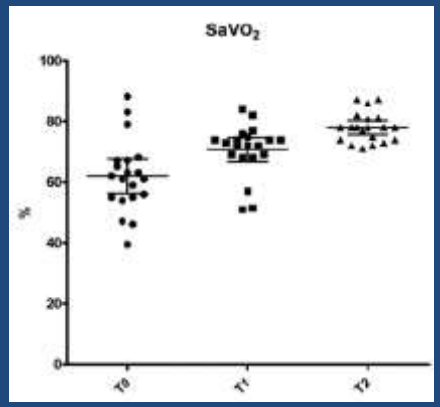
DOI: 10.1177/0885066617740849

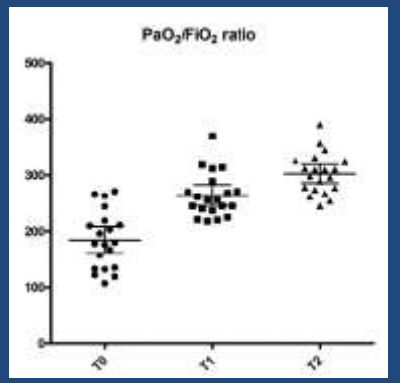












Question 2a: Should NIV be used in ARF due to cardiogenic pulmonary oedema?

Over 30 trials have been published on the use of CPAP and/or NIV compared with standard therapy or each other for patients with acute cardiogenic pulmonary oedema. The majority are small single-centre trials that span a period of 30 years, during which cardiogenic pulmonary oedema management, particularly in the setting of acute coronary syndrome, has evolved along with trial inclusion and exclusion criteria. Patients in cardiogenic shock have been almost universally excluded from trials and thus are not included in our NIV recommendations. Many trials have also excluded patients requiring acute revascularisation and some have excluded patients with acute coronary syndrome.

Recommendation

We recommend either bilevel NIV or CPAP for patients with ARF due to cardiogenic pulmonary oedema. (Strong recommendation, moderate certainty of evidence.)

TABLE 2 Recommendations for actionable PICO questions

Clinical indication[#]	Certainty of evidence[¶]	Recommendation
Prevention of hypercapnia in COPD exacerbation	⊕⊕	Conditional recommendation against
Hypercapnia with COPD exacerbation	⊕⊕⊕⊕	Strong recommendation for
Cardiogenic pulmonary oedema	⊕⊕⊕	Strong recommendation for
Acute asthma exacerbation		No recommendation made
Immunocompromised	⊕⊕⊕	Conditional recommendation for
***De novo* respiratory failure**		No recommendation made
Post-operative patients	⊕⊕⊕	Conditional recommendation for
Palliative care	⊕⊕⊕	Conditional recommendation for
Trauma	⊕⊕⊕	Conditional recommendation for
Pandemic viral illness		No recommendation made
Post-extubation in high-risk patients (prophylaxis)	⊕⊕	Conditional recommendation for
Post-extubation respiratory failure	⊕⊕	Conditional recommendation against
Weaning in hypercapnic patients	⊕⊕⊕	Conditional recommendation for

[#]: all in the setting of acute respiratory failure; [¶]: certainty of effect estimates: ⊕⊕⊕⊕, high; ⊕⊕⊕, moderate; ⊕⊕, low; ⊕, very low.

The management of patients with acute heart failure: oxygen therapy and ventilatory

Recommendations	Class	Level
Monitoring of transcutaneous arterial oxygen saturation (SpO_2) is recommended.	I	C
Measurement of blood pH and carbon dioxide tension (possibly including lactate) should be considered, especially in patients with acute pulmonary oedema or previous history of COPD using venous blood. In patients with cardiogenic shock arterial blood is preferable.	IIa	C
Oxygen therapy is recommended in patients with AHF and SpO_2 <90% or PaO_2 <60 mmHg (8.0 kPa) to correct hypoxaemia.	I	C
Non-invasive positive pressure ventilation (CPAP, BiPAP) should be considered in patients with respiratory distress (respiratory rate >25 breaths/min, SpO_2 <90%) and started as soon as possible in order to decrease respiratory distress and reduce the rate of mechanical endotracheal intubation. Non-invasive positive pressure ventilation can reduce blood pressure and should be used with caution in hypotensive patients. Blood pressure should be monitored regularly when this treatment is used.	IIa	B
Intubation is recommended, if respiratory failure, leading to hypoxaemia (PaO_2 <60 mmHg (8.0 kPa)), hypercapnia ($PaCO_2$ >50 mmHg (6.65 kPa)) and acidosis (pH <7.35), cannot be managed non-invasively.	I	C

European Heart Journal (2016) **37**, 2129-2200 - doi:10.1093/eurheartj/ehw 128

Approach to Acute Heart Failure in the Emergency Department

Benton R. Hunter, Jennifer Martindale, Osama Abdel-Hafez, Peter S. Pang

doi: 10.1016/j.pcad.2017.08.008

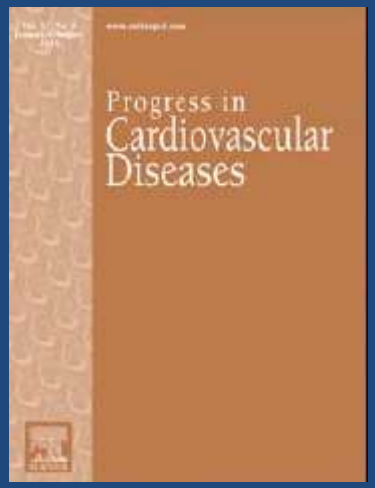


Table 2: Therapeutic Options for the Early Treatment of AHF – A Historical Perspective

1974[67]	2017
Sit the patient upright	Sit the patient upright
Oxygen	Oxygen
Positive pressure ventilation	Positive pressure ventilation
Morphine	Morphine
Diuretics	Diuretics
Intra-aortic balloon pump	Intra-aortic balloon pump
Phlebotomy	**Inotropes**
Rotating tourniquets	**Vasodilators / Nesiritide**

Table 3	Initial Dose	Infusion range	Recommendation Class (Evidence level)
Dobutamine	2-3 ucg/kg/min	2-20 ucg/kg/min	IIB (Level B
Milrinone		0.375-0.75 ucg/kg/min*	IIB (Level B)
Levosimendan		0.05 -0.2 ug/kg/min*	Not available in US
Dopamine	2-5 ucg/kg/min	2-50 ucg/kg/min	IIB (Level B)
Norepinephrine		0.2 – 1.0 ucg/kg/min	
Nitroglycerin	5-20 ucg/min (rapidly titrate to effect)	5-200 ucg/min (rapidly titrate to effect)	IIB (Level A)
Nitroprusside	5-10 mcg/min	0.25 ucg/kg/min – 10 ucg/kg/min	IIB (Level A)
Nesiritide		0.01 ucg/kg/min*	IIB (Level A)
ACE-I (enalaprilat)	1.25-5mg IV bolus q6 hrs	NA	NA
*Consider bolus dosing			

Table 3 shows commonly used inotropes and vasopressors. Although inotropes and vasodilators improve hemodynamics, to date, none are associated with better clinical outcomes. In fact, available inotropes have been associated with harm, though the

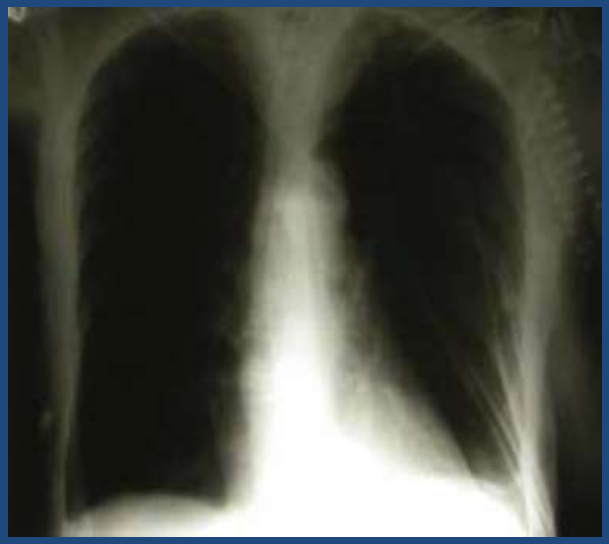
doi: 10.1016/j.pcad.2017.08.008

Caso Clinico 2

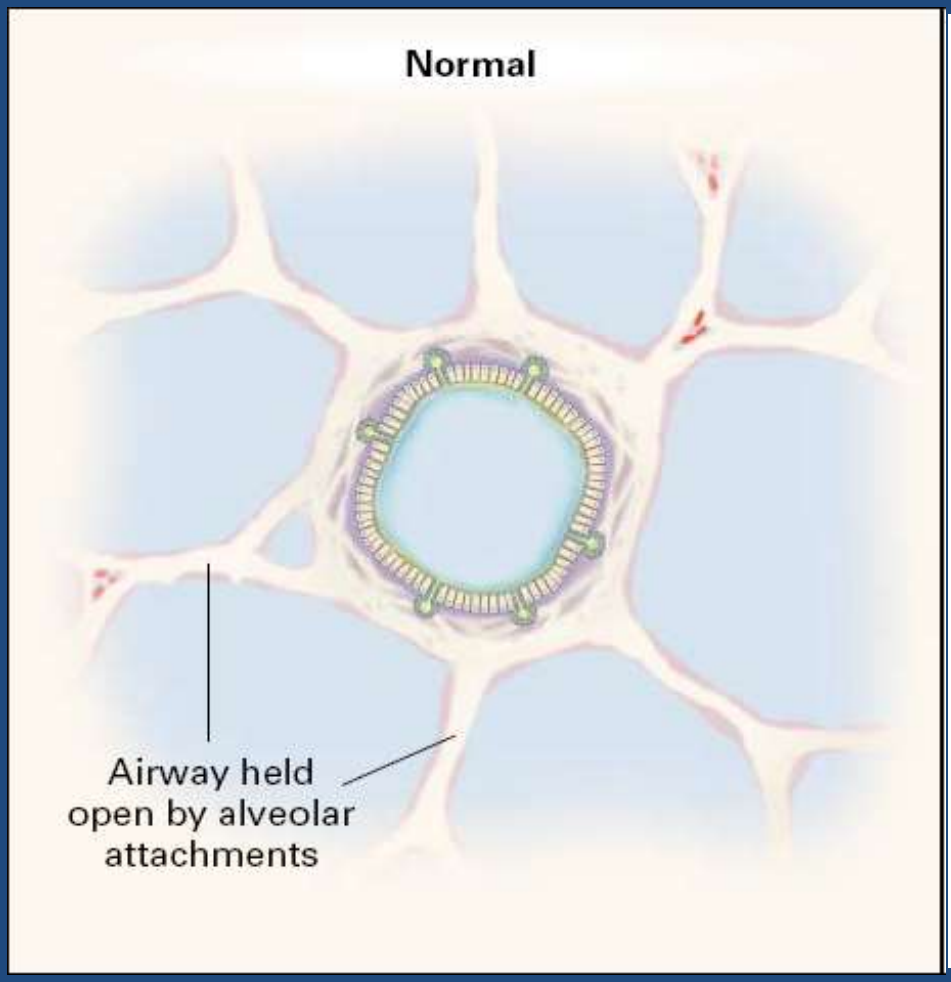
- Uomo, 75 anni
- Ex fumatore (ha smesso 15 aa fa)
- Precedente diagnosi di BPCO

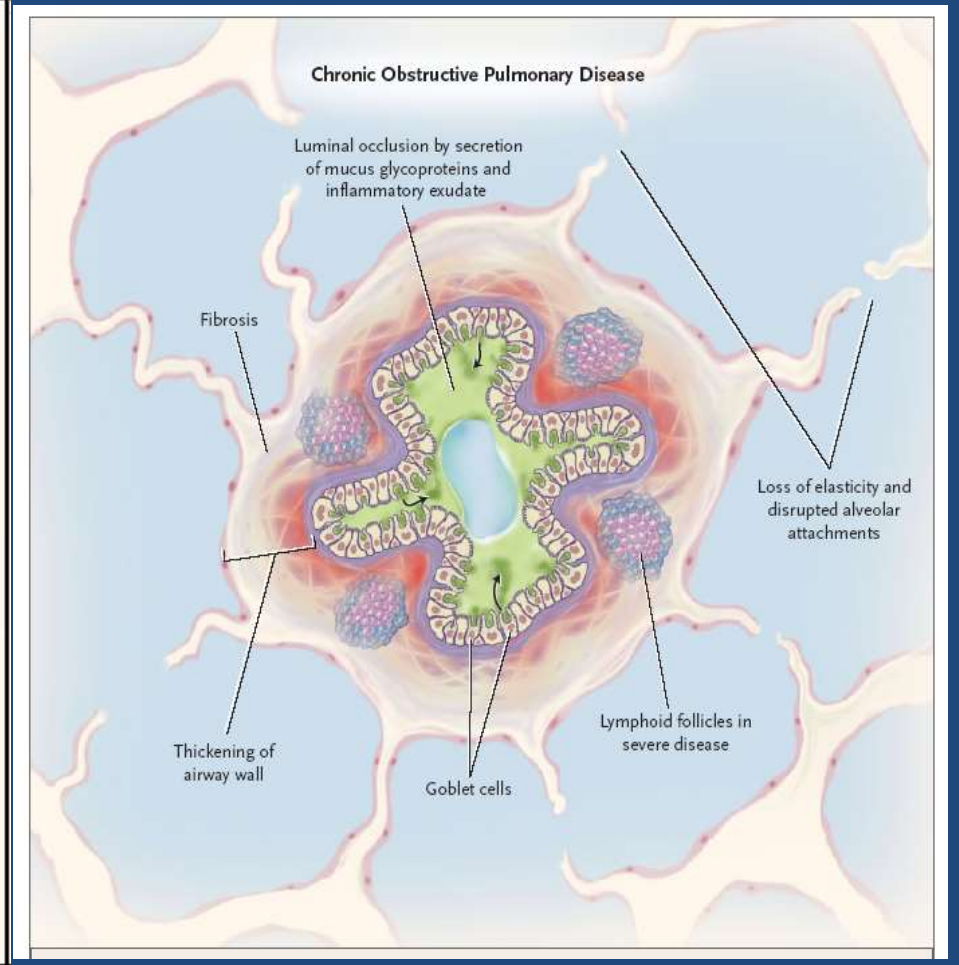
Caso Clinico 2

- In PS
  - PA: 210/110, FC=120/min, FR=33/min, SpO_2 86% in FiO_2 0.21, T= 36,5°
  - EGA: pH=7,25, PaO_2= 38,3, $PaCO_2$=85, HCO_3^-=36



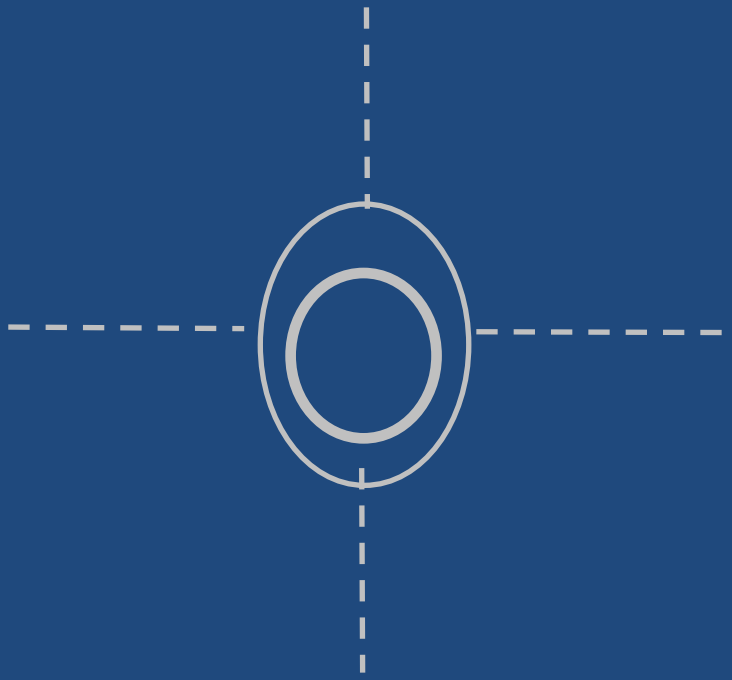
Istologia

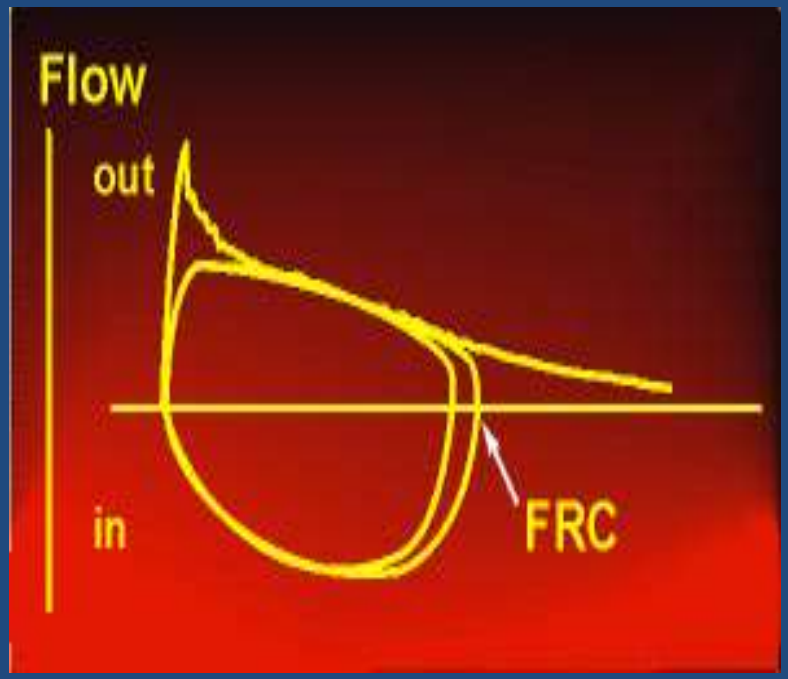




Barnes PJ N Engl J Med 2004; 350: 2635-37

Espirazione in BPCO

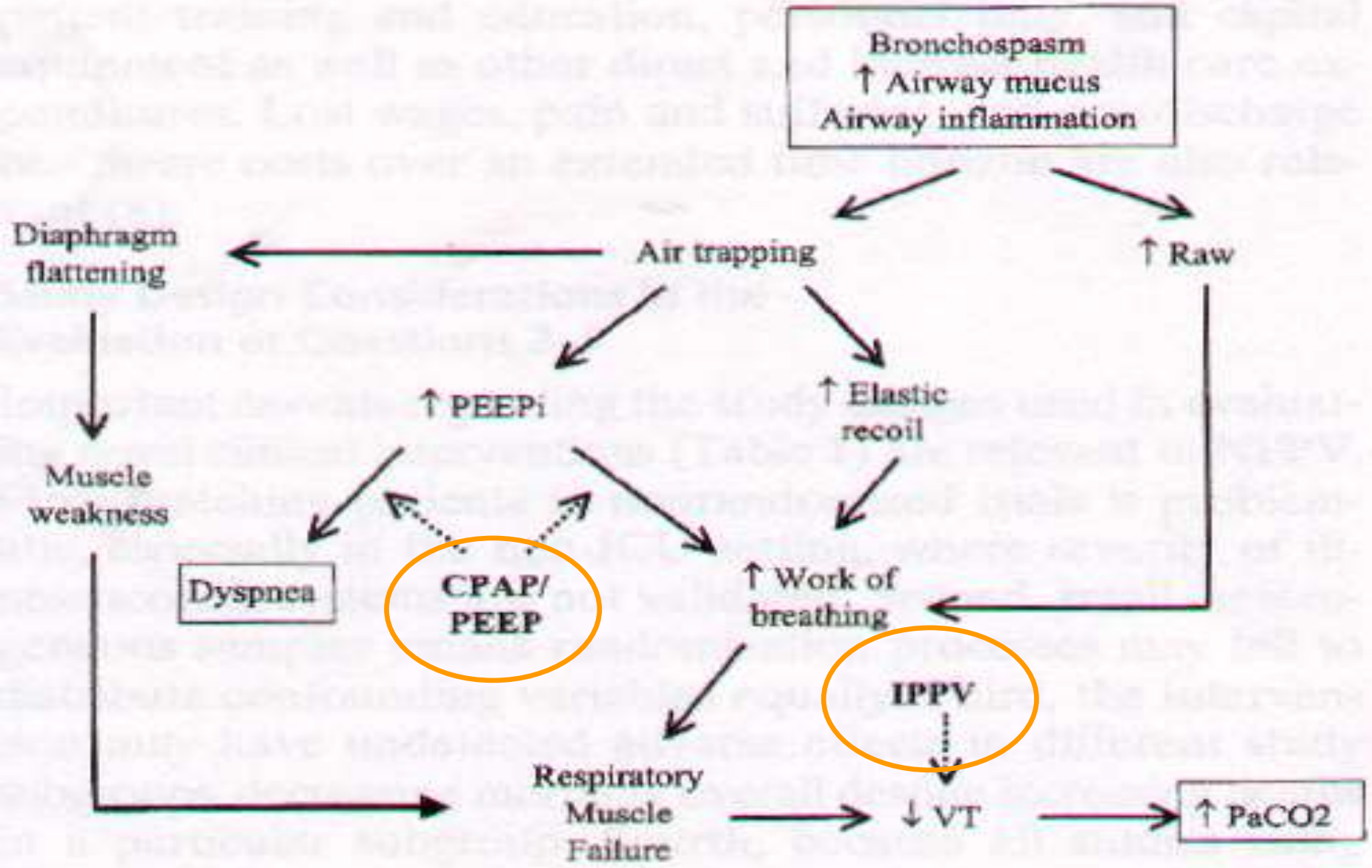




Lume del bronco in espirazione

Flow Volume Curve

Fattori determinanti di IRA



Non-invasive positive pressure ventilation to treat respiratory failure resulting from exacerbations of chronic obstructive pulmonary disease: Cochrane systematic review and meta-analysis

Josephine V Lightowler, Jadwiga A Wedzicha, Mark W Elliott, Felix S F Ram

- Lower mortality (relative risk 0.41) (95% confidence interval 0.26 to 0.64)),
- lower need for intubation

- greater improvements at 1 hour in pH, PaCO2 respiratory

- fewer complications associated with treatment
- shorter duration of stay in hospital

BMJ VOLUME 326 25 JANUARY 2003

Fig 3 Mortality in seven studies of non-invasive positive pressure ventilation (NPPV) as an adjunct to usual medical care

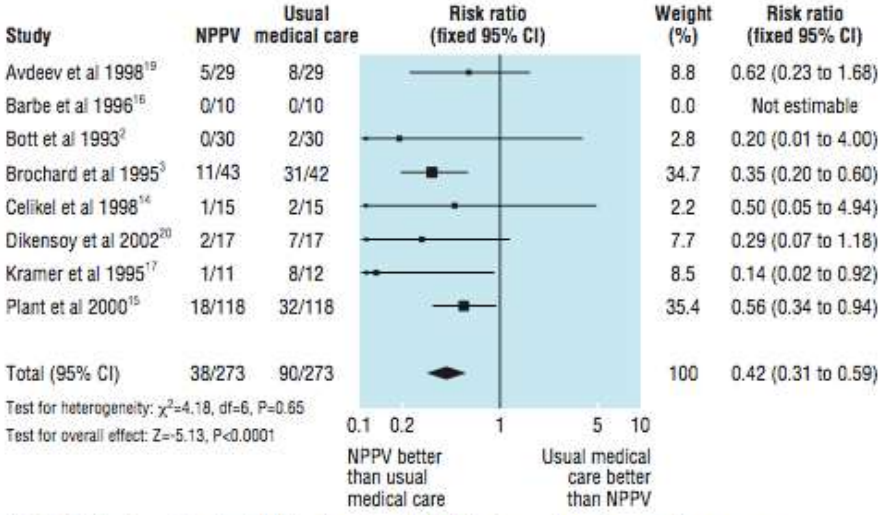


Fig 4 Risk of endotracheal intubation in eight trials of non-invasive positive pressure ventilation (NPPV) as an adjunct to usual medical care

Question 1b: Should NIV be used in established acute hypercapnic respiratory failure due to a COPD exacerbation?

Recommendations

We recommend bilevel NIV for patients with ARF leading to acute or acute-on-chronic respiratory acidosis (pH $\leqslant$7.35) due to COPD exacerbation. (Strong recommendation, high certainty of evidence.)

We recommend a trial of bilevel NIV in patients considered to require endotracheal intubation and mechanical ventilation, unless the patient is immediately deteriorating. (Strong recommendation, moderate certainty of evidence.)

Implementation considerations

1) Bilevel NIV should be considered when the pH is $\leqslant$7.35, P_{aCO_2} is >45 mmHg and the respiratory rate is >20–24 breaths·min^{-1} despite standard medical therapy.
2) Bilevel NIV remains the preferred choice for patients with COPD who develop acute respiratory acidosis during hospital admission. There is no lower limit of pH below which a trial of NIV is inappropriate; however, the lower the pH, the greater risk of failure, and patients must be very closely monitored with rapid access to endotracheal intubation and invasive ventilation if not improving.

Official ERS/ATS clinical practice guidelines: noninvasive ventilation for acute respiratory failure

Hospitalised patients are given much the same drug therapy as they were 25 yrs ago

The "ABC" approach

Table 1 What is known of pharmacological treatment

Bronchodilators

Short acting inhaled bronchodilators

- Improve symptoms and FEV_1
- No differences between different classes
- No additional benefit with combinations
- No differences between MDI and nebuliser use
- Recommend increasing dose and/or frequency of existing classes

Theophylline

- Discrete effects on symptoms and lung function (second line treatment)
- Numerous side effects

Systemic corticosteroids

- Improve symptoms, FEV_1 and PaO_2 in moderate to severe exacerbations
- Reduce treatment failure, relapse and length of hospital stay
- Induce more side effects (such as hyperglycaemia)

Antibiotics

- Less risk of treatment failure, mortality and sputum purulence in moderate to severe exacerbations
- Oral route is preferred and cheaper
- Unlikely benefit of short courses of antibiotics in most patients

FEV_1, forced expiratory volume in 1 second; PaO_2, arterial oxygen pressure.

(Rodriguez-Roisin 2005)

Official Journal of the Asian Pacific Society of Respirology
Respirology

Acute exacerbation of COPD

FANNY W. KO,[1] KA PANG CHAN,[1] DAVID S. HUI,[1] JOHN R. GODDARD,[2,3] JANET G. SHAW,[2,3] DAVID W. REID[2,3,4] AND IAN A. YANG[2,3]

Broncodilatatori short acting: evidenza di tipo C

Via di somm.	Dosaggio	Vantaggi	svantaggi
Nebulizzatore	Salbutamolo: 2,5 mg x 4 Ipratropio b: 0,5 mg x 1->4	Pz. non in grado di collaborare	Costi Trasmissione infezioni
MDI + spacer	Salbutamolo: 200 mcg x 4 Ipratropio b: fuori commercio	Somm. rapida Riduzione costi Training paziente	Solo pz. collaborante

Respirology (2016)
doi: 10.1111/resp.12780

Systemic corticosteroids

Table 2 Dose and duration of systemic corticosteroids for treatment of acute exacerbation of chronic obstructive pulmonary disease, as recommended by different clinical guidelines

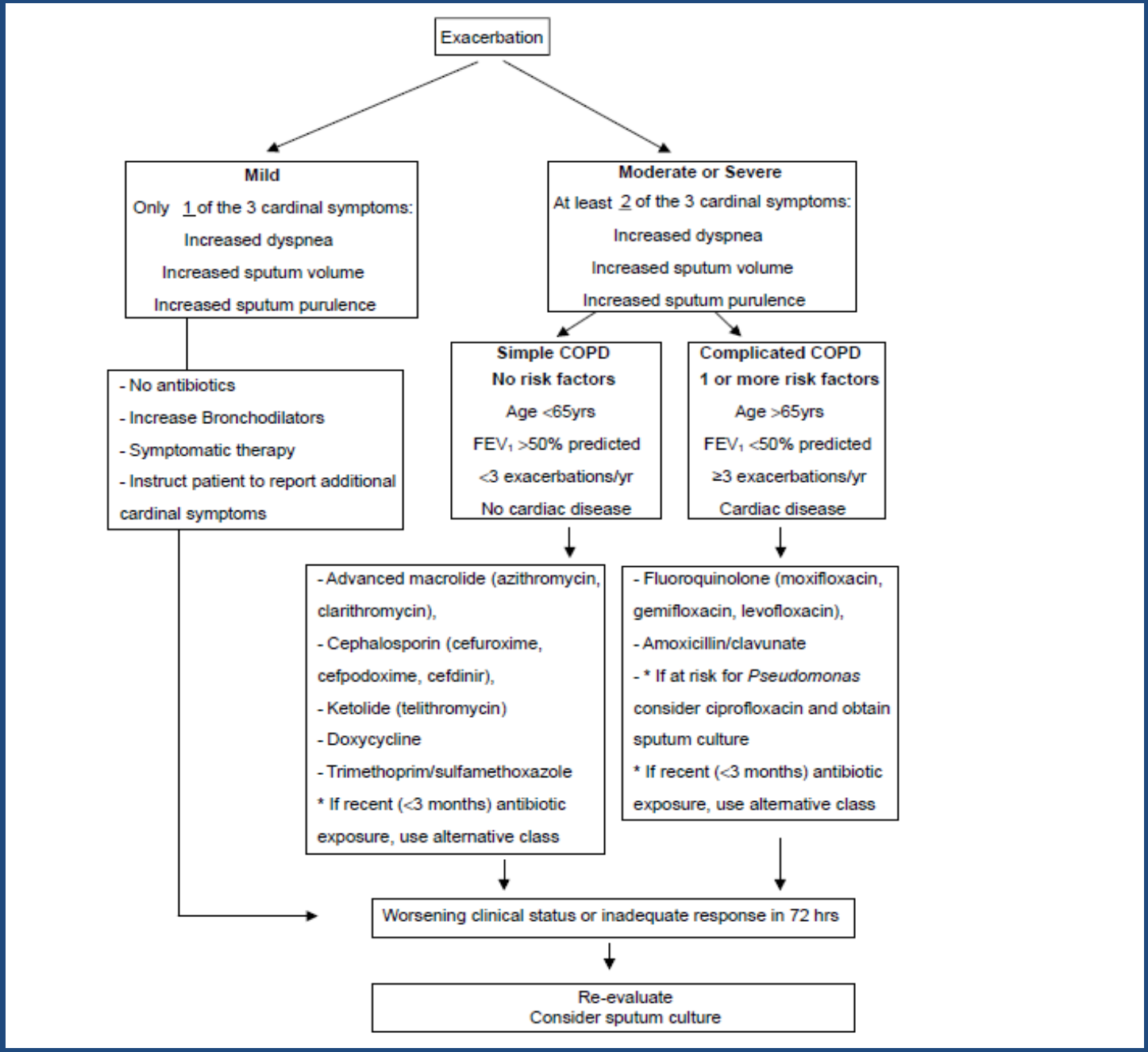
GOLD[3]	Oral prednisolone 40 mg/day × 5 days
JRS[60]	Oral prednisolone 30–40 mg/day × 7–10 days
TSANZ[61]	Oral prednisolone 30–50 mg/day × 5 days (tapering dose required for those receiving > 14 days)
MTS[62]	Oral corticosteroids, no longer than 14 days, dose not specified
PCCP[63]	Oral prednisolone 30–40 mg/day × 7–14 days

GOLD, Global Obstructive Lung Disease; JRS, Japanese Respiratory Society; MTS, Malaysian Thoracic Society; PCCP, Philippine College of Chest Physicians; TSANZ, Thoracic Society of Australia and New Zealand.

Respirology (2016)
doi: 10.1111/resp.12780

Antibiotics should be given to pts. With:

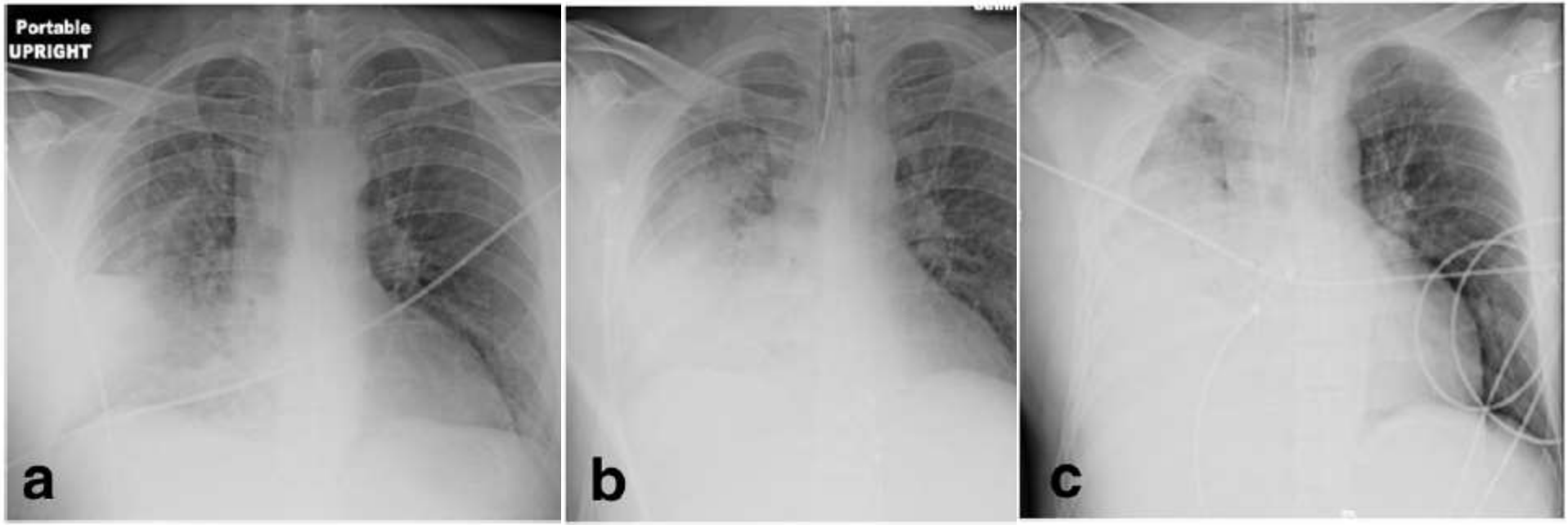
- Increase in dyspnea, sputum volume and purulence (evidence B)
- Two of the cardinal symptoms if one is increased purulence (evidence C)
- Need for mechanical ventilation (invasive or noninvasive) (evidence B)
- The recommended lenght of antibiotic therapy is 5-10 days (evidence D)



(Siddiqi and Sethi, 2008)

Caso clinico 3

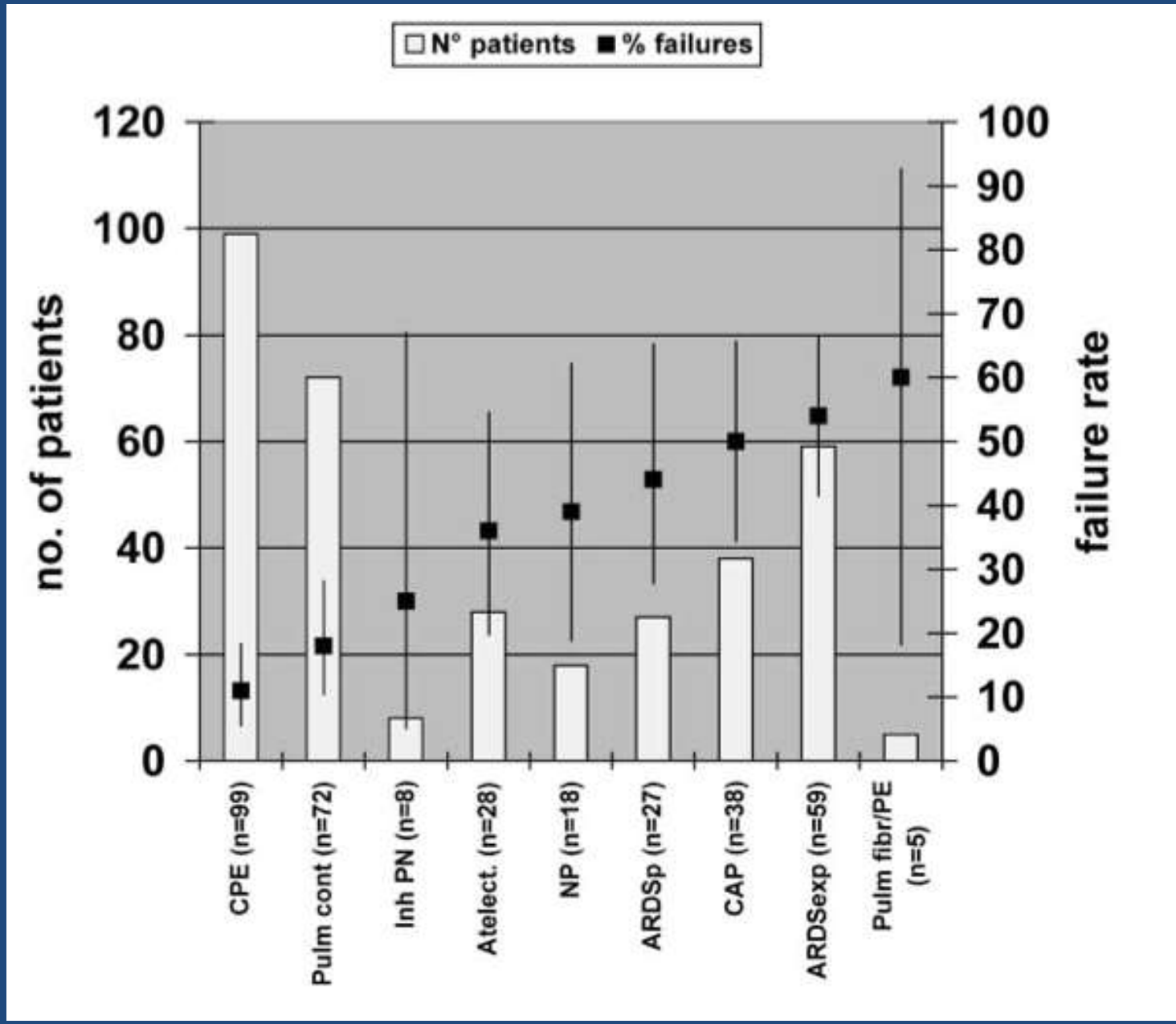
- Pz. Di 40 aa, TD. In PS per dispnea ingravescente e febbre.
- Pa 90/55; FR 38/min; FC 120/min; GCS 15/15; TC 39°C; Marezzato.
- pH 7.28; PaO2 45; PaCO2 38; HCO3- 17; Lac 4.1
- Tazobactam-piperacillina, Ciprofloxacina, Fluidi, steroidi, CPAP



Dopo 2 ore di trattamento con CPAP:
pH 7.23; PaO2 60 mmHg; Pa CO2 55; HCO3 -16; Lac 7.2
PA 80/?
Intubato e trasferito in RIA

Controllo Rx a 4 ore dall'ingresso e 4 l di fisiologica

Controllo Rx a 12 ore dall'ingresso
E 9 l di fisiologica.
PVC: 10 mmHg
Decesso 6 ore dopo per ipossiemia refrattaria



Antonelli M et al. Intensive Care Med 2001;27:1718-1728

Univariate and multivariate analysis of the risk factors for failure of noninvasive ventilation

Variables	No. of failures/total (%)	Univariate analysis		Multivariate analysis	
		OR	95% CI	OR	95% CI
Reason for ICU admission					
Medical	58/218 (27)	1.00			
Surgical/trauma	50/136 (37)	1.96	1.11–3.45		
Age, years					
≤40	18/93 (19.4)	1.00			
> 40	90/261 (34.5)	2.19	1.19–4.06	1.72	0.92–3.23
SAPS II					
< 35	55/236 (23.3)	1.00		1.00	
≥35	53/118 (44.9)	2.68	1.63–4.42	1.81	1.07–3.06
Underlying disease					
None or none of the following	97/333 (29)	1.00			
Diabetes	11/21 (52)	2.47	1.06–5.74		
Etiology of respiratory failure					
None of the following	42/225 (18.6)	1.00		1.00	
ARDS, CAP	66/129 (51.1)	4.77	2.86–7.96	3.75	2.25–6.24
Respiratory rate at baseline, breaths/min					
≤38	79/285 (27.7)	1.00			
> 38	29/69 (42)	1.89	1.06–3.37		
PaO2:FiO2 after 1 h of NPPV					
> 146	64/264[a] (24.2)	1.00		1.00	
≤146	44/89 (49.4)	3.06	1.79–5.21	2.51	1.45–4.35
Sepsis on admission					
No	77/295 (26.1)	1.00			
Yes	31/59 (52.5)	3.13	1.70–5.78		

[a] For one patient PaO2:FiO2 value 1 h after NIV was missing

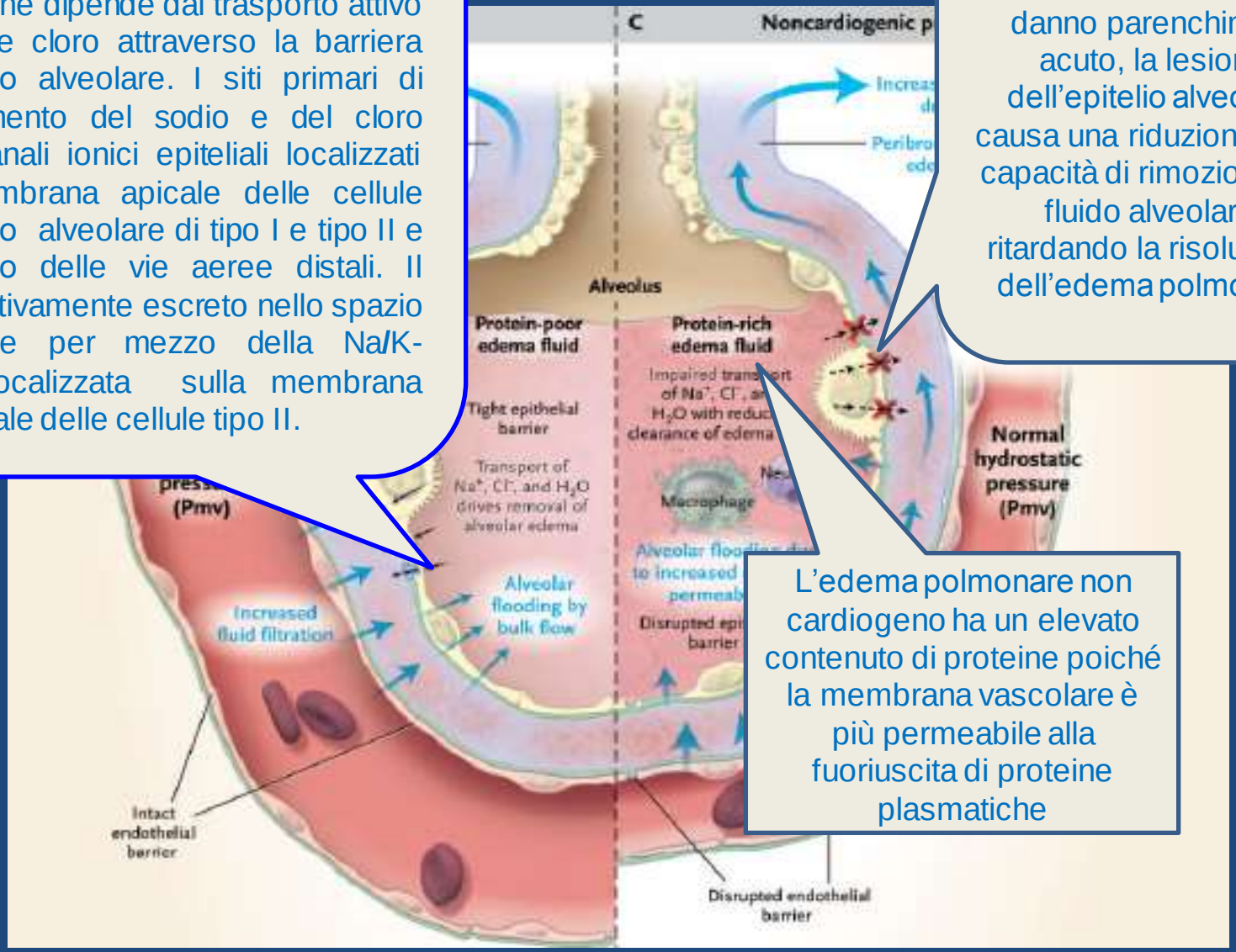
Antonelli et al. Intensive Care Med 2001

Outcome variables, complications and mortality

Variable[d]	Avoided intubation ($n = 246$)		Required intubation ($n = 108$)		P value
Duration of NIV (h), median (range)	48	(1–216)	24	(1–192)	0.06[a]
Length of stay (days), median (range)	5	(3–31)	9	(1–72)	< 0.001
No. of complications related to noninvasive ventilation (%)					
Nasal/facial skin necrosis	25	(10)	9	(8)	0.29[b]
Conjunctivitis	4	(2)	3	(3)	0.36[c]
Gastric distention	3	(1)	2	(2)	0.48[c]
No. of complications causing death in intensive care unit/total no. of complications (%)					
Ventricular fibrillation/cardiac arrest[d]	1/1	(0.4)	3/3	(3)	0.08[b]
Acute myocardial infarction	1/1	(0.4)	2/2	(2)	0.22[b]
Cardiogenic shock	5/6	(2)	8/8	(7)	0.19[b]
Pulmonary embolism	0/2	(1)	1/2	(1)	0.66[b]
Gastrointestinal bleeding	0/1	(0.4)	0/1	(1)	0.51[b]
Cerebral hemorrhage	0/0	(0)	0/1	(1)	0.30[b]
Ventilator-associated pneumonia	0/1	(0.4)	20/30	(28)	< 0.001
Severe sepsis and septic shock with multiple organ failure after study entry[e]	6/8	(3)	50/70	(64.8)	< 0.001
ICU Mortality, no. (%)[f]	13	(5.3)	64	(59.3)	< 0.001
ICU Mortality by subgroups, no. of deaths/total no (%)					
Pulmonary ARDS	0/15	(0)	4/12	(33)	0.03
Extra-pulmonary ARDS[g]	4/27	(15)	18/32	(56)	0.003
Community-acquired pneumonia	0/19	(0)	10/19	(53)	< 0.001
Nosocomial pneumonia	1/11	(9)	7/7	(100)	< 0.001
Inhalation pneumonia	1/6	(16)	1/2	(50)	10.0[c]
Cardiogenic pulmonary edema	7/89	(8)	9/10	(90.0)	< 0.001
Pulmonary embolism	0/2	(0)	2/2	(100)	0.33[c]
Mucous plugging and atelectasis	0/18	(0)	4/10	(40.0)	0.01[c]
Pulmonary fibrosis	0/0	(0)	1/1	(100)	
Pulmonary contusion and multiple trauma	0/59	(0)	8/13	(64)	< 0.001
Hospital mortality no (%)	20	(8.1)	69	(64)	< 0.001

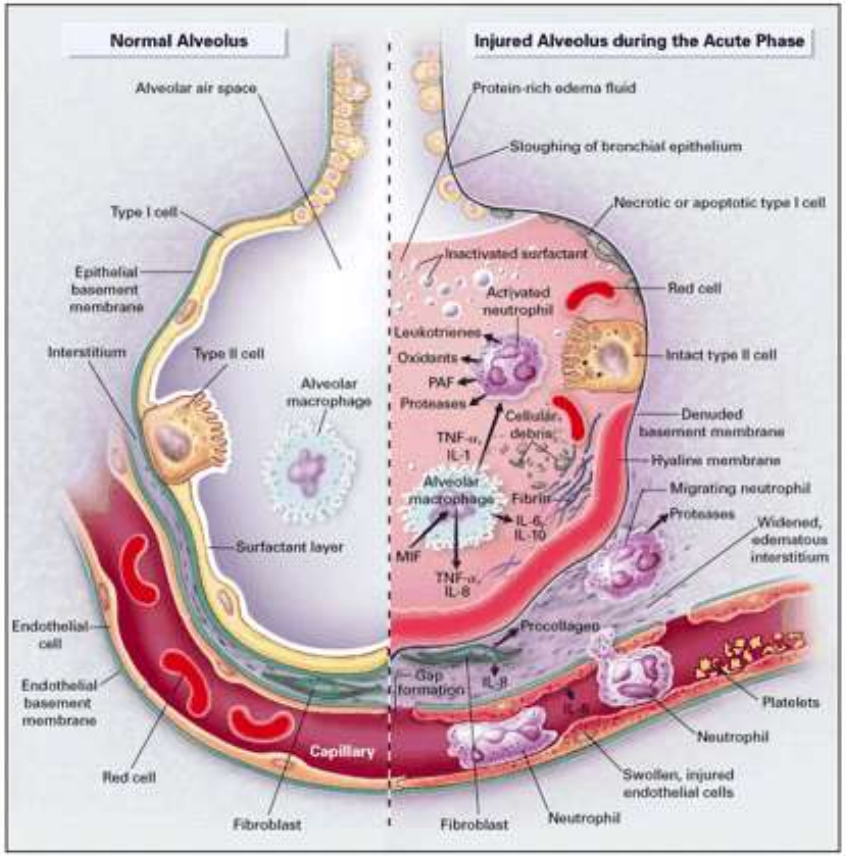
Antonelli et al. Intensive Care Med 2001

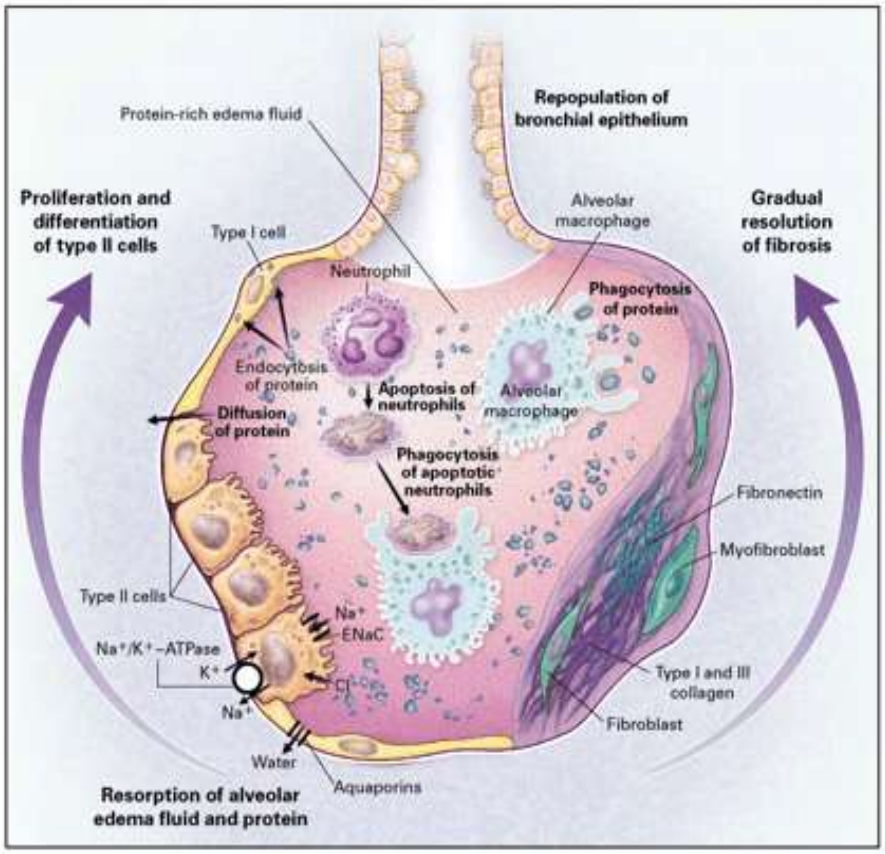
La rimozione di fluido dagli spazi aerei del polmone dipende dal trasporto attivo di sodio e cloro attraverso la barriera dell'epitelio alveolare. I siti primari di riassorbimento del sodio e del cloro sono i canali ionici epiteliali localizzati sulla membrana apicale delle cellule dell'epitelio alveolare di tipo I e tipo II e dell'epitelio delle vie aeree distali. Il sodio è attivamente escreto nello spazio interstiziale per mezzo della Na/K-ATPasi localizzata sulla membrana basolaterale delle cellule tipo II.



Nell'edema dovuto ad un danno parenchimale acuto, la lesione dell'epitelio alveolare causa una riduzione della capacità di rimozione del fluido alveolare, ritardando la risoluzione dell'edema polmonare

L'edema polmonare non cardiogeno ha un elevato contenuto di proteine poiché la membrana vascolare è più permeabile alla fuoriuscita di proteine plasmatiche


Normal Alveolus
Injured Alveolus during the Acute Phase
Alveolar air space
Type I cell
Epithelial basement membrane
Interstitium
Type II cell
Alveolar macrophage
Surfactant layer
Endothelial cell
Endothelial basement membrane
Red cell
Capillary
Fibroblast
Protein-rich edema fluid
Sloughing of bronchial epithelium
Necrotic or apoptotic type I cell
Inactivated surfactant
Activated neutrophil
Red cell
Leukotrienes
Oxidants
PAF
Proteases
Intact type II cell
Cellular debris
Denuded basement membrane
TNF-α, IL-1
Hyaline membrane
Alveolar macrophage
Fibrin
Migrating neutrophil
Proteases
IL-6, IL-10
MIF
Widened, edematous interstitium
TNF-α, IL-8
Procollagen
Gap formation
IL-8
Platelets
Neutrophil
Swollen, injured endothelial cells
Fibroblast
Neutrophil

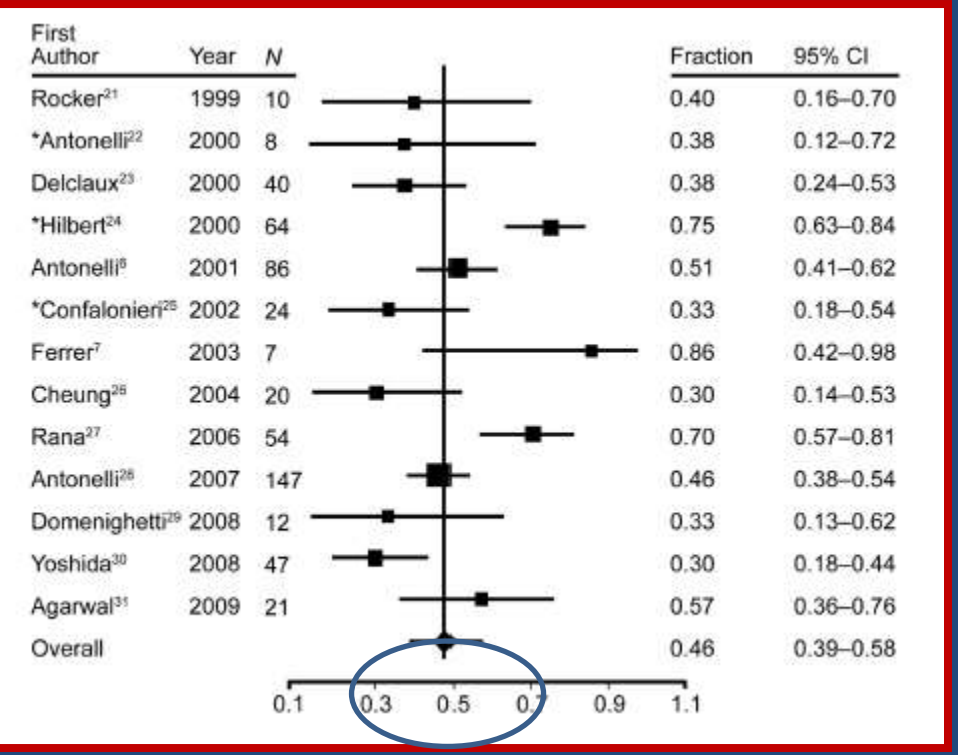

Protein-rich edema fluid
Repopulation of bronchial epithelium
Proliferation and differentiation of type II cells
Gradual resolution of fibrosis
Type I cell
Alveolar macrophage
Neutrophil
Phagocytosis of protein
Endocytosis of protein
Apoptosis of neutrophils
Alveolar macrophage
Diffusion of protein
Phagocytosis of apoptotic neutrophils
Fibronectin
Myofibroblast
Type II cells
Na+ ENaC
Na+/K+–ATPase
K+
Cl−
Na+
Type I and III collagen
Fibroblast
Water
Aquaporins
Resorption of alveolar edema fluid and protein

Role of Noninvasive Ventilation in Acute Lung Injury/Acute Respiratory Distress Syndrome: A Proportion Meta-analysis

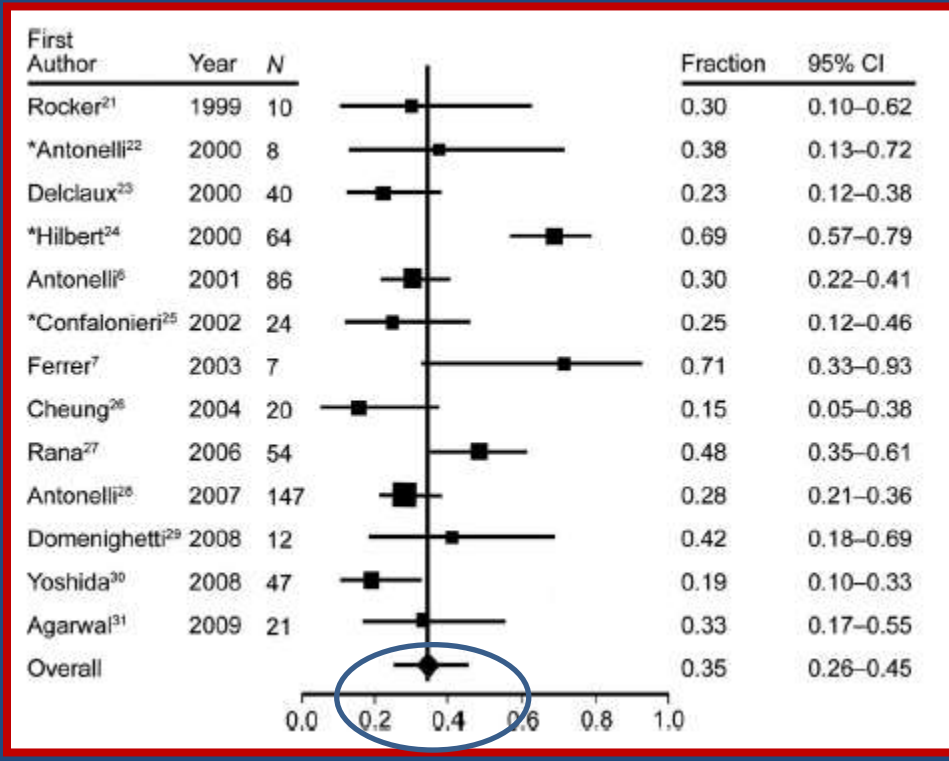
Ritesh Agarwal MD DM, Ashutosh N Aggarwal MD DM, and Dheeraj Gupta MD DM

RESPIRATORY CARE • DECEMBER 2010 VOL 55 NO 12

Intubation



Death



540 pts

Intensive Care Med (2012) 38:458–466
DOI 10.1007/s00134-012-2475-6

ORIGINAL

Table 3 Multivariate analysis of variables independently associated with non-invasive ventilation failure in the overall population

	Adj. OR	95% CI	*p* value	AUC	Optimal cut-off	Sensitivity (%)	Specificity (%)	Likelihood ratio	
								Positive	Negative
Maximum SOFA during NIV	1.442	1.187–1.753	<0.001	0.86	≥7	81	80	4.08	0.24
Worsening X-ray infiltrate 24 h after onset of NIV	84.23	16.74–423.8	<0.001	–	–	77	86	5.58	0.27
Heart rate 1 h after NIV onset, min^{-1}	1.064	1.029–1.100	<0.002	0.68	≥104	63	67	1.93	0.55
PaO_2/FiO_2 ratio 1 h after NIV onset, mmHg	0.980	0.965–0.996	0.012	0.78	<144	53	91	5.58	0.52
HCO_3 1 h after NIV onset, mEq/L	0.802	0.711–0.905	<0.001	0.77	<23	67	68	2.72	0.48

Adj. OR adjusted odds ratio, *CI* confidence interval, *SOFA* Sepsis-Related Organ Failure Assessment, *AUC* area under the ROC curve

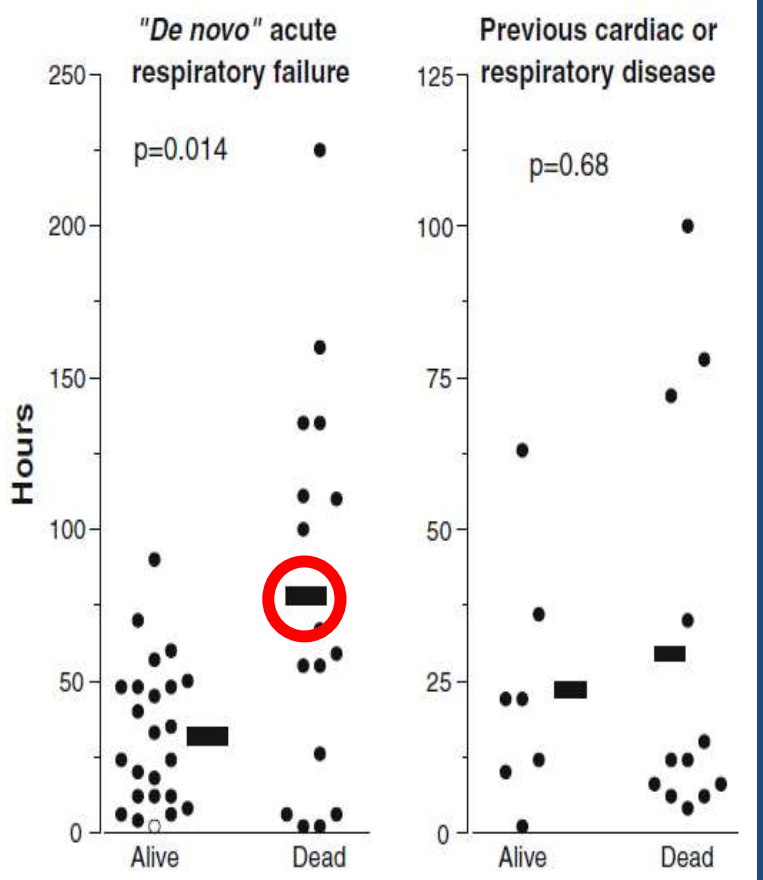


Fig. 1 Duration of non-invasive ventilation in patients who needed intubation and survived or died in the hospital. *Left panel* patients with "de novo" acute respiratory failure. *Right panel* patients with previous cardiac or respiratory disease. *Horizontal bars* represent mean values of survivors and non-survivors for intubated patients from each group

- Pz affetti da CAP+ preesistenti problemi cardiaci o respiratori rispondono meglio alla NPPV rispetto a quelli con «de novo» AhRF
- Nei pz affetti da «de novo» AhRF vi è una forte associazione tra ritardata IOT e riduzione della mortalità
- CONCLUSIONI: nei pz affetti da de novo AhRF in presenza di predittori di fallimento della NIV, ed in particolare se in trattamento con vasopressori, bisogna evitare di tardare la IOT al fine di ridurre la mortalità

Anna Maria Brambilla
Stefano Aliberti
Elena Prina
Francesco Nicoli
Manuela Del Forno
Stefano Nava
Giovanni Ferrari
Francesco Corradi
Paolo Pelosi
Angelo Bignamini
Paolo Tarsia
Roberto Cosentini

Helmet CPAP vs. oxygen therapy in severe hypoxemic respiratory failure due to pneumonia

Intensive Care Med 2014
DOI 10.1007/s00134-014-3325-5

Table 2 Primary endpoint

Met ETI criteria, *n* (%)	6
Major criteria	

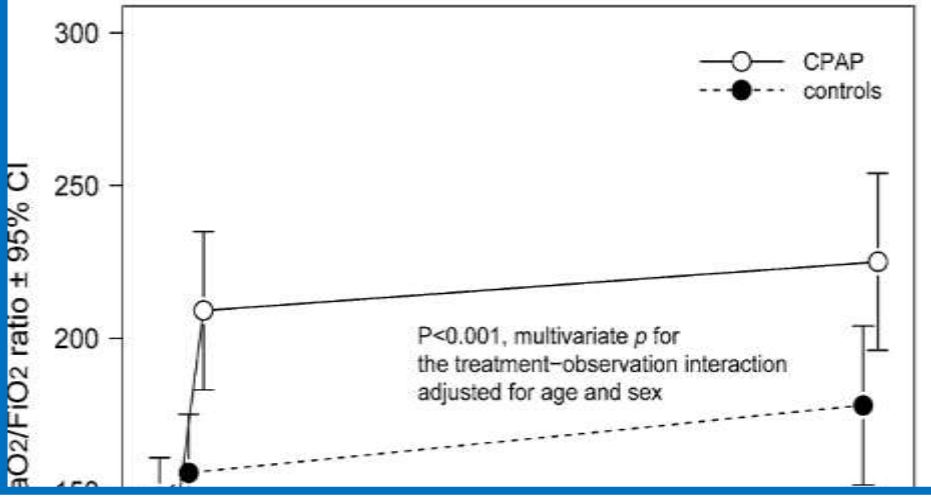


Table 3 Secondary endpoints

Characteristic	CPAP group ($n = 40$)	Control group ($n = 41$)	*p*
In-hospital mortality, *n* (%)	2 (5.0)	7 (17.1)	0.155[c]
Hospital length of stay, median (IQR) days[a]	14.5 (10.8–24.3) $n = 38$	14.0 (10.0–16.0) $n = 34$	0.122[d]
Discomfort to device, *n* (%)	6 (15.0)[b]	2 (4.9)	0.155[c]

Remaining cases:

Infectious Diseases Society of America/American Thoracic Society Consensus Guidelines on the Management of Community-Acquired Pneumonia in Adults

Lionel A. Mandell,[1,a] Richard G. Wunderink,[2,a] Antonio Anzueto,[3,4] John G. Bartlett,[7] G. Douglas Campbell,[8] Nathan C. Dean,[9,10] Scott F. Dowell,[11] Thomas M. File, Jr.[12,13] Daniel M. Musher,[5,6] Michael S. Niederman,[14,15] Antonio Torres,[16] and Cynthia G. Whitney[11]

Clinical Infectious Diseases 2007; 44:S27–72

36. Patients with hypoxemia or respiratory distress should receive a cautious trial of noninvasive ventilation unless they require immediate intubation because of severe hypoxemia (PaO_2/FiO_2 ratio, <150) and bilateral alveolar infiltrates. (Moderate recommendation; level I evidence.)

36. Patients with hypoxemia or respiratory distress should receive a cautious trial of noninvasive ventilation (NIV) unless they require immediate intubation because of severe hypoxemia (arterial oxygen pressure/fraction of inspired oxygen [PaO_2/FiO_2] ratio, <150) and bilateral alveolar infiltrates. (Moderate recommendation; level I evidence.)

Patients who do not require immediate intubation but who have either hypoxemia or respiratory distress should receive a trial of NIV [114, 288, 289]. Patients with underlying COPD are most likely to benefit. Patients with CAP who were randomized to receive NIV had a >25% absolute risk reduction for the need for intubation [114]. The use of NIV may also improve intermediate-term mortality. Inability to expectorate may limit the use of NIV [290], but intermittent application of NIV may allow for its use in patients with productive cough unless sputum production is excessive. Prompt recognition of a failed NIV trial is critically important, because most studies demonstrate worse outcomes for patients who require intubation after a prolonged NIV trial [288, 290]. Within the first 1–2 h of NIV, failure to improve respiratory rate and oxygenation [114, 289, 290] or failure to decrease carbon dioxide partial pressure (pCO_2) in patients with initial hypercarbia [114] predicts NIV failure and warrants prompt intubation. NIV provides no benefit for patients with ARDS [289], which may be nearly indistinguishable from CAP among patients with bilateral alveolar infiltrates. Patients with CAP who have severe hypoxemia (PaO_2/FiO_2 ratio, <150) are also poor candidates for NIV [290].

Conclusioni

Authors' conclusions

Non-invasive ventilation can reduce the risk of death in the ICU, endotracheal intubation, shorten ICU stay and length of intubation. Some outcomes and complications of non-invasive ventilation were varied according to different participant populations. Other than the oxygen therapy, we must mention the importance of standard treatment by physicians. The evidence is weak and we did not include participants with pulmonary tuberculosis and cystic fibrosis. More RCTs are required to answer these clinical questions. However, the review indicates that non-invasive ventilation may be more beneficial than standard oxygen supplementation via a Venturi mask for pneumonia.

non-invasive ventilation may be more beneficial than standard oxygen supplementation

The evidence is weak

More RCTs are required to answer these clinical questions.

Cochrane Database Syst Rev. 2012 Mar

NIV: efficacia

- > 90 % dei pazienti con EPAc
- 70 % dei pazienti con COPD Ex
- 50% dei pazienti con AHRF

PAZIENTE GIUSTO	MOMENTO GIUSTO	POSTO GIUSTO
Non controindicazioni alla NIV/CPAP non indicazioni immediate a IOT (p/F, emodinamica, danno d'organo) immunodepresso BPCO paziente DNI	stato di coscienza predittori di fallimento PRECOCI della NIV (p/F, FR, pH, HACOR…) predittori di fallimento TARDIVI della NIV (p/F, FR, pH…)	Staff: • medico • **infermieristico** • adeguato in numero • preparato Monitoraggio: • umano • tecnologico

RAPIDO PASSAGGIO ALLA IOT E VENTILAZIONE INVASIVA IN CASO DI FALLIMENTO DELLA NIV

Failure criteria

pH < 7.10 – 7.20

Hypercapnic coma

PaO2 < 60 mmHg notwithsanding maximal tolerated FiO2 or maximal tolerated IPAP

Shock, cardiac arrest

REVIEW Open Access

Timing of noninvasive ventilation failure: causes, risk factors, and potential remedies

Ezgi Ozyilmaz[1], Aylin Ozsancak Ugurlu[2] and Stefano Nava[3*]

Ozyilmaz *et al. BMC Pulmonary Medicine* 2014, **14**:19

BMC Pulmonary Medicine

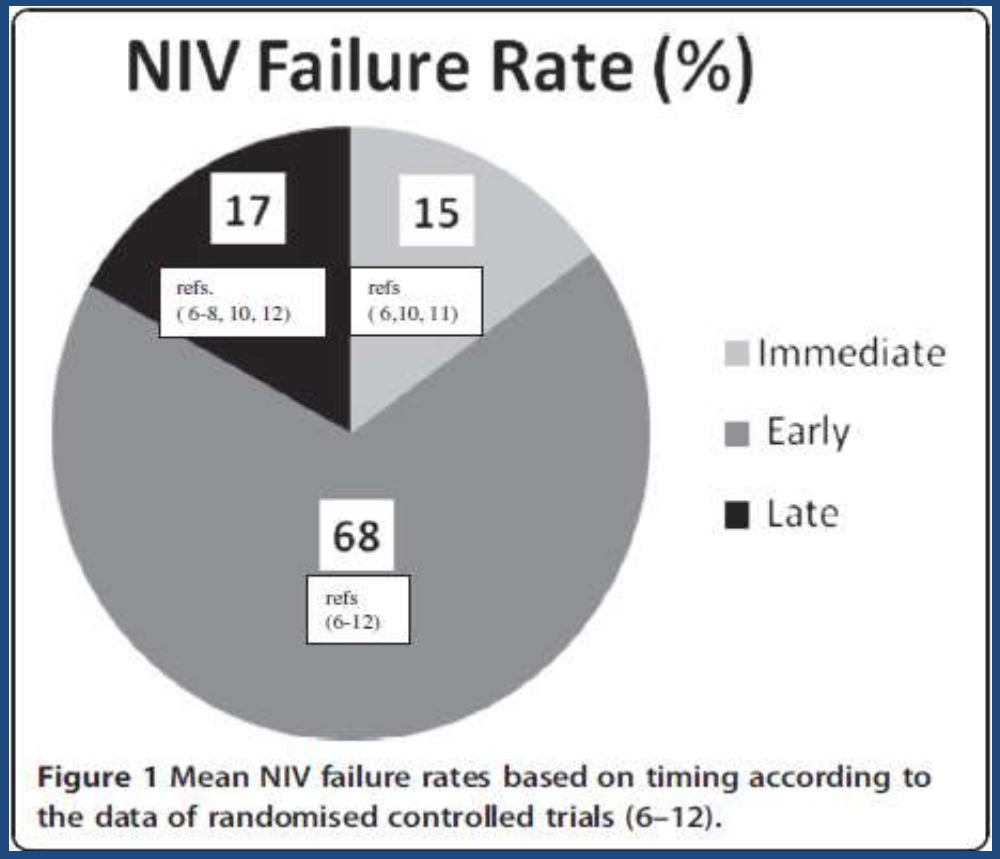


Figure 1 Mean NIV failure rates based on timing according to the data of randomised controlled trials (6–12).

Table 1 Indications and contraindications for NIV in acute care [4,16,17,35]

Indications:

A- Gas exchange:

- Acute or acute on chronic ventilator failure (best indication), $PaCO_2 > 45$ mmHg
- Ph < 7.35
- Hypoxemia (use with caution), PaO_2/FIO_2 ratio < 200

B- Bedside observations:

- Increased dyspnea- moderate to severe
- Tachypnoea (24 breaths per minute in obstructive, >30 per minute in restrictive)
- Signs of increased work of breathing, accessory muscle use, and abdominal paradox

Absolute contraindications:

- Cardiac or respiratory arrest
- Unable to fit mask

Relative contraindications:

- Non-respiratory organ failure (severe encephalopathy with GCS < 10, severe upper gastrointestinal bleeding, hemodynamic instability or unstable cardiac arrthythmia)
- Inability to cooperate/protect the airway
- Inability to clear respiratory secretions
- High risk of aspiration
- Recent facial surgery, trauma, or deformity
- Upper airway obstruction

NIV: Noninvasive ventilation, GCS: Glasgow coma scale.

Risk factors for Immediate NIV failure

Immediate	1. Weak cough reflex and/or excessive secretions	1a. Manual or mechanic chest physiotherapeutic techniques; b. Early fiberoptic bronchoscopy.
	2. Hypercapnic encephalopathy and coma	2a. Set a back-up rate ~ 15 b/min and/or use PCV; b. Decrease the F_iO2 level.
	3. Intolerance and psychomotor agitation	3. Judicious sedation
	4. "Fighting with the machine": Patient-ventilator asynchrony	4a. Closely monitor ventilator waveforms; b. Judicious sedation; c. Use a ventilator with an NIV platform; d. Change ventilatory parameters; e. Minimize air-leaks.

Risk factors for early NIV failure

Early Hypoxemic ARF	1. Baseline ABG and inability to correct gas exchange (P/F < 150)*
	2. Baseline severity scores (SAPS II >35)*
	3. The presence of ARDS/pneumonia/sepsis/multiorgan failure (OR: 4-28)*
	4. Increased respiratory rate (>25 breaths/min)*
	5. Miscellaneous: Delay between admission and NIV use, Number of fiberoptic bronchoscopes performed, Duration of NIV use, Increase in radiographic infiltrates within the first 24 hours, Causal diagnosis (as 'de novo')

Edema polmonare acuto cardiogeno

- NIV efficace > 90% dei casi
- SpO2 inferiore nei non responders (Tallman, Acad Emerg Med 2008)
- pH < 7.25; FE < 30%; PAS < 140 (Masip, Intensive Care Med 2003)
- pH < 7.03 (Shirakabe, J Cardiol 2010)
- PAS < 140; PAD < 90; Anemia; Età; Ipocapnia (Cosentini, Intensive Care Med, 2010)

Risk factors for early NIV failure

Hypercapnic ARF	1. Baseline ABG and inability to correct gas exchange (pH < 7.25)*
	2. Increased severity of disease
	3. Increased respiratory rate (>35 breaths/min, OR for baseline and after 2 hours of NIV: 2.66 and 4.95)*
	4. Mixed indices:
	GCS, APACHE II score, respiratory rate and pH
	Respiratory rate, random glucose level and APACHE II
	Anemia and World Health Organization Performance Status (WHO-PS)
	5. Miscellaneous: Poor nutritional status, Increased heart rate, Higher baseline C-reactive protein/white blood cell count, Lower serum potassium, Airway colonization by non-fermenting gram-negative bacilli

Monitoraggio

- Uso di standard di terapia e management
- Valutazione della risposta a terapia e ottimizzazione del settaggio del ventilatore
- Valutazione dei trend e delle condizioni di rischio per il paziente

Cosa monitorare?

- **Monitoraggio clinico**
  - Scala di Kelly (neurologico)
  - Esame del torace:
    - Fonendoscopio
    - ECO torace
  - Pattern respiratorio
  - Sincronia macchina paziente
  - Stato emodinamico
    - Valutazione cava

- **Parametri clinici**

Frequenza respiratroia

Frequenza cardiaca

Pressione arteriosa

Temperatura

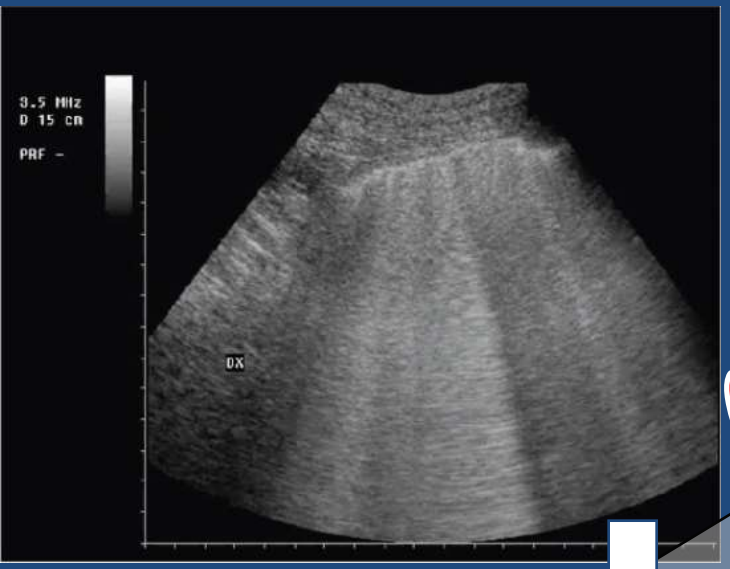
ECG

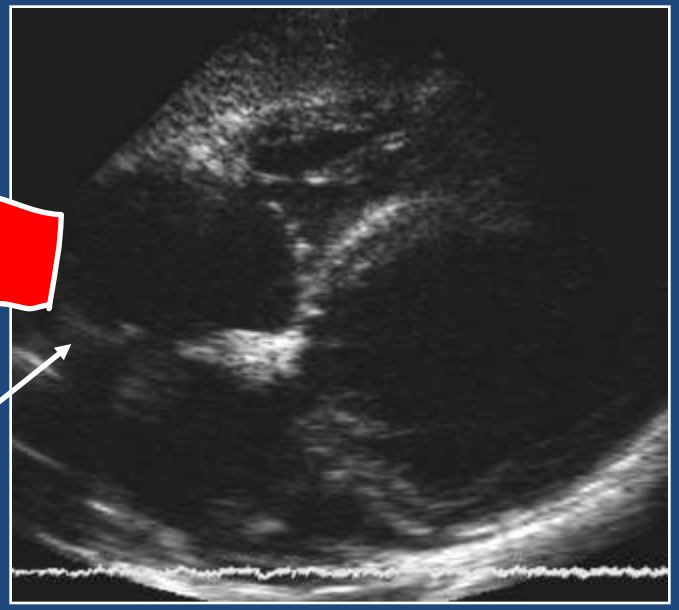
SpO_2

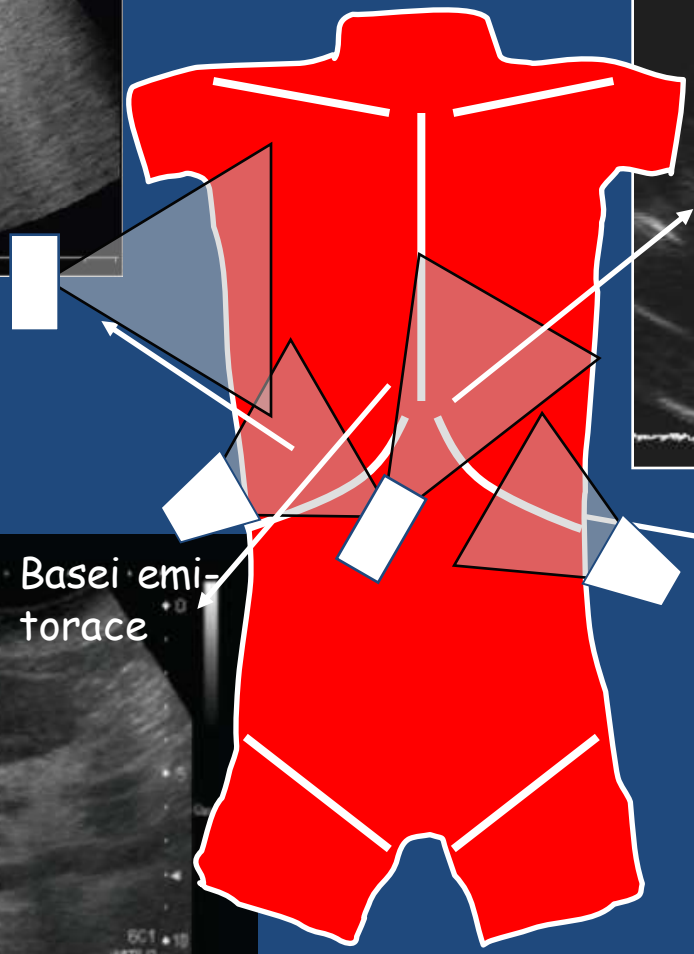
EGA
(timing variabile)

SCALA DI KELLY

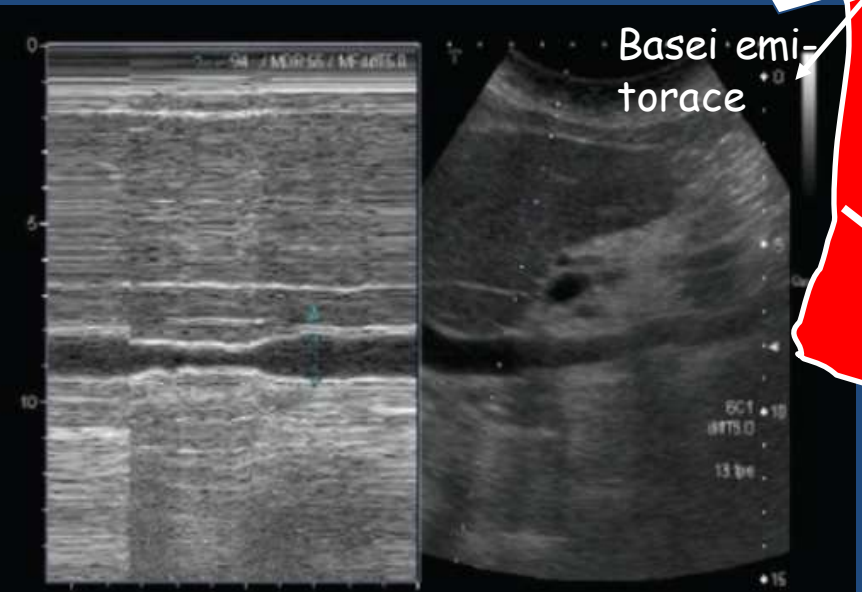
- Kelly 1 Pz sveglio, esegue tre ordini semplici
- Kelly 2 Pz sveglio, esegue solo ordini semplici
- Kelly 3 Pz sonnolento, risvegliabile a comando verbale
- Kelly 4 Pz sonnolento, risvegliabile a stimoli fisici
- Kelly 5 Pz in coma, senza alterazioni tronco-encefaliche
- Kelly 6 Pz in coma, con alterazioni tronco-encefaliche

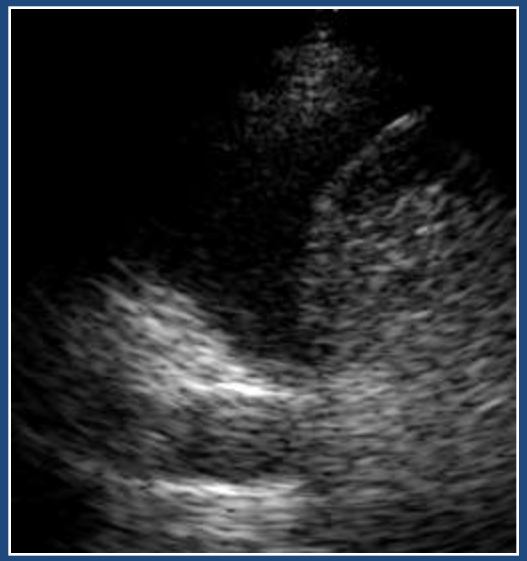






Basei emi-
torace





Frequenza respiratoria

-Correla con il grado di distress respiratorio

-FR elevata (>30 a/min) → il pz sta lavorando ai limiti superiori delle sue possibilità (↑ WOB)

- Riduzione della FR → migliora il pattern respiratorio (↓WOB)

Saturimetria (SpO_2)

- Parametro vitale da monitorizzare in continuo nelle prime 24 h (pulsossimetro)

-Con la CPAP → SpO2 ≥ 95%

-Con la NIV SpO2 > 90% (SpO2 tra 88-92 % nel BPCO)

- L'aumento è indice di miglioramento degli scambi gassosi solo se la FiO2 rimane costante!

Hb : ~ 15 mg
SpO2 = 100%

DO_2?

Hb : ~ 8 mg
SpO2 = 100%

EMOGAS ANALISI ARTERIOSA

Equilibrio acido-base

pH, HCO3, lattato, anion gap

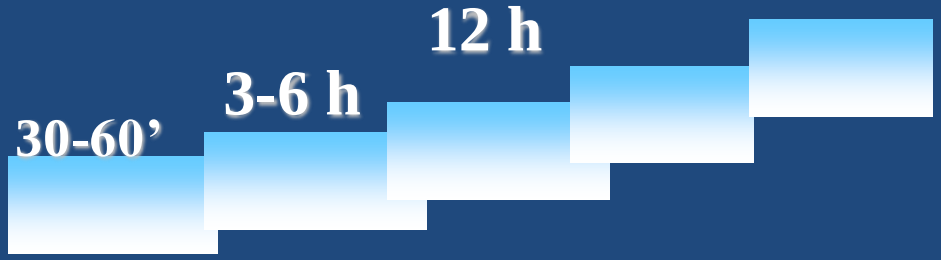
Ventilazione alveolare

PaCO2

Scambi gassosi

Δ(A-a)O_2, P/F, PaO2, SaO2

Monitoraggio emogasanalitico



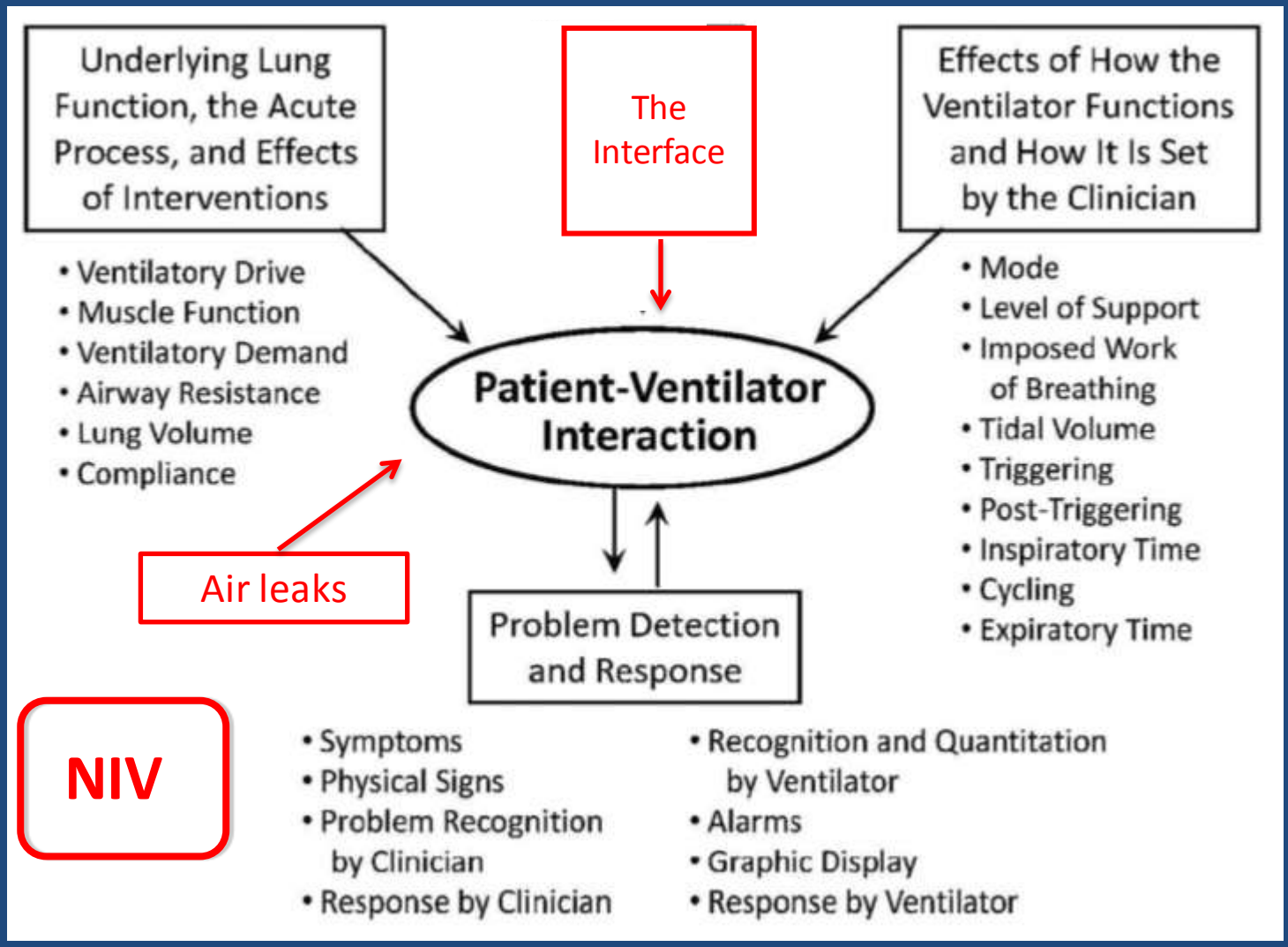
1 h da ogni modifica di parametri ventilatori

Se dopo 1-2 ore non miglioramento:

fatica muscolare, FR > 30, SpO2 < 90, pO2 < 60 mmHg, pH < 7,30, PaO2/FiO2 < 200

Patient-Ventilator Interaction

David J Pierson MD FAARC



Adapted from Respir Care. 2011 ;52:214-28

Patient-ventilator asynchrony is common but under-recognized and under-reported. PVA can occur with every mode of ventilation and in every phase of breathing.

The frequency of PAV is reported around 23%, but up to 93% of patients have at least one episode of PVA.

Camparota L .Bedside detection of patient-ventilation asynchrony. SIGNA VITAE 2016; 11(SUPPL 2): 31-34

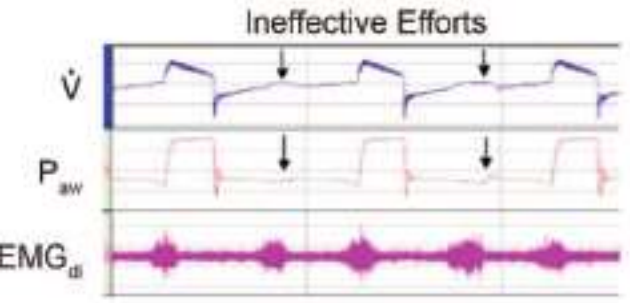
Table 2. Deleterious Effects of Patient-Ventilator Asynchrony

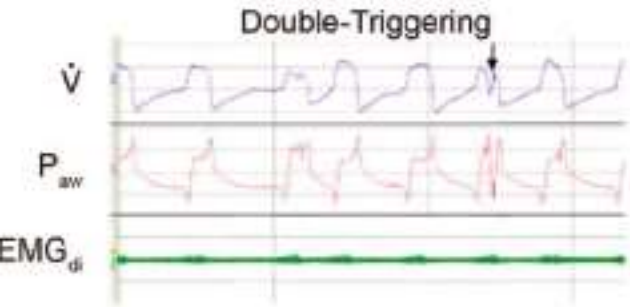
Patient fights the ventilator
More sedation required
Higher work of breathing
Muscle damage
Ventilation-perfusion problems
Dynamic hyperinflation
Delayed or prolonged weaning
Longer stay
Higher costs

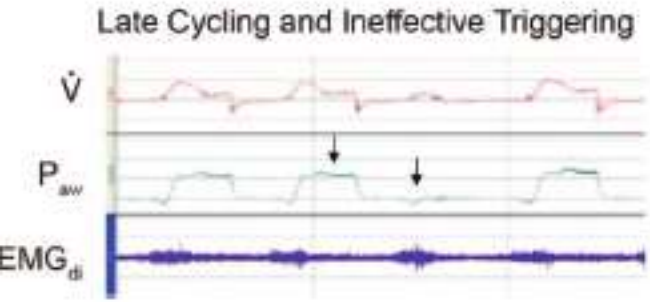
Nilsestuen and Hargett 2005

Patient–ventilator asynchrony during non-invasive ventilation for acute respiratory failure: a multicenter study

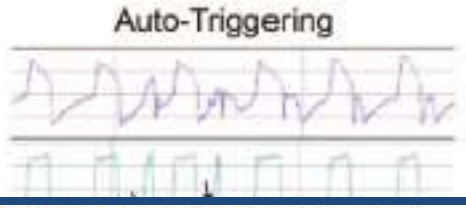
Vignaux et al. Intensive Care Med 2009



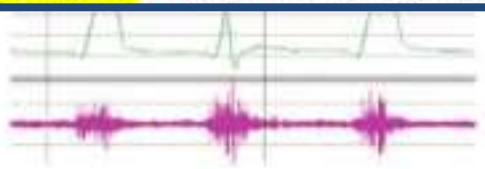




Asynchrony index (%) = number of asynchrony events/total respiratory rate × 100%



ratory parameters are shown in Table 2. The 2 factors predictive of an asynchrony index > 10% were the level of pressure support and the magnitude of leaks. The comfort score was higher in patients with an asynchrony index < 10%. No difference was observed in the intubation rate,



Choosing the Proper Interface for Positive Airway Pressure Therapy in Subjects With Acute Respiratory Failure

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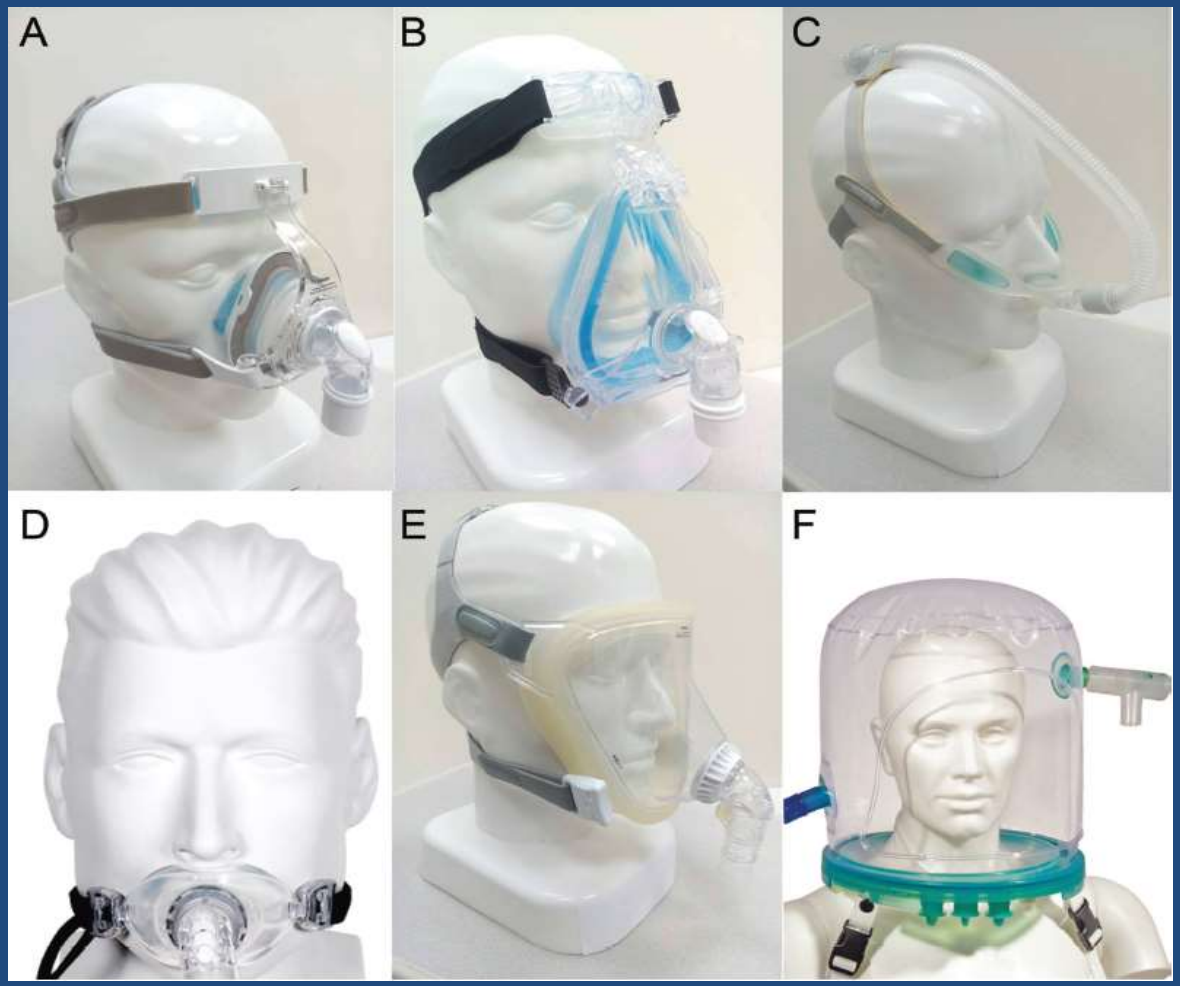
DOI: 10.4187/respcare.05787



Strategies to improve synchrony

Guarda il paziente

Parla con il paziente

Osserva la morfologia delle curve del ventilatore

Provvedi

Table 3. Strategies to Improve Synchrony With Noninvasive Ventilation

Trigger Synchrony
Adjust trigger sensitivity for the best balance between trigger effort and auto-triggering
Increase PEEP (expiratory positive airway pressure) to counterbalance auto-PEEP
Minimize unintentional leak with appropriate fitting of the interface
Treat underlying disease process (eg, bronchodilators to decrease airways resistance and air trapping)
Flow Synchrony
Use pressure-targeted or volume-targeted ventilation per patient comfort
Adjust inspiratory pressure with pressure-targeted ventilation; adjust flow and tidal volume with volume-targeted ventilation
Adjust rise time (pressurization rate) per patient comfort
Minimize unintentional leak with appropriate fitting of the interface
Reduce respiratory drive (eg, increase ventilation to treat acidosis)
Cycle Synchrony
Minimize unintentional leak with appropriate fitting of the interface
Use time-cycled (pressure control) rather than flow-cycled (pressure support) ventilation
Adjust flow cycle setting
Reduce pressure support setting
Treat underlying disease process (eg, bronchodilators to decrease airways resistance)
Mode Synchrony
Use backup rate if apnea or periodic breathing occurs


Grazie!