…E se non si svezza dalla NIV?

Paolo Groff

PS-MURG

Ospedale Civile “Madonna del Soccorso”

San Benedetto del Tronto

Caso clinico

Donna di 88 aa

Arriva in PS per dispnea ingravescente

In ambulanza: PA 110/70

SpO_2 82% (a.a.)

90% (O_2 con O.N. 24%)

Anamnesi

- Demenza senile con sd. ipocinetica
- Ipertensione Arteriosa
- Frequenti ricoveri per “bronchite”
- F.A. cronica

Tp. Domiciliare: Aricept, Seroquel, Exelon, Lanoxin, Kanrenol, Zestoretic. Cardirene. Seretide. Negli ultimi giorni Starcef, Bentelan.

Obiettività

FR 36/min; FC 120/min; Vigile non collaborante,

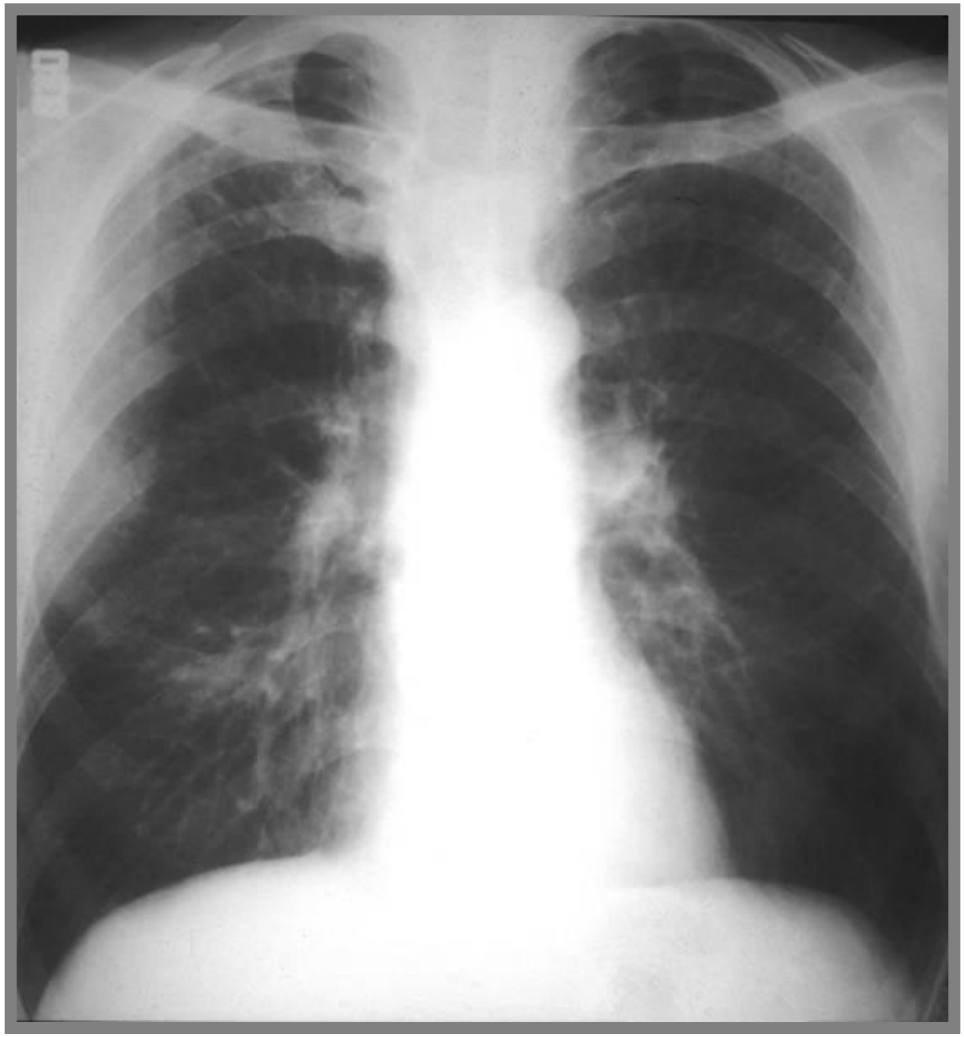
Respiro superficiale

- **MV marcatamente ridotto con sparsi sibili**
- **Cute fredda e sudata**
- **Marezzatura diffusa**

EGA

(O2-24%)

pH	7.25
pCO_2	83
pO2	66
HCO_3^-	36.4
Sat O_2	90



Terapia

- **Steroidi ev**
- **Diuretici EV**
- **Broncodilatatori aerosol in O_2**

NIV

- PSV + PEEP
- 10 + 3,5 con steps incrementali di 2 Cm H2O→ 23 + 5 Cm H2O
- Vtexp desiderato: 500 ml
- FiO2: 30%
- Trigger: -1L/min.
- Curva di pressurizzazione (rampa) ripida
- Trigger espiratorio: 50%

Dopo 8 ore

- EGA: pH 7.51; PaCO2 39; PaO2 81, HCO3 34.1 (FiO2 34%)
- Tentativo di sospensione: dispnea, reclutamento muscolatura acessoria, spO2: 86%
- Liquidi a 100 cc/h; Steroidi, Aerosol, antibiotici
- Riprende NIV con decremento PS
- Prosegue con cicli successivi di NIV per un totale di 42 ore
- Alla sospensione: pH 7.43; PaCO2 55; PaO2 65; HCO3 28.5 (FiO2 28%)

Se non mi si svezza dalla NIV:

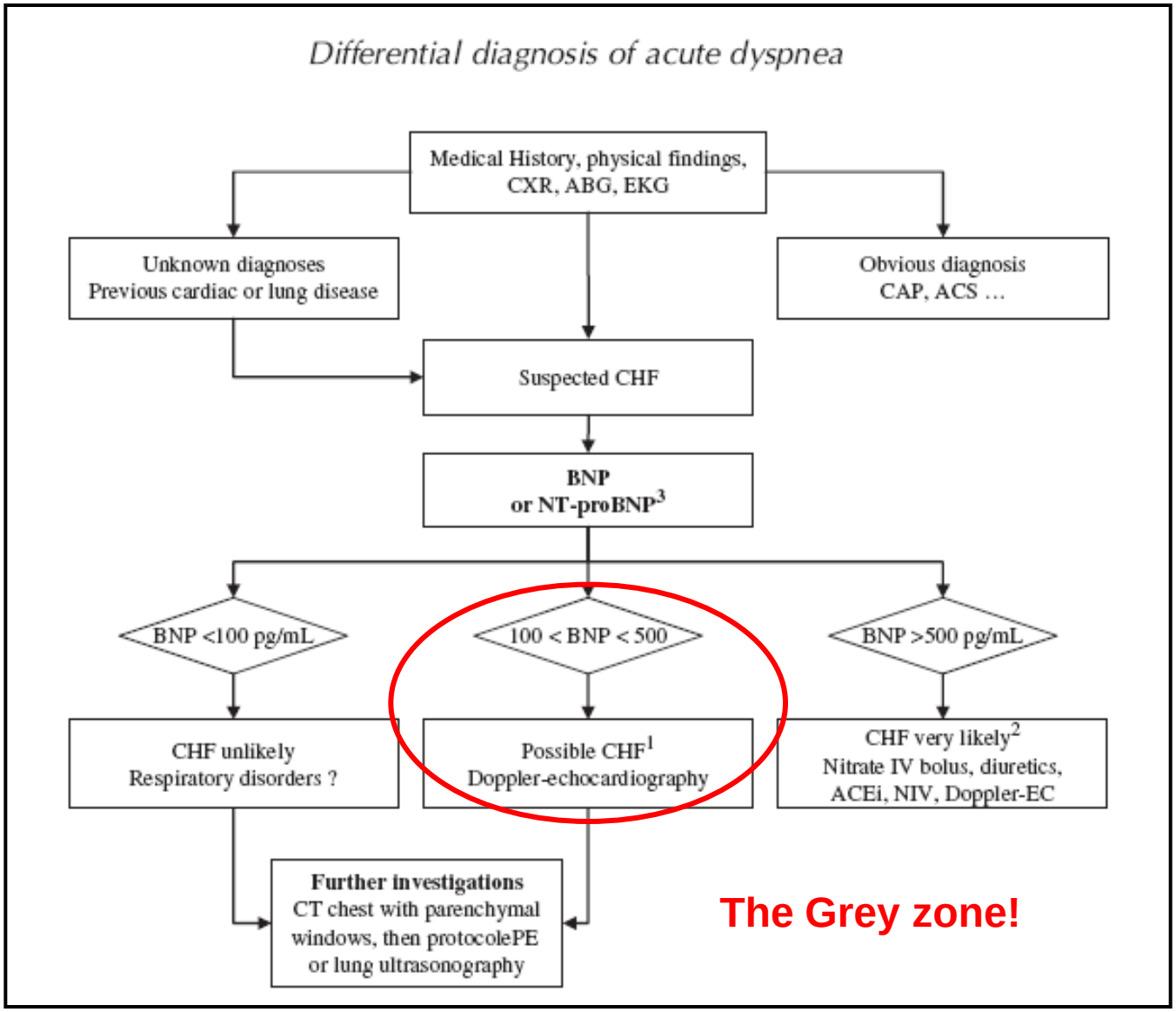
- Ho fatto una corretta diagnosi (e terapia) iniziale?
- Ho valutato la probabilità di successo della NIV e il rischio di fallimento tardivo?
- Ho curato il comfort del paziente e il sincronismo paziente-ventilatore?
- Ho interrotto correttamente la NIV?

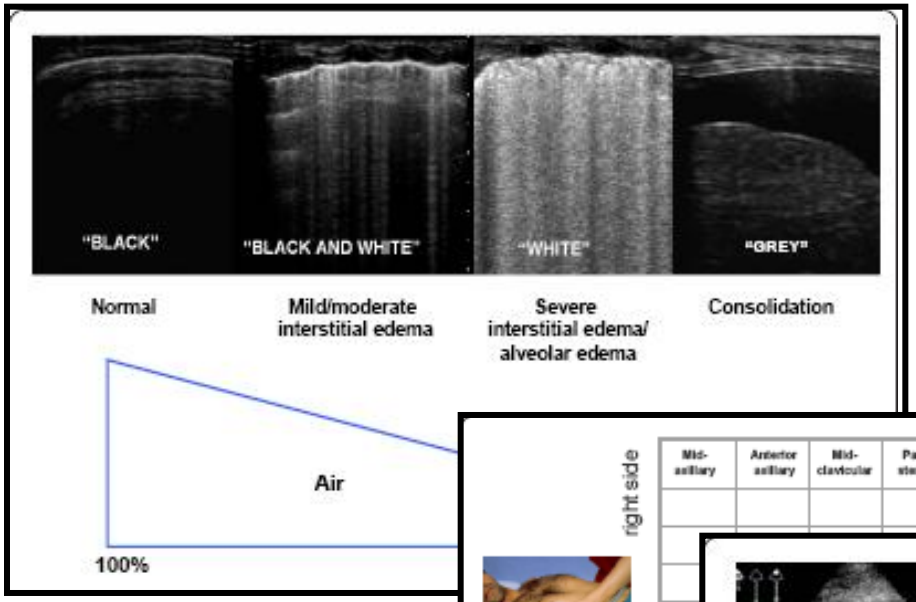
Natriuretic peptides (pros)

- BNP is an independent predictor of high left venticular end diastolic pressure and of capillary PA pressure (Maeda, Am Heart J 1998)
- Many studies have evaluated and validated NP in the diagnosis of CHF in acute dyspnea in middle aged pts (Maisel, NEJM 2002; Januzzi, Am J Cardiol 2005)
- NP testing alone is superior to clinical judgment alone for diagnosing CHF
- **There is evidence that a NP-guided strategy reduces hospital costs and improve outcome (Mueller, NEJM 2004; Moe, Circulation 2007; Throughton, Lancet 2000; Jourdain J Am Coll Cardiol 2007)**

Natriuretic Peptides (cons)

- Many factors influencing NP secretion (and cut-offs): Na intake, circadian variations, drugs and related hormones (corticosteroids, diuretics, ACEI, adrenergic agonists and antagonists), weight, gender, aging, renal function
- Many clinical conditions affecting NP circulating concentrations
- Different methods of analysis affecting variability: BNP vs. NT-proBNP; Bedside vs. laboratory tests; single antibodies (NT-proBNP) vs. different antibodies (BNP)


Differential diagnosis of acute dyspnea
Medical History, physical findings, CXR, ABG, EKG
Unknown diagnoses Previous cardiac or lung disease
Obvious diagnosis CAP, ACS …
Suspected CHF
BNP or NT-proBNP[3]
BNP <100 pg/mL
100 < BNP < 500
BNP >500 pg/mL
CHF unlikely Respiratory disorders ?
Possible CHF[1] Doppler-echocardiography
CHF very likely[2] Nitrate IV bolus, diuretics, ACEi, NIV, Doppler-EC
Further investigations CT chest with parenchymal windows, then protocolePE or lung ultrasonography
The Grey zone!



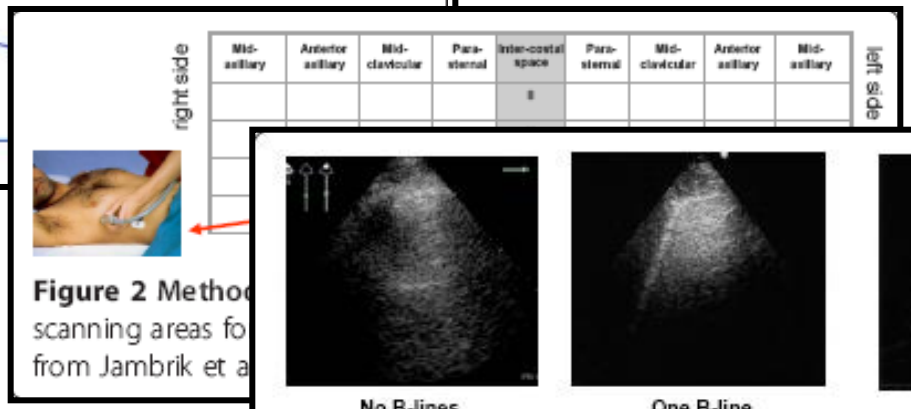


Figure 2 Metho
scanning areas fo
from Jambrik et a

Table 1 Scoring of B-lines

Score	Number of B-lines	Extravascular lung water
0	≤ 5	No sign
1	6 - 15	Mild degree
2	16 - 30	Moderate degree
3	> 30	Severe degree

(Modified from Picano et al, 2006 [16]).

Ultrasound lung comets for the differential diagnosis of acute cardiogenic dyspnoea: A comparison with natriuretic peptides ☆

L. Gargani [a,*], F. Frassi [a], G. Soldati [b], P. Tesorio [c], M. Gheorghiade [d], E. Picano [a]

[a] *Institute of Clinical Physiology, National Research Council, Pisa, Italy*
[b] *Hospital "Valle del Serchio", Castelnuovo Garfagnana, Lucca, Italy*
[c] *Clinica "Montevergine", Mercogliano, Avellino, Italy*
[d] *Division of Cardiology, Feinberg School of Medicine, Northwestern University, Chicago, IL, USA*

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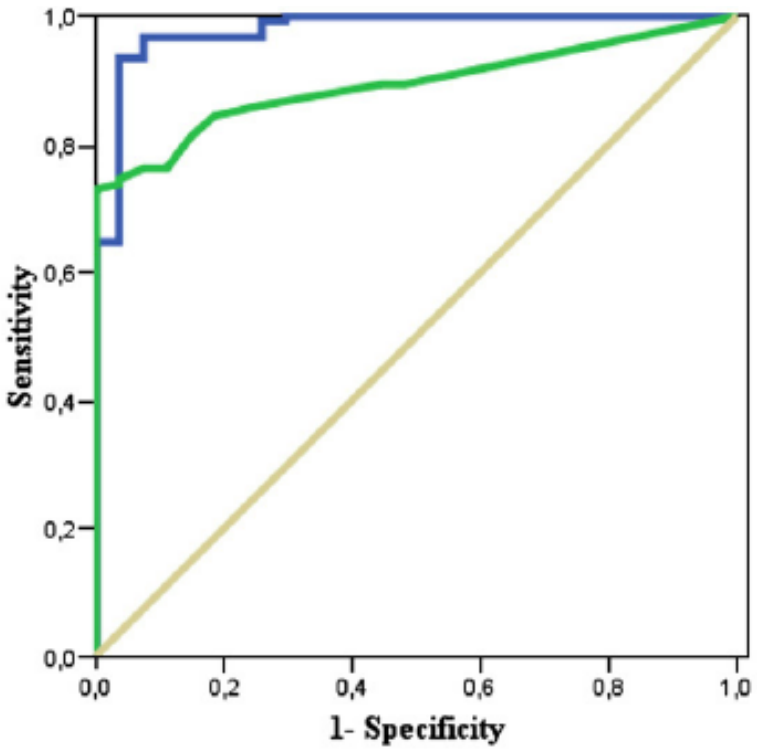
149 pts.



n=149
AUC NT-proBNP= .978 (.940-.995)
AUC ULCs= .893 (.832-.937)

NT-proBNP
Sensitivity:97%
Specificity:92.6%
Npv: 100%
Ppv:92%

ULCs
Sensitivity:81%
Specificity:85%
Npv: 45%
Ppv:97%

RESEARCH Open Access

Combination of lung ultrasound (a comet-tail sign) and N-terminal pro-brain natriuretic peptide in differentiating acute heart failure from chronic obstructive pulmonary disease and asthma as cause of acute dyspnea in prehospital emergency setting

Gregor Prosen[1,2], Petra Klemen[1,2,3], Matej Strnad[1,2] and Štefek Grmec[1,2,3,4*]

CRITICAL CARE

Prosen *et al. Critical Care* 2011, **15**:R114

218 pts.

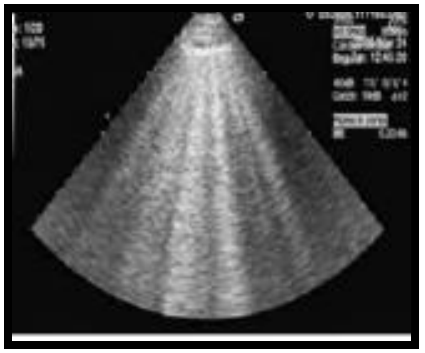


Table 3 Multiple logistic regression analysis of factors used for differentiation between HF-related and pulmonary-related acute dyspnea in prehospital emergency setting[a]

Factor	OR (95% CI)[b]	*P* value[c]
Ultrasound examination	53.7 (28.6 to 83.5)	< 0.001
NT-proBNP	14.3 (8.1 to 29.4)	< 0.001
Orthopnea	6.9 (1.9 to 18.39	< 0.001
Rales	5.1 (1.5 to 12.8)	0.014
Troponin T	2.1 (1.3 to 4.6)	0.018
$petCO_2$	7.6 (2.9 to 19.6)	< 0.001
HF medications	2.7 (1.3 to 5.1)	0.031
Asthma/COPD medications	0.12 (0.03 to 0.42)	0.028
Previous HF	7.4 (2.3 to 20.4)	< 0.001
Fever	0.17 (0.06 to 0.49)	0.017

Table 4 Test characteristics of ultrasound examination, modified Boston examination, NT-proBNP and combination of ultrasound examination and NT-proBNP[a]

Characteristic	Ultrasound examination[b]	Modified Boston criteria scoring	NT-proBNP	Ultrasound examination + NT-proBNP[c]	*P* value[d]
Sensitivity	100% (95% CI 98 to 100)	85% (95% CI 79 to 89)	92% (95% CI 88 to 95)	100% (95% CI 98 to 100)	< 0.01
Specificity	95% (95% CI 91 to 100)	86% (95% CI 82 to 90)	89% (95% CI 84 to 92)	100% (95% CI 97 to 100)	< 0.01
NPV	100% (95% CI 98 to 100)	80% (95% CI 77 to 85)	86% (95% CI 82 to 90)	100% (95% CI 98 to 100)	< 0.01
PPV	96% (95% CI 93 to 100)	90% (95% CI 86 to 93)	90% (95% CI 85 to 94)	100% (95% CI 96 to 100)	< 0.01
LR^+	20 (95% CI 1.98 to 89.94)	6.1 (95% CI 1.65 to 18.48)	8.36 (95% CI 1.72 to 33.86)	Infinite	< 0.01
LR^-	0	0.18 (95% CI 0.07 to 0.52)	0.09 (95% CI 0.02 to 0.23)	0	< 0.01
AUROC	0.94 (95% CI: 0.90 to 0.97)	0.86 (95% CI 0.80 to 0.91)	0.90 (95% CI 0.84 to 0.94)	0.99 (95% CI 0.98 to 1.00)	< 0.01

Mixed Acid-Base Disorders, Hydroelectrolyte Imbalance and Lactate Production in Hypercapnic Respiratory Failure: The Role of Noninvasive Ventilation

Claudio Terzano[1], Fabio Di Stefano[1], Vittoria Conti[1]*, Marta Di Nicola[2], Gregorino Paone[1], Angelo Petroianni[1], Alberto Ricci[1]

PLoS one

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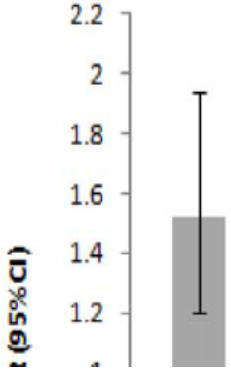

COPD exacerbation. Mixed respiratory acidosis–metabolic alkalosis patients were more likely to use NIV and were subjected to longer periods of ventilation compared to those with pure respiratory acidosis. The requirement for and duration of NIV was associated with low serum sodium and chloride, common findings in diuretic-induced metabolic alkalosis. The clinical

The use of diuretics for cardiovascular comorbidities was the main cause of metabolic alkalosis with hyponatremia and/or hypochloremia.

Table 4. Duration of NIV in groups of pa

Patients	Overall	NIV use (n, %)[a]	Hours of NIV (Mean ± SD)[b]
Overall	58	24, 41.4	42.4±10.5
Respiratory acidosis	36	10, 27.8	36.2±8.9
Mixed respiratory acidosis – metabolic alkalosis	17	11, 64.7	45.1±9.8
Mixed respiratory - metabolic acidosis	5	3, 60.0	53.3±4.1

Where Should Noninvasive Ventilation Be Delivered?

Nicholas S Hill MD

RESPIRATORY CARE • JANUARY 2009 VOL 54 NO 1

Table 2. Risk Factors for NIV Failure in Patients With Acute Hypercapnic Respiratory Failure

Poor neurologic score (Glasgow Coma Score < 11)
Tachypnea (> 35 breaths/min)
pH < 7.25
APACHE score > 29
Asynchronous breathing
Edentulous
Excessive air leak
Agitation
Excessive secretions
Poor tolerance
Poor adherence to therapy
No initial improvement within first 2 h of NIV:
No improvement in pH
Persistent tachypnea
Persistent hypercapnia

NIV = noninvasive ventilation
APACHE = Acute Physiology and Chronic Health Evaluation
(Based on data in References 20-22.)

Table 3. Risk Factors for NIV Failure in Patients With Acute Hypoxemic Respiratory Failure

Diagnosis of ARDS or pneumonia
Age > 40 y
Hypotension (systolic blood pressure < 90 mm Hg)
Metabolic acidosis (pH < 7.25)
Low oxygenation index (P_{aO_2}/F_{IO_2})
Simplified Acute Physiology Score II > 34
Failure to improve oxygenation within first hour of NIV (P_{aO_2}/F_{IO_2} > 175 mm Hg)

ARDS = acute respiratory distress syndrome
F_{IO_2} = fraction of inspired oxygen
(Based on data in References 23-25.)

Incidence and causes of non-invasive mechanical ventilation failure after initial success

Maurizio Moretti, Carmela Cilione, Auro Tampieri, Claudio Fracchia, Alessandro Marchioni, Stefano Nava

Thorax 2000;55:819–825

Table 1 Mean (SD) demographic, clinical, and functional characteristics recorded at baseline and during non-invasive mechanical ventilation (NIMV) at 1 and 24 hours in patients with COPD grouped according to the success of NIMV (group 1 = successful, group 2 = "late respiratory failure")

	Group 1 (n=106)	*Group 2 (n=31)*	*p value*
Age (years)	70 (8.8)	70 (10.1)	NS
pH			
On admission	7.25 (0.07)	7	
1 h	7.30 (0.06)	7	
24 h	7.36 (0.06)	7	
$PaCO_2$ (mm Hg)			
On admission	87.6 (17.1)	8	
1 h	76.6 (13.6)	7	
24 h	68.8 (13.2)	6	
Sao_2/FIo_2 at admission	2.0 (0.6)	1	
Heart rate (bpm)	97 (17.8)	1	
Respiratory rate (bpm)	26 (5.6)	2	
Arterial pressure (mm Hg)	105 (15)	9	
Compliance score	3.7 (1.0)	3	
APACHE II score	22.0 (4.5)	2	
ADL score	2.1 (0.8)	1	
No of complications of admission	0.45 (0.6)	0	
Community acquired pneumonia (no (%))	8 (7.5%)	7	
Albumin (mg/dl)	34.2 (3.3)	3	
Sodium (mEq/l)	139 (3.6)	1	
Potassium (mEq/l)	4.2 (0.6)	4	
Fasting glycaemia (mg/dl)	83.5 (8.8)	9	
FEV_1 (% pred)	17.6 (9.1)	1	
FVC (% pred)	45.6 (8.2)	4	
FEV_1/FVC	38.5 (7.6)	3	

ADL = activity of daily living score; FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity. Dynamic lung volumes were recorded in 61 and 18 patients of groups 1 and 2, respectively.

The logistic analysis performed on the variables recorded at the time of admission showed that activities of daily living, the number of complications, and pH recorded on admission strongly predicted the occurrence of "late failure" during NIMV. In the logistic analysis metabolic complication was, among the other complications recorded on admission, the only independently significant predictor of late failure.

Dependency on mask ventilation after acute respiratory failure in the intermediate care unit

A. Cuvelier*,¶, C. Viacroze*, J. Bénichou#, L.C. Molano*, M-F. Hellot#, D. Benhamou* and J-F. Muir*,¶

Eur Respir J 2005; 26: 289–297

TABLE 3 Long-term dependency on noninvasive ventilation (LTD-NIV) following hospitalisation in the respiratory intermediate care unit for an episode of acute hypercapnic respiratory failure depending on the arterial blood gases at stable state during the 6 mo

Independent predictive factors of LTD-NIV

Based on a multivariate analysis with stepwise logistic regression, lower baseline pH values and a noninfectious cause for the AHRF emerged as the only variables that were independently associated with subsequent LTD-NIV in the whole population of patients. The mutually adjusted odds

	No L						V	p-value
Subjects n								
$Pa,O_2/FI,O_2$	241.2						5.2	0.03
pH	7.42						.03	0.0001
Pa,CO_2 kPa	6.24±1.16	6.82±0.9	0.0018	6.45±1.45	6.74±1.10	0.39	6.0±0.8	6.9±0.8	0.0008
HCO_3 mEq·L^{-1}	30.7±4.8	30.8±3.0	0.58	31.1±5.9	29.8±2.7	0.78	30.4±3.6	31.1±3.1	0.35

Data are presented as mean±SD or n. COPD: chronic obstructive pulmonary disease; Pa,O_2: arterial oxygen tension; FI,O_2: inspired oxygen fraction; Pa,CO_2: carbon dioxide arterial tension; HCO_3: hydrogen carbonate.

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A. Cuvelier*,¶, C. Viacroze*, J. Bénichou#, L.C. Molano*, M-F. Hellot#, D. Benhamou* and J-F. Muir*,¶

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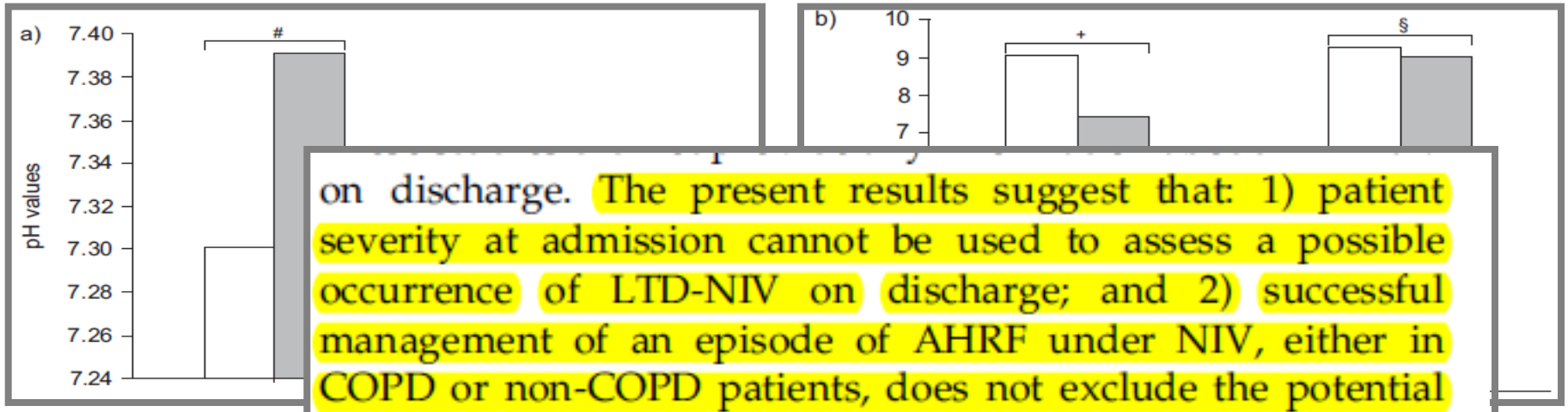
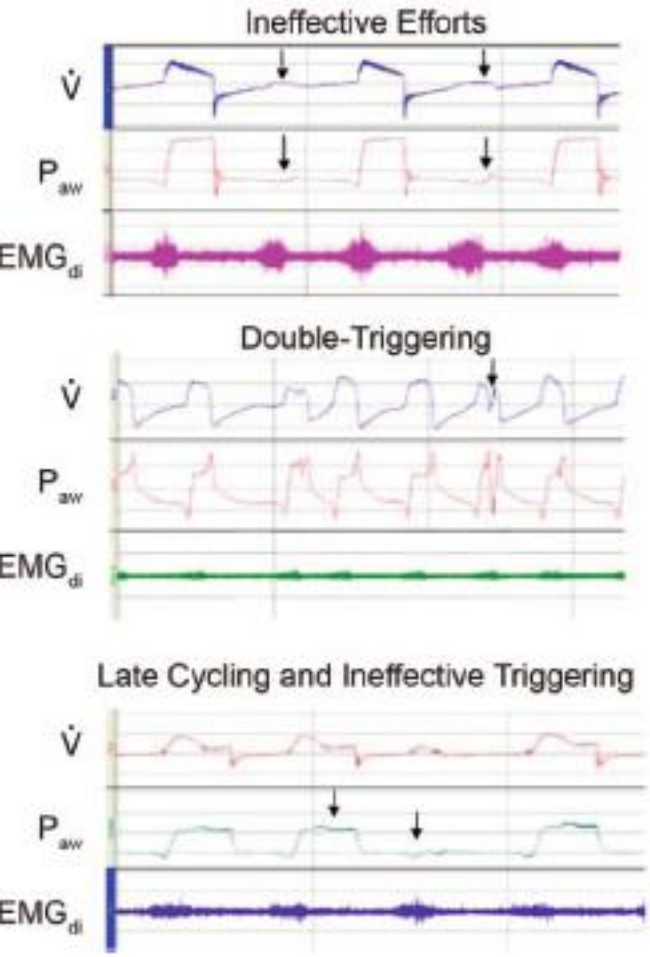


on discharge. The present results suggest that: 1) patient severity at admission cannot be used to assess a possible occurrence of LTD-NIV on discharge; and 2) successful management of an episode of AHRF under NIV, either in COPD or non-COPD patients, does not exclude the potential need for long-term NIV.

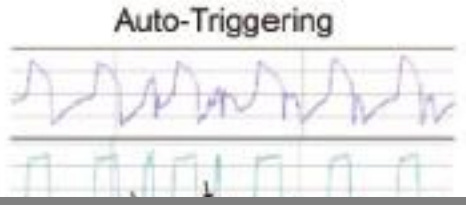
Patient-Ventilator Interaction During Noninvasive Ventilation

Dean R Hess PhD RRT FAARC

Respir Care 2011;56(2):153–165.



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,

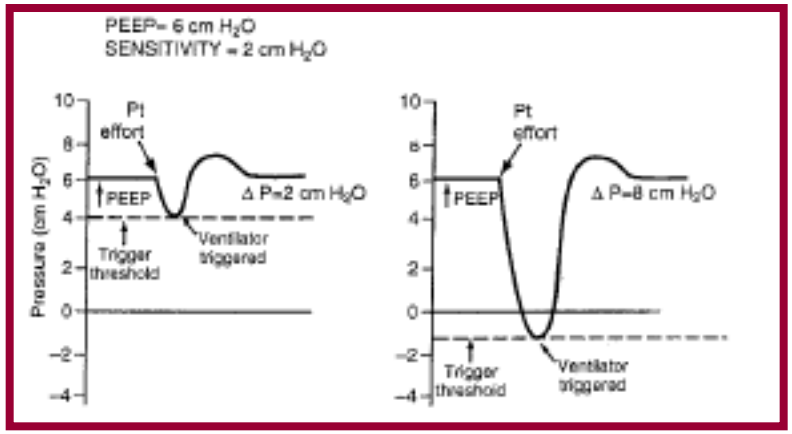
The interface and asynchrony

- Leaks: reduce the efficiency of NIV, pt tolerance, pt-ventilator asynchrony
- The interface can contribute to mechanical dead space and CO2 rebreathing (helmet)
- Worse triggering and increased inspiratory muscolar effort with the helmet
- Possible interface-ventilator mismatch when mixing vented masks from one manufacturer with ventilators from another manufacturer

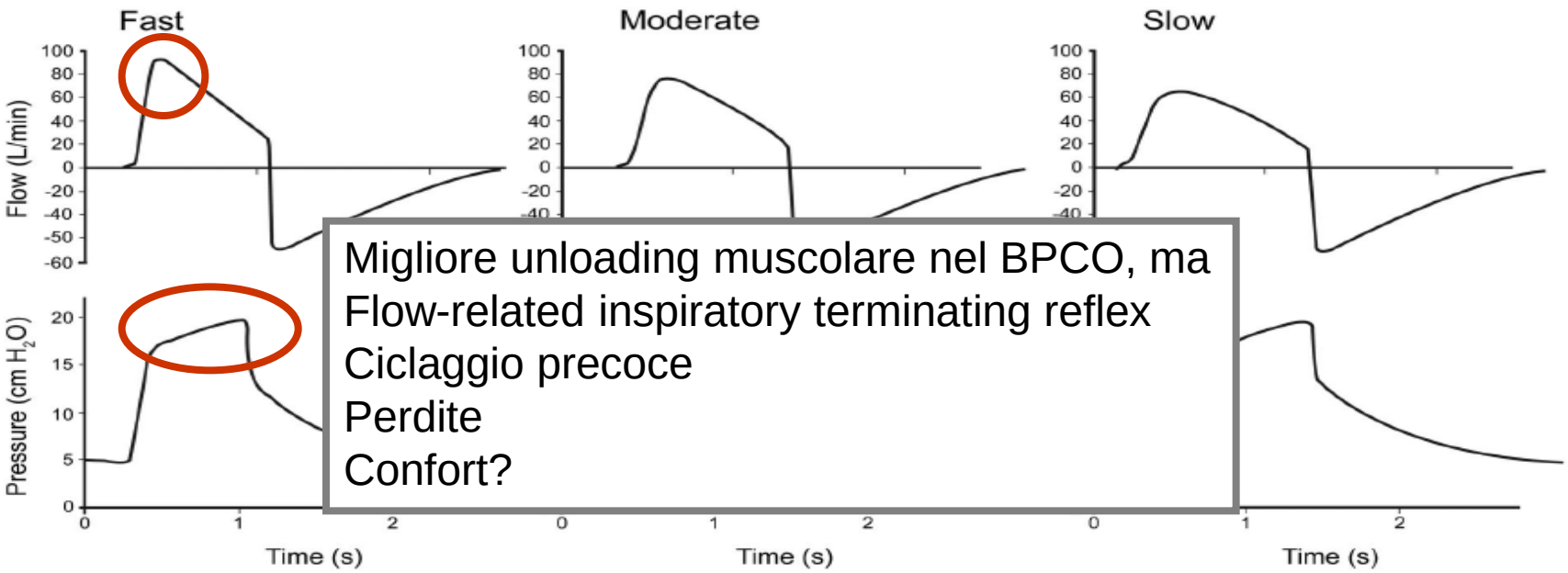
The ventilator and asynchrony

- With the single-circuit bilevel ventilators there is the potential for rebreathing: position of the leak, O2 inlet, expyratory pressure, plateau exhalation valve
- Leak detection algorithms to adjust for leak changes following IPAP and EPAP changes, as well as breath by breath changes due to fit of the interface

Trigger, flow, cycle asynchrony



perdite: riduzione del tempo espiratorio→aumento PEEPi→asincronia del trigger→sforzi inefficaci→prolungamento fase espiratoria→ decremento PEEPi→variabilità FR e Vt



Migliore unloading muscolare nel BPCO, ma
Flow-related inspiratory terminating reflex
Ciclaggio precoce
Perdite
Confort?

Strategies to improve synchrony

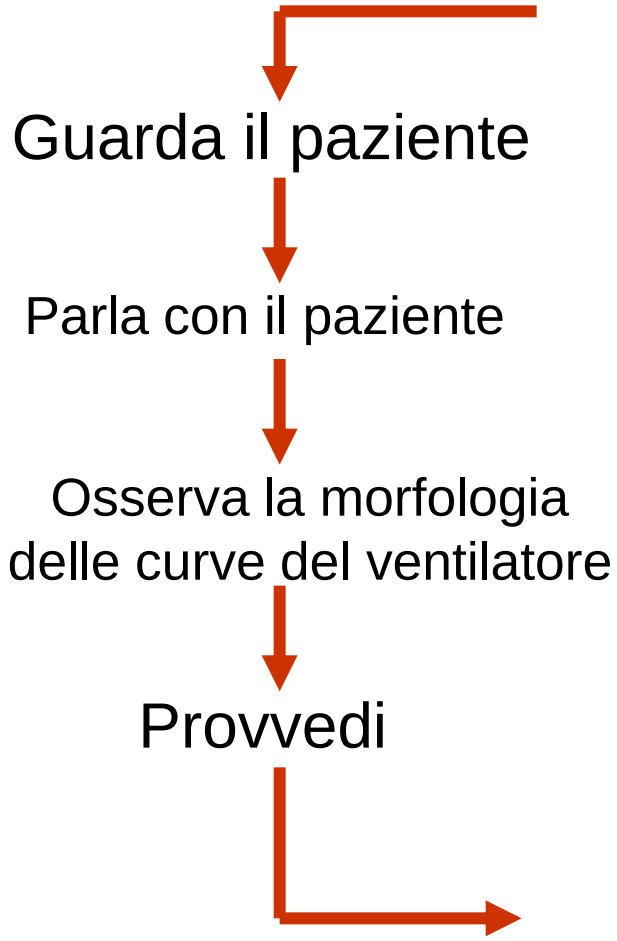


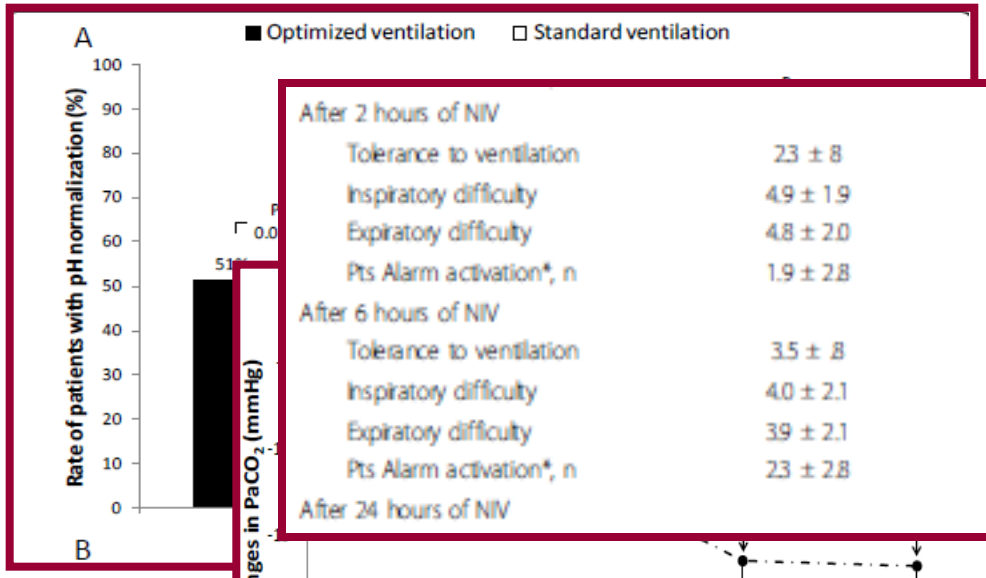
Table 3. Strategies to Improve Synchrony With Noninvasive Ventilation

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

Optimization of ventilator setting by flow and pressure waveforms analysis during noninvasive ventilation for acute exacerbations of COPD: a multicentric randomized controlled trial

Di Marco et al. Critical Care 2011, 15:R283
http://ccforum.com/content/15/6/R283

Fabiano Di Marco[1*], Stefano Centanni[1], Andrea Bellone[2], Grazia Messinesi[3], Alberto Pesci[3], Raffaele Scala[4], Andreas Perren[5] and Stefano Nava[6]



After 2 hours of NIV				
Tolerance to ventilation	2.3 ± 8	2.6 ± .7	1.9 ± .9	**.001**
Inspiratory difficulty	4.9 ± 1.9	4.7 ± 1.9	5.0 ± 1.9	.494
Expiratory difficulty	4.8 ± 2.0	4.8 ± 2.0	4.9 ± 2.0	.875
Pts Alarm activation*, n	1.9 ± 2.8	1.0 ± 1.9	3.3 ± 3.2	**.004**
After 6 hours of NIV				
Tolerance to ventilation	3.5 ± .8	3.7 ± .7	3.3 ± .9	.092
Inspiratory difficulty	4.0 ± 2.1	3.7 ± 2.1	4.2 ± 2.2	.442
Expiratory difficulty	3.9 ± 2.1	3.5 ± 2.0	4.4 ± 2.2	.124
Pts Alarm activation*, n	2.3 ± 2.8	1.1 ± 1.8	3.4 ± 3.2	**.015**
After 24 hours of NIV				

Table 4 Changes in ventilator setup

	All patients	Optimized ventilation	Standard ventilation	*P*
At the beginning of NIV				
PEEP, cmH_2O	5.1 ± 1.4	5.5 ± 1.7	4.7 ± .9	**.003**
PS, cmH_2O	13.6 ± 3.4	14.0 ± 3.7	13.2 ± 3.1	.152
Insp trigger*, L/minute	3.2 ± 2.0	2.3 ± 1.8	4.0 ± 1.7	**.002**
Expiratory trigger, %	40 ± 9	38 ± 9	42 ± 8	.086
Speed of pressurization	1.8 ± .6	1.7 ± .6	2.0 ± .4	**.024**
After 30 minutes of NIV				
PEEP, cmH_2O	5.4 ± 1.3	5.9 ± 1.4	4.9 ± .9	**.011**
PS, cmH_2O	14.3 ± 3.5	14.7 ± 3.7	14.0 ± 3.4	.604
Insp trigger*, L/minute	2.9 ± 2.0	2.0 ± 1.7	3.8 ± 1.8	**.001**
Expiratory trigger, %	40 ± 12	40 ± 14	40 ± 9	.600
Speed of pressurization	1.6 ± .5	1.4 ± .5	1.7 ± .4	**.011**
After 2 hours of NIV				
PEEP, cmH_2O	5.4 ± 1.2	5.9 ± 1.3	4.9 ± .9	**.030**
PS, cmH_2O	14.8 ± 3.9	14.9 ± 4.2	14.7 ± 3.7	.820
Insp trigger*, L/minute	2.9 ± 2.0	1.9 ± 1.7	4.1 ± 1.7	**0.001**
Expiratory trigger, %	37 ± 12	36 ± 14	38 ± 10	.337
Speed of pressurization	1.4 ± .5	1.3 ± .5	1.6 ± .5	.064
After 6 hours of NIV				
PEEP, cmH_2O	5.4 ± 1.4	5.8 ± 1.6	5.1 ± 1.0	**.015**
PS, cmH_2O	15.3 ± 4.0	15.5 ± 4.2	15.1 ± 4.0	.925
Insp trigger*, L/minute	3.0 ± 2.0	2.0 ± 1.7	3.9 ± 1.8	**.001**
Expiratory trigger, %	37 ± 11	36 ± 12	39 ± 10	.143
Speed of pressurization	1.6 ± .5	1.5 ± .6	1.7 ± .5	.134
After 24 hours of NIV				
PEEP, cmH_2O	5.4 ± 1.5	6.0 ± 1.8	4.9 ± .9	**.002**
PS, cmH_2O	14.8 ± 3.9	15.4 ± 4.2	14.3 ± 3.6	.426
Insp trigger*, L/minute	3.1 ± 1.9	2.4 ± 1.8	4.0 ± 1.8	**.009**
Expiratory trigger, %	37 ± 11	37 ± 12	37 ± 10	.708
Speed of pressurization	1.7 ± .6	1.5 ± .5	1.9 ± .6	.089

Di Marco, Nava 2011

Dependency on mask ventilation after acute respiratory failure in the intermediate care unit

A. Cuvelier*,¶, C. Viacroze*, J. Bénichou#, L.C. Molano*, M-F. Hellot#, D. Benhamou* and J-F. Muir*,¶

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1. **Condizioni di stabilità clinica + controllo dell'evento scatenante**
2. **Progressiva riduzione della sola NIV diurna sotto controllo clinico e ABG**
3. **Sospensione NIV notturna in base a ABG del giorno successivo a sosp. NIV notturna**
4. **NIV sospesa in assenza di acidosi (pH < 7.35) o dispnea negli <u>8 giorni</u> consecutivi**
5. **Se il weaning fallisce, si ripete la procedura con le stesse modalità**

NIV duration

Patients who benefit from NIV during the first hours of treatment should receive NIV for as long as possible during the first 24 hours [A]

Treatment should last until the acute cause has resolved, commonly 2-3 days [C]

In patients in whom NIV is successful (pH ≥7.35 achieved, resolution of underlying cause and symptoms, respiratory rate normalized) it is appropriate to start a weaning plan [C]

Conclusioni

- La mortalità intra-ospedaliera e l'intubazione non sono gli unici indici di risultato della NIV. Dal 20 al 30% dei pazienti peggiora dopo un iniziale successo o necessita di terapia prolungata
- E' possibile che ciò dipenda da un non corretto inquadramento iniziale del paziente (stato emodinamico), ma necessitano indici prognostici affidabili di questo tipo di outcome
- Un settaggio dei parametri di ventilazione individualizzato per ottenere un buon sincronismo paziente-ventilatore può essere un approccio valido
- Non disponiamo ancora di una procedura validata e "standardizzata" di weaning dalla NIV


Grazie!