

Gemelli

Monitoraggio NIV

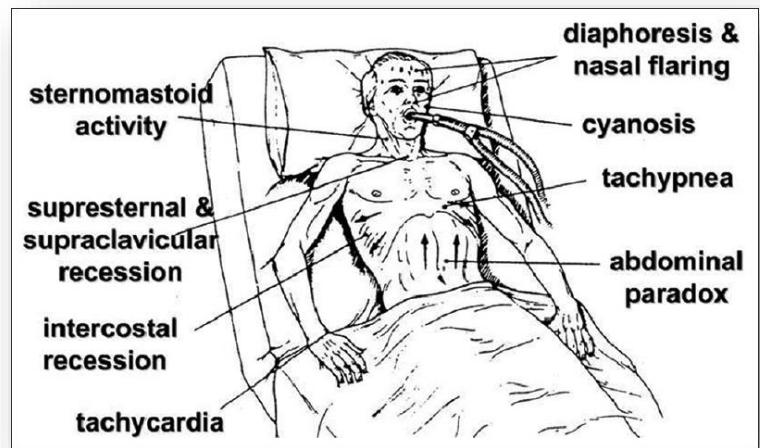


Mariano Alberto Pennisi

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Valutazione clinica

- Confort, tolleranza dell' interfaccia
- Dispnea e uso dei muscoli accessori
- Coscienza-sensorio (GCS, Kelly–Matthay score)
- Capacità di proteggere le vie aeree
- Distensione gastrica
- Scorse di valutazione della sedazione e del delirio(RASS, SAS, RSS)
- Score di gravità (SAPS II APACHE II)



Kelly–Matthay score (KMS)

Grade 1 = alert, follows complex 3-step command
Grade 2 = alert, follows simple commands
Grade 3 = lethargic, but arousable and follows simple commands
Grade 4 = stuporous (only intermittently follows simple commands even with vigorous attempts to arouse the patient)
Grade 5 = comatose, brain stem intact
Grade 6 = comatose with brain stem dysfunction

Noninvasive Versus Conventional Mechanical Ventilation

An Epidemiologic Survey

Am J Respir Crit Care Med Vol 163, pp 874-880, 2001
Internet address: www.atsjournals.org

ANNALISA CARLUCCI, JEAN-CHRISTOPHE RICHARD, MARC WYSOCKI, ERIC LEPAGE, LAURENT BROCHARD
and the SRLF Collaborative Group on Mechanical Ventilation

Paris XII Université, Department of Biostatistics, Medical Intensive Care Unit, Institut National de la Recherche et de la Santé Medicale U 492,
Henri Mondor Hospital, Créteil, France

A prospective survey was performed over a period of 3 wk among 42 intensive care units to assess the incidence of use and effectiveness of noninvasive mechanical ventilation (NIV) in clinical practice. All patients requiring ventilatory support for acute respiratory failure (ARF), either with endotracheal intubation (ETI) or NIV, were included. Ventilatory support was required in 689 patients, 581 with ETI and 108 (16%) with NIV (35% of patients not intubated on admission). Reasons for mechanical ventilation were coma (30%), cardiogenic pulmonary edema (7%), and hypoxemic (48%) and hypercapnic ARF (15%). NIV was never used for patients in coma (who were excluded from further analysis), but was used in 14% of patients with hypoxemic ARF, in 27% of those with pulmonary edema, and in 50% of those with hypercapnic ARF. NIV was followed by ETI in 40% of cases. The incidence of both nosocomial pneumonia (10% versus 19%, $p = 0.03$), and mortality (22% versus 41%, $p < 0.001$) was lower in NIV patients than in those with ETI. After adjusting for differences at baseline, Simplified Acute Physiology Score (SAPS) II (odds ratio [OR] = 1.05 per point; confidence interval [CI]: 1.04 to 1.06), McCabe/Jackson score (OR = 2.18; CI: 1.57 to 3.03), and hypoxemic ARF (OR = 2.30; CI: 1.33 to 4.01) were identified as risk factors explaining mortality; success of NIV was associated with a lower risk of pneumonia (OR = 0.06; CI: 0.01 to 0.45) and of death (OR = 0.16; CI: 0.05 to 0.54). In NIV patients, SAPS II and a poor clinical tolerance predicted secondary ETI. Failure of NIV was associated with a longer length of stay. In conclusion, NIV can be successful in selected patients, and is associated with a lower risk of pneumonia and death than is ETI.

TABLE 2. COMPARISON OF SUCCESS AND FAILURE OF NONINVASIVE VENTILATION IN UNIVARIATE ANALYSIS

	Success (n = 65)	Failure* (n = 43)	p Value
Age, yr	62 ± 16	66 ± 15	n.s.
SAPS II	30 ± 11	45 ± 27	< 0.005
ABG before ventilation			
Pa _{CO₂} , mm Hg	54 ± 23	63 ± 30	n.s.
pH	7.36 ± 0.09	7.30 ± 0.10	< 0.01
Pa _{O₂} /Fi _{O₂} , mm Hg	227 ± 79	206 ± 119	n.s.
ABG at Day 1			
Pa _{CO₂} , mm Hg	57 ± 19	60 ± 31	n.s.
pH	7.37 ± 0.08	7.34 ± 0.09	n.s.
Pa _{O₂} /Fi _{O₂} , mm Hg	232 ± 92	200 ± 115	n.s.
Copious secretions, yes/no [†]	9/55	14/27	< 0.05
Encephalopathy (no or moderate/pronounced) [‡]	43/17	18/19	< 0.01
Tolerance, good/poor	59/6	27/16	< 0.001
Leaks, minor/large	59/6	31/12	< 0.004
Mask, facial/nasal	56/9	40/3	n.s.

Definition of abbreviations: ABG = arterial blood gases; Fi_{O₂} = fraction of inspired oxygen; Pa_{CO₂} = arterial carbon dioxide tension; SAPS = Simplified Acute Physiology Score.

* Failure = need for endotracheal intubation.

[†] Three missing values.

[‡] Eleven missing values.

Scarsa tolleranza alla NIV

- Rassicurare il paziente
- Scegliere correttamente tipo e dimensioni dell'interfaccia
- Controllare la tenuta, regolare la tensione dei sistemi di ancoraggio (maschera) o delle cinghie (casco)
- Ottimizzare il supporto ventilatorio ( pressioni  riduce le perdite)

Valutare una lieve sedazione

Sedation agitation scale

Score	State	Behaviors
7	Dangerous Agitation	Pulling at ET tube, climbing over bedrail, striking at staff, thrashing side-to-side
6	Very Agitated	Does not calm despite frequent verbal reassurance, requires physical restraints
5	Agitated	Anxious or mildly agitated, attempting to get out of bed, down to verbal instructions
4	Calm and Cooperative	Calm, awakens easily, follows commands, interacts with family
3	Sedated	Difficult to arouse, awakens to verbal stimuli, shaking but drifts off
2	Very Sedated	Arouses to physical stimuli but does not follow commands
1	Unarousable	Minimal or no response to noxious stimuli, cannot communicate or follow commands

Richmond Agitation Sedation Scale (RASS)

Target RASS	RASS Description
+4	Combative, violent, danger to staff
+3	Pulls or removes tube(s) or catheters; aggressive
+2	Frequent nonpurposeful movement, fights ventilator
+1	Anxious, apprehensive, but not aggressive
0	Alert and calm
-1	awakens to voice (eye opening/contact) >10 sec
-2	light sedation, briefly awakens to voice (eye opening/contact) <10 sec
-3	moderate sedation, movement or eye opening. No eye contact
-4	deep sedation, no response to voice, but movement or eye opening to physical stimulation
-5	Unarousable, no response to voice or physical stimulation

Lung (2012) 190:597–603
DOI 10.1007/s00408-012-9403-y

REVIEWS

Noninvasive Positive Pressure Ventilation for Acute Respiratory Failure in Delirious Patients: Understudied, Underreported, or Underappreciated? A Systematic Review and Meta-analysis

Michael Charlesworth · Mark W. Elliott · John D. Holmes

Study or Subgroup	Delirium		No delirium		Weight	Risk Ratio M-H, Fixed, 95% CI	Year				
	Events	Total	Events	Total							
Carlucci 2001	19	36	18	61	77.9%	1.79 [1.09, 2.94]	2001				
Campo 2010	9	9	5	18	22.1%	3.28 [1.60, 6.73]	2010				
Total (95% CI)		45	79 100.0%		2.12 [1.41, 3.18]						
Total events		28	23								
Heterogeneity: Chi ² = 1.87, df = 1 (P = 0.17); I ² = 47%											
Test for overall effect: Z = 3.62 (P = 0.0003)											

Confusion Assessment Method for the ICU (CAM-ICU) Flowsheet

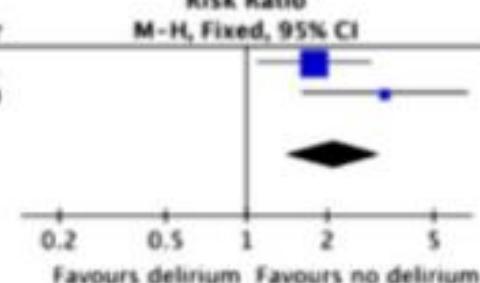
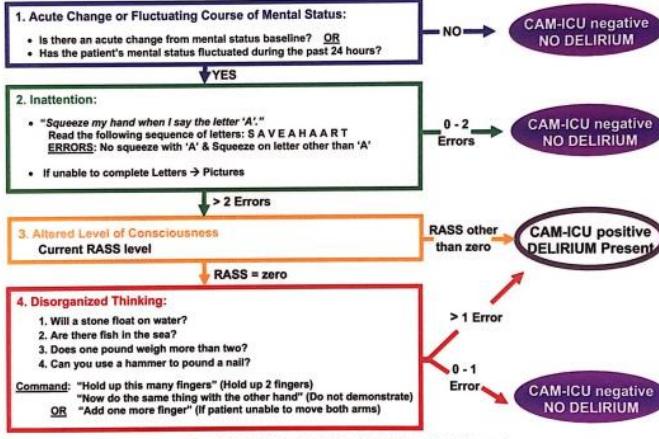
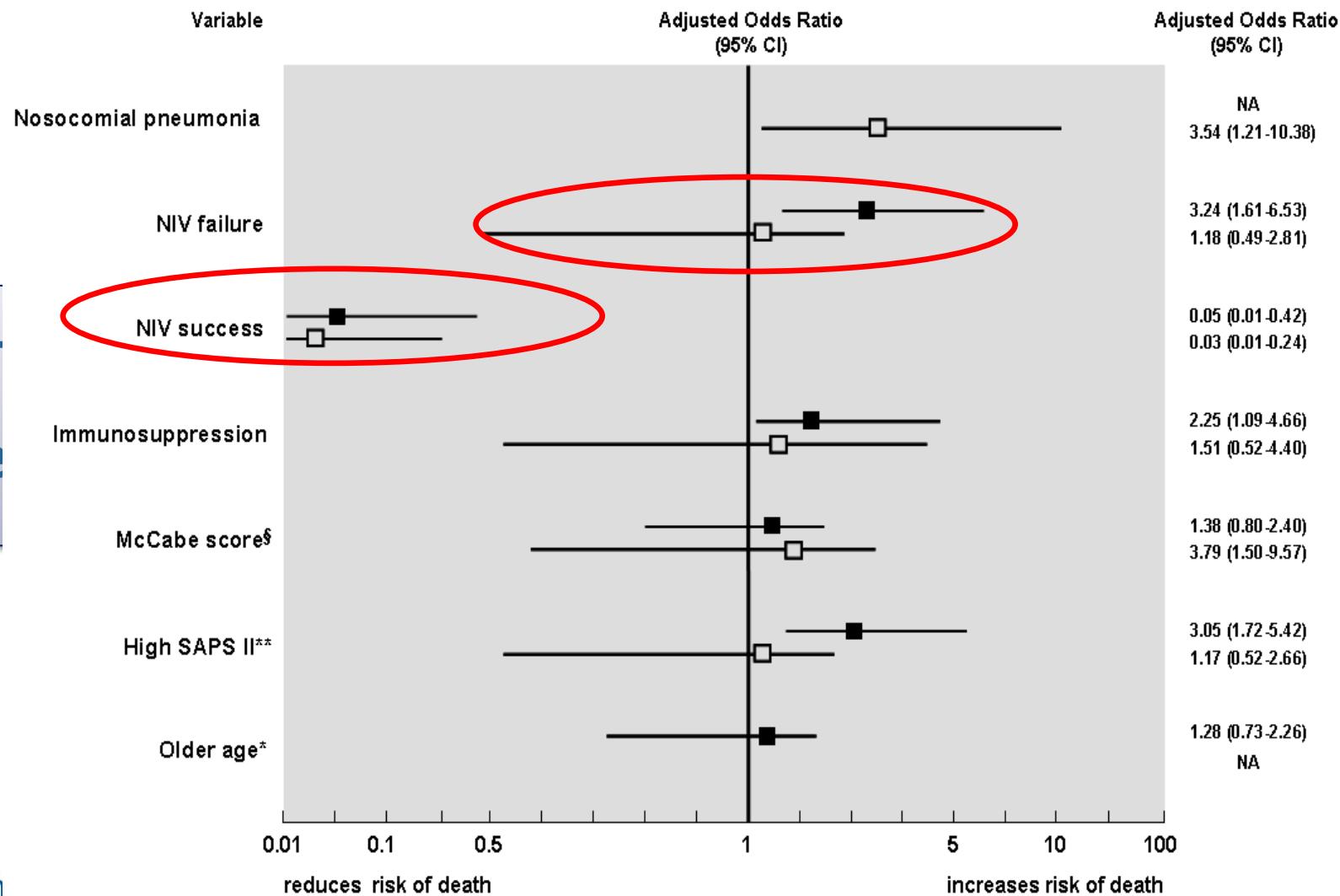


Fig. 2 Forest plot demonstrating the pooled risk ratio for delirium and NPPV failure in acute respiratory failure. Values on the right indicate that delirium has an unfavourable outcome

Alexandre Demoule
Emmanuelle Girou
Jean-Christophe Richard
Solenne Taille
Laurent Brochard

Benefits and risks of success or failure of noninvasive ventilation



M. Antonelli
G. Conti
M. L. Moro
A. Esquinas
G. Gonzalez-Diaz
M. Confalonieri
P. Pelaia
T. Principi
C. Gregoretti
F. Beltrame
M. A. Pennisi
A. Arcangeli
R. Proietti
M. Passariello
G. U. Meduri

Predictors of failure of noninvasive positive pressure ventilation in patients with acute hypoxic respiratory failure: a multi-center study

Table 2 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			
≥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			
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			
≤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

Parametri fisiologici

- SaO₂
- Frequenza respiratoria
- EGA (pH, PaCO₂, PaO₂)
- T CO₂
- End-tidal CO₂

Review

Non-invasive ventilation in acute respiratory failure

Stefano Nava, Nicholas Hill

Panel 4: How to apply NIV during first few hours

- Explain technique to patient (if competent)
- Choose correct interfaces and size
- Set pressures starting from low levels (ie, pressure support about 8 cm H₂O and external PEEP 4–5 cm H₂O)
- Place interface gently over face, holding it in place and start ventilation
- When patient tolerant, tighten straps just enough to avoid major leaks, but not too tight
- Set F₂O₂ on ventilator or add low-flow oxygen into the circuit, aiming for SO₂>90%
- Set alarms—low pressure alarm should be above PEEP level
- Be mindful of and try to optimise patient's comfort
- Reset pressures (pressure support increased to get expired tidal volume 6 mL/kg or higher—raise PEEP external to get oxygen saturation 90% or higher).
- Protect site of skin pressure from the interface (ie, artificial skin, wound-care dressing, or rotating interfaces)
- Consider use of mild sedation if patient is agitated
- Monitor comfort, respiratory rate, oxygen saturation, and dyspnoea every 30 min for 6–12 h then hourly
- Measure arterial blood gases at baseline and within 1 h from start
- Humidification advised for applications longer than 6 hours

NIV=non-invasive ventilation; PEEP=end-expiratory positive pressure; FIO₂=fraction of inspired oxygen; SO₂=oxygen saturation.

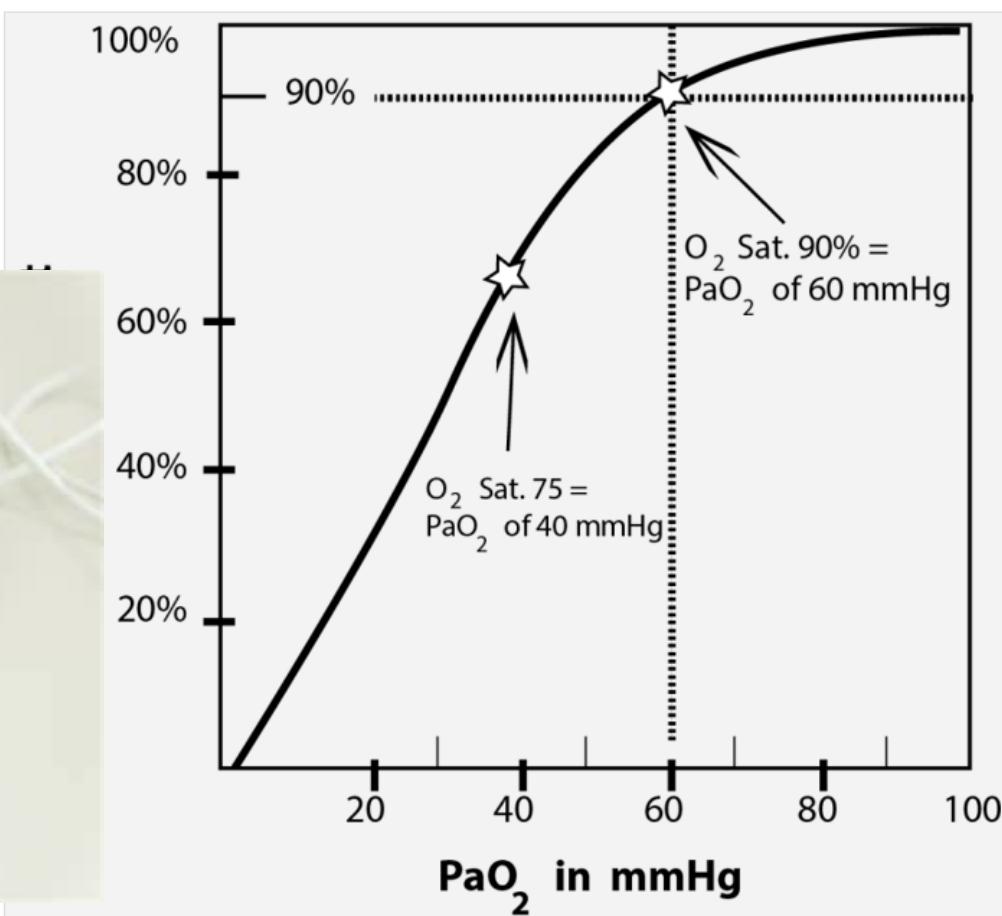
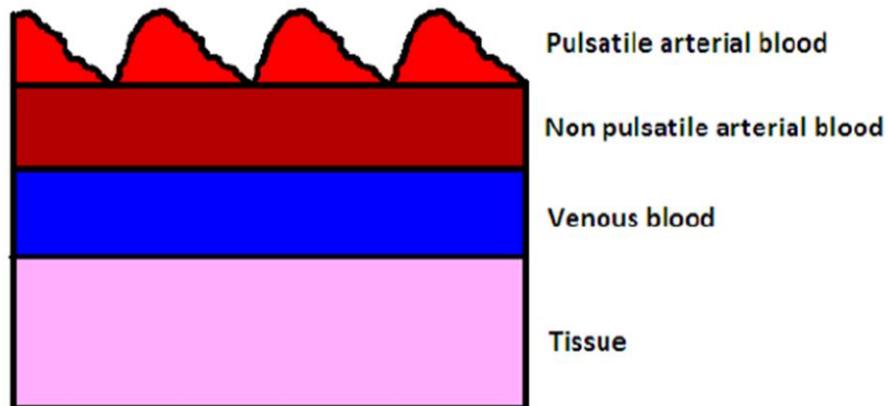
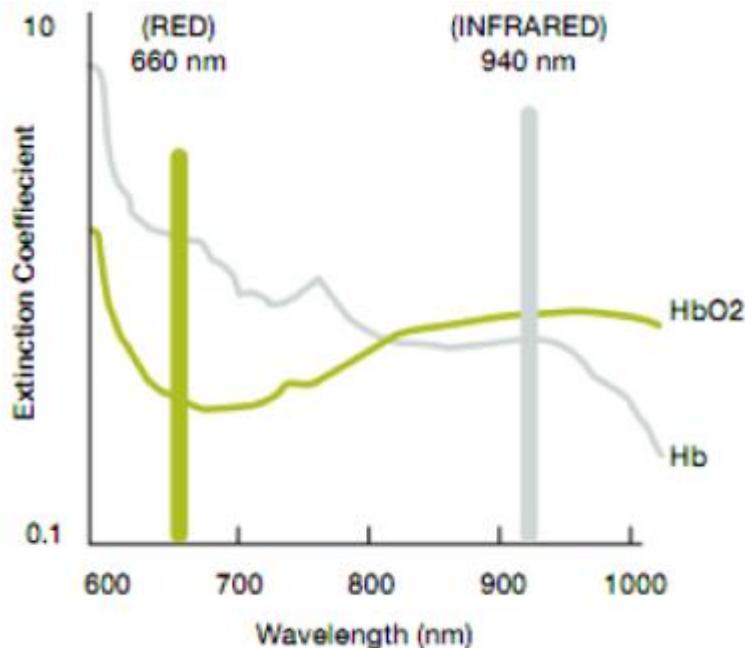
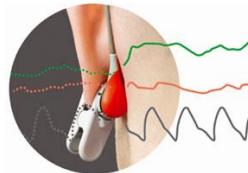


Figure 3. Hemoglobin light absorption graph



Transcutaneous PCO_2 Monitoring During Initiation of Noninvasive Ventilation



Jan H. Storre, MD; Boris Steurer, MD; Hans-Joachim Kabitz, MD;
Michael Dreher, MD; and Wolfram Windisch, MD



Background: To assess the efficacy of transcutaneous PCO_2 (PtCO_2) measurements for monitoring alveolar ventilation in patients requiring noninvasive positive-pressure ventilation (NPPV).

Methods: In a prospective study on method agreement pairs of PaCO_2 and PtCO_2 (SenTec Digital Monitor; SenTec AG; Therwil, Switzerland), measurements were performed every 10 min during the establishment of NPPV over a 4-h period in 10 patients (8 patients with COPD) presenting with acute-on-chronic hypercapnic respiratory failure, thus providing 250 pairs of measurement.

Results: Mean (\pm SD) PaCO_2 decreased from 67.2 ± 11.9 mm Hg (PtCO_2 , 65.5 ± 13.9 mm Hg) to 54.6 ± 8.8 mm Hg (PtCO_2 , 47.8 ± 8.8 mm Hg), and mean pH increased from 7.36 ± 0.03 to 7.44 ± 0.04 . Following PtCO_2 assessment, PtCO_2 in the ensuing 2-min period was the strongest predictor for PaCO_2 compared to PtCO_2 in the ensuing 5-min period and to real-time measurements. PtCO_2 was highly correlated with PaCO_2 ($r = 0.916$; $p < 0.001$), as determined by linear regression analysis. The mean difference between PaCO_2 and PtCO_2 was 4.6 mm Hg, and the limits of agreement (bias ± 1.96 SDs) ranged from -3.9 to 13.2 mm Hg, following the Bland and Altman analysis. Retrospective drift correction produced an even higher correlation ($r = 0.956$; $p < 0.001$) with lower limits of agreement (-1.7 to 7.5 mm Hg).

Conclusions: PtCO_2 measurements provide a sensitive, continuous, and noninvasive method for monitoring alveolar ventilation in pts who are receiving short-term NPPV therapy. The "gold standard" PtCO_2 value cannot be excluded.

Trial registration: www.uniklinik-freiburg.de/zks/live/uklregister/Oeffentlich.html Identifier: UKFO01271. **(CHEST 2007; 132:1810-1816)**

Parametri ventilatori

- Frequenza respiratoria
- VTE, 'VE
- Perdite
- Monitoraggio curve (flusso–tempo, pressione–tempo, capnografia)
- PEEPi
- Interazione paziente ventilatore

P_{AW}



V_T
 RR
 T_i

Carico dei
muscoli
respiratori

- Tachipnea
- discomfort
- ipercapnia

- Atrofia diaframmatica
- Iperinflazione
- Ciclaggio ritardato
- PEEPi
- Sforzi inefficaci
- discomfort

Livello ottimale di
supporto pressorio

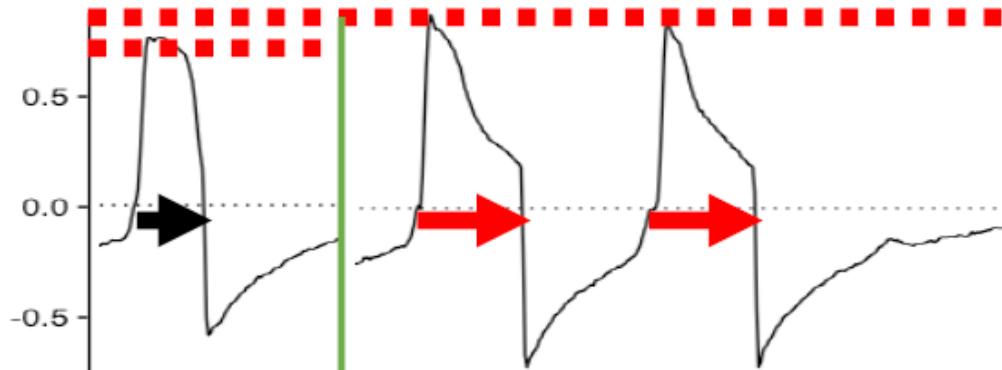


Livello di supporto pressorio

Cicli inspiratori prolungati

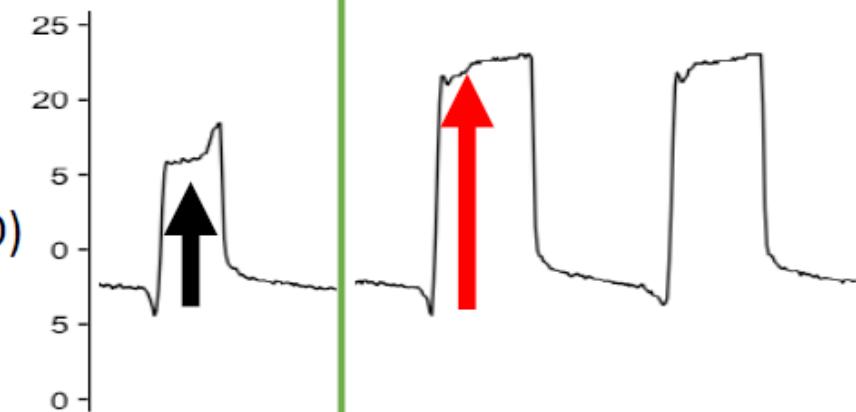
PSV 9

Flow
(L/S)



PSV 15

Paw
(cm H₂O)

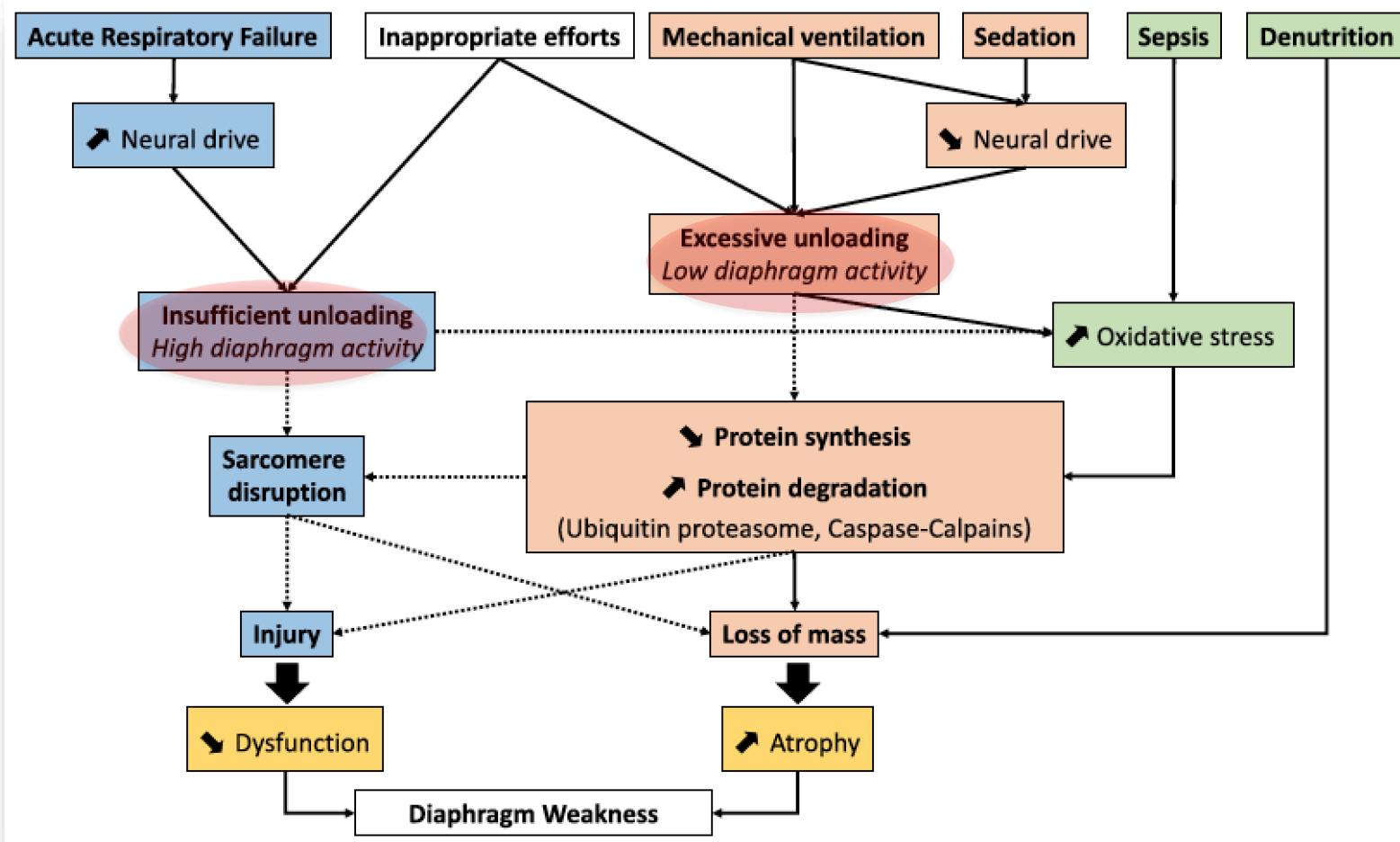


Meccanismi

- Aumento del supporto pressorio
- Picco di flusso elevato
- Tempo di insufflazione prolungato
- Elevato TV
- Tempo neurale ridotto
- Riduzione drive ventilatorio e dello sforzo inspiratorio

Peso
(cm H₂O)





Dres M et al ICM (2017) 43:1441–1452



Danno strutturale del diaframma + atrofia

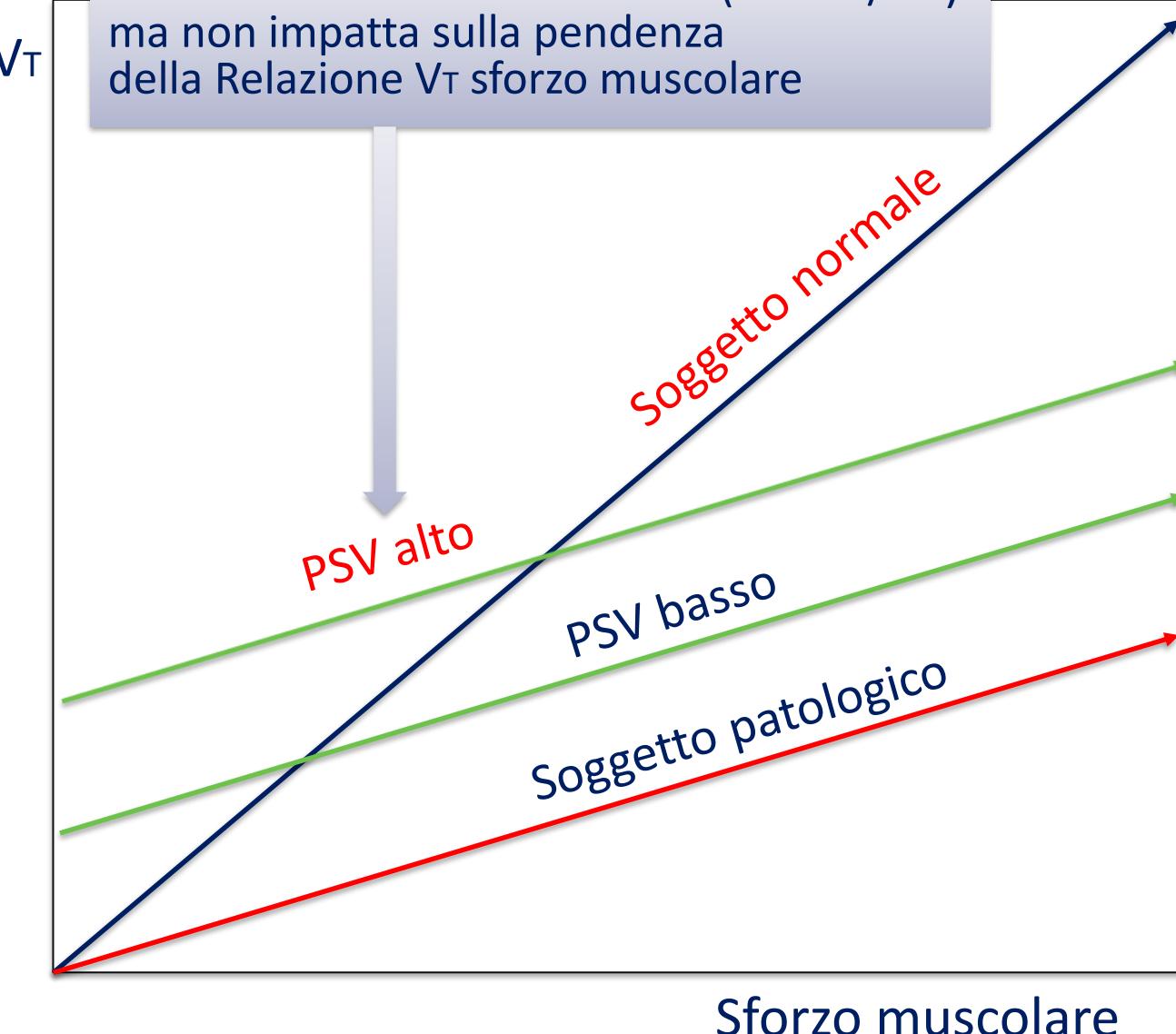
Riduzione della capacità del diaframma di generare forza



Valutazione dello sforzo respiratorio durante ventilazione assistita: cosa monitorare

- ventilazione in volume : curva di pressione
frequenza respiratoria
- ventilazione in pressione : *frequenza respiratoria*
- P0.1

L'aumento del PS aumenta il V_T ($VT = PS/E_{RS}$)
ma non impatta sulla pendenza
della Relazione V_T sforzo muscolare



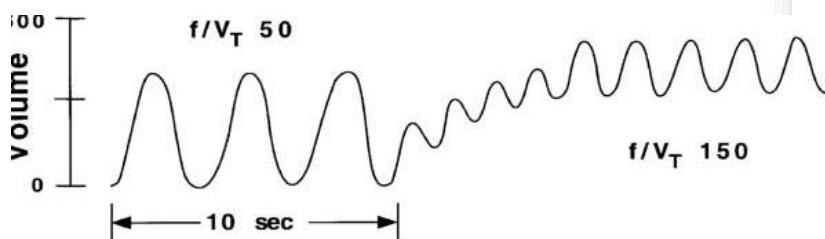
Original Research

The Rapid Shallow Breathing Index as a Predictor of Failure of Noninvasive Ventilation for Patients With Acute Respiratory Failure

Katherine M Berg MD, Gerald R Lang RRT, Justin D Salciccioli, Eske Bak, Michael N Cocchi MD, Shiva Gautam PhD, and Michael W Donnino MD

An aRSBI of > 105 is associated with need for intubation and increased in-hospital mortality.

Whether pts with an elevated aRSBI could also have benefitted from an increase in NIV settings remains unclear



RR 25 breaths/min Vt 250 mL/breath RSBI of
(25 breaths/min)/(0.25 L) = 100 breaths/min/L

Table 2. Multivariate Analysis for aRSBI as a Predictor of Intubation*

	Intubation		
	Odds Ratio	95% CI	P
aRSBI \geq 105	3.70	1.14–11.99	.03
Age	0.96	0.93–0.99	.02
Pneumonia	3.56	1.38–9.18	.009
COPD exacerbation	0.23	0.06–0.88	.03

* Variables were included in multivariate analysis if univariate $P < .10$.
aRSBI = assisted rapid shallow breathing index

Table 3. Multivariate Analysis for aRSBI as a Predictor of In-Hospital Mortality*

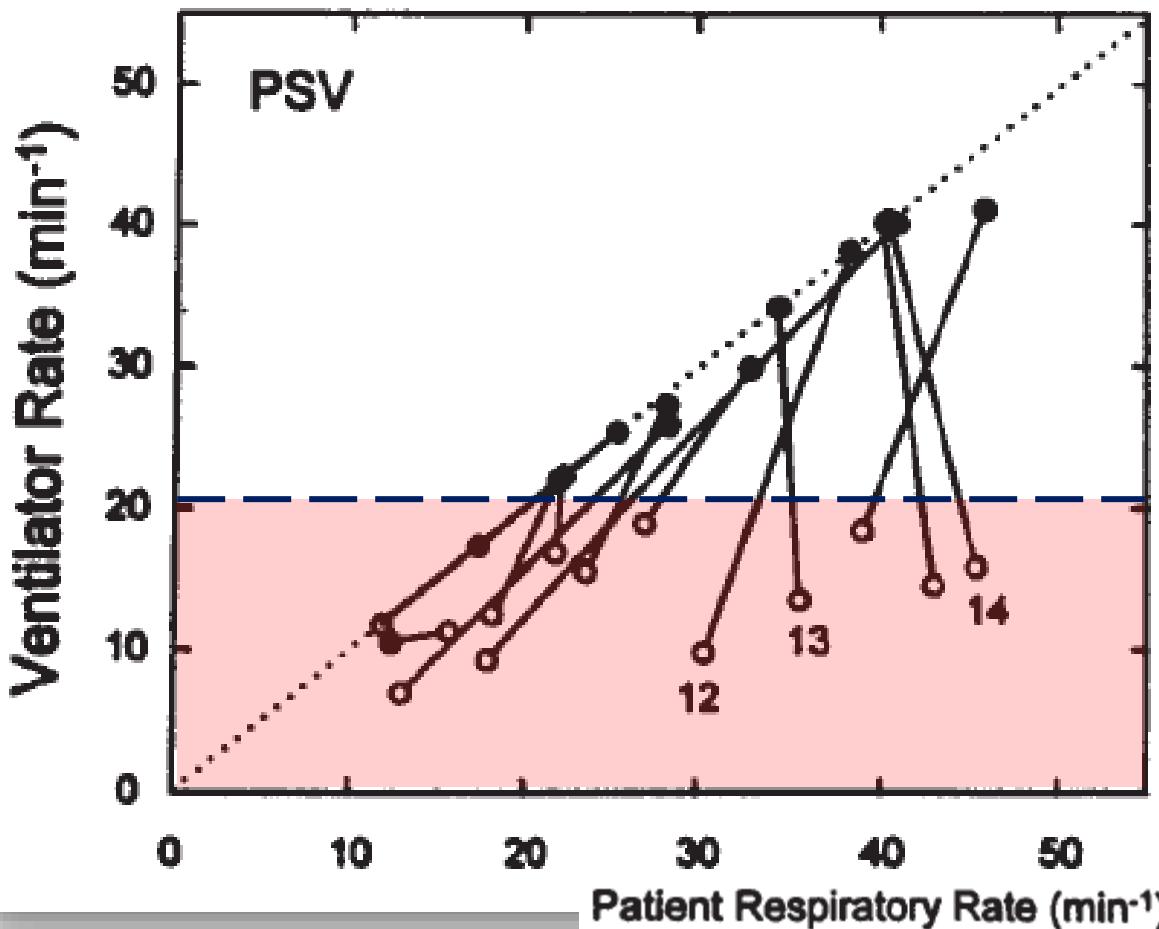
	In-Hospital Mortality		
	Odds Ratio	95% CI	P
aRSBI \geq 105	4.51	1.19–17.11	.03
Age	1.06	1.01–1.11	.03
Sepsis	3.10	0.68–14.13	.14

* Variables were included in multivariate analysis if univariate $P < .10$.
aRSBI = assisted rapid shallow breathing index

Response of Ventilator-dependent Patients to Different Levels of Pressure Support and Proportional Assist

ELENI GIANNOULI, KIM WEBSTER, DAN ROBERTS, and MAGDY YOUNES

Sections of Respiratory and Critical Care Medicine, Department of Medicine, University of Manitoba,
Winnipeg, Manitoba, Canada



At low level of support, the data points (*solid dots*) fall near the line of identity.

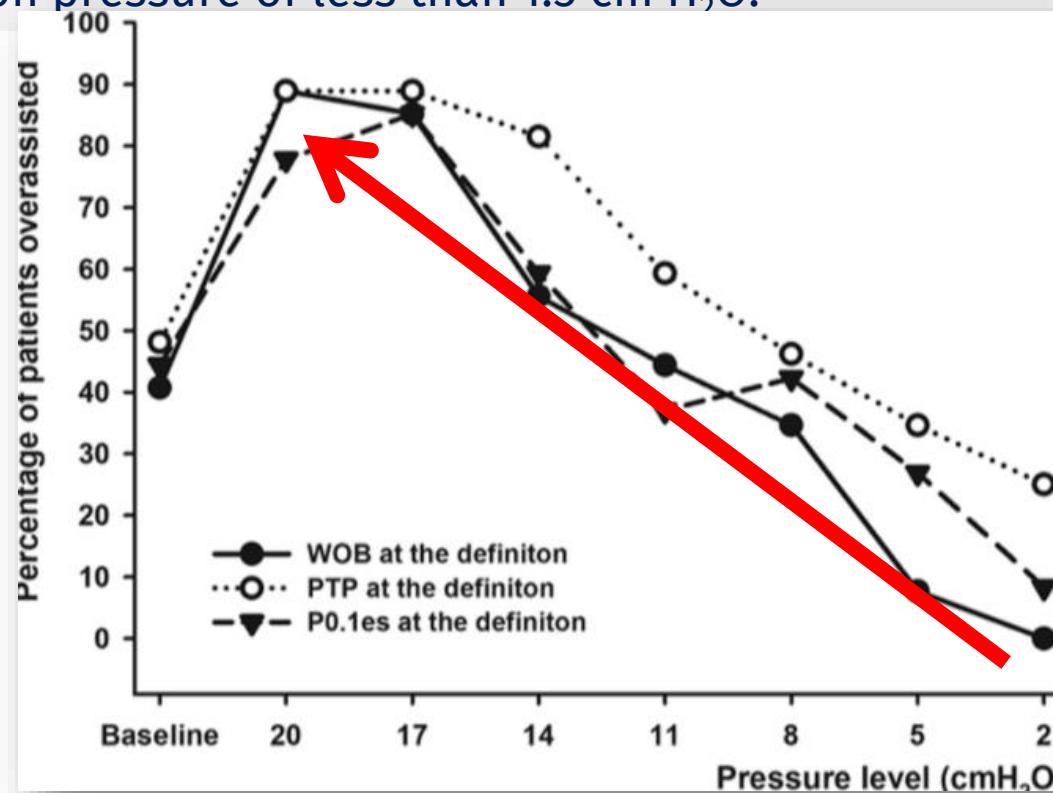
With high-level PSV the data points (*open circles*) deviate from the line of identity in all but two patients.

Accuracy of Invasive and Noninvasive Parameters for Diagnosing Ventilatory Overassistance During Pressure Support Ventilation*

Renata Pletsch-Assuncao, RT, PhD¹; Mayra Caleffi Pereira, RT, MSc¹; Jeferson George Ferreira, RT^{1,2}; Letícia Zumpano Cardenas, RT, PhD^{1,2}; André Luis Pereira de Albuquerque, MD, PhD^{1,3}; Carlos Roberto Ribeiro de Carvalho, MD, PhD¹; Pedro Caruso, MD, PhD^{1,2}

Overassistance was defined as:

- work of breathing less than 0.3 J/L or $\geq 10\%$ of ineffective inspiratory effort.
- inspiratory esophageal pressure-time product of less than 50 cm H₂O s/min
- esophageal occlusion pressure of less than 1.5 cm H₂O.



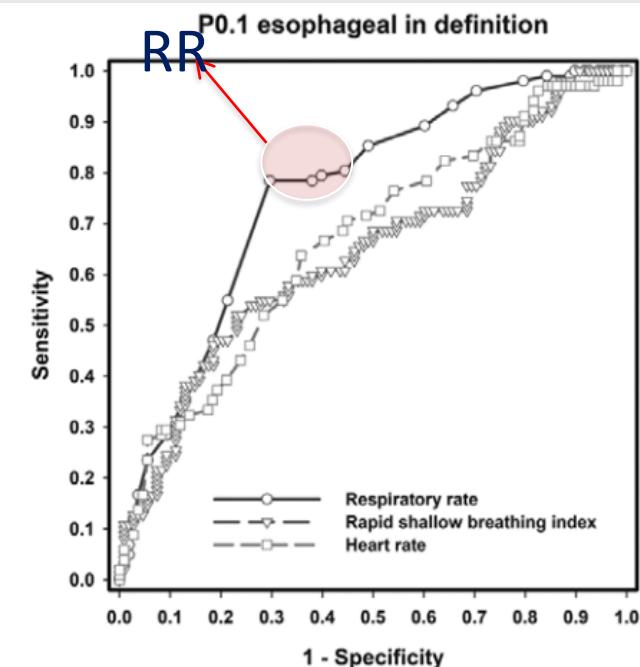
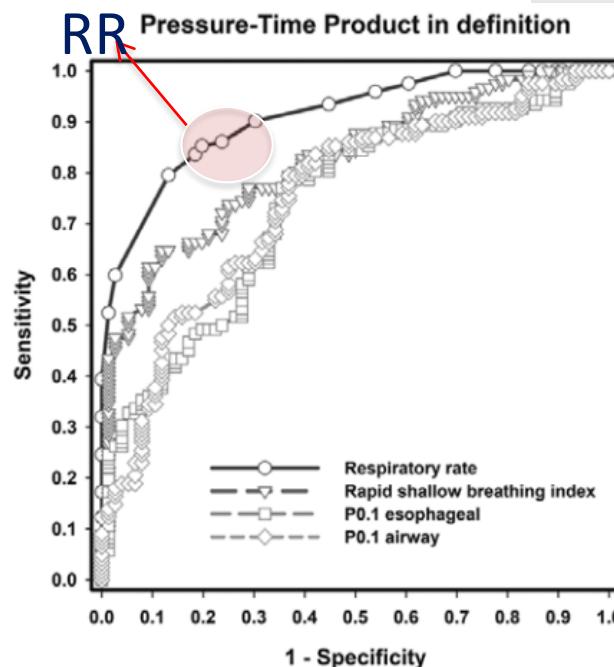
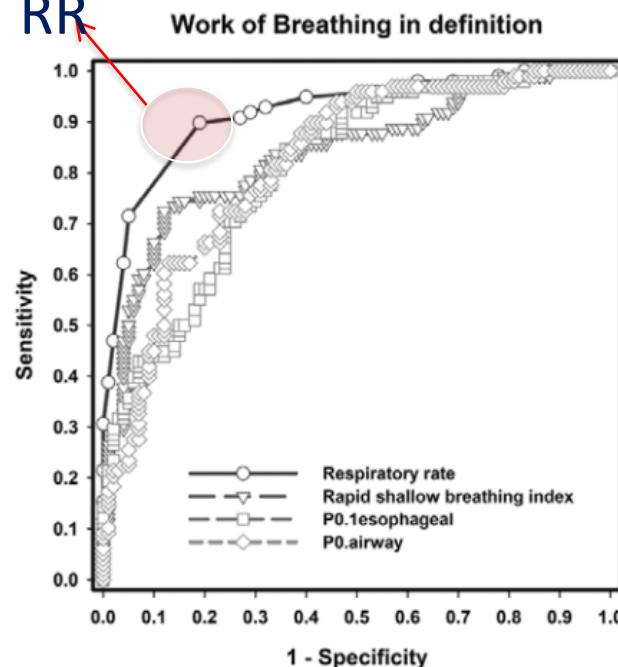
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RR



In all definitions, the RR had the greatest accuracy for diagnosing overassistance

(ROC area = 0.92; 0.91 and 0.76 for work of breathing, pressure-time product and esophageal occlusion pressure in definition, respectively) and always with a cutoff of 17 incursions per minute.

In all definitions, a $\text{RR} \leq 12$ confirmed overassistance (100% specificity), whereas a $\text{RR} \geq 20$ excluded overassistance (100% sensitivity)

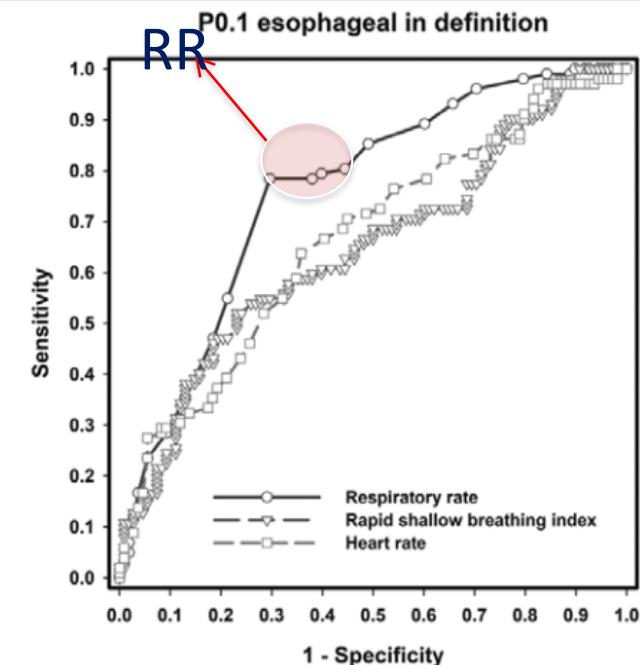
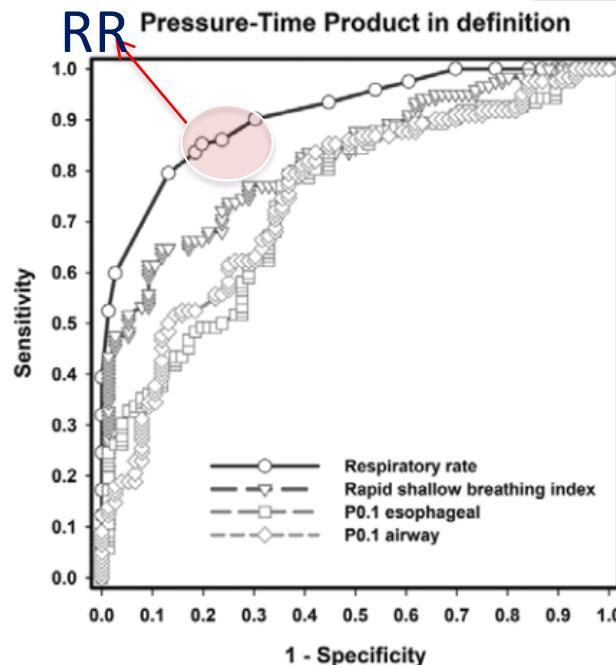
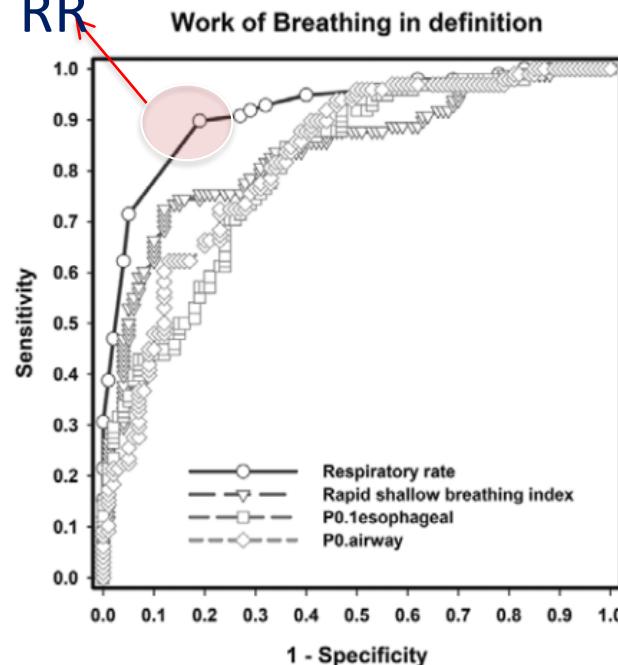
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RR



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In all definitions, a **RR \leq 12 confirmed overassistance (100% specificity)**, whereas a **RR > 20 excluded overassistance (100% sensitivity)**

ORIGINAL

Assessment of heart rate, acidosis, consciousness, oxygenation, and respiratory rate to predict noninvasive ventilation failure in hypoxemic patients



Jun Duan*, Xiaoli Han, Linfu Bai, Lintong Zhou and Shicong Huang

Methods: The test cohort comprised 449 patients with hypoxemia who were receiving NIV. This cohort was used to develop a scale that considers heart rate, acidosis, consciousness, oxygenation, and respiratory rate (referred to as the HACOR scale) to predict NIV failure, defined as need for intubation after NIV intervention. The highest possible score was 25 points. To validate the scale, a separate group of 358 hypoxemic patients were enrolled in the validation cohort.

Demographics	Test cohort		<i>p</i> ^a	Validation cohort		<i>p</i> ^a	<i>p</i> ^b
	NIV failure (N = 215)	NIV success (N = 234)		NIV failure (N = 141)	NIV success (N = 217)		
Age (years)	66 ± 17	65 ± 17	0.51	67 ± 17	65 ± 17	0.38	0.86
Male gender (%)	161 (75%)	153 (65%)	0.03	99 (70%)	156 (72%)	0.81	0.70
Diagnosis							
Pneumonia	104 (48%)	141 (60%)	0.01	74 (53%)	132 (61%)	0.13	0.43
ARDS	61 (28%)	24 (10%)	<0.01	27 (19%)	18 (8%)	<0.01	0.02
Pulmonary cancer	30 (14%)	16 (7%)	0.02	21 (15%)	16 (7%)	0.03	>0.99
Pulmonary embolism	6 (3%)	15 (6%)	0.08	3 (2%)	12 (6%)	0.18	0.86
Heart failure	2 (1%)	13 (6%)	<0.01	4 (3%)	16 (7%)	0.10	0.16
Others	12 (6%)	25 (11%)	0.25	12 (9%)	23 (11%)	0.59	0.46
Data collected at NIV initiation							
APACHE II score	19 ± 6	16 ± 5	<0.01	18 ± 5	14 ± 4	<0.01	<0.01
Systolic blood pressure (mmHg)	131 ± 25	132 ± 25	0.65	133 ± 28	134 ± 30	0.77	0.20
Diastolic blood pressure (mmHg)	79 ± 17	80 ± 15	0.35	79 ± 17	80 ± 17	0.85	0.85
Heart rate (beats/min)	124 ± 24	110 ± 24	←	120 ± 23	113 ± 24	<0.01	0.67
Respiratory rate (breaths/min)	34 ± 8	30 ± 7	←	34 ± 7	31 ± 7	<0.01	0.85
pH	7.40 ± 0.11	7.44 ± 0.08	←	7.42 ± 0.10	7.43 ± 0.08	0.09	0.53
PaCO ₂ (mmHg)	38 ± 17	38 ± 13	0.77	37 ± 13	37 ± 12	0.57	0.19
PaO ₂ /FiO ₂	137 ± 65	179 ± 83	←	146 ± 68	165 ± 63	<0.01	0.79
GCS	14.4 ± 1.7	14.8 ± 0.8	←	14.3 ± 1.6	14.8 ± 0.6	<0.01	0.84

Heart rate, Acidosis (pH), Consciousness (GCS), Oxygenation, and Respiratory rate (HACOR) were independent predictors of NIV failure in the test cohort.

ORIGINAL

Assessment of heart rate, acidosis, consciousness, oxygenation, and respiratory rate to predict noninvasive ventilation failure in hypoxemic patients

Jun Duan¹, Xiaoli Han, Linfu Bai, Lintong Zhou and Shicong Huang



HACOR scale

- Heart rate,
- Acidosis,
- Consciousness,
- Oxygenation
- Respiratory Rate

Hacor >

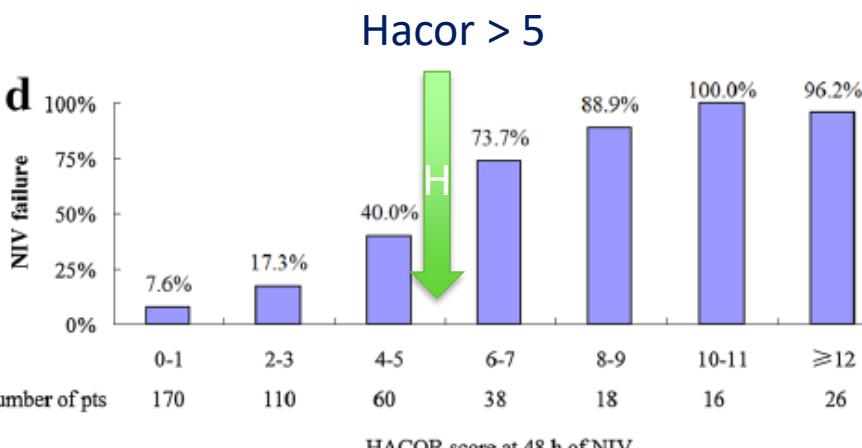
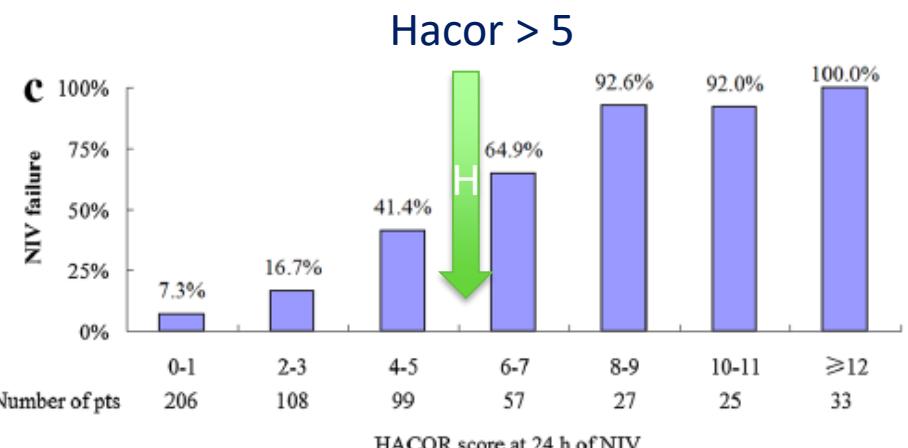
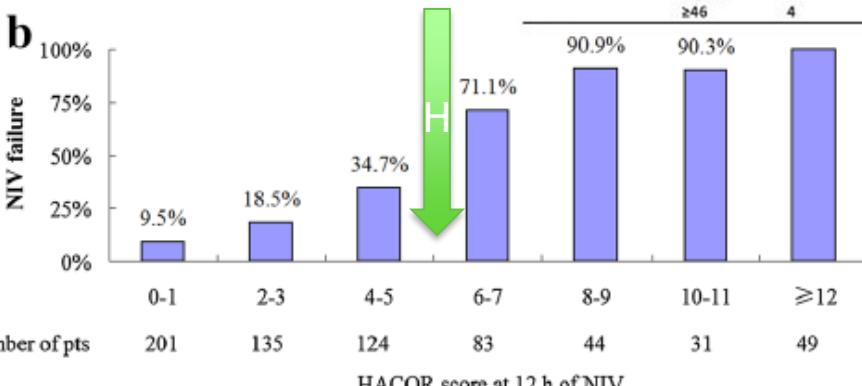
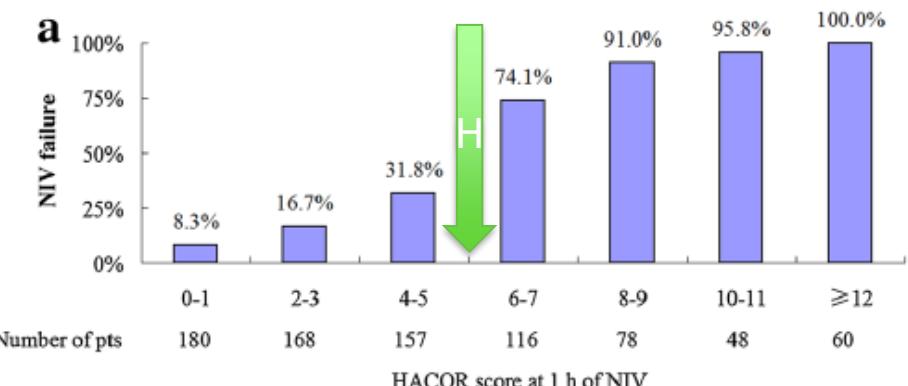


Fig. 1 Noninvasive ventilation (NIV) failure rate in patients with different HACOR (heart rate, acidosis, consciousness, oxygenation, and respiratory rate) scores at 1, 12, 24, and 48 h of NIV

Variables	Category (i)	Assigned points
Heart rate, beats/min	≤120	0
	≥121	1
pH	≥7.35	0
	7.30–7.34	2
	7.25–7.29	3
GCS	<7.25	4
	15	0
	13–14	2
	11–12	5
PaO ₂ /FiO ₂	≤10	10
	≥201	0
	176–200	2
	151–175	3
	126–150	4
	101–125	5
Respiratory rate, breaths/min	≤100	6
	≤30	0
	31–35	1
	36–40	2
	41–45	3
	≥46	4

ORIGINAL

Assessment of heart rate, acidosis, consciousness, oxygenation, and respiratory rate to predict noninvasive ventilation failure in hypoxicemic patients

Jun Duan*, Xiaoli Han, Linfu Bai, Lintong Zhou and Shicong Huang

Variables	Category (j)	Assigned points
Heart rate, beats/min	≤ 120	0
	≥ 121	1
pH	≥ 7.35	0
	$7.30\text{--}7.34$	2
	$7.25\text{--}7.29$	3
	<7.25	4
GCS	15	0
	13–14	2
	11–12	5
	≤ 10	10
PaO ₂ /FiO ₂	≥ 201	0
	176–200	2
	151–175	3
	126–150	4
	101–125	5
	≤ 100	6
Respiratory rate, breaths/min	≤ 30	0
	31–35	1
	36–40	2
	41–45	3
	≥ 46	4

HACOR score improves in pts with NIV success and remains unaltered in patients with NIV failure.

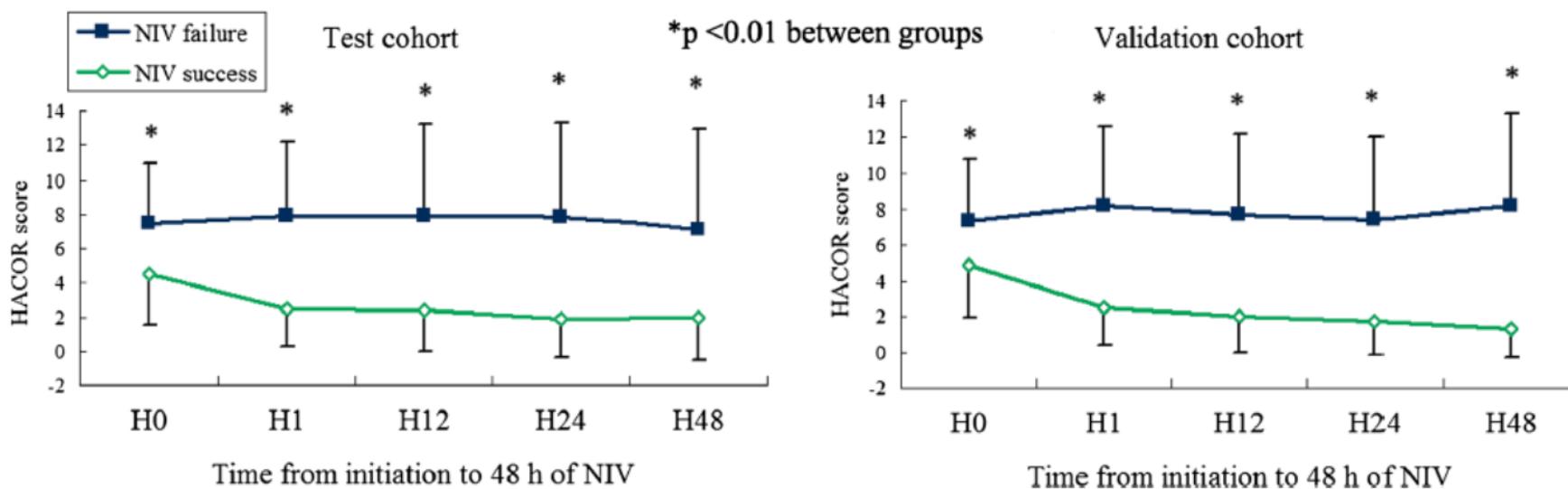


Fig. 2 The HACOR score in patients with NIV failure and success from initiation to 48 h of NIV

ORIGINAL

Assessment of heart rate, acidosis, consciousness, oxygenation, and respiratory rate to predict noninvasive ventilation failure in hypoxicemic patients

Jun Duan*, Xiaoli Han, Linfu Bai, Lintong Zhou and Shicong Huang



The diagnostic accuracy for NIV failure of a HACOR score > 5 at 1 hour of NIV was 81.8% (test cohort) and 86% (validation cohort).

Table 4 Predictive power of noninvasive ventilation failure diagnosed by the HACOR score assessed at 1 h, 12 h, 24 h and 48 h of NIV

NIV time points	AUC (95% CI)	Cutoff point	SE (%)	SP (%)	PPV (%)	NPV (%)	Diagnostic accuracy (%)	LR+	LR-
1 h of NIV ($N = 807$)	0.89 (0.87–0.91)	>5	73.9	91.4	87.1	81.6	83.7	8.54	0.29
12 h of NIV ($N = 667$)	0.87 (0.85–0.90)	>5	66.9	92.3	85.0	81.0	82.3	8.72	0.36
24 h of NIV ($N = 555$)	0.88 (0.85–0.90)	>5	61.5	93.4	83.1	82.1	82.3	9.30	0.41
48 h of NIV ($N = 438$)	0.87 (0.83–0.90)	>5	60.3	95.6	86.7	83.5	84.2	13.8	0.42

HACOR heart rate, acidosis, consciousness, oxygenation, and respiratory rate, AUC area under the curve of receiver operating characteristics, CI confidence interval, SE sensitivity, SP specificity, PPV positive predictive value, NPV negative predictive value, LR+ positive likelihood ratio, LR– negative likelihood ratio, NIV noninvasive ventilation

The diagnostic accuracy for NIV failure remained above 80% regardless of NIV duration, diagnosis, age, or disease severity (APACHE 2 score).

ORIGINAL



Assessment of heart rate, acidosis, consciousness, oxygenation, and respiratory rate to predict noninvasive ventilation failure in hypoxemic patients

Jun Duan*, Xiaoli Han, Linfu Bai, Lintong Zhou and Shicong Huang

HACOR scale

- Heart rate,
- Acidosis,
- Consciousness,
- Oxygenation
- Respiratory Rate

Table 5 Early versus late intubation in patients with a HACOR score of >5 at 1 h of noninvasive ventilation

NIV time points and hospital mortality	Intubation at ≤ 12 h ($N = 88$)	Intubation at >12 h ($N = 175$)	p
HACOR score at NIV initiation	8.9 ± 3.9	7.7 ± 3.0	<0.01
HACOR score at 1 h of NIV	11.4 ± 4.0	8.8 ± 3.1	<0.01
HACOR score before intubation	12.2 ± 4.7	11.4 ± 5.0	0.22
Time from NIV initiation to intubation (h)	5 (2–18)	53 (22–132)	<0.01
Hospital mortality	58 (66%)	138 (79%)	0.03

Values in table are presented as the mean \pm SD, the median \pm IQR, or the number of patients with the IQR in parenthesis, or as a number with the percentage in parenthesis, as appropriate

HACOR heart rate, acidosis, consciousness, oxygenation, and respiratory rate, NIV noninvasive ventilation

Among pts with a HACOR score >5, early ETI improved hospital mortality

Parametri ventilatori

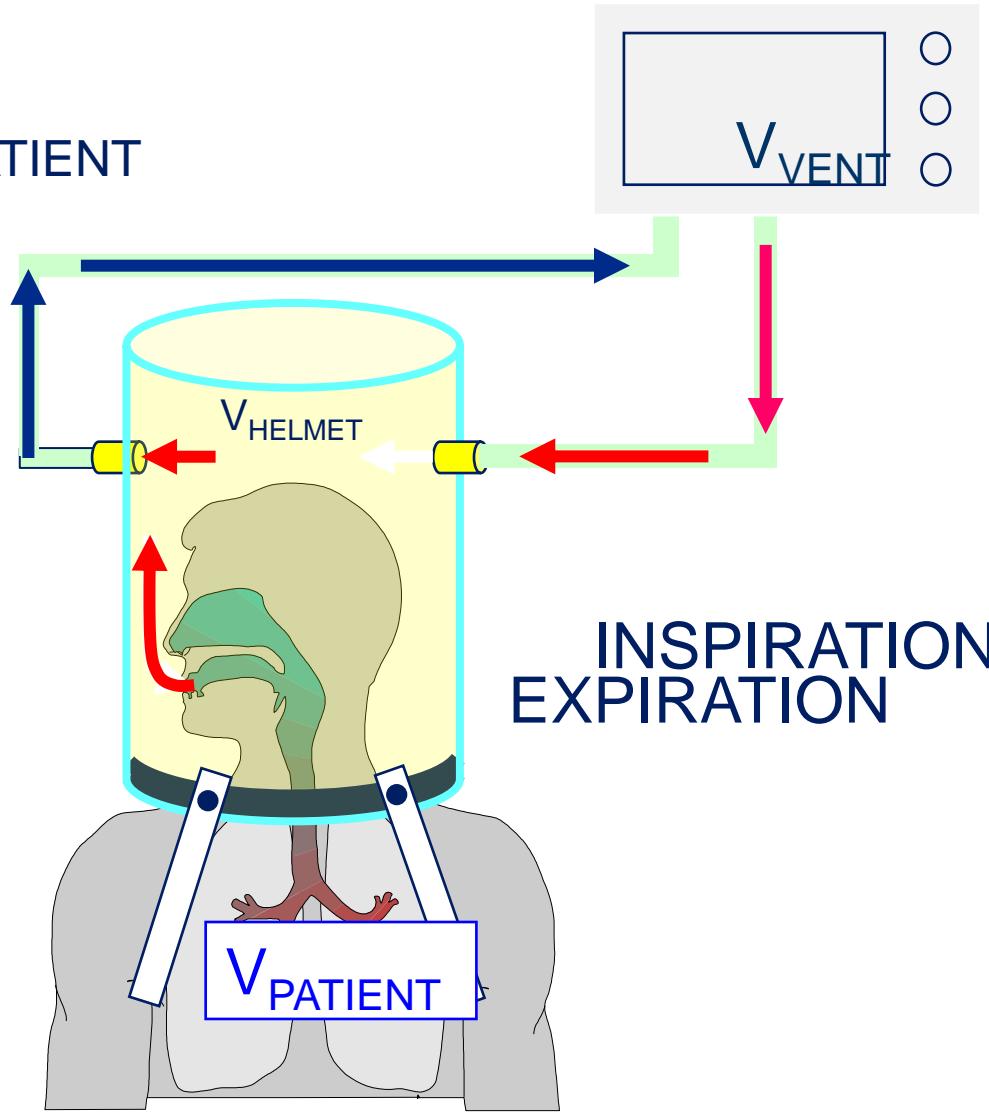
- Frequenza respiratoria
- VTE, 'VE
- Perdite
- Monitoraggio curve (flusso–tempo, pressione–tempo, capnografia)
- PEEPi
- Interazione paziente ventilatore

Monitoring of patient's volume and flow is impossible

$$V_{VENT} = V_{HELMET} + V_{PATIENT}$$

V_{HELMET} depends on:

- PS level
- PEEP
- Helmet volume
- Helmet rigidity
- Helmet fixation
- Leaks
- Resp. muscles activity
- Resp. Mechanics (R, C)



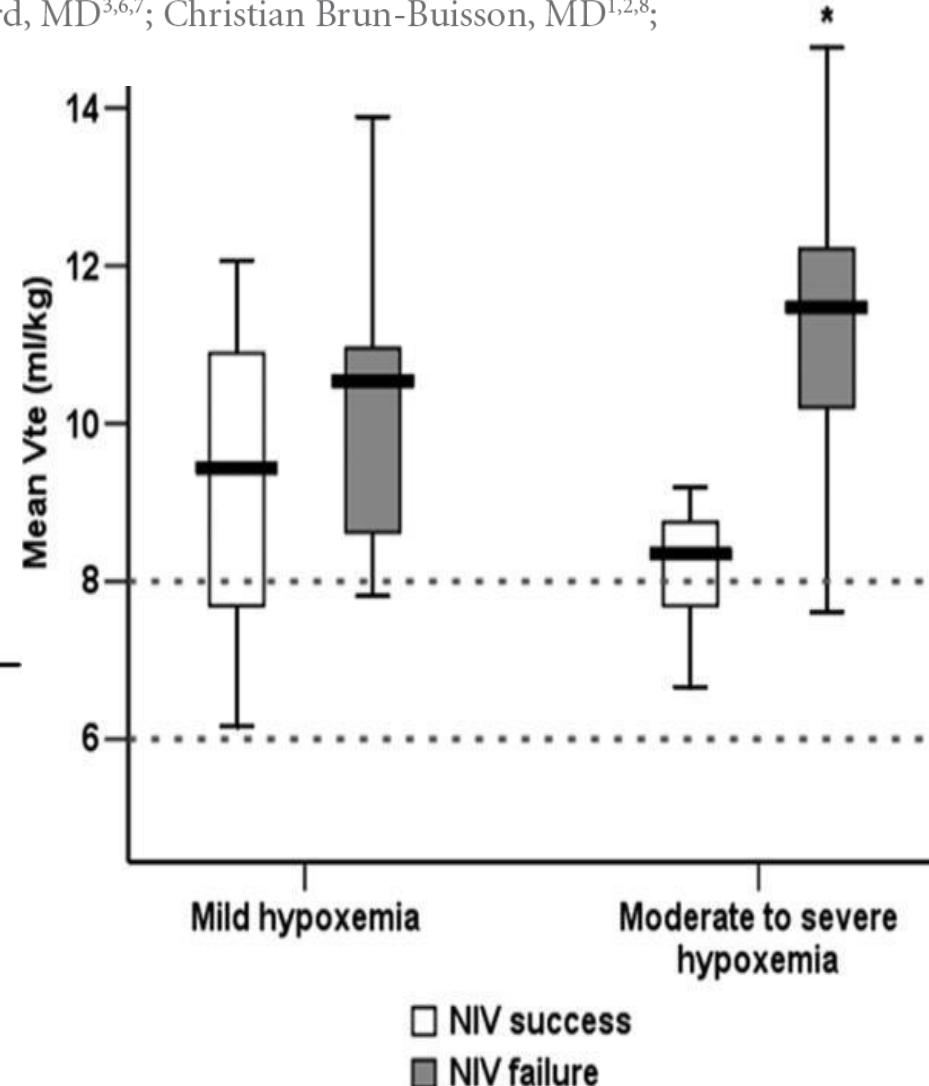
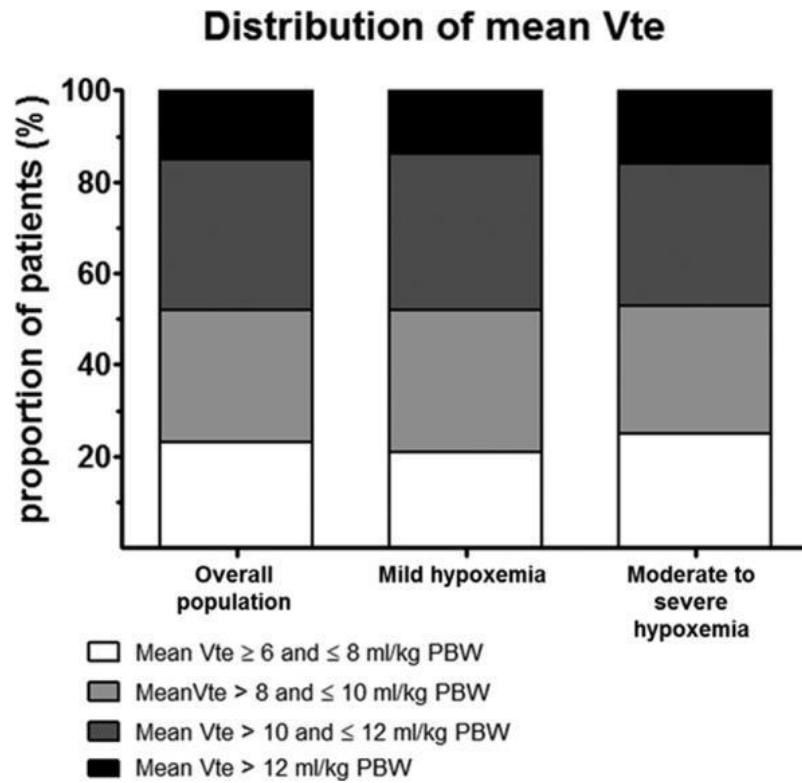
Failure of Noninvasive Ventilation for De Novo Acute Hypoxemic Respiratory Failure: Role of Tidal Volume*

Guillaume Carteaux, MD^{1,2,3}; Teresa Millán-Guilarte, MD⁴; Nicolas De Prost, MD, PhD^{1,2,3};

Keyvan Razazi, MD^{1,2,3}; Shariq Abid, MD, PhD³; Arnaud W. Thille, MD, PhD⁵;

Frédérique Schortgen, MD, PhD^{1,3}; Laurent Brochard, MD^{3,6,7}; Christian Brun-Buisson, MD^{1,2,8};

Armand Mekontso Dessap, MD, PhD^{1,2,3}



Failure of Noninvasive Ventilation for De Novo Acute Hypoxemic Respiratory Failure: Role of Tidal Volume*

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Armand Mekontso Dessap, MD, PhD^{1,2,3}

Risk Factors	Unadjusted Hazard Ratio (95% CI)	p	Adjusted Hazard Ratio (95% CI) ^a	p
Simplified Acute Physiology Score II (30)	1.026 (1.008–1.043)	0.011	1.024 (1.007–1.041)	0.013
Immunosuppression	2.207 (1.054–4.622)	0.045	1.351 (0.598–3.056)	0.476
Pao ₂ /Fio ₂ before NIV	0.995 (0.990–1.001)	0.114	0.995 (0.989–1.001)	0.109
Mean expired tidal volume during NIV, per mL/kg predicted body weight	1.318 (1.109–1.567)	0.002	1.286 (1.069–1.547)	0.008

NIV = noninvasive ventilation.

^aAdjusted hazard ratio obtained by Cox regression.

Failure of Noninvasive Ventilation for De Novo Acute Hypoxemic Respiratory Failure: Role of Tidal Volume*

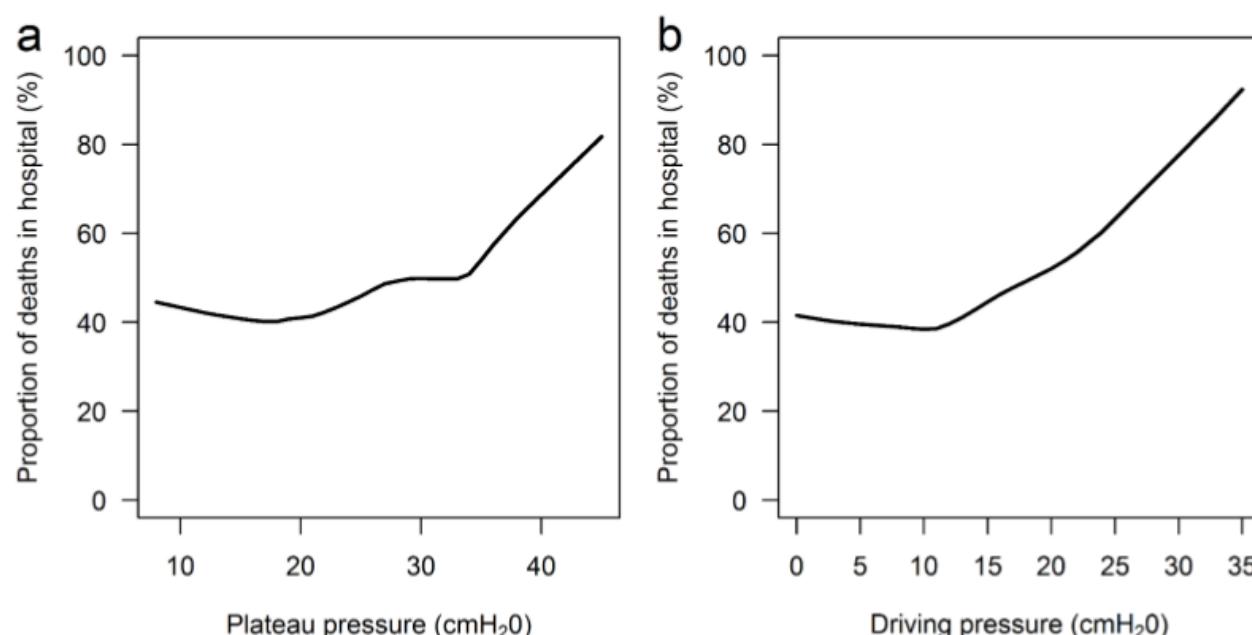
Guillaume Carteaux, MD^{1,2,3}; Teresa Millán-Guilarte, MD⁴; Nicolas De Prost, MD, PhD^{1,2,3}; Keyvan Razazi, MD^{1,2,3}; Sharif Abid, MD, PhD⁵; Arnaud W. Thille, MD, PhD⁶; Frédérique Schortgen, MD, PhD^{1,2,3}; Laurent Brochard, MD^{3,6,7}; Christian Brun-Buisson, MD^{1,2,8}; Armand Mekontso Dessap, MD, PhD^{1,2,3}

Ventilatory and Hemodynamic Data	NIV Success (n = 30)	NIV Failure (n = 32)	p
Positive end-expiratory pressure, cm H ₂ O			
During NIV	5 (5–5)	5 (5–5)	0.50
After intubation	NA	10 (5–14) ^b	
Pressure support level, cm H ₂ O			
During NIV	7.8 (7.2–9.3)	7.5 (7.0–8.3)	0.28
NIV H1	8.0 (7.0–10.5)	8.0 (7.0–10.0)	0.07
Before intubation	NA	8.0 (7.0–8.0)	
V _{te} , mL/kg PBW			
During NIV	8.5 (7.6–10.2)	10.6 (9.6–12.0)	0.001
Respiratory mechanics after intubation			
Static compliance of the respiratory system, mL/cm H ₂ O	NA	27 (18–36)	

PIP 13 cmH₂O

$$10.6 \times 70 = 742 \text{ ml}$$

$$742/27 + \text{PEEP} = 32 \text{ cmH}_2\text{O}$$



Intensive Care Med (2016) 42:1865–1876
DOI 10.1007/s00134-016-4571-5

SEVEN-DAY PROFILE PUBLICATION



Potentially modifiable factors contributing to outcome from acute respiratory distress syndrome: the LUNG SAFE study

John G. Laffey^{1,2,3} Giacomo Bellani^{3,4} Tai Pham^{5,6,7} Eddy Fan^{8,9} Fabiana Madotto¹⁰ Ednan K. Bajwa¹¹ Laurent Brochard^{12,13} Kevin Clarkson¹⁴ Andres Esteban¹⁵ Luciano Gattinoni¹⁶ Frank van Haren¹⁷ Leo M. Heunks¹⁸ Kiyoyasu Kurashiki¹⁹ Jon Henrik Laake²⁰ Anders Larsson²¹ Daniel F. McAuley²² Lia McNamee²² Nicolas Nini¹⁵ Halbo Olz²³ Marco Ranieri²⁴ Gordon D. Rubenfeld²⁵ B. Taylor Thompson¹¹ Hermann Wrigge²⁶ Arthur S. Slutsky^{12,13,27} Antonio Pesenti^{28,29} and The LUNG SAFE Investigators and the ESICM Trials Group

Parametri ventilatori

- VTE, 'VE
- Frequenza respiratoria
- Perdite
- Monitoraggio curve (flusso–tempo, pressione–tempo, capnografia)
- PEEPi
- Interazione paziente ventilatore

Laurence Vignaux
Frédéric Vargas
Jean Roeseler
Didier Tassaux
Arnaud W. Thille
Michel P. Kossowsky
Laurent Brochard
Philippe Jollivet

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

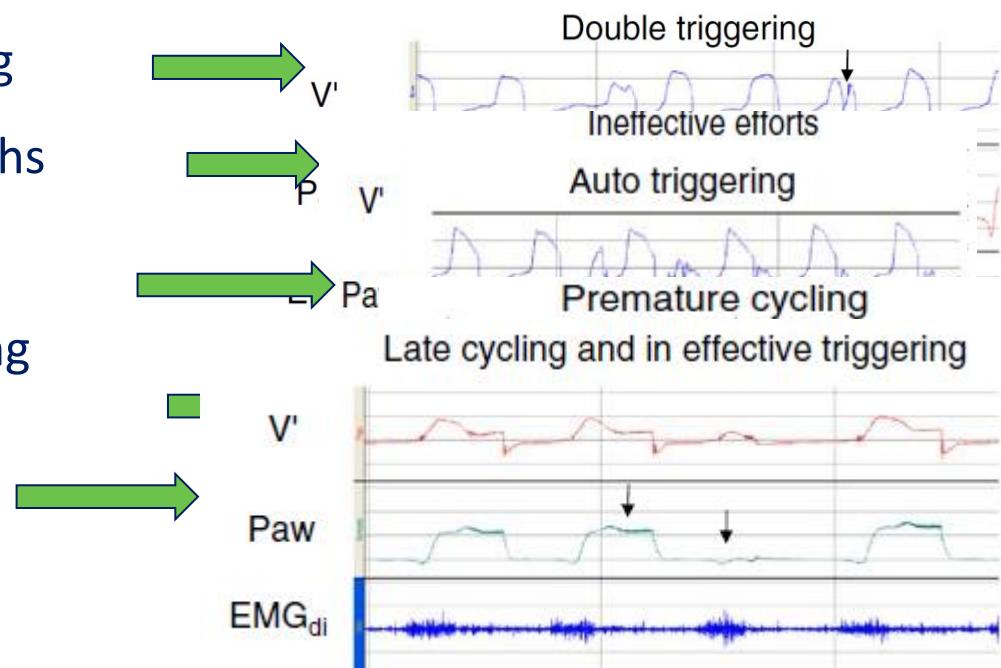
Vignaux et al., Intensive Care Med 2009; 35: 840-846

60 NIV pts (55%Hypercapnic)

Asynchrony prevalence in NIV:

AI > 10% in 26 pts (43%) median AI 26 (15-54%)

- 9 pts (15%) double triggering
- 8 pts (13%),ineffective breaths
- 8 pts (13%) auto-triggering
- 7 pts (12%) premature cycling
- 14 pts (23%) late cycling



Asynchrony Index (AI)

% of breaths that are asynchronous

Number of Asynchrony Events $\times 100$

Total Respiratory Rate

IEE + IEI + DT + AT $\times 100$

Ventilator breaths +Ineffective efforts

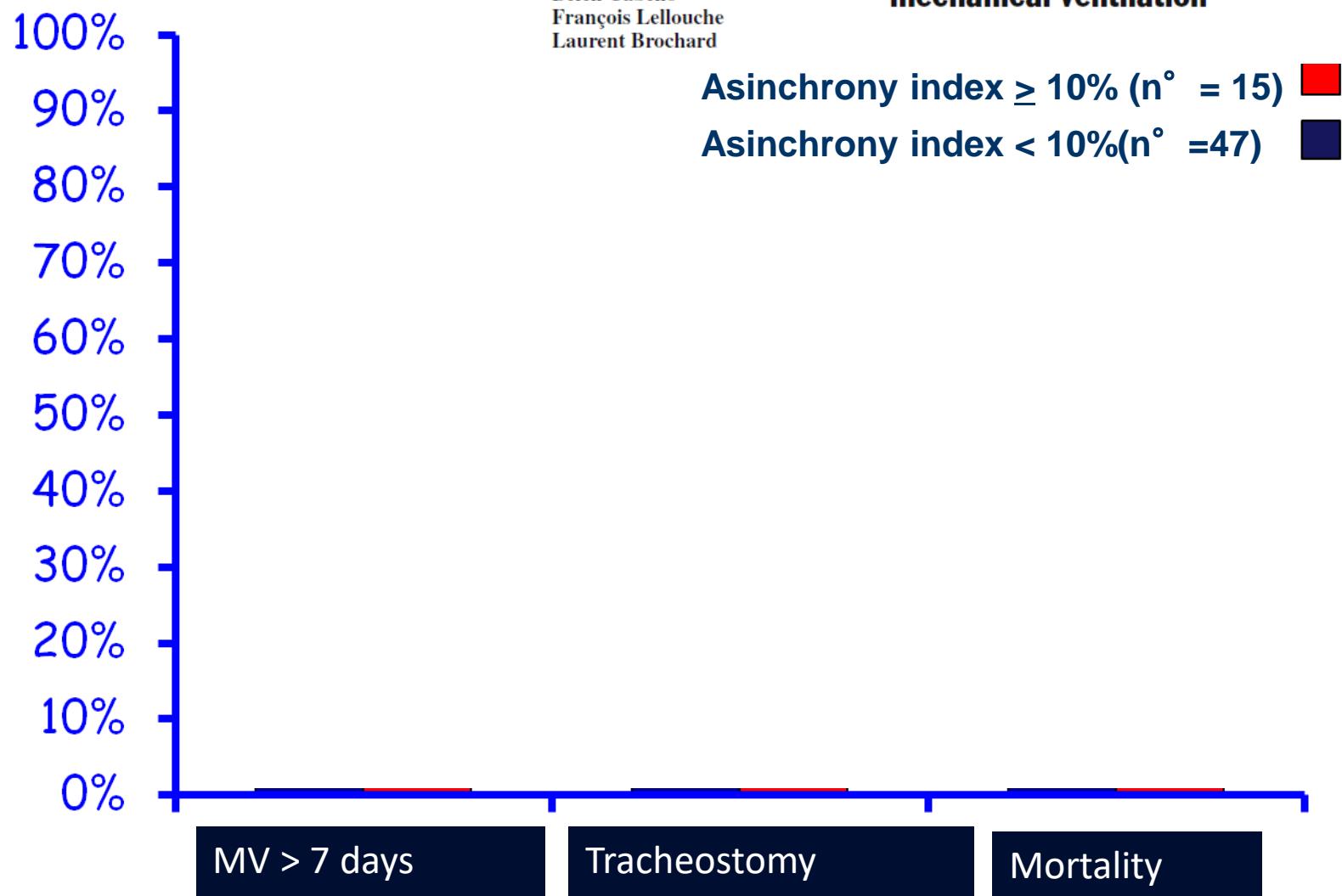
Asynchrony Index $\geq 10\%$ \rightarrow High

Vitacca, M. Chest (2004) 126: 851-859

Thille, A. Intensive Care Med (2006) 32:1515-1522

Arnaud W. Thille
Pablo Rodriguez
Belen Cabello
François Lellouche
Laurent Brochard

Patient-ventilator asynchrony during assisted mechanical ventilation



Lluís Blanch
Ana Villagra
Bernat Sales
Jaume Montanya
Umberto Lucangelo
Manel Luján
Oscar García-Esquirol
Encarna Chacón
Anna Estruga
Joan C. Oliva
Alberto Hernández-Abadia
Guillermo M. Albaiceta
Enrique Fernández-Mondejar
Rafael Fernández
Josefina Lopez-Aguilar
Jesús Villar
Gastón Murias
Robert M. Kacmarek

Asynchronies during mechanical ventilation are associated with mortality

Table 2 Relationship between AI and duration of MV, reintubation, tracheostomy, and ICU and hospital mortality by comparing patients AI ≤ 10 % vs AI > 10 %

	AI ≤ 10 % (n = 44)	AI > 10 % (n = 6)	p value
Length of MV (days)	6 [5.0; 15.0]	16 [9.7; 20.0]	0.061
Reintubation	9 (20 %)	0 (0 %)	0.57
Tracheostomy	14 (32 %)	2 (33 %)	0.999
ICU mortality	6 (14 %)	4 (67 %)	0.011*
Hospital mortality	10 (23 %)	4 (67 %)	0.044*

Data are expressed as numbers and percentages or as medians and interquartile ranges

MV mechanical ventilation, ICU intensive care unit, AI asynchrony index

* Significant at $p < 0.05$



Efficacy of ventilator waveform observation for detection of patient–ventilator asynchrony during NIV: a multicentre study

Federico Longhini ^①, Davide Colombo², Lara Pisani³, Francesco Idone⁴, Pan Chun⁵, Jonne Doorduin⁶, Liu Ling⁵, Moreno Alemani⁷, Andrea Bruni⁸, Jin Zhaochen⁹, Yu Tao¹⁰, Weihua Lu¹⁰, Eugenio Garofalo⁸, Luca Carenzo², Salvatore Maurizio Maggiore¹¹, Haibo Qiu⁵, Leo Heunks¹², Massimo Antonelli⁴, Stefano Nava³ and Paolo Navalesi⁸

ABSTRACT The objective of this study was to assess ability to identify asynchronies during noninvasive ventilation (NIV) through ventilator waveforms according to experience and interface, and to ascertain the influence of breathing pattern and respiratory drive on sensitivity and prevalence of asynchronies.

35 expert and 35 nonexpert physicians evaluated 40 5-min NIV reports displaying flow–time and airway pressure–time tracings; identified asynchronies were compared with those ascertained by three examiners who evaluated the same reports displaying, additionally, tracings of diaphragm electrical activity. We determined: 1) sensitivity, specificity, and positive and negative predictive values; 2) the correlation between the double true index (DTI) of each report (*i.e.*, the ratio between the sum of true positives and true negatives, and the overall breath count) and the corresponding asynchrony index (AI); and 3) the influence of breathing pattern and respiratory drive on both AI and sensitivity.

Sensitivities to detect asynchronies were low either according to experience (0.20 (95% CI 0.14–0.29) for expert *versus* 0.21 (95% CI 0.12–0.30) for nonexpert, $p=0.837$) or interface (0.28 (95% CI 0.17–0.37) for mask *versus* 0.10 (95% CI 0.05–0.16) for helmet, $p<0.0001$). DTI inversely correlated with the AI ($r^2=0.67$, $p<0.0001$). Breathing pattern and respiratory drive did not affect prevalence of asynchronies and sensitivity.

Patient–ventilator asynchrony during NIV is difficult to recognise solely by visual inspection of

In conclusion, recognising patient–ventilator asynchrony during NIV by visual inspection of the ventilator-displayed waveforms is difficult.

Because the use of invasive means to precisely detecting patient's own respiratory activity is unreasonable for most patients undergoing NIV, the future development of dedicated tools for these purposes is advisable

Monitoraggio della pressione esofagea

CONCISE CLINICAL REVIEW



The Application of Esophageal Pressure Measurement in Patients with Respiratory Failure

Evangelia Akoumianaki¹, Salvatore M. Maggiore², Franco Valenza³, Giacomo Bellani⁴, Amal Jubran⁵, Stephen H. Loring⁶, Paolo Pelosi⁷, Daniel Talmor⁶, Salvatore Grasso⁸, Davide Chiumello⁹, Claude Guérin¹⁰, Nicolo Patroniti⁴, V. Marco Ranieri¹¹, Luciano Gattinoni¹², Stefano Nava¹³, Pietro-Paolo Terragni¹¹, Antonio Pesenti⁴, Martin Tobin⁵, Jordi Mancebo¹⁴, and Laurent Brochard¹⁵

Intensive Care Med (2016) 42:1360–1373
DOI 10.1007/s00134-016-4400-x

REVIEW



Esophageal and transpulmonary pressure in the clinical setting: meaning, usefulness and perspectives

Tommaso Mauri¹, Takeshi Yoshida^{2,3,4}, Giacomo Bellani⁵, Ewan C. Goligher^{6,7,12}, Guillaume Carteaux^{8,9}, Nuttapol Rittayamai^{10,11,12}, Francesco Mojoli¹³, Davide Chiumello^{1,14}, Lise Piquilloud^{15,16}, Salvatore Grasso¹⁷, Amal Jubran¹⁸, Franco Laghi¹⁸, Sheldon Magder¹⁹, Antonio Pesenti^{1,14}, Stephen Loring²⁰, Luciano Gattinoni^{1,14}, Daniel Talmor²⁰, Lluís Blanch²¹, Marcelo Amato²², Lu Chen^{11,12}, Laurent Brochard^{11,12*}, Jordi Mancebo²³ and the PLeUral pressure working Group (PLUG—Acute Respiratory Failure section of the European Society of Intensive Care Medicine)

Classificazione generale

1. Asincronie di trigger inspiratorio

- a. Trigger delay → Sforzi inefficaci
- b. Autotrigger

2. Asincronie di ciclaggio espiratorio

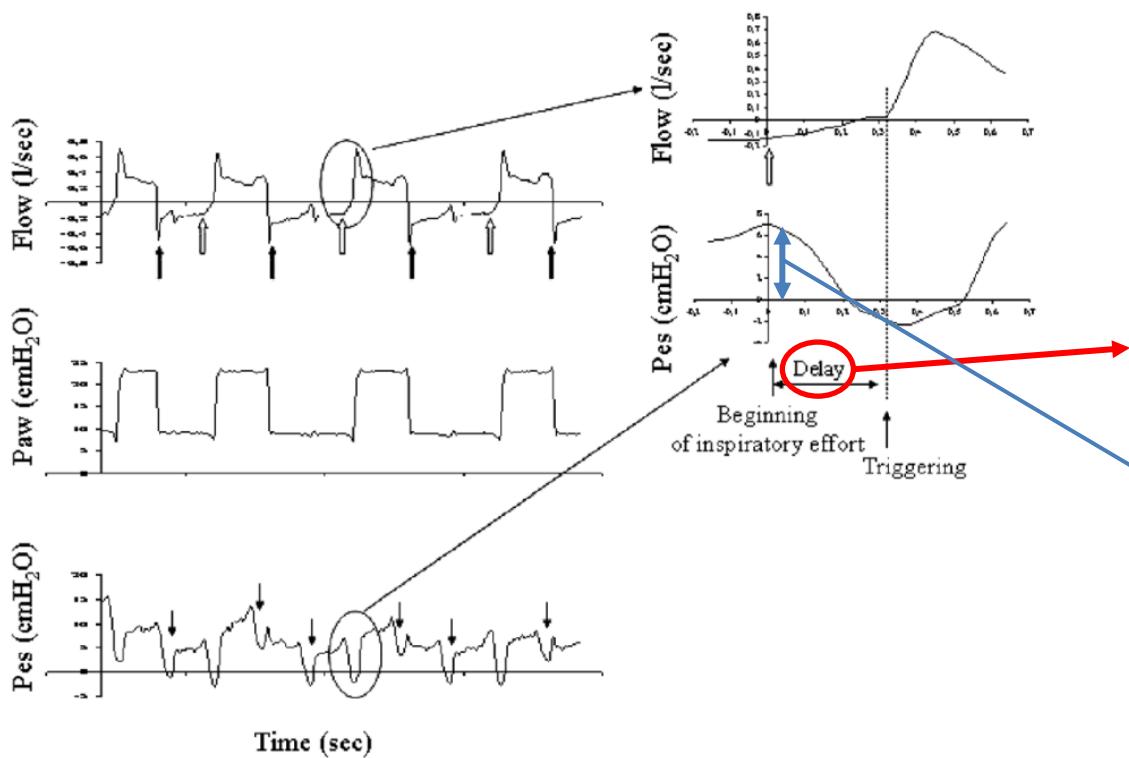
- a. Ciclaggio espiratorio precoce (Short cycle) → Doppio trigger
- b. Ciclaggio espiratorio tardivo → Ciclaggio a tempo (*hang-up*)

3. Altre asincronie

- a. Flow starvation
- b. Reverse triggering non armonico

Asincronie di trigger inspiratorio

Trigger delay



$$P_{mus} (+ P_{aw}) = P_{res} + P_{el} + \textcolor{red}{P_{PEEPi}}$$

Il flusso inspiratorio (pos.) inizia solo quando il carico dato dalla PEEPi viene controbilanciato dallo sforzo inspiratorio (deflessione sul tracciato Paw)

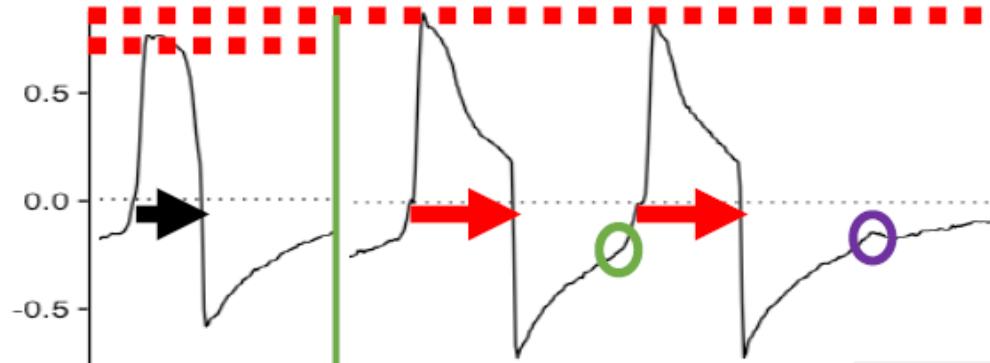
= PEEPi !

Sforzi inefficaci

PSV 9

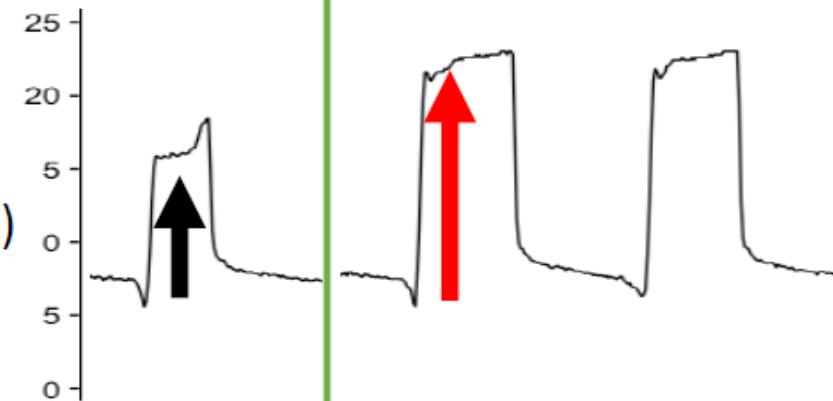
PSV 15

Flow
(L/S)



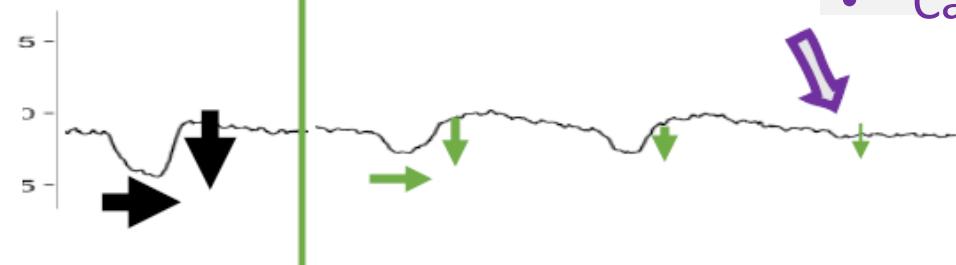
Meccanismi

Paw
(cm H₂O)

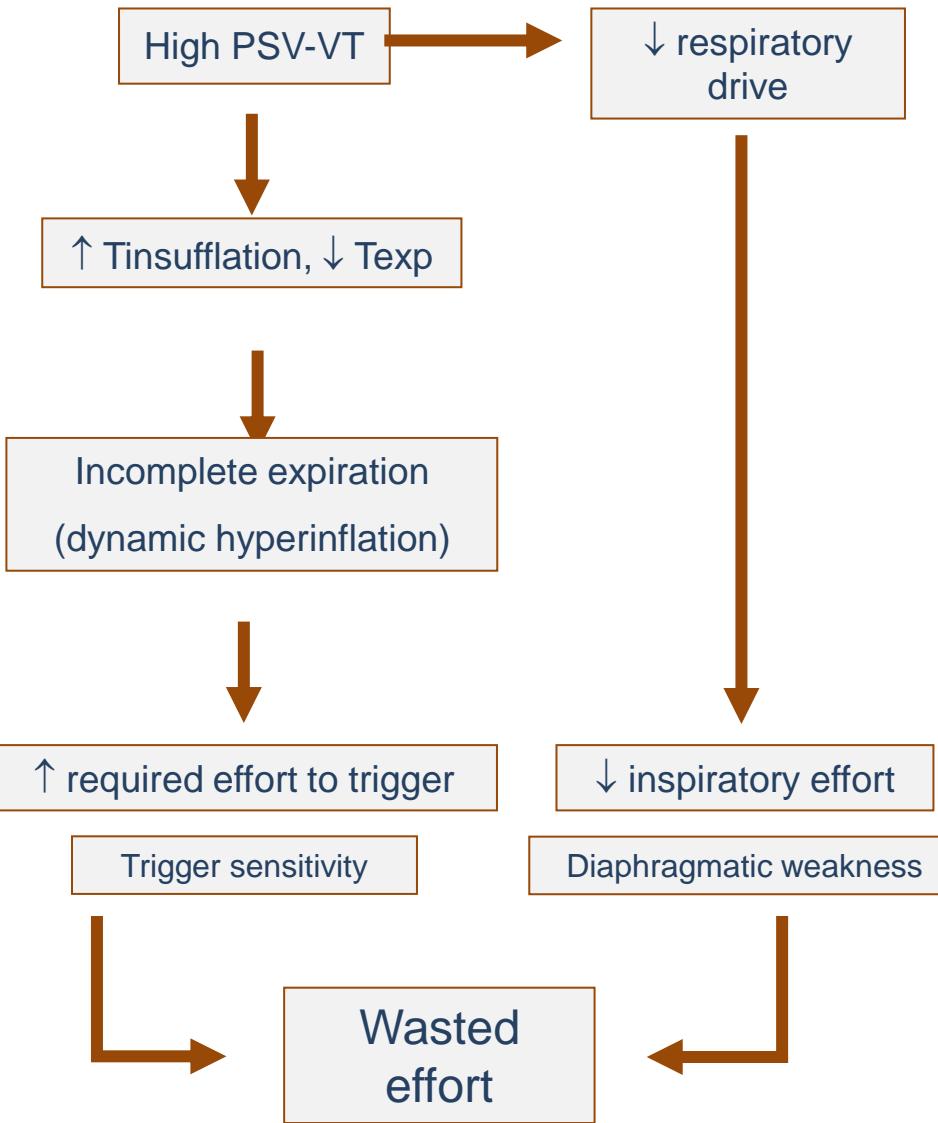


- Supporto pressorio eccessivo
- Riduzione drive ventilatorio e sforzi inspiratori
- ↑compliance ↑ Costante di tempo
- ↑Resistenze
- iperinflazione
- Sforzi insp. prima della completa esp.
- Carico extra dovuto auto PEEP

Peso
(cm H₂O)



8 PEEP 14 PSV



8 PEEP 8 PSV



Conseguenze cliniche del ciclaggio prolungato e degli sforzi inefficaci in corso di PSV

Supporto eccessivo

Riduzione del drive ventilatorio e dello sforzo inspiratorio

Atrofia diaframmatica



Sforzi inefficaci
Basso RR sullo schermo del ventilatore

Raddoppio RR
e riduzione Vt

Aumento supporto

Passaggio in Ventilazione controllata

Ritardata estubazione

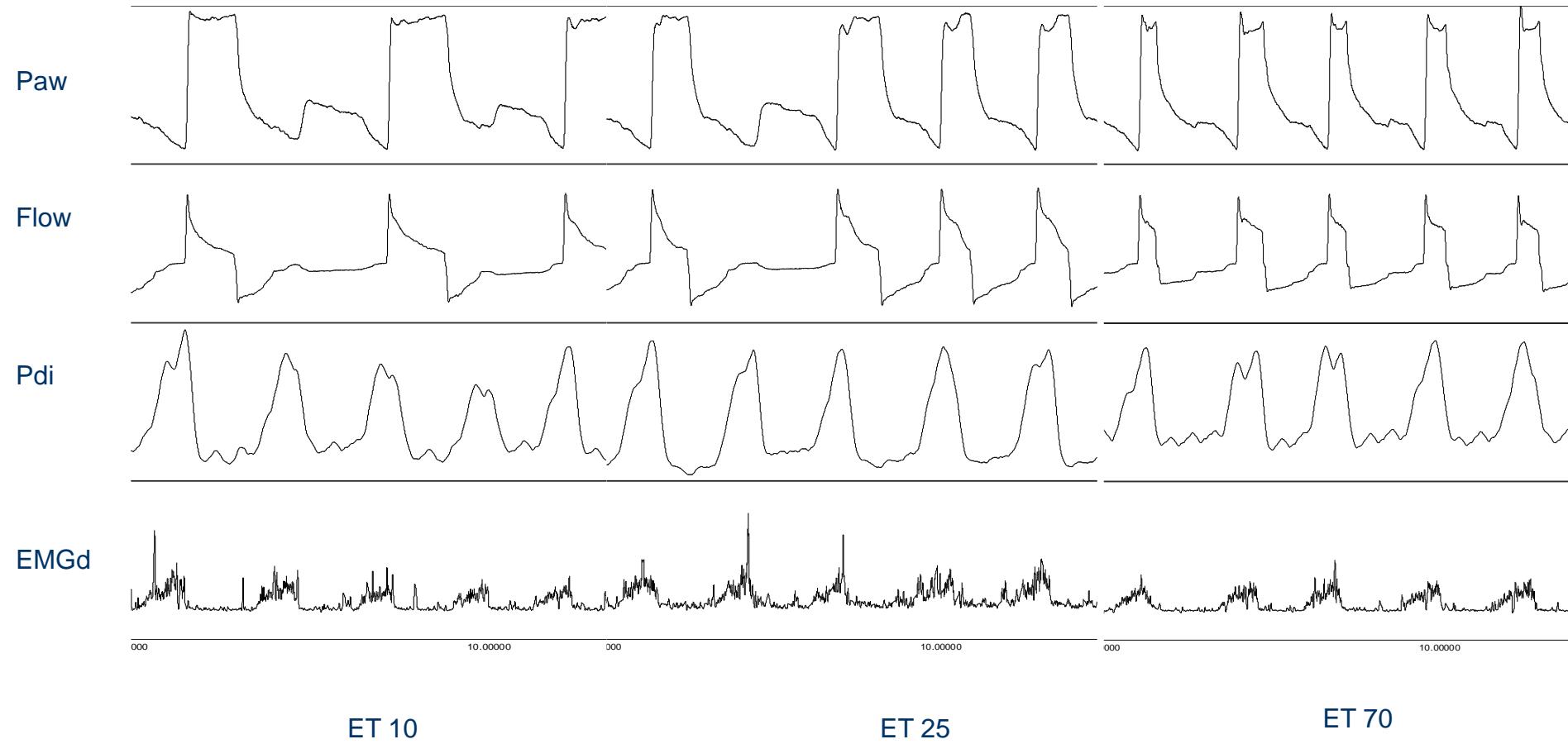
Se il clinico riduce il supporto

Reazione del clinico

Impact of Expiratory Trigger Setting on Delayed Cycling and Inspiratory Muscle Workload

Didier Tassaux, Marc Gaignier, Anne Battisti, and Philippe Jolliet

Medical Intensive Care, University Hospital, Geneva, Switzerland; and Medical Intensive Care, Ste. Marguerite University Hospital, Marseille, France

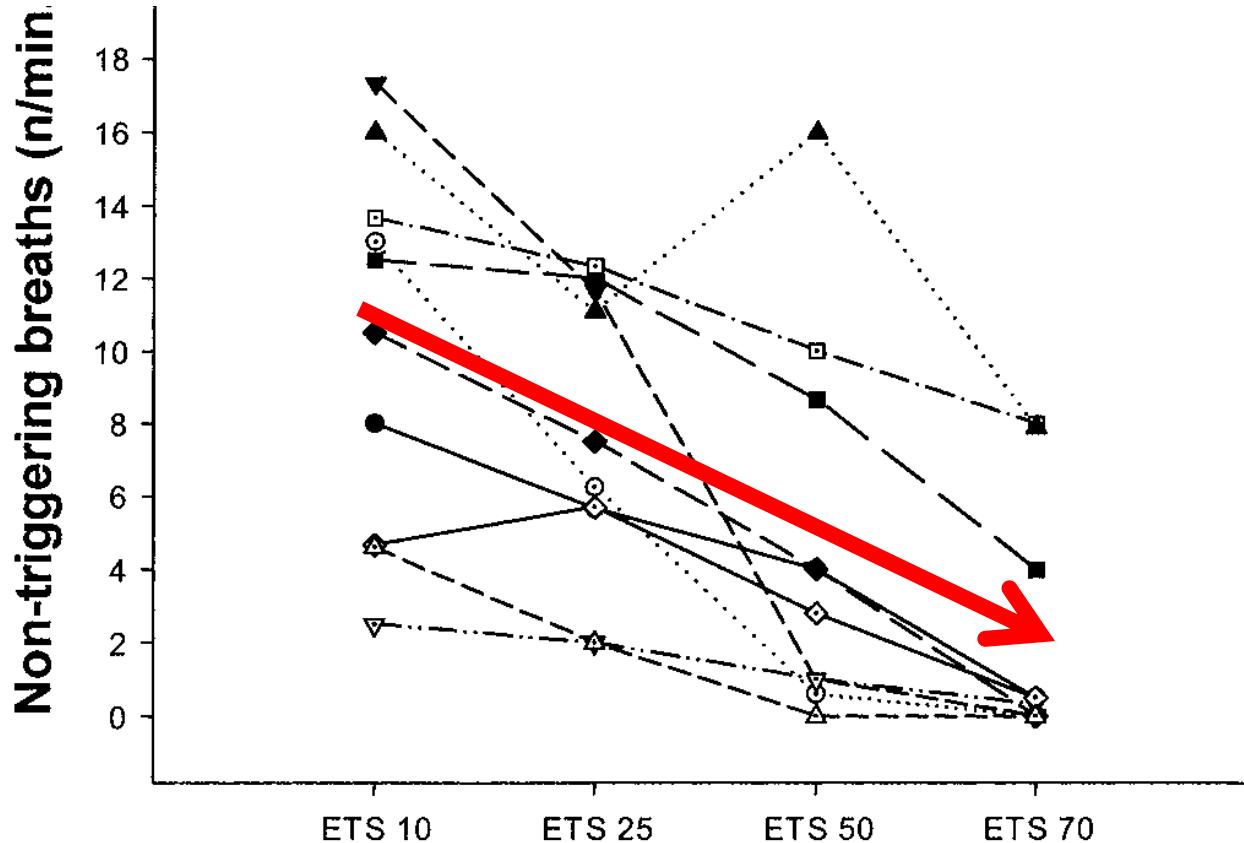


ET= Expiratory Trigger as % of peak inspiratory flow

Impact of Expiratory Trigger Setting on Delayed Cycling and Inspiratory Muscle Workload

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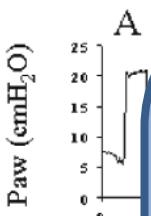


Setting expiratory trigger at a higher % of peak inspiratory flow in COPD pts improves patient–ventilator synchrony

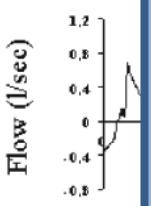


Asincronie di trigger inspiratorio

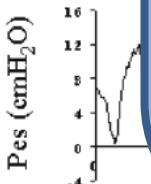
Sforzi



Diagnosi: distorsione del flusso (++) o deflessione Paw (>0.5 cmH₂O) non seguiti dall'erogazione dell'atto, FR ventilatore > FR paziente

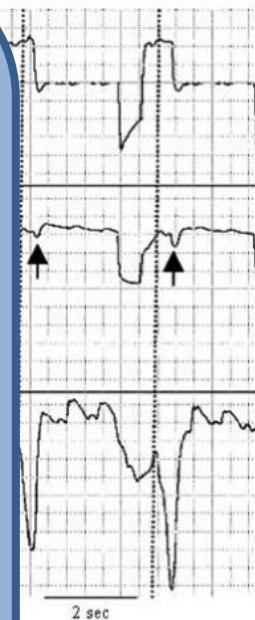


Cause: PEEPi, ↑↑ Psupp, basso drive respiratorio, ↓↓ sensibilità trigger inspiratorio



Soluzioni: ↑ tempo espiratorio (↑ % trigger exp. → ↓ tempo espiratorio), ↓ Psupp, ↑ sensibilità trigger inspiratorio, PEEPe 80% della PEEPi?

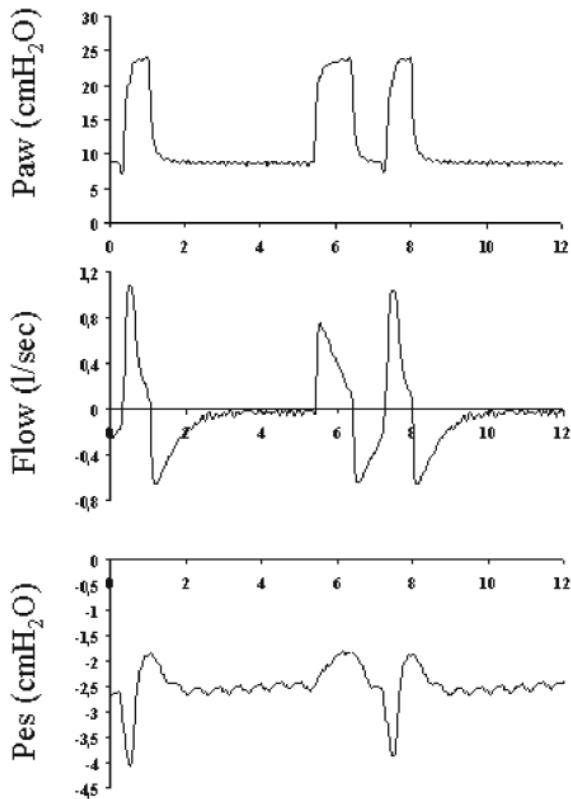
NB: se coesistono sforzi inefficaci e doppi trigger, priorità alla correzione degli sforzi inefficaci (fame d'aria dovuta agli sforzi inefficaci → ↑↑ drive inspiratorio negli atti successivi → doppi trigger!)





Asincronie di trigger inspiratorio

Autotrigger



Diagnosi: atto inspiratorio meccanico erogato senza essere preceduto da deflessione negativa Paw

Cause: oscillazioni cardiache, condensa, perdite, ↑↑ sensibilità del trigger inspiratorio

Soluzioni: rimuovere condensa dai tubi (modificare sistema di umidificazione?), correggere le perdite, ↓ sensibilità del trigger inspiratorio

Asincronie di trigger espiratorio

Ciclaggio espiratorio precoce (short cycle) e doppi cicli

Diagnosi:

Ciclaggio espiratorio precoce: distorsione delle tracce di flusso e Paw, $T_{insp} < \frac{1}{2} T_{insp}$ medio (T_{insp} medio = tempo durante il quale il flusso è positivo, calcolato su 30 atti)

Doppi cicli: due atti meccanico separati da un $T_{exp} < \frac{1}{2} T_{insp}$ medio

Cause: ↑↑ drive/sforzo inspiratorio, ↓↓ Psupp, ↑↑ % trigger exp. (inspirazione meccanica troppo corta), ciclaggio a tempo con un tempo inspiratorio massimo troppo basso, $\downarrow\downarrow$ costante di tempo del sistema respiratorio, cause cliniche neurologiche (encefalopatia/respiro di Kussmaul)

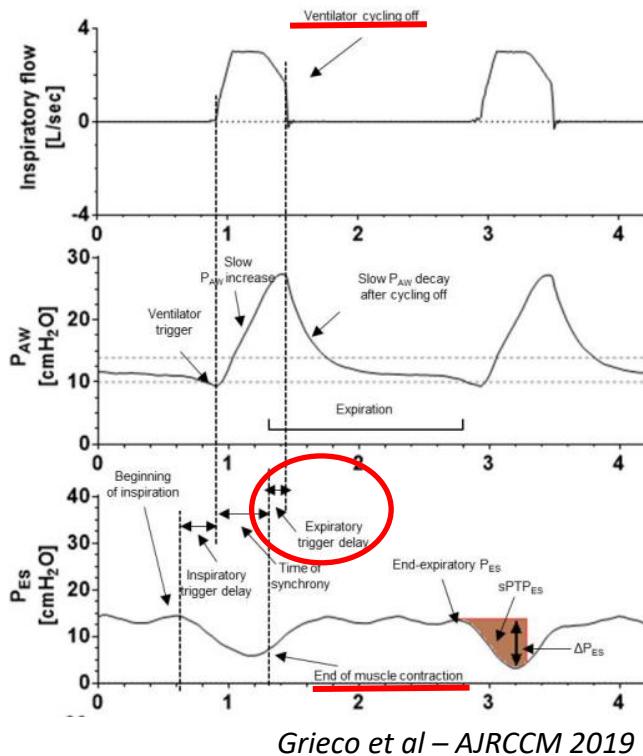
Soluzioni: esclusione cause neurologiche, \uparrow Psupp se sospetta sotto-assistenza ($\downarrow V_t$), \uparrow % trigger exp. (allungamento inspirazione meccanica), \uparrow tempo inspiratorio massimo (0.8 - 1 sec)

NB: se segni di ↑↑ drive/sforzo nel setting acuto, ventilazione protettiva controllata vs. controllo del drive respiratorio?



Asincronie di trigger espiratorio

Ciclaggio espiratorio tardivo e ciclaggio a tempo (*hang-up*)



Diagnosi:

*Ciclaggio espiratorio tardivo: T_{insp} > 2*T_{insp medio}*

Ciclaggio a tempo: segnalato dal ventilatore

Cause: perdite!!, ↓↓ % trigger exp. (inspirazione meccanica troppo lunga), ↑↑ costante di tempo del sistema respiratorio

Soluzioni: correggere le perdite, ↑ % trigger exp. (idealemente sopra il flusso delle perdite; accorciamento inspirazione meccanica), settare un tempo inspiratorio massimo di 0.8-1 sec, passare a una modalità ciclata a tempo (++ PACV)



Altre asincronie

Flow starvation



PSV/PACV: ↑ Psupp / Pinsp

VACV: ↑ Flusso o Vt vs. passare a modalità pressometrica assistita, dove lo sforzo muscolare del paziente concorre a determinare il picco di flusso inspiratorio

Pham et al – Crit Care Clin 2018

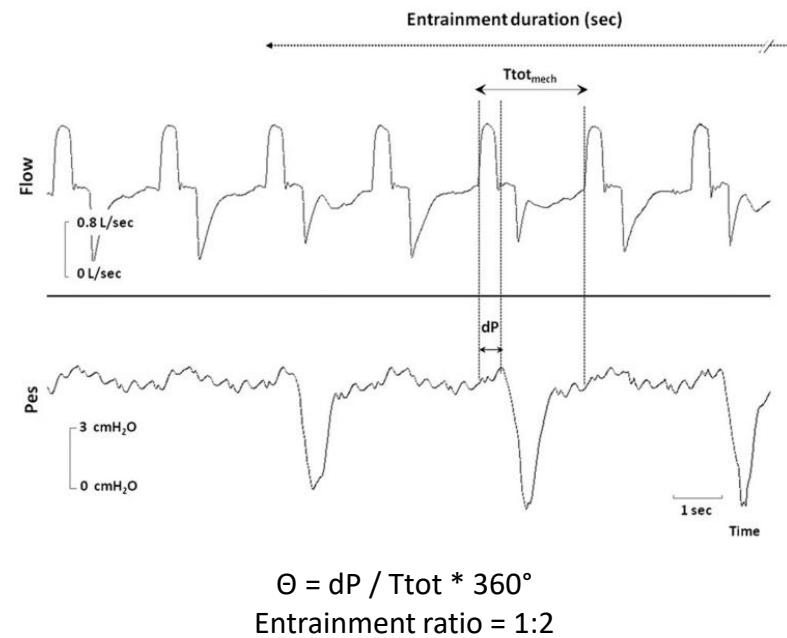
Cause: insufficiente flusso inspiratorio (i centri bulbari sono soddisfatti dal flusso; ++ se ↓ Vt), ↑↑ drive/sforzo inspiratorio (++ se ↑ Vt)

Soluzioni: ↑ flusso inspiratorio vs. ↓ drive respiratorio / ventilazione meccanica protettiva controllata?



Altre asincronie

Reverse triggering non armonico



Cause: ++ sedazione profonda, ↓ FR impostata



CHEST

Original Research

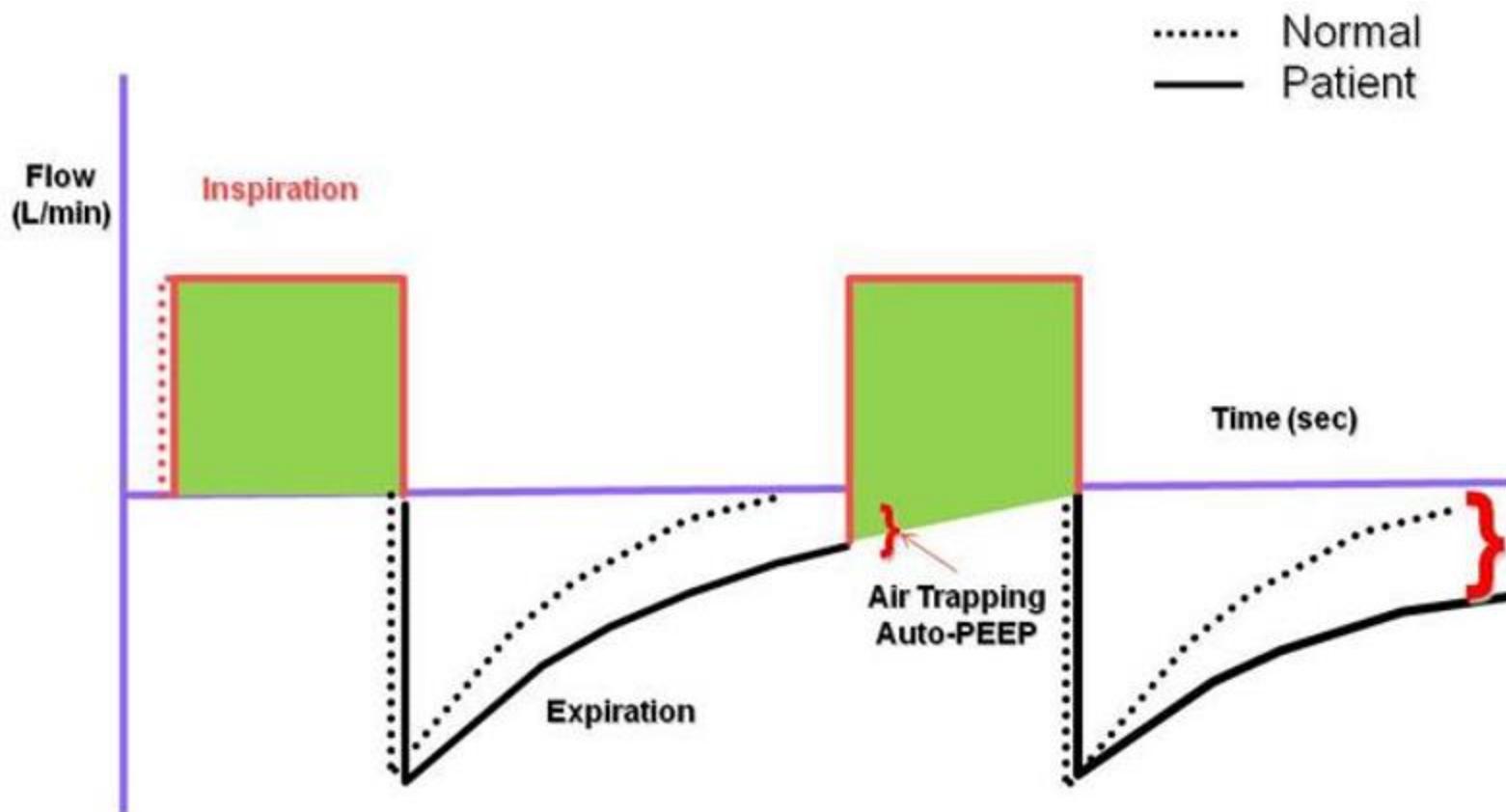
CRITICAL CARE

Mechanical Ventilation-Induced Reverse-Triggered Breaths

A Frequently Unrecognized Form of Neuromechanical Coupling

Evangelia Akoumianaki, MD; Aissam Lyazidi, PhD; Nathalie Rey, MD;
Dimitrios Matamis, MD; Nelly Perez-Martinez, MD; Raphael Giraud, MD;
Jordi Mancebo, MD; Laurent Brochard, MD; and Jean-Christophe Marie Richard, MD, PhD

sensibilità del trigger inspiratorio (non risolve le conseguenze dell'attività muscolare respiratoria incontrollata!)

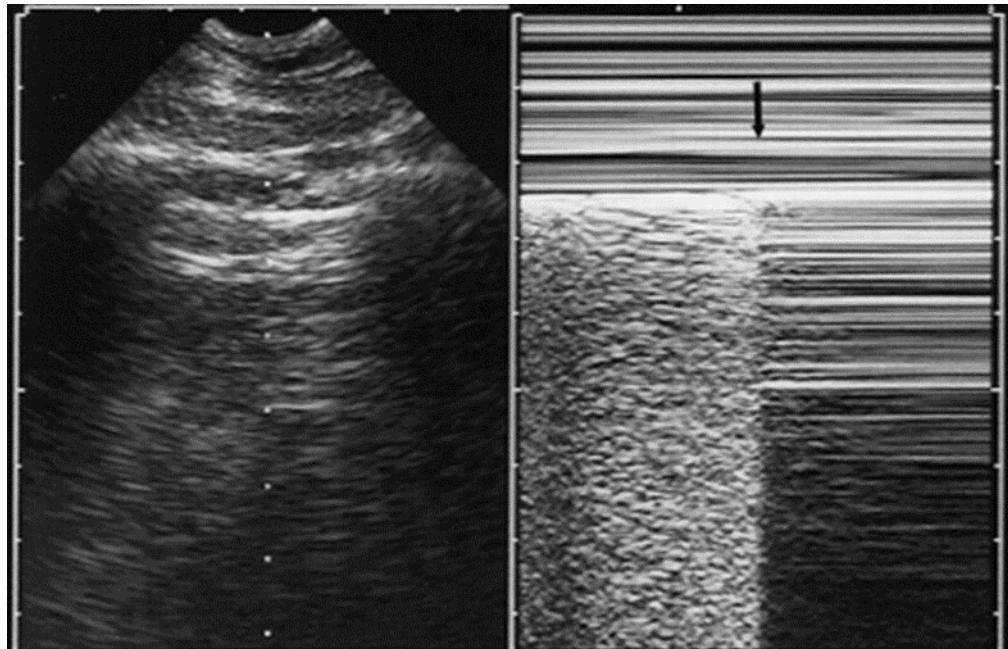


Valutazione radiologica e ultrasonografica

- Rx torace
- Tomografia computerizzata
- Ecografia polmonare e diaframmatica

Pneumotorace

- Assenza del Gliding
- Comparsa di linee A
- Assenza di linee B
- Lung Point: VPP 100%



The “lung point”: Association of absent lung sliding with “A lines”

M-mode: This sudden replacement of the physiologic granulous pattern by a horizontal pattern, at a precise location of the chest wall, is specific to pneumothorax.

RESEARCH

Open Access



A novel non-invasive method to detect excessively high respiratory effort and dynamic transpulmonary driving pressure during mechanical ventilation

Michele Bertoni^{1,2}, Irene Telias^{3,4}, Martin Umer^{3,5}, Michael Long⁶, Lorenzo Del Sorbo^{3,5}, Eddy Fan^{3,5,7}, Christer Sinderby^{3,4}, Jennifer Beck^{3,4}, Ling Liu⁸, Haibo Qiu⁸, Jenna Wong⁵, Arthur S. Slutsky^{3,4}, Niall D. Ferguson^{3,5,7,9,10}, Laurent J. Brochard^{3,4} and Ewan C. Goligher^{3,5,10,11*}

Abstract

Background: Excessive respiratory muscle effort during mechanical ventilation may cause patient self-inflicted lung injury and load-induced diaphragm myotrauma, but there are no non-invasive methods to reliably detect elevated transpulmonary driving pressure and elevated respiratory muscle effort during assisted ventilation. We hypothesized that the swing in airway pressure generated by respiratory muscle effort under assisted ventilation when the airway is briefly occluded (ΔP_{occ}) could be used as a highly feasible non-invasive technique to screen for these conditions.

Methods: Respiratory muscle pressure (P_{mus}), dynamic transpulmonary driving pressure ($\Delta P_{L,dyn}$, the difference between peak and end-expiratory transpulmonary pressure), and ΔP_{occ} were measured daily in mechanically ventilated patients in two ICUs in Toronto, Canada. A conversion factor to predict $\Delta P_{L,dyn}$ and P_{mus} from ΔP_{occ} was derived and validated using cross-validation. External validity was assessed in an independent cohort (Nanjing, China).

Results: Fifty-two daily recordings were collected in 16 patients. In this sample, P_{mus} and ΔP_L were frequently excessively high: P_{mus} exceeded 10 cm H₂O on 84% of study days and $\Delta P_{L,dyn}$ exceeded 15 cm H₂O on 53% of study days. ΔP_{occ} measurements accurately detected $P_{mus} > 10$ cm H₂O (AUROC 0.92, 95% CI 0.83–0.97) and $\Delta P_{L,dyn} > 15$ cm H₂O (AUROC 0.93, 95% CI 0.86–0.99). In the external validation cohort ($n = 12$), estimating P_{mus} and $\Delta P_{L,dyn}$ from ΔP_{occ} measurements detected excessively high P_{mus} and $\Delta P_{L,dyn}$ with similar accuracy (AUROC ≥ 0.94).

Conclusions: Measuring ΔP_{occ} enables accurate non-invasive detection of elevated respiratory muscle pressure and transpulmonary driving pressure. Excessive respiratory effort and transpulmonary driving pressure may be frequent in spontaneously breathing ventilated patients.

Keywords: Mechanical ventilation, Artificial respiration, Acute lung injury, Myotrauma, Respiratory monitoring

Conclusions: Measuring ΔP_{occ} enables accurate non-invasive detection of elevated respiratory muscle pressure and transpulmonary driving pressure. Excessive respiratory effort and transpulmonary driving pressure may be frequent in spontaneously breathing ventilated patients.

Effects of Gas Leak on Triggering Function, Humidification, and Inspiratory Oxygen Fraction During Noninvasive Positive Airway Pressure Ventilation*

Eriko Miyoshi, MD; Yuji Fujino, MD; Akinori Uchiyama, MD;
Takashi Mashimo, MD; and Masaji Nishimura, MD

Objectives: During noninvasive positive pressure ventilation (NPPV), the gas leak that commonly occurs around the mask can render NPPV ineffective. We evaluated the effects of gas leak on inspiratory trigger function during NPPV with bilevel pressure and ICU ventilators. In addition, we evaluated the effects of gas leak on fraction of inspired oxygen (FIO_2) and humidification.

Methods: Air leak was created at the airway opening of a model lung by establishing several different-size holes in the circuit. During simulated spontaneous breathing, we evaluated inspiratory trigger performance of two bilevel pressure ventilators (BiPAP Vision and BiPAP S/T-D; Respiration; Murrysville, PA) and two ICU ventilators (Puritan-Bennett 7200ae and Puritan-Bennett 840; Tyco Healthcare; Mansfield, MA). Inspiratory delay time and inspiratory trigger pressure were analyzed. FIO_2 at the airway opening and inside the model lung were evaluated during BiPAP S/T-D ventilation at supplemental oxygen flows of 3, 6, 9, 12 and 15 L/min. Measured oxygen concentration was compared to mathematically predicted levels. Finally, using two heated humidifiers, we evaluated the effect of gas leak on humidification.

Results: The bilevel pressure ventilators triggered properly at all levels of gas leak, and inspiratory triggering was more effective than with the ICU ventilators. Delivered FIO_2 with the BiPAP S/T-D ventilator was affected by gas leak and could be predicted mathematically unless the gas leak was large. With large gas leaks, although relative humidity was maintained, absolute humidity decreased.

Conclusion: Gas leak affected triggering of ICU ventilators, FIO_2 of the BiPAP S/T-D ventilator, and humidity with both types of humidifiers. *(CHEST 2005; 128:3691–3698)*

Key words: gas leak; humidification; inspiratory fraction of oxygen; noninvasive positive pressure ventilation; triggering function

Abbreviations: AH = absolute humidity; DT = inspiratory delay time; FIO_2 = fraction of inspired oxygen; NPPV = noninvasive positive pressure ventilation; PEEP = positive end-expiratory pressure; PI = inspiratory trigger pressure; PIP = peak inspiratory pressure; RH = relative humidity; RR = respiratory rate

RESEARCH

Open Access

A novel non-invasive method to detect excessively high respiratory effort and dynamic transpulmonary driving pressure during mechanical ventilation

Michele Bertoni^{1,2}, Irene Telias^{3,4}, Martin Umer^{3,5}, Michael Long⁶, Lorenzo Del Sorbo^{3,5}, Eddy Fan^{3,5,7}, Christer Sinderby^{3,4}, Jennifer Beck^{3,4}, Ling Liu⁸, Haibo Qiu⁸, Jenna Wong⁵, Arthur S. Slutsky^{3,4}, Niall D. Ferguson^{3,5,7,9,10}, Laurent J. Brochard^{3,4} and Ewan C. Goligher^{3,5,10,11*} 

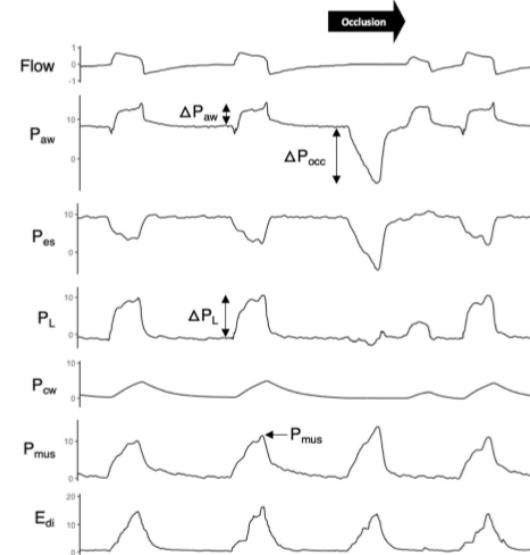


Fig. 1 Representative tracings obtained during the airway occlusion maneuver. Flow, airway pressure (P_{aw}), esophageal pressure (P_{es}), and diaphragm electrical activity (E_{di}) were recorded while a one-way end-expiratory occlusion permitting expiratory flow but not inspiratory flow (black arrow) was applied at a random interval. Transpulmonary pressure (P_L), obtained by digital subtraction of P_{aw} from P_{es} , signifies the dynamic stress applied to the lung. Chest wall elastic recoil pressure (ΔP_{occ}) was estimated by multiplying tidal volume by predicted chest wall elastance. Inspiratory effort was quantified by the peak inspiratory muscle pressure, P_{mus} , estimated as the difference between ΔP_{occ} and ΔP_{es} . Baseline P_{mus} is 0 cm H₂O by definition). Note that peak E_{di} did not differ between occluded and non-occluded breaths

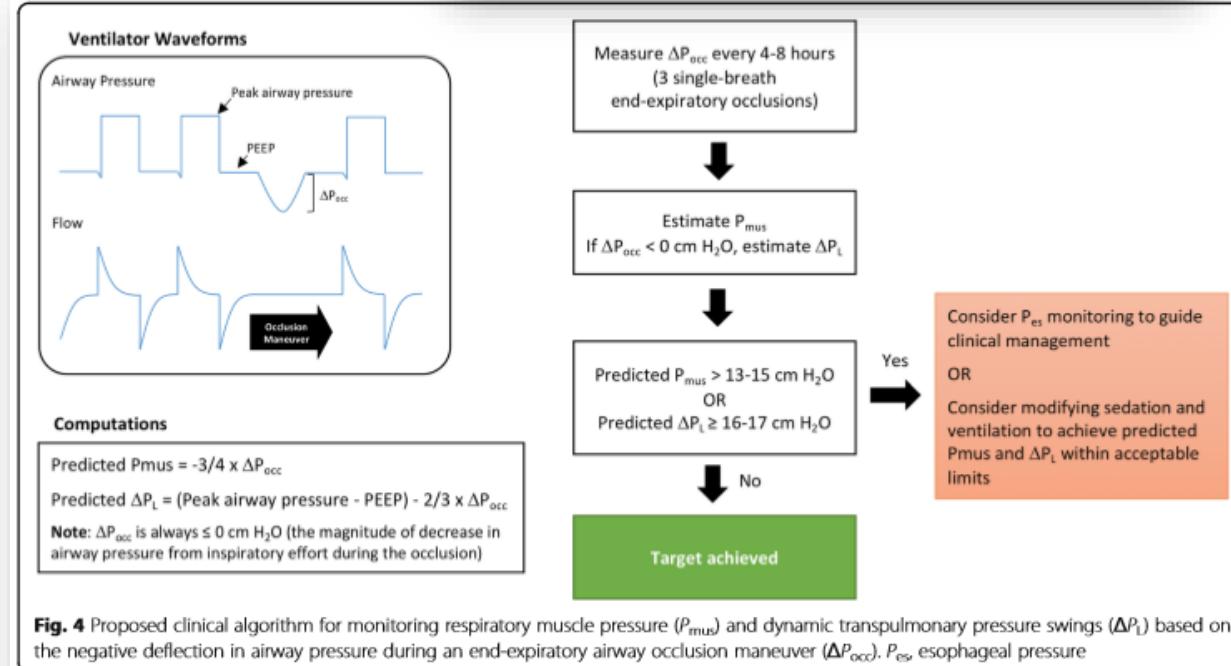


Fig. 4 Proposed clinical algorithm for monitoring respiratory muscle pressure (P_{mus}) and dynamic transpulmonary pressure swings (ΔP_L) based on the negative deflection in airway pressure during an end-expiratory airway occlusion maneuver (ΔP_{occ}). P_{es} , esophageal pressure

RESEARCH

Open Access

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

Fabiano Di Marco^{1*}, Stefano Centanni¹, Andrea Bellone², Grazia Messinesi³, Alberto Pesci³, Raffaele Scala⁴, Andreas Perren⁵ and Stefano Nava⁶

Abstract

Introduction: The analysis of flow and pressure waveforms generated by ventilators can be useful in the optimization of patient-ventilator interactions, notably in chronic obstructive pulmonary disease (COPD) patients. To date, however, a real clinical benefit of this approach has not been proven.

Methods: The aim of the present randomized, multi-centric, controlled study was to compare optimized ventilation, driven by the analysis of flow and pressure waveforms, to standard ventilation (same physician, same initial ventilator setting, same time spent at the bedside while the ventilator screen was obscured with numerical data always available). The primary aim was the rate of pH normalization at two hours, while secondary aims were changes in PaCO₂, respiratory rate and the patient's tolerance to ventilation (all parameters evaluated at baseline, 30, 120, 360 minutes and 24 hours after the beginning of ventilation). Seventy patients (35 for each group) with acute exacerbation of COPD were enrolled.

Results: Optimized ventilation led to a more rapid normalization of pH at two hours (51 vs. 26% of patients), to a significant improvement of the patient's tolerance to ventilation at two hours, and to a higher decrease of PaCO₂ at two and six hours. Optimized ventilation induced physicians to use higher levels of external positive end-expiratory pressure, more sensitive inspiratory triggers and a faster speed of pressurization.

Conclusions: The analysis of the waveforms generated by ventilators has a significant positive effect on physiological and patient-centered outcomes during acute exacerbation of COPD. The acquisition of specific skills in this field should be encouraged.

Trial registration: ClinicalTrials.gov NCT01291303.

Keywords: chronic obstructive pulmonary disease, acute exacerbation, non invasive ventilation, ventilators

