Polmonite da Covid 19:

Trattamento dell'Insufficienza respiratoria acuta ipossiemica

MA Pennisi

Gemelli

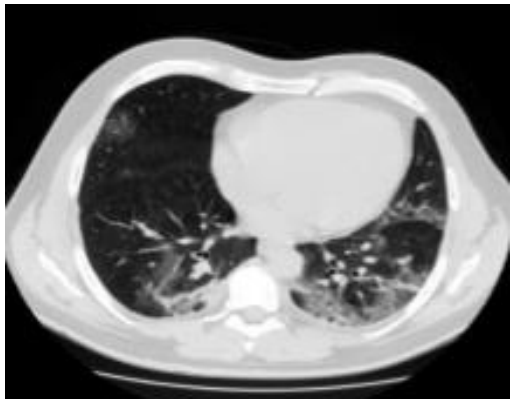
Gattinoni et al. Critical Care (2020) 24:154
https://doi.org/10.1186/s13054-020-02880-z

Critical Care

EDITORIAL Open Access

COVID-19 pneumonia: ARDS or not?

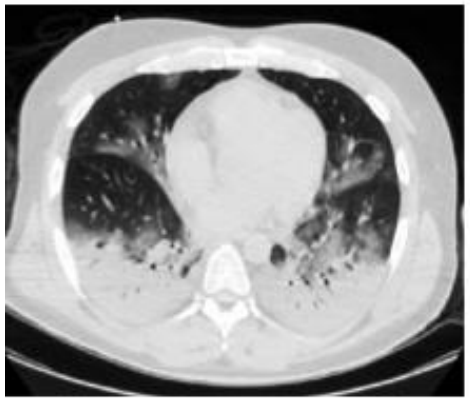
Luciano Gattinoni[1*], Davide Chiumello[2] and Sandra Rossi[3]



Type 1: Near normal pulmonary compliance with isolated viral pneumonia

In these patients, severe hypoxemia is associated with respiratory system compliance > 50 ml/cmH_2O. The lung's gas volume is high, the recruitability is minimal, and the hypoxemia is likely due to the loss of hypoxic pulmonary vasoconstriction and impaired regulation of pulmonary blood flow. Therefore, severe hypoxemia is primarily due to ventilation/perfusion (V_A/Q) mismatch. High PEEP and prone positioning do not improve oxygenation through recruitment of collapsed areas, but redistribute pulmonary perfusion, improving the V_A/Q relationship. Lung CT scans in those patients confirm that there are no significant areas to recruit, but the right-to-left venous admixture is typically around 50%.

SILI ?



Type 2: Decreased pulmonary compliance

In 20–30% of these COVID-19 patients admitted to the intensive care unit (ICU), severe hypoxemia is associated with compliance values < 40 ml/cmH_2O, indicating severe ARDS [3]. It is certainly possible that their lower compliance (i.e., lower gas volume and increased recruitability) is due to the natural evolution of the disease, but we cannot exclude the possibility that this severity of damage (increased edema) results in part from the initial respiratory management. Indeed, some of these hypoxemic patients receive CPAP or non-invasive ventilation before ICU admission and present with very high respiratory drives, vigorous inspiratory efforts, and highly negative intrathoracic pressures. Therefore, in addition to viral pneumonia, those patients likely have self-inflicted ventilator-induced lung injury [4].

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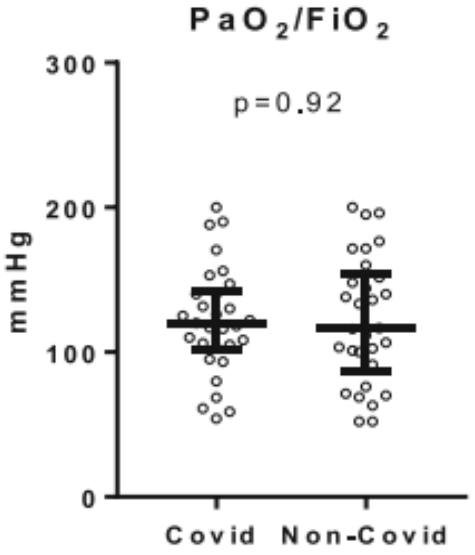
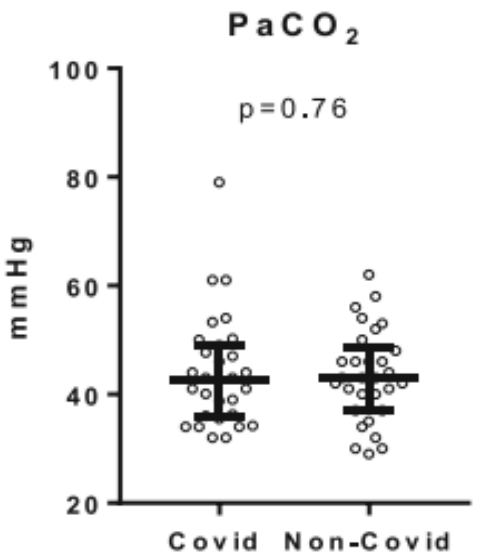
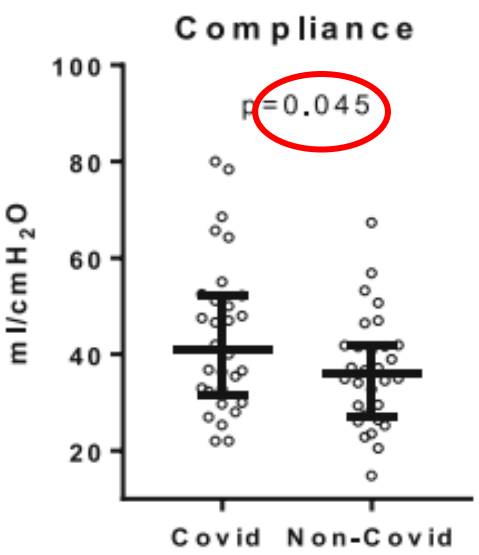
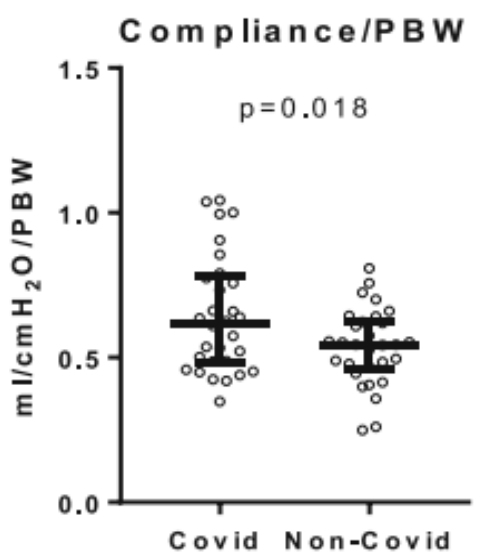
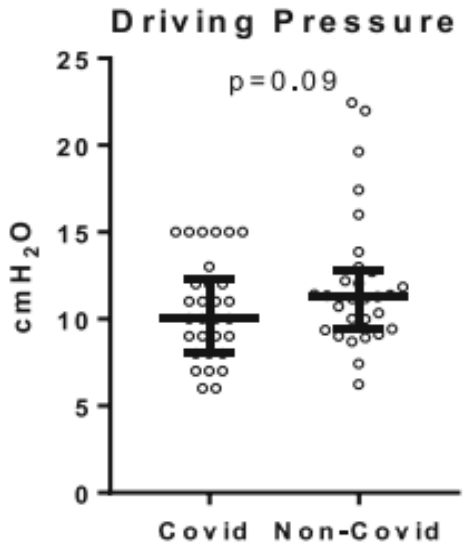
COVID-19 ARDS

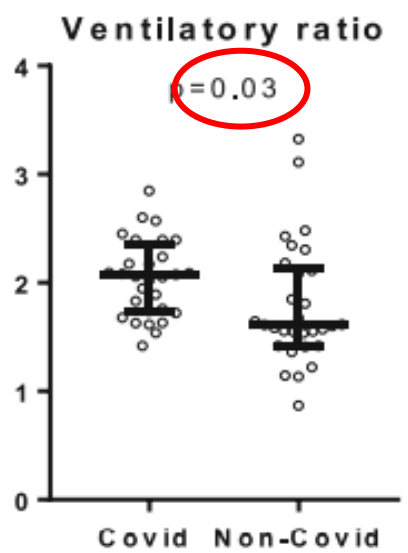


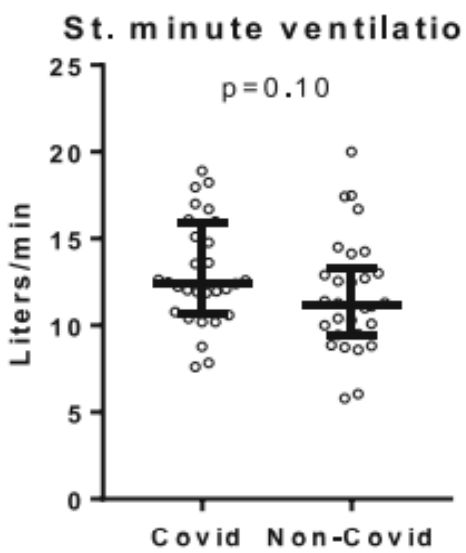


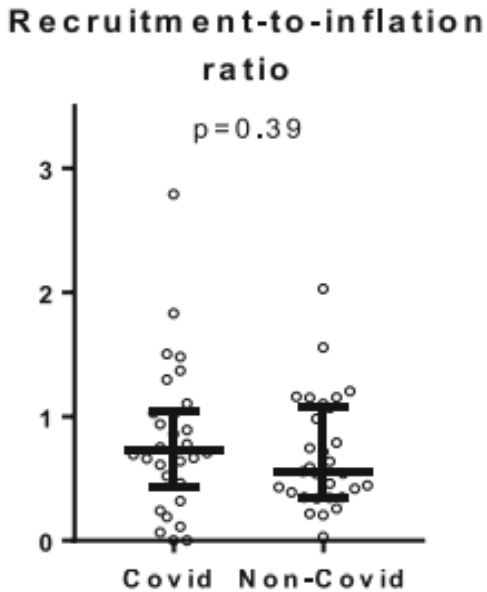




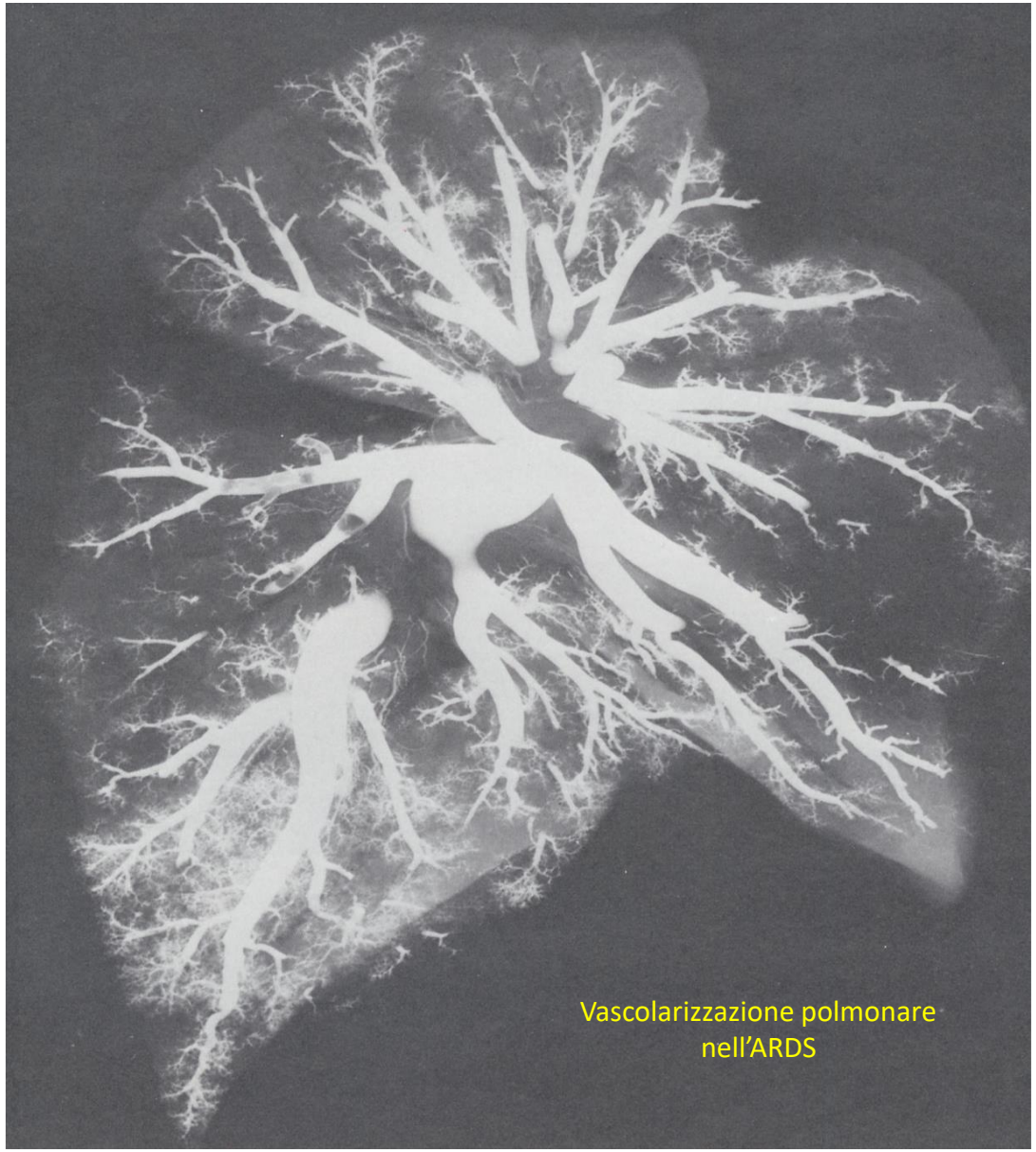








Ipertensione polmonare nell'ARDS



Grieco *et al. Critical Care* (2020) 24:529
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Critical Care

RESEARCH Open Access

Respiratory physiology of COVID-19-induced respiratory failure compared to ARDS of other etiologies

Early after establishment of mechanical ventilation, pts with COVID-19 show a conventional ARDS phenotype, with heterogeneity in respiratory mechanics, aeration loss related to the degree of hypoxemia, and inter-individually variable recruitability.

Physiological differences between COVID-19 pts and ARDS from other etiologies appear clinically negligible.

Until other data emerge, **clinicians treating COVID-19 patients should adhere to most recent guidelines regarding ARDS management**

Gestione non invasiva dell'insufficienza respiratoria acuta ipossiemica

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

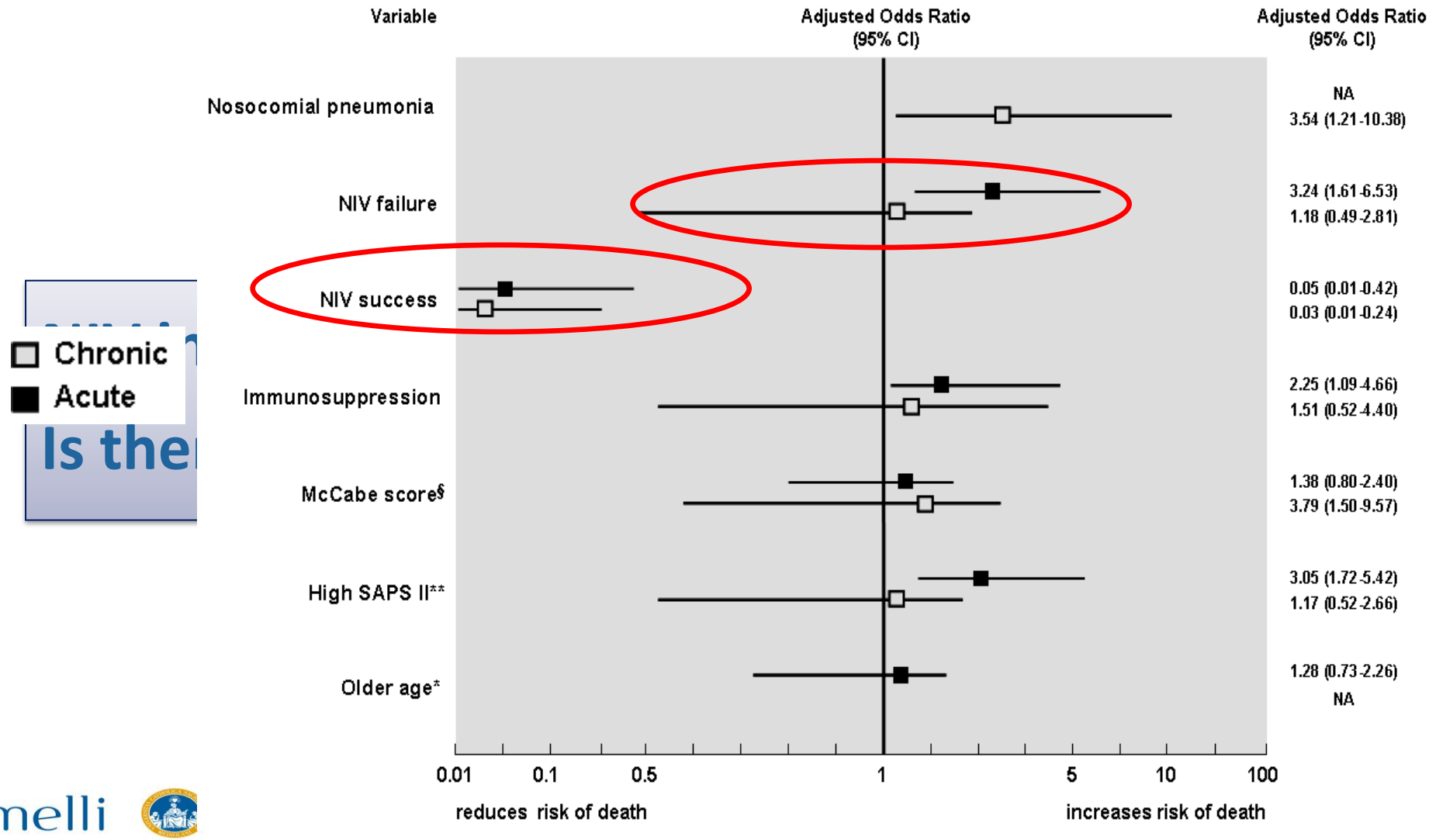
[#]: all in the setting of acute respiratory failure; [¶]: certainty of effect estimates: ⊕⊕⊕⊕, high; ⊕⊕⊕, moderate; ⊕⊕, low; ⊕, very low.

Intensive Care Med (2006) 32:1756–1765
DOI 10.1007/s00134-006-0324-1

ORIGINAL

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

Benefits and risks of success or failure of noninvasive ventilation



Chronic
Acute

Is the

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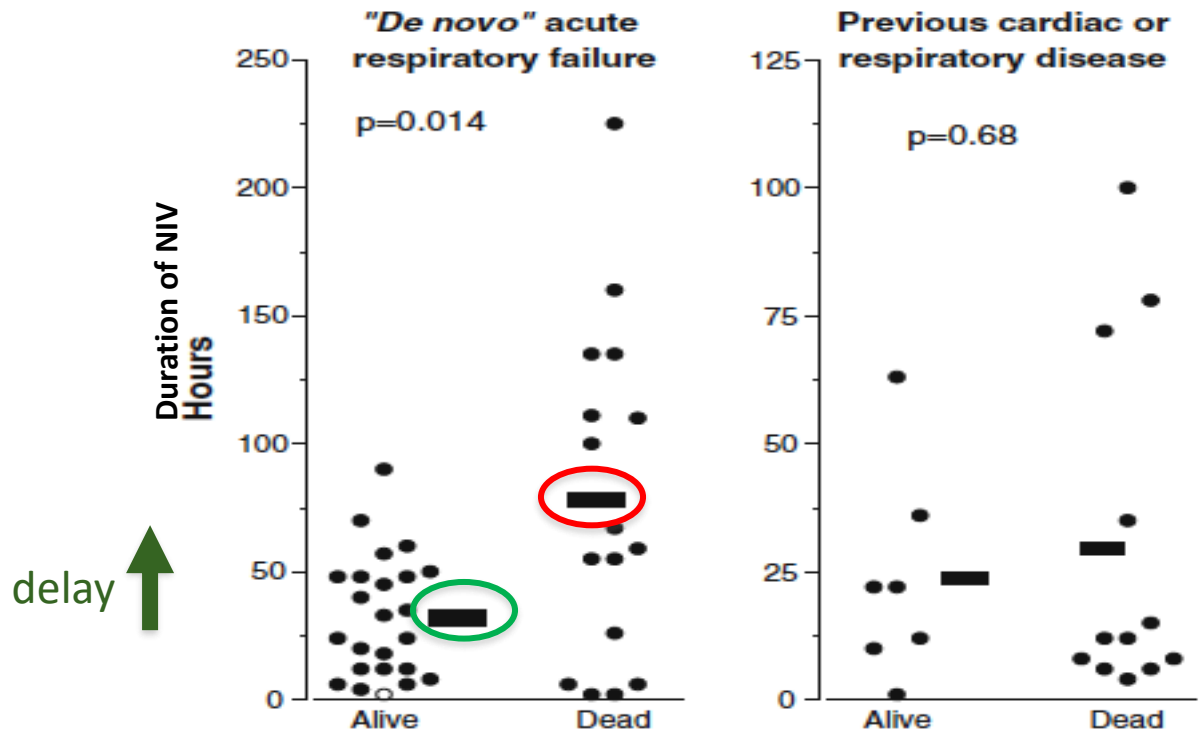
DELAYED INTUBATION INCREASES MORTALITY

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

ORIGINAL

Andres Carrillo
Gumersindo Gonzalez-Diaz
Miquel Ferrer
Maria Elena Martinez-Quintana
Antonia Lopez-Martinez
Noemi Llamas
Maravillas Alcazar
Antoni Torres

Non-invasive ventilation in community-acquired pneumonia and severe acute respiratory failure



Carrillo A et al. Intensive Care Med 2012;38:458-466

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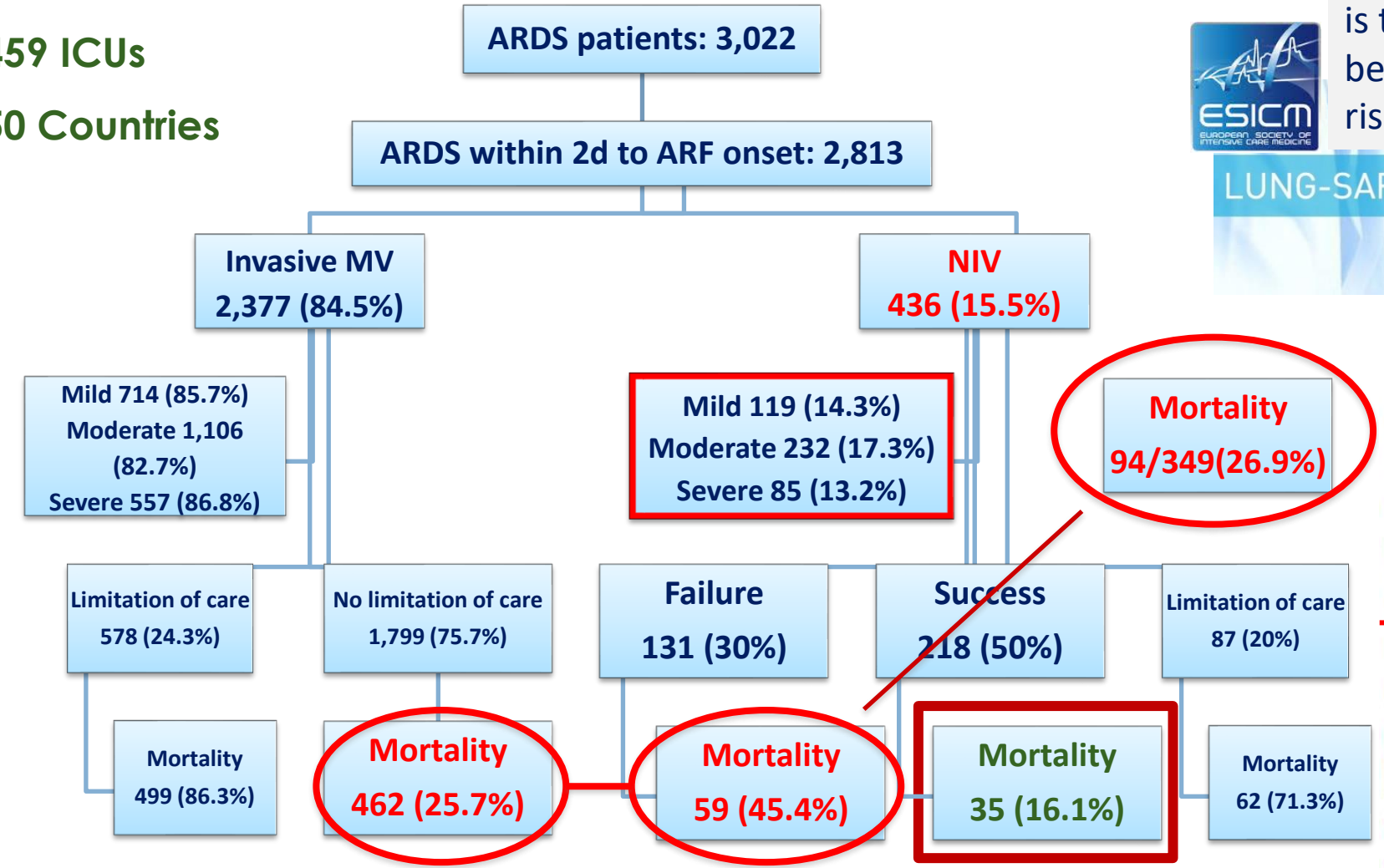
NIV IN PATIENTS WITH ARDS: THE LUNG-SAFE ANALYSIS

459 ICUs

50 Countries

NIV as a first-line strategy in pts with AHRF is the balance between the potential benefits of avoiding intubation and the risks deriving from SILI.



In conclusion, in a large cohort of patients with ARDS, NIV was used in 15% of cases, and was used to a similar extent across the severity categories. NIV failure occurred in more than one-third of patients with ARDS and in almost half of patients with moderate and severe ARDS. Mortality rates in patients that failed NIV were high. Of concern, NIV was associated with a worse adjusted ICU mortality than invasive-MV in patients with a Pa_{O_2}/FI_{O_2} lower than 150 mm Hg.

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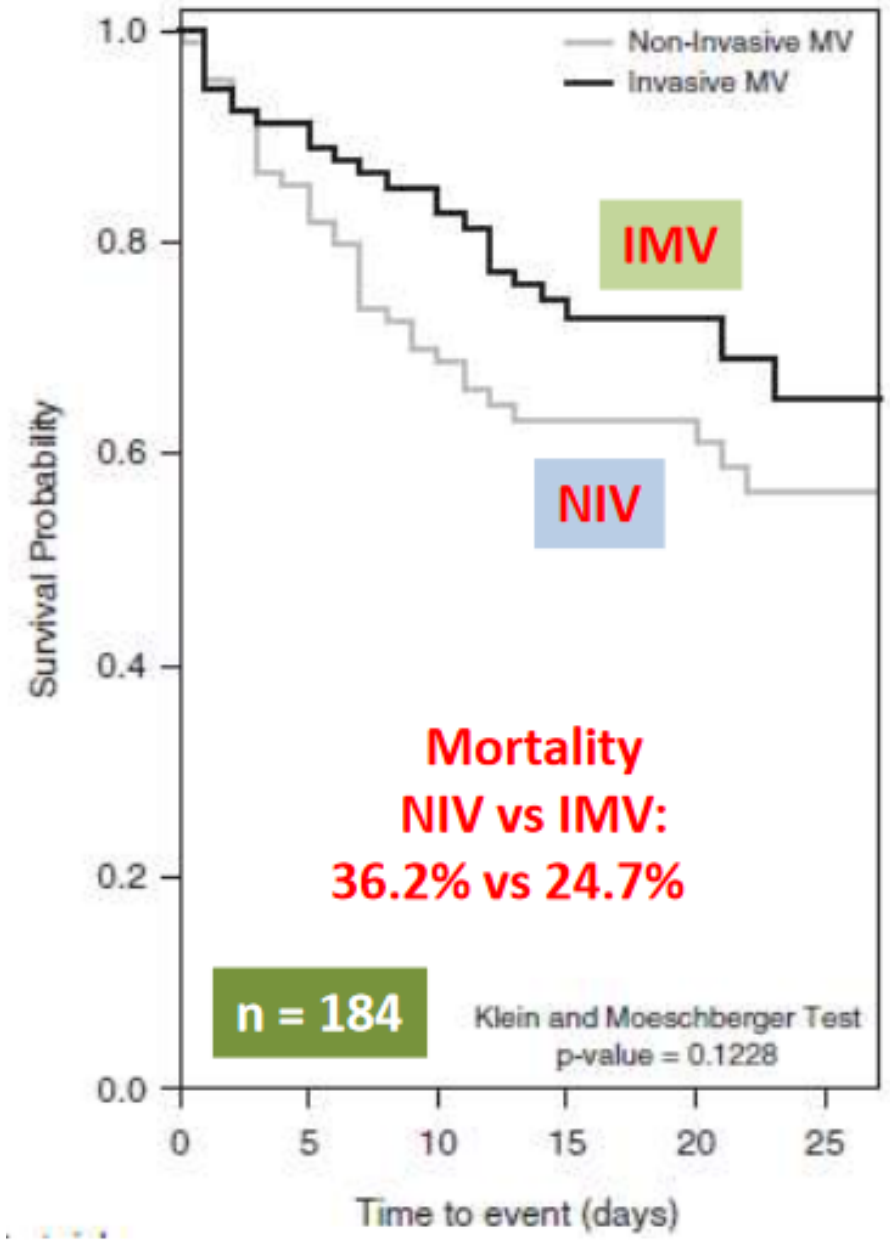
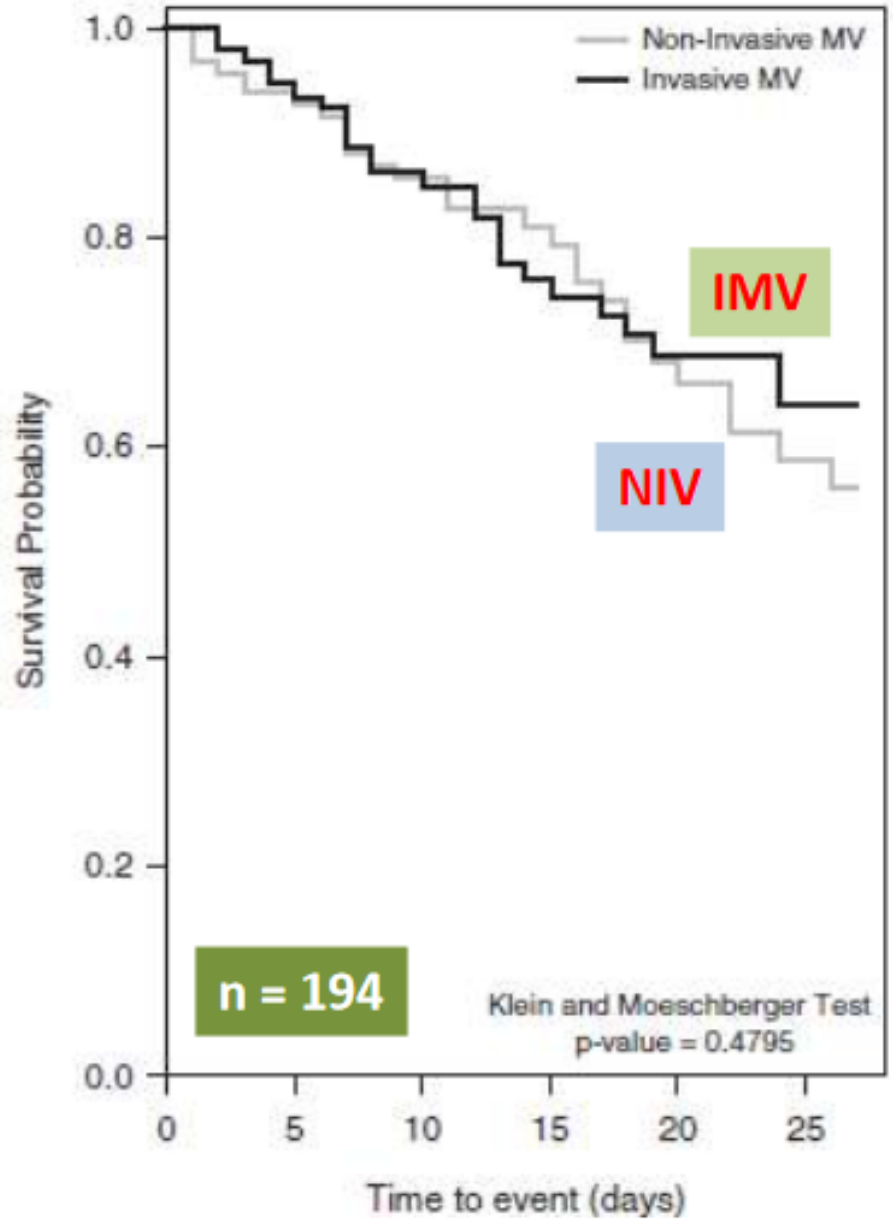
Bellani G et al. Am J Resp Crit Care Med 2016;10.1164/rccm.201606-1306OC (18-October-2016)

PaO2/FIO2 <150 mmHg



PaO2/FIO2 ≥150 mmHg



Bellani G et al. AJRCCM 2017;195(1):67–77

A multiple-center survey on the use in clinical practice of noninvasive ventilation as a first-line intervention for acute respiratory distress syndrome*

Massimo Antonelli, MD; Giorgio Conti, MD; Antonio Esquinas, MD; Luca Montini, MD; Salvatore Maurizio Maggiore, MD, PhD; Giuseppe Bello, MD; Monica Rocco, MD; Riccardo Maviglia, MD; Mariano Alberto Pennisi, MD; Gumersindo Gonzalez-Diaz, MD; Gianfranco Umberto Meduri, MD

Prospective, multiple-center cohort study
147 pts in 3 european ICUs

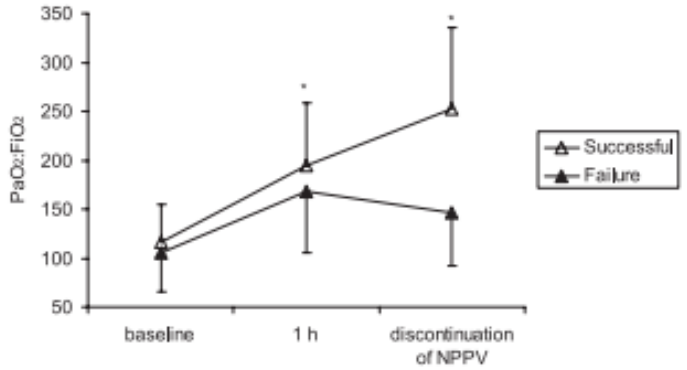


Figure 2. Pao_2:Fio_2 ratio over time in patients who avoided (successful) and required (failure) intubation. Discontinuation of noninvasive positive pressure ventilation (*NPPV*) corresponds to the discontinuation of ventilation for patients who avoided intubation and timing of endotracheal intubation for those who required intubation. *$p < .01$ between the two groups.

Table 5. Univariate and multivariate analysis of risk factors for endotracheal intubation

Variable	No. of Endotracheal Intubations/Total (%)	Univariate Analysis OR	95% CI	*p* Value[a]	Multivariate Analysis OR	95% CI	*p* Value[a]
Age, yrs							
≤58	28/77 (36)	1			1		
>58	40/70 (57)	2.33	1.2–4.52	.01	1.4	0.66–3	.38
Gender, male	50/93 (54)	2.38	1.18–4.78	.01	2.1	0.93–4.64	.07
SAPS II							
≤34	25/78 (32)	1			1		
>34	43/69 (62)	3.5	1.77–6.92	.0003	3.6	1.66–7.7	.001
Δ RR							
>4	32/89 (36)	1			1		
4	36/58 (62)	2.91	1.47–5.78	.002	1.94	0.86–4.36	.1
pH after 1 hr							
>7.37	25/75 (33)	1			1		
≤7.37	43/72 (60)	2.96	1.51–5.8	.001	1.91	0.85–4.31	.11
Pao_2/Fio_2 after 1 hr							
>175	28/79 (35)	1			1		
≤175	40/68 (59)	2.92	1.49–5.72	.001	2.34	1.1–5.15	.03

NIV applied as first-line intervention in ARDS avoided intubation in 54% of pts

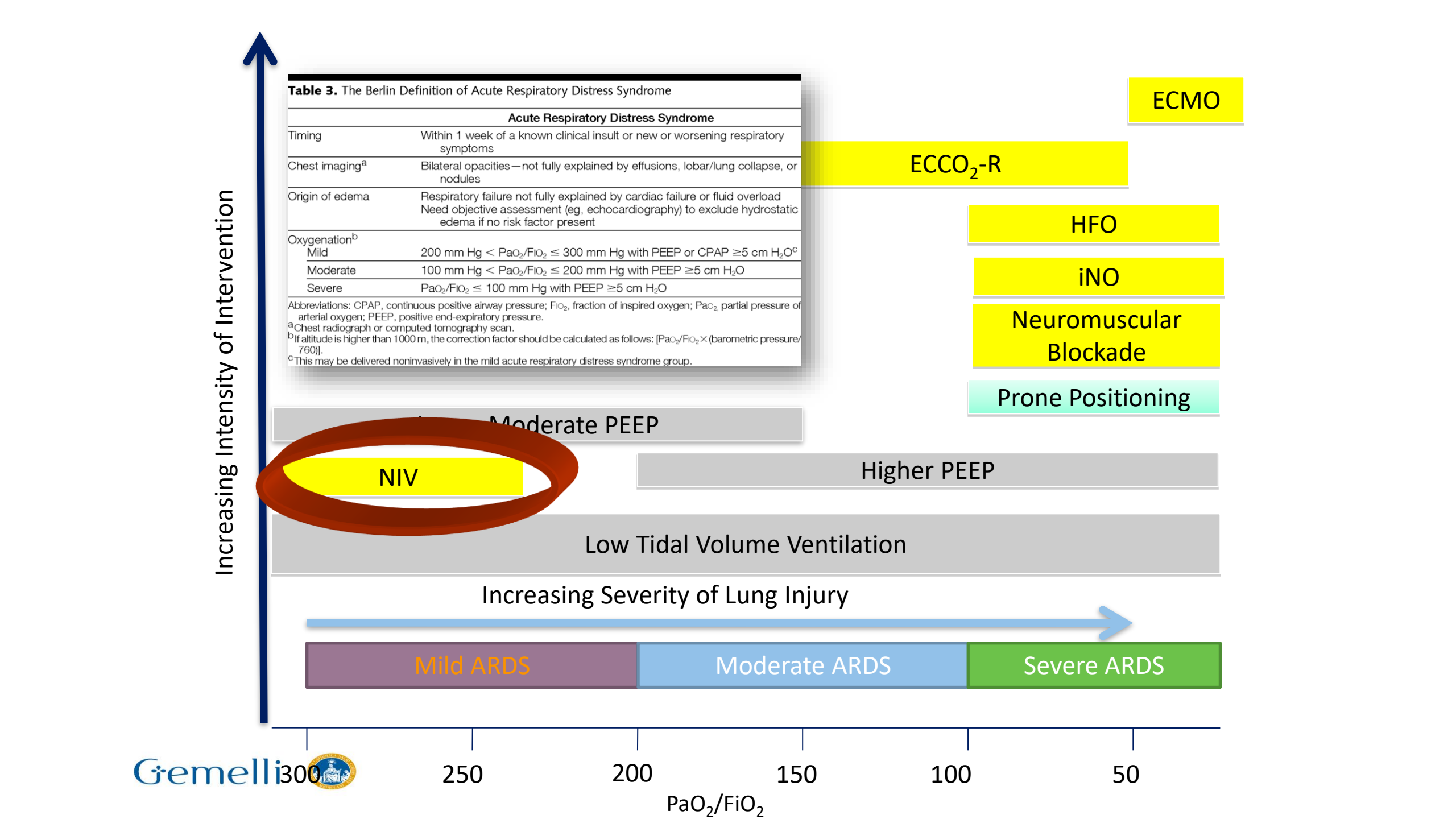


Table 3. The Berlin Definition of Acute Respiratory Distress Syndrome
Acute Respiratory Distress Syndrome
Timing
Within 1 week of a known clinical insult or new or worsening respiratory symptoms
Chest imaging[a]
Bilateral opacities—not fully explained by effusions, lobar/lung collapse, or nodules
Origin of edema
Respiratory failure not fully explained by cardiac failure or fluid overload
Need objective assessment (eg, echocardiography) to exclude hydrostatic edema if no risk factor present
Oxygenation[b]
Mild
200 mm Hg < PaO2/FIO2 ≤ 300 mm Hg with PEEP or CPAP ≥5 cm H2O[c]
Moderate
100 mm Hg < PaO2/FIO2 ≤ 200 mm Hg with PEEP ≥5 cm H2O
Severe
PaO2/FIO2 ≤ 100 mm Hg with PEEP ≥5 cm H2O
Abbreviations: CPAP, continuous positive airway pressure; FIO2, fraction of inspired oxygen; PaO2, partial pressure of arterial oxygen; PEEP, positive end-expiratory pressure.
[a] Chest radiograph or computed tomography scan.
[b] If altitude is higher than 1000 m, the correction factor should be calculated as follows: [PaO2/FIO2 × (barometric pressure/760)].
[c] This may be delivered noninvasively in the mild acute respiratory distress syndrome group.
ECMO
ECCO2-R
HFO
iNO
Neuromuscular Blockade
Prone Positioning
Moderate PEEP
NIV
Higher PEEP
Low Tidal Volume Ventilation
Increasing Intensity of Intervention
Increasing Severity of Lung Injury
Mild ARDS
Moderate ARDS
Severe ARDS
300
250
200
150
100
50
PaO2/FiO2
Gemelli

The NEW ENGLAND JOURNAL of MEDICINE

REVIEW ARTICLE

C. Corey Hardin, M.D., Ph.D., *Editor*

Noninvasive Respiratory Support for Adults with Acute Respiratory Failure

Laveena Munshi, M.D., Jordi Mancebo, M.D.,* and Laurent J. Brochard, M.D.

From the Interdepartmental Division of Critical Care, University of Toronto (L.M., L.J.B.), the Critical Care Department Sinai Health System (L.M.), and Keenan Research Centre for Biomedical Science, Li Ka Shing Knowledge Institute, Unity Health Toronto (L.J.B.) — all in Toronto; and the Intensive Care Department, Hospital Universitari de La Santa Creu I Sant Pau, Barcelona (J.M.). Dr. Brochard can be contacted at laurent.brochard@unityhealth.to or at the Keenan Research Centre for Biomedical Research, Li Ka Shing Knowledge Institute, Unity Health Toronto, 209 Victoria St., Toronto, ON, M5B 1T8, Canada.

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N Engl J Med 2022;387:1688-98.
DOI: 10.1056/NEJMra2204556

ACUTE RESPIRATORY FAILURE IS A COMMON INDICATION FOR ADMISSION to an intensive care unit. Invasive mechanical ventilation, particularly positive-pressure ventilation, has been the cornerstone of the management of severe forms of acute respiratory failure since the 1950s.[1] However, despite major advancements in critical care management, the complications and mortality associated with intubation and positive-pressure ventilation are not insignificant.[2] Efforts to circumvent invasive mechanical ventilation through the use of noninvasive devices have therefore garnered much attention. For some conditions, such as cardiogenic pulmonary edema and chronic obstructive pulmonary disease (COPD) exacerbations, noninvasive respiratory support is highly beneficial,[3] whereas for hypoxemic respiratory failure, the presence of associated conditions such as sepsis and shock[4] may make the use of noninvasive respiratory support risky and its benefits more difficult to delineate.

Three main methods of noninvasive support are used in the acute care setting: a high flow of gas delivered through a large-bore nonocclusive nasal cannula (i.e., high-flow nasal cannula), continuous positive airway pressure (CPAP), and noninvasive ventilation (i.e., pressure-support ventilation with positive end-expiratory pressure [PEEP]). In this review, we provide an overview of the physiological effects, different configurations, clinical indications, and evidence for the use of noninvasive respiratory support in adults with acute respiratory failure.

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ARDS : Self inflicted Lung Injury (SILI)

FIFTY YEARS OF RESEARCH IN ARDS

Spontaneous Breathing during Mechanical Ventilation

Risks, Mechanisms, and Management

Takeshi Yoshida[1,2,3,4], Yuji Fujino[4], Marcelo B. P. Amato[5], and Brian P. Kavanagh[1,2,3]

[1]Translational Medicine, [2]Department of Critical Care Medicine, and [3]Department of Anesthesia, Hospital for Sick Children, University of Toronto, Toronto, Ontario, Canada; [4]Intensive Care Unit, Osaka University Hospital, Suita, Japan; and [5]Laboratório de Pneumologia LIM-09, Disciplina de Pneumologia, Heart Institute (InCor) Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil

Abstract

Spontaneous respiratory effort during mechanical ventilation has long been recognized to improve oxygenation, and because oxygenation is a key management target, such effort may seem beneficial. Also, disuse and loss of peripheral muscle and diaphragm function is increasingly recognized, and thus spontaneous breathing may confer additional advantage. Reflecting this, epidemiologic data suggest that the use of partial (vs. full) support modes of ventilation is increasing.

Notwithstanding the central place of spontaneous breathing in mechanical ventilation, accumulating evidence indicates that it may cause—or worsen—acute lung injury, especially if acute respiratory distress syndrome is severe and spontaneous effort is vigorous. This Perspective reviews the evidence for this phenomenon, explores mechanisms of injury, and provides suggestions for clinical management and future research.

Keywords: mechanical ventilation; acute respiratory distress syndrome; spontaneous breathing; ventilator-induced lung injury

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Am J Respir Crit Care Med Vol 195, Iss 8, pp 985–992, Apr 15, 2017

CRITICAL CARE PERSPECTIVE

Mechanical Ventilation to Minimize Progression of Lung Injury in Acute Respiratory Failure

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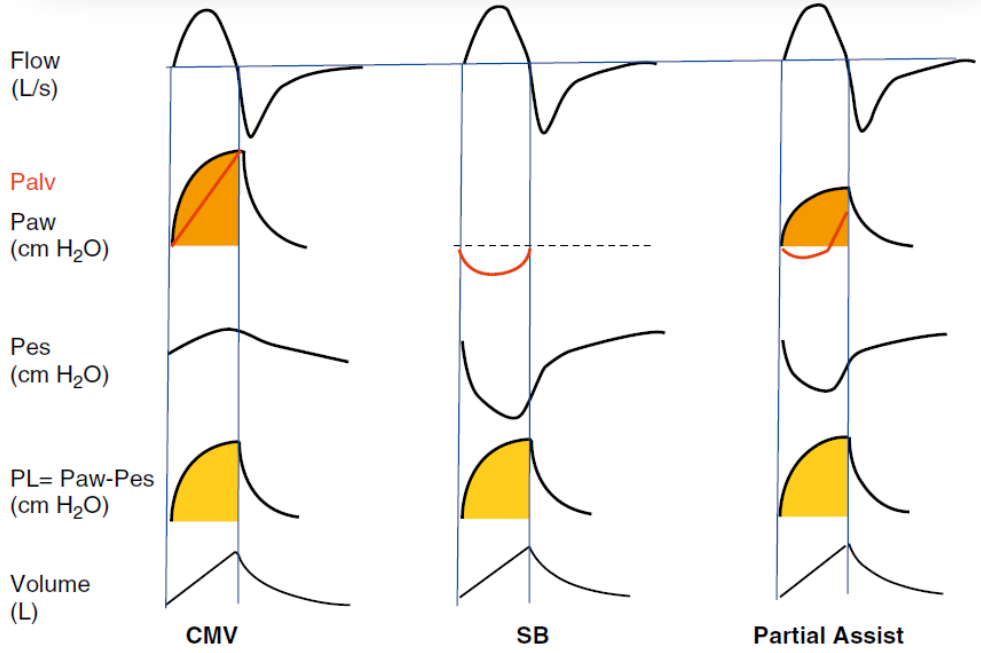
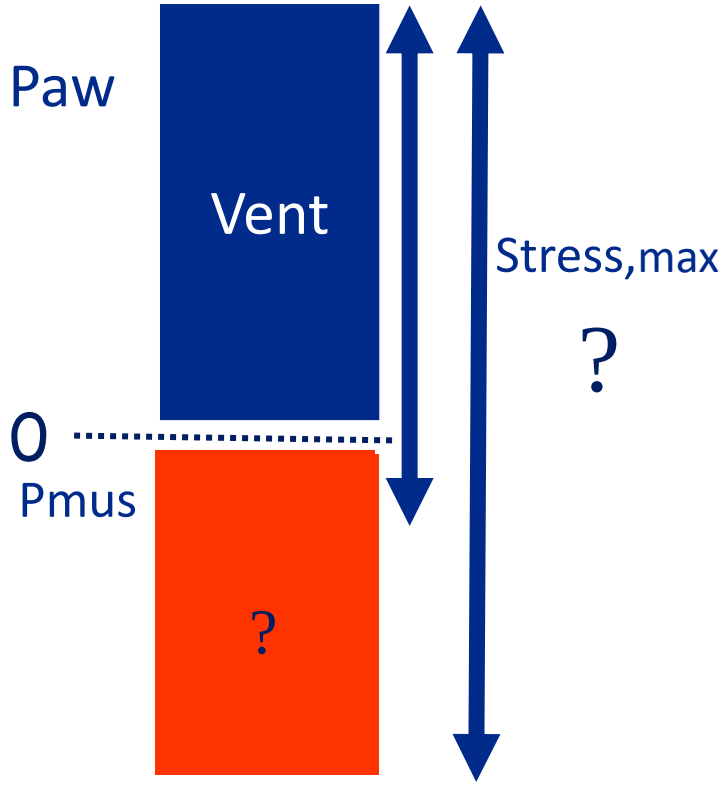


Figure 1. Illustration of how the same transpulmonary pressure can be generated by a mechanical breath during controlled mechanical ventilation, a spontaneous breath, or a combination of the two during partial ventilatory support. From *top* to *bottom*: flow, alveolar pressure (in *red*) and airway opening pressure, esophageal pressure, calculated transpulmonary pressure, and tidal volume. *Vertical blue lines* delimit inspiration time. CMV = controlled mechanical ventilation; Palv = alveolar pressure; Paw = airway opening pressure; Pes = esophageal pressure swings; P_L = transpulmonary pressure; SB = spontaneous breath.



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FIFTY YEARS OF RESEARCH IN ARDS

Spontaneous Breathing during Mechanical Ventilation

Risks, Mechanisms, and Management

Takeshi Yoshida[1,2,3,4], Yuji Fujino[4], Marcelo B. P. Amato[5], and Brian P. Kavanagh[1,2,3]

[1]Translational Medicine, [2]Department of Critical Care Medicine, and [3]Department of Anesthesia, Hospital for Sick Children, University of Toronto, Toronto, Ontario, Canada; [4]Intensive Care Unit, Osaka University Hospital, Suita, Japan; and [5]Laboratório de Pneumologia LIM-09, Disciplina de Pneumologia, Heart Institute (InCor) Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil

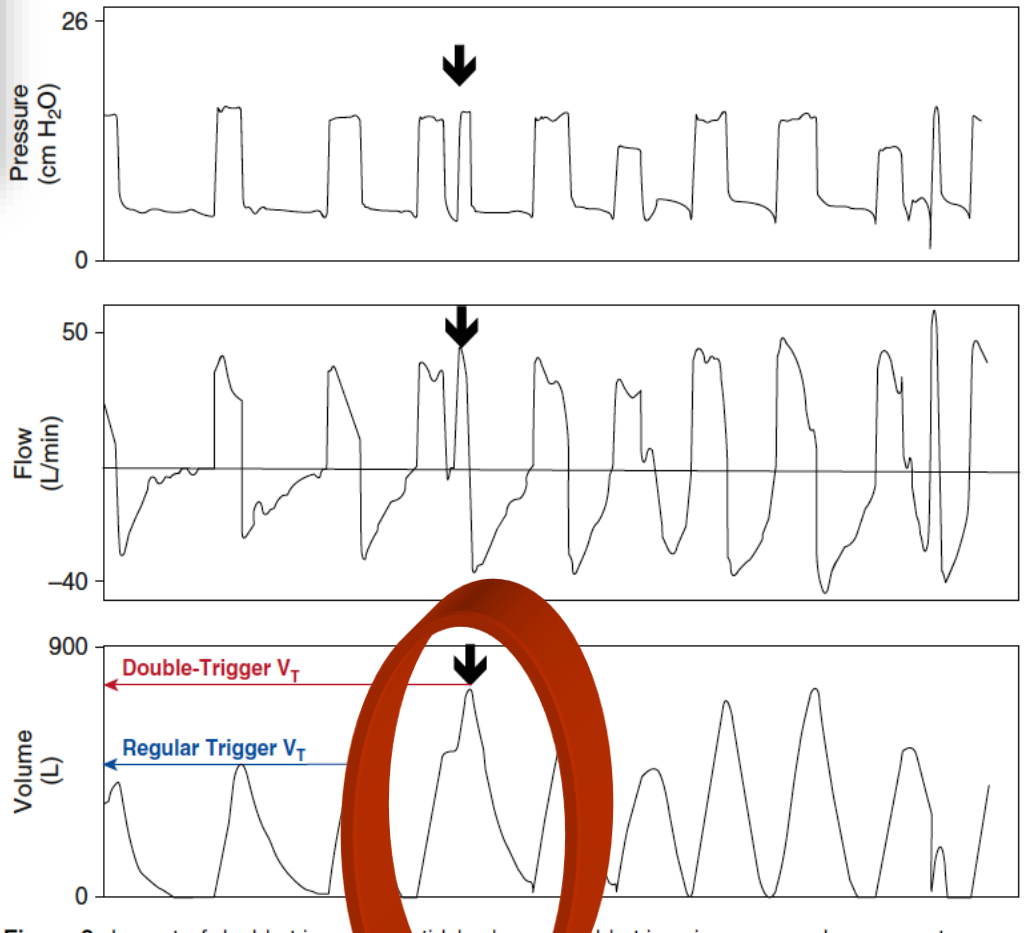

Figure 3. Impact of double trigger[illegible] tidal volume. [illegible]uble triggering occurs when a spontaneous effort triggers a (second) ventilator br[illegible]efore the in[illegible] breath has been completely exhaled (*arrow*). The pressure–time trace (*top*) and flow[illegible]ce (*mi[illegible]le*) demonstrate the occurrence of the additional breath but do not give a sense that both [illegible] are summed; this is apparent from the volume–time trace (*bottom*) indicating that the double triggering results in a substantially larger (potentially injurious) VT (*red*) compared with regular triggering (*blue*). Adapted by permission from Reference 56.

CRITICAL CARE PERSPECTIVE

FIFTY YEARS OF RESEARCH IN ARDS

Spontaneous Breathing during Mechanical Ventilation

Risks, Mechanisms, and Management

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[1]Translational Medicine, [2]Department of Critical Care Medicine, and [3]Department of Anesthesia, Hospital for Sick Children, University of Toronto, Toronto, Ontario, Canada; [4]Intensive Care Unit, Osaka University Hospital, Suita, Japan; and [5]Laboratório de Pneumologia LIM-09, Disciplina de Pneumologia, Heart Institute (InCor) Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil

Pressione trans vascolare
Pcap-Ppl =12 - 10 = +2

Presssione trans vascolare
Pcap-Ppl =8- - 20 = +28

Ventilazione
Meccanica
controllata

Ventilazione In PSV

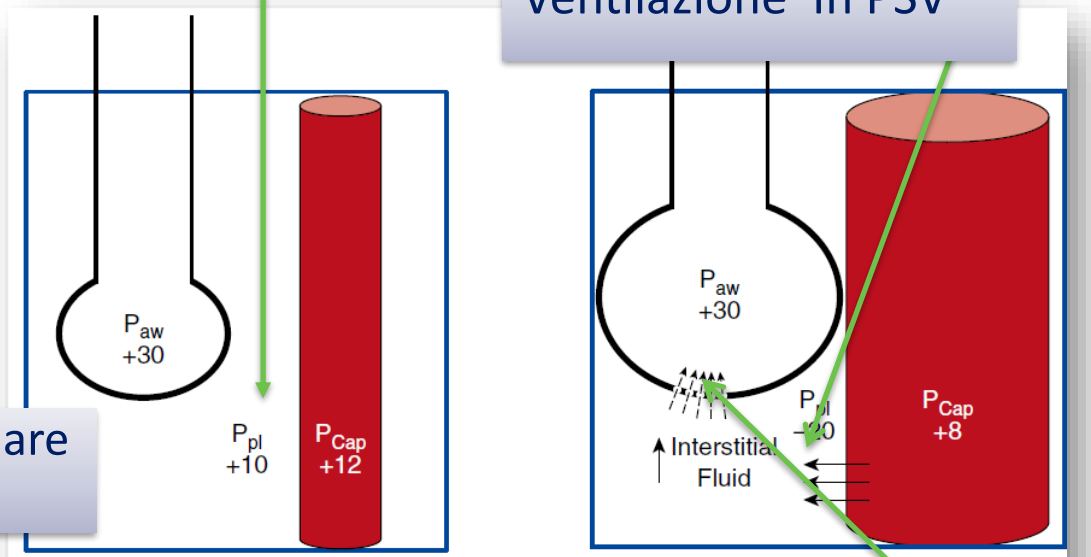

Figure 1. Spontaneous effort and transpulmonary and transvascular pressures. During a mechanical breath (*left*), the transpulmonary pressure (Paw – Ppl = P_L) distending the lung is +20 (30 – 10); the pulmonary blood vessels are compressed by the positive-pressure breath and the transvascular pressure (Pcap – Ppl) is low (assume 12 – 10 = 2). When spontaneous effort is added (*right*), the P_L (30 + 20 = +50) is greater, thereby increasing the V_T and causing lung injury. In addition, the negative Ppl (−20) distends the pulmonary blood vessels and increases perfusion; the transvascular pressure is greater (assume 8 – −20 = +28), increasing fluid shift to the interstitium. In the presence of injury, permeability (and therefore propensity to alveolar edema) is increased. Paw = airway pressure; Pcap = capillary hydrostatic pressure; Ppl = pleural pressure.

Pressione transpolmonare
Paw-Ppl =30-10= 20

Transpulmonary Pressure
Paw-Ppl =30+20 = 50

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CRITICAL CARE PERSPECTIVE

FIFTY YEARS OF RESEARCH IN ARDS

Spontaneous Breathing during Mechanical Ventilation

Risks, Mechanisms, and Management

Takeshi Yoshida[1,2,3,4], Yuji Fujino[4], Marcelo B. P. Amato[5], and Brian P. Kavanagh[1,2,3]

[1]Translational Medicine, [2]Department of Critical Care Medicine, and [3]Department of Anesthesia, Hospital for Sick Children, University of Toronto, Toronto, Ontario, Canada; [4]Intensive Care Unit, Osaka University Hospital, Suita, Japan; and [5]Laboratório de Pneumologia LIM-09, Disciplina de Pneumologia, Heart Institute (InCor) Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil

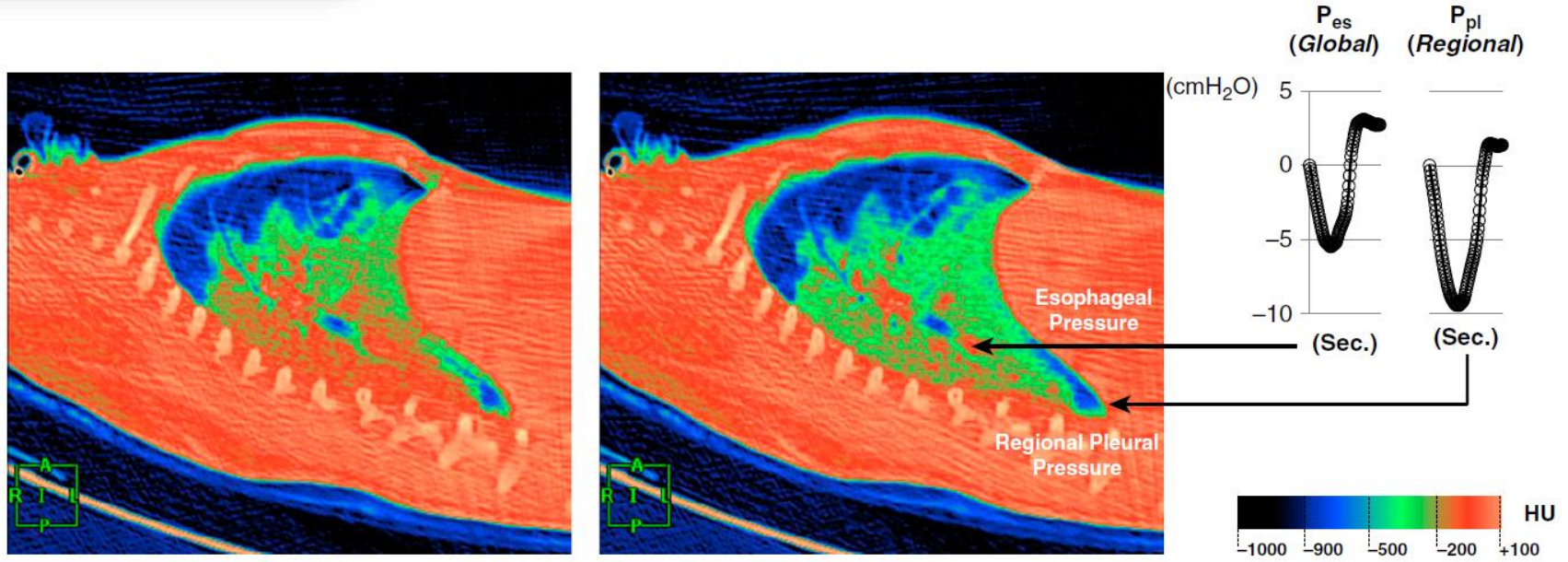


Figure 2. Spontaneous effort and distribution of regional ventilation and pleural pressure. Dynamic computed tomographic scan in end-expiration (*left*) demonstrates that the aerated lung (*blue*) is nondependent, while the dependent lung is densely atelectatic (*red*). At end-inspiration during a spontaneous breath (*middle*), there is little change in the nondependent aerated lung (*blue*); the dependent lung, previously densely atelectatic (*red*), is now partially aerated (*green/red*) (i.e., tidal recruitment). The inspiratory pleural pressure traces (*right*), measured at the *arrow tips*, show the negative deflections ("swings") in regional Ppl and global Pes during inspiration. However, the "swing" in regional Ppl is greater (by twofold) than the "swing" in Pes, indicating that diaphragm contraction results in greater distending pressure applied to the regional lung near the diaphragm, compared with the pressure transmitted to the remainder of the lung (i.e., Pes). A = anterior; HU = Hounsfield units; I = inferior; L = left; P = posterior; Pes = esophageal pressure; Ppl = pleural pressure; R = right.

Noninvasive Ventilation for patients with Hypoxemic ARF

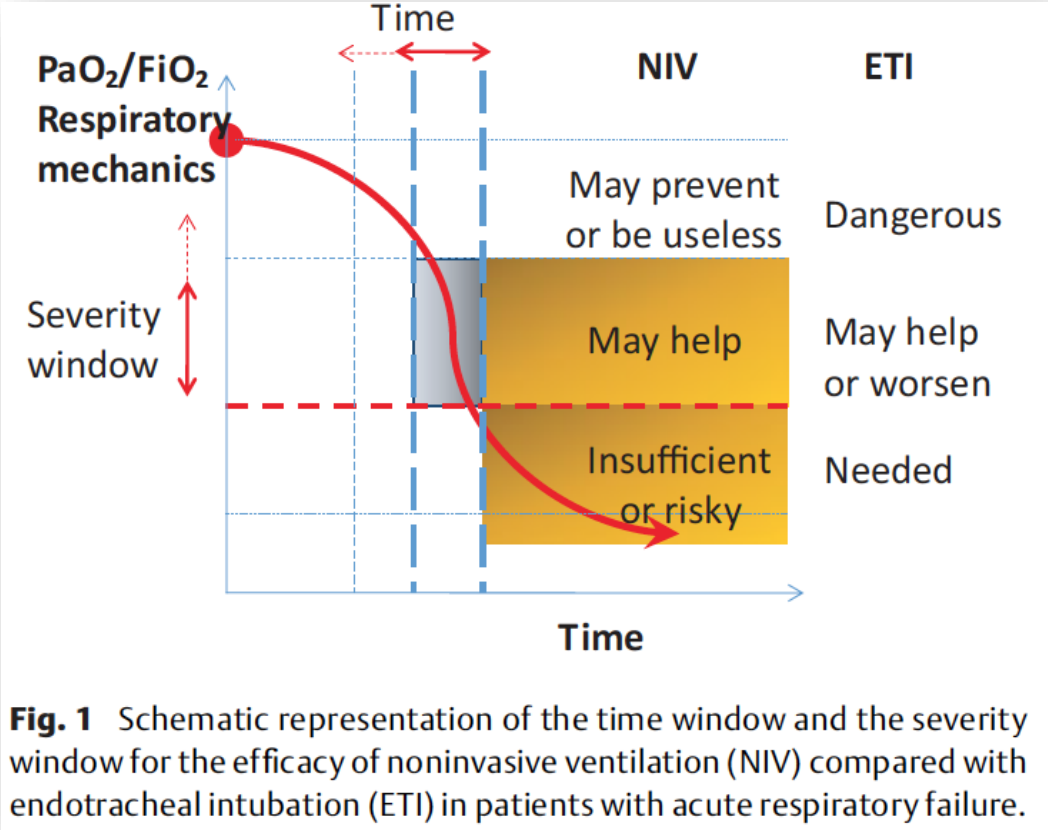


Fig. 1 Schematic representation of the time window and the severity window for the efficacy of noninvasive ventilation (NIV) compared with endotracheal intubation (ETI) in patients with acute respiratory failure.

Come identificare i pazienti in cui i rischi della NIV superano i vantaggi?

Semin Respir Crit Care Med 2014;35:492–500.

Gemelli

Intensive Care Med (2001) 27: 1718–1728
DOI 10.1007/s00134-001-1114-4

ORIGINAL

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 hypoxemic respiratory failure: a multi-center study

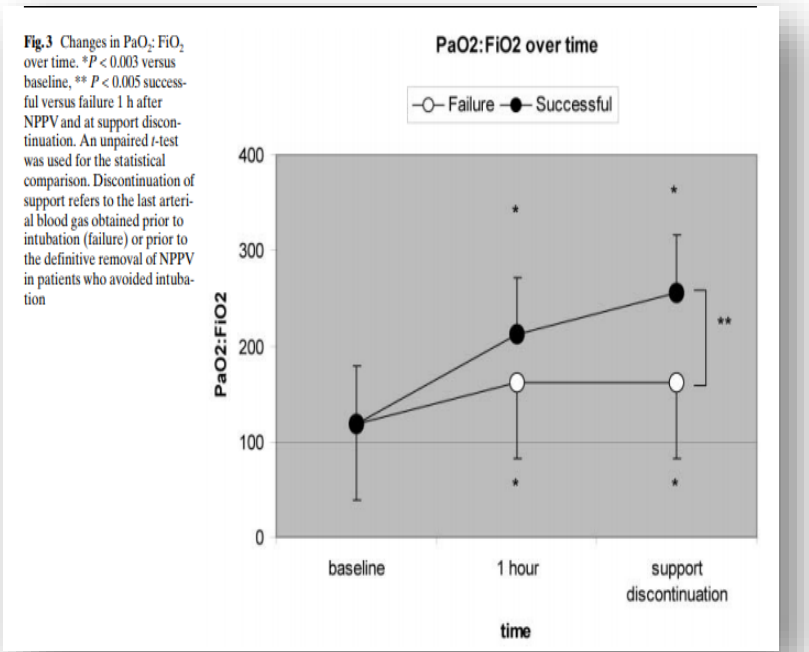


Fig.3 Changes in PaO_2: FiO_2 over time. *$P < 0.003$ versus baseline, ** $P < 0.005$ successful versus failure 1 h after NPPV and at support discontinuation. An unpaired *t*-test was used for the statistical comparison. Discontinuation of support refers to the last arterial blood gas obtained prior to intubation (failure) or prior to the definitive removal of NPPV in patients who avoided intubation

CLINICAL INVESTIGATIONS

Failure of Noninvasive Ventilation for De Novo Acute Hypoxemic Respiratory Failure

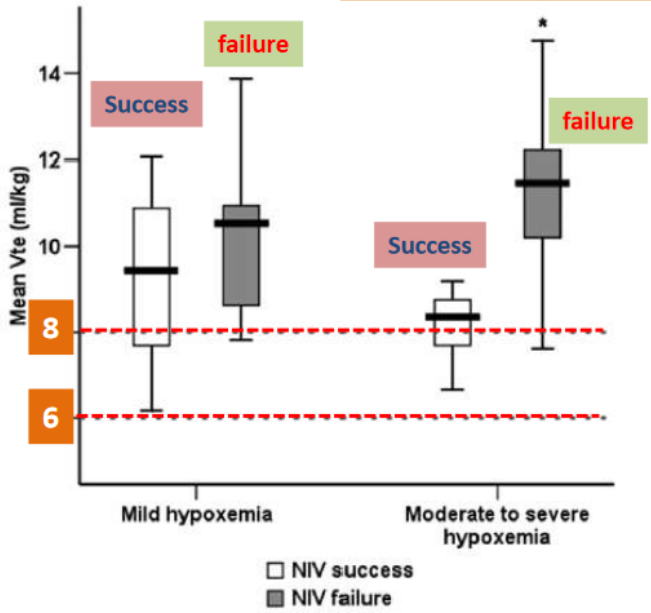
Role of Tidal Volume*

Carteaux, Guillaume MD[1,2,3]; Millán-Guilarte, Teresa MD[4]; De Prost, Nicolas MD, PhD[1,2,3]; Razazi, Keyvan MD[1,2,3]; Abid, Shariq MD, PhD[3]; Thille, A
PhD[1,3]; Brochard, Laurent MD[3,6,7]; Brun-Buisso
MD, PhD[1,2,3] Author Information

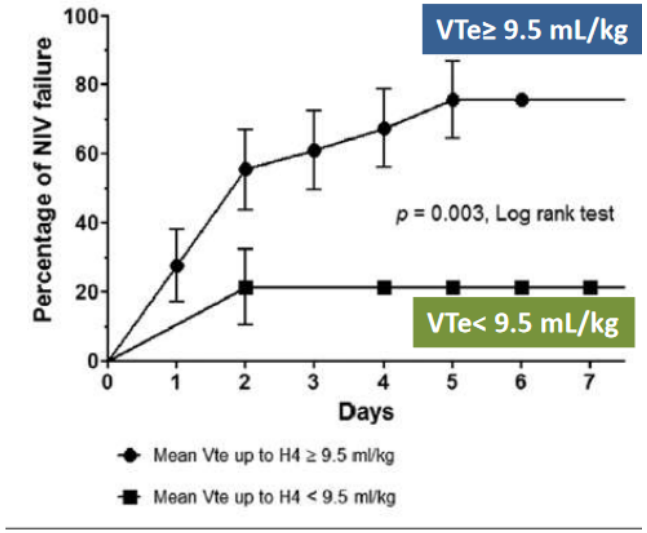
Critical Care Medicine: February 2016 - Volume

mild hypoxemia
200 < PaO2/FiO2 ≤ 300

Moderate to severe hypoxemia
PaO2/FiO2 ≤ 200 mm Hg



Moderate to severe hypoxemia
PaO2/FiO2 ≤ 200 mm Hg



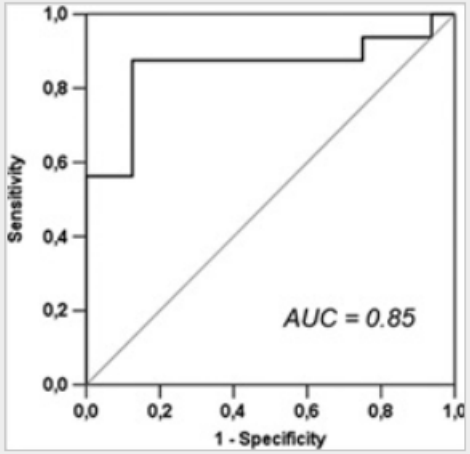


Figure 3: Receiver operating characteristic (ROC) curves of mean expired tidal volume (Vte) during noninvasive ventilation (NIV) to predict NIV failure in patients with moderate-to-severe hypoxemia (PaO_2/FIO_2 ratio ≤ 200 mm Hg; *n* = 33). AUC = area under the ROC curve.

In pts with $PaO_2/FIO_2 \leq 200$ mm Hg, Vte >9.5 mL/kg PBW predicted NIV failure (sensitivity 82% specificity of 87%).

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

Guillaume Carteaux, MD[1,2,3]; Teresa Millán-Guilarte, MD[4]; Nicolas De Prost, MD, PhD[1,2,3]; Keyvan Razazi, MD[1,2,3]; Shariq Abid, MD, PhD[3]; Arnaud W. Thille, MD, PhD[5]; 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]

Ventilatory and Hemodynamic Data	NIV Success ($n = 30$)	NIV Failure ($n = 32$)	p
Positive end-expiratory pressure, cm H_2O			
During NIV	5 (5–5)	5 (5–5)	0.50
After intubation	NA	10 (5–14)[b]	
Pressure support level, cm H_2O			
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)	
Vte, 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_2O	NA	27 (18–36)	

PIP 13 cmH2O

10.6 x 70 =742 ml

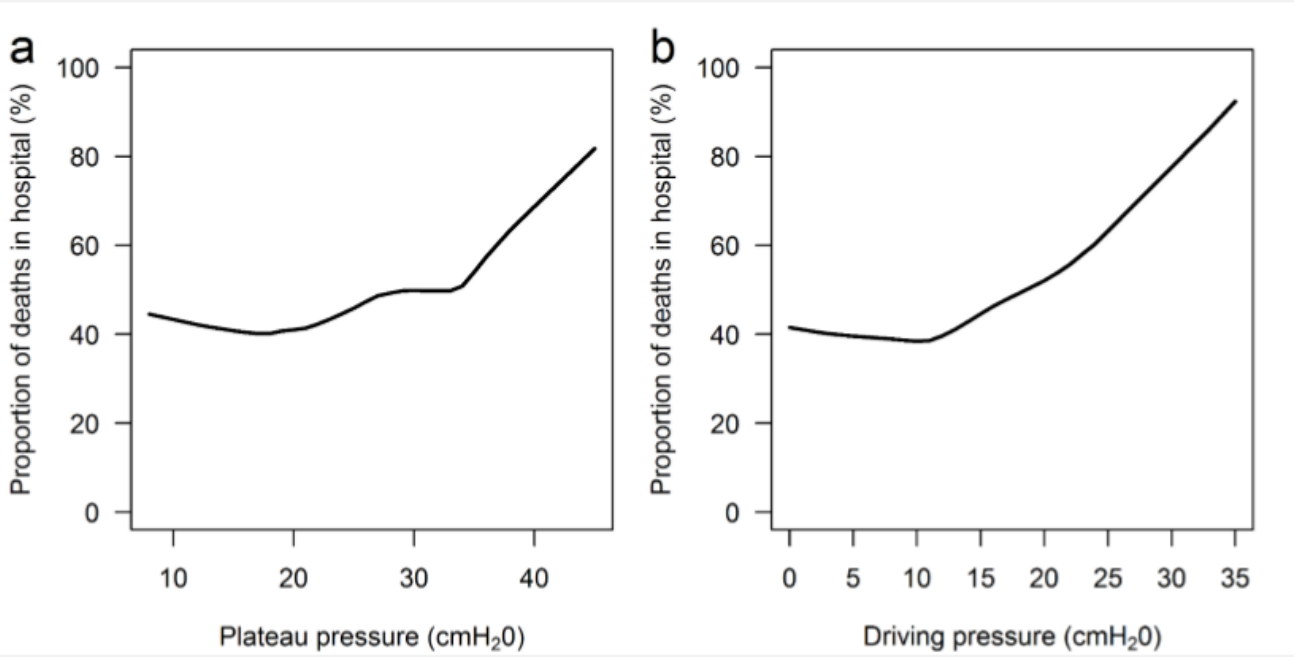
742/27 + PEEP= 32 cmH2O

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*], Giacomo Bellani[3,4], Tài Pham[5,6,7], Eddy Fan[8,9], Fabiana Madotto[10], Ednan K. Bajwa[11], Laurent Brochard[12,13], Kevin Clarkson[14], Andres Esteban[15], Luciano Gattinoni[16], Frank van Haren[17], Leo M. Heunks[18], Kiyoyasu Kurahashi[19], Jon Henrik Laake[20], Anders Larsson[21], Daniel F. McAuley[22], Lia McNamee[22], Nicolas Nin[15], Haibo Qiu[23], Marco Ranieri[24], Gordon D. Rubenfeld[25], B. Taylor Thompson[11], Hermann Wrigge[26], Arthur S. Slutsky[12,13,27], Antonio Pesenti[28,29] and The LUNG SAFE Investigators and the ESICM Trials Group



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AJRCCM Volume 202 Number 4 | August 15 2020

ORIGINAL ARTICLE

Early Inspiratory Effort Assessment by Esophageal Manometry Predicts Noninvasive Ventilation Outcome in *De Novo* Respiratory Failure

A Pilot Study

Roberto Tonelli[1,2], Riccardo Fantini[1], Luca Tabbì[1], Ivana Castaniere[1,2], Lara Pisani[3], Maria Rosaria Pellegrino[1], Giovanni Della Casa[4], Roberto D'Amico[5], Massimo Girardis[6], Stefano Nava[3], Enrico M. Clini[1], and Alessandro Marchioni[1]

[1]Respiratory Diseases Unit, Department of Medical and Surgical Sciences; [2]Clinical and Experimental Medicine Doctoral Program, [4]Radiology Unit and [5]Statistics Unit, Department of Diagnostics, Clinical and Public Health Medicine, and [6]Intensive Care Unit, Department of Surgical, Medical, Dental and Morphological Sciences related to Transplants Oncology and Regenerative Medicine, University Hospital of Modena, University of Modena and Reggio Emilia, Modena, Italy; and [3]Department of Specialistic, Diagnostic and Experimental Medicine, University of Bologna, Bologna, Italy

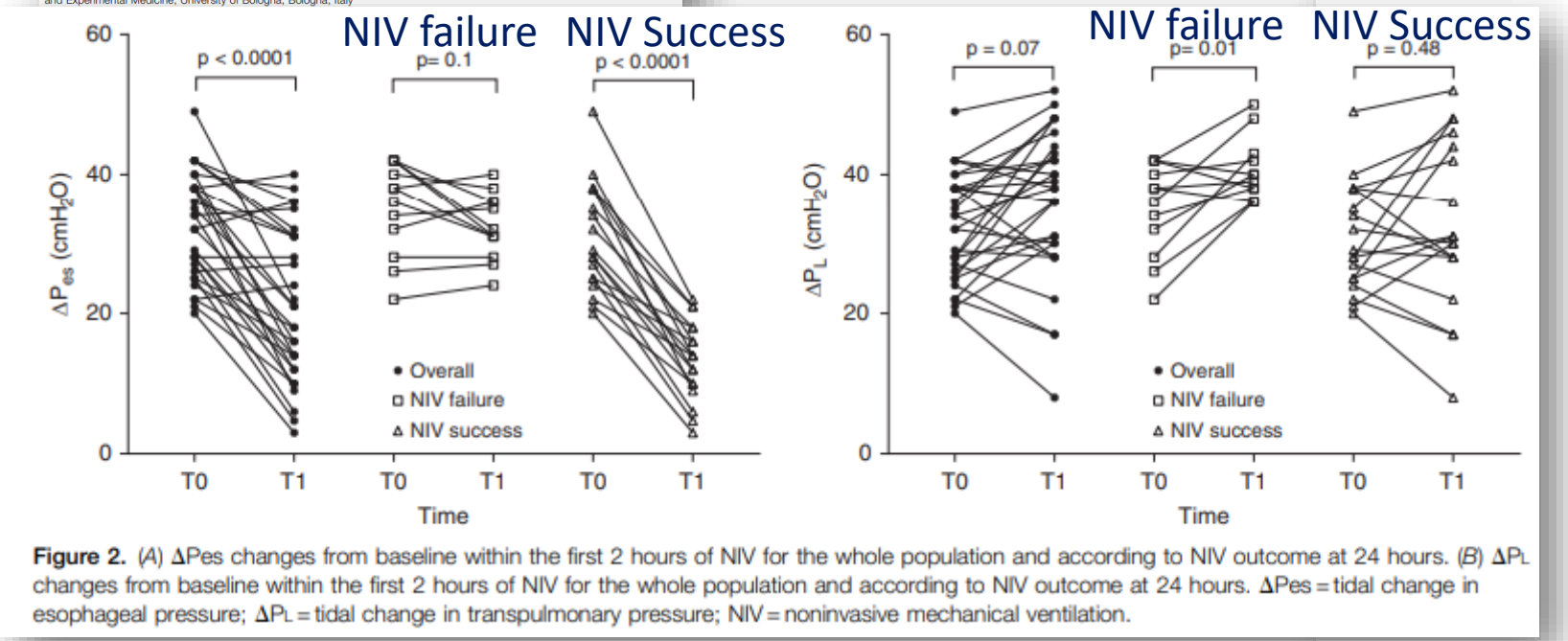


Figure 2. (*A*) ΔPes changes from baseline within the first 2 hours of NIV for the whole population and according to NIV outcome at 24 hours. (*B*) ΔPL changes from baseline within the first 2 hours of NIV for the whole population and according to NIV outcome at 24 hours. ΔPes = tidal change in esophageal pressure; ΔPL = tidal change in transpulmonary pressure; NIV = noninvasive mechanical ventilation.

Table 3. Association between Physiological and Clinical Variables and NIV Failure at 24 Hours

Feature	OR	95% CI	*P* Value
ΔPes < 10 cm H_2O post 2 h NIV	15	2.8–110	0.001
VTE > 9.5 ml/kg of PBW	7.9	1.5–72	0.02
HACOR score >5 post 2 h NIV	6.3	0.9–49	0.046
RR > 30 bpm	5.5	0.8–112	0.14
Pa_{O_2}/FI_{O_2} ratio < 150 mm Hg	2	0.5–9.8	0.4
VTE/ΔPL ratio < 0.33 ml/kg/cm H_2O	2	0.4–9.8	0.36

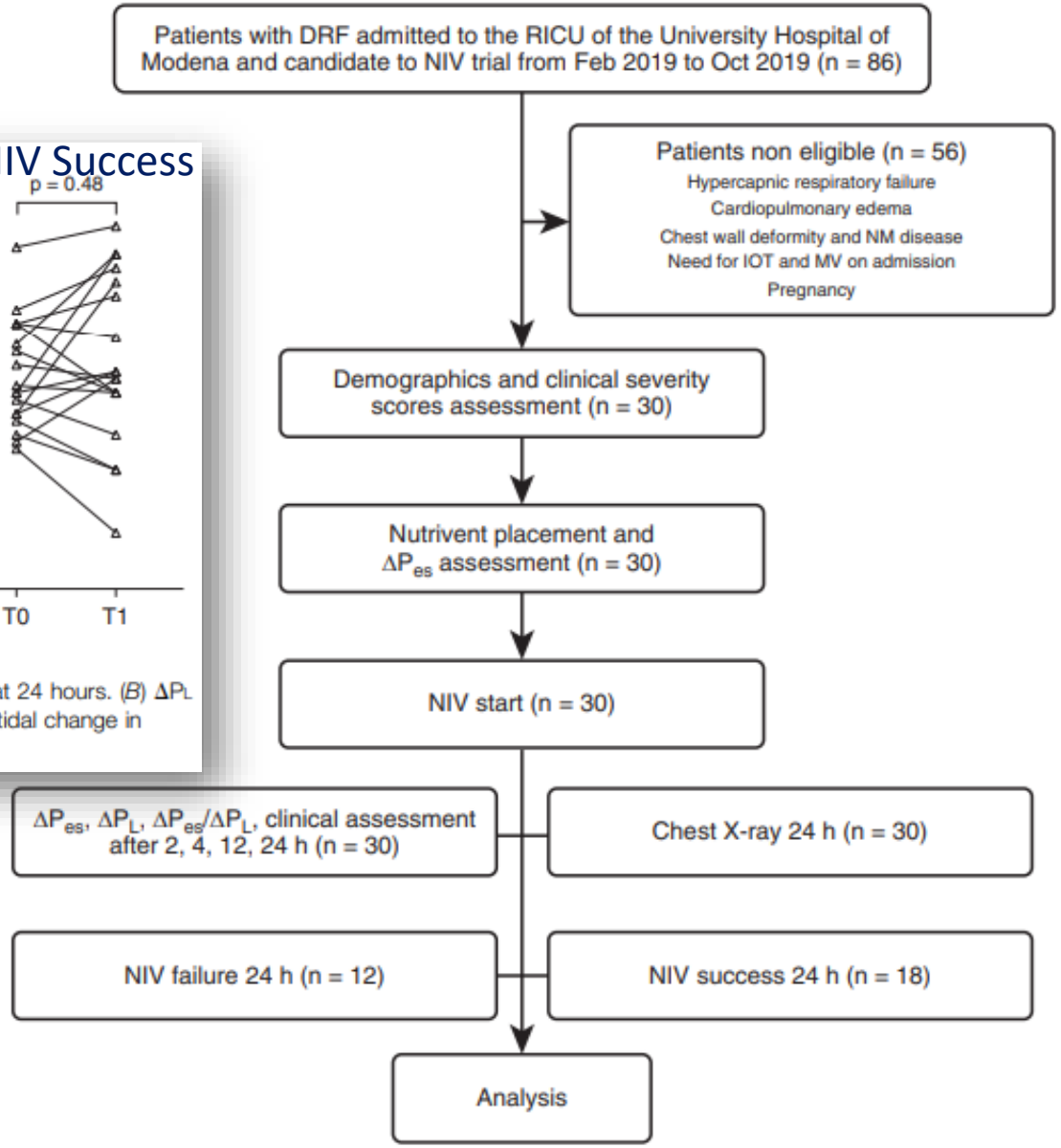


Figure 1. Flowchart for patients in this study. ΔPes = tidal change in esophageal pressure; ΔPL = tidal change in transpulmonary pressure; DRF = *de novo* respiratory failure; IOT = intubation (orotracheal); MV = mechanical ventilation; NIV = noninvasive MV; NM = neuromuscular; RICU = respiratory ICU.

NARRATIVE REVIEW

Non-invasive ventilatory support and high-flow nasal oxygen as first-line treatment of acute hypoxemic respiratory failure and ARDS

Domenico Luca Grieco[1,2*], Salvatore Maurizio Maggiore[3,4], Oriol Roca[5,6], Elena Spinelli[7], Bhakti K. Patel[8], Arnaud W. Thille[9,10], Carmen Silvia V. Barbas[11,12], Marina Garcia de Acilu[5,13], Salvatore Lucio Cutuli[1,2], Filippo Bongiovanni[1,2], Marcelo Amato[14], Jean-Pierre Frat[9,10], Tommaso Mauri[7,15], John P. Kress[7], Jordi Mancebo[16] and Massimo Ant[1,2]

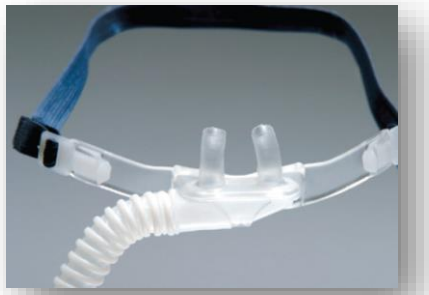
Paziente ipossiemico fallimento della NIV e valutazione SILI

Table 2 Relevant physiological measures for monitoring of hypoxemic patients on noninvasive respiratory support

Parameter	Monitoring technique/score calculation	Clinical thresholds associated with risk of failure	Limitations
SpO_2/FiO_2	Pulse oximetry	< 120 and/or worsening trend	Underestimation of severity with low $PaCO_2$
PaO_2/FiO_2	Arterial blood gas analysis	< 150–200 mmHg and/or worsening trend	Intermittent
Respiratory Rate	Clinical examination	> 25–30 and/or not decreasing with support	Poorly correlated with effort
Expired tidal volume	Ventilator	> 9–9.5 ml/kg PBW	Not feasible during HFNO, standard helmet NIV
ΔP_{ES}	Esophageal balloon catheter	> 15 cmH_2O and/or reduction < 10 cmH_2O during NIV	Needs some expertise
ROX	(SpO_2/FiO_2)/Respiratory Rate	< 2.85 at 2 h of HFNO initiation < 3.47 at 6 h of HFNO initiation < 3.85 at 12 h of HFNO initiation	Validated only for HFNO
HACOR scale[a]	Heart rate, acidosis, consciousness, oxygenation and respiratory rate[a]	> 5 at 1 h of NIV initiation	Intermittent, time consuming, validated only for NIV

PBW predicted body weight, *NIV* noninvasive ventilation, *HFNO* high-flow nasal oxygen, *DeltaPes* inspiratory effort

[a] The HACOR score is calculated as the sum of the scores for each individual variable, assigned as follows. Heart rate: ≤ 120 beats/min = 0, ≥ 121 beats/min = 1; pH: ≥ 7.35 = 0, 7.30–7.34 = 2, 7.25–7.29 = 3, < 7.25 = 4; Glasgow Coma Scale score: 15 = 0, 13–14 = 2, 11–12 = 5, ≤ 10 = 10; PaO_2/FiO_2 ratio: ≥ 201 mmHg = 0, 176–200 mmHg = 2, 151–175 mmHg = 3, 126–150 mmHg = 4, 101–125 mmHg = 5, ≤ 100 mmHg = 6; Respiratory rate: ≤ 30 breaths/min = 0, 31–35 breaths/min = 1, 36–40 breaths/min = 2, 41–45 breaths/min = 3, ≥ 46 = 4

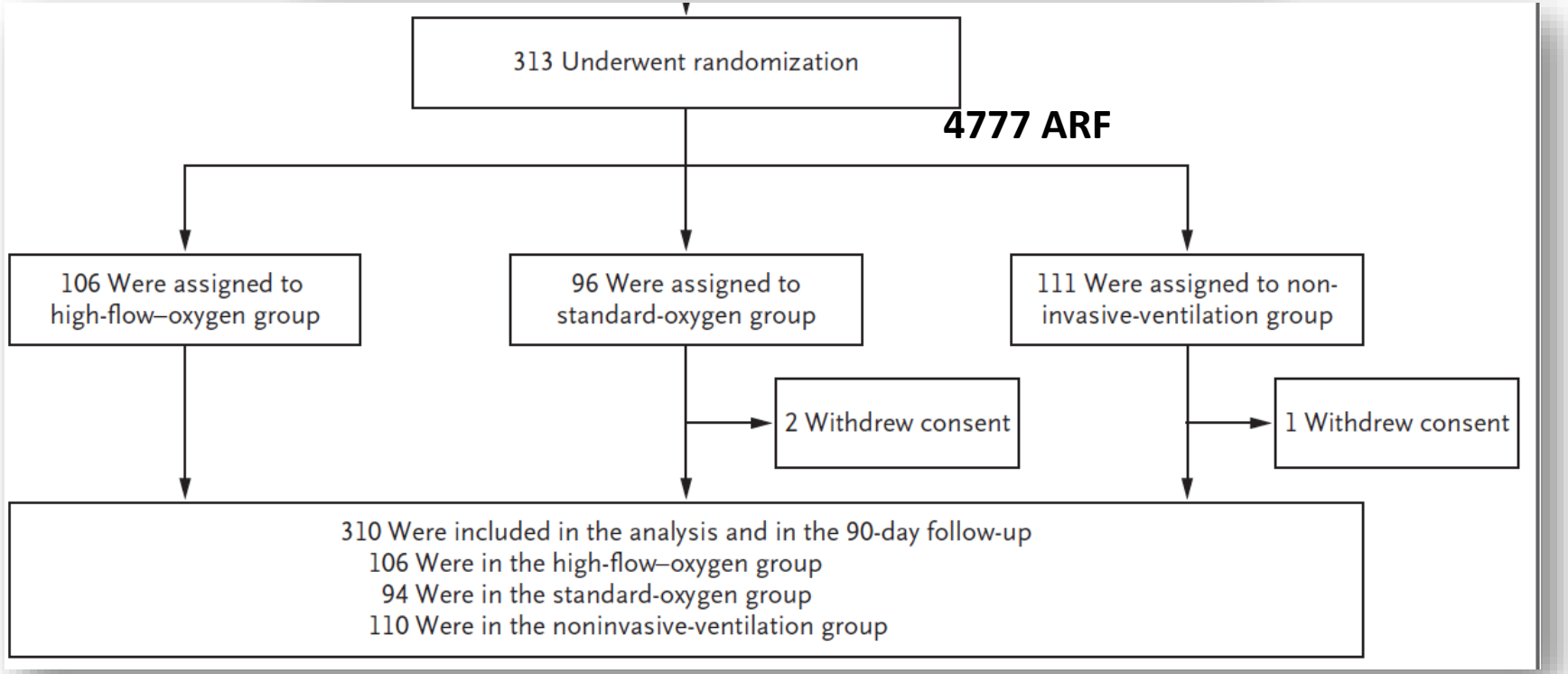


The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 JUNE 4, 2015 VOL. 372 NO. 23

High-Flow Oxygen through Nasal Cannula in Acute Hypoxemic Respiratory Failure

Jean-Pierre Frat, M.D., Arnaud W. Thille, M.D., Ph.D., Alain Mercat, M.D., Ph.D., Christophe Girault, M.D., Ph.D., Stéphanie Ragot, Pharm.D., Ph.D., Sébastien Perbet, M.D., Gwénael Prat, M.D., Thierry Boulain, M.D., Elise Morawiec, M.D., Alice Cottereau, M.D., Jérôme Devaquet, M.D., Saad Nseir, M.D., Ph.D., Keyvan Razazi, M.D., Jean-Paul Mira, M.D., Ph.D., Laurent Argaud, M.D., Ph.D., Jean-Charles Chakarian, M.D., Jean-Damien Ricard, M.D., Ph.D., Xavier Wittebole, M.D., Stéphanie Chevalier, M.D., Alexandre Herbland, M.D., Muriel Fartoukh, M.D., Ph.D., Jean-Michel Constantin, M.D., Ph.D., Jean-Marie Tonnelier, M.D., Marc Pierrot, M.D., Armelle Mathonnet, M.D., Gaëtan Béduneau, M.D., Céline Delétage-Métreau, Ph.D., Jean-Christophe M. Richard, M.D., Ph.D., Laurent Brochard, M.D., and René Robert, M.D., Ph.D., for the FLORALI Study Group and the REVA Network*



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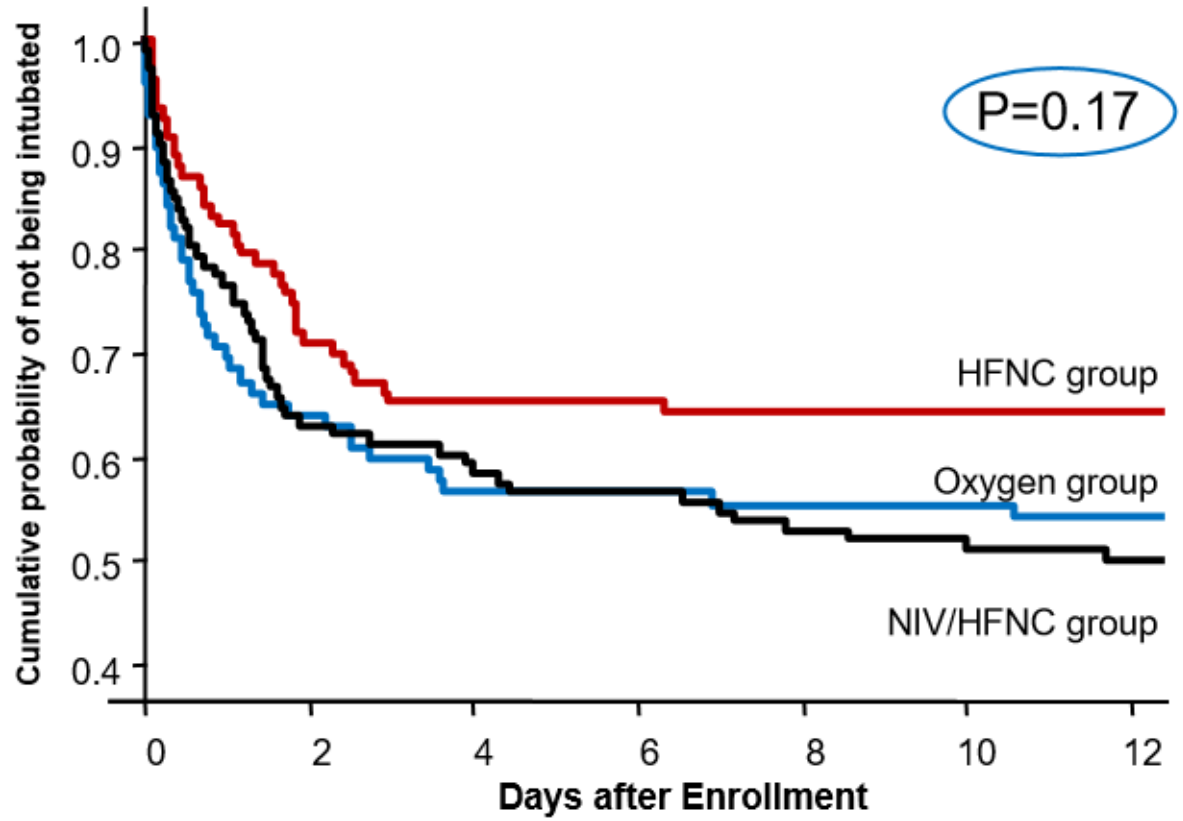
Primary outcome: Intubation rate

	Oxygen group (n=94)	HFNC group (n=106)	NIV/HFNC group (n=110)	P Value
Intubation – no. (%)	44 (46.8)	40 (37.7)	55 (50.0)	0.17
Interval between baseline and intubation – hours	15 [5-39]	27 [8-46]	27 [8-53]	0.27



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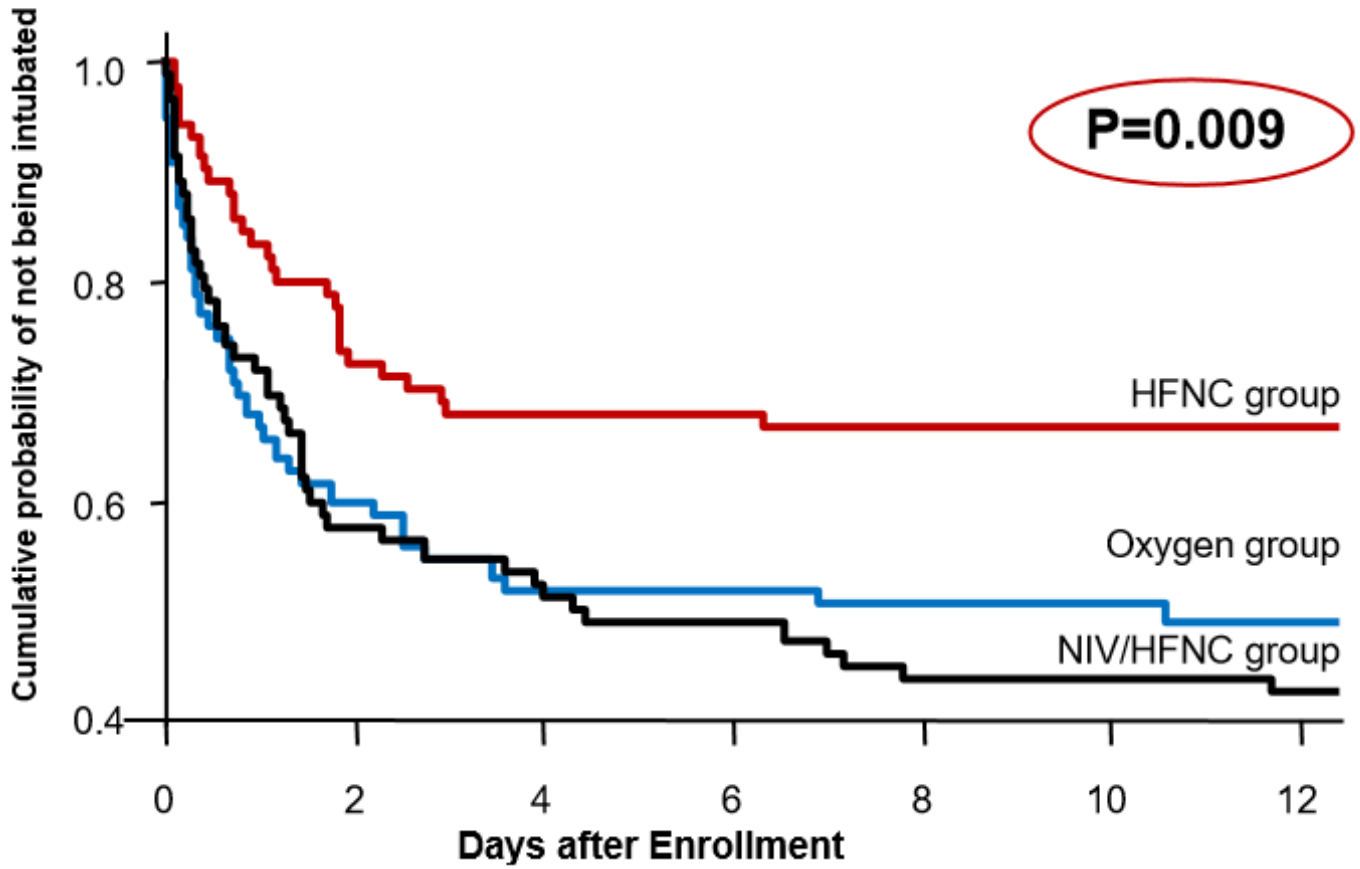
Intubation rate in patients with P/F ≤200 (n=238)

	Oxygen group (n=74)	HFNC group (n=83)	NIV/HFNC group (n=81)	P Value
Intubation – no. (%)	39 (52.7)	29 (34.9)	47 (58.0)	<0.01



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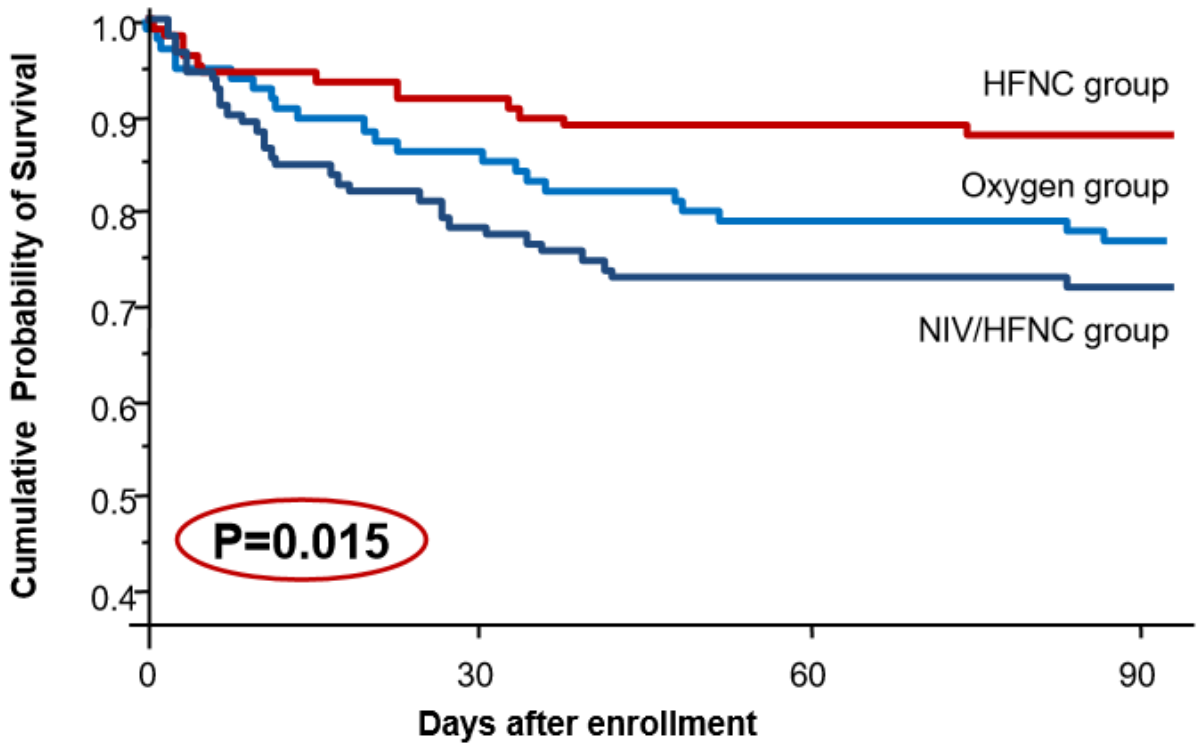
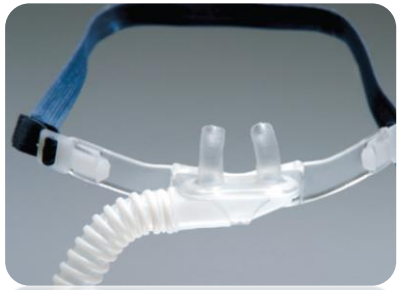
Secondary outcomes:
in-ICU mortality, day 90 mortality, Day 28 VFD

	Oxygen group (n=94)	HFNC group (n=106)	NIV/HFNC group (n=110)	P Value
ICU mortality – no. (%)	18 (19.1)	12 (11.3)	27 (24.5)	<0.05
Mortality at day 90– no. (%)	22 (23.4)	13 (12.3)	31 (28.2)	<0.05
Ventilator-free days at day 28 – day	22±10	24±8	19±12	<0.05

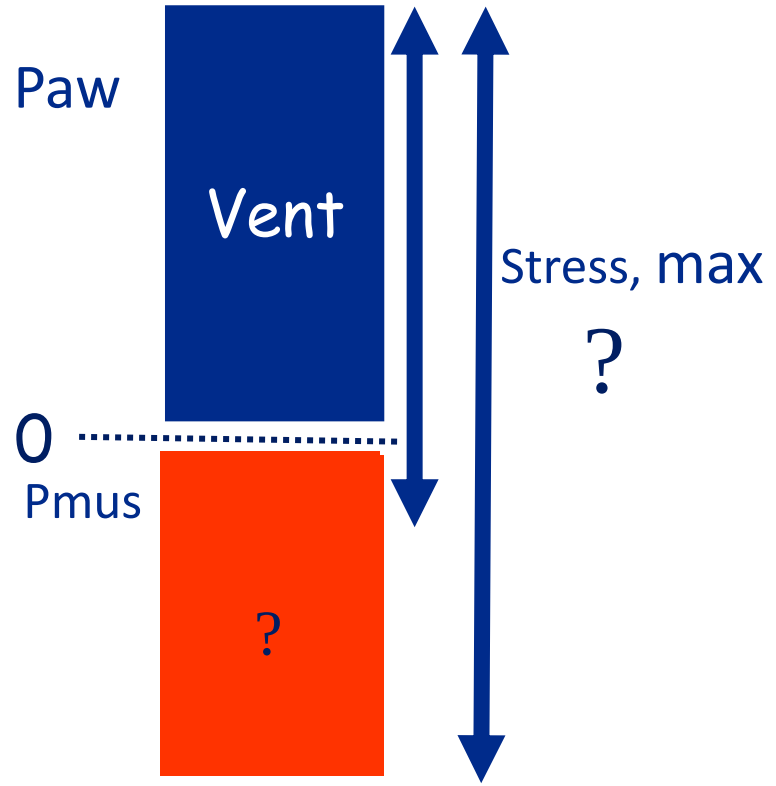




- NIV applied intermittently and without standardized protocol and balanced experience of the centres involved
- NIV Intermittence has unavoidably induced derecruitment.
- Cross over
- Difference in mortality due to higher no. of circulatory shock in the NIV group respect
- If this is true, why pts had a delay of intubation instead a prompt invasive mechanical ventilation?

NIV

Self inflicted
Lung Injury (SILI)



Respiratory non-invasive therapies for COVID-19: recommendations from scientific societies

Scientific society (country), ref nr	Non-invasive respiratory therapy first option
SEPAR (Spain)[56]	HFNC
AIPO (Italy)[57]	Helmet CPAP
ESICM/SCCM (EU/US)[58]	HFNC
SPP (Portugal)[51]	HFNC or CPAP
NHS (UK)[59]	CPAP
WHO[60]	HFNC or NIV
CTS (China)[61]	HFNC
ANZICS (Australia/New Zealand)[62]	HFNC
Multiple Societies (Germany)[63]	Helmet NIV

Abbreviations: SEPAR-Sociedad Española de Patologia Respiratória; AIPO-Associazone Italiana Pneumologi Ospedalieri; ESICM-European Society of Intensive Care Medicine; SCCM-Society of Critical Care Medicine SPP-Sociedade Portuguesa de Pneumologia; CTS-Chinese Thoracic Society, ANZICS-Australian and New Zealand Intensive Care Society.

Wink JC & Ambrosino N Pulmonol 2020;26(4):213-20

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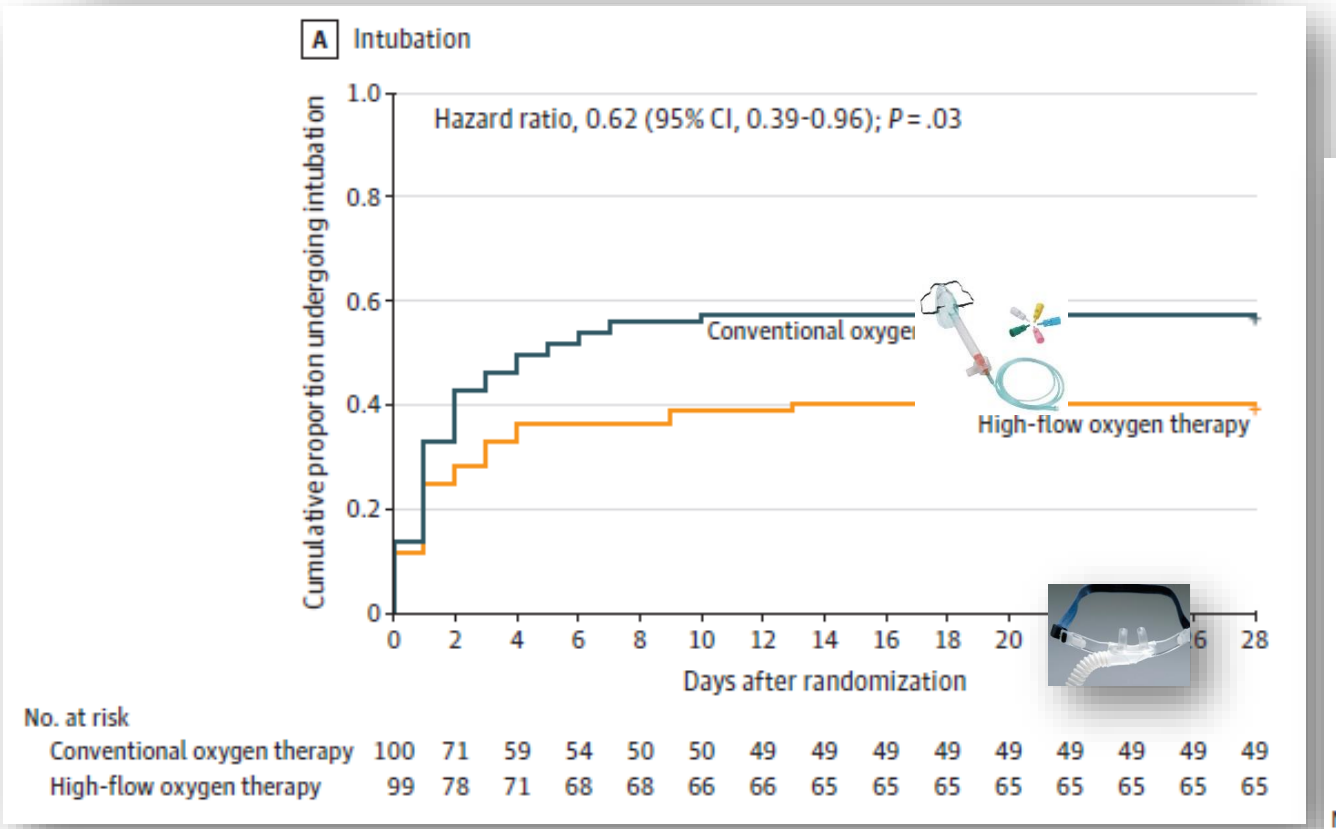
JAMA | Original Investigation

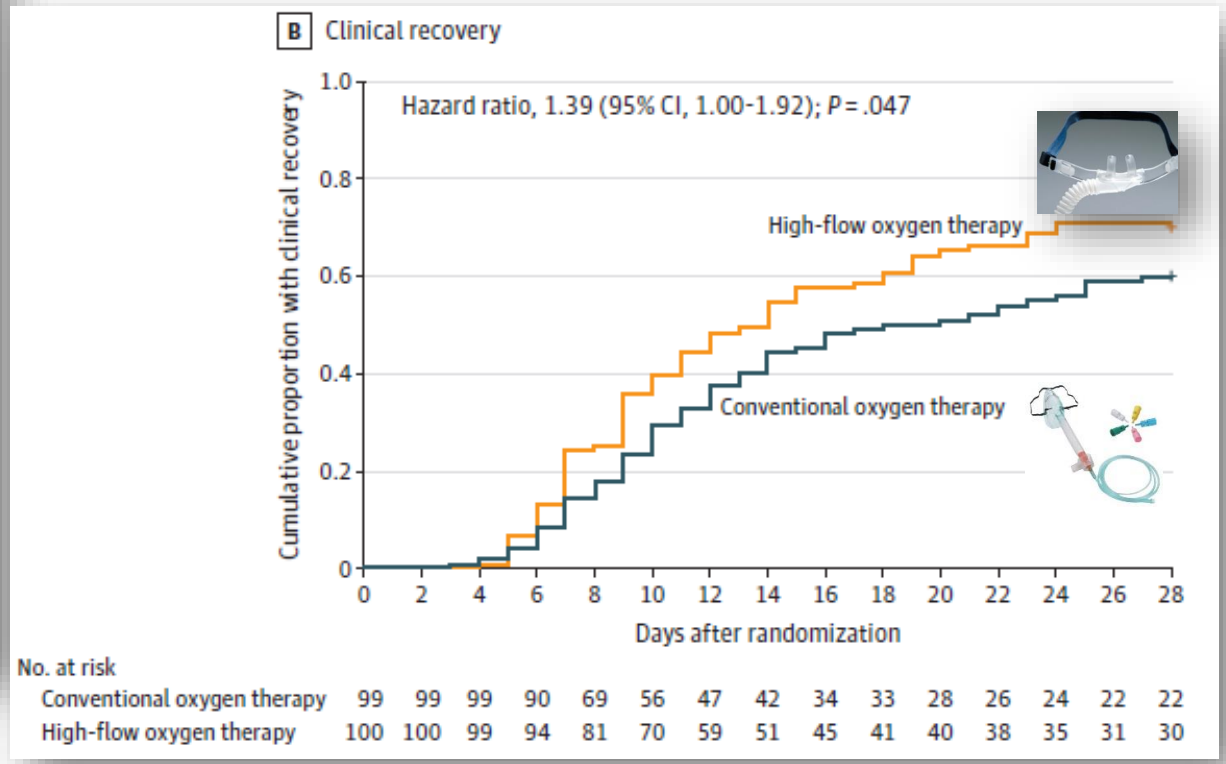
Effect of High-Flow Oxygen Therapy vs Conventional Oxygen Therapy on Invasive Mechanical Ventilation and Clinical Recovery in Patients With Severe COVID-19

A Randomized Clinical Trial

Gustavo A. Ospina-Tascón, MD, PhD; Luis Eduardo Calderón-Tapia, MD; Alberto F. García, MD, MSc; Virginia Zarama, MD; Freddy Gómez-Álvarez, MD; Tatiana Álvarez-Saa, MD; Stephania Pardo-Otálvaro, MD; Diego F. Bautista-Rincón, MD; Mónica P. Vargas, MD; José L. Aldana-Díaz, MD; Ángela Marulanda, MD; Alejandro Gutiérrez, MD; Janer Varón, MD; Mónica Gómez, MD; María E. Ochoa, MD; Elena Escobar, MD; Mauricio Umaña, MD; Julio Díez, MD; Gabriel J. Tobón, MD, PhD; Ludwig L. Albornoz, MD; Carlos Augusto Celemín Flórez, MD; Guillermo Ortiz Ruiz, MD, PhD; Eder Leonardo Cáceres, MD; Luis Felipe Reyes, MD, PhD; Lucas Petri Damiani, MSc; Alexandre B. Cavalcanti, MD, PhD; for the HiFLo-Covid Investigators

Among pts with severe COVID-19, use of high-flow oxygen through a nasal cannula significantly **decreased need for mechanical ventilation** support and **time to clinical recovery** compared with conventional low-flow oxygen therapy.





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The Journal of Emergency Medicine, Vol. 61, No. ■, pp. 37–40, 2021

0736-4679/$ - see front matter

https://doi.org/10.1016/j.jemermed.2021.03.006

Selected Topics: Prehospital Care

Logistical Challenge With Prehospital Use of High-Flow Nasal Oxygen Therapy in COVID-19-Induced Respiratory Distress: A Case Report

Romain Kedzierewicz, MD, Clément Derkenne, MD, Adrien Fraudin, MD, Paola Vanhaecke, MD, Romain Jouffroy, MD, Daniel Jost, MD, and Bertrand Prunet, PDH

Emergency Medical Service, Paris Fire Brigade, Paris, France

Reprint Address: Romain Kedzierewicz, MD, Emergency Medical Service, Paris Fire Brigade, 1 Place Jules Renard, 75017 Paris, France

Figure 1. High-flow nasal oxygen therapy device being used in a resuscitation ambulance.

HFOT could be used to improve oxygenation, if conventional O2 failed and there is no urgent indication for intubation or as a surrogate while waiting for intubation

Batteria

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O2 ALTA PRESSIONE

Feasibility and clinical impact of out-of-ICU noninvasive respiratory support in patients with COVID-19-related pneumonia

Cosimo Franco[1,16], Nicola Facciolongo[2,16], Roberto Tonelli[3,4], Roberto Dongilli[5], Andrea Vianello[6], Lara Pisani[7], Raffaele Scala[8], Mario Malerba[9], Annalisa Carlucci[10], Emanuele Alberto Negri[2], Greta Spoladore[11], Giovanna Arcaro[6], Paolo Amedeo Tillio[9], Cinzia Lastoria[12], Gioachino Schifino[7], Luca Tabbì[4], Luca Guidelli[8], Giovanni Guaraldi[13], V. Marco Ranieri[14], Enrico Clini[4,17] and Stefano Nava[15,17]

Observational study, data from 670 pts with confirmed Sars CoV 2 disease referred to pulmonology units in 9 hospitals

TABLE 3 Clinical outcomes and relative probability for the whole population and according to ventilatory support

	Total	HFNC	OR (95% CI)	p-value	CPAP	OR (95% CI)	p-value	NIV	OR (95%CI)	p-value
Patients	670 (100)	163 (24.3)			330 (49.3)			177 (26.4)		
30-day mortality										
Crude	180 (26.9)	26 (15.9)	0.43 (0.3–0.7)	<0.01	100 (30.3)	1.4 (0.9–2)	0.05	54 (30.5)	1.3 (0.5–1.9)	0.20
Adjusted#			0.52 (0.2–1.2)	0.10		1.7 (0.8–4.3)	0.11		1.1 (0.3–3.7)	0.88
ETI										
Crude	178 (26.6)	47 (28.8)	1.1 (0.8–1.7)	0.45	82 (24.8)	0.8 (0.6–1.2)	0.32	49 (27.7)	1.1 (0.7–1.6)	0.80
Adjusted#			1.5 (0.6–4.1)	0.39		0.9 (0.5–1.7)	0.76		1.2 (0.5–3.3)	0.65
30-day mortality/ETI										
Crude	312 (46.5)	62 (38)	0.6 (0.4–0.9)	0.01	156 (47.3)	1.06 (0.8–1.4)	0.7	94 (53)	1.4 (1.2)	0.04
Adjusted#			0.89 (0.4–2.1)	0.79		0.9 (0.5–1.7)	0.76		1.1 (0.5–2.7)	0.78
Length of hospital stay days	20.3±13.2	19.2±13.3	0.91 (0.4–1.13)	0.87	19.8±12.1	0.95 (0.5–1.14)	0.82	21.5±15.1	1.2 (0.6–1.5)	0.47

Data are presented as n (%) or mean±SD, unless otherwise stated. Clinical outcomes and relative probability from fitting a logistic regression model for the whole study population and according to ventilatory support. Statistical significance was set at $p<0.05$. HFNC: high-flow nasal cannula; CPAP: continuous positive airway pressure; NIV: noninvasive mechanical ventilation; ETI: endotracheal intubation. #: adjusted for age, baseline arterial oxygen tension/inspiratory oxygen fraction ratio, number of comorbidities and steroid usage.

The 3 modes of NRS had similar impact on

- **Mortality**
- **ETI rate**
- **Hospital LOS**

TABLE 2 Fraction of active professional healthcare workers and percentage of infection

	At work	Infected
Physician	108	8 (7.4)
Nurse	210	29 (13.8)
Healthcare assistant	45	5 (11)
Physiotherapist	16	0 (0)
Total	379	42 (11.1)

Data are presented as n or n (%).

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Massive numbers of clinicians have been infected during the COVID-19 outbreak, which has raised **concerns around implementing aerosol-generating procedures**. Consequently, there appears to be a trend to avoid HFNC.

The scientific evidence of generation and dispersion of bio-aerosols via HFNC summarised here shows a similar risk to standard oxygen masks

Oxygen device	Flow rate L·min^{-1}	Dispersion distance cm
HFNC	60	17.2±3.3
	30	13.0±1.1
	10	6.5±1.5
Simple mask	15	11.2±0.7
	10	9.5±0.6
Non-rebreathing mask	10	24.6±2.2
Venturi mask at F_{IO_2} 0.4	6	39.7±1.6
Venturi mask at F_{IO_2} 0.35	6	27.2±1.1

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Li J et al , ERJ 2020; 55: 20008982

Preliminary Findings on Control of Dispersion of Aerosols and Droplets During High-Velocity Nasal Insufflation Therapy Using a Simple Surgical Mask

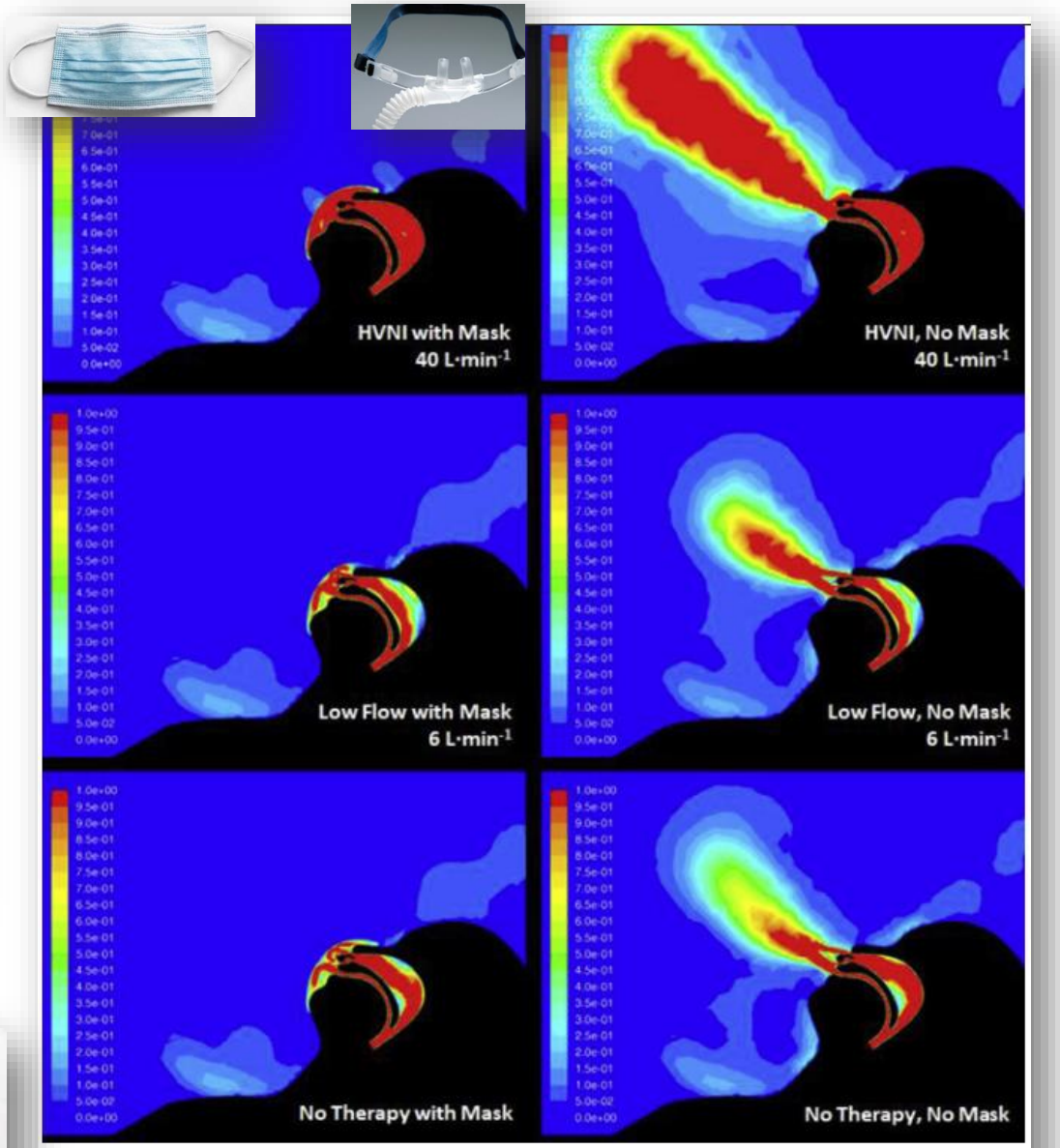
Implications for the High-Flow Nasal Cannula

Check for updates

To the Editor:

Coronavirus disease 2019 (COVID-19) pneumonia presents with severe hypoxemic respiratory failure, caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The primary mode of transmission appears to be droplet-borne. Respiratory support and high levels of oxygen are required in the acute treatment of these patients. High-flow therapies have been included as part of the possible management of COVID-19.[1,2] One such modality is high-flow therapy, including high-velocity nasal insufflation (HVNI), high-flow nasal oxygen (HFNO), and high-flow nasal cannulation (HFNC). HVNI shares characteristics with HFNC/HFNO, in that all reliably deliver high flows of oxygen-enriched gas at high F_{IO_2} to the patient via an open nasal interface. High-flow therapy has demonstrated the ability to help manage hypoxic or type I respiratory failure.[3,4] All high-flow therapies also share the same issue of potential aerosol generation.

Recent correspondence has raised questions about health-care worker safety during the use of noninvasive ventilation and HFNC therapies.[5] HFNC has been studied and found to have limited particle dispersion when the cannula is properly fitted.[6] A recent recommendation has advised the use of a surgical mask over the face of the patient while wearing the high-flow therapy device to help reduce inadvertent aerosol.[7] This is the initial report of a study using computational fluid dynamic (CFD) simulation to determine the ability of a mask to reduce the velocity of exhaled gas flow and capture particles during HVNI.



Download : Download high-res image (714KB)

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Figure 1. Velocity map of gas flow for all tested settings. HVNI= high-velocity nasal insufflation

The addition of a simple type I surgical mask may provide an effective tool to further reduce droplet deposition due to exhaled gas flow, except at mask leaks

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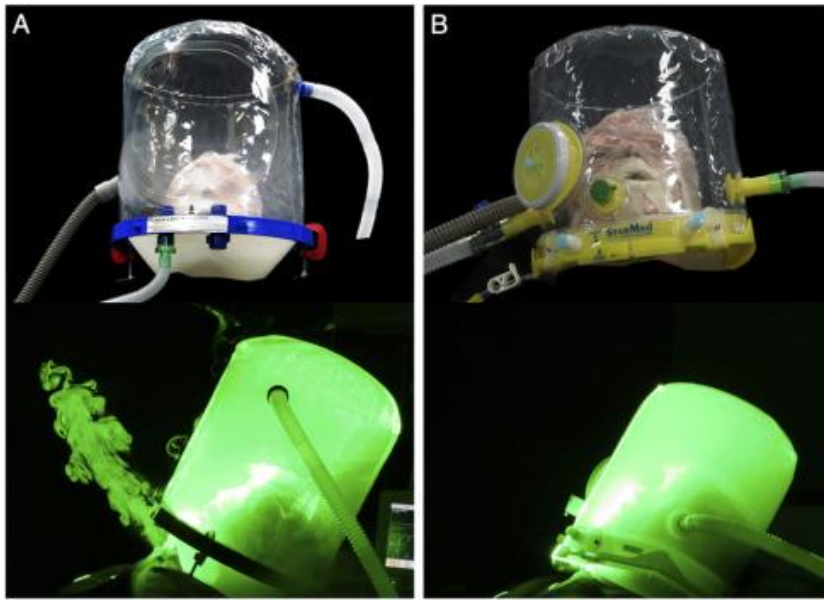
[Original Research **Critical Care**]

CHEST

Exhaled Air Dispersion During Noninvasive Ventilation via Helmets and a Total Facemask

David S. Hui, MD, FCCP; Benny K. Chow, PhD; Thomas Lo, MSc; Susanna S. Ng, MBChB; Fanny W. Ko, MD, FCCP; Tony Gin, MD; and Matthew T. V. Chan, MD

CHEST2015; 147(5): 1336 - 1343



Method	Maximum exhaled air dispersion distance
Oxygen *via* nasal cannula 5 L·min^{-1}	100 cm
Oxygen *via* oronasal mask 4 L·min^{-1}	40 cm
Oxygen *via* Venturi mask F_{IO_2} 40%	33 cm
Oxygen *via* non-rebreathing mask 12 L·min^{-1}	<10 cm
CPAP *via* oronasal mask 20 cmH_2O	Negligible air dispersion
CPAP *via* nasal pillows	33 cm
HFNC 60 L·min^{-1}	17 cm (62 cm sideways leakage if not tightly fixed)
NIV *via* full face mask: IPAP 18 cmH_2O, EPAP 5 cmH_2O	92 cm
NIV *via* helmet without tight air cushion: IPAP 20 cmH_2O, EPAP 10 cmH_2O	27 cm
NIV *via* helmet with tight air cushion: IPAP 20 cmH_2O, EPAP 10 cmH_2O	Negligible air dispersion

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Ferioli M et al Eur Respir Rev 2020; 29:200068

Effect of Noninvasive Ventilation Delivered by Helmet vs Face Mask on the Rate of Endotracheal Intubation in Patients With Acute Respiratory Distress Syndrome A Randomized Clinical Trial

Bhakti K. Patel, MD; Krysta S.Wolfe, MD; Anne S. Pohlman, MSN; Jesse B. Hall,

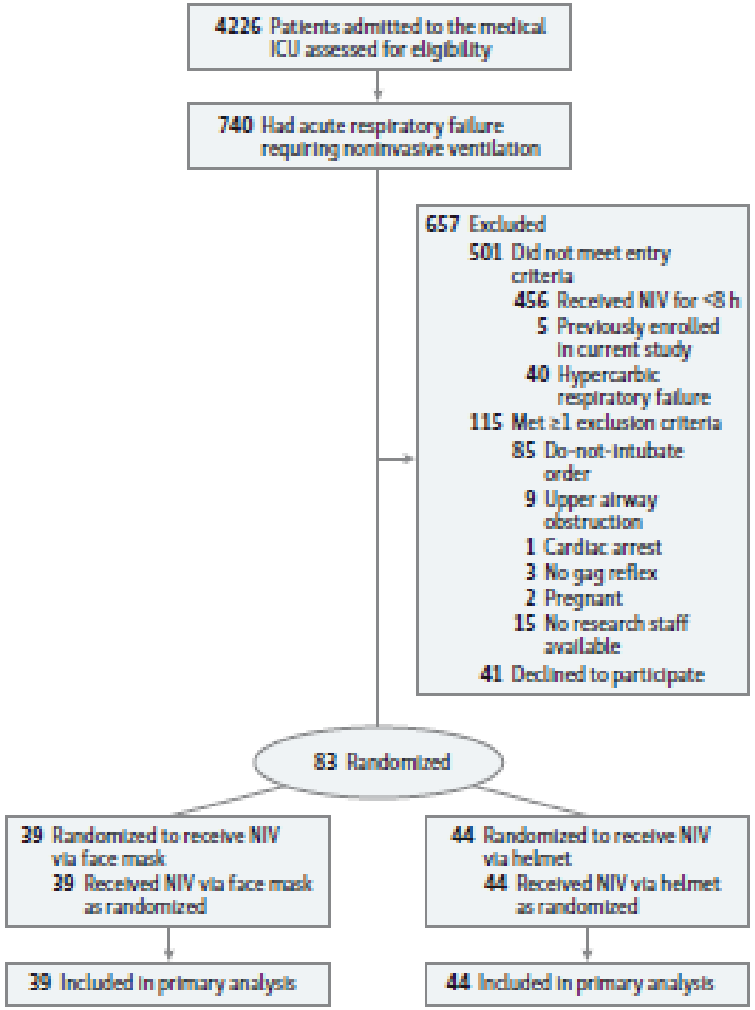
Single center RCT on 83 ARDS pts

Table 3. Level of Respiratory Support and Physiologic Parameters During Noninvasive Ventilation

	Noninvasive Ventilation, Median (IQR)		
	Face Mask (n = 39)	Helmet (n = 44)	*P* Value
Respiratory support with NIV[a]			
Duration of NIV, h	26.4 (7.0-60.0)	19.8 (8.4-45.6)	.68
PEEP, cm H_2O	5.1 (5.0-8.0)	8 (5.0-10.0)	.006
Pressure support, cm H_2O	11.2 (10.0-14.5)	8 (5.6-10.0)	<.001
FIO_2, %	60 (50.0-68.6)	50 (40.0-60.0)	.02
SpO_2, %	95.3 (92.3-96.7)	96.2 (94.8-98.4)	.13
Respiratory rate, breaths/min			
Baseline	28.3 (22.1-34.4)[b]	27.7 (21.5-34.6)[b]	
After randomization	29.1 (22.1-37.6)	24.5 (20.4-30.5)	

JAMA June 14, 2016 Volume 315, Number 22

Figure 1. Flow of Participants Through Study



ICU indicates intensive care unit; NIV, noninvasive ventilation.

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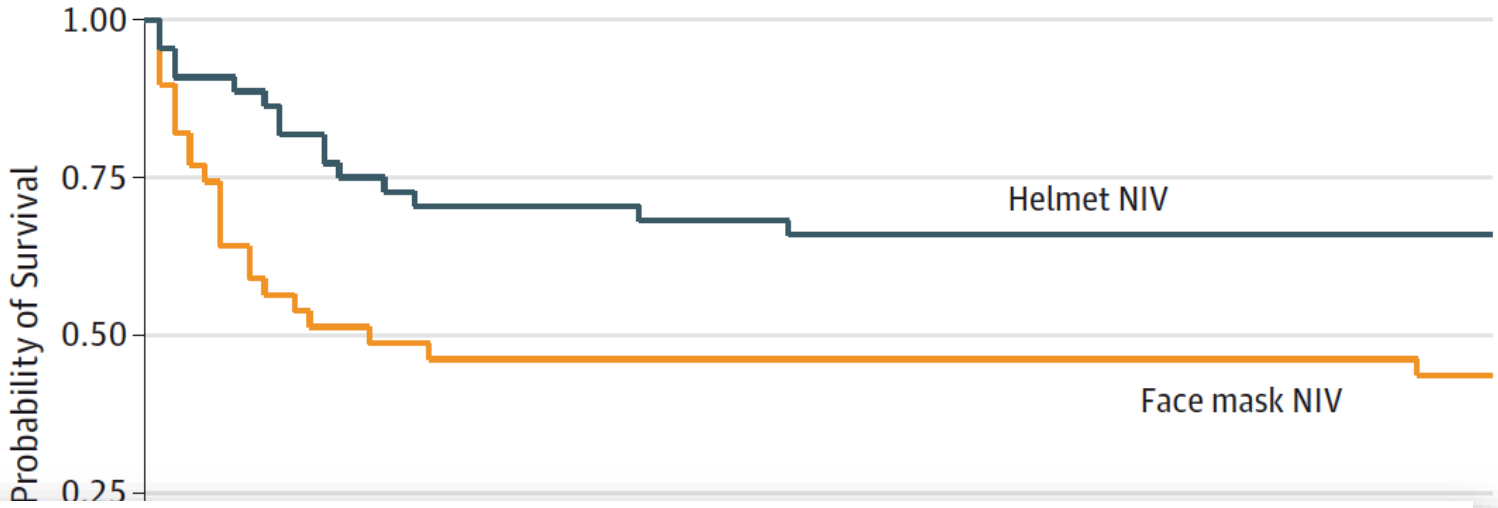
Table 2. Primary and Secondary Outcomes and Adverse Events

	Absolute

Figure 2. Probability of Survival From Randomization to Day 90



For patients with ARDS, treatment with helmet NIV was associated with a significant reduction of intubation rates compared with delivery by face mask. There was also a statistically significant reduction in 90-day mortality with helmet NIV.

Face mask	39	20	18	18	18	18	17
Helmet	44	33	31	29	29	29	29

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	Continuous positive airway pressure (n = 380)	High-flow nasal oxygen (n = 418)	Conventional oxygen therapy (n = 475)
Initial intervention details and prone positioning			
Continuous positive airway pressure			
Positive end-expiratory pressure, mean (95% CI), cm H_2O	(n = 304) 8.3 (8.1-8.5)	NA	NA
Delivery device, No. (%)		NA	NA
Noninvasive ventilation[a]	147 (38.7)	NA	NA
Continuous positive airway pressure	173 (45.5)	NA	NA
Other[b]	24 (6.3)	NA	NA

POPULATION

844 Men
429 Women

Adults with COVID-19-related acute hypoxemic respiratory failure

Mean age: 57 years

LOCATIONS

48 Acute care hospitals in the UK and Jersey

INTERVENTION

1273 Patients randomized
1260 Patients analyzed

380
CPAP
Administered per local protocols

418
HFNO
Administered per local protocols

475
Conventional oxygen therapy
Standard of care; oxygen via nasal cannula

PRIMARY OUTCOME

A composite of tracheal intubation or mortality within 30 days

FINDINGS

Tracheal intubation or mortality within 30 days

CPAP: 36.3% (137 of 377 patients)

HFNO: 44.3% (184 of 415 patients)

Conventional oxygen therapy
vs CPAP: **44.4%** (158 of 356 patients)
vs HFNO: **45.1%** (166 of 368 patients)

CPAP vs conventional therapy was significant.
Absolute difference, −8% (95% CI, −15% to −1%)

HFNO vs conventional therapy was not significant.
Absolute difference, −1% (95% CI, −8% to 6%)

Perkins GD, Ji C, Connolly BA, et al; RECOVERY-RS Collaborators. Effect of noninvasive respiratory strategies on intubation or mortality among patients with acute hypoxemic respiratory failure and COVID-19: the RECOVERY-RS randomized clinical trial. *JAMA*. Published January 24, 2022. doi:10.1001/jama.2022.0028

JAMA | Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

Association of Noninvasive Oxygenation Strategies With All-Cause Mortality in Adults With Acute Hypoxemic Respiratory Failure

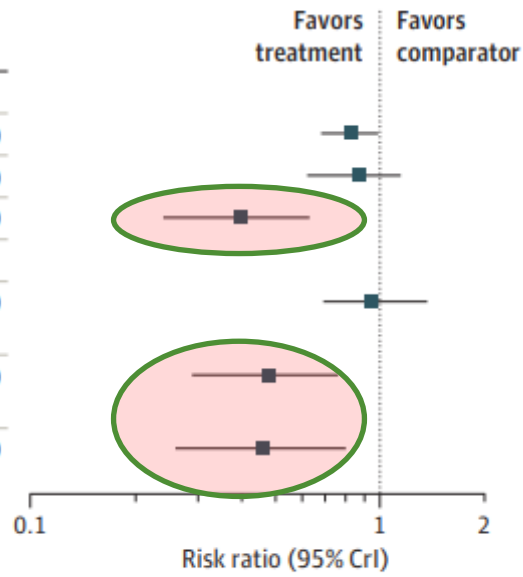
A Systematic Review and Meta-analysis

Bruno L. Ferreyro, MD; Federico Angriman, MD, MPH; Laveena Munshi, MD, MSc; Lorenzo Del Sorbo, MD; Niall D. Ferguson, MD, MSc; Bram Rochwerg, MD, MSc; Michelle J. Ryu, MLIS; Refik Saskin, MSc; Hannah Wunsch, MD, MSc; Bruno R. da Costa, MSc, PhD; Damon C. Scales, MD, PhD

Figure 3. Forest Plots for the Association of Noninvasive Oxygenation Strategies With Study Outcomes

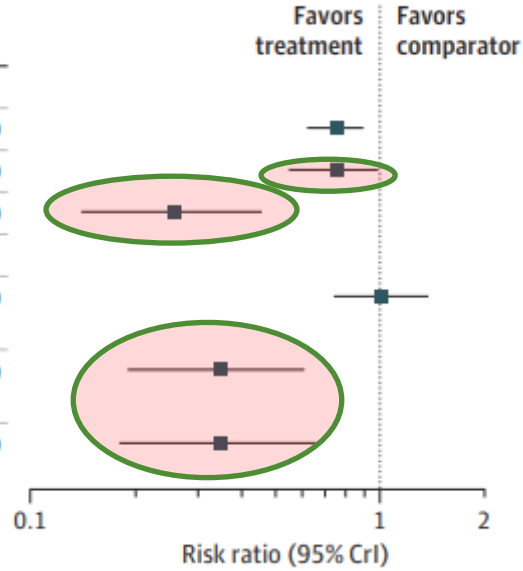
A All-cause mortality

	No. of patients	No. of trials	Quality	Absolute risk difference (95% CrI)	Network risk ratio (95% CrI)
Compared with standard oxygen					
Face mask noninvasive ventilation	1725	14	Moderate	-0.06 (-0.15 to -0.01)	0.83 (0.68-0.99)
High-flow nasal oxygen	1279	3	Moderate	-0.04 (-0.15 to 0.04)	0.87 (0.62-1.15)
Helmet noninvasive ventilation	330	3	Low	-0.19 (-0.37 to -0.09)	0.40 (0.24-0.63)
Additional comparisons					
Face mask noninvasive ventilation vs high-flow nasal oxygen	216	1	Low	-0.02 (-0.14 to 0.07)	0.95 (0.69-1.37)
Helmet noninvasive ventilation vs face mask noninvasive ventilation	83	1	Low	-0.13 (-0.27 to -0.05)	0.48 (0.29-0.76)
Helmet noninvasive ventilation vs high-flow nasal oxygen	0	0	Low	-0.15 (-0.34 to -0.05)	0.46 (0.26-0.80)



B Intubation

	No. of patients	No. of trials	Quality	Absolute risk difference (95% CrI)	Network risk ratio (95% CrI)
Compared with standard oxygen					
Face mask noninvasive ventilation	1725	14	Moderate	-0.12 (-0.25 to -0.05)	0.76 (0.62-0.90)
High-flow nasal oxygen	1479	5	Moderate	-0.11 (-0.27 to -0.01)	0.76 (0.55-0.99)
Helmet noninvasive ventilation	330	3	Low	-0.32 (-0.60 to -0.16)	0.26 (0.14-0.46)
Additional comparisons					
Face mask noninvasive ventilation vs high-flow nasal oxygen	450	3	Low	0.00 (-0.13 to 0.10)	1.01 (0.74-1.38)
Helmet noninvasive ventilation vs face mask noninvasive ventilation	83	1	Low	-0.20 (-0.40 to -0.09)	0.35 (0.19-0.61)
Helmet noninvasive ventilation vs high-flow nasal oxygen	0	0	Low	-0.20 (-0.43 to -0.08)	0.35 (0.18-0.66)



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Research

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Effect of Helmet Noninvasive Ventilation vs High-Flow Nasal Oxygen on Days Free of Respiratory Support in Patients With COVID-19 and Moderate to Severe Hypoxemic Respiratory Failure

The HENIVOT Randomized Clinical Trial

Domenico Luca Grieco, MD; Luca S. Menga, MD; Melania Cesarano, MD; Tommaso Rosà, MD; Savino Spadaro, MD, PhD; Maria Maddalena Bitondo, MD; Jonathan Montomoli, MD, PhD; Giulia Falò, MD; Tommaso Tonetti, MD; Salvatore L. Cutuli, MD; Gabriele Pintaudi, MD; Eloisa S. Tanzarella, MD; Edoardo Piervincenzi, MD; Filippo Bongiovanni, MD; Antonio M. Dell'Anna, MD; Luca Delle Cese, MD; Cecilia Berardi, MD; Simone Carelli, MD; Maria Grazia Bocci, MD; Luca Montini, MD; Giuseppe Bello, MD; Daniele Natalini, MD; Gennaro De Pascale, MD; Matteo Velardo, PhD; Carlo Alberto Volta, MD; V. Marco Ranieri, MD; Giorgio Conti, MD; Salvatore Maurizio Maggiore, MD, PhD; Massimo Antonelli, MD; for the COVID-ICU Gemelli Study Group

POPULATION

88 Men
21 Women

Adults in the intensive care unit with COVID-19 and moderate to severe hypoxemic respiratory failure

Median age: 65 years

LOCATIONS

4
ICUs in Italy

PaO2/FiO2≤200 entro 24 ore dall'ammissione

INTERVENTION

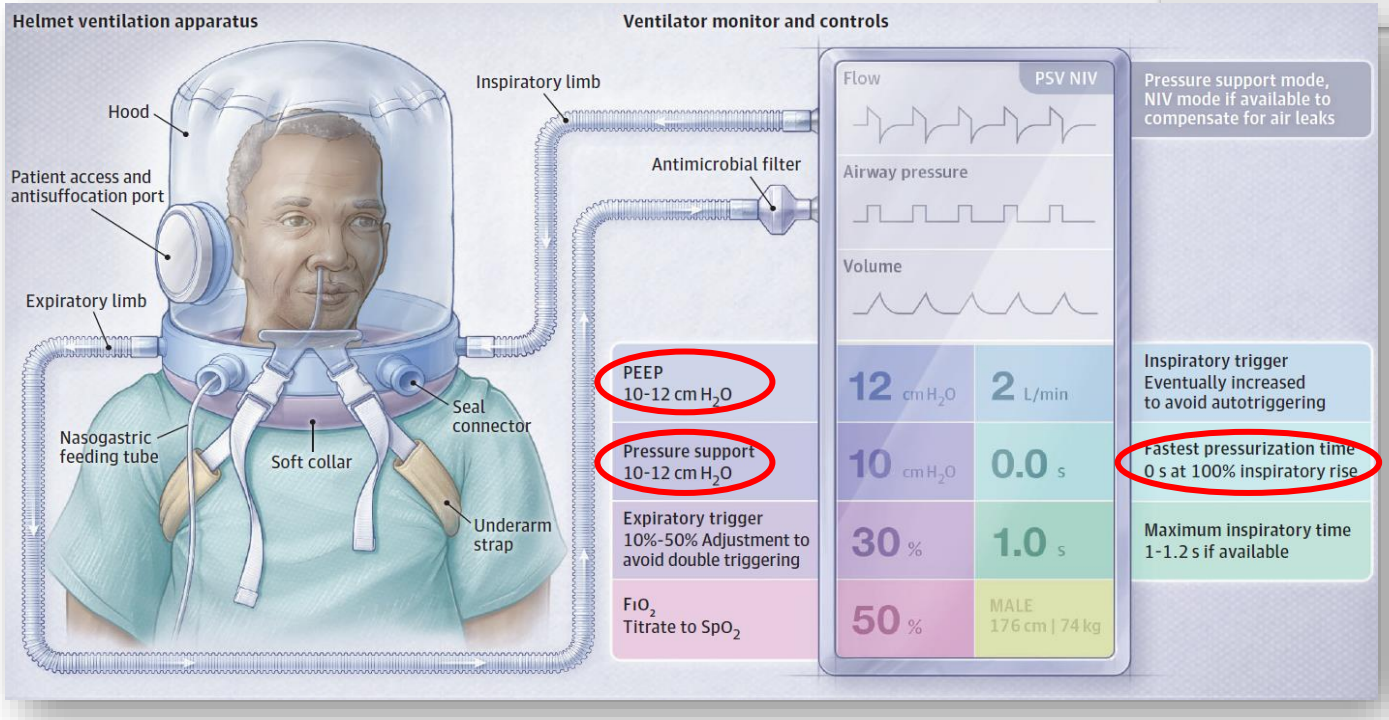
109 Patients analyzed

54
Helmet ventilation
Continuous treatment with helmet noninvasive ventilation for at least 48 hours followed by high-flow nasal oxygen

55
High-flow oxygen
High-flow nasal oxygen alone

PRIMARY OUTCOME

Median number of days free of respiratory support within 28 days after enrollment



Impostazioni

- FiO_2: 50-70%
- Flusso: 60 lpm
- Temperatura: 31-37°C

Grieco et al, JAMA 2021

Research

JAMA | Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

Effect of Helmet Noninvasive Ventilation vs High-Flow Nasal Oxygen on Days Free of Respiratory Support in Patients With COVID-19 and Moderate to Severe Hypoxemic Respiratory Failure

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Outcome primario

FINDINGS

Median respiratory support–free days

Helmet ventilation

Day 0 — **20 Days** (IQR, 0-25) — Day 28

Respiratory support–free

High-flow oxygen

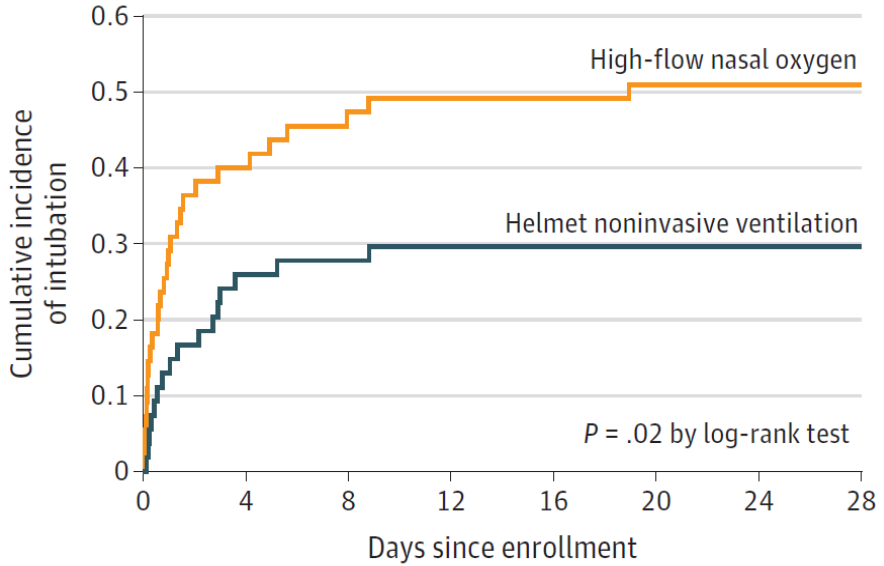
Day 0 — **18 Days** (IQR, 0-22) — Day 28

Respiratory support–free

Mean between-group difference was not statistically significant:

2 days (95% CI, −2 to 6)

Outcome secondari – IOT secondo criteri predefiniti



No. at risk

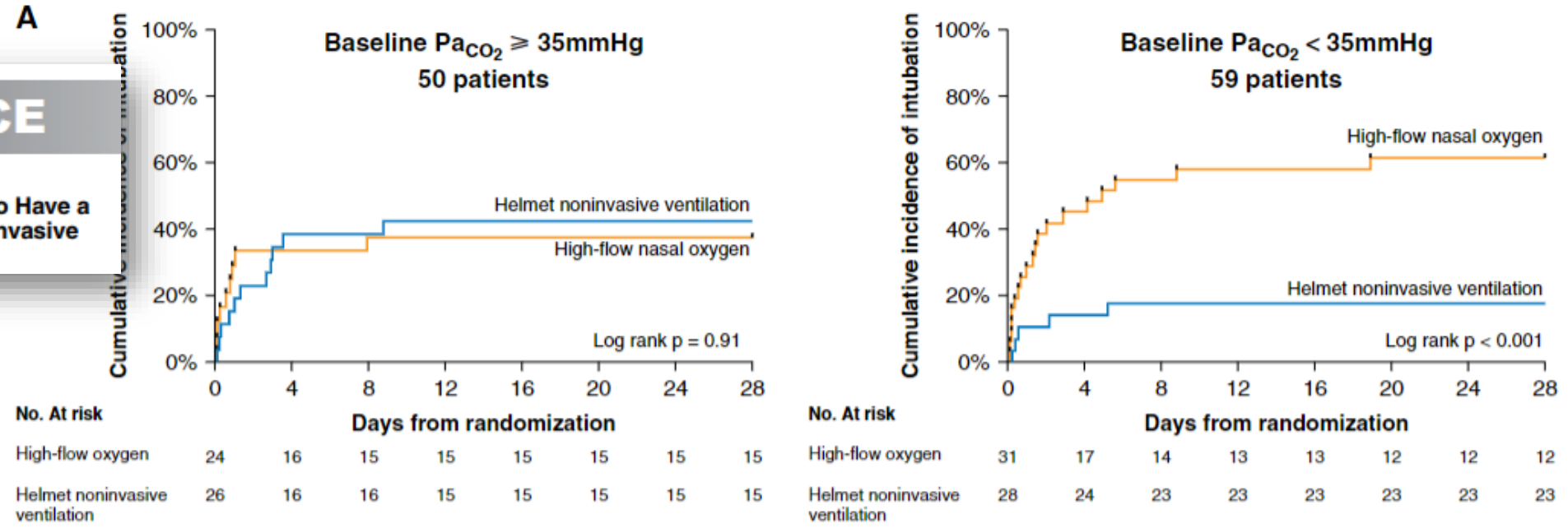
High-flow nasal oxygen	55	34	30	28	28	27	27	27
Helmet noninvasive ventilation	54	41	39	38	38	38	38	38

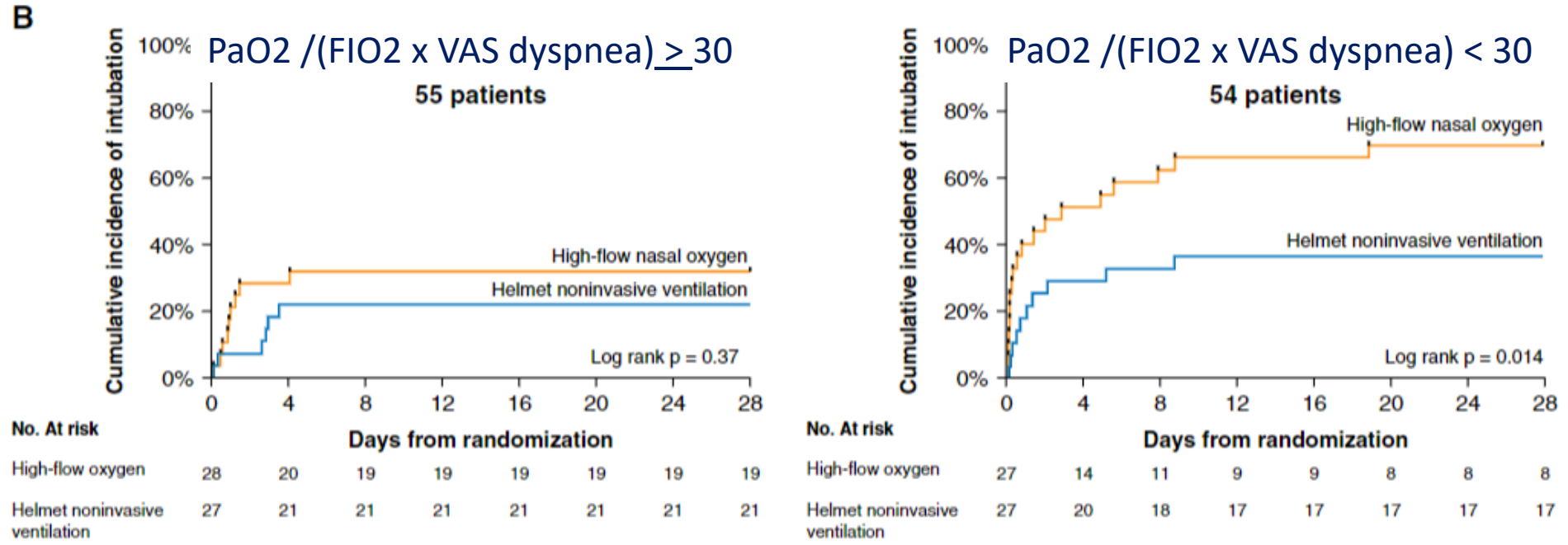
Grieco et al, JAMA 2021

CORRESPONDENCE

Check for updates

Phenotypes of Patients with COVID-19 Who Have a Positive Clinical Response to Helmet Noninvasive Ventilation





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Journal of Critical Care 30 (2015) 1390–1394

Contents lists available at ScienceDirect

Journal of Critical Care

journal homepage: www.jccjournal.org

Prone positioning improves oxygenation in spontaneously breathing nonintubated patients with hypoxemic acute respiratory failure: A retrospective study☆,☆☆,★

Vittorio Scaravilli [a,*], Giacomo Grasselli [b], Luigi Castagna [a], Alberto Zanella [a], Stefano Isgrò [b], Alberto Lucchini [b], Nicolò Patroniti [a,b], Giacomo Bellani [a,b], Antonio Pesenti [a,b]

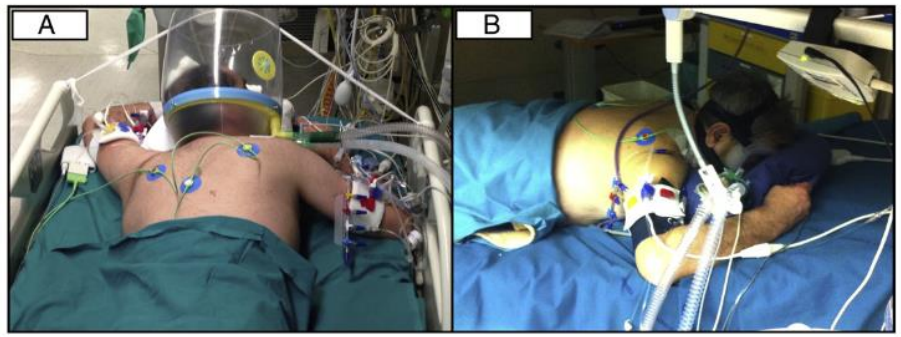


Fig. 2. Pictures of 2 representative non-intubated, spontaneously breathing patients undergoing prone position. Panel A shows prone positioning in a patient with helmet continuous positive airway pressure. Panel B shows prone positioning in a patient with mask non-invasive ventilation.

PP was feasible and improved oxygenation in non-intubated, spontaneously breathing pts with ARF

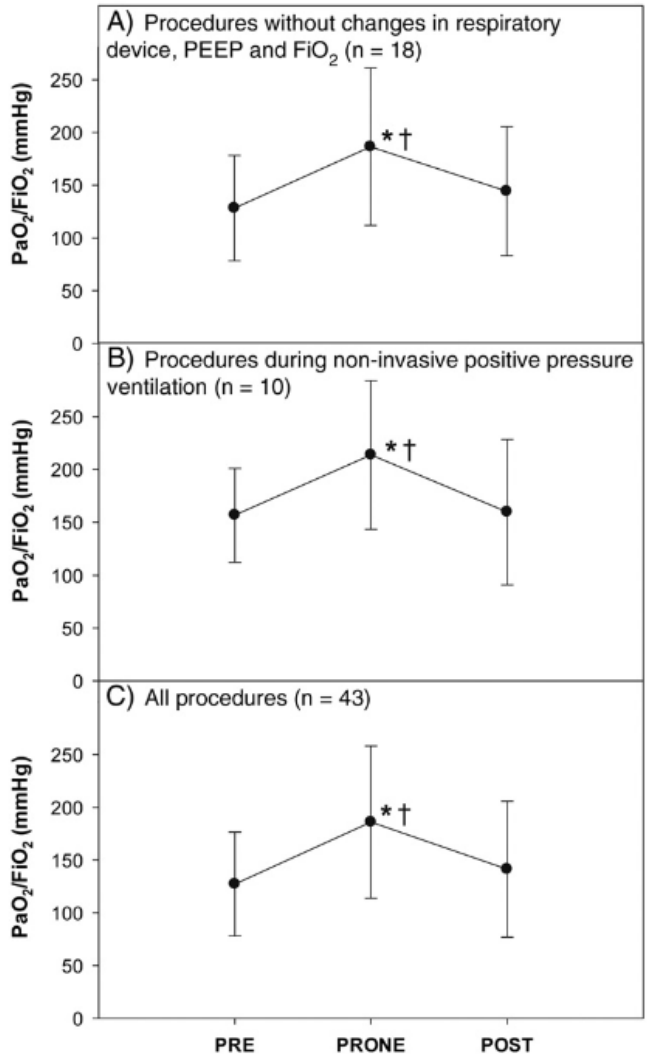


Fig. 1. Pao_2/Fio_2 during the three study steps. PRE, 1 to 2 hours before pronation; PRONE, during prone positioning; POST, 6 to 8 hours after resupination. Panel A shows Pao_2/Fio_2 of the subset of pronation procedures without changes in respiratory device, PEEP, and Fio_2 (n = 18). Panel B shows Pao_2/Fio_2 of the subset of pronation procedures performed during non-invasive positive pressure ventilation (n = 10). Panel C shows Pao_2/Fio_2 of all the performed pronation procedures. Data are represented as mean ± standard deviations. * $P < .05$ vs. PRE step and † $P < .05$ vs. POST step.

Gemelli

Pronazione in respiro spontaneo?

Grieco *et al. Critical Care* (2023) 27:315
https://doi.org/10.1186/s13054-023-04600-9

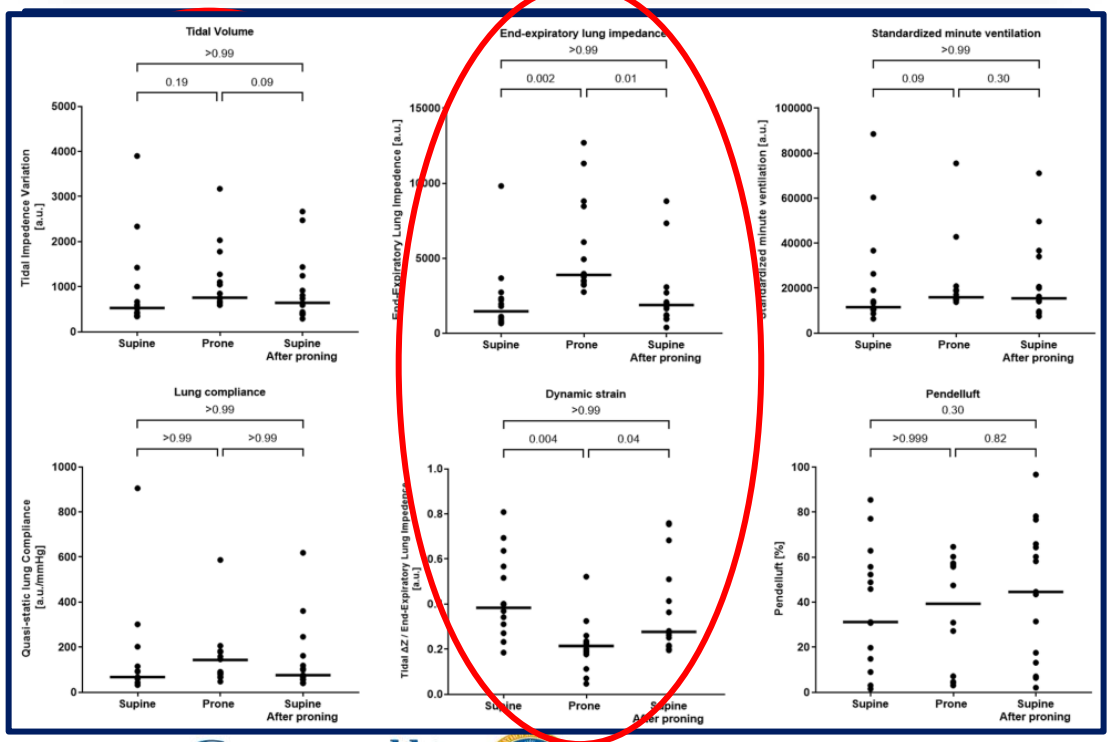
Critical Care

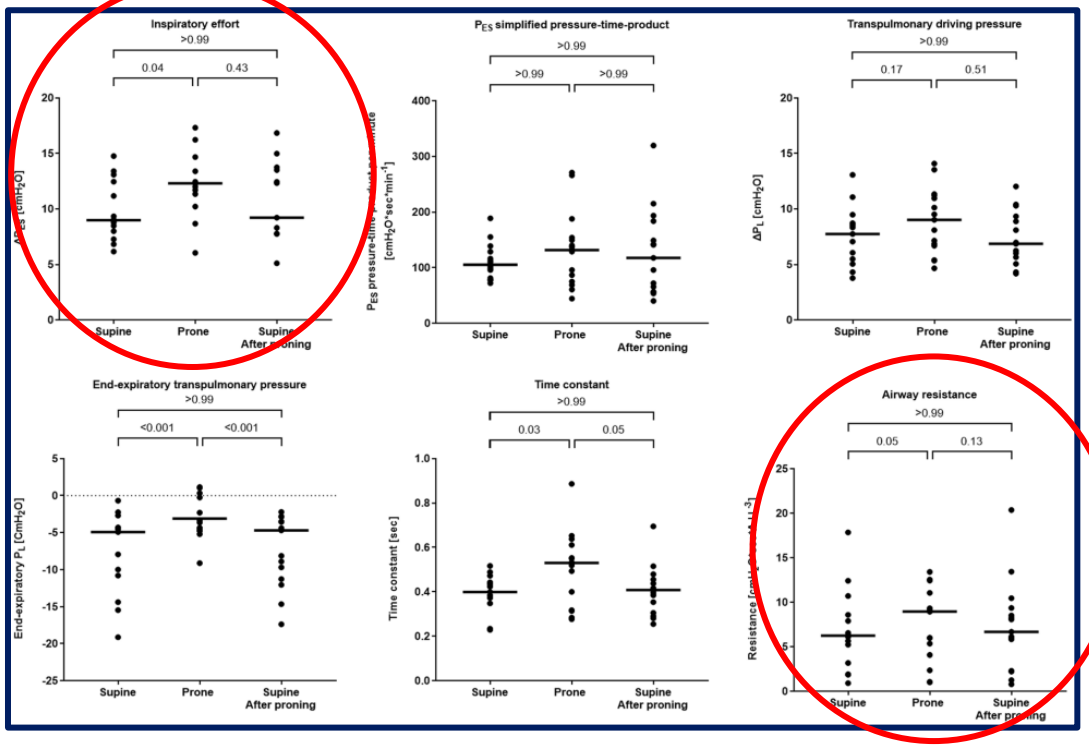
RESEARCH Open Access

Physiological effects of awake prone position in acute hypoxemic respiratory failure

Domenico Luca Grieco[1,2*], Luca Delle Cese[1,2], Luca S. Menga[1,2], Tommaso Rosà[1,2], Teresa Michi[1,2], Gianmarco Lombardi[1,2], Melania Cesarano[1,2], Valentina Giammatteo[1,2], Giuseppe Bello[1,2], Simone Carelli[1,2], Salvatore L. Cutuli[1,2], Claudio Sandroni[1,2], Gennaro De Pascale[1,2], Antonio Pesenti[3], Salvatore M. Maggiore[4,5] and Massimo Antonelli[1,2]

Lo strain dinamico è il principale responsabile del VILI, ma ΔPes e pendelluft sono i prinicipali determinanti dello P-SILI!





Modifiche gravitazionali sulle piccole vie aeree, rotazione laterale del collo

Intensive Care Med (2023) 49:840–843
https://doi.org/10.1007/s00134-023-07048-1

EDITORIAL

Personalized noninvasive respiratory support for acute hypoxemic respiratory failure

Domenico Luca Grieco[1,2*], Laveena Munshi[3,4] and Lise Piquilloud[5]

Probabilmente non esiste una strategia perfetta per tutti i pazienti:

Individualizzazione!

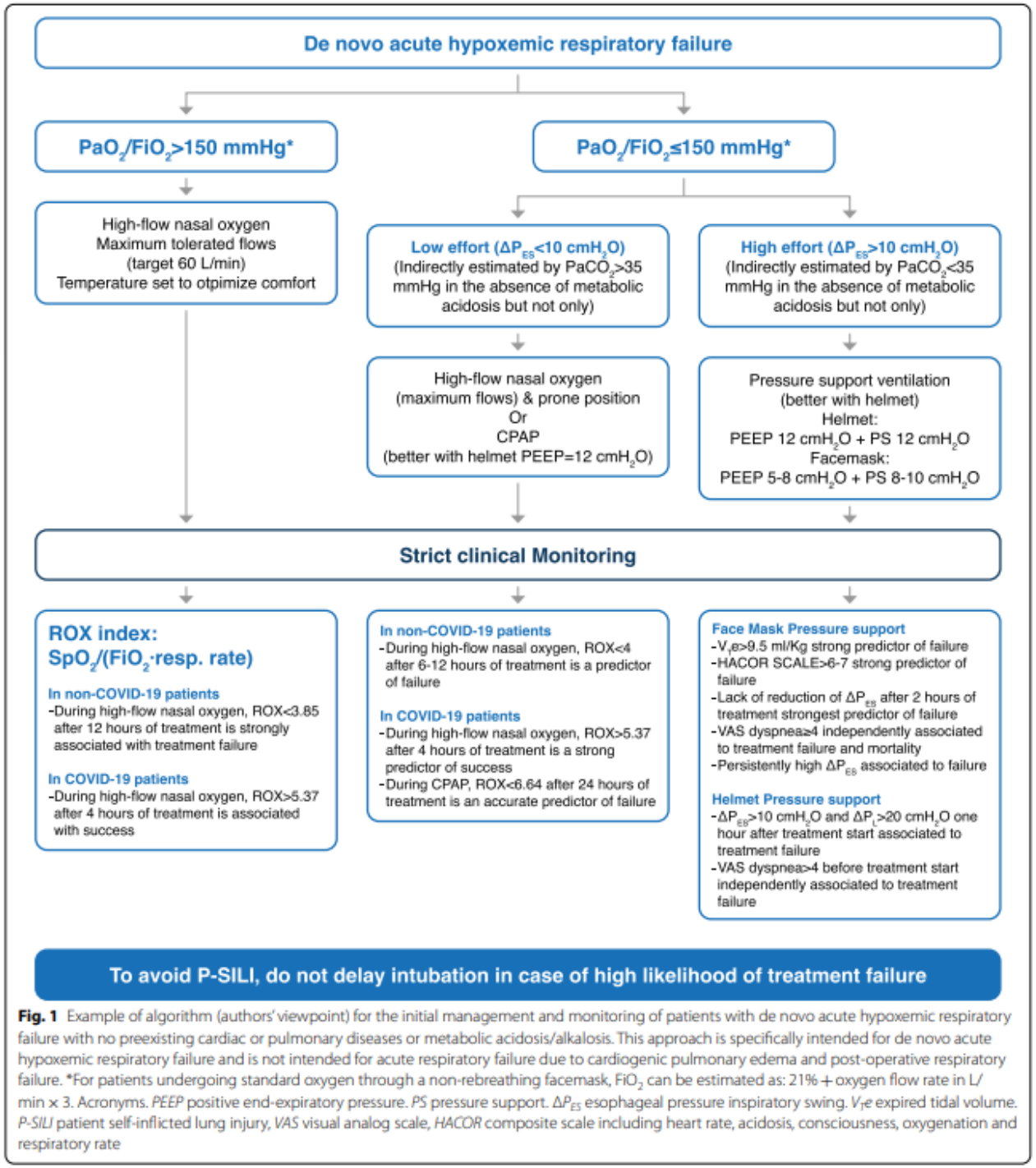


Fig. 1 Example of algorithm (authors' viewpoint) for the initial management and monitoring of patients with de novo acute hypoxemic respiratory failure with no preexisting cardiac or pulmonary diseases or metabolic acidosis/alkalosis. This approach is specifically intended for de novo acute hypoxemic respiratory failure and is not intended for acute respiratory failure due to cardiogenic pulmonary edema and post-operative respiratory failure. *For patients undergoing standard oxygen through a non-rebreathing facemask, FiO_2 can be estimated as: 21% + oxygen flow rate in L/min × 3. Acronyms. *PEEP* positive end-expiratory pressure. *PS* pressure support. ΔP_{ES} esophageal pressure inspiratory swing. V_Te expired tidal volume. *P-SILI* patient self-inflicted lung injury, *VAS* visual analog scale, *HACOR* composite scale including heart rate, acidosis, consciousness, oxygenation and respiratory rate