La 4ème Journée COmmune de Réanimation

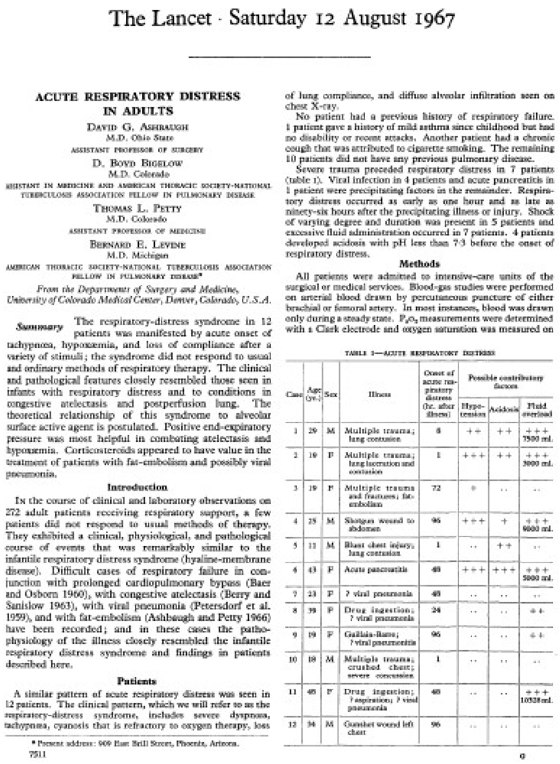
Nouvelles définitions du SDRA

AYACHI JIHENE

MCA Réanimation Médicale Kairouan

31/05/2025

Tout ce que nous savons sur le SDRA, nous l’avons appris en 1967



The Lancet · Saturday 12 August 1967

ACUTE RESPIRATORY DISTRESS IN ADULTS

DAVID G. ASHBAUGH
M.D. Ohio State
ASSISTANT PROFESSOR OF SURGERY
D. BOYD BIGELOW
M.D. Colorado
ASSISTANT IN MEDICINE AND AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE
THOMAS L. PETTY
M.D. Colorado
ASSISTANT PROFESSOR OF MEDICINE
BERNARD E. LEVINE
M.D. Michigan
AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE*

*From the Departments of Surgery and Medicine,
University of Colorado Medical Center, Denver, Colorado, U.S.A.*

Summary The respiratory-distress syndrome in 12 patients was manifested by acute onset of tachypnœa, hypoxæmia, and loss of compliance after a variety of stimuli; the syndrome did not respond to usual and ordinary methods of respiratory therapy. The clinical and pathological features closely resembled those seen in infants with respiratory distress and to conditions in congestive atelectasis and postperfusion lung. The theoretical relationship of this syndrome to alveolar surface active agent is postulated. Positive end-expiratory pressure was most helpful in combating atelectasis and hypoxæmia. Corticosteroids appeared to have value in the treatment of patients with fat-embolism and possibly viral pneumonia.

Introduction

In the course of clinical and laboratory observations on 272 adult patients receiving respiratory support, a few patients did not respond to usual methods of therapy. They exhibited a clinical, physiological, and pathological course of events that was remarkably similar to the infantile respiratory distress syndrome (hyaline-membrane disease). Difficult cases of respiratory failure in conjunction with prolonged cardiopulmonary bypass (Baer and Osborn 1960), with congestive atelectasis (Berry and Sanislow 1963), with viral pneumonia (Petersdorf et al. 1959), and with fat-embolism (Ashbaugh and Petty 1966) have been recorded; and in these cases the pathophysiology of the illness closely resembled the infantile respiratory distress syndrome and findings in patients described here.

Patients

A similar pattern of acute respiratory distress was seen in 12 patients. The clinical pattern, which we will refer to as the respiratory-distress syndrome, includes severe dyspnœa, tachypnœa, cyanosis that is refractory to oxygen therapy, loss of lung compliance, and diffuse alveolar infiltration seen on chest X-ray.

No patient had a previous history of respiratory failure. 1 patient gave a history of mild asthma since childhood but had no disability or recent attacks. Another patient had a chronic cough that was attributed to cigarette smoking. The remaining 10 patients did not have any previous pulmonary disease.

Severe trauma preceded respiratory distress in 7 patients (table I). Viral infection in 4 patients and acute pancreatitis in 1 patient were precipitating factors in the remainder. Respiratory distress occurred as early as one hour and as late as ninety-six hours after the precipitating illness or injury. Shock of varying degree and duration was present in 5 patients and excessive fluid administration occurred in 7 patients. 4 patients developed acidosis with pH less than 7·3 before the onset of respiratory distress.

Methods

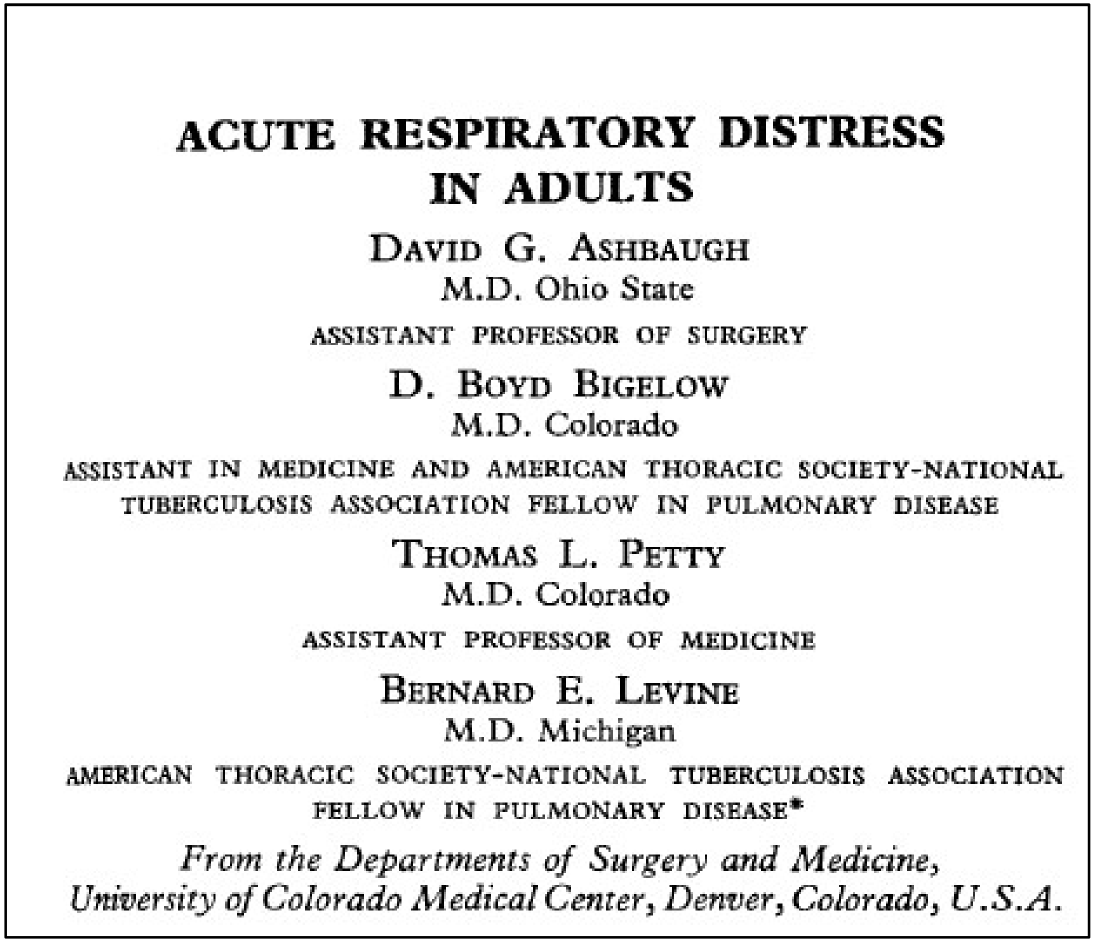
All patients were admitted to intensive-care units of the surgical or medical services. Blood-gas studies were performed on arterial blood drawn by percutaneous puncture of either brachial or femoral artery. In most instances, blood was drawn only during a steady state. P_aO_2 measurements were determined with a Clark electrode and oxygen saturation was measured on

TABLE I—ACUTE RESPIRATORY DISTRESS

Case	Age (yr.)	Sex	Illness	Onset of acute respiratory distress (hr. after illness)	Possible contributory factors		
					Hypotension	Acidosis	Fluid overload
1	29	M	Multiple trauma; lung contusion	8	++	++	+++ 7500 ml.
2	19	F	Multiple trauma; lung laceration and contusion	1	+++	++	+++ 3000 ml.
3	19	F	Multiple trauma and fractures; fat-embolism	72	0	..	..
4	25	M	Shotgun wound to abdomen	96	+++	+	+++ 6000 ml.
5	11	M	Blunt chest injury; lung contusion	1	..	++	..
6	43	F	Acute pancreatitis	48	+++	+++	+++ 5000 ml.
7	23	F	? viral pneumonia	48	..	..	..
8	39	F	Drug ingestion; ? viral pneumonia	24	..	..	++
9	19	F	Guillain-Barre; ? viral pneumonitis	96	..	..	++
10	18	M	Multiple trauma; crushed chest; severe concussion	1	..	..	..
11	48	F	Drug ingestion; ? aspiration; ? viral pneumonia	48	..	..	+++ 10528 ml.
12	34	M	Gunshot wound left chest	96	..	..	..

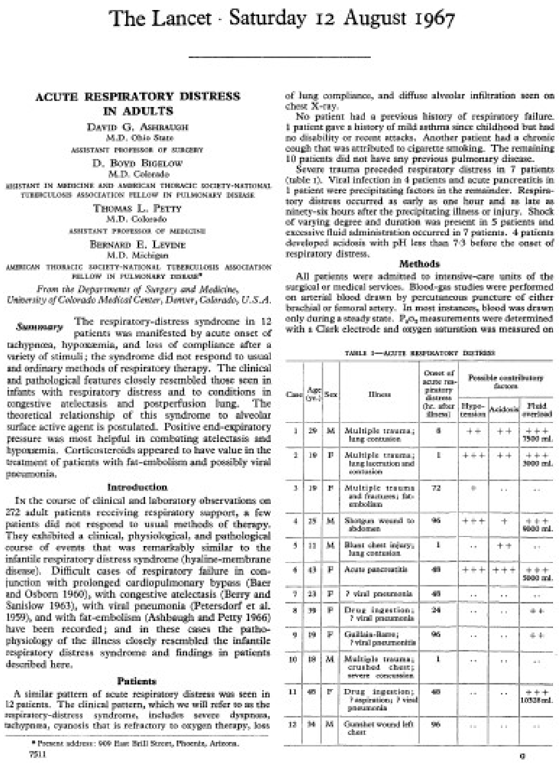
---

* Present address: 909 East Brill Street, Phoenix, Arizona.



ACUTE RESPIRATORY DISTRESS
IN ADULTS
DAVID G. ASHBAUGH
M.D. Ohio State
ASSISTANT PROFESSOR OF SURGERY
D. BOYD BIGELOW
M.D. Colorado
ASSISTANT IN MEDICINE AND AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE
THOMAS L. PETTY
M.D. Colorado
ASSISTANT PROFESSOR OF MEDICINE
BERNARD E. LEVINE
M.D. Michigan
AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE*
*From the Departments of Surgery and Medicine,
University of Colorado Medical Center, Denver, Colorado, U.S.A.*

Tout ce que nous savons sur le SDRA, nous l'avons appris en 1967



The Lancet · Saturday 12 August 1967

ACUTE RESPIRATORY DISTRESS IN ADULTS

David G. Ashbaugh
M.D. Ohio State
ASSISTANT PROFESSOR OF SURGERY

D. Boyd Bigelow
M.D. Colorado
ASSISTANT IN MEDICINE AND AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE

Thomas L. Petty
M.D. Colorado
ASSISTANT PROFESSOR OF MEDICINE

Bernard E. Levine
M.D. Michigan
AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE*

From the Department of Surgery and Medicine, University of Colorado Medical Center, Denver, Colorado, U.S.A.

Summary The respiratory-distress syndrome in 12 patients was manifested by acute onset of tachypnœa, hypoxæmia, and loss of compliance after a variety of stimuli; the syndrome did not respond to usual and ordinary methods of respiratory therapy. The clinical and pathological features closely resembled those seen in infants with respiratory distress and to conditions in congestive atelectasis and postperfusion lung. The theoretical relationship of this syndrome to alveolar surface active agent is postulated. Positive end-expiratory pressure was most helpful in combating atelectasis and hypoxæmia. Corticosteroids appeared to have value in the treatment of patients with fat-embolism and possibly viral pneumonia.

Introduction

In the course of clinical and laboratory observations on 272 adult patients receiving respiratory support, a few patients did not respond to usual methods of therapy. They exhibited a clinical, physiological, and pathological course of events that was remarkably similar to the infantile respiratory distress syndrome (hyaline-membrane disease). Difficult cases of respiratory failure in conjunction with prolonged cardiopulmonary bypass (Baer and Osborn 1960), with congestive atelectasis (Berry and Sanislow 1963), with viral pneumonia (Petersdorf et al. 1959), and with fat-embolism (Ashbaugh and Petty 1966) have been recorded; and in these cases the pathophysiology of the illness closely resembled the infantile respiratory distress syndrome and findings in patients described here.

Patients

A similar pattern of acute respiratory distress was seen in 12 patients. The clinical pattern, which we will refer to as the respiratory-distress syndrome, includes severe dyspnœa, tachypnœa, cyanosis that is refractory to oxygen therapy, loss of lung compliance, and diffuse alveolar infiltration seen on chest X-ray.

No patient had a previous history of respiratory failure. 1 patient gave a history of mild asthma since childhood but had no disability or recent attacks. Another patient had a chronic cough that was attributed to cigarette smoking. The remaining 10 patients did not have any previous pulmonary disease.

Severe trauma preceded respiratory distress in 7 patients (table I). Viral infection in 4 patients and acute pancreatitis in 1 patient were precipitating factors in the remainder. Respiratory distress occurred as early as one hour and as late as ninety-six hours after the precipitating illness or injury. Shock of varying degree and duration was present in 5 patients and excessive fluid administration occurred in 7 patients. 4 patients developed acidosis with pH less than 7·3 before the onset of respiratory distress.

Methods

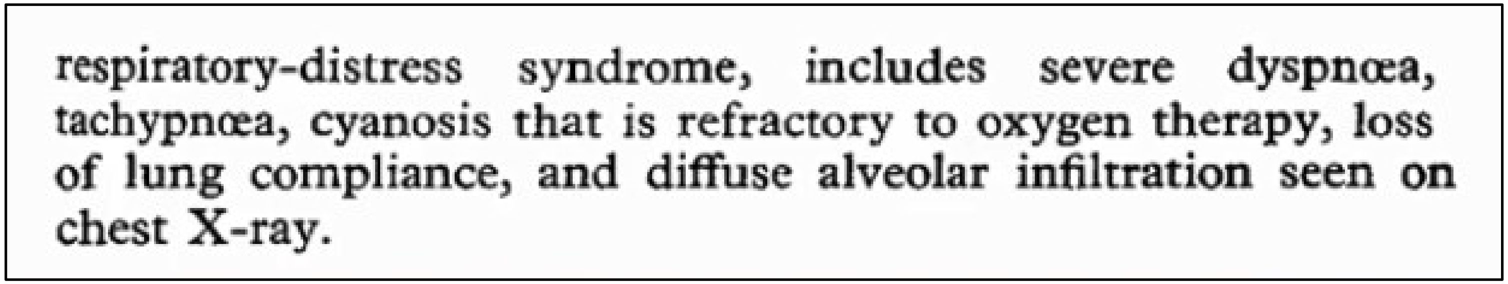
All patients were admitted to intensive-care units of the surgical or medical services. Blood-gas studies were performed on arterial blood drawn by percutaneous puncture of either brachial or femoral artery. In most instances, blood was drawn only during a steady state. P_aO_2 measurements were determined with a Clark electrode and oxygen saturation was measured on

TABLE I—ACUTE RESPIRATORY DISTRESS

Case	Age (yr.)	Sex	Illness	Onset of acute respiratory distress (hr. after illness)	Possible contributory factors		
					Hypotension	Acidosis	Fluid overload
1	29	M	Multiple trauma; lung contusion	8	++	++	+++ 7500 ml.
2	19	F	Multiple trauma; lung laceration and contusion	1	+++	++	+++ 3000 ml.
3	19	F	Multiple trauma and fractures; fat-embolism	72	+	..	..
4	25	M	Shotgun wound to abdomen	96	+++	+	+++ 9000 ml.
5	11	M	Blunt chest injury; lung contusion	1	..	++	..
6	43	F	Acute pancreatitis	48	+++	+++	+++ 5000 ml.
7	23	F	? viral pneumonia	48	..	..	..
8	39	F	Drug ingestion; ? viral pneumonia	24	..	..	++
9	19	F	Guillain-Barre; ? viral pneumonitis	96	..	..	++
10	18	M	Multiple trauma; crushed chest; severe concussion	1	..	..	..
11	48	F	Drug ingestion; ? aspiration; ? viral pneumonia	48	..	..	+++ 10528 ml.
12	34	M	Gunshot wound left chest	96	..	..	..

---

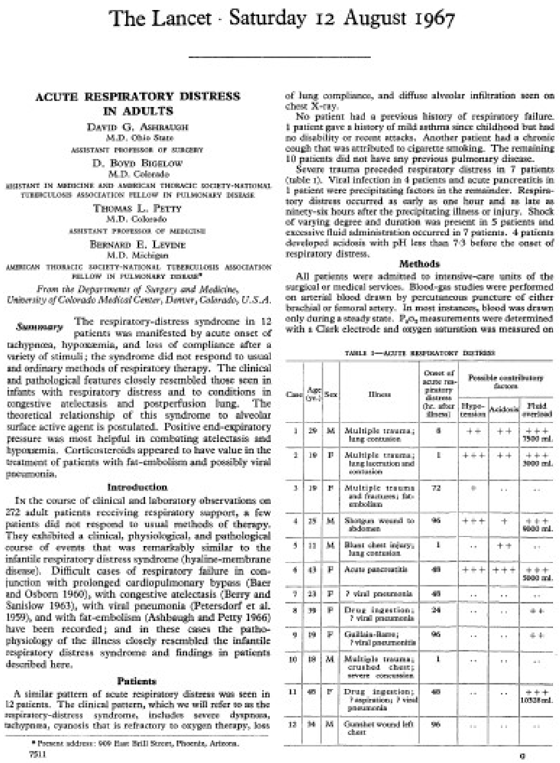
* Present address: 909 East Brill Street, Phoenix, Arizona.



respiratory-distress syndrome, includes severe dyspnœa, tachypnœa, cyanosis that is refractory to oxygen therapy, loss of lung compliance, and diffuse alveolar infiltration seen on chest X-ray.

Début brutal, hypoxémie, faible compliance et anomalies radiologiques

Tout ce que nous savons sur le SDRA, nous l'avons appris en 1967



The Lancet · Saturday 12 August 1967

ACUTE RESPIRATORY DISTRESS IN ADULTS

David G. Ashbaugh
M.D. Ohio State
ASSISTANT PROFESSOR OF SURGERY

D. Boyd Bigelow
M.D. Colorado
ASSISTANT IN MEDICINE AND AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE

Thomas L. Petty
M.D. Colorado
ASSISTANT PROFESSOR OF MEDICINE

Bernard E. Levine
M.D. Michigan
AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE*

From the Departments of Surgery and Medicine, University of Colorado Medical Center, Denver, Colorado, U.S.A.

Summary The respiratory-distress syndrome in 12 patients was manifested by acute onset of tachypnœa, hypoxæmia, and loss of compliance after a variety of stimuli; the syndrome did not respond to usual and ordinary methods of respiratory therapy. The clinical and pathological features closely resembled those seen in infants with respiratory distress and to conditions in congestive atelectasis and postperfusion lung. The theoretical relationship of this syndrome to alveolar surface active agent is postulated. Positive end-expiratory pressure was most helpful in combating atelectasis and hypoxæmia. Corticosteroids appeared to have value in the treatment of patients with fat-embolism and possibly viral pneumonia.

Introduction

In the course of clinical and laboratory observations on 272 adult patients receiving respiratory support, a few patients did not respond to usual methods of therapy. They exhibited a clinical, physiological, and pathological course of events that was remarkably similar to the infantile respiratory distress syndrome (hyaline-membrane disease). Difficult cases of respiratory failure in conjunction with prolonged cardiopulmonary bypass (Baer and Osborn 1960), with congestive atelectasis (Berry and Sanislow 1963), with viral pneumonia (Petersdorf et al. 1959), and with fat-embolism (Ashbaugh and Petty 1966) have been recorded; and in these cases the pathophysiology of the illness closely resembled the infantile respiratory distress syndrome and findings in patients described here.

Patients

A similar pattern of acute respiratory distress was seen in 12 patients. The clinical pattern, which we will refer to as the respiratory-distress syndrome, includes severe dyspnœa, tachypnœa, cyanosis that is refractory to oxygen therapy, loss of lung compliance, and diffuse alveolar infiltration seen on chest X-ray.

No patient had a previous history of respiratory failure. 1 patient gave a history of mild asthma since childhood but had no disability or recent attacks. Another patient had a chronic cough that was attributed to cigarette smoking. The remaining 10 patients did not have any previous pulmonary disease.

Severe trauma preceded respiratory distress in 7 patients (table I). Viral infection in 4 patients and acute pancreatitis in 1 patient were precipitating factors in the remainder. Respiratory distress occurred as early as one hour and as late as ninety-six hours after the precipitating illness or injury. Shock of varying degree and duration was present in 5 patients and excessive fluid administration occurred in 7 patients. 4 patients developed acidosis with pH less than 7·3 before the onset of respiratory distress.

Methods

All patients were admitted to intensive-care units of the surgical or medical services. Blood-gas studies were performed on arterial blood drawn by percutaneous puncture of either brachial or femoral artery. In most instances, blood was drawn only during a steady state. P_aO_2 measurements were determined with a Clark electrode and oxygen saturation was measured on

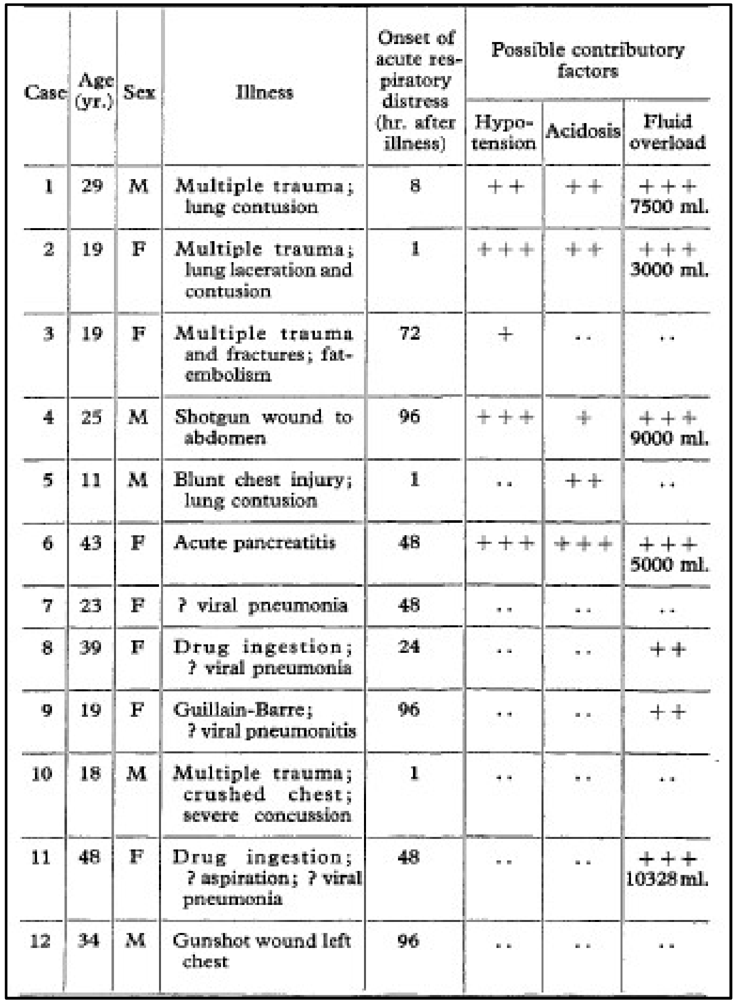
TABLE I—ACUTE RESPIRATORY DISTRESS

---

* Present address: 909 East Brill Street, Phoenix, Arizona.

7511

G

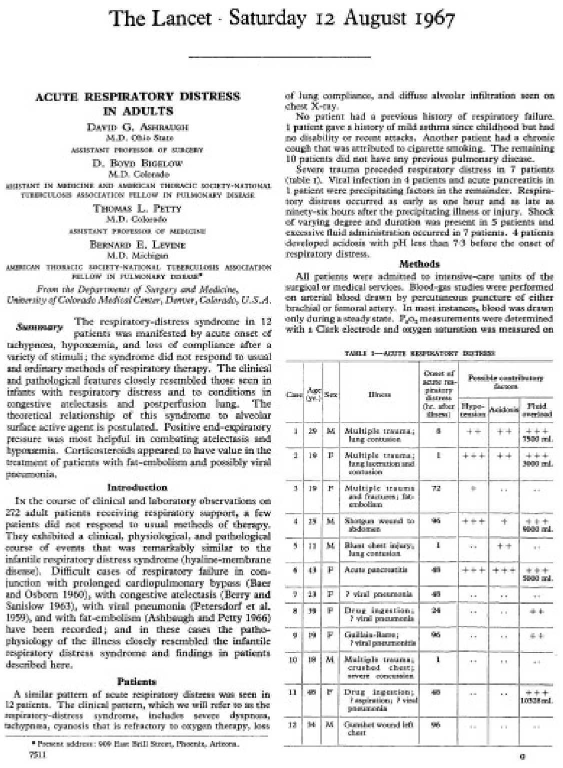


Case	Age (yr.)	Sex	Illness	Onset of acute respiratory distress (hr. after illness)	Possible contributory factors		
					Hypotension	Acidosis	Fluid overload
1	29	M	Multiple trauma; lung contusion	8	++	++	+++ 7500 ml.
2	19	F	Multiple trauma; lung laceration and contusion	1	+++	++	+++ 3000 ml.
3	19	F	Multiple trauma and fractures; fat-embolism	72	+	..	..
4	25	M	Shotgun wound to abdomen	96	+++	+	+++ 9000 ml.
5	11	M	Blunt chest injury; lung contusion	1	..	++	..
6	43	F	Acute pancreatitis	48	+++	+++	+++ 5000 ml.
7	23	F	? viral pneumonia	48	..	..	..
8	39	F	Drug ingestion; ? viral pneumonia	24	..	..	++
9	19	F	Guillain-Barre; ? viral pneumonitis	96	..	..	++
10	18	M	Multiple trauma; crushed chest; severe concussion	1	..	..	..
11	48	F	Drug ingestion; ? aspiration; ? viral pneumonia	48	..	..	+++ 10328 ml.
12	34	M	Gunshot wound left chest	96	..	..	..

Facteurs de risque:

- **Trauma**
- **Pancréatite**
- **Pneumonie**
- **Inhalation**
- **Embolie graisseuse**
- **Choc**

Tout ce que nous savons sur le SDRA, nous l’avons appris en 1967



The Lancet · Saturday 12 August 1967

ACUTE RESPIRATORY DISTRESS IN ADULTS

DAVID G. ASHBAUGH
M.D. Ohio State
ASSISTANT PROFESSOR OF SURGERY

D. BOYD BIGELOW
M.D. Colorado
ASSISTANT IN MEDICINE AND AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE

THOMAS L. PETTY
M.D. Colorado
ASSISTANT PROFESSOR OF MEDICINE

BERNARD E. LEVINE
M.D. Michigan
AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE*

From the Department of Surgery and Medicine, University of Colorado Medical Center, Denver, Colorado, U.S.A.

Summary The respiratory-distress syndrome in 12 patients was manifested by acute onset of tachypnœa, hypoxæmia, and loss of compliance after a variety of stimuli; the syndrome did not respond to usual and ordinary methods of respiratory therapy. The clinical and pathological features closely resembled those seen in infants with respiratory distress and to conditions in congestive atelectasis and postperfusion lung. The theoretical relationship of this syndrome to alveolar surface active agent is postulated. Positive end-expiratory pressure was most helpful in combating atelectasis and hypoxæmia. Corticosteroids appeared to have value in the treatment of patients with fat-embolism and possibly viral pneumonia.

Introduction

In the course of clinical and laboratory observations on 272 adult patients receiving respiratory support, a few patients did not respond to usual methods of therapy. They exhibited a clinical, physiological, and pathological course of events that was remarkably similar to the infantile respiratory distress syndrome (hyaline-membrane disease). Difficult cases of respiratory failure in conjunction with prolonged cardiopulmonary bypass (Baer and Osborn 1960), with congestive atelectasis (Berry and Sanislow 1963), with viral pneumonia (Petersdorf et al. 1959), and with fat-embolism (Ashbaugh and Petty 1966) have been recorded; and in these cases the pathophysiology of the illness closely resembled the infantile respiratory distress syndrome and findings in patients described here.

Patients

A similar pattern of acute respiratory distress was seen in 12 patients. The clinical pattern, which we will refer to as the respiratory-distress syndrome, includes severe dyspnœa, tachypnœa, cyanosis that is refractory to oxygen therapy, loss of lung compliance, and diffuse alveolar infiltration seen on chest X-ray.

No patient had a previous history of respiratory failure. 1 patient gave a history of mild asthma since childhood but had no disability or recent attacks. Another patient had a chronic cough that was attributed to cigarette smoking. The remaining 10 patients did not have any previous pulmonary disease.

Severe trauma preceded respiratory distress in 7 patients (table I). Viral infection in 4 patients and acute pancreatitis in 1 patient were precipitating factors in the remainder. Respiratory distress occurred as early as one hour and as late as ninety-six hours after the precipitating illness or injury. Shock of varying degree and duration was present in 5 patients and excessive fluid administration occurred in 7 patients. 4 patients developed acidosis with pH less than 7·3 before the onset of respiratory distress.

Methods

All patients were admitted to intensive-care units of the surgical or medical services. Blood-gas studies were performed on arterial blood drawn by percutaneous puncture of either brachial or femoral artery. In most instances, blood was drawn only during a steady state. P_aO_2 measurements were determined with a Clark electrode and oxygen saturation was measured on

TABLE I—ACUTE RESPIRATORY DISTRESS

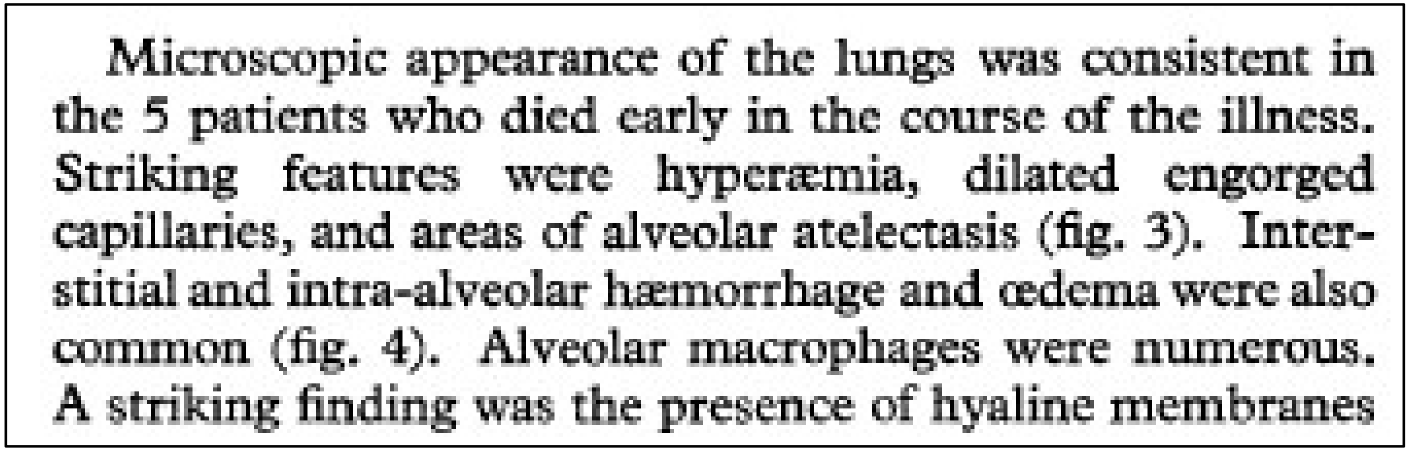
Case	Age (yr.)	Sex	Illness	Onset of acute respiratory distress (hr. after illness)	Possible contributory factors		
					Hypotension	Acidosis	Fluid overload
1	29	M	Multiple trauma; lung contusion	8	++	++	+++ 7500 ml.
2	19	F	Multiple trauma; lung laceration and contusion	1	+++	++	+++ 3000 ml.
3	19	F	Multiple trauma and fractures; fat-embolism	72	+	..	..
4	25	M	Shotgun wound to abdomen	96	+++	+	+++ 9000 ml.
5	11	M	Blunt chest injury; lung contusion	1	..	++	..
6	43	F	Acute pancreatitis	48	+++	+++	+++ 5000 ml.
7	23	F	? viral pneumonia	48	..	..	..
8	39	F	Drug ingestion; ? viral pneumonia	24	..	..	++
9	19	F	Guillain-Barre; ? viral pneumonitis	96	..	..	++
10	18	M	Multiple trauma; crushed chest; severe concussion	1	..	..	..
11	48	F	Drug ingestion; ? aspiration; ? viral pneumonia	48	..	..	+++ 10528 ml.
12	34	M	Gunshot wound left chest	96	..	..	..

---

* Present address: 909 East Brill Street, Phoenix, Arizona.

7511

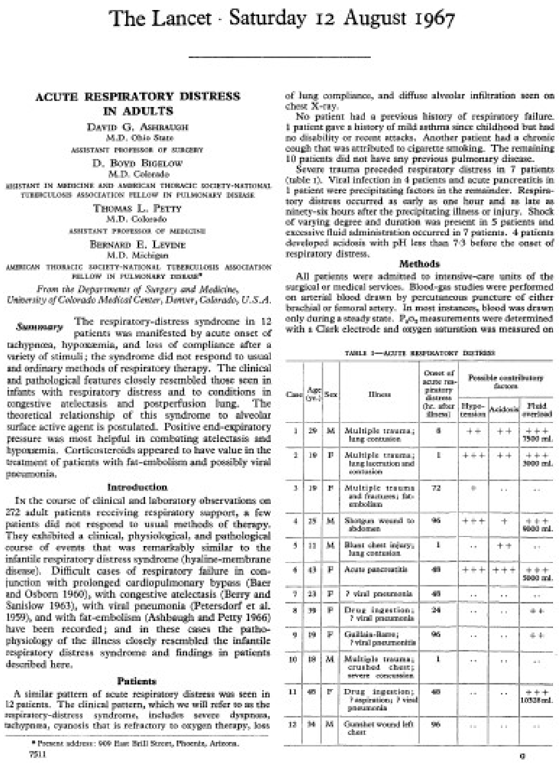
G



Microscopic appearance of the lungs was consistent in the 5 patients who died early in the course of the illness. Striking features were hyperæmia, dilated engorged capillaries, and areas of alveolar atelectasis (fig. 3). Interstitial and intra-alveolar hæmorrhage and œdema were also common (fig. 4). Alveolar macrophages were numerous. A striking finding was the presence of hyaline membranes

Signes pathologiques classiques : membranes hyalines, progression vers fibrose

Tout ce que nous savons sur le SDRA, nous l’avons appris en 1967



The Lancet · Saturday 12 August 1967

ACUTE RESPIRATORY DISTRESS IN ADULTS

David G. Ashbaugh
M.D. Ohio State
ASSISTANT PROFESSOR OF SURGERY

D. Boyd Bigelow
M.D. Colorado
ASSISTANT IN MEDICINE AND AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE

Thomas L. Petty
M.D. Colorado
ASSISTANT PROFESSOR OF MEDICINE

Bernard E. Levine
M.D. Michigan
AMERICAN THORACIC SOCIETY-NATIONAL TUBERCULOSIS ASSOCIATION FELLOW IN PULMONARY DISEASE*

From the Department of Surgery and Medicine, University of Colorado Medical Center, Denver, Colorado, U.S.A.

Summary The respiratory-distress syndrome in 12 patients was manifested by acute onset of tachypnea, hypoxemia, and loss of compliance after a variety of stimuli; the syndrome did not respond to usual and ordinary methods of respiratory therapy. The clinical and pathological features closely resembled those seen in infants with respiratory distress and to conditions in congestive atelectasis and postperfusion lung. The theoretical relationship of this syndrome to alveolar surface active agent is postulated. Positive end-expiratory pressure was most helpful in combating atelectasis and hypoxemia. Corticosteroids appeared to have value in the treatment of patients with fat-embolism and possibly viral pneumonia.

Introduction

In the course of clinical and laboratory observations on 272 adult patients receiving respiratory support, a few patients did not respond to usual methods of therapy. They exhibited a clinical, physiological, and pathological course of events that was remarkably similar to the infantile respiratory distress syndrome (hyaline-membrane disease). Difficult cases of respiratory failure in conjunction with prolonged cardiopulmonary bypass (Baer and Osborn 1960), with congestive atelectasis (Berry and Sanislow 1963), with viral pneumonia (Petersdorf et al. 1959), and with fat-embolism (Ashbaugh and Petty 1966) have been recorded; and in these cases the pathophysiology of the illness closely resembled the infantile respiratory distress syndrome and findings in patients described here.

Patients

A similar pattern of acute respiratory distress was seen in 12 patients. The clinical pattern, which we will refer to as the respiratory-distress syndrome, includes severe dyspnœa, tachypnœa, cyanosis that is refractory to oxygen therapy, loss of lung compliance, and diffuse alveolar infiltration seen on chest X-ray.

No patient had a previous history of respiratory failure. 1 patient gave a history of mild asthma since childhood but had no disability or recent attacks. Another patient had a chronic cough that was attributed to cigarette smoking. The remaining 10 patients did not have any previous pulmonary disease.

Severe trauma preceded respiratory distress in 7 patients (table I). Viral infection in 4 patients and acute pancreatitis in 1 patient were precipitating factors in the remainder. Respiratory distress occurred as early as one hour and as late as ninety-six hours after the precipitating illness or injury. Shock of varying degree and duration was present in 5 patients and excessive fluid administration occurred in 7 patients. 4 patients developed acidosis with pH less than 7·3 before the onset of respiratory distress.

Methods

All patients were admitted to intensive-care units of the surgical or medical services. Blood-gas studies were performed on arterial blood drawn by percutaneous puncture of either brachial or femoral artery. In most instances, blood was drawn only during a steady state. P_aO_2 measurements were determined with a Clark electrode and oxygen saturation was measured on

TABLE I—ACUTE RESPIRATORY DISTRESS

Case	Age (yr.)	Sex	Illness	Onset of acute respiratory distress (hr. after illness)	Possible contributory factors		
					Hypotension	Acidosis	Fluid overload
1	29	M	Multiple trauma; lung contusion	8	++	++	+++ 7500 ml.
2	19	F	Multiple trauma; lung laceration and contusion	1	+++	++	+++ 3000 ml.
3	19	F	Multiple trauma and fractures; fat-embolism	72	*	..	..
4	25	M	Shotgun wound to abdomen	96	+++	+	+++ 9000 ml.
5	11	M	Blunt chest injury; lung contusion	1	..	++	..
6	43	F	Acute pancreatitis	48	+++	+++	+++ 5000 ml.
7	23	F	? viral pneumonia	48	..	..	..
8	39	F	Drug ingestion; ? viral pneumonia	24	..	..	++
9	19	F	Guillain-Barre; ? viral pneumonitis	96	..	..	++
10	18	M	Multiple trauma; crushed chest; severe concussion	1	..	..	..
11	48	F	Drug ingestion; ? aspiration; ? viral pneumonia	48	..	..	+++ 10528 ml.
12	34	M	Gunshot wound left chest	96	..	..	..

---

* Present address: 909 East Brill Street, Phoenix, Arizona.

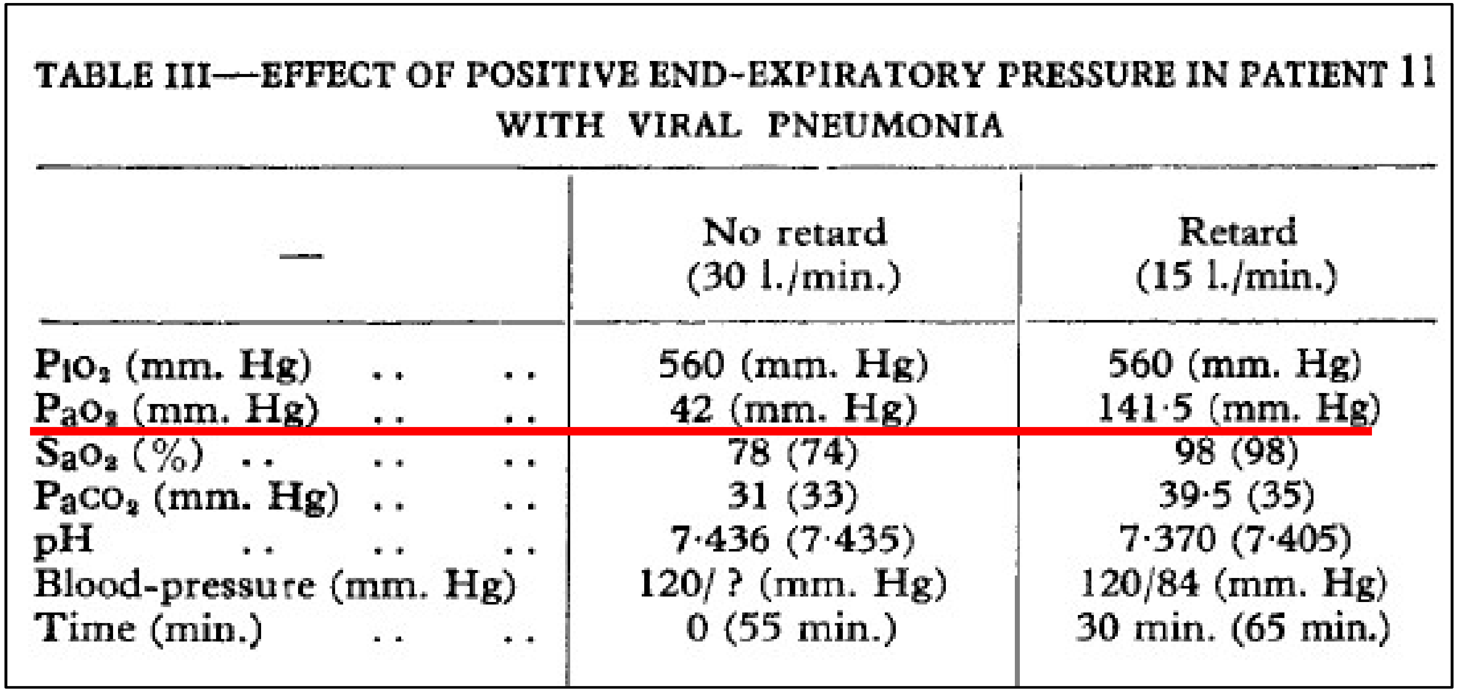
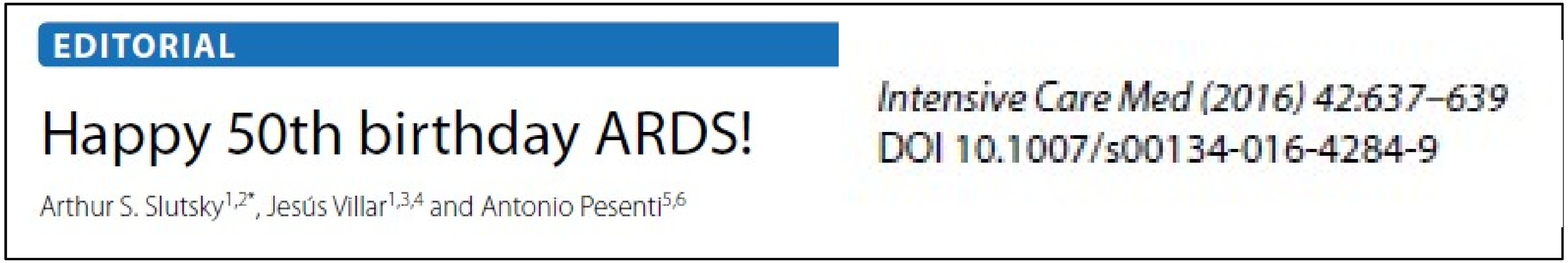


TABLE III—EFFECT OF POSITIVE END-EXPIRATORY PRESSURE IN PATIENT 11 WITH VIRAL PNEUMONIA

—	No retard (30 l./min.)	Retard (15 l./min.)
P_IO_2 (mm. Hg)	560 (mm. Hg)	560 (mm. Hg)
P_aO_2 (mm. Hg)	42 (mm. Hg)	141·5 (mm. Hg)
S_aO_2 (%)	78 (74)	98 (98)
P_aCO_2 (mm. Hg)	31 (33)	39·5 (35)
pH	7·436 (7·435)	7·370 (7·405)
Blood-pressure (mm. Hg)	120/ ? (mm. Hg)	120/84 (mm. Hg)
Time (min.)	0 (55 min.)	30 min. (65 min.)

Amélioration de l’oxygénation avec la PEP

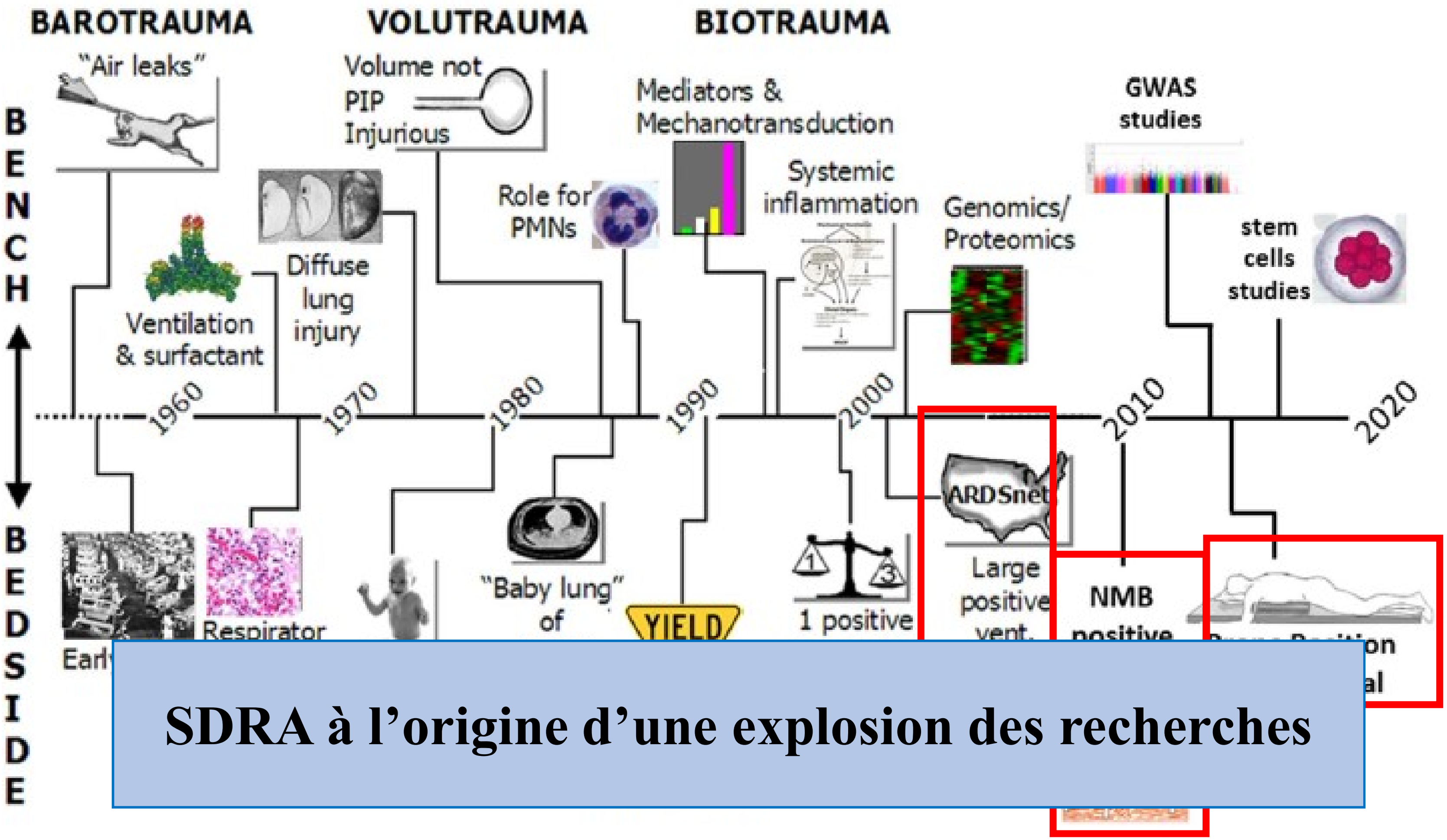


EDITORIAL

Happy 50th birthday ARDS!

Arthur S. Slutsky[1,2*], Jesús Villar[1,3,4] and Antonio Pesenti[5,6]

Intensive Care Med (2016) 42:637–639
DOI 10.1007/s00134-016-4284-9


BAROTRAUMA
VOLUTRAUMA
BIOTRAUMA
"Air leaks"
Volume not PIP Injurious
Mediators & Mechanotransduction
GWAS studies
BENCH
Role for PMNs
Systemic inflammation
Genomics/ Proteomics
stem cells studies
Ventilation & surfactant
Diffuse lung injury
1960
1970
1980
1990
2000
2010
2020
BEDSIDE
Earl
Respirator
"Baby lung" of
YIELD
1 positive
1
3
ARDSnet
Large positive vent.
NMB positive
on
al

SDRA à l'origine d'une explosion des recherches

The American-European Consensus Conference on ARDS

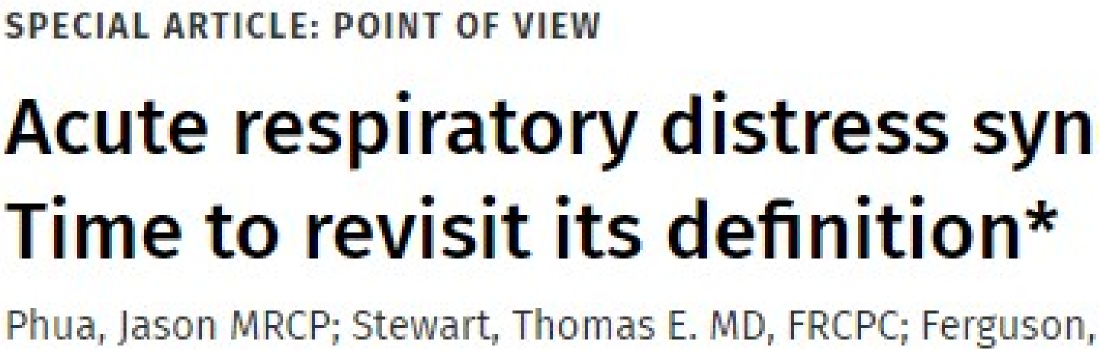
Definitions, Mechanisms, Relevant Outcomes, and Clinical Trial Coordination

Bernard et al, AJRCCM 1994; 149:818-24

- **Début aigue**
- **PaO2/FiO2≤ 200**
- **Infiltrat radiologique bilatéral à la RP**
- **Absence d’insuffisance cardiaque, d’hypervolémie ou PAPO ≤ 18**

Les limites de l’AECC 1994

- **Début aigue**: Pas de définition précise : heures, jours, semaine..
- **PaO2/FiO2≤ 200** sans tenir compte de la PEP et de la FiO2
- **Infiltrat radiologique bilatéral à la RP** : Problème d’interprétation
- **PAPO élevée** et SDRA peuvent coexister



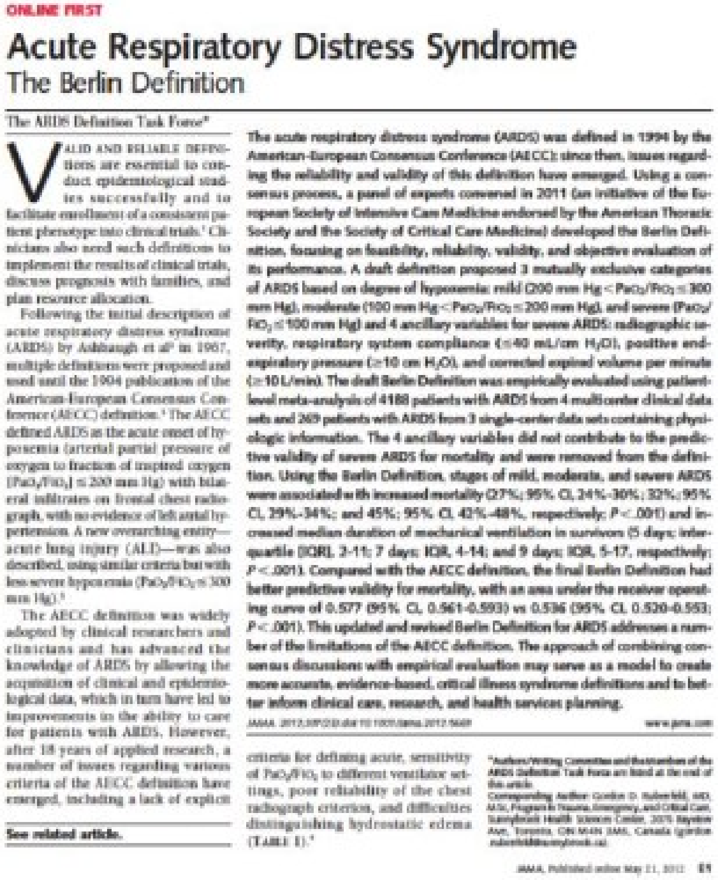
SPECIAL ARTICLE: POINT OF VIEW

Acute respiratory distress syndrome 40 years later: Time to revisit its definition*

Phua, Jason MRCP; Stewart, Thomas E. MD, FRCPC; Ferguson, Niall D. MD, MSc, FRCPC

Crit Care Med 2008 ; 36:2912-2921

Update de la définition du SDRA



ONLINE FIRST

Acute Respiratory Distress Syndrome
The Berlin Definition

The ARDS Definition Task Force*

The acute respiratory distress syndrome (ARDS) was defined in 1994 by the American-European Consensus Conference (AECC); since then, issues regarding the reliability and validity of this definition have emerged. Using a consensus process, a panel of experts convened in 2011 (an initiative of the European Society of Intensive Care Medicine endorsed by the American Thoracic Society and the Society of Critical Care Medicine) developed the Berlin Definition, focusing on feasibility, reliability, validity, and objective evaluation of its performance. A draft definition proposed 3 mutually exclusive categories of ARDS based on degree of hypoxemia: mild (200 mm Hg $<$ $PaO_2/FIO_2 \leq 300$ mm Hg), moderate (100 mm Hg $<$ $PaO_2/FIO_2 \leq 200$ mm Hg), and severe ($PaO_2/FIO_2 \leq 100$ mm Hg) and 4 ancillary variables for severe ARDS: radiographic severity, respiratory system compliance (≤40 mL/cm H_2O), positive end-expiratory pressure (≥10 cm H_2O), and corrected expired volume per minute (≥10 L/min). The draft Berlin Definition was empirically evaluated using patient-level meta-analysis of 4188 patients with ARDS from 4 multicenter clinical data sets and 269 patients with ARDS from 3 single-center data sets containing physiologic information. The 4 ancillary variables did not contribute to the predictive validity of severe ARDS for mortality and were removed from the definition. Using the Berlin Definition, stages of mild, moderate, and severe ARDS were associated with increased mortality (27%; 95% CI, 24%-30%; 32%; 95% CI, 29%-34%; and 45%; 95% CI, 42%-48%, respectively; $P < .001$) and increased median duration of mechanical ventilation in survivors (5 days; interquartile [IQR], 2-11; 7 days; IQR, 4-14; and 9 days; IQR, 5-17, respectively; $P < .001$). Compared with the AECC definition, the final Berlin Definition had better predictive validity for mortality, with an area under the receiver operating curve of 0.577 (95% CI, 0.561-0.593) vs 0.536 (95% CI, 0.520-0.553; $P < .001$). This updated and revised Berlin Definition for ARDS addresses a number of the limitations of the AECC definition. The approach of combining consensus discussions with empirical evaluation may serve as a model to create more accurate, evidence-based, critical illness syndrome definitions and to better inform clinical care, research, and health services planning.

JAMA. 2012;307(23):doi:10.1001/jama.2012.5669 www.jama.com

VALID AND RELIABLE DEFINITIONS are essential to conduct epidemiological studies successfully and to facilitate enrollment of a consistent patient phenotype into clinical trials.[1] Clinicians also need such definitions to implement the results of clinical trials, discuss prognosis with families, and plan resource allocation.

Following the initial description of acute respiratory distress syndrome (ARDS) by Ashbaugh et al[2] in 1967, multiple definitions were proposed and used until the 1994 publication of the American-European Consensus Conference (AECC) definition.[3] The AECC defined ARDS as the acute onset of hypoxemia (arterial partial pressure of oxygen to fraction of inspired oxygen [PaO_2/FIO_2] ≤200 mm Hg) with bilateral infiltrates on frontal chest radiograph, with no evidence of left atrial hypertension. A new overarching entity—acute lung injury (ALI)—was also described, using similar criteria but with less severe hypoxemia ($PaO_2/FIO_2 \leq 300$ mm Hg).[3]

The AECC definition was widely adopted by clinical researchers and clinicians and has advanced the knowledge of ARDS by allowing the acquisition of clinical and epidemiological data, which in turn have led to improvements in the ability to care for patients with ARDS. However, after 18 years of applied research, a number of issues regarding various criteria of the AECC definition have emerged, including a lack of explicit criteria for defining acute, sensitivity of PaO_2/FIO_2 to different ventilator settings, poor reliability of the chest radiograph criterion, and difficulties distinguishing hydrostatic edema (TABLE 1).[3]

See related article.

*Authors/Writing Committee and the members of the ARDS Definition Task Force are listed at the end of this article.
Corresponding Author: Gordon D. Rubenfeld, MD, MSc, Program in Trauma, Emergency, and Critical Care, Sunnybrook Health Sciences Center, 2075 Bayview Ave, Toronto, ON M4N 3M5, Canada (gordon.rubenfeld@sunnybrook.ca).

JAMA®
The Journal of the American Medical Association

The ARDS Definition Task Force

Acute Respiratory Distress Syndrome:
The Berlin Definition

Published online May 21, 2012

An initiative of

EUROPEAN SOCIETY OF INTENSIVE CARE MEDICINE

Endorsed by

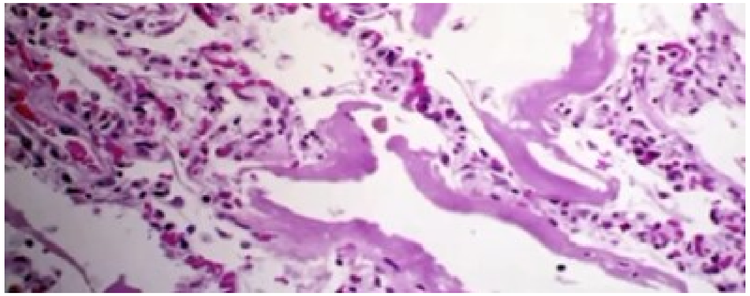
AMERICAN THORACIC SOCIETY 1905 ATS.

Society of Critical Care Medicine
The Intensive Care Professionals

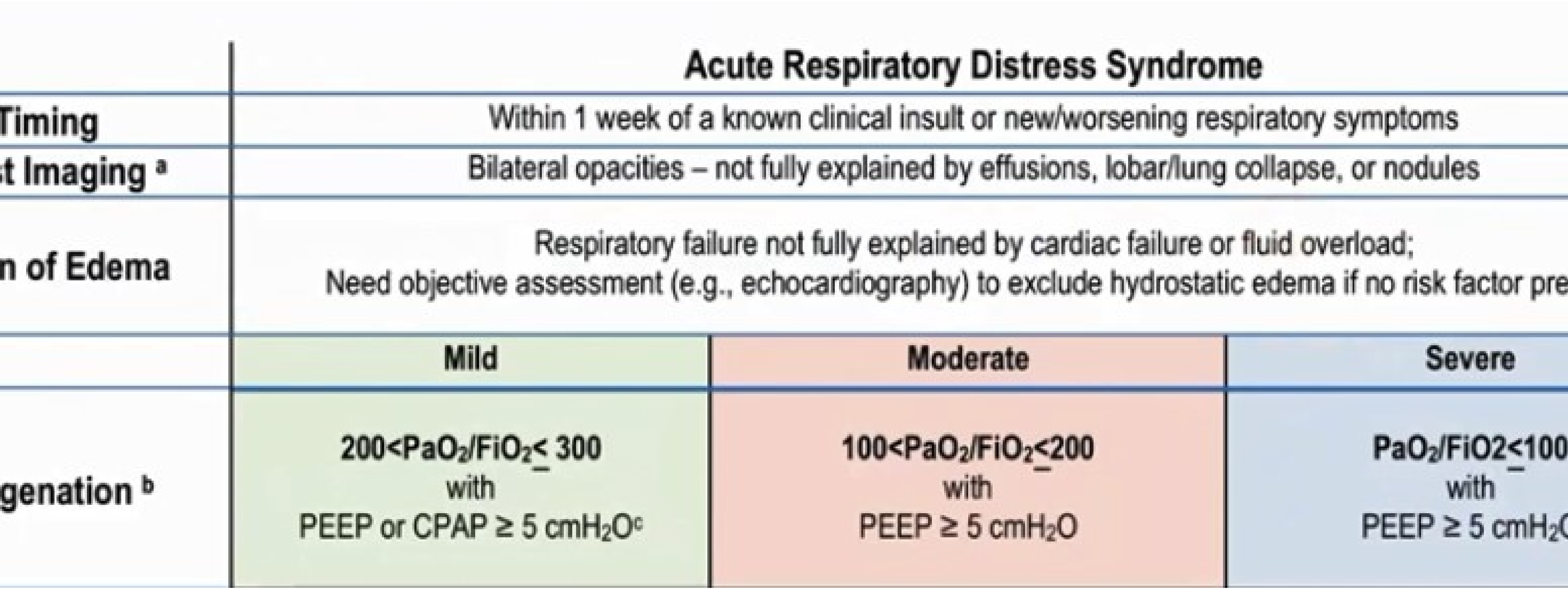
Andrew RHODES
Anders ESTEBAN
Karen PICKETT

Définition de BERLIN 2012

- Atteinte pulmonaire inflammatoire aigue et diffuse
- Augmentation de la perméabilité capillaire, du poids pulmonaire et perte du tissu pulmonaire aéré
- Clinique : hypoxémie, opacités bilatérales, baisse de la compliance
- Atteinte alvéolaire diffuse

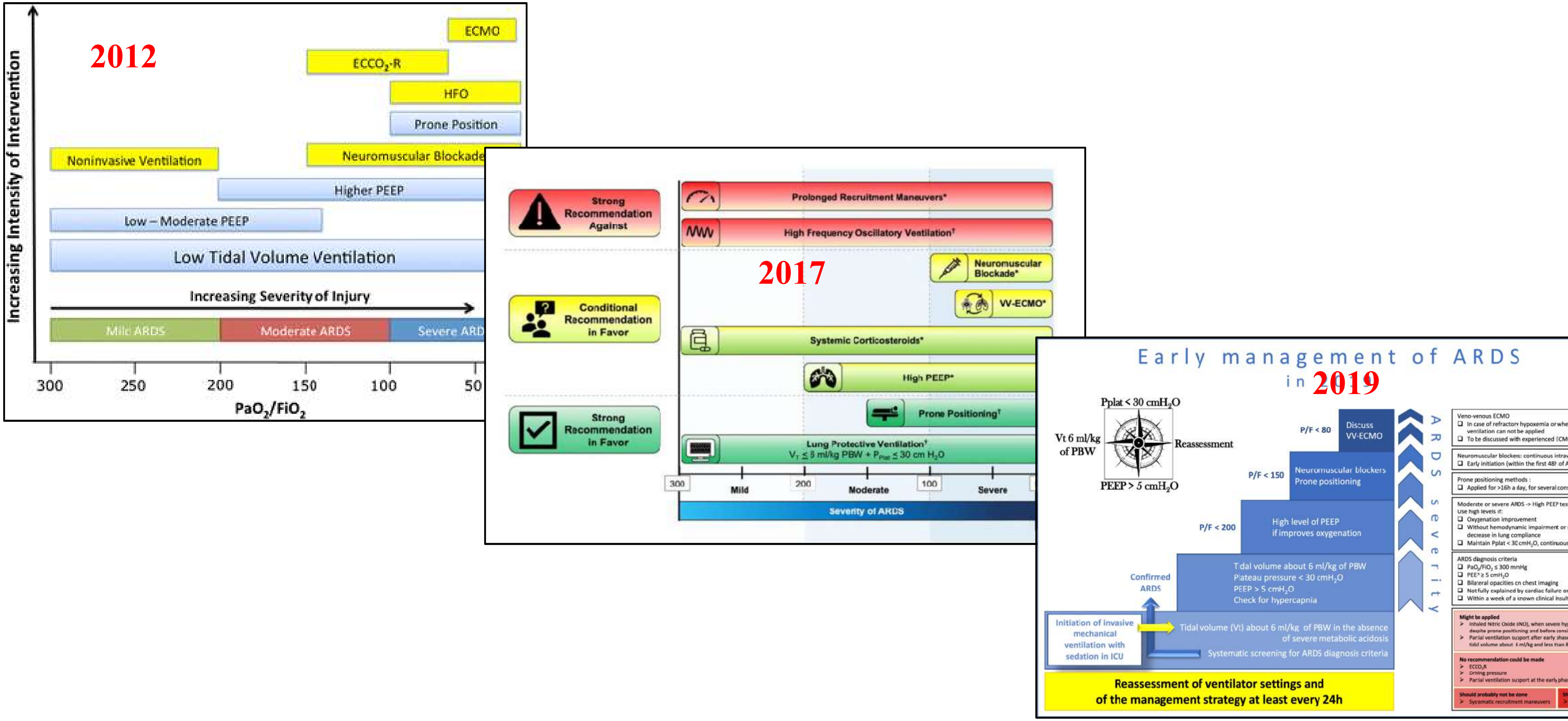


Définition de BERLIN 2012



	Acute Respiratory Distress Syndrome		
Timing	Within 1 week of a known clinical insult or new/worsening respiratory symptoms		
t Imaging [a]	Bilateral opacities – not fully explained by effusions, lobar/lung collapse, or nodules		
n of Edema	Respiratory failure not fully explained by cardiac failure or fluid overload; Need objective assessment (e.g., echocardiography) to exclude hydrostatic edema if no risk factor pre		
	Mild	Moderate	Severe
genation [b]	$200 < PaO_2/FiO_2 \leq 300$ with PEEP or CPAP ≥ 5 cmH_2O[c]	$100 < PaO_2/FiO_2 \leq 200$ with PEEP ≥ 5 cmH_2O	$PaO_2/FiO2 \leq 100$ with PEEP ≥ 5 cmH_2

Depuis la Définition de BERLIN 2012


2012
Increasing Intensity of Intervention
ECMO
ECCO$_2$-R
HFO
Prone Position
Noninvasive Ventilation
Neuromuscular Blockade
Higher PEEP
Low – Moderate PEEP
Low Tidal Volume Ventilation
Increasing Severity of Injury
Mild ARDS
Moderate ARDS
Severe ARD
300
250
200
150
100
50
PaO$_2$/FiO$_2$
2017
Strong Recommendation Against
Conditional Recommendation in Favor
Strong Recommendation in Favor
Prolonged Recruitment Maneuvers*
High Frequency Oscillatory Ventilation†
Neuromuscular Blockade*
VV-ECMO*
Systemic Corticosteroids*
High PEEP*
Prone Positioning†
Lung Protective Ventilation†
$V_T \leq 8$ ml/kg PBW + $P_{Plat} \leq 30$ cm H_2O
300
Mild
200
Moderate
100
Severe
Severity of ARDS
Early management of ARDS
in 2019
Pplat < 30 cmH$_2$O
Vt 6 ml/kg of PBW
Reassessment
PEEP > 5 cmH$_2$O
P/F < 80
Discuss VV-ECMO
P/F < 150
Neuromuscular blockers
Prone positioning
P/F < 200
High level of PEEP
if improves oxygenation
Confirmed ARDS
Tidal volume about 6 ml/kg of PBW
Plateau pressure < 30 cmH$_2$O
PEEP > 5 cmH$_2$O
Check for hypercapnia
Initiation of invasive mechanical ventilation with sedation in ICU
Tidal volume (Vt) about 6 ml/kg of PBW in the absence of severe metabolic acidosis
Systematic screening for ARDS diagnosis criteria
Reassessment of ventilator settings and
of the management strategy at least every 24h
ARDS severity
Veno-venous ECMO
☐ In case of refractory hypoxemia or whe
ventilation can not be applied
☐ To be discussed with experienced ECM
Neuromuscular blockers: continuous intrav
☐ Early initiation (within the first 48h of A
Prone positioning methods :
☐ Applied for >16h a day, for several con
Moderate or severe ARDS -> High PEEP tes
Use high levels if:
☐ Oxygenation improvement
☐ Without hemodynamic impairment or
decrease in lung compliance
☐ Maintain Pplat < 30 cmH$_2$O, continuous
ARDS diagnosis criteria
☐ PaO$_2$/FiO$_2$ ≤ 300 mmHg
☐ PEEP ≥ 5 cmH$_2$O
☐ Bilateral opacities on chest imaging
☐ Not fully explained by cardiac failure o
☐ Within a week of a known clinical insul
Might be applied
➢ Inhaled Nitric Oxide (iNO), when severe hy
despite prone positioning and before cons
➢ Partial ventilation support after early phas
tidal volume about 6 ml/kg and less than 8
No recommendation could be made
➢ ECCO$_2$R
➢ Driving pressure
➢ Partial ventilation support at the early pha
Should probably not be done
➢ Systematic recruitment maneuvers

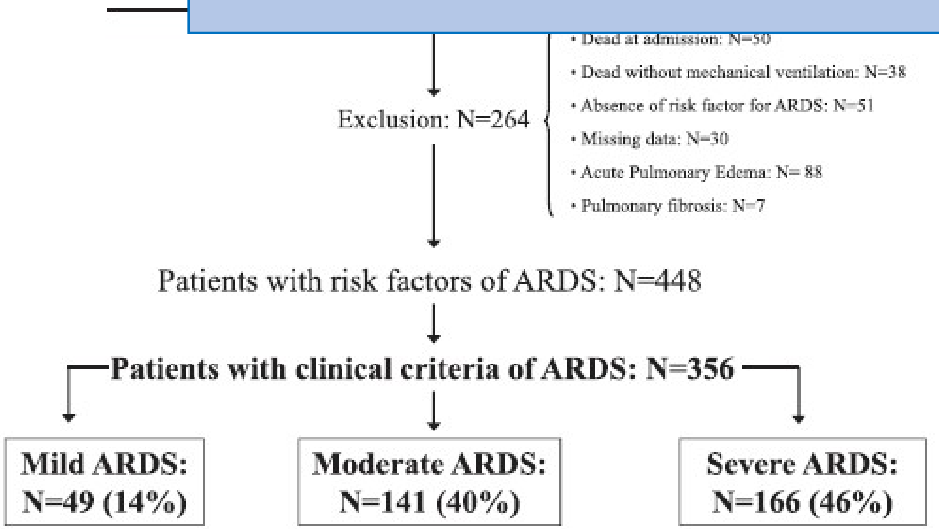
Définition de BERLIN oui MAIS….

Comparison of the Berlin Definition for Acute Respiratory Distress Syndrome with Autopsy

AJRCCM 2013

Arnaud W. Thille[1,3], Andrés Esteban[1], Pilar Fernández-Segoviano[2], José-Maria Rodriguez[2], José-Antonio Aramburu[2], Oscar Peñuelas[1], Irene Cortés-Puch[1], Pablo Cardinal-Fernández[1], José A. Lorente[1], and Fernando Frutos-Vivar[1]

La définition n’est pas parfaite si on prend en considération le critère d’atteinte alvéolaire diffuse surtout pour les stades minimes et modérés



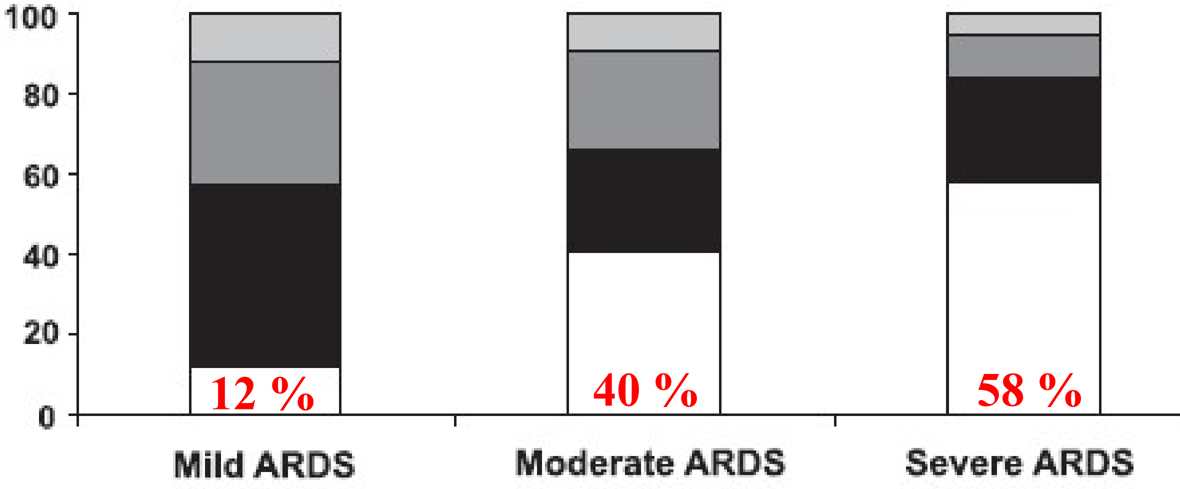
- Dead at admission: N=50
- Dead without mechanical ventilation: N=38
- Absence of risk factor for ARDS: N=51
- Missing data: N=30
- Acute Pulmonary Edema: N= 88
- Pulmonary fibrosis: N=7

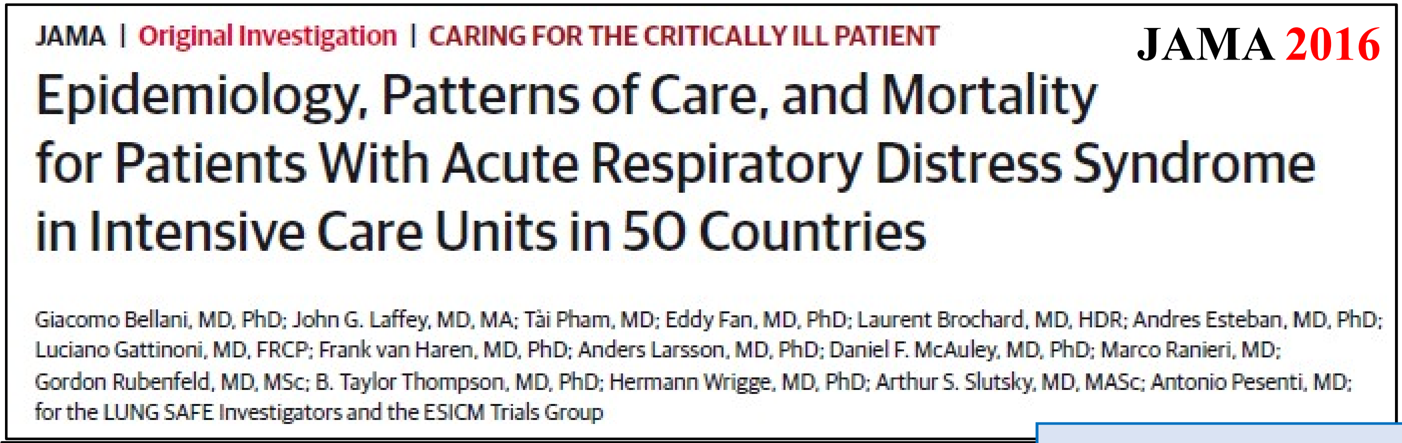
Exclusion: N=264

Patients with risk factors of ARDS: N=448

Patients with clinical criteria of ARDS: N=356

Mild ARDS: N=49 (14%)	Moderate ARDS: N=141 (40%)	Severe ARDS: N=166 (46%)





JAMA | Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

JAMA 2016

Epidemiology, Patterns of Care, and Mortality for Patients With Acute Respiratory Distress Syndrome in Intensive Care Units in 50 Countries

Giacomo Bellani, MD, PhD; John G. Laffey, MD, MA; Tài Pham, MD; Eddy Fan, MD, PhD; Laurent Brochard, MD, HDR; Andres Esteban, MD, PhD; Luciano Gattinoni, MD, FRCP; Frank van Haren, MD, PhD; Anders Larsson, MD, PhD; Daniel F. McAuley, MD, PhD; Marco Ranieri, MD; Gordon Rubenfeld, MD, MSc; B. Taylor Thompson, MD, PhD; Hermann Wrigge, MD, PhD; Arthur S. Slutsky, MD, MASc; Antonio Pesenti, MD; for the LUNG SAFE Investigators and the ESICM Trials Group

Etude LUNG-SAFE
459 Réanimations dans 50 pays
Été 2014 sur un mois
Recueil prospectif des données des patients avec VM
29144 patients

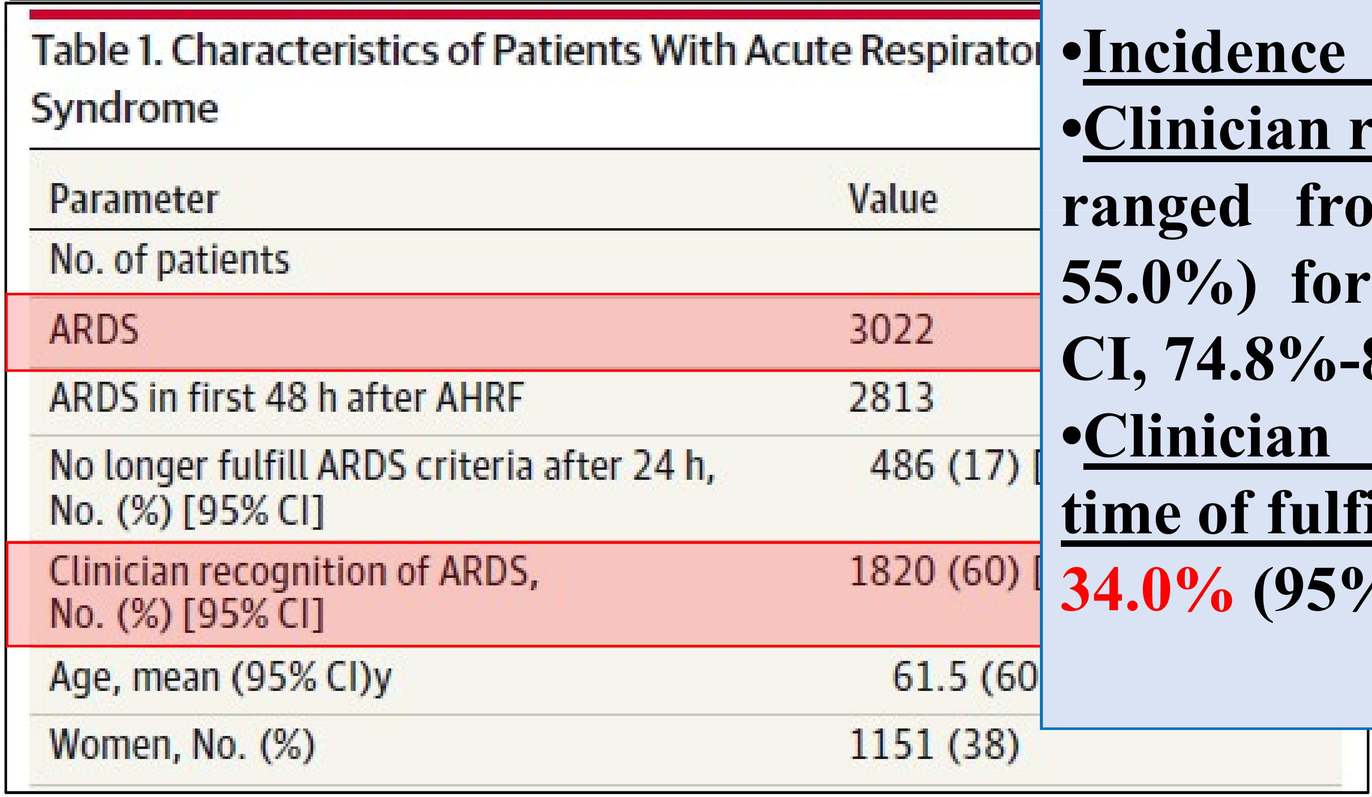


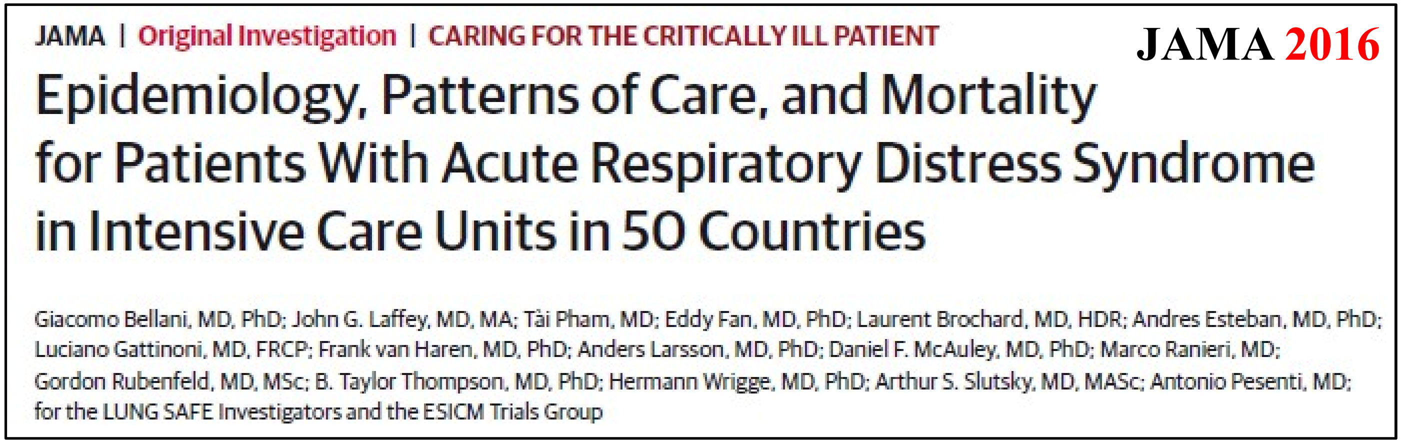
Table 1. Characteristics of Patients With Acute Respirator
Syndrome

Parameter	Value
No. of patients	
ARDS	3022
ARDS in first 48 h after AHRF	2813
No longer fulfill ARDS criteria after 24 h, No. (%) [95% CI]	486 (17) [
Clinician recognition of ARDS, No. (%) [95% CI]	1820 (60) [
Age, mean (95% CI)y	61.5 (60
Women, No. (%)	1151 (38)

•Incidence of ARDS : 10,4%

•Clinician recognition of ARDS ranged from 51.3% (95% CI, 47.5%-55.0%) for mild ARDS to 78.5% (95% CI, 74.8%-81.8%) for severe ARDS.

•Clinician recognition of ARDS at the time of fulfillment of ARDS criteria was 34.0% (95% CI, 32.0-36.0)



JAMA | Original Investigation | CARING FOR THE CRITICALLY ILL PATIENT

Epidemiology, Patterns of Care, and Mortality for Patients With Acute Respiratory Distress Syndrome in Intensive Care Units in 50 Countries

Giacomo Bellani, MD, PhD; John G. Laffey, MD, MA; Tài Pham, MD; Eddy Fan, MD, PhD; Laurent Brochard, MD, HDR; Andres Esteban, MD, PhD; Luciano Gattinoni, MD, FRCP; Frank van Haren, MD, PhD; Anders Larsson, MD, PhD; Daniel F. McAuley, MD, PhD; Marco Ranieri, MD; Gordon Rubenfeld, MD, MSc; B. Taylor Thompson, MD, PhD; Hermann Wrigge, MD, PhD; Arthur S. Slutsky, MD, MASc; Antonio Pesenti, MD; for the LUNG SAFE Investigators and the ESICM Trials Group

JAMA 2016

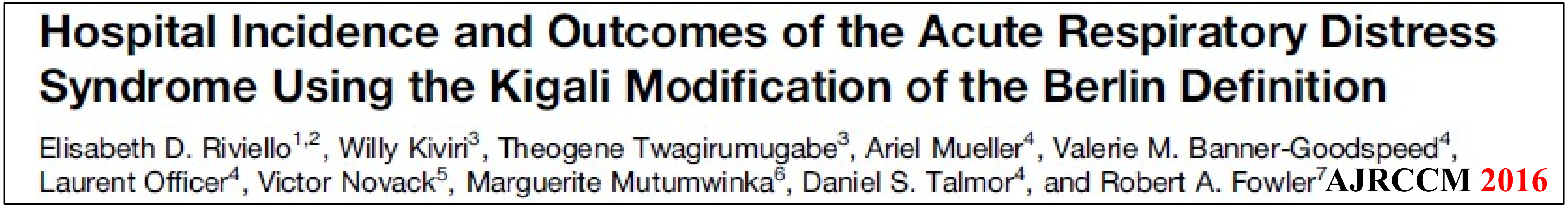
Etude LUNG-SAFE
459 Réanimations dans 50 pays
Eté 2014 sur un mois
Recueil prospectif des données des patients avec VM
29144 patients

Diagnosis of ARDS was frequently delayed.

This syndrome appeared to be underrecognized, undertreated, and associated with a high mortality rate (40%).

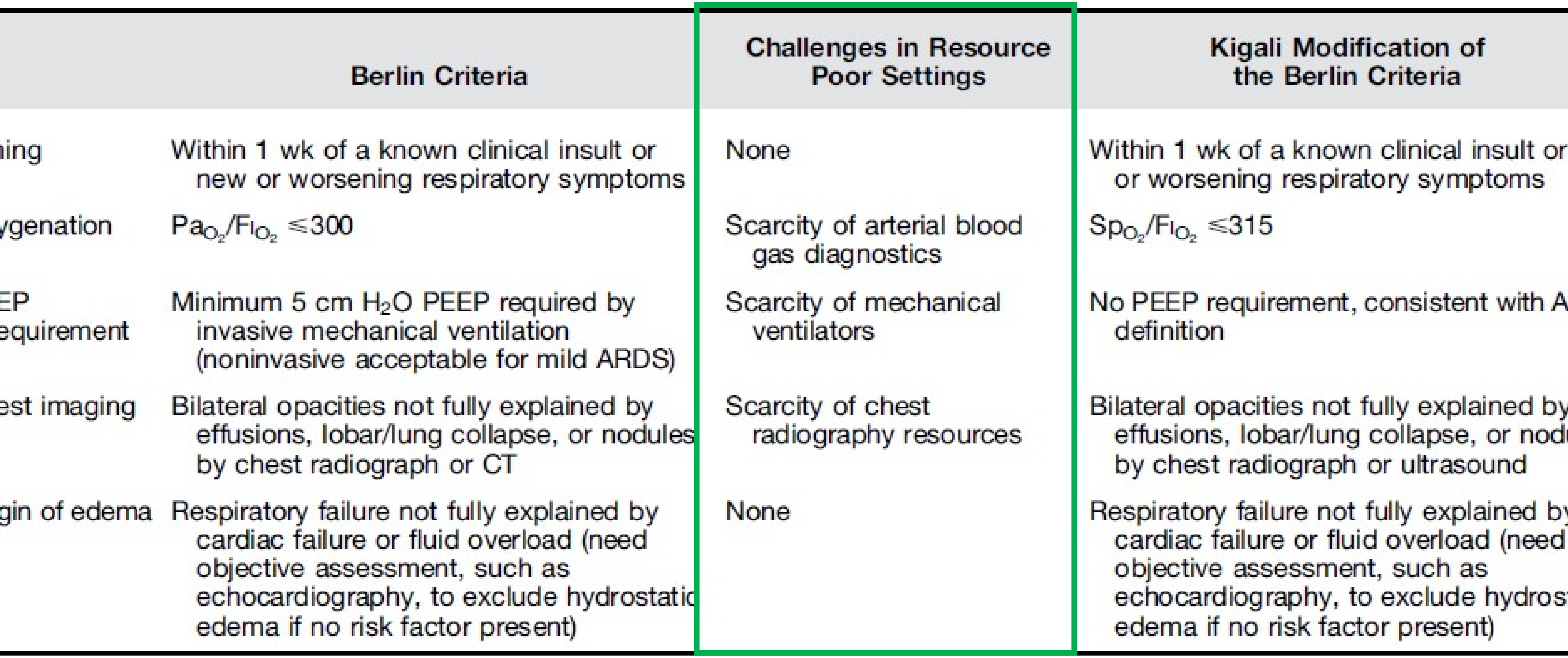
These findings indicate the potential for improvement in management of patients with ARDS.

« Re »Définition de KIGALI 2016



Hospital Incidence and Outcomes of the Acute Respiratory Distress Syndrome Using the Kigali Modification of the Berlin Definition

Elisabeth D. Riviello[1,2], Willy Kiviri[3], Theogene Twagirumugabe[3], Ariel Mueller[4], Valerie M. Banner-Goodspeed[4], Laurent Officer[4], Victor Novack[5], Marguerite Mutumwinka[6], Daniel S. Talmor[4], and Robert A. Fowler[7]**AJRCCM 2016**

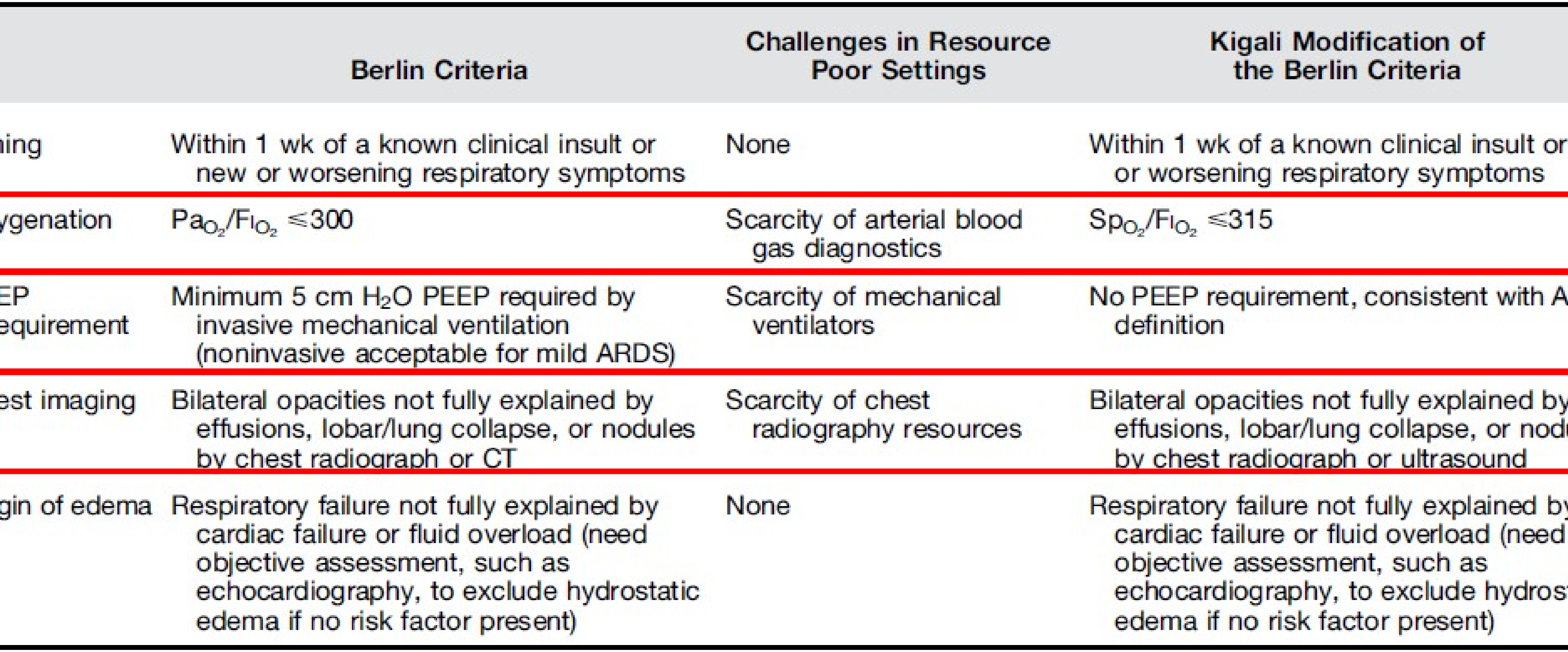


	Berlin Criteria	Challenges in Resource Poor Settings	Kigali Modification of the Berlin Criteria
ing	Within 1 wk of a known clinical insult or new or worsening respiratory symptoms	None	Within 1 wk of a known clinical insult or or worsening respiratory symptoms
ygenation	$Pa_{O_2}/FI_{O_2} \leq 300$	Scarcity of arterial blood gas diagnostics	$Sp_{O_2}/FI_{O_2} \leq 315$
EP equirement	Minimum 5 cm H_2O PEEP required by invasive mechanical ventilation (noninvasive acceptable for mild ARDS)	Scarcity of mechanical ventilators	No PEEP requirement, consistent with A definition
est imaging	Bilateral opacities not fully explained by effusions, lobar/lung collapse, or nodules by chest radiograph or CT	Scarcity of chest radiography resources	Bilateral opacities not fully explained by effusions, lobar/lung collapse, or nodu by chest radiograph or ultrasound
gin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload (need objective assessment, such as echocardiography, to exclude hydrostatic edema if no risk factor present)	None	Respiratory failure not fully explained by cardiac failure or fluid overload (need objective assessment, such as echocardiography, to exclude hydros edema if no risk factor present)

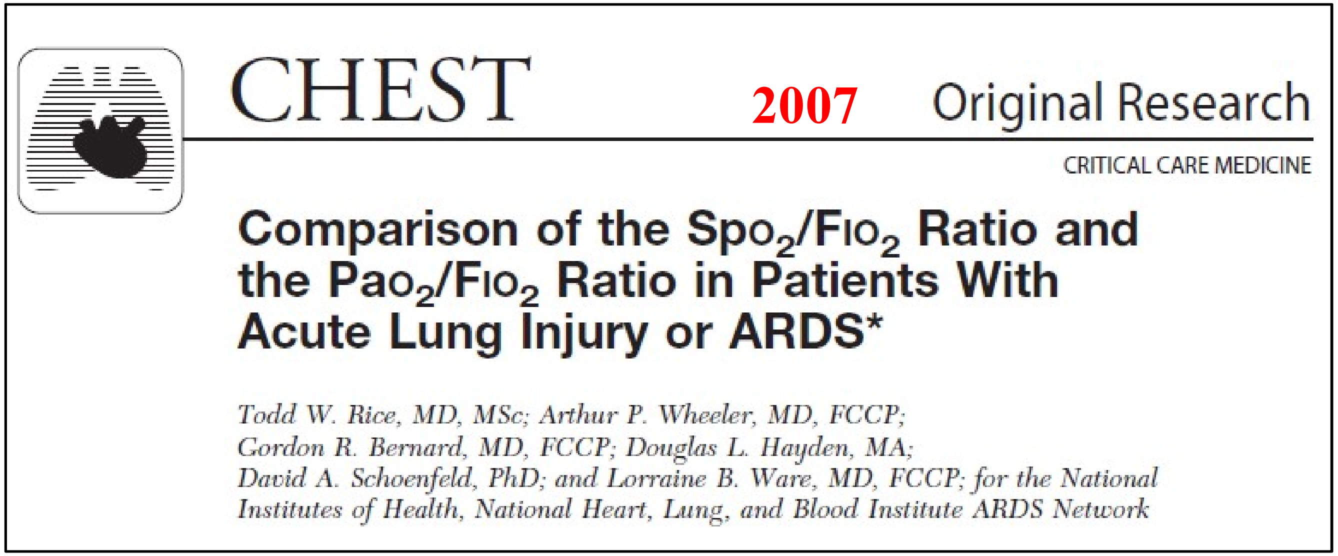
Hospital Incidence and Outcomes of the Acute Respiratory Distress Syndrome Using the Kigali Modification of the Berlin Definition

Elisabeth D. Riviello[1,2], Willy Kiviri[3], Theogene Twagirumugabe[3], Ariel Mueller[4], Valerie M. Banner-Goodspeed[4], Laurent Officer[4], Victor Novack[5], Marguerite Mutumwinka[6], Daniel S. Talmor[4], and Robert A. Fowler[7]

AJRCCM 2016



	Berlin Criteria	Challenges in Resource Poor Settings	Kigali Modification of the Berlin Criteria
ing	Within 1 wk of a known clinical insult or new or worsening respiratory symptoms	None	Within 1 wk of a known clinical insult or or worsening respiratory symptoms
ygenation	$Pa_{O_2}/FI_{O_2} \leq 300$	Scarcity of arterial blood gas diagnostics	$Sp_{O_2}/FI_{O_2} \leq 315$
EP equirement	Minimum 5 cm H_2O PEEP required by invasive mechanical ventilation (noninvasive acceptable for mild ARDS)	Scarcity of mechanical ventilators	No PEEP requirement, consistent with A definition
est imaging	Bilateral opacities not fully explained by effusions, lobar/lung collapse, or nodules by chest radiograph or CT	Scarcity of chest radiography resources	Bilateral opacities not fully explained by effusions, lobar/lung collapse, or nodu by chest radiograph or ultrasound
gin of edema	Respiratory failure not fully explained by cardiac failure or fluid overload (need objective assessment, such as echocardiography, to exclude hydrostatic edema if no risk factor present)	None	Respiratory failure not fully explained by cardiac failure or fluid overload (need objective assessment, such as echocardiography, to exclude hydros edema if no risk factor present)

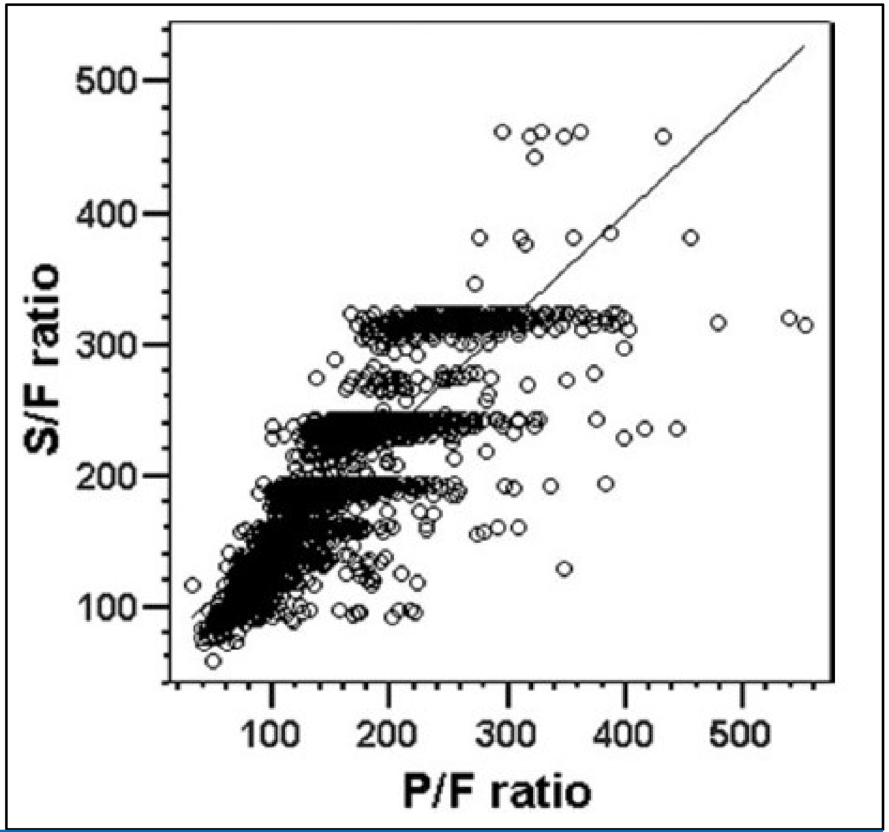


CHEST 2007 Original Research

CRITICAL CARE MEDICINE

Comparison of the SpO_2/FIO_2 Ratio and the PaO_2/FIO_2 Ratio in Patients With Acute Lung Injury or ARDS*

Todd W. Rice, MD, MSc; Arthur P. Wheeler, MD, FCCP; Gordon R. Bernard, MD, FCCP; Douglas L. Hayden, MA; David A. Schoenfeld, PhD; and Lorraine B. Ware, MD, FCCP; for the National Institutes of Health, National Heart, Lung, and Blood Institute ARDS Network



S/F ratios correlate with P/F ratios.

S/F ratios of 235 and 315 correlate with P/F ratios of 200 and 300, respectively, for diagnosing and following up patients with ALI and ARDS.

Pulse oximetry for the diagnosis and management of acute respiratory distress syndrome

Lancet Respir Med 2022;
10: 1086–98

Katherine D Wick, Michael A Matthay, Lorraine B Ware

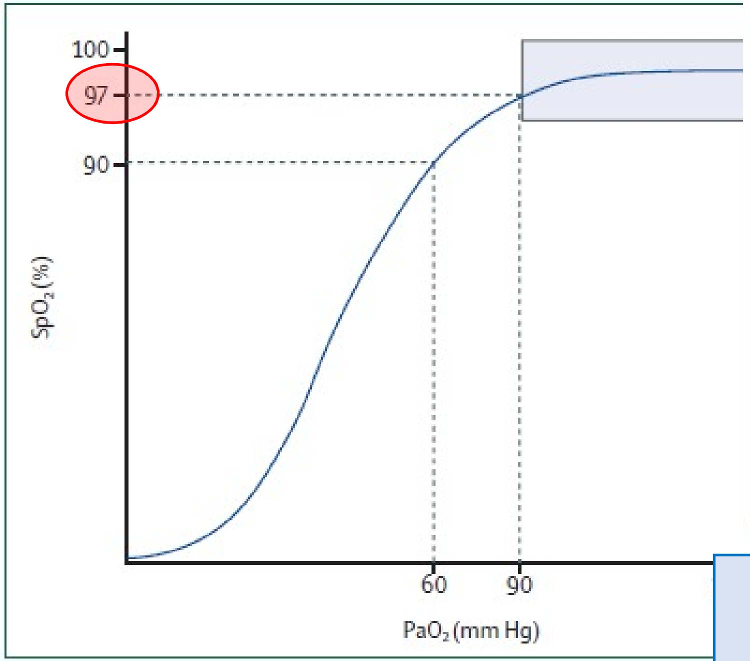
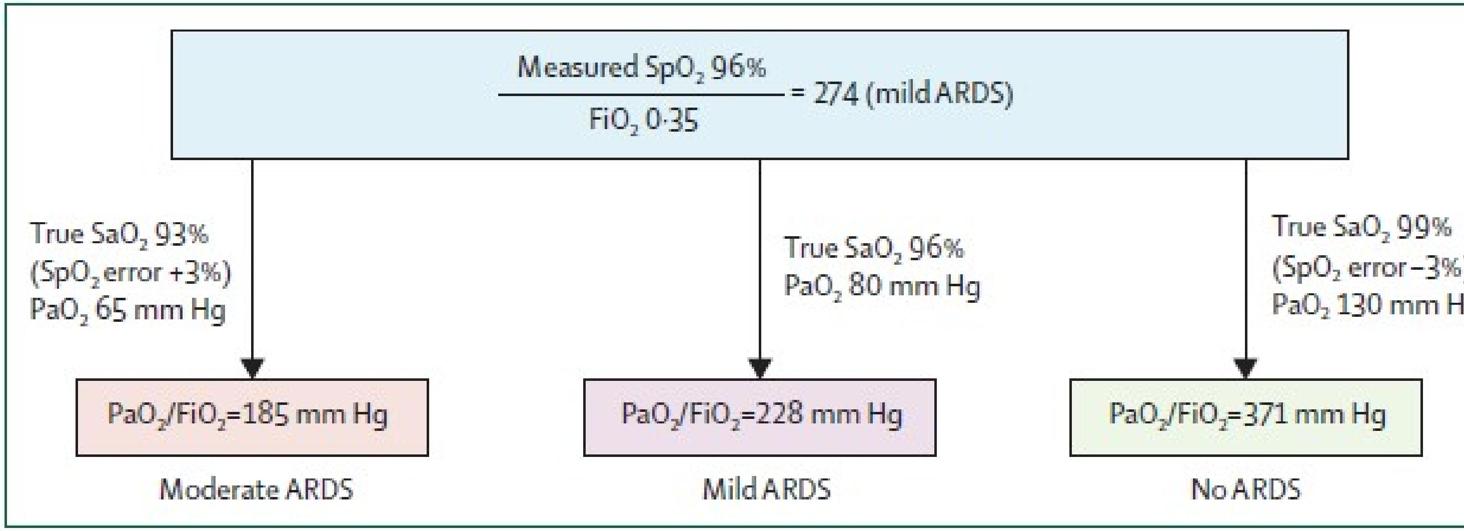


Figure 1: Oxyhaemoglobin dissociation curve



$$\frac{\text{Measured } SpO_2 \text{ } 96\%}{FiO_2 \text{ } 0{\cdot}35} = 274 \text{ (mild ARDS)}$$

True SaO_2 93%
(SpO_2 error +3%)
PaO_2 65 mm Hg

PaO_2/FiO_2=185 mm Hg

Moderate ARDS

True SaO_2 96%
PaO_2 80 mm Hg

PaO_2/FiO_2=228 mm Hg

Mild ARDS

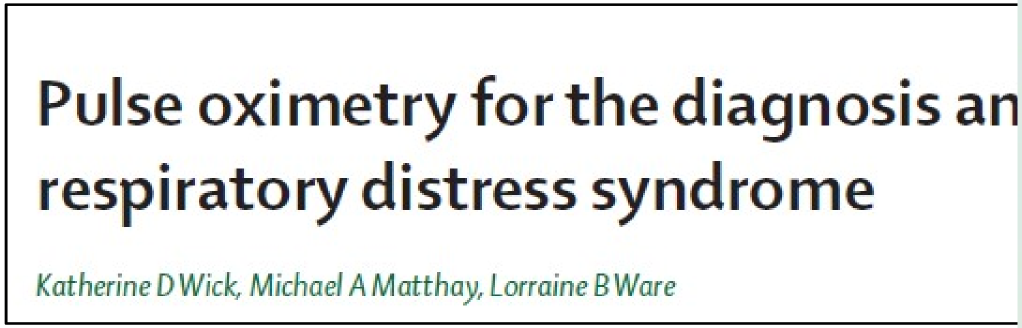
True SaO_2 99%
(SpO_2 error −3%
PaO_2 130 mm H

PaO_2/FiO_2=371 mm Hg

No ARDS

SDRA : SpO2/FiO2 <315

SpO2 <97%



Pulse oximetry for the diagnosis an respiratory distress syndrome

Katherine D Wick, Michael A Matthay, Lorraine B Ware

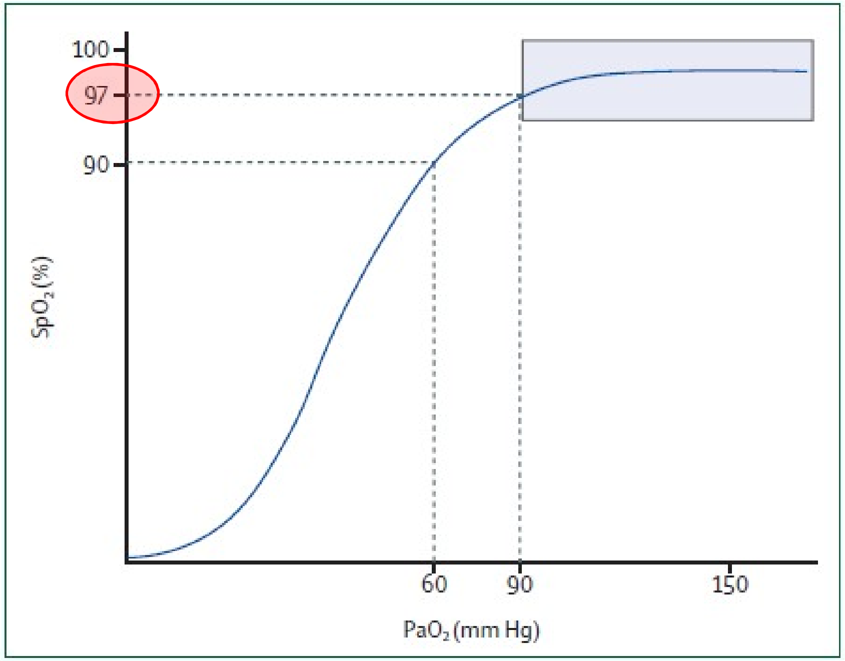
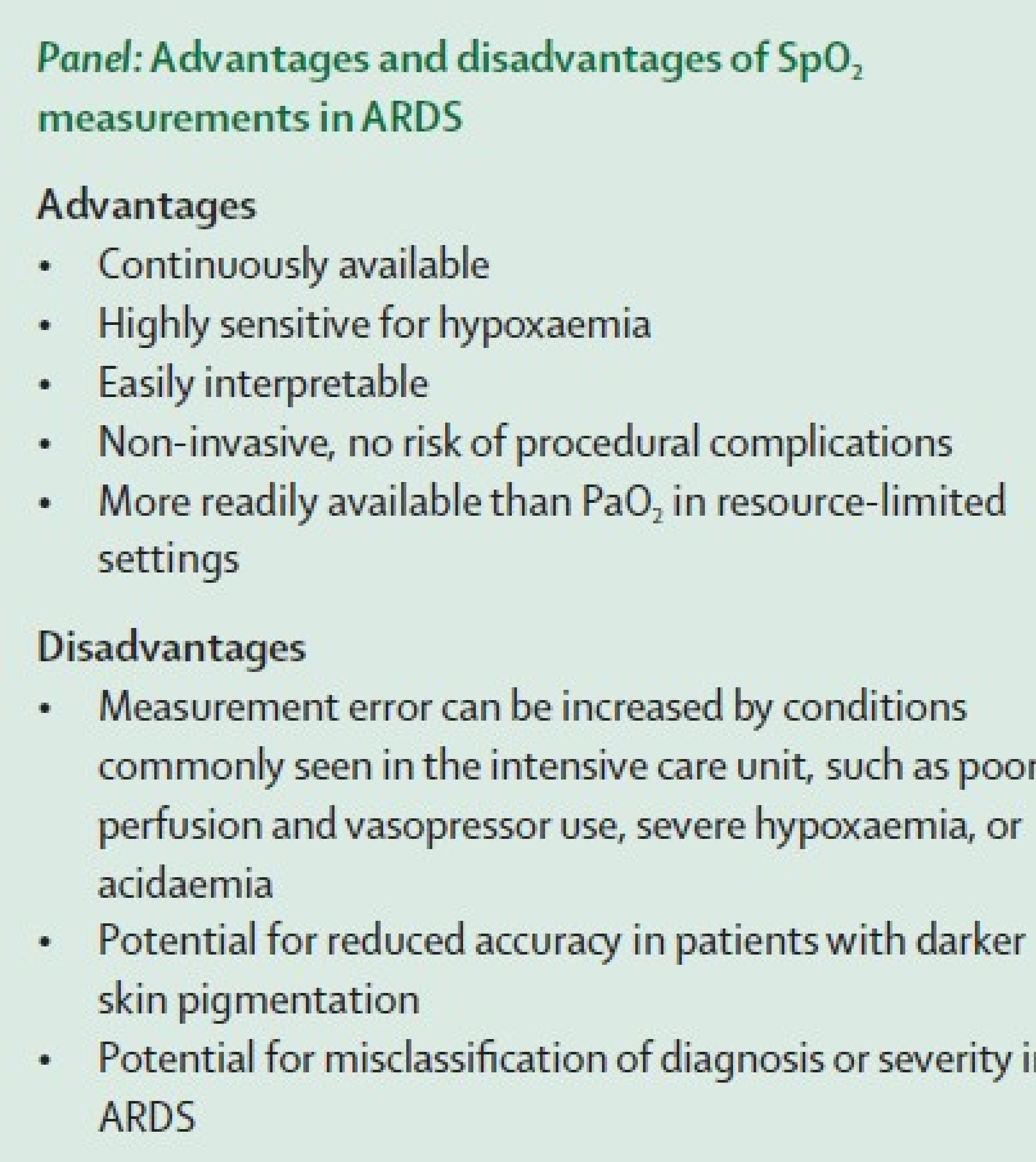


Figure 1: **Oxyhaemoglobin dissociation curve**



Panel: Advantages and disadvantages of SpO_2 measurements in ARDS

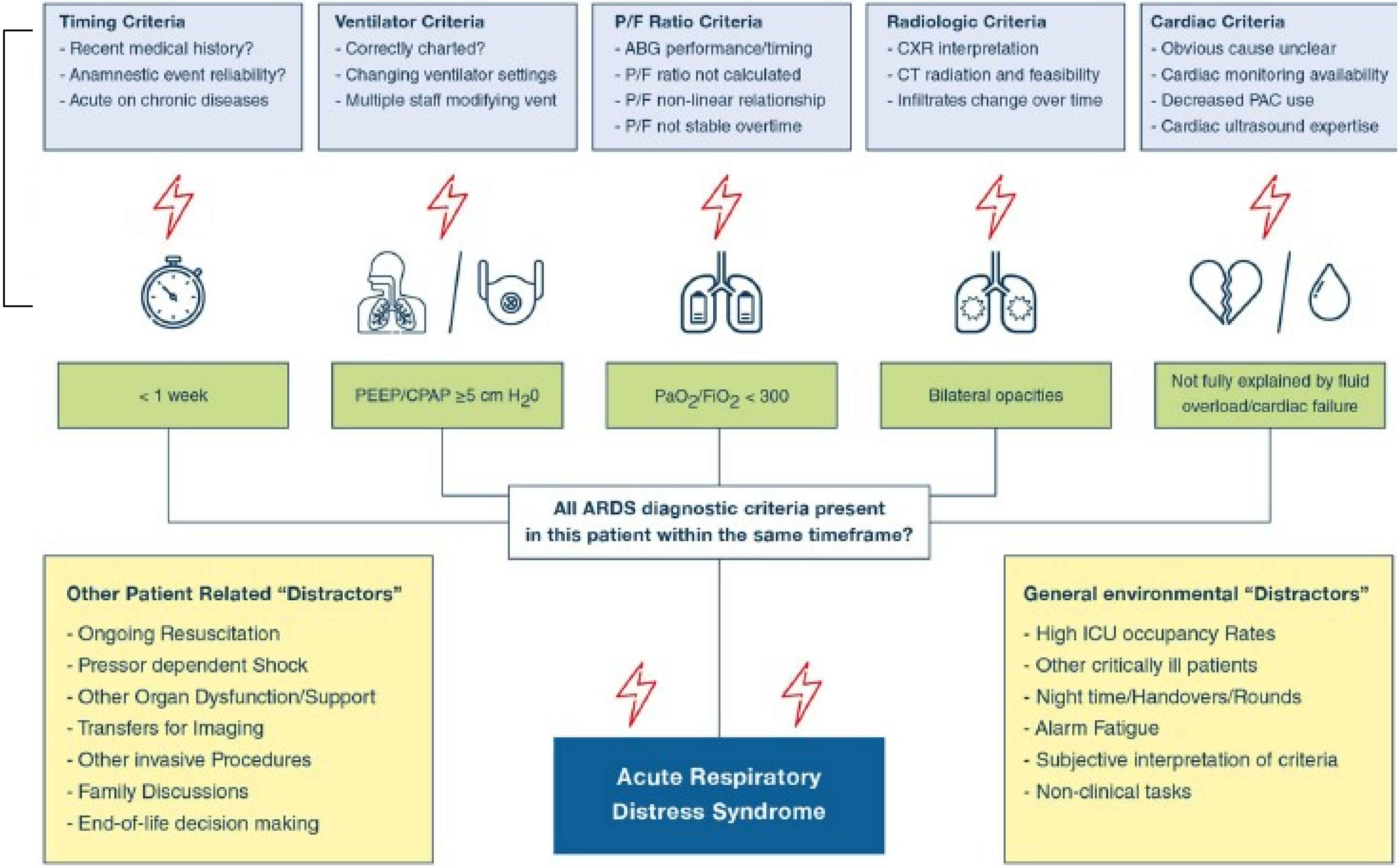
Advantages

- Continuously available
- Highly sensitive for hypoxaemia
- Easily interpretable
- Non-invasive, no risk of procedural complications
- More readily available than PaO_2 in resource-limited settings

Disadvantages

- Measurement error can be increased by conditions commonly seen in the intensive care unit, such as poor perfusion and vasopressor use, severe hypoxaemia, or acidaemia
- Potential for reduced accuracy in patients with darker skin pigmentation
- Potential for misclassification of diagnosis or severity i ARDS

Encore pas d’actualisation de la définition de BERLIN….



Timing Criteria
- Recent medical history?
- Anamnestic event reliability?
- Acute on chronic diseases

Ventilator Criteria
- Correctly charted?
- Changing ventilator settings
- Multiple staff modifying vent

P/F Ratio Criteria
- ABG performance/timing
- P/F ratio not calculated
- P/F non-linear relationship
- P/F not stable overtime

Radiologic Criteria
- CXR interpretation
- CT radiation and feasibility
- Infiltrates change over time

Cardiac Criteria
- Obvious cause unclear
- Cardiac monitoring availability
- Decreased PAC use
- Cardiac ultrasound expertise

< 1 week

PEEP/CPAP ≥5 cm H_20

$PaO_2/FiO_2 < 300$

Bilateral opacities

Not fully explained by fluid overload/cardiac failure

All ARDS diagnostic criteria present in this patient within the same timeframe?

Other Patient Related "Distractors"
- Ongoing Resuscitation
- Pressor dependent Shock
- Other Organ Dysfunction/Support
- Transfers for Imaging
- Other invasive Procedures
- Family Discussions
- End-of-life decision making

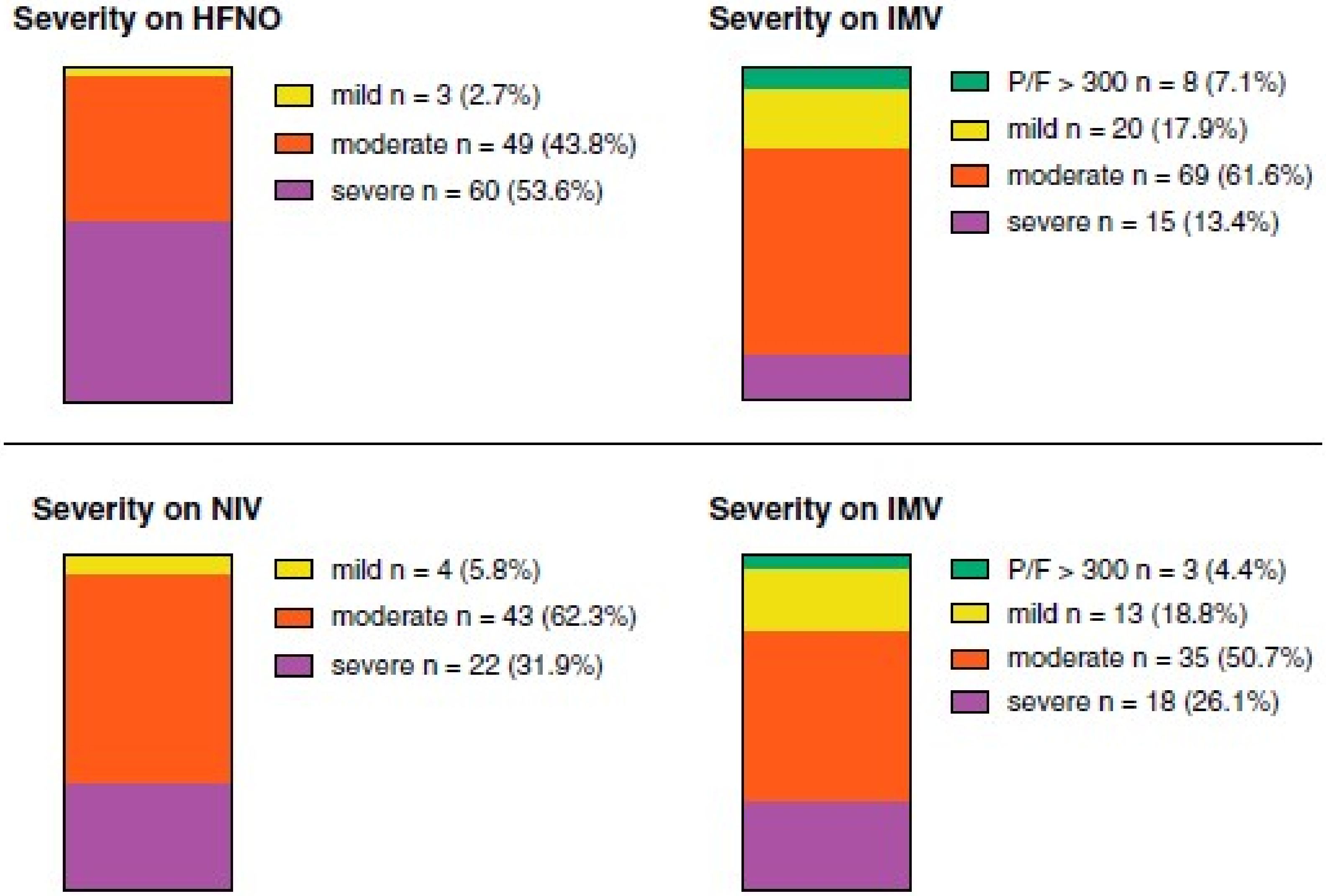
Acute Respiratory Distress Syndrome

General environmental "Distractors"
- High ICU occupancy Rates
- Other critically ill patients
- Night time/Handovers/Rounds
- Alarm Fatigue
- Subjective interpretation of criteria
- Non-clinical tasks

RIGINAL ARTICLE **Ranieri AJCCM 2022**

gh-Flow Nasal Oxygen for Severe Hypoxemia
ygenation Response and Outcome in Patients with COVID-19

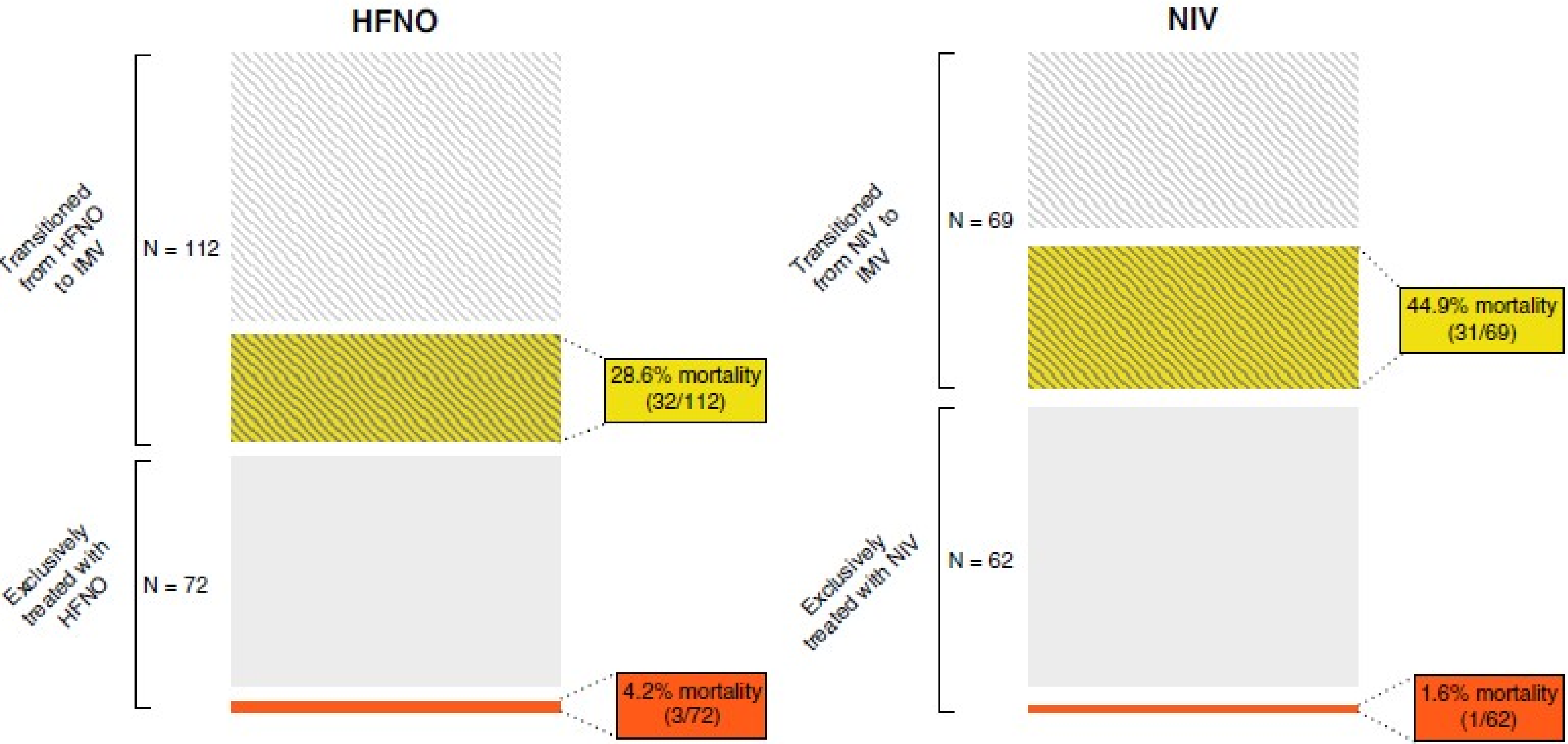
Etude rétrospective multicentric
Patients COVID-19 + critères S
131 avec VNI (51% IOT)
181 avec OHD (61% IOT)



RIGINAL ARTICLE **Ranieri AJCCM 2022**

gh-Flow Nasal Oxygen for Severe Hypoxemia
ygenation Response and Outcome in Patients with COVID-19

Etude rétrospective multicentri
Patients COVID-19 + critères S
131 avec VNI (51% IOT)
181 avec OHD (61% IOT)

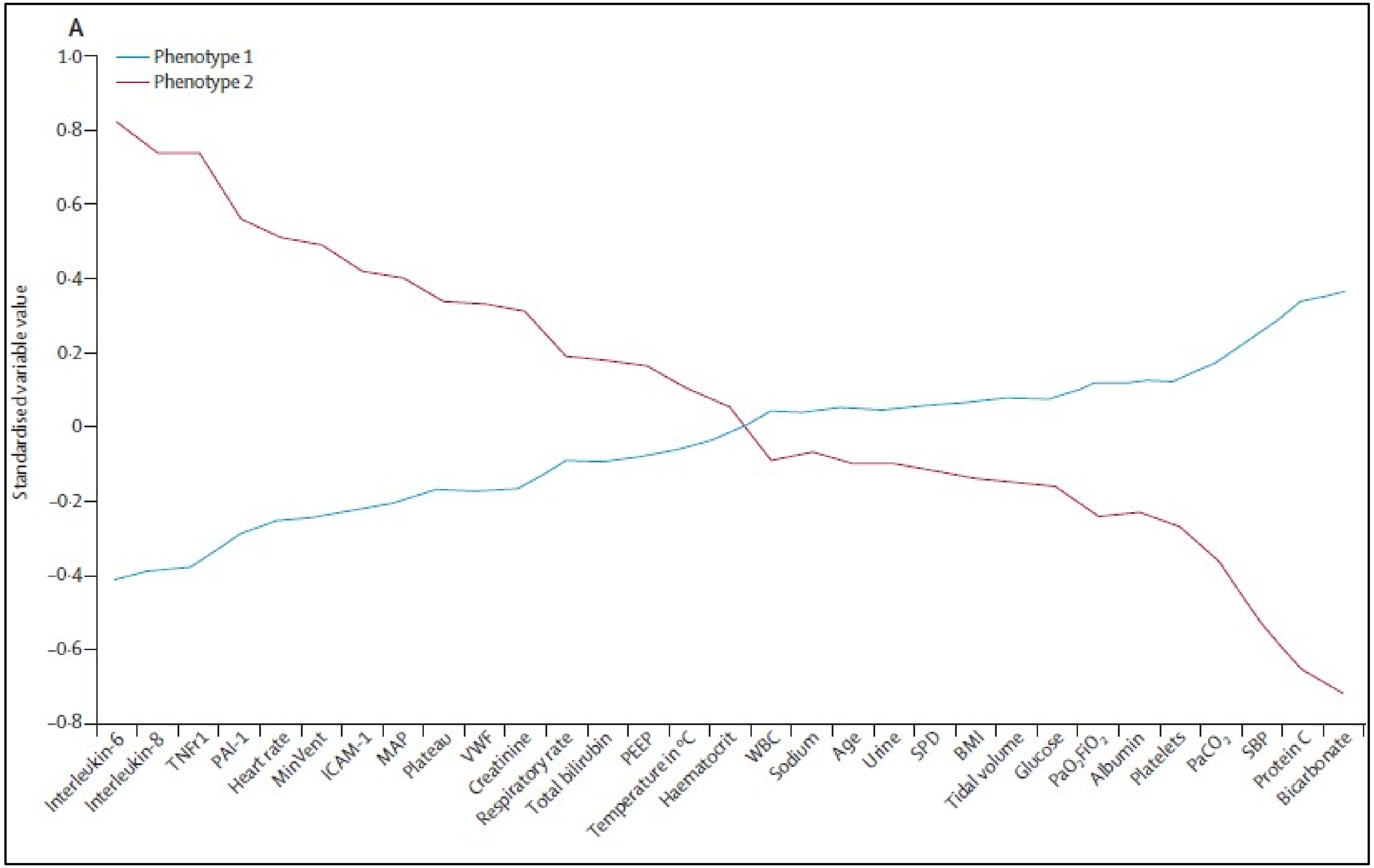


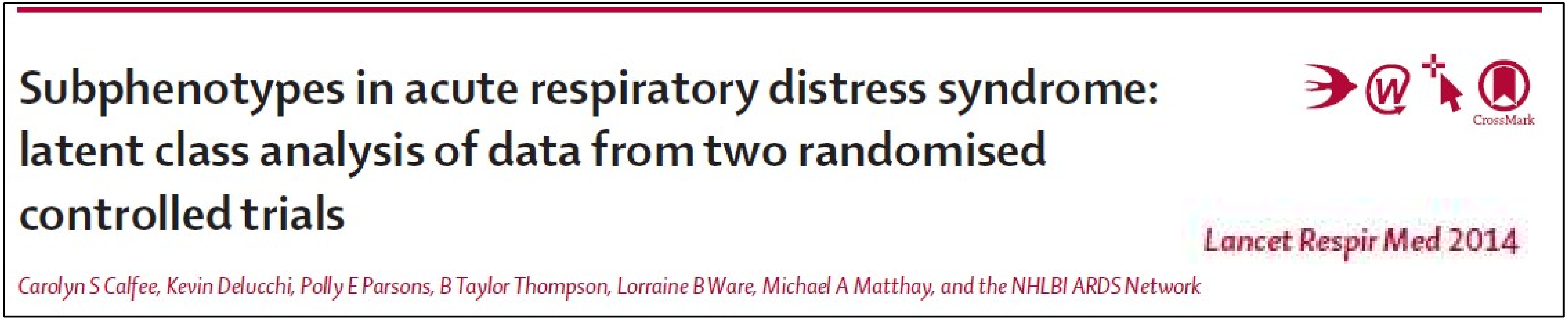
Subphenotypes in acute respiratory distress syndrome: latent class analysis of data from two randomised controlled trials

Lancet Respir Med 2014

Carolyn S Calfee, Kevin Delucchi, Polly E Parsons, B Taylor Thompson, Lorraine B Ware, Michael A Matthay, and the NHLBI ARDS Network

ARMA and ALVEOLI trials
Latent class modeling
Identify subphenotypes using clinical and biological data

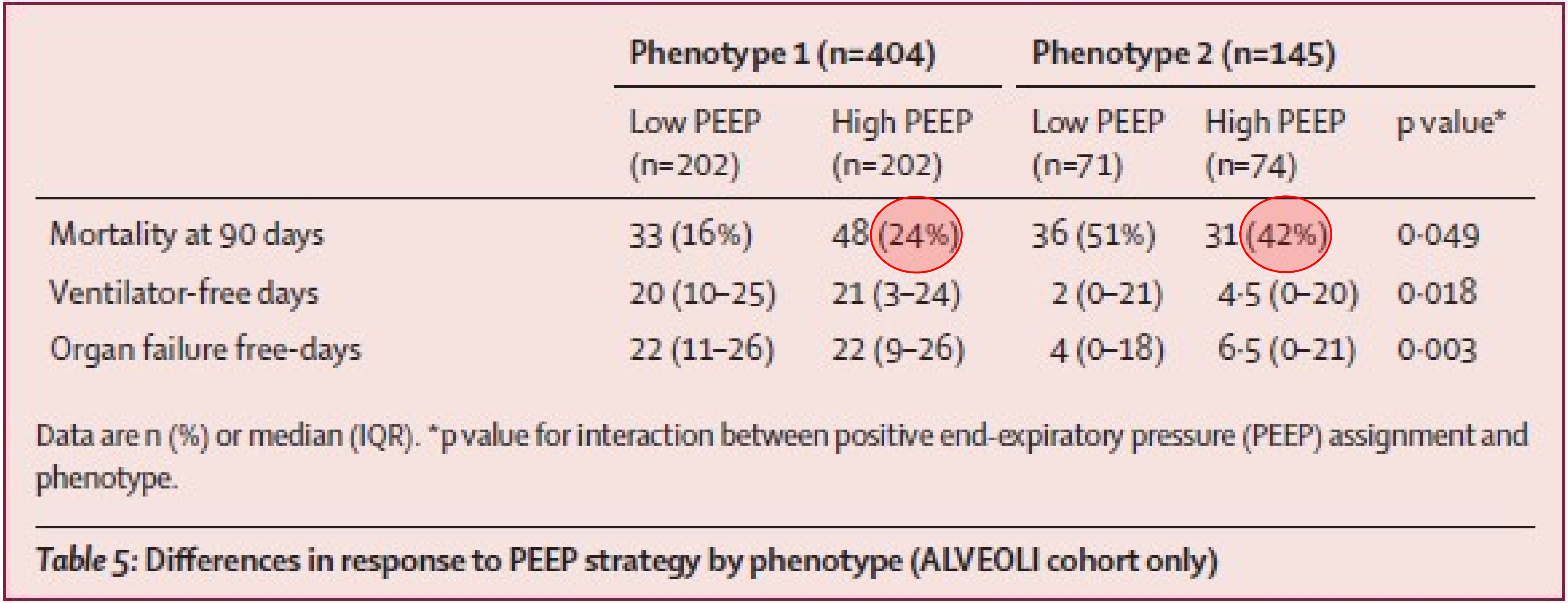




Subphenotypes in acute respiratory distress syndrome: latent class analysis of data from two randomised controlled trials

Lancet Respir Med 2014

Carolyn S Calfee, Kevin Delucchi, Polly E Parsons, B Taylor Thompson, Lorraine B Ware, Michael A Matthay, and the NHLBI ARDS Network



	Phenotype 1 (n=404)		Phenotype 2 (n=145)		
	Low PEEP (n=202)	High PEEP (n=202)	Low PEEP (n=71)	High PEEP (n=74)	p value*
Mortality at 90 days	33 (16%)	48 (24%)	36 (51%)	31 (42%)	0·049
Ventilator-free days	20 (10–25)	21 (3–24)	2 (0–21)	4·5 (0–20)	0·018
Organ failure free-days	22 (11–26)	22 (9–26)	4 (0–18)	6·5 (0–21)	0·003

Data are n (%) or median (IQR). *p value for interaction between positive end-expiratory pressure (PEEP) assignment and phenotype.

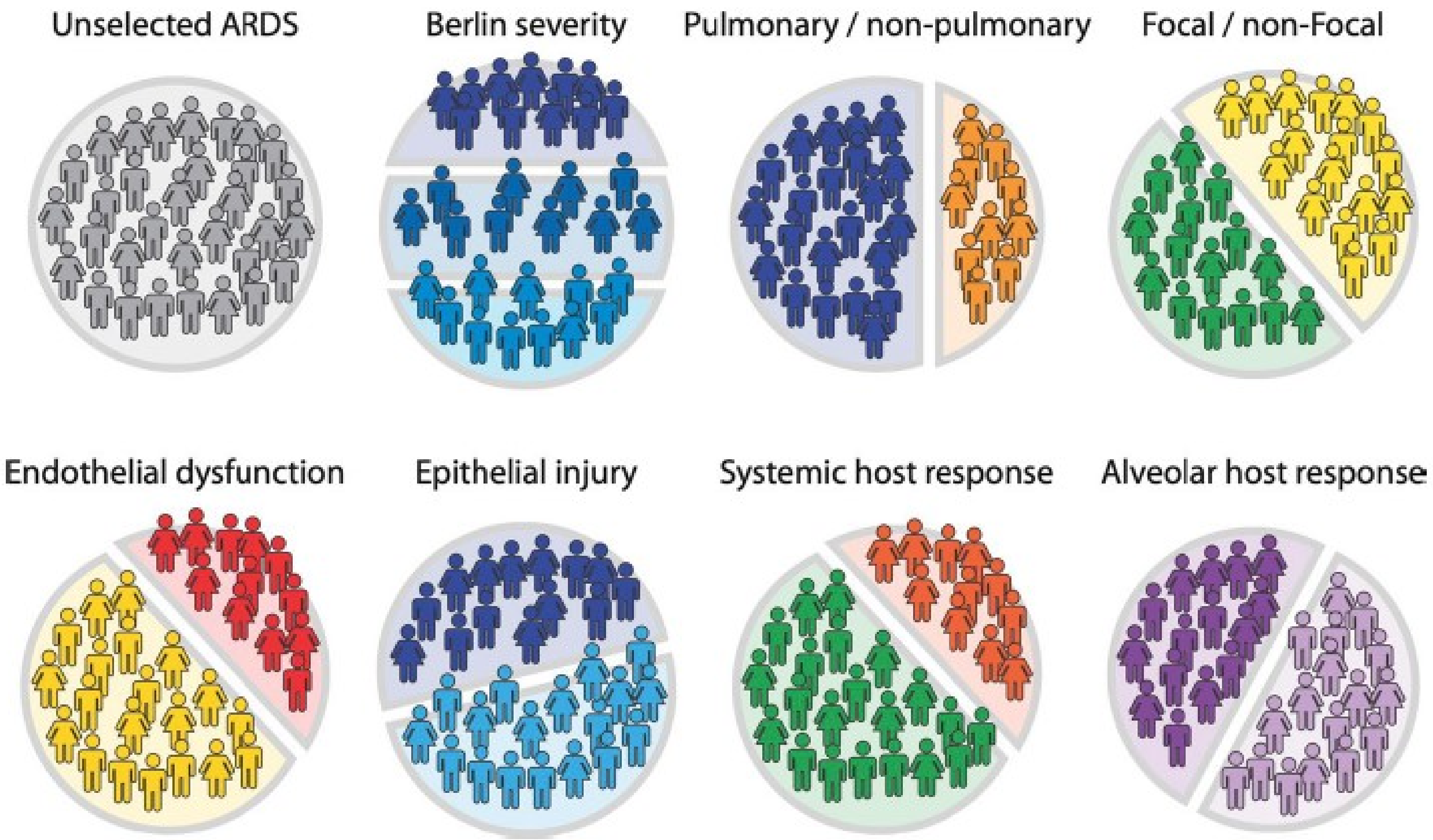
Table 5: Differences in response to PEEP strategy by phenotype (ALVEOLI cohort only)

« Un » SDRA Mais « des » Maladies….

REVIEWS
Open Access

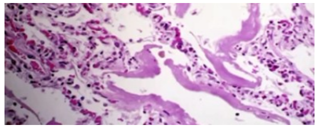
Towards a biological definition of ARDS: are treatable traits the solution?

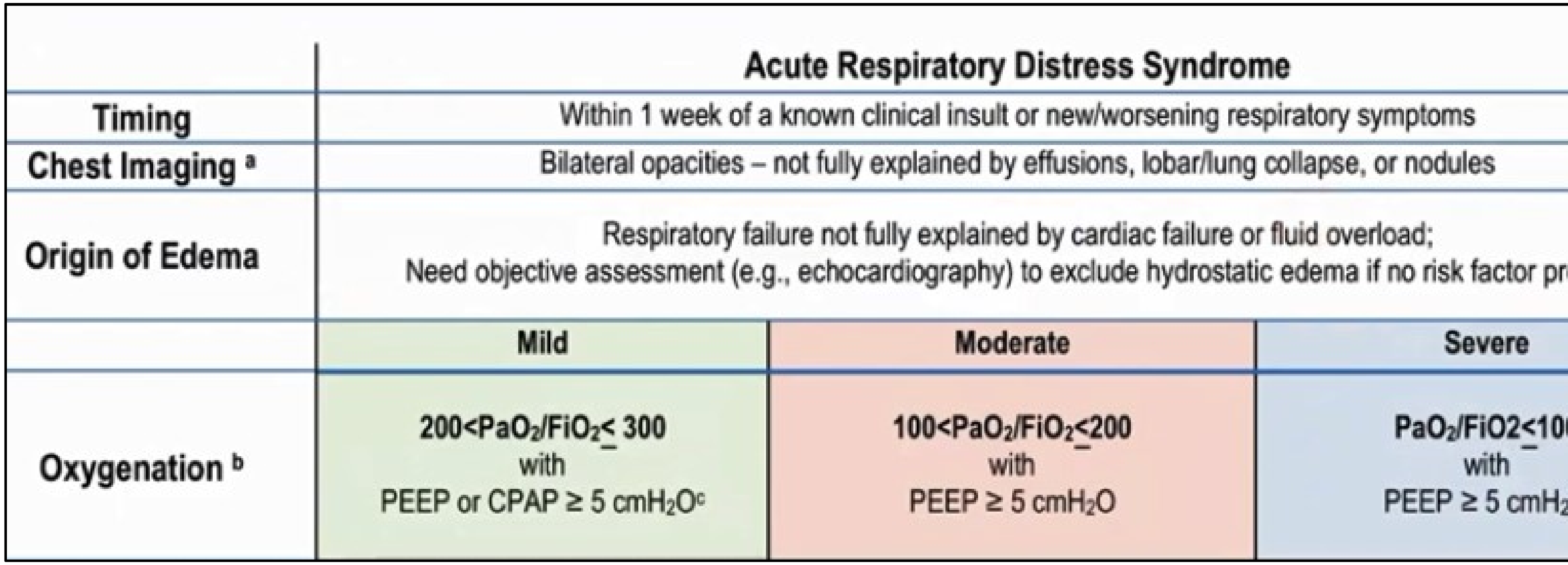
Intensive Care Medicine Experimental (2022) 10:8


Unselected ARDS
Berlin severity
Pulmonary / non-pulmonary
Focal / non-Focal
Endothelial dysfunction
Epithelial injury
Systemic host response
Alveolar host response

Définition de BERLIN 2012

- **Atteinte pulmonaire inflammatoire aigue et diffuse**
- **Augmentation de la perméabilité capillaire, du poids pulmonaire et perte du tissu pulmonaire aéré**
- **Clinique : hypoxémie, opacités bilatérales, baisse de la compliance**
- **Atteinte alvéolaire diffuse**



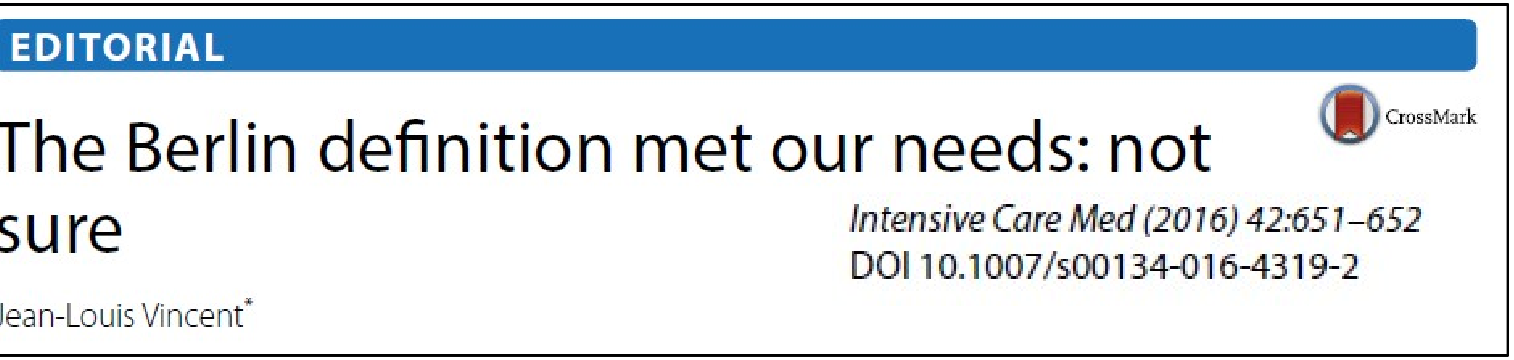


	Acute Respiratory Distress Syndrome		
Timing	Within 1 week of a known clinical insult or new/worsening respiratory symptoms		
Chest Imaging [a]	Bilateral opacities – not fully explained by effusions, lobar/lung collapse, or nodules		
Origin of Edema	Respiratory failure not fully explained by cardiac failure or fluid overload; Need objective assessment (e.g., echocardiography) to exclude hydrostatic edema if no risk factor pr		
	Mild	Moderate	Severe
Oxygenation [b]	$200<PaO_2/FiO_2\leq 300$ with PEEP or CPAP ≥ 5 cmH_2O^c	$100<PaO_2/FiO_2\leq 200$ with PEEP ≥ 5 cmH_2O	$PaO_2/FiO2\leq 10$ with PEEP ≥ 5 cmH_2

Une nouvelle définition: Pourquoi ????

Une nouvelle définition : Pourquoi???

- Place de la définition du SDRA dans les pays aux **ressources sanitaires limités ?**
- Les patients en **VS sous OHD** et selon quels **réglages (FiO2, débit)?**
- Place du **SpO2/FiO2 (degré d'hypoxémie?)** comme alternative au PaO2/FiO2?
- Place de **l'échographie thoracique** pour évaluer l'atteinte parenchymateuse pulmonaire ?
- Place des **marqueurs biologiques et différents phénotypes** de SDRA?

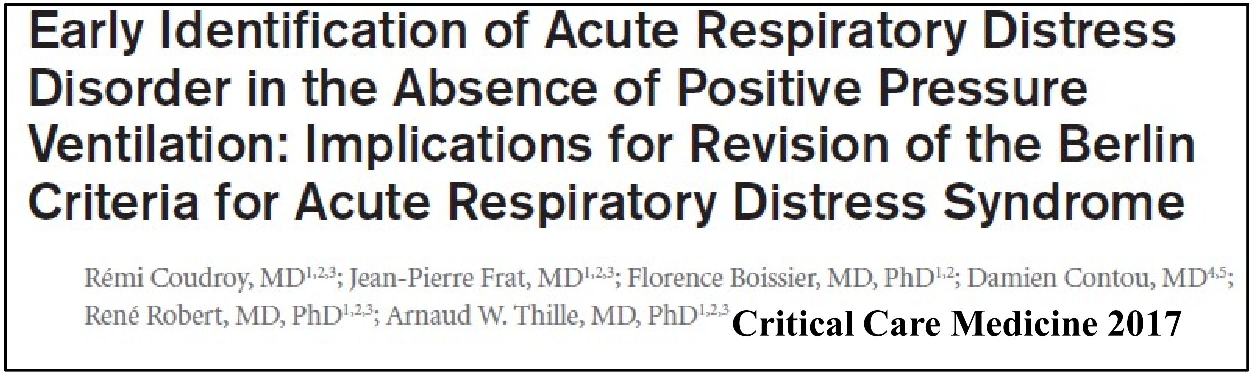


EDITORIAL

The Berlin definition met our needs: not sure

Intensive Care Med (2016) 42:651–652
DOI 10.1007/s00134-016-4319-2

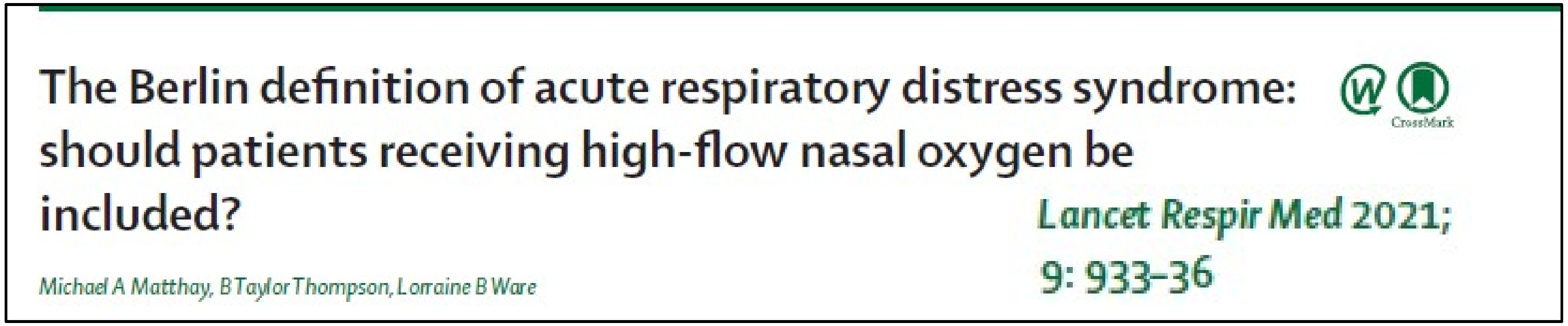
ean-Louis Vincent*



Early Identification of Acute Respiratory Distress Disorder in the Absence of Positive Pressure Ventilation: Implications for Revision of the Berlin Criteria for Acute Respiratory Distress Syndrome

Rémi Coudroy, MD[1,2,3]; Jean-Pierre Frat, MD[1,2,3]; Florence Boissier, MD, PhD[1,2]; Damien Contou, MD[4,5]; René Robert, MD, PhD[1,2,3]; Arnaud W. Thille, MD, PhD[1,2,3]

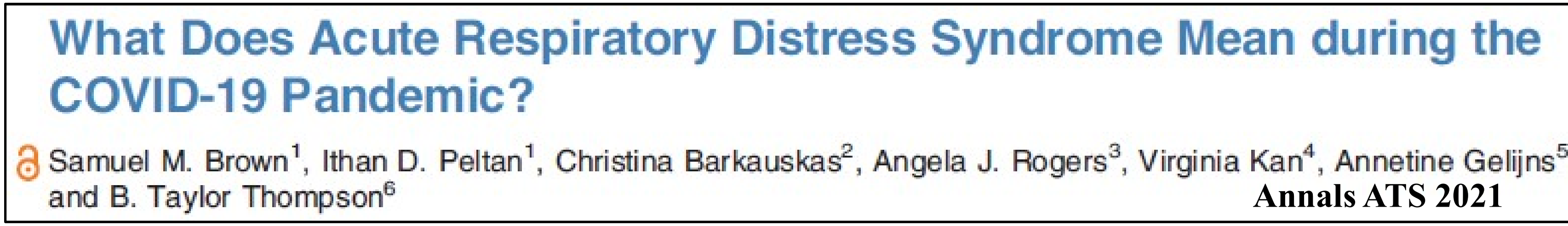
Critical Care Medicine 2017



The Berlin definition of acute respiratory distress syndrome: should patients receiving high-flow nasal oxygen be included?

Lancet Respir Med 2021; 9: 933–36

Michael A Matthay, B Taylor Thompson, Lorraine B Ware



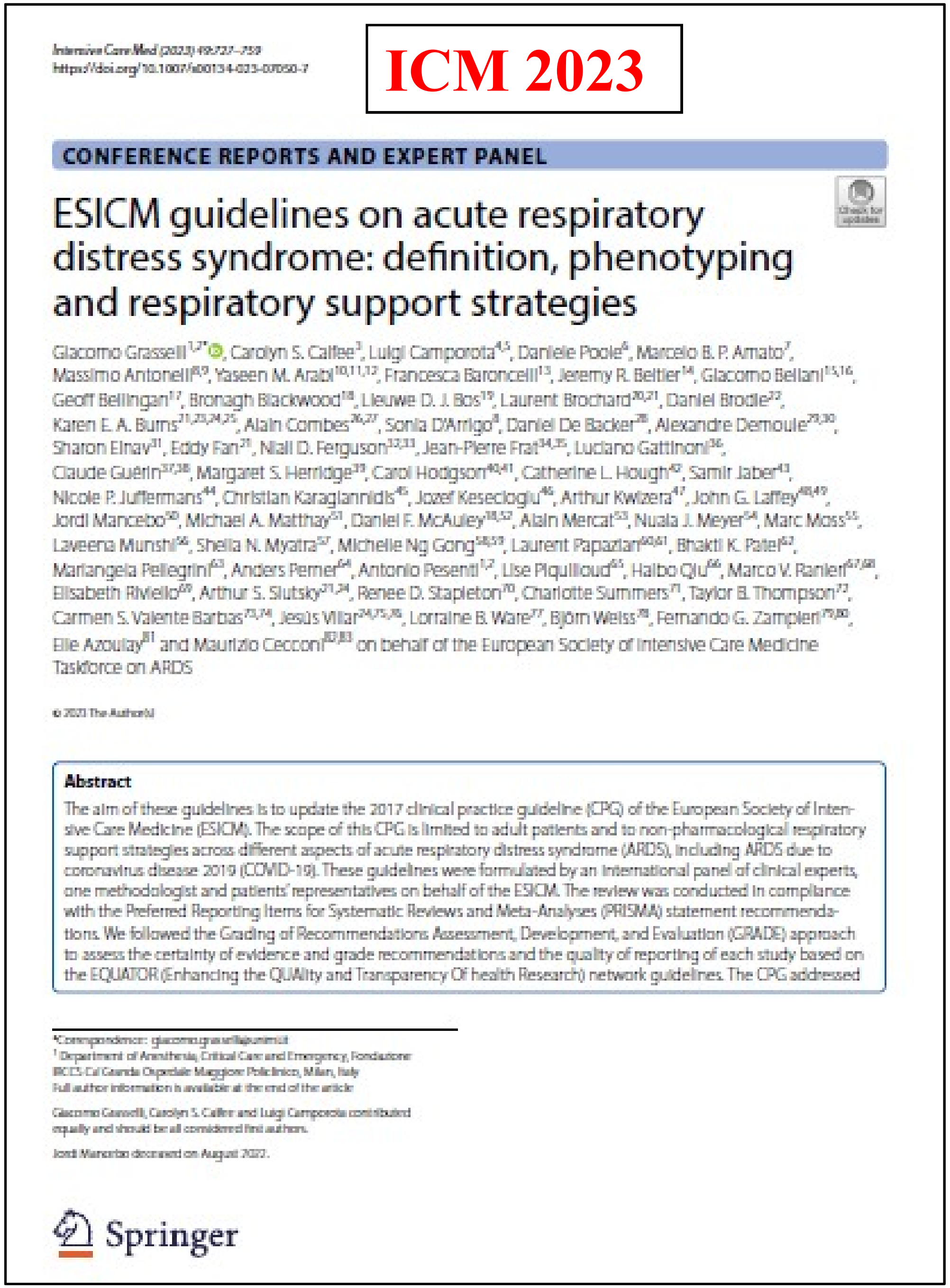
What Does Acute Respiratory Distress Syndrome Mean during the COVID-19 Pandemic?

Samuel M. Brown[1], Ithan D. Peltan[1], Christina Barkauskas[2], Angela J. Rogers[3], Virginia Kan[4], Annetine Gelijns[5] and B. Taylor Thompson[6]

Annals ATS 2021

Et donc... la nouvelle Définition

ICM 2023



Intensive Care Med (2023) 49:727–759
https://doi.org/10.1007/s00134-023-07050-7

CONFERENCE REPORTS AND EXPERT PANEL

ESICM guidelines on acute respiratory distress syndrome: definition, phenotyping and respiratory support strategies

Giacomo Grasselli[1,2*], Carolyn S. Calfee[3], Luigi Camporota[4,5], Daniele Poole[6], Marcelo B. P. Amato[7], Massimo Antonelli[8,9], Yaseen M. Arabi[10,11,12], Francesca Baroncelli[13], Jeremy R. Beitler[14], Giacomo Bellani[15,16], Geoff Bellingan[17], Bronagh Blackwood[18], Lieuwe D. J. Bos[19], Laurent Brochard[20,21], Daniel Brodie[22], Karen E. A. Burns[21,23,24,25], Alain Combes[26,27], Sonia D'Arrigo[8], Daniel De Backer[28], Alexandre Demoule[29,30], Sharon Einav[31], Eddy Fan[21], Niall D. Ferguson[32,33], Jean-Pierre Frat[34,35], Luciano Gattinoni[36], Claude Guérin[37,38], Margaret S. Herridge[39], Carol Hodgson[40,41], Catherine L. Hough[42], Samir Jaber[43], Nicole P. Juffermans[44], Christian Karagiannidis[45], Jozef Kesecioglu[46], Arthur Kwizera[47], John G. Laffey[48,49], Jordi Mancebo[50], Michael A. Matthay[51], Daniel F. McAuley[18,52], Alain Mercat[53], Nuala J. Meyer[54], Marc Moss[55], Laveena Munshi[56], Sheila N. Myatra[57], Michelle Ng Gong[58,59], Laurent Papazian[60,61], Bhakti K. Patel[62], Mariangela Pellegrini[63], Anders Perner[64], Antonio Pesenti[1,2], Lise Piquilloud[65], Haibo Qiu[66], Marco V. Ranieri[67,68], Elisabeth Riviello[69], Arthur S. Slutsky[21,24], Renee D. Stapleton[70], Charlotte Summers[71], Taylor B. Thompson[72], Carmen S. Valente Barbas[73,74], Jesús Villar[24,75,76], Lorraine B. Ware[77], Björn Weiss[78], Fernando G. Zampieri[79,80], Elie Azoulay[81] and Maurizio Cecconi[82,83] on behalf of the European Society of Intensive Care Medicine Taskforce on ARDS

© 2023 The Author(s)

Abstract

The aim of these guidelines is to update the 2017 clinical practice guideline (CPG) of the European Society of Intensive Care Medicine (ESICM). The scope of this CPG is limited to adult patients and to non-pharmacological respiratory support strategies across different aspects of acute respiratory distress syndrome (ARDS), including ARDS due to coronavirus disease 2019 (COVID-19). These guidelines were formulated by an international panel of clinical experts, one methodologist and patients' representatives on behalf of the ESICM. The review was conducted in compliance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement recommendations. We followed the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach to assess the certainty of evidence and grade recommendations and the quality of reporting of each study based on the EQUATOR (Enhancing the QUAlity and Transparency Of health Research) network guidelines. The CPG addressed

---

*Correspondence: giacomo.grasselli@unimi.it

[1] Department of Anesthesia, Critical Care and Emergency, Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico, Milan, Italy

Full author information is available at the end of the article

Giacomo Grasselli, Carolyn S. Calfee and Luigi Camporota contributed equally and should be all considered first authors.

Jordi Mancebo deceased on August 2022.

Springer

AJRCCM 2023

WORKSHOP

A New Global Definition of Acute Respiratory Distress Syndrome

Michael A. Matthay[1,2,3], Yaseen Arabi[5], Alejandro C. Arroliga[6], Gordon Bernard[7], Andrew D. Bersten[8], Laurent J. Brochard[10], Carolyn S. Calfee[1,2,3], Alain Combes[11], Brian M. Daniel[4], Niall D. Ferguson[12,13], Michelle N. Gong[14], Jeffrey E. Gotts[15], Margaret S. Herridge[16], John G. Laffey[17], Kathleen D. Liu[1,2], Flavia R. Machado[18], Thomas R. Martin[19], Danny F. McAuley[20], Alain Mercat[21], Marc Moss[22], Richard A. Mularski[23], Antonio Pesenti[24,25], Haibo Qiu[26], Nagarajan Ramakrishnan[27], V. Marco Ranieri[28], Elisabeth D. Riviello[29], Eileen Rubin[30], Arthur S. Slutsky[10], B. Taylor Thompson[31], Theogene Twagirumugabe[32], Lorraine B. Ware[9], and Katherine D. Wick[33]

Abstract

Background: Since publication of the 2012 Berlin definition of acute respiratory distress syndrome (ARDS), several developments have supported the need for an expansion of the definition, including the use of high-flow nasal oxygen, the expansion of the use of pulse oximetry in place of arterial blood gases, the use of ultrasound for chest imaging, and the need for applicability in resource-limited settings.

Methods: A consensus conference of 32 critical care ARDS experts was convened, had six virtual meetings (June 2021 to March 2022), and subsequently obtained input from members of several critical care societies. The goal was to develop a definition that would *1*) identify patients with the currently accepted conceptual framework for ARDS, *2*) facilitate rapid ARDS diagnosis for clinical care and research, *3*) be applicable in resource-limited settings, *4*) be useful for testing specific therapies, and *5*) be practical for communication to patients and caregivers.

Results: The committee made four main recommendations: *1*) include high-flow nasal oxygen with a minimum flow rate of ≥30 L/min; *2*) use $Pa_{O_2}:FI_{O_2} \le 300$ mm Hg or oxygen saturation as measured by pulse oximetry $Sp_{O_2}:FI_{O_2} \le 315$ (if oxygen saturation as measured by pulse oximetry is ≤97%) to identify hypoxemia; *3*) retain bilateral opacities for imaging criteria but add ultrasound as an imaging modality, especially in resource-limited areas; and *4*) in resource-limited settings, do not require positive end-expiratory pressure, oxygen flow rate, or specific respiratory support devices.

Conclusions: We propose a new global definition of ARDS that builds on the Berlin definition. The recommendations also identify areas for future research, including the need for prospective assessments of the feasibility, reliability, and prognostic validity of the proposed global definition.

Keywords: ARDS; acute lung injury; pulmonary edema

Acute respiratory distress syndrome (ARDS) is a clinical syndrome of acute hypoxemic respiratory failure due to lung inflammation, not caused by cardiogenic pulmonary edema. It was first described in 1967 (1), and in 1988, a more explicit clinical definition quantified the severity of physiologic respiratory impairment (the lung injury score) (2). Since then, the clinical definition of ARDS has been revised, first by an American–European consensus conference convened in 1992 by the American Thoracic Society and the European Society of Intensive Care Medicine (3) and subsequently by the ARDS Definition Task Force convened in Berlin in 2012 by the European Society of Intensive Care Medicine (4, 5). Each revision of the definition was made with the goal of providing a definition that would consistently and accurately identify patients with similar characteristics for clinical care and epidemiological, observational, and interventional research studies. Although the Berlin definition of ARDS was a major step forward, some of i limitations were recognized soon after publication. Specifically, it was recognized that its requirement for noninvasive ventilation (NIV) or invasive ventilation could not be met in settings in which these modalities are not available (6).

In the decade since the Berlin definitio was published, several developments in the management and study of ARDS have

---

(Received in original form March 20, 2023; accepted in final form July 24, 2023)

This article is open access and distributed under the terms of the Creative Commons Attribution Non-Commercial No Derivatives License 4. For commercial usage and reprints, please e-mail Diane Gern (dgern@thoracic.org).

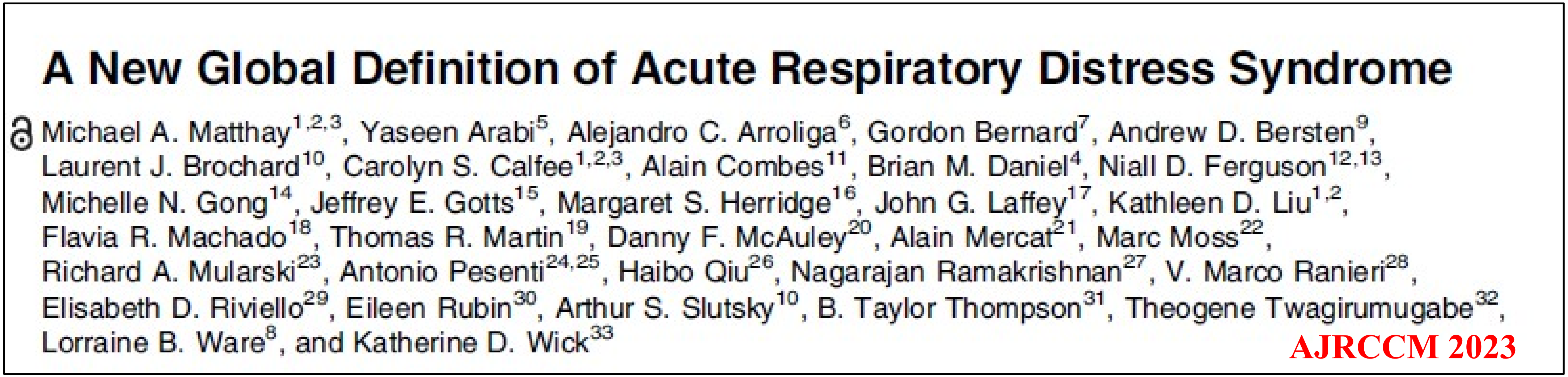
This workshop was not an Official American Thoracic Society Workshop. Therefore, it was not initiated, funded, reviewed, or approved by the leadership of the ATS.

Correspondence and requests for reprints should be addressed to Michael A. Matthay, M.D., Department of Medicine and Anesthesiology, University of California, San Francisco, Parnassus Avenue, M-917, San Francisco, CA 94143. E-mail: michael.matthay@ucsf.edu.

This article has a related editorial and viewpoint.

This article has an online supplement, which is accessible from this issue's table of contents at www.atsjournals.org.

Am J Respir Crit Care Med Vol 209, Iss 1, pp 37–47, Jan 1, 2024
Copyright © 2024 by the American Thoracic Society
Originally Published in Press as DOI: 10.1164/rccm.202303-0558WS on July 24, 2023
Internet address: www.atsjournals.org



A New Global Definition of Acute Respiratory Distress Syndrome

Michael A. Matthay[1,2,3], Yaseen Arabi[5], Alejandro C. Arroliga[6], Gordon Bernard[7], Andrew D. Bersten[9], Laurent J. Brochard[10], Carolyn S. Calfee[1,2,3], Alain Combes[11], Brian M. Daniel[4], Niall D. Ferguson[12,13], Michelle N. Gong[14], Jeffrey E. Gotts[15], Margaret S. Herridge[16], John G. Laffey[17], Kathleen D. Liu[1,2], Flavia R. Machado[18], Thomas R. Martin[19], Danny F. McAuley[20], Alain Mercat[21], Marc Moss[22], Richard A. Mularski[23], Antonio Pesenti[24,25], Haibo Qiu[26], Nagarajan Ramakrishnan[27], V. Marco Ranieri[28], Elisabeth D. Riviello[29], Eileen Rubin[30], Arthur S. Slutsky[10], B. Taylor Thompson[31], Theogene Twagirumugabe[32], Lorraine B. Ware[8], and Katherine D. Wick[33]

AJRCCM 2023

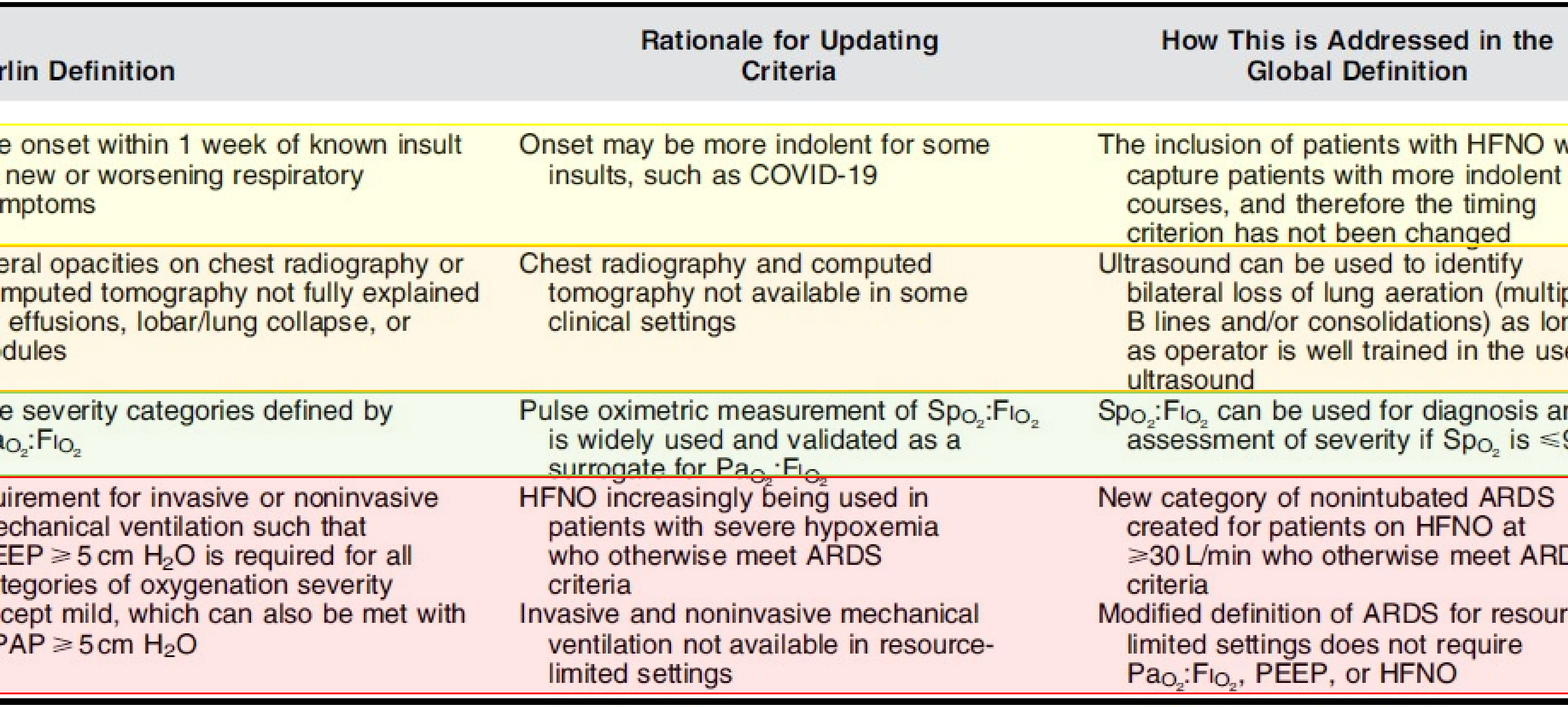
- **32 experts internationaux**
- **Avis et commentaires de 21 sociétés savantes de réanimation**

Conceptual model: ARDS is an acute, diffuse, inflammatory lung injury precipitated by a predisposing risk factor, such as pneumonia, nonpulmonary infection, trauma, transfusion, burn, aspiration, or shock. The resulting injury leads to increased pulmonary vascular and epithelial permeability, lung edema, and gravity-dependent atelectasis, all of which contribute to loss of aerated lung tissue. The clinical hallmarks are arterial hypoxemia and diffuse radiographic opacities associated with increased shunting, increased alveolar dead space, and decreased lung compliance. The clinical presentation is influenced by medical management (position, sedation, paralysis, positive end-expiratory airway pressure, and fluid balance). Histological findings vary and may include intraalveolar edema inflammation, hyaline membrane formation, and alveolar hemorrhage.

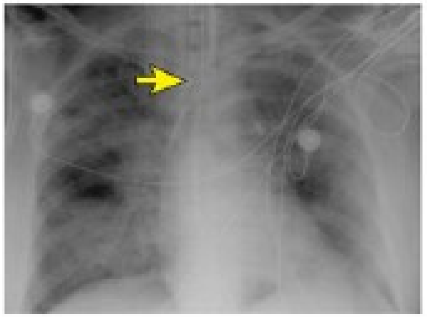
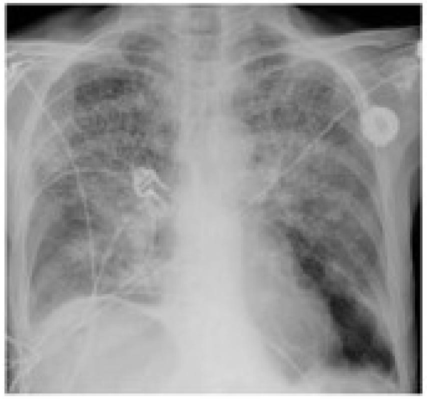
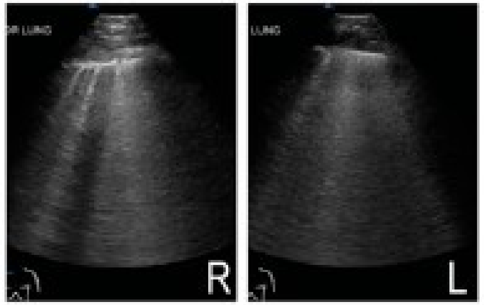
	Criteria That Apply to All ARDS Categories
Risk factors and origin of edema	Precipitated by an acute predisposing risk factor, such as pneumonia, nonpulmonary infection, trauma, transfusion, aspiration, or shock. Pulmonary edema is not *exclusively or primarily* attributable to cardiogenic pulmonary edema/fluid overload, and hypoxemia/gas exchange abnormalities are not primarily attributable to atelectasis. However, ARDS can be diagnosed in the presence of these conditions if a predisposing risk factor for ARDS is also present.
Timing	Acute onset or worsening of hypoxemic respiratory failure within 1 week of the estimated onset of the predisposing risk factor or new or worsening respiratory symptoms.
Chest imaging	Bilateral opacities on chest radiography and computed tomography or bilateral B lines and/or consolidations on ultrasound[*] not fully explained by effusions, atelectasis, or nodules/masses

	Criteria That Apply to Specific ARDS Categories		
	Nonintubated ARDS[†]	Intubated ARDS	Modified Definition for Resource-Limited Settings
Oxygenation[§][||]	$Pa_{O_2}:FI_{O_2} \leq 300$ mm Hg or $Sp_{O_2}:FI_{O_2} \leq 315$ (if $Sp_{O_2} \leq 97\%$) on HFNO with flow of ≥ 30 L/min or NIV/CPAP with at least 5 cm H_2O end-expiratory pressure	Mild[¶]: $200 < Pa_{O_2}:FI_{O_2} \leq 300$ mm Hg or $235 < Sp_{O_2}:FI_{O_2} \leq 315$ (if $Sp_{O_2} \leq 97\%$) Moderate: $100 < Pa_{O_2}:FI_{O_2} \leq 200$ mm Hg or $148 < Sp_{O_2}:FI_{O_2} \leq 235$ (if $Sp_{O_2} \leq 97\%$) Severe: $Pa_{O_2}:FI_{O_2} \leq 100$ mm Hg or $Sp_{O_2}:FI_{O_2} \leq 148$ (if $Sp_{O_2} \leq 97\%$)	$Sp_{O_2}:FI_{O_2} \leq 315$ (if $Sp_{O_2} \leq 97\%$)[†]. Neither positive end-expiratory pressure nor a minimum flow rate of oxygen is required for diagnosis in resource-limited settings.

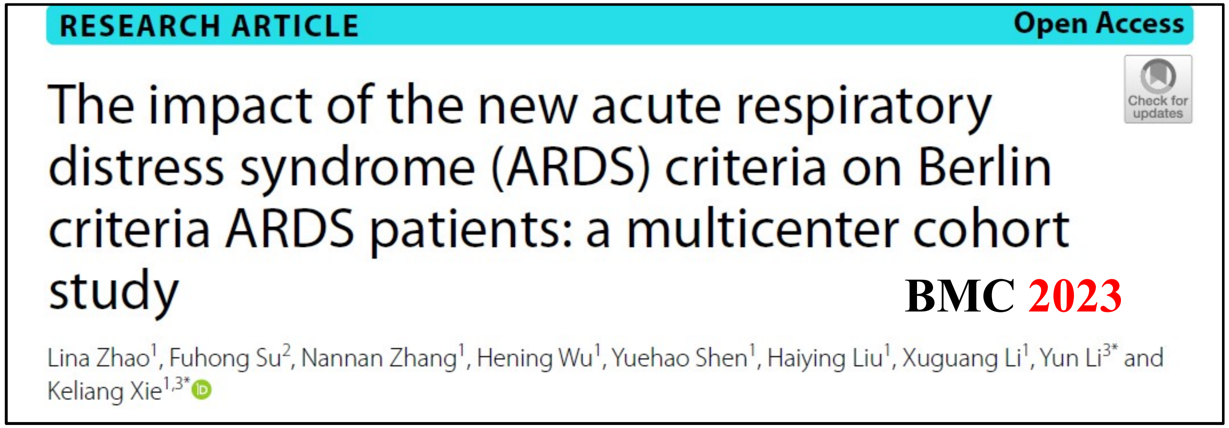
Et donc quelles évolutions ?



rlin Definition	Rationale for Updating Criteria	How This is Addressed in the Global Definition
e onset within 1 week of known insult new or worsening respiratory mptoms	Onset may be more indolent for some insults, such as COVID-19	The inclusion of patients with HFNO w capture patients with more indolent courses, and therefore the timing criterion has not been changed
eral opacities on chest radiography or mputed tomography not fully explained effusions, lobar/lung collapse, or dules	Chest radiography and computed tomography not available in some clinical settings	Ultrasound can be used to identify bilateral loss of lung aeration (multip B lines and/or consolidations) as lor as operator is well trained in the use ultrasound
e severity categories defined by $_{O_2}$:FI_{O_2}	Pulse oximetric measurement of Sp_{O_2}:FI_{O_2} is widely used and validated as a surrogate for Pa_{O_2}:FI_{O_2}	Sp_{O_2}:FI_{O_2} can be used for diagnosis ar assessment of severity if Sp_{O_2} is ≤9
uirement for invasive or noninvasive echanical ventilation such that EEP ≥ 5 cm H_2O is required for all tegories of oxygenation severity cept mild, which can also be met with PAP ≥ 5 cm H_2O	HFNO increasingly being used in patients with severe hypoxemia who otherwise meet ARDS criteria Invasive and noninvasive mechanical ventilation not available in resource-limited settings	New category of nonintubated ARDS created for patients on HFNO at ≥30 L/min who otherwise meet ARD criteria Modified definition of ARDS for resour limited settings does not require Pa_{O_2}:FI_{O_2}, PEEP, or HFNO

Patient Description		Imaging	Oxygenation	ARDS Categorie
	68-year-old M with abdominal sepsis, septic shock, and acute hypoxemic respiratory failure		Mechanically ventilated $\mathrm{Fi}_{\mathrm{O}_2}$ 0.5 $\mathrm{Pa}_{\mathrm{O}_2}$ 75 P/F = 150 mm Hg	**Intubated ARDS** Severity: Moderate *Typical patient included* *prior Berlin definition*
	54-year-old F with history of breast cancer, COVID-19 pneumonia, and worsening shortness of breath for the past 6 days		High-flow nasal oxygen HFNO 40L/min $\mathrm{Fi}_{\mathrm{O}_2}$ 0.80 $\mathrm{Sp}_{\mathrm{O}_2}$ 91% S/F = 114	**Nonintubated ARDS** *New category in Global* *definition*
	39-year-old F with abdominal sepsis and gram-negative bacteremia in a small under-resourced hospital without blood gases, radiography, or mechanical ventilation		Supplemental oxygen by face mask at 15L/min $\mathrm{Fi}_{\mathrm{O}_2}$ 0.6 $\mathrm{Sp}_{\mathrm{O}_2}$ 85% S/F = 142	**ARDS in resource-limited settings** *New category in global* *definition, consistent* *with the Kigali modificat*

Nouvelle définition globale 2023

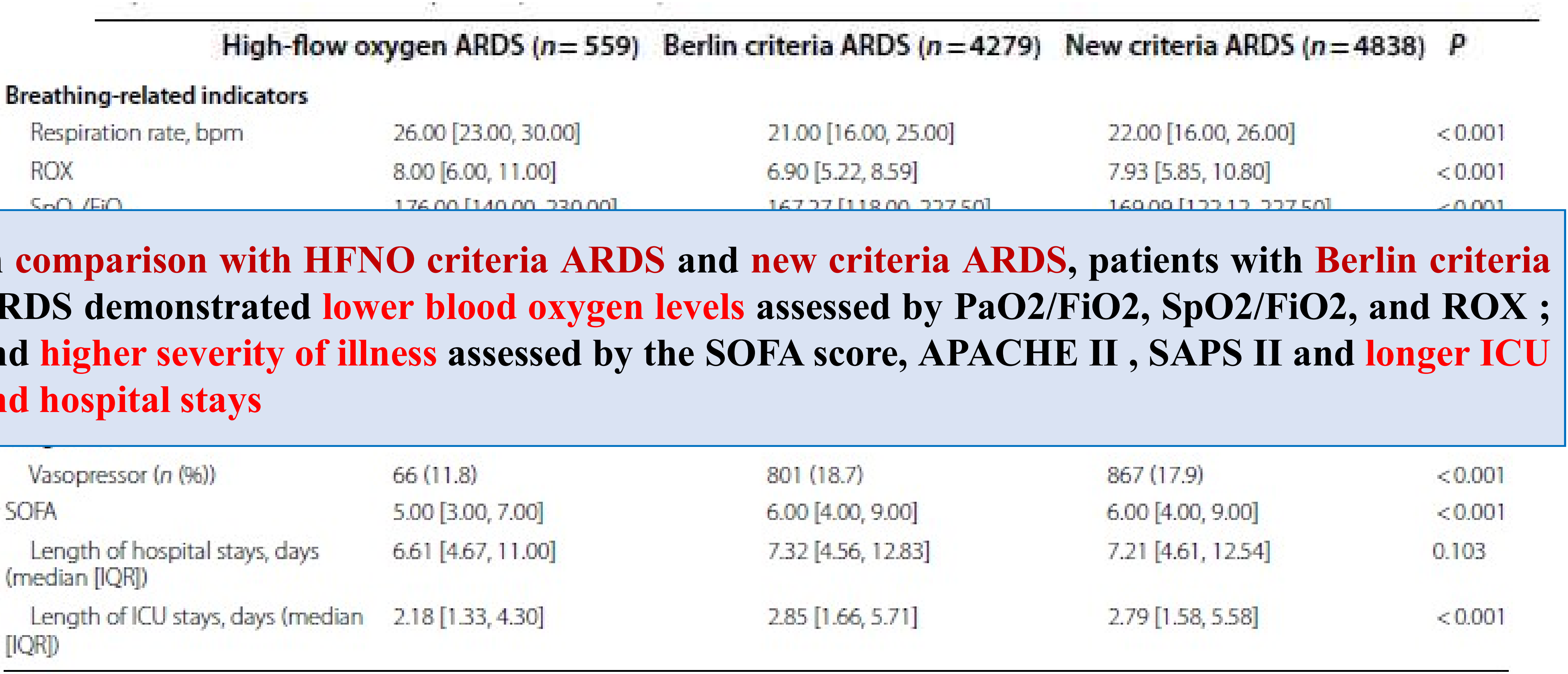


RESEARCH ARTICLE **Open Access**

The impact of the new acute respiratory distress syndrome (ARDS) criteria on Berlin criteria ARDS patients: a multicenter cohort study

BMC 2023

Lina Zhao[1], Fuhong Su[2], Nannan Zhang[1], Hening Wu[1], Yuehao Shen[1], Haiying Liu[1], Xuguang Li[1], Yun Li[3*] and Keliang Xie[1,3*]



	High-flow oxygen ARDS (*n* = 559)	Berlin criteria ARDS (*n* = 4279)	New criteria ARDS (*n* = 4838)	*P*
Breathing-related indicators				
Respiration rate, bpm	26.00 [23.00, 30.00]	21.00 [16.00, 25.00]	22.00 [16.00, 26.00]	< 0.001
ROX	8.00 [6.00, 11.00]	6.90 [5.22, 8.59]	7.93 [5.85, 10.80]	< 0.001
SpO_2/FiO_2	176.00 [140.00, 230.00]	167.27 [118.00, 227.50]	169.09 [[illegible], 227.50]	< 0.001
Vasopressor (*n* (%))	66 (11.8)	801 (18.7)	867 (17.9)	< 0.001
SOFA	5.00 [3.00, 7.00]	6.00 [4.00, 9.00]	6.00 [4.00, 9.00]	< 0.001
Length of hospital stays, days (median [IQR])	6.61 [4.67, 11.00]	7.32 [4.56, 12.83]	7.21 [4.61, 12.54]	0.103
Length of ICU stays, days (median [IQR])	2.18 [1.33, 4.30]	2.85 [1.66, 5.71]	2.79 [1.58, 5.58]	< 0.001

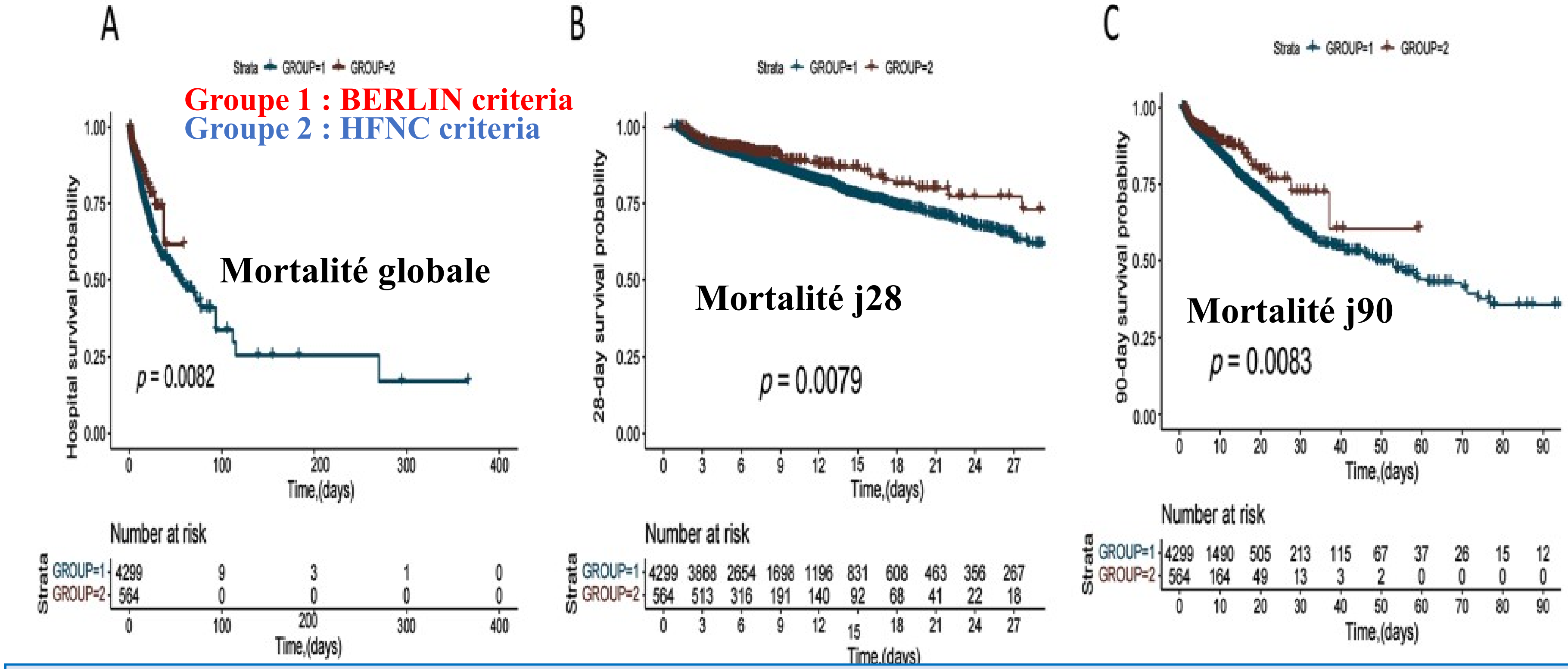
In comparison with HFNO criteria ARDS and new criteria ARDS, patients with Berlin criteria ARDS demonstrated lower blood oxygen levels assessed by PaO2/FiO2, SpO2/FiO2, and ROX ; and higher severity of illness assessed by the SOFA score, APACHE II , SAPS II and longer ICU and hospital stays

RESEARCH ARTICLE **Open Access**

The impact of the new acute respiratory distress syndrome (ARDS) criteria on Berlin criteria ARDS patients: a multicenter cohort study

BMC 2023

Lina Zhao[1], Fuhong Su[2], Nannan Zhang[1], Hening Wu[1], Yuehao Shen[1], Haiying Liu[1], Xuguang Li[1], Yun Li[3*] and Keliang Xie[1,3*]



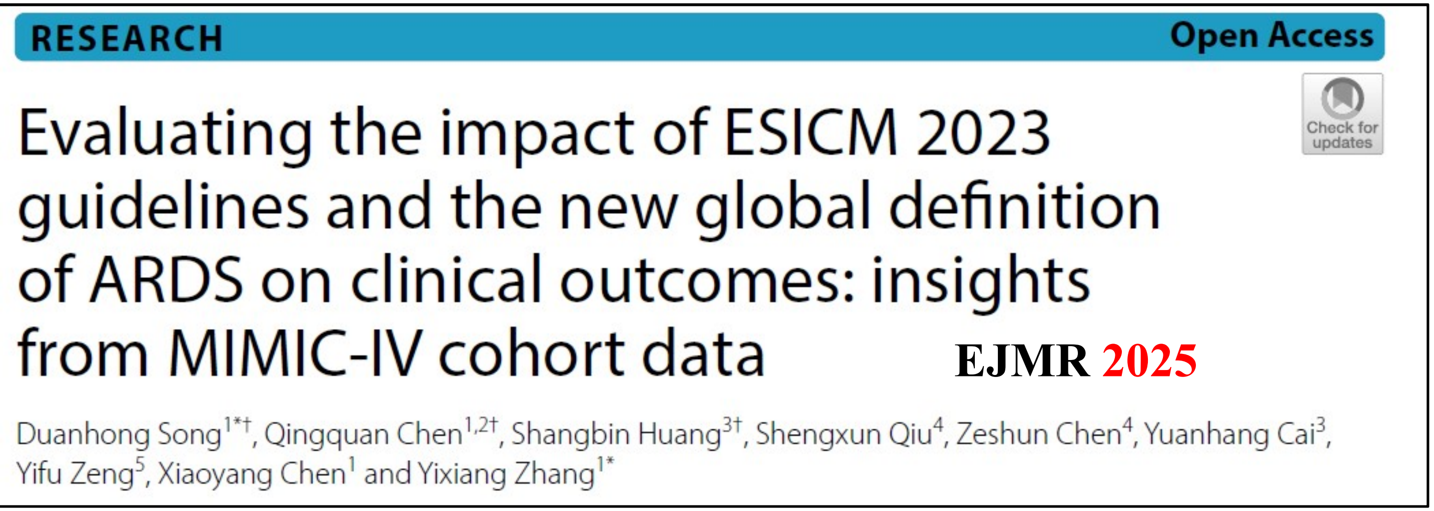
Groupe 1 : BERLIN criteria
Groupe 2 : HFNC criteria

Mortalité globale

Mortalité j28

Mortalité j90

Berlin criteria ARDS patient survival rates were lower than HFNO criteria ARDS patients



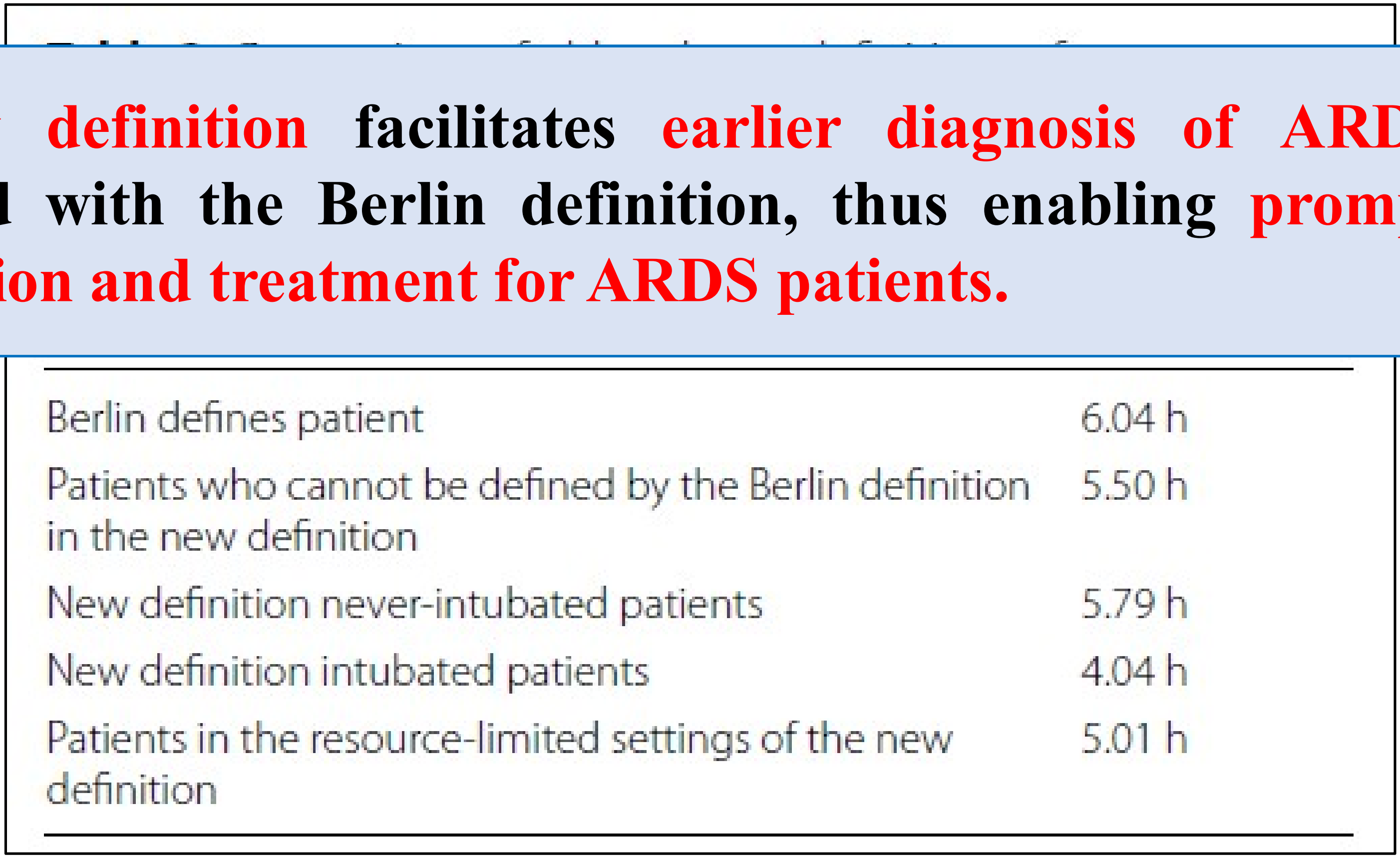
RESEARCH **Open Access**

Evaluating the impact of ESICM 2023 guidelines and the new global definition of ARDS on clinical outcomes: insights from MIMIC-IV cohort data

EJMR 2025

Duanhong Song[1*†], Qingquan Chen[1,2†], Shangbin Huang[3†], Shengxun Qiu[4], Zeshun Chen[4], Yuanhang Cai[3], Yifu Zeng[5], Xiaoyang Chen[1] and Yixiang Zhang[1*]

The new definition facilitates earlier diagnosis of ARDS compared with the Berlin definition, thus enabling prompt intervention and treatment for ARDS patients.



Berlin defines patient	6.04 h
Patients who cannot be defined by the Berlin definition in the new definition	5.50 h
New definition never-intubated patients	5.79 h
New definition intubated patients	4.04 h
Patients in the resource-limited settings of the new definition	5.01 h

Nouvelle définition oui MAIS….

RESEARCH **Open Access**

Limitations of SpO_2 / FiO_2-ratio for classification and monitoring of acute respiratory distress syndrome—an observational cohort study

Critical care 2025

Rolf Erlebach[1*†], Una Pale[2†], Tilman Beck[2], Sasa Markovic[2], Marko Seric[2], Sascha David[1] and Emanuela Keller[2]

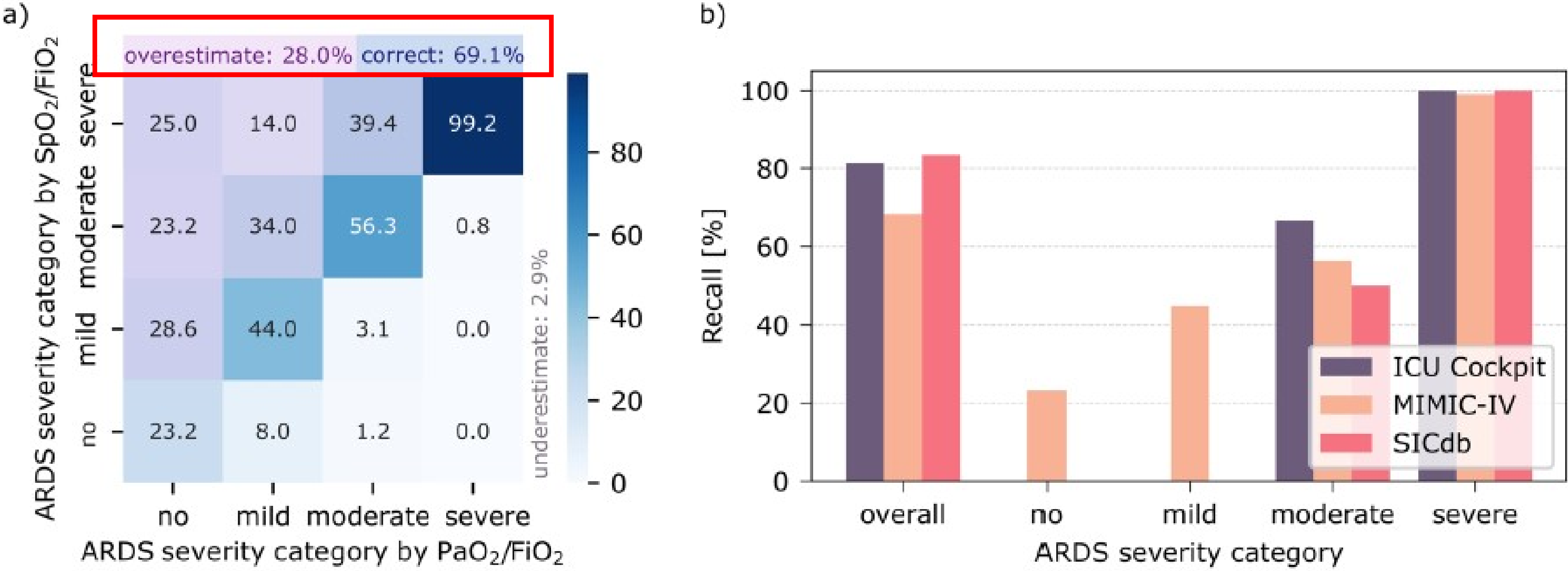
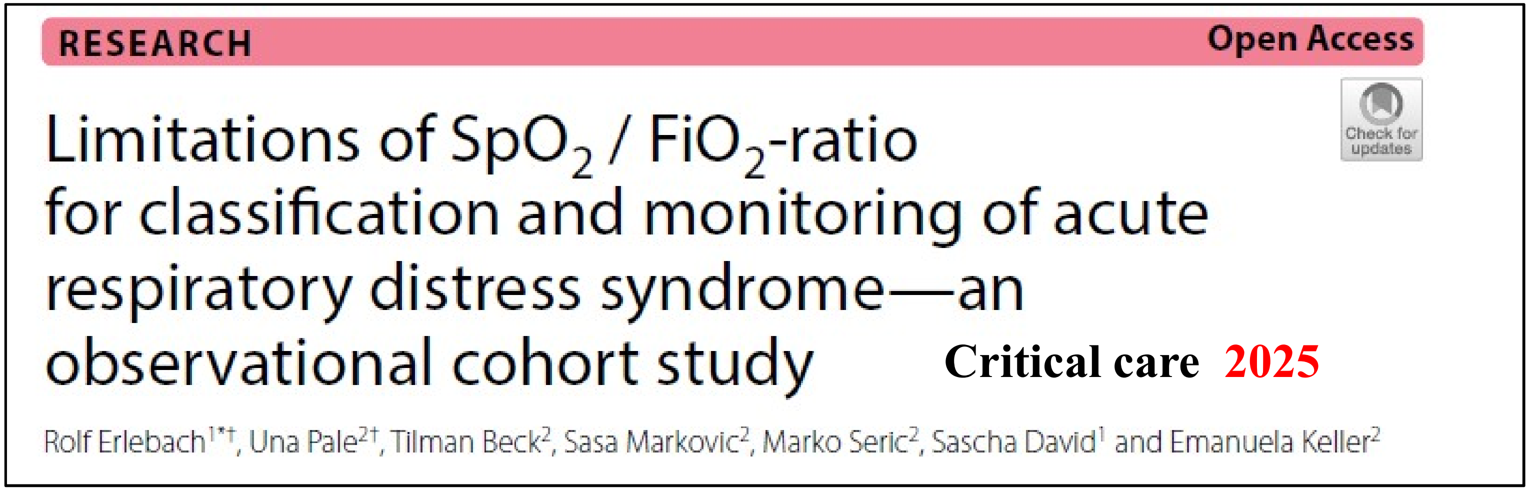


Fig. 1 Performance of the SpO_2/FiO_2-ratio for ARDS severity classification evaluated on admission level and presented as **a** confusion matrix and **b** recall (sensitivity) per ARDS category. Numbers are presented as percentages of PaO_2/FiO_2 category


RESEARCH **Open Access**

Limitations of SpO_2 / FiO_2-ratio for classification and monitoring of acute respiratory distress syndrome—an observational cohort study

Critical care 2025

Rolf Erlebach[1*†], Una Pale[2†], Tilman Beck[2], Sasa Markovic[2], Marko Seric[2], Sascha David[1] and Emanuela Keller[2]

lar, FiO2 settings were
widely distributed
the PaO2/FiO2-ratio
more distinct using the
2/FiO2-ratio categories

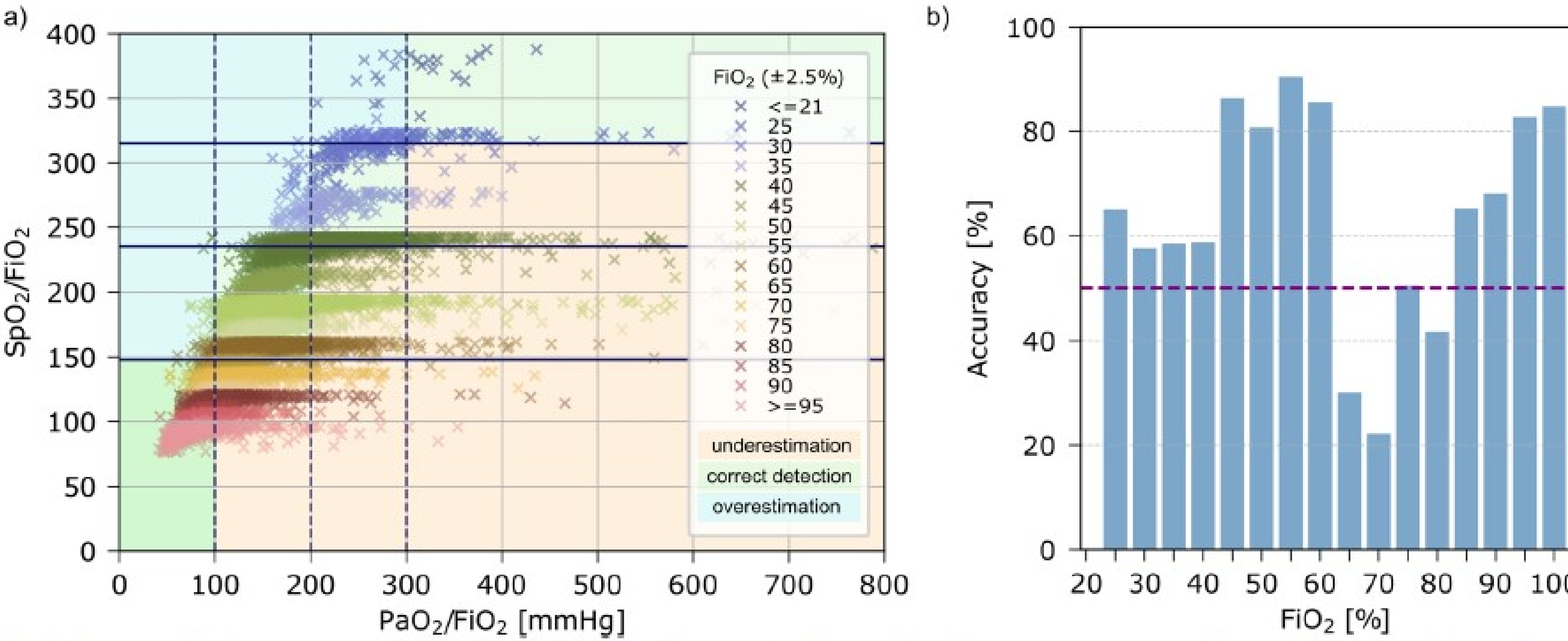
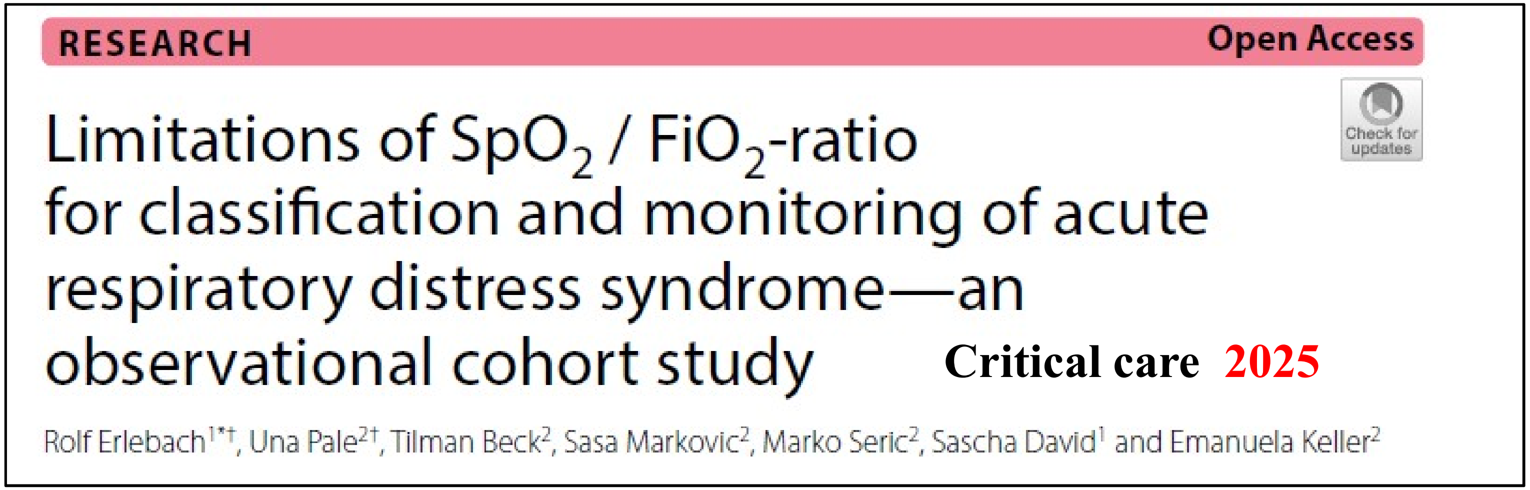


Fig. 2 Impact of FiO_2 on accuracy of SpO_2/FiO_2-ratio. **a** PaO_2/FiO_2 vs. SpO_2/FiO_2 colored by FiO_2. Lines represent thresholds of SpO_2/FiO_2 (solid) and PaO_2/FiO_2 (dashed). **b** ARDS severity classification accuracy in respect to FiO_2. Dashed line marks an accuracy of 50%


RESEARCH **Open Access**

Limitations of SpO_2 / FiO_2-ratio for classification and monitoring of acute respiratory distress syndrome—an observational cohort study

Critical care 2025

Rolf Erlebach[1*†], Una Pale[2†], Tilman Beck[2], Sasa Markovic[2], Marko Seric[2], Sascha David[1] and Emanuela Keller[2]

The use of the SpO2/FiO2-ratio for severity classification in ARDS interchangeably with the PaO2/FiO2-ratio is limited by its high dependence on FiO2 settings, the inability to follow disease progression and the imprecision of SpO2 measurements. This discrepancy in severity classification may influence treatment decisions and patient selection in clinical trials.

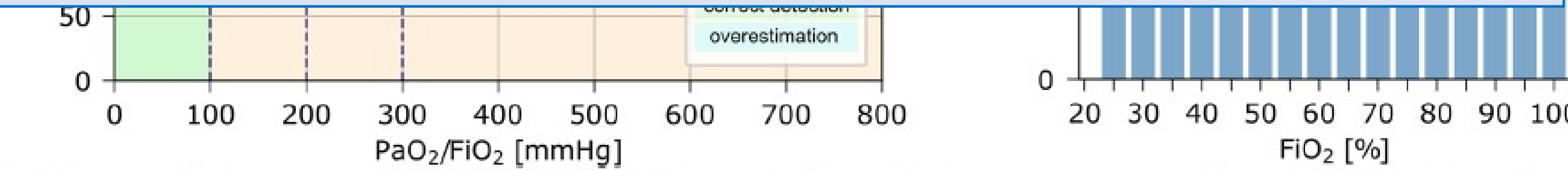


Fig. 2 Impact of FiO_2 on accuracy of SpO_2/FiO_2-ratio. **a** PaO_2/FiO_2 vs. SpO_2/FiO_2 colored by FiO_2. Lines represent thresholds of SpO_2/FiO_2 (solid) and PaO_2/FiO_2 (dashed). **b** ARDS severity classification accuracy in respect to FiO_2. Dashed line marks an accuracy of 50%

Messages clés

- **Nouvelle définition = « fusion » Berlin+ Kigali**
- **Pas de modifications des critères diagnostics usuels** :
  - infiltrat alvéolaire bilatéral non prioritairement cardiogénique apparaissant ou s’aggravant en 1 semaine ou moins
- **Inclusion des SDRA en OHD → rattrape la pratique actuelle**
- **Seuils SpO2/FiO2**
  - Plus complexes, moyen de dépistage ou de suivi plus que de diagnostic ?
  - Intérêt probable si pas de KTA

Messages clés

- Echographie pulmonaire → Variabilité IO, chronophage…à évaluer

- **Peu de modifications pour des pays développés**
- **Evolution majeure pour les pays avec ressources limitées**