26e CONGRÈS NATIONAL

الجمعية التونسية لطب الانعاش
ASSOCIATION TUNISIENNE DE REANIMATION

Fonction ventriculaire droite au cours du SDRA

Dr Merhabene Takoua

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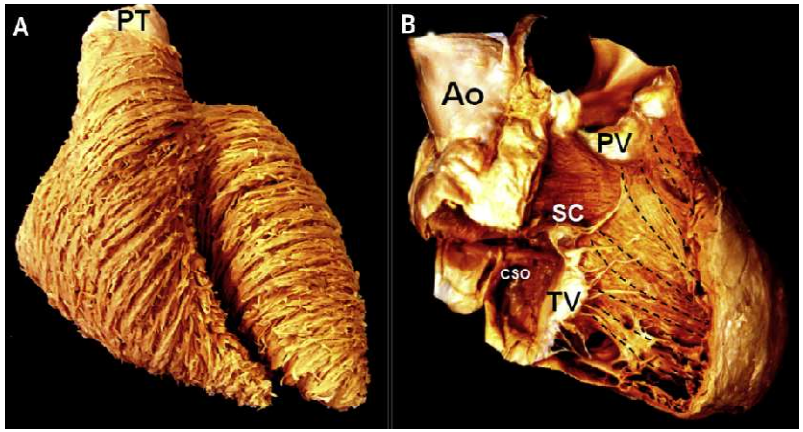
Research Unit for Respiratory Failure and Mechanical Ventilation UR22SP01

جامعة تونس المنار
UNIVERSITÉ DE TUNIS EL MANAR

Plan

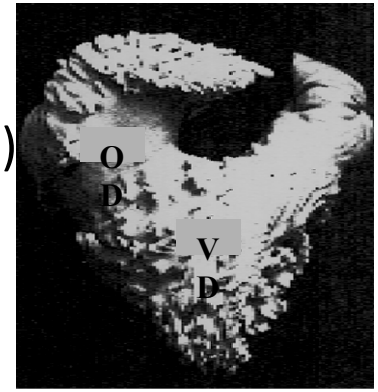
- Rappel (anatomo-physiologique)
- Epidémiologie- définition
- Physiopathologie dysfonction VD-SDRA
- Diagnostic d'une défaillance VD
- Traitement de la défaillance VD au cours du SDRA
- Pronostic

Le VD : structure anatomique



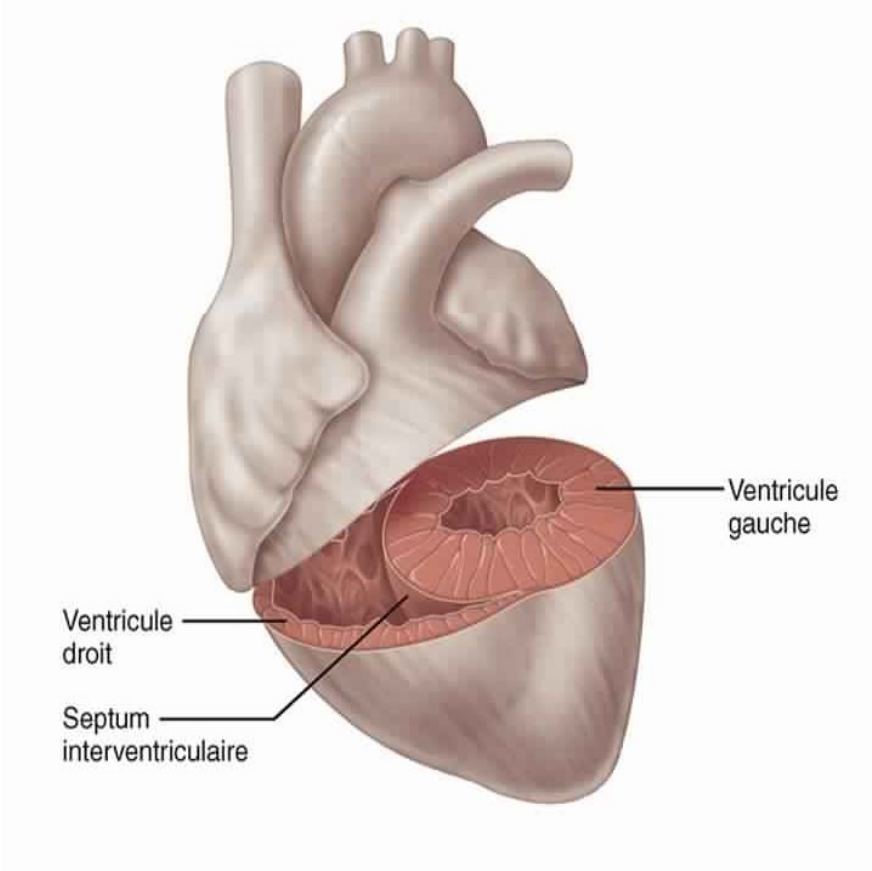
- Structure musculaire trabéculaire/arrangement musculaire circulaire-longitudinal
- VD= géométrie complexe (forme de soufflet), pyramide à base triangulaire
- 2 chambres
  - Sinus: chambre d'admission, générateur de débit pendant le systole
  - Conus: chambre de chasse, régulateur de pression
- Contraction triphasique (hétérogène et asynchrone):
  - Contraction du muscle papillaire (abaissement du plancher tricuspidien), sinus puis conus
  - Mouvement paroi libre → SIV
  - Contraction septal et de la paroi libre du VG (expulsion du sang)
- Paroi libre fine (<5mm) incapable de s'adapter à

une augmentation brusque et importante de la post charge



Anatomie VD/VG

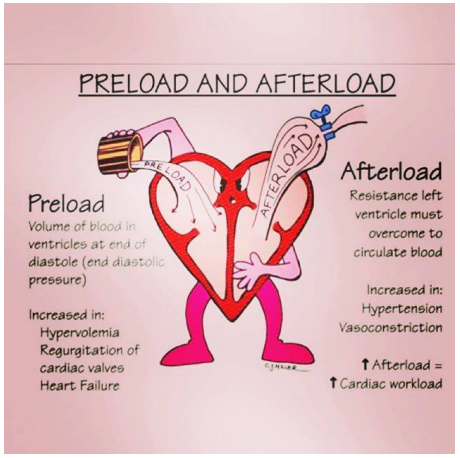
VD= loin d'être un simple conduit passif entre la circulation systémique et pulmonaire



- VD adossé au VG
- Flux sanguin hélicoïdal (apex puis septum)
- Masse musculaire =1/6 VG
- Vascularisation CD+++
- Débit coronaire systolo-diastolique
- Débit sanguin VG/VD=1.5 (VG= 20-40% débit VD)
- Consommation O2= 1/2VG
- VD= travail en débit ≠ VG travail en pression
- Système à basse pression (résist 6 x moins élevées que la circulation systémique) et haute compliance
- Élastance plus basse
- Longueur du sarcomère à pression identique VD>VG

Stephanazzi et al, Ann Fr Anes Réa 1997

Rappel physiologique (2)



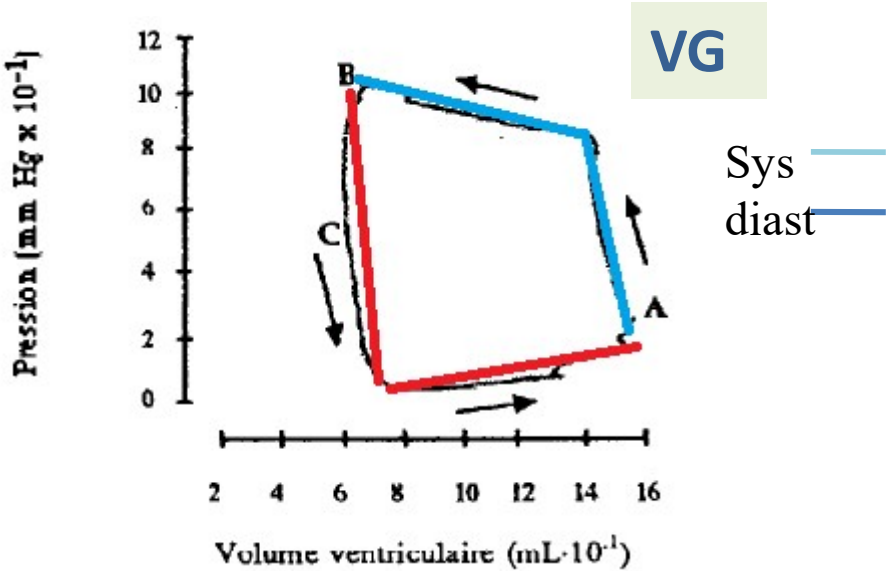
- Fonction VD:
  - Fonction systolique limitée: éjection continue du volume sanguin dans la circulation pulmonaire (faible pression)

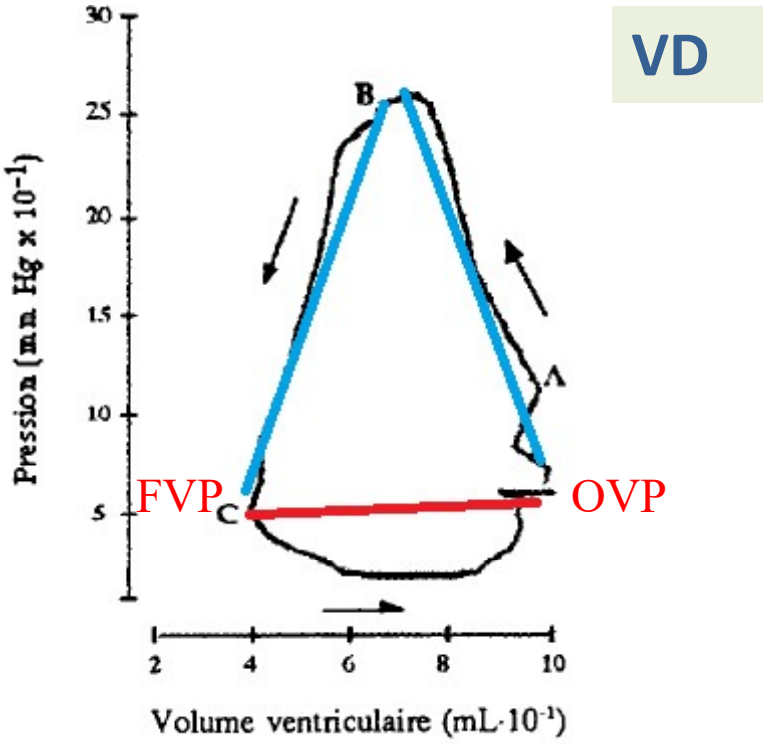
⇒ Adaptation réduite à une augmentation de pression

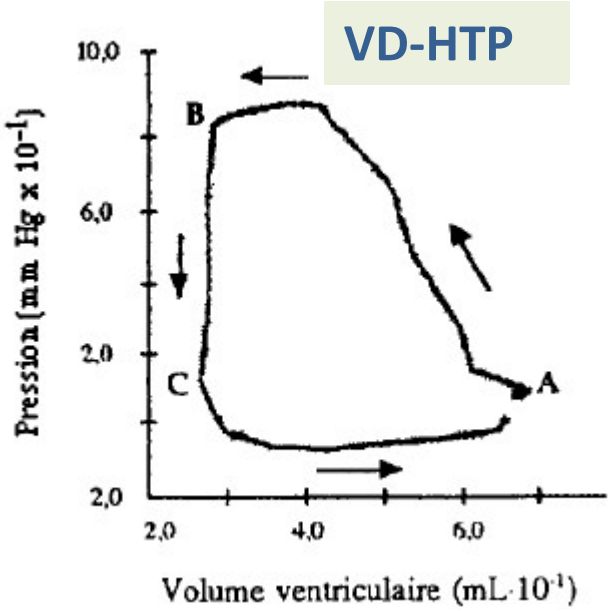
  - Fonction diastolique tolérante (faible élastance)

⇒ Dilatation facile en cas de surcharge volumétrique

Fonction systolique du VD (Courbe pression-volume)







- Contraction en torsion péristaltique
  - Augmentation progressive de la vélocité
  - Le pic tardif
  - La décroissance lente

Stephanazzi et al, Ann Fr Anes Réa 1997

Définition dysfonction VD

V Baron 1985, Vincent 1995 ICM

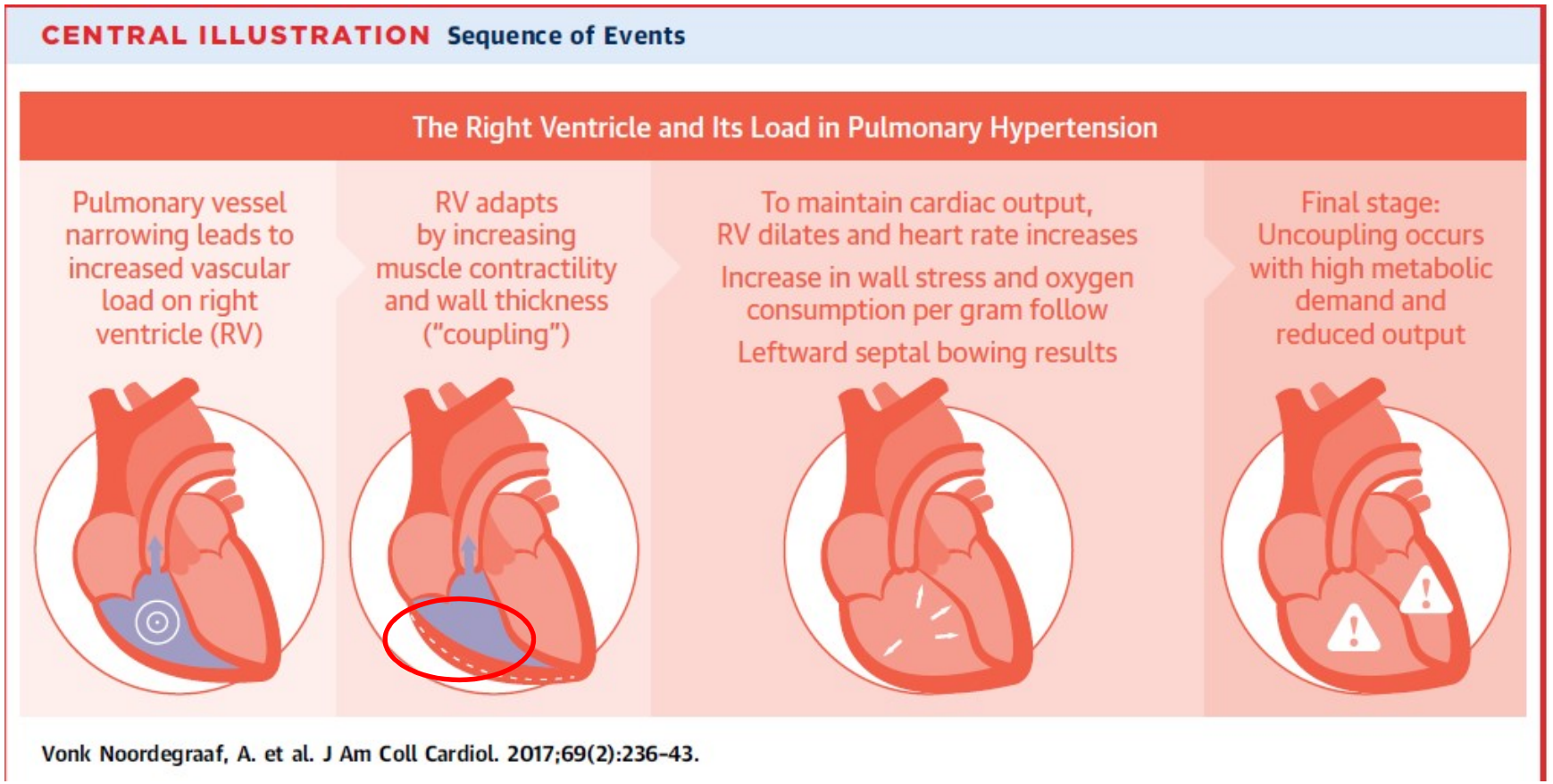
- Dysfonction VD : altération de la relation précharge-VES
  - Diminution de la contractilité
  - Surcharge pressionnelle
  - Surcharge volumétrique

Couplage VD-AP

- Défaillance VD : atteinte de la fonction cardiaque droite responsable d'un hypodébit cardiaque

Acute right ventricular (RV) failure can be defined as a rapidly progressive syndrome with systemic congestion resulting from impaired RV filling and/or reduced RV flow output. Most often it is associated with increased RV afterload or preload and consequent RV chamber dilatation and tricuspid regurgitation

European Journal of Heart Failure (2016) 18, 226–241
doi:10.1002/ejhf.478



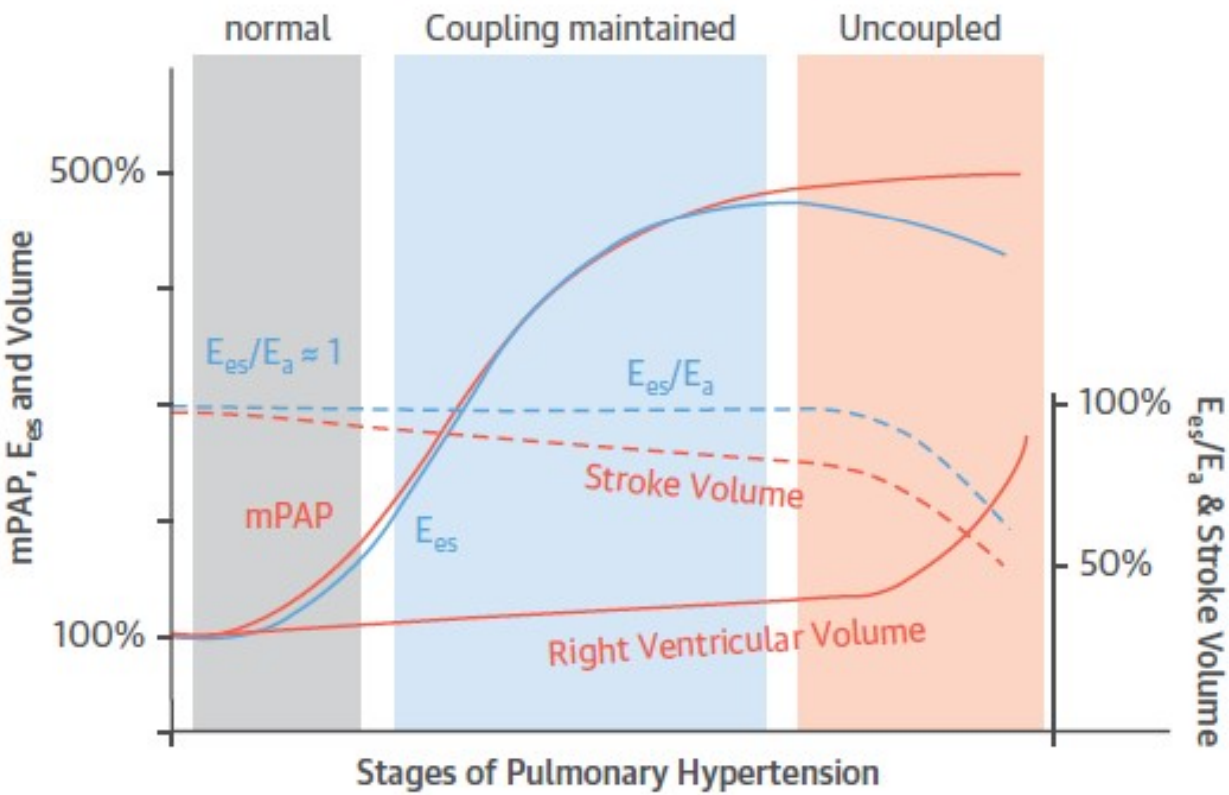
Coupling= augmentation de la contractilité pour vaincre la post charge et maintenir le débit et préserver l'éfficience (transfert d'énergie d'une structure élastique vers le réseau artériel)

Uncoupling= survenue de la dilatation avec augmentation du stress mural

Anatomy, Function, and Dysfunction of the Right Ventricle

JACC State-of-the-Art Review

FIGURE 4 Changes of the Main Parameters in PAH Progression



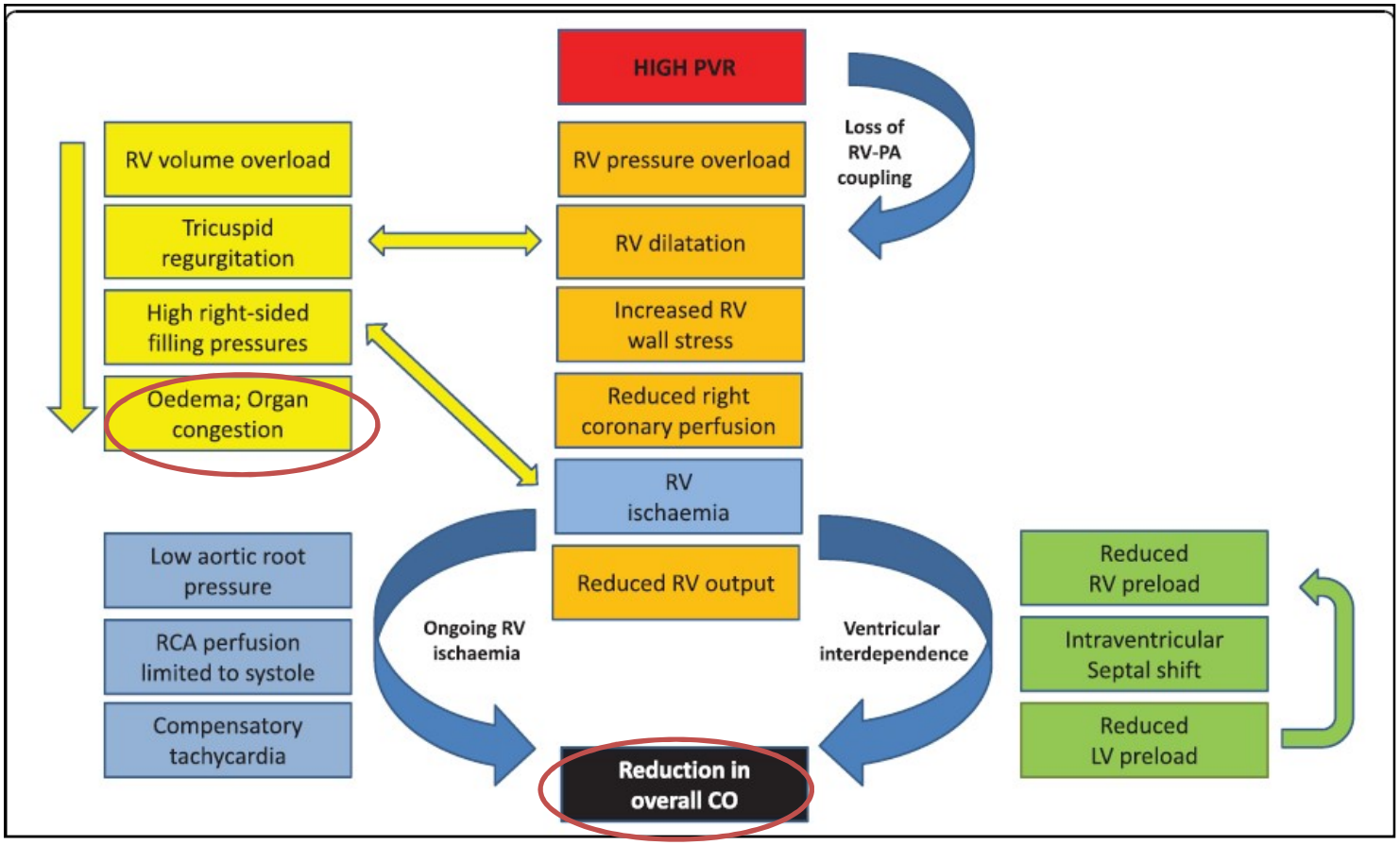
Price et al. Critical Care 2010, 14:R169
http://ccforum.com/content/14/5/R169

CRITICAL CARE

RESEARCH Open Access

Pulmonary vascular and right ventricular dysfunction in adult critical care: current and emerging options for management: a systematic literature review

Laura C Price[1*†], Stephen J Wort[1†], Simon J Finney[1], Philip S Marino[1], Stephen J Brett[2]



Occurrence IVD= perte de la réserve cardiaque

Table 1 Causes and differential diagnosis of acute right ventricular failure

Acute left ventricular failure
Right ventricular ischaemia/infarction
Acute pulmonary embolism
Exacerbation of chronic lung disease and/or hypoxia
Acute lung injury or respiratory distress syndrome
Sepsis
Chronic pulmonary hypertension (groups 1–5)
Pericardial disease (tamponade)
Arrhythmias (supraventricular or ventricular tachycardia)
Congenital heart disease (e.g. atrial or ventricular septal defect, Ebstein's anomaly)
Valvulopathies (e.g. tricuspid valve regurgitation, pulmonary valve stenosis)
Cardiomyopathies (e.g. arrhythmogenic right ventricular dysplasia, familial, idiopathic)
Myocarditis or other inflammatory diseases
Cardiac surgery (e.g. cardiac transplant or left ventricular assist device implantation)
Haematological disorders (e.g. acute chest syndrome in sickle cell disease)

GROUP 3 PH associated with lung diseases and/or hypoxia

- 3.1 Obstructive lung disease or emphysema
- 3.2 Restrictive lung disease
- 3.3 Lung disease with mixed restrictive/obstructive pattern
- 3.4 Hypoventilation syndromes
- 3.5 Hypoxia without lung disease (e.g. high altitude)
- 3.6 Developmental lung disorders

2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension

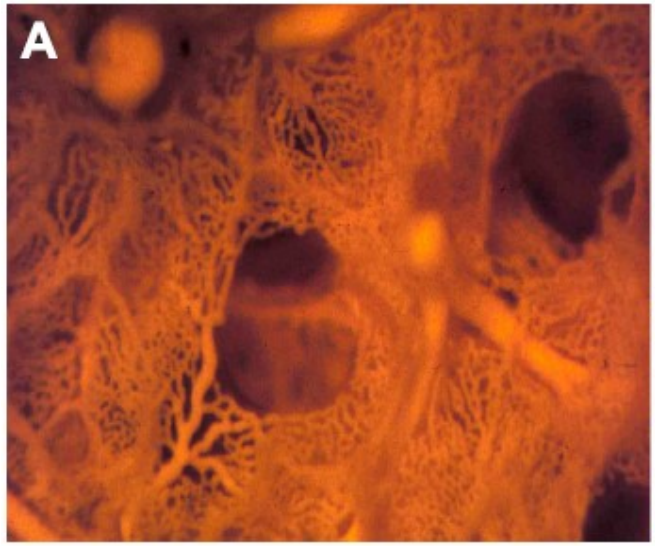
European Journal of Heart Failure (2016) 18, 226–241
doi:10.1002/ejhf.478

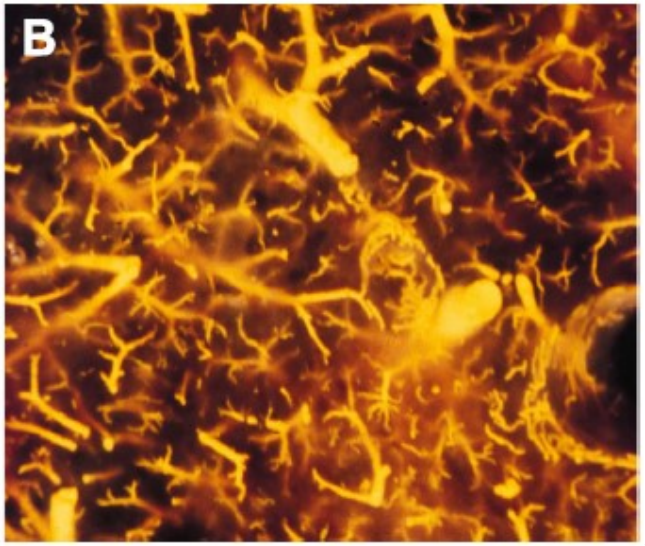
Vascular Obstruction Causes Pulmonary Hypertension in Severe Acute Respiratory Failure*

Warren M. Zapol, M.D.; Koichi Kobayashi, M.D.; Michael T. Snider, M.D.; Reginald Greene, M.D.; and Myron B. Laver, M.D.

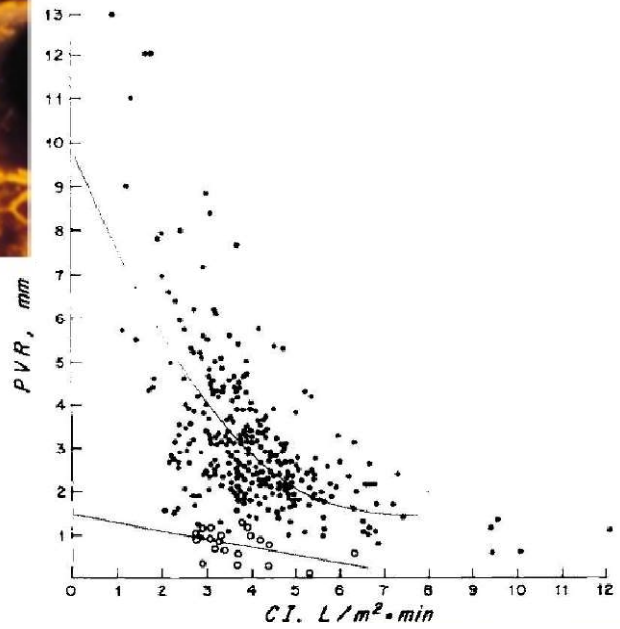
19TH ASPEN LUNG CONFERENCE

CHEST 71: 2, FEBRUARY, 1977 SUPPLEMENT





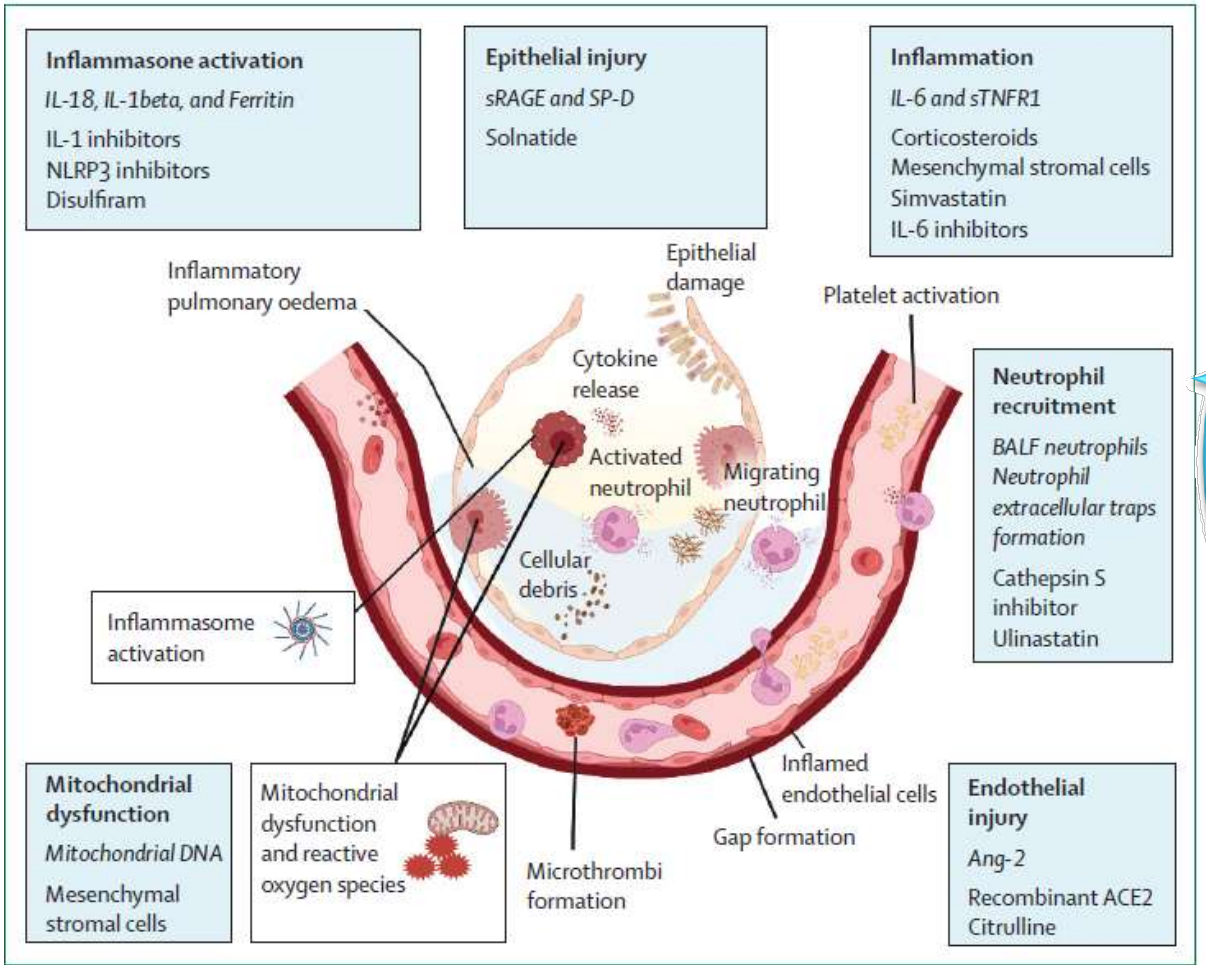
Obstruction de la microcirculation pulmonaire

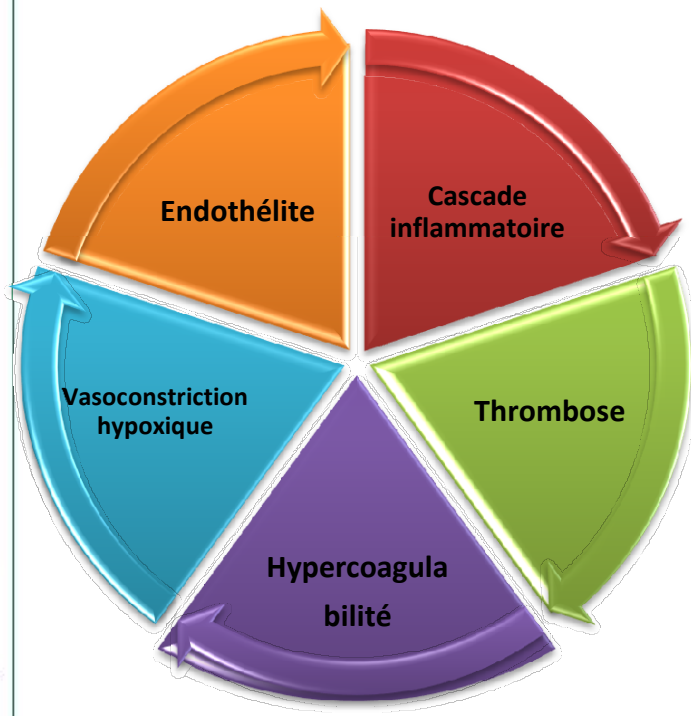


Acute Respiratory Distress Syndrome 2022 2

Acute respiratory distress syndrome in adults: diagnosis, outcomes, long-term sequelae, and management

Ellen A Gorman, Cecilia M O'Kane, Daniel F McAuley

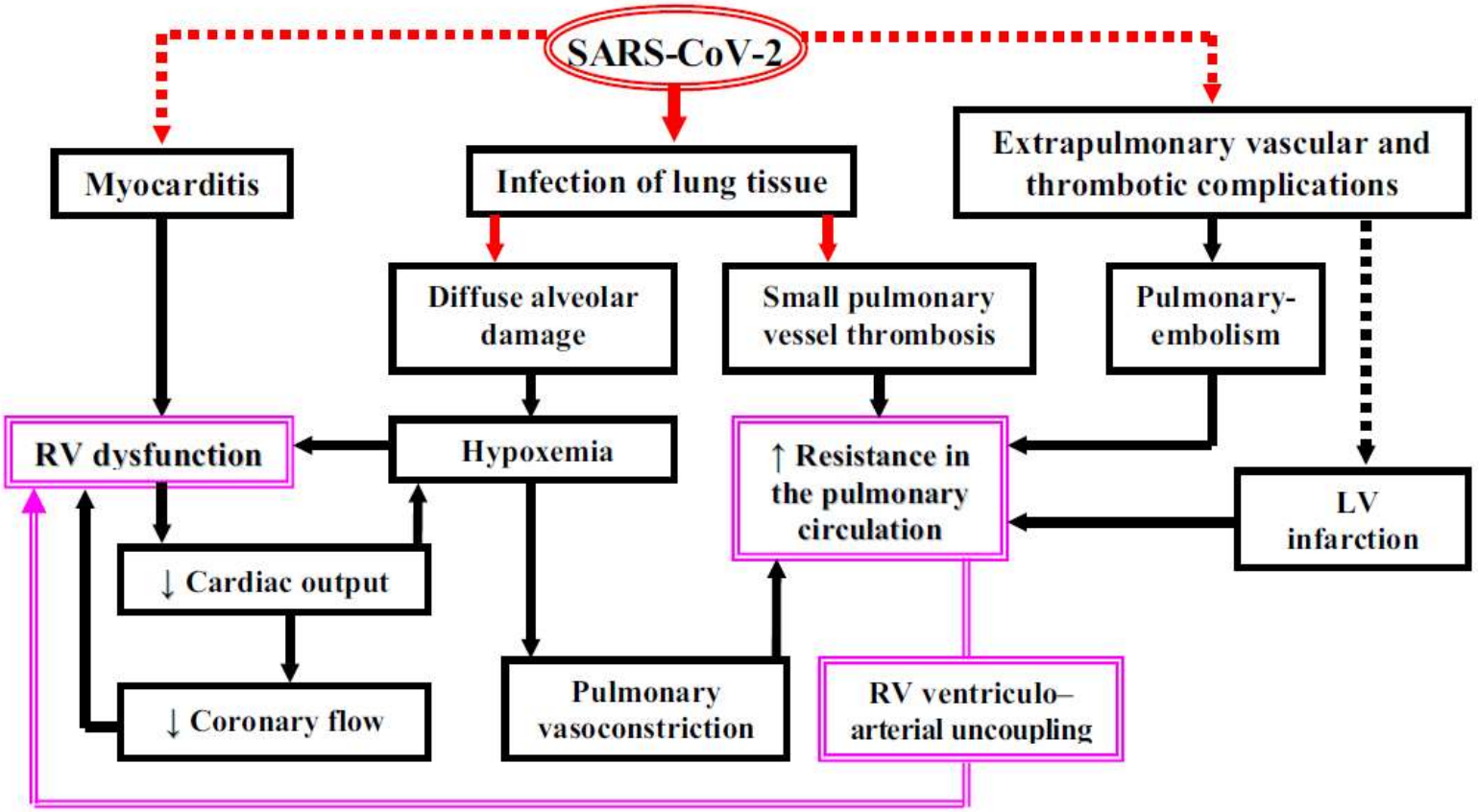




Heart Failure Reviews (2022) 27:1325–1339
https://doi.org/10.1007/s10741-021-10108-7

Heart–lung interactions in COVID-19: prognostic impact and usefulness of bedside echocardiography for monitoring of the right ventricle involvement

COVID-19 Related ARDS



Effets VM/VD

↑ pression intrathoracique

↑volume pulmonaire

↑de la pression alvéolaire

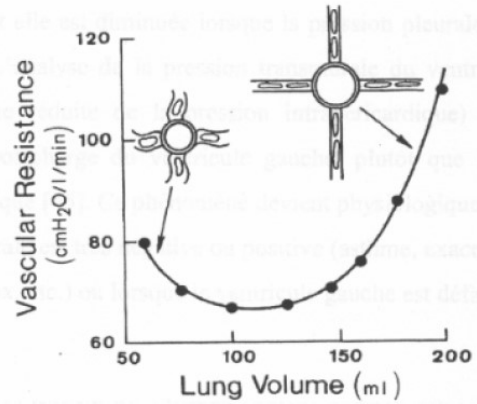
↑Pression pleurale: chute du retour veineux

Modification de la géométrie ventriculaire

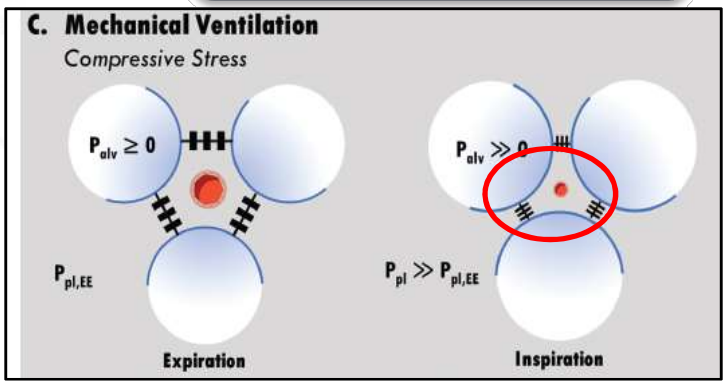
Réduction des volumes télé diastoliques

Augmentation résistances vasculaires pulmonaires

↓↓Précharge VD



↑↑Post charge VD (Elast art)



Physiological and Pathophysiological Consequences of Mechanical Ventilation Silva et al.

Seminars in Respiratory and Critical Care Medicine © 2022.

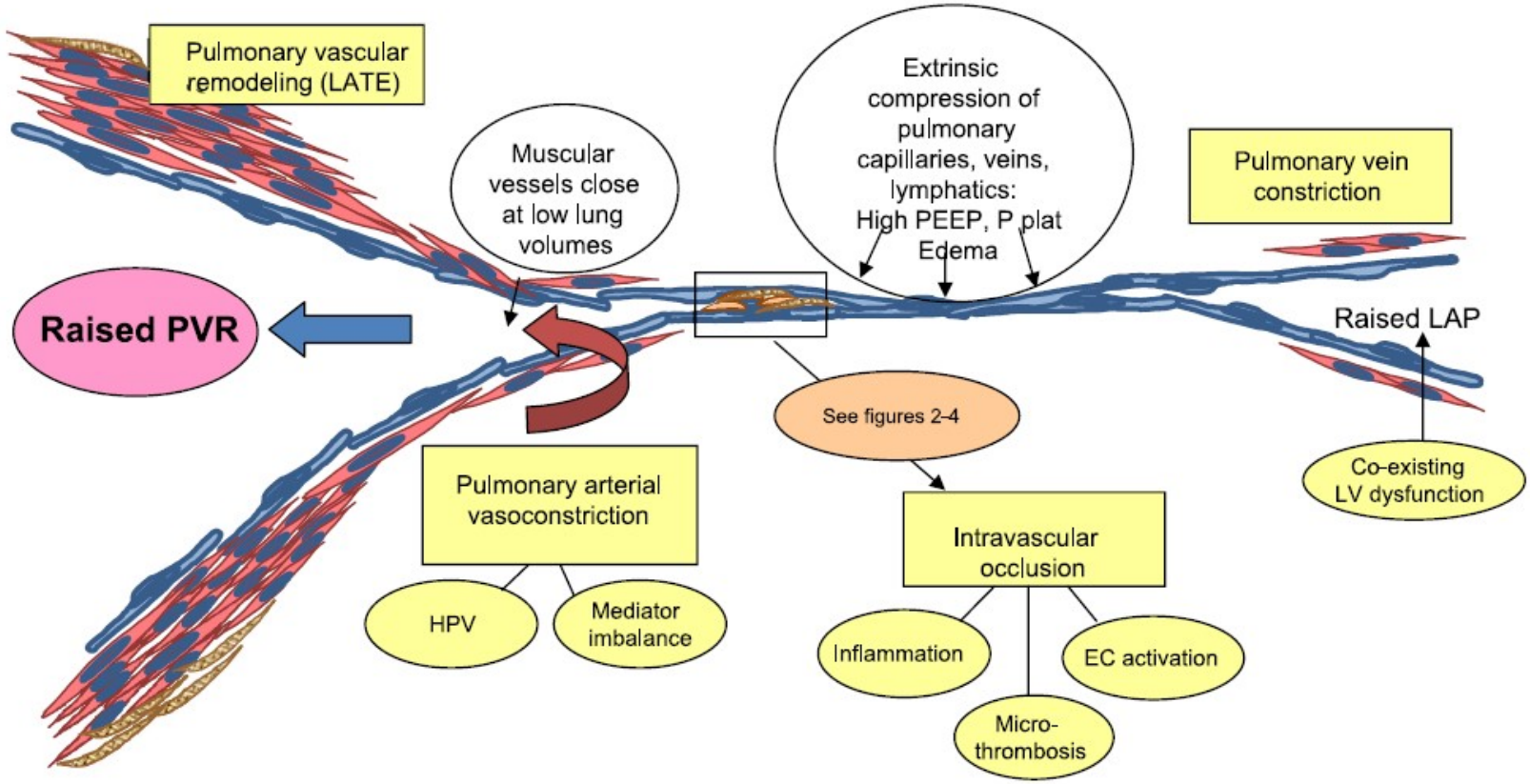
Am J Physiol Lung Cell Mol Physiol 302: L803–L815, 2012.
First published January 13, 2012; doi:10.1152/ajplung.00355.2011.

Review

Pathophysiology of pulmonary hypertension in acute lung injury

Laura C. Price,[1] Danny F. McAuley,[2] Philip S. Marino,[1] Simon J. Finney,[1] Mark J. Griffiths,[1]* and Stephen John Wort[1]*

[1]*Department of Critical Care, Royal Brompton Hospital, National Heart and Lung Institute, Imperial College London, London, United Kingdom; and* [2]*Respiratory Medicine Research Programme, Centre for Infection and Immunity, Queen's University, Grosvenor Road, Belfast, United Kingdom*



Intensive Care Med (2009) 35:1850–1858
DOI 10.1007/s00134-009-1569-2

ORIGINAL

Armand Mekontso Dessap
Cyril Charron
Jérôme Devaquet
Jérôme Aboab
François Jardin
Laurent Brochard
Antoine Vieillard-Baron

Impact of acute hypercapnia and augmented positive end-expiratory pressure on right ventricle function in severe acute respiratory distress syndrome

	LP strategy	HP/HR strategy	HP/LR strategy
P_{plat} (cmH_2O)	22.1 (18.9–27.0)	23.0 (20.1–28.0)	22.0 (19.5–26.2)
$PEEP_{tot}$ (cmH_2O)	5.4 (5.0–6.0)	11.0 (10.0–12.0)*	10.0 (10.0–12.0)*
P_{aw} (cmH_2O)	11.8 (10.8–12.7)	15.5 (14.8–16.9)*	15.5 (13.8–16.7)*
V_T (mL)	548 (468–605)	336 (260–360)*	356 (281–398)*
V_T (mL/kg)	8.5 (8.3–8.9)	5.3 (4.5–6.1)*	5.8 (4.5–7.4)*
Respiratory rate (bpm)	15 (15–20)	26 (25–30)*	15 (15–20)#
Minute ventilation (L/min)	8.3 (7.2–9.8)	8.4 (7.1–8.8)	5.3 (4.6–7.5)*,#
$PaCO_2$ (mmHg)	52 (43–68)	71 (60–94)*	75 (53–84)*
pH	7.30 (7.24–7.35)	7.17 (7.12–7.23)*	7.20 (7.16–7.25)*
PaO_2/FiO_2 ratio (mmHg)	88 (60–110)	103 (74–138)*	101 (81–137)*
Alveolar recruitment at elastic distending pressure of			
15 cmH_2O (mL)[a]	64 (40–89)	151 (77–245)*	133 (67–197)*
17.5 cmH_2O (mL)[a]	42 (36–65)	117 (50–184)*	116 (44–146)*
PEEP-induced increase in end-expiratory lung volume (mL)	215 (110–269)*	520 (310–614)*	547 (392–561)*
Dead space fraction (% of V_T) ($n = 7$)[b]			
Total dead space	72 (60–77)	88 (73–90)*	72 (55–80)#
Airway dead space	35 (31–47)	61 (46–67)*	42 (29–50)#
Alveolar dead space	25 (23–29)	20 (14–23)	25 (16–31)

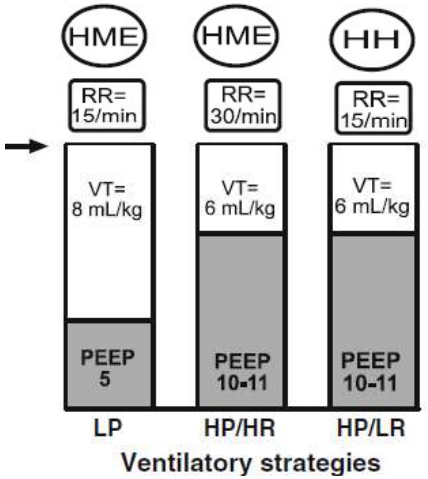


Table 3 Echocardiographic variables

	LP strategy	HP/HR strategy	HP/LR strategy
LV ejection fraction (%)	62 (57–71)	64 (49–67)	59 (53–66)
LV fractional area contraction (%)	58 (55–65)	57 (53–70)	62 (54–65)
LV maximal systolic elastance (mmHg/mL)	5.5 (3.9–6.3)	5.2 (4.1–6.6)	5.4 (4.3–7.2)
Superior vena cava collapsibility index (%)	0 (0–26)	0 (0–8)	0 (0–23)
RV stroke index (cm^3/m^2)	22 (20–32)	17 (10–26)*	16 (11–27)*
Cardiac index (L/min/m^2)	2.60 (1.53–3.54)	1.87 (1.16–2.98)*	1.89 (1.38–3.35)*
End-systolic eccentricity index	1.10 (1.02–1.25)	1.19 (1.07–1.54)*	1.28 (1.00–1.52)*
End-diastolic RV/LV area ratio	0.64 (0.56–0.77)	0.85 (0.62–1.10)*	1.0 (0.69–1.20)*,#
End-diastolic RV/LV area ratio >0.6	7 (64%)	9 (82%)	9 (82%)

Intensive Care Med (2016) 42:862–870
DOI 10.1007/s00134-015-4141-2

SEVEN-DAY PROFILE PUBLICATION

Armand Mekontso Dessap
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Acute cor pulmonale during protective ventilation for acute respiratory distress syndrome: prevalence, predictors, and clinical impact

Table 2 Factors associated with acute cor pulmonale in patients with acute respiratory distress syndrome

Variable	Odds ratio (95 % CI) by logistic regression	
	Univariate	Multivariable[a]
Pneumonia as cause of ARDS	2.54 (1.79–3.62), $p < 0.01$	2.73 (1.84–4.05), $p < 0.01$
Respiratory settings on TEE day		
Tidal volume <7 mL/kg	1.70 (1.17–2.47), $p < 0.01$	I/NR
Respiratory rate ≥30 breaths/min	1.70 (1.11–2.60), $p = 0.02$	I/NR
Plateau pressure ≥27 cmH_2O	1.91 (1.33–2.73), $p < 0.01$	I/NR
Compliance <30 ml/cmH_2O	1.91 (1.33–2.73), $p < 0.01$	I/NR
Driving pressure ≥18 cmH_2O[b]	2.16 (1.51–3.10), $p < 0.01$	2.28 (1.53–3.38), $p < 0.01$
Arterial blood gases on TEE day		
PaO_2/FiO_2 ratio <150 mmHg	2.41 (1.49–3.92), $p < 0.01$	2.60 (1.50–4.52), $p < 0.01$
$PaCO_2$ ≥48 mmHg	2.95 (2.06–4.21), $p < 0.01$	2.39 (1.62–3.52), $p < 0.01$

Table 3 The acute cor pulmonale risk score

Parameter	Score
Pneumonia as cause of ARDS	1
Driving pressure ≥18 cmH_2O[a]	1
PaO_2/FiO_2 ratio <150 mmHg	1
$PaCO_2$ ≥48 mmHg	1
Total score	0–4

ROC-AUC 0,7; IC 0,62-0,78; p<0,001

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DOI 10.1007/s00134-015-4141-2

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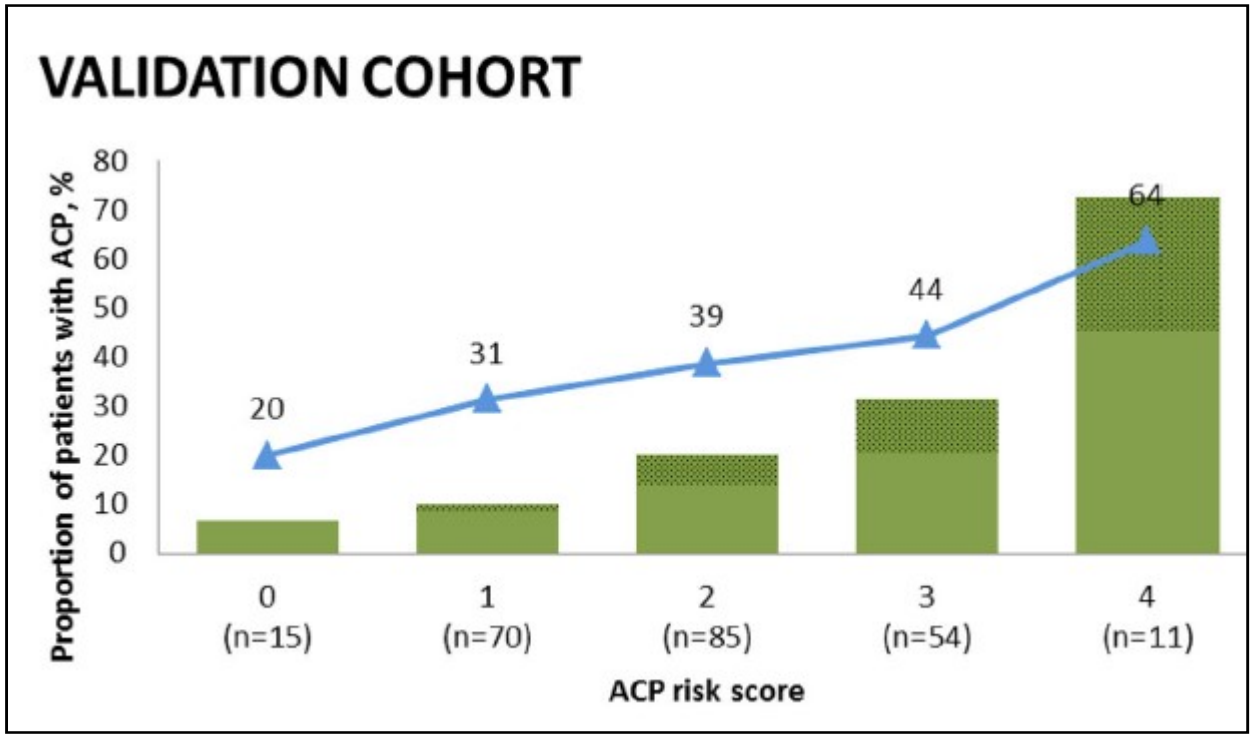
Acute cor pulmonale during protective ventilation for acute respiratory distress syndrome: prevalence, predictors, and clinical impact



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Acute cor pulmonale during protective ventilation for acute respiratory distress syndrome: prevalence, predictors, and clinical impact

22% CPA

Sato *et al. Crit Care* (2021) 25:172
https://doi.org/10.1186/s13054-021-03591-9

Critical Care

RESEARCH Open Access

The impact of right ventricular injury on the mortality in patients with acute respiratory distress syndrome: a systematic review and meta-analysis

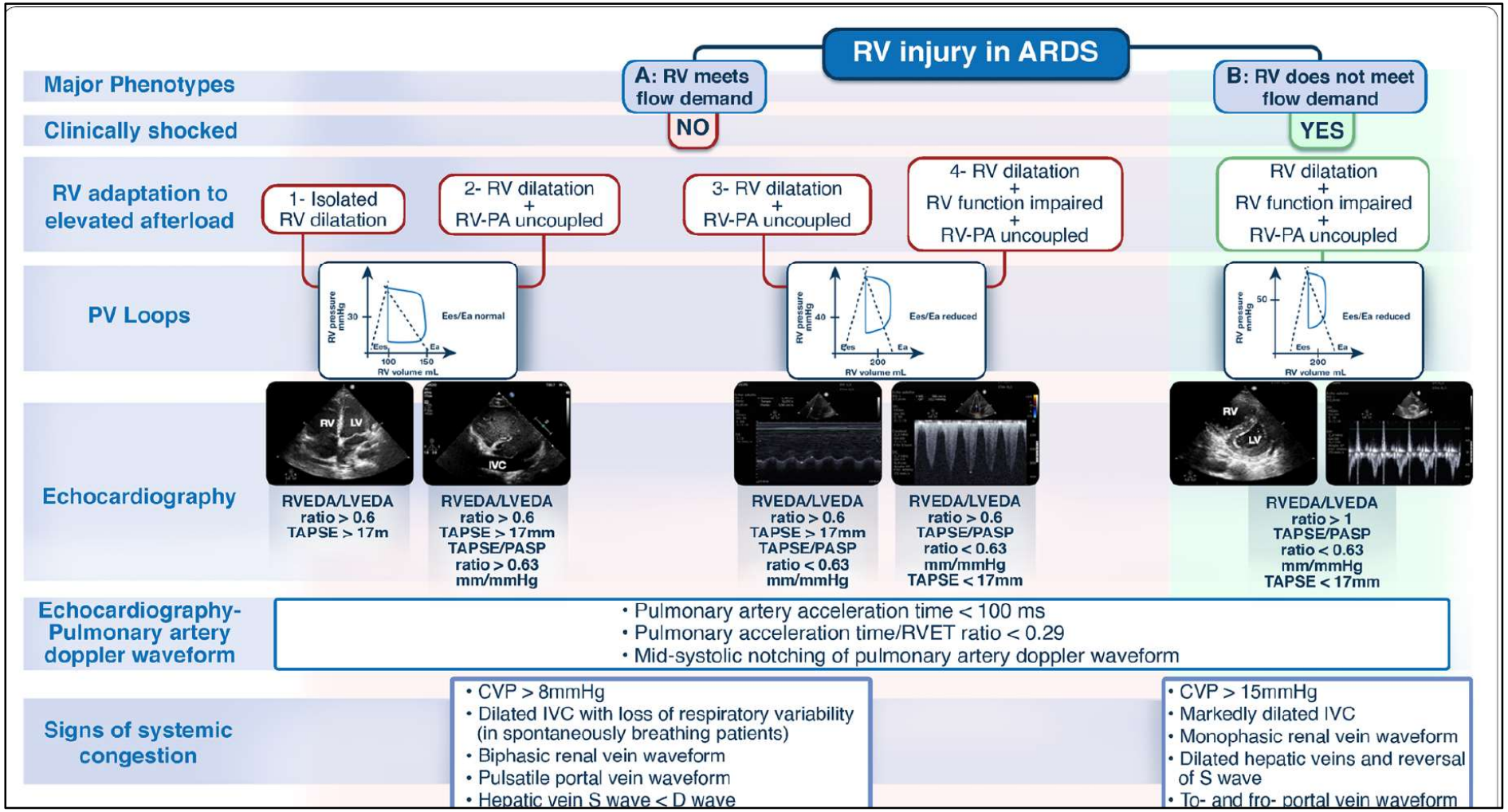
Ryota Sato[1], Siddharth Dugar[1,2*], Wisit Cheungpasitporn[3], Mary Schleicher[4], Patrick Collier[5], Saraschandra Vallabhajosyula[6,7,8,9] and Abhijit Duggal[1,2]

21%

Acute right ventricular injury phenotyping in ARDS

Vasileios Zochios[1,2*], Hakeem Yusuff[1,3], Matthieu Schmidt[4,5] on behalf of Protecting the Right Ventricle Network (PRORVnet)

ICM, octobre 2022

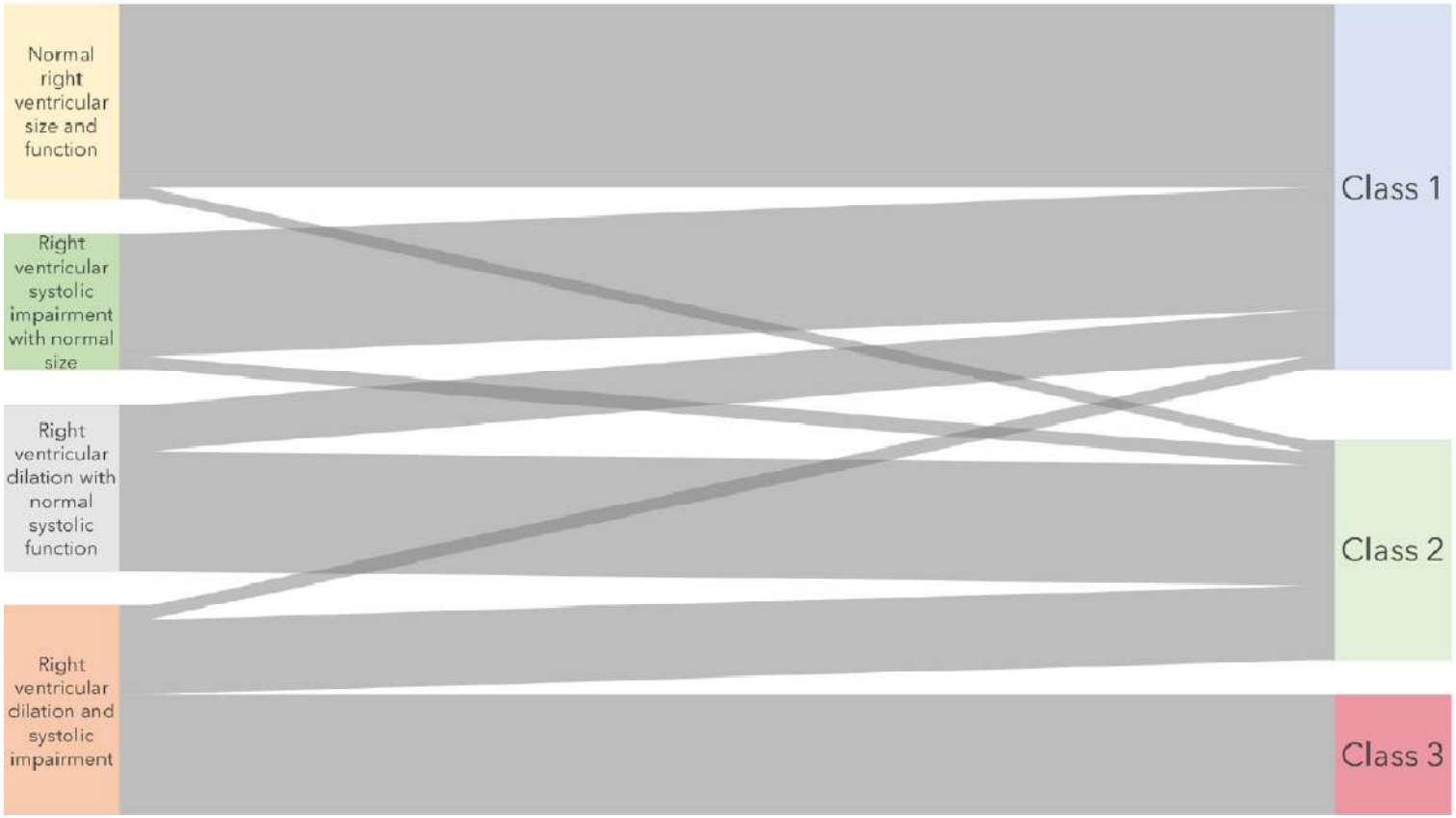


Anaesthesia 2022, 77, 763–771 doi:10.1111/anae.15700

Original Article

Cardiovascular subphenotypes in patients with COVID-19 pneumonitis whose lungs are mechanically ventilated: a single-centre retrospective observational study

M. Chotalia,[1] M. Ali,[2] J. E. Alderman,[2] J. M. Patel,[3] D. Parekh[3] and M. N. Bangash[3]



52%

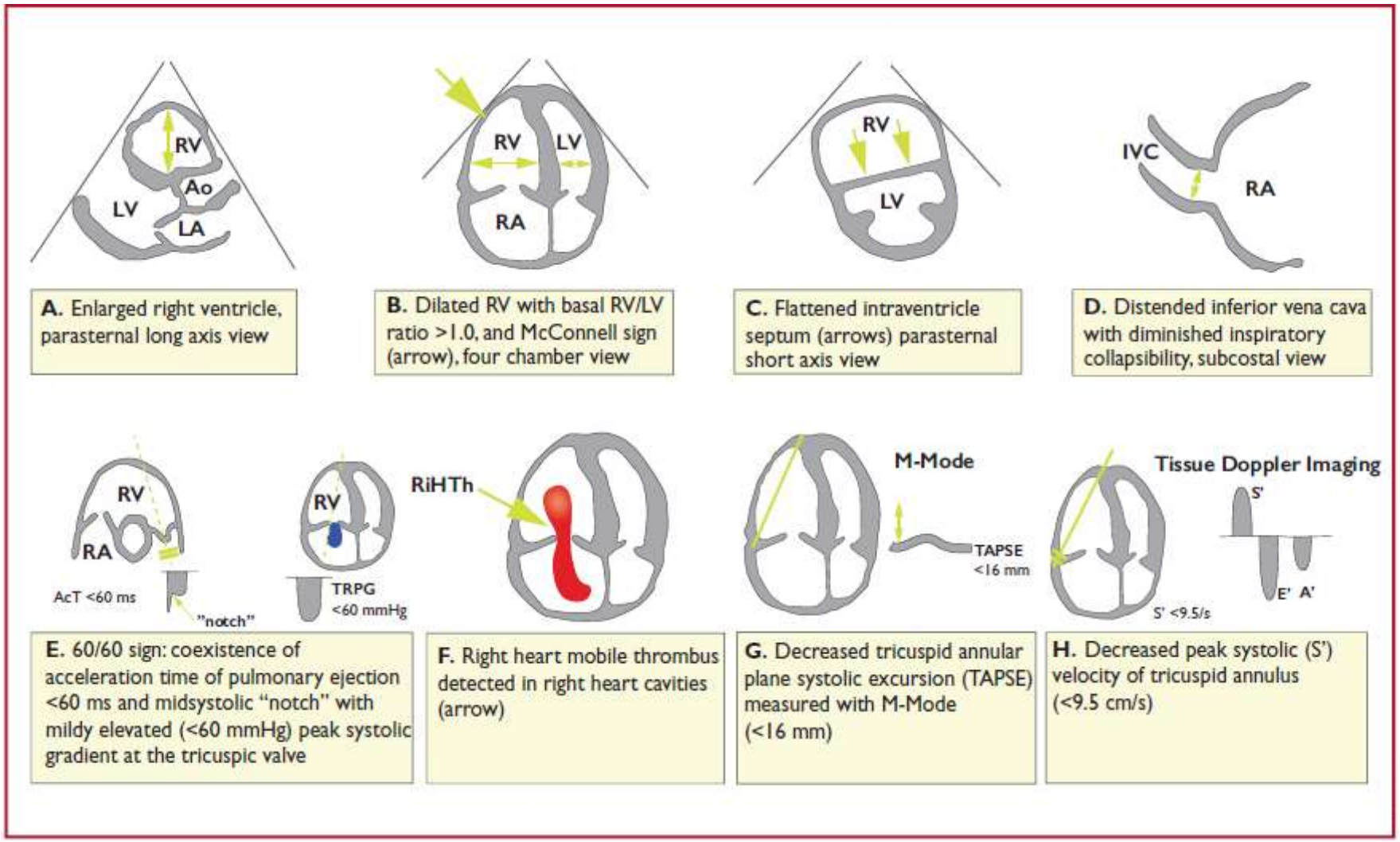
31%

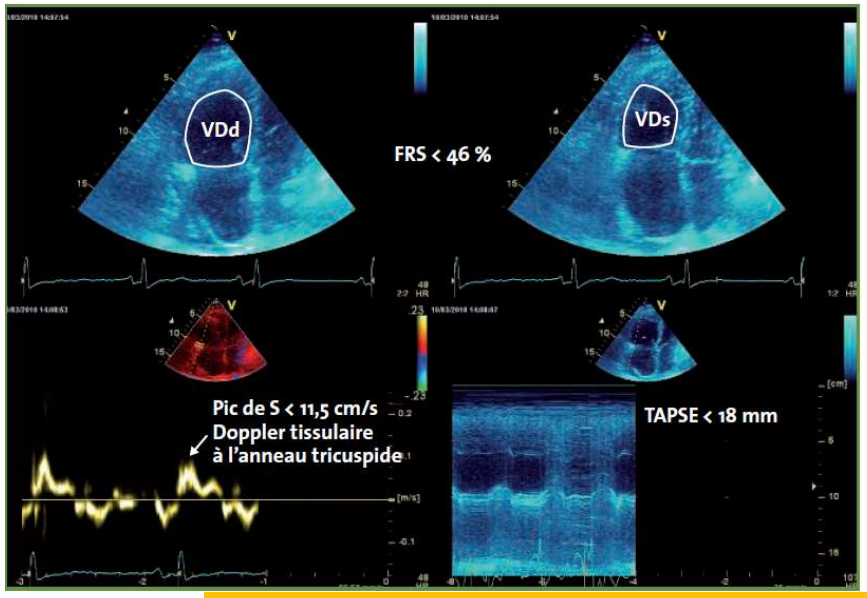
17%

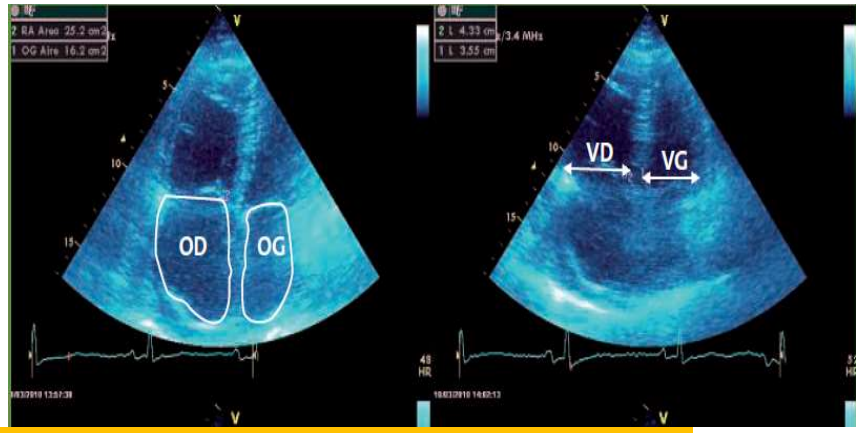
Table 1 Comparison of baseline characteristics and clinical variables in cardiovascular subphenotypes. Values are mean (SD), number (proportion) or median (IQR [range])

	All n = 305	Class 1 n = 158	Class 2 n = 95	Class 3 n = 52	p value
Age; y	57 (12)	55 (13)	60 (12)	58 (10)	0.010
Sex; male	219 (72%)	104 (66%)	74 (78%)	40 (77%)	0.076
Day of transthoracic echocardiogram	8 (5–13 [1–31])	8 (4–13 [1–26])	9 (5–14 [1–31])	7 (4–12 [1–24])	0.482
Acute respiratory distress syndrome severity					0.014
Mild	54 (18%)	36 (23%)	12 (13%)	6 (12%)	
Moderate	199 (65%)	105 (67%)	61 (64%)	33 (63%)	
Severe	52 (17%)	17 (11%)	22 (23%)	13 (25%)	
PaO_2:F_IO_2	20 (16–25 [7–39])	22 (17–27 [10–39])	19 (14–24 [8–39])	18 (13–22 [7–39])	< 0.001
$PaCO_2$; kPa	7 (6–9 [4–15])	7 (6–9 [4–13])	8 (7–10 [4–13])	7 (7–10 [6–15])	0.031
pH	7.34 (7.28–7.39 [7.00–7.50])	7.34 (7.29–7.39 [7.10–7.50])	7.32 (7.27–7.37 [7.10–7.50])	7.31 (7.22–7.37 [7.00–7.40])	0.016
White blood cell count; x 10^9 l^{-1}	13 (9–16 [0–44])	12 (9–16 [1–32])	13 (10–18 [1–39)	12 (8–16 [0–44])	0.188
C-reactive protein; mg.ml^{-1}	132 (65–221 [5–554])	115 (59–189 [5–409])	148 (74–249 [5–449])	167 (76–266 [5–554])	0.001
Troponin; ng.l^{-1}	26 (9–161 [4–72,304])	7 (15–62 [4–1995])	26 (9–132 [4–2983])	132 (35–1109 [10–72,304])	< 0.001
D-dimer; ng.ml^{-1}	1349 (617–3392 [104–42,230])	1035 (456–2680 [104–13,349])	1978 (855–4286 [113–30,093])	2498 (664–6403 [109–42,230])	0.002
Chest radiograph opacification score (0–16)	8 (6–10 [3–16])	8 (6–8 [3–14])	8 (6–10 [4–16])	9 (8–11 [4–16])	< 0.001
Dead space fraction	0.74 (0.65–0.81 [0.25–0.92])	0.72 (0.64–0.80 [0.25–0.92])	0.74 (0.65–0.80 [0.25–0.89)	0.78 (0.72–0.86 [0.32–0.92])	0.001
Dynamic compliance; ml.cmH_2O^{-1}	28 (21–34 [10–77])	29 (21–38 [17–77])	26 (21–33 [15–61])	24 (19–32 [10–49])	0.019
Peak airway pressure; cmH_2O	26 (21–30 [12–42])	25 (20–29 [12–30])	28 (22–30 [14–34])	29 (25–32 [20–42])	< 0.001
Positive-end expiratory pressure; cmH_2O	8 (6–10 [4–16])	8 (5–10 [4–14])	8 (6–10 [4–14])	8 (7–10 [4–16])	0.056
Urine output; ml.kg^{-1}.h^{-1}	0.60 (0.31–0.94 [0–2.70])	0.70 (0.46–1.0 [0–2.40])	0.58 (0.22–0.82 [0–2.70])	0.37 (0.14–0.66 [0–2.60])	< 0.001
Second vaso-active agent	24 (8%)	3 (2%)	9 (10%)	12 (23%)	< 0.001
Prone ventilation	219 (72%)	108 (69%)	70 (74%)	40 (77%)	0.419
Neuromuscular blockade	269 (89%)	136 (86%)	85 (89%)	49 (92%)	0.261
Renal replacement therapy	154 (51%)	68 (43%)	53 (56%)	33 (64%)	0.018
90-day mortality	113 (37%)	35 (22%)	40 (42%)	38 (73%)	< 0.001

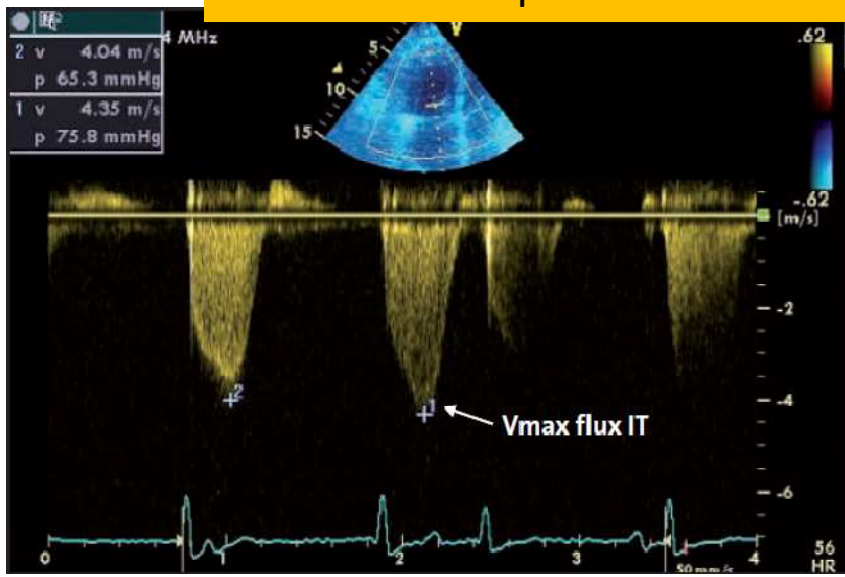
Critères échographiques (ESC 2019)

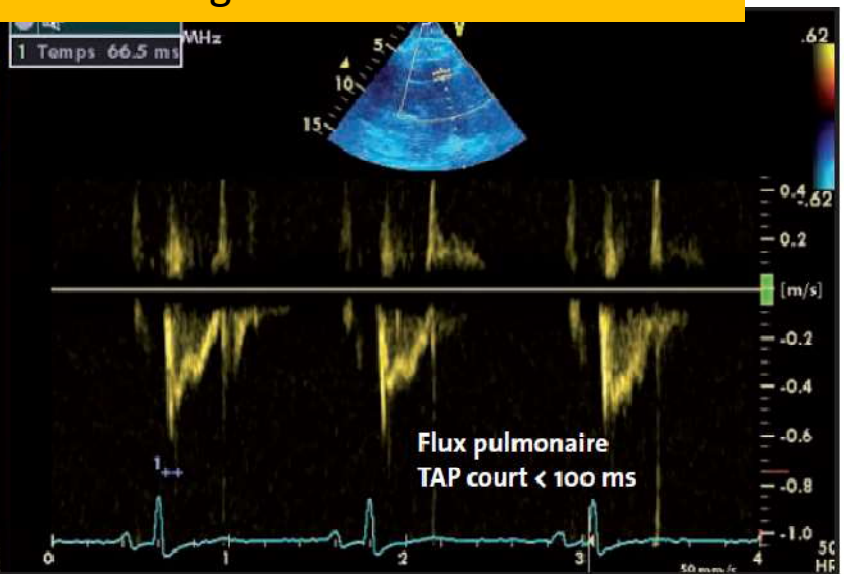






- Géométrie complexe/asynchronisme de contraction
- S' et TAPSE Exploration de la fonction longitudinale du VD





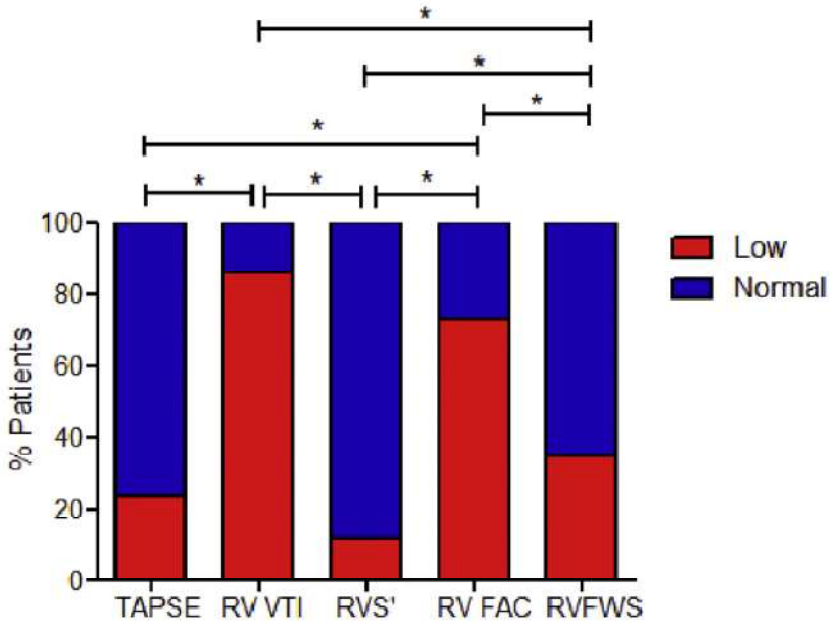
International Journal of Cardiology 327 (2021) 251–258

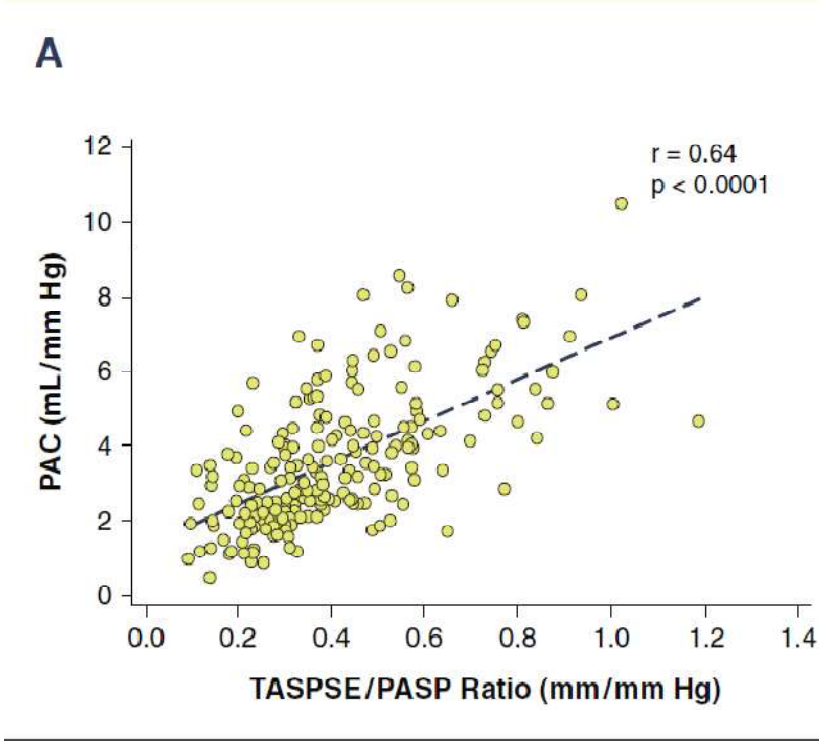
Contents lists available at ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Right ventricular dysfunction in critically ill COVID-19 ARDS





JACC: CARDIOVASCULAR IMAGING

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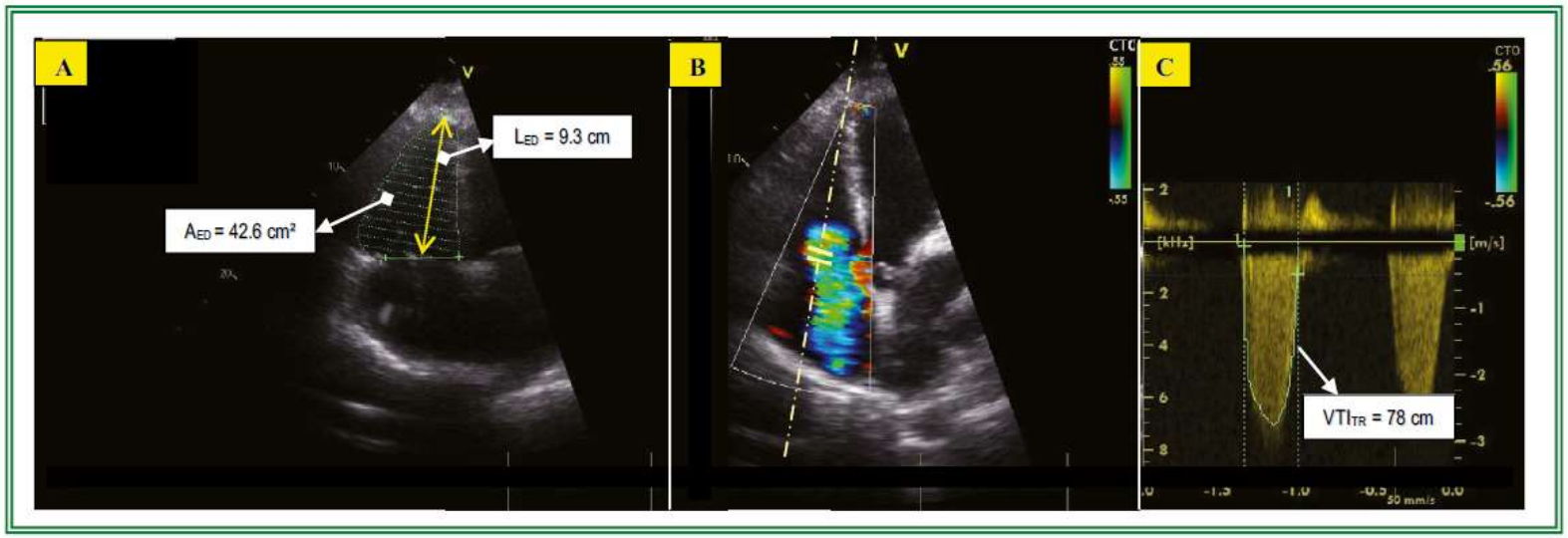
http://dx.doi.org/10.1016/j.jcmg.2016.12.024

Heart Failure Reviews (2022) 27:1325–1339
https://doi.org/10.1007/s10741-021-10108-7

Heart–lung interactions in COVID-19: prognostic impact and usefulness of bedside echocardiography for monitoring of the right ventricle involvement

Michael Dandel[1,2]

Right Ventricular Load adaptation index

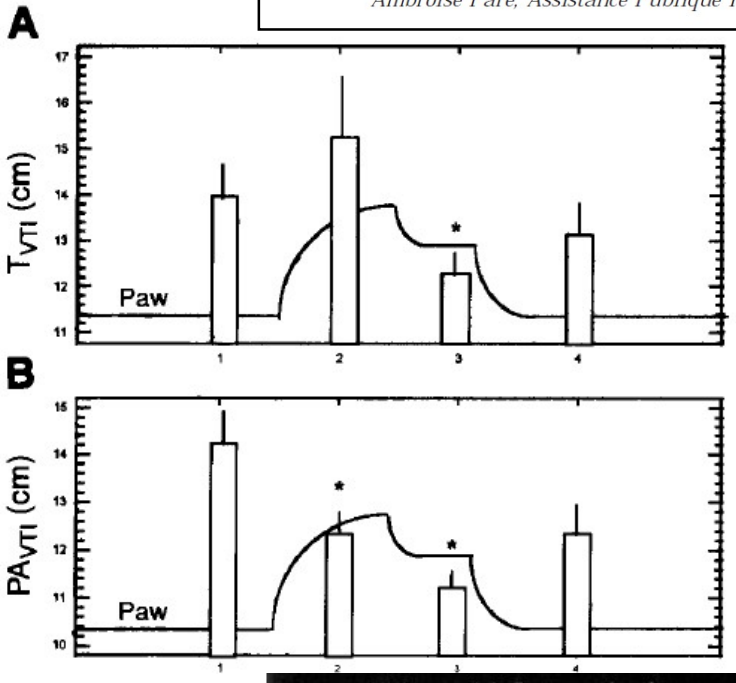


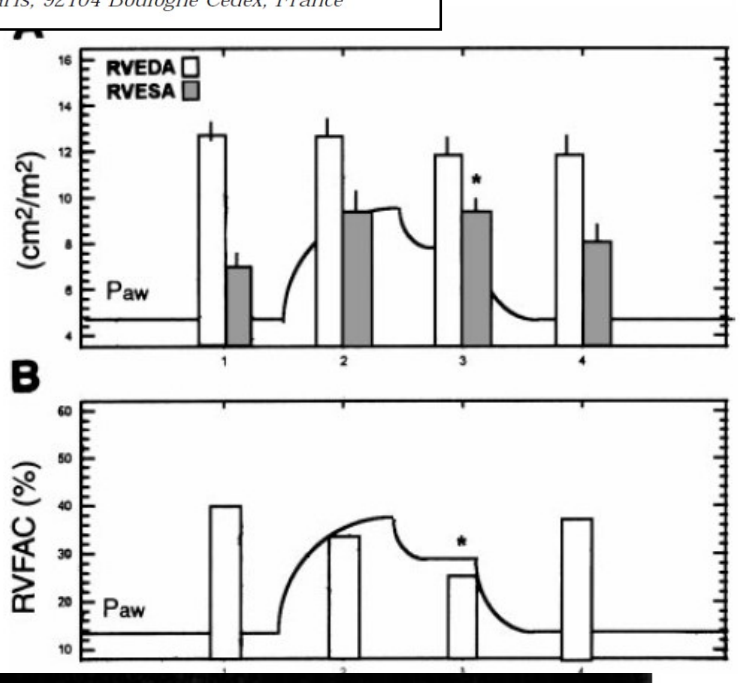
$$LAI_{RV} = \frac{VTI_{TR}\,(cm) \bullet L_{ED}\,(cm)}{A_{ED}\,(cm^2)} = \frac{78\ cm \bullet 9.3\ cm}{42.6\ cm^2} = 17.0$$

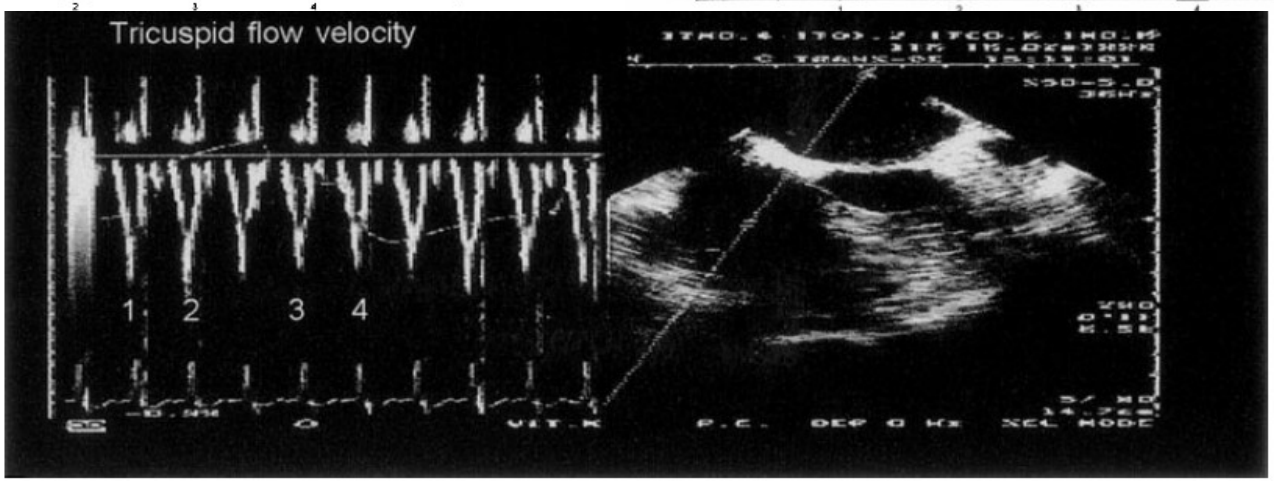
Fig. 2 Calculation of the right ventricular load adaptation index (LAI_{RV}). **a** Measurement of the right ventricular (RV) end-diastolic area (A_{ED}) and long axis lengths in the apical 4 chamber view. **b** and **c** Measurement of the tricuspid regurgitation velocity–time integral (VTI_{TR}) using the continuous wave Doppler. The LAI_{RV} value of 17.0 is reduced and indicates that in this patient with pulmonary arterial hypertension (pulmonary arterial systolic and mean pressure: 48 mmHg and 37 mmHg, respectively), the RV dilation is more pronounced than one would expect on the basis of its present afterload. The limited adaptation possibilities to the increased afterload are exceeded, and a reduction of pulmonary vascular resistance is highly needed in order to prevent further aggravation of RV dysfunction

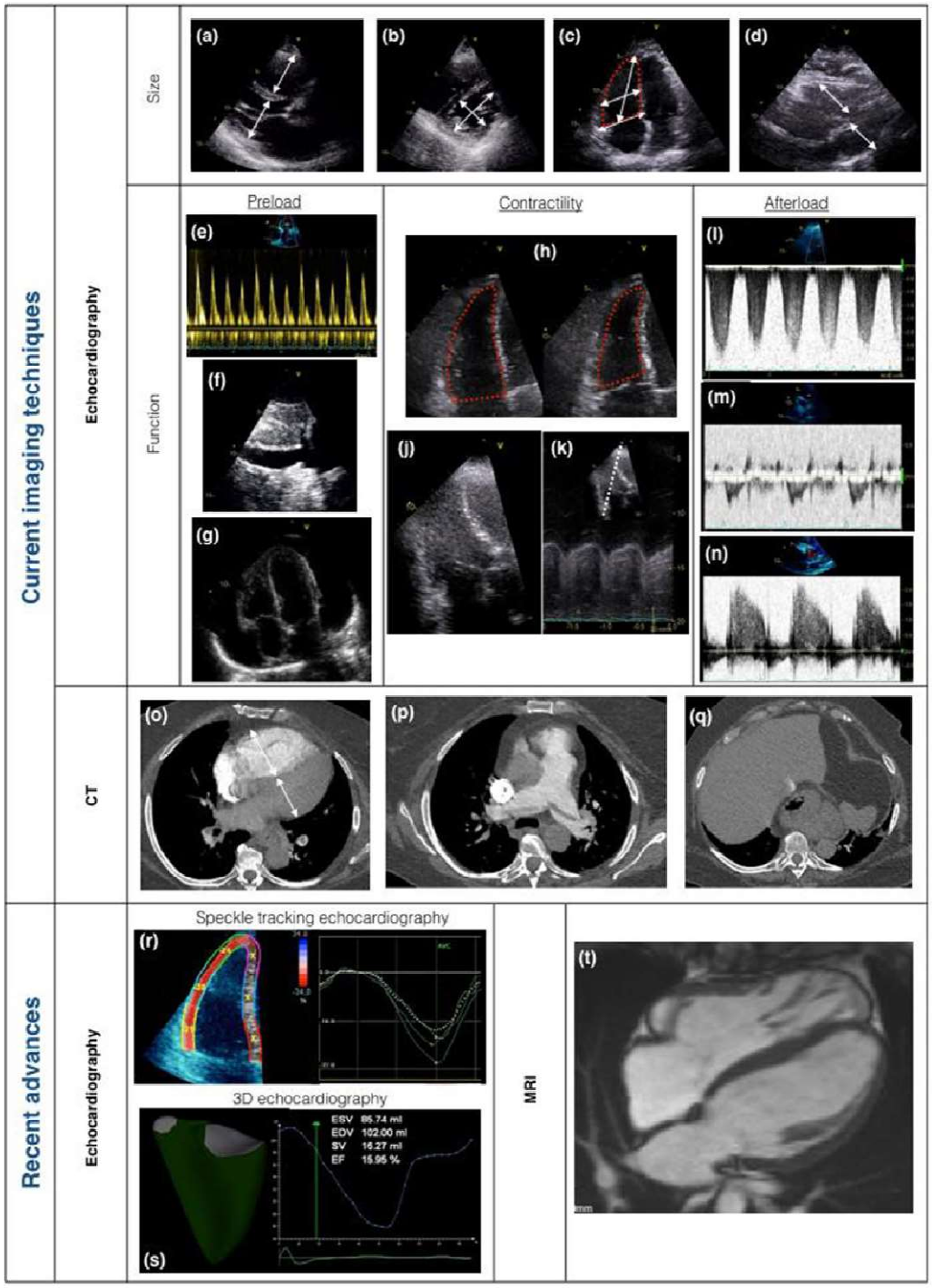
Cyclic changes in right ventricular output impedance during mechanical ventilation

ANTOINE VIEILLARD-BARON,[1] YANN LOUBIERES,[1] JEAN-MARIE SCHMITT,[1]
BERNARD PAGE,[1] OLIVIER DUBOURG,[2] AND FRANÇOIS JARDIN[1]
[1]*Medical Intensive Care Unit and the* [2]*Department of Cardiology, University Hospital Ambroise Paré, Assistance Publique Hôpitaux de Paris, 92104 Boulogne Cedex, France*







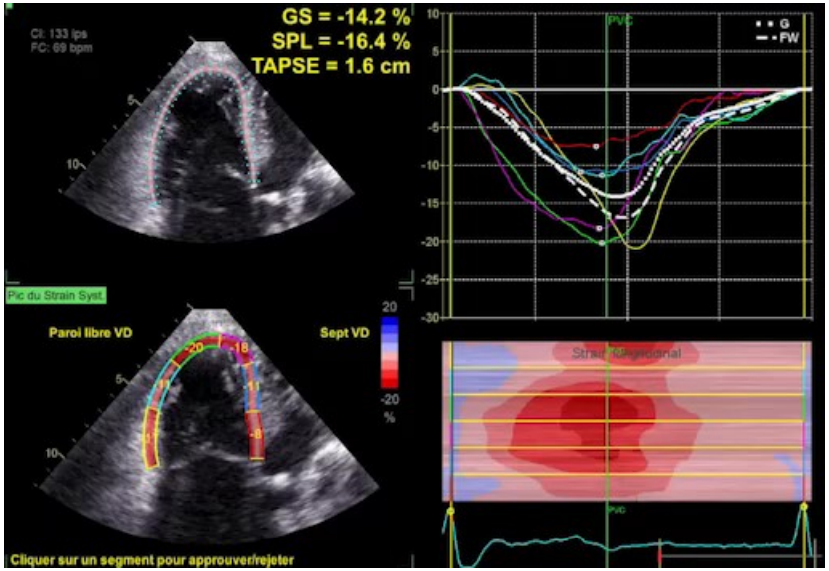


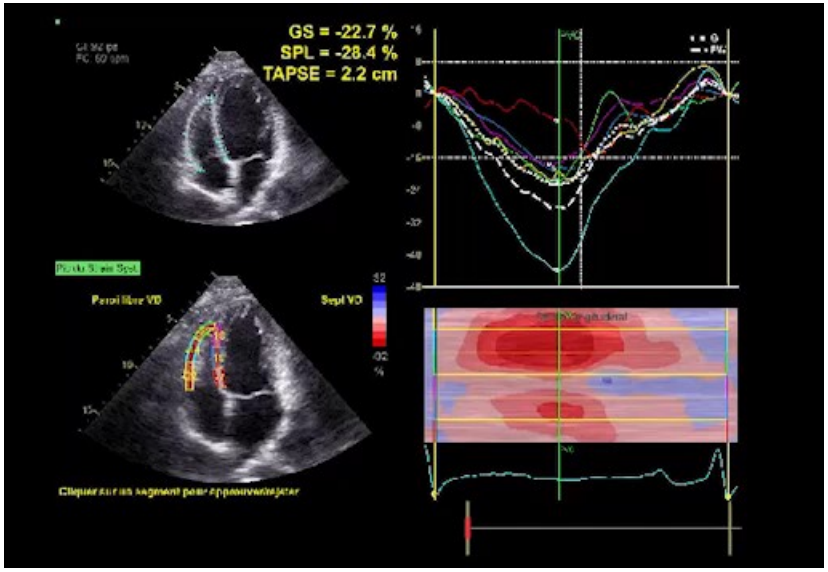
REVIEW

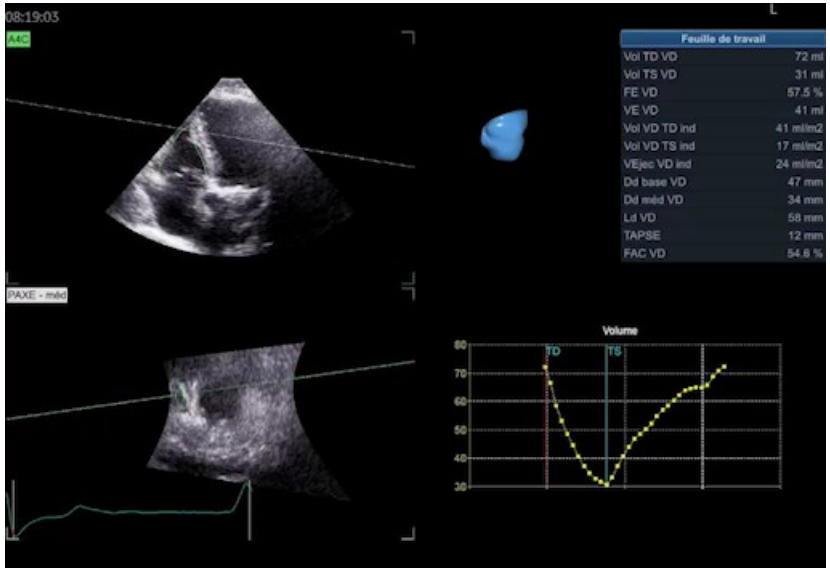
Diagnostic workup, etiologies and management of acute right ventricle failure

A state-of-the-art paper

Antoine Vieillard-Baron[1,2*], R. Naeije[3], F. Haddad[4], H. J. Bogaard[5], T. M. Bull[6], N. Fletcher[7], T. Lahm[8], S. Magder[9], S. Orde[10], G. Schmidt[11] and M. R. Pinsky[12]


GS = -14.2 %
SPL = -16.4 %
TAPSE = 1.6 cm
Pic du Strain Syst
Paroi libre VD
Sept VD
Strain Régional
Cliquer sur un segment pour approuver/rejeter


GS = -22.7 %
SPL = -28.4 %
TAPSE = 2.2 cm
Paroi libre VD
Sept VD


08:19:03
A4C
Feuille de travail
Vol TD VD 72 ml
Vol TS VD 31 ml
FE VD 57.5 %
VE VD 41 ml
Vol VD TD ind 41 ml/m2
Vol VD TS ind 17 ml/m2
VEjec VD ind 24 ml/m2
Dd base VD 47 mm
Dd méd VD 34 mm
Ld VD 58 mm
TAPSE 12 mm
FAC VD 54.6 %
PAXE - méd
Volume
TD
TS

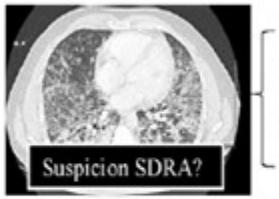
Méd. Intensive Réa (2018) 27:161-171
DOI 10.3166/rea-2018-0019

MISE AU POINT / *UPDATE*

Monitorage hémodynamique dans le SDRA : que savoir en 2018

Hemodynamic Monitoring in ARDS: What to Know in 2018

D. Rousset · B. Riu-Poulenc · S. Silva



Installation < 7 jours

Imagerie compatible

Rapport PaO_2 /FiO_2 altéré avec PEP ≥ 5 cmH_2O

Pas de participation cardiaque majeure au tableau de défaillance respiratoire : **échographie**

SDRA

Instabilité hémodynamique? → **Eliminer CPA : échographie**

+/- TDTP

Evaluation précharge-dépendance
Echo/TDTP : collapsibilité VCS, Test LJP, remplissage titré, TOTE

\+ → Remplissage vasculaire

\- → Vasopresseurs

Recherche défaillance droite ou HTAP sévères : échographie et PVC

\+ → CAP

\- → Echographie et/ou TDTP

- **CAP:**
- Monitorage invasif complet, continu
- mesure RVP, PAPO, PVC, PAP
- Monitorage SvO_2
- **Thermodilution TP:**
- Estimation EPEV, IPV

Sato et al. Crit Care (2021) 25:172
https://doi.org/10.1186/s13054-021-03591-9

Critical Care

RESEARCH Open Access

The impact of right ventricular injury on the mortality in patients with acute respiratory distress syndrome: a systematic review and meta-analysis

Ryota Sato[1], Siddharth Dugar[1,2*], Wisit Cheungpasitporn[3], Mary Schleicher[4], Patrick Collier[5], Saraschandra Vallabhajosyula[6,7,8,9] and Abhijit Duggal[1,2]

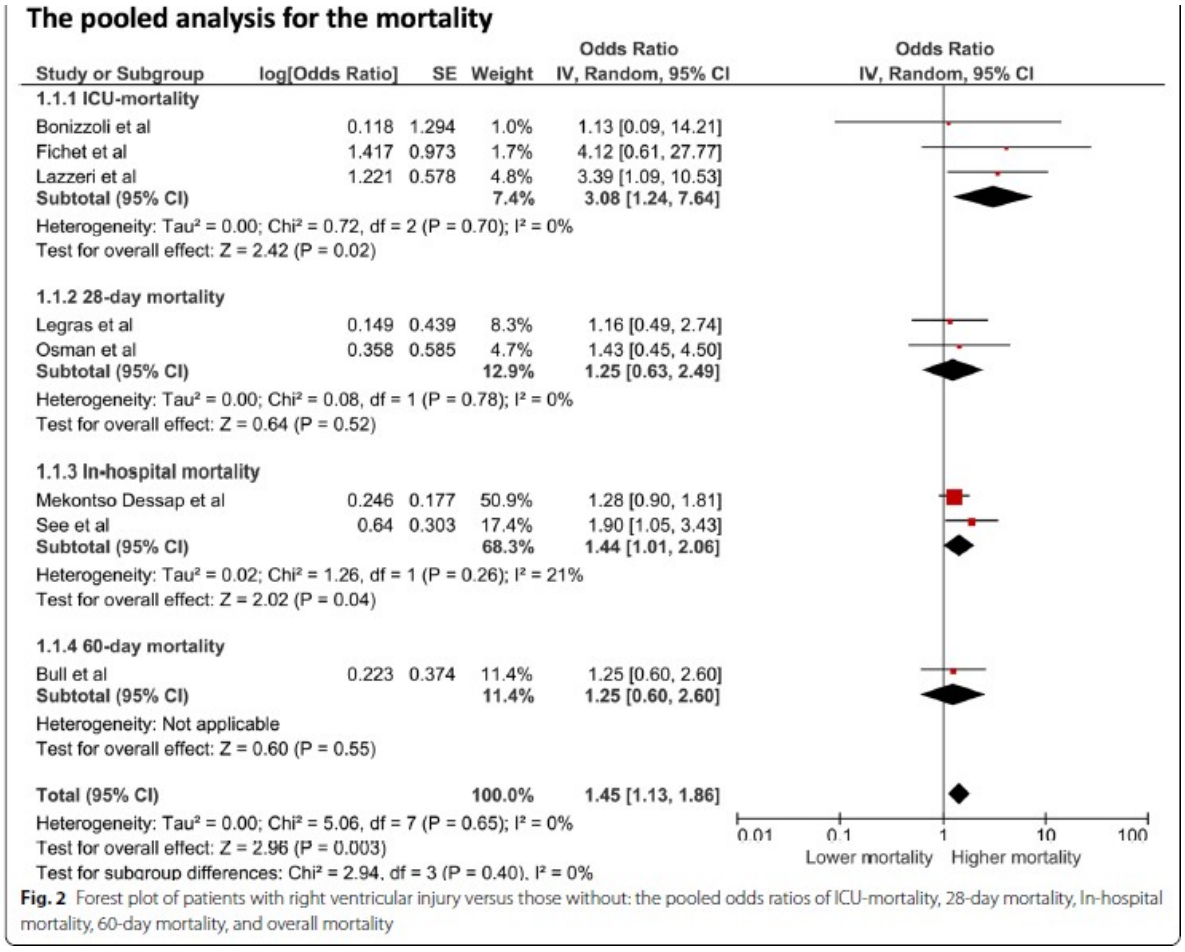
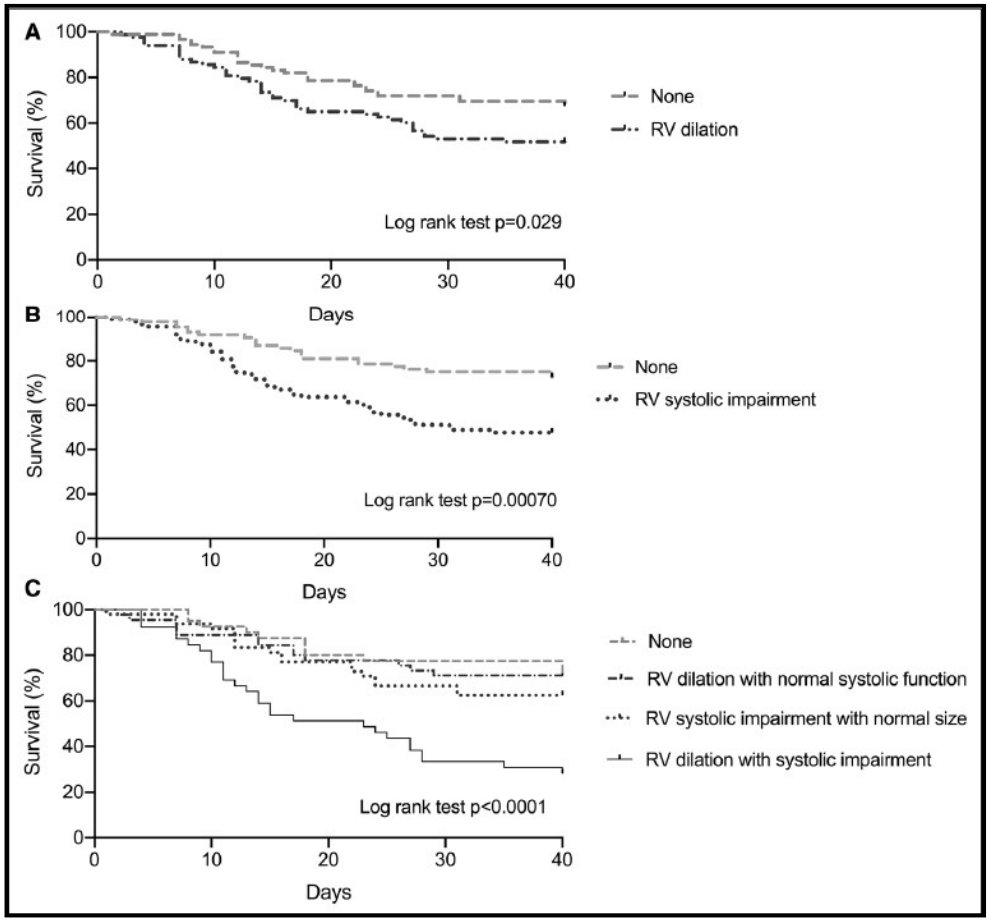
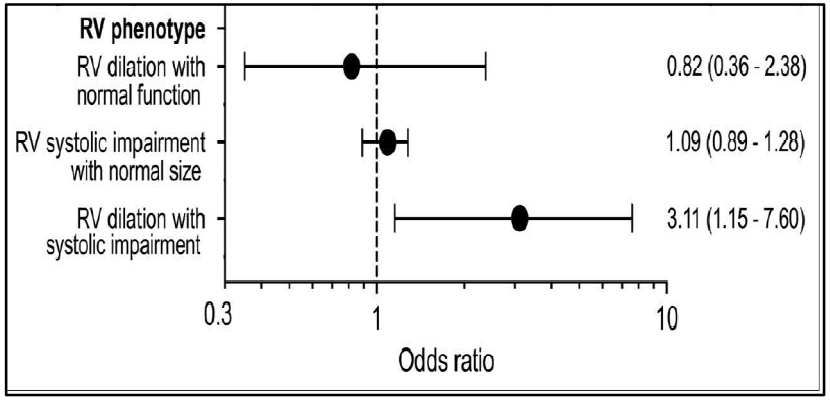


Fig. 2 Forest plot of patients with right ventricular injury versus those without: the pooled odds ratios of ICU-mortality, 28-day mortality, In-hospital mortality, 60-day mortality, and overall mortality

Right Ventricular Dysfunction and Its Association With Mortality in Coronavirus Disease 2019 Acute Respiratory Distress Syndrome*

CCM 2021

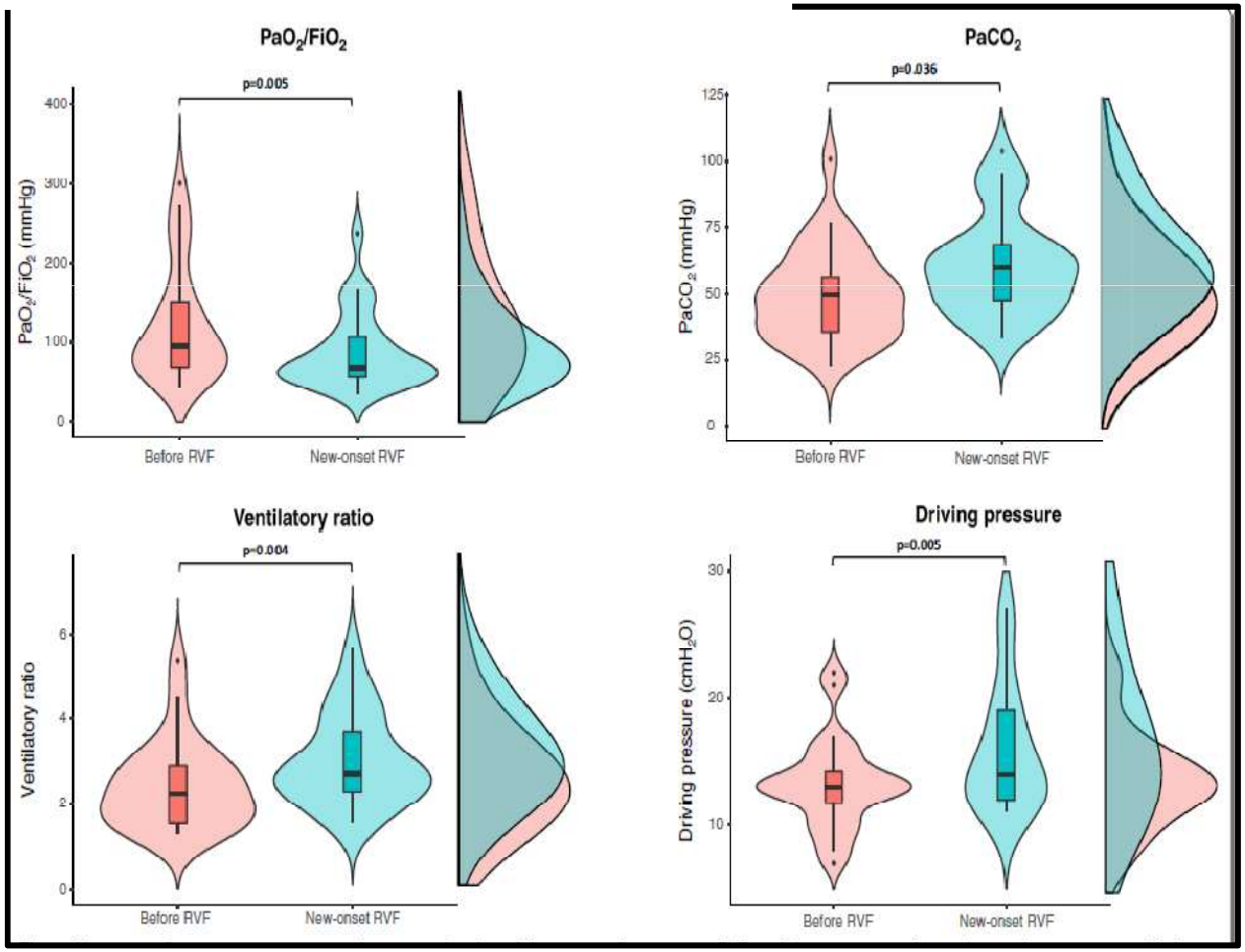




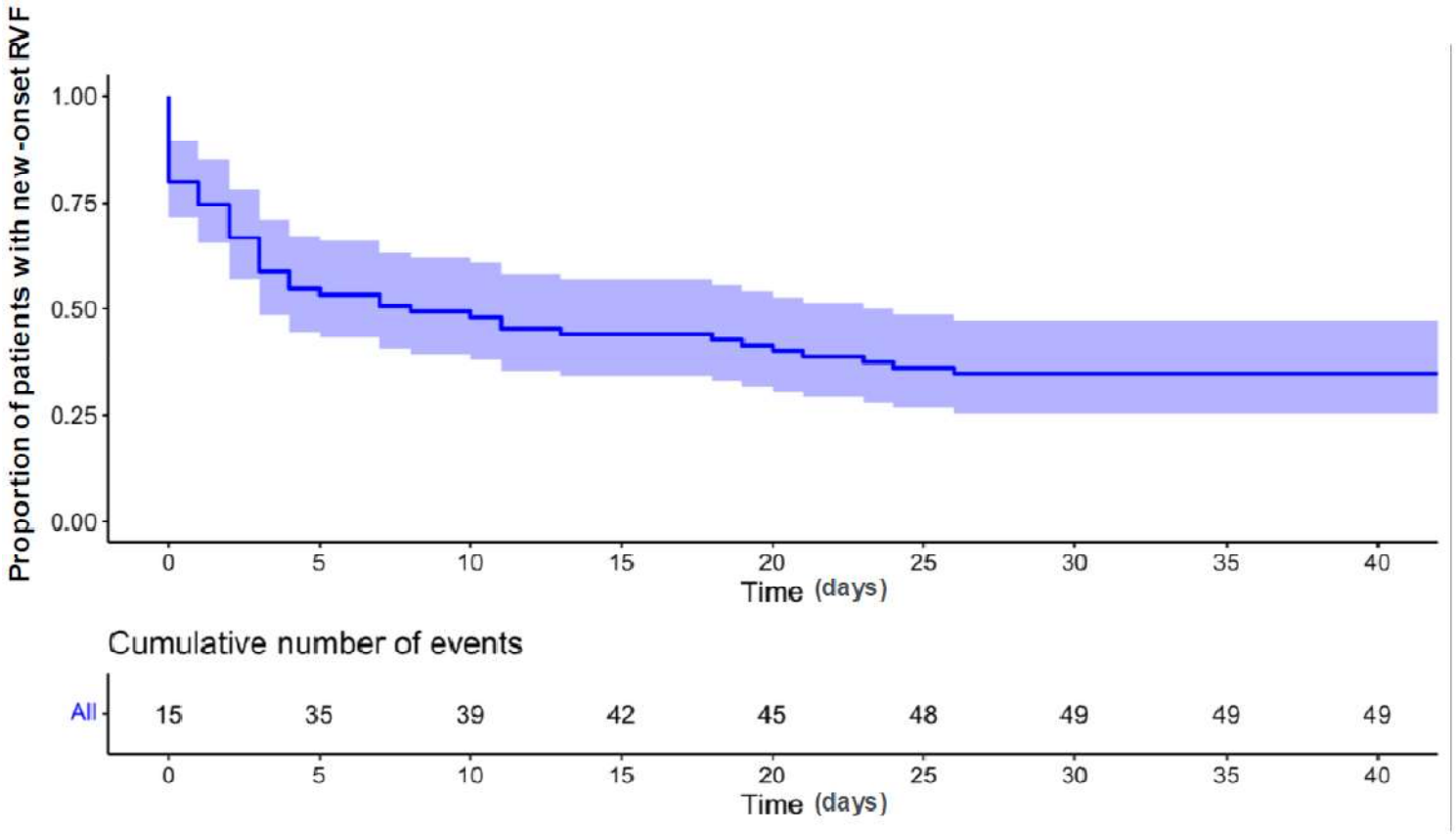
Right ventricular failure is strongly associated with mortality in patients with moderate-to-severe COVID-19-related ARDS and appears related to respiratory worsening

Intensive Care Med (2022) 48:765–767
https://doi.org/10.1007/s00134-022-06730-0

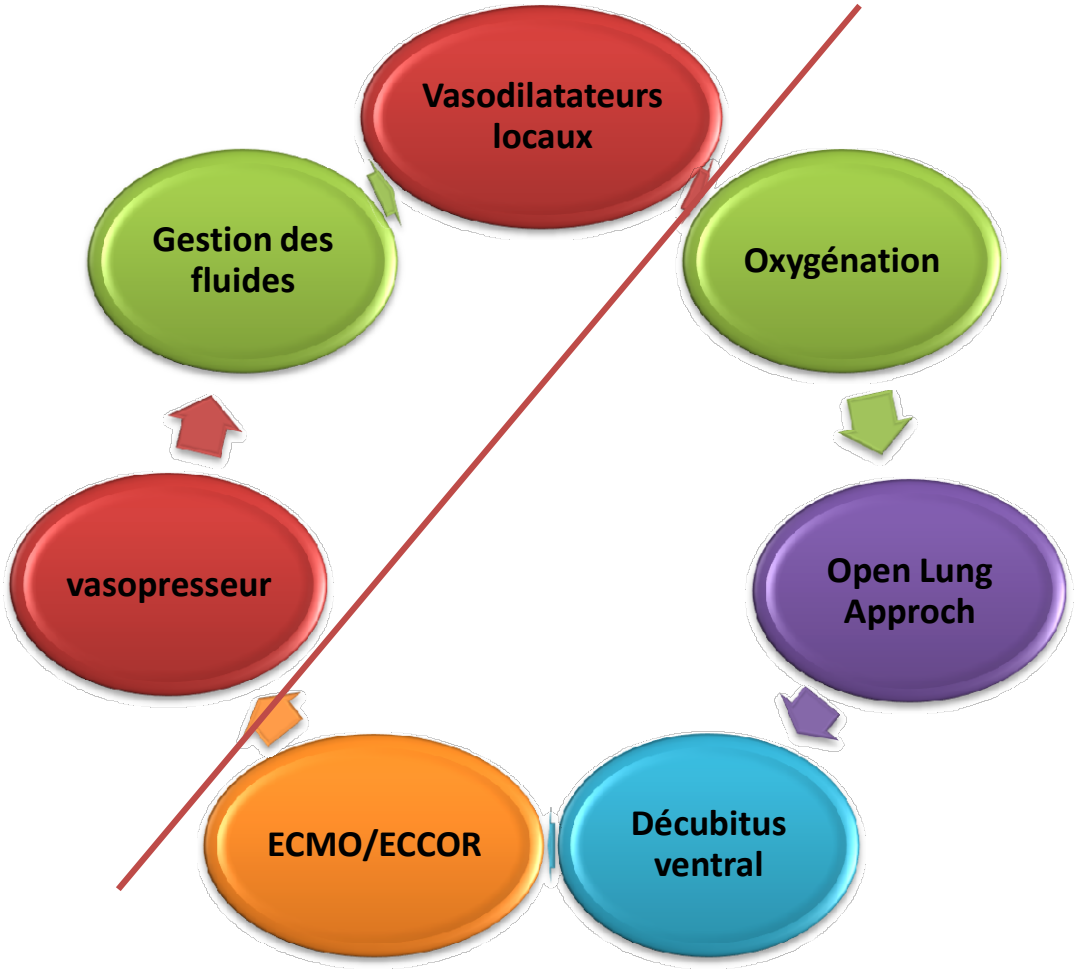
Bruno Evrard[1,2,6*], Marine Goudelin[1,2], Bruno Giraudeau[3], Bruno François[1,2,4] and Philippe Vignon[1,2,4,5]

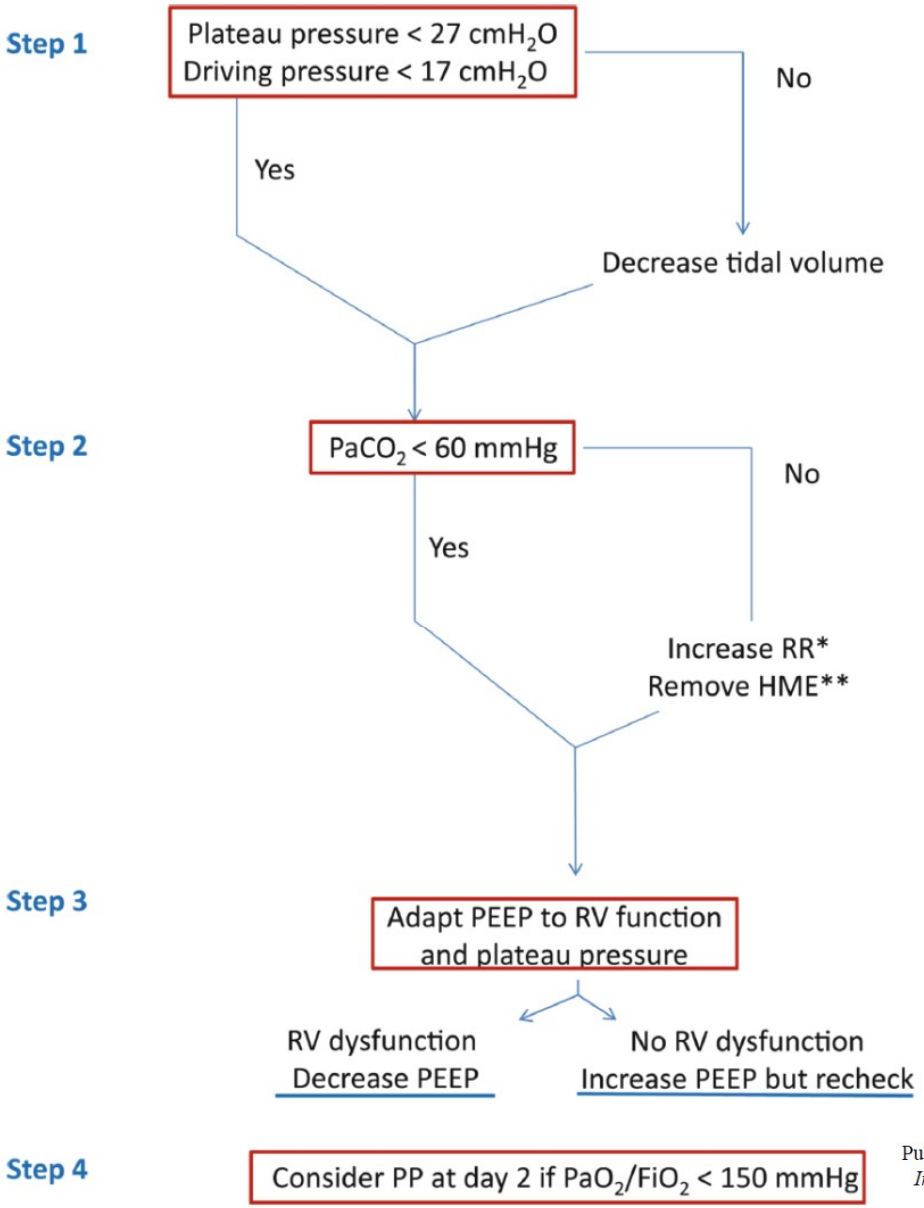


A Time lag between new-onset RVF and initiation of vasopressor



Traitement: approche multimodale



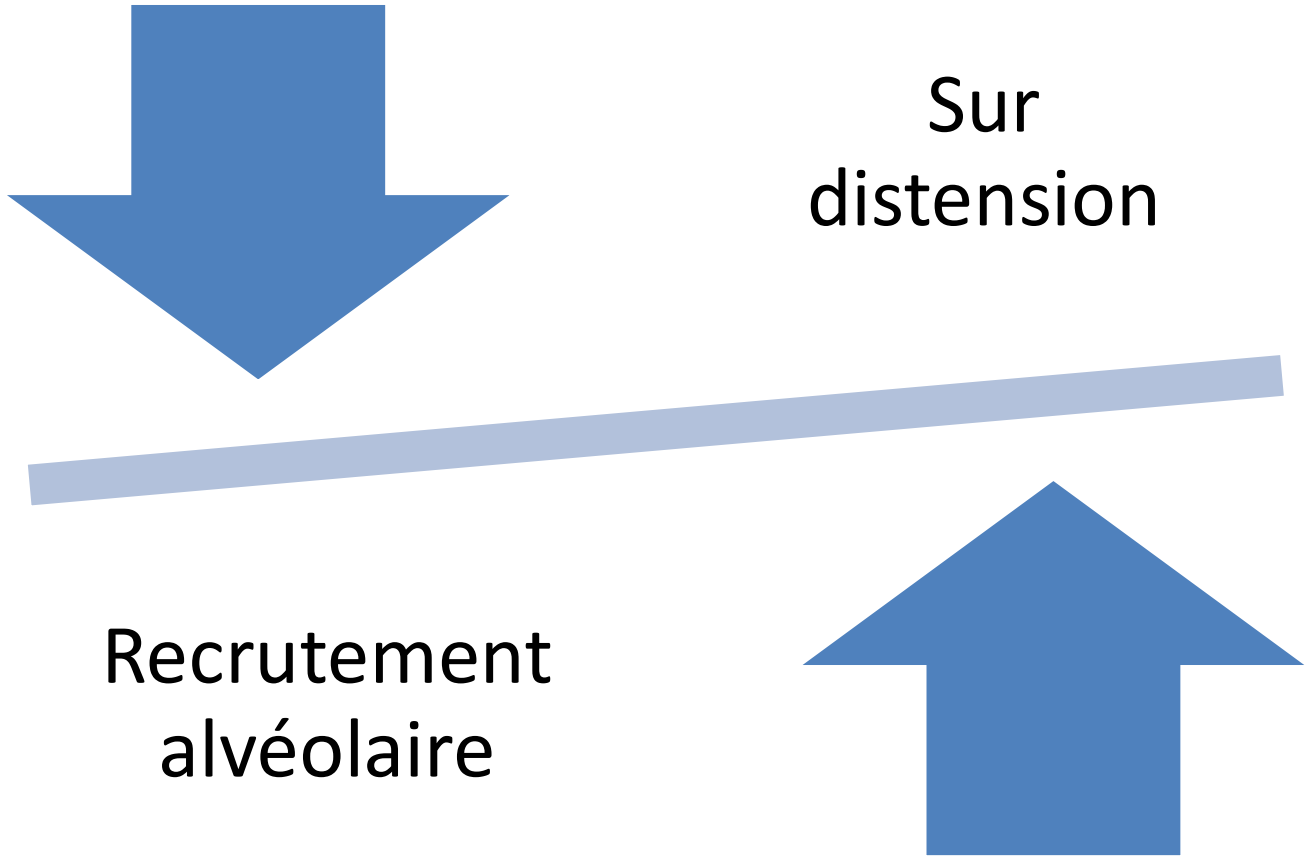


Approche ventilatoire préventive

Published in final edited form as:
Intensive Care Med. 2013 October ; 39(10): 1836–1838. doi:10.1007/s00134-013-3045-2.

Acute cor pulmonale in ARDS

Best PEEP

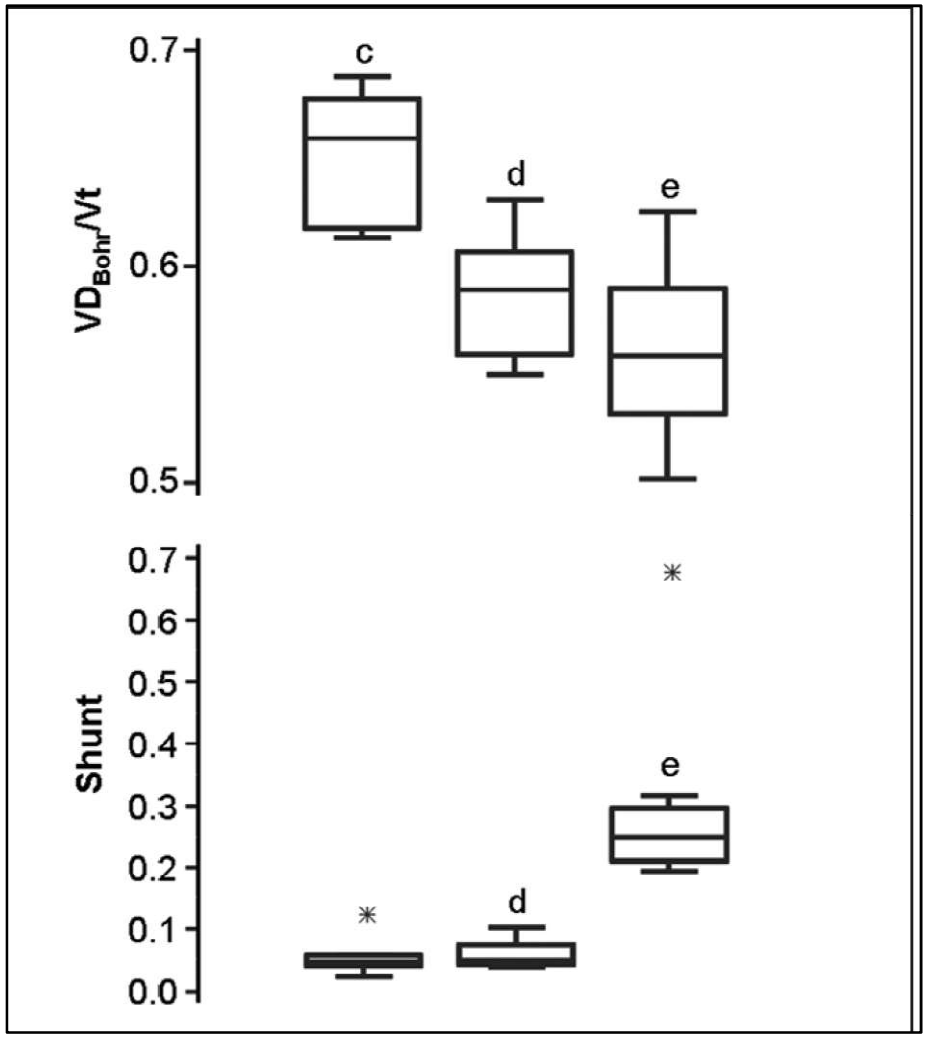


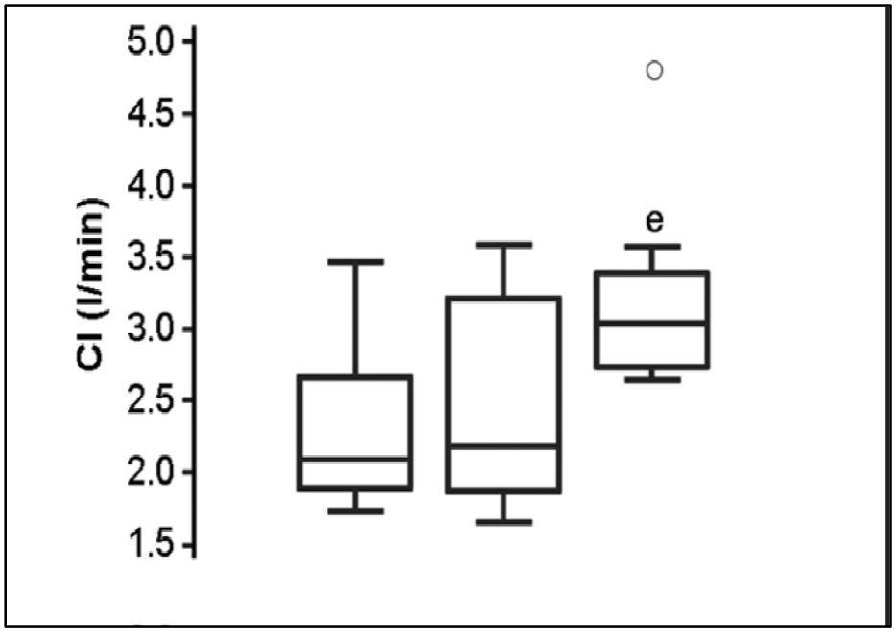
The Open Lung Approach Improves Pulmonary Vascular Mechanics in an Experimental Model of Acute Respiratory Distress Syndrome

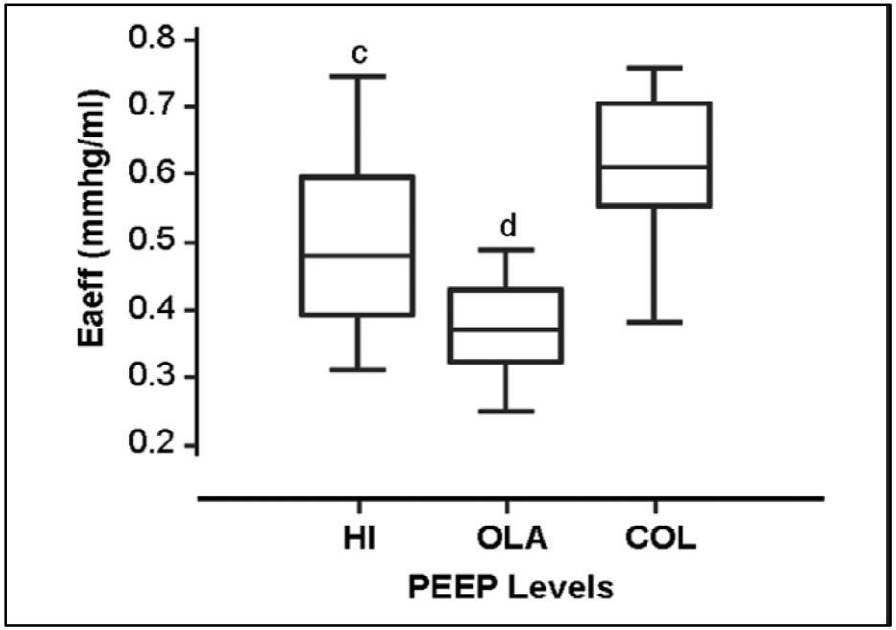
March 2017 • Volume 45 • Number 3

TABLE 2. Systemic Hemodynamic and Oxygen Transport Variables During the Evaluated Experimental Conditions

	Baseline	Acute Respiratory Distress Syndrome	p[a]	Hyperinflation	Open Lung Approach	Collapse
Heart rate (beats/min)	92 (19)	102 (20)	0.288	113 (27)	110 (28)	113 (24)
Mean systemic arterial pressure (mm Hg)	96 (23)	85 (15)	0.505	79 (15)	78 (15)	74 (9)
Central venous pressure (mm Hg)	12 (3)	14 (2)	0.003	17 (3)	16 (2)[b]	14 (4)[c]
Cardiac index (L/min)	3.14 (0.56)	3.74 (0.57)	0.005	2.31 (0.63)	2.47 (0.75)	3.22 (0.72)[c]
Mixed venous hemoglobin oxygen saturation (%)	65 (10)	50 (13)	0.197	50 (13)	55 (12)	58 (17)
O_2 delivery (mL/min)	407 (63)	390 (94)	0.03	293 (73)	306 (87)	380 (71)[c]
O_2 consumption (mL/min)	166 (31)	173 (23)	0.093	151 (22)	148 (20)	149 (56)
O_2 extraction	0.42 (0.1)	0.46 (0.1)	0.002	0.53 (0.12)	0.51 (0.11)	0.4 (0.16)[c]
PEEP (cm H_2O)	7.8 (0.2)	8.6 (1)	< 0.001	22.9 (1.3)[e]	19 (1.3)[c]	10 (1.3)[d]
PPlat (cm H_2O)	14.3 (1.4)[b]	26.3 (3.7)	0.002	33.3 (2)[e]	25.9 (1.6)	23.5 (2.8)[d]
Mean airway pressure (cm H_2O)	10.3 (0.3)[b]	14.8 (1.3)	< 0.001	26.6 (1.6)[e]	21.7 (1.5)[c]	15.5 (1.3)[d]
Driving pressure (DP = PPlat-PEEP) (cm H_2O)	6.4 (1.5)[b]	17.8 (4.2)	0.003	10.3 (1.0)[e]	6.8 (0.8)[c]	13.5 (3.2)
Airway resistance (cm H_2O/L/s)	5.7 (1)[b]	15.1 (2.9)	0.001	7.6 (0.9)[e]	6 (0.8)[c]	9.9 (2.2)[d]
Dynamic respiratory system compliance (mL/cm H_2O)	34 (11)[b]	12 (3)	< 0.001	19 (3)[e]	29 (2)[c]	15 (4)


0.7
0.6
0.5
VD_{Bohr}/Vt
c
d
e
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0.0
Shunt
e
d

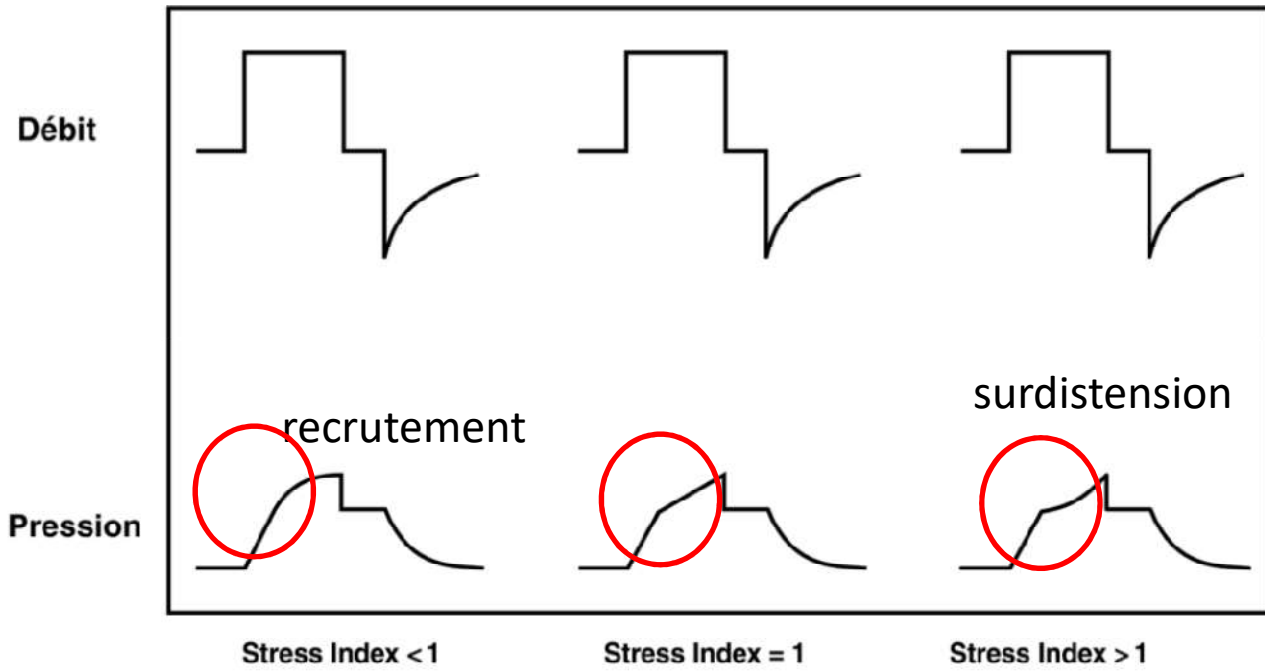

5.0
4.5
4.0
3.5
3.0
2.5
2.0
1.5
CI (l/min)
e


0.8
0.7
0.6
0.5
0.4
0.3
0.2
Eaeff (mmhg/ml)
c
d
HI
OLA
COL
PEEP Levels

Published in final edited form as:
Intensive Care Med. 2008 December ; 34(12): 2291–2299. doi:10.1007/s00134-008-1301-7.

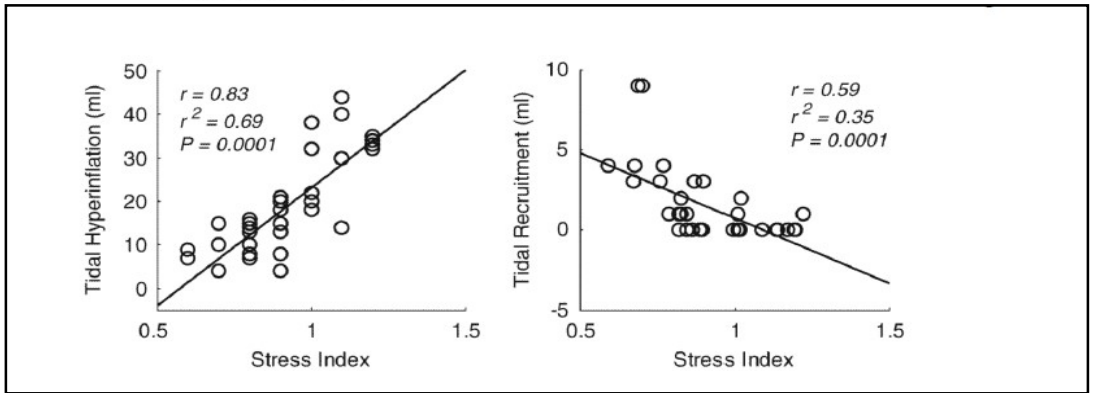
Stress index

Ability of dynamic airway pressure curve profile and elastance for positive end-expiratory pressure titration



Analyse de la courbe de pression d'insufflation en ft du temps en VAC à débit continu

➡ Réflète la compliance du syst resp en VAC

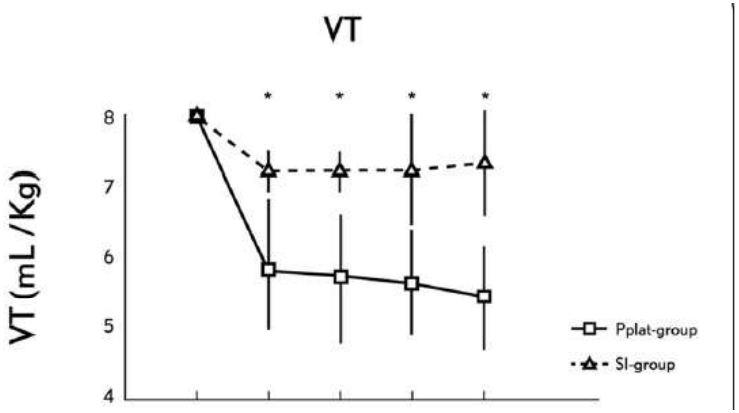


DOI 10.1186/s13054-014-0726-3

CRITICAL CARE

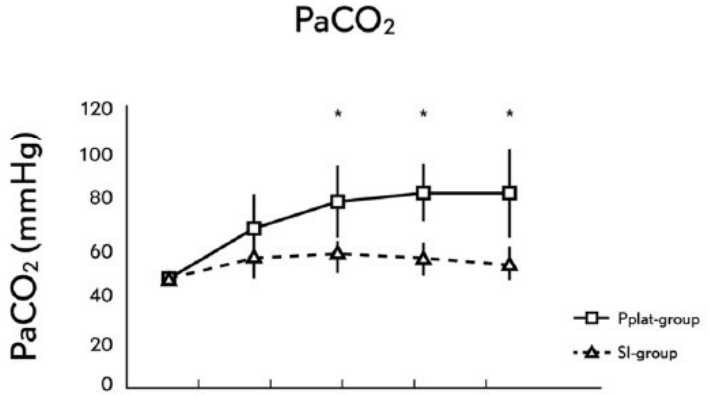
RESEARCH Open Access

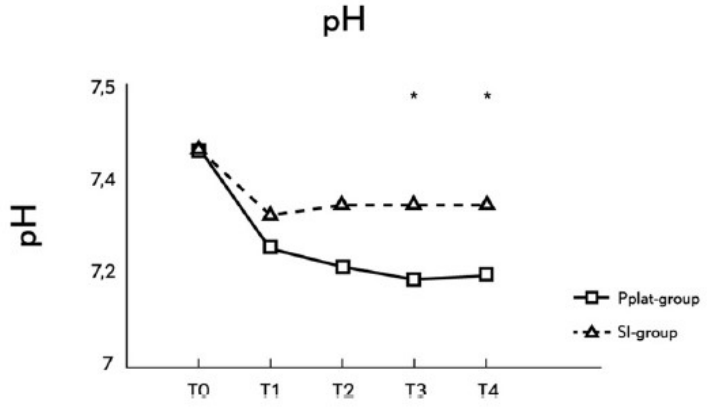
Adjusting tidal volume to stress index in an open lung condition optimizes ventilation and prevents overdistension in an experimental model of lung injury and reduced chest wall compliance

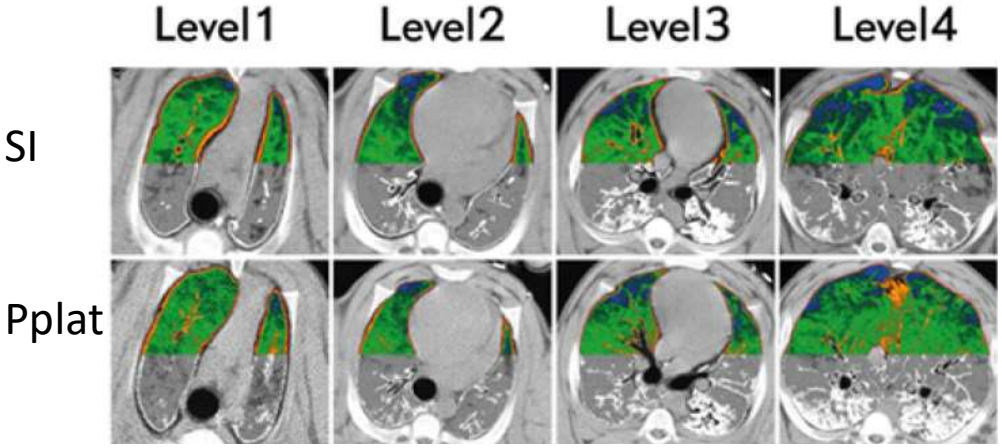


Ajustement volume courant selon 2 statégies

- Pplat= 30 cmH2O
- SI (0,85-1,05)



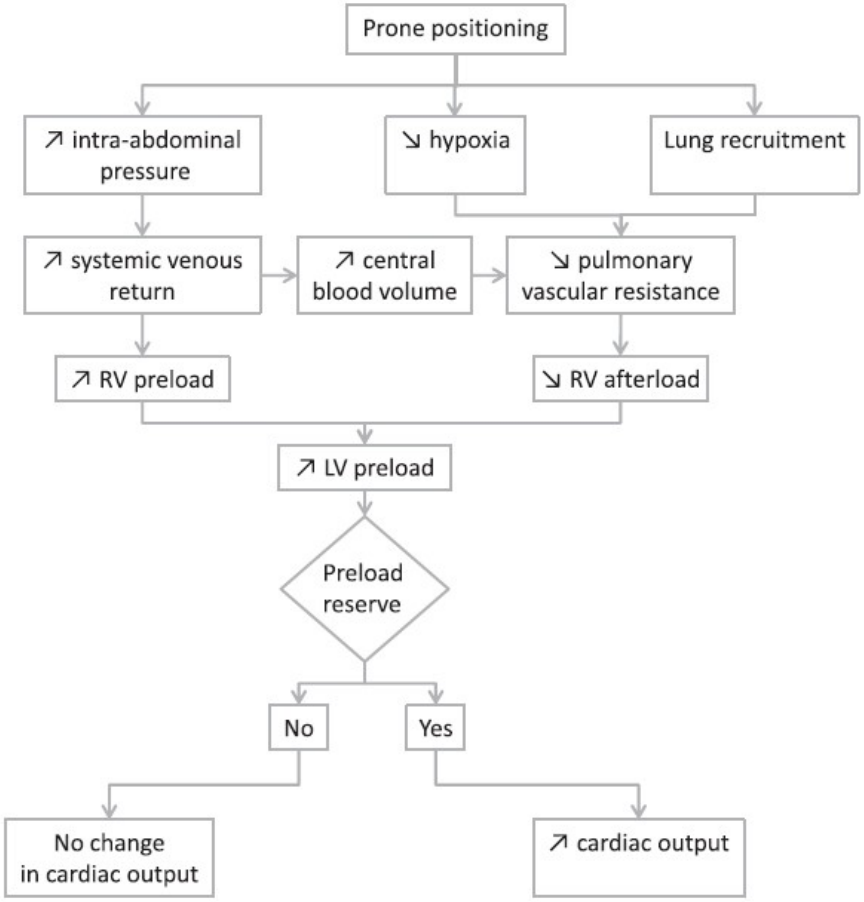


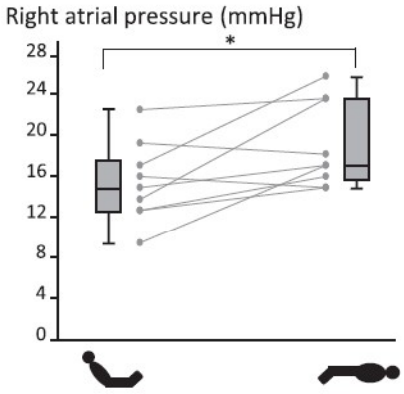


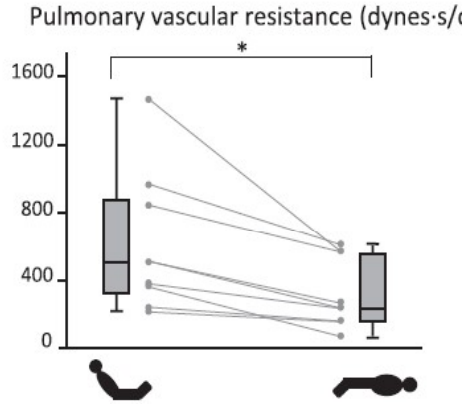
Beneficial Hemodynamic Effects of Prone Positioning in Patients with Acute Respiratory Distress Syndrome

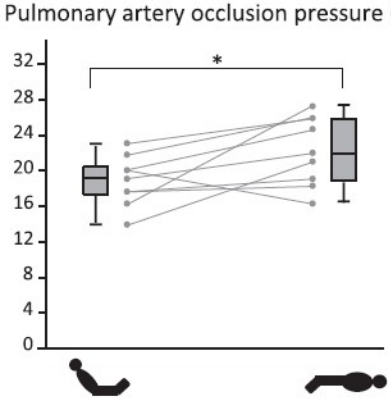
Mathieu Jozwiak[1,2], Jean-Louis Teboul[1,2], Nadia Anguel[1,2], Romain Persichini[1,2], Serena Silva[1,2], Denis Chemla[2,3], Christian Richard[1,2], and Xavier Monnet[1,2]

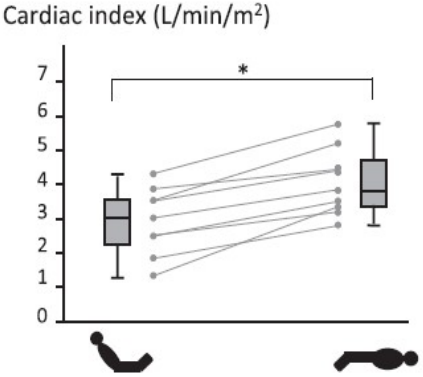
AJRCCM 2013





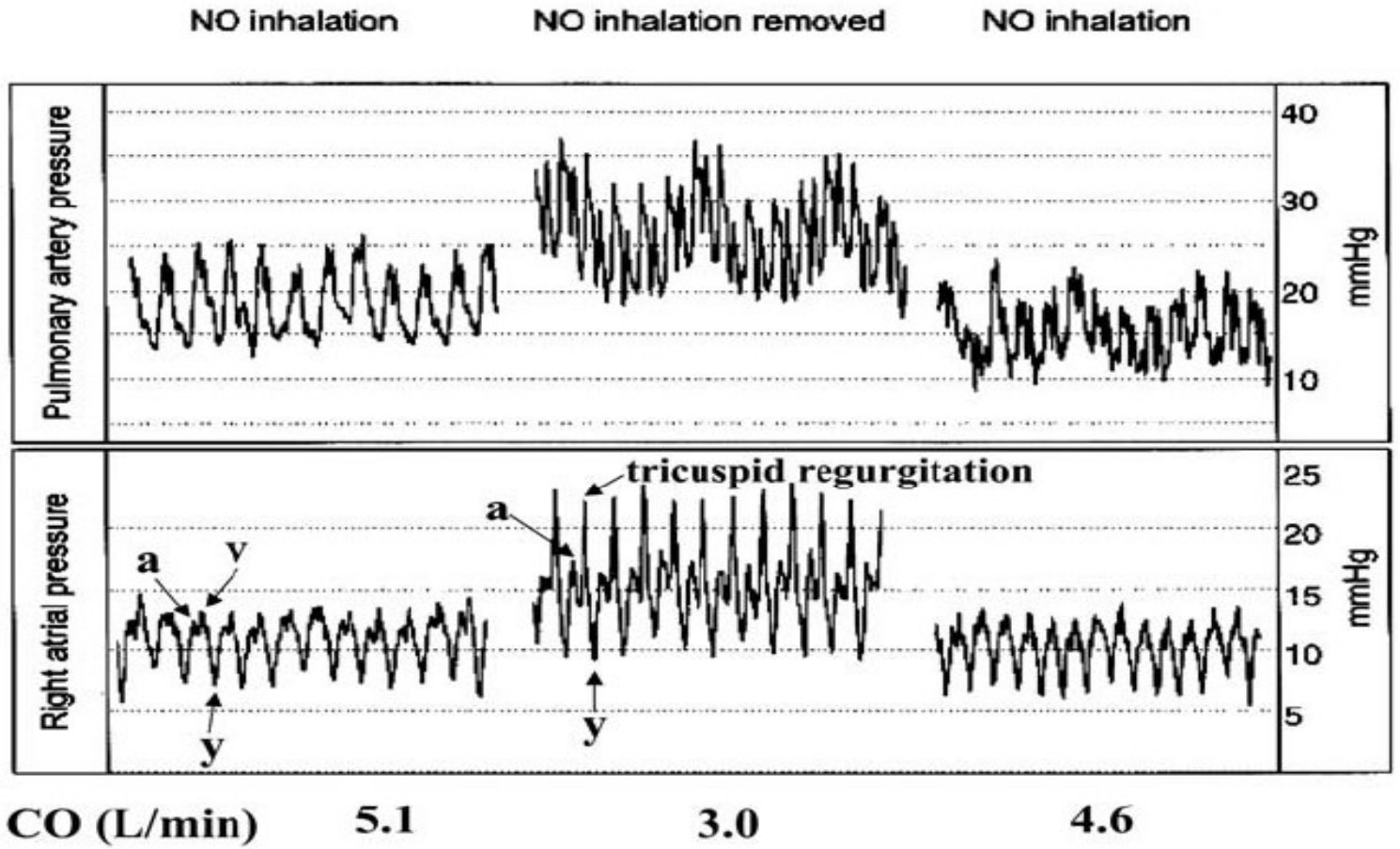






Vasodilatateurs : NO inhalé

Mebazaa , ICM 2004



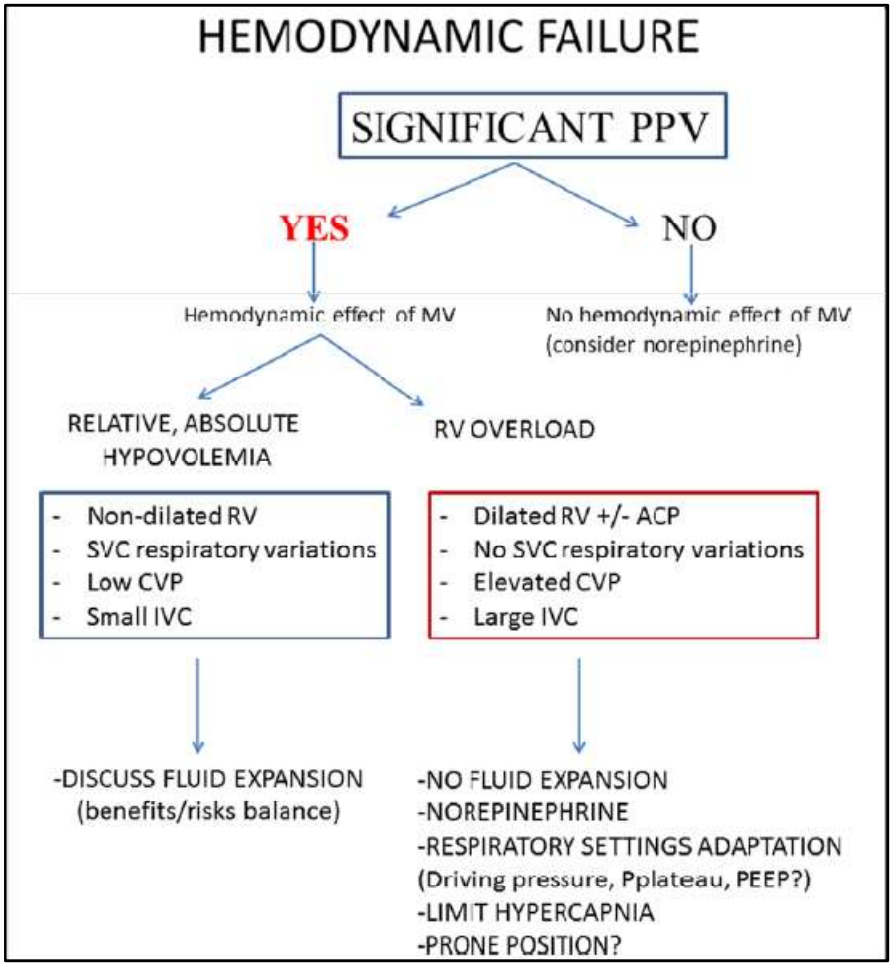
- ↘ shunt et ↘ hypoxie, ↘ PAP, ↘ RVP et ↘ POD, ↘ VTDVD et ↗ FEVD
- Inhalé : moins d'effets systémiques
  ⇨ ↗ DC si dysfonction VD mais plus d' ↗ dès que fonction VD Nl

Intensive Care Med (2016) 42:739–749
DOI 10.1007/s00134-016-4326-3

REVIEW

Experts' opinion on management of hemodynamics in ARDS patients: focus on the effects of mechanical ventilation

A. Vieillard-Baron[1,2,3*], M. Matthay[4], J. L. Teboul[5,6], T. Bein[7], M. Schultz[8], S. Magder[9] and J. J. Marini[10]



Veno-venous extracorporeal CO_2 removal improves pulmonary hemodynamics in a porcine ARDS model

P. Morimont[1,2], J. Guiot[1,2], T. Desaive[2], V. Tchana-Sato[2], N. Janssen[2], A. Cagnina[2], D. Hella[3], F. Blaffart[3], J.-O. Defraigne[2,4], B. Lambermont[1,2]

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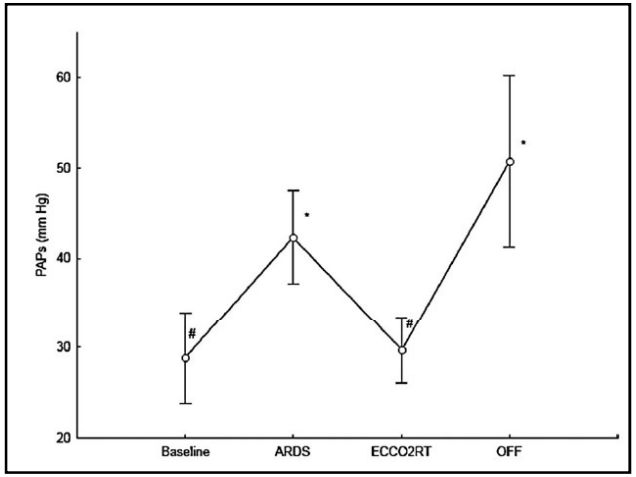
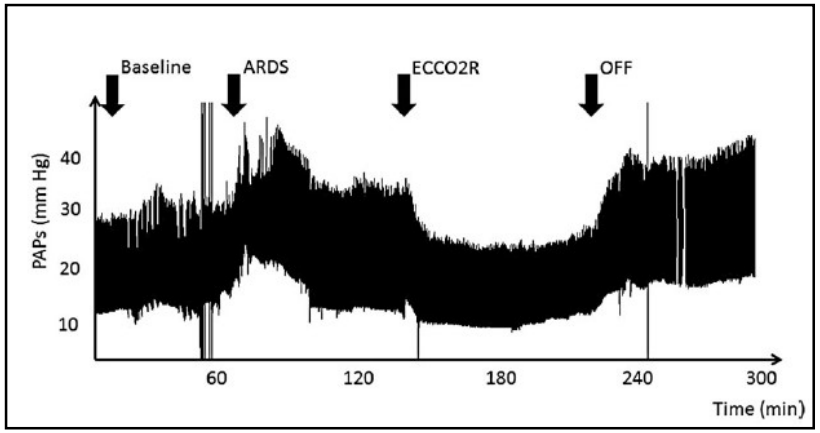
[3]School of Perfusion, University of Liege, Liege, Belgium
[4]Department of Cardiothoracic Surgery, University Hospital of Liege, Liege, Belgium





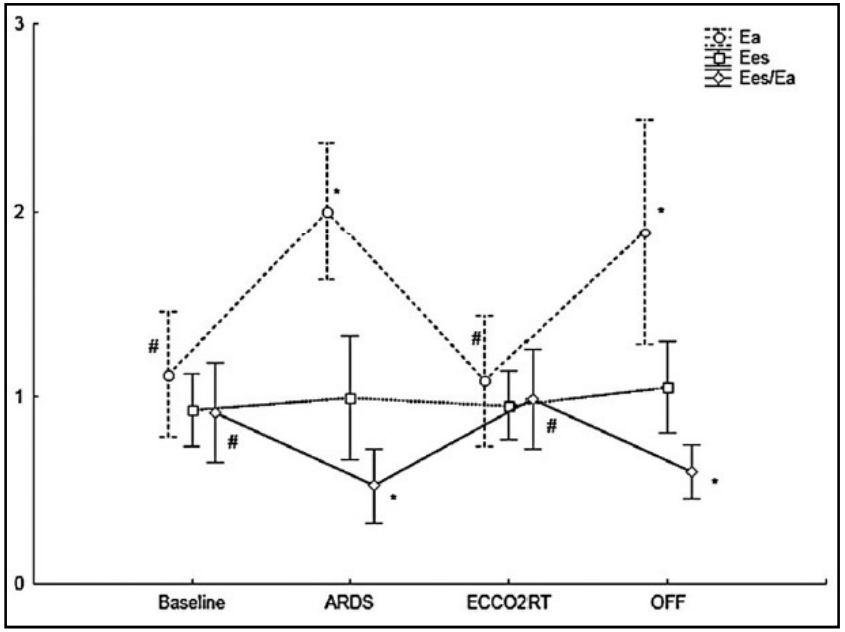


Fig. 3. Evolution of pulmonary arterial elastance (Ea, mmHg/ml), RV end-systolic elastance (Ees, mmHg/ml) and RV ventriculo–arterial coupling index (Ees/Ea).

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Editorial

Protecting the Right Ventricle Network (PRORVNet): Time to Defend the "Forgotten Ventricle"?