

Dysfonction cardiovasculaire liée à la COVID-19



Professeur Yazine MAHJOUR

Réanimation CTVR

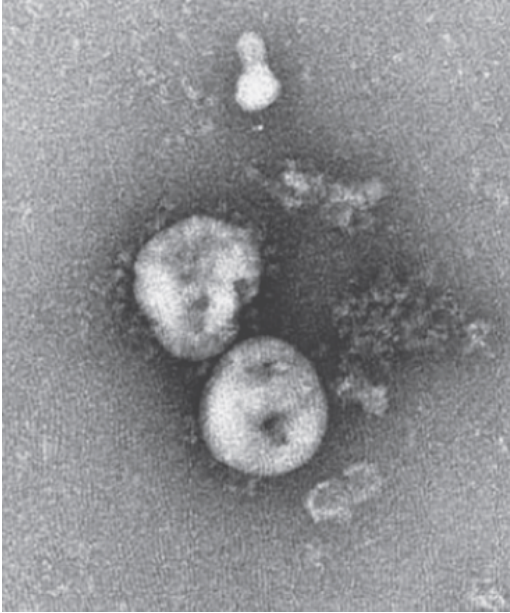
Pôle d'Anesthésie-Réanimation

CHU Amiens-Picardie

AMIENS-FRANCE

Conflit d'intérêt

AUCUN



- Virus à ARN enveloppé appartenant à la famille des *Coronaviridae*, genre betacoronavirus
- Chez l'homme: six espèces de coronavirus connues
 - hCoV saisonniers: 229E, OC43, NL63, HKU1
 - CoV émergents à pathogénicité accrue
 - SRAS -CoV : létalité de 10%
 - MERS-CoV : létalité de 37%

EDITORIAL

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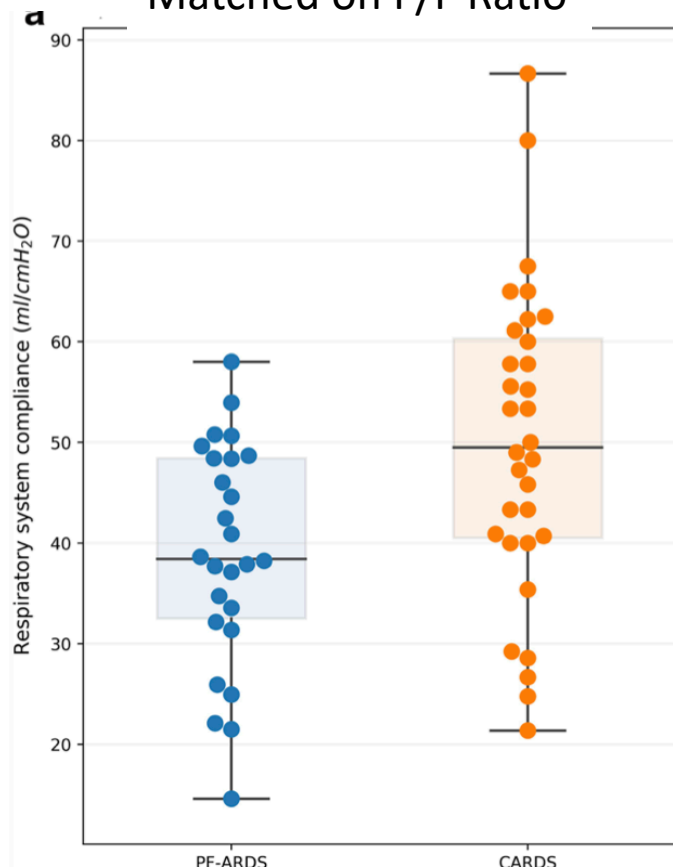
COVID-19 pneumonia: ARDS or not?

Luciano Gattinoni^{1*}, Davide Chiumello² and Sandra Rossi³



Type 1: Near normal pulmonary compliance with isolated viral pneumonia

Matched on P/F Ratio



ORIGINAL

Physiological and quantitative CT-scan characterization of COVID-19 and typical ARDS: a matched cohort study

Davide Chiumello¹, Mattia Busana², Silvia Coppola¹, Federica Romitti², Paolo Formenti¹, Matteo Bonifazi², Tommaso Pozzi¹, Maria Michela Palumbo², Massimo Cressoni³, Peter Herrmann², Konrad Meissner², Michael Quintel², Luigi Camporota⁴, John J. Marini⁵ and Luciano Gattinoni^{2*} 

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Specific features of COVID-19-related ARDS

- **Injury site**
 - Mainly respiratory system
 - Alveolar epithelial cells
- **Specificity of clinical features**
 - Clinical symptoms were inconsistent with the severity of laboratory and imaging findings
 - Clinical manifestations were relatively mild

Differences from ARDS caused by other factors

- **Timing of onset**
 - 8-12 days
- **Respiratory system compliance**
 - Lung compliance might be relatively normal in some COVID-19-related ARDS patients
- **Severity based on oxygenation index**
 - Three categories ($PEEP \geq 5 \text{ cmH}_2\text{O}$)
 - Mild ($200 \text{ mmHg} \leq PaO_2/FiO_2 < 300 \text{ mmHg}$)
 - Mild-moderate ($150 \text{ mmHg} \leq PaO_2/FiO_2 < 200 \text{ mmHg}$)
 - Moderate-severe ($PaO_2/FiO_2 < 150 \text{ mmHg}$)
- **Management protocols**
 - **HFNO**
 - HFNO can be safe even in some moderate-severe patients
 - The timing of invasive mechanical ventilation is very important
 - **Corticosteroids**
 - The effects of corticosteroids were uncertain

al Care

n Access



RESEARCH LETTER

Open Access

PEEP levels in COVID-19 pneumonia



Vasiliki Tsolaki^{1*} , Ilias Siempos², Eleni Magira², Stelios Kokkoris², George E. Zakynthinos¹ and Spyros Zakynthinos²

Table 1 Respiratory and hemodynamic data in 17 mechanically ventilated patients with COVID-19, before and after PEEP reduction

	Before	After	Difference between before and after (95% CI) ^a	P value
PEEP, cm H ₂ O	15.6 ± 1.7	11.1 ± 1.9	4.5 (4.0 to 5.0)	< 0.001
PaO ₂ /FiO ₂ , mm Hg	115.8 ± 30.5	121.9 ± 26.3	− 6.1 (− 13.8 to 1.6)	0.11
PaCO ₂ , mm Hg	49.7 ± 10.4	42.9 ± 5.6	6.8 (3.4 to 10.1)	0.001
Cst, ml/cm H ₂ O	60.0 ± 8.3	63.2 ± 7.4	− 3.2 (− 4.9 to − 1.5)	0.001
Noradrenaline, µg/kg/min	0.21 ± 0.12	0.08 ± 0.08	0.13 (0.09 to 0.16)	< 0.001
MAP, mm Hg	67.2 ± 6.1	69.1 ± 2.6	− 1.9 (− 4.4 to 0.5)	0.11
Fluid balance, ml	1980 ± 764 ^b	942 ± 331 ^c	1039 (760 to 1317)	< 0.001

Data are expressed as mean ± standard deviation

^aData are expressed as mean difference [95% confidence interval (CI)]; ^bfluid balance during 12 h before PEEP reduction; ^cfluid balance during 12 h following PEEP reduction

PEEP positive end-expiratory pressure, PaO₂ partial pressure of arterial oxygen, FiO₂ fraction of inspired oxygen, PaCO₂ partial pressure of arterial carbon dioxide, Cst static compliance of the respiratory system, MAP mean arterial pressure

LETTER

Open Access

Severe Covid-19 disease: rather AVDS than ARDS?



Yazine Mahjoub^{1*}, Daniel Oscar Rodenstein² and Vincent Jounieaux³

REVIEW

Open Access

COVID-19

The vascular endothelium: the cornerstone of organ dysfunction in severe SARS-CoV-2 infection

Laure-Anne Teuwen^{1,2*}

Stéphanie Pons^{1,2}, Sofiane Fodil³, Elie Azoulay³ and Lara Zafrani^{1,3*}

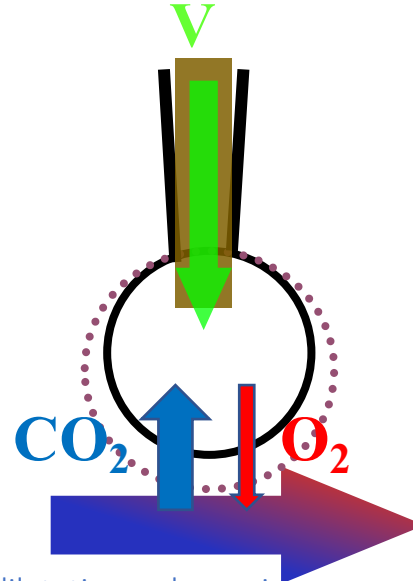
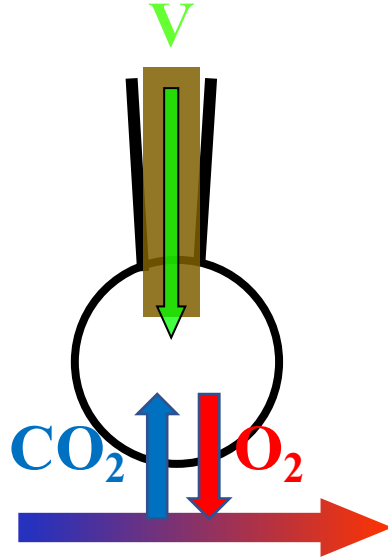
On the basis of emerging evidence ... cells are essential contributors to the initiation ... discuss current insights into the link between endothelial ... changes and propose novel therapeutic strategies.



Facing COVID-19: endothelial dysfunction, thrombosis, and dysregulation of inflammation

Daniel E. Leisman¹, Clifford S. Deutschman² and Matthieu Legrand^{3*}

Hypothèse physiopathologique



Vasodilatation pulmonaire responsable d'un
shunt intrapulmonaire
(syndrome hépato-pulmonaire)
Hypoxémie, hyper débit cardiaque ? ↓RVP ?

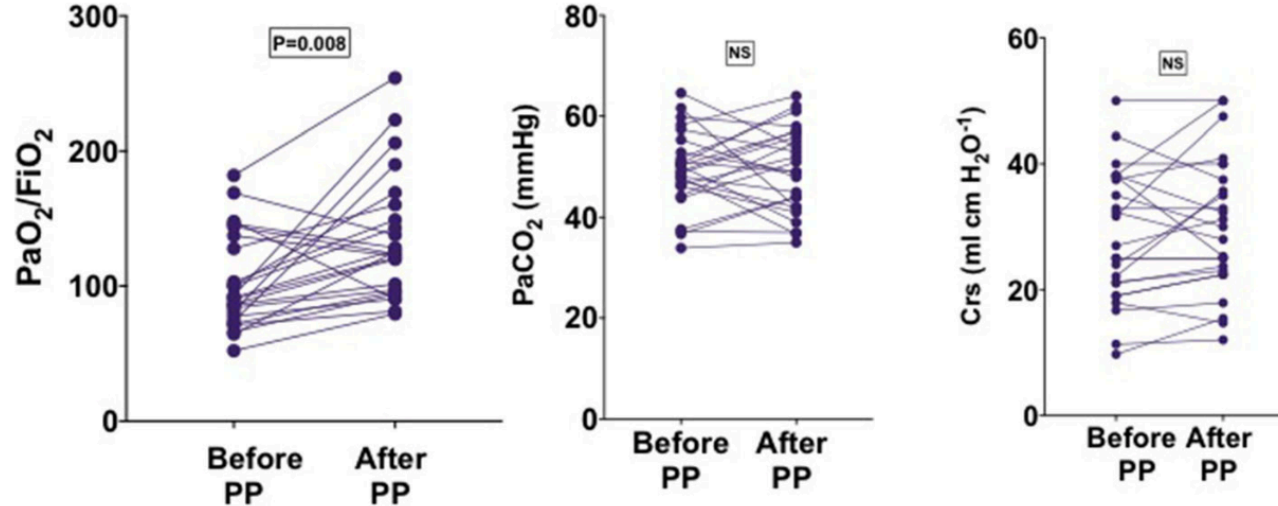
Hypoxemia and prone position in mechanically ventilated COVID-19 patients: a prospective cohort study

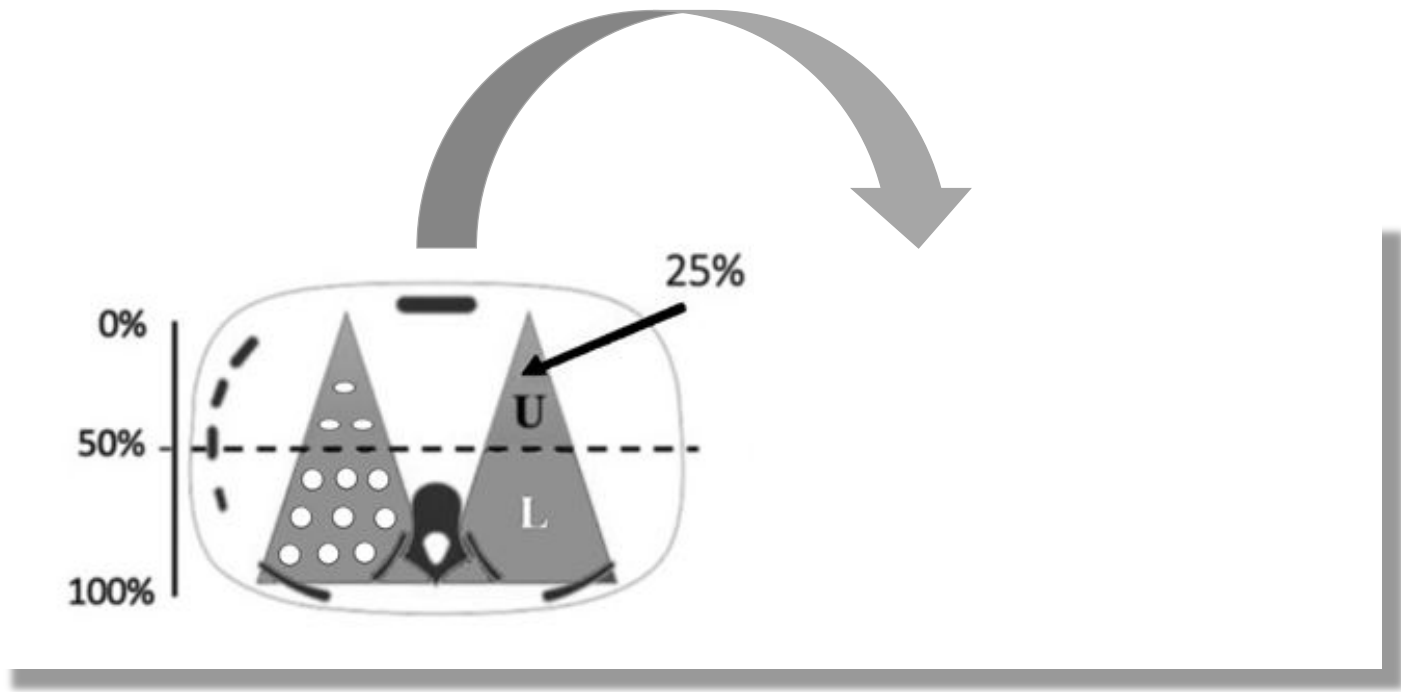
Osama Abou-Arab, MD, PhD · Guillaume Haye, MD · Christophe Beyls, MD ·
Pierre Huette, MD · Pierre-Alexandre Roger, MD · Mathieu Guilbart, MD ·
Michaël Bernasinski, MD · Patricia Besserve, MD · Faouzi Trojette, MD ·
Hervé Dupont, MD, PhD · Vincent Jounieaux, MD, PhD · Yazine Mahjoub, MD,
PhD



Canadian Journal
of Anesthesia

Can J Anesth/J Can Anesth (2021)
68:262-263
DOI 10.1007/s12630-020-01844-9





Modification du shunt due à la forme conique des hémithorax



Inhaled nitric oxide for critically ill Covid-19 patients: a prospective study

Abou-Arab *et al. Crit Care* (2020) 24:645
<https://doi.org/10.1186/s13054-020-03371-x>

Osama Abou-Arab^{1*} , Pierre Huette¹, Fanny Debouvries¹, Hervé Dupont¹, Vincent Jounieaux² and Yazine Mahjoub¹

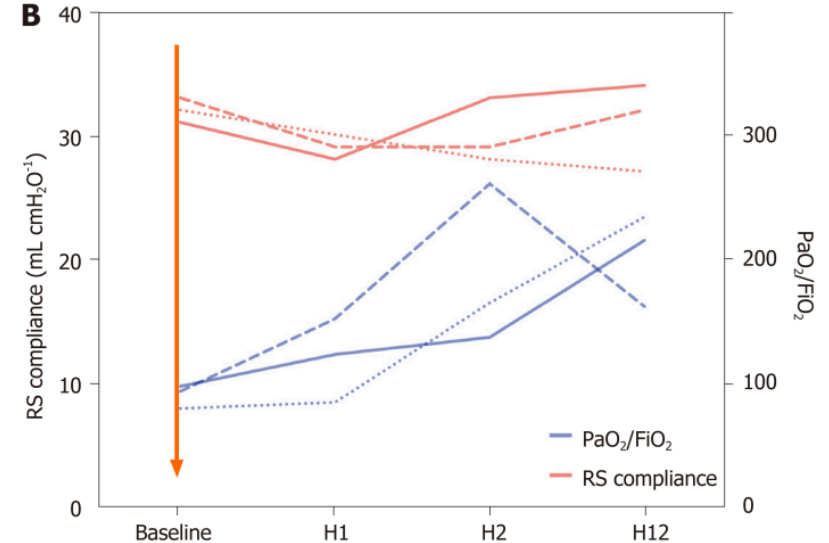
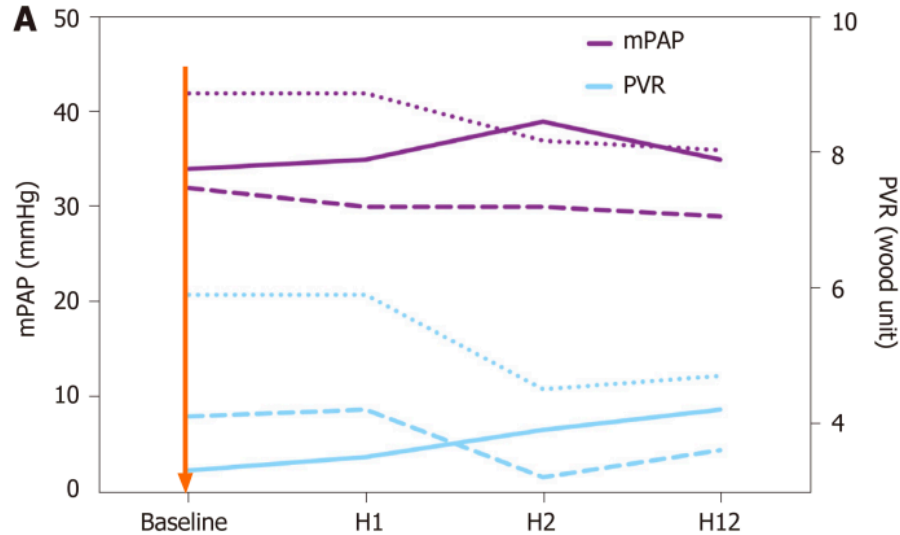
Variables	Non-responder (<i>n</i> = 12)	Response rate 65%	<i>P</i> value
PaCO ₂ (mmHg)			
Baseline	49 [36–56]	48 [42–60]	0.363
After iNO	47 [42–60]	47 [38–52]	0.444
PaO ₂ /FiO ₂			
Baseline	134 [83–173]	70 [63–100]	<u>< 0.0001</u>
After iNO	125 [92–144]	144 [107–175]	0.068
<i>P</i> value before/after iNO	0.005	< 0.0001	

Almitrine for COVID-19 critically ill patients – a vascular therapy for a pulmonary vascular disease: Three case reports

World J Clin Cases 2021 May 16; 9(14): 3385-3393

World J Clin Cases 2021 May 16; 9(14): 3385-3393

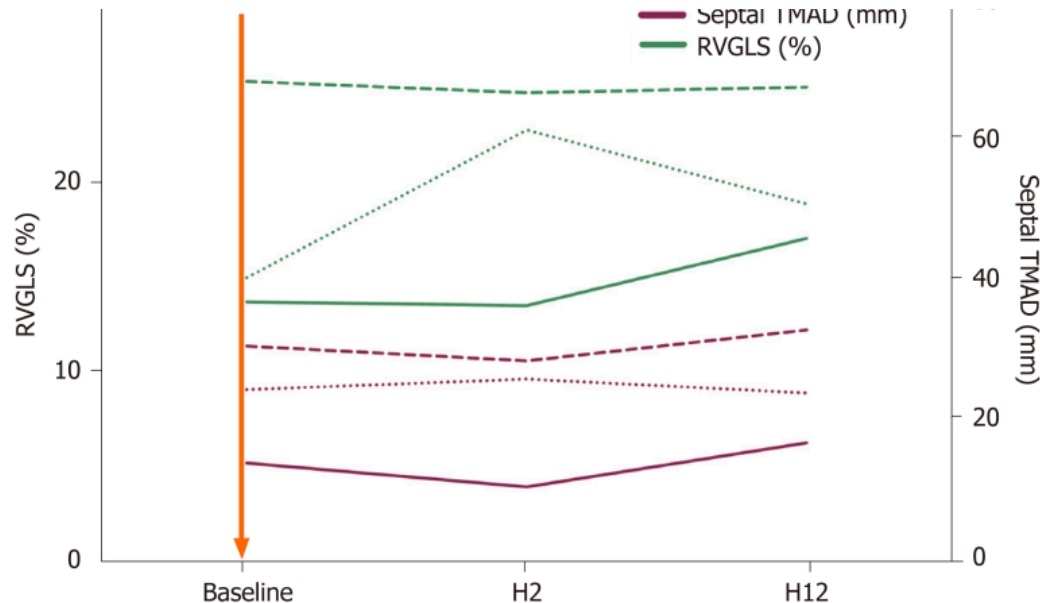
Pierre Huette, Osama Abou Arab, Vincent Jounieaux, Mathieu Guilbart, Mohamed Belhout, Guillaume Haye, Hervé Dupont, Christophe Beyls, Yazine Mahjoub



Almitrine for COVID-19 critically ill patients – a vascular therapy for a pulmonary vascular disease: Three case reports

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Pierre Huette, Osama Abou Arab, Vincent Jounieaux, Mathieu Guilbart, Mohamed Belhout, Guillaume Haye, Hervé Dupont, Christophe Beyls, Yazine Mahjoub



FIRST REPORTED

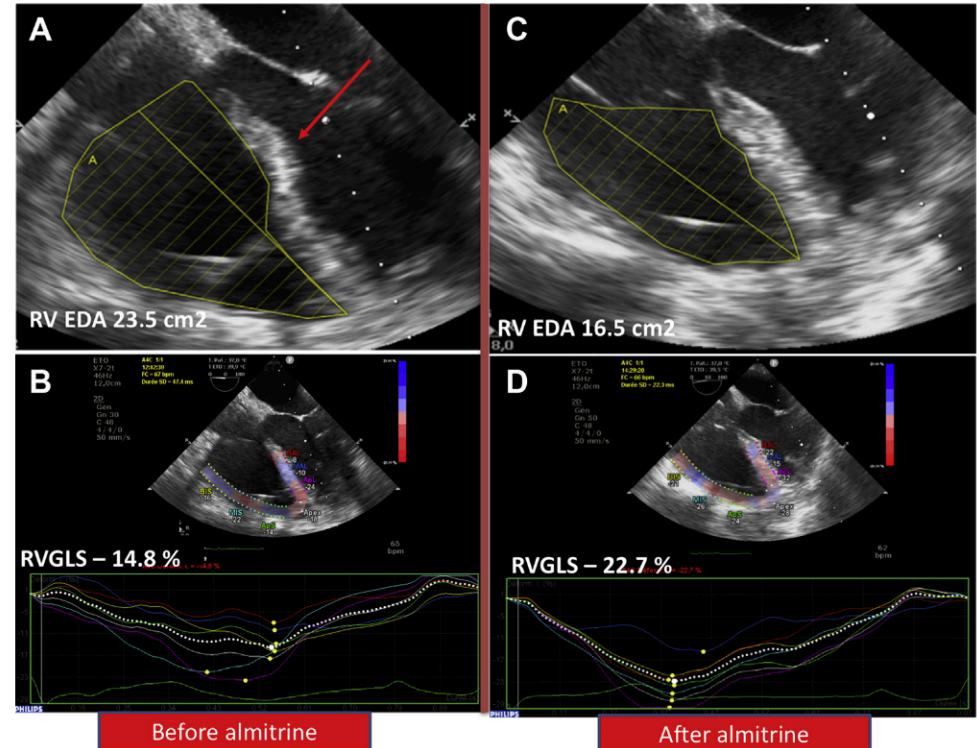
CASE REPORT: CLINICAL CASE

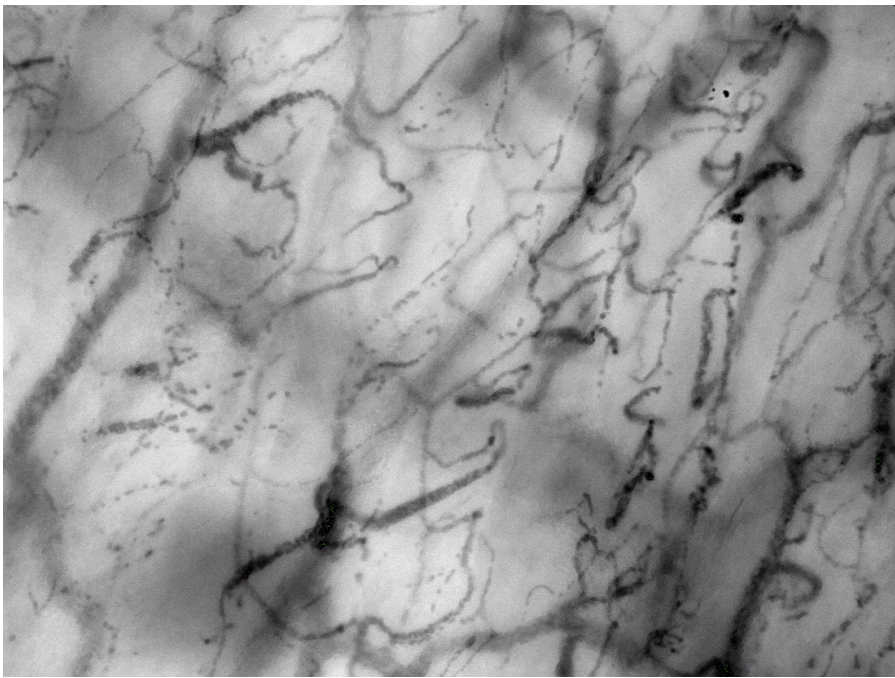
Acute Cor Pulmonale in COVID-19-Related ARDS Improvement With Almitrine Infusion



Pierre Huette, MD, Christophe Beyls, MD, Mathieu Guilbart, MD, Guillaume Haye, MD, Fatim-Zahra Najid, MD, Benjamin Mestan, MD, Pierre-Alexandre Roger, MD, Hervé Dupont, MD, PhD, Osama Abou-Arab, MD, PhD, Yazine Mahjoub, MD, PhD

FIGURE 1 Transesophageal Echocardiography Before and After Almitrine Infusion





RESEARCH ARTICLE

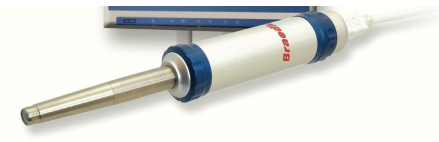
Microvascular flow alterations in critically ill COVID-19 patients: A prospective study

Osama Abou-Arab^{1*}, Christophe Beyls¹, Abdelilah Khalifa¹, Mathieu Guilbart¹, Pierre Huet¹, Stéphanie Malaquin¹, Benoit Lecat¹, Pierre-Yves Macq¹, Pierre Alexandre Roger¹, Guillaume Haye¹, Michaël Bernasinski¹, Patricia Besserve¹, Sandrine Soriot-Thomas², Vincent Jounieaux³, Hervé Dupont¹, Yazine Mahjoub¹

¹ Department of Anesthesiology and Critical Care Medicine, Amiens University Hospital, Amiens, France

PLOS ONE

February 8, 2021

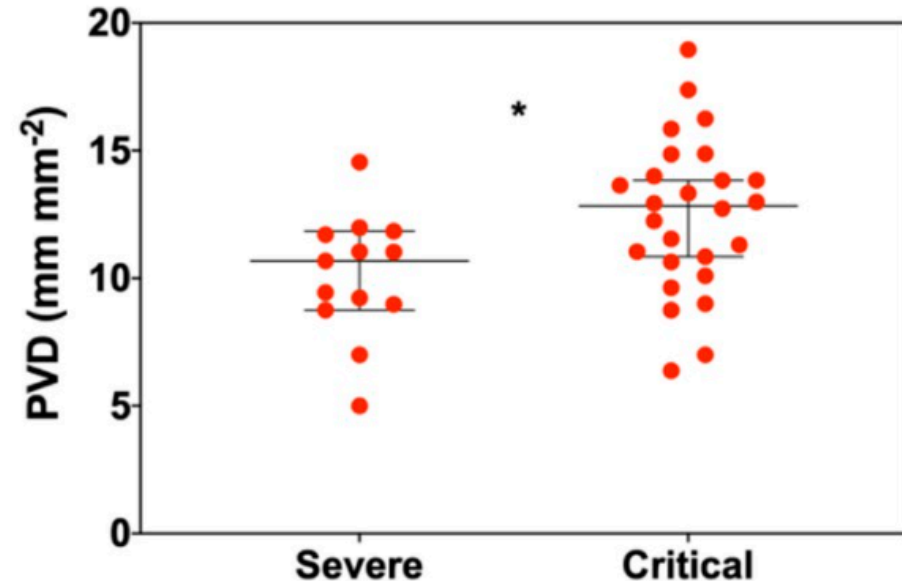
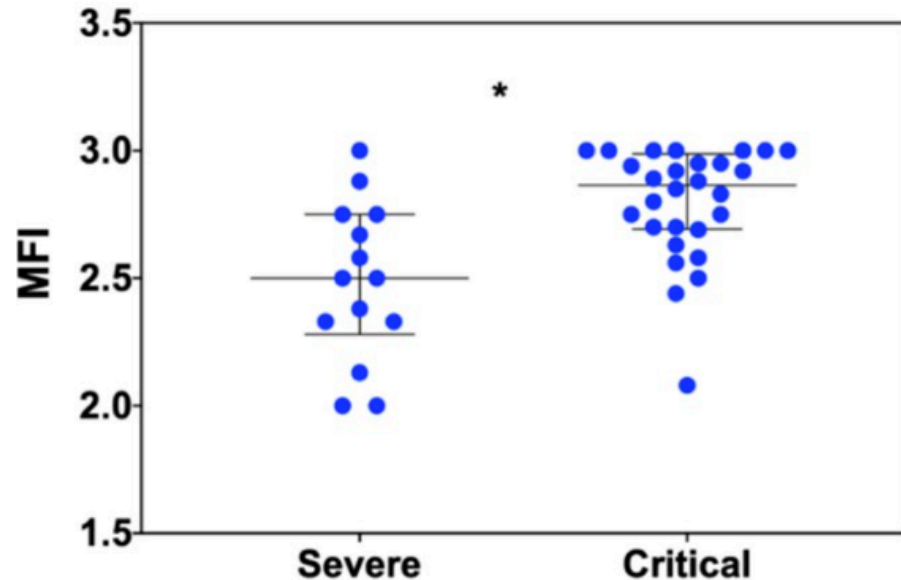


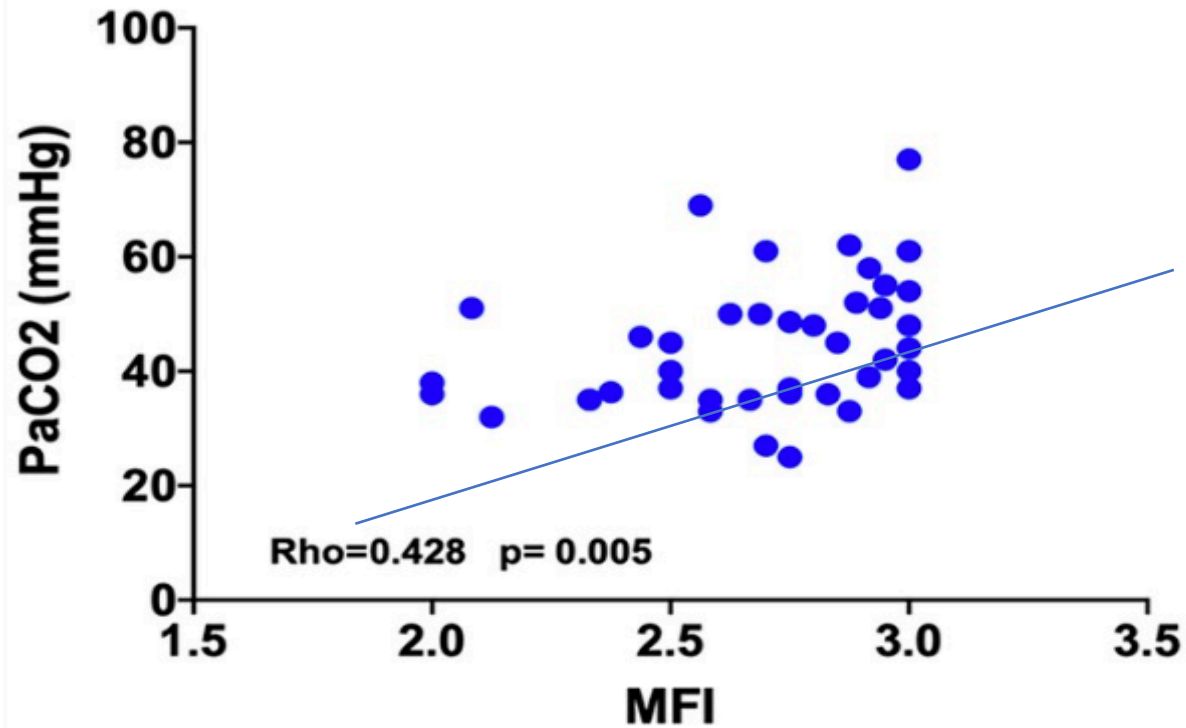
- 43 patients
- Réanimation ou USC
- Comparaison “severe” vs “critical”

Microvascular flow alterations in critically ill COVID-19 patients: A prospective study

Osama Abou-Arab^{1*}, Christophe Beyls¹, Abdelilah Khalipha¹, Mathieu Guilbart¹, Pierre Huet¹, Stéphanie Malaquin¹, Benoit Lecat¹, Pierre-Yves Macq¹, Pierre Alexandre Roger¹, Guillaume Haye¹, Michaël Bernasinski¹, Patricia Besserve¹, Sandrine Soriot-Thomas², Vincent Jounieaux³, Hervé Dupont¹, Yazine Mahjoub¹

¹ Department of Anesthesiology and Critical Care Medicine, Amiens University Hospital, Amiens, France,



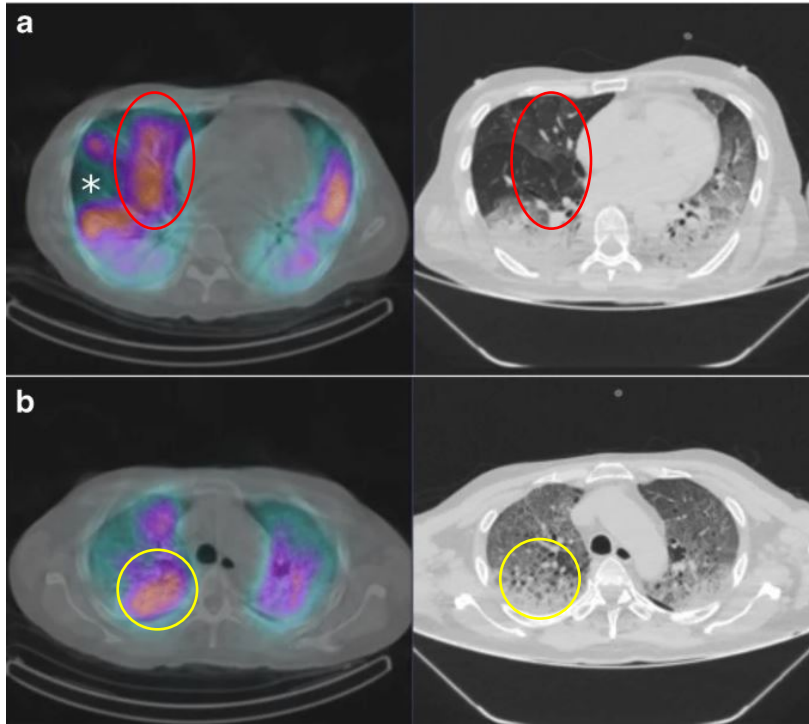


The importance of lung hyperperfusion patterns in COVID-19-related AVDS



European Journal of Nuclear
Medicine and Molecular Imaging

Vincent Journeaux¹ · Yazine Mahjoub² · Isabelle El-Esper³ · Daniel Oscar Rodenstein⁴

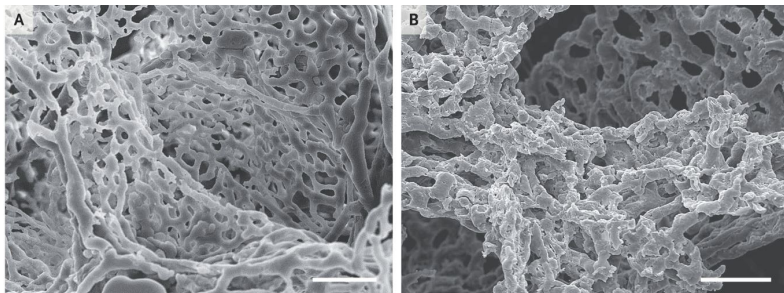


Hypervascularisation dans
des territoires « sains »
(vasodilatation
pulmonaire)

Toutes les anomalies V/Q
observées au cours de
l'infection Covid-19 ne sont
pas attribuables à des EP.

Hypervascularisation dans
des territoires condensés
(par perte de la
vasoconstriction
pulmonaire hypoxique)

Autopsies



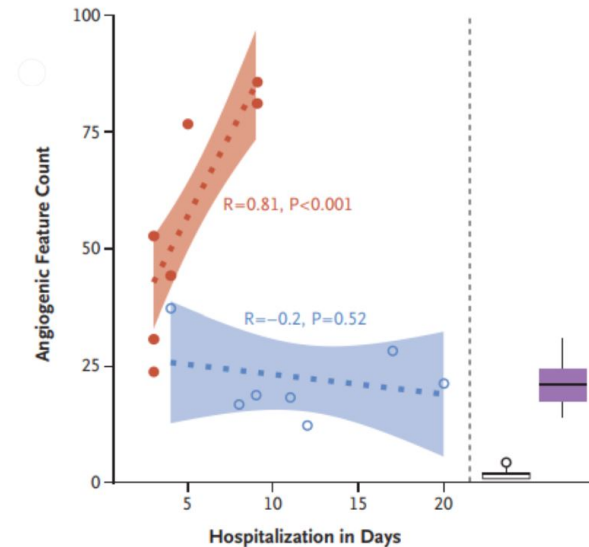
Ackermann M et al.

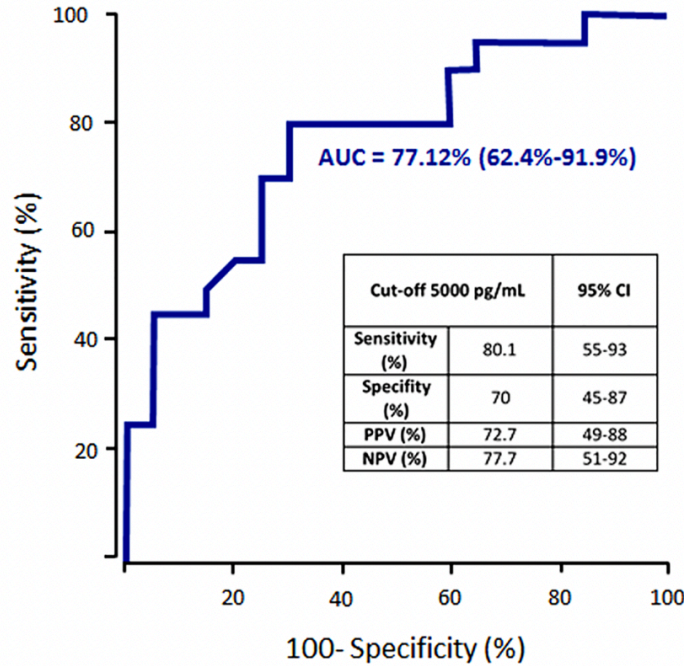
Pulmonary Vascular Endothelialitis, Thrombosis, and Angiogenesis in Covid-19.

N Engl J Med. 2020. DOI: 10.1056/NEJMoa2015432.

- Ultrastructural damage to the endothelium,
- Presence of Sars-Cov-2 in endothelial cells of
- Pulmonary angiogenesis observed at day 4 hospital admission and significantly increases with time

C Intussusceptive Angiogenic Features over Time





Angiopietin-2 as a marker of endothelial activation is a good predictor factor for intensive care unit admission of COVID-19 patients

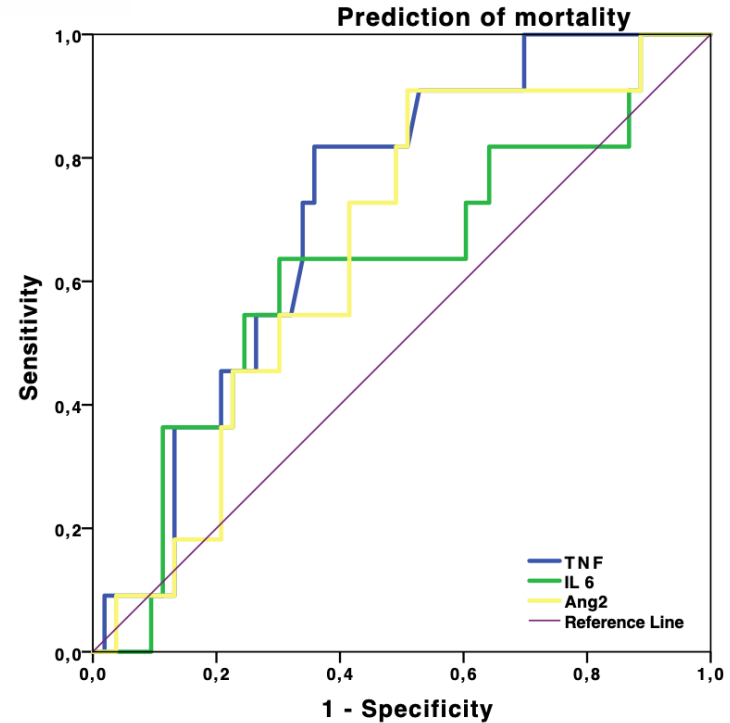
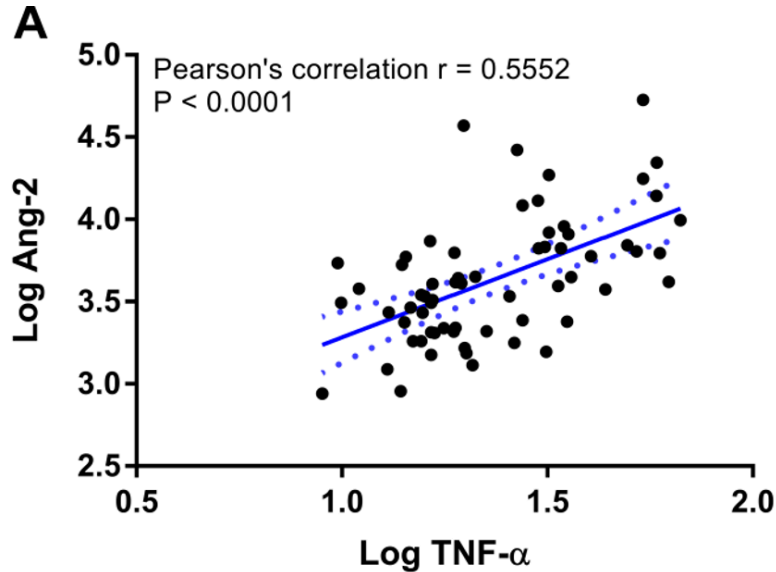
David M. Smadja^{1,2} · Coralie L. Guerin^{1,3} · Richard Chocron^{4,5} · Nader Yatim^{6,7} · Jeremy Boussier^{6,7} · Nicolas Gendron^{1,2} · Lina Khider⁸ · Jérôme Hadjadj^{7,9} · Guillaume Goudot⁸ · Benjamin Debuc¹⁰ · Philippe Juvin¹¹ · Caroline Hauw-Berlemont¹² · Jean-Loup Augy¹² · Nicolas Peron¹² · Emmanuel Messas^{4,13} · Benjamin Planquette^{1,14} · Olivier Sanchez^{1,14} · Bruno Charbit¹⁵ · Pascale Gaussem^{1,16} · Darragh Duffy^{6,7} · Benjamin Terrier^{17,18} · Tristan Mirault^{4,13} · Jean-Luc Diehl^{1,19}

Biological characteristics of COVID-19 patients at admission according to level of care (medical ward or ICU direct admission)

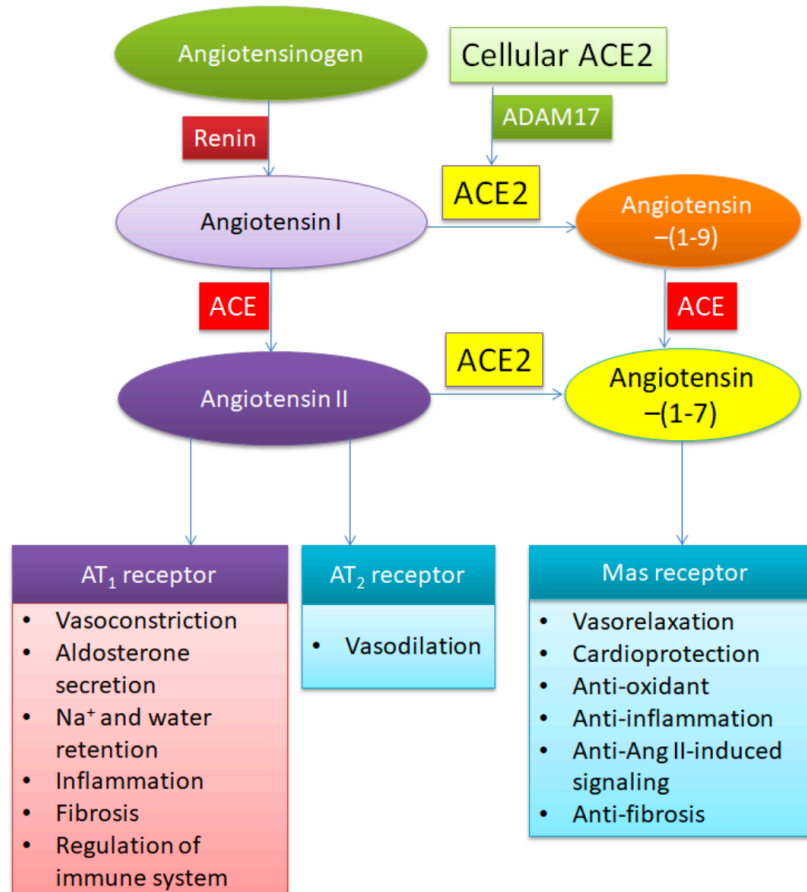
	Medicine patients (<i>n</i> = 20)	ICU patients (<i>n</i> = 20)	<i>p</i> value
Angiopietin-2	4385.7 [3185.9, 5667.1]	6574.1 [5354.4, 11,375.9]	0.003

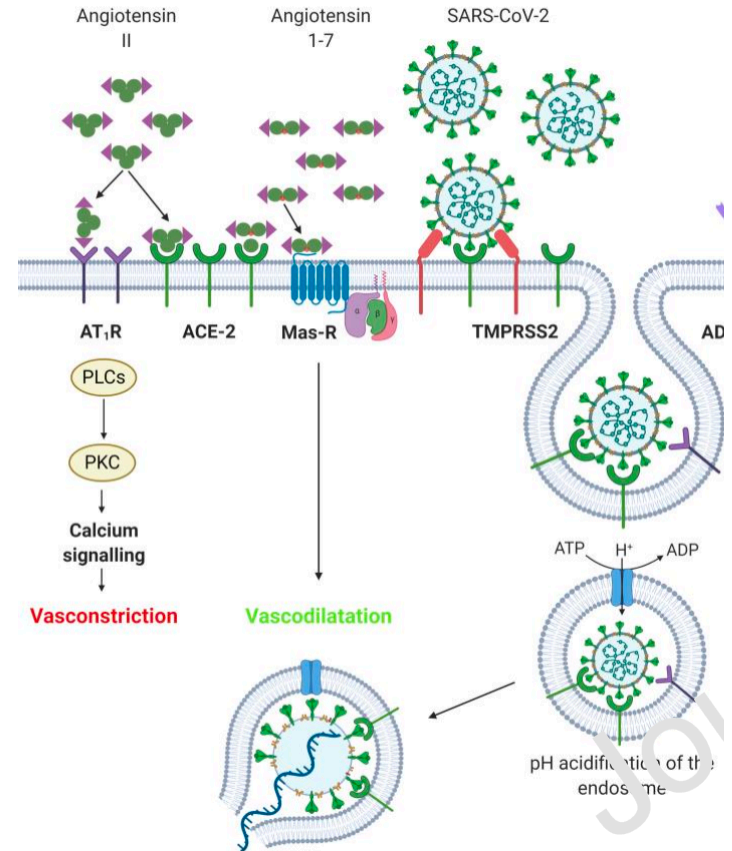
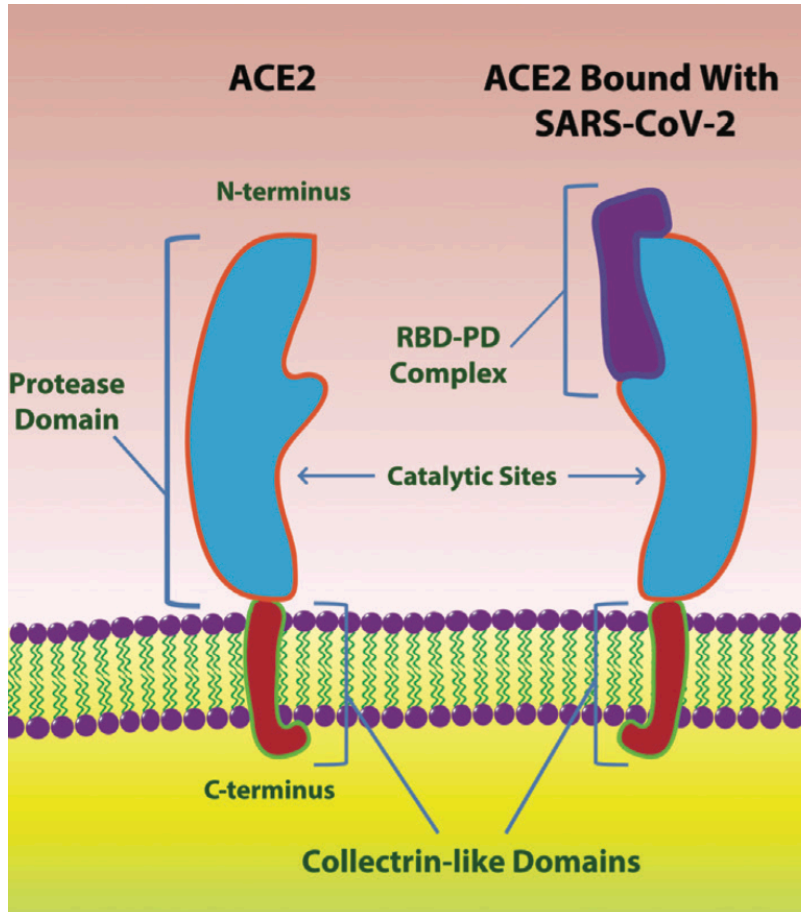
Association between inflammation, angiopoietins, and disease severity in critically ill COVID-19 patients: a prospective study

Osama Abou-Arab^{1,2,*}, Youssef Bennis², Pierre Gauthier¹, Cedric Boudot², Gwladys Bourdenet³, Brigitte Gubler⁴, Christophe Beyls¹, Hervé Dupont³, Said Kamel² and Yazine Mahjoub¹

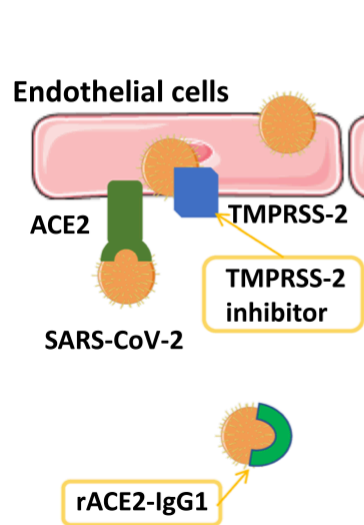


ACE and ACE2 in RAAS

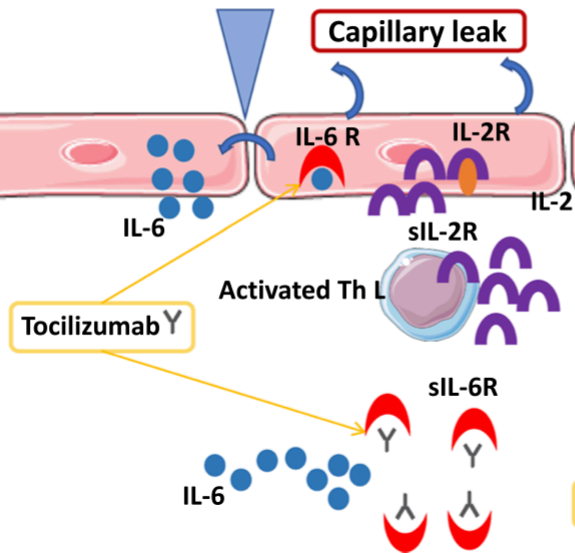




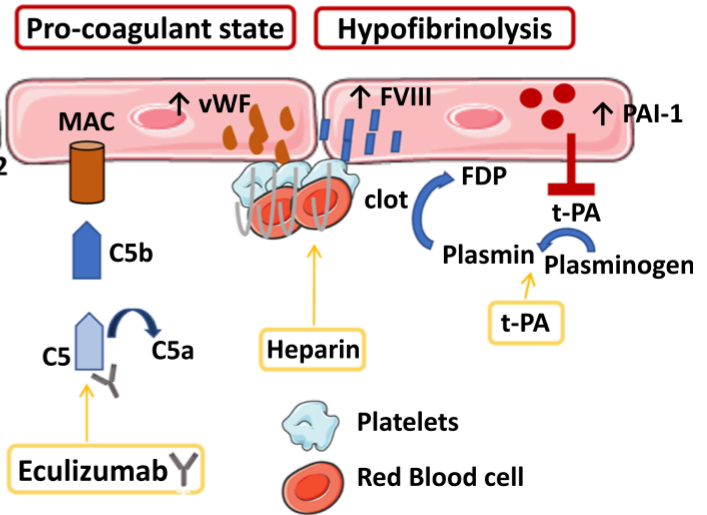
A) SARS-CoV-2 ENTRY



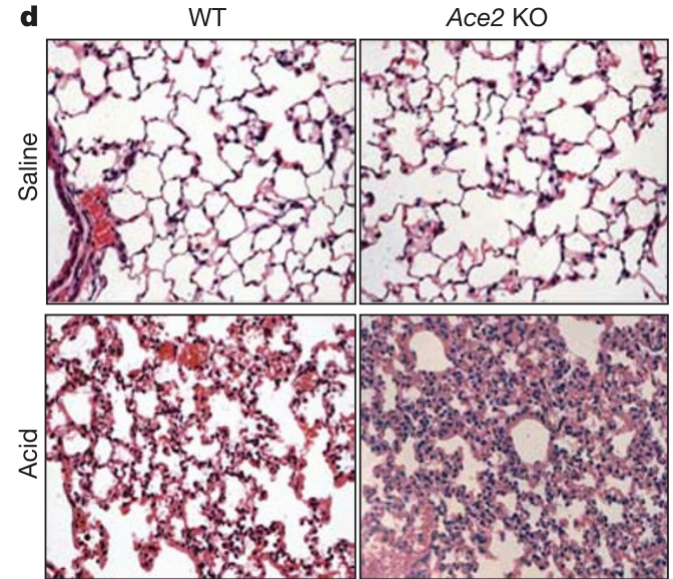
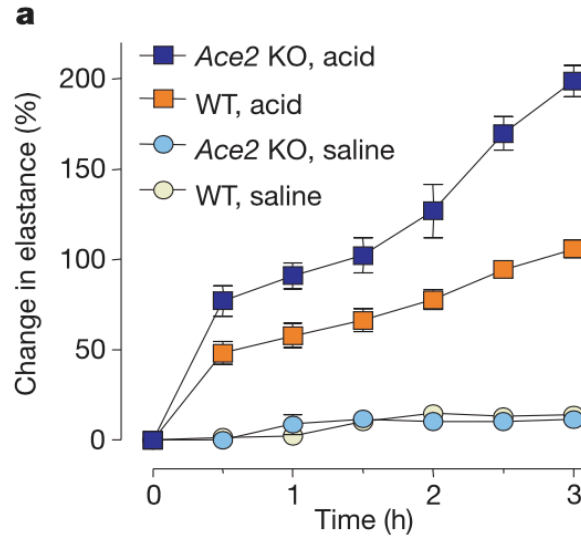
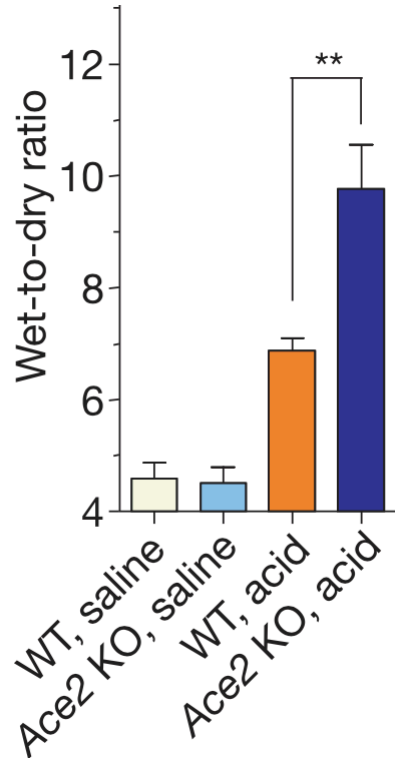
B) CYTOKINES



C) THROMBOSIS



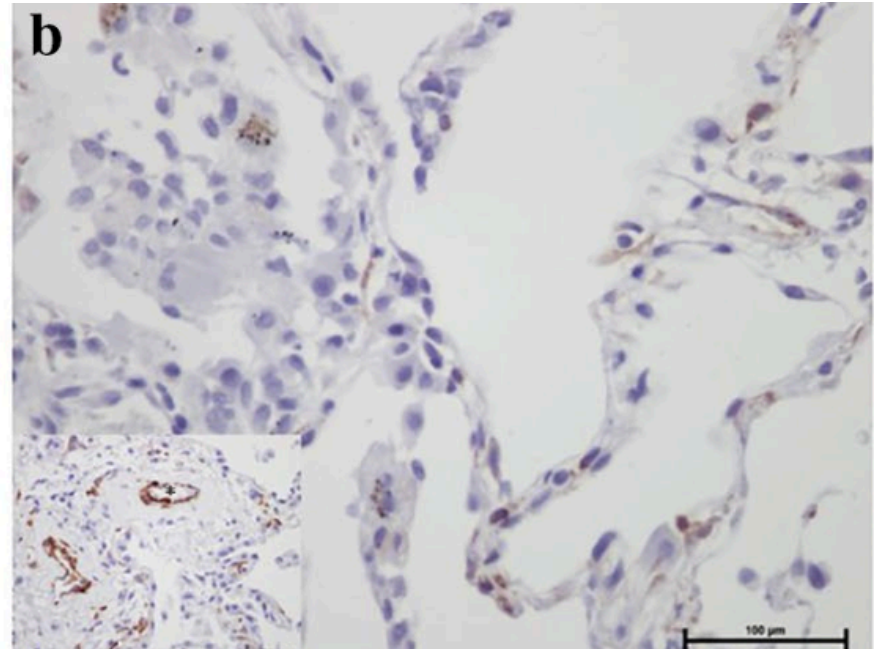
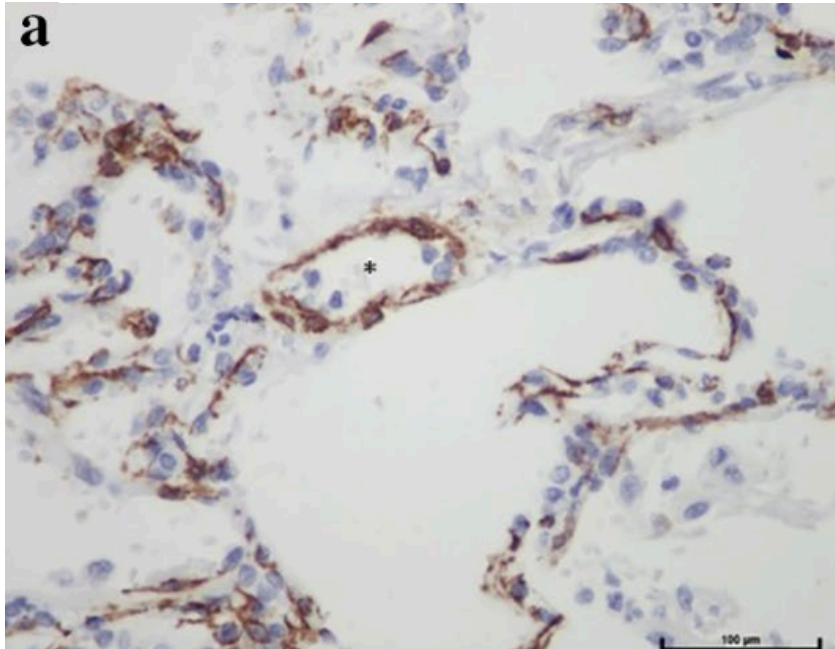
Effet protecteur des Récepteurs ACE-2



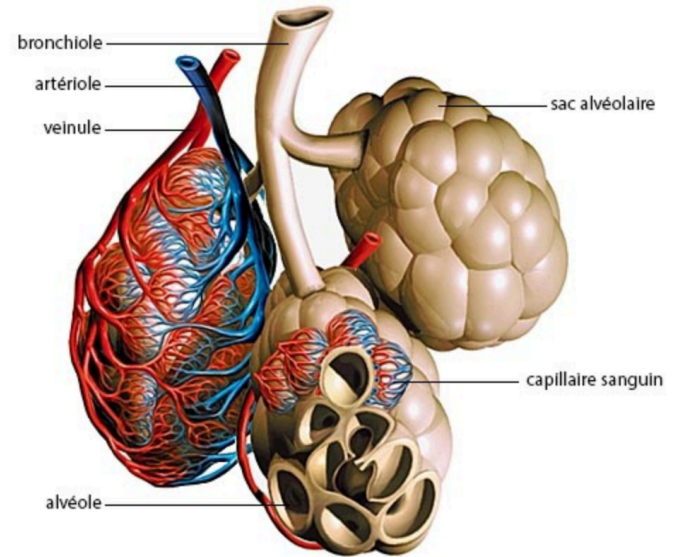
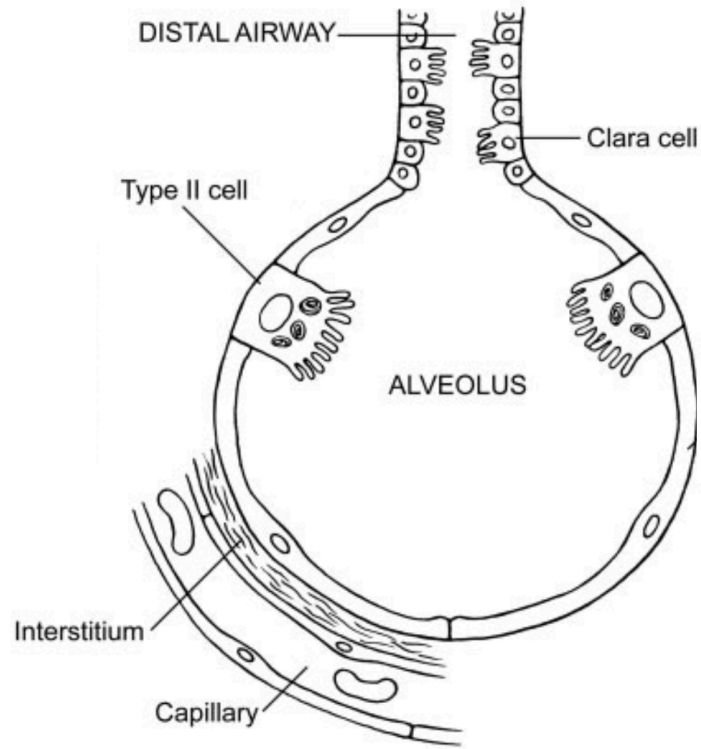
Imai Y, Nature 2005

Pericyte alteration sheds light on micro-vasculopathy in COVID-19 infection

Nathalie Cardot-Leccia¹, Thomas Hubiche², Jean Dellamonica³, Fanny Burel-Vandenbos¹
and Thierry Passeron^{2,4*} 

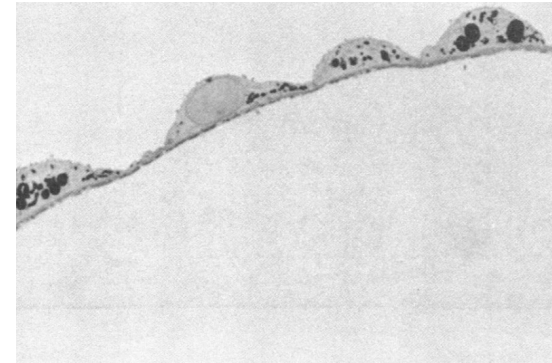
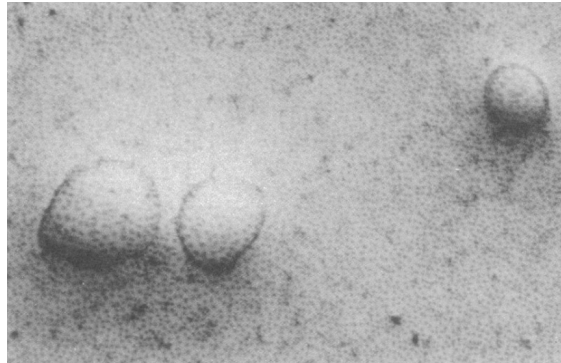
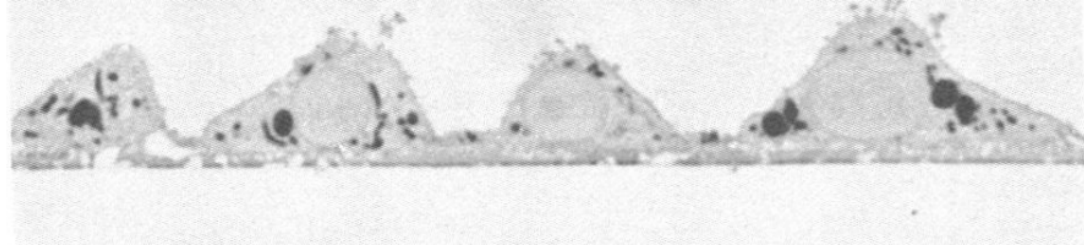


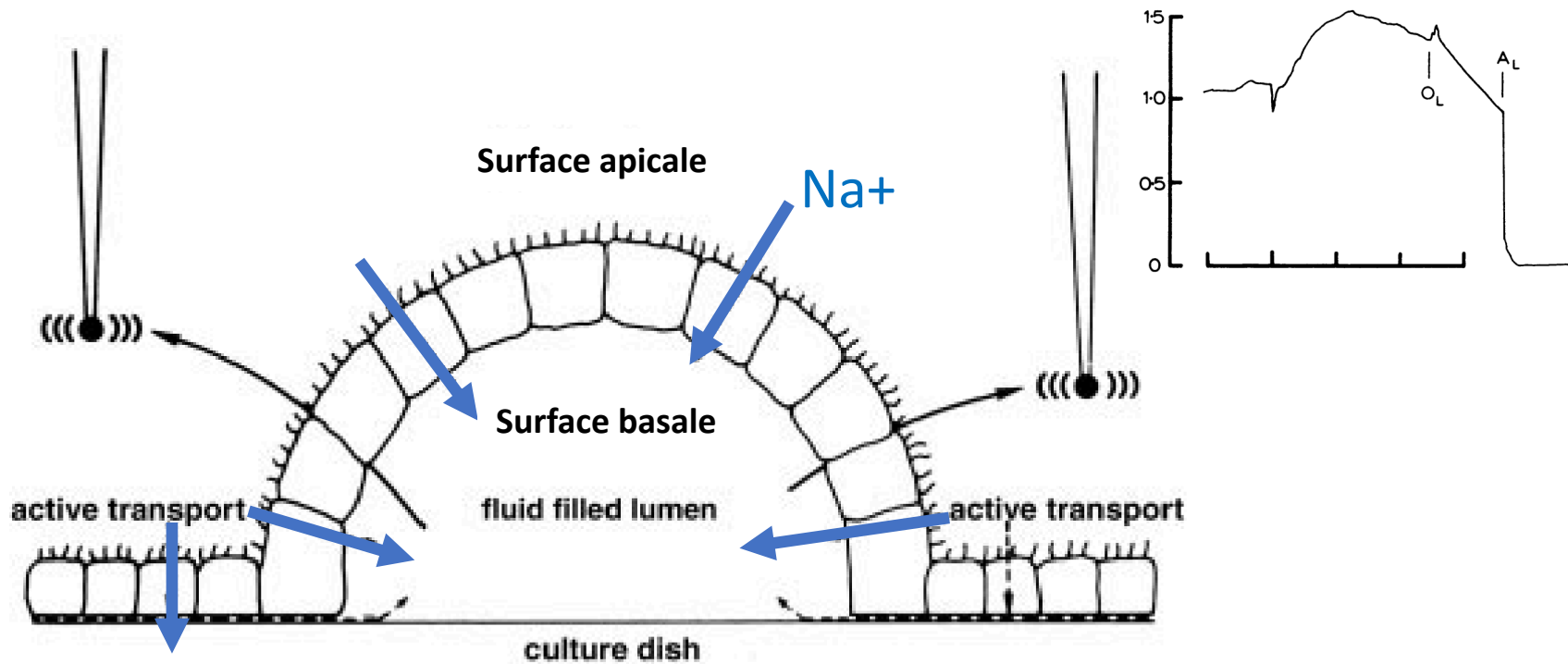
Alvéole



Clairance liquidienne du poumon sain

- Culture de pneumocyte de type II
- Formation de dôme
- Inhibition par l'amiloride





ACE

Lésion endothéliales

Lésion pérycites

Œdème interstitiel + Néoangiogenèse

Shunt (hypoxie)

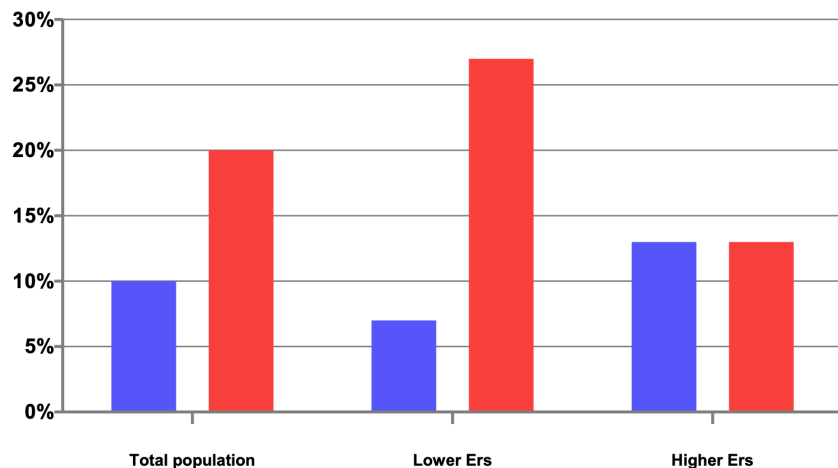
Œdème alvéolaire (hypoxie)

Atteinte de la compliance



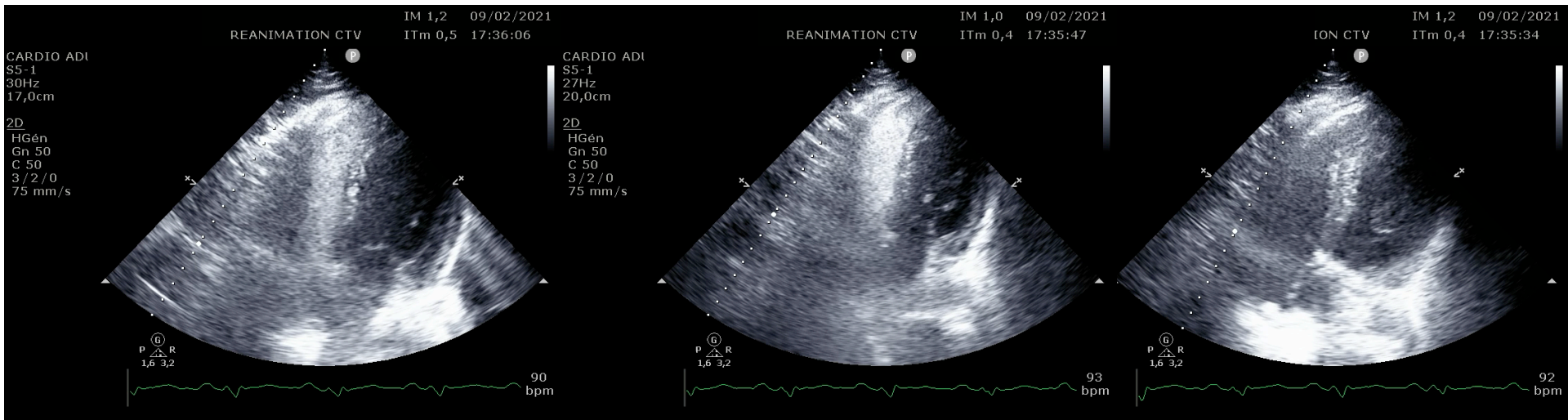
Is hypoxemia explained by intracardiac or intrapulmonary shunt in COVID-19-related acute respiratory distress syndrome?

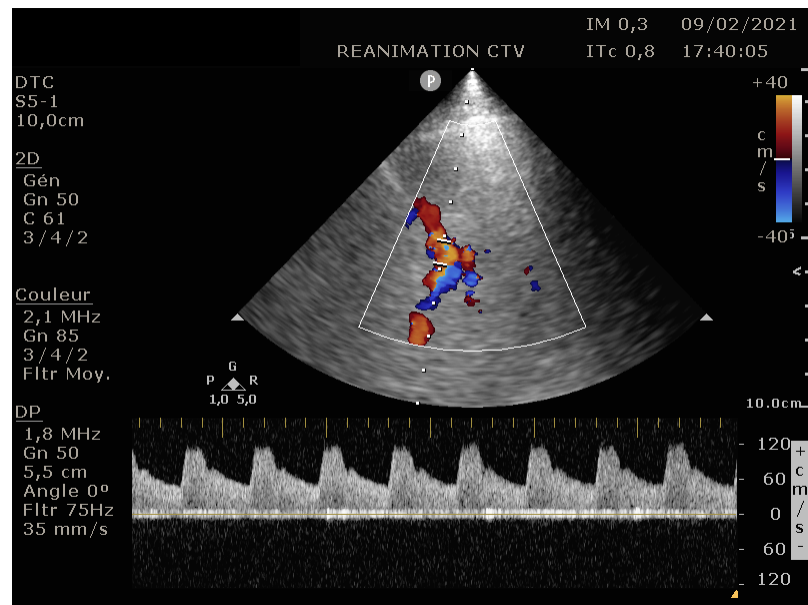
Paul Masi^{1,2*} , François Bagate^{1,2}, Thomas d'Humières^{3,4}, Lara Al-Assaad³, Laure Abou Chakra³, Genevieve Derumeaux^{3,4} and Armand Mekontso Dessap^{1,2}



ETT: 7%

ETO: 16-19%





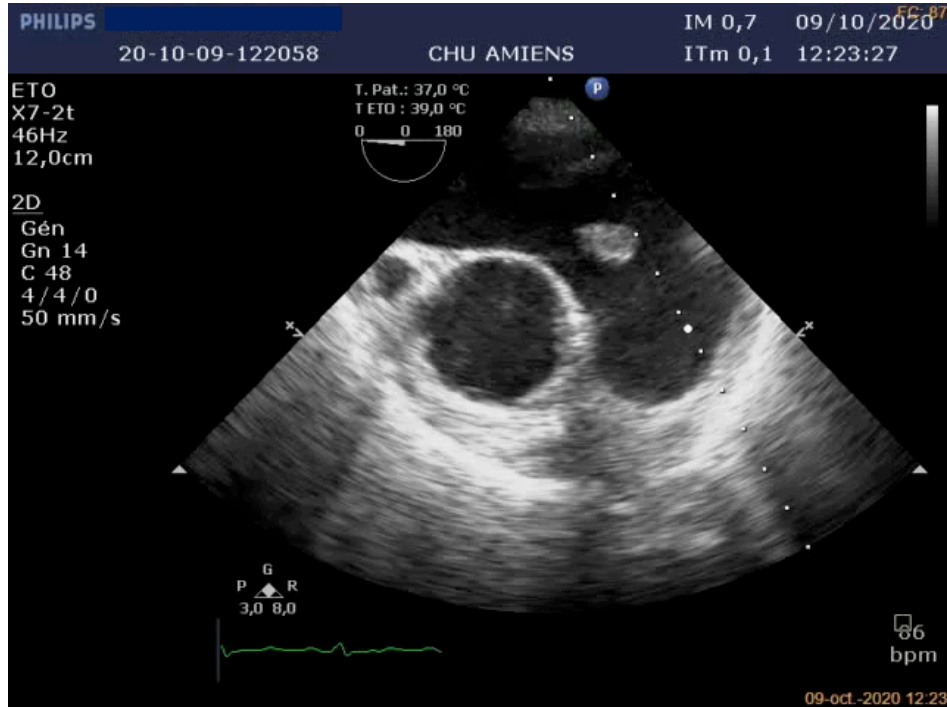
Pulmonary Embolism and Deep Vein Thrombosis in COVID-19: A Systematic Review and Meta-Analysis

Young Joo Suh, MD, PhD • Hyunsook Hong, PhD • Mickaël Ohana, MD, PhD • Florian Bompard, MD • Marie-Pierre Revel, MD, PhD • Clarissa Valle, MD • Alban Gervaise, MD, PhD • Julien Poissy, MD • Sophie Susen, MD, PhD • Guillaume Hékimian, MD • Mathieu Artifoni, MD • Daniel Periard, MD • Damien Contou, MD • Julie Delaloye, MD • Bienvenido Sanchez, MD • Cheng Fang, FRCR • Giorgio Garzillo, MD • Hasti Robbie, MD • Soon Ho Yoon, MD, PhD

Parameter	Incidence (%)	Heterogeneity*
PE		
Overall	16,5%	NA
Study design		
Retrospective (<i>n</i> = 20)	15.5 (10.4, 22.6)	<.001, 0.93
Prospective (<i>n</i> = 2)	25.1 (11.1, 47.2)	<.001, 0.93
Study population†		
Non-ICU or various (<i>n</i> = 12)	24,7%	<.001, 0.95
ICU or critically ill patients (<i>n</i> = 18)		<.001, 0.82
Anticoagulation		
None or unknown (<i>n</i> = 2)	21.0 (15.6, 27.8)	0.43, <0.001
Yes (<i>n</i> = 20)	16.0 (10.8, 23.0)	<.001, 0.93
Proportion of patients undergoing CT pulmonary angiography		
Part of patients or unknown (<i>n</i> = 15)	11.3 (6.7, 18.4)	<.001, 0.92
All patients (<i>n</i> = 7)	30.2 (21.0, 41.3)	<.001, 0.91
DVT		
Overall	14,8%	NA
Study design		
Retrospective (<i>n</i> = 15)	18.8 (10.5, 31.5)	<.001, 0.95
Prospective (<i>n</i> = 3)	3.0 (0.4, 19.5)	<.001, 0.91
Study population†		
Non-ICU or various (<i>n</i> = 8)	21,2%	<.001, 0.93
ICU or critically ill patients (<i>n</i> = 13)		<.001, 0.93

- 27 études
- 3342 patients

Embolie pulmonaire



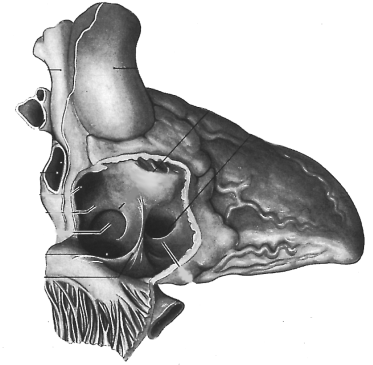
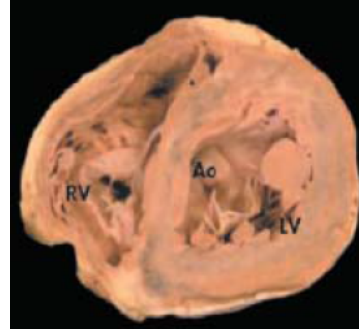
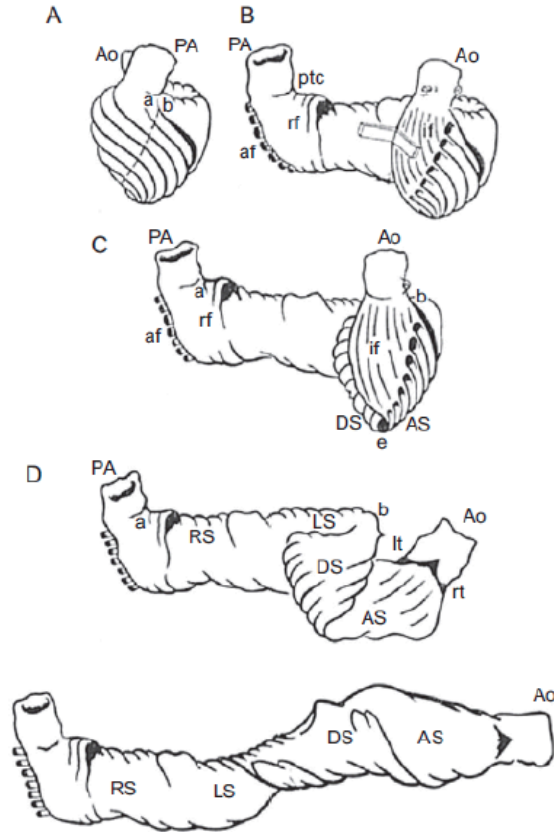
- Malgré anticoagulation efficace
- Thrombus tronc de l'AP

Atteinte Cardiaque

Cœur Droit

- Atteinte virale intrinsèque
- CPA / embolie pulmonaire, SDRA
- dysfonction systolique?

Right Ventricle Anatomy



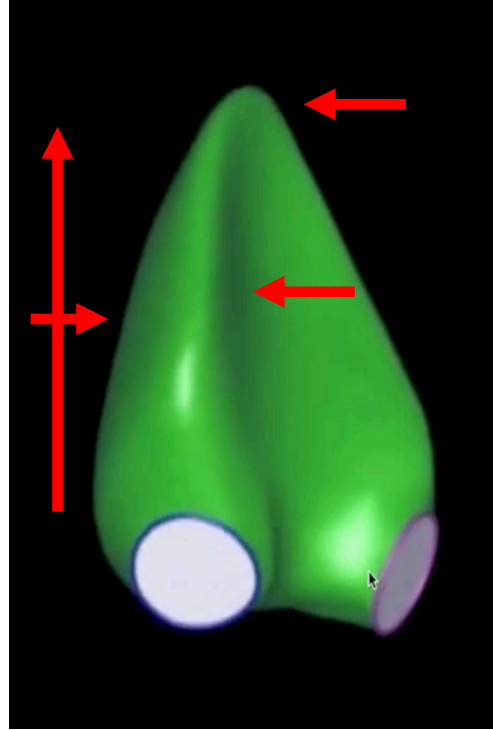
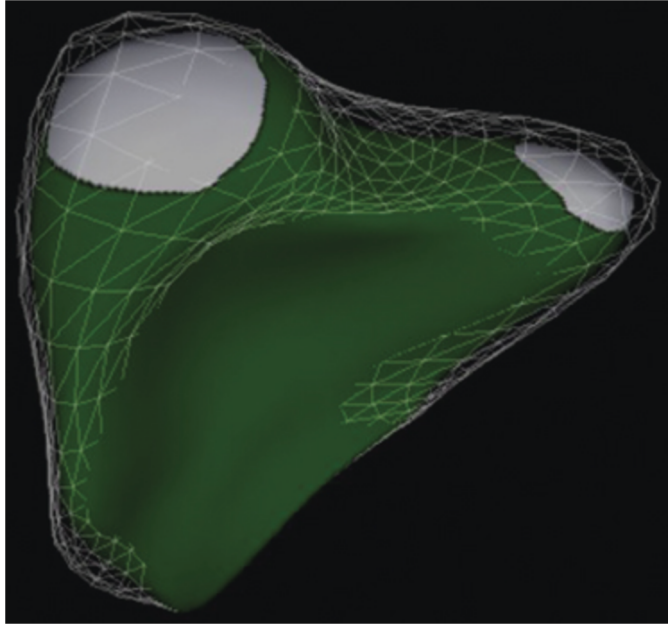
RV lacks transverse fibers

Predominantly longitudinal fibers

Area RV > LV

Weight RV = 1/6 LV

La contraction du VD

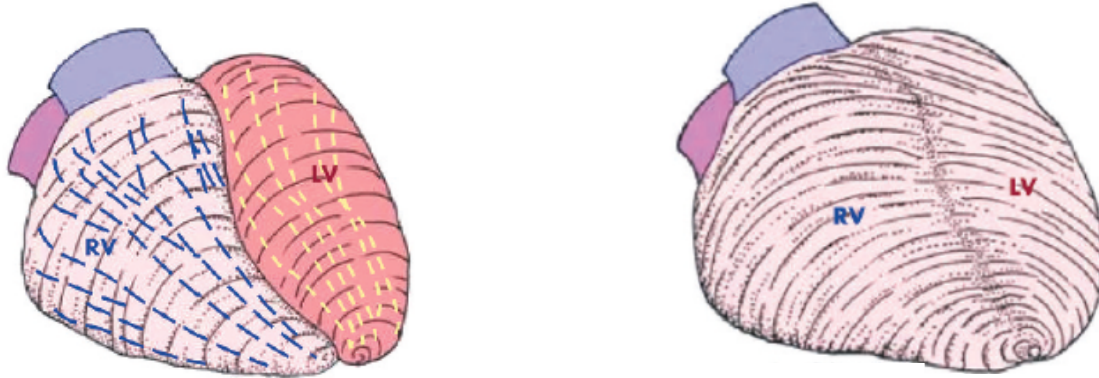


RV-LV interaction

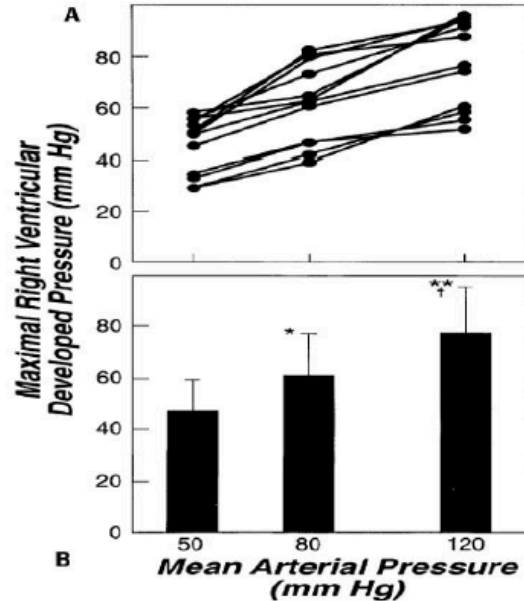
- Superficial fibers
- Septum
- pericardium

Interaction RV-LV

Separated Deep layers of Fibres but shared superficial fibres encircling RV and LV



Role of interventricular septum



Maximal RVDP (mm Hg)

Mean arterial pressure

50 mm Hg

$47.5 \pm 12.1^*$

80 mm Hg

$60.7 \pm 15.2^*$

120 mm Hg

$77.7 \pm 17.9^*$

Working septum (at 80 mm Hg)

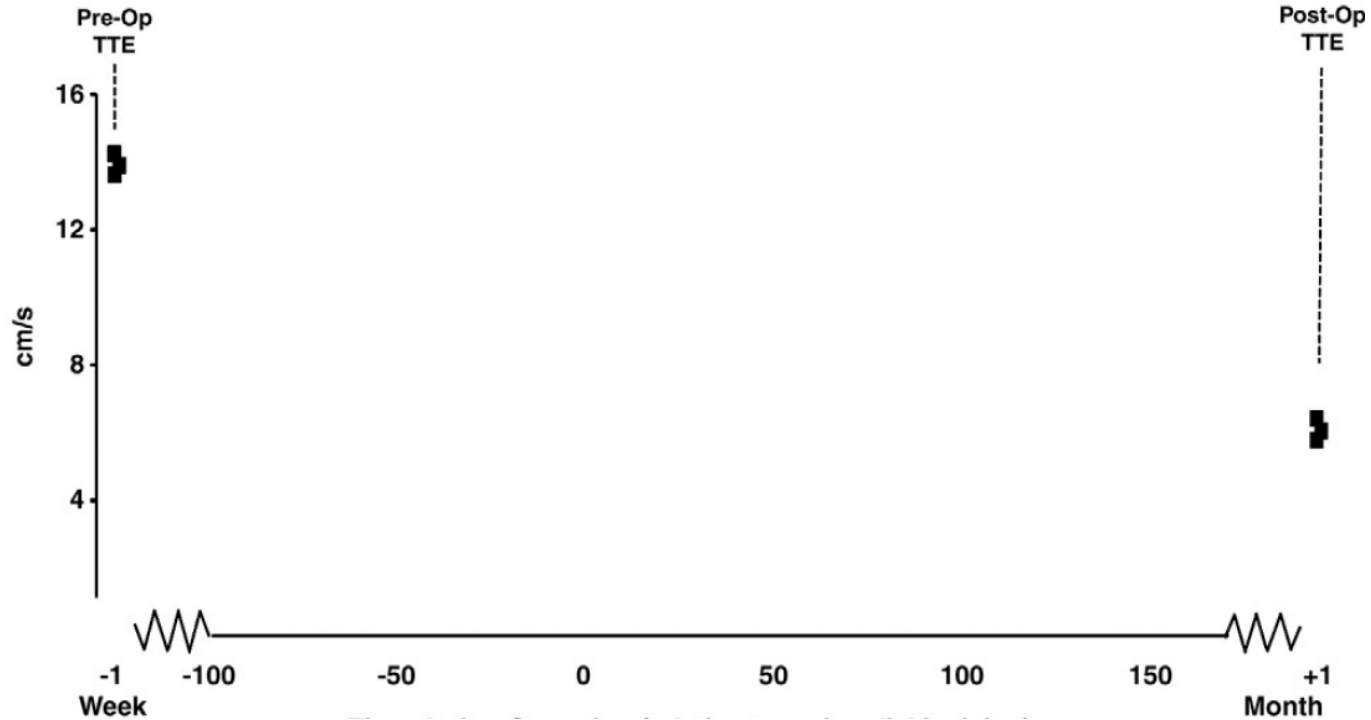
60.7 ± 15.4

Inactivated septum (at 80 mm Hg)

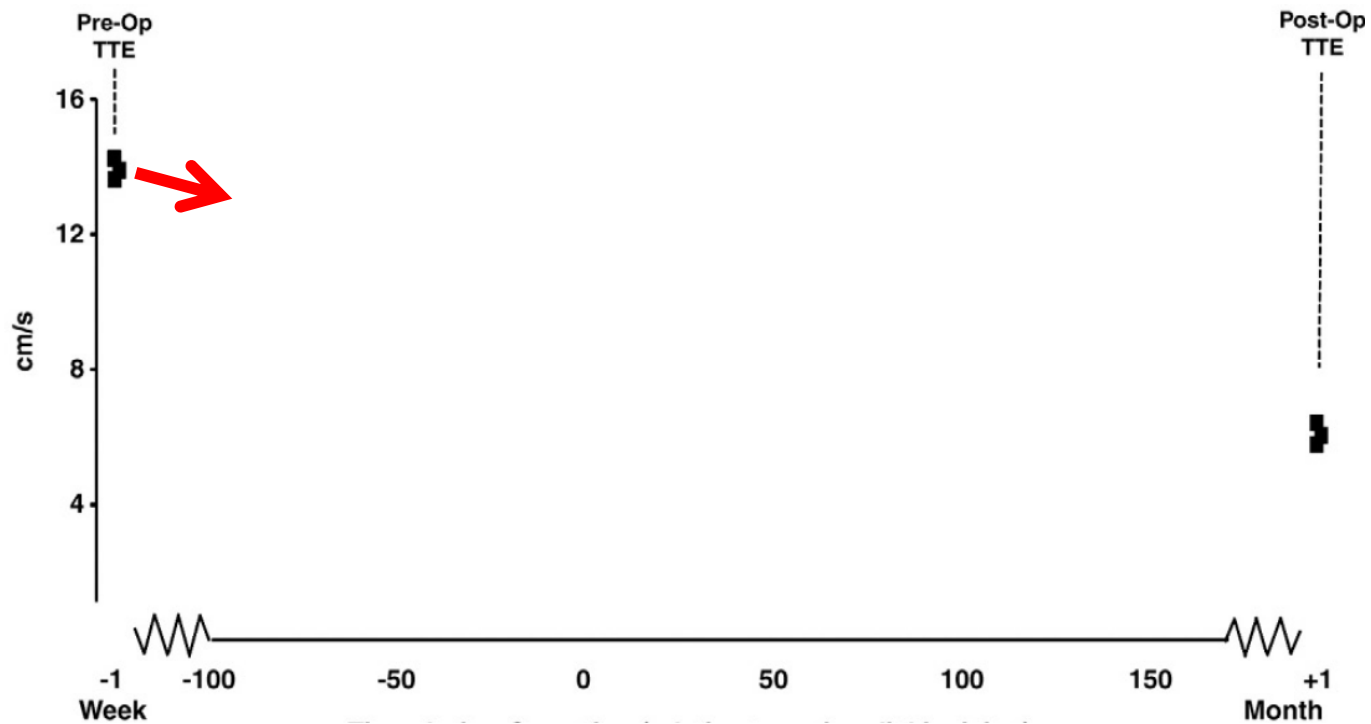
$39.8 \pm 11.2^\dagger$

Septum contribution about 65 %

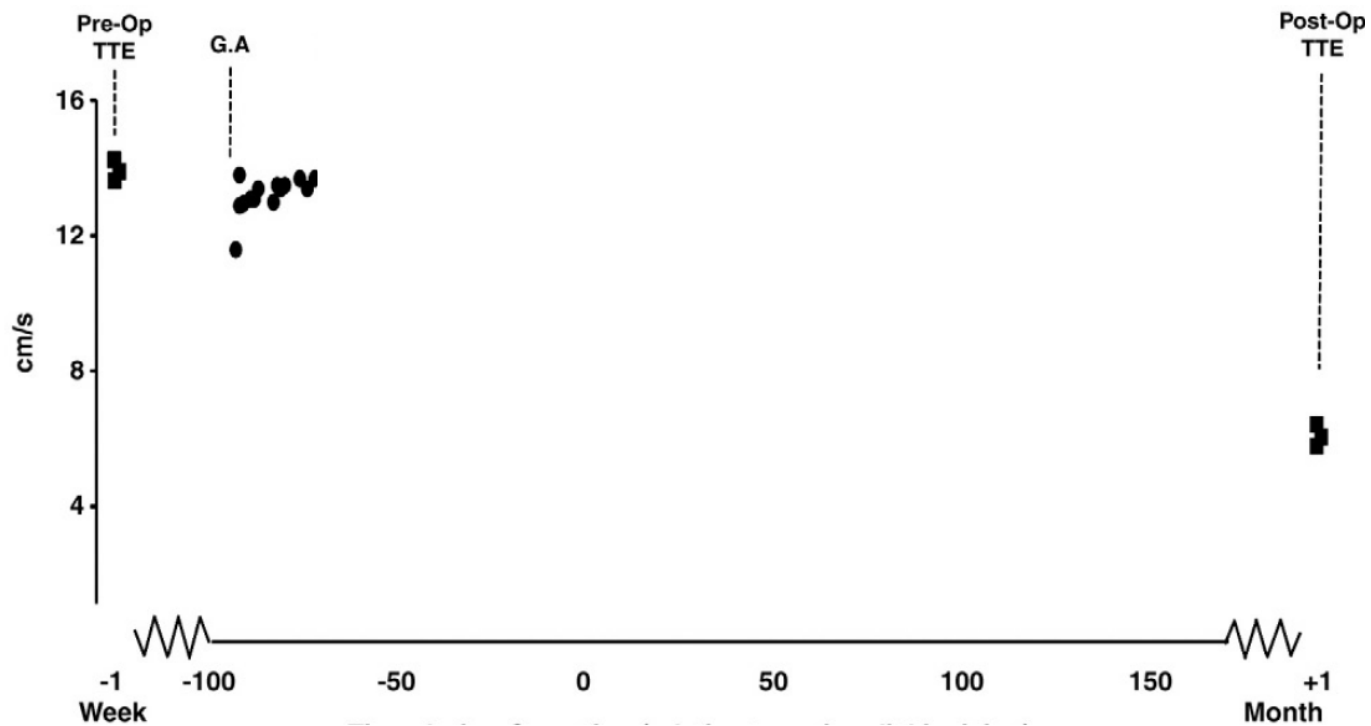
Role of the pericardium



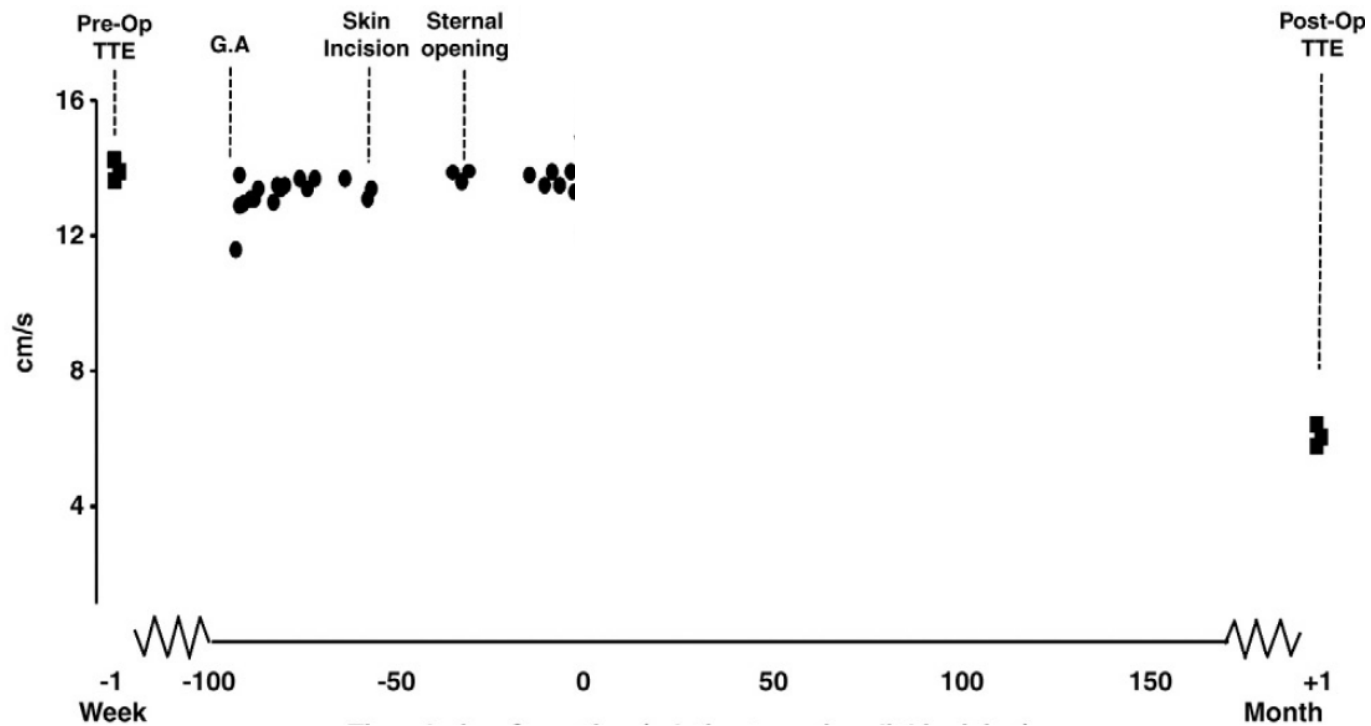
Unsworth B, Am Heart J 2012



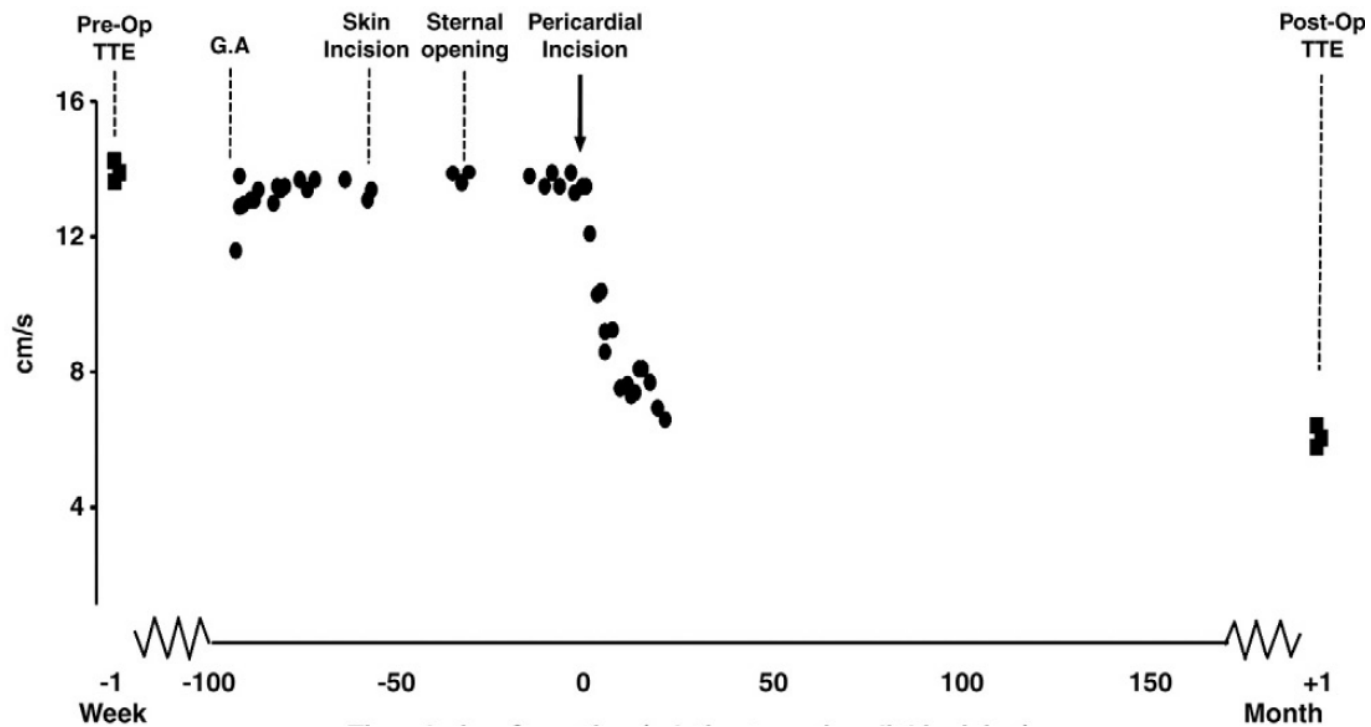
Unsworth B, Am Heart J 2012



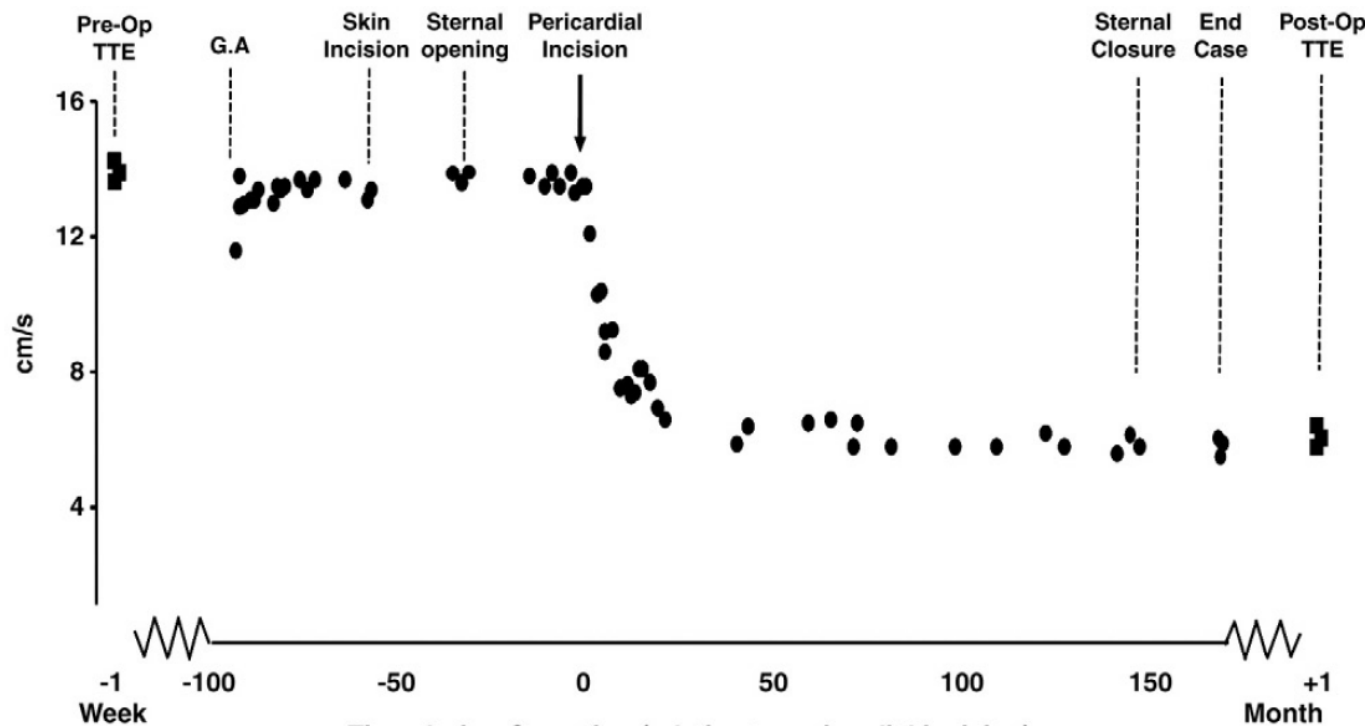
Unsworth B, Am Heart J 2012



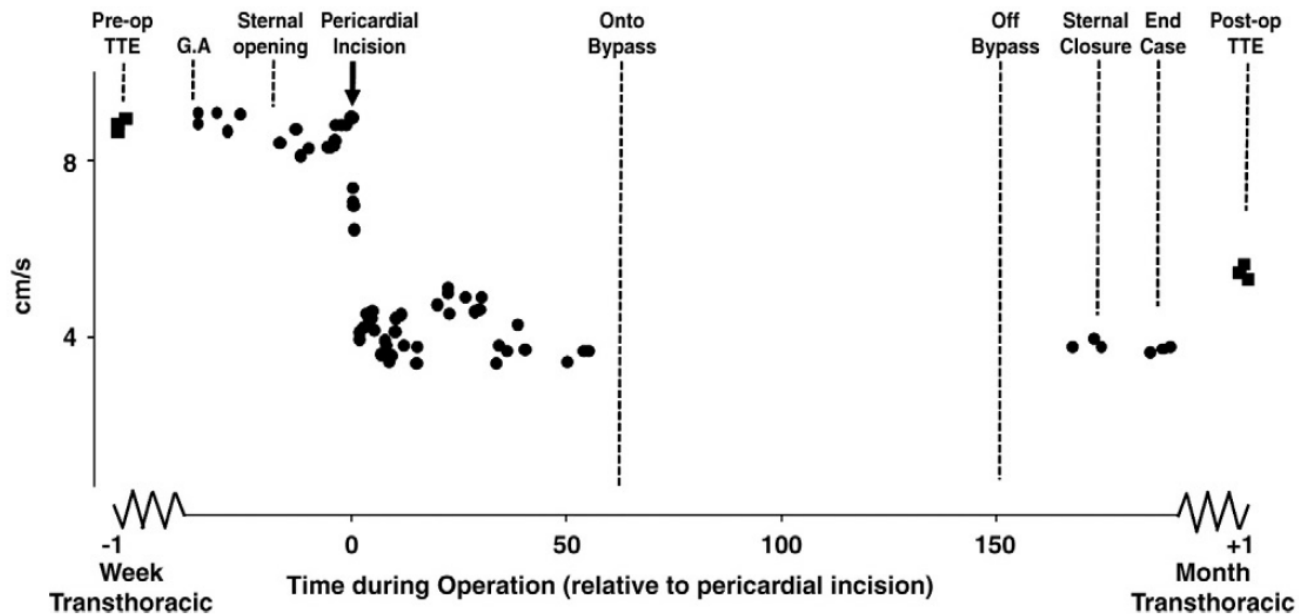
Unsworth B, Am Heart J 2012



Unsworth B, Am Heart J 2012



Unsworth B, Am Heart J 2012



RV-LV interdependance

$$\Delta V_1 = \frac{C_s}{C_s + C_r} \Delta V_r + C_1 \Delta P_p$$

V1= LV volume

Vr= RV Volume

Cs= Septum compliance

Cr= RV free wall compliance

C₁= LV free wall compliance

Pp=Pericardial pressure

Little WC, *Circ Res* 1984

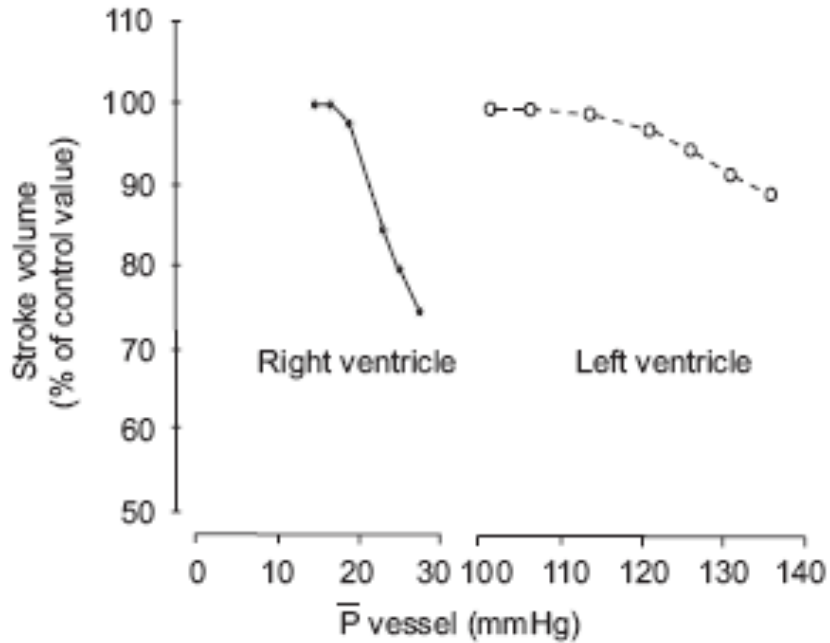
Santamore WP, *Am Rev Resp* 1986

Much of the RV work is done by LV contraction

Right heart response to acute stress

- RV is able to dilate quickly to acute stress (adapting diastolic fn)
 - Volume overload
 - Pressure overload
- RV is very sensitive to acute increase in afterload

Acute afterload increase



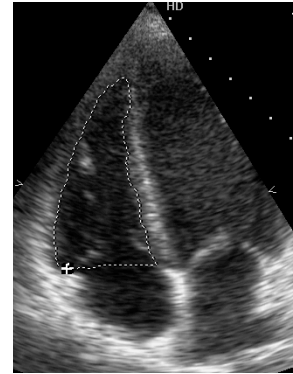
$$\text{FAC} = \text{RVEDA} - \text{RVESA} / \text{RVEDA} \times 100\%$$

Normal values

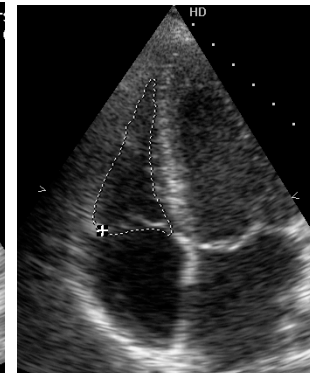
EDA: $20 \pm 4 \text{ cm}^2$

ESA: $11 \pm 3 \text{ cm}^2$

FAC = $46 \pm 7 \%$



S télédiast



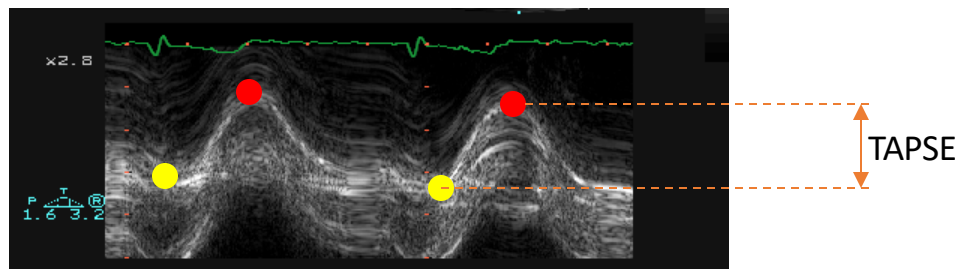
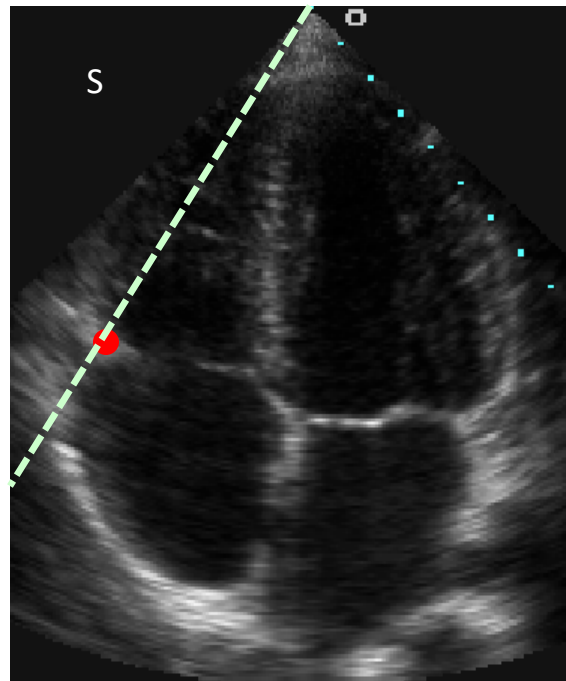
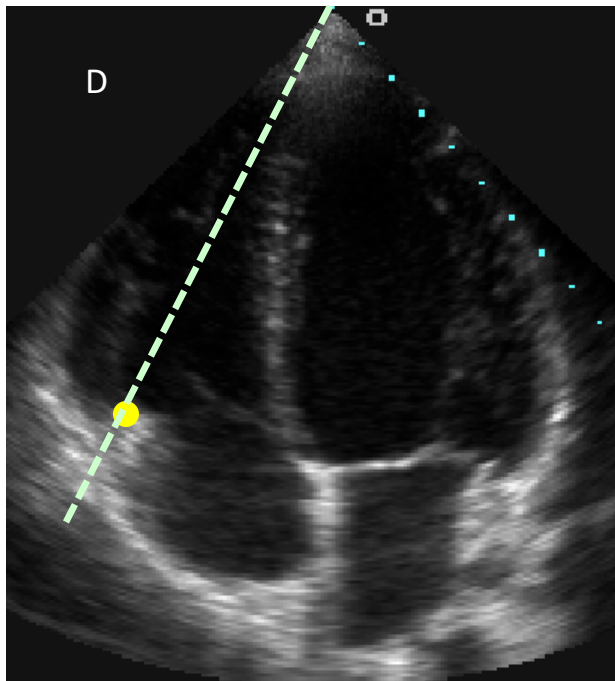
S télésyst

Triulzi. *Echocardiography* 1984

Correlation with radionuclid RV EF VD ($r = 0,8$)

Reproducibility 14%

Kaul. *Am H J* 1984

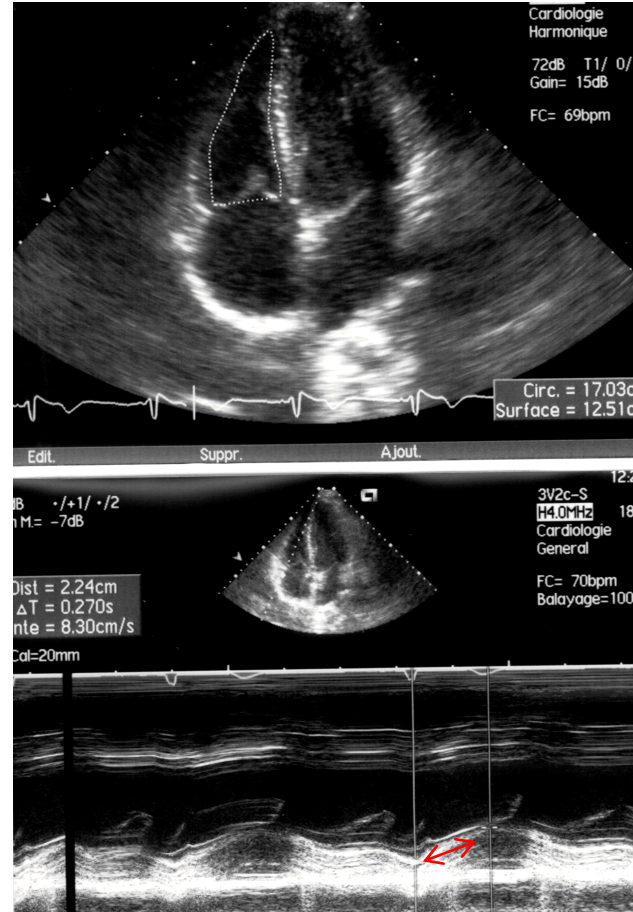


TAPSE

N > 17 mm.

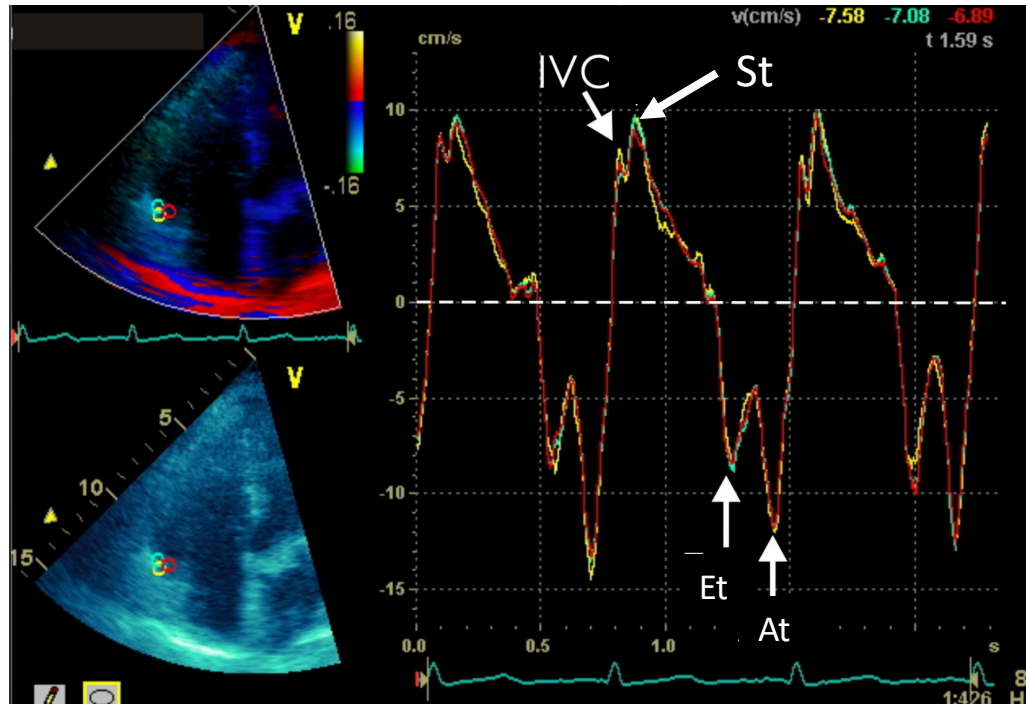
RV Dysfonction < 12mm

Severe RV dysfonction < 8.5mm



Tissue Doppler Imaging: Maximal velocity of tricuspid annulus systolic wave: St

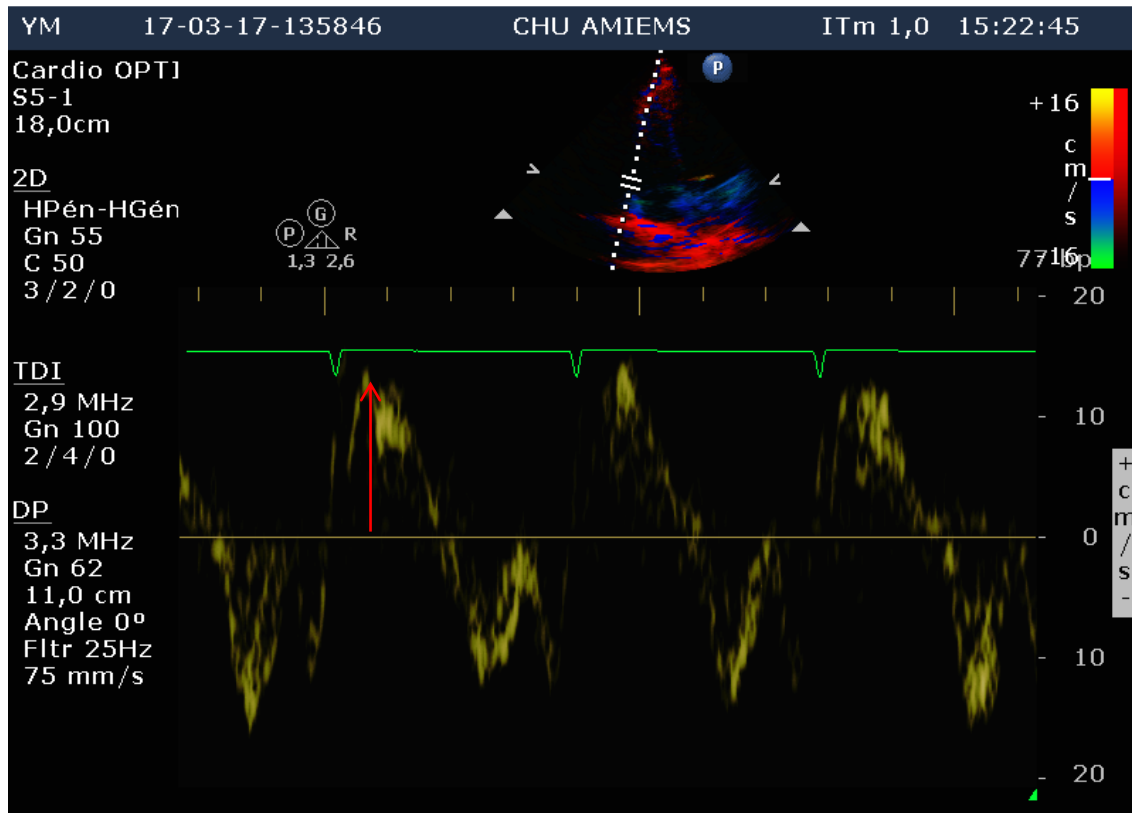
Measurement of myocardial longitudinal velocity



St maximal velocity

normal: $15,5 \pm 2,6$ cm/s

RV dysfunction: St < 9,5 cm/s

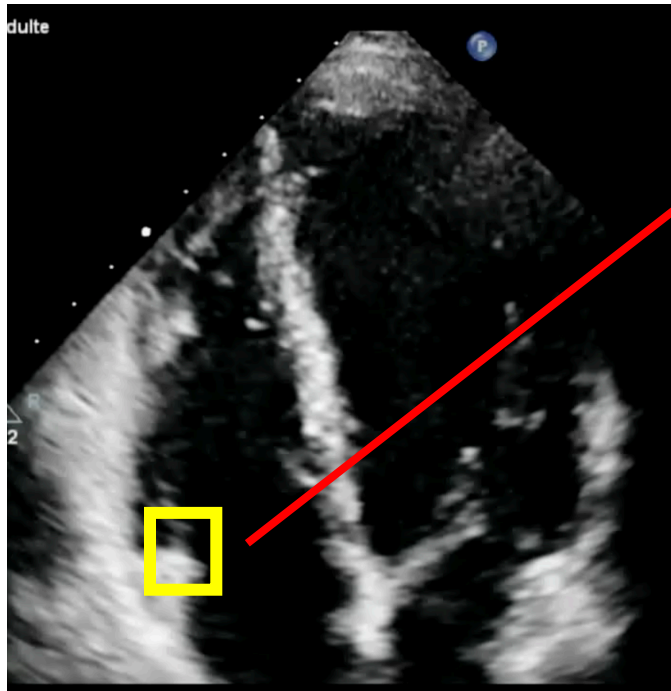


Mahjoub Y, Crit Care Med 2009

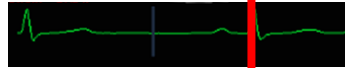
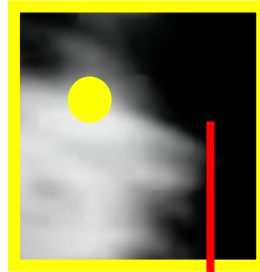
Meluzin J, Eur Heart J, 2001

Turhan S, Echocardiogr 2007

Le speckle tracking et 2D - strain



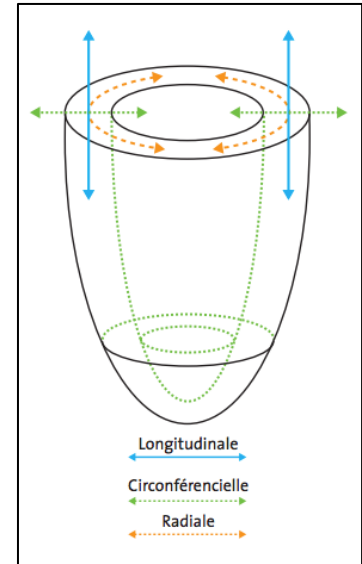
Frame n



Frame $n+1$

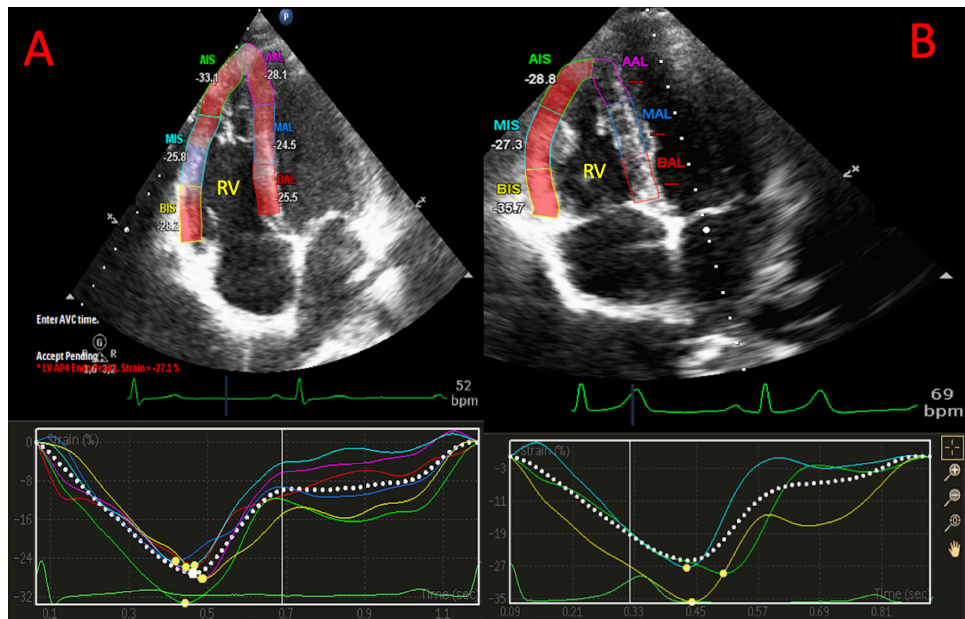
Diagram illustrating the strain calculation. It shows a vertical zigzag line representing a segment of length L_0 in the initial state and a shorter zigzag line of length L_1 in the final state.

$$\text{Strain} = \frac{L_1 - L_0}{L_0} \times 100$$





Le 2D Strain - Référence



ESC

European Society
of Cardiology

European Heart Journal - Cardiovascular Imaging (2018) 19, 591–600
doi:10.1093/ehjci/ey042

**EACVI CONSENSUS
DOCUMENT**

Standardization of left atrial, right ventricular, and right atrial deformation imaging using two-dimensional speckle tracking echocardiography: a consensus document of the EACVI/ASE/Industry Task Force to standardize deformation imaging

Variabilité mais peu de différence significative entre les techniques ou logiciel

Cut off selon pathologie (-20%)

Badano, EHJ CI, 2018

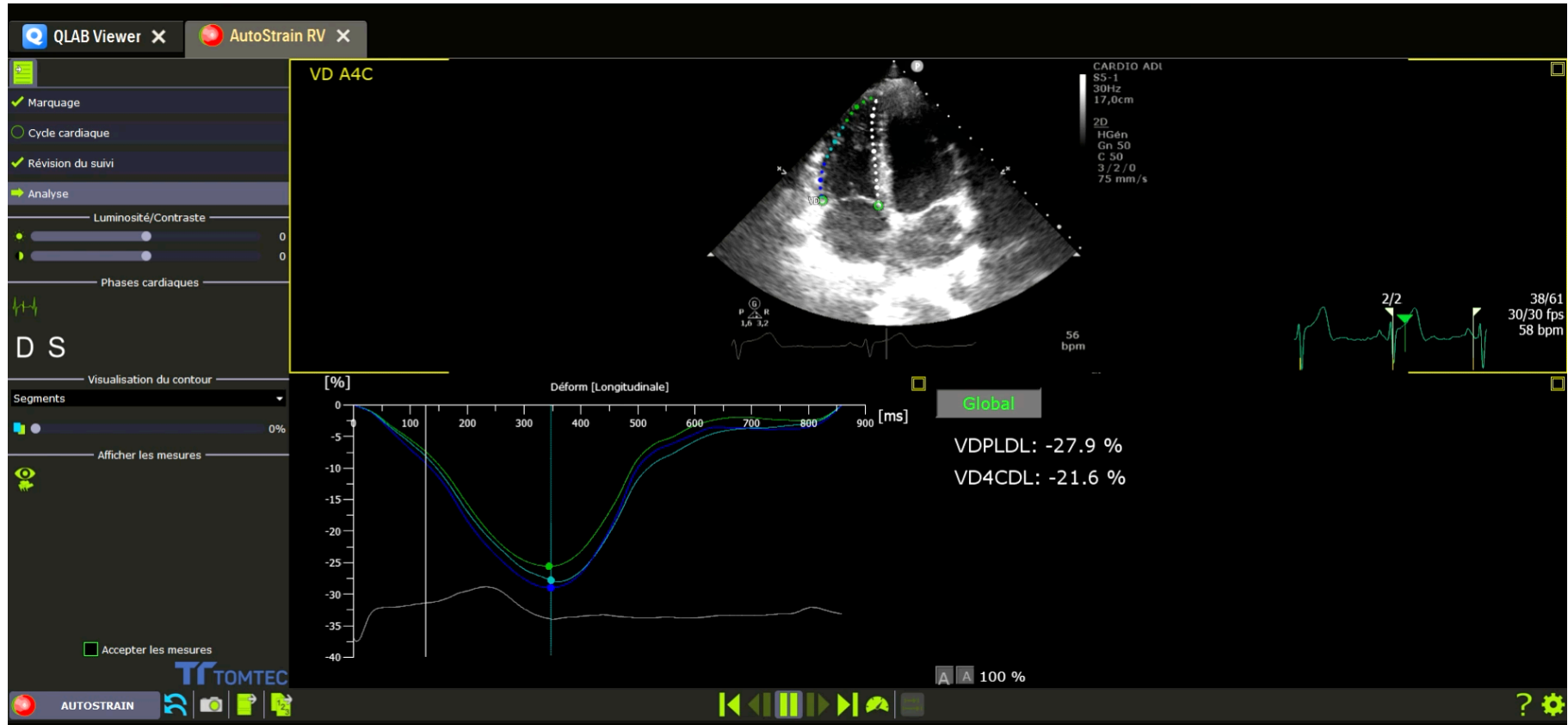
Il'Giovine, JASE, 2018



Centre Hospitalier Universitaire
Amiens - Picardie
80054 Amiens Cedex 1
www.chu-amiens.fr



Le strain VD - AutoStrain



Dysfonction droite Covid-19

Observational Study

> [Int J Cardiol.](#) 2021 Mar 15;327:251-258.

doi: 10.1016/j.ijcard.2020.11.043. Epub 2020 Nov 23.

Right ventricular dysfunction in critically ill COVID-19 ARDS

Caroline Bleakley ¹, Suveer Singh ², Benjamin Garfield ², Marco Morosin ², Elena Surkova ³,
Ms Sundhiya Mandalia ⁴, Bernardo Dias ³, Emmanouil Androulakis ⁵, Laura C Price ⁶,
Colm McCabe ⁶, Stephen John Wort ⁶, Cathy West ³, Wei Li ⁷, Rajdeep Khattar ⁷,
Roxy Senior ⁷, Brijesh V Patel ², Susanna Price ⁸

Observational Study

> [Echocardiography.](#) 2021 Feb;38(2):222-229. doi: 10.1111/echo.14966.

Epub 2020 Dec 24.

A pilot study on right ventricular longitudinal strain as a predictor of outcome in COVID-19 patients with evidence of cardiac involvement

> [J Intensive Care Med.](#) 2021 Mar 30;8850666211006335. doi: 10.1177/08850666211006335.

Online ahead of print.

Right Ventricular Strain Is Common in Intubated COVID-19 Patients and Does Not Reflect Severity of Respiratory Illness

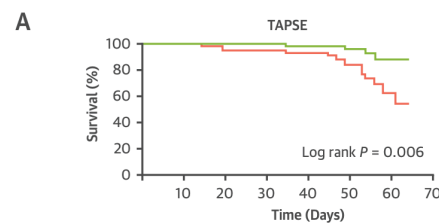
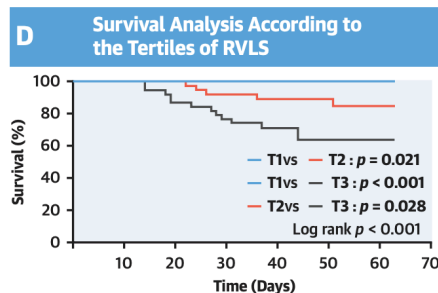
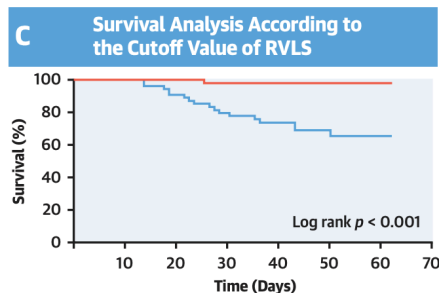
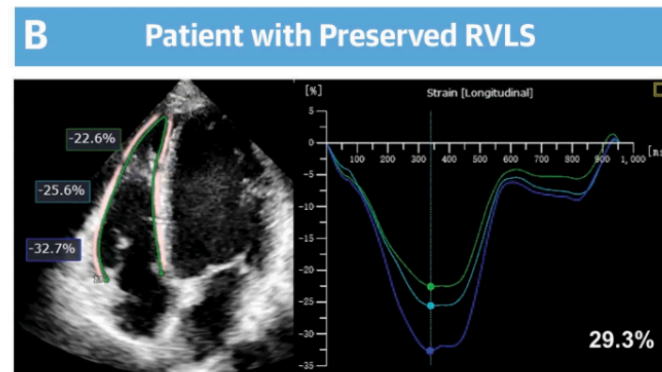
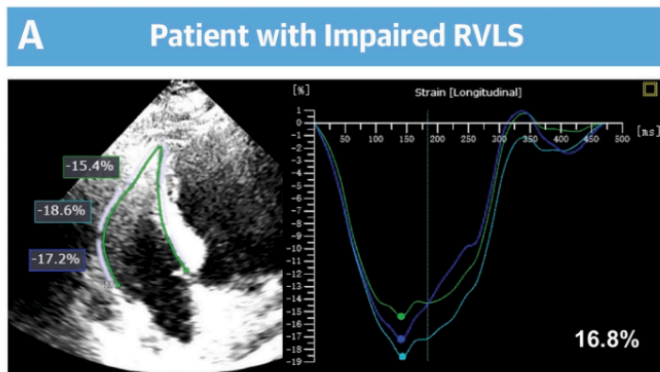
Comment

> [JACC Cardiovasc Imaging.](#) 2020 Aug;13(8):1859. doi: 10.1016/j.jcmg.2020.06.019.

Epub 2020 Jul 4.

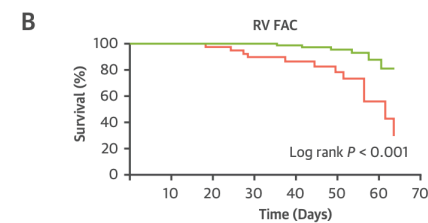
Right Ventricular Longitudinal Strain: A New Prognostic Tool for COVID-19?

COVID et 2D strain



Number at risk

TAPSE >23 mm	56	56	56	54	45	35	4	0
TAPSE ≤23 mm	64	64	59	51	38	27	5	0



Number at risk

FAC >43.5%	78	78	78	73	61	47	5	0
FAC ≤43.5%	42	42	37	32	22	15	4	0

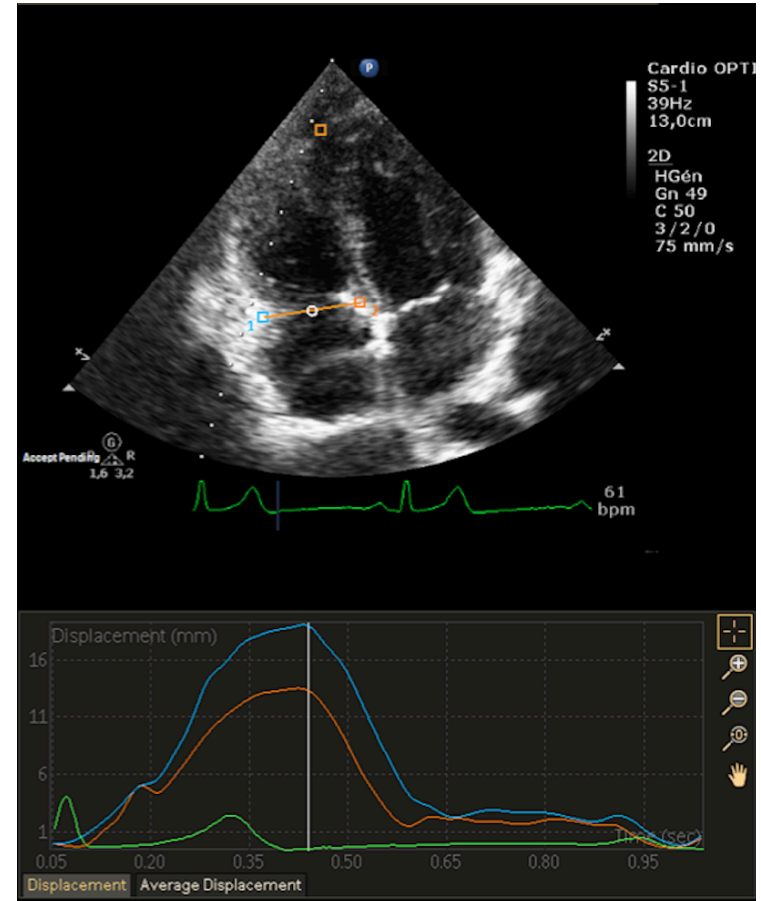
Le TAD ou TMAD

Application Philips – TMAD tricuspide en 2D

- Le TAD latéral (mm)
- Le TAD septal (mm)
- Le RVLSF (%)

Avantages :

- Angle indépendant
- Limite de l'endocarde
- Rapidité





Patients



BB00

N° du patient 18-10-11-090635

Date de naissance Inconnu

Sexe Inconnu



Système

Aide

PHILIPS

QLAB Viewer

aCMQ

aCMQ

Global

Défini par utilis.

Déplacement annulaire(TMAD)

Cycles cardiaques

Région d'intérêt

Sélect. une boucle

Boucle 1

Confirmer une vue

A4C

A2C

Dessiner/éditer ROI

Dessiner

Editer

Superpositions et mesures

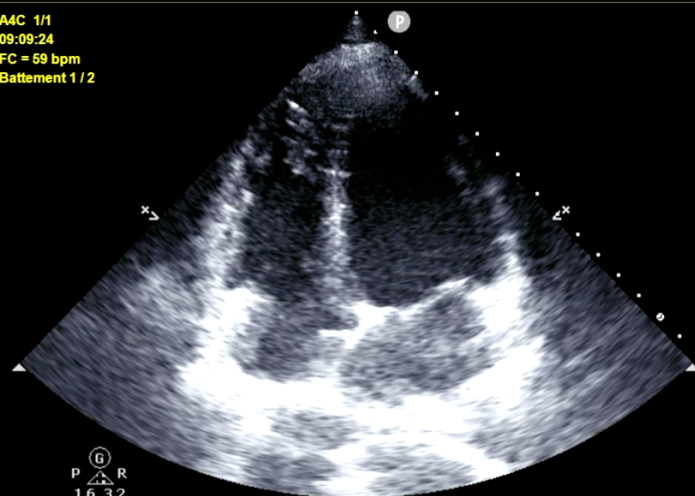
Afficher et masquer

A4C 1/1

09:09:24

FC = 59 bpm

Battement 1 / 2



CARDIO ADI

S5-1

30Hz

17,0cm

2D

HGén

Gn 50

C 50

3 / 2 / 0

75 mm/s

P 1,6
R 3,2

56
bpm

1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31 32 33 34 35

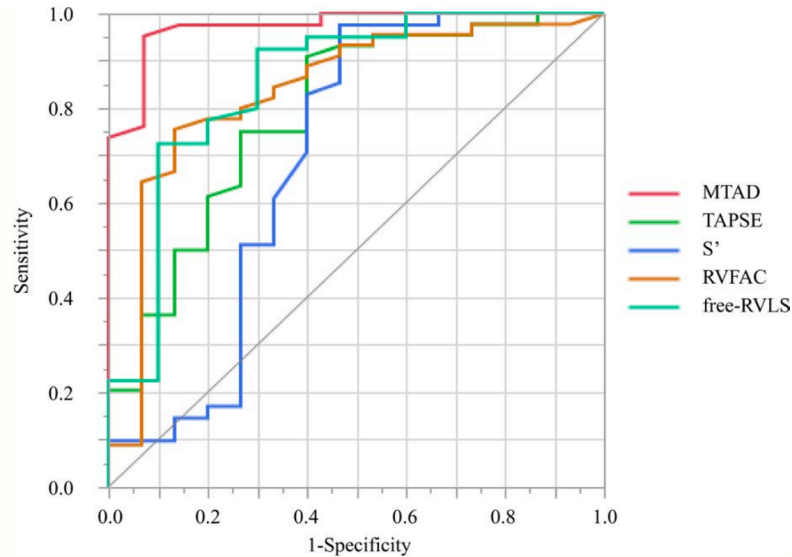
1/31 [3/61] 0.00s/0.99s (0.033s, 30Hz, 0.000s)



LAPTOP-I29SQ5A2\beyls

29-mars-2021 13:14

TAD et dysfonction VD – ETT 3D



	AUC	Cutoff value	Sensitivity	Specificity
MTAD	0.97	<14.7	93	95
Free-RVLS	0.86	>-16.2	70	92
RVFAC	0.84	<36	87	76
TAPSE	0.79	<16.5	60	91
S'	0.70	<9.5	53	98

Maniwa et al, Echocardiography, 2018

Dysfonction VD < 45% en ETT 3D (n=81)

AUC supérieur du RV-LSF vs TAPSE, RVFAC et RV FWLS pour prédire la dysfonction VD

TAD et dysfonction VD - IRM

Conventional parameters of RV function and TMAD parameters.

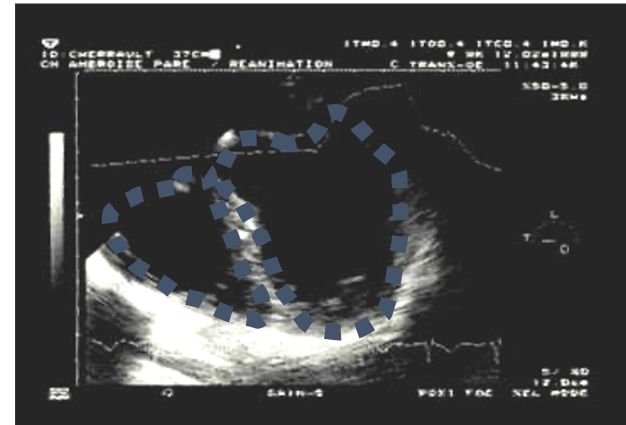
Parameter	Group I (n=182)	Group II (n=43)	P
Conventional parameters			
TAPSE, mm	15.12 ± 3.95	19.50 ± 2.88	<.001
RIMP	0.75 ± 0.28	0.50 ± 0.08	<.001
RVFAC, %	33.40 ± 9.32	50.12 ± 7.96	<.001
s', cm/s	10.98 ± 2.95	12.99 ± 3.05	<.001
e'/a'	0.51 ± 0.04	0.74 ± 0.29	.308
TMAD parameters			
TMAD1, mm	13.82 ± 4.18	17.10 ± 3.52	<.001
TMAD2, mm	8.44 ± 3.00	11.60 ± 2.87	<.001
TMADm, mm	11.78 ± 3.25	14.37 ± 2.83	<.001
TMADm%	15.48 ± 4.81	21.76 ± 4.31	<.001

Variable	AUC	Standard Error	95% CI
RV-LSF (%)	0.975	0.030	0.841-1.000
TAPSE	0.815	0.112	0.631-0.932
RIMP	0.833	0.114	0.653-0.944
RVFAC	0.963	0.035	0.822-0.999
s'	0.889	0.086	0.720-0.974

Li et al, Medicine, 2018

Patients avec HTP pré-capillaire

AUC supérieur du RV-LSF vs TAPSE et vs RVFAC pour prédire la dysfonction VD en IRM



Major Dilatation : RV EDA/LV EDA > 1

Usefulness of Right Ventricular Longitudinal Shortening Fraction to Detect Right Ventricular Dysfunction in Acute Cor Pulmonale Related to COVID-19

Christophe Beyls, MD^{*,1,2}, Yohann Bohbot, MD[†],
Pierre Huette, MD^{*}, Thomas Booz, MD^{*}, Camille Daumin, MD^{*},
Osama Abou-Arab, MD, PhD^{*}, Yazine Mahjoub, MD, PhD^{*}

^{*}Department of Anesthesiology and Critical Care Medicine, Amiens University Hospital, Amiens, France

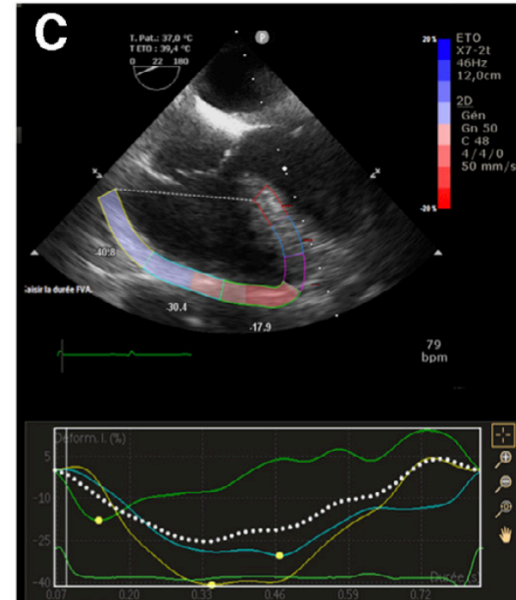
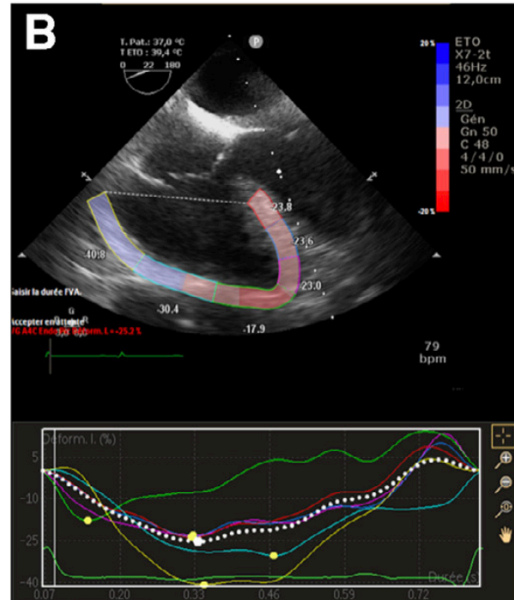
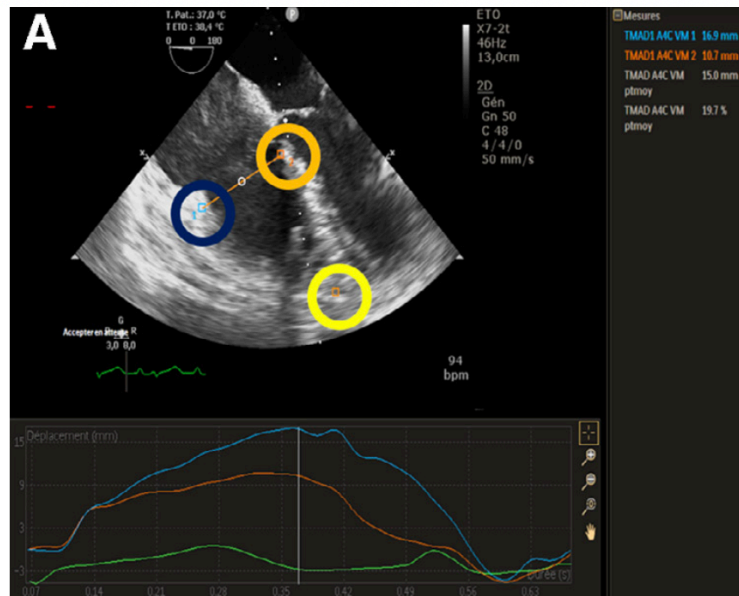
[†]Department of Cardiology, Amiens University Hospital, Amiens, France

COVID et CPA

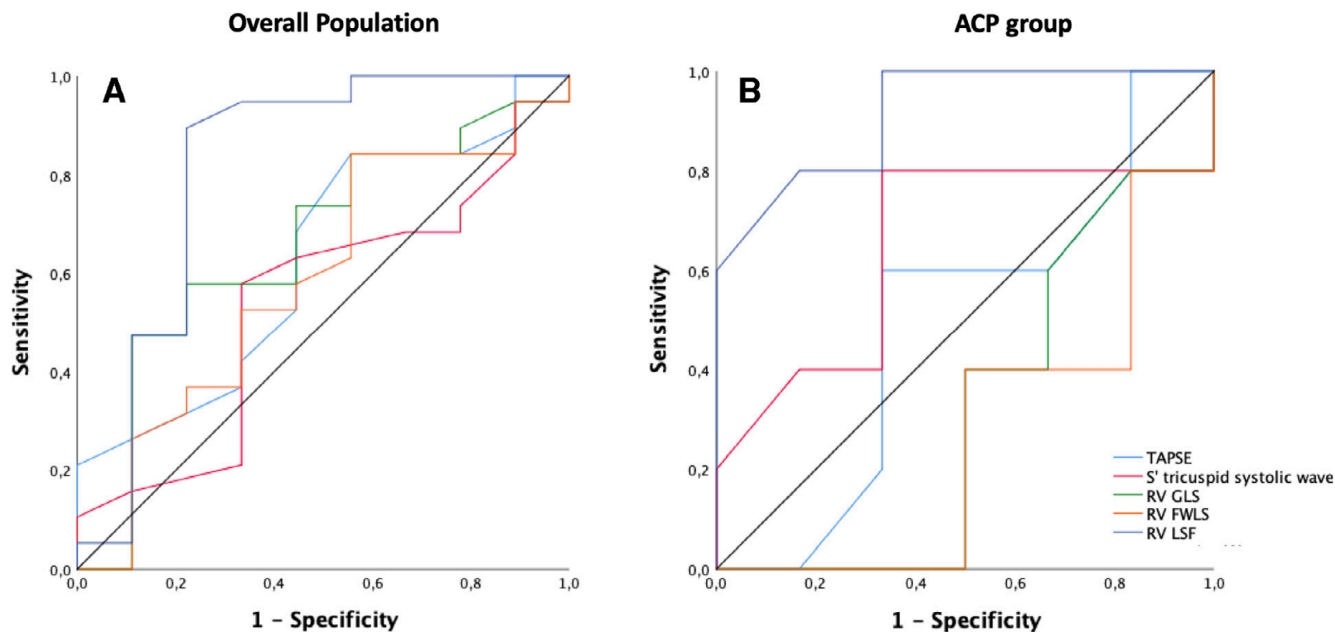
Contents lists available at [ScienceDirect](https://www.sciencedirect.com)

Journal of Cardiothoracic and Vascular Anesthesia

journal homepage: www.jcvaonline.com



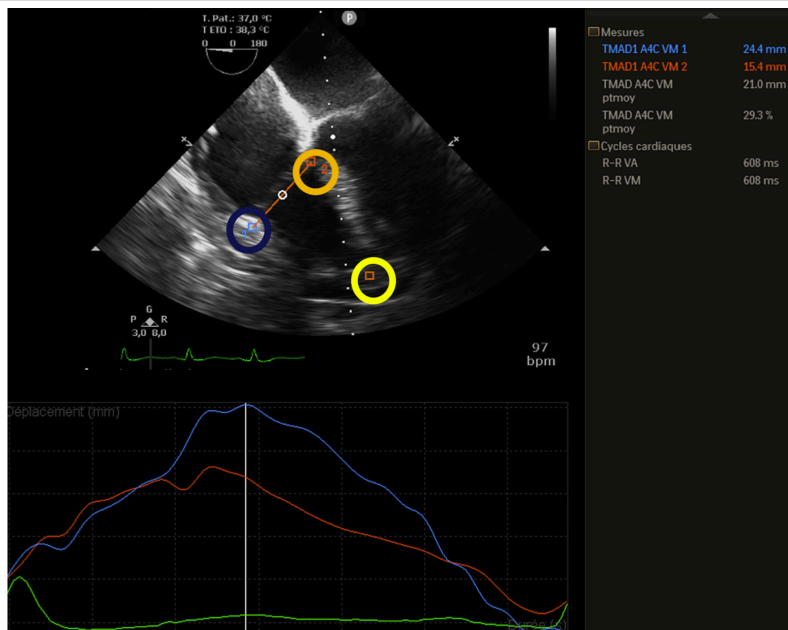
COVID et CPA



ETO
41% CPA

AUC supérieur du RV-LSF vs RV FWLS, RV GLS et marqueur conventionnels pour prédire la dysfonction VD

Reproductibilité et SDRA



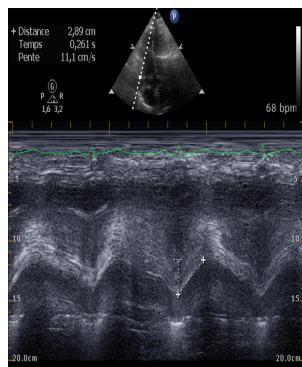
2D-STE parameters	ICC for intra-operator	IC95%	ICC for inter-operator	IC95%
<i>TAD parameters</i>				
RV-LSF (%)	0.96	0.74-0.98	0.93	0.74-0.98
TAD _{lat} (mm)	0.98	0.93-0.99	0.89	0.58-0.97
TAD _{sep} (mm)	0.96	0.85-0.98	0.93	0.73-0.98
<i>Strain parameters</i>				
RVGLS (%)	0.92	0.72-0.98	0.92	0.68-0.98
RVFWLS (%)	0.88	0.6-0.97	0.84	0.37-0.96

ETO réalisée en décubitus ventrale – Faisabilité 100 % - Pas d'ES

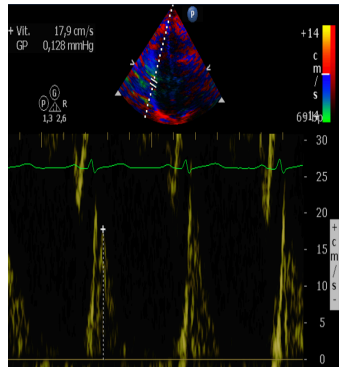
Monitoring of the right ventricle and volume

4 CAVITE MULTIPARAMETRIQUE

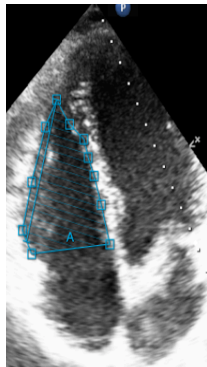
MARQUEURS CONVENTIONNELLS



< 17 mm

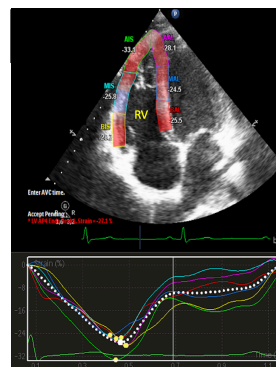


< 9.5 cm/s

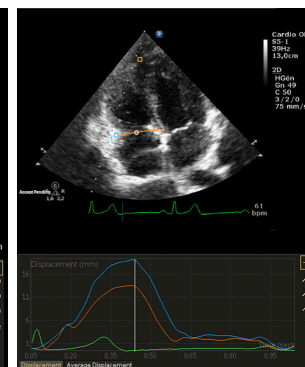
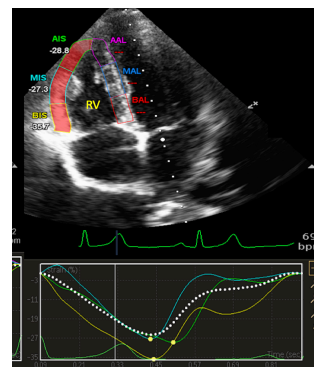


< 35%

MARQUEURS 2D STE



< 21% - Range ++



?

Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China

Chaolin Huang*, Yeming Wang*, Xingwang Li*, Lili Ren*, Jianping Zhao*, Yi Hu*, Li Zhang, Guohui Fan, Jiuyang Xu, Xiaoying Gu, Zhenshun Cheng, Ting Yu, Jiaan Xia, Yuan Wei, Wenjuan Wu, Xuelei Xie, Wen Yin, Hui Li, Min Liu, Yan Xiao, Hong Gao, Li Guo, Jungang Xie, Guangfa Wang, Rongmeng Jiang, Zhancheng Gao, Qi Jin, Jianwei Wang†, Bin Cao†

www.thelancet.com Vol 395 February 15, 2020

1. *Lancet* 2020; 395: 476–81. doi:10.1016/S0140-6736(20)30183-2

	All patients (n=41)	ICU care (n=13)	No ICU care (n=28)	p value
Hypersensitive troponin I, pg/mL	3·4 (1·1–9·1)	3·3 (3·0–163·0)	3·5 (0·7–5·4)	0·075
>28 (99th percentile)	5/41 (12%)	4/13 (31%)	1/28 (4%)	0·017
B-type natriuretic peptide, pg/mL	0·1 (0·1–0·1)	0·1 (0·1–0·1)	0·1 (0·1–0·1)	0·021

Cardiac Injury in COVID-19

A. DIRECT VIRAL INJURY

B. MEDICATION TOXICITY

Previous medical history and medication toxicity can affect patients with COVID disease and cause cardiac injuries.

C. UNSTABLE HAEMODYNAMIC

Hypoxaemia and hypoperfusion can cause myocardial injury and cardiac injury.

D. ↑ SYSTEMATIC INFLAMMATION

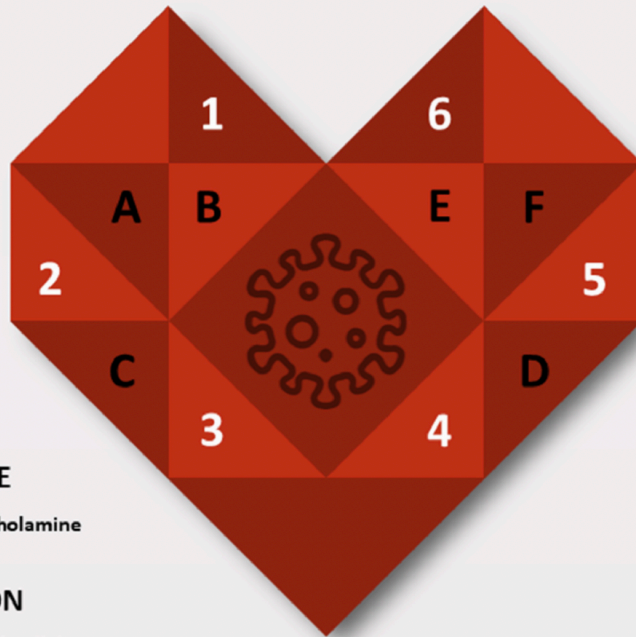
Elevations in inflammatory markers, such as interleukin (IL)-6, ferritin, and erythrocyte sedimentation rate, increases systemic inflammation.

E. ↑ ENDOGENOUS CATECHOLAMINE

Extreme anxiety leading to further endogenous catecholamine release, causing myocardial electrical instability.

F. ACE2 RECEPTOR DOWNREGULATION

SARS-CoV infection causes robust downregulation of cellular ACE2 expression levels and it has been suggested that the SARS-CoV effect on ACE2 is involved in the severity of disease. The decline of ACE2 expression is dependent on the efficiency of HCoV-NL63 replication, and that HCoV-NL63 and SARS-CoV both affect cellular ACE2 expression during infection.



1. CHEST PAIN

One of the most common manifestation in early cardiac injury.

2. ARRHYTHMIA

Such as ventricular tachycardia, atrial tachycardia, and atrioventricular block.

3. MYOCARDIAL INJURY

Direct myocardial injury due to viral involvement of cardiomyocytes and the effect of systemic inflammation appear to be the most common mechanisms responsible for cardiac injury.

4. HEART FAILURE

The onset of symptoms several days after influenza-like syndrome may reflect these proposed mechanisms with potential myocyte dissemination of the virus, the immune system's activation, and, ultimately, the clinical onset of heart failure.

5. SHOCK

Major complications during hospitalization included ARDS, cardiac injury, and shock.

6. SUDDEN CARDIAC DEATH

CONCLUSION

- COVID-19 = Maladie vasculaire qui touche le poumon
- Nécessite une approche vasculaire du poumon



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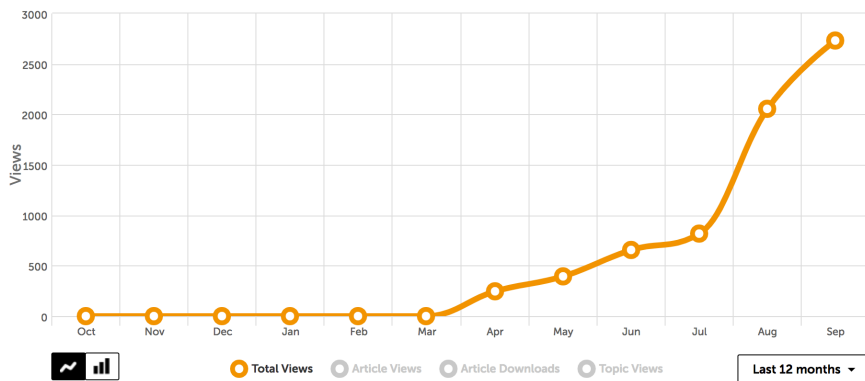
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Merci pour votre attention