Tretman pacijenata sa COVID-19 u jedinici intenzivne njege sa posebnim osvrtom na ARDS

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Respiratorno udruženje u Bosni i Hercegovini

Stručni skup „Najbolja pulmološka praksa u vezi sa COVID19
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Sažetak

- Epidemiološki podaci
- ARDS vs.*COVID-19 ARDS*
- Patofiziologija ARDS-a u COVID-19
- Klinička slika (fenotip “L” i “H”)
- Dijagnostičke procedure i laboratorijski parametri
- Kriteriji za prijem pacijenta u ICU
- Tretman ICU/neintubiranog pacijenta
- Tretman intubiranog pacijenta
- Antikoagulantna terapija
- Izazovi

Epidemiološki podaci

Tzotzos et al. Critical Care (2020) 24:516
https://doi.org/10.1186/s13054-020-03240-7

Critical Care

RESEARCH LETTER Open Access

Incidence of ARDS and outcomes in hospitalized patients with COVID-19: a global literature survey

Table 1 Incidence of ARDS, ICU admission, IMV treatment, and outcome in hospitalized COVID-19 patients

Study author, online publication date, doi	Location	Number of patients, *N*	Patients with ARDS	Patients transferred to ICU	Patients who received IMV	Mortality hospitalized patients
Huang C, 24/1; doi: https://doi.org/10.1016/S0140-6736(20)30183-5	Wuhan, China	41	12/41 (29)	13/41 (32)	4/41 (10)	6/41 (15)
Wang D, 7/2; doi: https://doi.org/10.1001/jama.2020.1585						
Xu X,19/2; doi: https://doi.org/10.1136/bmj.m606						
Zhou F, 11/3; doi: https://doi.org/10.1016/S0140-6736(20)30566-3						
Wu C, 13/3; doi: https://doi.org/10.1001/jamainternmed.2020.0994						
Dreher M, 17/4; doi: https://doi.org/10.3238/arztebl.2020.0271						
Yao Q, 24/4; doi: https://doi.org/10.20452/pamw.15312						
Aggarwal S, 26/4; doi: https://doi.org/10.1515/dx-2020-0046						
Inciardi R, 8/5; doi: https://doi.org/10.1093/eurheartj/ehaa388						
Hong K, 11/5; doi: https://doi.org/10.3349/ymj.2020.61.5.431						
Yang L, 26/5; doi: https://doi.org/10.1016/j.jcv.2020.104475						
Suleyman G, 1/6; doi: https://doi.org/10.1001/jamanetworkopen.2020.12270						
Zheng Y, 10/4; doi: https://doi.org/10.1016/j.jcv.2020.104366						
Chen N, 30/1; doi: https://doi.org/10.1016/S0140-6736(20)30211-7						
Chen T, 26/3; doi: https://doi.org/10.1136/bmj.m1091						
Yu T, 27/4; doi: https://doi.org/10.1016/j.clinthera.2020.04.009						
Ciceri F, 12/6; doi: https://doi.org/10.1016/j.clim.2020.108509						
Total (weighted average)						

Data are presented as *n/N(%)*
Abbreviations: *ARDS* acute respiratory distress syndrome, *COVID-19* coronavirus disease
[a]From a hospitalized cohort of 799 COVID-19 patients, data available for 274 patients
[b]Patient numbers for Chen T study not included

Table 2 Incidence of ARDS, IMV treatment, and outcomes in COVID-19 patients admitted to an ICU

Study author, online publication date[a]	Location	ICU patients with ARDS	ICU patients who received IMV	Mortality ICU patients	Mortality ARDS patients	Mortality IMV patients	Prevalence of ARDS in non-survivors
Dreher M, 17/4	Aachen, Germany	24/24 (100)	24/24 (100)	3/24 (13)	3/24 (13)	3/24 (13)	3/7 (43)
Yao Q, 24/4	Huanggang, China	17/17 (100)	10/17 (59)	12/17 (71)	12/45 (27)	10/10 (100)	12/12 (100)
Hong K, 11/5	Daegu, South Korea	13/13 (100)	11/13 (85)	4/13 (31)	4/18 (22)	NA	4/5 (80)
Xu X,19/2	Zhejiang, China	1/1 (100)	1/1 (100)	0/1 (0)	0/1 (0)	0/1 (0)	NA
Wu C, 13/3	Wuhan, China	NA	6/53 (11)	NA	44/84 (52)	6/6 (100)	44/44 (100)
Zhou F, 11/3	Wuhan, China	NA	NA	39/50 (78)	50/59 (85)	31/32 (97)	50/54 (93)
Zheng Y, 10/4	Chengdu, China	15/32 (47)	NA	3/32 (9)	3/15 (20)	NA	3/3 (100)
Suleyman G, 1/6	Detroit, Michigan, USA	104/141 (74)	114/141 (81)	57/141 (40)	NA	52/114 (46)	NA
Huang C, 24/1	Wuhan, China	11/13 (85)	4/13 (31)	5/13 (38)	NA	NA	NA
Wang D, 7/2	Wuhan, China	22/36 (61)	21/36 (58)	6/36 (17)	NA	NA	NA
Yang L, 26/5	Yichang, China	21/29 (72)	16/29 (55)	14/29 (48)	NA	NA	NA
Chen T, 26/3	Wuhan, China	NA	NA	NA	113/196 (58)	17/17 (100)	113/113 (100)
Aggarwal S, 26/4	Des Moines, Iowa, USA	NA	5/8 (63)	3/8 (38)	NA	NA	NA
Inciardi R, 8/5	Brescia, Italy	NA	NA	NA	17/19 (89)	NA	17/26 (65)
Chen N, 30/1	Wuhan, China	NA	NA	NA	11/17 (65)	NA	11/11 (100)
Ciceri F, 12/6	Milan, Italy	NA	NA	NA	65/245 (27)	NA	65/82 (79)
Yu T, 27/4	Dongguan, China	NA	NA	NA	NA	NA	NA
Total (weighted average)		228/306 (75)	212/335 (63)	146/363 (40)	322/722 (45)	119/203 (59)	322/357 (90)

Data are presented as *n/N(%)*
Abbreviations: *ARDS* acute respiratory distress syndrome, *COVID-19* coronavirus disease 2019, *ICU* intensive care unit, *IMV* invasive mechanical ventilation, *NA* not available: insufficient data for calculation
[a]For study reference see Table 1

EDITORIALS

Check for updates

Biological Mechanisms of COVID-19 Acute Respiratory Distress Syndrome

Am J Respir Crit Care Med Vol 202, Iss 11, pp 1489–1502, Dec 1, 2020

ARDS vs.*COVID-19 ARDS (CARDS)*

ARDS ⟶ isključen kardiogeni edem pluća i ostali mogući uzroci akutnog hipoksemijskog respiratornog zatajenja i bilateralnih plućnih infiltrata!!!
(Berlinski kriteriji)

Perspective

It is time to update the ARDS definition: It starts with COVID-19-induced respiratory failure☆

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ARTICLE INFO

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COVID-19
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Respiratory failure
Silent hypoxemia
Histopathological changes

ABSTRACT

Coronavirus disease 2019 (COVID-19) may rapidly worsen respiratory failure, thereby leading to death. COVID-19-induced respiratory failure exhibits some atypical characteristics, silent hypoxemia, and high lung compliance. Some histopathological changes associated with COVID-19-induced respiratory failure differ from those of classic acute respiratory distress syndrome (ARDS). However, compared with classical ARDS, COVID-19-induced respiratory failure has a similar timing of onset, clinical syndromes, radiological profile, and mortality rate in the intensive care unit (ICU). Respiratory failure induced by COVID-19 is a type of ARDS and is currently underdiagnosed. This condition stretches the definition of classic ARDS; therefore, an updated definition is warranted.

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≥5cmH_2O)
    - Mild (200mmHg≤PaO_2/FiO_2<300mmHg)
    - Mild-moderate (150mmHg≤PaO_2/FiO_2<200 mmHg)
    - Moderate-severe (PaO_2/FiO_2<150mmHg)
- **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

Li and Ma Critical Care (2020) 24:198

Patofiziologija ARDS-a u COVID-19

american physiological society | JOURNAL OF APPLIED PHYSIOLOGY

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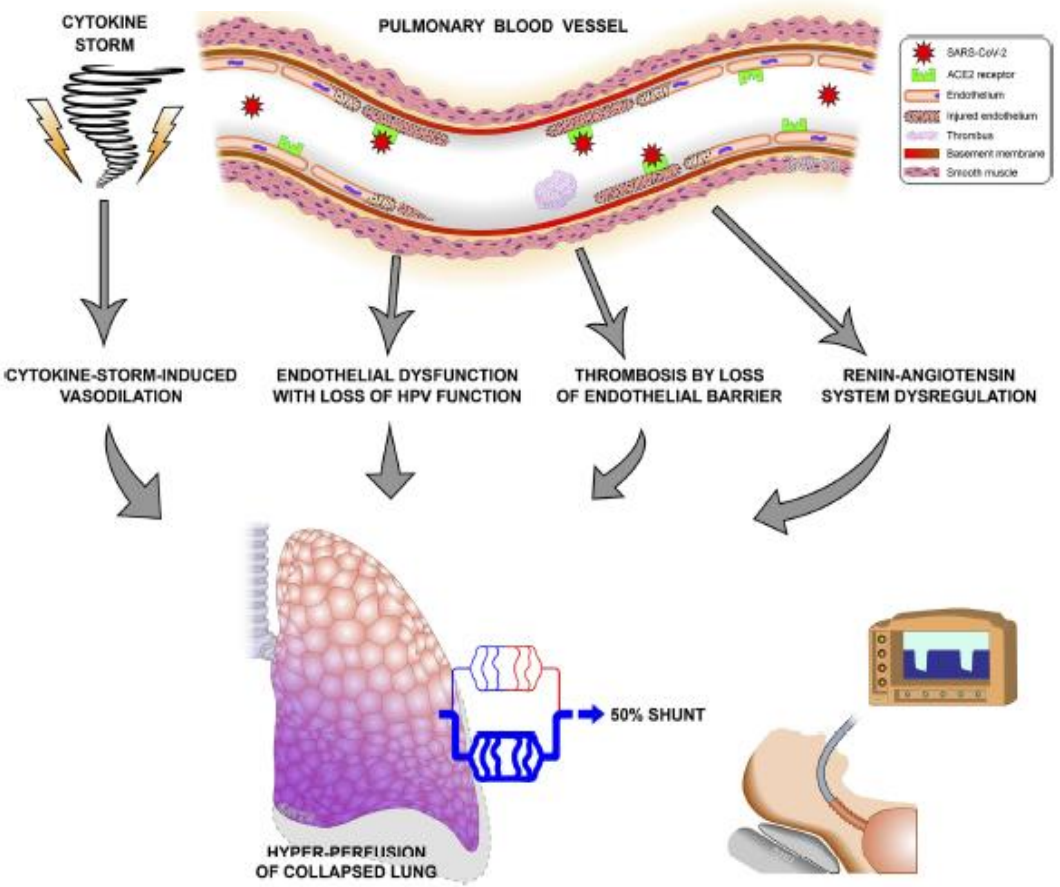
REVIEW

Physiology in Medicine

Functional pathophysiology of SARS-CoV-2-induced acute lung injury and clinical implications



- *Hipoksemijsko respiratorno zatajenje:* citopatski efekti virusa na pneumocite; nivo surfaktanta↓; nastanak atelektaza i difuzne alveolarne hemoragije; formiranje hijalinih membrana; *"silent hypoxemia"*

- *Citokinska oluja:* Neu i Mono → porast proinflamatornih citokina (IL-6), IL-1 i TNF

- *COVID-19- uzrokovana hiperkoagulabilnost:* protrombotsko stanje nastalo kao rezultat povišenja nivoa fibrina i fibrinogena

Klinička slika (fenotip "L" i "H")

Intensive Care Med (2020) 46:1099–1102
https://doi.org/10.1007/s00134-020-06033-2

EDITORIAL

COVID-19 pneumonia: different respiratory treatments for different phenotypes?

Luciano Gattinoni[1*], Davide Chiumello[2], Pietro Caironi[3,4], Mattia Busana[1], Federica Romitti[1], Luca Brazzi[5] and Luigi Camporota[6]

COVID-19 pneumonia, Type L

At the beginning, COVID-19 pneumonia presents with the following characteristics:

- *Low elastance.* The nearly normal compliance indicates that the amount of gas in the lung is nearly normal [4].
- *Low ventilation-to-perfusion (VA/Q) ratio.* Since the gas volume is nearly normal, hypoxemia may be best explained by the loss of regulation of perfusion and by loss of hypoxic vasoconstriction. Accordingly, at this stage, the pulmonary artery pressure should be near normal.
- *Low lung weight.* Only ground-glass densities are present on CT scan, primarily located subpleurally and along the lung fissures. Consequently, lung weight is only moderately increased.
- *Low lung recruitability.* The amount of non-aerated tissue is very low; consequently, the recruitability is low [5].

COVID-19 pneumonia, Type H

The Type H patient:

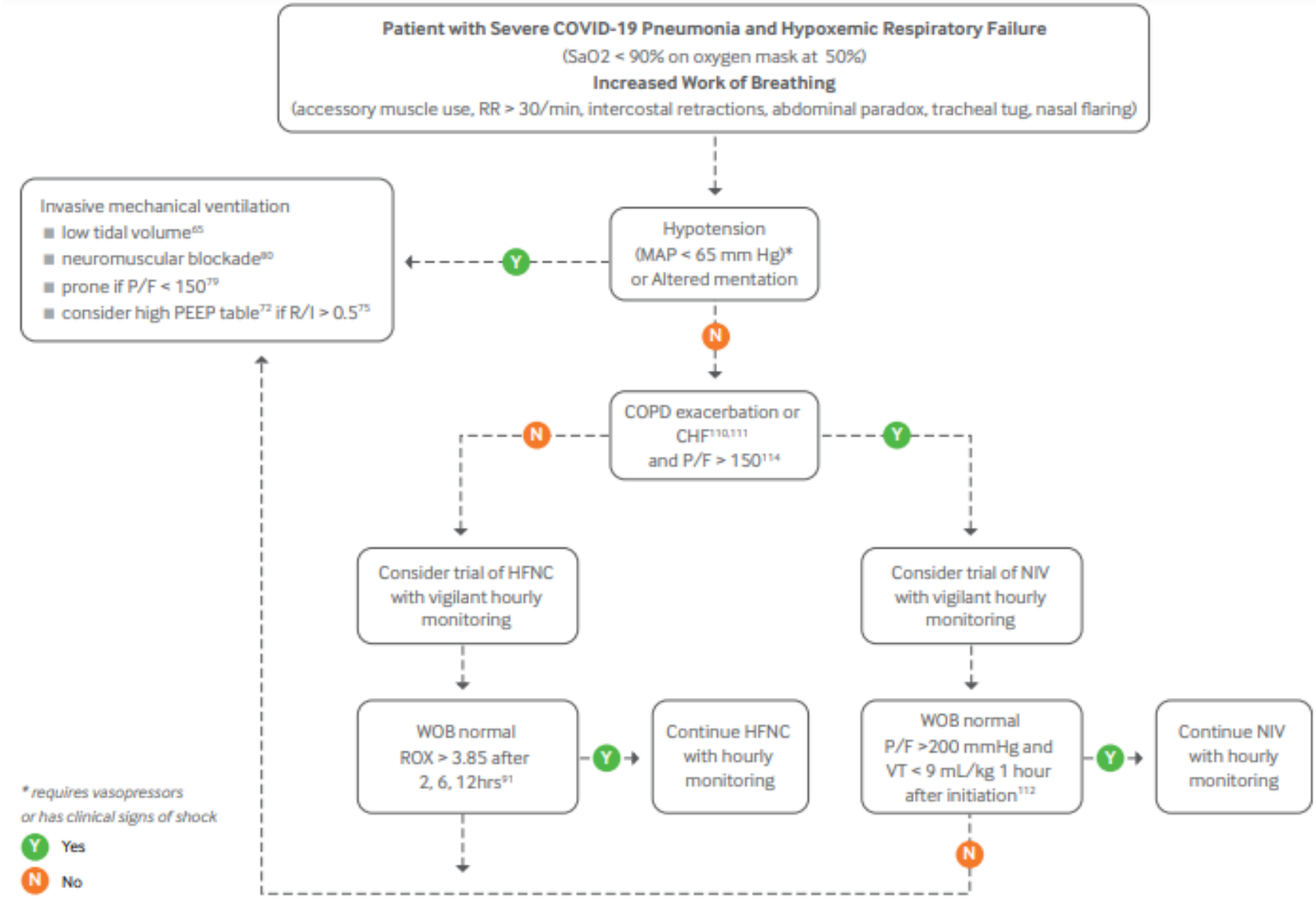
- *High elastance.* The decrease in gas volume due to increased edema accounts for the increased lung elastance.
- *High right-to-left shunt.* This is due to the fraction of cardiac output perfusing the non-aerated tissue which develops in the dependent lung regions due to the increased edema and superimposed pressure.
- *High lung weight.* Quantitative analysis of the CT scan shows a remarkable increase in lung weight (> 1.5 kg), on the order of magnitude of severe ARDS [12].
- *High lung recruitability.* The increased amount of non-aerated tissue is associated, as in severe ARDS, with increased recruitability [5].

Dijagnostičke procedure

- *Klinički pregled pacijenta*
- *Laboratorijski testovi*: neu↑, limf↓, AST↑, ALT↑, kreatinin, urea, CRP↑, PCT, feritin↑ i faktori koagulacije, PV, fibrinogen, D-dimer↑
- *Radiološke slikovne tehnike*:
  - Rtg p/c PA/AP
  - CT grudnih organa
  - UZ pluća
- *Faktori visokog rizika za kliničko pogoršanje i razvoj ARDS*: starost ≥65 godina, T>39C, RR≥30/min,spO2≤ 93% i plućni infiltrati>50%
- *Kriteriji za prijem pacijenta u ICU*: potreba za kiseoničkom potporom 6–8 l/min kako bi se postigla spO2≥ 90–92%, respiratorno zatajenje, šok, multiplo organsko zatajenje, kao i pacijenti pod visokim rizikom za kliničko pogoršanje stanja

Tretman neintubiranog pacijenta/Ventilacija

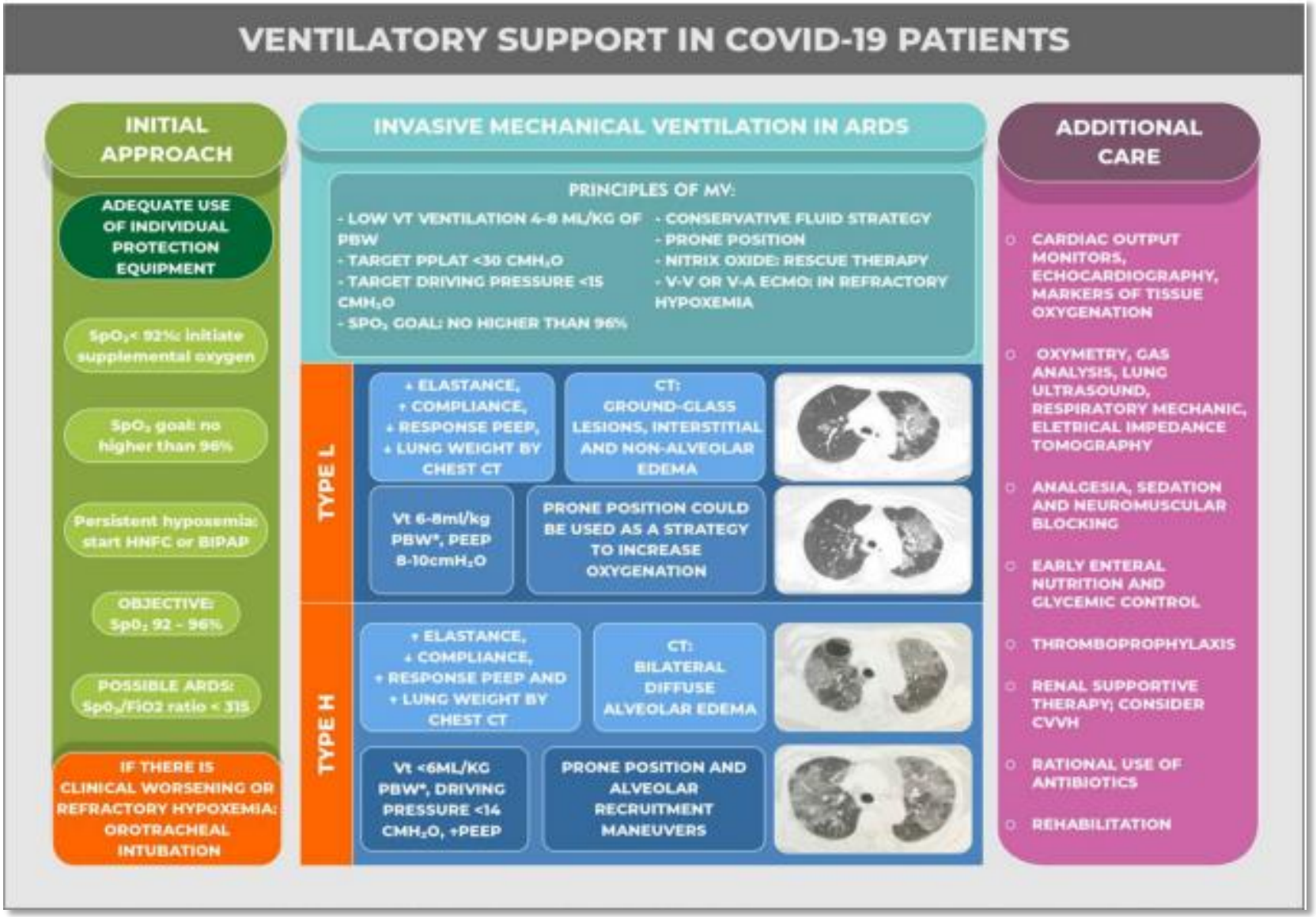
- Proniranje
- *Blaga do umjerena dispnea* i hipoksemija, bez adekvatnog odgovora na *low-flow* NC -HFNC uz proniranje
- NIV (CPAP vs.BiPAP) –alveolarni recruitment; posebni entiteti (HOPB, CHF)
- Alarmi: respiratorni pogon↑,umor respiratornih mišića i neadekvatna mehanika disanja, pogoršanje respiratorne acidoze kao i sniženo stanje svijesti – ETI i MV se ne smiju odlagati!!!
- *Prijeteći CARDS*- respiratorni pogon↑ može generirati visoke transpulmonalne pritiske -"patient self-inflicted lung injury" (P-SILI)



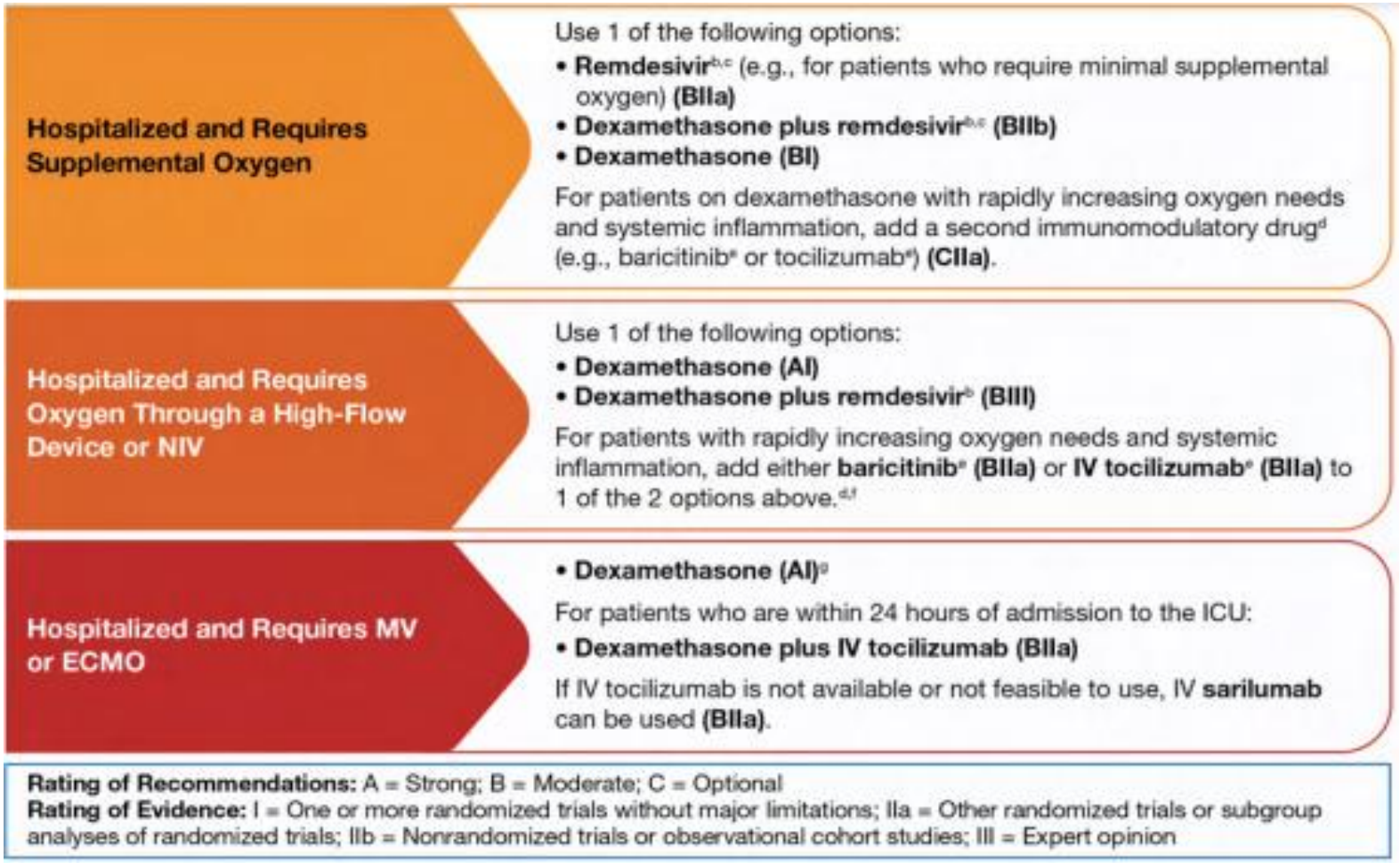
Attaway AH, Scheraga RG, Bhimraj A, Biehl M, Hatipoğlu U. Severe covid-19 pneumonia: pathogenesis and clinical management. BMJ. 2021 Mar 10;372:n436.

Tretman intubiranog pacijenta/Ventilacija

Hajjar et al. Ann. Intensive Care (2021) 11:36



Tretman ICU pacijenta/ Medikamentozni pristup



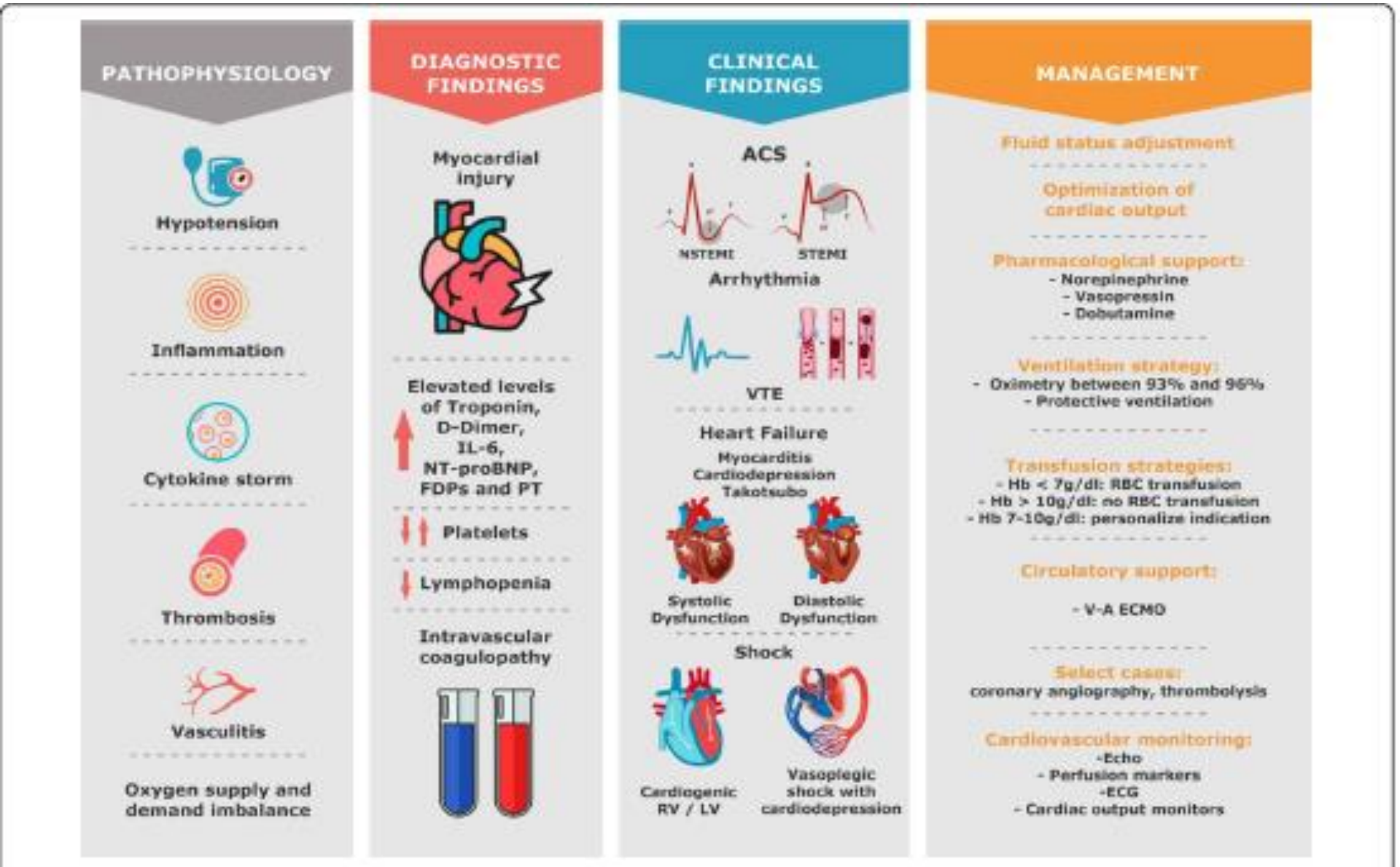
https://files.covid19treatmentguidelines.nih.gov/guidelines/covid19treatmentguidelines.pdf Pristup: 21.02.22.

Hajjar et al. Ann. Intensive Care (2021) 11:36
https://doi.org/10.1186/s13613-021-00820-w

Annals of Intensive Care

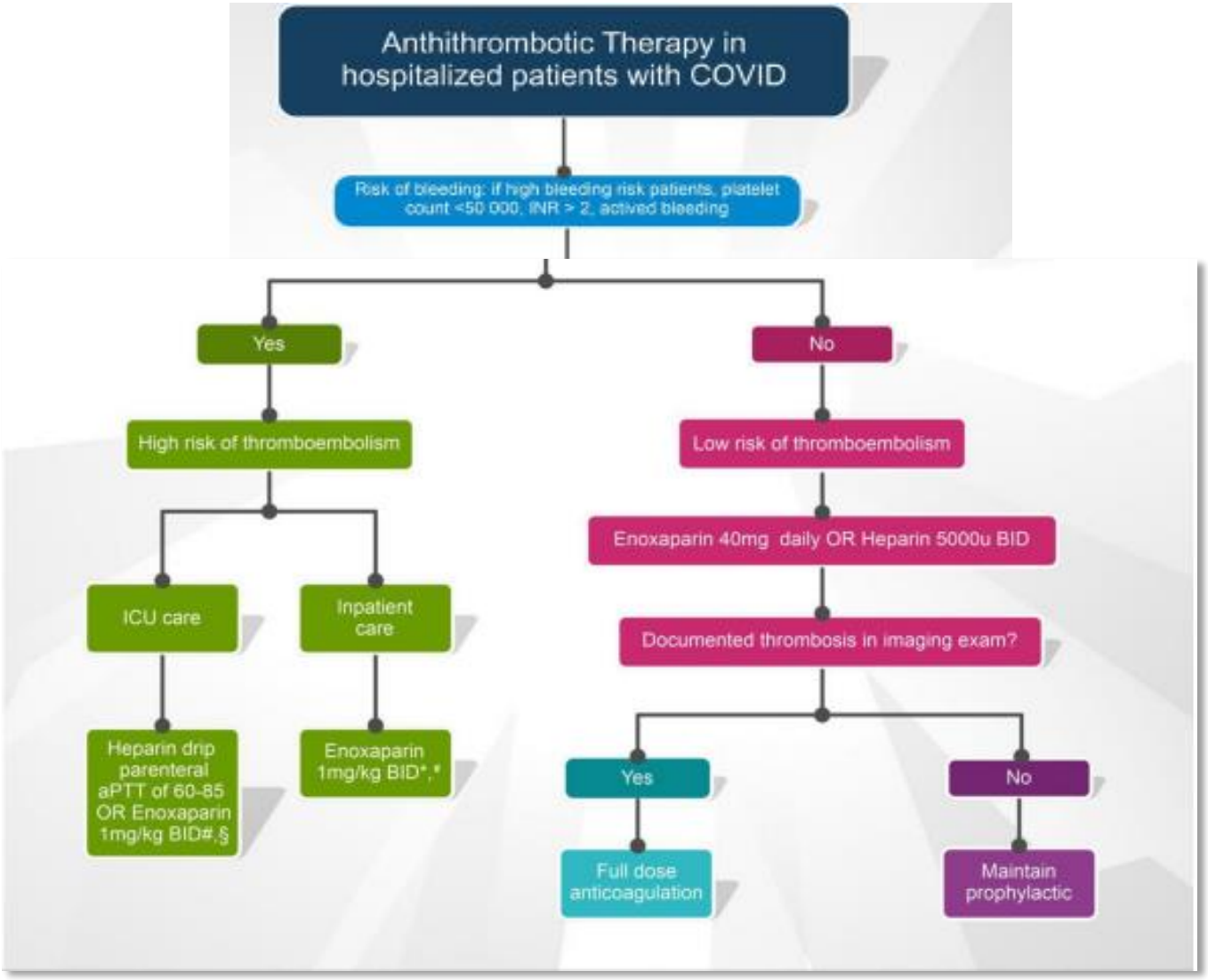
REVIEW Open Access

Intensive care management of patients with COVID-19: a practical approach



Antikoagulantna terapija

Hajjar et al. Ann. Intensive Care (2021) 11:36



IZAZOVI: *NO, ECMO*

Role of Pulmonary Vasodilator Therapies

Selective pulmonary vasodilation is thought to improve ARDS secondary to redistribution of blood from poorly ventilated areas to those with higher ventilation, thereby decreasing the shunt fraction and correcting hypoxemia. *Nitric Oxide* and *Prostaglandins* (e.g. PGI_2 [epoprostenol]), despite its pulmonary vasodilatory properties have failed to demonstrate a mortality benefit in ARDS.[44] Nitric Oxide use during the COVID-19 patients is controversial.[45] Some authors advocate for its potential anti-viral activity following the results of a study performed during the SARS-CoV outbreak in 2004.[46] Currently there is no recommendation for the use of pulmonary vasodilators in patients with ARDS due to COVID-19, other than a last resource (rescue therapy) for refractory hypoxemia.[23]

In order to determine patient eligibility, the ELSO consensus on CARDS provides a forthright algorithm centered in providing conservative management (prone positioning, neuromuscular blockade, pulmonary vasodilators, high PEEP, recruitment maneuvers) for patients with PaO2/FiO2 ratio < 150 mmHg. If the patient develops worsening refractory hypoxemia (PaO2/FiO2 ratio < 80 mmHg for > 6 h, *or* PaO2/FiO2 ratio < 50 mmHg for > 3 h) or signs of poor tissue perfusion and hypercarbia (pH < 7.25 with partial pressure of arterial carbon dioxide [PaCO2] > 60 mmHg), then the patient should be considered for ECMO, assuming no contraindications are present. Also, for patients with PaO2/FiO2 ratio > 150 mmHg, but with signs of poor tissue perfusion and hypercarbia, ECMO should be considered as well.[48]

Jose R. Navas-Blanco; Roman Dudaryk
Management of Respiratory Distress Syndrome due to COVID-19 Infection. BMC Anesthesiol. 2020;20(177)

Hvala za pažnju!!