# HEMODYNAMIC MANAGEMENT IN COVID-19

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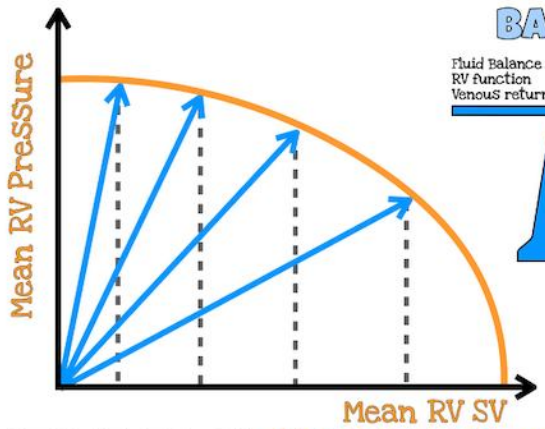
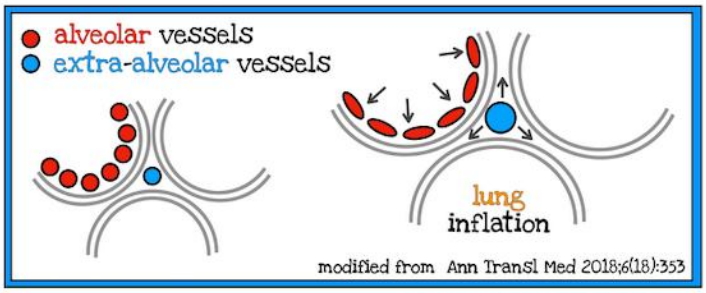
According to SSC guidelines (ICM 2020 Mar 28:1-34.) for adults with COVID-19 & shock, recommend measuring dynamic parameters to assess fluid responsiveness is suggestd (weak recommendation, LQE), and for acute resuscitation, suggest use of:
conservative fluid administration strategy (weak, very LQE)
crystalloids over colloids (strong; moderate QE); balanced crystalloids preferred over unbalanced (weak, moderate QE)
norepinephrine as first-line vasoactive agent (weak, LQE), and either vasopressin or epinephrine if NE not available (weak, LQE).
Physiology of right ventricle is complex, and still not fully understood; basically, the right ventricle is not able to increase SV after a sudden increase in its afterload: everytime we increase RV afterload, responses are a reduction of RVSV, and increase in Mean RV Pressure. This is particularly true considering pulmonary vascuar resistances. There is a main strategy to decrease the shunt within the lung, which is hypoxemic vasoconstriction. On the other hand, if we apply PEEP or increase the volume of the lung, we are going to increase the RV afterload: whe need to balance this 2 forces for RV.




## BALANCE

| | |
|---|---|
| Fluid Balance | Respiratory Compliance |
| RV function | MV: PEEP/Driving P applied |
| Venous return | Oxygenation |




modified from Ann Transl Med 2018;6(18):353

## HYPOVOLEMIA ?

★ Most COVID-19 patients do not show signs of shock at the very beginning of presentation of the disease, but are frequently hypovolemic at admission to ED/ICU, due to prolonged fever period at home, tachypnea and GI symptoms (ie diarrhea). Effects of MV are higher in hypovolemic patients (RV preload decreases because the increase in pleural pressure induces a compression of SVC, and an increase in intramural right atrial pressure; RV afterload increases: West zones I/II increase because pulmonary capillaries are compressed - Michard, Anesthesiology 2005). Before applying high level of PEEP we should try to fix the balance between RV pre/afterload, and trying to correct hypovolemia.
Fluid needed? if you need to administer fluids, it could be better to use small amount of fluids administered in a short time ie
mini fluid challenge = 100 ml over 1 minute: SAP (also non invasive) increase > 10% = possibility that patient will respond to fluid administration is high = high sensitivity (but low specificity) to detect SV changes
fluid challenge = 4 ml/Kg over 10 minutes
Rate of administration is important: the lower the rate of administration, the lower the response in terms of pressure variables (implying that with faster rates of infusion, we could also try to use non invasive parameters to assess the response to fluid administration.

## START FROM CLINICAL EXAMINATION

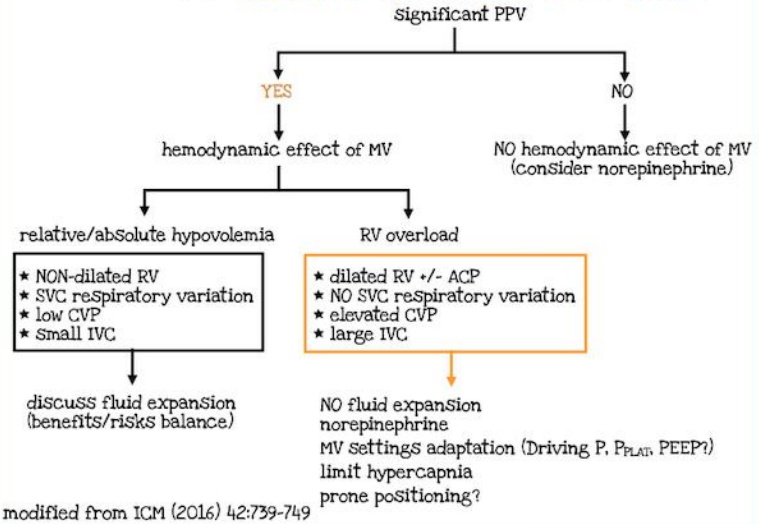
★ Hemodynamic management should start from clinical examination! At the bedside, is advisable to use simple markers of tissue dysoxia: easy to obtain, reproducible, quite reliable (Cecconi M et al. Minerva Anesthesiologica 2014):
★ capillary refill time
★ lactates
★ gap in $CO_2$ = venous-to-arterial $CO_2$ difference = $PvCO_2$ - $PaCO_2$
★ skin mottling
★ $SvcO_2$ = central venous oxygen saturation

## HEMODYNAMIC MONITORING

if these signs are present, & it's probable that CO manipulation may improve tissue perfusion consider
★ hemodynamic monitoring as tool to allow to define best cardiac output for the selected patient; hemodynamic monitoring should be considered as a second step after careful basic assessment of the eventual presence of clinical signs of tissue dysoxia.

★ Add bedside echocardiography to basic hemodynamic monitoring to assess right ventricular function too more complex hemodynamic monitoring could be used... but just for selected patients! Pulse Pressure Variation could be used (together with echo) to assess right ventricular function in ARDS patients; high PPV, indicating that SV markedly changes during swings of positive pressure, may suggest that the patient is fluid responsive. But PPV could be NOT reliable to assess fluid responsivness in ARDS due to low $V_T$, low lung compliance, and due to impact breathing activity. Nevertheless if PPV is high (>12 to 13 %), with no signs of relative/absolute hypovolemia, this could be related to the distension of the RV: PPV may be used at the bedside to quickly assess impact on RV of changes in its afterload: ie if raises up only changing MV settings, probably we are going to affect RV function.
Lung parenchyma involvement in COVID-19 patients is not necessarily related to the compliance of the system: setting PEEP in this population could be really challenging, particularly at the beginning of the disease, and we should be careful in applying MV settings without monitor impact on RV.
Do not forget role of functional hemodynamic tests (challenges to CV systems; is patient responder or not? ie end-expiratory occlusion test - hold respiration for 15-30" looking at the SV - passive leg raising, and mini-fluid challenge are validated in ARDS/low compliance states), and echocardiography to assess right ventricular function (also consider high risk for pulmonary embolism) and IVC collapsibility (quick but limited value).
★ Pay also attention to hospital acquired infections (and consequent septic shock)

## PPV BASED MANAGEMENT OF HEMODYNAMIC FAILURE




modified from ICM (2016) 42:739-749

## CLINICAL EXAMINATION

easy/simple/reliable at the bedside also for NOT (or NOT experienced) intensivists/ICU nurses & NOT ICU settings during the surge
skin mottling, venous saturation, refill time...

OPTIMIZE [PEEP not before VOLEMIA]

ECHO ie RV dilation/TAPSE
HEMODYNAMIC MONITORING

RV FUNCTION
PPV - EVLWI
to support conservative fluid balance

NEW ONSET HYPOTENSION ??
promptly address!

CO
SVRI - ARTERIAL TONE ← HEMODYNAMIC MONITORING → vs DILATED RV / NORMAL

exclude/confirm septic shock related to secondary infections (long ICU stay/MV

exclude/confirm pulmonary embolism