CRITCARE BITES

ACUTE RIGHT VENTRICULAR FAILURE

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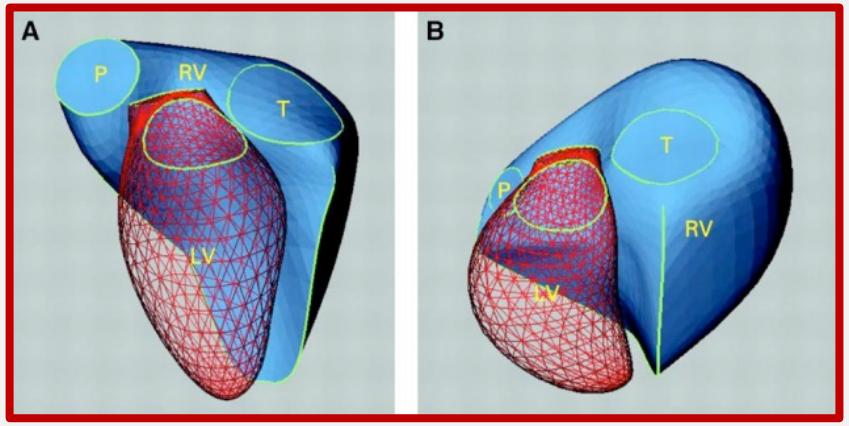
MAD FOR MEDICINE

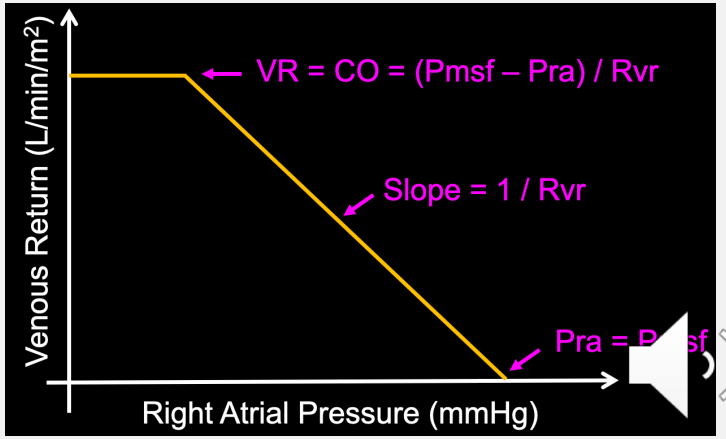
OVERVIEW

- Right ventricle anatomy and physiology
- Causes of acute right ventricular failure
- Pathophysiology
- Assessment
- Management
- Monitoring

RIGHT VENTRICLE ANATOMY AND PHYSIOLOGY

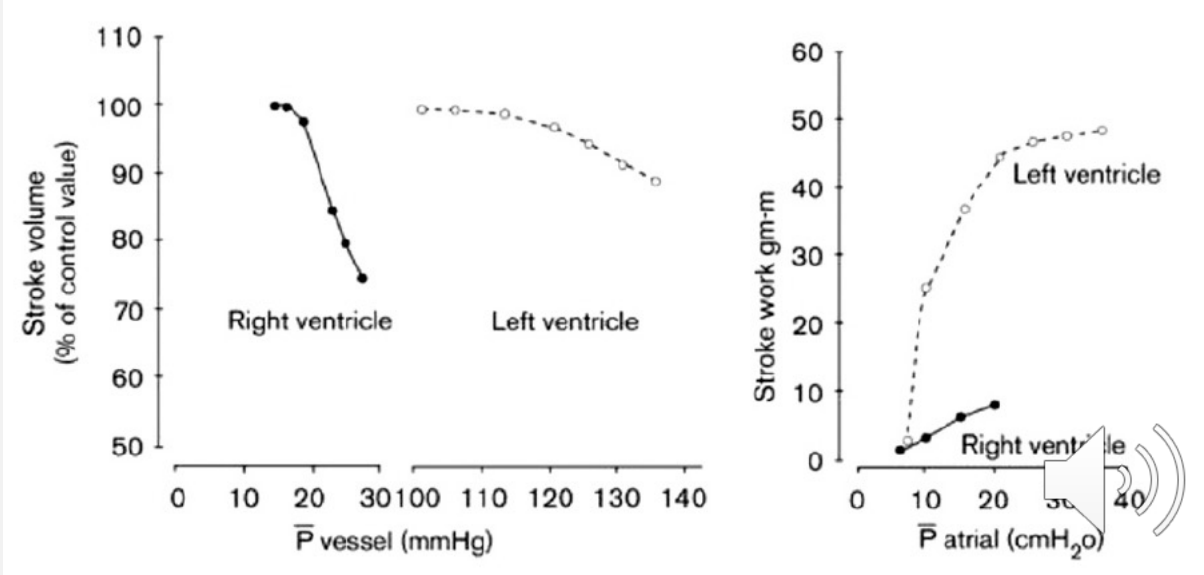
- RV has a unique **crescent shape** that **adheres to and shares the interventricular septum with the LV.**
- The RV free wall is comprised of a superficial transversely oriented muscle layer and deep longitudinally oriented layer from apex to base
- **RV contraction occurs in 3 phases:** Contraction of papillary muscles → Movement of RV free wall towards IV septum → Contraction of LV causing a wringing motion (LV contributes 20-40% of RV systolic function); this results in a peristaltic motion, and movement is predominantly in the **longitudinal direction** (apex moving towards tricuspid valve)
- The primary function of the RV is to **maintain a low right atrial pressure to facilitate venous return** (and hence cardiac output). This is achieved through a nearly **continuous ejection of blood** (both systole and diastole) from the RV to the lungs through a **low pressure, low resistance and high compliance pulmonary vasculature**.





RIGHT VENTRICLE ANATOMY AND PHYSIOLOGY

- The RV is thin walled and is able to accommodate increase venous return, **but unable to acutely overcome rises in pulmonary vascular resistance**
- Of note also, the RV is perfused both during systole and diastole (unlike the LV which is only perfused in diastole)



CAUSES OF RIGHT VENTRICULAR FAILURE

- Volume overload: Tricuspid regurgitation
- Impaired contractility: Myocardial infarction, myocarditis
- Increased afterload:
  - Decompensation of pre-existing pulmonary hypertension
  - **Pulmonary embolism**
  - **ARDS with positive pressure ventilation**

PATHOPHYSIOLOGY

- Can be incited either by a **increase in preload, reduction in contractility or increase in afterload**
- **Ventricular interdependence** – LV filling is reduced (diastolic dysfunction) because
  - Bulging of interventricular septum into LV because of RV dilation
  - Reduced LV preload as RV cannot pump blood out
- **RV ischemia** because
  - LV fails > Reduced cardiac output and hypotension
  - Increased RV wall tension reduces pressure gradient between systemic blood pressure and RV pressure
- **Functional tricuspid regurgitation**
  - RV dilatation causes functional TR
  - This increases RA pressures and reduces venous return, which exacerbates cardiac output

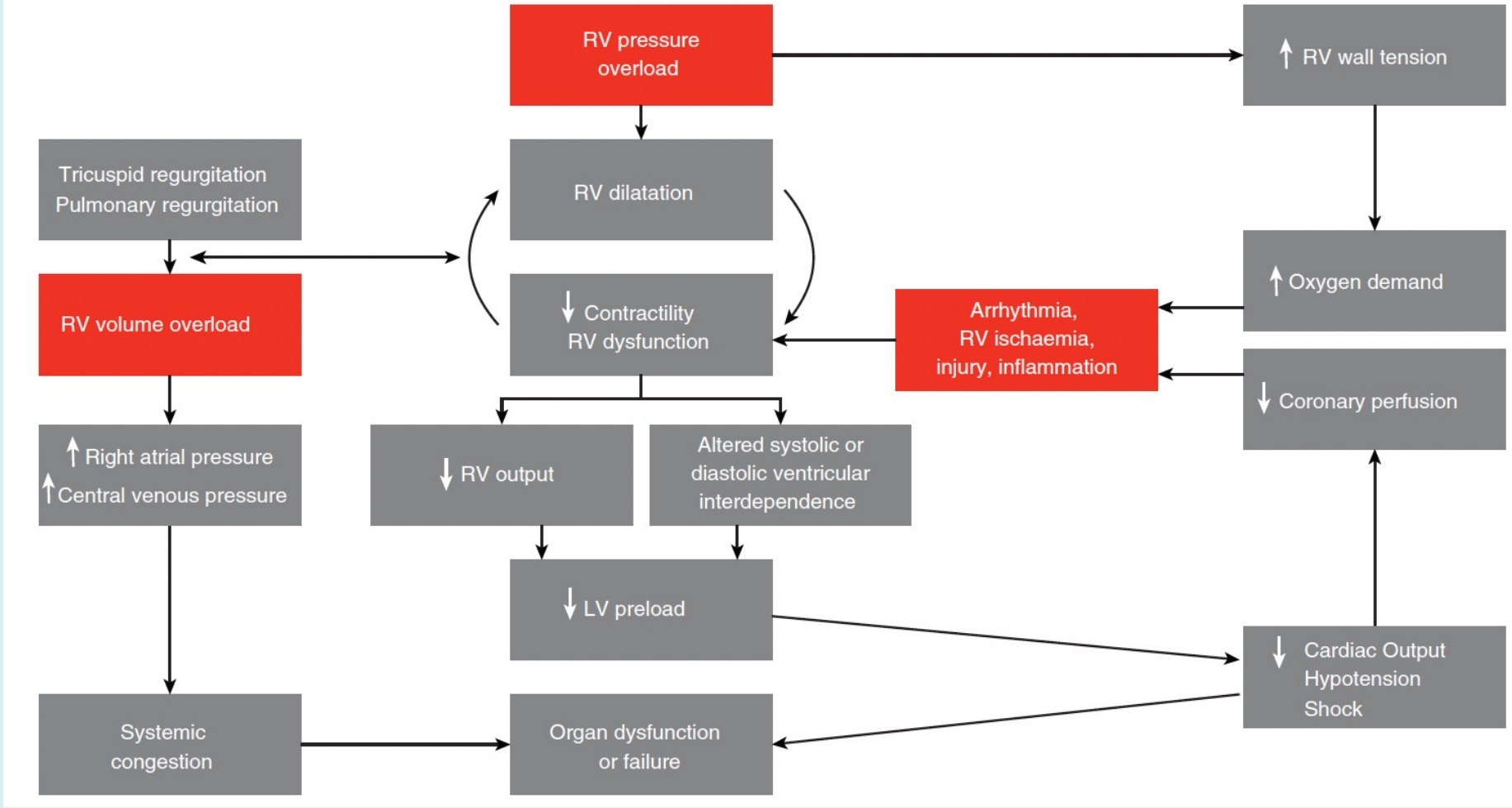


Figure 1 Pathophysiology of acute right ventricular failure. LV, left ventricular; RV, right ventricular.

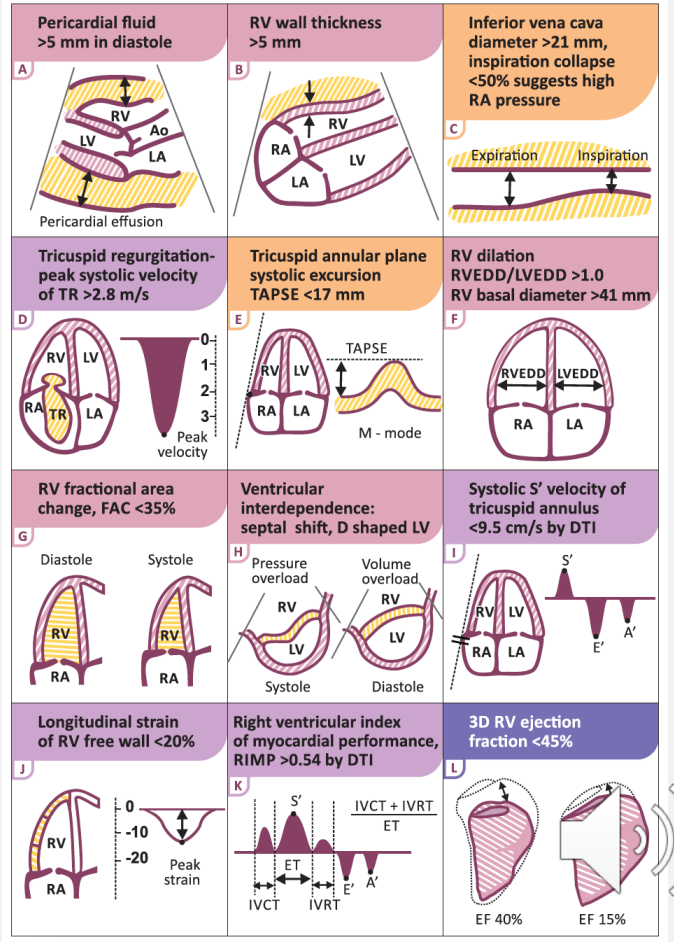
Contemporary management of acute right ventricular failure: a statement from the Heart Failure Association and the Working Group on Pulmonary Circulation and Right Ventricular Function of the European Society of Cardiology

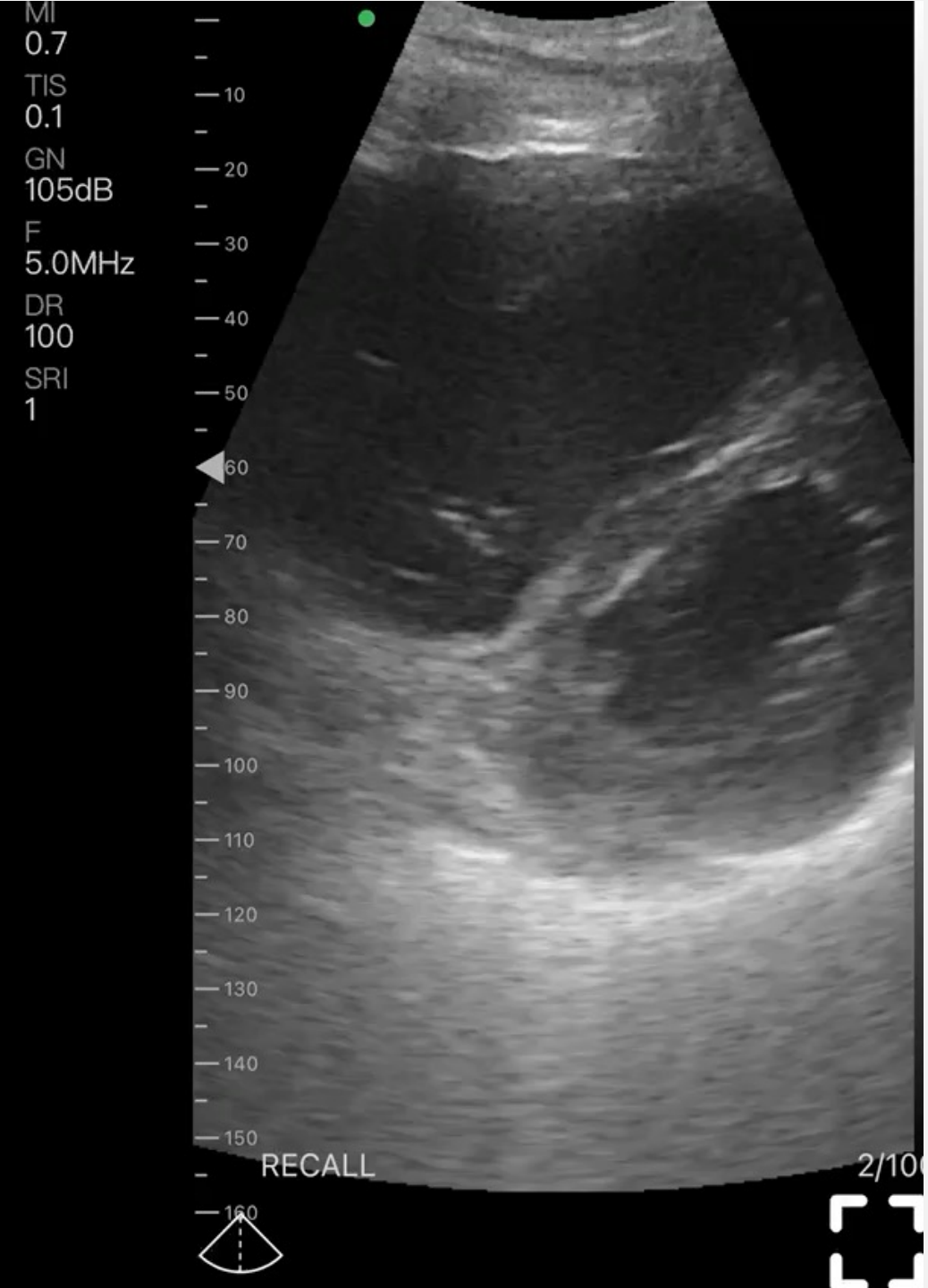
CLINICAL PRESENTATION

- Presentation depends whether acute or chronic
- Congestive symptoms include peripheral edema, abdominal pain/discomfort (hepatic congestion), raised JVP
- Forward failure results in clinical features of shock and organ dysfunction
- Symptoms can also relate to underlying cause – myocardial infarction, pulmonary embolism, ARDS
- High index of suspicion in patients with underlying pulmonary hypertension, and in patients who may appear fluid responsive but deteriorate with fluid therapy

ECHOCARDIOGRAPHY

- RV dilation (RVEDD/LVEDD > 1)
- Septal flattening and paradoxical motion
- TAPSE < 17mm
- RVSP: [4 x (peak TR jet on continuous wave doppler)] + RAP
- RV fractional area change < 35%
- IVC dilation and minimal variability
- McConnell's sign: RV free wall akinesis with apical sparing (not sensitive, but specific for pulmonary embolism)
- RV wall thickness > 5mm suggests chronicity
- 60/60 sign: Coexistence of RVOT acceleration time <60ms and PASP < 60mmHg in the context of RV failure is suggestive of acute (rather than chronic elevation in afterload)

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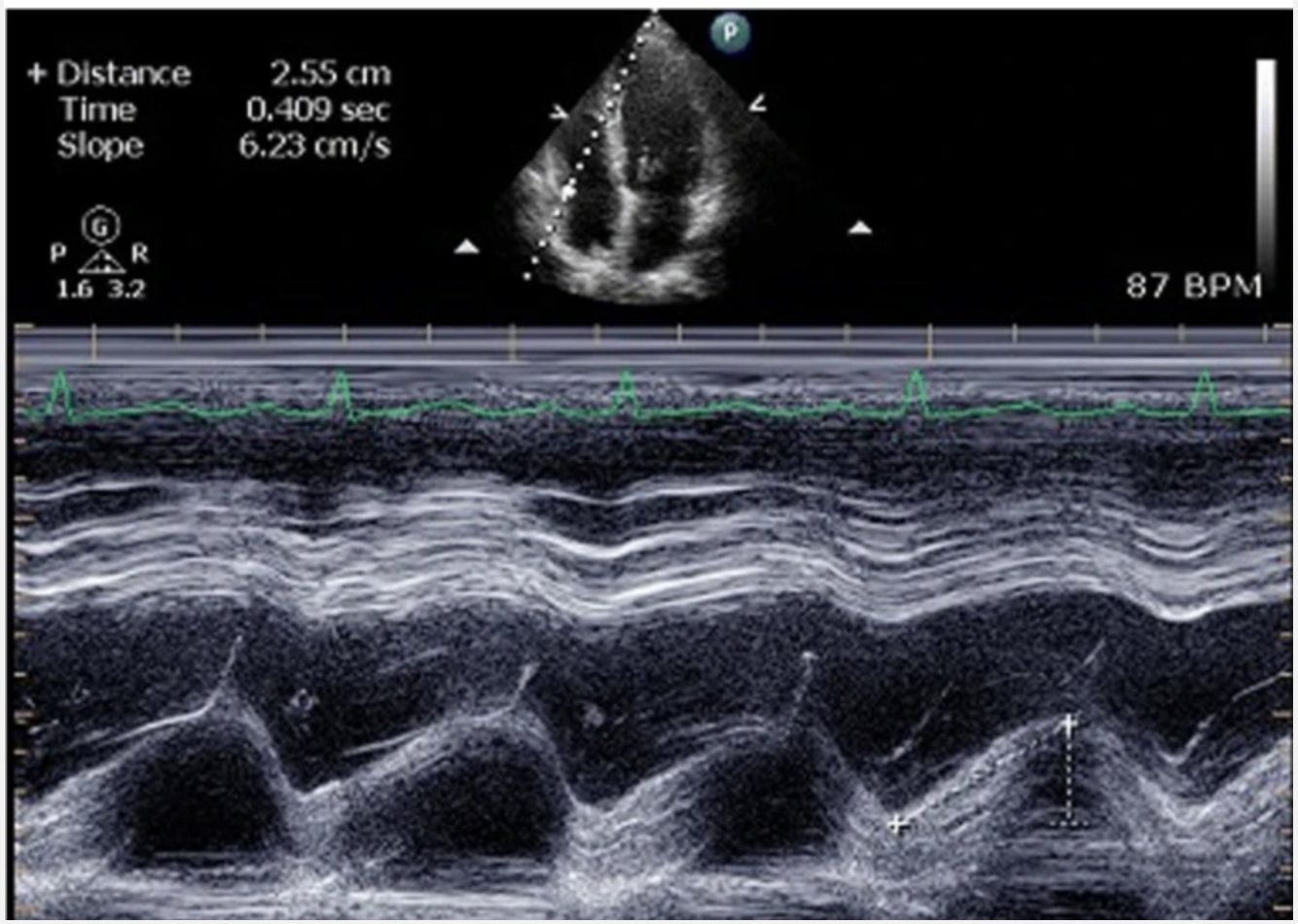
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MANAGEMENT

- Treat underlying etiology
- Avoid hypoxemia, hypercapnia, acidosis
- Optimise ventilatory settings
- Haemodynamic management
  - Vasopressors to maintain MAP
  - Optimise preload
  - Improve RV contractility
  - Reduce RV afterload
- Mechanical circulatory support

VASOPRESSORS

- The role of vasopressors is to maintain coronary perfusion pressure – of note the **RV is perfused both in systole and diastole**, hence an adequate MAP should be targeted
- **Noradrenaline** is usually the initial vasopressor of choice, however it does **increase pulmonary vascular resistance** to some degree
- **Vasopressin** is a suitable alternative as it does not increase pulmonary vascular resistance – but it is less titratable, and does not have any inotropic effect

OPTIMISATION OF PRELOAD

- A normal functioning RV is preload dependent, however it is also very afterload sensitive (especially if there is RV dysfunction)
- As such, the RV requires an adequate amount of preload, however especially in the context of RV dysfunction secondary to high afterload, excessive preload can be particularly deleterious because of resulting ventricular interdependence
- Bedside ultrasound features of septal bowing suggests pressure overload impairing ventricular filling and may indicate need for fluid removal which can be achieve via diuresis or ultrafiltration

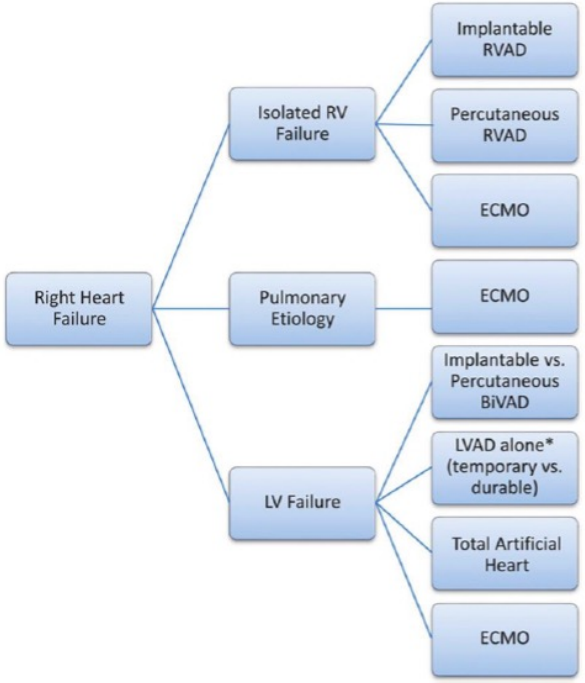
IMPROVE RV CONTRACTILITY: INOTROPES

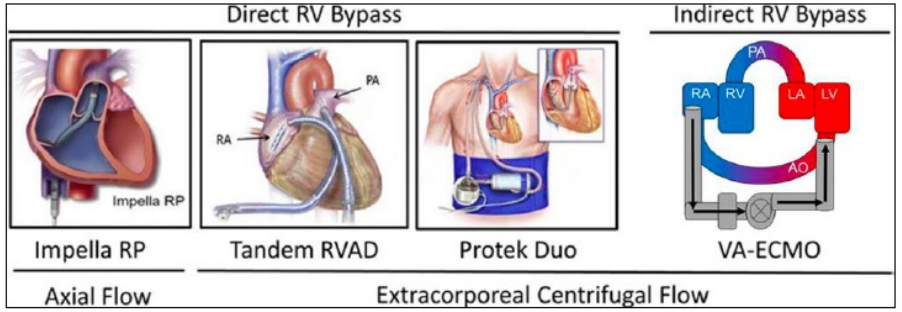
- Options include **milrinone, dobutamine, levosimendan**
- **Milrinone significantly reduces pulmonary vascular resistance**
- Both inodilators can cause hypotension, hence an adequate MAP should be achieved prior to initiation
- Inodilators may also increase myocardial oxygen consumption hence close monitoring is indicated

RV AFTERLOAD REDUCTION

- Inhaled agents like nitric oxide and iloprost reduce RV afterload
- Inhaled agents are preferred to IV agents like GTN and nitroprusside because they **act selectively at well ventilated lung units** without worsening V/Q matching; IV agents cause vasodilation even at poorly ventilated lung units, causing shunting in these areas and dead space in the well ventilated units through a 'steal phenomenon'

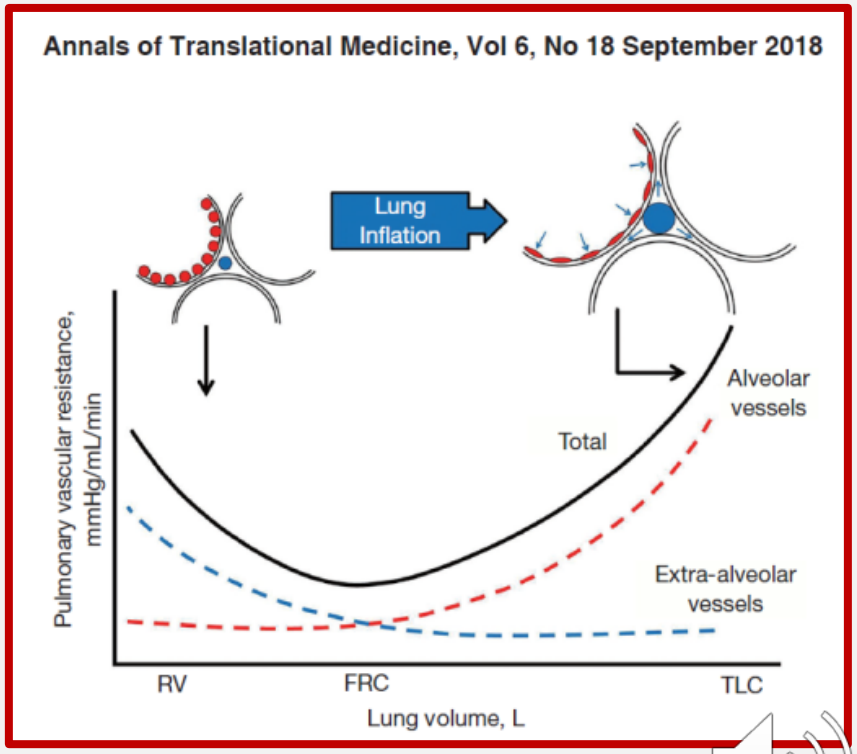
MECHANICAL CIRCULATORY SUPPORT

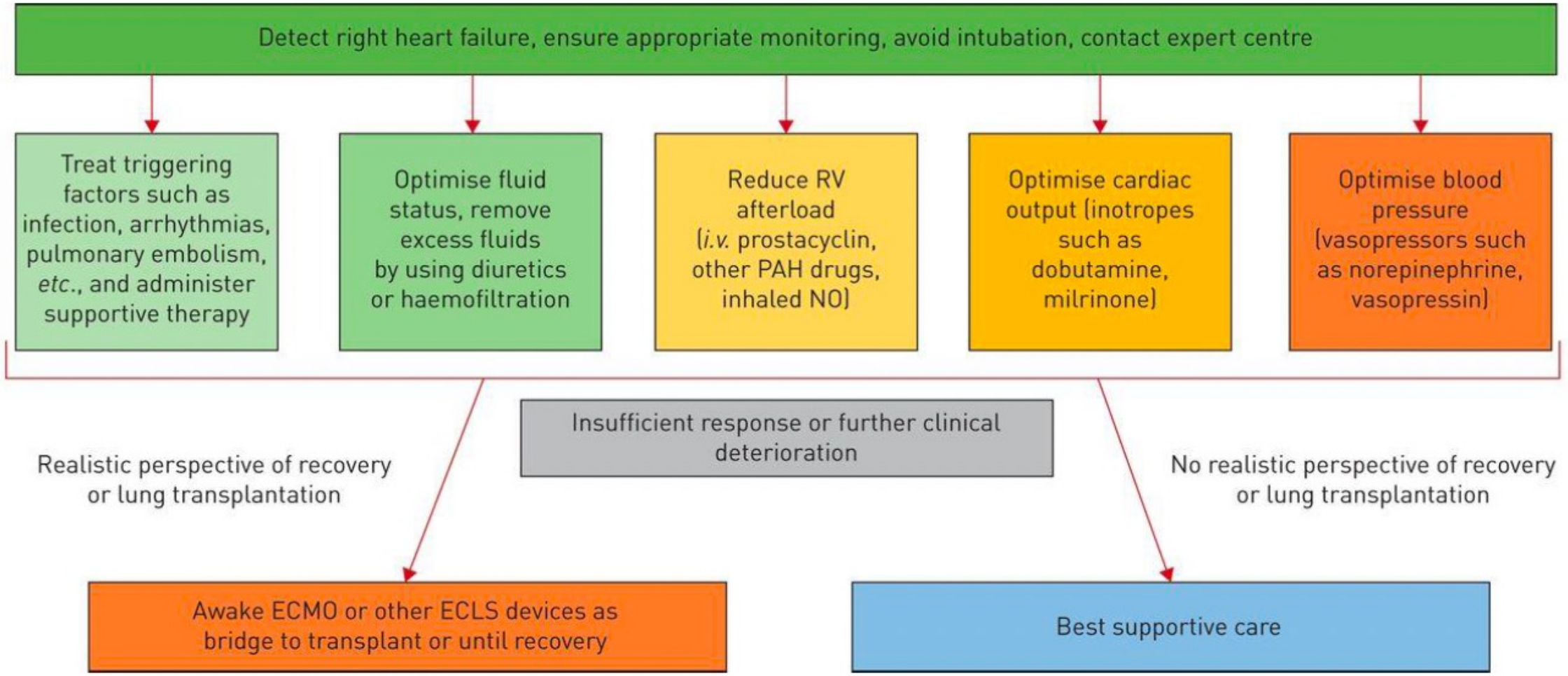




MECHANICAL VENTILATION

- ARDS is a common cause of acute cor pulmonale
- Excessive transpulmonary pressures may result in increased pulmonary vascular resistance due to compression of alveolar vessels
- Generally keep PEEP < 10-12, if higher PEEP is used in the context of concomitant ARDS, consider echocardiographic assessment during titration
- Aim plateau pressure < 27mmHg and driving pressure < 17mmHg
- Avoid hypoxemia, hypercarbia (<60mmHg) and acidosis – permissive hypercapnia should be avoided in RV failure
- Caution with too high respiratory rates that may predispose to autoPEEP and dynamic hyperinflation
- **Consider prone positioning in ARDS**





Intensive care, right ventricular support and lung transplantation in patients with pulmonary hypertension. European Respiratory Journal. 2018.

MONITORING

- **Caution with interpreting PPV/SVV and IVC variability as markers of fluid responsiveness**
- **Repeated echocardiographic assessment**
- **ScvO2, CO2 gap, lactate**
- **CVP** can be used to guide fluid removal
- **Pulmonary artery catheter** (gold standard)
  - Right sided indices: RVEF, RVEDVi, PVRI
  - Global indices: SvO2, cardiac index

CONCLUSION

- RV fails when subjected to high afterload → RV spiral of death (ventricular interdependence, impaired LV filling, RV ischemia, functional TR)
- High index of suspicion to diagnose, usually requires echocardiographic assessment
- Certain aspects of management are counter intuitive – hypotension does not mean more fluids, hypoxemia does not mean more PEEP, permissive hypercapnia may not be suitable
- Optimise BP, preload, contractility and afterload. Optimise ventilator.
- Monitoring – Ideally with PA catheter. If no PA catheter, use ultrasound, CVP and perfusion markers (lactate, ScvO2, CO2 gap)