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What's new: the management of acute right ventricular decompensation of chronic pulmonary hypertension

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In healthy individuals, the right ventricular-pulmonary circulation is a low-pressure, high-compliance system. In high afterload states, the right ventricle (RV) adapts by increasing contractility (homeometric autoregulation) and preload (heterometric autoregulation) [1, 2]. Pulmonary hypertension (PH), defined as a mean pulmonary artery pressure >25 mmHg, results from any physiologic process that increases RV afterload, most commonly left ventricular (LV) disease resulting in a high left atrial pressure and post-capillary pulmonary venous congestion [3]. Pre-capillary PH (PAH) results from high RV loading despite normal pulmonary venous pressure. PAH may present with RV dysfunction, a physiologic state where the RV ventriculo-vascular unit is unable to perform some necessary functions. However, many patients with PAH present to the intensive care unit (ICU) in overt RV failure. In this condition, regardless of etiology, the RV is unable to meet increased loading demands leading to RV dilation, tricuspid regurgitation, and increased right atrial pressure reducing forward flow, coronary perfusion, and perhaps systemic hypotension [4]. This presents unique problems for hemodynamic optimization, intubation, and ventilator management. This paper offers strategies to address the complicated physiology of PH with RV failure in the ICU. See Fig. 1 for a summary of our recommendations.

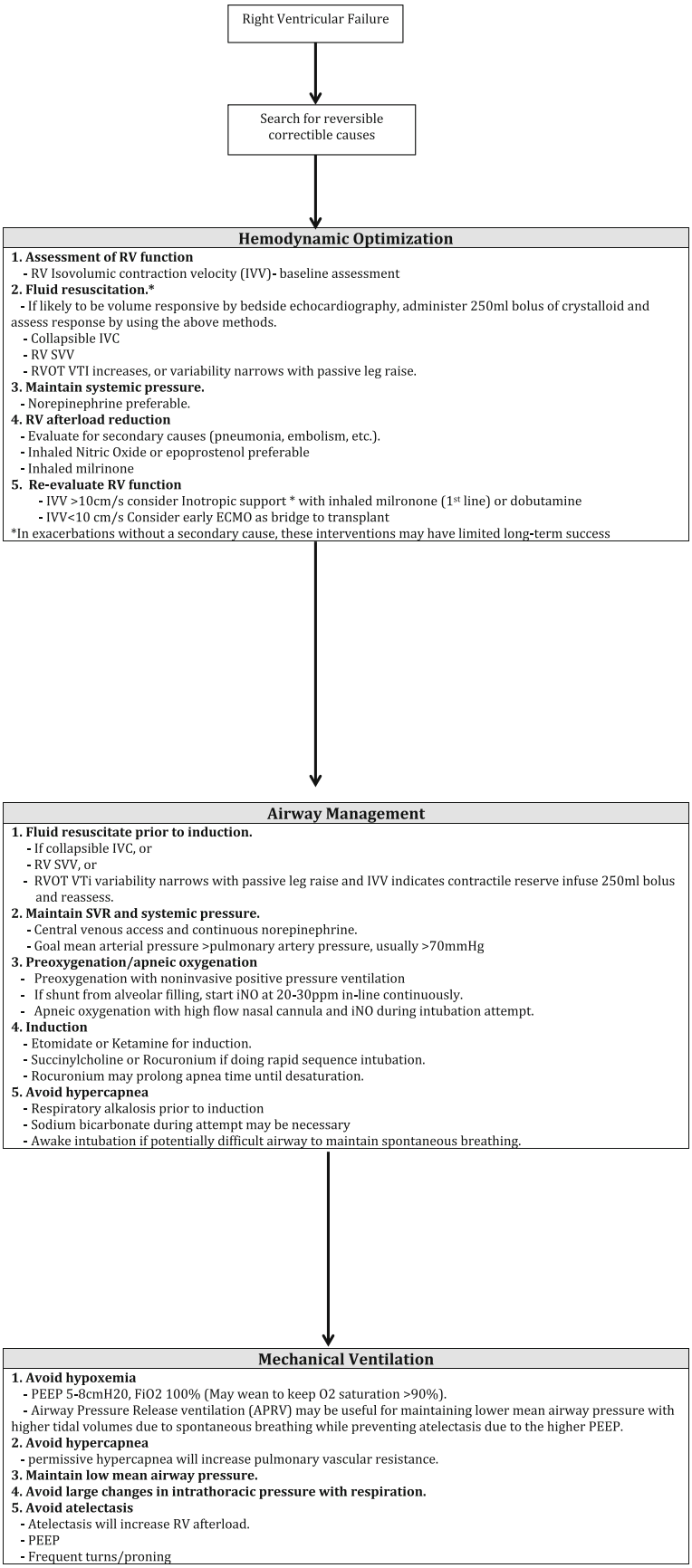
Hemodynamic optimization

Fluid management

In most patients presenting with decompensated RV function, regulatory mechanisms have reached their maximum, rendering interventions that augment contractility or volume loading of limited utility. Volume overloading an already failing RV not only adds to diastolic wall tension and worsens LV filling directly, but it also leads to acute LV diastolic dysfunction and further reduction in stroke volume [5]. Consequently, euvolemia is critically important, yet assessment of volume responsiveness is quite challenging as traditional methods are unreliable. If the patient shows signs of hepatic or renal congestion, or severe RV dilation on initial echo assessment, fluid loading is contraindicated.

Volume responsiveness is often indicated by high stroke volume variation or Doppler velocity–time integral (VTI); however, in the failing RV, the LV will usually be preload-responsive. Some patients present with RV volume overload and organ congestion, despite a preload-responsive LV, and may require diuresis rather than fluid resuscitation. Data suggest that VTI or stroke volume variation alone may indicate RV dysfunction [6]. If VTI is used as an index of responsiveness, it may be done very cautiously at the RV outflow tract and determined before and after a small fluid challenge [6]. We recommend 250 ml or a passive leg raise. In intubated patients, RV outflow variability can be induced by the mechanical ventilator exclusive of volume responsiveness.

Fig. 1 Summary of challenges and recommendations



Assessment of RV function

Numerous quantitative measurements of RV contractile function have been proposed. Pre-ejection phase measurements may be superior to more load-dependent measurements such as tricuspid annular plane systolic excursion (TAPSE) in the measurement of contractility, adaptation, and perhaps clinical outcome [7, 8]. Our experience is that isovolumic contraction velocity (IVV) >10 cm/s obtained using tissue Doppler of the RV lateral wall can predict contractile reserve. In this setting, we are more likely to conduct fluid challenge and/or incorporate inotropic therapy early versus hasty referral for extracorporeal membrane oxygenation (ECMO).

Afterload reduction and hemodynamic support

Reversing correctible causes of increased RV loading and afterload reduction are the mainstays of therapy [9]. This often presents a problem, as many pulmonary vasodilators have systemic effects and systemic hypotension should be avoided. Accordingly, agents such as inhaled nitric oxide (iNO), inhaled epoprostenol, or inhaled milrinone are often the preferred pulmonary vasoactive agents due to their limited effect on the systemic circulation [10, 11]. Systemic hypotension due to a drop in systemic vascular resistance (SVR) can lead to a detrimental drop in RV coronary perfusion [12]. Strict vasoconstrictors (i.e. phenylephrine or vasopressin) should be avoided in favor of norepinephrine, which has been shown to maintain stroke volume with little change in pulmonary afterload [13]. As RV stroke work in PH patients is often exceedingly high, inotropic agents serve little role other than "bridging" until afterload reduction, ECMO, or transplant occur [14, 15].

Intubation and mechanical ventilation

Recently, it was shown that the need for mechanical ventilation was the strongest unadjusted risk factor for mortality in patients with PAH admitted to the ICU [16]. Intrathoracic pressure changes with respiration have an exaggerated effect on hemodynamics in the patient with PH, worsening cardiopulmonary interactions, and making intubation extremely risky. Increased intrathoracic pressure with positive pressure ventilation decreases venous return, especially in the hypovolemic patient. Combined with increased RV afterload from positive pressure ventilation, RV performance can worsen with transition to mechanical ventilation, worsening shunt and hypoxemia, and precipitating cardiovascular collapse [3]. When possible, work of breathing and gas exchange should be supported with non-invasive positive pressure ventilation (NIPPV) and positive end-expiratory pressure (PEEP).

Careful preparation prior to intubation is critical. Hypoxemia and hypercapnea increase pulmonary artery vasoconstriction and RV afterload [17, 18]. Preoxygenation can be difficult due to shunt physiology from intracardiac lesions and/or VQ mismatch, and should be performed with the goal of hyperoxia using NIPPV [19]. During intubation, apneic oxygenation with a high-flow nasal cannula should be performed [20]. Pulmonary vasodilators (iNO) can augment oxygenation at low concentrations (20–30 ppm), and can be delivered in-line continuously during the intubation process. The concentration can be increased afterwards for maximal pulmonary artery vasodilation.

During preoxygenation, respiratory alkalosis should be attempted to avoid apnea-induced hypercapnea. Premedication with fentanyl may be considered to blunt the hypertensive response to laryngoscopy. In low SVR states, norepinephrine should be started prior to induction with a mean arterial pressure goal > pulmonary artery pressure. Hemodynamically neutral sedatives such as etomidate should be used for induction. Neuromuscular blocking agents should be considered as they may improve intubating conditions and first-attempt success rate. When choosing post-intubation sedation and analgesia, the goal should be to maintain normotension and SVR.

The goals of mechanical ventilation are: (1) avoid hypoxemia and hypercapnea, (2) maintain a low mean airway pressure, and (3) avoid alveolar collapse, all of which increase RV afterload [17, 18, 21, 22]. A cycle of RV failure occurs when any of the above changes result in an increased RV afterload and decreased cardiac output, resulting in further hypoxia and under-filling of the LV. Hypotension ensues from the decreased cardiac output, further compromising the RV. PEEP and spontaneous breathing can obviate some of these difficulties. Prone positioning may relieve RV afterload by improving oxygenation, especially in patients with RV preload reserve [23]. ECMO can be used until definitive RV reduction or transplant is achieved, as patients can be kept awake, extubated, and spontaneously breathing after cannulation [14, 15].

Summary

In decompensated PH, regardless of etiology, the treatment of choice is afterload reduction through modification of secondary causes and pulmonary vasoactive therapy. When faced with intubation, attention to hemodynamic optimization, preoxygenation, and induction is critically important. It is important to continue all PH-targeted therapy the patient is taking for pulmonary hypertension

prior to intubation, including parenteral and subcutaneous medications. Ultimately, rescue therapies (ECMO, right-ventricular assist device) may be required as a bridge to transplant in the suitable patient.

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