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Right heart failure: A narrative review for emergency clinicians

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ABSTRACT

Introduction: Right heart failure (RHF) is a clinical syndrome with impaired right ventricular cardiac output due to a variety of etiologies including ischemia, elevated pulmonary arterial pressure, or volume overload. Emergency department (ED) patients with an acute RHF exacerbation can be diagnostically and therapeutically challenging to manage.
Objective: This narrative review describes the pathophysiology of right ventricular dysfunction and pulmonary hypertension, the methods to diagnose RHF in the ED, and management strategies.
Discussion: Right ventricular contraction normally occurs against a low pressure, highly compliant pulmonary vascular system. This physiology makes the right ventricle susceptible to acute changes in afterload, which can lead to RHF. Patients with acute RHF may present with an acute illness and have underlying chronic pulmonary hypertension due to left ventricular failure, pulmonary arterial hypertension, chronic lung conditions, thromboemboli, or idiopathic conditions. Patients can present with a variety of symptoms resulting from systemic edema and hemodynamic compromise. Evaluation with electrocardiogram, laboratory analysis, and imaging is necessary to evaluate cardiac function and end organ injury. Management focuses on treating the underlying condition, optimizing oxygenation and ventilation, treating arrhythmias, and understanding the patient's hemodynamics with bedside ultrasound. As RHF patients are preload dependent they may require fluid resuscitation or diuresis. Hypotension should be rapidly addressed with vasopressors. Cardiac contractility can be augmented with inotropes. Efforts should be made to support oxygenation while trying to avoid intubation if possible.
Conclusions: Emergency clinician understanding of this condition is important to diagnose and treat this life-threatening cardiopulmonary disorder.

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1. Introduction

Right heart failure (RHF) is defined as impaired right ventricular (RV) contractility due to myocardial dysfunction, elevated pressures, or volume overload [1]. The true prevalence of RHF is challenging to estimate and depends on the definition of RHF in the individual study and the imaging study used to determine right ventricular function. As opposed to left-sided heart failure (LHF) in which patients are categorized in distinct classes (such as the New York Heart Association Classification) [2], RHF is not currently divided into stages. Nevertheless, there are over 950,000 emergency department (ED) visits in the United States per year for an acute heart failure exacerbation, with 80% of heart failure exacerbations admitted from the ED setting [3]. Current literature suggests RHF is observed in 3–9% of all acute heart failure admissions [4-7]. Using these data, the number of patients with RHF admitted from the ED in the United States annually ranges between 22,800 to 68,400. In a study of the European CHARITEM registry, RHF was present in 2.2% of all patients with acute decompensated heart failure [8]. Additionally, RHF is estimated to account for at least one fifth of patients who have LHF with preserved ejection fraction [8,9].

While RHF is more rare compared to LHF, the presence of RHF contributes to patient morbidity and mortality. Overall mortality from RHF is estimated to be between 6% to 14% [4,5]. RHF can be a result of many underlying conditions including pulmonary hypertension (PH), RV infarction, valvular pathology, cardiomyopathy, thromboembolism, congenital conditions, and chronic pulmonary conditions [8]. Compared to patients with normal right ventricular function, the presence of right heart dysfunction in a patient who also has LHF with reduced ejection fraction increases mortality 2.4 fold [10]. In patients with acute inferior ST elevation myocardial infarction (STEMI), RV dysfunction occurs in 30–50% of cases [11]. The SHOCK trial found that only 5% of the 933

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patients with myocardial infarction induced cardiogenic shock had shock due to RV failure [12]. However, inhospital mortality for this group was over 50%, similar to patients with cardiogenic shock due to left ventricular failure [12]. In patients diagnosed with acute pulmonary embolism (PE), the presence of RV dysfunction on echocardiogram is estimated to increase mortality 2.4 to 3.5 fold [13].

The presentation of RHF can overlap with LHF, or it may be concomitant with other acute pathologies. Thus, the diagnosis of an acute RHF exacerbation can be challenging in the ED, but as discussed, decompensated RHF significantly increases morbidity and mortality. It is therefore important that ED clinicians can diagnose and appropriately treat patients with RHF, as their presentation is complex and may vary based on the underlying pathology.

2. Methods

A literature review of PubMed and Google Scholar databases was performed for articles up to January 2022, using the keywords 'right heart failure' OR 'pulmonary hypertension' for this narrative review. The authors included retrospective and prospective studies, systematic reviews and meta-analyses, and other narrative reviews. Guidelines and international/national organization websites were also included. The literature search was restricted to studies published or translated into English. Authors reviewed all relevant articles and decided which studies to include for the review by consensus, with focus on emergency medicine-relevant articles, including guidelines. A total of 82 resources were selected for inclusion in this review.

3. Discussion

3.1. RV physiology

Due to the variety of causes of PH and RHF, patient presentations can vary and are typically dependent on the underlying cause and the presence of a sudden change in right heart hemodynamics resulting in acute or chronic RHF. Acute RHF commonly occurs due to an abrupt increase in RV afterload or decrease in myocardial function, rather than acute changes in volume status [1]. Understanding the unique physiology of the RV can help determine the appropriate treatment for patients with RHF.

As opposed to the left ventricle (LV) which contracts in a concentric twisting motion, 75% of RV contraction occurs via a longitudinal contraction with a bellows-like inward movement mechanism of the RV free wall [14,15]. In normal physiology, the pulmonary vasculature is a highly compliant, low resistance system, so generating RV output requires approximately one fifth the amount of energy needed to generate LV output [16]. LV ejection fraction may augment 20 to 40% of the RV ejection fraction [17,18]. Additionally, the RV ejection fraction is inversely proportional to pulmonary artery pressure [19]. This results in an RV systolic function that is highly sensitive to changes in afterload, but more accommodating to changes in preload [17]. Fig. 1 compares the physiology of the RV to the LV.

The response of the RV to elevated pulmonary vascular resistance (PVR) can be both adaptive and maladaptive [20]. Adaptive RV changes include cardiomyocyte hypertrophy and remodeling similar to patients with compensated LHF, resulting in preserved systolic and diastolic function [21]. Patients with adaptive changes may eventually develop chronic RHF and have examination findings consistent with heart failure such as jugular venous distension (JVD), abdominal distention, and leg edema but clinically are not typically in extremis [21]. Maladaptive changes include RV dilatation leading to tricuspid annular dilation and tricuspid regurgitation [22]. The clinician may note a systolic murmur [21]. Elevated right atrial pressure is a concerning sign for decompensated RHF and associated with higher all-cause mortality [23]. Additionally, maladaptive structural changes to the right heart can increase the risk of dysrhythmias [21,24].

Right Ventricular Physiology	Left Ventricular Physiology
Longitudinal squeeze	Concentric, twisting squeeze
20-40% cardiac output augmented by LV	No augmentation of cardiac output by RV
Thin walled myocytes with more collagen content	Thickened, muscular, myocytes
EF inversely proportional to PVR	EF less sensitive to SVR
Squeezes against highly compliant system	Squeezes against low compliance system

Fig. 1. Left and Right Ventricle Physiology. RV = right ventricle, LV = left ventricle, EF = ejection fraction, PVR = pulmonary vascular resistance, SVR = systemic vascular resistance.

The thin wall of the RV also makes it more susceptible to hypotension and more dependent on adequate coronary perfusion pressure compared to the LV [1]. In 80% of the population the right coronary artery (RCA) and its branches perfuse the RV free wall and the inferior portion of interventricular septum [25,26]. In normal physiology, the RV is perfused by the RCA during both diastole and systole [27]. However, in patients with right heart changes due to PH, RCA perfusion occurs mostly during diastole [27]. Therefore, RV perfusion decreases in proportion to elevations in RV pressure, and if RV pressure becomes greater than the systemic blood pressure, RV ischemia ensues, thus worsening RHF [27,28].

In the acute phase of RHF, the RV is preload dependent [1,25-27]. The RV's response to acutely increased preload or increased afterload is similar to the LV in that ventricular dilatation occurs initially to preserve stroke volume [1]. However, RV over-distension acutely and RV hypertrophy over time decrease RCA perfusion leading to RV ischemia [27]. Additionally, over-distension of the RV can lead to bowing of the interventricular septum into the LV, limiting LV filling volume and reducing cardiac output, leading to hypotension and poor systemic perfusion [17,18]. This process is termed ventricular interdependence [18]. The combination of both the RV bulging into the LV and decreased contractility from RV ischemia contributes to cardiogenic shock in a patient with acute RHF [28]. The physiologic changes occurring in RHF are summarized in Fig. 2.

3.2. Pulmonary hypertension

One of the common pathologic causes of chronic RHF is increased RV strain over time due to PH [20,29]. Normal mean pulmonary artery pressure is 15 mmHg [30]. PH has been classically defined as mean pulmonary artery pressure greater than 25 mmHg as measured by right heart catheterization (though some refinements to this definition have been made more recently but are not pertinent to ED management) [30]. The gold standard of RV dynamic measurements such as RV ejection fraction and stroke volume is cardiac magnetic resonance imaging (MRI); however, echocardiogram may also be utilized [31]. An RV pressure greater than 35 mmHg on echocardiogram strongly suggests PH [32].

PH is caused by a number of etiologies divided in 5 different subcategories. LHF is the most common cause of PH, as chronically elevated LV pressure leads to increased pressures upstream and pulmonary vascular remodeling [30,33]. This is classified as Group 2 PH [30]. Group 3 PH develops from chronic lung conditions such as chronic obstructive pulmonary disease (COPD) or pulmonary fibrosis and is the second most common cause of PH [30,33]. The chronic hypoxic state and respiratory acidosis cause pulmonary vascular changes leading to PH in patients with Group 3 PH [30]. Other groups of PH include sequela of thromboemboli (Group 4) and multifactorial mechanisms (Group 5) [30]. Lastly, pulmonary arterial hypertension (PAH) (previously known as primary pulmonary hypertension) is classified as Group 1

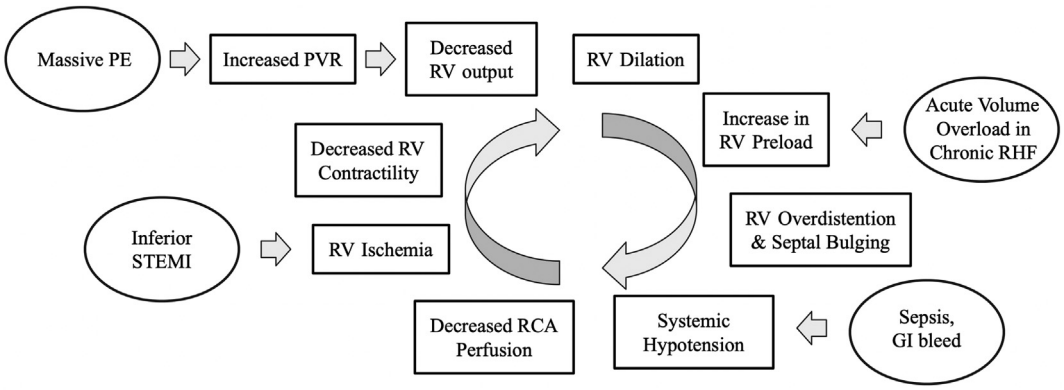

Fig. 2. Cyclic physiology of right heart failure. Ovals represent examples of underlying causes leading to right heart failure. PVR = pulmonary vascular resistance, RV = right ventricle, RHF = Right heart failure, PE = Pulmonary Embolism, STEMI = ST elevation myocardial infarction, RCA = right coronary artery.

PH and is a rarer entity caused by genetic conditions such as connective tissue disorders, toxins, congenital heart disease, human immunodeficiency virus (HIV), schistosomiasis, and idiopathic conditions [30]. RHF is the leading cause of death in patients with PAH [20,34].

Patients with PH often have a delay to diagnosis [33]. Therefore, it is important to consider PH (and subsequent RHF) in patients with unexplained dyspnea and risk factors for PH such as chronic obstructive pulmonary disease (COPD), PE, LHF, or connective tissue disorders [33]. The most common complaint in patients with PH is dyspnea [35]. Depending on the severity of RHF, the patient may also present with chest pain, fatigue, peripheral edema, or lightheadedness [35]. Syncope and pre-syncope in a patient with PH or RHF are concerning and warrant further evaluation for dysrhythmia, valvular pathology, and elevated RV pressures [25].

3.3. ED evaluation and diagnosis

Initial ED evaluation for RHF includes electrocardiogram (ECG), chest radiograph (CXR), and laboratory analysis such as cardiac markers (i.e. troponin, brain natriuretic peptide [BNP]), electrolytes, and renal and hepatic function panels to diagnose the underlying cause as well as determine the severity of RHF [36,37]. Patients with cardiogenic shock will demonstrate signs of end organ damage on laboratory analysis (i.e., acute kidney injury, elevated BNP, elevated liver function tests, etc.) [38,39]. However, these laboratory abnormalities may be present in patients with chronic RHF if symptoms occur gradually [40]. Therefore, trends in laboratory studies may be useful to assess the acuity of injury if prior laboratory results are available [40]. An elevated troponin, increased BNP, or increase in creatinine have been associated with increased morbidity and mortality in patients with RHF [36,39].

Common ECG findings in patients with PH include right axis deviation (70% of patients with PH), right bundle branch block, normal sinus rhythm, and ST depression or T wave inversions in V1 and the inferior leads [41,42]. Chronic RHF can lead to increased right atrial pressure and increases the risk of dysrhythmias, which can acutely worsen heart failure [21,24]. The most common dysrhythmias seen in patients with PH include AV nodal reentrant tachycardia (also known as supraventricular tachycardia [SVT]), atrial fibrillation, and atrial flutter [24]. If the ECG is concerning for STEMI, emergent coronary reperfusion is indicated, which can prevent complications such as cardiogenic shock [43]. In cases where RV failure is due to an acute myocardial infarction, nitrates or diuretics should be avoided, as the decrease in preload can result in patient decompensation [44].

Imaging studies such as CXR and chest computed tomography (CT) may demonstrate secondary signs of PH, which may aid in the diagnosis of RHF. CXR findings in a patient with RHF include RV enlargement, marked by a globular shaped cardiac silhouette with loss of the retrocardiac space on the lateral projection [45,46]. An enlarged right atrium may be present in a patient with chronic RHF and is noted on CXR by rightward displacement of the cardiac silhouette beyond the spine [45,46]. Pulmonary edema and pleural effusions are less common in isolated RHF unless it is combined with LHF [29,32]. Signs of PH on CXR include an enlarged pulmonary artery with distal branches that have a "pruned" appearance, meaning that the arteries dramatically taper towards the periphery of the CXR [45,46]. Retrospective studies have found that a main pulmonary artery diameter greater than 30 mm on chest CT is predictive of PH [47]. One study found a 96% positive predictive value for PH if the diameter of the main pulmonary artery was greater than the proximal ascending aorta on chest CT [47]. A chest CT to evaluate for PE may be an important component of the evaluation in a patient with known PH, as reduced pulmonary blood flow from chronic RHF is a risk factor for developing an acute thromboembolism [25].

Point of care ultrasound (POCUS) is an important tool to aid in the diagnosis and management of patients with RHF. While one goal of POCUS in RHF is to assess RV function, POCUS can also evaluate for mimics of RV failure such as cardiac tamponade, as well as other conditions. POCUS assessments particular to RHF include RV free wall thickness, inferior vena cava (IVC) assessment, tricuspid annular plane of systolic exertion (TAPSE), RV to LV ratio, and interventricular wall flattening [48].

A starting point for assessing RV function using POCUS is the subxiphoid view to measure RV free wall thickness. A free wall less than 5 mm is normal, while a thickened free wall indicates chronic RV failure [49]. The differential for a thickened RV free wall includes right ventricular hypertrophy from chronic PH, LV failure, chronic PE, and cardiomyopathies [49]. Fig. 3 shows a thickened RV wall in a patient with chronic RHF. In an acute obstructive PE, the RV diameter may be dilated but with a thin RV free wall [49,50]. However, RV wall thickening is indicative of chronic changes to the RV muscle as opposed to a sudden acute change where the RV has not had time to remodel and thicken [50]. Echocardiographic findings for a PE such as a dilated RV may vary based on the acuity and size of PE burden [50]. In general, echocardiographic findings for PE have higher specificity compared to sensitivity, but bedside POCUS assessment of right heart function is useful because it may guide PE therapy [50].

The clinician can also assess the IVC in patients with RHF starting with a subxiphoid view, then turning the probe to a sagittal plane to visualize the IVC and right atrium junction. IVC diameter measurement can be obtained via M-mode set between 0.5 cm and 3.0 cm from the IVC / right atrial junction [49]. An IVC diameter greater than 2.1 cm and less than 50% collapse with patient sniff suggests high right atrial pressures [49]. In acute RHF this may indicate excess volume or an acute change in RV afterload, but this measurement must be correlated

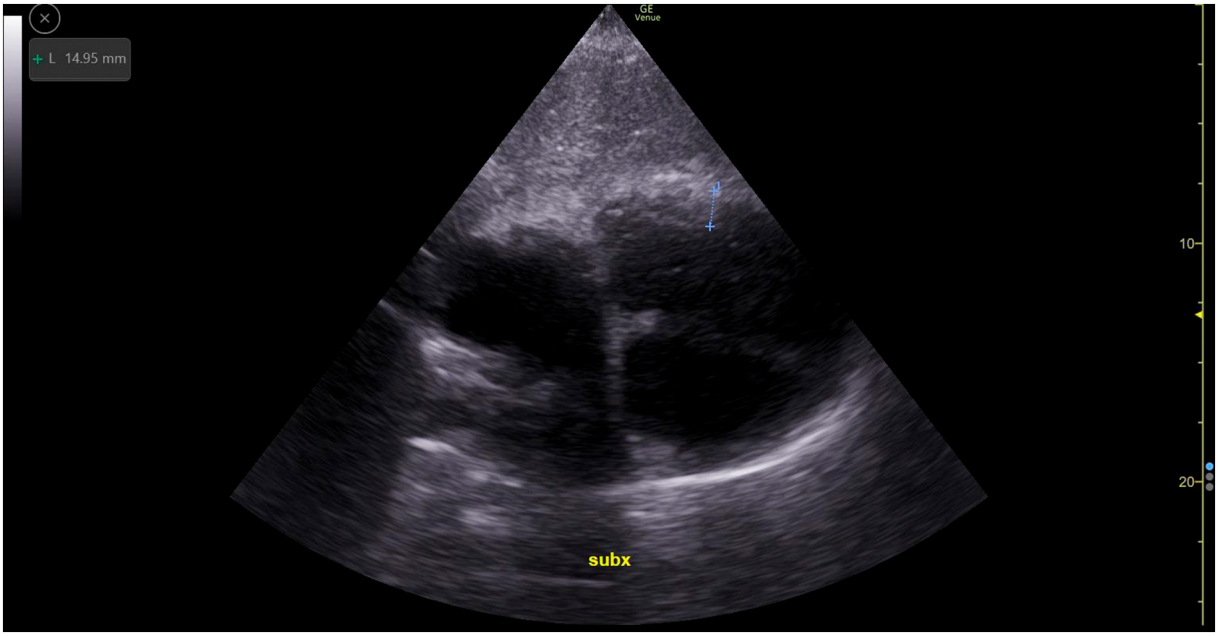

Fig. 3. Thickened RV wall.

with history and physical examination [44]. IVC measurements are more difficult to interpret clinically in a patient with chronic PH and chronically elevated right atrial pressures; however, IVC measurements are useful in making a new diagnosis of PH [48,51]. Video 1 demonstrates a dilated IVC with minimal respiratory variation in a patient who is volume overloaded.

The apical 4 chamber view is also used to assess the overall RV size and ratio of the RV to the LV. A normal RV to LV ratio is 0.6:1.0 (i.e., a normal RV should be about two thirds of the size of the LV) [48,52]. An RV to LV ratio of 1:1 or an RV basal diameter greater than 41 mm is concerning for RV dilation [48,49]. Video 2 demonstrates a dilated RV in a patient with chronic RHF.

TAPSE measurement can assist in assessment of RV systolic function [48,49]. This can be measured by placing the M mode cursor over the lateral portion of the tricuspid annulus in an apical 4 chamber view [49]. Since RV function is dependent on longitudinal contraction, assessing the ascent and descent of the tricuspid annulus from systole to diastole using M mode can evaluate RV function [48,49]. Normal measurement from peak to valley of the tricuspid motion in M-mode is greater than 17 mm [49,53]. Obtaining a TAPSE measurement is relatively easy with an adequate apical 4 chamber view. Fig. 4 demonstrates a normal TAPSE measurement. However, TAPSE cannot differentiate between acute and chronic RV dysfunction [50]. Other limitations include decreased accuracy in patients with regional differences in RV function, and the numerical value can be affected by the angle of the M-mode cursor over the tricuspid annulus [49].

If present, McConnell's sign is indicative of RV strain and decreased function. McConnell's sign is demonstrated in the apical 4 chamber view with a hypokinetic RV free wall at the basal and mid segments, but normal movement of the apical segment [44]. One meta-analysis found McConnell's sign to be 97% specific for the diagnosis of PE [50].

Finally, the parasternal short axis view can assess for interventricular flattening. This is also known as the “D-sign”, as the LV takes on a D shape, rather than the circular LV shape, due to increased RV pressure or volume [49]. In extreme cases septal dyskinesia may be present, especially at the apex of the RV in which the RV bulges into the LV, thus compromising the patient's hemodynamics indicating RV overdistension and excess preload [49]. Video 3 shows a patient with acute on chronic RHF with volume overload and interventricular flattening on the parasternal short axis view.

3.4. Management

One of the primary tenets of diagnosis and management of RHF is to find and treat the underlying cause of the RHF exacerbation. RHF can present acutely with signs of cardiogenic shock or more insidiously [20,25]. Patients with massive PE, chronic COPD who develop an acute respiratory illness, or acute on chronic RHF with systemic venous congestion as a result of longstanding increased RV afterload can all present in RHF [20]. If the patient has a PE causing right heart strain and hemodynamic compromise, treatment with thrombolytics or a surgical embolectomy is indicated [54]. If acute hypoxia from a COPD exacerbation is improved with supplemental oxygen and inhaled beta agonists and anticholinergics, RV hemodynamics should also improve [32,44].

While searching for and treating the underlying etiology, hemodynamics must be optimized, including preload, afterload, and contractility [20,32]. Patients with an acute dysrhythmia such as atrial fibrillation benefit from rapid electrical cardioversion, as loss of atrial kick can further impair RV cardiac output [24,55]. Rate control of dysrhythmias with beta antagonists or calcium channel blockers is not recommended in RHF, as these medications will worsen cardiac output in this setting [55].

A failing RV is preload dependent, and an accurate assessment of volume status is necessary [20,44]. Systemic hypotension does not necessarily indicate that the patient is intravascularly depleted. Certain underlying conditions, such as sepsis or volume loss, could be the cause of hypotension and worsening RV dynamics due to low preload [55]. However, if excess preload is present, RV dilatation ensues, which worsens RV ischemia from increased wall tension, may cause tricuspid regurgitation, and even lead to interventricular bowing and cardiogenic shock [18,22]. History, examination, and POCUS are important initially and on reassessment to assess RV hemodynamics [44]. If the patient appears intravascularly depleted, small boluses of isotonic fluids (250 ml boluses) and frequent patient reassessment are recommended [32,44]. If POCUS findings are notable for RV overdistention and interventricular bowing, diuretics are indicated despite the patient's hypotension [20,56]. If the patient does not respond to the initial treatment, higher doses of diuretics may be beneficial [57]. However, a patient may also be diuretic resistant, so close monitoring of urine output is needed, as renal replacement therapy may ultimately be needed to remove volume [58,59].

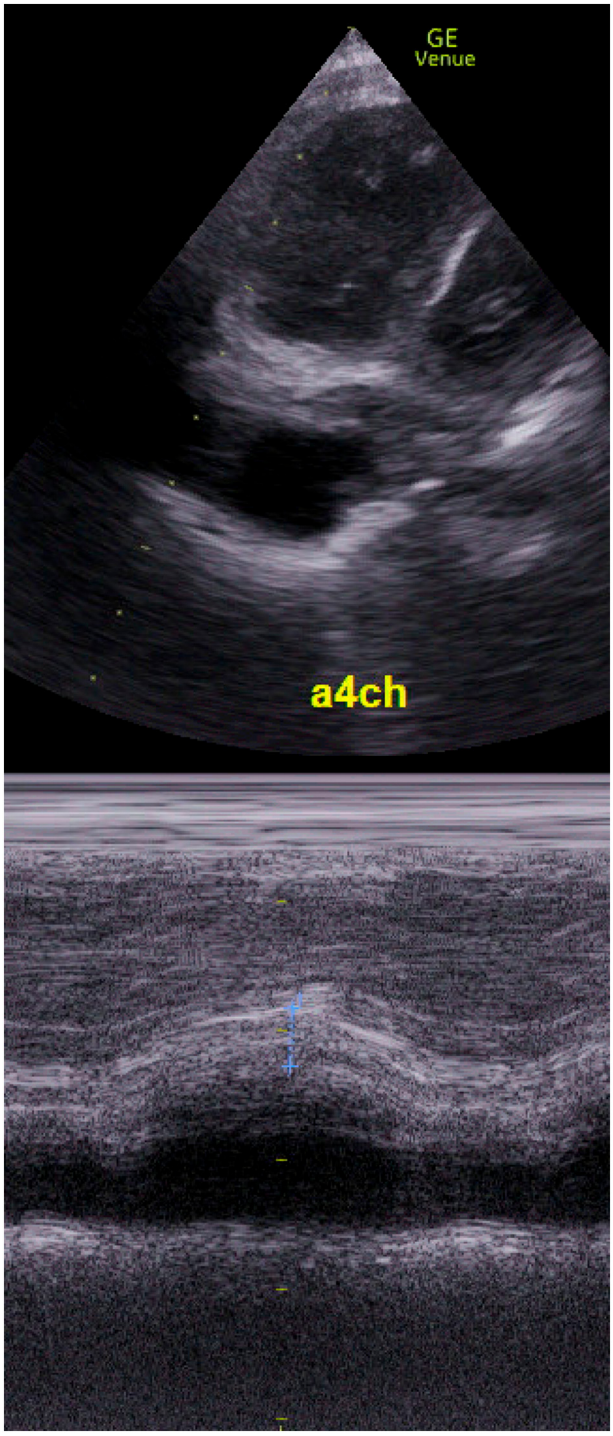

Fig. 4. Normal TAPSE measurement in M-mode.

When considering afterload, hypoxia and acidosis worsen PH by causing pulmonary vasoconstriction. Therefore, supplemental oxygen is warranted if the patient is hypoxic. High flow nasal cannula (HFNC) or nebulized beta agonists in a patient with RHF due to obstructive lung disease may improve RV afterload and RV function [32,55]. Hypervolemia also increases afterload, so if the patient is volume overloaded, then diuresis can assist [59]. Intravenous (IV) antihypertensive medications will also reduce systemic blood pressure leading to decompensation and are not specific enough to reduce pulmonary arterial hypertension alone to be useful in RHF [20]. However, inhaled nitric oxide can be considered if available in the ED, as some studies have shown beneficial effects on pulmonary vascular resistance and oxygenation [60]. Inhaled nitric oxide has isolated effects to ventilated areas of the lungs compared to IV pulmonary vasodilators and can improve V/Q mismatch [61]. Phosphodiesterase-5 inhibitors, such as sildenafil, and IV prostaglandins, such as epoprostenol, are not well studied in critically ill patients with acute RHF [62,63]. Initiating pulmonary vasodilators such as inhaled nitric oxide is best done in consultation with the pulmonary or critical care specialist, as not all forms of PH will benefit from pulmonary arterial vasodilators [64].

Patients with chronic PAH (Group 1 PH) who are already on a continuous pulmonary arterial vasodilator infusion, such as treprostinil, must remain on this infusion. Withdrawal can precipitate a pulmonary hypertension crisis and acute RHF [65]. While most patients on chronic pulmonary vasodilators receive this medication via central access, if the medication has been disrupted, thus driving the patient into RHF, it is acceptable to restart IV pulmonary vasodilators via a peripheral IV if central access is not immediately available [25]. The half-life of treprostinil is 2 to 4 h, but other infusions may have a shorter half-life [66]. For example, epoprostenol has a half-life of less than 5 min [66]. If a disruption in the continuous infusion is identified, the medication should be rapidly restarted [65].

Dobutamine, a selective beta-1 agonist, or milrinone, a selective phosphodiesterase-3 inhibitor, may be utilized to improve cardiac contractility and lower PVR [67,68]. The lowest possible dose of either inotropic medication is recommended to limit the risk of dysrhythmia. Milrinone may be preferred for patients on chronic beta blockers, since it does not affect beta receptors [67]. However, dobutamine has a half-life of 2 min compared to the half-life of milrinone which is 2–4 h [67]. While direct comparisons of these medications specifically for RHF are sparse, the limited evidence on RHF and trials in patients with advanced decompensated heart failure in general show similar clinical outcomes with either medication [69-71]. Given the shorter half-life, lower cost, and ED availability, dobutamine may be a preferable choice for use in the ED. While both of these medications can improve RV hemodynamics, systemic hypotension may occur especially with higher doses of these medications [72].

If the patient is hypotensive, vasopressors are recommended [32]. Hypotension can worsen RV ischemia, as RCA perfusion will be compromised if pulmonary vascular resistance exceeds systemic vascular resistance [27,28]. The goal is to keep systemic pressure above RV systolic pressure [20]. If available, a prior echocardiogram may provide a recent RV pressure measurement. Norepinephrine is an optimal first-line choice for hypotension because it increases systemic blood pressure with minimal effects on the pulmonary vasculature (especially if used in lower doses) [72]. Additionally, beta-1 adrenergic effects of norepinephrine may augment contractility [72]. Vasopressin is another effective vasopressor for refractory hypotension in RHF and may also have nitric-oxide based mechanisms to reduce PVR [73]. However, there are limited data for vasopressin in RV failure [44]. Phenylephrine should be avoided in patients with acute RHF, as it can worsen pulmonary hypertension and does not improve contractility [74].

Endotracheal intubation should be avoided if possible [75]. The increase in intrathoracic pressure after intubation and mechanical ventilation will worsen RV hemodynamics, decrease preload, and potentially lead to a peri-intubation cardiac arrest [55,75]. High flow nasal cannula (HFNC) or even non-invasive positive pressure ventilation (NIPPV) with low end expiratory pressures should be attempted in most cases before intubation [32,76]. These may improve oxygenation and ventilation, thus improving RV hemodynamics [20]. However, NIPPV should be used with caution, as this can worsen preload [55]. A trial of inhaled nitric oxide or inhaled epoprostenol is recommended by some experts prior to intubation in order to prevent cardiovascular collapse, but this may not benefit all patients (for example RHF secondary to LHF) [64,77]. In RV failure without hypoxemia, inhaled nitric oxide at 30–80 ppm via nasal cannula or inhaled epoprostenol during pre-oxygenation or apenic oxygenation can reduce pulmonary vascular resistance [77]. However, inhaled

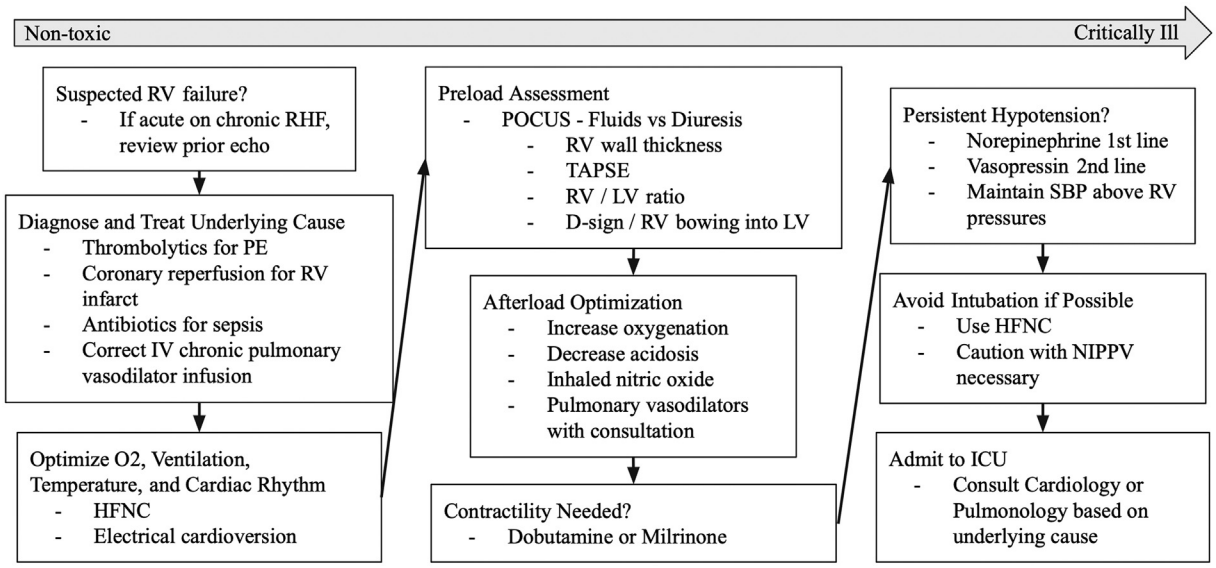

Fig. 5. Summary of key management points in a patient presenting with acute RHF.

nitric oxide at lower concentrations (less than 30 ppm) should be used if the patient is already hypoxemic prior to intubation, as higher concentrations can worsen V/Q mismatch [77].

If intubation is required, the most experienced clinician should perform the procedure, with consideration of vasopressor infusion during the sedation and intubation [78]. When considering the method of intubation, a case series demonstrated successful intubation utilizing awake intubation techniques in patients with RHF, but hypotension was still a frequent complication, occurring in 55% of the patients [79]. In this case series both atomized lidocaine in the oropharynx and supraglottic area and viscous lidocaine were used to achieve topical analgesia, and systemic sedation was utilized with a goal to achieve anxiolysis and analgesia [79]. Once a patient with RHF is placed on mechanical ventilation, the lowest positive-end expiratory pressure (PEEP) and lowest tidal volume (TV) as possible should be used while avoiding hypoxia and hypercarbia, as higher PEEP and TV levels will decrease venous return and worsen RV preload [78]. This differs from the typical management of LHF with pulmonary edema in which higher PEEP is generally recommended [80]. The goal plateau pressure should be less than 30 mmHg in order to decrease compression of the pulmonary vasculature [25]. It is also important to avoid hypercapnia and hypoxia once intubated as these will worsen pulmonary vasoconstriction [78,81].

If the patient continues to decompensate despite optimal medical management, extracorporeal membrane oxygenation (ECMO) may be considered. While the literature on ECMO in RV failure is limited, veno-venous ECMO can be considered in cases of isolated hypoxemic respiratory failure, whereas veno-arterial ECMO can be considered patients with intrinsic RV myocardial infarction or in RV failure with concomitant LV failure [82]. In most cases the goal of ECMO in the setting of RV failure is to rest the heart while supporting other organs and perfusing the RV. However, ECMO can also be considered as a temporary treatment such as in a patient with a massive PE waiting for thrombectomy or as bridge therapy to implantation of an RV assist device [82]. Mechanical RV assist devices (RVADs) are under study, but use is currently limited to end stage disease or as a bridge to transplantation in patients with PH [20]. Fig. 5 summarizes key management components for the patient with RHF.

4. Conclusion

While the diagnosis and management of RHF can be complex, understanding the underlying pathophysiology and RV hemodynamics can assist with treatment decisions. The initial step in management is to treat and reverse the underlying cause of the RHF exacerbation. POCUS is a useful adjunct to clinical assessment especially in a hypotensive patient who may either benefit from diuresis if the patient is volume overloaded and the RV is overdistended, or fluids if the patient is volume depleted. After preload optimization, contractility can be augmented with dobutamine or milrinone. Early use of vasopressors, such as norepinephrine or vasopressin, in a hypotensive patient is important to prevent continued cardiovascular collapse. A trial of HFNC or NIPPV is recommended if the patient is in respiratory distress. Intubation should be avoided if possible in a patient with RHF, as positive pressure mechanical ventilation can worsen right heart hemodynamics. It is essential that emergency physicians are able to diagnose and treat this life-threatening condition in order to reduce patient morbidity and mortality.

CRediT authorship contribution statement

Matthew Kostura: Supervision, Visualization, Writing – original draft, Writing – review & editing. **Courtney Smalley:** Visualization, Validation, Writing – original draft, Writing – review & editing. **Alex Koyfman:** Validation, Supervision, Conceptualization, Writing – review & editing. **Brit Long:** Conceptualization, Supervision, Visualization, Writing – original draft, Writing – review & editing.

Declaration of Competing Interest

None.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ajem.2022.05.030.

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