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REVIEW

Perioperative right ventricular dysfunction in adult patients undergoing non-complex cardiac surgery: diagnosis and management

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Abstract

Even though there is no precise consensus on definition of the right ventricular dysfunction and the right ventricular failure, their reported incidence after cardiac surgery ranges from 0.04% to 2.9%. Right ventricular dysfunction is clinically important because it has been consistently shown to have a negative impact on the perioperative outcomes. In this article we explore current evidence on perioperative diagnosis and management of adult patients with right ventricular dysfunction who are undergoing non-complex cardiac surgery. We propose criteria for the right ventricular dysfunction, describe its pathophysiological mechanisms, diagnostic and therapeutic options as well as current challenges. The function of the right ventricle is determined by preload, afterload, contractility, ventricular interdependence, and heart rhythm. These five factors should all be assessed in a particular patient. Right ventricular dysfunction is not always easy to confirm. Transthoracic and transesophageal echocardiography are the mainstay of diagnosis. Also, clinical and laboratory findings should be considered especially when the patient approaches the extreme of right ventricular dysfunction spectrum—right ventricular failure. Right ventricular failure should be anticipated, and management focused on its prevention and treatment of the underlying cause. The clinical aim is to optimise the volume status, minimise the right ventricular afterload, optimise right ventricular free wall and interventricular septum performance, and to maximise the left heart pressure work. If the patient does not respond to conservative therapy, mechanical support should be promptly considered.

Keywords

Right ventricle; Right ventricular dysfunction; Right ventricular failure; Heart failure; Cardiac surgery; Management; Diagnosis; Definition

1. Introduction

The right ventricle (RV) has unique haemodynamic properties. Its anatomical position and functionality are in series with the left ventricle (LV). Thus, right ventricular dysfunction (RVD) impacts on the LV and cardiac output regardless of its cause and even if it originates from the LV problems. There has been emphasis on importance of recognizing RVD in patients undergoing cardiac surgery as, for example, preoperative RVD is a strong predictor of survival after cardiac surgery [1–5]. Also, "*de novo*" or worsened intraoperative and postoperative RVD leads to increased length of stay, higher morbidity, and higher mortality [5]. The aetiopathogenesis of RVD in cardiac surgery includes ischemia, pulmonary hypertension, reperfusion lung injury, pulmonary embolism, acute unloading of the LV after left ventricular assist device (LVAD) insertion and sepsis [4, 6]. Intraoperatively, RVD typically leads to a difficult weaning from the cardiopulmonary bypass (CPB) and is also associated with increased perioperative mortality [7].

While we have evidence of perioperative RVD in cardiac surgery impacting patient outcomes, there is much less consensus on the definition of RVD. One of the problems is that RVD and right ventricular failure (RVF) are often used interchangeably in scientific literature. However, RVF is the extreme of the RVD spectrum and can be better defined as "inability of RV to maintain enough blood flow through pulmonary vasculature to achieve adequate LV filling" [8]. On the other hand, RVD can be vaguely defined as evidence of abnormal RV structure or function [9], but there is a significant lack of consensus on how to specify and diagnose it. For example, a just recently published paper on RVD in COVID-19 patients reported a much lower prevalence and a much higher mortality [10] than a meta-analysis of 29 studies on the topic [11]. The difference most likely results from more stringent inclusion criteria and missing the less sick patients [12]. This is only one example which reflects the fact that there is no

universal definition of RVD.

When making a diagnosis of RVD, several echocardiographic parameters can be considered in addition to assessing adequate filling of the RV:

- tricuspid annular plane systolic excursion (TAPSE) (Fig. 1a).
- pulsed tissue Doppler S wave (S') (Fig. 1b).
- fractional area change (FAC) (Fig. 1c).
- RV size/dilatation (Fig. 1d and **Supplementary Video**).
- pulmonary artery pressure.
- right ventricular-arterial uncoupling.
- interventricular septal flattening (Fig. 1e).
- paradoxical septal motion.

With this review we aimed to explore the current evidence on perioperative diagnosis and management of RVD in adult patients undergoing non-complex cardiac surgery and thus providing a synopsis of how to optimise routine care of these patients.

2. Diagnostic criteria for RVD

There is a need for a clear and universal definition of RVD which would be the basis for further research on the topic. Thus, for the patients undergoing cardiac surgery we suggest the cut-off values for the definition of RVD to be at the lower threshold, *i.e.*, less stringent inclusion criteria including the less sick patients too. This is because patients with RVD have lower physiological reserve of the RV and have worse perioperative outcomes. The RVD diagnosis would lead to a more vigilant care in the entire perioperative period. We suggest that any individual criterion present in Table 1 is sufficient for the diagnosis of RVD.

Clinical and laboratory findings also play a role in the diagnostic management, but they present at a later stage when RVD approaches the far end of the spectrum—RVF with liver congestion. We do suggest that clinical signs of congestion should be sought for in the entire perioperative period even though they are a late sign of RVD. For example, in this way one can detect a slowly progressing pericardial effusion ultimately leading to cardiac tamponade in a patient following cardiac surgery who is already admitted to the regular ward. Increased laboratory values of troponin, brain natriuretic peptide, elevated liver enzymes and lactate should indicate further echocardiographic assessment of the RV (Table 1).

3. Pathophysiology

RVD and RVF occur as a consequence of volume or pressure overload or due to intrinsic myocardial contractile dysfunction. While the LV is adapted to the high-pressure system of systemic arterial circulation, the RV is adapted to the low-pressure system of pulmonary circulation and is more suited to accommodate fluctuating blood volumes due to variations in venous return without the increase in end diastolic pressure. Fluid overload of the RV and bulging of the septum towards the LV lead to the reduction of stroke volume of the LV. In this case the septum moves because the pericardium constraints RV free wall from dilating outwards. While an abrupt increase in afterload leads to only a slight decrease in stroke volume of the LV, it causes a marked decrease in RV stroke volume once the compensatory capacity of the Frank-Starling mechanism is exceeded [16, 17]. Further dilatation of the RV stretches the tricuspid annulus which in turn increases preload and worsens RV stretch.

Thus, the function of RV is determined by preload, afterload, contractility, ventricular interdependence, and the heart rhythm. To understand RVF we must always assess these five components [17].

4. Preoperative period

4.1 Preoperative acute RVF

Acute RVF commonly occurs after a sudden increase in RV afterload or due to the intrinsic myocardial dysfunction, rather than from volume overload. Generally, pulmonary embolism is the main cause of a sudden increase in RV afterload. The effect of pulmonary embolism on RV is determined by anatomical site of the embolus, release of humoral growth factors and hypoxic vasoconstriction. When thrombotic occlusion extends to more than 50% of the lung vessels and, in turn, pressure elevation occurs, the unconditioned RV can overcome a mean pulmonary arterial pressure of up to 40 mmHg [17]. Resultant increase in pulmonary artery pressure causes RV strain with dilatation and hypokinesia, tricuspid regurgitation with annular dilatation, which leads to reduced RV stroke volume [16].

Volume overload occurs in a variety of clinical conditions with tricuspid and pulmonary regurgitation and aggressive fluid resuscitation in the postsurgical or critical care setting. Experiments in animal models show that volume overload is tolerated better than pressure overload [16].

However, the most common cause of RVF before cardiac surgery is intrinsic myocardial dysfunction caused by RV myocardial infarction. Ischemic injury decreases RV contractility and leads to RV dilatation and dyskinesis. Decrease in RV compliance causes reduction in RV stroke volume, which worsens ventricular interdependence, leading to hemodynamic collapse. RV involvement in myocardial infarction has a worse prognosis, since Shock and Core trials found, that patients with inferior myocardial infarction and RV myocardial involvement remain at high risk for death despite revascularisation [16, 18, 19].

In acute RVF caused by pulmonary embolism, RV myocardial infarction or cardiac tamponade, avoidance of fluid depletion is particularly important. Ensuring adequate preload in these situations can lead to hemodynamic optimisation until underlying cause is managed [20–22].

4.2 Preoperative chronic RVF

Chronic RVF can be a consequence of heart disease or lung disease [17]. Left heart disease leads to increased left atrial pressures, which will result in pulmonary hypertension (PH) even in absence of intrinsic pulmonary arterial disease. Persistent changes in left atrial pressure eventually lead to elevated pulmonary vascular resistance, through intimal fibrosis and medial hypertrophy of pulmonary arteries and reduced vasodilator response. As the pulmonary vasculature becomes less compliant, accompanying endothelial dysfunction leads to

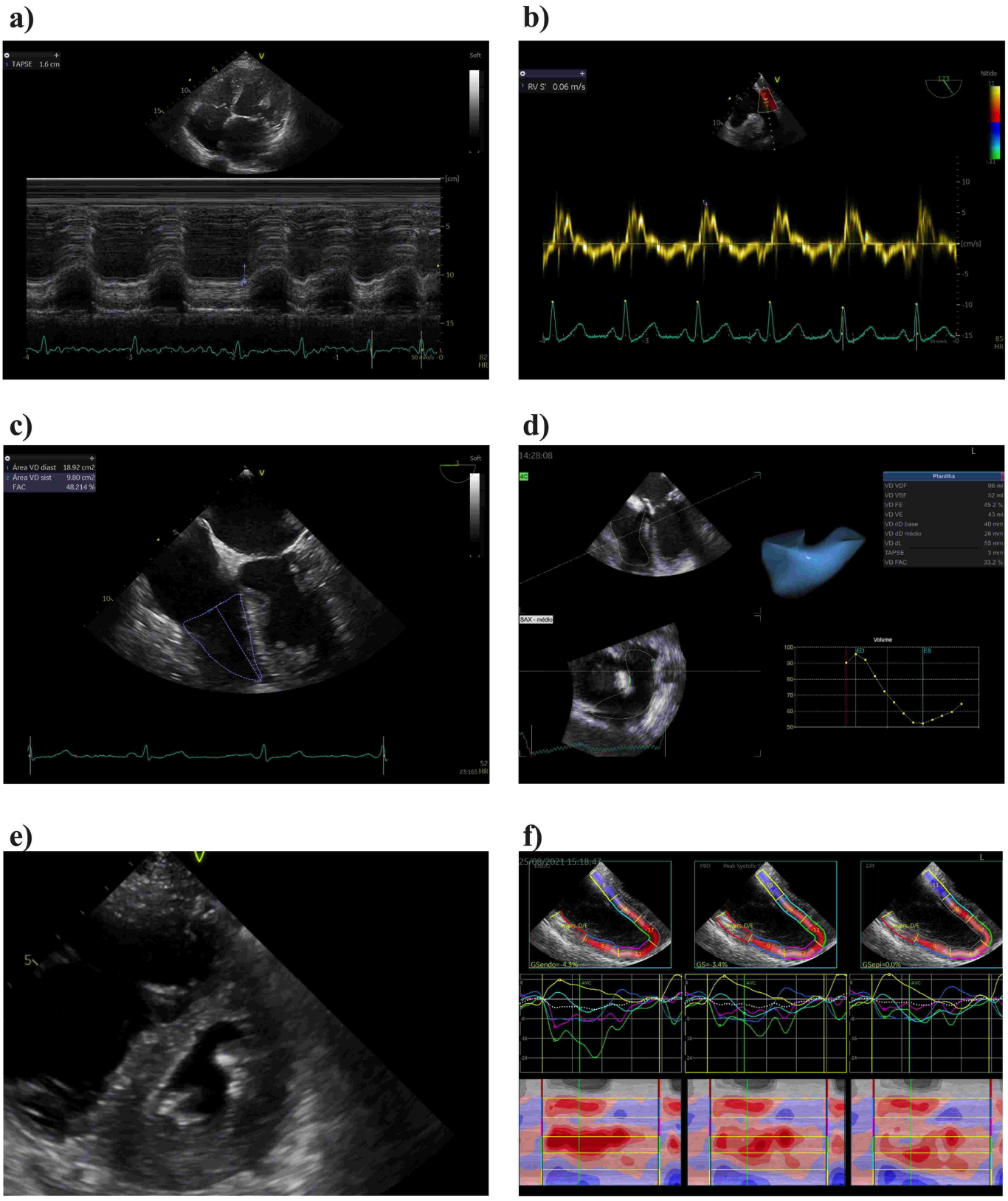

FIGURE 1. Examples of echocardiographic parameters for assessing the right ventricular dysfunction. (a) tricuspid annular plane systolic excursion of 16 mm. (b) pulsed tissue Doppler S wave (S') of 6 mm. (c) fractional area change of the right ventricle. (d) right ventricular size in three-dimensional mode. (e) interventricular septal flattening. (f) regional longitudinal shortening of the right ventricle with strain echocardiography.

TABLE 1. Criteria for diagnosing the right ventricular dysfunction (RVD) in the presence of adequate filling of the right ventricle (RV) [1–15]. We propose that any individual criterion is sufficient for diagnosing RVD in patients undergoing cardiac surgery.

Echocardiographic assessment of the RV	Suggested cut-off value for RVD
tricuspid annular plane systolic excursion	<17 mm
pulsed tissue Doppler S wave velocity (S') in the basal segment of the RV free wall	<10 cm/s
fractional area change (ideally measured in at least 2 planes)	<35%
mean pulmonary artery pressure	>20 mmHg
RV-arterial uncoupling (ratio of tricuspid annular plane systolic excursion and systolic pulmonary artery pressure)	<0.635 mm/mmHg
RV dilatation (ideally in three-dimensional echocardiography)	Yes
Septal flattening present	Yes
Paradoxical septal motion	Yes

RV, right ventricle; RVD, right ventricular dysfunction.

reduction in nitric oxide production and increased endothelin production. These changes can initially be reversible, but eventually become fixed. Atrial fibrillation and hypoxia due to chronic congestion may worsen RV function in patients with PH [16]. Also, arrythmias can worsen RVD and pericardial disease may also alter RV preload and ventricular interdependence [17]. In constrictive pericarditis the RV filling is impaired and venous return decreased which results in elevated systemic venous pressure, congestion and reduced cardiac output [23]. This is first treated medically, while surgical intervention is reserved for the end-stage disease with operative mortality of up to 20% [23].

RVF due to lung disease is also called "*cor pulmonale*". The most common respiratory disease causing RVF is chronic obstructive pulmonary disease which increases RV afterload through different mechanisms: obliteration of vascular beds, hypercapnia and acidosis, pulmonary hyperinflation, airway resistance, endothelial dysfunction, and hypoxia. Hypoxic pulmonary vasoconstriction results in elevated pulmonary pressures and when persistent vascular remodelling and fixed PH [17].

Initially, PH causes RV wall thickening with increased RV systolic pressure and increased end-diastolic volume. With longer duration of PH, the RV transitions to maladaptive phase which is characterised by inflammation, impaired angiogenesis, and fibrosis [16].

4.3 Preoperative diastolic RVD

Some recent studies have suggested that already diastolic RVD can contribute to worse perioperative outcome in cardiac surgery patients [24, 25]. However, this topic warrants further investigation.

4.4 Preoperative management

4.4.1 Fluid management

In chronic RVF careful monitoring and avoiding fluid overload is the mainstay of therapy. Patients are advised to weigh daily to quickly spot fluid retention [17]. Initial assessment of the patient should include assessment of the severity of fluid overload and identifying and treating the underlying cause. We should consider discontinuation of drugs that cause fluid retention, reduce salt intake, reduce fluid intake, and use 5% glucose for diluting intravenous drugs instead of 0.9% saline. Serum creatinine and glomerular filtration rate should be checked and if the latter is less than 20 mL/min, the patient should be referred to a nephrologist. If glomerular filtration rate is over 20 mL/min, we should consider oral loop diuretic. Fluid overloaded patients should aim to lose 0.5–1 kg per day. If the patient is not losing weight, the dose of loop diuretic can be increased, and other classes of diuretics can be added [26]. The goals of volume management in chronic RVF are to maintain sufficient preload for adequate cardiac filling while providing relief from RV volume overload, ventricular interdependence, and congestion [27].

4.4.2 Oral medication

Renin-angiotensin-aldosterone system inhibitors and beta blockers are not recommended in patients with PH regardless of RVF, unless associated with hypertension, coronary artery disease, or LV failure. Patients with biventricular dysfunction should be managed according to current practice guidelines for the management of chronic heart failure [27]. Phosphodiesterase inhibitors can also be used in the setting of PH.

5. Intraoperative period

5.1 Intraoperative causes of RVD

Acute exacerbation of RV function during cardiac surgery can also result from abnormal preload, reduced contractility of RV and/or pressure overload. Any combination of these three sources is of particular importance. Acute increase in RV afterload can be a result of acidosis, protamine, hypoxia and/or positive pressure ventilation [27]. This can abruptly decrease RV stroke volume. RV contractility can be acutely hindered by direct myocardial injury due to ischemia, reperfusion injury and/or stunning [27]. Abnormal preload ranges from inadequate filling (*e.g.*, relative hypovolemia, altered heart rate and rhythm—such as loss of atrial kick) to overfilling due to

excessive transfusion/infusion.

5.2 Intraoperative assessment of RV function

For adequate intraoperative assessment the preoperative state of RV function, current hemodynamic findings, and transoesophageal echocardiography (TEE) examination results should be considered.

With pulmonary artery catheter in place, we have continuous monitoring of the systolic and diastolic pulmonary artery pressures, right and left atrial pressures, cardiac output, and pulmonary vascular resistance [28]. Multiple hemodynamic parameters are associated with RVD (Table 2). However, compared to imaging techniques, they are inconsistent across studies.

Combined with invasive hemodynamic assessment, echocardiography is essential for diagnosing the RVD/RVF. With the use of ultrasound, RV size and function can be thoroughly quantified. Trans-tricuspid pressure gradient is a reliable measurement of pulmonary artery systolic pressure. Quantitative assessment of global RV function is recommended by at least one of the following: FAC, TAPSE, doppler tissue imaging-derived systolic S' velocity of the tricuspid annulus or RV index of myocardial performance. RV global and regional longitudinal shortening may be estimated by strain echocardiography (Fig. 1f). RV ejection fraction estimation with two-dimensional echocardiography is not recommended [28]. If available, 3D echocardiography is recommended to assess RV size and function.

Echocardiography is a useful tool to determine what causes RVD/RVF and distinguish whether it is right ventricular systolic dysfunction or right sided pressure/volume overload. In the presence of systolic RVD, we see TAPSE <17 mm, RV FAC <35%, and RV S' <10 cm/s (Table 1). Parameters that are pointing to right sided pressure/volume overload are: RV basal end-diastolic diameter >41 mm, RV versus LV end diastolic diameter ratio >1.0, septal shift, or D-shaped LV in systole and/or diastole, RV thickness >5 mm, inferior vena cava diameter >21 mm and collapsibility <50%, and tricuspid regurgitation peak systolic velocity >2.8 m/s [28]. RV dilatation can present as D-shaping where the interventricular septum is moving towards LV. In the case of isolated D-shaping, the RV is volume overloaded. If D-shaping is present during the whole cardiac cycle, RVF is caused by increased afterload. The best view to visualise D-shaping is the transthoracic parasternal short-axis view or transgastric short axis view in TEE. RV dilatation can also be seen in the 4-chamber view where a RV:LV diameter ratio >0.6 indicates a RV dilatation and a RV:LV ratio >1 indicates a severe RV dilatation. A severe RV dilatation causes stretching of tricuspid annulus which in turn leads to functional tricuspid regurgitation, but this can also be a consequence of pulmonary hypertension.

5.3 Intraoperative management of RVD

In cardiac surgery, RVD should be anticipated, and management should be focused on prevention. Thus, RV afterload should be minimised, RV performance should be optimised (free wall and interventricular septum), and left heart pressure work should be maximised [29]. Avoidance of atrioventricular asynchrony and maintenance of sinus rhythm is important in preventing deterioration of RV function [6]. Patients with known PH, RVD, severe ventricular dysfunction, long CPB period should be identified and managed by multidisciplinary teams [28].

Intraoperative factors that influence RVD are suboptimal myocardial protection, myocardial stunning after long duration of CPB, air or thromboembolism to the right coronary artery and mechanical occlusion or kinking of the right coronary button or bypass graft. Simultaneous anterograde-retrograde cardioplegia provides superior RV myocardial protection. Excessive transfusion should be avoided to prevent increased RV volume load. In the case of hemodynamic instability after sternal closure, delayed sternal closure can be considered. Whether delayed sternal closure is efficacious for RVF is not known. In a small study, routine delayed sternal closure after

TABLE 2. Hemodynamic parameters of right ventricular dysfunction (RVD) [28].

Hemodynamic parameter	Measurement/Calculation	Threshold for RVD/RVF
RAP	RAP (or CVP)	>15 mmHg (after LVAD)
Right-to-left discordance of filling pressures	RAP: PCWP	>0.63 (after LVAD) >0.86 (in acute myocardial infarction)
Pulmonary artery pulsatility index	(PASP-PADP)/RAP	<1.0 (in acute myocardial infarction) <1.85 (after LVAD)
RV stroke work index	(MPAP-CVP) xSVI	<0.25–0.3 mmHg·L/m^2 (after LVAD)
PVR	(MPAP-PCWP)/CO	>3.6 WU (after LVAD)
Pulmonary artery compliance	SV/(PASP-PADP)	<2.5 mL/mmHg (in chronic heart failure, RV-PA coupling in PAH)

Abbreviations: CO, cardiac output; CVP, central venous pressure; LVAD, left ventricular assist device; MPAP, mean pulmonary artery pressure; PA, pulmonary artery; PADP, pulmonary artery diastolic pressure; PAH, pulmonary artery hypertension; PASP, pulmonary artery systolic pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RV, right ventricle; RVD, right ventricular dysfunction; RVF, right ventricular failure; SV, stroke volume; SVI, stroke volume index (SVI = cardiac index/heart rate); WU, Woods units in mmHg/L/min.

implantation of an LVAD did not prove to be beneficial in reducing complications associated with hemodynamic instability including cardiac tamponade or RVF [30].

Hemodynamic instability after CPB or weaning failure will be associated with reduced cardiac output and low near-infrared spectroscopy values indicating poor tissue perfusion. Recognition of RV function focuses on visual heart assessment, RV pressure waveform analysis and echocardiographic assessment of the diastolic diameter, systolic function, and assessment of RV outflow tract obstruction. Severity of hemodynamic instability correlates with a gradient above 25 mmHg, which can be confirmed with TEE. If we rule out mechanical compression, inotropic support should be reduced, volume administered and reduction in heart rate should be considered. If we confirm RVF, it is important to rule out any early surgical complication such as compression of the pulmonary artery or obstruction at the pulmonary artery anastomosis. Priority is to avoid the vicious circle of hypotension and subsequent RV ischemia. We should strive for heart rate and rhythm optimisation, optimal fluid management, follow principles of protective ventilation without reduced or excessive lung volumes, avoid drugs that could increase pulmonary artery pressure and maintain normal acid-base status. Ischemia and evidence of LV dysfunction must be excluded with potential need for medical or surgical reperfusion. Sometimes, isolated RVF will be present, which can occur in severe pulmonary reperfusion syndrome, in patients with reduced RV reserve and during carbon dioxide embolism. When pulmonary artery resistance is elevated, RV afterload reduction can lead to drastic improvement, which can be achieved with inhaled nitric oxide (iNO), inhaled prostacyclin and inhaled milrinone alone or in combination. In RVF, we strive to increase RV contractility as long as there is no LV outflow tract obstruction. Here, phosphodiesterase inhibitor or beta agonist alone or in combination with vasopressin or alpha agonist can be used to maintain right coronary perfusion pressure [7].

5.3.1 Minimising RV afterload

Before adding specific vasodilators, the following measures should be instituted [29]:

- adequate oxygenation.
- adequate depth of anaesthesia and paralysis.
- hypercarbia and acidosis should be avoided.
- pleural spaces void of air, blood, or effusion.
- appropriate mechanical ventilation.
- clear airway and breath sounds without wheezing.

Once these have been assured, then pulmonary specific vasodilators should be used such as iNO with the initial dose of 10 ppm [29].

5.3.2 Maximising RV function

Approximately half of the RV function is derived from the free wall and half from the interventricular septum [29]. The RV during CPB should be well protected with antegrade and retrograde cardioplegia. RV blood supply should be sufficient (revascularisation plan, graft kinking, *etc.*) and inotropic support should be initiated if needed.

5.3.3 Maximising left heart pressure work

The interventricular septum has significant contribution to RV function. Weaning from CPB should be done with as much developed LV pressure as possible. For patients receiving LVAD this might require some reduction in LVAD flow and allow some LV filling to increase LV developed pressure [29].

5.3.4 Mechanical ventilation

Hypercapnia increases pulmonary artery pressure, independently of the presence of hypoxia. Acidaemia and hypoxemia produce pulmonary vasoconstriction, with synergistic effect. Chronic hypoxemia can produce vascular remodelling with muscularization and narrowing of small pulmonary arteries. Both, vasoconstriction and remodelling, increase pulmonary vascular resistance and RV afterload. Mechanical ventilation in patients with RVD/RVF can have unfavourable hemodynamic effects. Heart and intrathoracic vessels are highly compliant, so changes in pleural pressure cause similar changes within these structures. Application of positive pressure with mechanical ventilation increases pleural pressure. Preload is dependent on pressure gradient between right atrium and systemic veins and by venous resistance. A positive pressure breath increases pleural and consequently right atrial pressure (RAP), which reduces the pressure gradient driving venous return, and RV preload falls. PEEP also augments pleural and RAP throughout the respiratory cycle. A drop in venous return is caused primarily by an increase in venous resistance due to narrowing of the hepatic veins and the superior vena cava [31, 32].

The RV pumps blood into highly compliant pulmonary vasculature at low pressures and is sensitive to changes in afterload. Changes in RV output are mainly mediated through changes in intrathoracic pressure. The most important factor of RV afterload is resistance of the pulmonary circulation. During the respiratory cycle, the lowest pulmonary vascular resistance is found at end-expiration at functional residual capacity. Positive end-expiratory pressure (PEEP) increases lung volume and transpulmonary pressure throughout the respiratory cycle, it magnifies the hemodynamic effect of mechanical ventilation and causes a continuous elevation in pulmonary vascular resistance. The extent of RV compromise is comparable between different modes of ventilation, in the case of similar mean airway pressures and tidal volumes [31, 33].

The heart chambers are enclosed in relatively noncompliant pericardium and share a common septum. Changes in the size of one ventricle can alter the size of the other. Normally the interventricular septum bows to the RV. When RV afterload increases, RV pressure rises, stroke volume falls and the ventricle dilates. Interventricular septum is pushed towards the LV, which reduces its compliance and impedes LV filling. Decreased RV stroke volume and reduced LV filling causes LV preload and stroke volume to fall, which can lead to cardiogenic shock. Mechanical ventilation can cause an acute increase in PVR which can lead to refractory shock in patients with pre-existing RVD. Low tidal volumes and minimal PEEP with prevention of hypoxemia and acidemia may prevent or reduce these haemodynamic changes [32].

5.3.5 Central extracorporeal membrane oxygenation (ECMO) support

In the case of post-cardiotomy shock with severe RVF, central veno-arterial ECMO may be the best option due to the already inserted aortic and atrial/bicaval cannulas [34]. Despite there is a higher rate of bleeding and acute renal failure in central ECMO in comparison to peripheral ECMO, no difference in overall survival was found by a recent meta-analysis [35]. The advantage of central cannulation is also in preventing the "Harlequin syndrome" since it is improving cardiac decompression and proximal aortic flow [36, 37].

6. Postoperative RVD and RVF

The overall incidence of RVD/RVF after cardiac surgery is not exactly known as the topic is understudied and the definitions are variable. One French study found a 2.9% incidence RVF following cardiac surgery [38] and acute refractory RVF ranges from 0.04 to 0.1% [4]. The mechanisms of RVF after cardiac surgery are more complex than the traditional pathophysiology of RVF which is focusing on hemodynamics [39]. There are many causes which can lead to RVF after cardiac surgery: prolonged CPB, poor myocardial protection during the CPB, preexisting RVD/RVF, arrhythmias, metabolic acidosis, hypothermia, RVF after LVAD insertion and pulmonary embolism [40]. In addition, air embolism, microthrombi, malfunction of a coronary bypass and hypotension can aggravate ischemia of the RV. Following CPB, several factors can cause "*de novo*" or aggravate a pre-existing PH: ischemia-reperfusion injury, pulmonary embolism, heparin-, protamine- or transfusion-induced pulmonary vasoconstriction, hypoxia, hypercapnia, and acidosis [41]. A sudden increase of afterload is not well tolerated by RV and causes RV dilatation with an increase in cardiac workload and contractile dysfunction which leads to diminished cardiac output, coronary perfusion and worsening cardiac failure [6, 42]. RVF has the potential to aggravate vasoplegia after cardiac surgery. Venous congestion following RVF triggers the release of inflammatory mediators in the liver which itself is the greatest source of vasodilatory cytokines [43, 44]. RVF has a great morbidity and mortality due to the resulting multiorgan failure [45].

6.1 Diagnosis of RVD and RVF in the intensive therapy unit

In every patient with signs of shock, a thorough clinical, point-of-care echocardiography and laboratory examination should be performed to exclude or confirm the cause. Signs of systemic congestion are difficult to assess after cardiac surgery and elevated liver enzymes could be a consequence of cardiopulmonary bypass and the accompanying systemic inflammation per se [46]. The diagnosis of RVD/RVF should be based on all available information and in the clinical context of the patient. All potential differentials for the shock should be systematically considered. Echocardiography is the gold standard for bedside evaluation of cardiac function and a basic point-of-care exam can also confirm or exclude potential reversible causes of shock (*e.g.*, tension pneumothorax, cardiac tamponade, left ventricular failure) [47].

Due to its unique shape, it is difficult to estimate the RV ejection fraction. Wanner and Filipovic propose to assess three key findings of RVF: signs of RV dilatation, signs of impaired RV systolic function and sign of increased RV preload [48] (see section 5.2). The most useful and easily obtained echocardiographic estimation for RV function is TAPSE, measured with TTE in the apical 4-chamber view or with TEE in the mid-oesophageal view [49]. TAPSE values $<$17 mm indicate impaired RV systolic function. The limitation of TAPSE is that it can overestimate RV systolic function in massive tricuspid regurgitation [48]. A deep description of echocardiographic assessment is behind the scope of this paper, but the interested reader is referred to the two guidelines [28, 50].

The pulmonary artery catheter is still the gold standard to diagnose pulmonary arterial hypertension and its aetiology, but due to lack of standardised use has some pitfalls in interpreting the measured values [51]. Due to its potential for life-threatening complications, it should be used only in complex cases or cases resistant to therapy [48]. Based on scientific data, the threshold PH was lowered from 25 mmHg to 20 mmHg mean pulmonary arterial pressure recently and the phenotypes of PH were redefined [13]. It is important to distinct pre-capillary, isolated post-capillary and combined pre-capillary and post-capillary PH to choose the appropriate therapy [48]. If pulmonary artery catheter is used, the measured values should always be evaluated together and in concert with clinical status of the patient.

6.2 Treatment (Fig. 2)

The best treatment of RVF is to prevent its evolvement and the vicious cycle of multiple organ failure due to venous congestion and diminished cardiac output. Avoiding increases (especially sudden) in pulmonary vascular resistance, decreases in systemic blood pressure causing coronary hypoperfusion, and greater derangements of volemia are essential measures to prevent RVF. Clinicians are often focusing on LV failure and aggressively treat the LV which in turn can worsen RV function by reducing the RV preload due to diuretics and/or increasing the RV afterload due to high PEEP. When managing LV failure, the potential harm for the RV should be kept in mind. Recognition and treatment of potential reversible causes (*e.g.*, myocardial infarction, pulmonary embolism, tension pneumothorax, cardiac tamponade, sepsis) is crucial. The therapy of RVF should aim to optimise coronary perfusion, optimise RV preload, increase RV contractility, maintain sinus rhythm, and lower RV afterload. In general, optimisation of oxygenation and normocapnia should be obtained. If possible, invasive mechanical ventilation should be avoided (see section 5.3.4). To correct hypoxia, high-flow nasal cannula can be used [52]. Another option to avoid intubation is a trial of non-invasive ventilation with careful titration of PEEP and pressure support [48]. If mechanical ventilation is necessary, an optimal patient-ventilator synchrony and adequate analgosedation should be attained [48]. To diminish the impact of mechanical ventilation on RV afterload high tidal volumes and high PEEP values (typically above 8 cm H_2O) should be avoided as well as pleural overcharge and bronchospasm [53]. As long as the appropriate PEEP value allows additional

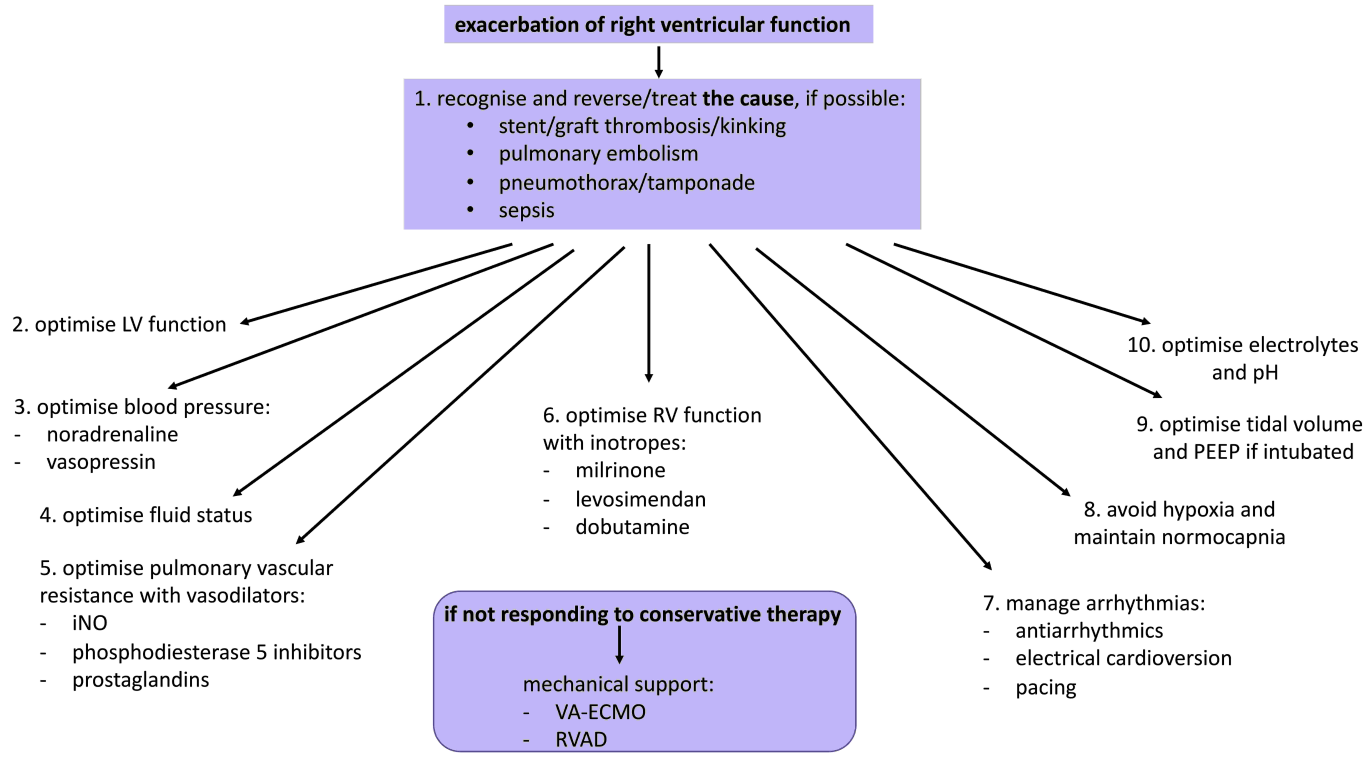

FIGURE 2. Management of the acute exacerbation of right ventricular function in the perioperative period. LV, left ventricle; iNO, inhaled nitric oxide; RV, right ventricle; ECMO, extracorporeal membrane oxygenation; RVAD, right ventricular assist device; PH, pulmonary hypertension; PEEP, Positive end-expiratory pressure; VA, veno-arterial.

lung recruitment and prevention of hypoxic vasoconstriction, PEEP is not contraindicated [48]. To prevent an additional deterioration of myocardial contractility a normal acid-base equilibrium and normothermia should be maintained [53].

6.2.1 Vasopressors

To improve coronary perfusion in hypotensive patients, vasopressors should be used. In acute RVF, systemic hypotension should be managed aggressively to avoid rapid deterioration and noradrenaline is the drug of choice [54]. Noradrenaline improves coronary perfusion without increasing pulmonary vascular resistance at lower dosing and improves ventricular systolic interaction. Vasopressin acts on V1 receptors and causes vascular smooth muscle contraction. In low doses it causes pulmonary and systemic vasodilation, but in higher doses it causes systemic vasoconstriction. It has antidiuretic effects and not fully elucidated cardiac effects [55]. In acute RVF data for vasopressin are lacking, but it seems to be a promising drug due to its selectivity for systemic circulation [28, 48].

6.2.2 Volume optimisation

Because RV is highly dependent on optimal volume status hypovolemia and hypervolemia should both be avoided. Since it is often misunderstood, that RV can tolerate liberate volume loading, a careful assessment of volume status is needed [47]. If hypovolemia is identified (which is mostly the case during the first hours after surgery), careful volume resuscitation should be provided [8]. In case of hypervolemia, which mostly appears after the first day after surgery following volume resuscitation, loop diuretics should be given. If hypervolemia persists despite the use of diuretics and judicious volume administration, hemofiltration should be started. Hypervolemia reduces venous return by increasing central venous pressure [56] and causes dilatation of the RV in diastole which may displace the interventricular septum and cause a diminished output of the LV [57].

6.2.3 Pulmonary vasodilators

Pulmonary vasodilators lower the pulmonary vascular resistance which represents the RV afterload. There are 3 types of pulmonary vasodilators: iNO, prostaglandins, and phosphodiesterase 5 inhibitors.

iNO is a highly selective pulmonary vasodilator with an extremely short half-time and rapid onset of action. iNO increases intracellular cyclic guanosine monophosphate (cGMP) levels. The inactivation by haemoglobin in lung capillaries prevents systemic vasodilation. It has greater effect on blood vessels in good-ventilated lung areas. Some authors suggest doses up to 40 ppm [29], yet a small sized study found that doses higher than 10 ppm had no additional effect on reducing pulmonary vascular tone [58]. Toxic effects of iNO are dose dependent and include methemoglobinemia, accumulation of free oxygen radicals, and nitrogen dioxide [59]. Weaning from iNO or prostaglandins can cause rebound pulmonary hypertension [53].

Prostaglandins epoprostenol (E1 prostaglandin) and the prostacyclin analogue iloprost are potential pulmonary vasodilators with rapid onset of action and a short half-life. They increase intracellular cyclic adenosine monophosphate

(cAMP) levels. They can both be administered as an inhalation without the need of special equipment, also in spontaneously breathing patients. Administered as inhalations are haemodynamically safe and if administered in combination with iNO potentiate its vasodilatory effect. Epoprostenol can be administered as an inhalation or intravenously and has no secondary toxic effects.

Phosphodiesterase 5 inhibitors act through inhibition of the metabolism of cGMP. They have systemic vasodilatory effects and longer half-lives (4–18 hours) and should be used with caution in haemodynamicaly unstable patients.

Other pulmonary vasodilators, such as riociguat or endothelin receptor antagonists should not be used in acute RV failure due to data of increased mortality and systemic vasodilatory effects [8].

In acute RVF it is important to use selective pulmonary vasodilators (inhaled form) to prevent systemic vasodilation and aggravation of RVF [60].

6.2.4 Inotropes

The most often used inotropes in RVF are levosimendan and milrinone. The use of dobutamine can also be considered.

Levosimendan is a unique drug with cardiovascular and some extravascular effects. In Europe it has been widely used for acute and chronic left-sided heart failure over the last 20 years [61]. By binding to calcium saturated troponin C it exerts positive inotropy without having a negative impact on heart muscle relaxation or oxygen consumption. It also strengthens contractions of diaphragm and skeletal muscles [62]. By binding to potassium channels it has a vasodilatory effect which improves organ perfusion and reduces preload and afterload of both ventricles. A recent meta-analysis of 10 studies using levosimendan in acute RVF found a statistical important short-term efficacy of improving RVF and systolic pulmonary artery pressure and pulmonary vascular resistance [63].

Milrinone is a positive inotrope which acts through inhibition of the phosphodiesterase III. It causes pulmonary vasodilation and improves RV contractility. It causes less tachycardia than dobutamine, but it can worsen systemic vasodilation. Due to its primarily renal clearance, it should be used with caution in acute renal failure [54, 64].

Dobutamine is a positive inotrope acting on the β1 and β2 receptors and alpha1 adrenoceptors. At lower doses it improves RV contractility and simultaneously causes pulmonary vasodilation. Its main unwanted effects are the risk of systemic hypotension, increased myocardial oxygen consumption and a risk for arrhythmias [65]. It should be used with caution in haemodynamically unstable patients and patients with arrhythmias [52].

6.2.5 Managing cardiac rate and rhythm

In severe RVF all kinds of arrhythmias can occur. The most common arrhythmias are sinus tachycardia as a consequence of maximal sympathetic tone, atrial fibrillation or atrial flutter. Bradyarrhythmias are mostly a sign of terminal RVF or of severe damage of the conduction system [48]. The most important measure is to treat the underlying cause. In haemodynamically unstable patients with atrial fibrillation or flutter, electrical cardioversion should be tried with great care regarding analgosedation which should not worsen the haemodynamic status. In stable patients a careful trial with amiodarone or ibutilide can be tried [47]. Since beta blockers and calcium channel blockers can precipitate a cardiovascular collapse, they should never be given for rate control in acute RVF [48, 66].

If severe tricuspid regurgitation is present, bradycardia would worsen the regurgitation and heart rate should be maintained at the upper level of normal range.

If bradyarrhythmia is present, the underlying cause should be identified and as soon as possible treated. For immediate treatment adrenaline should be titrated in small doses to the desired effect, since it also improves the systemic pressure. In resistant cases, transvenous pacing should be attained, keeping in mind the potential technical complications and the potential of triggering a malignant arrhythmia [48].

6.2.6 Mechanical support for the RV

Mechanical support devices are invasive options carrying high risk of bleeding and infection complications. However, they are the only specific therapy to target RVF [67, 68]. If the patient does not respond to conservative therapy, an early decision about mechanical support should be made [67, 69]. The two options include right ventricular assist device (RVAD) and ECMO.

RVAD is a good option as a bridge to recovery in ischaemic or post-infarction RVF. RVAD can be inserted percutaneously (right jugular vein, right femoral vein) or surgically, where the surgeon inserts one cannula through a median sternotomy or thoracotomy into the right atrium and another one into the main pulmonary artery. The purpose is to connect the right atrium to pulmonary artery via the pump to bypass the RV. Modern percutaneously inserted RVAD cannulas are dual lumen cannulas with one lumen positioned in the right atrium and the other in the main pulmonary artery. A detailed description of haemodynamic effects of different devices is described elsewhere [67].

ECMO enables cardio-respiratory support for patients with hypoxic respiratory failure and/or cardiogenic shock. It is an invasive therapy which allows an aggressive lung rest and/or a full or partial cardiovascular support depending on the cannulation site. Due to its invasiveness and potential life-threatening complications, the indication for ECMO must be carefully weighed against potential harm [37]. After cardiac surgery, the common indications for ECMO are cardiogenic shock resistant to medical therapy and intraaortic balloon pump, post-cardiotomy shock (unable to separate from CPB which we described in section 5.3.5), primary graft failure or hyperacute rejection following heart transplantation. It can be used as a bridge to recovery, as a bridge to RVAD/LVAD or as a bridge to heart transplant. ECMO can also be used as a periprocedural support in high-risk percutaneous coronary intervention, in RVF after the LVAD insertion or as an option of extracorporeal resuscitation [70, 71]. It can be used in case of acute respiratory distress syndrome (ARDS) or septic shock which can appear as a late complication after surgery [70]. The most common contraindications to ECMO are disseminated malignancy, unrecoverable heart disease in patients which are

not candidates for heart transplant/ventricular assist device, known severe brain injury, known chronic end-stage disease (*e.g.*, liver cirrhosis), unwitnessed or prolonged cardiac arrest, unrepaired aortic dissection, severe aortic insufficiency, severe chronic pulmonary hypertension [70].

The literature regarding ECMO effectiveness in acute RVF is scarce [67, 68, 72]. We have listed selected case reports in Supplementary Table 1. In case of acute RVF, especially if this is a consequence of increased afterload, veno-arterial ECMO can be used to bypass the RV [73]. In veno-arterial ECMO blood is drawn from right atrium, oxygenated, and returned to a central artery. In veno-veno-arterial ECMO blood is drawn from the right atrium and pulmonary artery and after oxygenation returned to a central artery. We have already described another central ECMO cannulation approach in section 5.3.5. Regardless of the cannulation site, veno-arterial ECMO is usually effective in unloading the RV but might not be effective in decompressing LV because there are two afterload dependent pumps working against each other: LV and ECMO. In case of LV distension and intraventricular or intra-atrial stasis, a LV unloading device should be used (surgically inserted vent, intraaortic baloon pump or transaortic valve axial pump device) [36, 74]. Peripheral cannulation seems to be suited for late RVF after cardiac surgery, replacing central ECMO in case a longer need for veno-arterial ECMO exists, or as an urgent rescue therapy for bridging the time to surgery [36, 68]. The advantages of peripheral cannulation may be avoiding resternotomy, facilitating chest compression during cardiopulmonary resuscitation and reducing infections [36].

7. Conclusions

We described pathophysiological mechanisms of the acute and chronic RVD/RVF in the perioperative period with the diagnostic and therapeutic options for their management. Preoperative acute RVF commonly occurs due to pulmonary embolism, myocardial ischemia, and volume overload. During and after cardiac surgery there are additional causes related to the surgical procedure, CPB, and metabolic derangements. Due to the unique shape of the RV, it is not always easy to diagnose RVD. The best treatment of RVD is its prevention since there is no ideal medication or procedure for treating a developed RVD. Especially regarding the diastolic dysfunction of RV further studies are needed.

AVAILABILITY OF DATA AND MATERIALS

Not applicable.

AUTHOR CONTRIBUTIONS

AMP and MZ—designed the study, AMP, MZ, RB, DS and DM—performed the literature search and wrote the manuscript. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

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CONFLICT OF INTEREST

The authors declare no conflict of interest. Marko Zdravkovic is serving as one of the Guest editors of this journal. We declare that Marko Zdravkovic had no involvement in the peer review of this article and has no access to information regarding its peer review. Full responsibility for the editorial process for this article was delegated to KPV.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found, in the online version, at https://oss.signavitae.com/mre-signavitae/article/1673249069107953664/attachment/Supplementary%20material.docx.

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