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REVIEW

A standardized definition for right ventricular failure in cardiac surgery patients

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Abstract

Right ventricular failure (RVF) is a significant cause of mortality and morbidity after cardiac surgery. Despite its prognostic importance, RVF remains under investigated and without a universally accepted definition in the perioperative setting. We foresee that the provision of a standardized perioperative definition for RVF based on practical and objective criteria will help to improve quality of care through early detection and facilitate the generalization of RVF research to advance this field. This article provides an overview of RVF aetiology, pathophysiology, current diagnostic modalities, as well as a summary of existing RVF definitions. This is followed by our proposal for a standardized definition of perioperative RVF, one that captures RV structural and functional abnormalities through a multimodal approach based on anatomical, echocardiographic, and haemodynamic criteria that are readily available in the perioperative setting (Central Image).

Keywords Right ventricular failure; Cardiac surgery; Perioperative care; Intensive cardiac care unit

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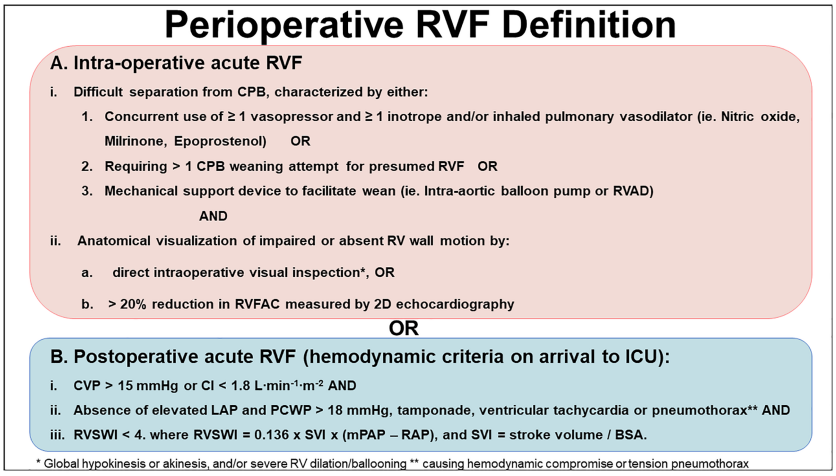
Central Image: defining perioperative right ventricular failure

Right ventricular failure (RVF) carries a significant burden of morbidity and mortality following cardiac surgery, necessitating early recognition and prompt treatment. We propose a multimodal, standardized definition of RVF, which is further classified into intraoperative and/or post-operative phases. Intraoperative RVF is characterized by difficult separation from cardiopulmonary bypass, defined by the need for pharmacologic or mechanical support and objective evidence of RV contractile dysfunction. The diagnosis of post-operative RVF is based on both traditional and derived haemodynamic criteria on arrival to the cardiac surgical intensive unit.



Abbreviations: BSA, body surface area; CI, cardiac index; CPB, cardiopulmonary bypass; CVP, central venous pressure; ICU, intensive care unit; LAP, left atrial pressure; mPAP, mean pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; RAP, right atrial pressure; RV, right ventricle; RVAD, right ventricular assist device; RVF, right ventricular failure; RVFAC, right ventricular fractional area change; RVSWI, right ventricular stroke work index; SVI, stroke volume index.

Introduction

Although right ventricular failure (RVF) is infrequent following routine cardiac surgery, it remains a significant mediator of perioperative and long-term morbidity and mortality.[1–5] The incidence of severe acute perioperative RVF is 0.1% after cardiotomy, up to 18% after orthotopic heart transplantation (OHT),[5] and 20–30% after left ventricular assist device (LVAD) implantation. RVF typically presents intraoperatively as difficulty in separation from cardiopulmonary bypass (CPB) and as low cardiac output syndrome or multi-organ dysfunction in the early post-operative setting. The occurrence of RVF is more frequent following valvular surgery, especially in the presence of pulmonary hypertension (PH),[6] OHT,[5] LVAD insertion,[7] and surgery for ischaemic[8] and adult congenital heart disease.[9]

Right ventricular failure in the perioperative setting is difficult to diagnose and even more challenging to treat. Refractory post-operative RVF is associated with up to 22–90% likelihood of in-hospital mortality,[2,9–11] as well as significant morbidity, including prolonged mechanical ventilation, intensive care unit (ICU) length of stay, renal and hepatic failure, and increased need for inotropic and mechanical circulatory support.[7,12–14] Despite its prognostic importance in cardiac surgery patients, no universally accepted definition of RVF exists in this setting.[15] The lack of a standardized definition also prevents the incorporation of RVF in major cardiac surgery mortality risk models.[16] The advent of perioperative echocardiography has enabled earlier and more rapid assessment of RV anatomy/function and hence aetiology-specific management. This modality, along with commonly accepted clinical and haemodynamic criteria, could be used to formulate a standardized definition of RVF in the perioperative setting. In turn, a standardized definition would enhance communication between surgeons, anaesthesiologists, and intensivists, improving patient care and outcomes, as well as enhancing the practical utility and comparability of research studies.

In this article, we will review perioperative RVF pathophysiology and RV assessment modalities, summarize existing RVF definitions, and propose a new, standardized definition in the context of cardiac surgery. Although current literature does not support a fully data-driven RVF definition specific to the perioperative setting, our proposed definition is based on clinical expertise, backed by encompassing RV diagnostic modalities, and their prognostic data in cardiac surgery patients. Our definition also addresses the shortcomings found in previously proposed definitions of RVF based on a thorough literature review. The importance of creating a perioperative definition of RVF specifically in the context of cardiac surgery is grounded in the physiologic distinction between the cardiac operating room and the ambulatory care setting. Specifically, during cardiac surgery, the diseased heart needs to adapt to ischaemia–reperfusion, the CPB circuit, shifts in loading conditions, mechanical ventilation, as well as newly imposed haemodynamic changes after valvular reconstruction.

Right ventricular physiology and pathophysiology

The primary role of the RV is to pump venous return from the right atrium into the pulmonary circulation, allowing blood oxygenation by the lungs. The pulmonary vasculature is normally a low impedance circuit, such that the RV is optimized for high volume capacitance and low afterload states. Additionally, the RV is a thin-walled structure, with its peristaltic contraction is derived from its free wall longitudinal fibres and the interventricular septum (IVS),[17] with an inflow to outflow delay of 20–40 ms.[18]

Right ventricular free wall

The RV free wall is perfused by marginal branches of the right coronary artery (RCA), whereas its posterior wall is supplied by the posterior descending artery and the anteroseptal wall from the left anterior descending artery.[19] Unlike the LV, the RV is perfused during both systole and diastole and possesses dense collateral vessels. RV function is therefore highly dependent on coronary perfusion pressure (CPP) (CPP = aortic diastolic pressure − RV end-diastolic pressure).[19] As such, the RV is particularly vulnerable to decreases in systemic pressure and increased pulmonary artery pressures.[17,20]

Ventricular interdependence

Right ventricular function is also influenced by ventricular interdependence (VI), the direct mechanical LV–RV interactions through the IVS.[17] The higher intracavitary pressure of the LV transmits through the IVS, creating a scaffold against which the RV free wall can contract. This pressure differential between ventricles (i.e. transeptal gradient)[21] dictates normal septal position, allowing systolic VI to contribute up to 60% of global RV function.[17,22] In diastolic VI, which occurs throughout the cardiac cycle, the loading of one ventricle affects the pressure–volume relationship of the other. Diastolic VI is dependent on the continuity of myocardial fibres between the IVS and RV and can occur without an intact pericardium.[23]

Preload

The RV is thin-walled, smaller mass with higher compliance than the LV. It is due to these anatomic considerations that the RV can readily accommodate varying preloads and is more sensitive to acute changes in afterload,[2,3,24,25] with typical mean pressures of 25/4 mmHg (range 15–30/1–7 mmHg), maximal ventricular elastance of 1.3 mmHg/mL,[26] and arterial elastance of 1.0 mmHg/mL.[27] In cases of severe RV volume or pressure overload, a reversal of the transeptal gradient shifts the IVS to the left, reducing LV diastolic filling and systemic cardiac output.[25]

Afterload

Right ventricular afterload is not only dependent on pulmonary valve function and pulmonary vascular resistance (PVR) but also pulmonary arterial impedance.[25] PVR increases in the setting of CPB through release of inflammatory mediators (i.e. prostaglandins and leukotrienes), as well as direct endothelial and reperfusion injury of the pulmonary circulation.[28] Physiologic states such as hypoxia, hypercarbia, acidosis, and hypothermia, as well as extremes of ventilatory volumes, or excessive transfusions contribute to increased RV afterload.[28,29]

Aetiologies of right ventricular failure in cardiac surgery

Physiologically, RVF is defined as an inability of the RV to provide adequate blood flow through the pulmonary circulation at normal right atrial pressures.[2] Clinically, RVF manifests as a triad of hypotension, elevated central venous pressure (CVP) $>$ 15 mmHg, and clear lungs.[24,30] In more advanced states, RVF can result in arrhythmias and poor forward flow leading to shock, systemic congestion, and multi-organ failure, particularly acute kidney injury. Common aetiologies of RVF in the setting of cardiac surgery are summarized in *Figure 1* (*Figure 1*, adapted from Jabagi *et al*.).[4]

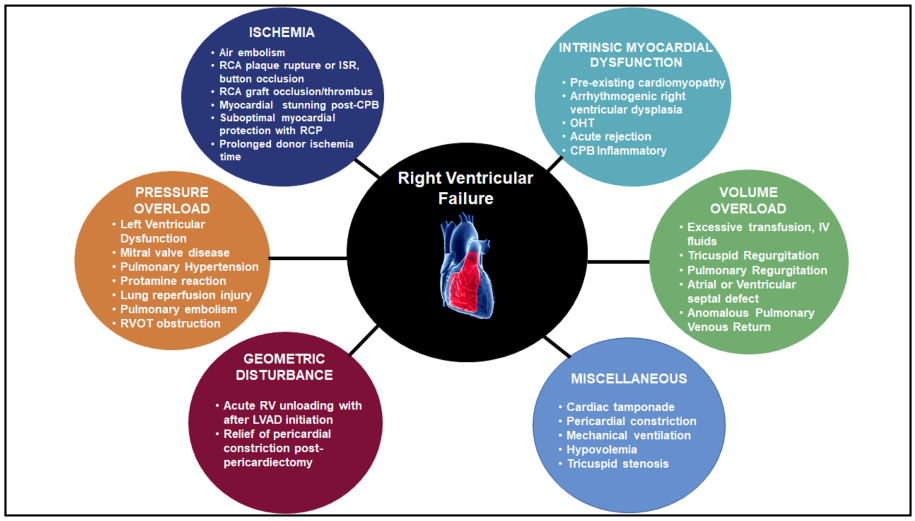
Intrinsic right ventricular myocardial dysfunction in the setting of cardiac surgery

Intrinsic RVF is most commonly attributed to poor myocardial preservation with exclusive retrograde cardioplegia use[8] and is further exacerbated by inflammation, prolonged CPB duration,[31] and pre-existing RV dysfunction.[32] RV ischaemia from coronary air embolism, plaque rupture, and RCA graft occlusion are other important contributing factors.[2] Additionally, large preoperative RV infarcts have been identified as a major cause of perioperative mortality.[33]

Pressure and volume overload

Right ventricular failure may be secondary to elevated PVR in the context of PH,[29] as well as LV failure, anatomic or functional RV outflow obstruction,[1] mitral valve disease, and congenital heart disease,[34] all of which could be exacerbated by CPB-related cytokine release, reperfusion injury, or

Figure 1 Aetiologies of perioperative right ventricular failure. CPB, cardiopulmonary bypass; ISR, in-stent restenosis; LVAD, left ventricular assist device; OHT, orthotopic heart transplant; RCA, right coronary artery; RCP, retrograde cardioplegia; RV, right ventricular; RVOT, right ventricular outflow tract. Reproduced and modified with permission from the authors.[4]



protamine reaction.[29] In the setting of OHT, 20% of early mortality can be attributed to RVF due to the prevalence of pre-existing PH in heart failure patients.[5]

Right ventricular volume overload can also be iatrogenic through excessive fluid administration,[12] as well as in the context of congenital heart defects (e.g. atrial septal defects with left to right shunt, Tetralogy of Fallot, and pulmonary regurgitation).[9] Severe tricuspid regurgitation may also cause inadequate forward flow and RV distension.[2]

Right ventricular failure in the setting of left ventricular assist device

Finally, RV geometry is critical in the setting of mechanical circulatory support. LVADs acutely unload the LV, causing a leftward IVS shift, thereby reducing IVS contribution to RV contraction.[21] This results in RV dilation and failure to maintain adequate output to match LVAD flows, ultimately leading to device suction events, low cardiac output, and eventual RV burnout.[15,35]

Modalities to assess right ventricular function

Gold standards for right ventricular function assessment

Currently, two gold standards are available: cardiac magnetic resonance imaging (cMRI) as the non-invasive standard and right heart catheterization (RHC) as the invasive standard. In the perioperative setting, cMRI is rendered impractical due to presence of epicardial pacer leads, prolonged ventilation, and cost.[4,14] Instead, RHC-derived haemodynamic measurements are more often used to define RVF, in addition to PA and RV pressure waveform morphology,[36] making haemodynamic measures the more practical gold standard for RVF assessment in the perioperative setting.

Echocardiographic right ventricular assessment

Transesophageal echocardiography (TEE) is widely available in the perioperative setting. The American Society of Echocardiography[37] recommends using at least one quantitative echocardiographic measure to assess the RV when dysfunction is suspected. These standardized measures include RV size, septal morphology and position, and surrogates of RV ejection fraction (RVEF). RV size is best measured by the basal diameter on the mid-oesophageal four-chamber view, where $>$42 mm indicates dilatation. Septal flattening in diastole is generally indicative of volume overload, while septal shift throughout the cardiac cycle is consistent with pressure overload.[37] Although RVEF is directly measurable, it relies on numerous volumetric assumptions and is rarely used perioperatively.[38]

Right ventricular fractional area change (RVFAC) is defined as the ratio of end-diastolic and end-systolic areas, with normal values ranging between 35% and 60%. It correlates with RVEF measurements by cMRI.[37] More recently, three-dimensional RVEF measurement has been validated and shown to be reliable and reproducible.[38] Due to the RV's primarily longitudinal contraction, tricuspid annular

plane excursion (TAPSE) is one of the most commonly used echocardiographic surrogates of RVEF and is well correlated with both cMRI and RHC-derived RVEF measurements.[39] TAPSE is defined as the distance that the tricuspid valve annulus descends towards the apex during contraction. Normal TAPSE is defined as 21–27 mm, and $<$17 mm is indicative of severe RV systolic dysfunction.[37] A major limitation of RVFAC and TAPSE is that they are both load-dependent metrics.

Tissue Doppler imaging of basal wall velocity, S′, is a load-independent measure of RV function that was previously applied in the LVAD population[40] but remains to be validated in the setting of routine cardiac surgery.[2] RV strain is another tissue Doppler-based modality for assessing RV function. It is defined as percentage change in myocardial deformation[37] and independently correlates with MRI measurements.[41] RV global longitudinal strain of $> -21\%$ is associated with mortality in patients undergoing cardiac surgery.[42] Despite strong correlations between RV strain and gold standard metrics, it is highly angle dependent and variable and lacks normative data.[43] Lastly, the RV myocardial performance index (RVMPI) is a global measure of the relationship between both ejection and non-ejection work of the RV.[37] RVMPI $>$ 0.5 suggests RV systolic or diastolic dysfunction. RVMPI has been validated in the settings of PH[42] and provides risk stratification for patients undergoing high-risk valvular surgery.[44]

Right ventricular assessment in the setting of left ventricular assist device

Some RHC-derived haemodynamic measures have been proposed in recent years. Amongst these, the PA pulsatility index (PAPi), defined as the ratio of pulmonary artery pulse pressure and CVP, is associated with RVF and need for an RV assist device (RVAD) after an LVAD procedure.[45] Other RHC-derived haemodynamic parameters such as CVP/PCWP have been described as independent predictors of RVF after LVAD implantation with a cut-off value of $>$0.63,[35] while the morphology of RV pressure wave tracings serves to provide additional physiological insight.[46,47] However, these haemodynamic measures suffer from load dependence and artefacts and remain to be validated in the cardiac surgery setting.

Biomarkers in the assessment of right ventricular failure

To date, few studies have examined the utility of biomarkers in diagnosing RVF, especially in the perioperative setting. However, several biomarkers of inflammation and myocyte injury/stress have shown superior potential in perioperative RV assessment, including Galectin 3, ST2/sST2, CRP, cTN/hs-cTn, and BNP/NT-proBNP.[4,14] Unfortunately, these specific markers are currently under clinical investigation and are not yet available for clinical use. Other important and clinically available markers of RVF include liver function tests (LFTs), serum creatinine, and glomerular filtration rate. Hepatic dysfunction is common in the setting of RVF and is primarily driven by passive venous congestion or low cardiac output.[4] The degree of elevation in LFTs correlates with the severity of RVF and hepatic congestion.[4] Similarly, RVF correlates with markers of renal injury through similar mechanisms.[48]

Pressure–volume loops

Pressure–volume (PV) loops are the gold standard for measuring real-time cardiac function, although mainly used in preclinical studies. By plotting simultaneously ventricular pressure against volume, PV loops provide a quantitative measure of a load-independent parameter of contractility—represented as the end-systolic pressure–volume relationship.[49] Morphological differences between the curves are indicative of increased load. However, PV loop assessment requires the use of a conductance catheter inserted into the RV, which is not always feasible in the perioperative setting.[50]

Previous definitions of right ventricular failure

Despite numerous methods available for perioperative RV assessment, there is so far no single diagnostic modality that is practical, accurate, and reproducible. Indeed, the lack of a standardized definition of perioperative RVF is a significant barrier to advancing the field, as clinical reports and research findings have not been generalizable across RVF studies. With numerous limitations and no single diagnostic technique to definitively characterize RV function in the perioperative setting, there is need for a universal definition that carefully encompasses anatomical, echocardiographic, and haemodynamic data.

Various criteria to define RVF have been proposed to date, but failure to distinguish between acute and chronic RVF are major limitations, as these entities are pathophysiologically and prognostically distinct. A summary of currently proposed definitions and their limitations for perioperative RVF in patients undergoing cardiac surgery is provided in *Table 1*.

Table 1 Previous studies defining perioperative right ventricular failure in cardiac surgery and associated limitations

Study	RVF definition	Population	Mortality (%)	Incidence (%)	Association with RVF	Definition limitations
Kaul and Fields[46]	CVP > 18, CI < 2.2, normal LAP.	Meta-analysis of patients undergoing cardiac surgery.	45–75	0.04–20		Parameters may not be specific to RVF and are dependent on volume status.
Maslow *et al.*[32]	RVFAC < 35%.	Patients undergoing CABG with LVEF < 25%, *n* = 41.	24	—	RV dysfunction associated with higher mortality.	RVFAC is affected by loading conditions.
Dávila-Román *et al.*[51]	Severe hypokinesis in >2 segments[a], RVFAC < 25%, and RV dilation (RVESV > 3 mm).	Low cardiac output syndrome post-cardiotomy, *n* = 75.	44	42	Haemodynamics alone could not detect RVF.	Load-dependent measure.
Moazami *et al.*[52]	Need for RVAD.	Patients after cardiotomy requiring right-sided support.	57	—		Excludes medically managed RVF.
Reichert *et al*[11]	RVFAC < 35%.	Hypotensive patients after cardiac surgery.	90	40		Load-dependent measure.
Denault *et al.*[10]	Haemodynamic instability, >20% RVFAC reduction, visualization of impaired RV wall motion.	High-risk surgical patients with PH.	22	15		Limited to the intraoperative setting.
Haddad *et al.*[44]	RVFAC < 32% or RVMPI > 0.5.	Patients undergoing mitral valve surgery, *n* = 50.	74	—	Pre-CPB RV dysfunction associated with post-operative circulatory failure and late mortality.	Echocardiographic parameters are affected by loading conditions.
Schuuring *et al.*[9]	Elevated jugular venous pressure and RV dysfunction on echo defined as TAPSE < 15 mm or RV S′ < 11 cm/s.	Patients undergoing surgery for congenital heart disease.	75	4.4		Measurement of jugular venous pressure is unreliable.
Ternacle *et al.*[53]	RV global longitudinal strain > −21%.	Patients post-cardiotomy, *n* = 250.	22	39.6		Angle-dependent measure, high degree of variability.
Gudejko *et al.*[54]	Need for RVAD, inotrope, or pulmonary vasodilator for >14 days.	LVAD patients, *n* = 110.	—	33	PAPi, CVP.	Lacks visual assessment and characterization of the RV.
Ochiai *et al.*[55]	RVF requiring RVAD.	LVAD patients, *n* = 245.	—	9	RVSWI, PAP.	Excludes medically managed RVF.
Grant *et al.*[56]	Need for RVAD or inotrope support >14 days.	LVAD patients, *n* = 117.	19	40	Reduced RV strain.	Excludes medically managed RVF. Inotropic therapy length confounded by practice variations and LVF.
Kavarana *et al.*[57]	Need for inotropes >14 days or RVAD.	LVAD patient, *n* = 69.	43	30	RVSWI.	Excludes medically managed RVF. Inotropic therapy length confounded by practice variations and LVF.
Matthews *et al.*[58]	IV inotropes >14 days iNO for ≥48 h, RVAD, or hospital discharge on an inotrope.	LVAD patient, *n* = 197.	—	35	RVF risk score (vasopressors, transaminitis, and renal dysfunction).	Inotropic therapy length confounded by practice variations and LVF.
Drakos *et al.*[59]	IV inotropes >14 days iNO for ≥48 h or RVAD.	LVAD patient, *n* = 175.	38	44	High PVR.	Inotropic therapy length confounded by practice variations and LVF.

(*Continues*)

Table 1 (continued)

Study	RVF definition	Population	Mortality (%)	Incidence (%)	Association with RVF	Definition limitations
Kormos *et al*.[35]	Need for inotrope support >14 days, need for RVAD, or late inotrope support >14 days after implant.	HeartMate II implantation, n = 484.	29	20	CVP/PCWP > 0.63.	Excludes medically managed RVF. Inotropic therapy length confounded by practice variations and LVF.
Fitzpatrick *et al*.[60]	Need for RVAD.	LVAD patients n = 266.	56	37	CI ≤ 2.2, RVSWI < 0.25 mmHg/L/m^2, preop RV dysfunction.	Excludes medically managed RVF.
LaRue *et al*.[61]	INTERMACS definition of severe (>14 day inotropes) or severe acute RVF (RVAD).	LVAD patients, retrospective, n = 445.	47–68	31		

CABG, coronary artery bypass grafting; CI, cardiac index; CPB, cardiopulmonary bypass; CVP, central venous pressure; iNO, inhaled nitric oxide; INTERMACS, Interagency Registry for Mechanically Assisted Circulatory Support; LAP, left atrial pressure; LVAD, left ventricular assist device; LVEF, left ventricular ejection fraction; LVF, left ventricular failure; PAPi, pulmonary artery pulsatility index; PCWP, pulmonary capillary wedge pressure; PH, pulmonary hypertension; PVR, pulmonary vascular resistance; RV, right ventricle; RVAD, right ventricular assist device; RVESV, right ventricular end-systolic volume; RVF, right ventricular failure; RVFAC, right ventricular fractional area change; RVMPI, right ventricular myocardial performance index; RVSWI, right ventricular stroke work index; TAPSE, tricuspid annular plane systolic excursion.
[a]RV segments defined as inferior, anterior, or free wall.

Echocardiographic definitions

Perioperative assessment of RV function is often challenging, given the interplay between intrinsic biventricular myocardial performance and dynamic changes in loading conditions.[4,13,14,62] Challenges to quantitative TEE assessment are due to the complex RV geometry and poor endocardial definition from trabeculation,[15] while qualitative evaluation is subjective to interobserver variability[12,63] and may not correlate well with the actual degree of venous congestion and clinical organ dysfunction.[4] Perioperative transthoracic echocardiography (TTE) is challenged by the presence of mechanical ventilation, chest tubes, and sterile bandages.[4,37] Lastly, echocardiographic measurements are often load dependent and more likely to reflect acute changes in RV preload or afterload rather than intrinsic myocardial contractility.[24,37]

The simplest definitions of RVF in the literature are based solely on echocardiographic characterization of RV dysfunction. TAPSE < 17 mmHg has been shown to be associated with higher post-operative mortality, inotrope requirement, and length of ICU stay after valvular surgery.[64,65] Maslow *et al*. defined RV dysfunction as RVFAC < 35% pre-CPB and demonstrated its association with early and late mortality, as well as longer ICU length of stay.[32] Based on this definition, Reichert *et al*. reported prevalence of up to 40% for isolated RVF in those undergoing cardiac surgery, with an associated 90% mortality of those who become haemodynamically unstable.[11] Using a definition of RVFAC < 30% or RVMPI > 0.5 in high-risk patients undergoing valvular surgery, Haddad *et al*. found RVF to be an important predictor of perioperative mortality.[44] Unfortunately, the absence of strict consensus between echocardiographic criteria across these definitions precludes direct comparison or validation of these parameters. As demonstrated in *Table 1* (summary of commonly proposed RVF definitions), to date, there are no standard echocardiographic criteria to define RVF.

Haemodynamic definitions

Right heart catheterization is commonly performed in the setting of cardiac surgery. Despite the CVP being frequently used as a surrogate for right-sided filling pressure, it is highly load dependent and unreliable in the setting of rapid fluid shifts and mechanical ventilation.[1,10] The use of PAP is inadequate as a standalone measure of RV function in patients with PH, as PAP tends to pseudo-normalize as RVF worsens.[25,30] In addition, PAP is dependent on LV function, mechanical manipulation of LV during surgery, as well as circulating volume.[29] Even thermodilution-derived cardiac output may become unreliable in the presence of tricuspid regurgitation or very low cardiac output states.[62]

The triad of RAP $>$ 8 mmHg, cardiac index $<$ 2.2 L/min/m^2, and normal left-sided filling pressure is associated with perioperative mortality rates of up to 75%.[46] However, haemodynamic criteria alone may be confounded by baseline volume status, as well as common pathological states such as tamponade, pneumothorax, pulmonary embolism, and PH in the perioperative setting. Dávila-Román *et al*. found that haemodynamics alone could not specifically distinguish post-operative RV dysfunction.[51] Recently, a three-item criterion for perioperative RVF was proposed by Denault *et al*. for non-LVAD patients, consisting of (i) difficult or complex separation from CPB, (ii) $>$20% reduction in RVFAC as measured by two-dimensional echocardiography, and (iii) direct intraoperative visualization of anatomically impaired or absent RV wall motion.[10] Although this definition incorporates haemodynamic instability and need for pharmacologic support, it is specific to the intraoperative setting and is not entirely applicable post-operatively.

Defining right ventricular failure after left ventricular assist device

Several criteria for post-operative RVF have been centred on the LVAD population, where RVF is most common.[59] The incidence of RVF as defined by Turner's criteria of need for inotropes $>$ 14 or nitric oxide $>$ 48 h or need of RVAD was 43% after LVAD implantation.[66] Studies using some or all of these criteria also reported higher rates of short-term and long-term mortality in those who developed RVF[57] (*Table 1*). In some cases, the diagnosis of RVF was based solely on the need for RVAD support,[55,60] although criteria for RVAD varied widely between studies.[35,56,58,59] Similarly, the need for inotropic therapy for $>$14 days[29,35,56,58] is confounded by practice variations across institutions. Various studies have demonstrated RV stroke work index (RVSWI), defined as work performed by RV each contraction indexed to heart rate and mean arterial pressure,[66] was predictive of RVF when RVSWI $<$ 4.[55,57,60] Additionally, a PAPi $<$ 1.85 has also been shown to be a sensitive predictor of RVF after LVAD implantation.[45,67]

To date, the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) criteria stand as the only formally accepted definition of RVF in the setting of LVAD implantation. The INTERMACS criteria include persistent signs and symptoms of RV dysfunction as evidenced by a CVP $>$ 18 mmHg and a cardiac index $<$ 2.0 L/min/m^2, requiring either RVAD or inhaled nitric oxide or inotropic therapy for $\geq$14 days after LVAD implantation.[42] When applied to a recent LVAD cohort,[61] the INTERMACS criteria identified a much higher RVF incidence (67%) as compared with previous criteria in similar settings (30–40%).[58,59,61,66] Despite this standardized definition, the data showed differences in outcome between subgroups of varying inotropic durations between 15–21 and $>$21 days, bringing into question the 14 day mark used in the INTERMACS definition.

Proposed standardized definitions in the setting of non-left ventricular assist device cardiac surgery

Herein, we propose a definition for RVF that captures RV structural and functional abnormalities, through a multimodal approach that incorporates anatomical, echocardiographic, and haemodynamic measurements that are readily available in the perioperative setting. Our proposed definition is based on multidisciplinary clinical expertise and readily available perioperative RV diagnostic modalities and is backed by available prognostic data to address the limitations of previously proposed definitions. By employing multiple diagnostic parameters, we also foster a team-based approach to the diagnosis and treatment of perioperative RVF, by involving surgeons to provide direct visual evaluation of RV function, anaesthesiologists and intensivist to provide additional confirmatory measures, and pursuant opportunity for team-based management discussions. Furthermore, we provide separate criteria for diagnosing RVF in the intraoperative and post-operative phases, to address the distinct physiologic challenges and available diagnostic modalities in these settings (Central Image).

Conclusions

The importance of a standardized definition for perioperative RVF cannot be understated. We have proposed a comprehensive definition of perioperative RVF in patients undergoing non-LVAD cardiac surgery using an expert-driven, multidisciplinary approach that builds on physiologic and prognostic data as described in state-of-the-art literature. This definition objectively combines important anatomical, echocardiographic, and haemodynamic criteria to improve the detection, prevention, and treatment of RVF in clinical practice, as well as in studies examining RVF to help accelerate the expansion of knowledge in this important area. Further prospective studies are needed to validate this definition as relating to outcomes in various cardiac surgery patient cohorts.

Clinical perspectives

The need for a perioperative definition of right ventricular failure (RVF) is urgent and stems from physiologic differences of cardiac surgery from the ambulatory setting. A standardized, consensus-based definition for RVF will enhance communication between clinicians as well as comparability

between research studies. We propose an expert consensus definition for perioperative RVF, based on available data and a multimodal approach that incorporates anatomical, echocardiographic, and haemodynamic measurements that are readily available in this setting. We further classify our definition into the intraoperative and post-operative phases to enable early detection, treatment, and prevention.

Translational outlook

Right ventricular failure is a major source of mortality and morbidity following cardiac surgery and is challenging to treat. The lack of a standardized definition for perioperative RVF not only prevents proper risk stratification and early treatment but also our ability to consistently interpret clinical research findings. A standardized perioperative RVF definition based on objective criteria will facilitate more rapid advancement in the care of patients who undergo cardiac surgery. Our proposed definition is grounded by expert opinion and guided by best available evidence. It may serve as an important first step to accelerate research and knowledge translation and improve patient outcomes.

Conflict of interest

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