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Clinical presentation and management of right ventricular dysfunction

E. Murphy[1,2] and B. Shelley[1,2,*]

[1]Golden Jubilee National Hospital, Glasgow, UK and [2]Academic Unit of Anaesthesia Critical Care and Pain Medicine, Glasgow University, Glasgow, UK

*Corresponding author: Benjamin.Shelley@glasgow.ac.uk

Learning objectives

By reading this article you should be able to:

- Describe a variety of conditions in which right ventricular (RV) dysfunction may occur.
- Describe a strategy for management of RV dysfunction, including an approach to rate, rhythm, perfusion, preload, contractility, and afterload.
- Be aware of the complex interaction between mechanical ventilation and RV dysfunction.
- Have a basic understanding of the mechanical support options available when pharmacological management fails.

Key points

- RV dysfunction may be encountered in a wide variety of clinical scenarios.
- Identifying the underlying aetiology is important in order to optimise RV function.
- Key management principles involve optimising rate, rhythm, perfusion, and preload, whilst maintaining contractility and minimising afterload.
- 'Traditional' mechanical ventilation strategies may worsen RV dysfunction.

The importance of identifying and managing right ventricular (RV) dysfunction is recognised increasingly by both anaesthetists and intensivists. In the face of an ageing population, with a broad range of increasingly complex comorbidities, the importance of RV function is applicable to far more patients than just those undergoing cardiothoracic surgery. Early recognition of RV dysfunction is essential to prevent RV failure (RVF) and improve morbidity and mortality. An approach to RV structure and function was discussed in an earlier article in this journal.[1] The aim of this second article is to provide an overview of the clinical conditions leading to RV dysfunction and outline an approach to managing these patients.

RVF is a heterogeneous syndrome, with a wide variety of aetiologies (both acute and chronic); individualised treatment is required.[2] Whilst there is no consensus definition for RVF, the term acute right heart syndrome has recently been described; acute right heart syndrome is defined as a rapidly progressive syndrome with systemic congestion resulting from impaired RV filling and or reduced RV flow output.[2]

A high index of suspicion is required in patients with potential acute RV dysfunction, as signs and symptoms may be non-specific. Acute signs of RVF are mainly a result of low cardiac output (CO) or systemic venous congestion. They include signs of hypoperfusion with deranged liver function tests, increased urea creatinine and lactate concentrations, and decreased venous oxygen saturations, all of which are non-specific to the diagnosis of RVF. Signs and symptoms of chronic RVF, such as ascites, exertional dyspnoea, reduced exercise tolerance, and ankle swelling may not always be

Emma Murphy MRCP *is a clinical research fellow based in cardiothoracic anaesthesia and intensive care at the Golden Jubilee Hospital, Glasgow, UK.*

Ben Shelley FRCA FFICM MD *is a consultant in cardiothoracic anaesthesia and intensive care at the West of Scotland Heart and Lung Centre, which includes the Scottish Pulmonary Vascular Unit, the Scottish National Advanced Heart Failure Unit (including mechanical circulatory support and cardiac transplantation) and the Scottish Adult Congenital Cardiac Service. He has an established research programme examining right ventricular function after lung resection.*

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present. Early use of transthoracic echocardiography (TTE) is recommended when RVF is suspected to aid early diagnosis, because management is challenging and patients often have a limited number of specific clinical signs.[3]

Clinical conditions in which RVF may be encountered

RV function can be impaired by volume or pressure overload, or by a reduction in myocardial contractility. Dysfunction is triggered by an injury or stress to the right ventricle with adaptation dependent on the nature of the insult, the duration of the disease, and time of onset (i.e. birth, childhood, or adulthood). Acute events, as seen in myocardial infarction (MI) or pulmonary embolism (PE), may quickly progress to RVF because of impaired contractility or acutely increased afterload. In chronic diseases, such as pulmonary hypertension (PH) or congenital cardiac disease, a gradual increase in RV afterload allows adaptive mechanisms to develop which preserve CO over a longer period of time before decompensation occurs.

Generally, the right ventricle adapts better to volume overload, as seen with tricuspid regurgitation (TR) and atrial septal defects, and it can withstand these conditions for a long period of time without significant change in RV systolic function. Acute pressure overload, as seen in PE, leads to a rapid increase in pulmonary vascular resistance (PVR), afterload, and RV wall tension, which quickly leads to RV dilatation and failure. Chronic pressure overload, which occurs commonly in PH, results in the right ventricle being able to adapt as the pressure increases gradually over time. Multiple compensatory measures exist in chronic pressure overload including myocyte hypertrophy, expansion of the extracellular matrix, reduction in wall stress, and upregulation of neurohormonal systems. Over time, the gradual increase in afterload results in a decrease in contractility, leading to progressive RV dilation and ultimately failure.

When considering the aetiology of RV dysfunction, it can be useful to categorise factors into those affecting preload, afterload, and contractility. Increased preload may occur in TR and atrial septal defects. Increased afterload may occur with PH and PE. Decreased contractility may be encountered in RV infarction, arrhythmias, or sepsis. In critically ill patients, RV dysfunction can often be multifactorial (e.g. with sepsis-induced acute respiratory distress syndrome [ARDS]) where RV function may be impaired by both increased afterload and reduced contractility.

Pulmonary hypertension

PH is defined by a mean pulmonary artery pressure (PAP) $\geq$25 mm Hg at rest, and is the commonest cause of RVF.[4] The WHO classifies PH into five broad classifications: (i) pulmonary arterial hypertension, (ii) PH secondary to left heart disease, (iii) PH associated with lung disease, (iv) chronic thromboembolic PH, and (v) miscellaneous including haematological, metabolic, and systemic disorders.[4] Classifications 1, 3, and 4 are 'precapillary' and demonstrate a low or normal pulmonary capillary wedge pressure (a surrogate measure of left atrial pressure).

In PH, remodelling of the pulmonary vasculature, hypoxic vasoconstriction, and disruption and fibrosis of small pulmonary vessels results in a steady increase in PVR. Increased afterload and disruption of neuroendocrine and autocrine signals leads to cardiomyocyte loss, myocardial ischaemia, and remodelling. Initially, this increased afterload can be compensated for by RV hypertrophy, but eventually, the ventricle can no longer compensate and dilatation and dysfunction occur. The ability of the right ventricle to adapt to pressure overload determines prognosis in PH; the presence of a dilated right ventricle is predictive of poor survival.[5]

It can be challenging and complex to manage patients with PH in critical care; pulmonary vasodilators, which are used less commonly in general ICUs, may be required and early advice from a centre with special expertise in PH should be considered.[6] An in-depth review of the critical care management of patients with PH has been published recently.[6]

Special attention should be given to patients with pre-existing PH undergoing surgery, as they are at high risk of deterioration. The chosen anaesthetic technique should aim to prevent further iatrogenic increases in PAP by preventing hypoxia, hypercapnia, and acidosis. The use of PEEP, intraoperative patient positioning, and pneumoperitoneum can all increase afterload. Intraoperative invasive monitoring is likely to be required and the use of transoesophageal echocardiography and CO monitoring devices may be appropriate. Neuraxial techniques can be used; however, the risk of cardiovascular instability with sympathetic blockade must be considered. These patients are likely to require critical care after surgery with a high risk of postoperative RVF. Depending on the nature and urgency of the surgery, referral to a PH centre for advice may be appropriate.

Left ventricular failure

The commonest cause of PH is left-sided heart failure, which includes LV systolic dysfunction, LV diastolic dysfunction, and left-sided valvular heart disease.[4] LV failure is classified as postcapillary PH (class II) and is associated with a high pulmonary capillary wedge pressure.[4] In this instance, PH occurs as a result of passive backward transmission of raised left-sided filling pressures. Associated RV dysfunction often goes unrecognised despite a prevalence of around one-fifth of patients with LV failure.[7] RVF is an important independent predictor of survival in patients with LV failure.[7] As with all cases of RVF, monitoring of both the left and right ventricles with echocardiography is essential. In patients with RVF as a result of raised left-sided filling pressures, there may be benefit from strategies used in the management of LV failure, including revascularisation, diuresis, beta blockers and ACE inhibitors. In some patients awaiting surgery, it may be beneficial to delay surgery in order to optimise medical management first.

Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is the most common cause of cor pulmonale and leads to an increase in RV afterload secondary to changes in pulmonary vascular structure and mechanics, and lung hyperinflation. Patients with COPD who subsequently develop RV dysfunction have an increased risk of admission to hospital and mortality.[8]

It is often not appreciated that these changes are not just limited to those with severe lung disease; studies demonstrate remodelling of the pulmonary vasculature occurs in those with mild disease and in patients who smoke with normal lung function.[9] As such, there is good evidence to show that patients with mild to moderate COPD (well within the realms

of lung function seen in patients presenting for a wide range of elective surgeries) have exercise limitation resulting from cardiac (i.e. RV) rather than respiratory insufficiency. Arguably, patients with COPD should be considered 'at risk' of RV dysfunction during the perioperative period and may be less tolerant of the stresses of mechanical ventilation and critical illness. The development of disproportionate haemodynamic collapse after intubation in the patient with COPD or 'single organ' postoperative respiratory failure are classic examples of this.

Pulmonary embolism

PE is the most common cause of acute RV dysfunction; assessment of RV function is essential, as mortality is directly related to the extent of RVF.[10] Mechanical obstruction of pulmonary vessels results in acutely increased RV afterload. Acute dilatation and stretching of the RV muscle occurs when more than 30% of the pulmonary vasculature is blocked, thus increasing RV wall tension with an increasing oxygen demand whilst simultaneously decreasing perfusion.[11] In most cases of PE, as resolution of the embolus occurs over time, PAP declines and RV function returns to normal.

Chronic thromboembolic PH is a rare complication of PE and occurs as a result of incomplete resolution of the clot with the formation of a fibrotic and flow-limiting thrombus within the pulmonary vasculature. These changes lead to an increase in afterload with remodelling of both the pulmonary vasculature and the right ventricle. Pulmonary endarterectomy may be appropriate for some patients; however, RV dysfunction may persist after surgery as a result of the extent of RV remodelling which has taken place.[12]

Acute respiratory distress syndrome

Acute cor pulmonale has been reported in 20–25% of patients with ARDS with good evidence to suggest that RV dysfunction is independently associated with poor outcome.[13] A high index of suspicion is therefore required for the potential diagnosis of PH and RV dysfunction in those with ARDS as the clinical signs are often non-specific.

ARDS causes direct injury to the pulmonary circulation caused by hypoxic vasoconstriction, extrinsic vascular compression as a result of interstitial oedema, vasoconstrictor mediator release, and blood vessel remodelling. Endothelial dysfunction is a common feature alongside mechanical obstruction as a consequence of thromboemboli, particularly in the larger pulmonary arteries, veins, and lymphatics, whilst smaller vessels become occluded by neutrophils and platelets.[14] This leads to an increase in PVR, RV afterload, and the development of PH and RVF. In patients with ARDS, these effects are magnified as airway pressures increase in combination with the pulmonary vasoconstrictive effects of hypoxia and hypercapnia.

It is increasingly being recognised that modern ventilatory practices with low tidal volumes and high PEEP have potentially adverse effects on RV function, with some experts recommending an 'RV protective' approach to mechanical ventilation in ARDS, whereby PEEP is manipulated in parallel with echocardiographic assessment of the right ventricle. This 'RV protective' strategy focuses on limiting plateau pressure to reduce lung stress, improving oxygenation to limit the effect of hypoxic vasoconstriction, and preventing hypercapnia.[15] Tidal volume and PEEP should aim to maintain a plateau pressure <27 cmH_2O, a driving pressure <18 cmH_2O and ideally a P_{ACO_2} <48 mmHg.[15] If the ventilatory frequency is adjusted, this should be done carefully as this may induce intrinsic PEEP and dynamic hyperinflation, worsening RV dysfunction. Ventilation in the prone position has been shown to induce alveolar recruitment and reduce RV afterload.[16]

Obesity and obstructive sleep apnoea

Obesity is an independent risk factor for cardiovascular disease and has been shown to cause a wide spectrum of cardiovascular changes which can ultimately result in heart failure. Obesity can lead to RV dysfunction as a result of increased CO, obesity-hypoventilation syndrome, and obstructive sleep apnoea (OSA). Studies have shown the presence of RV dilatation, increased right ventricle wall thickness, and subclinical RV diastolic and systolic impairment may be present in young obese patients with no pre-existing cardiovascular disease.[17]

In OSA, hypoxic pulmonary vasoconstriction occurs during apnoeic episodes leading to remodelling of the pulmonary microcirculation increasing PVR, and ultimately leading to the development of PH and subsequent RV dysfunction.[18] Negative intrathoracic pressure, occurring during inspiration against an occluded pharynx, increases venous return, and RV preload.[18] Coexisting cardiovascular disease and hypoxaemia may also play an important role in RV dysfunction in these patients. Imaging of the right ventricle by TTE can be challenging and more advanced techniques may be needed.

Careful attention to the provision of mechanical ventilation is also required to prevent both atelectasis and further RV dysfunction; this may require a similar balanced approach to that recommended for patients with ARDS.

Right ventricular myocardial infarction (RVMI)

As the right ventricle is supplied by the right coronary artery in 80% of the population, right coronary artery occlusion can lead to significant RV ischaemia. Generally, the right ventricle is considered to be more tolerant of ischaemia compared with the left ventricle, because coronary perfusion occurs throughout the cardiac cycle, and myocardial oxygen demand is comparatively lower.[19] However, patients with an RVMI have an increased risk of tachyarrhythmias, cardiogenic shock and death, because of interruption in blood flow to the atrioventricular node and the lack of collateral blood supply in the right ventricle. Prognosis in RVMI depends on the location of the MI, the presence of complications, pre-existing RV dysfunction, and successful coronary reperfusion. Similar to left ventricular myocardial infarction, systolic function in patients who survive RVMI may recover after the initial ischaemic episode as 'stunning' resolves.

Congenital heart disease

Several different types of congenital heart disease are associated with RV dysfunction. Both atrial and ventricular septal defects with left-to-right shunt and tricuspid and pulmonary regurgitation may lead to volume-overloaded RVF. Chronically increased pulmonary blood flow can occur in large septal defects leading to pulmonary endothelial damage, thrombosis, RV hypertrophy, and pulmonary vascular remodelling. If unchecked, progressive hypertrophy and increasing right-

sided systolic pressure result in 'suprasystemic' right-sided pressures and reversal of the initially left-to-right shunt. This results in right-to-left shunting and consequent systemic hypoxemia as seen in Eisenmenger's syndrome.[19 20]

Pressure-overloaded RVF occurs with pulmonary stenosis, RV outflow tract obstruction after correction of tetralogy of Fallot, and repair of transposition of the great arteries. Whilst the right ventricle is initially able to adapt, longstanding obstruction if untreated leads to progressive TR and RV dilation and failure.[20] Over time, pulmonary regurgitation leads to progressive RV dilatation necessitating timely pulmonary valve (PV) replacement before RV dysfunction ensues.[20] Adult patients with tetralogy of Fallot repaired in childhood are becoming an increasingly large cohort in congenital cardiac centres.

Cardiac surgery

A degree of RV dysfunction is commonplace after cardiac surgery; however, the underlying aetiology is not clear. Besides the effects of myocardial stunning resulting from direct myocardial ischaemia during cardiopulmonary bypass, the right ventricle is particularly susceptible to postoperative dysfunction because of air embolisation (to which the right ventricle is particularly predisposed as a result of the anterior location of the right coronary ostia), and increased PVR after operation. Where systemic vasodilatation is common after cardiopulmonary bypass, PVR commonly increases as a result of the activation of inflammatory mediators, the accumulation of extravascular lung water, persistent pulmonary derecruitment and protamine-induced PH.

PH, RV dilatation and dysfunction, and functional TR are common sequelae of mitral valve disease. At the time of mitral valve surgery, the tricuspid valve may also be repaired. Careful attention to the right ventricle after tricuspid valve surgery is paramount. The presence of TR can lead to overestimation of baseline RV function before surgery, as the latter is 'flattered' by the presence of TR (i.e. the ease of emptying when blood may flow in an anterograde or retrograde direction can make the function 'look' better than it is). Repair of the valve can then lead to an effective increase in afterload, which may unmask impaired RV function.

Left ventricular assist devices

Left ventricular assist devices (LVADs) are used as a bridge to transplant or recovery in patients with severe heart failure. An inlet cannula is inserted into the left atrium (LA) or LV cavity, whilst an outflow graft is attached to the ascending aorta providing isolated support to the left side of the heart. Adequacy of RV function is a major determinant of outcome after LVAD insertion. In comparison to the low CO state encountered before the operation, the right ventricle is challenged after LVAD implantation by the need to match the increased LV output, which leads to an effective increase in preload. In addition, unloading of the LV by a VAD can alter the shape and size of the right ventricle leading to a direct effect on function.[21]

Heart transplantation

Despite advances in the perioperative management of heart transplantation, acute RVF still accounts for a significant number of complications and early deaths. Whilst the aetiology of RVF after transplantation is multifactorial, two predominant mechanisms are described. Firstly, an ischaemic insult to the right ventricle may occur as a result of prolonged ischaemic time and suboptimal myocardial protection, leading directly to primary graft failure. Secondly, the presence of pre-existing or acquired PH in the recipient can result in the exposure of the previously 'afterload-naive' (and therefore not adapted) transplanted organ to acutely increased afterload.

Management

The management of RVF should naturally aim to identify and treat any underlying aetiology. Generic treatment goals include optimising rate, rhythm, perfusion, preload, augmenting myocardial contractility, and minimising afterload (Fig. 1). This requires a careful balance of therapy to ensure cardiac filling combined with vasopressor and inotropic support. In rare cases, surgical management in the form of mechanical circulatory support may be required.

Optimise rate and rhythm

In general, it is considered preferable to keep the right ventricle beating faster and where possible in sinus rhythm (SR). A relatively high HR (it is not uncommon to pace the immediately post-transplanted heart at 110 bpm), prevents excessive RV distension and subsequent distortion of the left ventricle and minimises TR. Additionally, in conditions of failure where stroke volume may be limited, a higher HR promotes CO. Clinicians should always be aware however of the effects of tachycardia on the LV blood supply and on its function.

In theory, preservation of SR offers significant haemodynamic benefits, improving ventricular preload and atrial emptying and reducing atrial pressures. In many patients, non-SR is a chronic situation however; attempts to restore SR through chemical or electrical cardioversion should therefore be reserved for circumstances where acute or paroxysmal arrhythmias are causing or exacerbating a patient's condition.

Maintaining perfusion

The importance of maintaining systemic arterial pressure and right coronary perfusion pressure is often underappreciated in RV dysfunction, where systemic hypotension can be detrimental to the failing right ventricle. Vasoconstrictor agents, such as noradrenaline or vasopressin (Table 1), are often required to maintain systemic arterial pressure, indirectly resulting in improved RV function and global tissue perfusion.

Intra-aortic balloon pump (IABP) counter pulsation is a commonly used circulatory assist device that improves coronary and systemic blood flow.[22] Whilst these devices are generally considered to provide left-sided support, improvement of right coronary perfusion can aid RV function. The IABP inflates and deflates in harmony with the cardiac cycle; as diastolic aortic pressure is augmented, coronary perfusion pressure is increased, improving coronary blood flow and increasing myocardial oxygen supply.

Optimise preload

Optimising RV preload is important in patients with RV dysfunction; even a functionally impaired ventricle has an

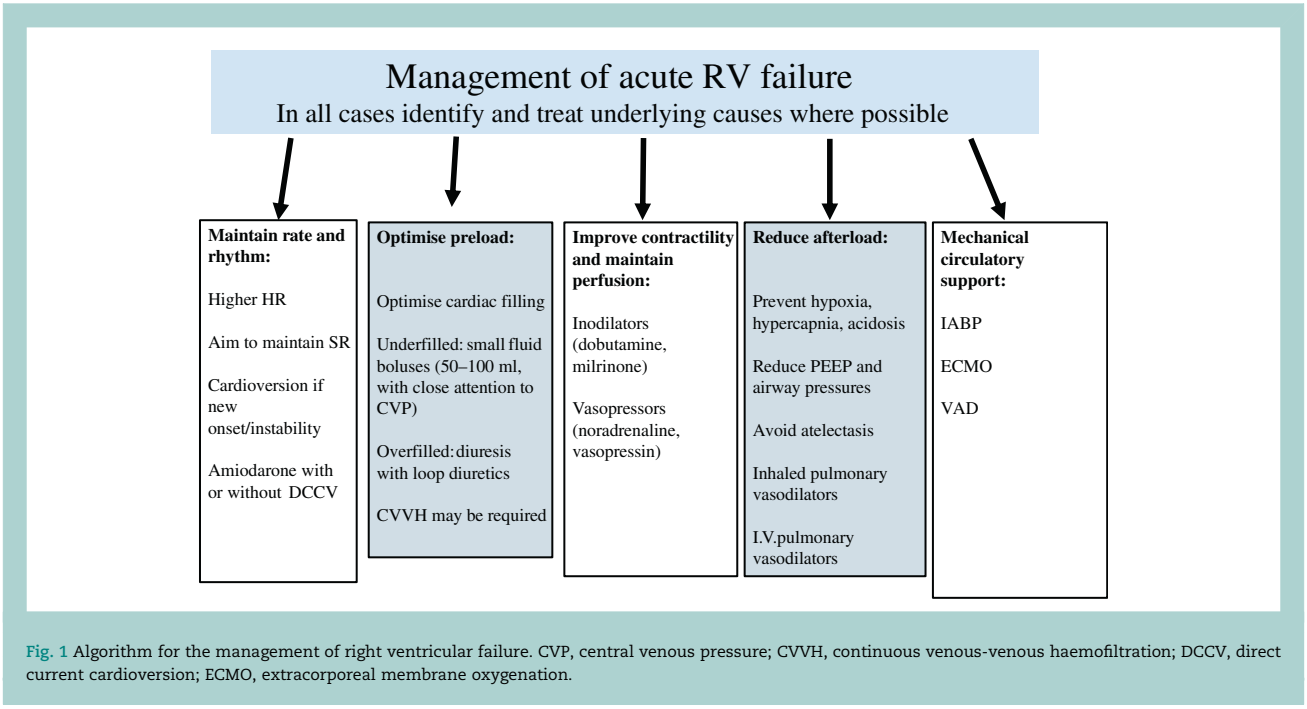

Fig. 1 Algorithm for the management of right ventricular failure. CVP, central venous pressure; CVVH, continuous venous-venous haemofiltration; DCCV, direct current cardioversion; ECMO, extracorporeal membrane oxygenation.

optimal level of preload. Given the potential for deleterious effects of overdistension, however, it is common practice to assess fluid responsiveness in patients with RV dysfunction by using small volume fluid boluses (50–100 ml) whilst paying close attention to central venous pressure. In patients with RV dilatation, any increase in intravascular volume can worsen dilatation and further impair LV diastolic filling. In the dilated right ventricle, marked diuresis is often warranted to offload the ventricle and reduce right-sided filling pressures.

Exaggerated, ventilation-induced (i.e. in response to cyclically varying afterload) changes in SV and CO are common in the failing right ventricle and can easily be misinterpreted as signs of 'fluid responsiveness', rendering monitors assessing SV or systolic pressure variation less useful in this context. Often, the only reliable method of assessing preload responsiveness is the judicious administration of a fluid bolus and observing the net change in SV or CO using an appropriate CO monitor.

Reduce afterload

Minimising afterload is a key component of RV management, though many patients presenting with RVF have chronic lung or cardiac diseases and consequently PH that cannot easily be reversed. Iatrogenic increases in PVR should be avoided by careful avoidance of hypoxia, hypercapnia, and acidosis. When ventilating patients with RVF, airway pressures should be minimised and PEEP used judiciously; both atelectasis and excessive PEEP will increase PVR (Fig. 2). Again, these goals appear in conflict to those used in patients with ARDS, where permissive hypercapnia and higher levels of PEEP are commonplace.

I.V. or inhaled pulmonary vasodilators may be used to reduce afterload by targeting pathways implicated in PH. I.V. pulmonary vasodilators (e.g. glyceryl trinitrate, epoprostenol, inodilators) cause indiscriminate vasodilation of the pulmonary vascular bed, blunting hypoxic pulmonary vasoconstriction, leading to a worsening of any ventilation-perfusion ($\dot{V}/\dot{Q}$) mismatch and potentially causing hypoxia. Systemic vasodilation occurs in parallel and can cause systemic hypotension.

Inhaled vasodilators, such as nitric oxide, reduce PVR only in areas of the lung that are well ventilated and so improve $\dot{V}/\dot{Q}$ matching by increasing flow to these areas, improving oxygenation and decreasing PVR. Nitric oxide has a fast onset of action with a short half-life and has been shown to improve RV systolic function, mixed venous oxygen saturations, and haemodynamics in patients with acute RVF. Nitric oxide may be of benefit in the perioperative and early postoperative period in patients with PH during cardiac surgery. There is evidence for a reduction in PAP and an improvement in hypoxia in other clinical scenarios such as ARDS, PE, and COPD; however, the evidence for an improvement in overall outcome is lacking. Disadvantages include expense, the need for toxicity monitoring, platelet inhibition, prolonged bleeding time, and the potential for rebound hypoxaemia and PH. Weaning of nitric oxide can be challenging and slow weaning over hours to days may be required to avoid vasoconstriction and rebound PH or hypoxaemia.

Oral phosphodiesterase type-5 inhibitors, such as sildenafil, reduce PVR and improve RV contractility, but caution should be exercised because of their long terminal half-life (4–18 h) and risk of systemic hypotension as a result of systemic vasodilatation. In practice, these agents tend to be used in stable disease rather than in the acute phase.

Optimise contractility

Inodilatory vasoactive drugs, such as dobutamine (a beta agonist), or milrinone (a phosphodiesterase-3 inhibitor), are a natural choice in RV dysfunction as they promote increased contractility with a simultaneous reduction in afterload

Table 1 Commonly used pharmacological agents in RVF. SVR: systemic vascular resistance. *A loading dose of milrinone may be given but increases the risk of systemic hypotension.

Drug	Classification	Dose (i.v. unless stated)	Receptor	Effects	Onset	Advantages	Disadvantages
Noradrenaline	Vasopressor	0.02–0.2 μg kg^{-1} min^{-1}	α_1, β_1	Vasoconstriction, ↑SVR, ↑myocardial O_2 delivery, ↑PVR	Quick (min)	Cheap, easy to titrate, familiarity	Arrhythmias, ↑PVR in higher doses
Vasopressin	Vasopressor	1–4 units min^{-1}	V_1, V_2	Vasoconstriction, ↑SVR, pulmonary vasodilatation at low doses via endothelial nitric oxide pathway, ↑myocardial O_2 delivery	Quick (min)	Catecholamine-sparing, less ↑PVR than noradrenaline, easy to titrate	Expensive, bradycardia, splanchnic ischaemia
Dobutamine	Inodilator	2.5–10 μg kg^{-1} min^{-1}	β_1,β_2	Inotropy, ↑contractility, ↓SVR, PVR	Quick (min)	Easy to titrate, cheap	↑O_2 demand, tachyarrhythmias, systemic hypotension
Milrinone	Inodilator	0.375–0.75 μg kg^{-1} min^{-1}*	PDE_3 inhibitor	Inotropy, ↑contractility, ↓SVR, PVR	Long half-life (2.5 h)	Pulmonary vasodilatation	Systemic hypotension, expensive
Levosimendan	Inodilator	Loading dose: 6–12 μg kg^{-1} min^{-1} over 10 min followed by infusion of 0.1 μg kg^{-1} min^{-1}	Calcium sensitiser	↑Contractility	Slow	No effect on myocardial oxygen demand	Expensive, tachycardia, hypotension, headache
Sildenafil	Pulmonary vasodilator	10 mg t.d.s Oral: 20–100 mg t.d.s.	PDE_5 inhibitor	↓PVR,↑contractility	Slow	Oral administration for patients with chronic disease	Long terminal half-life (4–18 h), ↓SVR
Epoprostenol	Pulmonary vasodilator	1–2 ng kg^{-1} min^{-1} Nebulised: 0.2–0.3 ml min^{-1} of a 10–20 μg ml^{-1} solution	Prostacyclin	↓PVR, ↑$\dot{V}/\dot{Q}$mismatch	Quick	As efficient as nitric oxide	Systemic hypotension with i.v. administration, flushing, headaches

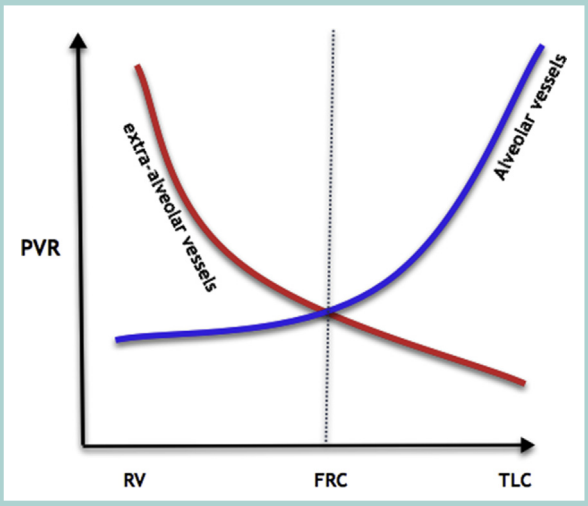

Fig. 2 Schematic representation of the relationship between lung volume and PVR. Atelectasis compresses extra-alveolar bloods vessels which increases PVR. Alveolar distension, occurring at high lung volumes, compresses intra-alveolar blood vessels which increases PVR. FRC, functional residual capacity; RV, residual volume; TLC, total lung capacity.

(Table 1). Both agents cause a reduction in systemic vascular resistance and can lead to profound systemic hypotension, especially with milrinone. As such, systemic vasoconstrictors are often required in conjunction with inodilators to counteract the adverse effects described.

Levosimendan, a calcium sensitiser, may improve coronary perfusion and contractility without increasing myocardial demand; however, its role in RVF needs further exploration.

Mechanical support

In isolated RVF refractory to medical therapy, or RVF in the context of an LV support device several options exist for mechanical circulatory support of the right ventricle. It is important to consider mechanical support early in order to prevent irreversible end organ damage.

Peripheral venoarterial (in contrast to venovenous) extracorporeal membrane oxygenation provides respiratory and biventricular support via cannulae in the right atrium (inserted via a peripheral vein) and in the subclavian or femoral artery.

Surgically implanted VADs provide isolated RV support classically via cannulae in the right atrium or ventricle and an outflow cannula in the pulmonary artery. In recent years, so-called 'percutaneously inserted' VAD devices such as the Impella RP (Abiomed, Danvers, Massachusetts, USA) and TandemHeart RVAD (LivaNova, London, UK) devices have become increasingly popular, providing isolated RV support without the need for sternotomy, by accessing the pulmonary circulation via a transvalvular systemic venous approach.

Conclusions

RV dysfunction and failure can occur as a result of a wide variety of pathophysiology and is becoming more frequent in patients presenting for general anaesthesia or to intensive care. Initial management should aim to identify potential underlying reversible causes, optimise preload and reduce afterload. Close attention should be paid to rate, rhythm and perfusion. Pharmacological management may include a combination of inotropes, vasopressors and pulmonary vasodilators.

Declaration of interest

The authors declare that they have no conflicts of interest.

MCQs

The associated MCQs (to support CME/CPD activity) will be accessible at www.bjaed.org/cme/home by subscribers to *BJA Education*.

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