Review

What role does the right side of the heart play in circulation?

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Abstract

Right ventricular failure (RVF) is an underestimated problem in intensive care. This review explores the physiology and pathophysiology of right ventricular function and the pulmonary circulation. When RVF is secondary to an acute increase in afterload, the picture is one of acute cor pulmonale, as occurs in the context of acute respiratory distress syndrome, pulmonary embolism and sepsis. RVF can also be caused by right myocardial dysfunction. Pulmonary arterial catheterization and echocardiography are discussed in terms of their roles in diagnosis and treatment. Treatments include options to reduce right ventricular afterload, specific pulmonary vasodilators and inotropes.

Introduction

The lungs are the only organs to receive the entire cardiac output. The role of the 'right side of the heart' is to accept the blood from systemic circulation and pump it through the pulmonary circulation. Even if the cardiac output from the right and left sides of the heart are approximately equal (there is a physiological shunt for the bronchial vessels), the way in which these two cardiac outputs are conducted by the ventricles is very different. Differences between the right and left sides of the heart are multiple and numerous. These differences contribute to both normal and abnormal physiology and pathophysiology. Much attention has recently been directed toward the left ventricle (LV), whereas the role played by the right ventricle (RV) has remained comparatively neglected. This is paradoxical when we consider that intensive care units (ICUs) have been using a 'right heart catheter', namely the pulmonary artery catheter (PAC), for more than 20 years. The fundamental duty of an intensivist could be simplified to ensuring that there is adequate cardiac output to systemic circulation. This is clearly a goal that should be achieved in any patient, but we should not discount the role of the pulmonary circulation in cardiac homeostasis because the left and right sides of the heart are interrelated in terms of function.

Important differences between right and left sides of the heart

Anatomy

The right atrium (RA) and the RV form the right side of the heart. The RA receives venous blood from superior and inferior venae cavae, ejecting it through the pulmonary arteries. The tricuspid and pulmonary valves prevent regurgitation into the RA and RV, respectively. The right ventricular myocardium is about one-third the thickness of its left counterpart because it is a comparatively low pressure system. During systole, the LV protrudes into the RV. These chambers are separated by the septum and are surrounded by pericardium. These basic anatomical arrangements explain why compliance of one ventricle can modify that of the other. This interaction is known as diastolic ventricular interaction and is important in understanding the pathophysiology of right heart failure.

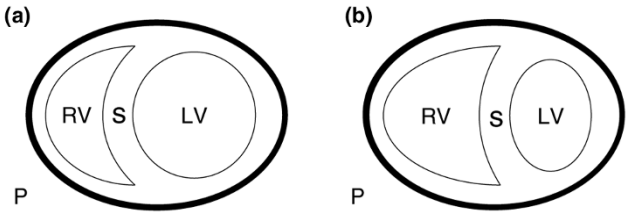
Physiology

In the average person, the systolic pressure of the RV reaches a mere 25 mmHg, which is in contrast to the 120 mmHg systolic pressure of the LV. This difference in pressure is essentially due to the discrepancy in compliance between pulmonary and systemic blood vessels, with pulmonary vessels having a relatively large diameter and thin walls (conditions that allow for the high compliance of the pulmonary system). Like any vessel in the body, the pressure in the pulmonary system depends on cardiac ouput, resistance and compliance. An increase in pulmonary arterial pressure could be due to any combination of increases in the first two factors or a decrease in the third. The pulmonary vascular resistance has a particular dependency on alveolar oxygen tension, whereby alveolar hypoxia leads to pulmonary arterial vasoconstriction.

Pulmonary compliance has a value of about 2 ml/mmHg. This allows the right heart to pump the blood with a blood

ARDS = acute respiratory distress syndrome; ICU = intensive care unit; iNO = inhaled nitric oxide; LV = left ventricle; MI = myocardial infarction; PAC = pulmonary artery catheter; PAH = pulmonary artery hypertension; PAWP = pulmonary artery wedge pressure; PE = pulmonary embolism; RA = right atrium; RCA = right coronary artery; RV = right ventricle; RVEF = right ventricular ejection fraction; RVEDVI = right ventricular end-diastolic volume index; RVF = right ventricular failure.

Figure 1



Leftward movement of the interventricular septum. **(a)** Position of the interventricular septum during systole in normal conditions. **(b)** Dilatation of the right ventricle, which moves the septum over into the cavity of the left ventricle at the end of prolonged right systole. The pericardium surrounds the heart and does not allow excessive dilatation of the right ventricle without displacement of the interventricular septum. LV, left ventricle; P, pericardium; RV, right ventricle; S, septum.

pressure lower than the systemic arterial pressure, ensuring that the pulmonary circulation is a 'low pressure system' [1]. Being a pump that works under low pressure conditions, the RV is affected by an increase in afterload [2,3], even when the oxygen consumption in the right heart is accounted for. The RV is mainly perfused by the right coronary artery (RCA) and partially by the left coronary arteries. The RCA is perfused in both systolic and diastolic phases in the healthy human as a result of the low systolic pressure (25 mmHg), which does not occlude the vessel that has a systemic pressure. When afterload is high, and therefore the pressure necessary to contract the RV successfully is also high, partial occlusion of the RCA may occur, leading to ischaemia.

Pathophysiology of right ventricular failure

Increased afterload

In ICUs there are many conditions that can lead to an increase in right ventricular afterload. These include pulmonary embolism (PE), positive pressure ventilation, acute respiratory distress syndrome (ARDS) and increased pulmonary vascular resistance (especially after cardiac surgery or in hypoventilatory states).

When there is an increase in right ventricular afterload, right ventricular systole takes longer, even if the ejection time is reduced. This elongated systole can last longer than left ventricular systole. Because the RV is still contracting as the left begins to relax, the septum displaces to the left while the LV starts diastole and is moved back rightward when left systole begins again. In summary, the net result is an abnormal reversal of the left-to-right pressure gradient at end-systole/onset of diastole and an altered cardiac performance (Figure 1) [4]. Septal dyskinesia seen at echocardiography is considered a sign of increased right ventricular afterload. This mechanism involves the diastolic ventricular interaction.

Dilatation of right heart is a compensatory mechanism that allows the RV to maintain stroke volume despite a decreased ejection fraction [5,6]. When this enlargement is acute and due to a sudden increase in afterload, it is termed acute cor pulmonale. The two main causes are massive PE and ARDS [7-9]. More importantly, when the underlying cause of acute cor pulmonale is treated, the situation reverses [5]. If intervention does not occur, enlargement of the RV can lead to tricuspid annular dilatation with regurgitation, an increase in right ventricular end-diastolic volume and leftward movement of the septum, with final impairment of the LV [10]. This progression can be rapid because the RV has a peculiar auto-aggravation cycle. Tricuspid insufficiency can aggravate splanchnic congestion, resulting in decreased venous return and therefore preload. The cardiac output, already compromised because of the low preload state of the LV induced by the right ventricular failure (RVF), decreases further. Consequential systemic hypotension exacerbates the impairment in organ perfusion and coronary artery perfusion. Cardiac ischaemia (commonly the next pathophysiological observation) is characterized by further aggravation of cardiac performance and its cycle.

Pulmonary embolism

Acute PE represents an example of the aetiology of increased right ventricular afterload. In massive embolism blood flow is obstructed, resulting in a low cardiac output with systemic hypotension. Jardin and coworkers [11] reported a study conducted in 14 patients with massive PE. In these patients, during the acute phase both the RA and the RV showed signs of hypertrophy and elevated right atrial pressure, with reversal of trans-septal diastolic pressure gradient. More importantly, the LV was impeded by the leftward movement of the septum and had a reduction in left ventricular diastolic area. As described by Morris-Thurgood and Frenneaux [12], further volume loading in this case can have detrimental effects enhancing diastolic ventricular interaction. Belenkie and colleagues [13] studied volume loading in a canine model, before and after pulmonary embolization. After embolization, volume loading moved the septum leftward and led to reduced left ventricular area index and left ventricular stroke work. Different findings were reported by Mercat and coworkers [14]; they evaluated 30 patients with acute massive PE and found that fluid loading (500 ml dextran 40) improved cardiac output. The latter study highlighted the patients who improved the most after the fluid loading, namely those who had a lower right atrial pressure at baseline, which suggests that when an interventricular dependency phenomenon was present there was a lesser response to fluid loading. It is possibly untrue that fluid loading should be avoided in the case of right ventricular failure, but the fluid management should only correct hypovolaemia. Clinicians must be aware of the consequences of excessive fluid loading, namely impairment in left ventricular (LV) performance and worsening of the 'auto-aggravation cycle'.

Mechanical ventilation

Intermittent positive pressure ventilation is often used alongside treatments to optimize left ventricular function, with benefits in terms of reductions in both preload and afterload. The RV has a different response to intermittent positive

pressure ventilation; the preload decreases but pulmonary vascular resistance and consequently afterload increase. This is further accentuated with positive end-expiratory pressure. If intermittent positive pressure ventilation is necessary in the setting of RVF, then high levels of plateau pressure and positive end-expiratory pressure should be avoided to minimize the right heart–lung interaction. For the same reason, high respiratory rates, which can lead to air trapping and intrinsic positive end-expiratory pressure, should be avoided. The minimal level of plateau pressure, mean airway pressure and positive end-expiratory pressure necessary for pulmonary recruitment should be maintained.

Acute respiratory distress syndrome

In ARDS two factors contribute to right heart failure. First, increased pulmonary vascular resistance occurs as a result of the distal occlusion of the pulmonary arterial bed. Second, most patients require mechanical ventilation to maintain adequate oxygenation and, as described above, intermittent positive pressure ventilation can worsen RVF. The incidence of acute cor pulmonale in ARDS is about 25% [8] if a protective ventilation strategy (low tidal volume/low plateau pressure) is used. A study conducted in 1985, when high tidal volume was used (≥13 ml/kg), identified an incidence of acute cor pulmonale in ARDS of 65% [4,15].

Hypoventilation state

With respiratory failure, as occurs in patients with chronic obstructive pulmonary disease, some areas of the lungs are hypoventilated. This stimulates the pulmonary vasoconstrictor reflex, thereby increasing pulmonary vascular resistance. Concomitant hypercapnia and acidosis are often present, and this worsens both vascular resistance and ventricular contractility. In these situations, acidosis should be corrected as well as hypercapnia.

Sepsis

Right ventricular dysfunction, as demonstrated with radionuclide angiography and echocardiography, is common in sepsis (up to 32%) [4,16,17]. RVF should be kept in mind in all septic patients who do not respond to volume expansion.

Reduced right ventricular contractility

Chronic heart failure

The response to chronic increased afterload of the RV is characterized by an initial dilatation and then by tricuspid regurgitation and low cardiac output. In recent years a number of studies have supported right ventricular performance being an important prognostic determinant in chronic heart failure [2] and, most importantly, right ventricular ejection fraction (RVEF) and pulmonary arterial pressure are independent predictors of survival [18].

Right ventricular myocardial infarction

There is a high prevalence of single vessel coronary disease in myocardial infarction (MI) of the RV compared with MI of the LV. When shock is present in patients with MI of the RV, the mortality is high and similar to that in patients with cardiogenic shock from infarction of the LV [19]. The aetiology of such MIs can be related back to an occlusion of the proximal part of the RCA. This leads to depressed right ventricular function and decreased left ventricular preload. Cardiac output is low and systemic hypotension appears as a consequence, even if left ventricular function is normal [20]. Right heart failure can be exacerbated if the posterior portions of the interventricular septum are also involved, because the septum plays an important role in right ventricular contraction [4,21,22].

Pulmonary arterial hypertension is usually absent but MIs have been reported to occur more frequently when right ventricular hypertrophy (which can be consequent to sustained increased afterload) is present. Indeed, many RCA occlusions do not lead to MI. The simple explanation for this is that myocardial hypertrophy increases oxygen demand and thereby leads to more ischaemic damage [10].

Assessment devices

Because the clinical presentation of right heart failure is very nonspecific, it is difficult to make a diagnosis of RVF by clinical examination alone. In the ICU scenario there are essentially two ways to assess right-sided function: pulmonary artery catheterization and echocardiography. The latter can be divided into transthoracic and transoesophageal methods. Magnetic resonance and radionuclide ventriculography are two other valid methods of assessment, but because their use at the bedside is difficult we do not consider them here. Electrocardiography and troponin assessment are also useful when MI is suspected.

Pulmonary artery catheter

Pulmonary arterial catheterization is the most invasive and oldest method of assessing right-sided cardiac function. It is the only available continuous monitoring device that permits evaluation of the efficacy of treatment. In fact, it permits continuous monitoring of right atrial pressure, right ventricular pressure, pulmonary arterial pressure, pulmonary artery wedge pressure (PAWP), continuous cardiac output and mixed venous oxygen saturation. Modern PACs can also be used to measure RVEF and right ventricular end-diastolic volume [23-25]. The diagnosis of RVF is made in situations of systemic hypotension, decreased cardiac output and low mixed venous saturation, and when there is a high right atrial pressure that is higher than a usually normal PAWP. Diagnosis of pulmonary artery hypertension (PAH) is easily made by assessing pulmonary pressure. Fluid loading in RVF can be dangerous.

New catheters permit continuous measurement of right ventricular end-diastolic volume index (RVEDVI) and RVEF. RVEDVI is a novel preload index. Interestingly, it seems that better understanding and use of RVEDVI result from its

Table 1

Haemodynamic and arterial pressure responses to volume challenge and dobutamine

Parameter	Baseline	Response to volume challenge	Response to dobutamine (5 μg/kg per min)
MAP	83	80	74
RAP	12	16	13
MPAP	18	28	16
PAWP	12	12	11
CI	1.8	1.9	1.8
RVEF	33	25	38
RVEDVI	145	190	163
Svo_2	54	52	60
Lactate	3.1	3.5	3.2

These measurements are from a 67-year-old critically ill postoperative patient. See text for further details. CI, cardiac index; MAP, mean arterial pressure; MPAP, mean pulmonary arterial pressure; PAWP, pulmonary artery wedge pressure; RAP, right atrial pressure; RVEDVI, right ventricular end-diastolic volume index; RVEF, right ventricular ejection fraction; Svo_2, mixed venous oxygen saturation.

consideration together with RVEF, a parameter that can define the contractility status of the RV. Indeed, as Sarnoff and Berglund [3] reported, contractility is derived from a series of ventricular function curves, with each curve having a particular ejection fraction and optimal preload (end-diastolic volume). Thus, it appears that correct interpretation of RVEDVI is strongly dependent on RVEF [26]. Currently, there is little evidence to support routine use of RVEDVI and RVEF in RVF.

Table 1 shows how RVEDVI and RVEF can be used to guide fluid and drug management. In this clinical example, a patient monitored with a PAC was given fluids and dobutamine. After the volume challenge the CI did not improve and the RVEDV increased. The RVEF decreased and the mean pulmonary artery pressure increased from 18 to 28 mmHg, indicating that the patient was not responding to volume expansion. The mixed venous oxygen saturation did not change and lactate level increased. Consequent administration of dobutamine without further volume expansion increased RVEF from 25% to 38%. At the same time, mean pulmonary artery pressure decreased from 28 to 16 mmHg. Although the CI does not improve, the improvement in cardiac performance is reflected by decreases in mean pulmonary artery pressure and lactate levels, and increases in RVEF and mixed venous oxygen saturation.

One of the primary aims in the setting of acute RVF is to achieve a reduction in right ventricular afterload. The best way to gauge the effectiveness of this treatment strategy is to monitor the patient continuously. This can only be done using the PAC.

Echocardiography

The role of echocardiography has expanded in recent years. The most powerful characteristic of this technique is that it permits immediate qualitative diagnosis of heart disease, simultaneously allowing for estimation of flows and pressures. In order to explore the right side of the heart, transthoracic echocardiography can be used as long as the patient remains unventilated. When mechanical ventilation is being used, a transoesophageal approach should be used, resulting in better imaging. Diagnosis of RVF is made qualitatively and, as emphasized by Jardin and Viellard-Baron [4], septal dyskinesia is the hallmark of RVF. Transoesophageal echocardiography may confirm an initial suspicion of RVF (high central venous pressure) if absence of the respiratory variation in vena cava diameter is detected. Both transthoracic and transoesophageal echocardiography can be used to estimate pulmonary pressures. The qualitative aspect of echocardiography allows a diagnosis of RVF to be made along with recognition of its cause. It is possible to recognize RVF secondary to a PE, ARDS, or coronary obstruction simply by analyzing the different recorded images [27]. Furthermore, echocardiography can assess both the right and left sides of the heart, and can allow measurements to be taken in systolic and diastolic phases that can be repeated, allowing intermittent monitoring of the efficacy of treatment.

Treatment strategies (with an emphasis on monitoring techniques)

The aim of treatment for right heart failure is to achieve optimal 'contraction status' by breaking the auto-aggravation cycle. A decrease in afterload should always be sought and an adequate preload should be maintained with appropriate fluid administration. Although overload should be avoided, hypovolaemia is unacceptable. Inotropes can be used to optimize ventricular contraction. Afterload can be decreased with the use of drugs and by minimizing the effects of ventilation on the heart. While providing these supportive therapies, definitive therapies such as coronary reperfusion should be delivered.

Management of myocardial infarction

Reperfusion of the occluded coronary is beneficial and should be a primary goal in proven right-sided infarction [28,29]. The goals suggested by Pfisterer [20] for right ventricular infarction are as follows: early recognition, early reperfusion, maintenance of adequate preload, reduction in right ventricular afterload (inotropes, balloon counterpulsation), preservation of right ventricular synchrony and avoidance of vasodilators, nitrates, morphine and β-blockers. It is very important that the left ventricular pressure be kept as close to normal as possible; this can help to restore the normal position of the interventricular septum and thereby improve contraction of the RV [10].

Fluid therapy

Because of the interventricular dependence phenomenon, volume overload can dilate the RV so much that it may impair left ventricular function and the entire circulation. Conversely, adequate fluid loading is necessary to achieve optimal contraction. So what should happen at the bedside in a patient with RVF? In the ICU fluid therapy is often guided by fluid challenges. Mercat and coworkers [14] discovered that, in acute PE, fluid challenges improve cardiac output, especially in patients with low baseline atrial pressure, who probably represent those in whom interventricular dependence is not yet playing a substantial role. Using mean pulmonary arterial pressure as a guide to fluid challenge, it seemed reasonable to stop filling beyond a mean pulmonary artery pressure of 30 mmHg [5,30]. This is supported by findings reported by Atherton and coworkers [31], even though their population included patients with chronic heart failure. In that study the investigators used a device that results in acute volume unloading by generating lower body suction of 30 mmHg, and they measured how right and left volumes changed. Interestingly, among the patients with heart failure, those with an increased left ventricular end-diastolic volume after the volume unloading were also those with higher right resting atrial pressures, mean pulmonary arterial pressure, and PAWP.

During the past few years a new way to assess fluid responsiveness in ventilated patients has become popular. Pulse pressure variation, evaluated by a simple analysis of the arterial pressure wave, has been proven to identify patients who may benefit from fluid challenge. In the setting of the patient with RVF undergoing mechanical ventilation, pulse pressure variation can be high not because of hypovolaemia alone but also because of ventricular impairment exacerbated by the mechanical ventilation [27,32].

Therefore, if a fluid challenge is followed by an increase in filling pressure without change in cardiac output, then fluid loading should be stopped. PAC is a valuable tool in this context because it allows the clinician to assess pulmonary pressure and CI on a continuous basis. The efficacy of fluid challenge can be monitored by watching changes in CI, SVI, RVEDVI and RVEF. Fluid removal with diuretics or haemofiltration may then be beneficial [5]. However, fluid withdrawal is effected, it is important that cardiac output monitoring be used to assess the efficacy and safety of treatment.

Drugs

Drugs are employed either to decrease pulmonary vascular resistance or to increase cardiac contractility.

Vasodilators

Inhaled nitric oxide (iNO) is a vasodilator that can be given to ventilated patients. It causes vasodilatation by increasing levels of cGMP in vascular smooth muscle cells. cGMP has a short half-life because it is degraded by endogenous phosphodiesterases. On entering the pulmonary circulation, iNO binds with plasma proteins and is immediately inactivated by haemoglobin. In this way its effects are localized solely to the pulmonary vasculature [33]. Moreover, its effect is to vasodilate only ventilated areas of the lungs, maintaining an efficient ventilation/perfusion ratio. Indeed, systemic administration of vasodilators such as nitroglycerine or prostaglandin E_1 can improve RVF, but by being unselective for ventilated areas these agents can worsen the ventilation/perfusion ratio [34,35]. iNO is considered the 'gold standard' in the management of PAH. Its role has been studied in primary PAH, and in this way it has been used to identify patients who would benefit from long-term treatment with nifedipine [36]. In acute RVF its ability to lower pulmonary artery resistance and pressure has been proved, even though it has not been demonstrated to decrease mortality in those patients who respond to the therapy [37]. iNO can be associated with a rebound increase in pulmonary arterial pressure, especially when its use is prolonged and its withdrawal sudden [38]. Its role is more controversial when we consider its effect on the LV. Indeed, some reports indicate that a decrease in cardiac output and an increase in PAWP may occur during iNO therapy for left ventricular impairment [39,40]. On the other hand, in a small study in patients with severe isolated right ventricular dysfunction [37] it was found that high concentrations of iNO (35-40 parts/million) not only decrease afterload but also increase cardiac output. Considering all the published studies, iNO appears to be a rapid acting and effective treatment in acute RVF.

Prostacyclin (prostaglandin I_2) is both a vasodilator and a potent inhibitor of platelet aggregation. It can be administered systemically or locally as inhaled prostacyclin as an alternative to iNO [5]. The preferred method of administration is inhalation for two reasons: first, it vasodilates ventilated areas of lung; and second, no PAH rebound on finishing treatment has been described. Systemic prostacyclin is available for administration by intravenous [41], subcutaneous [42], and oral [43] routes. Iloprost is a prostacyclin derivate molecule (carbacyclin). In comparison with prostacyclin and iNO, it has a longer half-life and its effects persist for 1 hour after administration [5].

Sildenafil is an inhibitor of phosphodiesterases that was initially used in the management of erectile dysfunction. Its effect is greater on pulmonary than on the systemic circulation because it inhibits phosphodiesterase-5 more than its analogous isoforms. This particular enzyme is expressed mainly in pulmonary vessels and in the corpora cavernosa. Oral administration has proven to reduce hypoxia-induced PAH. In cardiac surgery sildenafil has been used to wean patients from inhaled iNO [44]. Its synergistic effects both with iNO and iloprost have been demonstrated.

Interestingly, Ghofrani and coworkers [45] found that oral sildenafil but not intravenous prostacyclin induced pulmonary

vasodilatation in patients with pulmonary fibrosis, thereby maintaining an optimal ventilation/perfusion ratio. Combinations of sildenafil and iNO can be used to achieve effects on PAH better than those with administration of a single drug [46].

Inotropes

Selective right heart inotropes do not exist. Contractility can be manipulated with the use of inotropes that are usually used in the management of left ventricular failure. These drugs act on the entire myocardium, improving both right and left ventricular contractility. In fact, increased left ventricular contraction can, in turn, facilitate right ventricular contraction. In the ICU β-mimetics such as dobutamine can be used to promote myocardial contraction and to improve cardiac output. Indeed, phosphodiesterase inhibitors and a new class of drugs, namely the calcium sensitizers, can also be used. Levosimendan is the first calcium sensitizer to be employed in clinical practice. In animal models, in comparison with milrinone, levosimendan was reported to increase cardiac output without increasing intracellular calcium levels, and so without increasing oxygen demand [47]. Moreover, the LIDO trial reported lower mortality at 6 months for levosimendan in comparison with dobutamine in low output cardiac failure [48]. More important in the context of right heart failure is that it acts as a vasodilator by opening ATP-sensitive potassium channels. It decreases pulmonary capillary wedge pressure, pulmonary artery pressure and mean right atrial pressure. Compared with dobutamine, levosimendan produces a similar increase in cardiac output but profoundly greater decreases in PAWP [49]. However, it is not a selective pulmonary vasodilator, and can lead to systemic hypotension. Whatever inotrope is used, cardiac output monitoring is essential for guiding therapy. The PAC can provide the clinician with information on both cardiac output and pulmonary pressure changes during inotrope use.

Systemic vasoconstrictors

It is not easy to arrive at a clinical diagnosis of RVF. The first clinical sign is commonly systemic hypotension. In the ICU this is usually treated with fluid administration and infusion of a vasoconstrictor. However, caution is needed because vasoconstrictors can also have a detrimental effect on the pulmonary vasculature by increasing pulmonary vascular resistance. In clinical practice, norepinephrine (noradrenaline) increases mean arterial pressure and this in turn improves coronary perfusion, as well as left and right ventricular contraction. Animal studies demonstrated the effect of norepinephrine on pulmonary resistance during heart failure [50,51] and revealed that norepinephrine restores mean arterial pressure and improves cardiac output. Right ventricular impairment can persist for some time after resolution of PAH [52]. In one model of RVF, after elimination of PAH, dobutamine was shown to be better than noradrenaline in restoring both cardiac output and mean arterial pressure [51].

Conclusion

RVF is a commonly under-diagnosed condition in the ICU. The diagnosis can be made using either pulmonary arterial catheterization or echocardiography. Treating the cause, decreasing afterload and restoring good contractility are paramount; the aims of therapy should be to reduce excessive pulmonary artery pressures and to improve cardiac output and systemic arterial pressure. Fluids should be given only to counteract hypovolaemia (because the risk for worsening both right and left contraction is high), with fluid loading guided by cardiac output monitoring. Pulmonary vasodilators and inotropes may be indicated. Monitoring the results of treatment can be achieved continuously with an *in situ* PAC and repeated echocardiographic measurements. RVEF and RVEDVI are two new variables that may be useful in monitoring right heart performance in the setting of RVF. Studies evaluating the efficacy of therapy guided by such variables are awaited.

Competing interests

AR has worked as a consultant for Edwards Lifesciences. AR and MC perform research that is partly funded by Edwards Lifesciences. EJ declares that he has no competing interests.

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