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# RIGHT VENTRICULAR DYSFUNCTION AND ANESTHESIA

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## INTRODUCTION

Until recently, the right ventricle (RV) was considered as a moderately passive conduit between the systemic and pulmonary circulations. This belief was supported by studies showing that complete destruction of the right ventricular free wall in dogs had no detectable impairment on overall cardiac performance[1]. However, investigations in the 1970s demonstrated that right ventricular failure (RVF) has significant hemodynamic effects and consequences on cardiac performance[2].

Right ventricular failure has a similar incidence to left-sided heart failure, with each affecting about 1 in 20 of the population[3]. In contrast with left-sided heart failure, which is often a chronic, progressive disease with a mortality four to eight times greater than that of age-matched general population[4], the outcome of RVF is broadly dependent on the underlying cause, resulting in either an acute or chronic condition. The importance of the right ventricular involvement in heart failure is illustrated by the fact that ischaemia following a myocardial infarction involving both the right and the left ventricle, results in a greater mortality than isolated left ventricular ischemia [5,6].

## NORMAL PHYSIOLOGY OF THE RIGHT VENTRICLE

The right ventricle provides low-pressure perfusion of the pulmonary vasculature, but is sensitive to changes in loading conditions and intrinsic contractility. Factors that affect right ventricular preload, RV afterload or left ventricular function can adversely influence the functioning of the RV and induce a worsen right ventricular failure (RVF).

### Right Ventricular Preload

Preload can be defined as the initial stretching of the cardiac myocytes prior to contraction. In the normal heart, right ventricular preload is determined by the volume of blood that fills the RV at the end of passive filling and atrial contraction (i.e., the end-diastolic volume). Factors which can enhance ventricular preload include mean systemic pressure (determined by blood volume and venous capacitance), and the venous resistance, which is influenced by blood volume, that affects the geometry of the veins (Figure 1).

### Right Ventricular Afterload

The pressure-volume characteristics for the RV differ markedly from those of the left ventricle[7]. The main difference remains is that the RV has only brief periods of isovolemic contraction and relaxation. RV ejection normally occurs at very low pressures which means that 1) blood is transferred to the pulmonary vasculature with minimal oxygen consumption and 2) right ventricular emptying is very sensitive to changes in afterload. Thus, RV function worsens parallel to elevation of the pulmonary artery pressure, main determinant of the right ventricular afterload.

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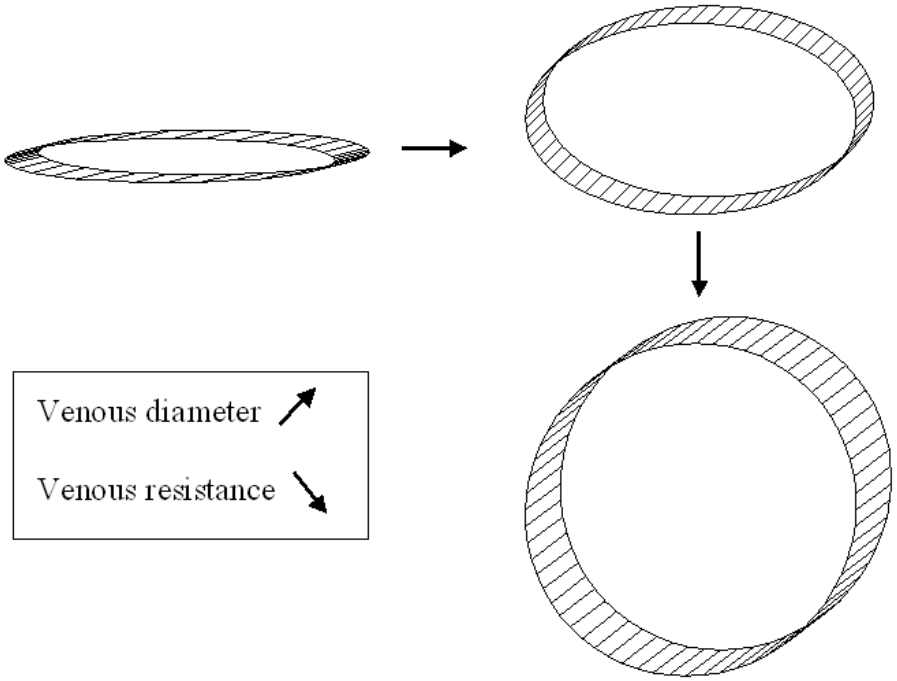


Figure 1:

**The Right Heart Vascularization**

Physiologically, right coronary artery perfusion occurs during both diastole and systole, in contrast to the left coronary artery, that supplies the left ventricular muscle mostly during diastole. However, if systolic pulmonary arterial pressure increases, RV wall stress increases occluding intramural vessels during systole, leading to a pattern of perfusion similar to that of the LV. Right coronary flow occurs therefore predominantly during diastole. Under such conditions, diastolic arterial pressure should be optimal in order to maintain sufficient right and left coronary blood flows [8].

**Normal Pressures in the Right Heart Cavities**

Normal values are presented in Table 1.

Table 1: Normal pressures in the right heart cavities.

| Variable | Value |
|---|---|
| Right atrial pressure * | |
| Mean | 0 – 7 mmHg |
| Right ventricular pressure | |
| Systolic | 15 – 25 mmHg |
| Diastolic | 0 – 8 mmHg |
| Pulmonary artery pressure | |
| Systolic | 15 – 25 mmHg |
| Diastolic | 8 – 15 mmHg |
| Mean | 10 – 20 mmHg |
| Wedge | 6 – 12 mmHg |
| Pulmonary vascular resistance | 100 – 250 dynes/s par $cm^5$ |

* 0 – 4 mmHg during spontaneous breathing, and 2 – 7 mmHg under mechanical ventilation (normal lungs, ZEEP)

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## PATHOPHYSIOLOGY OF RIGHT HEART FAILURE

Various pathophysiologic findings may be encountered during right heart failure, depending on the underlying cause. A summary of these phenomenons is presented in Figure 2. RV failure is usually secondary to a combination of decreased RV contractility, increased RV pressure, and increased RV volume.

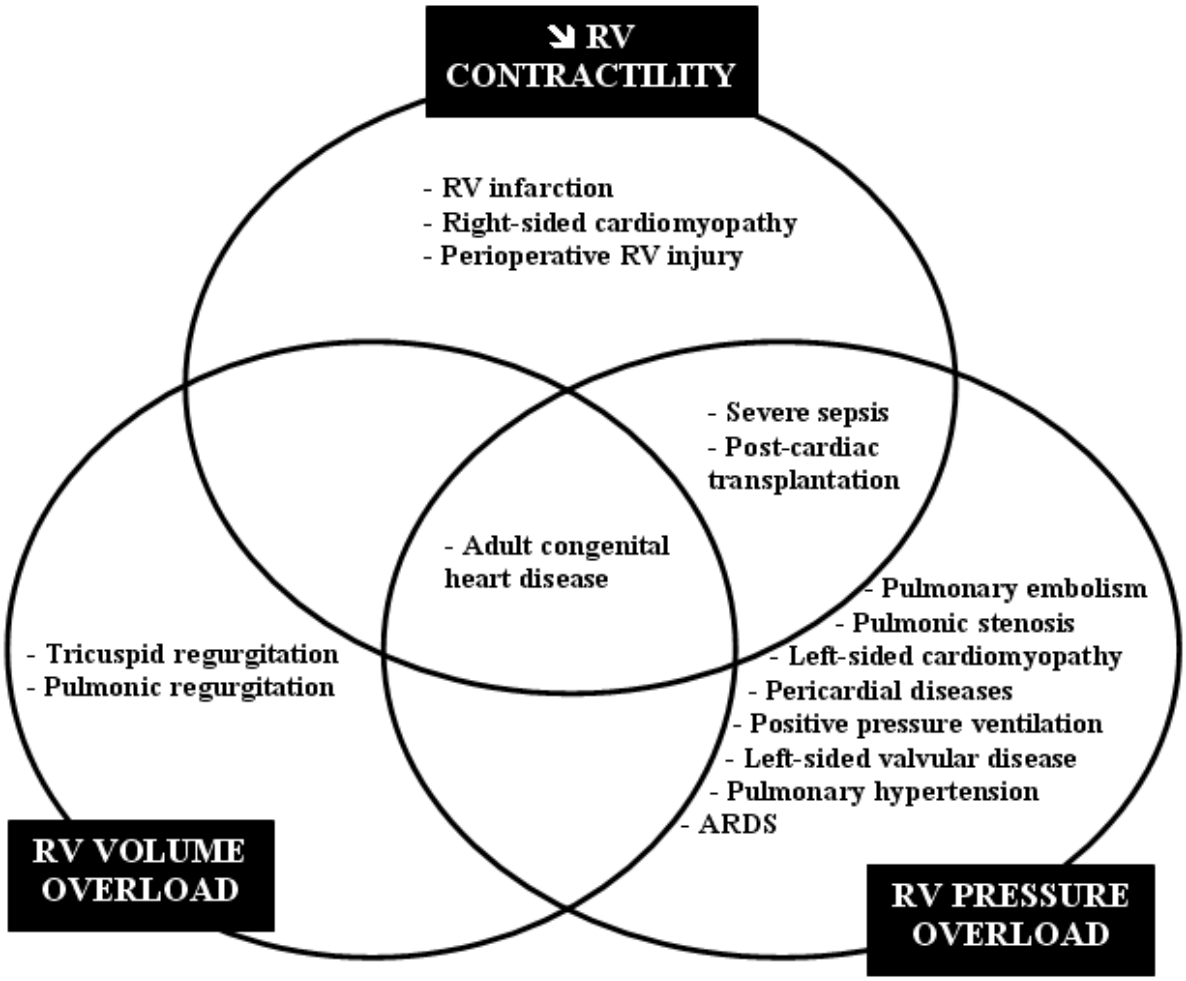


Figure 2: Conditions associated with RV failure categorized by initial pathophysiology (from [20], with permission).

### Right Ventricular Contractile Impairment

This condition occurs most often as a consequence of right ventricular ischemia and infarction. Usually, right ventricular infarction is due to proximal occlusion of the RCA. In this condition, the RV is unable to contract against normal pulmonary artery pressures (see Figure 3). Accordingly, RV ischemia rapidly leads to RV dilatation with a concomitant rise in RV diastolic pressure. Pressure elevation causes a shift of the interventricular septum towards an already underfilled left ventricle. RV dilatation in the setting of limited pericardial compliance leads to increased intrapericardial pressures and additional constraint on the filling of both ventricles[9]. These changes in RV mechanics lead to depressed right-sided output, decreased LV preload and, subsequently, a reduced overall cardiac output [10].

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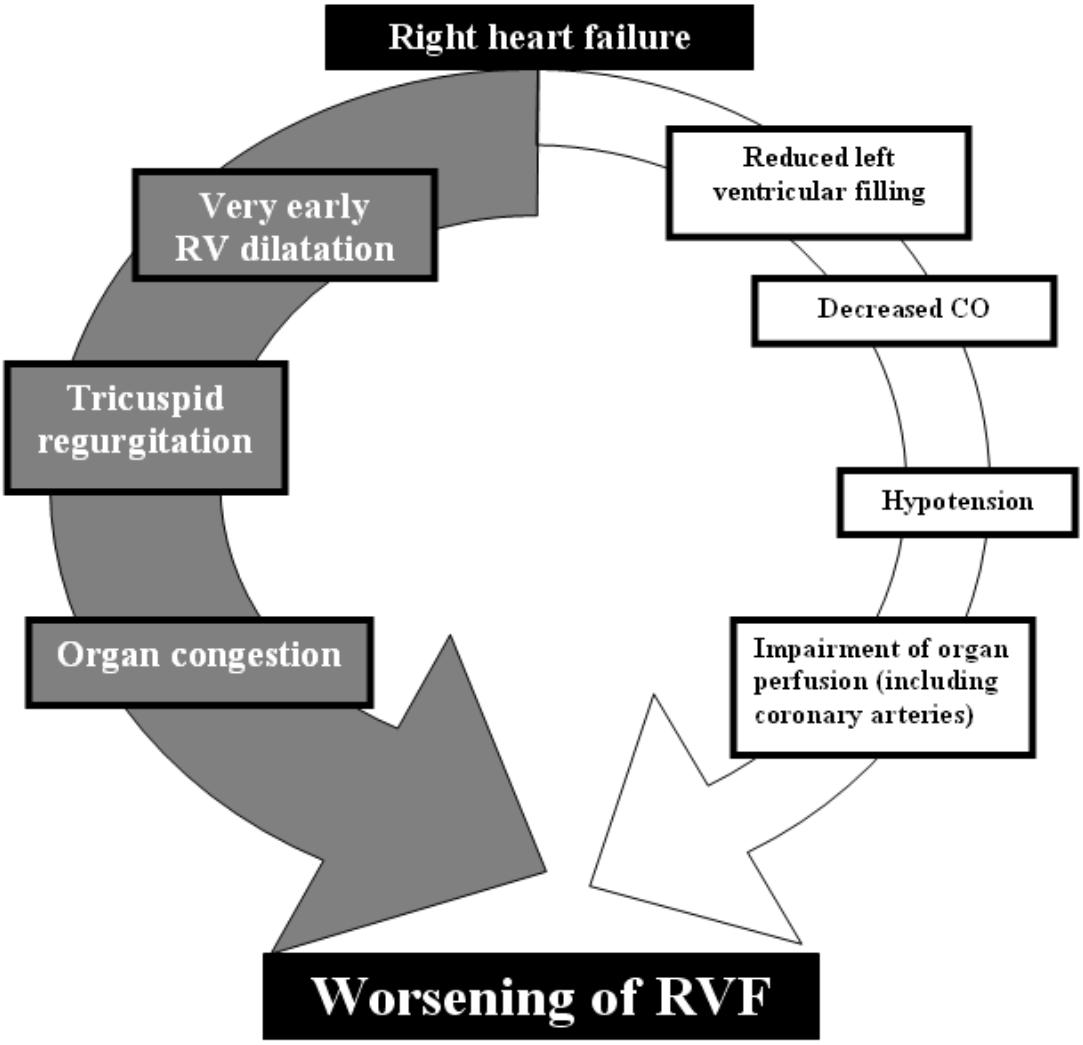


Figure 3: Vicious cycle of auto-aggravation. This pathophysiologic pathway is specific to the RV. This cascad of events must be prevented as soon as possible and implies that any sign of RVF should result in an immediate treatment in order to avoid this vicious cycle.

**Consequences of an Increase in Right Ventricular Afterload**

*Chronic Pulmonary Hypertension*

As described above, increased PAP impairs both coronary perfusion and RV performance. Under normal conditions, RCA perfusion occurs during diastole and systole. A sudden elevation in PAP will reduce systolic perfusion and potentially reduce oxygen supply to the RV at a time when oxygen demand is increased. In addition, as already described, the right ventricle emptying is very sensitive to changes in afterload. In patients with pulmonary hypertension, the RV dilates to maintain stroke volume despite reduced ejection fraction. Increased afterload prolongs the isovolaemic contraction phase and ejection time and, therefore, increases myocardial oxygen consumption. Accordingly, in a patient with decreased RCA perfusion, it is important to reduce right ventricular afterload to improve the oxygen supply/demand balance in the RV and maintain right ventricular function.

*Acute Cor Pulmonale*

Acute cor pulmonale is the consequence of a sudden increase in RV afterload, most often due to a massive pulmonary embolism or acute respiratory distress syndrome in adults[11,12]. In ARDS patients, circulating vasoconstrictors, increased sympathic tone, microvascular obstruction and hypoxic vasoconstriction contribute altogether to increase RV afterload[13]. In response to the abrupt rise in impedance to right ventricular ejection, RV cavity dilates, RV

ejection is impaired, and circulatory failure may occur anytime. Acute cor pulmonale may be completely or partially reversible if RV afterload returns to normal range (Figure 4).

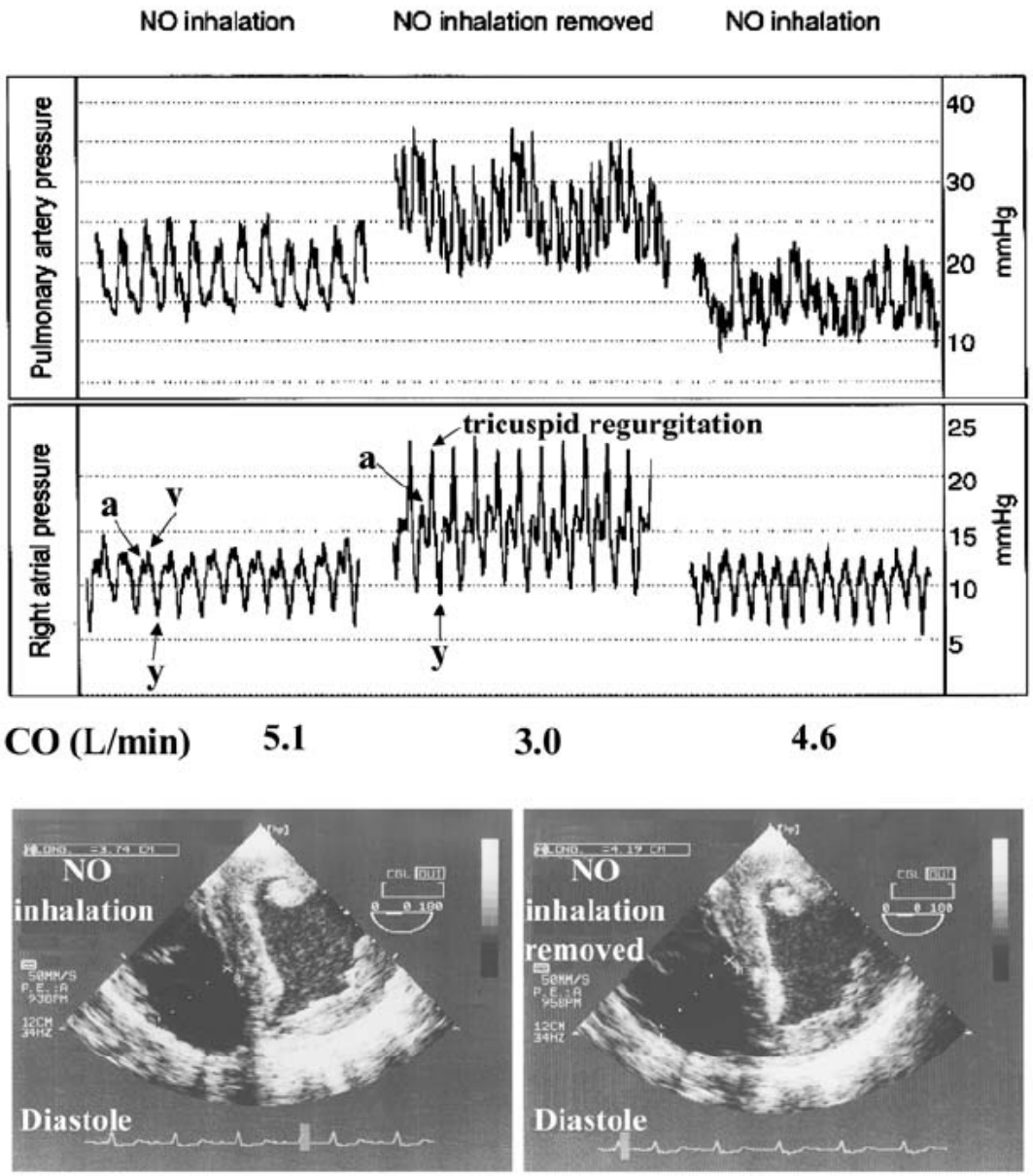


Figure 4: Hemodynamic changes observed with and without inhaled nitric oxide (NO) in a patient with ischemic right ventricular failure. 2D echocardiography shows dilated RV, and an inter-ventricular septum that is shifted from right to left, giving a characteristic "D shape" to the short axis view of the LV. When inhaled NO was removed, the slight increase in pulmonary artery pressure (from 18 to 25 mmHg) resulted in right ventricular dilatation (from 37 to 42 mm) and decreased cardiac output. Simultaneously, right atrial pressure waveform indicated acute tricuspid regurgitation (large "v" waves). (Reproduced from[19], with permission).

**Effect of the Increase in Right Ventricular Volume**

Volume overload is common during RVF and physicians should be aware that additional fluid loading may further dilate the RV, increase tricuspid regurgitation, and, subsequently, worsen hepatic and renal congestion. Although, infusing fluids may seem logical with the prospect of recruiting preload, this effect is potentially limited beyond a mean pulmonary artery pressure of 30 mmHg in most RVF patients[14], and therefore caution is warranted when considering volume loading in any patient with suspecte RVF (Figure 5).

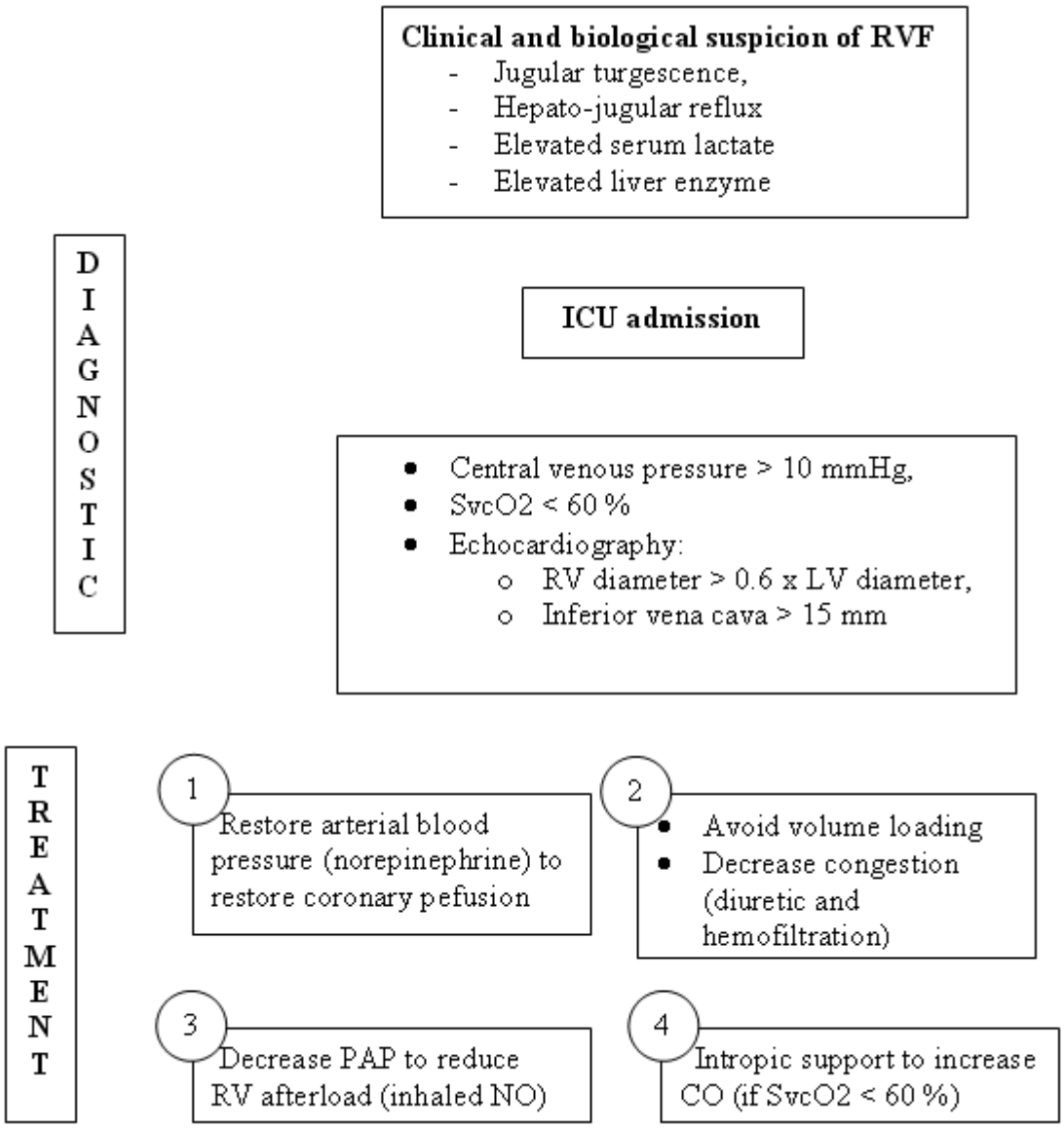


Figure 5: Management algorithm of RVF.

**Ventricular Interdependence**

There is a high degree of ventricular interdependence due to the existence of a non-compliant pericardium surrounding both ventricles[15]. Any acute increase in the size of one chamber will occur at the expenses of the other one[16]. Indeed, increases in the end-diastolic volume of the left ventricle are transmitted to the RV by movement of the interventricular septum toward the right cavity, increasing the end-diastolic pressure of the RV[17]. Similarly, when right ventricular end-diastolic volume is increased, the interventricular septum shifts towards the left cavity during diastole due to restrictions imposed by the pericardium on the RV as the cavity volume increases. This leftward shift impairs left ventriclar filling due to the reduction in left ventriclar volume and compliance.

Ventricular interdependence can also cause RVF during left ventricular assist device support. As the left ventricular assist device restores cardiac output, the sudden increase in venous return may precipitate acute right ventricular failure[18]. It is therefore crucial to monitor right ventricular function during the first days following insertion of a left ventricular assist device and to provide inotropic support to the RV if required.

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### The Vicious Cycle of Auto-Aggravation

Compared to the left ventricle, RVF progresses quickly from compensated to end-stage heart failure because of a vicious cycle of auto-aggravation (Figure 3). This is quite specific to the RV and rarely seen in isolated left ventricular failure. As can be seen in Figure 4, although modest, a sudden increase in RV afterload (inhaled NO withdraw) on an ischemic RV immediately dilates the RV, induces a tricuspid regurgitation and decreases cardiac output.

## CONCLUSION

Understanding physiology and physiopathology of the right ventricule is essential for the management of right ventricular failure. Intensivists and anesthesiologists should be aware that the RV is an organ at risk in critically ill patients and in some surgical patients. Therapeutic management of RVF depends upon underlying etiology, but primarily involves breaking the vicious circle of reduced cardiac output. This will allow restoring adequate oxygen delivery to the myocardium and reducing right ventricular overload. Treatment of RVF should therefore focus on alleviating congestion (limit volume loading), increase right coronary artery perfusion pressure and flow, improving right ventricular contractility and/or reducing right ventricular afterload (avoid mechanical ventilation and/or avoid high airway pressure).

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