

Is Right Ventricular Cardiogenic Shock Important To You?

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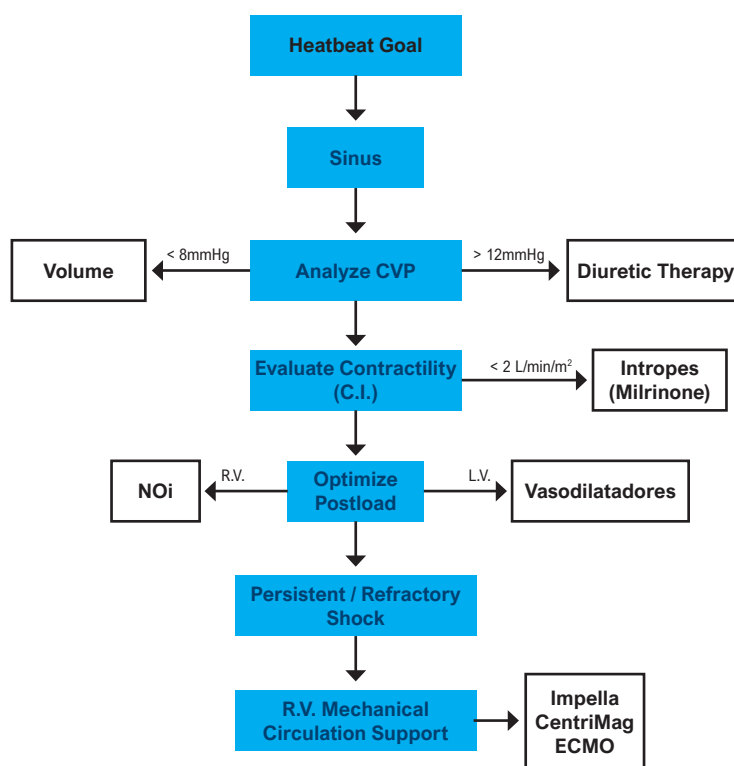
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Central Illustration: Is Right Ventricular Cardiogenic Shock Important To You?



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CVP: Central Venous Pressure; C.I.: Cardiac Index; R.V.: Right Ventricle; L.V.: Left Ventricle; NOi: Nitrous Oxide Inhalation; ECMO: extracorporeal membrane oxygenation.

Keywords

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Introduction

In the scenario of cardiogenic shock, attention was almost exclusively focused on the left ventricle (LV). Currently the concept that the circulatory system involves the interdependence between the LV and the right ventricle (RV) has been reinforced, and researchers seek to achieve a clearer understanding of the role of the right ventricle in cardiogenic shock.¹

In the 1940s, in search of a more in-depth understanding of the role of the RV, the American doctor and researcher

Isaac Starr and his team carried out a series of experiments on animals, in which they caused severe damage to the right ventricle. Observing only a small increase in peripheral venous congestion, they concluded that “*the weakness on the right side of the heart... appears to be less important*” in the establishment of heart failure (HF).²

These concepts have perhaps placed the RV on the margins of research for many years. However, today, it is well-known that it plays an extremely relevant role in the pathophysiological context of cardiogenic shock. This leads to the need to better understand the behavior of RV disorders.²

In this context, reaching an early etiological recognition and diagnosis is crucial, as clinical therapy can range from the mechanical or pharmacological removal of obstructive thrombi, to the need for the use of vasodilators, inotropes, myocardial revascularization or even ventricular assist devices (VADs).³

Pathophysiology

RV function is influenced by preload and postload, myocardial contractility, and factors such as pericardial conformity and conditions that alter chest pressure.¹

In chronic increases in pulmonary vascular resistance (PVR), the RV has the ability to hypertrophy and dilate. This adaptation serves to reestablish ventricular-vascular coupling in the short term, but can lead to dilation, tricuspid regurgitation, RV hypertrophy, and ischemia. In the acute state of RV failure, these adaptations do not occur and the RV response is more blunted, leading to RV cardiogenic shock, which is a hemodynamic problem caused by a compromised function of the ventricle, valves, or vasculature.⁴⁻⁶

Therefore, it is understood that the management of RV cardiogenic shock not only requires an understanding of the anatomical and physiological particularities of the RV, but also a rapid identification and treatment of the underlying causes and related pathophysiological disorders.^{7,8}

Primary mechanisms of RV cardiogenic shock include: contractile failure secondary to ischemic or inflammatory myocardial damage, volume overload due to right-sided valve insufficiency, increased venous return, pressure overload resulting from left HF, worsening of the PVR, or acute pulmonary embolism.⁹

Although isolated RV infarction is relatively rare, RV ischemic involvement has been observed in 40-50% of all patients with inferior infarction, and these have a higher risk of cardiogenic shock, arrhythmia, and death.^{10,11}

Diagnosis

Making the diagnosis of RV cardiogenic shock remains a major challenge in clinical practice. Patients with RV cardiogenic shock show signs of hypoperfusion, hypotension, and cold extremities. In etiologies involving chronic RV heart failure (HF), these manifestations may be associated with symptoms of systemic congestion, characterized as: hepatomegaly, abdominal distension, ascites, and peripheral edema.¹²

The mechanism to explain the clinical exuberance is that the right heart dilates and can displace the interventricular

Table 1 – Causes of right ventricular failure and related etiologies

Parameters	Acute Insufficiency	Chronic Dysfunction
Preload		Atrial septal defect
	Acute Kidney Injury	Tricuspid or pulmonary insufficiency
	Acute Valvular Insufficiency	Ventricular septal defect
	Patent foramen ovale	High Output Heart Failure
Postload		Fisenmenger syndrome
	Pulmonary thromboembolism	
	Hypoxia of pulmonary origin (Pneumonia, ARDS)	Pulmonary valve stenosis
	Pressure ventilation Positive	Pulmonary artery stenosis
Contractility	RV infarction	
	Myocarditis	Arrhythmogenic RV Dysplasia
	Supraventricular or ventricular tachycardia	

RV: right ventricle; ARDS: acute respiratory distress syndrome.

septum to the left, thus compromising LV filling, reducing its performance and causing hypoperfusion and systemic congestion, resulting in a marked increase in transaminases and circulating lactic acids, triggering the pathophysiological mechanisms of shock. In the laboratory, the measurement of atrial natriuretic peptide (BNP) is quite sensitive, but not very specific for the diagnosis of RV insufficiency and can be useful as evidence of RV impairment, particularly when there is no associated LV dysfunction.^{13,14}

Recently, an analysis of Culprit-Shock generated a score with good prediction of mortality from shock related to acute myocardial infarction. This score, however, uses markers that are not widely available, such as cystatin C and interleukin-6, among others that are more easily accessible, such as NT-proBNP and lactate.¹⁵

Echocardiography is the most useful non-invasive tool to evaluate RV anatomy and function, cardiac output, inferior vena cava, and valvular heart disease. However, it has limitations because it depends on good imaging quality and the performance of the operator. Cardiac magnetic resonance imaging (MRI) and computed tomography (CT) of the heart are less useful in bedside management.¹⁶

Recently, with the greater availability of bedside USG devices, Point-of-care-ultrasound (POCUS) is a useful tool in RV assessment. To evaluate RV preload and postload, the following parameters can be used: relative size of the RV in relation to the LV; shape of the interventricular septum and investigation of McConnell's sign. When evaluating the contractile function, the displacement of the tricuspid annulus in relation to the RV apex during systole (TAPSE) is measured. It is important to highlight, however, that visual impression is the most widely used parameter to evaluate RV systolic function when using POCUS. The accuracy of visual estimation depends on the examiner's experience.^{17,18}

Hemodynamic assessment through pulmonary artery catheterization, Swan-Ganz, continues to be the gold standard in the diagnosis of RV cardiogenic shock. Indicated in RV shocks with pulmonary artery hypertension, in cases where the echocardiogram windows are inadequate, this helps to define the components of vasoplegia versus the pump deficit.¹⁹

Although several formulas for evaluating pulmonary hemodynamics have been developed to quantify RV postload, including RV dysfunction, diastolic and transpulmonary gradient, pulmonary artery elasticity, compliance, or impedance, none of these formulas alone characterizes RV failure. It is also essential to define the severity of the cardiogenic shock while these hemodynamic measurements are performed to contextualize the data obtained.²⁰⁻²²

Treatment

As noted in the central figure, treatment of RV cardiogenic shock should seek to address the underlying cause of RV dysfunction and improve blood flow to the lungs and body. The objective should be to stabilize and provide hemodynamic support to the patient and carry out specific measures based on the etiological diagnosis.⁴

1) Volume optimization

Even considering that patients with predominant RV shock are dependent on preload, it is important to highlight that most RV failure is caused, associated, or worsened by volume overload. These patients will often be congested, with distended myocardial fibers. A fluid removal strategy reduces the distension of myocardial fibers, resulting in an improvement in coronary perfusion and more effective ventricular contraction, less pulmonary congestion, and, consequently, less hypoxia. Therefore, essential components to preload management are the optimization of central venous pressure, the prevention of ventricular distension, as well as the management of circulating volume status.²³

In patients with RV insufficiency and signs of venous congestion, diuretics are often the first option to optimize volume status. The elevated renal venous pressure often found in these patients contributes to decreased renal blood flow and reduces perfusion pressure, which decreases the effectiveness of the diuretic. Therefore, in patients with RV cardiogenic shock with significant renal congestion, adequate doses of diuretics are crucial to achieve the desired effect and maintain renal perfusion.^{24,25}

A continuous infusion of loop diuretics is often necessary to maintain the decongestive effect. If decongestion is insufficient, rapid intensification of the dose of loop diuretics should be considered, combining diuretics with different mechanisms of action or the use of ultrafiltration.²³

2) Vasopressors

In cardiogenic shock, the use of vasopressors has proven benefits, mainly indicated to restore blood pressure (MAP target of ≥ 65 mmHg) and improve coronary and systemic perfusion. In RV cardiogenic shock, vasopressin can be

considered the first choice, as it has little interference with peripheral vascular resistance, and some experimental studies suggest that it may have a pulmonary vasodilation effect, which leads to a decrease in RV postload.^{26,27}

In cases of refractory cardiogenic shock, one should not use the combination of noradrenaline and vasopressin, given that the latter does not have a positive chronotropic effect. After this association, one should consider a ventricular assist device.

Rationally, it should be used for the shortest possible time and in the lowest possible dose, since vasoconstrictors in general increase left ventricular postload, reduce cardiac output, increase myocardial oxygen consumption, and can induce arrhythmias.²⁸

3) Inotropes

If cardiac output is unsatisfactory, inotropes can be considered with the aim of increasing output and improving renal perfusion. However, the risk of arrhythmia and cardiac ischemia must be considered.⁴

In this context, dobutamine, levosimendan, and milrinone can be used for management. It is important to note that levosimendan and milrinone have an inodilatory action, especially for those who have pulmonary hypertension due to LV involvement, as they promote inotropism and pulmonary vasodilation, reducing RV postload. However, particular care must be taken in patients with RV infarction, since these medications increase oxygen consumption through the cardiomyocyte and may predispose to arrhythmias.²³

In a meta-analysis published in 2018, there was an increase in mortality in the group that used epinephrine in cardiogenic shock, possibly due to the arrhythmogenic potential of this drug.²⁹

There is a lack of robust evidence regarding the best drug in the management of RV shock. Dobutamine and Milrinone were recently compared in the context of cardiogenic shock, and no difference was found in primary or secondary outcomes.³⁰

4) Devices

Recognizing the patient who is deteriorating, even despite pharmacological treatment, and who requires mechanical circulatory support (MCS), is essential for good results, as premature implantations of these devices can expose patients to unnecessary therapies and risks of related complications, since the delay in its installation may not be able to reverse the shock in more advanced stages.²³

Auxiliary propaedeutic measures can help determine the appropriate time to start MCS, based on the careful monitoring of hemodynamic and metabolic parameters. If hemometabolic parameters do not improve, despite optimized pharmacological management over a few hours, SCM is recommended. Although the use of a hemodynamic device, such as the Swan Ganz, has not improved survival in a broad population of patients with HF, observational data have demonstrated a positive association with survival in cardiogenic shock.³¹⁻³³

Ventricular assist device options for RV *cardiogenic* shock include venoarterial extracorporeal membrane oxygenation (VA-ECMO), the TandemHeart flow centrifugal pump, and the axial flow Impella RP catheter. These devices can be categorized according to their mechanism of action as direct RV bypass or indirect RV bypass systems. Impella RP and TandemHeart move blood from the right atrium to the pulmonary artery, directly bypassing the RV. By contrast, ECMO moves and oxygenates blood from the right atrium to the femoral artery, indirectly bypassing the RV. As a result, these systems have different hemodynamic effects depending on whether the patient has isolated RV or biventricular failure.³⁴

Intra-aortic balloon counterpulsation (IABP) failed to increase cardiac output in patients with advanced HF and RV involvement. For these reasons, IABP is not considered a primary ventricular assist device in RV *cardiogenic* shock.^{35,36}

The most recent introduction to the VADs is the Impella RP microaxial flow catheter. Since 2013, several reports have described the successful use of the Impella RP for RV failure in the context of heart surgery. In 2015, the RECOVER RIGHT trial prospectively studied the utility of this device for refractory RV failure, allowing for the weaning of inotropic and vasopressor support. Impella RP has been used to support patients with RV failure associated with malignant ventricular arrhythmias and severe mitral valve regurgitation.³⁷⁻⁴¹

Conclusion

RV cardiogenic shock is a serious medical condition that requires a rapid and accurate diagnosis and treatment.

Knowledge and awareness about this condition are essential for an early detection and appropriate intervention in order to improve the prognosis of affected patients. Continuous research and updating of health professionals in this area are essential to advance the treatment and management of RV cardiogenic shock.

Author Contributions

Conception and design of the research, Acquisition of data and Critical revision of the manuscript for important intellectual content: Andrade MVS, Aragão CAS, Figueiredo RMP, Almeida BM, Rocha LG; Writing of the manuscript: Figueiredo RMP, Almeida BM, Rocha LG.

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Ethics approval and consent to participate

This article does not contain any studies with human participants or animals performed by any of the authors.

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