Management of Right Heart Failure in the Intensive Care Unit

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KEYWORDS

• Right heart failure (RHF) • Right ventricular failure (RVF) • Pulmonary arterial hypertension (PAH) • Pulmonary hypertension (PH) • Pulmonary vasodilators • Atrial septostomy • Potts shunt

KEY POINTS

- Right heart failure is a syndrome of multiple possible causes that can eventually result in circulatory failure and death.
- Hemodynamic support of a failing right ventricle must address its preload, contractile, and afterload states.
- Additional adjunctive support measures to optimize electromechanical coupling and ventilator support remain important interventions.
- Rescue interventions to offload a failing right ventricle continue to have a role in the management of this highly morbid syndrome.

INTRODUCTION

Right heart failure (RHF) is a clinical syndrome that arises from a disturbance of the right-sided circulatory system and results in elevated venous pressure and/or aberrant delivery of blood to the pulmonary circulation. It represents a pathologic disturbance of any component of the right heart circulatory system, which consists of the venous system up to the level of the pulmonary capillaries and, therefore, has both systemic (the systemic veins up to the level of the pulmonic valve) and pulmonary components (the precapillary pulmonary circulation).[1] Within this framework lies the more specific concept of right ventricular failure (RVF), which is often a major component of RHF. Conceptualized hemodynamically, RVF occurs at the point at which cardiac output (CO) and blood pressure drop despite an increased RV end-diastolic pressure (RVEDP).[2] It can be suspected when the right atrial pressure (RAP) to pulmonary artery occlusion pressure ratio is less than or equal to 0.8 to 1, usually with a low cardiac index (CI).[3] Exacerbating this presentation is the negative impact of a failing, distended right ventricle (RV) on left ventricle (LV) function with further reduction of CO.[4] The RV may fail when subjected to pressure or volume overload, ischemia, intrinsic myocardial disease, or from pericardial limitation.[5]

DIAGNOSTIC CLUES

The classic physical examination findings in a patient in RVF include an elevated jugular venous pressure with a prominent v wave. On auscultation, one may note a prominent pulmonic component of the second heart sound and the holosystolic murmur of tricuspid regurgitation (TR). On palpation, one may note a right ventricular heave, hepatomegaly, ascites, and/or lower extremity edema. Although the pulmonary examination may point to underlying lung disease, the suggestion of pulmonary edema is not consistent with isolated RVF and suggests possibilities such

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as left-sided heart disease or noncardiogenic causes.

Laboratory studies can help determine the underlying cause of RVF if it is indeed secondary to pulmonary arterial hypertension (PAH) with serologic markers of connective tissue disease, human immunodeficiency virus infection, and the viral hepatitides with concurrent assessment of liver function. Polycythemia, suggestive of chronic hypoxemia, can be associated with the World Health Organization Group 3 causes of pulmonary hypertension (PH), which can ultimately result in cor pulmonale. Both overdistension and ischemia of the RV predispose to elevations of cardiac enzymes and B-type or pro-B-type natriuretic peptide. Positive biomarkers are well-described in pulmonary embolism and confer greater mortality risk.[6,7]

Electrocardiography remains frequently used in the initial assessment of acute heart failure but is an insensitive test of PH, pulmonary embolism, or RVF. Nonetheless, one may see evidence of right axis deviation, an R:S wave of greater than 1 in V_1, an R wave greater than 0.5 mV in V_1 and electrocardiographic signs of right atrial enlargement (p pulmonale) with P wave amplitude greater than 2.5 mm in leads II, III, and aVF; or greater than 1.5 mm in V_1.[8] The qR pattern in lead V_1, in which one sees the negative deflection of a q wave immediately followed by an upward R wave, has also been associated with RV strain and adverse outcomes in patients presenting with acute pulmonary embolism.[9]

ASSESSMENT OF RIGHT HEART FAILURE

The management of RVF should be predicated on an assessment of the preload, the RV contractility, and the afterload. The preload stress of RV is intimately tied with the volume status of the patient (see later discussion) (Fig. 1).

When suspecting the clinical syndrome of RVF, an assessment of the contractile state is essential. The right ventricular ejection fraction (RVEF) is a commonly used index for this. Nonetheless, its value depends highly on the loading conditions of the RV and may not provide an accurate reflection of contractility.[10] The RV chamber size is larger than the LV's, rendering the RVEF less than its counterpart and ranges from 40% to 76% depending on the method of its analysis, with MRI and radionuclide angiography being the most accurate. Unfortunately, neither modality is practical for assessing a critically ill patient.

Although different than contractility, CO is a relevant clinical parameter and can be obtained with placement of a pulmonary artery catheter (PAC). Given the absence of improved outcomes data in favor of empiric placement of PACs in the critically ill, its use must be considered in the context of the information it provides and how that might alter management.[11] Furthermore, different techniques used to measure CO are not necessarily equivalent; there can be significant discrepancy between sequentially obtained Fick and thermodilution measurements of CO.[12] Echocardiography, conversely, is a noninvasive test

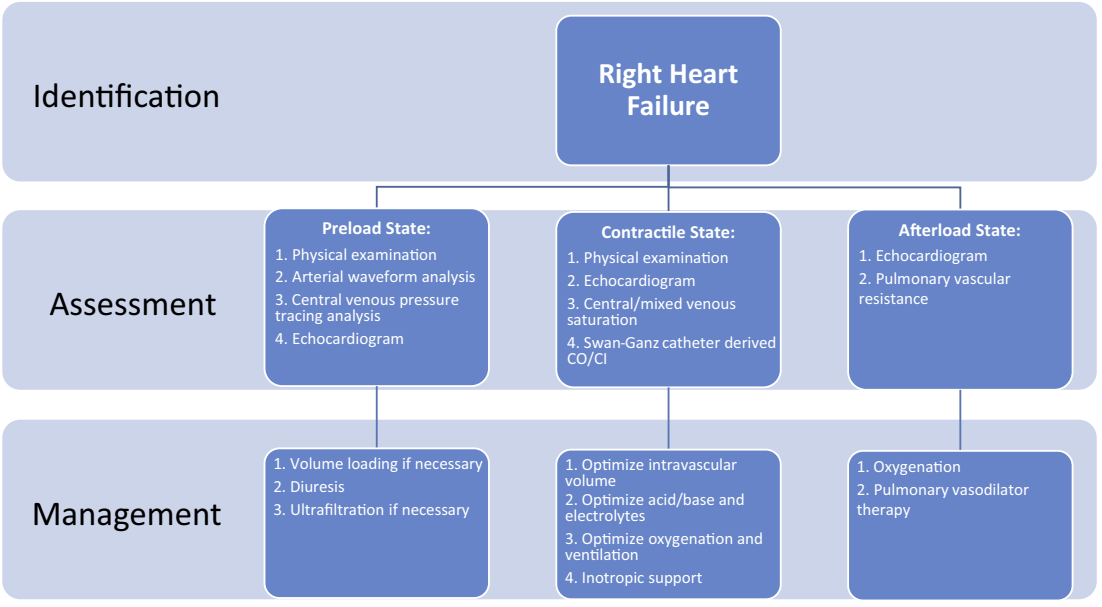

Fig. 1. Summarized assessment and management of the preload, contractile, and afterload states in the setting of right heart failure.