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CE

Right Ventricular Failure

Barbara Leeper, MN, RN-BC, APRN, CNS M-S, CCRN

ABSTRACT

Interest in the right ventricle has increased because of advances in pulmonary hypertension treatment, improved diagnostic technology, and increased implantation of left ventricular assist devices and other mechanical circulatory assist devices. Right ventricular dysfunction is an independent predictor of mortality in patients with chronic heart failure. The purpose of this article is to describe the normal structure and function of the right ventricle, causes of right ventricular dysfunction leading to right ventricular failure, diagnostic hemodynamic assessments, and management of right ventricular failure in the critical care unit.

Key words: heart failure, myocardial infarction, pulmonary hypertension, right ventricular dysfunction

The right ventricle (RV) has been described as the forgotten chamber, indicating that its only significance is to deliver venous blood to the pulmonary vascular bed and subsequently to the left ventricle (LV). Recently, interest in the RV has increased because of advances in the management of pulmonary hypertension, improved diagnostic technology, and increased implantation of left ventricular assist devices (LVADs) and other mechanical circulatory assist devices for the management of cardiogenic shock.[1] Right ventricular dysfunction, defined as evidence of abnormal RV structure or function,[1] is a powerful and independent predictor of mortality in patients with chronic heart failure.[2] This article describes the normal structure and function of the RV, causes of RV dysfunction leading to RV failure, diagnostic and emerging hemodynamic assessments, and management of RV failure in the critical care unit.

Right Ventricular Physiology

The RV is a crescent-shaped chamber that has a thin free wall (2 to 3 mm thick) and is wrapped around the LV.[3] Normally the chamber is highly compliant and volume dependent. The RV has one-sixth the muscle mass of the LV.[4] However, the end-diastolic volume of the RV is larger (range, 49-101 mL/m^2) than that of the LV (range, 44-89 mL/m^2). The higher RV end-diastolic volume is associated with a lower ejection fraction (range, 40%-60%) than the LV ejection fraction (range, 50%-55%). The RV contracts with a bellows-type motion, resulting in movement of the RV free wall toward the interventricular septum. The highly compliant RV muscle allows it to tolerate large increases in venous return. However, the RV is much less tolerant of increased afterload than is the LV.[3]

Determining factors of RV function include systemic venous return (preload), pressure in

Barbara Leeper is Clinical Nurse Specialist, Cardiovascular Services, Baylor University Medical Center, 3500 Gaston Avenue, Dallas, TX 75252 (Bobbi.Leeper@BSWHealth.org).

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Table 1: Comparison of Right Ventricle and Left Ventricle Characteristics[a]

Characteristics	Right Ventricle	Left Ventricle
Shape	From the side: triangular Cross section: crescent	Elliptical
Volume, mL/m^2	49-101	44-89
Muscle mass, g/m^2	<35 (one-sixth of left ventricle muscle mass)	<130 (men) <100 (women)
Ejection fraction, %	40-68	57-74
Ventricular compliance	Higher compliance than left ventricle	
Adaptation to disease	Better adaptation to volume overload states	Better adaptation to pressure overload states

[a] Adapted from Haddad et al.[6] Used with permission from Wolters Kluwer.

the pulmonary vascular bed (afterload), and the contractile properties of the RV free wall and interventricular septum. The RV generates one-fourth of the work of the LV because much of the RV contractile force maintains forward blood flow out of the chamber into a highly compliant, low-resistance pulmonary vascular bed.[4] Generally, pulmonary vascular resistance is less than one-tenth of the systemic vascular resistance, allowing it to adapt to variations in volume rather than pressure.[1] The RV has a lower peak systolic pressure than the LV. The crescent shape of the RV makes it much less tolerant of acute increases in afterload. The RV is not able to acutely generate a mean pressure greater than 40 mm Hg. Stroke volume falls linearly as RV afterload increases. Rapid increases in afterload can cause the RV to dilate, damaging the muscle tissue and causing RV failure.[5] Refer to Table 1 for a comparison of RV and LV characteristics.

Ventricular Interdependence

According to the concept of ventricular interdependence, the size, shape, and compliance of 1 ventricle affect the size, shape, and pressure-volume relationship of the other ventricle through mechanical interactions. In other words, the forces from 1 ventricle are transmitted to the other through the myocardium and pericardium.[1] The determinants of this relationship include the ventricular septum, pericardium, and continuity of the pericardial fibers between the RV and LV.[4] The concept becomes important with changes in preload and/or afterload, for example during the respiratory cycle or abrupt postural changes. This interaction plays an important role in RV pathophysiology.[4]

Pathophysiology of RV Failure

Acute RV Failure

Right ventricular failure can occur abruptly or slowly over time. Situations causing an abrupt increase in afterload, such as pulmonary embolism, hypoxia, and acidosis, are associated with the abrupt onset of RV failure. Conditions causing acute impairment of RV contractility, such as myocarditis and myocardial ischemia, also contribute to an abrupt onset of RV failure. As RV stroke volume begins to fall, the RV dilates, causing tricuspid regurgitation. The regurgitant tricuspid valve worsens the RV dilatation and affects LV filling because of ventricular interdependence. As the RV dilates, the interventricular septum flattens. The LV end-diastolic pressure increases, impairing LV filling during diastole and reducing cardiac output (Figure 1).[1] Systolic interactions also occur between the 2 chambers. An estimated 20% to 40% of RV systolic pressure is generated from contraction of the LV. As the RV filling pressure increases, coronary sinus congestion occurs, impeding coronary blood flow and contributing to the development of RV ischemia. The increased right-sided filling pressure contributes to venous congestion, negatively affecting renal and hepatic function and worsening fluid retention and RV failure.[1]

Chronic RV Failure

Chronic RV failure is commonly caused by the development of pulmonary hypertension over time, most often related to LV failure. Chronic RV volume overload, such as tricuspid regurgitation, can lead to RV failure. Long-term pressure or volume overload is initially associated with the development of RV

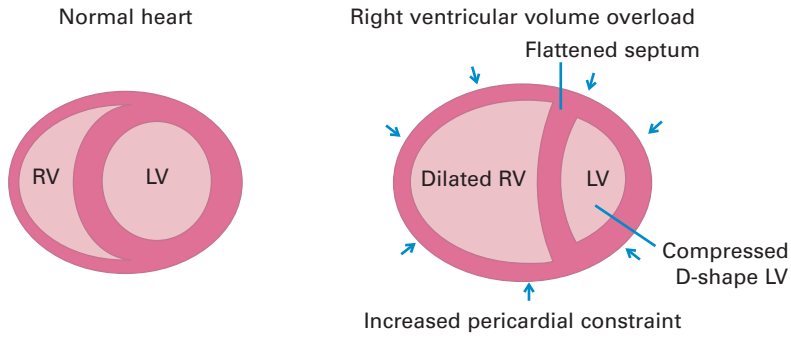

Figure 1: Ventricular interdependence in right ventricular failure.[1] Normally the right ventricle (RV) is crescent shaped and attached to the intraventricular septum. When the RV begins to fail, the chamber dilates and is limited by the pericardium. Right ventricular pressure increases, causing the septum to flatten and reduce the size of the left ventricle (LV) chamber. Left ventricular end-diastolic volume is reduced and stroke volume decreases. Reproduced from Haddad et al.[7] Used with permission from Wolters Kluwer.

hypertrophy. Both systolic pressure and RV end-diastolic volume increase as the RV dilates. Right ventricular dilatation compresses the interventricular septum, impairing LV filling and eventually causing equalization of the diastolic pressures of both ventricles. The pericardium limits the amount of RV dilatation, further increasing RV pressure and impairing LV filling (Figure 1). As the RV begins to decompensate, pulmonary vascular resistance and right atrial pressure (RAP) increase. Cardiac output begins to fall followed by a reduction in pulmonary artery (PA) pressure while pulmonary vascular resistance may remain elevated. The combination of the lower PA pressure and higher pulmonary vascular resistance has been identified as an ominous clinical indication.[1]

Causes of RV Failure

Myocardial Infarction

Right ventricular myocardial infarction (MI) accompanies 35% to 50% of all inferior wall MIs. If the right coronary artery is occluded proximally to the RV branches, RV systolic dysfunction accompanied by acute RV dilatation will occur.[1] Right ventricular MIs can also occur with occlusion of the circumflex artery and the left anterior descending coronary artery. The left anterior descending coronary artery supplies the anterior wall of the RV as well as the interventricular septum. If a septal MI is present, the RV is impacted considerably. As much as 40% of the RV contractile force is derived from the interventricular septum.[8] If the force from the septum is lost, RV myocardial contractility is severely impaired. Approximately 25% to 50% of patients with a RV MI experience some hemodynamic compromise, which is associated with a high mortality rate.[1]

Left Ventricular Assist Devices

At least 20% to 50% of patients with an implanted LVAD are estimated to experience acute onset of RV failure, contributing to significant postoperative morbidity and mortality.[1] The LVAD increases venous return and right ventricular preload. The RV may become overwhelmed and dilate. Tricuspid insufficiency occurs and the interventricular septum shifts to the left, flattening out and causing a reduction of RV stroke volume. The effect on the LV is reduced preload and decreased LVAD flow. If the patient has pulmonary hypertension, the risk for acute RV failure is increased. In some patients the dysfunctional RV may not be able to compensate for the increased afterload associated with pulmonary hypertension.

Pulmonary Embolism

The abrupt onset of pulmonary embolism in a major branch of the PA has immediate (within minutes) effects on the RV, causing a marked increase in afterload. The incidence of acute RV failure in this setting is 25% to 60%.[1] Patients may present with syncope and atrial arrhythmias.

Pulmonary Hypertension

Pulmonary vascular pressures determine the afterload of the RV. As chronic pulmonary hypertension develops, the RV initially adapts

to the increased PA pressure by developing concentric hypertrophy, preserving systolic and diastolic function. When a 4- to 5-fold increase in PA pressure occurs, the RV is no longer able to compensate and RV function declines. The chamber dilates and RV failure ensues.[9] Patients with long-standing pulmonary hypertension may have PA pressures that equal or exceed systemic pressures.

Assessment of RV Failure

Right ventricular failure is assessed via a thorough physical examination for clinical manifestations including distended neck veins, particularly during inspiration (Kussmaul sign); peripheral edema; hepatomegaly; and ascites. Another indication is the abdominal-jugular reflux sign, a sustained increase of more than 3 cm in the height of the jugular venous distention for at least 15 seconds with calm spontaneous breathing.[1] Right ventricular failure can also be tested by compressing the liver along the right costal margin with the head of the bed at a 45° angle. This test is positive if jugular venous distention increases while the liver is being compressed. If RV hypertrophy is present, a precordial heave can be palpated. The examination should include auscultation for tricuspid regurgitation, which would be associated with dilatation of the chamber.

Other assessments include 12-lead electrocardiography, chest radiography, and echocardiography. Echocardiography allows for assessment of RV size, competence of the tricuspid and pulmonic valves, and presence of abnormal wall motion associated with impaired contractility. However, the use of echocardiography for diagnosis can be challenging because the complex structure of the RV can lead to inaccurate measurements and interobserver variability. Magnetic resonance imaging provides specific information about RV volume but is limited in patients with implantable pacemakers and defibrillators.[2,10]

Hemodynamic Assessments

Right ventricular dysfunction is not easily identified by clinical imaging or physical examination. Therefore, right heart catheterization with a PA catheter (PAC) is the gold standard to assess RV function.[2,8] Right heart catheterization can be performed as an outpatient procedure to evaluate the RV or in the intensive care unit to provide continuous monitoring for patients with MI, advanced heart failure, LVAD, heart transplant, or cardiogenic shock. The PAC directly measures RAP, PA and pulmonary artery occlusion pressure (PAOP), mixed venous oxygen saturation, and cardiac output and allows for calculation of cardiac index, systemic vascular resistance, pulmonary vascular resistance, stroke work index, PA pulsatility index (PAPi), and cardiac power index.[8,11] Various hemodynamic variables have been identified as risk factors for RV failure, particularly following the implantation of a LVAD. One of these risk factors is elevated RAP, especially when it is disproportionate to the rise in PAOP. Elevated RAP is considered to be a marker for RV dysfunction. Generally RAP has been considered the most reliable sign of increased PAOP, but approximately 25% to 30% of patients have a discordance between the right-sided and left-sided filling pressures.[2] Normally the RAP to PAOP ratio is about 0.5. A higher ratio is an indication of RV dysfunction. For patients undergoing LVAD implantation, a RAP to PAOP ratio of greater than 0.63 is associated with RV failure following the surgical procedure.[1]

Another parameter that uses the sum of the RAP and PAOP, called the *congestion index*, has been used as a predictor of all-cause mortality in patients with heart failure.[12] The sum of the PAOP plus the RAP equal to 30 mm Hg obtained following therapeutic interventions and assessed early in the initial hospitalization may be able to predict heart failure survival as well as readmission rates at 6 months. The sum may be a warning signal for more aggressive intervention, but researchers indicate it requires further investigation.[12]

The PAPi is a hemodynamic parameter that predicts RV failure in patients with inferior wall MI and those undergoing LVAD implantation.[2,13-15] The PAPi is the ratio of PA pulse pressure to RAP (RV preload), calculated as the difference between systolic and diastolic PA pressures divided by RAP. Systolic PA pressure may reflect RV contractility, whereas PA diastolic pressure indicates left atrial filling pressure.[2] Lower PAPi values (2 or less) are clinically significant. The PAPi has been shown to predict RV failure and the need for a RV assist device after implantation of a LVAD. The PAPi has also been proven to be superior to the RAP to PAOP ratio and the RV stroke work index. Overall, the PAPi is a

Table 2: Hemodynamic Parameters Associated With Right Ventricular Dysfunction[a]

Variable	Reference Value	RV Dysfunction	Severe RV Dysfunction
RAP, mm Hg	<5	>10	>15
PAS, mm Hg PAD, mm Hg	15-25 8-15		
RAP/PAOP	Approximately 0.5	>0.63	>0.86
PVR, WU	0.25-2	3.6	After LVAD
PAPi	>2.0	<2.0	<1.5 <1.0 in AMI after LVAD < 1.85
RVSWI, g/m/m^2	51-61	<450	<300

Abbreviations: AMI, acute myocardial infarction; LVAD, left ventricular assist device; PAD, pulmonary artery diastolic pressure; PAOP, pulmonary artery occlusion pressure; PAPi, pulmonary artery pulsatility index; PAS, pulmonary artery systolic pressure; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RV, right ventricular; RVSWI, right ventricular stroke work index; WU, Wood unit.
[a] Data were derived from Konstam et al[1] and Lala et al.[8]

strong predictor of adverse clinical events in patients with severe symptomatic heart failure. Additionally, studies have shown the PAPi to be a significant predictor of death or hospitalization at 6 months.[2] Refer to Table 2 for a summary of the hemodynamic parameters associated with RV failure.

Management of RV Failure

The management of RV failure should target the cause. Right ventricular MI, contrasted with PH, requires different and specific approaches. A general approach is to optimize RV preload, afterload, and contractility.

Right ventricular preload requirements differ based on what the PVR may be. If normal in a patient with RV MI, RAP needs to be optimized to above normal levels to maintain cardiac output. If PVR is increased, giving volume can overload the RV causing displacement of the septum toward the LV and inhibiting LV filling. In general, recommendations are to keep the RAP between 8 and 12 mm Hg, adjusting as necessary for the individual patient. Monitoring central venous oxygen saturation and RAP through a central line can be helpful for assessing the right-sided filling pressures and CVP. Using a PAC with continuous monitoring of mixed venous oxygen saturation (SvO_2) capability may prove beneficial for monitoring cardiac output and systemic organ perfusion because of the potential for marked reductions in cardiac output and cardiogenic shock.[3]

In many patients, RV failure can be attributed to increased afterload. Management should be directed toward the primary cause (eg, correcting hypercapnia, alveolar hypoxia, or acidemia). Arterial oxygen saturation should be kept higher than 92%. The biggest challenge to RV function is pulmonary hypertension. In patients with pulmonary hypertension and RV failure, pulmonary vasodilators should be considered. For intubated patients receiving mechanical ventilation, inhaled nitric oxide or a prostacyclin derivative (eg, epoprostenol) can be administered. Both have been shown to improve the RV ejection fraction and overall pulmonary hemodynamics and to reduce ventilation-perfusion mismatching, thereby improving oxygenation.[3] Other pulmonary vasodilators that are administered intravenously, have rapid onset of action, and have short half-lives are also effective for reducing afterload.

Impaired RV myocardial contractility in acute RV failure is due to 3 interrelated mechanisms: (1) overstretching of the RV free wall, (2) derangements in cellular metabolism, and (3) myocardial ischemia caused by a reduction in coronary blood flow. Coronary perfusion of the RV decreases during systole in patients with PA hypertension. The PA pressure often exceeds normal systemic pressure. If systemic hypotension is present, care must be taken to avoid overloading the RV during fluid resuscitation. A vasopressor may be used to raise the systemic pressure in patients with RV failure. Vasopressors must also be used with care to avoid further increases in PA pressures. The use of an inotrope may be indicated only when preload, afterload, and myocardial ischemia have been addressed. Inotropes increase the risk of tachyarrhythmias, both atrial and ventricular.

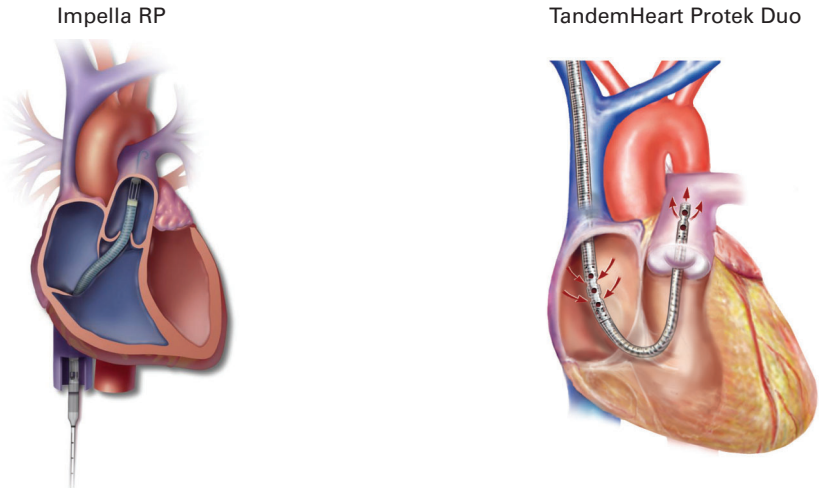

Figure 2: Temporary right ventricular assist devices. These devices are inserted percutaneously and advanced through the inferior or superior vena cava into the right atrium and right ventricle and out into the pulmonary artery. Venous blood in the right atrium is taken into the device via an intake valve and ejected into the pulmonary artery. This process reduces the work of the right ventricle and contributes to maintaining adequate left ventricular flow and cardiac output. Abiomed Impella RP image reproduced with permission of Abiomed. TandemHeart ProTek Duo image reproduced with permission of CardiacAssist, Inc.

Sinus tachycardia is considered a poor prognostic indicator in patients with RV failure.[3]

Mechanical Circulatory Support for RV Failure

Mechanical circulatory support for RV failure can be considered when medical management is insufficient. Mechanical circulatory support may be used as a bridge to recovery, ventricular assist device implantation, or heart transplantation. Research indicates that 42% to 75% of patients with acute RV failure may recover sufficiently to allow the mechanical circulatory support device to be removed. The type of device is determined by the cause of the RV failure.[16]

Newer RV assist devices (Impella RP, Abiomed; TandemHeart pVAD with Protek Duo cannula, CardiacAssist) have been used successfully to manage RV failure after RV infarction, cardiac surgery, LVAD implantation, and cardiac transplantation.[17] Refer to Figure 2 for examples of RV assist devices.

Extracorporeal membrane oxygenation (ECMO) may be considered, particularly in patients with massive pulmonary emboli, chronic thromboembolic pulmonary hypertension, or PA hypertension. Venoarterial ECMO pumps blood from the venous circulation to the arterial circulation, allowing the RV to rest while maintaining systemic oxygenation. Extracorporeal membrane oxygenation has been used for RV support in patients requiring a LVAD and for patients with acute RV failure after cardiac transplantation.[10] It also may be used as a bridge to recovery or as a bridge to another intervention. Occasionally patients receiving ECMO require further RV offloading with a RV assist device.

Mechanical support of the RV is being performed with increasing regularity in patients with RV failure and a LVAD.[16] Treatment of severe RV failure depends on prompt identification and biventricular support.[15] The results of some studies suggest that planned biventricular support following LVAD implantation in patients at high risk for RV failure produces superior outcomes.[15] Treatment of severe RV failure depends on prompt identification and early consideration of biventricular support.

Nursing Implications

Many nursing considerations must be evaluated when caring for a patient with RV failure. Understanding the clinical presentation, diagnostic approaches, and medical, pharmacologic, and device interventions is important. Nurses must have strong assessment skills to provide safe and effective care for this patient population. Early identification of the onset of RV failure is key. Attention to subtle changes in a

patient's hemodynamic profile allows for earlier intervention and prevention of cardiogenic shock.

Nurses may encounter a variety of clinical situations involving patients with RV failure. A patient with a history of heart failure may initially present following a right heart catherization, and depending on the hemodynamic assessment the provider may decide to leave the PAC in place and admit the patient to the intensive care unit for closer monitoring. The initial goal in this situation may be to optimize biventricular performance via diuresis and/or inotropic support. A patient with an inferior RV MI or anteroseptal MI might develop cardiogenic shock because of severe RV failure, and the cardiologist may elect to insert a temporary RV assist device to support the patient. A patient with severe pulmonary hypertension and RV failure might develop cardiogenic shock requiring ECMO support and pulmonary vasodilators.

Regardless of the clinical situation, the nurse is a key member of the multidisciplinary team and is responsible for understanding all of the mechanical and pharmacological interventions for each patient. The nurse must ensure that hemodynamic measurements are obtained using best practices and that variances in measurements are avoided. Equally as important, the nurse must focus on basic nursing care to protect the patient's skin, prevent hospital-acquired infections, provide psychological support, and educate the patient and the patient's family members.

Conclusions

Right ventricular failure represents a very real threat to patients' survival in the acute situations presented in this article. The use of a PAC to treat this patient population has proven to be beneficial, especially when combined with new hemodynamic parameters. Many advances have been made in the understanding of RV failure, but experts have indicated that more research is needed.

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CE Evaluation Instructions

This article has been designated for CE contact hour(s). The evaluation tests your knowledge of the following objectives:

1. Differentiate the normal physiologic aspects of the right ventricle from those of the left ventricle.
2. Describe the pathophysiological processes contributing to acute onset of right ventricular failure.
3. Discuss the management of right ventricular failure in the context of preload, afterload, and contractility.

Contact hour: **1.0**
Synergy CERP Category: **A**

To complete evaluation for CE contact hour(s) for article #ACC3111, visit www.aacnacconline.org and click the "CE Articles" button. No CE evaluation fee for AACN members. This expires on March 1, 2023.

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