

Right-sided heart failure

Give the right side of the heart the attention it's due.

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RIGHT-SIDED HEART FAILURE (HF) doesn't get the attention that left-sided HF receives, but it's an important factor in HF symptom severity and mortality. Right-sided HF, which can cause or worsen pulmonary hypertension and left-sided HF, is defined as a structural or functional abnormality of the right heart circulatory system that impedes blood delivery to the pulmonary system or elevates venous pressures at rest or with exercise. Right ventricular dysfunction is the terminology used when a structural or functional abnormality that affects the right side of the heart is present, but patients are asymptomatic. Understanding right-sided HF pathophysiology, causes, and medical management can help nurses facilitate evidence-based care for their patients.

How the right ventricle works

The right ventricle is a thin-muscle, crescent shaped, low-pressure chamber that can accommodate large volumes of blood, which it

ejects across the low resistance of the pulmonary circuit (the circulation between the heart and the lung).

Blood enters the ventricle from the venous circulation (systemic venous return) and exits to the pulmonary circuit. The right ventricle's workload is much lower than the left (its contraction has lower peak systolic pressure and higher steady-state volume than the left ventricle), linking right ventricle function to its preload (circulating blood volume) and afterload (the force against which the right ventricle needs to generate enough pressure to overcome the pressure in the pulmonary vasculature), with afterload being the primary determinant of normal right ventricular function.

When pulmonary artery pressure rises (even slightly), right ventricle stroke volume decreases. The right ventricle becomes inefficient and uses more energy to maintain the balance between ventricular load and pulmonary arterial pressure. Unlike the left ventricle, which is perfused primarily during diastole, right ventricle coronary perfusion occurs during systole and diastole. If the right ventricle becomes pressure-overloaded, right ventricle coronary perfusion pressure (blood flow) decreases, increasing the risk for right ventricular ischemia. Ultimately, the right ventricle works best when the energy needed to produce contraction (to eject blood) occurs under low pressure.

Causes of right-sided HF

Right-sided HF has many causes. In adults without congenital heart diseases, it can occur after myocarditis, right ventricular myocardial infarction, cardiac surgery or transplantation,



LEARNING OBJECTIVES

1. Describe physiologic changes associated with right-sided heart failure (HF).
2. Discuss clinical findings of right-sided HF.
3. Discuss management of right-sided HF.

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Ventricular interdependence

Ventricular interdependence causes the interventricular septum to shift to the left and flatten, which causes the left ventricle cavity to become compressed and “D”-shaped, rather than maintaining its normal “O” shape.

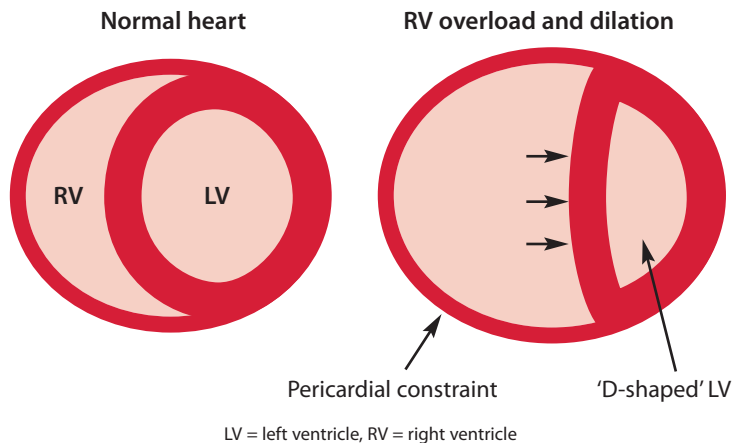
acute pulmonary embolus, tricuspid valve regurgitation, pulmonary valve disease, and arrhythmogenic right ventricular cardiomyopathy (a rare genetic disease of cardiac myocytes). Primary pulmonary arterial hypertension causes pulmonary vasculature alterations that lead to increased pulmonary resistance and right-sided HF, and alterations in pulmonary vasculature (for example, chronic thromboembolic disease) may lead to increased pulmonary vascular resistance, pulmonary embolus, and, ultimately, right-sided HF.

More commonly, though, right-sided HF occurs as left-sided HF advances and secondary pulmonary hypertension becomes prominent. Secondary pulmonary hypertension can occur in HF with reduced ejection fraction (HFrEF) and in HF with preserved ejection fraction (HFpEF), including restrictive cardiomyopathies. When HFpEF is associated with right-sided HF, distinguishing whether the cause is a primary right ventricular pathology or secondary pulmonary hypertension can be difficult because the right ventricle is afterload-dependent (it depends on pressure in the pulmonary artery). Secondary hypertension that results from chronic lung conditions (such as adult respiratory distress syndrome and chronic obstructive lung disease) also can lead to right-sided ventricular dysfunction and HF.

Right-sided HF prevalence can be as high as 50% of patients who are hospitalized for acutely decompensated HFrEF because decompensation frequently is coupled with pulmonary hypertension. According to Amsalleem and colleagues, Harjola and colleagues, and Ponikowski and colleagues, prevalence varies with HFrEF etiology. For example, 60% of patients with dilated cardiomyopathy and 16% with ischemic cardiomyopathy have right-sided HF. In patients with HFpEF, 33% to 50% had right-sided HF. For patients who are in the early postoperative phase after receiving a left ventricular assist device, 20% develop right-sided HF.

Acute right-sided HF

Acute right-sided HF occurs when right ventricular afterload abruptly increases (caused by pulmonary embolus, hypoxia, or acidosis) or right ventricle contractility decreases (frequently caused by ischemia, myocarditis, or postcardiotomy shock from perioperative my-



ocardial ischemia or injury). The right ventricle can handle volume changes (except excessive transfusion), but a sudden change in pressure can abruptly decrease right ventricular stroke volume. If contractility is impaired by an abrupt event, the right ventricle dilates, prompting tricuspid regurgitation, which exacerbates right ventricular dilation.

Pericardial constraint that prevents the right ventricle from enlarging outward creates ventricular interdependence, which disrupts left ventricular filling. (See *Ventricular interdependence*.) As left ventricular end-diastolic pressure increases and transmural filling pressure decreases, diastolic filling is impeded. Because less blood is available during left ventricular systole, systemic hypoperfusion results. As right heart filling pressures increase, systemic venous congestion increases, leading to hepatic congestion and impaired renal function, which can aggravate fluid retention and worsen right-sided HF.

Chronic right-sided HF

Chronic right-sided HF is associated with increased right ventricular afterload caused by pulmonary hypertension, which typically is caused by left-sided HF. Another common cause is chronic volume overload from left ventricular remodeling, as seen in HFrEF and HFpEF. If the right ventricle has myocyte loss or becomes hypertrophied and fibrotic, right ventricular systolic pressure increases and decompensation leads to a rise in peripheral vascular resistance and right atrial pressure. Over time, cardiac output declines.

Signs and symptoms

Signs and symptoms of acute and chronic right-sided heart failure (HF) worsen as right ventricular function worsens.

Acute right-sided HF

Signs

- Atrial and ventricular dysrhythmias
- High transaminase level
- Increased jugular venous pressure
- Prominent V wave during hemodynamic monitoring
- Right-sided third heart sound
- Tricuspid valve holosystolic murmur

Symptoms

- Cool extremities
- Diaphoresis
- Dyspnea (from diminished peripheral oxygen delivery)
- Hypotension
- Listlessness
- Pulmonary edema (if secondary to left-sided HF)
- Tachycardia

Chronic right-sided HF

Signs

- Atrial tachydysrhythmias (if elevated right atrial pressure)
- Hepatic congestion (right upper-quadrant discomfort) and reduced perfusion
 - Hyperbilirubinemia (in patients with pulmonary hypertension)
 - Moderate-to-severe tricuspid regurgitation (versus mild severity)
 - Prolonged prothrombin time and elevated international normalized ratio
 - Serum markers of cholestasis (elevated bilirubin and alkaline phosphate)
- Interstitial edema and increased inter-abdominal pressure
 - Malnutrition and reduced absorption
- Peripheral edema (most prominent sign)
- Reduced serum albumin
- Visceral edema (caused by toxins in the gut lumen that leak into the bloodstream)
- Worsening renal function (due to increased central venous pressure and renal vein pressure)
 - Decreased urine output
 - Fluid retention
 - Increased diuretic dosage
 - Increased serum creatinine and blood urea nitrogen reflecting worsened status

Symptoms

- Exercise intolerance (due to reduced cardiac output)
- Fatigue (due to reduced cardiac output)
- Ventricular tachycardia/heart block (can lead to sudden cardiac death)

Acute-on-chronic left-sided HF signs and symptoms

- Ascites
- Hepatomegaly
- Peripheral edema

If declining cardiac output is accompanied by declining pulmonary artery pressure in the presence of high pulmonary vascular resistance, mortality risk increases. In patients with an intact pericardium and right ventricular dilation, ventricular interdependence can lead to reduced cardiac output, impaired coronary blood flow, and increased peripheral and abdominal congestion.

Clinical characteristics and evaluation

Clinical characteristics of right-sided HF are caused by increased pressure in the right atrium (central venous pressure) and reduced left ventricular filling as a result of the effect of ventricular interdependence. Elevated central venous pressure impedes lung lymphatic drainage, so in patients with pulmonary hypertension resulting from left-sided heart diseases, lung fluid clearance decreases and excessive pulmonary edema can lead to pleural effusion. (See *Signs and symptoms*.)

The kidneys, liver, and GI tract also are affected by chronic right-sided HF. Poor renal function and end-stage liver disease can increase mortality risk. In addition, renal function deterioration may be a marker of worsening cardiac function as a result of HF. If the patient is experiencing worsening renal function and elevated right-sided filling pressures, placing a pulmonary artery catheter may help determine true volume status and the need for loop diuretic therapy.

Patients with severe right-sided HF may be emaciated, tachypneic, and cyanotic, and they may have elevated jugular venous pressure. For patients with a noncompliant right ventricle, a Kussmaul's sign may unmask venous hypertension. (See *Kussmaul's sign*.) If pulmonary hypertension is the cause of right-sided HF, clinicians may hear a prominent pulmonic component of the second heart sound on auscultation. If coronary heart disease is part of the diagnosis, the pulmonic component of the second heart sound may be soft or absent.

Diagnostic testing

In addition to physical assessment, testing for right-sided HF consists of 12-lead ECG, echocardiogram (or alternative cardiac size and function tests, such as cardiac magnetic resonance imaging or positive emission

tomography scan), and hemodynamic assessment.

ECG

Chronic right-sided HF is associated with a right-axis deviation and large P wave amplitude in leads II, III, and aVF. Sinus tachycardia is common, and the V1 lead may have a qR pattern. In addition, atrial fibrillation and atrial flutter are common.

Echocardiogram

The right ventricle's retrosternal position, proximity to the chest wall, and complex non-geometric shape create inter-observer variability on echocardiograms. However, when using tissue Doppler, tricuspid annular plane systolic excursion (TAPSE) and right ventricular functional area change (RVFAC) are reproducible, feasible, easily obtained measures that reflect longitudinal right ventricular shortening. Reference limits of right ventricular function based on TAPSE and RVFAC are ≥ 1.8 cm (≥ 18 mm) and $\geq 35\%$, respectively.

Hemodynamic assessment

Right atrial pressure >15 mmHg is a marker of right ventricular dysfunction, especially when the elevation is disproportionate relative to the rise in pulmonary artery wedge pressure (normal right atrial/pulmonary artery wedge pressure ratio is about 0.5). In addition, elevated right atrial pressure will cause large atrial contraction waves (A waves) and large V waves, which signal a poorly compliant right atrium and tricuspid regurgitation.

Medical management

Managing acute right-sided HF focuses on reduced volume and preload, enhanced myocardial contractility, and reduced right ventricular afterload with pharmacologic therapies and mechanical circulatory support. (See *Acute right-sided HF: Medical management*.) In acute right-sided HF, the goal is decreased left atrial pressure to reduce congestion and central venous pressure to <12 mmHg (ideal is <8 mmHg) and reduced pulsatile right ventricular loading.

When medical therapies aren't effective, temporary mechanical circulatory support with axial flow or extracorporeal centrifugal flow devices provide short-term right ventricular support. Surgical options (tricuspid and

Kussmaul's sign

If a patient's right ventricle is noncompliant, Kussmaul's sign (increased right internal jugular venous pressure on inspiration) may be present. Check for this sign by performing the abdominojugular reflux test. Elevate the head of the bed to 45 degrees, and ask the patient to breathe calmly. Note the pulsations in the internal jugular vein, then use the palm of your hand to apply firm pressure for at least 15 seconds (various sources list a range from 10 to 60 seconds) on the right upper-quadrant of the abdomen while you observe the vein. A positive Kussmaul's sign is defined as an observed increase in internal jugular venous pressure (>3 cm) on inspiration that is sustained for more than 15 seconds.

Acute right-sided HF: Medical management

Medically managing acute right-sided heart failure (HF) includes managing volume, improving systolic contraction, and decreasing pulmonary and systemic vascular resistance

Volume management

Manage volume using I.V. loop diuretics.

- Response may be suboptimal as a result of hypotension, elevated central venous pressure, renal venous congestion, low cardiac output, or oliguric acute kidney injury.
- Administer in patients with volume overload even if they're hypotensive, but manage low blood pressure by repositioning (flat or flatter in the bed) or drug therapies that enhance contractility.

If the patient doesn't respond to I.V. loop diuretics, consider these renal replacement therapies:

- Veno-venous hemofiltration
- Ultrafiltration (Assess blood pressure to ensure maintenance of optimal intravascular volume. Don't remove fluid at a rate that exceeds the ability of extravascular fluid to shift into intravascular space.)

Vasoactive therapies

These therapies reduce afterload to enable more effective systolic contraction and to decrease pulmonary and systemic vascular resistance:

- I.V. vasodilators
 - Nitroglycerin
 - Sodium nitroprusside
- Oral vasodilators
 - Phosphodiesterase-5 inhibitors

Enhance contractility with short-term infusions of I.V. inotropes, such as

- dobutamine, which has a short half-life with a rapid onset and offset and a lower risk of hypotension than milrinone
- milrinone (if the patient also is receiving beta-blocker therapy), which is less likely than dobutamine to cause drug tolerance but more likely to cause hypotension when administered as a bolus.

If the patient is hypotensive (systolic blood pressure <80 to 90 mmHg), optimal perfusion can be achieved using a combination drug with inotropic and vasopressor properties to improve coronary artery perfusion, augment arterial pressure, and reduce the risk of right ventricular myocardial ischemia.

- Alpha agonist (phenylephrine)
- Dopamine
- Norepinephrine
- Epinephrine

Chronic right-sided HF: Medical management

Medically managing chronic right-sided heart failure (HF) includes volume management, beta blockade, and arterial vasodilation, as well as treating pulmonary arterial hypertension.

Volume management

Volume is managed using loop diuretics and sodium restriction.

- Loop diuretics
 - Intensity of diuretic should be based on cause and severity of right-sided HF and coexisting renal disease.
 - Serum laboratory monitoring is required to prevent development of prerenal azotemia.
 - Large diuretic doses may be required due to upregulation of the renin-angiotensin-aldosterone system:
 - Visceral edema reduces diuretic absorption and tubular drug delivery.
 - Chronic sodium-potassium-chloride blockade from diuretic therapy can lead to hypertrophied distal nephron and rebound sodium absorption.
 - Combination therapy of loop and thiazide diuretics may augment natriuresis (via sequential nephron blockade)
 - The loop diuretic torsemide has consistent absorption (80% bioavailability) compared to furosemide (bioavailability ranges from 10% to 90%).
- Sodium restrictions (Recommendations are imprecise.)
 - Restrict sodium to reduce congestion in bi-ventricular HF or isolated right-sided HF
 - In 2013, the National Heart Failure guidelines from the American Heart Association/American College of Cardiology for dietary sodium restrictions were revised from <2,000 mg/day to <3,000 mg/day. The right-sided HF 2018 scientific statement also recommends <3,000 mg/day.
 - Restricting fluid to 1.5 to 2.0 liters/day may reduce congestion and hyponatremia.

Renin-angiotensin-aldosterone system inhibitors and beta blockers

- Small-scale studies support the use of the following medications for patients who have chronic HF with reduced ejection fraction: an angiotensin-converting enzyme inhibitor or an angiotensin receptor blocker, beta-blocker, and mineralocorticoid receptor blocker.

Pulmonary arterial hypertension management

Pulmonary artery vasodilator therapy relieves right ventricular afterload.

- Prostaglandin analogs include
 - I.V. epoprostenol
 - inhaled iloprost
 - inhaled treprostinil
- Phosphodiesterase-5 inhibitors include
 - sildenafil
 - tadalafil
- Endothelin receptor antagonists provide short-term hemodynamic benefits but don't reduce morbidity and mortality.
 - Right-sided HF is associated with hepatic congestion. Endothelin receptor antagonists increase hepatic aminotransferases, so the patient's liver function should be monitored.

pulmonary artery procedures) offer intermediate-term support. When patients meet inclusion and exclusion criteria, total artificial

heart, durable mechanical circulatory support (left ventricular assist) device, and cardiac transplantation offer long-term support.

Management goals for chronic right-sided HF include relieving congestion, inhibiting neuroendocrine stimulation with medications that block or inhibit the actions of hormones of the renin-angiotensin-aldosterone system and beta-adrenergic system, and increasing cardiac output and right-sided ejection fraction. If the patient has pulmonary arterial hypertension, management focuses on vasodilating the pulmonary vasculature. (See *Chronic right-sided HF: Medical management*).

Nursing implications

Understanding the diverse pathophysiology of acute and chronic right-sided HF helps nurses recognize and identify these conditions and promptly communicate with providers to enhance right ventricular unloading (for example, avoiding excessive volume administration) and improve left ventricular function. Right ventricular unloading is important because it may minimize central and renal venous engorgement and reduce the risk or severity of cardiorenal syndrome.

Nurses also can provide patient education about sodium and fluid restriction goals; however, large-scale research study findings on these restrictions aren't currently available. In addition, nurses can advocate for optimal medical therapies based on the cause of right-sided HF and provide psychological support to patients and families, given the poor prognosis.

Helping patients

Right-sided HF is associated with impaired renal and hepatic function that increases morbidity and mortality. Management is aimed at palliative support to relieve congestion, improve exercise tolerance, and reduce afterload. Nurses can help patients understand the pathophysiology of their symptoms and the importance of seeking care when symptoms worsen.

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Access references at myamericannurse.com/?p=74385.

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Please mark the correct answer online.**1. Afterload refers to the**

- blood volume that is ejected by the right ventricle.
- total blood volume in the heart during systole.
- force the left ventricle needs to generate to overcome the pressure in the pulmonary vasculature.
- force the right ventricle needs to generate to overcome the pressure in the pulmonary vasculature.

2. Which statement about secondary pulmonary hypertension and heart failure (HF) is correct?

- It occurs in HF with moderate ejection fractions and left-side dysfunction.
- It occurs in HF with reduced ejection fraction and with preserved ejection fraction.
- It occurs only in HF with reduced ejection fraction.
- It occurs only in HF with preserved ejection fraction.

3. Which statement related to acute right-sided HF is correct?

- It's commonly caused by hypertension in the pulmonary vasculature.
- It occurs when right ventricular afterload abruptly increases or right ventricle contractility decreases.
- It occurs when right ventricular afterload abruptly decreases or right ventricle contractility increases.
- It's commonly caused by hypertrophy of the left ventricle.

4. Ventricular interdependence causes the

- left ventricle cavity to become "O"-shaped.
- right ventricle cavity to become compressed.
- interventricular septum to shift to the right.
- interventricular septum to shift to the left.

5. Which of the following would lead you to suspect your patient was experiencing acute-on-chronic left-sided HF?

- Dehydration
- Pulmonary edema
- Hepatomegaly
- Hypertension

6. Which of the following physical assessment findings would lead you to suspect your patient was experiencing acute right-sided HF?

- Right-sided third heart sound
- Right-sided fourth heart sound
- Bicuspid valve holosystolic murmur
- Aortic valve holosystolic murmur

7. An ECG finding that could indicate chronic right-sided HF is

- left axis deviation and large P wave amplitude in leads II, III and aVF.
- right axis deviation and large P wave amplitude in leads II, III and aVF.
- sinus bradycardia.
- a qR pattern in lead aVF.

8. A sign of right ventricular dysfunction is a right atrial pressure that is

- <10 mmHg.
- <15 mmHg.
- >15 mmHg.
- >10 mmHg.

9. Which of the following would be most helpful for managing volume in a patient with acute right-sided HF?

- Loop diuretics
- Beta blockers
- Phosphodiesterase-5 inhibitors
- Oral vasodilators

10. Which of the following medications might be used as a short-term I.V. infusion to enhance contractility in a patient with acute right-sided HF?

- Nitroglycerin
- Phosphodiesterase-5
- Sodium nitroprusside
- Dobutamine

11. Which statement about the use of loop diuretics in patients with chronic right-sided HF is correct?

- Torsemide has consistent absorption (80% bioavailability).
- Furosemide has consistent absorption (70% bioavailability).
- Large diuretic doses usually are not required because of upregulation of the renin-angiotensin-aldosterone system.
- Large diuretic doses may be required because of downregulation of the renin-angiotensin-aldosterone system.

12. Although dietary sodium restriction recommendations are imprecise, guidelines from the American Heart Association/American College of Cardiology recommend that patients with chronic right-sided HF keep their sodium intake to

- <1,000 mg/day.
- <2,000 mg/day.
- <3,000 mg/day.
- <4,000 mg/day.

13. An example of a phosphodiesterase-5 inhibitor is

- sildenafil.
- I.V. epoprostenol.
- treprostinil.
- inhaled iloprost.

14. A patient who has chronic right-sided HF with reduced ejection fraction may be prescribed all of the following *except*

- an angiotensin-converting enzyme inhibitor.
- a mineralocorticoid receptor stimulator.
- an angiotensin receptor blocker.
- a beta blocker.