Reducing blood pressure in acute decompensated heart failure

By Daniel L. Arellano, RN, ACNP-BC, CCRN, CEN

Heart failure (HF) affects an estimated 5.7 million individuals in the United States age 20 and older, with 870,000 new cases of HF diagnosed annually. In 2010, there were over 1 million primary care provider office visits and 676,000 ED visits with a primary diagnosis of acute HF, making it one of the leading causes of hospital admissions. A review of the Acute Decompensated Heart Failure National Registry finds that almost 50% of patients admitted with HF had a BP of greater than 140/90 mm Hg upon admission.

It is therefore paramount for clinicians to understand the treatment modalities for acute hypertensive HF. The main treatment goals are to establish euvolemia, reduce afterload, and restore adequate perfusion and oxygenation. The purposes of this article are to discuss the pathophysiology of acute decompensated HF, review pharmacologic agents useful in reducing BP in the setting of acute HF, and highlight pharmacologic agents to avoid.

Pathophysiology of acute decompensated HF

HF is characterized by systemic perfusion that is inadequate to meet metabolic requirements or to accommodate systemic venous return resulting from an impaired myocardium. Damage to the myocardium results from a variety of causes, including: coronary artery disease, hypertension, diabetes mellitus, valvular disease, infection, and cardiac toxins. HF can be divided into two categories: systolic and diastolic, which can affect both the right and left ventricles. Systolic HF is characterized by impaired ventricular contraction and ejection; diastolic HF is characterized by impaired relaxation and ventricular filling. Approximately 70% of patients experience systolic HF compared to 30% with diastolic HF. These conditions are not mutually exclusive, and most patients with systolic HF have some component of diastolic HF as well.

The spectrum of HF is also described as it relates to the left and right ventricle. Left ventricular (LV) systolic dysfunction is defined as an LV ejection fraction of less than 40%. Common causes include ischemic heart disease, uncontrolled hypertension, and cardiac toxins. Damage to cardiac myocytes lowers cardiac contractility and causes a decrease in systemic perfusion. The resulting increase in LV end-diastolic volume leads to elevated left atrial pressures, which in turn increases the pressure inside lung capillaries. Increased pressure forces fluid from the pulmonary capillaries into the alveoli and causes pulmonary congestion and shortness of breath. LV diastolic dysfunction presents similar symptoms when impaired relaxation and ventricular filling cause hypoperfusion and pulmonary congestion.

Right ventricular dysfunction is most commonly caused by either hypoxic pulmonary disease or LV dysfunction. When the right ventricle is unable to fill or eject adequate amounts of blood, it leads to an elevated right atrial pressure. This contributes to increased pressure in the vena cava and impaired venous drainage from the liver, gastrointestinal tract, and lower extremities, resulting in hepatomegaly, abdominal pain, and peripheral edema.

Compensatory mechanisms. The body has a series of compensatory mechanisms to combat hypoperfusion resulting from HF. These compensatory mechanisms include the Frank-Starling Mechanism, neurohormonal activation, and ventricular remodeling. The Frank-Starling law of the heart states that increases in end-diastolic volume creates a stretch on the myocardium, which responds with an increase in cardiac output. This compensatory mechanism is typically exhausted as HF worsens.

Neurohormonal activation is another compensatory mechanism utilized to regulate cardiac output. Decreases in BP stimulate the sympathetic nervous system to release catecholamines (norepinephrine and epinephrine). This causes an increase in heart rate, BP, and cardiac contractility while also triggering peripheral vasoconstriction to shunt blood to critical organs. The kidneys sense a decrease in cardiac output and activate the renin-angiotensin aldosterone system, which causes vasoconstriction and sodium retention to increase BP.

Chronic activation of these systems causes the third compensatory mechanism of ventricular remodeling. Ventricular remodeling is defined as a change to the size, shape, structure, and function of the ventricle. Chronic hemodynamic stress can lead to increased ventricular hypertrophy, which can cause increases in BP and increased workload of the heart. Over time, the hypertrophic tissue can become fibrotic and impair contractility.
Reducing BP in acute decompensated HF

As discussed, almost 50% of patients admitted with HF had a BP of greater than 140/90 mm Hg upon admission to the hospital. The pathologic mechanisms involved are likely a combination of the Frank-Starling Mechanism, neurohormonal activation, and ventricular remodeling. Therefore, clinicians must guide antihypertensive therapy to reduce preload, reduce afterload, and optimize contractility (see Pharmacologic agents to reduce BP in hypertensive acute HF). Practice guidelines released from the American College of Cardiology and the American Heart Association recommend hospitalization during acute HF exacerbations. Therefore, most of the interventions discussed below are parenteral therapies intended for administration and monitoring in the hospital setting.

**Diuresis.** Studies have shown that early initiation of diuresis can reduce mortality in patients presenting with evidence of fluid overload and acute decompensated HF. The initial dose should be equivalent to or exceed the daily dose for patients already receiving diuretic therapy. Administration can be achieved either by I.V. bolus or continuous infusion. Close observation of urinary output and frequent assessment of pulmonary congestion are indicated. Modifications in dose may be required to relieve symptoms, reduce volume, and avoid hypotension. Nursing interventions must include strict intake and output, daily body weights, and serial labs to monitor electrolytes.

Loop diuretics, such as furosemide, torsemide, or bumetanide, are recommended to achieve diuresis in patients with acute HF. Subsequent dosing and/or the addition of a second diuretic, such as a thiazide, may be required. Patients with a history of kidney failure or those experiencing refractory congestion may require the use of ultrafiltration with hemodialysis to expedite euvoelma.

**Vasodilators.** The use of vasodilators to reduce BP in acute hypertensive HF is strongly recommended. Nitroglycerin helps promote venous dilatation, decreases preload, and helps to rapidly reduce pulmonary congestion. It can be administered either orally, sublingually, transdermally, or I.V. Nitroglycerin has a half-life of 1 to 3 minutes, making it an excellent drug for rapid I.V. titration during acute hypertensive, decompensated HF.

Common adverse reactions include flushing, hypotension, headache, and reflex tachycardia. Nitroglycerin is the single best option for reducing BP in acute decompensated HF because of its short half-life and powerful vasodilatory properties.

Sodium nitroprusside may also be useful in reducing BP in acute HF. It acts as both a venous and arterial dilator to reduce both preload and afterload while dilating pulmonary vasculature. The data demonstrating sodium nitroprusside’s efficacy in acute HF are limited. However, it is certainly an effective therapy for reducing BP in acute HF based on its mechanism of action. Sodium nitroprusside can cause marked hypotension, and therefore, invasive BP monitoring with the use of an arterial line is recommended. Therapy duration should be minimized because prolonged infusions have been associated with thiocyanate toxicity, particularly in the setting of kidney dysfunction.

The use of I.V. nesiritide (human brain natriuretic peptide) in hypertensive acute HF is somewhat controversial. Nesiritide acts as brain natriuretic peptide to decrease LV filling pressure by binding to vascular smooth muscle, causing arterial and venous vasodilation. Nesiritide has a much longer half-life than nitroglycerin or nitroprusside, and adverse reactions such as hypotension may persist longer.

Effects on cardiac output, urinary output, and sodium excretion vary. One study found the use of nesiritide in conjunction with diuretics more rapidly decreased the severity of dyspnea compared to diuretics alone. However, several larger trials found only a slight impact on dyspnea and no improvement in mortality or hospital readmission rates. It is important to monitor creatinine levels during and after therapy, as nesiritide may decrease kidney function. Nesiritide use has decreased, but it remains an alternative therapy for BP management in HF.

Morphine sulfate offers another alternative strategy to reduce BP in acute hypertensive HF. Morphine causes venous pooling and peripheral vasodilation, which subsequently decrease myocardial oxygen demand and reduce preload. These mechanisms cause a reduction in BP and decrease the heart’s workload. Morphine sulfate also alleviates dyspnea and pain associated with acute decompensated HF. Small to moderate I.V. doses are ideal to avoid oversedation and acute hypotension.

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**Pharmacologic agents to reduce BP in hypertensive acute HF**

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<td>• Initial dose should equal or exceed chronic dose</td>
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<th>Vasodilators</th>
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<td>• Nitrates</td>
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<td>• Nitroprusside infusion</td>
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<td>• Nesiritide</td>
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<td>• Morphine sulfate</td>
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<td>• Hydralazine</td>
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Hydralazine may also be useful in reducing BP in acute hypertensive HF. It acts as a direct arterial vasodilator but has little effect on the venous system. It has been shown to be safe in patients experiencing acute HF and can be administered either via I.V. or orally. Hydralazine has been shown to work best when used in combination with nitrates, particularly in the Black population. Hydralazine use is contraindicated in patients with coronary artery disease, mitral valve rheumatic heart disease, and also in patients experiencing an elevated heart rate due to its activation of the baroreflex and subsequent reflex tachycardia.

■ ACE inhibitors
The literature provides limited guidance for the use of the I.V. angiotensin-converting enzyme (ACE) inhibitor enalaprilat in this setting. However, based on mechanism of action, it is a suitable alternative to reduce BP in acute hypertensive HF. Enalaprilat acts to inhibit the conversion of angiotensin I to angiotensin II in the renin angiotensin aldosterone system, thereby reducing BP. ACE inhibitors have been shown to reduce BP and mortality in chronic HF; however, there are limited studies to indicate efficacy in reducing BP in acute HF. It is recommended to closely monitor kidney function when using high amounts of diuretics to assess for kidney dysfunction.

■ Pharmacologic agents to avoid
Calcium channel blockers and beta-blockers should be avoided when managing hypertensive acute HF. Calcium channel blockers act by decreasing the transport of calcium into the cardiac tissues and within blood vessels. This results in vascular relaxation, increased supply of blood and oxygen to the heart, and a reduction in cardiac workload. The decrease in calcium influx can have negative inotropic and chronotropic effects, which negate the goal of increasing cardiac contractility in acute decompensated HF.

Beta-blockers should also be avoided when managing hypertensive acute HF. Beta-blockers act by thwarting the effects of sympathetic nerve stimulation and circulating catecholamines at beta-adrenoreceptors within the heart and throughout the body. This is the primary effect desired in patients with chronic HF because it reduces myocardium sensitivity to hypertensive compensatory mechanisms. However, many clinicians elect to decrease total beta-blocker dosing by halving or discontinuing use in the setting of acute decompensated HF requiring hospitalization to minimize the effect of decreased inotropy. Overall, the use of calcium channel blockers and beta-blockers is not recommended to reduce BP in acute decompensated HF due to negative inotropic effects.

■ Understanding principles
Acute decompensated HF is one of the leading causes of hospital admission. It is important for clinicians to understand the principles of reducing BP in acute hypertensive HF. The main goals are to establish euvolemia via diuresis, reduce BP via vasodilation, and ensure adequate perfusion and oxygenation. This can best be achieved with a combination of loop diuretics, nitrates, and close observation.

REFERENCES
7. Lilly LS. Pathophysiology of Heart Disease: A Collaborative Project of Medical Students and Faculty. 5th ed. Baltimore: Lippincott Williams & Wilkins; 2011.
18. Daniel L. Arellano is critical care nurse practitioner at Division of Anesthesiology and Critical Care University of Texas MD Anderson Cancer Center, Houston, TX, and an instructor of the Nursing Department at Family Health University of Texas Health Science Center School of Nursing, Houston, Tex. The author has disclosed that he has no financial relationships related to this article.

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