In patients with chronic heart failure, fluid retention (or hypervolemia) is often the stimulus for acute decompensated heart failure that requires hospitalization. The pathophysiology of fluid retention is complex and involves both hemodynamic and clinical congestion. Signs and symptoms of both hemodynamic and clinical congestion should be assessed serially during hospitalization. Core heart failure drug and cardiac device therapies should be provided, and ultrafiltration may be warranted. Critical care, intermediate care, and telemetry nurses have roles in both assessment and management of patients hospitalized with acute decompensated heart failure and fluid retention. Nurse administrators and managers have heightened their attention to fluid retention because the Medicare performance measure known as the risk-standardized 30-day all-cause readmission rate after heart failure hospitalization can be attenuated by fluid management strategies initiated by nurses during a patient’s hospitalization. (Critical Care Nurse. 2012;32[2]:20-32,34)

The term heart failure is defined as a clinical syndrome of decreased exercise tolerance and fluid retention due to structural heart disease (eg, cardiomyopathy or valvular disorders). Acute decompensated heart failure denotes development of progressive signs and symptoms of distress that require hospitalization in patients with a previous diagnosis of heart failure.1 Although many markers of acute decompensated heart failure are related to fluid retention,2 patients may not have classic signs and symptoms of clinical congestion, such as respiratory distress, crackles, interstitial/alveolar edema, elevated jugular venous pressure or jugular venous distension, findings on chest radiographs, and an S₃ heart sound. Patients may have hemodynamic congestion, defined as an increase in left ventricular filling and/or intravascular pressures.3 Hemodynamic congestion is a form of fluid retention that occurs earlier than does clinical congestion and indicates that the clinical manifestations of fluid retention may be imminent.3 Even when signs and symptoms of clinical congestion are relieved, patients may still have hemodynamic congestion that could lead to progression of heart failure and worsening prognosis.3 Thus, optimal assessment of fluid status and management of both hemodynamic and clinical congestion are integral components of nursing care.

Congestion in any form is a hallmark of acute decompensated heart failure that stems from a cyclical detrimental process involving low fluid volume and raised intravascular pressures, which ultimately leads to reduced cardiac output and systemic hypoperfusion.
cardiac output, arterial underfilling, activation of neurohormonal systems, and dysregulation between the heart and kidneys.\(^4\) Two large US heart failure registries, Acute Decompensated Heart Failure Registry (ADHERE)\(^5\) and Organized Program to Initiate Lifesaving Treatment in Hospitalized Patients with Heart Failure (OPTIMIZE-HF)\(^6\) collected data on the clinical features of patients hospitalized for acute decompensated heart failure. According to both registries, cardiogenic shock was uncommon, accounting for 2% or less of all cases. However, hypervolemic states were prevalent and often included dyspnea (89% and 90%, respectively), crackles (67% and 65%, respectively), and peripheral edema (66% and 65%, respectively) regardless of whether or not the ejection fraction was less than 40% (indicating systolic left ventricular dysfunction) or normal (indicating heart failure with preserved ejection fraction, or diastolic dysfunction).

In this review, I briefly describe the complex pathophysiological processes of hypervolemia in hospitalized patients with decompensated heart failure and discuss fluid management strategies, many of which can be nurse-led or nurse-facilitated. Critical care, intermediate care, telemetry, and general care nurses have many opportunities to assess patients’ fluid status, correct hypervolemia, and ensure that fluid management strategies are in place before a patient is discharged. Patients must also understand their roles in assessing, monitoring, and treating hypervolemia at home to optimize health-related clinical outcomes.

Pathophysiology of Hypervolemia

In patients with normal hemodynamic, neurohormonal, cardiac, and renal processes, an increase in total blood volume is associated with an increase in renal levels of sodium and water excretion\(^4\) (Figure 1). Renal excretion of sodium and water is due to a series of reflexes that maintain normal total body volume when atrial pressure increases. Thus, any increase in atrial pressure leads to a diminished release of arginine vasopressin (antidiuretic hormone), increased release of atrial natriuretic peptide, and decreased renal sympathetic tone.\(^8\)

However, in patients with acute decompensated heart failure, total blood volume is not the determinant of renal excretion of sodium and water; the integrity of arterial circulation is a key factor in euvoemia.\(^4\) Patients with heart failure have either decreased cardiac output that causes underfilling of the arterial circulation or high cardiac output that prompts systemic arterial vasodilatation and underfilling of the arterial circulation.\(^4\) In order to compensate, total blood volume is increased by expansion of blood volume in the venous circulation and systemic vascular resistance (afterload) increases.\(^5\) Increased afterload combined with impaired systolic performance also leads to an acute increase in left ventricular end-diastolic pressure. An acute increase in left ventricular end-diastolic and pulmonary venous pressures causes an increase of pressure in the alveoli. When the absorptive capabilities of the alveoli cells are overwhelmed, pulmonary congestion occurs.\(^9\)

Further, in acute decompensated heart failure, normal reflexes stimulated by increased atrial pressure are blunted by reflexes initiated in the high-pressure arterial circulation. For example, an increase in total blood volume associated with decompensated heart failure prompts activation of the renin-angiotensin-aldosterone system, leading to production of angiotensin II.\(^3\) Angiotensin II has many physiological effects, including peripheral and renal vasoconstriction (to restore arterial pressure and improve cardiac output), increased thirst, and stimulation of the sympathetic nervous system. Angiotensin II increases synthesis of aldosterone, leading to renal reabsorption of sodium and sodium retention.\(^10\) Activation of the sympathetic nervous system leads to elevated plasma levels of norepinephrine that stimulate \(\alpha\)-receptors in the nephron, enhancing reabsorption of sodium in the proximal tubules.\(^8,10\) In addition, \(\beta\)-receptors in the juxtaglomerular apparatus stimulate the renin-angiotensin-aldosterone system, further enhancing proximal tubular reabsorption of sodium.\(^4\) Normally,
atrial natriuretic peptide increases glomerular filtration rate and excretion of water and sodium; however, in advanced heart failure, these effects are attenuated by renal vasoconstriction and a reduction in sodium delivery to the distal nephron. Arginine vasopressin is released as a result of arterial underfilling. Arginine vasopressin increases plasma and urine osmolalities and leads to peripheral arterial vasoconstriction and water reabsorption in the cells of the distal tubule and collecting duct in the kidney, promoting hyponatremia. Figure 2 provides a global depiction of interacting events and responses that occur in patients with reduced cardiac output and fluid overload.

Thus, activation of neurohormonal systems leads to worsening retention of sodium and water that contributes to pulmonary congestion, hyponatremia, and edema. Ultimately, a vicious cycle occurs, with activation of neurohormonal systems leading to worsening cardiac function and further stimulation of neurohormonal systems. In addition to the pathophysiological processes of acute decompensated heart failure set in motion when
total blood volume increases because of arterial underfilling, increased left ventricular filling (diastolic) pressure and myocardial stretch (left ventricular dilatation) are also powerful mechanisms of neurohormonal activation and hypervolemia that can further impair cardiac function.2

Assessment of Hypervolemia

Accurate assessment of hypervolemia is important, because freedom from hypervolemia after hospitalization has been associated with improvement in long-term clinical outcomes. Lucas et al11 assessed patients 4 to 6 weeks after hospital discharge for 5 signs of hypervolemia: orthopnea, peripheral edema, weight gain, need to increase baseline diuretic dose, and jugular venous distension. Patients with any 3 of the 5 signs 6 weeks after discharge had a 3-fold increase in mortality at 2 years after the index hospitalization.

In another study,12 investigators defined clinical exacerbation of heart failure as the occurrence of at least 2 of the following: new or worsening edema, increased body weight, worsened dyspnea, worsened orthopnea, worsened paroxysmal nocturnal dyspnea, and increased jugular venous distension, all of which are indications of hypervolemia. In 189 outpatients with heart failure, episodes of clinical exacerbation were assessed over time. More episodes of clinical exacerbation were associated with an increased rate of hospitalization for heart failure, an increased risk of
mortality over a 2-year period, and a greater likelihood of lower quality of life, lower functional status, and poorer exercise tolerance.

In other studies, repeated hospitalizations for heart failure decompensation were associated with all-cause mortality, even after adjustments for patients’ characteristics. Moreover, hemodynamic congestion is not benign. In one study, increased blood volume was associated with increased pulmonary artery wedge pressure and increased risk for death or urgent heart transplantation at 1 year.

Although assessment of hypervolemia is important, some nuances are worth mentioning. First, although some signs and symptoms (weight gain, nocturia, elevated jugular venous pressure, lower extremity edema, positive hepatojugular reflux, paroxysmal nocturnal dyspnea, and crackles) were significant predictors of decompensated heart failure in patients treated in an emergency department, the overall sensitivity (the probability that signs or symptoms assessed were present in patients who actually had worsening heart failure) of each sign or symptom was low, even though specificity (the probability that signs or symptoms present were absent in patients who actually had worsening heart failure) was high. Thus, signs and symptoms commonly associated with decompensated heart failure were not helpful in diagnosing heart failure as the current problem. Invasive hemodynamic monitoring may be needed to assess intracardiac pressures.

Second, Mueller et al characterized daily dyspnea, edema, and body weight in patients with heart failure for 1 month, and Albert et al determined if signs and symptoms differed between ambulatory and hospitalized patients. Although dyspnea was positively and significantly associated with edema, changes in body weight were not routinely associated with dyspnea or edema. Hence, although changes in body weight might be associated with hospitalization for heart failure and repeat hospitalization for worsening heart failure, weight gain may not occur in patients with acute decompensated heart failure. Lack of association of body weight with dyspnea or edema could have many causes, including failure to monitor a patient’s weight, offset of weight gain from fluid by weight loss from cachexia, and minimal weight gain because of diminished appetite due to ascites.

Finally, hemodynamic congestion may not be associated with physical findings of hypervolemia. In a study of ambulatory nonedematous patients, physical findings of hypervolemia were infrequent and were not associated with increased blood volume. In patients with acutely decompensated heart failure, pulmonary artery wedge pressures can be elevated even though crackles and edema are absent or infrequent, and jugular venous pressures may not be elevated.

The biomarker B-type natriuretic peptide (BNP) may not be an ideal marker of volume status. In one study, levels of BNP increased with worsening heart failure and correlated with New York Heart Association functional class. However, in another study, after treatment, patients’ hemodynamic parameters correlated better with changes in blood volume than with changes in BNP values. Although the sample size was small, the researchers thought that BNP values changed more slowly than did blood volume and were better for showing long-term rather than instantaneous volume status.

Likewise, O’Neill et al found that BNP levels were not accurate predictors of serial hemodynamic changes in hospitalized patients with advanced heart failure. Even though an initial decrease in BNP levels was associated with early improvement in hemodynamic parameters, a change in BNP level was not associated with a change in pulmonary artery wedge pressure. Research results reinforce the need to use more than 1 method to assess initial volume status, to determine the effectiveness of therapies, and to inform clinical decisions.

Fluid Management Strategies

The guidelines of the American College of Cardiology and American Heart Association and the Heart Failure Society of America include recommendations for management of patients with chronic heart failure during acute episodes that require hospitalization. The recommendations should be followed to ensure optimal management with evidence-based therapies. During both hospitalization and outpatient care, the aims of fluid management strategies for left ventricular systolic dysfunction and left ventricular dysfunction with preserved ejection fraction are relief of signs and symptoms of hypervolemia, stabilization of hemodynamic status without further
damage of cardiac myocytes, and minimization of preventable recurrences of hypervolemia that require hospitalization for heart failure decompensation.

In patients with hypervolemia, signs and symptoms are the tip of the iceberg in regard to congestion. Pathophysiological changes in hypervolemia include low cardiac output, arterial underfilling, elevation in left ventricular diastolic pressures, and neuroendocrine activation. Thus, managing hemodynamic congestion manifested by increased left ventricular filling pressure but no constellation of signs and symptoms is just as important as managing clinical congestion. Core medications for heart failure and cardiac resynchronization therapies are first-line strategies for managing hypervolemia because the interventions attenuate neurohormonal activation and prevent progression, or promote reversal, of left ventricular remodeling that can worsen congestion. Unless contraindicated, all patients should take an angiotensin-converting enzyme inhibitor (eg, lisinopril, enalapril, or captopril) or angiotensin II receptor blocker (eg, valsartan or candesartan) and a β-blocker (eg, carvedilol, metoprolol succinate, or bisoprolol). Patients hospitalized with advanced systolic heart failure often meet indications for an aldosterone antagonist (eg, spironolactone or eplerenone) or hydralazine-and-nitrate combination therapy and cardiac resynchronization therapy.

Finally, when patients have hypoperfusion and diuretic-resistant elevations in cardiac filling pressures, intravenous inotropic or vasopressor therapies are indicated to maintain systemic perfusion and preserve or improve end-organ performance.

Loop diuretics are the hallmark pharmacological treatment for hypervolemia. Because oral agents, especially furosemide, have irregular intestinal absorption and can have altered pharmacokinetics and pharmacodynamics, intravenous administration is preferred during the early part of hospital therapy. When administered intravenously, loop diuretics rapidly relieve signs and symptoms of pulmonary congestion by lowering left ventricular filling pressures. Initially, loop diuretics should be administered at a dose that is higher than the total daily outpatient dosage. Urine output and signs and symptoms of hypervolemia must be serially assessed so that the dosage of a diuretic can be titrated to a patient’s needs. Adverse events associated with use of diuretics include electrolyte imbalances (hypokalemia and hypomagnesemia) leading to serious dysrhythmias, hypotension (especially when vasodilator therapy is used concomitantly), and worsening renal function. Electrolyte levels, hemodynamic parameters, and overall fluid volume status must be carefully monitored and managed. Diuretic resistance is common in patients with advanced heart failure because of hypertrophy of distal tubule epithelial cells, increased activation of the renin-angiotensin-aldosterone system, and decreased glomerular filtration rate. Strategies to overcome diuretic resistance are provided in Table 1.

Some therapies developed to directly or indirectly relieve hypervolemia were promising in early research but did not improve short- and long-term quality of life, morbidity, and mortality in large-scale randomized controlled trials. A1 adenosine receptor antagonists, vasopressin receptor antagonists, and levosimendan resulted in removal of excess fluid or improved cardiac output in heart failure in clinical trials but were not approved by the Food and Drug Administration because the medications did not decrease the number of hospitalizations for heart failure or mortality rates. Likewise, early clinical outcomes did not differ between treatment groups in acutely decompensated, hospitalized patients with heart failure and stable hemodynamic function.

<table>
<thead>
<tr>
<th>Table 1 Strategies to overcome diuretic resistance&lt;sup&gt;a&lt;/sup&gt;</th>
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<tbody>
<tr>
<td>1. Infuse the agent as a continuous intravenous infusion: for example, furosemide at 5-40 mg/h or bumetanide at 0.1-0.5 mg/h</td>
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<tr>
<td>2. Administer 2 diuretic agents at the same time: for example, a loop diuretic and an agent that blocks the distal tubule</td>
</tr>
<tr>
<td>* Intravenous chlorothiazide (500-1000 mg), given 30 minutes before administration of an intravenous loop diuretic</td>
</tr>
<tr>
<td>* Oral metolazone (2.5-10 mg) given with an oral loop agent</td>
</tr>
<tr>
<td>3. Rotating loop diuretic agents: for example, switching or alternating between oral furosemide and torsemide</td>
</tr>
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<sup>a</sup> No large, randomized trials that provide evidence of the effectiveness of these strategies have been done. Based on data from Jessup et al and the Heart Failure Society of America.
status who were randomized to continuous intravenous infusion of milrinone or placebo. More patients sustained hypotension requiring intervention and had new atrial arrhythmias in the milrinone group. Further, use of milrinone in patients who had worsening renal function (increasing serum levels of urea nitrogen) during hospitalization did not have improved outcomes despite minor improvements in renal function.

As a therapeutic procedure, ultrafiltration is the mechanical removal of fluid from the vasculature. Whole blood passes across a hemofilter (a semipermeable membrane) to yield plasma water in response to a pressure gradient created by the filtrate compartments and hydrostatic pressures in blood and also by oncotic pressure produced by plasma proteins. The ultrafiltration device extracts blood from and then returns it to the venous circulation via separate access points with large venous catheters (known as a venovenous technique) and an extracorporeal blood pump. Vascular catheters can be placed in the femoral, internal jugular, or subclavian veins and in large peripheral veins. The procedure can be performed only once, continuously or intermittently.

Unlike the situation in fluid removal with diuretics, which is primarily hypotonic, the sodium content in the ultrafiltrate is equal to the amount of sodium in the water component of plasma. In addition, diuretic therapy can cause hypovolemia, which enhances renal secretion of renin and activation of neurohormones. In ultrafiltration, fluid is removed from the blood at the same rate at which fluid is reabsorbed from the edematous interstitium; therefore, prolonged intravascular hypovolemia does not occur and neurohormonal activation is not stimulated.

Ultrafiltration became a more clinically relevant option after a portable, peripheral venovenous system became available and the results of a multicenter, randomized, controlled research trial corroborated the usefulness of the treatment. When peripheral ultrafiltration was compared with diuretic therapy in patients with acute decompensated heart failure, patients treated with ultrafiltration had greater weight loss, decreased need for vasoactive drugs, and reduced 90-day rate of rehospitalization. However, in a small single-center study in patients with very advanced heart failure and diuretic resistance, ultrafiltration was associated with variable fluid removal, and renal function worsened in 45% of patients during therapy. Overall, in 3 of 5 trials of peripheral ultrafiltration with a portable system, readmission was not improved in 3 studies, and signs and symptoms were significantly reduced in only 1 study.

Other disadvantages of ultrafiltration therapy are patients’ costs, need for training nurses in the procedure, nurse staffing, excessive volume removal (resulting in hypotension and worsening prerenal azotemia), and catheter-related or system complications, such as infection, thrombosis, air embolism, or hemorrhage due to disconnection of the venous return catheter. To date, peripheral ultrafiltration has not been better than aggressive intravenous diuretic therapy in improving signs and symptoms, causing weight loss, or preventing complications.

Nurses must understand medically appropriate care recommendations and advocate for patients during daily rounds with physicians and pharmacy care providers.
the depth or breadth of content delivered (program intensity), or assessment methods used to determine and enhance patients’ understanding. Additionally, not all programs were effective in preventing hospitalizations, even if patients had improvement in knowledge or self-care.48-51

Nursing Implications

Assessment

Because of the nuances of hypervolemia assessment in heart failure, nurses must not base decisions on volume status on a single method of assessment or on only a few variables. Physical signs and symptoms must be assessed along with patients’ subjective perceptions of clinical changes in status, such as worsening exercise intolerance or changes in New York Heart Association functional class (Table 2). A valuable assessment variable for hypervolemia may be history of recent hospitalization for heart failure. Nurses should ask patients about recent hospital events, especially if patients use more than a single health care center to meet health needs. Specific issues and tips for assessing hypervolemia are provided in Table 3.

In lieu of invasive hemodynamic monitoring to measure intracardiac pressures and definitively determine hemodynamic congestion, clinicians can be trained to use other technology. Portable, handheld, pocket-sized ultrasound machines can be used to determine left ventricular function, detect pericardial effusions, predict intravenous fluid responsiveness, and identify important valvular defects.64,65 For patients with implantable cardioverter defibrillators that also measure intrathoracic impedance, impedance data (on intrathoracic fluid) can be downloaded by using a wand system similar to that used to download pacemaker data. The impedance report provides data about the presence of thoracic congestion. In a study66 of 23 patients, impedance values measured by using an implantable cardioverter defibrillator were compared with pulmonary artery wedge pressures measured noninvasively by using echocardiography. The results indicated a strong correlation between high wedge pressure and low intrathoracic impedance.

Fluid Management

Currently, a gap exists between clinical expectations for use of evidence-based treatment recommendations and actual practice. Disparities are prevalent in the quality of care in heart failure at both the patient67-69 and hospital level.67,68,70 Nurses must understand medically appropriate care recommendations and advocate for patients during daily rounds with physicians and pharmacy care providers. Nurses should participate in quality improvement programs that focus on monitoring the adherence of health care providers’ use of heart failure medications chosen on the basis of research evidence and recommendations for use of cardiac devices. Nurses should also participate in quality improvement programs that focus

<table>
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<th>Table 2</th>
<th>New York Heart Association functional classificationa and examples</th>
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<tr>
<td>Functional status</td>
<td>Definition</td>
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<tr>
<td>I, Asymptomatic</td>
<td>Ordinary physical activity does not cause symptoms</td>
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<tr>
<td>II, Mild</td>
<td>Ordinary physical activity may be slightly limited by symptoms but no symptoms at rest</td>
</tr>
<tr>
<td>III, Moderate</td>
<td>Physical activity is markedly limited because of symptoms</td>
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<tr>
<td>IV, Severe</td>
<td>Physical activity cannot be carried out without symptoms; symptoms occur at rest</td>
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a Signs and symptoms: dyspnea, fatigue, chest pain, palpitations.
on the understanding of patients and patients’ families of education received, adherence to early (7-day) follow-up care, and adherence to the nonpharmacological plan of care. Nurses should participate on interdisciplinary collaborative teams to implement strategies to improve quality of care and conformity with recommended guidelines for management of heart failure. The need is great for nurses to develop and participate in programs that ease the transition of patients and informal caregivers from hospital to home and focus on disease management. Nurse-led initiatives can facilitate safe and effective care before patients are discharged, increase awareness of patients’ and informal caregivers’ barriers to optimal self-care, and prepare patients and informal caregivers to adhere to the plan of care.

<table>
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<tr>
<th>Table 3 Hypervolemia assessment issues and tips</th>
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<tr>
<td><strong>Issue 1: Insensitivity of common signs and symptoms of heart failure</strong>&lt;sup&gt;15-17&lt;/sup&gt;</td>
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<tr>
<td>• Ask multiple questions, in a variety of ways, to obtain a well-rounded picture of the incidence and level (mild, moderate, or severe) of signs and symptoms.</td>
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<tr>
<td>- For example, when assessing dyspnea, do not just ask if it had worsened before the patient came to the hospital or outpatient clinic. Patients may decrease activity level to prevent worsening of dyspnea.</td>
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<tr>
<td>□ Ask what activities the patient has given up or is doing more slowly or less frequently because of dyspnea.</td>
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<tr>
<td>□ Ask if caregivers have changed behaviors to minimize patient’s dyspnea.</td>
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<tr>
<td>- For example, if a patient complains of difficulty sleeping, you might learn that he or she has nocturia. Once the problem is identified, ask the patient about:</td>
</tr>
<tr>
<td>□ Changes in dietary behaviors (eating away from home or a change in purchasing practices)</td>
</tr>
<tr>
<td>□ Change in food preparation (new meal planner or cook)</td>
</tr>
<tr>
<td>□ Recent nonadherence to medications for heart failure</td>
</tr>
<tr>
<td>□ New or worsening thirst leading to increased fluid intake</td>
</tr>
<tr>
<td>□ Use of ibuprofen or other over-the-counter drugs that cause sodium and water retention</td>
</tr>
<tr>
<td>• Use noninvasive and internal monitoring features of cardiac devices to aid in assessment when elevations in pulmonary artery wedge pressure cannot be directly assessed.&lt;sup&gt;52&lt;/sup&gt;</td>
</tr>
<tr>
<td>- When measurement of thoracic impedance is available as a feature of an implantable cardioverter-defibrillator device, assess thoracic impedance levels.&lt;sup&gt;53&lt;/sup&gt;</td>
</tr>
<tr>
<td>- If respiratory distress or clinical evidence of impaired perfusion and intracardiac filling pressures cannot be assessed clinically, use invasive hemodynamic monitoring.&lt;sup&gt;1&lt;/sup&gt;</td>
</tr>
<tr>
<td><strong>Issue 2: Neuroendocrine activation leading to hypervolemia could be due to nonoptimal medication regimen or medication nonadherence</strong>&lt;sup&gt;1,28,54&lt;/sup&gt;</td>
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<tr>
<td>• Obtain medication reconciliation related to therapies used before hospitalization.</td>
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<tr>
<td>- Report findings and patient’s rationale for not taking medications as prescribed.</td>
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<tr>
<td>- Report core medications not prescribed for heart failure.</td>
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<tr>
<td>- Report identified contraindications to medications for heart failure.</td>
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<tr>
<td>- Report medications not for heart failure that could worsen heart failure.</td>
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<tr>
<td><strong>Issue 3: Nonadherence to self-care program for heart failure may have multiple, patient-specific causes that may be related to or beyond knowledge and skills deficits</strong>&lt;sup&gt;16,55-58&lt;/sup&gt;</td>
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<tr>
<td>• Carefully assess patient’s rationale for nonadherence. Consider economic issues (transportation or costs of care), social issues (caregiver support, loneliness),&lt;sup&gt;57&lt;/sup&gt; psychological issues (depression, anxiety),&lt;sup&gt;58&lt;/sup&gt; and cognition issues.&lt;sup&gt;59&lt;/sup&gt;</td>
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<tr>
<td>• Assess health literacy.&lt;sup&gt;60&lt;/sup&gt;</td>
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<tr>
<td>• Assess number of heart failure health care provider services as patients may be receiving mixed or confusing advice.&lt;sup&gt;61&lt;/sup&gt;</td>
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<td><strong>Issue 4: Be aware of laboratory and clinical parameters that may be helpful in determining hypervolemia, renal dysfunction that aggravates hypervolemia,</strong>&lt;sup&gt;4&lt;/sup&gt; or other medical conditions associated with decreased cardiac output and neuroendocrine activation leading to hypervolemia.&lt;sup&gt;1,28,62,63&lt;/sup&gt;</td>
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<tr>
<td>• Assess creatinine clearance (estimated glomerular filtration rate).</td>
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<tr>
<td>• Assess increases ion serum levels of urea nitrogen and creatinine during hospitalization.</td>
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<tr>
<td>• Assess hemoglobin level for presence of anemia that could be due to hemodilution.</td>
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<tr>
<td>• Assess serum sodium level for hyponatremia, which is a marker of an increase in plasma levels of arginine vasopressin (and water retention).</td>
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<tr>
<td>• Assess for elevated blood pressure that may be associated with stimulation of the sympathetic nervous system.</td>
</tr>
<tr>
<td>• Assess QRS duration ≥120 ms associated with cardiac dyssynchronization that can lead to mitral regurgitation and pulmonary congestion as well as hypervolemia from neuroendocrine activation associated with poor cardiac performance.</td>
</tr>
<tr>
<td>• Assess serum levels of troponins I and T, which are highly sensitive markers of myocardial injury. Elevated values (troponin I &gt;1 µg/L or troponin T &gt;0.1 µg/L) could indicate cardiac myocyte damage as the precipitant of hypervolemia.</td>
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<tr>
<td>• Assess worsening burden of comorbid conditions that could cause acute deterioration in renal function (eg, worsening diabetes or hypertension).</td>
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Patients’ nonadherence to methods for managing heart failure has been associated with acute decompensated heart failure leading to hospitalization.2,7 Nonadherence to diet, medication, or fluid restriction was cited as a reason for readmission by 25% of patients and 26% of informal caregivers.2 However, only 14% of cardiologists and 13% of heart failure nurses thought nonadherence was the primary reason for hospitalization.2 Cardiologists and heart failure nurses were more likely to cite other diseases, nonoptimal medical regimens, knowledge deficits, and delay in seeking help as reasons for hospitalization. Among participants in the study,7 33% of patients and 23% of heart failure nurses cited improving adherence to heart failure therapies as the primary intervention to prevent readmissions. Adequate professional help was identified by 35% of family caregivers as the most important intervention. Cardiologists identified 2 primary interventions as equally important: improving adherence and adequate professional help. Nurses have an opportunity and a responsibility to help patients improve adherence to regimens for managing heart failure.

Research results highlight the need for greater vigilance in optimal assessment of possible causes of patients’ nonadherence with the heart failure plan of care so that an individualized approach can be developed. Nurses must ensure consistency of hospital-based education and counseling related to fluid management (weight monitoring, fluid restriction when ordered, and low-sodium diet). Nurses must also ensure consistency in the delivery of interventions to manage heart failure (including those targeting clinicians and informal caregivers) and in assessment of the effectiveness of the interventions by studying clinical outcomes and cost of care so that deficiencies can be corrected. Attention to emerging knowledge and evidence-based practices is paramount to a successful program focused on patients, patients’ families and patients’ informal caregivers. Revised national guideline recommendations and new research that provides important and generalizable findings should be the basis for standards of clinical care.

Summary

Hyypervolemia (both hemodynamic and clinical congestion) is an import predictor of worsening heart failure, morbidity leading to hospitalization for heart failure, and mortality. Hyypervolemia can be difficult to recognize when common signs and symptoms of clinical congestion are not manifested during an acute congestive exacerbation. Clinical congestion often occurs later than elevated left ventricular filling pressure (hemodynamic congestion) does, necessitating use of multiple measures and methods of monitoring hyypervolemia.

Nurse-led or nurse-facilitated delivery of interventions to manage heart failure may decrease practice gaps associated with worsening heart failure due to hyypervolemia.


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49. Whellan DJ, Ousdiagn GT, Akitab SM, et al; PARTNERS study investigators. Combined heart failure device diagnostics identify patients at higher risk of subsequent heart failure hospitalizations: results from PARTNERS HF (Program to Access and
See page for full list of references.
Fluid Management Strategies in Heart Failure

Facts

In patients with chronic heart failure, fluid retention (or hypervolemia) is often the stimulus for acute decompensated heart failure that requires hospitalization. The pathophysiology of fluid retention is complex and involves both hemodynamic and clinical congestion. Signs and symptoms of both hemodynamic and clinical congestion should be assessed serially during hospitalization. Core heart failure drug and cardiac device therapies should be provided, and ultrafiltration may be warranted. Adherence to heart failure medications improves cardiac function, leading to improvement in volume status.

During both hospitalization and outpatient care, the aims of fluid management strategies for left ventricular systolic dysfunction and left ventricular dysfunction with preserved ejection fraction are relief of signs and symptoms of hypervolemia, stabilization of hemodynamic status without further damage of cardiac myocytes, and minimization of preventable recurrences of hypervolemia that require hospitalization for heart failure decompensation.1,2 Strategies to overcome diuretic resistance are provided in the Table.

Because of the nuances of hypervolemia assessment in heart failure, nurses must not base decisions on volume status on a single method of assessment or on only a few variables. Physical signs and symptoms must be assessed along with patients’ subjective perceptions of clinical changes in status, such as worsening exercise intolerance or changes in New York Heart Association functional class. A valuable assessment variable for hypervolemia may be history of recent hospitalization for heart failure. Nurses should ask patients about recent hospital events, especially if patients use more than a single health care center to meet health needs.

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Table

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<tr>
<td>2. Administer 2 diuretic agents at the same time: for example, a loop diuretic and an agent that blocks the distal tubule</td>
</tr>
<tr>
<td>‣ Intravenous chlorothiazide (500-1000 mg), given 30 minutes before administration of an intravenous loop diuretic</td>
</tr>
<tr>
<td>‣ Oral metolazone (2.5-10 mg) given with an oral loop agent</td>
</tr>
<tr>
<td>3. Rotating loop diuretic agents: for example, switching or alternating between oral furosemide and torsemide</td>
</tr>
</tbody>
</table>

a No large, randomized trials that provide evidence of the effectiveness of these strategies have been done. Based on data from Jessup et al1 and the Heart Failure Society of America.2

1. Which statement best defines features of heart failure due to structural heart disease?
   a. Orthopnea and sleep disordered breathing
   b. Decreased exercise tolerance and fluid retention
   c. Cough and orthopnea
   d. Hypovolemia and decreased exercise tolerance

2. What is the key factor to ensure euvolemia in heart failure patients?
   a. The integrity of the arterial circulation
   b. Normal kidney function
   c. Decreased levels of B-type natriuretic peptide
   d. Increased level of renin

3. Which of the following are physiological effects of angiotensin II?
   a. Acts as a neurotransmitter and sodium excretion
   b. Activates renal vasodilatation and sodium retention
   c. Inhibits the release of anti-diuretic hormone and B-type natriuretic peptide
   d. Activates renal vasoconstriction and stimulates the sympathetic nervous system

4. What is the mechanism for the stimulation of the renin-angiotensin-aldosterone system?
   a. Decrease in arterial volume
   b. Hyponatremia and antidiuretic hormone
   c. Production of cortisol by the adrenal gland
   d. Receptors in juxtaglomerular apparatus of the kidney

5. Which factor has been associated with long-term improvement in heart failure patients?
   a. Drinking fluids to prevent thirst
   b. Freedom from hypervolemia after hospitalization
   c. Weight loss when overweight, obese, or extremely obese
   d. Keeping serum sodium levels between 130-135 mmol/L

6. What did multiple researchers find to be true regarding weight gain in acute decompensated heart failure?
   a. Weight gain was commonly reported when even mild dyspnea was present
   b. Weight gain was associated with systolic dysfunction (ejection fraction of <40%)
   c. Weight gain may not occur in patients, even if dyspnea or edema are present
   d. Weight gain was commonly observed in patients with edema

7. After initial treatment (first 24 hours) of hypervolemia in patients with acute decompensated heart failure, what laboratory value is not an accurate predictor of heart failure status?
   a. Potassium
   b. Sodium
   c. B-type natriuretic peptide
   d. Glomerular filtration rate

8. What medications should all patients with heart failure take, unless otherwise contraindicated?
   a. Digoxin, β-blocker, and angiotensin-converting enzyme inhibitor
   b. Angiotensin-converting enzyme inhibitor or angiotensin II receptor blocker and β-blocker
   c. Aldosterone inhibitor, loop diuretic, and angiotensin-converting enzyme inhibitor
   d. β-Blocker, thiazide diuretic, and hydralazine/nitrates combination

9. What class of medication is considered the hallmark pharmacological treatment for hypervolemia in heart failure?
   a. Angiotensin-converting enzyme inhibitor
   b. β-Blocker
   c. Loop diuretic
   d. Aldosterone inhibitor

10. What adverse events are associated with the use of loop diuretics?
    a. Hypokalemia and hypomagnesemia
    b. Hypocalcemia and hyperkalemia
    c. Hyperphosphatemia and hyponatremia
    d. Hypermagnesemia and hypercalcemia

11. Which strategies were most effective for promoting adherence to prescribed therapies and preventing rehospitalization?
    a. Sending patients home with written self-care materials
    b. Ensuring patients know how to record daily weight and report changes
    c. Education before discharge and remote monitoring
    d. Diet class provided to family members after discharge

12. What percentage of patients and informal caregivers cited non-adherence to diet, medications, or fluid restriction as the reason for readmission?
    a. 46% and 38%, respectively
    b. 14% and 46%, respectively
    c. 12% and 15%, respectively
    d. 25% and 26%, respectively

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

1. 1. [ ] 2. [ ] 3. [ ] 4. [ ] 5. [ ] 6. [ ] 7. [ ] 8. [ ] 9. [ ] 10. [ ] 11. [ ] 12. [ ]
   a. [ ] b. [ ] c. [ ] d. [ ] e. [ ] f. [ ] g. [ ] h. [ ] i. [ ] j. [ ] k. [ ] l. [ ]

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Fluid Management Strategies in Heart Failure
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