Patients with heart failure and pulmonary edema are often admitted to the critical care unit. Many of these patients have severe peripheral edema, which may be associated with exudates and wounds of the lower extremities and which present a challenge to critical care nurses. Little information is available on treatment of peripheral edema in the intensive care unit or in patients with unstable hemodynamic status. Nursing care is based on available evidence, findings on chest radiographs, and hemodynamic status. Medications that contribute to peripheral edema should be evaluated and discontinued if possible. An appropriate mattress surface with an underpad that promotes wicking away of moisture should be selected. The patient’s lower extremities should be elevated according to his or her current pulmonary status, and skin-protective interventions should be instituted. Multilayer compression wraps should be avoided until the patient’s hemodynamic status is stable and the patient can get out of bed. (Critical Care Nurse. 2011;31[4]:21-29)

Heart failure is often associated with peripheral edema. Patients with decompensated heart failure may have an unstable hemodynamic status and have both pulmonary and peripheral edema. Medical treatment for patients with decompensated heart failure is usually focused on relieving pulmonary congestion and optimizing hemodynamic pressures. Little or no direction may be offered on the treatment of peripheral edema or on interventions to protect the skin integrity of the lower extremities. In addition, the traditional treatments used to reduce lower-extremity edema may add to pulmonary congestion. Thus critical care nurses are left in a quandary about the treatment of lower-extremity edema, containment of exudate, and prevention of skin breakdown. Attempts to contain exudate may be detrimental to skin integrity if the products used are inappropriate or are not applied properly. In this article, I discuss the causes of and contributing factors to peripheral edema in heart failure and offer advice on the care of critically ill patients with lower-extremity edema.

Causes of Peripheral Edema in Patients With Heart Failure

Causes of peripheral edema in patients with heart failure (Figure 1 and Table 1) are related to both left- and right-sided heart failure, medications, and compensatory changes that influence hydrostatic pressure, fluid retention, and autoregulation in the...
vascular system. In addition, patients chronically ill with heart failure may have nutritional deficiencies that cause decreases in the serum level of albumin and result in decreased oncotic pressure.

Albumin accounts for about 55% of total serum protein. Serum protein contributes to colloid osmotic-oncotic pressure associated with retention of sodium and water in the vascular bed. When the serum level of albumin decreases (normal range, 3.4-5.4 g/dL), colloid osmotic-oncotic pressure decreases. When colloid pressure decreases to less than hydrostatic pressure, fluid leaks from the vascular tree into the interstitial space. Patients with decompensated heart failure are often short of breath. Shortness of breath contributes to nutritional and albumin deficiency because patients have difficulties preparing and consuming meals that provide adequate nutrition. Protein-rich foods such as meats require mastication, a requirement that may limit consumption in patients with low energy levels and shortness of breath.

In right-sided heart failure, the right ventricle is unable to pump blood into the pulmonary circulation, and blood backs up into the right atrium, superior vena cava, and peripheral tissues. This backup results in edema of distal peripheral tissues below the level of the heart, including the liver and the lower extremities.

In left-sided heart failure, cardiac output decreases, causing activation of the sympathetic nervous system and neurohormonal compensatory mechanisms that ultimately result in peripheral edema. Activation of

**Figure 1** Causes of peripheral edema in patients with heart failure.

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the renin-angiotensin system causes release of aldosterone, vasoconstriction of peripheral vessels, and retention of sodium and water. Antidiuretic hormone is also released, resulting in increased water reabsorption in the renal collecting ducts, which contributes to intravascular and ultimately interstitial fluid. Decreased cardiac output may reduce the ability of cells to pump out excess sodium and thus further contribute to edema.1(pp910,911)

Levels of brain natriuretic peptide are elevated in both right- and left-sided heart failure and may contribute to the development of peripheral edema in patients with heart failure. This peptide is released when the ventricle becomes stretched from increased volume. Brain natriuretic peptide acts on the renal tubules to retain sodium and water. It also causes vasodilatation and aids diuresis by increasing glomerular filtration pressure.2 Endothelin levels are also increased in heart failure. The actions of endothelin include renal vasoconstriction and sodium and water retention.3(p238) Sodium and water retention in the vascular tree contribute to formation of edema.3(p238)

Medications associated with the development of peripheral edema include calcium channel blockers, β-blockers, insulin, glyburide, and metformin. Although the exact mechanism by which these medications cause edema is not specified, many of them cause vasodilatation or vasoconstriction. The occurrence of peripheral edema is considered an adverse drug reaction. The weight gain anticipated from hypoglycemic agents is less than 2 kg, but when hypoglycemic agents are combined with other medications, weight gain may be twice the anticipated weight gain from hypoglycemic agents alone.2 When peripheral edema occurs in a patient taking a calcium channel blocker, the dosage of the blocker should be decreased or the medication discontinued.3(p238)

Last, the calf muscle pump (action of the calf muscles during activity to pump venous blood upward) in patients with decompensated heart failure is often ineffective because of a decrease in the patients’ physical activity. Patients with decompensated heart failure often have decreased activity tolerance, and the additional effort to mobilize edematous extremities, which are heavy and swollen, further contributes to immobility. In the hospital, patients are often in bed and immobile.

A weight gain of 2.25 kg indicates retention of approximately 2 L of fluid. Pitting edema may indicate that approximately 4 L of fluid are sequestered in the interstitial space.

### Table 1: Contributing factors in edema

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Pathophysiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydrostatic</td>
<td>Increased systolic pressure causes movement of fluid from areas of high pressure (the vascular bed) to areas of lower pressure (the interstitial spaces). Endothelin is a protein released in heart failure that causes arterial vasoconstriction, which causes higher pressure in the arterial capillary system than in the venous system.</td>
</tr>
<tr>
<td>Decreased oncotic/osmotic pressure</td>
<td>Albumin (a protein) aids in retaining fluid in the vascular bed. When levels are low, fluid is not retained in the vascular system and leaks into the interstitial spaces, causing edema.</td>
</tr>
<tr>
<td>Increased capillary permeability</td>
<td>Increased capillary permeability is influenced by the release of endotoxins and cellular mediators. Brain natriuretic protein is one of the cellular mediators released in congestive heart failure that influence capillary permeability.</td>
</tr>
<tr>
<td>Renin-angiotensin system</td>
<td>Renin and angiotensin affect the kidneys’ retention of sodium and water. When the renin-angiotensin system is stimulated to retain sodium and water, water and pressure in the vascular bed increase. Higher pressures due to increased water and sodium increase the movement of water and sodium into the interstitial space.</td>
</tr>
<tr>
<td>Medications</td>
<td>Medications that cause vasodilatation or vasoconstriction Medications with adverse drug reactions that include edema</td>
</tr>
<tr>
<td>Functional ability</td>
<td>Decreased activity causes decreased ability of calf muscles to pump, decreasing venous return and increasing venous stasis. Impaired nutritional intake decreases protein intake; decreased serum level of protein decreases colloid osmotic-oncotic pressure and encourages leakage of fluid into the interstitial space.</td>
</tr>
</tbody>
</table>
Mrs F, a 66-year-old, came to the emergency department because she had chest pain and shortness of breath. She had a history of hypertension and type 2 diabetes. Although findings on an electrocardiogram were nondiagnostic, laboratory values were consistent with non–ST-segment elevation myocardial infarction. The serum troponin level was 9 ng/mL (normal, 0.00-0.09 ng/mL), and the serum level of creatine kinase was 265 U/L (normal, <135 U/L; to convert units per liter to microkatalis per liter, multiply by 0.0167) with an MB fraction of 16.8 ng/mL (normal, <3.0 ng/mL). The level of brain natriuretic peptide was 800 pg/mL (normal, 0-100 pg/mL).

Upon admission to the emergency department, Mrs F had an arterial oxygen saturation of 90% when breathing 4 L of oxygen by nasal cannula. However, she rapidly became increasingly short of breath and was switched to 10 L of oxygen by face mask and then 15 L by nonrebreather mask. Improvement in oxygen saturation and shortness of breath was minimal. Findings on chest radiographs were consistent with pulmonary edema (Figure 2). Mrs F was tachycardic with a heart rate of 103/min and tachypneic with respirations of 39/min. Her blood pressure was 207/77 mm Hg. She was given 160 mg of furosemide intravenously, 2 doses of 0.4 mg nitroglycerin sublingually, and then a continuous intravenous infusion of nitroglycerin at 90 μg/min. She was intubated and transferred to the intensive care unit.

Before admission, Mrs F was taking the following medications: aspirin 81 mg/d orally, furosemide 40 mg twice daily orally, glargine (Lantus) insulin 100 units each night subcutaneously, lisinopril 20 mg/d orally, lovastatin 40 mg/d orally, allopurinol 100 mg/d.

**Treatment Strategies for Patients With Peripheral Edema and Decompensated Heart Failure**

Unfortunately, evidence-based treatment of peripheral edema in patients with decompensated heart failure is limited to the use of diuretics. The contributing causes of peripheral edema should be investigated for their potential role in the edema. Because pulmonary edema and heart failure are relieved with use of diuretics, the serum level of brain natriuretic peptide will decrease, and the peptide will no longer contribute to edema. Medications that contribute to the formation of edema should be assessed and should be discontinued or changed to evaluate their effect on continued peripheral edema. Bilateral peripheral edema is generally related to venous insufficiency and heart failure. If lymphedema is the cause of either unilateral or bilateral peripheral edema, treatment may be similar, but diuretic therapy will not be helpful. Unilateral peripheral edema suggests the possibility of deep vein thrombosis, and ultrasound should be performed to determine if deep vein thrombosis is present.
orally, and metoprolol tartrate 100 mg/d orally. In the intensive care unit, she was given ondansetron 40 mg/d intravenously, intravenous heparin infusion titrated according to activated partial thromboplastin time, and epifibatide (Integrilin) 180-μg bolus dose and continuous infusion at 2 μg/kg per minute. Furosemide 40 mg twice daily also was ordered.

Transesophageal echocardiography showed reduced left ventricular systolic function and akinesis of the inferior apical wall. The estimated left ventricular ejection fraction was 45% with moderate elevation of pulmonary artery pressure.

Mrs F was treated with mechanical ventilation with a fraction of inspired oxygen of 40% and pulmonary end expiratory pressure 5 cm H₂O and was able to maintain a PaO₂ of 80 mm Hg with aggressive administration of a diuretic. The edema in the lower extremity was rated as 4+, pitting, and weeping. In an effort to absorb the excessive exudate, nursing staff applied circumferential gauze dressings from ankle to knee under the sequential compression device and placed plastic-backed pads under the lower extremities. Upon removal of the gauze dressings 24 hours after the application, the left lower extremity had a skin tear and evidence of cellulitis (Figure 3). A deep indentation was present at the ankle. A wound care consultation was obtained. The wound care nurse ordered a low air loss mattress overlay, cleansing of the lower extremities twice daily, and application of a skin-barrier cream after cleansing. Further orders included leaving the lower extremities open to air and application of petrolatum gauze to the skin tear that would be changed every 12 hours. Antibiotics were ordered by the physician to treat the cellulitis. The foot of the bed was elevated, and an absorbent pad recommended by the manufacturer of the mattress overlay was placed under the lower extremities to absorb exudate.

When her hemodynamic status was stable, Mrs F was placed on a progressive ambulation protocol and was transferred to the medical surgical unit. Compression wraps were applied to her lower extremities by the wound care nurse. At the time of discharge, Mrs F was referred to the wound care nurse for follow-up for wound care, and compression hose specific to the patient’s leg measurements were ordered by the nurse at the time of discharge.

The American Heart Association and the American College of Cardiology have published guidelines on the treatment of patients with decompensated heart failure. These guidelines address initial treatment and treatment of pulmonary edema but do not address the treatment of peripheral edema. Initial treatment strategies for patients with decompensated heart failure include use of loop diuretics to reduce volume overload and pulmonary congestion. In patients with severe heart failure with pulmonary edema, the addition of a vasodilator such as nitroglycerin, sodium nitroprusside, or nesiritide is recommended if blood pressure permits.⁶

Severe edema predisposes patients to skin breakdown. Skin tears over bony prominences and pressure ulcers, especially on the heels, are common in patients with edema of a lower extremity. Venous insufficiency is a common finding in patients with chronic heart failure and may be manifested by the presence of hemosiderin staining of the lower extremities. Hemosiderin staining is due to deposits of the breakdown products of red blood cells and usually occurs in...
the area above the ankle and below the knee. The medial and lateral malleoli are particularly predisposed to skin tearing due to edema. The skin may also appear tense and scaly.

Traditional treatments for patients with heart failure with peripheral edema include elevation of the affected extremity above the heart level, exercises (eg, pedal-push exercises) that activate the calf muscle pump, use of sequential compression devices, and use of elastic compression stockings or layered compression wraps. Scheduled range-of-motion exercises facilitated by nurses or referrals to physical therapy for assistance in providing appropriate therapy to increase venous return in patients on bed rest may help decrease edema.

Elevation of the legs decreases edema only slightly; addition of elastic wraps may result in a further decrease. If a patient has weeping lower-extremity edema, elastic wraps must be changed frequently. Care must be taken to ensure that the wrap does not shift or crease, causing constriction, particularly around the heel and ankle (Figure 3). A pad (made of components similar to those used in diapers) that absorbs and wicks moisture should be placed under exuding extremities. Procedure pads or simple plastic-backed pads should not be used. They do not wick away moisture, and moisture close to the skin contributes to skin maceration and potential skin breakdown. In addition, the patient’s heels should be elevated off the bed to prevent the development of pressure ulcers on the heels.

Sequential compression devices may be helpful in reducing edema, but the sleeves do not absorb exudate and the addition of gauze or other padding to absorb exudates may interfere with the pressure redistribution or increase pressure over fragile skin areas and contribute to the development of skin tears or pressure ulcers. If removed improperly, gauze dressings may stick to the skin and tear it when removed. Sequential compression devices may be set to variable levels of pressure and offer compression at alternating areas from ankle to knee or from ankle to thigh. Orders for sequential compression devices may include the addition of compression stockings. The additive effect of sequential compression devices and compression stockings in the prevention of deep vein thrombosis is controversial, but the use of elastic stockings or compression stockings in addition to the device may be helpful in reducing edema and will not decrease the effectiveness of the devices in enhancing venous return. The tubing of sequential compression devices may promote the development of pressure ulcers, especially in patients with severe edema, so care must be taken to keep the tubing away from direct contact with the skin or under the extremities. Use of intravenous unfractionated heparin or subcutaneous low-molecular-weight heparin should be considered because use of sequential compression devices in a patient with severely swollen extremities may not be effective in preventing deep vein thrombosis.

Compression wraps are a well-documented intervention for the treatment of lower-extremity edema in outpatients, but no studies have been published that address use of the wraps in inpatients. If a patient with pulmonary edema admitted to the critical care unit has compression wraps, the wraps should be removed to determine if infection or cellulitis is present and to prevent the potential complication of compartment syndrome. If a compression wrap contains a zinc-based primary layer, the zinc cream must be removed to allow skin assessment. Mineral oil removes the zinc-based cream easily. Compression wraps are minimally elastic, and as heart failure progresses, the compression wraps may act like a cast, causing compression of nerves and blood vessels.

Application of compression wraps in patients with severely decompensated heart failure may be inappropriate. These patients need diuretic therapy, and the wraps will decrease the vascular volume in the periphery but may increase the volume in the pulmonary tree, increasing pulmonary edema. The size of the lower extremity will change, and the size of the compression wrap will be inappropriate when the size of the extremity decreases. Application of compression wraps before diuresis is completed may result in more fluid in the lungs and a worsening of pulmonary congestion.

Compression wraps include several layers of dressing. The first layer is an impregnated gauze bandage that includes a zinc or calamine additive. The next layer is a padding bandage. The following 2 layers are compressive elastic and adherent products (Figures 4 and 5). Compression wraps are usually changed weekly. Acutely ill patients with decompensated heart failure should have a complete skin assessment performed daily, at a minimum, to
determine if cellulitis is present or if pressure-associated ulceration has developed. In addition, compression wraps should be applied by a nurse who is competent in applying them.1,pp910,911 Wound care nurses are usually the only nurses competent in the application of compression wraps.

Regardless of the type of dressing or skin covering, the skin surface should be protected and cleansed. Physiological saline, mild soap, or rinseless cleansers should be used to cleanse the skin surfaces. Application of skin-barrier cream or ointment to skin surfaces may be helpful to protect skin integrity and prevent epidermal degradation due to maceration. Possible creams and ointments include commercial products with zinc, petrolatum, or other moisturizing and protective agents. Zinc-based skin barriers may leave a residue on the skin even after cleansing that makes assessment of skin color difficult.

Low air loss therapy with mattress overlays or mattress replacement products may help reduce skin moisture by constant motion of air across the mattress surface. The underpad suggested by the manufacturer should be used; otherwise the therapeutic drying action may be lost. In the intensive care unit, the key components in choices of care include the ability to assess skin surfaces easily and as often as a patient’s condition requires.

Critical care nurses should consider all the variables that contribute to leg edema in formulating the plan of care for a patient (Table 2). Adjunctive therapies may be beneficial in preventing skin breakdown and reducing edema.

When to Begin Treatment for Peripheral Edema

Treatment for peripheral edema includes elevation of the affected extremities, administration of diuretic agents, use of compression, and active and passive range-of-motion exercises that activate the calf muscle pump. In a patient with decompensated heart failure and pulmonary edema, elevation of the lower extremities may make the patient’s condition more unstable and contribute to pulmonary congestion. After diuretic therapy is started, the nurse must determine when to safely elevate the lower extremities to decrease peripheral edema. If the patient is in respiratory distress, elevation of the legs should be delayed until oxygen saturation measured by pulse oximetry is acceptable, respiratory rate is normal, and signs of respiratory distress are absent. If a patient can only tolerate having the head of the bed elevated more than 30°, elevation of the legs may place the patient at greater risk for sacral pressure ulcers. Range-of-motion and plantar flexion exercises increase activation of the calf muscle pump whether or not a patient has sequential compression devices on the lower extremities.

Madias7 found that the amplitude of the electrocardiogram

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**Figure 4** Patient’s left lower extremity wrapped in compressive elastic products.

**Figure 5** Patient’s left lower extremity wrapped in adherent products.
Table 2 Strategies in the care of patients with edema of the lower extremities

<table>
<thead>
<tr>
<th>Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevate the edematous extremities</td>
</tr>
<tr>
<td>Evaluate medications that may contribute to the development of edema</td>
</tr>
<tr>
<td>Use underpads that are designed to wick away moisture</td>
</tr>
<tr>
<td>Consider treatments that are additive:</td>
</tr>
<tr>
<td>1. Administer diuretics</td>
</tr>
<tr>
<td>2. Raise the extremity above the level of the heart</td>
</tr>
<tr>
<td>3. Protect the skin with a skin barrier</td>
</tr>
<tr>
<td>4. Use an absorbent underpad</td>
</tr>
<tr>
<td>5. Consider application of elastic bandages that will be changed when soiled</td>
</tr>
<tr>
<td>6. Perform range-of-motion exercises</td>
</tr>
<tr>
<td>7. Use sequential compression devices</td>
</tr>
<tr>
<td>8. Consider a low air loss mattress or overlay to aid in drying exudate</td>
</tr>
</tbody>
</table>

Tracing may be influenced by thoracic fluid volume. For example, the amplitude of QRS complexes fluctuates with fluid volume in pulmonary edema. In addition, T-wave amplitude may be influenced by the degree of peripheral edema in patients with heart failure. The increase in amplitude of the QRS complex and the T wave may indicate safe timing to elevate the lower extremities, but more research is needed.

Findings on chest radiographs can also be used to determine when diuretic therapy has been sufficient to allow treatment of peripheral edema. However, no information in the literature supports the timing of interventions to safely decrease peripheral edema in patients with decompensated heart failure. In fact, on a chest radiograph, evidence of fluid may lag behind clinical indications of shortness of breath. If a patient is intubated and receiving mechanical ventilation, elevating the lower extremities before extubation may be prudent to determine the effect of venous return on pulmonary status. Again, no evidence-based studies support this practice.

**Conclusion**

Patients with acute decompensated heart failure and peripheral edema are a challenge for critical care nurses. Physicians may not be knowledgeable about the appropriate nursing care to protect the lower extremities from injury. In the intensive care unit, nurses who care for patients with decompensated heart failure need a basic understanding of evidence-based practice for the treatment of patients with peripheral edema. If an advanced practice nurse is not available to offer guidance, critical care nurses must be knowledgeable about appropriate treatment of exudative peripheral edema to prevent adverse outcomes.

Key factors in the care of patients with pulmonary congestion and peripheral edema include when to initiate treatment such as compression wraps or elastic wraps plus sequential compression devices to decrease peripheral edema and how to prevent problems in skin integrity by using skin-barrier products and protecting bony prominences and heel pressure.

Although further research is necessary to determine the appropriate timing for various therapies, critical care nurses must be cognizant of therapies to avoid when a patient’s pulmonary status is unstable and of therapies that may ultimately cause a patient harm.

**References**

CE Test  Test ID C114: Care of the Lower Extremities in Patients With Acute Decompensated Heart Failure

Learning objectives: 1. Describe the pathophysiology of peripheral edema associated with acute decompensated heart failure. 2. Identify the limitations of current evidence-based treatment for critically ill patients with peripheral edema and acute decompensated heart failure. 3. Discuss appropriate treatment strategies and their associated nursing considerations and implications related to care of patients with peripheral edema and acute decompensated heart failure.

1. Fluid leaks from the vascular tree into the interstitial space when colloid pressure decreases to less than which of the following pressures?
   a. Osmotic-oncotic pressure
   b. Hydrostatic pressure
   c. Mean arterial pressure
   d. Central venous pressure

2. Current evidence-based treatment of peripheral edema in patients with decompensated heart failure is limited to which of the following therapies?
   a. Administration of diuretics
   b. Elevation of lower extremities
   c. Frequent range-of-motion exercises
   d. Application of elastic or compression stockings

3. Which of the following medications may contribute to peripheral edema?
   a. Insulin, β-blockers, nonsteroidal anti-inflammatory drugs
   b. Calcium channel blockers, metformin, and glyburide
   c. Nitroglycerin, insulin, and β-blockers
   d. Albumin, insulin, metformin, and glyburide

4. Decreased cardiac output is thought to reduce the ability of cells to do which of the following?
   a. Use oxygen
   b. Pump out excess sodium
   c. Maintain membrane integrity
   d. Utilize glucose

5. Pitting edema indicates what amount of fluid is sequestered in the interstitial space?
   a. 2.5 L
   b. 3 L
   c. 4 L
   d. 4.5 L

6. Diuretic therapy would most likely be effective for which of the following?
   a. Unilateral peripheral edema
   b. Bilateral peripheral edema associated with pulmonary edema
   c. Unilateral peripheral edema associated with lymphedema
   d. Bilateral peripheral edema associated with lymphedema

7. Which of the following measurements is most valuable in determining whether sodium nitroprusside can be used as treatment for a patient with severe heart failure and pulmonary edema?
   a. Left ventricular ejection fraction
   b. Pulmonary artery pressure
   c. Heart rate
   d. Blood pressure

8. Which of the following therapies are suggested to decrease moisture in patients with peripheral edema associated with heart failure?
   a. Circumferential gauze dressing to contain exudate
   b. Sequential compression devices
   c. Compression wraps
   d. Low air-loss mattress therapy

9. Which of the following interventions should be avoided in a patient with acute decompensated heart failure and pulmonary edema?
   a. Removal of compression wraps before diuretic therapy
   b. Application of compression wraps before diuretic therapy
   c. Performance of range-of-motion exercises before the patient can tolerate having the head of the bed elevated less than 30°
   d. Elevation of the patient’s lower extremities while intubated and receiving mechanical ventilation

10. Which of the following indicates that it is safe to elevate the legs of a patient with acute decompensated heart failure?
    a. Patient’s BNP level has increased; chest radiograph shows significant improvement of pulmonary edema
    b. Patient’s supplemental oxygen requirements have decreased; chest radiograph shows worsening of pulmonary edema
    c. Patient’s BNP level has decreased; amplitude of QRS complexes has decreased
    d. Patient’s supplemental oxygen requirements have decreased; amplitude of QRS complexes has increased

11. An underpad should be used in conjunction with low air loss therapy with mattress overlays and/or mattress replacement products to ensure which of the following?
    a. Reduced compression of nerves and blood vessels
    b. Reduced skin moisture
    c. Reduced deep vein thrombosis
    d. Reduced breakdown of red blood cells

12. Edema of the lower extremities particularly predisposes the lateral and medial malleoli to which of the following?
    a. Hemosiderin staining
    b. Pressure ulcers
    c. Skin tears
    d. Development of cellulitis

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

1. □ a □ b □ c □ d  2. □ a □ b □ c □ d  3. □ a □ b □ c □ d  4. □ a □ b □ c □ d  5. □ a □ b □ c □ d  6. □ a □ b □ c □ d  7. □ a □ b □ c □ d  8. □ a □ b □ c □ d  9. □ a □ b □ c □ d  10. □ a □ b □ c □ d  11. □ a □ b □ c □ d  12. □ a □ b □ c □ d

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Care of the Lower Extremities in Patients With Acute Decompensated Heart Failure
Karen L. Cooper

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