

Cardiovascular Emergencies: Pericardial Effusion and Cardiac Tamponade

Jo Ann Flounders, MSN, CRNP, OCN®, CHPN

Cardiac tamponade is a life-threatening emergency that occurs when excessive fluid in the pericardial space, called a pericardial effusion, creates increased pressure in the pericardial sac that compromises the heart's ability to fill and pump. Consequently, cardiac output decreases and systemic perfusion is impaired (Dietz & Flaherty, 1993; Hunter, 1998). The distinction between pericardial effusion and cardiac tamponade is important. Pericardial effusion is an anatomic diagnosis of abnormal pericardial fluid accumulation that has no hemodynamic consequences, whereas cardiac tamponade is a physiologic diagnosis of varying amounts of pericardial fluid that causes increased pressure and resultant hemodynamic consequences (Harken, Hammond, & Edmunds, 1997).

Etiology

Although cardiac tamponade can be caused by malignant or nonmalignant conditions, malignant disease resulting in pericardial effusion is a major cause of cardiac tamponade in many patients (Keefe, 2000; Knoop & Willenberg, 1999; Markiewicz, Borovik, & Ecker, 1986; Pass, 1997). Malignant invasion of the pericardium or myocardium may be the result of metastatic tumors or direct extension of a primary tumor (DeMichele & Glick, 2001). Whereas primary tumors of the heart, such as mesotheliomas and sarcomas, occur rarely, malignant invasion of the heart by metastases or direct extension occurs more frequently (Knoop & Willenberg; McAllister, Hall, & Cooley, 1999). Malignancies that cause secondary tumor involvement, either by metastasis or local extension, include lung, breast, and esophageal cancers. Hodgkin's and non-Hodgkin's lymphomas frequently involve the mediastinum and pericardium. Leukemias can infiltrate the myocardium and cause small effusions (Keefe; Knoop & Willenberg). Sarcomas, melanomas, and liver, gastric, and pancreatic malignancies also can metastasize to the heart.

Cancer treatment also may cause pericardial effusion with resultant cardiac tamponade (Knoop & Willenberg, 1999). Radiation therapy of more than 4000 cGy to the mediastinal area can cause pericarditis, leading to the development of car-

diac tamponade (Harken et al., 1997; Knoop & Willenberg; Smeltzer & Bare, 1996). Anthracyclines, such as doxorubicin or daunomycin, can be responsible for the development of pericardial effusions (Chabner & Myers, 1993).

People with cancer may experience pericardial effusions and cardiac tamponade caused by nonmalignant conditions, although the incidence is less compared to malignant causes. Diseases such as rheumatoid arthritis, systemic lupus erythematosus, hypoalbuminemia, renal failure, or hypothyroidism may cause increased serous fluid in the pericardial sac or serous effusion (Bullock, 2000; Lawler, 1999). Infectious pericarditis caused by bacteria, fungus, virus, or tuberculosis can cause cardiac tamponade as a result of accumulation of large volumes of infected fluid. Other cardiovascular conditions, such as chest trauma, aneurysm, improper insertion of a cen-

Goal for CE Enrollees:

To further enhance nurses' knowledge of cardiovascular emergencies of patients with cancer.

Objectives for CE Enrollees:

- On completion of this CE, the participant will be able to
1. Describe two cardiovascular emergencies for patients with cancer.
 2. Describe medical management of the cardiovascular emergencies.
 3. Discuss nurses' role in the care of patients with cardiovascular emergencies.

Jo Ann Flounders, MSN, CRNP, OCN®, CHPN, is a nurse practitioner at Consultants in Medical Oncology and Hematology in Drexel Hill, PA.

Digital Object Identifier: 10.1188/03.ONF.E48-E55

tral venous catheter, or complications secondary to angiography, can cause serosanguinous pericardial effusion and subsequent tamponade (Bullock; Schafer, 1997).

Physiology

The heart and segments of the great vessels are surrounded by the pericardium, a thin, tough fibrous sac with two layers (Knoop & Willenberg, 1999; Maxwell, 1997; Schafer, 1997). The inner layer, called the visceral pericardium, is a serous membrane connected to the surface of the heart. The outer fibrous membrane of the pericardium is the parietal layer, which provides strength and protection and is in direct contact with the chest wall at the left sternal position (Schafer). Between the visceral membrane and the parietal membrane is the pericardial cavity that cushions the myocardium.

The mesothelial cells of the visceral pericardium produce 10–50 ml of clear serous lubricating fluid that prevents friction between the membranes during contraction and relaxation of the heart (Mangan, 1992). This pericardial fluid arises from the lymphatic channels surrounding the heart (Schafer, 1997) and normally is reabsorbed and drained by the lymphatic system into the mediastinum and right heart cavities (Uaje, Kahsen, & Parish, 1996).

Pathophysiology

Excessive fluid can accumulate in the pericardial sac for a variety of reasons (Beauchamp, 1998). As malignant invasion of the pericardium occurs, the invasive malignancy or the pericardial tissue itself may produce excess fluid in response to the malignant process (Smeltzer & Bare, 1996). Pericardial fluid can collect when lymphatic and venous flow is obstructed by tumor, therefore preventing reabsorption of fluid (Mangan, 1992). Invasive tumors also can bleed, and because blood accumulates more rapidly than transudates or exudates, these pericardial effusions tend to progress to cardiac tamponade more rapidly (Keefe, 2000).

Cardiac tamponade results when cardiac function is impaired by pressure exerted by pericardial effusion. As pericardial fluid increases, the increased pressure in the pericardium compresses all four chambers of the heart (Beauchamp, 1998; Bullock, 2000; Knoop & Willenberg, 1999; Mangan, 1992). The right atrium and right ventricle initially are compressed, leading to decreased right atrial filling during diastole. As less venous blood returns to the right atrium, increased venous pressure results, which is evidenced by jugular vein distention, edema, hepatomegaly, and increased diastolic pressure. Continued compression of the heart leads to decreased diastolic filling of the ventricles as well, causing decreased stroke volumes, decreased cardiac output, and poor tissue perfusion. The body attempts to compensate with cardiac stimulation by the adrenergic nervous system, resulting in tachycardia and peripheral vasoconstriction. Decreased tissue perfusion causes activation of the renin-angiotensin-aldosterone system as the body attempts to increase blood volume and improve stroke volume (Bullock). As a result, workload on the failing heart is greatly increased. As these compensatory mechanisms are exhausted, a cycle of increased fluid, with decreased cardiac output and decreased venous return, will cause circulatory collapse and lead to shock, cardiac arrest, and death if not corrected (Schafer, 1997).

The severity of cardiac tamponade depends on the amount of pericardial effusion, the rate of accumulation, and the degree of pericardial compromise (Schafer, 1997). Onset may be gradual or rapid. If fluid accumulates slowly, the parietal pericardium is able to stretch to compensate for the increased pressure and large amounts of fluid may be tolerated without symptoms. However, when fluid accumulates rapidly, the pericardial pressure rises quickly, compensatory mechanisms cannot adapt, and severe cardiac tamponade results (Bullock, 2000; Knoop & Willenberg, 1999; Schafer; Smeltzer & Bare, 1996).

The risk of cardiac tamponade for patients with cancer corresponds to the etiologic factors that cause cardiac tamponade. Figure 1 presents a summary of patients with cancer who are at risk for cardiac tamponade.

Assessment

Signs and symptoms of cardiac tamponade are related to the amount and rapidity of onset of pericardial fluid accumulation and require a careful and thorough history and review of symptoms. Clinical manifestations increase in severity if pericardial fluid rapidly accumulates or if a small pericardial effusion progresses into a large effusion (Knoop & Willenberg, 1999; Mangan, 1992; Schafer, 1997). Therefore, patients may be initially asymptomatic when pericardial effusions are small or develop slowly.

Early symptoms of pericardial effusion with tamponade include

1. Dyspnea, which is usually the most common presenting symptom (Shepherd, 1997)
2. Retrosternal chest pain that increases when patients are supine and decreases when leaning forward because of compression of the heart
3. Cough, dysphagia, hoarseness, or hiccups caused by mechanical compression of nerves of the esophagus, bronchi, and trachea (Uaje et al., 1996)
4. Dizziness, lightheadedness, or agitation caused by hypoxia
5. Weakness, fatigue, and malaise resulting from decreased cardiac output
6. Palpitations
7. Vague gastrointestinal complaints, including anorexia, nausea, and vomiting because of visceral congestion and venous stasis (Mangan, 1992; Pass, 1993).

-
- Patients with malignancies that metastasize to the pericardium, such as breast, lung, esophagus, gastrointestinal, and hepatic malignancies; sarcoma; and melanoma
 - Patients with hematologic malignancies, such as leukemia and lymphoma
 - Patients with primary tumors of the heart, such as mesothelioma and sarcoma
 - Patients who have been treated with radiation therapy of 4000 cGy or greater to the mediastinal area
-

Figure 1. Patients With Cancer at Risk for Development of Cardiac Tamponade

Note. Based on information from Hunter, 1998; Knoop & Willenberg, 1999.

Late symptoms of cardiac tamponade include

1. Increasing retrosternal chest pain, causing patients to assume a forward-leaning position
2. Progressive dyspnea at rest and orthopnea caused by decreased cardiac output and hypoxia, as well as decreased lung expansion caused by the enlarged mediastinum
3. Peripheral edema resulting from venous congestion
4. Confusion, restlessness, or apprehension caused by hypoxia and decreased cerebral perfusion (Beauchamp, 1998; Hunter, 1998; Knoop & Willenberg; Mangan; Schafer 1997; Shepherd).

Physical Examination

Pericardial effusions frequently are not suspected until the late signs and symptoms of cardiac tamponade occur. This is primarily a result of the fact that early symptoms may be nonspecific or falsely attributed to tumor progression. In addition, symptoms of increased pericardial effusion and cardiac tamponade resemble symptoms of pulmonary complications and often are mistaken for pulmonary or pleural metastasis. Many patients with malignant pericardial tamponade also have pleural effusions (i.e., accumulation of fluid in the pleural space between the lung and chest wall), as well as malignant parenchymal pulmonary involvement (Beauchamp, 1998; Shepherd, 1997). Consequently, diagnosis of pericardial effusion and tamponade is challenging, although imperative, because if not recognized and treated aggressively, the outcome potentially is fatal.

After consideration of risk factors and review of symptoms indicative of cardiac tamponade, a physical examination must be completed. Possible early signs of cardiac tamponade include

1. Muffled heart sounds, undetectable or weak apical pulse, or positional pericardial friction rub caused by increased pericardial fluid
2. Mild tachycardia of 100 beats per minute caused by compensatory adrenergic stimulation
3. Mild peripheral edema and abdominal distention as a result of venous and visceral congestion
4. Fever caused by an inflammatory process in the pericardium (Beauchamp, 1998; Bickley, 1999; Hunter, 1998; Mangan, 1992).

As cardiac tamponade progresses, patients will appear critically ill. Physical examination may reveal pulsus paradoxus or hepatojugular reflux, which are possible features of cardiac tamponade (Schafer, 1997). Pulsus paradoxus is a decrease or absence of the amplitude of the pulse on inspiration (Beauchamp, 1998). Normally, a decrease in systolic blood pressure occurs with inspiration because breathing causes cyclic cardiocirculatory changes (Knoop & Willenberg, 1999). However, this mechanism is exaggerated during cardiac tamponade because the myocardium is constricted and inspiration causes the diaphragm to exert additional pressure on the pericardial sac. The left ventricle receives less blood, causing decreased stroke volume, decreased cardiac output, and, therefore, decreased blood pressure during inspiration (Beauchamp; Mangan, 1992; Schafer).

Pulsus paradoxus often is a late sign of cardiac tamponade that can be detected by assessing blood pressure with a sphygmomanometer. Inflate the cuff to 20 mmHg above the normal systolic pressure. While slowly deflating the cuff, observe the patient's respiratory rate and note the mmHg when Kortokoff

sounds (i.e., the tapping sounds heard during auscultation of a blood pressure) are heard only during expiration. Note the mmHg when sounds are heard during both inspiration and expiration. Pulsus paradoxus is a difference greater than 10 mmHg between the first Kortokoff sound during expiration and the mmHg when sounds are heard during both inspiration and expiration. In patients with cardiac tamponade, sounds are heard during expiration alone for a prolonged period of time (Beauchamp, 1998; Mangan, 1992).

Hepatojugular reflux is defined as an elevation in jugular venous pressure by 1 cm or more (Schafer, 1997). Elevate the head of the bed to a position at which jugular venous pulsations are visible, usually 30°–45°. Exert pressure continuously over the right upper quadrant of the abdomen for 30–60 seconds while observing the jugular pressure. Increased venous congestion causes an increase in jugular pressure with abdominal pressure resulting in positive hepatojugular reflux (Dietz & Flaherty, 1993; Schafer).

In addition to pulsus paradoxus and hepatojugular reflux, other late signs of cardiac tamponade include

1. Resting tachycardia greater than 100 beats per minute caused by adrenergic stimulation to compensate for reduced cardiac output
2. Tachypnea caused by decreased cardiac output, hypoxia, and limited expansion of the lung resulting from the enlarged mediastinum
3. Hypotension from reduced cardiac output
4. Narrow pulse pressure caused by compression of the heart that, in turn, causes decreased systolic and increased diastolic blood pressure
5. Pale, ashen, and diaphoretic skin with cool, clammy extremities and peripheral cyanosis from reduced cardiac output, hypoxia, and peripheral vasoconstriction
6. Increased central venous pressure, marked jugular venous pressure, ascites, hepatomegaly, and peripheral edema, caused by venous congestion
7. Oliguria progressing to anuria as a result of decreased renal blood flow
8. Impaired consciousness, progressing to obtundation, caused by decreased cerebral perfusion (Beauchamp, 1998; Hunter, 1998; Knoop & Willenberg, 1999; Mangan, 1992; Schafer, 1997; Shepherd, 1997).

Diagnostic Studies

Although not diagnostic for cardiac tamponade, a routine chest x-ray and electrocardiogram (ECG) may be the initial tests that identify abnormalities and reveal the need for more diagnostic testing (Mangan, 1992). An anterior-posterior chest x-ray may demonstrate an enlarged cardiac silhouette resulting from the increased amount of fluid in the pericardial sac (Lawler, 1999). Chest x-rays in more than half of patients with pericardial effusions demonstrate cardiac enlargement, mediastinal widening, or hilar adenopathy (DeMichele & Glick, 2001). The heart may develop a water-bottle appearance on chest x-ray, with loss of the normal contours of the pericardial reflection (Pass, 1993). However, the chest x-ray cannot differentiate between possible causes of an enlarged heart shadow, and, because of this lack of specificity, the chest x-ray does not provide conclusive evidence to support a pericardial effusion (Mangan; Schafer, 1997).

The ECG changes often are nonspecific, but may show tachycardia, premature contractions, and electrical alternans

(i.e., an ECG pattern of alternating amplitude of the P wave and the QRS complex with every other beat, caused by excessive heart movement in the increased fluid of the pericardium) (DeMichele & Glick, 2001; Knoop & Willenberg, 1999; Mangan, 1992). Diffused low voltage (less than 5 mm) in the limb leads and precordial leads of an ECG can be an indication of significant pericardial effusion.

The most specific and sensitive noninvasive test for pericardial effusion or cardiac tamponade is the two-dimensional echocardiogram (2-D echo), which has become the imaging technique of choice when malignant pericardial effusion is suspected (DeMichele & Glick, 2000; Shepherd, 1997). A 2-D echo uses painless ultrasound waves to create a picture of the heart, including the function of the heart. The classic echocardiographic finding suggestive of cardiac tamponade is collapse of the right atrium and ventricle because of the pressure of the increased pericardial fluid (Beauchamp, 1998). One benefit of the 2-D echo is that it can be completed at bedside if patients are too ill to be transported. Other imaging techniques used to detect cardiac tamponade may be magnetic resonance imaging and computerized tomography scans, both of which can detect small and large effusions, pericardial thickening, and pericardial masses (Lawler, 1999; Shepherd).

Medical Management of Cardiac Tamponade

Cardiac tamponade is a life-threatening emergency. The immediate goal of treatment for cardiac tamponade is the removal of the pericardial fluid to restore hemodynamic stability. The degree of decrease in cardiac output and hemodynamic instability must be considered because emergency intervention may be needed to prevent a fatal outcome.

Supportive care of patients with cardiac tamponade includes ongoing pharmacologic therapy to maintain blood pressure and cardiac functioning during all treatment modalities (Uaje et al., 1996). Mild cardiac tamponade initially may be treated cautiously with diuretics, such as furosemide or spironolactone, as well as with corticosteroids, such as prednisone. Administration of blood products, plasma, and saline will expand circulatory volume and delay circulatory collapse. Oxygen therapy may be initiated. Vasoactive drugs may be used to maintain perfusion. Isoproterenol can increase heart rate and low-dose dopamine can improve cardiac contractility (Beauchamp, 1998; Braunwald, 1997; Schafer, 1997).

Several options for treatment of cardiac tamponade exist. Percutaneous pericardiocentesis, a drainage technique frequently used as the initial treatment for cardiac tamponade, also is useful as a diagnostic tool (Beauchamp, 1998; Harkin et al., 1997; Knoop & Willenberg, 1999; Lawler, 1999; Mangan, 1992; Pass, 1993). Under guidance by either cardiac catheterization or 2-D echo, a needle is inserted percutaneously into the pericardial sac to aspirate the fluid and decompress the tamponade. Echocardiographically guided pericardiocentesis can be performed at bedside. Unguided aspiration only should be used in extreme emergency situations. The benefit of pericardiocentesis is that the tamponade is relieved quickly, but complications of the procedure include puncture of the cardiac muscle, abscess, dysrhythmia, and infection.

Pericardiocentesis also is useful as tool for cytologic diagnosis. The aspirated pericardial fluid is classified as either a transudate or an exudate. A transudate, which has low protein levels, is a fluid that has leaked from blood vessels as a result

of mechanical factors, such as cirrhosis. An exudate, which is rich in protein, is a fluid that has leaked from blood vessels with increased permeability. Malignant effusions often are exudates that contain cellular debris resulting from irritation of the serous membrane by sloughed cancer cells (Maxwell, 1997). Malignant effusions usually are serosanguinous, as compared to the clear straw color of normal pericardial fluid (Schafer, 1997). Cytologic examination of the fluid is necessary to determine the etiology of the effusion and determine appropriate medical management.

Pericardial fluid may reaccumulate, therefore a definitive medical or surgical treatment usually follows pericardiocentesis to prevent recurrence of cardiac tamponade.

Pericardial sclerosis involves instillation of chemicals into the pericardial sac through a pericardial catheter after the pericardial space has been drained. The purpose is to create an inflammatory response with resultant fibrosis and sclerosis to prevent reaccumulation of fluid in the pericardial space. Some of the chemicals used are doxycycline, bleomycin, cisplatin, or vinblastine (Hunter, 1998; Pass, 1993).

Surgical procedures also can provide effective palliative treatment for recurrent malignant cardiac tamponade. A partial pericardiectomy, or pericardial window, creates a surgical opening in the pericardium to drain fluid into the pleural or peritoneal compartments. However, pericardial drainage will be less than optimal if a pleural effusion or peritoneal effusion (i.e., ascites) already exists (Harken et al., 1997). A total pericardiectomy (i.e., removal of the visceral pericardium) is used to treat radiation-induced constrictive pericarditis (Hunter, 1998). These procedures are performed in the operating room under general anesthesia (Beauchamp, 1998). Another surgical procedure is percutaneous balloon pericardiectomy, which uses a balloon-tipped catheter to tear the pericardium and create a window for drainage. This procedure usually is performed in a cardiac catheterization lab.

After the patient has been stabilized, long-term goals can be addressed, including treatment of any underlying malignancy (Schafer, 1997). Treatment choices will depend on patients' primary diagnosis, performance status, and stage and aggressiveness of the disease (Lawler, 1999; Mangan, 1992). Radiation therapy can be used when tumors of the pericardium are radiosensitive. Radiation therapy is contraindicated when the area involved has been irradiated previously. Systemic anti-neoplastic therapy may be administered if the malignancy is chemotherapy sensitive (DeMichele & Glick, 2001).

Nursing Care

Recognition of early signs and symptoms of pericardial effusion and cardiac tamponade can allow for treatment before life-threatening circulatory collapse occurs. Nurses frequently are able to perceive subtle changes in patient status. Accurate and thorough ongoing assessment of cardiopulmonary and hemodynamic status is necessary to identify early abnormal changes. Nursing assessment includes strict monitoring of vital signs, including assessment for pulsus paradoxus, as well as assessment of level of consciousness, ECG tracings, respiratory status, and skin and temperature changes (Smeltzer & Bare, 1996). Accurate monitoring of intake and output is necessary, including assessment for edema or oliguria and anuria. Regular assessment also includes examination for positive hepatjugular reflux as well as measurement of abdominal girth to detect ascites (Schafer, 1997).

Nursing interventions include pain management, care of pericardial catheters, position changes to minimize shortness of breath, postoperative care, and administration of oxygen (Hunter, 1998; Schafer, 1997). Instructions regarding appropriate and timely physician notification of complications that occur after discharge can help to initiate early interventions. Assessment and intervention for the side effects of radiation therapy, such as fatigue and skin alterations, as well as the side effects of chemotherapy, such as nausea, vomiting, stomatitis, or pancytopenia, can aid in minimizing symptoms. Nurses should assess for possible ineffective coping and depression. Referral for home nursing services or hospice care should be considered as needed.

Additional Cardiac Complications of Cancer Therapy

In addition to pericardial effusion and cardiac tamponade, people with cancer may develop any cardiovascular disease (Keefe, 2000). Oncology nurses should be aware that acute heart failure might be a complication experienced by patients with cancer and that heart failure occasionally is caused by the cancer treatment itself. Anthracyclines, such as doxorubicin, and the anthracenedione mitoxantrone can cause signs and symptoms of heart failure as a result of treatment-induced cardiomyopathy caused by myocyte damage and myocardial cell loss (Keefe; Steinherz & Yahalom, 1993). ECG changes that have been detected after administration of anthracyclines include supraventricular dysrhythmias, heart block, and ventricular tachycardia. In addition, ECG-gated pool scans can be completed to assess global left ventricular function and ejection fraction. The ECG-gated pool scan is a procedure in which the measured amount of radioactive tracer in the chambers of the heart is proportional to the volume of blood in the chambers (Bullock & Henze, 2000). ECG-gated pool scans have shown major decreases in ejection fraction that reach a nadir within 24–48 hours after anthracycline administration. As a result, congestive heart failure can occur in certain patients (Chabner & Myers, 1993).

Tachycardia is the initial sign that an anthracycline is causing cardiomyopathy, followed by decreased exercise tolerance. However, these signs sometimes are overlooked and attributed to other causes, such as anemia, pain, or fever (Keefe). Fluid overload occurs next. The response of a normal heart to increased blood volume usually is dilatation of the chambers of the heart and hypertrophy of the ventricles (Bullock, 2000). However, anthracycline-induced cardiomyopathy is associated with little increase in left ventricular size, and,

therefore, patients may experience severe symptoms of heart failure (Keefe). Avoidance of anthracycline-induced cardiomyopathy includes measurement of the ejection fraction and ongoing evaluation of patients for symptoms, including referral to a cardiologist for high-risk patients, monitoring cumulative dose, and dose adjustment or discontinuation when necessary. Treatment includes diuretics, digitalis, and angiotensin-converting enzyme inhibitors.

Cardiac complications can occur as a side effect of other cancer treatments. Trastuzumab (Herceptin® [Genentech, Inc., South San Francisco, CA]) is a recently developed monoclonal antibody to the HER2 receptor (Cooper & Cooper, 2001; Keefe, 2000). The incidence of heart failure increases when trastuzumab is administered in conjunction with an anthracycline such as doxorubicin or epirubicin. Therefore, a baseline ejection fraction always should be obtained before administration of trastuzumab to detect underlying asymptomatic left ventricular dysfunction (Keefe). Patients must be monitored closely during therapy with trastuzumab.

Cardiotoxicity also can be caused by administration of 5-fluorouracil, which can cause coronary artery spasm with resultant angina, dysrhythmias, myocardial infarction, cardiac arrest, or sudden death (Keefe, 2000; Steinherz & Yahalom, 1993). Cardiac toxicity occurs very rarely with initial doses and is more common with subsequent doses toward the end of a five-day infusion. Deaths have been reported to occur in symptomatic patients who continued therapy despite warning signs and symptoms (Keefe).

Conclusion

Nurses must be aware that cardiac complications can occur as a complication of cancer treatment. Oncology nurses commonly administer chemotherapy and have frequent and prolonged encounters with patients. Ongoing, accurate, and thorough cardiovascular assessment is necessary to detect dysrhythmias or changes in functional status, such as increased weakness, decreased endurance, or exercise intolerance. Early detection of subtle changes and signs and symptoms of cardiac complications can prevent increased cardiotoxicity.

The author would like to thank John Sprandio, MD, of Consultants in Medical Oncology and Hematology, Drexel Hill, PA, for reviewing this manuscript.

Author Contact: Jo Ann Flounders, MSN, CRNP, OCN®, CHPN, can be reached at joann@theflounders.com, with copy to editor at rose_mary@earthlink.net.

References

- Beauchamp, K. (1998). Pericardial tamponade: An oncologic emergency. *Clinical Journal of Oncology Nursing*, 2, 85–95.
- Bickley, L. (1999). *Bates' guide to physical examination and history taking* (7th ed.). Philadelphia: Lippincott.
- Braunwald, E. (1997). Cardiac tamponade. In E. Braunwald (Ed.), *Heart disease: A textbook of cardiovascular medicine* (pp. 1446–1496). Philadelphia: Saunders.
- Bullock, B. (2000). Altered cardiac function. In B. Bullock & R. Henze (Eds.), *Focus on pathophysiology* (pp. 455–502). Philadelphia: Lippincott.
- Bullock, B., & Henze, R. (Eds.). (2000). *Focus on pathophysiology*. Philadelphia: Lippincott.
- Chabner, B., & Myers, C. (1993). Antitumor antibiotics. In V. DeVita, S. Hellman, & S. Rosenberg (Eds.), *Cancer principles and practice of oncology* (4th ed., pp. 374–384). Philadelphia: Lippincott-Raven.
- Cooper, M., & Cooper, M. (2001). Systemic therapy. In R. Lenhard, R. Osteen, & T. Gansler (Eds.), *Clinical oncology* (pp. 175–215). Atlanta, GA: American Cancer Society.
- DeMichele, A., & Glick, J. (2001). Cancer-related emergencies. In R. Lenhard, R. Osteen, & T. Gansler (Eds.), *Clinical oncology* (pp. 733–764). Atlanta, GA: American Cancer Society.
- Dietz, K., & Flaherty, A. (1993). Oncologic emergencies. In S. Groenwald, M. Frogge, M. Goodman, & C. Yarbro (Eds.), *Cancer nursing* (3rd ed., pp. 800–839). Boston: Jones and Bartlett.

- Harken, A., Hammond, G., & Edmunds, L. (1997). Pericardial diseases. In L. Edmunds (Ed.), *Cardiac surgery in the adult* (pp.1303–1317). New York: McGraw Hill.
- Hunter, J. (1998). Structural emergencies. In J. Itano & K. Taoka (Eds.), *Core curriculum for oncology nursing* (3rd ed., pp. 340–354). Philadelphia: Saunders.
- Keefe, D. (2000). Cardiovascular emergencies in the cancer patient. *Seminars in Oncology*, 27, 244–255.
- Knoop, T., & Willenberg, K. (1999). Cardiac tamponade. *Seminars in Oncology Nursing*, 15, 168–173.
- Lawler, P. (1999). Effusions. In C. Yarbrow, M. Frogge, & M. Goodman (Eds.), *Cancer symptom management* (2nd ed., pp. 419–433). Boston: Jones and Bartlett.
- Mangan, C. (1992). Malignant pericardial effusions: Pathophysiology and clinical correlates. *Oncology Nursing Forum*, 19, 1215–1223.
- Markiewicz, W., Borovik, R., & Ecker, S. (1986). Cardiac tamponade in medical patients: Treatment and prognosis in the echocardiographic era. *American Heart Journal*, 111, 1138–1142.
- Maxwell, M. (1997). Malignant effusions and edemas. In S. Groenwald, M. Frogge, M. Goodman, & C. Yarbrow (Eds.), *Cancer nursing* (4th ed., pp. 721–741). Boston: Jones and Bartlett.
- McAllister, H., Hall, R., & Cooley, D. (1999). Tumors of the heart and pericardium. *Current Problems in Cardiology*, 24, 57–116.
- Pass, H. (1993). Malignant pleural and pericardial effusions. In V. DeVita, S. Hellman, & S. Rosenberg (Eds.), *Cancer principles and practice of oncology* (pp. 2586–2598). Philadelphia: Lippincott-Raven.
- Schafer, S. (1997). Oncologic complications. In S. Otto (Ed.), *Oncology nursing* (3rd ed., pp. 406–476). St. Louis: Mosby.
- Shepherd, F. (1997). Malignant pericardial effusion. *Current Opinion in Oncology*, 9, 170–174.
- Smeltzer, S., & Bare, B. (1996). Oncology: Nursing the patient with cancer. In S. Smeltzer & B. Bare (Eds.), *Brunner and Suddarth's textbook of medical-surgical nursing* (8th ed., pp. 309–316). Philadelphia: Lippincott-Raven.
- Steinherz, L., & Yahalom, J. (1993). In V. DeVita, S. Hellman, & S. Rosenberg (Eds.), *Cancer principles and practice of oncology* (pp. 2370–2385). Philadelphia: Lippincott-Raven.
- Uaje, C., Kahsen, K., & Parish, L. (1996). Oncology emergencies. *Critical Care Nursing Quarterly*, 18(4), 26–34. 