



## Perioperative issues in patients with cancer

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General internists and family practice physicians frequently act as primary care physicians or consultants in the perioperative assessment and management of patients with cancer. Among patients with cancer, approximately 75% undergo surgical resection for cure [1], and about 90% for cure or palliation [2]. The perioperative care of a cancer patient can be challenging because of several disease-related considerations. Perhaps the primary consideration is the patient's diagnosis, including the extent of disease and planned cancer-related treatment to be performed in addition to surgery. The natural history of the cancer and the effects of any previous chemotherapy or radiation therapy should also be considered. In managing care of these patients, as with any preoperative evaluation, the physician should have a basic understanding of the surgical procedure and should know whether the surgery is intended for cure or palliation. The physician must also obtain a thorough medical history and physical examination to assess adequately comorbid conditions, such as hypertension, diabetes, cardiac, pulmonary, or renal disease. The preoperative medical evaluation should include an assessment of the patient's nutritional status, functional status, and pain. In general, organ dysfunction is managed with standard treatments, regardless of whether a patient has cancer. Physicians should pay particular attention to pain management, which is overlooked too frequently by clinicians despite the high priority patients often place on this aspect of their care. Finally, because most patients with cancer who are

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undergoing surgery have many caregivers, including surgeons, oncologists, radiation oncologists, and possibly other medical subspecialists, one role of the general internist often is to coordinate the patient's overall care. Frequently, the physician knows the patient well enough to have a frank conversation with the patient and the patient's family about the expectations of treatment. This conversation can provide an opportunity to ascertain the patient's wishes regarding resuscitation. In sum, general internists frequently play an important and complex role in the perioperative care of patients with cancer. Indeed, the complexity of this role has led to proposals that hospitals should implement coordination mechanisms to improve delivery of multidisciplinary care to patients with cancer [3].

### **Direct and indirect effects of cancer**

#### *Performance status*

Patients with cancer vary widely in their performance status. Some patients are ambulatory and fully functional in activities of daily living, whereas others are severely debilitated. A patient's performance status depends on many factors, including the type of cancer, extent of disease, side effects of cancer-related treatments, and presence of comorbid medical conditions. Performance status reflects many disparate parameters and is a general and robust prognostic indicator for surgical outcome and mortality [4].

#### *Malnutrition*

Patients with cancer can be significantly malnourished for a variety of reasons. They can have impaired eating and drinking functions because of pain, nausea, stomatitis, or tumor of the oropharynx or gastrointestinal tract. Vigorous nutritional replacement for 1 week before surgery in patients with at least moderate undernutrition has been shown to reduce perioperative mortality and morbidity, including postoperative wound infection [5,6]. Both parenteral and enteral nutritional replacements have been effective, and the use of nutrients that modulate the immune system might have additional benefits [7].

#### *Pain*

Cancer, particularly in cases of advanced disease, is frequently accompanied by pain recalcitrant to standard therapies. Treating the patient's pain and addressing the concerns of the patient and his or her family about addiction to pain medication are important aspects of the preoperative evaluation. Referral to a pain specialist may be appropriate, but the physician can usually provide significant palliation in the immediate perioperative period. One fundamental approach to treatment includes (1) a base of

scheduled acetaminophen or a nonsteroidal anti-inflammatory drug; with (2) an opiate (scheduled or as needed); and (3) if needed, a low dose of a sedating tricyclic antidepressant, such as amitriptyline, 10 to 50 mg, at bedtime for sleep. In some patients, nonsteroidal anti-inflammatory drugs should be avoided in the preoperative period because of the potential for platelet inhibition and additional operative blood loss. The specific opiate used and the route of administration should be individualized on the basis of the likely palliative effect and toxic effects for that patient. It is important to discuss the inevitable sedation and constipation that accompany opiate use and to guide patients on how to manage these side effects.

### *Cardiopulmonary considerations*

Patients with tumors in or adjacent to the upper airway are at risk for airway obstruction. Laryngeal obstruction is the most common type of airway obstruction among patients with head and neck tumors. Stridor or other concerning findings at the time of preoperative medical evaluation should be assessed on the same day by an otolaryngologist. High-dose steroids, such as dexamethasone, can effectively relieve obstruction by reducing edema. High-dose steroids, however, are likely to cause insulin resistance and can cause delirium. Furthermore, even with preoperative steroids, some patients require tracheotomy to bypass laryngeal obstruction before tumor resection.

In patients with an anterior or middle mediastinal mass, airway obstruction or cardiopulmonary arrest can develop in any phase of general anesthesia [8]. The preoperative evaluation of a patient with an anterior or middle mediastinal mass should include a detailed review for respiratory symptoms including stridor, dyspnea, wheezing, and orthopnea. In addition, a CT or MRI of the chest should be obtained and echocardiography, to look for cardiac compression by the mass, and spirometry, including inspiratory and expiratory flow volume loops [9]. Patients with any evidence of tracheobronchial tree or cardiac compression may require special anesthetic care and precautions including (1) fiberoptic intubation while awake; (2) spontaneous ventilation throughout surgery; (3) capability quickly to reposition the patient to lateral, prone, or sitting position as needed; (4) availability of a rigid bronchoscope in the event of a collapsed airway; and (5) standby femoral-femoral cardiopulmonary bypass in the event of cardiovascular collapse [9].

Among patients with cancer, pericardial effusion can be caused by chemotherapy or infection, but is most commonly caused by metastases to the pericardium. Patients with a significant pericardial effusion may have progressive dyspnea, chest discomfort, and, sometimes, upper abdominal discomfort that is usually relieved when the patient leans forward. Findings on physical examination in patients with pericardial effusion include jugular venous distention; narrowed pulse pressure; distant heart sounds; and excessive respiratory variation in blood pressure (pulsus paradoxus). Electrocardiograms show low voltage. Echocardiography confirms the diagnosis and

allows assessment of the impact of the effusion on diastolic filling and global cardiac function [10]. Furthermore, echocardiography can help guide transcatheter pericardial drainage performed with local anesthesia [11]. Sclerosis sometimes provides lasting treatment; the effusion is drained, and 30 to 60 mg of bleomycin is infused through the drainage catheter [12]. Although definitive drainage of a malignant pericardial effusion can be obtained with subxiphoid pericardotomy [13], systemic chemotherapy can also often provide substantial palliation [14].

In patients with cancer, pleural effusions can develop, owing either to toxicity of treatment or, more likely, to the cancer itself. Preoperative thoracentesis is not always necessary; however, spirometry should be performed and arterial blood gases measured before elective surgery for patients with symptomatic or otherwise evident pleural effusions. Tisi et al [15] have suggested criteria for identifying patients who require thoracentesis. In the postoperative period, patients who have undergone thoracentesis should be monitored closely and regularly for the reaccumulation of pleural fluid (eg, monitor with chest radiography).

### *Endocrine and metabolic*

Hyponatremia in patients with cancer can be a direct consequence of a brain tumor, a result of a paraneoplastic syndrome, or a toxic side effect of medication, and can be exacerbated by poor oral intake. Common presentations of hyponatremia include malaise, fatigue, and confusion, but delirium, seizure, coma, and death do occur. Furthermore, even among patients who do not have brain tumors, focal neurologic deficits can be a presenting symptom of hyponatremia [16]. Patients with a rapid decrease in serum sodium concentration are more likely to have symptoms; however, patients with serum sodium above 130 mEq/L are rarely symptomatic. The proximate cause of hyponatremia is often the syndrome of inappropriate secretion of antidiuretic hormone. Some cancers, such as small cell lung cancer, are more frequently associated with syndrome of inappropriate secretion of antidiuretic hormone. Criteria for diagnosis of syndrome of inappropriate secretion of antidiuretic hormone are a serum sodium level less than 135 mEq/L, plasma osmolality less than 280 mOsm/kg, urine sodium greater than 20 mEq/L, and urine osmolality greater than 500 mOsm/kg. In addition, patients with syndrome of inappropriate secretion of antidiuretic hormone usually have normal renal, adrenal, and thyroid functions, and a normal extracellular fluid volume. In managing patients with hyponatremia, the physician must carefully review all medications, because excretion of free water can be impaired by many medications, including morphine, cyclophosphamide, vincristine, chlorpropamide, amitriptyline, clofibrate, and thiazide diuretics. Discontinuation of such medication and restriction of fluids is the first line of treatment in mild cases of hyponatremia. Recalcitrant cases require acute therapy, which includes intravenous administration of saline and treatment with a

loop diuretic to induce excretion of free water. Long-term treatment for recalcitrant cases can include both restriction of fluids to 500 to 1000 mL/d and orally administered demeclocycline, 300 to 600 mg twice daily. Treatment of the cancer, however, such as chemotherapy for patients with small cell lung cancer, may be the most effective therapy for hyponatremia. Although it is preferable to correct hyponatremia before surgery, asymptomatic hyponatremia is not associated with increased perioperative risk, provided that the patient maintains a normal extracellular fluid volume [17].

Hypercalcemia is a common complication of cancer, occurring in up to 5% of all cancer patients [18]. The cancers most commonly associated with hypercalcemia are breast cancer, non-small cell lung cancer, and multiple myeloma. Hypercalcemia also occurs in patients with renal cell carcinoma and squamous cell cancer of the upper orodigestive tract. In general, the prognosis is poor because hypercalcemia is a manifestation of advanced cancer [19]. Exceptions are hypercalcemia caused by breast cancer or multiple myeloma, which may respond well to treatment. Regardless of the malignancy, treatment of the hypercalcemia is beneficial because it can help to relieve symptoms and improve quality of life. The symptoms of hypercalcemia are nonspecific and include fatigue, nausea, abdominal pain, constipation, depression, and delirium. Patients with a serum calcium concentration greater than 14 mg/dL usually are symptomatic. As with hyponatremia, symptoms are usually a function of both the degree of hypercalcemia and the rapidity with which it develops. Findings of electrocardiography include a prolonged QT interval and a flattened T wave. The severity of the hypercalcemia should determine the aggressiveness of the therapy. Patients with significant hypercalcemia are usually dehydrated. Acute treatment includes vigorous intravenous hydration and concomitant diuresis with a loop diuretic. In the perioperative period, the therapeutic goal is to normalize both the intravascular volume and the calcium level. Although it is preferable to correct hypercalcemia before surgery, asymptomatic hypercalcemia is associated with minimal risk in euvolemic patients if the corrected calcium concentration is maintained at less than 12 mg/dL [20,21]. Longer-term treatment has traditionally included focal radiation therapy to shrink bone metastases; however, intravenous or oral bisphosphonates act systemically to prevent and treat cancer-related osteoporosis and to relieve bone pain. Determining the best type of bisphosphonate to use and the optimal route of administration and dosing schedule is an active area of research.

### *Hematologic considerations*

Hypercoagulability should be assumed in patients with cancer and may be caused by increased plasma levels of clotting factors, cytokines, cancer procoagulant A, or increased release of tissue plasminogen activator. Several tumor types are more commonly associated with thrombosis, especially

mucin-producing adenocarcinomas, such as those arising from the pancreas, lung, and gastrointestinal tract. The risk of postoperative deep vein thrombosis is as high as 29% among all patients with cancer [22] and is even higher among patients with additional risk factors for deep vein thrombosis, such as obesity, advanced age, orthopedic or neurologic surgery, and impaired mobility. In evaluating a patient with cancer before surgery, the physician faces the challenging task of estimating the risk for perioperative deep vein thrombosis and, in collaboration with the surgeon, determining the appropriate level of prophylactic treatment. A range of effective, prophylactic treatments is available. Static compression stockings are effective and have few substantial side effects. Pneumatic compression stockings are even more effective, but are more expensive and many patients find them less comfortable and more difficult to use. Stockings can be combined with medication for anticoagulation, which decreases the risk of clotting, but increases the risk of bleeding. The least aggressive and simplest medication for anticoagulation is low-dose, unfractionated heparin, 5000 U administered subcutaneously twice or three times daily. More aggressive and more expensive, but better controlled treatment is titrated, intravenously administered unfractionated heparin, or subcutaneous low-molecular-weight heparin at prophylactic doses. The same anticoagulant medications at higher (ie, treatment) doses provide the most aggressive prophylactic therapy against deep vein thrombosis. The goal of therapy is to balance the patient's risk of deep vein thrombosis with the risk of bleeding complications, a difficult clinical judgment for which no formula or protocol is available. Determining the duration of anticoagulation after surgery is also a challenging task. Several studies have indicated that, in high-risk patients, such as those undergoing surgery for abdominal or pelvic cancer, 1 month of postoperative anticoagulation is superior to shorter durations [23].

### *Thrombocytosis*

Thrombocytosis in patients with cancer can be a consequence of splenectomy, iron deficiency, or more commonly a subacute inflammatory condition. Treatment with anagrelide (Agrylin) to reduce the platelet count must be based on a consideration of the individual's risk for thrombosis, but this drug generally is used at doses needed to maintain a platelet count below 1,000,000/mm<sup>3</sup>.

### *Granulocytosis*

Granulocytosis or leukemoid reaction to the cancer without infection occurs with a variety of solid tumors and lymphomas. Evaluation of the patient's history, physical examination, and limited laboratory testing is appropriate to rule out infection, but specific treatment is rarely needed.

### *Neurologic considerations*

Neurologic problems are common in patients with cancer [24]. Except for routine chemotherapy, neurologic complications are the most common reason for hospitalization of patients with cancer. Brain metastases are the most frequent neurologic complication, occurring in up to one third of patients with cancer in some settings. The most typical presenting symptoms are headache, confusion, delirium, and focal neurologic deficit. Papilledema is an unreliable sign of central nervous system metastases, occurring in only about one fourth of patients with such metastases. Leptomeningeal metastases and epidural spinal cord compression are also common. Leptomeningeal metastases should be suspected in patients who develop multifocal neurologic deficits, especially multifocal cranial neuropathies. Lung and breast cancers, melanoma, and lymphoma are common causes of leptomeningeal metastases. Emergent treatment of spinal cord compression with high-dose corticosteroid, radiation therapy, or surgical decompression is often necessary to preserve neurologic function. Delirium is not always caused by metastatic tumor; cancer and treatment-related metabolic encephalopathy are common nonmetastatic causes of delirium. Stroke can be caused by a variety of tumor-related conditions, including direct tumor invasion; side effects of chemotherapy; and cancer-related coagulopathy, such as thrombotic (marantic) endocarditis. Radiation therapy is the most commonly used palliative treatment for brain metastases. Chemotherapy or surgical resection of tumors may be appropriate for selected patients. Paraneoplastic syndromes that affect neuromuscular function are relatively rare but are of particular concern in the perioperative period because treatment with anesthetic agents can exacerbate neuromuscular dysfunction, leading to respiratory failure or delayed extubation. For example, myasthenia gravis occurs in up to 50% of patients with thymoma, and Lambert-Eaton myasthenia occurs in up to 5% of patients with small cell lung cancer [25]. Symptoms of Lambert–Eaton syndrome include proximal muscle weakness, diminished or absent deep tendon reflexes, and autonomic neuropathy. Treatment of the cancer can mitigate these symptoms, whereas calcium channel antagonists can exacerbate the symptoms and are contraindicated [26].

## **Effects of cancer therapy**

### *Chemotherapy*

When evaluating a cancer patient before surgery, the physician should obtain a list of the chemotherapeutic agents the patient has received and the dose, schedule of administration, and dates of therapy. Toxicity of chemotherapy can develop months after administration is completed and can affect many organ systems. The toxicity of some agents, such as bleomycin and doxorubicin, is strongly correlated with the cumulative dose.

### *Pulmonary toxicity*

Bleomycin can have significant pulmonary toxicity in up to 10% of patients, at least in part because lung tissue is deficient in the enzymes that inactivate this drug. Bleomycin pulmonary fibrosis is usually associated with cumulative doses greater than 450 U; however, there are reports of this effect at lower doses [27]. Advanced patient age, the presence of pre-existing lung disease, and a history of radiation therapy can lower the total dose at which toxicity is likely to be seen [28]. In the preoperative evaluation of patients with sufficient cumulative doses of bleomycin the physician must look for pulmonary symptoms, including dyspnea, pleuritic chest pain, and nonproductive cough, and physical findings, including basilar fine crackles. Patients who have any such symptoms should be evaluated fully with spirometry, arterial blood gases, and assessment of diffusing capacity. Patients with restrictive lung disease, an increased alveolar-arterial oxygen gradient, and a decreased diffusing capacity are likely to benefit from special pulmonary care in the perioperative period [29]. In addition to bleomycin, many other chemotherapeutic agents can also cause interstitial pneumonia or pulmonary fibrosis, including busulfan; chlorambucil; cyclophosphamide; melphalan; methotrexate; nitrosoureas (carmustine BCNU, lomustine CCNU, or semustine-methyl CCNU, all of which cause pulmonary complications in up to 25% of patients); and vinca alkaloids with mitomycin. Many of these agents have also been associated with other specific pulmonary toxic effects including bronchiolitis obliterans with organizing pneumonia, pulmonary infiltrates with eosinophilia, noncardiac pulmonary edema, and pleural effusion. Vinca alkaloids with mitomycin have also been reported to induce or exacerbate asthma [30].

### *Cardiac toxicity*

Doxorubicin and daunorubicin can have considerable adverse effects on the heart. A dose-dependent drug-induced cardiomyopathy can manifest as sinus tachycardia, premature atrial or ventricular contractions, nonspecific ST and T-wave changes, and low-voltage QRS complex [31]. The potentially toxic cumulative doses are 550 mg/m<sup>2</sup> for doxorubicin, and 600 mg/m<sup>2</sup> for daunorubicin [31]. Patient characteristics and exposures, such as pre-existing heart disease, radiation therapy, and exposure to other chemotherapeutic agents, can lower the cumulative dose at which a cardiomyopathy develops.

### *Hepatotoxicity*

Several chemotherapeutic agents are potentially hepatotoxic. Methotrexate commonly causes transient, mild elevations of serum transaminases; however, daily administration of the drug for several months can also cause hepatic fibrosis [32]. Other potentially hepatotoxic chemotherapeutic agents include L-asparaginase; cytosine arabinoside; plicamycin (mithramycin); and 6-mercaptopurine. Although most hepatotoxicity caused by chemotherapeu-

tic agents is transient, hepatic function should be assessed in patients with potential hepatotoxicity. In patients with potential for chemotherapy-related hepatotoxicity, prothrombin time is an appropriate test to ensure that the patient has adequate hepatic synthetic function before surgery is performed.

### *Nephrotoxicity*

Several chemotherapeutic agents are potentially nephrotoxic. For example, *cis*-platinum commonly causes a dose-dependent, transient toxicity to the distal tubular epithelium leading to hypomagnesemia. Streptozotocin frequently causes renal tubular toxicity that can progress to Fanconi's syndrome [33]. Other agents that cause renal toxicity are mitomycin-c, mithramycin, and high-dose methotrexate. For virtually all patients who have recently undergone chemotherapy serum blood urea nitrogen, creatinine, and electrolyte levels should be measured before surgery.

### *Myelosuppression*

In general, chemotherapeutic agents that interfere with DNA synthesis and repair cause myelosuppression. Physicians must try to anticipate the timing of chemotherapy-related myelosuppression and avoid scheduling elective surgery for periods when the patient is neutropenic and thrombocytopenic. If feasible, surgery should be delayed until the myelosuppression has resolved. Recombinant hematopoietic growth factors should be used as necessary to shorten the period of neutropenia ( $< 500/\text{mm}^3$ ). A platelet count of at least  $50,000/\text{mm}^3$  is generally considered adequate for most surgical procedures, but the specific procedure, and the functionality of the platelets, must also be considered.

### *Diabetes mellitus*

High-dose corticosteroid treatment is frequently a component of chemotherapy for lymphoma or an adjunctive treatment to reduce edema or nausea. Such treatment also causes insulin resistance and frequently induces diabetes mellitus in patients with cancer. Patients receiving corticosteroids should undergo frequent monitoring of blood glucose and receive insulin or oral hypoglycemic agents as needed. In addition, streptozocin [33], L-asparaginase [34], interleukin-2 [35], and interferon- $\alpha$  [36] can damage or suppress pancreatic islet cells and cause insulin-deficient diabetes mellitus. Patients who have received these agents also should undergo frequent glucose monitoring and receive insulin as needed.

### *Adrenal insufficiency*

Most commonly, adrenal insufficiency in patients with cancer is secondary to suppression of the hypothalamic-pituitary-adrenal axis because of corticosteroid treatment. A course of corticosteroid lasting at least 2 weeks

can cause measurable suppression for up to 1 year. Other agents that can cause adrenal insufficiency are aminoglutethimide and metyrapone, which are used in the treatment of prostate, breast, and adrenocortical cancers, and mitotane, which is used in the treatment of adrenocortical cancer. If time permits, patients with potential adrenal insufficiency should undergo assessment of the hypothalamic-pituitary-adrenal axis using a corticotropin stimulation test to determine if stress-dose steroid is needed. If such testing cannot be performed before surgery, the following general rules might be useful. Patients taking corticosteroid medication should continue to receive this treatment at the usual dose up to the day of surgery [37]. For procedures involving only minor physiologic stress, the patient's usual dose of steroid should be continued throughout the perioperative period; however, for more stressful procedures, supplemental steroid treatment is indicated. Patients taking prednisone at a dose of 5 mg/d or less usually do not require steroid supplementation. Patients taking prednisone at doses greater than 5 mg/d might have a suppressed hypothalamic-pituitary-adrenal axis and should receive supplemental steroid for physiologically stressful procedures. Furthermore, patients taking prednisone at doses of at least 5 mg/d who develop perioperative complications might also benefit from use of a supplemental steroid. For example, for a procedure involving minor stress, such as inguinal herniorrhaphy, a single dose of either hydrocortisone hemisuccinate, 25 mg, or methylprednisolone, 5 mg, administered intravenously at the start of surgery should be adequate. For a more stressful procedure, such as cholecystectomy or hemicolectomy, hydrocortisone hemisuccinate, 50 mg intravenously every 8 hours, or methylprednisolone, 10 mg administered intravenously every 6 hours starting at the time of surgery and tapered over 1 to 2 days should be adequate. For a major surgery, such as major cardiothoracic or abdominal surgery, hydrocortisone hemisuccinate, 100 mg administered intravenously every 8 hours, or methylprednisolone, 20 mg administered intravenously every 6 hours tapered over 2 to 3 days, should be adequate. Although corticosteroid therapy can be life-saving in selected patients, use of steroid therapy should be limited to minimize consequent osteoporosis, glucose intolerance, and other associated adverse effects.

### **Radiation therapy**

When performing a preoperative evaluation of a patient with cancer or a history of malignancy, the physician should ascertain whether the patient has received radiation therapy, and, if so, what part of the body was irradiated. Radiation-induced scarring of the jaw and neck can cause airway narrowing or limit neck flexibility, but more commonly causes glandular hypofunction, leading to xerostomia, primary hypothyroidism, and primary hypoparathyroidism. Concomitant radical neck dissection or hemithyroidectomy increases the risk of hypothyroidism and hypoparathyroidism

[38,39]. The greater the dose of radiation, the greater the risk of radiation-induced scarring [40,41]. Among patients who received mantle radiation for Hodgkin's lymphoma, the threshold radiation dose for hypothyroidism seems to be about 10 Gy. In one study, 45% of patients who received 30 Gy or more had hypothyroidism 20 years after treatment [39]. In another study, 60% of such patients had an elevated level of thyroid-stimulating hormone 10 to 18 years after treatment [42]. Before surgery the serum thyroid-stimulating hormone level should be measured in patients who have more than a 10 Gy total dose of radiation to the neck. Hypoparathyroidism is less common but should be considered in patients who have hypothyroidism caused by neck radiation.

Hypothalamic-pituitary dysfunction can be caused by radiation therapy to the brain, base of the skull, or upper neck [43]. The total radiation dose and the rate of delivery are proportionate to the severity of the dysfunction. The development of radiation-induced hypothalamic dysfunction is usually insidious and can occur many years after radiation treatment. Panhypopituitarism manifests as hypotension, hypothermia, and hypoglycemia; however, nonspecific fatigue and weakness are the usual presenting symptoms. Testing for serum levels of pituitary hormones can be reserved for patients with symptoms of this condition who have received radiation therapy to areas near the pituitary gland.

Patients who have received radiation therapy to the chest can develop pulmonary and cardiac toxicity. Lymphocytic radiation pneumonitis can develop up to several months after chest irradiation, and may occur outside the radiation field. Chronic pulmonary scarring may also develop [44]. Although focal pulmonary scarring in patients who have received radiation for neck or breast cancer does not usually require any additional testing, patients who have received radiation therapy to more than one third of the chest or who have symptoms might benefit from spirometry and arterial blood gas testing. Chronic radiation-induced pulmonary damage manifests as decreased lung volume and compliance, and an impaired diffusing capacity [27,45]. Radiation therapy to the mediastinum without adequate heart shielding can cause pericarditis. In one study, pericarditis was found in approximately 5% of patients who received at least 40 Gy to more than half their heart volume [46]. Pericarditis can present months to years after radiation treatment and can present as acute pericarditis, as an asymptomatic effusion, as cardiac tamponade, or as constrictive pericarditis [47]. Radiation of the heart is also associated with conduction abnormalities and with premature coronary artery disease [48]. For those who have previously received mediastinal irradiation, preoperative screening with electrocardiography is appropriate, even in young patients.

## **Summary**

The perioperative care of patients with cancer can be an exciting challenge. The physician must consider many factors, including the cancer

diagnosis, the extent of disease, treatment received, the presence of comorbid conditions, and the patient's prognosis and must understand the impact of these factors on the planned surgical procedure. In this setting, the physician has the opportunity to perform an essential role in the perioperative management of patients with cancer.

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