

Surgical Emergencies in the Cancer Patient

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Rondi Marie Kauffmann
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 Springer

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Foreword

Plus ça change, plus c'est la meme chose

Proverb: JBA Karr 1849

Three decades have passed since I edited the textbook “Surgical Emergencies in the Cancer Patient” [1]. This was one of two companion texts, where my colleagues and my mentors shared their knowledge and experience in treating cancer patients by publishing on Medical as well as Surgical Emergencies in the Cancer Patient.

In the Preface to the current book also entitled “Surgical Emergencies in the Cancer Patient,” the editors acknowledge the tremendous changes that have occurred in the practice of Surgical Oncology since then.

This book presents multidisciplinary treatment algorithms that embrace virtually every aspect of Cancer Biology and Therapeutics today. Treatment of the critically ill cancer patients now has far greater treatment options. The specialty-specific complications and emergencies are reviewed in far greater depth than we were able to do so long ago.

Indeed, so much has changed ... but has it? The Yankees’ baseball legend Yogi Berra once said “it’s deja vu all over again,” and in many ways it is. What then remains the same? What has not changed is the fundamental importance of early recognition that there is a problem, a prompt multidisciplinary response, and a well-coordinated rescue, recovery, and follow-up. Complications are rarely “opportunistic,” and a senior-level team response must be available “24/7/365.”

Centers of Excellence provide this level of coverage while attracting sufficient individual surgeon and specialty-specific patient referrals to justify in economic terms the costs involved.

Looking back on the nighttime and weekend surgical emergencies of the past, what has changed is how so many are now handled expeditiously because of technologic advances in and focused expertise with therapeutic endoscopy and image-directed intervention. Consider for a moment how easily deep abscesses are drained and airway, esophageal, colonic, or vascular stents are placed. Tiny ports have replaced incisions as minimally invasive imaging, and instrumentation continues to develop. Forgive an “old-timer” for wondering if a “Fellowship” may one day be needed to gain expertise in open exploration of a “difficult” abdomen or chest.

It is humbling to find that a few copies of a certain book with the same title published in 1987 can still be purchased on the Internet—for as little as 89 cents! Perhaps the Hall of Fame catcher who claimed “he really didn’t say everything he said” would agree—it’s really all the same only different.

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Reference

1. Turnbull ADM. Surgical emergencies in the cancer patient. Chicago: Year Book Medical Publishers Inc; 1987.

Preface

Cancer surgery has made tremendous progress in the last three decades. Surgery is an essential part of any curative strategy for solid tumors, and over the last years, even the most intricate operations have been perfected and deemed safe. Operations such as pancreatectomies, hepatectomies, esophagectomies, or laryngectomies that used to have prohibitive mortality can now be performed in most major centers with less than 3 % mortality. Recent studies have shown that such great outcomes are highly dependent upon recognition and rescue from complications during the recovery process. At times, the rescue requires urgent or emergent surgery.

Cancer patients also develop complications from their primary or metastatic tumors, or from the complex multimodality therapies to which they are subjected. Many of these complications, including bowel obstruction, autoimmune colitis, airway obstruction, abscesses, and other problems, may also require life-saving surgical intervention. Thus, urgent or emergent surgical intervention is instrumental for favorable outcomes in many patients after bone marrow transplantation for hematologic malignancies or for disseminated solid tumors even when cure is not possible.

This book summarizes surgical emergencies in the cancer patient. The book is separated into three sections. We begin with a discussion of the special biology and physiology of the cancer patient, particularly one who is undergoing chemotherapy or radiation therapy. Conduct and complications of surgery in patients with coagulopathy, immunosuppression, or wound healing issues related to antineoplastic therapy is summarized. Then, workup, indications for surgery, and basic surgical approach for emergencies related to specific organ systems are summarized in second section. Finally, special considerations in surgical emergencies are presented, including a discussion of infected ports, of emergencies in bone marrow or solid organ transplant patients, and of MIS approaches for surgical intervention. Overall, this is meant as a guide to surgical decision-making and surgical conduct in the important population of cancer patients and cancer survivors.

This book is intended for anyone working in a modern hospital or cancer center. The authorship of this work includes experienced surgical oncologists, medical oncologists, transplantation specialists, anesthesiologists, and interventional radiologists. We thank them for their contributions and this most wonderful collaboration.

We thank our teachers, residents, clinical fellows, and colleagues who have shared their knowledge and experience with us. We thank our patients who inspire us to be superior clinicians, to constantly strive to improve the field. We thank our editor at Springer Daniel Dominguez. Finally, we thank our families and particularly our spouses Nicole, Adam, Tim, Madhuri, and Jennifer for the patience and support they have given us to do our daily investigative and clinical work and then to complete a work such as this.

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Preoperative Evaluation of the Cancer Patient for Emergency Surgery

1

Russell J. Gray and Michael W. Lew

Introduction

In cancer surgical emergencies there are aspects of the disease and treatment that may complicate management and require special attention. Oncologic surgical emergencies may be related to the cancer itself, namely invasion by the tumor (i.e., gut, airway or vascular obstruction, or brain mass lesions), or complications as a result of cancer care, such as chemotherapy, immunotherapy, or radiation (i.e., perforation, infection). Optimal medical care enhanced by effective communication between all care providers, including oncologic, medical, surgical, and anesthesia teams, is hoped to improve patient outcomes and reduce perioperative complications.

The preoperative assessment of the patient with a cancer emergency begins with a review of the medical history, often a challenging task, because patients may be poor historians or mentally compromised by their medical condition or age, since most cancer diagnoses are made in older adults. The evaluation includes past medical, surgical, and anesthetic histories, allergies, drug intolerance and dependency, current medication list, and most

recent intake. Malignant hyperthermia risk should be ruled out. Although NPO status should be known, all emergency cases are usually considered full stomach and at risk for pulmonary aspiration of gastric contents. Imaging and laboratory tests may guide some aspects of management.

Physical examination assesses vital signs, airway, heart, lungs, carotid arteries, bruising, and possible vascular cannulation sites. The risk of transfusion, invasive line placement, difficult airway, as well as postoperative respiratory support should be discussed with the patient whenever possible. Blood products must be available if clinically indicated.

The American Society of Anesthesiologists' Risk Classification

Perioperative risk should be determined and discussed with the patient and the perioperative care team. The American Society of Anesthesiologists' (ASA) Physical Status Classification is the most commonly used physical status assessment tool worldwide. Although it lacks precision and specificity, it is helpful in quantifying the general physiological reserve of patients at the time of assessment [1].

Despite the simplicity of this classification, sometimes it is not easy to assign an accurate ASA class, especially for elderly patients.

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Preoperative impaired cognition, low albumin levels, previous falls, low hematocrit levels, functional dependence, and multiple comorbidities are more closely related to 6-month mortality and post-discharge rehabilitation in the elderly undergoing major surgical procedures. In the “Preoperative Assessment of Cancer in the Elderly” (PACE) study, functional dependency, fatigue, and abnormal performance status were associated with a 50% increase in the relative risk of postoperative complications. Several studies show poor outcomes for elderly patients undergoing emergency noncardiac surgery [2].

The cancer patient may present with significant metabolic, anatomic, and physiologic derangements that require immediate intervention and pose increased risks for emergency surgery. Significant anterior mediastinal masses, pericardial and pleural effusions, cardiac tamponade, and superior vena cava syndrome are common oncologic complications. Such abnormalities may complicate the anesthetic induction. Common hematological abnormalities that may require perioperative management include anemia, thrombocytopenia, or pancytopenia.

Tumor lysis syndrome is common in patients with high-grade non-Hodgkin lymphomas, acute leukemia, and large cancer cell mass. It is usually caused by cytotoxic therapy and results in the release of tumor cellular components into the bloodstream. It is important to know that tumor lysis syndrome can significantly increase the risk for hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia, and azotemia. Additionally, nausea and vomiting, acute kidney injury, seizures, and cardiac arrhythmias may develop.

Preoperative Cardiovascular Risk Assessment

Cardiovascular risk assessment for the probability of major adverse cardiac events (MACE) should be determined prior to surgery. Cardiovascular risk can be assessed using the Revised Cardiac Risk Index (RCRI), or the American College of Surgeons National Surgical

Quality Improvement Program (ACS NSQIP) Surgical Risk Calculator [3].

According to the American College of Cardiology/American Heart Association (ACC/AHA) guidelines, a low-risk procedure is one in which the combined surgical and patient characteristics predict a risk of MACE, (myocardial infarction (MI), heart failure, ventricular fibrillation/tachycardia, stroke, or death) of <1%. Procedures with a risk of MACE of $\geq 1\%$ are considered at elevated risk. The patient undergoing emergency oncologic surgery usually falls in the elevated risk category [3].

The revised cardiac risk index (RCRI), a validated risk assessment tool for major noncardiac surgery, is the most commonly used risk assessment tool among anesthesiologists and perioperative medicine physicians. The RCRI is simple and allows the practitioner to calculate the risk of major adverse cardiac complications for major surgery rapidly. Major cardiac complications are MI, cardiac arrest, heart failure (HF), ventricular fibrillation (VF), pulmonary edema, and complete heart block.

There are six independent predictors of cardiac risk: (1) ischemic heart disease, (2) cerebrovascular disease, (3) diabetes (on insulin therapy), (4) creatinine ≥ 2 mg/dl (GFR <30 ml/min), (5) heart failure, and (6) high-risk surgery. Thus according to the RCRI classification, patients with 0 or 1 predictor(s) of risk would have a low risk of MACE. Patients with two or more predictors would have an elevated risk. The RCRI was validated in nonemergency major noncardiac surgical procedures [4, 5].

The American College of Surgeons recently developed a universal, Web-based, patient-specific risk prediction tool (ACS NSQIP) to calculate procedural specific surgical risk. The surgeon can enter 21 preoperative risk factors (including functional status, disseminated cancer, prior cardiac events, CHF, and emergency surgery) that will predict eight outcomes (morbidity, mortality, pneumonia, cardiac events, surgical site infection, urinary tract infection, venous thrombus embolism, and renal failure). The ACS NSQIP risk calculator offers an opportunity to

improve shared decision making and informed consent [3, 6].

Functional capacity is a reliable predictor of perioperative and long-term adverse cardiac events. The emergency surgical patient usually presents acutely and often chronically debilitated. Knowledge of the patient's baseline functional status gives an estimate of perioperative risk. There is an inverse relationship of functional capacity and MACE. Functional capacity is expressed in terms of metabolic equivalents (METs). Less than four METs is considered poor functional capacity. Girish et al. observed that the inability to climb two flights of stairs was associated with a positive predictive value of 82 % for the development of postoperative complications such as myocardial infarction, heart failure, arrhythmia, pulmonary embolism, pneumonia, and death [7].

Coronary Artery Disease

High-risk surgical procedures and emergency surgery are independent risk factors for patients with known or suspected cardiac disease. Risk factors for MACE include age ≥ 55 , prior coronary events, and cerebrovascular disease. The emergency surgical patient is assessed for the presence of cardiovascular disease. Symptoms of fatigue, dyspnea, or shortness of breath may be the result of multiple etiologies. A history of receiving cardiotoxic chemotherapeutic drugs or radiation therapy to the chest may be the cause of heart failure and ischemic heart disease. A complaint of chest pain may be due to chemoradiation therapy or preexisting coronary artery disease. Atypical chest pain, often due to anxiety, is not uncommon among cancer patients.

Late complications of radiation therapy include pericarditis, accelerated coronary artery disease, restrictive cardiomyopathy, valvular stenosis, and cardiac conduction system defects (arrhythmias). Radiation to the head and neck may cause carotid artery stenosis, increasing the risk of perioperative stroke. Sonny et al. found a lack of association between carotid artery stenosis detected on carotid ultrasound and the inci-

dence of stroke and MI [8]. Independent risk factors for stroke include age ≥ 62 , having an MI within 6 months of surgery, preoperative acute renal failure or dialysis, history of stroke or TIA, hypertension requiring medication, being a current smoker, presenting severe COPD, and having a BMI of 30–40 kg/m² [9].

Emergency noncardiac surgery provides no time for medical optimization of patients with known or suspected coronary artery disease. Myocardial oxygen supply–demand imbalance is the predominant cause of perioperative MI (type 2 PMI), compared to plaque rupture (type 1 PMI). Thus, the goal is to monitor for ischemia, to prevent even modest increases in heart rate, and to avoid hypotension and decreased cardiac output. Studies show that beta-blocker use can reduce perioperative events.

Therefore, the risk and benefits of beta-blocker use should be considered. However, beta-blockers should not be started on the day of surgery. The potential risks include stroke (incidence is far less common than MACE), noncardiac-related death, hypotension, and inability to maintain cardiac output during active bleeding or infection [10]. Long-term beta-blockade should not be discontinued in the absence of significant bradycardia or hypotension. Statin use may improve perioperative outcomes. Patients experiencing a postoperative MI after noncardiac surgery have a hospital mortality rate of 15–25 %, and nonfatal perioperative MI is an independent risk factor for cardiovascular death and for nonfatal MI during the 6 months following surgery [11, 12].

Focusing on an emergency geriatric cohort, Chong et al. observed the incidence of postoperative troponin I elevation (>0.03 ng/ml), 1-year all-cause mortality rates, and cardiac events in an emergency orthopedic geriatric population [13]. The incidence of a troponin I rise postoperatively was 52.9 %. Postoperative acute MI was diagnosed in 9.8 %, and at 1 year, 70 % of these patients were dead. At 1 year, 32.4 % had sustained a cardiac event (MI, CHF, or major arrhythmias). All-cause mortality was 20.6 % at 1 year; 37 % with an associated postoperative troponin rise died versus 2.1 % without a rise. There was a higher incidence of postoperative troponin

I rise in older patients undergoing emergency surgery, which also correlated with an increase in cardiac events and 1-year mortality. Consistent with similar studies, the majority of troponin I rises were asymptomatic. Moreover, postoperative B-type natriuretic peptide (BNP) and troponin I were shown to predict short- to medium-term mortality in patients undergoing emergency gastrointestinal surgery [14].

Coronary stents are commonplace today and represent significant risks perioperatively for stent thrombosis and bleeding. The risk of stent thrombosis is due to the prothrombotic state induced by the surgical stress response and the disruption of dual-antiplatelet therapy. The risk of bleeding is due to the continuation of the antiplatelet therapy. Emergency surgery is associated with a higher incidence of cardiac events than elective procedures. Bare metal stents have a threefold increased risk (12% vs. 4.4%), while drug-eluting stents (DES) have a 3.5-fold increase in risk (18% vs. 4.7%) [15]. New, second-generation DES have reduced rates of thrombosis and MIs, compared to first-generation DES (i.e., eluting everolimus, zotarolimus vs. eluting sirolimus and paclitaxel) [16].

During emergency surgery, the challenge is to minimize the incidence and severity of potential stent thrombosis-induced myocardial ischemia and bleeding by closely monitoring the patient, and ensuring the availability of immediate percutaneous coronary intervention (interventional cardiac catheterization laboratory). Early detection of myocardial ischemia and infarction is essential. Transferring the patient for immediate percutaneous coronary intervention (PCI) within 90 min is critical. Mortality increases 50% with delayed reperfusion (3% at 30 min to 4.3% at 90 min) [15].

Dual-antiplatelet therapy (DAPT) commonly consists of aspirin and clopidogrel (Plavix). The incidence of MACE is inversely related to the time of stent insertion and the timing of surgery. Patients undergoing surgery less than 30 days after stent placement have the greatest risk for adverse cardiac events (Fig. 1.1) [17].

Rates of coronary stent-related complications in patients undergoing noncardiac surgery range from 0.6 to 45%, with a mortality rate of 2.6–4.9%. The mortality rates have been as high as 85% in the report by Sharma (2004) [18]. Current guidelines

by ACC/AHA recommend that noncardiac surgery be delayed until 30 days after bare-metal stent implantation, and 3–6 months after drug eluting stent implantation if delayed surgical risk is higher than the risk of stent thrombosis [16]. After drug-eluting stent implantation, the advice is for at least a 6-month delay for elective noncardiac surgery [16]. Cancer and surgery are associated with an increased inflammatory response and a prothrombotic state. Perioperative risk factors for stent thrombosis and bleeding are listed in Table 1.1, and include discontinuation of DAPT, diabetes, renal failure, and low ejection fraction [15, 16]. Risk factors for bleeding are bleeding history, female sex, low body weight, kidney disease, advanced age, chronic NSAID therapy, diabetes, and chronic steroid therapy [16].

The cardiology consultant may advise in weighing the risk of bleeding against that of thrombosis. Although in most instances clopidogrel cannot be continued, all efforts should be made to continue aspirin. If aspirin is contraindicated or there is a high risk of bleeding from surgical sites, antiplatelet drugs may have to be discontinued. DAPT should be restarted as soon as possible after the surgical procedure. If a platelet transfusion is required, the short half-life of clopidogrel will not interfere with the function of transfused platelets [15, 16].

Heart Failure

Heart failure (HF) is an independent risk factor for perioperative adverse events. Elderly heart failure patients have a substantially higher risk of operative mortality and hospital readmission than other patients, including those with coronary disease, admitted for the same noncardiac procedure [19, 20].

Survival after surgery for patients with a left ventricular ejection fraction (LVEF) less than 29% is significantly worse (59% mortality) than for those with an LVEF greater than 29% (18% mortality) [21]. Early diastolic dysfunction was found to be a strong and independent risk predictor of mortality in cancer patients presenting with septic shock, while the diastolic dysfunction was not associated with exposure to cardiotoxic drugs [22].

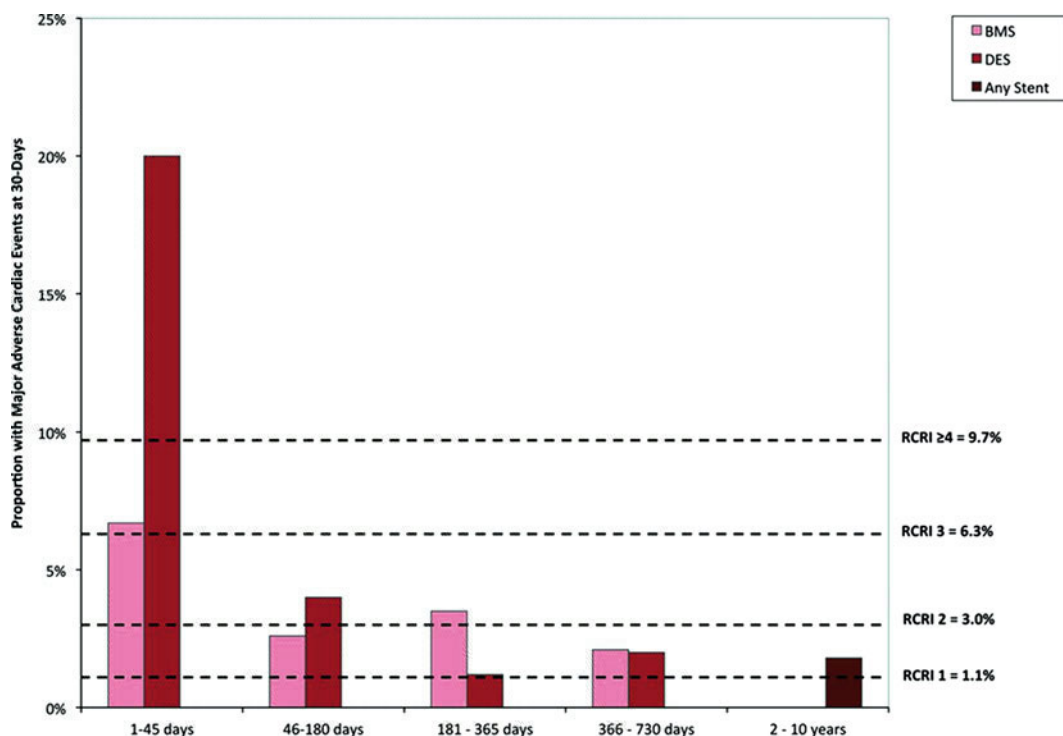


Fig. 1.1 Proportion of patients with major adverse cardiac events (death, readmission for acute coronary syndrome, coronary revascularization) within 30 days after elective noncardiac surgery, based on the interval between the most recent coronary stent insertion and subsequent noncardiac surgery. The *red columns* represent proportions for individuals who received bare metal stents (BMS), drug-eluting stents (DES), or either type of stent (for stent insertions 2–10 years before noncardiac surgery). For comparison,

the *horizontal dashed lines* represent event rates for individuals who did not undergo coronary revascularization within 10 years before noncardiac surgery, as stratified by their Revised Cardiac Risk Index scores [17]. From “Risk of Elective Major Noncardiac Surgery After Coronary Stent Insertion: A Population-Based Study” by D. N. Wijeyesundera et al., 2012, *Circulation*, 126, p1359. Copyright © by the American Heart Association, Inc. Reproduced by permission

Emergency surgery allows limited or no time for medical optimization. Transesophageal echocardiography (TEE), central venous pressure (CVP), pulmonary artery catheter (PAC), or non-invasive cardiac output monitors may guide perioperative management. Heart failure medications should be continued; exceptions include drugs with potential severe adverse effects such as renin-angiotensin system inhibitors and beta-blockers. Intraoperative hypotension should be avoided and treated aggressively. Acute or decompensated heart failure may require diuretic, inotropic, and vasodilator therapy [23]. Temporary use of inotropic therapy (dobutamine, dopamine, norepinephrine, and phosphodiesterase inhibitors) may be necessary to treat hypotension, low perfusion, and cardiac output. Intraoperative fluid

management is a major challenge for the anesthesiologist; goal-directed therapy (GDT) may be useful [23]. A cardiology consultant may be needed to assist in the perioperative management of implanted cardiac devices (ICD).

Valvular Heart Disease

Mild valvular lesions may be well tolerated by patients undergoing surgery, but the risk of perioperative adverse events is increased for patients with significant valvular disease undergoing noncardiac surgery. Patients with known or suspected valvular heart disease should undergo echocardiography to quantify the severity of valvular disease, and determine ventricular function, and

Table 1.1 Perioperative risk factors for stent thrombosis [15]

Acute coronary syndrome
Congestive heart failure
Diabetes
Renal impairment
Coronary anatomy (total stent length, multiple stents, bifurcation-lesion)
Advanced age
Prior brachytherapy
Cancer
Elective surgery
Emergency surgery

From “Coronary stents: factors contributing to perioperative major adverse cardiovascular events” by P. Barash and S. Akhtar, 2010, Br. J. Anaesth., 105, page i5. Copyright © 2010 by P. Barash and S. Akhtar. Reproduced with permission

atrial and ventricular pressures. The perioperative risk of emergency noncardiac surgery can be minimized by avoiding fluid overload, choosing the appropriate anesthetic, and using appropriate intraoperative monitoring (arterial line, TEE, or PAC).

Aortic Stenosis

Aortic stenosis (AS) is present in 1–2 % of all patients greater than 65 years of age and in 3–8 % of all patients greater than 75 years of age [24, 25]. The estimated rate of cardiac complications in patients with undiagnosed severe AS undergoing noncardiac surgery is 10–30 % [25]. Symptomatic and severe AS patients undergoing emergency noncardiac surgery are at a significant risk for major adverse cardiac events and mortality.

The murmur of aortic stenosis is a harsh, crescendo-decrescendo murmur, heard loudest at the right upper sternal border and radiating to the carotid arteries. Peripheral pulses have a significantly delayed upstroke and diminished intensity (pulsus parvus tardus). With severe AS, the aortic valve area is less than 1 cm²; the mean transvalvular pressure gradient is >50 mmHg, and peak blood velocity >4 m/s. An aortic valve gradient ≥40 mmHg is significant for major adverse cardiac events.

Table 1.2 Rates of 30-day MACE and mortality in AS patients and controls stratified by emergency vs. elective surgery [26]

	AS patients	Controls	P for difference
MACE			
Emergency surgery	163/1051 (15.5 %)	120/1051 (11.4 %)	0.006
Elective surgery	66/1772 (3.7 %)	52/1772 (2.9 %)	0.19
Mortality			
Emergency surgery	225/1051 (21.4 %)	179/1051 (17.0 %)	0.01
Elective surgery	67/1772 (3.8 %)	51/1772 (2.9 %)	0.13

Abbreviations: AS aortic stenosis, MACE major adverse cardiovascular event

From “Noncardiac surgery in patients with aortic stenosis: a contemporary study on outcomes in a matched sample from the Danish health care system” by C. Andersson et al., 2014, Clin. Cardiol., 37, p 682. Copyright © 2014 by Wiley Periodicals, Inc. Reproduced by permission

Hemodynamic instability (i.e., hypotension, tachycardia) as a result of anesthetics and surgical stress decreases coronary perfusion, causing arrhythmias, myocardial ischemia, heart failure, and death. In a Dutch study, emergency surgery patients with aortic stenosis had higher event rates than elective surgery patients, and symptomatic patients had higher event rates than asymptomatic patients (Fig. 1.2 and Table 1.2) [26]. Management goals should be to avoid tachycardia; maintain sinus rhythm, a normal preload, and left atrial pressure; and avoid hypotension. Phenylephrine or norepinephrine can be used to treat hypotension [21, 24].

Mitral Stenosis

Mitral stenosis (MS) is more common in women [27]. Patients may present with fatigue, palpitations, hemoptysis, or atrial fibrillation. Patients usually are not symptomatic until the mitral valve area falls below 2.5 cm². Exertional dyspnea is the most common symptom when the mitral valve area is less than 1.5 cm². Patients should be assessed for pulmonary hypertension, pulmonary edema, and right heart failure. Patients

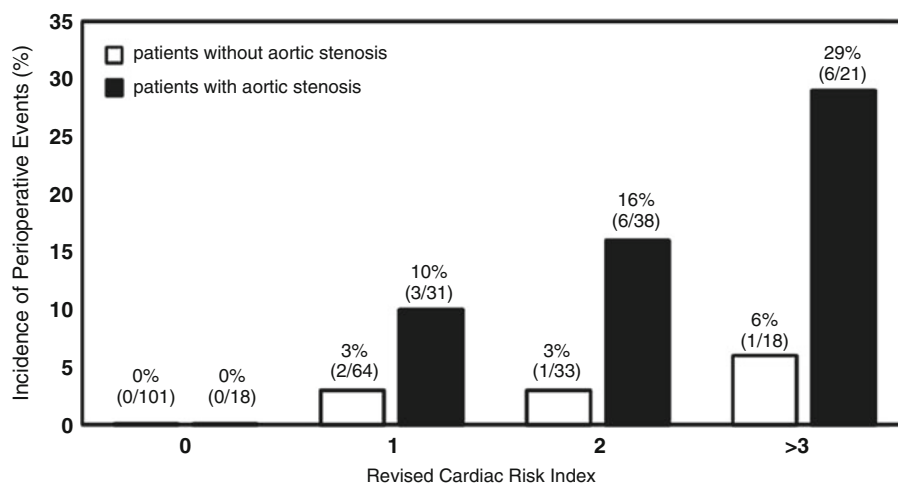


Fig. 1.2 Incidence of perioperative mortality and nonfatal myocardial infarction in patients with aortic stenosis compared to control. Results are based on the absence or presence of aortic valve stenosis, and on Revised Cardiac Risk Index (RCRI). RCRI, 1 point for each of the following: high-risk surgery, ischemic heart disease, history of heart failure, history of cerebrovascular disease, insulin-

dependent diabetes, preoperative creatinine >2.0 mg/dl [25]. From “Aortic stenosis: an underestimated risk factor for perioperative complications in patients undergoing noncardiac surgery” by M. D. Kertai et al., 2004, *Am. J. Med.*, 116, p 12. Copyright © 2004 by Exerta Medica. Adapted with permission

with severe mitral stenosis having emergency noncardiac surgery are at increased risk for adverse events and should be managed similar to patients with AS (monitor intravascular volume and avoid tachycardia and hypotension) [21].

Aortic Regurgitation and Mitral Regurgitation

Acute aortic regurgitation (AR) and acute mitral (MR) regurgitation represent cardiac surgical emergencies beyond the scope of this chapter. Clinical characteristics of aortic regurgitation include a high-pitch decrescendo diastolic murmur, loudest at the right upper sternal border, systolic hypertension, and a wide pulse pressure. The chest X-ray may reveal a large dilated left ventricle. Patients with mitral regurgitation may present with complaints of shortness of breath, palpitations (as a result of atrial fibrillation), poor functional status, and cachexia. The murmur of mitral regurgitation is best auscultated at the cardiac apex, and is a holosystolic murmur.

In the absence of decompensated left ventricular function, patients with chronic AR, MR, and tricuspid regurgitation (TR) are able to tolerate noncardiac surgery. The goals are to maintain

adequate intravascular volume, to avoid fluid overload, and to avoid increased afterload (Table 1.3) [28].

Perioperative Fluid Management

Fluid and electrolyte disorders are often present in the cancer patient undergoing emergency surgery. The anesthesiologist should be prepared to initiate or continue fluid resuscitation and correct electrolyte, and acid-base abnormalities. Sepsis, dehydration, anemia, coagulopathies, and acute bleeding are often present and require immediate perioperative management. The goal of fluid management is to maintain effective circulating volume, adequate tissue perfusion and oxygenation. The anesthesiologist must decide the appropriate amount and type of intravenous fluids to administer, based on the surgical procedure and the clinical status of the patient (sepsis, dehydration or fluid overload, and comorbidities). The administration of crystalloids versus colloids remains controversial. Fluid management may affect postoperative morbidity, mortality, and hospital length of stay.

Table 1.3 Hemodynamic goals in patients with valvular disease [28]

	HR	Contractility	Preload	Afterload	Concerns	Drugs
AS	n/↓	n/↑	↑	↑	Maintain SR	Phenylephrine
					Spinal anesthesia relatively contraindicated	Norepinephrine
					Avoid too low HR (fixed CO)	
					Immediate defibrillation if VT/VF (CPR ineffective)	
AI	↑	n/↑	↑	↓		Ephedrine
						Epinephrine
MS	↓	n	n/↑	↑	Maintain SR	Phenylephrine
					If other than SR control HR	Norepinephrine
					Avoid precipitators of PHT	Epinephrine
MR	↑	n/↑	↑	↓	Often underlying cardiac dysfunction (not apparent from EF)	Ephedrine
						Epinephrine
						Norepinephrine
HCM	↓	↓	↑	↑	Avoid increase in contractility	β-Blocker
					Avoid β-agonists	Phenylephrine
						Norepinephrine

HR heart rate, AS aortic stenosis, SR sinus rhythm, CO cardiac output, VT ventricular tachycardia, VF ventricular fibrillation, CPR cardiopulmonary resuscitation, AI aortic insufficiency, MS mitral stenosis, PHT pulmonary hypertension, MR mitral regurgitation, EF ejection fraction, HCM hypertrophic cardiomyopathy

From “Anesthetic considerations in the patient with valvular heart disease undergoing noncardiac surgery” by A. J. Mittnacht et al., 2008, Semin. Cardiothorac. Vasc. Anesth., 12, p 40. Copyright © 2008 by Sage Publications. Reproduced with permission

The avoidance of the detrimental effects of organ hypoperfusion, excessive intravascular volume, edema, electrolyte abnormalities, and acute kidney injury (synthetic starches) will reduce complications. There are two approaches to perioperative fluid therapy: (1) the “traditional formula approach,” estimating fluid requirements based on weight, type of surgery, and the nature of the fluid loss, or (2) Goal Directed Therapy (“GDT,”) which is a direct measurement of physiological variables, such as cardiac output, systemic vascular resistance, and tissue oxygen content.

The traditional approach (recipe/cookbook approach) of fluid administration is based on a predetermined rate of intravenous infusion along with replacing observed intraoperative losses, which is often inaccurate and erratic. Several studies show harm with this approach since it may fail to account for preoperative loss or replacements, and cardiovascular status or disease (i.e., heart failure, CAD, sepsis). With large fluid shifts and surgical insults, it becomes

difficult to determine the correct amount of fluid to infuse [29, 30].

GDT is based on measuring physiologic variables related to cardiac output or tissue O₂ delivery, and the administration of fluids, red blood cells, and possibly inotropic and vasodilator therapy as needed (fluid responsiveness) to improve tissue perfusion and ultimately patient outcome. Inadequate fluid administration can lead to a reduced effective circulating volume, by the diversion of blood toward vital organs such as the brain and the heart, and away from non-vital organs (gut, kidney, liver, and skin), and can result in tissue hypoperfusion and hypoxia (Fig. 1.3 and Table 1.4) [31].

Excess fluid in the intravascular compartment leads to increased pressure in the venous circulation and results in loss of fluid from the intravascular space into the interstitial space. Furthermore, this leads to peripheral and pulmonary edema, compromised systemic and/or local tissue oxygenation, and to multi-organ

Fig. 1.3 The classic relationship between perioperative volume status and perioperative complications. The relationship describes a “U” shape with an increased risk of complication for both perioperative hypovolemia and perioperative hypervolemia, emphasizing the importance of perioperative fluid optimization [31]. From “Guiding Goal-Directed Therapy” by K. Suehiro et al., 2014, *Curr. Anesthesiol. Rep.*, 4, p361. Copyright © 2014 by Springer. Reproduced by permission

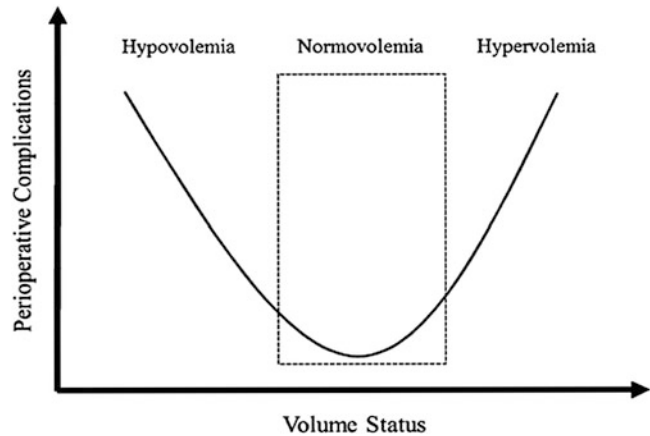


Table 1.4 Complications associated with hypervolemia and hypovolemia [31]

Complications of hypervolemia	Complications of hypovolemia
Increases venous pressure resulting in loss of fluid from the intravascular to interstitial space which can lead to pulmonary and peripheral edema impairing tissue oxygenation	Reduces effective blood circulatory volume resulting in diversion of blood flow from non-vital organs (skin, gut, kidneys) to vital organs (heart and brain)
Increases demand on cardiac function	Activates the sympathetic nervous and renin-angiotensin system
Decreases tissue oxygenation with delayed wound healing	Increases inflammatory response
May cause coagulation disturbances through hemodilution	May also lead to vasopressor agent administration which may increase hypoperfusion and ischemia ^a
Is associated with increased daily fluid balance and mortality. ^b Chappell et al. ^c also demonstrate a relationship between weight gain related to excessive fluid administration and mortality	

From “Guiding Goal-Directed Therapy” by K. Suehiro et al., 2014, *Curr. Anesthesiol. Rep.*, 4, p 362. Copyright © 2014 by Springer. Reproduced by permission

^aMurakawa K, Kobayashi A. Effects of vasopressors on renal tissue gas tensions during hemorrhagic shock in dogs. *Crit Care Med.* 1988; 16:789–92

^bChappell D, Jacob M, Hofmann-Kiefer K, Conzen P, Rehm M. A rational approach to perioperative fluid management. *Anesthesiology.* 2008; 109:723–40

^cRosenberg AL, Dechert RE, Park PK, Bartlett RH. Review of a large clinical series: association of cumulative fluid balance on outcome in acute lung injury: a retrospective review of the ARDSnet tidal volume study cohort. *J Intensive Care Med.* 2009; 24:35–46

dysfunction (Table 1.4) [29, 32]. The correlation of pulmonary artery occlusion pressure and central venous pressure as a reliable measure of left and right ventricular preload has been rejected in recent studies (Fig. 1.4). Neither pulmonary artery occlusion pressure nor central venous pressure appears to be a useful predictor of ventricular preload or stroke volume with respect to optimizing cardiac performance (Fig. 1.5) [32].

Current evidence demonstrates that CVP or PAC should not be used to make clinical decisions regarding fluid management [33]. It has been demonstrated that noninvasive or minimally invasive monitoring can be used to predict fluid responsiveness in a highly predictable and accurate way [34]. These monitors include esophageal Doppler, bioreactance/bioimpedance, and pressure waveform analysis, which use an arterial catheter or a finger probe [35].

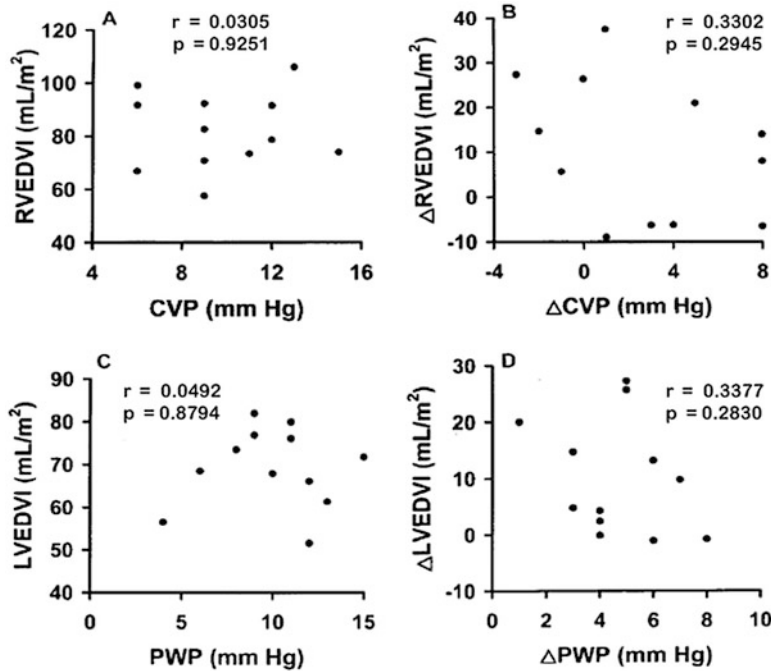


Fig. 1.4 Relationship between **a**, initial central venous pressure (CVP) and right ventricular end-diastolic volume index (RVEDVI); **b**) changes in central venous pressure and RVEDVI in response to saline; **c**) initial pulmonary artery occlusion pressure (PWP) and left ventricular end-diastolic volume index (LVEDVI); and **d**) changes in PWP and LVEDVI in response to saline in group 1 subjects. No significant relationship was found between initial values for central venous pressure and RVEDVI or

changes in these variables following 3 l of saline infusion. Similar negative results were found for the relationship between PWP and LVEDVI [32]. From “Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects” by A. Kumar et al., 2004, *Crit. Care Med.*, 32, p 694–5. Copyright © 2004 by Lippincott Williams & Wilkins. Reproduced by permission

GDT can be applied to perioperative fluid management on a patient-specific basis and has gained widespread acceptance in clinical practice, in spite of conflicting data. Several studies suggest that GDT can improve postoperative outcomes with lower complication and mortality rates, shorter hospital length of stay, and lower cost of surgery (Fig. 1.6) [31, 36].

The overall conclusion is that GDT of some type is probably beneficial for high-risk patients and has few documented adverse effects.

Transfusions

Anemia is not uncommon in cancer patients requiring emergency surgery. Anemia can contribute to increased morbidity and mortality in

patients unable to tolerate increased oxygen demands (frailty, sepsis, coronary artery disease, heart failure). The American Association of Blood Banks has established clinical practice guidelines for transfusions. Current guidelines recommend not transfusing asymptomatic hemodynamically stable patients without CAD at hemoglobin >7 – 8 g/dl. In postoperative patients with heart disease the recommendation is to maintain the hemoglobin ≥ 8 g/dl.

Transfusion requirements in surgical oncologic patients have been examined. In a randomized controlled trial in an oncologic ICU, restrictive (hemoglobin <7 g/dl) and liberal (hemoglobin <9 g/dl) transfusion strategies were compared for reducing mortality and severe clinical complications among patients having major cancer surgery. The liberal transfusion strategy with a hemoglobin

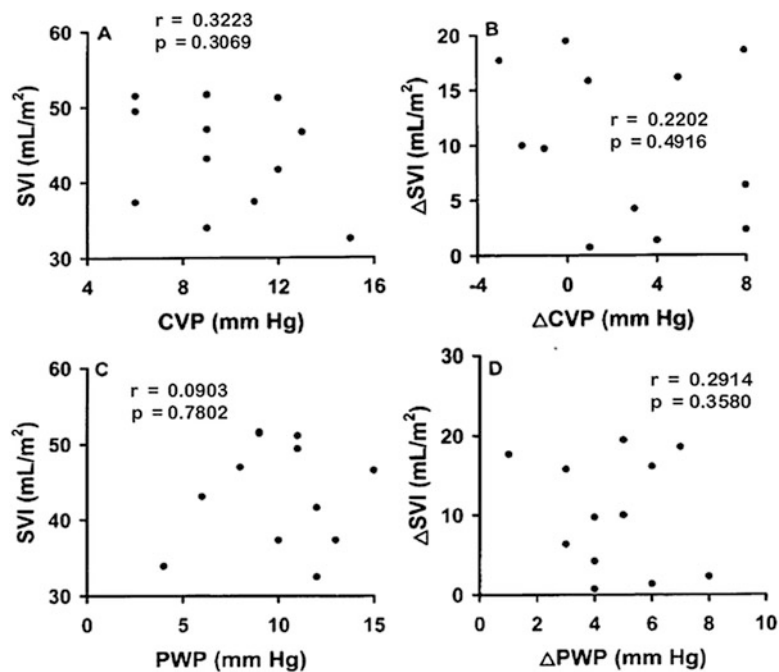
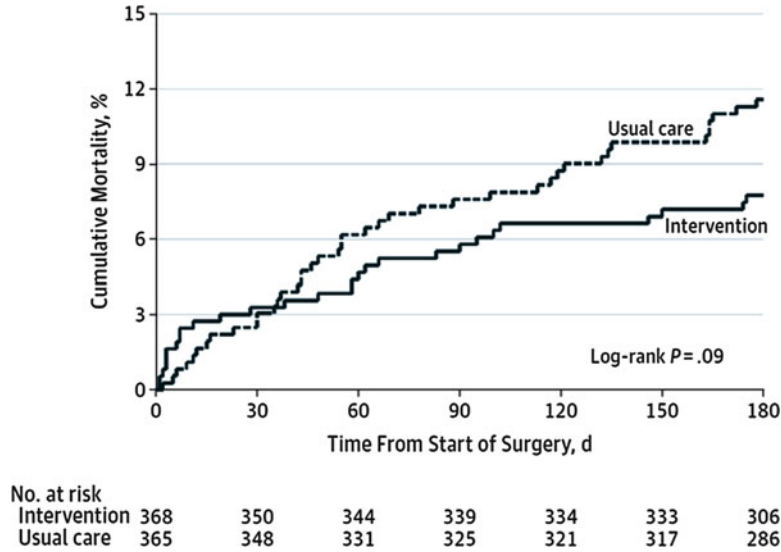


Fig. 1.5 Relationship between **a**, initial central venous pressure (CVP) and stroke volume index (SVI); **(b)** changes in central venous pressure and SVI in response to saline; **(c)** initial pulmonary artery occlusion pressure (PWP) and SVI; and **(d)** changes in PWP and SVI in response to saline in group 1 subjects. No significant relationship was found between initial values for either central venous pressure or

PWP and SVI or changes in these variables following 3 l of saline infusion [32]. From “Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance, or the response to volume infusion in normal subjects” by A. Kumar et al., 2004, *Crit. Care Med.*, 32, p694-5. Copyright © 2004 by Lippincott Williams & Wilkins. Reproduced by permission

Fig. 1.6 Cumulative incidence of mortality up to 180 days after surgery using a cardiac output-guided hemodynamic therapy algorithm intervention vs. usual care [36]. From “Effect of a perioperative, cardiac output-guided hemodynamic therapy algorithm on outcomes following major gastrointestinal surgery: A randomized clinical trial and systematic review” by R. M. Pearse, et al., 2014, *JAMA*, 311, p2187. Copyright © 2014 American Medical Association. Reproduced with permission



trigger of 9 g/dl was associated with fewer major postoperative complications than the restrictive strategy with a hemoglobin trigger of 7 g/dl (19.6% vs. 35.6%) [37, 38].

Sepsis

The septic patient undergoing emergency surgery for source control is common among cancer patients. The anesthesiologist is frequently involved in the management of septic or septic shock patients prior to taking the patient to the operating room. Resuscitation during the first 6 h of recognition of sepsis was associated with a 15.9% absolute reduction in 28-day mortality. Obtaining adequate intravenous and arterial cannulation is an immediate priority, while peripheral access is achievable [39, 40].

The history, physical examination, and laboratory values should give clues about the severity of the septic state. Vital signs, such as the blood pressure, heart rate, respiratory rate, oxygen saturation, warm perfused skin versus cold and mottled, and mental status, will give clues about the volume and perfusion status.

Ventilation, oxygenation, and acid–base status can be assessed by arterial blood gases, and lactate levels. Renal function and perfusion can be assessed by blood urea nitrogen, creatinine, and pH. Among critically ill cancer patients, the rate of acute kidney injury (AKI) is between 12 and 49%. The most common cause of AKI in patients with critical illness is sepsis, especially in patients older than 60, or with uncontrolled cancer and poor performance status. In the setting of multi-organ dysfunction, mortality increases with the number of affected organs [41].

Initial laboratory studies should also include a CBC, liver enzymes, coagulation panel, and mixed venous oxygenation (SvO₂). The CXR and ECG should be reviewed to assess for ARDS, pulmonary edema, myocardial ischemia, and arrhythmias. The insertion of a central venous catheter should be considered for the purpose of fluid resuscitation, vasopressor and inotropic therapy, central venous oxyhemoglobin saturation (ScvO₂), and central venous pressure monitoring.

1. Initial resuscitation during the first 6 h of sepsis-induced hypoperfusion should include maintaining a CVP between 8 and 12 mmHg, MAP $\geq$ 65 mmHg, urine output $\geq$ 0.5 ml/kg/h, superior vena cava oxygenation saturation (ScvO₂), or mixed venous oxygen saturation (SvO₂) 70% or 65%, respectively. Figure 1.7 gives an example of using ScvO₂ as a guide for GDT end points. ScvO₂ data can be collected via CVP line insertion which has less risk compared to PAC [31].
2. Next, targeting resuscitation to normalize lactate in patients with elevated lactate levels as a marker of tissue hypoperfusion (Fig. 1.7) [31].

Guidelines for fluid administration have been established by the Society of Critical Care Medicine (Surviving Sepsis Campaign) [42]. Since its inception, mortalities associated with the sepsis spectrum have decreased [42].

Preoperative Pulmonary Evaluation

Unlike cardiovascular preoperative assessment, organized algorithms and guidelines for pulmonary risk stratification are lacking. Major pulmonary risk factors include chronic obstructive pulmonary disease, obstructive sleep apnea (OSA), obesity hypoventilation syndrome (OHS), and pulmonary hypertension.

OSA challenges the anesthesiologist in many ways. Repetitive chronic airway obstruction during sleep causes hypoxemia, pulmonary hypertension, cor pulmonale, and fatal cardiac dysrhythmias. Vastly prevalent but greatly undiagnosed, OSA can be suspected by history and physical examination, where the STOP-BANG Questionnaire has shown good predictive value [43]. High grade of suspicion of OSA would dictate a higher degree of postoperative monitoring.

OHS is characterized by daytime hypercapnia (PaCO₂ >45 mmHg), sleep-disordered breathing, and obesity (BMI >30 kg/m²) [44]. Ninety percent of patients with OHS have OSA. The perioperative management of patients with OHS is similar to patients with OSA; but OHS patients

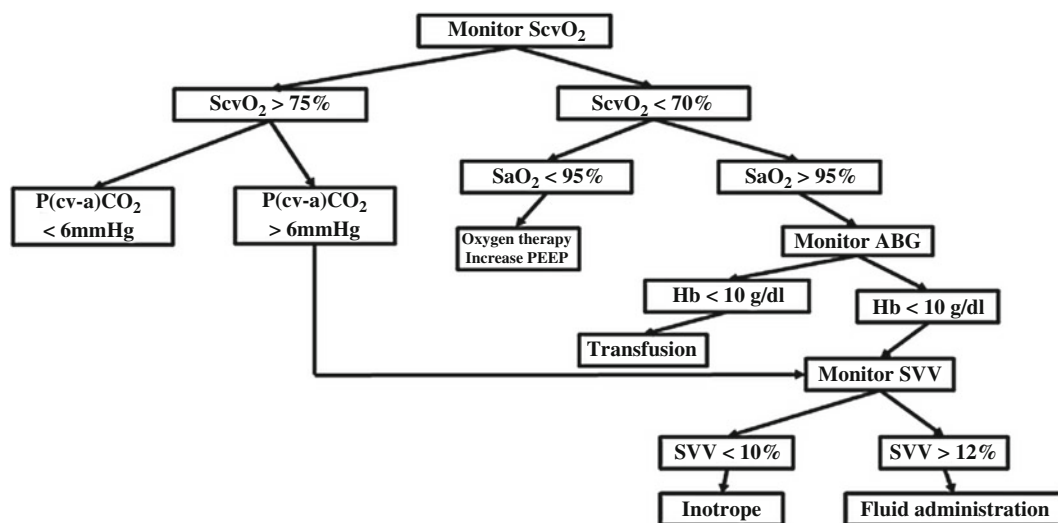


Fig. 1.7 $ScvO_2$ central venous oxygen saturation, SaO_2 arterial oxygen saturation, $PEEP$ positive end-expiratory pressure, ABG arterial blood gas, Hb hemoglobin, SVV stroke volume variation. The example protocol using $ScvO_2$ adapted from the report by Vallet et al. (Tissue oxygenation parameters to guide fluid therapy. *Transfus.*

Altern. Transfus. Med. 2010; 11:113–7). In this protocol, the goal for $ScvO_2$ is to be maintained above 75 % [31]. From “Guiding Goal-Directed Therapy” by K. Suehiro et al., 2014, *Curr. Anesthesiol. Rep.*, 4, p 371. Copyright © 2014 by Springer. Reproduced by permission

have a smaller reserve which increases their acuity and risk. Their respiratory drive is less reactive, and they have a high risk of respiratory failure postoperatively. Similar to OSA, OHS often goes undiagnosed until an untoward outcome occurs. Because of chronic hypoxemia, the development of pulmonary hypertension is increased with progression to right ventricle failure and ultimately cor pulmonale.

Pulmonary hypertension (mean arterial pressure at rest >25 mmHg) can be precipitated and exacerbated by acidosis, hypoxemia, hypercapnia, lung injury, and positive pressure ventilation. Air, bone marrow, or cement embolism also results in increases in pulmonary pressure. Increasing pulmonary artery pressure leads to right ventricular failure and cardiogenic shock; diastolic perfusion decreases, worsening right-side function and decreasing cardiac output. Pulmonary hypertension predisposes patients to a higher incidence of congestive heart failure, hemodynamic instability, sepsis, increased ICU stay, prolonged mechanical ventilation, and respiratory failure. Increased mean arterial pulmonary pressure was a risk factor for postoperative

mortality with a rate of 3.5 % and mortality for emergency surgery at 15 % [45].

Assessing the severity and etiology of the pulmonary hypertension is recommended. Perioperative PA catheter monitoring has the advantage of guiding vasodilator therapy.

Data thus obtained may serve as a factor in considering the necessity and urgency of the surgical procedure and evaluate the morbidity and mortality risk to the patients. Pulmonary vascular dilators such as inhaled nitric oxide, prostacyclin, or sildenafil have been used to decrease pulmonary pressures [46]. The avoidance of hypercarbia, hypoxia, hypervolemia, hypothermia, and acidosis is important in anesthesia.

The preoperative evaluation of the patient undergoing noncardiac thoracic surgery has traditionally been based on assessing a patient's preoperative pulmonary status with pulmonary function testing. Recently, functional status in the form of a fragility index has been proposed to further elucidate preoperative morbidity and mortality in the elderly population [47]. In an NSQIP study, over 6300 patients undergoing thoracic surgery were evaluated and assessed

regarding being independent (does not require assistance), partially dependent (some assistance for ADL), and totally dependent (require total assistance). The results demonstrated that non-independent patients had an increased incidence of postoperative infection, failure to wean from mechanical ventilation, and prolonged intubation, and were eight times more likely to die [48].

Airway Management

Respiratory distress in cancer patients can arise from various etiologies: the cancer itself, surgery, radiation, chemotherapy, sepsis, and/or aspiration. It is the most common admission into the ICU in cancer patients and up to 76 % of all cancer patients who are mechanically ventilated have fatal outcomes [49]. Airway obstruction can present either as an acute or a gradual process. However, it is the acute airway obstruction that must be addressed without delay. Failure to promptly and strategically manage the airway could result in brain death or cardiac arrest.

Complicating airway management is the fact that obesity and OSA have become increasingly prevalent over the years. These two comorbid conditions correlate with difficult laryngoscopy, difficult intubation, difficult mask ventilation, and increased risk of aspiration [50]. Aspiration is the leading cause of anesthesia-related death, so identifying and minimizing the risk is essential. Patients at risk for aspiration include conditions that delay gastric emptying, recent oral intake, chronic opioid use, and obesity. Not only does the obese patient pose an aspiration risk, but also is an airway management risk because of altered anatomy such as decreased neck mobility, relatively large tongue, edematous pharyngeal mucosa, poor mask fit ventilation, and a decreased functional residual capacity which predisposes to oxygen desaturation.

Airway management in the oncologic emergency setting is one of the most challenging tasks for the anesthesiologist, because in most instances, oncologic airway emergencies are synonymous with a difficult airway. The American Society of Anesthesiologists task force devel-

oped the Difficult Airway Algorithm (DAA) to systematically manage the difficult airway (Fig. 1.8) [51]. The ultimate success of the DAA depend upon the anesthesiologist's clinical judgment and expertise in the appropriate use of airway devices and techniques.

Airway obstruction can be classified either as upper or lower airway obstruction. In the cancer patient, the most common causes of upper airway obstruction are an enlarging mass (i.e., tumor or hematoma) or chemotherapy/radiation-induced changes (i.e., mucositis). Noninfectious causes of airway obstruction include airway edema, severe tracheomalacia, and tracheal stenosis.

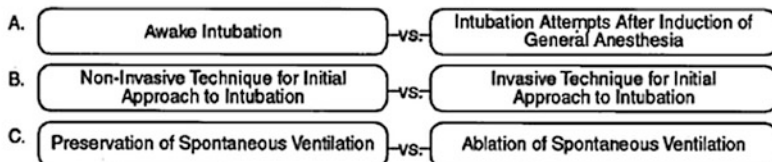
The early clinical presentation of upper airway obstruction includes restlessness and exertional dyspnea. Late findings include wheezing, orthopnea, tachycardia, diaphoresis, sternal retraction, and stridor. In severe cases, bradycardia, cyanosis, obtundation, and death may ensue within minutes of initial presentation. In the event of an acute upper airway obstruction the airway is best secured with an endotracheal tube while maintaining spontaneous ventilation either by utilizing topical anesthetic or an inhalational anesthetic. Neuromuscular tone in the spontaneous breathing patient is the pillar that prevents complete airway collapse; therefore, this tone should not be abolished by the administration of a neuromuscular blocking agent.

Hemoptysis is another cause of airway obstruction with neoplasm representing 7–19 % of these cases. Bronchoscopy is the single most important technique for determining the cause and location of the bleeding. When severe bleeding or hemodynamic instability exists the patient should be transferred to the operating room where flexible or rigid bronchoscopy can be performed in a controlled environment. General anesthesia is the most commonly employed anesthetic technique. If bleeding cannot be controlled, the bleeding side can be isolated with lung separation utilizing a double-lumen tube or bronchial blocker.

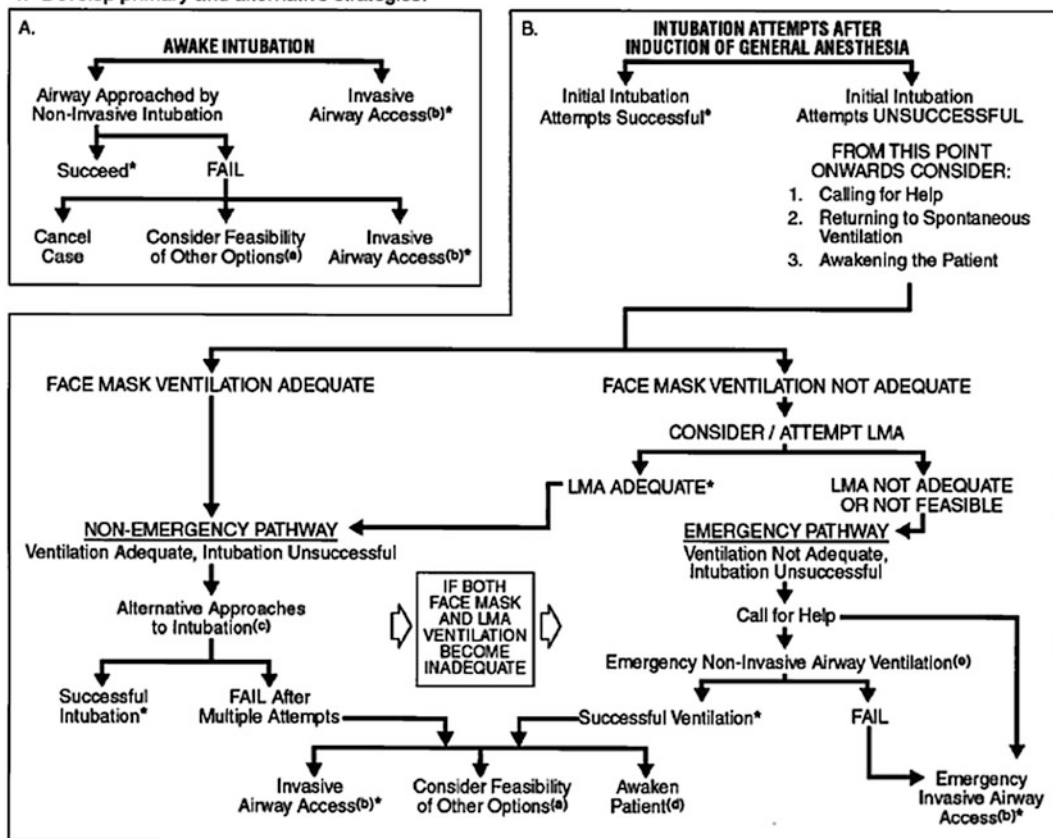
In the emergent oncologic surgical patient, using an endotracheal tube to secure and manage the airway occurs in the vast majority of patients. If the intubation isn't successful, the use of a

DIFFICULT AIRWAY ALGORITHM

1. Assess the likelihood and clinical impact of basic management problems:
 - A. Difficult Ventilation
 - B. Difficult Intubation
 - C. Difficulty with Patient Cooperation or Consent
 - D. Difficult Tracheostomy
2. Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management
3. Consider the relative merits and feasibility of basic management choices:



4. Develop primary and alternative strategies:



* Confirm ventilation, tracheal intubation, or LMA placement with exhaled CO₂

a. Other options include (but are not limited to): surgery utilizing face mask or LMA anesthesia, local anesthesia infiltration or regional nerve blockade. Pursuit of these options usually implies that mask ventilation will not be problematic. Therefore, these options may be of limited value if this step in the algorithm has been reached via the Emergency Pathway.

b. Invasive airway access includes surgical or percutaneous tracheostomy or cricothyrotomy.

c. Alternative non-invasive approaches to difficult intubation include (but are not limited to): use of different laryngoscope blades, LMA as an intubation conduit (with or without fiberoptic guidance), fiberoptic intubation, intubating stylet or tube changer, light wand, retrograde intubation, and blind oral or nasal intubation.

d. Consider re-preparation of the patient for awake intubation or canceling surgery.

e. Options for emergency non-invasive airway ventilation include (but are not limited to): rigid bronchoscope, esophageal-tracheal combitube ventilation, or transtracheal jet ventilation.

Fig. 1.8 Difficult airway algorithm [51]. From "Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway" by the

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