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Anesthesia for Patients with Acute Heart Failure Syndromes

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This chapter provides a conceptual framework for anesthesia for cardiac surgery, and discusses selected practical issues concerning anesthesia for patients with acute heart failure syndromes (AHFSs). We first develop the conceptual framework by selectively presenting recent knowledge (including definitions and pathophysiology) relevant for anesthesia and postanesthesia care of patients with AHFS. We then discuss diagnosis, treatment decisions, and procedures. Finally, a case report is presented.

Conceptual Framework

The definitions of acute heart failure syndromes (AHFSs) used in this chapter are those recently published by the European Society of Cardiology (ESC) guidelines (1): acute decompensated heart failure (de novo or as decompensated congestive heart failure [CHF]), hypertensive acute heart failure, pulmonary edema, cardiogenic shock, and high output failure (1). Cardiogenic shock is defined in the ESC guidelines as a syndrome characterized by evidence of tissue hypoperfusion induced by heart failure after correction of preload (1). This definition constitutes a change from previous criteria that were mainly based on values of cardiac index and arterial blood pressure (see below).

The causes of AHFS have been detailed in the ESC guidelines (1). The search for a cause should be done rapidly. When a correctable causes (surgical or nonsurgical) is not found, therapy should be aimed at correction of the precipitating factor(s) and symptomatic therapy.

A frequent cause of cardiogenic shock is acute myocardial infarction (AMI). In this context, the mechanism of cardiogenic shock is related mainly to isolated left ventricular systolic dysfunction (79% of cases), but also to isolated right ventricular systolic dysfunction (2.8%), severe mitral regurgitation (6.8%), ventricular septal rupture (3.9%), and tamponade (1.4%) (2). Thus, the mechanism of cardiogenic shock is not isolated left ventricular systolic dysfunction in 20% of patients presenting with AMI. Hemodynamic signs are not helpful for recognizing these 20% of cases, nor is the presence or absence of pulmonary congestion. Understanding the mechanisms of cardiac dysfunction in patients with AHFS is necessary in order to provide adequate therapy but also in order to anticipate, prevent, and correct the potential effects of anesthetic drugs.

New Pathophysiologic Paradigms and Therapeutic Approaches

There are two new pathophysiologic paradigms that are relevant for this chapter. The first is the paradigm of a therapeutic window for all shock states; the second is the interaction between cardiogenic shock and the inflammatory response.

Therapeutic Window of Shock States

Whatever the initial cause of shock, its persistence for prolonged periods of time leads to vital organ dysfunction that will evolve by itself to multiple organ failure even if the initial cause of shock is successfully corrected. The duration of

"prolonged" depends on the type of shock. It is minutes for anaphylactic shock (3), and a few hours for hemorrhagic, cardiogenic, and septic shock. Therefore, there is a therapeutic window for each type of shock when adequate therapy decreases the probability of subsequent occurrence of multiple organ failure. Compliance with the therapeutic window paradigm has been shown to decrease mortality in patients with severe sepsis (4). All shock states therefore represent an acceleration of the symptom/sign-diagnosis-therapy cycle.

Interaction Between Cardiogenic Shock and the Inflammatory Response

Cardiogenic shock was classically defined by the presence of arterial hypotension (systolic blood pressure <90 mmHg for at least 30 minutes or the need for supportive pharmacologic or mechanical measures to maintain a systolic blood pressure >90 mmHg) and end-organ hypoperfusion (e.g., cool extremities or a urine output of <30 mL/h). The hemodynamic criteria were a cardiac index (CI) of no more than 2.2 L/min/m² and a pulmonary capillary wedge pressure (PCWP) of at least 15 mmHg.

This canonical clinical and hemodynamic presentation can be complicated by clinical and biologic signs that usually define systemic inflammatory response syndrome SIRS (e.g., fever >38.5°C, leukocytosis). In a recently published substudy of the SHOCK (SHould we emergently revascularize Occluded Coronaries for cardiogenic shock) trial, it was shown that of the 302 enrolled patients, 59 (20%) presented with signs of SIRS (out of which two had signs of SIRS before the onset of cardiogenic shock and three that could not be properly evaluated) (5). Of the remaining 54 patients, 40 had documented infection by positive cultures (mostly blood cultures) and 14 had negative bacterial cultures (5). Median (interquartile range [IQR]) systemic vascular resistance values expressed as dyne/s/cm⁵ were significantly ($p = 0.006$) lower for patients with cardiogenic shock and SIRS [i.e., 1051 (862–1486), $n = 31$ for patients with SIRS and positive bacterial cultures, and 1174 (705–1370), $n = 11$ for patients with SIRS and negative bacterial cultures] as compared to patients with cardiogenic shock without

SIRS [1402 (1088–1807), $n = 168$]. Duration of stay in the intensive care unit (ICU) and in the hospital was significantly longer for the patients with SIRS. The mortality of patients with SIRS and positive bacterial cultures, after adjustment for age and use of coronary artery bypass grafting, was significantly higher than for controls (no SIRS) (odds ratio 2.2; 95% confidence interval [CI] 1.32–3.76; $p = 0.008$) (5). Three parameters predicted the occurrence of positive bacterial cultures in patients with cardiogenic shock: younger age, the use of CABG, and lower initial systemic vascular resistance (SVR) (5). For each decrease in SVR of 200 dyne/s/cm⁵ upon the initial hemodynamic evaluation, the odds ratio of subsequent culture-positive SIRS was 1.21 (95% CI, 1.04–1.40; $p < 0.05$) (5). The unifying hypothesis proposed by the authors is that in patients with SIRS, low SVR predisposes to endothelial damage and a leakage syndrome in which normal barriers against infection are disrupted (5).

This study confirmed previous reports and demonstrates that in up to 20% of patients with cardiogenic shock following AMI, SIRS, most frequently a consequence of bacterial infection, significantly increases mortality (5). The mediators that contribute to the reduced SVR are probably cytokines and excessive production of nitric oxide due to deregulated activation of the inducible isoform of the nitric oxide synthase (NOS₂) in both cardiac tissue (6) and vascular bed (7). An interaction between NOS and cyclooxygenase isoforms could contribute to the cardiovascular abnormalities of SIRS (8).

There are several relevant implications of the new cardiogenic shock and inflammation paradigm: (1) Clinical and hemodynamic signs of inadequately low vascular resistance can be a presentation of cardiogenic shock complicated by SIRS. (2) A vasoconstrictor is probably part of the therapeutic armamentarium because it could improve left-ventricular to aorta coupling and increase coronary perfusion pressure. Nevertheless, vasoconstrictors could decrease mesenteric blood flow and result in bowel ischemia. (3) When hypnotics and opioids are prescribed for the sedation and anesthesia of patients with inadequately low SVR, they can result in further vasodilatation and reduced cardiac preload and arterial hypotension. This may require either

volume expansion to optimize preload or increased doses of vasoconstrictors. (4) Bacterial cultures should be prescribed routinely in such patients. The diagnosis of infection could probably be enforced by high values (>2 ng/mL) of procalcitonin (PCT), although it has been suggested that much higher PCT concentrations (>10 ng/mL) are predictive of infection in patients with cardiogenic shock (9). (5) If a bacterial infection is suspected, probabilistic anti-infectious therapy should be instituted rapidly (4), given the fact that mortality was significantly increased in culture-positive SIRS as compared to negative-culture SIRS and no SIRS patients, all with cardiogenic shock (5).

New Therapeutic Approaches

The most important therapeutic approach, in the authors' opinion, relevant for patients with AHFS who must undergo anesthesia, is the more and more frequent use of percutaneous cardiopulmonary support (PCPS) devices. Uni- or biventricular assist devices have been developed for different indications (bridge to transplantation, destination therapy in patients with contraindications to transplantation, and in a smaller number of patients as bridge-to-recovery). For many years they were inserted nearly exclusively by thoracotomy and were mainly intended for cardiac surgery patients. More recently, the use of PCPS devices has been extended to other situations such as cardiogenic shock due to a variety of causes, including acute intoxications and cardiopulmonary arrest (10). Results of PCPS devices in terms of survival are particularly encouraging in patients with cardiogenic shock as compared with patients with cardiovascular arrest. In a recently published randomized study in patients with cardiogenic shock after AMI, it was shown that the use of PCPSs resulted in improved hemodynamic and metabolic status as compared with the classic intraaortic balloon pump (IABP) counterpulsation (11). Nevertheless, the improved hemodynamic status with PCPS was associated with a higher incidence of severe complications and did not translate into improved 30 days survival (11). This observation suggests the requirement for additional studies in this field in order to improve survival in patients with PCPS.

The anesthetic and sedation techniques for the insertion and follow-up of PCPS have not been standardized. A whole array of anesthetic regimens has been reported (12). Anesthesiologists familiar with cardiac surgery and cardiopulmonary bypass techniques will not encounter any specific difficulties in the management of such patients and the PCPS devices, except that in many cases these patients undergo procedures outside the cardiac surgery operating room. For anesthesiologists unfamiliar with the above-mentioned situations, it is necessary to acquire the basic knowledge and skills in cardiopulmonary bypass, cannulas, pumps, oxygenators, anticoagulation, weaning from cardiopulmonary bypass (CPB), and the risk of bleeding. It is beyond the aims of this chapter to review the literature on PCPS devices. Nevertheless, their availability in an institution can dramatically change the management of patients with cardiogenic shock. The key to their success is early insertion. Ideally, algorithms for their insertion, according to specific clinical situations, should be implemented in order to respect the therapeutic window paradigm. One such algorithm is proposed in Figure 67.1. It has been suggested that recovery of cardiac function in such patients could be predicted by simple parameters such as increased end-tidal CO_2 concentrations and decreased arterial lactate.

Practical Issues Concerning Anesthesia for Patients with Acute Heart Failure Syndromes

There are several clinical situations in patients with AHFS that may require the intervention of the anesthesia team: (1) emergent cardiac surgery; (2) emergent nonsurgical myocardial revascularization procedures (such as percutaneous transluminal coronary angioplasty [PTCA]) with or without prior insertion of a PCPS device; and (3) emergent noncardiac surgery. Anesthesia for emergent cardiac surgery is discussed in major textbooks of cardiac anesthesia and will not be covered here. The two latter situations (items 2 and 3 on the above list) are the subject of this chapter because they are rarely discussed in textbooks. There are no evidence-based recommen-

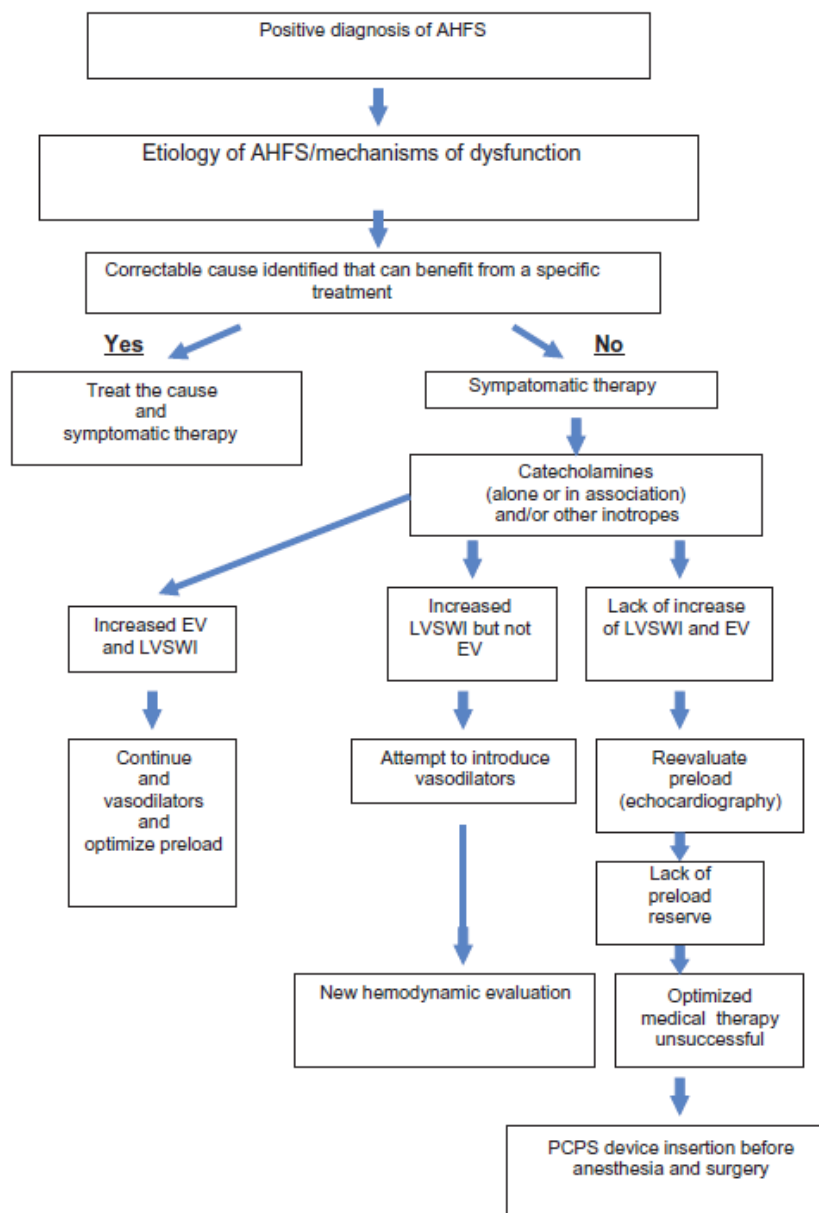


FIGURE 67.1. Algorithm for insertion of percutaneous cardiopulmonary support (PCPS) device in patients with acute heart failure syndromes. EV, ejection volume; LVSWI, left ventricular stroke work index.

Preoperative Evaluation

The time devoted to preoperative evaluation is short. The goals are to gather information concerning the positive and differential diagnosis of the AHFS, its causes and mechanisms, the therapy already instituted, and the response to therapy. A brief medical and surgical history should focus on chronic cardiovascular problems and medications as well as frequently associated comorbidities. These pieces of information are essential in order to anticipate, prevent, and correct (1) possible further alterations of cardiovascular performance by anesthetic drugs (Fig. 67.2); (2) changes in vital organ (lung, kidney, central nervous system, gut) function induced by the AHFS and its therapy; and (3) possible correctable factors that could increase oxygen transport.

Details concerning the onset of the AHFS should include therapy (inotropes, vasodilators, diuretics) as well as the time of the last oral intake in conscious patients without tracheal intubation. In patients already sedated and with tracheal intubation, the occurrence of cardiorespiratory arrest and external cardiac massage should be documented. The conditions of tracheal intubation could suggest the possibility of inhalation. Obtaining information on recent medication is important. Early (day 0 to day 1) administration of β -adrenergic antagonists in patients presenting with AMI has been reported to significantly increase the early risk of cardiogenic shock despite delayed beneficial effects through reduction of the rate of reinfarction. The increased risk of cardiogenic shock upon early administration of β -adrenergic blocking drugs in patients with

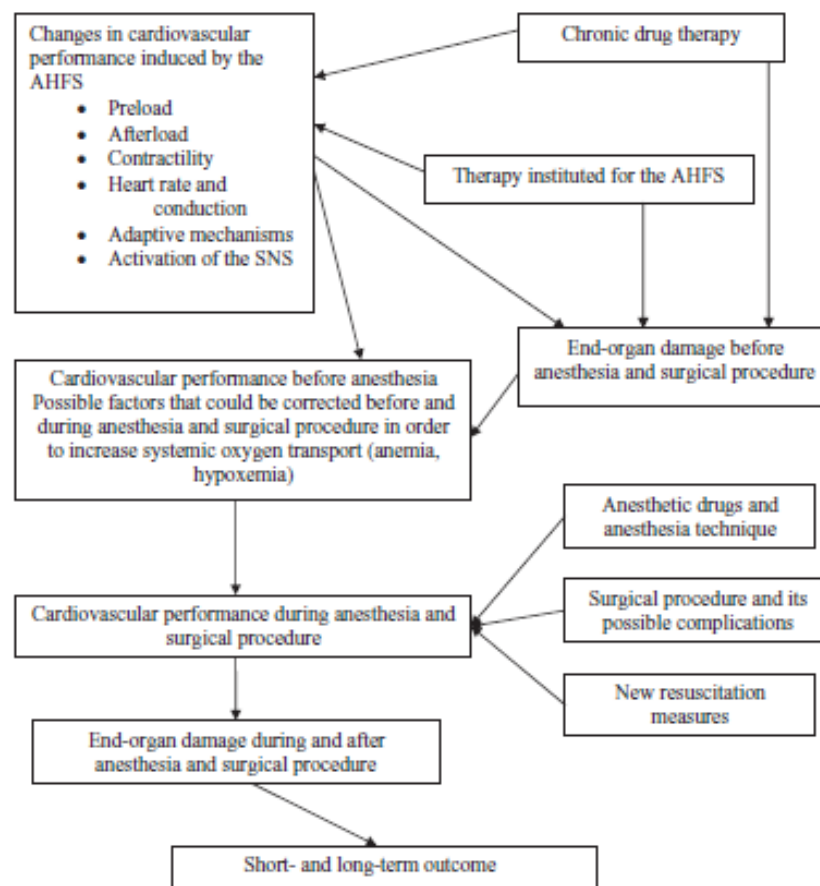


FIGURE 67.2. Impact of anesthesia and surgical procedure on short- and long-term outcomes. SNS, sympathetic nervous system.

AMI has triggered a recommendation to use β -adrenergic antagonists only in patients with AMI and stable hemodynamics. The occurrence of cardiogenic shock in patients with chronic or acute administration of β -adrenergic blocking drugs decreases the effects of β -adrenergic agonists and is an accepted indication for the use of positive inotropes such as levosimendan or phosphodiesterase inhibitors that have effects independent of the β -adrenergic receptor (13,14). Knowing the types and total amount of volume expanders already infused will help in interpreting the concentration of hemoglobin and plasma proteins.

Clinical examination should include vital signs such as noninvasive measurement of arterial blood pressure in both arms (even in the presence of a radial artery catheter), heart rate, respiratory rate in patients with spontaneous ventilation, temperature, and diuresis if a Foley catheter is in place. The neurologic examination is performed by taking into consideration the medication already used (hypnotics, opioids, neuromuscular blocking drugs). Conditions of lung ventilation (tidal volume, rate, positive end-expiratory pressure [PEEP], peak and plateau pressure values) should be recorded. Vascular access should be checked (caliber, location, back flow when possible for central venous catheters because vascular access could have been performed under substandard conditions).

The medical workup should focus on (1) systemic consequences of the AHFS such as acidosis (pH on blood gas, lactic acidosis), hemostasis abnormalities due to hemodilution or to disseminated intravascular coagulation (DIC) (PT, PTCA, fibrinogen, platelets, D-dimers), rhabdomyolysis (creatinine phosphokinase [CPK], myoglobin); (2) myocardial damage (troponin I); (3) alteration of vital organ function such as lung ($\text{PaO}_2/\text{FiO}_2$, PaCO_2), kidney (creatinine, BUN), liver (aspartate aminotransferase [ASAT], alanine aminotransferase [ALAT], bilirubin, coagulation factors), bowel (intraabdominal pressure); and (4) possible signs of infection (blood, urine and tracheal aspiration cultures, C-reactive protein, procalcitonin). Blood grouping and detection of irregular antierythrocyte antibodies are routine.

Imaging is usually limited to echocardiography and chest radiography, which provide helpful

information on the cardiac silhouette, lung edema, and the proper location of central venous catheters and tracheal tube.

The preoperative evaluation in patients who must undergo anesthesia despite the presence of an AHFS is challenging. The short symptoms/signs-diagnosis-therapy cycle must be translated into diagnoses, treatment decisions, and procedures.

Diagnoses

At the end of the preoperative evaluation, the anesthesiologist should be able to estimate the following: (1) The severity of the AHFS, which is demonstrated by the impairment of hemodynamic parameters (cardiac index, left ventricular stroke work, estimators of preload), and its consequences on oxygen transport and consumption (SvO_2 , lactic acidosis) as well as the amount of support (pharmacologic, mechanical) required to maintain those values. (2) The mechanisms of the AHFS should be documented as carefully as possible by preoperative and, if not detailed enough, by intraoperative echocardiography. Understanding the causes and mechanisms of the AHFS is mandatory in order to provide adequate therapy within the therapeutic window. It is essential to understand whether the patient has extracardiac (tamponade; pulmonary embolism) versus cardiac dysfunction, or myocardial versus valvular dysfunction. If myocardial dysfunction is the cause of the AHFS, then it is important to document systolic versus diastolic dysfunction, and left ventricular versus right ventricular versus biventricular failure. (3) The alterations of end-organ function due to the AHFS and its therapy (neurological, lung, renal, liver, bowel, metabolic) should be documented. (4) The occurrence of problems such as inhalation upon tracheal intubation should be documented. (5) The possible factors that could be corrected in order to increase oxygen transport such as preload, afterload, atrioventricular (AV) asynchrony, and anemia should be documented. (6) Major electrolyte (potassium, calcium, magnesium) and metabolic abnormalities (hyperglycemia) should be documented.

Treatment Decisions

The severity of the impairment and the mechanism of AHFS should allow the anesthesiologist to address two issues. The first is whether the cardiac dysfunction is reversible in the short term, that is, in minutes (e.g., cardiac tamponade), potentially reversible after a period of hours to days (myocardial revascularization, acute valvular dysfunction that can be corrected by cardiac surgery), or probably not reversible (a history of recurrent myocardial infarction in a patient with chronic heart failure). If the cardiac dysfunction is not rapidly reversible, the second issue is whether the pharmacologic support is sufficient to avoid vital organ dysfunction and multiple organ failure, or whether the patient should receive a PCPS device before the index surgery or procedure. There are no widely accepted criteria for the institution of PCPS in this context. Other decisions concern strategies for transportation to the operating room, anesthesia induction (rapid sequence if the patient is not already intubated), maintenance of anesthesia, and postoperative care.

Procedures

Procedures done before induction of anesthesia will depend on the balance between, on the one hand, the necessity to rapidly correct the cause of AHFS (e.g., rapid myocardial revascularization by PTCA) and, on the other hand, the necessity to stabilize the hemodynamic and metabolic status of the patient before the procedure. When time is available, procedures include implementation of monitoring (cardiovascular, respiratory, temperature). There is a lack of consensus about the choice of a specific cardiovascular monitoring strategy (invasive versus noninvasive). Frequently, arterial and central venous catheters are required. There are no proved benefits of pulmonary artery catheters on mortality. Also, when time is available, preanesthesia improvement of vascular access should be attempted. The lack of sufficient and secure intravascular lines should be anticipated. The intravenous lines for inotropes and anesthetic drugs administration should be separated. A Y-type trifurcated intravenous extension set with a low (<0.5 mL) priming volume is helpful to simultaneously infuse several anesthetic drugs

with the lowest possible dead volumes. In addition, intravascular access for rapid volume expansion should be available.

The Anesthesia Procedure

The therapeutic goals upon induction of anesthesia are as follows: (1) preserve life through maintenance of the cerebral and coronary perfusion pressure; (2) avoid further worsening of preload and afterload conditions; (3) minimize changes in heart rate that could worsen myocardial ischemia or valvular dysfunction; (4) avoid complications such as inhalation if the patient's trachea is not already intubated; (5) avoid explicit awareness upon tracheal intubation due to inadequately low effect site concentrations of hypnotics and opioids; (6) avoid worsening of end-organ function (lung, renal) by inadequate volume expansion. The therapeutic goals for maintenance are as follows: (7) continue to provide hemodynamic stability as well as "cardioprotection"; (8) avoid anesthetic drugs over- and underdosing by monitoring depth of anesthesia; (9) maintain homeostasis (temperature, hemoglobin, glycemia, electrolytes); (10) prevent and correct hemostasis abnormalities to avoid excessive bleeding during surgery.

For most AHFS patients who require anesthesia, recovery will take place in an ICU. Tracheal extubation is delayed until cardiopulmonary and other vital organs functions have stabilized. Pain therapy is usually administered in the context of postoperative sedation of a patient with intubated trachea, mechanical ventilation, and in the most severe cases multiple organ dysfunction or failure.

Providing Hemodynamic Stability (Goals 1 to 3 and 7)

The main immediate concerns when choosing anesthetic drugs for induction and maintenance of anesthesia are preservation of (nearly) physiologic cerebral and coronary perfusion pressure together with minor changes in heart rate. Such hemodynamic stability is often, but not always, associated with preserved myocardial oxygen balance and no electrocardiogram (ECG) signs of myocardial ischemia (usually estimated by changes of the ST segment). Other, less immediate

concerns are anesthetic drug-mediated myocardial protection, especially in patients who will be confronted with myocardial ischemia/reperfusion sequences such as those who will undergo cardiac surgery.

These general considerations require several comments. First, in addition to the intrinsic effects of anesthetic drugs on cardiovascular function, their integrated effects in a given patient depend on chronic and acute preoperative medication (β -adrenergic receptor antagonist for instance) as well as on their effects on the autonomic nervous system. Second, in most, if not all, published studies, any mean differences in terms of hemodynamic effects between anesthetic regimens are lower than interpatient variability within the same regimen. In other words, whatever the choice of anesthetic drugs, the main challenge, yet unsolved in the literature, is the titration of anesthesia for an individual patient. Two algorithms for titration of anesthetic drugs upon anesthesia induction have been proposed in patients without AHFS and could partially be extrapolated to patients with AHFS (15,16). Third, in the most difficult cases, hemodynamic stability is a secondary goal, just after avoidance of inhalation by rapid sequence induction. In these cases, once the airway is secured, correction, rather than prevention of hemodynamic instability, is the only choice.

For induction of anesthesia, the hypnotic drug with the highest therapeutic index on hemodynamic stability is etomidate because it preserves sympathetic outflow and autonomic reflexes. This has been demonstrated in prospective randomized clinical studies and in studies analyzing thousands of patients in routine clinical practice. Experimental data also suggest that in contrast to other hypnotics, the pharmacokinetic and pharmacodynamic effects of etomidate are not altered by shock states. Several studies have raised concerns for an effect of etomidate, even given in single bolus infusion, on cortisol metabolism and subsequent infraclinical adrenal insufficiency. Supplementation with low-dose corticosteroids could be helpful in the postoperative period in patients with cardiogenic shock who received etomidate for anesthesia induction.

For maintenance of anesthesia, it has been shown in patients undergoing coronary artery bypass graft surgery that inhaled (sevoflurane and

desflurane) as compared to intravenous (propofol and midazolam) hypnotics resulted in better postoperative myocardial function (17). Although sevoflurane and desflurane have not been specifically investigated in patients with AHFS, clinical experience suggests that these two drugs could be a reasonable choice for these patients during maintenance of anesthesia mainly because their hemodynamic effects are no worse than those of other drugs such as propofol and because of their clinically relevant cardioprotective effects. Sevoflurane has been shown to better preserve left ventricular function in elderly patients as compared to propofol. Sevoflurane has also been shown to have cardiac sympatholytic effects, whereas propofol does not. This effect could be beneficial in patients with myocardial ischemia in order to avoid further tachycardia. Inhaled anesthetics are mainly used in the operating room. For patients who require anesthesia in the catheter laboratory, intravenous anesthetics are the first choice.

The choice of opioid is also based on hemodynamic stability upon induction and maintenance. From a pharmacokinetic point of view, the most interesting drug is remifentanyl, especially when administration by target-controlled infusion (TCI) through commercially available devices (in Europe) allows more reproducible titration. The advantages of remifentanyl are its short onset time and short contextual half-life that allows rapid decrease of plasma and effect site concentrations, even in the presence of liver or renal dysfunction. The main concern with remifentanyl upon induction, when given by bolus, is the occurrence of severe bradycardia and even systolic especially when patients receive preoperative β -adrenergic receptor antagonists or calcium channel blockers such as diltiazem. Administration with TCI devices by targeting plasma and not the effect compartment results in lower plasma concentrations and less severe bradycardia.

If muscle relaxation is required during the procedure, the choice of the neuromuscular blocking during maintenance of anesthesia should be based on its effects on heart rate, especially through interaction with the opioids. Vecuronium as opposed to pancuronium and suxamethonium, when associated with etomidate and fentanyl, results in the highest incidence of bradycardia requiring atropine. It also should be based on the

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pharmacokinetic properties and the possible alterations induced by renal and hepatic impairment.

Avoiding Perioperative Awareness (Goals 5 and 8)

Many anesthesiologists, when providing anesthesia for patients with AHFS, focus their attention and efforts on hemodynamic stability. Although this is an accepted proof of quality of anesthesia for patients with AHFS, providing comfort and avoiding explicit awareness are also necessary. The occurrence of explicit perioperative awareness is reported by most patients as being one of the worst possible personal experiences (18).

Recent evidence from observational and prospective (18) trials have shown that patients with altered cardiovascular reserve are at increased risk of perioperative awareness because of inadequately low concentrations of hypnotics and opioids for a given intensity and duration of nociceptive stimulation. Interestingly, it was shown that an interval of inadequately "shallow" anesthesia as short as 1 to 2 minutes was probably sufficient to result in explicit awareness. Risk factors of perioperative explicit awareness, in addition to reduced cardiovascular reserve, were cardiac, abdominal/thoracic, or orthopedic surgery (versus all other types of surgery) (18).

There are several complementary methods of preventing or dealing *a posteriori* with explicit awareness that have recently been recommended in a "sentinel event alert" of the Joint Commission on Accreditation of Healthcare Organization (October 6, 2004, issue 32; available at www.jcaho.org). Among other methods, this document, together with a document from the American Society of Anesthesiology (ASA) (available at www.asahq.org) suggests that in patients at risk of perioperative explicit awareness, the use of depth of anesthesia monitors can reduce by approximately 80% the incidence of such episodes. These recommendations are based on observational and randomized-controlled trials. In addition to the decrease in the incidence of perioperative explicit awareness, monitoring depth of anesthesia is probably helpful in avoiding anesthetic drugs overdose in such patients. Anesthesia overdose has been incriminated (although no formal proof

was provided) in increasing long-term mortality after surgery (19). We think that the above-cited studies and the recommendations of JCAHO and ASA are arguments in favor of monitoring depth of anesthesia in all patients with AHFS who must undergo general anesthesia. This is a financial and organizational challenge, especially for patients who require anesthesia outside of the operating room. A few algorithms for titration of anesthesia in order to avoid under- and overdosing of anesthetic drugs have been published (15,16).

Other Concerns for the Perioperative Period, Not Specific of Patients with Acute Heart Failure Syndromes

In all patients undergoing anesthesia, maintenance of homeostasis is a routine goal. This means avoiding hypothermia, severe anemia, and hyperglycemia.

End of Surgery, and Transfer to the Recovery Area or Intensive Care Unit

Part of the anesthesia plan is the choice of the recovery unit and the transfer from the operating room. This requires the choice of a sedation regimen. Continuation of the opioid infusion at a lower dose concentration than that used during surgery is reasonable. An effect site remifentanyl concentration of 1 to 2 ng/mL is adequate in most situations in the absence of nociceptive stimulation. If an inhaled anesthetic was used during maintenance, an intravenous hypnotic should be started in the operating room sufficiently early to provide adequate hypnosis during transfer to and installation in the recovery unit. Propofol, given by TCI, at 1 to 1.5 µg/mL is a reasonable choice. Defining such a sedation regimen in each institution is useful for patients with AHFS who are frequently transferred between several locations of the hospital.

Clinical Case

A 78-year-old man with a history of chronic arterial hypertension, tobacco use, old myocardial infarction, peripheral vascular disease, and

abdominal aorta aneurysm was scheduled for angiography with possible percutaneous iliac angioplasty without the supervision of the anesthesia team. The patient was taken to the operating room, and positioned on the operation table after insertion of one intravenous cannula. Oxygen ($\text{FiO}_2 = 0.5$) was given through a face mask. After the first attempt of femoral artery cannulation, the patient became agitated and complained of severe shortness of breath, and the anesthesia team was asked to intervene.

Upon arrival of the anesthesia team, the patient was conscious and complained of worsening shortness of breath. His respiratory rate was 60/min and he was cyanotic. Pulse oxymetry revealed that oxygen saturation was 73%. Arterial blood pressure was 245/130 mmHg and heart rate was 110 bpm. Lung auscultation revealed fine crackles in the two lung fields. The FiO_2 was increased to 1, and the patient was given two intravenous bolus injections of 2 mg of nicardipine, 20 mg of furosemide, and 2 mg of isosorbide dinitrate. Arterial blood pressure decreased to 160/95 mmHg without changes in heart rate and there was no improvement in the shortness of breath. Oxygen saturation decreased to 34% and the patient did not respond anymore to verbal commands.

The patient was administered 0.5 mg/kg of etomidate, 15 μg of sufentanil, and 30 mg of atracurium, and tracheal intubation was performed without problems and manual ventilation was performed for 5 minutes with oxygen. Anesthesia was maintained with 2% sevoflurane. Oxygen saturation increased to 95%. Mechanical ventilation was instituted with a tidal volume of 8 mL/kg, a respiratory rate of 12/min, and a PEEP of 8 cm H_2O . Lung auscultation revealed crackles in the two lung fields and symmetric breath sounds. Blood pressure decreased to 120/75 mmHg and heart rate was 110 bpm. A second intravenous cannula was inserted with a three-way device and anesthesia was maintained with TCI (Base Primea®, Fresenius Vial, Brézins, France), remifentanyl (3 ng/mL), and sevoflurane 2%. Depth of anesthesia was monitored with a BIS XP® monitor (Aspect Medical System, Newton, MA, USA) and sevoflurane concentrations were adapted to maintain BIS values between 50 and 55. Pulse oxymetry revealed oxygen saturation of 99%, the FiO_2 was decreased to 0.6, and the surgical and anesthesia

team decided to continue the procedure. After 2 hours, oxygen saturation was stable, arterial blood pressure was 130/80 mmHg, and the patient was taken to the ICU under propofol sedation (TCI, Diprifusor®, 2 $\mu\text{g/mL}$). A Foley catheter was inserted and a chest x-ray was performed and revealed acute pulmonary edema. Transthoracic echocardiography revealed a left ventricular ejection fraction of 0.5 and an undilated right ventricle. The propofol infusion was stopped; 30 minutes later, the remifentanyl infusion was stopped. The patient was weaned from the mechanical ventilation 2 hours after arrival in the ICU and received oxygen through a face mask for the following hours.

This clinical case concerns anesthesia in a patient with AHFS, diagnosed as hypertensive acute heart failure (signs and symptoms of AHF in the presence of high arterial blood pressure and preserved left ventricular systolic function). Because respiratory and hemodynamic functions were rapidly stabilized, it was possible to continue the procedure. Therapy to reduce arterial blood pressure was instituted according to the ESC guidelines (1). Anesthesia was induced and maintained by taking into account the above-described goals.

Conclusion

Providing anesthesia for patients presenting with AHFS is challenging for anesthesiologists not familiar with cardiac surgery. This field is uncharted territory because there are no evidence-based recommendations. The easiest way to adopt an anesthesia plan in such patients, in our opinion, is to define goals that should include, but not be limited, to hemodynamic stability. Through careful titration of anesthetic drugs, it is often possible to provide both hemodynamic stability and to avoid anesthetic drug over- and underdose.

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