A Practical Approach to the Perioperative Management of Heart Failure

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Abstract

Demographic aging and its implications for healthcare delivery is a global concern. Heart failure is the leading cause of hospital admission in patients older than age 65. It is a major risk factor for postoperative morbidity and mortality, representing one of the most challenging and expensive problems in medicine and surgery. Every anesthesia provider must be familiar with the definition, classification, pathogenesis, and perioperative management associated with heart failure. This review will focus on five key questions that anesthesiologists can incorporate into a strategic approach to managing the surgical patient with heart failure.

Keywords: systolic heart failure; diastolic heart failure; cardiomyopathy; anesthesia; preoperative evaluation; blood volume management

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Introduction

The terms “coronary artery disease” and “congestive heart failure” are common listings on the pre-anesthesia medical record. Coronary artery disease is a structural and readily conceptual diagnosis. In contrast, the term “CHF” is often vaguely listed on the pre-anesthesia record and poorly characterized in patients for non-cardiac surgery. Furthermore, many aspects of heart failure (HF) are poorly understood. Every anesthesia provider must be familiar with the definition, classification, pathogenesis, and treatment strategies associated with HF. Comprehensive guidelines for the diagnosis and treatment of HF were first published in 1995, were revised in 2001, 2009, and recently in 2013 [1,2].

As survival in patients with coronary disease has increased, so has the prevalence of heart failure [3]. Patients with HF have a significantly higher risk of postoperative mortality than patients with coronary disease, even in minor procedures [4]. From the year 2010 to 2030 the prevalence of all cardiovascular disease is projected to increase by 10%, but the prevalence of HF is projected to increase by 25%, such that 1 in 33 Americans will have heart failure [5]. In the same time frame, total costs for HF are estimated to increase from $31 billion to $70 billion [3]. Heart failure is the most common reason for hospital admission in patients older than age 65 [6]. Taken together, HF is emerging as one of the most challenging and expensive problems in medicine and surgery. In a recent surgical outcomes study of HF patients, adverse events within one month of non-cardiac surgery occurred in 30%; of these were death (8%), myocardial infarction (15%), and HF exacerbation (25%). Patient factors that increased risks of these complications included age>80, diabetes, and ejection fraction (EF) less than 30% [7]. Due to the broad nature of this topic, this review will focus on five key questions that anesthesiologists can incorporate into a strategic approach to managing the surgical patient with heart failure.

What is the type and cause of heart failure?

The common definitions used to classify the type of HF are summarized in Table 1. Heart failure is the
syndrome that results from a reduced filling of blood, or reduced ejection of blood from the ventricle. The unifying theme in HF is the clinical presentation of dyspnea and fatigue which may limit exercise tolerance. Fluid retention may lead to pulmonary venous congestion and peripheral edema. Together, the ultimate consequence is a reduction in quality of life and life expectancy [8]. While there is no single test that confirms the diagnosis of HF, the categorical feature of systolic HF is an EF less than 40%, compared to an EF greater than 50% in diastolic HF, while individuals with an EF between 41 and 49% are considered intermediate.

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
<th>Comments/features</th>
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<tbody>
<tr>
<td><strong>Heart Failure</strong></td>
<td>Structural or functional impairment of ventricular filling or ejection</td>
<td>Lifetime risk is 20% for Americans over age 40. Mortality is 50% within 5 years of diagnosis.</td>
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<tr>
<td><strong>HFrEF</strong></td>
<td>HF with reduced EF, formerly systolic HF: Inability of heart to move blood forward at a sufficient rate to meet the metabolic demands of the body</td>
<td>EF typically &lt; 40% Prevalence less than half of HF cases. Major causes are ischemic heart disease and idiopathic.</td>
</tr>
<tr>
<td><strong>HFpEF</strong></td>
<td>HF with preserved EF, formerly diastolic HF: Ability of heart to move blood forward only if the cardiac filling pressures are abnormally high</td>
<td>EF typically &gt; 50% Prevalence more than half of HF cases and more common in women. Major cause is hypertension.</td>
</tr>
<tr>
<td><strong>HF intermediate EF</strong></td>
<td>HF with intermediate EF</td>
<td>EF between 41-49%. Treated similarly to HFpEF</td>
</tr>
<tr>
<td><strong>Cardiomyopathy, LV dysfunction</strong></td>
<td>Structural and functional disorders of the myocardium</td>
<td>Includes a wide range of abnormalities that cause HF</td>
</tr>
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<td><strong>Dilated Cardiomyopathy</strong></td>
<td>Myocardial dysfunction characterized by ventricular dilatation and decreased contractility</td>
<td>Most commonly idiopathic in origin and associated with better survival than other causes of cardiomyopathy; 25% of cases improve in a short time</td>
</tr>
<tr>
<td><strong>Peripartum cardiomyopathy</strong></td>
<td>LV dysfunction in the last trimester of pregnancy, unknown cause but risk factors include multiparity, advanced maternal age, African descent, long-term tocolysis.</td>
<td>After 6 months: improved LV function in 50% of patients; much worse prognosis if LV function does not improve (6 year mortality 50%)</td>
</tr>
<tr>
<td><strong>Compensated HF</strong></td>
<td>Moving up the Frank-Starling curve (Na+ and water retention), LV hypertrophy, neurohumoral activation</td>
<td>Improving NYHA class</td>
</tr>
<tr>
<td><strong>Decompensated HF</strong></td>
<td>Increased cardiac workload due to increases in metabolic demand, increased circulating volume, decreased contractility</td>
<td>Worsening NYHA class</td>
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The heart consumes more energy than any other organ, pumping about ten tons of blood throughout the body per day. When cells in the myocardium become deprived of energy, this abnormality of “myocardial energetics” plays a major role in progression of HF. There are three components of cellular energy metabolism: 1) substrate utilization: the uptake and conversion of free fatty acids and glucose into the Kreb’s cycle; 2) oxidative phosphorylation: producing ATP from the mitochondrial respiratory chain; and 3) transfer and utilization of high-energy phosphates (ATP) to the myofibrils [9]. Dysfunction in all three components of myocardial energetics is early and integral to HF...
pathogenesis. Future therapeutic approaches will target these molecular and metabolic pathways. In contrast to the common clinical presentation of HF, the causes of HF are widely variable. Growing evidence suggests that there are unique characteristics in risk factors, pathophysiology, treatment, and outcomes in systolic vs. diastolic heart failure [10]. Figure 1 displays common and contrasting features of systolic and diastolic HF.

Causes of HF can be classified as cardiogenic or non-cardiogenic. Cardiogenic causes of HF are inherently related to pathophysiology of the myocardium, the valves, and the pericardium, and can be classified as structural and functional. Non-cardiogenic causes of HF are non-cardiac pathologies that are injurious to myocardium or evoke a physiological state that resembles heart failure.

Figure 1. Common and contrasting features of heart failure with reduced HF (HFrEF) and heart failure with preserved HF (HFpEF). HFrEF or systolic HF is generally associated with an acute injury to myocardium, or a genetic abnormality injurious to myocardium. The pathophysiology of HFpEF, or diastolic HF, is insidious in onset primarily from poorly controlled hypertension, but also confounded by aging and poorly controlled comorbidities. While initial physiologic adaptations are common between syndromes, the maladaptive processes distinguish the two. The arrows represent the cycle of heart failure that continues when inadequate therapies exacerbate the syndrome.

For structural changes in systolic HF, ischemic heart disease is the leading cardiogenic cause. Myocardial infarction changes the ventricular geometry within hours to days. The area of myocardium expands and becomes thinner. Global remodeling results in ventricular dilatation (eccentric hypertrophy), decreased systolic function, mitral valve dysfunction, and aneurysm formation [11]. For diastolic HF, hypertensive heart disease is the leading cardiogenic cause associated with remodeling, as the ventricular walls become thickened (concentric hypertrophy) and ejection fraction is preserved.

Chemotherapy induced cardiomyopathy can be attributed to a variety of agents, with the most established class being anthracyclines. Doxorubicin (Adriamycin) toxicity is thought to be due to oxidative stress, decreased antioxidant enzymes, and possibly iron accumulation in cardiomyocytes [12,13]. The incidence of heart failure after doxorubicin is dependent on age and dose, with an accelerated risk of myocardial toxicity when doses exceed 550 mg/m² [14]. Trastuzumab (Herceptin) is a monoclonal antibody against HER2 protein for the treatment of positive HER2 breast cancer [15]. Trastuzumab myocardial toxicity is not related to dose, but potential cardiotoxicity increases when combined with anthracycline therapy, and the effects are greater if the cumulative doxorubicin dose is > 300 mg/m² [16]. Other common chemotherapy agents that are associated with myocardial depression are mitoxantrone (Novantrone), cyclophosphamide (Cytoxan),
ifosfamide (Ifex), all-trans retinoic acid (Tretinoin), and imatinib (Gleevec) [17].

Detailed examples of additional structural causes of HF are beyond the scope of this review but include: gene mutations, inborn errors of metabolism, toxins (i.e., alcohol, smoking, illicit drugs, lead poisoning), infections such as viral (i.e., HIV), bacterial, Lyme disease, fungal, parasitic (i.e., Chagas disease), and idiopathic. Indeed, the leading cause of non-ischemic dilated cardiomyopathy is idiopathic, present in up to 50% of patients where no definitive cause is found [18].

Functional causes of HF are illustrated in Figure 2. Functional causes may overlap with structural causes that reduce contractile function, such as myocardial infarction. However, a major cause of non-ischemic contractile dysfunction is chronic volume overload due to either severe mitral regurgitation or severe aortic regurgitation. This leads to chamber dilation and eccentric hypertrophy. Pressure overload is a functional cause of diastolic HF, where chronic hypertension or chronic aortic stenosis lead to ventricular remodeling which increases wall thickness in attempt to reduce the wall stress through LaPlace’s relation for a hollow sphere:

$$\sigma = \frac{P \times r}{2h},$$

where \(\sigma\) = wall tension, \(P\) = pressure, \(r\) = radius, and \(h\) = wall thickness.

Figure 2. Functional causes of left sided heart failure. Impaired contractility results from a combination of structural damage to myocardium, most commonly by myocardial infarction and ischemic heart disease, with functional causes that perpetuate the syndrome of heart failure, such as increased sympathetic nervous system activity and neurohormonal activation. Pressure overload can induce left ventricular (LV) systolic or diastolic dysfunction. Diastolic dysfunction is the abnormal relaxation or myocardial stiffness associated with chronic hypertension, pressure overload, and myocardial thickening. Without proper management diastolic dysfunction can progress to LV systolic dysfunction or HF with preserved EF.

Diastolic dysfunction is the abnormal relaxation associated with chronic hypertension, pressure overload, and myocardial thickening. Without proper management it can progress to HF with preserved EF. Other causes of diastolic dysfunction that aren’t directly caused from pressure overload include hypertrophic cardiomyopathy and restrictive cardiomyopathy.

Another common functional cause of HF relevant to the anesthesiologist is tachyarrhythmia-induced cardiomyopathy. A typical surgical presentation of tachyarrhythmia-induced cardiomyopathy is atrial fibrillation with rapid ventricular response and
associated arterial thrombosis (i.e., ischemic stroke, mesenteric ischemia, or cold leg). Emergency echocardiography may reveal severe global hypokinesis with a severely depressed ejection fraction. When caught early, these tachyarrhythmia-induced cardiomyopathies are at least partially reversible with rate control and medical management [19].

The pathophysiology of systolic HF is a complex interaction between injury of the myocytes and extracellular matrix, ventricular remodeling, sympathetic nervous system activation, and activation of the renin-angiotensin-aldosterone system [20]. Sympathetic nerve activity is normally increased in aging, but substantially increased during HF [21]. Circulating catecholamine levels (norepinephrine, epinephrine) are increased, which leads to salt and fluid retention, peripheral vasoconstriction, and down-regulation of beta-1 adrenergic receptors in the heart [22]. Vagal function in the heart is also reduced in proportion to the severity of heart failure [23,24].

Concomitant with overdriving sympathetic nerve traffic, HF is associated with an over-active renal angiotensin-aldosterone system (RAAS) [25]. This further increases salt and fluid retention, and the RAAS hormones mediate some of the maladaptive effects on the myocardium[26,27]. Natriuretic peptides are released, and circulating cytokines such as interferons and tumor necrosis factor further mediate dysfunctional myocardial remodeling [28].

Interestingly, co-morbid conditions tend to segregate between systolic and diastolic HF. Previous myocardial infarction is the major risk factor for systolic HF, followed by sleep apnea, hypertension, and diabetes. Diastolic HF is associated with a greater variety of diseases, with the major risks being obesity, hypertension, and diabetes, followed by chronic lung disease, dialysis, and sleep apnea [11].

What is the stage and class of heart failure?
The patient’s functional status remains a key feature in preoperative cardiac evaluation. In previous ACC/AHA guidelines for preoperative clinical predictors of anesthesia risk, decompensated HF was classified as a major clinical risk factor, and compensated or prior HF, an intermediate risk factor [29]. The most recent ACC/AHA guidelines for preoperative cardiovascular evaluation list decompensated HF as an “active cardiac condition,” for which the HF patient should undergo evaluation and optimization of HF before elective surgery [30]. Compensated HF or prior HF is now considered a clinical risk factor warranting further assessment of functional capacity, other clinical risk factors, and surgical risk. Adapted from the original Goldman criteria, the Revised Cardiac Risk Index (RCRI) lists “History of HF” as one of six independent predictors of cardiac risk in the determination of an individual’s RCRI [31]. More recently, Sabate and colleagues identified HF as an independent risk factor for major adverse cardiac and cerebrovascular events following non-cardiac surgery [32].

Class: The New York Heart Association (NYHA) functional classification correlates with quality of life and survival. Because the paramount symptom of HF is dyspnea on exertion, the NYHA class stratifies patients based on the symptom severity as described in Table 2. Class IV HF has a worse prognosis than most cancers in the U.S., with a one-year mortality approaching 45% [11].

Stage: Historically, there have not been widespread screening efforts for HF. Risk factors for HF were either poorly defined or poorly publicized. In an attempt to prevent or reverse HF progression, the ACCF/AHA guidelines developed clinical staging criteria to identify patients at risk for—or in early stages of—HF [1]. The staging criteria allow for patients to be stratified into clinical trials. Results of these trials form the guidelines for medical management. In contrast to NYHA class where patients can move up or down a continuum of functional status, patients move through the stages of HF in one direction, not reverse.

What are the current medications?
By understanding the treatment strategy, summarized in Table 2, one can determine a patient’s current HF stage. The medications in HF serve to reduce cardiac sympathetic stimulation, lessen sodium and water retention, and decrease afterload, collectively breaking the cycle of physiological adaptations and pathologic maladaptations depicted in Figure 1.

On the electrocardiogram, a prolonged QRS complex is present in approximately 1/3 of HF patients, which is associated with worse outcome [33]. The associated bundle branch block produces a loss of ventricular coordination called dyssynchrony or mechanoeenergetic uncoupling which hastens maladaptive remodeling [34]. Cardiac resynchronization therapy (CRT) is an attempt to coordinate ventricular depolarization. With pacer
leads in both ventricles, CRT brings synchronized contraction to the ventricles, which reverses pathologic remodeling, may improve mitral regurgitation if present, improve ejection fraction, and can improve symptoms and NYHA class [35].

Table 2. Stage, class, and treatment algorithm of HF

<table>
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<tr>
<th>NYHA Functional Classifications</th>
<th>ACCF/AHA Stages of HF</th>
<th>Treatment Algorithm</th>
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<tbody>
<tr>
<td>A</td>
<td>At high risk for HF but without structural heart disease or symptoms of HF</td>
<td>Risk-factor reduction, education; treat hypertension, diabetes, dyslipidemia. Smoking cessation and avoidance of cardiotoxic agents</td>
</tr>
<tr>
<td>I</td>
<td>No limitations of physical activity. Ordinary physical activity does not cause symptoms of HF</td>
<td>Structural heart disease but without signs or symptoms of HF</td>
</tr>
<tr>
<td>I</td>
<td>No limitation of physical activity. Ordinary physical activity does not cause symptoms of HF</td>
<td>Structural heart disease with prior or current symptoms of HF</td>
</tr>
<tr>
<td>II</td>
<td>Slight limitation of physical activity, Comfortable at rest, but ordinary physical activity results in symptoms of HF</td>
<td>Structural heart disease</td>
</tr>
<tr>
<td>III</td>
<td>Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes symptoms of HF</td>
<td>Refractory HF requiring specialized interventions</td>
</tr>
<tr>
<td>IV</td>
<td>Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest.</td>
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ACCF, American College of Cardiology Foundation; AHA, American Heart Association; HF, heart failure; NYHA, New York Heart Association; MI, myocardial infarction; ARB, angiotension receptor blocker. Table adapted from reference [1].

There is strong evidence to suggest that CRT is indicated in patients with left ventricular EF less than or equal to 35%, a QRS complex greater than 120 milliseconds, a left bundle branch pattern on ECG, sinus rhythm, and NYHA class II or III symptoms [1]. Weaker evidence suggests a benefit of CRT in patients with atrial fibrillation with EF less than 35%, optimal medical therapy, and fully
dependent on ventricular pacing [1]. Importantly, approximately 80% of biventricular pacers will include an automated internal cardiac defibrillator (ICD).

There are two strong indications for ICD’s in HF. This first is to prevent sudden cardiac death in patients with non-ischemic cardiomyopathy, ischemic cardiomyopathy > 40 days post myocardial infarction, an EF less than or equal to 35%, and have a NYHA class II or III with a reasonably predicted survival beyond one year [1]. The second indication is to prevent sudden cardiac death in patients with an EF less than or equal to 30%, with NYHA class I symptoms while receiving optimal medical management, who are also expected to live beyond one year [1]. CRT is not indicated for patient with comorbidities and an poor expected survival. One must weigh the benefits against the potential for worsening quality of life because of the discomfort from shock delivery.

What workup does this patient need before surgery?

The patient with HF presents in one of three ways: 1) A noticeable onset of exertional dyspnea or decrease in exercise tolerance; 2) Fluid retention; 3) With no symptoms or with symptoms of another cardiac disorder or noncardiac disorder. The complete history and physical exam will delineate cardiac versus noncardiac causes of presentation. The routine ECG and chest X-ray have low sensitivity and specificity for diagnosing a cardiac cause of HF, but are useful in the general assessment of cardiac and pulmonary pathology.

The most useful diagnostic test is echocardiography to address three main questions: 1) ejection fraction; 2) left ventricular structure, dimension, wall motion; and 3) non-left ventricular abnormalities (i.e., valve, pericardium, right ventricle). Additional echocardiography indices will include atrial size and pressure, characteristics of LV filling, and pulmonary arterial pressure. The comprehensive echocardiogram will also provide a baseline for future comparison and therapeutic management. Laboratory testing will include natriuretic peptides (BNP and/or NT-proBNP)Figure 3, complete blood count, urinalysis, electrolyte panel, hemoglobin A-1c, lipid panel, renal, liver, and thyroid function, as well as viral screening.

**Figure 3. Brain-type natriuretic peptide.** BNP is also called brain type natriuretic peptide because it was first discovered in brain tissue. In cardiac myocytes, dilation and pressure overload on the cell causes expression of the gene that translates the pro-hormone. The pro-hormone gets cleaved into an N-terminal pro-BNP that is inactive but measurable, and C-terminal BNP which is the active hormone that inhibits the renin angiotensin aldosterone system (RAAS), promotes diuresis and vasodilation, and thereby decreases preload and afterload. NT-proBNP has a half-life of 1-2 hours and BNP has a half-life of 20 minutes.
The pre-operative holding area: Is this patient in heart failure? Certainly patients in acute HF should not undergo elective procedures, and anesthesia providers must be prepared to diagnose and triage HF patients in the holding area. A meta-analysis of HF diagnostic criteria in patients presenting to the emergency room with dyspnea can be extrapolated to our pre-operative evaluations [36]. If a patient is dyspneic, the positive predictors that suggest HF are listed in Table 3. The strongest predictor of HF in a dyspneic patient is medication non-adherence. Atrial fibrillation is an important predictor of HF because the prevalence of a-fib in patients with acute HF is greater than 30% [37]. Of note, the reason serum BNP or NT-proBNP is not listed as a positive predictor is because it can also be elevated in advanced age and critical illness. This would include renal failure, acute coronary syndrome, lung disease with cor pulmonale, acute pulmonary embolus, and high output cardiac states such as sepsis. However, if BNP is low, this suggests that a patient does not have HF. The most negative predictors that would dissuade the diagnosis of HF are also listed in Table 3.

<table>
<thead>
<tr>
<th>Table 3. Is this patient in heart failure...do I cancel this case?</th>
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<tr>
<td><strong>Suggested of systolic HF (HFrEF):</strong></td>
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<tr>
<td>Comorbidities</td>
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<tr>
<td><strong>Predictors of acute decompensated HF</strong></td>
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<tr>
<td><strong>Positive predictors:</strong></td>
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<tr>
<td>History</td>
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<tr>
<td>Physical exam</td>
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<tr>
<td>Chest X-Ray</td>
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<tr>
<td>Electrocardiography</td>
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<td>Monitors/Laboratory</td>
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*indicates that while BNP and/or NT-proBNP is elevated in acute HF, there are many other conditions associated with an elevation of these markers.
Preoperative medications: For preoperative continuation of anti-hypertensive agents, concerns have arisen with respect to profound and refractory intraoperative hypotension in patients who take an angiotensin converting enzyme inhibitor (ACEI) or an angiotensin receptor blocker (ARB). A study at our institution found that discontinuation of ACEI/ARB therapy at least 10 hour before anesthesia was associated with a reduced risk of immediate postinduction hypotension [38]. Therefore, our pre-operative evaluation clinic recommends that patients hold their ACEI and ARB on the day of surgery, provided that their blood pressure is well-controlled. If their hypertension is poorly controlled at the time of their preoperative visit, these medications are continued on the day of surgery. The lipid-lowering agent is generally taken at night, and while postoperative outcomes data are less clear on HF patients, the use of preoperative statins has shown a reduction in postoperative atrial fibrillation and hospital length-of-stay in cardiac surgery patients [39] and therefore can be continued up to surgery.

Specific beta-blockers in HF management are bisoprolol, carvedilol, or sustained-release metoprolol [1]. These medications should be continued on the day of surgery [40,41]. Diuretics should be held on the morning of surgery, primarily because patients are fasting and therefore may be at risk of volume depletion with the use of their diuretic. Aldosterone antagonists (i.e., spironolactone, eplerenone), digoxin, and long-acting nitrates can be continued on the day of surgery.

Pacemaker interrogation: Pacemaker settings vary by patient and device manufacturer. Interrogation of these devices is essential to determine whether a patient’s heart rhythm is pacemaker dependent and if so, the device should be re-programmed to asynchronous demand pacing (e.g., VOO). The device settings and magnet responses should be reviewed with the manufacturer and/or the hospital pacemaker service. If neither is available and the device is a single chamber (right atrium) or dual-chamber (right atrium + right ventricle) pacemaker, a magnet placed on the chest should convert a pacemaker to VOO. Importantly, these responses are not universal, particularly for older devices. This stresses the importance of reviewing all pacemakers prior to surgery.

Stage B patients with asymptomatic ischemic cardiomyopathy, who are at least 40 days after myocardial infarction, and have an LVEF ≤30%, may have an internal cardiac defibrillator (ICD) to prevent sudden cardiac death. Defibrillators are accompanied by either single chamber, dual chamber, or biventricular pacemakers. If a biventricular pacemaker device has an ICD, a magnet should inactivate the defibrillator but may not change the pacemaker settings. To prevent unnecessary shocks the ICD should be inactivated in the holding area. Continuous ECG monitoring is indicated until reactivation.

Stage C patients may have a biventricular pacemaker with or without an implantable defibrillator. Again, magnet placement should deactivate the defibrillator, but will not change the pacemaker settings. For electrical interference, these devices are programmed with a ventricular noise reversion back-up mode, referred to as the “VNR.” This is the automatic default setting when the pacemaker senses electrical noise such as electrocautery. When electrical interference is sensed, the device typically converts to VOO at a rate between 60-90 bpm. These devices should be interrogated postoperatively to confirm the desired settings and reactivation of the ICD.

How should I care for this patient during and after surgery?

Induction and maintenance of anesthesia: An awake arterial catheter prior to induction is helpful for any patient in acute or decompensated HF presenting for emergency surgery, for any HF patient in a higher functional class or stage of HF, and for any HF patient with severe systolic or diastolic dysfunction, pulmonary hypertension, or significant valvular disease. Rapid atrial fibrillation in a HF patient would also indicate arterial line placement, with aggressive attempts to adequately stabilize ventricular rate control prior to surgery.

The choice of medications for induction is less critical when real-time monitoring of heart rate and arterial pressure allow for careful titration of these agents. For example, propofol or thiopental have the potential to cause profound hypotension in the HF patient because of the chronically elevated sympathetic nervous system activity [42,43]. These agents can be given simultaneously with pressor administration. Etomidate and ketamine have less effect on sympathetic-mediated maintenance of blood pressure [42,44]. The sympathomimetic effect of ketamine may increase myocardial oxygen demand, but this can be offset by short-acting beta-blockade. Maintenance of anesthesia in HF patients is generally dependent on the surgical procedure, the expected duration of procedure, blood loss, fluid
shifts, and the need for additional monitoring techniques. **Hemodynamic supportive infusions for perioperative management:** The agents useful for maintaining cardiac performance and blood pressure support are listed in Table 4. Patients with severe systolic dysfunction or dilated cardiomyopathy may develop hypotension secondary to low cardiac output. If the patient has adequate intravascular volume, then inotropes (dopamine, dobutamine) should be initiated. If blood pressure is stable, then these patients may benefit from the vasodilatory and inotropic effect of milrinone. Milrinone may be bolused over 10 minutes prior to infusion.

<table>
<thead>
<tr>
<th>Table 4. Intraoperative supportive medications and considerations</th>
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<tr>
<td><strong>Indication / Drug</strong></td>
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<tr>
<td>Systolic dysfunction (↓ CO + congestion)</td>
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<tr>
<td>Dopamine</td>
</tr>
<tr>
<td>Dobutamine</td>
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<tr>
<td>Milrinone</td>
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<tr>
<td>Diastolic dysfunction (↑ BP + congestion)</td>
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<tr>
<td>Nitroprusside</td>
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<tr>
<td>Nitroglycerin</td>
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<tr>
<td>Volume Overload</td>
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<td>Furosemide</td>
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For patients with diastolic HF, an imbalance in myocardial oxygen supply and demand, particularly in the setting of volume overload and severe hypertension, may lead to deterioration of cardiac function and flash pulmonary edema. Nitroglycerin generally decreases preload by venodilation in order to reduce myocardial oxygen demand, while the arteriodilator effect reduces afterload. Nitroprusside provides potent afterload reduction. Both agents must be titrated with caution as patients with diastolic HF are particularly volume sensitive. With either decompensated systolic or diastolic HF, furosemide is the agent of choice for diuresis. Based on the oral bioavailability (60%), the initial I.V. bolus dose is approximately one-half the daily oral dose, with additional doses titrated to effect. Nesiritide is a recombinant natriuretic peptide that has been shown to improve dyspnea and hemodynamic parameters in patients with decompensated heart failure [45] but no improvement in renal function or mortality [46]. Because the half-life and duration of action of nesiritide is longer than the nitric oxide mediated vasodilators, its use is limited in the perioperative period. **Monitors:** Regardless of surgery, the ACCF/AHA guidelines recommend invasive hemodynamic monitoring in HF patients whose fluid status, perfusion, or systemic or pulmonary vascular resistance is uncertain; whose systolic pressure...
remains low, or is associated with symptoms, despite initial therapy; whose renal function is worsening with therapy; or those who require parenteral vasoactive agents[1]. Because of pathophysiologic relevance of intravascular volume for both systolic and diastolic HF, the most critical aspect of intraoperative and postoperative monitoring for HF patients involves careful fluid management. “Static” variables are those that estimate cardiac filling pressure or cardiac filling volume (as an estimate of end-diastolic volume, or preload) [47]. Central venous catheters are placed for tracking changes in filling pressure but in isolation will not discriminate between changes in ventricular dysfunction and volume overload. The pulmonary arterial catheter estimates changes in left ventricular filling pressure (pulmonary diastolic and wedge pressure) and provides thermodilution-based measures of cardiac output.

Even with minimal user training, transesophageal echocardiography (TEE) provides real-time image estimates of end-diastolic ventricular volume, and global and regional ventricular contractile function [48]. TEE is often considered the monitor of choice to differentiate between cardiogenic, hypovolemic, and vasodilatory causes of shock. The intrathoracic thermodilution method utilizes a central venous line for injection of iced saline, and a thermodilution catheter in a proximal artery (i.e., femoral or axillary) to sense the temperature change from the cold saline injection [49]. The accompanying commercial monitor module estimates stroke volume and intrathoracic blood volume and global end-diastolic volume without dependence on the respiratory cycle.

“Dynamic” variables estimate changes in intravascular volume based on characteristics of the arterial waveform that vary with the respiratory cycle [47]. A regular rhythm is also necessary. During positive pressure ventilation, systolic blood pressure variation is the difference between the maximum and minimum systolic pressure values over the respiratory cycle. Hypovolemia is suggested when systolic pressure variation is greater than 10 mmHg. Similarly, pulse pressure (PP) will vary over the respiratory cycle in accord with hypovolemia.

Pulse pressure variability can also be derived from one respiratory cycle and is calculated as the maximal PP minus the minimal PP, divided by the average of these two pressures, expressed as a percentage. Similarly, stroke volume (SV) varies over the respiratory cycle in accord with hypovolemia. The calculation of SV variability requires a commercial module on the arterial line that analyzes the pulse contour to estimate SV. SV variability is derived from one respiratory cycle and is calculated as the maximal SV minus the minimal SV divided by the average of these two pressures. It is also expressed as a percentage.

Finally, commercial modules exist that determine variability in the amplitude of the pulse oximetry waveform. The variability in pulse oximeter plethysmography waveform (ΔPOP) is calculated as the maximal minus minimal plethysmographic amplitude, divided by the maximal amplitude. This too can be determined over one respiratory cycle.

All of the examples of static and dynamic methods of tracking intravascular volume changes have advantages and disadvantages, including cost and training expertise. Moreover, ventricular systolic and diastolic dysfunction associated with HF may have a confounding effect on these variables. No readily available technique has demonstrated superiority. In this context, one must practice the basics of fluid management, including maintenance fluid requirements, surgery-specific insensible losses, urine output, estimated blood loss, and careful fluid resuscitation.

Postoperative Management: Postoperative planning for HF patients depends on the preoperative functional status, the complexity of the procedure including intravascular fluid shifts, and potential for postoperative acute decompensating HF. Cardiac devices must be interrogated and reactivated when available. Large intraoperative fluid shifts increase the risk for major adverse cardiac events and acute congestion. The entire clinical picture must be considered for postoperative monitoring with a low threshold for intensive care.

In summary, HF is a syndrome with multiple classifications, etiologies, and treatment strategies. Understanding the recently-standardized treatment algorithm will facilitate the preoperative evaluation. Anesthesia providers need to recognize compensated versus decompensated HF in order to guide decision-making. Careful anesthetic planning, device interrogation, fluid management, and postoperative monitoring will optimize perioperative management.

Competing interests
The authors declare that they have no conflict of interests.

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