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Perioperative management of geriatric patients with heart failure

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Heart failure (HF) is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood. In the United States, the prevalence of HF is estimated to be 2.5%, with nearly 6 million people carrying this diagnosis[1]. Both the incidence and the prevalence of HF are increasing. Unfortunately, end-stage HF is expensive to treat and is associated with considerable morbidity and mortality[2]. Strategies for treating end-stage HF aim to limit disease progression, prolong life, and improve quality of life[3].

Coronary artery disease is the underlying casue of HF in approximately 2/3 of patients with left ventricular systolic dysfunction[3]. Nonischemic causes of systolic dysfunction include valvular disease, myocarditis, myocardial toxins [alcohol or chemotherapeutic agents], hypertension, or idiopathic dilated cardiomyopathy. The cardinal symptoms of HF are dyspnea and fatigue, due to inadequate cardiac output and/or elevated left ventricular filling pressure causing pulmonary congestion. Right-sided heart failure is associated with hepatic congestion and peripheral edema.

Several adaptive mechanisms come into play when the cardiac output is inadequate to meet metabolic demands. Initially, the left ventricular end-diastolic volume increases, which causes an improvement in pump function and cardiac output via the Starling mechanism. As the left ventricle dilates and hypertrophies, a change in its geometry occurs, and it becomes more spherical (ventricular remodeling). Sympathetic activation leads to elevation of circulating catecholamine concentrations, with increased heart rate and excessive peripheral vasoconstriction, as well as activation of the renin-angiotensin-aldosterone system, with increased sodium retention. Vasopressin becomes an important vasoconstrictor, since chronic activation of the sympathetic nervous system leads to a vasopressin-deficient state.

Patients with Stage C HF are defined as having current or prior symptoms of HF (e.g., dyspnea, fatigue, and reduced exercise tolerance) associated with underlying structural heart disease[3]. Patients at this stage are treated with drugs, such as angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), aldosterone-receptor blockers, β-blockers, and other agents for symptomatic relief such as digoxin, diuretics, and other vasodilators. Antiarrhythmic agents and other measures, such as dietary salt restriction and possibly treatment with electrophysiological devices, are also commonly utilized[3,5]. In decompensated heart failure, positive inotropes are often used, including agents such as digoxin, dobutamine, milrinone, and calcium sensitizers such as levosimendan. However, with the exceptions of digoxin and possibly levosimendan, positive inotropic substances have been found to be detrimental in the long-term treatment of HF, because they contribute to the development of malignant ventricular tachyarrhythmias and increase the incidence of sudden cardiac death[4]. Therefore, treatment of HF has shifted away from focusing on directly improving myocardial performance and instead emphasizes interruption of the neurohumoral responses that are known to be deleterious. Although such medical management can temporarily ameliorate the symptoms of HF in many patients, the disease itself often progresses into an inexorable downward spiral[2].

Patients classified as having Stage D (end stage) HF are those with advanced structural heart disease who have marked symptoms at rest despite maximal medical therapy and who require specialized intervention. In addition to surgical options, such interventions include continuous (not intermittent) intravenous inotropic infusions and end-of-life strategies, e.g., hospice care[2,3]. Approximately 5-10% of all patients with HF have such end-stage, refractory disease[6].

While the focus of chronic therapy is based upon interruption of the neurohumoral responses, acute management of HF patients, e.g. perioperative management, focuses on maintaining hemodynamic stability and preserving end-organ function. Insertion of an arterial catheter for continuous monitoring of arterial blood pressure and intermittent monitoring of blood gases is wise. Also, insertion of a CCO/SVO_2 pulmonary artery catheter before or after anesthetic induction may be considered to continuously assess PA pressures, cardiac output, and trends in mixed venous oxygen saturation. Transesophageal echocardiography may be particularly useful to define the causes of episodes of hypotension and assess changes in myocardial function.

Patients with severe HF are certainly prone to hypotension during induction of anesthesia due to low ejection fraction, intravascular volume depletion, autonomic dysfunction, and use of ACE inhibitors. Also, even if preload, LV function, and RV function are all adequate, systemic perfusion pressure may be too low due to «vasodilatory shock» causes by ACE inhibitors, amiodarone, side effects from milrinone or dobutamine, arginine vasopressin deficiency, or sepsis. If refractory to standard vasopressor and/or inotropic agents, an infusion of vasopressin (1 – 4 U/hour) or a dose of methylene blue (1.5 – 2.0 mg/kg administered over 20-30 min) may be required[7,8].

Patients with severe HF may have preexisting coagulopathy caused by hepatic congestion associated with significant hemodynamic deterioration, uremia secondary to renal insufficiency, and prior anticoagulant and antiplatelet therapy. If a major surgical procedure is planned, the anesthesiologist should plan for large-bore intravenous or central venous access and a transfusion system suitable for rapidly administering blood products.

Patients with end-stage HF may become candidates for surgical therapy when maximal medical therapy, including treatment with intravenous inotropes, cannot maintain adequate cardiac output to prevent organ dysfunction. Until recently, cardiac transplantation was considered the only definitive treatment for advanced HF. Certainly, cardiac transplantation represents the definitive therapy for terminal HF; it is associated with excellent 1-year survival (> 80%), 5-year survival (60%), and functional capacity[9]. However, whereas > 10,000 patients are on a heart transplant waiting list, fewer than 2,200 donor hearts are available each year[6]. It is this mismatch between the increasing number of potential candidates for cardiac transplantation and the relatively fixed number of donors, as well as the large number of acute HF deaths, that continues to stimulate the search for alternative surgical therapies.

During the last four decades, advances in mechanical circulatory support have expanded the surgical options for treating end-stage HF[10]. In the late 1980s, left ventricular assist devices (LVADs)–which were originally designed in the 1970s as long-term left ventricular replacement devices– became widely used as bridges to transplantation. Furthermore, as the universal waiting period for donor hearts has increased, circulatory assistance has become necessary for longer periods. Increasing clinical experience, the introduction of improved pulsatile LVADs, and the introduction of a new generation of continuous-flow LVADs have created the potential for the use of these devices in patients as an alternative to transplantation[11]. In some cases, long-term LVAD support has resulted in the recovery of native heart function[10,12,13]. Indeed, LVADs may find use as platforms for other therapies for reversing end-stage HF, including pharmacologic therapies meant to enhance reverse remodeling (such as lisinopril, carvedilol, spironolactone, and losartan) [14] or gene-or cell-based therapies[2].

The role of the right ventricle (RV) in cardiovascular disease has been recently reviewed[15,16]. RV function may be impaired in pulmonary hypertension, valvular heart disease, coronary artery disease, and in patients with left-sided HF. The RV has an important role in prognosis after valvular heart surgery, coronary artery bypass surgery, heart transplantation, and left ventricular assist device insertion[17-19]. Severe refractory RV failure requiring prolonged inotropic support or RV assist device insertion occurs in approximately 0.1% of patients after cardiotomy, in 2-3% of patients after heart transplantation, and in 20-30% of patients receiving an LVAD[18]. Recognition of high-risk patients and early management of RV dysfunction, as well as advances in pulmonary vasodilators and RV assist device (RVAD) technology, may decrease the incidence of refractory postoperative RV failure[19].

In summary, as the average age of the population increases, the epidemic of HF can be expected to continue. Therefore, anesthesiologists are now confronted more frequently with patients with advanced HF and with those who have had relatively new and innovative surgical procedures for HF. Although patients with end-stage HF still have a poor prognosis, development of novel therapies for these patients is ongoing.

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