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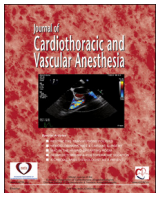

Review Article

Low-Cardiac-Output Syndrome After Cardiac Surgery

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OVER THE PAST DECADE, there has been a significant decline in cardiac surgery–associated mortality, despite an increase in procedural complexity. Although the average perioperative mortality currently is 1% to 2%, the rate of major cardiovascular complications remains high.[1-3] Low-cardiac-output syndrome (LCOS) is the most common and the most serious complication and is associated with increased morbidity, short- and long-term mortality, and healthcare resource utilization.[4,5] This syndrome is characterized by decreased heart pump function, leading to reduced oxygen delivery (DO_2) and subsequent tissue hypoxia.[6] The most common definition of LCOS also includes decreases in the cardiac index (CI) to < 2.0 L/min/m^2 and a systolic blood pressure of < 90 mmHg, in conjunction with signs of tissue hypoperfusion (cold periphery, clammy skin, confusion, oliguria, elevated lactate level) in the absence of hypovolemia. The use of inotropic agents or mechanical circulatory support always is required to improve patient hemodynamics.[7,8]

Acute renal failure, neurologic and pulmonary complications, and atrial fibrillation are the most common consequences of LCOS.[4,9-11] Furthermore, mortality among patients who develop LCOS after cardiac surgery can exceed 20%.[7] Numerous demographic and intraoperative and postoperative factors might be responsible for the development of LCOS.[5,7,12] High-risk cardiac patients, especially those with preoperative left ventricular (LV) systolic dysfunction (left ventricular ejection fraction [LVEF] $< 35\%$), develop LCOS more frequently than do patients with a normal LVEF[13] and must receive special attention during the perioperative period. Thus, prompt identification of LCOS is necessary to enable goal-directed therapy to maximize DO_2 and restore tissue metabolism and organ function, thus improving the clinical outcome.[14]

This review article aims to summarize the current data regarding the pathophysiology, diagnosis, prevention, and treatment of LCOS after cardiac surgery.

Risk Factors and Predictors

To date, several risk factors and predictors have been recognized (Table 1). Furthermore, a number of outcome prediction models have been developed, including the

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Table 1
Predictors and Risk Factors of Postoperative LCOS

Preoperative factors	Age > 65 years[16,17]
	LVEF < 50%[16,17]
	On-pump CABG[17]
	DM and CKD[18,19]
	Malnutrition[20]
Intraoperative factors	CPB duration[16]
	Emergency surgery[17]
	Incomplete revascularization[17]
Laboratory predictors	Hemoglobin[21]
	TLC < 2,000 cells per microliter[22]
	NT-proBNP[23]
	BNP[24,25]
	hFABP[26]

Abbreviations: BNP, brain natriuretic peptide; CABG, coronary artery bypass grafting; CKD, chronic kidney disease; CPB, coronary artery bypass; DM, diabetes mellitus; hFABP, heart fatty acid binding protein; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal of the prohormone brain natriuretic peptide; TLC, total lymphocyte count.

commonly used EuroSCORE, which predicts perioperative cardiovascular alterations.[15] Independent significant risk factors for LCOS, including advanced age (> 65 years), impaired LV function (< 50%), on-pump coronary artery bypass grafting (CABG), emergency surgery or cardiopulmonary bypass (CPB), and incomplete revascularization, have been described.[16,17] Diabetes mellitus and preoperative renal dysfunction are not separate predictors, but in combination they increase the risk of LCOS by 50%.[18,19] Importantly, there have been changes in the recognized risk factors over time. Thus, during the last 20 years, common risk factors such as hypertension, being female, triple-vessel disease, and left main vessel disease are no longer statistically significant, whereas the risk associated with low preoperative ejection fraction has doubled.[7] Another risk factor is malnutrition, which is associated with a 2-fold increase in the probability of postoperative inotropic support and independently predicts adverse clinical outcomes.[20]

Considering that cardiovascular disease, per se, and cardiac surgery with CPB cause profound alterations in systemic metabolism and endocrine function, many trials have been conducted to determine the biochemical predictors of various complications, including LCOS. The predictive impact of low hemoglobin levels was shown in one prospective cohort study.[21] In addition, a preoperative total lymphocyte count of < 2,000 cells/μL was associated with a high incidence of postoperative inotropic support.[22] Preoperative levels of brain natriuretic peptide and the N-terminal of the prohormone brain natriuretic peptide in adult patients undergoing cardiac surgery have been shown to be predictors of prolonged inotropic support, hospitalization, and 30-day mortality.[23,24] Furthermore, another study demonstrated that a brain natriuretic peptide level of > 82 pg/mL at admission to the intensive care unit (ICU) in patients who underwent aortic valve surgery was an independent predictor of postoperative heart failure.[25]

Muehlschlegel et al prospectively studied a cohort of 1,298 patients undergoing primary CABG with CPB to assess the predictive value of heart fatty acid binding protein levels as early markers of perioperative myocardial injury, ventricular dysfunction, and all-cause mortality.[26] In-hospital ventricular dysfunction was defined as a new requirement for the use of 2 or more inotropes or the new placement of an intra-aortic balloon pump (IABP) or ventricular assist device during either the intraoperative period, after the patient was weaned from CPB, or postoperatively in the ICU. After adjusting for clinical predictors of ventricular dysfunction, the peak postoperative heart fatty acid binding protein level (immediately after CPB weaning) was found to be an independent predictor of postoperative ventricular dysfunction.

Pathophysiology

Most interventions that include CPB with cardioplegic arrest lead to myocardial dysfunction, which typically results from ischemic/reperfusion injury of the heart. The persistence of such dysfunctions may vary from temporary (up to 24 hours), for stunning, to persistent, in cases of profound ischemia and myocardial infarction. The contributing factors include preoperative myocardial dysfunction, degree of myocardial protection, systemic inflammatory responses, and alterations in signal transduction systems.[27]

The following pathophysiologic mechanisms of LCOS should be highlighted: (1) LV systolic dysfunction, (2) right ventricular (RV) systolic dysfunction, and (3) diastolic dysfunction, also called heart failure with preserved ejection fraction (Fig 1). The aforementioned mechanisms may occur in isolation or in combination. Conditions such as valvular heart disease, pulmonary hypertension, mechanical valve dysfunction, and respiratory failure, also contribute to LCOS development.

LV Systolic Dysfunction

LV function is derivative of preload, afterload, and contractility; LV systolic dysfunction occurs due to loss of functional myocytes or a decrease in their function. In most cases, the loss of functional myocytes develops as a result of necrosis due to impaired coronary circulation and ischemia/reperfusion injury or the less-understood phenomenon of apoptosis. A loss of function of vital myocytes commonly is transient during stunning or may be refractory to reversal with conditions such as infection; tachycardia; cardiac valvular disease; metabolic abnormality (acidosis, hypoglycemia, hypocalcemia); exposure to cardiac toxins; idiopathic dilated cardiomyopathy; and genetic disorders (familial dilated cardiomyopathy, hypertrophic cardiomyopathy, muscular dystrophies).[28] The impairment of cardiac response to preload leads to dramatic decreases in cardiac output (CO) and oxygen delivery to other organs, increased left atrial pressure and capillary wedge pressure, and cardiogenic pulmonary edema. Although the LV usually works against relatively high systemic arterial pressure, the significant afterload increase also may induce LV systolic dysfunction.