Introduction

Stem cell therapy is another potential treatment of heart failure. Stem cell therapy has shown promise in the treatment of ischemic heart disease both in the laboratory and in small clinical studies. Autologous bone marrow and peripheral blood stem cells transplanted in patients with acute myocardial infarction improved cardiac function. However, until double-blind, randomized controlled trials are performed, the true bene t of this innovative treatment remains unknown. Patients with chronic heart failure, despite good medical management, may experience episodes of pulmonary edema or other signs of acute volume overload. ese patients may require hospitalization for intensive management if diuretics fail to relieve their symptoms. Other patients may experience exacerbations of heart failure associated with acute myocardial ischemia or infarction, worsening valvular dysfunction, infections, or failure to maintain an established drug regimen [1]. Fonarow and associates described a risk strati cation system for in-hospital mortality in acutely decompensated heart failure using data from a national registry. Low, intermediate, and high-risk patients with mortality were identi ed using blood urea nitrogen, creatinine, and systolic BP on admission. ese patients will require all the standard medications, as outlined in previous sections, and may also require infusions of vasodilators or positive inotropic drugs. Intravenous vasodilators have long been used to treat the symptoms of low CO in patients with decompensated chronic heart failure. In general, vasodilators reduce ventricular lling pressures and SVR while increasing SV and CO. NTG is commonly used for this purpose and has been studied in numerous clinical trials. It is o en initially e ective at relatively small doses but frequently requires progressively increasing doses to counteract tachyphylaxis [2]. NTG is associated with dose-dependent arterial hypotension. Brain natriuretic peptide is a acid peptide that is mainly secreted from the cardiac ventricles. Physiologically, BNP functions as a natriuretic and diuretic. It also

heart failure, but only in selected patients. When drug treatment proves unsuccessful, heart failure patients may require invasive therapy, including ventricular assist devices, biventricular pacing, coronary artery by-pass with or without surgical remodeling, or even cardiac orthotopic transplantation. Acute heart failure is a frequent concern of the cardiac anesthesiologist, particularly at the time of separation from cardiopulmonary bypass. e new onset of ventricular dysfunction and a low CO state a er aortic clamping and reperfusion is a condition with more pathophysiologic similarity to cardiogenic shock than to chronic heart failure and is typically treated with positive inotropic drugs, vasopressors, if needed, and/or mechanical assistance [5]. e latter more commonly takes the form of intra-aortic balloon counter-pulsation and less commonly includes one of the several available ventricular assist devices. Most patients undergoing cardiac surgery with CPB experience a temporary decline in ventricular function, with a recovery to normal function in a period of roughly a day. us, pathophysiologic explanations must acknowledge the temporary nature of the low-output syndrome a er CPB. Most likely, this results from one of three processes, all related to inadequate oxygen delivery to the myocardium: acute ischemia, hibernation, or stunning [6]. All three processes would be expected to improve with adequate revascularization and moderate doses of positive inotropic drugs, consistent with the typical progress of the cardiac surgery patient. All three processes would be expected to be more troublesome in patients with pre-existing chronic heart failure, pulmonary hypertension, or arrhythmias. e need for inotropic drug support a er CPB can o en be anticipated based on data available in the preoperative medical history, physical examination, and imaging studies. In a series of consecutive patients undergoing elective CABG, it was observed that increasing age, decreasing le ventricular ejection fraction, female sex, cardiac enlargement, and prolonged duration of CPB were all associated with an increased likelihood that the patient would be receiving positive inotropic drugs on arrival in the intensive care unit. Similarly, in a study of patients undergoing cardiac valve surgery, it was found that increasing age, reduced le ventricular ejection fraction, and the presence of CAD all increased the likelihood that a patient would receive positive inotropic drug support [7]. Whereas all positive inotropic drugs increase the strength of contraction in non-infarcted myocardium, mechanisms of action di er. ese drugs can be divided into those that increase cyclic adenosine monophosphate for their mechanisms of action and those that do not. e agents that do not depend on cAMP form a diverse group, including cardiac glycosides, calcium salts, calcium sensitizers, and thyroid hormone. In contrast to chronic heart failure, cardiac glycosides are not used for this indication, owing to their limited e cacy and narrow margin of safety. Calcium salts continue to be administered for ionized hypocalcemia and hyperkalemia, which are common occurrences during and a er cardiac surgery. Increased Ca2+ in bu er solutions bathing cardiac muscle in vitro unquestionably increase inotropy. Calcium sensitizers, speci cally levosimendan, function by binding to troponin C in a calcium-dependent fashion. us, levosimendan does not impair diastolic function because its a nity for troponin C declines with Ca during diastole. Although several reports have described the successful use of levosimendan in patients recovering from CABG, clinical experience with this agent remains limited and there is no consensus as to how and when this agent should be used, relative to other, better established agents. Intravenous thyroid hormone has been studied extensively as a positive inotrope in cardiac surgery. ere are multiple studies supporting the existence of euthyroid sick syndrome with persistently reduced concentrations in blood a er cardiac surgery in both children and adults [8]. ere are also data suggesting that a er ischemia and reperfusion, T 3 increases inotropy faster than and as potently as isoproterenol. Nevertheless, randomized controlled clinical trials have failed to show e cacy of T3 a er CABG. e cAMP-dependent agents form the mainstays of positive inotropic drug therapy a er cardiac surgery. ere are two main classes of agents: the phosphodiesterase inhibitors and the -adrenergic receptor agonists. ere are many di erent phosphodiesterase inhibitors in clinical use around the world, including enoximone, inamrinone, milrinone, olprinone, and piroximone [9]. Comparisons among the agents have failed to demonstrate important hemodynamic di erences. Reported di erences relate to pharmacokinetics and rare side e ects, typically observed with chronic oral administrations during clinical trials. All members of the class produce rapid increases in contractile function and CO and decreases in SVR. e e ect on BP is variable, depending on the pre-treatment state of hydration and hemodynamics; nevertheless, the typical response is a small decrease in BP. ere is either no e ect on HR or a small increase. Inamrinone and milrinone have been shown to be e ective, rst-line agents in patients with reduced preoperative le ventricular function. Milrinone, the most commonly used member of the class, is most o en dosed at a loading dose and maintenance infusion. It is o en given in combination with a -adrenergic receptor agonist. Among the many -adrenergic receptor agonists, the agents most o en given to patients recovering from cardiac surgery are dopamine, dobutamine, and epinephrine. Dopamine has long been assumed to have dose-de ned receptor speci- city. At small doses, it is assumed to have an e ect mostly on dopaminergic receptors. At intermediate doses, -adrenergic e ects are said to predominate; and at doses of -adrenergic receptor e ects predominate. Nevertheless, the relationship between dose and blood concentration is poorly predictable. Dopamine is a relatively weak inotrope that has a predominant e ect on HR rather than on SV. Dobutamine is a selective adrenergic receptor agonist. Most studies suggest that it causes less tachycardia and hypotension than isoproterenol [10]. It has been frequently compared with dopamine, where dobutamine s greater tendency for pulmonary and systemic vasodilation is evident. Dobutamine has a predominant e ect on HR, compared with SV, and as the dose is increased more than there are further increases in HR without changes in SV. Epinephrine is a powerful adrenergic agonist, and, like dopamine, demonstrates di ering e ects depending on the dose. At small doses, despite an almost pure -adrenergic receptor stimulus, there is almost no increase in HR. Clinicians have long assumed that epinephrine increases HR more than dobutamine administered at comparable doses. Nevertheless, in patients recovering from cardiac surgery, the opposite is true: dobutamine increases HR more than epinephrine. Other -adrenergic agonists are used in speci c circumstances. For example, isopro-terenol is o en used a er cardiac transplantation to exploit its powerful chronotropy and a er correction of congenital heart defects to exploit its pulmonary vasodilatory e ects. Norepinephrine is exploited to counteract profound vasodilation

Conclusion

Capsaicin, oil of camphor, Curcumin, cod liver oil and green tea have been used as natural/herbal analgesics and anti-in ammatory agents..

Acknowledgement

None

Con ict of Interest

None

References

1. Stevenson M, Wickline A (2020) 23-hour TKA in 10 opioid pills or less through 90 days: A non-selected prospective consecutive one year cohort.