Journal of Pain & Relief
ISSN: 2167-0846
Francesco, J Pain Relief 2022, 11:11
Mini Review
Open Access

Acute Therapy Management of Chronic Heart

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Abstract

Among the promising non pharmacologic therapies for the management of heart failure are the implantable defibrillators and biventricular pacemakers. In the companion trial, cardiac resynchronization therapy with a pacemaker combined with an implantable defibrillator significantly decreased the likelihood of death from or hospitalization for heart failure when compared with conventional pharmacologic therapy.

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 benefit 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. These 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 stratification 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 identified using blood urea nitrogen, creatinine, and systolic BP on admission. These 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 filling 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 often initially effective 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 serves as a counter regulatory hormone to Ang II, norepinephrine, and endothelin by decreasing the synthesis of these agents and by direct vasodilation. As the clinical severity of heart failure increases, the concentrations of BNP in blood also increase. As a result, measurements of BNP in blood have been used to evaluate new onset of dyspnea. BNP concentrations in blood increase with decreasing left ventricular ejection fraction; therefore, measurements of this mediator have been used to estimate prognosis. BNP concentrations decline in response to therapy with ACE inhibitors, Ang II antagonists, and aldosterone antagonists.

Discussion

In addition, recombinant BNP has been released as a drug indicated for patients with acute heart failure and dyspnea with minimal activity. Nesiritide produces arterial and venous dilatation through increasing cGMP. Nesiritide does not increase HR and has no effect on cardiac inotropy. It has a rapid onset of action and a short elimination half-life. In clinical studies, loading doses have maintenance doses. Studies have shown that nesiritide reduces symptoms of acute decompensated heart failure similarly to NTG, with-out development of acute tolerance. Patients receiving nesiritide experienced fewer adverse events than those receiving NTG. However, the mortality rate at 6 months was higher in the patients receiving nesiritide than in the NTG group. Compared with dobutamine, nesiritide was associated with fewer instances of ventricular tachycardia or cardiac arrest. Positive inotropic drugs, principally dobutamine or milrinone, have long been used to treat decompensated heart failure, despite the lack of data showing an outcome benefit to their use. In the past, some chronic heart failure patients would receive intermittent infusions of positive inotropic drugs as part of their maintenance therapy [3]. Small studies consistently demonstrate improved hemodynamic values and reduced symptoms after administration of these agents to patients with heart failure. Studies comparing dobutamine to milrinone for advanced decompensated heart failure showed large differences in drug costs, favoring dobutamine, and only small hemodynamic differences, favoring milrinone. Nevertheless, placebo-controlled studies suggest that there may be no role what-soever for discretionary administration of positive inotropes to patients with chronic heart failure. In one study, 951 hospitalized patients with decompensated chronic heart failure who did not require intravenous inotropic support were assigned to receive infusion of either milrinone or saline. Meanwhile, all patients received ACE inhibitors and diuretics as deemed necessary. Total hospital days did not differ between groups; however, those receiving milrinone were significantly more likely to require intervention for hypotension or to have new atrial arrhythmias [4]. A subanalysis of these results found that patients suffering from ischemic cardiomyopathy were particularly subject to adverse events from milrinone. At the present, positive inotropic drug support can be recommended only when there is no alternative. Thus, dobutamine and milrinone continue to be used to treat low CO in decompensated

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Received: 22-Oct-2022, Manuscript No.JPAR-22-81719; **Editor assigned:** 24-Oct-2022, PreQC No. JPAR-22-81719(PQ); **Reviewed:** 07-Nov-2022, QC No. JPAR-22-81719; **Revised:** 12-Nov-2022, Manuscript No. JPAR-22-81719(R); **Published:** 19-Nov-2022, DOI: 10.4172/2167-0846.1000470

Citation: Francesco (2022) Acute Therapy Management of Chronic Heart. J Pain Relief 11: 470.

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. The new onset of ventricular dysfunction and a low CO state after 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]. The 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. Thus, pathophysiologic explanations must acknowledge the temporary nature of the low-output syndrome after 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. The need for inotropic drug support after CPB can often 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 left 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 left 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 differ. These drugs can be divided into those that increase cyclic adenosine monophosphate for their mechanisms of action and those that do not. The 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 efficacy and narrow margin of safety. Calcium salts continue to be administered for ionized hypocalcemia and hyperkalemia, which are common occurrences during and after cardiac surgery. Increased Ca2+ in buffer solutions bathing cardiac muscle in vitro unquestionably increase inotropy. Calcium sensitizers, specifically levosimendan, function by binding to troponin C in a calcium-dependent fashion. Thus, levosimendan does not impair diastolic function because its affinity 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. There are multiple studies supporting the existence of euthyroid sick syndrome with persistent reduced concentrations in blood after cardiac surgery in both children and adults [8]. There are also data suggesting that after ischemia and reperfusion, T 3 increases inotropy faster than and as potently as isoproterenol. Nevertheless, randomized controlled clinical trials have failed to show efficacy of T3 after CABG. The cAMP-dependent agents form the mainstays of positive inotropic drug therapy after cardiac surgery. There are two main classes of agents: the phosphodiesterase inhibitors and the β-adrenergic receptor agonists. There are many different 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 differences. Reported differences relate to pharmacokinetics and rare side effects, 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. The effect on BP is variable, depending on the pre-treatment state of hydration and hemodynamics; nevertheless, the typical response is a small decrease in BP. There is either no effect on HR or a small increase. Inamrinone and milrinone have been shown to be effective, first-line agents in patients with reduced preoperative left ventricular function. Milrinone, the most commonly used member of the class, is most often dosed at a loading dose and maintenance infusion. It is often given in combination with a β-adrenergic receptor agonist. Among the many β-adrenergic receptor agonists, the agents most often given to patients recovering from cardiac surgery are dopamine, dobutamine, and epinephrine. Dopamine has long been assumed to have dose-defined receptor speci-ficity. At small doses, it is assumed to have an effect mostly on dopaminergic receptors. At intermediate doses, β-adrenergic effects are said to predominate; and at doses of α-adrenergic receptor effects predominate. Nevertheless, the relationship between dose and blood concentration is poorly predictable. Dopamine is a relatively weak inotrope that has a predominant effect on HR rather than on SV. Dobutamine is a selective β-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 effect 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 differing effects depending on the dose. At small doses, despite an almost pure β-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 β-adrenergic agonists are used in specific circumstances. For example, isopro-terenol is often used after cardiac transplantation to exploit its powerful chronotropy and after correction of congenital heart defects to exploit its pulmonary vasodilatory effects. 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-inflammatory agents..

Acknowledgement

None

Conflict of Interest

None

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