MINI REVIEW

Experimental heart failure

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ABSTRACT

More than 150 years ago, vagus nerve stimulation was tried out for the first time in an experimental setting. In the 1980's and 1990's, vagus nerve stimulation was demonstrated to have significant antiarrhythmic effects in both conscious and anaesthetized animals, especially during acute myocardial ischemia. Chronic Heart Failure (CHF) is characterized by autonomic dysregulation, which is marked by a persistent rise in sympathetic drive and a decline in parasympathetic activity. Poor long-term outcomes in HF patients are predicted by sympathetic overdrive and elevated heart rate. The widespread use of selective and non-selective beta-adrenergic receptor blockers is the classic example of pharmacologic medicines that partially reduce sympathetic activity being used as an effective long-term therapy for patients with HF. In contrast, despite its complicated cardiovascular effects, modulating parasympathetic activity as a potential treatment for HF has gotten little study over the years. In this article, we examine the outcomes of recent experimental animal experiments that suggest the potential use of electrical Vagus Nerve Stimulation (VNS) as a long-term therapy for the management of chronic Heart Failure (HF). The review will examine the effects of chronic VNS on Left Ventricular (LV) function as well as the impact of VNS on cytokine production and nitric oxide generation as potential modifiers of the HF state. Finally, we will briefly examine several nerve stimulation techniques that are also being researched as prospective therapeutic methods for the management of chronic HF.

Keywords: Ventricular function; Heart failure; Autonomic imbalance

INTRODUCTION

Heart valve disease, which can be brought on by a variety of conditions such as rheumatic fever and congenital abnormalities, continues to be a substantial cause of morbidity and mortality [1]. Currently, valve replacement is the main method of treatment for aortic valve disease. Although congestive Heart Failure (HF) treatment has evolved significantly in recent years, particularly with the concurrent prescribing of ACE inhibitors, ARBs, beta-blockers, and AAT antagonists, mortality is still high due to various circumstances [2-5]. Treatment for a failing heart should target the underlying illness, alleviate incapacitating symptoms, and focus on methods to stop ventricular remodeling. A very effective non-medical strategy using biventricular pacing was introduced to the toolbox for treating Heart Failure (HF) with cardiac resynchronization therapy, however it is only applicable to patients with a large QRS complex and mechanical dyssynchrony. The current work further emphasises the requirement for exact constitutive models and careful experimentation when simulating BHV function and design [6-9]. Increased sympathetic activity, decreased parasympathetic tone, or both are known to cause life threatening ventricular tachyarrhythmias, which are intimately related to heart failure. In a model with healed myocardial infarction, exercise testing, and intermittent ischemia, experimental animal findings in conscious dogs convincingly show that raising vagal tone with right Vagus Nerve Stimulation (VNS) can prevent ventricular tachyarrhythmias [10-13].

LITERATURE REVIEW

Observations on vagus nerve stimulation

In 2004, a report on the first significant animal model of VNS in HF was published. Rats with anterior myocardial infarction and HF were randomly assigned to either VNS or sham stimulation. With stimulus intensity capable of lowering HR by 20-30 bpm, stimulation was conducted for 10 seconds per minute [14]. After 140 days, there was a noticeable improvement in left ventricular function and a drop in mortality from 50% to 14% compared to untreated animals. In a dog model of micro embolization induced HF, increased left ventricular function with VNS had similar favorable outcomes. Since both the treated group and the control group were continuously paced at the same rate, heart rate reduction was not a factor in this model. With their micro embolization HF model, Sabbah et al. revealed significant findings [15-17]. Left ventricular function was enhanced by low intensity vagus stimulation with no change in heart rate, and detrimental HF biomarkers were significantly reduced. Patients with structural heart disease, ischemic and non-ischemic cardiomyopathy, diminished LVEF, and clinical signs of advanced HF were chosen for the first VNS in humans (NYHA class II–III). Patients had to be on stable, effective medication and in sinus rhythm with a resting heart rate between 60 and 110 bpm. The vital physiological function of autonomic balance for the heart and the potential to correct its imbalance by raising vagus tone with electrical stimulation have both been well established, especially for patients with HF. However, better arrhythmia management is still not possible at this time [18-20].

A completely new area of study and therapeutic application has been made possible by the non-pharmacologic therapy of HF as an "addon" method. Cardiac resynchronization therapy has a lot of experience and produces great results. Cardiac contractility modulation has been developed for the treatment of HF, although it has not yet found a wide ranging application. Another viable electrical treatment for drug resistant hypertension appears to be carotid baroreceptor stimulation. The spinal cord stimulation is a different location to promote parasympathetic activity to have a positive effect on HF and/or ventricular arrhythmias. Although studies utilizing this approach have begun, there are currently no known outcomes for HF patients [21-23].

Statistical evaluation: The combined analysis of the two stages of the trial was reported as the major result because safety was the study's key endpoint, which was the presence of all system and procedure related adverse effects. Separate results on the second group of patients (n 14 24) are also presented with regard to effectiveness. The report includes all of the 1 year follow-up data that are currently accessible. For normal and non-normal distributions, data are reported as mean+SD or median and interquartile range, respectively. Data were analyzed using repeated measures ANOVA, followed by a Bonferroni multiple comparison test, or Kruskal-Wallis ANOVA, followed by a Wilcoxon signed rank test, depending on the variables that were examined at baseline, three, and six months after the follow-up. With

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the appropriate paired t-test or Mann-Whitney test, one year's worth of data were compared to baseline data.

Side effects and safety: During the course of the trial, three patients including one of the two class IV patients died. The authors believed that these mortality events and bouts of abrupt cardiac decompensation were predicted given the severity of HF and were unrelated to the investigational device. It can be challenging to say with certainty that a clinical state is unrelated to the investigational technique, as is the case when trials are conducted on chronic patients with severe diseases. Despite this broad consideration, the results indicate that safety was good without any significant AEs that could be definitively linked to the device. Higher stimulation amplitudes were frequently hindered by the existence of local side effects, such as dysphonia, but overall these AEs were anticipated given the extensive experience with epileptic patients.

DISCUSSION

The mechanism of VNS's positive effect is the most crucial unanswered question. Reduced heart rate may be significant and is indicative of the antiadrenergic effects of elevated vagus tone. Recent experimental data and the clinical results of the initial use in patients, however, show that the beneficial effect is still measurable even in the absence of a clinically meaningful HR drop. The increased parasympathetic activity is evidently there from the improved HR variability. VNS's acute effectiveness is difficult to evaluate, making it difficult to forecast how the body will react.

CONCLUSION

The mechanism of VNS's positive effect is the most crucial unanswered question. Reduced heart rate may be significant and is indicative of the antiadrenergic effects of elevated vagus tone. Recent experimental data and the clinical results of the initial use in patients, however, show that the beneficial effect is still measurable even in the absence of a clinically meaningful HR drop. The increased parasympathetic activity is evidently there from the improved HR variability. VNS's acute effectiveness is difficult to evaluate, making it difficult to forecast how the body will react. The current results demonstrate that continuous VNS in symptomatic CHF patients and LV systolic dysfunction may be safe and tolerated, but caution must be exercised when evaluating a small non-controlled clinical trial. At a 1 year follow-up, this innovative therapeutic strategy enhanced LVEF, lowered LV systolic volume, and improved quality of life.

To determine whether vagus nerve stimulation can actually function as a novel nonpharmacological strategy for the treatment of symptomatic HF, it should be put to the test in a controlled clinical trial.

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