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Vasovagal syncope-role of closed loop stimulation pacing[★]

Mohammed Ruzieh*, Blair P. Grubb

Penn State Heart and Vascular Institute, 500 University Drive, PO Box 850, MC H047, Hershey, PA 17033, United States

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ABSTRACT

The benefit of conventional pacing in vasovagal syncope remains controversial and is currently recommended for patients with recurrent syncope and documented asystole. In the last two decades, a growing body of evidence has emerged supporting the use of a new sensing technique called closed loop stimulation or CLS, to treat refractory vasovagal syncope. CLS uses a sensing algorithm that can detect variation in cardiac contractility and respond to drop in blood pressure by increasing the heart rate. Multiple observational and randomized studies have assessed its efficacy and showed its superiority to conventional pacing in reducing the burden of syncopal attacks in patients with cardio-inhibitory vasovagal syncope.

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Contents

Introduction
Rationale and evidence of closed loop stimulation (CLS) in vasovagal syncope
Retrospective cohort studies
Prospective non-randomized studies
Randomized trials of CLS pacing vs. conventional pacing
In summary
Who should get DDD-CLS pacemaker
Conclusion
Acknowledgment
References

Introduction

Syncope is defined by transient loss of consciousness with spontaneous and rapid recovery [1]. The most common form of syncope is vasovagal (VVS) or neuro-cardiogenic syncope, accounting for at least 20% of the cases, and generally has a benign prognosis [2]. The mechanism behind VVS involves vasodilation and/or bradycardia or asystole resulting in hypotension and decreased cerebral perfusion leading to transient loss of consciousness [3–5]. Based on response to head-up tilt table test (HUTT), vasovagal (VVS) can be further divided into mixed, cardio-inhibitory or vasodepressor types [6,7] (Table 1).

The initial treatment strategy for VVS is conservative and focuses on life style modifications and avoidance of triggers that could precipitate syncope. When this fails, medical therapy is usu-

E-mail address: mruzieh@pennstatehealth.psu.edu (M. Ruzieh).

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ally the next step [8]. However, despite medical therapy, many patients remain symptomatic, and pacemakers were investigated as a treatment option for recurrent VVS when associated with asystole or bradycardia (type II response on HUTT). Clinical trials for pacemakers in VVS are conflicting, and it is currently given a Class IIb recommendation (Level of Evidence: C) [8].

Recently, a new sensing and pacing algorithm that responds to change in cardiac contractility has emerged, known as dual-chamber pacing with closed loop stimulation [DDD-CLS]). Multiple observational and randomized trials have investigated CLS pacing and found it superior to conventional pacing in patients with refractory cardio-inhibitory VVS.

In this review, we describe the available literature on the use of CLS pacing in patients with refractory cardio-inhibitory VVS.

Rationale and evidence of closed loop stimulation (CLS) in vasovagal syncope

In patients with VVS syncope, an increase in cardiac contractility may precede fall in blood pressure and heart rate [9,10], and

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^{*} Corresponding author.

Table 1 Classification of VVS based on response to HUTT.

Type of response	Heart rate response	Blood pressure response
Mixed (Type I) Cardio-inhibitory (Type II)	Heart rate falls < 40 bpm for < 10 s, with or without asystole for < 3 s Type IIa: Heart rate falls < 40 bpm for > 10 s without asystole or with asystole for < 3 s Type IIb: Asystole > 3 s	Blood pressure falls; before or at the time of bradycardia or a systole
Vasodepressor	Constant heart rate or drop up to 10% of its peak	

VVS: vasovagal syncope; HUTT: head-up tilt table test; bpm: beats per minutes.

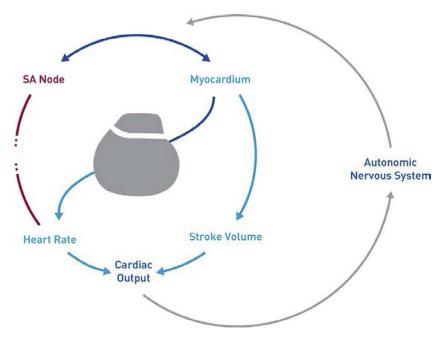


Fig. 1. CLS monitors myocardial contraction dynamics to regulate heart rate. Decreased right ventricular filling (such as in the early stages of syncope) leads to increase lead impedance that triggers CLS algorithm.

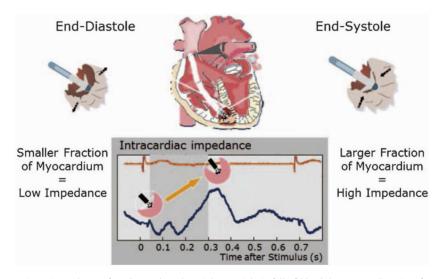


Fig. 2. The lead impedance changes in various phases of cardiac cycle. When right ventricle is full of blood, less myocardium interferes with the lead tip and thus the impedance is low. The opposite happens when there is less blood the right ventricle.

in some instances, bradycardia is not the cause syncope but rather follows it due to a vasovagal reflex effect. Thus, Conventional pacing systems would increase heart rate too late in the episode and be less likely to fully prevent it.

Closed Loop Stimulation (CLS) by Biotronik Inc (Biotronic, Germany) is a sensing strategy that tracks variation in myocardial contractility by measuring localized cardiac impedance between

the right ventricular lead and the device [11] (Fig. 1). At rest, impedance measurements are collected to create a baseline wave form. When there is reduced right ventricular filling (such as in the early stages of syncope), a greater fraction of the myocardium interferes with the lead tip, increasing its impedance (Fig. 2). The increase in lead impedances triggers CLS algorithm and responds by increasing pacing rate, even before the onset of bradycardia or

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