



Gastric Dysrhythmia Corrected by Dual Chamber Pacemaker Implantation

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Highlights

Nausea has been defined as an “unpleasant painless subjective feeling that one will imminently vomit”. While nausea and vomiting are often thought to exist on a temporal continuum, this is not always the case. There are situations when severe nausea may be present without emesis and less frequently, when emesis may be present without preceding nausea. The underlying mechanisms involved in nausea are complex and encompass psychological states, the central nervous system, autonomic nervous system, gastric dysrhythmias, and the endocrine system. We report a 92-year-old male patient with depression, hypothyroidism, and intermittent severe sinus dysfunction, causing nausea, vomiting, and fatigue, pre-syncope and low cerebral output. Dual chamber pacemaker implantation was performed and 24 hours after the implantation of the device all the symptoms disappeared, the parameters of the pacemaker were stable, and the patient was discharged. At the 1st and the 3rd month after implantation the patient remained asymptomatic. Over-activity of autonomic outflow may be a determinant for overall nausea intensity, probably, may be a potential therapeutic target to be corrected, at least in part, by a dual chamber pacemaker implantation.

Keywords: Autonomic nervous system; central nervous system; gastric dysrhythmias; pacemaker; vomiting.

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Introduction

Nausea has been defined as an “unpleasant painless subjective feeling that one will imminently vomit” [1]. While nausea and vomiting are often thought to exist on a temporal continuum, this is not always the case. There are situations when severe nausea may be present without emesis and less frequently, when emesis may be present without preceding nausea. Most individuals report that nausea is more common, more disabling, feels worse and lasts longer than vomiting [2]. Despite this, there is a clear understanding of the mechanisms underlying nausea; much of which is about nausea concomitant with emesis. The underlying mechanisms involved in nausea are complex and encompass psychological states, the central nervous system, autonomic nervous system, gastric dysrhythmias, and the endocrine system.

Characteristic physiological changes (sweating, pallor, salivation, increase in blood pressure, tachycardia, cutaneous vasoconstriction, decreased gastrointestinal motility) occurring before vomiting are mediated by the autonomic nervous system (ANS) and are well described [3]. Afferent signaling arises from vagal inputs, reflecting mechanical or chemical stimuli [4]. La

Count and colleagues have shown that bursts of cardiovagal modulation precedes transition to a higher level of nausea, perhaps by prompting interoceptive re-evaluation by the subject culminating in rating nausea at a higher level [5]. This autonomic outflow during nausea is likely modulated by the central nervous system (CNS). While some areas of the brain, such as the insula appear to be modulating both sympathetic as well as parasympathetic responses, there also appears to be a divergent central control for autonomic response of nausea [6]. Thus, ANS outflow and the CNS network controlling it could be determinant of overall nausea intensity, and understanding them in more detail could be of therapeutic importance.

In this case we report a 92-year-old male patient with depression, hypothyroidism, and scrotal hernia with surgical indication. However, surgery was contraindicated due to intermittent severe sinus dysfunction, causing nausea, vomiting, and fatigue, pre-syncope and low cerebral output. He was taking cilostazol 50mg twice a day, acetylsalicylic acid 100mg daily, levothyroxine sodium 50µg daily and citalopram 20mg daily. The ECG showed sinus bradycardia, divisional anterior-superior block, intra and interatrial conduction disorder, and right bundle branch

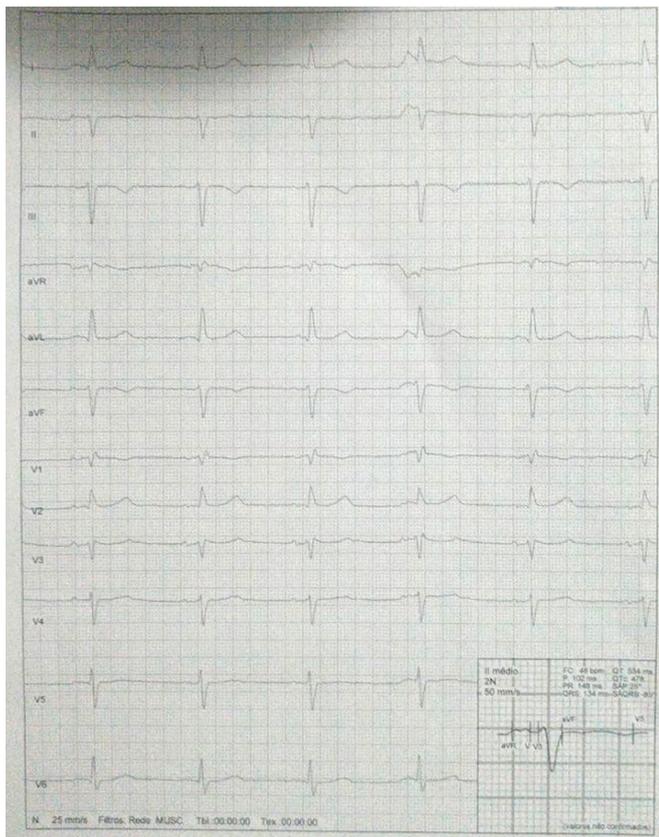


Figure 1. The ECG showing sinus bradycardia, divisional anterior-superior block, intra and interatrial conduction disorder, and right bundle branch block.

block (Figure 1). All the other exams, such as transthoracic echocardiogram, blood and urine tests, chest radiography, upper gastrointestinal endoscopy, abdominal ultrasonography, and cranial magnetic resonance imaging were normal. According to the current Brazilian Guidelines for Implantable Electronic Cardiac Devices of the Department of Artificial Cardiac Pacing, there was indication for a DDDR pacemaker implant [7].

The device implantation was performed; the rate-adaptive function was activated and programmed with a lower rate of 60 bpm and an upper rate of 120 bpm. We also programmed the paced atrioventricular interval to 140 to 220 ms and activated the AV delay management algorithm that automatically searches for intrinsic conduction to prevent unnecessary right ventricular pacing in these cases of sinus node disease. The auto mode switching function was activated. Auto mode switching occurred when the atrial rate exceeded 170 to 180 bpm for a specific number of beats or period of time. The atrial tachycardia/AF diagnostic suite provided detailed historical data, allowing us to identify and evaluate therapy for improved management of patients. Atrial sensitivity was programmed to 0.5 mV. Twenty-four hours after the implantation of the device all the symptoms disappeared, the parameters of the pacemaker were stable, and the patient was discharged. During the evaluation at the 1st and the 3rd month after the implantation the patient remained asymptomatic.

In conclusion, over activity of ANS outflow and resulting CNS network effects, leading to a severe sinus dysfunction that

appeared to be determinant for severe nausea, may be a potential therapeutic target to be corrected, at least in part, by a dual chamber pacemaker implantation.

List of abbreviations

ANS - autonomic nervous system
CNS - central nervous system
DDDR pacemaker - dual chamber pacemaker
ECG - electrocardiogram

Declarations of Interest

The author declare no conflicts of interest.

Acknowledgements

The authors state that they abide by the "Requirements for Ethical Publishing in Biomedical Journals" [8].

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