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Case Report

A profound case of neurally mediated syncope with asystole after septoplasty☆

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Abstract Vasovagal syncope (VVS) is an alarming yet benign condition that may present postoperatively for the first time in otherwise healthy patients. Although VVS is associated anecdotally with nasal manipulation, no data have been found to quantify this incidence with otolaryngology surgeries. We present a case of profound, recurrent syncope and documented asystole with an initial diagnosis of glossopharyngeal neuralgia. We conclude with a discussion of neurally mediated syncope particular to the perioperative setting. It is essential to recognize neurocardiogenic etiology to differentiate it from other more concerning causes of syncope and asystole.
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1. Introduction

It has been well established that stimulation of the vagus nerve causes bradycardia, syncope, and asystole. Although the overall incidence of postoperative syncopal events is unknown, vasovagal reactions occur in approximately 2% of patients undergoing dental procedures [1]. Vasovagal syncope (VVS), also referred to as neurocardiogenic syncope [1-4], is one of the subtypes of neurally mediated (reflex) syncopes [1,4,5]. Vasovagal syncope is the most common cause of loss of consciousness in apparently normal patients [1,6,7]. Asystole is associated with syncopal events 50% of the time [4] and may be especially concerning in the perioperative setting; however, no specific definition (eg, duration) of asystole exists [8]. Studies using the controlled environment of tilt table testing have shown that the length of asystole associated with syncope varies from three to 34 seconds [9-16]. However, despite considerable study of VVS, its pathophysiology remains to be fully elucidated [1].

Although nasal manipulation and surgery are associated with VVS, little information exists in the literature. A case of immediate postoperative syncope and asystole following septoplasty surgery is presented. The patient showed multiple episodes of syncope and documented asystole that persisted for several days despite medical treatment and the discontinuation of suspected triggers.

2. Case report

A 25 year old, ASA physical status 1 man presented to the Ear, Nose and Throat (ENT) Clinic for evaluation of worsening bilateral nasal obstruction. Daily intranasal

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steroid and saline irrigation failed to improve his symptoms. He had no known allergies; he denied smoking and use of illicit drugs or ethanol; and he was not taking any other medications. Physical examination was notable for septal deviation to the left and bilateral turbinate hypertrophy. Based on his history, examination, and failed medical management, surgery was recommended.

The patient underwent a routine uncomplicated septoplasty and bilateral inferior turbinoplasty with coblation and outfracturing. Anesthesia consisted of midazolam preoperatively, fentanyl, propofol, and succinylcholine for induction and endotracheal intubation, and maintenance with sevoflurane. Doyle splints were placed and a drip pad was placed under the patient's nose at the end of surgery. Estimated blood loss was less than 50 mL, and the patient received 1,200 mL of Ringer's Lactate during the 53-minute surgery. He was extubated and transported to the Postanesthesia Care Unit (PACU) in stable condition and receiving 6 L of oxygen via simple mask.

Approximately 45 minutes after the end of the surgery and while in the PACU, the patient lost consciousness; telemetry showed asystole for approximately 15 seconds with concurrent absence of palpable peripheral pulses. This episode resolved before initiation of ACLS protocol (Fig. 1A). No seizure-like activity was noted. The anesthesiologist was called; a 12-lead electrocardiogram (ECG) showed sinus rhythm with mildly prolonged QT intervals. The patient denied specific complaints following the episode.

Approximately 2 1/2 hours later, and while the anesthesiologist was at the bedside, the patient said, "I am not feeling good." Telemetry showed sinus bradycardia at 40 bpm. The anesthesiologist administered 0.5 mg of intravenous (IV) atropine without response. His rhythm progressed to asystole for 20 seconds, which required an additional 0.5 mg of atropine (Fig. 1B). Both events were not associated with any medication dosing. A Cardiology consult was obtained. As a possible vagal reflex from glossopharyngeal nerve stimulation via the nasal packing was suspected, the Doyle splints were removed and the patient was monitored overnight. All laboratory studies were normal except for mildly elevated serum glucose. The patient did well overnight; a repeat ECG was normal and he was discharged home.

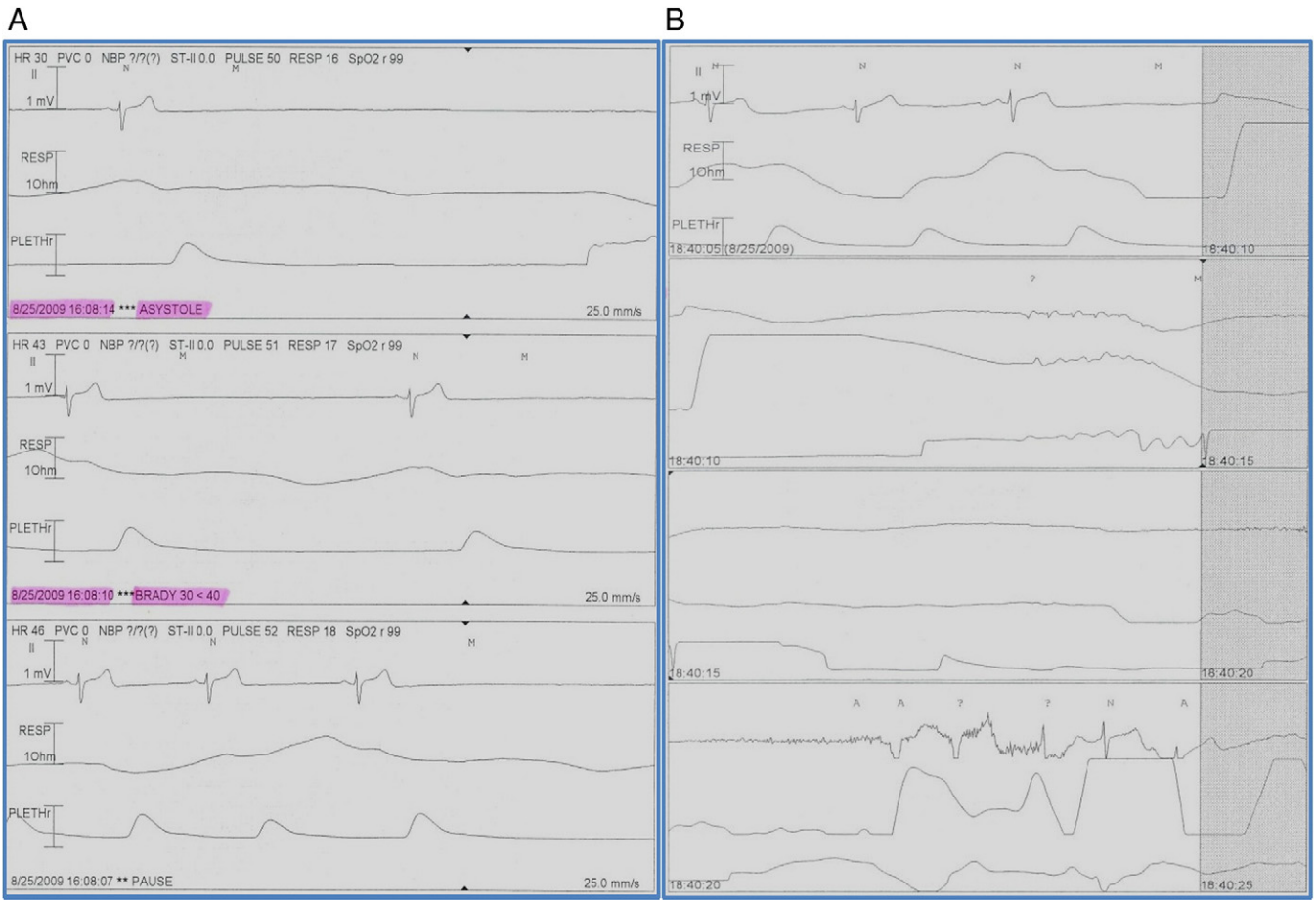

Fig. 1 **A**. Telemetry recording during first syncopal event in the Postanesthesia Care Unit (PACU) lasting approximately 15 seconds. **B**. Telemetry recording of second syncopal event in the PACU lasting approximately 20 seconds. Each strip represents 5 seconds. The three lines from top to bottom are the electrocardiogram, respirations, and plethysmographic waveform.