Pop and Drop

Michael Witcik, MD, FACC; Joshua Meskin, MD, FACC

ABSTRACT
A 70-year-old man presented to cardiology clinic with recurrent syncope associated with consumption of carbonated beverages. Ambulatory monitoring revealed multiple symptomatic ventricular pauses. A barium esophagram was unremarkable, and the patient underwent placement of a dual chamber pacemaker for deglutition syncope with resolution of symptoms. A challenge of carbonated beverage was given, resulting in bradycardia that initiated an appropriate pacing response. This case illustrates the need for a thorough history when defining the etiology of syncope.

INTRODUCTION
Despite advancing medical technology, a thorough history continues to be integral to optimal patient care. This is illustrated by a case of a 70-year-old man who presented an unusual form of recurrent syncope.

CASE PRESENTATION
A 70-year-old man presented to cardiology clinic with recurrent syncope after consumption of cold carbonated beverages. The episodes typically resulted in weakness and flushing; however, one episode was associated with head trauma. The patient reported regaining full consciousness after several seconds without residual symptoms. There was no history of dysphagia or odynophagia. He denied any history of neurological disease. There was no associated chest pain, dyspnea, or palpitations. Past medical history was significant for coronary artery bypass surgery and rate-controlled atrial fibrillation. Family history was unremarkable for any arrhythmias, and social history was noncontributory.

On examination, the patient appeared well. He was afebrile and normotensive with normal heart and respiratory rates. Cardiac auscultation revealed an irregular rhythm without a significant murmur or gallop. No jugular vein distension was present. Pulmonary examination revealed clear lung fields. The rest of the examination, including abdominal and neurological exam, was normal. Laboratory evaluation was unremarkable. The chest radiograph did not reveal any cardiopulmonary abnormality. The 12-lead electrocardiogram demonstrated atrial fibrillation with a ventricular rate in the 60s. Transthoracic echocardiogram revealed normal left ventricular size and function, mild aortic valve regurgitation, and otherwise no significant abnormalities. The patient underwent ambulatory event monitoring that revealed atrial fibrillation with multiple ventricular pauses, up to 4 seconds, associated with swallowing and symptoms of significant pre-syncope (Figure 1). Barium esophagram did not demonstrate underlying esophageal pathology.

The patient underwent placement of a dual chamber permanent pacemaker with resolution of symptoms and ventricular pauses. After pacemaker placement, a challenge of carbonated beverage was given, resulting in bradycardia that initiated an appropriate pacing response (Figure 2). Subsequently, the patient has been free of recurrent syncope.

DISCUSSION
Deglutition syncope, or swallow syncope, was described as early as 1906. It is a relatively rare type of situational syncope that is related to the ingestion of solid foods or liquids and can be independent of underlying esophageal or cardiac disease. Through 2011, 80 cases have been reported in the English literature. Deglutition syncope can, however, be associated with various esophageal abnormalities such as spasm, carcinoma, and stricture. Additionally, there are cases that report association with achalasia, esophageal diverticulum, and hiatal hernia.

The typical presentation includes lightheadedness or syncope during or immediately after swallowing food or liquids. The episodes can be intermittent and unpredictable. The ingestion can be of any size, consistency, or temperature; however, carbonated
beverages have been the implicated agent in many cases. The physical examination and resting electrocardiogram are usually normal. Ambulatory electrocardiographic monitoring often reveals the causative bradyarrhythmia. Atrioventricular block is the most common cause of syncope and cases of sinus arrest, sinoatrial block, sinus bradycardia, and ventricular asystole have also been reported. The pathophysiology of this disorder is not completely understood; however, it appears that it is due to a hypersensitive vagal reflex triggered by mechanoreceptors in the esophagus in response to swallowing. Mechanoreceptors in the esophageal wall sense distension and send signals to the cardioinhibitory center in the medulla. The efferent limb of reflex pathway is postulated to be the vagus nerve, which innervates the sinoatrial and atrioventricular nodes. Vagal stimulation leads to nodal suppression that can result in slowing or cessation of conduction and transient inappropriate bradyarrhythmias. If prolonged, this can lead to a reduction of cardiac output, hypotension, diminished cerebral perfusion, and syncope. Additionally, there are reports that suggest the efferent pathway results in vasodilation. During continuous hemodynamic monitoring, a vasodepressor response has been noted followed by bradycardia. The decrease in systemic blood pressure preceding bradycardia may be due to withdrawal of sympathetic neural activity, activation of cholinergic vasodilation, or effects of local humoral substances such as nitric oxide. This vasodepressor mechanism is important to recognize as permanent pacemaker placement may not alleviate symptoms.

CONCLUSION
The diagnosis of deglutition syncope requires careful acquisition of the temporal relationship between swallowing solid food or liquids and lightheadedness or syncope. It can cause significant impairment of quality of life and can result in significant injury, especially if it occurs while driving. A thorough history is imperative to the diagnosis of this disorder.

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REFERENCES
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